Papillomatous Lesions in Wild Juvenile Atlantic Salmon, Salmo salar L., in New Brunswick, Canada

Author: Rand, Thomas G.

Source: Journal of Wildlife Diseases, 21(3) : 324-326

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-21.3.324

ly and only viral neoplasms occur with any apparent significant prevalence (Clark, 1973, Southwest. Vet. 26: 185–188). The adenomas of the thyroid in this 11-yr-old fisher agree with the observations of thyroid neoplasia in captive wild mammals, predominantly in older carnivores (Schlumberger, 1955, Brookhaven Symp. No. 7, pp. 168–191). Fisher longevity in the wild is usually less than 10 yr (Powell, 1982, The Fisher; Life History, Ecology, and Behavior, University of Minnesota Press, Minneapolis, 217 pp.). The oldest fisher in a recent survey in this laboratory of 396 trapper-harvested fishers was 7 yr.

The microscopic characteristics of the adenomas in the fisher were comparable to follicular adenomas of the canine thyroid (Leav et al., 1976, Am. J. Pathol. 83: 61–122) but had both small (microfollicular) and large (macrofollicular) irregular follicles containing varying amounts of colloid. Bilateral involvement was present in the fisher, but is uncommon in dogs and cats (Leav et al., 1976, op. cit.). Thyroid function was not assessed and clinical signs were not specific enough to determine if the thyroid was hyper- or hypofunctional.

Luteinized interstitial masses in the ovaries of the owl monkey (*Aotus trivirgatus*) are composed of a foamy outer layer of steroidogenic pigmented cells derived from cortical stroma. An inner mass of smaller, darker, nonpigmented inactive cells are derived from the luteinized theca interna of involuted follicles (Hertig et al., 1976, Lab. Anim. Sci. 26: 1041–1067). The origin of the interstitial cells and hormonal function in the fisher is unknown. The histologic and morphologic changes observed in the uterus were indicative of luteal function. The cause of the bilateral ovarian luteinization in an aged female fisher with no history of breeding is unknown.

The animal was raised in captivity which allowed her to reach an age not normally achieved under natural conditions. A review of the literature revealed little information on naturally occurring diseases of fishers. Additional research into parasites and diseases is needed in order to more effectively manage this animal.

Paraffin-embedded blocks of the adenoma described herein have been deposited in the Armed Forces Institute of Pathology, Washington, D.C. 20306, USA (NERCWD #84:9478; AFIP #1962601).

Scientific Contribution No. 1082, Storrs Agricultural Experiment Station, University of Connecticut, Storrs, Connecticut 06268, USA. We thank Ms. Joyce Czikowsky for technical assistance.

Journal of Wildlife Diseases, 21(3), 1985, pp. 324–326

Papillomatous Lesions in Wild Juvenile Atlantic Salmon, *Salmo salar* L., in New Brunswick, Canada

Thomas G. Rand, Bermuda Aquarium, Museum and Zoo, P.O. Box 145, Flatts, Bermuda

In September 1979, 144 Atlantic salmon parr were captured in Catamaran Brook, a small tributary of the Little Southwest Miramichi River, New Brunswick, Canada (46°51'N, 66°11'W). Greyish-white or pink lesions up to 2.5 cm in diameter and 2 mm thick were on the body or fin surfaces of six of 144 (4.2%) of the specimens (Fig. 1). The lesions grossly resembled papillomata described in several species of fish (Roberts, 1978,

Received for publication 20 March 1984.

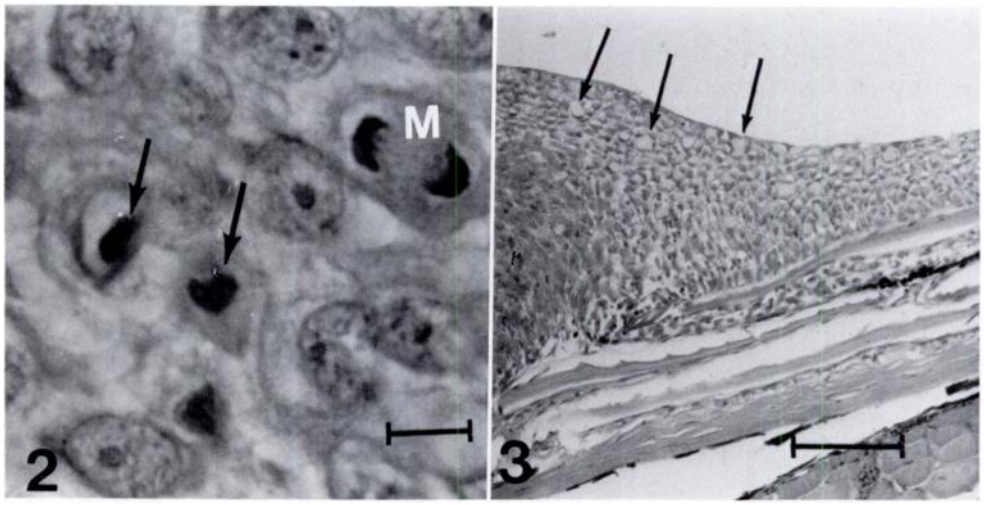
FIGURE 1. Papillomatous lesion on flank of an Atlantic salmon parr. (Bar = 1 cm.)

Fish Pathology, Bailliere Tindall, London, England, 318 pp.). Although papillomatous lesions have been reported in juvenile Atlantic salmon from several European countries and the United States (Carlisle and Roberts, 1977, J. Wildl. Dis. 13: 230–234), there are no published reports to date from juvenile Atlantic salmon in Canada.

All fish were fixed in acetic acid–formalin–alcohol immediately after capture. Pieces of skin and fins were excised and decalcified overnight in Kristensen's solution (Humason, 1972, Animal Tissue Techniques, Freeman, San Francisco, California, 546 pp.). They were then washed in cold, running tap water, dehydrated in an ethanol series, cleared in xylene, embedded in a commercial wax and sectioned at 7 μm. Sections were stained with Mayer's hematoxylin and eosin. Tissue samples have been deposited in the Armed Forces Institute of Pathology, Washington, D.C. 20306 (AFIP #1964946).

Lesions were composed of neoplastic cells which varied greatly in size and shape and which differed strikingly from normal epidermal cells. The neoplastic cells were generally smaller and more crowded, and arranged in a swirling pattern or in cords or whorls quite unlike the layered structure of the normal epidermis. The cytoplasm of most neoplastic cells was vacuolated and poorly stained. Nuclei were characteristically highly pleomorphic, hyperchromatic and anaplastic. Mitotic figures were numerous and the chromatin in most of the cells was coarse and clumped (Fig. 2). Pyknotic and necrotic cells were ubiquitous but larger necrotic areas were located centrally in the lesions. A few leukocytes were sometimes associated peripherally with these areas, indicating, possibly, a low level of inflammation. There was a sharp boundary between normal epidermis and papillomatous tissue (Fig. 3), marked by the greater density of the tumor cells, and the presence of a large number of mucus cells in the normal epidermis, which were absent within the lesions. Normal epidermis was displaced outwardly and overlapped the circumference of the lesions.

Lesions studied here were grossly and microscopically similar to papillomata described by Bylund et al. (1980, J. Fish Dis. 3: 525–528), Carlisle and Roberts (1977, op. cit.), and Chromwall (1976, Zoon 4: 109–114). Epidermal papillomata in wild and hatchery Atlantic salmon have been



FIGURES 2, 3. 2. Mitotic figure (M) and clumped chromatin (arrows) in nuclei of papillomatous cells of an Atlantic salmon parr. ×1,000. (Bar = 5 μm.) 3. Section through boundary area between normal epidermis and papillomatous tissue of an Atlantic salmon parr. The normal epidermis with abundant mucus cells (arrows) is seen at the right. The neoplastic cells which are smaller, more crowded and have lost the orderly pattern of the normal epidermis are seen at the left. ×100. (Bar = 100 μm.)

referred to as salmon wart disease (Bylund et al., op. cit.) and salmon pox (Roberts and Bullock, 1976, Oceanogr. Mar. Biol. Annu. Rev. 14: 227–246). They are benign proliferative lesions of the epidermis observed mainly in juvenile Atlantic salmon (Carlisle and Roberts, 1977, op. cit.) though older fish also can be afflicted (Bylund et al., 1980, op. cit.). Although the etiology of the disease is unknown, chemical agents which are claimed to cause papillomatous lesions in some fishes (Roberts, 1978, op. cit.) were ruled out as causative agents in salmon by Bylund et al. (1980, op. cit.) who observed the disease in wild salmon living under natural conditions unaffected by man. An infectious agent is indicated by the work of Bylund et al. (1980, op. cit.). They showed that in cultured fish the condition became epizootic in some tanks, but was absent in others with the identical food and water supplies. Carlisle (1977, J. Wildl. Dis. 13: 235–239) observed viruslike particles in papillomata, but there is still doubt whether these caused the lesions (Pitcher and Fryer, 1980, CRC Crit. Rev. Microbiol. 8: 1–25).

Papillomatosis is not usually considered a serious disease of salmon because the lesions slough and the fish usually recover (Carlisle and Roberts, 1977, op. cit.) although ulceration may occur. Mortality is generally low, though Bylund et al. (1980, op. cit.) reported an epizootic in a hatchery where about half the affected fish died. Secondary mycotic and bacterial infections which resulted in ulceration and probable loss of osmoregulatory function were implicated as causes of death of cultured salmon by Bylund et al. (1980, op. cit.) and Carlisle and Roberts (1977, op. cit.). As lesions disrupt normal skin coloration and mechanically impede swimming performance (Carlisle and Roberts, 1977, op. cit.) they perhaps make affected wild fish more susceptible to predation.

I am grateful to Mr. David Gluns, who helped to collect the fish and to Drs. David Cone and Michael Wiles who provided research facilities, helpful advice and generous hospitality at Saint Mary's University, Halifax, Nova Scotia, Canada for the preparation of this manuscript.