Sudden mortality caused by cardiac deformities following seining of preharvest farmed Atlantic salmon (*Salmo salar*) and by cardiomyopathy of postintraperitoneally vaccinated Atlantic salmon parr in British Columbia

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Case 1

This site contained approximately 450 000, intraperitoneally (IP) vaccinated Atlantic salmon, weighing 5 kg. The salmon were evenly distributed over 12 seacages, which measured 35 m^2 at the surface and extended to a depth of 17 m. The ambient water temperature was 8.5°C. The fish were fed a commercial ration. Approximately 10% to 15% of each preharvest seine of 3000 salmon rolled onto their backs within 20 min following seining. Mortality of up to 5% was recorded for each seine. Moribund salmon were observed lying motionless on the bottom of the seacage with muscular fasciculations of the flank.

On necropsy, hearts from fresh mortalities lacked the normal pyramidal shape. Cardiac deformities included a hypoplastic or aplastic septum transversum, with herniation of the elongated pyriform to crescent-shaped heart into the peritoneal cavity (leading to craniomedial ventral depression of the hepatic parenchyma and occasional adhesions); situs invertus (up-side-down heart within an intact pericardial sac); and ventricular hypoplasia with ascites. In a few fish, the liver and spleen were located on the opposite side within the peritoneal cavity and there were malformed (foreshortened or convoluted) swimbladders.

Microscopically, in virtually all the fish, there was moderate to marked thickening of myofibers and loss of sarcoplasmic striation, with hyalinization and occasional nuclear enlargement, extending from the outer compact layer of the myocardium moderately deeply into the spongy layer. There were also mild to moderate scattered accumulations of lymphocytes and plasma cells, and fewer histiocytes, around multiple intramural coronary vessels and, intermittently, throughout the epicardium. Within select sections of liver, there was mild, circumferential, subintimal fibrosis of the central vein. Serum samples taken from moribund fish that had cardiac deformities on necropsy had vitamin E and selenium levels within normal ranges. While the site was being harvested, sudden death attributed to cardiac deformities continued to occur following handling, crowding, grading, and transport. Cumulative preharvest seining mortality from cardiac deformities exceeded 20% of the total site population.

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Case 2

This case involved post-IP-vaccinated parr, weighing 18 g, that were maintained in outdoor, covered, 2-meter diameter, circular fiberglass tanks that received single-pass surface water at approximately 8°C. Each tank contained approximately 30 000 parr. Incubation temperatures ranging from 9°C to 14°C had been used earlier in the freshwater production phase in order to accelerate the growth from buttoning-up of the yolk sac through first feeding (0.15 g body weight (BW) up to parr (5.0 g BW). An arbitrary sample of 60 parr was collected from the tanks and sacrificed by anesthetic overdose. On necropsy, the hearts of 8 fish were found to be hypoplastic.

Microscopically, throughout the spongy layer of the ventricular myocardium and, to a much lesser extent, the atrial myocardium, there were multifocally extensive regions of myofiber degeneration; this was characterized by a loss of sarcoplasmic hyalinization or vacuolation and scattered compensatory nuclear enlargement. There were enlarged, vesicular to fusiform nuclei with rare nuclear rowing. There was mild to moderate endocardial hypertrophy and hyperplasia with scattered lymphohistiocytic infiltrate with occasional thrombus formation. Cumulative losses attributed to the above described cardiomyopathy for each tank transferred to seacages over the succeeding 12 wk averaged 5% to 10%, excluding culls, nonsmolts, precocious males, and transport losses.

Malformations of the heart, including hypoplastic septum transversum, situs invertus, hypoplasia or aplasia of the ventricular compact myocardium, and hemangiomas have been observed in Atlantic salmon from British Columbia raised in seacages (1). The mortality rate attributed to cardiac deformities has not been recorded. Factors contributing to cardiac deformities may include stock selection and high ambient water temperatures during early freshwater incubation (1,2). The common sequela from the cardiac deformities observed in this case was compensatory cardiac hypertrophy and reduced cardiac output, which presumably resulted in impaired cardiovascular function and failure. Cardiomyopathy syndrome (CMS), which has also been

termed "acute heart failure" or "heart rupture," has been described in marine-farmed Atlantic salmon stocks that were otherwise in good condition in Norway and the Faeroe Islands (1,2). It differs from the case reported here because CMS is a chronic progressive disease with serious losses, typically occurring in the autumn, 12 to 18 mo after transfer to seacages at an average bodyweight of 3 to 4 kg. Moribund CMS fish were lethargic. There was marked exophthlmos, pitting of the skin due to edema and congestion, and hemorrhage around the pectoral fins. In addition, lesions due to CMS typically included fibrinous peritonitis, ascitic fluid, and blood or blood clots surrounding the heart. The atrium and sinus venosus were usually dilated and contained clots. The microscopic lesions of CMS are similar to the lesions recorded from this case (1,2). Although infectious agents, such as nodavirus (3) and *Diphylbothrium dendriticum*, have been associated with CMS in production stocks, the history of elevated ambient water temperature to accelerate growth in fry under 5.0 g BW and the lack of demonstrable pathogens suggest a metabolic or production etiology rather than an infectious disease (2).

References

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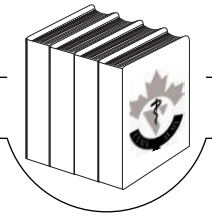
3. Sindre G, Totland GK, Kryvi H. Detection of a nodavirus-like agent in heart tissue from reared Atlantic salmon (*Salmo salar*) suffering from cardiac myopathy syndrome (CMS). Dis Aqua Org 1997;29:79–84.

BOOK REVIEW



COMPTE RENDU DE LIVRE

Samuel WM, Pybus M, Kocan AA. ***Parasitic Diseases of Wild Mammals.*** Iowa State University Press, Ames, 2001, ISBN 0-8138-2978-X, US$94.95.

We are fortunate to share Canada with a splendid wildlife resource. At home, traveling, or on vacation, interactions with animals in the wild enhance our understanding of nature and our responsibilities to our ecosystem, enrich the landscape, and liberate our spirit. The past several decades have generated major developments in our knowledge of many aspects of the biology of wild animals, in particular factors that may affect wildlife health. Canadians have made many significant contributions to this progress.

In 1971, John Davis and Roy Anderson edited *Parasitic Diseases of Wild Mammals.* Now, 30 years later, Bill Samuel, Margo Pybus, and Alan Kocan have assembled the "long overdue" 2nd edition. Read the preface, a stimulating overture, and you will know that this new book, like its predecessor, is a landmark in the literature of wildlife parasitology. The editors have selected authors who have written with great authority, clarity, and a generally uniform approach on arthropod, helminth, and protozoan parasites that meet carefully selected criteria: they cause disease in wild mammals, have the potential to cause disease, are of zoonotic importance; are "emerging" diseases, or are of particular public interest. Twenty-five groups of parasites are included.

This book has many strengths. Perhaps the greatest is the thoroughness of the material presented. Each chapter is an excellent, comprehensive review, and almost all chapters include information summarized in clear and concise tables. Other strengths are the broad range of parasites included and the worldwide view, although there is perhaps a slight emphasis on North America. One caution! Obviously, this book does not include all wild mammals or all parasites of the hosts listed in the index, only those that meet the criteria mentioned above. Another strength, making the book attractive to a nonspecialist audience, is its freedom from complex parasitological and disease terminology. The book is well designed, clearly printed — other than some of the photographs — and strongly bound. The lists of literature cited are very helpful — other than the occasional missed reference — while the index, a good measure of user-friendliness, is exemplary in its detail. This book should be in the libraries of all those working in wildlife biology, parasitology, or veterinary medicine. It will serve as a valuable resource for those in the field, and in management agencies, in research and educational institutions, and for a variety of students.

Any deficiencies do not compromise the book's great value. Although several authors refer to the population effects of selected parasites on their hosts, perhaps the next edition will contain a chapter focused on some of the recent observational, experimental, and theoretical studies that have pushed forward this intriguing and important area of wildlife parasitology. Such a chapter could serve as a useful overview of the principles underlying the relationships between wild animals, their parasites, and their population health. Also, as the world faces climate change, a chapter addressing the interface between this phenomenon and wildlife parasitic disease, even from a theoretical perspective, would have been timely. And what happened to a chapter on "pulmonary" lungworms?

In summary, this book is effective on several levels. It is an excellent resource for people interested in wild mammals and their parasites, it is an exciting introduction for those entering the disciplines of wildlife biology or wildlife parasitology, and, perhaps inadvertently, it is an outstanding text on aspects of host-parasite biology. A book of a similar scope and standard on parasites of domestic animals would be a challenge, but surely it would be widely attractive.

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