

Unravelling the common etiology of related, but pathologically divergent, diseases

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Abstract

Piscine orthoreovirus Strain PRV-1 is the causative agent of heart and skeletal muscle inflammation (HSMI) in Atlantic salmon (*Salmo salar*). Given its high prevalence in net pen salmon, debate has arisen on whether PRV poses a risk to migratory salmon, especially in British Columbia (BC) where commercially important wild Pacific salmon are in decline. Various strains of PRV have been associated with diseases in Pacific salmon, including erythrocytic inclusion body syndrome (EIBS), HSMI-like disease, jaundice syndrome, and jaundice/anemia in Japan, Norway, Chile and Canada. Herein, we examine the developmental pathway of HSMI and jaundice/anemia associated with PRV-1 in farmed Atlantic and Chinook (*Oncorhynchus tshawytscha*) salmon in BC, respectively. A molecular viral disease development (VDD) biomarker panel differentiated fish that were merely viral carriers from those in an active viral disease state, and In situ hybridization (ISH) localized PRV-1 within developing lesions in both diseases. The two diseases showed dissimilar pathological pathways, as indicated by the preponderance of inflammatory lesions in heart and skeletal muscle in Atlantic salmon, and the development of degenerative-necrotic lesions in kidney and liver in Chinook salmon. However, our data indicate that a species-related difference in PRV load tolerance in red blood cells can explain these differences. Moreover, complete viral genome sequencing revealed no consistent differences in the sequence of PRV-1 variants intimately involved in the development of both diseases, suggesting that migratory Chinook salmon may be at more than a minimal risk of disease from exposure to the high levels of PRV occurring on salmon farms.

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Biography

Kristina Miller holds a PhD from Stanford University (1992) and is currently the Head of Genetics and Genomics at Fisheries and Oceans Canada. She is also an adjunct professor at UBC. Dr. Miller is on the

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