

In vitro and *in vivo* properties and pathogenesis of the white sturgeon iridovirus,
WSIV, in juvenile white sturgeon, *Acipenser transmontanus*

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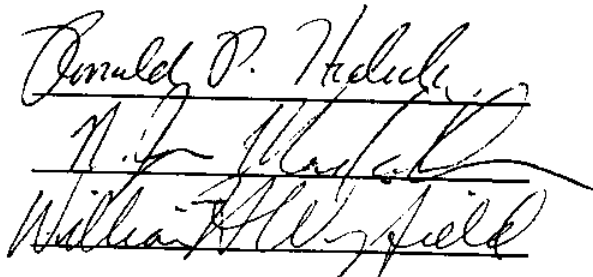
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Abstract

A cell line derived from sturgeon gonadal tissues, WSGO, produced up to 12-fold more white sturgeon virus (WSIV) than the reference spleen cell line (WSS-2). Phase photography and transmission electron microscopy revealed that cell hypertrophy, hyper-refractility and dense cytoplasmic inclusions were associated with proliferation of cytoplasmic vesicles and viral assembly sites. *In vitro* properties that were consistent with chronic *in vivo* infections included a lengthy multiplication cycle, low concentration of WSIV produced and released from the cell and non-cytolytic cytopathic effect. In comparison with other members of the Iridoviridae, the biological properties of WSIV most closely resembled those of the fish lymphocystiviruses (FLDV).

Juvenile white sturgeon were bath-challenged and one of the key histopathological indicators was a clear pericellular space that surrounded individual enlarged Malpighian cells. Microscopic signs were observed up to 10 d before gross clinical signs appeared. In addition, previously unknown sites of infection were observed in the olfactory organs,

oropharyngeal cavity and barbels which elucidated the pathogenic mechanism of WSIV and the cause of the associated wasting syndrome.

An indirect fluorescent antibody test (IFAT) was developed to detect WSIV in tissue imprints of bath-challenged sturgeon juveniles. WSIV antigens were prominent in epithelial cells of olfactory, gill and esophageal tissues during the initial stages of infection and the intensity of fluorescent staining paralleled the tissue concentrations of WSIV. Imprints of the gills and olfactory organs provided a rapid and accurate means of diagnosing inapparent WSIV carriers and this approach required fewer personnel and less processing time than conventional methods .

Sturgeon fry were bath-challenged with WSIV and the course of infection was monitored at water temperatures of 10, 14, 19 and 23°C. Mortality rates were significantly elevated at higher water temperatures whereas plasma protein concentrations and hematocrits were lower. WSIV-infected fish held at lower water temperatures experienced prolonged epizootics, elevated cumulative mortality and chronically depressed condition factor (K), hepatosomatic index (HSI) and specific growth rate (G). Gill hyperplasia was more common at higher water temperatures whereas olfactory lesions were prominent at low temperatures. WSIV-induced anorexia and starvation, due to lesions in the chemosensory and tactile tissues of the olfactory organs, esophagus and barbels, could explain the pathological basis of the wasting syndrome.

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