High virulence differences among phylogenetically distinct isolates of the fish rhabdovirus viral hemorrhagic septicaemia virus are not explained by variability of the surface glycoprotein G or the non-virion protein Nv

Viral hemorrhagic septicaemia virus (VHSV) is an important viral pathogen in European rainbow trout farming. Isolates from wild marine fish and freshwater trout farms show highly different virulence profiles: isolates from marine fish species cause little or no mortality in rainbow trout following experimental waterborne challenge, whilst challenge with rainbow trout isolates results in high levels of mortality. Phylogenetic analyses have revealed that the highly virulent trout-derived isolates from freshwater farms have evolved from VHSV isolates from marine fish host species over the past 60 years. Recent isolates from rainbow trout reared in marine zones show intermediate virulence. The present study aimed to identify molecular virulence markers that could be used to classify VHSV isolates according to their ability to cause disease in rainbow trout. By a reverse genetics approach using a VHSV-related novirhabdovirus [infectious hematopoietic necrosis virus (IHNV)], four chimaeric IHNV–VHSV recombinant viruses were generated. These chimaeric viruses included substitution of the IHNV glyco- (G) or non-structural (Nv) protein with their counterparts from either a trout-derived or a marine VHSV strain. Comparative challenge experiments in rainbow trout fingerlings revealed similar levels of survival induced by the recombinant (r)IHNV–VHSV chimaeric viruses regardless of whether the G or Nv genes originated from VHSV isolated from a marine fish species or from rainbow trout. Interestingly, recombinant IHNV gained higher virulence following substitution of the G gene with those of the VHSV strains, whilst the opposite was the case following substitution of the Nv genes.

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