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Viral haemorrhagic septicaemia virus (VHSV) is the economically most important viral disease in European rainbow trout farming. The virus was introduced to fresh water farms in the 1950ies from a reservoir of VHSV in the marine environment. Isolates from wild marine fish and fresh water farms are difficult to distinguish serologically but they show different virulence profiles: marine isolates typically cause little or no mortality in rainbow trout fry following experimental waterborne challenge, while freshwater isolates often kill the majority of the fish. Genetic analysis reveal that the change in host range (to include rainbow trout) likely have occurred several times. Virus from the marine environment therefore continues to represent a threat to the expanding trout aquaculture industry in the marine environment. Identification of potential virulence markers are therefore of great importance.
By a reverse genetics approach using the related novirrhabdovirus infectious hematopoietic necrosis virus (IHNV) as basis, four hybrid IHNV-VHSV variants were generated. These chimeric variants included substitution of the IHNV glyco(G) or nonstrutrual (Nv) protein with the corresponding G or Nv-protein from either a freshwater or a marine VHSV strain. Following rescue of the hybrid viruses, comparative challenge experiments in rainbow trout fingerlings have been performed. The pathogenicity of the recombinant IHNV-VHSV hybrid viruses were similar, regardless of whether the G or Nv originate from marine or fresh water VHSV. Recombinant IHNV gained higher virulence following substitution of the homologous G gene with the VHSV G gene, while the opposite was the case following substitution of the Nv gene. These findings suggest that higher virulence of VHSV compared to IHNV might be related to the G protein, while the VHSV Nv may not efficiently support *in vivo* propagation of IHNV.

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