Generation of an infectious clone of AMDV and identification of capsid residues essential for infectivity in cell culture.

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Abstract: Pathogenic strains of Aleutian mink disease virus (AMDV) such as Utah-1 do not replicate in cell culture (e.g., Crandell Rees feline kidney cells) while the in vitro-adapted AMDV strain ADV-Gorham (ADV-G) is not pathogenic. Here, we constructed a full-length infectious clone (pADV-G). Alignment of the VP2 gene of ADV-G with that of other AMDV strains revealed many amino acid (a.a.) residues conserved among pathogenic isolates that differed in ADV-G. Four virulence-associated, conserved residues of pADV-G VP2 were studied by site-directed mutagenesis (H92A, Q94S, Y115F, and I116L). Mutation of residue 92 or 94 decreased viral-transcription and viral-infectivity levels, whereas mutation of residue 115 or 116 did not affect viral-infectivity in CRFK cells. These results indicated that VP2 residues 92 and 94, both located on the surface of the viral capsid, are critical for AMDV infectivity in vitro.

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