

Salmon cells SHK-1 internalize infectious pancreatic necrosis virus by macropinocytosis

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We have previously shown that infectious pancreatic necrosis virus (IPNV) enters the embryo cell line CHSE-214 by macropinocytosis. In this study, we have extended our investigation into SHK-1 cells, a macrophage-like cell line derived from the head kidney of Atlantic salmon, the most economically important host of IPNV. We show that IPNV infection stimulated fluid uptake in SHK-1 cells above the constitutive macropinocytosis level. In addition, upon infection of SHK-1 cells, IPNV produced several changes in actin dynamics, such as protrusions and ruffles, which are important features of macropinocytosis. We also observed that the Na⁺/H⁺ pump inhibitor EIPA blocked IPNV infection. On the other hand, IPNV entry was independent of clathrin, a possibility that could not be ruled out in CHSE 214 cells. In order to determine the possible role of accessory factors on the macropinocytic process, we tested several inhibitors that affect components of transduction pathways. While pharmacological intervention of PKI3, PAK-1 and Rac1 did not affect IPNV infection, inhibition of Ras and Rho GTPases as well as Cdc42 resulted in a partial decrease in IPNV infection. Further studies will be required to determine the signalling pathway involved in the macropinocytosis-mediated entry of IPNV into its target cells.