Effects of Z-FA-FMK drug treatment on aquatic reoviral and rhabdoviral replication

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Piscine reovirus (PRV) is a reovirus correlated with heart and skeletal muscle inflammation (HSMI) in Norwegian Atlantic salmon (*Salmo salar*) but does not cause apparent symptoms in infected BC fish. Z-FA-FMK is a drug that irreversibly inhibits cysteine proteases and has been shown to inhibit reoviral replication and infectious particle production. This research tested this compound to observe if PRV replication is effectively inhibited and to assess whether it affects other viruses such as infectious hematopoietic necrosis virus (IHNV). Atlantic salmon erythrocytes were infected with PRV, treated with 2, 20, and 200 μM Z-FA-FMK, and e*x vivo* PRV transcription levels and erythrocyte were assessed. Additionally, live Atlantic salmon were injected with PRV inoculum along with 2000 μM Z-FA-FMK. IHNV was tested for Z-FA-FMK susceptibility by infecting CHSE-214 (Chinook salmon embryo) cells, which were treated with 2, 20, and 200 μM Z-FA-FMK, and transcription levels and plaque assays were used to determine susceptibility. No significant inhibition of PRV transcription was observed for 2 and 20 μM concentrations and the decrease in transcription observed at 200 μM was most likely a result of decreased cell viability due to necrosis/apoptosis induction by the drug. PRV was not significantly affected by the drug *in vivo* with live fish. IHNV transcription and viral particle formation were not affected by the drug at 2 and 20 μM concentrations but were at 200 μM concentrations. The results indicate that PRV infects host cells with different proteins than most reoviruses and is notably different in its mechanism of infection.