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Juvenile gilthead sea bream immune response against RGNNV or RGNNV/SJNNV infections

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Viral nervous necrosis is one of the main threats for the Mediterranean aquaculture. It is caused by the nervous necrosis virus (NNV, *Betanodavirus* genus), with a genome composed of two positive-sense, ssRNA segments: RNA1, encoding the viral polymerase; and RNA2, encoding the capsid protein. Betanodaviruses have been clustered into four species: RGNNV, SJNNV, BFNNV and TPNNV. In addition, reassortment between RGNNV and SJNNV segments has also been detected. NNV can affect a wide range of fish species; however, not all viral species are equally virulent to all susceptible fish species; this issue is especially relevant concerning European sea bass and gilthead sea bream, which are frequently co-cultured. Sea bass is highly susceptible to RGNNV, whereas RGNNV/SJNNV (RNA1/RNA2) reassortants cause low mortality in this fish species. On the contrary, RGNNV/SJNNV reassortants cause high mortality in sea bream larvae, and this fish species is reluctant to RGNNV infections. The outcome of viral infections depends on the specific virus-host interaction. Understanding the mechanisms responsible for this differential interaction is crucial to control viral diseases in aquaculture. This study focuses on the analysis of the immune gene transcription in brain of 3-g sea bream experimentally infected with betanodaviruses isolated from sea bass (DlNNV, RGNNV) or sea bream (SaNNV, RGNNV/SJNNV). We have analysed genes suggested to be relevant in controlling RGNNV infection in sea bass: inflammatory genes, apoptotic genes, stress genes, and IFN I-system-related genes.

Mortalities or symptoms were not recorded in any experimental group for 30 days, and the quantification of viral genome evidenced that only SaNNV replicated in sea bream brain. Principal component analysis clustered samples according to the viral isolate from 1 day post-infection onwards, and evidenced differences in the immune response against both viruses. Specifically, sea bream response against DlNNV is characterized by a higher *rtp3* transcription early after the infection, a longer-lasting transcription of the anti-inflammatory gene *il-10* and a stronger induction of *casp1* and *hsp70*. These genes should be targets for future studies in order to elucidate their role in hampering the replication of RGNNV in sea bream.

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