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Senegalese sole immune response against betanodavirus recombinants harboring modifications in the 3' terminal region of the RNA1

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The nervous necrosis virus (NNV) is the etiological agent of the viral nervous necrosis (VNN), a disease affecting a high number of fish species worldwide. NNV genome is composed of two segments RNA1 and RNA2, encoding the RNA-dependent RNA polymerase and the capsid protein, respectively. NNV has been classified into four species: striped jack nervous necrosis virus (SJNNV), tiger puffer nervous necrosis virus (TPNNV), red-spotted grouper nervous necrosis virus (RGNNV), and barfin flounder nervous necrosis virus (BFNNV). Furthermore, reassortants between RGNNV and SJNNV have been reported, such as wt160 isolated from Senegalese sole (*Solea senegalensis*), which presents a RGNNV-RNA1 and a SJNNV-RNA2 type segments, and causes 100% mortality in this fish species. This isolate exhibited differences in the 3' non-coding region (NCR) of both genomic segments when compared to the reference strains of each genotype.

In this study, the effect on virulence of the substitutions observed in the 3'NCR of the wt160-RNA1 has been evaluated, by the development of two recombinants harbouring mutations at position 3073 and 3093, which make the wt160-RNA1 similar to the reference RGNNV. Moreover, immune response of Senegalese sole against the infection with these recombinants compared to the wild-type, has been evaluated using an 112-OpenArray®.

The infection with the recombinants r3073 and r3093 decreased the mortality to 29.3% and 25.3%, respectively. Furthermore, the number of differential expressed genes (DEGs) was higher at 3 days than at 2 days post-inoculation (p.i.), after the infection with the three viruses, being the number of DEG quite similar among viruses. Significant differences between DEG fold changes after infection with the mutants and the wt160 will be discussed. It should be highlighted that at 2 days p.i., the interferon stimulated gene (ISG) gig1 was not expressed after the infection with r3073 and r3093. However, at 3 days p.i. this gene was expressed at the highest level after the infection with the three viruses. Moreover, the infection with the wt160 induced the down-regulation of the genes gilt (related to antigen processing and presentation) and magel2 (related to protein ubiquitination process), which was not observed after infections with r3073.