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Understanding the role of European sea bass RTP3 protein in the course of betanodavirus infections

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Nervous necrosis virus (NNV, Betanodavirus genus) is the causative agent of the viral nervous necrosis, the main viral disease affecting European sea bass. Betanodavirus genome is composed of two single-stranded, positive-sense segments: RNA1 (viral polymerase) and RNA2 (capsid protein), and this virus has been classified into four species, being RGNNV and SJNNV the viral species most frequently detected in the Mediterranean area. Numerous studies have pointed out the importance of the host immune response to defeat betanodavirus infections, highlighting the relevant transcription of a new virus responsive gene, rtp3. This gene encodes the RTP3 protein, which belongs to the receptor-transporting protein (RTP) family. Previously, our research group has described two rtp3 genes within the genome of European sea bass (rtp3X1, rtp3X2). The aim of the present study is to analyse the role of sea bass RTP3 proteins against betanodavirus infections, performing an in vivo transcription analysis and evaluating the RTP3 anti-NNV activity using an in vitro approach. The transcription profile of rtp3X1 and X2 was analysed by qPCR in sea bass injected with LPS, poly I:C, or RGNNV. The results revealed significant rtp3X1 and X2 transcription after LPS and poly I:C inoculation, although the values obtained for rtp3X2 were higher than those recorded for rtp3X1. The highest transcription of both rtp3 genes was induced by RGNNV infection, recording upregulation of rtp3X2 extremely high in brain. The anti-NNV activity was evaluated against RGNNV and SJNNV infections, inoculating E-11 cell lines permanently expressing the sea bass RTP3 proteins and quantifying viral replication in those cells, using E-11 cells as control. In RTP3 X1-expressing cells, the number of RGNNV RNA1 and RNA2 copies were significantly higher compared with values detected in E-11 cells, whereas in RTP3 X2-expressing cells a significant reduction of both viral segments was recorded. These results were corroborated by viral titration. On the contrary, viral replication of SJNNV was not significantly lower by the presence of any RTP3 protein. This study contributes to further understand the European sea bass response against betanodavirus, suggesting an important role of RTP3 X2 in controlling NNV infections. Funding: Agencia Estatal de

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