

NODAVIRUS INFECTION ALTERS THE KISSPEPTINS PATHWAY IN EUROPEAN SEA BASS BRAINY. VALERO¹, A. CUESTA², M. ARIZCUN¹, M.A. ESTEBAN², J. MESEGUER², E. CHAVES-POZO^{1*}¹*Instituto Español de Oceanografía, Puerto de Mazarrón, Spain*²*University of Murcia, Murcia, Spain*

In vertebrates, the kisspeptins pathway in the brain is an essential upstream regulatory element of the hypothalamus - pituitary - gonad (HPG) axis, which regulates reproduction. In European sea bass it has been described two forms of kisspeptin genes, kisspeptin 1 (*kiss1*) and kisspeptin 2 (*kiss2*), and two forms of G-coupled protein receptor for kisspeptins, *gpr54-1b* and *gpr54-2b*. The kisspeptins pathway activation is involved in the release of GnRH in the brain, which subsequently, is involved in the release of the gonadotropin hormones (LH and FSH) in the pituitary, and in turn acts in the regulation of the steroidogenesis and gametogenesis in the gonad. One of the pathogens that greatly affects the brain is nodavirus (VNNV), a known vertically transmitted virus, which causes the viral encephalopathy and retinopathy (VER) disease. Previous studies showed that, in addition to infect the brain and cause a considerable fish death in European sea bass specimens, VNNV is able to colonize and replicate into the gonad, altering the sex steroid hormone levels and triggering a high immune response into the tissue. In this study, we have determined the alteration on the expression levels of *kiss1* and 2, *gpr54-1b* and -2b, *gnrh1*, 2 and 3 genes and also the GnRH receptor (*gnhr2a*) gene in the brain of European seabass males upon VNNV intramuscular or intravitreally infection. This study represents an advance on our knowledge about the interaction between host and VNNV, needed to understand how this virus is able to avoid the immune response of adult fish to spread to the progeny.

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