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Molecular characterization of Glutaminyl-peptide cyclotransferase（QPCT）in *Scylla paramamosain* and its role in *Vibrio alginolyticus* and white spot syndrome virus (WSSV) infection

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Abstract

Glutaminyl-peptide cyclotransferase (QPCT) catalyzes the posttranslational modification of an N-terminal glutamate of proteins to pyroglutamate. This renders the protein more resistant to protease degradation, more susceptible to hydrophobic interactions, aggregation, and neurotoxic. In this study, we evaluated the influence of QPCT in the crab *Scylla paramamosain* infected with white spot syndrome virus (WSSV) or with *Vibrio alginolyticus*. A cDNA clone, encompassing the entire 2,445 bp of the *S. paramamosain* QPCT gene, containing a 1,113 bp open reading frame (ORF) encoding a 370 amino acid protein was cloned from *S. paramamosain*. Real-time PCR indicated that *QPCT* was primarily expressed in the digestive tract of *S. paramamosain*, was up-regulated in hemocytes after infection with *V. alginolyticus*, and down-regulated in hemocytes after infection with WSSV. Knockdown of *QPCT* expression by double-stranded RNA (QPCT-dsRNA) resulted in down-regulation of prophenoloxidase (proPO) and crustin antimicrobial peptide, whereas myosin-II-essential-light-chain-like-protein was significantly up-regulated in hemocytes at 24 h post QPCT-dsRNA treatment. WSSV challenge in crabs treated with QPCT-dsRNA resulted in a reduction in viral burden and in the apoptotic rate of crab hemocytes, while the phagocytic activity of crab hemocytes and overall mortality rate were increased. This suggests that WSSV might take advantage of QPCT to benefit its replication. In contrast, *V. alginolyticus* infection in crabs treated with QPCT-dsRNA indicated that the apoptotic rate and phagocytic activity of hemocytes, and overall incidence of mortality, were increased compared to mock-treated animals, indicating