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

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12 **Abstract**

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

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