

Functional characterization of Pacific white shrimp *Litopenaeus vannamei* heat shock protein 90 (LvHSP90) in response to white spot syndrome virus (WSSV) infection

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Abstract: Stress proteins (SPs) including heat-shock proteins (HSPs), are molecular chaperones essential for cellular homeostasis. The functions of HSPs include chaperoning misfolded proteins, protecting cells from stress, and participating in the host immune responses. Moreover, HSPs also cooperate with numerous viruses to promote viral infection. In this study, we investigate the role of heat shock protein 90 (LvHSP90) in white spot syndrome virus (WSSV) infection in Pacific white shrimp *Litopenaeus vannamei*. The expression of LvHSP90 was significantly up-regulated upon white spot syndrome virus (WSSV) infection. Gene silencing of LvHSP90 followed by WSSV infection resulted in 60% higher cumulative mortality rate in the LvHSP90-silenced shrimp. In addition, the transcription of WSSV gene were highly decreased in the LvHSP90-silencing group. In WSSV infected hemocyte cells, LvHSP90 protein showed accumulation in the nuclear zone, where viral replication is located and the number of WSSV-infected cells was significantly lower than control groups after LvHSP90 silencing. Injection of shrimp with WSSV that co-incubated with recombinant LvHSP90 (rLvHSP90) showed an increased mortality rate up to 90% compared to only 40% mortality in the control groups and shrimp also had increased viral copy number. Taken together, these results suggested that LvHSP90 plays a crucial role in promoting WSSV infection.

Keywords: Heat shock protein 90; *Litopenaeus vannamei*; white spot syndrome virus



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