Developmental and Comparative Immunology 63 (2016) 18–26

Contents lists available at ScienceDirect

Developmental and Comparative Immunology

journal homepage: www.elsevier.com/locate/dci

Dorsal transcription factor is involved in regulating expression of crustin genes during white spot syndrome virus infection

Xin Huang, Wen Wang, Qian Ren*

Jiangsu Key Laboratory for Biodiversity & Biotechnology and Jiangsu Key Laboratory for Aquatic Crustacean Diseases, College of Life Sciences, Nanjing Normal University, 1 Wenyuan Road, Nanjing 210046, PR China

ARTICLE INFO

Article history:
Received 4 March 2016
Received in revised form
11 May 2016
Accepted 11 May 2016
Available online 13 May 2016

Keywords:
Dorsal
Crustins
White spot syndrome virus
Innate immunity
Macrobrachium rosenbergii

ABSTRACT

Nuclear factor-kappa B (NF-κB) pathways play important roles in innate immune responses. In this study, we identified a dorsal homolog (*MrDorsal*) from freshwater prawn *Macrobrachium rosenbergii*. The full-length cDNA of *MrDorsal* comprised 2533 bp with an open reading frame of 1986 bp, which encoded a peptide of 661 amino acid residues. Amino acid sequence analysis showed that MrDorsal contains a Rel homolog domain and an IPT/TIG (i.e., Ig-like, plexin, and transcription factors) domain. The signature sequence of dorsal protein FRYMCEG existed in the deduced amino acid sequence. Sequence analysis showed that MrDorsal shared high similarities with Dorsal from invertebrate species. *MrDorsal* was abundant in the hemocytes and gills of healthy prawns but minute levels were detected in other tissues. The expression of *MrDorsal* was significantly upregulated 48 h after the white spot syndrome virus (WSSV-) challenge. Knockdown of *MrDorsal* using double-stranded RNA could suppress the transcription of *crustin* genes (*MrCrustin2 and MrCrustin4*) in gills of prawns after 48 h of the WSSV challenge. Results indicated that *MrDorsal* was involved to regulate the expression of *crustin* genes and it might play potential important roles during WSSV infection.

1. Introduction

Innate immune system is crucial in host defense against animal pathogens. The innate immune response is activated by pattern recognition receptors that recognize pathogen-associated molecular patterns, such as lipopolysaccharides, peptidoglycans, and mannans (Medzhitov and Janeway, 1997). Pathogenic infection triggers diverse humoral and cellular activities through signal transduction pathways, which are broadly conserved in both insects and mammals (Borregaard et al., 2000). Among intracellular signaling cascades, Rel/NF-κB family plays a central role in the transcription of some immune effectors (Hoffmann and Reichhart, 2002). The Rel/NF-κB family of transcription factors regulate gene expression in inflammation, immune response, apoptosis, embryonic morphogenesis, cell proliferation, and differentiation (Dixit and Mak, 2002; Govind, 1999; Hayden and Ghosh, 2008). The mammalian Rel/NF-κB family includes p65 (RelA), RelB, c-Rel, p50/p105, and p52/p100, which can be divided into two classes. Three members have been identified in *Drosophila melanogaster*, including Relish, Dorsal, and Dif (Ghosh et al., 1998). They all share a highly conserved Rel homology domain (RHD) in the N-terminal region, which is responsible for DNA binding, dimerization and interaction with the regulatory proteins. Nuclear localization signal is a short sequence located at the C-terminus of RHD and is required in the case of nuclear import.

In *D. melanogaster*, two major signal pathways (Toll or Imd pathway) leading respectively to the production of antifungal or antibacterial peptides (*AMPs*) have been reported (De Gregorio et al., 2002; Hoffmann and Reichhart, 2002). Dorsal and Dif are two of the NF-κB proteins that interact with Cactus, an IκB-related inhibitor. Degradation of Cactus in response to Toll receptor signaling releases Dorsal or Dif for nuclear translocation and interaction with target gene regulatory sites (Busse et al., 2007). In *Drosophila*, the Toll pathway is important in response to infection of fungi, several Gram-positive bacteria, and virus through the synthesis of *AMPs* (Rutschmann et al., 2000, 2002; Valanne et al., 2011). Similar to mammalian p105, Relish is triggered by the Imd pathway in response to infection of Gram-negative bacteria, which activates the gene expression of *AMPs* (Lemaitre and Hoffmann, 2007).

As invertebrates, shrimps lack a true adaptive immune system and rely on various innate immune responses to fight against

* Corresponding author.
E-mail address: renqian0402@126.com (Q. Ren).

http://dx.doi.org/10.1016/j.dci.2016.05.006
0145-305X/

(A)

```
agtcagtgcccagtagtagacttaactgtgcctctgctcggtttggtcccctgggagatga
gggcgtcaattggcagtgactccttctaagtattcgtgtgctggcggtatggcggaccca
                                                  M  A  D  P     4
gggtctgtgagcatgcgaaacaccaacacgctcgactataacatcagcgatatcattgat
 G  S  V  S  M  R  N  T  N  T  L  D  Y  N  I  S  D  I  I  D    24
gttatcaaagaagactaccaagacttagagacagaatatggcggagccgttgtcggaagt
 V  I  K  E  D  Y  Q  D  L  E  T  E  Y  G  G  A  V  V  G  S    44
gcgataggcagttcagacaattactctgtcgtgacgcaggaatcttgcaacgatcctgat
 A  I  G  S  S  D  N  Y  S  V  V  T  Q  E  S  C  N  D  P  D    64
tatgcactcaaaaggaaagcttacgtgaaaatattggagcagcctcaggccaaagctctg
 Y  A  L  K  R  K  A  Y  V  K  I  L  E  Q  P  Q  A  K  A  L    84
cgatttcgttatatgtgcgagggtcggtcggctgggtcgatacctggagttcgaagcact
 R  F  R  Y  M  C  E  G  R  S  A  G  S  I  P  G  V  R  S  T   104
gcagataataaaactttcccaggcatacaggtggttggttacaagggtcccgcggtagtc
 A  D  N  K  T  F  P  G  I  Q  V  V  G  Y  K  G  P  A  V  V   124
gtagtgtcgtgtgtaaccgtcgatccccctacagaccgcacccccacaacctggtcggg
 V  V  S  C  V  T  V  D  P  P  Y  R  P  H  P  H  N  L  V  G   144
aaagaaggctgtaaaaaaggaatatgtacaatgacgatcagtagcgacaccatgcagtgc
 K  E  G  C  K  K  G  I  C  T  M  T  I  S  S  D  T  M  Q  C   164
atgttctcgaacttgggaatacagtgcatcaaaaagcgcgacgtggaagacgctctgaaa
 M  F  S  N  L  G  I  Q  C  I  K  K  R  D  V  E  D  A  L  K   184
ctgagagaggagattcgcgttgacccttttcaaactggattcagccatcgaaaccagcct
 L  R  E  E  I  R  V  D  P  F  Q  T  G  F  S  H  R  N  Q  P   204
cagagtatcgacctgaattcgctaagactgtgtttccaagtgttcctcgaaggctcggag
 Q  S  I  D  L  N  S  L  R  L  C  F  Q  V  F  L  E  G  S  E   224
aagggcaaatttacgttccccttgaagccagtggtgtctgatcctatatatgacaaaaaa
 K  G  K  F  T  F  P  L  K  P  V  V  S  D  P  I  Y  D  K  K   244
gccgcctgtgatctcaacatatgcaaattgagtgattgcacgagtagtgttgctggcggt
 A  A  C  D  L  N  I  C  K  L  S  D  C  T  S  S  V  A  G  G   264
aaagaaataatactcttatgcgagaaagtcactaaagaagacatccaggtacgcttttat
 K  E  I  I  L  L  C  E  K  V  T  K  E  D  I  Q  V  R  F  Y   284
gaagtcaaagacggaaggatagaatgggaggcattcggggatttcaggcttctgatgta
 E  V  K  D  G  R  I  E  W  E  A  F  G  D  F  Q  A  S  D  V   304
cataaacaggtggcaatatccttcaaaactccaagatataagacactggaggtggaaaat
 H  K  Q  V  A  I  S  F  K  T  P  R  Y  K  T  L  E  V  E  N   324
cctgtgaaagtatttgtacaactactgcgaccaacggacaagagcaccagcgaaccaagg
 P  V  K  V  F  V  Q  L  L  R  P  T  D  K  S  T  S  E  P  R   344
aacttccatttcttgcctctggattcagatcctttgtatgtgaaacgaaaagtggccaag
 N  F  H  F  L  P  L  D  S  D  P  L  Y  V  K  R  K  V  A  K   364
ttggggaacagcagcaatgcggacaaattggtgaagtttgtcgctgagaatgttggtcct
 L  G  N  S  S  N  A  D  K  L  V  K  F  V  A  E  N  V  G  P   384
ggagtttatggggacgtagctatgtcgggagctggagctgtgcagcgacctgttataatc
 G  V  Y  G  D  V  A  M  S  G  A  G  A  V  Q  R  P  V  I  I   404
gcaaatcgaacaaaattcggtgatagaataaagaaggaacctacagatataggctcccct
```