Serine/arginine (SR)-rich protein B52 is involved in Dscam alternative splicing in *Litopenaeus vannamei*

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Alternative splicing (AS) is the essential process of maturation in precursor messenger RNA (pre-mRNA). AS can generate two to several thousands of mRNA isoforms from a single gene. Many splicing regulators are involved in AS. These include splicing activators and splicing repressors. One of the well-known splicing activators is the serine/arginine (SR)-rich protein family. In *Drosophila*, SR protein B52 regulates AS in several genes, including the Down syndrome cell adhesion molecule (Dscam). By using mutually exclusive alternative splicing, the arthropod Dscam isoform variability supports these proteins to function as putative antigen-specific receptors. In this study, the shrimp B52 gene from *Litopenaeus vannamei* (LvB52) was isolated and characterized. The open reading frame of LvB52 contains 1,149 bp encoding 382 amino acids. The deduced LvB52 protein includes two RNA recognition motifs (RRM) at the N terminus, and an arginine/serine rich domain (RS rich domain) at the C terminus. Based on domain architecture, LvB52 appears to be a functional SR protein homolog. Tissue tropism analysis revealed that LvB52 is expressed in most tissues, especially stomach and muscle. *in vivo* dsRNA silencing of LvB52 induced abnormal exon exclusion in the LvDscam cytoplasmic tail, but not in the Ig2-Ig3 region. An increase of element 3 skipping event was observed in the cytoplasmic tail of LvB52-silenced shrimp. We also found that abnormal exon exclusion of the Ig7 region occurred commonly, even in normal shrimp. After white spot syndrome virus infection, a significant increase in the expression of total LvDscam, tail-less LvDscam, membrane-bound LvDscam and LvB52 was observed after 24 hpi. Taken together, our data suggest that LvB52 acts as a splicing activator that regulates alternative splicing events in LvDscam.