



PRESS RELEASE (2017/03/16)

The regulation mechanism of the NF- κ B-like transcription factor Relish through the polyamine-modification catalyzed by transglutaminase

Drosophila has an immune signal pathway, called the immune deficiency (IMD) pathways. The final IMD pathway-dependent signal is transmitted through proteolytic conversion of the nuclear factor- κ B (NF- κ B)-like transcription factor Relish to the active N-terminal fragment Relish-N. Relish-N is then translocated from the cytosol into the nucleus for the expression of IMD-controlled genes. We previously demonstrated that transglutaminase (TG) suppresses the IMD pathway by polymerizing Relish-N to inhibit its nuclear translocation. On the other hand, we also demonstrated that orally ingested synthetic amines are TG-dependently incorporated into Relish-N. It remains unclear, however, whether polyamine-containing Relish-N retains transcriptional activity. Here we used mass spectrometry analysis and recombinant proteins, and show that the TG-modified Gln residues are located in the DNA-binding region of Relish-N. Moreover, *in vivo* experiments demonstrated that Relish-N was TG-dependently modified by polyamines, and that this modification reduced transcription of IMD pathway-controlled antimicrobial peptide genes. These findings suggest that TG regulates Relish-N-mediated transcriptional activity by incorporating polyamines into Relish-N and via protein-protein crosslinking. For more information about this research, see [Transglutaminase-catalyzed Incorporation of Polyamines Masks the DNA-binding Region of the Transcription Factor Relish](#).

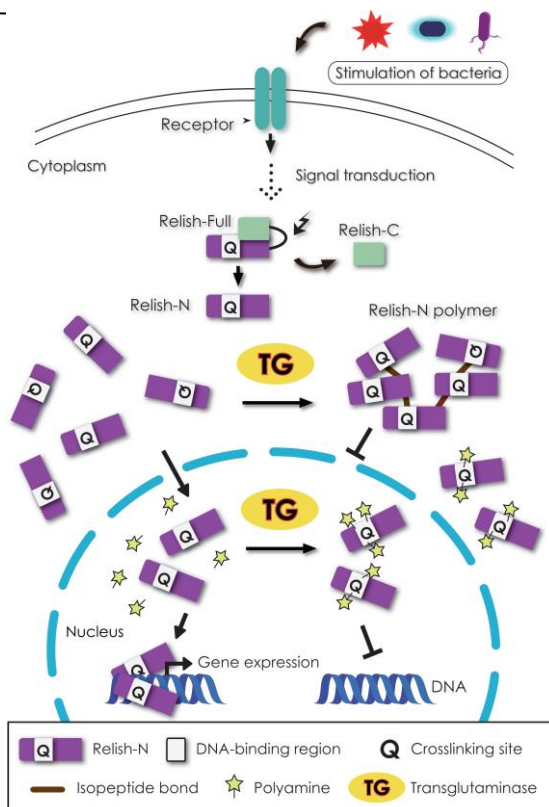


Figure: TG-catalyzed regulation of the IMD pathway in *Drosophila*

The IMD pathway is driven by stimulation of bacteria. The pathway controls the production of antimicrobial peptides by the activation of the nuclear factor- κ B (NF- κ B)-like transcription factor Relish. Relish is endo-proteolytically activated by a protease to be converted to the N-terminal fragment of Relish (Relish-N) containing the DNA-binding region with transcriptional activity and the C-terminal fragment (Relish-C). The resulting Relish-N is translocated into a nucleus, and functions as a transcription factor. TG regulates Relish-N-mediated transcriptional activity by incorporating polyamines into specific Gln (Q) residues of Relish-N and via protein-protein crosslinking of Relish-N molecules.

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