Abstract

The adaptor protein STING plays an essential role in innate immune system signalling. This transmembrane protein is anchored in the membrane of the endoplasmic reticulum. The STING protein is expressed in the cells of the immune system, in epithelial cells and pancreatic cells. The STING protein is activated by binding to the cyclic dinucleotides which induce the polymer formation. Then, the STING protein translocates to the Golgi apparatus, where it recruits the kinase TBK1 which phosphorylates the STING protein. Subsequently the protein recruits the transcription factor IRF3 that is also phosphorylated by TBK1. Phosphorylated IRF3 detaches from the complex, forms a dimer and translocates to the nucleus where it initiates the transcription of interferon genes. After signalling the STING protein is degraded via the autophagic pathway, thus terminating its signalization. Viruses, as intracellular parasites, need to modulate the signalling pathways of innate immunity for their effective multiplication within the cell. Therefore, the STING protein can serve as a target for certain viral proteins that inhibit STING-mediated signalling, allowing the viruses to evade the immune system.

Key words: STING, interferon, autophagy, virus, innate immunity