**Virus-Host Interactions: Innate immunity of DHX9, a nuclear RNA helicase and Viral Immune Evasion**

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DExD/H-box RNA helicases are enzymes that unwind duplex nucleic acids in an ATP-dependent manner and primarily involved in all facets of RNA metabolism. Multiple DExD/H-box RNA helicases also play important roles in innate immunity. DExD/H-box helicase 9 (DHX9), or RNA helicase A (RHA), is an abundant multifunctional nuclear protein. While DHX9 facilitates replication of many viruses including HIV-1, HCV, and FDMV, recent studies suggest that DHX9 may act as a cytosolic DNA and RNA sensor in innate immune cells. In this seminar, I will show you a detailed mechanism of DHX9 in the context of DNA virus infection. Macrophage-specific knockout and fibroblast-specific knockdown of DHX9 impaired antiviral innate immunity against DNA viruses. DNA virus infection did not induce the cytoplasmic translocation of nuclear DHX9. DHX9 enhanced NF-кB-mediated transactivation in the nucleus, which required its ATPase/helicase domain, but not the DNA-sensing domain. Nuclear DHX9 was essential for the recruitment of RNAPII rather than NF-кB/p65, to the promoters containing NF-κB binding sites in the chromatin level. Our results show a DNA-sensing independent role of nuclear DHX9 as a transcription coactivator in stimulation of NF-кB-mediated innate immunity against DNA virus infection. In addition, I will further discuss how the virus may counteract DHX9-dependent NF-кB transactivation.