Distinct Immune Escape Mechanisms of Bovine Cronavirus Nucleocapsid by Suppressing beta Interferon Production via Retinoic Acid-inducible Gene I-like Receptor Pathway

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Abstract

The present study aimed to explore if bovine coronavirus nucleocapsid (BCoV N) impacts beta interferon (IFN-β) production in the host cells and to reveal further molecular mechanism of BCoV pathopoiesis. Human embryonic kidney (HEK) 293T cells were transientlly transfected with pCMV-Myc-BCoV-N recombinant plasmids, then infected with the vesicular stomatitis virus (VSV). Expression levels of IFN-β mRNA were detected using qPCR. The results determinated that pCMV-Myc-BCoV-N recombinant plasmids of 1347bp was successfully constructed and transcribed into HEK 293T cells. Western-blotting assay indicated that BCoV-N recombinant plasmids had excellent antigenicity. BCoV-N recombinant proteins inhibited dose-dependently IFN-β production mediated by Vesicular stomatitis virus (VSV) ($P<0.01$). Furthermore, MDA5, MAVS, TBK1 and IRF3 could promote transcription levels of IFN-β mRNA. But, BCoV-N proteins demoted IFN-β levels induced by MDA5, MAVS, TBK1 and IRF3. Expression levels of MDA5, MAVS, TBK1 and IRF3 mRNAs were reduced in retinoic acid-inducible gene I-like receptor (RLR) pathway. In conclusion, BCoV-N reduced IFN-β levels in RLR pathway of HEK 293T cells. BCoV-N protein inhibited IFN-β production and activation of RLRs signal pathway. Our findings demonstrated a new mechanism evolved by BCoV to inhibit type I IFN production and provided a solid scientific basis for revealing the pathogenesis of BCoV, which is beneficial for developing novel strategy of the diagnose and therapy of BCoV disease.

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