

Immune Escape of Bovine Parvovirus by VP1 Inhibiting IFN- β Production through RIG-I like Receptors Pathway

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Abstract

Objective The present study aimed to explore if bovine parvovirus (BPV) impacts beta interferon (IFN- β) production and to reveal further molecular mechanism of BPV immune escape. **Method** The pCMV-Myc-BPV-VP1 recombinant plasmid was verified with both double enzyme digestion and sequence. HEK 293T cells were transfected with this recombinant protein, then infected with the vesicular stomatitis virus (VSV). Expression levels of IFN- β mRNA were detected using qPCR. Results Expression level of BPV VP1 mRNA in pCMV-Myc-BPV-VP1 group was significantly higher than those of the untreated group (UT) and pCMV-Myc vector group. BPV virus copies in bovine turbinate (BT) cells of BPV-VP1 group were raised ($P < 0.05$) with an increment of 5.8×10^4 . Expression levels of IFN- β mRNA of BPV VP1 group in HEK 293T cells were decreased ($P < 0.01$). Following treatment of TBK1 and IRF3(5D), IFN- β expression levels in HEK 293T cells were depressed. Expression levels of TBK1, IRF3(5D), MDA5 and MAVS were lower than those of their self treatment. **Conclusion** pCMV-Myc-BPV-VP1 could heighten transcription levels of VP1 protein in BT cells, promote BPV proliferation and ascend the production of IFN- β . Overexpression of pCMV-Myc-BPV-VP decreased IFN- β mRNA expression in HEK 293T cells and inhibited IFN- β production induced by TBK1 and IRF3(5D). Furthermore, BPV VP1 obviously declined expression levels of TBK1, IRF3(5D), MDA5 and MAVS in RLR pathway. Our findings revealed a new mechanism evolved by BPV VP1 to inhibit type I IFN production and provided a solid basis into the immunosuppression of BPV, which is beneficial for developing novel strategy of therapy of BPV disease.

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Table 1. Premiers for BPV VP1 amplification.doc available at <https://authorea.com/users/498881/articles/579617-immune-escape-of-bovine-parvovirus-by-vp1-inhibiting-ifn-%CE%B2-production-through-rig-i-like-receptors-pathway>



