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Yin Yang-1 (YY1), a transcription factor that is part of the polycomb group protein (PcG) family, is a direct target of miR218 in human glioma cells. It was demonstrated that YY1 promoted glioma cell proliferation and miR-218 could inhibit glioma cell proliferation by targeting YY1, and indirectly reduced the degradation of p53. Together the results indicate that miR-218 functions as a tumor suppressor in human glioma and suggest that overexpression of miR-218 may be a potential strategy for the treatment of human glioma in the future ¹⁾

Although transcription factor Yin Yang 1 (YY1) has been reported to have a dual activator/repressor role during the regulation of interferon beta (IFN- β) promoter activity, the roles of YY1 in the regulation of upstream signaling pathways leading to IFN-1 induction and IFN-1 signaling during viral infection remain to be elucidated. METHODS:

The roles of YY1 in IFN-1 production and IFN-1 signaling were investigated using immunoblotting, real-time PCR, small interfering RNA (siRNA)-mediated YY1 knockdown, YY1 overexpression by transient transfection, and co-immunoprecipitation, using mouse cells. RESULTS:

YY1 was shown to interact with STAT1 in the absence of viral infection. Following viral infection, YY1 protein expression levels were decreased. YY1 knockdown led to a considerable downregulation of phosphorylated (p) TBK1 and pIRF3 expressions, while YY1 overexpression significantly upregulated pTBK1 and pIRF3 expression levels and promoted virus-induced IFN-β production. Additionally, YY1 knockdown led to a significant upregulation of pSTAT1, pSTAT2 and antiviral interferon-stimulated genes, and inhibited viral replication. CONCLUSION:

We demonstrated here that YY1 interacts with STAT1 and dynamically regulates the induction of IFN-1 production and activation of IFN-1 signaling in different stages during viral infection ²⁾.

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