



## **A New Pathway for Cellular Antiviral Response**

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Upon infection with many RNA viruses, the cytoplasmic RIG-I pathway activates the latent transcription factor IRF-3 causing its nuclear translocation and the induction of many antiviral genes including those encoding interferons. In addition to transcription of antiviral genes, IRF-3 activated by RIG-I signaling pathway can efficiently trigger cellular apoptosis program. Using appropriate mutants of IRF-3, we demonstrated that the apoptotic activity was independent of its transcriptional activity. Apoptosis was triggered by the interaction of IRF-3 with the pro-apoptotic protein Bax, their co-translocation to the mitochondria and the resulting activation of the mitochondrial apoptotic pathway. Our current studies further uncover that members of DNA viruses can activate a similar apoptotic response via IRF-3 and Bax. Selective ablation of the IRF-3/Bax mediated apoptotic pathway significantly augmented virus replication and pathogenesis, demonstrating the importance of apoptosis in antiviral response. The presentation will highlight how this newly discovered IRF-3/Bax mediated apoptotic pathway significantly contributes to the host's overall protection against virus infection.