Limin Chen, Virol Mycol 2018, Volume 7
DOI: 10.4172/2161-0517-C1-022

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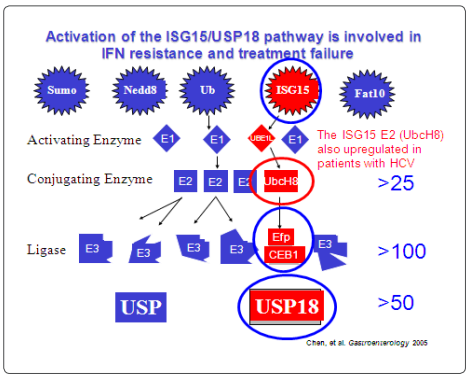
Activation of the ubiquitin-like ISG15/USP18 signaling pathway contributes to interferon resistance of HCV and HBV



Activation of the type-I Interferon (IFN) signaling pathway poses the first line of defense against many virus infections, including HCV and HBV. Our previous work identified, using high throughput gene expression profiling, 18 differentially-expressed hepatic genes between treatment responders (Rs) and Non-Responders (NRs) to IFN treatment of patients chronically infected with HCV. Three out of these 18 genes are involved in the same ubiquitin-like ISG15/USP18 signaling pathway, with higher expression levels in the pre-treatment liver tissues of NRs, indicating that activation of the ISG15/USP18 signaling contributes to treatment non-response leading to persistent infection. Similar findings were observed in chronic HBV infection. Mechanistically, some of these ISGs, such as ISG15 and ISG16 stimulated HCV replication and blunted IFN anti-HCV activity. All these data point out that type-I IFN signaling is a "double-edged" sword, while activation of this pathway is indeed necessary to control viral spread, over-activation of this pathway leading to the activation of the ISG15/USP18 signaling actually benefits virus to facilitate its persistent infection.

Biography

Limin Chen is the Affiliate Scientist with the University of Toronto and also a Professor with the Chinese Academy of Medical Sciences and Peking Union Medical College. Currently he is the Director and Chief Scientific Officer of the Center for Transfusion Transmitted Diseases, Institute of Blood Transfusion, CAMS/PUMC, Member of the American Association for Studies of Liver Diseases and Canadian Association for Studies of Liver. He has obtained his MD, MSc in Biochemistry and Molecular Biology in China, PhD in Molecular Genetics at the University of Toronto. He has obtained his Postdoctoral training both at the Merck Research Laboratories and at the Harvard Medical School.

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