Gene-viral interactions in hepatitis C; A critical role for IFN-λ3 gene variation

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Hepatitis C virus (HCV) is one of the most endemic viral pathogen that currently infects more than 170 million people worldwide. HCV causes acute and chronic hepatitis that progress to liver cirrhosis or hepatocellular carcinoma. HCV patients have two possible outcomes of infection, clearance or persistent infection. Approximately 20 % of primary HCV infected-people successfully clear the HCV, while the remaining 80 % of cases fail to do so. The interaction between host and HCV viral components ultimately determines the balance between the virus and host. Host factors, such as genetic background or immune responsiveness, dictate the distinct outcomes. Viral infection of a cell activates a panel of pattern recognition receptors that mediate antiviral innate and adaptive host immune responses to inhibit viral replication and dissemination. However, HCV utilizes multifaceted strategies to evade or subvert the host immune responses.

HLA haplotypes such as HLA-A03, -B27, -B57 and HLA-DRB1*0101, -DRB1*0401, -DRB1*1101, and -DQB1*0301 and Interleukin-28B, also known IFN-λ3, gene variation have been reported to influence HCV eradication either therapeutically or spontaneously. Human mDCs type 2, also known $BDCA3^{+}$DCs, are the potent producers of IFN-λ3 in HCV infection, suggesting the possibility that these cells could play a key role in developing therapeutic HCV vaccine. This work aims to review current state of researches that focused on the interaction of Viral-host immune system.