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Investigation of therapeutic potential of Cytokine IL-33 in hepatitis

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A new member cytokine IL-33 has recently joined the family of IL-1 because of its 11th number in the family it is also nominated as IL-1F11. In human and mice, main source of IL-33 is liver fibrotic cells and Hepatic stellate cells (HSC) when are in their activated form. To explore the functional role of IL-33 in viral related liver pathology, murine model of hepatitis was developed by injecting Poly I:C and hepatoprotective function of IL-33 was by administration of pre-treatment of mice with recombinant IL-33 (rIL-33). The poly I:C represents a relevant viral hepatitis model in human, because poly I:C is a virus-related dsRNA mimetic which plays role in increasing the IL-33 level in fulminant hepatitis. The poly I:C activates the NK cells in liver that leads to induction of inflammation. The present proposal was to decode the hepatoprotective role of IL-33 and underlying mechanism in viral dsRNA mimetic and poly I:C mediated acute liver diseases i.e. Hepatitis in murine model. Serum biochemical parameters like dosage of aspartate aminotransferase (AST), alanine aminotransferase (ALT) was carried out by diagnostic kit in biochemistry auto-analyzer that displayed in higher amount in those mice that were challenged with Poly I:C and their level was observed lower in post-treated rIL-33 group after Poly I:C administration and the quantitative measurement of serum INF-γ and TNF-α was performed using Albcam's INF-γ and TNF-α mice ELISA kits results of both these pro-inflammatory cytokines were same like ALT/AST. Level of these cytokines was also higher in Poly I:C challenged group and lower in post-treated rIL-33 group. These results confirmed the therapeutic effect of IL-33 in hepatitis or liver related diseases. The results were statistically analyzed by T test and one way ANOVA that proven the level of liver biomarkers and pro-inflammatory were significantly differ in rIL-33 treated mice trials.

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