

IL25 and IL23 on Diabetic Mice

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ABSTRACT

BACKGROUND

Type1 diabetes (T1D) is a complex chronic autoimmune inflammatory disease in which induced cytokine production creates dynamic environments for evolution and activity of effector as well as regulatory cells. Low-dose IL-2 has been applied for treatment recent-onset T1D in NOD mice. Different IL-2 formulations, for instance nanoparticle encapsulation, have been implicated to improve the therapeutic utility of low dose IL-2 to achieve precise suppression of pathological immune responses.

OBJECTIVES:

So, the current study intended to evaluate the effect of ultra low dose rhIL-2 therapy on the inflammatory process in multiple low dose streptozotocin (MLD STZ) induced T1D as well as the influence of chitosan nanosphere encapsulation on the proposed effect.

METHODS:

Male inbred Balb/c mice were divided into 5 groups; Normal (Normal mice), T1D: (T1D induced by MLD-STZ), T1D (Nano): T1D mice treated with free CS-TPP-NPs, T1D (rIL-2): T1D

mice treated with free rhIL-2, and T1D (rIL-2/Nano): T1D mice treated with rhIL-2 encapsulated into CS-TPP-NPs. Levels of IL-23 and IL-25.

RESULTS:

showed that both rhIL-2 and rhIL-2/CSNPs significantly decreased IL-23 as compared to corresponding group in T1D with maximum decrease at day 21 post treatment. Both rhIL-2 and rhIL-2/CSNPs significantly decreased IL-25 at days 7 and 14. All through the three time intervals, IL-25 remained significantly higher in rhIL-2/CSNPs than in rhIL-2.

CONCLUSIONS:

Ultra-low dose rhIL-2 ameliorated the diabetic state at day 7. rhIL-2/CSNP is more prone to induce an anti-inflammatory state. The anti-diabetic effect of rhIL-2 or rhIL-2/CS-NPs is not directly dependent on alterations in IL-23 and IL-25 production.

KEYWORDS:

Interleukin-2, Interleukin-23, Interleukin-25, Type1 diabetes.

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