Increased level of type I Interferon (IFN) during type I diabetes (T1D) induces apoptosis in spleen-homing T cells

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Abstract:

Type I diabetes (TID) is an autoimmune disease characterized by abnormalities in the defense mechanisms against a variety of infectious agents. Susceptibility to infections occurring in diabetic individuals is attributed to the decrease in the number of lymphocytes, which is probably a clinical consequence of the occurrence of apoptosis described in diabetes. TID is associated with increased cytokines that dampen lymphocytes proliferation, functions and subsequently increase risk to infection. Previous studies have reported an increase in IFN-α level which is associated with TID pathogenesis. In the present study, we further investigated the effect of blocking type I IFN receptor signaling pathway on the lymphocyte proliferation and functions within spleen as a secondary lymphoid organ in a streptozotocin (STZ)-induced type I diabetic mouse model. Three groups of mice were used (10 mice in each group): group 1, control non-diabetic mice; group 2, diabetic mice; and group 3, diabetic mice intraperitoneal injected with anti-IFNAR1 (10 mg/kg body weight once/day for up to 20 days). We found that diabetic mice exhibited increase in the apoptosis, DNA fragmentation, chromatin condensation and cell shrinkage; prolonged elevation in IFN-α and TNF-α levels and obvious reduction in spleen-homing T lymphocytes as compared to control mice. Interestingly, blocking type I IFN receptor of diabetic mice significantly decreased (P

Keywords:

Diabetes mellitus, IFN-α, apoptosis, lymphocytes, spleen.

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