Polyacetylenic compound and butanol fraction from Bidens pilosa can modulate the differentiation of helper T cells and prevent autoimmune diabetes in non-obese diabetes mice

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Abstract

Compelling evidence suggests that infiltrating CD4+ type I helper T (Th1) cells in the pancreatic islets play a pivotal role in the progression of diabetes in non-obese diabetic (NOD) mice. We demonstrate in the present report that a butanol fractionof B. pilosa suppressed the development of diabetes, helped maintain levels of blood sugar and insulin in NOD mice in a dose-dependent manner and elevated the serum IgE levels regulated by Th2 cytokines in NOD mice. Moreover, the butanol fraction inhibited the differentiation of naive helper T (Th0) cells into Th1 cells but enhanced their transition into type II helper T (Th2) cells using an in vitro T cell differentiation assay.

Two polyacetylenic compounds, 2-beta-D-glucopyranosyloxy-1-hydroxy-5(E)-tridecene-7,9,11-triyne and 3-beta-D-glucopyranosyloxy-1-hydroxy-6(E)-tetradecene-8,10,12-triyne, identified from the butanol fraction also prevented the onset of diabetes like the butanol fraction. The latter compound showed a stronger activity for T cell differentiation than the former. In summary, the butanol fraction of B. pilosa and its polyacetylenes can prevent diabetes plausibly via suppressing the differentiation of Th0 cells into Th1 cells and promoting that of Th0 cells into Th2 cells.