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Immunological and protective activities of Gamguntang (GGT) on experimental thyroiditis model

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Abstract

A variety of animal models for experimental autoimmune thyroiditis (EAT) exist which may arise as a result of different mechanisms. One model extensively employed involves the immunization of susceptible mice (H-2 k) with autologous thyroglobulin in complete Freunds adjuvant (CFA) or lipopolysaccharide (LPS). In this model, mononuclear cells consisting of both CD4+ and CD8+ T cells, together with macrophages and a few B cells, infiltrate the gland. There has been much speculation on the relative contribution of T helper type 1 (Th1) and Th2 subsets in this model and there is some evidence that the EAT induced in normal CBA/J (I-Ak) is characteristic of a Th1 response. However, It was also reported that the Th2 subset may be involved, as in their model of granulomatous EAT, inhibiting the Th1 subset with anti-interferon-γ (IFN-γ) antibody did not prevent thyroid infiltration. The crude herbal formulation, GGT

is an immunomodulator showing marked down-regulation of several experimental autoimmune diseases. In this study, its effect on different experimental models of thyroid disease was investigated. Although very effective at preventing thyroid infiltrates in mice immunized with mouse deglycosylated thyroglobulin and complete Freunds adjuvant and in spontaneous models of thyroiditis, it completely failed to modify experimental autoimmune thyroiditis (EAT) induced in mice immunized with mouse thyroglobulin and lipopolysaccharide. There was no significant shift in the observed isotypes of anti-mouse thyroglobulin antibodies and only anti-mouse thyroglobulin antibodies in the spontaneous model were completely down-modulated by the GGT. One surprising fact to emerge was that GGT-treated donor mice, although protected from thyroid lesions themselves, were still able to transfer EAT showing that they must have been effectively primed while being treated with GGT. It is possible that the drug down modulated EAT by interfering with the trafficking of primed effector cells.