

ABSTRACT

THESIS: The effects of the aqueous extract of *Lonicera japonica* on antigen-stimulated T cell function

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PAGES: 40

Lonicera japonica is a honeysuckle species commonly used in traditional Chinese medicine to treat diverse ailments such as headache, fever, cough and sore throat. The aqueous fraction of *L. japonica* has been shown to have a variety of anti-inflammatory properties in addition to *L. japonica* being hailed as an immune enhancer. Previous studies examining flavonoids, common constituents of *L. japonica* extracts, have demonstrated that flavonoids have varying abilities to inhibit lymphocyte functions. The goal of these studies was to examine the effect of the aqueous fraction of *L. japonica* on T cell functions. Purified murine T cells were assessed for proliferation using an MTT assay following stimulation with plate bound anti-CD3 and soluble anti-CD28 antibodies and/or treatment with *L. japonica*. The expression of CD25, CD95, CD152, and CD178 as well as cell death using propidium iodide staining were analyzed on CD4⁺ T cells using flow cytometry. An ELISA was used for assessing IL-2 production. Neutralization assays were performed using α CD178 or α CD152 antibodies in determining alterations in T cell death or proliferation. *L. japonica* inhibited T cell proliferation and reduced CD25 expression on activated T cells, while having no effect on IL-2 production. In naïve and

activated CD4⁺ T cells, *L. japonica* increased the expression of CD95, CD152, and CD178 in addition to cell death. Neutralization of CD178 or CD152 did not abrogate increased cell death or reduced proliferation resulting from *L. japonica* treatment. Our results showed that the aqueous extract of *L. japonica* is inhibitory to T lymphocyte proliferation and potentially occurs through decreased expression of CD25. In addition, *L. japonica* increased cell death which was independent of upregulated CD95 and CD178 expression. Findings from this work provide insights into the immunomodulatory properties of *L. japonica* and subsequent effects on T cell responses.