EFFECTS OF THE STEROID DIGOXIN ON B LYMPHOCYTES, ANTIBODY PRODUCTION AND ANTI-INFLAMMATORY PROFILE IN A MURINE MODEL

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**Introduction:** Digoxin is a cardiotonic glycoside that inhibits the Na+/K+ ATPase pump. Beyond its well-known cardiovascular effects, Digoxin also modulates the immune system, though its role in immunoregulation, particularly of B lymphocytes, remains underexplored. Understanding Digoxin~~'s~~ effects on B lymphocytes is relevant as these cells are fundamental to the humoral adaptive response. To date, no study has investigated the impact of Digoxin on B lymphocyte regulation. **Objectives:** This study aims to investigate the effects of Digoxin *in vivo* and *in vitro* on B lymphocyte homeostasis in central and peripheral lymphoid organs and the anti-inflammatory effects in Balb/c and C57BL/6 mice. **Material and Methods:** Balb/c and C57BL/6 mice received intraperitoneal injections of 0.3125 mg/Kg Digoxin elixir (equivalent to clinical doses) or RPMI medium alone (control group) for three consecutive days. Twenty-four hours post-injection, mice were euthanized, and organs and peripheral blood were collected. Cytokine secretion from lymphocytes stimulated *in vitro* and treated or not with Digoxin was measured using immunoenzymatic assay. **Results:** In Balb/c mice treated with Digoxin, there was an increase in B lymphocytes in the spleen and mesenteric lymph node, different from that observed in C57BL/6 mice in which there was a decrease in this population. This increase in B lymphocytes in Balb/c mice was not attributed to enhanced proliferation, viability, activation status or increased production in the bone marrow. Additionally, Digoxin administration *in vivo* elevated serum levels of total immunoglobulins M and G. *In vitro*, Digoxin reduced the secretion of pro-inflammatory cytokines IL-17, IL-6, and IL-2. **Conclusions:** Digoxin treatment increased the humoral response in Balb/c mice and reduced inflammatory cytokines, suggesting its potential use as a drug against chronic inflammation.