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Aryl Hydrocarbon Receptor Activation Enhances Total IgA Secretion in Leishmania Major-Infected Mice

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Secretory IgA (antibody A) plays a critical role in neutralizing microbial toxins, regulating non-pathogenic gut microbe populations, and protecting against infections at mucous membranes, like the airways or gut. Altered IgA levels have been associated with increased risk of allergies, infection, and autoimmunity. The aryl hydrocarbon receptor (AhR) is a regulator of immune function, including IgA levels. Chemicals in food or the environment can affect IgA levels, and this has been proposed as a risk factor for human disease. In mice, activation of the AhR with a single dose of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), an environmental pollutant, has been shown to alter fecal IgA levels. The goal of this study was to investigate a mechanism to explain altered fecal IgA levels after TCDD exposure. Female C57Bl/6 mice were administered TCDD orally at 0 or 40 μg/Kg of body weight one day prior to subcutaneous infection with one million Leishmania major promastigotes. Three weeks later, antibody levels were measured in serum and fecal extract by ELISA. TCDD exposure did not significantly change total IgA, IgM, or IgG1 levels in serum nor did it significantly change L. major-specific IgA levels in serum or fecal extract. However, relative to control animals, TCDD exposure significantly increased total fecal IgA levels by 61% ($P = 0.013$). Conversely, the level of mRNA for IgA heavy chain (measured by RT-RT-PCR) was significantly reduced by 54% ($P = 0.006$). These results suggest that AhR activation enhances total IgA secretion in the gut but does not enhance antigen-specific IgA responses following pathogen challenge. One explanation for these results could be relocation of B cells from the spleen, bone marrow, and other tissues to the gut. These findings suggest a potential mechanism by which environmental chemical exposures can dysregulate immunity to the detriment of human health.