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***Tryptophan Metabolites and Their Role in IL10-R1 Expression***

Within the gut lives a diverse microbial flora. Intestinal epithelial cells (IEC) need to maintain a functional barrier in order to protect the body from this antigenic luminal environment. The immune system's inflammatory response to invasion of antigens in the IEC layer can lead to disruption and damage to the mucosal lining. This compromises the barrier function of the epithelium, resulting in an increase of bacterial diffusion across the intestinal epithelia as well as an increase in inflammation. Regulatory T cells (Treg) can mediate the pro-inflammatory response through the signaling of the cytokine interleukin-10. IL-10 and its receptor IL-10R1 play key roles in suppressing inflammation resulting from a disruption of intestinal epithelial homeostasis and epithelial barrier. Recent research has shown the activation of the aryl hydrocarbon pathway induces IL-10R1 IECs. There is currently a gap in our knowledge regarding the specific tryptophan metabolites implicated in the upregulation IL10-R1 in intestinal epithelial cells via the aryl hydrocarbon receptor pathway. Based on previous findings, we hypothesize that specific tryptophan metabolites including indole-3-carboxaldehyde (IAI), indole-3-propionate (IPr) and indole-3-acetic acid (IAa) upregulate IL-10R expression through the AhR pathway in intestinal epithelial cells.

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