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Treatment with Interleukin-22 (IL-22) Protects Against Necrotizing Enterocolitis (NEC) by Enhancing Mucosal Healing

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Necrotizing enterocolitis (NEC) is the leading cause of death from gastrointestinal disease in premature infants. NEC is characterized by an exaggerated pro-inflammatory response, gut barrier dysfunction and impaired mucosal healing. Interleukin-22 (IL-22) has been shown to provide gut barrier maintenance and attenuate intestinal inflammation in adult animal models. Therefore, we hypothesized that IL-22 plays a role in protecting against NEC by reducing intestinal inflammation. We evaluated IL-22 as a treatment strategy *in vivo* using a NEC mouse model, which utilizes a combination of formula feedings and hypoxia. We used breastfed (BF) mice as a healthy negative control and formula-fed (FF) NEC mice as a sick positive control. Our results supported our hypothesis of the role of IL-22 in protecting against NEC. We found disruption of the small intestinal architecture in the histology of NEC FF compared to the BF controls. Importantly, in the NEC mice that received treatment with IL-22, we found restoration in gross appearance of the intestine and improvement in the intestinal architecture compared to NEC FF mice and they appeared similar to the BF controls. We further found that treatment with IL-22 increases the expression of CD4+ T helper cells, which suggests that these T helper cells are mediating protection against the intestine. Taken together, we conclude that IL-22 contributes to protection against NEC, which may be related to enhanced immune cell expression in the gut. This raises the possibility that treatment with IL-22 may protect premature infants from NEC.