

Employing Lymphoid Resident Commensal Bacteria to Modulate Chronic Inflammatory Diseases

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Background & Unmet Need

- The mammalian GI tract is colonized by a vibrant community of microorganisms, with roles in nutrient metabolism, resisting infection, and immune system development
- Translocation of commensal bacteria can lead to pro-inflammatory immune response and disease pathogenesis (e.g., IBD, metabolic disorders)
- **Unmet Need:** Improved understanding of translocation of commensal bacteria and strategies for modulating the host microbiome to treat chronic inflammatory diseases

Technology Overview

- **The Technology:** Lymphoid-tissue-resident commensal bacteria (LRCs) as key regulators of the pro-inflammatory response and potential biotherapeutic agents to treat chronic inflammatory disease
- LRCs colonize and persist within murine dendritic cells (DCs) and modulate cytokine production to curb inflammation
- The induced cytokines protected mice from lethal intestinal damage

Inventors: Inventors: Gregory Sonnenberg Nicholas Bessman

Patents: US Patent <u>10,596,206</u>

Publications: Fung et al. Immunity. 2016

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Cornell Reference: D-7744

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Technology Applications

- Treatment and prevention of inflammatory bowel disease (IBD) including Chron's Disease, Ulcerative Colitis, and IBS
- Treatment and prevention of other diseases mediated in part by the gut microbiome (e.g., diabetes, obesity, cancer)
- Consumer probiotic for maintenance of a healthy intestinal tract

Technology Advantages

- Colonization of LRCs may lead to long-term reduction of the pro-inflammatory response
- Co-administration of LRCs with IL-10 and IL-22 may enhance efficacy



Figure 1: Lymphoid-tissue-resident commensal bacteria (LRCs) regulate the host immune system through induction of IL-10 family cytokines

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