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Probiotics and innate signaling pathways contribute to protective responses in experimental allergic asthma

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Asthma is a chronic inflammatory disorder of the airways that is clinically characterized by recurrent airflow obstruction, wheezing and airway hyperresponsiveness (AHR). The disease is considered to be a Th2/Th17-driven disease associated with airway eosinophilia, and mucus metaplasia. We studied the capacity of probiotic bacteria to protect from experimental allergic asthma. Probiotic bacteria (*Bifidobacterium, Lactobacillium and Streptococcus*), administration prevents mice from experimental allergic asthma. In probiotic-treated mice, we observed a decreased AHR, eosinophilia in the bronchoalveolar fluid and impaired TH2 production of cytokine and chemokine levels in lungs. Mechanistic studies showed that the protection was associated with an increased frequency of $CD4^{+}CD25^{+}FoxP3^{+}T$ cells and was an IL-10-dependent effect since administration of an anti-IL-10 receptor antibody completely abolished the therapeutic effect.

Probiotics may function as a combinatorial signalling through several Toll-like receptors (TLRs). Interestingly, distinct TLR stimulation may also prevent allergic inflammation and AHR, since the contribution of dysregulated innate immune responses leading the pathogenesis of allergic disease was unknown, we examined the role of innate pathways in the pathogenesis of an airway inflammation by studying $MyD88^{-/-}$ mice that had concurrent deficiencies in TLR signaling pathways. Global deficiency of the common TLR adaptor MyD88 offered partial protection from allergic disease.

Taken together, these results suggest that probiotics have a key role at host-microbial interfaces in the prevention of human allergic disease in promoting immunoregulation. The potential role of the MyD88 signaling pathway, which may play a substantial role in the pathogenesis of human allergic disease, in the response to probiotics will be discussed.

Biography

Nathalie Thieblemont has completed her Ph.D in immunology from Paris 6 University, Paris in 1993 and postdoctoral studies from the Rockefeller University, NY and then moved to Merck Research Laboratories, NJ. She joined the National Center of Scientific Research, Paris as Principal Investigator in 2001. Her research emphasizes the role of the Toll-Like Receptor signaling pathway in immunity. She has published over than 30 papers in international peer-reviewed journals. By addressing the hygiene hypothesis, she started to investigate the role of TLR/MyD88 signaling pathway in responses to probiotics.

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