Review of ecology of the gut microbiota and human immunity

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Abstract

Coevolution of humans and the ecological system of trillions of symbiont gut microorganisms belonging to Bacteroidetes, Firmicutes and Archaea, has resulted in a mutually beneficial partnership. The human immune system tolerates the presence of gut microbiota and, in return, the microbiota has enhanced the fundamental functions of the immune system as evident by increased susceptibility of germ-free guinea pigs to *Shigella flexneri* infection and that of germ-free mice to *Listeria monocytogenes* and Salmonella ser. Typhimurium, which is readily reversed by reintroduction of specific commensal bacteria. Introduction of commensal bacteria has been found to increase the expression of the antibacterial protein, lectin that augments innate immunity. Germ-free mice are documented as neutropenic and their neutrophils demonstrate reduced nitric oxide generation, superoxide generation and phagocytic ability. Also germ-free mice have comparatively less intestinal antigen presenting cells, which was corrected by introduction of Escherichia coli. *Bactereoides fragilis* is known to produce a unique zwitterionic polysaccharide, which after processing by antigen presenting cells stimulates CD4 T helper cells. Intestinal CD8 T cells in germ-free mice are diminished in number and cytotoxicity, portraying that the gut microbiome is essential for the potency of the acquired immune system. Clostridia, through increased levels of butyrate, promotes increased expression of Treg cells that police acquired immune responses to prevent allergy and inflammation. Certain pathogens have developed mechanisms to destabilize the gut microbiome as means of gaining access to the human body. *Salmonella* Typhimurium induces inflammation, which leads to a disadvantageous change in the composition of the commensal microbiome and suppresses its growth, so that colonization resistance provided by the commensals is negated. In inflammatory bowel disease, the Firmicutes and Bacteroides species reduce in number, leading to an overgrowth of proteobacteria, suggesting that dysbiosis could be a causative factor in autoimmune diseases. Further research is required to improve our knowledge of the gut microbiome and its role in the immunity of Sri Lankans, because the number and the diversity of the gut microbiome depends greatly on ethnicity, geographical variations, dietary habits, health status and iatrogenic factors among other determinants.

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