The immune response to Prevotella bacteria in chronic inflammatory disease - DTU Orbit (28/04/2018)

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The microbiota plays a central role in human health and disease by shaping immune development, immune responses and metabolism, and by protecting from invading pathogens. Technical advances that allow comprehensive characterization of microbial communities by genetic sequencing have sparked the hunt for disease-modulating bacteria. Emerging studies in humans have linked the increased abundance of Prevotella species at mucosal sites to localized and systemic disease, including periodontitis, bacterial vaginosis, rheumatoid arthritis, metabolic disorders and low-grade systemic inflammation. Intriguingly, Prevotella abundance is reduced within the lung microbiota of patients with asthma and chronic obstructive pulmonary disease. Increased Prevotella abundance is associated with augmented T helper type 17 (Th17) -mediated mucosal inflammation, which is in line with the marked capacity of Prevotella in driving Th17 immune responses in vitro. Studies indicate that Prevotella predominantly activate Toll-like receptor 2, leading to production of Th17-polarizing cytokines by antigen-presenting cells, including interleukin-23 (IL-23) and IL-1. Furthermore, Prevotella stimulate epithelial cells to produce IL-8, IL-6 and CCL20, which can promote mucosal Th17 immune responses and neutrophil recruitment. Prevotella-mediated mucosal inflammation leads to systemic dissemination of inflammatory mediators, bacteria and bacterial products, which in turn may affect systemic disease outcomes. Studies in mice support a causal role of Prevotella as colonization experiments promote clinical and inflammatory features of human disease. When compared with strict commensal bacteria, Prevotella exhibit increased inflammatory properties, as demonstrated by augmented release of inflammatory mediators from immune cells and various stromal cells. These findings indicate that some Prevotella strains may be clinically important pathobionts that can participate in human disease by promoting chronic inflammation.
ISI indexed (2011): ISI indexed yes
Web of Science (2011): Indexed yes
BFI (2010): BFI-level 1
Scopus rating (2010): SJR 0.121 SNIP 0.897
Web of Science (2010): Indexed yes
BFI (2009): BFI-level 2
Scopus rating (2009): SJR 0.122 SNIP 0.928
Web of Science (2009): Indexed yes
BFI (2008): BFI-level 1
Scopus rating (2008): SJR 0.111 SNIP 0.921
Web of Science (2008): Indexed yes
Scopus rating (2007): SJR 0.122 SNIP 0.964
Web of Science (2007): Indexed yes
Scopus rating (2006): SJR 0.122 SNIP 0.887
Web of Science (2006): Indexed yes
Scopus rating (2005): SJR 1.64 SNIP 0.921
Web of Science (2005): Indexed yes
Scopus rating (2004): SJR 1.409 SNIP 0.793
Scopus rating (2003): SJR 1.329 SNIP 0.836
Scopus rating (2002): SJR 1.293 SNIP 0.789
Scopus rating (2001): SJR 0.121 SNIP 0.76
Scopus rating (2000): SJR 0.111 SNIP 0.836
Scopus rating (1999): SJR 0.107 SNIP 0.827

Original language: English
cytokines, inflammation, inflammatory disease, mucosa, Prevotella, T cells
DOIs:
10.1111/imm.12760
Source: FindIt
Source-ID: 2363398362
Publication: Research - peer-review › Journal article – Annual report year: 2017