SOFTWARE

BACTERIA, VITAMIN D AND ALLERGY

30.05.2016

Bacterial (and fungal) gut diversity are believed to influence primary allergic sensitization as well as early vitamin D supplementation. The question is – again – could there be any connection?

No doubt, there are numerous antagonistic mechanisms of the human host linking bacteria flora and allergen recognition/presentation. 1952: "Hay factor", 2004: LPS/D3 antagonism, 2005: D3 blocks DC maturation, 2007: DCs need a co-inflammatory signal, 2009: bacteria-induced vitamin D receptor dysfunction, 2010: Salmonella more aggressive with turned off vitamin D receptor…

But what happens when ingesting overdoses of vitamin D supplements? 500-1000 kU vitamin D is an about 100fold overdose compared to physiological intake of a newborn. AFAIK bacteria do not utilize vitamin D, so it is rather unlikely that there are direct host-independent effects.

Only recently I found a paper already published last year "Effects of high doses of vitamin D3 on mucosa-associated gut microbiome vary between regions of the human gastrointestinal tract". As a main outcome Bashir et al. observe a reduction in opportunistic pathogens and an increase in bacterial richness:

> Vitamin D3 supplementation changed the gut microbiome in the upper GI tract (gastric corpus, antrum, and duodenum). We found a decreased relative abundance of Gammaproteobacteria including Pseudomonas spp. and Escherichia/Shigella spp. and increased bacterial richness. No major changes occurred in the terminal ileum, appendiceal orifice, ascending colon, and sigmoid colon or in stools, but the CD8+ T cell fraction was signicantly increased in the terminal ileum.

Like the authors, I do not know of any similar study. The mechanism is rather unclear leading the authors to speculate

Reducing such an inflammatory environment by vitD3 could diminish the competitive advantage of opportunistic pathogens, such as Escherichia/Shigella spp. or Pseudomonas spp. which are evolutionary better adapted to inflammation and can outcompete commensal bacteria. In return, a low inflammatory environment allows beneficial bacteria such as Bacteroidetes to outcompete opportunistic pathogens, resulting in increased phylotype richness which we found in this study.

The only paper I remember from the literature is Cantorna et al 2013 cited but not evaluated by Bashir 2015:

Cyp KO and VDR KO mice had more bacteria from the Bacteroidetes and Proteobacteria phyla and fewer bacteria from the Firmicutes and Deferribacteres phyla in the feces compared with wild-type ... The mechanisms by which the dysbiosis occurs in VDR KO and Cyp KO mice included lower expression of E-cadherin on gut epithelial and immune cells and fewer tolerogenic dendritic cells that resulted in more gut inflammation in VDR and Cyp KO mice compared with wild-type mice.