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The Celiac Disease & the Microbial Flora

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A lifelong immune-mediated, multisystem disorder that develop in genetically predisposed individuals when exposed to dietary gluten, Characterized by elevated titers of celiac-specific autoantibodies, and inflammatory enteropathy with a wide range of gastrointestinal and extra-intestinal manifestations and problems (S. Guandalini, 2014).

The celiac individuals have abnormal microbial flora compared to healthy ones. There is dysbiosis of the microbial flora with villus atrophy and lymphocytes in the villous architecture.

(Chang F 2005)

The dysbiosis could be a cause and a result in the course of the disease.

The microbial dysbiosis is the imbalance of the microbial flora of the person concerned where bacteria with inflammatory cytokine overcome those with anti-inflammatory cytokines.

Reduction in *Bifidobacteria* species and/or *Lactobacillus* species relative to gram negative bacteria and pathobionts which have the potential to aggregate and maybe a predisposing factor in the course of the disease with their production of inflammatory cytokine especially after the ingestion of gliadin. The inflammatory cytokines include interferon gamma (IFN-γ), tumor necrosis factor alpha (TNF-α), and interleukin 12 (IL-12) (E. Leonetti., *et al*. 2011).

Studies mentioned that breast feeding may ameliorate the symptoms and delay it manifestation, by acting on the tight junctions and keep the microbial flora in balance. "The effect of the good bacteria, the bifidogenic nature of the breast milk".

Also, studies show that the alteration of the composition of the microbial flora of the celiac patient by the good flora produce anti-inflammatory cytokines which counter effect the inflammatory cytokines which are elicited by the pathogenic bacteria. (Di Cagno R., *et al*. 2011) (Nóra Judit Béres., *et al*. 2014). (K. Lindfors., *et al*. 2008).

So the question is Can designed probiotic or fecal transplant of a heathy patient change the course of cealiac disease and decrease or stop the intestinal and extra intestinal manifestation of the disease??? (Fasano Jose U., *et al*. 2016).

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