

The gut immune system, inflammatory bowel diseases, and the body immune homeostasis: modern treatment strategies

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ABSTRACT

The digestive tract is nowadays conceived as a barrier organ constituted by a mucosal membrane separating the gut lumen from the inner milieu. The gut lumen is laden by a myriad of antigens brought about by the diet, but also pertaining to the overwhelming bacterial species of the gut microbiome. The mucosal cell population comprehends epithelial cells, and a variety of immune reactive cells. Of them, the mononuclear types effecting innate responses are endowed by membrane signaling receptors and, as a rule, are sensing the polysaccharides of bacterial cell walls; non-tolerated signals may then push the chain reaction on, to end in full activation of inflammation mediators. Acquired immunity is in turn mainly effected by T-cell types, some of them, behaving as autoreactive cells, may induce metastatic inflammation beyond bowel boundaries,

partly explaining the so-called extra-intestinal manifestations of inflammatory bowel disease (IBD). The scenario is further complicated by the possible influence of epigenetic factors: diet, stress, smoking, drugs. Being IBD a low-penetrance disorder, for the full phenotype to develop, a critical mass of the above listed factors (typically, a disturbed membrane permeability, an immune stimulus, and an epigenetic factor) must occur. In the century since the full description of IBD, a variegated plethora of measures have been attempted. Some updated designs are now under scrutiny. Microbiota engineering, apoptosis modulation, and diet modification are just a few of the measures that we are arbitrarily describing here.

Key words: Inflammatory bowel disease, gut immunopathology, gut apoptosis, IBD modern treatment

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