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*Actis GC**

Gastroenterologist and Immuno-Rheumatologist Emeritus, The Medical Center Office and Turin General Hospitals, Corso Einaudi 18a, Turin, 10129, Italy

Abstract

The digestive tract, one of the body's barrier organs, integrates digestive and immune functions. A lingering down-graded inflammatory state witnesses the evolutionary capacity to express immunity safely in both defensive and regulatory roles. It may be forecasted that tomorrow's scientist shall be full-time busy in studying and translating the digestive functions in physiology and disease, to the point of obscuring the role of internal medicine specialties.

Keywords: Inflammatory Bowel Disease; Barrier Organs; Inflammation; Microbiome; Digestive Immunity

Background

The digestive tract as a barrier organ

One of the top achievements of modern science has been the reckoning of the existence in the human body of anatomical/functional entities named "barrier organs" [1]. For short, barrier organs are conduits or surfaces that mark the threshold between the outer world (thought of as an antigenic load) and the inner world (ideally a sterile domain). The threshold is supposed to be an epithelial or mucosal sheath lying on a highly reactive lymphoid tissue, which will mount a defensive inflammatory reaction against any threat to trespass the threshold [2]. A subtle control system downgrading the response will try to avoid devastating immune clashes at any time [2]. There are at least two other body barrier organs: the skin and the lungs. Notably, the latter exhibit an interesting interplay with the gut immune system: for example, antigens tolerated in the lung microenvironment, will be so also at the gut level [3]. From this preface, it seems clear that the main inherent function of barrier organs, chiefly the gut, is the

Handling of inflammation

a. Inflammation as an integration of feeding functions**:** The onset of some drowsiness after a meal pertains to lay people knowledge. Some authors have named it "post-prandial dip" [4] and recent research has shed interesting light on the underlying biochemical steps [5]. Briefly, an insulin response normally follows post-prandial hyperglycemia; contextually, a wave of lipopolysaccharide (originating from the ingested food load) raises omental macrophages, which, nourished by glucose availability, will release the pro-inflammatory IL-1 beta. This will target the insulin-producing cells which are rich of IL-1 beta receptors, and the overall inflammatory system level will be upgraded [5]. This chain of events tells then two messages: 1) feeding functions are tightly integrated with an inflammatory response; 2) insulin release itself is an inflammatory event directed by IL-1 beta, a classic pro-inflammatory mediator. Other implications, such as the relationship between feeding disorders and inflammation and cognitive decline in today's society [6] can easily be forecasted to become crucial soon, yet they extend beyond the present topic.

b. "Active" inflammation either gut indwelled (B1), or trespassing bowel borders (B2) (neurodegenerative disease complex).

B1. The mostly known and studied examples of indwelled digestive disorders are the Inflammatory Bowel Diseases (IBD), that comprise Crohn's Disease, an entity marked by the formation of granulomatous inflammatory patches potentially everywhere in the gut; and Ulcerative Colitis, a vasculitis-like lesion confined to the colonic mucosa [7]. The socio-economic and medical challenges posed by IBD in the century upon its description are the subject of a vast ever-increasing literature. Restricting the focus to the IBD forecast, a couple of aspects may be found to be worth of note. Firstly, many authors nowadays imagine the existence of an inflammatory continuum spanning from phenomena purely considered as functional (irritable bowel syndrome, for example), connecting ill-defined (organic?) disorders (microscopic colitis), all the way to the

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Correspondence:
Actis GC, Gastroenterologist and Immuno-Rheumatologist Emeritus, The Medical Center Office and Turin General Hospitals, Corso Einaudi 18a, Turin, 10129, Italy.
Tel: *0039-011-59 1388*
Fax: *0039-011-50 5564*
E-mail: *Actis_g@libero.it*
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severest IBD presentations, with inflammation intensity being the only difference factor [8]. Besides its cultural contents, this novel mind frame clearly purports issues of treatment positioning, with the underlying suggestion that, in an extreme top-down strategy [9], irritable bowel syndrome should receive immune suppressors to halt its progression from the very beginning.

B2. Recent developments are suggesting that inflammation originating in the upper GI tract may migrate cranially, creating a favorable background for the breaking out of neurodegenerative disease, including Parkinson's, Alzheimer, and Amyotrophic Lateral Sclerosis. For short, according to this hypothesis, common agents such as H. Pylori in susceptible individuals could induce the enteric nervous system (ENS) to produce the defensive prion-like protein alpha-synuclein; migrating cranially, this protein could come about to disturb the nervous conduction at the mid-brain level, resulting in full-blown Parkinson's in predisposed individuals [10]. In the near future, this knowledge can not be ignored to imply that devastating neurologic diseases plaguing our Western World could hopefully be limited by eradicating some gut infectious agents, and, trivially, by increasing people's oral hygiene [11].

c. Scientists of this century are maximally excited by the discovery of the microbiome, a population of some 10^{14} bacteria indwelling the colon [12]. On one side, the microbiome may respond to outside stimuli, and on the other side, its changes can incite inflammation from the local barrier organ system. Thus, the microbiome may act as a linking signal connecting the world outside with the inner body environment. Feeding changes (resulting from a huge factor list including food availability, cooking recipes, feeding time, income levels, single versus family percentages) are among the mostly effective forces to microbiome changes and inflammation [13]. The challenges we are facing now, in terms of automation in industry, unemployment increase, and migrations, all confer an undeniable social dimension to what cannot be considered a merely medical issue.

The above statements witness that feeding is just one of the gut functions, and the inherent presence of inflammation into gut physiology and disease confers to gut disorders the character of a generalized trans-organ (systemic) process.

It may be forecasted that such an expansion of gastroenterological disciplines will progressively question the survival of internal medicine as an autonomous specialty [14,15].

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