Irritable Bowel Syndrome: a psycho-somatic disease?

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Irritable Bowel Syndrome (IBS) is a clinical entity which associates abdominal pain or discomfort and transit disorders, in the absence of an otherwise recognisable anatomical/biochemical abnormality of the gut. The Rome Criteria form the basis for a positive diagnosis of IBS, and relies mainly on symptoms assessment and their duration. IBS is a highly prevalent disease, which accounts for up to half of specialised consultations in the gastro-intestinal arena. Except in rare settings, many more women than men consult for IBS.

For many decades, evidence for a patho-physiologic abnormality at the origin of IBS has been lacking. Hence, it is no surprise that the scientific debate on the origin of IBS has traditionally been centered around two opposite poles: one advocating the prevailing role of psychological disturbances (a "utility" hypothesis, whenever objectives measures cannot link a particular abnormality to the disease), the other one advocating the existence of motor/sensitive abnormalities at the origin of the disease. In this context, it is legitimate to hypothesise that the primary predisposing insult in IBS patients and perhaps also the mechanism for sustaining the condition, are located somewhere on the brain-gut axis.

Epidemiological studies have clearly shown that the presence of some psycho-social factors is much higher in IBS patients than in normals. However, some of these studies also suggest that such factors contribute more to the patients' behavior with regards to seeking treatment for IBS than to the disease itself. On the other hand, motility studies have failed to identify an anomaly which is typical of IBS; most of the "abnormal" intestinal motor patterns identified in IBS patients can be elicited under certain conditions in normals. More recently, repeated experiments comparing IBS patients and normals, have shown that IBS patients tended, as a group, to exhibit a lower threshold for pain elicited by the intraluminal distension of the gut at various levels, and has prompted the theory of hypersensitive gut. Interestingly, this hypersensitive state is not global, as attested by the normal or even increased somatic pain thresholds found in the same IBS patients. Thus, SmithKline Beecham's hypothesis is that the primary insult in IBS is represented by an anomaly specifically located in the gut wall at the 5-HT4 receptor level, driving the hypersensitivity and most of the gastro-intestinal symptomatology. Given the rich innervation of the gut, and its ability to interact with the central nervous system, one could easily conceive that such anomaly in the gut may also drive reactive effects at different levels of the CNS.