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Dealing with Our 'In-Vironment': New Aspects of Inflammatory Bowel Disease Pathogenesis and Therapy

Our understanding of the pathogenesis of inflammatory bowel disease (IBD) has dramatically changed during recent years. The focus of research has shifted from adaptive immunity to genetic risk factors and now to disturbances in innate immunity and the interactions of the mucosal immune system with the content of the gut – our 'in-vironment'. The in-vironment is a term introduced by Michael Mayerfeld Bell in his book 'An Invitation to Environmental Sociology'. He describes it as the 'human body, which is continuously interacting with the environment'. He points to the fact that eating and drinking are particular ways for humans to interact with their environment.

The groundbreaking insights gained in the last years indicate that the genetic factors only contribute 50% to the risk of developing IBD and that environmental factors may trigger or even cause the disease. These factors mediate their effects via uptake into the human body. They may change the composition of our microbiome

making it more proinflammatory or aggressive. Other factors may directly act on the intestinal barrier function.

Our therapeutic approaches still mainly focus on regulating adaptive immunity. Only recently have the barrier function of the gut mucosa and the defense function of the innate immune system come into focus. A recent FALK meeting entitled 'Dealing with our 'In-vironment': New Aspects in IBD Pathogenesis and Therapy' was, therefore, centered around recent discoveries that may change future treatment strategies. In a 2-day meeting, genetic risk factors in IBD, antibacterial defense mechanisms, the epithelial barrier as a border to the in-vironment, environmental factors in pathogenesis, disease markers, new therapeutic approaches and future therapeutic direction were discussed. The articles summarized here cover the most important aspects of the meeting and highlight the role of the in-vironment in IBD pathogenesis.

Gerhard Rogler, Zürich

Inflammatory bowel disease (IBD) is a chronic and life-long disease characterized by gastrointestinal tract inflammation. It is caused by the interplay of the host's genetic predisposition and immune responses, and various environmental factors. Despite many treatment options, there is no cure for IBD. Natural products have significant potential as therapeutic agents with an increasing role in health care. Given that natural products display great structural diversity and are relatively easy to modify chemically, they represent ideal scaffolds upon which to generate novel therapeutics. This review focuses on the pathology, currently available treatment options for IBD and associated challenges, and the roles played by natural products in health care. The chronic inflammatory bowel diseases (IBD), Crohn's disease (CD) and ulcerative colitis (UC), are recognized as important causes of gastrointestinal disease in children and adults. IBD occurs worldwide, although it is more common in some regions (United States, United Kingdom, and Scandinavia) than in others, with incidence rates of 4 to 10/100,000 persons per year and prevalence rates between 40 to 100/100,000 persons (49). IBD is most commonly diagnosed between the third and fourth decades of life, with no difference noted between males and females. Inflammatory Bowel Diseases (IBDs) are complex, multifactorial disorders characterized by chronic relapsing intestinal inflammation. The two major subtypes of IBD are Ulcerative Colitis (UC) and Crohn's Disease (CD). Ulcerative Colitis and CD are important worldwide health problems, with an incidence in Europe of 12.7 and 24.3 per 100,000 person-years, respectively, and prevalence of 0.5 and 1.0%. New advances in understanding IBD pathogenesis explain important disease mechanisms, including not only the innate and adaptive immunity, but also interactions between genetic, microbial, and environmental factors (4). Genetics and Pathogenesis. Over the past few decades, there have been important advances in our understanding of genetic contributions to IBD.