Oral microbiota, intestinal microbiota and inflammatory bowel disease

By Prof. Denis Bourgeois, France

**Intestinal microbiota**

There exists a close relationship between the human host and the intestinal microbiota—a mixed community of microorganisms that protect the intestine from being colonised by exogenous pathogens. In a healthy individual, the host and microbiota coexist in mutual harmony, allowing both to function properly. The balance of the intestinal microbial ecosystem can be disrupted by a number of factors, such as antibiotics, vaccinations, certain foods and stress. An intestinal bacterial disorder primarily manifests in terms of quantitative changes in bacterial location, causing excessive bacterial growth in the intestine. This can damage the intestinal mucosal barrier, thereby releasing enterotoxins as a means to increase intestinal epithelial permeability so that bacteria and products can enter the intestinal lamellae, causing an immune dysregulation of the mucous membranes and inducing inflammatory bowel disease (IBD). Changes in intestinal microbes are associated with the development of IBD.

IBD comprises a group of idiopathic diseases characterised by chronic inflammation of the bowel. This inflammation may affect any part of the gastrointestinal tract. IBD represents a group of two principal intestinal disorders: Crohn’s disease (CD) and ulcerative colitis. These two disorders have distinct clinical and pathological features, yet they do overlap.

The pathogenesis of CD is most notably associated with a deterioration of the immune system, which becomes incapable of destroying bacteria, viruses and other potentially harmful foreign organisms, as well as the intestinal microbiota. There is currently good evidence that the intestinal flora or microbiota plays a key role in the development of IBD. Recent studies have shown that certain strains of intestinal bacteria are responsible for ulceration and chronic inflammation in IBD. Ulcerative colitis, as opposed to what was initially believed, is not an autoimmune disease, but rather an infectious disease related to an imbalance in the intestinal microbiota.

According to He et al., the CD microbiota is grouped into two distinct meta-communities, which would indicate subject variation in the structure of the microbiome. Specific functional changes in the CD meta-community show increased levels of pro-inflammatory hexa-acylated lipopolysaccharides and a reduced potential to synthesise short-chain fatty acids. Moreover, disruption of ecological networks in CD is associated with reduced growth rates of many bacterial species. The authors concluded that the microbiota of CD patients can be layered into two distinct meta-communities, in which the most seriously disrupted meta-community exhibits functional potentials that substantially deviate from those of a healthy individual, with a possible implication for the pathogenesis of CD.

Various explanations have been advanced, such as the hygiene hypothesis, which blames the frequent use of antibiotics and microbicidal compounds; the partial elimination of enteric microflora after suffering from infectious acute gastroenteritis; certain food components, for example refined sugars used in developed countries, which could promote the growth of certain types of bacterial species; and even certain types of toothpaste.

**Oral microbiome**

Individuals’ oral microbiomes are highly specific at the species level, although overall, the human oral microbiome is largely homogenous. If the symbiotic balance between the host and the microbiota of the oral cavity is disrupted, the microbiota may become harmful. Distinctions in microbial composition have been found between carious and caries-free microbiomes, as well as periodontally diseased and periodontally healthy microbiomes. Although caries and periodontitis are clearly bacterial diseases, they are not infectious diseases in the classical sense, since they result from a number of factors: commensal microbiota, host susceptibility and environmental factors, such as diet and smoking.

The literature on interdental applied to carious lesions is extremely limited. However, it has been established that the effective presence of the red complex, particularly *Porphyromonas gingivalis*, a pathogen of heart disease and other systemic diseases, is a strong indicator of the need to develop new methods to disrupt interdental biofilm through daily oral hygiene. Indeed, it has been shown that low levels of *P. gingivalis* (< 0.01% of the total load) were able to induce changes in the composition of the biofilm. Likewise, the presence of *Candidas albicans* in significant quantities in the interdental spaces is cause
Understanding the interaction between the intestinal microbiota, pathogens and the human host could lead to new strategies, notably by modifying the composition of the intestinal microbiota.

*Helicobacter pylori*, a bacterium known to irritate the stomach lining and induce chronic gastritis, as well as poor periodontal health. This observation is supported by existing literature on the subject, which suggests that dental plaque may harbour *H. pylori* and cause recurrences of gastric infection.

A 2017 study by Hujoel and Lingström traced an overview of the historical role of nutrition in the development and prevention of dental caries, gingival bleeding and periodontal disease. Given how much recommendations on nutrition have changed over time—the World Health Organization has only since 2015 recommended the restriction of sugar intake, for example—it is interesting to see that the current evidence suggested a low-carbohydrate diet high in non-vegetable fats, micronutrients (e.g. vitamin C and B₁₂) and protein was correlated with periodontal health. However, the ability to absorb these nutrients can be influenced by gastrointestinal health. As the Canadian Society of Intestinal Research has reported, the improper functioning of the gastrointestinal tract can reduce nutrient absorption, leading to vitamin and mineral deficiencies that may cause oral lesions and tongue inflammation.

**Editorial note:** A list of references can be obtained from the publisher.