Colonic fermentation: a neglected topic in human physiology education

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The large bowel has seemingly never been very attractive (2). As stated by Sir Arthur Hurst (3a):

> No organ in the body is so misunderstood, so slandered and maltreated as the colon.

Human physiology textbooks tend to limit their discussion of colonic functions to those of absorbing water and electrolytes and storing waste material. However, the colon is a highly active metabolic organ, containing an exceedingly complex society of microbes (1).

By means of fermentation, gastrointestinal microbes break down nutrients that cannot be hydrolyzed by mammalian host enzymes and thus play an important role in digestive physiology, not only in plant-eating animals (herbivores) but also in humans (omnivores). A substantial amount of energy, otherwise lost in feces, is conserved by colonic salvage of calories, mainly derived from complex carbohydrates (“grass”). The microbial flora may consume enough “grass” to cover ~5--10% of our daily energy demand (5). In fact, energy obtained from microbial fermentation products may contribute to the development of obesity (9).

Colonic fermentation yields both gases (e.g., hydrogen, methane, and carbon dioxide) and short-chain fatty acids (SCFAs; e.g., acetic, propionic, and butyric acids), which exert several effects of major physiological and pathophysiological importance. The production and absorption of SCFAs facilitate the uptake of electrolytes and water and reduce the osmotic effect of unabsorbed carbohydrate molecules. Hence, diarrhea will ensue if colonic fermentation capacity is impaired (e.g., antibiotic-associated diarrhea) or overwhelmed (e.g., lactase deficiency) (6). Colonocytes rely mainly on nutrition from the colonic lumen (the “milieu extérieur”) and have a preference for using butyrate as an energy substrate. “Starvation colitis” may develop if access to SCFAs is hindered, and failure of β-oxidation of SCFAs has been proposed as a pathogenetic mechanism for ulcerative colitis (7). SCFAs, especially butyrate, display antineoplastic properties and may prevent the development of colorectal cancer (3). Furthermore, microbial fermentation products influence gastrointestinal motility and sensitivity and may play a role in the pathogenesis of irritable bowel syndrome (4, 8).

Unfortunately, comparative physiology is no longer a part of the medical curriculum. The microbial contribution to mammalian digestion is a well-recognized concept in veterinary medicine, but humans also eat “grass.” We feel that time has come to acknowledge the human colon as a digestive organ. A description of colonic fermentation and its consequences should be included in every textbook of human physiology.

**REFERENCES**