Author/s:
Kamm, MA

Title:
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Date:
2020-01-01

Citation:
Kamm, M. A. (2020). Processed food affects the gut microbiota: The revolution has started. Journal of Gastroenterology and Hepatology Australia, 35 (1), pp.6-7. https://doi.org/10.1111/jgh.14976.

Persistent Link:
https://hdl.handle.net/11343/275226

Processed Food Affects the Gut Microbiota - the Revolution has Started

Editorial for J Gastro Hep

Michael A Kamm, MD PhD

Professor of Gastroenterology

Affiliations

1. St Vincent's Hospital, Melbourne Australia
2. University of Melbourne, Melbourne, Australia

Correspondence

Professor Michael A. Kamm

St Vincent's Hospital

Victoria Parade

Fitzroy

Melbourne 3065

Australia

Phone: + 61 3 9417 5064

Fax: + 61 3 9416 2485

Email: mkamm@unimelb.edu.au

This is the author manuscript accepted for publication and has undergone full peer review but has not been through the copyediting, typesetting, pagination and proofreading process, which may lead to differences between this version and the Version of Record. Please cite this article as doi: 10.1111/jgh.14976

Inflammatory and metabolic gut and liver diseases appear to be “modern” diseases, whose incidence has increased dramatically in the West in the last 50 years, and in the developing world in the past 25 years (1,2). In parallel with these temporal and geographic changes in disease prevalence there have been huge changes in the way food is produced. With the industrial revolution in the late 1700s, and more markedly after World War I, local agriculture and fresh food consumption changed in the West to mechanised and industrial food production and processing. After World War II, developed nations embarked on economically-driven further change from locally grown and consumed fresh food to food that is stored, processed, chemically altered, and widely distributed (3). Recently this has become a *global process* as the world became a *single market*.

Processed food now constitutes a large part of the world’s food consumption. The proportion of food that is ultra-processed, that is chemically modified, is 60% in the USA, 51% in the United Kingdom, 33% in Australia, and about 14% in Southern European Mediterranean countries. It used to be much less in Asia, but is increasing rapidly. Chemicals are added to food to improve appearance, improve longevity and enhance taste. These chemicals include emulsifiers, preservatives, colourings, non-sugar sweeteners and anti-oxidants. The number of additives permitted by regulatory authorities to be included in food is greater than 300 in Australia, much greater in the USA, and poorly defined in many countries. The approval for use of food additive has historically been based on animal toxicity studies (3).

The gut microbiota, the vast ecosystem of microorganisms in our gut, is now regarded as central to a range of gut-related diseases, such as inflammatory bowel disease, Crohn’s disease and ulcerative colitis, non-alcoholic fatty liver disease (NAFLD), non-alcoholic steato-hepatitis (NASH), obesity and diabetes (4). It may also play a role in many non-gut disorders. Recent studies have demonstrated that chemical food additives interact with the gut microbiota to predispose to, contribute to the development of, or cause many of these conditions.

One of the first mechanistic studies to demonstrate that food additives might be harmful in this respect came from Chassaing and colleagues (5). They reported that low concentrations, as would be found in human processed food, of two commonly-used emulsifiers, carboxymethylcellulose and polysorbate-80 (P-80), induced low grade inflammation and metabolic syndrome in wild-type mice. These emulsifiers also induces colitis in predisposed mice. Emulsifier-induced metabolic syndrome is associated with destruction of the protective mucous layer, microbiota epithelial encroachment, altered species composition and inflammation. These destructive changes are not direct chemical effects of the emulsifiers on the epithelium but mediated via alterations in the enteric microbiota.

Further studies have highlighted which opportunistic pro-inflammatory organisms, that is “pathobionts”, proliferate, and which anti-inflammatory organisms are inhibited, when the enteric environment is altered by chemical food additives. When Rodriguez-Palacios et al (6) fed sucralose-maltodextran (an artificial sweetener - Splenda) to mice with a Crohn’s-like ileitis and healthy mice there was an expansion of *Proteobacteria* and associated inflammation in susceptible mice.

Jimenez Loayza et al demonstrated *in vitro* (7) that the commonly-used food emulsifiers sodium sulfite & P-80 inhibit the growth of *Faecalibacterium prausnitzii,* a key anti-inflammatory organism.

In the recent elegant study by Furuhashi *et al* in this issue of the Journal (8) the effect of dietary emulsifiers on small-intestinal microbiota, a key determinant of gut immunity, was examined. Mice fed P-80 demonstrated increased *Gammaproteobacteria* abundance and decreased microbial diversity. P-80 pretreatment also exacerbated small-intestinal lesions induced by indomethacin. Ileal culture demonstrated that P-80 significantly increased the abundance of *Proteus mirabilis*. *Proteus* are motile, swarming organisms - this is one of their numerous virulence mechanisms (9). In their study (8) adding P-80 to an agar plate culture increased the diameter of *P. mirabilis* colonies, suggesting that the growth and virulence of pathobionts are enhanced by this emulsifier. *Proteus mirabilis* has recently been implicated as a potentially causative organism in active Crohn’s disease (10). The accumulating data that increased abundance and virulence of this organism are induced by changes in food that have occurred simultaneous with the increasing Crohn’s disease prevalence make for a credible link.

Studies such as that by Furuhashi et al (8) should stimulate us to explore further food -microbiota interactions, and the diseases in which they play some pathophysiological part. Food additive effects on the gut microbiota, and their potential to cause microbiota-related diseases, now need to be included as part of their safety evaluation.

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