



Dietary Emulsifier Exposure and Mucosal Barrier Markers in Adults With Quiescent Ulcerative Colitis: A Review of the Evidence

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Abstract

Adults whose ulcerative colitis is in remission often ask whether processed food, and food additive emulsifiers in particular, could weaken the colonic barrier and bring on a flare. This narrative review sets out what the published literature can and cannot say in answer. It first describes the layered structure of the colonic barrier and the markers used to assess it, from sugar permeability probes to mucus thickness and confocal endomicroscopy, and notes that faecal calprotectin is a marker of inflammation and not of barrier integrity. It then examines the experimental work, which shows that carboxymethylcellulose and polysorbate 80 thin the mucus layer, alter the microbiota and promote colitis in susceptible mice, and the much smaller body of human work: a controlled feeding study in healthy volunteers, one small randomised trial of carrageenan in ulcerative colitis in remission, and cohort studies that use ultra-processed food as a proxy for additive intake. The chain of inference from mouse to patient is plausible but incomplete. No published study has measured emulsifier intake and direct barrier markers together in adults with quiescent disease. The review identifies exposure measurement, marker validity and the separation of barrier change from subclinical inflammation as the main obstacles, and outlines the studies needed to resolve them.

Keywords: *Ulcerative colitis, Food additive emulsifiers, Carboxymethylcellulose, Intestinal permeability, Mucus layer, Ultra-processed food*

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1. Introduction

Ulcerative colitis is a chronic inflammatory disease of the colonic mucosa that follows a relapsing and remitting course. Treatment now aims beyond the control of symptoms at endoscopic healing (Turner et al., 2021), yet a patient whose disease is quiescent by every clinical measure still carries a mucosa that differs from a healthy one and still faces a risk of flare that is hard to predict (Ungaro et al., 2017). People in remission want to know what they should eat to stay there, and clinicians have had little firm evidence

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to offer in reply.

Among the dietary candidates, food additive emulsifiers have drawn particular attention since [Chassaing et al. \(2015\)](#) reported that two synthetic compounds, carboxymethylcellulose and polysorbate 80, thinned the protective mucus layer, changed the gut microbiota and promoted colitis in genetically susceptible mice. The suspicion was older than that paper: [Roberts et al. \(2013\)](#) had already proposed rising emulsifier consumption as an explanation for the rising incidence of Crohn's disease. For ulcerative colitis the idea has obvious appeal. The disease is one of the mucosal surface, emulsifiers are surface-active molecules, and the consumption of processed food has grown in the same countries and decades in which inflammatory bowel disease has become common.

This review asks a narrow question: what does the published evidence say about emulsifier exposure and markers of mucosal barrier integrity in adults with ulcerative colitis in remission? Quiescent disease is the state in which dietary advice is most often sought, and it is also the state in which an effect on the barrier could, in principle, be told apart from the damage done by active inflammation. The short answer is that the evidence is largely indirect, and the purpose of the article is to show where each link in the argument is strong and where it is not.

2. Scope and Approach

This is a narrative review of the published literature. It draws on experimental studies in animals and in laboratory models of the human microbiota, controlled feeding studies in people, dietary trials in inflammatory bowel disease, cohort studies of ultra-processed food, and earlier reviews and professional guidance. No systematic search was carried out, and the article does not attempt a quantitative synthesis. The term emulsifier is used in the broad sense common in this literature. It covers true emulsifiers such as polysorbates and lecithins and also the thickening and stabilising hydrocolloids, such as carboxymethylcellulose and carrageenan, that are usually grouped with them ([Cox et al., 2021](#)).

3. The Colonic Barrier and Its Markers

The colonic barrier is a layered structure, shown schematically in Figure 1. Its outermost element is mucus, organised around the gel-forming mucin MUC2 into two layers: a loose outer layer that houses bacteria and a dense inner layer that, in health, keeps them away from the epithelium ([Johansson et al., 2008](#)). Mice lacking Muc2 develop colitis spontaneously ([Van der Sluis et al., 2006](#)). In people with active ulcerative colitis the inner layer is penetrable to bacteria, and [Johansson et al. \(2014\)](#) found the same defect in some patients whose disease was in remission. That observation is the main reason to think that quiescent colitis might be unusually sensitive to anything that disturbs mucus.

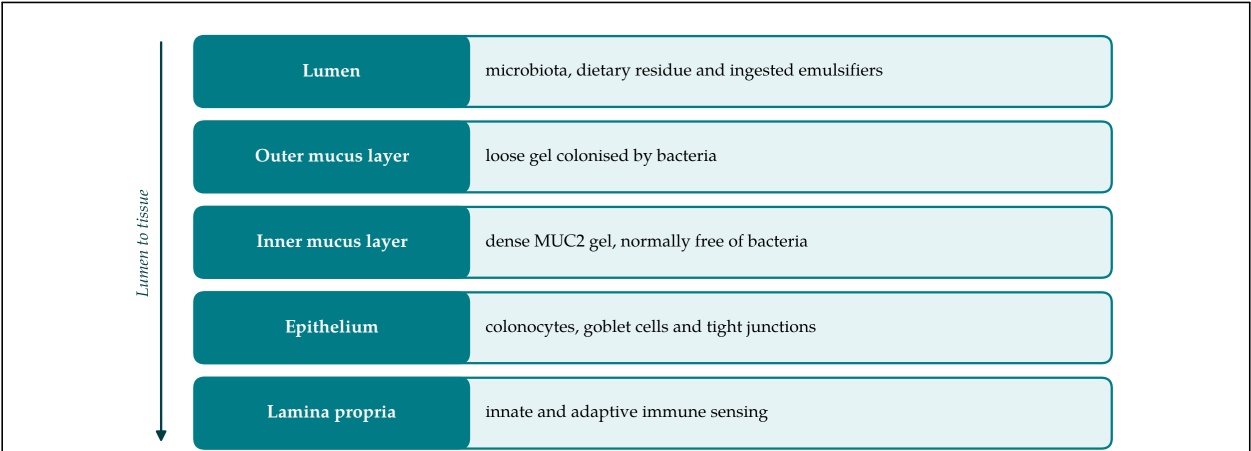


Figure 1: Layers of the colonic barrier at which dietary emulsifiers have been proposed to act, from the lumen to the lamina propria

Beneath the mucus lies a single layer of epithelial cells sealed by tight junctions, whose permeability is regulated and is increased by inflammatory cytokines (Turner, 2009). Below that, immune cells of the lamina propria respond to whatever crosses. A change at any level can be called a change in barrier function, which is why the phrase needs to be tied to a specific measurement whenever it is used.

No single marker captures the barrier as a whole (Bischoff et al., 2014). Functional tests measure the urinary recovery of ingested sugar probes. The familiar lactulose and mannitol pair mainly reflects the small intestine, because both sugars are fermented in the colon; sucralose, which resists fermentation, has been used to extend the test to the large bowel. Circulating markers such as lipopolysaccharide-binding protein are taken as indirect signs of bacterial translocation. Tissue measures include the thickness of the mucus layer, which is lost in routine processing and needs special fixation, the distance between bacteria and the epithelial surface, and the expression of tight junction proteins. Confocal laser endomicroscopy can show epithelial gaps and fluorescein leakage in vivo, and such findings predicted relapse in patients with inflammatory bowel disease in remission (Kiesslich et al., 2012). Faecal calprotectin, although often reported beside these measures, reflects neutrophil traffic into the lumen. It is a marker of inflammation and should not be read as a marker of barrier integrity.

4. Emulsifiers in the Food Supply and the Problem of Exposure

Emulsifiers are amphiphilic or gel-forming compounds added to food to keep oil and water mixed, to improve texture and to extend shelf life. They are found in bread, ice cream, sauces, spreads, confectionery and plant-based milks, among many other products, and they are authorised additives with their own E numbers in the European system. Table 1 lists the compounds most often discussed in relation to the gut, with a summary of the experimental and human evidence for each.

Table 1: Emulsifiers and thickeners most often studied in relation to the gut barrier, with the nature of the evidence for each			
Additive (E number)	Typical foods	Experimental findings	Human evidence
Carboxymethylcellulose (E 466)	Ice cream, sauces, gluten-free bakery	Thinner mucus, altered microbiota and colitis in susceptible mice Chassaing et al., 2015	Feeding study in healthy adults Chassaing et al., 2022; none in colitis
Polysorbate 80 (E 433)	Ice cream, sauces, some desserts	As above in mice; more bacterial translocation in cell models Roberts et al., 2010	No controlled feeding study in colitis
Carrageenan (E 407)	Dairy desserts, plant-based milks, processed meat	Long used to induce colitis in animal models	One small trial in colitis in remission Bhattacharyya et al., 2017
Lecithins (E 322)	Chocolate, margarine, bakery	Little effect on microbiota ex vivo Naimi et al., 2021	No direct barrier studies in colitis
Mono- and diglycerides of fatty acids (E 471)	Bread, spreads, ice cream	Sparse; rarely tested in barrier models	Intake estimates only

The practical obstacle to studying these compounds in people is that exposure is very hard to measure. Labels state that an additive is present, not how much. Food composition tables do not record additive content, dietary questionnaires were not designed to capture it, and manufacturers change recipes without notice (Cox et al., 2021). Most observational work has therefore fallen back on the intake of ultra-processed food as defined by the NOVA classification (Monteiro et al., 2019). That proxy bundles emulsifiers together with sweeteners, colours, refined starch, fat, salt and a low fibre intake, so it cannot isolate the effect of any one of them.

Regulatory approval offers less reassurance on this specific question than might be assumed. The European Food Safety Authority re-evaluated the celluloses, including carboxymethylcellulose, and saw no need for a numerical acceptable daily intake (EFSA, 2018). Such assessments rest on conventional toxicological endpoints. As Halmos *et al.* (2019) point out, they were not designed to detect changes in the microbiota or the mucus layer, and they consider the general population and not people with an already abnormal mucosa.

5. Experimental Evidence

The central experiment remains that of Chassaing *et al.* (2015). Mice given carboxymethylcellulose or polysorbate 80 at low concentrations developed low-grade intestinal inflammation and features of metabolic syndrome, and mice predisposed to colitis by deletion of the interleukin-10 or Toll-like receptor 5 genes developed overt disease more often and more severely. Bacteria were found closer to the epithelium, and the mucus layer was thinner. The effect depended on the microbiota: it did not occur in germ-free animals and could be transferred to them by faecal transplantation. This points to an indirect mechanism, in which emulsifiers change the composition and behaviour of the bacterial community and the altered community then erodes or breaches the mucus.

Other models agree in outline. Carboxymethylcellulose given to interleukin-10 deficient mice caused bacterial overgrowth and inflammation of the small intestine (Swidsinski *et al.*, 2009). In cell culture, polysorbate 80 increased the translocation of *Escherichia coli* across epithelial monolayers and M cells, whereas soluble plant fibres reduced it (Roberts *et al.*, 2010). Long-term exposure of mice to either synthetic emulsifier also promoted tumour development in a model of colitis-associated cancer (Viennois *et al.*, 2017).

Work with human microbiota outside the body adds two points. In a simulator of the human intestinal ecosystem, both compounds raised the proinflammatory potential of the community, notably its content of bioactive flagellin, and the treated microbiota induced inflammation when transferred to germ-free mice (Chassaing *et al.*, 2017). A later screen of twenty commonly used emulsifiers found that they are not alike. Many altered microbiota composition or its inflammatory potential, while lecithin was among those with little effect (Naimi *et al.*, 2021). Speaking of emulsifiers as a single exposure is therefore misleading.

These findings have well-known limits as a guide to human disease. The murine colon and its mucus differ from the human in scale and in bacterial ecology. The animals received the additive continuously in drinking water, without the food matrix in which people consume it. Two compounds account for most of the data, and they are not the emulsifiers eaten in the greatest quantity. The experiments establish that a mechanism exists. They do not establish that it operates at ordinary human intakes.

6. Evidence in Humans

The most direct human evidence comes from a controlled feeding study in healthy volunteers who were housed as inpatients and given an emulsifier-free diet with or without added carboxymethylcellulose (Chassaing *et al.*, 2022). Over a short period the additive increased abdominal discomfort after meals, reduced microbial diversity and depleted faecal short-chain fatty acids. In a subset of participants bacteria were seen closer to the epithelium, which echoes the encroachment described in mice. The study was small and brief, the dose was above usual dietary intake, and the participants did not have colitis. It shows that a human response is possible and that people vary in their susceptibility.

Only one randomised trial has enrolled patients with ulcerative colitis in remission and manipulated an additive of this class. Bhattacharyya *et al.* (2017) placed participants on a carrageenan-free diet and assigned them to capsules of carrageenan or placebo. Relapses occurred in the carrageenan group and not in the placebo group. The number of participants was very small, and the outcomes were clinical relapse and inflammatory markers, not direct measures of mucus or permeability. The trial is best regarded as a signal that deserves replication.

Dietary restriction has been tested for feasibility. Sandall *et al.* (2020) showed that people with Crohn's

disease could follow a low-emulsifier diet, which matters because any trial of avoidance depends on adherence. Comparable work in ulcerative colitis in remission, with barrier markers as endpoints, has not been published to the knowledge of this review.

Cohort studies give a mixed picture. In a large multinational cohort, higher intake of ultra-processed food was associated with a higher risk of incident inflammatory bowel disease (Narula *et al.*, 2021). In three cohorts of health professionals in the United States the association was seen for Crohn's disease but not for ulcerative colitis (Lo *et al.*, 2022). These studies concern the onset of disease and not the course of established colitis, and their exposure measure is the crude proxy already described. They neither confirm nor exclude an effect of emulsifiers on the barrier in remission.

Professional guidance reflects this uncertainty. The International Organization for the Study of Inflammatory Bowel Diseases advised that it may be prudent to limit foods containing carboxymethylcellulose, polysorbate 80 and carrageenan, while stating that the supporting evidence is of low quality and comes mostly from animals (Levine *et al.*, 2020).

7. Interpreting Barrier Markers in Quiescent Disease

Even a well-conducted study in patients in remission would face three problems of interpretation. The first is that remission is not a uniform baseline. Clinical and endoscopic remission can coexist with residual histological activity, and current treatment targets treat histology as an adjunct and not as a formal goal (Turner *et al.*, 2021). Two patients labelled quiescent may thus start from different states of the mucosa.

The second is direction of effect. Inflammatory cytokines open tight junctions and deplete goblet cells (Turner, 2009), so a rise in permeability after an exposure may be the result of a small increase in inflammation and not its cause. Separating the two requires markers of inflammation and of the barrier to be measured together and repeatedly. The third problem is the validity of the markers themselves. Sugar probe tests vary with transit and renal function, blood markers of translocation are nonspecific, and tissue measures depend on biopsy site and handling (Bischoff *et al.*, 2014).

8. Gaps and Priorities for Research

The most obvious gap is the absence of any study that measures emulsifier intake and direct barrier markers together in adults with quiescent ulcerative colitis. Filling it calls for two kinds of work. Controlled supplementation or controlled feeding, with blinding where the additive allows it, can test individual compounds over weeks, using mucus thickness, bacterial distance from the epithelium and endomicroscopy as endpoints beside calprotectin and histology. Longer pragmatic trials of low-emulsifier diets can test whether avoidance changes relapse rates, provided the comparison diet is matched for fibre and overall food quality.

Both depend on better exposure assessment. Databases of additive content by brand, biomarkers of intake and food records designed around additives are all needed before observational studies can move beyond ultra-processed food as a proxy. Research should also widen beyond carboxymethylcellulose and polysorbate 80 to the compounds eaten in larger amounts, and should ask why individuals differ in their response, since the feeding study in healthy volunteers suggests that the microbiota a person starts with shapes the effect (Chassaing *et al.*, 2022).

9. Conclusion

The proposition that dietary emulsifiers impair the colonic barrier in ulcerative colitis rests on coherent experimental biology and on very little direct human evidence. Certain emulsifiers disturb the microbiota and the mucus layer in mice and in models of the human gut, and one of them does so in healthy people at high intake. Whether usual dietary exposure alters barrier markers, or the risk of relapse, in adults whose colitis is in remission has not been shown. Until it has, advice to limit heavily processed food can be defended on general nutritional grounds and as a reasonable precaution, but it should be presented to patients as precaution and not as established fact.

Conflict of interest

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