ECCO Consensus on Dietary Management of Inflammatory Bowel Disease

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1

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INTRODUCTION

Diet is gaining recognition amongst clinicians, researchers, and the inflammatory bowel disease [IBD] community as a potential modifier to change the course of the disease. The role of diet in the management of IBD has evolved beyond adjustment for nutritional needs and is now applied as therapy to influence disease activity and complications. With growing recognition of diet, there is a demand for high-quality dietary research to guide practice and consideration on where to best position diet in the treatment paradigm. This has led to the commissioning of this first European Crohn's and Colitis Organisation [ECCO] consensus on dietary management of IBD, with the goal of providing evidence-based recommendations to guide clinical practice.

METHODOLOGY

This consensus followed ECCO standardised methodology and was directed by four project coordinators, selected by the ECCO Guideline Committee to represent an adult gastroenterologist and ECCO guideline committee member [HG], a paediatric gastroenterologist [RH], and dietitians [VS and EPH], all with expertise in diet and IBD. A panel of 25 experts was selected from a competitive pool of applicants that consisted of 12 IBD dietitians, eight adult gastroenterologists, and five paediatric gastroenterologists. Panel members were allocated to one of five working groups. One patient representative from the European Federation of Crohn's and Ulcerative Colitis Associations was assigned to each group.

The topics of the working groups were decided by the project coordinators to comprehensively cover the following: i) Diet as therapy to induce remission of IBD; ii) Diet as therapy to maintain remission of IBD; iii) Dietary management of comorbidities and special conditions of IBD; iv) Nutritional assessment and optimisation for IBD; and v) Diet as prevention of IBD development. Each working group was led by a project coordinator and a fifth working group leader was recruited [MCEL]. Within each working group, clinically relevant research questions were formulated using the the Population, Intervention, Comparison, and Outcome [PICO] approach[1] when possible, in accordance with ECCO standard operating procedures (SOPs) for OCEBM based guidelines.. These informed systematic literature searches performed by a professional librarian [completed January 2024] using PubMed/Medline, Embase, and the Cochrane Central databases. Abstracts from each PICO were screened for relevance by two panel members. Full texts of potentially relevant abstracts were retrieved and evaluated for consensus inclusion by a panel member. Consensus statements and supporting text were drafted for each PICO and allocated a level of evidence [EL] score using the Oxford Centre for Evidence-Based Medicine[2]. When considering *Diet as prevention of IBD development* only prospective cohort studies were included in data analysis, with EL2 ascribed for papers best fitting with 'inception cohort studies'.

All statements underwent three voting rounds and feedback by panel members and patient representatives, followed by revisions. The second voting round was extended to all applicants to the consensus with opportunity for feedback, but consensus was calculated from panel member votes only. Panel members and patient representatives met in person or online in September and December 2024 for the final discussion and consensus vote. Consensus was defined as > 80% agreement amongst panel members and patient representatives and is presented for each statement. Only statements achieving consensus were included.

TRANSLATION AND EXECUTION OF DIETARY MANAGEMENT

Statement 1: In the absence of a specific dietary intervention that is recommended by an IBD healthcare professional, healthy eating guidelines should be followed by people with IBD, as recommended for the general population. [EL5] [Consensus: 100%]

Statement 2: All people with IBD should have access to a dietitian with experience in IBD. [EL5] [Consensus: 96%]

To drive engagement with diet as a routine part of IBD care, to enable diet as both a primary and adjunctive therapy, and to facilitate high-quality dietary research, it is essential to include specialist dietetic input in clinical care and research. *Statement 2* is arguably the most important statement within this consensus. Recommendations for nutritional assessment and execution of dietary interventions are outlined through the statements of this consensus, based on evidence for its efficacy and safety. Where evidence is lacking, as indicated by EL5, mechanism-based reasoning should ensue without compromise to safety. Ideally, nutritional assessment should be performed by a dietitian, who can assess and correct abnormalities in nutrition and eating behaviour. Where no such dietetic expertise is available, a healthcare professional with training in nutrition assessment and optimisation should be consulted. A dietitian well-versed in IBD should execute and monitor IBD-specific dietary interventions, particularly diets of restrictive nature, as such diets carry nutritional and psychosocial risks and require skilled dietetic supervision.

DIETARY MANAGEMENT OF PATIENTS WITH IBD

The dietary management of patients with IBD is categorised in three areas [Figure 1], including dietary therapy to induce and maintain remission of IBD, dietary management of comorbidities and special conditions of IBD, and nutritional assessment and optimisation for IBD.

Dietary therapy to induce and maintain remission of IBD

Consensus evaluation of dietary therapies and supplements for both induction and maintenance of remission of IBD is presented in this section and are summarised in Table 1 and Table 2. Several areas of interest had insufficient evidence to make a recommendation. Most evidence for dietary interventions was of low quality, with few studies measuring endoscopic, radiological or biochemical endpoints or durability of response. Hence, positioning dietary therapies in treatment algorithms is challenging. It is often appropriate to consider dietary intervention in conjunction with medical therapy. Descriptions of defined diets given recommendations for use in the management of IBD are summarised in Table 3.

Exclusive enteral nutrition and partial enteral nutrition

Statement 3.1: Exclusive enteral nutrition [EEN] is effective for the induction of clinical and endoscopic remission in children [EL1] and adults [EL2] with mild-to-moderate Crohn's disease [CD]. [Consensus 96.6%]

Statement 3.2: Partial enteral nutrition [PEN] provided in high volumes could improve disease activity, although details of optimal dose are unclear. The use of ≤ 50% PEN is not recommended for the induction of remission of CD. [EL3] [Consensus 90.0%]

Statement 3.3: Polymeric formulas for EEN and PEN are equally effective to elemental and semi-elemental formulas and should be recommended. [EL2] [Consensus 89.7%]

Statement 3.4: The use of EEN or PEN as induction therapy for ulcerative colitis is not recommended. [EL2] [Consensus 100%]

Exclusive enteral nutrition [EEN] is a treatment that replaces all food with oral nutrition supplements, usually for 6–8 weeks. Partial enteral nutrition [PEN] replaces a proportion of food with oral nutritional supplements.

The efficacy of EEN inducing remission of active Crohn's disease [CD] in paediatric and adult populations is supported by several systematic reviews and meta-analyses[3-13]. A Cochrane review, including 27 studies and 1011 adult and paediatric participants with CD, showed high remission rates for both children [83%] and adults [43%], with no difference between enteral formulas, including fat content and elemental versus polymeric composition[10]. While remission rates between elemental and polymeric formulas are comparable, polymeric formulas may be better tolerated[14]. EEN remission rates were comparable with corticosteroids [EEN: 83%, corticosteroids: 61%] for children but not for adults [EEN: 45%, corticosteroids: 73%] in intention to treat analysis[10]. However in per protocol analysis, there was equivalence between remission rates in adults too (RR 0.93, 95% CI 0.75 to 1.14), suggesting the therapies are equally effective in adults when EEN is tolerated. The equal clinical efficacy of EEN and corticosteroids in the paediatric population, both for newly diagnosed and relapsing CD, has been reported elsewhere[6,12,13]. Normalisation of C-reactive protein [CRP] and faecal calprotectin did not differ between groups, while EEN was superior to corticosteroids for endoscopic and histological healing[5,12,13]. Interestingly, recent paediatric studies have shown similar efficacy for remission induction for EEN and infliximab[15,16].

EEN use in adults has become more common[10,15,17-20], with studies with good adherence demonstrating equal efficacy in adult and paediatric cohorts[4]. Two randomised controlled trials [RCT] that compared EEN with corticosteroids reported excellent tolerance and similar efficacy between groups[21,22]. Mucosal healing rates as high as 79% and a significant reduction in bowel-wall thickness have been reported for EEN in prospective observational studies of adult patients[17,23]. Emerging data

also support the use of EEN in abdominal abscesses, intestinal fistulae, and inflammatory intestinal strictures[24-27]. There are additionally emerging retrospective data suggesting that EEN may augment response to advanced therapies, with a multicentre study of patients with ileal CD commencing advanced therapy demonstrating mucosal healing in 85.7% of those also receiving EEN for 16 weeks, compared with 23.7% receiving advanced therapy alone[28].

In paediatrics, PEN is likely less effective than EEN, although the proportion of enteral nutrition has varied between studies. The first trial comparing PEN and EEN in CD found lower clinical remission for PEN [15%] versus EEN [42%] after 6 weeks, although both reduced disease activity, with stronger benefits in EEN[29]. However, in a separate study when PEN accounted for 47% of caloric intake, remission was achieved by 50%, 76%, and 73% in PEN, EEN, and biologic groups, respectively, based on faecal calprotectin[16]. When PEN provided 80–90% of caloric intake, 87% clinical response, 65% remission, and improvements in biochemical outcomes were observed irrespective of disease location[30]. However, for a similar protocol only 42% completed the prescribed course due to lack of response [25%] and intolerance [23%][31].

In adults, PEN that provides at least 50% of caloric intake alongside adalimumab led to remission rates and inflammatory marker improvements similar to EEN, outperforming regular diet[32].

Data on the use of EEN and PEN for the treatment of active ulcerative colitis [UC] are limited. A single small open label RCT suggests that EEN may reduce hospital stay and corticosteroid failure in patients with acute severe UC, but these findings require replication[33].

Statement 4.1: Partial enteral nutrition [PEN] could be considered for the maintenance of remission in Crohn's disease [EL2], including as an adjunct to medical therapy [EL3], with the most convincing evidence of efficacy for PEN comprising >35% daily energy requirements [EL2]. However, details of optimal duration and psychological impact of long-term PEN are unknown. [Consensus 96%]

Statement 4.2 There is insufficient evidence to recommend PEN for the maintenance of remission of ulcerative colitis. [EL5] [Consensus 88%]

PEN as maintenance therapy for CD has been evaluated by prospective trials, retrospective trials, and RCTs as reported in several meta-analyses and systematic reviews[34-37].

The data are broadly supportive of PEN for maintenance of clinical remission in CD in adults and children. The most recent systematic review and meta-analysis, which consisted of 64 studies, including 11 RCTs, revealed efficacy of PEN as maintenance therapy when prescribed at higher doses[35]. The risk of clinical relapse was significantly reduced at intake > 35% of energy requirements (35–50% PEN, odds ratio [OR]: 0.42; 95% confidence interval [CI]: 0.27–0.65) and even lower with > 50% PEN [OR: 0.27; 95% CI: 0.08–0.88]. A follow-up of 268 patients with CD demonstrated that maintenance PEN > 900 kcal/day was associated with reduced risk of hospitalisation (hazard ratio [HR]: 0.62; 95% CI: 0.44–0.87, $p = 0.007$)[38]. Conversely, no studies with PEN < 35% have described benefit[39]. PEN may also aid corticosteroid weaning in refractory CD[40].

Most studies did not include endoscopic or biochemical endpoints[35-37]. One prospective observational study did not find a difference between endoscopic outcomes of PEN compared with usual diet at 12 months (relative risk [RR]: 0.79; 95% CI: 0.57–1.10)[41], although a prospective study from the same group did show a reduction in 12-month endoscopic relapse rate in 40 participants post ileal or ileocolic resection [RR: 0.43; 95% CI: 0.21–0.89][42]. The efficacy of polymeric and elemental formulations is comparable[43].

When considering PEN to augment response to medical therapy, this has been best studied in people receiving anti-tumour necrosis factor [TNF] therapy. One meta-analysis, including data from clinical trials and prospective and retrospective cohort studies evaluating PEN with infliximab or adalimumab, revealed the maintenance effect of the PEN group to be 70.5% [203/288] compared with 53.8% [306/569], yielding a pooled OR for long-term remission or response of 2.19 [95% CI: 1.49–3.22][34]. A meta-analysis focusing on three studies showed improved 12-month infliximab efficacy with PEN > 600 kcal/day clinical remission rates for CD compared with infliximab alone (74.5% [79/106] vs 49.2% [62/126], OR: 2.93; 95% CI: 1.66–5.17, $p < 0.01$)[34]. However, there are limited data on endoscopic and biochemical outcomes, with details on immunogenicity and immunomodulator use also lacking.

There is no evidence to support PEN for maintaining remission in UC.

The Crohn's Disease Exclusion Diet

Statement 5.1: The Crohn's disease exclusion diet [CDED] with partial enteral nutrition [PEN] is recommended for induction of remission in children [EL2] and could be considered in adults [EL3] with mild-to-moderate Crohn's disease [CD]. [Consensus 100%]

Statement 5.2: There is insufficient evidence to support the use of the CDED without PEN as induction therapy for CD. [EL3] [Consensus 100%]

Statement 5.3: There is no evidence to support the use of the CDED, with or without PEN, as induction therapy for ulcerative colitis. [EL5] [Consensus 100%]

Statement 5.4: There is insufficient evidence to recommend the CDED for maintenance of remission of CD. [EL3] [Consensus 96%]

The Crohn's disease exclusion diet [CDED], combined with PEN covering 50% of energy requirements, is a 6-week induction therapy [described as 'Phase 1'- further phases explained below] for treating paediatric and adult populations with CD. This 6-week diet comprises 50% PEN, using a polymeric oral nutritional supplement, mandatory inclusion of five foods of specified dose to be eaten daily, and an allowance of 14 foods that may be eaten. All other foods are disallowed.

The efficacy of CDED for inducing remission of active CD has been assessed in various studies[44-48]. RCT data from paediatric participants with active CD revealed that CDED with 50% PEN was better tolerated and equally as effective as EEN in inducing remission at 6 weeks [CDED+PEN: 80%; EEN: 73.5%]. Both therapies significantly reduced faecal calprotectin [CDED+PEN: median 3126 to 1744µg/g, $p = 0.002$; EEN: 2647 to 1021µg/g, $p = 0.011$][49]. Subsequent analysis showed that rapid response [94%] and remission [81%] based on faecal calprotectin levels within 3 weeks predicted sustained control at 6 weeks[50]. These data are supported by retrospective evidence comparing CDED with 50% PEN and EEN [remission rate CDED+PEN: 75%; EEN: 66%][51]. Those receiving CDED with 50% PEN had superior growth, although 80% of the cohort had 1–2 weeks of EEN prior to therapy[51]. A prospective study revealed that CDED with PEN and EEN had similar clinical [CDED+PEN: 85%; EEN: 81%], endoscopic [CDED+PEN: 54%; EEN: 50%], and mucosal healing rates [CDED+PEN: 39%; EEN: 44%] after 6 weeks, although PEN covered 75% of energy needs[52]. Preliminary data suggest that CDED with 50% PEN may be more effective for treatment-naïve children than those with prior biologic therapy[45].

In adults, an open-label pilot trial found similar 6-week remission rates between CDED with 1000 kcal PEN [68%] and CDED alone [57%, $p = 0.46$][53]. An observational trial supported use of the CDED with 50% PEN, which induced remission in 77% and response in 83% of adults with CD[54].

For maintenance therapy, a step-down diet has been assessed, which involves reducing PEN to 25% of energy requirement and an expanded range of permitted foods for 6 weeks [Phase 2], then a restricted diet without PEN [Phase 3]. This maintenance therapy has been assessed in two small studies of mild-to-moderate CD with follow-up to 24 weeks after the aforementioned 6-week induction diet. One of the studies in advanced therapy-naïve adults with mild-to-moderate CD (defined as Harvey Bradshaw Index [HBI] 5–14) compared CDED with 1000 kcal PEN to CDED alone[53]. In the 20 participants who achieved remission at week 6, 80% were still in sustained clinical remission at week 24 [12 CDED+PEN; 8 CDED alone][53]. At week 24, 35% [14/40] of participants were also in endoscopic remission [simple endoscopic score-CD ≤ 3; 72.8% decrease from baseline in n = 22][53]. A real-world follow-up of patients treated with CDED to maintain remission demonstrated that remission was maintained in 83% [20/24] of patients at week 12[44]. However, there was significant heterogeneity, with 54.5% using concurrent medical therapy.

The only long-term analysis of Phase 3 CDED as maintenance therapy was a 52-week observational study in a limited number of children with CD in deep remission withdrawing from immunomodulator or anti-TNF monotherapy, whereby there was no difference in week-52 clinical remission between those who followed CDED (67% [5/9]) compared with a free diet (55.6% [5/7], $p = 0.63$)[55].

Parenteral nutrition

Statement 6.1: While total parenteral nutrition is effective as induction therapy for IBD, with stronger evidence for Crohn's disease [CD] than ulcerative colitis [UC] [EL1], given the risk of harm, parenteral nutrition is not recommended. [EL3] [Consensus 90%]

Statement 6.2: Total parenteral nutrition is not recommended for maintenance of remission of CD [EL4] or UC. [EL5] [Consensus 100%]

Intravenous feeding, described as parenteral nutrition [PN] or total [exclusive] parenteral nutrition [TPN], has been investigated in the treatment of active IBD.

When given to people with active CD or UC, PN improved symptoms, weight, mucosal healing, and subsequent corticosteroid reduction and surgery avoidance in historic studies conducted before 1990[56-60]. A systematic review and meta-analysis of PN in treatment of IBD [15 studies qualitatively, 10 quantitatively] revealed improvements in disease activity and albumin but no increase in body weight. Ten of the studies assessed PN for treatment of active IBD, while the other studies focused on surgical outcomes[4]. A controlled trial of PN with PEN in hospitalised patients with IBD demonstrated superior clinical response compared with a regular hospital diet, with benefits in high-sensitivity CRP, erythrocyte sedimentation rate [ESR], and albumin[61].

Of the two studies that assessed PN in an active CD cohort, similar remission rates were observed for TPN compared with polymeric EEN and PN [71%, 58%, and 60%, respectively] with food in one trial[62], which was similar to the other, showing comparable efficacy of TPN and EEN for treating CD[63]. A retrospective sub-analysis of 12 participants with CD colitis compared TPN treatment alone with a cohort of 2 × 11 participants with moderate-to-severe UC receiving corticosteroids with TPN or a hospital diet, respectively, and showed that TPN led to symptom resolution and nutritional improvements in the participants with CD[64]. No TPN-related differences in outcomes were seen in the participants with UC[64]. Indeed, another study confirmed the equivalence of TPN and EEN for inducing remission in a population with UC[21]. Regarding safety, a randomised study with similar efficacy outcomes of TPN and EEN among patients with IBD found higher liver dysfunction rates in the TPN group [TPN: 62%; EEN: 6%, $p = 0.002$][65], while the risks of catheter-related infections with TPN was

previously described in a meta-analysis[66]. Given these risks and the efficacy equivalence to EEN, PN is not recommended as induction therapy for CD or UC.

For maintenance of remission, there is no evidence to support the use of TPN for CD or UC. TPN use in those with refractory CD led to high rates of 12-month clinical remission [61%], but corticosteroids were not discontinued and participants only received TPN for a mean 25.5 [± 1.1] days with no clear objective maintenance endpoints and no evidence of superiority over EEN or medical therapies[59].

The Mediterranean diet

Statement 7.1: There is insufficient evidence to recommend the Mediterranean diet [MD] as induction therapy for Crohn's disease [CD] or ulcerative colitis [UC]. [EL3] [Consensus 100%]

Statement 7.2: The MD could be considered for maintenance of remission in UC as an adjunct to medical therapy. There is insufficient evidence to recommend the MD for maintenance of remission in CD. [EL3] [Consensus 83%]

The Mediterranean diet [MD], widely recognised for metabolic benefits, has shown mixed results in managing IBD.

A 12-week RCT in children with IBD revealed that the MD lowered inflammatory activity indices, CRP, and faecal calprotectin compared with those who remained on habitual diet, although the results were confounded by corticosteroid use[67]. A 6-month MD intervention in participants with IBD improved metabolic and inflammatory markers, although most participants were already in remission, limiting its utility as an induction therapy[68]. This limitation also affected another trial in CD where the MD achieved modest symptomatic and biomarker responses, but 85% of participants had normal baseline faecal calprotectin levels [< 250 µg/g][69].

For maintenance of remission, one prospective interventional study assessed 84 adult patients with UC and 58 with CD who followed the MD for 52 weeks[68]. Although the study revealed improvements in clinical and biochemical outcomes associated with dietary adherence, there were significant biases in the analysis, such as exclusion of participants who commenced corticosteroids[68].

A single-centre prospective cohort study followed 693 patients with IBD [UC: n=373; CD: n=320] for a median of 27 months (interquartile range [IQR]: 22–29 months), using the PREMIMED questionnaire to assess adherence to the MD in addition to assessing activity levels[70]. A sub-analysis of participants with UC showed that those who adhered to the MD were less likely to flare [p = 0.034], had lower faecal calprotectin [p = 0.018], and had reduced need for corticosteroids [p = 0.023]. Median time to relapse was also greater in those who adhered to the MD (MD adherence: 14 months [IQR: 6.8 – 19.5]; non-adherence: 5.5 months [IQR: 3.8–15], p = 0.03). A similar trend was found in participants with CD, although this did not reach statistical significance.

The Specific Carbohydrate Diet

Statement 8.1: There is insufficient evidence to support the use of the specific carbohydrate diet [SCD] as induction therapy for Crohn's disease [CD] or ulcerative colitis [UC]. [EL3] [Consensus 96.2%]

Statement 8.2: There is insufficient evidence to recommend the SCD for maintenance of remission of CD or UC. [EL4] [Consensus 100%]

The Specific Carbohydrate Diet [SCD] excludes disaccharides and polysaccharides found in grains, legumes, starchy vegetables, dairy, and manufactured food.

For induction therapy with the SCD, initial small retrospective studies of up to 26 children with mild-to-moderate IBD showed improvements in disease activity indices and CRP[71-73]. However, prospective analysis of the SCD revealed variable results. Three small paediatric observational studies of up to 12 participants showed clinical improvements over 12 weeks, although results were mixed over 26 months[74-76]. In another small trial of children with mild-to-moderate CD, 12 weeks of the SCD improved disease activity indices and CRP in 4 participants, as did a modified SCD and a whole-food diet in 4 and 2 participants, respectively[77]. In a large trial of 191 adults randomised to the SCD or the MD, both cohorts had improvements in symptoms but no differences in inflammatory biomarkers, including faecal calprotectin. Many participants had quiescent disease on study entry[69]. Adherence to the SCD remains challenging, with high dropout rates in some studies due to adverse events or lack of response[78].

Studies assessing the SCD as maintenance therapy for IBD are small, heterogenous, and exclusive to the paediatric population. In 7 children with active CD following the SCD for 52 weeks, clinical response [HBI: 0.1 ± 0.4 and Paediatric CD Activity Index: 5.4 ± 5.5, $p = 0.016$ and $p = 0.027$, respectively] and mucosal response [assessed by capsule endoscopy; 2 children had mucosal healing] were maintained, with no patients requiring changes to medical therapy[74]. Conversely, no significant benefits were seen in clinical or biochemical outcomes in paediatric participants with IBD [CD: n = 20; UC: n = 6] following the SCD over 3–48 months[72]. Ten children were unable to tolerate the diet.

The semi-vegetarian diet

Theoretical and preliminary clinical data suggest that the semi-vegetarian diet [SVD] may be of benefit for maintaining IBD remission, although data are currently too limited and heterogenous to draw conclusions. The SVD includes rice, vegetables, fruit, potatoes, fish once per week, meat every 2 weeks, and regular exercise. Sweets, bread, cheese, margarine, fast foods, carbonated drinks, juices, alcohol, and smoking are discouraged. A Japanese group studied the role of a SVD in three CD and two UC open-label populations [CD: n = 68; UC: n = 107], mostly initiated during a period of hospitalisation[79-84]. There was heterogeneity between clinical studies in clinical outcomes and use of endoscopy, with some studies undertaken only in responders. These studies demonstrated a reduction in clinical relapse in participants with CD [> 90% remission at 2 years] and UC [25% relapse rate at 1–2 years] associated with normalisation of CRP in approximately half of participants when measured. However, with the various changes advocated, it is difficult to attribute the benefits to SVD. No consensus was reached for the role of a SVD in IBD management.

Red and processed meat

Statement 9: A reduction in the intake of red and processed meat could be considered for maintenance of remission in ulcerative colitis. [EL3] [Consensus 96%]

Meat, particularly red and processed meat, has been implicated in the pathogenesis of IBD, albeit most data exist for its association with UC[85,86]. Furthermore, an average daily red meat intake of only 76 g has been associated with incident colorectal cancer[87], which may be relevant to an IBD population with an already increased risk[88]. The role of meat in established IBD is not clear and its role in predicting relapse has been assessed, mostly through observational studies.

Two studies have assessed the contribution of meat intake to disease flare in a population of combined CD and UC in remission. This included association data from a longitudinal study conducted in two distinct geographic Dutch cohorts that revealed that patients with quiescent IBD [CD: n = 440; UC: n = 284] had lower intake of red meat than those who had a recent flare [38.1 ± 23.7 vs 46.2 ± 34.8 g/day, $p = 0.028$][89]. This was consistent with the second study, a retrospective survey that assessed dietary

patterns in patients with IBD. This study revealed that a diet low in processed meats, if also low in refined carbohydrates and high in fruits and vegetables, was associated with reduced disease activity indices[90]. Associations have also been observed between processed meat and all-cause mortality in a retrospective cohort of 5763 patients with IBD[91].

For CD, studies have examined the role of meat intake in maintaining remission, as in the Food and Crohn's Disease Exacerbation Study [FACES], which assessed time to flare in people with quiescent status[92]. A high red and processed meat intake did not decrease time to disease flare and there was no significant difference in relapse rates between the high meat diet group [62%] versus the low meat diet group [42%][92]. However, this internet-based study had much lower adherence rates in the low meat group [57.3% vs 98.5%]. Lastly, in children with CD completing a course of EEN, reintroduction of meat was associated with an increase in faecal calprotectin[93].

For maintenance of UC remission, the link between red and processed meat was made in one prospective cohort study in 191 subjects with quiescent UC where intake increased risk of relapse when comparing the highest to lowest tertiles [OR: 5.19; 95% CI: 2.1–12.9][94]. This was reflected in a retrospective study of dietary patterns in patients with IBD showing an inverse association between consumption of processed meats and risk of active UC[90].

Reduction of dairy

Statement 10: Cow's milk protein elimination is not recommended as an adjunct induction therapy for ulcerative colitis. [EL3] [Consensus 100%]

Exclusion of cow's milk protein and dairy products in general is a common practice among people with IBD[95]. There are limited data to support the benefit of a dairy-free diet in the management of IBD. In a RCT in children with new-onset UC, a diet that eliminated cow's milk protein as an adjunct to induction treatment with corticosteroids, mesalamine, or both did not increase the proportion of children who achieved clinical remission when compared with an unrestricted diet[96]. No studies have been performed to explore the benefit of cow's milk elimination in disease outcomes in CD. However, the routine use of dairy-based nutritional supplements in the management of active CD with EEN contradicts any notions of a harmful role of cow's milk protein in CD[97].

The efficacy of *Bifidobacterium*-fermented milk [BFM] as a maintenance therapy in UC was investigated in two Japanese trials. While BFM was effective for symptomatic relief in the first pilot trial, results were not replicated in the larger, placebo-controlled trial in 195 patients[98,99]. No studies were identified on maintaining remission in UC.

High- and low-fibre diets

Statement 11.1: There is no consistent evidence to make recommendations on the use of low- or high-fibre diets as induction therapy for IBD. [EL3] [Consensus 95.2%]

Statement 11.2: Neither low- nor high-fibre diets are recommended for maintenance therapy for Crohn's disease or ulcerative colitis. [EL3] [Consensus 96%]

It is currently unclear if the amount and type of fibre play important roles in the management of IBD.

One of the first retrospective case studies on fibre, published in 1979, demonstrated that hospital admissions were significantly lower and shorter in patients with CD who followed an unrefined, high-fibre diet[100]. In a study published in 1985, a low residue/fibre diet did not differ from a high residue/fibre diet when comparing clinical disease activity, need for hospitalisation or surgery, new

complications, nutritional status, or postoperative recurrence in adults with CD[101]. A recent Cochrane systematic review found that the effect of a high-fibre diet on induction of clinical remission in people with active CD was uncertain[102]. Most studies focused on specific fibre supplementation, with one study focusing on the effect of a high-fibre diet, inclusive of wheat bran, on induction of remission in adults with active CD. This study revealed improved health-related quality of life [QoL] and gastrointestinal symptoms but did not demonstrate benefit in biochemical outcomes[103]. These data are consistent with a previous systematic review of RCTs, which concluded that there was no benefit of fibre supplementation for active CD and inconsistent data for UC[104].

For maintaining remission of IBD, there was no clear evidence that either high- or low-fibre diets predispose to disease flare. A sub-analysis within a meta-analysis[36], including two RCTs[105,106], revealed that a high-fibre diet was not superior in maintaining clinical remission compared with a diet low in fibre and high in refined carbohydrates in 556 patients with clinically quiescent CD [RR: 1.04; 95% CI: 0.83–1.30]. However, another RCT showed higher 1-year clinical relapse rates in patients with CD who followed a diet rich in fibre compared with a dietary protocol excluding symptom-triggering foods [100% vs 30%, $p < 0.05$][107]. This study lacked objective measures of inflammation. However, there was a similar observation in a cross-sectional study in 14 children with CD entering clinical remission following treatment with EEN, where median fibre intake during early food reintroduction was higher in those with higher faecal calprotectin levels compared with those with lower faecal calprotectin [median: 12.1 g/day; IQR: 11.2–19.9 vs median 9.9 g/day; IQR: 7.6–12.1, $p = 0.03$][93]. However, a prospective study of patients with CD showed no such association[108]. In a more recent crossover trial of 17 patients with UC in remission or with mild clinical disease, administration of a high-fibre and low-fat diet [median fibre intake: 25.6 g/day] for 4 weeks, compared with a 'healthier' version of a typical American diet [median fibre intake: 18.1 g/day], improved patients' QoL scores but did not demonstrate improved biochemical outcomes[109].

The role of fibre in risk of surgery and hospitalisation is also unclear, with two large prospective studies showing differing results[110,111]. In the first study of over 5000 adults from the UK Biobank, fibre intake in the highest quartile was negatively associated with surgery after a mean follow-up of 11.2 years [HR: 0.63; 95% CI: 0.44–0.89, $p = 0.009$, with a protective effect seen from fibre sourced from fruit and vegetables on sensitivity analysis[111]. In contrast, in a study of over 1000 patients with quiescent IBD, no significant associations were observed between fibre intake and hospitalisation with a median 3-year follow-up [HR: 1.016; 95% CI: 0.99–1.05, $p = 0.294$][110]. Fibre restriction in the context of symptomatic strictures in CD is detailed in *Dietary management of comorbidities and special conditions of IBD*.

The low FODMAP diet

Statement 12: The low FODMAP diet is not recommended for maintenance of remission in Crohn's disease [EL2] and there is no evidence to support its use for maintenance of remission in ulcerative colitis. [Consensus 100%]

The low fermentable oligosaccharides, disaccharides, monosaccharides and polyols [FODMAP] diet is a therapeutic diet commonly utilised for treatment of irritable bowel syndrome [IBS].

In a meta-analysis including 4 RCTs evaluating the low FODMAP diet to maintain remission in quiescent CD compared with normal diet, there was no impact on inflammatory markers after remission was achieved with conventional therapy[112]. No studies were identified that examined the ability of the low FODMAP diet to maintain remission in UC.

Food-specific IgG-guided diet

Statement 13: A food-specific IgG-guided diet is not recommended as induction therapy for IBD. [EL3] [Consensus 100%]

People with IBD may have specific food intolerances. The clinical utility of measurement of serum immunoglobulin G [IgG] to guide food elimination has been studied, with no studies to date supporting this practice.

In active disease, one open-label study using a food-specific IgG-guided diet in CD showed improved clinical disease activity and ESR but no changes in albumin or CRP[113]. A subsequent trial with a sham diet as comparator arm found modest improvements in CD activity and QoL, but no changes in inflammatory biomarkers[114]. Similar subjective markers, such as abdominal pain, were measured in a crossover study that showed no specific improvements from an IgG-guided diet compared with a sham diet in CD; however, 45% of patients dropped out due to poor compliance[115]. In UC, IgG-guided diets improved Mayo scores, BMI, and albumin, but not endoscopic outcomes[116].

Following induction therapy with EEN, one study evaluated food re-introduction based on IgG, including a retrospective study of 64 adults where no difference in relapse rates were seen between those following IgG exclusion and those on unrestricted diet (relapse rate from IgG diet: 12.5% [4/32]; relapse rate from normal diet: 25% [8/32])[117]. No studies were identified on maintaining remission in UC.

A low microparticle diet

Statement 14: A low microparticle diet is not recommended as induction therapy for Crohn's disease. [EL2] [Consensus 100%]

Microparticles, including titanium dioxide and aluminosilicates, are used as food additives.

Two RCTs explored the effect of a low microparticle diet on disease activity in adults with active CD. In the first trial of 20 patients with active corticosteroid-treated CD, a 4-month low microparticle diet significantly reduced CD activity index compared with a control group[118]. However, these preliminary data were not replicated in a subsequent larger, single-blinded, multicentre, placebo-controlled trial[119].

Other diets targeting active IBD

Statement 15.1: There is insufficient evidence to support the use of the CD Treatment-with-EATing [CD TREAT] diet as induction therapy for Crohn's disease [CD] or ulcerative colitis [UC]. [EL3] [Consensus 100%]

Statement 15.2: There is insufficient evidence to support the use of the Autoimmune Protocol diet [AIP] [EL4], IBD-Anti-Inflammatory Diet [AID] [EL4], 4-strategies-to-SUlfide-Reduction [4 SURE diet] [EL4], or UC exclusion diet [UCED] [EL2] as induction therapy for CD, UC, or both. [Consensus 100%]

Statement 15.3: There is no evidence to make recommendations on the benefit of a gluten-free diet for induction of remission in IBD. [EL5] [Consensus 100%]

The Crohn's Disease Treatment-with-EATing [CD-TREAT] diet is a food-based therapy designed to replicate the composition and microbial effects of EEN. While early studies, including an animal model of ileitis and a small open-label trial of 5 paediatric patients, showed promising anti-inflammatory benefits and faecal calprotectin reduction, larger controlled trials are needed to confirm these findings[120]. There were no published studies that evaluated CD-TREAT as maintenance therapy.

Targeting both CD and UC, the autoimmune protocol [AIP] diet is an extension of the Palaeolithic diet that is high in meat but eliminates grains, legumes, certain vegetables, dairy, nuts, seeds, and certain sugars, oils, and food additives. In an open-label study in 9 CD and 6 UC patients, QoL, disease activity indices and faecal calprotectin improved. Among those with follow-up endoscopy, 6 of 7 patients showed improvement after 11 weeks[121,122].

The IBD-Anti-Inflammatory Diet [IBD-AID] is a step-down diet that restricts foods and textures across all food groups. In a small open-label study with 22 adults, *Clostridia* and *Bacteroides* increased, but clinical outcomes were not measured[123]. A retrospective series of 40 adults with IBD [of whom 13 did not attempt the diet] demonstrated symptom improvement and medication reduction in 11 participants[124].

Targeting UC, the 4-strategies-to-SUlfide-REduction [4-SURE] diet reduces total and sulphur-containing proteins and sulphur- and nitrate-containing additives, whilst increasing resistant starch and non-starch polysaccharides. An 8-week study in 28 patients with active UC showed clinical response in 48%, improved faecal calprotectin [from 400 to 175 µg/g, $p = 0.02$], and endoscopic improvement in 36%. Faecal short-chain fatty acids [SCFA] increased by 69%, and QoL improved by 10 points [$p < 0.001$][125].

The Ulcerative Colitis Exclusion Diet [UCED] aims to not only reduce total and sulphur-rich protein but also to reduce saturated and polyunsaturated fats and all food additives while including tryptophan, pectin, and resistant starch. An improved disease activity index was observed in 37.5% [9/24] of children with active disease following the UCED after 6 weeks[126]. A subsequent trial in adults comparing UCED to faecal microbiota transplantation revealed that a greater proportion of participants on UCED achieved clinical remission [40%] and endoscopic improvement [27%]. Mucosal healing was seen in 4/15 in the UCED group[127].

Large controlled trials are needed before formal recommendations on these diets can be made.

Although the literature search encompassed search terms to evaluate the role of both a gluten-free diet and intermittent fasting on IBD disease activity, no relevant studies were found. A weak signal of sustained remission to 26–52 weeks was found in a meta-analysis combining studies of various dietary strategies in quiescent UC[128], but serious imprecisions were identified in all studies and individual study results were of low or very low certainty. Pooling studies with distinct interventions for meta-analysis is also of limited clinical utility.

Dietary supplements to induce and maintain remission of IBD

Consensus evaluation for dietary supplements to induce and maintain remission of IBD are described below and detailed in Table 2.

Curcumin and QingDai

Statement 16.1: Curcumin could be used as an adjunct therapy to mesalamine for induction of remission in mild-to-moderate ulcerative colitis [UC]. [EL3] However, the optimal formulation, duration, and dose are unclear, and due to potential toxicity, medical supervision is advised. [EL5] [Consensus 100%]

Statement 16.2: There is evidence to support curcumin for maintenance of remission in UC, although the optimal formulation, duration, and dose are unclear, with risk of toxicity at high doses. Curcumin is not recommended for maintenance therapy in Crohn's disease. [EL3] [Consensus 96%]

Statement 16.3: QingDai with or without curcumin may be considered as an adjunct for induction of remission of mild-to-moderate UC. [EL3] Due to potential toxicity, medical supervision is advised [EL5]. [Consensus 100%]

Curcumin, the active compound of turmeric and QingDai [QD, also known as indigo naturalis] is a plant-based compound, both studied in adults with UC.

Meta-analyses and a systematic review suggest that combining curcumin with mesalamine may help induce remission in patients not responding to mesalamine alone. The dosages and formulations of curcumin varied in the studies, ranging from 550 mg to 4 g per day, with treatment durations between 4 weeks and 6 months[129-131].

Four studies [2 RCTs, 1 case series, and 1 observational study] examined curcumin supplementation at doses of 2–3 g/day between 9 weeks and 49 months as an adjunct maintenance therapy for IBD[130,132-134]. In post-operative CD, 3 g/day curcumin in addition to azathioprine in 31 subjects was not effective in preventing post-operative disease recurrence compared with placebo and azathioprine [curcumin: 67.7%; placebo: 58.1%, p = 0.60][133]. In fact, higher rates of severe disease recurrence occurred in the curcumin group [curcumin: 54.8%; placebo: 25.8%, p = 0.034]. In UC, one trial found 2 g/day curcumin with oral mesalamine was more effective at maintaining remission at 6 months compared with placebo and mesalamine [curcumin: 4.44, 95% CI: 0.54–15.15; placebo: 15.15, 95% CI: 8.19–32.71, p = 0.049][130], with a relapse rate at 6 months of 4% [curcumin] versus 18% [placebo] [RR: 0.24; 95% CI: 0.05–1.09, p = 0.06][135]. This treatment effect was not sustained at 12 months[130], with comparable relapse rates of 22% [curcumin] versus 32% [placebo] [RR: 0.70; 95% CI: 0.35–1.40, p = 0.31][135].

Some reports suggest curcumin has potential hepatotoxicity[136]. Curcumin may interact with medications, including anticoagulants and diabetes drugs, and may modulate oestrogen receptors[136-140]. It would therefore seem sensible that curcumin be used under clinical supervision with monitoring of liver biochemistry, particularly for long-term and high-dose use.

QingDai has shown efficacy in treating active moderate-to-severe UC in two Japanese RCTs[141,142]. A European trial demonstrated that a combination of curcumin and QD [CurQD] activated the mucosal aryl hydrocarbon receptor pathway and was superior to placebo in inducing clinical remission and achieving endoscopic improvement in those with moderate-to-severe UC, including biologic-experienced patients[143]. Similar results were observed in a multi-centre retrospective cohort of 88 UC patients; 48% of these had prior biologic or small-molecule therapy. Patients received 1.5 g of QD and 1.5 g of curcumin for an 8-week induction phase[144]. Side effects were rare and included headaches, elevated liver enzymes, and cases of intussusception or pulmonary hypertension[141-144]. Clinical supervision would seem appropriate, with dose adjustments for headaches, liver enzyme tests 1 month after starting, and periodic testing every 3–6 months. Further research is needed to determine the optimal dosing, duration, and positioning of CurQD in UC management.

Fibre supplements

Statement 17.1: There is insufficient evidence to recommend psyllium, germinated barley, resistant starch, oat bran, wheat bran, and non-starch polysaccharides for induction therapy in ulcerative colitis [UC] [EL3], and there is no evidence for induction therapy in Crohn's disease [CD]. [Consensus 100%]

Statement 17.2: There is insufficient evidence to recommend psyllium, germinated barley, oat bran, mastiha, or QingDai supplements for maintenance of remission in CD or UC. [EL3] [Consensus 100%]

A wide range of fibre supplements have been examined for induction of remission, maintenance of remission, or both in IBD. Assessment with objective markers of disease activity is limited.

Derived from the husk of *Plantago ovata*, psyllium [also known as ispaghula] is predominantly composed of the non-starch polysaccharide arabinoxylan and has shown some promise in symptom management for patients with UC, but lacks robust evidence for remission induction[145]. In a small [n = 36] double-blinded, placebo-controlled crossover trial of 3.5 g/day ispaghula husk for maintenance of remission, 4/36 participants relapsed within 4 months (ispaghula husk: n = 1 [3%]; placebo: n = 3 [8%], *p*-value not reported)[146]. In an open-label, parallel-group, multicentre trial, 20 g/day *Plantago* seeds were compared with 1.5 g/day mesalamine or combined therapy for 12 months[147]. Although numerically fewer relapses were observed in the combination group, no differences in the probability of maintaining remission at 12 months was observed upon examination of treatment failure rates [Mantel-Cox test, $p = 0.67$; Breslow test, $p = 0.58$]. No studies evaluated psyllium as maintenance therapy for CD.

Wheat bran is a fibre that contains 46% non-starch polysaccharides. In a small intervention study, wheat bran provided daily for 18 months was well tolerated and reduced bile acid faecal excretion among juvenile UC patients but showed no effect on disease activity parameters[148].

The only other fibre supplement assessed, germinated barley fibre, led to reduced frequency of diarrhoea in patients with UC and may also reduce levels of inflammatory markers when provided at 20–30 g/day. However, there was no published comparison to control or placebo[149-152]. There were insufficient data from two prospective open-label trials of 105 adults with quiescent UC who received 20–90 g/d germinated barley as an adjunct to conventional therapy[149,153]. While significant treatment effects were observed in one study up to 12 months [lower disease activity scores, lower withdrawal rates, reduced corticosteroid use, and lower cumulative recurrence rates compared with control; all $p < 0.05$][153] and symptomatic benefit was observed in both trials, germinated barley warrants further evaluation in trials of longer duration with objective endpoints before a clear recommendation can be made[149,153].

A recent systematic review with a meta-analysis on the effect of resistant starches on active IBD was impeded by significant methodological challenge[154]. Outcomes from two prospective trials suggest that oat bran is ineffective at maintaining remission as an adjunct therapy in quiescent UC[155,156].

Mastiha, a traditional herb, as an adjunct to conventional medical therapy was not superior to placebo at maintaining remission in quiescent IBD [placebo: 23.5%; mastiha: 17.6%, $p = 0.549$][157]. There were no studies evaluating supplementation with indigo naturalis or QingDai in the maintenance of remission in quiescent UC or CD.

Prebiotics

Statement 18.1: Prebiotics are not recommended as induction therapy for Crohn's disease [CD]. [EL2] [Consensus 100%]

Statement 18.2: There is insufficient evidence to recommend prebiotics as induction therapy for ulcerative colitis [UC]. [EL3] [Consensus 100%]

Statement 18.3: Prebiotics are not recommended for maintenance of remission in CD or UC. [EL3] [Consensus 100%]

Prebiotics are a class of non-digestible nutrients that undergo selective fermentation by host microorganisms into bioactive molecules [such as SCFA], which confer a health benefit[158].

Individual studies of prebiotics have suggested potential benefits, but further research is required to confirm their efficacy and optimal use[159-164].

The prebiotic effects of the common laxative lactulose [10 g/day] were explored in a 4-month pilot study of patients with active CD [n=17] and UC [n=14]. Lactulose did not have any beneficial effects on clinical activity indices, endoscopic scores, or immunohistochemical parameters but also did not detrimentally alter stool frequency[165].

In quiescent disease, a systematic review and meta-analysis of prebiotic use in IBD demonstrated that in a pooled analysis, prebiotics did not reduce risk of UC flare [RR: 0.84; 95% CI: 0.57–1.26, 4 studies; n = 332][166]. Germinated barley foodstuff trended towards prevention of clinical relapse [RR: 0.40; 95% CI: 0.15–1.03, n = 59], while inulin, oat bran, and *P. ovata* showed no difference compared with controls[166]. For CD, inulin and lactulose were no different than controls for induction of remission, and fructo-oligosaccharides were no different than controls for maintenance of remission[166]. The overall certainty of evidence was very low, with larger studies needed to draw firm conclusions.

Vitamin D, omega-3, and glutamine

Statement 19.1: There is insufficient evidence to recommend vitamin D for induction therapy of IBD. [EL2] [Consensus 100%]

Statement 19.2: Omega-3 fatty acid [EL2] and glutamine [EL3] supplementation are not recommended for induction therapy in IBD. [Consensus 100%]

Statement 19.3: Omega-3 fatty acid and vitamin D supplementation are not recommended for maintenance of remission in Crohn's disease or ulcerative colitis. [EL1] [Consensus 100%]

A recent Cochrane review on the utility of vitamin D in active IBD, which assessed 22 RCTs with 1874 participants, could not draw any conclusions on clinical response for UC as the certainty of evidence was low. There were no data on efficacy for induction of remission in CD[167]. This review and four other meta-analyses consistently did not support vitamin D for maintenance of remission of IBD[167-174].

A Cochrane review of six studies assessed omega-3 fatty acids for induction of remission in IBD. One small study showed positive benefits, while others suggested minor benefits for secondary outcomes[175]. However, small sample sizes and poor study quality prevented definitive conclusions. A more recent systematic review of 83 RCTs [41 751 participants], including 13 studies in active IBD, concluded that long-term supplementation with omega-3, omega-6, and total polyunsaturated fatty acids [PUFA] had minimal effect on IBD activity[168]. Meta-analysis data also did not support use of these supplements in maintenance of remission[167-174].

A systematic review of glutamine for treating active IBD analysed seven studies that used oral, enteral, or parenteral administration. The results showed no significant changes in anthropometry, biochemical parameters, or disease activity[176].

Although there are potential health benefits for optimising these nutrients, their impact on IBD disease activity has not been proven.

Dietary management of comorbidities and special conditions of IBD

Management of IBD involves more than controlling the inflammatory disease process. Diet plays an important role in treating complications of IBD and concerns unrelated to inflammation.

IBS-like symptoms

Statement 20.1: In adults with quiescent IBD and IBS-like symptoms, a low FODMAP diet is recommended to treat persistent gastrointestinal symptoms. [EL1] [Consensus: 100%]

Up to one-third of people with IBD in remission have IBS-like symptoms[177], which is approximately three-fold more than the general population.

A low FODMAP diet has the greatest efficacy for managing IBS compared with other dietary interventions[178] and is currently recommended in IBS management guidelines, if general lifestyle and dietary advice fail[179]. Two meta-analyses evaluated the efficacy of a low FODMAP diet for treatment of overall symptoms in adults with IBD in remission[112,180]. The most recent meta-analysis, which included four RCTs[181-184], showed that a low FODMAP diet improved overall gut symptoms compared with a controlled diet [RR: 0.47; 95% CI: 0.33–0.66][112]. Improvements were seen in people with CD and UC. There were no differences observed between IBS subtypes, which are classified according to predominant bowel habits. These data are supported by observational trials[185,186] and studies that showed a worsening of symptoms with FODMAP provocation on a background low FODMAP diet in the same population[187,188]. There are limited data on the effects of a low FODMAP diet in children with IBS-like symptoms[189]. Dietary guidance and monitoring with a dietitian are suggested for appropriate application and customisation to prevent ongoing over-restriction of diet.

Statement 20.2: In people with quiescent IBD and IBS-like symptoms, there is insufficient evidence to support the use of high-fibre foods, fibre supplements, or a low-fibre diet to treat persistent gastrointestinal symptoms. [EL3] [Consensus: 100%]

Fibre is the only other dietary component that has been assessed in people with quiescent IBD and persistent gastrointestinal symptoms. However, there are limited quality data supporting the efficacy of high-fibre foods[103,155], fibre supplements[146], and a low-fibre diet[101] in reducing IBS-like symptoms.

Strictures

Statement 21.1: While there are no data supporting a modified or low-fibre diet to manage stricturing Crohn's disease [CD], mechanism-based reasoning suggests that a diet low in bulking fibre in people with stricturing CD and obstructive symptoms might reduce the risk of developing intestinal obstruction. [EL5] [Consensus: 100%]

People with stricturing CD are at risk of intestinal obstruction. Obstructive symptoms typically include abdominal pain, nausea, vomiting, abdominal distension, absence of gas and stool passage, or combinations thereof.

It has long been convention to suggest a low bulking fibre diet to reduce the risk of intestinal obstruction. This is solely mechanism-based reasoning, utilising anecdotal associations of obstructive symptoms and intestinal obstruction with high-fibre foods. No studies have investigated the role of a modified or low-fibre diet in preventing intestinal obstruction. However, it is common practice to suggest a low bulking fibre diet in people with stricturing CD and obstructive symptoms. There is no consensus on the definition of a low bulking fibre diet; a proposed categorisation of food based on functional gastrointestinal effects[190] is detailed in Table 4. Although it is not common practice to

restrict fermentable and viscous fibres to reduce risk of intestinal obstruction, many foods contain a mix of fibre types [Table 4]. Dietary guidance and monitoring to customise dietary treatment and ensure nutritional adequacy are suggested, preferably with a dietitian experienced in IBD.

Statement 21.2: Exclusive enteral nutrition could be considered to treat inflammatory strictures in Crohn's disease. [EL3] [Consensus: 100%]

Two observational studies that included people with stricturing CD who completed 12 weeks of EEN revealed a decrease in bowel-wall thickness and an increase in luminal cross-sectional area[25,191], indicating that EEN could reduce risk of intestinal obstruction in people with inflammatory strictures. This is consistent with retrospective studies. One case-control study revealed that 13/51 [25%] participants treated with at least 2 weeks of pre-operative EEN avoided their planned surgery, compared with participants who received no nutrition support, who all proceeded to surgery[192]. Another study of 87 participants with complicated CD [48 with stricturing disease] revealed that participants who received 4 weeks of EEN were 4-fold less like to require surgery[26]. There are limited quality data supporting the use of EEN to prevent intestinal obstruction.

Preoperative nutritional care

Statement 22.1: Exclusive enteral nutrition provided for at least 2 weeks might reduce the inflammatory burden in people with Crohn's disease who are awaiting surgical intervention and might prevent the planned surgery. [EL4] [Consensus: 96%]

Many people with IBD will require surgery during their disease course. Provision of nutrition support preoperatively is recognised to improve postoperative outcomes and is incorporated in protocols for colorectal surgery[193]. Research assessing the role of preoperative nutrition on clinical and surgical outcomes in people with IBD have been limited mostly to people with CD who have received EEN.

Preoperative EEN for at least 2 weeks can reduce the disease burden[194] and therefore reduce the extent and even requirement for surgery[26,192,195,196].

Statement 22.2: To reduce risk of postoperative complications in people with Crohn's disease who are awaiting surgical intervention, provision of preoperative nutrition is recommended, with most evidence supporting 4 weeks of exclusive enteral nutrition [EEN]. [EL3] A minimum of 10 days of oral nutritional supplementation might be considered if EEN is not feasible. [EL4] [Consensus: 100%]

A meta-analysis of five retrospective and observational trials that evaluated the effect of preoperative nutrition compared with standard care without nutrition support in people with CD undergoing surgery showed that at least 4 weeks of EEN or TPN reduced the risk of postoperative complications [OR: 0.26; 95% CI: 0.07–0.99][197]. A sub-analysis indicated that this reduced risk was largely associated with EEN[196,198]. Subsequent retrospective and observational trials on EEN for at least 2 weeks have supported this finding[195,199,200]. This included retrospective studies in children and adults with CD undergoing surgery, indicating that even PEN reduced the risk of postoperative complications compared with no nutritional support[194,201,202]. One of the studies provided 10–14 days of nutrition support as part of a perioperative home management programme, which also involved nutrition screening, endurance training, pre- and postoperative drug review, and patient education[202]. Furthermore, limited observational data suggest that EEN leads to a reduction in inflammatory markers, such as cytokines[203,204], CRP[192,198-200,205], and CD clinical disease activity scores[195]. Although one comparative trial showed no differences in postoperative complications from EEN compared with no nutritional support, EEN was given only to high-risk patients in this trial[206]. Since this meta-analysis, there has been limited-quality evidence supporting the use of PN over standard care for the

prevention of postoperative complications[207]. Clinical decisions on type and duration of preoperative nutrition depend on timing of surgery, clinical risk, access to a dietitian, other dietary requirements [e.g. for malnutrition, obesity, strictures, pregnancy], and preferences of the person with IBD.

Statement 22.3: There are insufficient data to define the optimal preoperative nutrition care in reducing risk of postoperative complications in people with ulcerative colitis. [EL4] [Consensus: 100%]

There are limited data on preoperative nutrition in people with UC. A retrospective study in people with UC undergoing surgery showed similar rates of postoperative complications, excluding line infections, in those provided EEN compared with PN[208]. However, another trial in people with acute severe UC on corticosteroids who were randomised to receive either EEN or PN showed that EEN was superior to PN in preventing postoperative infection in a subset of 20 patients who had undergone colectomy[209].

Intestinal resection

Statement 23.1: There are insufficient data to support a low-fat diet for the treatment of bile-acid diarrhoea in people with Crohn's disease who have undergone intestinal resection involving the terminal ileum. [EL4] [Consensus: 100%]

There is no specific dietary advice in the setting of intestinal resection and re-anastomosis. However, in people with CD who have undergone resection involving the terminal ileum, there is an increased risk of ileal malabsorption of vitamin B12 and bile acids, with subsequent development of vitamin B12 deficiency and type I bile-acid diarrhoea, respectively. Assessment and correction of vitamin B12 deficiency is described under *Nutritional assessment and optimisation for IBD*.

Bile-acid diarrhoea is caused by the dysregulation of bile-acid recycling within the enterohepatic circulation, leading to delivery of unabsorbed bile acids to the colon where they increase stimulating fluid, mucus, or sodium secretion[210]. Data on the role of diet in treating bile-acid diarrhoea in people with CD after an ileal resection are limited. An observational trial of EEN with a low-fat elemental formula for 8–15 days in 6 patients [4 with CD] with bile-acid diarrhoea following an ileal resection led to reduced faecal bile-acid output, weight, and frequency[211]. Whether the improvement in bile-acid diarrhoea was due to the low-fat composition or another component of the EEN formula is unknown. Two other observational studies of patients with bile-acid diarrhoea in the absence of IBD, with or without bile-acid sequestrants, revealed improvements in gastrointestinal symptoms with a low-fat diet[212,213]. Given the chronic nature of bile-acid diarrhoea, use of ongoing EEN is not feasible.

Statement 23.2: In people with Crohn's disease who have undergone intestinal resection and have a history of calcium oxalate nephrolithiasis, mechanism-based reasoning suggests that adequate hydration, increased dietary calcium, supplementary calcium citrate, a low oxalate diet, or combinations thereof might reduce oxalate absorption and oxalate stone formation. [EL5] [Consensus: 100%]

Calcium oxalate nephrolithiasis is over 2-fold more common in people with CD following intestinal resection than those without prior intestinal surgery[214].

A case-control study revealed that in patients with CD, two-thirds of whom had undergone an ileal resection, had higher intestinal absorption and urinary excretion of oxalate compared with healthy controls[215]. However, there were no studies specifically in people with IBD assessing dietary treatment of oxalate stones. Mechanism-based reasoning suggests that adequate hydration, increased dietary

calcium, supplementary calcium citrate, and a low oxalate diet will reduce oxalate absorption and oxalate stone formation. This is supported by trials assessing a low-oxalate diet in those with idiopathic hyperoxaluria[216].

Stomas

Statement 24: In people with IBD and an ileostomy, fibre intake could increase ileostomy output, but likely not above what is considered normal volume. [EL3] [Consensus: 100%]

Many people with IBD will require a temporary or permanent stoma. Diet may influence the content of stoma bags.

The impact of dietary fibre on stoma output has been investigated in six randomised controlled cross-over trials, predominately in people with UC with an ileostomy, although these trials included small numbers and were not sufficiently blinded. Compared with diets with < 20 g dietary fibre, a diet containing ≥ 28 g dietary fibre from fruits, vegetables, grains[217,218], inclusion of oat-bran bread[219], rye bread[220], brewer's spent grain[221], or potato or kidney bean flakes[222] increased ileostomy total and dry weight. However, all ileostomy outputs remained within what is generally accepted as normal volume. No other symptoms were examined. In two similar cross-over trials in people with IBD and an ileostomy, fibre supplementation with 3.3 g of psyllium husk and 5 g guar gum, but not psyllium seed, provided three times daily increased wet ileostomy output compared with no fibre supplementation[223,224]. Guar gum doubled ileostomy output, reduced output viscosity, and increased stoma bag emptying frequency compared with usual diets[224], although background fibre intake was not evaluated.

Survey data indicate that constipation, defined as the absence of stool evacuation ≥ 2 days with abdominal discomfort, flatulence, and painful passage of stool, is common and occurs in up to 29% of people with a colostomy, although only a small proportion of participants surveyed had IBD[225,226]. Lifestyle modification counselling, including recommending 20–35 g/day fibre and 2–3 L/day fluids led to symptom resolution in 50–60% of people with a colostomy and constipation, although this did not benefit the small sample of people with IBD[225,226]. No contraindications for fibre or fluid were identified[225,226], and it is common practice to encourage adequate fibre and fluid intake as part of a healthy diet.

Ileal pouch-anal anastomoses

Statement 25.1: In people with ulcerative colitis and an ileal pouch-anal anastomosis, fermentable fibre might improve pouch function but there is insufficient evidence to support non-fermentable fibre to improve pouch function. [EL4] [Consensus: 100%]

Many people with UC and an ileal pouch-anal anastomosis [IPAA] associate certain foods with a worsening of symptoms and restrict their diet[227]. However, data informing the role of diet in pouch function, pouchitis development, and pouchitis treatment are limited.

Pouch function: Fibre directly or indirectly has been the most studied for its effect on pouch function in people with UC and an IPAA. There may be symptomatic benefits from fermentable fibre [Table 4], as two placebo-controlled crossover studies showed that the fermentable fibre supplements inulin[228] and fructo-oligosaccharides[229] improved clinical disease activity scores and reduced stool frequency. Dietary sources of fermentable fibre may act similarly, as suggested by an open-label study showing improved pouch function in 6/12 symptomatic patients who were provided a specific diet to increase oligosaccharides and to regulate total and sulphur-containing dietary protein[230]. Assessment of the

inverse [via reduction in dietary fermentable fibres] in an open-label study of a low FODMAP diet in 5 patients showed variable results[231]. There is no evidence of a discernible effect of non-fermentable fibre on pouch function, as methylcellulose did not provide symptomatic benefit in an open-label crossover study[232].

Statement 25.2: In people with ulcerative colitis and an* ileal pouch-anal anastomosis*, adherence to a Mediterranean diet and daily fruit intake might be associated with a reduced risk of pouchitis development. There are insufficient data on diet in treatment of pouchitis. [EL4] [Consensus: 100%]

Pouchitis development: The impact of specific nutrients, food groups, and dietary patterns on pouchitis development has been assessed. The only factor associated with previous pouchitis was oligosaccharide intake, as shown in a cross-sectional study[233]. Lower fruit consumption is associated with pouchitis development in observational and case-control studies[234,235]. While a prospective cohort study in participants with IPAA did not reveal any specific dietary pattern associated with pouchitis development[236], an observational study associated adherence to a MD with lower faecal calprotectin levels[237]. This associated benefit did not clearly reduce risk of pouchitis development over 8 years of follow-up[237].

Pouchitis treatment: EEN may reduce bowel frequency and clinical disease activity scores, as shown in an open-label study of 7 people with pouchitis[238], although clinical symptom improvement from EEN might not be consistent[239]. Improvements in a modified clinical disease activity score were demonstrated in 15 people with pouchitis following the CDED over 24 weeks, although 7 people withdrew from the study before completion[240].

No improvement of bowel frequency was seen with green tea polyphenol in a retrospective study of people with pouchitis[241].

Short-bowel syndrome

Statement 26.1: In people with Crohn's disease and short-bowel syndrome, isotonic high sodium oral rehydration solutions might help maintain good hydration. [EL4] [Consensus: 100%]

Studies assessing the role of oral dietary therapies in people with short-bowel syndrome [SBS] or intestinal failure, specifically in CD, are lacking. As such, clinical practice in people with CD and SBS is usually based on research in cohorts that did not include people with CD or included people with SBS due to CD and other aetiologies. The role of oral rehydration solutions, macronutrient absorption, and glutamine supplementation has been investigated. PN strategies in people with CD and SBS are beyond the scope of this consensus.

Dehydration, both from reduced absorption and increased losses from diarrhoea, is common in people with SBS. Isotonic oral nutritional solutions are often used to enhance fluid absorption. Various oral rehydration solutions have been tested in people with CD and SBS, albeit mostly in small series or case reports, with a focus on short-term physiological studies. Isotonic high sodium oral rehydration solutions may help to replace luminal sodium losses in SBS[242-248].

Statement 26.2: In people with Crohn's disease and short-bowel syndrome, a diet with a higher fat:carbohydrate ratio containing medium-chain triglycerides might decrease energy losses in those with colon in continuity. [EL3] [Consensus: 100%]

Macronutrient absorption has been assessed in people with SBS with and without a colon. In those with a colon, unblinded crossover studies revealed that faecal energy losses were less after following a diet with a higher fat:carbohydrate ratio[249,250]. However, there were conflicting findings on the

differences being due to fat versus carbohydrate excretion. These results may be because of a difference in those who had fat maldigestion or malabsorption, which is common with SBS and poses a risk of deficiencies in fat-soluble vitamins and essential fatty acids[251]. The type of fat ingested may be of importance, as indicated in a randomised crossover study of 7 people with SBS and a colon, whereby inclusion of medium-chain compared with only long-chain triglyceride consumption, provided in both food and enteral nutrition, led to improved energy absorption[252]. Medium-chain triglycerides can be absorbed directly in the small bowel without requiring micelle formation and the presence of bile. In those without a functioning colon, the fat:carbohydrate ratio did not affect absorption in several studies[250,252,253]. Type of fat matter also did not affect absorption, as shown in another crossover study of 7 people with a high jejunostomy that compared an elemental diet containing medium-chain triglycerides to a polymeric formula containing only long-chain triglycerides[253].

Statement 26.3: In people with Crohn's disease and short-bowel syndrome, oral or subcutaneous glutamine does not improve markers of intestinal growth, function, and adaption. [EL3] [Consensus: 100%]

Supplementation [oral or subcutaneous] with the amino acid glutamine, often in combination with growth hormone, has been examined for possible benefits on intestinal growth, function, and adaption. Double-blinded, placebo-controlled crossover studies have shown no convincing effects on intestinal morphology, gastrointestinal transit, D-xylose absorption, or stool losses. However, the studies had small sample sizes and a mix of those with and without a colon[254-256].

Orofacial granulomatosis

Statement 27.1: A cinnamon- and benzoate-free diet might assist in the management of perioral manifestations of Crohn's disease, within the spectrum of orofacial granulomatosis, to reduce lip and oral inflammation and lip enlargement. There is insufficient evidence to support other dietary restrictions, although exclusive enteral nutrition might have a role. [EL4] [Consensus: 96%]

Orofacial granulomatosis [OFG] is an uncommon condition characterised by lip swelling that may have associated perioral erythema and intra-oral changes [ulceration or fissuring]. While OFG can be isolated, some individuals also have or will subsequently develop CD. Given the low prevalence of OFG, most research on the role of diet as treatment has provided low-level evidence, and only short-term outcomes have been reported.

The most commonly researched diet in OFG has been a cinnamon- and benzoate-free diet. In a retrospective study of 32 people with OFG, of whom 9 also had inflammation identified on ileocolonoscopy, a large proportion had improved lip and oral inflammatory activity after following the diet for 8 weeks[257]. The findings of this study were consistent with three single case reports that showed reduced gingival inflammation and enlargement following several weeks of a cinnamon-free or benzoate-free [or both] diet[258-260].

Restriction of phenolic acid and monosodium glutamate has also been examined. A low phenolic acid diet led to improved symptoms in 7/10 people with OFG [1 person had concurrent CD][261] and two case reports of people with isolated OFG achieved responses after exclusion of monosodium glutamate[262,263]. A 6-week course of EEN with an elemental formula in a series of 22 children with OFG, [12 with concurrent CD] led to improved lip appearance and clinical disease activity scores in those with and without concurrent CD[264].

Statement 27.2: Allergy testing to predict dietary response for orofacial granulomatosis may not be valuable. [EL3] [Consensus: 96%]

Many conditions that affect the orofacial region have an allergy-basis, including the proposed aetiology of OFG. Indeed, 73% of 264 people with OFG within a large cohort of people with oral diseases had urticaria on allergy patch testing, with more than half reacting to benzoic acid, 38% to cinnamaldehyde, and many to related food additives, perfumes, and flavourings[265]. Subsequent dietary restrictions resulted in subjective improvements in more than a third of those with OFG[265]. In contrast, there were variable results on the predictive value of allergy testing for dietary response[266].

Perinatal nutrition

The nutritional management of women with IBD and their infants throughout the prenatal, antenatal and infant periods was covered in a 2023 ECCO consensus on perinatal care in IBD and therefore has not been revisited within this consensus[267].

Nutritional assessment and optimisation for IBD

Consensus evaluation of the evidence for nutritional assessment and optimisation for IBD is outlined as anthropometric, micronutrient, and dietary approaches.

Anthropometry

Statement 28: Malnutrition and sarcopenia are negatively associated with clinical outcomes in IBD. Screening to identify the need for nutritional assessment is recommended. [EL3] [Consensus: 96.6%]

Malnutrition [including both undernutrition and overnutrition] arises from an imbalance between nutrient intake and requirements, leading to abnormal body composition and diminished physical and mental function[268]. This imbalance can lead to nutritional deficiencies and sarcopenia, characterised by low muscle mass and decreased muscle strength or physical performance, and increased visceral fat associated with chronic disease, including cardiovascular and metabolic conditions. Malnutrition is highly prevalent in IBD, during active disease and remission[269], and is associated with increased hospitalisations, disease flares, need for surgery, and post-operative complications[270-278]. Furthermore, malnutrition and sarcopenia are correlated with disease activity and elevated levels of faecal calprotectin[277]. Identification of malnutrition involves using a validated screening tool that is both effective and easy to use. Nutritional assessment in IBD at diagnosis and in patients who are at risk of malnutrition is particularly important, necessitating the use of various anthropometric measurements and assessment of body composition [Table 5]. Body composition measurements, such as reduced muscle mass and excessive visceral adipose tissue, are independently associated with negative clinical outcomes[272,273,277,279-285]. A detailed nutritional assessment paradigm is presented in Figure 2.

Statement 29: Body mass index does not accurately represent body composition in people with IBD. Longitudinal assessment of nutritional status using body mass index in combination with assessments of body composition, muscle function, or both is recommended. [EL3] [Consensus: 93.3%]

Historically, nutritional assessment has relied solely on weight and body mass index [BMI]. However, these have limitations in nutritional assessment of people with IBD and are often misleading in people who are overweight and obese but are actually malnourished[271,286]. Weight and BMI also do not accurately predict body composition[286-288]. As sarcopenia is not exclusively linked to undernutrition and can occur in people who are overweight and obese, this can lead to undiagnosed sarcopenia in IBD. Additionally, body composition measurements reveal a high prevalence of nutritional risk in people with IBD in clinical remission that may not be captured by BMI alone[286,289]. Therefore, incorporating additional anthropometric measures to evaluate body composition and muscle function is important in clinical practice decisions for IBD management. The Global Leadership Initiative on Malnutrition [GLIM] includes reduction in muscle mass as one of the phenotypic criteria for diagnosing malnutrition[268]. According to GLIM, techniques such as dual-energy X-ray absorptiometry, bioelectrical impedance analysis, ultrasound, computed tomography, and magnetic resonance imaging are recommended for assessing muscle mass. Additionally, GLIM recommends grip strength as a measure of muscle function due to its practicality and accessibility, particularly since muscle function often declines more rapidly than muscle size, making grip strength a useful complementary tool[268].

Micronutrients

Anaemia, or reduction of haemoglobin, is common in IBD[290-292] and has a prevalence ranging from 6–74%, with two-thirds of patients presenting with anaemia at IBD diagnosis[290,293]. Anaemia leads to fatigue and negatively impacts patient QoL, cognitive functions, and IBD disease outcomes[290]. Anaemia in IBD is defined according to the World Health Organization criteria[294] [Table 6]. Anaemia is commonly caused by deficiencies in iron, folate, vitamin B12, or combinations thereof. Specific recommendations for iron, folate, and vitamin B12 monitoring in people with IBD and supplementation therapies for correction of deficiencies are described elsewhere[293,295] and are beyond the scope of this consensus.

Statement 30: Haemoglobin and iron status should be monitored in all people with IBD. [EL2] [Consensus: 100%]

The most common forms of anaemia in people with IBD are iron deficiency anaemia [IDA], anaemia of chronic disease [ACD], and anaemia from combined causes. In patients with IBD, the diagnosis of IDA and ACD is challenging as they often overlap, particularly as iron deficiency relates to the degree of inflammation, which is commonly problematic in chronic disease. In patients without clinical, laboratory, endoscopic, or radiological signs of active inflammation, serum ferritin < 30 µg/L indicates iron deficiency. In the presence of active inflammation [CRP > 10 mg/L], serum ferritin up to 100 µg/L indicates ACD. Transferrin saturation < 16% is diagnostic of IDA when inflammatory markers are not available[296]. If serum ferritin is 30–100 µg/L, a combination of iron deficiency and ACD is likely[293,295]. The initial screening for anaemia and iron deficiency should be performed in all patients with IBD through full blood count, serum ferritin, and CRP[293]. If anaemia is confirmed, red blood cell indices, such as red cell distribution width and mean corpuscular volume, reticulocyte count, differential blood cell count, serum ferritin, transferrin saturation, and CRP concentration should be measured. Based on the results and each patient's clinical situation, serum levels of vitamin B12, folic acid, haptoglobin, reticulocytes, lactate dehydrogenase, soluble transferrin receptor, creatinine, and urea could benefit clinical assessment. If the cause of anaemia is still unclear despite an appropriate extensive workup, advice from a haematologist may be necessary. Patients in remission or with mild disease activity should be monitored every 6–12 months; such measurements should be performed at least every 3 months for those with moderate or severe disease activity. Serum levels of vitamin B12 and folic acid should be measured at least annually in patients at risk [e.g. small-bowel disease or resection] or in the absence of thiopurine use in case of macrocytosis [Table 7][293,295,296]. The long-term effects of anaemia on IBD disease course are debated. However, pre-operative anaemia is associated with poorer surgical outcomes [i.e. a higher risk of postoperative morbidity and mortality and post-surgical complications][297]. A recent Italian multicentre study conducted on 5416 adults with IBD revealed that severe anaemia was linked to increased fatigue and poorer QoL. Despite this, only two-thirds of patients received adequate supplementation[290].

Iron deficiency is the most common micronutrient deficiency in patients with IBD[290]. Long-term iron deficiency can impact patient QoL and cognitive function, even in the absence of anaemia[292]. A systematic review of 39 studies conducted in paediatric IBD showed that iron deficiency is common, with iron deficiency in up to 95% of patients at diagnosis and up to 70% of patients continue to have iron deficiency after 2 years[296].

Statement 31: Serum folate should be monitored in people with IBD, as they are at risk of deficiency. [EL2] [Consensus: 100%]

Folate [vitamin B9] is required for DNA replication, metabolism of amino acids, and maturation of red blood cells. Folate is found in legumes and legume flours, green vegetables, wholegrains, offal, mussels, and folic acid fortified foods. Folate is absorbed in the duodenum and serum folate

concentration is an indicator of recent folate status and intake. Although there is no consensus on the specific serum folate level indicating deficiency, the risk of megaloblastic anaemia significantly increases below the serum folate cut-off value of 7 nmol/L[294]. Systematic reviews of studies on children and adults with IBD suggest that folate deficiency is rare[296,297] but that serum folate is significantly lower in those with IBD than controls[298]. Furthermore, patients who live in countries without mandatory folic acid food fortification [e.g. Europe and Asia] were more likely to have lower serum folate than those living in countries with mandatory fortification [e.g. United States, Africa][296,298]. Patients taking methotrexate should be supplemented with prophylactic folic acid as the drug inhibits folate uptake. Furthermore, to reduce the risk of neural tube defects, folic acid supplementation is recommended for at least 4 weeks preconception and continued for the first 12 weeks of pregnancy[267].

Statement 32: People with Crohn's disease [CD] who have had an ileal resection > 20 cm, people with IBD who follow a vegan diet, or both are at high risk of vitamin B12 deficiency and prophylactic supplementation could be considered. Monitoring of vitamin B12 in people with CD and ileal involvement is recommended [EL3] [Consensus: 100%]

Vitamin B12 is essential for cellular metabolism, DNA and nerve myelin synthesis, and maturation of red blood cells. Vitamin B12 is found only in animal products and some vitamin B12 fortified foods. Vitamin B12 is stored in the liver but prolonged inadequate dietary intake or poor absorption depletes liver stores. Vitamin B12 requires gastric-produced intrinsic factor to support its absorption in the terminal ileum. Therefore, patients with prolonged active ileal CD, ileal resections, or both are at increased risk of deficiency compared against those with colonic CD or UC[299,300]. Serum vitamin B12 is not a sensitive marker of deficiency[300] and clinical symptoms of deficiency overlap with those of active IBD, making the diagnosis of vitamin B12 deficiency challenging. Methylmalonic acid, with or without holotranscobalamin [the metabolically active form of vitamin B12], could be used as a more sensitive marker of deficiency[300]. Symptoms of vitamin B12 deficiency include fatigue, diarrhoea, constipation, poor concentration, frequent infections, poor appetite, numbness in extremities, and depression. During conception and pregnancy, low serum vitamin B12 is associated with adverse pregnancy outcomes[267]. Besides those with active ileal disease or resection, the prevalence of serum vitamin B12 deficiency in IBD is rare[296,297,299,300]. A meta-analysis revealed that people with CD and ileal resection > 20 cm were predisposed to vitamin B12 deficiency[301]. A multivariate analysis of a large retrospective observational study [n = 381] revealed that the odds of vitamin B12 deficiency were higher in patients with ileal resection > 20 cm [OR: 6.7; 95% CI: 3.0–15.0] or active ileal disease [OR: 3.9; 95% CI: 2.2–6.9][300]. A systematic review found that those who follow a vegan diet are also at high risk of deficiency due to low oral intake of vitamin B12[302]. Prophylactic supplementation could be considered for patients at higher risk of deficiency. Oral, sublingual, or intramuscular vitamin B12 improves status [Table 7], although those with ileal resections may not respond as well to oral supplementation[299].

Statement 33: Vitamin D insufficiency is common in people with IBD and may be associated with adverse disease outcomes. Monitoring of serum vitamin D is recommended [EL3] [Consensus: 100%]

Vitamin D, in conjunction with calcium, magnesium, and phosphate, is necessary to achieve and maintain optimal bone density. There are few dietary sources of vitamin D [cod liver oil, fatty fish; Table 7]; synthesis of vitamin D from ultraviolet radiation on the skin is the major contributor to maintenance of adequate status. In IBD, prevalence of deficiency in some countries varies by season, skin pigmentation, or sun exposure behaviours [Table 7] and as such prophylactic supplementation could be considered[303-305]. While vitamin D deficiency is common both in the IBD and general population, optimal repletion strategies in those with IBD is not known. This is indicated by paediatric

trials suggesting that vitamin D supplementation does not preclude vitamin D deficiency[306,307] and a Canadian cohort study showing that pregnant women with IBD [n = 61] receiving vitamin D supplements were more likely to be deficient than pregnant women without IBD [n = 574][308]. However, normalisation of vitamin D status may be associated with improved inflammatory markers, lower risk of hospitalisation and surgery, and fewer disease relapses in both adults and children with IBD[170,172,303].

Statement 34: Monitoring of micronutrient levels should be considered in people with IBD. Ideally, this should be performed during clinical and biochemical remission, because levels may be falsely altered during active disease [EL2] [Consensus: 100%]

Although limited data are available, active IBD is linked to low serum levels of vitamins and minerals[297], including iron, zinc, selenium, copper, manganese, folic acid, thiamine, and vitamins D, B12, A, C, E, K, and B6[297]. Many of these nutrients are acute-phase reactants, making interpretation of findings challenging in the presence of elevated CRP[291]. Micronutrient deficiencies are more prevalent in hospitalised patients and in those with active IBD and long-standing CD[297,309,310]. However, some reports suggest that micronutrient deficiencies persist even during disease remission[311,312]. A systematic review and meta-analysis revealed zinc deficiency in approximately 50% of adults with IBD, particularly CD[313], and commonly at diagnosis[296]. Limited data are available on the clinical outcomes of zinc deficiency in IBD. Some studies suggest poorer clinical outcomes [higher risk of surgery and hospitalisations] in zinc-deficient individuals with IBD than in those with normal zinc levels[297,314,315]. Similar data were reported in children[291,316]. The efficacy of zinc supplementation is poorly understood. Small studies suggest that different zinc formulations may improve serum zinc levels and possibly clinical outcomes[297,317].

Although data on vitamins A, E, and C and selenium are limited, deficiency of these micronutrients seems to be rare[296].

Overall, minimal data are available on the impact of trace elements on the course of IBD. Recently, a single-centre, randomised, double-blind, placebo-controlled trial evaluating the efficacy of an over-the-counter multivitamin and mineral supplement to reduce the risk of infection in 320 non-deficient patients with IBD in remission receiving immunomodulators, biologics, or both found no significant differences between supplemented and placebo groups over 24 weeks[318].

Diet

Beyond nutritional status of people with IBD, problems with eating behaviour are common and can lead to nutritional inadequacies and excesses, in addition to negative effects on social eating and enjoyment. Dietary assessment is a core skill of all dietitians, including identification of issues with eating behaviours followed by counselling on corrective strategies and monitoring behaviour change. However, all clinicians can screen for dietary abnormalities. Table 8 describes such behavioural alarm features that may indicate dietary inadequacies, excesses, or both that warrant referral to a dietitian.

Gaps in dietary intake have been assessed in patients with CD and UC. Dietary components that require particular attention in an IBD population include total energy, iron, vitamin C, calcium, and dietary fibre.

Statement 35: Dietary intake is generally lower in active disease than in remission in people with Crohn's disease or ulcerative colitis [EL2]. Assessment of dietary intake in people with IBD is recommended, as dietary restriction is common, often resulting in inadequate energy and nutrient intake. [EL3] [Consensus: 100%]

Energy intake, as estimated by dietary recall or food diaries, is lower in adults and children with CD and UC when compared with recommended intake[319-321] and controls, regardless of disease activity[322-325]. Intake for dietary fibre, vitamin A, vitamin D, vitamin K, thiamine, folate, calcium, magnesium, iron, zinc, phosphorus, and selenium are often inadequate for persons with CD or UC[323,324,326-334]. Dietary assessment in children is of particular importance to ensure dietary recommendations are met and to prevent growth deficits[322,325,335,336].

While dietary restriction is common among all people with IBD, specific attention should be given to people with IBD and a stoma or an IPAA due to high rates of self-perceived intolerances[227]. Indeed, survey data show that almost half [15/32] of people with IBD and a newly-established or well-established ileostomy avoided certain foods to prevent increased output, gas, odour, blockage, visible food in the stoma bag, or pain[337]. Similar food restrictions were identified in people with an IPAA[227]. As with other populations applying dietary restrictions, there may be a higher risk of micronutrient deficiencies, as suggested by a sub-analysis of 12 people with an established ileostomy not meeting certain reference nutrient intakes[337]. Restrictive eating behaviour is of particular concern in people with IBD, as a fear of eating, food avoidance behaviour, or disordered eating patterns are more frequently encountered than observed in the general population; dietary assessment may help identify those at risk[338,339].

Management of increased nutritional requirements and correction of micronutrient deficiencies in women with IBD is reported elsewhere[340].

Statement 36: Dietary intake of iron and vitamin C is often low in people with IBD. Assessment of iron and vitamin C intake is recommended. [EL3] [Consensus: 93.1%]

In the absence of IDA or ACD, adequate dietary iron intake helps maintain iron stores[341]. Dietary iron intake [high concentrations found in red meat; Table 7] is usually lower in people with active IBD, including children and pregnant women[329,336,340,341] than in healthy persons, although there is inconsistency in whether dietary iron intake increases in people in remission[325,334,342]. Given the higher iron requirements, cross-sectional studies found that girls[343] and women[321,332,344] of menstruating age are less likely than males to meet targets. Non-haem iron absorption is improved with dietary and supplementary vitamin C [dietary sources include fruit and vegetables; Table 7], however vitamin C intake is also lower in individuals with IBD compared to healthy people[329,331,332,334,336,341,345]. Given the low consumption of iron and vitamin C in people with IBD, referral for dietary assessment should be considered in those identified as deficient during quiescent disease, particularly for menstruating women and those on restrictive diets, including vegetarians and vegans. Recommendations for people unable to meet recommended iron intake from food alone and needing iron supplementation are specified elsewhere[293,295].

Statement 37: Dietary calcium intake is often below recommendations for optimal bone health in people with IBD. Assessment of calcium intake is recommended. [EL3] [Consensus: 100%]

Osteopenia and osteoporosis are prevalent in people with IBD due to inflammation and corticosteroid use, which increases bone resorption[346]. Adequate calcium intake is needed to promote bone mineralisation. Compared with healthy populations, calcium intake is lower during active and inactive disease in people with IBD, including children and pregnant women, and is also lower than the recommended intake for optimal bone health[321,325,328,329,331-336,340,343,344]. As milk products are rich in calcium, reduced calcium intake is even more prevalent in those who avoid dairy[331,334,344]. Referral for dietary assessment should be considered in people with IBD who restrict dairy intake.

Statement 38: Assessment of dietary fibre intake is recommended in people with IBD, as intake is often below recommendations. [EL2] [Consensus: 96.4%]

Dietary fibre is important for normal laxation, gut health, and disease prevention. Compared with healthy people, those with IBD often have lower dietary fibre intake[320,325,331,336,340,342,343,347-350], varying between 8.8–22.7 g/day[321,324,325,327,329,331,342,351,352]. This is well below most recommended guidelines of 25–30 g/day[309,320,321,324-327,331-333,340,343,344,346-349,351-354]. The data for adults and children are similar and disease phenotype or activity have minimal influence on intake.

Despite suggestions that fibre restriction may increase the risk of a flare in CD[347] or UC[350], many people with IBD minimise fibre to prevent a flare. The clinical practice of a targeted fibre restriction in people with IBD is indicated only for people with stricturing CD [see *Dietary management of comorbidities and special conditions of IBD*].

Where available, referral for dietary assessment of fibre should be considered in people with IBD who restrict fibre or plant-based foods [i.e. fruit, vegetables, whole grains, legumes, nuts, and seeds] or those with suspected poor-quality diets.

THE ROLE OF DIET IN IBD PREVENTION

Diet is a key environmental factor that plays a role in the development of IBD [Figure 1]. Identifying pre-disease dietary components that increase or decrease the risk of IBD development has been ascertained through epidemiological cohort studies that repeatedly assessed food intake via food frequency questionnaires or 24-h dietary recall in a large healthy population that was observed for several years or decades. Identifying people who develop illness, such as IBD, allows for assessment of baseline dietary risk factors. For the purpose of this review, only cohort studies conducted prospectively were assessed for consensus inclusion. This was done to minimise the inherent bias from data collected retrospectively.

Diet as prevention of IBD development

Prospective cohort studies have investigated associations between macronutrients, micronutrients, other food components, and more recently, dietary patterns, such as healthy eating patterns and classification for levels of food processing [Table 9]. The association between breastfeeding and IBD development in the offspring is also included [Table 9].

Food components

Statement 39: Increased docosahexaenoic acid [n-3] is associated with lower risk of Crohn's disease and ulcerative colitis [UC]. A high intake of arachidonic acid [n-6] is associated with UC. Increased oleic acid [n-9] and a high intake ratio of n-3:n-6 polyunsaturated fatty acids are associated with lower risk of UC. [EL2] [Consensus: 100%]

Several prospective cohort studies have assessed specific food components in the development of IBD.

Several studies have investigated the association between total fat, total long-chain fatty acids, and specific omega-3 [n-3], omega-6 [n-6], and omega-9 [n-9] long-chain fatty acids, in addition to intake ratios of n-3:n-6 and IBD development.

Total fat and long-chain fatty acids: The European Prospective Investigation into Cancer and Nutrition [EPIC], one of the largest and oldest European cohort studies, has broadly assessed the link between the intake of total fat and long-chain fatty acids and CD using matched controls[355]. No associations were found[355].

n-3: Only two studies assessed intake of total n-3 with the development of UC and CD, with neither study finding associations[356,357]. However, the individual n-3 fatty acids docosahexaenoic acid [DHA] and eicosapentaenoic acid [EPA], commonly found in fish and shellfish, were assessed in four cohort studies[355-358], with variable results. Two EPIC studies showed a reduced risk of UC development with DHA intake[357,358]. This included a study where DHA intake 0.13–0.80 g/day was associated with reduced risk of UC when corrected for total energy, cigarette smoking, and other long-chain fatty acids [OR: 0.17; 95% CI: 0.04–0.78][357]. The second study showed similar results [OR: 0.23; 95% CI: 0.06–0.97, adjusted for various factors including age, gender, total energy, and the other fatty acids][358]. Another study assessing risk for CD where consumption of 0.31–1.49 g/day DHA [corrected for smoking, total energy, BMI, dietary vitamin D] and the other fatty acids was associated with an over 16-fold reduction in the risk of developing CD [OR: 0.06; 95% CI: 0.01–0.72][355]. EPA was not associated with UC risk[357].

n-6: n-6 arachidonic acid, found in animal-derived foods, was associated with developing UC in a Danish cohort [OR: 4.16; 95% CI: 1.56–11.04, adjusted for gender, smoking, aspirin and NSAID use, and other fatty acids][359] and within the UK cohort of EPIC [OR: 6.09; 95% CI: 1.05-35.23]; adjusted for total energy, aspirin use, cigarette smoking, social class, and the dietary intakes of linoleic acid and n-3 fatty acids[360].

n-9: Consumption of oleic acid, a large constituent of olive oil, was associated with a lower risk of UC (OR 0.03 [0-0.56])[360].

n-3:n-6 ratio: In the all-female Nurses' Health Study [NHS] I, the risk associations of n-3:n-6 ratio for UC and CD were explored[361]. Compared with the lowest quintile, women in the highest quintile of n-3:n-6 PUFA intake had a lower risk of developing UC but not CD [HR: 0.69; 95% CI: 0.49–0.98, adjusted for age, cohort, smoking, energy intake, BMI, oral contraceptive and postmenopausal hormone therapy use, and NSAID and aspirin use][361]. Although no associations were seen for n-3:n-6 ratios, in

a subsequent study including NHS I and NHS II, integration of targeted genetic profiling revealed a significantly reduced risk of UC from n-3:n-6 ratio intake in four genotypes (GG genotype of rs4646904 single nucleotide polymorphism [SNP], GT/TT genotype of rs1290617 SNP, GG genotype of rs3794987 SNP of CYP4F3 locus, and T-genotype of rs3834458 SNP of FADS2 locus). No such protective effect was seen in genotype analysis for CD[356].

Statement 40: Increased dietary fibre intake is associated with lower risk of IBD, particularly from fruit and high-fibre bread for Crohn's disease and from high-fibre cereals for ulcerative colitis. [EL2] [Consensus: 96.7%]

Three studies investigated dietary fibre intake in the development of CD and UC[362-364]. The impact of dietary fibre on all forms of IBD was investigated in one cohort, which showed a reduced risk of developing IBD in all quintiles of fibre intake in an adjusted model [highest quintile HR: 0.74; 95% CI: 0.58–0.93][364]. The reduced risk was largely associated with fibre sourced from bread and cereal [highest quintiles- bread HR: 0.85; 95% CI: 0.75–0.96, cereal HR: 0.84; 95% CI: 0.71–0.99]. When separated into IBD phenotypes, there were consistently no associations found between dietary fibre intake and UC development[362,363]. A sub-analysis considering food sources of fibre found cereal fibre lowered the risk of developing UC [HR: 0.79; 95% CI: 0.64–0.98][364], but this was inconsistent with another study[362].

Total fibre intake reduced risk of developing CD in two of three cohort studies[362,364]. Fibre intake ≥ 24.3 g/day, comparable to most dietary guidelines, and particularly sourced from fruit [5.8 g/day] reduced risk of CD, whether corrected for age alone [fibre HR: 0.53; 95% CI: 0.35–0.80, fruit fibre HR: 0.51; 95% CI: 0.35–0.76] or for age and other factors [fibre HR: 0.59; 95% CI 0.39–0.90, fruit fibre HR: 0.57; 95% CI: 0.38–0.85][362]. Data from the UK biobank found a similar association with total fibre and fibre from fruit. Fibre sourced from bread was also associated with a lower risk of developing CD [total fibre HR: 0.48; 95% CI: 0.32–0.72, fruit fibre HR: 0.79; 95% CI: 0.64–0.98, bread fibre HR: 0.75; 95% CI: 0.57–0.98][364]. These results were adjusted for intake of various foods and food groups [fruit, vegetables, bread, cereal, fish, and meat] and other variables. EPIC found no association with fibre or fibre sources and CD[363].

Statement 41: Vitamin D intake is not associated with risk of Crohn's disease; however, higher vitamin D intake is associated with a lower risk of ulcerative colitis. [EL2] [Consensus: 93.3%]

Vitamin D plays an important role in the regulation of calcium and bone health and may have anti-inflammatory effects[365]. In IBD, this concept was suggested by one study showing a negative association between plasma vitamin D concentrations and CD onset, although this was not the case for UC[366] or in studies of similar design[367], including a genome-wide case-control study of 34 915 Europeans [CD: n = 12 194; UC: n = 12 366][368].

Four prospective cohort studies investigated the association between either supplementary or dietary vitamin D intake and risk of CD or UC development. No study reported an association between vitamin D intake and CD[367,369,370], although only one study revealed a lower risk of UC [HR: 0.64; 95% CI: 0.37–1.10, *p* for trend 0.04)[366]. Although intake for each quartile was not reported in this study, for each 100 IU/day increase in total vitamin D intake there was a 10% reduction in UC risk [multivariate HR: 0.90; 95% CI: 0.83–0.98, *p* = 0.02]. Calcium intake had no association with risk of CD or UC in one study[370].

Statement 42: There is an association between higher zinc intake and lower risk of Crohn's disease, but not ulcerative colitis. [EL2] [Consensus 100%]

Zinc plays many essential functional roles, including immunity, wound healing, and blood clotting. People with chronic diarrhoea, including those with IBD, are at greater risk of zinc deficiency. Vegetarians and vegans are at particular risk of zinc deficiency as animal products, such as meat, fish, and cheese are rich in zinc. The specific role of zinc adequacy and deficiency has not been investigated in risk of IBD development overall. However, zinc intake was associated with a lower risk of CD in one study [HR: 0.74; 95% CI: 0.50–1.10, *p* for trend 0.003][371]. This association remained when assessing zinc intake from diet alone[371]. Compared with individuals consuming < 8 mg/d zinc, those consuming 8–16 mg/day and > 16 mg/day zinc had a reduced risk of CD [HR: 0.69; 95% CI: 0.44–1.08 and HR: 0.52, 95% CI: 0.32–0.86, respectively]. In contrast, there was a positive association between zinc intake and UC risk, however, despite a significant trend [*p* = 0.007 across quintiles], the 95% CIs for all quintiles crossed 1.00 and therefore no consistent association was found[371]. This finding was consistent in another cohort study [reduced risk of CD HR: 0.12; 95% CI: 0.02–0.73, *p* for trend 0.02] showing no association between zinc intake and UC[372]. The intake in each tertile and the intake required to reduce risk were not reported. Mean follow-up in this cohort was only 2.3 years and the median time from last dietary analysis to disease diagnosis was < 1 year; thus, latent undiagnosed disease may have impacted the results.

Statement 43: Intakes of iron, folate, and vitamin B12 are not associated with risk of Crohn's disease or ulcerative colitis. [EL2] [Consensus: 96.6%]

Iron plays important roles in energy production and supporting immunity. Deficiencies can lead to anaemia and fatigue. Iron deficiency is common in established active IBD, due to inflammation and associated impaired absorption and increased losses [see *Nutritional assessment and optimisation for IBD*]. In the general population, iron deficiency is also common in menstruating women and particularly in vegetarians and vegans, as meat is a rich dietary source of haem iron, which has higher bioavailability than plant sources of iron. Two prospective cohort studies assessed the association of iron intake and did not reveal a link between iron intake and developing CD or UC[369,373]. One of the studies considered intake of both total dietary iron and dietary haem iron in an all-female population[373]. The model was adjusted for menopausal status but not for dietary restrictions [e.g. vegetarian or vegan].

Deficiencies in folate, vitamin B12, or both can also lead to anaemia, but evaluation of serum inadequacies and pre-disease intake has not been assessed in high-quality prospective cohort trials. However, data from a Mendelian randomisation study from several European genome-wide association studies [CD: n = 12 194; UC: n = 12 366] found no association between folate status and IBD, CD, or UC[368]. Although there were greater odds of CD in those with high vitamin B12 status [OR: 1.10; 95% CI: 1.00–1.21, *p* = 0.04], adjustment for an outlier single nucleotide polymorphism was thought to have biased the risk estimate.

Statement 44: Intakes of vitamins A, C and E are not associated with risk of ulcerative colitis. [EL2] [Consensus: 96.7%]

As part of the EPIC cohort exploring the intake of numerous micronutrients and development of UC, no association was identified between intakes of carotene, retinol, vitamin C, or vitamin E and subsequent UC[369]. There was no investigation of the association with CD risk.

Statement 45: Polyphenol intake is not associated with risk of Crohn's disease or ulcerative colitis. [EL2] [Consensus: 96.4%]

Through a range of mechanisms, including antioxidant activity, polyphenols are non-essential nutrients that may have protective effects against development of many chronic diseases. EPIC explored risk associations with intake of numerous non-nutrient bioactive polyphenols[374]. There were no associations between risk of CD and intake of total polyphenols or total or individual flavonoids. There was a reduced risk with higher flavone intakes, which was significant for the trend across quartiles [p = 0.03]. However, only the third quartile was statistically significant [OR: 0.33; 95% CI: 0.15–0.69, in only 12 cases] but not the fourth [OR: 0.61; 95% CI: 0.28–1.3, in 30 cases, lignans or total or individual phenolic acids][374]. There was a reduced risk of CD with higher intake of resveratrol [OR: 0.40; 95% CI: 0.20–0.82, p for trend 0.02][374]. Individual intake corresponding to the different quartiles associated with risk were not reported. There were no associations between intake of total polyphenols or intake of total or individual polyphenol classes with UC.

Dietary patterns

Rather than assessing the risk of IBD development with individual nutrients, consideration of more comprehensive dietary intake, either through food groups or the combination of food groups in dietary patterns, may be more indicative of interactions of foods as eaten together and possibly overall diet quality. Food groups describe the categorisation of foods sharing similar nutritional properties, such as dairy [consisting of milk, yoghurt, and cheese, which are rich in calcium and protein]. The removal of entire food groups with the specific intent of mitigating risk of IBD presents safety concerns and may potentially cause macronutrient or micronutrient deficiencies [or both], as in the case of dairy exclusion causing calcium deficiency. For this reason, any recommendation to exclude food groups from the diet should be done with caution. Several studies have investigated food groups and dietary patterns and their association with IBD development.

Statement 46: Healthy eating patterns are associated with lower IBD risk and therefore are recommended. These often include high consumption of fruits, vegetables, whole grains, nuts and seeds, legumes, and fish and low consumption of red meat, processed meats, and high-sugar foods and beverages. [EL2] [Consensus: 83.3%]

Food groups: A recent meta-analysis of cohort studies reported reduced risk for both CD and UC in adults with higher dairy intake. However, this reduced risk did not appear to persist when adjusted for alcohol, BMI, and energy intake[375]. A systematic review and meta-analysis reported inverse associations between fruit and vegetable intake with the development of CD, UC, and IBD[376], although many studies were case-control in design and findings were variable. Results from a large prospective cohort suggested that higher fruit intake was associated with a decreased risk of CD and no association with UC, but no adjustment was made for other dietary components or level of food processing[362]. Other prospective cohorts did not find an association[377,378]. There were no clear associations between intake of meat [including red, white, and processed meat][85,86,375,377,379,380], alcohol[381], and sweetened beverages[382,383] with development of IBD. Most published studies did not adjust for other dietary components or patterns, which limits generalisability.

Healthy dietary patterns: Studies utilising a dietary pattern approach have the advantage of capturing the complex interactions between foods and nutrients. In recent years, multiple studies that focused on healthy dietary patterns, using different definitions, have been conducted on large cohorts with long-term follow-up, showing a seemingly protective effect between adherence to a healthy lifestyle [including diet] and risk of IBD. The largest of these assessments included the NHS that translated modifiable diet and other lifestyle factors into a 'Healthy Lifestyle Score', which was higher for consumption of fruits, vegetables, nuts, dietary fibres, at least two servings of fish per week, nil-to-moderate alcohol consumption, low red meat consumption, maintaining a normal BMI, being a non-

smoker, and engaging in physical activity. The population attributable risk for a healthy lifestyle score was 61.1% [95% CI: 16.8–84.9%] for CD and 42.2% [95% CI: 1.7–70.9%] for UC[384]. These data were validated in three large European cohorts, including the EPIC and the Swedish Mammography Cohort/Cohort of Swedish Men cohorts, generating a population attributable risk of 48.8–60.4% and 56.3–46.8% for CD and UC, respectively[384]. Data from the UK biobank suggest adherence to a healthy lifestyle score, which was calculated based on smoking status, BMI, sleep duration, physical activity, low alcohol consumption, and a diet including high consumption of fruits, vegetables, and whole grains, consumption of ≥ 2 servings of fish/week and low consumption of red meat, processed meat, and refined grains, could mitigate genetic risk of IBD. Individuals with a high genetic risk who adhered to a healthy lifestyle experienced an almost 50% reduction in the likelihood of developing CD and UC[385]. This is consistent with studies using a UK-based dietary index showing that low nutritional quality scores are associated with increased risk of CD, although this was not the case with UC[386]. Adherence to a cardioprotective diet was also inversely associated with the development of CD and UC[387].

Commonly associated with health outcomes to prevent many chronic diseases, the MD has also been investigated in IBD prevention. Adherence to the MD was inversely associated with CD, but not UC, in large Swedish cohorts[388,389]. However, this was inconsistent in other populations, likely confounded by inconsistencies in its definition[390]. Moreover, other diet-quality scores, including healthy eating index, did not show consistent associations with CD or UC risk[390,391]. Only one cohort group has investigated early-life impact on diet, following participants from birth, indicating that a high-quality diet at 1 year of age was inversely associated with the risk of CD and UC, suggesting that even short exposure to dietary habits may have significant effects on IBD risk[392].

Unhealthy dietary patterns: As opposed to healthy eating patterns, the 'Western diet' is often the model of an unhealthy diet. While there is no clear definition of a Western diet, it is often characterised as a diet high in fat, sugar, salt, animal protein, and low in dietary fibres. Meta-analyses of cohort and case-control studies have shown inconsistent results when comparing high and low categories of Western diet patterns, with no associations demonstrated when only cohort studies are included[390,391,393,394].

Foods associated with circulating inflammatory markers: Another method of assessing dietary patterns is to apply indices developed through associations of known inflammatory markers. This was the strategy undertaken in the development of the Empirical Diet Inflammatory Pattern [EDIP] score, which identified 18 inflammatory and anti-inflammatory foods associated with CRP and circulating cytokines, although the foods selected for study inclusion were ambiguous. Conflicting results between populations prevented conclusions on the association between EDIP and IBD risk[377,395,396].

Statement 47: Ultra-processed food [UPF] intake is associated with the development of Crohn's disease but not ulcerative colitis. Intake of UPF should be limited consistent with healthy eating patterns. [EL1] [Consensus: 88.9%]

There is a growing interest in the role of food processing in the pathophysiology of IBD. Ultra-processed food [UPF], defined by the Nova classification system[397], typically contains at least five ingredients, extracted from foods or derived from processing of food components, and can contain food additives. Several prospective cohort studies have investigated food processing on the risk of developing CD and UC. A meta-analysis of these studies, comprising five large cohorts of > 1 million participants followed long-term across different geographical sites, showed that UPF intake was associated with an increased risk of subsequent CD [highest quartile 45–51% versus lowest quartile

13–21% of energy intake, OR: 1.71; 95% CI: 1.36–2.14][398]. Low-processed diets were inversely associated with CD risk [OR: 0.71; 95% CI: 0.53–0.94][398]. No associations were found with UC[398].

There are several challenges that need consideration when following the recommendation made for UPF. Firstly, there is some ambiguity on the definition of UPF, which encompasses a large range of food, thus identifying some UPFs can be challenging. Secondly, no clear 'safe' threshold for consumption of UPF has been identified, with some studies arbitrarily using 20% of energy intake or one serving/day as a reference. Thirdly, UPF differ greatly in their nutritional quality and there was no adjustment for diet quality in the published cohort studies. Due to these constraints, a recommendation of complete avoidance of UPF cannot be made. Recommendations for UPF intake are that they should be limited consistent with healthy eating patterns. Food availability and nutritional adequacy should not be compromised when giving recommendations for UPF intake, as complete avoidance is usually impractical.

Breastfeeding

Statement 48: There are well-recognised benefits of breastfeeding for the health of both the mother and infant but there is currently insufficient evidence supporting breastfeeding as a significant protector against subsequent IBD development. [EL2] [Consensus: 96.6%]

The health benefits of breastfeeding are well-recognised for both the infant and mother[399].

The role of breastfeeding in preventing later development of IBD in the infant has been assessed in many studies. Although three meta-analyses concluded a benefit from breastfeeding[400-402], this was mostly from case-control studies. Recommendations are based on four cohort studies to limit the biases inherent in case-control methodology.

The first cohort study that prospectively assessed breastfeeding and risk of IBD onset included data from British birth cohorts [MRC National Survey of Health & Development of 1946 and 1958 National Child Development Study] that followed participants to age 43 and 33 years, respectively, at the time of analysis[403]. No associations were identified for risk of CD or UC development with any breastfeeding compared with no breastfeeding[403]. The other collection of Scandinavian prospective birth cohorts from three countries [Norway 1999–2008, Sweden 1997–1999, Denmark 1996–2002] also found no difference between any duration of breastfeeding and development of CD or UC[404].

Early-life factors and subsequent risk of paediatric IBD in the Scottish population from 1981–2017 were explored, where no difference between exclusive breastfeeding or exclusive formula feeding at 6 weeks was seen[405].

FUTURE DIRECTIONS

This first ECCO consensus on dietary management of IBD supports the role of diet in improving clinical outcomes for patients with IBD. The procedure for determining when and what dietary assessments and treatments should be implemented is detailed in Figure 1. Many evidence gaps within our IBD dietary management paradigm were identified through the consensus process. Advancing diet-focused IBD research would be facilitated by increasing the numbers of research dietitians and an increased provision of targeted financial support. Proposed research topics to further knowledge and improve clinical care for patients with IBD are described in Table 10. Ongoing dietary research is crucial to provide optimal dietary management in IBD.

Conflict of interest statement:

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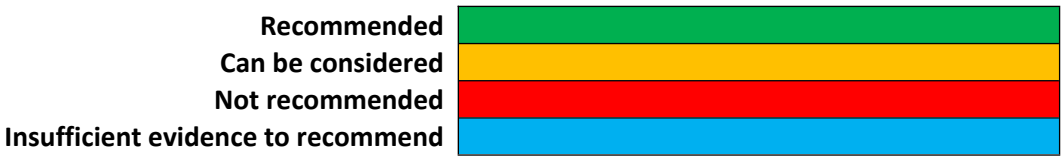
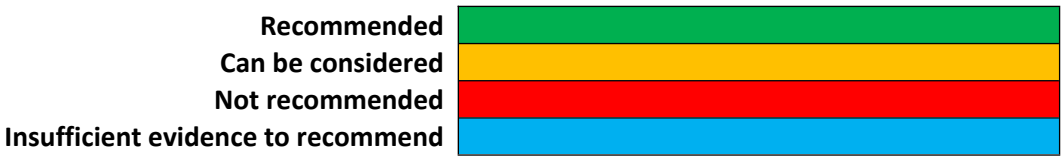
Table 1. Supporting evidence for dietary interventions to induce and maintain remission of Crohn's disease and ulcerative colitis



Dietary intervention	Crohn's disease		Ulcerative colitis	
	Induction of remission	Maintenance of remission	Induction of remission	Maintenance of remission
EEN				
PEN				
CDED + PEN				
CDED				
PN				
Mediterranean Diet				
SCD				
Reduction of red meat				
Cow's milk protein elimination				
BFM				
High fibre diet				
Low fibre diet				
Low FODMAP diet				
Food-specific IgG-guided diet				
Low microparticle diet				
CD-TREAT				
AIP				
IBD-AID				
4-SURE diet				
UCED				
Gluten-free diet				
Intermittent fasting				

EEN	Exclusive Enteral Nutrition
PEN	Partial Enteral Nutrition
PN	Parenteral Nutrition
CDED	The Crohn's Disease Exclusion Diet
SCD	The Specific Carbohydrate Diet
BFM	*Bifidobacterium*-fermented milk
FODMAP	Fermentable Oligosaccharides, Disaccharides, Monosaccharides, and Polyols
IgG	Immunoglobulin G
CD-TREAT	Crohn's Disease TReatment-with-EATing
AIP	Autoimmune Protocol
IBD-AID	Inflammatory Bowel Disease Anti-Inflammatory Diet
4-SURE	4 Strategies to SUlfide REduction
UCED	Ulcerative Colitis Exclusion Diet

Table 2. Supporting evidence for dietary supplements to induce and maintain remission of Crohn's disease and ulcerative colitis



Dietary supplement	Crohn's disease		Ulcerative colitis	
	Induction of remission	Maintenance of remission	Induction of remission	Maintenance of remission
Curcumin*				
QingDai*				
Curcumin and QingDai*				
Psyllium				
Germinated barley				
Resistant starch				
Oat bran				
Wheat bran				
Non-starch polysaccharides				
Mastiha				
Prebiotics				
Vitamin D**				
Omega-3				
Glutamine				

* optimal dose and duration are unclear

** although not recommended for induction or maintenance of remission, vitamin D supplementation is not contraindicated in IBD and supplementation may confer benefit, especially when deficiency is present

Table 3. Rationale and description of defined diets recommended in the management of IBD

Dietary intervention	Description of diet and rationale	Key food inclusions/exclusions [not an exhaustive list]
Exclusive enteral nutrition (EEN)	Total exclusion of food using a nutritionally complete liquid enteral nutrition formula reduces intestinal inflammation and promotes mucosal healing in Crohn's disease.	**Inclusion**: 100% of energy intake from polymeric (whole protein), semi-elemental (oligopeptide) or elemental (amino acid based) formula. **Exclusion**: All foods, beverages (except water and enteral nutrition formula).
Partial enteral nutrition (PEN)	Partial replacement of food with enteral nutrition formula reduces intestinal inflammation in Crohn's disease.	**Inclusion**: 35–50% of estimated energy requirements from polymeric, semi-elemental or elemental formula. **Exclusion**: None.
Crohn's disease exclusion diet (CDED) + PEN	Structured exclusion of food components (e.g., emulsifiers, maltodextrin, carrageenan, gluten) impairs intestinal barrier function, leading to dysbiosis, combined with PEN reduces intestinal inflammation in Crohn's disease.	**Inclusion**: 14 whole foods including chicken, eggs, rice, bananas, apples, potatoes; 50% of estimated energy requirements from enteral nutrition formula. **Exclusion**: For food components - gluten, dairy, processed foods, additives and high-fat animal products.
Mediterranean diet	High intake of plant-based foods rich in fibre, antioxidants, healthy fats, limited protein from meat supports a diverse microbiota and reduces inflammation in IBD.	**Inclusion**: Fruits, vegetables, legumes, whole grains, olive oil, fish, nuts. **Exclusion**: Red and processed meats, sugary drinks, ultra-processed foods.
Low FODMAP diet	FODMAPs are fermentable short-chain carbohydrates responsible for increased intestinal water and colonic gas, decreased FODMAP intake reduces IBS-like symptoms in susceptible individuals.	**Inclusion**: Defined food lists of low FODMAP foods, such as meat and fish, oats, rice, lactose-free milk, low FODMAP fruits and vegetables. **Exclusion**: Defined list of high FODMAP foods, such as onion, garlic, wheat, legumes, apples, milk.
Modified or low fibre diet	A diet low in bulking fibre reduces risk of intestinal obstruction in people with stricturing Crohn's disease.	**Inclusion**: White bread, white rice, peeled well-cooked vegetables,

		banana, peeled apple, pureed legumes. **Exclusion**: Wholegrain bread, brown rice, raw vegetables, edible fruit peel, whole legumes.
Low oxalate diet	A low oxalate diet reduces risk of oxalate stone formation in people at risk of calcium oxalate nephrolithiasis.	**Inclusion**: Most meats, grains, many fruits and vegetables, such as onion, potato, mushroom, avocado, banana, apple. **Exclusion**: Nuts, nut butters, cocoa, rhubarb, beetroot, spinach, berries, soy products.
Cinnamon- and benzoate-free diet	Elimination of foods containing cinnamon and benzoates reduces allergy-like responses associated with orofacial granulomatosis.	**Inclusion**: Most meats, whole grains, fruits, vegetables and dairy. **Exclusion**: Cinnamon, benzoic acid, sodium benzoate, and processed foods containing these additives.
Healthy eating patterns	A healthy eating pattern reduces risk of IBD development.	**Inclusion**: Whole grains, fruits, vegetables, nuts, seeds, legumes and fish. **Exclusion**: Red meat, processed meats, high-sugar foods and beverages.

Table 4. Example foods and supplements containing fermentable, viscous, and bulking fibre [not an exhaustive list]

Food group	Foods containing fermentable fibre	Foods containing viscous fibre	Foods containing bulking fibre
Grains	• Wheat breads • Wheat pasta	• Oats • Barley	• Whole-grain breads • Bran-based cereals • Barley • Quinoa • Brown rice
Vegetables	• Onion • Garlic • Potato [particularly cooked and cooled]	• Vegetable peel [e.g. potato skin]	• Raw vegetables [e.g. salads] • Broccoli • Cauliflower • Corn • Celery • Vegetable peel [e.g. potato skin]
Fruit	• Apple • Pear • Stone fruit [e.g. apricots, plums] • Banana [particularly green]	• Apple • Pear • Stone fruit [e.g. apricots, plums]	• Edible fruit peel [e.g. apple peel] • Edible seeds [e.g. passionfruit] • Dried fruit
Meat/alternatives	• Legumes [whole and pureed, e.g. baked beans, chickpeas, lentils]	• Legumes [whole and pureed, e.g. baked beans, chickpeas, lentils]	• Whole legumes [e.g. baked beans, lentils and chickpeas] • Whole nuts and seeds
Dairy/alternatives	-	-	-
Fibre supplements	• Inulin	• Psyllium	• Cellulose

Table 5. Advantages and disadvantages of tools assessing body composition

Tool	Advantages	Disadvantages	Marker of sarcopenia	Marker of visceral adiposity
BMI	Free, rapid, easy to measure and track	Does not provide information about body composition, can misclassify malnutrition, cannot detect sarcopenia	Poor	Moderate
Unintentional weight loss	Free, easy to measure and track, indicates recent nutritional changes	Does not provide information about body composition, cannot detect sarcopenia	Poor	Poor
MUAC	Inexpensive, rapid, easy to measure	Does not accurately reflect body composition, cannot detect sarcopenia, less sensitive to changes of nutritional status	Poor	Poor
MAMC	Inexpensive, provides more detailed information about muscle mass than MUAC, practical indicator of muscle mass when combined with triceps skinfold	Requires triceps skinfold measurement to calculate, more time-consuming, interpretation requires age and gender-specific reference values, limited in accuracy compared with more advanced techniques	Poor	N/A
Waist circumference	Inexpensive, rapid, easy to measure, practical indicator of visceral adiposity	Does not accurately reflect muscle composition, cannot detect sarcopenia	N/A	Good
Handgrip strength	Moderate cost, non-invasive, easy to perform, can detect sarcopenia by assessing muscle strength, sensitive to changes of nutritional status	Interpretation requires age and gender-specific reference values	Good with muscle mass measure	N/A
Quadriceps muscle strength	Moderate cost, non-invasive, can detect sarcopenia by assessing muscle strength of lower body	Requires specialised equipment and training, interpretation requires age and gender-specific reference values	Good with muscle mass measure	N/A

Bioimpedance	Moderate cost, non-invasive, rapid, easy to use, generally portable, can detect sarcopenia by measuring body composition, sensitive to changes of nutritional status	Requires specialised equipment and training, might be less accurate especially in certain populations [e.g. obese or cachectic patients]	Good with muscle strength measure	N/A
DEXA	Highly accurate for body composition assessment, can detect sarcopenia, can measure bone density, can assess visceral adiposity	Expensive, requires specialised equipment and training, very low dose of radiation	Reference method	Reference method
CT	Can detect sarcopenia by measuring body composition, providing precise measurements of skeletal muscle mass, can assess visceral adiposity	Expensive, requires specialised equipment and training, high dose of radiation	Reference method	Reference method
MRI	Can detect sarcopenia by measuring body composition, can assess visceral adiposity, provides detailed images without radiation exposure	Very expensive, limited availability and time consuming, requires specialised equipment and training	Reference method	Reference method
Ultrasound	Moderate cost, portable, non-invasive, provides imaging of muscle and subcutaneous fat layers without radiation exposure	Requires specialised equipment and training, less accurate for body composition assessment, validation studies are required before a recommendation for sarcopenia detection can be made	N/A	Moderate

Table assessment tools summary are based on these references[1-7]

BMI	Body Mass Index
CT	Computed tomography
DEXA	Dual Energy X-ray Absorptiometry
MAMC	Mid-upper Arm Muscle Circumference
MRI	Magnetic Resonance Imaging
MUAC	Mid-Upper Arm Circumference
N/A	Not applicable

Table 6. Definition of anaemia according to WHO criteria [2024][8]

Age or sex group	Mild to moderate anaemia [haemoglobin g/L]	Severe anaemia [haemoglobin g/L]
Children, 6–23 months	< 105	< 70
Children, 24–59 months	< 110	< 70
Children, 5–11 years	< 115	< 80
Girls, 12–14 years [non-pregnant]	< 120	< 80
Boys, 12–14 years	< 120	< 80
Women, 15–65 years [non-pregnant]	< 120	< 80
Men, 15–65 years	< 130	< 80
First trimester pregnancy	< 110	< 70
Second trimester pregnancy	< 105	< 70
Third trimester pregnancy	< 110	< 70

Table 7. Characterisation of relevant micronutrients in IBD, food sources, deficiencies, and recommended monitoring

Micronutrient	Role in body	Food sources	Symptoms of deficiency	Definition of deficiency	Populations to consider prophylactic supplementation	Monitoring
Iron*	Formation of haemoglobin	High bioavailability: Meat, seafood, and poultry Low bioavailability: Iron-fortified breakfast cereals and breads, legumes, spinach, nuts and some dried fruits	Weakness, fatigue, lack of energy, and problems with concentration and memory	CRP < 10 mg/L and ferritin < 30 µg/L CRP > 10 mg/L and ferritin < 100 µg/L CRP not available TfS < 16%	Pregnancy, infancy, those on vegetarian or vegan diets, those with anaemia of chronic disease	At diagnosis and at least every 12 months in patients in remission, at least every 3 months in those with active disease
Folate*	DNA replication, metabolism of amino acids, maturation of red blood cells	Green vegetables, legumes/legumes flours, whole grains, offal, folate-fortified foods	Fatigue, mouth ulcers, decreased appetite, pale skin, dizziness, poor appetite, diarrhoea, irritability	Serum folate < 7 nmol/L or 3 g/L	Those on restrictive diets, those on sulfasalazine or methotrexate, pregnancy/pregnancy planning	At diagnosis and annually

Vitamin B12*	DNA synthesis, nerve myelin synthesis, cellular metabolism, maturation of red blood cells	Animal products, vitamin B12 fortified plant foods	Fatigue, diarrhoea or constipation, poor concentration, frequent infections, poor appetite, numbness in extremities, depression	Variable definitions [148–185 pmol/L or 200– 250 pg/mL; serum vitamin B12 poor marker], elevated methylmalonic acid	Those on vegan diet, those with ileal resection > 20 cm	At diagnosis and annually
Vitamin D	Maintains bone health alongside calcium and phosphorus, supports gut absorption of calcium, magnesium, phosphorus	Cod liver oil, fatty fish, vitamin D supplemented foods and drinks	Osteopenia and osteoporosis	< 50 nmol/L or < 30 ng/dL	Those with limited sunlight exposure, those at far distance from the equator	At diagnosis and annually
Vitamin C	Tissue repair, formation of collagen, production of neurotransmitters , enhances non-haem iron absorption	Fruit, vegetables	Poor wound healing, easy bruising or bleeding, fatigue	30–80 µmol/L if CRP < 10 mg/L	Those on restrictive or poor-quality diets	If deficiency suspected

Zinc	Cellular metabolism, immune function, protein and DNA synthesis, wound healing, and cell signalling and division	Meat, seafood	Alopecia, delayed growth [in children], frequent infections, hair loss, poor wound healing, loss of appetite, infertility, loss of taste and smell	< 9 mml/L [74 µg/dL; fasting] < 8.4 mml/L [postprandial]	Those on vegetarian or vegan diets, pregnancy and lactation, people with chronic diarrhoea	At diagnosis and annually in people with Crohn's disease [not during active disease]
Calcium	Builds and maintains bones and teeth	Dairy, fish with edible bones, calcium-fortified dairy alternatives	Osteopenia and osteoporosis	Serum calcium markers poor indicator of deficiency	Those with active IBD, frequent or long-term corticosteroid use, children, those who restrict dairy	N/A
Magnesium	Energy production, maintains nerve and muscle function, immunity, maintains calcium regulation	Green vegetables, soybeans, nuts, spices, cocoa	Loss of appetite, nausea, vomiting, muscle spasms, abnormal heart rhythm	0.6–1.0 mmol/L	People with chronic or severe diarrhoea	If deficiency suspected

*For supplementation guidelines refer to Gordon et al. 2024 ECCO guidelines on extraintestinal manifestations in IBD[9]

Table 8. Dietary alarm features to indicate referral for dietary assessment

High-risk behaviour	Possible contribution to malnutrition
Skipping meals	Not meeting nutritional requirements or overeating at other times
Continual eating	Small food volumes leading to undereating or not stimulating satiety, enabling overeating
Inappropriate eating times [e.g. overnight]	Avoidance of 'normal' eating leading to under- or overeating
Extreme dietary restriction	Food restriction resulting in undereating or compensation with high-energy foods
Lack of diet variety	Nutritional gaps, limited variety, or including only high-energy foods
Poor knowledge of intake	Poor food choice leading to nutritional gaps or excesses
Fussy eating	Insufficient nutritional intake or including only high-energy foods
Disordered eating	Intentional or inappropriate food restriction [or both] or binge eating

Table 9. Associations of macronutrients, micronutrients, dietary patterns, and breastfeeding with development of Crohn's disease and ulcerative colitis from prospective cohort studies

Food component	Prospective cohort study	Participants [n]	Association with Crohn's disease	Association with ulcerative colitis	Reference
Macronutrients					
Total fat and long-chain fatty acids	EPIC	229 702	None	-	[10]
n-3	EPIC	25 639		↓ DHA	[11]
n-3	NHS I and NHS II	238 130	None	None	[12]
n-3	EPIC	229 702	↓ DHA	-	[10]
n-3	EPIC	203 193	-	↓ DHA	[11]
n-6	DCH	57 053	-	↑ arachidonic acid	[13]
n-6	EPIC	25 639	-	↑ arachidonic acid	[14]
n-9	EPIC	25 639	-	↓ oleic acid	[14]
n-3:n-6 ratio	NHS I and NHS II	170 805	None	↓	[15]
n-3:n-6 ratio	NHS I and NHS II	238 130	↓ in certain genotypes	None	[12]
Fibre	NHS I and NHS II	170 776	↓ total fibre and fibre from fruit	None	[16]
Fibre	EPIC	401 326	None	None	[17]
Fibre	UK Biobank	470 669	↓ total fibre and fibre from fruit and bread	↓ fibre from cereal	[18]
Micronutrients and polyphenols					
Vitamin D	NHS I	72 719	None	↓	[19]
Vitamin D	E3N	67 572	None	None	[20]
Vitamin D	NHS I	359 728	None	None	[21]
Vitamin D	EPIC	260 686	-	None	[22]
Zinc	NHS I and NHS II	170 776	↓	None	[23]
Zinc	NutriNet Santé	359 728	↓	None	[24]
Iron	NHS I and NHS II	165 331	None	None	[25]
Iron	EPIC	260 686	-	None	[22]
Polyphenols	EPIC	401 326	↓ resveratrol	None	[26]
Food groups					

Dairy	Meta-analysis of 11 cohort studies	4 302 554	None [after adjusted for energy, BMI, and alcohol intake]	None [after adjusted for energy, BMI, and alcohol intake]	[27]
Fruit and vegetables	Meta-analyses of 3–4 cohort studies	211 767 fruit 211 540 vegetables	↓	↓	[28]
Meat	Meta-analysis of 11 cohort studies	4 302 554	None	None	[27]
Meat	PURE	116 087	None*	None*	[29]
Meat	DCH	56 468	-	-	[30]
Meat	EPIC	413 593	None	↑ total and red meat	[31]
Meat	NHS II	39 511	None	None	[32]
Meat	E3N	67 581	None	↑ animal protein	[33]
Alcohol	EPIC	262 451	None	None	[34]
Sweetened beverages	SMC/CoSM	83 042	None	None	[35]
Sweetened beverages	UK Biobank	121 490	↑ sugar-sweetened beverages	None	[36]
Dietary patterns					
Healthy Lifestyle Score	NHS I, NHS II, HPFS, EPIC and SMC/CoSM	208 070 primary cohort 482 229 validation cohort	↓ Healthy Lifestyle Score	↓ Healthy Lifestyle Score	[37]
Healthy Lifestyles Score	UK Biobank	429 515–430 384	↓ Healthy Lifestyle Score alone and in combination with polygenic risk scores	↓ Healthy Lifestyle Score alone and in combination with polygenic risk scores	[38]
Nutritional quality	EPIC	394 255	↓ UK Food Standards Agency modified nutrient profiling system score	None	[39]
Cardioprotective diet	UK Biobank	482 887	↓ cardioprotective diet score	↓ cardioprotective diet score	[40]

Mediterranean diet score	SMC/CoSM	83 147	↓ modified Mediterranean diet score	None	[41]
Several healthy eating scores	SMC/CoSM	83 147	↓ healthy eating scores	None	[42]
Mediterranean diet score	EPIC	366 351	None	None	[43]
Several healthy eating scores	Lifelines	125 445	↓ Lifelines diet score	↑ red meat, processed meat, and poultry	[44]
Healthy eating index	ABIS and MoBA	81 280	↓ Medium and high diet quality score	↓ Medium and high diet quality score	[45]
Healthy eating patterns	Meta-analysis of 6 studies including 4 case-control studies	264 211	↓ healthy eating pattern	None	[46]
Western dietary pattern	Meta-analysis of 6 studies including 2 cohorts	39 511–222 419	None [cohorts only]	None	[47]
EDIP score	NHS I, NHS II, and HPFS	208 834	↑ higher inflammatory score	None	[48]
EDIP score	PURE	28 428	None	None	[29]
EDIP score	EPIC	32 633	↑ higher inflammatory score	None	[49]
UPF	Meta-analysis of 5 studies	1 068 425	↑ UPF	None	[50]
Breastfeeding					
Breastfeeding	NSHD	3 322	None	None	[51]
Breastfeeding	DNBC, ABIS and MoBA	148 737–169 510	None	None	[52]

* Processed meat intake associated with increased risk of IBD but not when adjusted for Crohn's disease or ulcerative colitis

ABIS and MoBA All Babies in Southeast Sweden Cohort and Norwegian Mother, Father, and Child Cohort
BMI body mass index
DCH Danish Diet, Cancer and Health cohort
DHA docosahexaenoic acid
DNBC Danish National Birth Cohort
E3N Etude Épidémiologique des femmes de la Mutuelle Générale de l'Education National
EDIP Empirical Dietary Inflammatory Pattern
EPIC European Prospective Investigation into Cancer and Nutrition
HPFS Health Professional Follow-up Study

n-3	omega-3 fatty acids
n-6	omega-6 fatty acids
n-9	omega-9 fatty acids
NHS	Nurses' Health Study
NSHD	The 1946 National Survey of Health & Development
PURE	Prospective Urban Rural Epidemiology cohort
SMC/CoSM	Swedish Mammography Cohort / Cohort of Swedish Men
UPF	Ultra-Processed Food

Table 10. Proposed dietary research priorities for improvement of IBD knowledge and care

Area	Research need	Example
Dietary therapy to induce and maintain remission of IBD	Develop and validate objective tools to assess dietary adherence and investigate the impact of compliance on treatment outcomes	Design prospective studies using metabolomic and isotope-based biomarkers and digital dietary tracking tools to quantify adherence and correlation with clinical, inflammatory, and microbiome-based outcomes in dietary interventions for IBD
	Elucidate the biological pathways through which EEN induces remission in Crohn's disease.	Conduct integrated multi-omic studies in both human patients and complementary animal/in vitro models to investigate how EEN modulates gut microbiota, microbial metabolites, mucosal immunity, and epithelial barrier function.
	Assess the efficacy and mechanisms of whole-food dietary interventions in induction and maintenance of remission in IBD, including the exclusion of ultra-processed foods [UPF], gluten and specific components identified as inflammatory in preclinical models	Randomised controlled trials comparing whole-food diets that exclude UPFs and specific components—such as emulsifiers, artificial sweeteners, gluten, and sugar/refined carbohydrates.
	Investigate the efficacy of combining dietary therapy with advanced pharmacological treatments to enhance induction of remission	Randomised controlled trials evaluating dietary interventions [e.g. CDED, whole-food diets, EEN] as adjuncts to biologics or small molecules
	Development of strategies to maintain remission	Development of existing dietary strategies that have shown promise, but currently with insufficient evidence to make a firm recommendation, such as CDED as maintenance therapy, PEN in CD, low red and ultra-processed meat in UC, Vegetarian diet in IBD, Mediterranean diet in UC.
Dietary management of comorbidities and special conditions of IBD	Prevention of postoperative complications	Assessment of 10 days of oral nutritional supplementation and 2 weeks EEN, as part of the ERAS programme, in people with IBD awaiting surgery
	Improvement of pouch function and prevention of pouchitis	Development and assessment of dietary therapies in people with UC and an ileal pouch-anal anastomosis
	Treatment of persistent gastrointestinal symptoms	Development and assessment of therapies in adults and children

		with quiescent IBD that minimise dietary burden
Nutritional assessment and optimisation of IBD	Practical nutritional assessment	Identification of the optimal nutrition assessment tools for use by clinicians and people with IBD
	Longitudinal nutritional assessment	Research that reports longitudinal nutritional status assessment data using BMI in combination with assessments of body composition, muscle function, or both
Diet as prevention of IBD development	Mechanism(s) of diet conferring protection or risk of IBD development	Preclinical studies to evaluate the effects of well-defined diets on IBD pathogenesis
	Optimal diet for preventing IBD	Evaluation of a well-defined diet in healthy people followed for many years

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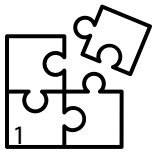

Access to an IBD dietitian

	IBD	Crohn's disease	Ulcerative colitis
Diet as **induction** of remission		EEN* PEN CDED + PEN	Curcumin QingDai
Diet as **maintenance** of remission		PEN	Curcumin Mediterranean diet Red/processed meat ↓
Diet for **comorbidities / special conditions**	Low FODMAP diet for IBS-like symptoms*	Preoperative EEN EEN for strictures Cinnamon / benzoate-free diet for OFG	Fermentable fibre, mediterranean diet, daily fruit for IPAA
Nutritional **assessment & optimisation**	Screening for malnutrition Global nutritional and body composition assessment Micronutrient monitoring during remission Assessment of dietary intake		
Diet as **prevention** of development	↓risk: healthy eating, fibre	↓risk: fruit, high fibre breads, zinc, DHA ↑risk: ultra-processed food*	↓risk: high fibre cereals, high n-3:n-6 ratio PUFAs, DHA, oleic acid, vitamin D ↑risk: arachidonic acid

* Evidence Level 1