

# British Dietetic Association consensus guidelines on the nutritional assessment and dietary management of patients with inflammatory bowel disease

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## Abstract

**Background:** Despite increased awareness of diet and nutrition being integral to the management of patients with inflammatory bowel disease (IBD), there are gaps in the knowledge of IBD healthcare providers regarding nutrition. Furthermore, high quality evidence on nutritional assessment and dietary management of IBD is limited. A Delphi consensus from a panel of experts allows for best-practice guidelines to be developed, especially where high quality evidence is limited. The aim was to develop guidelines for the nutritional assessment and dietary management of IBD using an eDelphi online consensus agreement platform.

**Methods:** Seventeen research topics related to IBD and nutrition were systematically reviewed. Searches in Cochrane, Embase<sup>®</sup>, Medline<sup>®</sup> and Scopus<sup>®</sup> electronic databases were performed. GRADE was used to develop recommendations. Experts from the IBD community (healthcare professionals and patients with IBD) were invited to vote anonymously on the recommendations in a custom-built online platform. Three rounds of voting were carried out with updated iterations of the recommendations and evaluative text based on feedback from the previous round.

**Results:** From 23,824 non-duplicated papers, 167 were critically appraised. Fifty-five participants completed three rounds of voting and 14 GRADE statements and 42 practice statements achieved 80% consensus. Comprehensive guidance related to nutrition assessment, nutrition screening and dietary management is provided.

**Conclusions:** Guidelines on the nutritional assessment and dietary management of IBD have been developed using evidence-based consensus to improve equality of care. The statements and practice statements developed demonstrate the level of agreement and the quality and strength of the guidelines.

## KEYWORDS

diet, Crohn's disease, inflammatory bowel disease, nutritional assessment, ulcerative colitis

## Key points

- Comprehensive guidelines related to nutrition assessment, nutrition screening and dietary management of inflammatory bowel disease have been developed.

- Fourteen GRADE statements and 42 practice statements achieved 80% consensus agreement
- Fifty-five participants comprising experts from the inflammatory bowel disease (IBD) community (healthcare professionals and patients with IBD) took part in the online consensus voting

## INTRODUCTION

Despite increased awareness of diet and nutrition being integral to the management of patients with inflammatory bowel disease (IBD), there are still gaps in the knowledge of IBD healthcare providers regarding nutrition.<sup>1,2</sup> Furthermore, high quality evidence on the nutritional assessment and dietary management of patients with IBD is limited. Consensus from a panel of experts known as a Delphi process allows for best-practice guidelines to be developed, especially where high quality evidence is limited. The aim of this research was to develop guidelines for the nutritional assessment and dietary management of IBD using an eDelphi online consensus agreement platform (<https://www.edelphi.org>).

## METHODS

An expert IBD panel including two patients, clinicians and researchers was created to discuss pertinent topics related to diet and IBD to include in the guideline. Sixteen research topics were identified from the research literature, clinical practice and gaps in the evidence base (see Supporting information, Table S1).

### Search strategy

Systematic reviews for each topic were undertaken in line with recommendations of the Cochrane Handbook for Systematic Reviews of Interventions<sup>3</sup> and reported in line with the guidelines of Preferred Reporting Items for Systematic reviews and Meta-Analyses.<sup>4</sup> A Population, Intervention, Comparison and Outcomes framework and literature search strategy criteria were developed (see Supporting information, Tables S2 and S3) to search Cochrane, Embase®, Medline® and Scopus® electronic databases for topics on nutritional assessment and dietary management of IBD up until March 2022. Identification, screening, eligibility and inclusion of eligible papers were agreed between the researchers in advance and published prior to the literature searches being conducted (PROSPERO 2018 CRD42018096818; PROSPERO 2019 CRD42019138650).

References were imported into a bibliographic database and duplicates were removed automatically (EndNote X9; Thomson Reuters). Titles and abstracts

were screened against the PICO eligibility criteria and potentially eligible full text articles were then obtained and screened against the eligibility criteria by two researchers independently. Reference lists of potential studies were cross-referenced for other studies of potential relevance. The percentage agreement in study eligibility and a kappa statistic were calculated to check concordance between reviewers.<sup>3</sup> Disparities in study eligibility were resolved through discussion with a third researcher.

Reasons for excluding studies are provided (see Supporting information, Table S4).

### Data extraction and risk of bias

Data were extracted from each eligible study relating to the patient or group, the intervention, the comparator, outcomes measured and the study design. A standardised data extraction sheet was developed, and two reviewers extracted the data from eligible papers. Discrepancies were reviewed and resolved.

Risk of bias tools appropriate to the study design were used. Two reviewers independently assessed risk of bias using seven domains: adequacy of randomisation, allocation concealment, blinding methods, complete outcome data, selective reporting and other sources of bias.<sup>3</sup> Any disparities were resolved through discussion with a third reviewer.

Where data were available from randomised controlled trials (RCTs), this was used to answer the research question and lower quality studies were not considered when developing evidence statements. Where no RCT data were available lower quality studies were considered when developing evidence statements.

### GRADE approach

Research papers were critically appraised using the GRADE tool.<sup>5</sup> GRADE analysis of the evidence was performed where two or more studies reported data for the same outcome. For dichotomous outcomes (e.g., remission), frequencies were entered to obtain an odds ratio (OR). For continuous outcomes (e.g., disease activity score), standardised mean differences were calculated.

To address the research questions where data were expected to be available for a range of interventions,

dietary interventions were categorised and each of these were separately assessed for available outcomes using the GRADE approach. The categories of nutrition outcome were; complementary alternative medicine, elimination diets, enteral nutrition, fibre (including prebiotics), nutrients, probiotics and whole diets.

Where a recently published systematic review was available, data from this were combined with data from studies published subsequent to the last search date of the systematic review.

Furthermore, where evidence was of low quality and GRADE Statements were not possible, recommendations to inform clinical practice were developed as Practice Statements.

## Consensus

GRADE Statements and Practice Statements underwent consensus voting using an eDelphi online platform. IBD experts from British Dietetic Association Gastroenterology Specialist Group, British Society of Gastroenterology, Crohn's and Colitis UK and patients with IBD were invited to vote anonymously on the statements.

Three rounds of voting took place. Participants used a five-point Likert scale to vote (strongly disagree, disagree, neutral, agree, strongly agree). After each voting round, statements that did not reach 80% consensus were reformulated and taken to the next round of voting or removed. In rounds 2 and 3, updated iterations of the statements were voted on and evaluative text based on feedback from the previous round was available.

## RESULTS

From 23,824 non-duplicated papers, 167 were critically appraised (see Supporting information, Table S5). Overall, the evidence was of generally low quality and risk of bias assessment is provided for all studies linked to GRADE statements but not practice statements (Table 1).

Fifty-five out of an initial 114 participants completed three rounds of voting and 14 GRADE statements and 42 practice statements achieved 80% consensus (Table 2). Comprehensive guidance related to nutrition assessment, nutrition screening and dietary management [induction of disease remission, maintenance of disease remission, functional gut symptoms, strictures, surgery, stoma, short bowel syndrome, fistula, pouchitis and special situations; e.g., orofacial granulomatosis (OFG)] are provided below. Four GRADE statements and nine practice statements were removed as a result of not reaching the consensus cut off.

## NUTRITION ASSESSMENT: ANTHROPOMETRY AND BODY COMPOSITION

**PRACTICE STATEMENT:** Disease activity affects body composition in inflammatory bowel disease; therefore, it is desirable to assess nutritional status longitudinally using body mass index in combination with assessments of body composition and/or muscle function. Agreement 96.2%.

Anthropometric measurement of nutrition assessment in patients with IBD in clinical practice is challenging and most often relies solely on weight and body mass index (BMI). Systemic inflammation affects body composition, often with a reduction in muscle mass (i.e., fat free mass) and an increase in fat mass. Sarcopenia is common in IBD, even in overweight patients,<sup>6</sup> and relying on weight and BMI may mask changes in body composition; thus, other anthropometric measures available in clinical practice are important in IBD and were systematically reviewed.

### BMI

Thirty studies reported BMI, 11 studies matched healthy controls and IBD patients for BMI so were excluded from the GRADE analysis.<sup>7–17</sup> The remaining 19 studies included 1077 patients with Crohn's disease (CD), 426 patients with UC and 4255 healthy controls.<sup>18–36</sup>

### Crohn's disease

Eight studies reported BMI in remission in CD.<sup>18–22,26,33,35</sup>

Four studies found BMI was similar between patients with CD in remission ( $n = 151$ ) and healthy controls ( $n = 193$ )<sup>21,22,26,35</sup> and four studies found BMI was lower in patients with CD in remission ( $n = 266$ ) compared to healthy controls ( $n = 225$ )<sup>18–20,33</sup>

One study reported BMI in active CD<sup>18</sup> and a further six studies in active CD and CD in remission.<sup>23,24,27,30,34,36</sup>

Four studies found BMI was similar between patients with active CD or CD in remission ( $n = 163$ ) and healthy controls ( $n = 174$ )<sup>23,24,27,34</sup> and two studies found BMI was lower in patients with active CD or CD in remission ( $n = 286$ ) compared to healthy controls ( $n = 2006$ ).<sup>30,36</sup>

### Ulcerative colitis

Two studies assessed BMI in ulcerative colitis (UC) patients in remission.<sup>20,26</sup> Both studies found BMI was

TABLE 1 Risk of bias

Question/Author (year)	Study type	Q 1	Q 2	Q 3	Q 4	Q 5	Q 6	Q 7	Q 8	Overall
<i>Nutritional assessment</i>										
Ardizzone et al. (2000)	Case control	+	+	?	?	+				Unclear
Bastos et al. (2017)	Case control	+	+	+	?	?				Unclear
Benjamin et al. (2011)	Case control	?	+	?	?	?				Unclear
Buning et al. (2015)	Case control	+	+	?	?	+				Unclear
Capristo et al. (1998)	Case control	+	+	+	?	+				Unclear
Capristo et al. (1998)	Case control	+	+	+	?	+				Unclear
Cox et al. (2021)	Case control	+	+	+	?	?				Unclear
Cuoco et al. (2008)	Case control	+	+	?	?	?				Unclear
Dalekos et al. (1998)	Case control	+	+	?	+	+				Unclear
D'Odorico et al. (2001)	Case control	+	+	?	+	+				Unclear
Duggan et al. (2004)	Case control	+	+	+	+	+				Low
Dumitrescu et al. (2014)	Case control	+	+	?	?	+				Unclear
Emerenziani et al. (2017)	Case control	+	+	+	+	+				Low
Filippi et al. (2006)	Case control	+	+	+	?	+				Unclear
Geerling et al. (1998)	Case control	+	+	+	+	+				Low
Geerling et al. (1999a)	Case control	+	+	+	+	+				Low
Geerling et al. (1999b)	Case control	+	+	+	+	+				Low
Geerling et al. (2000a)	Case control	+	+	+	+	+				Low
Gentschew et al. (2012)	Case control	+	+	?	?	?				Unclear
Ghoshal and Shukla (2008)	Case control	+	+	?	+	+				High
Gilman et al. (2006)	Case control	+	+	+	+	+				Low
Grunbaum et al. (2013)	Case control	+	+	+	+	+				Low
Guerreiro et al. (2007)	Case control	+	+	+	+	?				Unclear
Hengstermann et al. (2008)	Case control	+	+	?	?	?				Unclear
Hinks et al. (1988)	Case control	+	+	?	?	+				Unclear
Jahnsen et al. (2003)	Case control	+	+	+	+	+				Low
Joseph et al. (2009)	Case control	+	+	+	+	+				Low
Kallel et al. (2011)	Case control	+	+	+	+	+				Low
Katznelson et al. (2003)	Case control	+	+	?	?	+				Unclear
Kawakami et al. (2005)	Case control	+	?	?	+	+				High
Khadgawat et al. (2008)	Case control	+	+	+	+	+				Low
Krzystek-Korpacka et al. (2010)	Case control	+	+	+	+	+				Low
Lomer et al. (2004)	Case control	+	+	+	+	+				Low
Lu et al. (2016)	Case control	+	+	?	?	+				Unclear
Magro et al. (2018)	Case control	+	+	+	+	+				Low
Mijač et al. (2010)	Case control	+	+	+	?	+				High
Mingrone et al. (1999)	Case control	+	+	+	?	+				Unclear
Mohammadi et al. (2017)	Case control	+	+	?	+	+				Low
Molnar et al. (2017)	Case control	+	?	+	+	+				Unclear
Opstelten et al. (2018)	Case control	+	?	?	+	?				Unclear
Powell et al. (2013)	Case control	+	+	+	+	+				Unclear
Principi et al. (2018)	Case control	+	+	+	?	+				Low
Ringstad et al. (1993)	Case control	+	+	+	+	+				Low
Rizzi et al. (2012)	Case control	?	?	+	?	?				Unclear

(Continues)

Salacinski et al. (2013)	Case control	+	?	+	+	+			Low
Schneider et al. (2008)	Case control	+	?	+	?	+			Unclear
Sturniolo et al. (1998)	Case control	+	+	?	+	+	+		High
Suibhne et al. (2012)	Case control	+	?	+	+	+	+		Unclear
Szentmihalyi et al. (2009)	Case control	?	?	?	?	?	+		Unclear
Tajika et al. (2004)	Case control	+	?	?	?	?	?		Unclear
Tan et al. (2014)	Case control	+	+	+	+	+	?		Low
Tjellesen et al. (1998)	Case control	+	?	+	+	+	?		Unclear
Ueda et al. (2008)	Case control	+	+	+	+	+	+		Unclear
Ulivieri et al. (2000)	Case control	+	?	+	+	+	+		Low
Ulivieri et al. (2001)	Case control	+	?	+	+	+	+		Low
Valentini et al. (2008)	Case control	+	+	+	+	+	+		Low
Vernia et al. (2014)	Case control	+	?	?	?	+	+		Unclear
Wendland et al. (2001)	Case control	+	+	+	+	+	+		Low
Wiroth et al. (2005)	Case control	+	?	+	?	+	+		Unclear
Zaltman et al. (2014)	Case control	+	?	?	+	+	+		Unclear
Zhang et al. (2017)	Case control	+	+	+	+	+	+		Unclear

*Nutritional screening*

Csontos et al. (2017)	Case series	?	+	?	+	+	?		High
Keetarut et al. (2017)	Case series	+	?	+	+	+	+		Unclear
Sandhu et al. (2016)	Case series	?	?	+	?	+	+		High
Takaoka et al. (2015)	Case series	?	+	+	+	+	+		High
Takaoka et al. (2017)	Case series	?	+	?	+	+	+		High
Dell'Era et al. (2016)	Cohort	+	?	?	+	?	+	+	Unclear
Mazzuoli et al. (2012)	Case series	?	+	+	+	+	+		High
Fiorindi et al. (2021)	Case series	?	+	+	+	+	+		High
Taylor et al. (2021)	Case series	?	+	+	?	+	?		High
Jansen et al. (2016)	Case series	+	?	?	?	+	+		Unclear
Haskey et al. (2018)	Case series	+	+	?	?	+	+		Unclear
Wall et al. (2019)	Case series	+	+	?	?	+	+		Unclear
Vigano et al. (2018)	Case series	?	+	?	?	+	+		High

*Oral nutrition support*

Dell'Era et al. (2016)	Cohort	+	?	?	+	?	+	?	Unclear
Harries et al. (1983)	RCT	?	?	?	+	+	?	?	Unclear
Sökülmez et al. (2014)	Case series	+	?	?	+	+	+		High
Verma et al. (2000)	RCT	?	?	?	?	+	?	?	Unclear

*Induction of remission*

## Exclusive enteral nutrition

Gassull et al. (2002)	RCT	+	+	+	+	+	+		Low
Gonzalez-Huix et al. (1993)	RCT	+	?	+	?	+	+		High
Lindor et al. (1992)	RCT	?	?	+	?	+	+		High
Lochs et al. (1991)	RCT	?	?	+	?	+	+		High
Malchow et al. (1990)	RCT	+	?	?	?	+	+		Unclear
Pfeffer-Gik et al. (2016)	Case series	+	?	+	?	+	+		High
Savarymattu et al. (1985)	RCT	?	?	?	?	+	+	?	Unclear
Wall et al. (2018)	Case series	+	?	?	+	+	+		High
Whole food or elimination diets									
Sigall-Boneh et al. (2014)	Case series	?	?	?	+	?	+		Unclear
Gunasekeera et al. (2016)	RCT	+	+	+	?	+	+		Low
Bartel et al. (2008)	RCT	?	?	?	?	+	?	+	Unclear

Lomer et al. (2001)	RCT	?	?	?	+	+	?	+	Unclear
Lomer et al. (2005)	RCT	+	?	?	+	+	+	+	Low
Chiba et al. (2017)	Cohort	?	+	+	?	?	-	?	Unclear
Fibre									
Benjamin et al. (2011)	RCT	?	+	?	?	?	?	+	Unclear
Steed et al. (2010)	RCT	+	?	+	+	+	+	+	Unclear
Valcheva (2019)	RCT	?	?	-	?	+	+	+	High
Fish oil									
Seidner et al. (2005)	RCT	+	+	+	+	?	?	?	Unclear
Wiese et al. (2011)	Case series	-	-	?	-	+	?		High
Curcumin									
Banerjee et al. (2017)	RCT	?	?	?	+	+	+	+	Unclear
Lang et al. (2015)	RCT	+	+	+	+	+	+	+	Low
Singla et al. (2014)	RCT	+	+	+	+	+	+	+	Low
Sugimoto (2020)	RCT	+	?	+	+	-	?	+	High
Cannabis									
Irving (2018)	RCT	+	+	+	+	-	+	+	High
Probiotics									
Chen (2020)	RCT	+	?	+	?	?	?	+	Unclear
Kato et al. (2004)	RCT	+	+	?	+	+	?	+	Unclear
Krag et al. (2013)	RCT	+	-	-	+	-	-	+	High
Malchow et al. (1997)	RCT	?	?	+	+	?	+	+	Unclear
Matthes et al. (2010)	RCT	+	?	+	+	+	?	+	Unclear
Ng et al. (2010)	RCT	?	?	+	+	+	?	+	Unclear
Petersen et al. (2014)	RCT	?	+	+	?	+	?	+	Unclear
Plein and Hotz (1993)	RCT	?	?	+	?	?	-	?	High
Schultz et al. (2004)	RCT	?	?	?	?	+	+	+	Unclear
Sood et al. (2009)	RCT	+	+	?	?	-	-	+	High
Tamaki et al. (2016)	RCT	+	+	+	?	+	?	+	Unclear
Tursi et al. (2010)	RCT	+	?	+	+	+	?	+	Unclear
Vejdani et al. (2017)	RCT	+	-	-	?	+	?	+	High
<i>Remission maintenance</i>									
Whole food or elimination diets									
Albenberg (2019)	RCT	+	+	-	?	+	?	-	High
Keshteli et al. (2016)	RCT	?	?	-	?	+	-	?	High
Partial enteral nutrition									
Takagi et al. (2006)	RCT	+	+	+	+	+	+	+	Low
Triantafillidis et al. (2010)	RCT	-	-	-	-	-	-	+	High
Hanai et al. (2012)	RCT	+	?	?	?	+	+	+	Unclear
Curcumin									
Bommelaer (2020)	RCT	+	?	+	+	+	+	-	High
Hanai et al. (2012)	RCT	+	?	?	?	+	+	+	Unclear
Fibre									
Hallert et al. (1991)	RCT	?	?	+	+	+	?	+	Unclear
James et al. (2015)		+	+	-	+	+	-	-	High
Mutlu et al. (2016)	RCT	-	-	?	?	-	-	?	High

(Continues)

## Vitamin D

Jorgensen et al. (2010)	RCT	+	+	+	+	+	+	+	Low
Narula et al. (2017)	RCT	?	+	+	+	+	+	+	Low

## Probiotics

Bjarnason (2019)	RCT	+	+	+	?	?	?	+	Unclear
Matsuoka (2019)	RCT	+	+	+	+	+	?	+	Unclear
Bourreille et al. (2013)	RCT	+	+	+	+	?	+	?	Unclear
Willert et al. (2010)	RCT	?	?	?	?	?	?	+	High
Cui et al. (2004)	RCT	?	?	+	+	?	+	+	High
Wildt et al. (2011)	RCT	+	?	+	?	+	+	+	High
Yoshimatsu et al. (2015)	RCT	+	+	+	+	+	+	?	High

## Surgery

Beaupel et al. (2017)	Case control	+	?	+	+	+	+	+	High
Guo et al. (2016)	Case control	+	+	+	?	?	+	+	Unclear
Heerasing et al. (2014)	Case control	+	+	?	+	+	+	+	Low
Li et al. (2014)	Case control	+	?	+	+	?	+	+	Unclear
Li et al. (2015)	Case control	+	+	?	?	?	+	+	Unclear
Meade (2022)	Case control	+	?	?	?	?	?	+	Unclear
Wang et al. (2016)	Case control	+	?	+	+	+	+	+	Low
Zhang et al. (2017)	Case control	+	?	+	+	+	+	+	Unclear
Zhu et al. (2017)	Case control	+	?	+	+	+	+	+	Unclear
Yamamoto et al. (2020)	Case control	+	+	?	+	+	+	+	Unclear

## Strictures

Hu et al. (2017)	Case series	?	?	+	+	+	?	+	Unclear
Teahon et al. (1990)	Case series	?	+	+	+	+	?	+	High
Xie et al. (2010)	Case series	+	?	+	+	+	+	+	High
Yang et al. (2017)	Case series	+	?	?	+	+	+	+	Unclear

## Pouchitis

Gionchetti et al. (2000)	RCT	+	+	+	+	+	+	+	Low
Gionchetti et al. (2003)	RCT	+	+	+	+	+	+	+	Low
Mimura et al. (2004)	RCT	+	+	+	+	+	+	+	Low
Pronio et al. (2008)	RCT	?	?	+	+	+	+	+	High
Brown et al. (2004)	RCT	?	?	+	+	+	?	+	Unclear

## Fistula

Yan et al. (2014)	Case series	?	?	+	+	+	?	+	High
Yang et al. (2017)	Case series	+	?	?	+	+	+	+	Unclear

## Functional symptoms

Cox et al. (2018)	RCT	?	?	?	?	?	?	+	Unclear
Halmos et al. (2016)	RCT	+	?	+	+	+	?	+	Unclear
Pedersen et al. (2017)	RCT	+	+	+	+	+	?	+	High

## Orofacial granulomatosis

White et al. (2006)	Case control	?	+	?	+	?	+	+	High
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## Key

Low bias	+
Unclear bias	?
High bias	+

Note: For RCTs, there are seven risk of bias questions; for cohort studies, there are eight risk of bias questions; for case-control studies, there are five risk of bias questions; for case series, there are six risk of bias questions.

Abbreviation: RCT, randomised controlled trial.



TABLE 2 Grade recommendations and % consensus

GRADE recommendations	% consensus
<b>Nutrition assessment</b>	
Fibre intake is likely to be low in inflammatory bowel disease and should be included as part of a nutritional assessment. (GRADE very low quality)	88.1
Calcium intake should be assessed in Crohn's disease and ulcerative colitis patients because patients may not meet their recommended intake. (GRADE very low quality)	96.3
Iron intake should be assessed in Crohn's disease and ulcerative colitis patients as patients may not meet their recommended intake. (GRADE very low quality)	96.2
<b>Nutrition screening</b>	
MUST may be used to screen patients with inflammatory bowel disease for risk of malnutrition. (GRADE very low quality)	85.5
<b>Induction of remission</b>	
To induce remission in active Crohn's disease, exclusive enteral nutrition (EEN) is less effective than corticosteroids. EEN may be used in mild to moderate disease where avoidance of corticosteroids is intended, and dietetic expertise is available. (GRADE very low quality)	84.6
In some patients with mildly active ulcerative colitis, taking specific probiotics alongside usual medication may support induction of disease remission. (GRADE moderate quality)	88.7
<b>Remission maintenance</b>	
There is insufficient evidence to support the use of high dose vitamin D for maintaining remission in Crohn's disease. (GRADE very low quality)	81.6
Partial enteral nutrition alongside routine medication may support Crohn's disease remission maintenance. (GRADE very low quality)	88.0
A probiotic mixture of 8 bacterial strains may maintain remission in chronic relapsing pouchitis. Very limited evidence may support prophylactic use after pouch formation surgery to prevent initial pouchitis onset. There is no evidence to support the use of other probiotics to maintain remission in pouchitis. (GRADE low quality)	85.4
<b>Surgery</b>	
There is limited evidence that pre-surgical exclusive enteral nutrition may reduce the length of post-surgical hospitalisation in patients with Crohn's disease. (GRADE very low quality)	84.3
Pre-surgical exclusive enteral nutrition may lower the risk of post-surgical infectious complications in patients with Crohn's disease. (GRADE very low quality)	92.0
<b>Strictureing disease</b>	
Exclusive enteral nutrition for 4–12 weeks may induce remission in Crohn's disease patients with inflammatory strictures. (GRADE very low quality)	84.3
<b>Fistulating disease</b>	
There is very limited evidence that fistulating Crohn's disease may respond to exclusive enteral nutrition. (GRADE very low quality)	82.0
<b>Functional symptoms</b>	
A low FODMAP diet may improve global functional bowel symptoms in quiescent or mildly active inflammatory bowel disease. (GRADE very low quality)	90.2

Abbreviations: CD, Crohn's disease; IBD, inflammatory bowel disease; UC, ulcerative colitis.

similar between patients with UC in remission ( $n = 37$ ) and healthy controls ( $n = 68$ ). One study in active UC found BMI was lower in patients ( $n = 53$ ) compared to healthy controls ( $n = 30$ ).<sup>29</sup>

Four studies assessed BMI in active UC and UC in remission.<sup>24,27,28,34</sup> Two studies found similar BMI between patients ( $n = 41$ ) and healthy controls ( $n = 43$ )<sup>28,34</sup> and two studies found BMI was lower in

active UC and UC in remission ( $n = 106$ ) compared to healthy controls ( $n = 106$ ).<sup>24,27</sup>

## IBD

Four studies assessed BMI in IBD patients.<sup>25,29,31,32</sup> Three studies found BMI was significantly lower in active



IBD and IBD in remission ( $n = 303$ ) compared to healthy controls ( $n = 1540$ )<sup>25,29,31</sup> and one found BMI was similar between patients with IBD in remission ( $n = 150$ ) and healthy controls ( $n = 100$ ).<sup>32</sup>

## Fat mass

Seventeen studies assessed fat mass in 920 CD patients, 195 UC patients and 983 healthy controls.<sup>8,14–24,27,33,35–37</sup>

### Crohn's disease

Two studies assessed fat mass in active CD.<sup>8,18</sup> There were 56 CD patients and 120 healthy controls in the analysis. One study showed that fat mass was significant lower in active CD compared to healthy controls,<sup>18</sup> whereas the other study did not.<sup>8</sup>

Nine studies assessed fat mass in CD in remission.<sup>16–22,33,35</sup> Fat mass was significantly lower in CD in remission ( $n = 115$ ) compared to healthy controls ( $n = 100$ ) in three studies<sup>19–21</sup> and the remaining six studies found no differences between groups: patients ( $n = 403$ ) and healthy controls ( $n = 356$ ).<sup>16–18,22,33,35</sup>

Six studies assessed fat mass in active CD and CD in remission.<sup>14,15,23,24,27,36</sup> There were no differences in fat mass between the patients ( $n = 143$ ) and healthy controls ( $n = 143$ ) in three studies<sup>23,24,27</sup> and fat mass was lower in patients ( $n = 246$ ) compared to healthy controls ( $n = 341$ ) in the other three studies.<sup>14,15,36</sup>

### Ulcerative colitis

Two studies assessed fat mass in UC in remission.<sup>16,20</sup> There were no differences in fat mass between the patients ( $n = 16$ ) and healthy controls ( $n = 20$ ) in one study<sup>20</sup> and fat mass was higher in patients ( $n = 50$ ) compared to healthy controls ( $n = 61$ ) in the other.<sup>16</sup>

Three studies assessed fat mass in active UC and UC in remission.<sup>24,27,37</sup> All studies found no differences in fat mass between the patients ( $n = 129$ ) and healthy controls ( $n = 106$ ).

## Percentage fat mass

Fifteen studies assessed percentage fat mass in IBD with 664 CD patients, 190 UC patients and 675 healthy controls.<sup>7,12,13,17–20,22–24,27,29,33,35,38</sup>

### Crohn's disease

Two studies assessed percentage fat mass in active CD ( $n = 152$ ) and showed that percentage fat mass was

significantly lower compared to healthy controls ( $n = 130$ ).<sup>18,29</sup>

Eight studies assessed percentage fat mass in CD in remission. Two studies showed that percentage fat mass was significantly lower in patients ( $n = 61$ ) compared to healthy controls ( $n = 75$ ),<sup>19,20</sup> one study showed that percentage fat mass was significantly higher in patients ( $n = 31$ ) compared to healthy controls ( $n = 98$ )<sup>35</sup> and the remaining five studies showed similar percentage fat mass between groups: patients ( $n = 299$ ) and healthy controls ( $n = 272$ ).<sup>17,18,22,33,38</sup>

Six studies assessed percentage fat mass in active CD and CD in remission. Two studies showed that percentage fat mass was significantly higher in patients ( $n = 51$ ) compared to healthy controls ( $n = 39$ ).<sup>7,12</sup>

And the remaining four studies showed similar percentage fat mass between groups: patients ( $n = 243$ ) and healthy controls ( $n = 199$ ).<sup>13,23,24,27</sup>

## Ulcerative colitis

One study assessed percentage fat mass in active UC and showed it was lower in patients ( $n = 53$ ) compared to healthy controls ( $n = 30$ ).<sup>29</sup>

Two studies assessed percentage fat mass in UC in remission and showed no differences between patients ( $n = 31$ ) and healthy controls ( $n = 85$ ).<sup>20,38</sup>

Two studies assessed percentage fat mass in active UC and UC in remission. One study showed that percentage fat mass was significantly higher in patients ( $n = 60$ ) compared to healthy controls ( $n = 60$ )<sup>27</sup> and the other study showed similar percentage fat mass between groups: patients ( $n = 46$ ) and healthy controls ( $n = 46$ ).<sup>24</sup>

## Fat free mass

Fat free mass was measured in 18 studies<sup>8,9,14–24,27,33,35,37,38</sup> in 836 CD patients, 210 UC patients and 814 healthy controls and analysis demonstrated inconsistent findings.

### Crohn's disease

Two studies assessed fat free mass in active CD and both found that fat free mass was significantly lower in patients ( $n = 1366$ ) compared to healthy controls ( $n = 120$ ).<sup>8,18</sup>

Ten studies assessed fat free mass in CD in remission. Two studies found fat free mass was significantly lower in patients ( $n = 154$ ) compared to healthy controls ( $n = 188$ ),<sup>18,35</sup> and one study reported lower fat free mass for males ( $n = 33$ ) but not females ( $n = 61$ ) when patients were compared with healthy controls (males  $n = 20$ , females  $n = 41$ ).<sup>16</sup> The remaining seven studies found fat free mass was similar between patients ( $n = 291$ ) and healthy controls ( $n = 272$ ).<sup>17,19–22,33,38</sup>

Six studies assessed fat free mass in a mixed population of active CD and CD in remission. Four studies found that fat free mass differed between patients ( $n = 179$ ) and healthy controls ( $n = 170$ ).<sup>9,24,27</sup>

The most recent study separated patients into those on conventional medication ( $n = 22$ ) and those on a biologic ( $n = 23$ ) and, when compared with healthy controls ( $n = 20$ ), found lower fat free mass for patients on conventional medication but not on a biologic.<sup>9</sup>

One study found that patients with newly diagnosed CD ( $n = 20$ ) had higher fat free mass compared to healthy controls ( $n = 20$ ) but patients who had CD for more than 5 years ( $n = 30$ ) had lower fat free mass compared to healthy controls ( $n = 40$ ).<sup>23</sup>

One study analysed patients as one group and found higher fat free mass in the patients ( $n = 23$ ) compared to healthy controls ( $n = 23$ )<sup>24</sup> and another found that fat free mass was lower in the patients ( $n = 60$ ) compared to healthy controls ( $n = 60$ ).<sup>27</sup> The remaining two studies found no differences in fat free mass between groups (patients  $n = 96$ , healthy controls  $n = 87$ ).<sup>14,15</sup>

## Ulcerative colitis

No studies assessed fat free mass in active UC.

Three studies assessed fat free mass in UC in remission. In two studies, there was no difference between patients ( $n = 31$ ) and healthy controls ( $n = 85$ ).<sup>20,38</sup> In the third study, male patients ( $n = 17$ ) had lower fat free mass compared to male healthy controls ( $n = 20$ ) but female patients ( $n = 33$ ) had similar fat free mass compared to healthy controls ( $n = 41$ ).<sup>16</sup>

Three studies assessed fat free mass in a mixed population of active UC and UC in remission and found no differences in fat free mass between patients ( $n = 129$ ) and healthy controls ( $n = 106$ ).<sup>24,27,37</sup>

## Percentage fat free mass

Two studies assessed percentage fat free mass in 61 CD patients in remission, 16 UC patients in remission and 75 healthy controls.<sup>19,20</sup> One study showed that percentage fat free mass was lower in patients ( $n = 43$ ) compared to healthy controls ( $n = 55$ ),<sup>19</sup> whereas the other study showed similar percentage fat free mass between groups: patients ( $n = 18$ ) and healthy controls ( $n = 20$ ).<sup>20</sup>

No studies in UC were identified.

## Visceral adipose tissue

Three studies assessed visceral adipose tissue in a mixed population of active CD and CD in remission with 101

CD patients and 67 healthy controls.<sup>7,12,13</sup> All studies found visceral adipose tissue was higher in patients compared to healthy controls.

No studies in UC were identified.

## Waist to hip ratio

Two studies assessed waist to hip ratio, one in CD in remission<sup>22</sup> and the other in a mixed population of active CD and CD in remission.<sup>12</sup> Fifty patients with CD and 52 healthy controls were included in the analysis. Both studies found no difference in waist to hip ratio between patients and healthy controls.<sup>12,22</sup>

## Muscle strength

In health, muscle mass is a strong predictor of muscle strength. In IBD, muscle strength is likely to be affected by disease activity. Longitudinal assessment of muscle function can be useful for assessing long-term effects of disease activity on muscle strength and endurance.

Handgrip and lower limb strength are reliable measures of muscle function; however, in clinical practice, handgrip strength is more practical, quicker and cheap assessment tool compared to assessment of lower limb strength.

Muscle strength was assessed in seven studies.<sup>15-17,22,24,36,39</sup> There were 437 patients with CD, 96 patients with UC and 491 healthy controls with inconsistent results.

## Crohn's disease

One study found hamstring and quadricep muscle strength was lower in male patients with CD in remission ( $n = 14$ ) compared to healthy controls ( $n = 14$ ) but there was no difference in female patients ( $n = 18$ ) and healthy controls ( $n = 18$ ).<sup>22</sup>

One study assessed hamstring and quadricep muscle strength in a mixed population of active CD and CD in remission and found similar strength between patients ( $n = 23$ ) and healthy controls ( $n = 23$ ).<sup>24</sup>

One study found knee extension and flexion peak torque were lower in patients with CD in remission ( $n = 19$ ) compared to healthy controls ( $n = 19$ ) and found fatigues rates were higher in patients compared to healthy controls.<sup>39</sup>

Two studies assessed handgrip strength in patients with CD in remission. One study found handgrip strength to be lower in patients ( $n = 94$ ) compared to healthy controls ( $n = 61$ ),<sup>16</sup> whereas the other found handgrip strength was lower in male patients ( $n = 17$ ) compared to male healthy controls ( $n = 10$ )

but not female patients ( $n = 24$ ) compared to female healthy controls ( $n = 15$ ).<sup>17</sup> The latter study found that handgrip endurance was lower in female patients but not male patients compared to healthy controls.

Two studies assessed handgrip strength in a mixed population of active CD and CD in remission and found handgrip strength was lower in patients ( $n = 78$ ) compared to healthy controls ( $n = 75$ ).<sup>15,36</sup>

## Ulcerative colitis

One study assessed hamstring and quadricep muscle strength in a mixed population of active UC and UC in remission and found similar strength between patients ( $n = 46$ ) and healthy controls ( $n = 46$ ).<sup>24</sup>

One study in UC in remission assessed handgrip strength and found that it was lower in patients ( $n = 50$ ) compared to healthy controls ( $n = 61$ ).<sup>16</sup>

## Practice tips

Table 3 provides information on available nutritional assessment anthropometric tools. A practical guide on how to use different anthropometric tools in nutritional assessment is presented elsewhere.<sup>41</sup> Suboptimal mean age and sex specific handgrip strength values are provided in Tables 3–5.

Multidisciplinary team meetings are a vital part of the patient care pathway. From a nutritional assessment perspective, body composition, muscle strength and bone mineral density may be altered, particularly when patients are taking corticosteroids, thus nutritional concerns and management should be discussed as part of the IBD MD team.<sup>47</sup>

## NUTRITION ASSESSMENT: MICRONUTRIENTS

**PRACTICE STATEMENT:** In inflammatory bowel disease, clinical interpretation of some serum micronutrients may be unreliable during inflammation. Agreement 96.2%.

One study has provided what C-reactive protein (CRP) levels are useful for clinical interpretation of certain micronutrients<sup>48</sup>:

CRP < 20 mg L<sup>-1</sup> plasma zinc;  
CRP < 10 mg L<sup>-1</sup> plasma selenium, vitamins A and D;  
CRP < 5 mg L<sup>-1</sup> vitamin B6 and vitamin C

This means that where CRP is above the cut-off provided, the micronutrient level may not be accurate. Further information is available.<sup>49,50</sup>

**PRACTICE STATEMENT:** A comprehensive nutritional assessment in inflammatory bowel disease patients includes the following serum micronutrients: folic acid, vitamin B<sub>12</sub>, vitamin D, iron, zinc, magnesium and selenium. Review recent tests and determine the frequency of monitoring depending on disease activity, dietary intake and micronutrient supplementation. Agreement 90.2%.

In clinical practice, only patients with micronutrient deficiencies are treated with supplementation, however, most studies only report serum concentrations of

**TABLE 3** Tools for nutritional assessment in inflammatory bowel disease

Body mass index (BMI)	BMI uses weight and height to calculate if weight is healthy. When used in isolation, BMI provides limited data for nutritional assessment. Relying on weight and BMI may mask IBD related changes in fat mass and fat-free mass
Handgrip strength	A reliable measure of muscle strength and muscle reserve
Mid-upper arm circumference (MUAC)	A measure of nutritional depletion that is less affected by fluid status
Tricep skinfold thickness (TSF)	A measure of fat mass that is more reliable than BMI
Mid-arm muscle circumference	A measure of fat-free mass derived from MUAC and TSF
Bioelectrical impedance	A measure of fat mass and fat-free mass
Waist to hip ratio	A measure of abdominal fat
Dual-energy X-ray absorptiometry	A measure of bone mineral density
Novel radiological scans	Computerised tomography <sup>40</sup> and magnetic resonance imaging can be used to measure abdominal fat mass

Note: Further practical advice is available in Sandall et al.<sup>41</sup>

TABLE 4 Evaluation of hand grip strength (HGS)

Males			Females		
Age (years) <sup>42</sup>	Mean HGS (kg)	Suboptimal	Age <sup>42</sup>	Mean HGS (kg)	Suboptimal
15–19	29.6	<25.2	15–19	23.9	<20.3
20–24	41.5	<35.3	20–24	28.4	<24.1
25–29	48.8	<41.5	25–29	30.6	<26.0
30–34	51.6	<43.9	30–34	31.4	<26.7
35–39	51.6	<43.9	35–39	31.3	<26.6
40–44	50.3	<42.8	40–44	30.7	<26.1
45–49	48.8	<41.5	45–49	29.9	<25.4
50–54	47.6	<40.5	50–54	28.7	<24.4
55–59	46.2	<39.3	55–59	27.5	<23.4
60–64	44.6	<37.9	60–64	26.5	<22.5
65–69	42.3	<36.0	65–69	25.3	<21.5
70–74	39.1	<33.2	70–74	23.5	<20.0
75–79	35.6	<30.3	75–79	21.4	<18.2
80–84	32.2	<27.4	80–84	19.1	<16.2
85–89	28.5	<24.2	85–89	16.6	<14.1
90–94	24.7	<21.0	90–94	14.2	<12.1
95+	No data		95+	No data	

Age and sex specific mean values for the maximum value of available attempts taken with permission from Dodds et al.<sup>43</sup> Suboptimal HGS is mean HGS (three attempts) that is less than 85% of the mean for age and sex.<sup>44</sup>

micronutrients and not deficiency rates which is a major limitation of the current research literature. Only two studies report on micronutrient deficiency rates for patients and healthy controls.<sup>16,51</sup> The nutrients selected are based on the available literature.

## Folic acid

Five studies assessed serum folic acid and/or folic acid deficiency rates.<sup>16,22,24,51,52</sup>

The studies included 283 patients with CD, 189 patients with UC and 272 healthy controls. One study reported that they excluded patients who were receiving folic acid supplementation within the previous 6 months,<sup>52</sup> two studies included patients who took folic acid supplements<sup>16,22</sup> and two studies did not describe whether patients received folic acid supplementation or not.<sup>24,51</sup>

## Crohn's disease

All four studies demonstrated there were no differences in serum folic acid levels between healthy controls and patients with CD in remission<sup>22</sup> or mixed populations of patients with active CD or CD in remission.<sup>24,51,52</sup>

One study found similar folic acid deficiency rates for CD patients in remission in one of 94 (1.0%) and no healthy controls (0%) ( $p > 0.05$ ).<sup>16</sup> Another study reported folic acid deficiency rates were significantly higher

TABLE 5 Recommendations for monitoring of micronutrient status in IBD

Nutrient	Remission	Active disease (outpatients)	Reference
Iron	Full blood count, serum ferritin, C-reactive protein every 6–12 months	Every 3 months	Dignass et al. <sup>45</sup>
Folic acid	Annually (if at risk – small bowel disease or resection). Sulfasalazine and methotrexate impair folate absorption	Annually (if at risk – small bowel disease or resection)	Dignass et al. <sup>45</sup>
Vitamin B <sub>12</sub>	Annually (if at risk – ileocaecal resection, vegan or avoiding meat and dairy)	Annually (if at risk – small bowel disease or resection)	Sandall et al., <sup>41</sup> Dignass et al. <sup>45</sup>
Folic acid and vitamin B <sub>12</sub>	Every 3–6 months (if at risk – small bowel disease or resection)	Every 3–6 months (if at risk – small bowel disease or small bowel resection)	Maaser et al. <sup>46</sup>
Vitamin D	Seasonal variation will influence interpretation	Measure in those with active disease – supplement and then re-measure to check they have responded	Sandall et al., <sup>41</sup> Maaser et al. <sup>46</sup>
Vitamin K, selenium, vitamin A, vitamin C, zinc, vitamin B <sub>6</sub> , vitamin B <sub>1</sub>	Malabsorption parameters should be assessed at regular intervals in all patients with IBD  Only in clinical scenario that could be exacerbated by micronutrient deficiency (e.g., poor wound healing) or in patients at greater risk (e.g., small bowel disease or resection)		Maaser et al. <sup>46</sup>

Abbreviation: IBD, inflammatory bowel disease.

at 30 of 105 (28.8%) in CD patients compared to two of 60 (3.7%) in healthy controls ( $p < 0.001$ ).<sup>51</sup>

## Ulcerative colitis

Two studies assessed serum folic acid levels between healthy controls and mixed populations of patients with active UC or UC in remission and found no differences between groups.<sup>24,51</sup>

One study found similar folic acid deficiency rates for UC patients in remission (1/54 [2.0%]) and no healthy controls (0%) ( $p > 0.05$ ).<sup>16</sup> One study reported folic acid deficiency rates were eight of 99 (8.6%) in UC patients compared to two of 60 (3.7%) in healthy controls, these rates were not significantly different ( $p = 0.278$ ).<sup>51</sup>

## Vitamin B<sub>12</sub>

Five studies assessed serum vitamin B<sub>12</sub> and/or vitamin B<sub>12</sub> deficiency rates.<sup>16,22,24,51,52</sup>

Micronutrient deficiency rates were reported in CD patients and healthy controls in two studies.<sup>16,51</sup>

The studies included 283 patients with CD, 189 patients with UC and 272 healthy controls. One study reported that they excluded patients who had received vitamin B<sub>12</sub> supplementation within the previous 6 months,<sup>52</sup> two studies included patients who had received intramuscular vitamin B<sub>12</sub><sup>16,22</sup> and two studies did not report whether patients did or did not receive vitamin B<sub>12</sub> supplementation.<sup>24,51</sup>

## Crohn's disease

One study reported similar serum vitamin B<sub>12</sub> levels between CD patients ( $n = 32$ ) in remission and healthy controls ( $n = 32$ ).<sup>22</sup> Another study also reported similar serum vitamin B<sub>12</sub> levels but included a mixed population ( $n = 45$ ) of active CD and CD patients in remission compared to healthy controls ( $n = 53$ ).<sup>51</sup> Two studies reported significantly lower serum vitamin B<sub>12</sub> levels for mixed populations of patients ( $n = 112$ ) with active CD or CD in remission compared to healthy controls ( $n = 126$ ).<sup>24,52</sup>

One study found similar vitamin B<sub>12</sub> deficiency rates for CD patients in remission (9/94 [9.6%]) and no healthy controls (0%) ( $p > 0.05$ ).<sup>16</sup> One study reported vitamin B<sub>12</sub> deficiency rates were significantly higher at 10 of 45 (22.2%) in a mixed population of CD patients compared to four of 53 (7.5%) in healthy controls ( $p = 0.039$ ).<sup>51</sup> rates.

## Ulcerative colitis

Two studies assessed serum vitamin B<sub>12</sub> levels between mixed populations of patients with active UC or UC in

remission ( $n = 139$ ) and healthy controls ( $n = 76$ ) and found no differences between groups.<sup>24,51</sup>

One study found similar vitamin B<sub>12</sub> deficiency rates for UC patients in remission (4/50 [8.0%]) and no healthy controls (0%) ( $p > 0.05$ ).<sup>16</sup> One study reported similar vitamin B<sub>12</sub> deficiency rates of seven of 93 (7.5%) for mixed populations of patients with active UC or UC in remission and healthy controls (4/53 [7.5%]) ( $p = 0.971$ ).<sup>51</sup>

## Vitamin D

There are 11 studies (five in remission and six in mixed IBD) measuring vitamin D status.

## Crohn's disease

Eleven studies, five in remission<sup>10,26,38,39,53</sup> and six in mixed CD, were included.<sup>54–59</sup>

Five studies showed that CD with active disease or CD in remission ( $n = 267$ ) did have significantly lower vitamin D compared to healthy controls ( $n = 394$ )<sup>10,53,55,56,59</sup> and six studies showed similar levels (CD,  $n = 239$ ; HC,  $n = 247$ ).<sup>26,38,39,54,57,58</sup>

Nine studies assessed vitamin D deficiency using different cut offs for suboptimal vitamin D status:

Five studies used  $<50 \text{ nmol L}^{-1}$  and one study found significantly more CD patients (27/34 [79%]) had vitamin D deficiency compared to healthy controls (17/34 [50%]) ( $p < 0.05$ ).<sup>56</sup> and the others did not report a difference.<sup>26,39,55,57</sup>

Two studies in CD in remission used  $<40 \text{ nmol L}^{-1}$ . One found more patients [9/47 [19.1%]]<sup>10</sup> had a higher vitamin D deficiency rate compared to healthy controls (2/47 [4.3%]) ( $p < 0.05$ ) and the other found similar rates.<sup>53</sup>

One study used  $<25 \text{ nmol L}^{-1}$  and found similar rates of vitamin D deficiency between patients ( $n = 33$ ) and healthy controls ( $n = 15$ ).<sup>58</sup>

One study used  $<25 \text{ nmol L}^{-1}$  in winter and  $<70 \text{ nmol L}^{-1}$  in summer/autumn and found significantly more patients with CD in remission (18/32 [56%]) had vitamin D deficiency compared to healthy controls (9/32 [28%]) ( $p < 0.01$ ).<sup>22</sup>

## Ulcerative colitis

Five studies, three in remission UC<sup>10,26,38</sup> and two in mixed UC<sup>55,59</sup> were included.

Three studies showed that vitamin D in active UC or UC in remission ( $n = 189$ ) was lower than healthy controls ( $n = 316$ )<sup>10,55,59</sup> and two studies showed similar levels (UC,  $n = 36$ ; HC,  $n = 113$ ).<sup>26,38</sup>

Three studies assessed vitamin D deficiency using different cut offs for suboptimal vitamin D status.



Two studies used  $<50 \text{ nmol L}^{-1}$  and both found similar vitamin D deficiency rates between patients with UC and healthy controls.<sup>10,26,38,39,53–59</sup>

One study in UC in remission used  $<40 \text{ nmol L}^{-1}$  and found similar vitamin D deficiency rates between patients with UC and healthy controls.<sup>10,53,55,56,59</sup>

## Zinc

Ten studies assessed serum zinc or zinc deficiency rates.<sup>11,16,22–24,60–64</sup> There were 375 CD patients, 398 UC patients and 451 healthy controls.

Zinc is mostly intracellular and disease activity and  $\text{CRP} > 20 \text{ mg L}^{-1}$  will limit interpretation in IBD.

## Crohn's disease

One study in active CD showed no difference in serum zinc levels between patients ( $n = 22$ ) and healthy controls ( $n = 11$ ).<sup>63</sup> Two studies showed lower serum zinc levels for patients in remission ( $n = 32$ )<sup>22</sup> and patients with long standing CD in remission ( $n = 32$ ) compared to healthy controls ( $n = 64$ ).<sup>23</sup> However newly diagnosed patients with CD ( $n = 20$ ) in remission had similar zinc levels compared to healthy controls ( $n = 20$ ).<sup>23</sup> Two studies with mixed populations of active CD or CD in remission ( $n = 70$ ) showed similar serum zinc levels compared to healthy controls ( $n = 146$ ).<sup>24,61</sup>

One study showed rates of zinc deficiency were similar between patients with CD in remission (4/94 [4.2%]) and no healthy controls (0%).<sup>16</sup>

## Ulcerative colitis

Four studies in mixed populations of active UC or UC in remission assessed serum zinc levels. Two studies showed higher zinc in patients ( $n = 198$ ) compared to healthy controls ( $n = 150$ ),<sup>61,62</sup> one study found similar levels between groups (UC,  $n = 24$ ; HC,  $n = 10$ )<sup>64</sup> and one study found serum zinc levels lower in UC patients ( $n = 46$ ) compared to healthy controls ( $n = 23$ ).<sup>24</sup>

One study showed rates of zinc deficiency were similar between patients with UC in remission (1/50 [2.0%]) and no healthy controls (0%).<sup>16</sup>

## IBD

Two studies did not separate patients with CD and patients with UC. Both showed serum zinc levels were significantly higher in patients compared to healthy controls, one was carried out in active IBD<sup>60</sup> and the

other was in a mixed population of patients with active IBD or IBD in remission.<sup>11</sup>

## Copper

Four studies assessed serum copper levels.<sup>22,24,61,63</sup> There were 113 CD patients, 163 UC patients and 200 healthy controls.

## Crohn's disease

One study in active CD<sup>63</sup> and one study in CD in remission<sup>22</sup> showed no difference in serum copper levels between patients ( $n = 43$ ) and healthy controls ( $n = 54$ ). Two studies with mixed populations of active CD or CD in remission showed differing results with similar serum copper levels between patients ( $n = 23$ ) and healthy controls ( $n = 23$ ) in one study<sup>24</sup> and higher serum copper levels in patients ( $n = 47$ ) compared to healthy controls ( $n = 123$ ) in the other study.<sup>61</sup>

No copper deficiency rates were reported.

## Ulcerative colitis

Two studies with mixed populations of active UC or UC in remission showed differing results with similar serum copper levels between patients ( $n = 46$ ) and healthy controls ( $n = 23$ ) in one study<sup>24</sup> and higher serum copper levels in patients ( $n = 117$ ) compared to healthy controls ( $n = 123$ ) in the other study.<sup>61</sup>

No copper deficiency rates were reported.

## Magnesium

Three studies assessed serum magnesium levels or magnesium deficiency rates.<sup>16,22,24</sup> There were 149 CD patients, 96 UC patients and 126 healthy controls included in the analysis.

## Crohn's disease

One study assessed serum magnesium levels in CD patients in remission and showed significantly lower levels of magnesium in all patients ( $n = 32$ ) compared to healthy controls ( $n = n = 32$ ).<sup>22</sup>

One study in a mixed population of active CD and CD in remission showed similar magnesium levels between patients and healthy controls.<sup>24</sup>

Magnesium deficiency was 27 of 94 (28.7%) in patients with CD in remission and four of 61 (6.6%) in healthy controls ( $p > 0.05$ ).<sup>16</sup>

## Ulcerative colitis

One study assessed serum magnesium levels in a mixed population of active UC and UC in remission and showed significantly lower levels of magnesium in patients compared to healthy controls.<sup>24</sup>

Magnesium deficiency was 11 of 50 (22.0%) in patients with UC in remission and 6.6% in healthy controls. This was significantly different to healthy controls for female patients ( $p = 0.033$ ) but not male patients ( $p > 0.05$ ).<sup>16</sup>

## Selenium

Eight studies assessed serum selenium levels or selenium deficiency rates.<sup>11,16,22,24,61,64–66</sup> There were 684 CD patients, 328 UC patients and 1214 healthy controls.

## Crohn's disease

One study in CD in remission showed lower selenium levels for patients ( $n = 32$ ) compared to healthy controls ( $n = 32$ ).<sup>22</sup> Four studies included mixed populations of patients with active CD or CD in remission, three of these showed lower selenium levels for patients ( $n = 435$ ) compared to healthy controls ( $n = 1013$ )<sup>61,65,66</sup> and one study showed no differences between groups (patients,  $n = 23$ ; HC,  $n = 23$ ).<sup>24</sup>

One study showed high rates of selenium deficiency in patients with CD in remission (58/94 [61.7%]) and healthy controls (19/61 [31.1%]) with a significant difference between groups for males ( $p < 0.001$ ) but not females ( $p > 0.05$ ).<sup>16</sup>

## Ulcerative colitis

Three studies assessed serum selenium levels in mixed populations of patients with active UC or UC in remission. Two studies showed significantly lower selenium levels in patients ( $n = 46$ ) compared to healthy controls ( $n = 23$ )<sup>24,64</sup> and one study showed similar levels between groups (patients,  $n = 117$ ; HC,  $n = 123$ ).<sup>61</sup>

One study showed no difference in rates of selenium deficiency in patients with CD in remission (20/50 [40.0%]) and healthy controls (19/61 [31.1%]) ( $p > 0.05$ ).<sup>16</sup>

## IBD

One study in patients with IBD failed to show any difference in serum selenium levels for comparisons between active IBD or IBD in remission ( $n = 167$ ) and healthy controls ( $n = 45$ ).<sup>11</sup>

## Iron

**PRACTICE STATEMENT:** Iron is an acute phase reactant therefore interpretation of blood test results should consider inflammatory status. Anaemia of chronic disease often co-exists with iron deficiency anaemia in inflammatory bowel disease. Agreement 94.4%.

Use serum ferritin, CRP, transferrin saturation and serum iron to assess presence of iron deficiency and haemoglobin, blood count and other micronutrients when diagnosing iron deficiency anaemia.

Three studies assessed serum iron levels.<sup>29,64,67</sup> The analysis included 191 patients with CD, 287 patients with UC and 280 healthy controls. No studies assessed iron deficiency rates in IBD compared to healthy controls.

## Crohn's disease

Two studies in patients with CD showed significantly lower iron levels for active disease ( $n = 110$ )<sup>29,67</sup> and remission<sup>67</sup> compared to healthy controls ( $n = 135$ ).

## Ulcerative colitis

Three studies in active UC show serum iron levels are significantly lower in patients ( $n = 162$ ) compared to healthy controls ( $n = 145$ ).<sup>29,64,67</sup> In UC in remission results are inconsistent with one study showing iron levels are significantly lower in UC ( $n = 8$ ) than in healthy controls ( $n = 10$ )<sup>64</sup> and the other showing similar levels between groups (UC,  $n = 93$ ; HC,  $n = 105$ ).<sup>67</sup>

## NUTRITIONAL ASSESSMENT: ENERGY AND MACRONUTRIENTS

**PRACTICE STATEMENT:** Dietary intake in inflammatory bowel disease (IBD) may be affected by differing food choices during periods of disease activity and remission and is an essential part of nutritional assessment in inflammatory bowel disease. Agreement 100%.

Suboptimal dietary intake is one of the contributing factors to reduced nutritional status in IBD. Estimates of dietary intake using a food diary, diet history or 24-h



recall are achievable in clinical practice. Dietary restriction is common in patients with active disease and in remission and is likely to affect diet quality.<sup>68</sup> Probing questions regarding dietary avoidances enable an appreciation of dietary restrictions that may be associated with deficits in nutritional intake. Dietary intakes of energy, macronutrients and calcium and iron intake were assessed.

**PRACTICE STATEMENT:** Energy and macronutrient intake may be altered in inflammatory bowel disease and should be included as part of a nutritional assessment. Agreement 92.2%.

There were 15 studies that assessed energy intake<sup>12,18,19,21–25,31,32,34,69–72</sup> with 856 patients with CD, 443 patients with UC and 2554 healthy controls. All studies included patients with CD and seven studies included patients with UC. Active IBD and IBD in remission were included in eight studies and seven studies included patients only in remission.

## Energy

Similar energy intakes compared to healthy controls ( $n = 1940$ ) were observed in eight of 15 studies in CD patients ( $n = 572$ )<sup>12,21–24,31,69,72</sup> and four of seven studies in UC patients ( $n = 258$ ) compared to 1607 healthy controls.<sup>24,31,34,72</sup> Lower energy intakes compared to healthy controls ( $n = 767$ ) were found in six of 14 studies in CD patients ( $n = 381$ )<sup>18,19,25,32,70,71</sup> and three of six studies in UC patients ( $n = 167$ ) compared to healthy controls ( $n = 527$ ).<sup>25,32,70</sup> One study found that CD patients ( $n = 20$ ) had significantly higher energy intake compared to healthy controls ( $n = 31$ ) but this study had a small sample size and these patients also had significantly higher protein and carbohydrate intakes.<sup>34</sup>

## Protein

Fourteen studies assessed protein intake<sup>12,18,19,21–25,31,34,69–72</sup> with 725 patients with CD, 359 patients with UC and 2474 healthy controls.

Eight of studies showed there was no difference in protein intakes between patients with CD ( $n = 539$ ) and healthy controls ( $n = 487$ ),<sup>19,21–24,69,71</sup> and patients with UC ( $n = 161$ )<sup>24</sup> and healthy controls ( $n = 107$ ). Four studies showed significantly lower intakes for patients with CD ( $n = 187$ ) compared to healthy controls ( $n = 547$ ),<sup>12,18,25,70</sup> two studies showed significantly lower intakes for UC patients ( $n = 101$ ) compared to healthy controls ( $n = 427$ )<sup>25,70</sup> and two studies showed a higher percentage of total energy from protein for CD ( $n = 111$ ) and UC ( $n = 97$ ) patients compared to healthy controls ( $n = 1500$ ).<sup>31,34</sup>

## Fat

There were 15 studies that assessed fat intake<sup>12,18,19,21–24,28,31,32,34,69–72</sup> with 849 patients with CD, 400 patients with UC and 2525 healthy controls. Fourteen studies included patients with CD and seven studies included patients with UC. Active IBD and IBD in remission were included in eight studies and seven studies included patients only in remission.

Fat intake was significantly lower in CD patients ( $n = 154$ ) compared to healthy controls ( $n = 1560$ ) in three of 13 studies<sup>19,31,34</sup> and in three of six studies in UC patients ( $n = 109$ ) compared to healthy controls ( $n = 1512$ ).<sup>28,31,34</sup>

Fat intake was significantly higher in CD patients ( $n = 121$ ) compared to healthy controls ( $n = 486$ ) in two of 13 studies<sup>32,70</sup> and in one of six studies in UC patients ( $n = 66$ ) compared to healthy controls ( $n = 100$ ).<sup>32</sup>

Fat intake was similar between CD patients ( $n = 607$ ) and healthy controls ( $n = 515$ ) in nine of 14 studies<sup>12,18,21–24,69,71,72</sup> and three of seven studies in UC patients ( $n = 175$ ) compared to healthy controls ( $n = 493$ ).<sup>24,70,72</sup>

## Carbohydrate

There were 15 studies that assessed carbohydrate intake<sup>12,18,19,21–24,28,31,32,34,69–72</sup> with 849 patients with CD, 400 patients with UC and 2525 healthy controls. Fourteen studies included patients with CD and seven studies included patients with UC. Active IBD and IBD in remission were included in eight studies and seven studies included patients only in remission.

There were no differences in carbohydrate intake between CD ( $n = 483$ ) and healthy controls ( $n = 806$ ) in nine of 14 studies<sup>12,18,19,21,22,32,69,70,72</sup> and UC ( $n = 227$ ) and healthy controls ( $n = 207$ ) in three of seven studies.<sup>24,32,72</sup> Compared with healthy controls ( $n = 1643$ ), higher carbohydrate intake was found in five of 13 studies for CD patients ( $n = 261$ )<sup>23,24,31,34,71</sup> and four of six studies for UC patients ( $n = 155$ ) compared to healthy controls ( $n = 1898$ ).<sup>28,31,34,70</sup>

## Fibre

**STATEMENT:** Fibre intake is likely to be low in inflammatory bowel disease and should be included as part of a nutritional assessment. (GRADE very low quality) agreement 88.1%.

Seven studies assessed fibre intake, three had a low risk of bias<sup>22,24,32</sup> and four had unclear risk of bias<sup>31,70–72</sup> in 471 CD patients, 226 UC patients and 2142 healthy controls.

Fibre intake was significantly lower in patients with CD ( $n = 439$ ) compared to healthy controls ( $n = 2142$ ) in six of seven studies<sup>22,31,32,70–72</sup> and patients with UC ( $n = 341$ ) compared to healthy controls ( $n = 2062$ ) in four of five studies.<sup>31,32,70,72</sup>

## Calcium

**STATEMENT:** Calcium intake should be assessed in Crohn's disease and ulcerative colitis because patients may not meet their recommended intake. (GRADE very low quality) agreement 96.3%.

Ten studies assessed calcium intake, three had a low risk of bias,<sup>22,24,53</sup> four had an unclear risk of bias<sup>71–74</sup> and three had high risk of bias<sup>25,28,75</sup> in 906 IBD patients (CD,  $n = 425$ ; UC,  $n = 329$ ; unknown,  $n = 152$ ) and 837 healthy controls.

Four studies included patients in remission<sup>22,53,71,73</sup> and six studies included patients with active disease or disease in remission.<sup>24,25,28,72,74,75</sup>

Six of nine studies found no significant difference in calcium intake between CD ( $n = 381$ ) and healthy controls ( $n = 708$ )<sup>22,24,25,72–74</sup> and four of six studies between UC ( $n = 278$ ) and healthy controls ( $n = 557$ ).<sup>25,28,72,74</sup> Two studies found lower intakes compared to healthy controls, one in UC ( $n = 46$ ) compared to healthy controls ( $n = 23$ )<sup>24</sup> and one in mixed IBD ( $n = 152$ ) compared to healthy controls ( $n = 73$ ).<sup>75</sup>

## Iron

**STATEMENT:** Iron intake should be assessed in Crohn's disease and ulcerative colitis as patients may not meet their recommended intake. (GRADE very low quality) agreement 96.2%.

Eight studies assessed dietary iron intake with three showing low risk of bias,<sup>22,24,69</sup> three showing unclear risk of bias<sup>21,72,73</sup> and two showing high risk of bias<sup>25,28</sup> in 348 patients with CD, 233 patients with UC and 336 healthy controls.

Three out of seven studies showed that iron intake was lower in CD ( $n = 215$ ) compared to healthy controls ( $n = 216$ )<sup>25,69,72</sup> and two of five studies showed that iron intake was lower in UC ( $n = 170$ ) compared to healthy controls ( $n = 125$ ).<sup>25,72</sup> All other studies (4/7) showed no differences in iron intake.<sup>21,22,24,28,73</sup> One study reported

that only 32% of patients with CD met the recommended intake for iron.<sup>69</sup>

## NUTRITIONAL SCREENING

**PRACTICE STATEMENT:** Screening for nutritional risk may be considered an integral part of inflammatory bowel disease (IBD) care in the IBD multidisciplinary care setting. Agreement 98.2%.

BMI is not an accurate marker of malnutrition in IBD patients and reduced lean body mass may be missed in patients with a normal or high BMI. In addition, dietary exclusion to manage symptoms is common in IBD and avoidance of specific foods and/or food groups increases the risk of micronutrient deficiencies. Thus, nutrition screening tools that rely heavily on BMI may not be capturing some patients at nutritional risk.

**STATEMENT:** MUST may be used to screen patients with inflammatory bowel disease for risk of malnutrition GRADE very low quality. Agreement 85.5%.

Ten studies assessed malnutrition risk using the Malnutrition Universal Screening Tool (MUST), four were unclear risk of bias<sup>76–79</sup> and six were high risk of bias.<sup>80–85</sup> These studies included 80 inpatients (CD,  $n = 62$ ; UC,  $n = 18$ )<sup>76,82–84</sup> and 936 outpatients (CD,  $n = 582$ ; UC,  $n = 347$ ; unclassified IBD,  $n = 7$ ).<sup>77,80,81,84</sup>

Combining the results from all ten studies, MUST scores for all IBD patients were 751/1080 (70%) for low risk, 128/1080 (12%) for moderate risk and 198/1080 (18%) for high risk. For outpatients, MUST scores were 734/998 (74%) for low risk, 112/998 (11%) for moderate risk and 151/998 (15%) for high risk. For inpatients, MUST scores were 17/80 (21%) for low risk, 16/80 (20%) for moderate risk and 47/80 (59%) for high risk. The MUST tool assesses risk of malnutrition and the other tools assess nutrition risk.<sup>78,79,86</sup>

The Saskatchewan IBD nutrition risk tool (SaskIBD-NR) uses a score based on symptoms, weight loss and food restriction. In 110 IBD outpatients (CD,  $n = 75$ ; UC,  $n = 35$ ), it showed significant agreement with a registered dietitian or gastroenterologist assessment ( $k = 0.83$ ,  $p < 0.001$ ), whereas MUST showed a lack of agreement ( $k = 0.15$ ,  $p = 0.12$ ).<sup>79</sup> The SaskIBD-NR had better sensitivity (82.6% vs. 26.1%), specificity (97.7% vs. 87.4%), positive predictive value (90.5% vs. 35.3%) and negative predictive value (95.5% vs. 81.7%) than MUST. In patients with a BMI  $> 25.0 \text{ kg m}^{-2}$ , the SaskIBD-NR had better sensitivity (63.6% vs. 27.3%) but not specificity

(73.2% vs. 90.2%) than MUST to identify patients malnourished using Subjective Global Assessment (SGA).<sup>87</sup> In patients waiting for surgery,<sup>85</sup> SaskIBD-NR and MUST performed similarly and did not identify all patients malnourished according to the Global Leadership Initiative on Malnutrition (GLIM) criteria but did identify a similar percentage of patients malnourished according to ESPEN criteria.

Malnutrition inflammatory risk tool (MIRT) includes BMI, unintentional weight loss and CRP. In a study of 55 patients with CD, seven (12.7%) were mild to severely malnourished (SGA B or C) and nine (16.4%) had a MIRT score  $\geq 3$  which was associated with worse clinical outcomes at 6 months.<sup>86</sup> The number of malnourished patients according to SGA reduced from seven to three over the 6-month period and it is not reported whether MIRT identified all malnourished patients.<sup>86</sup> In a surgical IBD cohort, MIRT successfully identified malnourished patients using GLIM criteria.<sup>85</sup>

The Inflammatory Bowel Disease-Nutrition Screening tool (IBD-NST) uses a score based on BMI, weight loss, active disease and nutrition concerns.<sup>78</sup> In 101 IBD outpatients (CD,  $n = 61$ ; UC,  $n = 33$ ; IBDU,  $n = 7$ ), 12 patients who were low risk for MUST were high risk for IBD-NST as a result of having a flare of IBD symptoms and concerns about their nutrition. IBD-NST was compared with SGA, MUST, hand grip strength, mid-arm muscle circumference and BMI. Unlike SGA and MUST, IBD-NST nutrition risk was not predicted by BMI (area under the curve = 0.262 [SE = 0.06]; 95% confidence interval [CI] = 0.17–0.40).

There are now multiple screening tools available. The best screening tool for clinical practice will depend on the purpose of nutrition screening and the capacity of the health service to deliver appropriate nutrition interventions.<sup>86</sup>

## Self-screening

Four recent studies from Canada<sup>81,87</sup> and the UK<sup>77,78</sup> have found that patients can accurately self-screen for risk of malnutrition using MUST<sup>77,78,81,87</sup> IBD-NST,<sup>78</sup> SaskIBD-NR<sup>87</sup> or abridged patient-generated SGA.<sup>87</sup> The use of self-screening may be a more cost-effective method of identifying patients who are likely to benefit from further nutritional assessment and dietetic input.

## ORAL NUTRITIONAL SUPPORT

**PRACTICE STATEMENT:** Oral nutritional supplementation may improve nutritional status in patients with inflammatory bowel disease. Agreement 88.9%.

Four studies met the inclusion criteria.<sup>76,88–90</sup> Three studies only included 91 CD patients,<sup>76,88,89</sup> and one study included 13 CD and 25 UC patients.<sup>90</sup>

All studies used different oral nutritional supplementation products for different durations.

One study was a crossover study in outpatients with active CD or CD in remission. Patients received normal diet for 2 months or normal diet supplemented with oral nutritional supplement (ONS) (Ensure Plus; Abbott) for 2 months or vice versa. Both groups had an increase in energy intakes, weight and mid-arm muscle circumference ( $p < 0.05$ ). Comparisons between groups were not presented.<sup>88</sup>

One study allocated patients with CD in remission to oral nutritional supplements (Elemental 028; Scientific Hospital Supplies) ( $n = 21$ ) or a normal diet ( $n = 18$ ) for 12 months.<sup>89</sup> Weight and BMI increased and more patients remained in remission at 12 months in the ONS group compared to the normal diet group.

One study in 25 inpatients with active CD gave ONS (Modulen IBD; Nestle) for 3 months to eight patients and dietary counselling and normal hospital diet to 17 patients. In the ONS group, weight, BMI, MUST, SGA and handgrip strength all improved after 3 months, whereas, in the dietary counselling and normal hospital diet group, only handgrip strength improved.<sup>76</sup>

One study in active IBD (UC,  $n = 25$  UC; CD,  $n = 13$ ) randomised patients to receive ONS (Novasource; Nestle) in addition to hospital diet or hospital diet for 3 weeks. Patients who refused ONS were transferred to the hospital diet group. SGA, protein and energy intake and disease activity scores improved in both groups. No comparisons between groups were presented.<sup>90</sup>

Despite limited evidence in IBD, that ONS improves nutritional status, dietary advice with or without ONS may improve nutritional status in adults with malnutrition.<sup>91</sup>

## INDUCTION OF REMISSION

### Enteral nutrition

**STATEMENT:** To induce remission in active Crohn's disease, exclusive enteral nutrition (EEN) is less effective than corticosteroids. EEN may be used in mild to moderate disease where avoidance of corticosteroids is intended, and dietetic expertise is available. (GRADE very low quality) agreement 84.6%.

A recent systematic literature review of exclusive enteral nutrition (EEN) to induce disease remission in adults with Crohn's disease reviewed literature published from inception to July 2017.<sup>92</sup> No further studies in adults

were identified after this date. Six RCT published from 1985 to 2002 were included in an intention to treat and per protocol meta-analysis of EEN versus corticosteroids to induce remission.<sup>93–98</sup> The intention to treat meta-analysis found 87 of 194 (45%) patients on EEN and 116 of 158 (73%) patients on corticosteroids achieved disease remission with risk ratio of 0.65 (95% CI = 0.52–0.82) favouring corticosteroids. The per protocol meta-analysis induction of remission results also favoured corticosteroids with 87 of 149 (58%) patients on EEN and 116 of 158 (73%) on corticosteroids with a risk ratio of 0.82 (95% CI = 0.70–0.95). These RCTs were reported between 1985 and 2002. The palatability of and methods used to administer current enteral nutrition formula have significantly improved since then. In addition, evidence from recent paediatric RCTs suggest that EEN is equivalent to corticosteroids at inducing disease remission.<sup>92,99,100</sup> Recent guidelines suggest that EEN may be considered as an alternative to corticosteroids in patients with mildly active CD.<sup>47,101</sup>

**PRACTICE STATEMENT:** In steroid-refractory or steroid-intolerant disease, exclusive enteral nutrition may be used as an adjuvant to induce disease remission in patients with active Crohn's disease starting immunosuppressive and/or biologic therapy. Agreement 84.3%.

Two case series reported improvement in CRP and disease activity in patients with active Crohn's disease treated with exclusive enteral nutrition as an adjuvant to aminosaliclates, immunosuppressive and/or biologic therapy.<sup>102,103</sup> One case series in 16 adults with active Crohn's disease treated with exclusive enteral nutrition as an adjuvant to existing Crohn's disease medications (details not provided)<sup>102</sup> showed significant reductions in Harvey Bradshaw index ( $6.5 \pm 5.8$  to  $2.4 \pm 3.3$ ) ( $p = 0.001$ ) and CRP ( $3.5\text{--}0.88$  mg dL<sup>-1</sup>) ( $p = 0.023$ ). Another case series in 25 patients with active Crohn's disease treated with EEN included 13 patients on stable doses of mesalazine ( $n = 9$ ), immunosuppressants ( $n = 3$ ) or biologic ( $n = 1$ ) medications. Only 14 of 25 (56%) patients completed EEN and achieved disease remission (Harvey Bradshaw Index < 5).<sup>103</sup> The outcomes of patients on concurrent medications were not reported separately. The two case series suggest that remission may be induced in patients with active Crohn's disease treated with EEN in addition to concurrent medications but this needs to be confirmed in controlled studies. The use of exclusive enteral nutrition to induce disease remission in patients with active disease on an optimised regimen of immunosuppressant and/or biologic medication has not been described in the literature.

A recent open label RCT of 7 days of EEN in acute severe UC has investigated whether EEN can improve

the response to intravenous corticosteroids.<sup>104</sup> On an intention-to-treat analysis, corticosteroid treatment failed in eight of 32 (25%) patients who received EEN versus 13 of 30 (43%) who ate a normal diet ( $p = 0.051$ ). Larger high-quality studies are warranted to confirm whether EEN can augment the effectiveness of corticosteroids in acute severe UC.

**PRACTICE STATEMENT:** Exclusive enteral nutrition, either used as a primary or adjuvant therapy, may be used for a minimum of 6 weeks to induce disease remission and achieve mucosal healing in patients with mild to moderate Crohn's disease in which corticosteroids are contradicted or the patient chooses to avoid corticosteroids. Agreement 84.3%.

Access to dietetic expertise and use of a defined pathway are essential for successful use of exclusive enteral nutrition.<sup>47,105</sup> Practical guidance is provided in Table 6.

## Whole food or elimination diets

**PRACTICE STATEMENT:** There is insufficient evidence to support the use of a whole food diet to induce disease remission in adults with active inflammatory bowel disease. Agreement 87.5%

Practical dietary advice for IBD is provided in Table 7. Ten whole food dietary exclusion interventions to induce remission in adults with active IBD were reviewed. These are anti-inflammatory IBD diet, low FODMAP (i.e., fermentable oligosaccharides, disaccharides, monosaccharides and polyols) diet, Crohn's disease exclusion diet (CDED), IgG4, Western diet, low microparticle diet, semi-vegetarian plant-based diet, Mediterranean diet, specific carbohydrate diet (SCD) and Crohn's disease Treatment with EATING diet (CD-TREAT).<sup>109–112</sup> The results of a feasibility study of a low emulsifier diet in adults with active IBD are also promising.<sup>113</sup> However, high quality evidence is needed for most of these whole food interventions before they may be recommended as alternative treatments to current management strategies recommended as primary treatment for active disease.

A 2019 Cochrane review concluded that there is insufficient evidence to determine whether exclusion diets induce disease remission in Crohn's disease or ulcerative colitis.<sup>114</sup> Some of their reviewed studies did not meet our inclusion criteria.<sup>115–117</sup> Subsequent to this review, new



**TABLE 6** Practical considerations for using exclusive enteral nutrition (EEN) in Crohn's disease

- For successful EEN, ensure the whole multi-disciplinary team support its use
- Some patients may choose EEN rather than pharmaceutical management
- Corticosteroids may be contra-indicated in some patients and EEN is a suitable alternative to induce disease remission
- EEN can be used as an adjunct to other pharmaceutical management options
- Always provide patients with detailed information on how to implement EEN in a structured manner
- Calculate nutritional requirements based on resting energy expenditure ( $25\text{--}30\text{ kcal kg}^{-1}\text{ day}^{-1}$ )<sup>106</sup> and protein ( $1\text{ g kg day}^{-1}$ )<sup>107</sup>
- Be prepared to modify nutritional requirements depending on physical activity level especially if patients report being hungry once EEN is established
- Provide different flavours and product samples of EEN, some will have different flavour options
- Use a starter regimen to commence EEN and where applicable gradually increase EEN over 3 days as food is reduced and stopped
- EEN can often be commenced in the out-patient setting and most patients can continue with their usual day-to-day activities
- Where hospital admission is necessary, consider refeeding risk and monitor urea and electrolytes, phosphate and magnesium daily until energy requirements are established
- Ensure EEN prescription is approved by the primary care team at the recommended dose and duration
- Once the target regimen is met, EEN can induce clinical remission within 10 days, however mucosal healing takes 6 weeks. Discuss the appropriate duration with the patient and multi-disciplinary team
- The majority of patients can tolerate EEN orally, however if patients are struggling to meet target volumes consider nasogastric feeding
- Regular support from a dietitian is vital to achieve and maintain patient motivation with EEN for the whole duration
- Ensure dietetic support is available to troubleshoot issues with tolerance, adherence, dental care, stool colour and symptoms. Consider adjusting EEN volume and the concentration of a powder feed
- Once EEN is established, consider issues around motivation to continue for the whole EEN duration and discuss other food intake, social interaction, peer pressure and hunger
- EEN protocols will normally allow some other fluids (e.g., water, weak black tea and coffee or diluted squash)
- Provide information on how to reintroduce food after a period of EEN and where appropriate gradually reducing the volume of enteral nutrition as more food is introduced

evidence from RCTs in adults with IBD has been published. Below is a summary of the evidence for each whole food dietary exclusion intervention.

## CDED

The CDED is phased dietary approach comprised of an initial 6 weeks of partial enteral nutrition (50% of energy requirements) and an exclusion diet (50% of energy requirements) followed by a second 6 weeks

**TABLE 7** Practical dietary advice for inflammatory bowel disease (IBD)

- Discourage self-directed non-evidence-based exclusion diets as they can promote nutritional deficiency and low diet quality
- Many dietary components have been associated with IBD, however, there is no evidence to support restriction of any one dietary component

### For stable inflammatory bowel disease

- Include a wide range of fruit, vegetables, nuts and seeds and wholegrains (with exception of stricturing disease) to meet energy and nutritional requirements. There is no need to restrict fibre intake, except in stricturing disease (see below)
- Include a variety of protein-rich foods, with a reduction in animal fat and processed meat. Limit red and processed meat to no more than twice weekly (approximately  $150\text{ g week}^{-1}$ ) to reduce risk of colon cancer<sup>108</sup>
- Limit intake of high fat, high sugar and high salt foods – guidance on label reading may be useful
- Consider vitamin D supplementation throughout the year especially if vitamin D deficient

### Stricturing inflammatory bowel disease

- Stricturing disease may need an individualised approach with (i) avoidance of fibrous foods (i.e. tough outer skins and stalks of fruit and vegetables, tough/grisly meat) and (ii) inclusion of foods rich in soluble fibres accompanied by fluids consumed at the same time

### Steroids

- Steroids reduce calcium absorption and can lead to bone resorption, supplement patients on steroids with vitamin D and calcium, do not continue long term unless calcium intake is below  $800\text{ mg day}^{-1}$ . Calcium without vitamin D may increase cardiovascular risk.<sup>47</sup>

of partial enteral nutrition (25% of energy requirements) with a slightly less restricted diet.<sup>118</sup> It was first described in 2014 with a case series of 13 adults (six males, seven females) aged greater than 18 years. Subsequently, a RCT in paediatric Crohn's disease compared CDED with EEN and found that the CDED was as effective as EEN to induce disease remission<sup>111</sup> and a pilot RCT in adults with mild-to-moderate Crohn's disease was published in 2022.<sup>112</sup> The RCT in adults compared the CDED without partial enteral nutrition to the CDED with partial enteral nutrition. The pilot study found that, after 6 weeks of treatment, 57% of the CDED alone versus 68% ( $p = 0.462$ ) of the CDED with partial enteral nutrition group were in clinical remission.<sup>112</sup>

## CD-TREAT

The CD-TREAT diet is a personalised diet designed to replicate the gut microbiome changes that have been observed in patients treated with EEN.<sup>109</sup> It contains a moderate amount of complex carbohydrate, higher amounts of protein, is low in fibre and includes an oral

multivitamin tablet. A non-randomised study in five children showed that, after 8 weeks of the dietary treatment, disease activity and faecal calprotectin reduced significantly.<sup>109</sup> An open-label trial in 32 adults with mild-to-moderately active Crohn's disease has been conducted. The trial showed that 23 of 32 adults completed the 8-week treatment and patients who strictly adhered to the treatment had a significant reduction in faecal calprotectin and 80% achieved clinical disease remission.<sup>110</sup>

### Low FODMAP diet<sup>119</sup>

The low FODMAP diet is a phased dietary approach that initially restricts the intake of short-chain fermentable carbohydrates. One RCT compared the low FODMAP diet to a normal diet in patients with mild-to-moderately active IBD ( $n = 13$ ) or IBD in remission ( $n = 65$ ) with co-existing functional bowel symptoms.<sup>120</sup> The low FODMAP diet had no significant effect on disease activity compared with the normal diet group for simple clinical colitis activity index or Harvey Bradshaw index. Research supports a low FODMAP diet to manage functional bowel symptoms rather than induce disease remission for active IBD.<sup>120,121</sup>

### Anti-inflammatory diet

The anti-inflammatory IBD diet is a phased dietary approach that includes modification of dietary carbohydrates, increased foods with pre- or probiotic properties, intake of anti-inflammatory fats and modification of eating pattern or dietary texture. The use of the anti-inflammatory IBD diet, in addition to usual IBD medications, has been reported in an uncontrolled, retrospective case series of 40 IBD patients.<sup>119</sup> The case series reported that 4 weeks of the strict dietary regimen improved disease activity in 10 of 11 patients who had a complete data set and 24 of 40 (60%) self-reported a good or very good response to the diet.

### SCD and Mediterranean diet

The SCD is a phased dietary treatment that includes carbohydrate rich foods that contain only monosaccharides, excludes foods rich in disaccharides (e.g., sucrose and lactose) and polysaccharides (e.g., grains, starchy vegetables and specific legumes). The diet includes unprocessed protein-rich foods and excludes most processed foods. Use of the SCD as a treatment for active IBD has been described in various paediatric case series. There has been one randomised superiority trial comparing 12 weeks of the SCD to a

Mediterranean diet in adults with mild-to-moderately active Crohn's disease ( $n = 194$ ).<sup>122</sup> Participants received all their meals and snacks for the first 6 weeks. At baseline, faecal calprotectin  $>250 \mu\text{g g}^{-1}$  and high-sensitivity CRP  $> 5 \text{ mg L}^{-1}$  were reported in 38% and 67% of participants, respectively. At week 6, less than half the participants (46.5% on SCD and 43.5% on Mediterranean diet) were in remission using the short Crohn's disease activity index, less than 35% of participants had a faecal calprotectin  $<250 \mu\text{g g}^{-1}$  and less than 5% had high-sensitivity CRP  $< 5 \text{ mg L}^{-1}$ . The SCD was not found to be superior to the Mediterranean diet and neither diet resulted in normalised serum CRP concentration.<sup>122</sup>

### IgG4-guided exclusion diet<sup>111,118</sup>

The IgG4-guided exclusion diet is based on the premise that improvement in functional gut symptoms have been shown after the individualised exclusion of foods based on IgG4 reactivity. A multi-centre, double-blind, randomised, controlled trial of the IgG4-guided exclusion diet was conducted in 98 adults with active Crohn's disease.<sup>123</sup> Patients either received dietary advice on the guided exclusion diet or a sham diet for 4 weeks, and 70 patients were taking immunomodulatory medication. In the intention to treat analysis, significant improvements in Crohn's disease activity index (CDAI) were observed in patients taking the exclusion diet (mean change 55.7; interquartile range [IQR] = -113 to 216) compared with patients taking the sham diet (mean change 16.8; IQR = -196 to 196). However the results do not report how many patients achieved disease remission (CDAI  $<150$ ). Faecal calprotectin and CRP were not included in the intention to treat analysis and the per protocol analysis in 76 patients who completed the study showed there were no significant differences in change of faecal calprotectin (0) ( $p = 0.19$ ) or CRP (47) ( $p = 0.13$ ) between groups. The use of IgG4 exclusion diet in patients with active Crohn's disease reduced disease activity symptoms but did not result in significantly greater improvements in markers of inflammation than a sham diet.

### Organic pre-Western diet

The Organic pre-Western diet hypothesis is that dietary intake in Western industrialised countries has changed substantially since the start of the 20th century and that exclusion of foods and practices associated with industrialisation will promote intestinal healing in mild to moderately active Crohn's disease. This theory was tested in a RCT of 18 patients with active Crohn's disease (CDAI = 150–220) and

ulceration of the small or large bowel visible on magnetic resonance imaging (MRI). Concomitant use of a stable dose of medical therapy was permitted.<sup>124</sup> The dietary intervention involved following an organically produced diet of red meat, spelt sourdough, and small amounts of fresh butter, rape oil, tea, tap water and rock salt. Fruit and vegetables were excluded as a result of the inability to ensure that they were always organically produced. Patients were also instructed to use baking soda toothpaste, avoid using a dishwasher and rinse plates in water after washing by hand. The control group were advised to eat a low-fat, high carbohydrate diet, with avoidance of fibre-rich fruit and vegetables and red meat. Both groups received vitamin B complex and vitamin C intramuscular injections every 3 weeks. Eight patients were randomised to the intervention (three withdrew at baseline after receiving dietary education) and 10 were randomised to the control diet (one withdrew after receiving dietary education). After 6 weeks of dietary treatment, MRI improvements were observed in three of eight (38%) of the intervention group and one of 10 (10%) of the control group and remission, defined as CDAI < 150, was achieved by four of eight (50%) patients in intervention group and seven of 10 (70%) of patients in control group. An RCT in 213 CD patients in remission (CDAI < 150) showed no difference in the rate of disease flare ups between a diet low in red meat with less than one serving of red or processed meat per month compared to a diet with more than two servings of red or processed meat per week.<sup>125</sup> The use of this restrictive diet, which required intramuscular vitamin supplementation to meet nutritional requirements, requires further scientific evidence to determine efficacy.

## Low microparticle diet

A low microparticle diet to induce CD remission was investigated in a RCT.<sup>126</sup> Microparticles are not only present in the environment, but also are increasing in the food supply as food additives. A pilot RCT of a low microparticle diet in patients taking corticosteroids showed that the diet, in conjunction with a low calcium intake, may result in disease remission occurring more quickly compared with a normal diet.<sup>127</sup> A subsequent multicentre RCT was conducted where 83 patients were randomised to one of four groups for 16 weeks: calcium controlled low microparticle diet with a placebo supplement or a 400 mg day<sup>-1</sup> calcium supplement or a calcium controlled normal microparticle sham diet with a placebo supplement or a 400 mg day<sup>-1</sup> calcium supplement. All patients started on 30 mg day<sup>-1</sup> reducing dose of prednisolone and could take aminosalicylate (Pentasa) but no other CD medication. At week 16, there were no statistically significant differences in

disease activity, CRP or faecal calprotectin between the low and normal microparticle diets or the normal and low calcium groups. The adjuvant use of low microparticle diet and/or a low calcium intake, in addition to a reducing dose of prednisolone, did not provide additional benefit over and above a normal microparticle diet.

## Semi-vegetarian plant-based diet

The use of semi-vegetarian plant-based diet in conjunction with anti-tumour necrosis factor  $\alpha$  medication Infliximab to induce remission of active CD was investigated in a multi-centre cohort study.<sup>128</sup> Patients (35 adults [26 biologic naïve] and 11 children) received enteral nutrition for 3–7 days prior to the first infliximab infusion at week 0 and started on a lacto-ovo-semi-vegetarian diet for 6 weeks, which included fish once a week and red meat once a fortnight. Infliximab infusions were also given at weeks 2 and 6. At baseline, seven of 35 (20%) were in remission (CDAI < 150) and, at week 6, remission was observed in 33 of 35 (94%) adults and CRP normalised in 28 of 35 (80%) adults. The major limitation of this study is the lack of a control group who received infliximab induction therapy without dietary intervention. RCT evidence of efficacy of the semi-vegetarian plant-based diet in conjunction with biological therapy is required before a clinical recommendation can be made.

## Fibre

**PRACTICE STATEMENT:** There is insufficient evidence to use prebiotic fibre to treat active ulcerative colitis. Agreement 87.0%.

Two systematic reviews of fibre (supplement or whole diet intervention) as a treatment to induce remission in IBD were performed.<sup>129,130</sup> Five RCTs were identified that included 114 patients with active UC.<sup>131–135</sup> All studies assessed the effects of a prebiotic, two of which were combined with a probiotic compared with a placebo. Study duration varied from 2 weeks to 12 months and there was no homogeneity of outcomes to which GRADE criteria could be applied. Compared with placebo the findings from these studies are summarised below.

Germinated barley fibre (20–30 g day<sup>-1</sup>) led to lower overall disease score at 4 weeks ( $p = 0.045$ ), mainly driven by reduced frequency of diarrhoea.<sup>134</sup>

Fructo-oligosaccharide (12 g day<sup>-1</sup>) plus probiotic led to greater reduction in sigmoidoscopy score at



4 weeks but failed to reach significance ( $p = 0.06$ ) and there was no difference in clinical disease activity score.<sup>131</sup>

Oligofructose inulin ( $12 \text{ g day}^{-1}$ ) alongside mesalazine ( $3 \text{ g day}^{-1}$ ) for 2 weeks had no effect on clinical outcomes compared to placebo alongside mesalazine ( $3 \text{ g day}^{-1}$ ), although faecal calprotectin was lower than baseline in the prebiotic group only ( $p < 0.05$ ).<sup>132</sup>

Galacto-oligosaccharide ( $5.5 \text{ g day}^{-1}$ ) with a probiotic led to similar endoscopic score at 1 year compared with placebo. However, there was a significant reduction in faecal myeloperoxidase for the intervention arm compared to placebo ( $p < 0.05$ ).<sup>133</sup>

Oligosaccharide inulin ( $7 \text{ g day}^{-1}$ ) with a probiotic and micronutrients for 2 months led to lower interleukin (IL)-6 ( $p < 0.05$ ), IL-8 ( $p < 0.01$ ) and lymphocyte expression ( $p < 0.05$ ) but clinical outcomes were not reported.<sup>135</sup>

No further RCTs in UC patients have been identified subsequent to the 2014 systematic review of fibre by Wedlake et al.<sup>129</sup>

**PRACTICE STATEMENT:** There is insufficient evidence to use prebiotic fibre to treat active Crohn's disease. Prebiotic fibre may increase abdominal symptoms. Agreement 96.3%.

Three studies that measured the effect of prebiotic fibre on clinical outcomes in 156 patients with active CD<sup>124,136,137</sup> have been systematically reviewed.<sup>124,129,136,137</sup>

An organic high-fibre diet ( $n = 8$ ; mean fibre content  $46 \text{ g day}^{-1}$ ) was compared with a control low-fibre diet ( $n = 10$ ; mean fibre content  $16 \text{ g day}^{-1}$ ).<sup>124</sup> There was no difference between groups for remission rates and on an intention to treat basis, with three of eight patients achieving mucosal healing in the high fibre group compared to one of 10 in the control group.

Oligofructose inulin ( $12 \text{ g day}^{-1}$ ) alongside a probiotic showed no difference in remission rates at 6 months in patients with active CD ( $n = 19$ ) compared to placebo ( $n = 16$ ); however, the treatment group had a significant improvement in histological scores from baseline but the control did not. There were no changes in either group in bowel habit, biochemical markers or IBD questionnaire scores.<sup>137</sup>

Oligofructose inulin ( $15 \text{ g day}^{-1}$ ,  $n = 54$ ) compared to placebo ( $n = 49$ ) for 4 weeks in active CD patients demonstrated no improvement in clinical outcomes and worse flatulence and abdominal pain and rumbling after prebiotic, although lower numbers of IL-6 and higher numbers of IL-10 expressing dendritic cells in the prebiotic group were reported.<sup>136</sup>

No further randomised controlled trial were identified after the publication of the systematic review by Wedlake et al.<sup>129</sup>

## Vitamin D

Low serum vitamin D is common in IBD. Adjuvant high dose vitamin D supplementation improves serum vitamin D concentrations but there is conflicting evidence on the effect of vitamin D supplementation on disease activity.<sup>138,139</sup>

Public Health England recommend that all adults should consider taking a daily vitamin D supplement of  $10 \mu\text{g}$  from October to March to improve their vitamin D status.

## Fish oil

**PRACTICE STATEMENT:** There is insufficient evidence that a fish oil supplement reduces disease activity in active inflammatory bowel disease. Agreement 88.9%.

An RCT in patients with mild to moderately active UC ( $n = 121$ ) found that partial enteral nutrition (33% of energy) supplemented with fish oil ( $2.5 \text{ g day}^{-1}$  eicosapentaenoic acid and  $1 \text{ g day}^{-1}$  docosahexaenoic acid) and fructo-oligosaccharide ( $6.5 \text{ g day}^{-1}$ ) had no effect on clinical outcomes or histology after 6 months compared to a carbohydrate-based placebo.<sup>140</sup> At baseline, 50% of patients were taking prednisone and/or sulfasalazine and medication doses could be adjusted based on patient symptoms. The effect of the fish oil could not be extrapolated from the mixed enteral nutrition and drug therapy; however, there were no differences between the intervention and placebo in reduction in disease activity.

An open label study in mild to moderately active CD ( $n = 20$ ) using partial enteral nutrition (33% of energy) supplemented with fish oil ( $2.5 \text{ g day}^{-1}$  eicosapentaenoic acid and  $1 \text{ g day}^{-1}$  docosahexaenoic acid) and fructo-oligosaccharide ( $6.5 \text{ g day}^{-1}$ ) was conducted.<sup>141</sup> At baseline, 50% of patients were taking corticosteroids. A number of adverse effects were attributed to the intervention in active CD including nausea, excessive flatulence, faecal incontinence, hot flushes and abdominal pain, although it would be difficult to determine the ingredient that induced the effects. Half of the patients achieved 85% compliance with the nutrition intervention associated with a  $>2\%$  increase in plasma phospholipid eicosapentaenoic acid ( $n = 10$ ). This group showed a significant improvement in CDAI change ( $-47.8$  [ $-65$  to  $-37$ ]) ( $p = 0.049$ ), whereas patients with 54% compliance ( $n = 10$ ) had no change in CDAI ( $-8.1$  [ $-54.6$  to  $40.1$ ]) ( $p = 0.99$ ). At 4 months, CDAI was significantly less for the 85% compliance patients ( $116 \pm 95$ ) compared with the 54% compliance patients ( $262 \pm 87$ ) ( $p < 0.005$ ). The number of patients who achieved disease remission was not reported. These results need to be interpreted with caution as a result of the study being open label with small numbers, the use of corticosteroids and the

inability to extrapolate the effect of the fish oil from the enteral nutrition.

## Complementary and alternative medicine

**PRACTICE STATEMENT:** There is insufficient evidence to recommend the routine use of complementary or alternative medicine in inflammatory bowel disease. Agreement 81.5%.

A systematic review of complementary and alternative medicines to improve disease activity and markers of inflammation in IBD was published in 2015.<sup>142</sup> The systematic review concluded that there was a lack of homogeneity in the literature and that complementary and alternatives therapies may be effective, although further research is required to confirm their efficacy. Subsequent to this systematic review, nine further RCTs to improve disease activity in active IBD have been published: seven using a curcumin supplement<sup>143–149</sup> and two using cannabidiol-rich botanical extract.<sup>150,151</sup> The evidence and recommendation for curcumin supplementation in active UC is described below. Two RCTs showed cannabidiol-rich botanical extract versus placebo improved disease activity<sup>150,151</sup> but not endoscopic healing.<sup>151</sup> Further details are available in recent reviews and guidelines.<sup>47,152</sup>

## Curcumin

**PRACTICE STATEMENT:** In patients with active ulcerative colitis, curcumin supplementation in addition to mesalamine may improve clinical response, however optimal dose, formulation and duration are unknown. Agreement 81.1%.

Six RCTs in 352 patients with mild to moderately active UC were identified. Five studies reported a benefit of curcumin supplementation compared to placebo on (i) disease activity (reported in five studies)<sup>144–148</sup> and (ii) endoscopic score (reported in three studies).<sup>146–148,153</sup> One study did not show benefit.<sup>143</sup> Curcumin was provided as an adjuvant therapy to mesalamine. Interventions were between 1 and 3 months and studies used different formulations (capsules, enema) and dose (50 mg to 3 g) of curcumin. Clinical response (fall of greater or equal to 3 points of the simple clinical colitis activity index) was reported in five studies and was observed in 85 of 175 (49%) of patients taking curcumin compared with 52 of 177 (29%) of patients on placebo. Endoscopic remission (Partial Mayo score of less than or equal to 1)

was observed in 36 of 83 (43%) patients taking curcumin compared to 10 of 81 (12%) on placebo.

In one small RCT, 30 patients (2:1 ratio) with mild to moderately active CD were randomised to curcumin or placebo for 12 weeks; eight of 20 patients in the curcumin group achieved remission compared with zero of 10 patients in the placebo group.<sup>149</sup>

## Probiotics

Eight systematic reviews of probiotics in active IBD were reviewed for RCTs that met the inclusion criteria.<sup>154–161</sup> One further study also met the inclusion criteria.<sup>162</sup>

**STATEMENT:** In some patients with mildly active ulcerative colitis, taking specific probiotics alongside usual medication may support induction of disease remission. (GRADE moderate quality) agreement 88.7%.

Ten RCTs assessed the effect of probiotics in active UC alongside usual medication, mostly 5-aminosalicylic acid. One study had low risk of bias,<sup>163</sup> five studies had unclear risk of bias<sup>162,164–167</sup> and four studies had high risk of bias.<sup>168–171</sup> The analysis included 356 patients randomised to a probiotic and 311 randomised to placebo. Different probiotics were used across studies. Seven studies showed more patients achieved remission with probiotics (124/272 [46%]) compared to placebo (75/277 [27%]). The probiotics were: De Simone Formulation in three studies<sup>166,167,169</sup>; Yakult *Bifidobacterium breve* strain Yakult, *Bifidobacterium bifidum* strain Yakult and a *Lactobacillus acidophilus* strain in one study<sup>164</sup>; *Bifidobacterium longum* 536 (BB-536) in one study<sup>170</sup>; Profermin *Lactobacillus plantarum* 299v in one study<sup>168</sup>; and *Lactobacillus casei* Zhang, *Lactobacillus plantarum* P-8 and *Bifidobacterium animalis* subsp. *lactis* V9 in one study.<sup>162</sup> Three studies showed similar remission rates between probiotics (53/112 [47%]) and placebo (40/62 [65%]) with these probiotics: *Escherichia coli* nissle in two studies,<sup>163,165</sup> and *Lactobacillus casei* strain ATCC PTA-3945 in one study.<sup>171</sup>

In practice, if patients want to try a probiotic, they should be given the evidence highlighting which ones have been shown to be beneficial in ulcerative colitis (for practical guidance, see Table 8).

**PRACTICE STATEMENT:** There is no evidence for probiotics to induce disease remission in Crohn's disease. Agreement 81.1%.

Three studies have assessed probiotics versus placebo to induce remission in 54 patients with CD.<sup>178–180</sup> One

TABLE 8 Practical guidance for probiotics

Disease and activity	Patients given probiotic (n)	Probiotic	References
Ulcerative colitis			
Induce remission	77	De Simone Formulation (vivomix)	Ng et al., <sup>166</sup> Tursi et al., <sup>167</sup> Sood et al. <sup>169</sup>
	14		
	65		
	10	Yakult <i>Bifidobacterium breve</i> strain Yakult, <i>Bifidobacterium bifidum</i> strain Yakult and a <i>Lactobacillus acidophilus</i>	Kato et al. <sup>164</sup>
	28	<i>Bifidobacterium longum</i> 536 (BB-536)	Tamaki et al. <sup>170</sup>
	32	Profermin <i>Lactobacillus plantarum</i> 299v	Krag et al. <sup>168</sup>
	12	<i>Lactobacillus casei</i> Zhang, <i>Lactobacillus plantarum</i> P-8 and <i>Bifidobacterium animalis</i> subsp. <i>lactis</i> V9	Chen et al. <sup>162</sup>
Remission maintenance	15	Bifid triple viable capsule (BIFICO)	Cui et al. <sup>172</sup>
Pouchitis			
Relapsing	76	De Simone Formulation (vivomix)	Gionchetti et al., <sup>173</sup> Gionchetti et al., <sup>174</sup> Mimura et al. <sup>175</sup> Pronio et al. <sup>176</sup>
Remission	7	<i>Bifidobacterium longum</i> BB-536	Brown et al. <sup>177</sup>
Crohn's disease			
Induce remission	10	<i>Saccharomyces boulardii</i>	Plein and Hotz <sup>178</sup>
Remission maintenance	—	No studies	

study demonstrated improvement in Crohn's disease activity index for the probiotic *Saccharomyces boulardii* ( $n = 10$ )<sup>178</sup> compared to placebo ( $n = 7$ ) and two showed no difference between probiotics *Lactobacillus rhamnosus* GG and *E. coli* Nissle 1917 (6/19 [32%]) compared to placebo (6/18 [33%]).<sup>179,180</sup>

## REMISSION MAINTENANCE

### Whole food or elimination diets

**PRACTICE STATEMENT:** There is a lack of evidence to recommend any specific elimination diet for maintaining remission in patients with inflammatory bowel disease. Agreement 85.2%.

There is much interest in aspects of the modern Western lifestyle that may contribute to intestinal inflammation in IBD. From a dietary perspective, processed foods and the use of preservatives and emulsifiers may be important and studies are underway to evaluate these dietary components.

In a retrospective case-control study, a diet that excluded immunoreactive foods was compared to a control diet to maintain Crohn's disease remission for 3 months following EEN to induce disease remission.<sup>181</sup> Disease relapse was similar between groups ( $p = 0.337$ ) with relapse in four of 32 in the exclusion diet group and eight of 32 in the control diet group after 3 months.

In a prospective pilot RCT, CDED with partial enteral nutrition was compared to CDED without partial enteral nutrition.<sup>112</sup> The CDED is described as a progressive high protein, low animal fat, low haem, low gluten, and low additive diet with exposure to fibre. Remission maintenance was compared at 24 weeks in the intention to treat group ( $n = 40$ ). In the CDED with partial enteral nutrition group, from week 13, patients did not have to include any enteral nutrition and were only allowed to eat foods from CDED, which is the same intervention as CDED without partial enteral nutrition. At 6 weeks, 25 patients were in remission and, at 24 weeks, 20 remained in remission, 12 of 19 patients from the CDED with partial enteral nutrition group and eight of 21 patients from the CDED without partial enteral nutrition group ( $p = 0.113$ ).<sup>112</sup>

Further trials are awaited; however, no recommendations can be made on the use of such diets at this time.<sup>114,125,182</sup>

## Anti-inflammatory diet

**PRACTICE STATEMENT:** There is insufficient evidence to recommend the anti-inflammatory diet to maintain inflammatory bowel disease remission. Agreement 94.1%.

An anti-inflammatory diet comprising foods rich in dietary fibre, prebiotics (fructans and galacto-oligosaccharides), antioxidants (fruits and vegetables), probiotics (fermented foods) and omega-3 polyunsaturated fatty acids (oily fish), as well as low in red meat, sugar and alcohol, was assessed in an RCT of 28 UC patients in remission with a high risk of bias.<sup>183</sup> It showed similar relapse rates and faecal calprotectin at 6 months for the anti-inflammatory diet (5/14) compared to healthy eating advice based on Canada's Food Guide (4/14). The group following Canada's Food Guide had a significant increase in faecal calprotectin over 6 months but the group on the anti-inflammatory diet did not. There is a high chance of a type 2 error in this study and large multi-centre RCTs demonstrating a positive effect on relapse rates and mechanistic understanding are needed before this diet can be recommended.

## Enteral nutrition

**STATEMENT:** Partial enteral nutrition alongside routine medication may support Crohn's disease remission maintenance. (GRADE very low quality) agreement 88.0%.

Three RCTs addressed the efficacy of partial enteral nutrition (oral nutritional supplementation) (420–1200 kcal day<sup>-1</sup>) for maintaining remission in CD.<sup>184–186</sup> One had low risk of bias,<sup>185</sup> one had unclear risk of bias<sup>184</sup> and one had high risk of bias.<sup>186</sup> Combining the data from these three studies was possible for the outcome of remission maintenance. More patients who received partial enteral nutrition 56 of 94 (60%) maintained remission compared to patients who did not (34/88 [39%]) (OR = 2.34, 95% CI = 1.29–4.24).

Low quality observation studies have investigated the use of partial enteral nutrition (300–900 kcal day<sup>-1</sup>) to prevent loss of response to biologic anti-tumour necrosis factor  $\alpha$  therapy.<sup>187–193</sup> However, it is unclear whether there is a beneficial effect compared with no enteral nutrition.

There is no evidence to recommend partial or exclusive enteral nutrition for maintaining remission in patients with UC.

## Complementary and alternative medicine

**PRACTICE STATEMENT:** There is insufficient evidence to recommend the use of complementary or alternative medicinal products to maintain remission in inflammatory bowel disease. Agreement 84.6%.

There is a lack of evidence to recommend complementary and alternative medicines to maintain disease remission in IBD.<sup>142,152</sup> One RCT investigated 2 g day<sup>-1</sup> curcumin compared to placebo for 6 months in patients with quiescent UC and reported significantly fewer patients relapsed following supplementation with curcumin (2/43 [5%]) compared to placebo (8/39 [21%]) ( $p = 0.04$ ). Curcumin was given in addition to standard medication (sulfasalazine or mesalamine).<sup>194</sup> In another RCT in 62 post-operative patients with Crohn's disease receiving azathioprine, curcumin was no more effective than placebo in preventing endoscopic or clinical recurrence in Crohn's disease at 6 months.<sup>195</sup>

## Fibre

**PRACTICE STATEMENT:** There is insufficient evidence for the use of a high fibre diet or fibre supplementation for maintaining remission in inflammatory bowel disease. Agreement 82.4%.

The recommended intake of fibre is 30 g day<sup>-1</sup>; however, most patients with IBD do not meet this level.<sup>22,72</sup> Patients perceive that fibre exacerbates symptoms<sup>196</sup> and therefore often avoid it during a disease flare. However, in stable disease without strictures, there is no research evidence to support restriction of fibre.<sup>197</sup>

In patients with UC in remission, psyllium appears safe to use and could be used alongside standard medication should patients wish to try it.<sup>198,199</sup>

Two systematic reviews of fibre (supplement or whole diet intervention) as a treatment to maintain remission in IBD were performed<sup>129,130</sup> and, despite identifying five studies including 232 patients with quiescent UC<sup>198–202</sup> and four studies including 465 patients with quiescent CD,<sup>203–206</sup> there was high heterogeneity among studies with varied criteria for assessing outcome and neither meta-analysis, nor GRADE summary of the evidence was possible.<sup>198–201,203–206</sup> The largest trial of 105 patients compared remission maintenance over 12 months for 1.5 g day<sup>-1</sup> mesalamine (24/37 [65%]) with 20 g day<sup>-1</sup>



psyllium fibre (21/35 [60%]) or a combination of both treatments (21/30 [70%]) ( $p = 0.67$ ) and concluded that the treatments were equivalent for remission maintenance.<sup>198</sup> For the other four studies,<sup>199–202</sup> either clinical outcomes were not reported or the results were reported as percentages without the data to ascertain the number of patients in each group; therefore, the outcome of remission maintenance in UC could not be analysed using GRADE. One further RCT conducted subsequent to these systematic reviews compared prebiotics (15 g day<sup>-1</sup> oligofructose and inulin) with placebo for 6 months and showed no difference in relapse rates for prebiotics (11/35) and control (10/41).<sup>207</sup>

Four studies of CD in remission including 465 patients were identified, and all four studies reported disease outcomes.<sup>129</sup> However, the interventions were heterogenous and the evidence could not be analysed using GRADE. One study of 20 patients showed that a low fibre exclusion diet led to increased remission maintenance rates compared to a 'fibre-rich' refined carbohydrate diet<sup>205</sup>; however, the other three studies of 445 patients showed no difference between placebo and fibre intervention in clinical outcomes.<sup>203–206</sup>

One study compared the effect of an anti-IBD diet (a diet low in animal fat, grains, additives, and high in monounsaturated and  $\omega$ -3 fatty acids) with fructooligosaccharides or placebo and reported that relapse occurred more frequently in the fructooligosaccharides group (6/19 [32%]) than the anti-IBD diet group (0/16 [0%]) ( $p = 0.035$ ), although neither group was significantly different to the placebo group with respect to the rate of relapse (4/19 [21%]).<sup>208</sup>

An RCT in 32 quiescent IBD patients with functional gut symptoms demonstrated that 12 g day<sup>-1</sup> fructans exacerbated abdominal pain, bloating, flatulence and urgency compared to a placebo (12 g day<sup>-1</sup> glucose) after 3 days of supplementation.<sup>209</sup>

## Nutrients

### Vitamin D

**STATEMENT:** There is insufficient evidence to support the use of high dose vitamin D for maintaining remission in Crohn's disease. GRADE very low quality agreement 81.6%.

Two RCTs in 128 patients with quiescent CD and low risk of bias reported 12-month supplementation with vitamin D.<sup>210,211</sup> One study reported six of 46 (13%) patients taking 1200 IU day<sup>-1</sup> vitamin D relapsed compared to 14 of 48 (29%) taking placebo ( $p = 0.056$ ),<sup>210</sup> whereas the second study reported six of

18 (33%) patients taking 10,000 IU day<sup>-1</sup> relapsed compared to 11 of 16 (69%) taking 1000 IU day<sup>-1</sup>.<sup>211</sup> GRADE assessment showed very low quality evidence that vitamin D supplementation had no significant effect on relapse rates at 12 months. As a general health measure vitamin D should be monitored and replaced in deficient individuals.

## Probiotics

**PRACTICE STATEMENT:** There is no evidence for probiotics to provide benefit to maintain disease remission in Crohn's disease. Agreement 90.2%.

Six systematic reviews of probiotics to maintain remission were assessed for RCTs that met the inclusion criteria.<sup>154,155,158,159,161,212</sup> Three studies have assessed probiotics versus placebo to maintain disease remission for 4 weeks to 12 months in CD.<sup>213–215</sup> None of the studies demonstrated lower relapse rates for probiotic (52/133 [39%]) compared to placebo (50/125 [40%]).

**PRACTICE STATEMENT:** There is insufficient evidence to recommend probiotics to maintain disease remission in ulcerative colitis. Agreement 81.8%.

Four studies have assessed probiotics versus placebo to maintain disease remission in UC.<sup>172,214,216,217</sup> One study showed that probiotics (3/15 [20%]) had a lower relapse rate compared to placebo (14/15 [93%]) following 8 weeks,<sup>172</sup> whereas the other studies showed similar rates between probiotics (29/83 [35%]) and placebo (32/61 [52%]) at 4 weeks<sup>214</sup> and 12 months.<sup>216,217</sup>

## SURGERY

Four systematic reviews have assessed the effect of exclusive enteral nutrition on outcomes in pre-surgical CD.<sup>218–221</sup> Ten retrospective case-control studies met the inclusion criteria, and one of these was a study reported subsequent to the systematic reviews. Two studies had low risk of bias,<sup>222,223</sup> seven had unclear risk of bias<sup>224–230</sup> and one had high risk of bias.<sup>231</sup>

These studies reported on at least one of length of post-surgical hospital stay, post-surgical infectious complications or intra-operative stoma rate in patients with CD undergoing surgery for bowel resection, fistula repair or abscess not suitable for drainage. All of studies compared those patients who received pre-surgical

exclusive enteral nutrition to patients who did not receive any nutrition intervention prior to surgery. Most information was from studies at low or unclear risk of bias using the risk of bias tool specifically for assessing case-control studies.

**STATEMENT:** There is limited evidence that pre-surgical exclusive enteral nutrition may reduce the length of post-surgical hospitalisation in patients with Crohn's disease. (GRADE very low quality). Agreement 84.3%.

Three studies reported mean length of post-surgical hospitalisation was 7.9 days in 288 patients who received pre-operative exclusive enteral nutrition compared to 9.9 days in 200 patients who received no nutrition intervention. Between 2 and 8 weeks (mean 6 weeks) of pre-surgical exclusive enteral nutrition was associated with a mean of 2.0 days of reduction in the length of hospital stay.<sup>222,228,232</sup>

One study reported that mean length of post-surgical hospitalisation for high risk surgical patients ( $n = 35$ ) was 7.5 (2.2) days after 3 weeks of pre-surgical exclusive enteral nutrition, which is similar to the length of stay (8.3 [6.2] days) ( $p = 0.222$ ) of low-risk surgical patients who did not receive pre-surgical nutrition ( $n = 21$ ).<sup>231</sup>

**STATEMENT:** Pre-surgical exclusive enteral nutrition may lower the risk of post-surgical infectious complications in patients with Crohn's disease. (GRADE very low quality). Agreement 92.0%.

Nine studies reported the rate of post-surgical infectious complications in patients receiving pre-surgical exclusive enteral nutrition for 2–12 weeks (mean 6 weeks) was 93 of 720 (13%) compared to a rate of 158 of 551 (29%) in patients with no nutrition intervention.<sup>222–226,228,230–232</sup> Eight studies reported a significantly lower rate of post-surgical infectious complication in patients provided with exclusive enteral nutrition.<sup>222–226,228,230,232</sup> One study did not report a significant difference because it compared high risk patients with exclusive enteral nutrition to low-risk patients without exclusive enteral nutrition.<sup>231</sup>

Low quality evidence suggests the rate of intra-operative stoma formation in patients receiving pre-surgical exclusive enteral nutrition may be lower than those receiving standard care,<sup>224,226,228,231</sup> although this requires validation in high quality studies.

Similarly, two of the studies reported avoidance of surgery in more patients after starting pre-surgical exclusive enteral nutrition compared to standard care.<sup>222,228</sup>

## STRICTURES

**STATEMENT:** Exclusive enteral nutrition for 4–12 weeks may induce remission in Crohn's disease patients with inflammatory strictures. (GRADE very low quality). Agreement 84.3%.

Four case series studies<sup>233–236</sup> met the inclusion criteria and reported on at least one of remission or bowel wall thickness in patients with inflammatory or fibrous strictures following treatment with EEN. Information was from two studies at unclear risk of bias<sup>233,236</sup> and two studies at high risk of bias.<sup>234,235</sup>

Combined results from four studies showed that 98 of 114 (85%) patients with stricturing CD achieved remission following exclusive enteral nutrition between 4 and 12 weeks.<sup>233–236</sup> Three studies reported validated measures of clinical response ( $>70$  point reduction in CDAI) and remission (CDAI  $< 150$ )<sup>233,235,236</sup> and one study measured remission as a return to pre-relapse well-being.<sup>234</sup> Despite EEN, 30 of 114 (26%) patients with stricturing CD required surgery after 4–12 weeks of EEN. One study had some overlap between patients that entered remission (reporting pre-relapse level of well-being) and those that went on to require surgery.<sup>234</sup>

Two case series reported a reduction in bowel wall thickness following EEN in stricturing IBD.<sup>233,236</sup> Controlled studies are needed to confirm what impact EEN has on bowel wall thickness and the duration needed to achieve clinical remission and mucosal healing.

## POUCHITIS

**STATEMENT:** A probiotic mixture of eight bacterial strains (De Simone Formulation) may maintain remission in chronic relapsing pouchitis. Very limited evidence may support prophylactic use after pouch formation surgery to prevent initial pouchitis onset. There is no evidence to support the use of other probiotics to maintain remission in pouchitis. (GRADE low quality). Agreement 85.4%.

Five RCTs met the inclusion criteria and reported the effect of probiotics on at least one of maintenance of remission in quiescent pouchitis, reduction in pouch disease activity score in active pouchitis or quality of life. All studies compared probiotic treatment to placebo. Information was from three studies at low risk of bias,<sup>173–175</sup> one study at unclear risk of bias<sup>176</sup> and one study at high risk of bias.<sup>177</sup>

Five RCTs reported rates of remission maintenance in patients with chronic or naïve pouchitis. Four studies

used a probiotic mixture of 8 bacterial strains (De Simone Formulation) and showed that 68 of 76 (89%) patients maintained remission compared to 24 of 68 (35%) in the placebo group.<sup>173–176</sup> Two of these studies measured IBD quality of life scores and reported an improvement with the probiotic.<sup>174,175</sup> One further study showed *Bifidobacterium longum* BB-536 maintained remission in six of seven (86%) patients compared to three of five (60%) patients in the placebo group, although the study was underpowered to detect a difference.<sup>177</sup>

Two other studies showed no benefit of probiotic over placebo in reducing pouch disease activity score, however the sample sizes were relatively small and duration of studies was short.<sup>237,238</sup>

## STOMA

**PRACTICE STATEMENT:** Appetite may be reduced following ileostomy or colostomy formation so encourage small frequent energy dense meals to meet nutritional requirements. Agreement 81.6%.

There are no studies on dietary intake immediately following ileostomy or colostomy formation. The appetite may be reduced, so encourage small frequent energy dense meals to meet nutritional requirements and use oral nutritional supplements where appropriate.

**PRACTICE STATEMENT:** Encourage a wide variety of foods for people with an ileostomy or colostomy to ensure a healthy varied diet. No foods are specifically contraindicated, however some foods have been associated with problems. Agreement 94.0%.

There is limited evidence on the dietary management of an ileostomy or colostomy. A wide variety of foods is encouraged. Various foods have been associated with odour, flatus, increased residue, irritation and increased water content of stoma output (Table 9).<sup>239–246</sup> Not all of these foods are likely to cause symptoms in isolation and larger portions, or multiple foods from one group within a meal, are more likely to cause symptoms whereas small portions may be well tolerated.

Many of the foods associated with increased flatus and increased liquid stoma volume are also high in FODMAPs. FODMAPs are short-chain fermentable carbohydrates that have been associated with increased delivery of endogenous fluid into the gastrointestinal lumen. An RCT of a high versus a low FODMAP diet in 10 patients with an ileostomy and no active disease demonstrated that the low FODMAP diet reduced the wet and dry stoma content; however, the reduction may not be clinically significant.<sup>247</sup> Whether a low FODMAP diet is beneficial in patients with an ileostomy to maintain hydration status alongside a reduction in stoma volume has not been assessed.

**PRACTICE STATEMENT:** Encourage patients with an ileostomy or colostomy to have a dietary fibre intake in line with population recommendations. Dietary fibre may reduce the volume of stoma output but it depends on the physiological properties of the dietary fibre. Agreement 80.4%.

The evidence does not support recommending a different fibre intake for people with an ileostomy or colostomy compared with the general population. Dietary fibre may reduce the volume of stoma output but it depends on the physiological properties of the dietary fibre. A study in 10 patients (CD,  $n = 5$ ; UC,  $n = 5$ ) with an ileostomy compared a diet high in sucrose and refined cereals with a

**TABLE 9** Foods potentially leading to symptoms associated with stoma

Symptom	Foods
Stool odour	asparagus, beans, broccoli, Brussels sprouts, cabbage, cauliflower, egg, fish, garlic, onions
Increased flatus	beans and pulses, beer and lager, broccoli, Brussels sprouts, cabbage, carbonated drinks, cauliflower, cucumber, turnip
Increased stoma residue	beans and pulses, cabbage (raw only), carrot (raw only), celery, citrus fruit, coconut, dried fruit, fruit and vegetable skins, lettuce, mushrooms, nuts and seeds, pineapple, popcorn, sweetcorn, tomatoes
Irritation	carrot (raw only), chilli, citrus fruit, nuts and seeds
Increased liquid stoma volume	alcohol, strawberries, grapes, peaches, raisins, bananas, prune juice, baked beans, whole wheat cereals, sweetcorn, apples, potatoes, bread, pineapple, pears, rhubarb spicy food, Chinese food, fried food

*Note:* Not all of these foods are likely to cause symptoms in isolation and larger portions, or multiple foods from one group within a meal, are more likely to cause symptoms whereas small portions may be well tolerated.



diet low in sucrose and unrefined cereals. They showed that effluent weight was lower for the low sucrose and unrefined cereal diet compared with the high sucrose and refined cereal diet.<sup>248</sup>

A study in 38 patients with an ileostomy investigated the effect of adding 7 g day<sup>-1</sup> psyllium husk to a low fibre diet on stoma bag use, as a proxy for stoma output volume.<sup>249</sup> The addition of psyllium husk resulted in decreased output volume and patients used one ostomy bag less per day than patients who did not use psyllium husk.

## FISTULA

Two uncontrolled case series studies met the inclusion criteria (Table 1) and reported on rates of fistula closure in patients with CD after 12 weeks treatment with EEN. Because the only data available is from uncontrolled studies these were assessed using the QATQS risk of bias tool as recommended in the Cochrane handbook, both studies were considered weak evidence or at high risk of bias.<sup>236,250</sup>

**STATEMENT:** There is very limited evidence that fistulating Crohn's disease may respond to exclusive enteral nutrition. (GRADE very low quality). Agreement 82.0%.

Combined results from two studies, one at unclear risk of bias<sup>236</sup> and one at high risk of bias,<sup>250</sup> showed that 57 of 81 (70%) patients that received 12 weeks of EEN had fistula closure by the end of treatment.<sup>236,250</sup> These findings may be exaggerated because patients unable to tolerate EEN were excluded from analysis; therefore, the data are per protocol only. There is no evidence that EEN is superior to standard care because there was no comparator group in either study.

## SHORT BOWEL SYNDROME

**PRACTICE STATEMENT:** Parenteral nutrition is required where patients cannot meet their nutritional needs via the enteral route. Agreement 91.8%.

Parenteral nutrition is beyond the scope of these guidelines and recommendations are available in the ESPEN 2017 guidelines on chronic intestinal failure in adults.<sup>251</sup> Use of the enteral nutrition and/or oral diet is to be encouraged where it is not contraindicated to stimulate intestinal adaptation. Avoid long periods of patients being left nil by mouth.

**PRACTICE STATEMENT:** Site and extent of resection and integrity of the remaining bowel are important considerations in the clinical assessment of short bowel syndrome. Agreement 97.9%.

The normal small intestine varies hugely in length, however following resection a residual length of <200 cm is an indication of a short bowel and can lead to nutritional deficiencies if not appropriately managed. For patients without a colon (jejunostomy), the ability to reabsorb fluid and electrolytes is lost. Where the residual jejunal length is <100 cm, parenteral replacement of fluids and electrolytes will be required. Where the jejunal length is <50 cm, long-term parenteral nutrition will additionally be required.<sup>252</sup> Understanding the integrity and which parts of the residual ileum and colon are present will determine what absorptive capacity remains; thus, where disease is still present, longer lengths of bowel may lead to short bowel syndrome.

**PRACTICE STATEMENT:** Encourage patients with short bowel syndrome to eat a wide variety of normal foods to compensate for hyperphagia caused by malabsorption. Agreement 84.8%.

Energy and macronutrient absorption are limited in patients with short bowel syndrome. Three studies in patients with ileum <200 cm (12 patients with a jejunostomy and 46 patients with a jejuno-colic anastomosis) have demonstrated that absorption of energy and macronutrients is approximately two-thirds of dietary intake indicating a need for hyperphagic diets in short bowel syndrome.<sup>253–255</sup> Energy and protein requirements may be increased from 30 to 60 kcal kg<sup>-1</sup> day<sup>-1</sup> and 1.25–1.5 g kg<sup>-1</sup> day<sup>-1</sup>, respectively.<sup>253–255</sup>

**PRACTICE STATEMENT:** The bowel adapts over time to improve nutrient absorptive capacity agreement 91.7%.

Following extensive ileal resection, parenteral nutrition is often needed in the short term. It is important that the enteral route is used where possible because hormonal growth factors, glucagon like growth factor-2, nutrients, bile and pancreatic secretions will enable morphological and functional adaptation of the gut, particularly distal to the anastomosis.<sup>256</sup> The ileum dilates and increased villi crypt height and depth improve tolerance of enteral

nutrition and oral diet.<sup>256</sup> The process can take months or even years and the exact mechanisms are not fully understood.

**PRACTICE STATEMENT:** In patients with short bowel syndrome where the colon is preserved (jejunocolic anastomosis), a diet high in complex carbohydrates and low in fat is advised. Consider using medium-chain triglycerides to replace some long-chain triglycerides and ensure the diet is not deficient in essential fatty acids and fat-soluble vitamins. Agreement 89.8%.

In patients with a jejunocolic anastomosis where small bowel length <200 cm, a diet containing 50%–60% energy from carbohydrate enables salvage of up to 1000 kcal day<sup>-1</sup> from fermentation of unabsorbed carbohydrates in the colon.<sup>257,258</sup>

A low fat diet (20%–30% energy from fat) can reduce diarrhoea; however, it may be unpalatable. Substituting long-chain triglycerides with medium-chain triglycerides increases fat absorption from 23% to 58% and energy absorption from 46% to 58% without increasing stool output.<sup>259</sup>

Ensure the diet contains adequate amounts of essential fatty acids and fat-soluble vitamins and, if not, use supplements to prevent deficiency.

**PRACTICE STATEMENT:** In patients with short bowel syndrome and a jejunostomy, a diet with normal carbohydrate and fat intakes are advised. Agreement 89.1%

In patients with short bowel syndrome and a jejunostomy, advise a normal amount of energy from carbohydrate (40%–50%) and fat (30%–40%) in the diet. Evidence indicates that patients with a jejunostomy benefit from the concentrated source of fat in the diet rather than a low fat diet.<sup>253</sup>

**PRACTICE STATEMENT:** In patients with short bowel syndrome where the colon is preserved (jejunocolic anastomosis), a diet low in oxalates will prevent renal calculi. Agreement 80.4%.

Preferential binding of calcium and magnesium to unabsorbed long-chain fatty acids in the colon releases oxalate which may be absorbed and predispose patients to developing renal calculi. A diet low in oxalates and moderate in fat is advised (Table 10).<sup>252</sup>

**TABLE 10** Dietary sources of oxalates that should be avoided in patients with short bowel syndrome and a colon

- Spinach
- Beetroot
- Rhubarb
- Chocolate
- Cocoa and carob
- Peanuts and almonds
- Bran flakes
- Tea (more than 2–3 cups day<sup>-1</sup>)
- Parsley

*Note:* Oxalate content may vary by country of origin, season and cooking method. Advise avoidance of high oxalate foods alongside an adequate calcium intake (800–1200 mg day<sup>-1</sup>) or calcium supplement if dietary intake is inadequate.<sup>260</sup>

**PRACTICE STATEMENT:** Lactose intolerance in short bowel syndrome should be assessed on a case by case basis. Agreement 88.0%.

A study in 14 stable patients with short bowel syndrome demonstrated that lactose was well tolerated as a 20-g load or as part of a normal lactose containing diet; thus, there is no need to routinely restrict lactose.<sup>261,262</sup>

**PRACTICE STATEMENT:** An increase in dietary fibre intake does not improve macronutrient or fluid absorption in short bowel syndrome. Agreement 82.6%.

A study in six patients with a jejunocolic anastomosis showed that pectin fibre had little effect on colonic fermentation and starch was more important for macronutrient and fluid absorption.<sup>263</sup> The bulking effect of dietary fibre is not helpful in short bowel syndrome and dietary fibre can inhibit fat and mineral absorption.<sup>264</sup> Thus, assess dietary fibre intake and consider a trial of fibre reduction if stool output is high. Anecdotally, some patients with a jejunostomy find taking a fibre supplement improves (thickens) effluent consistency.

**PRACTICE STATEMENT:** In patients with a jejunostomy at risk of dehydration, advise use an oral rehydration solution to optimise sodium and fluid absorption. Agreement 95.7%.

To optimise sodium and electrolyte absorption in jejunostomy patients at risk of dehydration as a result of increased gastrointestinal losses, limit hypotonic and hypertonic oral fluids to 500 ml day<sup>-1</sup>. Give an oral

rehydration solution containing 90 mmol L<sup>-1</sup> sodium (20 g glucose, 3.5 g sodium chloride and 2.5 g sodium bicarbonate dissolved in 1 L of water).<sup>265–267</sup>

**PRACTICE STATEMENT:** In all patients with a jejunostomy, encourage sprinkling salt onto food. Add 0.5–1 g of salt per day. Agreement 87.5%.

Adding salt to food (0.5–1 g day<sup>-1</sup>) is useful for maintaining good hydration status in patients with a jejunostomy.

## FUNCTIONAL BOWEL SYMPTOMS

In line with clinical guidelines, patients with IBD and functional gut symptoms should be provided with first line dietary and lifestyle advice before implementing any dietary restrictions.<sup>268–271</sup> Three RCTs met the inclusion criteria and reported on at least one of global symptom improvement of functional symptoms or improvement in composite functional symptom score in patients with IBD following a low FODMAP diet compared to either a placebo diet or habitual diet. Information was from two studies at unclear risk of bias<sup>121,272</sup> and one study at high risk of bias.<sup>120</sup>

**STATEMENT:** A low FODMAP diet may improve global functional bowel symptoms in quiescent or mildly active inflammatory bowel disease. (GRADE very low quality). Agreement 90.2%.

Two studies reported a combined global symptom response to the low FODMAP diet of 44 of 71 (62%) compared to 23 of 70 (33%) following a control diet (OR = 3.3, 95% CI = 1.7–6.7) ( $p = 0.0007$ ).<sup>120,121</sup> One study was an RCT that compared the low FODMAP diet with a placebo sham diet<sup>121</sup> and the other study was a non-blinded study comparing the low FODMAP diet with habitual diet<sup>120</sup>; therefore, the evidence was downgraded.

**PRACTICE STATEMENT:** Dietitian-led low FODMAP education in inflammatory bowel disease may be used for persistent bowel symptoms in the absence of inflammation. This involves short-term FODMAP restriction, FODMAP reintroduction to tolerance and personalisation for long-term management. Agreement 93.9%.

Two studies reported symptom scores following the low FODMAP diet compared a control diet.<sup>120,272</sup> One study was a small cross-over study in patients with quiescent CD ( $n = 9$ ) and reported that visual analogue scale scores were lower in the last 2 weeks following the low FODMAP diet (13.5 mm, 95% CI = 5.9–21.1) compared to the typical Australian diet (24.8 mm, 95% CI = 12.6–37.0) ( $p < 0.001$ ).<sup>272</sup> The second study was an open label study in both active and inactive IBD and reported that median (IQR) irritable bowel syndrome severity scoring system (IBS-SSS) scores at week 6 were lower (115 [33–169]) after the low FODMAP diet compared to the habitual diet (170 [91–288]) ( $p = 0.02$ ).<sup>120</sup>

Two studies have assessed provocation with FODMAPs in IBD patients with functional symptoms who had first been instructed to follow a low FODMAP diet.<sup>209,273</sup> One RCT in 32 patients demonstrated that 12 g day<sup>-1</sup> fructans exacerbated abdominal pain, bloating, flatulence and urgency compared to a placebo (12 g day<sup>-1</sup> glucose) after 3 days of supplementation,<sup>209</sup> whereas the other in 19 patients showed that provocation with FODMAPs and placebo led to similar pain and bloating levels as at baseline.<sup>273</sup>

## SPECIAL SITUATIONS

### Extra-intestinal manifestations

**PRACTICE STATEMENT:** There is no evidence for providing dietary advice to manage extraintestinal manifestations of inflammatory bowel disease, however optimisation of nutritional status and induction of disease remission are important considerations. Agreement 91.8%.

Refer to BSG guidelines and ECCO guidelines on how to manage extra-intestinal manifestations of IBD.<sup>47,274,275</sup>

### OFG

One case series met the inclusion criteria and reported on either clinical response in patients with OFG following a nutrition intervention.

**PRACTICE STATEMENT:** A cinnamon and benzoate free diet may be effective in reducing oral disease activity score in patients with orofacial granulomatosis. Agreement 93.6%.

Patients with OFG ( $n = 32$ ) received dietary advice on a cinnamon and benzoate free diet for 8 weeks. Only 25 patients completed the study and nine patients had gut involvement. A clinical response was reported in 18 of 25 (72%) patients.<sup>276</sup> A cinnamon and benzoate free diet is a first-line treatment option for patients with OFG; however, the mechanisms are not understood. Dietary resources on a cinnamon and benzoate free diet are available online ([www.kcl.ac.uk/ofg](http://www.kcl.ac.uk/ofg)) for healthcare professionals working with patients with OFG.

## Upper gastrointestinal Crohn's disease

**PRACTICE STATEMENT:** There is no evidence for providing dietary advice to manage upper gastrointestinal Crohn's disease; however, optimisation of nutritional status and induction of disease remission are important considerations. Agreement 83.3%.

Proton pump inhibitors are often used to treat upper gastrointestinal Crohn's disease and may affect iron, vitamin B<sub>12</sub>, vitamin C and vitamin D, and possibly magnesium status, as well as bone mineral density. Refer to BSG guidelines and ECCO guidelines on how to manage upper gastrointestinal Crohn's disease.<sup>47,101,274</sup>

## Perianal IBD

**PRACTICE STATEMENT:** There is no evidence for providing dietary advice to manage perianal inflammatory bowel disease; however, optimisation of nutritional status and induction of disease remission are important considerations. Agreement 96.0%.

Refer to BSG guidelines and ECCO guidelines on how to manage perianal IBD.<sup>47,274,277</sup>

## CONCLUSIONS

These British Dietetic Association consensus guidelines on the nutritional assessment and dietary management of IBD have been developed with input from all the major UK stakeholders involved in the management of IBD including patient representation through collaboration with Crohn's and Colitis UK. Involving the IBD multi-disciplinary team has enabled these guidelines to consider that patient choice is central to the development of treatment plans.

The guidelines have been developed to improve access to the current research evidence and ultimately aim to increase equality of care in nutritional assessment and dietary management of IBD. They comprise the most up-to-date recommendations with statements and practice statements agreed by consensus on nutritional assessment and nutritional screening tools, as well as dietary management strategies, for treatment of malnutrition; induction and maintenance of disease remission; surgery; stricturing disease; pouchitis; stoma; fistula; short bowel syndrome; functional bowel symptoms; and special situations including OFG.

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## CONFLICT OF INTERESTS

The authors have no conflicts of interest. All members of the eDelphi Consensus Group signed conflicts of interest forms during the development of these guidelines.

## AUTHOR CONTRIBUTIONS

Miranda C. E. Lomer was the grant holder. Miranda C. E. Lomer is the IBD lead of the Gastroenterology Specialist Group of the BDA. Miranda C. E. Lomer, Bridgette Wilson and Catherine L. Wall were involved in the conception, development and analysis of the guidelines. Miranda C. E. Lomer and Catherine L. Wall recruited the expert IBD panel. Miranda C. E. Lomer recruited members of the IBD community to take part in the consensus voting. Miranda C. E. Lomer, Bridgette Wilson and Catherine L. Wall wrote, edited and approved the final version of the manuscript submitted for publication.

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## SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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