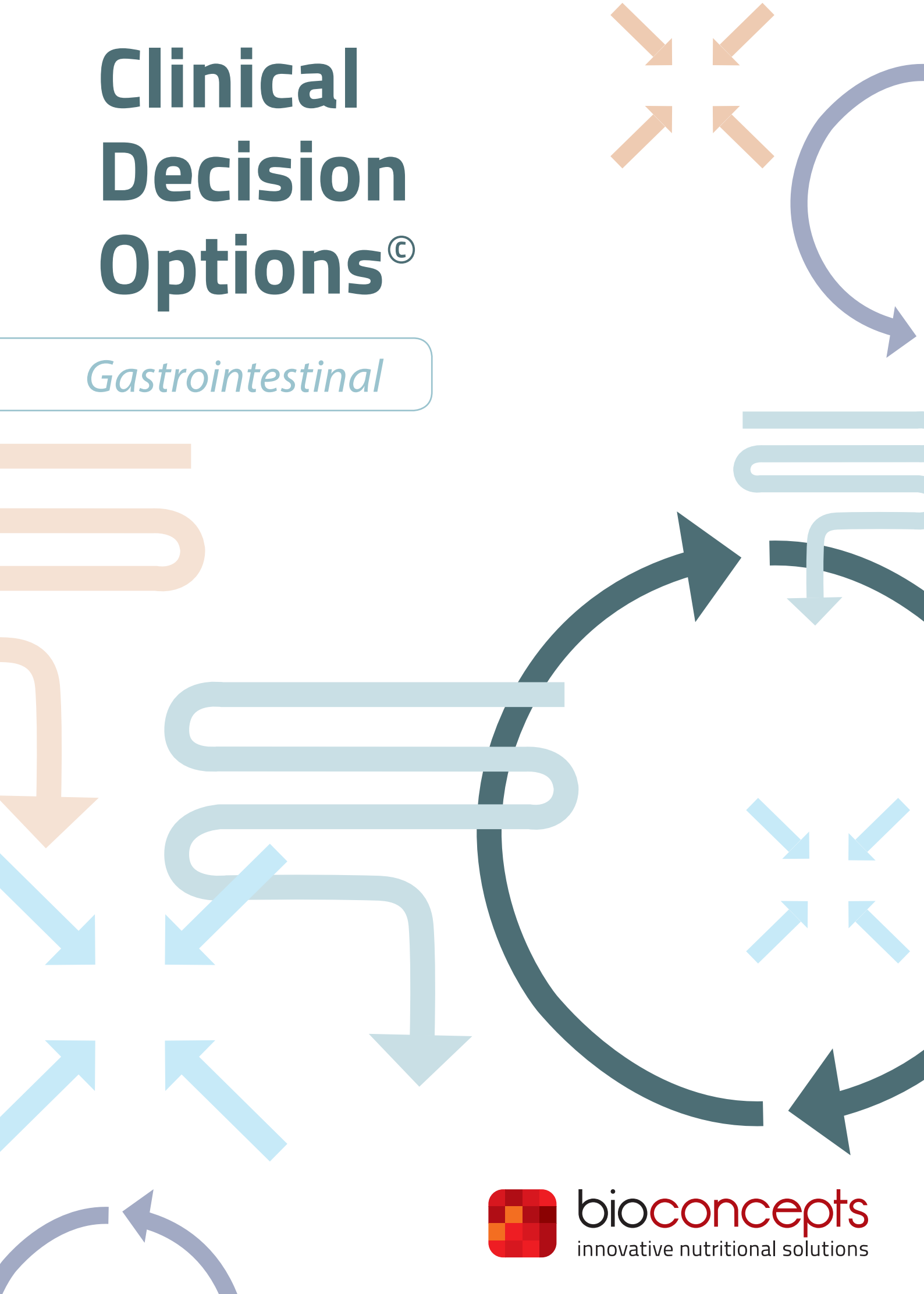
Clinical Decision Options©

Gastrointestinal



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Introducing Bio Concepts **Clinical Decision Options (CDO)©**

What is a CDO?

The Bio Concepts Clinical Decision Options (CDO) is a new tool created to support your clinical choices and deliver superior information for therapeutic considerations, strategies and outcomes.

What does the CDO offer as a clinical tool?

Bio Concepts has developed CDOs to assist you in your critical decision-making process, as an adjunct to your own clinical experience and expertise, in the formulation of effective treatment plans for your patients. This Gastrointestinal CDO is designed to help identify symptoms, determine appropriate testing, interpret results and guide your clinical considerations with researched ingredients to deliver the best possible clinical outcomes. The Bio Concepts Gastrointestinal CDO also includes clinical outlines for gut repair, function and maintenance and wherever you see these icons 1 2 3 refer to pages 12 and 13 of this booklet.
Important note: Throughout this booklet we have highlighted the 'red flag' medical referral moments with this icon !

Table of Contents

Produced strictly for practitioner use only. All the information provided is generic, not diagnostic or prescriptive.

Common GIT Disorder Presentations

There are many gastrointestinal symptoms and disorders that patients may experience. Among the most common are adverse food reactions and GIT infection.

Presentation 1

Adverse Food Reactions

Refer to page 4-7

Symptoms associated with adverse food reactions:

- Abdominal pain
- Bloating
- Constipation & diarrhoea
- Nausea
- Hypersensitivity
- Breathing difficulties & asthma
- Headaches & migraine
- Fluid retention
- Skin rashes, acne, eczema & psoriasis
- Anxiety & depression
- Dizziness, fatigue & lethargy
- Pre-existing conditions:
  - Gastric ulcers
  - Irritable Bowel Syndrome (IBS)
  - Coeliac Disease
  - Crohn's Disease (CD)
  - Arthritis
  - Autism
  - Epilepsy
  - Fibromyalgia
  - Multiple Sclerosis
  - Chronic Fatigue Syndrome

Presentation 2

GIT Infection

Refer to page 8-11

Symptoms associated with GIT infection:

- Nausea
- Vomiting
- Fever
- Loss of appetite
- Fatigue
- Muscle aches
- Dehydration
- Headache
- Mucous or blood in the stool
- Bloating
- Abdominal pain
- Diarrhoea
- Constipation
- Belching
- Flatulence
- Weight loss

Presentation 3

Refer to page 4-11

Symptoms associated with adverse food reactions & GIT infection:

- Any combination of the above listed symptoms.

The CDO tool is not for use in a medical emergency. Refer immediately for acute abdominal pain emergencies which include: "worst ever ripping or tearing" to describe pain; fever; occult blood or blood in the urine; pregnancy; uncontrolled vomiting; lightheadedness on standing; acute onset of pain; intensifying pain over time; trauma to abdomen; abdominal pain with walking; distended abdomen; excessive abnormal sweating with pain; pain which awakens from sleep or a pulsatile abdominal mass.

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Adverse Food Reactions

Presentation 1 or 3

Immune Mediated

Autoimmune Food Reactions

T-cell mediated eg. Coeliac Disease

Investigations:
- Diet & symptom diary *(See Bio Concepts Diet & Symptom Diary)*

Refer: to the GP for Coeliac serology testing and diagnostic investigations
- Autoantibodies: tTG-IgA, EMA-IgA, DGP-IgG (indicative, not diagnostic)
- Gene testing: HLA-DQ2 & HLA-DQ8 (shows genetic predisposition, not diagnostic)
- Small intestinal endoscopy & biopsy (diagnostic)
- A Complete Digestive Stool Analysis (CDSA) may reveal more information about inflammatory markers and digestion which may be useful ***Refer to GIT Infection Chart (pages 7-10)***

Important note: Dietary intake of gluten is required for accurate testing results. If a patient is undergoing testing, do not remove gluten from the diet until investigations are complete; 21% of Coeliac patients have no symptoms; 27% present with diarrhoea, weight loss & malabsorption; other common presentations include constipation, anaemia, osteoporosis, neurological disorders or dermatitis herpetiformis.

Symptom management: Patients who have Coeliac Disease or gluten sensitivity may experience symptoms such as nausea, vomiting, abdominal pain and diarrhoea during the testing process due to increased gluten intake advised during this time. Support with herbal digestive teas (fennel, peppermint, ginger, chamomile), digestive enzymes, adequate hydration & supplementation to manage the potential increase in nutrient loss.

Mechanisms behind autoimmune conditions include chronic inflammation driven by abnormal cytokine biology & higher than normal levels of autoreactive CD4+ T-cells

Refer: Depending on the severity of the case, patients may be referred to a specialist physician for assessment and to monitor their condition periodically.

Investigate:
Consider other food reactions
Refer to Adverse Food Reactions Chart (pages 4-7)
If dysbiosis or GIT infection is suspected
Refer to GIT Infection Chart (pages 8-11)
Consider a differential diagnosis.

1 2 3

Clinical Considerations:
Phase 1-3: Gut Repair, Function & Maintenance

Main goals: Reduce inflammation & repair the gut lining, support the microbiota & optimal immune function & address nutritional deficiencies commonly associated with Coeliac Disease if present (protein, Vitamins A & D, Iron, B12, Folate, Zinc & Magnesium).

Diet: Strict lifelong avoidance of gluten-containing foods, consider cross-reactive foods, diverse whole food diet, no alcohol, consider a Mediterranean Diet (high in Omega-3).

Lifestyle: Avoid cooking items such as toasters & chopping boards that come in contact with gluten-containing foods, be cautious when eating out, gluten-free products are not always highly nutritious – keep the diet as diverse as possible to support the core microbiota & prevent nutritional deficiencies. Minimise stress which often exacerbates Coeliac disease & avoid environmental triggers (autoimmune disease can move in and out of remission based on health and lifestyle factors).

Food Allergies

IgE mediated reactions usually occur immediately or within a few hours of consumption

Refer: Patients experiencing breathing difficulty, swelling of the tongue or any symptoms suggesting anaphylaxis require urgent assessment by a specialist physician.

Investigations:
- Antigen specific serum IgE testing
- Diet & symptom diary may assist to identify the likely food trigger. Common food allergies include milk, eggs, nuts, soy, sesame, fish & shellfish.
- Elimination diet & food challenge may be required if the patient is reacting to multiple foods.
- Assess all supplement ingredients used during this time (if any permitted) for possible triggers. *(See Bio Concepts Elimination Diet handout).*

1 2 3

Clinical Considerations:
Phase 1-3: Gut Repair, Function & Maintenance

Main goals: Reduce symptoms, strict avoidance of reactive foods generally for life (sensitisation programs may be offered as an option through a specialist immunologist), support immune function & stabilise mast cells, support digestion, reduce inflammation & repair the gut lining & prolonged gut healing may improve food tolerance.

Diet: Strict avoidance of reactive foods, diverse, whole food diet, a long-term restricted diet is not recommended, diversifying the diet as much as possible to include high quality prebiotic foods may help to enhance the core microbiota and, in turn, support a healthy immune response.

Investigate:
Consider other food reactions
Refer to Adverse Food Reactions Chart (pages 4-7)
If dysbiosis or GIT infection is suspected
Refer to GIT Infection Chart (pages 8-11)
Consider other IgE reaction causes such as topical or inhaled triggers. Consider a differential diagnosis.

Peptic Ulceration and GORD

Investigations:
- Hydrogen breath test (*H.pylori*)
- Barium oesophagram
- Endoscopy & biopsy

1 2 3

Clinical Considerations:
Phase 1-3: Gut Repair, Function & Maintenance

Main goals: Reduce symptoms, reduce BMI, insulin resistance, reduce stress & detrimental lifestyle factors, address hypochlorhydia and support digestion. Avoid nonsteroidal anti-inflammatory drugs (NSAIDs) both as a causal and maintaining factor.

Refer: Patients should consult their prescribing physician for assessment of their medications eg. Proton-pump inhibitors (PPIs), nitroglycerine, anticholinergics & benzodiazapenes may impact digestive health.

Diet: Avoid trigger foods eg. chocolate, coffee, alcohol (also affects sphincter function), chilli & fatty meals; protein sufficiency is important to ensure healthy collagen production 0.8-1.2g/kg, encourage a diverse, whole food diet adapted for weight loss if required; eat slowly & small meals more regularly. Avoid peppermint oil which is commonly used for indigestion but may negatively affect sphincter function.

Food Sensitivities

IgG, IgA & IgM mediated reactions usually occur hours to days after consumption

Investigations:
- Antigen specific serum IgG/IgG4 & IgA testing
- Serum LPS & zonulin testing (non-specific intestinal barrier function tests)
- Lactulose/mannitol testing (indicative of intestinal permeability from all causes)
- Diet & symptom diary *(See Bio Concepts Diet & Symptom Diary)*
- Elimination diet *(See Bio Concepts Elimination Diet handout)*

Likely aetiology: intestinal permeability

1

Clinical Considerations:
Phase 1-3: Gut Repair, Function & Maintenance

Main goals: Reduce symptoms, remove reactive foods/food groups, promote gut repair, reduce inflammation, stabilise mast cells & prevent histamine release, support immune function & microbiota diversity. Withhold fermentable fibre until inflammation &/or dysbiotic overgrowth have resolved to avoid symptom exacerbation. Support digestion & nutrient assimilation.

Diet (Phase 1-3): Diverse, whole food diet, strictly avoid reactive foods & include therapeutic gut healing foods such as bone broths. Replace & balance the nutrients lacking due to eliminated foods. Consider additional dietary modifications if required. FODMAPs diet short-term only as restricted diets may compromise long-term outcomes for the microbiota.

Investigate:
Consider other food reactions
Refer to Adverse Food Reactions Chart (pages 4-7)
If dysbiosis or GIT infection is suspected
Refer to GIT Infection Chart (pages 8-11)
Consider a differential diagnosis.

2

Clinical Considerations:
Phase 2: Gut Repair & Function
Main goals: Continue reducing inflammation, soothe the GIT mucous membrane. Introduce fermentable fibres to help restore microbiota balance & diversity & improve bowel function/motility. Reduce pathogenic load. If present ***refer to GIT Infection Chart (pages 8-11).*** Address the gut-brain axis & relieve anxiety (if present) & support detoxification.

3

Clinical Considerations:
Phase 3: Gut Repair & Maintenance

Main goals: Continue active maintenance of the GIT integrity, epithelial barrier function & gap junctions. Reduce pathogenic load. If present ***refer to GIT Infection Chart (pages 8-11).*** Promote the growth of beneficial bacteria & microbiota diversity. Continue to reduce inflammation. Educate patients on how to avoid exacerbations by considering dietary & lifestyle choices eg. alcohol consumption. Prevent infection if travelling overseas.

Continue Phase 3 Repair & Maintenance until symptoms subside and then consider re-introducing eliminated foods one-by-one into the diet from least reactive to most reactive, while staying on the Phase 3 supportive nutrients. If an adverse reaction occurs, consider eliminating this food for a longer period, continue Phase 3 Repair & Maintenance and attempt reintroduction again later in the healing process. Encourage an ongoing diverse diet, including Fructooligosaccharide (FOS) & Galactooligosaccharide (GOS)-containing foods to enhance the foundations of the core microbiota & diversify native populations.

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Adverse Food Reactions

Presentation 1 or 3

Non-Immune Mediated

Non-Toxic Reactions

Food Intolerances

Food intolerances can be dependent on dose; minimal amounts of a certain food or food group may not cause a reaction, however, moderate to large doses may eg. 25g of fructose will cause symptoms in up to 50% of the general population.

Enzymatic

eg. lactose or fructose intolerance

Pharmacological

eg. amines, salicylates

Biochemical

eg. caffeine & additives: MSG, colours, preservatives, nitrates, sulfites

Investigations:
- Diet & symptom diary *(See Bio Concepts Diet & Symptom Diary)*
- Elimination diet *(See Bio Concepts Elimination Diet handout)*
- Hydrogen/methane breath testing (to inform the dietary source of malabsorption eg. lactose)

Important note: Avoid antibiotics for 1 month prior to breath testing to avoid false negative results.

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Likely aetiology: intestinal permeability, genetic factors, enzyme deficiency

Clinical Considerations: 1 2 3
Phase 1-3: Gut Repair, Function & Maintenance

Main goals: Reduce symptoms, remove reactive foods/food groups, reduce inflammation, promote gut repair, support immune function, stabilise mast cells & prevent histamine release, support microbiota diversity, improve bowel function & motility & support detoxification.

Withhold fermentable fibre until inflammation &/or dysbiotic overgrowth have resolved to avoid symptom exacerbation. Support digestion & nutrient assimilation.

Diet: Diverse, whole food diet (organic where possible as reactions may be associated with herbicides or pesticides), apply dietary modifications eg. low amine diet, low FODMAP diet, lactose free diet, avoid additives, preservatives, colours (largely pre-packaged foods) & reassess after completing the 3 Phases of gut healing.

Replace & balance the nutrients lacking due to eliminated foods.

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Investigate:
Consider other food reactions
Refer to Adverse Food Reactions Chart (pages 4-7)
If dysbiosis or GIT infection is suspected
Refer to GIT Infection Chart (pages 8-11)
Consider a differential diagnosis.

Toxic Reactions

Food Poisoning

eg. Hepatitis A, *Salmonella, Listeria, E.coli*

! ***Refer:*** Patients experiencing acute infection require assessment by a physician & minimal exposure to other people due to the highly infectious nature of these conditions.

Investigations:
- Stool analysis
- Anti-HAV

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Investigate:
Refer to Infection Chart (pages 8-11) for clinical considerations for post infection recovery.

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Investigate:
Consider other food reactions
Refer to Adverse Food Reactions Chart (pages 4-7)
If dysbiosis or GIT infection is suspected
Refer to GIT Infection Chart (pages 8-11)
Consider a differential diagnosis.

SIBO or Candida

Suspected

Investigations:
Refer to Infection Chart (pages 8-11)

IBS

Up to 50% of patients may have SIBO

Investigations:
- IBS: Rome III & IV criteria, consider food sensitivity testing ***Refer to Food Sensitivities (page 4)***
- Diverticular Disease: colonoscopy/endoscopy
- Symptoms: flatulence, dyspepsia, constipation/diarrhoea, impaired detoxification

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IBS or Functional GIT Disorders

Clinical Considerations: 1 2 3
Phase 1-3: Gut Repair, Function & Maintenance

Main goals: Reduce symptoms, remove reactive foods/food groups (if any), promote gut repair, reduce inflammation, support immune function, support microbiome diversity, withhold fermentable fibre until inflammation and/or dysbiotic overgrowth have resolved, support digestion & nutrient assimilation. Address the gut-brain axis: stress appears to stimulate the sympathetic nervous system which impairs optimal digestion, leading to symptoms. There are altered serotonin levels in some patients with functional GIT disorders. Serotonin is required to stimulate the enteric nervous system to initiate secretion and peristalsis. Too much serotonin = diarrhoea and too little = constipation. Medications may alter gut flora & increase the chances of dysbiotic overgrowth (NSAIDs, antibiotics, contraceptive pill, antihistamines, PPIs & increase the chance of nutrient deficiencies. Prolonged diarrhoea may cause nutrient loss: refer for testing if required (Full Blood Count, Urea & Electrolyte Count, Iron Studies etc).

Diet: Refer to Food Intolerances (page 4)

GIT Infection

Presentation 2 or 3

Pathology-driven differential diagnosis of GIT infection

Stool analysis

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Microbial overgrowth

Positive for *E. coli; Enterococcus faccium; Aeromonas spp.; Campylobacter spp.; Shigella spp.; Salmonella spp.; Yersinia enterocolitica; Citrobacter freundii; Staphylococcus aureus; Staphylococcus spp.; Helicobacter pylori; Klebsiella pneumoniae; Streptococcus spp.; Giardia intestinalis; Cryptosporidium spp.; Entamoeba histolytica; Blastocystis spp.; Candida albicans; Candida spp.;* round worms; tape worms; flukes

1 2

Clinical Considerations:
Week 1 to 12

1. Cycle antimicrobials containing thyme, clove and oregano, alongside herbs black walnut, wormwood, pau d'arco, barberry and garlic for 4 days with *Saccharomyces boulardii* for 3 days until asymptomatic.
2. ***Phase 1: Gut Repair (page 12)***

Diet: Ensure adequate hydration and recommend easily digestible foods such as smoothies, soups, slow cooked meals; include fruits and cooked vegetables (for easy digestion), ground flaxseeds and oat bran to aid mucous production in the colon to help reduce the colonic pH making an inhospitable environment for parasites.

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1 2 3

Asymptomatic:
Phase 1-3: Gut Repair, Function and Maintenance (page 12-13)

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1 2 3

Symptomatic:
Consider biofilm eradication with Quercetin, NAC, Vitamin C and essential oils oregano, thyme and clove. Once asymptomatic, move on to ***Phase 1: Gut Repair (page 12)*** for 6-12 weeks then ***Phase 2: Gut Repair & Function (page 12)***.

If still asymptomatic, go on to ***Phase 3: Gut Repair & Maintenance (page 13).***

Investigate:
Negative to the above pathogens, look at other important markers in stool analysis (page 10); or ***Refer to Adverse Food Reactions Chart (pages 4-7).***

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Investigate:
Other stool analysis markers (see page 10) and differential diagnosis. ***Refer to Adverse Food Reactions Chart (pages 4-7).***

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Candida albicans

An opportunitistic yeast infection that produces symptoms in a dysbiotic gut environment. Genital candidiasis (most common in women): extreme vaginal itching with soreness and redness in the area; white clumpy discharge that can resemble cottage cheese; and painful intercourse. Oral thrush (infants, the elderly and those with compromised immune systems): white spots inside the mouth and on the tongue; sore and red in the mouth and throat; sometimes difficulty swallowing.

Clinical Considerations:
Step 1: 3-4 weeks
Anti-fungals: Clove, oregano and thyme essential oils; wormwood and barberry
Saccharomyces boulardii (as effective as the anti-fungal medication Nystatin) and *B. longum* BB536 (decreased number of candida counts compared to controls)
Cycle anti-fungals for 4 days with *Saccharomyces boulardii* for 3 days
Hydrochloric acid and digestive enzyme support
Immune support – Vitamin C, Quercetin, Vitamins A & D, Zinc, Selenium and Rutin

Diet: strictly avoid all yeast- and mould-containing foods and refined sugars and carbohydrates, alcohol, vinegar and dairy products; follow a low carbohydrate diet; include garlic in cooking.

Other considerations:
Stress weakens the immune system & cortisol increases blood glucose.
Mix together *Saccharomyces boulardii* and *B. longum* BB536 with plain yoghurt or coconut oil and apply topically. Continue this treatment if still symptomatic; and reiterate importance of sugar free diet.

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Step 2:
Asymptomatic: reducing inflammation, gut healing and balancing microbiome for 6-8 weeks.
Candida overgrowth can damage the gastrointestinal mucosa, potentially leading to GI symptoms, nutrient malabsorption and increased absorption of antigenic macromolecules which can lead to ongoing allergic or autoimmune reactions in those who are susceptible. Metabolic products from candida organisms, along with other toxins can be absorbed across the gut epithelium into the systemic circulation (in those with significant intestinal permeability), leading to wide-ranging symptoms. Once asymptomatic, restore microbiome diversity, soothe GIT, reduce inflammation and improve bowel motility for 4-6 weeks. If still asymptomatic, go on to a maintenance program to maintain gap junctions; and GIT integrity and epithelial barrier function; reduce pathogenic load and promote the growth of beneficial bacteria.

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1 2 3

Intestinal permeability test

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Clinical Considerations:
Phase 1: Gut Repair (pages 12-13)

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Investigate:
Other stool analysis markers and differential diagnosis. ***Refer to Adverse Food Reactions Chart (pages 4-7).***

H. pylori urea breath test

H.pylori

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1 2 3

Clinical Considerations:
Phase 1: Gut Repair
NAC; Vitamin C; Omega-3 fatty acids
Once asymptomatic, proceed to ***Phase 2 & 3: Gut Repair, Function & Maintenance (pages 12-13)***

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Investigate:
Other stool analysis markers and differential diagnosis. ***Refer to Adverse Food Reactions Chart (pages 4-7).***

Hydrogen and methane breath test for SIBO

Wait at least one month after colonoscopy, barium studies, colonics and antibiotic use before testing. One week before testing stop probiotics, probiotic foods and herbal antimicrobials. One day before testing stop laxatives, digestive enzymes & hydrochloric acid supplements.

SIBO

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1 2 3

Clinical Considerations:

Step 1: 4-6 weeks
Phase 1: Gut Repair

Diet: remove fermentable starches and fibres to starve bacteria of fuel so they begin to die-off. Consider digestive support with enzymes and hydrochloric acid supplementation.

Step 2: 4-6 weeks
Continue Phase 1: Gut Repair

Antimicrobials; N-acetylcysteine for biofilm eradication after 2-3 weeks of antimicrobial therapy to reduce potential heavy die-off; bowel motility support with 5-HTP, ginger and acetyl-l-carnitine, B vitamins including choline; continue digestive support with enzymes and hydrochloric acid supplementation.

Diet: Re-introduce some starches like rice and quinoa

During both steps, you may consider removing foods high in histamines such as fermented foods, spinach, aged meats, bone broth that has been cooked for hours, tinned fish and meats; oxalates such as almonds, spinach, quinoa and cacao; and salicylates such as nightshades, zucchini, berries, avocado and coconut and olive oils.

Depending on symptoms after Step 2, the patient will either go back to Step 1 or if asymptomatic, start slowly reintroducing their normal diet.

Move on to ***Phase 2 & 3: Gut Repair, Function & Maintenance (pages 12-13)***

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Investigate:
Other stool analysis markers and differential diagnosis. ***Refer to Adverse Food Reactions Chart (pages 4-7).***

GIT Infection

Presentation 2 or 3

Stool analysis

Pathology-driven differential diagnosis of GIT infection

Investigate: Other important markers in Stool Analysis

Muscle fibres

Incomplete protein digestion; could indicate hypochlorhydria or achlorhydria; low levels of pancreatic enzymes or high meat consumption.

1 2 3

Clinical Considerations:
Ensure appropriate meat consumption; hydrochloric acid supplement; digestive enzyme support.

Phase 1-3: Gut Repair, Function & Maintenance (pages 12-13)

Vegetable fibres

Possible amylase deficiency. Inadequate chewing or eating quickly

Clinical Considerations:
Hydrochloric acid supplement; digestive enzyme support.
Talk to the patient about mindful eating.

Butyrate & total SCFAs

Reflect amounts of beneficial flora and fibre intake

Butyrate range
10.8 - 33.5

High

- Bacterial overgrowth
- High soluble fibre intake

Clinical Considerations:
Phase 1: Gut Repair (page 12)
Dietary fibre only (withhold supplemental fermentable fibre until inflammation &/or dysbiotic overgrowth have resolved);
Multistrain probiotic;
Omega-3 fatty acids.

Low

- Inadequate colonic function
- Low beneficial flora
- Inadequate fibre intake
- Can lead to obesity & decreased insulin sensitivity

Clinical Considerations:
Phase 2: Gut Repair & Function (page 12)
Reduce inflammation; soothe the GIT mucous membrane; introduce fermentable fibres to restore microbiota balance & diversity via increased butyrate production & improve bowel function & motility.

Fat stain

Fat malabsorption and steatorrhoea Pancreatic insufficiency leading to lipase insufficiency for normal lipid hydrolysis; mostly due to pancreatic inflammation.

Clinical Considerations:
Supplement with lipase-containing digestive enzymes
Bile salt deficiency – assess liver function (CYP450 inhibition); ensure phospholipids/cholesterol in a 10:1 ratio; adequate taurine and glycine; healthy gut microbiome; SNS/PSNS balance; consider circadian rhythm as bile acid synthesis occurs during the wake cycle during the day; check methylation and protein intake.

Malabsorption due to compromised intestinal mucosa, small intestinal disease, surgery, or medications.

1 2 3

Clinical Considerations:
Phase 1-3: Gut Repair, Function & Maintenance (page 12-13)

Lactoferrin

Marker for GIT inflammation
Elevated in IBD but not IBS

Calprotectin

Marker for GIT Inflammation.
Elevated in IBD but not IBS

Lysozyme

Moderate elevations are associated with yeast infections and dysbiosis; marked elevations are indicative of IBD

1 2 3

Clinical Considerations:
Phase 1-3: Gut Repair, Function & Maintenance (page 12-13)

White Blood Cells & Mucous

Both can be present in bacterial and parasitic infections, in cases of mucosal irritation and IBD.

Secretory IgA (sIgA)

Elevated levels are consistent with increased immune response relating to GI infections.

Clinical Considerations:
Refer to Infection Chart (previous page)

IBD

Refer: If markers point towards IBD, refer to GP for diagnosis & co-management.

1

Clinical Considerations:
Main goals: reduce symptoms and improve immune regulation for sustained results.

Phase 1: Gut Repair
Prevent malnutrition – common deficiencies are Iron, Folate, B12 and fat-soluble vitamins due to loss of appetite, bleeding from mucosal lesions, impaired absorption, inflammation, medications and surgery. Zinc, Selenium and Magnesium deficiencies due to diarrhoea and low dietary intake.

Note: BMI is not a reliable measure of nutritional status.

For Ulcerative colitis (UC) and Crohn's Disease (CD), avoid the stabiliser carrageenan which is present in foods such as ice-cream, cottage cheese and milk chocolate.

Avoid maltodextrin and emulsifiers as they are involved in disease development. Emulsifiers disrupt the intestinal mucous layer, increase intestinal permeability and promote bacterial translocation across the epithelium. Maltodextrin promotes *E.coli* biofilms and adhesion to epithelial cells and macrophages.

Increase intake of filtered water as chlorinated water can irritate the colon. If the medication sulfasalazine is being taken, consider B12 and Folate injections. Recommend 5-6 small meals daily. Cabbage juice contains substance U for ulcer healing.

Include fruit, vegetables and fish and avoid processed foods, red meat, sugar and alcohol. Increase protein intake as requirements are higher in active disease state. Monitor closely.

Caloric restriction reduces inflammation and improves immune response: Th17, Th1 and mTOR, increases Treg, autophagy, increases *Faecalbacterium prausnitzii* and *Akkermansia muciniphilia*.

Ulcerative colitis

1

Phase 1: Gut Repair (page 12), including Zinc carnosine to aid wound healing for up to 12 weeks; supplement with *Lactobacillus rhamnosus* GG (ATCC 53103) and *Bifidobacterium longum* BB536.

Nutritional supplementation should begin before elective surgery.

Check Iron levels and consider supplementation as appropriate (Anaemia is one of the most important features of IBD. Prevalence in adult IBD patients is up to 50%).

Magnesium is often deficient due to diarrhoea, consider supplementation as appropriate

N-acetylcysteine - a glutathione precursor, attenuates disease progression.

Vitamin D - supplement if deficient.

2 3

Phase 2 & 3: Gut Repair, Function & Maintenance (pages 12-13)

Crohn's Disease

1

Phase 1: Gut Repair (page 12)

Consider the following deficiencies common in Crohn's Disease: Iron, B1, B2, B12, Zinc, Magnesium, Potassium, Folate (if taking Methotrexate), Vitamins A, C, D, E and K.
Suggest a medication review with the GP.
Consider digestive enzymes and hydrochloric acid support with every meal; digestive enzymes in between meals help to break up clots.

Vitamin D - supplement if deficient.

2 3

Phase 2 & 3: Gut Repair, Function & Maintenance (pages 12-13)

Omega-3 fatty acids

Repair Function Maintenance

Phase 1: Repair

Week 1 - 4

1. Remove reactive foods/food groups
2. Promote gut repair by improving GIT integrity & epithelial barrier & gap junction function: Glutamine, Turmeric, Quercetin, Zinc carnosine, Vitamins A & D
3. Reduce inflammation: Turmeric, Quercetin, Glutamine, Vitamins A & D, Zinc carnosine, Omega-3 fatty acids
4. Support immune function, stabilise mast cells & prevent histamine release: Quercetin, Zinc, Vitamins A & D
5. Reduce pathogenic load & support microbiome diversity: Quercetin, Turmeric (Quercetin & Turmeric act as non-fermentable prebiotics), Glutamine, Vitamins A & D & multi-strain probiotics
6. Withhold fermentable fibre until inflammation & dysbiotic overgrowth have resolved
7. Support digestion & nutrient assimilation: hydrochloric acid & digestive enzymes

Assess symptoms at weeks 2 & 4*

1. Continue Phase 1 Gut Repair for another 2-4 weeks & then reassess (Phase 1 Gut Repair may require 12 or more weeks depending on the severity of the case & compliance)
2. Consider further investigations (other immune or non-immune mediated reactions, CDSA to assess microbiology & key markers of digestion, absorption & inflammation – ***Refer to infection Chart (pages 7-10***)
3. Consider cross-reactive foods
4. Address lifestyle factors such as stress & exercise that impact greatly on gut health
5. Other contributing factors such as alcohol consumption & medications: eg. NSAIDs, antibiotics & PPIs

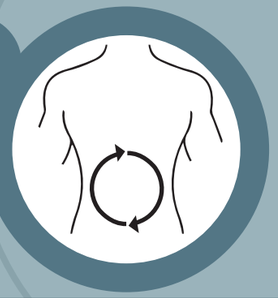
Phase 2: Repair & Function

Week 5 - 8

1. Introduce fermentable fibre: FOS, Psyllium, Slippery elm
2. Continue reducing inflammation: Turmeric, FOS, Licorice, Omega-3 fatty acids
3. Soothe the GIT mucous membranes: Slippery elm, Marshmallow, Licorice
4. Reduce pathogenic load: FOS, Marshmallow, Licorice, Psyllium
5. Restore microbiome balance & diversity: FOS, Marshmallow, Licorice, Psyllium, Slippery elm, Turmeric
6. Improve bowel function & motility: FOS, Psyllium, Lemon balm
7. Address the gut-brain axis & relieve anxiety: Psyllium, FOS, Lemon balm, Turmeric
8. Support detoxification: Turmeric, Licorice, Psyllium, B vitamins, Antioxidants, Sulphur-containing amino acids

Assess symptoms at weeks 6 & 8*

1. Continue Phase 2 Gut Repair & Function for another 2-4 weeks & then reassess (Phase 2 Gut Repair may require 12 or more weeks depending on the severity of the case & compliance)
2. Consider further investigations - other immune or non-immune mediated reactions, CDSA to assess microbiology & key markers of digestion, absorption & inflammation – ***Refer to infection Chart (pages 7-10***)
3. Consider cross-reactive foods
4. Address lifestyle factors such as stress & exercise that impact greatly on gut health
5. Other contributing factors such as alcohol consumption & medications: eg. NSAIDs, antibiotics & PPIs
6. Refer for a second opinion if required



Phase 3: Repair & Maintenance

Week 9 - 12

1. Continue reducing inflammation: Zinc carnosine, *S.boulardii*, Omega-3 fatty acids
2. Maintain gap junctions, GIT integrity & epithelial barrier function: Zinc Carnosine
3. Reduce pathogenic load: *S. boulardii*
4. Promote the growth of beneficial bacteria: *S. boulardii*
5. Promote microbiome diversity: *S. boulardii*
6. Prevent infection if travelling overseas: *S. boulardii*
7. Improve *H.pylori* eradication rates: Zinc carnosine, *S. boulardii*
8. Reduce negative effects associated with food reactions: Zinc carnosine, *S. boulardii*

Assess symptoms at week 12*

1. Continue Phase 3 Gut Repair & Function for another 2-4 weeks and then reassess (Phase 3 Gut Repair may require 12 or more weeks depending on the severity of the case & compliance)
2. Consider further investigations - other immune or non-immune mediated reactions, CDSA to assess microbiology & key markers of digestion, absorption & inflammation – ***Refer to infection Chart (pages 7-10)***
3. Consider cross-reactive foods
4. Address lifestyle factors such as stress & exercise that impact greatly on gut health
5. Other contributing factors such as alcohol consumption & medications: eg. NSAIDs, antibiotics & PPIs
6. Refer for a second opinion if required

Maintenance

Week 12+

1. Continue Phase 3 Repair & Maintenance until symptoms subside & then consider reintroducing eliminated foods (if possible) one-by-one into the diet from least reactive to most reactive, while staying on the Phase 3 supportive nutrients
   - If an adverse reaction occurs, consider eliminating this food for a longer period, continue Phase 3 Repair & Maintenance & attempt reintroduction again later in the healing process
2. Encourage an ongoing diverse, whole food diet including FOS & GOS-containing foods to enhance the foundations of the core microbiome & build native populations
3. Address lifestyle factors such as stress & exercise that impact greatly on gut health

*Each patient will have individual features that affect their time to recovery & may require more or less time to optimise gut healing. Let the patient's symptoms & feedback guide you through this process timeframe.

Refer to the Nutrient & Dosage Chart page 14-17 *for the recommeded dosages and treatment duration as outlined in research.*

Marked improvement

Little or no improvement

Researched Nutrient & Dosage Chart

Nutrient/ Herb	Clinically Researched Dose	Rationale/Research	Action
***B. longum* BB536**	112 - 427 million CFU daily	• *Bacteroides fragilis* - decreased pathogen at week 8 in treatment group, with no effects on controls.[1]	Antimicrobial
Betaine hydrochloride; Glutamic acid; Pepsin		• Supplemental HCl (available as Betaine HCl and often combined with Glutamic Acid), has been shown to significantly reduce stomach pH in induced hypochlorhydria.[2] • Pepsin is the predominant proteolytic enzyme in the stomach. It facilitates the breakdown of protein in the diet, and requires HCl for its activation. To function optimally, pepsin requires a pH between 1.8 and 3.5.[3] Its activity decreases when the pH reaches 5 or above.[4,3,5] In hypochlorhydric states, activation of endogenous pepsinogen into pepsin is stalled, consequently impairing protein digestion. However, as pepsinogen is activated by HCl or a combination of HCl and preformed pepsin, supplementation is able to correct this imbalance.[3]	Improve hydrochloric acid production and digestive capacity
Digestive enzymes: protease, bromelains, trypsin		• Trypsin is part of a family of protease enzymes that fragment proteins. Unlike pancreatic enzymes, bromelain has a relatively broad pH range through which it can remain effective, providing proteolytic activity in the stomach as well as in the intestine.[6] Bromelain may therefore offer additional support for protein digestion when combined with pancreatin.	Digestive support
Fructooligosaccharides FOS (Crohn's Disease)	2g+ daily	• Increases *Bifidobacterium* spp., *Lactobacillus* spp., and *Faecalbacterium prausnitzii*. Increases SCFAs.[7] • Reduces TNFα, IL-6, IL-8, IL-1β. Increases IL-10 producing dendritic cells. Reduces faecal calprotectin.[7]	Microbiome balance; anti-inflammatory
FOS	2.5g+ daily	• **Restores microbiome diversity** - nourishes *Faecalibacterium* and *Bifidobacterium* populations. Promotes butyrate production. Increases beneficial bacteria and reduces non-beneficial bacteria.[8] • **Improves bowel motility** - selectively stimulates the growth of *Bifidobacteria*, improves stool consistency and bowel function.[9,8] • **Anti-inflammatory** - improves gut barrier function, increases beneficial bacteria & reduces non-beneficial bacteria.[8] • **Anxiety** - increased faecal bifidobacterial concentrations with a subsequent improvement in anxiety.[9]	Microbiome balance; bowel function; anti-inflammatory; anxiolytic
Glutamine	10-15g for 8 weeks	• Major energy source for epithelial cells • Role in mucosal integrity • Depleted by infection • **Clinical trial:** 5g Glutamine TDS for 8 weeks restored intestinal permeability in post infectious IBS patients.[10] • Improves epithelial integrity, downregulates NF-kB, preserves gut barrier function, maintains tight gap junctions, and provides fuel for enterocytes.[10]	Protects gap junctions, gut healing, antimicrobial, improves microbiota
***Glycyrrhiza glabra* (Licorice)**		• **Restores microbiome diversity** - stimulates *Lactobacilli* and *Bifidobacterium* spp. Increases butyrate which reduces non-beneficial bacteria and favours mucin production.[11] • **Soothes the GIT** - soothes mucous membranes. Provides demulcent and anti-ulcerogenic actions.[12,13,14] • **Reduces inflammation** - provides antimicrobial, anti-ulcerogenic, anti-spasmodic and anti-inflammatory actions. Supports detoxification, providing antimicrobial, antioxidant and hepatoprotective actions.[12,13]	Microbiome balance; demulcent; anti-inflammatory; antimicrobial; anti-ulcerogenic; hepatoprotective
***Althea officinalis* (Marshmallow)**		• **Restores microbiome diversity** - promotes healthy bowel flora. Provides anti-inflammatory and antimicrobial actions.[15-17] • **Soothes the GIT** - relieves inflamed mucous membranes. Provides vulnerary and demulcent actions.[11,16,17]	Microbiome balance; demulcent; vulnerary
***Melissa officinalis* (Lemon balm)**		• **Regulates bowel motility** - provides carminative and antispasmodic actions.[18,19,20] • **Relieves anxiety -** improves functional gastrointestinal complaints associated with anxiety. Anxiolytic actions.[18,19]	Carminitive; antispasmodic; anxiolytic; antibacterial to *E.coli*
N-acetylcysteine		• Impairs *Staphylococcus aureus* biofilm formation without inducing cytotoxic effects[21]; NAC has good antibacterial properties and interferes with biofilm formation.[22]	Antimicrobial; detoxification support
N-acetylcysteine *(H.pylori)*	400mg three times daily + Clarithromycin & Lansoprazole	• NAC had an additive effect on eradication rates of *H.pylori* with medications; NAC may have improved the delivery of antibiotics to the infection site by reducing mucous thickness.[23]	Antimicrobial
Omega-3 fatty acids (IBD & *H.pylori*)		• Reduce inflammation in IBD[24] • Omega-3 PUFAs could reduce various *H. pylori*-associated gastric diseases, including gastric cancer by influencing multiple targets, including proliferation, survival, angiogenesis, inflammation, and metastasis.[25]	Anti-inflammatory; prebiotic
Omega-3 fatty acids (IBS, Functional GIT disorders, Coeliac Disease)		• Omega-3 PUFAs exert significant effects on the intestinal environment; on mood and cognitive functioning, such as anxiety and depression; and modulating the gut microbiota composition.[26] Omega-3 PUFAs can be considered prebiotics.	Prebiotic
Probiotics (IBD)		• Increase SCFAs, mucous secretion, IL-10; decrease TNFα, NF-kB, IL-1β, IL-β, TLR expression; enhance epithelial barrier function.[7]	Anti-inflammatory; improve gut barrier function
Psyllium		• **Restores microbiome diversity -** increases butyrate production. Shapes a desirable microbiome by correcting disturbed bowel flora.[27] • **Improves bowel motility** – softens the stool, reduces discomfort and straining.[27,28] • **Relieves anxiety** - improves bowel symptoms, microbiome diversity and quality of life parameters, including anxiety, physical discomfort and straining.[28] • **Supports detoxification** - binds bile acids which causes increased bile acid excretion. Provides mucilage & polysaccharides and normalises stool consistency and transit time.[27,29]	Microbiome balance; bowel function & motility; detoxification support; anxiolytic
Quercetin	500mg daily	• Inhibits histamine release[30] • Protects tight junction barrier function by regulating tight junction proteins and inflammatory signalling pathways.[31] • Gut microbial composition can be modulated by polyphenols.[32] • Quercetin has been demonstrated to prevent intestinal permeability in response to toxic agents, the rise in the levels of proinflammatory cytokines and pathologically provoked intestinal motility.[33] • NSAID-induced mitochondrial dysfunction[33] • **Meta-analysis:** Showed a significant reduction in circulating CRP at doses of 500mg daily.[34] • **Clinical trial:** Study of rheumatoid arthritis patients found 500mg of Quercetin reduced pain, stiffness and inflammatory markers including TNFα.[35] • Quercetin produced by microbial transformation demonstrated antimicrobial activity against *S. aureus* by effectively inhibiting the growth and dispersion of preformed biofilms[36]; induces apoptosis by modulating quorum sensing.[33]	Anti-inflammatory; antimicrobial; improves microbiota; non-fermentable prebiotic; protects gap junctions; gut healing

Researched Nutrient & Dosage Chart

Nutrient/ Herb	Clinically Researched Dose	Rationale/Research	Action
Saccharomyces boulardii	5-10billion CFU daily	• *Blastocystis hominis* - 77.7% of treatment group achieved a clinical cure at day 15; after one month clinical cure rate was 94.4%.[37] • *Candida albicans* - as effective as nystatin (anti-fungal) at reducing fungal infection, progressive fungal colonisation & incidences of clinical sepsis.[38] • *Clostridium difficile* - stool analysis showed those with lower *S. boulardii* after treatment had higher clostridium recurrence.[39] • *Giardia lamblia* - in combination with antibiotic therapy, led to reduced pathogen numbers.[40] • *H. pylori* - in children the eradication rate was 93.3% in treatment group with 30% reduction in side effects[41]; in adults it reduced side effects of triple therapy.[42]	Antimicrobial; microbiome balance
Ulmus rubra **(Slippery elm)**		• **Restores microbiome diversity** - provides polysaccharides and mucilage. Increases butyrate-producing bacteria.[11] • **Soothes the GIT** - provides mucilage to protect the mucosal barrier function in the GIT.[11,43]	Microbiome balance; demulcent; vulnerary; laxative
Thyme, Clove and Oregano oils, with the herbs Black walnut, Wormwood, Pau d'arco, Barberry and Garlic		See Bio Concepts' *Gastrointestinal Diverse Clinical Applications Chart*	Antimicrobial
Curcuma longa **(Turmeric)**		• **Restores microbiome diversity** - provides polyphenols for positive effects on gastrointestinal microbiota modulation and neuroendocrine system health (gut-brain axis). [32,44] • **Reduces inflammation -** reduces multiple markers of gastrointestinal inflammation such as NF-κB and TNF-α. Improves gut barrier function. Increases Intestinal Alkaline Phosphatase (IAP) and helps reduce the impact of endotoxins.[45,46,47] • **Supports detoxification** - supports detoxification by promoting bile flow and glucuronidation. Provides hepatoprotective and antioxidant actions.[45,48,49,50,51] • **Strengthens intestinal barrier function** - improves intestinal barrier function and reduces bacterial translocation.[52] • **Antimicrobial** - influences the function of immune cells involved in IBD, inhibits TLR4 and NF-kB and AP-1 signal transduction.[52]	Microbiome diversity; anti-inflammatory; supports detoxification; anti-microbial; non-fermentable prebiotic
Vitamin A		• Promotes epithelial cell proliferation and healing and increases mucous production from goblet cells. • Required for the development of immune tolerance in the intestine.[53] • Increases the abundance of *Lactobacillus* spp. in the gut • Regulates the expression of tight junction genes in epithelial cells and restores tight junctions.[54] • Strengthens the barrier function of epithelial cells in the absence of probiotic bacteria.	Promotes healthy cell turnover; vulnerary; microbiota balance; regulates gene expression; repairs gap junctions
Vitamin C		• The inhibitory effect of Vitamin C on biofilm formation proceeds by inhibition of quorum sensing and other stationary phase regulatory mechanisms underpinning biofilm development which specifically leads to inhibition of polysaccharide biosynthesis.[55]	Biofilm inhibiting
Vitamin D	1000IU-2000IU for 12 weeks	• Role in maintaining the integrity of the intestinal barrier. • Enhances the tight junctions that control mucosal permeability. • Deficiency leads to disruption of the gut barrier and dysbiosis; and low *Akkermansia municiphila* which is associated with ulcerative colitis.[56] • D3 supplementation diminishes the competitive advantage of opportunistic pathogens.[56] • **Clinical trial:** Patients with ulcerative colitis were given 1000IU-2000IU daily for 12 weeks. Both doses improved patients' quality of life and level of disease activity reduced in the 2000IU group only.[57]	Anti-inflammatory; improves microbiota; protects gap junctions; gut healing
Vitamin D (IBD)		• Exerts protective mechanisms through modulation of gut commensal bacteria, reducing inflammation and immunomodulatory actions[58] (reduces Th1, Th17, dendritic cell maturation, enhances epithelium barrier function and autophagy, increases IL-10, and reduces IFN-y, IL-17, IL-12).[58]	Anti-inflammatory; microbiome balance; improves gut barrier function
Zinc carnosine (ZnC)	75mg- 150mg daily for 4 weeks	• ZnC dissociates slowly in gastric secretions. This prolonged existence in the stomach maintains the healing effect for a longer time.[59] • Approved as an anti-ulcer drug; adheres to and penetrates ulcers which then undergo wound healing.[59] • **Clinical trial:** ZnC at 37.5mg BD given alongside standard doses of NSAIDs demonstrated no significant increase in intestinal permeability.[60] • **Clinical trial:** Patients taking daily low-dose aspirin were given 150mg ZnC for 4 weeks and showed a significant reduction in the median number of reddened lesions and erosions/ulcers on follow up capsule endoscopy.[61] • **Clinical trial:** Patients taking 74mg BD ZnC alongside triple therapy for *H.pylori* demonstrated significantly higher eradication (77.0%) compared to triple therapy alone (48.6%) with no serious adverse events.[62]	Protects gap junctions; anti-ulcerogenic; gut healing

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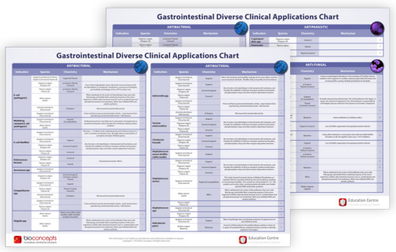
Related Resources

Elimination Diet Handout

Diet and Symptom Diary Notepad

Food and Lifestyle Diary Notepad



Gastrointestinal Diverse Clinical Applications Chart

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