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Management of Small Intestinal Bacterial Overgrowth in Adult Patients

ABSTRACT

The human gastrointestinal tract is a complex system of digestive pathways aided by mechanical processes, enzymes, transport molecules, and colonic bacteria. Occasionally, these bacterial components transplant to atypical locations due to various gastrointestinal imbalances or anatomical structural issues. This may lead to bacterial overgrowth of the small intestine, where minimal or no bacteria are normally found. Symptoms of small intestinal bacterial overgrowth may mimic those of various functional gastrointestinal diseases. Small intestinal bacterial overgrowth is typically diagnosed through hydrogen breath tests or jejunal aspirate culture. Current recommendations indicate antibiotics as the first-line treatment to eradicate or modify the bacterial overgrowth to a more favorable state. Nutritional support is also indicated to correct deficiencies and aid in symptom alleviation. As small intestinal bacterial overgrowth is common in other conditions, much of the research for this area is based on findings in codisease states rather than independent disease research. To provide accurate recommendations for small intestinal bacterial overgrowth, more targeted research is needed.

The human gastrointestinal tract includes yeasts, parasites, viruses, and bacteria that collectively contribute to the normal function of the gastrointestinal tract and, sometimes, to the pathogenesis of diseases in the intestines (Gasbarrini et al., 2007). Normal gastric acid pH, small intestinal motility, pancreatic enzymes, and the ileocecal valve all aid in prevention of bacteria growth throughout the small intestine (Gabrielli et al., 2009). In a healthy, adult patient, the small intestines are typically devoid of coliform bacteria or the number found is minimal (Bohm, Siwiec, & Wo, 2013). Normal concentrations for gut bacteria were found to be as follows: jejunum 10^{3-4} cfu/ml (colony-forming units per milliliter), terminal ileum 10^{7-9} cfu/ml, and colon 10^{10-12} cfu/ml aspirate, with 400–500 bacterial species represented (Quigley & Quera, 2006). The actual bacterial makeup of the small intestine in a healthy human is widely unknown at this point, but recent advances in sampling techniques may lead to additional information in the future (Dave et al., 2011; Quigley, 2014; Quintanilha et al., 2007; Zilberstein et al., 2007).

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Background

Within the oropharynx region, lactobacilli, enterococci, and oral streptococci are common (Quigley & Abu-Shanab, 2010). Streptococci, staphylococci, enterococci, lactobacilli, and corynebacteria are commonly found in the stomach and proximal small intestine (Bohm et al., 2013). The colon is the most densely populated segment of the bowel in relation to bacteria, with Firmicutes and Bacteroidetes dominating followed by Proteobacteria and Actinobacteria (Bäckhed et al., 2012; Gill et al., 2006). The colon may also include *Porphyromonas*, bifidobacteria, lactobacilli, and clostridia (Quigley & Abu-Shanab, 2010). The types and amounts of bacteria that are found within the intestinal tract are highly dependent on individual diet; those who consume a high plant-based diet will have more *Prevotella* bacteria than Bacteroidetes (Jeffery & O'toole, 2013).

Colonic bacteria are responsible for synthesis of B vitamins including folate and biotin. Vitamin K is also produced in the colon. In addition, undigested resistant starch and oligosaccharides are converted in the large intestine by bacteria to short-chain fatty acids, which are used to then produce acetate, propionate, and butyrate (Jeffery & O'toole, 2013). These short-chain fatty acids play a role in reducing intestinal pH to inhibit growth of pathogenic microorganisms and to increase the absorption rate of certain nutrients (Macfarlane & Macfarlane, 2012).

Butyrate is mainly used by colonocytes for energy, and the rest passes through the portal vein into the liver to be used in lipid biosynthesis along with a small portion of acetate (den Besten et al., 2013). Acetate is also metabolized in the citric acid cycle (Skutches, Sigler, Teehan, Cooper, & Reichard, 1983). Propionate circulates to the liver for use in gluconeogenesis (den Besten et al., 2013). Therefore, colonic microbiome contributes to health and metabolism of humans.

Bacteria have also been shown to play a role within the nervous system. There is evidence that the bacteria of the colon have a bidirectional communication role between the enteric peripheral nervous system and the central nervous system (Cryan & Dinan, 2012), which helps modulate intestinal immunity, sensitivity, motility, and secretion (Distrutti, Monaldi, Ricci, & Fiorucci, 2016). Humans can respond to bacterial communications through epithelial receptor-mediated signaling or direct stimulation of enteric neurons and immune cells. Conversely, through effects on intestinal motility, secretion, and immune function, the brain can exert influence over the enteric microbiota (Kelly et al., 2015), establishing a gut–brain axis connection. In addition, bacteria play a role in gut motility, as research in animals where the intestinal microbiome was altered to a germ-free environment led to profound alterations in gut motility that were reversed once typical intestinal microflora were reestablished (Caenepeel, Janssens, Vantrappen, Eyssen, & Coremans, 1989; Collins, Verdu, Denou, & Bercik, 2009; Distrutti et al., 2016).

Small Intestinal Bacterial Overgrowth

Despite the overriding presence of bacteria in the colon, the small intestine either is devoid of bacteria or has only a small bacterial content, usually of similar composition to the oropharynx. In a healthy adult, there are various mechanisms to prevent bacterial growth within the small bowel. The ileocecal valve prevents colonic bacteria from entering the small intestine, and gastric acid pH acts to prevent excessive oropharynx bacteria from passing into the duodenum (Bohm et al., 2013). Failure of these components may occur through anatomical anomalies such as fistulae and surgical alterations (Yang & Pimental, 2007).

Hypochlorhydria (Lewis, Potts, Malhotra, & Mountford, 1999; McEvoy, Dutton, & James, 1983), pernicious anemia (Parodi et al., 2009), presence of *Helicobacter pylori* bacteria, and long-term use of proton-pump inhibitors, through a related reduction in gastric acid pH, are also connected to bacterial overgrowth (Compare et al., 2010; Lombardo, Foti, Ruggia, & Chiecchio, 2010; Shindo, Machida, Koide, Fukumura, & Yamazaki, 1998). However, a retrospective study by Ratuapli et al. (2012) and a prospective study by Giamarellos-Bourboulis, Pyleris, Barbatzas, Pistiki, and Pimentel (2016) showed that there was no difference in bacterial overgrowth rates and use of proton-pump inhibitors.

Other contributing causes to bacterial overgrowth include impaired intestinal immunity (Parodi et al., 2009), altered small intestinal motility or clearance in Phase III contractions of the migrating motor complex (Gabrielli et al., 2009), conditions that cause disruptions to visceral enteric neurologic and muscular systems, alterations to small bowel anatomy such as duodenal or jejunal diverticula, strictures of small intestine, radiation, and inadequate pancreatic enzyme or bile acid secretions (Bohm et al., 2013; Goebel-Stengel et al., 2014; Quigley & Quera, 2006; Quigley & Abu-Shanab, 2010). In addition, whether as a direct consequence or as an epiphenomenon, bacterial overgrowth is connected to many gastrointestinal conditions including irritable bowel syndrome (IBS), inflammatory bowel disease, celiac disease, chronic pancreatitis, liver disease, scleroderma (Quigley & Abu-Shanab, 2010), and certain infectious diseases including Chagas and Lyme (Parodi et al., 2009). Recent research has proposed a definitive connection between IBS and small intestinal bacterial overgrowth (SIBO), despite past controversies concerning the connection (Giamarellos-Bourboulis et al., 2016). A synopsis of the contributing causes and linked conditions is presented in Table 1.

Small Intestinal Bacterial Overgrowth Diagnostic Testing

Diagnosis using jejunal aspirate culturing is the preferred method of testing (Goebel-Stengel et al., 2014), but because of the invasive nature of collecting the sample, possible contamination with oropharynx bacteria, low reproducibility, and the inability to culture all contents (Parodi et al., 2009; Quigley & Abu-Shanab, 2010), breath testing has been accepted as an alternate option. If an aspirate sample is available, a diagnosis of SIBO is typically made when the sample contains 10^5 cfu/ml of luminal fluid from proximal jejunum (Bohm et al., 2013; Parodi et al., 2009; Quigley & Abu-Shanab, 2010) or with lower numbers if colonic-type bacteria are found, or a bacterial species

TABLE 1. Contributing Causes of SIBO and Linked Conditions[a]

Contributing Causes of SIBO	Linked Conditions
Anatomic anomalies (fistulae or surgical alterations) Hypochlorhydria Pernicious anemia *H. pylori* bacteria Impaired intestinal immunity Altered motility Disruptions to visceral neurologic or muscular systems Diverticula Strictures Radiation treatment Inadequate pancreatic or bile enzyme secretions	IBS IBD Celiac disease Chronic pancreatitis Liver disease Scleroderma Chagas disease Lyme disease

Note. IBD = inflammatory bowel disease; IBS = irritable bowel syndrome; SIBO = small intestinal bacterial overgrowth.
[a]From Bohm et al. (2013), Compare et al. (2010), Gabrielli et al. (2009), Giamarellos-Bourboulis et al. (2016), Goebel-Stengel et al. (2014), Lewis et al. (1999), Lombardo et al. (2010), McEvoy et al. (1983), Parodi et al. (2009), Quigley and Quera (2006), Quigley and Abu-Shanab (2010), Shindo et al. (1998), and Yang and Pimental (2007).

that is not present in salivary or gastric fluids (Bauer et al., 2001; Gabrielli et al., 2009; Quigley & Abu-Shanab, 2010; Toskes, 1993).

The contaminating bacteria in SIBO often include *Streptococcus*, *Micrococcus*, *Escherichia*, *Staphylococcus* and *Klebsiella* species (Bouhnik et al., 1999). SIBO may be caused by bacterial overgrowth with bacteria from the upper respiratory tract, which is most linked with Gram-positive bacteria passing because of the failure of the gastric acid barrier. Overgrowth of colonic bacteria is most linked to Gram-negative bacteria and is typically associated with small bowel clearance impairment or alteration of gastrointestinal anatomy (Bohm et al., 2013; Parodi et al., 2009).

In the absence of jejunal aspirates, hydrogen breath testing may be used for evaluating the presence of bacterial overgrowth. The test is completed using glucose, lactulose, or d-xylose. Patients have a baseline hydrogen reading completed, then consume the chosen substrate, and complete subsequent hydrogen breath readings over a set time. Patients with an increase of hydrogen output of 20 ppm within 180 minutes or any increase over 90 minutes are considered to have a positive result indicative of SIBO. Some diagnostic standards require a double-peak pattern in hydrogen levels, showing an initial peak from SIBO fermentation and a second peak from colonic fermentation (Quigley & Abu-Shanab, 2010). The interpretation of breath tests for diagnosing SIBO is controversial because of low reproducibility and inconsistent results (Gibson & Barrett, 2010). Other researchers have presented the idea that an empiric 1-week trial with antibiotics with an improvement in symptoms is an alternate method for diagnosing SIBO (Bohm et al., 2013; Quigley & Quera, 2006).

SIBO Symptoms

Commonly accepted symptoms for SIBO include abdominal pain, bloating, and diarrhea (Banwell et al., 1981; Goebel-Stengel et al., 2014; Quigley, 2014). Weight loss (Parodi et al., 2009), abdominal distension, increased flatus, dehydration, fecal urgency, fecal incontinence, and nausea are also possible symptoms (Bohm et al., 2013). The symptoms of SIBO overlap with many other gastrointestinal disorders, making a symptom-based diagnosis complicated (Quigley & Abu-Shanab, 2010).

Nutritional Consequences

As the site of major nutrient uptake, alterations to the small bowel from bacteria lead to significant impairment of nutrient absorption among all macronutrients and some vitamins. Carbohydrate malabsorption occurs because of impaired activity of disaccharidases (Bohm et al., 2013), brush border hydrolases (Yang & Pimental, 2007), and monosaccharidases (Giannella, Rout, & Toskes, 1974; Sherman, Wesley, & Forstner, 1985) as well as early digestion by bacteria (Bohm et al., 2013). These malabsorbed carbohydrates form short-chain organic acids that may increase the osmolarity of intestinal fluid and contribute to diarrhea (Quigley & Quera, 2006). Some patients may show signs of lactose intolerance, but, in SIBO, this is thought to be a bacterial intolerance of lactose and not a deficiency in lactase enzyme (Pimentel, Kong, & Park, 2003; Vernia, Di Camillo, & Marinaro, 2001; Yang & Pimental, 2007).

Bacterial overgrowth also leads to deconjugation of bile acids within the proximal small bowel. These acids are then reabsorbed in the jejunum rather than in the ileum (Bohm et al., 2013), and this disruption of enterohepatic reabsorption, along with the deconjugation,

leads to impaired absorption of fats and fat-soluble vitamins A, D, E, and K (Bohm et al., 2013; Smith & Summa, 1999; Quigley & Quera, 2006). Furthermore, the deconjugated bile acids are toxic to intestinal mucosa, which may lead to additional malabsorption related to bile acid diarrhea (Bohm et al., 2013) or steatorrhea (Quigley & Quera, 2006).

Unconjugated bile acids, along with bacterial mucosal injury, also contribute to protein malabsorption through a decrease in amino acid uptake (Burke, Gracey, Thomas, & Malajczuk, 1975; Jones, Craigie, Tavill, Franglen, & Rosenoer, 1968; Strauss, 1961). In addition, inhibition of protein absorption is thought to occur through inactivation of pancreatic enzymes by bacterial proteases (Parodi et al., 2009). Vitamin B_{12} deficiency is also common in SIBO because of uptake and use by luminal bacteria (Bohm et al., 2013; Festen, 1991; Quigley & Quera, 2006; Saltzman & Russell, 1994).

Management

The management of SIBO is multifaceted and may require months of support or repeated treatment. At the present time, the recommended treatments are mainly empirical (Quigley, 2014) with minimal SIBO-specific research. The main components of treatment include correcting underlying causes (Bohm et al., 2013; Gabrielli et al., 2009; Gasbarrini et al., 2007; Quigley & Abu-Shanab, 2010), eradicating bacterial overgrowth, and treating nutritional deficiencies (Bohm et al., 2013; Quigley & Abu-Shanab, 2010). Surgical repair of intestinal fistulae, diverticula, blind loops, and strictures may be necessary to remove underlying causes (Bohm et al., 2013). In addition, prokinetic agents may be used to improve motility (Quigley & Quera, 2006), although this has yet to be shown to be fully effective (De Ponti & Tonini, 2001).

If applicable and possible, patients should also stop the use of narcotics to aid in improved motility (Bohm et al., 2013). To eradicate the overgrowth, or alter the growth for a more favorable pattern, nonabsorbable antibiotics, such as rifaximin or neomycin, are considered to be the main approach (Bohm et al., 2013; Corazza, Stefano, & Scarpignato, 2006; Dupont, 2014; Muir & Gibson, 2013). Because of the high rate of recurrence with SIBO, repeated courses of antibiotics or regular intake over months may be required with rotational use of various antibiotics to reduce likelihood of building resistance (Quigley & Abu-Shanab, 2010).

To treat nutritional deficiencies, patients may need supplementation with vitamin B_{12}, fat-soluble vitamins, pancreatic enzymes (Bohm et al., 2013), and calcium and magnesium (Quigley & Abu-Shanab, 2010). In addition, although the benefit in SIBO is unclear, a low-FODMAP diet may be recommended (Bohm et al., 2013; Gibson & Shepherd, 2012) to help alleviate any remaining symptoms including abdominal discomfort (Barrett & Gibson, 2012; Gibson & Shepherd, 2010), bloating, flatulence, pain, or diarrhea. A low-FODMAP diet reduces intestinal fermentation and symptom severity associated with gas production and abdominal distension (Barrett & Gibson, 2012).

FODMAP refers to fermentable oligosaccharides, disaccharides, monosaccharides, and polyols. These categories encompass galactooligosaccharides, fructans, lactose, fructose, sorbitol, and mannitol. Foods high in these components, shown in Table 2, are poorly absorbed and may contribute to abdominal distension, gas, and flatulence (Barrett & Gibson, 2012). Malabsorption of FODMAPs occurs because of a low-capacity fructose transport mechanism, lack of hydrolases for fructans and galactans, and no polyol-specific transporters along with an inability of these molecules to be absorbed through passive diffusion (Jeffery & O'toole, 2013). Improvements in symptoms may be related to a reduction in fermentation among small bowel bacteria or from a modulation of microbiota composition (Bohm et al., 2013).

Studies have shown a progressive efficacy of symptom reduction with a low-FODMAP diet over 6 weeks (Fedewa & Rao, 2014). Following that time, patients should reintroduce foods from individual categories to see whether the consumption of these foods renews symptoms (Barrett & Gibson, 2012). Maintaining a restrictive diet for this amount of time may be difficult for patients (Muir & Gibson, 2013), and without proper dietetic support, some patients may not be compliant or may unduly restrict their intake. As the diet is very restrictive and the nutritional adequacy is suspect (Barrett & Gibson, 2007; Fedewa & Rao, 2014), patients should be under the care of a dietetics expert (Foxx-Orenstein, 2016). Support from a dietitian with experience in a low-FODMAP diet has shown increased compliance (Barrett & Gibson, 2012), and the diet appears to be safe if implemented with professional dietetic support (Barrett & Gibson, 2007).

Additional recommendations may include probiotics (Gabrielli et al., 2009), but some researchers disagree with this recommendation because of a lack of research (Quigley & Abu-Shanab, 2010). Currently, there are insufficient data concerning the preferred strain, dose, monitoring mechanisms, and delivery methods for the use of probiotics in SIBO (Shanahan, 2003).

SIBO IBS

Although the connection between SIBO and IBS is controversial, there is significant symptom overlap (Lacy, 2015) and SIBO has been cited as an underlying issue for at least a subset of patients with IBS (Barbara et al.,

TABLE 2. FODMAP Dietary Components and Alternate Food Options[a]

FODMAP	Dietary Form	Foods High in FODMAP Component	Alternatives
Monosaccharide	Fructose	*Fruits:* apples, pears, nashi pears, clingstone peaches, mango, sugar snap peas, watermelon, cherries, canned fruit in natural juice *Honey* *Vegetables:* asparagus, artichokes, sugar snap peas	*Fruits:* banana, blueberry, cantaloupe, carambola, durian, grape, grapefruit, honeydew melon, kiwi, lemon, lime, orange, passion fruit, pawpaw, raspberry, strawberry, tangelo *Honey:* golden syrup, maple syrup *Sweeteners:* any except polyols or honey
Disaccharides	Lactose	*Milk:* cow, goat, and sheep *Ice-cream* *Custards* *Yogurt* *Cheeses:* soft and fresh *Butter*	*Milk:* lactose-free milk, rice milk *Ice-cream:* gelato, sorbet *Yogurts:* lactose-free yogurts *Cheeses:* hard cheeses
Oligosaccharides	Fructans and/or galactans	*Fruits:* watermelon, custard apple, white peaches, rambutan, persimmon *Vegetables:* artichokes, asparagus, beetroot, Brussels sprouts, broccoli, cabbage, fennel, garlic, leeks, okra, onions, peas, shallots, soy products (tofu, soy milk, miso, veggie-burgers) *Cereals:* wheat and rye in large amounts, couscous *Nuts and seeds:* pistachios *Legumes:* chickpeas, lentils, red kidney beans, baked beans *Inulin or chickory root* *Coffee*—more than one cup per day	*Fruits:* unsweetened cranberries, strawberries, cantaloupe, lemon, lime, tomato *Vegetables:* bamboo shoot, bok choy, capsicum, carrot, celery, chives, choko, choy sum, corn, eggplant, green bean, lettuce, parsnip, silverbeet, spring onion (green part only) *Onion/garlic substitute:* garlic-infused oil *Cereals:* gluten-free and spelt bread/cereal products
Polyols	Sorbitol, mannitol, maltitol, xylitol, erythritol, polydextrose, and isomalt	*Fruits:* apples, apricots, cherries, lychee, nashi pears, nectarines, pears, peaches, plums, prunes, watermelon *Vegetables:* avocado, cauliflower, mushrooms, snow peas *Sweeteners:* sorbitol, mannitol, xylitol, maltitol, isomalt, and others ending in –ol *Laxatives* *Sugar-free mints/gums*	*Fruits:* banana, blueberry, cantaloupe, carambola, durian, grape, grapefruit, honeydew melon, kiwi, lemon, lime, orange, passion fruit, pawpaw, raspberry *Sweeteners:* glucose, sucrose, other artificial sweeteners not ending in –ol

Note. FODMAP = fermentable oligosaccharides, disaccharides, monosaccharides and polyols.
[a]From Barrett and Gibson (2012), Fedewa and Rao (2014), Magge and Lembo (2012), and Mansueto, Seidita, D'Alcamo, and Carroccio (2015).

2016; Barrett & Gibson, 2012; Foley, Burgell, Barrett, & Gibson, 2014; Foxx-Orenstein, 2016; Spiegel, 2011) or as a treatable factor for some patients with IBS (Bohm et al., 2013). It has been argued that the two conditions are separate and distinct (Saadi & Mccallum, 2013) because of inconclusive data (Ford, Talley, Spiegel, & Moayyedi, 2009; Posserud, Stotzer, Bjornsson, Abrahamsson, & Simren, 2007; Yu, Cheeseman, & Vanner, 2010). As previously cited, a 2016 study presents findings that connect the disease states (Giamarellos-Bourboulis et al., 2016) but the connection is dependent on a clear definition for SIBO with standards for testing preferences, requirements for diagnosis, and clear symptomatic expectations.

Currently, treatments to eradicate bacteria in SIBO have been shown to improve IBS symptoms (Pimentel, Chow, & Lin, 2000). Despite the disputed connection, many of the recommendations made for the treatment of SIBO are based on symptomatic improvement in patients with IBS. More research with SIBO-specific populations is necessary to clearly delineate and define the two conditions and associated treatments. In addition, a clear clinical definition for SIBO is necessary to differentiate associated diseases (Quigley, 2014).

Conclusion

There are many recommendations for the management of SIBO. These recommendations include altering the bacterial makeup, treating nutritional deficiencies, and managing gastrointestinal symptoms. Many of the recommendations are based on symptomatic relief using research conducted with patients with SIBO or, more commonly, patients with SIBO and an additional condition or multiple conditions. Further research is necessary to determine any possible connections between SIBO and other gastrointestinal diseases. Despite the presence of SIBO in many other conditions, more SIBO-specific research needs to be conducted to determine targeted treatments and recommendations for this population. ✪

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