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Intestinal gas production by the gut microbiota: A review

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ABSTRACT

In addition to causing embarrassment, intestinal gas can be associated with more serious symptoms. This review provides an overview of gas production by the human gut microbiome and outlines foods associated with intestinal gas. *Bacteroides*, *Ruminococcus*, *Roseburia*, *Clostridium*, *Eubacterium, Desulfovibrio,* and *Methanobrevibacter* are among the most abundant microbes responsible for intestinal gas. More than 99% of intestinal gas is composed of hydrogen, carbon dioxide, and methane, while less than 1% is composed of other odiferous compounds. Food groups associated with intestinal gas include pulses, vegetables, fruits, grains, and, for some individuals, dairy. These foods are rich in non–digestible carbohydrates such as raffinose family oligosaccharides, fructans, polyols, and, for sensitive individuals, lactose. These carbohydrates are fermented by colonic bacteria and produce gases directly or by cross–feeding. Additional research on gas production by the gut microbiota and foods associated with gas may help mitigate the symptoms linked to intestinal gas.

1. Introduction

During typical metabolism of dietary and endogenous components in the large intestine, the gut microbiota of most healthy people can generate 0.2L–1.5L of gas per day (Mego, Accarino, Malagelada, Guarner, & Azpiroz, 2015; Serra, Azpiroz, & Malagelada, 1998; Suarez, Springfield, & Levitt, 1998). Although the gut microbiota produces many volatile compounds as part of its metabolism (including short chain fatty acids), the gases that make up the majority of this volume are hydrogen (H_2), carbon dioxide (CO_2), and methane (CH_4). These gases contribute to more than 99 % of the intestinal gas volume and are odorless (Suarez & Levitt, 2000). The unpleasant odor associated with intestinal gas comprises less than 1 % of intestine gas volume and is the result of the sulfur–containing trace gases such as hydrogen sulfide (H_2S), methanethiol (CH_3SH), and dimethyl sulfide [$(CH_3)_2S$] as well as other volatile compounds such as short chain fatty acids (Kalantar-Zadeh, Berean, Burgell, Muir, & Gibson, 2019; Suarez et al., 1998).

In general, besides being socially awkward or causing embarrassment in public circumstances, intestinal gas is normal for most healthy people (Tomlin et al., 1991). However, intestinal gas is reported to be associated with other abdominal symptoms such as bloating, constipation, belching, abdominal pain, and excessive passing of gas. Excessive intestinal gas can have a negative impact on the social well–being of an individual and may also be a symptom of chronic conditions such as irritable bowel syndrome (IBS) (Caldarella et al., 2002). Indeed, intestinal gas is one of the most common health complaints that makes people visit a gastroenterologist (Arzpiroz & Michael, 2010; Manichanh et al., 2014).

Unfortunately, most of the foods that promote human health, including vegetables, high–fiber grains, and legumes, are known to contribute to intestinal gas (Manichanh et al., 2014; Mego, Accarino, Malagelada, Guarner, & Azpiroz, 2015; Winham, Webb, & Barr, 2008). These foods have unabsorbed residues that can be fermented in the colon by gut bacteria and lead to gas production as a by–product of microbial metabolism (Mego et al., 2015). The purpose of this review is to provide an overview of the gas–producing pathways used by the human gut microbiome and to outline foods associated with intestinal gas. Emphasis will be on gas–producing pathways from carbohydrate fermentation, with only some mention of gas–producing pathways from other dietary components due to the limited information on these substrates.

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2. Fermentation of non-digestible residues by gut microbiota and production of gases

The human gut is host to trillions of bacteria largely composed of strictly anaerobic microorganisms. The overall microbial population in the gastrointestinal (GI) tract, also known as the gut microbiota, varies widely from one person to another based on different factors such as diet, host genetics, and environmental conditions (Holscher, 2017). Although microbes that colonize the intestinal lumen may vary widely between individuals, the majority of bacterial species belong to five phyla, which are Actinobacteria, Bacteroidetes, Firmicutes, Proteobacteria, and Verrucomicrobia (Rinninella et al., 2019). These microorganisms contribute significantly to the health and disease of the host (Tremaroli & Bäckhed, 2012).

Among other functions, bacteria in the colon have the ability to ferment the substrates that are not digested or absorbed in the upper GI tract. These include carbohydrates, proteins, fats, and other dietary components that escape digestion due to molecular or physical structural complexity (Yao et al., 2016). Primary fermenters break these nutrients down for energy and metabolism and release metabolites such as short chain fatty acids (e.g., acetate, propionate, and butyrate), branched-chain fatty acids, ammonia (NH_3), *N*-nitro compounds, phenolic and indolic compounds, and various gases as by–products or intermediates of fermentation (Bernalier-Donadille, 2010). Human gut microorganisms form complex microbial communities that depend on one another to harvest nutrients and energy to survive (Pimentel et al., 2013); in fact, metabolites produced by one strain in the community may be further utilized by another (Smith et al., 2019). The metabolites released by microbiotas differ across individuals (Rinninella et al., 2019; Smith et al., 2019). Although some microbial fermentation products, especially short chain fatty acids, impact human health and are beneficial to the host, gases produced during fermentation can cause adverse symptoms in some people.

Although the majority of species belong to five phyla, the two major bacterial phyla responsible for gas production in the gut belong to Bacteroidetes and Firmicutes and together they make up over 90 % of the total bacterial population of the human gut (Arumugam et al., 2013). Bacteria in these phyla produce primarily H_2 and CO_2 (Hylemon et al., 2018; Kalantar-Zadeh et al., 2019). CH_4 gas is also produced by colonic microbes from the metabolism of CO_2 and H_2 by Archaea in the colon. Minor gases such as H_2S and other sulfur–containing gases are produced in trace concentrations by sulfate–reducing bacteria (SRB), which can reduce sulfate compounds to H_2S (F. Suarez et al., 1997). Sulfate may be derived in the colon from sources such as proteins from animal foods which contain amino acids cysteine, methionine, and taurine, as well as carrageenan and other sulfated polysaccharides (Rey et al., 2013).

Table 1
Major gas producing microbes present in the human gut microbiome.

Phylum	Genus and/or species	Gas	Reference
Bacteroidetes	*Parabacteroides*	H_2 & CO_2	(Ezeji et al., 2021)
	Alistipes	H_2 & CO_2	(Oliphant & Allen-Vercoe, 2019)
	Bacteroides	H_2 & CO_2	(Smith et al., 2019)
Firmicutes	*Enterococcus*	H_2 & CO_2	(Robert & Bernalier-Donadille, 2003)
	Dorea	H_2 & CO_2	(Oliphant & Allen-Vercoe, 2019)
	Clostridium spp.	H_2 & CO_2	(Steer et al., 2001)
	Roseburia intestinalis	H_2 & CO_2	(Duncan et al., 2002)
	Ruminococcus	H_2 & CO_2	(Zheng et al., 2014)
	Anaerostipes caccae	H_2 & CO_2	(Schwiertz et al., 2002)
	Eubacterium rectale	H_2 & CO_2	(Duncan & Flint, 2008)
	Blautia	H_2 & CO_2	(Suzuki et al., 2018)
	Veillonella	H_2 & CO_2	(Aujoulat et al., 2014)
	Victivallis vadensis	H_2 & CO_2	(Zoetendal et al., 2003)
	Desulfotomaculum	H_2S	(Dordević et al., 2021)
Proteobacteria	*Desulfovibrio piger*	H_2S	(Rey et al., 2013)
	Desulfovibrio fairfieldensis	H_2S	(I. Kushkevych et al., 2019)
	Desulfovibrio desulfuricans	H_2S	(Kushkevych, 2016)
	Desulfobulbus	H_2S	(Dordević et al., 2021)
	Desulfomicrobium	H_2S	(Ivan V Kushkevych, 2014)
	Desulfomonas	H_2S	(Ivan V Kushkevych, 2014)
	Fusobacterium spp.	H_2S	(Mothersole & Wolthers, 2019)
	Bilophila	H_2S	(Braccia et al., 2021)
	Escherichia	H_2 & CO_2	(Suzuki et al., 2018)
Euryarchaeota	*Methanobrevibacter smithii*	CH_4	(Weaver et al., 1986)
	Methanosphaera stadtmanae	CH_4	(Fricke et al., 2006)
	Methannobrevibacter oralis	CH_4	(Scanlan et al., 2008)

2.1. Hydrogen

H_2 is the most predominant gas produced by colonic bacteria and is produced solely through bacterial fermentation of non–digestible substrates in the colon (Naito et al., 2018). Accordingly, breath H_2 has been used as a primary marker for diagnostic testing of carbohydrate malabsorption or small intestinal bacterial overgrowth. Fermentative metabolism by gut bacteria creates the potential for large quantities of H_2 gas to be produced within the gut. It has been reported that up to 1L of H_2 can be produced in 24 h, and this magnitude of gas can cause GI symptoms such as bloating, abdominal pain, and excessive flatus (Gasbarrini et al., 2009; Strocchi & Levitt, 1992).

The most abundant bacterial genera responsible for H_2 production in the colon are *Bacteroides*, *Ruminococcus*, and *Roseburia* (Duncan et al., 2002; Zheng et al., 2014). Other colonic taxa known to be associated with H_2 production include *Anaerostipes caccae*, *Clostridium* spp., *Eubacterium rectale*, *Enterococcus,* and *Victivallis vadensis* (Table 1) (Duncan & Flint, 2008; Ivan V. Kushkevych, 2013; Schwiertz et al., 2002; Steer, Collins, Gibson, Hippe, & Lawson, 2001; Zoetendal, Plugge, Akkermans, & de Vos, 2003).

The most common pathway used by bacteria to produce H_2 is the Embden–Meyerhof–Parnas pathway, also known as glycolysis. The majority of gut bacteria use this pathway to convert carbohydrates into pyruvate. The oxidation of reduced flavin (FADH) and nicotinamide adenine dinucleotides (NADH) by microbial hydrogenases is the reaction in the Embden–Meyerhof–Parnas pathway responsible for most H_2 produced in the colon (Fig. 1) (Den Besten et al., 2013; Hylemon et al., 2018). Other mechanisms by which H_2 can be produced include 1) cleavage of pyruvate to formate and subsequent metabolism by formate hydrogenlyase; 2) generation from pyruvate through the activity of pyruvate: ferredoxin oxidoreductase and hydrogenase (Carbonero et al., 2012; Louis et al., 2014; Macfarlane & Gibson, 1997).

The balance of H_2 concentration in the gut is crucial to colonic fermentation and the host. Indeed, high H_2 partial pressure can hinder bacterial fermentation. Therefore, to prevent the accumulation of H_2 in the gut, the removal of excess H_2 can be mediated by both the host and gut microbiota (Carbonero et al., 2012). About one–third of H_2 produced in the gut is utilized by other microbes in the colon and the remaining is

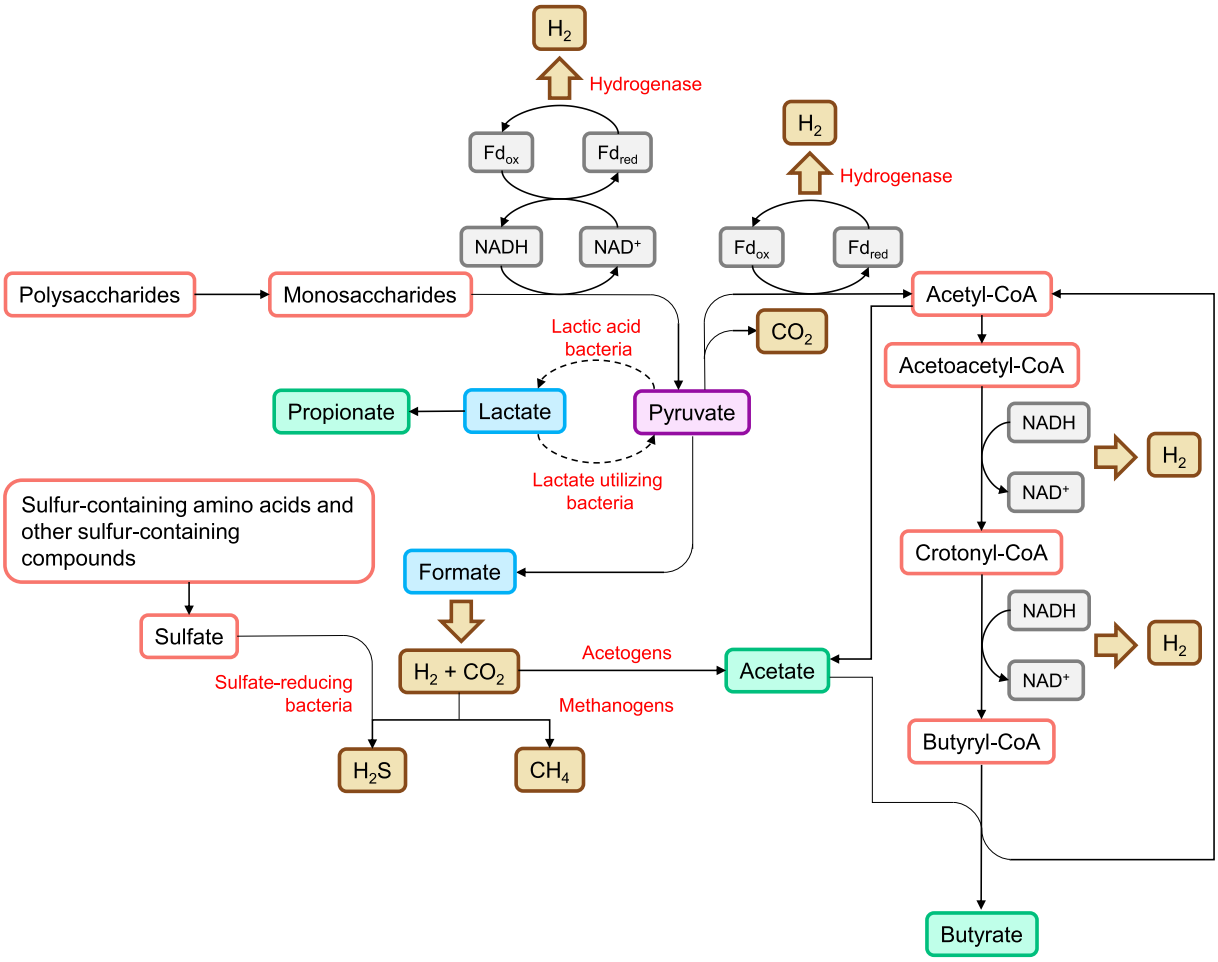

Fig. 1. Biochemical pathway of H_2, H_2S, CH_4, and CO_2 production from bacterial fermentation. Gases are shown in brown boxes; intermediate products of bacterial fermentation are shown in blue boxes and primary products of bacterial fermentation (short chain fatty acids) are shown in green boxes. Fd_{ox}, oxidized flavin adenine dinucleotide; Fd_{red}, reduced flavin adenine dinucleotide.

passed as flatus or excreted via breath (Christl et al., 1992; Hylemon et al., 2018). Hydrogenotrophic (H_2–utilizing) microbes responsible for converting H_2 into other metabolites include (1) methanogens, which use H_2 as the electron donor to reduce CO_2 and produce CH_4; (2) sulfate reducing bacteria, which reduce sulfate with H_2 to form H_2S; and (3) reductive acetogens that utilize the acetyl–CoA pathway to synthesize acetate from CO_2 and H_2 (Bernalier, Rochet, et al., 1996; Drake et al., 2008; Macfarlane & Gibson, 1997; Pimentel et al., 2013) (Table 2). These hydrogenotrophic bacteria prevent H_2 buildup in the colon that would thermodynamically inhibit fermentation and reduce the energy–extracting capacity of primary fermenters (Carbonero et al., 2012; Krajmalnik-Brown et al., 2012).

2.2. Carbon dioxide

CO_2 is one of the major gases produced by colonic bacteria during fermentative metabolism of dietary substrates in the colon (Montalto et al., 2009). During fermentation, CO_2 can be produced from conversion of pyruvate to acetyl–CoA or cleavage of pyruvate to formate, which is then metabolized to H_2 and CO_2 by formate hydrogenlyase (Macfarlane & Gibson, 1997) (Fig. 1). Colonic bacteria, predominantly in the phyla Firmicutes and Bacteroidetes, are responsible for dietary carbohydrate fermentation and CO_2 production as a by–product of fermentation (Table 1).

Most CO_2 is passively absorbed by the colonic mucosa and enters into circulation through enterocytes and is then excreted via exhaled breath (Christl et al., 1992). Alternatively, unabsorbed CO_2 can be excreted in flatus or metabolized by resident microorganisms of the gut microbiome. For example, CO_2 can be reduced by hydrogenotrophic (H_2–utilizing) microbes such as methanogens in the presence of H_2 to produce CH_4, and by reductive acetogens that utilize the acetyl–CoA pathway to synthesize acetate from CO_2 and H_2 (Fig. 1; Table 2) (Drake et al., 2008; Fricke et al., 2006).

2.3. Hydrogen sulfide

SRB are anaerobic H_2 utilizers that are part of the human gut microbiota. SRB use H_2 as an electron donor to reduce sulfate and generate H_2S (Fig. 1). H_2S concentrations in the human large intestine and feces ranges from 0.17 and 0.38 mmol/L (Florin, 1991; Magee et al., 2000b). Sulfate may be derived in the gut from several foods but is predominantly from sulfur–containing amino acids in proteins (Magee et al., 2000a). Indeed, an increase in fecal H_2S was correlated with increase in red meat consumption, a good source of sulfur-containing amino acids (Richardson et al., 2000).

SRB numbers in the colon are approximately 10^3 to 10^{11} CFU/ gram of human stool (Gibson et al., 1993). SRB are primarily members of the *Desulfovibrio* genus of γ–proteobacteria (Pimentel et al., 2013). Particularly, *Desulfovibrio piger* is the most frequent SRB present in the colon (Rey et al., 2013). Other genera of colonic SRB include *Desulfotomaculum, Desulfobulbus, Desulfomicrobium, Desulfobacter,* and *Desulfomonas* (Barton & Hamilton, 2007; Kushkevych, 2016; Kushkevych & Moroz, 2012; Ivan V. Kushkevych, 2014).

The abundance of SRB and the level of H_2S accumulation in the

Table 2
Gas-utilizing gut microbes.

Classification	Genus and/or species	Gas utilized	By-product	Reference
Sulfate-reducing bacteria	*Desulfovibrio piger*	H_2	H_2S	(Rey et al., 2013)
	Desulfovibrio fairfieldensis	H_2	H_2S	(I. Kushkevych et al., 2019)
	Desulfovibrio desulfuricans	H_2	H_2S	(Kushkevych, 2016)
	Desulfobulbus	H_2	H_2S	(Dordević et al., 2021)
	Desulfomicrobium	H_2	H_2S	(Ivan V Kushkevych, 2014)
	Desulfomonas	H_2	H_2S	(Ivan V Kushkevych, 2014)
	Desulfovibrio piger	H_2	H_2S	(Rey et al., 2013)
Methanogens	*Methanobrevibacter smithii*	H_2 & CO_2	CH_4	(Weaver et al., 1986)
	Methanosphaera stadtmanae	H_2 & CO_2	CH_4	(Fricke et al., 2006)
	Methannobrevibacter oralis	H_2 & CO_2	CH_4	(Scanlan et al., 2008)
Reductive acetogens	*Ruminococcus*	H_2 & CO_2	Acetate	(Bernalier, Rochet, et al., 1996; Macfarlane & Gibson, 1997b)
	Clostridium	H_2 & CO_2	Acetate	(Bernalier, Rochet, et al., 1996; Macfarlane & Gibson, 1997)
	Peptostreprococcus	H_2 & CO_2	Acetate	(Macfarlane & Gibson, 1997b)
	Streptococcus	H_2 & CO_2	Acetate	(Bernalier, Rochet, et al., 1996)
	Blautia hydrogenotrophica	H_2 & CO_2	Acetate	(Bernalier, Willems, et al., 1996; Liu et al., 2008)

human gut can have a health impact on the individual. High concentrations of H_2S have toxic effects on human tissues (I. Kushkevych et al., 2019; Schicho et al., 2006). H_2S can disrupt colonocyte metabolism and growth by impairing butyrate oxidation and protein synthesis (Roediger et al., 1993). Furthermore, high levels of H_2S can cause DNA damage of epithelial cells (Attene-Ramos et al., 2007). Consequently, several studies have suggested that sulfide is associated with intestinal disorders such as inflammatory bowel disease (IBD) (Gibson et al., 1991).

2.4. Ammonia

Colonic bacteria produce NH_3 by the deamination of amino acids. Different from the other gases discussed herein, NH_3 is primarily dissolved in the aqueous phase of the GI tract·NH_3 concentration detected in human feces varies from 10 to 30 mM and excretion has been shown to increase with a higher intake of proteins (Cummings et al., 1979; Hughes et al., 2000)·NH_3 exhibits several toxic effects by changing the morphology and intermediary metabolism of intestinal cells, which hinders DNA biosynthesis and diminishes the lifespan of cells (Visek, 1978). Other studies reported that ureterosigmoidostomy patients who have a luminal NH_3 concentration as high as 100 mM have an increased risk of developing tumors distal to the site of ureteric implantation (McConnell et al., 1979; Tank et al., 1973). Animal studies have also reported that elevated levels of NH_3 can damage the colonic mucus layer and reduce colonocyte lifespan (Andriamihaja et al., 2010; Lin & Visek, 1991). High levels of NH_3 caused the inhibition of butyrate oxidation in the colonocytes of rats (Cremin et al., 2003).

2.5. Methane

Methanogens, a group of microorganisms within the kingdom Euryarchaeota of the domain Archaea, can utilize H_2 as electron donors to reduce CO_2 to CH_4 (Fig. 1) (Scanlan et al., 2008). The process of conversion of CO_2 and H_2 to CH_4 has the effect of reducing total gas volume by a factor of 5 ($CO_2 + 4H_2 \rightarrow CH_4 + 2H_2O$: 5 mol gas → 1 mol gas) (Bauchop & Mountfort, 1981; Blaut, 1994). Therefore, methanogenic metabolism can decrease total gas volume (Pimentel et al., 2013). However, CH_4 production is considered a potential biomarker to diagnose symptoms and disease in GI disorders. High levels of CH_4 have been linked to decreased intestinal motility and are associated with constipation, especially in patients with IBS with constipation (Chatterjee et al., 2007; Kunkel et al., 2011; Pauff & Miller, 2012).

Substantial inter-individual differences exist in colonic methanogenesis. The abundance of methanogens in human fecal samples varies from undetectable to 10^9 CFU per g of stool and a threshold population of 10^7 is required to result in detectable levels of CH_4 in the breath (Pochart et al., 1992). Studies indicate *Methanobrevibacter smithii* is the most abundant Archaeal species present in the GI tract (Eckburg et al., 2005; Weaver et al., 1986). Other methanogenic species in the GI tract responsible for CH_4 production are *Methanosphaera stadtmanae* and *Methanobrevibacter oralis* (Fricke et al., 2006; Triantafyllou et al., 2014).

3. Intestinal gases and adverse symptoms

Although intestinal gas is normal for most healthy people and imparts no adverse effects, many patients with GI disorders attribute their symptoms to gas in the gut. Gases produced during fermentation by the gut microbiota can be eliminated from the GI tract via three means: 1) further metabolism by gas-consuming microorganisms into non-gaseous products (e.g., reductive acetogens converting H_2 and CO_2 to acetate); 2) absorption into the bloodstream and exhalation via the breath; and 3) through the anus as flatulence (Mego et al., 2017). In one study, it was shown that about 77 % of microbial gas is eliminated via the first two pathways, while the remaining 23 % is eliminated via the third, less desirable pathway (Mego et al., 2017). Patients that complain of frequent bloating and flatulence have been shown to have impaired clearance of gas from the GI tract (Salvioli et al., 2005). They also tend to experience adverse symptoms in response to gas volumes that are usually well-tolerated by healthy subjects (Malagelada et al., 2012; Mego et al., 2017).

Bloating and abdominal distension often present as isolated, transient episodes (Bendezú et al., 2015). Therefore, abnormalities in the quantity or location of gas production in the GI tract can result in adverse symptoms for the patient (Bendezú et al., 2015). For example, H_2 accumulation due to bacterial fermentation of carbohydrates in the small intestine contributes to the bloating and abdominal pain experienced by patients with small intestinal bacterial overgrowth (Azpiroz & Malagelada, 2005; Pimentel et al., 2000). Furthermore, rapidly-fermenting carbohydrates such as raffinose family oligosaccharides (RFO) are often implicated in causing bloating because they can cause rapid production of gas that is difficult to eliminate from the GI tract without distension (Price, Lewis, & Fenwick, 1988). Importantly, the rate at which gases can be eliminated from the GI tract before reaching the anus (i.e., via pathways 1 and 2 above) is not static, but can be modified by diet. For example, immediately after beginning consumption of galactooligosaccharides (GOS), a short-chain, rapidly fermenting non-digestible carbohydrate, total gas volume increased by 37 %; however, gas volume declined back to baseline levels after consumption continued for two weeks. Moreover, the volume of gas cleared from the lumen before reaching the anus tended to increase following two weeks of consumption (Mego et al., 2017). Therefore, continued consumption of flatulogenic foods may increase gas-consuming pathways by the microbiota and increase the ability of gas to be absorbed into the bloodstream to be eliminated in the breath.

Beyond the bloating, intestinal distension, and flatulence that can arise primarily by the major gases produced by the microbiota, some of the less abundant gases have shown specific adverse effects on patient health. In particular, high levels of CH_4 have been linked to decreased intestinal motility and are associated with constipation, especially in patients with IBS with constipation (Chatterjee et al., 2007; Kunkel et al., 2011; Pauff & Miller, 2012). Studies in human model systems demonstrated that high levels of CH_4 augmented intestinal contractile activity and slowed down the peristalsis (Pimentel et al., 2006). The rate of excretion of H_2 and CH_4 in IBS patients has been reported to be significantly higher compared to healthy individuals (King et al., 1998).

Both clinical and experimental studies have linked bacterial-generated H_2S to increased toxic effects, especially in patients with IBD. A high concentration of H_2S has toxic effects to the epithelial cell of the colon (Kushkevych et al., 2019; Schicho et al., 2006). For instance, one study demonstrated that high levels of H_2S can cause DNA damage to epithelial cells (Attene-Ramos et al., 2007). In addition, H_2S can impair metabolic activity of colonocytes, such as butyrate oxidation and protein synthesis. Consequently, several studies have suggested that sulfide is associated with intestinal disorders such as IBD (Gibson, Cummings, & Macfarlane, 1991). *In vitro* studies have shown that the quantity of SRB (such as *Desulfovibrio piger*) in stool samples from patients with ulcerative colitis (UC) were greater compared to stool samples from healthy individuals. Furthermore, these patients produced significantly more H_2S than healthy individuals (Levine et al., 1998; Loubinoux et al., 2002; Roediger et al., 1997). The removal of foods rich in sulfur-containing amino acids (milk, eggs, and cheese) has proven therapeutic to those with UC (Truelove, 1961).

Multiple studies reported that methanogens and SRB are associated with colorectal cancer (CRC). Methanogens and SRB provide a source of CH_4 and H_2S, respectively, and these molecules influence the colonic epithelium. For instance, there is a higher prevalence of CH_4 excretion among patients with CRC compared with healthy individuals (Flick et al., 1990; Holma et al., 2012; Peled, 1985; Piqué et al., 1984). In addition, higher stool H_2S was reported in individuals with a high risk of CRC (and UC) compared to healthy individuals. One study reported that thiosulfate sulfurtransferase, an enzyme responsible for H_2S detoxification, was present in lower abundance in biopsy tissue from patients with CRC (Ramasamy et al., 2006).

While there exist these relationships between intestinal gases and diseases, the molecular mechanisms by which these gases influence health have been explored only to a limited extent and should be further studied in the future.

4. Food and intestinal gas

Although there are many causes of intestinal gas, food is one of the main causes influencing gas symptoms (Hasler, 2006; Tomlin et al., 1991). A large portion of people associate intake of certain foods with the development of intestinal gas and other GI symptoms (Mego et al., 2015). This is especially true for foods rich in non–digestible, fermentable carbohydrates, proteins, and fats (Gibson, Varney, Malakar, & Muir, 2015). Recent studies have emphasized the relationship between GI disorders and food intake, and many people, particularly those with GI disorders want to know specific foods that can contribute to GI symptoms. Several studies have reported food groups and specific food items that cause intestinal gas and other GI symptoms in patients with GI disorders and healthy people. The most commonly reported flatulogenic food groups include pulses, vegetables, fruits, whole grains, and dairy products (MacDermott, 2007; Manichanh et al., 2014).

4.1. Pulses

Pulse foods including dry beans, lentils, peas, and others are an important staple food for many people across the world because of their nutritional, economic, and health benefits. Pulses are generally low in fat and an inexpensive rich source of proteins, vitamins, fibers, and minerals (Perera et al., 2020). However, despite all these benefits, many consumers avoid eating pulses, especially a wide variety of beans, because of the fear of excessive flatulence and stomach discomfort (Descrochers & Brauer, 2001; Fleming et al., 1985). It has been reported that increased flatulence is an expected outcome among some people after the inclusion of pulses in their diet, especially if fiber intake is already low (Perera et al., 2020; Tomlin et al., 1991). However, it has been reported that flatulence does decrease with more frequent consumption of pulses (Livesey, 2001). In a randomized controlled trial, 50 % of healthy subjects reported increased flatulence in the first week of consuming pinto or baked beans, but the reported percentage dropped to 38 % after the second week (Winham & Hutchins, 2011). Thereafter the percentage of people reporting flatulence symptoms consistently declined to 15–23 % for weeks 6–12. Similarly, in another randomized controlled trial, the impact of 28 consecutive days of consumption of pulses (chickpeas, lentils, and green peas) was assessed (Veenstra et al., 2010). A significant increase in flatulence in the early phase of the intervention was observed with the consumption of each pulse, but the level of flatulence significantly declined in the late phase. Based on these studies, the frequency of passing gas depends on the how frequently an individual consumes pulses, and the response can vary significantly from one individual to another.

Most pulses contain relatively high amounts of dietary fibers including resistant starch. Furthermore, they are particularly rich in soluble, fermentable oligosaccharides that belong to the RFOs (Table 3) (Elango et al., 2022). RFOs are non–reducing carbohydrates consisting of one to several 1 → 6–linked α–galactopyranosyl units linked to C-6 of the glucose moiety of sucrose (Andersen et al., 2005). Raffinose, stachyose, and verbascose are the common RFOs (Van den Ende, 2013). Raffinose and stachyose exist ubiquitously in plants, whereas verbascose is found in the vacuoles of only certain plants (Elsayed et al., 2014). These RFOs cannot be digested or absorbed in the small intestine due to the lack of α–galactosidase enzymes in humans to degrade RFOs (Kalantar-Zadeh et al., 2019). Therefore, once RFOs reach the colon, they undergo fermentation by colonic bacteria that produce gases as metabolic byproducts (Price et al., 1988). Consequently, consumption of legumes can cause increased flatulence in some people (Lacy et al., 2011; Naczk et al., 1997). Many studies have found that the removal of RFOs from plants through processing methods such as extrusion, soaking, autoclave, enzyme use, and boiling can reduce RFOs; and presumably reduce gas production (Çelem & Önal, 2022).

4.2. Vegetables and fruits

Vegetables that are high in fructans have been reported to be associated with increased flatulence in humans after consumption (Bruhwyler et al., 2009; Grabitske & Slavin, 2009). The fructans in vegetables and fruits are classified as inulin-type, which consist of one to several 2

Table 3
Variability in raffinose family oligosaccharides (mg/g) present in dry seeds of various plants (Elango et al., 2022; Price et al., 1988).

Crop	Raffinose	Stachyose	Verbascose
Dry Peas	4.1–10.3	10.7–26.7	0.0–26.7
Soybean	11.1.–18	31–48	–
Chickpea	8.1–9	15–19	–
Lentil	28.6–37	24.6–28.8	3.9–7.2
Green peas	30.1	35.4	15
Cowpea	12	34	9
Mung bean	4.1–5	17–20	–
Peanut	3	9	–
Red kidney bean	3.1	31.6	–
Green bean	2.5	34.3	–
Faba bean	2.3	10.7	11.4
Black eyed peas	4	4	–
Lima bean	6.9	30.3	–

→ 1–linked β-fructofruanosyl units linked to the fructose moiety of sucrose. The simplest fructan of this type is 1-kestose, which is β-fructofuranosyl-(2 → 1)-β-fructofuranosyl-(2 → 1)-α-glucopyranoside. Degrees of polymerization of inulin-type fructans usually ranges from 3 to about 60 (Cooper et al., 1996).

Inulin and fructooligosaccharides (FOS) are abundant in vegetables such as artichokes, asparagus, chicory root, garlic, onions, dandelion greens and leeks (Table 4). Fructans cannot be digested in the small bowel and result in excessive intestinal gas after bacterial fermentation in the colon. Two recent studies examined the effect of consuming inulin–containing foods on GI symptoms. The findings from both studies showed that the most frequent GI symptom reported by healthy volunteers after consuming inulin–rich diets was increased flatulence (Holscher et al., 2014; Slavin & Feirtag, 2011). In another study, FOS was reported to contribute to increasing the production of intestinal gas during 5 weeks of consumption of a FOS–rich diet (Alles et al., 1996).

Sugar alcohols, also known as polyols, are found naturally in some fruits and vegetables (Table 5) and have also been implicated in intestinal gas due to their poor absorption in the small intestine (Grembecka, 2015; Langkilde et al., 1994; Lenhart & Chey, 2017). For example, one study assessed the association between sorbitol malabsorption and GI symptoms when 7 healthy participants consumed different doses of sorbitol (Hyams, 1983). In a majority of subjects (4 of 7), ingestion of as little as 5 g of sorbitol was associated with significant increase in gas, while most subjects experienced severe symptoms (gas, bloating, cramps, and diarrhea) after consuming 20 g of sorbitol. Similarly in another study with 10 healthy volunteers, ingestion of hydro solution containing high dose of mannitol led to the highest rate of side effects, including flatulence, diarrhea, and abdominal pain compared to the ingestion of low dose of mannitol (Ajaj et al., 2004). Both these studies conclude that the malabsorption of sugar alcohols causes flatulence among participants, but the severity of the symptom depends on the dose of polyols.

Polyols are also commonly used as sugar–free sweeteners in chewing gums and beverages (Yao et al., 2014). Examples of polyols approved by the US FDA are mannitol, sorbitol, xylitol, isomalt, lactitol, maltitol, erythritol, and hydrogenated starch.

4.3. Whole grains

Grains such as wheat, corn, barley, rye, and oats are staple foods that most of the world's population rely on as the main proportion of the diet. However, nutritionists and governmental agencies recommend consuming whole rather than refined grains. The 2020–2025 Dietary Guidelines for Americans recommend eating at least 3 oz of whole grains/day due to the associated health benefits of whole grain consumption. Whole grains contain some soluble fibers and resistant starch that can be fermented, but mostly contain poorly fermentable carbohydrates such as cellulose and cross–linked arabinoxylan (Nirmala Prasadi & Joye, 2020). However, whole grains also contribute to a significant quantity of fructans to diets (Table 6). The fructans in grains are different from those found in fruits and vegetables. They are classified as levan–type (Gallagher et al., 2007; van den Ende et al., 2011; Yoshida & Tamura, 2011), which contain β-fructofuranosyl units linked by (2 → 6) bonds to sucrose, as in 6-kestose [β-fructofuranosyl-(2 → 6)-β-fructofuranosyl-(2 → 1)-α-glucopyranoside] (Roberfroid & Delzenne, 1998; Rossi et al., 2005). Levans from grains have been reported to reach about 90 degrees of polymerization (Roberfroid & Delzenne, 1998). Although the concentrations of fructans in grains are not high, wheat is the major source of naturally occurring fructans in the diets of Americans due to the frequent consumption of wheat (Moshfegh et al., 1999).

A substantial amount of research has linked whole grain consumption to increased flatulence. In an uncontrolled study, 55 % of IBS patients reported that bran made their GI symptoms worse including gaseous complaints (Francis & Whorwell, 1994). Controlled trials of wheat bran have also reported increased flatulence and abdominal discomfort in participants compared to placebo (Cann et al., 1984). Additionally, (Vuholm et al., 2017) investigated whether whole–grain wheat and whole–grain rye affects GI symptoms and found that intake of these whole grains was significantly associated with increased flatulence in healthy volunteers (Vuholm et al., 2017).

Although fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (FODMAPs) such as fructans found in wheat and other grains are commonly known to cause severe GI symptoms in IBS patients (Shepherd et al., 2008), it has been also reported that many people with non-Celiac gluten sensitivity may in fact be sensitive to FODMAPs in wheat rather than gluten (Molina-Infante et al., 2014). Consequently, several studies have evaluated different methods to reduce fructans in whole grain foods, which can presumably decrease

Table 4
Inulin and fructooligosaccharide contents of selected vegetables (Alles et al., 1996; Holscher et al., 2014; Loo et al., 1995; Moshfegh et al., 1999; Sabater-Molina et al., 2009; Slavin & Feirtag, 2011).

	Inulin (g/100 g)	Fructooligosaccharides (g/100 g)
Jerusalem artichoke	16.0–20	12.0–15.0
Globe artichoke	1.2–6.8	0.2–0.7
Chicory roots	35.7–47.6	19.6–26.2
Dandelion greens		
Raw	12.0–15.0	9.6–12.0
Cooked	8.1–10.1	6.5–8.1
Garlic	9.0–16	3.6–6.4
Onion		
Raw	1.1–7.5	1.1–7.5
Cooked	0.8–5.3	0.8–5.3
Leeks	3.0–10.0	2.4–8.0
Asparagurus		
Raw	2.0–3.0	2.0–3.0
Cooked	1.4–2.0	1.4–2.0

Table 5
Sorbitol and mannitol concentration of selected vegetables and fruits.

Category	Sorbitol (g/100 g)	Mannitol (g/100 g)
Vegetables		
Brussel sprouts	0.2	0
Brocolli	0.3	0
Cabbage	0.2	0
Cauliflower	0	2.6
Celery	0	1.5
Mushrooms	0.1	2.6
Sweet potatoes	0	0.3
Fruits		
Plum	2.4	0
Apple	1.2	0
Apricot	1.2	0
Blackberries	4.1	0
Cherries	0.7	0
Nectarine	1	0
Peach	0.9	0.5
Pear	2.3	0

Table 6
Inulin and fructooligosaccharide contents of selected grains (Alles et al., 1996; Holscher et al., 2014; Loo et al., 1995; Moshfegh et al., 1999; Sabater-Molina et al., 2009; Slavin & Feirtag, 2011).

Grain	Inulin (g/100 g)	Fructooligosaccharides (g/100 g)
Wheat		
Bran–raw	1.0–4.0	1.0–40
Flour–baked	1.0–3.8	1.0–3.8
Flour–boiled	0.2–0.6	0.2–0.6
Barley		
Raw	0.5–1	0.5–1.0
Cooked	0.1–0.2	0.1–0.2
Rye		
Baked	0.5–0.9	0.5–0.9

intestinal gas and other GI symptoms. One study evaluated the ability of different sourdough strains of yeast to degrade fructans in wheat flour during 96 h of fermentation. After the fermentation, *Saccharomyces cerevisiae* and *Torulaspora delbrueckii* isolated from Australian sourdough demonstrated the ability to significantly reduce the fructan content in the wheat flour compared to traditional baker's *S. cerevisiae* isolates ((Fraberger et al., 2018). In another study, (Pejcz, Spychaj, & Gil, 2020) also investigated the potential of *Lactobacillus plantarum* with extended fermentation time with *Saccharomyces cerevisiae* to degrade fructans in rye bread during rye dough fermentation. After 3 h of fermentation, the authors noticed that the content of fructans in bread was significantly decreased in rye bread fermented with *L. plantarum* and yeast than in bread fermented with baker's yeast alone (Pejcz et al., 2020). Therefore, based on these studies, the use of *Lactobacillus* strains and sourdough yeast can be an effective method to reduce the content of fructans in whole grain bread.

4.4. Dairy products

Milk and other dairy products are among the food items that aggravate GI symptoms among the considerable proportion of the adult population of the world that is deficient in lactase, an enzyme responsible for the hydrolysis of lactose, the primary carbohydrate in dairy foods (Campbell et al., 2009). In most mammals, lactase activity is high in children but decreases rapidly with age. In humans, about 30 % of the world's population is lactase persistent as adults, but the prevalence of lactase persistence varies widely in frequency across the human population (Bayless et al., 2017; Montgomery et al., 2007). Lactose is a unique carbohydrate present in mammalian milk, 7.2 g/100 ml in human milk and 4.7 g/100 ml in cow's milk (Solomons, 2002). When lactose is not hydrolyzed into its simple sugars (glucose and galactose) in the small intestine, it enters the large intestine where it serves as a fermentable substrate for colonic bacteria (Campbell et al., 2009). It is reported that patients suffering from lactase deficiency experience increased flatulence as a result of the fermentation of lactose in the colon (Le Nevé et al., 2019).

5. Conclusion

The gaseous by–products of microbial fermentation can have both direct (e.g., IBD, constipation, and DNA damage) and indirect (abdominal pain and bloating) effects on the host. It is clear that the presence of different strains of bacteria in the colon and various types of foods rich in non–digestible substrates play a role in aggravating GI symptoms, especially increased flatulence, in patients with GI disorders and healthy people. However, people with GI disorders often experience adverse symptoms in response to gas volumes that are usually well-tolerated by healthy subjects, and continued consumption of flatulogenic foods may increase gas-consuming pathways by the microbiota and increase the ability of gas to be absorbed into the bloodstream to be eliminated in the breath. Therefore, it is not possible to draw up a specific list of foods to recommend or avoid. Rather, further research examining the interactive effects of gut microbiome composition, gaseous products produced, and adverse symptoms experienced will advance the field toward reducing GI discomfort caused by gases in the GI tract.

6. Ethics statement

This study did not involve human or animal subjects. Therefore, no approvals for human or animal research were necessary.

Author contributions

Funding (D.R., S.L., M.S.); resources (D.R.); writing - first draft (E. M.); writing - reviewing and editing (E.M., D.R., S.L., M.S.). All authors read and approved the final version of the manuscript.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data was used for the research described in the article.

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