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What Do Gases in the Large Intestine Have to Do with Health?

Klaudia Anna Pawełek
Edward Szczeklik Specialist Hospital in Tarnów, Szpitalna 13 Street, 33-100 Tarnów
https://orcid.org/0009-0005-6166-6396
klaudia.ludew@gmail.com

Patrycja Kinga Marta
Fryderyk Chopin University Clinical Hospital in Rzeszow, Fryderyk Chopin Street 2, 35-055 Rzeszow
https://orcid.org/0009-0009-6972-8140
pat.martaa@gmail.com

Filip Maciej Huzarski
St. Queen Jadwiga Clinical District Hospital No. 2 in Rzeszow, Lwowska 60 Street, 35-301 Rzeszow
https://orcid.org/0009-0002-3773-5388
fhuzarski@gmail.com

Gabriela Monika Ferfecka
St. Queen Jadwiga Clinical District Hospital No. 2 in Rzeszow, Lwowska 60 Street, 35-301 Rzeszow
https://orcid.org/0009-0001-2908-9171
g.ferfeckaa@gmail.com

Magdalena Rosa-Bończak
Fryderyk Chopin University Clinical Hospital in Rzeszow, Fryderyk Chopin Street 2, 35-055 Rzeszow
https://orcid.org/0009-0005-7266-6930
magros1996@gmail.com

Agata Ossolińska

Fryderyk Chopin University Clinical Hospital in Rzeszow, Fryderyk Chopin Street 2, 35-055 Rzeszow
https://orcid.org/0009-0005-4941-7039
ossolinska.agata@gmail.com

Weronika Kłosowicz
Fryderyk Chopin University Clinical Hospital in Rzeszow, Fryderyk Chopin Street 2, 35-055 Rzeszow
https://orcid.org/0009-0006-1452-7411
weronikacebula11@gmail.com

Oliver Carlton
Poznan University of Medical Science General, oncological and colorectal surgery clinic Szwacjarska 3 Street, 61-285, Poznan
https://orcid.org/0009-0001-1506-626X
olic25@op.pl

Lucyna Stolarska
Stefan Żeromski Specialist Hospital Independent Public Health Care Facility, 66 Na Skarpie Housing Estate, 31-913 Kraków
https://orcid.org/0009-0009-0480-304X
lucynka.stolarska@gmail.com

Natalia Morawiecka
Fryderyk Chopin University Clinical Hospital in Rzeszow, Fryderyk Chopin Street 2, 35-055 Rzeszow
https://orcid.org/0009-0006-6043-8188
n.morawiecka@gmail.com

Abstract:

A healthy human gastrointestinal tract contains approximately 200 ml of gas, with an average daily

elimination of 600 ml. Sources of intestinal gases include swallowing air and products of intestinal fermentation, primarily in the large intestine. The gases consist of nitrogen, oxygen, carbon dioxide, hydrogen, and methane, with carbon dioxide and hydrogen being the most abundant. The gut-brain axis, regulated by the nervous system and gut microbiota, influences intestinal motility, hormone secretion, and metabolic processes. Gut microorganisms produce short-chain fatty acids (SCFAs), which play a significant role in regulating energy homeostasis and enteroendocrine hormone secretion.

Disruptions in gut microbiota, such as in irritable bowel syndrome (IBS) and small intestinal bacterial overgrowth (SIBO), lead to changes in gas production, discomfort, and gastrointestinal symptoms. Diagnosis of these conditions relies on breath tests and ruling out other pathologies. Additionally, carbohydrate intolerances, such as fructose or lactose intolerance, result in excessive gas production and malabsorption issues. Celiac disease, an autoimmune condition, causes damage to intestinal villi, impairing digestion and absorption. A comprehensive approach to diagnosing and treating these conditions, including dietary modifications and microbiological interventions, plays a crucial role in improving patients' quality of life.

Key words: abdominal bloating; irritable bowel syndrome; celiac disease; malabsorption syndrome; small intestine bacterial overgrowth.

Introduction

The gastrointestinal tract of a healthy individual contains approximately 200 ml of gas, and the average amount of gas expelled from the digestive tract is about 600 ml daily. Intestinal gases have two primary sources: the first is air swallowed during eating or speaking, and the second is the byproduct of intestinal fermentation, which mostly occurs in the large intestine. The gases present in the digestive tract include nitrogen, oxygen, carbon dioxide, hydrogen, and methane. Swallowed air mainly contains nitrogen and oxygen, while internal intestinal processes lead to the production of gases with varying compositions, such as methane, hydrogen, and carbon dioxide.

Carbon dioxide is generated by the neutralization of alkaline pancreatic juice by acidic gastric juice components and acids derived from food or digestion processes. Methane and hydrogen are produced through bacterial fermentation in the large intestine. In healthy individuals, carbon dioxide and hydrogen are the most abundantly produced gases, with small amounts of odorous gases like indole, skatole, and sulfur compounds. [1]

Composition and characteristics of intestinal gases.

Gas	Source	Characteristic
Azote (N_2)	It mainly comes from air swallowed during eating, drinking, and talking.	It makes up 50-80% of the volume of intestinal gases. It is not metabolized or absorbed. An essential component of air, odorless, and neutral to physiological processes.
Oxygen (O_2)	It comes from swallowed air.	It is quickly absorbed by the intestinal mucosa. Consumed by aerobic bacteria. Its concentration in the intestines is low.
Carbon Dioxide (CO_2)	It is produced through the neutralization of acids (e.g., hydrochloric acid) and the fermentation of food by intestinal bacteria.	It is one of the main gases in the gastrointestinal tract. It is absorbed by the intestines, transported to the lungs, and exhaled. It can cause bloating after consuming foods rich in fermentable sugars.
Hydrogen (H_2)	It is produced by intestinal bacteria during the fermentation of carbohydrates and fiber.	It is exhaled with breath. Its excess may indicate small intestinal bacterial overgrowth (SIBO).
Methane (CH_4)	Produced by methanogenic bacteria in the large intestine, which convert hydrogen and carbon dioxide into methane.	Methane is not present in all people. Methane production can affect slower intestinal transit and constipation. It is odorless.

Gas	Source	Characteristic
Hydrogen sulphide (H_2S)	It is produced during the breakdown of sulfur-containing proteins, such as cysteine and methionine.	It is intensely detectable in small amounts. It is responsible for the characteristic unpleasant smell of intestinal gases. It can be toxic in excess.
Indole and Skatole	They are formed from tryptophan as a result of protein breakdown.	They have an intense, unpleasant odor. Their presence indicates protein breakdown, especially of animal-origin proteins.
Ammonia (NH_3)	It is formed from the breakdown of nitrogen compounds, such as urea and amino acids, by intestinal bacteria.	It is absorbed by the intestinal wall and transported to the liver, where it is neutralized. Excessive amounts may indicate dysbiosis or liver disorders.

Source: Own study based on [2,3,4,5,6]

Intestinal Gases and the Gut-Brain Axis

The amount and type of gases produced during fermentation depend on the type of food consumed, the composition of the gut flora, and the efficiency of intestinal transit. Food intake and energy regulation are tightly controlled by the central and peripheral nervous systems. There are two important centers that regulate appetite: the hunger center, located in the lateral hypothalamic area, and the satiety center, found in the paraventricular nuclei of the hypothalamus. Bidirectional brain-gut communication plays a crucial role in regulating intestinal function. Sensory stimuli, such as sound, images, smells, visceral and somatic sensations, are processed in the central nervous system

using knowledge, memory, and emotions, and then reach the enteric nervous system, influencing motility, secretory processes, immune responses, and blood flow in the gastrointestinal tract.

It is believed that the hunger center is continuously active, and its activity increases during fasting under the influence of orexigenic peptides such as ghrelin and orexins. These peptides act on neurons in the arcuate nucleus of the hypothalamus, activating the release of neuropeptide Y and AgRP (agouti-related peptide), which increase the sensation of hunger. Conversely, anorexigenic peptides, such as cholecystokinin, peptide YY, and glucagon-like peptide-1, act on arcuate nucleus neurons, stimulating the release of pro-opiomelanocortin, α-melanocyte-stimulating hormone, and the CART system (cocaine and amphetamine-regulated transcript), which promote satiety and reduce appetite.

The functioning of hunger and satiety centers is influenced by numerous short-term neural and hormonal signals, primarily from the stomach and intestines. For example, gastric distension caused by food stimulates mechanoreceptors, while the presence of digested carbohydrates, proteins, or fats in the intestine activates chemoreceptors in the enteric nervous system, transmitting signals to centers in the brainstem, and subsequently to the arcuate nucleus and the satiety center [7,8].

Gut microbiota also play a significant role in regulating the gut-brain axis. Intestinal microorganisms metabolize undigested carbohydrate complexes, producing short-chain fatty acids (SCFAs), which can be absorbed and used as an energy source or act as signaling molecules. The main SCFAs produced by gut bacteria are acetate, propionate, and butyrate. Propionate can be used for glucose and lipid synthesis. SCFAs stimulate the secretion of peptide YY, which slows intestinal motility, delaying food transit and increasing nutrient absorption. Butyrate affects the body's energy homeostasis by stimulating leptin production in adipocytes and GLP-1 secretion by intestinal L-cells, as well as increasing thermogenesis, fatty acid oxidation, and mitochondrial activity in muscles and brown adipose tissue.

Additionally, gut microbiota transform primary bile acids into secondary bile acids, which act as signaling molecules by binding to receptors such as the farnesoid X receptor and G-protein-coupled receptors. Like SCFAs, secondary bile acids can influence the release of enteroendocrine hormones, including glucagon-like peptide-1 (GLP-1) [9,10].

Gases and Gastrointestinal Disorders

Irritable Bowel Syndrome (IBS) is a chronic functional disorder, characterized primarily by recurrent abdominal pain associated with bowel movements or changes in stool frequency and consistency. The causes of IBS are complex and not fully understood, but disturbances in the gut-brain axis play a key role. An important element of these disturbances is abnormalities in the gut microbiota, which affect neurogenic, endocrine, and immune mechanisms involved in regulating this axis.

The gut microbiota plays a crucial role in maintaining health. Dysbiosis, or disruption of microbiota balance, leads to a decrease in beneficial bacteria, such as *Lactobacillus* and *Bifidobacterium*, and an increase in pathogenic microorganisms, such as *Escherichia coli* and *Clostridium spp.* Disruption of the gut microbiota can exacerbate IBS symptoms by causing changes in intestinal motility, visceral hypersensitivity, and inflammation. In IBS, reduced diversity of *Lactobacillus*bacteria is often observed, which plays an important role in maintaining gut ecosystem balance and regulating the immune system.

One of the triggering factors for IBS can be a past gastrointestinal infection, affecting up to 20% of patients with the so-called post-infectious form of the disease. This group also frequently experiences small intestinal bacterial overgrowth (SIBO), which is one manifestation of dysbiosis. Visceral hypersensitivity, one of the main factors of IBS, results from abnormal sensory processing and neurotransmission disorders, including excessive production of serotonin and substance P. On the other hand, impaired bile acid absorption can contribute to the diarrheal form of IBS.

Psychosocial factors also influence the functioning of the gut-brain axis. Psychological stress can exacerbate IBS symptoms, as well as affect mood and the nervous system's response to pain. Studies show that in 50–80% of patients, stress plays a significant role in triggering and worsening symptoms. Diet, especially one rich in fermentable carbohydrates and polyols, can further aggravate the condition of patients. [11,12,13]

Small Intestine Bacterial Overgrowth (SIBO) is a condition characterized by excessive bacterial growth in the small intestine, leading to gastrointestinal symptoms. SIBO is a state of dysbiosis in which the natural balance of microorganisms in this section of the digestive tract is disrupted. In a healthy small intestine, the number of bacteria is limited – in the proximal part of the jejunum, the normal level is less than 10^3 colony-forming units (CFU). However, in SIBO, the small intestine

becomes colonized by an excessive amount of aerobic and anaerobic bacteria, disrupting normal intestinal function.

Bacterial overgrowth in SIBO can be divided into two main types. In the case of overgrowth in the upper digestive tract, Gram-negative bacteria dominate, such as *Streptococcus viridans* and *Prevotella spp.*, originating from the oral cavity. In contrast, the lower digestive tract is predominantly colonized by bacteria such as *Escherichia coli*, *Klebsiella pneumoniae*, *Enterococcus spp.*, *Proteus mirabilis*, and *Clostridium spp.* Factors that promote the development of SIBO in the upper sections of the intestine include slowed intestinal motility caused by medications, hypochlorhydria resulting from chronic use of proton pump inhibitors (PPIs), or atrophic gastritis. In the lower sections, SIBO may result from motility disorders or anatomical changes, such as intestinal diverticula.

Physiological defense mechanisms, such as acidic gastric juice, bile, pancreatic enzymes, secretory IgA, proper intestinal peristalsis, and the ileocecal valve, prevent excessive colonization of the small intestine. Dysfunction of these mechanisms can lead to the development of SIBO.

Excessive bacterial growth in the small intestine leads to intense carbohydrate fermentation and gas production, resulting in discomfort, bloating, and abdominal pain. The diagnosis of SIBO relies on breath tests measuring gases such as hydrogen and methane, which are byproducts of this fermentation. SIBO symptoms are nonspecific and include abdominal pain, bloating, diarrhea, belching, excessive gas production, and indigestion. These symptoms can vary in frequency, severity, and duration. In more severe cases, vitamin deficiencies (including B12 and fat-soluble vitamins), iron deficiency anemia, steatorrhea, and even dehydration can occur. Patients may also experience fatigue and concentration problems, although these symptoms are not specific to SIBO.

SIBO often coexists with other conditions, such as diabetes, irritable bowel syndrome, diverticular disease, or long-term PPI use. It can also be accompanied by overgrowth of methane-producing archaea or carbon dioxide-producing fungi. Proper diagnosis of SIBO requires ruling out other serious causes of symptoms, such as weight loss, gastrointestinal bleeding, or chronic inflammatory conditions [14,15,16].

Carbohydrate malabsorption disorders often manifest as bloating, belching, abdominal pain, and excessive gas production. Fructose intolerance, resulting from its impaired absorption in the small intestine, is a problem that may be more common than previously thought. It is believed to

contribute to gastrointestinal discomfort, especially in children under the age of 9 and adults with irritable bowel syndrome (IBS). Increased fructose consumption in recent decades, mainly due to the popularity of products rich in this sugar, highlights the significance of this issue.

Fructose occurs in three forms: as a simple sugar, a component of sucrose (with glucose), and in fructans, which are not digested in the small intestine. Fructose absorption depends on the GLUT5 transporter, and the presence of glucose or galactose can enhance this process. Fructans, on the other hand, are fermented in the large intestine, leading to gas production, including hydrogen and methane, and may exacerbate symptoms such as bloating and intestinal discomfort. Treatment includes a diet limiting fructose and fructans, especially products with a higher proportion of fructose to glucose. In many cases, such a diet alleviates symptoms and improves the patients' quality of life. [17]

Lactose malabsorption in the small intestine causes symptoms such as abdominal pain, bloating, loose stools, and excessive gas production. Lactose intolerance is usually a permanent, inherited condition but may be transient, for example, after infection or damage to the intestinal mucosa. Proper diagnosis of this condition is crucial because symptoms can be effectively relieved with simple dietary changes, and an accurate diagnosis helps avoid unnecessary tests and treatments. The enzyme lactase is located in the brush border of enterocytes in the small intestine. It breaks down lactose from food into glucose and galactose, which are then transported across the cell membrane. For proper absorption, enzyme activity and adequate time for lactose passage through the mucosa of the jejunum are necessary. In the absence or deficiency of lactase (hypolactasia), unabsorbed sugars osmotically attract fluids into the intestinal lumen. In addition to increasing the volume and liquidity of intestinal contents, unabsorbed lactose reaching the colon undergoes fermentation by bacteria. This process results in gas production and the breakdown of lactose into monosaccharides, which cannot be absorbed by the colonic mucosa. This leads to an increase in osmotic pressure and further fluid attraction into the intestine. In individuals with lactase deficiency, some of the carbohydrates reaching the colon may be converted by bacteria into short-chain fatty acids and absorbed. However, the overall effect of lactose consumption is a significant increase in the volume of fluids and gases in the intestines. Hypolactasia occurs in three forms: primary, secondary (resulting from mucosal damage), and congenital (rare). The most common is primary adult hypolactasia. Lactase activity naturally declines in all mammals after the milk-feeding period. In humans, this decline varies according to ethnic background: from 2% in Northern Europe to nearly 100% in Asia and South America. It is speculated that hypolactasia is a natural condition, and the

maintenance of lactase activity in adulthood in some populations is an evolutionary adaptation linked to dairy consumption. [18,19]

Celiac disease is a genetic disorder caused by an abnormal immune response to gluten in the diet. This leads to damage to the intestinal villi, resulting in malabsorption disorders. Celiac disease mainly occurs in individuals with certain HLA class II proteins – DQ2 (90-95%) or DQ8 (5-10%). The villous atrophy results in a decrease in the activity of enzymes, such as disaccharidases, and a reduction in the number of APUD cells producing intestinal peptides like cholecystokinin and secretin. These changes in the villi can be reversed by following a gluten-free diet.

Celiac disease is triggered by gluten, a protein found in the grains of wheat, rye, barley, and, to a lesser extent, oats. Gluten is located in the walls of cereal vesicles, not in their husks. Gluten in various cereals, such as gliadin in wheat, secalin in rye, hordein in barley, and avenin in oats, has a toxic effect on the intestinal villi. The fraction of alpha gliadin is particularly harmful, as it is difficult to digest in the gastrointestinal tract. In healthy individuals, the intestinal barrier prevents the absorption of gluten, but in those with celiac disease, its permeability increases. After crossing the intestinal barrier, undigested gluten peptides are converted by transglutaminase into glutamic acid, allowing them to bind more strongly with HLA DQ2 and DQ8 particles. This activates T and B lymphocytes, which release cytokines, leading to damage to the villi, deepening of the intestinal crypts, and an increase in lymphocytic infiltration. Antibodies are also produced against endomysium, gliadin, reticulin, and tissue transglutaminase.

The clinical symptoms of celiac disease can vary depending on the age at which the disease manifests. There are classic forms (with gastrointestinal symptoms, such as abnormal stools, large abdomen, malnutrition) and atypical forms, where extra-intestinal symptoms predominate, such as growth disturbances, anemia, or osteoporosis. There is also a latent form, where symptoms are mild, but histological changes in the intestinal villi are present. Celiac disease can also take a latent form, which may remain asymptomatic for a long time, and its diagnosis is based on serological tests and biopsy. [20,21,22,23]

Intestinal Gases as an Indicator of Gastrointestinal Health

The release of gases from the gastrointestinal tract is a natural process in the human body. These gases are produced as a result of intestinal fermentation (digestion processes) and swallowing air during eating, drinking, or talking. One of the reasons why the release of gases indicates good

health is the ability to expel excess gases from the body. If these gases are not expelled, they can cause discomfort, abdominal pain, or bloating.

In a study conducted by Koide A. et al., the relationship between irritable bowel syndrome (IBS) and the volume of intestinal gases was investigated. The volume of intestinal gases was measured using X-rays of the abdominal cavity in 30 IBS patients and 30 healthy controls. A strong correlation was found between the volume of intestinal gases and the occurrence of IBS, with the average gas volume in IBS patients being significantly higher than in the control group [24].

The presence of a healthy bacterial flora in the intestines is crucial for digestion and metabolism. These bacteria assist in digestion by enabling the breakdown of indigestible or poorly digestible food components, such as resistant starch, oligosaccharides, mucins, nitrogen compounds, and lipids. One of the byproducts of their action is the production of gases [25]. If the intestinal flora is well-balanced and fermentation processes occur properly, the expulsion of gases is a normal symptom. The lungs play an important role in removing volatile substances from the body. Studies have shown that exhaled air contains nearly 2000 different chemical compounds [26].

Hydrogen breath tests (HBT) are widely used, safe, and non-invasive diagnostic methods in gastroenterology. The test is based on the assumption that, under normal physiological conditions, hydrogen (H2) is either absent or present in very low concentrations in exhaled air. The production of H2 occurs mainly as a result of the activity of bacteria that colonize the colon [27]. In natural conditions, the bacterial flora of the gastrointestinal tract begins to colonize the intestines shortly after birth, and its composition remains relatively unchanged throughout life [28].

In a study conducted by Sachdeva S. et al., the role of small intestinal bacterial overgrowth (SIBO) in the pathogenesis of IBS was analyzed. The study examined 59 IBS patients and 37 healthy controls. SIBO was assessed using a breath test with 100 g of glucose after an overnight fast. The concentration of hydrogen and methane in exhaled air was measured at the beginning of the test and every 15 minutes after glucose administration for a total of 3 hours. Under physiological conditions, hydrogen and methane are not present in exhaled air. If present, it indicates that they were produced by bacteria colonizing the colon. In this situation, hydrogen is released, and in some cases, methane as well. These compounds dissolve into the blood vessels of the mucous membrane and are transported to the lungs, where their presence can be detected in exhaled air [6]. A constant increase in hydrogen or methane concentrations in exhaled air greater than 12 ppm above baseline was

considered diagnostic for SIBO. The overgrowth of intestinal bacteria occurred more frequently in IBS patients compared to the control group. Additionally, bloating was found to be a predictor of SIBO in IBS patients [29].

The expulsion of gases can also indicate proper intestinal motility, i.e., the ability of the intestines to contract and move food contents. Regular motility is essential for proper digestion and the elimination of waste products. In a study conducted by Harder H. et al., an attempt was made to correlate intestinal motility with negative sensations from the presence of gases in different parts of the intestines. The study involved 14 healthy volunteers. Each participant was given a gas infusion (12 ml/min) into the jejunum or rectum for one hour while the outflow of gases from the rectum was blocked, and then the gas clearance was measured during one hour of free evacuation from the rectum. It was observed that the same amount of trapped gas (720 ml) caused significantly more negative abdominal symptoms when infused into the jejunum compared to the rectum. The experiment showed that most healthy individuals tolerate large gas loads well due to rapid passage and gas evacuation. However, patients with functional bowel disorders, such as IBS or functional bloating, may experience significant discomfort due to impaired gas expulsion, even if the gas volume is normal [30].

Summary

Research suggests that the quantity and type of intestinal gases may be linked to certain bowel disorders, such as irritable bowel syndrome or small intestinal bacterial overgrowth. However, further studies are necessary to better understand these relationships.

In summary, the release of intestinal gases is a natural process in the human body and can indicate good health. If gas expulsion is excessively frequent or accompanied by severe abdominal pain, bloating, diarrhea, or constipation, it may suggest the presence of an intestinal disorder. An unpleasant odor of gases, especially if persistent and accompanied by other symptoms, may be associated with digestive disorders, food intolerance, or intestinal infections.

Understanding the composition of intestinal gases, the factors influencing their production, and the regulation of gut-brain centers is essential for gastrointestinal health and the overall condition of the body.

Disclosures

Author's contribution

Conceptualization: Klaudia Anna Pawełek

Methodology: Patrycja Kinga Marta

Software: Filip Maciej Huzarski

Check: Gabriela Monika Ferfecka, Natalia Morawiecka

Formal analysis: Magdalena Rosa-Bończak

Investigation: Agata Ossolińska

Resources: Weronika Kłosowicz

Data curation: Oliver Carlton

Writing - rough preparation: Klaudia Pawełek, Lucyna Stolarska

Writing - review and editing: Natalia Morawiecka

Visualization : Filip Maciej Huzarski

Supervision: Magdalena Rosa-Bończak, Agata Ossolińska

Project administration: Klaudia Pawełek

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