RESEARCH PAPER

Activated Charcoal for Gastrointestinal Gas and Bloating: Mechanisms, Clinical Evidence, and Safety Considerations- A Narrative Review

Diksha Shukla[1*], Kishalay Maity[2], Monisankar Pramanik[3], Kajal Sharma[4], Dr. Daisy sharma[5], Mainka Coguide[6*]

[1]CT group of institutions phase 2 urban state ,pratappura road, Jalandhar, Punjab -144020.*

[2]Arni school of Pharmacy,Arni University kangra, Himachal Pradesh.

[3]Rashbehari Pharmaceuticals Institute, Patanda, Panskura, District - Purba Medinipur, West Bengal, 721139.

[4]Lyallpur khalsa college technical campus, jalandhar,Punjab.

[5]CT group of institutions phase 2 urban state ,pratappura road, Jalandhar, Punjab -144020.

[6]CT group of institutions phase 2 urban state ,pratappura road, Jalandhar, Punjab -144020.

ABSTRACT

Excessive gas, flatulence, and abdominal bloating are frequent gastrointestinal problems that can significantly affect the quality of life. These symptoms are common in functional gastrointestinal diseases such as irritable bowel syndrome (IBS). The Source of intestinal gas are mainly the fermentation of undigested carbohydrates by bacteria and swallowing of air (aerophagia), which causes abdominal bloating, discomfort, and alteration of gastrointestinal motility. Activates charcoal is a porous carbon-based adsorbent with a large surface area that has been extensively investigated for the absorption of gases, toxins, and chemicals in the gastrointestinal tract. It remains in the gastrointestinal lumen, is neither absorbed nor metabolized, and is excreted in the feces. Also, activated charcoal has a high affinity for a number of drugs and chemicals, which inhibits absorption. While there are indications that activated charcoal has adsorptive properties useful in treating gas and bloating, more clinical studies are required to determine a standardized dose, long-term safety, and clinical effectiveness. The purpose of this narrative review is to assess the adsorptive ability, action, clinical evidence and safety of using AC for gastrointestinal gas and bloating.

Keywords: Activated charcoal; bloating; adsorption; intestinal gas; gastrointestinal gas; breath tests

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INTRODUCTION

Activated carbon, also called medicinal charcoal, is a carbonaceous adsorbent which contains very irregular and poorly energetic pores in terms of their energy and activity due to the destruction of the crystalline structure during its production [1]. Both the surface and internal surface of the activated charcoal is covered with these pores. Activated carbon has the ability to contain the maximum number of adsorbents compared to other types of carbon and surface area of carbon can vary between 300 m2/g to 5000 m2/g [2]. There are two types of activated charcoal: powdered and granular. The micron-sized activated charcoal powder was prepared by grinding the granular activated charcoal of millimeter size to powder. The adsorption kinetics and total adsorption capacity of micro-sized particles is higher than that of the granular particles. Typically, granular activated carbons have between 0.2 and 5 mm particle size. These are also easier to work with and will stay in place longer than powders [3]. The common charcoal is produced from peat, coal, wood, coconut shells or petroleum. The "medicinal" activated charcoal is processed to comply with the exact sorption and purity requirements set forth by the Pharmacopoeia. Thermal activation is a process of pyrolysis of carbonaceous material with controlled atmosphere that produce numerous “pores” or voids within the charcoal. These pores allow the chemical compounds to be adsorbed on the surface of the activated charcoal. Activated charcoal's physicochemical properties make it an ideal material for use in gastrointestinal applications, and specifically as an aid to the reduction of intestinal gas and bloating. The symptoms are typical for functional gastrointestinal disorders, including irritable bowel syndrome (IBS) [4]. Activated charcoal has a large surface area with porous structure and is able to bind and prevent the absorption of chemical substances. Activated charcoal side effects include constipation and stools that are black. Due to its toxin-adsorbing properties, activated charcoal has been suggested for treating a variety of health conditions. One of these benefits is that it helps with the

elimination of undigested drugs and pollutants which helps with renal health. Activated charcoal has been demonstrated to be effective in eliminating toxins from the urea digestible compound, which is the main one. The adsorptive properties of activated charcoal in the lumen of the intestine aid in the elimination of trapped gases and fluids.

Table1: Activated Charcoal Properties

Property	Description	Scientific basis
High Surface Area	Extremely large surface area (500–1500 m^2/g or more	Maximizes the density of the available active sites for molecular adsorption . [5]
Porous Structure	Contains micro-,meso and macropores	Enables adsorption of molecules of different sizes[5]
Strong Adsorptive Capacity	Binds gases, toxins, and chemicals	It is driven by physical forces (not chemical bonding) . [6]
Adsorption Mechanism	Surface-based interaction of molecules	Involves vander Waalsforces and weakintermolecular interactions .[6]
Chemical Inertness	Stable and non-reactive material	It does not undergo chemical changes in the body. [7]
Non-Absorbable	Not absorbed into bloodstream	It acts locally in the gastrointestinal tract. [7]

MECHANISM OF ACTION

In the digestive system, activated charcoal will bind with toxins, thus blocking the toxin from entering the blood stream. Poisons can only be absorbed directly by the activated charcoal when the poison is dissolved in liquid form. Activated charcoal, when taken orally, does not alter and is not absorbed through the gastrointestinal (GI) lumen either by enterohepatic recirculation, entero-enteric recirculation through active secretion, passive diffusion or if not absorbed from the gastrointestinal lumen. Activated charcoal is more effective at binding toxins when they are not ionised. Polar and water-soluble substances do not generally occur in the form of adsorption. Pharmacodynamics[8] is the reason that nonpolar, poorly watersoluble organic poisons are the most easily absorbed by activated charcoal.Activated charcoal has a decreased absorption in the systemic circulation with acetaminophen, aspirin, barbiturates, tricyclic antidepressants, theophylline, phenytoin and most inorganic and organic compounds [9].Alcohols, metals (like iron and lithium), electrolytes (magnesium, potassium or sodium), acids or alkalis are not efficiently adsorbed by activated charcoal due to their polarity. Superheated, high-surface-area, porous organic material particles are used to make activated charcoal, a powerful adsorbent powder. Due to the large surface area of the activated charcoal, a carbon-based network is formed that adsorbs chemicals rapidly. The use of activated charcoal (OAC) to reduce gastrointestinal lumen absorption of chemicals has been known for approximately 200 years, as it is not systemically absorbed, but rather binds to chemicals in the gastrointestinal lumen [10]. OAC is also used as an 'over the counter' tooth whitener and to relieve symptoms of gas. The doses used in clinical practice are generally between 12-100 grams. The OAC power is mixed with water to make a slurry that can be sometimes flavored with juice. Side effects of OAC are very uncommon, and their first symptom is usually gastrointestinal problems such as nausea, vomiting and fullness [10]. Two studies suggest that OAC protects against the gut microbiota by binding to and preventing the absorption of antibiotics in the lower gi tract [11]. This has a large surface area, and a network of carbon molecules is included which are functionalized with groups (such as carbonyl groups and hydroxyl groups) that are able to bind drugs in minutes after contact, preventing gastrointestinal absorption and toxicity. Activated charcoal has been utilized for treating a number of ailments, such as anthrax, vertigo and epilepsy. Recently, activated charcoal has been used as an antidote against many toxins, and as an anti-gas agent. Activated charcoal over extended periods of time has been found to be quite safe in large doses [12].

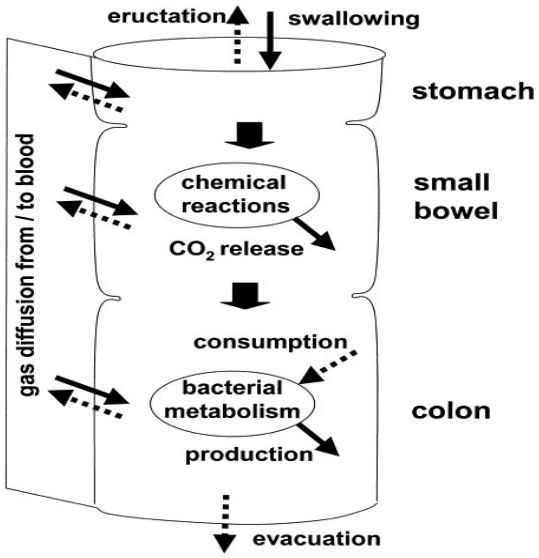


Figure 1 depicts the intestinal gas metabolism. Swallowing, chemical reactions, blood diffusion, and bacterial fermentation contribute to gas input. Gas output occurs through eructation, absorption, bacterial consumption, and anal evacuation. Gas transit determines the time of exposure to gas diffusion across the gut-blood barrier and bacterial consumption, potentially influencing intestinal gas volume, composition, and tolerance.

PHARMACOKINETICS

- **Absorption:** Activated charcoal is not absorbed by the gastrointestinal tract.
- **Elimination:** Activated charcoal is excreted in the feces.

1. Physiochemical Properties and Pore Dynamics

The therapeutic efficacy of activated charcoal (AC) is a result from its complex hierarchical pore network, which is often inhomogeneous owing to the breakdown of its crystalline structure during production.

- **Pore Classification:** According to International Union of Pure and Applied Chemistry (IUPAC), activated carbon can be classified as micropores (< 2nm), mesopores (2 – 50 nm) and macropores (> 50 nm). [13]
- **Surface Chemistry**: The presence of specific acidic and basic sites, such as carbonyl and hydroxyl functional groups, is critical for the sequestration of polar gaseous molecules and intraluminal toxins.[14]
- **Physical Forces**: The adsorption process is driven by physical forces, specifically vander-waals forces and weak intermolecular interactions, rather than chemical bonding.

2. Pharmacological Mechanism and Intraluminal Sequestration

- **Local Action:** Activated charcoal is not absorbed into systemic circulation. Remaining confined to the gastrointestinal lumen, it is subsequently eliminated via the feces.
- **Intraluminal Sequestration**: Activated Charcoal exerts its effect locally within the gastrointestinal tract without systemic absorption. By adsorbing gaseous molecules onto its high surface area network, it mitigates intraluminal pressure and bloating by lowering the total amount of free gas in the stomach.
- **Odor Neutralization**: It is particularly effective at absorbing volatile sulfur compounds (VSCs), which are responsible for the unpleasant smell of flatulence.[15]

3. Clinical Efficacy and Modern Evidence

The clinical role of AC has been clarified by recent meta-analyses and prospective studies (2021–2026):

- **Symptom Relief:** In a 2024 placebo-controlled study, activated charcoal plus simethicone was shown to be significantly superior to placebo in decreasing abdominal fullness, bloating and indigestion after 90 days of treatment ($P<0.05$) [16].
- **IBS Management:** AST-120 (spherical carbon adsorbent) is made up of mostly composed of carbon (about 96%) and includes spherical, black particles that are about 0.2–0.4 mm in size diameter with distinct pore size and nanopore volume distribution. This is a widespread porosity which provides a good and large surface area on which the binding is selective. It is thought that the AST-120's clinical utility may be in the ability to bind low molecules in the intestine's lumen in the lower digestive tract. The adsorptive profile of the AST- 120 has been shown to include neuroactive substances such as serotonin, Tyramine, tryptamine, histamine, caffeine and Toll-like receptor (TLR) ligands lipopolysaccharide (LPS) as well as bacterial adjuvants (fMLP, or formyl-methionyl-leucyl-cholic acid) and bile acids [17].

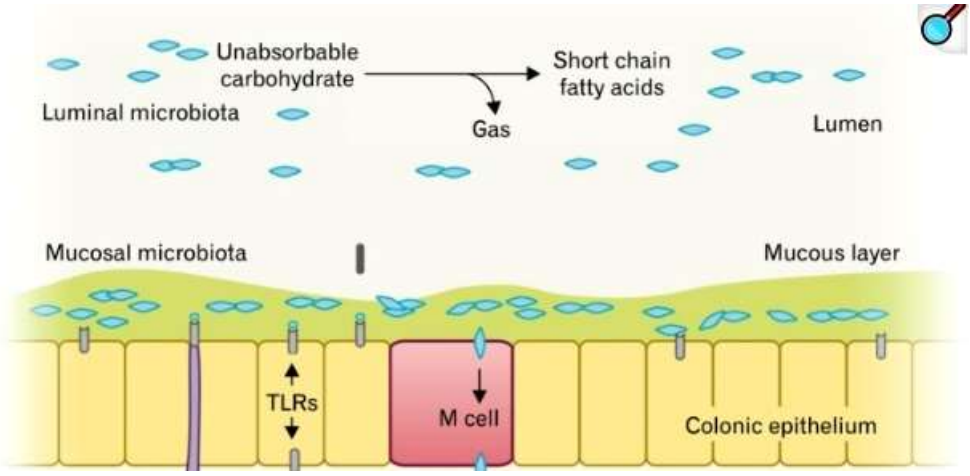
4. Latest Safety Interaction Guideline

For a wide range of harmful substances, activated charcoal is a nonspecific and nonabsorbable adsorbent.The interactions with a vast network of pores on the surface of activated charcoal, where binding takes place, are governed by simple physical chemical properties. The bound poison and activated charcoal complex is expelled in the stool. Since activated charcoal stops absorption throughout the gastrointestinal tract rather than merely trying to empty the stomach, it is conceptually superior to orogastric lavage. However, due to a few significant drawbacks, activated charcoal is not a cure-all. First, not all toxins are absorbed by activated charcoal. Iron and lithium are notable exceptions. [18]. For drugs that are well adsorbed, it typically takes ten times as much activated charcoal as the drug to ensure nearly total binding. [19].

PATHOGENESIS OF GAS AND BLOATING

Abnormal Gut Microbiota

There are over 500 distinct species of GI microbiota in adults, the majority of which are obligatory anaerobes and are crucial to the host immune system[20]. Because only a small percentage of these organisms can be cultured, little is known about the functions of different microbes in the GI tract . However, studies conducted over the past few decades have revealed that stool samples from IBS patients have altered colonic flora [21]. Parkes et al.[22] proposed that the luminal bacteria and the mucosa-associated bacteria are two ecosystems that comprise the GI microbiota (Fig. 2). By fermenting carbohydrates and producing gas, luminal microbiota, which make up the majority of the GI tract flora, contribute significantly to bloating and flatulence in IBS[22].



Gut homeostasis and the luminal and mucosal colonic microbiota. Parkes et al. [22] was modified.

SMALL INTESTINAL BACTERIAL OVERGROWTH

SIBO is a condition characterized by an overgrowth of bacteria in the small intestine. In patients who complain of bloating, there have been reports of increased gas production due to bacterial fermentation because of small intestinal bacterial overgrowth (SIBO). The idea that SIBO could be a significant pathogenesis of IBS was established by Pimentel et al.[23].Additionally, several studies found that taking antibiotics significantly improve symptoms such as bloating or abdominal pain[24]. Other studies have not confirmed these results, however; [25] reported a slight increase in numbers of bacteria cultured from the small intestine of IBS patients and no difference in H2 level in IBS patients' breath compared with non-IBS patients [26]. In addition, the lactulose breath test was considered to be an unreliable indicator of the relationship between bacterial overgrowth and IBS, and there was no association with bacterial alteration and symptom pattern. In another study, breath hydrogen concentration wasn't significantly different between IBS and control groups, and wasn't correlated with pain ratings in other patients with IBS due to the lack of objective diagnostic measurements and inconsistent data. [27]

ALTERED GUT MOTILITY

Moreover, loperamide, a drug that is known to reduce transit, induced constipation in normal volunteers, causing bloating in a conventional experiment, in IBS-C patients[30]. Rather than bloating, IBS-C patients with delayed orocecal and colonic transits have recently exhibited abdominal distention. [31]. Although some motor abnormalities have been documented in Asian IBS patients, such as delayed gastric emptying and slow intestinal transit, it remains controversial whether these abnormalities are exclusive to Asian IBS patients. Moreover, there are few data that correlate IBS symptoms with gut motility changes. A recent study found that changes in colon transit didn't really affect IBS symptoms of bloating and pain. [33]

FOOD INTOLERANCE AND CARBOHYDRATE MALABSORPTION

There is a general belief that diet can be a cause of abdominal symptoms; some attempts have been made to try and link food with symptoms associated with IBS. Overload of fibre has long been suspected of exacerbating IBS symptoms, through a reduction in small bowel motility or intraluminal bulking [34]. Also, increased lactose intolerance can be a factor when developing symptoms in people with IBS. Disaccharides are converted to monosaccharides in the small intestine by enzymes in the intestinal lumen, which are absorbed. If this is not the case, the disaccharide will be delivered to the colon where it's metabolized by the bacteria into short-chain carbonic acids and gases. So it could cause the symptom of bloating in those suffering with IBS who have malabsorbed the lactose [35]. In addition, a new hypothesis is suggested: high amounts of delivery of FODMAPs (fermentable oligo-, di-, and monosaccharides and polyols) to the small intestinal and colon can lead to gastrointestinal symptoms. FODMAPs are small molecules which are osmotically active and ferment much quicker than long-chain carbohydrates [36]. A high FODMAP diet has been demonstrated to trigger prolonged production of hydrogen in the gut, colonic distention from fermentation, increased fluid delivery to the colon from osmotic load within the gut lumen and induce GI symptoms, in an indirect way, due to increased bacterial proliferation, particularly of bifidobacterium. [37]

Differential Diagnosis of Gas and Bloating

A Clinical Reference Guide

Category	Condition	Key Features
Functional Disorders	Irritable Bowel Syndrome (IBS)	Recurrent abdominal pain, altered bowel habits, bloating — no structural cause
	Functional Bloating	Bloating without pain or altered motility; no identifiable organic cause
	Functional Dyspepsia	Upper abdominal discomfort, early satiety, belching
Dietary & Lifestyle	Excessive Gas-Producing Foods	Beans, lentils, cruciferous vegetables, fizzy drinks
	Aerophagia	Excessive air swallowing, often linked to eating quickly or anxiety
	Lactose Intolerance	Bloating, diarrhoea, and cramps after dairy consumption
Malabsorption	Coeliac Disease	Chronic bloating, diarrhoea, fatigue — triggered by gluten
	Small Intestinal Bacterial Overgrowth (SIBO)	Excess bacteria in small intestine causing gas, bloating, and diarrhoea
	Fructose/Sorbitol Malabsorption	Bloating and loose stools after consuming fruit or artificial sweeteners
Structural Causes	Intestinal Obstruction	Severe bloating, vomiting, inability to pass gas — medical emergency
	Hernia	Localised abdominal bulge with discomfort and bloating
	Adhesions	Post-surgical scarring causing trapped gas and pain
Motility Disorders	Gastroparesis	Delayed stomach emptying causing bloating, nausea, and early fullness
	Slow Transit Constipation	Infrequent bowel movements leading to gas build-up and distension
Inflammatory Conditions	Inflammatory Bowel Disease (IBD)	Crohn's or Ulcerative Colitis — bloating alongside bloody stool, weight loss
	Diverticular Disease	Left-sided pain, bloating, altered bowel habits in older adults
Gynaecological (Females)	Endometriosis	Cyclical bloating linked to menstrual cycle, pelvic pain
	Ovarian Cysts	Abdominal distension, pelvic discomfort
Systemic Conditions	Hypothyroidism	Slow gut motility causing constipation and bloating
	Diabetes (Autonomic Neuropathy)	Nerve damage slowing digestion, leading to bloating and fullness

EVALUATION OF GAS AND BLOATING

Breath Testing Hydrogen breath analysis can be used to establish that gaseous symptoms are due to carbohydrate intolerance. These methods are based on the measurement of the hydrogen generated by bacteria in the lumens when they break down the previously consumed test substrates.On the contrary, human tissues do not produce hydrogen when they expend carbohydrates. The person breathes into the machine at the beginning and 2 hours after drinking a sugar solution they are thought to malabsorb/maldigest. Within 120 minutes of consuming lactose, hydrogen increases of more than 20 parts per million can differentiate between lactase shortage and lactase sufficiency with 90% sensitivity.[38] Sucrose hydrogen breath analysis is used to diagnose sucrase-isomaltase deficiency [39]. The diagnosis of sorbitol or fructose malabsorption has been made by hydrogen breath analysis. For the hydrogen evaluation period, however, the period of time to assess for complex carbohydrate

maldigestion can be extended to 10 hours. Also, hydrogen breath tests can establish if there is bacterial overgrowth in the small intestine. If the hydrogen concentration is raised above normal levels, or the hydrogen levels raised in the first 30 minutes of eating, this can be considered a sign of overgrowth. Breath testing has been used for detecting bacterial overgrowth and has been shown to have 60-90% sensitivity and specificity when using lactulose and rice as the substrate [40]. However, the sensitivity of these approaches range from 17 to 33% for the detection of overgrowth; false negative results can occur in people who don't have any hydrogen-producing bacteria, and false positive results can occur in people with fast small bowel transit. In other centers, breath 14CO2 or 13CO2 is measured using 14C or 13C-labeled substrates, but this is only possible with special facilities [41]. If the diagnosis is questionable, bacterial overgrowth is confirmed by the presence of bacterial counts larger than 105 CFU/mL on quantitative culture of duodenal or jejunal secretions.[40] Finally, hydrogen breath testing with lactulose was used to estimate orocecal transit in patients with suspected intestinal pseudo-obstruction. The time taken for transit is recorded from the time of ingestion until an increase in breath hydrogen is detected (due to the colonic bacterial metabolism of the substrate). Lactulose stimulates the transit of contents in the small intestine.[42]

Table2:Comparison of Activated Charcoal with Alternative Treatments For Flatulence

Treatment	Mechanism of action	Clinical effectiveness	Advantages	Limitations	Reference
Activated Charcoal	Adsorption of gases and poisons through porous surfaces	Moderate; inconsistent research findings	Inexpensive,widely accessible,and capable of absorbing poisons	Non-selective;capable of binding nutrients and medications	**[43]**
Simethicone	Lowers gas bubble surface tension(anti-foaming)	Effective in relieving symptoms	Safe,quick-acting,and without systemic absorption	Does not reduce gas production	**[44]**
Probiotics	Alters the gut microbiota	Effects that vary and depend on strain	Enhances intestinal homeostasis and has long term advantages	Results from different studies are inconsistent	**[45]**
Rifaximin	A non-absorbable Antibiotic that lowers intestinal bacteria	Effective for bloating caused by IBS	Focuses on the underlying bacterial cause	Costly;chance ofrecurrence	**[46]**
Fiber Supplements	It enhances gut healthand bowel movements	Various outcomes based on the type	Helps constipation-related bloating	May exacerbate gas in gas patients	**[47]**

CONCLUSION

Activated charcoal is a well-known and commonly used adsorbent with promising therapeutic applications in the treatment of gastrointestinal gas and bloating. Its highly porous structure, large surface area, and strong physical adsorption ability allow for effective binding of intraluminal gases, poisons, and chemicals, lowering belly discomfort and enhancing digestive health. The mechanism of action appears to be primarily intestinal and is not taken up systemically, making the use of activated charcoal relatively safe in the short term. Current clinical data indicate that activated charcoal may be more effective in symptom relief for individuals with gastrointestinal symptoms of bloating or with functional gastrointestinal disease (e.g., irritable bowel syndrome) and that its use with drugs like simethicone may be useful. Although it has benefits, the therapeutic effectiveness of activated charcoal is still not clear, with conflicting results from investigations. Its non-selective adsorption can also interfere with nutritional absorption and co-administered drugs, emphasizing the importance of exercising caution and relying on evidence. In addition, safety concerns such as drug-drug interactions and contraindications must be carefully considered in clinical practice.

FUTURE DIRECTIONS

Future research should focus on large-scale, randomised controlled studies to create standardized dose regimens and confirm long-term safety and efficacy. Advanced formulations, such as modified or surface-engineered activated charcoal with specific adsorption capabilities, could aid in reducing drug-nutrient interactions. Furthermore, integration with new drug delivery methods and combination therapies may improve therapeutic outcomes.

Exploration of its involvement in microbiome modification, personalized medicine techniques, and targeted gastrointestinal drugs present potential opportunities. Standardization of clinical evaluation tools, such as breath tests and symptom rating systems, is required to produce more uniform and comparable data from studies.

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