RESEARCH PAPER

Therapeutic Mechanisms of Carbon Quantum Dots in the Treatment of Recurrent Gastrointestinal Ulcerative Colitis Disorders

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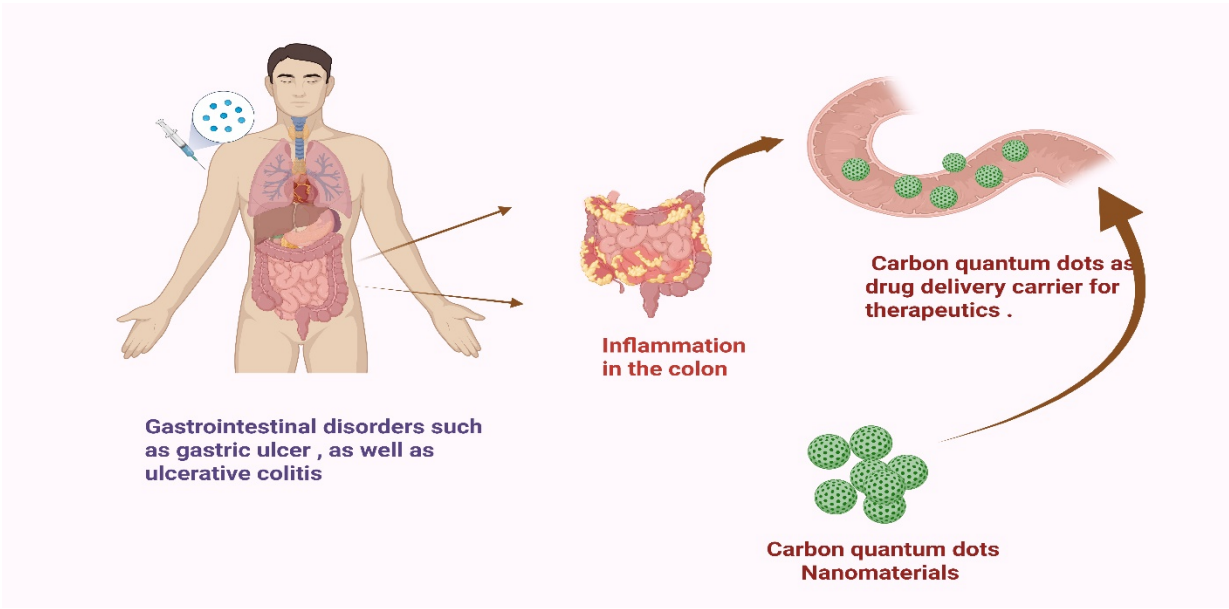
Graphical Abstract



ABSTRACT

Gastrointestinal disorders such as gastric ulcers (GU) and ulcerative colitis (UC) are well recognized for their tendency to recur after therapy, posing a significant problem in modern medicine. Carbon quantum dots (CQDs) have high-performing antioxidant and anti-inflammatory properties, making them particularly interesting research substrates for the study of gastrointestinal and colorectal disorders. This review focuses on the synthesis methods and properties of CQDs and simultaneously analyzes their mechanisms in ulcerative colitis and their therapeutic potential. The review initially highlights the importance of green synthesis and the use of herbal medicine in producing biologically derived CQDs. Subsequently, focusing specifically on the induction mechanisms of UC and GU, the following discussion aims to improve understanding of CQD's therapeutic mechanisms. In conclusion, the elucidation of the molecular mechanisms by which nanostructured CQDs protect the digestive tract and improve diseases such as gastric ulcers and ulcerative colitis has been accomplished and will therefore lead to a promising future for the treatment of gastrointestinal disorders.

Keywords: Carbon quantum dots, ulcerative colitis, gastric ulcers, green synthesis, antioxidant, anti-inflammatory, gastrointestinal disorders, nanotherapeutics

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Introduction

Gastrointestinal (GI) disorders include diseases of the oesophagus, stomach, small intestine, colon, and rectum. It is characterised by health issues and clinical signs of frequent diarrhoea, abdominal pain, bloating, nausea, dyspepsia, and constipation. Crohn's disease (CD) and ulcerative colitis (UC) are the two major types of inflammatory bowel disease (IBD) [1]. CD involves the entire gastrointestinal tract, spreading from the oral cavity to the anal canal. The final stretch of the ileum is affected in about 90% of patients with CD [1]. Gastric ulcers (GU) are a very common gastrointestinal-related disease triggered by a variety of pathogens, chronic tobacco use, long-term use of steroids and anti–inflammatory drugs, excess ethanol consumption, and most importantly, *Helicobacter pylori* infection. All these factors exert deleterious effects upon the gastric mucosal epithelium, with a disrupted epithelial barrier, the provocation of oxidative stress, and increased pro-inflammatory cytokine production converging to the clinical manifestation of GU. The pathogenesis of ulcerative colitis is characterized by a disruption of the regulation of intestinal immune responses and a break in the intestinal epithelial barrier. Intestinal epithelial cells form the major physical defense of the gastrointestinal tract, a role that relies on both intercellular and intracellular tight-junction proteins such as occludin, claudin-1, and zonula occludens [1]. Conventional pharmacotherapy for IBD includes anti-inflammatory drugs such as 5-aminosalicylic acid and corticosteroids, as well as immunosuppressive drugs such as azathioprine, 6-mercaptopurine, methotrexate, cyclosporine A, and tacrolimus. Additionally, antibodies against interleukin IL-12/IL-23 and adhesion molecules, such as ustekinumab and natalizumab, have been suggested as therapeutic options for IBD [2]. However, there is a need for new therapeutic strategies, as many patients do not respond to currently approved medications, including tumour necrosis factor (TNF antagonists and vedolizumab (9-11). Some of the formulations release the drug continuously along the gastrointestinal tract before the delivery system reaches the colon, thereby reducing drug availability and increasing the risk of systemic adverse effects. IBD predominantly affects the colon, and thus colon-specific drug delivery systems have gained significant interest in the context of IBD therapy [3]. These deficiencies highlight the need for novel drug delivery systems (DDS) that optimally release drugs at the site of colon inflammation without affecting normal tissues, thereby reducing drug-related side effects. The advent of new technologies has opened up therapeutic prospects for IBD treatment [4]. Specifically, nanomaterials have great potential for targeted drug delivery to specific sites of colonic inflammation via different mechanisms. These carbon-based nanostructures are categorised according to their basic carbon structure, surface functional groups, and unusual physicochemical properties. They included a number of variants such as (CQDs), graphene quantum dots (GQDs), carbon nano dots (CNDs), and carbon polymer dots (CPDs) [4]. Recent research has focused on the use of raw materials and plant extracts for the synthesis of carbon Quantum dots derived from herbal medicines (HM-CQDs) due to their minimal side effects.[5] These HM-CQDs exhibit significant medicinal effects and biological activities, which can be attributed to the active ingredients and chemical constituents of plants, such as polysaccharides, phenolic compounds, and alkaloids. Nevertheless, there are still challenges that can be linked to partial understanding of active constituents, synergistic interactions between extract constituents, instability of extracts, toxicity of solvents and poor solubility of bioactive compounds. All these issues are to be taken care of to support the development of definite products or novel formulations arising from medicinal plants [5]. HM-CQDs based drug delivery systems (DDS) have multiple important advantages, such as high local drug concentrations, maintaining pharmacological activity and therefore maximising therapeutic and curative activity, ensuring target drug delivery and minimize systemic adverse effects. The article presents a summary of modern and emerging systems of drug administration based on CQD for colon-targeted drug carrier and the treatment of ulcerative colitis (UC).

1. Methods for the synthesis of carbon quantum dots.

The synthesis of carbon quantum dots (CQDs) is currently under investigation because the mechanisms underlying their formation remain poorly understood. In this context, various molecular precursors, such as L-arginine, L-tyrosine, biotin, glutathione, citric acid, polyethyleneimine, and ammonium citrate, have been used as carbon sources to understand the pathways for CQD generation. An additional strategy to optimise the optical and therapeutic properties of CQDs is to dope them with heteroatoms or metallic elements.

Furthermore, larger carbon materials, such as activated carbon, graphene, carbon black, and graphite powder, can be used as carbon sources for CQD synthesis via thermal decomposition.[6]. The environmentally benign synthesis of CQD from biological and natural sources, especially from medicinal plants and agricultural sources, is of great significance. Moreover, medicinal plants, as naturally occurring organic precursors, are often used to prepare such medical nanoparticles. Over the years, numerous scholarly investigations have highlighted the remarkable therapeutic potential of medicinal plants in the treatment of a spectrum of pathologies, from cancers to gastric ulcers, IBD, and diabetes. Researchers faced difficulties due to limited understanding of the active constituents, synergistic effects among the components of the extract, low stability, toxicity issues associated with the solvent, and the low solubility of bioactive compounds, hindering the creation of stable end products and the formulation of innovative therapeutics based on medicinal plants. Recently, researchers have attempted to use more raw materials and plant extracts to synthesize HM-CQD [7]. These HM-CQDs exhibit interesting properties in medicinal and biological contexts owing to the presence of bioactive phytochemicals in plant materials. These constituents include polysaccharides, phenolic compounds, and alkaloids". Nevertheless, the structural, physicochemical, and biological characteristics of HM-CQDs vary widely across herbal medications. This heterogeneity stems from the peculiar phytochemical profiles of each plant species, which possess characteristic functional groups that can be detected on the surfaces of the obtained quantum nanoparticles after the carbonization process. Functional groups have a major impact on the diverse features of CQDs, including particle size, solubility in pure or complex systems, the chemical and optical properties of hydrocarbon-metalated CQDs, and biological and medicinal functionality. It is interesting to note that using plant materials as carbon precursors for medicinal applications offers additional advantages, such as increased accessibility, cost-effectiveness, safety, and an inherently non-toxic profile [6]

2. Synthesizing standard Methods of carbon quantum dots

The preparation of CQDs generally involves two methodologies: "top-down" and "bottom-up" approaches. The "top-down" approach involves breaking down large precursor molecules of graphene and graphite into nanoscale materials and requires acid treatments, hazardous chemicals, and elaborate machinery. On the other hand, the "bottom-up" method yields CQDs with uniform particle dimensions and high quantum efficiency by using precursors of natural carbon of suitable size, thereby providing a cost-effective, straightforward procedure [8, 9].

2.1 Hydrothermal and solvothermal methods

Hydrothermal synthesis is one of the most commonly used methods for synthesizing CQDs. This approach offers several significant advantages, including the use of double-distilled water as the solvent, which improves environmental sustainability. CQDs fabricated through this approach have various surface functional groups, including hydroxyl, amine, and carboxyl groups [10]. These features account for their high water solubility. Hydrothermal and solvothermal approaches have become popular for CQD preparation due to their simple preparation procedures, few experimental requirements, and ease of achieving high quantum yields [9].

2.2 Pyrolysis method

The most widely recognised technique for producing CQDs in vacuum synthesis is pyrolysis. During this procedure, a plant-based or carbonaceous precursor is first placed on a forerunner and later heated in a muffle furnace until it is converted to charcoal. This method is especially suitable for hard, woody plant components, such as roots and seeds. However, a wide range of rich carbon precursors, such as biomass, organic compounds, or polymers, can generally be used. Moreover, parameters such as pyrolysis temperature, heating rate, reaction time, and the presence of impurities significantly influence the properties of the resulting CQDs [11].

2.3 Microwave radiation method

Microwave-based synthesis is one of the fastest and cheapest methods for generating CQDs. This strategy involved exposing the mixture of carbon-based precursors to electromagnetic waves with wavelengths ranging from 1 nanometer to 1 meter, thereby promoting efficient carbonization and defect engineering in the quantum dots. The benefits of microwave-assisted synthesis include uniform heating, rapid and efficient processing, and the ability to achieve CQD synthesis with controllable fluorescence properties [12].

2.4 Herbal medicine-based carbon quantum dots synthesis

After analyzing all reported herbal precursors of CQD, it was found that the majority are medicinal plants, such as flowers, roots, seeds, fruits, and other plant parts, used

for synthesis. Herbal plants produce a range of bioactive and secondary metabolites with potential as therapeutic agents for the disease [7].

3. Properties of carbon quantum dots synthesis

The size of nanoparticles is of utmost importance in determining their unique physical and chemical properties, which are necessary for targeting specific sites to cure disease. As CQD dimensions shrink, their properties change due to size-dependent behavior. Quantum confinement effects are related to the dual nature of wave-particle matter at the nanoscale [13]. Since the size of the CQD is reduced below the Bohr exciton radius, there is spatial separation between an excited electron and its corresponding hole, which confines the electronic properties and results in the formation of different energy levels. Moreover, downscaling of CQD to the nanometer regime strongly increases the surface-to-volume ratio, thereby increasing their bandgap and shifting their emission and absorption spectra toward the blue end. Accordingly, the smaller CQDs have a higher energetic potential and thus emit at shorter wavelengths. In addition, it is particularly beneficial to increase the material's surface reactivity and to produce smaller nanostructured CQDs, which have widespread applications as catalysts and sensors.[9]. Photoluminescence is one of the most interesting and effective characteristics of CQD. Consequently, these materials are exploited in an extremely wide range of applications, such as sensing, catalysis, solar-energy conversion, biomedical imaging/diagnostic, phototherapy, light-emitting devices, photocatalysis, and supercapacitors [3]. CQD are synthesized and precipitated from a variety of precursors, and they exhibit different absorption spectra depending on the solvent used. In UV-Vis measurements, typically three characteristic bands of absorbance are observed, generally in the wavelength range from 200 nm to 750 nm. Zeta potential determinations are a key analytical method in the study of CQD and are a very important aspect of determining key parameters, such as surface charge and, thus, the colloidal stability of these nanomaterials within solution [9] This anlysed metric represented an electrical potential difference between the surrounding medium and the surface of the particles which means that indicated important impact on the stability and dispersion of CQDs nanomaterial's in different surroundings. Zeta potential results, which are either negative or positive, indicate good dispersion and high stability of the nanomaterials (a high zeta potential results in strong electrostatic repulsion, which decreases particle aggregation)[9].

4 Experimental models of Ulcer colitis

4.1Trinitrobenzene Sulfonic Acid Model (TNBS)

The trinitrobenzene sulfonic acid model is a well-developed model system of colitis induction and is often used to mimic the transmural inflammation of Crohn's disease [14]. This model is based on intrarectal instillation of 2,4,6-trinitrobenzene sulfonic acid (TNBS), a hapten commonly dissolved in ethanol that triggers a delayed-type hypersensitivity reaction in the colon [15]. The epithelium is damaged by ethanol and exposes trinitrobenzene sulfonic acid, allowing it to covalently modify proteins in the lumen and tissue. These changes make the proteins immunogenic, eliciting a strong T-lymphocyte-mediated immune response [16]. This process leads to the development of chronic inflammation, ulcerations, and fibrosis, which are very similar to the pathological processes of Crohn's disease in humans [14, 17].

Induced Ulcer Colitis using the TNBS model depends on the rodent strain, BALB/c and C3HeJ strains. Moreover, TNBS-induced ulcerative colitis was characterized by a T-helper-1-mediated immune response, with elevated interleukin-12 levels and other pro-inflammatory cytokines, including tumor necrosis factor alpha. Interferon gamma is one of the major cytokines implicated in the chronic inflammatory process [17]. Th1/Th17 T helper cells are the factors that drive the immune response and improve immunological profiling, as frequently observed in Intestinal disorder Crohn's disease. Acute TNBS administration can induce a pro-inflammatory Th1 response, as evidenced by increased expression of IL-12, IFN-γ, and TNF. This Th1 dominance distinguishes it from Th2-driven inflammation, which is typically observed in spontaneous models of ulcerative colitis, thereby emphasizing its suitability for studying the pathophysiology of Crohn's disease [18]. In the TNBS solution, the ethanol fraction aggravates the mucosal damage through its irritant action and by enhancing TNBS penetration through the bowel wall. The binding of TNBS to host proteins alters them, forming neoantigens that elicit a strong cell-mediated immune response, primarily involving CD4+ T cells, macrophages, and neutrophils, leading to transmural colitis. In the current histological evaluation, the specimen shows transmural inflammation, with prominent infiltration and edema. It frequently results in

architectural distortion and even the formation of granulomas, giving the appearance of mimicking the most important histological features of Crohn's disease[19].

5. Therapeutic mechanisms: Carbon quantum dots on Gastrointestinal Ulcerative Colitis Disorders

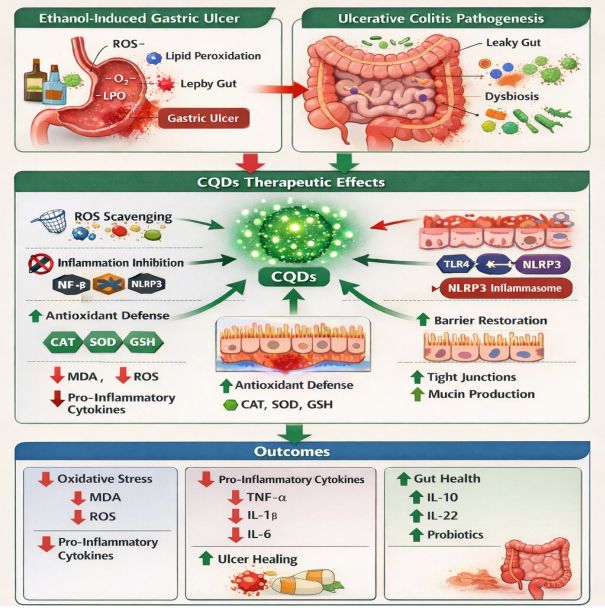

Fig 1. Mechanism of therapeutic action of carbon quantum dots on the treatment of ulcerative colitis.

Stomach ulcers are generally caused by an imbalance between environmental and defence factors, and alcohol is the most damaging factor, damaging the stomach's internal mucosa. The ethanol-induced gastrointestinal ulcer model has been histologically reported, with morphological, reparative, and recurrent features similar to those observed in human gastric mucosa, and can be produced with high reproducibility. Consumption of alcohol in an excessive manner not only attacks and affects the gastrointestinal mucosa, but also erodes the important protective barrier that protects the stomach and intestinal mucosa, and produces appreciable quantities of free radicals during metabolism and their absorption. Such free radicals may cause cell damage by breaking down lipid molecules in the cell membrane, a process called peroxidation, which can compromise the cell membrane and, ultimately, damage mucosal intestinal cells [5]. Free radicals are not efficiently neutralised, and they may cause oxidative damage to other important biological molecules, such as lipids, proteins, and DNA. Furthermore, alcohol affects the intestinal environment and natural flora and degrades beneficial factors. This creates a favorable environment for the growth of pathogenic microorganisms, thereby reducing beneficial gut microorganisms, which are important for protecting the mucosal membrane. Inflammation is a major mediator of ethanol-induced intestinal gastric ulcers. Damage to the mucosal membrane breaches the barrier between the gut's internal environment and external pathogens, initiating a self-perpetuating immune response involving macrophages and neutrophils that results in significant structural and functional changes. Concurrently, elevated free radical levels interfered with the antioxidant based defence mechanism and triggered activation of the NF-kB signalling pathway, leading to the synthesis and release of inflammatory cytokines TNF-α, IL-6, IL-1β, and intensification of inflammatory reactions. The cycle continues, culminating in apoptosis (cell death) and necrosis, which ultimately damage and kill intestinal and gastric cells. CQDs, which have therapeutic properties, showed a significant antiulcer effect in gastric cells. Empirical investigations have found that CQDs significantly reduce oxidative injury to the gastric mucosa, thereby increasing the expression of antioxidant enzymes, including catalase (CAT), glutathione peroxidase (GPx), and superoxide dismutase (SOD), as well as non-enzymatic antioxidants. Concurrently, they neutralise malondialdehyde (MDA), a marker of lipid peroxidation. Moreover, CQDs inhibited the NF-kBa/NLRP3 signalling cascade downstream of ROS by inhibiting the formation of reactive oxygen species (ROS), which dampens production of pro-inflammatory cytokines, including tumour necrosis factor TNF-alpha and interleukin-6. In addition, CQDs increase gastric mucosal defence factor concentrations to include "interleukin-10 prostaglandin E2 (PGE2) and mucin 5AC (MUC5AC)"[20]. These factors play essential roles in mitigating inflammation, maintaining mucosal cell membrane structural integrity, and synthesizing mucin glycoproteins, respectively [9]. Furthermore, nanomedicines can reduce the enzymatic activity of H^+/K^+-ATPase and pepsin. These are primary signs of gastric acid secretion, mucosal damage, and perturbation of the microbial environment, leading to changes in intestinal flora diversity and the growth of bacteria beneficial to the host's health in ulcerative colitis. Numerous studies have shown that overexpression of reactive oxygen species (ROS), such as the hydroxyl radical (OH^-), hydrogen peroxide (H_2O_2), and superoxide anion (O_2^-), plays a vital role in the aetiology and development of IBDs. It has been

documented that the overproduction of reactive oxygen species (ROS) is a principal etiological factor in the development of ulcerative colitis, as well as in the worsening of inflammation and the infection of the colon, due to the oxidative damage it causes to cellular macromolecules. Consequently, the suppression of oxidative stress, which can be done by eliminating excess ROS, is crucial for successful UC therapy [15]. CQD has a high surface area and functional groups, which suggest it can be used as an antioxidant. These include amine (NH_2), carboxyl (COOH), and hydroxyl (OH) groups and confer non-enzymatic antioxidant properties similar to those of reduced glutathione (GSH). These are the major compounds that could neutralize ROS, especially hydroxyl (OH) radicals, thus decreasing the levels of "malondialdehyde (MDA), nitric oxide (NO), and simultaneously increasing superoxide dismutase (SOD) activities in colonized tissue, and improving blood circulation. Moreover, CQDs behaved as antioxidant nano-enzymes with superoxide dismutase (SOD) like properties, thereby efficiently scavenging a significant amount of intracellular and extracellular superoxide (O_2) by converting it to H_2O_2 and oxygen. H_2O_2, however, can readily cross the cellular membrane, react with intracellular $Fe2^+$ ions, and generate highly reactive hydroxyl radicals via the Fenton reaction, thereby causing oxidative stress. Recent investigations show that doping CQD with metals, including I, C, and Zn, endows them with, in addition to the functionality of SODs, that of catalase (CAT) and glutathione peroxidase (GPx). This modification allows them to work well for the removal of H_2O_2. Nonetheless, the low catalytic enzymatic properties of CQDs in the ROS scavenging of certain ROS species are attributed to a relatively decreased capability for electron transfer. Introducing Cu, I, and Zn helps address this deficiency, leading to a significant increase in electron density and their quasi-enzymatic mechanistic activity. Recent investigations have shown that carbon quantum dots (CQDs), by modulating oxidative stress levels within cells, not only prevent tissue injury but also reduce pro-inflammatory cytokine production by inhibiting the activation of the Nuclear Factor Kappa (NF-kappaB)/NLRP3 signaling cascade in the colon. The successful accomplishment of this aim is attested by the ablation of reactive oxygen species and the concomitant inhibition of the TLR4 receptor that represents the principal activator of the excitation mechanism behind this particular inflammatory signalling pathway [9]. Inhibiting the NLRP3 pathway with CQDs, the expression and levels of ASC, Caspase-1, and IL-1β are naturally reduced, as well as the production of downstream cytokines TNF-α, IL-1β, IL-6, IL-17A, IL-23, and IL-12 [21]. Due to its ability to enhance the functional competence of both innate and adaptive immune components and to promote the generation of IL-17A in combination with granulocyte-macrophage colony-stimulating factor (GM-CSF) and IL-23, the reduction of GM-CSF plays a critical role in limiting neutrophil recruitment and reducing intestinal inflammation. Additionally, decreased GM-CSF reduces monocyte migration from the circulation to infected tissues and inhibits their maturation into fully developed macrophages [9, 22]. As a result, Myeloperoxidase (MPO) activity is attenuated, reducing inflammation and inhibiting the synthesis of pro-inflammatory cytokines ("TNF-α, IL-1β, IL-6"), accompanied by inhibition of leukocyte migration to inflammatory sites. It is after the amelioration of tissue damage that the application of CQDs promotes reorganisation of the affected area by up-regulating anti-inflammatory mediators interleukin-10 and interleukin-22, decreasing the overproduction of pro-cytokines, and preventing colonisation of the wound site by pathogenic microorganisms. Furthermore, not only does the application of carbon quantum dots promote the reparative process, but it also promotes the restoration of the intestinal epithelial integrity by increased expression of tight junction (amino acid) proteins. "occludin, claudin-1, Zonula occludens-1 and mucin-2, while simultaneously stimulating proliferation of beneficial probiotics Muribaculaceae, Lactobacillus, Lachnospiraceae, Roseburia, and Rikenella". As a consequence, colon length increases, the index of disease activity eases, nutrient absorption improves, and eventually body weight increases [9].

Table 1 Pathogenic factors, molecular mechanisms, and the effect of carbon quantum dots on ulcerative colitis disorders.

Disease Model	Key Pathogenic Factors	Major Molecular Mechanisms	Therapeutic Effects of CQDs	Biological Outcomes	References
Ethanol-Induced	Alcohol toxicit	NF-κB/NLRP3	✦ ROS neutralization;	MDA ↓, ROS	[9,23]

Gastric Ulcer	y; ROS overproduction; lipid peroxidation; microbiota imbalance	activation; ↑ TNF-α, IL-6, IL-1β; immune cell infiltration	↑ antioxidant enzymes (CAT, SOD, GPx); ⊖ NF-κB/NLRP3 signaling; ↓ inflammatory cytokines; ↺ microbiota balance	↓, TNF-α ↓, IL-6 ↓, IL-10 ↑, PGE_2 ↑, Antioxidant defense ↑	
Oxidative Stress (Gastric Tissue)	H_2O_2, •OH, O_2^- radicals	Lipid peroxidation; DNA damage; membrane injury	✦ ROS scavenging; ↓ lipid peroxidation	MDA ↓, membrane stability ↑	[9,24]
Ulcerative Colitis (UC)	Excess ROS; TLR4 activation; NLRP3 inflammasome	NF-κB/NLRP3 signaling; ASC & Caspase-1 activation; cytokine cascade	↑ SOD-like nanozyme activity; ⊖ TLR4/NF-κB/NLRP3 pathway; ↓ pro-inflammatory cytokines; ↺ epithelial barrier repair	MPO ↓, DAI ↓, cytokines ↓, SOD ↑, tight junction proteins ↑	[9]
Intestinal Barrier Dysfunction	Tight junction disruption; gut dysbiosis	Loss of occludin, claudin-1, ZO-1, MUC-2	↑ tight junction proteins; ↑ mucin secretion; ↺ microbiota homeostasis	Colon length ↑, body weight ↑, nutrient absorption ↑	[3,9,25]

Symbol Legend: ↑ enhancement/upregulation; ↓ reduction; ⊖ inhibition; ✦ neutralization; ↺ restoration/homeostasis.

Abbreviations: ROS – Reactive Oxygen Species; MPO – Myeloperoxidase; DAI – Disease Activity Index; SOD – Superoxide Dismutase; CAT – Catalase; GPx – Glutathione Peroxidase; TLR4 – Toll-like Receptor 4; NF-κB – Nuclear Factor kappa B; NLRP3 – NOD-like receptor protein 3 inflammasome.

Conclusion

Carbon quantum dots are primarily used as diagnostic and therapeutic nanomaterials, produced via environmentally benign processes using a variety of organic and biological precursors. The source material directly influences the properties of CQDs, including absorption, zeta potential, and photoluminescence, paving the way to tailor them for applications such as therapy, imaging, and sensing. Their size also depends on the synthesis technique and the materials used. CQDs exhibit little cytotoxicity toward normal gastrointestinal cells; however, determining safe dosage limits and their toxicity remains a challenge. Mechanistically, CQDs provide gastric protection and promote gastric ulcer recovery by acting as dual-nature antioxidants, with functional groups on the surface derived from the precursors. These groups interact with receptors that function in signaling mechanisms, thus influencing gene expression. In addition, CQDs modulate the inflammatory response and cell activity by attenuating oxidative stress, facilitating tissue repair and promoting wound healing, and inhibiting recurrent gastrointestinal

disorders. The demonstrated efficacy in preclinical studies indicates that CQDs have great potential as antiulcer nanomedicines. Subsequent research will focus on clinical applications, toxicity, and side effects, and will continue advancing these therapeutic agents in nanomedicine in the modern world. The current review lays a strong foundation for future studies on the potential therapeutic value of CQDs for treating gastrointestinal ulcers, synthesized from plant materials and natural components readily available, making them particularly beneficial for therapeutics.

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