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Carbon quantum dots: a versatile nanotechnology for personalized treatment in Alzheimer's disease

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Abstract

Alzheimer's Disease (AD) represents a global health crisis characterized by complex multifactorial etiology and severe pharmacological limitations imposed by the Blood–Brain Barrier (BBB). This narrative review critically synthesizes recent advancements (2021–2026) regarding the application of Carbon Quantum Dots (CQDs) as a transformative nanotechnological paradigm for AD management. Unlike traditional semiconductor quantum dots, CQDs synthesized via eco-friendly "green" protocols offer superior biocompatibility and unique physicochemical properties ideal for central nervous system interfacing. The article details the molecular mechanisms—specifically passive diffusion and carrier-mediated transport, alongside alternative intranasal and intrathecal routes—that enable surface-engineered CQDs to bypass or circumvent the BBB. A dual-functional analysis high lights the versatility of these structures: diagnostically, they facilitate ultra-sensitive pre symptomatic detection of amyloid-beta and Tau biomarkers through Near-Infrared (NIR) bioimaging and fluorescent biosensing; therapeutically, CQDs are presented as multi-target agents that exhibit intrinsic neuroprotective capabilities (inhibiting protein aggregation and scavenging Reactive Oxygen Species) while serving as advanced vectors for the targeted delivery of genetic materials and neurotrophic drugs such as memantine and insulin. Central to this review is the concept of personalized medicine, exploring "theranostic" platforms and stimuli-responsive smart delivery systems (*e.g.*, pH-sensitive) that adapt to the individual pathological microenvironment. The review concludes by addressing critical challenges regarding long-term biosafety, "protein corona" formation, and manufacturing scalability, while outlining future perspectives focused on autophagy reactivation and the standardization required for clinical translation.

Keywords: Alzheimer's disease, carbon quantum dots, nanomedicine, blood–brain barrier, theranostics, personalized medicine

Introduction

The context of the problem: the neurodegenerative crisis and current therapeutic limitations

Alzheimer's Disease (AD) represents a global health crisis and stands as the most prevalent form of dementia [1, 2]. While the classical histopathological hallmarks—extracellular accumulation of amyloid-beta (Aβ) plaques and intracellular formation of neurofibrillary tangles (NFTs) composed of hyperphosphorylated Tau protein—are well-documented, the fundamental etiology of the disease remains incompletely elucidated and highly complex [2, 3]. Recent evidence suggests that AD is not merely a proteinopathy but a multifactorial pathology involving early synaptic dysfunction, cholinergic deficits, severe oxidative stress, mitochondrial impairment, and chronic neuroinflammation mediated by microglia [1, 3]. This pathophysiological heterogeneity largely explains the high failure rate of "single target" therapies (*e.g.*, those solely removing amyloid), highlighting the critical need for multimodal therapeutic approaches [1, 3].

The efficacy of current treatments is severely compromised by the Blood–Brain Barrier (BBB), a highly selective endothelial structure that preserves cerebral homeostasis [2]. From a pharmacokinetic perspective, the BBB acts as an almost impenetrable filter for most therapeutic agents [1]. It actively restricts the passage of hydrophilic molecules and compounds with a molecular weight exceeding 500 Da, a threshold that excludes most proteins, monoclonal antibodies, and genetic fragments [4, 5]. Furthermore, the presence of active efflux pumps, such as P-glycoprotein (P-gp), rapidly expels drugs that manage to diffuse passively, preventing them from reaching effective therapeutic concentrations within the brain parenchyma [1, 4]. Consequently, over 98% of potential Central Nervous System (CNS) drugs fail to cross this barrier without an advanced transport vector [1, 4].

Carbon quantum dots (CQDs) – a versatile paradigm

To understand the innovation represented by Carbon Quantum Dots, it is necessary to first define the broader category of Quantum Dots (QDs). QDs are semiconductor nanocrystals, typically 2–10 nm in diameter, whose optoelectronic properties are governed by the quantum confinement effect [6]. Unlike bulk materials, the electron-hole pairs (excitons) in QDs are spatially confined in all three dimensions, which leads to discrete energy levels, allowing their optical emission spectra to be precisely tuned simply by altering the particle size [6]. While traditional QDs exhibit high quantum yields and photostability, they are predominantly composed of heavy metals — such as cadmium (Cd) or lead (Pb) — which pose severe risks of cytotoxicity and environmental bioaccumulation, limiting their application in clinical medicine [6].

Carbon Quantum Dots (CQDs), discovered during the purification of single-walled carbon nanotubes, represent the "green" successor to traditional semiconductor QDs [6, 7]. They are defined as zero-dimensional carbonaceous nanomaterials, generally smaller than 10 nm, characterized by a core structure of sp2/sp3 hybridized carbon atoms [6–8]. What distinguishes CQDs from other carbon allotropes (like graphene or nanotubes) is their amorphous or nanocrystalline nature combined with extensive surface passivation by functional groups (oxygen- or nitrogen-containing moieties) [6]. These structural characteristics confer unique physicochemical properties: unlike their heavy-metal counterparts, CQDs are chemically inert, highly soluble in water, and exhibit a superior biocompatibility profile, making them the ideal candidate for biological interfacing where safety is paramount [6].

The paradigm of nanomedicine: overcoming biological barriers and enabling early diagnosis

In response to the pharmacokinetic limitations of conventional pharmacology, nanomedicine has emerged as a pivotal strategy to bypass the BBB [1]. Nanocarriers offer the unique ability to encapsulate neurotrophic agents, protecting them from enzymatic degradation while facilitating their transport into the central nervous system through receptor-mediated transcytosis or adsorptive mechanisms [1, 4]. However, the transition from general nanocarriers to specific Carbon Quantum Dots (CQDs) represents a significant leap forward. Leveraging the unique physicochemical architecture described in the previous section, CQDs possess a superior surface-to-volume ratio compared to bulkier polymeric carriers [6]. This characteristic, combined with the abundance of surface functional groups (carboxyl, hydroxyl, and amino), facilitates extensive chemical functionalization. Such "molecular tunability" is essential for personalized medicine, enabling the conjugation of specific ligands that match the patient's distinct pathological profile [6].

Crucially, the most transformative advantage of CQDs lies in their intrinsic optical properties (tunable fluorescence), which address the greatest failure of current AD management: late diagnosis [2]. Clinical consensus suggests that therapeutic interventions often fail because they are administered after irreversible neuronal damage has

occurred [1]. CQDs can serve as ultra-sensitive contrast agents for bioimaging (particularly in the Near-Infrared windows, NIR-I and NIR-II), capable of detecting amyloid-beta aggregation and tau fibrillation in the prodromal or presymptomatic phase [2]. By visualizing these molecular hallmarks before the onset of cognitive decline, CQDs facilitate a "therapeutic window" where treatment can modify the disease trajectory, preventing the condition from reaching the stage where brain atrophy makes recovery impossible [1].

Objective of the review

This narrative review aims to critically synthesize and analyse the most recent scientific advancements— specifically focusing on literature published between 2021 and 2026—regarding the emerging role of Carbon Quantum Dots in the management of Alzheimer's Disease [1, 6]. While previous reviews have addressed general nanomedicine applications, this article specifically targets the versatility of CQDs as multifunctional "theranostic" platforms. The analysis covers the transition towards eco-friendly "green synthesis" methods, the molecular mechanisms facilitating Blood–Brain Barrier and Blood-Cerebrospinal Fluid Barrier penetration (such as receptor-mediated transcytosis), and the integration of these nanostructures into personalized treatment strategies [1, 4, 6]. From ultrasensitive biosensors for prodromal biomarker detection to "smart" drug delivery systems responsive to the pathological microenvironment, this review evaluates how CQDs can bridge the gap between early diagnosis and targeted therapy [1, 7].

Fundamentals of CQDs for Alzheimer's disease

This section elaborates on the unique physicochemical properties that transition CQDs from mere optical biomarkers into complex therapeutic agents. It provides a critical analysis of their synthesis methodologies—shifting from traditional approaches to eco-friendly protocols—and examines their intrinsic capacity to interact with the CNS by overcoming physiological barriers [1, 6].

Green synthesis and surface engineering

The period from 2021 to 2026 marks a definitive paradigm shift from toxic "top-down" methods to "bottom-up" green synthesis. By utilizing renewable precursors (*e.g.*, citric acid, glucose) via hydrothermal treatment, these protocols yield CQDs with superior intrinsic biocompatibility and reduced cytotoxicity, which are essential attributes for chronic neuro-applications [6, 9].

To ensure biological efficacy, surface engineering is critical for addressing hydrophilicity and targeting. Stability (*e.g.*, PEGylation) is achieved through surface passivation with Polyethylene Glycol (PEG), which creates a hydrophilic "stealth" layer. This modification prevents opsonization (immune recognition) and significantly prolongs systemic circulation time [1], allowing the nanocarriers to reach the brain. The presence of reactive surface moieties (-COOH, NH2) enables the conjugation of specific targeting ligands such as transferrin to facilitate receptor-mediated BBB crossing [1, 6], while CDs retaining precursor-derived surface functionalities — such as glucose analogues — can exploit endogenous carrier proteins for transport [10]. Furthermore, CDs of sufficiently small size (below 6 nm) may traverse the BBB passively through tight junction gaps without the need for any ligand conjugation [5]. Furthermore, doping the carbon lattice with heteroatoms modulates the surface charge to optimize cellular uptake and target specificity: nitrogen doping enriches surface amino and carboxyl groups that facilitate specific protein binding [11], while selenium doping confers antioxidant and electron-transfer capabilities particularly relevant to ROS scavenging [3].

Crossing the blood-brain barrier: mechanisms and strategies

Overcoming the Blood-Brain Barrier remains a primary challenge in managing Alzheimer's Disease, as this restrictive endothelial lining blocks the passage of most molecules larger than 500 Da [1, 2]. However, due to their small size, biocompatibility, and stability, CQDs can penetrate the CNS via three distinct strategies [5, 10].

The first involves transcytosis across the BBB, where nano-formulations can bypass the barrier using specific cellular transport mechanisms, including Receptor-Mediated Transcytosis (RMT), Carrier-Mediated Transcytosis (CMT), and Adsorptive-Mediated Transcytosis (AMT) [4]. Functionalization is key to this approach; for instance, studies have demonstrated that CQDs coated with targeting ligands can successfully cross the barrier via RMT [1, 6]. Notably, self-targeting CDs that retain precursor-derived surface motifs — such as glucose residues — can exploit endogenous transporter proteins without additional ligand conjugation [10], while ultrasmall CDs below 6 nm can traverse tight junction gaps through passive diffusion [5].

A second strategy is the nasal-brain route. This non-invasive approach allows instilled or inhaled CQDs to deposit on the nasal mucosa and travel directly to the CNS, effectively bypassing the BBB. Transport occurs via the olfactory and respiratory epithelial routes, as well as through trigeminal pathways [4].

Finally, via intrathecal administration, CQDs injected directly into the cerebrospinal fluid (CSF) reach the brain parenchyma through mechanisms that include bulk flow transport, movement through the glymphatic system, passive diffusion, and active transport across the blood–CSF barrier [4]. These complementary strategies expand the therapeutic landscape for CQD-based interventions in AD [1, 4].

Pathological mechanisms of AD targeted by nanotherapy

Once localized within the brain parenchyma, CQDs cease to be passive carriers and actively interface with the dysregulated molecular cascades of Alzheimer's Disease. The therapeutic relevance of these nanomaterials relies on their ability to modulate proteopathy, oxidative stress, and neuroinflammation through specific physicochemical interactions [1, 8].

Modulation of the amyloid-beta (Aβ) cascade

The aggregation of Aβ monomers into toxic oligomers and insoluble fibrils is driven by hydrophobic interactions and beta-sheet stacking [11]. CQDs, particularly those doped with nitrogen or functionalized with carboxyl groups, exert a potent anti-amyloidogenic effect by interfering with these kinetics [8, 11]. Through electrostatic forces and hydrogen bonding, CQDs bind to the Aβ monomers, sterically blocking the nucleation sites required for fibril growth [11]. Furthermore, recent data suggest that these nanostructures can disassemble pre-existing mature fibrils, thereby reduce the burden of extracellular plaque and mitigate synaptic toxicity [8].

Inhibition of Tau protein hyperphosphorylation

While amyloid plaques are extracellular, the neurofibrillary tangles (NFTs) formed by hyperphosphorylated Tau protein accumulate intracellularly, leading to microtubule destabilization. CQDs have demonstrated the capacity to penetrate neuronal membranes and interact directly with the Tau protein [12, 13]. Structural analyses reveal that this interaction is primarily governed by hydrophobic forces: less polar CQD surfaces exhibit significantly stronger inhibition of Tau aggregation than their hydrophilic counterparts, with molecular dynamics simulations confirming that CQDs bind to a hydrophobic cavity within the Tau fibril and engage key residues through hydrogen bonding and CH-π interactions [13]. This 'dual inhibition' capability — targeting both Aβ and Tau simultaneously — distinguishes CQDs from conventional monoclonal antibody therapies that typically address only a single pathological target [8].

Mitigation of oxidative stress (ROS scavenging)

Mitochondrial dysfunction and the subsequent overproduction of Reactive Oxygen Species (ROS) act as central mediators of neuronal apoptosis in AD. CQDs synthesized from carbon-rich precursors (*e.g.*, citric acid) or doped with Selenium possess intrinsic electron-donating and accepting capabilities, functioning as broad-spectrum nano-antioxidants [1, 3]. They effectively scavenge hydroxyl and superoxide radicals, protecting neuronal lipids and proteins from oxidative damage [3].

Immunomodulation of microglia

Chronic neuroinflammation, driven by the persistent activation of microglia towards a pro-inflammatory (M1) phenotype, exacerbates neurodegeneration. Emerging evidence indicates that carbon-based nanomaterials, including CQDs, can influence the immune microenvironment. By modulating signaling pathways, CQDs can facilitate the repolarization of microglia from the neurotoxic M1 state to the neuroprotective, anti-inflammatory M2 phenotype, thereby promoting tissue repair and reducing the secretion of damaging cytokines like TNF-α and IL-1β [1].

Diagnostic applications: from bio-imaging to smart sensors

Current clinical diagnosis of Alzheimer's Disease relies heavily on cognitive assessments and structural imaging (MRI, PET), which often detect pathological changes only at advanced stages where neuronal loss is irreversible [1, 2]. Nanotechnology based on Carbon Quantum Dots promises to revolutionize this landscape by providing tools for ultra-sensitive molecular detection in the prodromal phase [7, 9].

Fluorescent biosensors for biomarker detection (Aβ and Tau)

The fundamental property of CQDs that renders them superior to traditional dyes is their tunable, environment-dependent fluorescence. In recent literature, CQDs have been engineered as "turn-on" or "turn-off" fluorescent probes to quantify AD-specific protein aggregates. The predominant mechanism employed is Förster Resonance Energy Transfer (FRET). In this scenario, CQDs act as energy donors; in the presence of amyloid aggregates, the distance between the CQD and an acceptor (or the protein structure itself) shifts, resulting in a measurable quenching or restoration of the optical signal [2, 7].

To ensure high specificity and eliminate false positives from other cerebral proteins, CQD

surfaces are functionalized with molecular recognition elements, most notably aptamers (short single-stranded DNA/RNA sequences) or specific antibodies. Recent strategies involve CQDs conjugated with aptamers that selectively bind to toxic Aβ oligomers or hyperphosphorylated Tau [2, 8]. This interaction induces a conformational change that modulates the fluorescent signal, enabling the non-invasive detection of amyloid burden with exceptional sensitivity. CD-based sensors have demonstrated limits of detection in the picomolar or femtomolar range across a range of protein biomarkers [7]. Applied to Tau, such sensitivity opens the possibility for screening via accessible biological fluids rather than invasive lumbar punctures [2].

The revolution of near-infrared imaging (NIR-I and NIR-II)

A major limitation of optical bioimaging has been the poor tissue penetration of visible light and the high autofluorescence background of biological tissues [6]. CQDs emitting in the "biological transparency windows"—NIR-I (650–950 nm) and, more recently, NIR-II (1000–1700 nm)—have overcome these barriers [6, 7]. Through heteroatom doping or π-conjugation extension, the emission of CQDs is redshifted, allowing photons to penetrate the skull and deep cerebral tissues with minimal scattering [6].

Research from 2023–2026 has particularly emphasized the NIR-II window, which offers superior signal-to-noise ratios [6]. Functionalized NIR-CQDs have demonstrated the ability to cross the BBB and label amyloid plaques *in vivo* with high spatial resolution [1, 6]. Studies on transgenic murine models have shown that these nanostructures allow for the real-time visualization of plaque dynamics and drug distribution without the need for craniotomy, acting as safer, non-radioactive alternatives to PET tracers [1]. Due to their superior photostability compared to organic fluorophores, CQDs facilitate longitudinal monitoring of disease progression over extended periods [2, 6].

Multimodal imaging capabilities

To transcend the limitations of a single imaging modality, current trends focus on developing hybrid probes. CQDs are frequently doped with paramagnetic elements, such as Gadolinium or Manganese, to serve dual functions: acting simultaneously as fluorescent probes to provide high cellular resolution and as MRI contrast agents to offer macroscopic anatomical context [1, 6]. This multimodal capability allows clinicians to correlate the precise molecular localization of senile plaques (optical signal) with broader brain atrophy (MRI signal), providing a comprehensive clinical picture essential for personalized treatment planning [1, 6].

Advanced therapeutic strategies: drug carriers for established therapeutics

Building upon the intrinsic neuroprotective mechanisms detailed in Section II, the therapeutic utility of Carbon Quantum Dots is further amplified by their role as advanced vehicles for pharmacological agents. CQDs serve as ideal platforms for transporting neurotrophic drugs that otherwise exhibit poor solubility or an inability to cross the BBB [1, 2, 5, 6].

Optimizing current therapeutics: the case of memantine

Memantine is one of the few approved drugs for AD, yet its efficacy is often limited by non-specific distribution. Researchers have developed systems where memantine is covalently attached or loaded onto the CQD surface [12]. Results indicate that this memantine–CQD nano complex crosses the BBB significantly more efficiently than the free drug. Moreover, it exhibits a synergistic effect: the CQD inhibits protein aggregation via surface interactions, while memantine blocks NMDA receptors, providing superior neuronal protection [12].

Intranasal administration and insulin delivery (Nose-to-Brain)

Brain insulin resistance is a central feature of AD pathology [14]. Systemic administration of insulin poses risks (hypoglycaemia), while cerebral delivery is hindered by the BBB. A major innovation involves using CQDs as carriers for insulin via intranasal administration. This route bypasses the BBB, transporting insulin directly to the brain along the olfactory and trigeminal neural pathways [14]. Studies have demonstrated that "Insulin-CQDs" (Ins-CQDs) can restore insulin signaling in affected neurons and ameliorate cognitive deficits in animal models without affecting peripheral blood glucose levels, serving as a controlled-release matrix suitable for AD therapy [14].

CQDs and the concept of personalized treatment in AD

The transition from the conventional "one-size-fits-all" approach to precision medicine is critical in Alzheimer's Disease [1, 6]. Carbon Quantum Dots represent a fundamental tool in this paradigm shift, offering unique platforms that

integrate molecular detection, intelligent release, and therapeutic monitoring [1, 6].

Theranostic platforms: integrating diagnosis and therapy

The concept of "theranostics" is the central pillar of nanotechnology-mediated personalized treatment. CQDs possess the unique capability to function simultaneously as bio-imaging contrast agents and as drug carriers [1, 6]. In an ideal clinical scenario, the administration of a single CQD complex allows the clinician to precisely localize amyloid deposits (via NIR fluorescence) and release the therapeutic payload strictly in those regions [1]. This eliminates empirical treatment, ensuring that medication is delivered only upon molecular confirmation of the pathology [1].

Smart delivery conditioned by the pathological microenvironment

Personalizing treatment implies spatiotemporal control over drug release. CQDs have been developed as "smart" (stimuli-responsive) systems that react to the specific biochemical signatures of the AD-affected brain. A prominent example is pH-sensitive systems [1, 6]. The microenvironment surrounding senile plaques and lysosomal compartments exhibits an acidic pH (5.0–6.0), in contrast to the neutral pH (7.4) of healthy tissue/blood. CQDs are engineered with labile chemical linkages (*e.g.*, Schiff bases) that keep the drug tightly bound during systemic circulation but cleave rapidly once the nanostructure reaches the acidic lesion zone [1, 6]. This ensures maximum local efficacy while minimizing systemic toxicity [1].

Patient stratification and specific molecular targeting

CQD nanotechnology allows therapy to be tailored to the patient's distinct molecular profile. Since AD pathology is heterogeneous, CQD surface chemistry permits the attachment of specific ligands (aptamers, antibodies) to target the patient's dominant biomarker [1]. For instance, for patients with a major inflammatory component, CQDs can be functionalized to specifically target receptors on activated microglia, whereas for others, the priority might be delivering BACE1 inhibitors directly to neurons [1, 6]. This modularity transforms CQDs into a programmable medical platform [1, 6].

Discussion

However, the clinical translation of CQDs is currently impeded by several major obstacles that necessitate rigorous investigation. First, although "green synthesis" has reduced acute cytotoxicity, long-term biosecurity remains an open question. Concerns persist regarding the potential for nanostructure accumulation in filtration organs (liver, kidneys) or within the cerebral parenchyma following chronic administration [1, 6]. Additionally, the phenomenon of "protein corona" formation—the adsorption of plasma proteins onto the CQD surface immediately post-injection—can dramatically alter targeting properties and may trigger unforeseen immune reactions, effectively masking the specific ligands intended for BBB receptors [1].

Second, standardization and scalability of production represent a significant technological challenge. Current laboratory methods produce small quantities with notable batch-to-batch variation regarding size and degree of functionalization. For clinical utility, it is imperative to develop industrial protocols that guarantee absolute homogeneity of CQDs, as minimal variations in size or surface charge can radically alter pharmacokinetics and toxicity profiles [1, 6].

According to the most recent analyses (2025–2026), the future of CQDs in Alzheimer's therapy will diverge from simple aggregation inhibition towards restoring natural cellular clearance mechanisms and integrating advanced technology [1]. A vanguard direction involves designing CQDs that do not merely block plaque formation but actively reactivate autophagy (the cell's waste disposal system). In AD, autophagy is stalled; new generations of CQDs are being studied for their ability to restore autophagic flux, aiding neurons in digesting intracellular toxic aggregates and defective mitochondria [1].

Concurrently, the integration of CQD-based sensors into point-of-care devices and wearable sensors is anticipated. Due to their extreme stability, CQDs can be incorporated into microfluidic chips or dermal patches to detect Aβ and Tau levels from interstitial fluid or blood with laboratory-grade precision but at a fraction of the cost, facilitating massive population screening [1, 2]. Finally, regarding the regulatory framework and pharmaceutical standardization, for these nanostructures to reach the clinic, the immediate perspective involves establishing strict "Good Manufacturing Practice" (GMP) protocols specific to nanomaterials. Research must focus on eliminating batch-to-batch synthesis variations. Without precise control of the CQD surface, regulatory approval remains unattainable, regardless of success in animal models [1, 6].

Conclusion

The analysis of recent literature (2021–2026) confirms that nanotechnology based on Carbon

Quantum Dots represents a paradigm shift in the management of Alzheimer's Disease, offering solutions to historical pharmacological limitations. Unlike conventional therapeutic agents, CQDs possess the intrinsic capability to traverse the Blood–Brain Barrier through multiple mechanisms — including passive diffusion through tight junction gaps, carrier-mediated transport via endogenous glucose transporters, and receptor-mediated transcytosis when functionalized with appropriate targeting ligands — ensuring superior cerebral bioavailability. Furthermore, their chemical versatility allows for synergistic action: they function simultaneously as ultra-sensitive diagnostic platforms (via NIR imaging and biosensors), as dual inhibitors of protein aggregation (Aβ and Tau), and as vehicles for targeted neurotrophic drug delivery. This multifunctionality defines CQDs as ideal instruments for implementing personalized medicine, allowing treatment to be tailored to the specific pathological profile of the patient.

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Conflict of interest

The authors declare no conflict of interest.

References

1. Bhukta P, Sahu PK, Acharya B. Nanomedicine approaches for Alzheimer's disease: advances in drug delivery and theranostic applications. Next Nanotechnol. 2026;9:100495. doi:10.1016/j.nxnano.2026.100495.
2. Guo F, Li Q, Zhang X, et al. Applications of carbon dots for the treatment of Alzheimer's disease. Int J Nanomedicine. 2022;17:6621-38. doi:10.2147/IJN.S388030.
3. Zhou X, Hu S, Wang S, et al. Large amino acid mimicking selenium-doped carbon quantum dots for multi-target therapy of Alzheimer's disease. Front Pharmacol. 2021;12:778613. doi:10.3389/fphar.2021.778613.
4. Madadi AK, Sohn MJ. Advances in intrathecal nanoparticle delivery: targeting the blood cerebrospinal fluid barrier for enhanced CNS drug delivery. Pharmaceuticals (Basel). 2024;17(8):1070. doi:10.3390/ph17081070.
5. Araújo CF, Rodrigues RO, Bañobre-López M, Silva AMT, Ribeiro RS. Carbon dots as a fluorescent nanosystem for crossing the blood-brain barrier with plausible application in neurological diseases. Pharmaceutics. 2025;17(4):477. doi:10.3390/pharmaceutics17040477.
6. Pechnikova NA, Domvri K, Porpodis K, et al. Carbon quantum dots in biomedical applications: advances, challenges, and future prospects. Aggregate. 2025;6(3):e707. doi:10.1002/agt2.707.
7. Terzapulo X, Kassenova A, Loskutova A, Bukasov R. Carbon dots: review of recent applications and perspectives in bio-sensing and biomarker detection. Sens Biosensing Res. 2025;47:100771. doi:10.1016/j.sbsr.2025.100771.
8. Zhang W, Smith N, Zhou Y, et al. Carbon dots as dual inhibitors of tau and amyloid beta aggregation for the treatment of Alzheimer's disease. Acta Biomater. 2024;183:341-55. doi:10.1016/j.actbio.2024.06.001.
9. Kaurav H, Verma D, Bansal A, Kapoor DN, Sheth S. Progress in drug delivery and diagnostic applications of carbon dots: a systematic review. Front Chem. 2023;11:1227843. doi:10.3389/fchem.2023.1227843.
10. Seven ES, Seven YB, Zhou Y, et al. Crossing the blood-brain barrier with carbon dots: uptake mechanism and in vivo cargo delivery. Nanoscale Adv. 2021;3:3942-53. doi:10.1039/d1na00145k.
11. Liu H, Guo H, Fang Y, Wang L, Li P. Rational design of nitrogen-doped carbon dots for inhibiting β-amyloid aggregation. Molecules. 2023;28(3):1451. doi:10.3390/molecules28031451.
12. Zhang W, Kandel N, Zhou Y, et al. Drug delivery of memantine with carbon dots for Alzheimer's disease: blood-brain barrier penetration and inhibition of tau aggregation. J Colloid Interface Sci. 2022;617:20-31. doi:10.1016/j.jcis.2022.02.124.
13. Zhou Y, Kandel N, Bartoli M, et al. Structure-activity relationship of carbon nitride dots in inhibiting tau aggregation. Carbon. 2022;193:1-16. doi:10.1016/j.carbon.2022.03.021.
14. Camlik G, Bilakaya B, Ozsoy Y, Degim IT. A new approach for the treatment of Alzheimer's disease: insulin–quantum dots. J Microencapsul. 2024;41(1):18-26. doi:10.1080/02652048.2023.2282928.