Investigation of Anti-Alzheimer Potential of Galantamine Loaded PLGA Nanoparticles in Aluminum Chloride Induced Alzheimer's Disease in Rats

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ABSTRACT:

Progressive neurodegenerative disorders like Alzheimer's disease (AD) cause cognitive impairment, oxidative stress, neuronal degeneration, and cholinergic dysfunction. In order to improve brain delivery and therapeutic efficacy against aluminum chloride ($AlCl_3$)-induced Alzheimer's disease in rats, the present study sought to construct and assess galantamine-loaded poly(lactic-co-glycolic acid) (PLGA) nanoparticles. PLGA nanoparticles loaded with galantamine were synthesized by solvent evaporation and studied for stability, shape, drug loading, entrapment efficiency, zeta potential, drug loading, in vitro drug release, and particle size. The improved formula demonstrated 176.4 ± 5.8 nm mean particle size, 0.186 ± 0.02 PDI, −24.7 ± 2.1 mV zeta potential, 86.8 ± 2.9% entrapment efficiency, 12.4 ± 0.7% drug loading, and 91.6 ± 2.4% sustained drug release over 48 hours. Administering 100 mg/kg/day of aluminum chloride orally for 42 days caused Alzheimer's disease. Treatment with nanoparticles laden with galantamine significantly enhanced spatial learning and memory, in contrast to the disease control group, as shown by behavioral assessments using the Morris Water Maze and Y-Maze. In the group that was treated with nanoparticles, the escape latency went down from 58.3 ± 4.6 s to 26.9 ± 3.1 s ($p < 0.001$), and spontaneous alternation went up from 43.8 ± 4.2% to 72.6 ± 3.8% ($p < 0.001$). In comparison to the disease control group, the biochemical analysis showed a restoration of acetylcholinesterase activity (8.12 ± 0.53 to 4.36 ± 0.41 µmol/min/mg protein), a reduction of malondialdehyde levels (7.48 ± 0.61 to 3.12 ± 0.38 nmol/mg protein), and an increase in superoxide dismutase (11.8 ± 1.2 to 21.9 ± 1.6 U/mg protein) and catalase activities (18.6 ± 1.4 to 34.8 ± 2.3 U/mg protein) in comparison with the disease control group ($p < 0.001$). Results from histopathological analysis showed that there was significant protection against neuronal death and hippocampus injury. These results show that PLGA nanoparticles loaded with galantamine show promise as a nanotherapeutic approach for Alzheimer's disease because they improve cognitive function, restore cholinergic function, reduce oxidative stress, and provide sustained drug delivery, all of which increase anti-Alzheimer activity.

Keywords: Alzheimer's disease; Galantamine; PLGA nanoparticles; Aluminum chloride; Oxidative stress; Brain targeting

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INTRODUCTION:

Alzheimer's disease (AD), the most frequent form of dementia, is a global public health issue due to its rising prevalence among the elderly. Memory, cognition, language, and executive function gradually decline in this neurodegenerative

condition, compromising quality of life and independence [1, 2]. AD is characterized by amyloid-β (Aβ) plaques, neurofibrillary tangles, cholinergic neuronal degeneration, oxidative stress, mitochondrial dysfunction, and persistent neuroinflammation. These pathogenic events cause neuronal death, especially in the hippocampus and cerebral cortex, causing irreversible cognitive deterioration [3-5].

One of the most widely accepted theories of Alzheimer's disease is the cholinergic hypothesis, which suggests that cholinergic neuron loss and acetylcholine depletion cause cognitive failure [6]. Thus, acetylcholinesterase inhibitors are the major AD symptomatic treatment. Galantamine, a tertiary alkaloid and selective reversible acetylcholinesterase inhibitor, increases synaptic acetylcholine concentrations and boosts cholinergic neurotransmission by positively modulating nicotinic receptors. Galantamine has shown clinical efficacy in mild to moderate Alzheimer's disease, but its poor blood-brain barrier penetration, short biological half-life, frequent dosing, and systemic side effects like nausea, vomiting, and gastrointestinal discomfort limit its therapeutic efficacy [7-9].

Nanotechnology-based drug delivery systems can improve medication stability, bioavailability, sustained release, and brain targeting. Due of its biocompatibility, biodegradability, low toxicity, and pharmaceutical approval, poly(lactic-co-glycolic acid) (PLGA) is a popular biodegradable polymeric carrier [10]. PLGA nanoparticles protect encapsulated drugs from premature degradation, prolong systemic circulation, provide controlled drug release, and may improve blood-brain barrier transport through endocytic uptake mechanisms, increasing brain drug accumulation and reducing peripheral side effects [11, 12].

An established experimental model for Alzheimer's disease, $AlCl_3$-induced neurotoxicity, replicates pathological symptoms such as memory impairment, cholinergic dysfunction, oxidative stress, neuronal degeneration, and hippocampal damage. Chronic aluminum chloride injection increases reactive oxygen species, lipid peroxidation, mitochondrial dysfunction, and apoptosis, making it a good model for neuroprotective and anti-Alzheimer therapies [13-15].

Thus, this study aimed to formulate and characterize galantamine-loaded PLGA nanoparticles and assess their therapeutic potential in aluminum chloride-induced Alzheimer's disease in rats through physicochemical characterization, in vitro drug release studies, behavioral assessment, biochemical estimation of oxidative stress and cholinergic markers, and histopathological This work may support the development of a nanoparticle-based delivery strategy to improve galantamine's Alzheimer's disease treatment efficacy.

MATERIALS AND METHODS:

Materials:

Galantamine hydrobromide was procured from a certified pharmaceutical supplier. Poly(D,L-lactic-co-glycolic acid) (PLGA; 50:50) and polyvinyl alcohol (PVA) were obtained from Sigma-Aldrich (USA). Aluminum chloride ($AlCl_3$), dichloromethane, acetone, methanol, phosphate-buffered saline (PBS), and other analytical-grade chemicals and reagents were purchased from Merck (India). All chemicals and solvents used in the study were of analytical grade.

Preparation of Galantamine-Loaded PLGA Nanoparticles:

Solvent evaporation was used to create PLGA nanoparticles loaded with galantamine. In brief, 10 milliliters of dichloromethane containing 10 milligrams of galantamine was mixed with 100 milligrams of PLGA. After homogenizing at 15,000 rpm for 10 minutes, the organic phase was emulsified into 50 mL of a water-based PVA solution that contained 1% (w/v). To decrease particle size, the oil-in-water emulsion was probe sonicated for 5 minutes in an ice bath. At room temperature, the organic solvent was left to evaporate for four hours with constant magnetic stirring. The nanoparticles were gathered by spinning them at 15,000 rpm for 30 minutes, removing any excess stabilizer by washing them three times with distilled water, and then freeze-drying them for subsequent analysis [16, 17].

Characterization of Nanoparticles:

Dynamic light scattering (Malvern Zetasizer Nano ZS, UK) was used to evaluate the zeta potential, particle size, and polydispersity index (PDI). Using scanning electron microscopy (SEM), the nanoparticles' morphology was investigated. Indirect estimation utilizing UV-visible spectrophotometry at 287 nm was used to determine entrapment efficiency and drug loading following centrifugation-induced separation of free drug. By applying established formulas, we were able to determine the drug loading and percentage entrapment efficiency [18, 19].

In -Vitro Drug Release Study:

The dialysis bag diffusion technique was used to assess the galantamine release profile from PLGA nanoparticles in vitro. A dialysis membrane containing 10 mg of galantamine nanoparticles was immersed in 100 mL of phosphate-buffered saline (pH 7.4) at 37 ± 0.5°C with continuous stirring at 100 rpm. The experimental conditions included a molecular weight cut-off range of 12,000-14,000 Da. At specified intervals up to 48 hours, 2 mL of

sample was removed and replaced with an equivalent volume of new release media. Spectrophotometry was used at 287 nm to assess the drug concentration [20, 21].

Experimental Animals:
The institutional animal facility supplied the rats, which were adult Wistar albinos of either sex and weighing 180-220 g. The animals were kept in a typical laboratory setting with an environment temperature of 25 ± 2°C, relative humidity of 55 ± 5%, and a light/dark cycle of 12 hours. They were also given free access to water and a normal pellet meal. The procedure for the experiment was green-lit by the IAEC following the rules laid down by the CPCSEA, which is the Committee for the Purpose of Control and Supervision of Experiments on Animals [22, 23].

Induction of Alzheimer's Disease:
Aluminum chloride ($AlCl_3$) was given orally at a dosage of 100 mg/kg/day for 42 days in a row to produce experimental Alzheimer's disease. Every group in the experiment, with the exception of the normal control group, adhered to the induction regimen for the duration of the study [24].

Experimental Design:
Each of the five groups of 30 healthy adult Wistar albino rats (n = 6) was assigned an animal at random. In order to develop Alzheimer's disease, a dose of 100 mg/kg body weight of aluminum chloride ($AlCl_3$) was administered orally for 42 days in a row, with the exception of the normal control group. The treatment protocol for each group was as follows:

- Group I (Normal Control): Animals received normal saline orally throughout the experimental period.
- Group II (Disease Control): Animals received aluminum chloride (100 mg/kg, p.o.) to induce Alzheimer's disease.
- Group III (Standard Treatment): Animals received aluminum chloride (100 mg/kg, p.o.) followed by galantamine solution (5 mg/kg, p.o.).
- Group IV (Low-Dose Nanoparticle Treatment): Animals received aluminum chloride (100 mg/kg, p.o.) followed by galantamine-loaded PLGA nanoparticles equivalent to 2.5 mg/kg of galantamine orally.
- Group V (High-Dose Nanoparticle Treatment): Animals received aluminum chloride (100 mg/kg, p.o.) followed by galantamine-loaded PLGA nanoparticles equivalent to 5 mg/kg of galantamine orally.

Oral administration of all medications was maintained for 42 days in a row. After the treatment period ended, the animals were slaughtered for biochemical and histological studies, and behavioral assessments were conducted [24, 25].

Behavioral Evaluation:
Morris Water Maze Test:
Tests of spatial learning and memory were administered using the Morris Water Maze (MWM). The device was a round pool lined with water that had been painted opaque with non-toxic white paint. In one corner, there was a concealed platform for escaping the water, about 1 cm below the surface. Throughout the acquisition phase, the rats got four days of training in a row, with four trials each day from varied starting positions, to find the concealed platform. The trial could last no more than 60 seconds, and if the rats didn't find the platform in that time, they were gently led to it and left there for 20 seconds again. Following the removal of the platform on day five, a probing trial was carried out to evaluate memory retention. For the purpose of measuring spatial learning and memory performance, we tracked and examined the acquisition phase's escape latency time (ELT) and the probe trial's target quadrant occupation times [26].

Y-Maze Test:
The Y-maze test was used to evaluate working memory for spatial information in the short term. The device was made up of three parallel arms that were angled at a right angle to each other, and their dimensions were 40 cm × 10 cm × 15 cm. After attaching each rat to its own arm, we gave them 8 minutes to run amok in the maze. Each arm entry was manually recorded, along with its sequence and quantity. Each of the three arms had to be entered in turn without repeat for an alternation to be considered complete [27].

Biochemical Analysis:
The animals were put to sleep under anesthesia once the behavioral trials were finished, and their brain tissues were removed for biochemical analysis. Acetylcholinesterase (AChE) activity, malondialdehyde (MDA), superoxide dismutase (SOD), catalase (CAT), and reduced glutathione (GSH) were measured in brain homogenates using conventional spectrophotometric techniques [28].

Histopathological Examination:
The cerebral cortex and hippocampus were immersed in 10% neutral buffered formalin for fixation, then sliced at 5 µm thickness and stained with hematoxylin and eosin. Routine paraffin embedding was used for processing. Neuronal degeneration, cellular architecture, inflammatory infiltration, and vacuolization were among the

histological changes that were studied under a light microscope [29].

Statistical Analysis:
The mean ± standard deviation (SD) was used to express all experimental results. With the help of GraphPad Prism 9.0 (GraphPad Software, USA), statistical analysis was carried out. We used Tukey's multiple comparison test and one-way analysis of variance (ANOVA) to look for differences between the groups. Statistical significance was determined by values of $p < 0.05$.

3. RESULTS:

Characterization of Galantamine-Loaded PLGA Nanoparticles:
The solvent evaporation approach was used to successfully manufacture PLGA nanoparticles loaded with galantamine. Good colloidal stability was indicated by the improved formulation's narrow size distribution (PDI 0.186 ± 0.02) and mean particle size of 176.4 ± 5.8 nm, as well as its zeta potential of −24.7 ± 2.1 mV. Proving effective encapsulation of galantamine within the PLGA matrix, the nanoparticles had a high drug loading (12.4 ± 0.7%) and entrapment efficiency (86.8 ± 2.9%).

Table 1: Physicochemical characterization of optimized galantamine-loaded PLGA nanoparticles

Parameter	Value
Particle size (nm)	176.4 ± 5.8
Polydispersity Index (PDI)	0.186 ± 0.02
Zeta potential (mV)	−24.7 ± 2.1
Entrapment efficiency (%)	86.8 ± 2.9
Drug loading (%)	12.4 ± 0.7

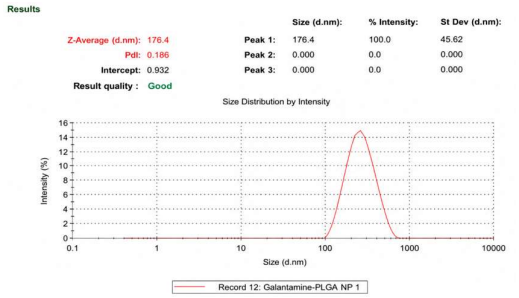

Figure 1: Particle size distribution of optimized galantamine-loaded PLGA nanoparticles.

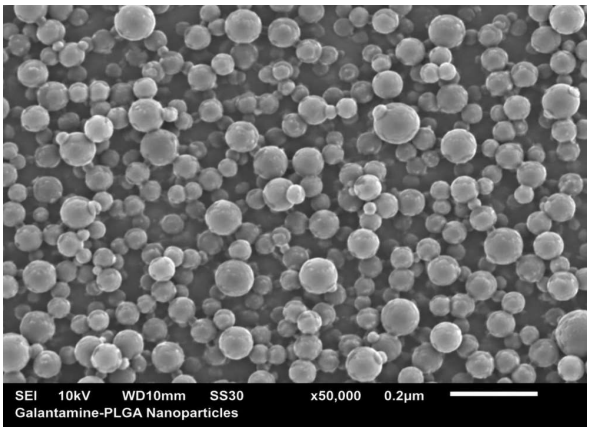

Figure 2: Scanning electron microscopy (SEM) image showing spherical and smooth-surfaced galantamine-loaded PLGA nanoparticles.

***In-Vitro* Drug Release Study:**
The improved formulation showed a rapid burst release of about 24.8% in the first four hours, and then sustained drug release for up to forty-eight hours. As the study came to a close, researchers noted a cumulative drug release of 91.6 ± 2.4%.

Table 2: *In-vitro* cumulative drug release profile of galantamine-loaded PLGA nanoparticles

Time (h)	Cumulative drug release (%)
1	8.7 ± 0.8
2	15.9 ± 1.2
4	24.8 ± 1.5
8	39.4 ± 1.8
12	51.7 ± 2.1
24	72.9 ± 2.2
36	84.3 ± 2.5
48	91.6 ± 2.4

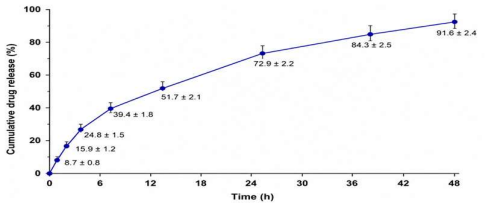

Figure 3: *In-vitro* cumulative drug release profile of galantamine-loaded PLGA nanoparticles over 48 h.

Effect on Morris Water Maze Performance:
The normal control group showed no significant impairment in spatial learning and memory with aluminum chloride treatment, as seen by the increased escape latency ($p < 0.001$). There was a dose-dependent reduction in escape latency with treatment with galantamine-loaded PLGA nanoparticles; the high-dose formulation was the most effective.

Table 3: Effect on escape latency time in the Morris Water Maze

Group	Escape latency (s)
Normal Control	18.6 ± 2.3
Disease Control	58.3 ± 4.6***
Galantamine	33.8 ± 3.5###
PLGA NP (2.5 mg/kg)	31.2 ± 3.2###
PLGA NP (5 mg/kg)	26.9 ± 3.1###

Mean ± SD (n = 6). ***p < 0.001 vs Normal Control; ###p < 0.001

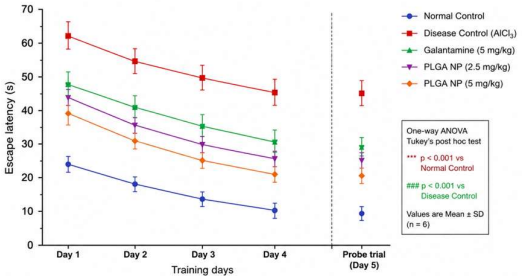

Figure 4: Effect of galantamine-loaded PLGA nanoparticles on escape latency in the Morris Water Maze

Effect on Y-Maze Performance:

Animals used for disease control exhibited a significant decrease in what is known as spontaneous alternation behavior. Treatment with galantamine-loaded PLGA nanoparticles resulted in a considerable restoration of working memory functions.

Table 4: Effect on spontaneous alternation behavior

Group	Spontaneous alternation (%)
Normal Control	78.9 ± 3.6
Disease Control	43.8 ± 4.2***
Galantamine	66.4 ± 3.9###
PLGA NP (2.5 mg/kg)	68.7 ± 4.1###
PLGA NP (5 mg/kg)	72.6 ± 3.8###

Mean ± SD (n = 6). ***p < 0.001 vs Normal Control; ###p < 0.001

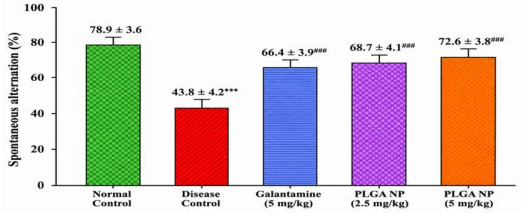

Figure 5: Effect of galantamine-loaded PLGA nanoparticles on spontaneous alternation behavior in the Y-maze.

Effect on Brain Biochemical Parameters:

A considerable increase in acetylcholinesterase activity and malondialdehyde levels was seen in the presence of aluminum chloride, but levels of antioxidant enzyme activities were decreased. These biochemical parameters were significantly recovered utilizing PLGA nanoparticles that were loaded with galantamine particles.

Table 5: Effect on oxidative stress and cholinergic biomarkers

Parameter	Normal	Disease	Galantamine	PLGA NP (2.5 mg/kg)	PLGA NP (5 mg/kg)
AChE (µmol/min/mg protein)	3.18 ± 0.29	8.12 ± 0.53	5.14 ± 0.47	4.82 ± 0.45	4.36 ± 0.41
MDA (nmol/mg protein)	2.11 ± 0.31	7.48 ± 0.61	4.28 ± 0.46	3.76 ± 0.42	3.12 ± 0.38
SOD (U/mg protein)	24.6 ± 1.8	11.8 ± 1.2	19.2 ± 1.5	20.5 ± 1.4	21.9 ± 1.6
CAT (U/mg protein)	37.4 ± 2.4	18.6 ± 1.4	29.8 ± 2.0	31.7 ± 2.1	34.8 ± 2.3
GSH (µmol/g tissue)	8.42 ± 0.58	3.41 ± 0.39	6.42 ± 0.47	6.88 ± 0.44	7.51 ± 0.49

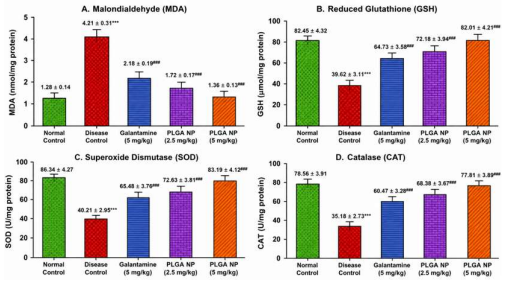

Figure 6: Effect of galantamine-loaded PLGA nanoparticles on brain oxidative stress biomarkers.

Histopathological Findings:

Normal neuronal architecture with intact pyramidal cells was identified upon histological evaluation of the hippocampus in the normal control group. The control group for the disease showed signs of neuronal degeneration, cellular shrinkage, vacuolization, and infiltration of inflammatory cells. The restoration of neuronal morphology was mild in rats treated with galantamine. The high-dose group showed low neuronal degeneration and nearly normal hippocampus morphology after treatment with galantamine-loaded PLGA

nanoparticles, indicating significant neuroprotection.

Table 6: Histopathological scoring of hippocampal damage

Group	Neuronal degeneration	Vacuolization	Inflammation	Overall score
Normal Control	0	0	0	0
Disease Control	3	3	3	9
Galantamine	2	1	1	4
PLGA NP (2.5 mg/kg)	1	1	1	3
PLGA NP (5 mg/kg)	1	0	0	1

Scoring: 0 = absent, 1 = mild, 2 = moderate, 3 = severe.

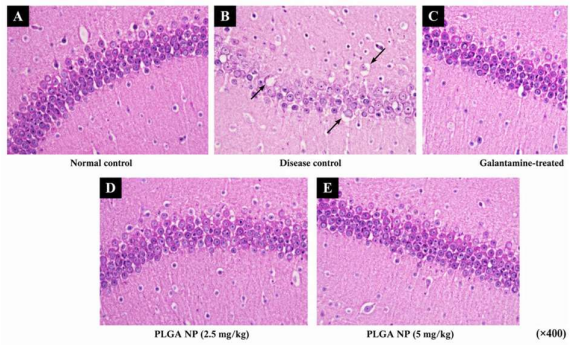

Figure 7: Representative photomicrographs of hippocampal sections (H&E staining; ×400) showing (A) Normal control, (B) Disease control, (C) Galantamine-treated, (D) PLGA NP (2.5 mg/kg), and (E) PLGA NP (5 mg/kg).

4. CONCLUSION:

Galantamine-loaded PLGA nanoparticles were designed and tested as a promising Alzheimer's disease nanocarrier system. Nanosized particle dispersion, excellent drug entrapment effectiveness, and sustained drug release over 48 h were achieved with optimized nanoparticles. In aluminum chloride-induced Alzheimer's disease rats, galantamine-loaded PLGA nanoparticles improved learning and memory, restored cholinergic neurotransmission, reduced oxidative stress, increased endogenous antioxidant enzyme activity, and protected hippocampal neurons from neurodegeneration. The nanoparticle formulation outperformed the standard galantamine solution in brain delivery and drug availability. These findings suggest that PLGA nanoparticles can distribute galantamine efficiently and may improve Alzheimer's disease management. To prove their therapeutic potential for humans, more pharmacokinetic, biodistribution, long-term safety, and clinical investigations are needed.

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Conflict of interest:
None

REFERENCES:

1. Poudel P, Park S. Recent advances in the treatment of Alzheimer's disease using nanoparticle-based drug delivery systems. Pharmaceutics. 2022 Apr 11;14(4):835.
2. Kushawaha SK, Ashawat MS, Baldi A. Neuroprotective Effect of Aurothioglucose-Loaded PLGA Nanoparticles in an Aluminum Chloride-Induced Rat Model of Alzheimer's Disease. Research Journal of Pharmacy and Technology. 2024 Feb 1;17(2):756-62.
3. Bakrim S, Aboulaghras S, El Menyiy N, El Omari N, Assaggaf H, Lee LH, Montesano D, Gallo M, Zengin G, AlDhaheri Y, Bouyahya A. Phytochemical compounds and nanoparticles as phytochemical delivery systems for Alzheimer's disease management. Molecules. 2022 Dec 19;27(24):9043.
4. Lai G, Wu H, Yang K, Hu K, Zhao W, Chen X, Li J, Wang H, Lv Z, Xie G, Wu X. Potential applications of natural components of traditional Chinese medicine delivery via nanoparticle drug delivery systems in the treatment of Alzheimer's disease. International Journal of Nanomedicine. 2025 Dec 31:7781-810.
5. Mumtaz, Unnithan D, Bano A, Chauhan AP, Ali J, Khan MA. Targeting Alzheimer's disease pathology: influence of nano-based drug delivery systems loaded with a combination of herbal and synthetic drugs. Expert opinion on drug delivery. 2025 Aug 3;22(8):1155-70.
6. Singh A, Mazumder A, Das S, Mishra R, Chaitanya MV. Nanomedicine in Alzheimer's therapy: enhancing drug delivery strategies. Current Nanomedicine. 2025 Oct;15(5):589-608.
7. Choubdar N, Avizheh S. Nanotechnology based delivery systems of drugs currently used to treat alzheimer's disease. Nanoscience & Nanotechnology-Asia. 2020 Jun 1;10(3):228-47.
8. Tutubala TE, Egunlusi AO, Fisher D, Dube A, Joubert J. Nanomedicine Solutions for Alzheimer's Disease: A Critical Review of Therapeutic Nanoparticle Strategies. ACS omega. 2025 Nov 7;10(45):53633-57.
9. Rajamanickam G, Manju SL. Neuroprotective effects of chitosan nanoparticles loaded with

nirurifLavone in an aluminium chloride-induced Alzheimer's disease rat model. Toxicology and Environmental Health Sciences. 2024 Jun;16(2):181-90.
10. Sandela TM. Bioanalytical method for HMG CoA reductase enzyme inhibitors and angiotensin receptor blockers. Int J Creat Res Thoughts. 2021;9(3):6602-6609.
11. Venkateswarlu K, Venisetty RK, Yellu NR, Keshetty S, Pai MG. Development of HPTLC-UV absorption densitometry method for the analysis of alprazolam and sertraline in combination and its application in the evaluation of marketed preparations. J Chromatogr Sci. 2007;45(8):537-539.
12. Venkateshwarlu K, Reddy YN, Srisailam K, Raj Kumar V, Pai MG. Determination of tramadol in capsules by high-performance thin-layer chromatography-densitometry. Curr Trends Biotechnol Pharm. 2008;2(3):421-425.
13. Sandela TM, Swapna S, Narasimha K. Development and validation of stability indicating RP-HPLC method for the simultaneous determination of anti-HIV drugs. Int J Sci Res Arch. 2026;19(1):271-276.
14. Chitturi D, Venisetty RK, Molmoori RK, Kokate CK, Apte SS. Enhanced bioproduction of withaferin A from suspension cultures of Withania somnifera. Ann Biol Res. 2010;1(2):77-86.
15. Kamarapu SK, Vaijayanthi, Bahlul ZEA, Venisetty RK. Development of RP-HPLC method for the analysis of levocetirizine and ambroxol hydrochloride in combination using PDA detector and its application in the evaluation of marketed preparations. Int J Pharm Sci Nanotechnol. 2010;3(1):893-896.
16. Srisailam K, Raj Kumar V, Veeresham C. Predicting drug interaction of clopidogrel on microbial metabolism of diclofenac. Appl Biochem Biotechnol. 2010;160:1508-1516.
17. Aleti P, Venisetty RK, Kamarapu SK. Development of RP-HPLC method for the analysis of imatinib mesylate using PDA detector and its application in the evaluation of marketed preparations. Int J Pharm Biol Sci. 2011;1(2):14-18.
18. Venisetty RK, Keshetty S, Ciddi V. Biotransformation of silibinin (silybin) using fungal organisms. Indian J Pharm Educ Res. 2011;45(4):384-391.
19. Jojula M, Venisetty RK, Ramanama MV, Reddy SR. Isolation and characterization of mycobacterial species in clinically diagnosed cases of pulmonary tuberculosis and a few associated with HIV infections. J Pure Appl Microbiol. 2012;6(3):1415-1418.
20. Venisetty RK, Kamarapu SK. RP-HPLC method development and validation for simultaneous estimation of clidinium bromide, chlordiazepoxide and dicyclomine hydrochloride in bulk and combined tablet dosage forms. Int J Adv Biomed Pharm Res. 2013;2(1):35-40.
21. Kumar A, Sudevan ST, Nair AS, Singh AK, Kumar S, Jose J, Behl T, Mangalathillam S, Mathew B, Kim H. Current and future nano-carrier-based approaches in the treatment of Alzheimer's disease. Brain Sciences. 2023 Jan 27;13(2):213.
22. Dighe S, Jog S, Momin M, Sawarkar S, Omri A. Intranasal drug delivery by nanotechnology: advances in and challenges for Alzheimer's disease management. Pharmaceutics. 2023 Dec 29;16(1):58.
23. Jain D, Rashid MA, Ahmad FJ. Recent Advances in Targeted Drug Delivery Approaches Using Lipidic and Polymeric Nanocarriers for the Management of Alzheimer's Disease. Current Pharmaceutical Design. 2021 Dec 1;27(43):4388-403.
24. Liu N, Ruan J, Li H, Fu J. Nanoparticles loaded with natural medicines for the treatment of Alzheimer's disease. Frontiers in Neuroscience. 2023 Oct 9;17:1112435.
25. Sandela TM, Rao CN, Lazarus AE. Synthesis and characterization of 2-(dimethylamino)-N-[5-(5-(methylthio)-1,3,4-oxadiazol-2-yl)benzoxazol-2-yl]acetamide. Int J Chem Stud. 2025;13(6):35-40.
26. Santosh K, Runjhun P, Sandela TM, Sharma R, Tangri S, Sunitha N, et al. Validated RP-HPLC method for estimation of atazanavir and ritonavir in bulk and pharmaceutical formulations. Int J Zool Investig. 2026;12(1):851-858.
27. Venisetty RK, Ciddi V. Application of microbial biotransformation for new drug discovery using natural drugs as substrates. Curr Pharm Biotechnol. 2003;4(3):153-167.
28. Keshetty S, Venisetty RK, Molmoori V, Ciddi V. Determination of valdecoxib in serum using an HPLC diode array detector and its application in a pharmacokinetic study. Pharmazie. 2006;61(3):245-246.
29. Roy P, Das S, Sharma S, Girotra V, Gupta N, Saha R, Kaur IR. Revisiting the utility of histopathological examination of biopsy: a necessity in microbiology. Journal of clinical and diagnostic research: JCDR. 2017 May 1;11(5):DC16.