Acupuncture and Herbal Medicine

Traditional Chinese medicine: a promising whole-process management for Alzheimer's disease

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Alzheimer's disease (AD) affects more than 57 million people worldwide, with a projected prevalence of 153 million by 2050[1]. The annual economic burden associated with AD already exceeds 1.3 trillion US dollars[2]. Notably, the prevalence of AD may be higher when estimated using biological rather than clinical criteria, thus underscoring the scale of the underlying disease burden[3]. For over two decades, the clinical management of AD has been dominated by symptomatic agents, including cholinesterase inhibitors and N-methyl-D-aspartate (NMDA) receptor antagonists, which provide modest and transient benefits without altering the underlying disease trajectory[4]. Although the recent approval of anti-amyloid monoclonal antibodies (aducanumab, lecanemab, and donanemab) represents a conceptual milestone, their clinical impact remains limited. Despite statistically significant reductions in cognitive decline, the clinical significance of these effects remains uncertain. The clinical use of these agents is further limited by frequent amyloid-related imaging abnormalities such as edema and hemorrhage[5]. These limitations reflect the complex nature of the disease that involves multiple pathogenic mechanisms[6]. Therefore, therapies directed at a single target are unlikely to achieve satisfactory and sustained efficacy across this heterogeneous disease spectrum. Next-generation AD therapeutics are likely to involve combination strategies that simultaneously address multiple pathophysiological processes[7].

This recognition has motivated a search for such therapeutic approaches. Traditional Chinese medicine (TCM) has accumulated centuries of clinical experience in managing cognitive decline and dementia-like conditions[8]. It is characterized by a multi-component, multi-target, and multi-pathway approach to disease treatment. Its defining therapeutic features conceptually align with the polypathogenic nature of AD. Over the past two decades, systematic clinical observations have led to the development of the kidney deficiency cascade hypothesis, a structured theoretical framework that, when combined with stage-adapted sequential therapy, provides whole-process management for AD. From this perspective, we outline the classical theoretical foundations of TCM for AD, examine modern pharmacological evidence supporting TCM-derived interventions, and present the kidney deficiency cascade hypothesis as an integrated clinical approach.

Theoretical foundations and therapeutic approaches of TCM for AD

The recognition of dementia-like syndromes in the Chinese medical literature dates back over two millennia. The seminal text *The Yellow Emperor's Inner Classic* established the foundational physiological axiom that "the kidney generates marrow, and the brain is the sea of marrow," suggesting an intrinsic connection between kidney function and cognitive integrity[8–9]. By the Ming and Qing dynasties, ancient scholars started classifying dementia as a distinct neurological disorder, explicitly linking cognitive decline to structural and functional impairments in the brain. TCM records have provided vital theoretical recognition of AD dementia.

Building on this historical foundation, contemporary scholars have synthesized their etiological understanding into a unifying framework. The TCM syndrome does not manifest as a single pattern during AD progression, but as an orderly pattern evolution process[10]. The kidney deficiency cascade hypothesis proposes that AD begins with kidney essence deficiency and progresses to phlegm muddiness, fire-heat disturbance, and blood stasis, ultimately resulting in severe toxicity and functional collapse. Notably, these pathogenic processes do not replace one another but superimpose and interact, creating an increasingly complex pathological terrain over the disease course. The evolution of these patterns reflects the general law of occurrence, development, and deterioration of AD, and provides a theoretical basis for the development of treatment strategies. Corresponding to this multifactorial understanding of pathogenesis, TCM employs a broad and integrated therapeutic approach. Herbal medicine constitutes the primary modality and is

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administered through pattern differentiation, a diagnostic methodology that matches treatment to an individual's unique combination of pathological factors.

The kidney deficiency cascade hypothesis and sequential therapy

These theoretical principles and empirical observations form a clinical framework that guides treatment approaches throughout the AD continuum. Based on analyses of patient cohorts and prospective clinical validation, the kidney deficiency cascade hypothesis proposes that AD progresses in an orderly sequence of overlapping pathogenic stages. These stages advance from initial kidney deficiency to intermediate phlegm muddiness, blood stasis, and fire-heat disturbance, ultimately resulting in severe toxicity and functional collapse[10]. A central principle of this model is that emerging stages do not replace the preceding ones, but rather superimpose upon them, creating an increasingly complex and treatment-resistant pathological environment as the disease advances (Figure 1). The early stage is driven by declining kidney essence and impaired cerebral marrow nourishment, manifesting predominantly as amnestic deficits that align with the preclinical and mild cognitive impairment phases of AD. The middle stage is characterized by phlegm muddiness, blood stasis, and fire-heat disturbance, which disrupts cerebral nutrient delivery, triggers neuroinflammatory cascades, and drives progressive cognitive and behavioral decline. Finally, the late stage is marked by severe toxicity and functional collapse, accelerating functional deterioration toward complete dependency.

This staging framework aligns with the current concepts of the AD continuum, outlined in the 2024 revised diagnostic criteria of the Alzheimer Association. These criteria established an integrated biological and clinical staging scheme incorporating tau pathology stages A to D and clinical severity stages 0 to 6, while explicitly recognizing that biological and clinical stages are frequently inconsistent[11]. Validation studies have demonstrated that 44% of amyloid-positive individuals exhibit a mismatch between clinical and biological staging, influenced by concurrent co-pathologies and variations in cognitive resilience[12]. The kidney deficiency cascade hypothesis accommodates this biological heterogeneity by attributing clinical progression not to a single molecular event with binary penetrance, but to the sequential accumulation, interaction, and mutual amplification of multiple pathological processes that vary across individuals and over time. This multifactorial stage-specific model provides a conceptual bridge between TCM emphasis on individualized pattern differentiation and the modern recognition of AD as a biologically heterogeneous syndrome.

Based on this hypothesis, the Sequential Therapy for Evolving Patterns (STEP regimen) was established for the longitudinal management of AD[10]. The STEP regimen has two distinct operational features compared with standard pharmacotherapy. First, kidney-tonifying therapy serves as a continuous therapeutic backbone from preclinical stages through severe dementia, addressing the foundational role of declining neurotrophic and neuroendocrine reserves in initiating and perpetuating the neurodegenerative cascade. This sustained approach parallels the chronic and uninterrupted administration of disease-modifying therapies in modern neurology. Second, targeted interventions are layered sequentially rather than substituted as new pathogenic mechanisms emerge. Specifically, formulas that resolve phlegm muddiness and blood stasis are introduced when metabolic and circulatory pathologies become clinically prominent; fire-purging and detoxifying interventions are deployed when inflammatory damage predominates; and vitality-restoring therapies are initiated in the advanced stages to prevent terminal functional collapse. Thus, the STEP regimen represents a dynamic, individualized framework rather than a rigid sequence of prescriptions, expanding the therapeutic portfolio in a structured, evidence-informed manner as the disease evolves. This approach

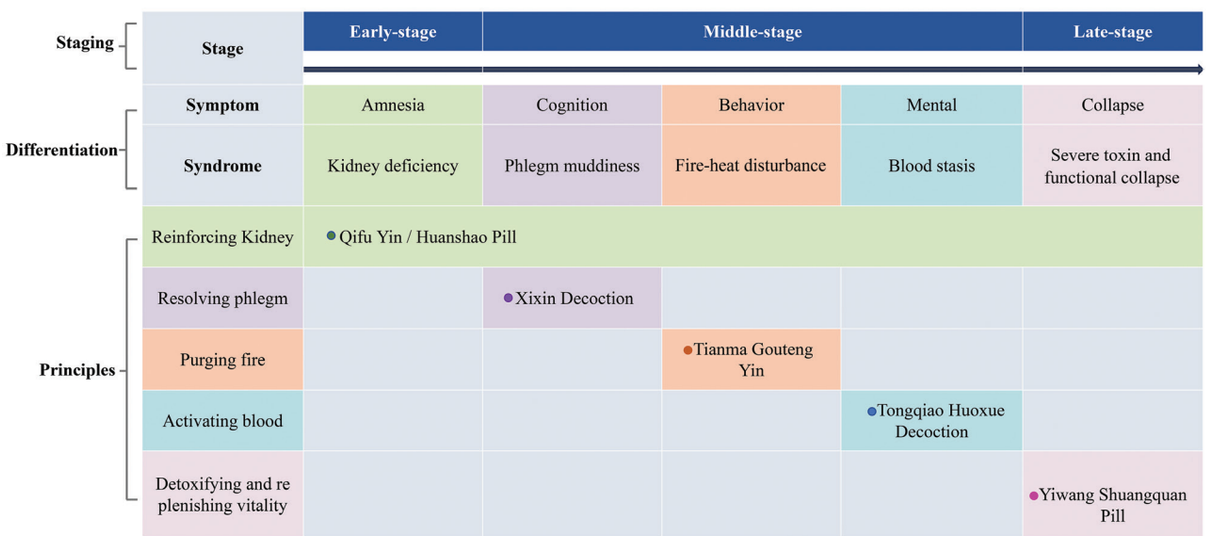


Figure 1. Sequential therapy for dementia (STEP regimen). STEP: Sequential Therapy for Evolving Patterns.

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differs fundamentally from conventional symptomatic management and single-target disease-modifying strategies.

Clinical evidence supporting this stage-adapted strategy continues to grow. In the early kidney deficiency–predominant stage, a 52-week randomized clinical trial demonstrated that Qinggong Shoutao Pill yielded significantly greater cognitive improvement than conventional therapy in patients with amnestic mild cognitive impairment, with the benefits sustained throughout the treatment period[13]. In the intermediate stage, as additional pathogenic cascades emerge, subtype-targeted interventions have demonstrated distinct efficacy. Specifically, phlegm-resolving granules improved cognitive function over 12 to 24 weeks in patients with the phlegm obstruction subtype, whereas Tianzhi granules produced superior cognitive outcomes over 6 months in patients with the fire-heat disturbance subtype[14]. In patients with prominent blood stasis, Ginkgo biloba extract EGb 761 enhanced cognition, behavior, and daily function over 22 to 24 weeks across multiple randomized clinical trials, improving a wide range of behavioral and psychological symptoms of dementia[15]. Moreover, real-world clinical data indicate that integrating sequential TCM with conventional care throughout AD progression yields sustained cognitive and behavioral improvements for at least 1 year[16]. Two comprehensive meta-analyses further reinforce these clinical observations. A meta-analysis of 23 randomized clinical trials involving 2,035 participants found that TCM formulations achieved significantly higher response rates and superior cognitive improvement compared with conventional monotherapy, as measured by the ADAS-Cog[17]. A systematic evidence map covering 187 clinical studies confirmed that herbal decoctions and acupuncture were the most extensively investigated modalities for AD[18]. This stage-adapted, multi-target strategy directly addresses a central limitation of single-target AD trials, specifically the flawed assumption that modulating a single pathway can effectively treat biologically heterogeneous and evolving disease processes.

Modern pharmacological mechanisms of TCM in AD

Pharmacological research has increasingly substantiated the clinical rationale for TCM-derived interventions for AD by elucidating their neuroprotective mechanisms. Beyond targeting the amyloid and tau pathways, these therapies engage the broader pathogenic network of AD, aligning with the multifactorial principles of TCM theory, as shown in Figure 2.

First, several well-characterized compounds are involved in core pathogenic pathways that are active throughout the AD continuum. Ginsenoside Rg1 upregulates the expression and enzymatic activity of the Aβ-degrading enzymes neprilysin and insulin-degrading enzyme while restoring aquaporin-4 polarization on astrocytic end feet to promote glymphatic clearance of interstitial solutes and facilitate the repair of meningeal lymphatic vessel function. Together, these mechanisms enhance the brain's intrinsic clearance capacity[19]. Curcumin directly binds Aβ oligomers and fibrils, inhibits their further aggregation, and promotes microglial phagocytic clearance of existing amyloid deposits[20]. Berberine inhibits beta-secretase 1 to reduce Aβ production and enhances autophagy-mediated clearance of aggregated proteins[21]. Icariin restores the activity of protein phosphatase 2A, the principal tau phosphatase downregulated in AD, thereby decreasing the hyperphosphorylated tau burden[22]. Salidroside reduces tau hyperphosphorylation through inhibition of GSK-3β and CDK5[23]. In the early stages of the disease, the predominant therapeutic mechanisms involve restoration of neurotrophic signaling and preservation of synaptic integrity. Ginsenoside Rg1 activates the BDNF-TrkB-CREB signaling axis, promoting hippocampal

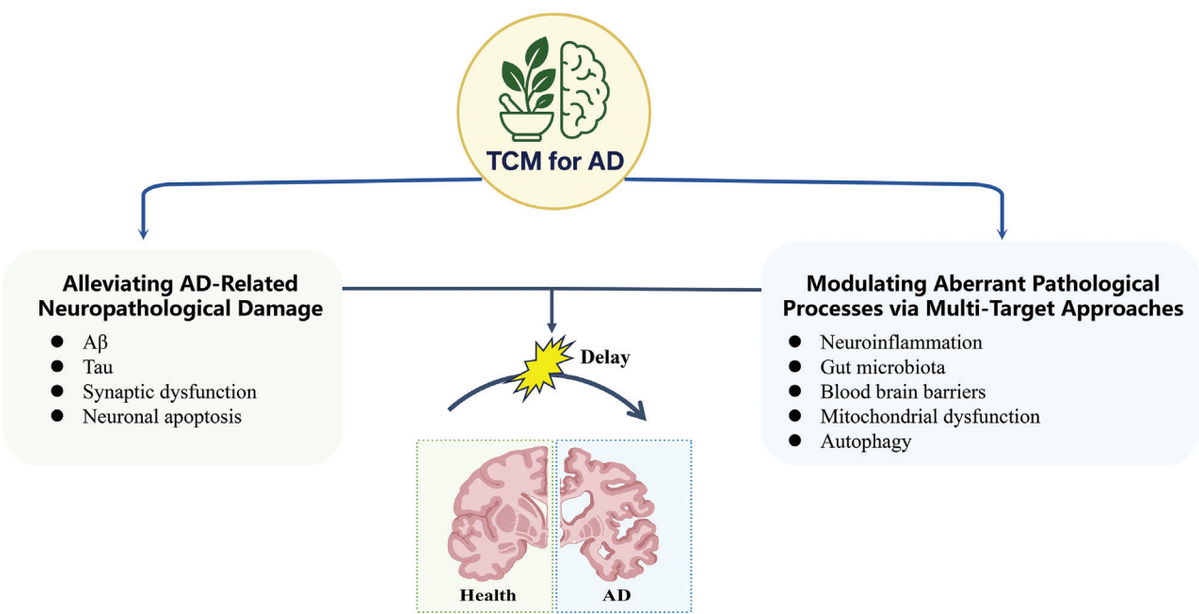


Figure 2. Multi-target pharmacological mechanisms of TCM in AD. AD: Alzheimer's disease; TCM: traditional Chinese medicine.

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neurogenesis and synaptic plasticity[19]. Polygalacic acid from *Polygala tenuifolia* enhances cholinergic transmission by inhibiting acetylcholinesterase activity while simultaneously upregulating the expression of choline acetyltransferase, thereby preserving cholinergic neurons that are among the earliest to degenerate in AD[24]. As the disease progresses to the middle stage, neuroinflammatory mechanisms become pathologically dominant. Cornuside, an iridoid glycoside from *Cornus officinalis*, ameliorates lipopolysaccharide (LPS)-induced cognitive dysfunction and inhibits microglial NOD-like receptor pyrin domain-containing 3 (NLRP3) inflammasome activation by enhancing Sirt1-mediated autophagy, reducing the maturation of interleukin (IL)-1β and IL-18[25]. Icariin alleviates cognitive dysfunction by reducing neuroinflammation through direct inhibition of the cGAS-STING signaling axis, reducing the phosphorylation of TBK1 and IRF3, and decreasing the expression of interferon-stimulated genes[26]. TCM-derived compounds regulate microglial polarization through multiple pathways, thereby restoring the disrupted neuroimmune balance in AD. At an advanced stage, the disease process is dominated by the activation of programmed neuronal cell death pathways. Bazi Bushen, a kidney-tonifying polyherbal formula, was recently shown to improve cognitive function in 5 × FAD mice by concurrently upregulating anti-apoptotic proteins, downregulating pro-apoptotic proteins, and attenuating hippocampal cellular senescence by reducing the expression of the senescence markers P21 and P16[27].

Notably, polyherbal formulations exert synergistic multi-pathway effects that transcend the actions of their individual constituents. The classic herb pair Yuanzhi-Shichangpu (*Polygala tenuifolia* and *Acorus tatarinowii*) restores blood-brain barrier integrity by upregulating the tight junction proteins ZO-1, occludin, and claudin-5 while improving mitochondrial bioenergetics in cerebral endothelial cells *via* the HIF-1α/GSK3β-Nrf2/PGC-1α signaling axis[28]. Similarly, various polyherbal formulations restore beneficial gut microbial taxa, including Lactobacillus and Bifidobacterium, suppress pro-inflammatory populations, and attenuate systemic inflammation through gut-brain axis modulation[29]. Collectively, this mechanistic evidence supports the premise that matching multicomponent pharmacological interventions to the dominant pathological mechanisms at each disease stage can achieve broader and more sustained therapeutic effects than single-target approaches.

Challenges and future directions

Despite a coherent theoretical foundation, promising clinical evidence, and an expanding mechanistic evidence base, the translational potential of TCM in AD remains poorly understood. The following three interconnected challenges require systematic attention. First, the quality of the clinical evidence must be improved. The most fundamental challenge in the whole-process management approach is the fragmentation of clinical evidence across the AD continuum. Research must transition from underpowered single-center studies to longitudinal cohort studies with embedded adaptive trials that track patients across disease stages and test the staged addition of therapeutic layers that incorporate objective biomarker endpoints. The Roman Consensus on Good Clinical Trials for TCM provides a timely methodological framework for this transition[30]. Second, chemical and pharmacological standardization are essential, as quality control is a prerequisite for accurate analysis of the molecular mechanisms of anti-AD effects. The botanical complexity of herbal preparations requires rigorous quality control, including high-resolution chemical fingerprinting of active formulations, standardized dosing regimens with defined pharmacokinetic profiles, and systematic evaluation of long-term safety, tolerability, and potential herb-drug interactions. Artificial intelligence (AI) provides an effective tool for addressing this challenge. AI-enabled network pharmacology can decode the complexity of multi-herbal formulations and disease-relevant biological networks on a scale that cannot be achieved using conventional experimental approaches[31]. Machine learning models trained on multiomics and clinical datasets can identify patient subgroups that are most likely to benefit from specific interventions. Natural language processing applied to classical TCM literature can systematically prioritize candidate formulas for modern evaluation. Third, the epistemological gap between TCM diagnoses and molecular disease staging remains unresolved. While the kidney deficiency cascade hypothesis provides a conceptual bridge, the empirical mapping of specific TCM patterns and defined biomarker profiles remains incomplete. Identifying biomarkers corresponding to each pattern-defined disease stage would create an empirically validated framework for cross-paradigm translation. Integrated data repositories that prospectively collect TCM pattern diagnoses, molecular biomarker profiles, neuroimaging data, and the long-term clinical outcomes of patients managed with sequential TCM therapy should be established. This infrastructure would enable retrospective and prospective analyses to refine stage transition criteria and optimize treatment algorithms.

AD is a systemic failure disorder that requires therapeutic strategies to address its underlying biological complexity. Grounded in a theoretical framework that conceptualizes disease progression as a dynamic, multistage process, TCM offers a stage-adapted, multi-target intervention that aligns with the modern understanding of AD as a multifactorial, heterogeneous syndrome. The classical pathogenic model, which encompasses kidney deficiency, phlegm muddiness, fire-heat disturbance, blood stasis, toxicity, and functional collapse, provides a structured nosology corresponding to the contemporary AD continuum. Its therapeutic range is supported by pharmacological and neuroscientific evidence demonstrating multipathway neuroprotection. Together, the kidney deficiency cascade hypothesis and its clinical translation as a STEP regimen constitute a coherent framework for longitudinal AD management that warrants rigorous evaluation against international evidence standards. Realizing this potential requires methodological rigor, biomarker-guided endpoints, chemical standardization, and open data sharing. These efforts may inform the rational design of future multi-target combination therapies for AD.

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Conflict of interest statement

The authors declare no conflict of interest.

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Author contributions

Jinzhou Tian, Jing Shi, and Lulu Yao conceived and designed the study. Lulu Yao wrote the manuscript. Jingnian Ni, Mingqing Wei, Ting Li, and Fuyao Li screened the literature and provided feedback on the drafts of the paper. Jinzhou Tian and Jing Shi reviewed the manuscript. All authors approved the final version of the manuscript.

Ethical approval of studies and informed consent

Not applicable.

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None.

Data availability

All data generated or analyzed during this study are included in this published article.

Declaration of generative AI in scientific writing

The authors declare that no generative artificial intelligence (AI) technology was used in the conception, drafting, or revision of this manuscript. All work and image creation were performed solely by human authors.

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