Review Article

Blood-Based Phosphorylated Tau Isoforms as Emerging Biomarkers for Alzheimer's Disease

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ABSTRACT

Alzheimer's disease (AD) is a progressive, currently incurable neurodegenerative disorder characterized by cognitive impairment. The neuropathology of AD is defined by two hallmark features: the extracellular deposition of amyloid-β (Aβ) plaques and the intracellular accumulation of neurofibrillary tangles composed of hyperphosphorylated Tau protein. Though cerebrospinal fluid (CSF) testing and imaging techniques such as positron emission tomography (PET) currently serve as diagnostic gold standards, CSF testing is invasive, and the latter is expensive and less accessible. Recent studies document the blood-based phosphorylated Tau (p-Tau) isoforms, particularly p-Tau181, p-Tau217 and p-Tau231, as promising next-generation noninvasive biomarkers for the early diagnosis of AD and disease monitoring. This review summarizes present evidence for clinical utility of blood-based p-Tau isoforms, specifically in terms of diagnostic validity, early detection ability, and ability to discriminate AD from other neurodegenerative disorders. Blood-based p-Tau testing holds the possibility to revolutionize AD diagnostic models, with prospects of far-reaching dissemination and early intervention.

Keywords: Alzheimer's Disease; Phosphorylated Tau; Blood-Based Biomarker; Cerebrospinal Fluid; Amyloid-β; Neurofibrillary Tangles; Diagnosis; Neurodegeneration

INTRODUCTION

Alzheimer's disease (AD) is a leading cause of dementia and a growing global public health concern. In 2019, it was estimated that over 55 million people were living with AD or related forms of dementia worldwide, and this number is projected to rise to 139 million by 2050 primarily due to population aging and increased life expectancy (1). AD is a progressive neurodegenerative disorder characterized by the gradual deterioration of cognitive functions, including diminished memory, disorientation and difficulty in reasoning (2). These symptoms profoundly impact on the daily lives of individuals affected by the disease and place significant emotional, physical, and financial burdens on families and caregivers.

The neuropathology of AD is defined by two hallmark features: the extracellular deposition of amyloid-β (Aβ) plaques and the intracellular accumulation of neurofibrillary tangles composed of hyperphosphorylated Tau protein. These pathological changes begin years, often decades, before the onset of clinical symptoms. Consequently, early

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and accurate detection of Aβ and Tau has become a central focus in AD diagnosis and monitoring. Current diagnostic methods primarily rely on neuroimaging and cerebrospinal fluid (CSF) analysis to detect these biomarkers (3). Positron emission tomography (PET) imaging can visualize both Aβ plaques and Tau tangles in vivo, which provide valuable insights into disease staging and progression. Likewise, CSF analysis enables quantification of soluble Aβ42 (which is typically decreased due to plaque deposition) and elevated levels of total Tau and phosphorylated Tau (p-Tau), reflecting neuronal injury and tangle pathology (Table 1). Although these diagnoses are highly informative and clinically validated, they are invasive and expensive. For example, obtaining CSF requires a lumbar puncture, an uncomfortable procedure with potential risks, which can limit its utility in routine clinical settings.

To overcome these limitations, growing research efforts have focused on the development of blood-based biomarkers that can capture the pathological signatures of AD. Among these, phosphorylated Tau (p-Tau), particularly isoforms such as p-Tau181, p-Tau217 and p-Tau231, has emerged as a promising biomarker candidate (4). These isoforms show strong concordance with CSF and PET-based measures of AD pathology, and their presence in blood opens the door to a more scalable, less invasive approach to early detection. This review examines the current advancements in blood-based p-Tau as a biomarker for AD, and its potential integration into clinical practice. It highlights how p-Tau testing could transform the current paradigm of AD diagnosis and monitoring, enabling earlier intervention, improving patient outcomes, and contributing to the global effort to reduce the burden of this devastating neurodegenerative disorder.

TAU / P-TAU IN CSF as biomarkers for AD

Tau proteins such as total Tau (t-Tau) and p-Tau in CSF serve as direct biochemical assessment of the pathological processes in brain and they are important biomarkers in the diagnosis of AD (5, 6). Tau protein plays a critical role in maintaining the structural integrity of neurons by stabilizing microtubules in axons. Under normal physiological conditions, Tau is highly soluble and minimally phosphorylated (7). However, in AD, Tau undergoes abnormal hyperphosphorylation which leads to its detachment from microtubules, loss of function and aggregation into neurofibrillary tangles (NFTs). These tangles disrupt intracellular transport, contribute to synaptic dysfunction, and ultimately lead to neuronal death (7). In CSF, elevated levels of t-Tau are generally interpreted as markers of neuronal and axonal injury, while p-Tau levels, phosphorylation sites at threonine 181 (p-Tau181), threonine 217 (p-Tau217) and threonine 231 (p-Tau231), are considered more specific indicators of AD pathology, closely linked to the formation and spread of NFTs (8-11).

Many studies have confirmed that t-Tau and p-Tau (p-Tau181, p-Tau217 and p-Tau231) in CSF are excellent biomarkers in the clinical diagnosis of AD (Table 2). For instance, Elevated p-Tau181 in CSF has been shown to differentiate AD from other neurodegenerative conditions such as Parkinson's disease dementia and Lewy body dementia, with high sensitivity and specificity (8). Recent studies suggest that p-Tau217 and p-Tau231 may offer even greater diagnostic accuracy, with improved ability to distinguish AD from other tauopathies and detect early-stage disease (9, 11). For instance, p-Tau217 has shown strong correlation with amyloid PET positivity and has been proposed as a robust predictor of progression from mild cognitive impairment (MCI) to AD dementia (12). In preclinical stages of AD, p-Tau levels in CSF begin to rise before significant cognitive decline is observable, making them valuable for early detection and risk stratification.

Despite the high diagnostic and prognostic value of

Table 1. Diagnostic methods for Alzheimer's disease, including CSF Aβ42/t-Tau/p-Tau, amyloid/tau PET, and plasma p-Tau

Method	Biomarkers Detected	Advantages	Limitations
CSF Lumbar Puncture	Aβ42, t-Tau, p-Tau181/217/231	High accuracy	Invasive, costly, requires expertise
PET Imaging	Aβ & tau tangles	Visualizes pathology in vivo	Expensive, limited access
Blood-based p-Tau	p-Tau181, p-Tau217, p-Tau231	Noninvasive, scalable, cost-effective	Validation ongoing

Summarizes targets, typical clinical utility, and major limitations (invasiveness, cost, accessibility). *Abbreviations:* Aβ, amyloid-β; PET, positron emission tomography; t-Tau, total tau; p-Tau, phosphorylated tau.

Table 2. Blood-based phosphorylated tau (p-Tau) isoforms as AD biomarkers: comparative strengths and stage of detection

Isoform	Strengths	Stage of AD Detection	References
p-Tau181	Correlates with Aβ & tau pathology; prognosis & differentiation from other dementias	MCI & symptomatic AD	13, 14, 15
p-Tau217	Higher sensitivity & specificity than p-Tau181; predicts progression	Early AD & MCI → AD dementia	16, 17, 18
p-Tau231	Detects earliest amyloid changes before tau deposition or cognitive decline	Preclinical AD	19, 20, 21

p-Tau181 correlates with Aβ and tau pathology and supports differential diagnosis and prognosis (most informative in MCI and symptomatic AD); p-Tau217 demonstrates higher sensitivity and specificity than p-Tau181 and tracks progression from early AD/MCI to dementia; p-Tau231 rises earliest with amyloid changes, enabling preclinical detection.

CSF-based p-Tau, their clinical application is limited by the invasive nature of lumbar puncture, potential side effects, and the need for specialized personnel and facilities, making it less accessible in primary care and low-resource settings.

Blood-based P-Tau181 as a biomarker for AD

The blood-based p-Tau181 has emerged as a highly promising non-invasive biomarker for the diagnosis and monitoring of AD. Recent studies have shown that plasma p-Tau181 levels strongly correlate with Aβ, tau pathology in the brain and neurodegeneration and cognitive decline. For instance, Janelidze *et al* studied plasma P-tau181 in three cohorts with a total of 589 individuals which include cognitively unimpaired participants, patients with mild cognitive impairment (MCI), AD dementia and non-AD neurodegenerative diseases (13). They found that plasma p-tau181 was increased at the MCI and dementia stages, which was well correlated with CSF p-tau181 and PET scans. Additionally, plasma P-tau181 can differentiate AD dementia from non-AD neurodegenerative diseases with an accuracy similar to that of Tau PET and CSF P-tau181 (13). High plasma p-tau181 was associated with subsequent development of AD dementia in cognitively unimpaired and MCI subjects, indicating that plasma P-tau181 is a noninvasive diagnostic and prognostic biomarker of AD.

In consistent, Mielke *et al* examined 269 participants consisting of 172 with cognitively unimpaired, 57 with MCI and 40 with AD dementia (14). All participants underwent brain PET imaging, MRI, and blood testing for total tau and p-Tau181. They reported that both plasma total tau and p-Tau181 levels were higher in patients with AD dementia than in cognitively unimpaired individuals. Particularly, plasma p-Tau181 showed a notably stronger association with both Aβ and tau PET imaging compared to total tau with greater sensitivity and specificity in predicting elevated brain Aβ levels than plasma total tau.

Moreover, Thijssen *et al*. examined whether p-Tau181 could differentiate between clinically diagnosed or autopsy-confirmed AD and frontotemporal lobar degeneration (15). They found that plasma p-Tau181 concentrations were increased by 3.5-fold in AD compared to controls and differentiated AD from both clinically and autopsy-confirmed frontotemporal lobar degeneration. Plasma p-Tau181 successfully identified individuals with amyloid β-PET positivity, independent of their clinical diagnosis, and showed a strong correlation with cortical tau accumulation as measured by PET imaging.

In summary, these findings suggest that plasma p-Tau181 could serve as a useful biomarker for detecting AD's-related brain changes and may be valuable as a noninvasive screening tool for identifying individuals with AD.

Blood-based P-Tau217 as a biomarker for AD

Similar to p-Tau181, p-Tau217 has emerged as a promising blood-based biomarker for AD, and it demonstrates superior sensitivity and specificity in AD diagnosis. While both p-Tau181 and p-Tau217 reflect AD-related neurofibrillary changes, studies have shown that p-Tau217 more accurately capture the presence and progression of Aβ and tau pathology in the brain, particularly during the early stages of AD development.

Palmqvist *et al*. conducted a large comparative study with enrollment of 1,402 participants from three selected cohorts: AD patients, patients with non-AD

neurodegenerative conditions (such as frontotemporal dementia), and cognitively healthy controls (16). They found that plasma p-Tau217 levels were significantly elevated in AD patients and displayed stronger correlations with Aβ-PET and tau-PET imaging results compared to p-Tau181. More importantly, p-Tau217 achieved a higher diagnostic accuracy in distinguishing AD from non-AD neurodegenerative diseases, particularly frontotemporal lobar degeneration which often presents similar clinical features but distinct underlying pathology from AD.

Meanwhile, Mattsson-Carlgren *et al*. reported a longitudinal study in two cohorts, 150 cognitively unimpaired participants and 100 MCI patients, followed for up to 6 years with repeated p-Tau217 measurements (17). They showed that plasma p-Tau217 levels begin to rise early in the AD disease process, even prior to symptom onset, and increase with amyloid deposition and subsequent tau accumulation in the brain (17). Notably, MCI patients who progressed to AD dementia exhibited accelerated p-Tau217 increases, while no significant change was observed in MCI patients who did not develop AD dementia or amyloid-negative participants. Longitudinal p-Tau217 elevations were associated with cognitive decline and brain atrophy, supporting blood-based p-Tau217 as a noninvasive biomarker with strong potential not only for diagnosing AD but also for predicting and monitoring AD disease progression.

Mendes *et al*. evaluated the diagnostic accuracy of plasma and CSF levels of three phosphorylated tau isoforms, p-tau217, p-tau181 and p-tau231, in identifying AD (18). The study included 114 participants, comprising 33 cognitively unimpaired individuals, 67 with MCI, and 14 diagnosed with dementia. These biomarkers were compared against continuous measures derived from amyloid and tau-PET scans. They found that both plasma and CSF p-tau217 exhibited stronger correlations with amyloid and tau PET imaging than the other p-tau variants. Specifically, plasma p-tau217 showed the highest diagnostic accuracy, significantly outperforming p-tau181 and p-tau231 in distinguishing between diagnostic groups and identifying PET positivity. These results highlight the superior performance of plasma p-tau217 as a diagnostic marker for AD. The findings suggest that plasma p-tau217 could become an important non-invasive tool for diagnosing and screening for AD, potentially reducing the need for more invasive procedures like CSF collection or PET imaging.

Collectively, these findings provide strong evidence that blood-based p-Tau217 is a highly sensitive and specific biomarker for AD. Its superior performance in early detection, disease staging, and differentiation from other neurodegenerative disorders positions it as a crucial tool in the AD diagnosis and prognosis.

Blood-based P-Tau231 as a biomarker for AD

Among the phosphorylated tau isoforms, p-Tau231 has gained increasing attention as an early and specific blood-based biomarker for Alzheimer's disease (AD). Unlike p-Tau181 and p-Tau217, which rise in response to both amyloid and tau pathology, p-Tau231 appears to be particularly sensitive to early Aβ changes, often preceding substantial tau deposition and measurable cognitive decline

Ashton *et al*. studied 171 participants, including individuals with AD, non-AD neurodegenerative diseases, and healthy controls(19). Their findings have demonstrated that plasma p-Tau231 levels are elevated in preclinical AD and are strongly correlated with Aβ-PET burden, even before tau-PET signals become detectable. Importantly, p-Tau231 outperformed p-Tau181 and p-Tau217 in identifying early Aβ pathology, suggesting its unique value in detecting AD at its earliest stage. Consistently, Janelidze *et al*. expanded on these findings in a comprehensive study involving 1,057 participants from the Swedish BioFINDER I and II cohorts, encompassing cognitively unimpaired individuals, MCI patients, AD dementia cases, and those with non-AD neurodegenerative diseases (20). The study measured plasma p-Tau217 in conjunction with tau-PET imaging and directly compared plasma levels of p-Tau231, p-Tau217, and p-Tau181 across the Alzheimer's disease continuum within the same cohorts (20). Notably, p-Tau231 levels were significantly elevated in cognitively unimpaired individuals who were Aβ-positive, identifying p-Tau231 as one of the earliest plasma markers to reflect amyloid pathology. This high sensitivity and early rise in plasma p-Tau231 underscore its potential as a valuable biomarker for detecting individuals at risk of AD prior to symptom onset. Moreover, Ferreira *et al*. analyzed plasma p-Tau231, p-Tau217+, p-Tau181 in a cohort of 225 participants, including 138 cognitively unimpaired and 87 cognitively impaired individuals (21). In the cognitively unimpaired group, plasma p-Tau231 and p-Tau217+ significantly improved demographic models for detecting Aβ-PET positivity, while no plasma biomarker enhanced detection of tau-PET positivity

at this early stage. Conversely, among impaired participants, p-Tau217+ and GFAP markedly improved identification of both Aβ and tau pathology beyond demographic factors, whereas p-Tau231 provided additional predictive value specifically for tau-PET positivity. These results suggest that plasma p-Tau231, as well as p-Tau217+ are sensitive indicators of early Aβ deposition, with their diagnostic utility evolving in later disease stages to reflect tau tangle accumulation.

In summary, these studies indicate that plasma p-Tau231 serves as a highly sensitive early indicator of amyloid pathology and is well-suited for preclinical AD detection. When used alongside other p-Tau isoforms, p-Tau231 can enhance the diagnostic precision and staging of AD. Its ability to detect pathological changes before the onset of clinical symptoms reinforces its value in early screening, clinical trial enrollment, and potential therapeutic intervention.

CLINICAL STUDIES OF P-TAU AS A BIOMARKER OF AD

To date, two clinical trials have been initiated to investigate blood-based p-Tau as a biomarker of AD (Table 3). The first clinical trial launched in July 2022 is a multicenter, non-interventional study currently recruiting participants from memory clinics in Montpellier, Nîmes, and Perpignan, France (NCT05427448). The trial is designed to determine whether blood-based biomarkers, including p-Tau181, Aβ (1-40), Aβ (1-42) and total tau, can serve as reliable biomarkers for Alzheimer's pathology by assessing the diagnostic accuracy of plasma biomarkers in comparison with CSF-based measures and multidisciplinary clinical diagnosis. Additional goal of this clinical trial is to examine if these plasma biomarkers can differentiate normal from pathological CSF profiles and to detect other neurodegenerative disorders, such as frontotemporal dementia and Lewy body disease. This trial planned to enroll 342 patients and will provide critical evidence supporting the use of blood-based p-Tau as a less invasive and more accessible diagnostic alternative to CSF sampling.

The second clinical trial, initiated in January 2024, focuses on evaluating plasma p-Tau217 as a biomarker for AD at the memory center of CHU Amiens, France, and is expected to conclude in June 2026 (NCT07071649). This study is designed to directly compare plasma p-Tau217 with established CSF amyloid and tau biomarkers in patients undergoing diagnostic evaluation for suspected AD. Approximately 40 participants, all aged 18 years or older and undergoing lumbar puncture as part of routine clinical workup, have been enrolled.

Together, these two trials represent complementary efforts to validate plasma-based p-tau isoforms as robust diagnostic markers of AD. If their projected outcomes are achieved, these studies could pave the way for less invasive, more accessible diagnostic tools that would expand early detection, reduce reliance on lumbar puncture, and improve patient stratification for therapeutic treatments.

CONCLUSION

Diagnosis of AD today depends on invasive CSF testing and costly PET imaging. Recently, blood-based p-Tau isoforms, including p-Tau181, p-Tau217 and p-Tau231, have emerged as promising noninvasive biomarkers for AD diagnosis and prognosis. This review highlights their capacity of diagnostic potentials to identify AD in its earliest stages and to distinguish it from other neurodegenerative diseases, thus offering diagnostic methods that is more accessible, scalable, and cost-effective.

Each p-Tau isoform possesses its own strengths: p-Tau181 is strongly correlated with Aβ and tau pathology and assists in AD differentiation and prognosis. P-Tau217 possesses higher sensitivity for early AD, and tracks disease progression with high

Table 3. Clinical Studies on Blood-Based Phosphorylated Tau Biomarkers for Alzheimer's Disease

Study	Biomarker	Design & Enrollment
NCT05427448	Plasma p-tau181, Aβ (1-40), Aβ (1-42), total tau	Prospective, non-interventional; ~342 participants from memory clinics
NCT07071649	Plasma p-tau 217	Interventional; ~40 suspected AD patients undergoing CSF testing

accuracy. P-Tau231 is best at detecting preclinical AD as it responds early to Aβ changes even prior to other tau markers or cognitive decline. Together, these blood-based p-Tau isoforms provide a full toolkit for the early and accurate diagnosis and staging of AD. Currently clinical trials (NCT05427448 and NCT07071649) of blood-based p-Tau biomarkers are ongoing. , Successful validation would pave the way for large-scale adoption, enabling earlier diagnosis, more effective patient stratification for clinical trials, and timely therapeutic interventions, advances that could fundamentally transform the management of AD.

CONFLICT OF INTERESTS

The author declares no conflicts of interest related to this work.

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