# Article information:

Relevance of the interplay between amyloid and tau for cognitive impairment in early Alzheimer's disease - ScienceDirect  
<https://www.sciencedirect.com/science/article/abs/pii/S0197458019301010>

# Article summary:

1. Amyloid β and tau are key hallmark features of Alzheimer's disease neuropathology.

2. Cognitive/memory performance correlated well with cerebrospinal fluid tau levels across early stages of AD, although the correlation is Aβ dependent.

3. The interplay between Aβ and tau may strengthen the interaction between both key features of the amyloid cascade hypothesis already at the start of the AD continuum.

# Article rating:

Appears moderately imbalanced: The article provides some useful information, but is missing several important points or pieces of evidence that would be required to present the discussed topics in a balanced and reliable way. You are encouraged to seek a more balanced perspective on the presented issues by exploring the provided research topics and looking at different information sources.

# Article analysis:

The article titled "Relevance of the interplay between amyloid and tau for cognitive impairment in early Alzheimer's disease" discusses the relationship between amyloid beta (Aβ) and tau, two hallmark features of Alzheimer's disease neuropathology. The article examines the interplay of Aβ and tau for cognitive impairment in early AD using cross-sectional analysis, measured by cerebrospinal fluid biomarkers (Aβ1–42, total tau [t-tau], and phosphorylated tau [p-tau181P]), and on cognitive performance by the repeatable battery for assessment of neuropsychological status (RBANS).

The article provides a comprehensive overview of the current understanding of AD pathology and its progression. It highlights the importance of Aβ as the initial pathological trigger in the disease continuum, interacting with tau to form NFT leading to widespread neuronal degeneration and dysfunction, cognitive decline, and dementia. The article also discusses genetic evidence from rare familial forms of AD as well as mutations in amyloid-β precursor protein and presenilins 1 and 2 genes that support the accumulation of Aβ as the causative factor for AD.

However, there are some potential biases in this article. For example, while it acknowledges that mutations in tau are not causative for AD but may result in dementia without an AD clinical or neuropathological phenotype, it does not explore this point further or consider how it might impact our understanding of AD pathology. Additionally, while it discusses preclinical evidence supporting a possible interplay between Aβ and tau, it does not provide any counterarguments or explore alternative hypotheses.

Furthermore, while the study found that cognitive/memory performance correlated well with CSF tau levels across early stages of AD, it notes that this correlation is Aβ dependent. However, it does not provide any evidence to support this claim or explore potential mechanisms underlying this relationship.

Overall, while this article provides a useful overview of the current understanding of AD pathology and its progression, it could benefit from a more balanced discussion of potential biases and alternative hypotheses. Additionally, it would be helpful to provide more evidence to support some of the claims made in the article.

# Topics for further research:

* Alternative hypotheses for the pathogenesis of Alzheimer's disease
* Role of inflammation in Alzheimer's disease progression
* Genetic risk factors for Alzheimer's disease beyond amyloid and tau
* Mechanisms underlying the correlation between CSF tau levels and cognitive impairment in AD
* The impact of non-AD tauopathies on dementia
* The role of synaptic dysfunction in Alzheimer's disease pathology

# Report location:

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