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Resumo	Alzheimer's disease (AD) is characterized by neuronal loss, attributed to amyloid-beta (Aβ) aggregation and accumulation. The autophagy-lysosomal pathway, including cathepsins B and D, is crucial for protein degradation and clearance, but it is impaired in some diseases. This review summarizes current knowledge on the dysregulation of this pathway in AD. Accumulating evidence suggests that Aβ overload impairs autophagy-lysosomal function and cathepsin activity, exacerbating Aβ accumulation and neurodegeneration. However, the precise mechanisms underlying these interactions remain elusive. Despite these challenges, targeting the lysosomal pathway emerges as a promising therapeutic strategy, and a comprehensive understanding of the autophagy-lysosomal system is essential to develop effective interventions for AD.
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