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Sommario/riassunto	<p>The incidence and prevalence of Alzheimer's Disease are increasing worldwide due to aging and comorbidities. The classical neuropathogenetic model, which concentrates on brain amyloid plaque deposition, neurofibrillary tangles, and cholinergic system dysfunction, is being expanded and integrated with newer molecular models. Understanding the relationships between the classical pathogenetic pathways and newer hypotheses is imperative for guiding future research and developing effective drugs. The existence of newer molecular mechanisms such as risk factors, comorbidities like vascular diseases and diabetes mellitus, and neuroanatomical/neurofunctional alterations as blood-brain barrier breakdown and neurovascular unit dysfunction, offer potential targets to improve Alzheimer's Disease diagnosis and treatment. This reprint provides an overview of the pathophysiologic role of newer and older molecular mechanisms of Alzheimer's Disease, shedding light on new insights for the development of new therapeutic approaches. It also highlights newer candidate markers of disease onset and progression.</p>