



Alzheimer's disease: From Molecular Basis to Therapy

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Message from the Guest Editors

Dear Colleagues,

Alzheimer's disease (AD) manifests as a chronic deterioration in cognition, primarily in memory, resulting from neuronal loss in specific regions of the brain. Genetics and pathology suggest that beta-amyloid build-up is the key process in the disease, but efforts to pharmaceutically reduce beta-amyloid have not resulted in an expected slowing of cognitive decline. Assuming that amyloid reduction is insufficient for therapeutic relief, other targets are required, and this necessitates new knowledge on AD pathogenesis. In this respect, the increasingly complex multidimensional molecular (multi-omic) data being generated from clinical samples and AD models are both exciting and extremely challenging. The scope of this Special Issue is an exploration of the different approaches that research groups are using to narrow this knowledge gap around AD pathogenesis. Authors are invited to submit original research or review articles which address linking molecular studies to novel therapeutic avenues or preventive strategies for AD.





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Message from the Editor-in-Chief

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