香港中文大學化學系
Department of Chemistry
THE CHINESE UNIVERSITY OF HONG KONG

The Chinese University of Hong Kong

Department of Chemistry Research Seminar Series

Battling Alzheimer's Disease by Controlling Amyloid Aggregation Pathways

Professor Jerry C. C. CHAN

Department of Chemistry
National Taiwan University
Taiwan

17 April 2026 (Friday)
4:30 PM
SC L3

Contact Person:
Professor Fuk Yee KWONG

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Jerry C. C. Chan

Department of Chemistry, National Taiwan University, Taiwan, R.O.C.

The aggregation of amyloid-β peptides is a central molecular event in Alzheimer's disease, yet the early oligomeric species believed to be most pathogenic remain difficult to isolate and characterize because of their transient nature and structural heterogeneity. In this talk, I will describe how nanoscale confinement can be exploited to actively control amyloid-β aggregation pathways and to access oligomeric assemblies that are otherwise inaccessible in bulk solution. By restricting peptide aggregation within confined nanoscale environments, fibril-associated nucleation processes can be suppressed, enabling the preparation of amyloid-β oligomers with well-defined sizes and improved structural homogeneity.

I will show that amyloid-β oligomers prepared under confined conditions exhibit size-dependent aggregation behavior, distinct structural features, and different dynamical properties, as revealed by kinetic analyses and solid-state NMR spectroscopy. Building on these structurally defined oligomeric states, we have developed DNA aptamers that recognize amyloid-β oligomers with high sensitivity. These aptamers provide a complementary molecular recognition strategy to antibodies and offer potential advantages in stability and assay reproducibility, highlighting their promise for diagnostic applications. Together, these results demonstrate how controlled confinement enables direct interrogation of early amyloid-β assembly events and supports the development of oligomer-targeted probes relevant to Alzheimer's disease.