

## **Internal Webinar**

### **Targeting Protein Condensates in Alzheimer's Disease: Mechanistic and Cellular Insights into LLPS and Its Modulators**

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Alzheimer's Disease is marked by the aggregation of misfolded proteins, mainly amyloid-beta ( $A\beta$ ) and tau, forming amyloid plaques and neurofibrillary tangles. Both intrinsically disordered tau and structured  $A\beta$  can undergo LLPS under certain conditions. Cellular models provide a dynamic environment to dissect these phenomena in physiologically relevant settings, offering deeper insights into spatiotemporal regulation of condensate dynamics, aggregation propensity, and cytotoxicity. In this proposal, we aim to elucidate the regulatory role of LLPS and PTMs on the aggregation behavior and toxicity of tau and  $A\beta$ . Using cellular systems, we will explore the induction of LLPS under disease-mimicking stress conditions, monitor their progression to pathological aggregates and evaluate the therapeutic potential of small-molecule modulators in disrupting aberrant LLPS and ameliorating AD-related toxicity. Together, these studies will contribute to a mechanistic framework for understanding the LLPS landscape in AD and inform novel therapeutic strategies targeting condensate biology.

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***14:30 Hrs***

