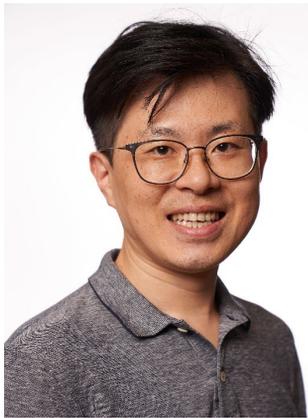




Fri, 10 Oct 2025 | 10 am | S3-05-02 Conference Room 1

Hosted by Assistant Prof Lin Zhewang

Mechanisms of protein aggregation and condensation: stalled nascent polypeptides and RNA-free TDP-43



By Young-Jun Choe

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About the Speaker

The speaker, Young-Jun Choe, received both his BSc and PhD degrees from Seoul National University, Korea. During his PhD, he investigated yeast prions and demonstrated how the soluble prion protein Sup35 initiates aggregation. After completing his doctorate, he moved to Germany to pursue a postdoctoral fellowship at the Max Planck Institute of Biochemistry under the supervision of Prof. Ulrich Hartl, a pioneer in chaperone-mediated protein folding. There, Dr. Choe initiated studies on co-translational protein quality control, focusing on ribosomes stalled during translation of defective mRNAs. In 2018, he established his independent laboratory at Nanyang Technological University as a Nanyang Assistant Professor.

Translation of defective mRNAs can cause ribosome stalling, generating incomplete and potentially toxic proteins. The ribosome-associated quality control (RQC) pathway detects stalled ribosomes and directs their incomplete products for degradation. RQC is a remarkably sensitive surveillance system, capable of detecting rare stalled ribosomes among a vast pool of actively translating ribosomes. However, this high sensitivity raises the question of whether RQC also targets ribosomes that pause transiently during normal mRNA translation. I will present our discovery that RQC is inhibited on transiently paused ribosomes, thereby preventing unnecessary protein degradation.

In the second part of my talk, I will discuss the unusual phase separation of the protein TDP-43. Aggregation of TDP-43 is a hallmark of amyotrophic lateral sclerosis (ALS), and phase separation is thought to precede the formation of these toxic aggregates. I will present our findings on how molecular chaperones interact with TDP-43 condensates, thereby remodeling their architecture.