東北大学 学際科学フロンティア研究所

学際融合グローバル研究者育成東北イニシアティブ

FRIS/TI-FRIS

第 21 回 FRIS ハブミーティング

第 5 回 TI-FRIS ハブミーティング

Hub Meeting

Recent understanding of disease-causing protein aggregation

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Nascent proteins fold to native structures for gain-of-function in soluble states. Failure of keeping native structures often induces protein aggregation with loss-of-function and gain-of-disease such as Alzheimer's disease (AD), Parkinson's disease (PD), type 2 diabetes mellitus (T2DM), and so on. Despite of numerous efforts, much remains to be elucidated on underlying principles of protein misfolding and aggregation from the microscopic and macroscopic point of view. Today, I will first show the microscopic molecular mechanisms of protein aggregation, amyloid fibrillation and amorphous aggregation, with the general description on the structures of amyloidogenic precursors and amyloid fibrils. Aggregation of several amyloid proteins including Alzheimer`s amyloid β peptides in AD, α-synuclein in PD, and insulin in T2DM will be explained based mostly on my previous and on-going studies. Macroscopic viewpoints including solubility, supersaturation, and phase diagram for amyloid formation will be next given with a new calorimetry and NMR-based assay. Several case studies which aimed at inhibiting toxic amyloid aggregation will be also addressed.

オンライン開催（Zoom）

2021. 5. 27

11:00 -12:00

参加登録

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FRIS Hub Meeting HP
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