The Biological and Biomedical Joint Seminar Series

(Hosted by the departments of Molecular & Cellular Biology, Chemistry & Biochemistry, Cellular & Molecular Medicine, and Plant Sciences)

"Probing the mechanism of inhibition of amyloid induced neurotoxicity by the chaperonin GroEL "

Marielle Wälti/Hegetschweiler

Assistant Professor
Chemistry & Biochemistry
The University of Arizona

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Hosted By: Pascale Charest (MCB)

Alzheimer's disease (AD) is a fatal neurodegenerative disease affecting 5.5 million Americans. Despite many decades of research there is still no known cure. AD is a protein misfolding disease, where the Alzheimer's protein, A□, aggregates from a random coil entity into fibrils. However, the nature of the toxic species in Alzheimer's disease remains unknown. More and more attention has been given to the possibility that A□ aggregates within mitochondria, rather than extracellular deposits of A□, may be responsible for the onset and progression of the disease. Nature has developed mechanisms to prevent disease-associated protein aggregation, e.g. by the introduction of heat shock proteins (Hsp's), which are overexpressed when cells undergo stress. The most important Hsp's in mitochondria are Hsp60 and Hsp70, whereas Hsp60 is the only essential chaperone in bacteria, yeast, and mammals. Here we study a potential mechanism how Hsp60 might slow down amyloid fibril formation and therefore reduces toxicity in neurons.

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