



Psychiatry and Neuroscience Seminar Series

Dr Ronald MELKI



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Prion-like propagation of alpha-synuclein assemblies in Parkinson's disease and other synucleinopathies

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Room R04-45, 102-108 rue de la santé - 75014 Paris

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The accumulation of protein aggregates within the central nervous system is the hallmark of several progressive neurodegenerative disorders (e.g. Alzheimer, Parkinson, Huntington and Creutzfeldt-Jacob diseases). These aberrant protein conformers are the consequence of the misfolding and aggregation of specific proteins. We demonstrated recently that the protein aggregates that are tightly associated to pathology spread from cell to cell in a prion-like manner reminiscent of PrP. The goals of our research are multiple: 1/ Decipher the molecular events leading to the aggregation of polypeptides that propagate from one cell to another and amplify by recruiting the soluble form of their constituting polypeptides, 2/ document the role of the players that maintain protein homeostasis within the cells, as the protein aggregates we are interested in form upon impairment of this cellular process, 3/ characterize the “functional” properties of the different high-molecular weight oligomeric species that form during assembly, and 4/ identify the routes used by these protein assemblies to spread from one cell to another, using a combination of cell biological, biochemical and biophysical methods.