Mathematical Biology Seminar

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3 pm – 657 CAB

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Stochastic Model of Prion's Disease: Nucleation-Polymerisation Model

The formation of fibrillar protein aggregates, often called amyloid fibrils, is a central feature of many human diseases, including Alzheimer's disease, Parkinson's disease and spongiform encephalopathies. It is generally accepted that spongiform encephalopathies result from the aggregation of a ubiquitous protein, the so-called prion protein, into amyloids. As a consequence, the dynamics of amyloid formation should explain the characteristics of the prion disease: infectiosity as well as sporadic and genetic occurrence, long incubation time, species barriers and strain specificities. The success of this amyloid hypothesis is due to the good qualitative accordance of this hypothesis with the observations. However, a number of difficulties arose when comparing quantitatively the in vitro obtained experimental results with the model, suggesting that some differences should hide important discrepancies.

I will consider some of the characteristics of the spongiform encephalopathies and will discuss some of the classical models of this disease and their limits. Finally, I will expose a stochastic model which aims to propose an additional on-path reaction that takes place before nucleation and that is responsible for the heterogeneity of structures generated during prion protein polymerization in vitro.

Join us for refreshments in CAB 549 immediately following the Seminar