ECMTB 2014
Modeling of Protein Aggregation and Transmission in Amyloid Diseases
Tuesday, June 17th from 1600 - 1730

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Amyloid diseases have a rich and fascinating history; the once heretical prion hypothesis has now become accepted biology and has connected a number of widely different diseases to a common biochemical process. Because of the diversity of amyloid diseases, mathematical models and approaches have been developed in distinct disease contexts. With this minisymposium, we intend to facilitate a multifaceted discussion of amyloid disease and transmission.

The Broad Diversity of Amyloid Dynamics

The central dogma of biology stipulates that DNA encodes RNA which encodes proteins. The shape - or confirmation - of a protein is highly connected to its function and was thought to be primarily encoded by biophysical constraints on the possible amino acid configurations. Amyloids represent an important departure from this common pathway because they are capable of aggregating into complexes of misfiled proteins.

Amyloid proteins are responsible for a large number of diseases such as Alzheimers, Huntingtons and Parkinsons disease in humans. In addition, prion diseases represent special class of protein aggregation disorders which spread through a population - cells or individuals - through a phenomena of protein only inheritance.

Minisymposium Speakers

- 1600 - 1630: Dr. Carola Kruse, INRIA
  Title: Investigation of a Nucleated-Polymerization Model applied to Polyglutamine Aggregation
- 1630 - 1700: Mr. Jason K. Davis, University of California, Merced
  Title: Enzyme-Limited Nucleated Polymerization Model of Prion Aggregation
- 1700 - 1730: Dr. Suzanne S. Sindi, University of California, Merced
  Title: Mathematical Modeling of Prion Protein Dynamics and Transmission in Yeast