



Protein aggregation, disease, milk and molecular chaperones

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Place » 4F seminar room

Abstract: *In vivo*, protein aggregation and precipitation are often a consequence of the unfolding or incorrect folding of proteins. These processes have been categorised by the term 'protein misfolding' that can result from the formation of highly structured entities known as amyloid fibrils. A diversity of diseases result from protein misfolding including cataract, Alzheimer's, Parkinson's and Huntington's diseases and haemodialysis-related amyloidosis. Different proteins are associated with each of these diseases. There are a variety of cellular mechanisms that minimise protein aggregation. Arguably, the most important of these is the expression of molecular chaperone proteins which interact with partially folded proteins to prevent their aggregation.

Mammalian milk has similarities to the above scenario. Milk is characterised by the presence of large protein aggregates known as the casein micelle. Micelle structure is maintained by the molecular chaperone action of caseins to prevent the inherent propensity of some of the casein proteins to form amyloid fibrils.

In this talk, some of our work investigating the structure and function of molecular chaperone proteins, particularly small heat-shock and casein proteins, and their interactions with aggregating proteins, will be described. Discussion of our investigations of small molecule inhibitors of amyloid fibril formation will also be presented.

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