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Expert Visit
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Protein Folding, Misfolding, Aggregation and Disease

More info: https://biologicalsciences.leeds.ac.uk/biological-sciences/staff/127/professor-sheena-radford

Watching Amyloid Form: From Test Tubes to Tissues

Many amyloid precursors are intrinsically disordered initially, yet fold to highly organised cross-β structures during amyloid formation. How this conformational transition occurs structurally is not clear, with the initiating steps in aggregation being difficult to study because of the dynamics and heterogeneity of the species involved. It is also clear that the energy landscape for aggregation into amyloid results in potentially many different amyloid folds. In this presentation I will discuss these concepts, drawing on recent results from our laboratory on the amyloidogenic proteins islet associated polypeptide (IAPP) involved in type 2 diabetes, α-synuclein in Parkinson's disease and Aβ in Alzheimer's disease. I will show how by combining kinetic analysis of amyloid assembly with structural analysis of fibril assembly *in vitro* and *in situ* we are beginning to link the pathway of structural conversion from the initial unfolded monomer to the cross-β amyloid fold. The insights are fuelling our quest to better understand the link between the structures of amyloid assemblies and the onset of cellular dysfunction and disease.

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