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Yeast assay for amyloid aggregation in proteopathies

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Amyloid proteins (including transmissible amyloids, prions) cause heritable, sporadic and infectious diseases in humans. Formation of the amyloid fibril is postulated to occur through a two-step process. First, the normal soluble protein is converted into small aggregates or nuclei of the prion isoform of that protein by a process called nucleation. Second, these nuclei seed the conversion of protein molecules containing the same or similar amino acid sequence thereby sequestering them into long fibrils. A similar molecular mechanism is employed by yeast prions, which are not homologous to known mammalian amyloid and prion proteins by sequence, and control heritable traits. We have developed a yeast-based assay that allows us to study the initial nucleation mechanism of any mammalian amyloidogenic protein. Here, we show that chimeric proteins composed of Sup35 fragments, including prion-forming domain and fused to aggregation-prone regions of mammalian prion protein (PrP), human amyloid beta (associated with Alzheimer's disease), human α-synuclein (associated with Parkinson's disease), human amylin (associated with type II diabetes), or the M-region of tumor suppressor protein 53 (associated with many forms of cancer), nucleate new Sup35 prions even in the absence of of the Rnq1 prion or any other pre-existing nuclei. Our data indicate that prion/amyloid properties of mammalian amyloidogenic proteins that are detected in yeast and mammalian (or *in vitro*) systems are controlled by the same sequence elements.

Biography

Zachery Deckner has completed his BS in Biology from GCSU and is currently a PhD student at Georgia Tech. He is working on identifying and studying new amyloidogenic proteins implicated in various proteopathies. He has mentored undergraduuate students and wants to take up a teaching career after his graduation.

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