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Predicting Prion Propensity of Human Proteins

Cascarina S; Ross E.

Abstract

In humans only a single prion-forming protein named PrPc (for " cellular prion protein") is currently known, yet many more neurodegenerative disorders involve aberrant protein aggregation. The classical model for these diseases has involved cell-autonomous aggregation, assuming that aggregation occurs independently in each cell within a diseased patient. However, more recent models have proposed a non-cell-autonomous progression of disease in which aggregates formed in one cell may be transmitted to neighboring cells. These aggregate seeds then cause aggregation of the soluble protein in the " infected" cells, similar to the prion diseases. Within the past few years, a number of proteins that exhibit prion-like aggregation and spread to neighboring tissues have been discovered in patients with Amyotrophic Lateral Sclerosis (ALS). Although ALS has been studied for a number of decades, these proteins were only recently linked to ALS by chance. This demonstrates a clear need for an accurate method to systematically identify additional proteins that may play a pathological role in neurodegenerative disorders.

Keywords:

Taking advantage of the compositional similarity of these proteins to the known yeast prions