Abstract Submitted
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Hunt for the Mysterious Prion Self-Replicator[1] OLGA HARRINGTON[2], University of South Florida — Amyloid diseases such as Alzheimer's disease and type II diabetes are characterized by the self-assembly of partially denatured proteins into micron-sized rigid fibrils. There is increasing evidence that amyloid diseases share important similarities with prion diseases, such as Mad Cow Disease. Our own laboratory has recently reported that metastable precursors of rigid fibrils, so called oligomers and protofibrils, can undergo self-replication from natively folded monomers. This is the in vitro hallmark of prion diseases. However, it remains a mystery how such prion particles are able to catalyze their self-replication. We are trying to gain insight into the structure of protofibrils formed by hen egg white lysozyme by testing the resistance of these aggregates to enzymatic digestion and analyzing which portions of the aggregates remain preserved. Using this process we want to accomplish two goals: First, we want to identify which portion of the original monomers form the (readily digestible) outer segments and which portions forms the stable core of these prion aggregates. In a second step, we will test whether the enzymatically ablated aggregates still retain their capacity for self-replication – thereby providing important insights into the structure of the mysterious prion self-replicator.

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