1096-VG-2704 **Jason Karl Davis*** (jdavis8@ucmerced.edu), 5200 N Lake Rd, School of Natural Sciences, Merced, CA 95343, and **Suzanne S Sindi** (ssindi@ucmerced.edu), 5200 N. Lake Rd, School of Natural Sciences, Merced, CA 95343. *An Enzymatic Model of Prion Aggregate Dynamics.*

Prion proteins are responsible for a variety of diseases in mammals such as Creutzfeldt-Jakob disease in humans and mad-cow diease in cattle. According to the prion hypothesis, misfolded versions of a protein appear and form prion aggregates, complexes of multiple misfolded proteins ranging in size from tens to hundreds of proteins. The prion state is infectious and spreads to healthy proteins by conversion of the healthy confirmation to the misfolded state (which increases the size of the aggregate). Prion aggregates also increase in number by fragmentation, thus increasing the number of templates which act to convert healthy proteins.

The dynamics of prion aggregates have been investigated with a number of mathematical models. Most mathematical models assume that the fragmentation rate is proportional to the size of the aggregate; we present yeast data to demonstrate the inadequacy of this assumption, then extend the model to include the effects of an enzymatic limitation. Experiments have shown that changing a separate protein's expression levels has measurable effects on the aggregate size distribution, suggesting its role as a molecular chaperone in the fragmentation process. We perform general analyses of our more complete model, then compare it with experimental data. (Received September 18, 2013)