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Interaction of Infectious and Noninfectious Proteins in Prion Disease: Models, Simulations, and Steady State Study

A prion is an infectious form of protein that differs from a naturally produced protein only in its folding. Prions are thought to cause several diseases, with BSE (Bovine Spongiform Encephalopathy) perhaps the most widely known example. Diseases associated with prions have very long incubation periods, are difficult to detect in all but the latest stages, and are highly fatal. These characteristics alone make study of prions interesting, but even more so, there is the question of prion replication. Proteins do not possess any nucleic acid. Without DNA or RNA, how does the structure copy itself and spread?

There is evidence that prions form polymers or aggregates, most likely with additional stability. Some or all of these polymers attach to the similar naturally produced protein and convert it to the infectious variety. Polymers also split. Altogether, both the overall quantity of infectious proteins, and the number of polymer strands, increase. To model these phenomena, we represent prion polymer length as a continuous structure variable. We obtain a system of two partial differential equations modeling interaction of the infectious and noninfectious conformations of prion protein within an infected individual. We use this system to create numerical simulations of disease progress within such an individual. Under some circumstances, we can simplify to a system of three ordinary differential equations. In the ODE case, we discuss steady states, their stability, and relative parameter changes that affect their viability.