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Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by cognitive decline and memory impairment. The exact mechanisms underlying AD are not yet fully understood, but it is known that Amyloid beta oligomers, which are small aggregates of $A\beta$ protein, play crucial roles in the pathogenesis of this disease. Recent studies suggest that $A\beta$ oligomers tend to bind to healthy prions and misfold the latter into a pathogenic form called PrP^{ol} . In this work we present a model that tries to describe the interaction between $A\beta$ oligomers and prions. we investigate equilibria and study their local stability.

$$\begin{aligned}\dot{U}(t) &= S_u - d_u U(t) - 2\delta_1 P(t)U^2(t) + 2\delta_2 P(t - \tau)U^2(t - \tau) \\ \dot{P}(t) &= S_p - d_p P(t) - \delta_1 P(t)U^2(t) \\ \dot{P}^{ol}(t) &= -d_{pc}P_c(t) + \delta_2 P(t - \tau)U^2(t - \tau)\end{aligned}$$

Where U, P and P^{ol} denote the concentrations in $A\beta$ oligomers, prions and pathogenic prions respectively.

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