## ALZHEIMER'S DISEASE: ANALYSIS OF A MATHEMATICAL MODEL INCLUDING THE ROLE OF PRION

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ABSTRACT. In this presentation we introduce a model accouting for the in vivo dynamics of Alzheimer's disease including the role of the prion protein onto memory impairment in the disease. We use a size-structured equation to describe the formation of  $\beta$  amyloid plaques coupled with three differential equations on the concentration of A $\beta$ -oligomers,  $PrP^{C}$  proteins (prion) and A $\beta$ -×-PrP<sup>C</sup> complex since this latter has been considered, in some recent findings, to be responsible for the synaptic toxicity. We prove well-posedness of the problem and stability results of the unique equilibrium, when the polymerization rate of  $\beta$ -amyloid is constant and then it is described as power law.

Finally, we discuss about possible implications of this model in drug design and an experimental validation.

Keywords: Prion; Alzheimer; Size-structured equation; Well-posedness; Stability

## References

- [1] Gimbel D A *et al.* (2010) Memory impairment in transgenic Alzheimer mice requires cellular prion protein, *The Journal of Neuroscience* 30:6367-74.
- [2] Cisse M, Wein L M (2009) A prion protein connection, Nature 457:1090-91.
- [3] Craft D L, Wein L M, Selkoe D J (2002) A mathematical model of the impact of novel treatments on the Aβ burden in the Alzheimer's brain, CSF and plama, Bulletin of Mathematical Biology 64:1011-31.

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