
AN AGGREGATED MATHEMATICAL MODEL FOR THE PROGRESSION OF ALZHEIMER'S DISEASE WITH TREATMENT IMPLICATIONS

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In the talk a phenomenological mathematical model for Alzheimer's Disease is formulated and analyzed as a dynamical system. The model is based on the amyloid-beta ($A\beta$) cascade hypothesis which states that the buildup of misfolded $A\beta$ -plaques in the brain is the trigger for the cognitive decline in Alzheimer's Disease.

In the model, the formation of $A\beta$ -proteins is aggregated into three compartments consisting of monomers, proto-oligomers, and oligomers and the net-effect of stimulating and inhibiting feedback loops on the production of monomers through pro- and anti-inflammatory cytokines is considered.

The resulting 4-dimensional nonlinear dynamical system exhibits a uniform dynamical structure irrespective of the numerical values of the parameters. This structure consists of a region of attraction of the trivial equilibrium point (corresponding to no disease) and a region of divergence of the trajectories (corresponding to the development of the disease). These regions are separated by a 3-dimensional stable manifold of a positive equilibrium point acting as stability boundary. Existence and location of this stability boundary are determined by a simple mathematical quantity which expresses the difference between stimulating and inhibiting parameters in the feedback loops [1].

The results are illustrated with numerical simulations presenting various possible scenarios. Current treatment approaches will be discussed as some parameters of the model allow for a direct interpretation of these aspects.

Literatura

- [1] U. Ledzewicz, E. Ratajczyk and H. Schättler, An aggregated mathematical model for the progression of Alzheimer's Disease with treatment implications, *Nonlinear Dynamics*, 2026, **114**:596, <https://doi.org/10.1007/s11071-026-12474-z>

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