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Dynamics of Amyloid Beta Protein Signaling under Complexity Reduction: A Mathematical Modeling and BooleSim Simulation Approach

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Understanding how complex biological systems behave over time is essential for predicting their future states, especially in diseases like Alzheimer's disease. Mathematical modeling of such systems offers a valuable approach to exploring not only the theoretical aspects of network behavior but also their biological and medical implications. This study investigates how reducing the complexity of the Amyloid Beta (Aβ) signaling pathways influences its dynamic behavior, using a Boolean network model built from data curated in the SIGNOR database. Boolean rules were applied, and network simulations were performed in BooleSim under different initial conditions reflecting healthy and disease-related cellular states. Complexity reduction involved removing non-essential interactions and simplifying regulatory motifs. Our findings show that while simplification can shorten the time to reach steady states, it may also result in the loss of important regulatory pathways, potentially affecting the biological validity of the model. Nevertheless, key nodes such as BACE1 and GSK3β retained their functional significance even in the simplified models, confirming their central role in Alzheimer's pathology. Simulated interventions targeting these molecules disrupted apoptosis-related signaling patterns, highlighting their relevance as potential therapeutic targets, consistent with current Alzheimer's drug development strategies targeting BACE1 and GSK3β. This work illustrates how careful simplification of biological networks can offer a practical framework for modeling complex neurodegenerative processes at the intersection of biology and computation.

Keywords: *Mathematical modeling, Complexity, Alzheimer's disease, Boolean model, Signaling pathway*

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