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Alzheimer's; Still in Need of a Proper Theoretical Framework

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With more than 55 million registered patients suffering from dementia and its impact on society through extensive pressure on families and the health care system, it seems to be incumbent to seek out the reason(s) underlying our failure to produce any medical/pharmaceutical treatment [1]. The answer, roughly speaking, lies with the theories proposed by scientists that failed to identify a substantial factor at work. Nevertheless, recent achievements in this field beginning from a series of neuropsychological studies especially since a decade ago, discovered beta-Amyloid as a determining factor giving rise to AD and its degenerative essence. The β-Amyloid proteins as a type of amino acid peptides can bind together to form a plaque aggregation in the brain targeting and killing the neurons. Having been identified as the culprit of AD, scientists started suggesting therapeutically protocols aiming to breach the aggregation of the β-Amyloid. These studies even though resulted in the production of a few medications all somehow failed to show any significant effect [2].

Therefore, a group of scientists recently tried to set aside β-Amyloid theory and instead, started extending their quest toward a new and more fruitful theory for DA which seems promising. While one group relates the disease to the leakage of toxic proteins from the blood barriers containing fat-carrying particles [3], the other scientists are focusing on the pattern formation of the grey matter to explain brain inflammation using a mathematical method called Hopf- Turing reaction-diffusion. Comprehending the interplay among inflammatory mediating factors is essential in providing effective, patient-specific care. Activated microglia and elevated con- centrations of inflammatory signaling molecules reflect the complex cascades associated with acute neuroinflammation and are predictive of recovery Microglia are the resident macrophages of the central nervous system. They play key roles in brain development and physiology during life and aging. Due to the microglia reaction resulting in neuronal dysfunctions (e.g., Amyloid and Tau aggregates). it was primarily believed to be incidentally involved in AD [4]. However, recent studies have remarkably shown multiple functionalities of microglia in the initiation and development of AD.

The reaction-diffusion mechanism, in its revised version, is able to provide a satisfactory account for the formation of neural patterns (inflammatory or toxic) through disturbing molec- ular diffusion-driven instability maintained by microglia. Furthermore, Hopf-Turing reaction- diffusion system puts forth two mechanisms, namely, self-activation and lateral inhibition that are taken together responsible for stability maintenance. Also, other mathematical concepts such as patterning robustness can equip us with an effective tool to predict the stability of the cortical patterns with the language of probability. This under-development model could for the first time incorporate all essential biomarkers of AD and yield a complete timeline of the initiation-development per patient.

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