Review of: "Somatostatin and the pathophysiology of Alzheimer’s disease"

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The author of the paper "Somatostatin and the Pathophysiology of Alzheimer's Disease" provides a thorough and current examination of a 1980s-era theory regarding the role of somatostatin in Alzheimer's disease.

The review is highly intriguing since it explains how amyloid plaques and interneuron somatostatin co-localization cause cell death, which results in cognitive abnormalities similar to those seen in Alzheimer's disease. A theoretical framework for the origin, development, and neuropsychological characteristics of AD is managed in the manuscript. The author employed the neuropeptide somatostatin (SST), which is released by somatostatin-positive interneurons (SST-IN), to highlight the disease's importance. While the former is known to have a significant role in memory, learning, cognition, and even sleep oscillations in associative cortices afflicted by AD, the latter has been demonstrated to support A cleavage and clearance in the brain. The study examines the data that suggest that SST-IN hyperactivity in the early stages of AD, along with SST-IN hypofunction and increased network activity, are the primary causes of the changes in the cortex and hippocampus.

The author also describes how, in the early stages of the disease, SST-INs can result in presynaptic inhibition of GABA-B, which inhibits and internalizes the GABA-B1A-APP complex responsible for the production of amyloid peptides.

Plaques are formed, dystrophic neurites are overactive, glial cells are early cortical and hippocampus hyperactive, oscillation frequencies are altered, there is indiscrimination, and information is lost in spatial, episodic, and semantic memory, among other symptoms. The article discusses SST models of AD and suggests one based on SST-IN hyperactivity and hypofunction. It also discusses the functional and cognitive ramifications of these altered functions and how they align with the neuropsychological profile of AD.