



LECTURE

Beyond the horizon in dementia

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Abstract

Our nearly 35 year journey in Alzheimer disease (AD) began with the linkage of senile dementia to AD and the molecular dissection of genetics and proteins that revealed the central players. How could a cure or even effective therapy have eluded us for so long?

Studying AD is like walking through the looking glass, for while everything is the same—e.g., there is no novel gene expression—it is also different in quantitative and qualitative relationships leading to endless studies suggesting therapeutic strategies. Do endless abnormalities mask the one distinct initiator or is the countless change instead the disease: possibly a single condition but made up of thousands of components that are touched by the key risk factor of AD—age—for we find the same changes in aged normal

people. Could it be the same changes of aging are AD because they are what maintains normal function with aging but are insufficient in AD to maintain physiology. Removal of these changes has not restored normal function and could do harm.

In Alois Alzheimer's time treatments revolutionized medicine by curing most diseases from the outside, infections and injury, and corrected many of defect (mutations) but since then we have made but modest impact in diseases of normal aging, degenerative diseases.

Instead progress has been made when we address the underlying issues: lifestyle, inflammation, and others, instead of eliminating the reactive changes. We might consider the same for AD as we work on more effective therapy.

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