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Dementia: Alzheimer as a run-away Auto-immune disease

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Dementia is a broad category of brain diseases that cause a long-term and often gradual decrease in cognitive, emotional, functional and behavioral ability, resulting ultimately in death. The affected person's consciousness is not impaired. The most common type of dementia is Alzheimer disease (AD), which makes up 50% to 70% of cases. Other common types include vascular dementia (25%), Lewy body dementia (15%), and frontotemporal dementia. Less common causes include normal pressure hydrocephalus, Parkinson's disease dementia, syphilis, and Creutzfeldt–Jakob disease among others. More than one type of dementia may exist in the same person. A small proportion of cases run in families. I will concentrate on AD which, once considered a rare disorder over the past few decades, has emerged from obscurity to become a major public health problem. Based on a lack of treatment, it has generally been considered as an irreversible, progressive brain disease. It is a chronic neurodegenerative disorder of poorly (or not) understood cause(s). Based on identified risk factors, beyond genetics, several theories (15 or more), have been propounded for its cause(s). Such a wide array of hypotheses is by itself indicative of our lack of true understanding and knowledge of the disease notwithstanding the fact that the disease has been identified since 1901 and has been the subject of a considerable number of publications dealing with it (in excess of 50,000, according to some authors). Despite claims by some research clinicians, there are currently no known treatments if only to stop or reverse its progression. Some of these alleged "treatments", including the advocated program ("DESS": Diet, Exercise, Stress, Sleep, and variations on this theme) are palliative in nature, temporarily improving symptoms, while the disease progresses unabated. One must keep in mind that risk is not causation and risk management is not cure!

Research has rather focused on diagnosing the condition before symptoms begin. Thus, a number of biochemical tests have been developed to attempt earlier detection. Again, however helpful, such tests are not curative. I will posit that the compromised integrity of the blood brain barrier is a component of the etiology of the disease, not a consequence of it. I will further submit that the root cause of the disease is the brain's autoimmune system having gone rogue (a sort of "run away" effect) in its unsuccessful attempts to maintain brain homeostasis between the antagonistic synaptoblastic and synaptoclastic pressures. The cure would be to balance these pressures by regulating the system rather than fiercely combating either the hyper-excited synaptoblastic pressures or/and suppressing the synaptoclastic ones. I will review and discuss the above factors and also offer some potential curative approaches, including natural and synthetic (chimeric antigen receptor CAR T-cells) cell-based immunotherapies utilizing Treg-cells.

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