

Biochemistry and medicine information in pharmacology

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Introduction

Alzheimer's disease (AD) is a chronic neurodegenerative disease that is often clinically characterized by a progressive dementia and cognitive decline that begins slowly and eventually get worse overtime. At the onset, the most common symptoms include short term memory loss usually manifested in the inability of the affected individuals to remember recent events or the names of the people they previously new. As the disease progresses, new symptoms may begin to emerge some of which include language problems, mood swings, disorientation, loss of motivation and behavioral issues among others. AD brains are particularly marked by neurofibrillary tangles, amyloid plaques, neuronal cell loss, innate immune responses as well as a prominent activation of the glial cells. This paper critically investigates the potential relationship between Alzheimer's disease, Beta-C kinins and brain derived neurotrophic factor (BDNF) as well as their improvement on the cognitive function of patients.

Numerous recent empirical studies suggest that the production of a class of brain derived neurotrophic factor (BDNF) is significantly diminished among the patients suffering from Alzheimer's disease. Generally, BDNF is normally produced in the entorhinal cortex area of the brain (the part involved with cognition and memory) throughout an individual's life Honea et al., 2013). It is widely believed that the production of brain derived neurotrophic factor is particularly enhanced by beta-C kinins, a class of neuropharmacological molecules some of which include AB 123.

Although the correlation between the lowered levels of BDNF and

Alzheimer's disease (AD) has not been effectively established, studies indicate that neurotrophic factors normally play a critical protective role against amyloid beta toxicity (Mattson, 2008, p. 97). This is further supported by the growing number of scientific studies in Alzheimer's disease that have revealed significant alterations in the immune responses including observable changes in the macrophage and lymphocyte distribution and activation.

In a recent study conducted by Weinstein et al(2014), the researchers examined the levels of BDNF in 2131 healthy adults and then followed them for ten years. The results revealed that up to 50% individuals with the highest baseline levels of BDNF were less likely to develop dementia compared to their counterparts with the lowest levels of the molecule. It was concluded that a higher level of BDNF may protect against the occurrence of memory loss and possible help in the prevention of AD. In this regard, AB123 can be clinically indicated by the presence of amelioration of cognitive dysfunction normally associated with Alzheimers disease (AD).

References

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BDNF Prevents and Reverses Alzheimer's Disease