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Interaction between Human Brain and the Heart during Alzheimer's Disease. Do These Organs Have a Similar Information Storage Mechanism?

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Abstract

There are reasons to believe that the presence of magnetite enables the human brain to store information. If so, the presence of magnetite in the human heart might suggest a similar function. Alzheimer's patients often experience heart problems. The amount of plaques in the heart is also increased in Alzheimer's disease and its composition is similar to the brain plaques. As the brain's neurons destruct during the disease, magnetite and prion proteins are released and possibly incorporated into the amyloid plaques, at least in the brain.

Keywords: Brain; Heart; Alzheimer's disease; Magnetite; Prions; Information; Amyloid plaques

Introduction

So far, an effort to develop a diagnostic test and cure for Alzheimer's Disease (AD) has been a mirage. One barrier is a better understanding of the underlying mechanisms involved in AD's formation and the storage of information in the human brain. It has been suggested that memory can be transformed and stored in the brain's neurons (storage neurons) involving a magnetite chain and a prion protein, the tandem model [1,2]. There is presented a quantum mechanical equation for the process in which such a chain of magnetite crystals is magnetized [3].

The Brain, Heart and Alzheimer Connection

Heart and brain health are interrelated. AD and heart disease share similar epidemiological and genetic profiles and biochemical characteristics. As in the brain, the amyloid-β (Aβ) components Aβ40 and Aβ42 are present in the heart, and their presence is increased in AD [4]. The complete understanding of these mechanisms is poorly understood. If an information storage mechanism could function in the brain, it could also function in the heart. Heart samples show magnetite levels at about five to ten times higher than in brain tissue [5]. Could magnetite be present in the plaques from the heart during Alzheimer's disease?

The Collapse of the Neurons

There might be a link between AD, magnetite and prions [6]. In Alzheimer's brains, Aβ build up around synapses as well as the formation of insoluble twisted strings of tau protein, probably hampers the signal strength of electrical impulses, interfere with communication from one cell to another, ultimately leading to memory loss. Normally the protein tau forms a part of a structure of microtubule that help to transport nutrients and other important substances from one part of the nerve cell to another. In AD the tau protein is released from the microtubules forming insoluble twisted fibers inside the neurons. The microtubules are destroyed and the transport system collapse. Lack of nutrients and disturbances in the signaling systems disables the neurons, preventing new information from reaching the storage prion. When a memory neuron collapses, its membrane is disrupted, the magnetic chain breaks up, and the magnetite crystals and the prions will be released. This is the end of the electromagnetic forces in connection to the signaling system. High levels of magnetite are found in patients with AD, especially in plaque core material [7-10]. It could be a link between released magnetite from the damaged neurons, Aβ and memory [10]. Magnetite nanoparticles may play a role in the damaging action of a magnetite-Aβ complex [11,12]. An increase in magnetite formation due to altered synthesis of biometals, could occur in connection to the development of AD. This magnetite together with that released from the damaged neurons could react with the plaques.

Understanding the metal associated with AD pathology is crucial in the development of therapies intended to diagnose, monitor and treat the disorder [13]. When the neurons are disrupted, metabolites are released that could be detected in a blood sample. This could lead to a diagnostic test in the future.

The Presence of Magnetite and Prions in the Amyloid-β

The magnetite-prion combination is a perfect match for being involved in information storage. They are both stable compounds.

There might be a physical interaction between Aβ and normal cellular prion protein PrPc playing an important role in the Aβ toxicity at the synapse level by perforating membranes [14]. The magnetite nanoparticles, PrPc, and Aβ, in connection to AD, may have a more prominent role than previously thought. It can bring new insights in the understanding of the magnetite-prion plaque complex, possibly also in connection to the heart problems.

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