

Alzheimer's Disease: What is the Connection between Amyloid Plaques, Magnetite and Memory?

Fredrik C Størmer*

Norwegian Institute of Public Health, Oslo, Norway

Abstract

The time it takes from the first plaque is formed and to clinical signs of Alzheimer's disease is observed, is unknown. I have described herein the possible connection between the plaque development and memory loss. The signals from the sense organs to the possible magnetite-prion part of the information storage in the neurons will be hampered and finally the neurons will disintegrate. Each step will probably affect the short term memory.

Keywords: Alzheimer's disease; Amyloid plaques; Neurons; Prions; Magnetite; Memory

Introduction

One of the most common signs of Alzheimer's disease (AD) is memory loss especially forgetting recently learned information. Before such loss of memory, a lot of processes have taken place in the brain. I shall focus on the processes that are occurring with the neurons involved in the storage of information during the development of the disease and its possible connection to the amyloid plaques.

The Steps Leading to Short Term Memory Loss

Amyloid- β (Ab) comes from a larger protein (amyloid- β precursor protein) found in the fatty membrane surrounding the nerve cells. In a healthy brain, these proteins are broken down to fragments, released and eliminated. In AD, the fragments (Ab) are formed and low amounts can act as a modulator of synaptic activity, with implications for memory and learning. It can accumulate in the extracellular space of the brain where it can form insoluble plaques.

Neurons are major sources of Ab in the brain and Ab is also present in the astrocytes. Astrocytes outnumber Ab by at least five-fold. Thus even a small level of astrocytic Ab production could make a significant contribution to Ab burden in AD. Loss of astroglial function and reactivity contributes to the ageing of the brain and to neurodegenerative diseases like AD [1]. Astrocytes are interacting with neurons, transport and other astrocytes via signaling and other processes. The formation of plaques could therefore gradually weaken the communication between the astrocytes and the neurons leading to destruction of the latter.

Insoluble twisted fibers are found inside the neurons. These tangles consist primarily of the protein tau, which forms part of the microtubule system. In AD, however, the tau protein is abnormal and disrupts the ability of neurons to strengthen connections with other brain cells, preventing memories from forming and the microtubule structures collapse [2].

Magnetite and its Connection to Alzheimer's Disease

Magnetite is an iron oxide (Fe_3O_4) which is widely distributed among organisms without being involved in any known biochemical reactions. Magnetite is present in the brain [3] and biogenic magnetite is associated with neurodegenerative diseases like AD and Parkinson. It has been shown that the amount of magnetite present is generally high in AD brain [4-6]. In plaque core material magnetite has been detected as the dominant iron compound [6,7].

The Storage of Information in the Neurons

Prion like proteins are critical for maintaining long term memory in mice [8] and the role of prions connected to memory is described [9]. They appear to be involved in the storage of information in the neurons of all animal species. The connection between Alzheimer's disease, magnetite and prions has been described [10].

It has been proposed that information is stored in prion-like proteins which can change shape when exposed to an electric impulse and that magnetite is involved. It could be assumed that magnetite and proteins are involved in a tandem mechanism in which incident impulses are received and reshaped by the nanocrystalline magnetite to a form that can be accepted by the proteins in which the information is stored [11]. Such chains of nanomagnetite crystals can easily be magnetized by an electric impulse but cannot hold the magnetism which drops to zero after each impulse. Therefore, magnetite is not likely to be the substance in which information is permanently stored. It is not surprising that these two stable compounds in the brain are involved in maintaining memory.

The Fate of the Neurons Involved in Memory Storage

It is a complicated route that the electric impulses have to travel from the sense organs to the final destination, the neurons where they are stored. When electric impulses are hampered by amyloid plaques they will be weakened in strength during their way. The neurons will not work properly due to several factors like lack of nutrients and disturbances in the signaling systems. Therefore new information will not be stored properly (short term memory). When a neuron collapses, the membrane is disrupted and this is the end of the electromagnetic forces in connection to the signaling system. Consequently the magnetite chain cannot be magnetized and the memory storage mechanism will not work. The membrane protecting the magnetite chain will also be disrupted and the magnetite crystals will be released.

*Corresponding author: Fredrik C Størmer, Norwegian Institute of Public Health, Oslo, Norway, Tel: +47 92268576; E-mail: fredrik.c.stormer@gmail.com

Received May 28, 2017; Accepted August 26, 2017; Published September 02, 2017

Citation: Størmer FC (2017) Alzheimer's Disease: What is the Connection between Amyloid Plaques, Magnetite and Memory? J Alzheimers Dis Parkinsonism 7: 366. doi: 10.4172/2161-0460.1000366

Copyright: © 2017 Størmer FC. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

It could be a connection between released magnetite from the damaged neurons and Ab that could lead to severe network deterioration. It has previously been suggested that magnetite nanoparticles have a more prominent role than previously suggested and may bring new insights in the understanding of the damaging action of a magnetite-Ab complex [7].

References

1. Hertz L, Peng L, Dienel (2007) Energy metabolism in astrocytes: High rate of oxidative metabolism and spatiotemporal dependence on glycolysis/glycogenolysis. *J Cereb Blood Flow Metab* 27: 219-249.
2. Tracy TE, Sohn PD, Minami SS, Wang C, Won Min S, et al. (2016) Acetylated tau obstructs KIBRA-mediated signaling in synaptic plasticity and promotes tauopathy-related memory loss. *Neuron* 90: 245-260.
3. Kirschvink JL, Kobayashi-Kirschvink A, Woodford BJ (1992) Magnetic biomineralization in the human brain. *Proc Natl Acad Sci USA* 89: 7683-7687.
4. Hautot D, Pankhurst QA, Khan N, Dobson J (2003) Preliminary evaluation of nanoscale biogenic magnetite in Alzheimer's disease brain tissue. *Proc Biol Sci* 270: S 62-64.
5. Pankhurst QA, Hautot D, Khan N, Dobson J (2008) Increased level of magnetic iron compounds in Alzheimer's disease. *J Alzheimers Dis* 13: 49-52.
6. Collingwood JF, Chong RKK, Kasama T, Cervera-Contard L, Rafael L, et al. (2008) Three-dimensional tomographic imaging and characterization of iron compounds within Alzheimer's plaque core material. *J Alzheimers Dis* 14: 235-245.
7. Teller S, Tahirbegi IS, Mir M, Samitier J, Soriano J, et al. (2015) Magnetite-amyloid- β deteriorates activity and functional organization in an *in vitro* model for Alzheimer's disease. *Scientific Reports* 5: 17261.
8. Fioriti L, Myers C, Huang Y-Y, Li X (2015) Long term memories are maintained by prion-like proteins. Columbia University Medical Center.
9. Si K, Kandel ER (2016) The role of functional prion-like proteins in the persistence of memory. *Cold Spring Harb Perspect Biol* 8: a021774.
10. Størmer FC, Bakkeiteig LS (2015) Is there a connection between Alzheimer's disease, magnetite and prions? *Austin J Clin Neurol* 2: 1044-1046.
11. Størmer FC, Alfsen EM (2015) Is a combination of magnetite and prions involved in memory storage in the human brain? *Med Hypotheses* 85: 111.

Citation: Størmer FC (2017) Alzheimer's Disease: What is the Connection between Amyloid Plaques, Magnetite and Memory? J Alzheimers Dis Parkinsonism 7: 366. doi: [10.4172/2161-0460.1000366](https://doi.org/10.4172/2161-0460.1000366)

OMICS International: Open Access Publication Benefits & Features

Unique features:

- Increased global visibility of articles through worldwide distribution and indexing
- Showcasing recent research output in a timely and updated manner
- Special issues on the current trends of scientific research

Special features:

- 700+ Open Access Journals
- 50,000+ editorial team
- Rapid review process
- Quality and quick editorial, review and publication processing
- Indexing at major indexing services
- Sharing Option: Social Networking Enabled
- Authors, Reviewers and Editors rewarded with online Scientific Credits
- Better discount for your subsequent articles

Submit your manuscript at: <http://www.omicsonline.org/submission/>