The Neurological Effects on the Four Lobes of the Brain as a Result of the Amyloid Beta Protein

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Abstract: Amyloid-beta is a protein found in the neocortex of the brain, associated with Alzheimer's disease dementia. Papers about dementia typically include overviews but rarely dive deeper. The goal of this paper is to critically review articles about the effects of accumulated amyloid beta via different lobes of the brain. We conducted a critical review focused on Alzheimer's disease, Parkinson's dementia, and amyloid-beta proteins. We identified five relevant sources related to declining cognition, and emotional wellbeing and functioning. Only one was an original empirical study. Further work is needed to better understand the role of amyloid-beta in the hippocampus. Future studies should integrate neuroimaging and cognitive testing to investigate how amyloid beta accumulation differs by brain region.

Keywords: amyloid-beta, Alzheimer's dementia, Parkinson's dementia, emotional wellbeing

Amyloid-beta is a protein found in the neocortex of the brain that is associated with Alzheimer's Disease when it builds up and kills brain cells during the process. Along with being the cause of Alzheimer's disease, accumulation of amyloid-beta contributes to cerebral amyloid angiopathy, neurodegenerative diseases, and cardiovascular issues (Pugazhenti, 2017). How is amyloid present in different diseases? To clarify, amyloid beta is found in the temporal lobe, more specifically, AB first accumulates in the entorhinal cortex, then extends to affect the entire hippocampus. Accumulation of AB in this region of the temporal lobe causes patients to clinically present episodic memory loss and loss of semantic knowledge. To date, the literature regarding AB has primarily focused on Alzheimer's dementia but has overlooked the effect amyloid-beta has on other lobes of the brain. To fill this gap, the purpose of this paper is to assess how would amyloid beta protein affect the rest of the body (e.g., senses, movement, etc.) as it accumulates in different lobes of the brain? The point of this project is to review past articles that are based around how the accumulation of the amyloid beta protein and how it affects the rest of the body via the different lobes of the brain.

Methods

To accomplish the goal described above, we conducted a critical review. The goal of a critical review is to comprehensively evaluate another's work, including weighing strengths and weaknesses and integrating their findings into the larger literature scope. One's stance should be based on knowledge and expertise, accumulated through lectures, readings, and personal experiences. A critical review should not simply summarize everything the author writes but should present key points. My critical review process is to summarize the key points of the author's article. I selected the articles based on how much evidence they gave to support effects that the protein had on the rest of the human body. For this critical review, we searched PubMed. Our search terms included: "amyloid beta" AND "dementia" AND "brain" AND "location" AND "lobes". We included articles that measured levels of amyloid beta in the brain, referenced any type and severity of dementia, were available in English, and described the location of amyloid in the brain. We filtered/screened studies by title/abstract and subsequently full text for relevance.

Results

Our search yielded 5 relevant articles. Of the 5 articles included in this critical review, data were derived from only 1 source. The study conducted to examine interference on the temporal lobe in dementia-diagnosed individuals (N=20) yielded that both groups of elderly individuals (those with dementia and those without) scored the same on trials that included high interference. However, healthy older adults significantly outperformed PD-diagnosed adults on trials that included low interference ($p<0.05$). Although the differences between healthy individuals and PD-diagnosed individuals were noticeable, both groups improved on low interference trials compared to high interference trials ($p<0.001$).

The first piece of evidence is from an article written by Younes et. al. in 2022. The key pieces of evidence that played a key role in writing this paper were percentages that explain how much cognitive thinking ability you lose from temporal lobe atrophy: Rigid thought process (18%) and a loss of empathy (27%); affecting both the temporal lobe and the frontal lobe. This piece of evidence aims to help the reader understand how much damage Alzheimer's dementia does to the temporal lobe using percentages as estimates. The authors' paper is clear and factual, which helps people who want to learn about the effect of Alzheimer's in the temporal lobe. The authors of this paper correctly sourced and cited all references in support of their claims. Another piece of evidence that was collected from this article were symptoms that affect the temporal lobe impairments in recognizing famous people, emotional theory of mind, and facial affect naming, directly affecting both anterior temporal lobes (ATL). The purpose of this piece of evidence was to show how much cognitive ability we lose when experiencing dementia, all of the articles show that we cognitive function in both the frontal and the temporal lobe.

This piece of evidence was written by Butler et. al. in 2019. The article gave a brief overview about Frontotemporal Dementia (FTD) and spoke about how FTD affects the human body as a whole. For example, FTD causes difficulties in social, emotional, and executive function, as well as causing changes in brain connectivity, which in turn causes neuronal loss and the spreading of proteinopathies. Key points include difficulties in speaking, expressing, and performing daily life functions. These daily life functions also include decision-making, flexible thinking, and self-control, which falls under the frontal lobe (more specifically the dorsolateral prefrontal cortex). To summarize, the author was successful in explaining the effect of FTD on the brain by describing the difficulties such as loss of executive function. This article was most likely directed towards someone who has foundational knowledge of FTD. The author did cite the sources that they were revealing in the correct APA Format.

The evidence listed here was written by Deford et. al. 2016. Unlike the others, this article includes a lab experiment done on elderly participants who were either healthy or have been diagnosed with Parkinson's dementia. The results were made with a 2 x 2 table that revealed evidence of a significant main condition ($F(1, 38) = 10.07$, $p < 0.01$, $\eta 2 = 0.21$) that caused an interference condition ($F(1, 38) = 73.01$, $p < 0.001$, $\eta 2 = 0.62$). The key points from this article showed how Parkinson's dementia affects multitasking, by negatively affecting the first task while trying to complete the second. The author was successful in explaining the results of the experiment, as well as citing the sources used in the introduction. The experiment included the medical background of the patients, and the step-by-step procedure of how the experiment was conducted.

The final piece of evidence was written by Borghesani et al. in 2022. This article gives an overview of FTD, and what parts of the brain it affects. The article explains that right-predominant anterior temporal lobe-predominant atrophy causes impairments in recognizing famous people, emotional theory of mind, and facial affect naming, directly affecting both anterior temporal lobes (ATL). The key point is that the cognitive ability to recognize famous people, emotional theory of mind, and facial affect naming had declined when the patient started developing FTD. The author does a great job in explaining FTD and its overlooked concepts to a mature audience and has made it clear enough for a proper critical review to be conducted. The citations have all been done correctly, and it helps readers deepen their understanding of FTD.

Discussion

The purpose of this paper was to review certain articles that wrote about dementia (any form) and how it affects both the brain and the peripheral nervous system. This is consistent with all 5 papers. We chose a critical review because of the strengths in improving decision-making, increasing self-awareness, and lessening the impact of cognitive biases. The biggest limitation of this review is the lack of existing literature about this topic, which resulted in less than 10 sources being used. From this point on, it is evident that doctors/neurological professionals should pay more attention to the effects of amyloid-beta on other parts of the brain like the parietal lobe and the cerebellum, not just the frontal and temporal lobes. To accomplish this goal, more imaging studies need to be conducted on the parietal lobe and the gray and white matter. This way future doctors can hopefully limit the effects on the human body, by understanding what other functions of the human body are affected. Not only should more imaging be conducted, an increase in studies on dementias such as Lewy body can help to develop a cure that can prevent the main symptoms from occurring.

In conclusion, amyloid-beta does indeed influence other lobes of the brain because studies show that amyloid-beta plaques cause cognitive impairment in the empathetic part of the brain, which is the main function of the temporal lobe. In the first article, right anterior temporal lobe atrophy causes a severe cognitive impairment in the empathetic region of the brain by a margin of 27%, Younes et al. (2022). Similarly, left and right anterior temporal lobe atrophy also causes difficulty in recognizing famous people, emotional theory of mind, and facial affect naming, directly affecting. The paper written by Defrod et al. in 2016 gives the process of an experiment that was done by the doctors that showed how both the temporal lobe and the frontal lobe have been affected because of the increase in interference. This interference makes multitasking a very difficult problem. To gradually correct this issue, more imaging studies should be conducted to help develop a cure that targets the main symptoms of the human brain and the peripheral nervous system.

References

Butler, P. M., & Chiong, W. (2019). Neurodegenerative disorders of the human frontal lobes. Handbook of clinical neurology, 163, 391–410. https://doi.org/10.1016/B978-0-12-804281-6.00021-5

Borghesani, V., DeLeon, J., & Gorno-Tempini, M. L. (2022). Frontotemporal dementia: A unique window on the functional role of the temporal lobes. Handbook of clinical neurology, 187, 429–448. https://doi.org/10.1016/B978-0-12-823493-8.00011-0

DeFord, N. E., Landy, K. M., Pirogovsky-Turk, E., Van Etten, E. J., Graves, L. V., Salmon, D. P., Filoteo, J. V., & Gilbert, P. E. (2016). The effect of interference on temporal order memory in individuals with Parkinson's disease. Brain and cognition, 107, 30–36. https://doi.org/10.1016/j.bandc.2016.05.008

Pugazhenthi, S., Qin, L., & Reddy, P. H. (2017). Common neurodegenerative pathways in obesity, diabetes, and Alzheimer's disease. Biochimica et biophysica acta. Molecular basis of disease, 1863(5), 1037–1045. https://doi.org/10.1016/j.bbadis.2016.04.017

Younes, K., Borghesani, V., Montembeault, M., Spina, S., Mandelli, M. L., Welch, A. E., Weis, E., Callahan, P., Elahi, F. M., Hua, A. Y., Perry, D. C., Karydas, A., Geschwind, D., Huang, E., Grinberg, L. T., Kramer, J. H., Boxer, A. L., Rabinovici, G. D., Rosen, H. J., Seeley, W. W., … Gorno-Tempini, M. L. (2022). Right temporal degeneration and socioemotional semantics: semantic behavioural variant frontotemporal dementia. Brain: a journal of neurology, 145(11), 4080–4096. https://doi.org/10.1093/brain/awac217