



NERVE CELL DETERIORATION ASSOCIATED WITH ALZHEIMER'S DISEASE



Formulate the Trend of Nerve Cell Decrease in Alzheimer's Patients.

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Abstract

Alzheimer's disease is an extremely serious condition that is challenging to diagnose. We have used experimental data to compare the rate of decay of entorhinal cortex (EC) neurons in various stages of Alzheimer's. We observed that the rate of EC neuron decay in the patients without Alzheimer's is close to zero, linear in mild cases, and quadratic in severe cases. We believe that described estimates may help to diagnose the disease as well as its stage.

Problem Statement

- ❖ 30% of people over the age of 85 suffer from Alzheimer's disease, making it the most common neurodegenerative disease (Hamann, 402).
- ❖ Laboratory test has yet to be established to diagnose the disease, so an autopsy remains the only reliable way to confirm the Alzheimer's (Hamann, 402).

To explore more accurate ways of diagnosing Alzheimer's disease, we compare the quantity of entorhinal cortex (EC) neurons in the of Alzheimer patients to the quantity of EC neurons in subjects that don't have Alzheimer's in hopes to find a pattern in the rate of EC neuron decrease that could serve as a marker characteristic for the disease.

EC is located at the medial temporal lobe, carries on important functions supporting the hippocampal formation and the neocortex in regards to memory formation, learning, and higher order cortical processing ("Entorhinal Cortex.").The EC is the first known area to be affected by Alzheimer's disease.

While neurofibrillary tangles and senile plaques are important signs of Alzheimer's disease onset, the occurrence of the two symptoms are also observed in healthy brains (Gomez-Isla, 4491).

The number of neurons in the entorhinal cortex is known to show a significant decrease in Alzheimer's patients, but not in a normal human's lifespan (Sutherland, 438).

Mathematical Approach

Figure 1: Neuronal counts in entire EC for control subjects

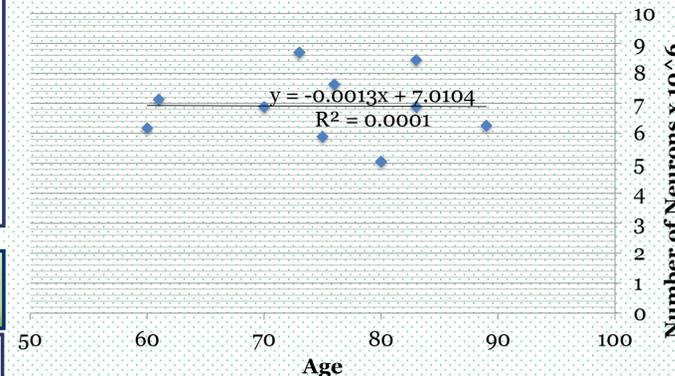


Figure 2: Neuronal counts in the entire EC for mild AD patients

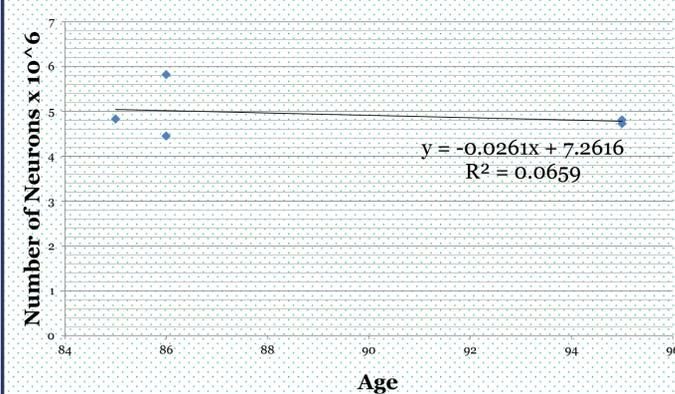
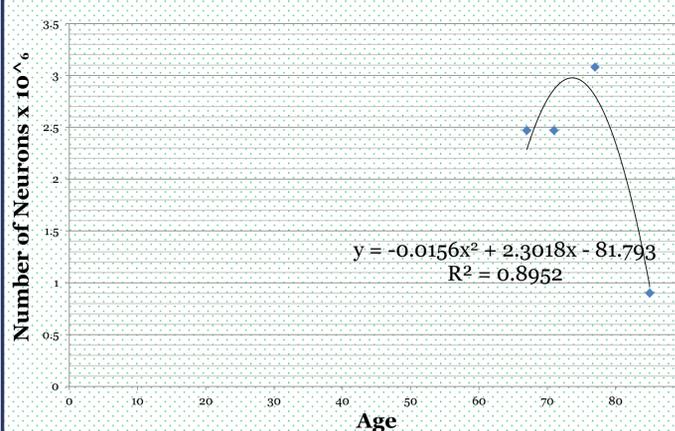


Figure 3: Neuronal Counts in the Entire EC for severe AD Subjects



In this work we use data reporting the decrease in number of entorhinal cortex (EC) neurons of Alzheimer's patients and normal subjects (Gomez-Isla, 4492). The data describes quantity of EC neurons for subjects in three categories according to their CDR: the control group, patients suffering a mild form of Alzheimer's disease, and patients suffering from severe Alzheimer's disease (see Figure 4). We have calculated the following lines of best fit for each of the groups.

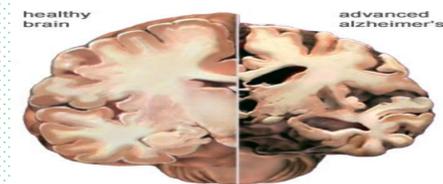
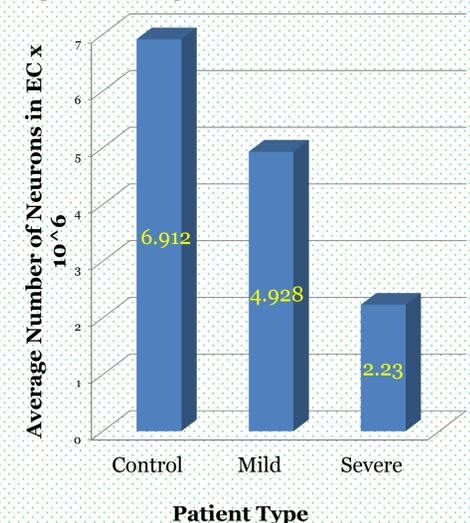


Figure 4: Average Number of Neurons (x10^6)



Discussion

The obtained equations suggest that the rate of EC neuron decay in the patients without Alzheimer's is close to zero, linear in mild cases, and quadratic in severe cases. However the used dataset was relatively small and so even a few outliers could have significantly affected our predictions. Hence we believe that a larger dataset could have significantly improved the reliability of our findings.

Conclusions

Our observations suggest that the patients with Alzheimer's gradually suffer increasingly faster entorhinal cortex (EC) neuron deterioration as the disease progresses while there is an insignificant loss of EC neurons in the entorhinal cortex during normal aging. For our estimates, we have used data generated by neurology service in the Massachusetts general hospital in Boston (Gomez-Isla 4492). However, the data is obtained from postmortem brain samples collected from individuals with and without Alzheimer's disease. Hence a method for estimating the quantity of EC neurons in live patients is a crucial step towards a better technique for diagnosing Alzheimer's. Perhaps integrating optical fractionator and EKG could help solve this problem.

References

"Entorhinal Cortex." 11 April 2012. [Scholarpedia](http://www.scholarpedia.org/article/Entorhinal_cortex). <http://www.scholarpedia.org/article/Entorhinal_cortex>.

Gomez-Isla, Teresa, et al. "Profound Loss of Layer II Entorhinal Cortex Neurons Occurs in Very Mild Alzheimer's Disease." *The Journal of Neuroscience* (1996): 4491-4500.

Hamann, Barbara P. "Disease: Identification, Prevention, and Control." *Boston: McGraw Hill* (2007): 402-403.

"Prevention and Treatment of Alzheimer's Disease." *The Light Party New Political Paradigm and Health Care* (2010): 11.

Sear, Who. "Alzheimer's Disease: Mild Memory Loss: An Inevitable Consequence of Ageing." *Regional Health Situation, and World AIDS Day, Health Report*. (2010).

Sutherland, Greg T., Michal Janitz and Jillian J. Kril. "Understanding The Pathogenesis Of Alzheimer's Disease: Will RNA-Seq Realize The Promise Of Transcriptomics?" *Journal Of Neurochemistry* 116.6 (2011): 937-946.