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Is too much brain activity connected to Alzheimer's disease?

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High baseline levels of neuronal activity in the best connected parts of the brain may play an important role in the development of Alzheimer's disease. This is the main conclusion of a new study appearing in *PLoS Computational Biology* from a group at VU University Medical Center in Amsterdam, the Netherlands.

In recent times, it has become clear that brain activity patterns change at an early stage in Alzheimer's disease. Moreover, there is reason to believe that, instead of being the consequence of structural damage, they might be the cause: recently, a direct influence of excessive regional neuronal activity on Alzheimer pathology was found in animal experiments. By showing that highly connected 'hub' regions (which display most Alzheimer pathology) indeed possess the highest levels of activity, the present study offers support for the unconventional view that brain dynamics may play a causal role in Alzheimer. As first author, Willem de Haan, says, "this implies that the investigation of factors regulating neuronal activity may open up novel ways to detect, elucidate and counter the disease".

Using a realistic computational model of the human cortex, the authors simulated progressive synaptic damage to brain regions based on their level of activity, and subsequently investigated the effect on the remaining network. With this 'activity dependent degeneration' model, they could not only offer an explanation for the distribution pattern of Alzheimer pathology but also reproduce a range of phenomena encountered in actual neurophysiological data of Alzheimer patients: loss

and slowing of neuronal activity, loss of communication between areas, and specific changes in brain network organization.

In upcoming projects the authors plan to verify the predictions from this study in patient data, but also to continue modeling studies. They conclude that: "the use of 'computational neurology' and network theory to unite experimental results and find plausible underlying principles in the growing bulk of human brain data seems inevitable".

More information: de Haan W, Mott K, van Straaten ECW, Scheltens P, Stam CJ (2012) Activity Dependent Degeneration Explains Hub Vulnerability in Alzheimer's Disease. PLoS Comput Biol 8(8): e1002582. doi:10.1371/journal.pcbi.1002582