

Increased dementia risk in women—a matter of proteins?

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Gender-specific differences between the levels and structures of proteins present in the white matter and the mitochondria of the brains of men and women suffering from dementia have been revealed for the first time in a study published in the open access journal *Molecular Brain*.

While previous studies have shown that women exhibit higher risk of dementia than men, the underlying mechanisms of this gender difference have remained elusive. The findings by researchers from Nanyang Technological University, Singapore may advance our understanding of the higher risk of dementia that has been observed in women compared to men, which the researchers suggest could lead to the development of new drugs for dementia treatment.

Associate Professor Sze Siu Kwan one of the co-authors of the study said: "The number of [dementia patients](#) is projected to triple by 2050 and there is an urgent need to identify key mechanisms of how dementia develops. Our findings and further study could have direct implications for our knowledge about the progression of dementia that could lead to the development of drugs for treatment of dementia."

The researchers used proteomics - the large-scale study of proteins, including their variations and changes - to analyze the proteins present in post-mortem brain tissues from five male and five female dementia patients and ten healthy controls. The researchers wanted to identify changes in structure and function of the proteins present in the white matter and the mitochondria of the temporal lobe - the part of the brain

involved in visual memory and the understanding of language.

Proteomics is an unbiased approach as, rather than testing a set of pre-defined, specific proteins, it allowed the researchers to look at thousands of proteins to identify changes associated with dementia.

Degenerative Protein Modifications (DPMs) are changes to proteins that are thought to cause the loss of [protein](#) function, similar to the way that steel loses strength when rusting. The protein shown to be most affected by these DPMs in the brains of dementia patients in this study is Myelin Basic Protein (MBP) which is important for the production of a protective myelin layer around the axons of nerve cells. Axons are part of the brain's [white matter](#) which transmits signals between different parts of the brain. A process called myelination provides an electrically insulating layer around axons, similar to the insulation around an electrical wire. Damage to that insulating layer can stop electrical impulses from being conducted properly, disrupting communication between different parts of the brain. DPMs that are thought to increase the dysfunction of MBP were found to be more pronounced in women than in men. Sze Siu Kwan said: "As DPMs are likely to critically influence protein function and activity in the central nervous system they can be novel drug targets for treatment of dementia."

The researchers detected changes in the presence of certain proteins in the mitochondria of patients suffering from dementia that indicate [mitochondrial dysfunction](#). Mitochondria are responsible for creating energy needed to sustain proper cell function, including brain cell function. Mitochondrial dysfunction can lead to cell injury or even cell death. As with changes to other proteins, disturbance to the proteins in the mitochondria was observed to be more pronounced in women than in men.

Xavier Gallart-Palau, the first author of the paper said: "The findings of this study indicate that proteomics can detect differences between male

and female dementia patients on a molecular level which cannot be detected by standard approaches."

The findings also provide new insight into the molecular basis of increased risk and severity in women suffering from dementia. They may be a step towards future clinical interventions targeted at reducing [dementia](#) risk in both men and women.

More information: Xavier Gallart-Palau et al. Gender differences in white matter pathology and mitochondrial dysfunction in Alzheimer's disease with cerebrovascular disease, *Molecular Brain* (2016). [DOI: 10.1186/s13041-016-0205-7](#)

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