



The University of Sydney



## Media release

### Discovery identifies mitochondria as major target in Alzheimer's disease

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In a collaborative effort, researchers at the Universities of Basel, Frankfurt and Sydney have made a major breakthrough that will help in the development of antioxidative treatments for Alzheimer's disease. This work is published in this week's early edition of the Proceedings of the National Academy of Sciences\*.

Brains of Alzheimer's disease (AD) patients are characterized by clumps of proteins that come in two flavors, as amyloid plaques and neurofibrillary tangles.

Researchers led by Professor Jürgen Götz from the University's Brain and Mind Research Institute (BMRI) firstly discovered that at an early stage when these deposits start to form, proteins that make up the mitochondria (the power horses of all cells) are altered in their expression. Professor Anne Eckert from the University of Basel next determined that not only are the proteins altered, but that their function is severely impaired. Specifically, the oxidative phosphorylation system that is essential for life goes awry.

“This publication is a convincing testimony that research gains from joining forces and that many breakthroughs are multi-disciplinary in nature”, says Jürgen Götz. Dr Ittner, co-author of the study and also based at the BMRI, states that “this study is another proof that plaques and tangles together damage the nerve cells.” The breakthrough study shows that plaques and tangles together do more harm to mitochondria than they would do alone.

By identifying a key role for mitochondria in the development of the disease it is hoped that anti-oxidative therapies may prevent or delay the onset of Alzheimer's disease.

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\* Virginie Rhein et al., (2009) Amyloid-beta and tau synergistically impair the oxidative phosphorylation system in triple transgenic Alzheimer's disease mice. *Proc Natl Acad Sci U S A*. 2009 Nov 2.