New hope for treating Alzheimer's Disease: A role for the FKBP52 protein

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New research in humans published today reveals that the so-called FKBP52 protein may prevent the Tau protein from turning pathogenic. This may prove significant for the development of new Alzheimer's drugs and for detecting the disease before the onset of clinical symptoms.

A study published online today in the *Journal of Alzheimer's Disease*, for the first time demonstrates that the FKBP52 protein, discovered by Prof. Etienne BAULIEU twenty years ago, may prevent hyperphosphorylation of Tau protein, which has been shown to characterise a number of cerebral neurodegenerative diseases, including Alzheimer's Disease (AD).

This work has been carried out by Professor Etienne Baulieu and his research team at Inserm (National Institute for medical research in France) with the support of philanthropists who help the Institut Baulieu, based in France.

Limited research exists on Tau and its role in the development of AD, but it is known that many neurodegenerative diseases are characterised by the deposition of pathological hyperphosphorylated forms of Tau protein, into structures known as 'Tau tangles'. The mechanism of Tau toxicity is unclear and there are currently no drug treatments targeting Tau, nor any biomarkers that predict the risk of a future "Tauopathy". Professor Baulieu decided to focus on Tau abnormalities and was the first to discover in 2010, an interaction between Tau, and the FKBP52

protein.

The new research takes his previous research to the next level. It demonstrates a direct correlation between high levels of hyperphosphorylated Tau protein and reduced levels of FKBP52, in brain cells from patients who have died following Alzheimer's Disease, compared with normal brain cells. This suggests that FKBP52 could control the aberrant production of pathogenic Tau. When FKBP52 is reduced in the nerve cells of AD patients, pathogenic Tau is free to accumulate and contribute to the degeneration of brain cells.

In conclusion, early measurement of FKBP52 levels could form the basis of a predictive test for Alzheimer's Disease before the onset of clinical symptoms, and new compounds modulating FKBP52's activity could become the next generation of treatments for the disease.

Commenting on this new research, Professor Baulieu said: "There is still a worrying lack of research into the causes of age-related brain disorders such as Alzheimer's Disease and dementia. I founded the Institut Baulieu, with the aim of being able to treat and even prevent these diseases.

Research on Tau has been very limited, and until recently, I was among the few scientists focusing on Tau pathology. The discovery of the FKBP52 protein is the only 'anti-Tau' perspective so far. Its reduced production in the brains of Alzheimer's patients marks a turning point in understanding this complex disease.

I believe it takes us one step closer to developing an effective treatment and possible predictive tests for the increasing number of people who may develop Alzheimer's Disease in our ageing societies."

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