

Prana drug reduces cognitive impairment

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Singapore: Prana Biotechnology announced the upcoming presentation of new data demonstrating the ability of PBT2 to reduce the damage to brain cells, caused by the accumulation of the tau protein and preventing subsequent cognitive impairment. The tau protein, along with the Abeta protein, are the two major proteins associated with Alzheimer's disease. PBT2 is currently in a phase II clinical trial, the Imagine trial, which is now fully enrolled and will be completed at the end of the year.

The findings are consistent with the improvement in cognition previously reported in transgenic Alzheimer's mice studies and in patients in a phase IIa clinical trial with PBT2 and further validate the metal targeting mechanism of action of PBT2. New data will be presented by Prana scientist and associate professor Paul Adlard at the 11th International Conference on Alzheimer's and Parkinson's disease, to be held in Florence, Italy, March 6-10, 2013.

Abeta has been the primary therapeutic target for disease modifying drugs developed for Alzheimer's disease. However, the clinical failure of several anti-Abeta drugs supports the view that targeting Abeta alone may be insufficient to improve outcomes for patients. The other hallmark pathological feature of Alzheimer's disease is the presence of neurofibrillary tangles, composed of abnormal tau protein.

In his presentation, entitled 'Metal chaperones are novel therapeutic agents for tauopathy', associate professor Adlard will present new data showing that treatment with PBT2 significantly improves cognition and reduces the abundance of tau aggregates through metal mediated mechanisms in a transgenic mouse model of tau overexpression.

Commenting on the significance of the new data, Ms Rudy Tanzi, the Rose and Joseph Kennedy Professor of Neuroscience at Harvard Medical School and Prana's chief scientific advisor, said, "These findings provide further evidence for PBT2 as a highly attractive therapeutic for Alzheimer's disease that targets both beta amyloid deposition and tangle formation. Translating these dual effects into the clinic could potentially provide tremendous benefit for patients."