IOS Press

JOURNAL OF ALZHEIMER'S DISEASE

PRESS RELEASE
FOR IMMEDIATE RELEASE

Contact:
Astrid Engelen
IOS Press
Tel: +31 20 688 3355
Fax: +31 20 620 3419
E-mail: a.engelen@iospress.nl

URL: www.j-alz.com

Investigators unveil new drug discovery tool for Alzheimer's disease

April 28, 2008 -- An article published in the April issue of the *Journal of Alzheimer's Disease* presents a detailed characterization of a new drug discovery tool for Alzheimer's disease. It demonstrates that an abnormal form of tau protein, as it occurs in Alzheimer's disease, can be produced in very simple cell models in an unambiguous way. Most importantly, it also shows an example of a chemical compound, found in nature, which is highly effective to completely suppress the abnormal changes of tau.

A simple cellular assay for the pathological modification of tau protein by abnormal hyperphosphorylation, as seen in Alzheimer's disease and a variety of other neurological disorders (tauopathies), has been developed by researchers at Sirenade Pharmaceuticals AG in Munich, Germany. The assay can be used to screen for drugs against these disorders, and the discovery of a very effective small molecule is described as an example.

Tau proteins are a family of neuron-specific proteins believed to play an important role in the organization of the skeleton of nerve cells. This protein has been known to be abnormally modified in degenerating neurons in Alzheimer's disease in a process called neurofibrillary degeneration. This form of neurodegeneration is thought to be a dominant cause of the massive loss of brain mass and function in many dementias, and its inhibition can be expected to change the prognosis of Alzheimer's disease significantly.

Cell models are essential tools to analyze the molecular mechanisms involved in specific disease processes, and also the mechanisms of drugs, which act against them. For the process of tau hyperphosphorylation, credible models with utility for drug discovery have

not been established to date. The model developed at Sirenade AG under the leadership of Dr. Hanno Roder, now at TauTaTis, Inc., fills this important gap.

"Inhibition of PHF-like Tau Hyperphosphorylation in SH-SY5Y Cells and Rat Brain Slices by K252a" by Gabriele Hübinger, Silvie LeCorre, Susanne Mühlbacher, Sandra Gordon, R. Paul Fracasso, Fred Hoffman, Sandrine Ferrand, Hans W. Klafki and Hanno M. Roder. *Journal of Alzheimer's Disease* 13 (3), April 2008.

# # #

Full text of the article is available to journalists upon request.

The *Journal of Alzheimer's Disease* (http://www.j-alz.com) is an international multidisciplinary journal to facilitate progress in understanding the etiology, pathogenesis, epidemiology, genetics, behavior, treatment and psychology of Alzheimer's disease. The journal publishes research reports, reviews, short communications, book reviews, and letters-to-the-editor. Groundbreaking research that has appeared in the journal includes novel therapeutic targets, mechanisms of disease and clinical trial outcomes. The *Journal of Alzheimer's Disease* has an Impact Factor of 3.058 according to Thomson Scientific Institute for Scientific Information's 2006 Journal Citation Reports. The Journal is published by IOS Press (www.iospress.nl).