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Promising compound restores memory loss and reverses symptoms of Alzheimer's

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A new ray of hope has broken through the clouded outcomes associated with Alzheimer's disease. A new research report published in January 2013 print issue of the *FASEB Journal* by scientists from the National Institutes of Health shows that when a molecule called TFP5 is injected into mice with disease that is the equivalent of human Alzheimer's, symptoms are reversed and memory is restored—without obvious toxic side effects.

"We hope that clinical trial studies in AD patients should yield an extended and a better quality of life as observed in mice upon TFP5 treatment," said Harish C. Pant, Ph.D., a senior researcher involved in the work from the Laboratory of Neurochemistry at the National Institute of Neurological Disorders at Stroke at the National Institutes of Health in Bethesda, MD. "Therefore, we suggest that TFP5 should be an effective therapeutic compound."

To make this discovery, Pant and colleagues used mice with a disease considered the equivalent of Alzheimer's. One set of these mice were injected with the small molecule TFP5, while the other was injected with saline as placebo. The mice, after a series of intraperitoneal injections of TFP5, displayed a substantial reduction in the various disease symptoms along with restoration of memory loss. In addition, the mice receiving TFP5 injections experienced no weight loss, neurological stress (anxiety) or signs of toxicity. The disease in the placebo mice, however, progressed normally as expected. TFP5 was derived from the regulator of a key brain enzyme, called Cdk5. The over activation of

Cdk5 is implicated in the formation of plaques and tangles, the major hallmark of Alzheimer's disease.

"The next step is to find out if this molecule can have the same effects in people, and if not, to find out which molecule will," said Gerald Weissmann, M.D., Editor-in-Chief of the FASEB Journal. "Now that we know that we can target the basic molecular defects in Alzheimer's disease, we can hope for treatments far better – and more specific – than anything we have today."

More information: Varsha Shukla, Ya-Li Zheng, Santosh K. Mishra, Niranjana D. Amin, Joseph Steiner, Philip Grant, Sashi Kesavapany, and Harish C. Pant. A truncated peptide from p35, a Cdk5 activator, prevents Alzheimer's disease phenotypes in model mice. FASEB J. January 2013 27:174-186; doi:10.1096/fj.12-217497

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