



PBT2 Demonstrates Rapid and Potent Effects in Cognition, Reduction of Brain Soluble Beta-amyloid and Significant Improvement in Synaptic Function in Mouse Models

- Morris Water Maze Test Data Demonstrates PBT2 Improves Memory Performance within 5 Days -

- Additional Studies Show Soluble Beta-amyloid in Brains of 15-month old Transgenic Alzheimer's Mice Dropped by 60% Within 24 Hours of Oral PBT2 Dosing -

Melbourne, Australia – July 20, 2006 – Professor Ashley Bush, MD, PhD, of the Mental Health Research Institute of Victoria (Australia) and co-founding scientist of **Prana Biotechnology Limited (NASDAQ: PRAN, ASX: PBT)** today presented data at the 10th International Conference on Alzheimer's Disease (ICAD) in Madrid demonstrating that in mouse models¹ PBT2:

- improved memory performance within five (5) days of oral dosing,
- rapidly reduced the levels of soluble beta-amyloid ("Abeta") in the brain, and
- restored normal function to Abeta impaired synapses.

Professor Bush's lecture, entitled, "Oral Treatment of APP Transgenic Mice with the Second-Generation 8-OH Quinoline, PBT2, Decreases Soluble Brain A β within 24 hours," was part of the "Hot Topics" session, which is devoted to exciting advances in Alzheimer's therapies.

In studies conducted by Professor Bush and his colleagues using the Morris Water Maze Test, it was demonstrated that PBT2 could quickly and significantly improve spatial memory – an important barometer of cognitive function – in seven-month old transgenic amyloid mice, which are a model for Alzheimer's disease. The Morris Water Maze Test involves remembering the location of a submerged platform, requiring the mouse to employ higher-level learning and spatial memory skills in order to successfully navigate the maze.

In addition, Professor Bush referenced studies he and colleagues performed on 15-month old transgenic Alzheimer's mice treated with 30 mg/kg PBT2, which showed the drug reduced soluble Abeta40 and Abeta42 levels by 60 percent within 24 hours of oral PBT2 administration. Professor Bush also presented mechanistic findings showing that PBT2 blocks the copper-dependent formation of amyloid oligomers, considered by many to be the toxic chemical entity leading to brain damage in Alzheimer's disease. Professor Bush showed that, by this mechanism, PBT2 in the rodent brain blocks synaptotoxicity caused by soluble beta-amyloid oligomers and restores LTP (long-term potentiation) – the neuronal electrical activity that underlies memory formation.

“This data is compelling and very exciting because it shows that PBT2 not only may facilitate the clearance of Abeta from the brain or prevent its production, but more importantly may improve cognition,” stated Professor Bush. “On the basis of the multiple encouraging results achieved to date, demonstrating that PBT2 has a rapid and potent mechanism of action, Prana is initiating a Phase II, double-blind, placebo-controlled trial of PBT2 in Alzheimer’s patients.

The observation that PBT1 (clioquinol)², a retired anti-amoebic drug, could halt cognitive decline in a pilot Phase IIa Alzheimer’s patient study was the original catalyst for the creation of Prana’s new generation MPAC (Metal Protein Attenuating Compound) chemical library. This platform of agents may have therapeutic utility in several key neurological disorders. Rodent pharmacokinetic studies have shown that the brain concentration of PBT2 is about 50-fold greater than clioquinol for an IV equivalent dose.

Geoffrey Kempler, Chairman and CEO of Prana, added, “We have made great strides in the development of our proprietary chemical library, and I believe we can, and will capitalize on our intellectual property in the MPAC platform in the future. We are very encouraged by these findings and look forward to confirming them in future human clinical trials.”

Safety studies in animals and healthy humans (Phase single and multiple dose), which were referenced by the researchers, indicate that PBT2 is well tolerated at doses proposed for Alzheimer’s treatment.

About 10th International Conference on Alzheimer’s Disease (ICAD)

The 10th International Conference on Alzheimer’s Disease is the largest gathering of Alzheimer researchers in history. Scientists from around the world will present and discuss the findings of more than 2,000 studies showcasing the newest treatment advances in Alzheimer’s disease and steps toward prevention.

About Prana Biotechnology Limited

Prana Biotechnology was established to commercialize research into Alzheimer’s disease and other major age-related neuro-degenerative disorders. The company was incorporated in 1997 and listed on the Australian Stock Exchange in March 2000 and listed on NASDAQ in September 2002. Researchers at prominent international institutions including the University of Melbourne, The Mental Health Research Institute and Massachusetts General Hospital, a teaching hospital of Harvard Medical School, discovered Prana’s technology.

For further information, please visit our web site at www.pranabio.com.

NB: this news was released in the United States at 10:15 a.m. (New York) on 19 July 2006 / 00:15 (Melbourne) on 20 July 2006.

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¹ The sample sizes studied in the tests conducted by Professor Bush and his colleagues was:

Morris Water maze study: n=7 (dosed) and 7 (vehicle)

24h study in 15month old Tg mice at 30mg/kg: n=7 (treated) and 8 (vehicle)

LTP experiment: n=8 (in each of 4 conditions)

² PBT1 (clioquinol) was found to be well tolerated and slowed cognitive decline in a pilot Phase II clinical trial of people with moderate stage Alzheimer's disease. Further trials of the drug were abandoned due to a manufacturing impurity, not a flaw in PBT1 itself.