

Neuron to Publish Prana's PBT2 preclinical research

- Significant cognitive benefits and reduction in Abeta demonstrates disease modification benefits
- Full data to be presented at ICAD in Chicago on July 29, 2008

Melbourne, Australia – July 10, 2008 – Prana Biotechnology Ltd (NASDAQ: PRAN, ASX: PBT) announced the publication of key research findings with its lead Alzheimer's Disease drug, PBT2. The article titled "Rapid restoration of cognition in Alzheimer's transgenic mice with 8-hydroxyquinoline analogs" is associated with decreased interstitial Abeta" appears in the current edition of the prestigious scientific journal *Neuron*, and can be viewed online.

The key findings reported are:

- PBT2 profoundly and rapidly improved cognition in transgenic mice.
- PBT2 prevented the formation of soluble Abeta oligomers, the form of Abeta believed to be the most toxic.
- PBT2 substantially reduced the amount of all forms of Abeta in the transgenic mouse brain, over a nine week period.
- PBT2, within hours of oral administration, significantly lowered soluble (interstitial) Abeta in the brain, sampled using *in vivo* microdialysis.
- Using a well established model for memory formation, PBT2 protected neurons in living brain tissue from the toxic effects of Abeta which impairs the signaling between neurons in Alzheimer's disease.

"The Alzheimer's field is eagerly awaiting the results of several clinical trials of therapies aimed at the toxic amyloid beta protein. Prana's PBT2 is uniquely positioned as an oral drug that neutralizes the toxicity of the amyloid beta protein and clears it from the brain. The positive findings in Alzheimer's mouse models along with the encouraging results from the phase II clinical trial of PBT2 greatly strengthen my belief that this drug will ultimately be shown to slow down disease progress in Alzheimer's patients," commented Dr. Rudolph Tanzi, Professor of Neurology at Harvard University, and Director of the Genetics and Aging Research Unit at MassGeneral Institute for Neurodegenerative Diseases.

In their discussion, the authors explain that healthy brain function is dependent upon the tightly regulated movement of metals within and between neurons. They speculate that with aging this restraint may be loosened, rendering the brain vulnerable to oxidative stress and the pernicious effects of Abeta accumulation. In the article, multiple modes of action for PBT2 are proposed based on the drug's intrinsic ability to capture and transport metals in the brain. This property explains how PBT2:

- inhibits the production of toxic free radicals and the formation of toxic "oligomeric" aggregates .
- promotes the solubilization and detoxification of Abeta aggregates and plaques
- transports metals into depleted neurons and thereby enhances the production of enzymes which break down Abeta, reducing its concentration in the brain.

The beneficial effects upon cognition are thus a net effect of the disaggregation of plaques, the detoxification of Abeta and enhanced removal of Abeta from the brain.

Associate Professor Robert Cherny, PhD, of the Mental Health Research Institute of Victoria (Australia), Head of Research of Prana Biotechnology Limited and a co-author on the paper, said "using *in vivo* microdialysis, we can monitor the effects of a drug on brain Abeta in real time in the conscious, freely-moving transgenic mouse. We can literally see the drug altering brain chemistry. The publication emphasises that the dramatic improvements in memory in mice seen with PBT2 are associated with reduction in this soluble (interstitial) Abeta."

Geoffrey Kempler, Chairman and CEO of Prana, noted that: "we are pleased that the research has been reported in *Neuron*, a highly regarded peer reviewed journal. Given this, together with the data from our recently completed Phase IIa trial (in press), we are very confident that our drug has the potential to be marketed as a treatment for Alzheimer's disease. Prana's approach is different to others because PBT2 targets toxic metal interactions in the brain."

The data will be presented at the 11th International Conference on Alzheimer's Disease (ICAD) in Chicago on July 29, 2008 by Associate Professor Robert Cherny describing key preclinical findings of PBT2 in a lecture entitled, "The 8-hydroxyquinoline analog PBT2 rapidly restores cognition and reduces soluble Abeta in Alzheimer's transgenic mice".

For access to the full journal article visit the company website at www.pranabio.com

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.

Forward Looking Statements

This press release contains "forward-looking statements" within the meaning of section 27A of the Securities Act of 1933 and section 21E of the Securities Exchange Act of 1934. The Company has tried to identify such forward-looking statements by use of such words as "expects," "intends," "hopes," "anticipates," "believes," "could," "may," "evidences" and "estimates," and other similar expressions, but these words are not the exclusive means of identifying such statements. Such statements include, but are not limited to any statements relating to the Company's drug development program, including, but not limited to the initiation, progress and outcomes of clinical trials of the Company's drug development program, including, but not limited to, PBT2, and any other statements that are not historical facts. Such statements involve risks and uncertainties, including, but not limited to, those risks and uncertainties relating to the difficulties or delays in financing, development, testing, regulatory approval, production and marketing of the Company's drug components, including, but not limited to, PBT2, the ability of the Company to procure additional future sources of financing, unexpected adverse side effects or inadequate therapeutic efficacy of the Company's drug compounds, including, but not limited to, PBT2, that could slow or prevent products coming to market, the uncertainty of patent protection for the Company's intellectual property or trade secrets, including, but not limited to, the intellectual property relating to PBT2, and other risks detailed from time to time in the filings the Company makes with Securities and Exchange Commission including its annual reports on Form 20-F and its reports on Form 6-K. Such statements are based on management's current expectations, but

actual results may differ materially due to various factors including those risks and uncertainties mentioned or referred to in this press release. Accordingly, you should not rely on those forward-looking statements as a prediction of actual future results.

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