



## **Prana Announces Publication in *Neurobiology of Aging Journal***

**MELBOURNE, Australia – January 18, 2008: Prana Biotechnology Limited (NASDAQ: PRAN / ASX: PBT)**, a biopharmaceutical company focused on the research and development of treatments for neurodegenerative disorders, today announced that the current online addition of *Neurobiology of Aging Journal*, has published a paper entitled, “Zinc and Copper Modulate Alzheimer A-beta Levels in Human Cerebrospinal Fluid,” which further supports Prana’s scientific approach in the treatment of Alzheimer’s Disease.

Results of the NIH study, conducted by Dorothea Strozyk, M.D. at The Albert Einstein College of Medicine in New York, and collaborators in the USA, Sweden and Australia, found that the removal of A-beta from the brain may be dependent upon maintaining normal levels of metals in the brain. The authors hypothesized that a breakdown in normal metal metabolism, accompanied by the accumulation of metals within the amyloid plaques, deprives metal dependent enzymes of the copper and zinc they need to drive the removal of A-beta from the brain.

Compounds synthesized by Prana chemists (MPACs) are expected to restore brain metals to normal levels by redistributing metals from A-beta plaques back to normal cells, rather than merely depleting brain tissue of metals. As a consequence, MPACs have been shown to restore normal function to A-beta impaired synapses and improve cognitive performance in mouse models of Alzheimer’s Disease. Unlike current approved Alzheimer’s therapies that only treat the symptoms of the disease, Prana’s lead MPAC compound, PBT2, targets the underlying cause of the disease.

Professor Ashley Bush senior author of the study from the Mental Health Research Institute of Victoria and Prana’s senior scientific consultant commented that, “Understanding that certain metal dependent enzymes degrade A-beta, these results show that the clearance of A-beta, which is damaged in Alzheimer’s Disease, could be influenced by metal levels in the CSF. As part of their mechanism of action, MPACs stop A-beta oligomers from trapping essential metals and return the metals to these clearance enzymes. This normalizes the removal of A-Beta, which we believe is key to a disease-modifying drug for Alzheimer’s Disease.”

### **About *Neurobiology of Aging Journal***

*Neurobiology of Aging Journal* publishes the results of studies in behavior, biochemistry, cell biology, endocrinology, molecular biology, morphology, neurology, neuropathology, pharmacology, physiology and protein chemistry in which the primary emphasis involves mechanisms of nervous system changes with age or diseases associated with age.

## **About Prana Biotechnology Limited**

Prana Biotechnology was established to commercialise research into Alzheimer's disease and other major age-related neurodegenerative 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 (Melbourne) and Massachusetts General Hospital, a teaching hospital of Harvard Medical School, contributed to the discovery of Prana's technology.

For further information, please visit the Company's web site at [www.pranabio.com](http://www.pranabio.com).

## **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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