



Cell publishes on the critical role of metals in Alzheimer's Disease.

New evidence strongly supports Prana's therapeutic strategy to regulate brain metals

Melbourne – September 3, 2010: Prana Biotechnology (NASDAQ:PRAN; ASX:PBT) today announced that Professor Ashley Bush, a co-founding scientist of Prana and a member of the Company's R&D Advisory Board, has in conjunction with the University of Melbourne, Australia, and Harvard Medical School, Boston, published an article that appears online today entitled: "Iron-export ferroxidase activity of beta-amyloid precursor protein is inhibited by zinc in Alzheimer's Disease" scheduled for the September 17 edition of **Cell**, the prestigious peer-reviewed, international biological research journal.

Prana's therapeutic strategy is to restore normal metal levels and distribution in the brain, such as zinc, which typically changes with aging and is exacerbated in Alzheimer's Disease, resulting in (i) reduced synaptic function and (ii) beta-amyloid reacting with the metals to form toxic oligomers that impair cognition.

The *Cell* publication now adds a third metal-dependent event, iron accumulation, which if left untreated, contributes to neuronal death in the Alzheimer's brain.

Sam Gandy, M.D Ph.D, Mount Sinai School of Medicine, New York, described this report, as "providing a major advance in deciphering the underlying causes of Alzheimer's Disease while at the same time pointing us toward a new and exciting strategy for treating or preventing the disease with a drug such as PBT2, which affects brain metals."

The evidence published in *Cell* shows that the source of beta-amyloid, the beta-amyloid precursor protein (APP), plays a hitherto unknown critical role in exporting iron out of neurons. If APP fails to carry out this role, iron builds up in the neurons contributing to oxidative stress, neurofibrillary tangle formation and ultimately neuronal cell death. Importantly, Prana scientists have demonstrated that synaptic zinc can prevent APP from performing its normal iron transporting role. Synaptic zinc levels and distribution changes in the Alzheimer's diseased brain, because zinc released into the synaptic space is drawn into the beta-amyloid that forms plaques in the synapse. This is problematic for the brain because neurons are deprived of the zinc required for neurotransmission and the zinc induces toxic beta-amyloid oligomer formation. Moreover, as now indicated by the **Cell** publication, the zinc caught up in the beta-amyloid can be exchanged or transported to the APP, impeding its ability to prevent iron buildup in neurons.

Accordingly, the maintenance of correct zinc levels in the brain is vital for neuronal function and ultimately cognition.

Prana's lead Alzheimer's drug, PBT2 acts as a zinc chaperone that transports zinc from the brain beta-amyloid deposits and returns it to neurons to facilitate normal neurotransmission. PBT2 has also recently been shown to have important neuroprotective and neurotrophic properties, consistent with its ability to prevent zinc from impairing the ability of APP to prevent iron overload.

Dr Rudolph Tanzi of Harvard Medical School, a Prana co-founding scientist and co-author of the **Cell** paper commented that "these findings are timely in distinguishing PBT2's unique mechanism of action from the recent clinical trial failures that target amyloid but did not address the need to restore synaptic metal homeostasis". PBT2 has completed a Phase 2a clinical trial where it significantly increased the cognitive performance of patients with Alzheimer's Disease after only 12 weeks of treatment.

Mr. Geoffrey Kempler, Executive Chairman of Prana, said, "The report is the most comprehensive validation to date of the importance of brain metals, which are the key therapeutic target of PBT2, in treating Alzheimer's Disease. These new findings strengthen the expectation that PBT2 will reverse brain damage in Alzheimer's Disease, and raise hopes that it will achieve further success in the next stage of clinical trials".

About Prana Biotechnology Limited

Prana Biotechnology was established to commercialise research into Alzheimer's Disease, Huntington'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.

About CELL

Cell publishes high impact papers that have come to form the foundation of contemporary life science research. *Cell* is at the forefront of exciting developments in biology, continually redefining the important areas of science with cutting edge papers. For further information visit the *Cell* website at cell.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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