



Prana Comments on *Archives of Neurology* Publication Which Highlights Critical Role of Brain Metals in Huntington Disease Progression

MGH team publishes data that supports the use of PBT2 in Huntington disease

Melbourne – 1 May 2012: Prana Biotechnology (NASDAQ:PRAN; ASX:PBT) today commented on the publication of new data of relevance to the current clinical trial, testing Prana's PBT2 as a treatment for Huntington disease.

The authors of the publication are led by Professor Diana Rosas of the Center for Neuroimaging of Aging and Neurodegenerative disease at Massachusetts General Hospital (MGH) in Boston. The paper, entitled "Alterations in Brain Transition Metals in HD", published in the *Archives of Neurology** describes how the rise in levels of Iron in the brains of people carrying the mutant gene which causes Huntington disease, correlates with the severity of symptoms and also predicts the time of disease onset. The article concluded that "an important and early role of altered metal homeostasis is suggested in the pathogenesis of Huntington disease" and this points to "metals as potential therapeutic targets". Selected patients in the current Reach2HD trial, testing PBT2, will be monitored using the imaging technology described in the publication.

According to Professor Rudy Tanzi, Prana's Chief Scientific Advisor and the Joseph P. and Rose F. Kennedy Professor of Neurology at Harvard University, "The new findings serve to further support the role of brain metal imbalances in the pathology of neurodegenerative diseases such as Alzheimer's disease and Huntington disease. It is becoming increasingly clear that PBT2 and Prana's other metal chaperone drugs could have broad utility as powerful modifiers of disease progress in a growing number of neurodegenerative diseases caused by misfolded proteins."

Ira Shoulson MD, Professor of Neurology, Pharmacology and Human Science at Georgetown University (Washington DC) and the Chair of the Executive Committee of the Huntington Study Group said "The data from the Harvard group are very encouraging and timely. The Reach2HD trial is underway and intended to characterize the safety and dosing parameters of PBT2, an experimental drug aimed at restoring function to neurons damaged by the pathological interaction between the mutant Huntingtin protein and transition metals. PBT2 has signaled some cognitive benefit in patients with Alzheimer disease that may also involve a pathological interaction between a protein and transition metals."

Huntington disease is a complex and severely debilitating genetic, neurodegenerative disease, for which there is no cure. It is caused by an abnormally high number of repeats of a DNA sequence (CAG) which encodes for the amino acid glutamine. The disease often affects young adults and, whilst associated with severe physical movement symptoms, progressively impacts the mind and emotions as well. The disease causes incapacitation and death about 15-25 years after onset. The disease affects 30,000 people in the US and about 70,000 worldwide.

There are no drugs either available or in development that have established clinical evidence for treating the cognitive decline associated with Huntington disease. In this study, Prana aims to demonstrate cognitive improvements as already demonstrated in a Phase IIa study in mild Alzheimer's patients treated with PBT2. The study will also investigate safety, functional, behavioural and motor benefits in this Huntington patient population.

PBT2 is concurrently being tested in a Phase II trial in Alzheimer's disease.

Key points from the publication

Using an advanced non-invasive MRI Imaging technique with pre-symptomatic and symptomatic patients, the authors show that:

- Iron levels in specific brain areas rise before symptoms appear and correlate with disease severity
- Iron levels increase with increased CAG repeat length
- Presymptomatic iron levels predict age of onset of symptoms

The authors found that the MRI data correlated well with the levels and anatomical distribution of iron in postmortem brain tissue.

**** Rosas HD, Chen YI, Doros G, Salat DH, Chen N-k, Kwong KK, Bush A, Fox J, Hersch SM (2012) Alterations in Brain Transition Metals in Huntington disease: An Evolving and Intricate Story. Arch Neurol archneurol.2011.2945.**

About Prana Biotechnology Limited

Prana Biotechnology was established to commercialize research into age-related neurodegenerative disorders. The Company was incorporated in 1997 and listed on the Australian Securities 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.

The Huntington Study Group

(www.huntington-study-group.org).

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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