



Prana accelerates PBT2 commercial development

- *Alzheimer's Disease Phase II Imaging Trial planned 3rd Qtr 2011*
- *Huntington's Disease Phase II Trial planned 4th Qtr 2011*

Melbourne – 20 April 2011: Prana Biotechnology (NASDAQ:PRAN; ASX:PBT) today announced its business strategy to accelerate development of its lead asset, PBT2. The strategy, that now includes Huntington's Disease, should enable Prana to target market approval for PBT2 several years sooner than previously planned and at considerably less cost.

The company has designed a Phase II placebo controlled double blind study in 100 mild Huntington's Disease (HD) patients, in Australia and the US, treated over 6 months. This trial will be conducted in parallel, to the previously announced 12 month Phase II brain imaging study in 40 mild Alzheimer's Disease (AD) patients, in Australia, supported by the US based Alzheimer's Drug Discovery Foundation. Recruitment for both trials is planned to commence in the second half of the year.

"We are excited by this strategy because from a commercial perspective these diseases are very complimentary. From our earlier Alzheimer's trial we showed that PBT2 significantly improves cognitive executive function. This is very relevant to Huntington's Disease given that these patients also suffer cognitive decline, for which there is no marketed treatment available. Success in the trial that we have announced today will position PBT2 as a frontrunner in the treatment of Huntington's Disease. We believe that PBT2 can bring the same cognitive benefits to Huntington's Disease patients that it did to Alzheimer's Disease patients" commented Mr Geoffrey Kempler, Prana's Executive Chairman.

"Because Huntington's Disease is a relatively uncommon disease, it is classed as an 'orphan indication' by regulators, a status that typically confers accelerated regulatory review by authorities and faster approval to market. That also means the cost will be considerably less" added Mr Kempler.

The Huntington's Disease Market

HD is a genetically inherited neurodegenerative disease resulting in severe motor dysfunction and cognitive decline. It affects 30,000 people in the US and about 70,000 worldwide. The only treatment currently approved for the disease only targets some of the motor loss symptoms and sells approx. \$250 million pa. There are no drugs in development that have established clinical evidence for treating cognitive decline. Prana aims, in this trial, to demonstrate the same cognitive benefits for HD patients that it has already demonstrated in AD patients treated with PBT2. A treatment for HD that addresses the underlying progression of the disease could generate sales of 750 million to \$1 billion pa.

What benefits has PBT2 already shown

In a Phase IIa trial of PBT2 in mild Alzheimer's Disease*, cognitive executive function was significantly improved in patients. Recently the company published that PBT2 was able to directly restore neurons critical to cognition in mouse models. In particular it was demonstrated that PBT2 increased the number of spines on the branches (or dendrites) of

neurons, an important means of permitting many more neurons to interconnect with any particular neuron thereby increasing the brain's capacity to carry out learning and memory functions.

These findings pointing to the ability of PBT2 to restore cognition in degenerative conditions, together with positive data achieved with PBT2 in mouse models of Huntington's Disease** provide confidence that PBT2 will be able to confer cognitive benefit to patients with HD and AD.

What Alzheimer's and Huntington's diseases have in common

Mechanistically, both AD and HD appear to involve a protein that combines with brain metals to become toxic. In Alzheimer's, Prana scientists believe that the formation of toxic A-beta protein oligomers leading to impaired synaptic function, is a metal dependent event. In Huntington's recent publications*** indicate that copper is critical in the formation of toxic oligomers of the Huntington's Disease protein, *huntingtin*, that causes the brain degeneration in HD patients.

"The demonstration that PBT2, a Metal Protein Attenuating Compound (MPAC) can redress metal imbalances in the brain and intercede in toxic oligomer formation offers a very promising therapeutic strategy to tackle Huntington's Disease" commented Dr Robert Cherny, Prana's Head of Research.

* Lannfelt *et al.* Lancet Neurology (2008) vol. 7, pp. 779-86; Lannfelt *et al.* Lancet Neurology (2009) vol. 8, pp. 981.

*Faux *et al.* J. Alzheimer's Disease 20 (2010) pp. 509-516

**Presented at the International Conference on Alzheimer's Disease, Honolulu Hawaii, 2010.

*** Fox *et al.* J. Bio. Chem. published as manuscript M110.199448

<http://www.jbc.org.Vcgi/doi/10.1074/jbc.M110.199448>

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.

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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