Prana's PBT434 Lowers Alpha-Synuclein and Prevents Neurodegeneration

Scientific Journal Acta Neuropathologica publishes pre-clinical data

MELBOURNE, Australia, and San Francisco USA 3 July 2017: Prana Biotechnology Ltd (ASX PBT: NASDAQ PRAN) today announced the article "**The novel compound PBT434 prevents iron-mediated neurodegeneration and alpha-synuclein toxicity in multiple models of Parkinson's disease"** was accepted for publication in the peer reviewed journal Acta Neuropathologica Communications. The peer reviewed article can be accessed from the following website: https://actaneurocomms.biomedcentral.com/articles/10.1186/s40478-017-0456-2

The publication is the culmination of ten years of research from scientists at the Florey Institute of Neuroscience and Mental Health, (Melbourne, Australia), investigating compounds from Prana Biotechnology's propriety chemical library. The novel drug candidate PBT434 is the first of a new generation of small molecules from the quinazolinone class of drugs that was specifically designed to block the accumulation and aggregation of alpha-synuclein, an abundant brain protein widely believed to be involved in the pathogenesis of Parkinson's disease and related disorders.

Not only was PBT434 shown to block alpha-synuclein accumulation, but it also prevented loss of nerve cells in the region of the brain primarily affected in Parkinson's disease, called the *substantia nigra*. To investigate the therapeutic potential of PBT434 to slow neurodegeneration, the researchers performed extensive animal testing in multiple Parkinson's disease models, including tests in mice that over-expressed the alpha-synuclein protein. These results showed that PBT434 lowered alpha-synuclein and its toxic effects and simultaneously improved motor performance.

If these findings are also observed in patients with diseases caused by alpha-synuclein, PBT434 could address a significant unmet medical need in preventing their progression.

The key findings from the publication in Acta Neuropathologica Communications are:

1. PBT434 prevents the formation of toxic alpha synuclein fibrils.
2. PBT434 prevents the formation of insoluble alpha synuclein in animals.
3. PBT434 prevents alpha synuclein mediated oxidative stress that induces cell death.
4. PBT434 protects against cell death and preserves neuronal circuitry in both transgenic and toxin mouse models of Parkinson's disease.
5. PBT434 improves motor behaviour in Parkinson's disease mouse models.
6. PBT434 normalises brain iron distribution.

David Stamler, M.D., Prana's Chief Medical Officer and Senior Vice President, Clinical Development said: "These findings are important because Parkinson's disease and the related synucleinopathies cause significant disability and diminish the independence of afflicted individuals. An agent which slows disease progression could have a great impact on reducing disease burden and improving quality of life. We are eager to begin clinical testing of PBT434."

The required animal testing has been completed and PBT434 is expected to begin human testing in a Phase 1 trial later this year.

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About Prana Biotechnology Limited

Prana Biotechnology was established to commercialise research into neurodegenerative diseases such as Alzheimer's disease, Huntington disease, and Parkinsonian disease. 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.

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 factions 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.