May 14, 2019

# ProMIS Neurosciences Announces First Quarter 2019 Results

*Company continues to show significant progress on developing highly selective antibodies targeting root cause of Alzheimer's and Parkinson's diseases and ALS*

TORONTO and CAMBRIDGE, MA, May 14, 2019 /PRNewswire/ - ProMIS Neurosciences, Inc. (TSX: PMN) (OTCQB: ARFXF), a biotechnology company focused on the discovery and development of antibody therapeutics targeting toxic oligomers implicated in the development of neurodegenerative diseases, today announced its operational and financial results for the three months ended March 31, 2019.

"Over the course of the first quarter of 2019, the value of our unique discovery and development platform was further evidenced as we made considerable progress in expanding our portfolio of opportunities in neurodegenerative diseases," stated Eugene Williams, ProMIS' Executive Chairman. Antibody candidates selectively targeting toxic forms of alpha synuclein for Parkinson's disease (PD) and toxic, aggregated forms of TDP43 for amyotrophic lateral sclerosis (ALS) were identified and further characterized to support ongoing pharmaceutical partnering discussions."

**Corporate Highlights**

- In January 2019, we completed a private placement of 9,560,000 common share units at a price of $0.23 per unit resulting in gross proceeds of approximately $2,198,800. Each unit consisted of one common share and one common share warrant. The common shares are subject to a four-month hold period from the date of issuance. The expiry of the warrant is subject to acceleration under certain conditions.

- In February 2019, we announced the identification of several antibody drug candidates showing best-in-class selectivity for toxic forms of alpha synuclein compared to other therapeutic antibodies in development for PD.

- Following this announcement, Dr. Neil Cashman, our CSO presented further results of

the Company's PD program at the International AD/PD 2019 Conference on March 31. The data presented showed how our novel drug discovery and development platform created antibodies that: 1) selectively bind to toxic forms of alpha synuclein while sparing healthy forms of alpha synuclein that are critical for proper cell metabolism and communication, and 2) block the neurotoxicity and the spread of the toxic forms of alpha synuclein in vitro.

- In January 2019, we appointed Timothy G. Rothwell to our Business Advisory Board. Mr. Rothwell is a highly accomplished pharmaceutical leader with more than 30 years of experience directing the development and commercialization of new therapies.

**Financial Results**

**Results of Operations – Three months ended March 31, 2019 and 2018**

Net loss for the three months ended March 31, 2019 was $2,446,577, compared to a net loss of $1,556,872 for the three months ended March 31, 2018, respectively. Included in the net loss amount for the three months ended March 31, 2019 were non-cash expenses of $263,872, representing share-based compensation and amortization of an intangible asset, compared to $329,011 for the three months ended March 31, 2018. The increase in the net loss in the three months ended March 31, 2019 reflects the costs associated with operating the Company's AD therapeutics program, increased contracted research and consultant salaries and associated costs, supporting its patent portfolio and general corporate expenditures.

Research and development expenses for the three months ended March 31, 2019 were $1,770,653, as compared to $698,007 in the three months ended March 31, 2018. The increase in research and development expense for the three months ended March 31, 2019 is primarily attributed to increased spending on external contract research organizations for internal programs, higher contracted research salaries and associated costs, patent costs, and higher share-based compensation.

General and administrative expenses for the three months ended March 31, 2019 were $675,924, as compared to $858,870 in the three months ended March 31, 2018. The decrease in general and administrative expense for the three months ended March 31, 2019 is primarily attributable to decreased share-based compensation offset by increased consultant salaries and associated costs.

**Outlook**

As a prelude to the first PMN310 clinical trial in AD, we anticipate using a novel biomarker approach that may show evidence of slowing of neuronal death early in the development program. To accomplish this, we plan to initiate a natural history evaluation of biomarker changes in untreated, early AD patients.

We will also continue to further characterize the potential benefits of our programs selectively targeting toxic aggregates of TDP43 in ALS and toxic forms of alpha synuclein in PD to further support ongoing pharmaceutical partnering discussions.

**About ProMIS Neurosciences, Inc.**

ProMIS Neurosciences, Inc. is a development stage biotechnology company focused on discovering and developing antibody therapeutics selectively targeting toxic oligomers implicated in the development and progression of neurodegenerative diseases, in particular Alzheimer's disease (AD), amyotrophic lateral sclerosis (ALS) and Parkinson's disease (PD). The Company's proprietary target discovery platform is based on the use of two complementary thermodynamic, computational discovery engines -ProMIS and Collective Coordinates – to predict novel targets known as Disease Specific Epitopes on the molecular surface of misfolded proteins. Using this unique precision approach, the Company is developing novel antibody therapeutics for AD, ALS and PD. ProMIS is headquartered in Toronto, Ontario, with offices in Cambridge, Massachusetts. ProMIS is listed on the Toronto Stock Exchange under the symbol PMN, and on the OTCQB Venture Market under the symbol ARFXF.

Company documents relating to the fiscal year 2018 annual report can be viewed on the System for Electronic Document Analysis and Retrieval (SEDAR) at the link below:

https://www.sedar.com/search/search_en.htm

Visit us at www.promisneurosciences.com or follow us on Twitter and LinkedIn

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