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ProMIS Neurosciences Publishes in the Journal of Biological Chemistry on the Interaction Between Pathogenic Proteins as a Treatment Target for ALS

Study results support ProMIS' TDP-43 misfolding-specific epitope as a potential therapeutic target for Amyotrophic Lateral Sclerosis (ALS)

CAMBRIDGE, Massachusetts and TORONTO, Ontario, April 09, 2024 (GLOBE NEWSWIRE) -- ProMIS Neurosciences Inc.(Nasdaq: PMN), a biotechnology company focused on the generation and development of antibody therapeutics targeting toxic misfolded proteins in neurodegenerative diseases such as Alzheimer's disease (AD), amyotrophic lateral sclerosis (ALS) and multiple system atrophy (MSA), today announced the publication of supportive preclinical data in the *Journal of Biological Chemistry* in an article titled, "Tryptophan residues in TDP-43 and SOD1 modulate the cross-seeding and toxicity of SOD1." ProMIS is developing antibodies selectively targeting misfolded forms of TDP-43 and SOD1.

ALS is a fatal neurodegenerative disease of motor neurons. Toxic aggregates of superoxide dismutase-1 (SOD1) and TAR DNA-binding protein 43 (TDP-43) in motor neurons are characteristic of ALS. The study showed that these two proteins interact such that misfolding of TDP-43 leads to misfolding and aggregation of SOD1 in a cell system and promotes motor neuron damage in zebrafish.

"Publication of these data underscores the connection of misfolded proteins and ALS and supports targeting our TDP-43-specific epitope with PMN267 as a potential therapeutic approach," stated Neil Warma, Chief Executive Officer of ProMIS Neurosciences. "PMN267 is advancing through preclinical development and is showing promise as a potential treatment for ALS. We are particularly pleased to have our data published in this well-regarded, peer-reviewed journal as it adds to the growing body of scientific knowledge on the pathogenic role of misfolded proteins in neurodegenerative diseases."

The study demonstrated a pathologic synergy between SOD1 and TDP-43 in cell culture and in zebra fish. The interaction between these 2 proteins was characterized at the molecular level. The results indicated that the misfolded TDP-43 epitope previously identified by ProMIS and targeted by PMN267 contains a tryptophan amino acid critical to the pathogenic interaction with SOD1 thereby providing further biological support for the potential of therapeutic intervention with PMN267.

"These findings provide an important contribution to our understanding of

ALS/FTLD molecular pathology and continue to validate our approach of targeting misfolded proteins as a therapeutic strategy that translates across multiple neurodegenerative diseases,” said Neil Cashman, M.D., Chief Scientific Officer and Co-founder of ProMIS.

The complete article can be accessed online [here](#).

About ProMIS Neurosciences Inc.

ProMIS Neurosciences Inc. is a clinical stage biotechnology company focused on generating and developing antibody therapeutics selectively targeting toxic misfolded proteins in neurodegenerative diseases such as Alzheimer’s disease (AD), amyotrophic lateral sclerosis (ALS) and multiple system atrophy (MSA). The Company’s proprietary target discovery engine applies a thermodynamic, computational discovery platform - ProMIS™ and Collective Coordinates - to predict novel targets known as Disease Specific Epitopes on the molecular surface of misfolded proteins. Using this unique approach, the Company is developing novel antibody therapeutics for AD, ALS and MSA. ProMIS has offices in Toronto, Ontario and Cambridge, Massachusetts.

Forward-Looking Statements

This press release contains forward-looking statements that are made pursuant to the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. Certain information in this news release constitutes forward-looking statements and forward-looking information (collectively, “forward-looking information”) within the meaning of applicable securities laws. In some cases, but not necessarily in all cases, forward-looking information can be identified by the use of forward-looking terminology such as “plans”, “excited to”, “targets”, “expects” or “does not expect”, “is expected”, “an opportunity exists”, “is positioned”, “estimates”, “intends”, “assumes”, “anticipates” or “does not anticipate” or “believes”, or variations of such words and phrases or state that certain actions, events or results “may”, “could”, “would”, “might”, “will” or “will be taken”, “occur” or “be achieved”. In addition, any statements that refer to expectations, projections or other characterizations of future events or circumstances contain forward-looking information. Specifically, this news release contains forward-looking information relating to the Company’s expectations regarding its clinical development of its lead product, PMN310, for Alzheimer’s disease. Statements containing forward-looking information are not historical facts but instead represent management’s current expectations, estimates and projections regarding the future of our business, future plans, strategies, projections, anticipated events and trends, the economy and other future conditions. Forward-looking information is necessarily based on a number of opinions, assumptions and estimates that, while considered reasonable by the Company as of the date of this news release, are subject to known and unknown risks, uncertainties and assumptions and other factors that may cause the actual results, level of activity, performance or achievements to be materially different from those expressed or implied by such forward-looking information, including, but not limited to, the Company’s ability to fund its operations and continue as a going concern, its accumulated deficit and the expectation for continued losses and future financial results. Important factors that could cause actual results to differ materially from those indicated in the forward-looking information include, among others, the factors discussed throughout the “Risk Factors” section of the Company’s most recently filed annual information form available on www.SEDAR.com, in Item 1A of its Annual Report on Form 10-K for the year ended December 31, 2022 and the section entitled “Risk Factors” in its Post-Effective Amendment No. 1 to Form S-1, filed March 17, 2023,

each as filed with the Securities and Exchange Commission, and subsequent quarterly reports. Except as required by applicable securities laws, the Company undertakes no obligation to publicly update any forward-looking information, whether written or oral, that may be made from time to time, whether as a result of new information, future developments or otherwise.

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