

Vigil Neuroscience Highlights Publication on ALSP Genetic Mutation Prevalence in Neurology Genetics

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- CSF1R pathogenic and likely pathogenic variants reported to have an approximate prevalence of 281 per 1 million -

- New research supports updated estimates to U.S. prevalence of approximately 19,000 and approximately 29,000 in the EU and UK -

WATERTOWN, Mass., Aug. 01, 2024 (GLOBE NEWSWIRE) -- <u>Vigil Neuroscience, Inc.</u> (Nasdaq: VIGL), a clinical-stage biotechnology company committed to harnessing the power of microglia for the treatment of neurodegenerative diseases, today announced a peer-reviewed research publication that reported new data on the prevalence and clinical significance of *CSF1R* gene variants in the UK population. The research, published in the journal *Neurology Genetics* and conducted by Wade *et al.*, suggests the estimated prevalence of adult-onset leukoencephalopathy with axonal-spheroids and pigmented glia (ALSP) is underreported in the U.S., EU, and UK.

The publication, titled "*CSF1R*-Related Disorder: Prevalence of *CSF1R* Variants and Their Clinical Significance in the UK Population," consisted of a search for pathogenic and likely pathogenic *CSF1R* variants among the sequencing data within the UK Biobank, in combination with medical history and MRI results. ALSP is caused by a mutated *CSF1R* gene, classifying it as a *CSF1R*-Related Disorder.

"Extrapolating from the frequency of pathogenic and likely pathogenic *CSF1R* variants in the UK Biobank, we can conservatively estimate that there are more than 2 million carriers worldwide," said David Lynch, M.D., Ph.D., Consultant Neurologist, National Hospital for Neurology & Neurosurgery and UCL Institute of Neurology in London, and corresponding author of the publication. "These data suggest that damaging mutations in *CSF1R* are more common than previously estimated in the general population and are associated with diagnoses for other cognitive, psychiatric and movement disorders. Increased genetic screening will help us better understand the prevalence of *CSF1R*-Related Disorders, like ALSP, as well as allow us to provide more timely diagnoses, specialized information, and services created to support people living with this devastating disease."

ALSP, a rare neurodegenerative disorder, is commonly misdiagnosed as other, more common neurodegenerative diseases, and can only be confirmed with a genetic test. To better understand the prevalence of ALSP, it is important to understand the prevalence of the causative genetic mutations. Prior to this publication, it was estimated there may be approximately 10,000 people living with ALSP in the U.S. with similar prevalence outside of the U.S. Based on these new data, the Company now estimates U.S. prevalence of ALSP is approximately 19,000 while the estimated combined EU and UK prevalence is approximately 29,000.

"Deepening our understanding of the prevalence of ALSP – and learning that it is likely higher than previously thought – is helpful not only for expanding disease awareness, but as a means of improving the journey to diagnosis," said Petra Kaufmann, M.D., F.A.A.N., Chief Medical Officer at Vigil. "Understanding the prevalence of a disease has a direct impact on its awareness in the healthcare community, improving the diagnostic process and the patient experience. This publication further underlines the sense of urgency we feel toward our ongoing efforts to address the severe unmet need of the ALSP community."

About Vigil Neuroscience

Vigil Neuroscience is a clinical-stage biotechnology company focused on developing treatments for both rare and common neurodegenerative diseases by restoring the vigilance of microglia, the sentinel immune cells of the brain. Vigil is utilizing the tools of modern neuroscience drug development across multiple therapeutic modalities in its efforts to develop precision-based therapies to improve the lives of patients and their families. Iluzanebart, Vigil's lead clinical candidate, is a fully human monoclonal antibody agonist targeting human triggering receptor expressed on myeloid cells 2 (TREM2) in people with adult-onset leukoencephalopathy with axonal spheroids and pigmented glia (ALSP), a rare and fatal neurodegenerative disease. Vigil is also developing VG-3927, a novel small molecule TREM2 agonist, to treat common neurodegenerative diseases associated with microglial dysfunction, with an initial focus on Alzheimer's disease (AD) in genetically defined subpopulations.

Forward-Looking Statements

This press release includes certain disclosures that contain "forward-looking statements" of Vigil Neuroscience ("Vigil" or the "Company") that are made pursuant to the safe harbor provisions of the federal securities laws, including, without limitation, express or implied statements regarding: estimates of the number of CSF1R carriers worldwide and the estimated prevalence of ALSP; and Vigil's ability to impact the diagnostic process and the patient experience, and the expected therapeutic benefits of our programs. Forward-looking statements are based on Vigil's current expectations and are subject to inherent uncertainties, risks and assumptions that are difficult to predict. Factors that could cause actual results to differ include, but are not limited to, risks and uncertainties related to uncertainties inherent in the development of product candidates, including the conduct of research activities and the conduct of clinical trials; whether results from preclinical studies and clinical trials will be predictive of the results of later preclinical studies and clinical trials; as well as the risks and uncertainties identified in the Company's filings with the Securities and Exchange Commission (SEC), including Vigil's Quarterly Report on Form 10-Q for the quarter ended March 31, 2024 and in any subsequent filings Vigil makes with the SEC. Forward-looking statements contained in this announcement are made as of this date, and Vigil undertakes no duty to update such information except as required under applicable law. Readers should not rely upon the information on this page as current or accurate after its publication date.

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