Amlogenyx

Amlogenyx Announces Expansion of Board of Directors and Formation of Scientific Advisory Board

Advancing a novel gene therapy to treat Alzheimer's disease

Appointed three Alzheimer's experts to newly formed Scientific Advisory Board

Gene therapy expertise enhanced on the board of directors with a new Independent member

NOVATO, Calif., August 11, 2025 – Amlogenyx Inc., a biotechnology company focused on developing gene therapies for Alzheimer's disease and other neurodegenerative diseases, today announced the appointment of Sukumar Nagendran, M.D., to the Board of Directors. The Company also announced the formation of its Scientific Advisory Board (SAB), with the appointment of three members, including Rudolph E. Tanzi, Ph.D., who will serve as chair, Frederick R. Maxfield, Ph.D. and Sangram S. Sisodia, Ph.D.

"I am honored to join the Board of Amlogenyx to support further advancement of its mission to revolutionize the treatment of Alzheimer's disease. This neurodegenerative disease steals precious years from individuals and profoundly affects families as they witness the gradual decline of their loved ones," said Dr. Nagendran. "With the exceptional expertise of the newly formed Scientific Advisory Board, Amlogenyx is uniquely positioned to advance a treatment option that could bring much-needed hope and relief to those affected by this devastating disease."

The appointment of Dr. Nagendran to the Board enhances strategic direction and governance of the Company at a critical time as it continues to advance the development of a one-time gene therapy for Alzheimer's disease that could significantly alter the course of the disease by preventing and reversing amyloid plaque build-up and promoting neuronal health.

Establishment of the SAB will provide guidance to the Amlogenyx scientific team on its proprietary approach utilizing an AAV9 gene therapy to deliver the protective enzyme cathepsin A (PPCA) directly to the brain, which has been demonstrated to reduce Aβ42 accumulation beyond the capabilities of traditional monoclonal antibody treatments in preclinical models of Alzheimer's disease.

Dr. Nagendran serves as president and head of Research and Development at Taysha Gene Therapies. He has more than 25 years of experience in gene therapy development, clinical development strategy, medical affairs, diagnostics, payer strategy and commercialization of therapeutic products. He previously held positions at Jaguar Gene Therapy, AveXis, Quest Diagnostics and Pfizer.

About the Scientific Advisory Board

Dr. Tanzi (chair) is the director of the Genetics and Aging Research Unit, director of the Henry and Allison McCance Center for Brain Health, and co-director of the Mass General Institute for Neurodegenerative Disease at Massachusetts General Hospital. He also serves as the Joseph P. and Rose F. Kennedy Professor of Neurology at Harvard Medical School. He co-discovered the first Alzheimer's disease gene, the amyloid precursor protein (APP) gene, and the two other early-onset familial Alzheimer's disease genes, presenilin 1 and presenilin 2. He and his team were trailblazers in using human stem cells to create three-dimensional mini human brain organoids and 3D neural-glial culture models of Alzheimer's disease, known as "Alzheimer's-in-a-Dish." These innovative models replicate key pathological hallmarks of Alzheimer's disease, significantly accelerating and reducing the cost of drug screening processes.

Frederick R. Maxfield, Ph.D., is a professor of biochemistry at Weill Cornell Medicine. He has made significant contributions through his application of quantitative imaging methods in membrane biology. He pioneered the use of fluorescence microscopy for the quantitative analysis of membrane traffic, leading to groundbreaking insights into the endocytic trafficking of proteins and lipids. His work included the initial demonstration that endosomes are acidic, a discovery that highlighted the essential role of acidification in many endosomal functions.

Sangram S. Sisodia, Ph.D., is the Thomas A. Reynolds Sr. Family Professor of Neurobiology, professor of neurology, and professor of the Neuroscience Institute at the University of Chicago. For more than three decades, he and his research team have developed a comprehensive research program that integrates genetics, neurobiology, molecular and cellular data to understand the cellular and molecular biology of key molecules linked to the onset of Alzheimer's disease. His groundbreaking contributions have significantly advanced the understanding of Alzheimer's disease. Dr. Sisodia's recent focus on the role of the microbiome in Alzheimer's continues to shape the field and propel innovative research.

About Amlogenyx

Amlogenyx is dedicated to the development of a novel gene therapy strategy to reduce amyloid accumulation in the brain in order to prevent and treat diseases like Alzheimer's disease. We are developing a novel protease therapy shown to reduce amyloid (Aβ42) accumulation in preclinical models of Alzheimer's disease. Amyloid accumulation has been shown to predict progression of Alzheimer's disease, and treatment with monoclonal antibodies to reduce amyloid has been shown to potentially slow the disease but not stop or reverse it. There is a clear need for better treatment options. Dr. Alessandra d'Azzo, Ph.D., at St. Jude Children's Research Hospital in Memphis identified a more potent method of degrading amyloid within neuronal cells that relies on the protective protein cathepsin A (PPCA)/neuraminidase (neu1) complex. Ultragenyx studied the effects of PPCA in vitro and showed it could potently degrade amyloid in various oligomeric and higher forms. Ultragenyx then worked with Dr. D'Azzo to develop an AAV9 gene therapy that can deliver supplemental PPCA enzyme to the brain and has shown a substantial reduction in Aβ42 accumulation in murine models of Alzheimer's disease that exceeds that observed with monoclonal antibodies. Amlogenyx is a subsidiary of Ultragenyx.

For more information on Amlogenyx, please visit the company's website at: https://www.amlogenyx.com/.

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