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PRESS RELEASE

Samumed Doses First Subject in Phase 1 Trial of SM07883, a Potential Treatment for Alzheimer's Disease

DYRK1A Inhibition as a Potential Treatment for Alzheimer's Disease Supported by Data Presented at International Conference on Alzheimer's and Parkinson's Diseases (AD/PD™ 2019)

SAN DIEGO – April 4, 2019 – Samumed, LLC, announced today that it has dosed the first subject in its phase 1 trial of SM07883, a potential treatment for Alzheimer's disease (AD). SM07883 is a novel, oral, small-molecule designed to reduce disease pathology by selectively inhibiting dual-specificity tyrosine phosphorylation-regulated kinase 1A (DYRK1A).

Yusuf Yazici, M.D., Chief Medical Officer of Samumed, said, "The accumulation of anomalous Tau proteins in the grey matter of Alzheimer's patients' brains has been strongly linked to the progression of the disease for decades. In preclinical studies, the observed significant reduction in Tau hyperphosphorylation and aggregation in animals treated with SM07883 compared to controls points to DYRK1A as a potential target for Alzheimer's disease and reinforces our rationale for proceeding with this phase 1 study."

This is a phase 1, open-label, dose-escalation study of a single ascending dose of orally administered SM07883 being conducted in healthy subjects in Australia. Sequential groups of healthy subjects will receive single doses of SM07883 (5 to 180 mg) with safety evaluations after each dose level. The primary endpoints of the study include safety, tolerability, and pharmacokinetics.

Samumed recently presented data at the 14th International Conference on Alzheimer's and Parkinson's Diseases (AD/PD™ 2019), held in Lisbon, Portugal from March 26-31, 2019. In an animal model of AD, triple transgenic (3xTG-AD) mice treated with SM07883 once per day for six months showed improved cognitive performance.

In other studies, SM07883 demonstrated selective and potent inhibition of DYRK1A activity, a protein that causes a biochemical modification (known as hyperphosphorylation) of Tau protein in the brain. In a mouse model of AD, daily treatment with SM07883 for three months demonstrated significant reduction in Tau hyperphosphorylation, the accumulation of Tau, the formation of neurofibrillary tangles (another abnormal brain tissue feature of AD), and improved behavior compared to vehicle.

About Alzheimer's Disease

Alzheimer's disease (AD), the most common cause of dementia, is a chronic neurodegenerative disease affecting an estimated 5.8 million people in the U.S. and over 46 million people worldwide. The disease is characterized by progressive memory loss and slow progression to

severe difficulty in accessing basic brain functions, prompting mental disorders. With the world's aging population, AD is quickly becoming "The Disease of the Century," a global epidemic, and a socioeconomic burden impacting families, social service, and healthcare delivery systems. Currently available therapies treat symptoms, not the disease itself, which is ultimately fatal.

About Samumed and SM07883

SM07883 is an oral small-molecule dual-specificity tyrosine phosphorylation-regulated kinase 1A (DYRK1A) inhibitor being developed as a potential therapy for the treatment of Alzheimer's disease. Preclinical data suggested that SM07883 reduced Tau and amyloid pathology, neuroinflammation, and improved behavior compared to controls. Additional information on Samumed's SM07883 Alzheimer's disease program can be found here: https://www.samumed.com/pipeline/detail.aspx?id=18.

Learn more about Samumed's technology platform and potential regenerative drug candidates at https://www.samumed.com/pipeline/default.aspx.

Corporate Contact:
Erich Horsley
Samumed, LLC
erich@samumed.com
858-365-0200

Investor Contact:
Ashley Robinson
LifeSci Advisors
arr@lifesciadvisors.com
617-535-7742

Media Contact:
Matt Middleman, M.D.
LifeSci Public Relations
matt@lifescipublicrelations.com
646-627-8384