Jacob Falck Hansen appointed as Chief Executive Officer of Vesper Bio

Copenhagen, Denmark, 01 July 2025 – Vesper Bio ApS ("Vesper" or "the Company"), a clinical stage biotech developing an oral therapy for frontotemporal dementia, today announces the appointment of Jacob Falck Hansen as Chief Executive Officer (CEO).

Jacob Falck Hansen brings more than 15 years of leadership experience from the pharmaceutical and biotech industry. He is a Partner at Lundbeckfonden BioCapital, the largest shareholder in Vesper Bio, and currently serves as board director at Notify Therapeutics and Kvantify, and as a board observer at Cytoki Pharma and SNIPR Biome.

Prior to joining Lundbeckfonden BioCapital, he was responsible for corporate strategy at Novo Nordisk A/S and acted as a trusted advisor to its executive management and board of directors. Jacob holds a MSc in Biochemistry and a PhD from the University of Copenhagen.

Jacob succeeds Paul Little, who has been overseeing Vesper Bio's successful journey from a preclinical to a clinical stage company.

Christian Elling, Managing Partner, SVP, Lundbeckfonden BioCapital said: *"Vesper is at a pivotal moment in its development, running a clinical trial in asymptomatic carriers of a mutation causing frontotemporal dementia. We are committed to maintaining this momentum and to bring hope to patients suffering from frontotemporal dementia. We would like to thank and acknowledge Paul Little for his leadership bringing Vesper to this exciting point and we wish him well with his future plans."*

Jacob Falck Hansen, Chief Executive Officer of Vesper, said: *"The company's pioneering work in sortilin biology and its commitment to addressing major unmet needs in neurodegenerative diseases are truly inspiring. I look forward to working with the talented Vesper team to advance our mission and bring transformative therapies to patients and their families."*

Vesper's lead candidate VES001 is currently in a Phase Ib/IIa trial in asymptomatic carriers of a mutation in the progranulin-coding gene (GRN), which causes a genetically-determined type of frontotemporal dementia (FTD), called FTD-GRN. The open-label trial assesses the clinical efficacy, safety and tolerability of VES001 and is expected to read out in H2 2025. For more information about the trial (NCT06705192), please visit www.clinicaltrials.gov.

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About Vesper Bio

Vesper Bio is a clinical stage biotech and world leader in sortilin receptor biology. Its lead programme uses a sortilin inhibitor to rebalance levels of progranulin in patients where the sortilin receptor would otherwise reduce circulating and extracellular progranulin, contributing to disease. Progranulin is a protein that the body uses to regulate cell growth, survival, repair and avoid inflammation. Low progranulin levels are believed to be a factor in cell dysfunction and damage in a range of indications across neurology. By normalising progranulin levels, Vesper believes its compounds will have a disease modifying effect, protecting and preserving the remaining cells.

Its lead compound, VES001, is a patient friendly, first-in-class, brain penetrant, oral treatment which targets progranulin deficiency, a major underlying cause of frontotemporal dementia (FTD). As an orally delivered small molecule, VES001 is able to cross the blood-brain barrier and is an ideal dosing method among these patients due to their rapidly declining mental state. The efficacy of VES001 is currently being evaluated in a Phase Ib/IIa trial in asymptomatic carriers of a mutation in the progranulin-coding gene (GRN), which causes a leading genetically determined type of frontotemporal dementia (FTD), called FTD-GRN.

About Frontotemporal Dementia (FTD)

Frontotemporal dementia (FTD), also known as frontotemporal lobar degeneration (FTLD), is a group of brain disorders that cause degeneration in the frontal and temporal lobes of the brain. FTD impacts a person's behaviour, judgement, communication and ability to participate in all activities of daily living. It is the most common cause of dementia in people under the age of 60 and is often misdiagnosed as Alzheimer's disease. FTD-GRN is a form of FTD caused by mutations of the progranulin gene (GRN), resulting in low progranulin levels. FTD-GRN is thought to account for a quarter of familial FTD cases. Low levels of progranulin are associated with cellular dysfunction, neuroinflammation and other neuronal damage across a variety of neurological indications.

For further information please visit, https://www.vesperbio.com/