

Summary of

NeuroRestore – a novel therapeutic concept for the treatment of Alzheimer’s disease

K. Önnestam¹, B. Nilsson¹, M. Rother¹, E. Rein-Hedin^{2,3}, J. Bylund², P. Anderer⁴, M. Kemethofer⁴, M.M. Halldin¹, J. Sandin^{1,5}, M. Segerdahl^{1,5}

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For questions or more information mail: marta.segerdahl@alzecurepharma.com

Alzheimer’s disease is a devastating neurodegenerative disease, affecting approximately 50 million patients worldwide. Not only patients, but also their families are heavily burdened with the reduced quality of life, associated disabilities, reduced cognition and activities of daily living as well as economic burden. Current symptomatic treatments have only minor efficacy. Anti-amyloid antibody therapies have become available in the USA, but not yet in the rest of the world. They can slow progression of the disease, but not improve cognition, and many patients will not be eligible for antibody therapies.

Brain-Derived Neurotrophic Factor (BDNF) and Nerve Growth Factor (NGF) are proteins that play a crucial role in the growth, development, and maintenance of neurons in the brain. They are involved in promoting the survival of existing neurons and the growth of new neurons and synapses. BDNF is often considered to be a key player in the plasticity of the brain, which refers to its ability to adapt and change in response to experiences and environmental factors.

Research suggests that BDNF levels and functioning are disrupted in individuals with Alzheimer’s disease, directly associated with the progression of neurodegeneration. BDNF is also involved in promoting neuroplasticity, which is the brain's ability to reorganize itself by forming new connections between neurons. Neuroplasticity is important for learning, memory, and adapting to stress. We have demonstrated in animal models that ACD856 can improve cognition in aged animals and in animals with neurodegeneration.

Compounds in AlzeCure’s NeuroRestore platform are so called Trk-PAMs, which stimulate specific signaling pathways in the central nervous system known as neurotrophins, including NGF and BDNF. The leading drug candidate in the platform, ACD856, has recently completed clinical phase I studies. In the published paper reporting data from the multiple ascending dose study we have demonstrated a good safety profile, good pharmacokinetic properties and positive proof-of-mechanism as shown by qEEG, supporting continued development of the program.

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References:

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