Cognition Therapeutics Elucidates Genetic Mutation that Protects Against Alzheimer's Disease

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Pittsburgh, October 19, 2020 — Cognition Therapeutics, Inc., a clinical stage neuroscience company developing drugs that treat neurodegenerative disorders by regulating cellular damage response pathways, today announced that a peer-reviewed manuscript, entitled, "Icelandic Mutant Human Amyloid-β 1-42 Peptide Forms Fewer Oligomers with Lower Binding Affinity than Wild-Type Peptide," has been published online in the Journal of Neurochemistry (doi:10.1111/jnc.15212).

It has been well documented that individuals with the A673T mutation ("Icelandic mutation") in the protein Aβ are four times less likely to develop Alzheimer's disease than are noncarriers, making this the strongest protective mutation discovered to date for this disease. Therapeutics that mimic the effects of protective protein mutations have proven effective in several human diseases and disorders. For this reason, the research community has shown great interest in understanding the biological changes caused by this mutation, with the aim of developing a therapeutic that replicates those conditions.

Cognition scientists conducted a series of studies to clarify the mechanism by which this genetic mutation confers protection against Alzheimer's disease. Cognition's research demonstrates that these mutant Aβ oligomers bind with a four-fold lower affinity to synapses in the brain than do normal oligomers. When bound to synapses, Aβ oligomers cause the brain cell dysfunction and memory failure characteristic of Alzheimer's disease. This suggests that the reduction in oligomer binding is the main reason Icelandic mutation carriers are four times less likely to develop Alzheimer's disease.

"These findings are an important validation of our approach to the treatment of Alzheimer's disease," explained Susan Catalano, Ph.D., Cognition's founder and chief science officer. "Our drug candidate, CT1812, is the only therapeutic currently in clinical development that has demonstrated an ability to reduce oligomer binding to brain cell synapses, and so is the only drug that mimics the effects of the strongest protective mutation yet discovered for Alzheimer's. This gives us great confidence that CT1812 will be able to provide a meaningful clinical benefit for Alzheimer's patients."

"This research will have a profound impact on Alzheimer's disease-modifying therapeutic research, as it lays out a clear mechanism for effective Alzheimer's therapeutics," concluded Lisa Ricciardi, president and CEO of Cognition Therapeutics. "Our lead candidate for Alzheimer's disease, CT1812, is designed to displace Aβ oligomers from their binding sites and has shown early clinical evidence that it improves biomarkers of the disease and may improve cognition in symptomatic patients. We look forward to completing the ongoing clinical trials of CT1812 to learn more about its activity and potential for disease modification in this underserved patient population."

About Cognition Therapeutics, Inc.
Cognition Therapeutics, Inc. has discovered and is developing a pipeline of novel, disease modifying, oral drug candidates to treat a broad array of neurodegenerative and neuro-ophthalmic disorders. Our pipeline compounds uniquely target the σ-2 receptor, a key regulator of the cellular damage response. CT1812, our lead product candidate, is being assessed in a comprehensive clinical program for Alzheimer's disease, including a 540-person Phase 2 study in collaboration with ACTC and supported by a competitive grant from the National Institute on Aging. Additional information about Cognition and its product candidates may be found online at www.cogrx.com.