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Company name: Modalis Therapeutics Corporation

Stock exchange listing: Tokyo Stock Exchange

Ticker/Code number: 4883

URL: <https://www.modalistx.com/en/>

Corporate Representative: Haruhiko Morita

A Patent Grant for "*Method for Treating Alzheimer's Disease by Targeting the MAPT Gene*"

Modalis Therapeutics (Tokyo, Japan & Waltham, MA; President and CEO: Haru Morita, "Modalis") today announced that the Japan Patent Office (JPO) has granted the company's patent application entitled "Method for Treating Alzheimer's Disease by Targeting the MAPT Gene" (Application No. JP 2023-501249).

This patent covers therapeutic methods and compositions utilizing Modalis' proprietary CRISPR-GNDM[®] epigenome editing technology to specifically and effectively suppress the expression of the human microtubule-associated protein tau (MAPT) gene. The invention is expected to provide a novel therapeutic approach for tauopathies, including Alzheimer's disease and frontotemporal dementia with parkinsonism linked to chromosome 17 (FTDP-17). The inventor of the patent are Dr. Talha Akbulut, Dr. Tetsuya Yamagata, and other members of Modalis' research and development team.

Across multiple large cohorts, tau PET burden shows stronger associations with cognitive decline and disease severity than amyloid PET or MRI, and is among the best predictors of longitudinal clinical progression. By suppressing abnormal accumulation and aggregation of phosphorylated tau protein, which is considered a key pathological factor in Alzheimer's disease, this technology has the potential to slow or prevent disease progression. Modalis will continue to advance the development of innovative therapy, MDL-104, that address critical unmet medical needs.

For more information, please contact:

Media Contact

Ms. Sawako Nakamura

sawako@modalistx.com

*Epigenome editing: The control of gene expression through modification of DNA, histones, non-coding RNA, etc., to turn genes on or off while leaving the gene sequence intact.

dCas9: An enzyme from which the cleavage activity of Cas9, a genome-editing cleavage enzyme, has been removed. It can be used for base substitution and epigenome editing by linking with transcription factors and other elements.