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October 20, 2026

Company name: Modalis Therapeutics Corporation
Stock exchange listing: Tokyo Stock Exchange Growth Market
Ticker/Code number: 4883
URL: <https://www.modalistx.com/en/>
Corporate Representative: Haruhiko Morita

MDL-101 Patent Application for LAMA2-RD Treatment Allowed in Japan

- The Japanese patent application “Method for Treating Muscular Dystrophy by Targeting LAMA1 Gene,” developed and filed by Modalis, has received a Notice of Allowance from the Japan Patent Office (JPO).
- The patent application is related to MDL-101, a therapeutic candidate for LAMA2-related muscular dystrophy (LAMA2-RD) that uses Modalis’ proprietary CRISPR-GNDM[®] epigenome editing technology.

Modalis Therapeutics Corporation (TSE: 4883; “Modalis”) announced today that it has received a Notice of Allowance from the Japan Patent Office (JPO) for a Japanese patent application (Japanese published application No. 2026-519313) related to MDL-101. The invention is entitled “Method for Treating Muscular Dystrophy by Targeting LAMA1 Gene.” Following completion of the required procedures, the application is expected to be granted as a Japanese patent.

The Japanese patent application was examined under the Japan Patent Office’s accelerated examination program, resulting in the present allowance. In addition, Modalis is proceeding with the examination process for the corresponding U.S. patent application through the Patent Prosecution Highway (PPH) program.

LAMA2-related muscular dystrophy (LAMA2-RD) is an inherited disorder caused by mutations in the LAMA2 gene, which encodes laminin $\alpha 2$, a component of the extracellular matrix surrounding muscle cells. Deficiency or impaired function of laminin $\alpha 2$ leads to various manifestations, including muscle weakness, hypotonia, joint contractures, and respiratory dysfunction.

MDL-101 is an investigational therapeutic candidate designed to selectively activate transcription of the LAMA1 gene, which is structurally and functionally similar to the LAMA2 gene, using Modalis’ proprietary CRISPR-GNDM[®] epigenome editing technology without cutting or altering the DNA sequence. By activating LAMA1, which is normally expressed at very low levels in mature skeletal

muscle, MDL-101 aims to induce expression of laminin $\alpha 1$ and compensate for the function of deficient or impaired laminin $\alpha 2$.

The LAMA1 and LAMA2 genes are too large to package in full into an adeno-associated virus (AAV) vector. Efficient gene delivery to skeletal muscles throughout the body also remains a challenge for nonviral delivery technologies. These limitations have posed technical challenges to developing LAMA2-RD treatments based on conventional gene replacement therapy. Rather than introducing a replacement gene, MDL-101 takes a differentiated approach using CRISPR-GNDM[®] to achieve sustained activation of endogenous LAMA1 gene expression. We believe MDL-101 has the potential to provide a new therapeutic option for patients with LAMA2-RD.

The allowed invention relates to methods of treating muscular dystrophy by targeting the LAMA1 gene and controlling its expression. We believe that the allowance of this invention in Japan strengthens the intellectual property foundation for our CRISPR-GNDM[®]-based approach to treating LAMA2-RD and represents an important step in protecting the potential future business value of MDL-101.

Guided by our corporate slogan, “Every life deserves attention,” Modalis is committed to advancing new treatments for patients living with diseases for which effective therapies remain limited or unavailable.

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