

This document has been translated from the Japanese original for reference purposes only.

In the event of any discrepancy between this translated document and the Japanese original, the original shall prevail. The Company assumes no responsibility for this translation or for direct, indirect or any other forms of damages arising from the translation.



September 10, 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

DMPK Patent Application for DM1 Treatment Allowed in the United States

- The U.S. patent application “Method for Treating Muscular Dystrophy by Targeting DMPK Gene,” developed and filed by Modalis, has received a Notice of Allowance from the United States Patent and Trademark Office (USPTO).
- The patent application is related to MDL-202, a therapeutic candidate for myotonic dystrophy type 1 (DM1) 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 United States Patent and Trademark Office (USPTO) for U.S. Patent Application No. 17/614,461, which is related to MDL-202. The invention is entitled “Method for Treating Muscular Dystrophy by Targeting DMPK Gene.” Following completion of the required procedures, the application is expected to issue as a U.S. patent.

Myotonic dystrophy type 1 (DM1) is an inherited disorder caused by an abnormal expansion of CTG repeats in the untranslated region of the DMPK gene. DMPK RNA containing the expanded repeat accumulates in cells and interferes with the function of RNA-binding proteins, leading to widespread abnormalities in RNA splicing. As a result, DM1 can affect multiple organs and systems, with manifestations including muscle weakness and myotonia as well as cardiac and respiratory complications.

MDL-202 is an investigational therapeutic candidate designed to selectively suppress transcription of the disease-causing DMPK gene using Modalis’ proprietary CRISPR-GNDM[®] epigenome editing technology, without cutting or altering the DNA sequence. In DM1, toxic RNA derived from DMPK plays a central role in disease pathogenesis. Rather than removing RNA after it has already been produced, MDL-202 is designed to intervene further upstream by suppressing its production at the transcriptional level.

Multiple therapeutic modalities targeting DMPK are currently being investigated, and we believe that therapeutic efficacy may depend not only on target selection but also on factors including delivery to relevant tissues, the magnitude of DMPK suppression, and the durability of that suppression. MDL-202 takes a differentiated approach by using CRISPR-GNDM[®] to achieve sustained control of gene expression itself, and we believe it has the potential to provide a new therapeutic option for patients with DM1.

The allowed claims cover methods of treating muscular dystrophy by targeting the DMPK gene and controlling its expression. We believe that the allowance of this invention in the United States strengthens the intellectual property foundation for our CRISPR-GNDM[®]-based approach to DM1 and represents an important step in protecting the potential future business value of MDL-202.

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.

End