Veritas In Silico

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The Jikei University School of Medicine and Veritas In Silico announce Patent Publication for Novel Nucleic Acid Therapeutic Targeting Amyotrophic Lateral Sclerosis (ALS)

The Jikei University School of Medicine, Research Center for Medical Science, Division of Regenerative Medicine (Minato-ku, Tokyo; Professor Doctor Hirotaka James OKANO M.D., PhD.,) and Veritas In Silico Inc. (Headquarters: Shinagawa-ku, Tokyo; Representative Director and CEO: Shingo Nakamura MBA PhD, hereinafter referred to as "VIS") are conducting joint research on developing a novel treatment for Amyotrophic Lateral Sclerosis (hereinafter referred to as "ALS"). A patent for new nucleic acid drug substances for ALS treatment originated from the Joint Research has now been published. Along with this announcement, an overview of the Joint Research is presented below.

The Joint Research has been initiated based on Professor Doctor Okano's discovery that the RNA/DNA-binding protein TDP-43, present within the cell nucleus, contains not only the “full-length TDP-43” that performs normal functions, but also several shorter splicing isoforms. This research revealed that ALS may be caused by a “functional decline of full-length TDP-43” involving “inhibitory truncated TDP-43,” specific generated from some of the shorter splicing isoforms of TDP-43. Professor Doctor Okano then conceived a therapeutic hypothesis: reducing the expression of the inhibitory truncated TDP-43 could restore the function of full-length TDP-43 impaired in ALS. Professor Doctor Okano published a paper on the function of TDP-43 splicing isoforms in October 2025. Concurrently, VIS designed and optimized nucleic acid drug candidates targeting two types of shorter isoforms generating inhibitory truncated TDP-43 using its AI-driven drug discovery platform, ibVIS®, and filed patents for nucleic acid drug substances that reduce their respective expression. As the examination process has now progressed, resulting in the publication of one of the patent applications.

The Joint Research reflects VIS's drug discovery support for Professor Doctor Okano's work, which holds the potential to pioneer new treatments for ALS, began even before the Joint Research was publicly disclosed. This initiative aims to accelerate drug discovery process for unmet medical needs by fostering early-stage collaboration between academia, which mainly aims to

elucidate disease mechanisms, and industry, which seeks to sufficiently enhance and optimize the performance of drugs used for treatment.

It is anticipated that this approach is expected to serve as a new third pathway for drug discovery from academia, complementing the existing methods of founding academia-spun-off ventures and transferring research to industry after its publication.

Please refer to the following pages for more details of the Joint Research and explanations.

東京慈恵会医科大学
THE JIKEI UNIVERSITY SCHOOL OF MEDICINE

Veritas In Silico

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- Patent Title: Nucleic Acid Therapeutics and Their Use
- Application Number: JP2025-575964
- International Patent Application Number: PCT/JP2025/025230
- Gene name: TDP-43 MP13
- Target disease: Amyotrophic Lateral Sclerosis (ALS)
- Novelty: First-in-class (no existing approved drug)
- Modality: Nucleic acid drug
- Target population: Approximately 300,000 worldwide, approximately 10,000 in Japan
- Revenue forecast in Japan: Estimate in progress
- Estimated development period: Estimate in progress (development schedule to be detailed)

VIS utilizes its proprietary AI-driven mRNA-targeting drug discovery platform, ibVIS®, to collaborate with partner pharmaceutical companies in creating small molecule drugs—the largest segment in the pharmaceutical market—through the AI-driven drug discovery. In parallel, VIS is advancing its own R&D in nucleic acid therapeutics, which are expected to show the strongest growth potential. This strategy reflects our growth vision: to pursue mRNA-targeted small molecule drugs while addressing rare diseases with unmet medical needs using nucleic acid therapeutics as the appropriate modality. It also represents our business strategy to further

differentiate VIS by leveraging the strength of our technology, which is applicable to drug discovery for both small molecule drugs and nucleic acid therapeutics.

ALS is a disease in which motor neurons in the brain and spinal cord to control muscles become impaired. This results in a gradual loss of movement in the muscles of the limbs, throat, tongue, and those necessary for breathing. This disease generally progresses rapidly, and many cases result in death within two to five years after onset. While ALS most commonly affects individuals in their 40s and older, with the highest number of patients in their 60s and 70s, cases of younger onset have also been reported. At the time of the survey, the global patient count was approximately 300,000, with about 10,000 in Japan. The male-to-female ratio among patients is approximately 1.3 to 1.5 times higher for males than for females. In most cases, ALS is not inherited. However, hereditary onset within families has been observed in approximately 5% of all cases. Furthermore, cases of frontotemporal dementia (FTD), which frequently co-occurs with ALS, share genetic, clinical, and pathological commonalities with ALS. It is believed that these conditions form a disease spectrum with shared etiology and pathophysiology.

Research has identified numerous ALS-causing genes, and pathological abnormalities in the RNA/DNA-binding protein TDP-43[*1], located in the cell nucleus, are detected in most ALS cases. Recent studies indicate that TDP-43 plays a significant role in the development and progression of ALS, suggesting that impaired TDP-43 function may contribute to the presence of "abnormal RNA" in ALS patients.

The Joint Research revealed that a specific splicing isoform of TDP-43 generates inhibitory truncated TDP-43, which induces number of "abnormal RNAs" that are only detected in the brains and spinal cords of ALS and FTD patients. These abnormal RNAs are produced as a result of the strong suppression of the function of full-length TDP-43. In light of these findings, we are proposing a novel therapeutic strategy for ALS. This strategy involves the reduction of inhibitory truncated TDP-43 expressed in the brain and spinal cord using nucleic acid therapeutics. The goal of this strategy is to restore full-length TDP-43 function.

● Outlines of the Patent and its Publication

As evidence suggests that inhibitory truncated TDP-43 may be involved in ALS onset, a therapeutic strategy is anticipated in which the splicing isoform generating inhibitory truncated TDP-43[*2] it is knocked down to reduce the inhibitory truncated TDP-43. The Joint Research findings were published in a research article in October 2025 (see the following section for research article details). Concurrently with the publication of the Joint Research article, VIS designed and optimized nucleic acid drug candidates to knock down two of the shorter splicing isoforms using AI-driven drug discovery. This work leveraged the AI-driven mRNA target drug discovery platform ibVIS®, generated by VIS, of which was developed using a multidisciplinary approach. VIS subsequently acquired the rights to both medicines from The Jikei University School of Medicine and became the sole patent applicant. As the examination process has now progressed, resulting in the publication of one of the patent applications.

● Title and Author of the Joint Research article

Title of Journal｜Journal of Cell Biology

Title of the Joint Research Article｜Dominant-negative isoform of TDP-43 is regulated by ALS-linked RNA-binding proteins

Author｜Minami Hasegawa-Ogawa, Asako Onda-Ohto, Takumasa Nakajo, Arisa Funabashi, Ayane Ohya, Ryota Yazaki, and Hirotaka James Okano

● Overview and Role Division of the Joint Research

The Jikei University School of Medicine has a long-standing commitment to ALS research, particularly from a medical standpoint. This includes analysis of human iPS cell models and pathological sections, yielding significant insights into the path mechanisms and causative genes of ALS. Since 2017, VIS has collaborated with The Jikei University School of Medicine to contribute to ALS therapy development. The collaboration has involved the design of nucleic acid therapeutics targeting genes predicted to be involved in the disease.

The Joint Research between the The Jikei University School of Medicine and VIS as a biotech company has been progressing since its inception. This approach, driven by the synergy between academia and biotech companies, is expected to enable effective and efficient drug discovery. Academia focuses on elucidating disease mechanisms and creating drugs, while biotech companies aim to enhance and optimize the performance of drugs for treatment.

In the Joint Research, The Jikei University School of Medicine will be responsible for evaluating drug efficacy using the relevant evaluation system and verifying treatment concepts using human iPS cells or the relevant animal models. VIS will be responsible for designing, optimizing and synthesizing drug candidate substances for proof-of-concept studies of the treatment concepts, as well as conducting in vitro evaluations[*3].

● Comments from Hirotaka James OKANO M.D., PhD., Division of Regenerative Medicine, Research Center for Medical Science, The Jikei University School of Medicine

The core strengths of The Jikei University School of Medicine lie in its ability to conduct clinical research, perform analyses using pathological sections, and develop treatments using human iPS cells for specific human diseases . We consider it highly significant that this research combines these strengths with VIS's drug discovery technology and capabilities, enabling us to advance drug discovery in parallel with the analysis of disease mechanisms. We will expedite our research to maximize this synergy and provide treatment options to patients suffering from intractable diseases as soon as possible.

● Comments from Tatsuya SASAKAWA, PhD., Executive Officer and General Manager of Research Strategy Division of VIS

In the Joint Research, we find it highly significant that our VIS technology is being applied to research on intractable diseases under the leadership of Dr. James Okano. We are also pleased with the steady progress of the research.

This research initiative represents a significant step forward in the field, aiming to rapidly develop nucleic acid therapeutics in parallel with disease mechanism research. It leverages the

profound clinical insights of The Jikei University School of Medicine, the pioneering research of Professor Doctor James Okano, who leads this collaborative effort, VIS's innovative methodologies, and the synergistic effects of AI-driven drug discovery utilizing the mRNA drug discovery platform ibVIS®.

We look forward to collaborating with The Jikei University School of Medicine and our team members to advance the practical application of nucleic acid drugs.

● Impact on Future Business Performance of VIS

The Joint Research expenses associated with the Joint Research will be respectively covered by The Jikei University School of Medicine and VIS, with no financial transactions planned between the parties. Expenses for the Joint Research are already included in the annual budget for FY2026, and it is anticipated that there will be no significant impact on the Business Results.

In case any matters requiring disclosure arise in the future, they will be promptly disclosed.

● Glossary for Reference

*1 TDP-43: TAR DNA-binding protein 43; normally present in the cell nucleus as an RNA/DNA-binding protein, it becomes abnormal in neurodegenerative diseases such as amyotrophic lateral sclerosis (ALS) and frontotemporal dementia (FTD).

*2 Splicing isoform: Multiple mRNA (messenger RNA) variants generating protein variants with different structures and functions. They are produced from a single gene through a mechanism called "alternative splicing."

*3 In vitro evaluation: An evaluation method conducted in artificial environments such as incubators or test tubes using cells or tissues.

For Further Information, Contact:

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