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NEWS RELEASE

SEAL Therapeutics updates on progress with SEAL technology at WMS conference, Co-Founder Dr. Reinhard wins WMS President's Award

Basel, Switzerland, October 16, 2022 – SEAL Therapeutics provides an update on its SEAL Technology at the 27th International Hybrid Annual Congress of the World Muscle Society (WMS) in Halifax, Canada.

The poster presentation entitled "Linker protein-mediated gene therapy ameliorates muscle and nerve pathology in mouse models for LAMA2-related congenital muscular dystrophy" by Co-founder and CSO Dr. Judith Reinhard receives the President's Award for best fundamental work.

"We are honored about the recognition of Dr. Reinhard's progress in developing this innovate gene therapy approach for LAMA2 MD by the WMS President's Award for best fundamental work", says **Prof. Markus Rüegg, Co-Founder and CEO of SEAL Therapeutics.** "Data shown demonstrate that AAV-based application of our linker proteins to LAMA2 MD mouse models significantly increases body- and muscle-weight, improves muscle histology and fiber size, reduces inflammation and improves muscle function. Together, our data provide a solid proof-of-principle for this gene therapy approach and we now aim to advance this technology towards clinical development."

About LAMA2 MD (Merosin-deficient congenital muscular dystrophy or MDC1A)

Congenital muscular dystrophies (CMDs) are a group of genetic muscle diseases with onset at birth or very early infancy, which cannot be treated. The more than 30 known forms of these neuromuscular diseases differ in the type of genetic defect and in the severity of disease progression. The muscles of the affected children progressively lose strength and degenerate over time. Progressive muscle weakness, joint contractures and respiratory insufficiency characterize most CMDs and patients often die before they reach adulthood.

Laminins are proteins of the extracellular matrix that are important in many tissues for the development, stability and survival of interacting cells. LAMA2-related muscular dystrophy (LAMA2 MD, also called MDC1A), is one of the most common forms of CMD. It is caused by mutations in the *LAMA2* gene encoding the α2 subunit of laminin-211, a protein that stabilizes muscle fibers. Children affected by LAMA2 MD usually suffer from poor muscle tone and strength already at birth, and are therefore called "floppy infants". Most of the affected children never learn to walk independently. The respiratory muscles are also weak and continue to degenerate, resulting in organ failure.

About the Simultaneous Expression of Artificial Linkers (SEAL) technology

The innovative gene therapy approach (called SEAL technology), developed by Prof. Markus Rüegg and Prof. Peter Yurchenco and their teams over the past 20 years, overcomes the lack of laminin-α2 in muscle tissue by providing molecular connections with other laminins and with the plasma membrane of the muscle fibers. Available data demonstrate that the simultaneous expression of two specifically designed linker proteins functionally corrects the primary pathology of laminin-α2 deficiency, leads to sustained improvement in muscle histology, increased muscle mass and strength, improved body weight, and results in a remarkable increase in life span compared to untreated animals [1-10].

About SEAL Therapeutics AG

SEAL Therapeutics AG, a spin-off of the Biozentrum of University of Basel, develops proprietary SEAL technology as potential gene therapy treatment of LAMA2-related muscular dystrophy (LAMA2 MD; also called MDC1A). The Company combines technology from the Biozentrum, University of Basel and Rutgers, The State University of New Jersey. SEAL Therapeutics intends to team-up with and support a qualified pharma partner with experience in advanced gene therapy technologies for clinical development and registration with the ultimate goal to make this innovative treatment approach available to LAMA2 MD patients and their families.

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