Dyne Therapeutics Presents New Preclinical Data for its Facioscapulohumeral Muscular Dystrophy Program During the FSHD Society International Research Congress

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- Leveraging the FORCE™ Platform, DYNE-302 Achieved Robust and Durable DUX4 Suppression and Functional Benefit in FSHD Preclinical Models
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WALTHAM, Mass., June 13, 2024 (GLOBE NEWSWIRE) -- Dyne Therapeutics, Inc. (Nasdaq: DYN), a clinical-stage muscle disease company focused on advancing innovative life-transforming therapeutics for people living with genetically driven diseases, today highlighted new preclinical data for DYNE-302, its product candidate for facioscapulohumeral muscular dystrophy (FSHD), that demonstrated robust and durable *DUX4* suppression and functional benefit. The data were presented during the 31st Annual FSHD Society International Research Congress, being held June 13-14, 2024, in Denver, Colorado.

"These encouraging data demonstrate that DYNE-302 exhibited prolonged activity in preclinical FSHD models, highlighting our innovative approach to targeting the genetic cause of this devastating and progressive muscle disease with no currently approved therapies," said Oxana Beskrovnaya, Ph.D., chief scientific officer of Dyne. "Additionally, the findings presented today build on the compelling clinical data to date from our DM1 and DMD programs, underscoring the modularity of the FORCE platform to conjugate different types of oligonucleotides to target the underlying disease mechanisms. Together these results reinforce our significant opportunity to advance a broad portfolio of therapeutic candidates for muscle diseases. We look forward to progressing DYNE-302 through IND/CTA-enabling studies."

FSHD is a severe muscle disorder resulting from aberrant expression of the *DUX4* gene leading to progressive wasting and skeletal muscle loss. Leveraging the FORCE platform, DYNE-302, consists of a fragment antibody (Fab) that binds to the transferrin receptor 1 (TfR1) which is highly expressed on muscle, conjugated to an siRNA designed to reduce *DUX4* expression.

Data presented during the congress were generated using an innovative hTfR1/iFLExD mouse model developed by Dyne that expresses the human transferrin 1 receptor (TfR1) and enables tunable DUX4 induction in skeletal muscle. In hTfR1/iFLExD mice, a single intravenous dose of DYNE-302 resulted in dose-dependent and robust reduction of the DUX4 transcriptome (D4T) that lasted up to three months, with benefit on muscle structure and function. DYNE-302 also demonstrated high *in vitro* potency in FSHD patient-derived myotubes.

Today's presentation entitled, "The FORCE™ platform achieves robust and durable *DUX4* suppression and functional benefit in FSHD mouse models" will be available in the Scientific Publications & Presentations section of Dyne's website following the session at https://www.dyne-tx.com/our-forcetm-publications/.

About Facioscapulohumeral Muscular Dystrophy (FSHD)

FSHD is a rare, progressive, genetic disease caused by a mutation in the *DUX4* gene, leading to skeletal muscle loss, muscle weakness and wasting. In healthy individuals, *DUX4*-driven gene expression is active for only a short time in early embryonic development. In individuals with FSHD, the *DUX4* gene remains "on" long after it is supposed to be silenced. This genetic mutation leads to surplus production of the DUX4 protein, which causes the gradual destruction of muscle cells throughout the body. People living with FSHD experience weakness in all major muscle groups, including the face, as well as joint and spinal abnormalities, and often limited mobility. An estimated 16,000-38,000 individuals in the United States and approximately 35,000 in Europe are affected by FSHD, but there are currently no approved therapies.

About the FORCE™ Platform

The proprietary FORCE™ platform drives Dyne's efforts to develop targeted, modern oligonucleotide therapeutics with the potential to be life-transforming for patients with serious muscle diseases. Dyne designed the FORCE platform using its deep knowledge of muscle biology and oligonucleotide therapeutics to overcome the current limitations in delivery to muscle tissue with the goal of stopping or reversing disease progression. The FORCE platform leverages the importance of transferrin receptor 1 (TfR1) in muscle biology as the foundation for its novel approach. TfR1, which is highly expressed on the surface of muscle cells, is required for iron transport into muscle cells. Dyne links therapeutic payloads to its TfR1-binding fragment antibody (Fab) to develop targeted therapeutics for muscle diseases.

About Dyne Therapeutics

Dyne Therapeutics is a clinical-stage muscle disease company focused on advancing innovative life-transforming therapeutics for people living with genetically driven diseases. With its proprietary FORCE™ platform, Dyne is developing modern oligonucleotide therapeutics that are designed to overcome limitations in delivery to muscle tissue. Dyne has a broad pipeline for serious muscle diseases, including clinical programs for myotonic dystrophy type 1 (DM1) and Duchenne muscular dystrophy (DMD) and a preclinical program for facioscapulohumeral muscular dystrophy (FSHD). For more information, please visit https://www.dyne-tx.com, and follow us on X, LinkedIn and Facebook.

Forward-Looking Statements

This press release contains forward-looking statements that involve substantial risks and uncertainties. All statements, other than statements of historical facts, contained in this press release, including statements regarding Dyne's strategy, future operations, prospects and plans, objectives of management, the potential of the FORCE platform, expectations regarding the initiation of additional preclinical studies or clinical trials of DYNE-302, expectations as to the relationship between data from the company's ongoing ACHIEVE clinical trial in DM1 and DELIVER clinical trial in DMD and existing or additional data for DYNE-302, and plans to provide future updates on pipeline programs, constitute forward-looking statements within the meaning of The Private Securities Litigation Reform Act of 1995. The words "anticipate," "believe," "continue," "could," "estimate," "expect," "intend,"

“may,” “might,” “objective,” “ongoing,” “plan,” “predict,” “project,” “potential,” “should,” or “would,” or the negative of these terms, or other comparable terminology are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Dyne may not actually achieve the plans, intentions or expectations disclosed in these forward-looking statements, and you should not place undue reliance on these forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in these forward-looking statements as a result of various important factors, including: uncertainties inherent in the identification and development of product candidates, including the initiation and completion of preclinical studies and clinical trials; uncertainties as to the availability and timing of results from preclinical studies and clinical trials; the timing of and Dyne’s ability to enroll patients in clinical trials; whether results from preclinical studies and initial data from early clinical trials will be predictive of the final results of the clinical trials or future trials; whether Dyne’s cash resources will be sufficient to fund its foreseeable and unforeseeable operating expenses and capital expenditure requirements; as well as the risks and uncertainties identified in Dyne’s filings with the Securities and Exchange Commission (SEC), including the Company’s most recent Form 10-Q and in subsequent filings Dyne may make with the SEC. In addition, the forward-looking statements included in this press release represent Dyne’s views as of the date of this press release. Dyne anticipates that subsequent events and developments will cause its views to change. However, while Dyne may elect to update these forward-looking statements at some point in the future, it specifically disclaims any obligation to do so. These forward-looking statements should not be relied upon as representing Dyne’s views as of any date subsequent to the date of this press release.

Contacts:

Investors
Amy Reilly
areilly@dyne-tx.com
857-341-1203

Media
Stacy Nartker
snartker@dyne-tx.com
781-317-1938