Keros Therapeutics Presents Additional Clinical Data from its KER-065 Program at the American Society of Bone and Mineral Research 2025 Annual Meeting

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LEXINGTON, Mass., Sept. 08, 2025 (GLOBE NEWSWIRE) -- Keros Therapeutics, Inc. (“Keros”) (Nasdaq: KROS), a clinical-stage biopharmaceutical company focused on developing and commercializing novel therapeutics to treat a wide range of patients with disorders that are linked to dysfunctional signaling of the transforming growth factor-beta (“TGF-ß”) family of proteins, today announced that it presented additional clinical data from its Phase 1 clinical trial of KER-065 in healthy male volunteers at the American Society of Bone and Mineral Research 2025 Annual Meeting on Saturday, September 6, 2025.

“We are pleased to present additional data that highlights the broad therapeutic potential of KER-065, including its robust bone anabolic activity,” said Jasbir S. Seehra, President and Chief Executive Officer. “These data continue to demonstrate the potential of KER-065 in Duchenne muscular dystrophy, where bone loss occurs as a result of the progressive muscle weakness and chronic use of corticosteroids.”

Clinical Presentation

Phase 1 Trial in Healthy Participants of KER-065, a Modified Activin Receptor Ligand Trap, Supports Development in Duchenne Muscular Dystrophy and Bone Disorders

This Phase 1 clinical trial was a randomized, double-blind, placebo-controlled, two-part dose escalation (single and multiple ascending dose) trial in healthy male volunteers. The primary objectives of this trial were to assess safety, tolerability and pharmacokinetics of KER-065. Exploratory endpoints include assessments of the pharmacodynamic effect on bone, adipose, muscle, cardiac tissue and fibrosis. Initial topline data from this trial was reported in March 2025.

As of the final data cut-off date of April 29, 2025, treatment with KER-065 was generally well-tolerated at all dose levels tested. No dose-limiting toxicities or serious adverse events were reported. One grade 4 treatment-emergent adverse event of elevated creatine kinase levels was observed, but deemed unrelated to treatment. The majority of the adverse events that were observed were mild to moderate in severity and resolved.

Additional results from this trial, as of the data cut-off date, demonstrated that treatment with KER-065 resulted in:

- Changes in bone biomarkers of increased bone formation and reduced bone resorption that were consistent with tissue level changes, as demonstrated by observed increases in bone mineral density (“BMD”)
- Whole body BMD improvements at Day 85 that were sustained through Day 141 (three months after the last dose in the multiple ascending dose cohorts), suggesting a balance between osteoblast and osteoclast activity
- Increased lumbar spine BMD following three doses that were sustained through Day 141

About KER-065

KER-065 is a novel ligand trap comprised of a modified ligand-binding domain derived from activin receptor type IIA and activin receptor type IIB that is fused to the portion of the human antibody known as the Fc domain. KER-065 is designed to act as a ligand trap and inhibit the biological effects of myostatin and activin A, two ligands that signal through activin receptors, to increase skeletal muscle regeneration, increase muscle size and strength, reduce body fat, reduce fibrosis of the skeletal muscle and increase bone strength. We are developing KER-065 for the treatment of neuromuscular diseases, with an initial focus on DMD.

About Duchenne Muscular Dystrophy (DMD)

DMD is the most common form of muscular dystrophy and results in muscle degeneration and premature death. DMD results from the lack of functional dystrophin protein that helps promote myofiber stability, caused by a gene mutation. The lack of dystrophin, an important structural component of muscle cells, causes muscle cells to have increased susceptibility to damage and to progressively die. Additionally, the absence of dystrophin in muscle cells leads to significant cell damage and ultimately causes muscle cell death and the replacement of muscle with fibrotic and fatty tissue. The replacement of muscle fibers with fatty and fibrotic tissue leads to progressive loss of muscle strength and function leading to immobility and respiratory and cardiac complications. In DMD patients, heart muscle cells progressively die and are replaced with scar tissue. This cardiomyopathy eventually leads to heart failure, which is currently the leading cause of death among those with DMD. The National Organization for Rare Disorders estimates that approximately one in every 3,500 male births is affected by DMD worldwide.

About Keros Therapeutics, Inc.

Keros is a clinical-stage biopharmaceutical company focused on developing and commercializing novel therapeutics to treat a wide range of patients with disorders that are linked to dysfunctional signaling of the TGF-ß family of proteins. Keros is a leader in understanding the role of the TGF-ß family of proteins, which are master regulators of the growth, repair and maintenance of a number of tissues, including blood, bone, skeletal muscle, adipose and heart tissue. By leveraging this understanding, Keros has discovered and is developing protein therapeutics that have the potential to provide meaningful and potentially disease-modifying benefit to patients. Keros’ lead product candidate, KER-065, is being developed for the treatment of neuromuscular diseases, with an initial focus on DMD. Keros’ most advanced product candidate, elritercept, is being developed for the treatment of cytopenias, including anemia and thrombocytopenia, in patients with myelodysplastic syndrome and in patients with myelofibrosis.

Cautionary Note Regarding Forward-Looking Statements

Statements contained in this press release regarding matters that are not historical facts are “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. Words such as “anticipates,” “believes,” “continue,” “expects,” “enable,” “intention,” “potential” and “will” or similar expressions are intended to identify forward-looking statements. Examples of these forward-looking statements include statements concerning: Keros’ expectations regarding its strategy, progress and timing of its clinical trials for KER-065; and the broad therapeutic potential of KER-065. Because such statements are subject to risks and uncertainties, actual results may differ materially from those expressed or implied by such forward-looking statements. These risks and uncertainties include, among others: Keros’ limited operating history and historical losses; Keros’ ability to raise additional funding to complete the development and any commercialization of its product candidates; Keros’ dependence on the success of its product candidates, KER-065 and elritercept; that Keros may be delayed in initiating, enrolling or completing any clinical trials; competition from third parties that are developing products for similar uses; Keros’ ability to obtain, maintain and protect its intellectual property; and Keros’ dependence on third parties in connection with manufacturing, clinical trials and preclinical studies.

These and other risks are described more fully in Keros’ filings with the Securities and Exchange Commission (the “SEC”), including the “Risk Factors” section of the Company’s Quarterly Report on Form 10-Q, filed with the SEC on August 6, 2025, and its other documents subsequently filed with or furnished to the SEC. All forward-looking statements contained in this press release speak only as of the date on which they were made. Except to the extent required by law, Keros undertakes no obligation to update such statements to reflect events that occur or circumstances that exist after the date on which they were made.

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