UNITED STATES SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): November 7, 2022

Keros Therapeutics, Inc.

(Exact name of registrant as specified in its charter)

Delaware (state or other jurisdiction of incorporation) 001-39264 (Commission File Number) 81-1173868 (I.R.S. Employer Identification No.)

99 Hayden Avenue, Suite 120, Building E
Lexington, Massachusetts
(Address of principal executive offices)

02421 (Zip Code)

Registrant's telephone number, including area code: (617) 314-6297

Not applicable

(Former name or former address, if changed since last report.)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:				
	Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)			
	Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)			

Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

\square Pre-commencement communications pursuant to Rule 1	3e-4(c) under the Exchange Act (17	CFR 240.13e-4(c))
Securities re	egistered pursuant to Section 12(b) c	of the Act:
Title of each class	Trading Symbol	Name of each exchange on which registered
Common Stock, \$0.0001 par value per share	KROS	The Nasdaq Stock Market LLC
Indicate by check mark whether the registrant is an emerging chapter) or Rule 12b-2 of the Securities Exchange Act of 193 Emerging growth company ⊠ If an emerging growth company, indicate by check mark if the or revised financial accounting standards provided pursuant to	4 (§240.12b-2 of this chapter). e registrant has elected not to use th	e extended transition period for complying with any new

Item 8.01 Other Events.

On November 7, 2022, Keros Therapeutics, Inc. (the "Company") issued a press release announcing results from a preclinical study of a research form of KER-012 on cardiac and pulmonary pathology in an established rat model of pulmonary arterial hypertension, which were presented at the American Heart Association 2022 Scientific Sessions on Monday, November 7, 2022.

A copy of the press release is attached as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description		
99.1	Press release dated November 7, 2022.		
104	Cover Page Interactive Data File (the cover page XBRL tags are embedded within the Inline XBRL document)		

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

KEROS THERAPEUTICS, INC.

By: /s/ Jasbir Seehra

Jasbir Seehra, Ph.D. Chief Executive Officer

Dated: November 7, 2022

Keros Therapeutics Presents Preclinical Data from its KER-012 Program at the American Heart Association 2022 Scientific Sessions

November 7, 2022, 4:01 PM EST

LEXINGTON, Mass., Nov. 7, 2022 (GLOBE NEWSWIRE) -- Keros Therapeutics, Inc. ("Keros") (Nasdaq: KROS), a clinical-stage biopharmaceutical company focused on the discovery, development and commercialization of novel treatments for patients suffering from hematological, pulmonary and cardiovascular disorders with high unmet medical need, today announced results from a preclinical study of a research form of KER-012 ("RKER-012") on cardiac and pulmonary pathology in an established rat model of pulmonary arterial hypertension ("PAH"), which were presented at the American Heart Association 2022 Scientific Sessions on Monday, November 7, 2022.

"We are pleased to have reported encouraging preclinical data from our KER-012 program showing that treatment with RKER-012 led to improvement in cardiac and pulmonary function in a rat model of PAH. This cardiopulmonary improvement was associated with reductions in inflammatory and fibrotic markers, including $TGF\beta$ -1, which is the most potent driver of fibrosis in the transforming growth factor-beta ("TGF- β ") family of ligands," said Jasbir S. Seehra, Ph.D., President and Chief Executive Officer of Keros. "We believe these data further validate that KER-012 has the potential to treat not only PAH but also other inflammatory and fibrotic diseases of the heart and the lungs."

RKER-012 improved cardiopulmonary function in a rat PAH model.

• Improvement in Cardiopulmonary Function in a Rat Model of Pulmonary Arterial Hypertension Observed with RKER-012, a Novel Activin Receptor Type II Ligand Trap, was Associated with Reduced Markers of Inflammation and Fibrosis in the Right Ventricle.

Keros combined administration of SUGEN5416, a tyrosine kinase inhibitor of vascular endothelial growth factor receptors 1/2, with exposure to chronic hypoxia to induce PAH in adult rats. RKER-012 was tested in this SUGEN/hypoxia ("SH") rat PAH model. Adult rats subjected to SH received either vehicle or 10 mg/kg of RKER-012 twice weekly for three weeks. Rats maintained under normal oxygen conditions ("normoxic controls") received only vehicle.

- Consistent with the development of cardiac and pulmonary impairment, vehicle-treated SH rats exhibited increases in Fulton index (p≤0.0001), which measures enlargement of the right ventricle ("RV") and systolic pulmonary arterial pressure ("sPAP") (p≤0.0001) relative to normoxic controls. In the SH rats, treatment with RKER-012 significantly attenuated increased Fulton index (p≤0.001) and prevented an increase in sPAP (p≤0.0001) compared to treatment with vehicle
- The RV of vehicle-treated SH rats exhibited significant increases in the gene expression of several known drivers of
 inflammation compared to normoxic controls. Treatment with RKER-012 prevented the increase in these markers in the
 RV of SH rats.
- Expression of fibrosis biomarkers were elevated in the RV of vehicle-treated SH rats compared to normoxic controls.
 Relative to vehicle-treated SH rats, RKER-012-treated SH rats had significantly reduced expression of most of those fibrosis biomarkers.

About KER-012

KER-012 is designed to bind to and inhibit the signaling of TGF-β ligands that stimulate smooth muscle hypertrophy and fibrosis, including activin A, activin B and myostatin (GDF8). Keros believes that KER-012 has the potential to increase the signaling of bone morphogenic protein ("BMP") pathways through this inhibition of activin A and activin B signaling, and consequently treat diseases such as PAH

that are associated with reduced BMP signaling. KER-012 is being developed for the treatment of PAH and for the treatment of cardiovascular disorders associated with cardiac hypertrophy.

About Keros Therapeutics, Inc.

Keros is a clinical-stage biopharmaceutical company focused on the discovery, development and commercialization of novel treatments for patients suffering from hematological, pulmonary and cardiovascular disorders with high unmet medical need. Keros is a leader in understanding the role of the TGF-β family of proteins, which are master regulators of red blood cell and platelet production as well as of the growth, repair and maintenance of a number of tissues, including blood vessels and heart tissue. Keros' lead protein therapeutic product candidate, KER-050, is being developed for the treatment of low blood cell counts, or cytopenias, including anemia and thrombocytopenia, in patients with MDS and in patients with myelofibrosis. Keros' lead small molecule product candidate, KER-047, is being developed for the treatment of functional iron deficiency. Keros' third product candidate, KER-012, is being developed for the treatment of pulmonary arterial hypertension and for the treatment of cardiovascular disorders associated with cardiac hypertrophy.

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 "believes," "further" and "potential," or similar expressions are intended to identify forward-looking statements. Examples of these forward-looking statements include statements concerning: Keros' expectations regarding its growth, strategy, progress and the design, objectives and timing of its clinical trials for KER-012; and the potential of KER-012 to treat PAH and other inflammatory and fibrotic diseases of the heart and the lungs. 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 lead product candidates, KER-050 and KER-047; 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; Keros' dependence on third parties in connection with manufacturing, clinical trials and pre-clinical studies; and risks relating to the impact on Keros' business of the COVID-19 pandemic or similar public health crises.

These and other risks are described more fully in Keros' filings with the Securities and Exchange Commission ("SEC"), including the "Risk Factors" section of the Company's Quarterly Report on Form 10-Q, filed with the SEC on November 3, 2022, 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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