Kymera Therapeutics Presents New Preclinical Lupus Data for KT-579, First-in-Class, Oral IRF5 Degrader, at EULAR and FOCIS Congresses

June 8, 2026

KT-579 demonstrated consistent disease-modifying activity comparable or superior to approved and clinically active therapies in multiple preclinical lupus models

KT-579 Phase 1 healthy volunteer trial ongoing, with data expected in 2H26

WATERTOWN, Mass., June 08, 2026 (GLOBE NEWSWIRE) -- Kymera Therapeutics, Inc. (NASDAQ: KYMR), a clinical-stage biopharmaceutical company advancing a new class of oral small molecule degrader medicines for immunological diseases, today announced the presentation of new preclinical data for KT-579, its potent, selective, oral IRF5 degrader, demonstrating disease-modifying activity in lupus models. The findings show that by selectively targeting and degrading IRF5, KT-579 offers a novel oral approach for complex, heterogeneous autoimmune diseases driven by multiple validated inflammatory pathways, including Type I interferons, pro-inflammatory cytokines and autoantibody responses. These data were presented at the European Alliance of Associations for Rheumatology (EULAR) Annual Meeting held June 3-6, 2026, in London, UK, and will be presented at the Federation of Clinical Immunology Societies (FOCIS) Annual Meeting being held June 9-12, 2026, in San Francisco, CA.

“Lupus remains a complex and heterogeneous autoimmune disease, with many patients continuing to experience inadequate disease control despite available therapies,” said Juliet Williams, PhD, Head of Research, Kymera Therapeutics. “The data presented at these key medical meetings reinforce KT-579’s potential as a novel oral approach to modulating multiple disease-driving pathways implicated in lupus, including Type I IFN, pro-inflammatory cytokine and B cell-driven responses. The consistent activity observed across patient-derived cells and multiple preclinical lupus models provides strong support for IRF5 degradation as a promising strategy to broadly address the underlying disease biology with a convenient oral medicine.”

The Company previously reported data demonstrating KT-579’s compelling profile in preclinical studies using human primary cell systems, patient-derived cells and *in vivo* disease models of lupus, rheumatoid arthritis, and inflammatory bowel disease showing activity comparable or superior to existing standards of care.

New data presented at EULAR and FOCIS further validate KT-579’s broad and consistent activity across preclinical models of lupus. In healthy donor and lupus patient-derived human cells, with or without a common SLE-associated polymorphism, KT-579 selectively degraded IRF5, blocked TLR-induced IRF5 nuclear translocation, and reduced key downstream inflammatory mediators, including Type I IFN and pro-inflammatory cytokines, as well as plasmablast differentiation and IgG production. *In vivo*, KT-579 demonstrated dose-dependent IRF5 degradation and inhibition of TLR7- and TLR9-induced cytokine release, including TNFα, IL-6, IL-12 and IFNβ. Across multiple lupus models spanning low to high Type I IFN signaling, KT-579 treatment led to reductions in disease-relevant biomarkers, including proteinuria, serum autoantibodies and kidney pathology, with activity comparable or superior to approved and clinically active agents tested. Together, these findings demonstrate consistent modulation of pro-inflammatory, Type I IFN and B cell-driven pathways, supporting KT-579’s potential as a first-in-class, oral approach in lupus and other chronic autoimmune diseases.

The KT-579 Phase 1 healthy volunteer trial is ongoing. The Phase 1 study is evaluating the safety, tolerability, pharmacokinetics and pharmacodynamics of single- and multiple-ascending doses of orally administered KT-579 compared to placebo. The key study aim is to show that KT-579 can robustly degrade IRF5 in blood at doses that are safe and well tolerated. The functional impact of IRF5 degradation on the induction of Type I interferons, pro-inflammatory cytokines, and inflammatory pathway gene transcripts will also be assessed with whole blood ex vivo stimulation assays. The Company expects to report data from the trial in the second half of 2026 and, following the healthy volunteer study, plans to initiate a patient proof-of-concept trial, likely in lupus.

European Alliance of Associations for Rheumatology (EULAR)

- **Title**: First-in-class Oral IRF5 Degrader, KT-579, Demonstrates Selective and Potent In Vitro and In Vivo Activity in Human Cellular Assays and Mouse Models of Lupus
- **Presenter:** Veronica Campbell, Senior Director, Immunology, Kymera Therapeutics
- **Type/Session:** Poster, Poster View VIII
- **Date/Time:** Saturday, June 6, 2026, at 10:15 AM BST

Federation of Clinical Immunology Societies (FOCIS)

- **Title**: Potent and Selective First-in-Class Oral IRF5 Degrader, KT-579, Inhibits Endosomal TLR-Induced Responses in SLE Derived PBMCs and Significantly Reduces Disease Activity in the MRL.lpr Mouse Lupus Model
- **Presenter:** Erik Corcoran, Principal Scientist, Kymera Therapeutics
- **Type/Session:** Poster, Autoimmune Diseases
- **Date/Time:** Thursday, June 11, 2026, at 7:00 PM PT

Copies of the EULAR and FOCIS posters will be available in the Resource Library section of Kymera's website.

About KT-579
KT-579 is an investigational, first-in-class, oral degrader of IRF5, a genetically validated transcription factor and master regulator of immunity, and currently in Phase 1 testing. By selectively degrading IRF5, KT-579 is designed to modulate multiple disease-driving pathways simultaneously, including Type I interferons, pro-inflammatory cytokines and autoantibody responses, offering the potential for biologics-like activity in a convenient oral medicine. In preclinical studies, KT-579 degraded IRF5 across multiple preclinical species and in all disease-relevant tissues. In preclinical models of lupus, rheumatoid arthritis (RA), and inflammatory bowel disease (IBD), KT-579 activity was equal to or more efficacious than small molecule

inhibitors and biologics currently marketed or in the clinic. In preclinical safety studies, KT-579 did not show any adverse effects of any type at all doses tested. KT-579 has the potential to be the first novel mechanism with broad utility in diseases where effective and well tolerated oral therapies are needed, such as lupus, IBD, RA, Sjögren's and others.

About Kymera Therapeutics
Kymera is a clinical-stage biotechnology company pioneering the field of targeted protein degradation (TPD) to develop medicines that address critical health problems and have the potential to dramatically improve patients' lives. Kymera is deploying TPD to address disease targets and pathways inaccessible with conventional therapeutics. Having advanced the first degrader into the clinic for immunological diseases, Kymera is focused on building an industry-leading pipeline of oral small molecule degraders to provide a new generation of convenient, highly effective therapies for patients with these conditions. Founded in 2016, Kymera has been recognized as one of Boston's top workplaces for the past several years. For more information about our science, pipeline and people, please visit www.kymeratx.com or follow us on X or LinkedIn.

Cautionary Note Regarding Forward-Looking Statements
This press release contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, as amended, including, without limitation, implied and express statements about our expectations regarding strategy, business plans and objectives on the development of our clinical and preclinical pipeline, including the therapeutic potential, clinical benefits and safety thereof, including for KT-579, the Phase 1 healthy volunteer data readout of KT-579 in the second half of 2026. The words "may," "might," "will," "could," "would," "should," "expect," "plan," "anticipate," "intend," "believe," "expect," "estimate," "seek," "predict," "future," "project," "potential," "continue," "target," "upcoming" and similar words or expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Any forward-looking statements in this press release are based on management's current expectations and beliefs and are subject to a number of risks, uncertainties and important factors that may cause actual events or results to differ materially from any forward-looking statements contained in this press release, including, without limitation, risks associated with: uncertainties inherent in the initiation, timing and design of future clinical trials, the availability and timing of data from ongoing and future trials and the results of such trials, whether preclinical results will be indicative of the results of clinical trials, the ability to successfully demonstrate the safety and efficacy of drug candidates, the timing and outcome of planned interactions with regulatory authorities, the availability of funding sufficient for our operating expenses and capital expenditure requirements and other factors. These risks and uncertainties are described in greater detail in the section entitled "Risk Factors" in the most recent Quarterly Report on Form 10-Q and in subsequent filings with the SEC. In addition, any forward-looking statements represent our views only as of today and should not be relied upon as representing our views as of any subsequent date. We explicitly disclaim any obligation to update any forward-looking statements. No representations or warranties (expressed or implied) are made about the accuracy of any such forward-looking statements.

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