Kymera Therapeutics Presents New Preclinical IBD Data for KT-579, a First-in-Class, Oral IRF5 Degrader, at Digestive Disease Week

May 5, 2026

KT-579 demonstrated activity comparable or superior to approved and clinically active therapies in preclinical IBD models

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

WATERTOWN, Mass., May 05, 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 an inflammatory bowel disease (IBD) model. 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 Digestive Disease Week (DDW) being held May 2-5, 2026, in Chicago, IL.

“Despite advances, many patients with chronic inflammatory diseases like IBD still cycle through therapies or remain inadequately controlled, in part because existing treatments don’t fully address the complexity of their disease or the realities of long-term management,” said Juliet Williams, PhD, Head of Research, Kymera Therapeutics. “KT-579 has the potential to deliver a novel oral mechanism that can modulate multiple disease-driving pathways simultaneously. The consistent activity we’ve observed across these pathways in preclinical models provides strong validation of this approach and its opportunity to more fully address disease biology. We believe this could translate into a meaningful new option for millions of patients in need of effective, safe, and convenient oral therapies.”

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 and rheumatoid arthritis, showing activity comparable or superior to existing standards of care. The new data presented at DDW further demonstrate KT-579’s consistent modulation of pro-inflammatory pathways. KT-579 showed impact on myeloid cell effector function, including potent inhibition of cytokines known to amplify inflammatory responses and promote Th1 and Th17 T cell activity in IBD.

In the TNBS model of IBD, KT-579 demonstrated activity comparable or superior to clinically relevant comparators, including a JAK inhibitor and biologic agents targeting integrins and TNF. Prophylactic dosing of KT-579 led to a significant reduction in disease activity score, including protection from body weight loss and maintenance of colon density. KT-579 demonstrated complete inhibition of key colon inflammatory cytokines, including TNFα, IL-1β and IL-6, and reduced pathological findings, including fewer inflammatory infiltrates, improved crypt structure integrity and absence of crypt abscesses. Transcriptomic analysis further demonstrated broad normalization of inflammatory, myeloid, and fibrosis-associated pathways, showing effects comparable to an anti-TNF agent. Collectively, these findings position KT-579 as a novel oral approach capable of broadly modulating pathogenic pathways in IBD and other complex autoimmune diseases, where current treatment options remain limited.

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.

Digestive Disease Week (DDW) 2026

- **Title**: Potent and Selective Oral IRF5 Degrader, KT-579, Demonstrates Robust Anti-inflammatory Activity and Disease Modulation in Preclinical In Vivo Models of Inflammatory Bowel Disease
- **Presenter:** Ryan Camire, PhD, Scientist, Immunology, Kymera Therapeutics
- **Type/Session:** Poster, Mechanisms of IBD Therapeutics
- **Date/Time:** Tuesday, May 5, 2026, at 12:30pm CT

A copy of the poster is 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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