Epirium Bio Presenting New Preclinical Data on its 15-PGDH Inhibitor Program in Inflammatory Bowel Disease at Digestive Disease Week® 2026

15-PGDH inhibition with Epirium's oral small molecules, a novel therapeutic approach in inflammatory bowel disease, reduces endoscopic disease severity, lowers inflammatory cytokines, and improves histologic scores in preclinical IBD models

San Diego, CA, May 5, 2026. Epirium Bio Inc. (Epirium), a clinical-stage biopharmaceutical company advancing medicines for neuromuscular, inflammatory, and fibrotic diseases, is presenting new translational data on its oral 15-PGDH inhibitor program in inflammatory bowel disease (IBD), including data on MF-300 and MF-1305 at Digestive Disease Week® (DDW) 2026, which is taking place May 2-5 in Chicago, IL. In mouse models of colitis, both compounds elicited significant improvements in markers of disease severity. MF-300, Epirium's Phase 2 ready compound, has demonstrated significant endoscopic improvement indicative of mucosal healing at all doses based on an initial efficacy readout, with additional data forthcoming. Separately, MF-1305 demonstrated improvement in disease activity index (DAI) scores, reduced key inflammatory cytokine levels in the colon, and improved histological scores.

Prostaglandin E2 (PGE2) is a key regulator of gastrointestinal homeostasis that promotes resolution of inflammation, supports epithelial barrier integrity, and drives mucosal repair, all processes that are impaired in IBD. 15-PGDH, the primary enzyme that degrades PGE2, is upregulated in the inflamed mucosa of patients with IBD, and PGE-Major Urinary Metabolite (PGE-MUM), a marker of systemic PGE2 turnover, has been shown to correlate with disease activity in both ulcerative colitis (UC) and Crohn's disease (CD). Published studies report approximately 30–40% higher PGE-MUM levels in active versus inactive UC and approximately 60% higher levels in active versus inactive CD. By inhibiting 15-PGDH, MF-300 and MF-1305 are oral therapies intended to restore physiological PGE2 signaling and promote mucosal healing through a mechanism that is distinct from currently available immunosuppressive and anti-inflammatory therapies for IBD.

"In order to maximize our treatment efficacy in IBD, we continue to need treatment options with novel mechanisms of action to be used alone or in combination with our current therapies," said Matthew Hamilton, M.D., Assistant Professor of Medicine at Harvard Medical School and gastroenterologist at Mass General Brigham. "What is compelling about 15-PGDH inhibition is that it aims to harness the body's capacity to

repair the mucosa, a mechanism that is distinct from existing therapies. The preclinical activity of MF-300 and MF-1305, combined with MF-300's Phase 1 pharmacodynamic effect on PGE-MUM, supports continued evaluation of this exciting approach in IBD."

Preclinical Study Details

Separate preclinical studies evaluated the respective effects of MF-300 and MF-1305 on disease severity markers in dextran sulfate sodium (DSS) mouse models of colitis, with initial findings reported for MF-300 and a more comprehensive dataset available for MF-1305.

In the MF-300 study, colitis was induced in male C57Bl/6 mice by 3% DSS administration for 5 days, followed by 14 days of daily MF-300 treatment at 2 clinically relevant doses (N=12 per dosage group), after which endoscopy was performed.

In the MF-1305 study, colitis was induced in female C57Bl/6NTac mice by 3.5% DSS administration for 8 days. MF-1305 was administered daily at 3 doses (N=10 per dosage group) beginning on day 3 and continuing for a further 3 days after DSS administration ended (total MF-1305 treatment duration = 9 days). Animals were then assessed for DAI scores, colon cytokine levels, and histological disease.

Key findings included:

- All doses of MF-300 significantly improved endoscopic measures of colitis severity, matching the response observed with the anti-p40 antibody positive control, a class of biologics used clinically in IBD that targets IL-12/23 signaling.

- All doses of MF-1305 improved DAI scores, comparable to the cyclosporine positive control, an established immunosuppressant used in IBD. MF-1305 also reduced inflammatory cytokines and improved histology scores, even exceeding the effects of cyclosporine in some endpoints.

- Collectively, these results support advancing 15-PGDH inhibitors as a novel oral therapeutic approach to promote mucosal healing in patients with IBD.

Details of the poster presentation include:

Title: Novel oral inhibitors of 15-PGDH improve disease outcomes in DSS mouse models of colitis

Session Title: Mechanisms of IBD Therapeutics

Presenter: Micah T. Webster, Ph.D.

Poster Number: Tu1476

The poster will be available in the “Posters and Publications” section of Epirium’s website, www.epirium.com.

About 15-PGDH Inhibition

15-hydroxyprostaglandin dehydrogenase (15-PGDH) is the main enzyme responsible for the degradation of prostaglandin E2 (PGE2), a key endogenous regulator of resolution of inflammation, and tissue repair. Across diseases characterized by prolonged or chronic inflammation and impaired tissue repair, including the gastrointestinal mucosa of patients with IBD as well as aged skeletal muscle in sarcopenia, 15-PGDH is transcriptionally upregulated. Pharmacologic inhibition of 15-PGDH is intended to restore physiological PGE2 levels for therapeutic benefits by promoting resolution of inflammation and tissue repair simultaneously.

About MF-300

MF-300 is an investigational, first-in-class, orally administered, small molecule inhibitor of 15-hydroxyprostaglandin dehydrogenase (15-PGDH). In a Phase 1 healthy volunteer study, MF-300 reduced prostaglandin E2 major urinary metabolite (PGE-MUM), a urinary marker of prostaglandin E2 (PGE2) turnover, providing pharmacodynamic evidence of 15-PGDH target engagement at clinically relevant doses. MF-300 is advancing into Phase 2b development in sarcopenia (age-related muscle weakness) and is also being evaluated preclinically in inflammatory bowel disease.

About Inflammatory Bowel Disease (IBD)

Inflammatory bowel disease (IBD) is a group of chronic, immune-mediated inflammatory disorders of the gastrointestinal tract, including ulcerative colitis and Crohn's disease. The U.S. Centers for Disease Control and Prevention estimates that up to 3 million Americans are living with IBD, which is characterized by an unpredictable, relapsing course of abdominal pain, diarrhea, bleeding, and urgency, and can lead to hospitalization, surgery, and serious long-term complications. Despite the growing availability of treatment options, relapse rates remain high, necessitating the development of novel agents to support mucosal healing.

About Epirium Bio

Epirium Bio is a clinical-stage biopharmaceutical company based in San Diego, California, developing an IP-protected platform of orally bioavailable small molecule inhibitors of 15-hydroxyprostaglandin dehydrogenase (15-PGDH). Epirium is leveraging 15-PGDH inhibition to address diseases affected by prolonged or chronic inflammation and impaired tissue repair, where elevated 15-PGDH activity blocks the proresolution

of inflammation and tissue repair pathways downstream of PGE2 signaling. This common mechanism is shared across distinct therapeutic indications, including neuromuscular diseases such as sarcopenia and spinal muscular atrophy, fibrotic diseases such as idiopathic pulmonary fibrosis, and inflammatory bowel disease. The Company's lead program, MF-300, is advancing into Phase 2b development for sarcopenia, with additional programs in earlier stages of development.

To learn more about Epirium, please visit www.epirium.com and follow us on LinkedIn.

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