MetaVia Presents Preclinical Data Supporting the Potential of DA-1726 to Address Liver Disease and Weight Loss at ObesityWeek® 2026

November 16, 2026

Statistically Significant Increase in Liver and Circulating FGF21 Expression

Greater Glucose Lowering and Comparable Weight Loss vs. Retatrutide with Less Reliance on Appetite Suppression

MetaVia Preparing to Initiate a Phase 2 Study in MetALD and ALD in the Second Half of 2027

CAMBRIDGE, Mass., Nov. 16, 2026 /PRNewswire/ -- **MetaVia Inc.** (Nasdaq: MTVA), a clinical-stage biotechnology company focused on transforming liver-related cardiometabolic diseases, today announced positive preclinical data from a head-to-head study comparing DA-1726, a novel GLP-1 (GLP1R)/glucagon (GCGR) dual-receptor agonist, with retatrutide, a triple GLP-1R/GIPR/GCGR agonist. The findings, presented on November 14, 2026, at ObesityWeek® 2026 in Washington, D.C., demonstrated that DA-1726 increased hepatic fibroblast growth factor 21 (*Fgf21)* gene expression and liver FGF21 protein levels, along with enhanced expression of genes associated with thermogenesis and energy expenditure. DA-1726 also demonstrated greater glucose lowering and achieved weight loss comparable to retatrutide with less suppression of food intake.

"Given our focus on liver-related metabolic disease, we are particularly encouraged that DA-1726 increased both gene expression and protein levels of hepatic *Fgf21*, a key hormone in the regulation of lipid and carbohydrate metabolism," said Hyung Heon Kim, President and Chief Executive Officer of MetaVia. "Greater glucose lowering and weight loss comparable to retatrutide, despite less suppression of food intake, further support DA-1726's differentiated metabolic profile. Together with our recent clinical data demonstrating substantial reductions in liver fat and body weight, these findings strengthen the rationale for advancing DA-1726 into a Phase 2 study in MetALD and ALD, targeted to begin in the second half of 2027."

The study compared DA-1726 to retatrutide in diet-induced obese (DIO) mice, assessing body weight, food intake, body composition, plasma metabolic parameters and thermogenesis-related gene expression. Animals received twice-weekly subcutaneous injections of DA-1726 at 300 or 500 nmol/kg or retatrutide at 100 or 200 nmol/kg for approximately three weeks. The higher doses were selected based on pharmacokinetic exposure estimates intended to approximate clinical exposures at 48 mg for DA-1726 and 12 mg for retatrutide.

DA-1726 demonstrated a statistically significant increase in hepatic expression of *Fgf21* ($p<0.05$), showing both increased liver FGF21 protein and circulating FGF21 ($p<0.001$) at the higher dose. DA-1726 also induced greater expression of genes associated with brown adipose tissue thermogenesis and white adipose tissue browning compared with retatrutide. At the higher dose, expression of thermogenesis-related genes, including *Ucp1*, *Ppargc1a* and *Fgf21*, was increased in brown and inguinal white adipose tissue, with several markers demonstrating significantly greater expression than observed with high-dose retatrutide. These findings suggest that glucagon receptor-associated metabolic pathways, including those involving FGF21 and thermogenesis, may contribute to DA-1726's weight-loss effects.

DA-1726 also demonstrated greater glucose lowering compared with retatrutide, along with favorable effects on lipid metabolism. Plasma glucose levels decreased by 43.0% and 39.2% for DA-1726, versus reductions of 21.5% and 32.5% for retatrutide, at the low and high doses for each compound, respectively. Both showed improvements in plasma lipid parameters, with DA-1726 reducing total cholesterol by 70.9% and 69.3%, compared with 60.9% and 69.0% for retatrutide, and LDL cholesterol by 89.9% and 88.4%, compared with 82.8% and 88.1% for retatrutide, respectively. Comparable or numerically greater improvements in glucose and lipid parameters were consistently observed with DA-1726.

In addition, DA-1726 achieved weight loss similar to retatrutide despite less suppression of food intake. After adjustment relative to DIO controls, DA-1726 achieved body weight reductions of 34.2% and 41.7% compared with reductions of 33.7% and 43.6% for retatrutide, at the low and high doses for each compound, respectively. However, DA-1726 reduced food intake by 19.6% and 31.2%, compared with 36.7% and 41.4% for retatrutide, respectively. Both treatments also demonstrated comparable effects on body composition, including substantial reductions in fat mass. These findings suggest a potentially greater contribution of GCGR-associated energy expenditure to weight loss with DA-1726 than with retatrutide.

Taken together, these findings highlight DA-1726's differentiated metabolic profile, demonstrating weight loss comparable to retatrutide with less suppression of food intake, along with increased hepatic *Fgf21* expression and protein levels and greater

expression of genes associated with thermogenesis. The results suggest that GCGR-associated energy expenditure may play a greater role in DA-1726's weight-loss effects, providing additional preclinical support for its potential in liver-related cardiometabolic diseases.

Presentation Details:

- **Title:** *DA-1726 Demonstrates Comparable Weight Loss and Distinct Metabolic Profile Compared With Retatrutide*
- **Session:** Gut Peptides, Appetite Signaling & Novel Therapeutics
- **Presenting Author:** Il Hoon Jeong, MS, Research Scientist, Dong-A ST Research Center
- **Poster Number:** Poster-229

A copy of the poster is available on the Posters section of the MetaVia website.

About DA-1726
DA-1726 is a novel GLP1R/GCGR dual agonist being developed to address various cardiometabolic indications, with potential in both Metabolic and Alcohol-Associated Liver Disease (MetALD) and Alcohol-Associated Liver Disease (ALD), which is designed for once weekly, subcutaneous administration. DA-1726 acts as a dual agonist of GLP-1 receptors (GLP1R) and glucagon receptors (GCGR), leading to weight loss through reduced appetite and increased energy expenditure. It is engineered with a 3:1 GLP1R:GCGR potency ratio intended to balance the complementary effects of GLP1R activation on appetite and glucose control with GCGR activation on energy expenditure and fat breakdown. DA-1726 has a well understood mechanism and preclinical tissue-distribution studies have shown that it concentrates most heavily in adipose tissue and in the liver. In a Phase 1 Part 3B interim analysis at Day 109, DA-1726 demonstrated a 13.5% placebo-adjusted reduction in body weight and a 68.0% placebo-adjusted reduction in liver fat, along with reductions in visceral and subcutaneous adipose tissue and relatively preserved lean mass. MetaVia is planning a Phase 2, 48-week, non-biopsy study of DA-1726 in MetALD and ALD, targeted to begin in the second half of 2027.

About MetaVia
MetaVia Inc. is a clinical-stage biotechnology company focused on transforming liver-related cardiometabolic diseases. The company is currently developing DA-1726 for the treatment of various cardiometabolic conditions, with potential in both Metabolic and Alcohol-Associated Liver Disease (MetALD) and Alcohol-Associated Liver Disease (ALD), and is advancing vanoglipel (DA-1241) in combination with resmetirom for the treatment of Metabolic Dysfunction-Associated Steatohepatitis (MASH). DA-1726 is a novel oxyntomodulin (OXM) analogue that functions as a glucagon-like peptide-1 receptor (GLP1R) and glucagon receptor (GCGR) dual agonist, engineered with a 3:1 GLP1R:GCGR potency ratio. OXM is a naturally-occurring gut hormone that activates GLP1R and GCGR, thereby decreasing food intake while increasing energy expenditure. In a Phase 1 Part 3B interim analysis at Day 109, DA-1726 demonstrated placebo-adjusted reductions of 13.5% in body weight and 68.0% in liver fat, along with reductions in visceral and subcutaneous adipose tissue and relatively preserved lean mass. A Phase 2, 48-week, non-biopsy study of DA-1726 in MetALD and ALD, is targeted to begin in the second half of 2027. Vanoglipel is a potential first-in-class drug candidate targeting G-protein-coupled receptor 119 (GPR119). It acts directly in the liver on pathways involved in steatosis, inflammation and fibrosis, while GPR119 activation in the pancreas and gut also supports glucose and metabolic regulation. In a Phase 2a clinical study, vanoglipel demonstrated direct hepatic action in addition to its glucose lowering effects. MetaVia is advancing vanoglipel in combination with resmetirom as a once-daily oral combination therapy for MASH, with a Phase 2a proof-of-concept study targeted to begin in the first half of 2027.

For more information, please visit www.metaviatx.com.

Forward Looking Statements
Certain statements in this press release may be considered forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Words such as "believes", "expects", "anticipates", "may", "will", "should", "seeks", "approximately", "potential", "intends", "projects", "plans", "estimates" or the negative of these words or other comparable terminology (as well as other words or expressions referencing future events, conditions or circumstances) are intended to identify forward-looking statements. Forward-looking statements are predictions, projections and other statements about future events that are based on current expectations and assumptions and, as a result, are subject to risks and uncertainties. Many factors could cause actual future events to differ materially from the forward-looking statements in this press release, including, without limitation, those risks associated with MetaVia's history of net losses, the sufficiency of its existing cash on hand to fund operations and raising additional capital; adverse global economic conditions; MetaVia's ability to execute on its commercial strategy; the timeline for regulatory submissions; the ability to obtain regulatory approval through the development steps of MetaVia's current and future product candidates; the ability to realize the benefits of the license agreement with Dong-A ST Co. Ltd., including the impact on future financial and operating results of MetaVia; the cooperation of MetaVia's contract manufacturers, clinical study partners and others involved in the development of MetaVia's current and future product candidates; potential negative interactions between MetaVia's product candidates and any other products with which they are combined for treatment; MetaVia's ability obtain, maintain, protect and/or enforce sufficient intellectual property rights to execute its commercial strategy; MetaVia's ability to initiate and complete clinical trials on a timely basis; MetaVia's ability to recruit subjects for its clinical trials; whether MetaVia receives results from MetaVia's clinical trials that are consistent with the results of preclinical and previous clinical trials; impact of costs related to the license agreement, known and unknown, including costs of any litigation or regulatory actions relating to the license agreement; the effects of changes in applicable laws, regulations or Nasdaq listing rules; the effects of changes to MetaVia's stock price; and other risks and uncertainties described in MetaVia's filings with the Securities and Exchange Commission, including MetaVia's most recent Annual Report on Form 10-K. Forward-looking statements speak only as

of the date when made. MetaVia does not assume any obligation to publicly update or revise any forward-looking statements, whether as a result of new information, future events or otherwise, except as required by law.

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