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 5, 2026

METAVIA INC.
(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-37809**	**47-2389984**
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

545 Concord Avenue, Suite 210 Cambridge, Massachusetts	**02138**
(Address of principal executive offices)	(Zip Code)

(857) 702-9600
(Registrant's telephone number, including area code)

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))
☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, par value $0.001 per share	MTVA	The Nasdaq Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01. Regulation FD Disclosures.

On November 5, 2026, MetaVia Inc. (the ***"Company"***) issued a press release announcing positive interim data from Part 3B of its Phase 1 clinical trial of DA-1726, a novel oxyntomodulin (OXM) analog targeting both GLP-1 (GLP1R) and glucagon (GCGR) receptors. A copy of the press release is furnished herewith as Exhibit 99.1 to this Current Report on Form 8-K (this ***"Report"***) and is incorporated herein by reference.

The information in Item 7.01 of this Report, including Exhibit 99.1 attached hereto, is furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the ***"Exchange Act"***), or otherwise subject to the liabilities of that Section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as expressly set forth by specific reference in such filing. The Company's submission of this Report shall not be deemed an admission as to the materiality of any information required to be disclosed solely to satisfy the requirements of Regulation FD.

Item 8.01. Other Events.

On November 5, 2026, the Company posted an updated corporate presentation to its website at https://ir.metaviatx.com/events-presentations/presentations, which the Company may use from time to time in connection with presentations, investor communications or conferences. A copy of the corporate presentation is attached as Exhibit 99.2 to this Report and is incorporated herein by reference.

Information contained on or accessible through any website reference in the press release or the corporate presentation is not part of, or incorporated by reference in, this Report, and the inclusion of such website addresses in this Report by incorporation by reference of the press release and the corporate presentation are as inactive textual references only.

Forward-Looking Statements

This Report, including Exhibit 99.1 and Exhibit 99.2 attached hereto, contains forward-looking statements within the meaning of the federal securities laws. These forward-looking statements are based on current expectations and are not guarantees of future performance. Further, the forward-looking statements are subject to the limitations listed in Exhibit 99.1 and Exhibit 99.2 and in the other reports that the Company has filed with the Securities and Exchange Commission, including that actual events or results may differ materially from those in the forward-looking statements.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number	Exhibit Description
99.1	Press Release dated November 5, 2026.
99.2	Corporate Presentation dated November 2026.
104	Cover Page Interactive Data File (embedded within 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.

METAVIA INC.

Date: November 5, 2026

By: /s/ Hyung Heon Kim
Hyung Heon Kim
President and Chief Executive Officer

MetaVia Announces Statistically Significant 68% Reduction in Liver Fat from DA-1726 Phase 1 Part 3B Interim Data

DA-1726 Also Demonstrated Statistically Significant 13.5% Placebo-Adjusted Weight Loss and Reduced Mean HbA1c from 5.79% to 5.33% in Participants Without Diabetes

No New Gastrointestinal Adverse Events Were Reported After Week 9 Through Day 109, with No Gastrointestinal Adverse Events Leading to Study Discontinuation

Full Part 3A and Part 3B Data Expected in the First Quarter of 2027

Results Support 48-Week, Non-Biopsy Phase 2 Study of DA-1726 in Up to 150 Patients with MetALD and ALD, Targeted to Begin in the Second Half of 2027

CAMBRIDGE, Mass., November 5, 2026 – MetaVia Inc. (Nasdaq: MTVA), a clinical-stage biotechnology company focused on transforming liver-related cardiometabolic diseases, today announced positive interim data from Part 3B of its Phase 1 clinical trial of DA-1726, a novel oxyntomodulin (OXM) analog targeting both GLP-1 (GLP1R) and glucagon (GCGR) receptors. The results, presented in an oral presentation at the Precision Liver Symposium on November 4, 2026, in Denver, Colorado, demonstrated a 68% placebo-adjusted reduction in liver fat and 13.5% placebo-adjusted body weight reduction following 16 weeks of once-weekly DA-1726 treatment in otherwise healthy adults with obesity.

At Day 109, 10 of 12 DA-1726-treated participants (83%) in the MRI substudy achieved a relative liver fat reduction of at least 30%, compared with none on placebo. All DA-1726-treated participants with baseline liver steatosis achieved MRI-PDFF below 5%. DA-1726 also lowered HbA1c from 5.79% at baseline to 5.33% at Day 109, a 0.24 percentage-point placebo-adjusted reduction in participants who were not diabetic at entry, with significant reductions in adipose tissue and relative preservation of lean tissue and muscle volume.

“These are the most compelling clinical data we have generated to date supporting DA-1726’s potential as a liver-focused cardiometabolic therapy,” said Hyung Heon Kim, President and Chief Executive Officer of MetaVia. “A 68% placebo-adjusted reduction in liver fat after approximately 16 weeks, together with all five participants who entered the study with liver steatosis achieving MRI-PDFF below 5%, provides a strong clinical rationale for expanding DA-1726 into liver disease. Importantly, these liver findings were accompanied by 13.5% placebo-adjusted weight loss, improvements in HbA1c, and significant reductions across visceral and subcutaneous adipose tissue, while lean tissue and muscle volume were relatively preserved. ”

Mr. Kim continued, “The next step for DA-1726 is clear. We intend to build on these results by advancing the program into both metabolic dysfunction and alcohol-associated liver disease (MetALD) and alcohol-associated liver disease (ALD), where there are currently no approved therapies specifically for these patient populations. MetALD is defined by a steatotic liver together with at least one cardiometabolic risk factor, and in 16 weeks DA-1726 moved all three with statistical significance: liver fat, body weight and HbA1c. MetaVia estimates that 10.7 million U.S. adults have MetALD. DA-1726’s dual GLP-1 and glucagon receptor mechanism, its concentration in liver and adipose tissue observed in preclinical studies, and the

significant liver fat reduction now demonstrated clinically, provide a strong biological and clinical foundation for this next stage of development. We expect to report full data from Part 3A and Part 3B in the first quarter of 2027 and are planning a 48-week, non-biopsy Phase 2 study in up to 150 subjects with MetALD and ALD, targeted to begin in the second half of 2027."

DA-1726 Part 3B Interim Results

Endpoint, Day 109 (16 weeks)	Baseline, DA-1726 arm	Change from baseline, DA-1726	Placebo-adjusted	p-value vs. placebo
Liver fat (MRI-PDFF), n=12 vs. 4	5.2%	All 5 participants with baseline steatosis (≥5%) fell below 5%	−68% (GMR 0.32)	<0.001
Body weight, n=15 vs. 4	98.6 kg (217 lbs)	−12.19 kg (−26.9 lbs; −11.9%)	−13.5%	<0.001
HbA1c, n=15 vs. 4	5.79%	−0.44 percentage points (to 5.33%)	−0.24 percentage points	0.006
Waist circumference, n=15 vs. 4	107.5 cm (42.3 in)	−9.6 cm (−3.8 in; −9.0%)	−9.1%	<0.001

DA-1726 64 mg target dose versus placebo. Placebo-adjusted body weight, waist circumference and HbA1c are differences in mean change from baseline versus placebo; liver fat is the geometric mean ratio (GMR) versus placebo in the MRI substudy.

Liver fat: The MRI substudy included 12 DA-1726-treated participants and four placebo participants. Mean baseline liver fat, as measured by MRI-PDFF, was 5.2% in DA-1726-treated participants and 8.0% in the placebo group. At Day 109, DA-1726 achieved a 68% placebo-adjusted reduction in liver fat (GMR 0.32; p<0.001). The absolute MRI-PDFF treatment difference versus placebo was −6.4 percentage points (p=0.007). 10 of 12 DA-1726-treated participants (83%) achieved a relative reduction in liver fat of at least 30%, compared with none of the four placebo participants (p=0.008).

Among the five DA-1726-treated participants with liver steatosis at baseline, defined as MRI-PDFF ≥5%, all five achieved MRI-PDFF below 5% by Day 109. Mean MRI-PDFF in these participants declined from 8.3% at baseline to 3.0% at Day 109, representing a 5.3 percentage-point reduction (p=0.005). The participant with the highest baseline MRI-PDFF experienced a reduction from 12.8% to 4.6%. In comparison, liver fat in the placebo group increased 40.4% from its own baseline.

Glycemic control: Mean baseline HbA1c was 5.79% in the DA-1726 arm and 5.65% in the placebo arm, with all participants in the non-diabetic range at entry. At Day 109, mean HbA1c in the DA-1726 arm fell from 5.79% to 5.33%, a 0.44 percentage-point reduction from baseline (p<0.001), and every DA-1726-treated participant with a Day 109 value showed a reduction of 0.3 to 0.8 percentage points. The placebo arm declined by 0.20 percentage points from its 5.65% baseline. The difference in mean change versus placebo was −0.24 percentage points (95% CI −0.41 to −0.07; p=0.006).

Body composition: MRI-based body composition analysis demonstrated significant reductions across measured adipose tissue compartments. At Day 109, visceral adipose tissue (VAT) was 22% lower versus placebo (GMR 0.78; p<0.001), abdominal subcutaneous adipose tissue (ASAT) was 19% lower versus placebo (GMR 0.81; p=0.002), and total adipose tissue (TAT) was 19% lower versus placebo (GMR 0.81; p<0.001).

Lean tissue and muscle volume were relatively preserved, with thigh muscle volume 9% lower versus placebo (GMR 0.91; p=0.018) and total lean tissue 8% lower versus placebo (GMR 0.92; p=0.007), compared with reductions of 19% to 22% across the measured adipose tissue compartments.

Safety and tolerability: No participant discontinued the study because of a gastrointestinal adverse event, and no new gastrointestinal adverse events were reported after Week 9, or Day 63, through the Day 109 interim assessment. Gastrointestinal adverse events were reported in nine of 15 DA-1726-treated participants, with most events mild to moderate in severity. Vomiting was reported in seven participants (46.7%), nausea in two participants (13.3%), diarrhea in two participants (13.3%), dyspepsia in two participants (13.3%), constipation in one participant (6.7%) and abdominal pain in one participant (6.7%).

One serious adverse event of severe vomiting was reported and considered possibly related to treatment. The participant recovered.

Precision Liver Symposium Presentation Details:

- **Title:** *DA-1726, a Dual GLP-1 and Glucagon Receptor Agonist, Leads to Significant Reduction in MRI-PDFF and Weight Loss with Relative Preservation of Lean Mass: A Randomized Placebo-Controlled Trial*
- **Presenting Author:** Sarah Browne, M.D., Chief Medical Officer of MetaVia

MetALD KOL Dinner at AASLD's The Liver Meeting 2026

MetaVia will host an invitation-only KOL dinner this evening during the American Association for the Study of Liver Diseases' (AASLD) The Liver Meeting 2026, focusing on MetALD and the evolving treatment landscape.

About the DA-1726 Phase 1 Part 3B Study
Part 3B of the Phase 1 trial of DA-1726 is a 16-week, randomized, placebo-controlled study in otherwise healthy adults with obesity. Participants receiving DA-1726 were titrated from 16 mg for 4 weeks, to 32 mg for 4 weeks, to the target dose of 64 mg, administered once weekly by subcutaneous injection. The interim analysis includes 15 participants treated with DA-1726 and four treated with placebo; an MRI substudy of 12 DA-1726-treated and four placebo participants assessed liver fat by MRI-PDFF and body composition.

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, a 68% placebo-adjusted reduction in liver fat and a 0.24 percentage-point placebo-adjusted reduction in HbA1c, 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, 68% in liver fat and 0.24 percentage points in HbA1c, 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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MetaVia Inc.

Transforming Cardiometabolic Diseases

November 2026

Nasdaq: MTVA

Disclaimers

This presentation (this "Presentation") includes forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995. Forward-looking statements include all statements th relate solely to historical or current facts and can be identified by the use of words such as "believes", "expects", "anticipates", "may", "will", "should", "seeks", "approximately", "intends", "projects" "estimates" or the negative of these words or other comparable terminology (as well as other words or expressions referencing future events, conditions or circumstances). Forward-looking stater 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. These forward-looking st include, but are not limited to, statements regarding the market size and potential growth opportunities of our current product candidates; the safety, efficacy, tolerability and other potential benefits, such loss, associated with our current product candidates; the competitive differentiators of our current product candidates; potential collaborations and/or licensing partners for our current product candi planned clinical trial activities for our current product candidates; the expected timeline for topline data release dates; and our planned submissions of investigational new drug applications and meetings wit Food and Drug Administration. Many factors could cause actual future events to differ materially from the forward-looking statements in this Presentation, including, without limitation, those risks associate ability to execute our commercial strategy; the sufficiency of our existing cash on hand to fund our operations and raising additional capital; adverse global economic conditions; our ability to execu commercial strategy; the timeline for regulatory submissions, regulatory steps and potential regulatory approval of our current and future product candidates; the ability to realize the benefits of t agreement with Dong-A ST Co. Ltd. (the "License Agreement"), including the impact on our future financial and operating results; the ability to integrate our current and future product candidates into our b a timely and cost-efficient manner; the cooperation of our contract manufacturers, clinical study partners and others involved in the development of our current and future product candidates; potentia interactions between our product candidates and other products for which they are combined for treatment; and our ability to initiate and complete clinical trials on a timely and in a cost-efficient ma planned clinical trials and our ability to recruit subjects for our clinical trials; the costs related to the License Agreement, known and unknown, including costs of any litigation or regulatory actions relat License Agreement; effects of the changes in applicable laws or regulations; the effects of changes to our stock price on the terms of the License Agreement and any future fundraising; and our ability maintain, protect and/or enforce sufficient intellectual property rights. Additional risks and uncertainties that could affect our financial results and business are more fully described in our Annual Report on f for the year ended December 31, 2025 ("2025 Form 10-K"), filed with the Securities and Exchange Commission ("SEC") on March 26, 2026, and in our other SEC filings. These forward-looking statements are information currently available to us and our current plans or expectations and are subject to a number of known and unknown uncertainties, risks and other important factors that may cause our actu performance or achievements expressed or implied by the forward-looking statements. These and other important factors are described in detail in the "Risk Factors" section of our 2025 Form 10-K, and filings with the SEC. Although we believe the expectations reflected in such forward-looking statements are reasonable, we can give no assurance that such expectations will prove to be correct. These forwa statements should not be relied upon as representing our views as of any date subsequent to this Presentation.

Disclaimers

No head-to-head study has been conducted between DA-1726 or vanoglipel and any product or product candidate shown in this Presentation. Data for products other than DA-1726 and vanoglipel are publicly available third-party sources, including company disclosures and peer-reviewed publications, and have not been generated or verified by the Company. Trials differ in patient population, dose, measurement method, and statistical approach (placebo-adjusted vs. own-arm change; MRI vs. DXA). Because of these differences, cross-trial comparisons should be interpreted with caution, and no co regarding the relative safety or effectiveness of DA-1726 or vanoglipel should be drawn from any comparisons presented in this Presentation.

This Presentation also may contain estimates and other statistical data made by independent parties and by us relating to market size and other data about our industry. This data involves a number of ass and limitations, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions and estimates of our future performance and the future performance of the market we operate are necessarily subject to a high degree of uncertainty and risk.

This Presentation speaks only as of the date of the information contained herein and the Company does not have any obligation to update or correct any information herein.

Investment Highlights

Two clinical-stage liver-metabolic assets with five catalysts over the next fifteen months

TWO ASSETS, ONE LIVER STRATEGY

MetaVia is now a liver-focused cardiometabolic company: DA-1726 toward MetALD / ALD, vanoglipel + resmetirom toward MASH — one disease continuum, two entry points.

DA-1726 — PART 3B INTERIM, DAY 109

−68%

liver fat, placebo-adjusted

−13.5% placebo-adjusted body weight and −0.24 pp HbA1c vs. placebo, lean mass relatively preserved; no new GI adverse events after Week 9.

DA-1726 — NEXT INDICATION

10.7M

US adults with MetALD, no approved therapy

A 3:1 GLP1R/GCGR agonist acting on hepatic fat and adipose. Phase 2 in MetALD / ALD targeted to start 2H 2027.

VANOGLIPEL — PHASE 2a, N=109

−22.8 U/L

ALT at Week 16 vs −6.7 on placebo

Liver fat and inflammation markers improved; weight-neutral; adverse events at placebo-like rates in the 108 treated, none leading to discontinuation.

VANOGLIPEL + RESMETIROM

2 species

synergy shown in mice and monkeys

Oral, once-daily fixed-dose combination MASH: better NAS than either drug alon weight loss only in combination. Phase 2 start 1H 2027.

FIVE CATALYSTS IN FIFTEEN MONTHS

H2 2026	Nov 2026	Q1 2027	1H 2027	2H 2027
FDA meeting on the combination data	Part 3B interim results — oral presentation, Precision Liver Symposium; Obesity Week poster	DA-1726 Part 3A / 3B topline	Combination Phase 2a in MASH — start	DA-1726 Phase 2 in MetALD / ALD

NASDAQ	MTVA	CASH, JUNE 30, 2026	$12.6M · no debt	LARGEST HOLDER	Dong-A ST, 26.8%	HEADQUARTERS	Massachusetts, U

DA-1726: Part 3B interim, n=15 active / 4 placebo. Vanoglipel: NCT06054815. Combination: MetaVia nonclinical studies in DIO-MASH mice and obese cynomolgus monkeys. Milestones assume regulatory and clinical success, which is not guaranteed.

MetaVia

A Liver-Focused Cardiometabolic Company

Two assets, one disease continuum — open white space at the MetALD / ALD end; a validated path and a $1B-a-year incumbent at the MASH end

VANOGLIPEL + RESMETIROM — oral, once-daily fixed-dose combination for MASH

DA-1726 — liver focused, MetALD / ALD

MASLD	MASH	MetALD	ALD
metabolic drivers, little or no alcohol	*steatohepatitis with fibrosis — 2 approved drugs*	*metabolic drivers plus moderate alcohol — 0 approved*	*alcohol-related liver disease — 0 approved*

MetALD / ALD — WHERE DA-1726 IS HEADED: NO ONE IS THERE

10.7M
US adults with MetALD
4.1% of the adult population

2.5×
prevalence rise since 1988–90
1.6% → 4.1%, tracking obesity, diabetes, alcohol

0
approved treatments
no dedicated Phase 3 program in the category

45% higher risk of premature death than abstainers without steatotic liver disease (adjusted HR 1.45). Glucagon-receptor agonism acts on hepatic fat and adipose — the tissues where DA-1726 concentrates (−68% liver fat at Day 109 in obesity).

MASH — WHERE THE COMBINATION COMPETES: THE PATH IS PROVEN

$364M
Rezdiffra net sales, Q2 2026
+71% YoY · ~$1.3B trailing twelve months

50,000+
patients on Rezdiffra
as of July 2026, more than double the prior year

2
approved MASH drugs
resmetirom (Mar 2024) · semaglutide (Aug 2025)

The incumbent leaves two gaps — most patients do not resolve NASH, and it is weight-neutral — and i itself building a combination. An oral, once-daily FDC that adds both builds on an established prescribe and payer base.

NEXT CLINICAL TRIALS

DA-1726 — Phase 2 in MetALD / ALD — Start 2H 2027

48 weeks	up to 150 subjects	non-biopsy	obese adults with MetALD / ALD

First dedicated trial in the category; non-invasive liver and metabolic endpoints, design to be finalized with FDA.

Vanoglipel + resmetirom — Phase 2a in MASH — Start 1H

12 weeks	~100 patients	non-biopsy	presumed MASH population

Randomized proof-of-concept, co-administration; modeled on the vanoglipel Phase 2a (NCT06054815).

MetALD: Younossi et al., Lancet Gastroenterol Hepatol 2026; Paik et al., J Clin Gastroenterol 2025. Rezdiffra: Madrigal Q2 2026 results. Approvals: REZDIFFRA USPI (2024); semaglutide MASH indication (2025). Milestones assume regulatory and clinical success, which is not guaran

MetaVia

MetaVia Inc. — DA-1726 & vanoglipel programs, November 2026

Leadership Team

EXECUTIVE LEADERSHIP



Hyung Heon Kim

Chief Executive Officer

- 20+ years in M&A, financing and corporate governance; 10+ years of licensing and M&A with Dong-A Group
- Former General Counsel and SVP, Dong-A ST and Dong-A Socio Group
- JD, Washington University School of Law



Sarah Browne, MD

Chief Medical Officer

- 20+ years in clinical development and regulatory science, specializing in liver and metabolic diseases, including MASH
- Former VP, Clinical Development at Altimmune; led pemvidutide through Phase 2 and Phase 3 planning
- Former FDA and NIH; MD, Virginia Commonwealth University



Mi-Kyung Kim, Ph.D., RPh

Chief Scientific Officer

- 25+ years in drug discovery research at Dong-A ST
- Diabetes, obesity, MASH and immune-mediated disease
- Ph.D., RPh, College of Pharmacy, Ewha Womans University



Marshall H. Woodworth

Chief Financial Officer

- 35+ years of financial experience; 2
  years working with life-science investors and analysts
- CFO of Nevakar, Braeburn Pharmaceuticals, Aerocrine and Fur
  Pharmaceuticals
- MBA, Indiana University

Biographies as of November 2026.

DA-1726 — Part 3B Interim Results at 16 Weeks

A once-weekly 3:1 GLP1R/GCGR dual agonist for obesity, with potential in MetALD/ALD. Day 109 interim: −68% placebo-adjusted liver fat, −13.5% placebo-adjusted body weight and −0.24 pp HbA1c vs. placebo, lean mass relatively preserved — and no new GI adverse events after Week 9.

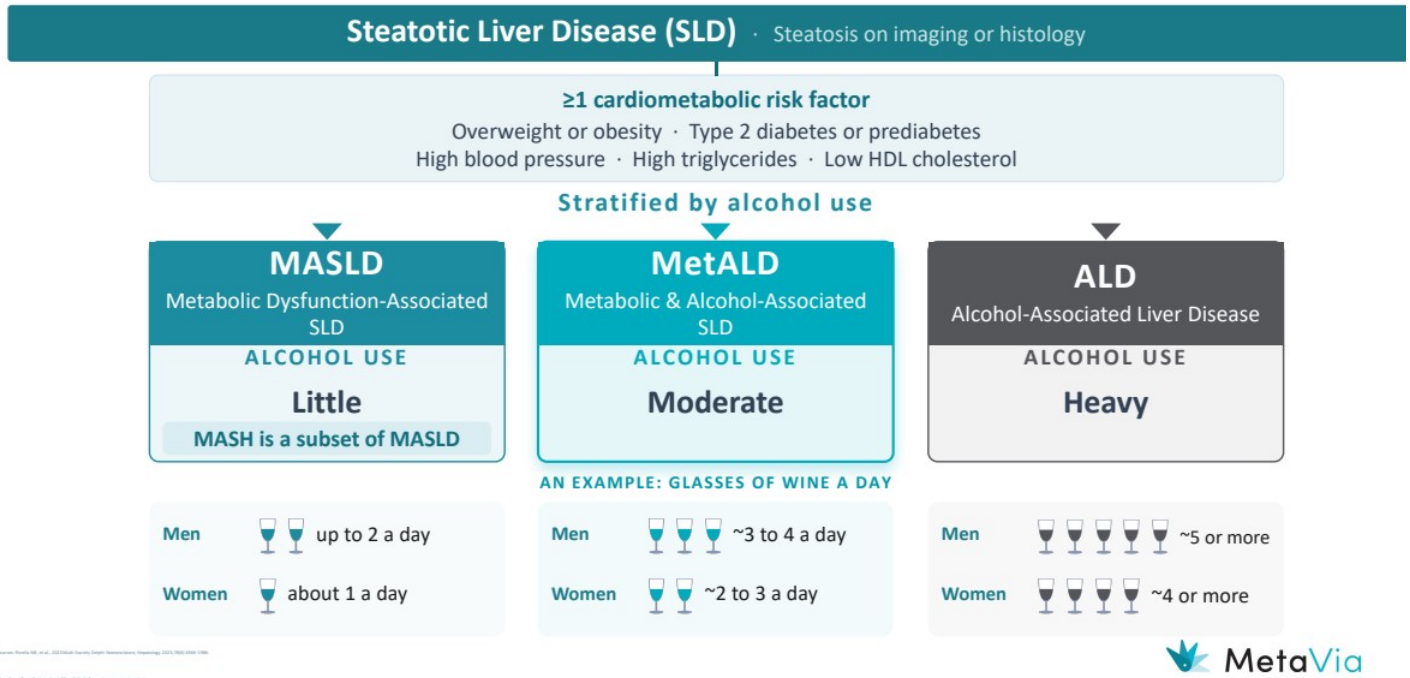
Part 3B Interim Highlights at 16 Weeks

- Placebo-adjusted liver fat reduction of −68% (MRI-PDFF, GMR 0.32, p<0.001). All five subjects with steatosis at entry (PDFF ≥5%) were below 5% by Day 109 — mean 8.3% → 3.0% (−5.3 pp, p=0.005).
- Placebo-adjusted body weight reduction of −13.5% (−13.5 kg; p<0.001) at Day 109 (~16 weeks), Part 3B interim (n=15 active vs. 4 placebo).
- HbA1c −0.44 pp on DA-1726 (p<0.001) and −0.24 pp vs. placebo (p=0.006) in a population without diabetes (baseline 5.79%); every subject with a Day 109 value fell by 0.3–0.8 pp.
- Consistent reductions across visceral and subcutaneous fat depots — VAT −22% (p<0.001), ASAT −19% (p=0.002), TAT −19% (p<0.001) — and waist circumference −9.1% (placebo-adjusted, p<0.001).
- Favorable safety and tolerability at Day 109: no new GI adverse events after Week 9 (Day 63) through study end (Day 109), and 0% study discontinuation due to GI AE.
- Favorable results support continued development toward Part 3A/3B topline in Q1 2027 and a Phase 2, 48-week, non-biopsy study in MetALD and ALD (up to 150 subjects), targeted to start in 2H 2027.

DA-1726 in MetALD / ALD

DA-1726 also carries potential in liver-focused MetALD/ALD, consistent with GCGR's role in adipose and hepatic fat. Planning for a Phase 2, 48-week, non-biopsy study in MetALD with up to 150 subjects, targeted to start in 2H 2027.

Metabolic and Alcohol-Associated Liver Disease (MetALD)
– A Newly Defined Disease Impacted by Cardiometabolic Risk and Moderate Drinking



MetaVia

MetaVia Inc. — DA-1726 & vanoglipel programs, November 2026

Sizing MetALD: A Wide Range, and Why

Published US prevalence estimates span 5.6M to 33M — nearly 6× — mostly because self-reported alcohol intake badly undercounts true consumption



FLOOR	COMPANY ESTIMATE	CEILING
5.6–5.8M	**10.7M**	**22–33M**
2.14–2.20% of US adults	4.10% of US adults	8–13% of US adults
NHANES self-report, uncorrected — Paik et al. 2025; Younossi et al. 2026 (uncorrected arm)	*NHANES self-report corrected & calibrated to NIAAA per-capita sales data — Younossi et al., Lancet Gastroenterol Hepatol 2026*	*Broader review-based steatosis definition (Leal-Lassalle et al. 2025, citing Kalligeros et al. 2024 & Ochoa-Allemant et al. 2023) — confirmed against primary source*

The core problem: self-reported drinking captures about a third of what's actually sold.

U.S. surveys capture only 22–32% of per-capita alcohol sales (BRFSS: 31.3%) — roughly 3 of the ~10 standard drinks per adult per week implied by NIAAA sales data. Heavier drinkers under-report more and respond to surveys less, pushing the heaviest-drinking tail furthest out of view.

Confirmed two independent ways — true intake runs **≈2×** reported levels:

Biomarkers: adding phosphatidylethanol (PEth) blood testing to questionnaires raised MetALD/ALD diagnoses up to 4× in one study; a review found 10–80% of self-reported MASLD patients already mee MetALD/ALD alcohol thresholds once biomarkers are used.
International pattern: the same survey-vs-sales gap shows up outside the US — England ~60% coverag and, on average, 36.5% across 39 surveys in 23 European countries — so this isn't a quirk of one US instrument.

Two further caveats on the headline numbers: *MetALD status is dynamic, not a fixed population — in one cohort only 38% of patients still met criteria two years later. And the treatment-eligible subset is smaller still: trials typically target F2+ fibrosis, 15–20% of the prevalent population.*

Sources: Younossi et al., Lancet Gastroenterol Hepatol 2026;11(6):495–508; Paik et al., J Clin Gastroenterol 2025; Leal-Lassalle et al., npj Gut and Liver 2025, citing Kalligeros et al., Clin Gastroenterol Hepatol 2024;22:1330–1332.e4, and Ochoa-Allemant et al., Hepatol Commun 2023;7:e0324; NIAAA Surveillance Report #122 (Slater & Al 2025); Nelson et al., Addiction 2010;105:1589–1596; Esser et al., J Stud Alcohol Drugs 2022;83(1):134–144; Kerr & Greenfield, Alcohol Clin Exp Res 2007;31(10):1714–1722; Stockwell et al., Addiction 2018;113:2245–2249; Díaz et al., Nat Rev Gastroenterol Hepatol 2025 & 2026, and underlying Vaz et al., J Hepatol 2025;83:1011–1022 (reclassification); Boniface & Shelton, Eur J Public Health 2013;23(6):1076–1081 (England); Kilian et al., Alcohol Alcohol 2020;55(5):554–563 (23 European countries).

MetaVia Inc. — DA-1726 & vanoglipel programs, November 2026

Why MetaVia Can Lead in MetALD

Nobody is set to generate MetALD-specific evidence — or a MetALD label — near term. The first program that does owns the indication and the regulatory conversation.

MetALD is empty.

no approved therapy; every MASH label excludes it

Big pharma is elsewhere.

MASH outcomes programs run to 2030 and beyond

DA-1726 is built for it.

liver fat, stiffness, drinking, weight, HbA1c — in one study

First to read out.

the first dedicated MetALD / ALD program readou
start targeted 2H 2027

WHY A MetALD LABEL IS UNLIKELY TO COME FROM BIG PHARMA NEAR TERM

Incremental patients, pricing conflict. Obesity and MASH labels already reach MetALD patients off-label; US pricing is per product, so a MetALD label adds payer friction, not revenue.

A new program, not an extension. Every MASH pivotal excluded alcohol above ~20–30 g/day; the FDA has not yet recognized MetALD as a distinct indication.

Capital committed to MASH through 2030. A US population roughly three times the size of MetALD on its floor estimate (~16.5M vs 5.6–5.8M).

WHY DA-1726 FITS THE GAP

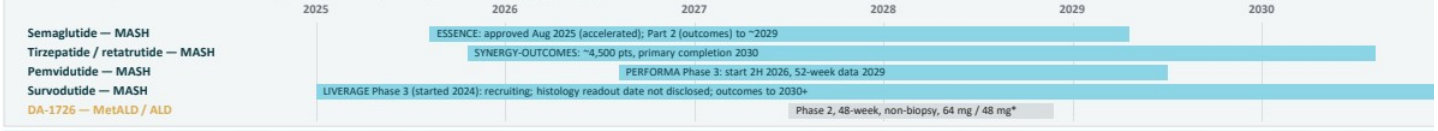
Strongest liver signal in the incretin class. Efinopegdutide beat semaglutide head-to-head on liver f
(72.7% vs 42.3%); survodutide's fibrosis improvement (up to 64.5% vs 25.9%, F2–F3) is among the largest reported.

Same receptor pair, more glucagon-weighted — and better tolerated. DA-1726's 3:1 GLP1R:GCGR
balance carries more glucagon activity than survodutide's (~8:1), the component behind the hepatic effect; −68% placebo-adjusted liver fat at 16 weeks, no discontinuations and no new GI adverse ever after Week 9 — vs 20% AE discontinuation for survodutide in MASH.

A purpose-built Phase 2 — our lead indication. 48-week, non-biopsy, 64 mg / 48 mg / placebo; MetALD and ALD; start targeted 2H 2027.

The lower-risk route in for a large franchise is a dedicated asset. Pricing, reimbursement and chan
considerations favor a licensed or acquired MetALD asset over a label extension.

THE WINDOW — WHERE THE LARGE PROGRAMS ARE POINTED THROUGH 2030



Field watch: Novo Nordisk (semaglutide combinations, completed) and Altimmune (pemvidutide RESTORE, data 2H 2027) run Phase 2 studies in ALD — heavy drinkers with fibrosis, not MetALD. The only MetALD-specific study of a major-class agent is a 12-week NIAAA tirzepatide trial.

** Target start in 2H 2027. Milestones assume regulatory and clinical success, which is not guaranteed. Gray boxes are prospective future studies; timing and occurrence subject to various factors. Sources: Sanyal et al., NEJM 2025 (ESSENCE; alcohol exclusion >20/30 g/day); Loomba et al., NEJM 2024 (SYNERGY-NASH); Noureddin et al., Lancet 2025 (IMPACT); Sanyal et al., NEJM 2024 (survodutide MASH Ph2) and Boehringer EASL 2024; Zimmermann et al., Mol Metab 2022 (survodutide receptor balance); Romero-Gómez et al., J Hepatol 2023 (efinopegdutide vs semaglutide); Novo Nordisk Aug 15, 2025 (Wegovy MASH approval); NCT07165028, NCT06632444 (LIVERAGE), NCT06409130, NCT07046819; Altimmune May and Aug 2026 updates; Estes et al., Hepatology 2018 (US NASH 16.5M in 2015) vs MetALD floor estimate 5.6–5.8M (slide 11); Rinella et al., Hepatology 2023; Acuña et al. 2025 and Pagadala et al. 2026, Metab Target Organ Damage. Competitor plans and timelines are as publicly disclosed and may change.*

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MetaVia Inc. — DA-1726 & vanoglipel programs, November 2026

DA-1726 Phase 2 in MetALD / ALD: Planned Study Design

Randomized, double-blind, placebo-controlled, non-biopsy — planned with leading hepatology investigators; start targeted 2H 2027

STUDY DESIGN AT A GLANCE (PLANNED)

PLANNED DESIGN — DIRECTIONAL



WHO

- Adults with MetALD or ALD — steatotic liver disease with alcohol intake above the MASLD threshold; MetALD is the focus, so a cardiometabolic risk factor and BMI ≥27 kg/m² are required, with no upper BMI limit
- At risk for F2–F3 fibrosis and progression on non-invasive tests; non-cirrhotic
- Mild-to-moderate alcohol use disorder permitted; severe AUD excluded

WHAT WE MEASURE

- Liver fat and liver stiffness — the primary and key secondary endpoints
- Fibrosis biomarkers (ELF, Pro-C3, FAST), liver enzymes
- Alcohol intake — PEth, heavy-drinking days, timeline follow-back
- Body weight and waist circumference
- Glycemic control — HbA1c

WHY IT IS BUILT THIS WAY

- Non-invasive endpoints make a 48-week study feasible and fast enroll — no biopsies
- Week 24 liver-fat primary reads early; Week 48 stiffness speaks fibrosis
- Drinking measured objectively (PEth) alongside self-report — reduction, not abstinence
- Two doses allow a dose-response read on the 64 mg target dos

Planned design elements are directional: endpoint hierarchy, eligibility thresholds, sample size and schedule remain subject to FDA feedback and the final protocol. MetALD / ALD per Rinella et al., Hepatology 2023. PEth: phosphatidylethanol, a direct blood marker of alcohol intake over the preceding 2–4 weeks.

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DA-1726: Part 3B Interim Results

MetaVia's own DA-1726 — a 3:1 GLP1R/GCGR dual agonist.

1 DA-1726: Mechanism & the Part 3B Trial

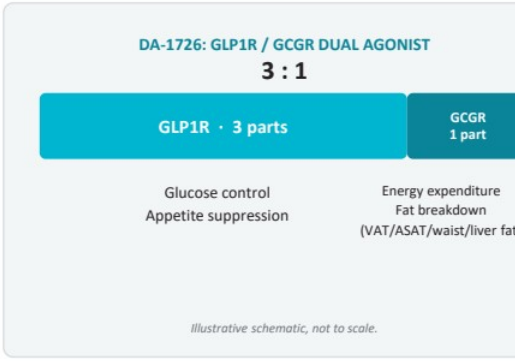
WHAT IS DA-1726?

Mechanism: A novel oxyntomodulin (OXM) analogue — a dual agonist of the GLP-1 receptor (GLP1R) and glucagon receptor (GCGR), dosed once weekly, subcutaneously. In development for obesity and potential for liver focused MetALD/ALD.

3:1 design ratio: Engineered with a 3:1 GLP1R:GCGR potency ratio. The 3:1 ratio of DA-1726 may provide an optimal balance to achieve a sustainable and tolerable therapeutic effect.

Why VAT/ASAT respond: Tissue-distribution study: DA-1726 concentrates most heavily in adipose tissue (abdominal subcutaneous, brown fat) and liver, consistent with GCGR's role in adipose breakdown.

Lean mass relatively preserved: Day 109: thigh muscle volume −9%, total lean tissue −8%, vs. −19–22% in fat compartments (VAT/ASAT/TAT) — consistent with fat-selective tissue distribution.



PART 3B TRIAL DESIGN AT A GLANCE

Design	Phase 1, Part 3B — 16-week, randomized, placebo-controlled obesity dose-titration study (16 mg → 32 mg → 64 mg SC, once weekly)
Eligibility	Adults 18–65 years with obesity (BMI 30–45 kg/m²), no diabetes, ALT/AST <1.25× ULN; randomized 4:1 to DA-1726 or placebo (n=15 vs. 4 in the safety population)
This dataset	Day 109 (~16-week) INTERIM analysis: n=15 active/4 placebo (weight, waist circumference, FibroScan); n=12 active/4 placebo (MRI sub-study: VAT/ASAT/TAT, liver f thigh muscle)
Upcoming public readouts	Part 3B interim results — oral presentation at the Precision Liver Symposium, Nov 2026; complete Part 3A and Part 3B topline data — Q1 2027

Every figure in this deck is drawn from this Part 3B interim dataset — not the company's forthcoming public topline readouts. *DA-1726's separate, completed Phase 1 MAD trial (2024, single/multiple ascending doses to 48 not combined with Part 3B data anywhere in this deck.*

2 Baseline Characteristics

Part 3B, safety population — DA-1726 (n=15) vs. placebo (n=4). Mean (SD) unless noted.

Characteristic	DA-1726 64 mg (n=15)	Placebo (n=4)
Age, years	43.7 (10.9)	50.3 (4.9)
Male, n (%)	11 (73.3)	1 (25.0)
Body weight, kg	98.6 (13.9)	86.7 (9.1)
Body mass index, kg/m²	34.0 (2.5)	32.9 (2.1)
BMI ≥35 kg/m², n (%)	5 (33.3)	1 (25.0)
Waist circumference, cm	107.5 (6.9)	99.5 (9.0)
HbA1c, %	5.79 (0.28)	5.65 (0.17)
Liver fat (MRI-PDFF), % — MRI sub-study, n=12 / 4	5.2 (3.4)	8.0 (6.9)
Steatosis (MRI-PDFF ≥5%), n (%) — MRI sub-study, n=12 / 4	5 (41.7)	2 (50.0)

Day 109 interim analysis; percentages based on n per arm.

3 Where DA-1726 Stands After 16 Weeks

MetALD = a steatotic liver + at least one cardiometabolic risk factor. DA-1726 moved all three in 16 weeks.

LIVER

Steatotic liver disease

-68%

Placebo-adjusted liver fat reduction

MRI-PDFF, GMR 0.32 vs. placebo, Day 109, n=12; **p<0.001**

CARDIOMETABOLIC RISK FACTORS

Weight / adiposity

-13.5%

Body weight, placebo-adjusted

−13.5 kg absolute difference; difference in mean change vs. placebo, Day 109, n=15 vs. 4; **p<0.001**

Glycemia

-0.24 pp

HbA1c, placebo-adjusted

Baseline 5.79%; −0.44 pp on DA-1726 vs. −0.20 on placebo, Day 109, n=15 vs. 4; **p=0.006**

Day 109 (~16 weeks) interim: MRI sub-analysis n=12 vs. 4; clinical readout n=15 vs. 4. All three results placebo-adjusted.

MetALD definition (Rinella et al., Hepatology 2023): hepatic steatosis on imaging or histology, at least one of five cardiometabolic criteria — BMI or waist circumference, fasting glucose / HbA1c or type 2 diabetes, blood pressure, triglycerides, HDL cholesterol — and weekly alcohol intake above the MASLD threshold (140–350 g women, 210–420 g men). Body weight and HbA1c are two of the five criteria.

4 Liver Fat

MRI-PDFF, Part 3B interim — Day 109 (~16 weeks)

5.2%

Baseline liver fat (MRI-PDFF)

64 mg dose, n=12. Placebo baseline 8.0% (n=4).

-68%

Placebo-adjusted (GMR)

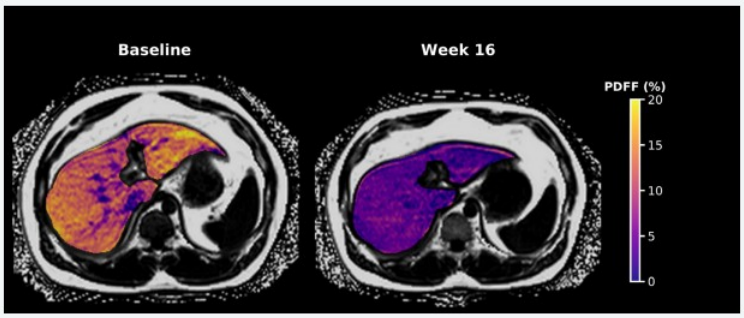
GMR 0.32, Day 109; **p<0.001**

5 of 5

Baseline steatosis (≥5%): all normalized

All five below 5% by Day 109 — e.g., the highest, 12.8% → 4.6%; mean 8.3% → 3.0% (−5.3 pp; **p=0.005)**

ONE SUBJECT — AXIAL MRI-PDFF LIVER FAT-FRACTION MAP, BASELINE → WEEK 16 (DAY 109)



10 of 12 (83%) achieved a ≥30% reduction in liver fat — vs. 0 4 on placebo (p=0.008)

≥30% relative reduction is the responder threshold used in MASH trials (Fisher's exact). Group result, same MRI sub-study: liver fat −68% placebo-adjusted (GMR 0.32, p<0.001), n=12 active / 4 placebo.

How to read the image

The color overlay maps the fat fraction (PDFF) of every pixel of liver tissue: yellow–orange is 15–20% fa purple is 0–5% (scale at right). At baseline the liver is largely orange — steatotic; at Week 16 it is unifor purple — liver fat has fallen into the normal range.

Single representative active-arm subject at the 64 mg target dose. Fat-fraction color map overlaid on the anatom image; same scale at both visits. A single subject is illustrative and not representative of the treatment effect.

Placebo: +40.4% from its own 8.0% baseline. Absolute MRI-PDFF treatment difference: −6.4 percentage points, p=0.007. ≥30% relative reduction is the responder threshold used in MASH trials.

5 Body Weight

Part 3B interim, obesity population — Day 109 (~16 weeks), n=15 active vs. 4 placebo, 64 mg target dose

98.6 kg

Baseline body weight

DA-1726 arm mean, 64 mg target dose, n=15 (placebo n=4).

-12.2 kg

Change from baseline, Day 109

DA-1726 arm (n=15): −12.19 kg, −11.9% (both p<0.001). Placebo arm (n=4): +1.35 kg, +1.6%.

-13.5%

Placebo-adjusted

Difference in mean % change vs. placebo, Day 109, n=15 vs. 4: −13.5 pp (−13.5 kg; 95% CI −18.6 to −8.5), two-sample t-test, multiple-imputation analysis; **p<0.001**

Also placebo-adjusted, Day 109: waist circumference −9.6 cm (−9.1 pp), p<0.001 — see the Waist Circumference slide; HbA1c −0.24 pp, p=0.006 — next slide.

6 Glycemic Control: HbA1c

Part 3B interim, obesity population without diabetes — Day 109 (~16 weeks), n=15 active vs. 4 placebo, 64 mg target dose

5.79%

Baseline HbA1c

DA-1726 arm mean, n=15 (placebo 5.65%, n=4). Prediabetes range (5.7–6.4%); no subject with diabetes.

-0.44 pp

Change from baseline, Day 109

DA-1726 arm (n=15): −0.44 pp, to 5.33% (95% CI −0.59 to −0.29; **p<0.001**). Every subject with a Day 109 value fell by 0.3–0.8 pp. Placebo arm (n=4): −0.20 pp.

-0.24 pp

Placebo-adjusted

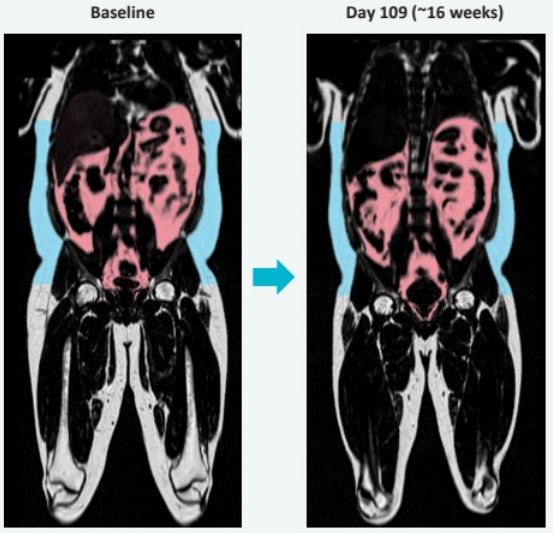
Difference in mean change vs. placebo, Day 109, n=15 vs. 4: −0.24 pp (95% CI −0.41 to −0.07), two-sample t-test, multiple-imputation analysis; **p=0.006**

A glycemic effect in a population without diabetes (fasting glucose 88.5 mg/dL at baseline). Glycemic control (HbA1c) is a secondary endpoint in the planned Phase 2 MetALD / ALD study.

7 Body Composition: Fat vs. Lean Mass

MRI sub-study (AMRA), Part 3B interim — 64 mg target dose, 16-wk titration (n=12 active vs. 4 placebo)

ONE SUBJECT, BASELINE → DAY 109 · AMRA FAT-COMPARTMENT SEGMENTATION



Single representative active-arm subject; individual values not shown. Group results at right.

FAT COMPARTMENTS — RELATIVE TREATMENT DIFFERENCE vs PLACEBO (GMR), DAY 109

−22%
VAT (visceral adipose tissue)
*Baseline 5.44 L; **p<0.001***

−19%
ASAT (subcutaneous)
*Baseline 10.16 L; **p=0.002***

−19%
TAT (total adipose tiss
*Baseline 29.6 L; **p<0.001***

LEAN / MUSCLE COMPARTMENTS — RELATIVELY PRESERVED

−9%
Thigh muscle volume
*vs. placebo, Day 109; **p=0.018***

−8%
TLT (total lean tissue)
*GMR 0.92 vs. placebo, Day 109; **p=0.007***

Fat-selective by design

Fat compartments fell 19–22% against an 8–9% change in lean compartments — consistent with DA-17 concentrating in adipose tissue and liver (tissue-distribution study) and with GCGR-driven fat breakdow Liver fat in the same sub-study: −68%, placebo-adjusted (see Liver Fat).

Baselines are the active-arm mean; reductions are relative treatment differences (GMR) vs. placebo at Day 109, MRI sub-study n=12 active / 4 placebo. Images: AMRA Profiler segmentation of one active-arm subject at the 64 mg target dose; pink = visceral, blue = abdominal subcutaneous adipose tissue. A single subject is illustrative and not representative of the treatment effect.

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8 Waist Circumference

Part 3B interim, obesity population — Day 109 (~16 weeks), n=15 active vs. 4 placebo, 64 mg target dose

107.5 cm

Baseline waist circumference

64 mg target dose, n=15 active (n=4 placebo).

-9.6 cm

Change from baseline, Day 109

DA-1726 arm (n=15): −9.6 cm, −9.0% (both p<0.001).
Placebo arm (n=4): 0.0 cm, +0.1%.

-9.1%

Placebo-adjusted

Difference in mean % change vs. placebo, Day 109, n=15 vs. 4: −9.1 pp (−9.6 cm), two-sample t-test, multiple-imputation analysis; **p<0.001**

9 Safety & Tolerability: GI Adverse Events

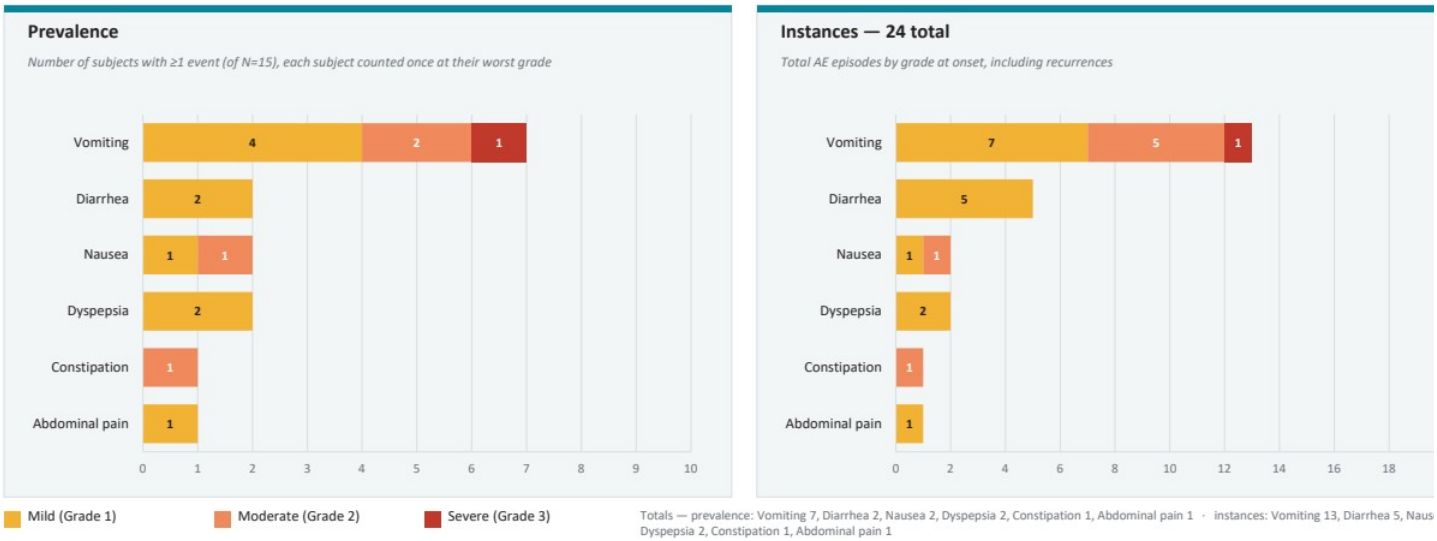
Maximum severity per subject, per preferred term — Part 3B interim (N=15 active, N=4 placebo). Placebo reported 0% on every term below (small n; not a powered comparison).

Preferred term	Mild	Moderate	Severe	Any grade
Any GI AE (Gastrointestinal disorders, SOC)	**5 (33.3%)**	**3 (20.0%)**	**1 (6.7%)**	**9 (60.0%)**
Vomiting	4 (26.7%)	2 (13.3%)	1 (6.7%)	**7 (46.7%)**
Nausea	1 (6.7%)	1 (6.7%)	0	**2 (13.3%)**
Diarrhea	2 (13.3%)	0	0	**2 (13.3%)**
Dyspepsia	2 (13.3%)	0	0	**2 (13.3%)**
Constipation	0	1 (6.7%)	0	**1 (6.7%)**
Abdominal pain	1 (6.7%)	0	0	**1 (6.7%)**

- **Notable AE:** 1 SAE — severe vomiting, possibly related, recovered. 0% study discontinuation due to GI AE.
- **No new GI AEs after Week 9 (Day 63) through study end (Day 109).**

10 GI Adverse Events: Prevalence vs. Instances

Same GI AE data as the prior table, visualized two ways: prevalence (subjects affected, worst grade each) vs. instances (total episodes, graded at onset) — Part 3B interim (N=15 active).



Totals — prevalence: Vomiting 7, Diarrhea 2, Nausea 2, Dyspepsia 2, Constipation 1, Abdominal pain 1 · instances: Vomiting 13, Diarrhea 5, Naus
Dyspepsia 2, Constipation 1, Abdominal pain 1

No new GI adverse events reported after Week 9 (Day 63) through study end (Day 109).



Day 0 *Day 63 (Week 9)*

Timeline reflects the trial-day GI AE reporting window, not a modeled daily-prevalence curve. Source: Part 3B interim analysis, Day 109.

MetaVia

11

DA-1726: Layered Patent Protection

Two granted composition-of-matter families — the core chemistry and a product-specific family — with exclusivity to 2041

29
granted patents
across both families

16
jurisdictions with granted patents
incl. US, Europe, Japan, China, Korea

2038
core chemistry protected to
Family 1, granted

2041
product-specific protection to
Family 2, granted and expanding

IN PLACE TODAY

Family 1 — composition of matter: core chemistry

GRANTED IN

United States (2) · Europe · Japan (2) · China · Korea (2) · Canada · Australia · India · Mexico · Russia · Israel · Saudi Arabia (2) · Singapore · Hong Kor · New Zealand · Philippines

20 granted patents · protection to 2038

ADDED PROTECTION

Family 2 — composition of matter: product-specific

GRANTED IN

Korea · Japan · China · Australia · India · Russia · Saudi Arabia · Hong Kong · New Zealand

9 granted patents · protection to 2041 · further grants expected in the US, Europe, Canada and 5 other markets

Source: company records, September 2026. All patents and applications are owned by Dong-A ST Co., Ltd. and licensed to MetaVia. Counts include divisional patents. Protection dates are estimated from the PCT filing date and exclude any patent term adjustment, extension or supplementary protection certificate that may be available.

Competitive Landscape

Five leading GLP-1-based obesity and MASH comparators — Tirzepatide, Retatrutide, Mazdutide, Survodutide, and Pemvidutide. *No head-to-head study has been conducted between DA-1726 and any product shown in this section; the data reflect different trials, populations, doses, and timepoints and are not intended as direct comparisons.*

12 Competitive Landscape: Positioning

Obesity and weight-related endpoints, positioned by mechanism, dosing, and development stage across all six programs.

	DA-1726	Tirzepatide	Retatrutide	Mazdutide	Survodutide	Pemvidutide
Mechanism	GLP-1/GCG dual agonist 3:1 GLP1R:GCGR ratio	GLP-1/GIP dual agonist	GLP-1/GIP/GCG triple agonist	GLP-1/GCG dual agonist	GLP-1/GCG dual agonist	GLP-1/GCG dual agoni
Dosing	Once weekly, SC	Once weekly, SC	Once weekly, SC	Once weekly, SC	Once weekly, SC	Once weekly, SC
Lead Trial	Part 3A/3B (Ph.1, obesity) Topline expected Q1 2027	SURMOUNT-1 (Ph.3, obesity)	TRIUMPH-1 (Ph.3, obesity)	Lilly Ph.2 (obesity)	SYNCHRONIZE-1 (Ph.3, obesity)	MOMENTUM (Ph.2, obes IMPACT (Ph.2b, MASH
Development Stage	Phase 1	Approved (obesity)	Phase 3	Approved (China, obesity); Ph.2 (US)	Phase 3	Phase 2
Positioning Notes	Adipose/liver-selective tissue distribution	First approved GLP-1/GIP dual agonist	Obesity-focused program	Co-developed with Innovent Biologics (China)	Parallel obesity and MASH programs	Parallel obesity and MASH programs

−68%
Liver Fat (placebo-adj.)

−13.5%
Body Weight (placebo-adj.)

−0.24 pp
HbA1c (placebo-adj.)

DA-1726's own interim data, shown on its own terms. Day 109 (~16-week) readout from Part 3B, n=15 active / 4 placebo, Phase 1 dose-titration study. Not compared numerically to any program abo — an early, small-sample interim readout is not comparable to the pivotal or later-stage data shown for other programs.

Positioning notes reflect each program's publicly disclosed mechanism, dosing, and development stage; they are not comparative efficacy or safety claims. Tirzepatide: Jastreboff et al., NEJM 2022 (SURMOUNT-1); Gastaldelli et al., Lancet Diabetes Endocrinol 2022. Retatrutide: Jastreboff et al., 2023; TRIUMPH-1. Mazdutide: Hsia et al., Lancet Diabetes Endocrinol 2026; NMPA approval, Innovent press release, June 2025. Survodutide: le Roux et al., NEJM 2026 (SYNCHRONIZE-1). Pemvidutide: Altimmune, MOMENTUM/IMPACT topline data.

13 Competitive Landscape: DA-1726 (MetaVia)

GLP1R/GCGR dual agonist (OXM analogue), 3:1 ratio · *Part 3B interim: Ph.1 obesity dose-titration (16→32→64 mg SC, QW) · Day 109 (~16 wk), n=15 active/4 placebo*

Body Weight

Baseline 98.6 kg

Day 109 (~16 wk):
−12.2 kg from baseline

−13.5% placebo-adjusted, p<0.001

Waist Circumference

Baseline 107.5 cm

Day 109 (~16 wk):
−9.6 cm from baseline

−9.1% placebo-adjusted, p<0.001

Liver Fat

Baseline 5.2% (MRI-PDFF, n=12)

Day 109 (~16 wk):
−68% *(GMR, placebo-adjusted)*

Placebo-adjusted, p<0.001 (GMR 0.32)

VAT / ASAT / TAT

VAT: −22%
*Baseline 5.44 L · Day 109; **p<0.001***

ASAT: −19%
*Baseline 10.16 L · Day 109; **p=0.002***

TAT: −19%
*Baseline 29.6 L · Day 109; **p<0.001***

GI Adverse Events — *Part 3B interim (N=15 active, N=4 placebo)*

Nausea	Vomiting	Diarrhea	Constipation	Disc. due to AE
13.3%	**46.7%**	**13.3%**	**6.7%**	**0%**

Notable AE: 1 SAE — severe vomiting, possibly related, recovered.

No new GI adverse events reported after Week 9 (Day 63) through study end (Day 109).



Timeline reflects the trial-day GI AE reporting window, not a modeled daily-prevalence curve.

14 Competitive Landscape: Tirzepatide

GLP-1/GIP dual agonist · Pivotal: SURMOUNT-1 (Ph.3, obesity) · 15 mg (comparator dose used throughout) · approved for obesity (Zepbound, Nov 2023)

Body Weight

Baseline 104.8 kg (SURMOUNT-1 trial-wide mean, all arms)

Early — wk 20 (pooled tirzepatide):
−12.8 kg own-arm; placebo −2.7 kg (key secondary endpoint)

Pivotal — 72 wk (15 mg): −20.9% (placebo −3.1%; −17.8 pp placebo-adjusted)

Waist Circumference

Baseline 114.1 cm (SURMOUNT-1 trial-wide mean)

Wk 72 by dose (5 / 10 / 15 mg), own-arm:
−14.0 / −17.7 / −18.5 cm placebo −4.0 cm

Pivotal — 72 wk (15 mg): −18.5 cm (placebo −4.0 cm; −14.5 cm placebo-adjusted, 95% CI −15.9 to −13.0)

Liver Fat

Baseline 16.7% (SURPASS-3, 15 mg, type 2 diabetes) · 18.4% (SYNERGY-NASH, MASH F2–F3)

Wk 52 (15 mg), T2D — SURPASS-3 MRI substudy:
−39.6% own-arm; degludec −11.2% (−28.4 pp); 10 mg arm −47.1%

MASH — SYNERGY-NASH, wk 52 (15 mg): −57.0% (own-arm; placebo −9.8%; −47.2 pp)

VAT / ASAT

VAT: −24.0% (15 mg)

MRI, 6.81 → 4.88 L · wk 52 — own-arm (degludec +8.1%)

ASAT: −19.4% (15 mg)

MRI, 10.34 → 8.42 L · wk 52 — own-arm (reported relative char

TAT: not reported

SURPASS-3 MRI substudy, type 2 diabetes population (n=296)

GI Adverse Events — SURMOUNT-1, 15 mg (Ph.3, obesity, 72 wk)

Nausea	Vomiting	Diarrhea	Constipation	Disc. due to AE
31.0%	**12.2%**	**23.0%**	**11.7%**	**6.2%**

Placebo: nausea 9.5%, vomiting 1.7%, diarrhea 7.3%, constipation 5.8%, discontinuation 2.6%. Most GI events mild-to-moderate, during dose escalation.

The approved benchmark — and a MASH program alongside it

Tirzepatide is the only program in this section with an obesity approval (Zepbound, FDA, November 2023). In SYNERGY-NASH (Ph.2, MASH with F2–F3 fibrosis, 52 weeks), MASH resolution without worsening of fibrosi was 44% / 56% / 62% at 5 / 10 / 15 mg vs 10% on placebo, with ≥1-stage fibrosis improvement in 51–55% vs 30%; at 15 mg, liver fat fell 57.0% and body weight 15.6% over 52 weeks. VAT / ASAT figures above come from a type 2 diabetes population (SURPASS-3 MRI substudy), not from the obesity or MASH trials.

Sources: Jastreboff AM et al., N Engl J Med 2022;387:205–216 (SURMOUNT-1; Table 2, Table 3); Gastaldelli A et al., Lancet Diabetes Endocrinol 2022;10:393–406 (SURPASS-3 MRI substudy; Table 2); Loomba R et al., N Engl J Med 2024;391:299–310 (SYNERGY-NASH; Table 2 and S Table S6); Zepbound US Prescribing Information (Nov 2023). Cross-trial figures are not head-to-head comparisons.

15 Competitive Landscape: Retatrutide

GLP-1/GIP/GCG triple agonist · *Pivotal: TRIUMPH-1 (Ph.3, obesity) · 12 mg (comparator dose used throughout)*

Body Weight

Baseline 112.7 kg (TRIUMPH-1 trial-wide average, all doses)

Early — wk 24 (12 mg):
−15.8% *placebo-adjusted (own-arm −17.5%; 95% CI −17.6 to −14.1)*

Pivotal — 80 wk (12 mg): −28.3%

Waist Circumference

Baseline 118.3 cm (TRIUMPH-1 trial-wide average, all doses)

Early — wk 24 (12 mg):
−11.8 cm *placebo-adjusted (−14.3 cm vs −2.5 cm placebo; NEJM 2023 Table S4)*

Pivotal — −20.5 cm (−17.3%)*

Liver Fat

Baseline 15.6–21.0% (12 mg, range across arms, not dose-specific)

Early — wk 24 (12 mg):
−82.4% *own-arm (placebo +0.3%)*

Pivotal — wk 48: −86.0% (own-arm; placebo −4.6%)

VAT / ASAT / TAT

VAT: Wk24 −28.0% → Wk48 −44.5%
(12 mg)
*MRI (AMRA), 12 mg — **own-arm***

ASAT: Wk24 −24.9% → Wk48 −43.5
(12 mg)
*MRI (AMRA), 12 mg — **own-arm***

Total fat mass (DXA): −18.7 pp (12 m

baseline 45.3 kg · wk 36 · vs placebo, p<0.0001 · T2D population (Coskun 2025)

GI Adverse Events — *TRIUMPH-1, 12 mg (Ph.3, obesity)*

Nausea	Vomiting	Diarrhea	Constipation	Disc. due to AE
42.4%	**25.3%**	**32.0%**	**26.1%**	**11.3%**

AEs of special interest: Hypersensitivity: 13%; Cardiac arrhythmia: 11%; Pancreatitis: 2%; Biliary/GB: 0%

Population changes the tolerability picture, not just dose

TRIUMPH-1's obesity-population rates (12 mg: nausea 42.4%, vomiting 25.3%, discontinuation 11.3%) are markedly higher than the same 12 mg dose in a T2D population (TRANSCEND-T2D-1: nausea 26.5%, vomiting 17.6%, diarrhea 22.8%, discontinuation 5.1% vs. 0% placebo). A reminder that tolerability comparisons must match population and duration, not only dose.

Sources: TRIUMPH-1 — Eli Lilly press release, May 21, 2026; TRANSCEND-T2D-1 — Eli Lilly press release, March 19, 2026.

**Pivotal waist-circ figure (TRIUMPH-1, Lilly press release, May 2026) supersedes an earlier Ph.2 estimate. Liver-fat and VAT/ASAT figures (Sanyal, Ph.2a MASLD substudy) are own-arm changes — placebo's own drift is noted on-card but not subtracted. Sources: Jastreboff et al., NE (Ph.2); Sanyal et al., Nat Med 2024 (Ph.2a MASLD substudy); Coskun et al., Lancet Diabetes Endocrinol 2025;13:674–684 (Ph.2 T2D body-composition substudy; total fat mass by DXA); TRIUMPH-1 (Ph.3, obesity; Lilly press release, May 2026).*

16 Competitive Landscape: Mazdutide

GLP-1/GCG dual agonist · *Pivotal: Lilly Ph.2 (obesity) · 16 mg for obesity endpoints; 6 mg for liver fat (GLORY-1, Ph.3)*

Body Weight

Baseline 109.7 kg

Early — wk 12 (9 mg):
−9.8% *placebo-adjusted*

Pivotal — 48 wk (16 mg): −22.3%

Waist Circumference

Baseline 115.8 cm (16 mg)

Early — wk 12 (9 mg):
−5.15 cm *placebo-adjusted, p=0.027*

Pivotal — −15.4 cm (≈−13.3%), p<0.001

Liver Fat

Baseline 21.3% MRI-PDFF (GLORY-1, 6 mg, LFC ≥10% subgroup, n=15; placebo 19.6%)

Wk 48 (6 mg) — ≥30% relative liver-fat reduction:
95.5% of patients vs 27.3% on placebo (LFC ≥5% cohort, n=22)

Pivotal — wk 48 (6 mg): −80.2%
own-arm; placebo −5.3% (GLORY-1, LFC ≥10% subgroup)

VAT / ASAT / TAF

VAT: −32.1% vs placebo (16 mg)
MRI · wk 48 · own-arm −33.8%, placebo −2.4 to −3.4% (Hsia 202 appendix)

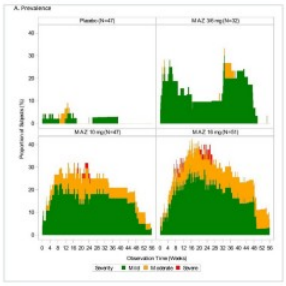
ASAT: −31.1% vs placebo (16 mg)
MRI · wk 48 · own-arm −33.5%, placebo −2.4 to −3.4% (Hsia 202 appendix)

TAF: −32.7% vs placebo (16 mg)
TAF = total abdominal fat (VAT + SAT); whole-body TAT not repo
MRI · wk 48 · own-arm −34.3%, placebo −2.4% (Hsia 2026, appe

GI Adverse Events — *Lilly Ph.2, 16 mg (Ph.2, obesity)*

Nausea	Vomiting	Diarrhea	Constipation	Disc. due to AE
60.8%	**45.1%**	**25.5%**	**35.3%**	**19.6%**

Placebo: nausea 25.5%, vomiting 2.1%, diarrhea 12.8%, constipation 10.6%, discontinuation 0% (Hsia 2026, appendix).



How it unfolds over time: Daily prevalence of combined N/V/D, by severity and dose arm, through week 56.

Source: Hsia et al., Lancet Diabetes Endocrinol 2026 — Suppl. Appendix, Fig. S6 (Panel A shown).

Sources: Ji et al., Ph.1b, eClinicalMedicine 2022 (Chinese overweight/obesity); Hsia et al., Lancet Diabetes Endocrinol 2026 (Ph.2, obesity), presented Obesity Week 2025; liver fat: Ji L et al., N Engl J Med 2025;392:2215–2225 (GLORY-1, Ph.3, Chinese adults; Suppl. Fig. S8: LFC ≥10% vs −5.3%; LFC ≥5% −73.2% vs +8.2%; ≥30% responders 95.5% vs 27.3%).

17 Competitive Landscape: Survodutide

GLP-1/GCG dual agonist · *Pivotal: SYNCHRONIZE-1 (Ph.3, obesity) · 6.0 mg (comparator dose used throughout)*

Body Weight

Baseline 108.8 kg (trial-wide, not dose-specific)

Early — wk 46 (4.8 mg):
−12.1% *placebo-adjusted*

Pivotal — 76 wk (6.0 mg): −16.6%

Waist Circumference

Baseline 115.0 cm (6.0 mg, overall pop.)

Early — wk 46 (4.8 mg):
−16.6 cm *own-arm — not placebo-adjusted, so not directly comparable to the pivotal figure; sourced from companion conference abstracts (primary paper's table was inaccessible)*

Pivotal — −6.1 cm, p<0.001 (placebo-adjusted)

Liver Fat

Baseline 17.9% (6.0 mg)

Early — wk 28 (6.0 mg):
−62.0% *own-arm (placebo −13.1%); pre-planned wk-28 interim, n=138 (Alkhouri et al., DDW 2024)*

Pivotal — wk 48: −62.0% (own-arm; placebo −5.7%)

VAT / ASAT / TAT

VAT: −22.2 pp vs PBO (6.0 mg)
MRI substudy · wk 76 · own-arm −34.0% vs −11.8% (NEJM 2026, 2)

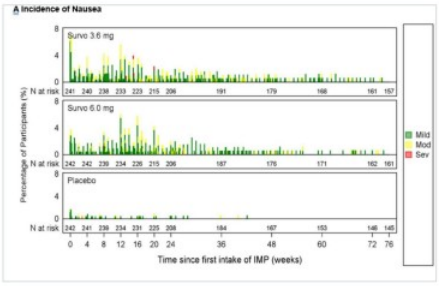
SAT: −18.3 pp vs PBO (6.0 mg)
MRI substudy · wk 76 · own-arm −28.1% vs −9.8% (NEJM 2026, 2)

Total fat: −18.3 pp vs PBO (6.0 mg)
MRI substudy · wk 76 · own-arm −27.8% vs −9.5% (NEJM 2026, 2)

GI Adverse Events — *SYNCHRONIZE-1, 6.0 mg (Ph.3, obesity)*

Nausea	Vomiting	Diarrhea	Constipation	Disc. due to AE
64.9%	**44.6%**	**39.7%**	**32.6%**	**24.8%**

Placebo: nausea 17.4%, vomiting 6.2%, diarrhea 18.6%, constipation 12.4%, discontinuation 5.4% (SYNCHRONIZE-1, Table 3).



How it unfolds over time: Weekly incidence of nausea, by severity, through week 76.

Source: le Roux et al., N Engl J Med 2026 — Suppl. Appendix, Fig. S7 (Panel A shown).

Liver-fat MRI-PDFF assessed at screening, week 28 and week 48. Sources: le Roux et al., NEJM 2026 (SYNCHRONIZE-1, Ph.3); le Roux et al., Lancet Diabetes Endocrinol 2024 (Ph.2 dose-finding, obesity); Sanyal et al., NEJM 2024;391:311–319 (week-48 liver fat); Alkhouri et al., Gastroenterology 2024 (DDW 2024 abstract 1057a; pre-planned week-28 interim analysis, n=138).

MetaVia

18 Competitive Landscape: Pemvidutide

GLP-1/GCG dual agonist · *Pivotal: MOMENTUM (Ph.2, obesity); IMPACT (Ph.2b, MASH) · 2.4 mg for obesity endpoints; 1.8 mg for liver fat (IMPACT)*

Body Weight

Baseline 104.0 kg

Early — wk 24 (2.4 mg):
−9.7% *placebo-adjusted, same trial as the pivotal figure*

Pivotal — 48 wk (2.4 mg): −15.6%
MASH (IMPACT, 1.8 mg, wk 24): −5.8% vs −0.5% placebo

Waist Circumference

Baseline Not disclosed (2.4 mg)

Early — wk 24 (2.4 mg):
−6.2 cm *interim readout, p<0.001*

Pivotal — Not yet public*
MASH (IMPACT, 1.8 mg, wk 24): −3.5% vs +0.9% placebo

Liver Fat

Baseline 19.0% MRI-PDFF (IMPACT, 1.8 mg; placebo 19.6%)

Wk 24 (1.8 mg) — ≥30% relative liver-fat reduction:
84% of patients vs 20% on placebo (n=212; no earlier MRI timepoint)

Pivotal — wk 24: −57.7% (own-arm; placebo −11.4%)

VAT / ASAT / TAT

VAT: −28.3% (2.4 mg)
*not disclosed · wk 48 — **own-arm***

SAT: −19.5% (subcutaneous, 2.4 mg
*not disclosed · wk 48 — **own-arm***

TAT: not reported

GI Adverse Events — *MOMENTUM, 2.4 mg (Ph.2, obesity)*

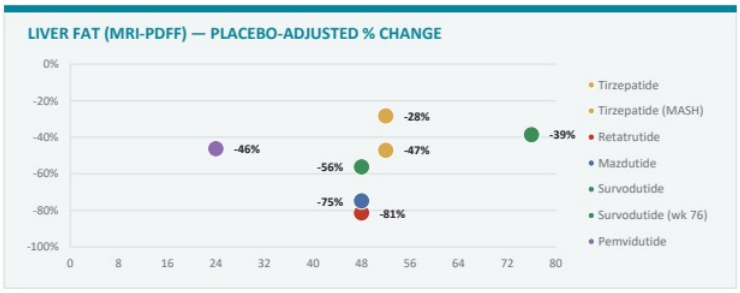
Nausea	Vomiting	Diarrhea	Constipation	Disc. due to AE
51.5%	**27.8%**	**18.6%**	**22.7%**	**19.6%**

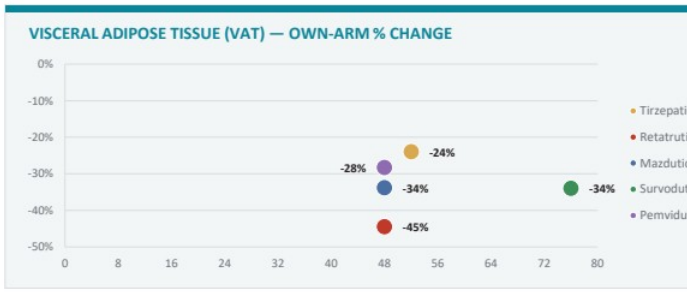
Placebo: nausea 11.3%, vomiting 3.1%, diarrhea 5.2%, constipation 8.2%, discontinuation 6.2% (no titration). MASH (IMPACT, 1.8 mg, wk 24): nausea 41%, diarrhea 21%; disc. for AE 1% vs 2%.

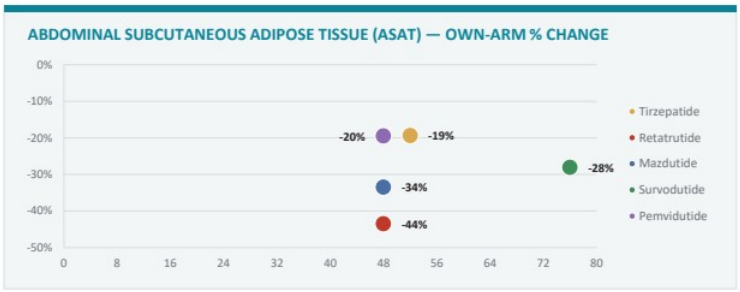
**48-wk waist-circumference data not yet disclosed. Sources: Altimmune press releases — MOMENTUM week-24 interim analysis (Mar 21, 2023: body weight −10.7% vs −1.0%, waist −10.2 vs −4.0 cm); MOMENTUM 48-week topline (Nov 30, 2023: body weight, GI adverse events); MRI body-composition substudy, EASD 2024, as corrected (Sept 10, 2024: VAT −28.3%, SAT −19.5%); IMPACT Ph.2b MASH: Noureddin M, Harrison SA, Loomba R, et al., Lancet, published online Nov 11, 2025 (n=212; baseline MRI-PDFF 19.0% [1.8 mg] vs 19.6% [placebo]; 1.8 mg live −57.7% vs placebo −11.4% at wk 24; ≥30% relative reduction 84% vs 20%; MASH resolution 52% vs 20%; body weight −5.8% vs −0.5%, waist −3.5% vs +0.9%; nausea 41% vs 14%, diarrhea 21% vs 8%, discontinuation for AE 1% vs 2% — Tables 2 and 3); MOMENTUM placebo GI rates: Altimmune topline slides, Nov 30, 2023; MOMENTUM 48-wk: Diabetes 2024;73(Suppl 1):262-OR (ADA 2024 oral).*

Competitive Landscape: Liver Fat, VAT, ASAT & TAT Over Time

Cross-trial comparison — each dot is one reported readout at the comparator dose. Liver fat placebo-adjusted; VAT / ASAT / TAT own-arm. Not head-to-head.









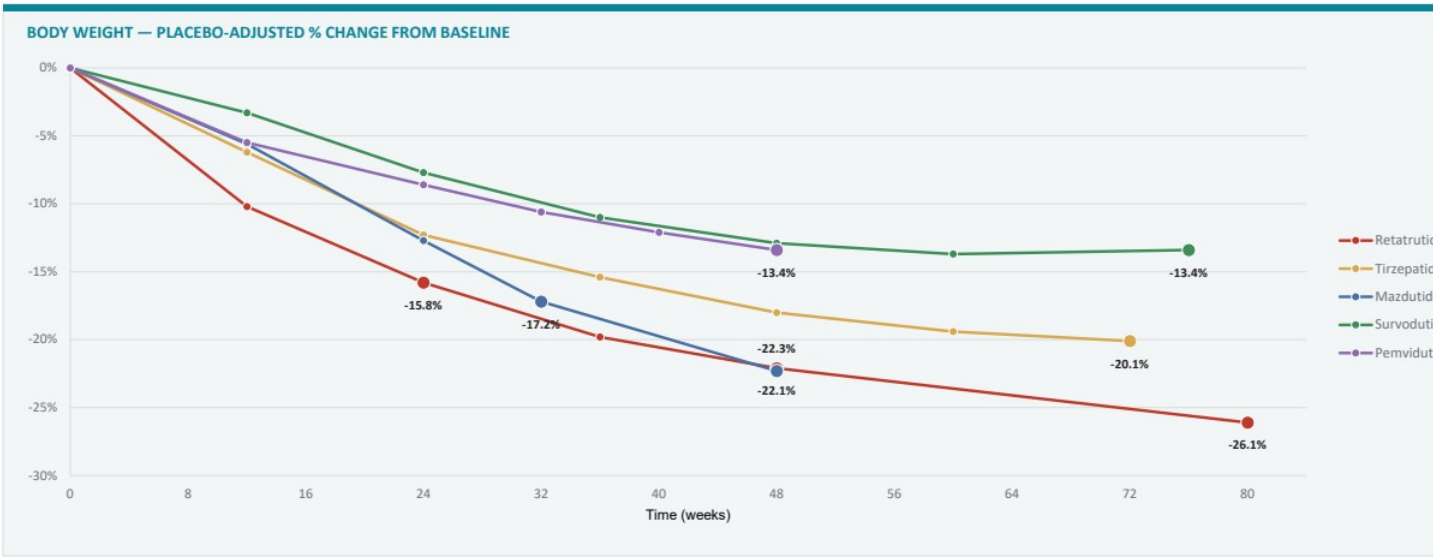
Time axis in weeks. Liver fat: placebo-adjusted relative change — tirzepatide 15 mg wk 52 –28.4 pp (ETD vs degludec, Gastaldelli 2022) and MASH F2–F3 wk 52 –47.2 pp (SYNERGY-NASH, Loomba 2024); retatrutide 12 mg wk 48 –81.4 pp (ETD, Sanyal 2024); mazdutide 6 mg wk 48, LFC ≥10% –74.9 pp (GLORY-1, Ji survodutide 6.0 mg wk 48 MASH –56.3 pp (Sanyal 2024) and wk 76 obesity –38.6 pp (SYNCHRONIZE-1, le Roux 2026); pemvidutide 1.8 mg wk 24 –46.3 pp (IMPACT, Noureddin 2025). Where no placebo-adjusted figure is published the value is drug-arm minus placebo-arm change. VAT / ASAT / TAT: own-arm MRI (pemvidutide placebo values not disclosed) — tirzepatide 15 mg wk 52 (Gastaldelli 2022); retatrutide 12 mg wk 48 (Sanyal 2024; its "TAT" is DXA total body fat, –18.7 pp vs placebo at wk 36 in T2D, Coskun 2025); mazdutide 16 mg wk 48 (Hsia 2026 appendix; "TAT" is total abdominal fat, VAT + SAT); survodutide 6 76 (le Roux 2026 Table 2); pemvidutide 2.4 mg wk 48 (Altimmune, Sept 2024); TAT not reported for tirzepatide or pemvidutide.

MetaVia

MetaVia Inc. — DA-1726 & vanoglipel programs, November 2026

Competitive Landscape: Weight Loss Over Time

Cross-trial comparison — placebo-adjusted % change from baseline at the comparator dose, early and pivotal readouts. Not head-to-head.



All values are placebo-adjusted percentage-point differences at the comparator dose (drug-arm minus placebo-arm change), efficacy estimand where the trial reports both. Large, labeled markers are published values: tirzepatide SURMOUNT-1 15 mg wk 72 −22.5% vs −2.4% = −20 (efficacy estimand; Jastreboff 2022); retatrutide Ph.2 12 mg wk 24 −15.8 pp and wk 48 −22.1 pp (Jastreboff 2023), wk 80 TRIUMPH-1 −28.3% vs −2.2% = −26.1 pp (Lilly, May 2026); mazdutide 16 mg wk 32 −18.1% vs −0.9% = −17.2 pp and wk 48 −22.3 pp (efficacy estimand; Hsia 20 survodutide 6.0 mg wk 76 −16.6% vs −3.2% = −13.4 pp (efficacy estimand; SYNCHRONIZE-1, le Roux 2026); pemvidutide MOMENTUM 2.4 mg wk 48 −15.6% vs −2.2% = −13.4 pp (Altimmune 2023). Small markers are intermediate values read from the published weight-over-time fig the same trials (SURMOUNT-1 Fig. 1B; retatrutide Ph.2 Fig. 1A; Hsia 2026 Fig. 2A, observed means; SYNCHRONIZE-1 Fig. 1B; MOMENTUM week-48 topline presentation, Nov 2023); they are approximate (±0.5 pp) and shown to illustrate trajectory only.

MetALD – A New Frontier with High Unmet Patient Need

10.7M

U.S. adults living with MetALD: 4.1% of the adult population.

0 Approved Treatments

No Dedicated Phase 3 Studies

Significant Market Size

Applying published national prevalence to adult populations, an estimated 35–60 million people across the U.S., EU4, UK, Japan and China (company estimate).

Growing

Prevalence has risen **2.5× since 1988–90** (1.6% to 4.1%), track rising obesity, diabetes and alcohol use.

High Mortality and Healthcare Costs

45% higher risk of premature death than healthy abstainers.

Sources: Younossi et al., Lancet Gastroenterol Hepatol 2026;11:495–508 (U.S.); Ho et al., Clin Gastroenterol Hepatol 2025;23:2423–2432 (global meta-analysis); Nakahata et al., Liver Int 2025;45:e70454 (Japan); Lin et al., Hepatol Commun 2025;9:e0785 (China); Schneider et al., Aliment Pharmacol Ther 2024 (UK).

Paik et al., J Clin Gastroenterol 2025.

MetaVia

Vanoglipel (DA-1241) + Resmetirom

Resmetirom leaves most patients without NASH resolution and delivers no weight loss. Vanoglipel addresses both — orally, once daily, with a placebo-like tolerability record in 108 patients — and the effect is synergistic, not additive.

1 The Incumbent Leaves Two Gaps Open

Resmetirom (Rezdiffra) — first approved MASH therapy. Its own label defines what a combination must add.

24–36%
achieve NASH resolution
Phase 3 MAESTRO-NASH, Month 12 — range across doses and two pathologist reads

64–76%
do not achieve resolution
complement of the labeled response rate

23–28%
improve fibrosis ≥1 stage
vs 13–15% on placebo

Weight-neutral
no effect on body weight
weight loss is not a labeled outcome

WHAT THE LABEL SAYS

- THR-β partial agonist, liver-directed (83.8% of T3 response at THR-β vs 48.6% at THR-α)
- Reduces liver fat (MRI-PDFF from Week 16, CAP at Week 52) and ALT/AST from Month 3
- Accelerated approval — continued approval contingent on the confirmatory outcomes trial
- Warnings: hepatotoxicity; gallbladder events; dose-dependent free-T4 decline

THE ALTERNATIVE IS INJECTABLE

- Semaglutide approved for MASH (Aug 2025) — the first incretin with biopsy-proven fibrosis improvement
- Brings weight loss, but at the cost of injection, GI tolerability and the lean-mass debat
- Distribution advantage: for many patients the first MASH drug is the GLP-1 they alrea take
- The field expects combination therapy to be the endgame — the question is which combination

RESMETIROM SAFETY PROFILE — LABEL TABLE 1: ADVERSE REACTIONS, INCIDENCE PER 100 PATIENT-YEARS (placebo / 80 mg / 100 mg)

Diarrhea	Nausea	Pruritus	Vomiting	Constipation	Abdominal pain	Dizziness	Discontinued for AE
14 / 23 / 33	9 / 18 / 15	4 / 6 / 10	4 / 7 / 8	4 / 5 / 8	4 / 5 / 7	1 / 4 / 4	4 / 5 / 8

Any GI adverse reaction: 57 / 73 / 89 per 100 PY. Diarrhea and nausea began early (median onset 6 and 5 days at 100 mg), were mild-to-moderate and were the most common reasons for discontinuation. Vanoglipel adds no mechanistic overlap (GPR119 agonism; TEAEs at placebo-like rates in 108 patients).

Sources: REZDIFFRA (resmetirom) US Prescribing Information, Sections 5, 6.1 (Table 1), 12.1, 12.2, 14 (Table 8); Clinical Trials Arena, "MASH therapy enters a more competitive era" (2026)

2 Vanoglipel Is Already De-Risked in Patients

Phase 2a, NCT06054815 — 16 weeks, randomized, double-blind, placebo-controlled, N=109 presumed MASH

−22.8 U/L
ALT at Week 16
vs −6.7 U/L placebo · 100 mg

−23.0 dB/m
Liver fat (CAP) at Week 16
vs −1.4 dB/m placebo

Week 4
HbA1c already falling
sustained to Week 16 in prediabetic / T2D subgroups

0
discontinuations for TEAE
on any vanoglipel arm (one on placebo)

LIVER INJURY, INFLAMMATION AND FIBROSIS MARKERS

- ALT reduction held regardless of glucose control — a direct hepatic effect, not a glycemic one
- Cell death (CK-18 M30), inflammation (hs-CRP, CCL2) and fibrosis (TIMP-1) markers all improved
- Liver stiffness (VCTE) fell significantly in the DPP4i-combination arm; CAP fell on monotherapy

PROFILE

- Once-daily oral GPR119 agonist; 50 mg and 100 mg studied
- Weight neutral — the weight loss in this program comes from the combination, not from vanoglipel alone
- Adding a DPP4 inhibitor did not add to the ALT effect — the hepatic benefit is vanoglipel's own

SAFETY AND TOLERABILITY — 16 WEEKS, SAFETY POPULATION (N=108)

n (%)	PBO N=32	50 mg N=14	100 mg N=26	100 mg + DPP4i N=36	All N=108
Any TEAE	18 (56.3)	9 (64.3)	16 (61.5)	22 (61.1)	65 (60.2)
Treatment-related TEAE	9 (28.1)	4 (28.6)	9 (34.6)	10 (27.8)	32 (29.6)
Severe TEAE	0	2 (14.3)*	0	0	2 (1.9)
Serious TEAE	0	0	0	1 (2.8)†	1 (0.9)
TEAE leading to drug discontinuation	1 (3.1)	0	0	0	1 (0.9)
Deaths	0	0	0	0	0

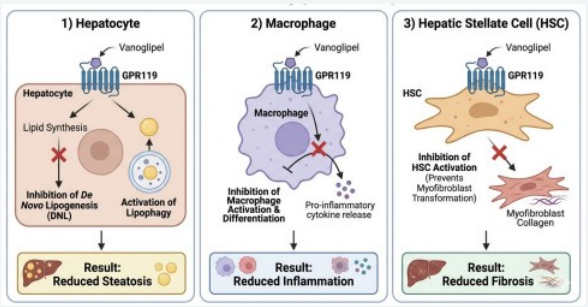
- Mostly mild GI events, at placebo-like rates — diarrhea 15.6% on placebo vs 3.8–14.3% on vanoglipel; nausea constipation and decreased appetite next most common
- † One SAE — vertigo on Day 23 (100 mg + DPP4i), resolved the next day, drug continued, assessed not related Two severe TEAEs (50 mg): ureterolithiasis and a limb injury, both unrelated
- No hypoglycemia reported. No Hy's law case: no ALT/AST ≥3× ULN with bilirubin ≥1.5× ULN. No treatment-re SAE.

Vanoglipel Phase 2a (NCT06054815), 2:1:2:2 randomization, 16 weeks; safety population N=108. Sources: Phase 2a study results (company data); Loomba et al., AASLD The Liver Meeting 2025 (#4012). One additional SAE (atrial fibrillation) occurred at screening before any study d and is not a TEAE.

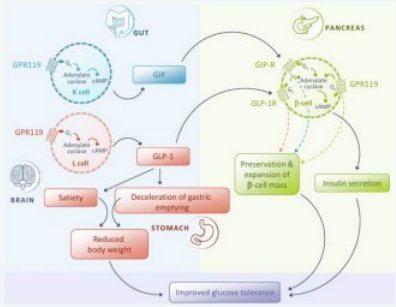
3 Two Mechanisms That Do Not Overlap

Vanoglipel acts on the cells that drive injury, inflammation and scarring; resmetirom acts on hepatic lipid handling

VANOGLIPEL — GPR119, DIRECT CELLULAR ACTION IN LIVER



VANOGLIPEL — EXTRAHEPATIC CONTEXT



RESMETIROM — THR-β, HEPATIC LIPID HANDLING

- Liver-directed THR-β partial agonist; THR-β is the dominant thyroid receptor in hepatocytes
- Drives hepatic fatty-acid oxidation and reduces intrahepatic triglycerides
- Leaves macrophage activation and stellate-cell fibrogenesis largely unaddressed

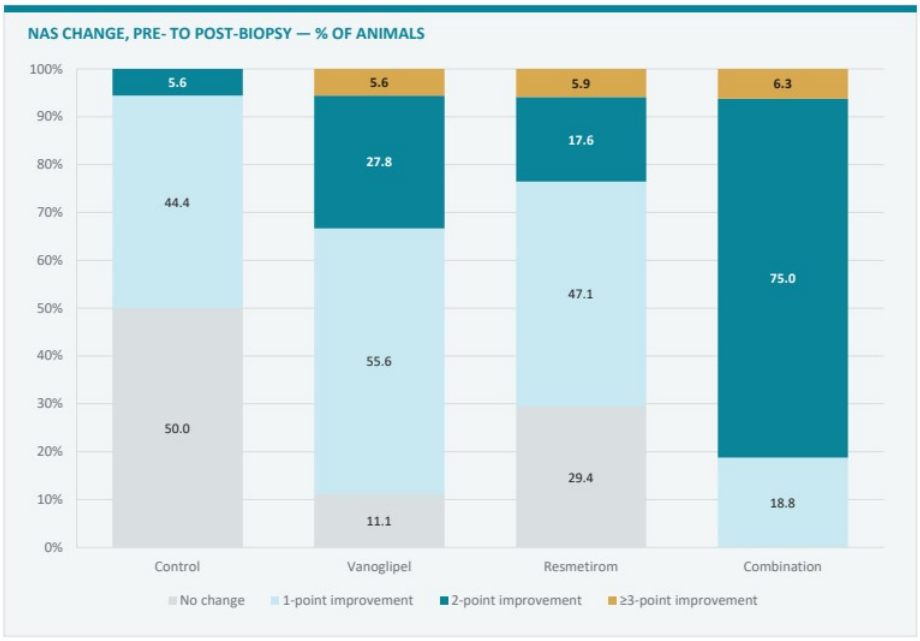
WHY THEY SHOULD ADD UP

- Different receptors, different cell types, different disease drivers
- Vanoglipel supplies the anti-inflammatory and anti-fibrogenic action resmetirom lacks
- Resmetirom supplies the potent steatosis reduction — and in combination, adipose tissue is recrui too
- Liabilities do not overlap either: vanoglipel showed no hepatotoxicity, gallbladder or thyroid-axis si in 16 weeks

Mechanism panels: Dong-A ST. Resmetirom pharmacology per USPI Section 12.1

4 Synergy I: the Combination Improved NAS in Every Mouse Treated

Resmetirom alone barely moves NAS. GAN DIO-MASH mice, paired pre-/post-treatment biopsy, 16 weeks, n=16–18 per group



100%

of combination animals improved ≥1 point

vs 50.0% control · 70.6% resmetirom · 88.9% vanoglipel

81%

of combination animals improved ≥2 points

vs 5.6% control · 23.5% resmetirom · 33.3% vanoglipel

MEAN NAS IMPROVEMENT

Combination	**+1.88**
Vanoglipel alone	**+1.28**
Resmetirom alone	**+1.00**
Control	**+0.56**

GAN DIO-MASH study (Gubra), per-animal data. Resmetirom alone did not improve NAS in the group-level analysis; improvement in all arms was driven mainly by steatosis. One-sided Fisher's exact, Bonferroni. Model-to-clinic translation: see Appendix (Gubra poster).

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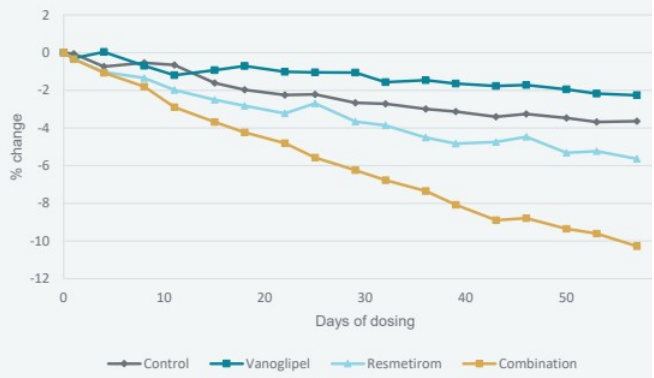
5 Synergy II: Weight Loss Appears Only When the Two Are Combined

Two species. Neither monotherapy reduced body weight; the combination did. Mouse food intake was unchanged; monkey intake fell with dosing

MICE — BODY WEIGHT, % CHANGE FROM BASELINE (16 WEEKS)



OBESE CYNOMOLGUS MONKEYS — BODY WEIGHT, % CHANGE (8 WEEKS)



−23.6%

mice: body weight vs control, day 114

control-adjusted · vanoglipel −3.0% · resmetirom +1.4%

−43.5%

mice: fat mass vs control (Echo MRI)

lean mass −6%; food intake unchanged

−10.3%

monkeys: combination body weight, day 56

from re-dosing baseline · vehicle −3.6% · monotherapies n.s.

−25.9%

monkeys: fat mass, combination

vs −5.4% vehicle · −11.7% vanoglipel · −26.3% resmeti

Mice: GAN DIO-MASH study (Gubra), n=15–18/group. Monkeys: obese cynomolgus study (WuXi), n=3–5/group, re-dosing baseline after a dosing holiday; per-animal % change averaged. Excluded: two resmetirom animals (GI events), one vehicle animal (tail wound). Model-to-clini translation: see Appendix (Gubra poster).

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6 Why It Is Synergistic: a Program Neither Drug Switches On Alone

Adipose tissue is remodeled — smaller, more numerous adipocytes, browning markers up, inflammation down

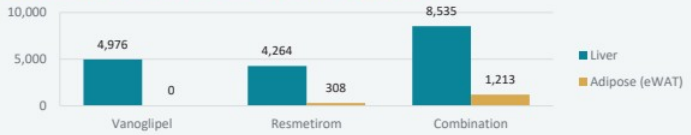
−36%
adipocyte size
µm² per cell, eWAT

+47.5%
adipocyte number
per mm², eWAT

+41%
UCP-1 protein
IHC, % fractional area · trend, p=0.07 (n.s.)

−50%
Galectin-3 (inflammation)
IHC, % fractional area

DIFFERENTIALLY EXPRESSED GENES vs CONTROL (RNA-seq)

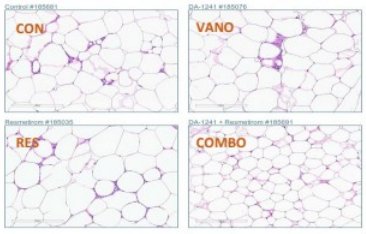


ADIPOSE TRANSCRIPTOME — SIGNIFICANT ONLY IN COMBINATION

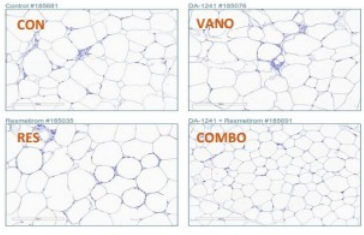
Cidea (browning)	+1.8	Dio2 (thermogenesis)	+2.7
Lgals3 (Galectin-3)	−1.2	Acta2 (α-SMA)	−0.6

log2 fold-change vs control, adjusted p<0.05. Vanoglipel alone: 0 regulated adipose genes; resmetirom alone: 308; combination: 1,213. Liver: 4,976 / 4,264 / 8,535 — the combination regulates roughly double either monotherapy, including a large pool of genes neither regulates alone.

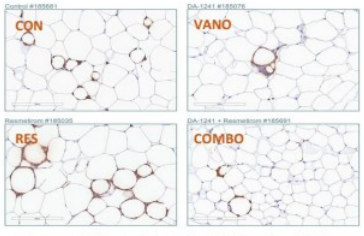
REPRESENTATIVE eWAT HISTOLOGY — H&E · UCP-1 · GALECTIN-3



Representative images of eWAT morphology at termination (magnification 20x, scale bar = 100 µm).



Representative images of eWAT stained with anti-UCP-1 (Abcam, Cat. ab209483) at termination (magnification 20x, scale bar = 100 µm).



Representative images of eWAT stained with anti-Galectin-3 (Biolegend, cat. 125402) at termina (magnification 20x, scale bar = 200 µm).

GAN DIO-MASH study (Gubra): terminal eWAT histomorphometry, n=16–18/group; bulk RNA-seq of liver and eWAT FFPE. UCP-1 shown at protein level (combination vs control, trend p=0.07, not statistically significant); Ucp1 transcript not significant in either tissue. Gene counts at adjusted p<0.05.

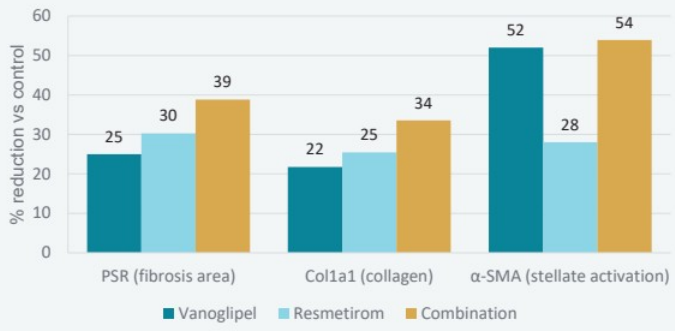
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7 What the Data Show — and What 2027 Will Answer

An honest ledger. The anti-fibrogenic signal is strong at the marker level; the regulatory endpoint is answered in the clinic

FIBROSIS AND FIBROGENESIS MARKERS — % REDUCTION vs CONTROL



HOW TO READ THE THREE MARKERS

PSR (picrosirius red) −39% with the combinat

Collagen area on histology — the quantitative readout of fibrosis burden

Col1a1 −34% with the combinat

Type I collagen protein — deposition of new scar matrix

α-SMA −54% with the combinat

Activated hepatic stellate cells — fibrogenesis in progress, the process vanoglipel targets directly

All three moved further with the combination than with either drug alone; each is steatosis-adjusted so the fibrosis signal is not an artifact of fat clearance. Same 16-week study and endpoints as presented at ADA 20

DEMONSTRATED

- Greater NAS improvement than either drug alone; all 16 combination-treated mice improved ≥1 point
- Weight and fat-mass loss in two species; mouse food intake unchanged
- Vanoglipel, 108 patients: placebo-like TEAE rates (56–64%), no related SAE, no discontinuations on the vanoglipel monotherapy arms
- Reduced fibrosis area, collagen and stellate activation; combination-unique adipose transcriptome

NOT YET DEMONSTRATED

- Histologic fibrosis-stage improvement — the regulatory endpoint, which the clinical program is designed to test
- Any efficacy or tolerability of the combination in humans (Phase 2a POC planned 1H 2027)
- Lean-mass sparing beyond control: combination −6.0% (mice) / −6.5% (monkeys) vs control −6%

MetaVia nonclinical studies — GAN DIO-MASH mice (Gubra) and obese cynomolgus monkeys (WuXi). All combination comparisons nonclinical.

Vanoglipel + Resmetirom: Potential for Industry-Leading Combo Therapy

Two direct liver MOAs in one once-daily oral fixed-dose combination, with weight-loss potential

TARGET PRODUCT PROFILE

Target Indication	MASH (F2/F3 +/- F4)		
Class	vanoglipel (GPR119 agonist) + resmetirom (THR-β agonist) combination therapy		
Mode of Action	**vanoglipel (GPR119)-mediated** • Inhibition of de novo lipogenesis • Inhibition of macrophage differentiation and inflammatory activation • Inhibition of TGFβ-induced hepatic stellate cell activation **resmetirom (THR-β)–mediated** • Improvement of hepatic lipid metabolism		**Synergistic Mechanisms** • Reduction of steatosis, inflammation, and fibrosis • Suppression of Col1a1, Col3a1, Ccl2, Lgals3 • White adipose remodeling with ↑UCP-1, ↑Cidea, ↑Dio2
End Points	**Primary**: NASH resolution with no worsening of fibrosis stage, and separately, ≥1-stage improvement in liver fibrosis with no worsening of NASH activity **Secondary**: MRI-PDFF, VCTE, ELF, FAST, NIS-4, body weight % change, waist circumference, body composition (fat vs. lean mass), HbA1c		
Route of Administration	Daily oral fixed-dose combination therapy		
Efficacy	Superior fibrosis and metabolic efficacy to resmetirom monotherapy		
	Histology	• Fibrosis improvement (≥1 stage): ≥ 40% • Fibrosis improvement (≥2 stages): ≥ 20%	• Inflammation reduction target ≥30% • MASH resolution target ≥ 50%
	Metabolic	• Weight loss up to 10% (fat-selective loss) • HbA1c: ~1.0 pp ↓	• TG: 50–70% ↓ • LDL-C: 30–40% ↓ • T-CHO: 50–60% ↓ • HDL-C: 20–30% ↑
Safety and Tolerability	• No titration, well-tolerated with GI safety profile similar to resmetirom • Minimal risk of hypoglycemia • No need for dose adjustment even in patients with impaired renal function • Potential for improvement in cardiovascular outcomes (non-fatal stroke, non-fatal myocardial infarction, and cardiovascular death)		

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9 From Combination Data to a Single Oral Tablet

The regulatory, clinical, safety, supply and IP steps between today's nonclinical synergy and a fixed-dose combination for MASH

REGULATORY

- FDA meeting on the combination data — H2 2026
- To settle: need for a combination toxicology study; biopsy requirement in Phase 2b
- Vanoglipel carries the new-molecule package; resmetirom's approved label supports the THR-β component

CLINICAL

- Phase 2a: 12-week, non-biopsy POC, ~100 patients, start 1H 2027 — modeled on the vanoglipel Phase 2a
- Phase 2b / 3 with the fixed-dose combination
- Target: NDA submission 2031

SAFETY — WHAT TO EXPECT

- Vanoglipel, 16 wk, N=108: TEAEs at placebo-like rates (56–64% across arms), no related SAE, no hypoglycemia, no Hy's law case
- Resmetirom label: GI events most common; hepatotoxicity and gallbladder warnings — no mechanistic overlap with GPR119
- Combination watch-points: additive GI tolerability; liver enzymes tracked as both efficacy and safety

SUPPLY & FORMULATION

- Resmetirom sourced commercially for clinical use; co-administered in Phase 2a
- Single-tablet FDC formulation developed during 2027 for Phase 2b / 3
- Once-daily oral, no renal dose adjustment, no hypoglycemia risk

INTELLECTUAL PROPERTY

- Vanoglipel composition-of-matter (2017) and MASH u (2018) patents
- MASH combined-use pater (2021); provisional for THR combined use in MASH / metabolic disease (2026)
- A single-tablet FDC could a up to 20 years of exclusivit

PATH TO A FIXED-DOSE COMBINATION

H2 2026	Late 2026 – Q2 2027	1H 2027	2027	2028 →	2031
FDA meeting on combination data	Combination toxicology study (if required)	Phase 2a POC — start	FDC formulation for Phase 2b / 3	Phase 2b / 3 with the FDC	NDA submission target

Patents held by Dong-A ST and licensed to MetaVia; Korean registrations 10-1726819, 10-2295300, 10-2538047, provisional 10-2026-0058472. Regulatory route and study requirements subject to FDA feedback. Milestones assume regulatory and clinical success, which is not guaranteed.

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10

A Large and Growing US Patient Opportunity for the Combination

Independent, patient-based assessment of the vanoglipel + resmetirom fixed-dose combination in MASH — US patients, launch 2032

US MASH PATIENT FUNNEL AT PEAK (2042) — WHERE THE COMBINATION FITS

~22M — **US adults with MASH**
prevalence ~19M in 2026, growing ~1% a year

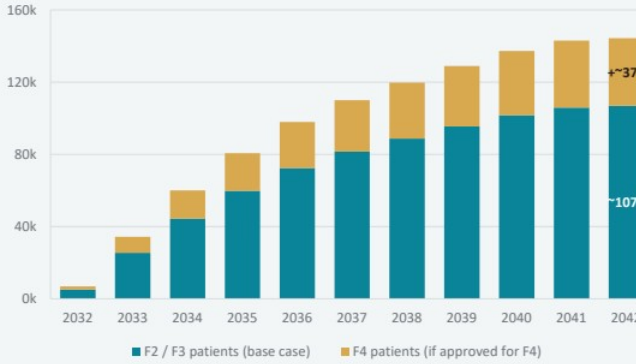
~8.9M — **diagnosed**
diagnosis rate rising from 13% today to 40% by 2034

~4.5M — **F2 / F3 fibrosis — the label population**
50% of diagnosed patients; a further ~0.9M (10%) are F4

~380,000 — **on a liver-directed therapy**
of ~1.7M F2 / F3 patients in specialist care; treatment rates 20% (F2) and 25% (F3) at peak

~107,000 — **on the vanoglipel FDC (base case)**
25% of F2 and 30% of F3 liver-directed patients; ~145,000 with F4 approval

PATIENTS ON THERAPY, 2032–2042 (THOUSANDS) — BASE CASE AND F4 UPSIDE



WHAT DRIVES THE FORECAST

13% → 40%
MASH diagnosis rate, today → 2034 (FIB-4 screening in T2D, non-invasive tests, referral pathways)

20% / 25%
F2 / F3 patients in specialist care on a liver-directed therapy at peak — up from ~10% today

25% / 30%
peak share of F2 / F3 liver-directed patients, with four competing liver-directed classes

10 years
ramp to peak from a 2032 launch, in line with oral fixed-dose combination analogs

Oral · fibrosis-first
KOLs: fibrosis improvement well above resmetirom, fully oral where F2 patients resist injectables, two validated mechanisms

Source: Independent US market assessment commissioned by MetaVia (revised September 24, 2026): six KOL and payer interviews, syndicated epidemiology and EMR data, EvaluatePharma analogs. Patient numbers are the assessment's base-case, patient-based model outputs; the F4 figures apply its F4-approval scenario (15% peak share). Forward-looking projections — actual results depend on clinical outcomes, approval, pricing and competition and are not guaranteed.

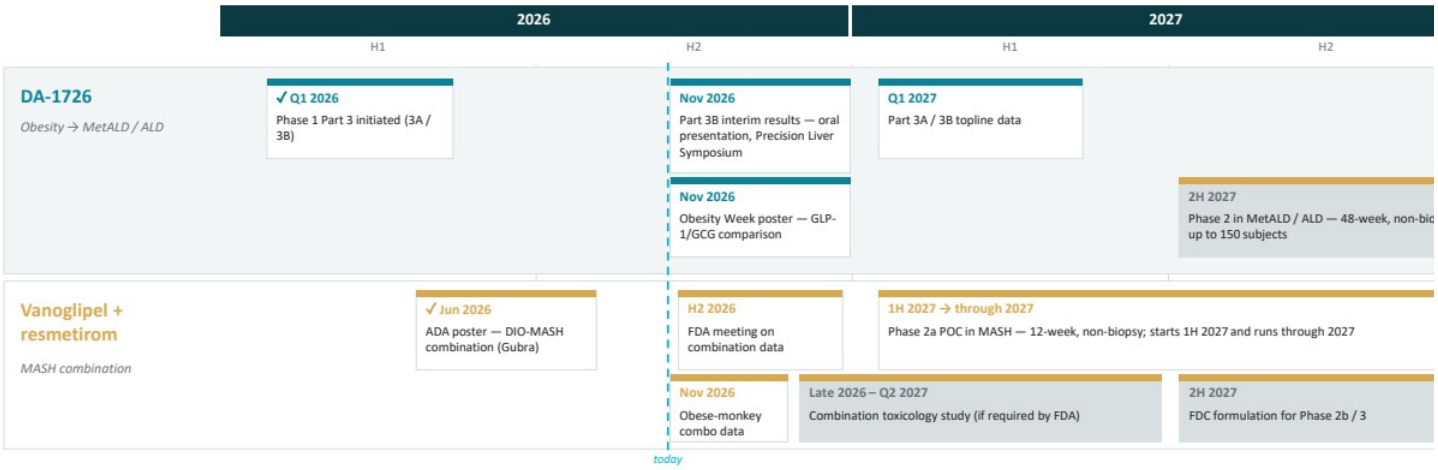
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Timeline and Financials

Near-term catalysts across both programs — DA-1726 obesity readouts and the vanoglipel + resmetirom combination path.

1 Multiple Near-Term Catalysts to Drive Shareholder Value

Both programs — DA-1726 obesity readouts and the MetALD / ALD start, then the vanoglipel + resmetirom combination path through 2027



DA-1726 — NEXT STEPS

Part 3B interim results — oral presentation at the Precision Liver Symposium — and Obesity Week poster — Nov 2026 · Complete Part 3A / Part 3B topline data — Q1 2027 · Phase 2, 48-week, non-biopsy MetALD/ALD study, up 150 subjects — start 2H 2027

VANOGLIPEL + RESMETIROM — NEXT STEPS

FDA meeting on combination data — H2 2026 · Combination tox study — late 2026 – Q2 2027 · Phase 2a, 12-week non-biopsy POC study — start 1H 2027 · Fixed-dose combination formulation for Phase 2b/3 — 2H 2027

Milestones assume regulatory and clinical success, which is not guaranteed · Gray boxes are prospective future studies; timing and occurrence subject to various factors

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2 Financial Overview and Capitalization

Balance sheet and capitalization as of June 30, 2026, and where capital is directed

$12.6M	Into 1Q 2027	6.58M	26.8%
cash and cash equivalents	**expected cash runway**	**common shares outstanding**	**held by Dong-A ST and affiliate**
as of June 30, 2026 · no debt	*based on the current operating plan*	*15.77M fully diluted*	*1,766,572 shares · licensor of both assets*

The 2027 clinical programs are contingent on additional financing.

CAPITALIZATION TABLE — JUNE 30, 2026

	Common stock equivalents
Common stock	6,575,656
Warrants (WAEP $7.94) [1]	8,959,788
Options (WAEP $4,240.22)	420
Restricted stock units	234,055
Fully diluted	**15,769,919**

(1) Includes (i) 2026 Series C warrants to purchase 4,478,322 shares, expiring January 2031, exercise price $3.10; (ii) 2026 Series D warrants to purchase 3,758,822 shares, expiring January 2028, exercise price $3.10; (iii) 2024 Series B milestone-based warrants to purchase 693,962 shares, expiring September 2029, exercise price $43.23; (iv) 2024 placement agent warrants to purchase 11,564 shares, expired July 2026, exercise price $54.0375; (v) 2022 Series B warrants to purchase 16,176 shares, expiring December 2027, assumed exercise price $0.00; and (vi) 2021 warrants to purchase 942 shares, expired July 2026, exercise price $15,919.20. No ratchets, price resets or anti-dilution provisions.

CAPITAL PRIORITIES

DA-1726

- Complete Part 3A / 3B — topline Q1 2027; Part 3B interim oral presentation (Precision Liv Symposium) and Obesity Week, Nov 2026
- Prepare the 48-week, non-biopsy Phase 2 in MetALD / ALD — start 2H 2027

VANOGLIPEL + RESMETIROM

- FDA meeting H2 2026; Phase 2a proof-of-concept — start 1H 2027
- Combination toxicology (if required) and single-tablet FDC formulation for Phase 2b / 3

CORPORATE

- Lean operating model — CROs, development partners and Dong-A ST; disciplined G&A
- Business-development optionality on both programs as data mature

Cash, share and warrant data per company records as of June 30, 2026. Runway estimate based on current operating plan. Milestones assume regulatory and clinical success, which is not guaranteed.

Positioned for Value Creation

Two assets on one liver continuum, both with human data, both with a defined next study and a date

WHAT WE HAVE

- DA-1726: −68% liver fat, −13.5% weight and −0.24 pp HbA1c, all placebo-adjusted, at Day 109, with no new GI adverse events after Week 9
- Vanoglipel: ALT, liver fat and inflammation markers improved in 109 patients; weight-neutral; placebo-like adverse-event rates, no discontinuations on monotherapy arms
- Combination: greater NAS improvement than either drug alone; weight loss only in combination, in two species

WHAT COMES NEXT

- H2 2026 — FDA meeting on the combination · Nov 2026 — Part 3B interim oral presentation (Precision Liver Symposium) and Obesity Week poster
- Q1 2027 — DA-1726 Part 3A / 3B topline
- 1H 2027 — combination Phase 2a start · 2H 2027 — DA-1726 Phase 2 in MetALD / ALD

WHY IT MATTERS

- MetALD is a 10.7M-patient US indication with nothing approved and no Phase 3 program
- The MASH incumbent has a $1B+ run-rate and building its own combination — the thesis is validated by the leader
- Both programs are liver-directed and differentiated from the weight-only incretin fi — one oral, one once-weekly

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Milestones assume regulatory and clinical success, which is not guaranteed. Combination data are nonclinical.



Appendix

Preclinical efficacy and clinical translatability of resmetirom in the GAN diet-induced obese and biopsy-confirmed mouse model of NASH

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BACKGROUND & AIM

Resmetirom (MGL-3196), a selective THR-β agonist, has been reported to improve histopathological outcomes in a recent 52-week phase-3 clinical trial (MAESTRO-NASH) in patients with non-alcoholic steatohepatitis (NASH). The present study aimed to //) evaluate the metabolic, biochemical and histopathological effects of resmetirom treatment in the Gubra-Amylin NASH (GAN) diet-induced obese (DIO) mouse model of fibrosing NASH; and //) compare to primary outcomes in the MAESTRO-NASH trial.

1 Study outline



Group	Animal	Gender	Number of animals	Treatment	Administration route	Dosing Frequency	Dose
1	LEAN-CHOW	Male	10	**Vehicle**	PO	QD	-
2	DIO-NASH	Male	16	**Vehicle**	PO	QD	-
3	DIO-NASH	Male	15	**Resmetirom**	PO	QD	3mg/kg

2 Metabolic and biochemical markers

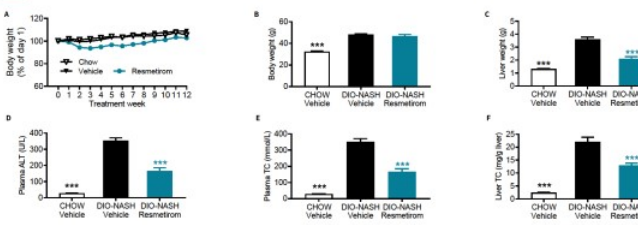


Figure 1. Resmetirom is weight-neutral and improves both hepatomegaly and biochemical markers in GAN DIO-NASH mice. **(A)** Body weight change relative to baseline (day 0). **(B)** Terminal body weight (gl). **(C)** Liver weight. **(D)** Plasma alanine aminotransferase (ALT). **(E)** Plasma total cholesterol (TC). **(F)** Liver total cholesterol (TC). ***p<0.001 compared to corresponding DIO-NASH vehicle control (Dunnett's test one-factor model).

3 NAFLD Activity Score and Fibrosis Stage

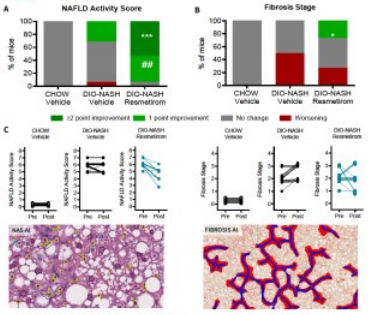


Figure 2. Resmetirom improves liver histopathological scores in GAN DIO-NASH mice. Histopathological scores were determined by Gubra Histopathological Objective Scoring Technique (GHOST) deep learning-based image analysis. **(A)** NAFLD Activity Score (NAS). **(B)** Fibrosis Stage. **(C)** Comparison of individual pre-post NAS and individual pre-post Fibrosis stage. Bottom panels: Representative HE and PSR photomicrographs used for GHOST evaluation. ##p<0.01 with 1 point improvement, *p<0.05 ǁ ≥1 stage fibrosis improvement, ***p<0.001 with more than 2-point improvement compared to corresponding DIO-NASH vehicle group (One-sided Fisher's exact test with Bonferroni correction). Bottom panels: Representative HE and PSR photomicrographs used for GHOST evaluation.

4 Quantitative histological markers of steatosis, inflammation and fibrosis

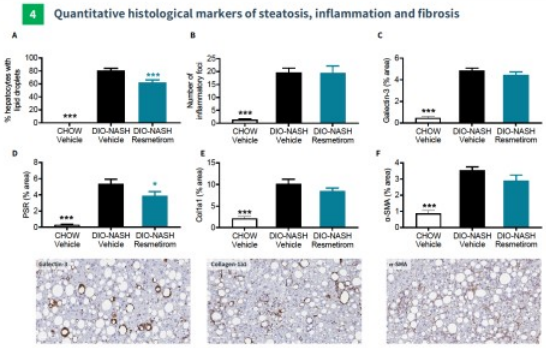


Figure 3. Resmetirom improves quantitative liver histological markers in GAN DIO-NASH mice. Histomorphometric assessments were performed by GHOST deep learning-based image analysis on scoring-associated variables (panels A-B) and conventional IHC image analysis (panels C-F). **(A)** % hepatocytes with lipid droplets. **(B)** Number of inflammatory foci. **(C)** % area of galectin-3. **(D)** % area of PSR. **(E)** % area of collagen-1a1. **(F)** % area of alpha-smooth muscle actin (a-SMA) as marker for stellate cell activation. Mean ± SEM. *p<0.05, ***p<0.001 to corresponding DIO-NASH vehicle group (Dunnett's test one-factor linear model). Bottom panels: Representative photomicrographs (scale bar, 100 µm).

5 Clinical translatability

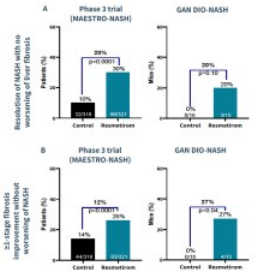


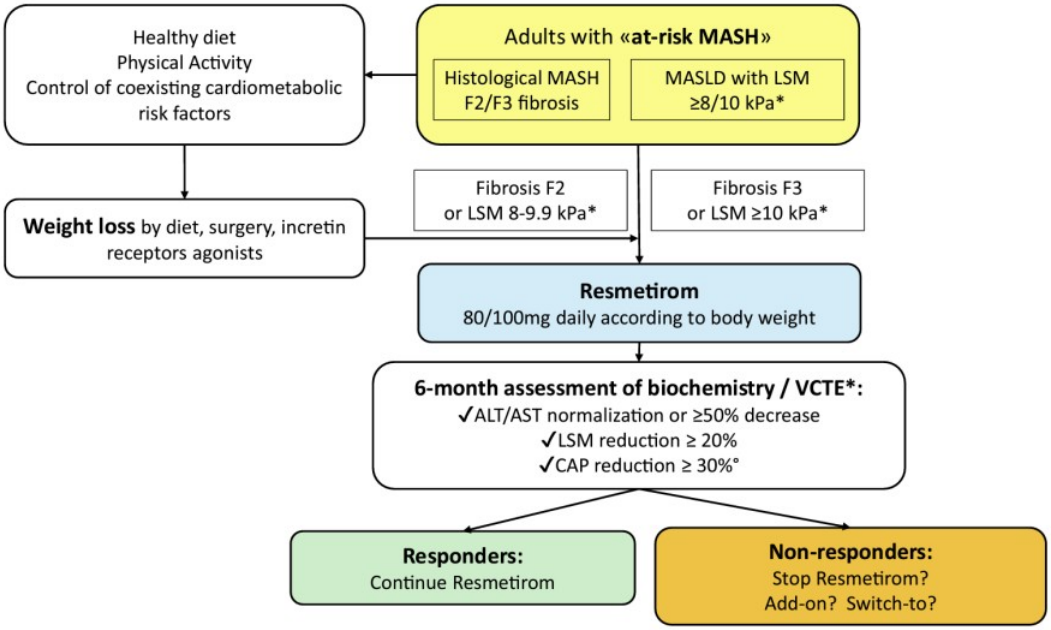
Figure 4. Resmetirom promotes NASH resolution and improves fibrosis stage in both GAN DIO-NASH mice and NASH patients. **(A)** Resolution of NASH (inflammation score ≤1; hepatocyte ballooning=0, with at least a 2-point reduction in NAS) with no worsening of liver fibrosis in GAN DIO-NASH mice compared to clinical phase-3 trial data (MAESTRO-NASH, press release Dec 19, 2022). **(B)** ≥1-stage fibrosis improvement without worsening of NASH in GAN DIO-NASH mice compared to the MAESTRO-NASH trial.

CONCLUSION

- Resmetirom reduces hepat ALT levels, and plasma/liver levels.
- Resmetirom promotes ≥2-p improvement in NAFLD Acti
- Resmetirom promotes 1-po improvement in Fibrosis Sta
- Resmetirom reduces quanti markers of steatosis and fib
- Resmetirom demonstrates efficacy on primary endpoi patients and GAN DIO-NASH
- These findings highlight the translatability of the GAN D model.

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Potential Algorithm to Manage Resmetirom Treatment in Clinical Practice



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