MetaVia Inc.

Transforming Cardiometabolic Diseases

November 2026

Nasdaq: MTVA

Forward Looking Statements

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 that do not 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”, “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). 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. These forward-looking statements 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 as weight 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 candidates; our 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 with the U.S. 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, the sufficiency of our existing cash on hand to fund our operations and raising additional capital; adverse global economic conditions; 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 the license 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 business in 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; potential negative interactions between our product candidates and other products for which they are combined for treatment; our ability to initiate and complete clinical trials on a timely and in a cost-efficient manner; our 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 relating to the 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 to obtain, maintain, protect and/or enforce sufficient intellectual property rights. These forward-looking statements are based on 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 actual results, 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 Annual Report on Form 10-K for the year ended December 31, 2025, and our other filings with the Securities and Exchange Commission. 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 forward-looking statements should not be relied upon as representing our views as of any date subsequent to 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 assumptions 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 markets in which we operate are necessarily subject to a high degree of uncertainty and risk.

Disclaimers & Risk Factors (cont'd..)

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 based on 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, duration, 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 conclusions regarding the relative safety or effectiveness of DA-1726 or vanoglipel should be drawn from any comparisons presented in this Presentation.

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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

−68.1%

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 for MASH: better NAS than either drug alone; weight loss only in combination. Phase 2a start 1H 2027.

FIVE CATALYSTS IN FIFTEEN MONTHS

H2 2026
FDA meeting on the combination data

Nov 2026
Part 3B interim results — oral presentation, Precision Liver Symposium; Obesity Week poster

Q1 2027
DA-1726 Part 3A / 3B topline

1H 2027
Combination Phase 2a in MASH — start

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

NASDAQ	MTVA	CASH, SEPTEMBER 30, 2026	$8.8M · no debt	LARGEST HOLDER	Dong-A ST, 26.2%	HEADQUARTERS	Massachusetts, USA

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.

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*

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 is itself building a combination. An oral, once-daily FDC that adds both builds on an established prescriber and payer base.

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 (−68.1% liver fat at Day 109 in obesity).

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 2027**

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 guaranteed.

MetaVia

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; 20+ years working with life-science investors and analysts
- CFO of Nevakar, Braeburn Pharmaceuticals, Aerocrine and Furiex Pharmaceuticals
- MBA, Indiana University

Biographies as of November 2026.

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

Steatotic Liver Disease (SLD) · Steatosis on imaging or histology

≥1 cardiometabolic risk factor

Overweight or obesity · Type 2 diabetes or prediabetes
High blood pressure · High triglycerides · Low HDL cholesterol

Stratified by alcohol use

MASLD	MetALD	ALD
Metabolic Dysfunction-Associated SLD	Metabolic & Alcohol-Associated SLD	Alcohol-Associated Liver Disease
ALCOHOL USE: **Little**	ALCOHOL USE: **Moderate**	ALCOHOL USE: **Heavy**
MASH is a subset of MASLD		

AN EXAMPLE: GLASSES OF WINE A DAY

	MASLD	MetALD	ALD
Men	up to 2 a day	~3 to 4 a day	~5 or more
Women	about 1 a day	~2 to 3 a day	~4 or more

Sources: Rinella ME, et al., 2023 Multi-Society Delphi Nomenclature, Hepatology 2023;78(6):1966–1986

One glass = 5 oz of wine, about 14 g of alcohol; counts are approximate.

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

5.6–5.8M

2.14–2.20% of US adults

NHANES self-report, uncorrected — Paik et al. 2025; Younossi et al. 2026 (uncorrected arm)

COMPANY ESTIMATE

10.7M

4.10% of US adults

NHANES self-report corrected & calibrated to NIAAA per-capita sales data — Younossi et al., Lancet Gastroenterol Hepatol 2026

CEILING

22–33M

8–13% of US adults

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 meet MetALD/ALD alcohol thresholds once biomarkers are used.

International pattern: the same survey-vs-sales gap shows up outside the US — England ~60% coverage 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, roughly 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 & Alpert, April 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 (PEth 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).

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 readout; 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

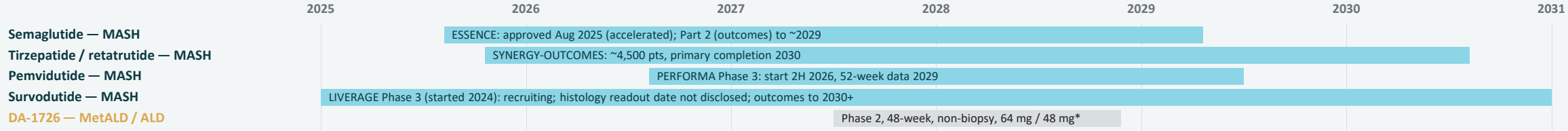
Strongest liver signal in the incretin class. Efinopegdutide beat semaglutide head-to-head on liver fat (72.7% vs 42.3%); survodutide's fibrosis improvement (up to 64.5% vs 25.9%, F2–F3) is among the largest reported.

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; −68.1% placebo-adjusted liver fat at 16 weeks, no discontinuations and no new GI adverse events 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 channel 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 9); 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.*

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

SCREENING

MetALD / ALD by non-invasive testing: liver fat on MRI, liver stiffness on FibroScan, alcohol intake confirmed by PEth

→

RANDOMIZE

up to 150 participants
1:1:1
double-blind

→

DA-1726 64 mg once weekly, titrated

DA-1726 48 mg once weekly, titrated

Placebo once weekly

→

WEEK 24

Primary endpoint
Liver fat (MRI-PDFF), relative change from baseline

→

WEEK 48

Key secondary
Liver stiffness (FibroScan)
End of treatment; 4-week follow-up

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 to enroll — no biopsies
- Week 24 liver-fat primary reads early; Week 48 stiffness speaks to fibrosis
- Drinking measured objectively (PEth) alongside self-report — reduction, not abstinence
- Two doses allow a dose-response read on the 64 mg target dose

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.

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: −68.1% 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.

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

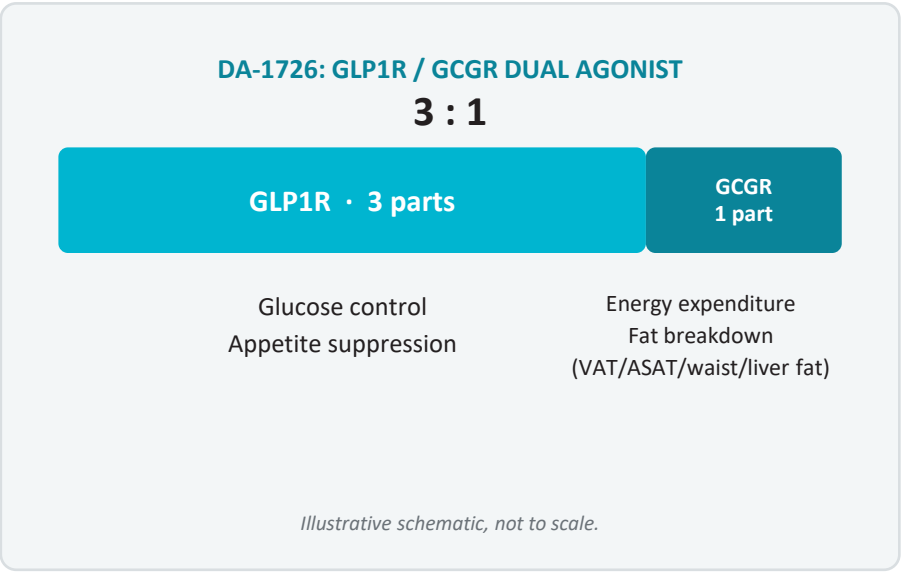
WHAT IS DA-1726?

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 fat, thigh muscle)
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

All DA-1726 figures in this deck are from the Part 3B Day 109 interim dataset presented at the Precision Liver Symposium in November 2026; complete Part 3A and Part 3B topline data are expected in Q1 2027. DA-1726's separate, completed Phase 1 MAD trial (2024, single/multiple ascending doses to 48 mg) is not combined with Part 3B data anywhere in this deck.

2 Baseline Characteristics

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)

Part 3B safety population — DA-1726 (n=15) vs. placebo (n=4); Day 109 interim analysis. Mean (SD) unless noted; 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

-68.1%

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

5.2%

Baseline liver fat (MRI-PDFF)

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

-68.1%

Placebo-adjusted (GMR)

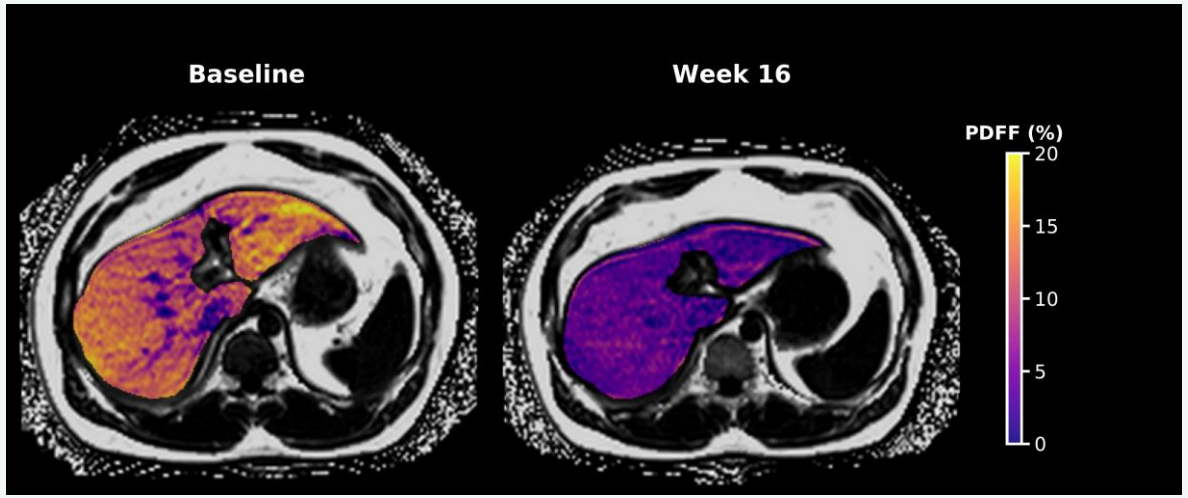
GMR 0.32, Day 109; **p<0.001**

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 of 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 −68.1% 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% fat, purple is 0–5% (scale at right). At baseline the liver is largely orange — steatotic; at Week 16 it is uniformly 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 anatomical 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, HbA1c and Uric Acid

BODY WEIGHT

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%; placebo (n=4): +1.35 kg, +1.6%.

-13.5%

Placebo-adjusted

Difference in mean % change vs. placebo, n=15 vs. 4: −13.5 pp (−13.5 kg); two-sample t-test, MI analysis; **p<0.001**

HBA1C

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 (n=4): −0.20 pp.

-0.24 pp

Placebo-adjusted

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

URIC ACID

Produced in the liver; rises with hepatic fat, insulin resistance and alcohol intake

5.89 mg/dL

Baseline serum uric acid

DA-1726 arm mean, n=15 (range 4.5–7.9; placebo 4.80, n=4). Normal range <7.0 mg/dL; no participant selected for gout or hyperuricemia.

−1.02 mg/dL

Change from baseline, Day 109

DA-1726 arm (n=15): −1.02 mg/dL (**p<0.001**). Every participant with a Day 109 value was below 7.0 mg/dL (range 3.4–6.9). Placebo arm (n=4): +0.45.

−1.47 mg/dL

Placebo-adjusted

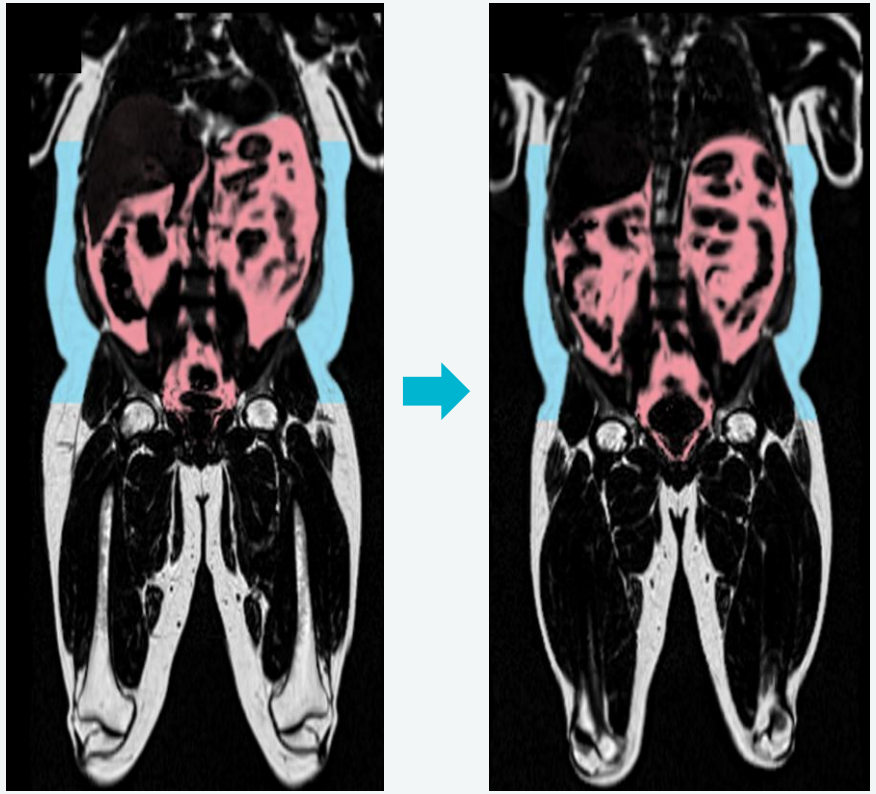
Difference in mean change vs. placebo, n=15 vs. 4: −1.47 mg/dL (95% CI −2.0 to −0.9); two-sample t-test, MI analysis; **p<0.001**

HbA1c effect observed in a population without diabetes (fasting glucose 88.5 mg/dL at baseline); HbA1c is a secondary endpoint in the planned Phase 2 MetALD / ALD study. Waist circumference: see Body Composition slide. Uric acid: normal range <7.0 mg/dL; a lower level from a normal baseline is relevant to the planned MetALD / ALD population, where hyperuricemia is common. Changes from baseline and differences vs. placebo use the multiple-imputation analysis; ranges are observed values.

6 Body Composition: Fat vs. Lean Mass

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

Baseline | Day 109 (~16 weeks)



Visceral adipose tissue (VAT)

Abdominal subcutaneous adipose tissue (ASAT)

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 tissue)
*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***

CENTRAL ADIPOSITY — WAIST CIRCUMFERENCE (TAPE MEASURE, FULL COHORT n=15 vs. 4)

−9.6 cm
Waist circumference, DA-1726 arm
Baseline 107.5 cm; −9.0%, Day 109; **p<0.001**

−9.1%
Placebo-adjusted
Difference in mean % change vs. placebo; **p<0.001**

Fat compartments fell 19–22% against an 8–9% change in lean compartments — consistent with DA-1726 concentrating in adipose tissue and liver (tissue-distribution study) and with GCGR-driven fat breakdown. Liver fat in the same sub-study: −68.1%, 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. Waist circumference from the full cohort (n=15 vs. 4).

7 Safety & Tolerability: GI Adverse Events

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%)**

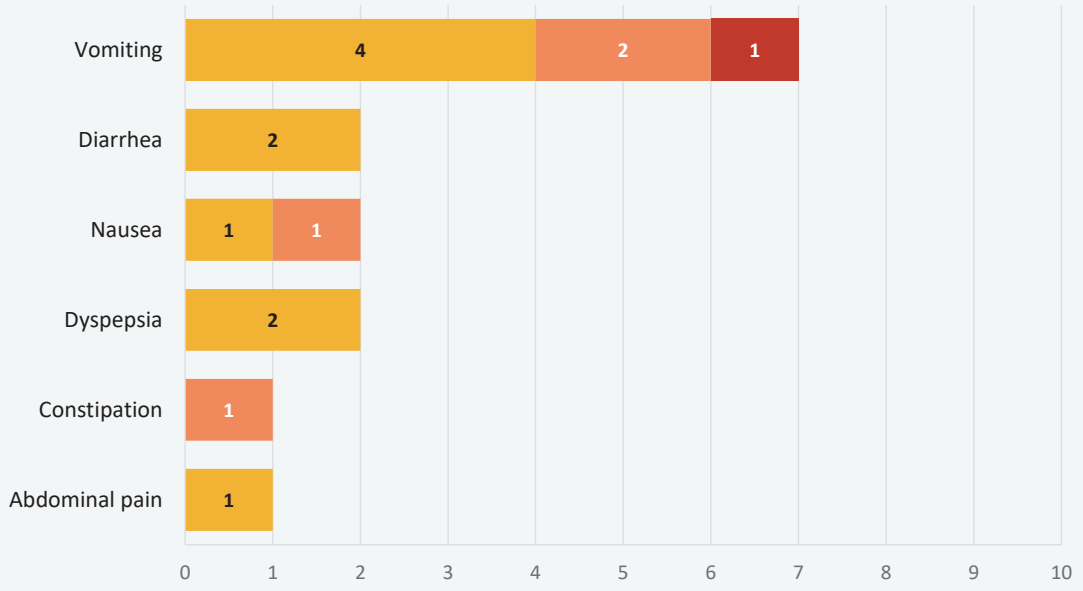
- **Notable AE:** 1 SAE — severe vomiting, possibly related, recovered. 0% study discontinuation due to GI AE.
- **No new GI AEs after Week 9 (Day 63) through study end (Day 109).**

GI adverse events by maximum severity per subject, per MedDRA preferred term — Part 3B interim (N=15 active, N=4 placebo). The placebo arm reported 0% on every term shown (small n; not a powered comparison).

8 GI Adverse Events: Prevalence vs. Instances

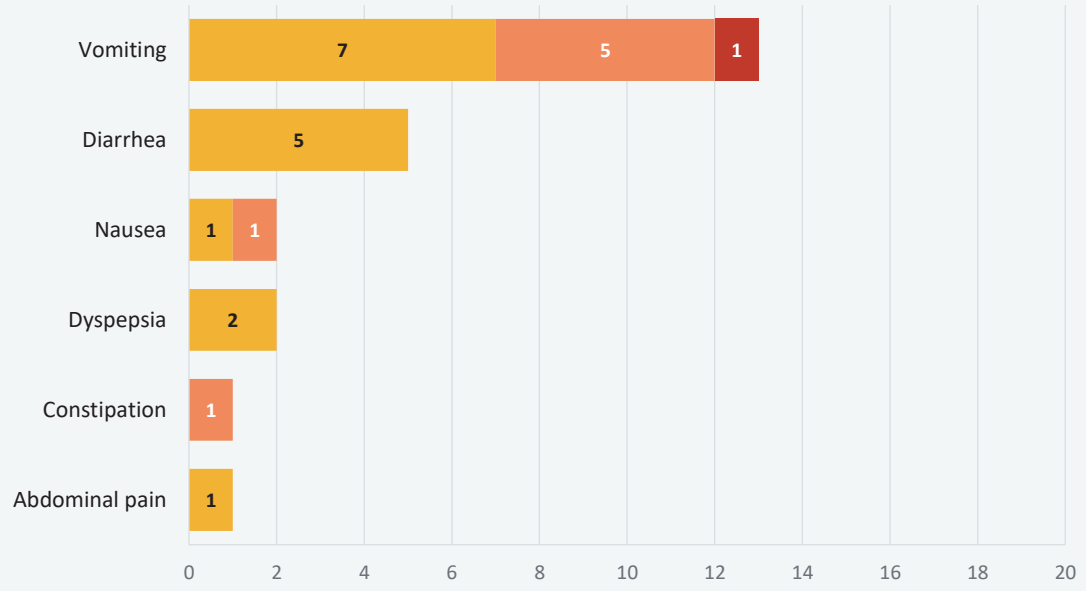
Prevalence

Number of subjects with ≥1 event (of N=15), each subject counted once at their worst grade



Instances — 24 total

Total AE episodes by grade at onset, including recurrences



Mild (Grade 1) Moderate (Grade 2) Severe (Grade 3)

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

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

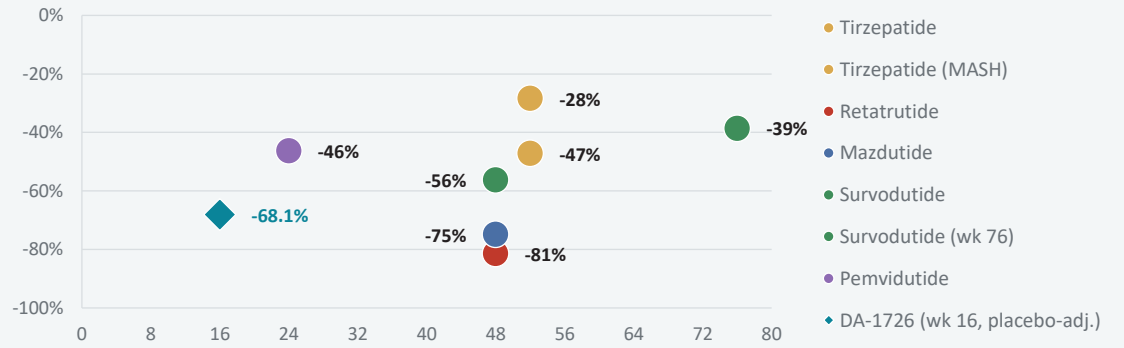


Timeline reflects the trial-day GI AE reporting window, not a modeled daily-prevalence curve. Source: Part 3B interim analysis, Day 109.

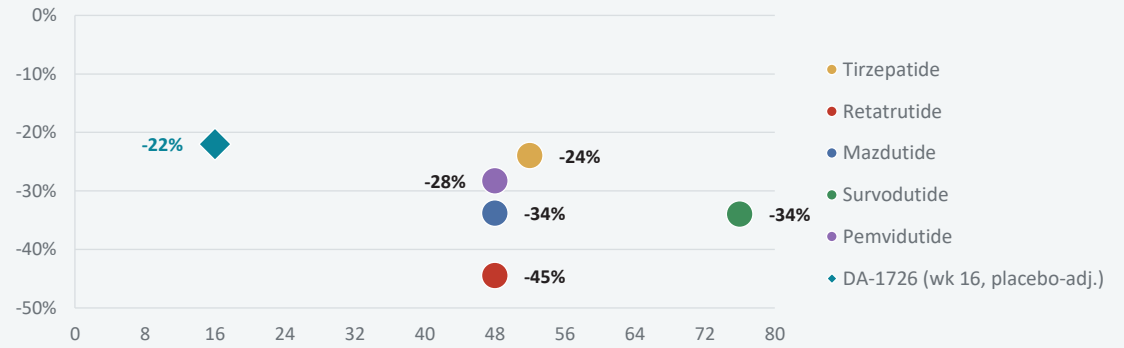
Competitive Landscape: Liver Fat, VAT, ASAT & TAT Over Time

Each dot: one published readout at the comparator dose. Liver fat placebo-adjusted; VAT / ASAT / TAT own-arm (DA-1726 placebo-adjusted).

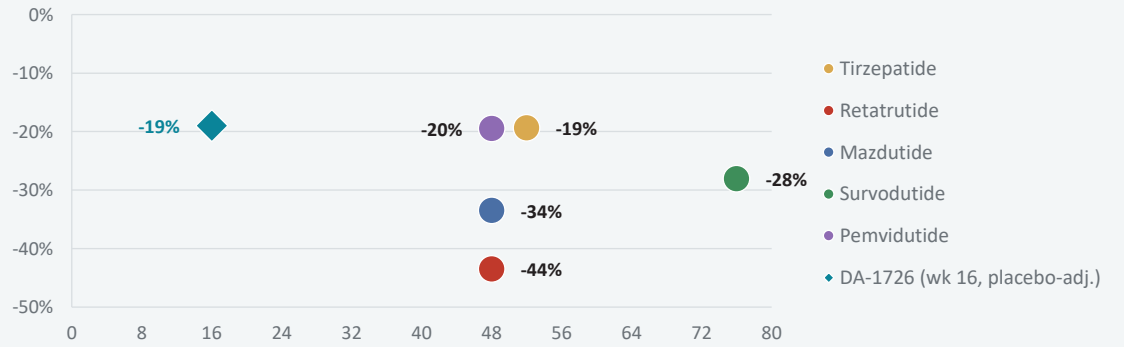
LIVER FAT (MRI-PDFF) — PLACEBO-ADJUSTED % CHANGE



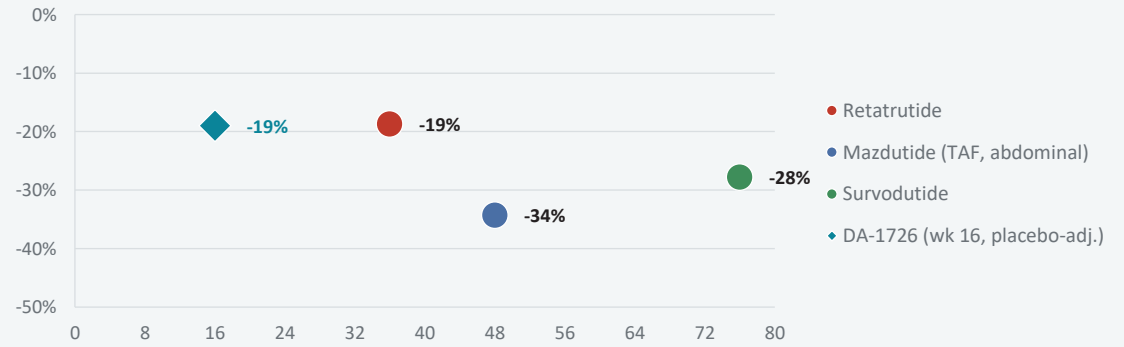
VISCERAL ADIPOSE TISSUE (VAT) — OWN-ARM % CHANGE



ABDOMINAL SUBCUTANEOUS ADIPOSE TISSUE (ASAT) — OWN-ARM % CHANGE



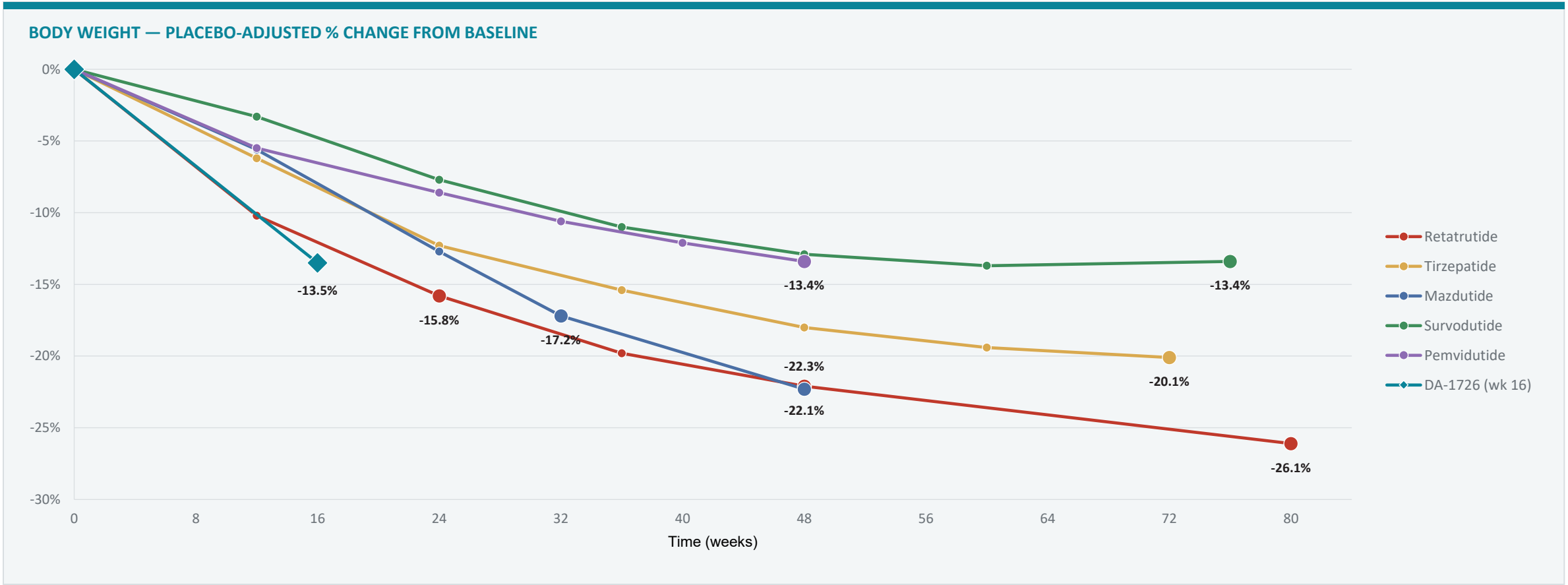
TOTAL ADIPOSE TISSUE (TAT) — OWN-ARM % CHANGE



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 2025); 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 values (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 6.0 mg wk 76 (le Roux 2026 Table 2); pemvidutide 2.4 mg wk 48 (Altimmune, Sept 2024); TAT not reported for tirzepatide or pemvidutide. DA-1726 (teal diamond): placebo-adjusted GMR values at Day 109 ≈ wk 16 (Part 3B interim, n=12 active / 4 placebo), shown for context only — an early, small-sample interim is not comparable to these later-stage readouts. Trials differ in population, duration and design.

Competitive Landscape: Weight Loss Over Time

Placebo-adjusted % change from baseline at the comparator dose, early and pivotal readouts, from each product's published trial.



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% = −20.1 pp (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 2026); 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); DA-1726 Part 3B 64 mg Day 109 = wk 16 −13.5 pp (difference in mean % change vs. placebo, n=15 vs 4, multiple-imputation analysis). Small markers are intermediate values read from the published weight-over-time figures of 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. DA-1726 (teal diamond) is an early, small-sample interim shown for context only; its 64 mg target dose was reached at Week 9 (16 → 32 → 64 mg titration), so the Day 109 readout reflects roughly 8 weeks at the target dose.

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%), tracking 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 debate
- Distribution advantage: for many patients the first MASH drug is the GLP-1 they already 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

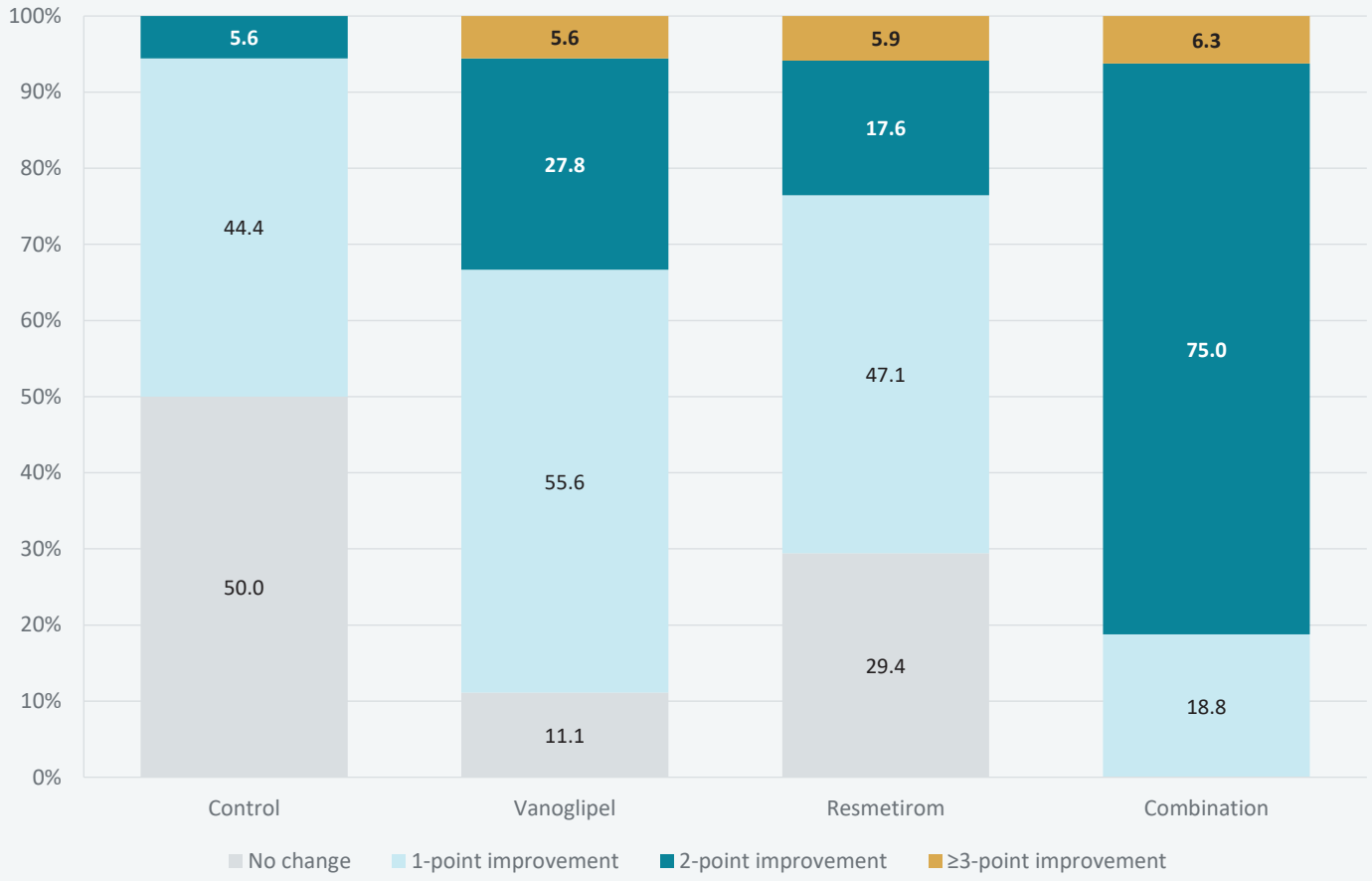
- Mostly mild GI events, at placebo-like rates — diarrhea 15.6% on placebo vs 3.8–14.3% on vanoglipel; nausea, constipation and decreased appetite next most common
- † One SAE — vertigo on Day 23 (100 mg + DPP4i), resolved the next day, drug continued, assessed not related. * Two severe TEAEs (50 mg): 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-related 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 drug and is not a TEAE.

3 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

NAS CHANGE, PRE- TO POST-BIOPSY — % OF ANIMALS



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

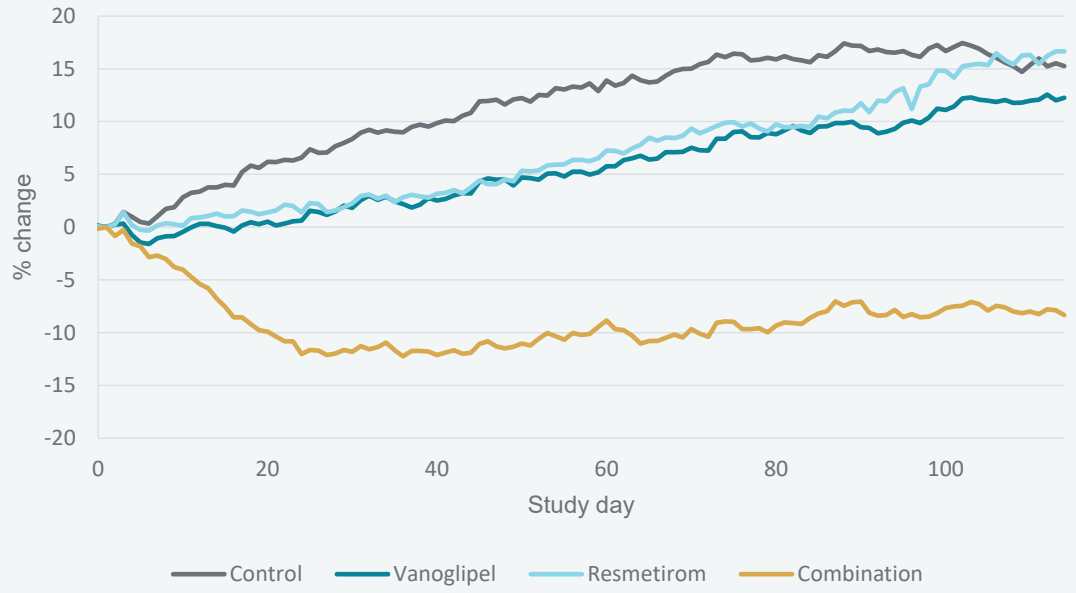
Combination	**+1.88 pts**
Vanoglipel alone	**+1.28 pts**
Resmetirom alone	**+1.00 pts**
Control	**+0.56 pts**

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.

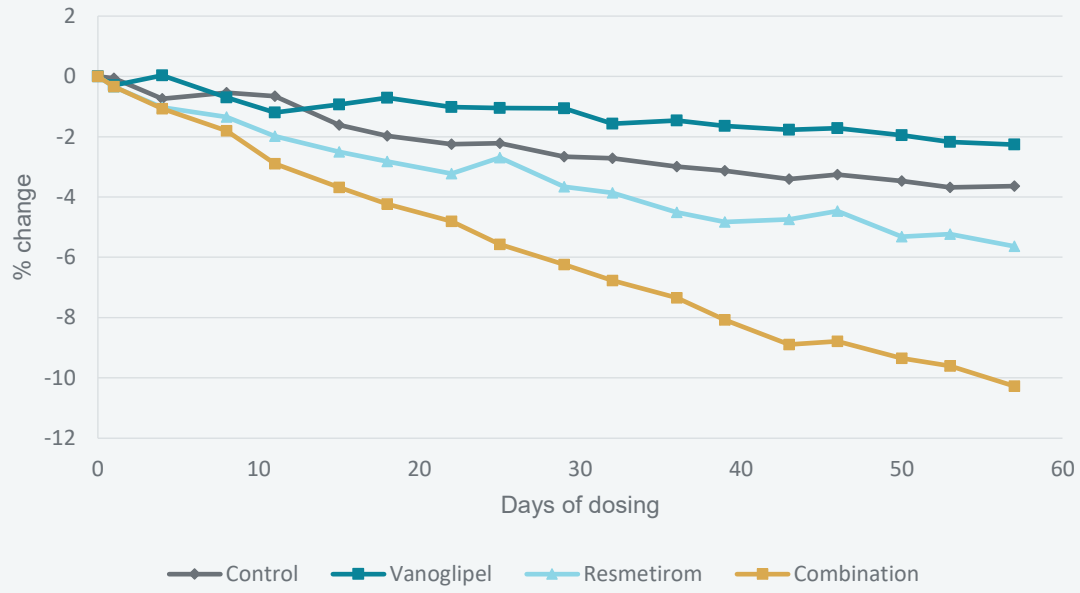
4 Synergy II: Weight Loss Appears Only When the Two Are Combined

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% resmetirom

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).

5 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 use (2018) patents
- MASH combined-use patent (2021); provisional for THR-β combined use in MASH / metabolic disease (2026)
- A single-tablet FDC could add up to 20 years of exclusivity

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

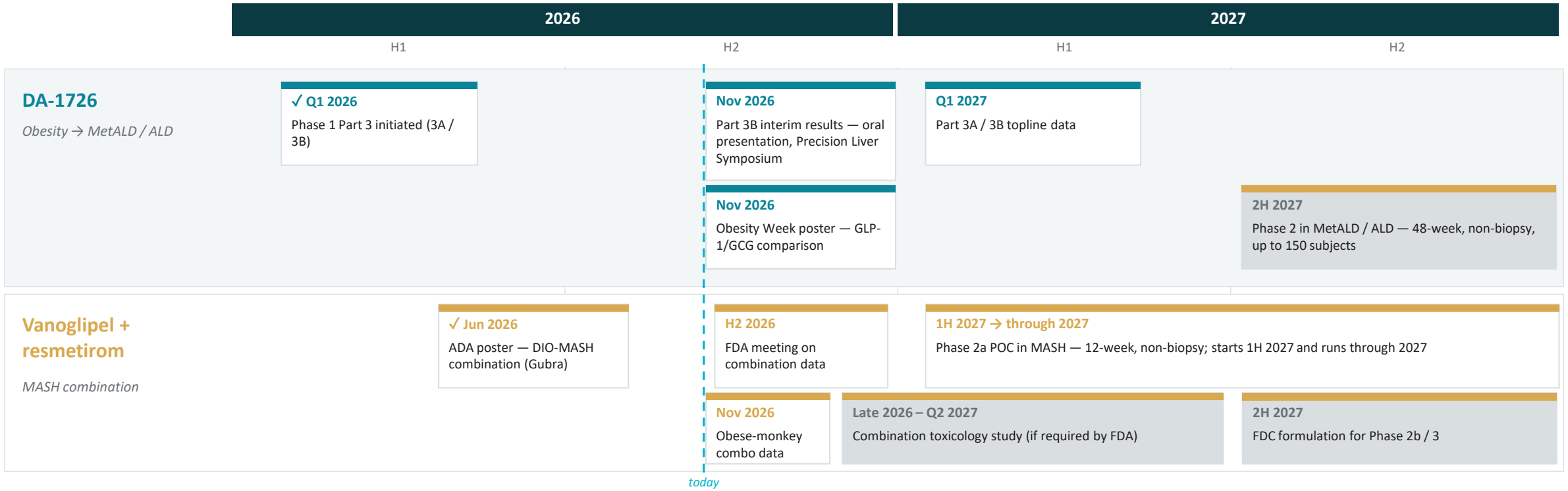
Patents held by Dong-A ST and licensed to MetaVia; Korean registrations 10-1726819, 10-2295300, 10-2538047, provisional 10-2026-0058472. Regulatory route and study requirements subject to FDA feedback. Milestones assume regulatory and clinical success, which is not guaranteed.

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 to 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

2 Financial Overview and Capitalization

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

$8.8M
cash and cash equivalents
as of September 30, 2026 · no debt

Into 1Q 2027
expected cash runway
based on the current operating plan

6.74M
common shares outstanding
15.92M fully diluted

26.2%
held by Dong-A ST and affiliate
1,766,572 shares · licensor of both assets

The 2027 clinical programs are contingent on additional financing.

CAPITALIZATION TABLE — SEPTEMBER 30, 2026

	Common stock equivalents
Common stock	6,742,889
Warrants (WAEP $7.94) [1]	8,947,282
Options (WAEP $4,240.22)	420
Restricted stock units	229,370
Fully diluted	**15,919,961**

(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, callable in the event of a positive readout of Phase 1b Part 3 data for DA-1726; (iii) 2024 Series B milestone-based warrants to purchase 693,962 shares, expiring September 2029, exercise price $43.23; and (iv) 2022 Series B warrants to purchase 16,176 shares, expiring December 2027, assumed exercise price $0.00. 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 Liver 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 September 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: −68.1% 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 is building its own combination — the thesis is validated by the leader
- Both programs are liver-directed and differentiated from the weight-only incretin field — one oral, one once-weekly

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

MetaVia

Appendix

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.*

1 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 agonist
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, obesity); 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

−68.1%
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 above — 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., NEJM 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.

2 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):
−68.1% *(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.

3 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 change)

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 fibrosis 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 Suppl. Table S6); Zepbound US Prescribing Information (Nov 2023). Figures are taken from each product's own published trial; populations, durations and designs differ.

4 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 mg)

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., NEJM 2023 (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).*

5 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 2026, appendix)

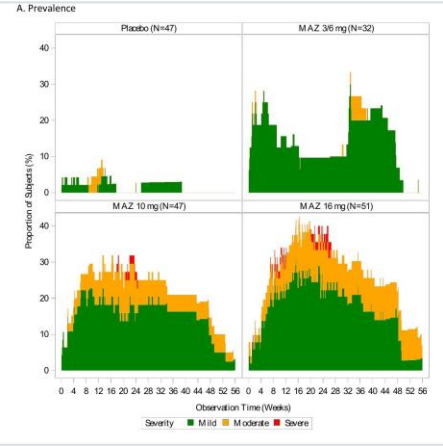
ASAT: −31.1% vs placebo (16 mg)
MRI · wk 48 · own-arm −33.5%, placebo −2.4 to −3.4% (Hsia 2026, appendix)

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

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% −80.2% vs −5.3%; LFC ≥5% −73.2% vs +8.2%; ≥30% responders 95.5% vs 27.3%).

6 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, Table 2)

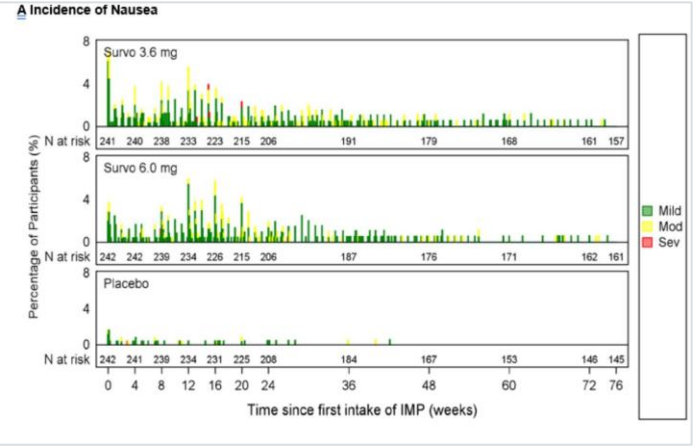
SAT: −18.3 pp vs PBO (6.0 mg)
MRI substudy · wk 76 · own-arm −28.1% vs −9.8% (NEJM 2026, Table 2)

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

7 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); Phase 2 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 liver fat −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).*