November 2026

Marea Therapeutics

Transformative cardioendocrine therapies

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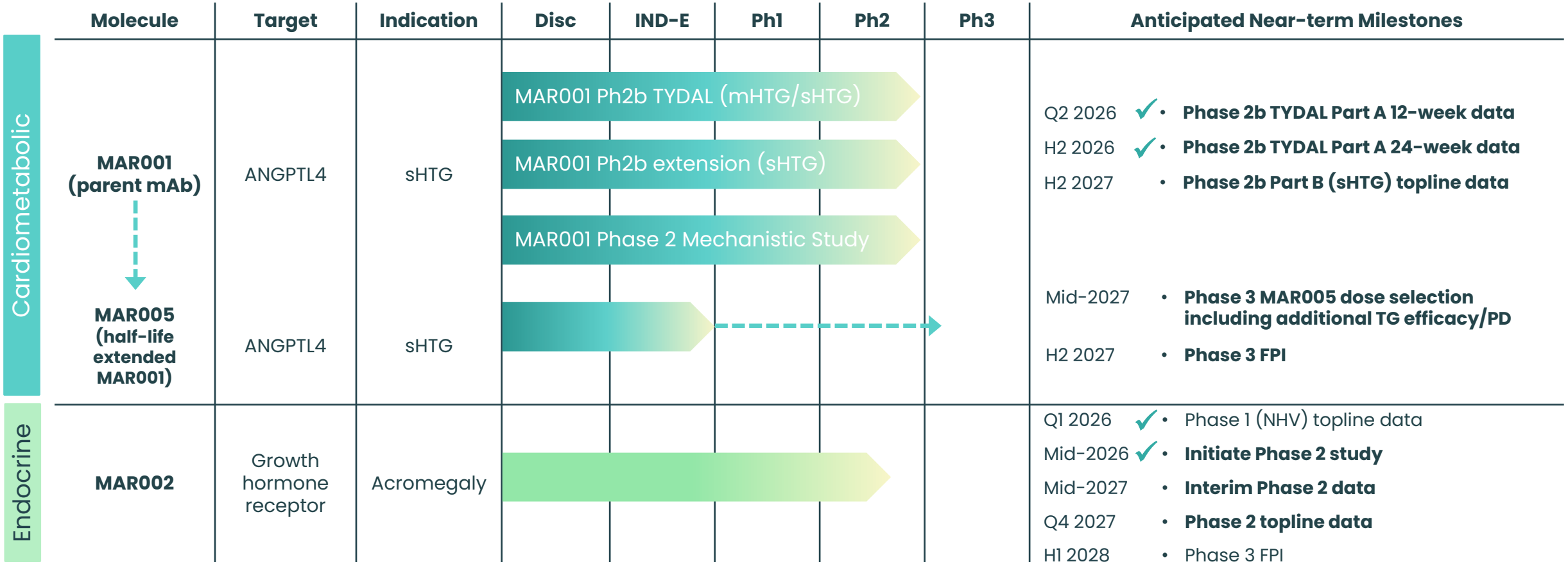
Two Clinical Assets Target Large, Underserved Cardioendocrine Markets

Two differentiated programs: MAR001/5 in a >$10B sHTG market; MAR002 in a >$1.4B acromegaly market

	Patient need	Clinical Activity	Differentiation	Opportunity
MAR001/5 **Severe Hypertriglyceridemia** ***Potent anti-ANGPTL4 sHTG candidate, clean metabolic profile***	**>3M** **US patients[1]** Pancreatitis risk; APOC3 drugs worsen diabetes and liver fat	**Matches APOC3[2]** Phase 2 showed competitive TG lowering in sHTG subgroup	**Clean metabolic profile** No HbA1c or liver-fat penalty to date vs. APOC3 class	**>$10B** **Projected US sHTG market[3]** Core: 1.8M patients with diabetes[4]
MAR002 **Acromegaly** ***First long-acting GHR antagonist for acromegaly***	**~30K** **US patients[5]** Highly morbid; >70% uncontrolled	**Matches best-in-disease[6]** Phase 1 IGF-1 on par with pegvisomant	**Better dosing and safety** Q2W dosing; no SAEs, ISRs, or LFT signals in Phase 1	**>$1.4B** **Projected acromegaly market[3]**

1. Christian JB, et al. Am J Cardiol 2011; 2. https://www.nejm.org/doi/abs/10.1056/NEJMoa2612463; 3. EvaluatePharma; 4. Zafrir B, et al. J Clin Lipidol 2018; 5. Burton T, et al. Pituitary 2016. 6. Marea data on file. MAR001/5: MAR001 (parent antibody) and MAR005 (formerly MAR001-HLE). GHR: growth hormone receptor; ISR: injection-site reaction; LFT: liver function test; Q2W: every 2 weeks; SAE: serious adverse event; sHTG: severe hypertriglyceridemia; SRL: somatostatin receptor ligand; TG: triglycerides.

Late-stage Cardioendocrine Pipeline with Multiple 2026–27 Readouts

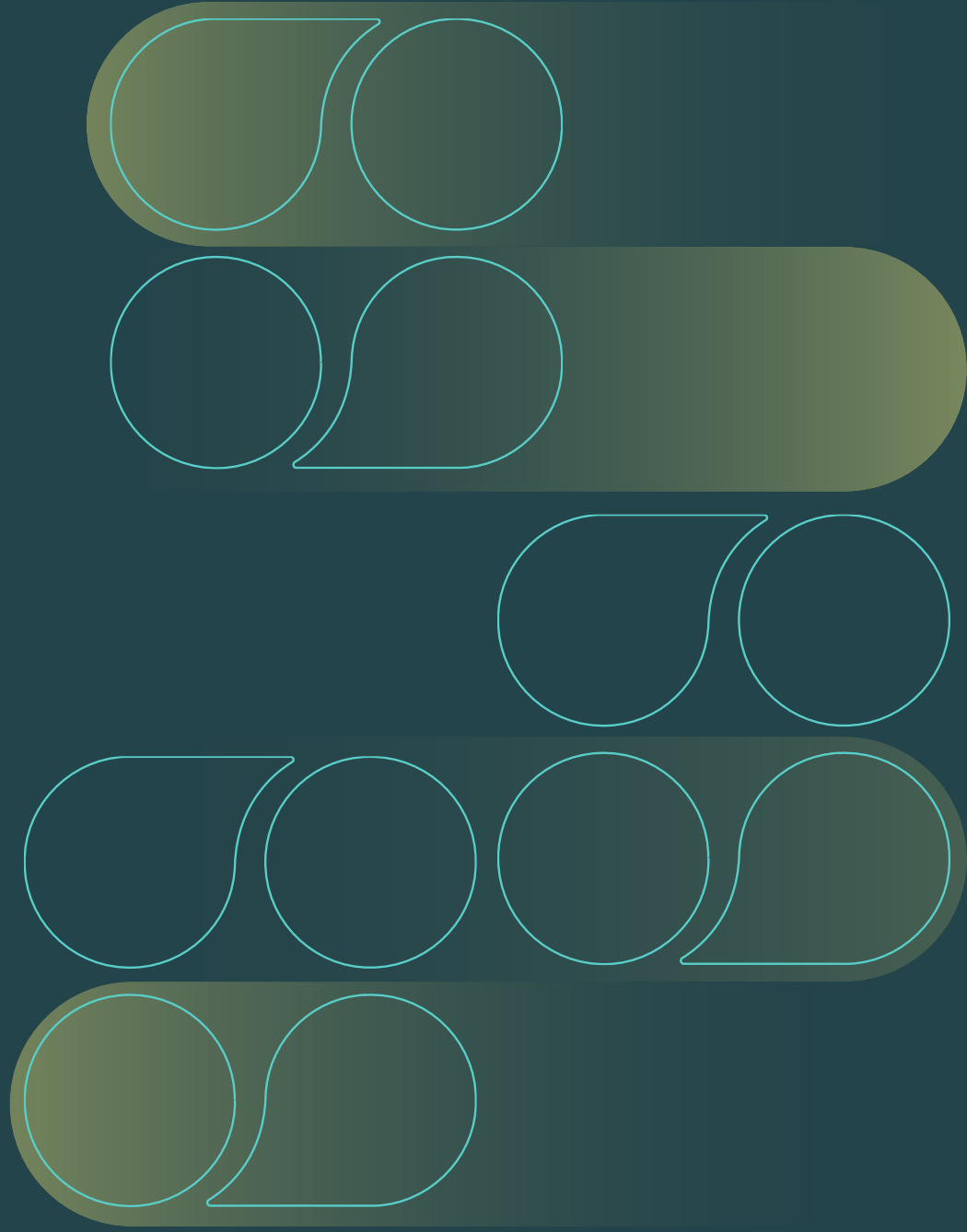


- MAR001 showed Phase 2 POC; MAR005 (half-life extended, improved PK) is our Phase 3 candidate; FDA agreed to PK bridging
- Marea also has developed a proprietary SiRNA platform and is developing adipose tissue targeted SiRNAs

Disc: discovery; FPI: first patient in; IND-E: IND-enabling; mHTG: moderate hypertriglyceridemia; NHV: normal healthy volunteer; PD: pharmacodynamics; PK: pharmacokinetics; POC: proof of concept; sHTG: severe hypertriglyceridemia; TG: triglycerides

MAR005 for sHTG

Potent sHTG candidate with a clean metabolic profile



marea

sHTG Is a Large Unmet Need Validated by APOC3 Inhibitors

APOC3 drugs cut TG 60–80% and pancreatitis events 78–85%, opening a >$10B US market

The unmet need

>3M

US adults with sHTG
- 1M at high risk of recurrent acute pancreatitis[1]
- Up to 20% mortality per episode[7]

60%

Comorbid T2DM
- 30% CAD, ~70% fatty liver: high CV/metabolic risk[2,3]

>$9B

Annual US cost burden
- Fibrates, omega-3s: ~30% TG lowering, no AP prevention data[4]

APOC3 inhibitors validate the opportunity

60–80%

TG reduction from baseline
- First class to reach <500 mg/dL in most patients[2,5]

78–85%

Reduction in AP events
- First proven AP reduction in sHTG[2,5]

>$10B

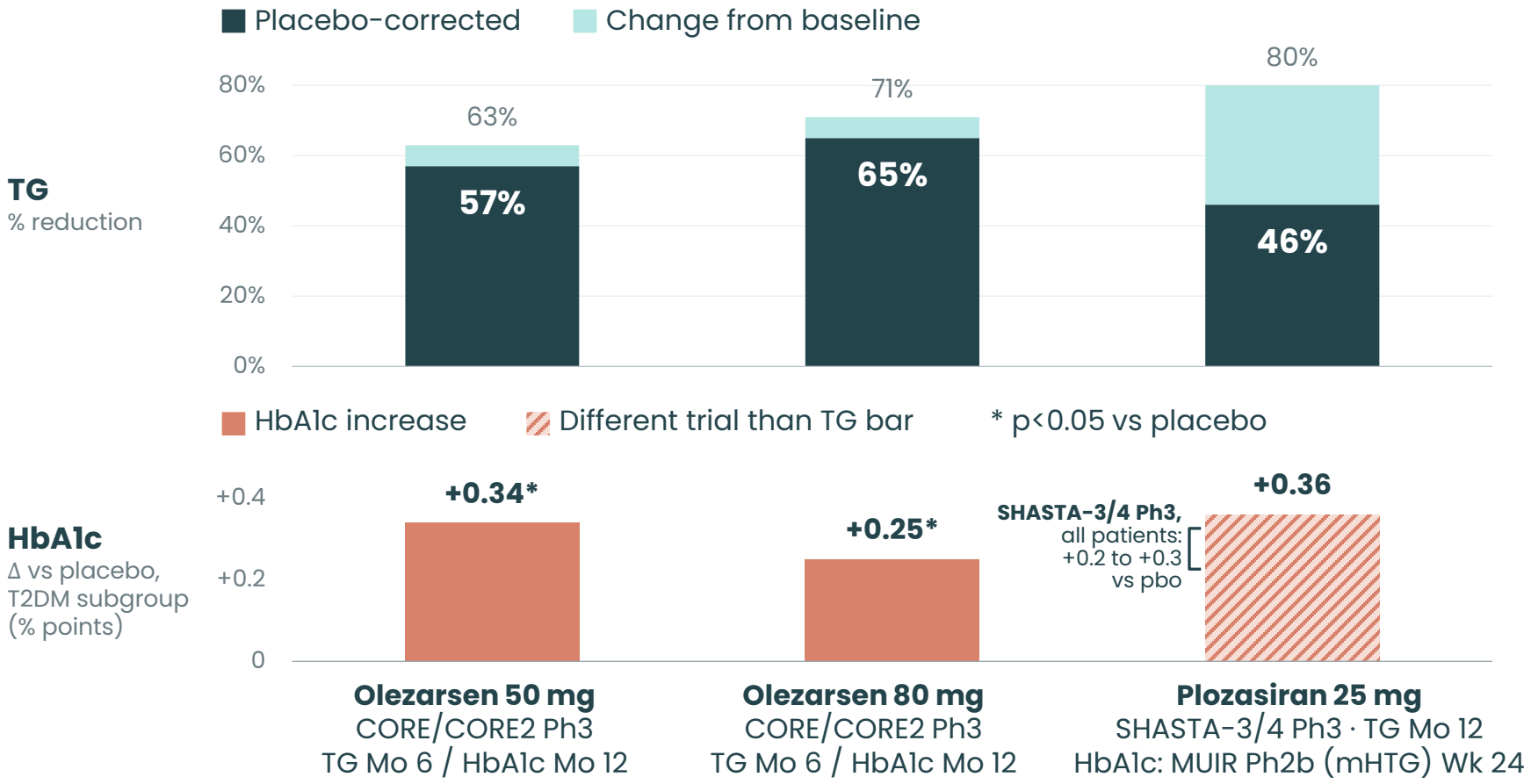
Projected US market
- Peak sales: olezarsen >$3B, plozasiran $3–4B; ~$45K annual list price[6]

But APOC3 efficacy comes at a cost

1. Christian JB, Am J Cardiol 2011. 2. Marston NA, NEJM 2026. 3. Gurevitz C, JACC Adv 2024. 4. Lee PJ, Nat Rev Gastroenterol Hepatol 2019. 5. Ionis AHA 2025 press release; Watts GF, NEJM 2026 (SHASTA-3/4). 6. EvaluatePharma; ClearView Healthcare Partners; peak sales are analyst/consensus estimates. 7. Lu J, BMC Gastroenterology. AP: acute pancreatitis; CAD: coronary artery disease; CV: cardiovascular; sHTG: severe hypertriglyceridemia; T2DM: type 2 diabetes; TG: triglycerides.

APOC3 Class Efficacy Comes at a Cost: HbA1c Increases in Patients with T2DM

Placebo-corrected TG down 46 to 65%, yet HbA1c rises 0.25 to 0.36 pts vs. placebo in T2DM



Additional safety signals

Glycemic AEs
Plozasiran: worsening glycemic control 14.3% vs 8.7% pbo (SHASTA-3/4 Ph3, ~60% T2DM)

Discontinuations
Plozasiran OLE: 18 of 26 AE-driven discontinuations (69%) were glycemic

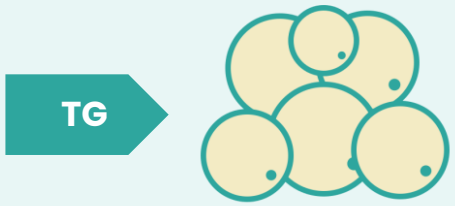
Liver fat
Olezarsen: HFF +2.3 / +4.2 pts vs pbo at Mo 12 (50 / 80 mg); plozasiran NS

SHASTA-3/4 data (TG, HbA1c, glycemic AEs): Watts NEJM 2026;395:1805-17. TG: olezarsen LS mean, n-weighted CORE/CORE2 (pbo-adj. -62.9/-72.2 and -49.2/-54.5), Mo 6; plozasiran Hodges-Lehmann median difference vs pbo, Mo 12, SHASTA-3/SHASTA-4 (-45/-47; mean of the two shown; median change from baseline -79%/-81%). HbA1c: T2DM subgroups; olezarsen Mo 12 (Marston NEJM 2026;394:429-41, Table S7). Plozasiran HbA1c: T2DM subgroup from MUIR Wk 24 (Ballantyne NEJM 2024). SHASTA-3/4 reports HbA1c in all patients only (+0.2 to +0.3 vs pbo, Mo 3 to Mo 9; Fig. S11); the prespecified T2DM subgroup and new diabetes medications by arm were not reported. The SHASTA-3/4 protocol directed investigators to adjust diabetes therapy back toward baseline HbA1c, which attenuates the observed placebo-corrected difference. Ballantyne Am J Prev Cardiol 2026. No head-to-head studies. AE: adverse event; HFF: hepatic fat fraction; LS: least squares; mHTG: moderate hypertriglyceridemia; Mo: month; NS: not significant; OLE: open-label extension; pbo: placebo; Ph: phase; T2DM: type 2 diabetes; TG: triglycerides; Wk: week.

Biology and Genetics Support ANGPTL4 Inhibition as Potential Best-in-Disease Profile

TG disposal into fat, not liver

ANGPTL4 inhibition (MAR001/5)



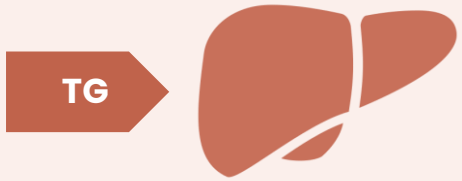
TG cleared into fat

- Unblocks LPL in adipose tissue
- Fat stores TG; no liver fat loading

> *… adipose enrichment [of ANGPTL4] may lead to a more favorable metabolic profile…*
> **— MAR001 Phase 2b authors, NEJM 2026**

vs

APOC3 inhibition (olezarsen / plozasiran)



TG routed to the liver

- Speeds liver uptake of TG remnants
- Shifts liver to fatty acid over glucose use
- Liver fat up (olezarsen); HbA1c up

> *Perhaps this apparent difference [in glycemic control] results from increased delivery of glycerol and fatty acid substrate to the liver…*
> **— Plozasiran Phase 3 authors, NEJM 2026**

Genetics predict differentiated impact on glycemic control*

ANGPTL4
T2DM risk and glucose **lower**

APOC3
T2DM risk and glucose **higher**

A mechanism with potential for avoiding metabolic penalties

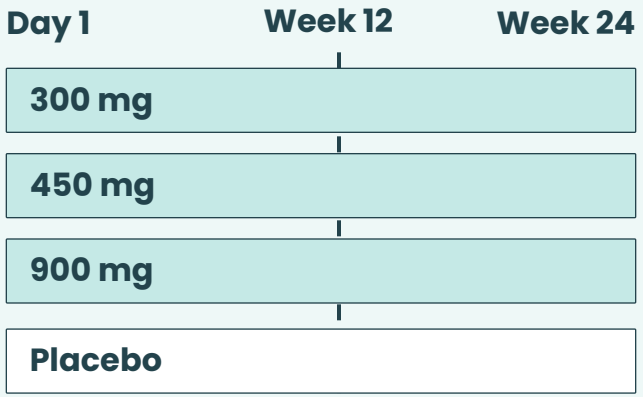
LPL: lipoprotein lipase; T2DM: type 2 diabetes; TG: triglycerides. Liver fat: olezarsen hepatic fat fraction +2.3/+4.2 pts vs placebo at Mo 12 (50/80 mg; Marston NEJM 2026); plozasiran not significant. *Genetics: Eur Heart J Open 2024;4(3):oeae035; doi:10.1186/s12933-026-03209-w; doi:10.1038/s41467-018-04611-z. Quotes from Discussion sections, bracketed text added: O'Donoghue ML et al., NEJM 2026 (TYDAL-TIMI 78); Watts GF et al., NEJM 2026;395:1805-17 (SHASTA-3/4).

TYDAL-TIMI 78 Phase 2b: Potent TG Lowering without Safety, Tolerability or HbA1c Signals

Primary endpoint met at every dose (P<0.001); larger effect at higher baseline TG

Study design

235 patients · 44% T2DM
Baseline TG 150–880 mg/dL



MAR001 Q4W, randomized 3:1
Primary: TG % change, week 12

	Change from baseline	Placebo-adjusted
All patients Placebo +5%; all doses P<0.001	**-34 to -40%**	**-39 to -46%**
TG ≥300 mg/dL (n=73) Placebo +9%; all doses P<0.001 (key secondary)	**-44 to -49%**	**-53 to -57%**

Safety through week 24

Serious Adverse events Pooled MAR001 vs placebo	**2.8%** vs 6.9%	**No HbA1c rise** **No liver enzyme signal**

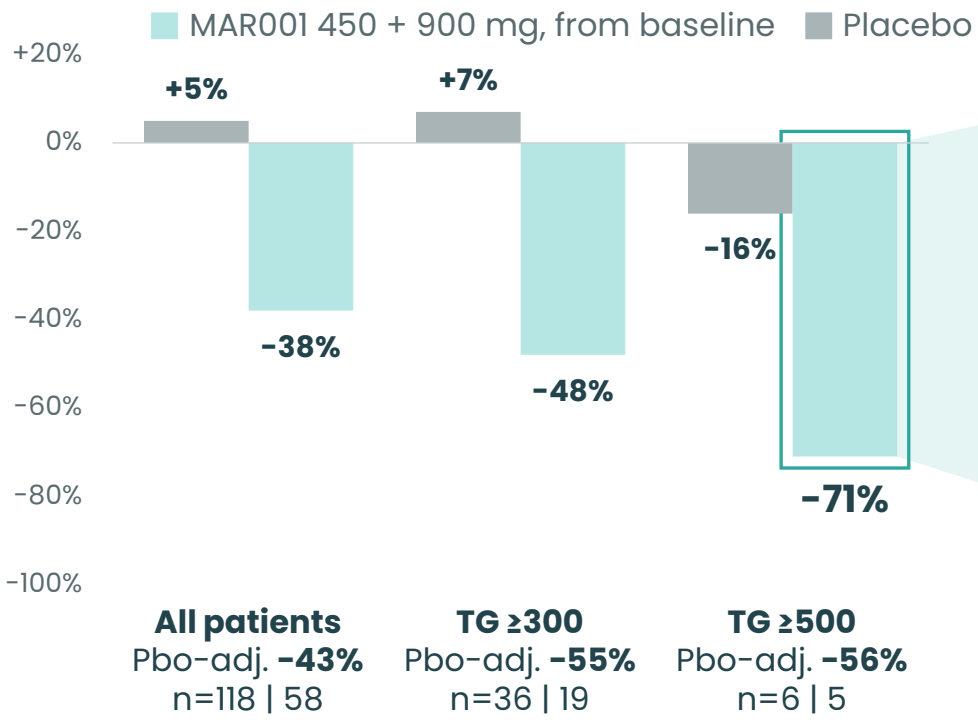


235 treated participants (236 randomized). Week 12 = mean of Weeks 10 and 12. LS mean % change (MMRM, on-treatment), range across 300/450/900 mg; TG ≥300 was a prespecified key secondary endpoint (hierarchical testing). LS: least squares; MMRM: mixed model for repeated measures; PK/PD: pharmacokinetics/pharmacodynamics; Q4W: every 4 weeks; T2DM: type 2 diabetes; TG: triglycerides. TYDAL-TIMI 78; https://www.nejm.org/doi/abs/10.1056/NEJMoa2612463

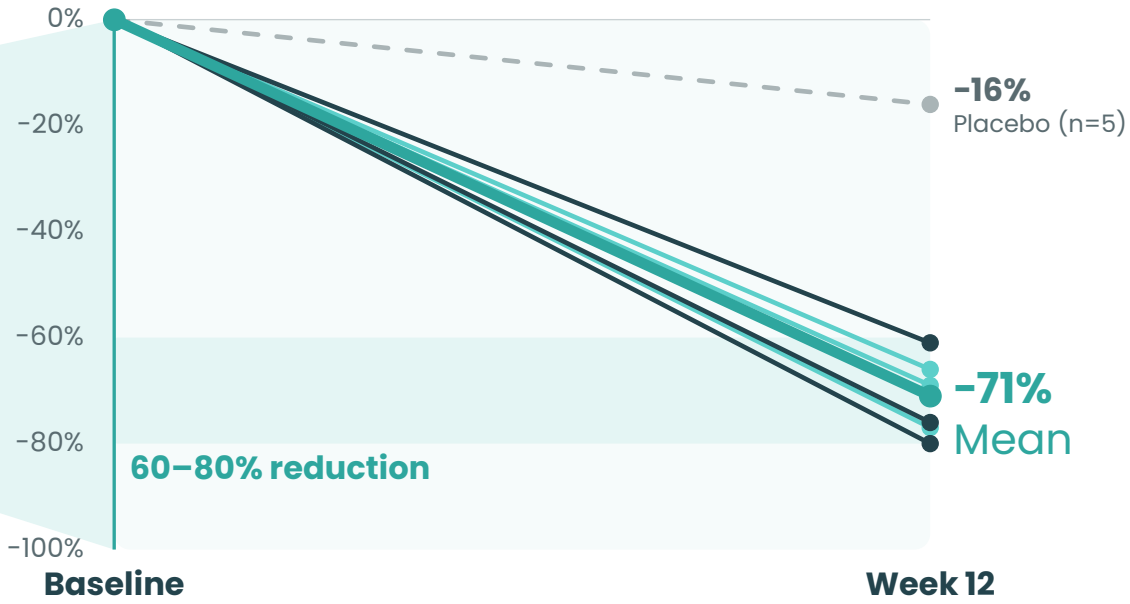
Reduction Scales with Baseline Triglycerides: Rises from 38% to 71%

Every sHTG patient on 450 or 900 mg reduced TGs 61 to 80% at Week 12

% change in TG from baseline, by baseline TG group



TG ≥500 mg/dL: each patient on 450 or 900 mg (n=6)

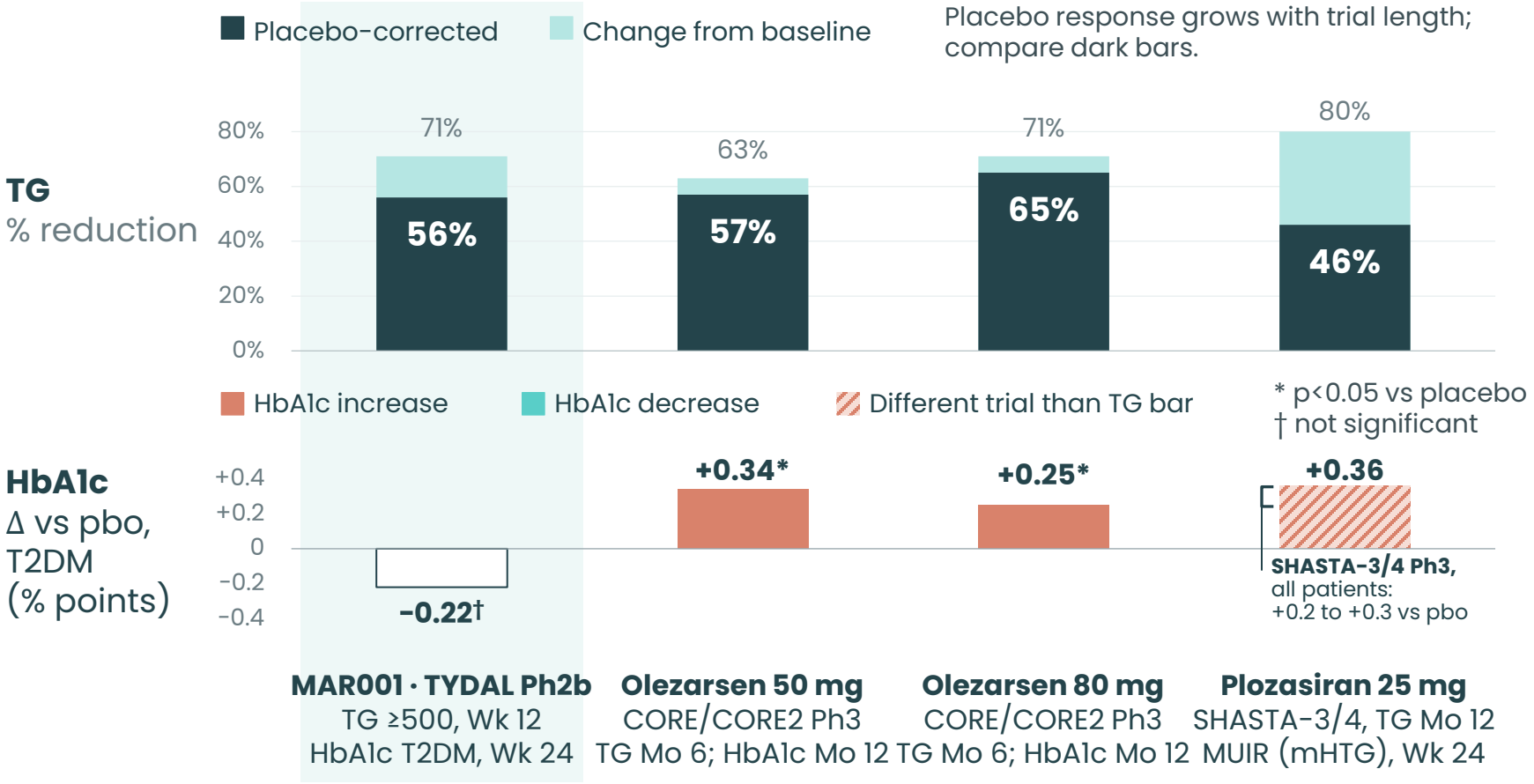


Part B (n~100, TG ≥500 mg/dL, 600 mg) designed to confirm in sHTG; topline H2 2027

TYDAL-TIMI 78 Part A, Week 12 (mean of Weeks 10 and 12). Left: LS mean % change (MMRM), average of the 450 and 900 mg arms (Marea calculation; doses bracket the 600 mg Part B dose) vs pooled placebo. Published pbo-adj. change, 450 / 900 mg: all patients -40.9% / -45.5%; TG ≥300 -53.7% / -57.2%. TG ≥500 (500–879 mg/dL) is descriptive (n=6 vs 5), data on file. Right: individual patients, baseline to Week 12; bold = mean, dashed = placebo. LS: least squares; MMRM: mixed model for repeated measures; Pbo-adj.: placebo-adjusted; sHTG: severe hypertriglyceridemia; TG: triglycerides. Source: O'Donoghue ML et al., NEJM 2026 (TYDAL-TIMI 78); Marea data on file.

MAR001: Potential Best-in-Disease Profile

Similar TG reduction; no HbA1c increase in T2DM



MAR001 Safety profile to date

Glycemic markers

- no adverse change in HbA1c, fasting glucose, insulin or HOMA-IR vs placebo

Liver

- no ALT/AST >3× ULN (0 vs 1 on placebo);
- no hepatic fat increase observed to date

Tolerability

- TEAEs 69.5% vs 77.6% pbo;
- injection-site reactions 14.1% vs 19.0% pbo

MAR001: LS mean, avg of 450/900 mg (n=3 each vs 5 pbo), TG ≥500, Wk 12 (TYDAL-TIMI 78, Table S3); HbA1c all doses pooled, T2DM (n=74 vs 29), Wk 24, data on file. Olezarsen: n-weighted CORE (-62.9/-72.2 pbo-adj.) and CORE2 (-49.2/-54.5), Mo 6; HbA1c T2DM, Mo 12 (Marston NEJM 2026;394:429-41, Table S7). Plozasiran (SHASTA-3/4, Watts NEJM 2026;395:1805-17): Hodges-Lehmann median difference vs pbo, SHASTA-3/SHASTA-4 (-45/-47; mean of the two shown), Mo 12; median change from baseline 79%/81%. Plozasiran HbA1c: T2DM subgroup from MUIR Wk 24 (Ballantyne NEJM 2024). SHASTA-3/4 reports HbA1c in all patients only (+0.2 to +0.3 vs pbo, Mo 3 to Mo 9; Fig. S11); the prespecified T2DM subgroup and new diabetes medications by arm were not reported. The SHASTA-3/4 protocol directed investigators to adjust diabetes therapy back toward baseline HbA1c, which attenuates the observed placebo-corrected difference. No head-to-head studies have been conducted; cross-trial comparisons are subject to inherent limitations and should be interpreted with caution. ALT/AST: liver enzymes; HOMA-IR: insulin resistance index; LS: least squares; mHTG: moderate hypertriglyceridemia; Mo: month; pbo: placebo; Ph: phase; T2DM: type 2 diabetes; TEAE: treatment-emergent adverse event; ULN: upper limit of normal.

MAR005 Is Designed for the Majority of sHTG Patients Who Also Have Diabetes

Glycemic neutrality: clinically meaningful, valued by physicians, large opportunity

0.3%

HbA1c rise matters in T2DM
Clinically meaningful: confers independent CV risk

#1

Physician-weighted attribute
for new sHTG drugs in T2DM

1.8M

US sHTG patients with T2DM
≥50% with uncontrolled glucose: the core of a >$10B market

> *Any new starts would probably go to the ANGPTL4 inhibitor … a much cleaner profile.*
> **Endocrinologist**

> *I would target the use of [MAR005] for patients who have diabetes or prediabetes, or I think are at high risk for developing diabetes.*
> **Cardiologist**

Potential best-in-disease profile for sHTG patients with prediabetes and diabetes

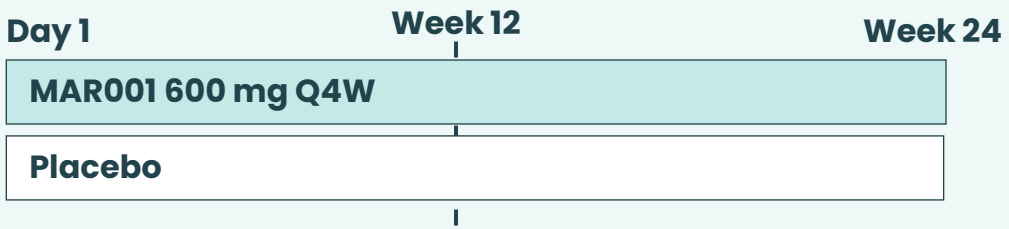
Sources: ClearView Healthcare Partners physician survey, Q1 2026 (N=151: 75 endocrinologists, 70 cardiologists, 6 lipidologists; plus 15 interviews with blinded conjoint). Luo S, et al. Diabetologia 2021;64(11):2502–2510. Zafrir B, et al. J Clin Lipidol 2018;12(4):928–936. NEJM 2021;384:2219–2228. Market size: EvaluatePharma; ClearView. CV: cardiovascular; sHTG: severe hypertriglyceridemia; T2DM: type 2 diabetes.

TYDAL-TIMI 78 Part B: Confirming Effects in sHTG; Next Data Catalyst

~100 patients with TG ≥500 mg/dL randomized to MAR001 600 mg vs placebo; topline H2 2027

Study design

~100 patients, nonfasting TG ≥500 and fasting TG ≥450 mg/dL
~50% T2DM enrichment



1:1 randomization; 24 weeks

Primary: fasting TG % change, Wk 12
Secondary: achieving <500 mg/dL
Exploratory: TG ≥880 subgroup

Study Goals

Strong TG drop

Majority of patients achieve TG <500 mg/dL

Metabolic neutrality

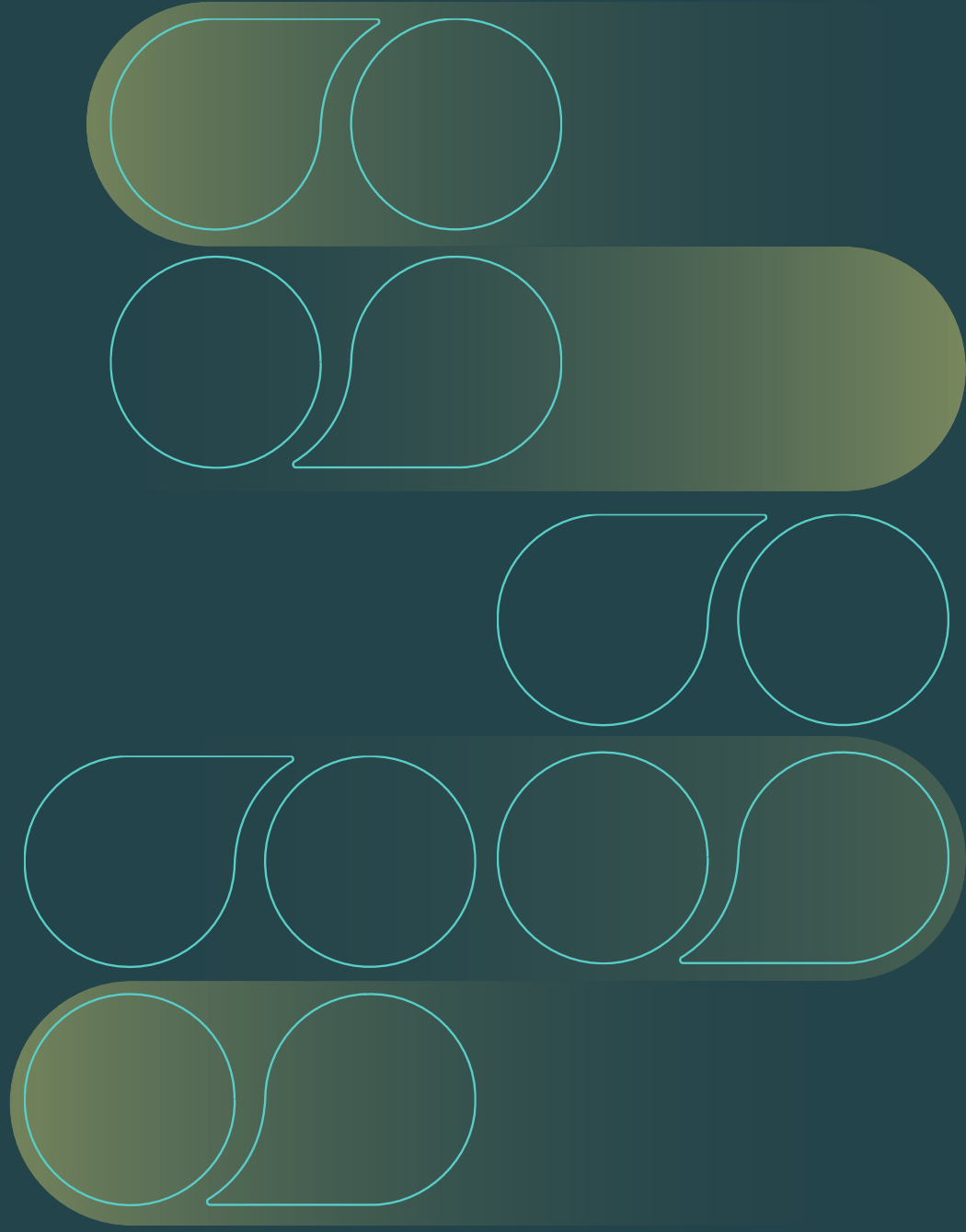
Clean safety

Next: TYDAL-TIMI 78 Part B topline data H2 2027

Olezarsen: Marston NA et al., NEJM 2026 (50 mg, Mo 6). Plozasiran: Watts GF et al., NEJM 2026 (SHASTA-3/4, Mo 12; placebo-adj. = Hodges-Lehmann median difference). Company targets; cross-trial caveats apply. AP: acute pancreatitis; FCS: familial chylomicronemia syndrome; ISR: injection-site reaction; MRI-PDFF: MRI proton density fat fraction; Q4W: every 4 weeks; RC: remnant cholesterol; sHTG: severe hypertriglyceridemia; T2DM: type 2 diabetes.

MAR002 for Acromegaly

First long-acting GHR antagonist candidate for acromegaly



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Acromegaly: Serious Disease with Inadequate Options for Medical Therapy

The Burden & Opportunity

~30K US patients

- Excess GH from a benign pituitary adenoma

Early mortality and high morbidity

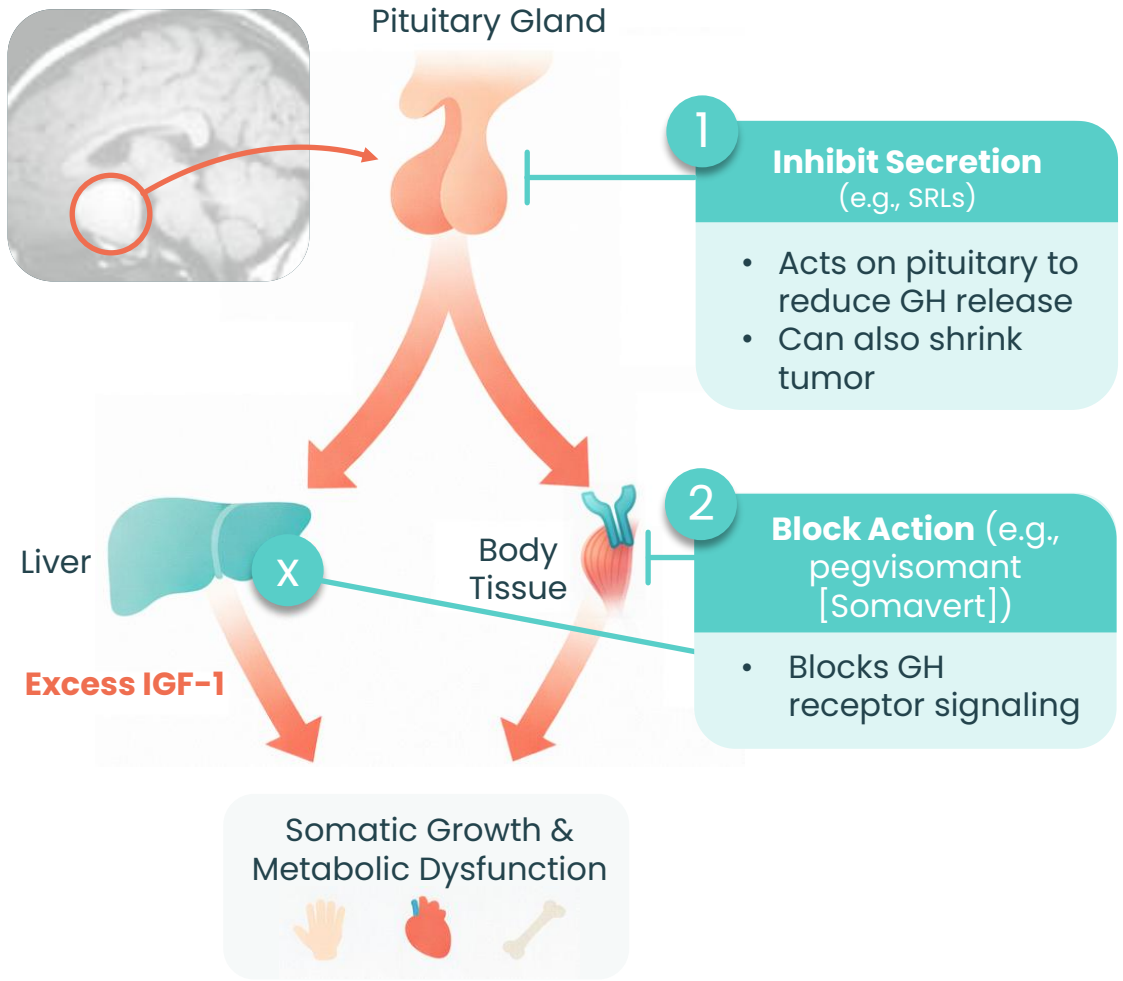
- Bone/organ overgrowth; cardiometabolic disease
- Median lifespan ~10 years shorter if untreated
- 2.5x mortality if IGF-1 is not normalized

Medical therapy goals

- Decrease tumor volume (some patients)
- Normalize IGF-1 levels (all patients)

Efficient development path: IGF-1 is an established clinical and regulatory endpoint

Two Approaches to Medical Therapy



Abbreviations: GH: growth hormone; IGF-1: insulin-like growth factor 1; SRL: somatostatin receptor ligand
Sources: UpToDate; Holdaway et al., Eur J Endo, 2008; Moustaki et al., Endocrine, 2023; van der Lely et al., Lancet, 2001

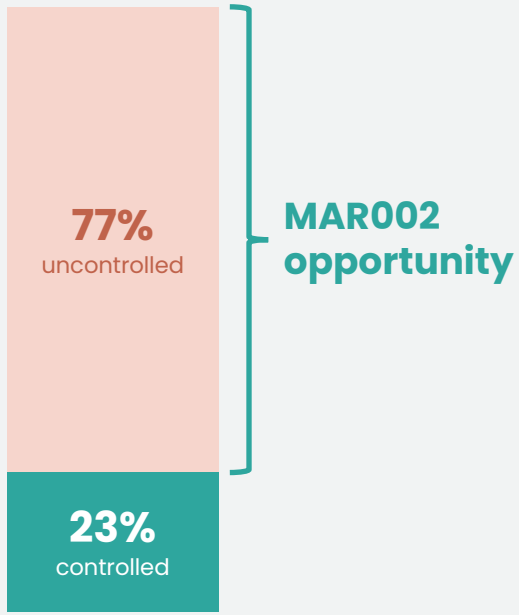
An Optimal GHRA Could Become the Treatment of Choice for Most Acromegaly Patients

1. SRLs first-line for ~80% after surgery → 2. >70% remain uncontrolled → 3. Add or switch to a GHRA

THE UNMET NEED

>70% of patients fail to respond to first-line SRLs

- First line for most patients (potential tumor volume benefit)
- Hyperglycemia (74% incidence), GI AEs

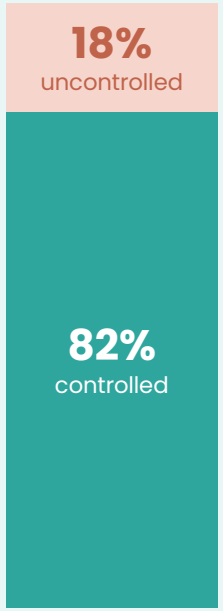


Paltusotine, treatment-naive, Phase 3

THE OPPORTUNITY

>80% of these patients respond to GHRAs but the only option, pegvisomant, falls short

Strong efficacy but high daily treatment burden.



Pegvisomant, GHRA-naive, Phase 3

An optimized, long-acting GHRA could address 3x more patients than SRLs: a >$1B opportunity

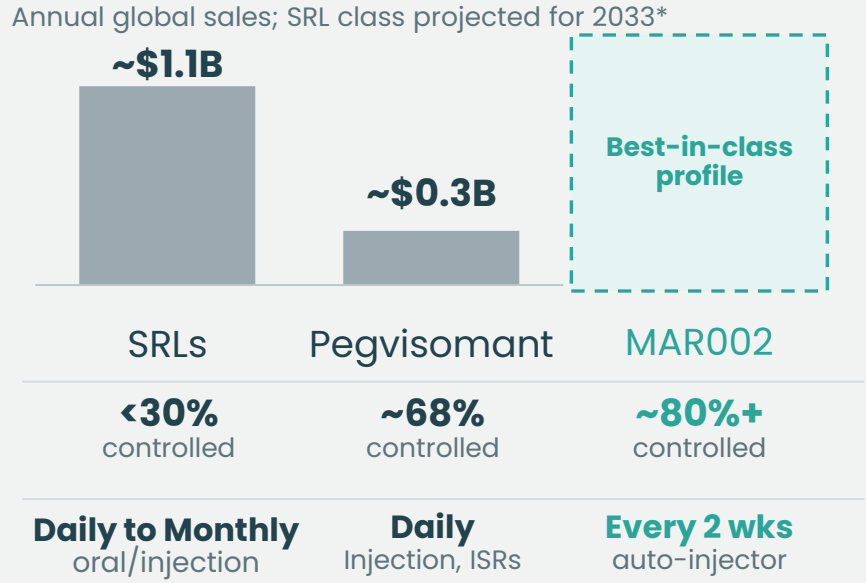
SRL: paltusotine, treatment-naive (n=22), Phase 3 PATHFNDR-2 (US label). GHRA: pegvisomant Phase 3, GHRA-naive (US label; Trainer, NEJM 2000). Market: Melmed & Giustina 2025; Fleseriu 2023; Moustaki 2023. Cross-trial comparisons have limitations.
Abbreviations: AE: adverse event; GHRA: growth hormone receptor antagonist; GI: gastrointestinal; SRL: somatostatin receptor ligand

MAR002: A Long-Acting GHRA Built to Fix Pegvisomant's Limits

MAR002 targets better control than SRLs with far less burden than pegvisomant

	Pegvisomant today	MAR002 target profile
Dosing	Daily injections (365 per year)	**Every 2 weeks (26 per year)**
Preparation	Vial and syringe reconstitution	**Auto-injector**
Tolerability	Injection-site reactions; LFT monitoring	**No LFT monitoring; minimal site reactions**
Efficacy	~68% IGF-1 control in real world	**~80%+ control in real world**

An optimal direct GHRA could be a large opportunity

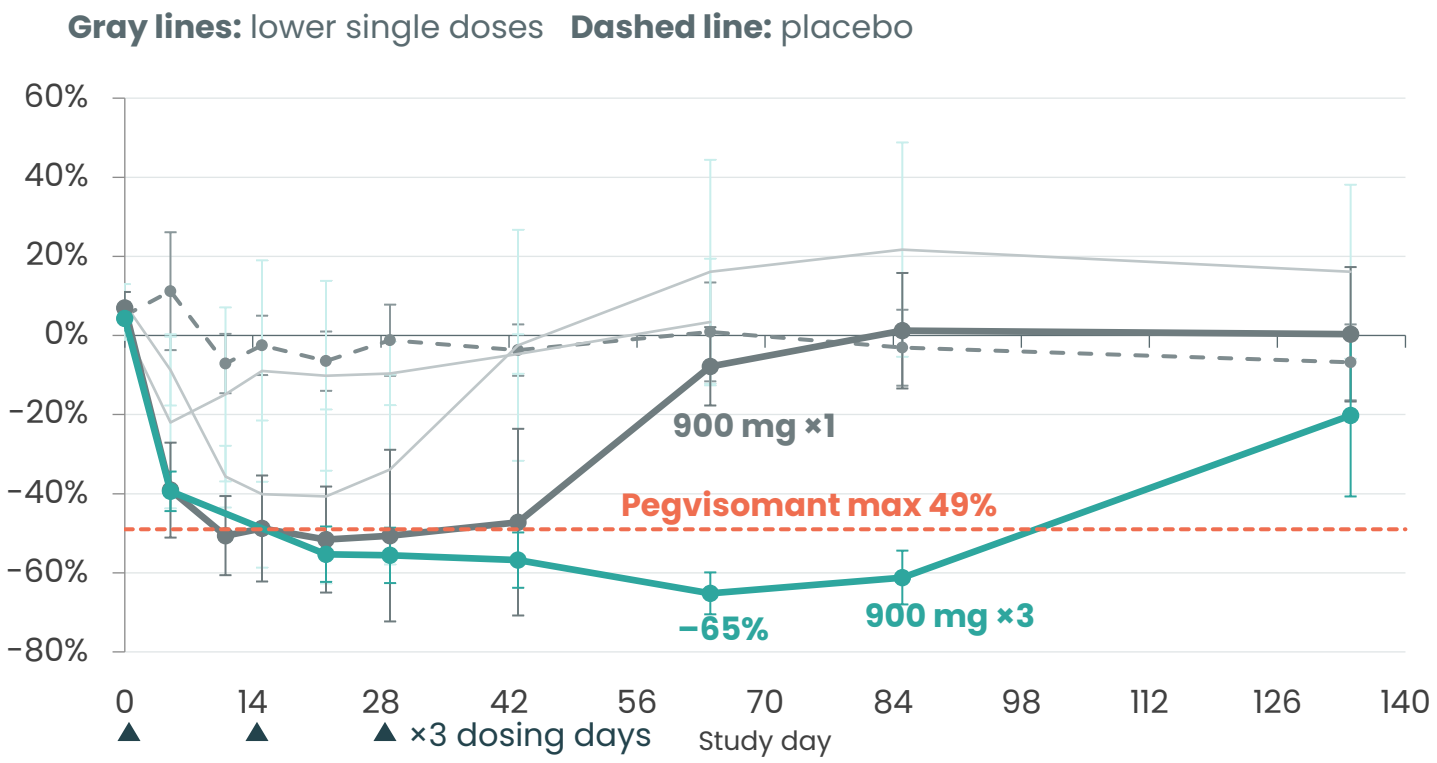


> *The daily injections were hard to sustain. Having that same level of control without the daily burden would be life-changing.*
>
> **Jill Sisco, President, Acromegaly Community**

*ClearView market research. MAR002 profile is a target. Sources: Melmed & Giustina 2025; Fleseriu 2023; Moustaki 2023; Carmichael 2014; Colao 2014; Trainer, NEJM 2000; ACROSTUDY registry (67.5% normal IGF-1); MAR002 Phase 1, Marea data on file.
Abbreviations: GHRA: growth hormone receptor antagonist; IGF-1: insulin-like growth factor 1; LFT: liver function test; SRL: somatostatin receptor ligand

Phase 1: Shows Deep, Durable IGF-1 Suppression and Supports Every-2-Week Dosing

Mean (±SEM) % change in IGF-1 by cohort in healthy volunteers: single doses 50–900 mg and 900 mg ×3



65%

Peak IGF-1 suppression
900 mg ×3 in HVs **vs 49% max** for daily pegvisomant in HVs

6 wks

Durable after a single dose
IGF-1 still 43% below baseline 6 weeks after one 900 mg dose (vs <4 days for pegvisomant)

✓

No safety signals
No SAEs, no ISR or LFT signals
Well tolerated at all doses (n=33 dosed)

PK/PD modeling supports 300 mg every 2 weeks to achieve ≥50% IGF-1 suppression

MAR-201 Phase 1, healthy men; central-lab IGF-1 (IDS-iSYS), mean ± SEM % change from baseline. 50 mg n=3; 150–900 mg n=6 each; 900 mg ×3 (Days 1, 15, 29) n=6; pooled placebo n=10. Selected visits shown; all visits in appendix. Pegvisomant: Thorner, JCEM 1999 (1.0 mg/kg SC, n=6). Cross-trial; not head to head.
Abbreviations: HV: healthy volunteer; IGF-1: insulin-like growth factor 1; ISR: injection-site reaction; LFT: liver function test; SAE: serious adverse event; SC: subcutaneous; SEM: standard error of the mean

MAR002: Emerging Data Supports Potential Best-in-Class Profile

Deeper, longer IGF-1 suppression than pegvisomant in healthy volunteers, with every-2-week dosing

	MAR002	Pegvisomant
In Vitro Potency with excess GH	90% suppression	24% suppression
Peak IGF-1 suppression Healthy volunteers	-64%	-49%
Days >45% suppressed Healthy volunteers	43 days	<4 days
Dosing Planned vs label	Every 2 weeks, SC	Daily SC injection
Tolerability Phase 1 vs label	No signals to date	LFT monitoring; ISRs

TROUGH-2 tests this profile in patients; topline Q4 2027

MAR002 (allosteric mAb): Kurylo, Endocrinology 2026; Marea Ph1 data on file (peak 900 mg ×3 SC; duration single 900 mg SC). Pegvisomant (competitive peptide): Thorner, JCEM 1999 (single 1.0 mg/kg SC, n=6); USPI 7/2023 (daily vial/syringe reconstitution). No head-to-head studies have been conducted; cross-trial comparisons are subject to inherent limitations and should be interpreted with caution.
Abbreviations: GH: growth hormone; IGF-1: insulin-like growth factor 1; ISR: injection-site reaction; LFT: liver function test; mAb: monoclonal antibody; SC: subcutaneous; USPI: US prescribing information

TROUGH-2 Phase 2 Will Set the Phase 3 Dose Across First- and Second-Line Patients

Randomized, double-blind, placebo-controlled; every-2-week dosing; first patient dosed Sep 30, 2026

Study design

~72 patients, active acromegaly
IGF-1 ≥1.3× ULN

Day 1 — **Week 10**

150 mg Q2W
300 mg Q2W
600 mg Q2W
Placebo

900 mg load; 3:1 vs placebo; then open-label extension

Primary: safety; % IGF-1 change
Secondary: AcroQoL, diary, ring size
Groups: untreated, pegvisomant washout, SRL/DA add-on

Study Goals

Beat SRL IGF-1 reduction

Match pegvisomant IGF-1 reduction

Q2W Delivery

Clean safety

Next: interim data mid-2027; topline Q4 2027; Phase 3 start H1 2028

MAR-202 protocol v2.1; IND 177488. Excludes patients controlled on SRL/DA or with HbA1c ≥9.0%. Pegvisomant: Trainer, NEJM 2000 (Wk 12 IGF-1 -50%/-63% vs -4% placebo). Paltusotine: Biller, JCEM 2026. DA: dopamine agonist; Q2W: every 2 weeks; SRL: somatostatin receptor ligand; ULN: upper limit of normal.

Multiple Value-Inflection Catalysts Through 2028

Two potentially first-in-class programs, both expected to be Phase 3-ready by early 2028

	Mid-2027	H2 2027	H1 2028
MAR001 / MAR005 sHTG	**MAR005 Phase 3 dose selection**	**TYDAL Part B sHTG topline + MAR005 Phase 3 start by end of 2027**	
MAR002 Acromegaly	**TROUGH-2 interim data**	**TROUGH-2 topline data (Q4 2027)**	**Phase 3 start**

Key data catalysts in 2027 to confirm profile and differentiation of both programs

Timing reflects current company expectations and is subject to change. sHTG: severe hypertriglyceridemia.

Thank You

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