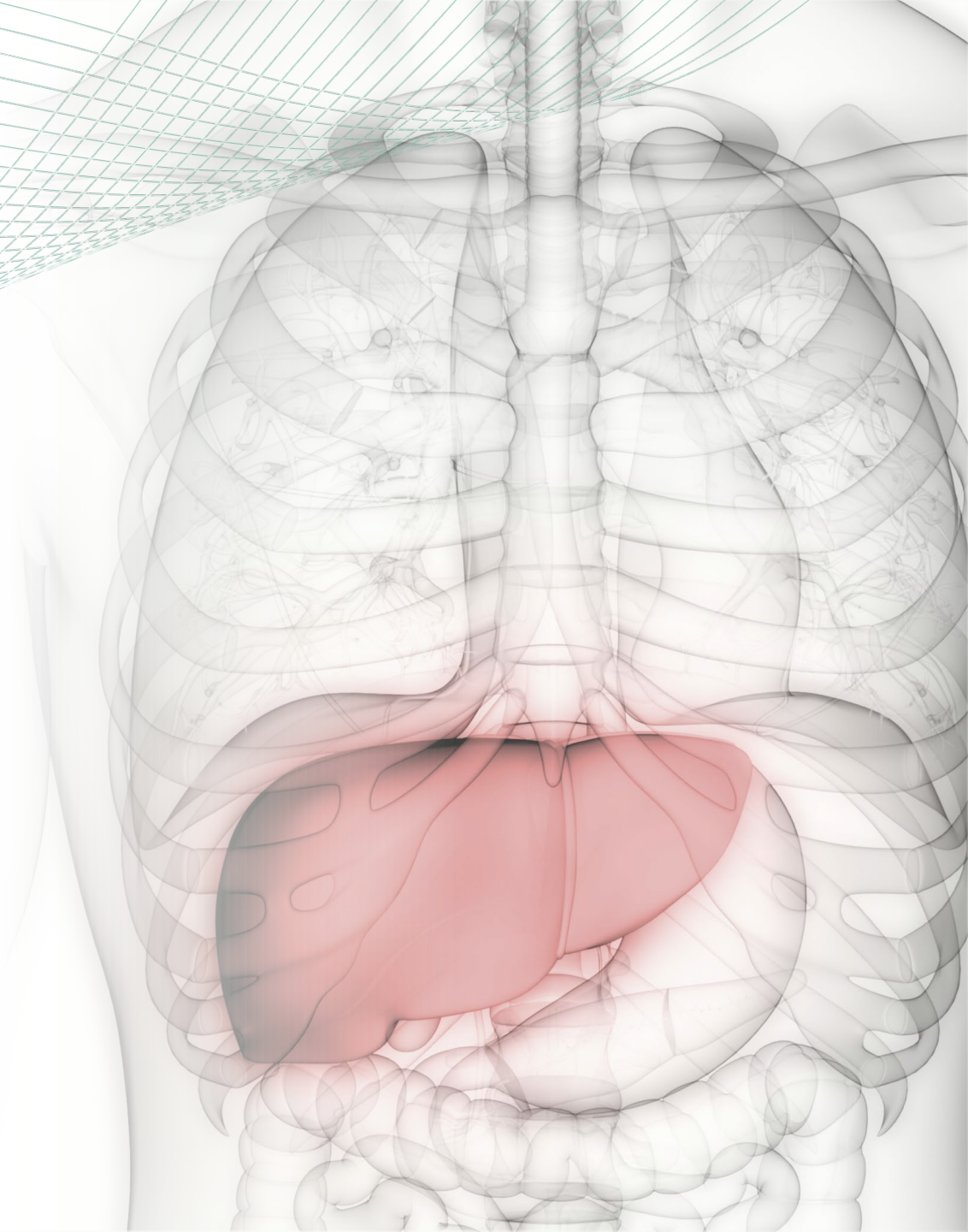
SAGIMET
BIOSCIENCES

Targeting Metabolic Dysfunction with Novel Therapies to Treat MASH, Acne & Cancer

March 2026



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Leadership Team with Proven Development and Commercialization Experience

Dave Happel *President & CEO*

>20 years of experience in executive leadership in biotech and pharma

Brought multiple innovative healthcare products to the market

Thierry Chauche *CFO*

>20 years of financial and operational leadership experience in finance and healthcare companies

Eduardo Martins *Chief Medical Officer*

>20 years of leadership of large-scale multinational clinical trials & global teams in pharma and biotech

Led clinical development team of cenicriviroc for MASH

Elizabeth Rozek *Chief Legal & Administrative Officer*

>20 years of legal experience including executive leadership of legal, IP and compliance functions in biopharma and biotech

Marie O'Farrell *Chief Scientific Officer*

>20 years of experience in R&D and translational medicine in biopharma and biotech

Successfully guided development for multiple clinical programs

Rob D'Urso *Senior Vice President, New Products*

>20 years of US and global leadership experience in dermatology

Pfizer raptor pharmaceuticals corp. MedImmune GILEAD DYNAVAX ALEXION Intercept U.S. Department of JUSTICE ISDIN

HORIZON AstraZeneca abbvie Genentech proventionbio NOVARTIS cognoa basilea PHARMACEUTICA Dr.Reddy's

Sagimet at a Glance

Unique MOA: FASN Inhibition

- Our lead molecule, denifanstat, is a novel fatty acid synthase (FASN) inhibitor with a differentiated MOA with the potential to target multiple underserved diseases
- Strong clinical data demonstrates denifanstat's proof of concept across multiple disease states

Denifanstat in MASH

- Denifanstat directly targets the 3 key drivers of MASH (metabolic dysfunction-associated steatohepatitis) – liver fat, inflammation, and fibrosis
- Successful outcome of Phase 2b trial; met both primary endpoints with significant reduction in fibrosis
- Pre-clinical data demonstrated synergistic effect of combination of FASN inhibitor and resmetirom
- Phase 1 pharmacokinetics (PK) clinical trial of a combination of denifanstat and resmetirom completed in December 2025, Phase 2 clinical combination trial with denifanstat and resmetirom in patients with MASH cirrhosis (F4) planned to initiate in 2H 2026

TVB-3567 in Acne

- Our follow-on FASN inhibitor, TVB 3567, received Investigational New Drug (IND) clearance in March 2025
- First-in-human Phase 1 clinical trial initiated in June 2025 for development of an acne indication

Strong IP, Cash Position, and Collaboration Potential

Strategic Collaboration with Ascletis in Acne

- Denifanstat met all primary and secondary endpoints in Phase 3 clinical trial in patients with moderate to severe acne vulgaris in China conducted by license partner for China, Ascletis
- Denifanstat was well-tolerated in Ascletis' open-label Phase 3 clinical trial
- Ascletis announced that Denifanstat NDA for the treatment of moderate to severe acne was accepted by China NMPA in December 2025

IP Portfolio

- Denifanstat:
  - Method of use patent—2036; potential PTE to 2041
  - Composition of matter patent—2032
- Combination of denifanstat and resmetirom:
  - Application filed 2024; if granted—2044; potential PTE to 2048
- TVB-3567:
  - Composition of matter patent—2035; potential PTE to 2038
  - Method of use application for TVB-3567 for acne filed 2025; if granted—2046

Cash Position

- Nasdaq: SGMT; $113.1M cash on hand*, expected to fund current operations through Q3 2027

*Cash, cash equivalents and marketable securities as of 12/31/2025

Development Pipeline: Multiple Indications and Clinical Milestones

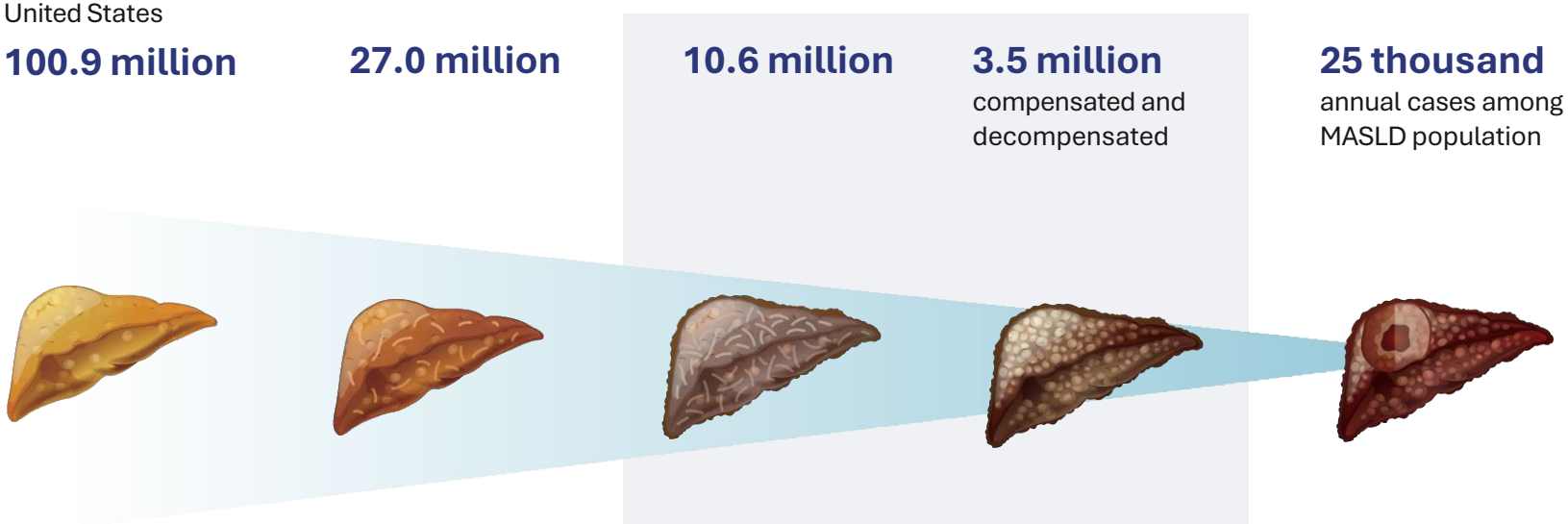
Therapeutic Area	Indication	Stage of Development	Expected Milestone / Status
Metabolic Disease	**MASH**	Denifanstat (Phase 2)	Phase 2b met histology primary and multiple secondary endpoints, data announced 1Q2024; FDA Breakthrough Therapy designation; Phase 3 ready (F2/F3 MASH)
		Denifanstat (Phase 1)	Phase 1 hepatic impairment results reported 1Q2024
		Denifanstat/resmetirom (Phase 1)	Phase 1 clinical PK trial completed in December 2025
Dermatology	**Acne**	TVB-3567 (Preclinical)	Phase 1 FIH initiated in June 2025
		歌礼 ascletis Denifanstat (ASC40) (Phase 3)	Phase 3 met all primary and secondary endpoints, data announced June 2025; NDA accepted by NMPA in December 2025*
Oncology	**Solid tumors**	TVB-3567 (Preclinical)	Identifying FASN-dependent tumor types for potential FASN inhibitor development
		Denifanstat (Phase 1)	

* Trial conducted in China by Ascletis, who has licensed development and commercialization rights to all indications in Greater China.

MASH: A Burgeoning Epidemic

Estimated Patients in 2030[1]

United States



MASH

- Complex disease with heterogeneous patient population
- Significant opportunity for differentiated MOA

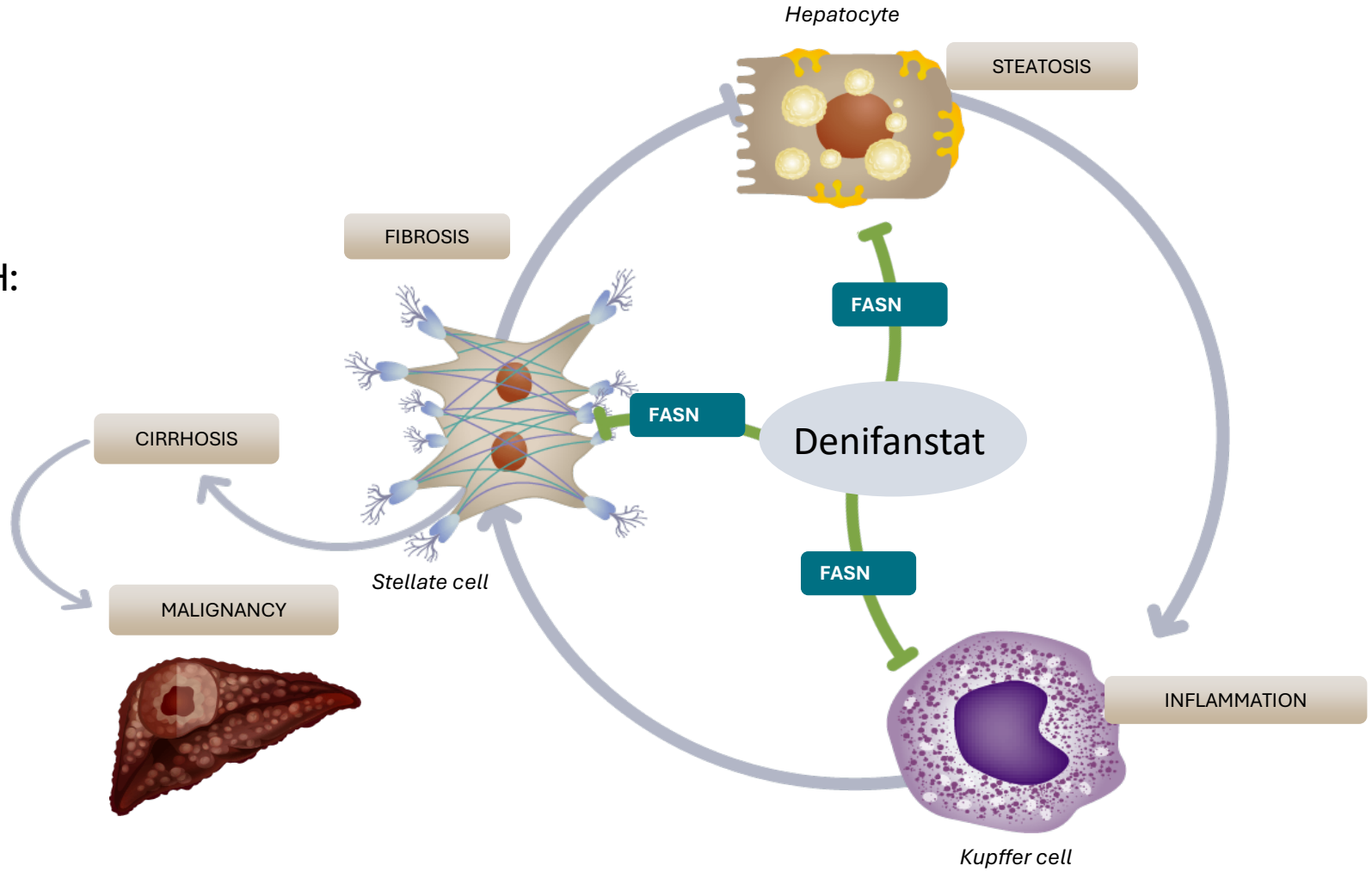
Note: MASH, or metabolic dysfunction-associated steatohepatitis, was formerly known as NASH, or nonalcoholic steatohepatitis.

1. Estes C, et al. *J Hepatol.* 2018;69(4):896-904.

FASN Inhibition Addresses Three Independent Mechanisms of MASH Development and Progression

Sagimet's lead drug candidate, denifanstat, is a specific and potent inhibitor of FASN that functions through three independent mechanisms in MASH:

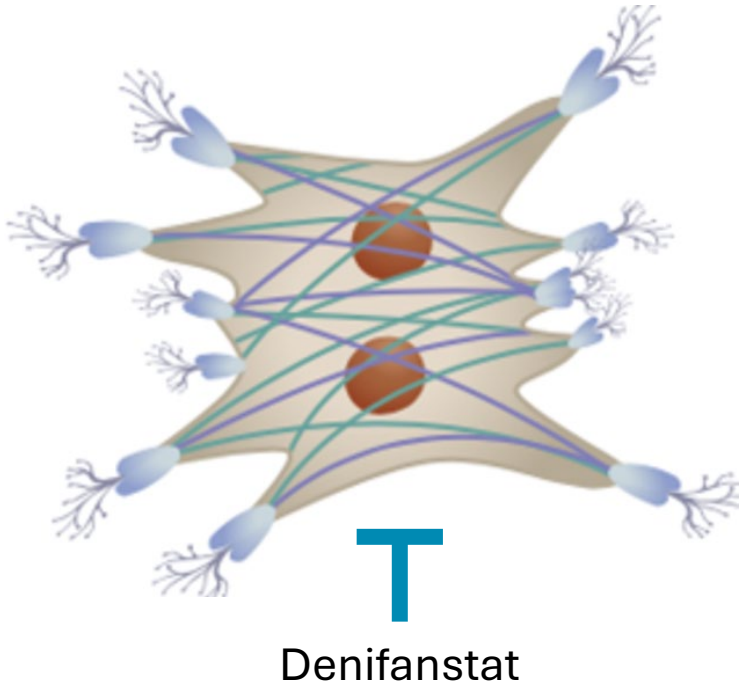
1. Blocking **steatosis** via inhibiting de novo lipogenesis in hepatocytes
2. Reducing **inflammation** via preventing immune cell activation
3. Blunting **fibrosis** via inhibiting stellate cell activation



FASN Inhibition Directly Blocks Human Liver Stellate Cell Function

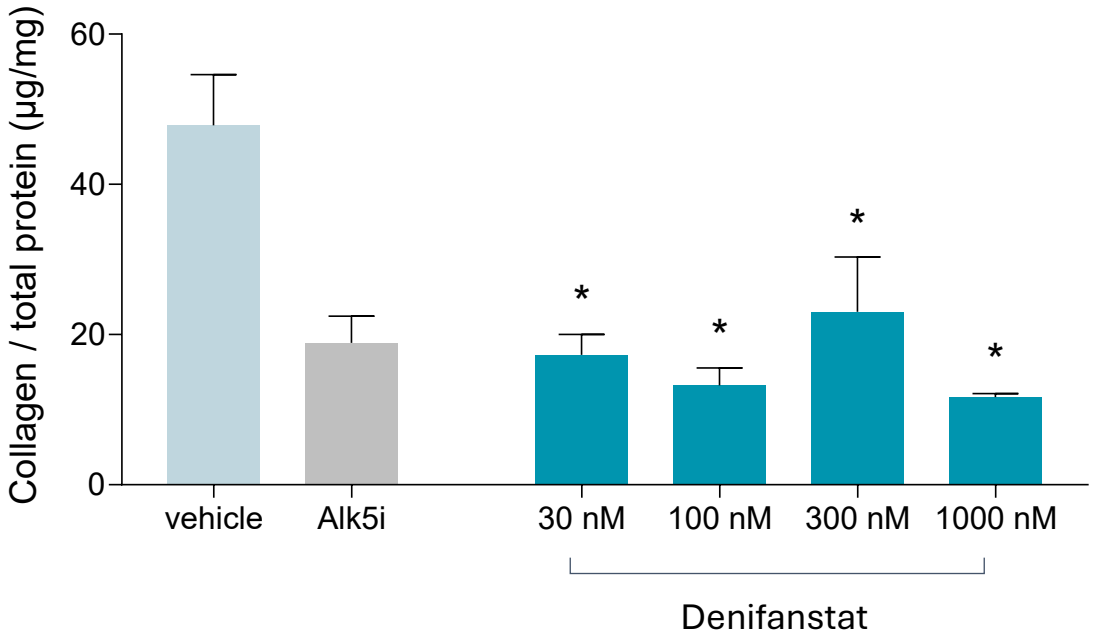
Stellate cells require DNL for fibrogenesis

Denifanstat blocks stellate cell activation



Denifanstat directly inhibits fibrogenic activity[1]

Primary human stellate cell assay

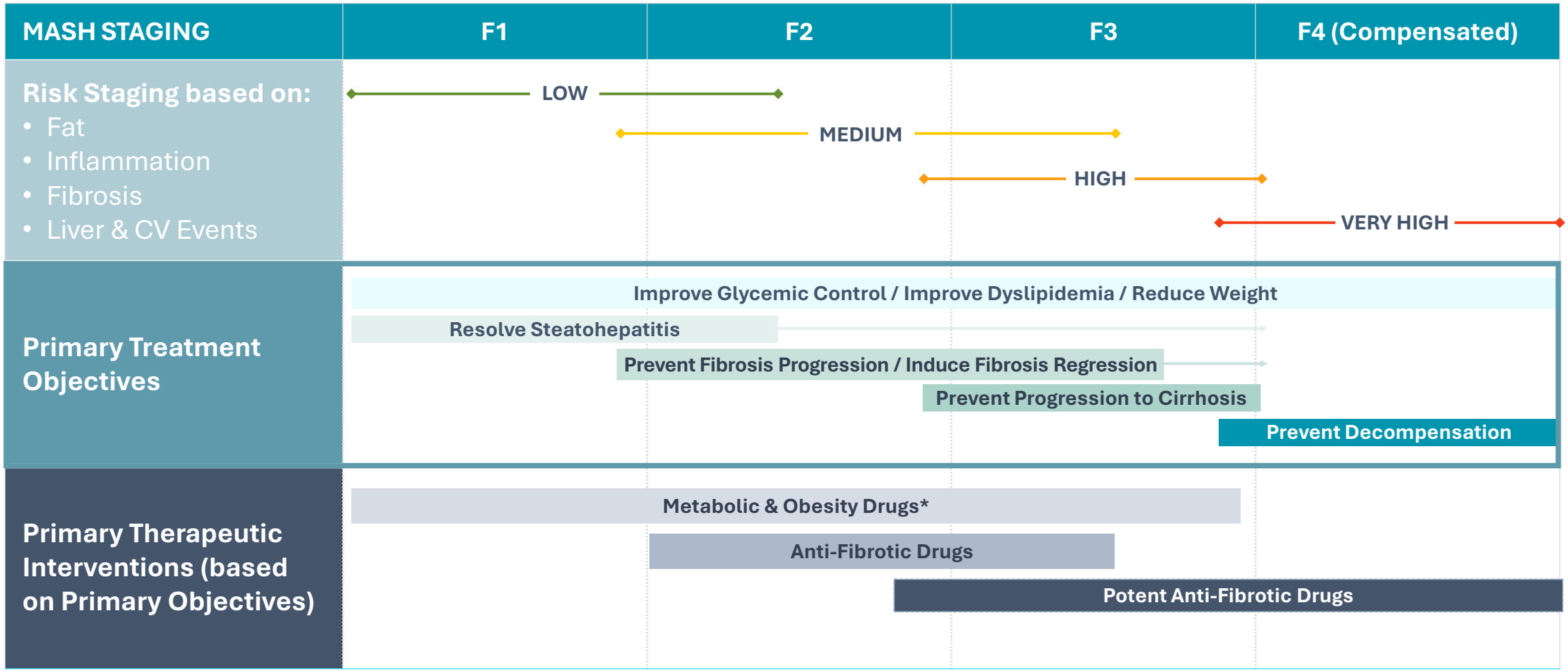


- Stimulated by TGF-beta to activate fibrogenesis
- Denifanstat showed similar inhibition to positive control ALK5 inhibitor

*p<0.05. DNL: de novo lipogenesis

1. O'Farrell M, et al. *Sci Rep*. 2022;12(1):15661. Sagimet Biosciences data on file.

Treatment Goals for MASH Across Fibrosis Staging



Cusi K, et al. *Endocr Pract*. 2022;28(5):528-562. Rinella ME, et al. *Hepatology*. 2023;77(5):1797-1835. EASL, et al. *J Hepatol*. 2024;81(3):492-542.
*Metabolic drugs are anticipated to be background therapy for obesity and type 2 diabetes, and earlier stages of fibrosis

Strong MASH Data Creates Opportunities to Reach Advanced Patient Populations

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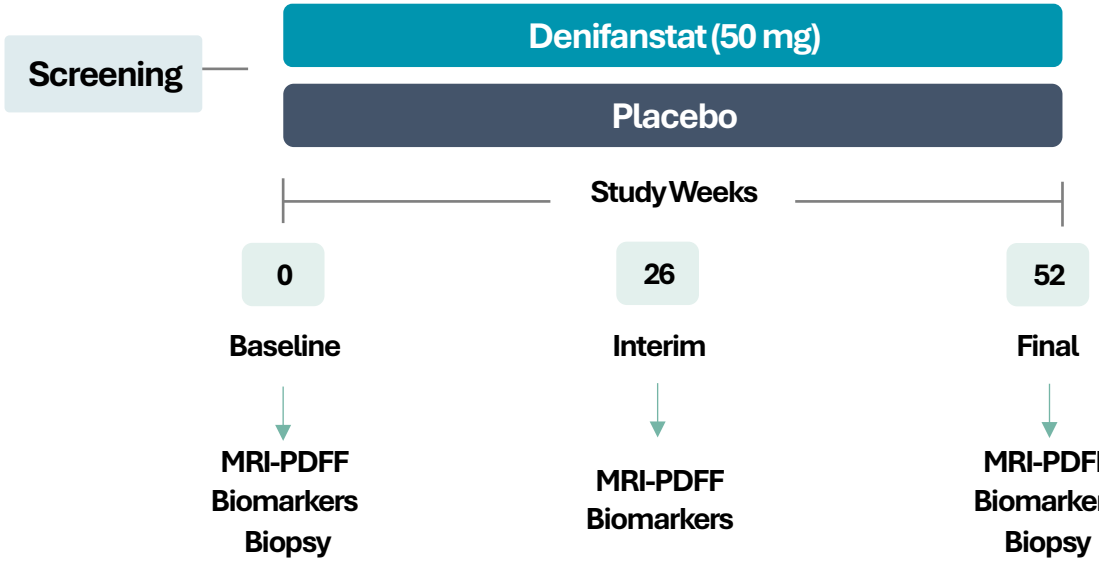
FASCINATE-2: Biopsy Trial Design Focused on Histological Endpoints

FASCINATE-2 Phase 2b trial design



- Biopsy confirmed F2-F3 MASH patients (n=168)
- 52 weeks, 2:1 randomization to 50mg or placebo, double-blind
- Single pathology reader: Dr. Pierre Bedossa
- AI digital pathology: HistoIndex

Primary endpoints

- NAS ≥2 points improvement w/o worsening of fibrosis
- MASH resolution + NAS ≥2 improvement w/o worsening of fibrosis

Selected secondary endpoints

- Improvement in liver fibrosis ≥1 stage without worsening of MASH as assessed by biopsy
- Digital AI pathology
- MRI-PDFF: absolute decrease, % change from baseline, % pts ≥30% reduction from baseline (responders)

AI: Artificial Intelligence, MRI-PDFF; magnetic resonance imaging derived proton density fat fraction, NAS; NAFLD Activity Score.

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100

FASCINATE-2: Baseline Characteristics Were Typical of the F2/F3 MASH Population

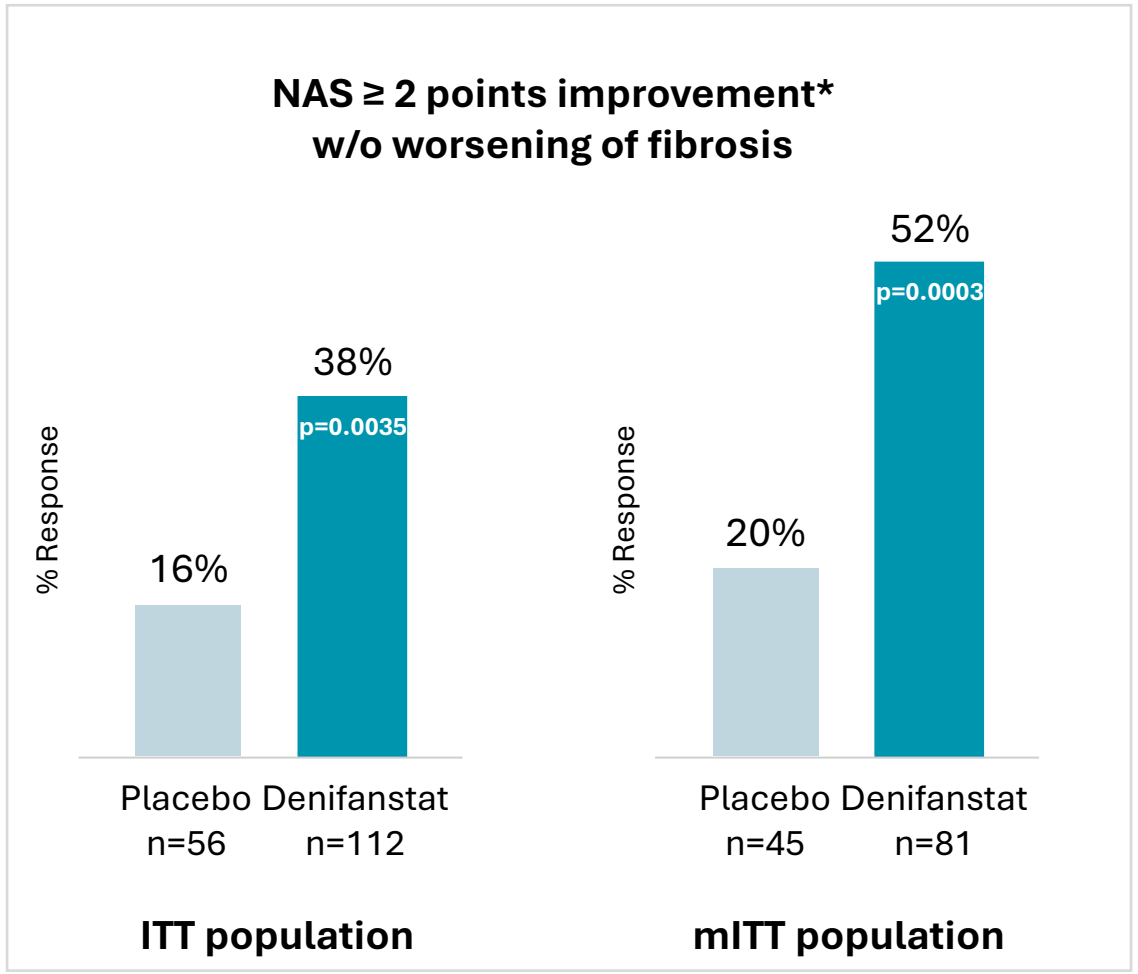
Parameter	Placebo, n=45	Denifanstat, n=81
Age, years	59.6 (+/- 10.9)	56.1 (+/- 10.8)
Sex, female	27 (60%)	48 (59%)
Race, White	41 (91%)	73 (90%)
Ethnicity, Hispanic or Latino	15 (33%)	27 (33%)
BMI, kg/m^2	36.5 (+/- 6.7)	34.6 (+/- 6.1)
Type 2 diabetes	27 (60%)	55 (68%)
ALT (alanine aminotransferase) U/L	67 (+/- 33)	57 (+/- 29)
AST (aspartate aminotransferase) U/L	52 (+/- 27)	48 (+/- 29)
Liver Fat Content (MRI-PDFF), %	19.0 (+/- 7.0)	16.6 (+/- 7.1)
Baseline liver biopsy NAS ≥ 5	34 (76%)	63 (78%)
Baseline liver biopsy F2/F3	22 (49%) / 23 (51%)	34 (42%) / 47 (58%)
Statin (at baseline)	21 (47%)	38 (47%)
GLP1-RA (at baseline)	4 (9%)	12 (15%)
LDL, mg/dL	103 (+/- 39)	96 (+/- 34)
Triglycerides, mg/dL	153 (+/- 67)	173 (+/- 79)
ELF (Enhanced Liver Fibrosis) Score	9.8 (+/- 0.8)	9.6 (+/- 0.8)
FAST (Fibroscan AST) Score	0.6 (0.19)	0.6 (0.20)

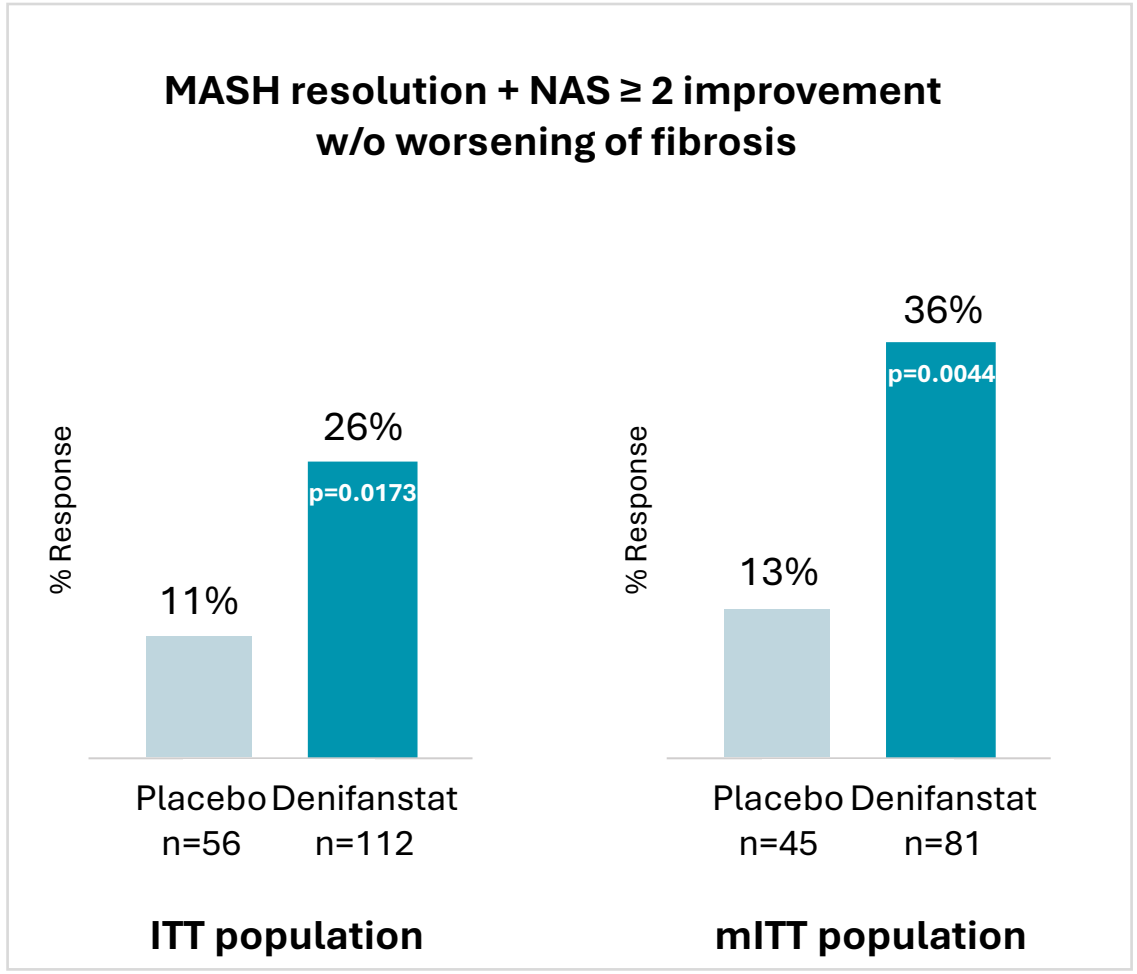
Modified intent-to-treat population (mITT) includes all patients with paired biopsies. Data are mean (SD) or n (%)

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100

Primary Endpoints: Liver Biopsy

Denifanstat Achieved Statistical Significance at Week 52



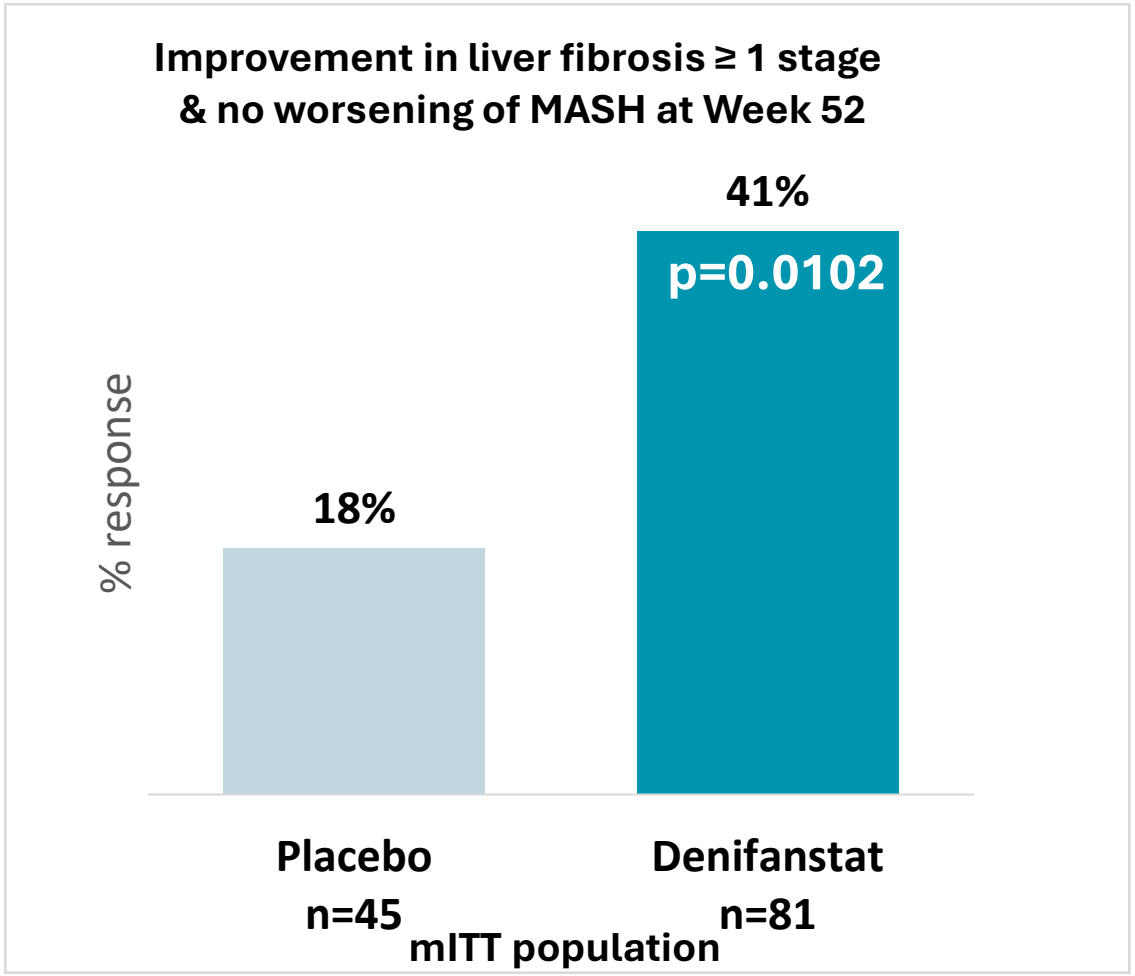


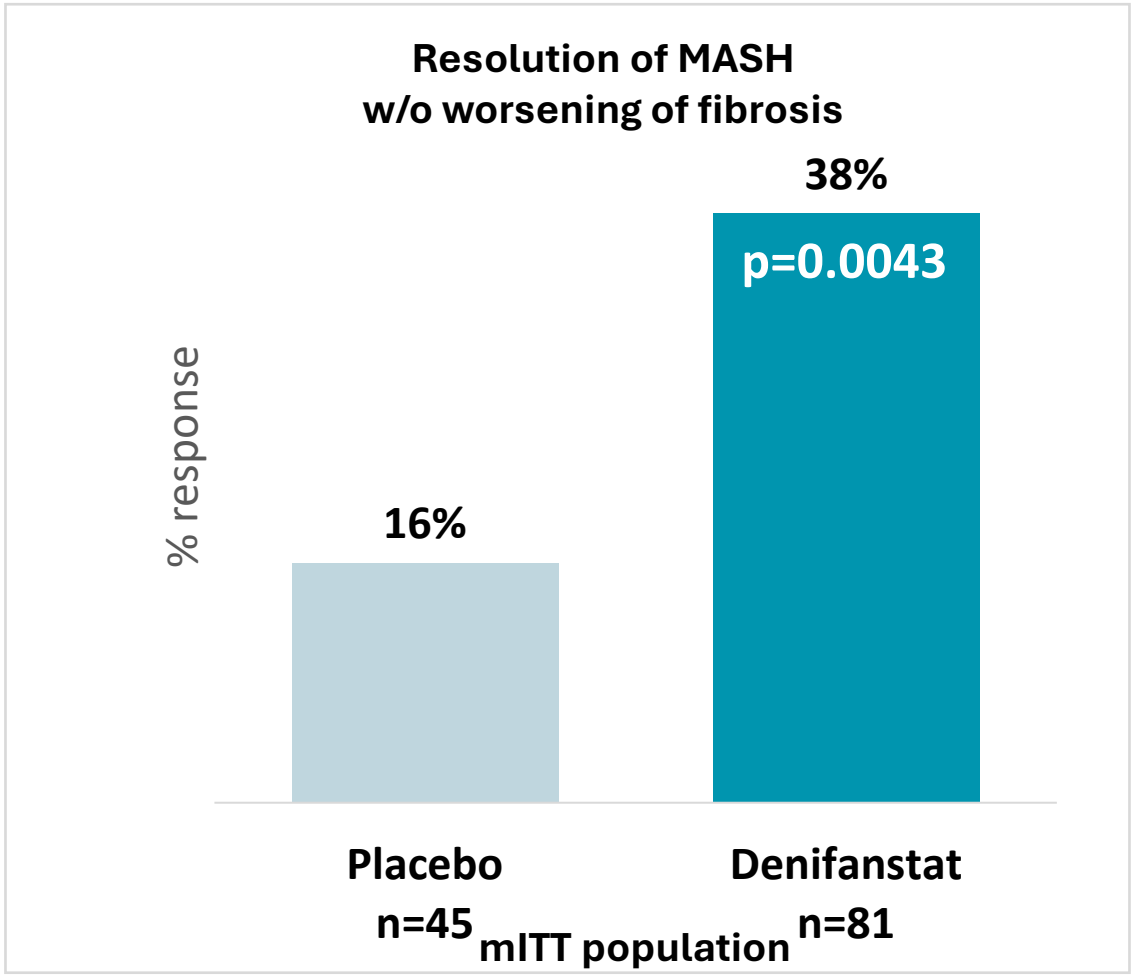
Cochran-Mantel-Haenszel Test – two sided at the 0.05 significance level. * ≥1-point improvement in ballooning or inflammation.

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100

Secondary Endpoints: Liver Fibrosis and MASH Resolution

Denifanstat Achieved Statistical Significance at Week 52





Cochran-Mantel-Haenszel Test – Two sided at the 0.05 significance level

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100

Secondary Endpoints: Liver Fibrosis

Denifanstat Achieved Statistically Significant Improvement of Fibrosis

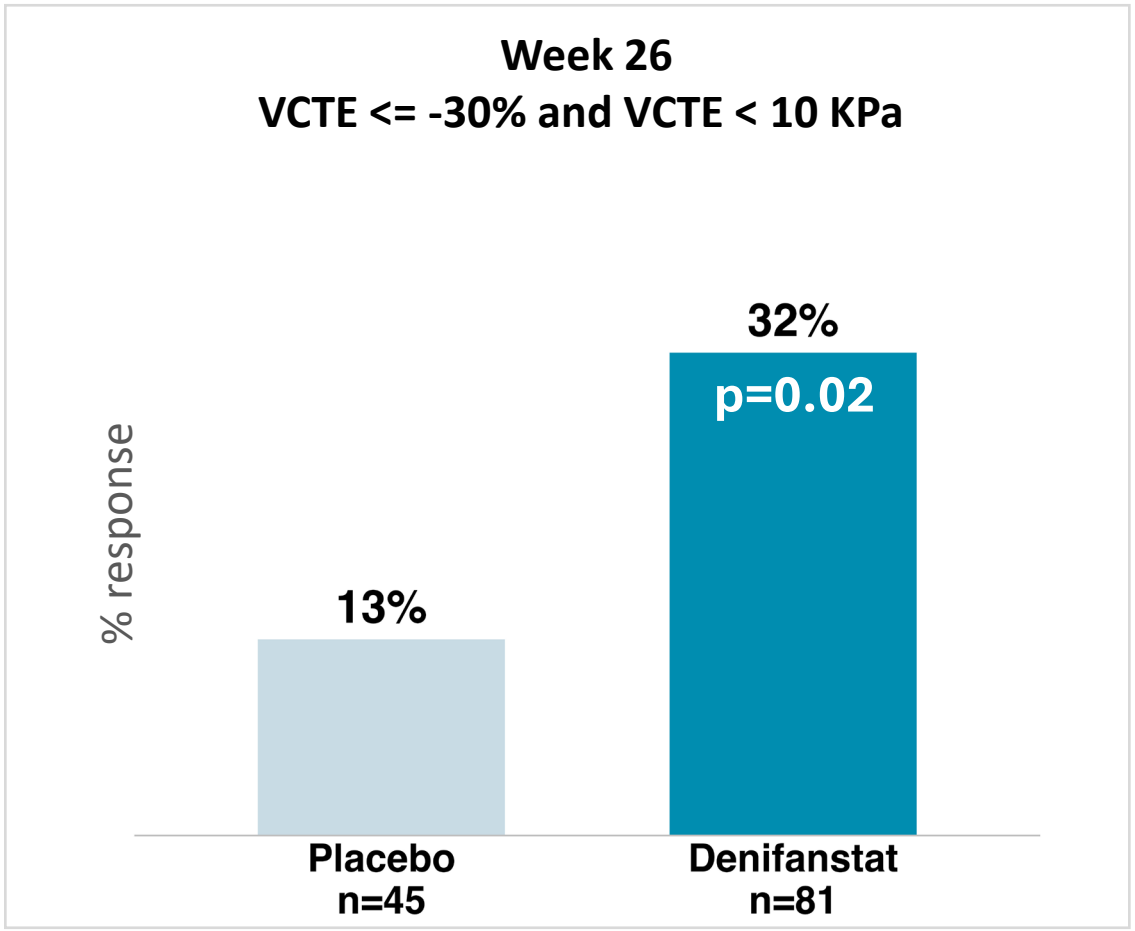
Fibrosis Endpoints	Subgroup	Placebo	Denifanstat	p-value
≥1 stage improvement in fibrosis w/o worsening of MASH	ITT	14%	30%	0.040**
	mITT	18%	41%	0.0102**
	F3	13%	49%	0.0032**
≥2 stage improvement in fibrosis w/o worsening of MASH	mITT	2%	20%	0.0065**
	F3	4%	34%	0.0065**
Progression to MASH cirrhosis (F4)	mITT	11%	5%	0.0386*

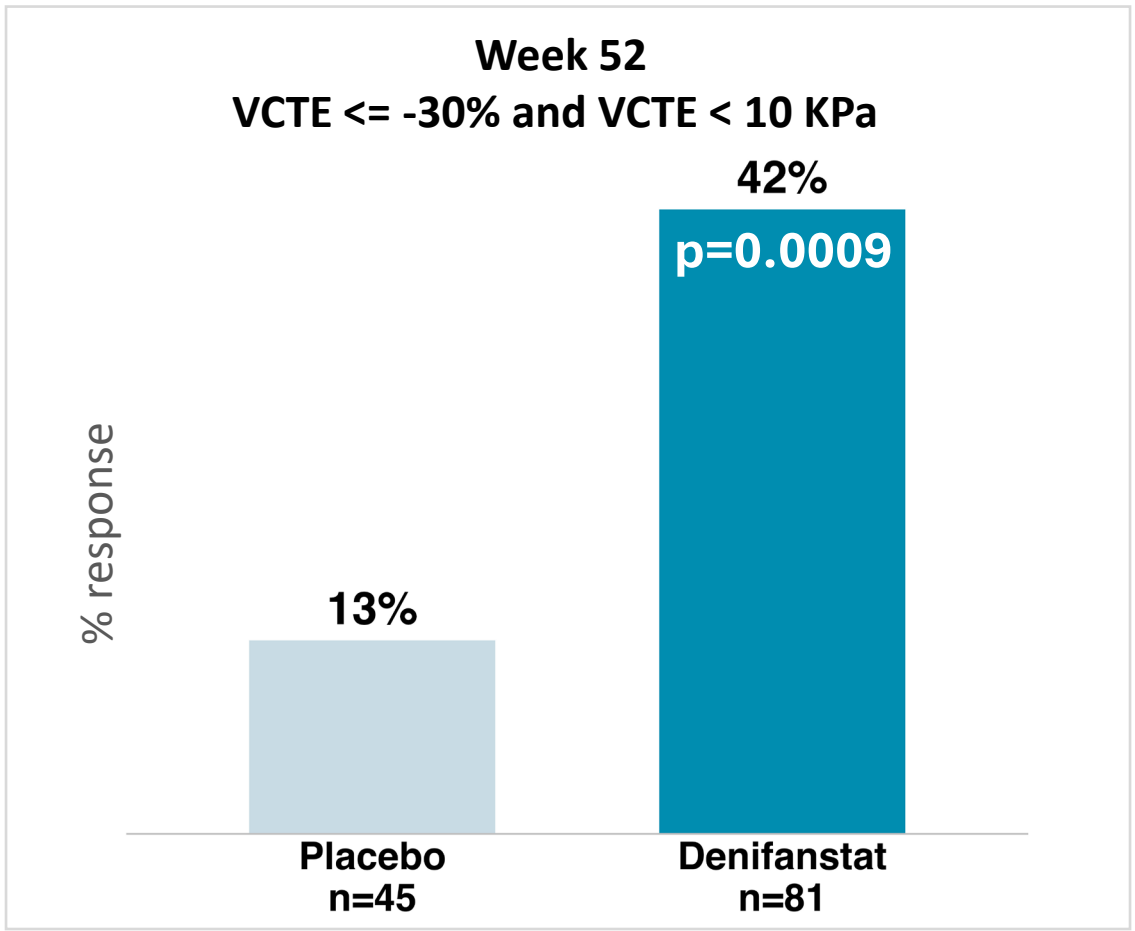
*One sided at the 0.05 significance level, **Two sided at the 0.05 significance level

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100.
Loomba R, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/Denifanstat_a_fatty_acid_synthase_FASN_inhibitor_shows_significant_fibrosis_improvement_and_MASH_resolution_in_FASCINATE-2_a_Ph2b_52_week.pdf

Denifanstat Achieved Statistically Significant VCTE Improvement at Weeks 26 and 52

FASCINATE-2 Phase 2b



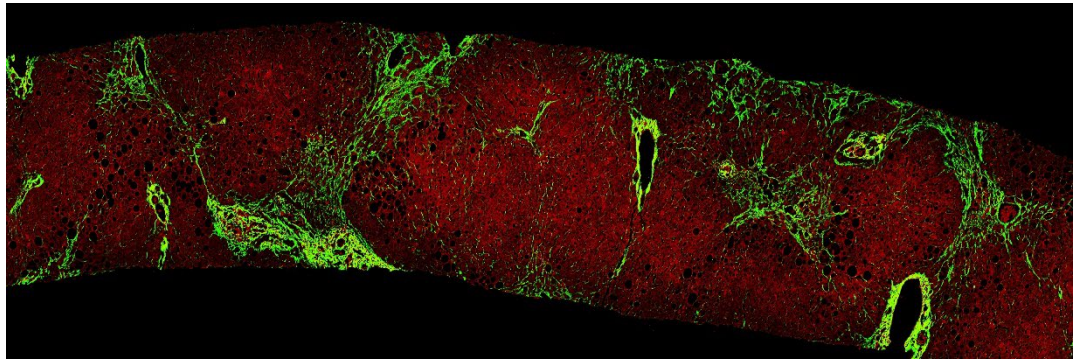


Sagimet Biosciences data on file. FASCINATE-2 posthoc analysis. mITT population. Chi-square test. VCTE: Vibration-controlled transient elastography. VCTE <=30% means magnitude of decline from baseline

Additional Fibrosis Analysis Using AI-based Digital Pathology

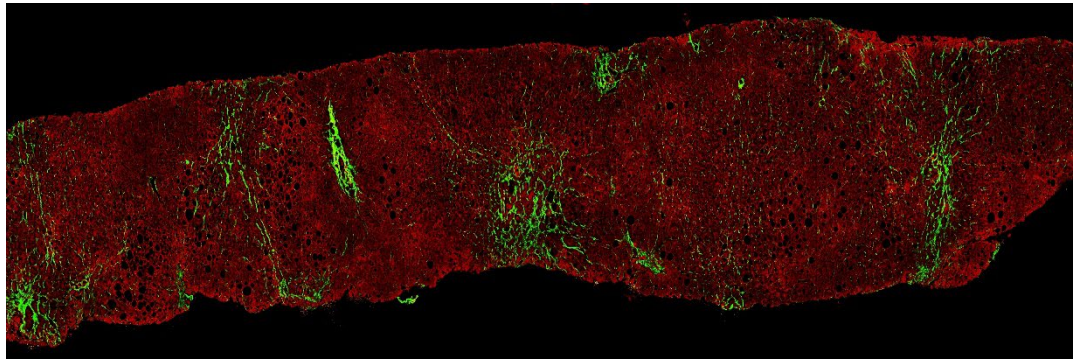
Digital Imaging Showed that Denifanstat Significantly Reduced Fibrosis in Advanced Patients

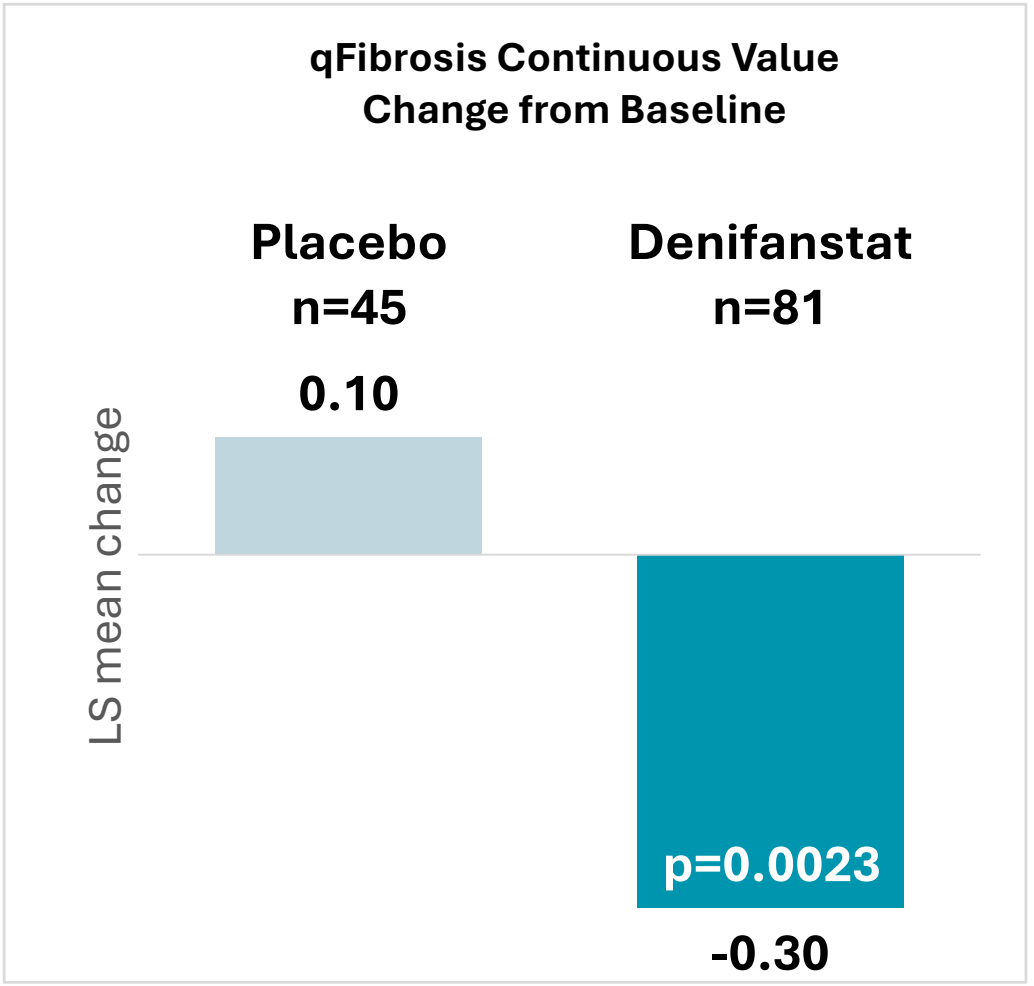
Pre-Treatment Pt A
NASH-CRN Fibrosis stage F3



Denifanstat

Post-Treatment Pt A
NASH-CRN Fibrosis stage F1

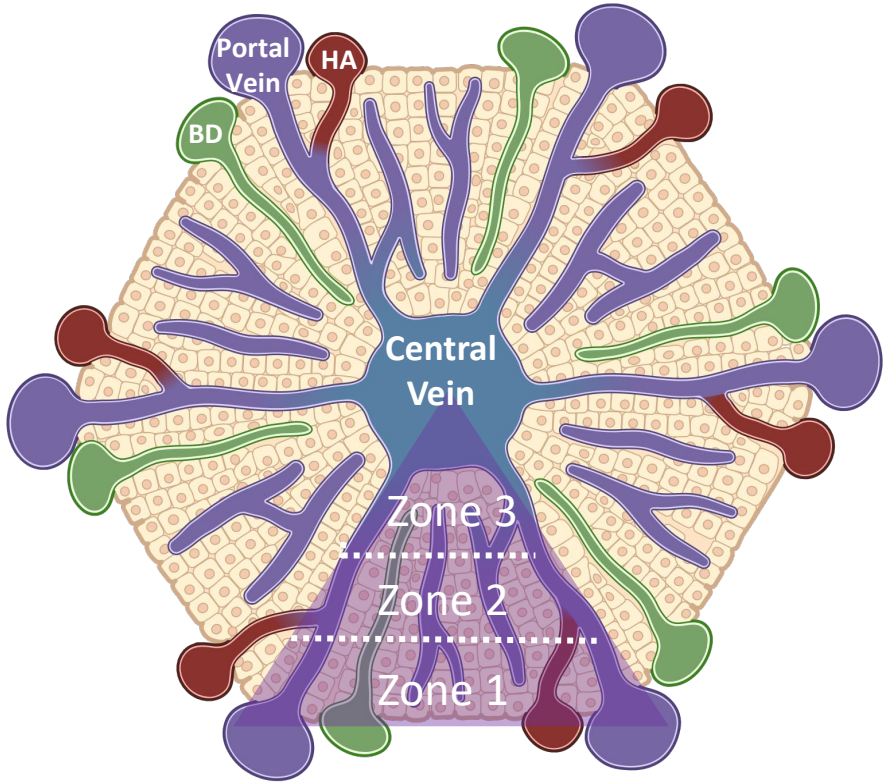




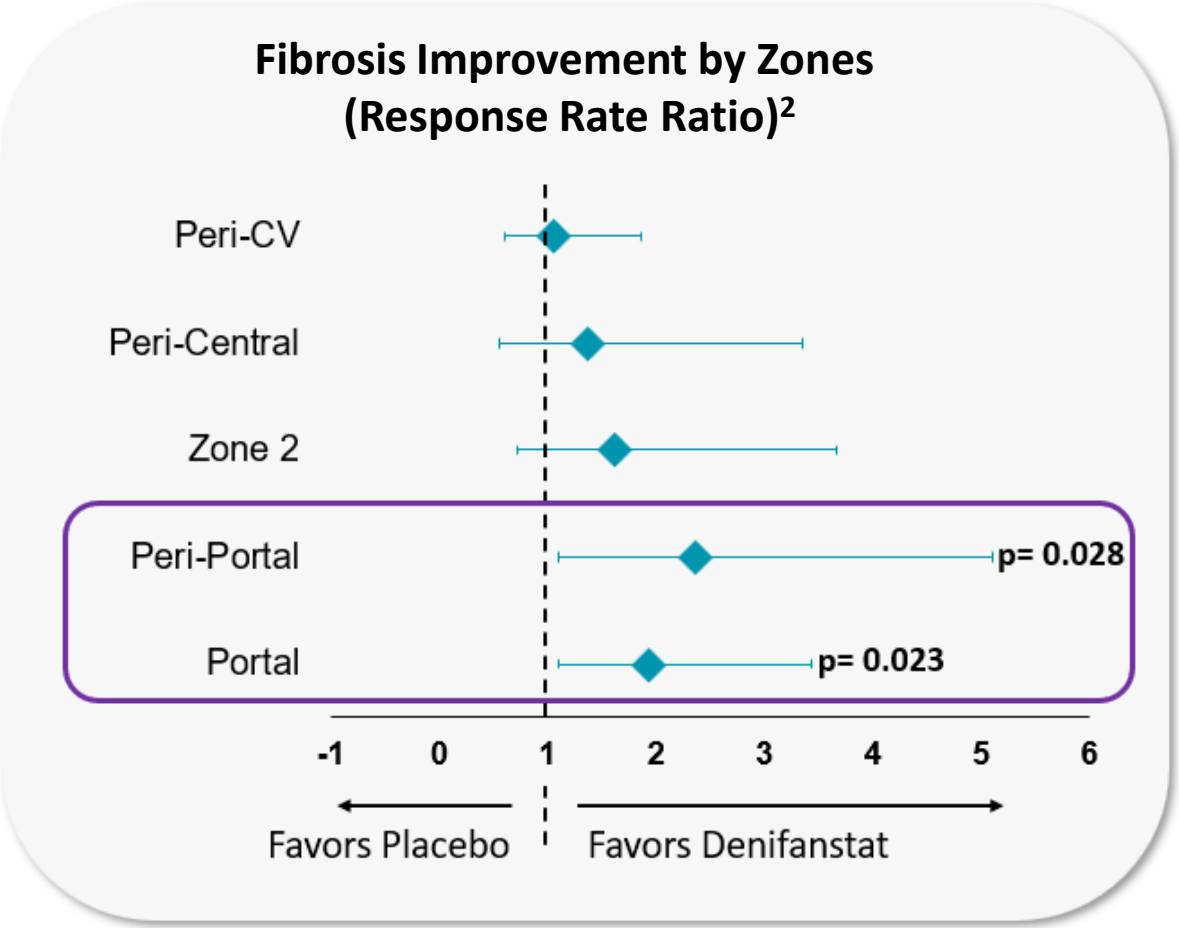
*One sided at the 0.05 significance level

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100

qFibrosis Zonal Analysis Demonstrated that Denifanstat Improves Parameters Linked to Liver Outcomes



Changes in periportal and portal zones have been correlated with liver outcomes and mortality by analysis of liver biopsies (n=452) from SteatoSITE study[1]



1. Kendall TJ, et al. *Liver Int.* 2024;44(10):2511-2516.

Response at the individual zonal parameter level was defined as "at least" 30% relative decrease from baseline.
2. Rinella M, et al. Presented at: AASLD 2024; November 15-19, 2024; San Diego, CA. Abstract 0170.

Denifanstat Potential in Patients with MASH Cirrhosis (F4)

Differentiated Mechanism of Action

- *In vitro* data demonstrates that denifanstat reduces pro-fibrotic signaling in stellate cells, suggesting that denifanstat has the potential to remove fibrotic scar tissue and reestablish the basal extracellular matrix (ECM) scaffold even in patients with MASH cirrhosis (F4)[1]
- Hepatocytes continue to be functional, and patients frequently have increased liver fat

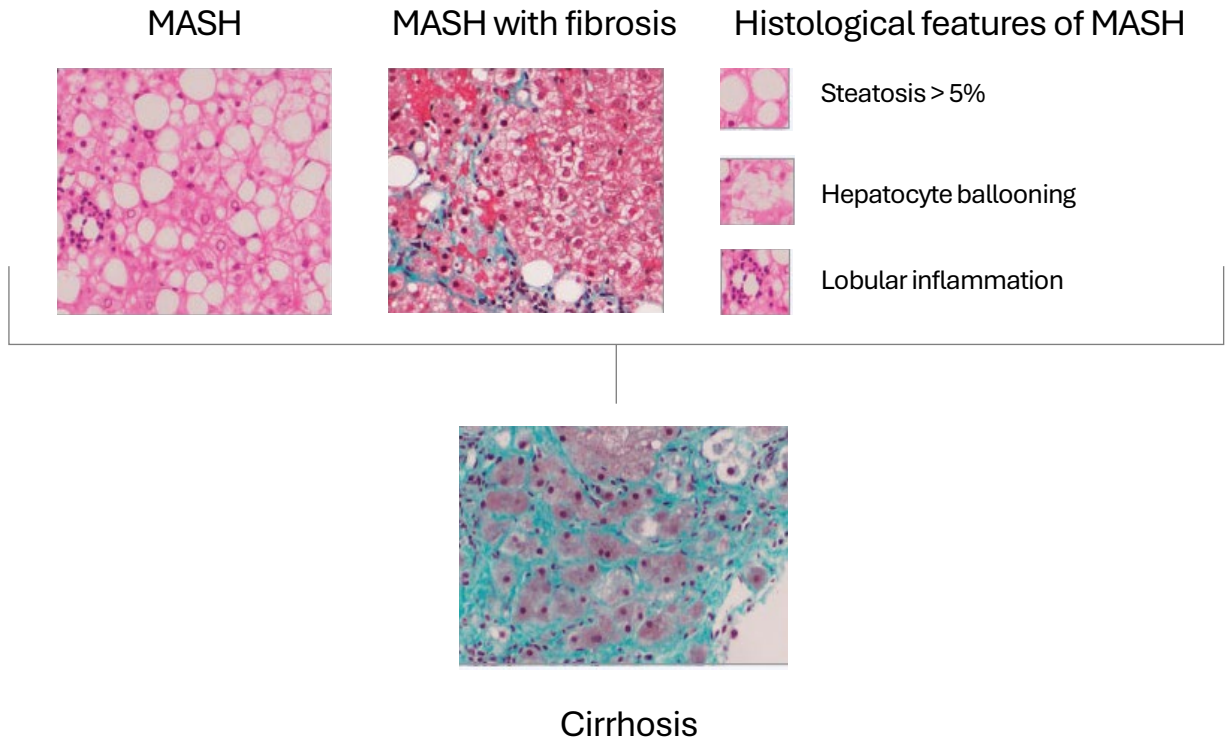
Clinical Data

- PK profiles in patients with MASH cirrhosis (F4) in the Phase 1 impaired hepatic function study[3]
- Positive impact on advanced fibrosis in patients in FASCINATE-2[4], including qF4 (quantification of fibrosis stage 4) patients based on AI-based digital pathology [5]

Next Step

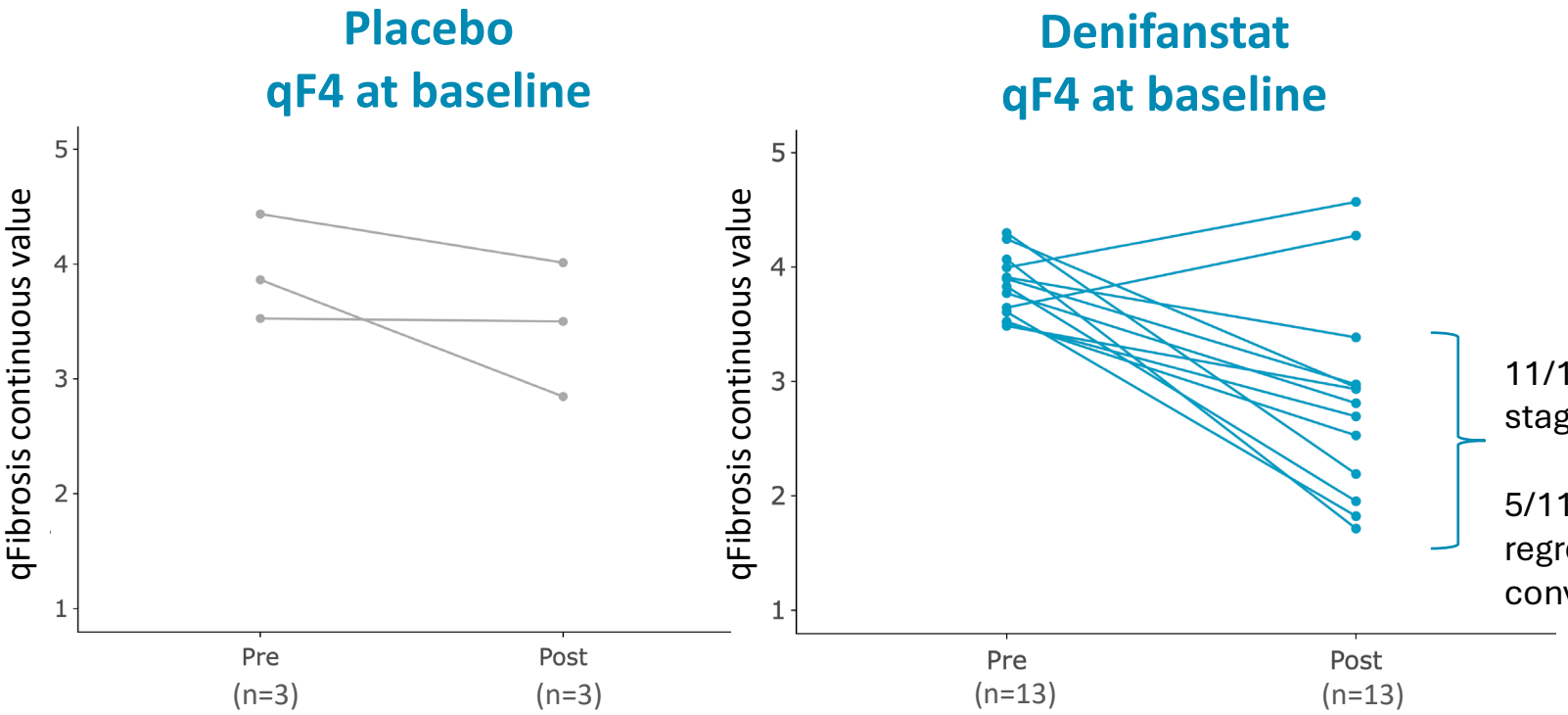
- Potential Phase 2 proof of concept in patients with MASH cirrhosis (F4)

~20% of Patients Progress to Cirrhosis [2]



1. Kamm DR, McCommis KS. *J Physiol.* 2022;600(8):1825-1837. 2. Sheka AC, et al. *JAMA.* 2020;323(12):1175-1183. 3. Sagimet Biosciences data on file. CLIN-009. 4. Loomba R, et al. *Lancet Gastroenterol Hepatol.* 2024;9(12):1090-1100. 5. Sagimet Biosciences data on file. FASCINATE-2 HistoIndex.

85% of qF4 Patients on Denifanstat Showed 1 to 2-Stage Reductions in Fibrosis



11/13 (85%) qF4 patients decreased by 1 or 2 qfibrosis stages measured by AI-based pathology

5/11 of qF4 patients showed ≥1 stage fibrosis regression, with 4 of these being 2-stage, measured by conventional pathology

- AI may detect fibrosis regression at an earlier point in time, compared to conventional pathology
- qF4 population (defined on AI platform by HistoIndex) are likely the most advanced subgroup of F3 patients in Phase 2b study

Sagimet Biosciences data on file. FASCINATE-2 HistoIndex.

FASCINATE-2: Safety

Denifanstat Was Generally Well-Tolerated

Event n (%)	Placebo (n=56)	Denifanstat 50mg (n=112)	Total (n=168)
Any adverse event (AE)	46 (82.1)	99 (88.4)	145 (86.3)
Adverse event related to denifanstat or placebo	20 (35.7)	51 (45.5)	71 (42.3)
Serious adverse event	3 (5.4)	13 (11.6)	16 (9·5)
TEAE leading to study drug discontinuation	3 (5.4)	22 (19.6)	25 (14.9)
Adverse events affecting ≥ 10% of patients			
COVID-19	6 (10.7)	19 (17.0)	25 (14.9)
Dry eye	8 (14.3)	10 (8.9)	18 (10.7)
Hair thinning	2 (3.6)	21 (18.8)	23 (13.7)

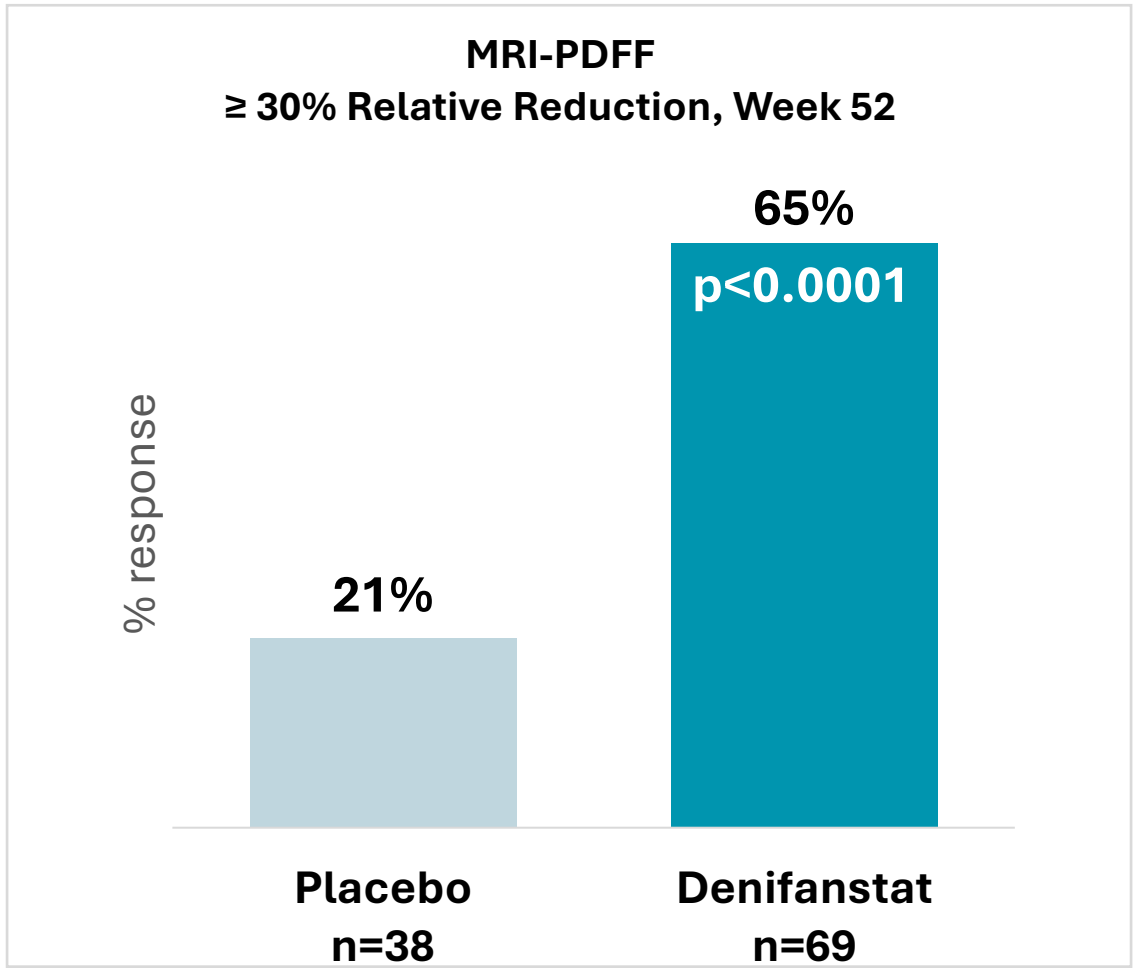
- No DILI (drug-induced liver injury) signal and no muscle wasting were detected, and GI (gastrointestinal) effects were comparable to placebo
- AE of hair thinning stabilized with a 2-to-4 week dose pause and then reversed with down titration or study completion
  - Only 7% of patients discontinued from the study with treatment-related hair thinning. Hair thinning in patients receiving GLP-1 ranges from 7% to 10% [1,2]
  - In two previous clinical studies of denifanstat, 2% of the patients on denifanstat experienced hair thinning at 50mg [3]

1. Wadden TA, et al. *Nat Med*. 2023;29(11):2909-2918. 2. Daniel S, et al. *J Drugs Dermatol*. 2025;24(4):413-415. 3. Sagimet Biosciences data on file. FASCINATE-1. Phase 2a study of denifanstat in acne conducted by Ascletis in China

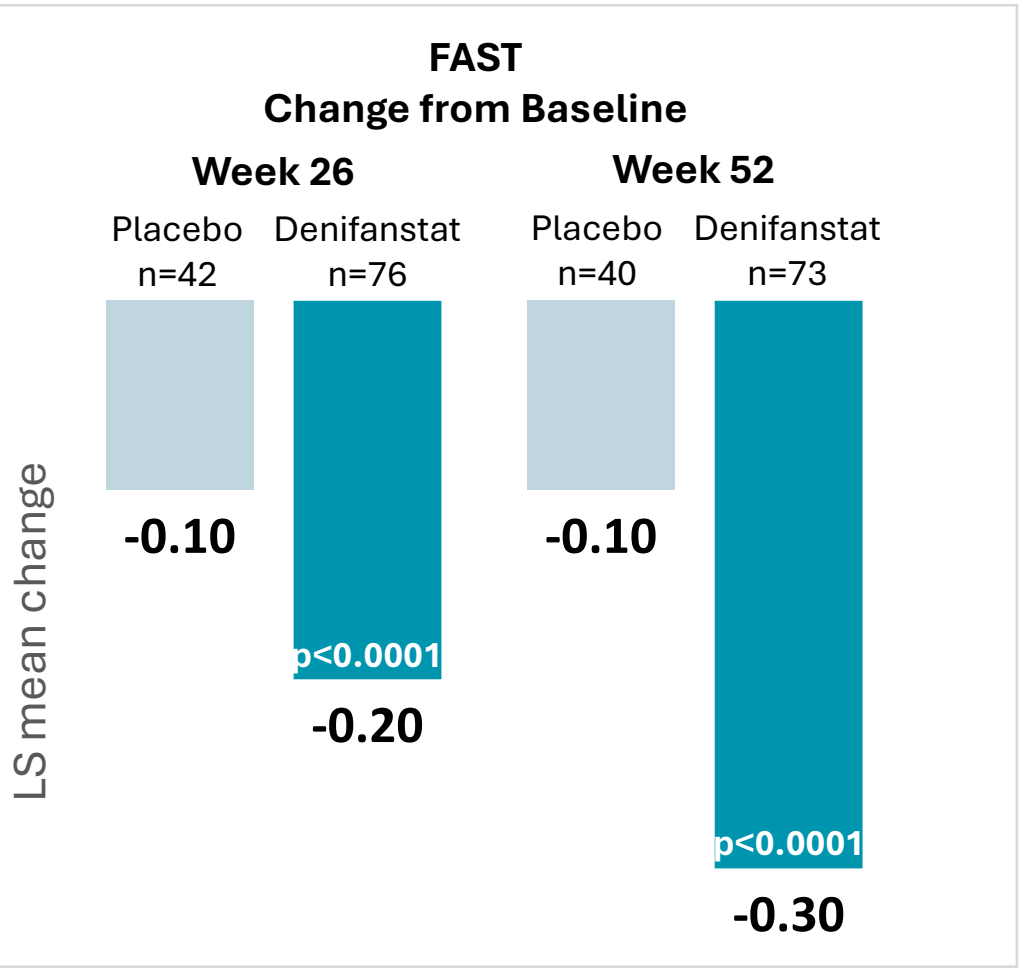
Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100

Denifanstat Decreased Liver Fat by MRI-PDFF and Reduced FAST Score

Denifanstat Achieved Statistical Significance



≥30% reduction: Cochran-Mantel-Haenszel Test. Relative reduction: Mixed-effects Model for Repeated Measures. mITT population. Two sided at the 0.05 significance level.

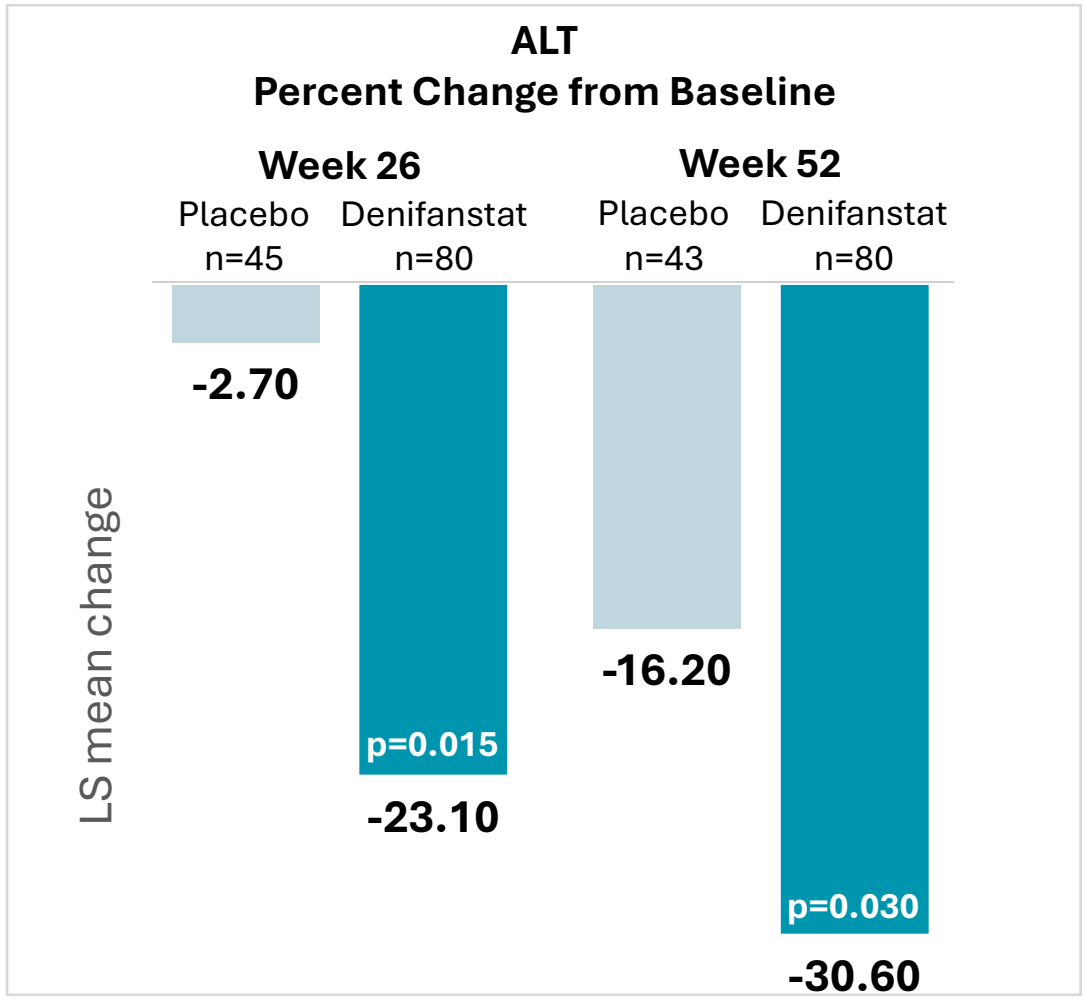


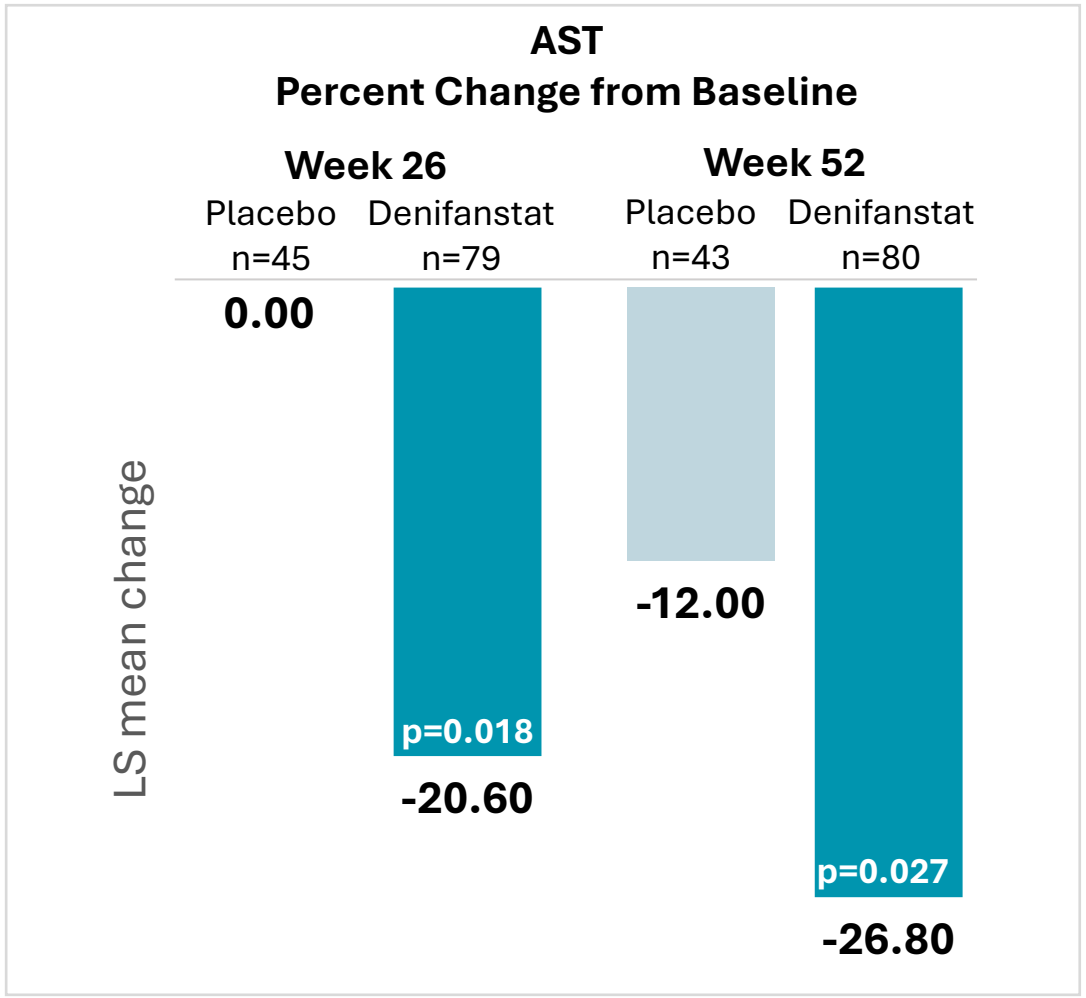
Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level.
mITT population.

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100. Sagimet Biosciences data on file.

Secondary Endpoints: Liver Enzymes

Denifanstat Decreased ALT and AST Levels



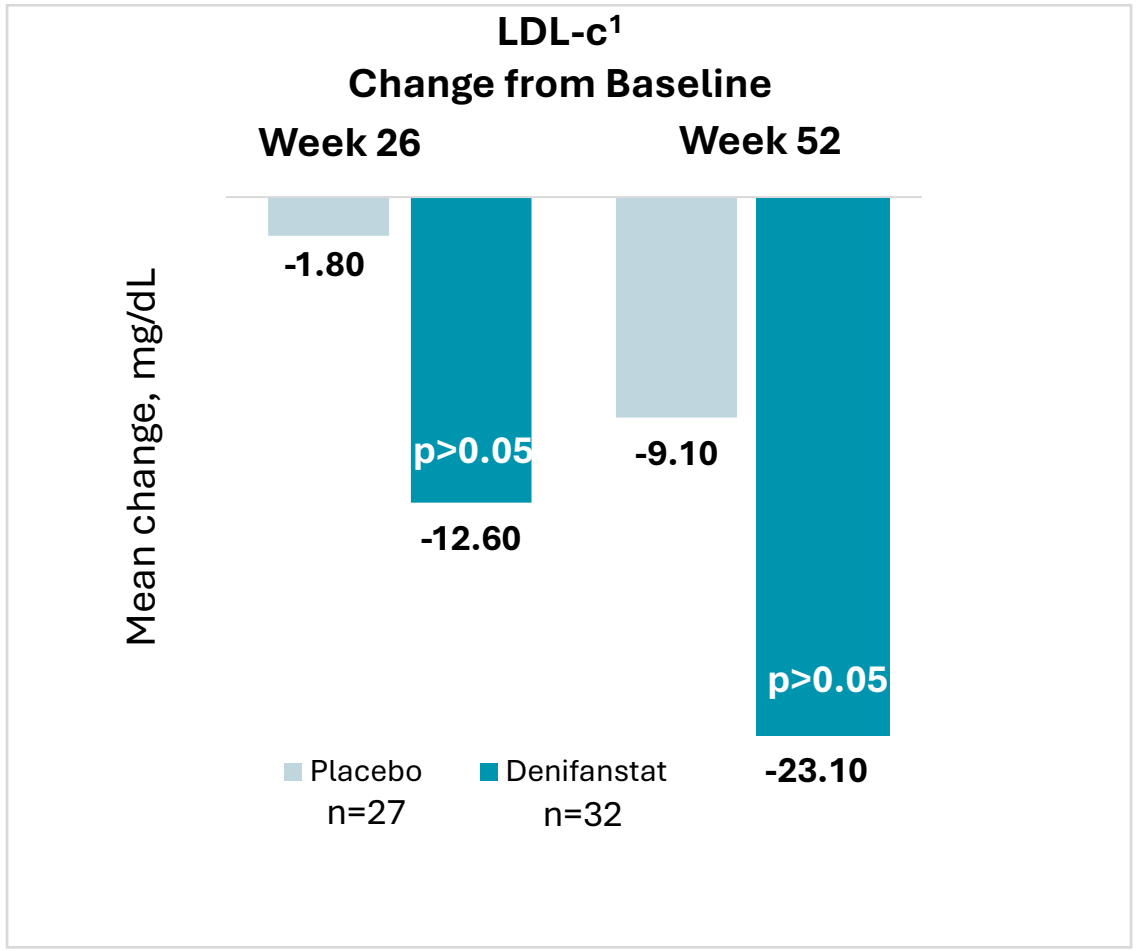


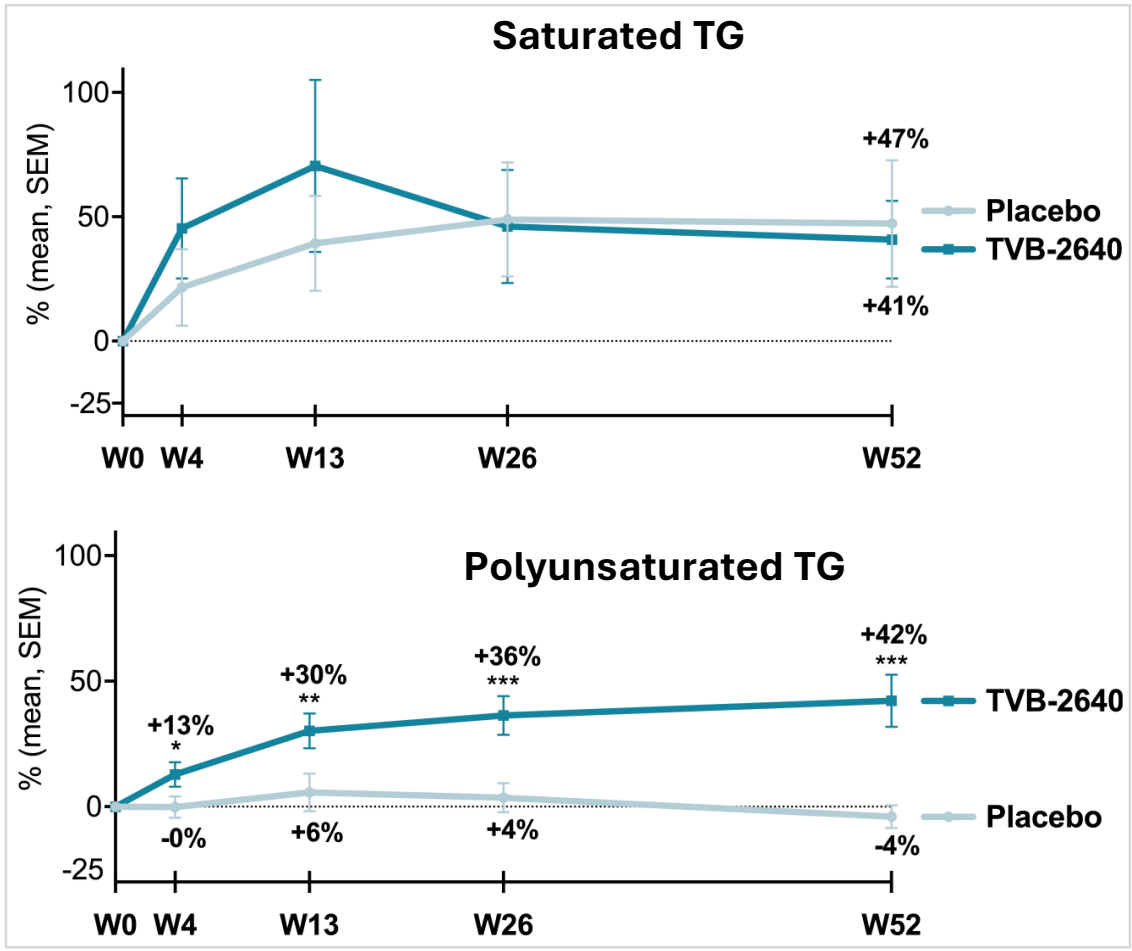
Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. mITT population

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100. Sagimet Biosciences data on file.

Cardiometabolic Health

Denifanstat Decreased LDL-c Levels and Increased Polyunsaturated Triglycerides

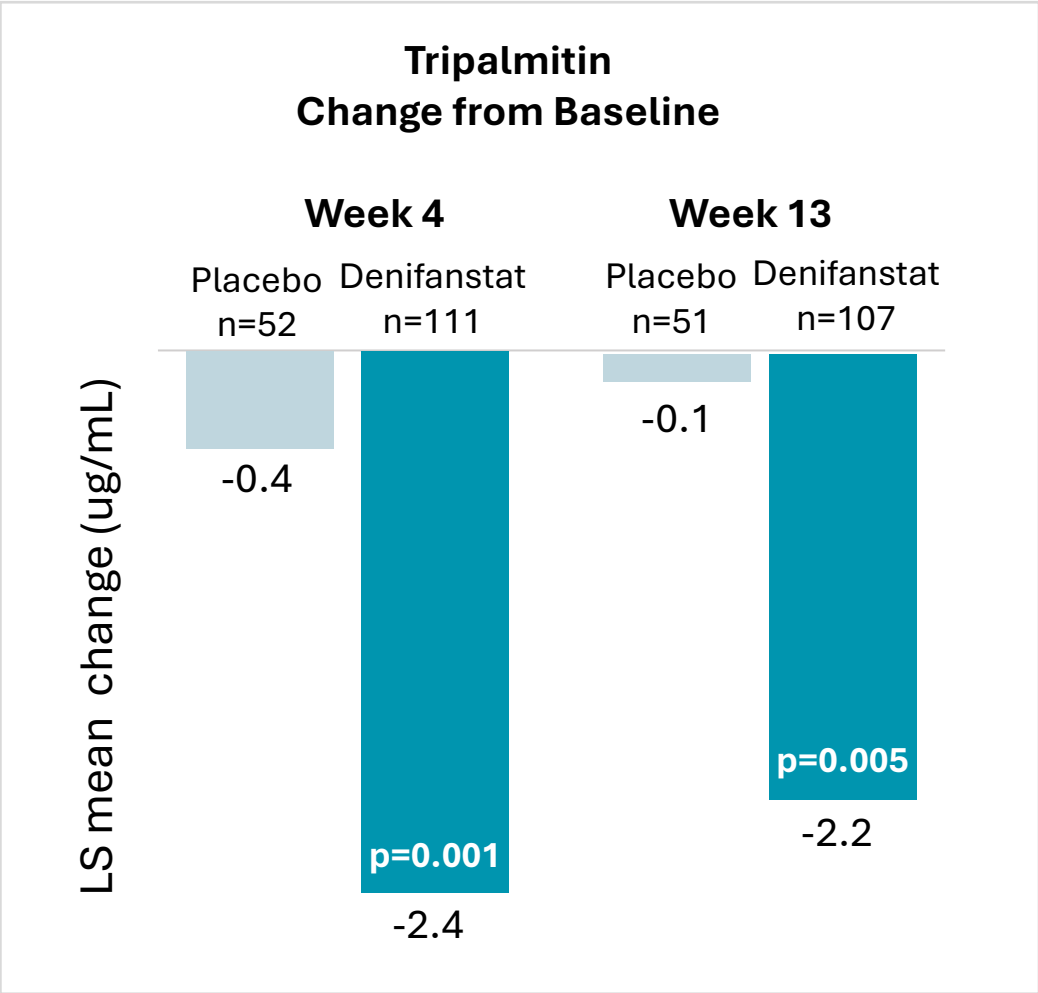




mITT population. Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. *p<0.05, **p<0.01, ***p<0.001. [1]For LDL-c, baseline > 100 mg/dL.

Loomba R, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/Denifanstat_a_fatty_acid_synthase_FASN_inhibitor_shows_significant_fibrosis_improvement_and_MASH_resolution_in_FASCINATE-2_a_Ph2b_52_week.pdf
Sagimet Biosciences data on file.

Denifanstat Reduced De Novo Lipogenesis



Tripalmitin:

- A saturated triglyceride which is a biomarker of DNL inhibition
- Reduced by denifanstat as early as week 4 of treatment

Next steps

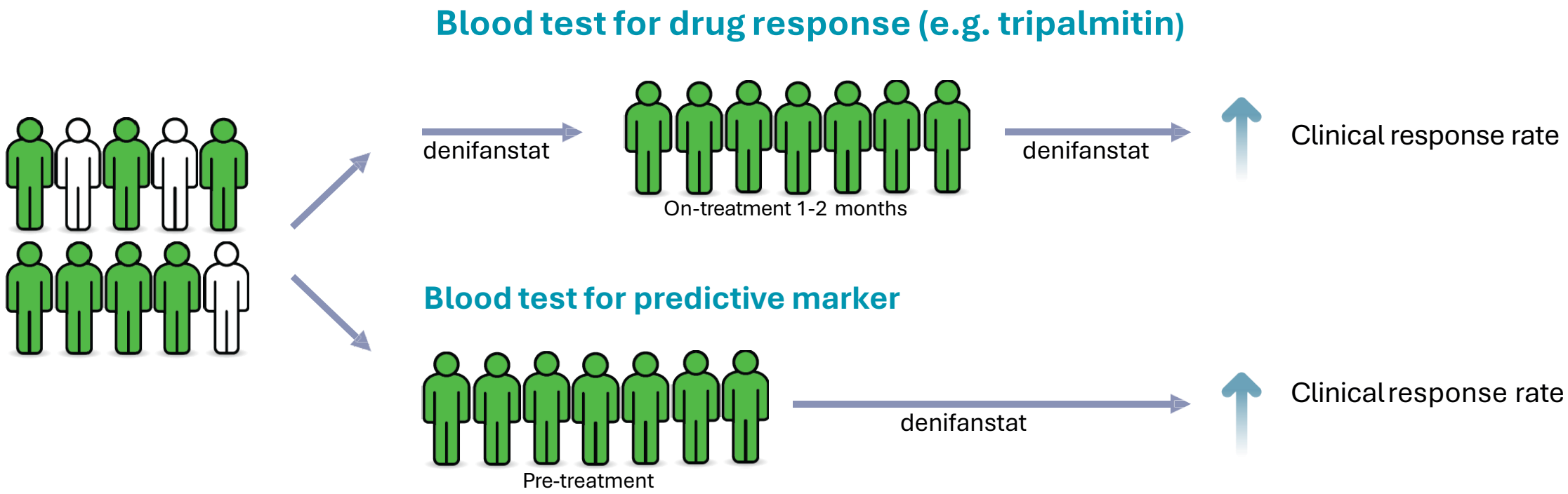
- Continue the development of tripalmitin and additional markers as potential biomarker(s) of treatment response for denifanstat

Two sided at the 0.05 significance level, ITT population

Loomba R, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/Denifanstat_a_fatty_acid_synthase_FASN_inhibitor_shows_significant_fibrosis_improvement_and_MASH_resolution_in_FASCINATE-2_a_Ph2b_52_week.pdf
Sagimet Biosciences data on file.

Precision Medicine: Blood Tests May Lead to Improved Patient Outcomes

- MASH is a multi-faceted disease and patients may benefit from being matched with optimal treatments
- Two approaches using blood tests are undergoing further evaluation
  - Drug response: 1-2 months after taking drug, tripalmitin identifies patients responding to drug treatment
  - Potential predictive marker: before taking drug, signature of 6 blood metabolites enriches for responders[1]



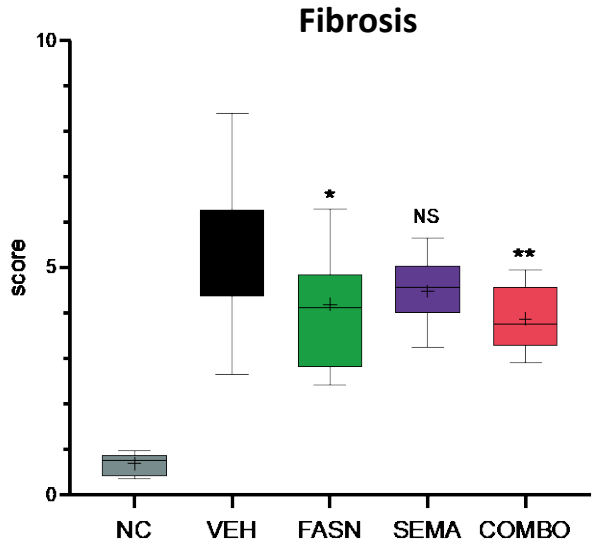
1. Signature has 6 metabolites: ursodeoxycholic acid, DL-2-aminocaprylic acid, sarcosine, glycoursodeoxycholic acid, D(-)-2-aminobutyric acid, PC(0-18:0/22:4). Accuracy 79%, PPV 88%, NPV 63%. Sagimet Biosciences data on file.

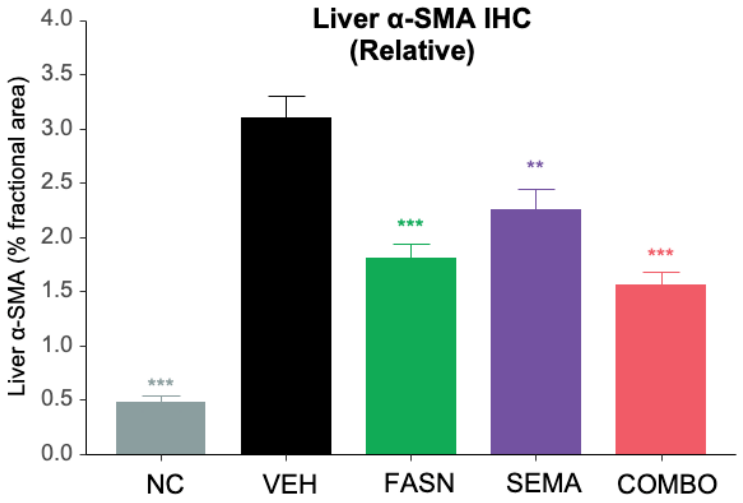
Combination Therapy Development Program for MASH

SAGIMET BIOSCIENCES

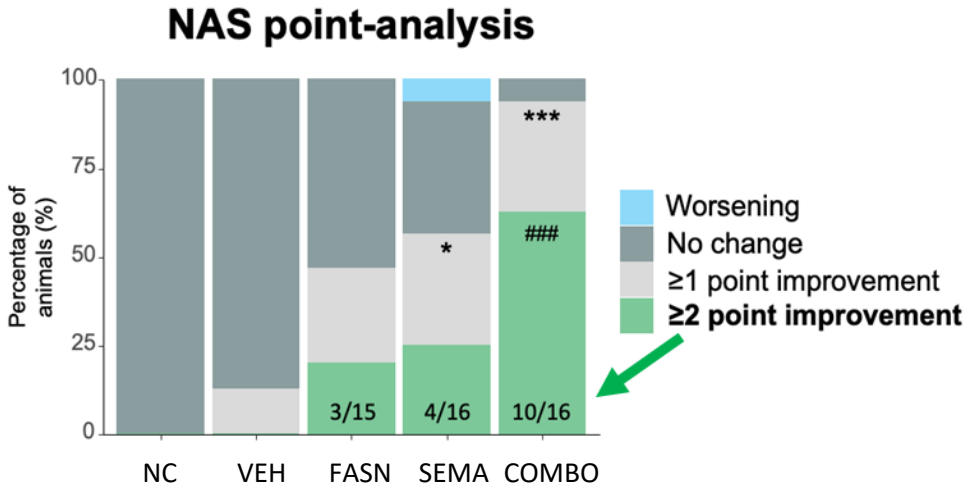
Combination of FASN Inhibitor and Semaglutide Improved Histological Features in MASH Mouse Model

In a mouse model, combination treatment showed: 1) an additive effect on fibrosis reduction, 2) a direct impact on stellate cells, and 3) a synergistic effect on NAS reduction





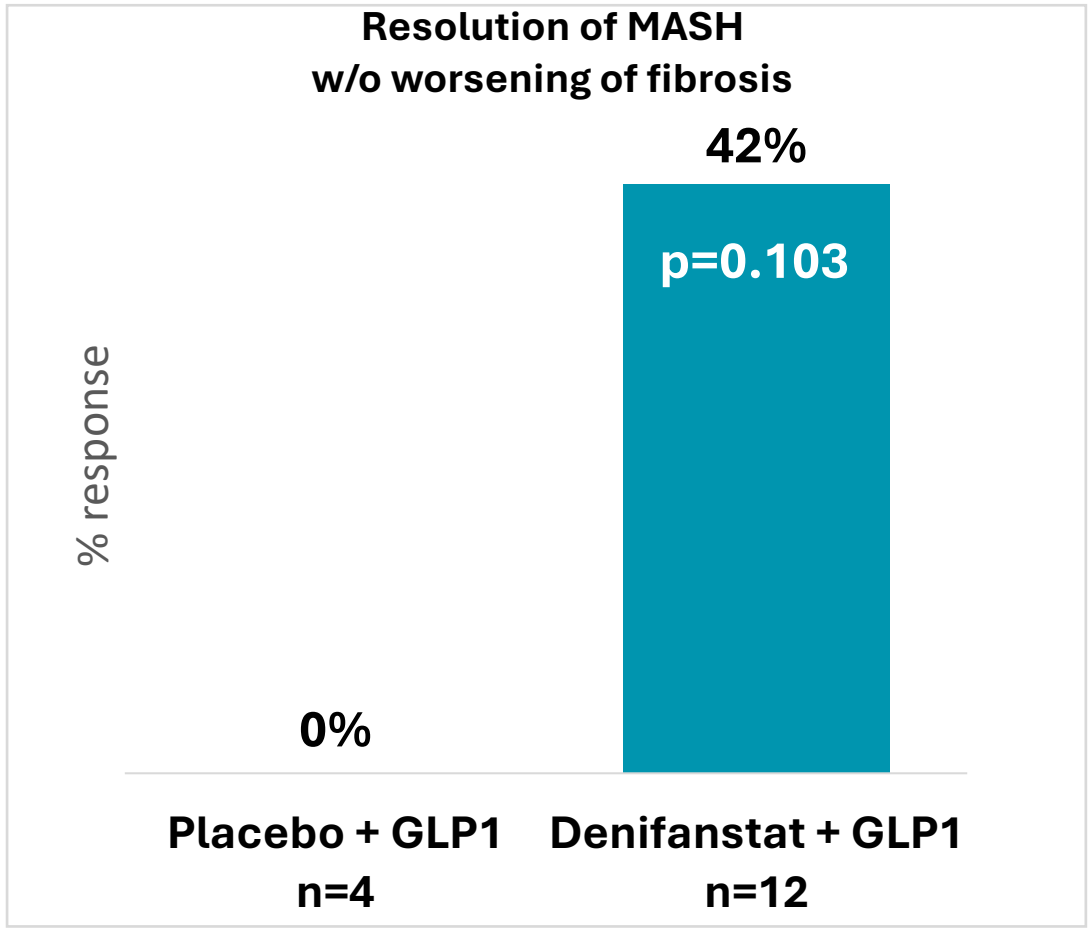
α-SMA: a marker of activated hepatic stellate cells



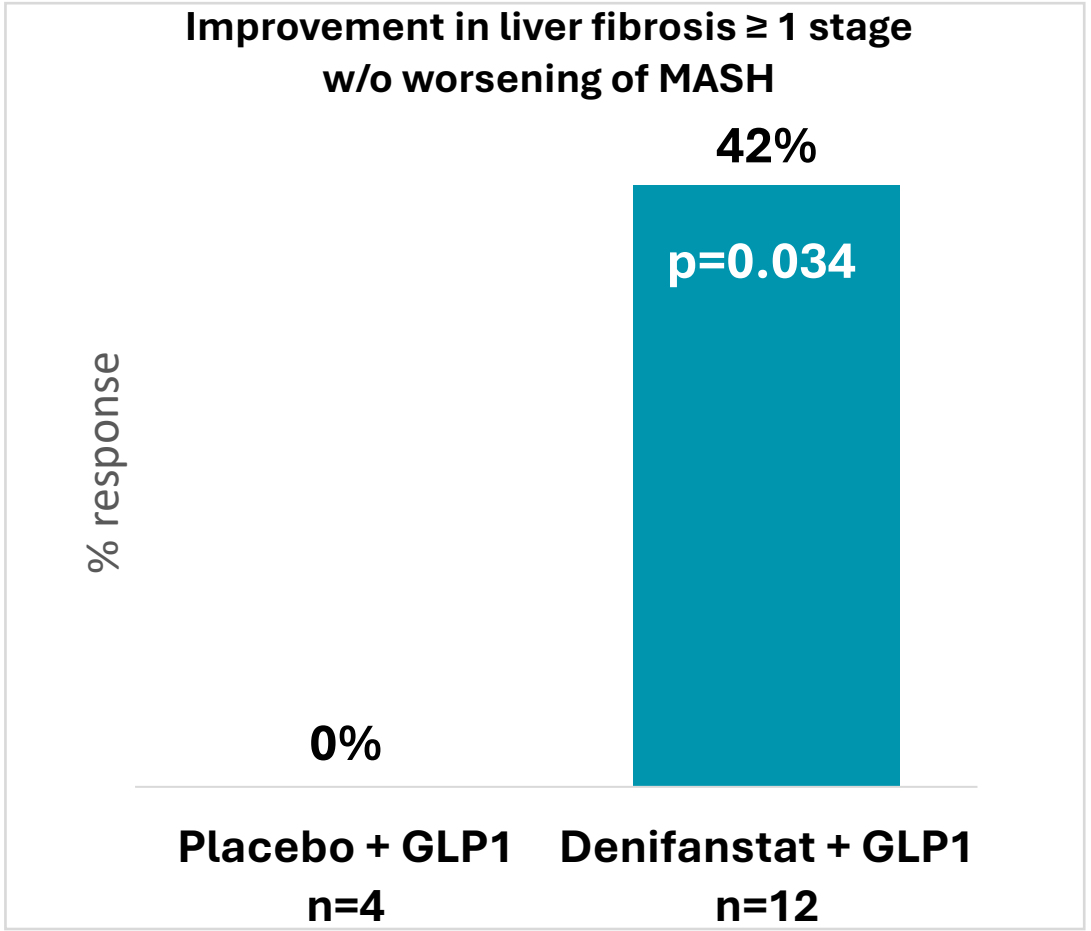
* p <0.05, ** p<0.01, *** p<0.001, ### p<0.001
GUBRA DIO MASH mice. PSR images were analyzed by FibroNest (PharmaNest), all scores shown with parenchymal correction
NS: not significant; NC: Normal chow diet control, VEH: MASH vehicle control, FASN: TVB-3664 (FASN inhibitor), SEMA: semaglutide, COMBO: TVB-3664/semaglutide
Tsai WW, et al. Presented at: AASLD 2023; November 10-14, 2023; Boston, MA. Abstract 2400-C.

Patient Subset on Stable GLP1-RA at Baseline: Liver Biopsy

FASCINATE-2 Phase 2b - Denifanstat Improved MASH Resolution and Fibrosis



Cochran-Mantel-Haenszel Test – One sided at the 0.05 significance level. mITT population
GLP patients were on stable dose for 6 months prior to first biopsy



AI digital pathology results also supports fibrosis improvement in patients receiving GLP1 and denifanstat

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100.
Loomba R, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/Denifanstat_a_fatty_acid_synthase_FASN_inhibitor_shows_significant_fibrosis_improvement_and_MASH_resolution_in_FASCINATE-2_a_Ph2b_52_week.pdf

Mechanism of Action Supports Combination Therapy Opportunity

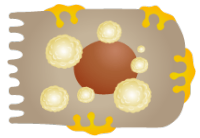
Potential improved clinical outcome for patients with combination therapy of denifanstat, a fat synthesis inhibitor + a fat oxidizer (THR-beta agonist)

Combination therapy potential:

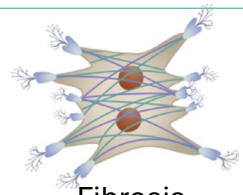
- Denifanstat MOA complementary to other MOAs – THR-beta, GLPs
- Opportunity for fixed dose combinations with other oral medications

Hypothesis: distinct and complementary mechanisms of the combination lead to synergistic effect

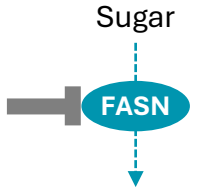
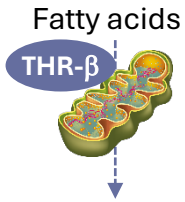
Preclinical combination studies in mouse models showed beneficial impact of FASN inhibitor + resmetirom combination on histology and MASH biomarkers[1]



Liver fat
(hepatocyte)



Fibrosis
(stellate cell)

Denifanstat Reduces de novo Lipogenesis		**Direct** - decreases de novo lipogenesis	**Direct** – decreases fibrogenesis in stellate cells, liver fat and lipotoxicity
Resmetirom Increases mitochondrial beta-oxidation		**Direct** - increases fatty acid oxidation and improves mitochondrial function	**Indirect** – decreases fibrosis due to decreased liver fat and lipotoxicity

MOA- Mechanism of Action

1. Tsai WW, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/2024-EASL-poster-GAN-model-final.pdf
Tsai WW, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/2024-EASL-poster-TNO-model-final.pdf

Potential Benefits of Combination Therapy in Advanced MASH Patients

A combination product could potentially offer an opportunity to serve patient groups with the strongest need of treatment, including those with stage 4 fibrosis

Characteristic	Denifanstat [1]	Resmetirom [2]	Potential Combination
Mechanism	**Direct** – decreases de novo lipogenesis **Direct** – decreases fibrogenesis in stellate cells, liver fat and lipotoxicity	**Direct** – increases fatty acid oxidation **Indirect** – decreases fibrosis due to decreased liver fat and lipotoxicity	Potential synergies in the MOA Note: THR-beta upregulates FASN
Formulation	Oral	Oral	Oral
Dosing	Once daily	Once daily	Once daily Fixed-Dose Combination (FDC)
Clinical Data	Met both primary endpoints in Phase 2b trial with significant reduction in fibrosis	Phase 3 data supported FDA approval for treatment of non-cirrhotic MASH	Potential synergistic effect

Note: These data are placebo-adjusted, derived from different clinical trials at different points in time, with differences in trial design and patient populations. As a result, cross-trial comparisons cannot be made, and no head-to-head clinical trials have been conducted.

1. Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100. 2. Harrison SA, et al. *N Engl J Med*. 2024;390(6):497-509.

Clinical Development Program for Denifanstat and Resmetirom Combination

Phase 1 pharmacokinetics (PK) trial for combination of denifanstat and resmetirom completed in December 2025

- Combination of denifanstat and resmetirom was generally well-tolerated over the duration of the study, with no safety signals
- No Serious Adverse Events (SAEs), no clinically significant laboratory results, and no treatment discontinuations

Subject to consultation with regulatory authorities, Phase 2 clinical trial with denifanstat and resmetirom in patients with MASH cirrhosis (F4) planned to initiate in 2H 2026

- Phase 2 proof-of-concept efficacy trial in patients with MASH cirrhosis (F4), for which there are no approved treatments
  - Potential clinical trial design, to be discussed with FDA:
    - 4-5 arms including monotherapy of each agent and up to two combination arms, versus placebo
    - At least 52 weeks combination treatment (up to 96 weeks) with interim readout planned at 52 weeks
    - Main efficacy endpoints: fibrosis improvement in liver biopsies and non-invasive markers of fibrosis
    - Non-invasive tests (NITs) for early readout to evaluate impact of the combination
    - Enrollment estimated between 12 and 18 months

Attractiveness of a Potential Denifanstat/Resmetirom Combination

Denifanstat in MASH

- Denifanstat directly targets the 3 key drivers of MASH (metabolic dysfunction-associated steatohepatitis) – liver fat, inflammation, and fibrosis
- Successful outcome of Phase 2b trial; met both primary endpoints with significant reduction in fibrosis [1]

Potential of a Fixed Dose Combination

- Pre-clinical data demonstrated synergistic effect of combination of FASN inhibitor and resmetirom [2]
- Combination of a Phase 3-ready drug candidate with the first drug approved for MASH
- IP for the combination of denifanstat and resmetirom:
  - Application filed 2024; if granted—2044; potential PTE to 2048
- Potential oral, once-daily product
- Potential to address an unmet need in patients with MASH cirrhosis (F4)

Development Program

- Phase 1 PK clinical trial of a combination of denifanstat and resmetirom completed in December 2025
- Phase 2 trial of denifanstat/resmetirom combination in F4 MASH patients is planned to initiate in 2H 2026
- Global license agreement with TAPI [3] for innovative forms of resmetirom API for the fixed dose combination program; Sagimet anticipates selecting one of the licensed innovative forms of resmetirom for combination with denifanstat in a fixed dose combination (FDC) tablet for use in a potential Phase 3 trial

1. Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100.
2. Tsai WW, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/2024-EASL-poster-GAN-model-final.pdf
Tsai WW, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/2024-EASL-poster-TNO-model-final.pdf
3. Assia Chemical Industries Ltd. (Assia), doing business as TAPI Technology & API Services (TAPI), a subsidiary of Teva Pharmaceutical Industries Ltd.

FASN Inhibition Offers Potential Benefit in Multiple Indications: Acne

SAGIMET BIOSCIENCES

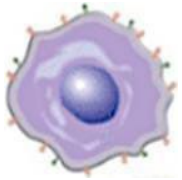
FASN Also Plays a Key Role in Other Diseases With Significant Unmet Need

FASN

Palmitate Lipid building block

Cancer cell

Membrane synthesis, intracellular signaling, protein modification



FASN in cancer

Supports tumor survival

Enables tumor proliferation

Establishes resistance to drugs

Sebocyte

Sebum production

FASN in acne

Increases sebum production

Exacerbates sebum composition

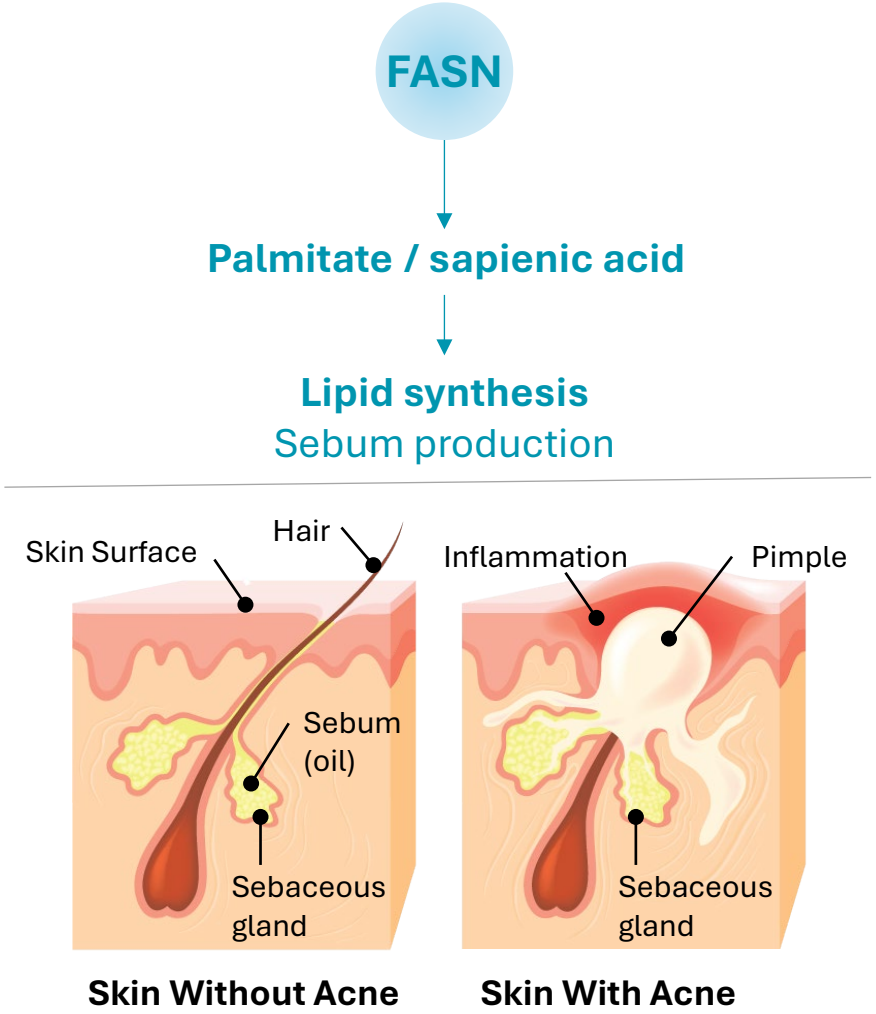
Acne Pathogenesis and Potential Role of FASN Inhibitors

Multifactorial pathogenesis of acne involves 4 key aspects:

- Increased sebum in sebaceous glands (80% of lipids produced through DNL)
- Abnormal or excessive follicular hyper-keratinization
- Accelerated bacterial growth (C. acnes)
- Localized inflammatory response

FASN is an attractive therapeutic target for acne

- Denifanstat directly reduced cutaneous (skin) sebum DNL lipids in two Phase 1 studies
- FASN inhibition has potential to reduce inflammation, through decreasing cytokine secretion and Th17 activation[1]

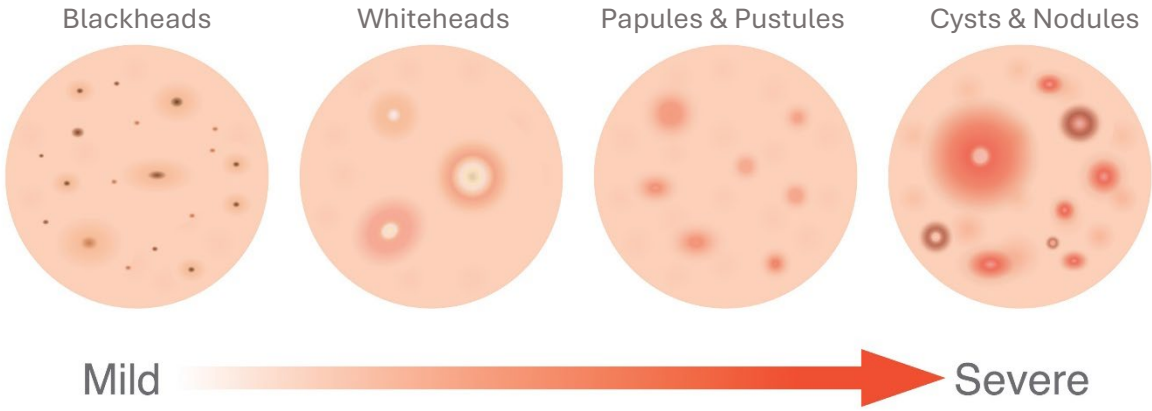


Heng AHS, Chew FT. *Sci Rep*. 2020;10(1):5754.

1. O'Farrell M, et al. *Sci Rep*. 2022;12(1):15661.

Acne Market Overview

Global acne market is expected to reach $17B in next decade[1]



5.1 million US acne patients are treated by dermatologists annually (total US acne market is 50 million people)[2,3]

- Acne is the #1 or #2 patient concern in dermatology offices and 65%+ of patients in dermatology offices have private insurance[4]
- Although acne treatments are currently available, dermatologists are open to new therapies (Seysara® Tablets & Winlevi® Cream)
- There is no cure for acne; due to its pathology, most patients require chronic management and multiple courses for flare control annually

Acne patients visiting a dermatologist are aligned to potential positioning of FASN inhibitor[4]

- 70% of patients presenting to dermatologists have moderate to severe disease[4]
- Approximately 70% of patients have inflammatory lesions, and 16% of patients are post-menopausal women[3]

1. www.expertmarketresearch.com/reports/acne-treatment-market
2. Bickers DR, et al. *J Am Acad Dermatol*. 2006;55(3):490-500. 3. American Academy of Dermatology. Burden of skin disease. 2017. www.aad.org/BSD.
4. Sagimet Biosciences data on file. Market research conducted in July 2024 among 50 dermatologists.

Acne Treatment Algorithm

Disease management involves flare and prevention intervention

Mild Disease	Moderate to Severe Disease	Severe (Cystic) Disease
Treatment includes topical agents used as mono-therapy, combination therapy, or with fixed dosed combination products	**Treatment approach adds oral products on top of the topical agents**	**Severe (cystic) patients are generally managed with isotretinoin (Accutane®)**
Main topical therapy categories • Retinoids • Benzoyl Peroxide • Antibiotics • Clascoterone • Salicylic Acid • Azelaic Acid	Main oral therapy categories: • Antibiotics (tetracyclines, sarecycline) • Hormonal contraceptives • Spironolactone (off-label) • Intralesional corticosteroids	Main therapy categories: • Isotretinoin

- Most acne patients receive skin care routines that include OTC cleansers and moisturizers to address AEs associated with their treatment

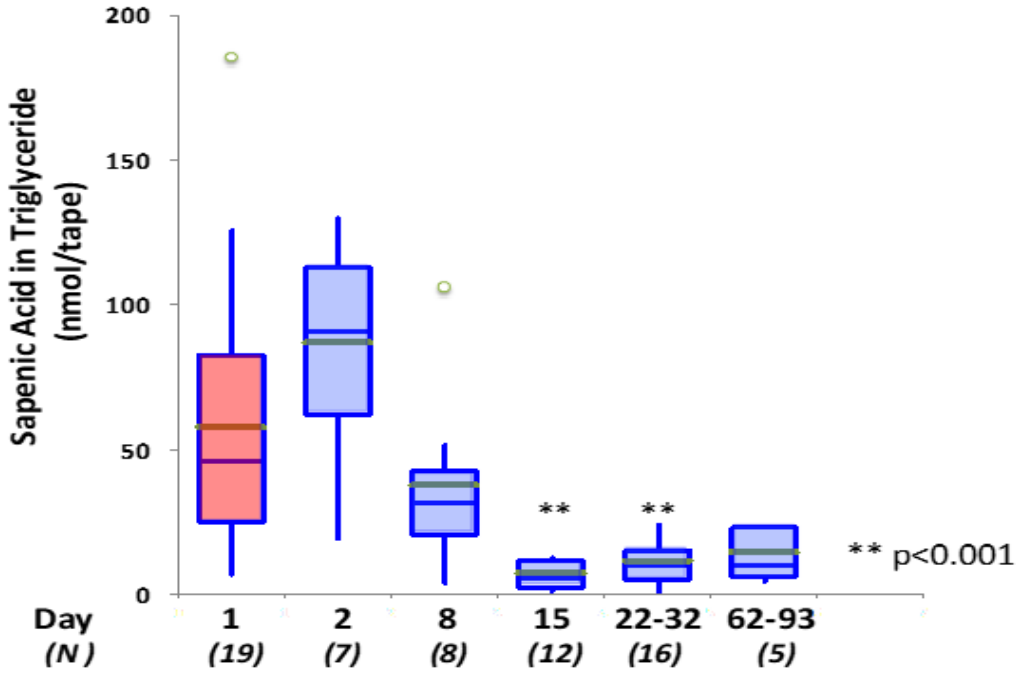
Clinical Data Support Mechanism of Action of a FASN Inhibitor in Acne

In multiple Phase 1 trials, FASN inhibitor demonstrated a decrease in DNL sebum lipids[1-3]

- FASN inhibitor demonstrated a >90% reduction in sebum lipids by day 15[1,2]
- FASN inhibitor maintained the reduced level of sebum lipids through the entire study[1,2]
- FASN inhibitor demonstrated a dose responsive impact on sebum lipids[1,2]

Note: denifanstat dose in this Phase 1 trial in cancer patients is several times higher than 50 mg dose tested in acne and MASH

Phase 1 oncology trial
Sebutape® assessment of cutaneous sebum lipids[1,2]



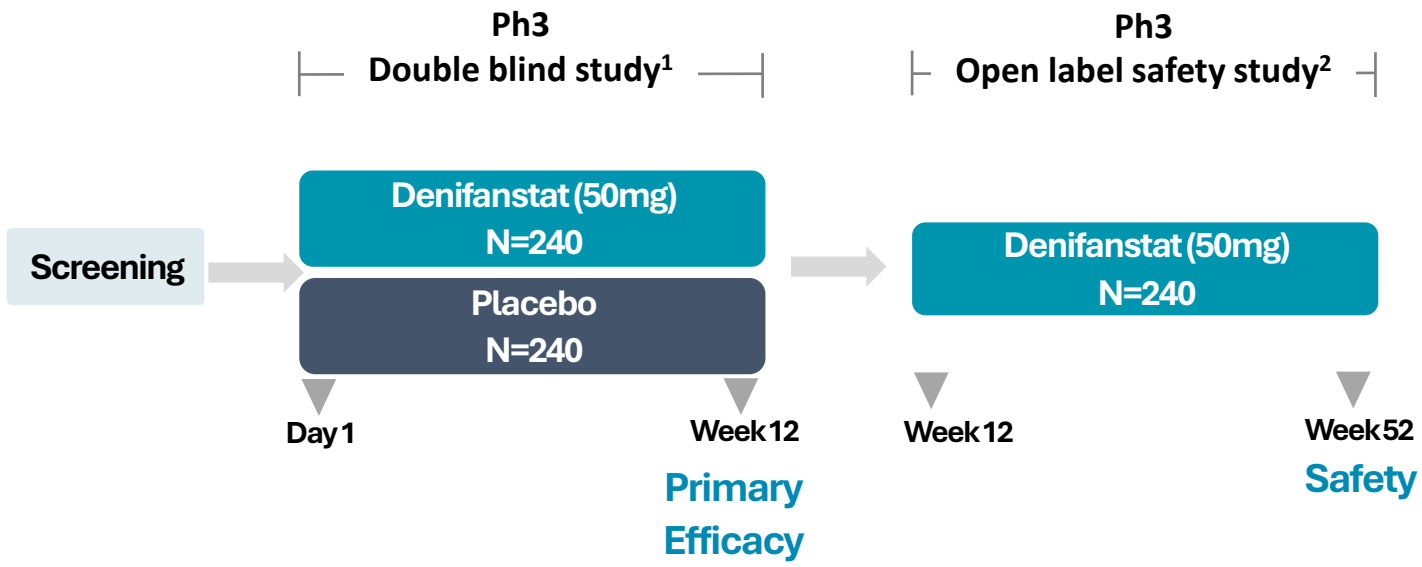
Days on therapy (# of subjects)

1. Duke G, et al. Presented at: EASL 2017; April 19-23, 2017; Amsterdam, The Netherlands. https://sagimet.com/wp-content/uploads/2017/05/3VBIO_EASLposter.pdf. 2. Falchook G, et al. *EClinicalMedicine*. 2021;34:100797.
3. Duke G, et al. Presented at: AASLD 2016; November 11-15, 2016; Boston, MA. https://sagimet.com/wp-content/uploads/2016/11/2016_AASLD_FASN_NASH_36x60_v10.pdf.

Ascletis Acne Phase 3 Clinical Trial Design

Denifanstat Phase 3 in acne

- Moderate to severe acne
- Multi-center placebo controlled
- 1:1 randomization
- Double-blind
- Once daily oral dosing
- 480 patients in China



Primary endpoints at week 12

- % patients who receive IGA success (defined as at least a 2-point reduction in IGA from baseline, and an IGA of 0 or 1 at week 12)
- % change of total lesion counts from baseline
- % change of inflammatory lesion counts from baseline

Key secondary endpoint at week 12

- % change of non-inflammatory lesion counts from baseline

1. ClinicalTrials.gov. NCT06192264. Study ASC40-303. https://clinicaltrials.gov/study/NCT06192264. 2. ClinicalTrials.gov. NCT06248008. Study ASC40-304. https://clinicaltrials.gov/study/NCT06248008.

Ascletis Acne Phase 3 Clinical Trial: All Primary and Secondary Endpoints Met

Baseline Characteristics	50mg denifanstat (n=240)	Placebo (n=240)
Total lesion count	102.2	102.1
Inflammatory lesion count	42.1	43.1
IGA=3 (moderate), %	85.8	85.8
IGA=4 (severe), %	14.2	14.2

Efficacy endpoints [1]	50mg denifanstat (n=240)	Placebo (n=240)	50mg denifanstat (placebo adjusted)	p value
% Treatment success [IGA] [2] (primary endpoint)	33.2	14.6	18.6	<0.0001
% Change in total lesion count (primary endpoint)	-57.4	-35.4	-22.0	<0.0001
% Change in inflammatory lesion count (primary endpoint)	-63.5	-43.2	-20.3	<0.0001
% Change in non-inflammatory lesion count (key secondary endpoint)	-51.9	-28.9	-23.0	<0.0001
Absolute change in total lesion count (secondary endpoint)	-58.3	-36.2	-22.1	<0.0001
Absolute change in inflammatory lesion count (secondary endpoint)	-26.6	-18.4	-8.2	<0.0001

Baseline demographics and efficacy endpoints of 50 mg denifanstat oral, once daily for 12 weeks versus Placebo (Intent-to-treat, ITT analysis change from baseline).

1. The efficacy data are LSMEANs.
2. Treatment success is defined as an Investigator's Global Assessment (IGA) score of 0 (clear) or 1 (almost clear) with at least a 2-point decrease from baseline.

Ascletis Acne Phase 3 Clinical Trial Safety Data

Denifanstat 50mg was generally well tolerated during the 12-week study

Treatment-emergent adverse events (TEAEs):

- TEAE incidence rates were comparable between denifanstat and placebo
- Only two categories of TEAEs had an incidence rate of 5% or more:
  - Dry eye (investigator reported as “dry eye” or “xerophthalmia”) in 10.9% of denifanstat-treated subjects vs 8.0% in the placebo group*
  - Dry skin reported in 6.3% of denifanstat-treated subjects vs 2.9% in the placebo group

Adverse events (AEs):

- All denifanstat-related AEs were mild or moderate
- No denifanstat-related grade 3 or 4 AEs
- No denifanstat-related serious AEs (SAEs)
- No deaths were reported

* The classifications of “dry eye” or “xerophthalmia” were not related to the AE grade.

Ascletis Acne Open Label Phase 3 trial

Denifanstat generally well-tolerated in the open label clinical trial

Treatment-emergent adverse events (TEAEs):

- Only two categories of TEAEs had an incidence rate of 5% or more with dry eye syndrome in 5.5% of denifanstat-treated subjects and dry skin reported in 5.2% of denifanstat-treated subjects

Adverse events (AEs):

- All denifanstat-related AEs were mild or moderate; no denifanstat-related Grade 3 or 4 AEs; no AE-related permanent discontinuations; Grade 1 hair thinning in the study was experienced by only 1 denifanstat-treated patient (which resolved within eight weeks while remaining in study without a change in dose); no deaths were reported

Serious adverse events (SAEs):

- No denifanstat-related SAEs; 2 non-denifanstat-related SAEs (1 breast lump, 1 contusion), both resolved

Efficacy Endpoints (secondary endpoints of the trial) :

- Efficacy endpoints (secondary endpoints of the trial) included the number of subjects with an IGA score decrease by at least 2 points, number of subjects dropping from an IGA score of 3 down to 0 or 1, the percentage reduction in total skin lesion count and the percentage reduction in inflammatory skin lesion count.
- Subjects treated with denifanstat showed improvements in all efficacy endpoints beyond those observed at 12 weeks

Ascletis data on file. Safety and efficacy endpoints of 50 mg denifanstat oral, once daily for 52 weeks versus placebo for 12 weeks and 50mg denifanstat oral once daily for 40 weeks

Second FASN Inhibitor TVB-3567 Entered FIH Phase 1

Phase 1 clinical trial initiated June 2025

A double-blind, randomized, placebo-controlled trial to assess the safety, tolerability, pharmacokinetics and pharmacodynamics of single and multiple ascending doses of TVB-3567 in healthy and acne participants

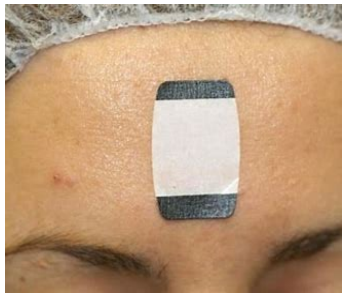
- **Includes sebum analysis as pharmacodynamic readout**

PART	DESIGN	PLANNED # of PARTICIPANTS
A	SAD	~56
B	Food effect	~12
C	MAD	~32
D	MAD/ACNE	~28

Sebumeter

Quantity of Sebum

Sebutape



Quality* of Sebum

Note: SAD = Single ascending dose. MAD = Multiple ascending dose

Each SAD/MAD cohort planned to include 6 participants on active and 2 on placebo. * Lipidomic analysis with focus on FASN-derived lipids.

ClinicalTrials.gov. NCT06989840. Study SB3567-CLIN-001. https://clinicaltrials.gov/study/NCT06989840.

Potential Clinical Development Program for TVB-3567 in Acne

Phase 1 trial initiated in June 2025

Goal to initiate Phase 2 trial in 2026, subject to consultation with regulatory authorities and outcome of Phase 1 trial

Step 1 - Phase 1 first-in-human pharmacokinetic (PK) clinical trial of TVB-3567 in healthy volunteers

- PK and pharmacodynamics (PD) evaluation to confirm profile
- Assess safety/tolerability
- Confirm potential doses for an acne Phase 2 trial

Step 2 - Phase 2 clinical trial in moderate to severe acne patients

- Upon completion of Phase 1, plan to consult with regulatory authorities regarding Phase 2 trial design, with goal of initiating Phase 2 trial in 2026
- Phase 2 trial design anticipated to be informed by the results of the Phase 1 trial, expect a 12-week dose ranging study in moderate to severe acne patients with lesion reduction, treatment success (IGA) as endpoints

Attractiveness of FASN Inhibition in Acne

FASN Inhibition in Acne

- Oral FASN inhibitors offer a novel mechanism of action for the potential treatment of moderate to severe acne
- Denifanstat met all primary and secondary endpoints in Phase 3 clinical trial in patients with moderate to severe acne vulgaris in China ; Denifanstat was generally well tolerated
- Denifanstat was well-tolerated in Ascletis' open-label Phase 3 clinical trial
- Denifanstat NDA for the treatment of moderate to severe acne accepted by NMPA in December 2025

Potential of TVB-3567 in Acne

- Acne market in dermatology is large (>50m people in the US) and aligned to those patients most likely to be prescribed an oral FASN inhibitor
- TVB-3567 IP:
  - Composition of matter patent—2035; potential PTE to 2038
  - Method of use application for TVB-3567 for acne filed 2025; if granted—2046

Development Pathways

- First-in-human Phase 1 clinical trial of TVB-3567 initiated in June 2025 for development in acne
  - Upon completion of TVB-3567 Phase 1, plan to consult with regulatory authorities regarding Phase 2 trial design, with goal of initiating TVB-3567 Phase 2 in 2026
  - Consulted with US FDA at end 2025 on the potential use of Ascletis Phase 3 data for the development of denifanstat in acne (e.g., as one of two registrational trials)

FASN Inhibition Offers Potential Benefit in Multiple Indications: Oncology

SAGIMET BIOSCIENCES

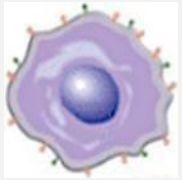
FASN Also Plays a Key Role in Other Diseases With Significant Unmet Need

FASN

Palmitate Lipid building block

Cancer cell

Membrane synthesis, intracellular signaling, protein modification



FASN in cancer

Supports tumor survival

Enables tumor proliferation

Establishes resistance to drugs

Sebocyte

Sebum production

FASN in acne

Increases sebum production

Exacerbates sebum composition

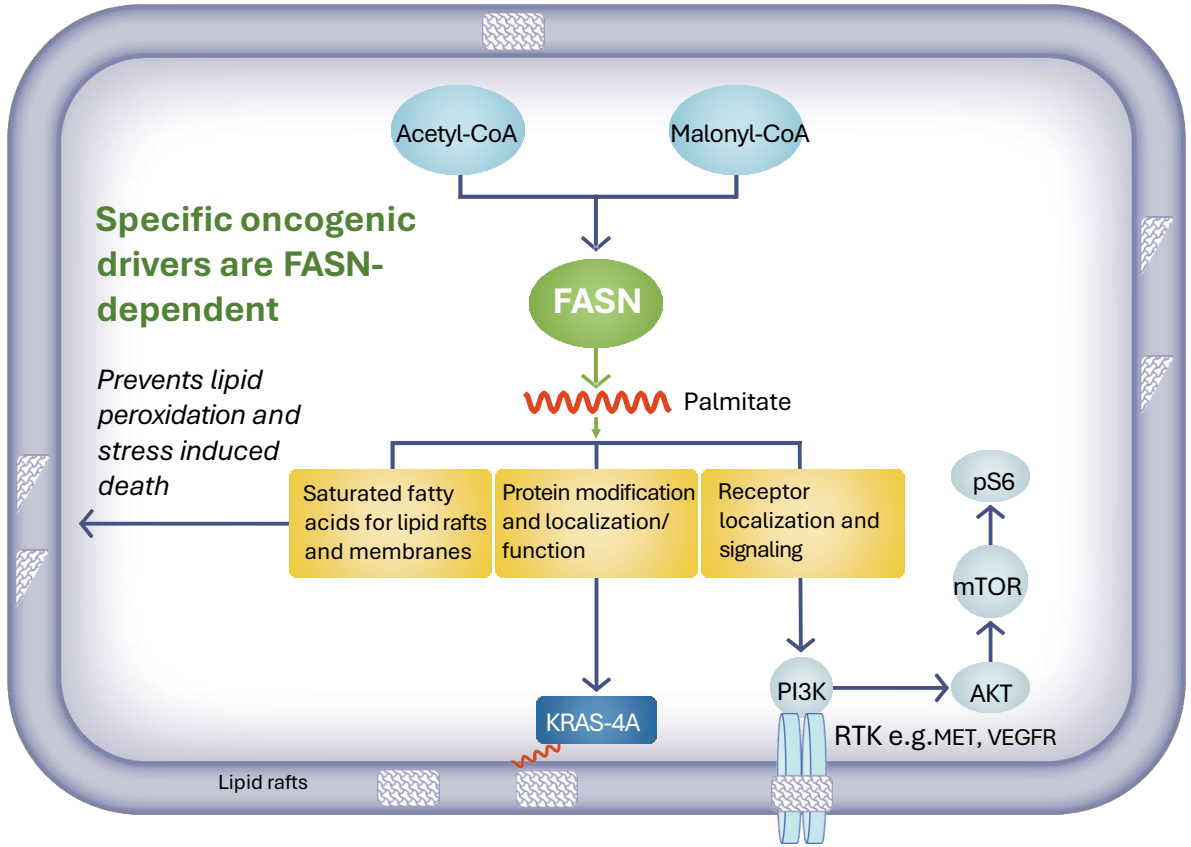
FASN Is Integral to Tumor Cell Proliferation and Survival

FASN dependence

- Certain cancers are dependent on DNL/FASN for proliferation especially downstream of driver oncogenes
- Strategy ➡ kill tumor cells and/or avoid drug resistance by combination of FASN inhibitor with drugs that inhibit driver oncogenes

Foundational Phase 1*

- 136 heavily pretreated patients received denifanstat
- Recommended Phase 2 dose defined
- Promising clinical activity consistent with proposed mechanism
  - KRASM NSCLC patients had significantly longer duration on study with denifanstat than KRASWT ($p<0.02$), and 91% KRASM had stable disease



Dietary fatty acids cannot compensate for de novo synthesized palmitate

KRASM – KRAS mutant. KRASWT- KRAS wild type

* Falchook G, et al. *EClinicalMedicine*. 2021;34:100797.

Cancer Program Focuses on 4 FASN-Dependent Tumor Types

Type	Status
Prostate	**Phase 1 ongoing** Investigator Sponsored at Weill Cornell, denifanstat combination with enzalutamide[1] Phase 1 results expected 1H2027
HCC	**Preclinical and translational work completed** Patient selection strategy by bioinformatics on primary samples Positive preclinical combination results[2] Phase 2-ready
NSCLC KRASM	**Preclinical and clinical evidence** Positive preclinical combination with KRAS inhibitor[3] Encouraging monotherapy Phase 1 results with denifanstat[4] Phase 2-ready
GBM	**Phase 2 completed** Positive investigator sponsored Phase 2 results[5] Ascletis announced cessation of China GBM program in August 2025 [6]

GBM (glioblastoma), HCC (hepatocellular carcinoma), KRASM (mutant KRAS), NSCLC (non small cell lung cancer)

1. ClinicalTrials.gov. NCT05743621. https://clinicaltrials.gov/study/NCT05743621. 2. Wang H, et al. *Hepatology*. 2022;76(4):951-966. 3. Liu Y, et al. *Lung Cancer*. 2021;153:73-80. 4. O'Farrell M, et al. Presented at: AARC 2016; April 16-20, 2016; New Orleans, LA. Abstract LB-214. 5. Kelly W, et al. *Clin Cancer Res*. 2023;29(13):2419-2425. 6. ClinicalTrials.gov. NCT05118776. Study ASC40-301. https://clinicaltrials.gov/study/NCT05118776.

Sagimet at a Glance

Unique MOA: FASN Inhibition

- Our lead molecule, denifanstat, is a novel fatty acid synthase (FASN) inhibitor with a differentiated MOA with the potential to target multiple underserved diseases
- Strong clinical data demonstrates denifanstat's proof of concept across multiple disease states

Denifanstat in MASH

- Denifanstat directly targets the 3 key drivers of MASH (metabolic dysfunction-associated steatohepatitis) – liver fat, inflammation, and fibrosis
- Successful outcome of Phase 2b trial; met both primary endpoints with significant reduction in fibrosis
- Pre-clinical data demonstrated synergistic effect of combination of FASN inhibitor and resmetirom
- Phase 1 pharmacokinetics (PK) clinical trial of a combination of denifanstat and resmetirom completed in December 2025, Phase 2 clinical combination trial with denifanstat and resmetirom in patients with MASH cirrhosis (F4) planned to initiate in 2H 2026

TVB-3567 in Acne

- Our follow-on FASN inhibitor, TVB 3567, received Investigational New Drug (IND) clearance in March 2025
- First-in-human Phase 1 clinical trial initiated in June 2025 for development of an acne indication