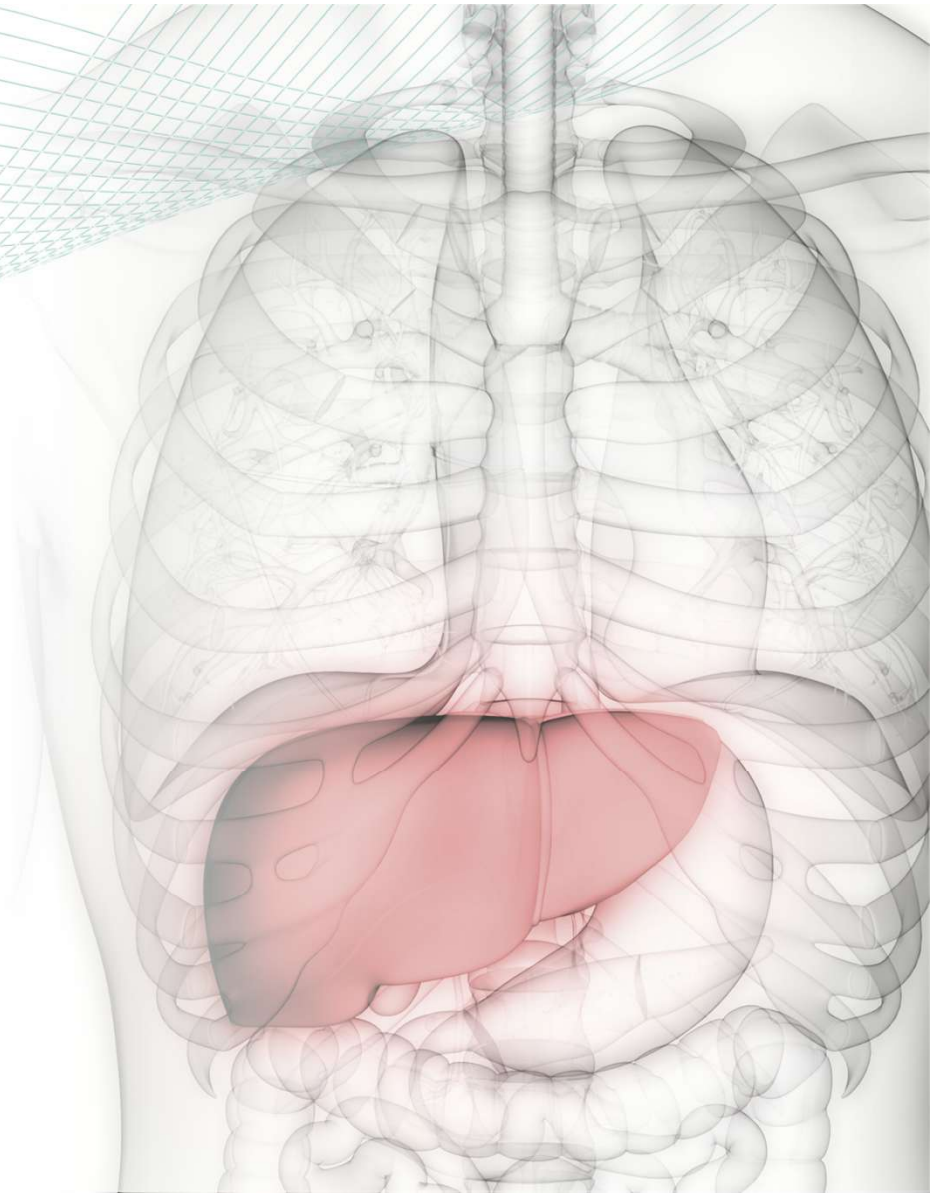
SAGIMET BIOSCIENCES

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

January 2025



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This presentation contains forward-looking statements within the meaning of, and made pursuant to the safe harbor provisions of, The Private Securities Litigation Reform Act of 1995. All statements contained in this document, other than statements of historical facts or statements that relate to present facts or current conditions, including but not limited to, statements regarding possible or assumed future results of operations, business strategies, research and development plans, regulatory activities, the presentation of data from clinical trials, Sagimet's clinical development plans and related anticipated clinical development milestones, market opportunity, competitive position and potential growth opportunities are forward-looking statements. These statements involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. In some cases, you can identify forward-looking statements by terms such as "may," "will," "should," "would," "expect," "plan," "anticipate," "could," "intend," "target," "project," "believe," "estimate," "predict," "potential," or "continue" or the negative of these terms or other similar expressions. The forward-looking statements in this presentation are only predictions. These forward-looking statements speak only as of the date of this presentation and are subject to a number of risks, uncertainties and assumptions, some of which cannot be predicted or quantified and some of which are beyond our control, including, among others: the clinical development and therapeutic potential of denifanstat or any other drug candidates we may develop; our ability to advance drug candidates into and successfully complete clinical trials, the risk the topline clinical trials may not be predictive of, and may differ from final clinical data and later-stage clinical trials; our ability to advance drug candidates into and successfully complete clinical trials within anticipated timelines, including our Phase 3 denifanstat program; that unfavorable new clinical trial data may emerge in other clinical trials of denifanstat, including Phase 3 clinical trials; that clinical trial data are subject to differing interpretations and assessments, including by regulatory authorities; our relationship with Ascletis, and the success of its development efforts for denifanstat; the accuracy of our estimates regarding our capital requirements; and our ability to maintain and successfully enforce adequate intellectual property protection. These and other risks and uncertainties are described more fully in the "Risk Factors" section of our most recent filings with the Securities and Exchange Commission and available at www.sec.gov. You should not rely on these forward-looking statements as predictions of future events. The events and circumstances reflected in our forward-looking statements may not be achieved or occur, and actual results could differ materially from those projected in the forward-looking statements.

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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

George Kemble *Executive Chairman*

>20 years of experience in R&D in biotech and pharma

Responsible for R&D of FluMist, first innovation in flu vaccines in 60+ years

Elizabeth Rozek *General Counsel*

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

Eduardo Martins *CMO*

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

Led clinical development team of cenicriviroc for MASH

Rob D'Urso *Senior Vice President of 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 disease states
- Clinical data demonstrates denifanstat's proof of concept across multiple disease states
- Denifanstat is highly differentiated as the only fat synthesis inhibitor currently in development

Phase 3 MASH program

- Denifanstat directly targets the 3 key drivers of MASH – liver fat, inflammation, and fibrosis
- Successful outcome of Phase 2b study; met both primary endpoints with significant reduction in fibrosis
- FDA Breakthrough Therapy designation granted for treatment of MASH (F2-F3 fibrosis)
- Phase 3 program initiated with sites activated & patients pre-screened in 4Q2024, FPI anticipated 1Q2025

Strategic Collaboration with Ascletis in Acne & Cancer

- Acne Phase 3 study enrollment completed in Nov 2024; topline results expected in 2Q2025
- GBM Phase 3 study in progress

Denifanstat IP Portfolio

- Method of use patent: 2036; Composition of matter patent: 2032
- Opportunities to lengthen one patent's life for up to 5 years via Patent Term Extension (US) or SPC (Europe)

Near Term Milestones & Cash Position

- NASDAQ: SGMT; $170.0M cash, cash equivalents and marketable securities at 3Q2024, expected to fund current operations through 2025
- Currently evaluating financing options to complete clinical development programs across indications

Development Pipeline: Indications and Clinical Milestones

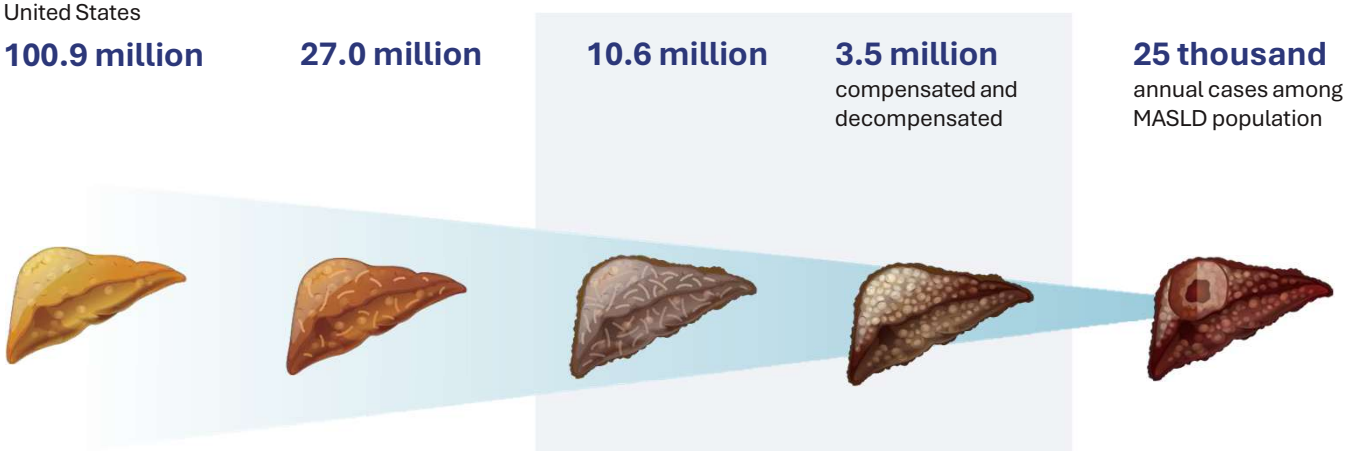
Therapeutic Area	Indication	Stage of Development	Expected Milestone / Status
Metabolic Disease	**MASH F2/F3 population**	Denifanstat (Phase 2 – Phase 3) Denifanstat (Phase 1 – Phase 2)	Phase 2b positive topline data announced 1Q2024; Pivotal Phase 3 program initiated 4Q2024, FPI anticipated 1Q2025; FDA Breakthrough Therapy designation Phase 1 hepatic impairment results reported 1Q 2024
Dermatology	**Acne**	TVB-3567 (Preclinical – Phase 1) Denifanstat (ASC40) (Phase 1 – Phase 3)	IND-enabling studies completed; evaluating timing to file IND Phase 3 clinical study enrollment completed in Nov 2024; topline results expected in 2Q2025
Oncology	**Solid tumors**	TVB-3567 (Preclinical – Phase 1) Denifanstat (Phase 1 – Phase 2)	Identifying FASN-dependent tumor types for potential FASN inhibitor development
	Recurrent glioblastoma (GBM)	Denifanstat (ASC40) (Phase 1 – Phase 3)	Phase 3 enrollment of 120 patients achieved in 3Q2023*

* Trials 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

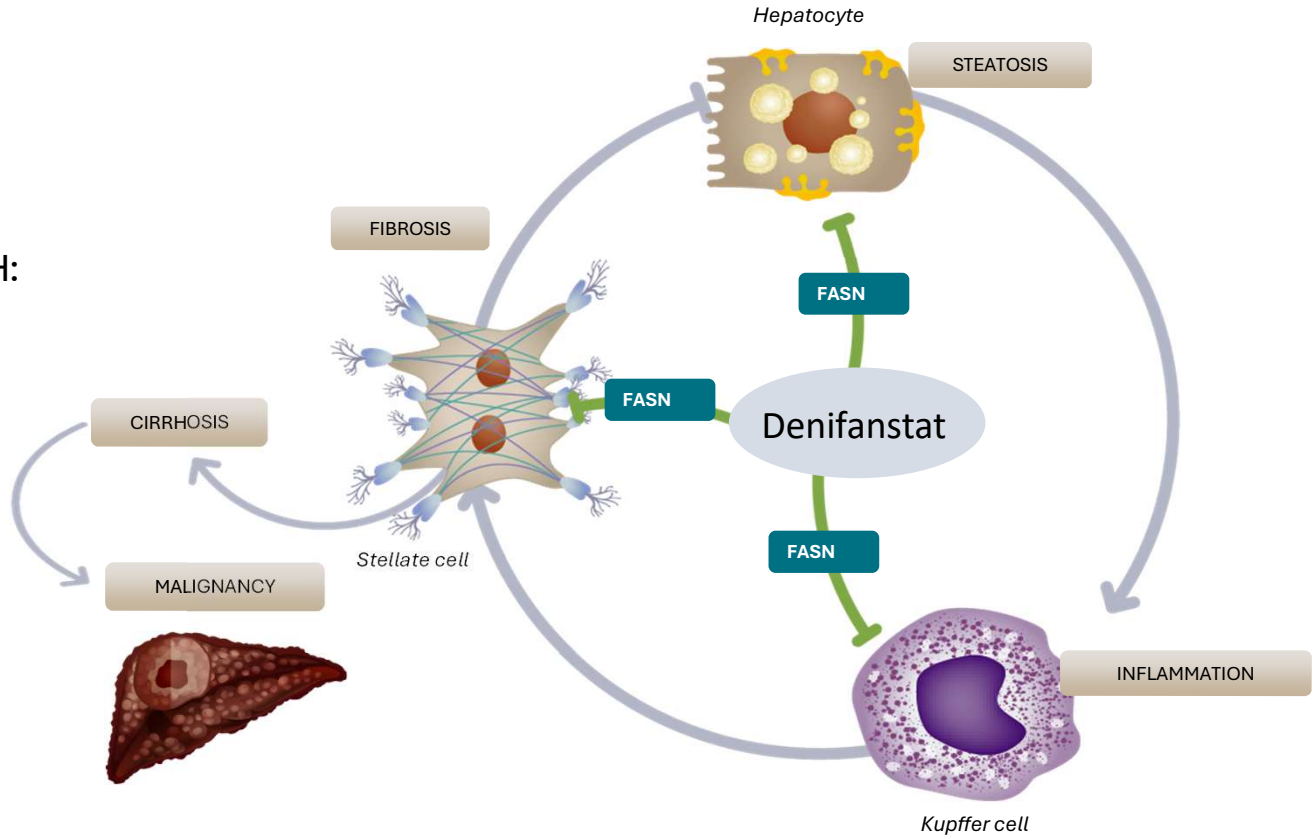
- Complex disease with heterogeneous patient population
- Significant opportunity for differentiated MOA

1 Estes, et al. 2018; http://dx.doi.org/10.1016/j.jhep.2018.05.036. Note: MASH, or metabolic dysfunction-associated steatohepatitis, was formerly known as NASH, or nonalcoholic steatohepatitis

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:

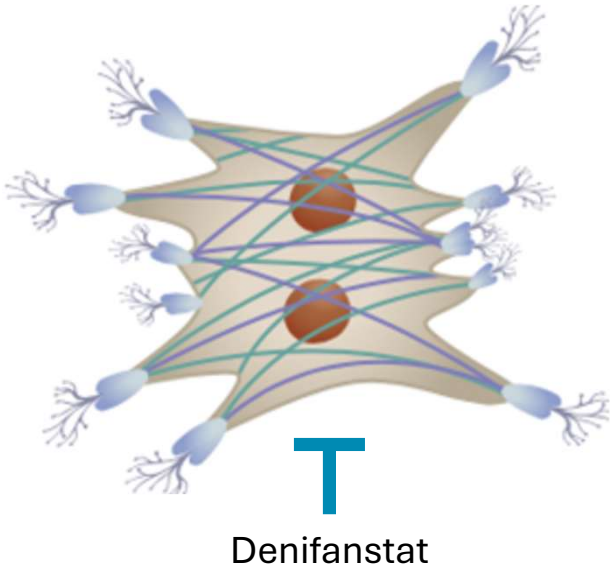
1. Blocking **steatosis** via inhibiting de novo lipogenesis in hepatocytes
2. Reducing **inflammation** via preventing immune cell activation
3. Blunting **fibrosis** via inhibiting stellate cell activation



FASN Inhibition Directly Blocks Human Liver Stellate Cell Function

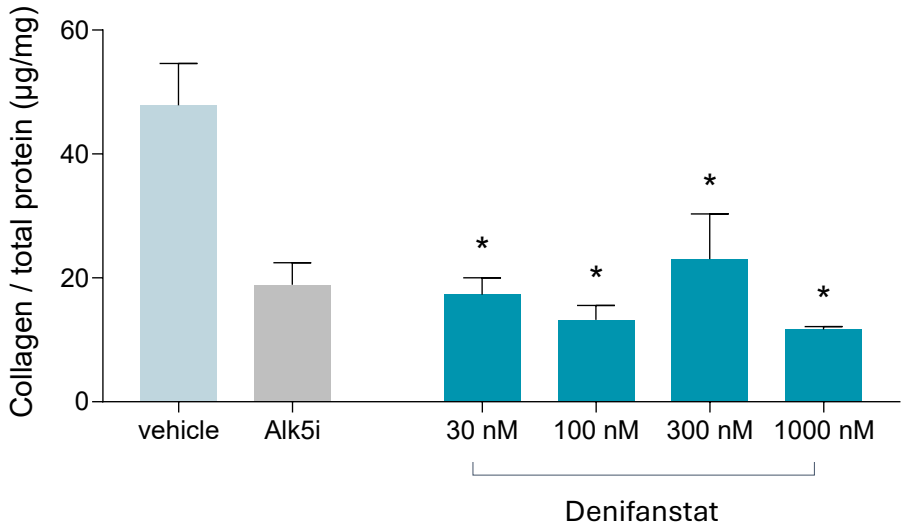
Stellate cells require DNL for fibrogenesis

Denifanstat blocks stellate cell activation



Primary human stellate cell assay

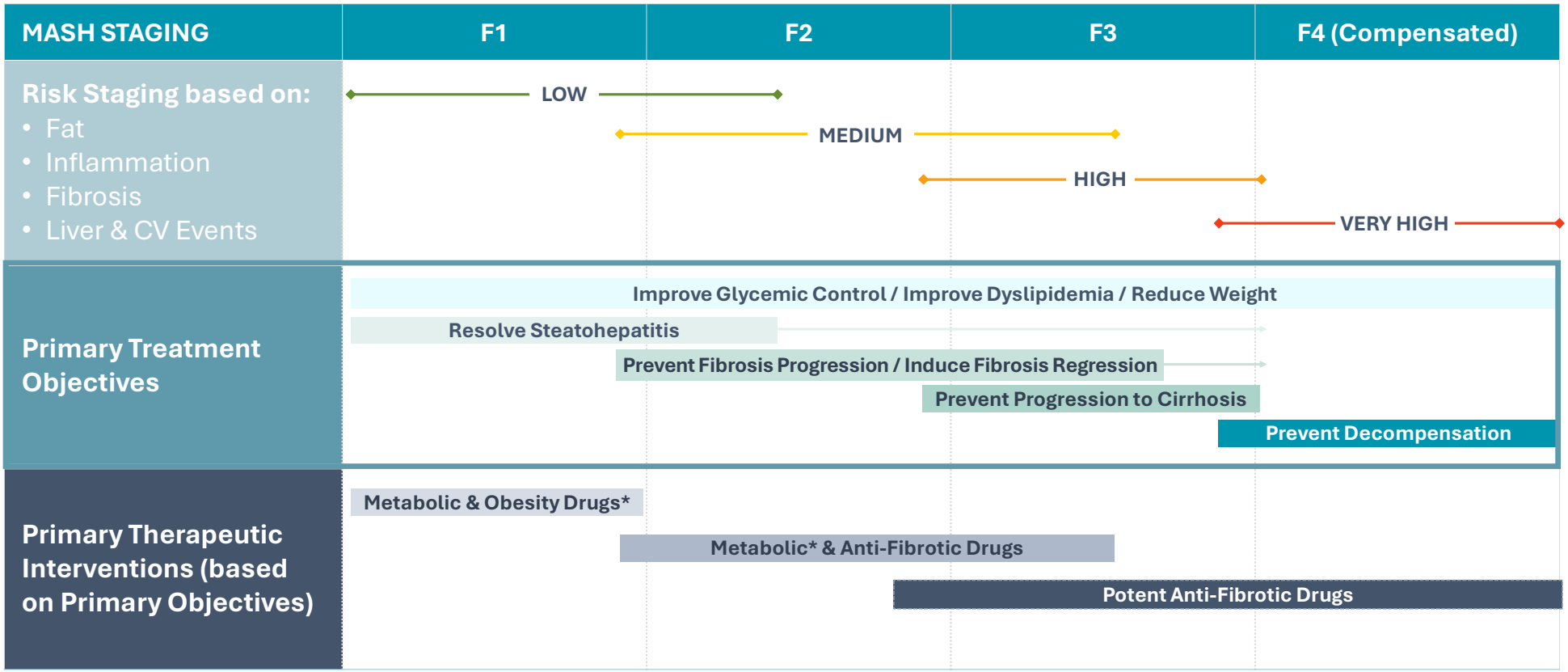
Denifanstat directly inhibits fibrogenic activity



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

*p<0.05. FASNi directly inhibits fibrosis published in O'Farrell et al.,2022. Scientific Reports. 12:15661

Treatment Goals for MASH Across Fibrosis Staging



Kusi et al. Endocrine Practice 28 (2022) 528-562. Rinella et al. Hepatology. 2023 May 01; 77(5): 1797–1835. Tacke et al. Journal of Hepatology, July 2024. vol. - 4 | 1–51
*Metabolic drugs are anticipated to be background therapy for obesity and type 2 diabetes, until clinical data support use in MASH

MASH Clinical Development Program

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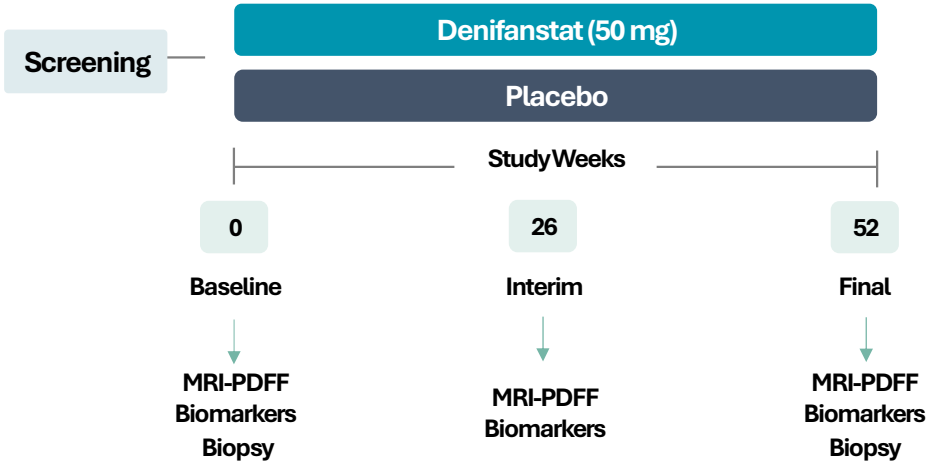
FASCINATE-2: Biopsy Trial Design Focused on Histological Endpoints

FASCINATE-2 Phase 2b trial design



- Biopsy confirmed F2-F3 MASH patients
- 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.

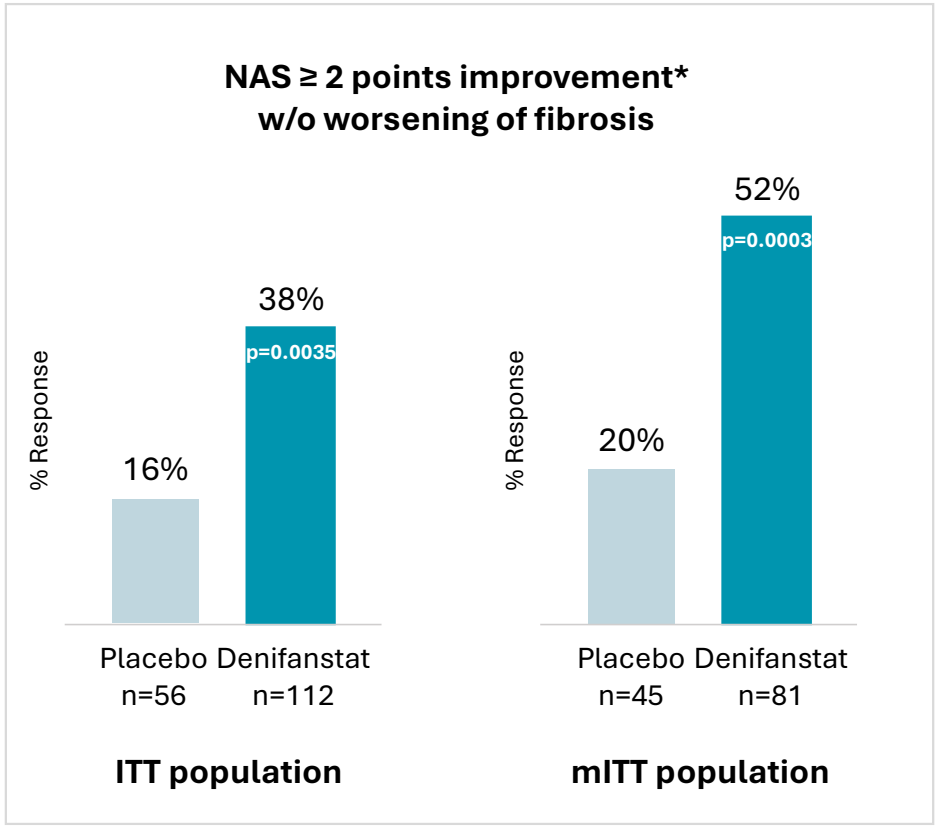
FASCINATE-2: Baseline Characteristics Were Typical of the F2/F3 MASH Population

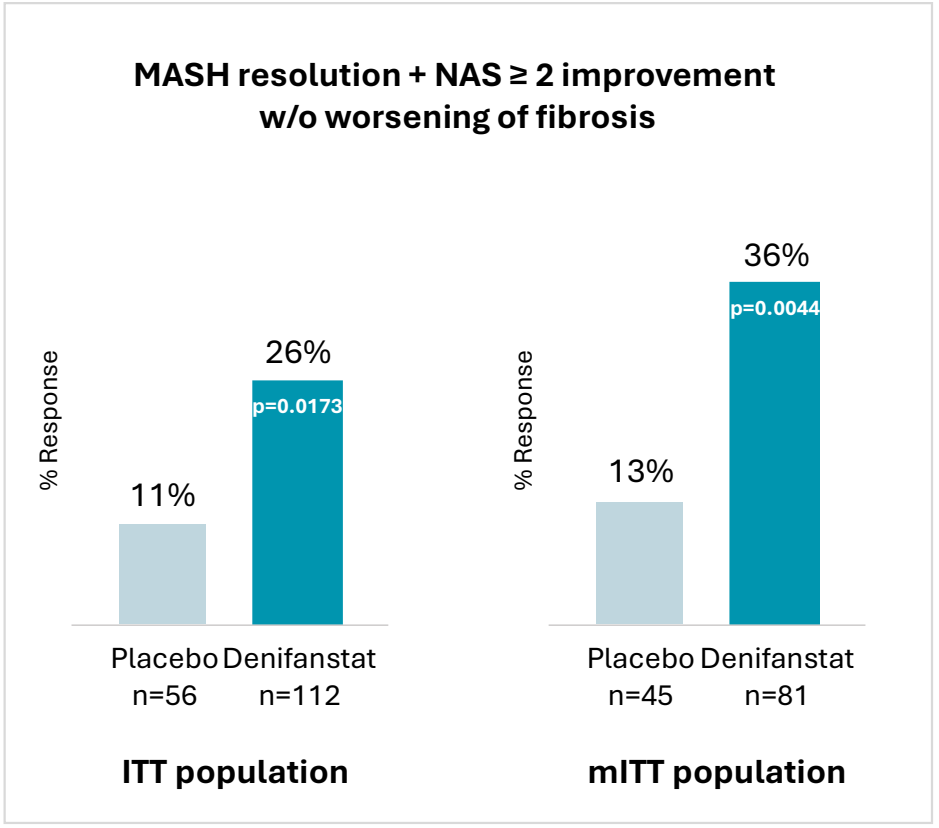
Parameter	Placebo, n=45	Denifanstat, n=81
Age, years	59.6 (+/- 10.9)	56.1 (+/- 10.8)
Sex, female	27 (60%)	48 (59%)
Race, White	41 (91%)	73 (90%)
Ethnicity, Hispanic or Latino	15 (33%)	27 (33%)
BMI, kg/m^2	36.5 (+/- 6.7)	34.6 (+/- 6.1)
Type 2 diabetes	27 (60%)	55 (68%)
ALT (alanine aminotransferase) U/L	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 (%)

Primary Endpoints: Liver Biopsy

Denifanstat Achieved Statistical Significance at 52 Weeks

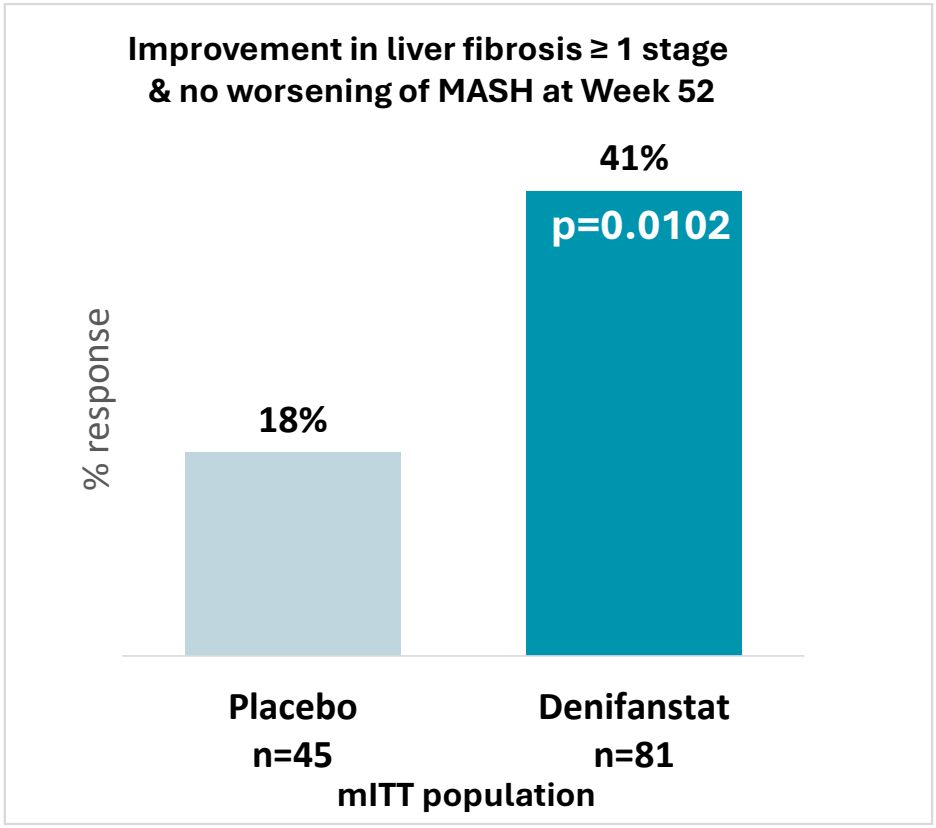


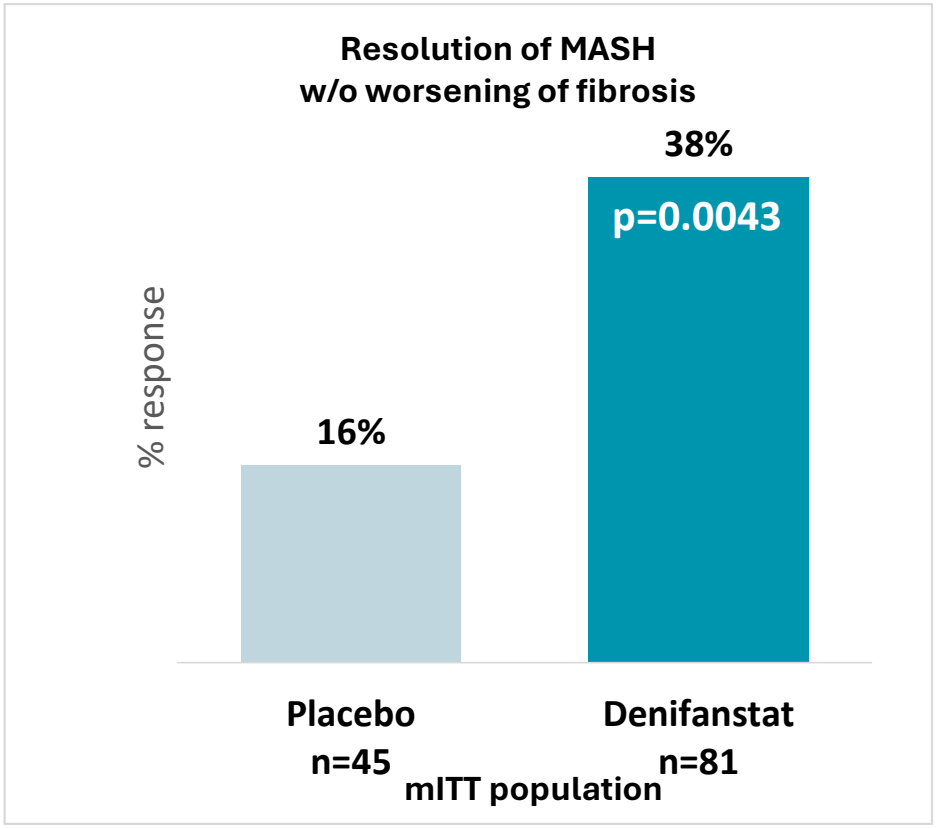


Cochran-Mantel-Haenszel Test – two sided at the 0.05 significance level. * ≥1-point improvement in ballooning or inflammation.

Secondary Endpoints: Liver Fibrosis and MASH Resolution

Denifanstat Achieved Statistical Significance





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

Secondary Endpoints: Liver Fibrosis

Denifanstat Achieved Profound Improvement of Fibrosis

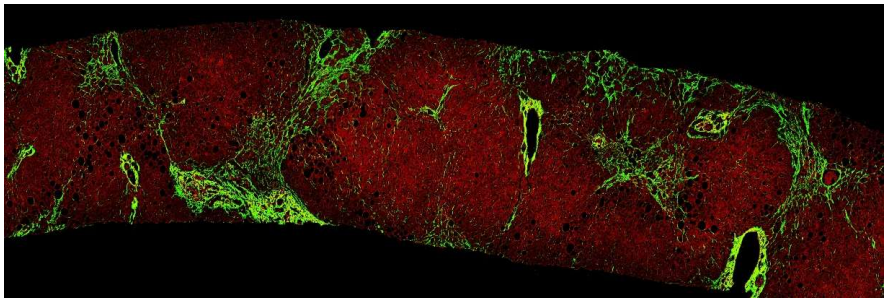
Fibrosis Endpoints	Subgroup	Placebo	Denifanstat	p-value
≥1 stage improvement in fibrosis w/o worsening of MASH	ITT	14%	30%	0.040**
	mITT	18%	41%	0.0103**
	F3	13%	49%	0.0032**
≥2 stage improvement in fibrosis w/o worsening of MASH	mITT	2%	20%	0.0065**
	F3	4%	34%	0.0065**
Progression to cirrhosis (F4)	mITT	11%	5%	0.0386*

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

Additional Fibrosis Analysis Using AI-based Digital Pathology

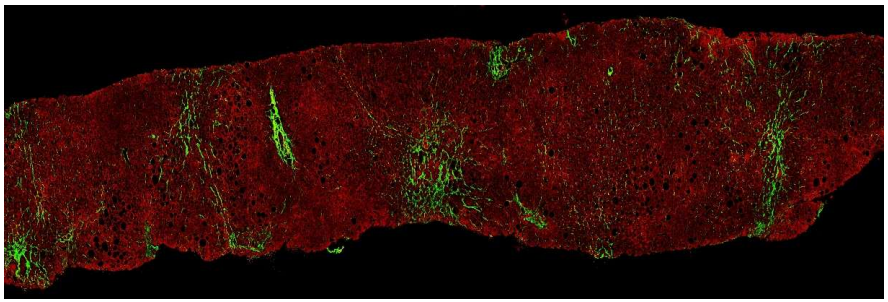
Supporting Evidence that Denifanstat Significantly Reduced Fibrosis

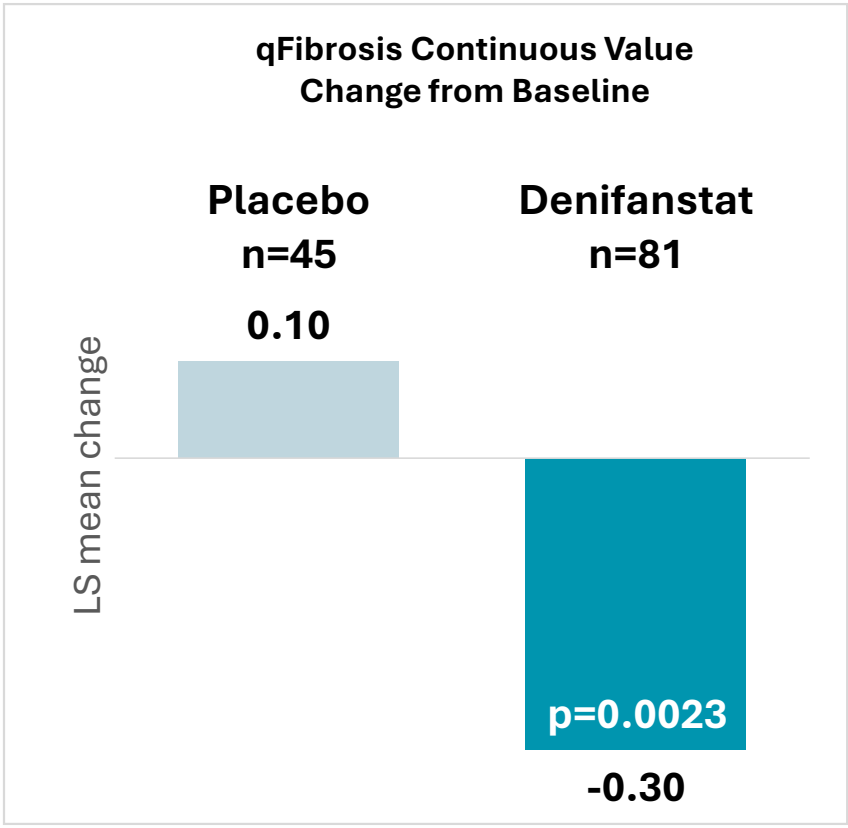
Pre-Treatment Pt A
NASH-CRN Fibrosis stage F3



Denifanstat

Post-Treatment Pt A
NASH-CRN Fibrosis stage F1

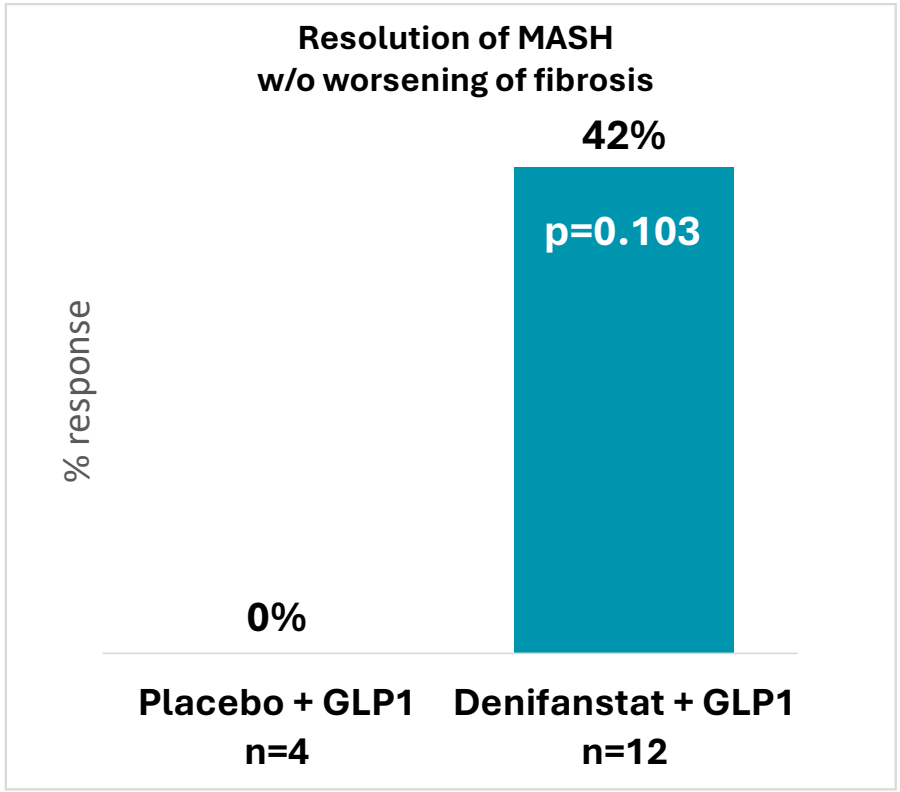


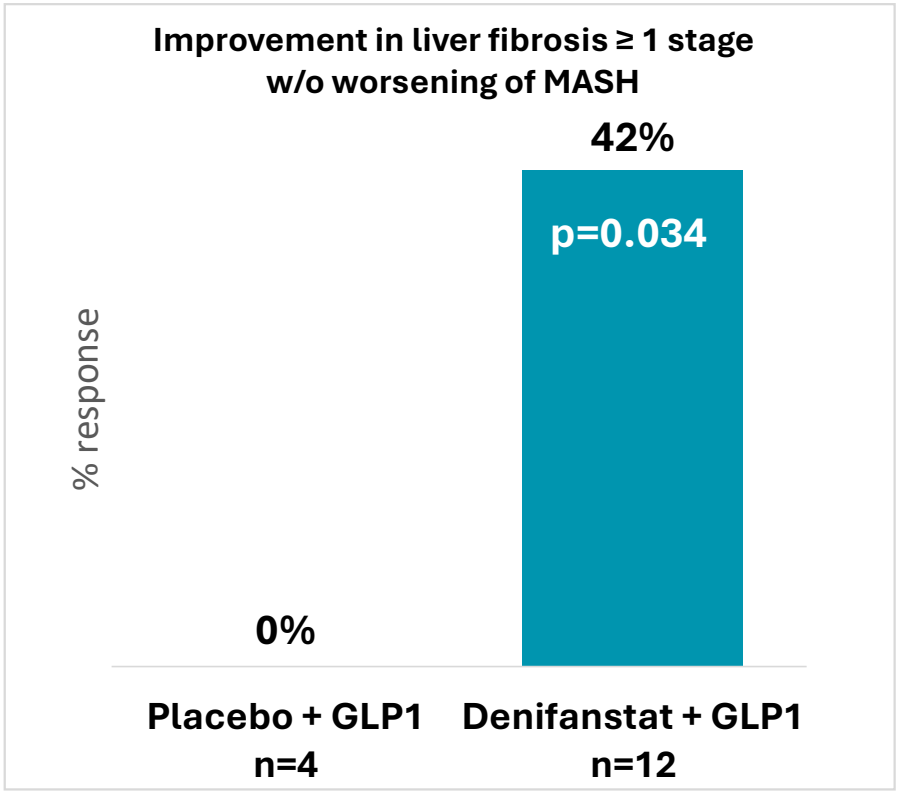


*One sided at the 0.05 significance level

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

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

FASCINATE-2: Safety

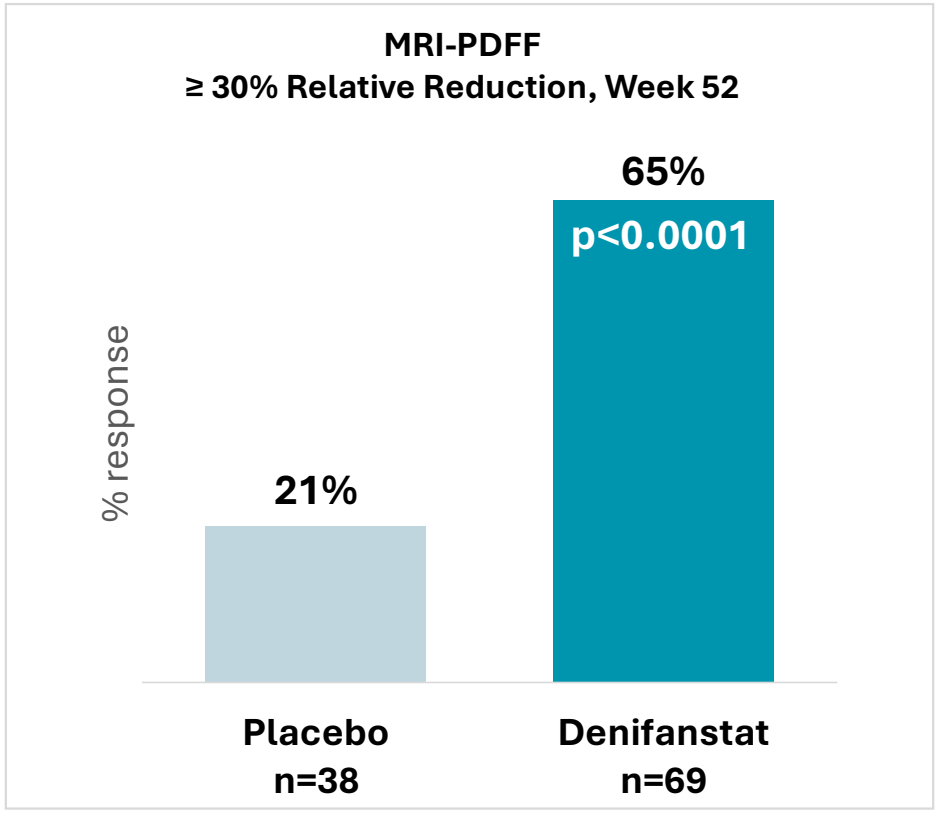
Denifanstat Was Generally Well Tolerated

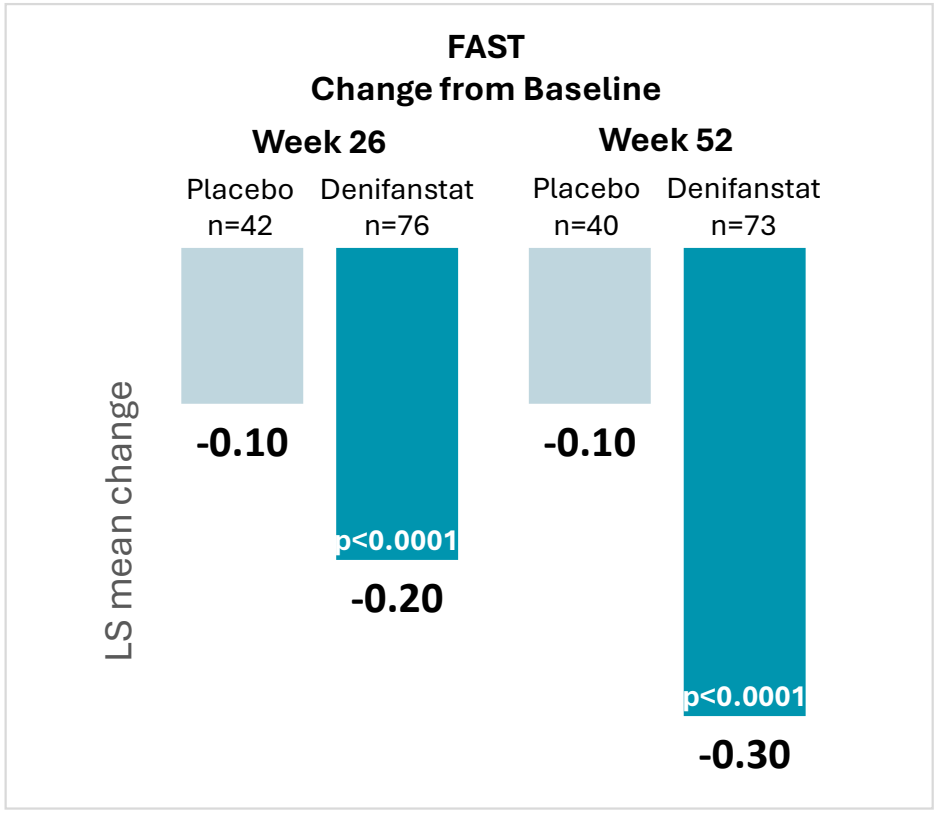
Event n (%)	Placebo (n=56)	Denifanstat 50mg (n=112)	Total (n=168)
Any adverse event	46 (82.1)	99 (88.4)	145 (86.3)
Adverse event related to denifanstat or placebo	20 (35.7)	51 (45.5)	71 (42.3)
Serious adverse event	3 (5.4)	13 (11.6)	16 (9·5)
TEAE leading to study drug discontinuation	3 (5.4)	22 (19.6)	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 signal and no muscle wasting were detected, and GI 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
  - Consistent with other MASH-related medications, only 6% of patients discontinued from the study with hair thinning
  - In previous clinical studies of denifanstat, <2% of the patients experienced hair thinning at 50mg

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

Denifanstat Achieved Statistical Significance



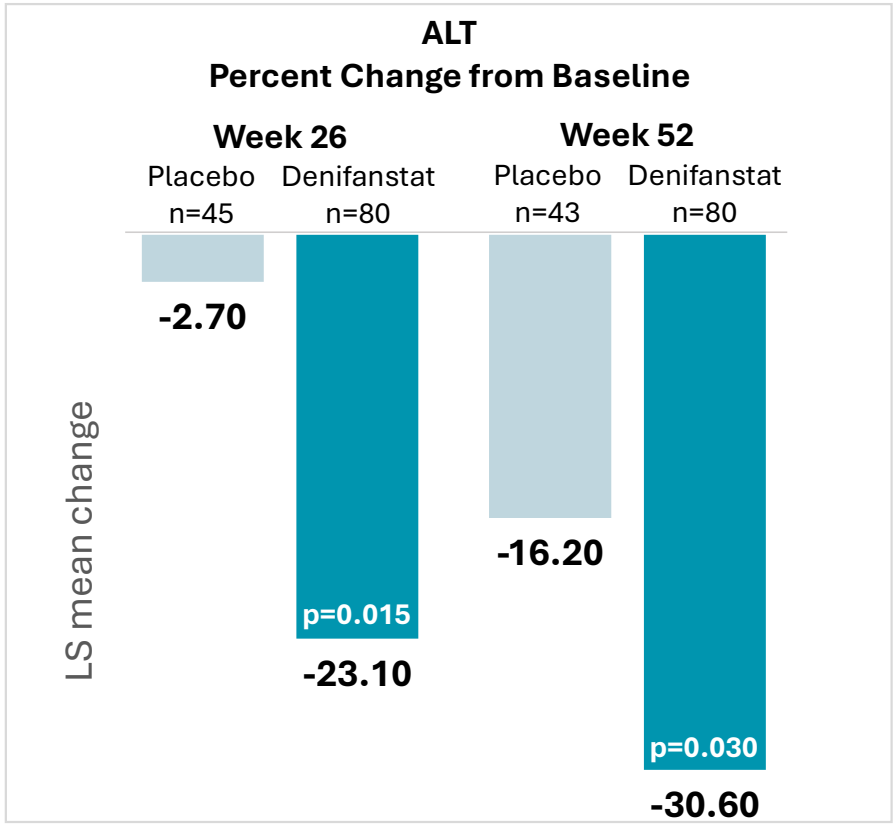


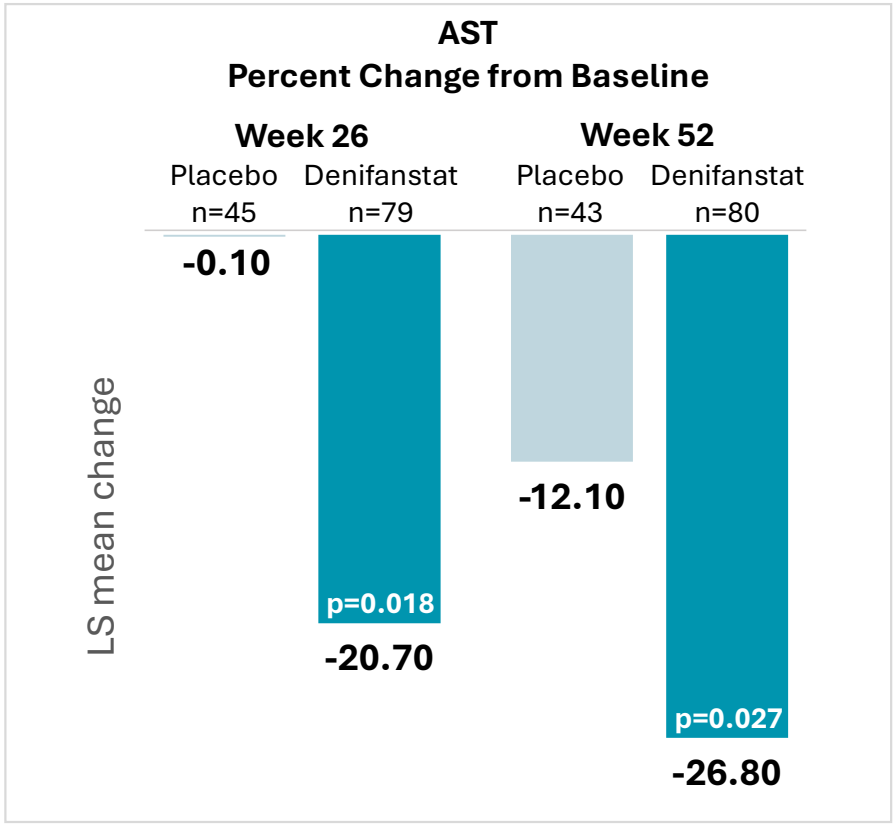
≥30% reduction: Cochran-Mantel-Haenszel Test. Relative reduction: Mixed-effects Model for Repeated Measures. mITT population. Two sided at the 0.05 significance level.

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

Secondary Endpoints: Liver Enzymes

Denifanstat Decreased ALT and AST Levels

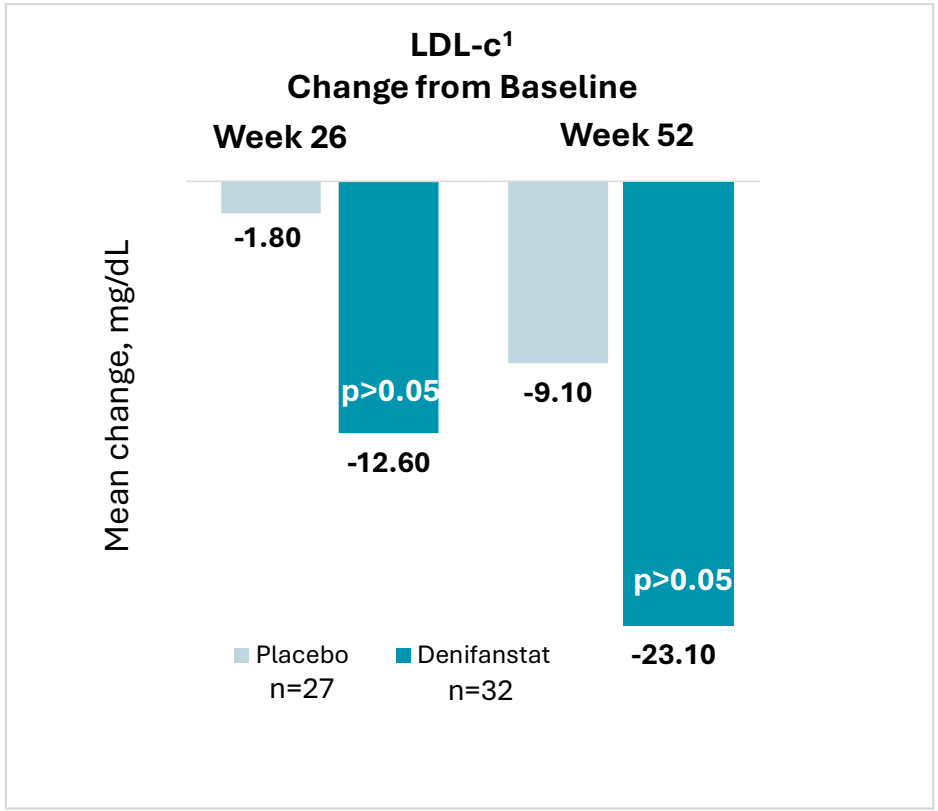


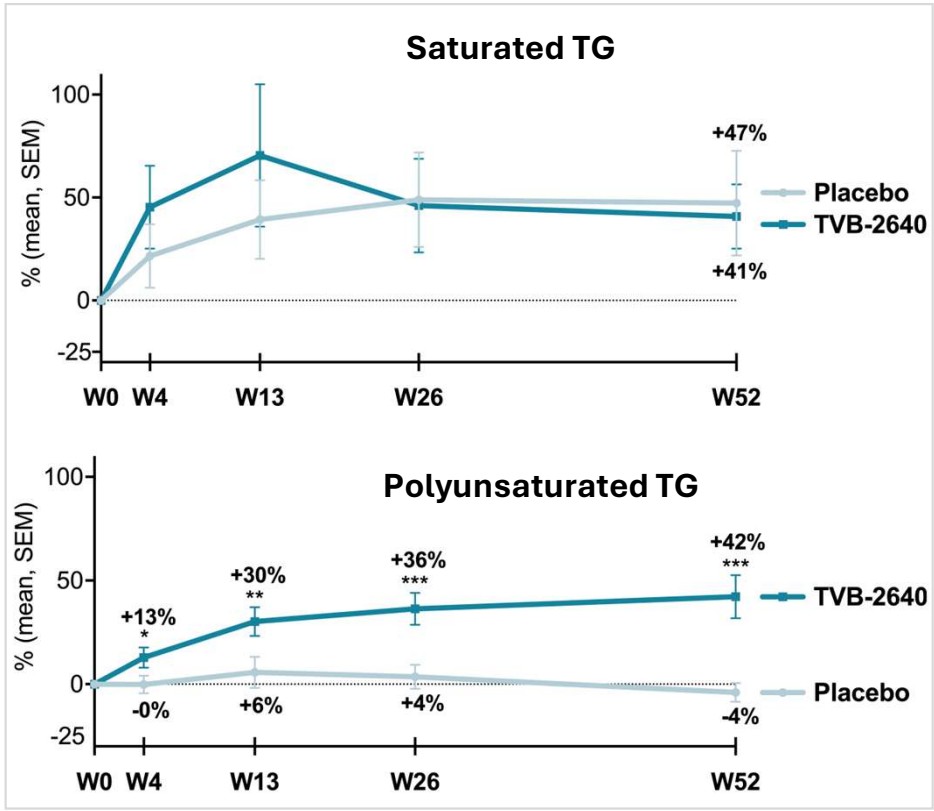


Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. mITT population

Cardiometabolic Health

Denifanstat Decreased LDL-c Levels and Increased Polyunsaturated Triglycerides

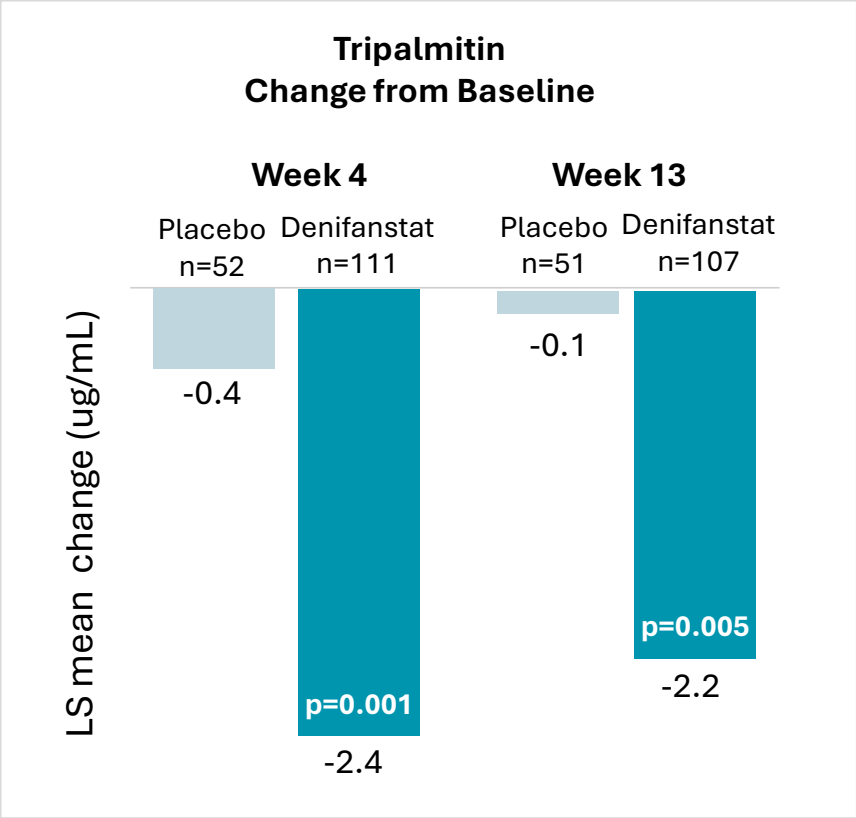




mITT population. Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. *p<0.05, **p<0.01, ***p<0.001

[1]For LDL-c, baseline > 100 mg/dL.

Denifanstat Rapidly and Robustly Reduced De Novo Lipogenesis



Tripalmitin

- A saturated triglyceride which is a biomarker of DNL inhibition
- Rapidly reduced by denifanstat as early as 4 weeks 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

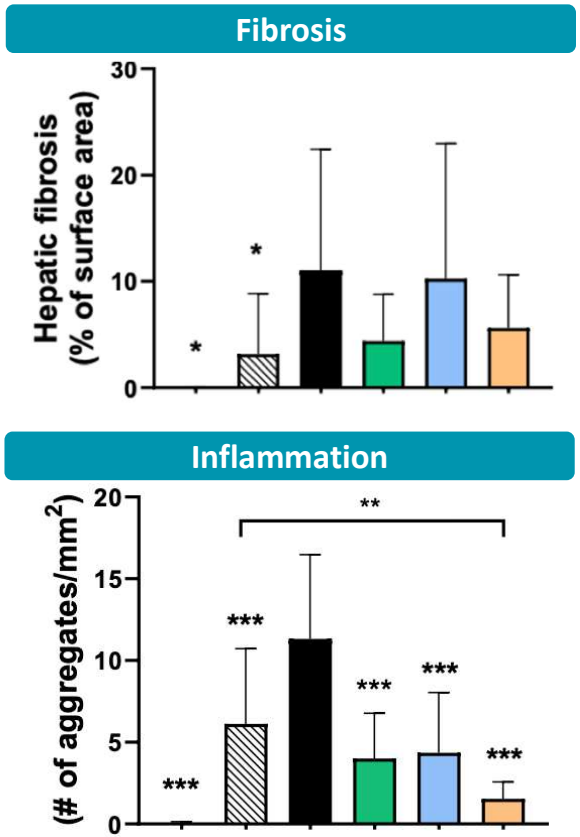
Mechanism of Action Supports Combination Therapy Opportunity

Potential improved clinical outcome for patients with combination therapy of denifanstat + fat burners

Combination therapy offers:

- Denifanstat MOA that is complementary to other MOAs – resmetirom, GLPs
- Opportunity for fixed dose combinations with other oral medications

Preclinical combination studies ongoing with a variety of other MASH, diabetes, metabolism and obesity molecules



MOA- Mechanism of Action

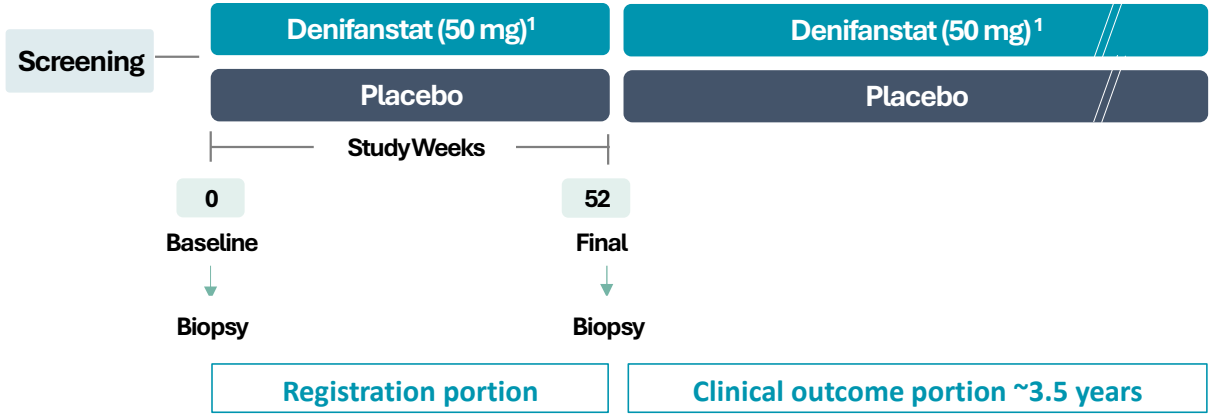
Tsai et al., EASL 2024, LDL knock-out MASH mice. * p<0.05; ** p<0.01; *** p<0.001

Phase 3 Program for Denifanstat in MASH

Multiple sites activated and patients in pre-screening

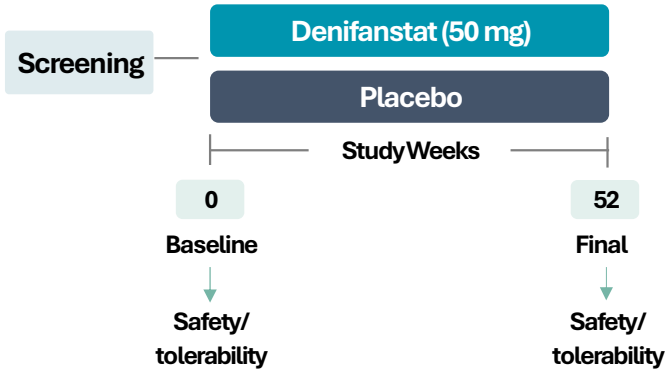
FASCINATE-3

- 1,260 biopsy confirmed F2-F3 MASH patients
- 52 weeks,double blind, placebo-controlled[1]
- Primary endpoints: liver biopsy assessments at 52 weeks, at which time Sagimet plans to seek accelerated approval in the US and Europe; the trial will continue until the required number of clinical outcomes is reached, estimated at 3.5 years



FASCINIT

- Up to 2,000 patients with suspected or confirmed diagnosis of MASLD/MASH
- 52 weeks, double blind, placebo controlled
- Primary endpoints: safety and tolerability at 52 weeks
- Secondary endpoints: non-invasive biomarkers



1 Study to include exploratory arm of ~100 patients on denifanstat 25mg

Denifanstat Potential in Cirrhotic (F4) Patients

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 cirrhotic (F4) patients[1]
- Hepatocytes continue to be functional, and patients frequently have increased liver fat

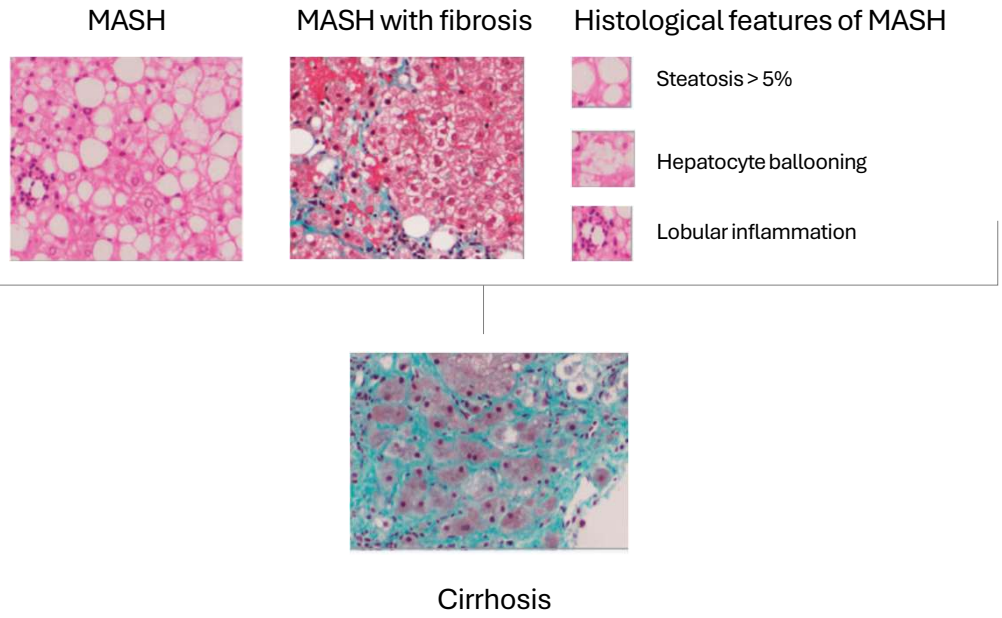
Supportive Clinical Data

- PK profiles in cirrhotic (F4) patients in the Phase 1 impaired hepatic function study[3]
- Positive impact on advanced fibrosis in patients in FASCINATE-2[4]

Next Step

- Phase 2b/3 trial in cirrhotic (F4) patients

~20% of Patients Progress to Cirrhosis [2]



1 Kamm DR and McCommis KS. doi: 10.1113/JP281061. 2 Sheka AC, et al. doi:10.1001/jama.2020.2298. 3. CLIN-009 data on file. 4. Loomba, et al. doi: 10.1016/S2468-1253(24)00246-2

Pediatric MASH Continues to be an Area of Significant Unmet Need

Pediatric MASH

- The prevalence rate of childhood MASLD is estimated at 5-10% in the general population and 10-20% of children with MASLD have advanced fibrosis[1]
- Pediatric MASLD has unique and aggressive histological features[2,3]
- Drugs approved for adults may not have the same efficacy in children[2]
- Effective therapies are urgently needed in pediatric patients[2]

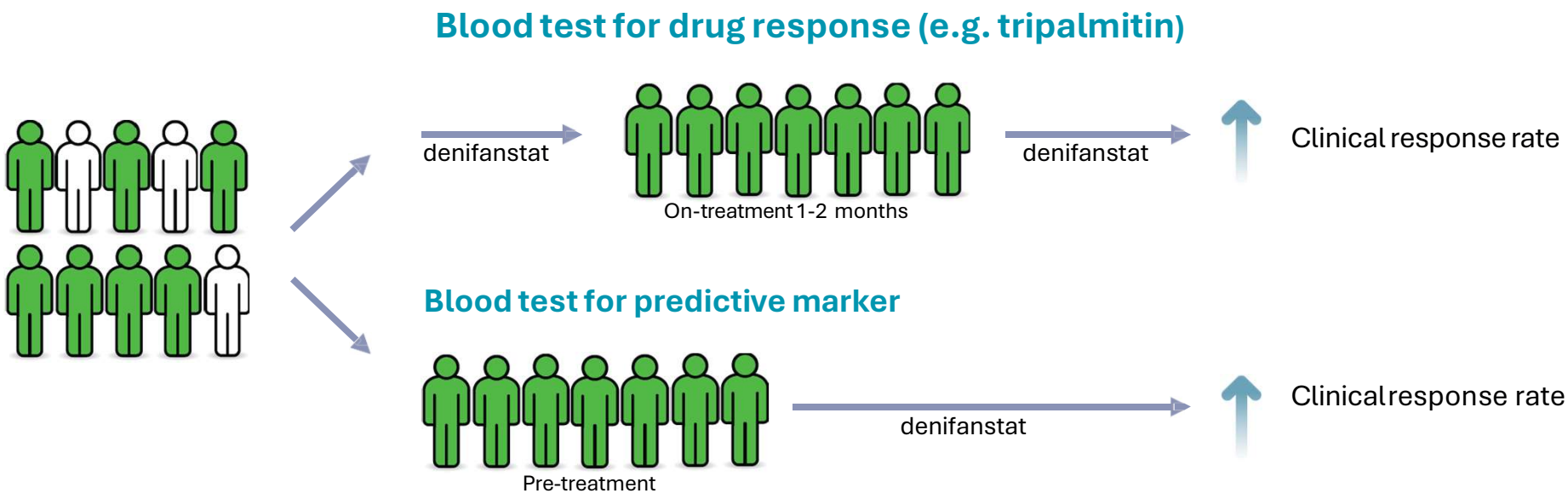
Next steps

- Phase 2 trial in pediatric MASH following:
  - Compilation of safety data across all denifanstat studies in young adults including FASCINATE-2
  - Nonclinical toxicology study in juvenile animals
  - Engagement with FDA

1Yu EL and Schwimmer JB. doi: 10.1002/cld.1027. 2 Softic S and Rohit K. doi: 10.1002/hep.32322. 3 Kleiner DE and Makhlouf HR. doi: 10.1016/j.cld.2015.10.011.

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]



1Signature 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%.

Additional Denifanstat Indications

SAGIMET BIOSCIENCES

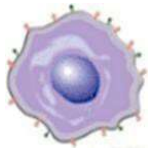
FASN Also Plays a Key Role in Other Diseases With Significant Unmet Need

FASN

Palmitate Lipid building block

Cancer cell

Membrane synthesis, intracellular signaling, protein modification



Sebocyte

Sebum production

FASN in cancer

Supports tumor survival

Enables tumor proliferation

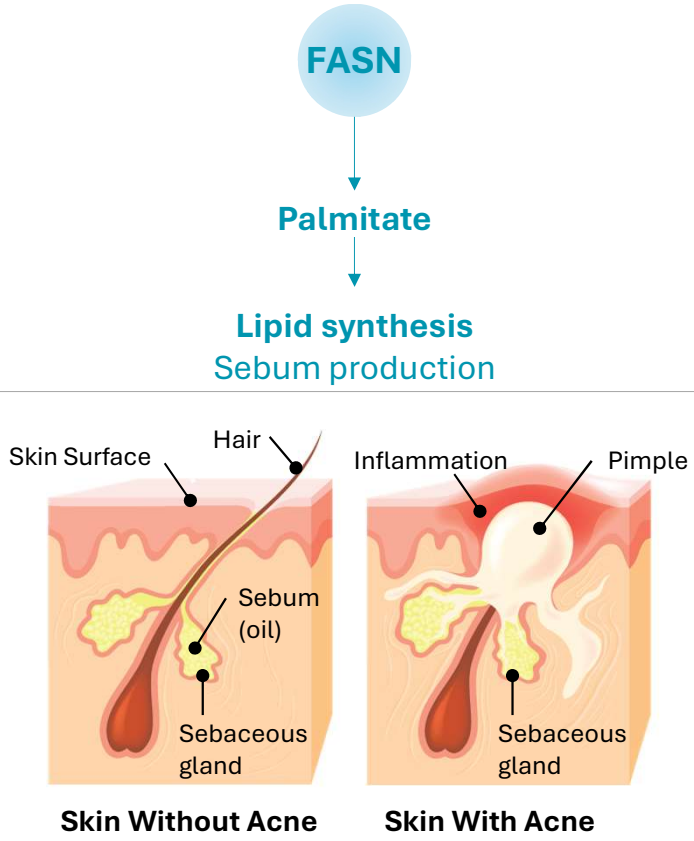
Establishes resistance to drugs

FASN in acne

Increases sebum production

Exacerbates sebum composition

DNL Pathway Plays a Critical Role in the Pathogenesis of Acne



Sebum is a significant part of acne pathogenesis

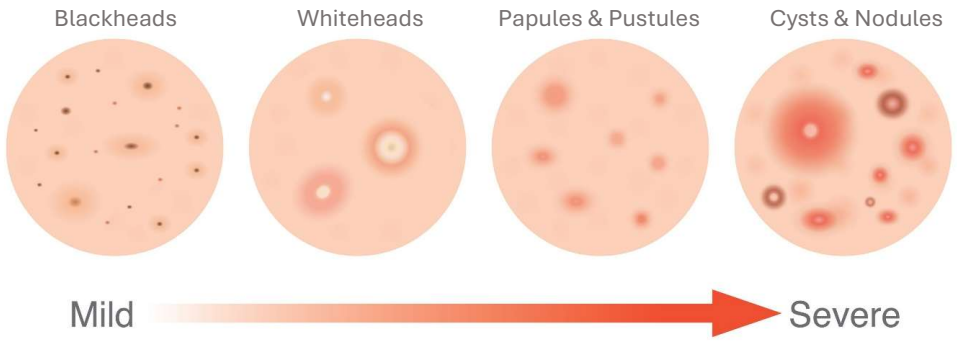
- Acne is associated with sebum overproduction by sebocytes in the skin
- Sebocytes rely on DNL/FASN to produce >80% of key sebum lipids such as palmitate and sapienic acid

FASN is an attractive therapeutic target for acne

- Acne clearance is directly associated with reduced sebum production
- Denifanstat directly reduced cutaneous (skin) sebum DNL lipids in two Phase 1 studies

Acne US Market Overview

Acne market in dermatology is large and highly aligned to a FASN inhibitor TPP value proposition



5.1 million US acne patients are treated by dermatologists annually (total US acne market is 50 million people)[1] [2]

- Acne is the #1 or #2 patient concern in dermatology offices and 65%+ of patients in dermatology offices have private insurance[3]
- 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 highly aligned to our TPP's value proposition and positioning[3]

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

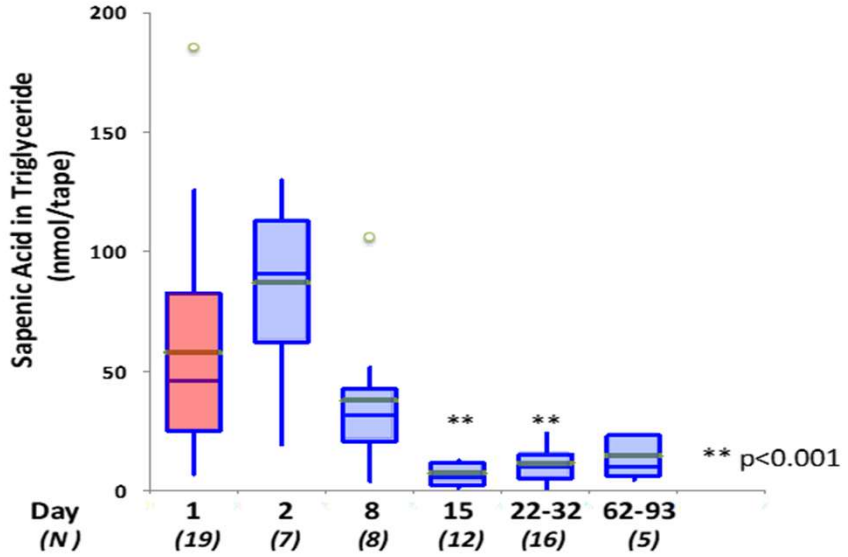
1 Bickers DR, Lim HW, Margolis D, Weinstock MA, Goodman C, Faulkner E et al. The burden of skin diseases: 2004 a joint project of the American Academy of Dermatology Association and the Society for Investigative Dermatology. Journal of the American Academy of Dermatology 2006;55:490-500
2 American Academy of Dermatology/Milliman. Burden of Skin Disease. 2017. www.aad.org/BSD
3 Sagimet market research conducted in July 2024 among 50 dermatologists, data on file

Data Support Mechanism of Action of a FASN Inhibitor in Acne

In multiple Phase 1 studies, FASN inhibitor demonstrated a decrease in DNL sebum lipids[1,2]

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

Phase 1 oncology study
Sebutape® assessment of cutaneous sebum lipids[1]



Days on therapy (# of subjects)

1 EASL 2017, Duke et al. /https://sagimet.com/wp-content/uploads/2017/05/3VBIO_EASLposter.pdf, Falchook et al. EClinicalMedicine 34 (2021) 100797

2 AASLD 2016, Duke et al., https://sagimet.com/wp-content/uploads/2016/11/2016_AASLD_FASN_NASH_36x60_v10.pdf

Ascletis Announced Positive Phase 2 Clinical Data in Acne Phase 3 Study Ongoing

Denifanstat Phase 2 in acne
by Ascletis in China

歌礼 ascletis

	EFFICACY RESULTS – 12 WEEKS			
	Placebo n=45	25 mg n=45	50 mg n=44	75 mg n=45
Total lesions^	-34.9%	-49.5%**	-51.5%**	-48.4%**
Inflammatory lesions^	-36.5%	-54.7%**	-56.7%**	-49.4%*
Non-inflammatory lesions^	-35.0%	-44.4%	-46.6%	-46.5
IGA (2-grade improvement)	15.6%	31.1%	31.8%	22.2%

* p<0.05. ** p<0.01. ^Lesion data are mean relative reduction from baseline to 12w, n= number in cohort. Ascletis has exclusive rights to denifanstat in Greater China

Phase 3 ongoing
by Ascletis in China

Multi-Center, Placebo-Controlled Phase 3 clinical trial of denifanstat (ASC40) in moderate to severe acne initiated by Ascletis in 4Q2023; enrollment completed in Nov 2024

Sagimet completed IND-enabling studies for its second FASN inhibitor TVB-3567

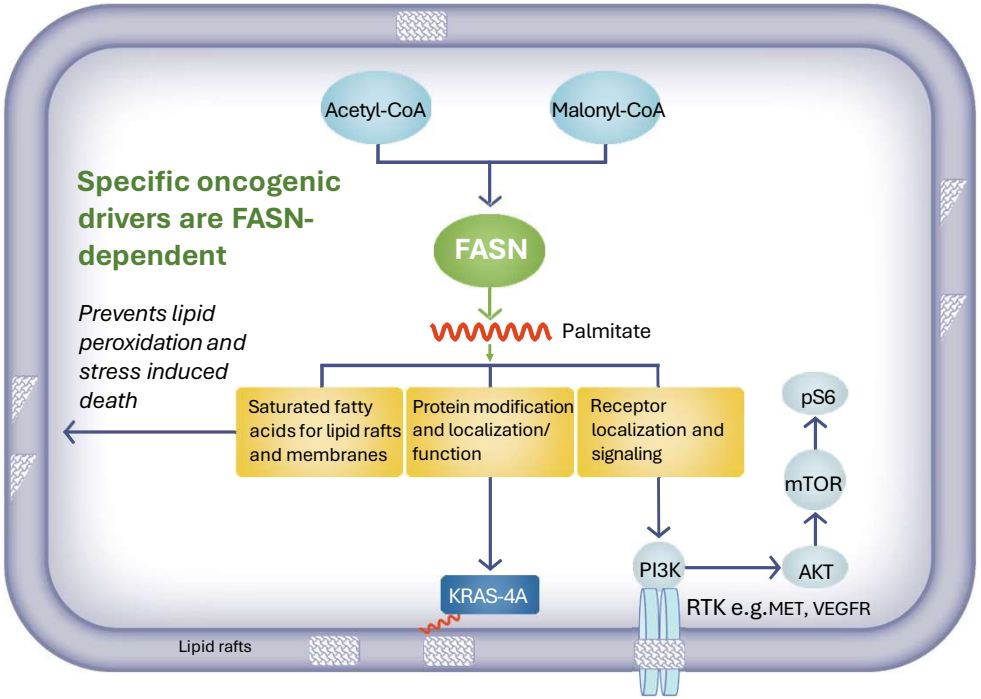
FASN Is Integral to Tumor Cell Proliferation and Survival

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

Cancer Program Focuses on 4 FASN-Dependent Tumor Types

Type	Status	Next milestone
GBM	**Phase 3 ongoing** In China by Ascletis, denifanstat combination with bevacizumab Positive investigator sponsored Phase 2 results*	Phase 3 study completion anticipated by end 2024
Prostate	**Phase 1 ongoing** Investigator Sponsored at Weill Cornell, denifanstat combination with enzalutamide	Phase 1 results expected 4Q2025
HCC	**Translational work ongoing** Patient selection strategy by bioinformatics on primary samples Positive preclinical combination results**	Potential Phase 2 study of FASN inhibitor in combination with a marketed kinase inhibitor, ideally via collaboration with an industry partner
NSCLC KRASM	**Preclinical and clinical evidence** Positive preclinical combination with KRAS inhibitor*** Encouraging monotherapy Phase 1 results with denifanstat	Potential Phase 2 study of FASN inhibitor in combination with a KRAS inhibitor, ideally via collaboration with an industry partner

*Brenner et al., 2023; **Wang at al., 2022; *** GBM (glioblastoma), HCC (hepatocellular carcinoma), KRASM (mutant KRAS), NSCLC (non small cell lung cancer)

Denifanstat: Differentiated MOA with Potential to Treat Multiple Disease States

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 disease states
- Clinical data demonstrates denifanstat's proof of concept across multiple disease states
- Denifanstat is highly differentiated as the only fat synthesis inhibitor currently in development

Phase 3 MASH program

- Denifanstat directly targets the 3 key drivers of MASH – liver fat, inflammation, and fibrosis
- Successful outcome of Phase 2b study; met both primary endpoints with significant reduction in fibrosis
- FDA Breakthrough Therapy designation granted for treatment of MASH (F2-F3 fibrosis)
- Phase 3 program initiated with sites activated & patients pre-screened in 4Q2024, FPI anticipated 1Q2025

Strategic Collaboration with Ascletis in Acne & Cancer

- Acne Phase 3 study enrollment completed in Nov 2024; topline results expected in 2Q2025
- GBM Phase 3 study in progress

Denifanstat IP Portfolio

- Method of use patent: 2036; Composition of matter patent: 2032
- Opportunities to lengthen one patent's life for up to 5 years via Patent Term Extension (US) or SPC (Europe)

Near Term Milestones & Cash Position

- NASDAQ: SGMT; $170.0M cash, cash equivalents and marketable securities at 3Q2024, expected to fund current operations through 2025
- Currently evaluating financing options to complete clinical development programs across indications