UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): July 2, 2024

SAGIMET BIOSCIENCES INC.
(Exact name of registrant as specified in its charter)

Delaware	**001-41742**	**20-5991472**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(I.R.S. Employer Identification No.)**

Sagimet Biosciences Inc.
155 Bovet Road, Suite 303,
San Mateo, California 94402
(Address of principal executive offices, including zip code)

(650) 561-8600
(Registrant's telephone number, including area code)

Not Applicable
(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

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

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

Title of each class	Trade Symbol(s)	Name of each exchange on which registered
Series A Common Stock, $0.0001 par value per share	SGMT	The Nasdaq Global Market

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

Emerging growth company ☒

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

Item 8.01 **Other Events.**

On July 2, 2024, Sagimet Biosciences Inc. (the "Company") updated information reflected in a slide presentation, which is attached as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated herein by reference. Representatives of the Company will use the updated presentation in various meetings with investors from time to time.

Item 9.01 **Financial Statements and Exhibits.**

(d) Exhibits

Exhibit No.	Document
99.1	Investor Presentation of Sagimet Biosciences Inc., dated July 2, 2024.
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURE

Pursuant to the requirements of the Securities Exchange Act of 1934, as amended, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Sagimet Biosciences Inc.

Date: July 2, 2024

By: /s/ David Happel
David Happel
Chief Executive Officer


SAGIMET
BIOSCIENCES
Targeting Metabolic Dysfunction with Novel Therapies to Treat MASH, Acne & Cancer
July 2024

Forward Looking Statements

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

Moreover, we operate in a dynamic industry and economy. New risk factors and uncertainties may emerge from time to time, and it management to predict all risk factors and uncertainties that we may face. Except as required by applicable law, we do not plan to publicly forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise.

Leadership Team with Proven Development and Commercialization

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 lea
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 e
of legal, IP and compliance functions in

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

Pfizer raptor MedImmune GILEAD DYNAVAX ALEXION Intercept

HORIZON AstraZeneca abbvie Genentech proventionbio NOVARTIS cognoa

SAGIMET BIOSCIENCES

FASN Inhibitor Denifanstat Offers a Unique and Validated Approach t

Unique MOA: FASN Inhibition

- As the only fat synthesis inhibitor, denifanstat directly targets the 3 key drivers of MASH – inflammation, and fibrosis
- FASN is a key enzyme in de novo lipogenesis responsible for excess liver fat in MASH
- Once daily oral administration, suitable for mono- or combination therapy

Positive FASCINATE-2 Phase 2b Data in MASH

- Met both primary endpoints in clinical trial: significant improvements in fibrosis with no v
- Improvement in more severe patients (stage F3) and demonstrated lack of progression to
- Enhanced treatment effect in patients with stable GLP therapy
- Generally well tolerated

Near Term Milestones & Cash Position

- Pivotal Phase 3 program expected to begin in 2H2024
- NASDAQ: SGMT; $193.7M cash* on hand, expected to fund current operations through 2

*Cash, cash equivalents and marketable securities as of March 31, 2024

Precision Medicine

- Tripalmitin and additional blood response markers under development as early biomarke engagement and treatment response

Strategic Collaboration with Ascletis in Acne & Cancer

- Acne Phase 3 study completion of enrollment anticipated by end 2024
- rGBM Phase 3 study interim analysis anticipated by end 2024

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

Denifanstat: A Novel Small Molecule FASN Inhibitor Protected By Str

Denifanstat

- Designed for once-daily, oral dosing
- Rigorous and de-risked development strategy
- Direct DNL inhibition demonstrated in Phase 1b
- Improvements in liver fat and other non-invasive biomarkers in Phase 2a
- Topline data of successfully completed 52-week Phase 2b biopsy study announced i
- Precision medicine approach to improve patient outcomes

Strong patent estate

- Denifanstat method of use expires in 2036
- Denifanstat composition of matter expires in 2032 (issued in all key commercial territo
- Opportunities exist to lengthen patent exclusivity of either composition patent or meth patent for up to 5 years via Patent Term Extension (US) or SPC (Europe)
- Currently building out global patent portfolio to further protect commercialization of de patent applications directed to formulations, methods of use, and synthetic methods, to extend exclusivity further

DNL = de novo lipogenesis

Development Pipeline: Indications and Clinical Milestones

Therapeutic Area	Indication	Stage of Development				Expected Milestone /
		Preclinical	Phase 1	Phase 2	Phase 3	
Metabolic Disease	MASH F2/F3 population			Denifanstat		Phase 2b positive topline d 1Q2024; MASH Phase 3 est
				Denifanstat		Phase 1 hepatic impairmer reported 1Q 2024
Dermatology	Acne		TVB-3567			IND-enabling studies comp timing to file IND
				Denifanstat (ASC40)		Phase 3 clinical study initia to be fully enrolled in 2024*
Oncology	Solid tumors		TVB-3567			Identifying FASN-depender potential FASN inhibitor de
				Denifanstat		
	Recurrent glioblastoma (GBM)			Denifanstat (ASC40)		Phase 3 enrollment of 120 achieved in 3Q 2023; pre-s interim analysis planned in

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

MASH: A Burgeoning Epidemic

Patients in 2016[1]

United States

MASLD	MASH	MASH mod-adv Fibrosis F2-F3	Cirrhosis F4	Hepatocellular carcinoma
85.3 million	**17.3 million**	**5.7 million**	**1.4 million** compensated and decompensated	**11 thousand** annual cases among MASLD population
Metabolic Dysfunction-Associated Liver Disease	Metabolic Dysfunction-Associated Steatohepatitis **F1**			

MASH

- Expected to
  double in siz
  2 decades[2]
- Complex dis
  heterogenec
  population
- Significant o
  differentiate

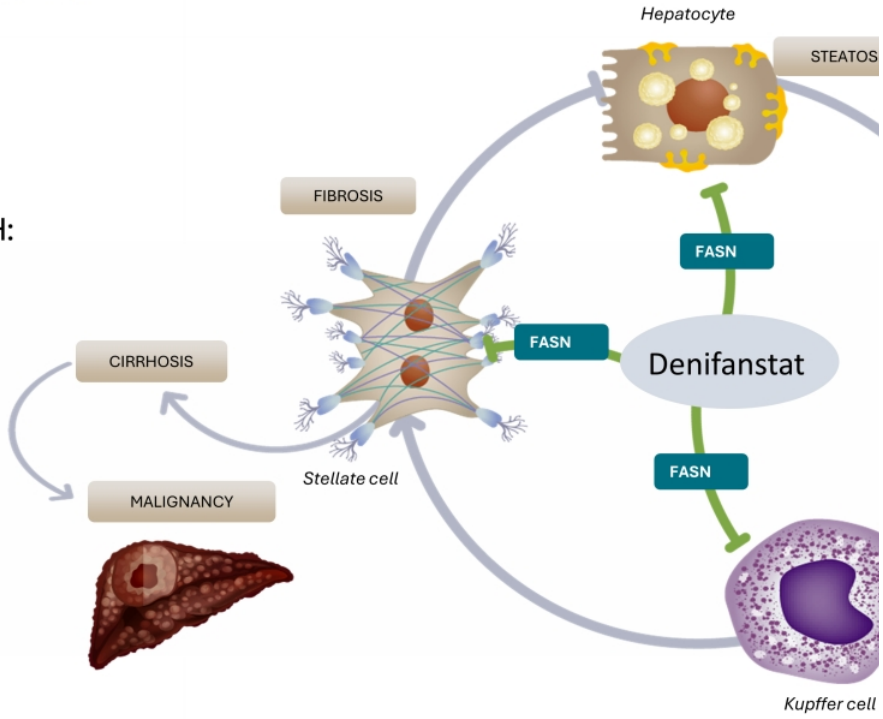
1 Estes, et al. 2018; http://dx.doi.org/10.1016/j.jhep.2018.05.036. Note: MASH, or metabolic dysfunction-associated steatohepatitis, was formerly known as NASH, or nonalcoholic

2 Yonoussi et al. 2023; The Growing Economic and Clinical Burden of Nonalcoholic Steatohepatitis (NASH) in the United States

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:

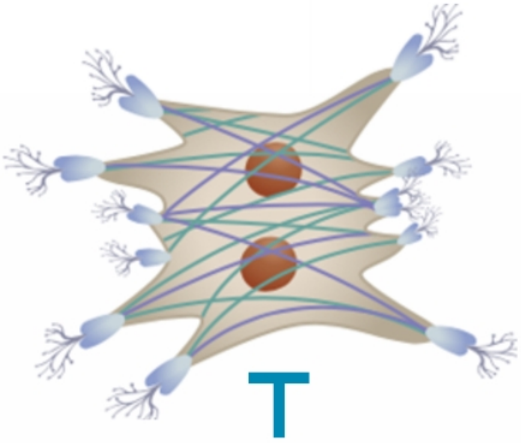
1. Blocking **steatosis** via inhibiting de novo lipogenesis in hepatocytes
2. Reducing **inflammation** via preventing immune cell activation
3. Blunting **fibrosis** via inhibiting stellate cell activation



FASN Inhibition Directly Blocks Human Liver Stellate Cell Function

Stellate cells require DNL for fibrogenesis

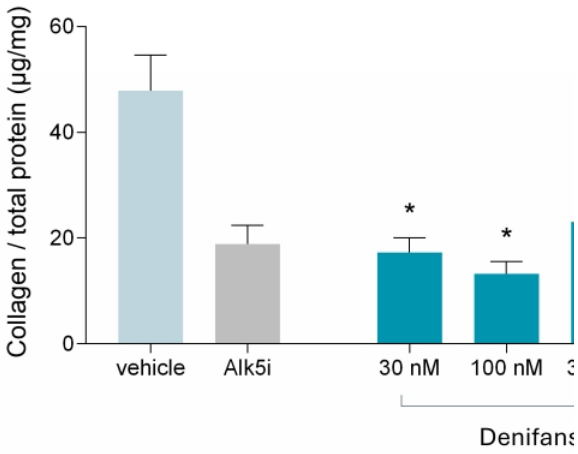
Denifanstat blocks stellate cell activation



Denifanstat

Primary human stellate cell a

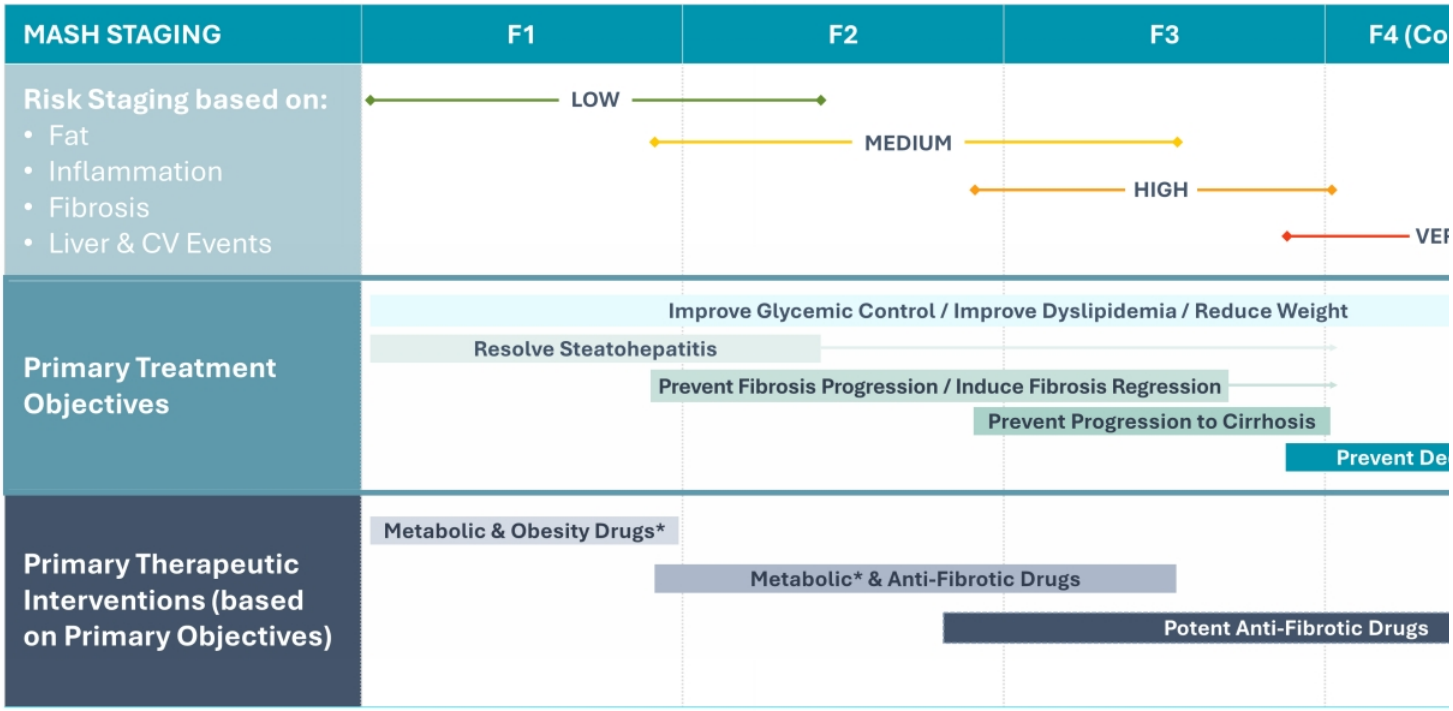
Denifanstat directly inhibits fibroge



- Stimulated by TGF-beta to activate fibro
- Denifanstat showed similar inhibition to 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

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MASH Clinical Development Program

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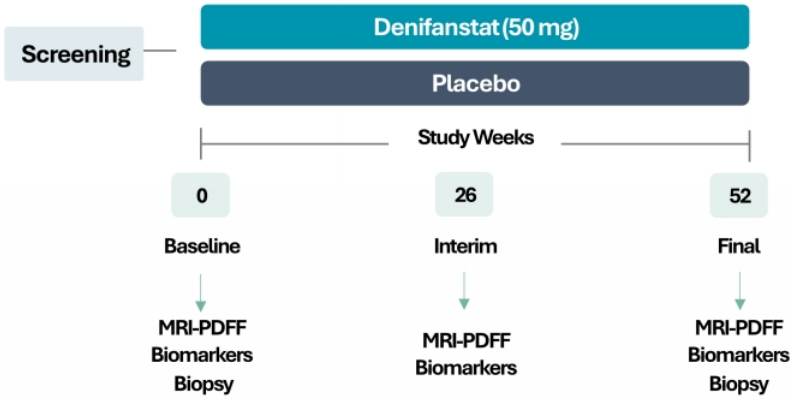
FASCINATE-2: Biopsy Trial Design Focused on Histological Endpoints

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 worse fibrosis
- MASH resolution + NAS ≥2 improveme worsening of fibrosis

Selected secondary endpoints

- Improvement in liver fibrosis ≥1 stage worsening of MASH as assessed by b
- Digital AI pathology
- MRI-PDFF: absolute decrease, % cha baseline, % pts ≥30% reduction from baseline (responders)

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

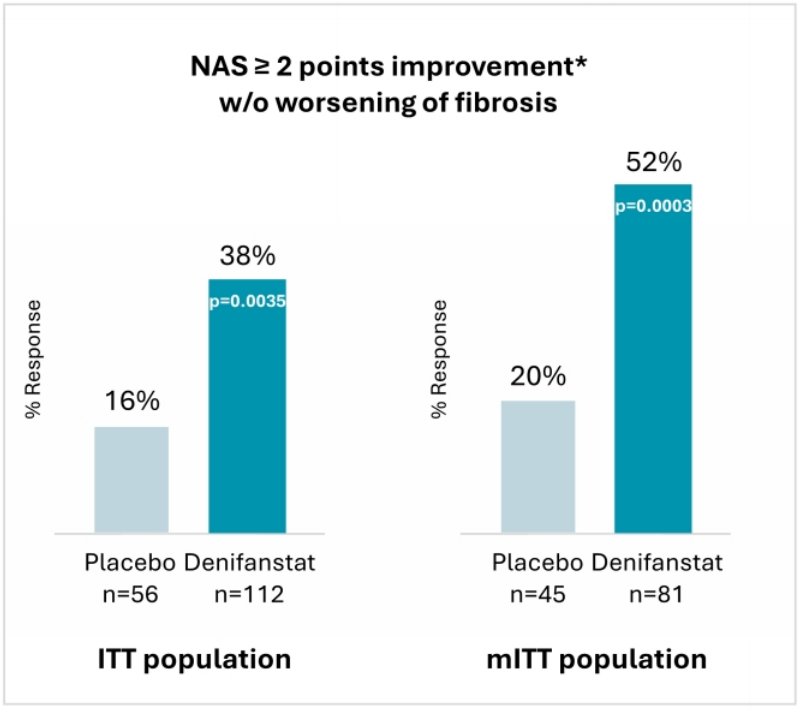
FASCINATE-2: Baseline Characteristics Were Typical of the F2/F3 MASH Po

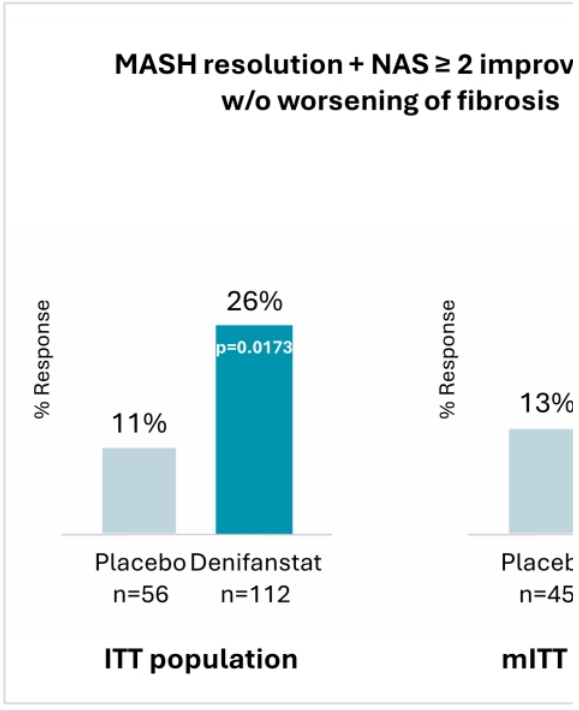
Parameter	Placebo, n=45	Denifanstat, n=81
Age, years	59.6 (+/- 10.9)	56.1 (+/- 10.8)
Sex, female	27 (60%)	48 (59%)
Race, White	41 (91%)	73 (90%)
Ethnicity, Hispanic or Latino	15 (33%)	27 (33%)
BMI, kg/m^2	36.5 (+/- 6.7)	34.6 (+/- 6.1)
Type 2 diabetes	27 (60%)	55 (68%)
ALT (alanine aminotransferase) U/L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

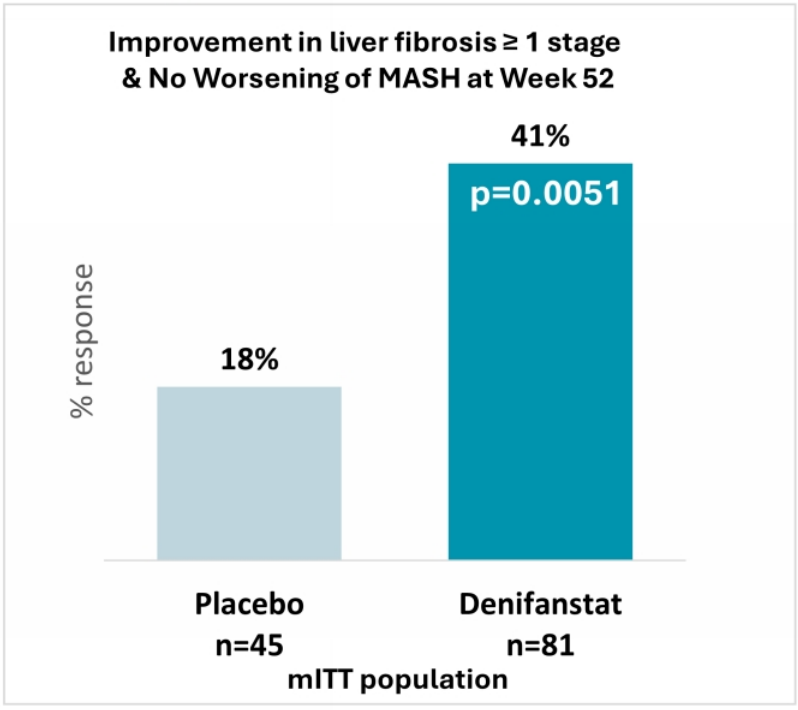




Cochran-Mantel-Haenszel Test – two sided at the 0.05 significance level. * ≥1-point improvement in ballooning or inflammation.

Secondary Endpoint: Liver Fibrosis

Denifanstat Achieved Statistical Significance



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

Secondary Endpoints: Liver Fibrosis

Denifanstat Achieved Profound Improvement of Fibrosis

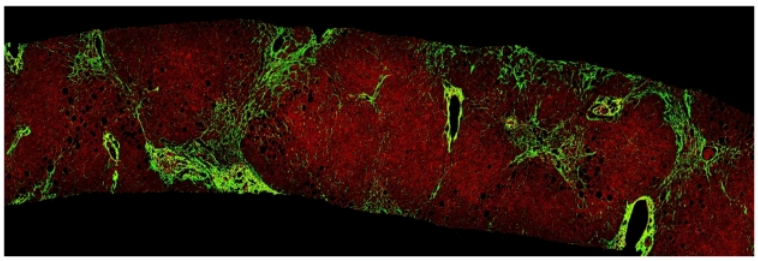
Fibrosis Endpoints	Subgroup	Placebo	Denifanstat
≥1 stage improvement in fibrosis w/o worsening of MASH	ITT	14%	30%
	mITT	18%	41%
	F3	13%	49%
≥2 stage improvement in fibrosis w/o worsening of MASH	mITT	2%	20%
	F3	4%	34%
Progression to cirrhosis (F4)	mITT	11%	5%

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

Additional Fibrosis Analysis Using AI-based Digital Pathology

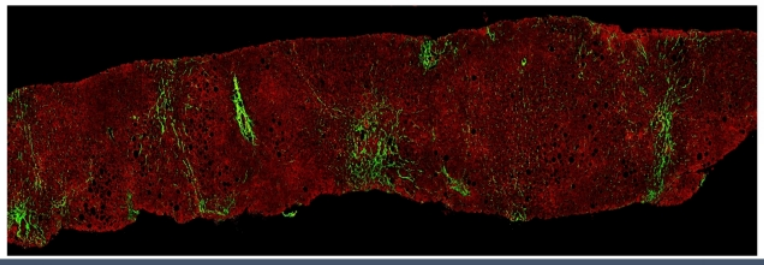
Supporting Evidence that Denifanstat Significantly Reduced Fibrosis

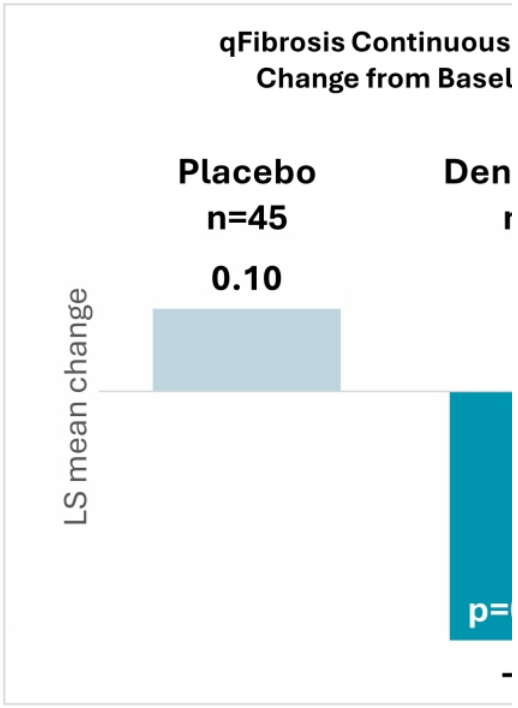
Pre-Treatment Pt A
NASH-CRN Fibrosis stage F3



Denifanstat

Post-Treatment Pt A
NASH-CRN Fibrosis stage F1

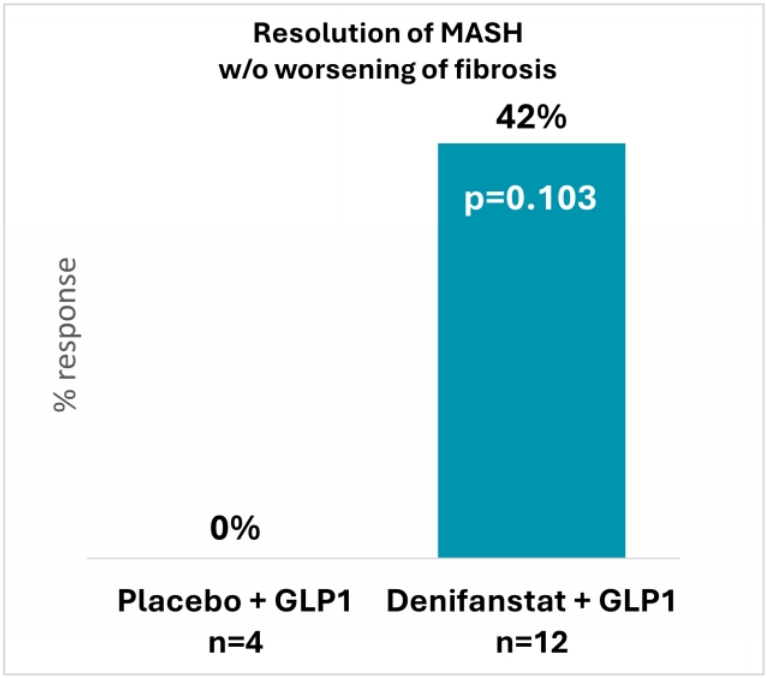


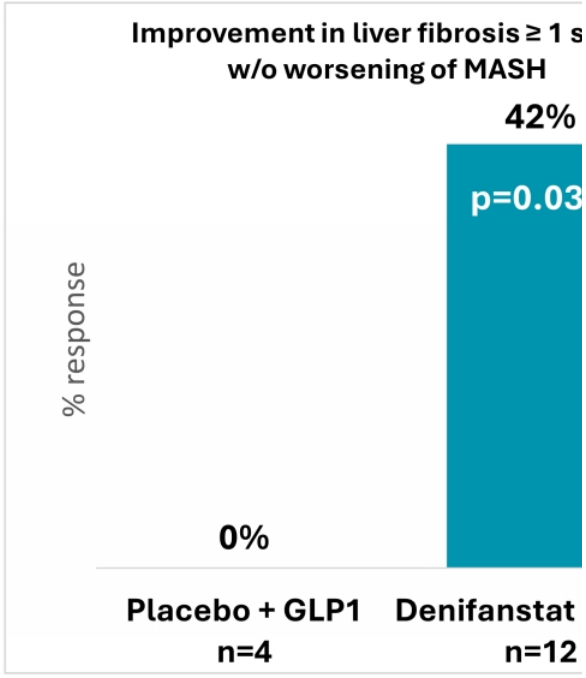


*One sided at the 0.05 significance level

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 re denifanstat

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FASCINATE-2: Safety

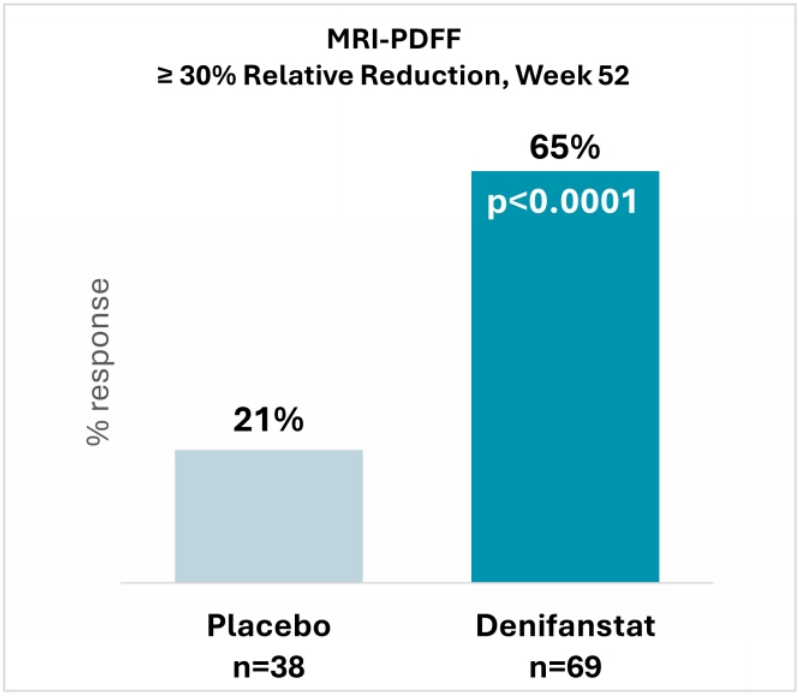
Denifanstat Was Generally Well Tolerated

Event n (%)	Placebo (n=56)	Denifanstat 50mg (n=112)	
Any adverse event	46 (82.1)	99 (88.4)	1
Adverse event related to denifanstat or placebo	20 (35.7)	51 (45.5)	7
Serious adverse event	3 (5.4)	13 (11.6)	
TEAE leading to study drug discontinuation	3 (5.4)	22 (19.6)	2
Adverse events affecting ≥ 10% of patients			
COVID-19	6 (10.7)	19 (17.0)	2
Dry eye	8 (14.3)	10 (8.9)	1
Hair thinning	2 (3.6)	21 (18.8)	2

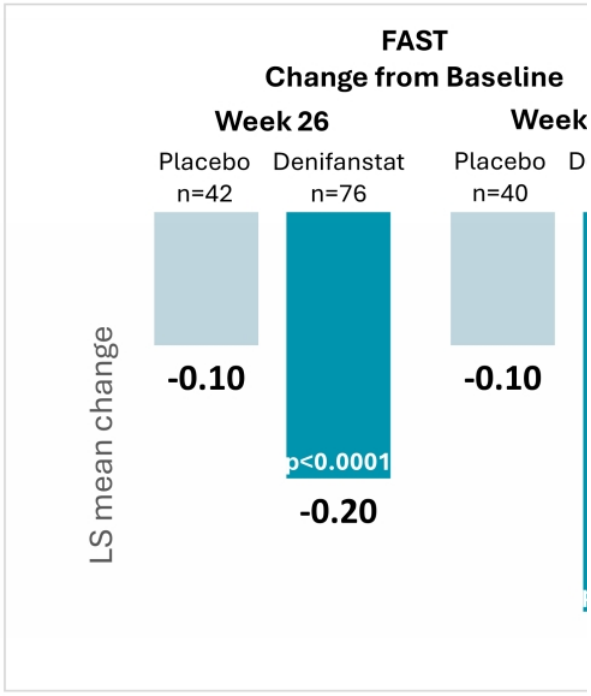
- No DILI signal and no muscle wasting were detected, and GI were comparable to placebo
- AE of hair thinning stabilized with a 2 to 4 week dose pause and then reversed with down titration or study completion
  - Consistent with other MASH-related medications, only 6% of patients discontinued from the 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 Sco

Denifanstat Achieved Statistical Significance



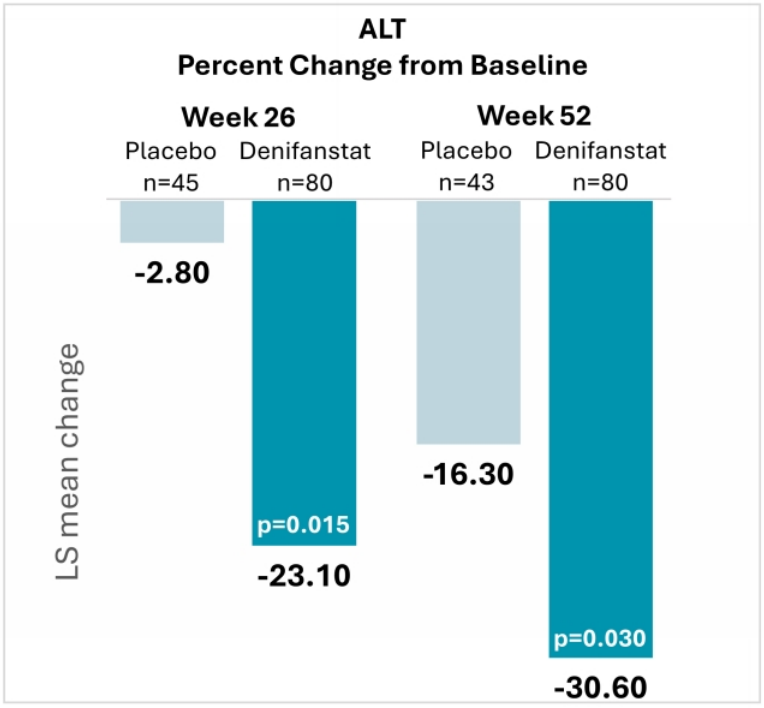
≥30% reduction: Cochran-Mantel-Haenszel Test. Relative reduction: Mixed-effects Model for Repeated Measures. mITT population. Two sided at the 0.05 significance level.

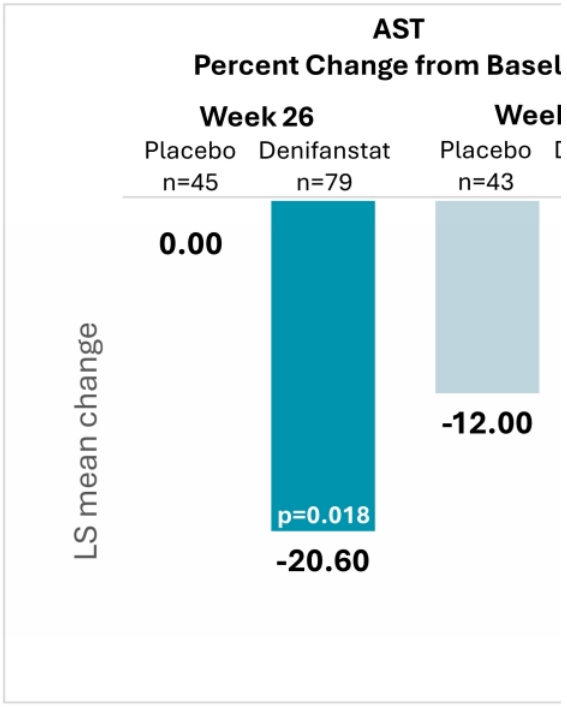


Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. mITT population.

Secondary Endpoints: Liver Enzymes

Denifanstat Decreased ALT and AST Levels

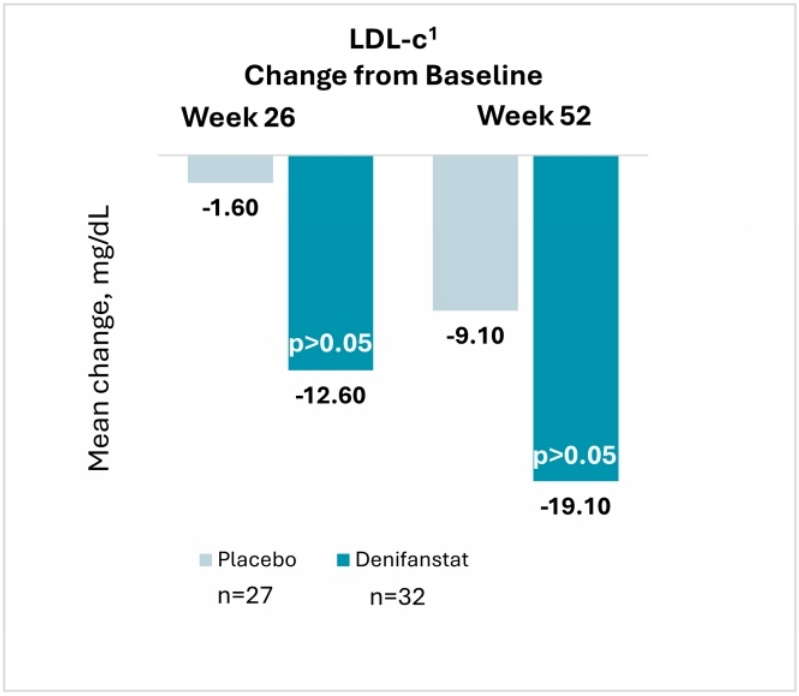


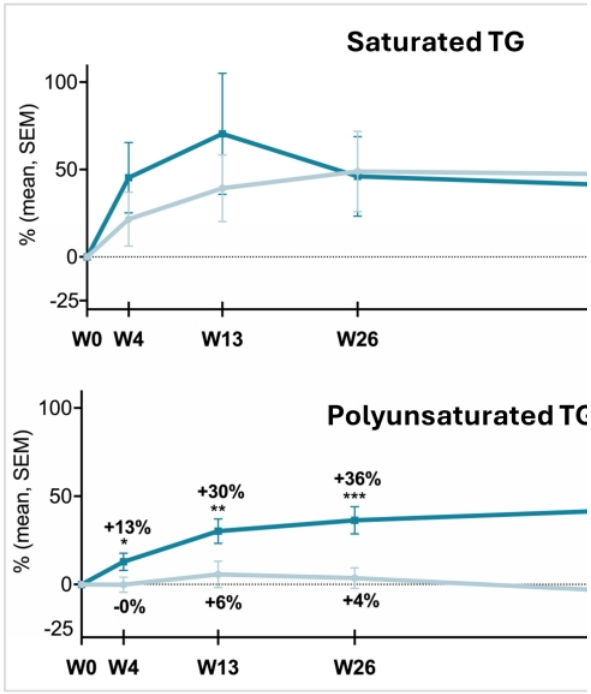


Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. mITT population

Cardiometabolic Health

Denifanstat Decreased LDL-c Levels and Increased Polyunsaturated Triglycerides

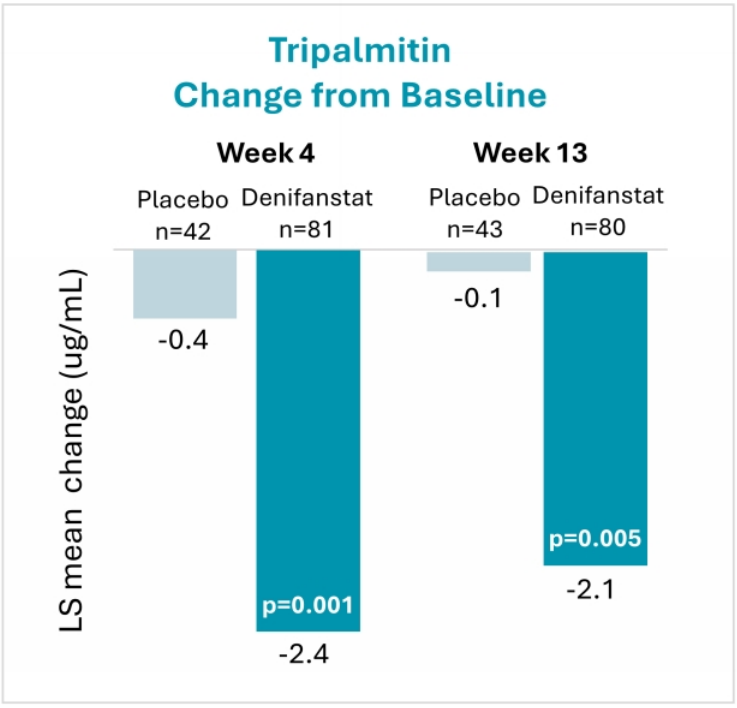




mITT population. Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. *p<0.05, **p<0.01, ***p<0.001

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

Denifanstat Rapidly and Robustly Reduced De Novo Lipogenesis



Tripalmitin

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

Next steps

- Continue the development of tripalmitir additional markers as potential biomark of treatment response for denifanstat

Two sided at the 0.05 significance level

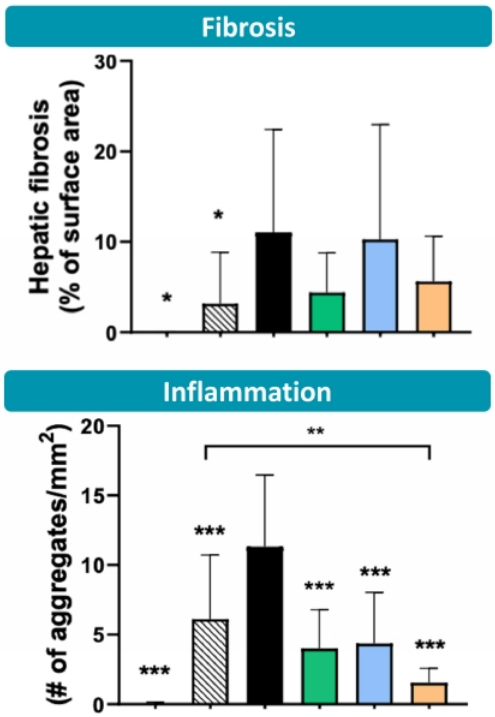
Mechanism of Action Supports Combination Therapy Opportunity

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

Denifanstat Potential in Cirrhosis

Compensated Cirrhotic Patients (MASH F4)

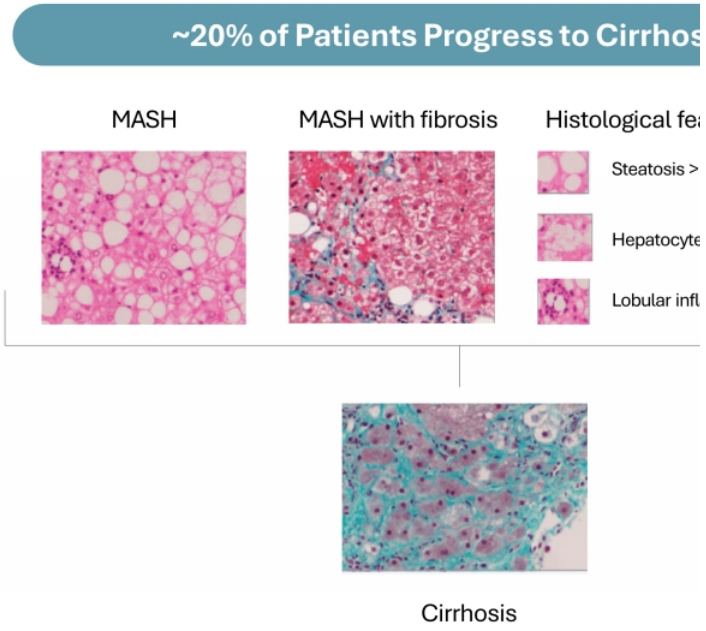
- Denifanstat reduces pro-fibrotic signaling stellate cells which retain the ability to remove the fibrotic scar and reestablish the basal ECM scaffold even in F4 MASH [1]
- Hepatocytes continue to be functional, and patients frequently have increased liver fat

Supportive Initial Data

- PK profiles in 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 MASH-F4



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. EASL 2024

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]

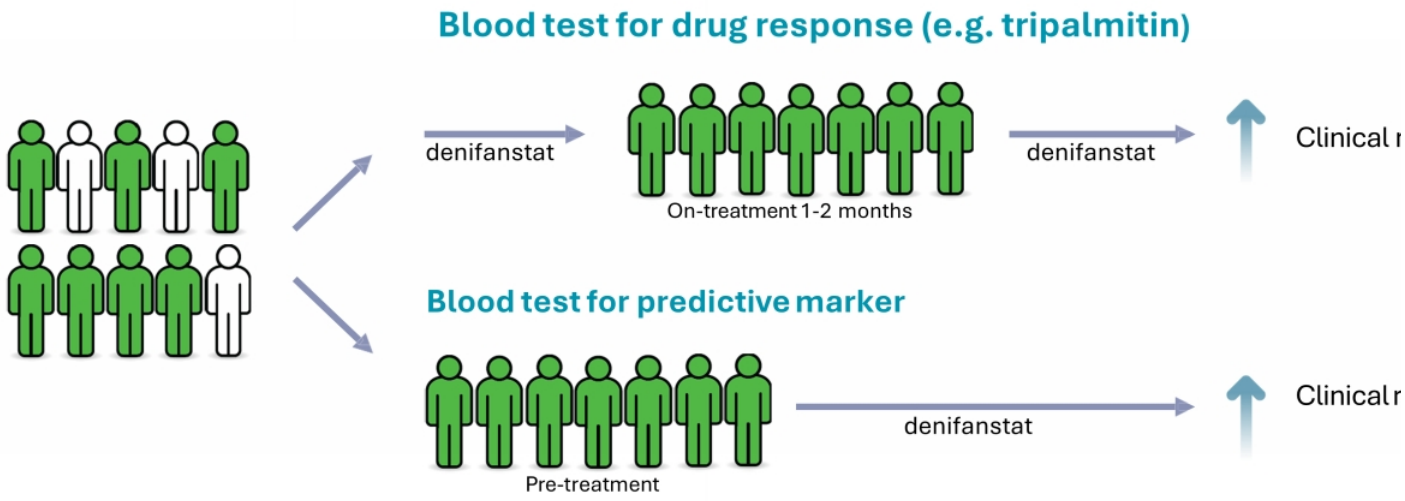
Next steps

- 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. 2Softic S and Rohit K. doi: 10.1002/hep.32322. 3Kleiner DE and Makhlouf HR. doi: 10.1016/j.cld.2015.10.011.

Precision Medicine: Blood Tests May Lead to Improved Patient Outco

- MASH is a multi-faceted disease and patients may benefit from being matched with optimal t
- 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%, PP

Additional Denifanstat Indications

SAGIMET BIOSCIENCES

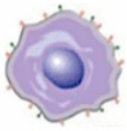
FASN Also Plays a Key Role in Other Diseases With Significant Unmet Ne

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

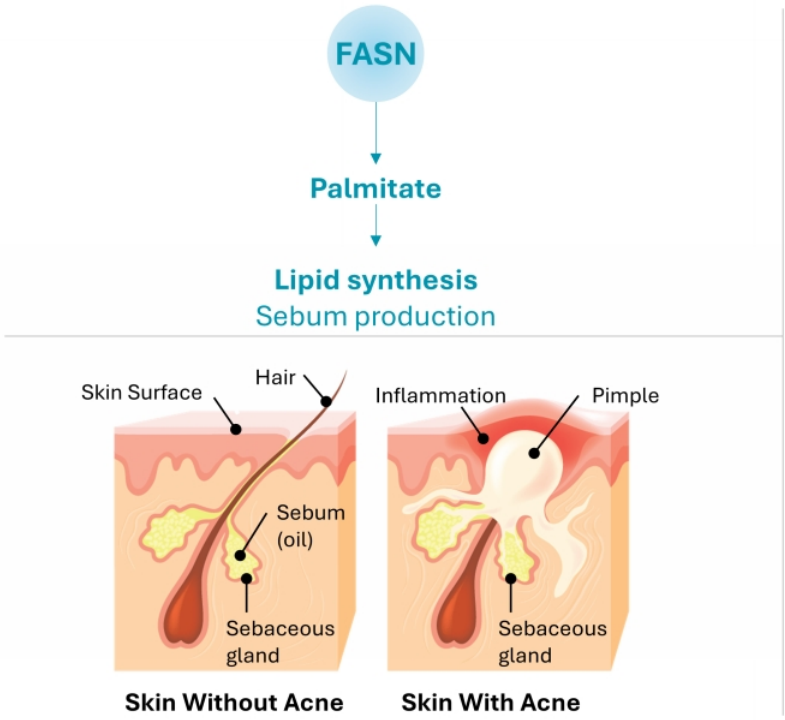
FASN in acne

Increases sebum production

Exacerbates sebum composition

SAGIMET BIOSCIENCES

DNL Pathway Plays a Role in the Pathogenesis of Acne



FASN is an attractive therapeutic targ

- Acne is associated with sebum overprodu sebocytes in the skin
- Acne resolution is associated with reduce production
- Sebocytes rely on DNL/FASN to make seb
  - >80% of key sebum lipids such as palmi sapienic acid are produced by DNL/FASN

Ascletis Announced Positive Early Clinical Data in Acne; Phase 3 Stu

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

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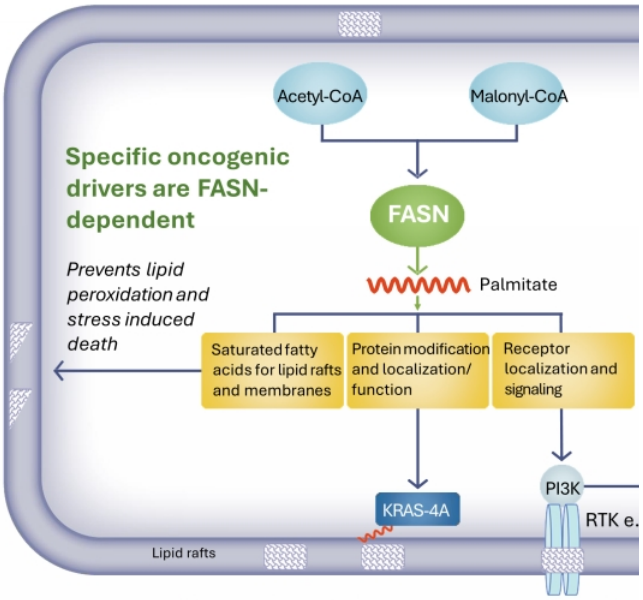
FASN Is Integral to Tumor Cell Proliferation and Survival

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 milest
GBM	**Phase 3 ongoing** In China by Ascletis, denifanstat combination with bevacizumab Positive investigator sponsored Phase 2 results*	Pre-specified interim analys 2024
Prostate	**Phase 1 ongoing** Investigator Sponsored at Weill Cornell, denifanstat combination with enzalutamide	Phase 1 results expected 4Q
HCC	**Translational work ongoing** Patient selection strategy by bioinformatics on primary samples Positive preclinical combination results**	Potential Phase 2 study of F combination with a markete ideally via collaboration with
NSCLC KRASM	**Preclinical and clinical evidence** Positive preclinical combination with KRAS inhibitor*** Encouraging monotherapy Phase 1 results with denifanstat	Potential Phase 2 study of F combination with a KRAS inh collaboration with an indust

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

FASN Inhibitor Denifanstat Offers a Unique and Validated Approach t

Unique MOA: FASN Inhibition

- As the only fat synthesis inhibitor, denifanstat directly targets the 3 key drivers of MASH – inflammation, and fibrosis
- FASN is a key enzyme in de novo lipogenesis responsible for excess liver fat in MASH
- Once daily oral administration, suitable for mono- or combination therapy

Positive FASCINATE-2 Phase 2b Data in MASH

- Met both primary endpoints in clinical trial: significant improvements in fibrosis with no v
- Improvement in more severe patients (stage F3) and demonstrated lack of progression to
- Enhanced treatment effect in patients with stable GLP therapy
- Generally well tolerated

Near Term Milestones & Cash Position

- Pivotal Phase 3 program expected to begin in 2H2024
- NASDAQ: SGMT; $193.7M cash* on hand, expected to fund current operations through 2

*Cash, cash equivalents and marketable securities as of March 31, 2024

Precision Medicine

- Tripalmitin and additional blood response markers under development as early biomarke engagement and treatment response

Strategic Collaboration with Ascletis in Acne & Cancer

- Acne Phase 3 study completion of enrollment anticipated by end 2024
- rGBM Phase 3 study interim analysis anticipated by end 2024

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