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): January 22, 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 7.01 Regulation FD Disclosure.

On January 22, 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.

The information contained in this Item 7.01 (including Exhibit 99.1) is being furnished and shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section and shall not be deemed to be incorporated by reference in any filing under the Securities Act of 1933, as amended, or the Exchange Act, except as shall be expressly set forth by specific reference in such filing.

Item 8.01 Other Events.

On January 22, 2024, the Company issued a press release announcing positive topline results from its Phase 2b FASCINATE-2 clinical trial of denifanstat in biopsy-confirmed F2/F3 non-alcoholic steatohepatitis (NASH) patients. A copy of this press release is attached as Exhibit 99.2 to this Current Report on Form 8-K and is incorporated herein by reference.

As described in the press release, representatives of the Company will host a live webcast to discuss the results from this clinical trial at 8:00 a.m. ET on January 22, 2024. A copy of the presentation to be used during the webcast is filed as Exhibit 99.3 to this Current Report on Form 8-K and is incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Document
99.1	Investor Presentation of Sagimet Biosciences Inc., dated January 22, 2024.
99.2	Press Release of Sagimet Biosciences Inc., dated January 22, 2024.
99.3	Sagimet Biosciences Announces Topline Results from Phase 2b FASCINATE-2 Clinical Trial.
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: January 22, 2024

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

Exhibit 99.1


SAGIMET
BIOSCIENCES
Targeting Metabolic Dysfunction with
Novel Therapies to Treat NASH, Acne and Cancer
January 2024

Forward Looking Statements

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

Proven Team with Development and Commercialization Experience A
Hepatology, Metabolic Disease and Oncology

Dave Happel
President & CEO

- Cognoa: President & CEO
  Chrono Therapeutics: President & CEO
  Senior Executive and Commercial roles at Horizon, Raptor, Dynavax, Chiron
- M.B.A. – Indiana State University; B.A. Chemistry – Indiana University

George Kemble
Executive Chairman

- AstraZeneca (formerly MedImmune, Aviron): SVP Research for Biologics & General Manager of California operations, VP Vaccine Research & Development for Vaccines
- Ph.D. – Stanford University, Dept of Microbiology & Immunology

Eduardo Martins
CMO

- Abbvie (formerly Allergan), Eiger BioPharmaceuticals, Gilead, Genentech, Dynavax, Intermune, SciClone
- D.Phil. – University of Oxford
  M.D. – Federal University of Rio de Janeiro, Brazil

Anthony Rimac
CFO

- Cognoa, ESCAPE Bio, Chrono Therapeutics, Aldea Pharmaceuticals, Adamas Pharmaceuticals, Aerovance
- M.B.A. – Santa Clara University; B.A. – University of California Santa Barbara

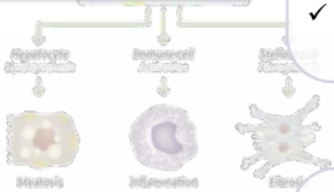
Elizabeth Rozek
General Counsel

- Cognoa, Basilea Pharmaceutica, US Department of Justice
- J.D. – University of California Berkeley
  M.A. – University of California San Diego
  B.A. – Brown University

Sagimet Investment Highlights

Critical role of FASN enzyme in NASH

- ✓ Key enzyme in de novo lipogenesis – responsible for excess liver fat in NASH
- ✓ FASN inhibition directly improves the 3 key drivers of NASH – liver fat, inflammation, fibrosis
- ✓ Differentiated MOA to treat growing underserved patient population



Denifanstat: FASN inhibitor with compelling clinical data

- ✓ FASCINATE-2 Phase 2b positive topline results
  - *NASH resolution without worsening of fibrosis with ≥2-point reduction in NAS (p=0.002)*
  - *≥2-point reduction in NAS without worsening of fibrosis (p=0.0001)*
  - *Fibrosis improvement by ≥ 1 stage with no worsening of NASH (p=0.005)*

Precision medicine is key differentiator

- ✓ Blood test confirms drug
- ✓ Predictive biomarkers ide responders
- ✓ Opportunity to personali optimize outcomes

Strong rationale for FASN in acne and cancer

Acne
- ✓ Clinical proof of concept est
  - Positive Phase 2 topline re May 2023 by Ascletis

Cancer
- ✓ Clinical proof of concept esta
  - Phase 3 rGBM trial enrollm analysis completed in Sept by Ascletis

Strong financial position

- ✓ Upsized IPO completed in Jul million of gross proceeds
- ✓ Cash and equivalents expecte operations through into the fi

Development Pipeline: Indications and Clinical Milestones

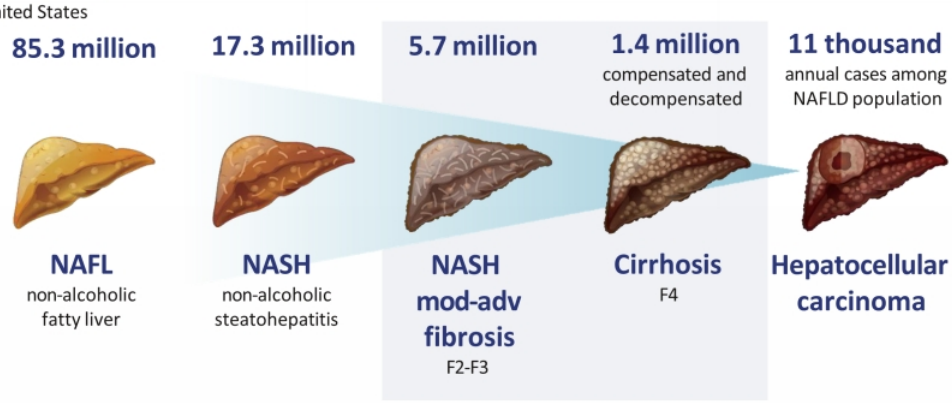
Therapeutic Area	Indication	Stage of Development				Expected Milest
		Preclinical	Phase 1	Phase 2	Phase 3	
Metabolic disease	NASH - F2/F3	TVB-2640				• Phase 2b successfu
		TVB-2640				• Phase 1 hepatic im 1Q 2024
Dermatology	Acne	TVB-3567				• IND 1H 2024 filing
		TVB-2640 (ASC40) 歌礼 ascletis				• Phase 3 clinical stu
Oncology	Solid tumors					• Patient selection a dependent tumor t
	Recurrent glioblastoma (GBM)	TVB-2640 (ASC40) 歌礼 ascletis				• Phase 3 enrollmen achieved in 3Q 202 planned*

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

NASH: A Burgeoning Epidemic

Patients in 2016[1]

United States



Disease challenges

- No approved drugs in U.S. or Europe
- Complex disease, heterogeneous patient population
- Improving regulatory clarity, but liver biopsy still required

Drug development challenges

- Many molecules moved forward on weak mechanism and data
- Inappropriate biomarkers for mechanism that did not translate to clinical benefit
- Safety: triglyceride elevations, LDL elevations, liver injury

Denifans

- ✓ Designed for on dosing
- ✓ Rigorous and de development str
- ✓ Direct DNL inhib demonstrated in
- ✓ Improvements o across biomarke
- ✓ Phase 2b fully-e biopsy results ex
- ✓ Precision medici to improve patie

DNL = de novo lipoge

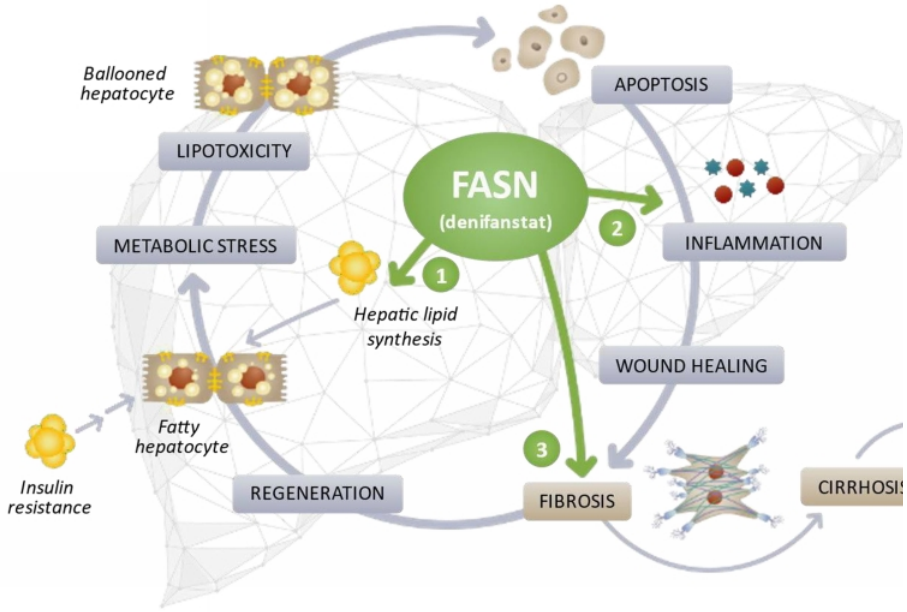
[1] *Estes, et al. 2018;* http://dx.doi.org/10.1016/j.jhep.2018.05.036

Denifanstat in NASH

Denifanstat: Differentiated Mechanism Believed to Target Key Driver

Denifanstat has independent mechanisms designed to:

1. Block **steatosis** via inhibiting de novo lipogenesis in hepatocytes
2. Reduce **inflammation** via preventing immune cell activation
3. Blunt **fibrosis** via inhibiting stellate cell activation



Adapted from Wegermann et al, Clinical Liver Disease, Vol 11, No 4, April 2018, DOI: 10.1002/cld.709

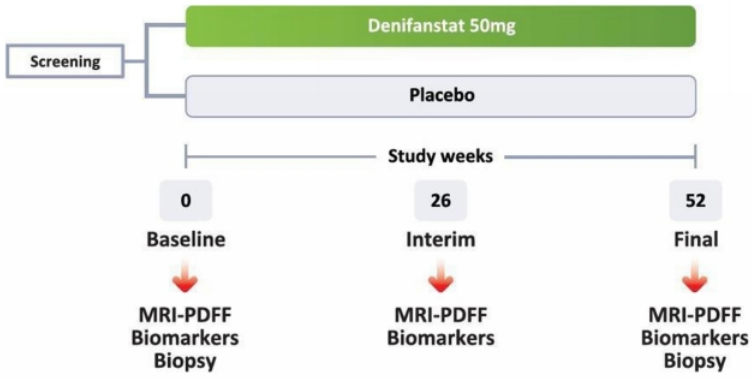
Denifanstat: Well Tolerated at 25/50mg Doses in FASCINATE-1

- No dose-related significant adverse events relative to placebo
- No serious AEs
- Majority of AEs were Grade 1; no Grade ≥3 drug-related AEs

Treatment Emergent Adverse Event (TEAE) Classification	Cohort 1			Cohort 2		Coho
	US Placebo N=31	US 25mg N=33	US 50 mg N=35	China Placebo N=9	China 50 mg N=21	US 75m N=1
Any TEAE	Gr 1: 12 (38.7%) Gr 2: 7 (22.6%)	Gr 1: 18 (54.5%) Gr 2: 7 (21.2%)	Gr 1: 12 (34.3%) Gr 2: 6 (17.1%)	Gr 1: 3 (33%) Gr 2: 2 (22%)	Gr 1: 11 (52%) Gr 2: 4 (19%) Gr 3: 2 (10%)	Gr 1: 3 (Gr 2: 6 (
TEAE leading to drug withdrawal	0	2 (6.1%)	0	0	0	4 (31%
Treatment Emergent Serious Adverse Event (SAE)	0	0	0	0	0	0
Drug related TEAE	Gr 1: 3 (9.7%) Gr 2: 1 (3.2%)	Gr 1: 10 (30.3%) Gr 2: 2 (6.1%)	Gr 1: 9 (25.7%) Gr 2: 1 (2.9%)	0	Gr 1: 9 (43%) Gr 2: 4 (19%)	Gr 1: 1 (Gr 2: 6 (

FASCINATE-2 Phase 2b Biopsy Trial Design
Measuring Histological Improvement

FASCINATE-2 Phase 2b trial design



- Biopsy confirmed F2-F3 NASH patients
- 52 weeks, 2:1 50mg or placebo, double-blind

Primary endpoints

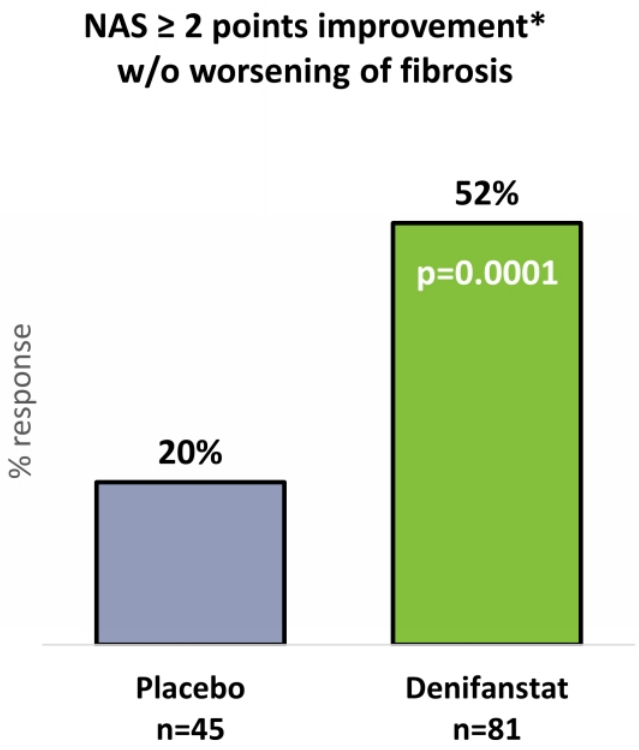
- NAS ≥2 points improvement w/o worsenin
  OR
- NASH resolution + NAS ≥2 improvement w/
  worsening of fibrosis

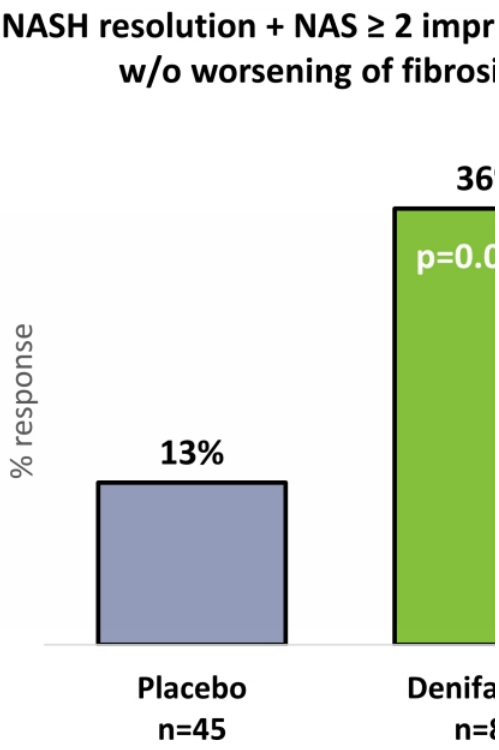
Other selected endpoints

- Improvement in liver fibrosis ≥1 stage wit
  worsening of NASH (Bx)
- Digital AI pathology
- MRI-PDFF: absolute decrease, % change f
  baseline, % pts ≥30% reduction from
  baseline (responders)

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

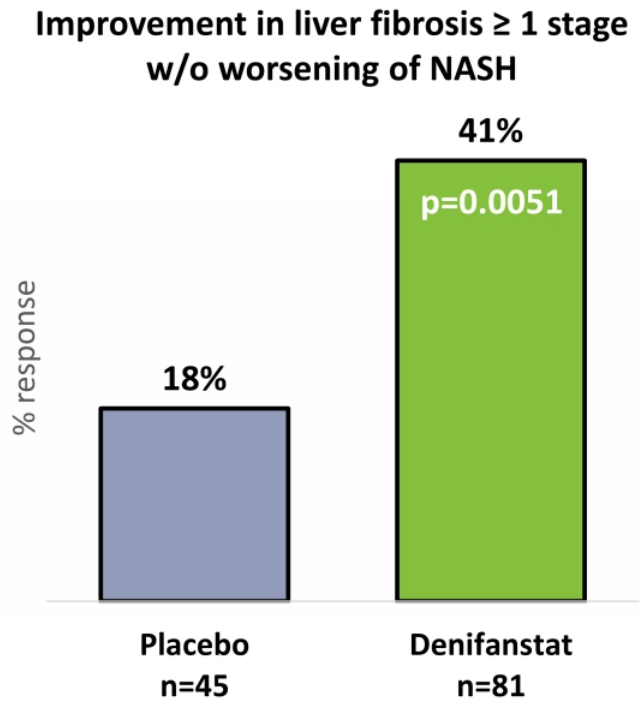
Primary Endpoints: Liver Biopsy
Denifanstat Achieved Statistical Significance

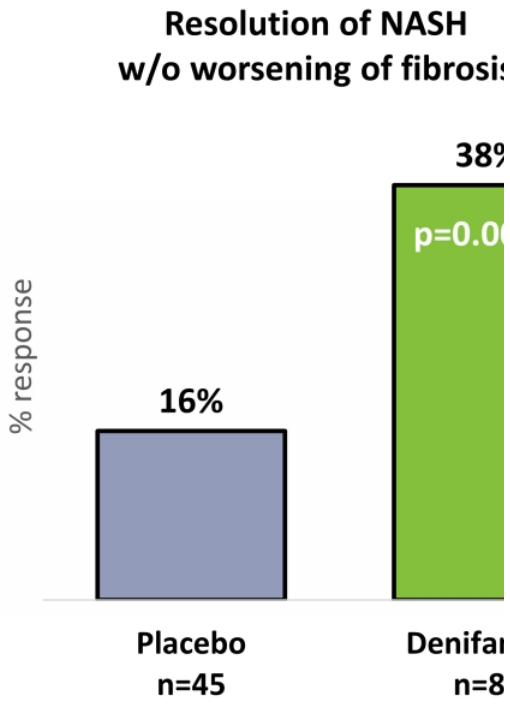




 Cochran-Mantel-Haenszel Test – One sided at the 0.05 significance level. mITT population. * ≥1-point improvement in ballooning or inflammation.

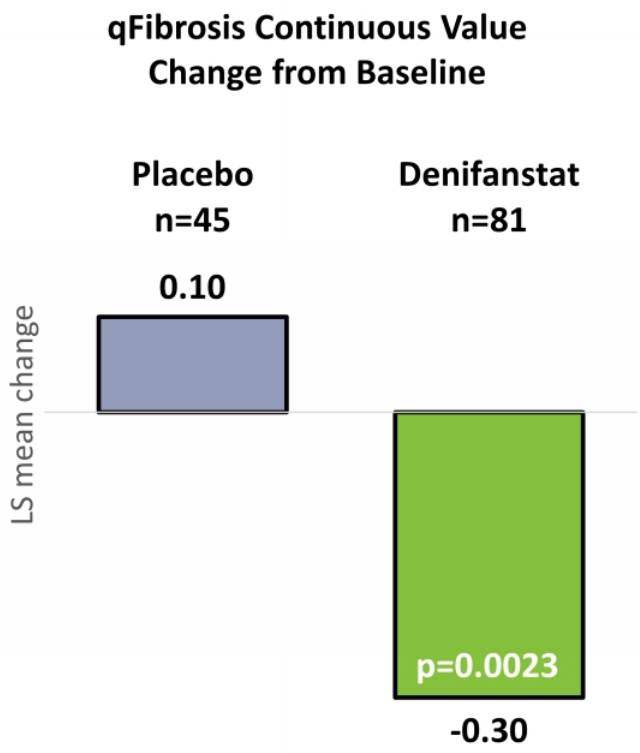
Secondary Endpoints: Liver Biopsy
Denifanstat Achieved Statistical Significance





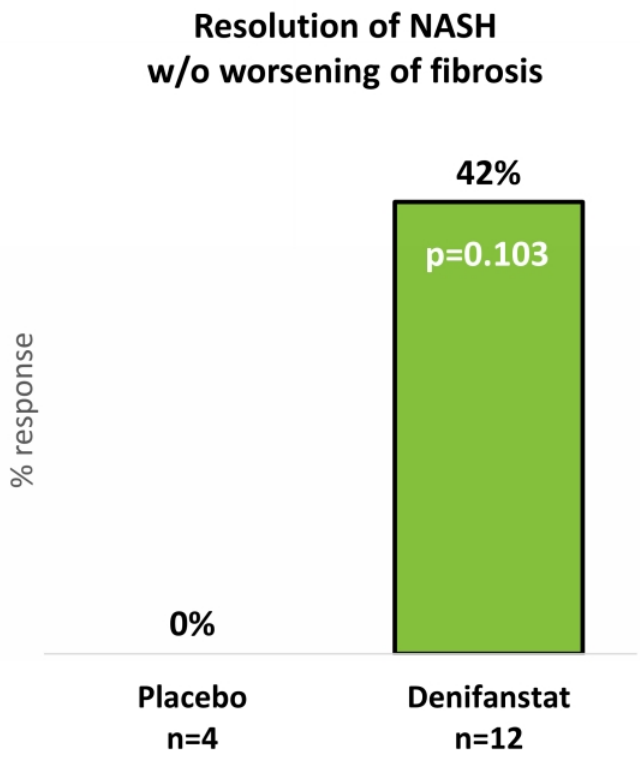
 Cochran-Mantel-Haenszel Test – One sided at the 0.05 significance level. mITT population

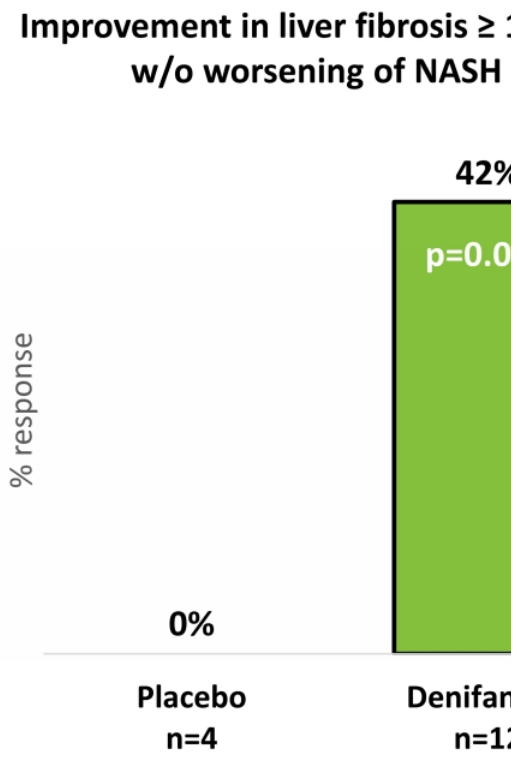
Independent Fibrosis Analysis by AI-based Digital Pathology
Supporting Evidence that Denifanstat Significantly Reduced Fibrosis



 Cochran-Mantel-Haenszel Test – One sided at the 0.05 significance level. LS means; least squares mean. HistoIndex platform. mITT population.

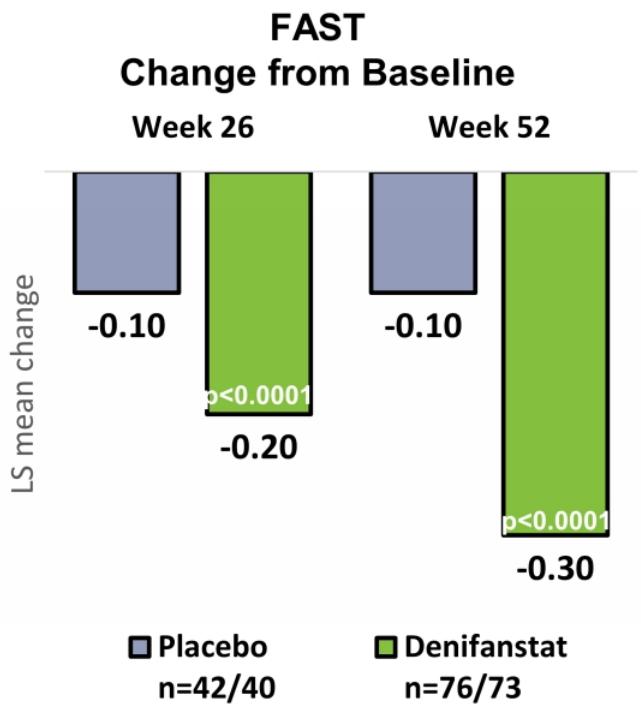
Patient Subset on Stable GLP1-RA at Baseline: Liver Biopsy
Denifanstat Improves NASH Resolution and Fibrosis

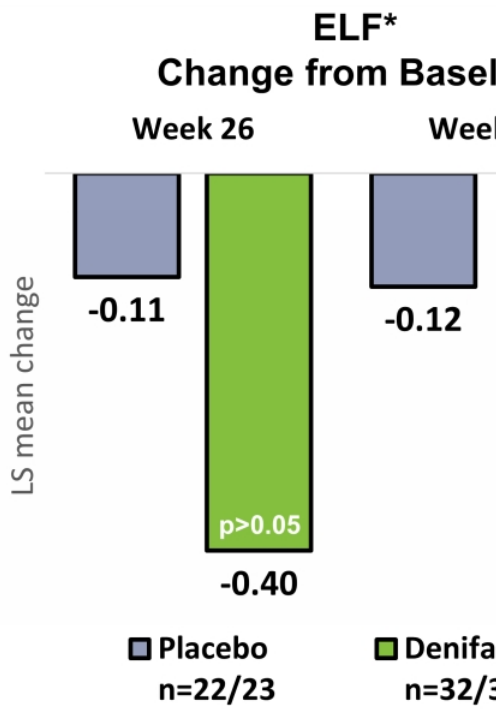




14 Cochran-Mantel-Haenszel Test – One sided at the 0.05 significance level. mITT population

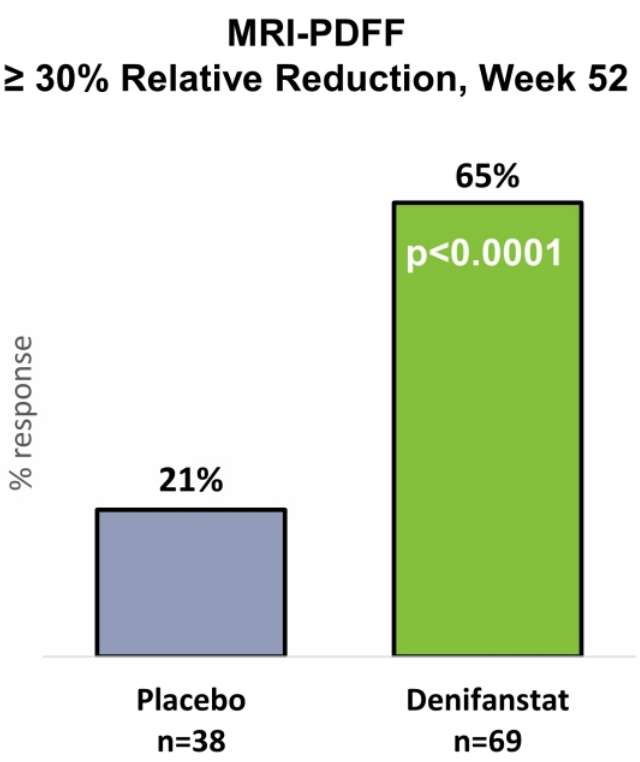
Biomarkers of Fibrosis
Denifanstat Decreased FAST Score and ELF

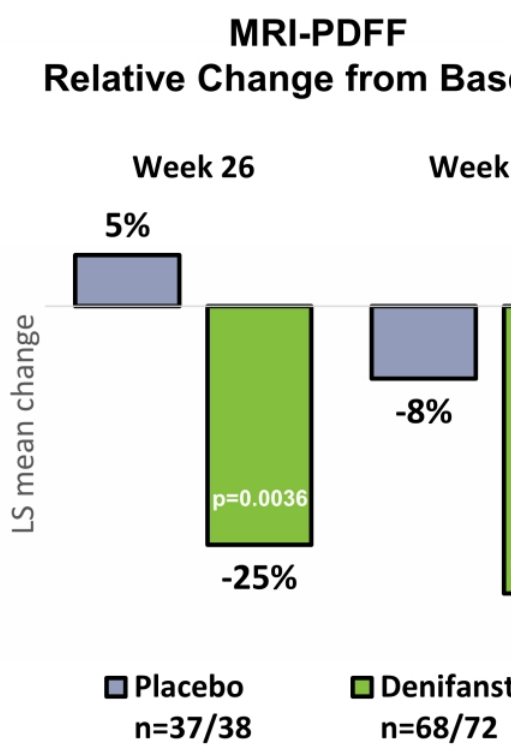




15 Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. mITT population. *Baseline ELF > 9.8 (mean).

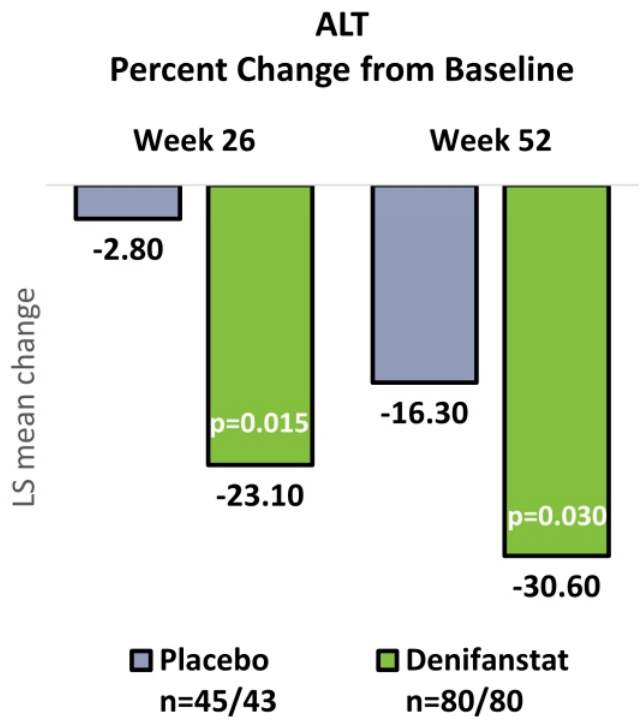
Secondary Endpoint: Liver Fat by MRI-PDFF
Denifanstat Achieved Statistical Significance

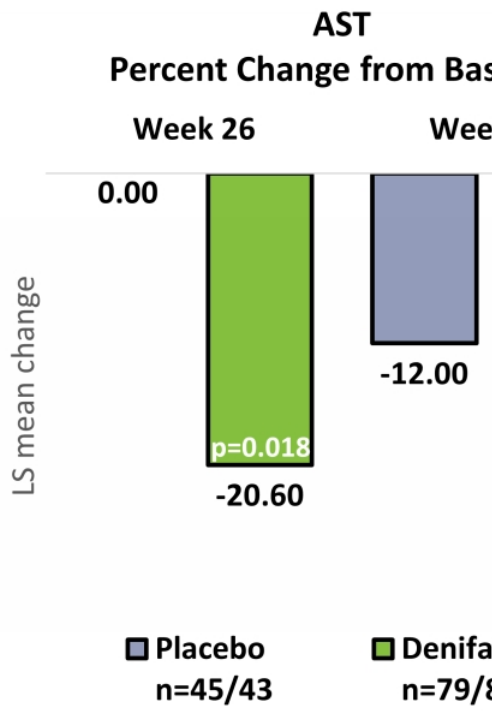




16 ≥30% reduction: Cochran-Mantel-Haenszel Test. Relative reduction: Mixed-effects Model for Repeated Measures . mITT population

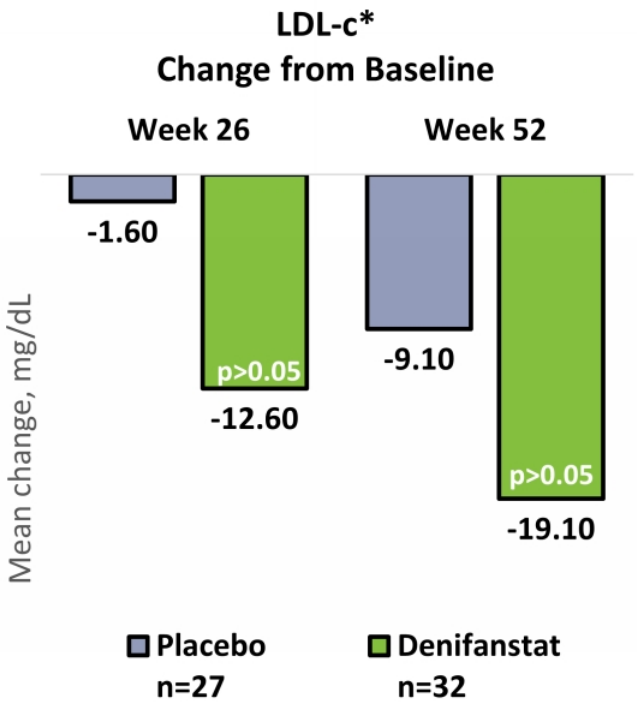
Secondary Endpoints: Liver Enzymes
Denifanstat Decreased ALT and AST Levels

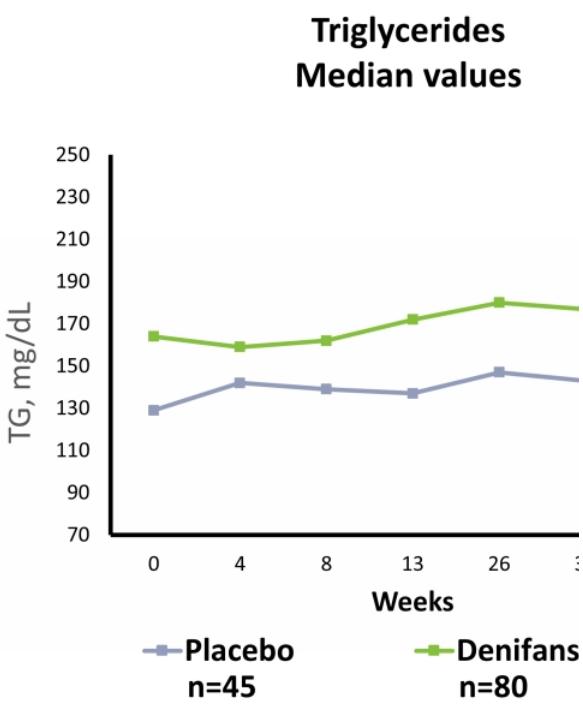




17 Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. mITT population

Cardiometabolic health
Denifanstat Decreased LDL-c Levels





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

FASCINATE-2: Safety
Denifanstat was Generally Well Tolerated

Parameter	Placebo n=56	Denifans N=11
Any TEAE (treatment emergent adverse event)	45 (80.4%)	96 (85.7
TEAE related to study drug	20 (35.7%)	51 (45.5
Most common TEAE related to study drug in ≥5% of patients by system organ class		
eye disorders	9 (16.1%)	17 (15.2
gastrointestinal disorders	5 (8.9%)	13 (11.6
skin and subcutaneous tissue disorders	4 (7.1%)	25 (22.3
TEAE leading to study drug discontinuation	3 (5.4%)	22 (19.6
TEAE with CTCAE Grade 3 (Severe) or higher*	3 (5.4%)	13 (11.6
SAE (none related to treatment)	3 (5.4%)	13 (11.6
Fatal TEAE	0	0

* No treatment-related AE was Grade 3 or higher

NASH Development Program

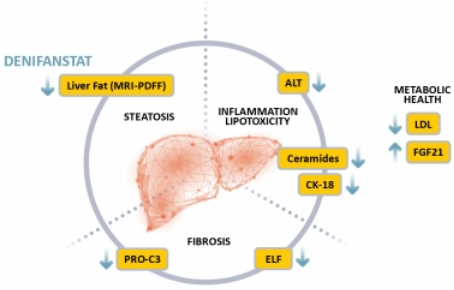
Progression from Phase 2b to Phase 3

Phase 2b – baseline Fibrosis stage

Interim cohort
F2 – 46.2%
F3 – 53.8%

Enrollment completed Sep 2022

Phase 2b – 26 weeks Non-invasive interim



Interim results released Nov 2022

Phase 2b – 52 weeks Histology

Primary endpoints
- NAS ≥2 improvement
  w/o worsening of fibrosis; or
  NASH resolution +
  NAS ≥2 improvement
  w/o worsening of fibrosis

Secondary endpoints
- Fibrosis ≥1 stage improvement
  w/o worsening of NASH
- Digital AI pathology

Topline data released Jan 2024

Ph Fibrosis en

Will use results ir patholog design a Phase 3

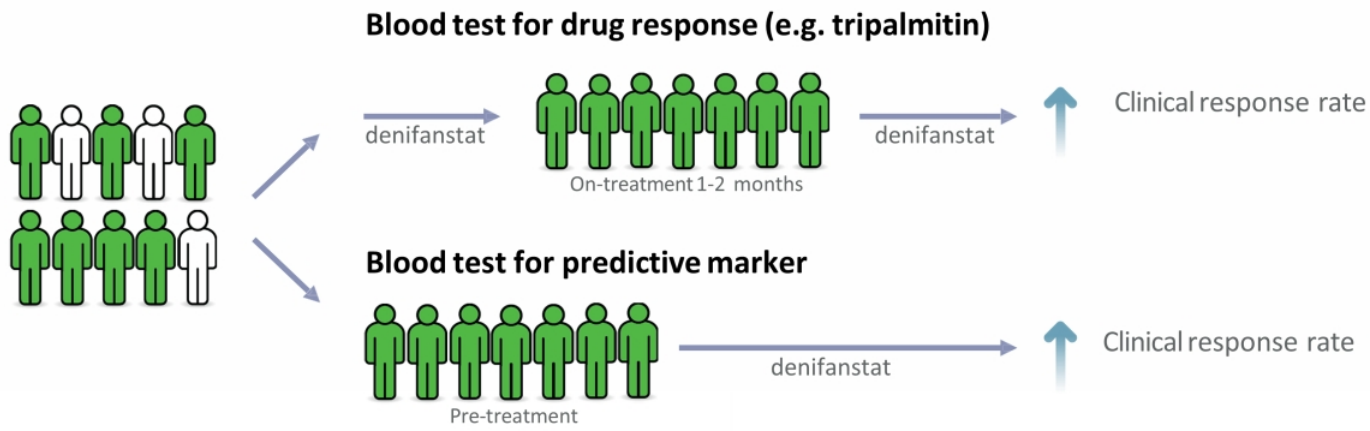
Startup act

We Believe Denifanstat is Differentiated in the Evolving NASH Landsc

Mechanism	FASN inhibitors	THRß Agonists	FGF-21	GLP-1 agonists	PPAR agonists	ACC inhibitors
Category	**DNL pathway**	Nuclear receptor	Growth factor	GLP-1	Nuclear receptor	DNL pathway
Route	**Oral**	Oral			Oral	Oral
Status	**Phase 2 complete**	Phase 3 complete	Phase 2 complete	Phase 2 complete	Phase 3 ongoing	Phase 2 complete
Challenges	• **Pivotal Phase 3 clinical study**	• Selectivity for beta isoform critical to avoid potential heart and bone safety issues	• Injectable • Nausea and diarrhea • Potential neutralizing antibodies • Higher expected COGS	• GI side effects including nausea • Lack of fibrosis improvement to date	• Weight gain, edema, GI side effects, anemia	• Combinations only • MOA causes triglyceride increases • Lack of fibrosis improvement as monotherapy

Precision Medicine: Blood Tests May Lead to Improved Patient Outc

- NASH is a multi-faceted disease and patients may benefit from being matched with optimal trea
- Two approaches using blood tests are undergoing further evaluation
  - Drug response: 1-2 months after taking drug, tripalmitin identifies patients responding to drug treatment
  - 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%.*

Strong Monotherapy Opportunity for Denifanstat in NASH

Expansion as backbone of combinations

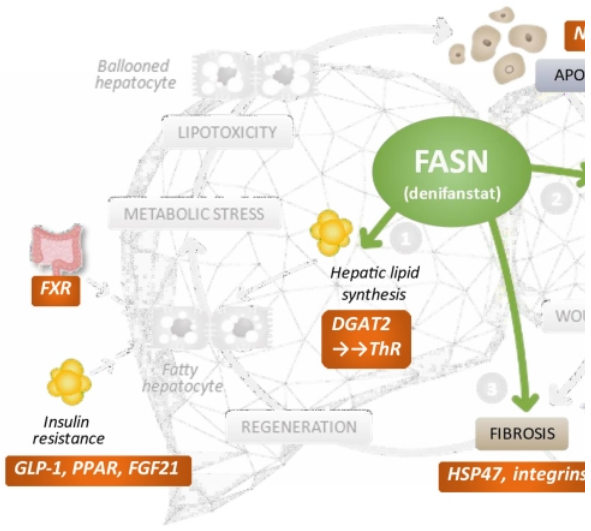
Denifanstat data support success as first line monotherapy

- ✓ Oral, once-daily tablet ideal for chronic administration
  - Tablets generally more affordable than complex biologics
- ✓ Potential to treat broad patient population
  - Including those with thyroid challenges
- ✓ Novel mechanism that acts directly upon liver
- ✓ Encouraging safety profile to date

Broaden market opportunity through combinations with denifanstat as backbone

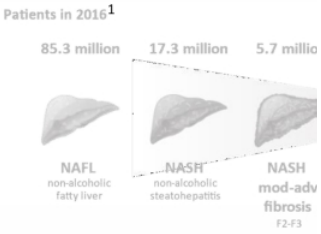
- Denifanstat's potential
  - ✓ Complementary to other mechanisms
  - ✓ Potential for fixed dose combinations with other oral medications
- ✓ Preclinical combination studies ongoing
  - NASH agents: anti-fibrotic, other metabolic agents
  - Co-morbidities: diabetes and other cardiovascular agents

Illustrative potential combo mech



Additional Expansion Opportunities in NASH

- **Compensated cirrhotic patients (NASH F4)**
  - Denifanstat directly targets stellate cells
  - Hepatocytes continue to be functional, and patients frequently have increased liver fat
  - Next steps
    - Characterize PK profile in patients with impaired hepatic function – Phase 1 results in 1Q 24
    - Positive impact on fibrosis in FASCINATE-2
    - Phase 2b/3 trial in NASH-F4

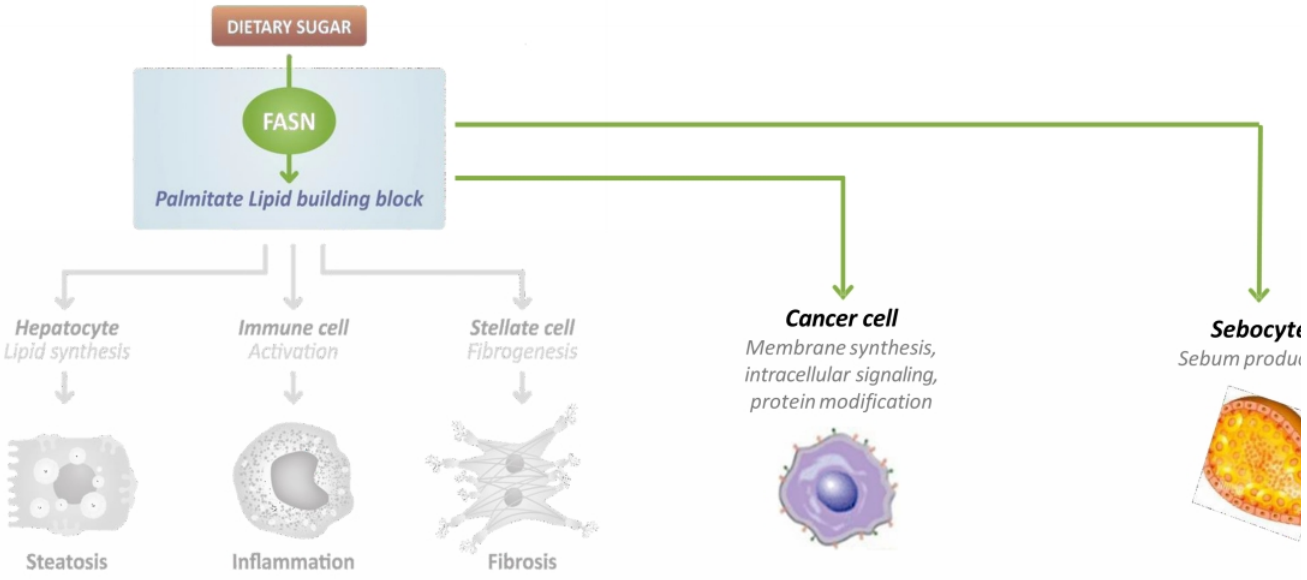


- **Pediatric NASH**
  - 23% of children with NAFLD have NASH at the time of diagnosis
  - Next steps
    - Compile safety data across all denifanstat studies in young adults including FASCINATE-2
    - Nonclinical toxicology study in juvenile animals – plan to initiate in 2024
    - Phase 2 trial in pediatric NASH

[1] *Estes, et al. 2018;* *http://dx.doi.org/10.1016/j.jhep.2018.05.036*

Other Indications

FASN Hyper-Activity Plays a Key Role in Multiple Diseases Beyond NA



FASN in NASH

1. Drives steatosis
2. Activates pro-inflammatory cells
3. Activates stellate cells leading to fibrosis

FASN in cancer

1. Supports tumor survival
2. Enables tumor proliferation
3. Establishes resistance to drugs

FASN in ac

1. Sebum
2. Sebum

DNL Pathway Plays a Role in the Pathogenesis of Acne

- Acne is associated with excess sebum production by sebocyte cells in the skin
- Acne resolution is associated with reduced sebum production
- Sebocytes upregulate and rely on DNL/FASN to make sebum
  - >80% of key sebum lipids such as palmitate and sapienic acid produced by DNL/FASN

=> FASN inhibition has potential therapeutic application

Phase 1 – sebum analysis
by Sagimet

- Denifanstat inhibited lipogenesis in skin
- Dose-dependent
- Proof of mechanism

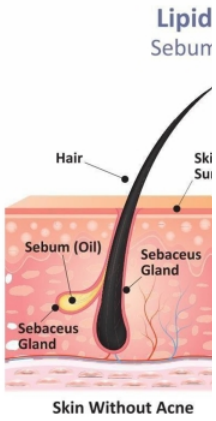
Phase 2 – acne
by Ascletis in China

歌礼 ascletis

- 12-week trial in moderate to severe acne
- 179 pts randomized to 25/50/75 mg denifanstat and placebo
- Endpoints: % change from baseline in lesion count and/or
  IGA score decreased by ≥ 2
  - Phase 3 study commenced in Q4 2023

Positive topline results announced May 2023

- Met primary and secondary endpoints
- Well-tolerated
- IND 1H 2024 filing planned



Skin Without Acne

Esler et al., Sci. Transl. Med.11, eaau8465 (2019). Figure adapted from kidshealth.org

FASN is Integral to Tumor Cell Proliferation and Survival

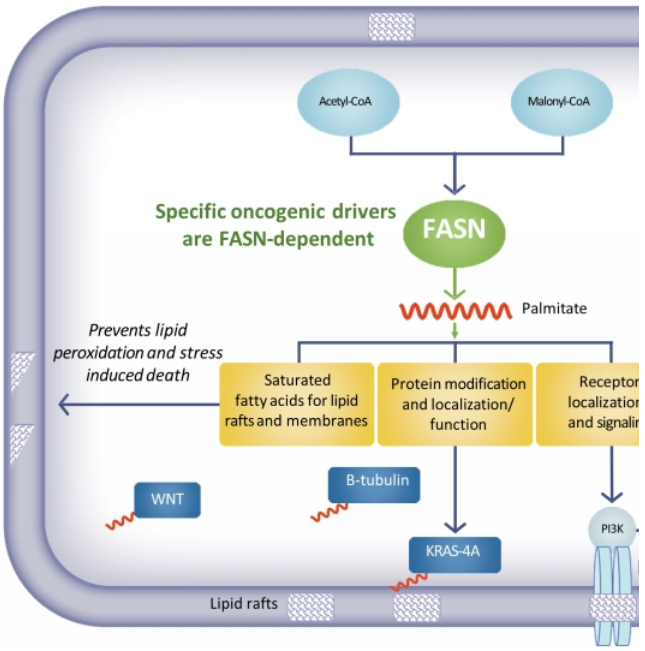
Reprogramed metabolism is one of the hallmarks of cancer

FASN-dependence

- Certain cancers are dependent on DNL/FASN for proliferation especially downstream of driver oncogenes
  - eg. KRASM in non-small cell lung cancer (NSCLC)
- Strategy –> exploit this vulnerability using FASN inhibition in the combination setting to cause death

Completed Phase 1 provides foundation

- 136 patients received denifanstat
- Heavily pretreated Phase 1 population
- 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 compensat
de novo synthesized palmitate

FASN-Dependent Tumor Types Identified that Meet Core Crite

Program focused on 4 selected tumor types

Core criteria

- ✓ FASN-dependent mechanism
- ✓ Preclinical or clinical POC shown
- ✓ Unmet clinical need
- ✓ Tractable clinical path including patient selection

Tumor type	Status	Next milest
NSCLC KRASM	**Preclinical ongoing** • Combination with KRAS inhibitor in mouse models ✓ Encouraging Phase 1 results with denifanstat	If positive, favo with a KRASM
HCC FASN-dependent	**Translational ongoing** • Patient selection bioinformatics ✓ Positive preclinical results	If patient selec would sponsor
Prostate FASN-dependent	**Phase 1 pending start** • Investigator Sponsored Trial at Weill Cornell, in combination with enzalutamide ✓ Positive preclinical results	Phase 1 result decision by Sag
GBM	**Phase 3 ongoing** • By Ascletis in China, in combination with bevacizumab ✓ Positive Phase 2 investigator sponsored trial results	Phase 3 result decision by Sag

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

Strong Financial Position and Intellectual Property Portfolio

Financial highlights Nasdaq: SGMT

- ✓ Upsized IPO completed in July 2023 raised $96.4 million of gross proceeds
- ✓ Cash and equivalents expected to fund current operations into the first quarter of 2025

Strong patent estate

- ✓ Composition of matter for denifanstat: 2032
- ✓ Issued in all key commercial territories
- ✓ Opportunities to lengthen exclusivity via Hatch-Waxman and synthesis/formulation applications

Development Pipeline: Indications and Clinical Milestones

Therapeutic Area	Indication	Stage of Development				Expected Milest
		Preclinical	Phase 1	Phase 2	Phase 3	
Metabolic disease	NASH - F2/F3	TVB-2640				• Phase 2b successfu
		TVB-2640				• Phase 1 hepatic im 1Q 2024
Dermatology	Acne	TVB-3567				• IND 1H 2024 filing
		TVB-2640 (ASC40) 歌礼 ascletis				• Phase 3 clinical stu
Oncology	Solid tumors					• Patient selection a dependent tumor t
	Recurrent glioblastoma (GBM)	TVB-2640 (ASC40) 歌礼 ascletis				• Phase 3 enrollmen achieved in 3Q 202 planned*

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

Exhibit 99.2

Sagimet Biosciences Announces Positive Topline Results from Phase 2b FASCINATE-2 Clinical Trial of Denifanstat in Biopsy-Confirmed F2/F3 NASH

Denifanstat achieved statistically significant results on primary and multiple secondary endpoints in a 52-week clinical trial of 168 NASH patients with stage 2 or 3 fibrosis

- *Primary efficacy endpoints:*
  - *NASH resolution without worsening of fibrosis with ≥2-point reduction in NAS (NAFLD Activity Score) in 36% of denifanstat-treated patients vs 13% with placebo* ***(p=0.0022)***
  - *≥2-point reduction in NAS (with ≥1-point improvement in ballooning or inflammation) and without worsening of fibrosis in 52% of denifanstat-treated patients vs 20% with placebo* ***(p=0.0001)***
- *Multiple secondary endpoints:*
  - *Fibrosis improvement by ≥ 1 stage with no worsening of NASH in 41% of denifanstat-treated patients vs 18% with placebo* ***(p=0.0051)***
  - *NASH resolution with no worsening of fibrosis in 38% of denifanstat-treated patients vs 16% with placebo* ***(p=0.0021)***
  - *MRI-PDFF decline from baseline ≥30% (responders) in 65% of denifanstat-treated patients vs 21% with placebo* ***(p<0.0001)***

Statistically significant improvements in additional markers of liver health, including artificial intelligence (AI) digital pathology-based fibrosis assessment, FAST Score, and ALT, and numerical improvements in LDL

Denifanstat was generally well-tolerated

Management to host live webcast at 8:00 a.m. ET on Monday, January 22, 2024

San Mateo, Calif., January 22, 2024 – Sagimet Biosciences Inc. (Sagimet, Nasdaq: SGMT), a clinical-stage biopharmaceutical company developing novel fatty acid synthase (FASN) inhibitors designed to target dysfunctional metabolic and fibrotic pathways, today announced positive topline results from its FASCINATE-2 Phase 2b clinical trial of denifanstat versus placebo in biopsy-confirmed non-alcoholic steatohepatitis (NASH) patients with stage 2 or stage 3 fibrosis (F2/F3) at week 52. In this trial, denifanstat, an oral, selective FASN inhibitor, showed statistically significant improvements relative to placebo on both of the primary endpoints of NASH resolution without worsening of fibrosis with ≥2-point reduction in NAS, and ≥2-point reduction in NAS without worsening of fibrosis. Denifanstat-treated patients also showed statistically significant fibrosis improvement by ≥ 1 stage with no worsening of NASH, and a greater proportion of MRI-derived proton density fat fraction (MRI-PDFF) ≥30% responders relative to placebo.

“Denifanstat is designed to reduce the three main drivers of NASH, including fat accumulation, inflammation, and fibrosis, both independently and in parallel. The week 52 biopsy results showed that denifanstat achieved statistical superiority over placebo in reduction of fibrosis, via two independent processes of traditional histopathology and AI digital pathology,” said Dave Happel, Chief Executive Officer of Sagimet. “Sagimet is committed to creating novel approaches to target dysfunctional metabolic pathways, and we believe these positive results represent a major advancement in that endeavor. Our next step will be holding an End-of-Phase 2 meeting with the FDA and starting our Phase 3 program for development of denifanstat in NASH with related fibrosis, which we anticipate to begin in the second half of 2024.”

“The over-activity of fatty acid synthase and increased de-novo lipogenesis or DNL plays a critical role in the development of NASH and its progression to cirrhosis,” commented Rohit Loomba, M.D., M.H.Sc., Professor of Medicine, Chief, Division of Gastroenterology and Hepatology, and Director, MASLD Research Center, University of California San Diego, who serves as a scientific advisor for Sagimet on its ongoing development of denifanstat. “Denifanstat is the only FASN inhibitor currently in clinical development for the treatment of NASH with related fibrosis. These data show that blocking fatty acid synthesis in the liver and DNL is a critical approach for NASH resolution and improvements in fibrosis. These results support denifanstat’s mechanism of action and the impact of addressing these multiple pathways simultaneously. Moreover, the safety profile supports the further development of denifanstat in NASH patients.”

Statistical Significance Achieved in Primary Endpoints and Improvements Across Other Endpoints at Week 52 of Denifanstat Treatment

	Denifanstat 50 mg (n=81)	Placebo (n=45)	P-value vs placebo
Primary Endpoints			
NASH resolution without worsening of fibrosis with ≥2-point reduction in NAS	36%	13%	0.0022
≥ 2-point decrease in NAS without worsening of fibrosis	52%	20%	0.0001
Other Endpoints			
Improvement of fibrosis by ≥ 1 stage with no worsening of NASH	41%	18%	0.0051
NASH resolution with no worsening of fibrosis	38%	16%	0.0021
AI digital pathology (qFibrosis)*	-0.3	0.1	0.0023
ALT % change from baseline	-30.5%	-17.2%	0.0300
MRI-PDFF responder rate**	65%	21%	<0.0001
FibroScan AST (FAST) score	-0.3	-0.1	<0.0001
LDL cholesterol (mg/dL)***	-19.1	-9.1	--

Modified intent-to-treat population (mITT) includes all patients with paired biopsies; includes Secondary Endpoints for which analysis has been completed as of the date of this press release.

**Artificial Intelligence (AI) digital pathology assessed by second harmonic generation (SHG, HistoIndex)*

*** MRI-PDFF responders are patients with ≥8% liver fat content at baseline who achieve a ≥30% relative reduction of liver fat at the end of treatment*

**** In study subjects with baseline LDL-C greater than 100 mg/dL; n=32 and n=27 denifanstat and placebo patients, respectively*

Safety and Tolerability

As in prior studies, no treatment-related serious adverse events (SAEs) were observed, and the majority of adverse events (AEs) were mild to moderate in nature (Grades 1 and 2). There were no Grade ≥3 treatment-related AEs. The most common treatment-related AEs by system organ class (observed in ≥5% of patients in the study) were eye disorders (denifanstat 15.2%, placebo 16.1%), gastrointestinal disorders (denifanstat 11.6%, placebo 8.9%), and skin and subcutaneous tissue disorders (denifanstat 22.3%, placebo 7.1%). The incidence of treatment emergent adverse events (TEAEs) leading to treatment discontinuation was 19.6% in the denifanstat group compared to 5.4% in placebo.

Webcast Information

Management will host a live webcast at 8:00 a.m. ET on Monday, January 22, 2024 to discuss the data; participants will have the opportunity to participate in a chat-based Q&A session. The webcast will be available here and in the Events & Presentation section of Sagimet's website at www.sagimet.com, with an archived replay available for approximately 90 days following the event.

About Phase 2b FASCINATE-2 Clinical Trial

The Phase 2b FASCINATE-2 clinical trial was a 52-week randomized, double-blind, placebo-controlled trial that evaluated the safety and histological impact of denifanstat compared to placebo in 168 biopsy-confirmed NASH patients with moderate-to-severe fibrosis (stage F2 or F3) with NAS ≥4.

Patients were randomized 2:1 to receive 50 mg denifanstat or placebo, taken orally once daily. An end-of-trial biopsy was assessed by a central pathologist for histological endpoints. Liver biopsies were also analyzed using AI-based digital pathology.

About Sagimet Biosciences

Sagimet is a clinical-stage biopharmaceutical company developing novel fatty acid synthase (FASN) inhibitors that are designed to target dysfunctional metabolic pathways in diseases resulting from the overproduction of the fatty acid, palmitate. Sagimet's lead drug candidate, denifanstat, is an oral, once-daily pill and selective FASN inhibitor in development for the treatment of NASH, for which there are no treatments currently approved in the United States or Europe. FASCINATE-2, a Phase 2b clinical trial of denifanstat in NASH with liver biopsy-based primary endpoints, was successfully completed with positive results. For additional information about Sagimet, please visit www.sagimet.com.

Forward-Looking Statements

This press release 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 press release, other than statements of historical facts or statements that relate to present facts or current conditions, including but not limited to, statements regarding: the presentation of data from clinical trials, Sagimet's clinical development plans and related anticipated development milestones, Sagimet's cash and financial resources and expected cash runway. These statements involve known and unknown risks, uncertainties and other important factors that may cause Sagimet's 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, these statements can be identified by terms such as "may," "might," "will," "should," "expect," "plan," "aim," "seek," "anticipate," "could," "intend," "target," "project," "contemplate," "believe," "estimate," "predict," "forecast," "potential" or "continue" or the negative of these terms or other similar expressions.

The forward-looking statements in this press release are only predictions. Sagimet has based these forward-looking statements largely on its current expectations and projections about future events and financial trends that Sagimet believes may affect its business, financial condition and results of operations. These forward-looking statements speak only as of the date of this press release 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 Sagimet's control, including, among others: the risks that top-line clinical trial results may not be predictive of, and may differ from, final clinical data and later-stage clinical trials; 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; the clinical development and therapeutic potential of denifanstat or any other drug candidates Sagimet may develop; Sagimet's ability to advance drug candidates into and successfully complete clinical trials; Sagimet's relationship with Ascletis, and the success of its development efforts for denifanstat; the accuracy of Sagimet's estimates regarding its capital requirements; and Sagimet's 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 Sagimet's 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 these forward-looking statements may not be achieved or occur, and actual results could differ materially from those projected in the forward-looking statements. Moreover, Sagimet operates in a dynamic industry and economy. New risk factors and uncertainties may emerge from time to time, and it is not possible for management to predict all risk factors and uncertainties that Sagimet may face. Except as required by applicable law, Sagimet does not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise.

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SAGIMET
BIOSCIENCES
Announces Topline Results from Phase 2b FASCINATE-2 Clinical Trial
Webcast on Monday, January 22, 2024 at 8:00 a.m. ET / 5:00 a.m. PT

Proven Team with Development and Commercialization Experience A
Hepatology, Metabolic Disease and Oncology

Dave Happel
President & CEO

- Cognoa: President & CEO
  Chrono Therapeutics: President & CEO
  Senior Executive and Commercial roles at Horizon, Raptor, Dynavax, Chiron
- M.B.A. – Indiana State University; B.A. Chemistry – Indiana University

George Kemble
Executive Chairman

- AstraZeneca (formerly MedImmune, Aviron): SVP Research for Biologics & General Manager of California operations, VP Vaccine Research & Development for Vaccines
- Ph.D. – Stanford University, Dept of Microbiology & Immunology

Eduardo Martins
CMO

- Abbvie (formerly Allergan), Eiger BioPharmaceuticals, Gilead, Genentech, Dynavax, Intermune, SciClone
- D.Phil. – University of Oxford
  M.D. – Federal University of Rio de Janeiro, Brazil

Anthony Rimac
CFO

- Cognoa, ESCAPE Bio, Chrono Therapeutics, Aldea Pharmaceuticals, Adamas Pharmaceuticals, Aerovance
- M.B.A. – Santa Clara University; B.A. – University of California Santa Barbara

Elizabeth Rozek
General Counsel

- Cognoa, Basilea Pharmaceutica, US Department of Justice
- J.D. – University of California Berkeley
  M.A. – University of California San Diego
  B.A. – Brown University

Forward Looking Statements

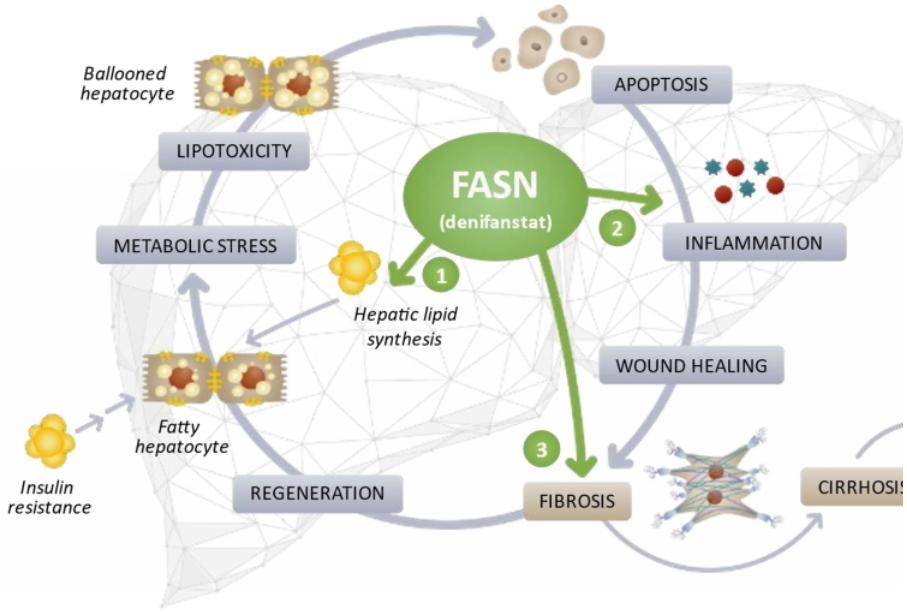
This presentation contains forward-looking statements within the meaning of, and made pursuant to the safe harb of, The Private Securities Litigation Reform Act of 1995. All statements contained in this document, other than sta historical facts or statements that relate to present facts or current conditions,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

Denifanstat: Differentiated Mechanism Believed to Target Key Drivers

Denifanstat has independent mechanisms designed to:

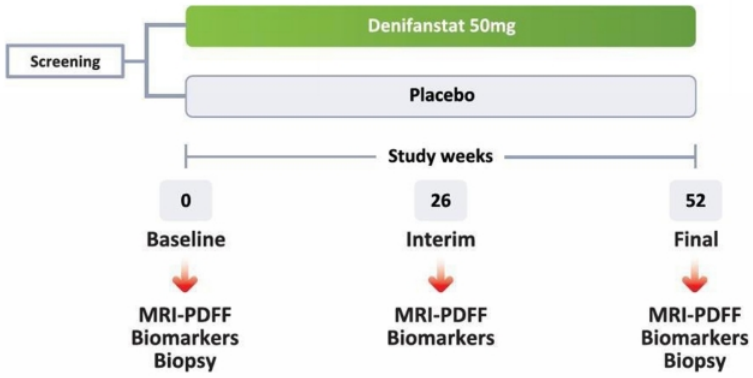
1. Block **steatosis** via inhibiting de novo lipogenesis in hepatocytes
2. Reduce **inflammation** via preventing immune cell activation
3. Blunt **fibrosis** via inhibiting stellate cell activation



Adapted from Wegermann et al, Clinical Liver Disease, Vol 11, No 4, April 2018, DOI: 10.1002/cld.709

FASCINATE-2 Phase 2b Biopsy Trial Design
Measuring Histological Improvement

FASCINATE-2 Phase 2b trial design



- Biopsy confirmed F2-F3 NASH patients
- 52 weeks, 2:1 50mg or placebo, double-blind

Primary endpoints

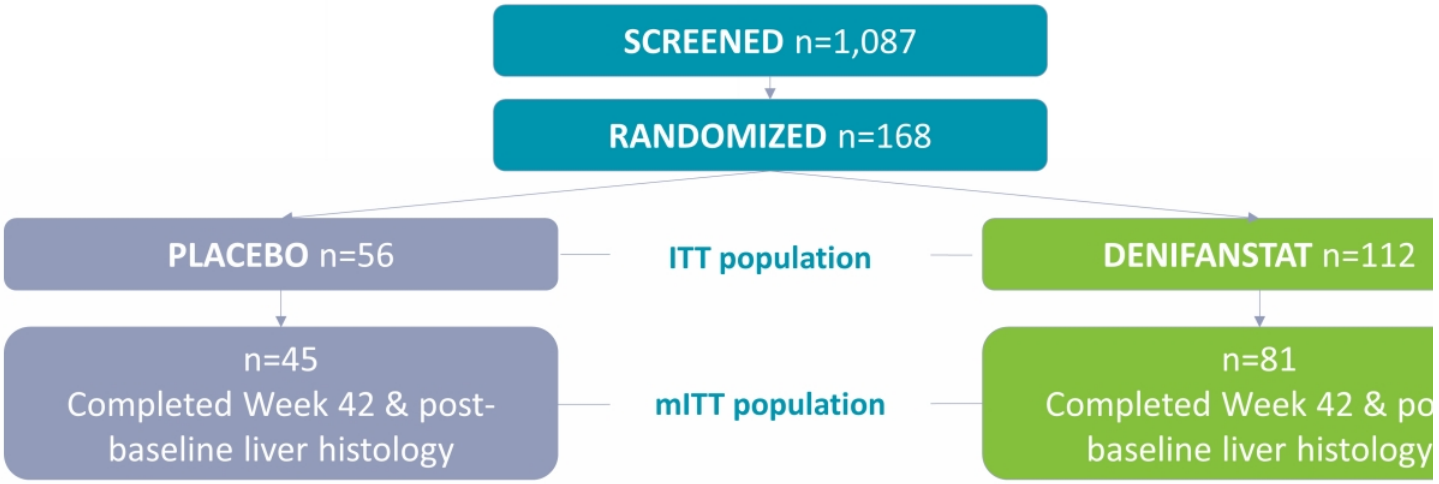
- NAS ≥2 points improvement w/o worsenin
  OR
- NASH resolution + NAS ≥2 improvement w/
  worsening of fibrosis

Other selected endpoints

- Improvement in liver fibrosis ≥1 stage wit
  worsening of NASH (Bx)
- Digital AI pathology
- MRI-PDFF: absolute decrease, % change f
  baseline, % pts ≥30% reduction from
  baseline (responders)

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

FASCINATE-2: Patient Disposition



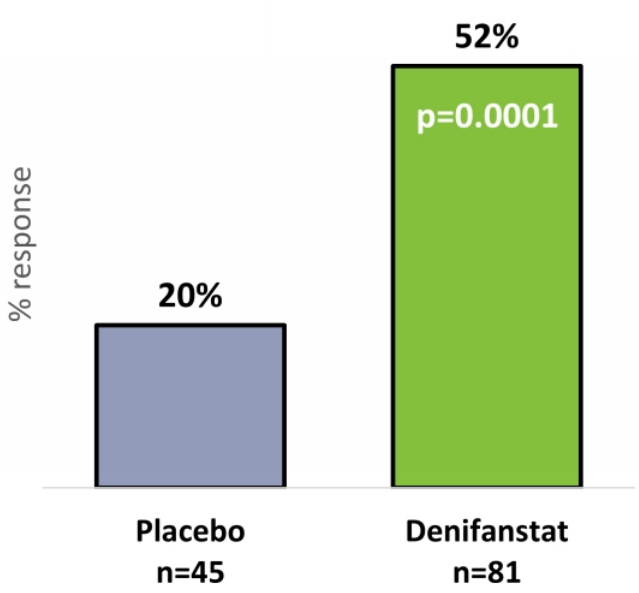
FASCINATE-2 Baseline Characteristics
Typical F2/F3 NASH Population

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)

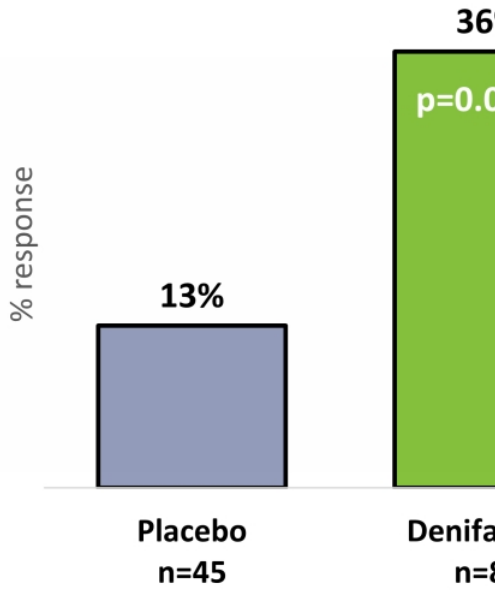
Modified intent-to-treat population (mITT) includes all patients with paired biopsies. Data are mean (SD) or n (%)

Primary Endpoints: Liver Biopsy
Denifanstat Achieved Statistical Significance

NAS ≥ 2 points improvement*
w/o worsening of fibrosis

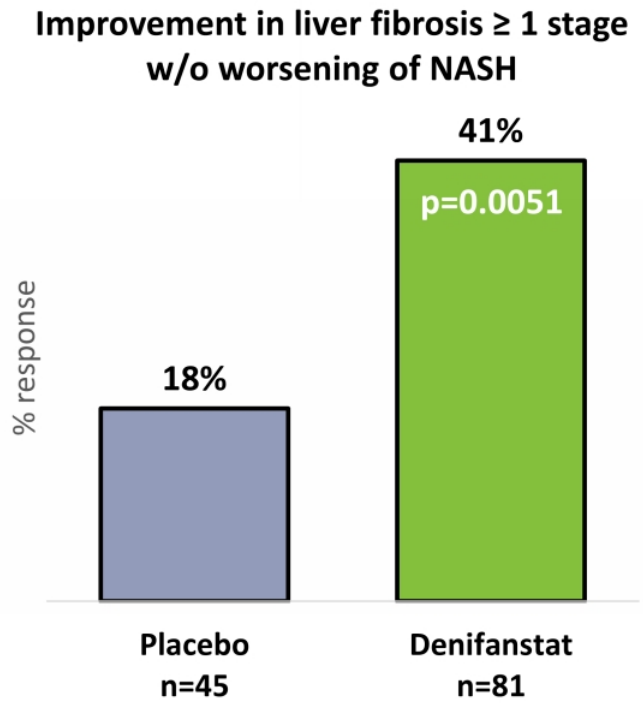


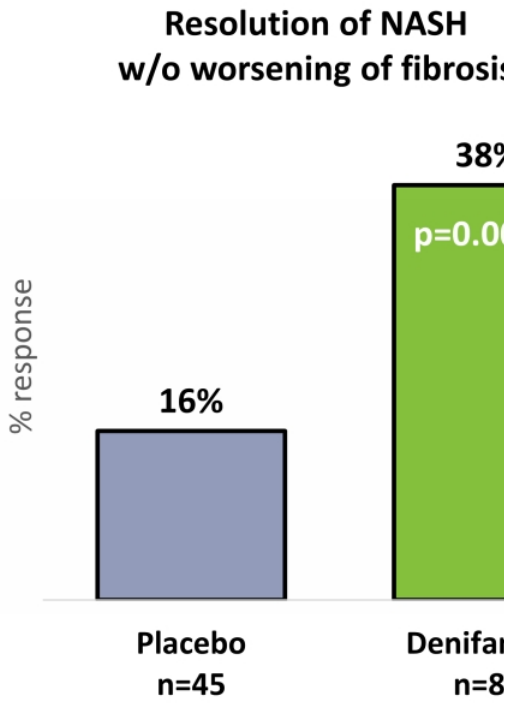
NASH resolution + NAS ≥ 2 impr
w/o worsening of fibrosi



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

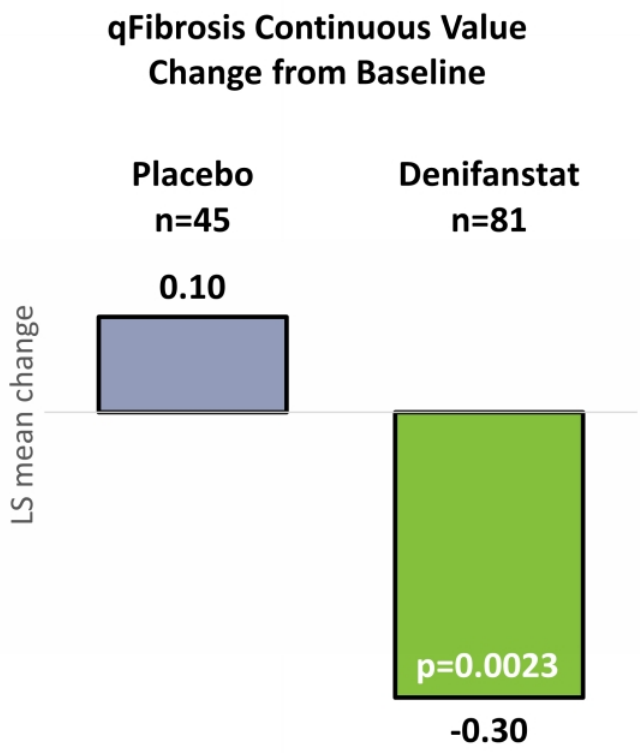
Secondary Endpoints: Liver Biopsy
Denifanstat Achieved Statistical Significance





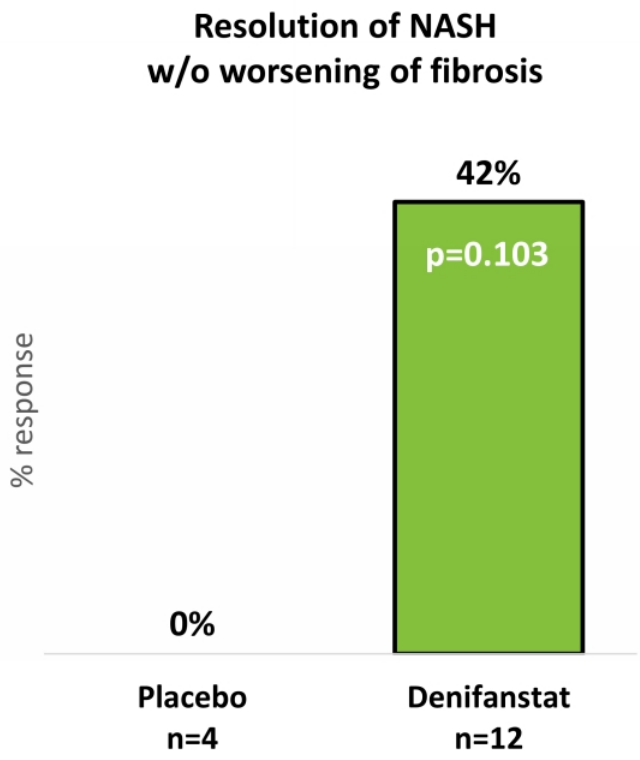
Cochran-Mantel-Haenszel Test – One sided at the 0.05 significance level. mITT population

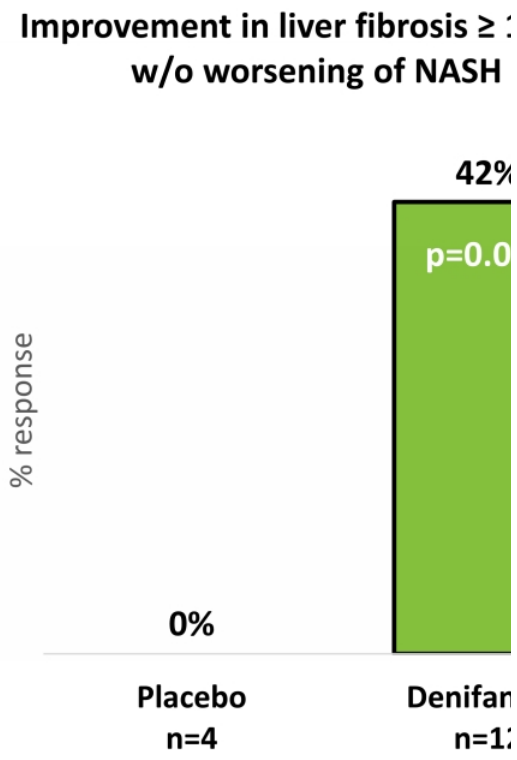
Independent Fibrosis Analysis by AI-based Digital Pathology
Supporting Evidence that Denifanstat Significantly Reduced Fibrosis



 Cochran-Mantel-Haenszel Test – One sided at the 0.05 significance level. LS means; least squares mean. HistoIndex platform. mITT population.

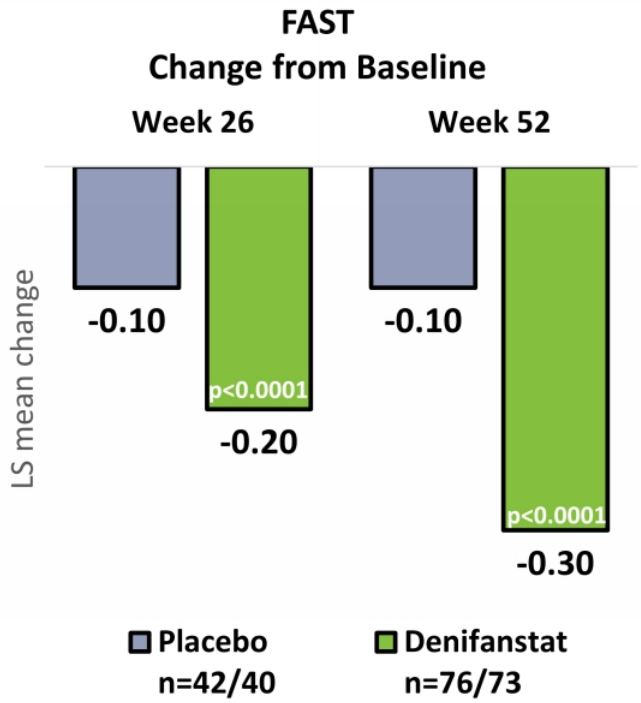
Patient Subset on Stable GLP1-RA at Baseline: Liver Biopsy
Denifanstat Improves NASH Resolution and Fibrosis

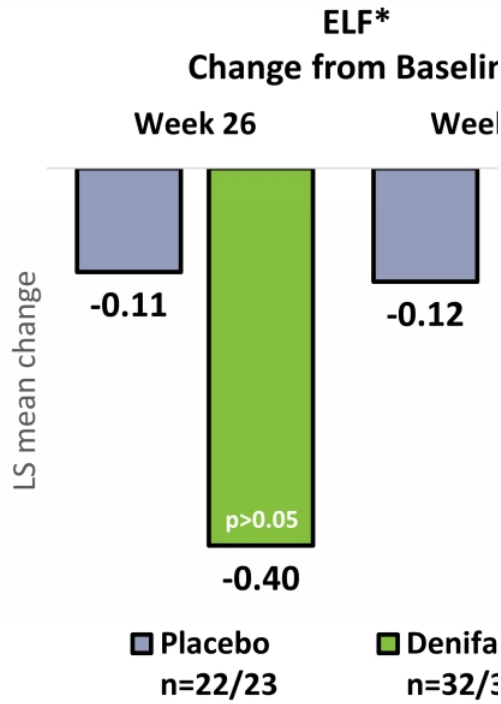




11 Cochran-Mantel-Haenszel Test – One sided at the 0.05 significance level. mITT population

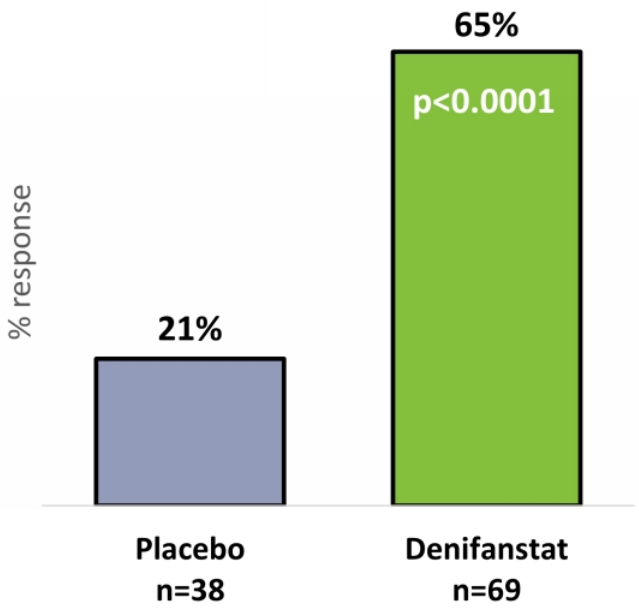
Biomarkers of Fibrosis
Denifanstat Decreased FAST Score and ELF

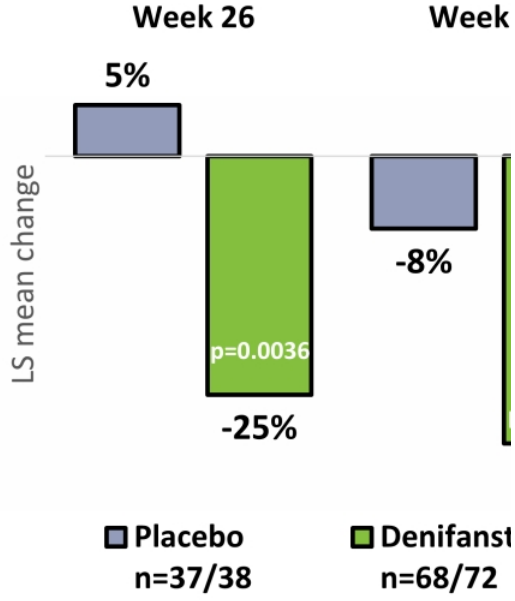




12 Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. mITT population. *Baseline ELF > 9.8 (mean).

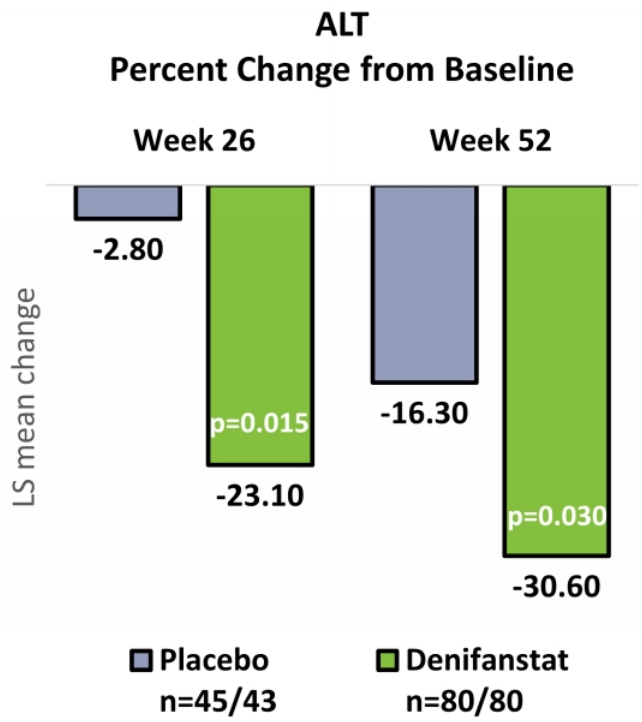
Secondary Endpoint: Liver Fat by MRI-PDFF
Denifanstat Achieved Statistical Significance

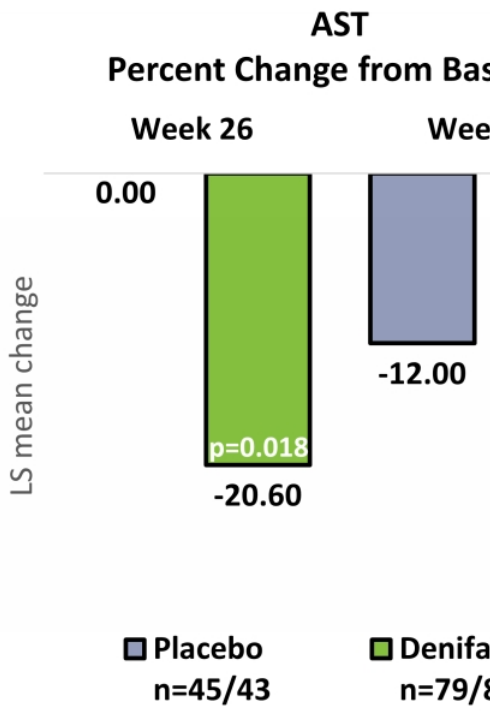




13 ≥30% reduction: Cochran-Mantel-Haenszel Test. Relative reduction: Mixed-effects Model for Repeated Measures . mITT population

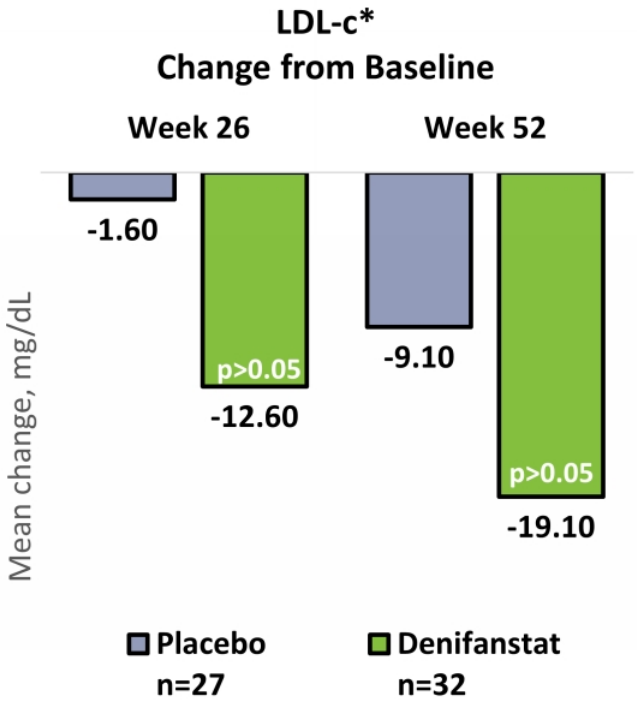
Secondary Endpoints: Liver Enzymes
Denifanstat Decreased ALT and AST Levels

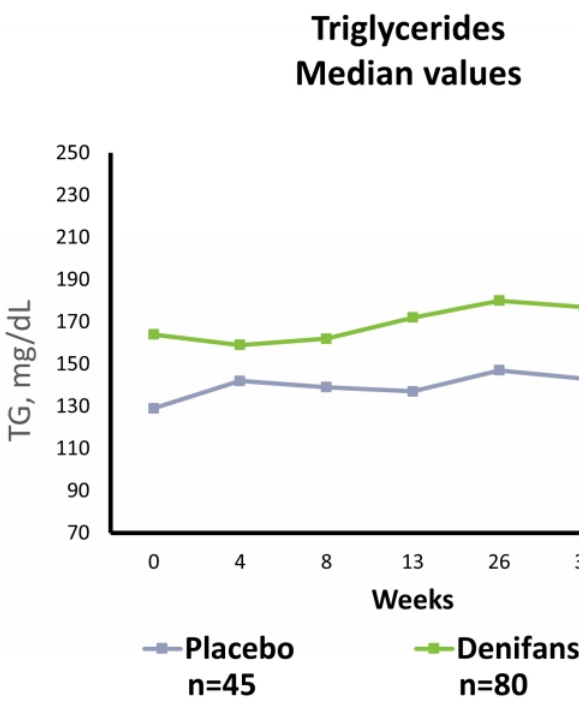




 Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. mITT population

Cardiometabolic health
Denifanstat Decreased LDL-c Levels





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

FASCINATE-2: Safety
Denifanstat was Generally Well Tolerated

Parameter	Placebo n=56	Denifans N=11
Any TEAE (treatment emergent adverse event)	45 (80.4%)	96 (85.7
TEAE related to study drug	20 (35.7%)	51 (45.5
Most common TEAE related to study drug in ≥5% of patients by system organ class		
eye disorders	9 (16.1%)	17 (15.2
gastrointestinal disorders	5 (8.9%)	13 (11.6
skin and subcutaneous tissue disorders	4 (7.1%)	25 (22.3
TEAE leading to study drug discontinuation	3 (5.4%)	22 (19.6
TEAE with CTCAE Grade 3 (Severe) or higher*	3 (5.4%)	13 (11.6
SAE (none related to treatment)	3 (5.4%)	13 (11.6
Fatal TEAE	0	0

* No treatment-related AE was Grade 3 or higher

Development Pipeline: Indications and Clinical Milestones

Therapeutic Area	Indication	Stage of Development	Expected Milest
		Preclinical / Phase 1 / Phase 2 / Phase 3	
Metabolic disease	NASH - F2/F3	TVB-2640	• Phase 2b successfu
		TVB-2640	• Phase 1 hepatic im 1Q 2024
Dermatology	Acne	TVB-3567	• IND 1H 2024 filing
		TVB-2640 (ASC40) (歌礼 ascletis)	• Phase 3 clinical stu
Oncology	Solid tumors		• Patient selection a dependent tumor t
	Recurrent glioblastoma (GBM)	TVB-2640 (ASC40) (歌礼 ascletis)	• Phase 3 enrollmen achieved in 3Q 202 planned*

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


End