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): May 23, 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 May 23, 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 in Item 7.01 of this Current Report on Form 8-K, including the information set forth in 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"), nor shall Exhibit 99.1 furnished herewith be deemed 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 a filing.

Item 9.01 Financial Statements and Exhibits

(d) Exhibits

Exhibit No.	Document
99.1	Investor Presentation of Sagimet Biosciences Inc., dated May 23, 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: May 23, 2024

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


SAGIMET
BIOSCIENCES
Targeting Metabolic Dysfunction with
Novel Therapies to Treat MASH, Acne and Cancer
May 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 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 statementsinvolve 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; 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.

Moreover, we operate 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 we may face. Except as required by applicable law, we do 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.

SAGIMET BIOSCIENCES

Proven Team with Development and Commercialization Experience Across 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

Thierry Chauche
CFO

- Provention Bio, Alexion Pharmaceuticals, Intercept Pharmaceuticals, Novartis
- MBA – The Wharton School of the University of Pennsylvania
- M.S. - Ecole Des Ponts ParisTech

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 MASH

- ✓ Key enzyme in de novo lipogenesis – responsible for excess liver fat in MASH
- ✓ FASN inhibition directly improves the 3 key drivers of MASH – 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 response
- ✓ Predictive biomarkers identify likely responders
- ✓ Opportunity to personalize treatment and optimize outcomes

Strong rationale for FASN in acne and cancer

Acne
- ✓ Clinical proof of concept established in Phase 1
- ✓ Positive Phase 2 topline results announced in May 2023 by license partner Ascletis
- ✓ Ascletis Phase 3 in severe acne vulgaris ongoing

Cancer
- ✓ Clinical proof of concept established in Phase 1
- ✓ Phase 3 rGBM trial enrollment for interim analysis completed in September 2023 by Ascletis

Strong financial position

- ✓ Upsized IPO completed in July 2023 raised $86.2 million of net proceeds
- ✓ Follow-on financing completed in January 2024 raised net proceeds of $104.7 million.
- ✓ Cash, cash equivalents and marketable securities were $193.7 million as of March 31, 2024, expected to fund current operations through 2025

SAGIMET BIOSCIENCES

Development Pipeline: Indications and Clinical Milestones

Therapeutic Area	Indication	Stage of Development	Expected Milestone / Status
Metabolic disease	MASH - F2/F3	TVB-2640 (Phase 2b successfully completed; MASH Phase 3 study planned to start 2H 2024)	• Phase 2b successfully completed; MASH Phase 3 study planned to start 2H 2024
		TVB-2640 (Phase 1 hepatic impairment results 1Q 2024)	• Phase 1 hepatic impairment results 1Q 2024
Dermatology	Acne	TVB-3567 (IND-enabling studies completed; evaluating timing to file IND)	• IND-enabling studies completed; evaluating timing to file IND
		TVB-2640 (ASC40) 歌礼 ascletis (Phase 3 clinical study initiated 4Q 2023*)	• Phase 3 clinical study initiated 4Q 2023*
Oncology	Solid tumors	(Patient selection and trial design in FASN-dependent tumor types ongoing)	• Patient selection and trial design in FASN-dependent tumor types ongoing
	Recurrent glioblastoma (GBM)	TVB-2640 (ASC40) 歌礼 ascletis (Phase 3 enrollment of 120 patients achieved in 3Q 2023; interim analysis planned*)	• Phase 3 enrollment of 120 patients achieved in 3Q 2023; interim analysis planned*

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

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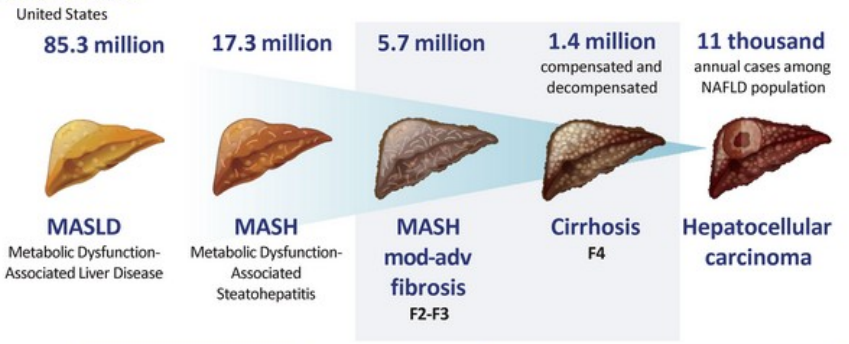
MASH: A Burgeoning Epidemic

Patients in 2016[1]

United States



Disease challenges

- Only one recently approved drug in U.S., and no approved drugs in 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

Denifanstat

- ✓ Designed for once-daily, oral dosing
- ✓ Rigorous and de-risked development strategy
- ✓ Direct DNL inhibition demonstrated in Phase 1b
- ✓ Improvements observed across biomarkers in Phase 2a
- ✓ Topline data of successfully completed Phase 2b announced in 1Q 2024
- ✓ Precision medicine approach to improve patient outcomes

DNL = de novo lipogenesis

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

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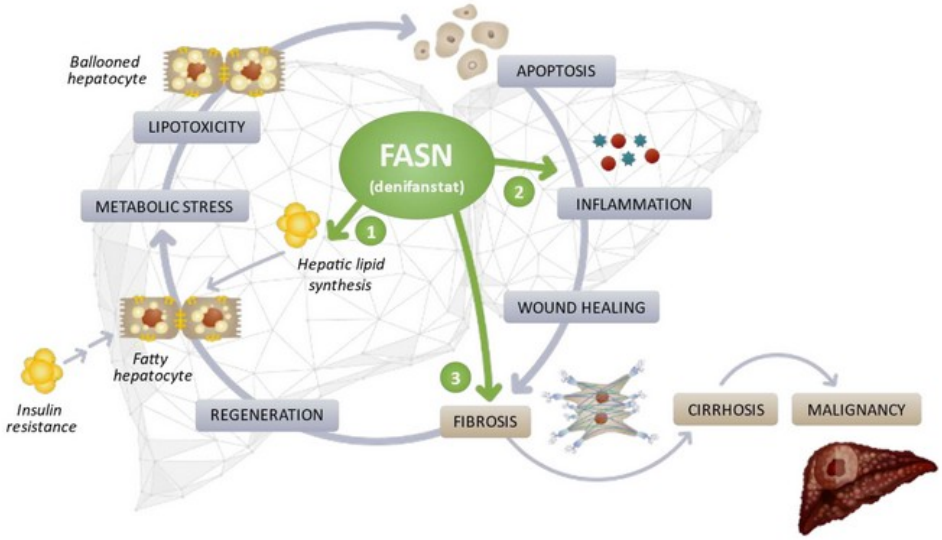
Denifanstat in MASH

Denifanstat: Differentiated Mechanism Believed to Target Key Drivers of MASH

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		Cohort 3
	US Placebo N=31	US 25mg N=33	US 50 mg N=35	China Placebo N=9	China 50 mg N=21	US 75mg N=13
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 (23%) Gr 2: 6 (46%)
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 (8%) Gr 2: 6 (46%)

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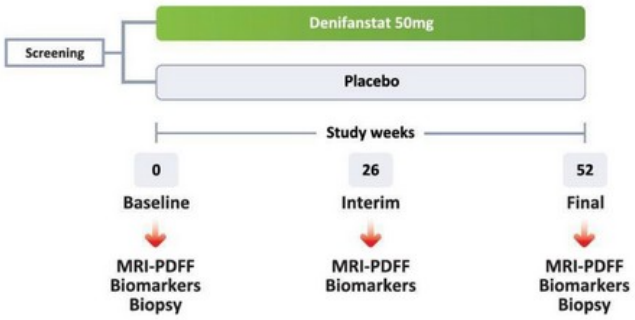
FASCINATE-2 Phase 2b Biopsy Trial Design
Measuring Histological Improvement

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

Other selected endpoints

- Improvement in liver fibrosis ≥1 stage without worsening of NASH (Bx)
- Digital AI pathology
- MRI-PDFF: absolute decrease, % change from 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 Score.

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FASCINATE-2 Baseline Characteristics
Typical 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 (%)

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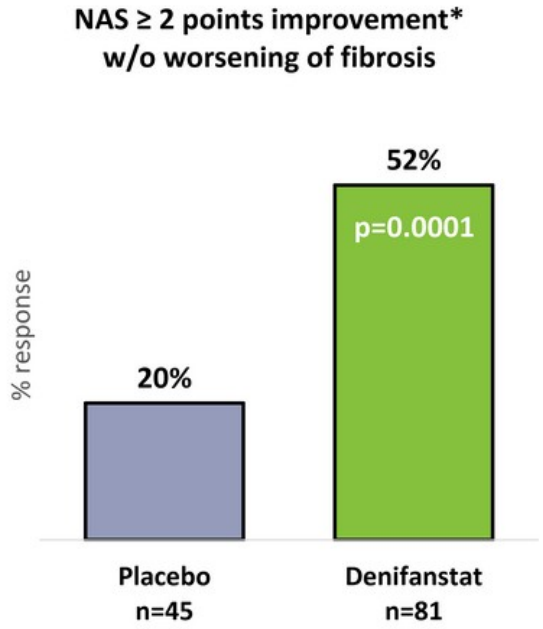
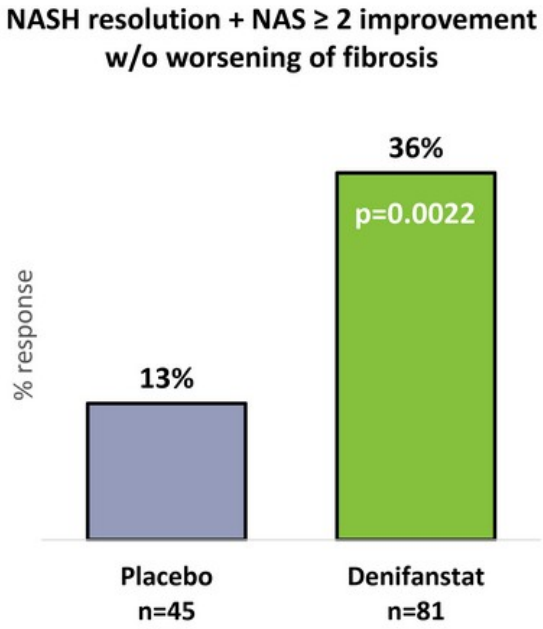
Primary Endpoints: Liver Biopsy
Denifanstat Achieved Statistical Significance





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

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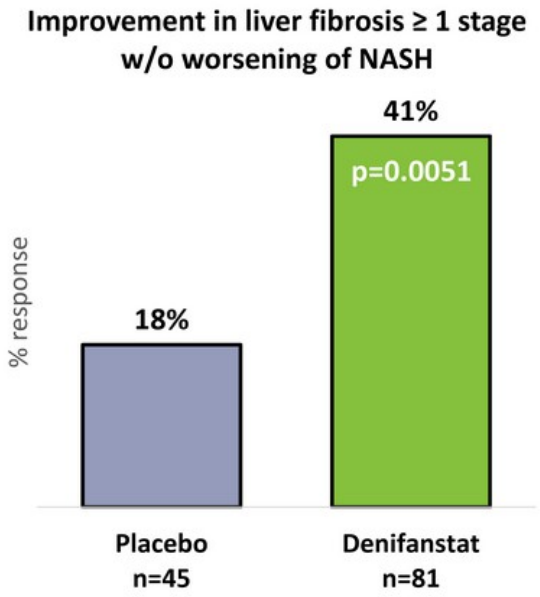
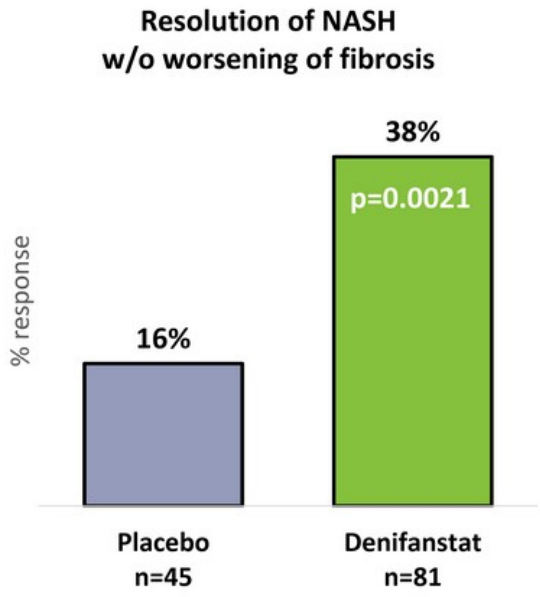
Secondary Endpoints: Liver Biopsy
Denifanstat Achieved Statistical Significance





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

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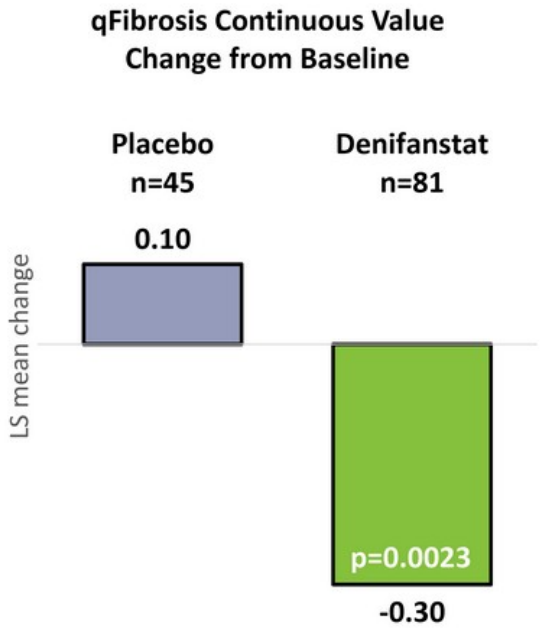
Independent Fibrosis Analysis by AI-based Digital Pathology
Supporting Evidence that Denifanstat Significantly Reduced Fibrosis



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

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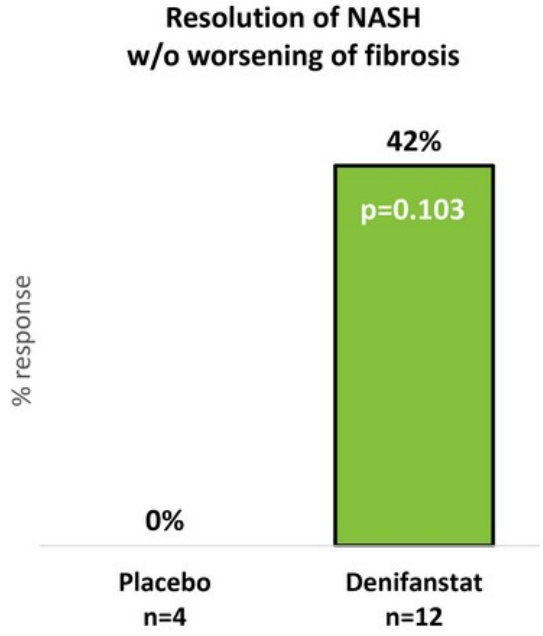
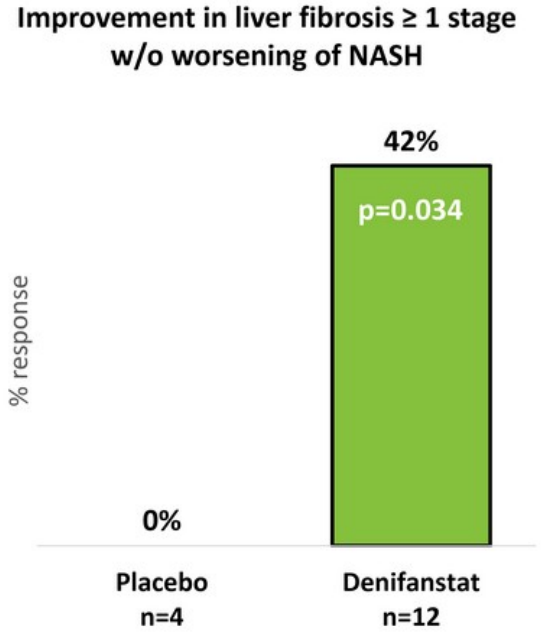
Patient Subset on Stable GLP1-RA at Baseline: Liver Biopsy

Denifanstat Improves NASH Resolution and Fibrosis





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

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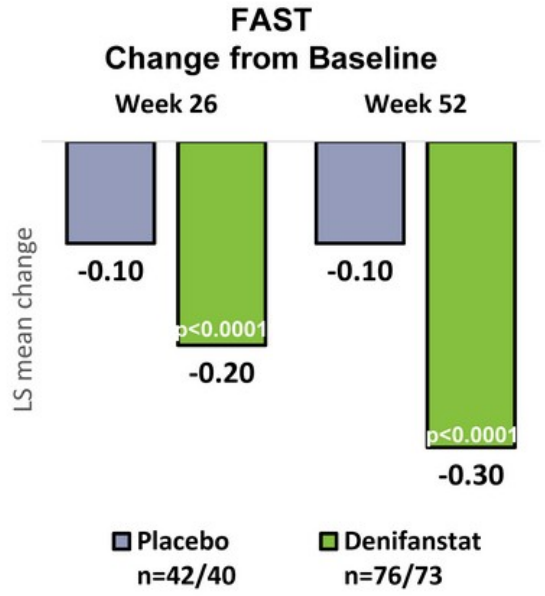
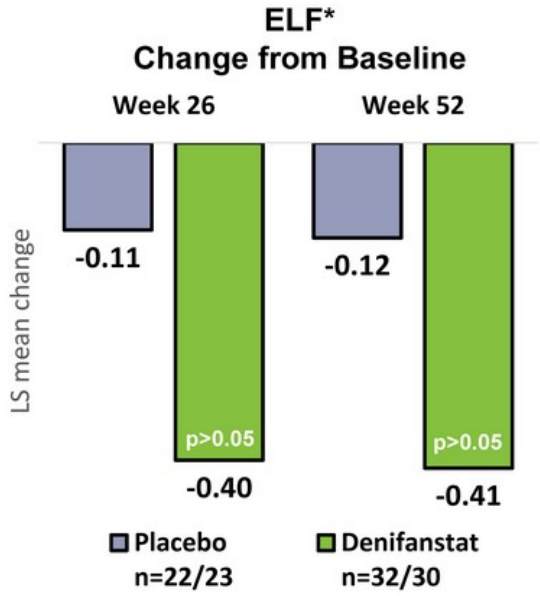
Biomarkers of Fibrosis
Denifanstat Decreased FAST Score and ELF





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

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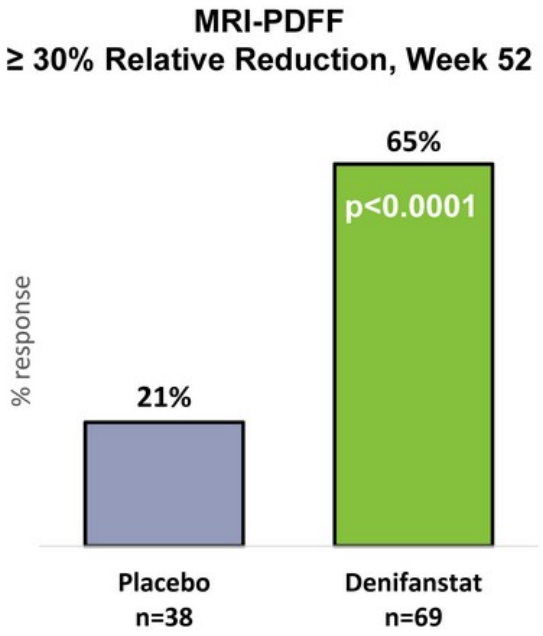
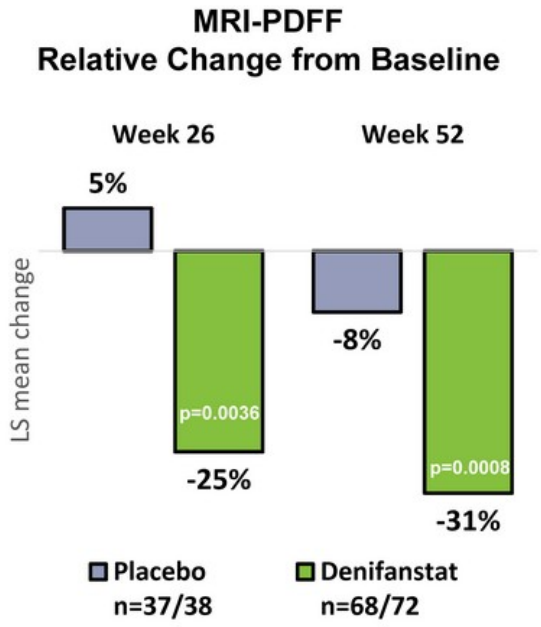
Secondary Endpoint: Liver Fat by MRI-PDFF
Denifanstat Achieved Statistical Significance





17 ≥30% reduction: Cochran-Mantel-Haenszel Test. Relative reduction: Mixed-effects Model for Repeated Measures . mITT population

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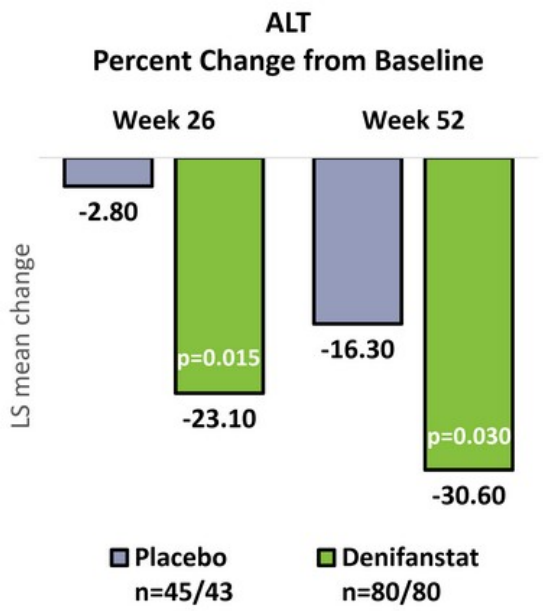
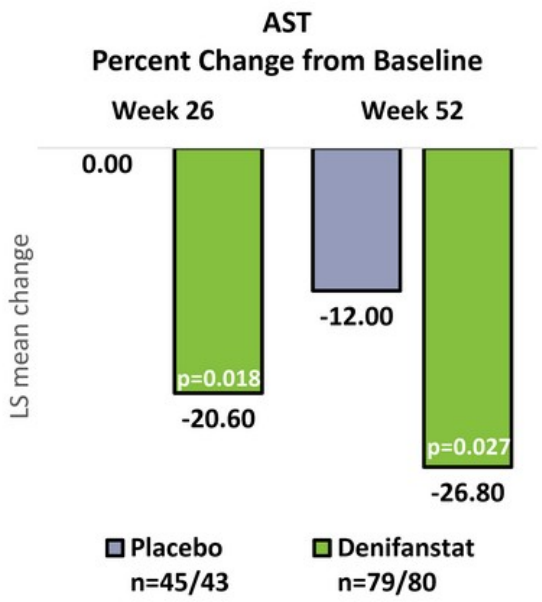
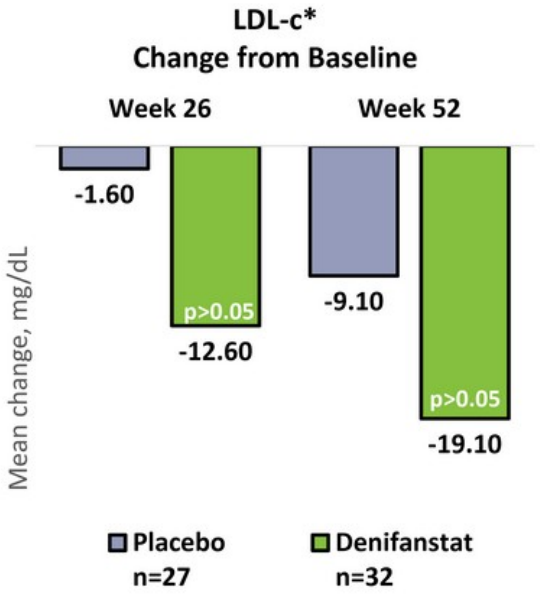
Secondary Endpoints: Liver Enzymes
Denifanstat Decreased ALT and AST Levels

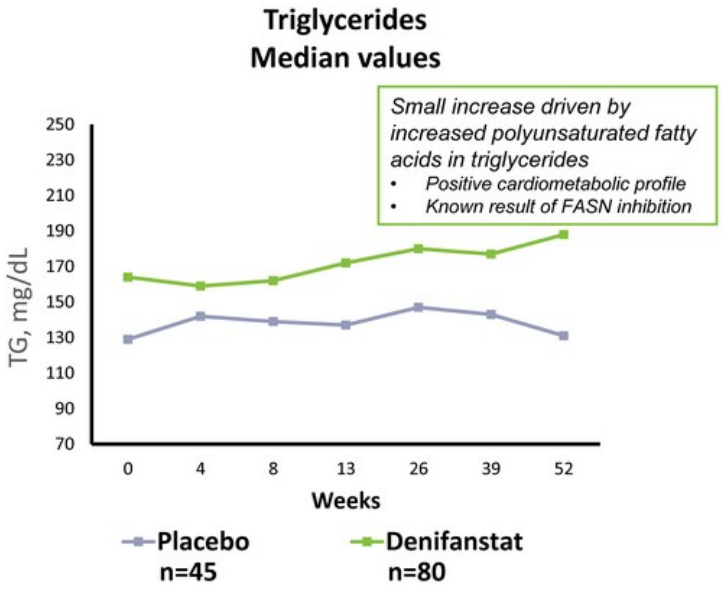




18 Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. mITT population

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	Denifanstat N=112
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

SAGIMET BIOSCIENCES

MASH Development Program

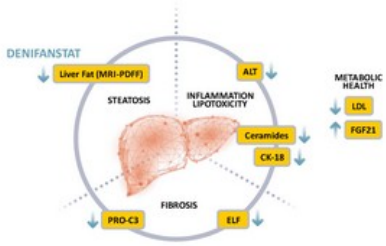
Progression from Phase 2b to Phase 3

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

Phase 3 Fibrosis endpoint - human

- Using Phase 2b results including AI pathology scores to design and power Phase 3
- Held end of Phase 2 Meeting with FDA in May 2024; in discussion with FDA regarding development plans and study protocol

MASH Phase 3 study planned to start 2H 2024

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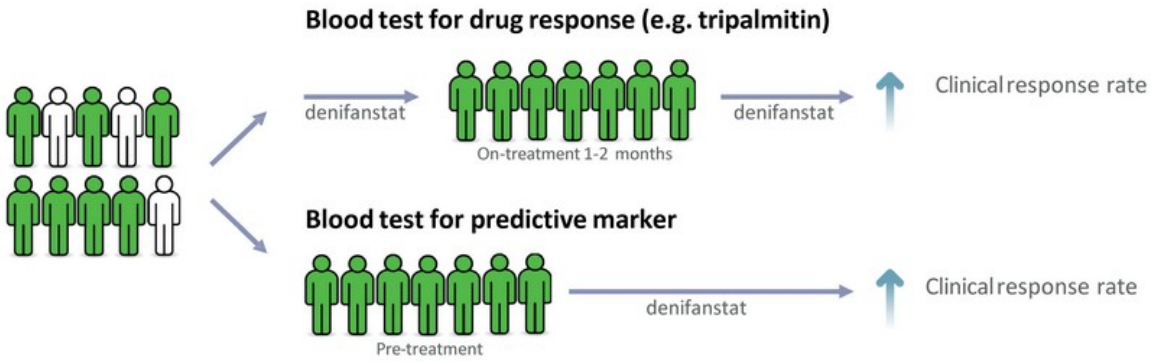
We Believe Denifanstat is Differentiated in the Evolving MASH Landscape

Mechanism	FASN inhibitors	THRß Agonists	FGF-21	GLP-1 agonists	PPAR agonists	ACC inhibitors	FXR agonists
Category	**DNL pathway**	Nuclear receptor	Growth factor	GLP-1	Nuclear receptor	DNL pathway	Nuclear receptor
Route	**Oral**	Oral			Oral	Oral	Oral
Status	**Phase 2 complete** **Phase 3 to start 2H 2024**	Approved March 2024	Phase 2 complete	Phase 2 complete	Phase 3 ongoing	Phase 2 complete	Phase 3 complete
Challenges	• Perceived market pressure from incretin class of weight loss drugs	• Diarrhea • Potential hormonal axis changes	• Bone loss • Injectable • Nausea and diarrhea • Potential neutralizing antibodies • Higher COGS	• GI side effects including nausea • Lack of fibrosis improvement to date • Muscle wasting	• Weight gain, edema, GI side effects, anemia • Possible liver injury	• Combinations only • MOA causes triglyceride increases • Lack of fibrosis improvement as monotherapy	• Mixed results from several programs • MOA causes pruritus and LDL-cholesterol increases

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
  - 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%.*

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Strong Monotherapy Opportunity for Denifanstat in MASH

Expansion as backbone of combinations

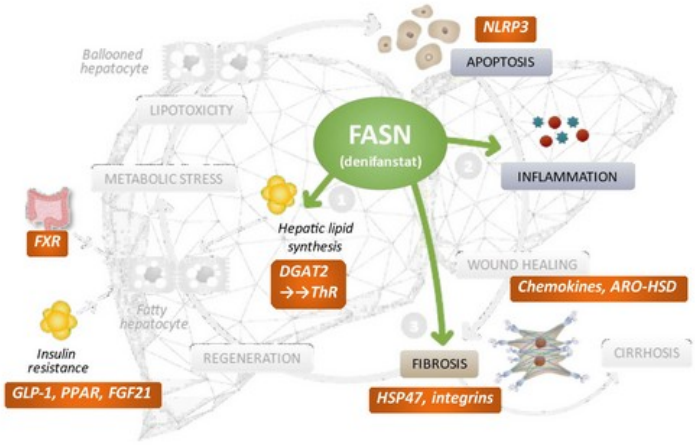
Denifanstat data support success as first line monotherapy

- ✓ 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
  - MASH agents: anti-fibrotic, other metabolic agents
  - Co-morbidities: diabetes and other cardiovascular agents

Illustrative potential combo mechanisms



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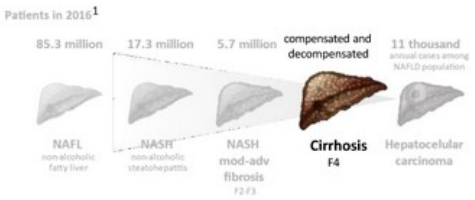
Additional Expansion Opportunities in MASH

- **Compensated cirrhotic patients (MASH 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 MASH-F4



- **Pediatric MASH**
  - 23% of children with NAFLD have MASH 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 MASH

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

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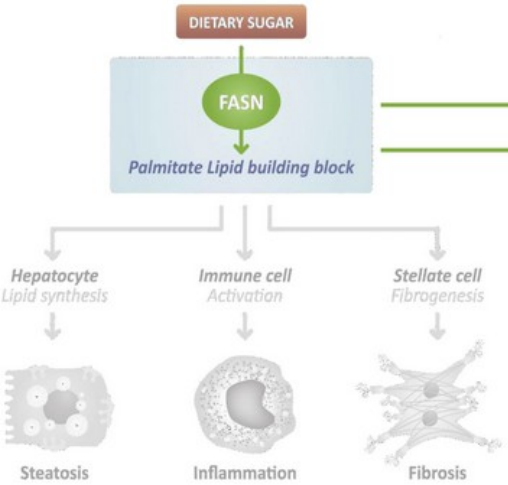
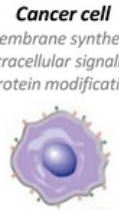
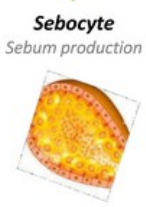
Other Indications

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







FASN in MASH

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 acne

1. Sebum production
2. Sebum composition

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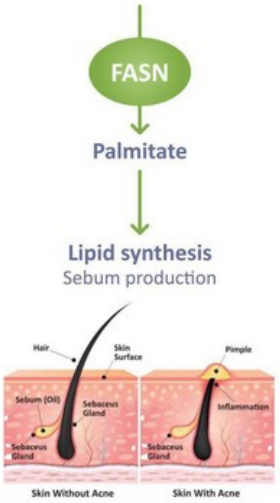
DNL Pathway Plays a Role in the Pathogenesis of Acne

FASN is an attractive therapeutic target for 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



Phase 1 – sebum analysis
by Sagimet

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

Phase 2 – acne
by Ascletis in China

	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%

Well tolerated across dose groups

* p <0.05 ** p <0.01

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FASN is Integral to Tumor Cell Proliferation and Survival

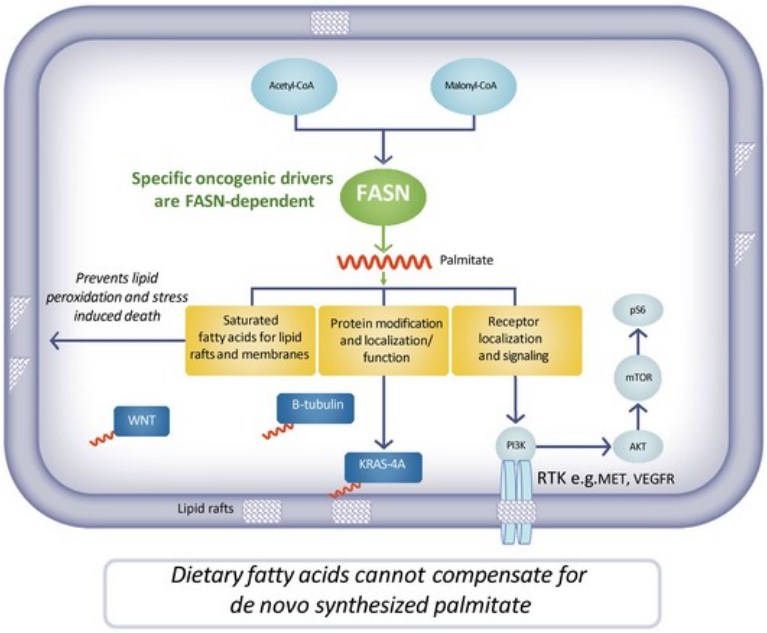
Reprogramed metabolism is one of the hallmarks of cancer

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 compensate for de novo synthesized palmitate

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FASN-Dependent Tumor Types Identified that Meet Core Criteria

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 milestone
NSCLC KRASM	**Preclinical ongoing** • Combination with KRAS inhibitor in mouse models ✓ Encouraging Phase 1 results with denifanstat	If positive, favor clinical collaboration with a KRASM industry partner
HCC FASN-dependent	**Translational ongoing** • Patient selection bioinformatics ✓ Positive preclinical results	If patient selection is tractable, Sagimet would sponsor a clinical study
Prostate FASN-dependent	**Phase 1 pending start** • Investigator Sponsored Trial at Weill Cornell, in combination with enzalutamide ✓ Positive preclinical results	Phase 1 results will inform clinical decision by Sagimet
GBM	**Phase 3 ongoing** • By Ascletis in China, in combination with bevacizumab ✓ Positive Phase 2 investigator sponsored trial results	Phase 3 results will inform clinical decision by Sagimet

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 $86.2 million of net proceeds
- ✓Follow-on financing completed in January 2024 raised net proceeds of $104.7 million.
- ✓Cash, cash equivalents and marketable securities were $193.7 million as of March 31, 2024, expected to fund current operations through 2025

Strong patent estate

- ✓ Denifanstat method of use: 2036
- ✓ Denifanstat composition of matter: 2032 (Issued in all key commercial territories)
- ✓ Opportunities exist to lengthen patent exclusivity of either composition patent or method of use 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 denifanstat via patent applications directed to formulations, methods of use, and synthetic methods, with potential to extend exclusivity further

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Development Pipeline: Indications and Clinical Milestones

Therapeutic Area	Indication	Stage of Development	Expected Milestone / Status
Metabolic disease	MASH - F2/F3	TVB-2640 (Phase 2b successfully completed; MASH Phase 3 study planned to start 2H 2024)	• Phase 2b successfully completed; MASH Phase 3 study planned to start 2H 2024
		TVB-2640 (Phase 1 hepatic impairment results 1Q 2024)	• Phase 1 hepatic impairment results 1Q 2024
Dermatology	Acne	TVB-3567 (IND-enabling studies completed; evaluating timing to file IND)	• IND-enabling studies completed; evaluating timing to file IND
		TVB-2640 (ASC40) 歌礼 ascletis (Phase 3 clinical study initiated 4Q 2023*)	• Phase 3 clinical study initiated 4Q 2023*
Oncology	Solid tumors	(Patient selection and trial design in FASN-dependent tumor types ongoing)	• Patient selection and trial design in FASN-dependent tumor types ongoing
	Recurrent glioblastoma (GBM)	TVB-2640 (ASC40) 歌礼 ascletis (Phase 3 enrollment of 120 patients achieved in 3Q 2023; interim analysis planned*)	• Phase 3 enrollment of 120 patients achieved in 3Q 2023; interim analysis planned*

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

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