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): August 14, 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 2.02 Results of Operations and Financial Condition

On August 14, 2024, Sagimet Biosciences Inc. (the "Company") issued a press release announcing its financial results for the quarter ended June 30, 2024. A copy of the press release is furnished as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated herein by reference.

The information contained in this Item 2.02 (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 (the "Securities Act"), or the Exchange Act, except as shall be expressly set forth by specific reference in such filing.

Item 8.01 Other Events.

On August 14, 2024, the Company updated information reflected in a slide presentation, which is attached as Exhibit 99.2 to this Current Report on Form 8-K and is incorporated herein by reference. Representatives of the Company will use the updated presentation in various meetings with investors from time to time.

Item 9.01 Financial Statements and Exhibits

(d) Exhibits

Exhibit No.	Document
99.1	Press Release of Sagimet Biosciences Inc., dated August 14, 2024
99.2	Investor Presentation of Sagimet Biosciences Inc., dated August 14, 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: August 14, 2024

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

Exhibit 99.1

Sagimet Biosciences Reports Second Quarter 2024 Financial Results and Provides Corporate Updates

Denifanstat Phase 2b FASCINATE-2 clinical trial 52-week data was presented in June at the European Association for the Study of the Liver (EASL) Congress

Preparations are ongoing to initiate a Phase 3 clinical development program for denifanstat in patients with metabolic dysfunction-associated steatohepatitis (MASH) in the second half of 2024

Two biotechnology industry leaders, Anne Phillips and Jennifer Jarrett, joined the Board of Directors effective August 1, 2024

Anticipated cash runway through 2025 with cash, cash equivalents and marketable securities totaling $188.5 million as of June 30, 2024

San Mateo, Calif., August 14, 2024 – Sagimet Biosciences Inc. (Nasdaq: SGMT), a clinical-stage biopharmaceutical company developing novel therapeutics targeting dysfunctional metabolic pathways, today reported financial results for the second quarter ended June 30, 2024, and provided recent corporate updates.

"In June, we presented the full Phase 2b FASCINATE-2 clinical trial 52-week biopsy results at EASL showing denifanstat's statistically significant fibrosis reduction in advanced F2 and F3 patients and a statistically significant delay in progression to cirrhosis," said David Happel, Chief Executive Officer of Sagimet. "We believe these encouraging data, which demonstrate denifanstat's mechanism of action as the only fat synthesis inhibitor that directly targets the three key drivers of MASH -- fat accumulation, inflammation, and fibrosis -- differentiate denifanstat from other therapeutics in the field. We plan to initiate the Phase 3 program for denifanstat in MASH in the second half of 2024 and intend to share the Phase 3 pivotal trial design later in the year. We look forward to progressing the development of denifanstat for patients living with MASH, a condition which has grown to epidemic levels worldwide."

Recent Corporate Highlights

- On June 6, 2024, Sagimet presented the full 52-week data from the ITT and F3 patient population in the Phase 2b FASCINATE-2 clinical trial of denifanstat at EASL. Key outcomes data included:
  - A statistically significant improvement in liver fibrosis by ≥1-stage without worsening of MASH at 52-weeks in ITT population and in patients with baseline stage 3 fibrosis.
    - ITT (denifanstat 30% vs. placebo 14%, p=0.0199), and
    - F3 mITT (denifanstat 49% vs. placebo 13%, p=0.0032)

- A statistically significant improvement in liver fibrosis by ≥2-stage without worsening of MASH at 52-weeks in mITT population and in patients with baseline stage 3 fibrosis.
  - mITT (denifanstat 20% vs. placebo 2%, p=0.0065), and
  - F3 mITT (denifanstat 34% vs. placebo 4%, p=0.0065)
- A statistically significant difference in progression to cirrhosis in F4 mITT population (denifanstat 5% vs. placebo 11%, p=0.0386).
- A statistically significant difference in fibrosis improvement by ≥ 1 stage with no worsening of MASH for patients on a stable background dose of a GLP-1RA (denifanstat 42% vs. placebo 0%, p=0.034) in mITT population.
- A statistically significant increase in beneficial polyunsaturated triglycerides at the end of 52 weeks of treatment (+42% denifanstat vs. -4% placebo, p<0.001) in the mITT population.
- Tripalmitin, a biomarker of denifanstat activity, showed an early and sustained reduction in de novo lipogenesis at 4-weeks (-2.4ug/ml with denifanstat vs. -0.4ug/mL placebo, p=0.001) and 13-weeks (-2.2ug/mL with denifanstat vs. -0.1ug/mL placebo, p=0.005) in the ITT population.

- On June 13, the Company hosted a conference call and webcast (link here) featuring 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, and Principal Investigator of the Phase 2b FASCINATE-2 clinical trial. In the webcast, Dr. Loomba reviewed denifanstat's strong fibrosis data as well as the preclinical data supporting the potential clinical use of denifanstat in combination with other medicines such as GLP-1s and thyroid-hormone receptor-beta (TRβ) agonists, including resmetirom, which he indicated could synergistically improve outcome of disease.
- On August 1, the Company announced the appointment to its board of directors of Dr. Anne Phillips and Jennifer Jarrett, two biotechnology industry leaders with extensive experience in clinical development, regulatory strategy, operations, and finance.

Anticipated Upcoming Milestones

- Following the company's End-of-Phase 2 meeting with the FDA in May 2024, discussions are currently ongoing with the FDA regarding the Phase 3 development plans for denifanstat in MASH. A contract with a Contract Research Organization (CRO) for the global Phase 3 development program has been executed and study start-up operational activities are ongoing. Pending the FDA's feedback, the company plans to start the Phase 3 program in the second half of 2024.
- The Phase 3 study of denifanstat in acne, conducted by license partner Ascletis Pharmaceuticals, is recruiting in China and expected to be fully enrolled by the end of 2024. This Phase 3 study was initiated after positive Phase 2 acne data reported in Q2 2023 [link].

Financial Results for the Three and Six Months Ended June 30, 2024

- **Cash and cash equivalents and marketable securities** as of June 30, 2024 was $188.5 million, including $104.7 million net proceeds from our January 2024 follow-on offering, which are expected to fund operations through 2025 based on management's current operating plan.

- **Research and development expense** for the three and six months ended June 30, 2024 was $6.3 million and $11.6 million, respectively, compared to $4.7 million and $9.2 million for the three and six months ended June 30, 2023, respectively.

- **General and administrative expense** for the three and six months ended June 30, 2024 was $4.3 million and $7.8 million, respectively, compared to $2.4 million and $4.7 million for the three and six months ended June 30, 2023, respectively.

- **Net loss** for the three and six months ended June 30, 2024 was $8.1 million and $14.7 million, respectively, compared to $6.8 million and $13.4 million for the three and six months ended June 30, 2023, respectively.

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 MASH. FASCINATE-2, a Phase 2b clinical trial of denifanstat in MASH with liver biopsy-based primary endpoints, was successfully completed with positive results. For additional information about Sagimet, please visit www.sagimet.com.

About MASH

MASH is a progressive and severe liver disease which is estimated to impact more than 115 million people worldwide, for which there is only one recently approved treatment in the United States and no currently approved treatments in Europe. In 2023, global liver disease medical societies and patient groups formalized the decision to rename non-alcoholic fatty liver disease (NAFLD) to metabolic dysfunction-associated steatotic liver disease (MASLD) and nonalcoholic steatohepatitis (NASH) to MASH. Additionally, an overarching term, steatotic liver disease (SLD), was established to capture multiple types of liver diseases associated with fat buildup in the liver. The goal of the name change was to establish an affirmative, non-stigmatizing name and diagnosis.

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 expected timing of the presentation of data from ongoing 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 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 within anticipated timelines, including its Phase 3 denifanstat program; 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.

Contact:
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LifeSci Advisors
JAllaire@LifeSciAdvisors.com

SAGIMET BIOSCIENCES INC.

CONDENSED STATEMENTS OF OPERATIONS AND COMPREHENSIVE LOSS

(Unaudited)

(in thousands, except for share and per share amounts)

	Three Months Ended June 30,		Six Months Ended June 30,	
	2024	2023	2024	2023
Operating expenses:				
Research and development	$ 6,313	$ 4,676	$ 11,575	$ 9,163
General and administrative	4,276	2,381	7,782	4,659
Total operating expenses	10,589	7,057	19,357	13,822
Loss from operations	(10,589)	(7,057)	(19,357)	(13,822)
Interest income and other	2,471	272	4,610	450
Net loss	$ (8,118)	$ (6,785)	$ (14,747)	$ (13,372)
Other comprehensive income (loss):				
Net unrealized income (loss) on marketable securities	(30)	13	(53)	84
Comprehensive loss	$ (8,148)	$ (6,772)	$ (14,800)	$ (13,288)
Net loss per share, basic and diluted	$ (0.25)	$ (35.80)	$ (0.48)	$ (71.39)
Weighted-average shares outstanding, basic and diluted	31,913,887	189,520	30,476,657	187,314

SAGIMET BIOSCIENCES INC.

CONDENSED BALANCE SHEETS

(Unaudited)

(in thousands)

	As of	
	June 30, 2024	December 31, 2023
Cash, cash equivalents and marketable securities	$ 188,491	$ 94,897
Total assets	$ 189,020	$ 96,719
Current liabilties	$ 5,728	$ 5,654
Stockholders' equity	$ 183,292	$ 91,065
Liabilities and stockholders' equity	$ 189,020	$ 96,719

Exhibit 99.2


SAGIMET
BIOSCIENCES
Targeting Metabolic Dysfunction with Novel Therapies to Treat MASH, Acne & Cancer
August 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 statements involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. In some cases, you can identify forward-looking statements by terms such as "may," "will," "should," "would," "expect," "plan," "anticipate," "could," "intend," "target," "project," "believe," "estimate," "predict," "potential," or "continue" or the negative of these terms or other similar expressions. The forward-looking statements in this presentation are only predictions. These forward-looking statements speak only as of the date of this presentation and are subject to a number of risks, uncertainties and assumptions, some of which cannot be predicted or quantified and some of which are beyond our control, including, among others: the clinical development and therapeutic potential of denifanstat or any other drug candidates we may develop; our ability to advance drug candidates into and successfully complete clinical trials, the risk the topline clinical trials may not be predictive of, and may differ from final clinical data and later-stage clinical trials; our ability to advance drug candidates into and successfully complete clinical trials within anticipated timelines, including our Phase 3 denifanstat program; that unfavorable new clinical trial data may emerge in other clinical trials of denifanstat, including Phase 3 clinical trials; that clinical trial data are subject to differing interpretations and assessments, including by regulatory authorities; our relationship with Ascletis, and the success of its development efforts for denifanstat; the accuracy of our estimates regarding our capital requirements; and our ability to maintain and successfully enforce adequate intellectual property protection. These and other risks and uncertainties are described more fully in the "Risk Factors" section of our most recent filings with the Securities and Exchange Commission and available at www.sec.gov. You should not rely on these forward-looking statements as predictions of future events. The events and circumstances reflected in our forward-looking statements may not be achieved or occur, and actual results could differ materially from those projected in the forward-looking statements.

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.

Leadership Team with Proven Development and Commercialization Experience

Dave Happel *President & CEO*

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

Brought multiple innovative healthcare products to the market

Thierry Chauche *CFO*

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

George Kemble *Executive Chairman*

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

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

Elizabeth Rozek *General Counsel*

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

Eduardo Martins *CMO*

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

Led clinical development team of cenicriviroc for MASH

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

HORIZON AstraZeneca abbvie Genentech proventionbio NOVARTIS cognoa basilea PHARMACEUTICA

FASN Inhibitor Denifanstat Offers a Unique and Validated Approach to MASH

Unique MOA: FASN Inhibition	• As the only fat synthesis inhibitor, denifanstat directly targets the 3 key drivers of MASH – liver fat, inflammation, and fibrosis • FASN is a key enzyme in de novo lipogenesis responsible for excess liver fat in MASH • Once daily oral administration, suitable for mono- or combination therapy
Positive FASCINATE-2 Phase 2b Data in MASH	• Met both primary endpoints in clinical trial: significant improvements in fibrosis with no worsening of MASH • Improvement in more severe patients (stage F3) and demonstrated lack of progression to cirrhosis • Enhanced treatment effect in patients with stable GLP therapy • Generally well tolerated
Near Term Milestones & Cash Position	• Pivotal Phase 3 program expected to begin in 2H2024 • NASDAQ: SGMT; $188.5M cash* on hand, expected to fund current operations through 2025 *Cash, cash equivalents and marketable securities as of June 30, 2024
Precision Medicine	• Tripalmitin and additional blood response markers under development as early biomarkers of target engagement and treatment response
Strategic Collaboration with Ascletis in Acne & Cancer	• Acne Phase 3 study completion of enrollment anticipated by end 2024 • rGBM Phase 3 study interim analysis anticipated by end 2024
Denifanstat IP Portfolio	• Method of use patent: 2036; Composition of matter patent: 2032 • Opportunities to lengthen one patent's life for up to 5 years via Patent Term Extension (US) or SPC (Europe)

Denifanstat: A Novel Small Molecule FASN Inhibitor Protected By Strong IP

Denifanstat

Designed for once-daily, oral dosing

Rigorous and de-risked development strategy

Direct DNL inhibition demonstrated in Phase 1b

Improvements in liver fat and other non-invasive biomarkers in Phase 2a

Topline data of successfully completed 52-week Phase 2b biopsy study announced in 1Q 2024

Precision medicine approach to improve patient outcomes

Strong patent estate

Denifanstat method of use expires in 2036

Denifanstat composition of matter expires in 2032 (issued in all key commercial 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

DNL = de novo lipogenesis

Development Pipeline: Indications and Clinical Milestones

Therapeutic Area	Indication	Stage of Development	Expected Milestone / Status
Metabolic Disease	MASH F2/F3 population	Denifanstat (Phase 2b) Denifanstat (Phase 1)	Phase 2b positive topline data announced 1Q2024; MASH Phase 3 estimated start 2H 2024 Phase 1 hepatic impairment results reported 1Q 2024
Dermatology	Acne	TVB-3567 (Preclinical) Denifanstat (ASC40) (Phase 2–3)	IND-enabling studies completed; evaluating timing to file IND Phase 3 clinical study initiated 4Q 2023; planned to be fully enrolled in 2024*
Oncology	Solid tumors	TVB-3567 (Preclinical–Phase 1) Denifanstat (Phase 1–2)	Identifying FASN-dependent tumor types for potential FASN inhibitor development.
	Recurrent glioblastoma (GBM)	Denifanstat (ASC40) (Phase 2–3)	Phase 3 enrollment of 120 patients achieved in 3Q 2023; pre-specified interim analysis planned in 2H 2024*

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

MASH: A Burgeoning Epidemic

Patients in 2016[1]

United States

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

MASH

- Expected to almost double in size within next 2 decades[2]
- Complex disease with heterogeneous patient population
- Significant opportunity for differentiated MOA

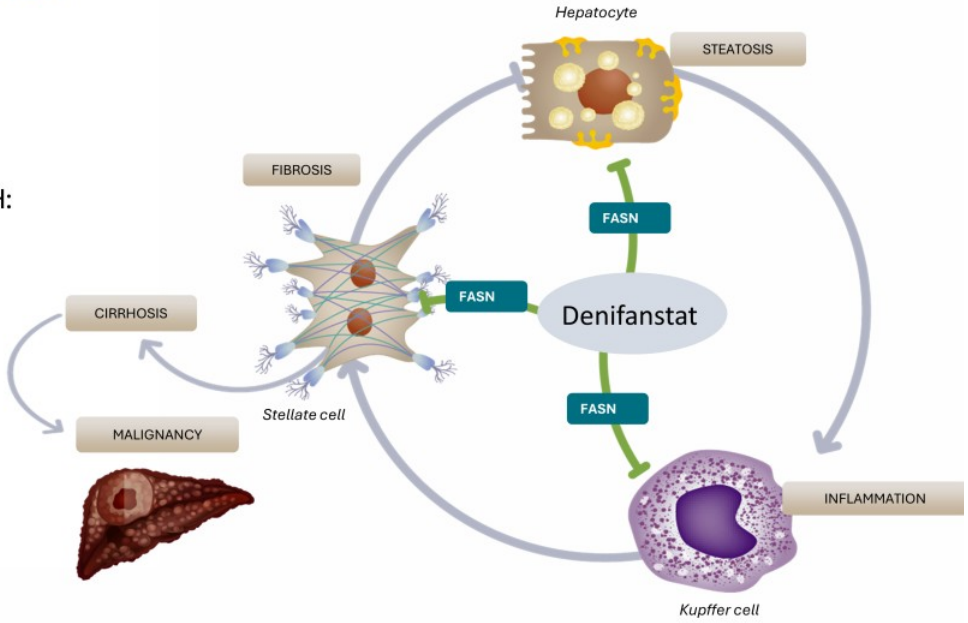
1 Estes, et al. 2018; http://dx.doi.org/10.1016/j.jhep.2018.05.036. Note: MASH, or metabolic dysfunction-associated steatohepatitis, was formerly known as NASH, or nonalcoholic steatohepatitis

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

FASN Inhibition Addresses Three Independent Mechanisms of MASH Development and Progression

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

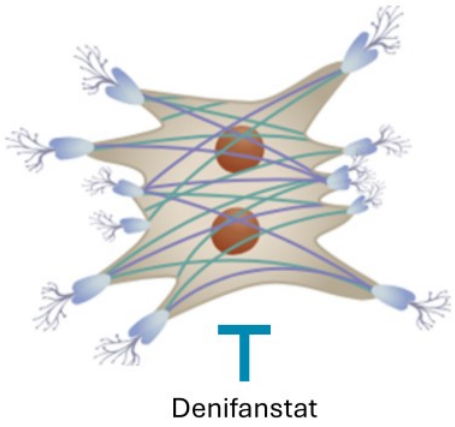
1. Blocking **steatosis** via inhibiting de novo lipogenesis in hepatocytes
2. Reducing **inflammation** via preventing immune cell activation
3. Blunting **fibrosis** via inhibiting stellate cell activation



FASN Inhibition Directly Blocks Human Liver Stellate Cell Function

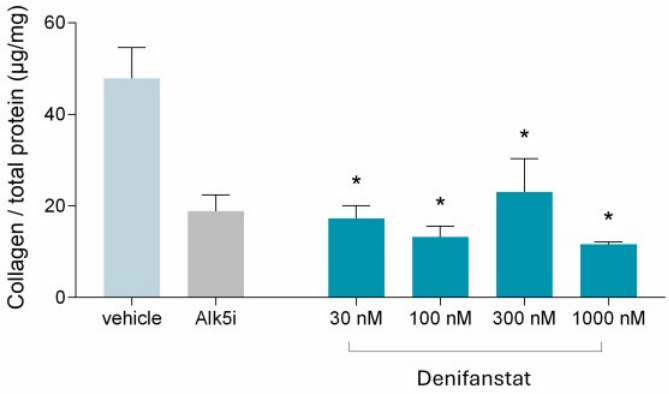
Stellate cells require DNL for fibrogenesis

Denifanstat blocks stellate cell activation



Primary human stellate cell assay

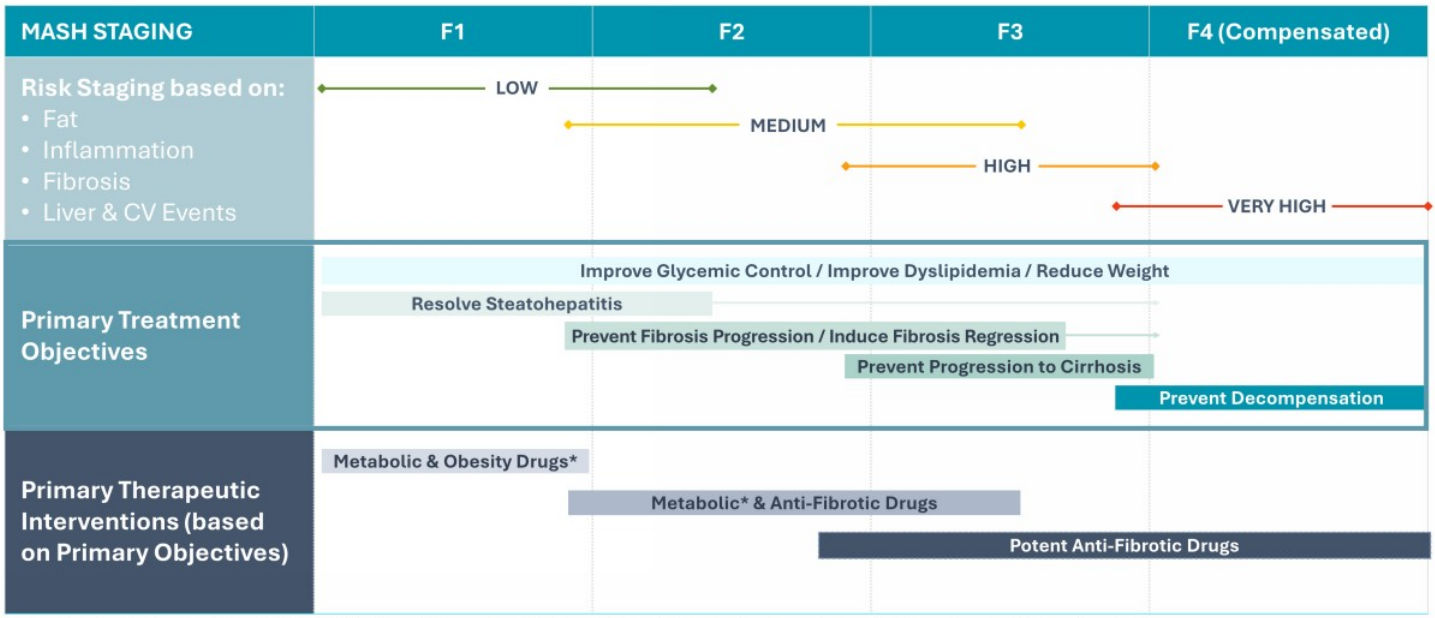
Denifanstat directly inhibits fibrogenic activity



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

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

Treatment Goals for MASH Across Fibrosis Staging



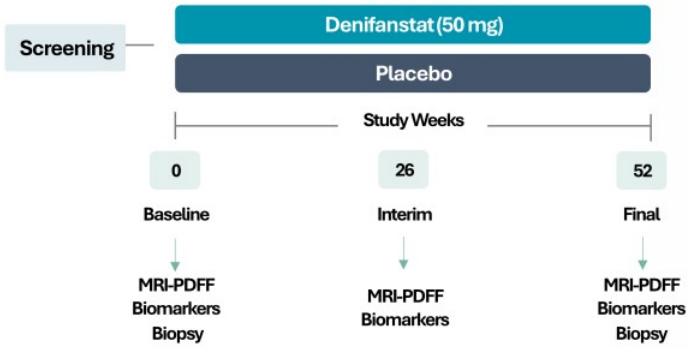
Kusi et al. Endocrine Practice 28 (2022) 528-562. Rinella et al. Hepatology. 2023 May 01; 77(5): 1797–1835. Tacke et al. Journal of Hepatology, July 2024. vol. - 4 | 1–51
*Metabolic drugs are anticipated to be background therapy for obesity and type 2 diabetes, until clinical data support use in MASH

MASH Clinical Development Program

SAGIMET BIOSCIENCES

FASCINATE-2: Biopsy Trial Design Focused on Histological Endpoints

FASCINATE-2 Phase 2b trial design



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

Primary endpoints

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

Selected secondary endpoints

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

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

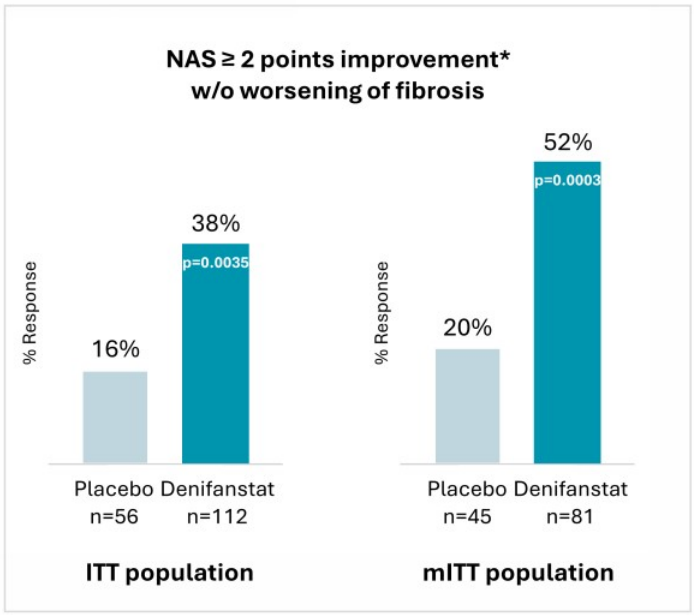
FASCINATE-2: Baseline Characteristics Were Typical of the F2/F3 MASH Population

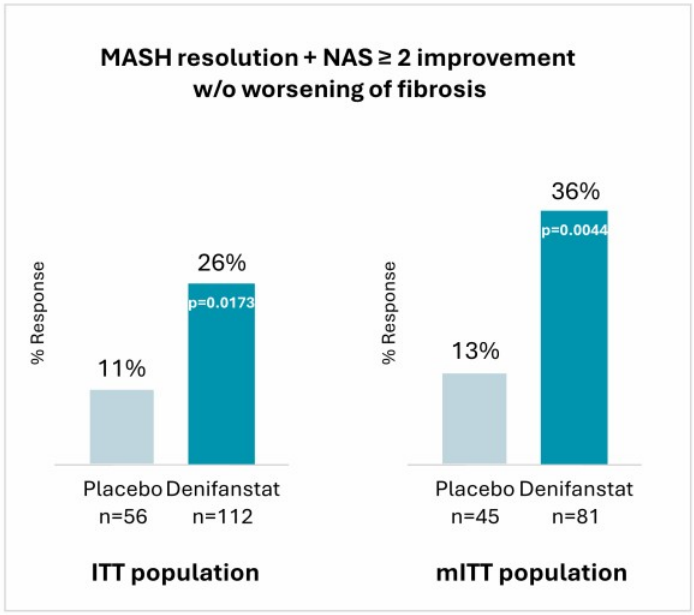
Parameter	Placebo, n=45	Denifanstat, n=81
Age, years	59.6 (+/- 10.9)	56.1 (+/- 10.8)
Sex, female	27 (60%)	48 (59%)
Race, White	41 (91%)	73 (90%)
Ethnicity, Hispanic or Latino	15 (33%)	27 (33%)
BMI, kg/m^2	36.5 (+/- 6.7)	34.6 (+/- 6.1)
Type 2 diabetes	27 (60%)	55 (68%)
ALT (alanine aminotransferase) U/L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

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

Primary Endpoints: Liver Biopsy

Denifanstat Achieved Statistical Significance at 52 Weeks

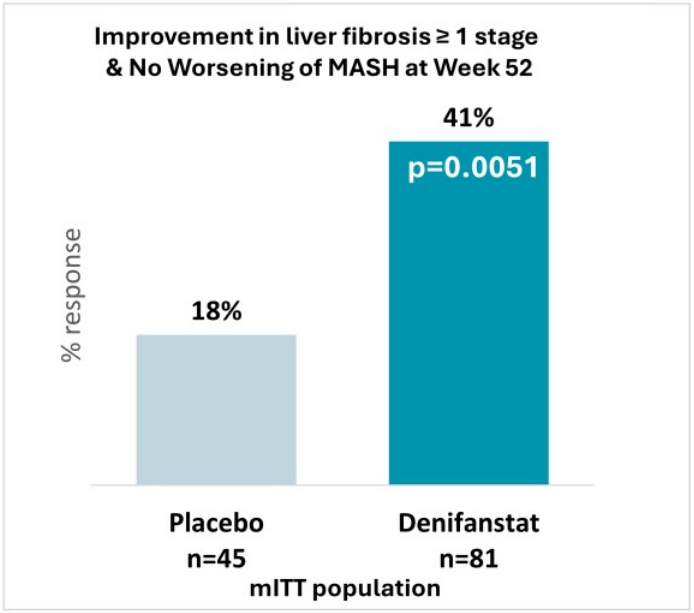


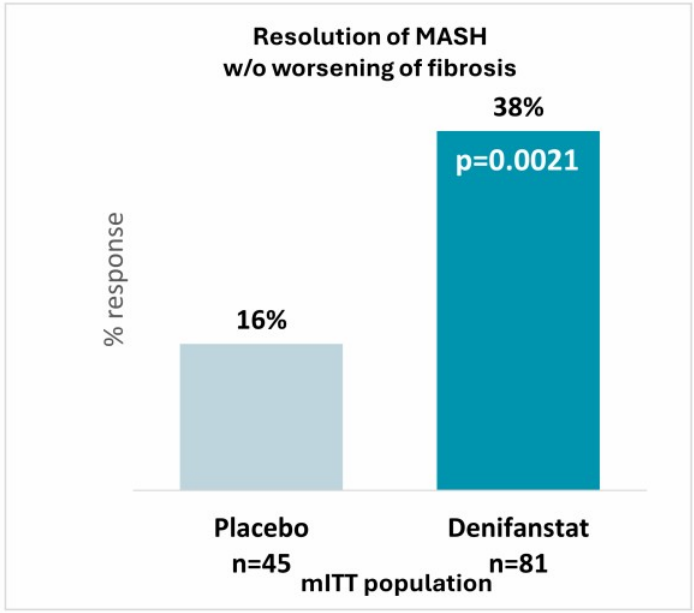


Cochran-Mantel-Haenszel Test – two sided at the 0.05 significance level. * ≥1-point improvement in ballooning or inflammation.

Secondary Endpoints: Liver Fibrosis and MASH Resolution

Denifanstat Achieved Statistical Significance





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

Secondary Endpoints: Liver Fibrosis

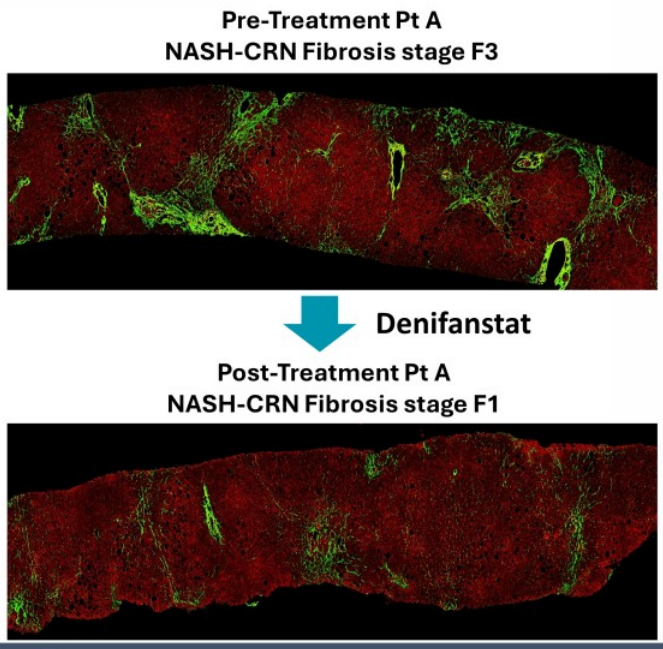
Denifanstat Achieved Profound Improvement of Fibrosis

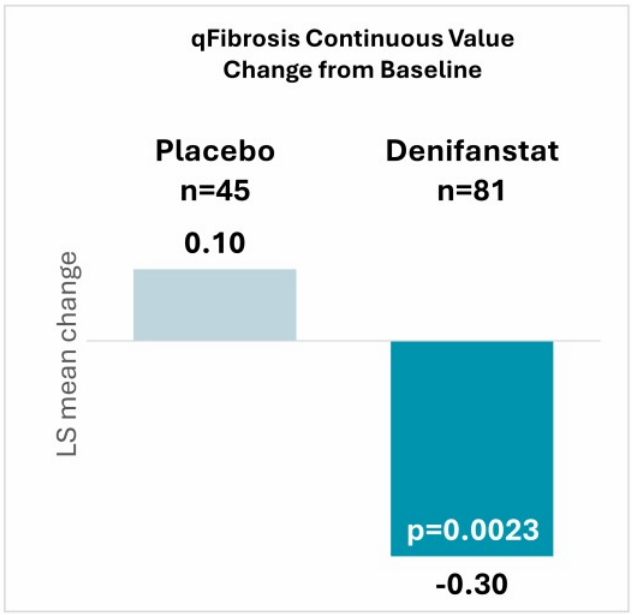
Fibrosis Endpoints	Subgroup	Placebo	Denifanstat	p-value
≥1 stage improvement in fibrosis w/o worsening of MASH	ITT	14%	30%	0.0199*
	mITT	18%	41%	0.0051*
	F3	13%	49%	0.0032**
≥2 stage improvement in fibrosis w/o worsening of MASH	mITT	2%	20%	0.0065**
	F3	4%	34%	0.0065**
Progression to cirrhosis (F4)	mITT	11%	5%	0.0386*

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

Additional Fibrosis Analysis Using AI-based Digital Pathology

Supporting Evidence that Denifanstat Significantly Reduced Fibrosis

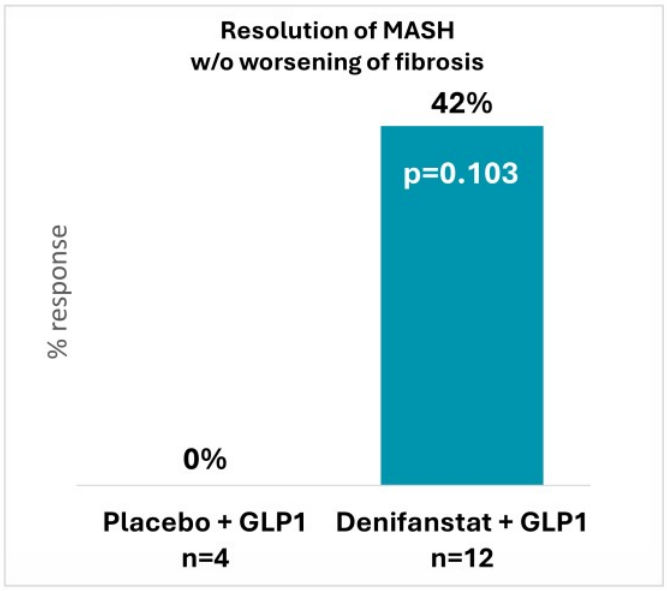




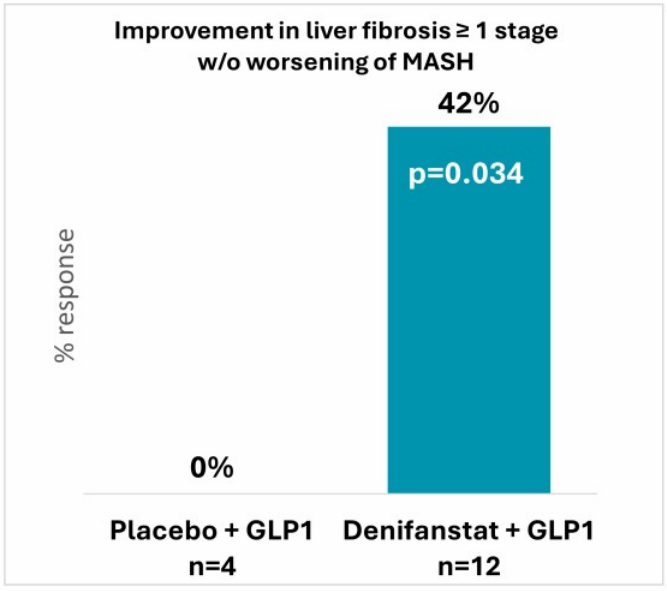
*One sided at the 0.05 significance level

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

Denifanstat Improved MASH Resolution and Fibrosis



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



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

FASCINATE-2: Safety

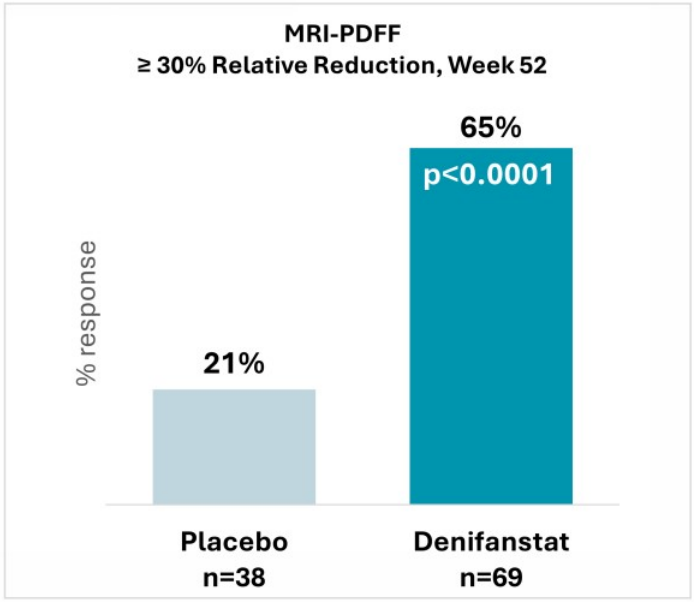
Denifanstat Was Generally Well Tolerated

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

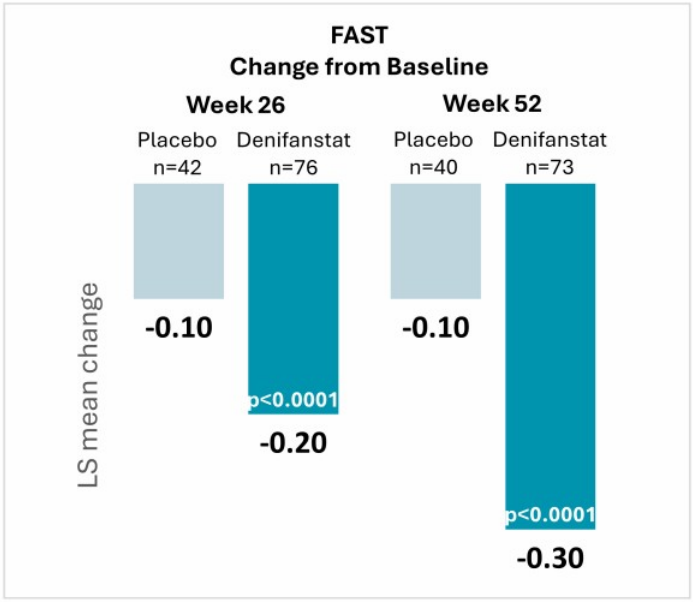
- No DILI signal and no muscle wasting were detected, and GI were comparable to placebo
- AE of hair thinning stabilized with a 2 to 4 week dose pause and then reversed with down titration or study completion
  - Consistent with other MASH-related medications, only 6% of patients discontinued from the study with hair thinning
  - In previous clinical studies of denifanstat, <2% of the patients experienced hair thinning at 50mg

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

Denifanstat Achieved Statistical Significance



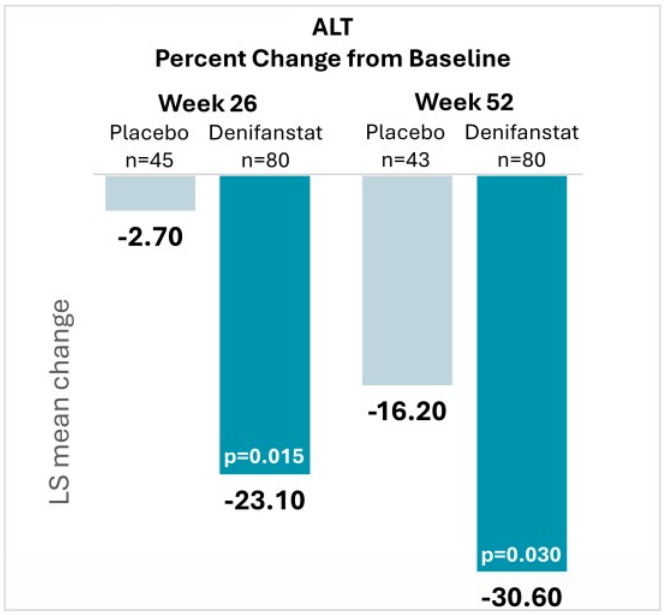
≥30% reduction: Cochran-Mantel-Haenszel Test. Relative reduction: Mixed-effects Model for Repeated Measures. mITT population. Two sided at the 0.05 significance level.

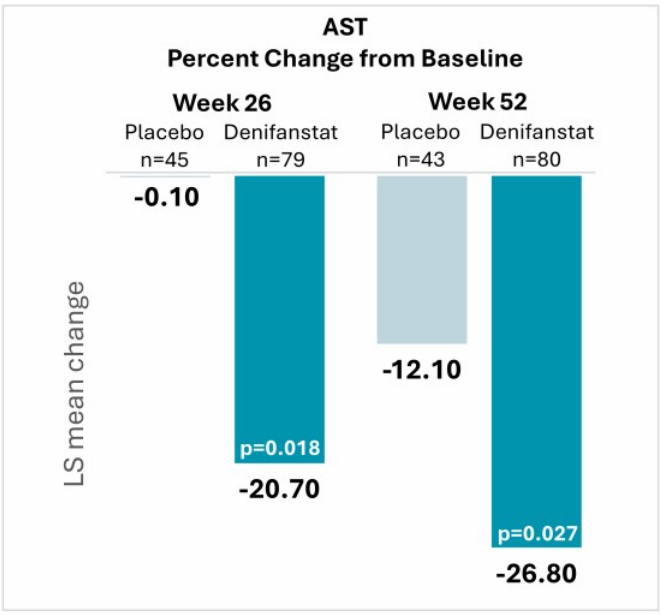


Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. mITT population.

Secondary Endpoints: Liver Enzymes

Denifanstat Decreased ALT and AST Levels

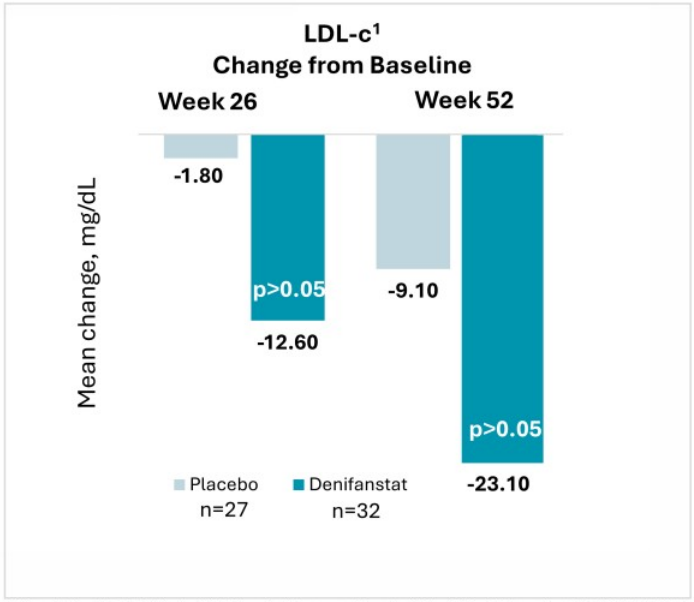


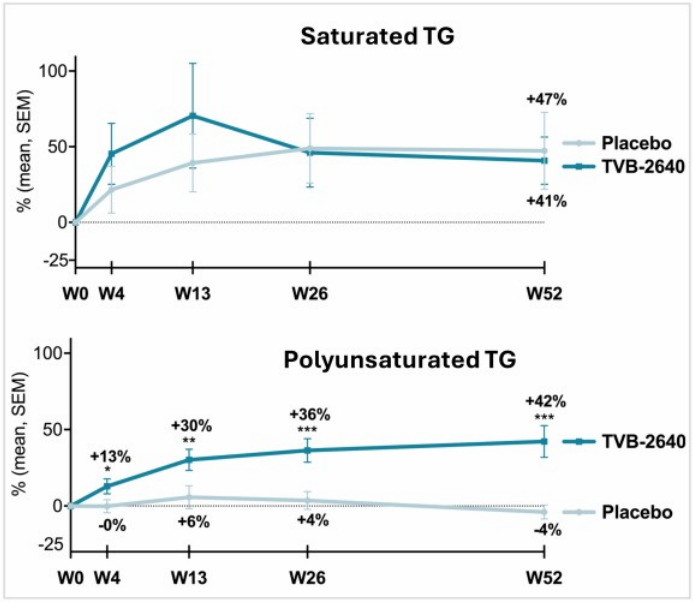


Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. mITT population

Cardiometabolic Health

Denifanstat Decreased LDL-c Levels and Increased Polyunsaturated Triglycerides

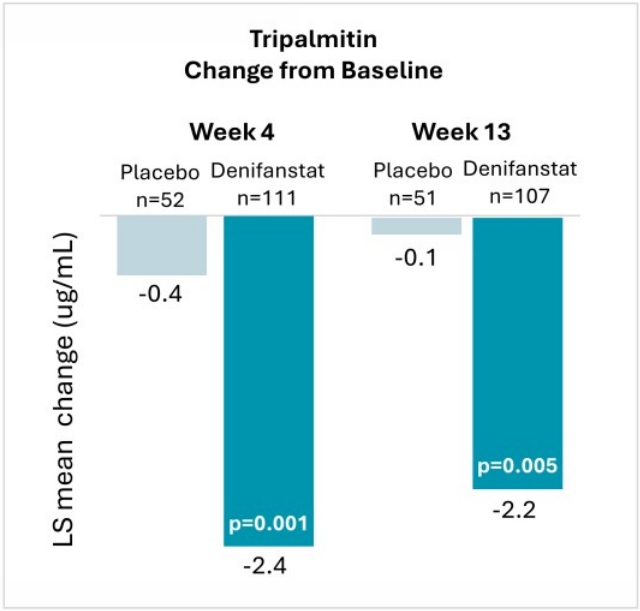




mITT population. Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. *p<0.05, **p<0.01, ***p<0.001

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

Denifanstat Rapidly and Robustly Reduced De Novo Lipogenesis



Tripalmitin

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

Next steps

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

Two sided at the 0.05 significance level, ITT population

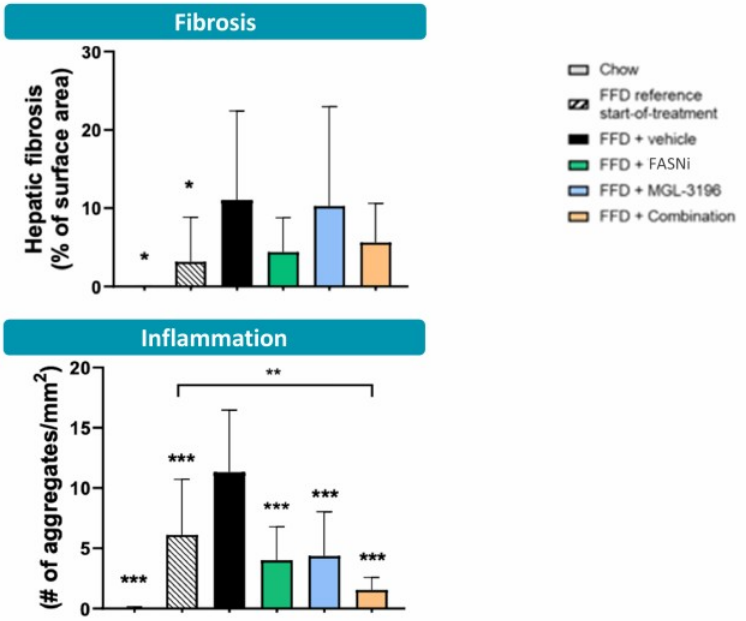
Mechanism of Action Supports Combination Therapy Opportunity

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

Combination therapy offers:

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

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



MOA- Mechanism of Action

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

Denifanstat Potential in Cirrhosis

Compensated Cirrhotic Patients (MASH F4)

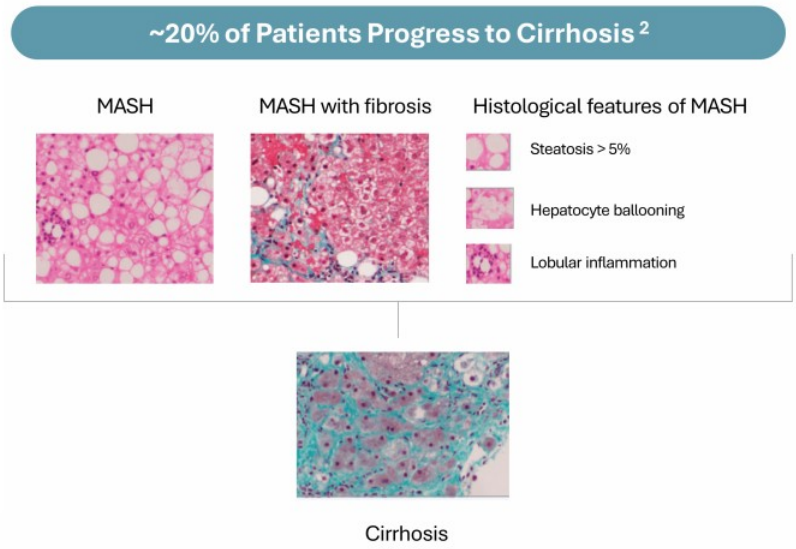
- Denifanstat reduces pro-fibrotic signaling stellate cells which retain the ability to remove the fibrotic scar and reestablish the basal ECM scaffold even in F4 MASH [1]
- Hepatocytes continue to be functional, and patients frequently have increased liver fat

Supportive Initial Data

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

Next Step

- Phase 2b/3 trial in MASH-F4



1 Kamm DR and McCommis KS. doi: 10.1113/JP281061. 2 Sheka AC, et al. doi:10.1001/jama.2020.2298. 3. CLIN-009 data on file. 4. Loomba, et al. EASL 2024

Pediatric MASH Continues to be an Area of Significant Unmet Need

Pediatric MASH

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

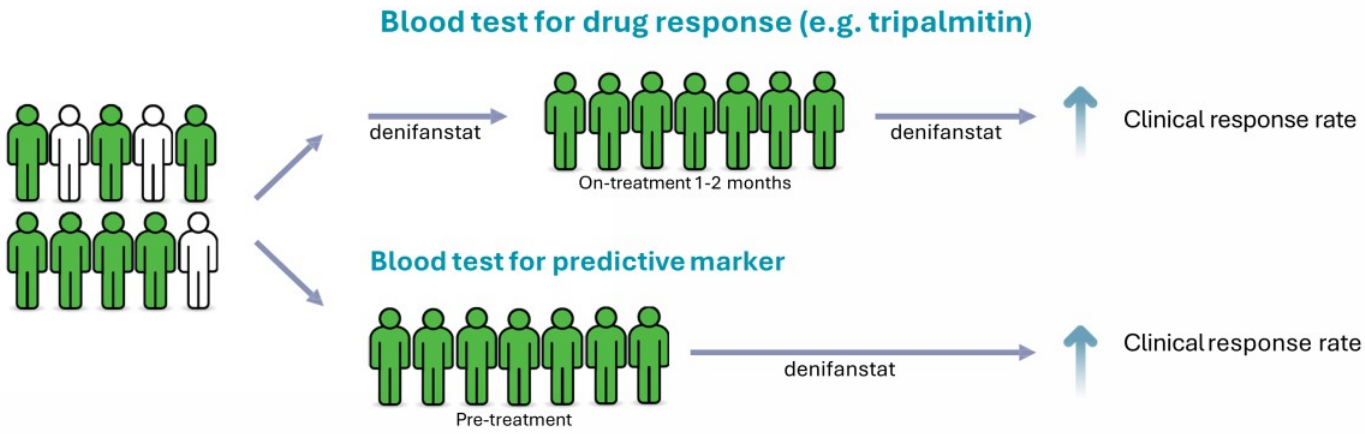
Next steps

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

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

Precision Medicine: Blood Tests May Lead to Improved Patient Outcomes

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



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

Additional Denifanstat Indications

SAGIMET BIOSCIENCES

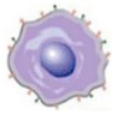
FASN Also Plays a Key Role in Other Diseases With Significant Unmet Need

FASN

Palmitate Lipid building block

Cancer cell

Membrane synthesis, intracellular signaling, protein modification



Sebocyte

Sebum production

FASN in cancer

Supports tumor survival

Enables tumor proliferation

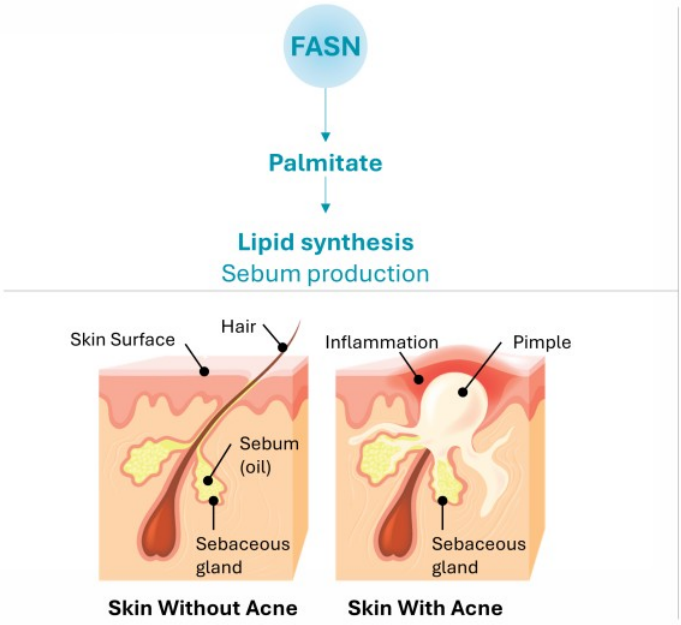
Establishes resistance to drugs

FASN in acne

Increases sebum production

Exacerbates sebum composition

DNL Pathway Plays a Role in the Pathogenesis of Acne



FASN is an attractive therapeutic target for acne

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

Ascletis Announced Positive Early Clinical Data in Acne; Phase 3 Study Ongoing

Denifanstat Phase 2 in acne
by Ascletis in China

歌礼 ascletis

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

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

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

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

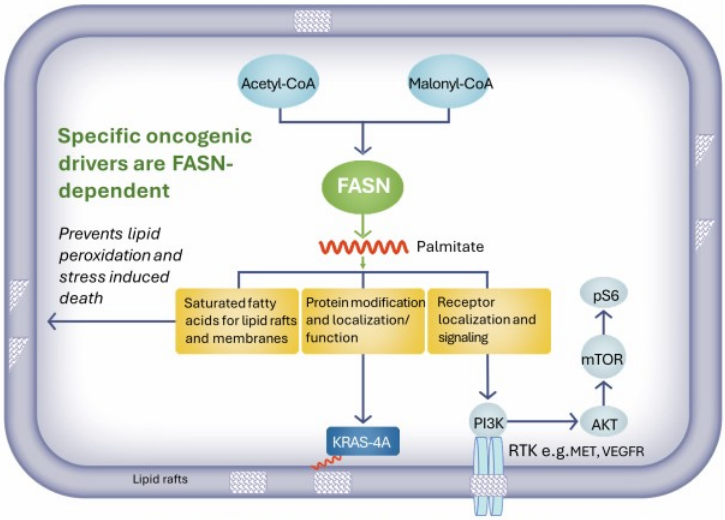
FASN Is Integral to Tumor Cell Proliferation and Survival

FASN dependence

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

Foundational Phase 1

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



Dietary fatty acids cannot compensate for de novo synthesized palmitate

KRASM – KRAS mutant. KRASWT- KRAS wild type

Cancer Program Focuses on 4 FASN-Dependent Tumor Types

Type	Status	Next milestone
GBM	**Phase 3 ongoing** In China by Ascletis, denifanstat combination with bevacizumab Positive investigator sponsored Phase 2 results*	Pre-specified interim analysis planned in 2H 2024
Prostate	**Phase 1 ongoing** Investigator Sponsored at Weill Cornell, denifanstat combination with enzalutamide	Phase 1 results expected 4Q 2025
HCC	**Translational work ongoing** Patient selection strategy by bioinformatics on primary samples Positive preclinical combination results**	Potential Phase 2 study of FASN inhibitor in combination with a marketed kinase inhibitor, ideally via collaboration with an industry partner
NSCLC KRASM	**Preclinical and clinical evidence** Positive preclinical combination with KRAS inhibitor*** Encouraging monotherapy Phase 1 results with denifanstat	Potential Phase 2 study of FASN inhibitor in combination with a KRAS inhibitor, ideally via collaboration with an industry partner

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

FASN Inhibitor Denifanstat Offers a Unique and Validated Approach to MASH

Unique MOA: FASN Inhibition

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

Positive FASCINATE-2 Phase 2b Data in MASH

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

Near Term Milestones & Cash Position

- Pivotal Phase 3 program expected to begin in 2H2024
- NASDAQ: SGMT; $188.5M cash* on hand, expected to fund current operations through 2025

*Cash, cash equivalents and marketable securities as of June 30, 2024

Precision Medicine

- Tripalmitin and additional blood response markers under development as early biomarkers of target engagement and treatment response

Strategic Collaboration with Ascletis in Acne & Cancer

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

Denifanstat IP Portfolio

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