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): February 2, 2026

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 February 2, 2026, Sagimet Biosciences Inc. (the "Company") issued a press release announcing that Ascletis Pharma Inc. announced on January 29th positive topline results in the open-label Phase 3 clinical trial evaluating the long-term safety of ASC40 (denifanstat) tablets in patients with moderate to severe acne. The full text of the Company's press release is furnished as Exhibit 99.1 to this Current Report on Form 8-K and incorporated herein by reference.

Also on February 2, 2026, 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.

The information in Item 7.01 of this Current Report on Form 8-K, including the information set forth in Exhibits 99.1 and 99.2, is being furnished and shall not be deemed "filed" for purposes of Section 18 of the Exchange Act, nor shall Exhibits 99.1 or 99.2 furnished herewith be deemed incorporated by reference in any filing under the Securities Act 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	Press Release of Sagimet Biosciences Inc., dated February 2, 2026.
99.2	Investor Presentation of Sagimet Biosciences Inc., dated February 2, 2026.
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: February 2, 2026

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

Exhibit 99.1

Sagimet Announces Positive 52-Week Data from License Partner Ascletis' Open-Label Phase 3 Clinical Trial Evaluating the Long-Term Safety of ASC40 (Denifanstat) Tablets in Patients with Moderate to Severe Acne

San Mateo, Calif., February 2nd, 2026 – Sagimet Biosciences Inc. (Nasdaq: SGMT), a clinical-stage biopharmaceutical company developing novel therapeutics targeting dysfunctional metabolic and fibrotic pathways, today announced that Ascletis Pharma Inc. issued a press release on January 29th reporting positive topline results in the open-label Phase 3 trial evaluating the long-term safety of ASC40 (denifanstat) tablets in patients with moderate to severe acne. Denifanstat is a once-daily oral small molecule fatty acid synthase (FASN) inhibitor being developed by Ascletis as ASC40 for acne in China and by Sagimet for MASH in the rest of the world. Sagimet has granted an exclusive license to denifanstat for China to Ascletis Bioscience Co. Ltd. (Ascletis), of which Ascletis Pharma Inc. is the parent company.

"The topline results from Ascletis' Phase 3 open-label acne trial in China build additional confidence in the clinical potential of FASN inhibition in acne," said David Happel, Chief Executive Officer of Sagimet. "These results demonstrate FASN inhibition's potential as a novel mechanism of action for the treatment of acne."

In June 2025, Ascletis announced that denifanstat (ASC40) met all primary, key secondary, and secondary endpoints in a 480-patient randomized, double-blind, placebo-controlled Phase 3 clinical trial (NCT06192264, ASC40-303) for the treatment of moderate to severe acne vulgaris (press release here).

"Following the 12-week data from the Phase 3 randomized double-blind denifanstat trial in moderate to severe acne patients, results from the 40-week open-label study are even more encouraging," said Dr. Neal Bhatia, Director of Clinical Dermatology at Therapeutics Clinical Research in San Diego and former Vice President of the American Academy of Dermatology. "For moderate to severe acne patients, who are currently underserved by older agents, the potential of a new therapeutic option would be a welcome addition to the current treatment armamentarium."

Clinical Results

The Phase 3 multi-center open-label clinical trial ASC40-304 (NCT06248008) was designed to determine the long-term safety of denifanstat in patients with moderate to severe acne vulgaris who were previously enrolled in the double-blind, randomized, placebo-controlled 12-week Phase 3 ASC40-303 trial. This open-label Phase 3 trial enrolled 240 subjects that received oral denifanstat 50 mg once daily for up to 40 weeks. Subjects who were originally randomized to denifanstat in ASC40-303 trial had a total of 52 weeks of denifanstat exposure.

Primary endpoints evaluated safety, and secondary endpoints evaluated efficacy, for up to 52 weeks of denifanstat treatment. Denifanstat was generally well tolerated, with the following:

- **Treatment-emergent adverse events (TEAEs):** Only two categories of TEAEs had an incidence rate of 5% or more, with dry eye syndrome in 5.5% of denifanstat-treated subjects and dry skin reported in 5.2% of denifanstat-treated subjects.
- **Adverse events (AEs):** All denifanstat-related AEs were mild or moderate; no denifanstat-related Grade 3 or 4 AEs; no AE-related permanent discontinuations; Grade 1 hair thinning in the study was experienced by only 1 denifanstat-treated patient (which resolved within eight weeks while remaining in study without a change in dose); no deaths were reported.
- **Serious adverse events (SAEs):** No denifanstat-related SAEs; 2 non-denifanstat-related SAEs (1 breast lump, 1 contusion), both resolved.

Subjects treated with denifanstat showed improvements in all efficacy endpoints (secondary endpoints of the trial) beyond those observed at 12 weeks. The endpoints were the number of subjects with an IGA[1] score decrease by at least 2 points, the number of subjects dropping from an IGA score of 3 down to 0 or 1, the percentage reduction in total skin lesion count, and the percentage reduction in inflammatory skin lesion count. Data are planned to be shared in upcoming congresses and publications.

1. Investigator's Global Assessment (IGA) score. IGA score 0: clear; IGA score 1: almost clear

About Sagimet Biosciences

Sagimet is a clinical-stage biopharmaceutical company developing novel FASN inhibitors designed to target dysfunctional metabolic and fibrotic pathways in conditions resulting from the overproduction of the fatty acid, palmitate. Denifanstat, an oral, once-daily pill, met all primary endpoints in its Phase 2b FASCINATE-2 clinical trial in MASH as well as all primary and secondary endpoints in Sagimet's license partner for China's Phase 3 clinical trial in moderate-to-severe acne. A combination of denifanstat and resmetirom was tested in a Phase 1 PK clinical trial and is planned to be developed for patients with MASH cirrhosis (F4). TVB-3567, a second oral FASN inhibitor which is planned to be developed for acne, is currently being tested in a Phase 1 first-in-human clinical trial. For additional information about Sagimet, please visit www.sagimet.com.

About FASN Inhibition and Acne

Over 50 million people suffer from acne in the U.S., with 5.1 million acne patients treated by dermatologists annually, making it one of the most prevalent skin diseases addressed by physicians.[1,2] There is no cure for acne; and due to its pathology, most patients require chronic management and multiple annual courses of treatment for flare control. Adherence to topical therapies is lower than with oral agents, with an estimated 30% to 40% of patients not adhering to their topical treatments.[3]

Patients with acne vulgaris have increased sebum production compared to non-acne populations which contributes to the pathogenesis of the disease. Increased sebum production is due to increased de novo lipogenesis (DNL) locally in the sebocytes. FASN is the last committed step in the DNL pathway which produces the majority (>80%) of key sebum lipids such as palmitate and sapienic acid in acne, and FASN also contributes to inflammatory pathways, making the inhibition of FASN a potentially impactful approach to address acne.

1. Bickers DR, et al. *J Am Acad Dermatol*. 2006;55(3):490-500.
2. American Academy of Dermatology. Burden of Skin Disease. 2017. www.aad.org/BSD.
3. Purvis CG, Balogh EA, Feldman SR. Clascoterone: How the Novel Androgen Receptor Inhibitor Fits Into the Acne Treatment Paradigm. Ann Pharmacother. 2021;55(10):1297-1299. doi:10.1177/1060028021992055.

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 timelines and anticipated development milestones, Sagimet's cash and financial resources and expected cash runway are forward-looking statements. 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, TVB-3567 or any other drug candidates or combination therapies developed by Sagimet; Sagimet's ability to advance drug candidates into and successfully complete clinical trials within anticipated timelines; 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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Exhibit 99.2


SAGIMET
BIOSCIENCES
Targeting Metabolic Dysfunction with Novel Therapies to Treat MASH, Acne & Cancer
February 2026

Forward-Looking Statements and Disclaimer

This presentation contains forward-looking statements within the meaning of, and made pursuant to the safe harbor provisions of, The Private Securities Litig Reform Act of 1995. All statements contained in this document, other than statements of historical facts or statements that relate to present facts or cu conditions, including but not limited to, statements regarding possible or assumed future results of operations, business strategies, research and develop plans, regulatory activities, the presentation of data from clinical trials, Sagimet's clinical development plans and related timelines and anticipated cl development milestones, market opportunity, competitive position and potential growth opportunities are forward-looking statements. These statements in known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially diff from any future results, performance or achievements expressed or implied by the forward-looking statements. In some cases, you can identify forward-lo statements by terms such as "may," "will," "should," "would," "expect," "plan," "anticipate," "could," "intend," "target," "project," "believe," "estimate," "pre "potential," or "continue" or the negative of these terms or other similar expressions. The forward-looking statements in this presentation are only predict These forward-looking statements speak only as of the date of this presentation and are subject to a number of risks, uncertainties and assumptions, sor which cannot be predicted or quantified and some of which are beyond our control, including, among others: the clinical development and therapeutic pote of denifanstat, TVB-3567 or any other drug candidates or combination therapies developed by Sagimet; our ability to advance drug candidates into 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 t our ability to advance drug candidates into and successfully complete clinical trials within anticipated timelines; that unfavorable new clinical trial data emerge in other clinical trials of our product candidates; that clinical trial data are subject to differing interpretations and assessments, including by regul authorities; our relationship with Ascletis, and the success of its development efforts for denifanstat; the accuracy of our estimates regarding our ca requirements; and our ability to maintain and successfully enforce adequate intellectual property protection. These and other risks and uncertainties described more fully in the "Risk Factors" section of our most recent filings with the Securities and Exchange Commission (SEC) and available at www.sec.gov should not rely on these forward-looking statements as predictions of future events. The events and circumstances reflected in our forward-looking statem 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 possibl 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 revis 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 Experie

Dave Happel *President & CEO*

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

Brought multiple innovative healthcare products to the market

Elizabeth Rozek *Chief Legal & Administrative Of*

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

Thierry Chauche *CFO*

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

Marie O'Farrell *Chief Scientific Officer*

>20 years of experience in R&D and translational medici biopharma and biotech

Successfully guided development for multiple clinical p

Eduardo Martins *Chief Medical Officer*

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

Led clinical development team of cenicriviroc for MASH

Rob D'Urso *Senior Vice President, New Product*

>20 years of US and global leadership experience in dermatology

Pfizer raptor pharmaceuticals MedImmune GILEAD DYNAVAX ALEXION Intercept JUSTICE

HORIZON AstraZeneca abbvie Genentech proventionbio NOVARTIS cognoa basilea

Sagimet at a Glance

Unique MOA: FASN Inhibition

- Our lead molecule, denifanstat, is a novel fatty acid synthase (FASN) inhibitor with a differentiated MOA with the potential to target multiple underserved diseases
- Strong clinical data demonstrates denifanstat's proof of concept across multiple disease states

Denifanstat in MASH

- Denifanstat directly targets the 3 key drivers of MASH (metabolic dysfunction-associated steatohepatiti – liver fat, inflammation, and fibrosis
- Successful outcome of Phase 2b trial; met both primary endpoints with significant reduction in fibrosis
- Pre-clinical data demonstrated synergistic effect of combination of FASN inhibitor and resmetirom
- Phase 1 pharmacokinetics (PK) clinical trial of a combination of denifanstat and resmetirom completed in December 2025, Phase 2 clinical combination trial with denifanstat and resmetirom in patients with MASH cirrhosis (F4) planned to initiate in 2H 2026

TVB-3567 in Acne

- Our follow-on FASN inhibitor, TVB 3567, received Investigational New Drug (IND) clearance in March 2
- First-in-human Phase 1 clinical trial initiated in June 2025 for development of an acne indication

Strong IP, Cash Position, and Collaboration Potential

Strategic Collaboration with Ascletis in Acne

- Denifanstat met all primary and secondary endpoints in Phase 3 clinical trial in patients with moderate to severe acne vulgaris in China conducted by license partner for China, Ascletis
- Denifanstat was well-tolerated in Ascletis' open-label Phase 3 clinical trial
- Ascletis announced that Denifanstat NDA for the treatment of moderate to severe acne was accepte by China NMPA in December 2025

IP Portfolio

- Denifanstat:
  - Method of use patent—2036; potential PTE to 2041
  - Composition of matter patent—2032
- Combination of denifanstat and resmetirom:
  - Application filed 2024; if granted—2044; potential PTE to 2048
- TVB-3567:
  - Method of use application for TVB-3567 for acne filed 2025; if granted—2046
  - Composition of matter patent—2035; potential PTE to 2038

Cash Position

- Nasdaq: SGMT; $125.5M cash on hand*, expected to fund current operations for 2 years

*Cash, cash equivalents and marketable securities as of 09/30/2025

Development Pipeline: Multiple Indications and Clinical Milestones

Therapeutic Area	Indication	Stage of Development				Expected Milestone / Status
		Preclinical	Phase 1	Phase 2	Phase 3	
Metabolic Disease	MASH	Denifanstat				Phase 2b met histology primary and multipl secondary endpoints, data announced 1Q2 FDA Breakthrough Therapy designation; Pha F2/F3 ready
		Denifanstat				Phase 1 hepatic impairment results reported 1Q2024
		Denifanstat/resmetirom				Phase 1 clinical PK trial completed in Dece 2025
Dermatology	Acne	TVB-3567				Phase 1 FIH initiated in June 2025
		Denifanstat (ASC40)				Phase 3 met all primary and secondary endp data announced June 2025; NDA accepted b NMPA in December 2025*
Oncology	Solid tumors	TVB-3567				Identifying FASN-dependent tumor types fo potential FASN inhibitor development
		Denifanstat				

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

MASH: A Burgeoning Epidemic

Estimated Patients in 2030[1]

United States

MASLD	MASH	MASH mod-adv Fibrosis F2-F3	MASH Cirrhosis F4	Hepatocellular carcinoma
100.9 million	**27.0 million**	**10.6 million**	**3.5 million** compensated and decompensated	**25 thousand** annual cases among MASLD population
Metabolic Dysfunction-Associated Liver Disease	Metabolic Dysfunction-Associated Steatohepatitis			

MASH

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

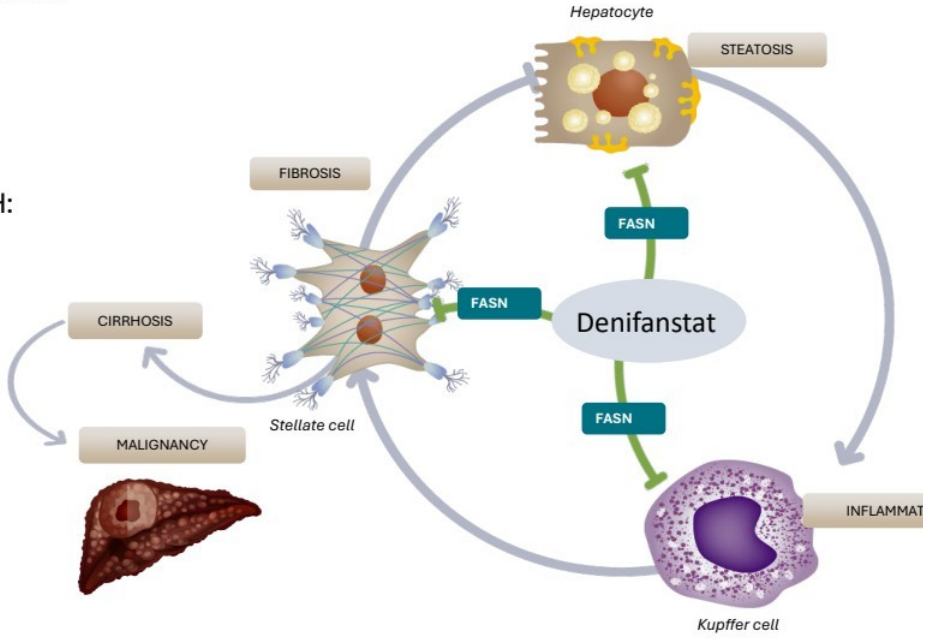
Note: MASH, or metabolic dysfunction-associated steatohepatitis, was formerly known as NASH, or nonalcoholic steatohepatitis.

1. Estes C, et al. *J Hepatol.* 2018;69(4):896-904.

FASN Inhibition Addresses Three Independent Mechanisms of MASH Development and Progression

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

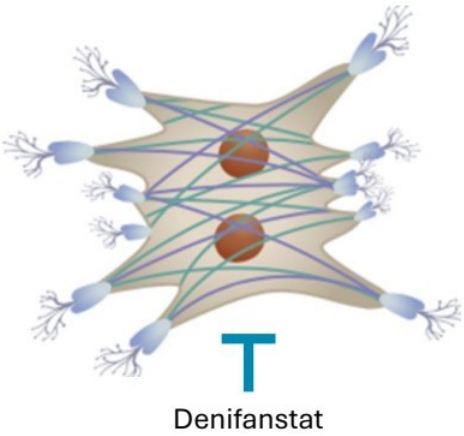
1. Blocking **steatosis** via inhibiting de novo lipogenesis in hepatocytes
2. Reducing **inflammation** via preventing immune cell activation
3. Blunting **fibrosis** via inhibiting stellate cell activation



FASN Inhibition Directly Blocks Human Liver Stellate Cell Function

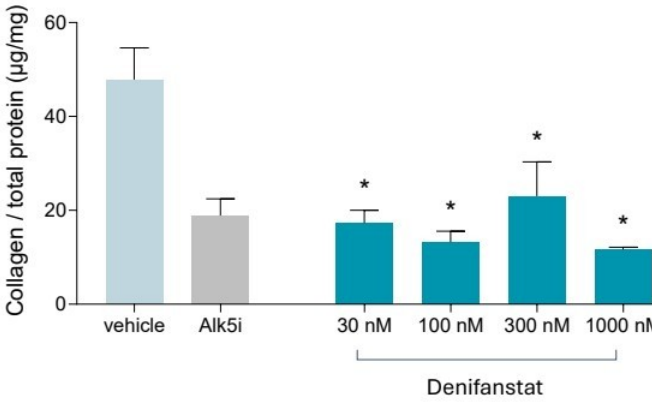
Stellate cells require DNL for fibrogenesis

Denifanstat blocks stellate cell activation



Denifanstat directly inhibits fibrogenic activ

Primary human stellate cell assay

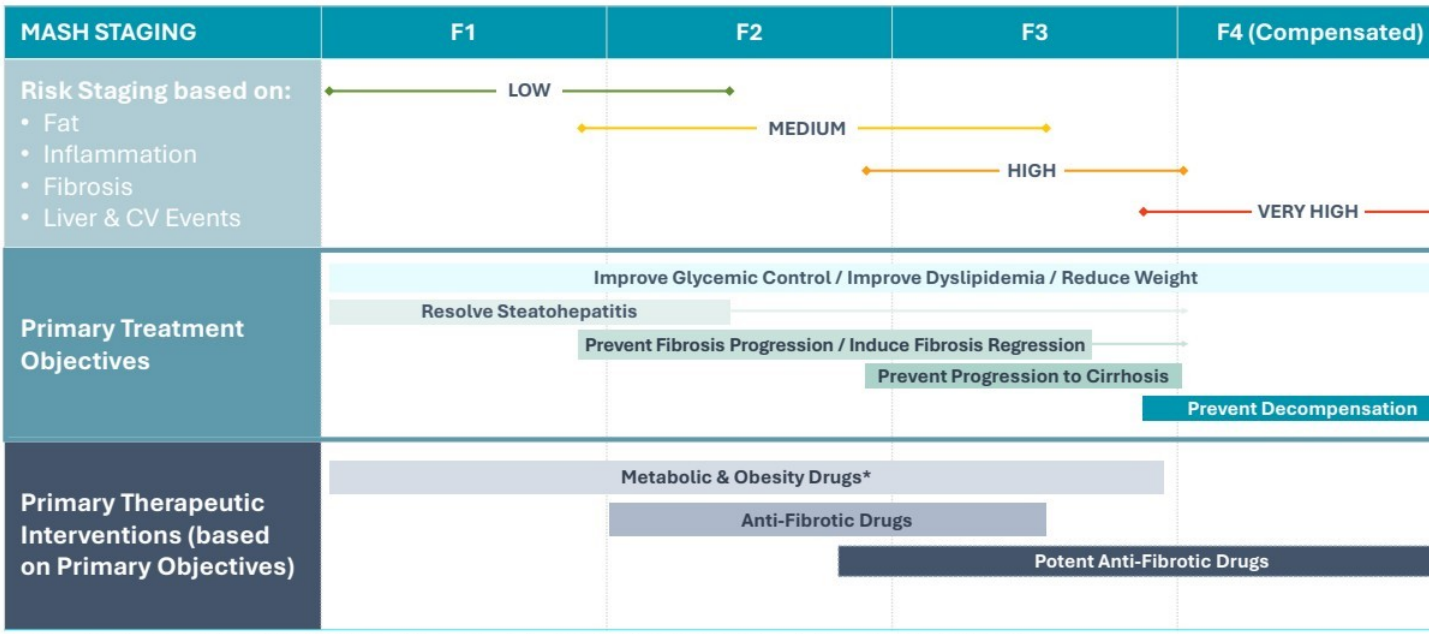


- Stimulated by TGF-beta to activate fibrogenesis
- Denifanstat showed similar inhibition to positive cont ALK5 inhibitor

*p<0.05. DNL: de novo lipogenesis

1. O'Farrell M, et al. *Sci Rep*. 2022;12(1):15661. Sagimet Biosciences data on file.

Treatment Goals for MASH Across Fibrosis Staging



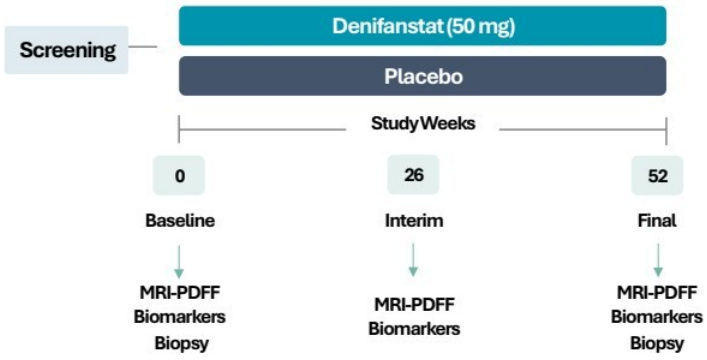
Cusi K, et al. *Endocr Pract.* 2022;28(5):528-562. Rinella ME, et al. *Hepatology.* 2023;77(5):1797-1835. EASL, et al. *J Hepatol.* 2024;81(3):492-542.
*Metabolic drugs are anticipated to be background therapy for obesity and type 2 diabetes, and earlier stages of fibrosis

Strong MASH Data Creates Opportunities to Reach Advanced Patient Populations

SAGIMET
BIOSCIENCES

FASCINATE-2: Biopsy Trial Design Focused on Histological Endpoints

FASCINATE-2 Phase 2b trial design



- Biopsy confirmed F2-F3 MASH patients (n=168)
- 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.

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100

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

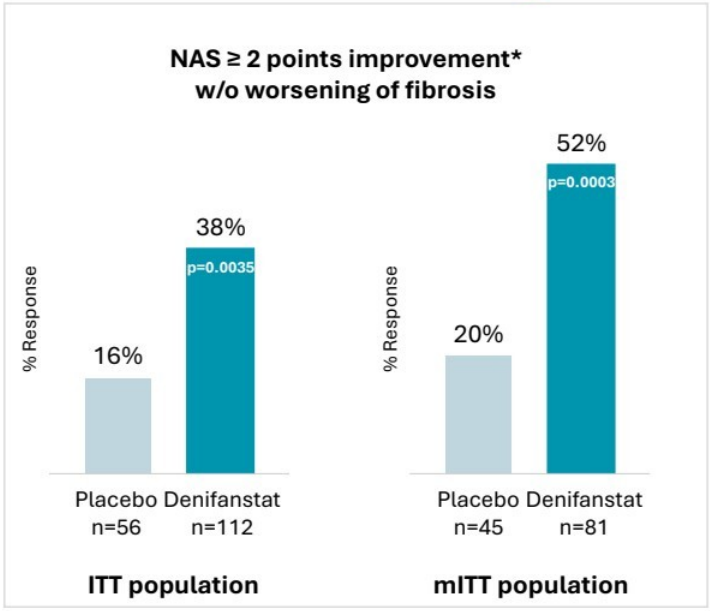
Parameter	Placebo, n=45	Denifanstat, n=81
Age, years	59.6 (+/- 10.9)	56.1 (+/- 10.8)
Sex, female	27 (60%)	48 (59%)
Race, White	41 (91%)	73 (90%)
Ethnicity, Hispanic or Latino	15 (33%)	27 (33%)
BMI, kg/m^2	36.5 (+/- 6.7)	34.6 (+/- 6.1)
Type 2 diabetes	27 (60%)	55 (68%)
ALT (alanine aminotransferase) U/L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

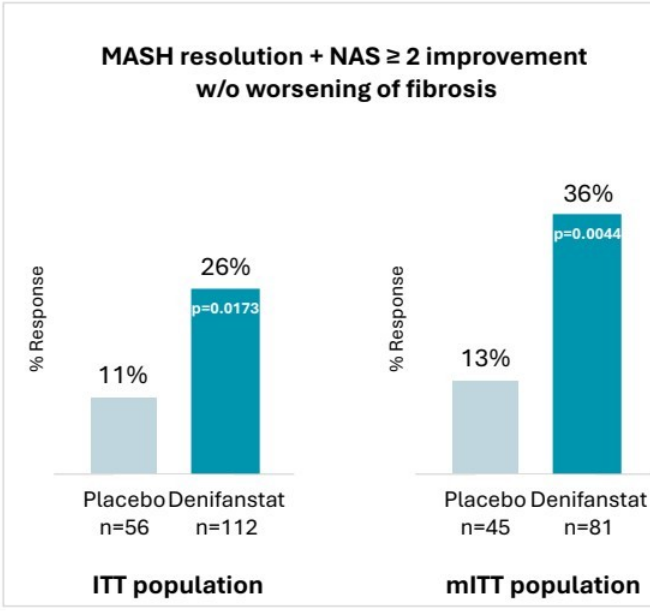
Modified intent-to-treat population (mITT) includes all patients with paired biopsies. Data are mean (SD) or n (%)

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100

Primary Endpoints: Liver Biopsy

Denifanstat Achieved Statistical Significance at Week 52



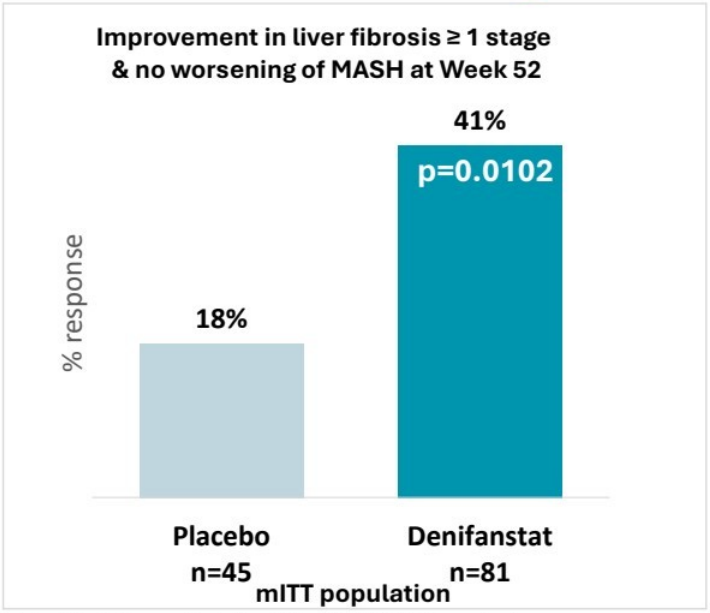


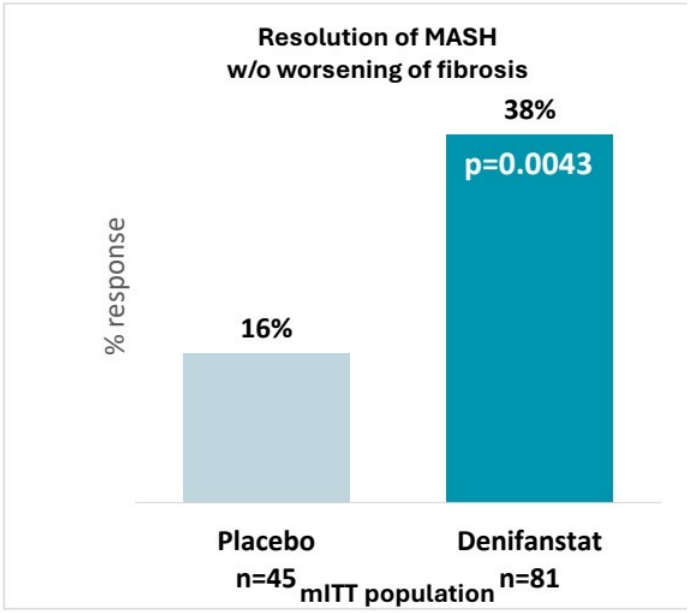
Cochran-Mantel-Haenszel Test – two sided at the 0.05 significance level. * ≥1-point improvement in ballooning or inflammation.

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100

Secondary Endpoints: Liver Fibrosis and MASH Resolution

Denifanstat Achieved Statistical Significance at Week 52





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

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100

Secondary Endpoints: Liver Fibrosis

Denifanstat Achieved Statistically Significant Improvement of Fibrosis

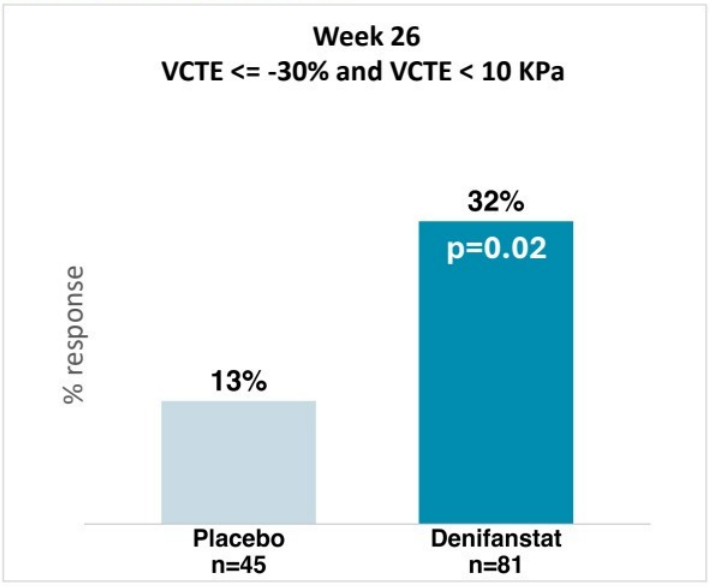
Fibrosis Endpoints	Subgroup	Placebo	Denifanstat	p-value
≥1 stage improvement in fibrosis w/o worsening of MASH	ITT	14%	30%	0.040**
	mITT	18%	41%	0.0102**
	F3	13%	49%	0.0032**
≥2 stage improvement in fibrosis w/o worsening of MASH	mITT	2%	20%	0.0065**
	F3	4%	34%	0.0065**
Progression to MASH cirrhosis (F4)	mITT	11%	5%	0.0386*

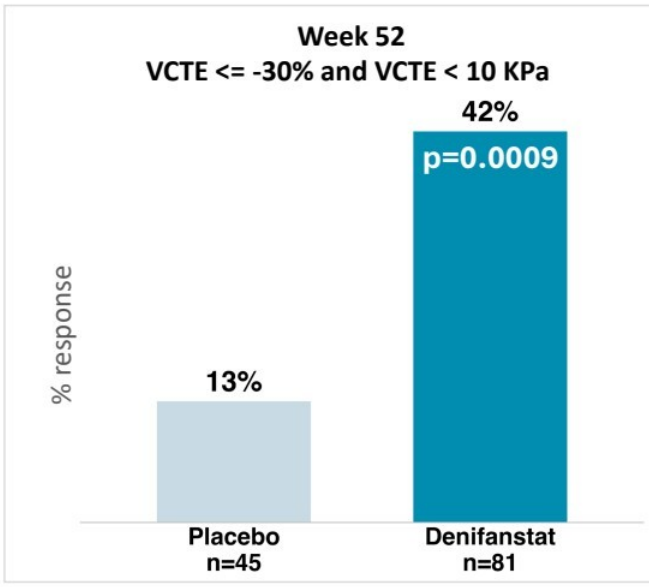
*One sided at the 0.05 significance level, **Two sided at the 0.05 significance level

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100.
Loomba R, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/Denifanstat_a_fatty_acid_synthase_FASN_inhibitor_shows_significant_fibrosis_improvement_and_MASH_resolution_in_FASCINATE-2_a_Ph2b

Denifanstat Achieved Statistically Significant VCTE Improvement at Weeks 2 and 52

FASCINATE-2 Phase 2b



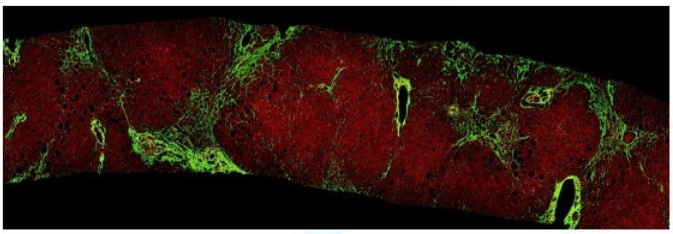


Sagimet Biosciences data on file. FASCINATE-2 posthoc analysis. mITT population. Chi-square test. VCTE: Vibration-controlled transient elastography. VCTE <=30% means magnitude of decline fro

Additional Fibrosis Analysis Using AI-based Digital Pathology

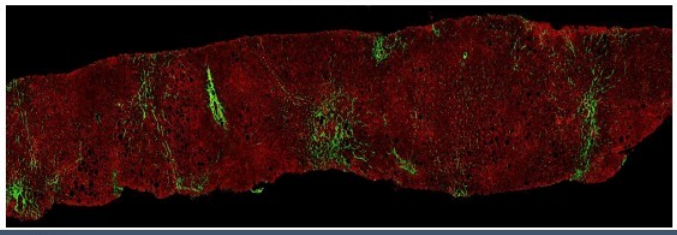
Digital Imaging Showed that Denifanstat Significantly Reduced Fibrosis in Advanced Patients

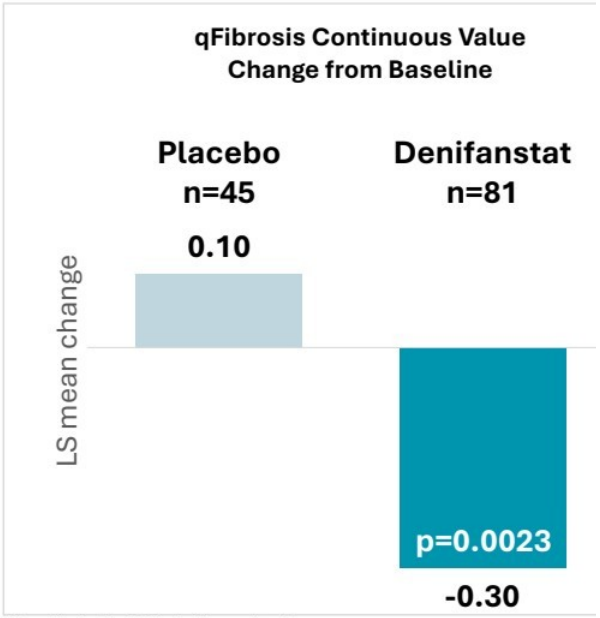
Pre-Treatment Pt A
NASH-CRN Fibrosis stage F3



Denifanstat

Post-Treatment Pt A
NASH-CRN Fibrosis stage F1

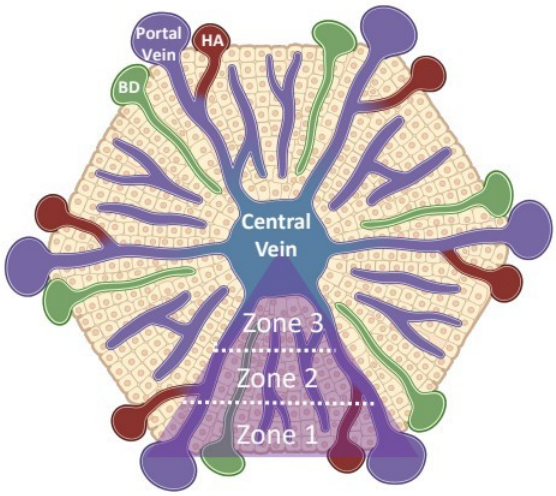




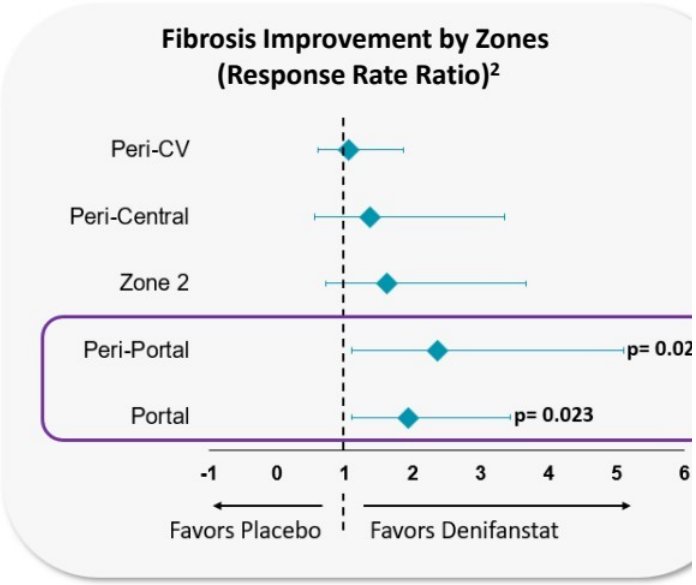
*One sided at the 0.05 significance level

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100

qFibrosis Zonal Analysis Demonstrated that Denifanstat Improves Parameters Linked to Liver Outcomes



Changes in periportal and portal zones have been correlated with liver outcomes and mortality by analysis of liver biopsies (n=452) from SteatoSITE study[1]



Response at the individual zonal parameter level was defined as "at least" 30% relative decrease f

1. Kendall TJ, et al. *Liver Int.* 2024;44(10):2511-2516.
2. Rinella M, et al. Presented at: AASLD 2024; November 15-19, 2024; San Diego, CA. Abstract 017

Denifanstat Potential in Patients with MASH Cirrhosis (F4)

Differentiated Mechanism of Action

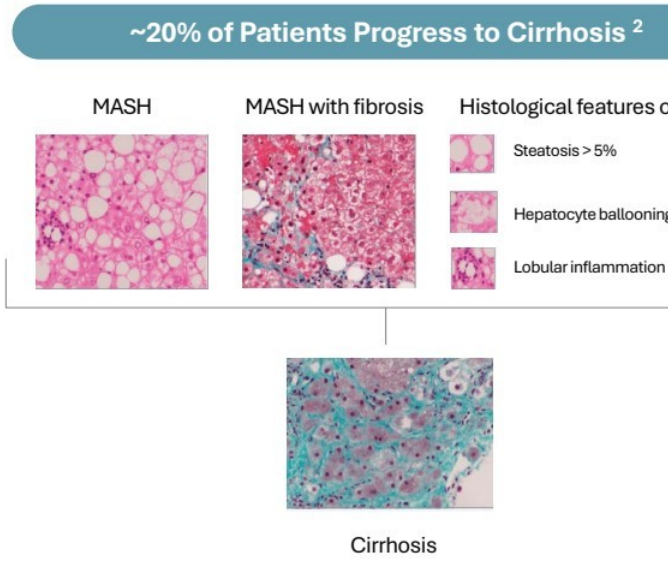
- *In vitro* data demonstrates that denifanstat reduces pro-fibrotic signaling in stellate cells, suggesting that denifanstat has the potential to remove fibrotic scar tissue and reestablish the basal extracellular matrix (ECM) scaffold even in patients with MASH cirrhosis (F4)[1]
- Hepatocytes continue to be functional, and patients frequently have increased liver fat

Clinical Data

- PK profiles in patients with MASH cirrhosis (F4) in the Phase 1 impaired hepatic function study[3]
- Positive impact on advanced fibrosis in patients in FASCINATE-2[4], including qF4 (quantification of fibrosis stage 4) patients based on AI-based digital pathology [5]

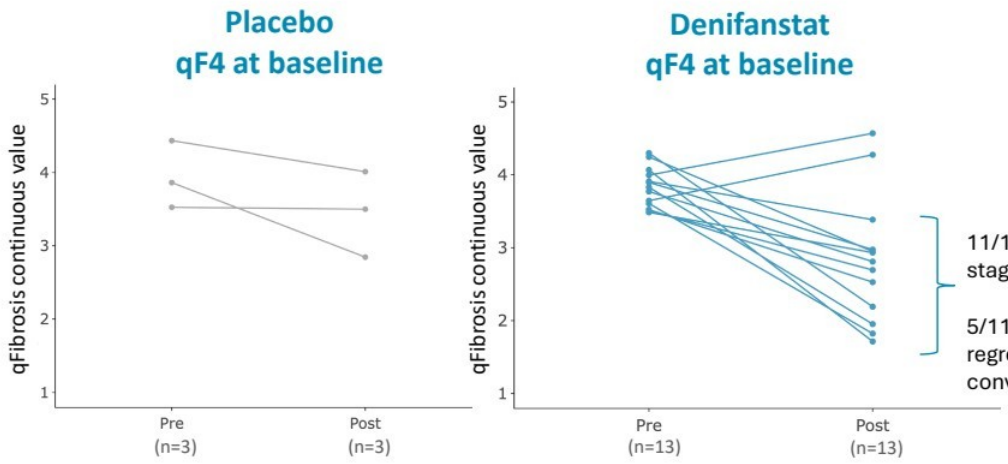
Next Step

- Potential Phase 2 proof of concept in patients with MASH cirrhosis (F4)



1. Kamm DR, McCommis KS. *J Physiol.* 2022;600(8):1825-1837. 2. Sheka AC, et al. *JAMA.* 2020;323(12):1175-1183. 3. Sagimet Biosciences data on file. CLIN-009. 4. Loomba R, et al. *Lancet Gastroenterol Hepatol.* 2024;9(12):1090-1100. 5. Sagimet Biosciences data on file. FASCINATE-2 HistoIndex.

85% of qF4 Patients on Denifanstat Showed 1 to 2-Stage Reductions in Fibros



11/13 (85%) qF4 patients decreased by 1 or 2 qfil stages measured by AI-based pathology

5/11 of qF4 patients showed ≥1 stage fibrosis regression, with 4 of these being 2-stage, measu conventional pathology

- AI may detect fibrosis regression at an earlier point in time, compared to conventional pathology
- qF4 population (defined on AI platform by HistoIndex) are likely the most advanced subgroup of F3 patients in Phase 2b study

Sagimet Biosciences data on file. FASCINATE-2 HistoIndex.

FASCINATE-2: Safety

Denifanstat Was Generally Well-Tolerated

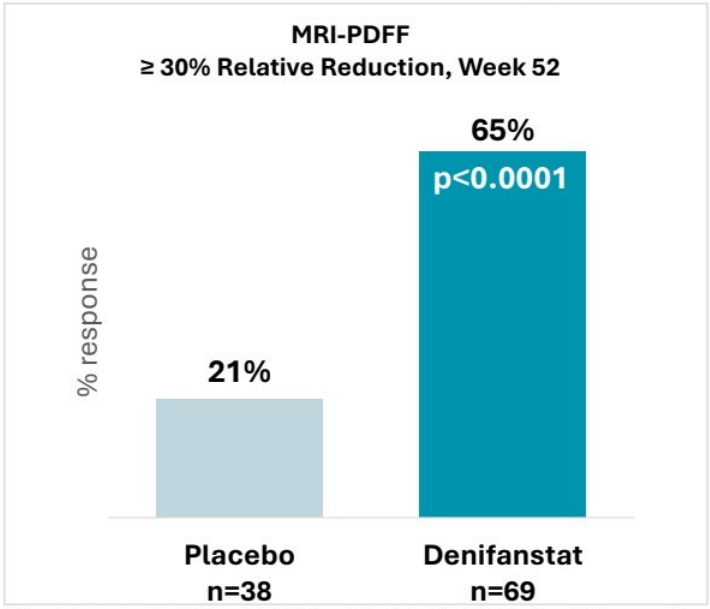
Event n (%)	Placebo (n=56)	Denifanstat 50mg (n=112)	Total (n=168)
Any adverse event (AE)	46 (82.1)	99 (88.4)	145 (86.3)
Adverse event related to denifanstat or placebo	20 (35.7)	51 (45.5)	71 (42.3)
Serious adverse event	3 (5.4)	13 (11.6)	16 (9·5)
TEAE leading to study drug discontinuation	3 (5.4)	22 (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 (drug-induced liver injury) signal and no muscle wasting were detected, and GI (gastrointestinal) effects were comparable to placeb
- AE of hair thinning stabilized with a 2-to-4 week dose pause and then reversed with down titration or study completion
  - Only 7% of patients discontinued from the study with treatment-related hair thinning. Hair thinning in patients receiving GLP-1 ranges 7% to 10% [1,2]
  - In two previous clinical studies of denifanstat, 2% of the patients on denifanstat experienced hair thinning at 50mg [3]

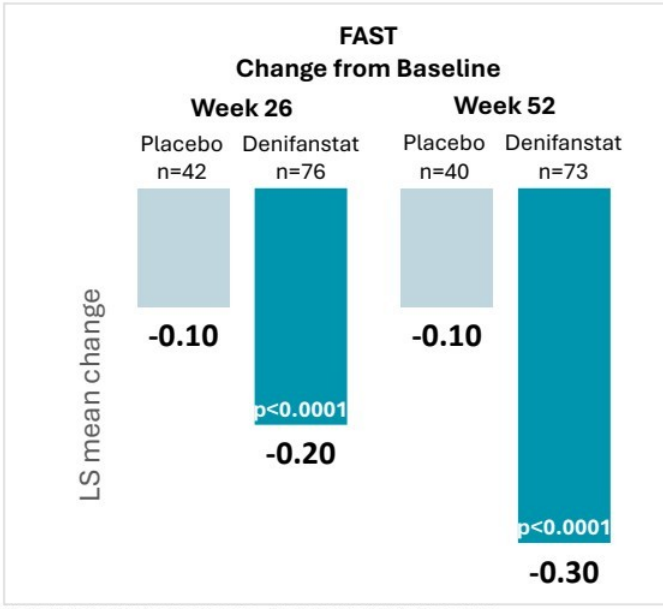
1. Wadden TA, et al. *Nat Med*. 2023;29(11):2909-2918. 2. Daniel S, et al. *J Drugs Dermatol*. 2025;24(4):413-415. 3. Sagimet Biosciences data on file. FASCINATE-1. Phase 2a study of denifanstat in acne by Ascletis in China
Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100

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.

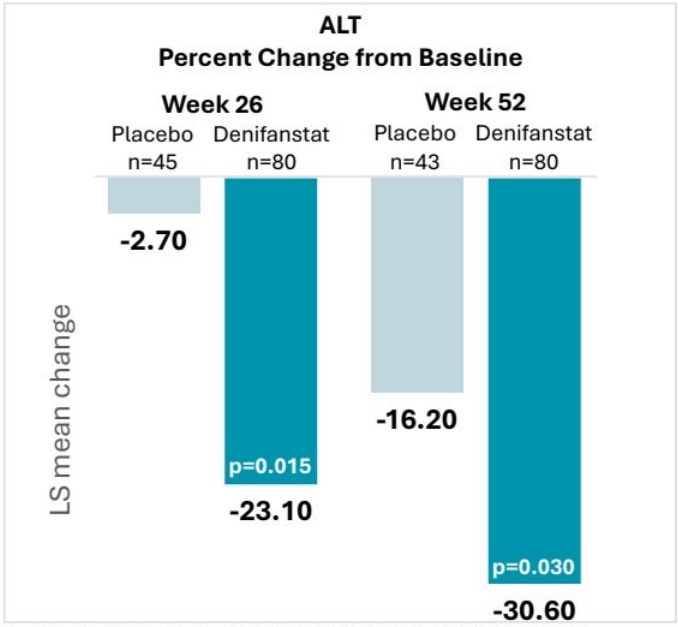


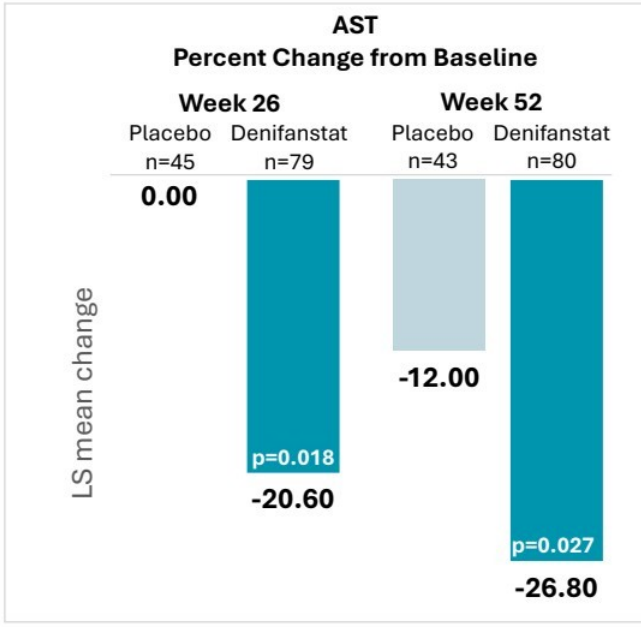
Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. mITT population.

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100. Sagimet Biosciences data on file.

Secondary Endpoints: Liver Enzymes

Denifanstat Decreased ALT and AST Levels



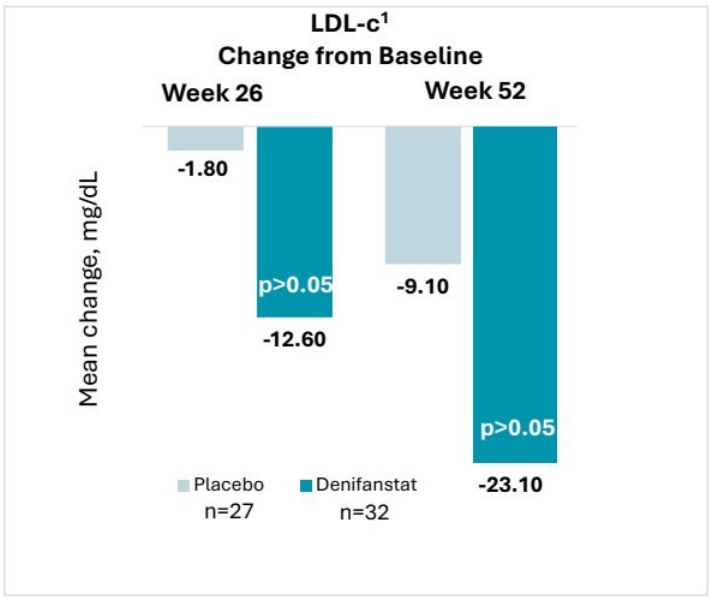


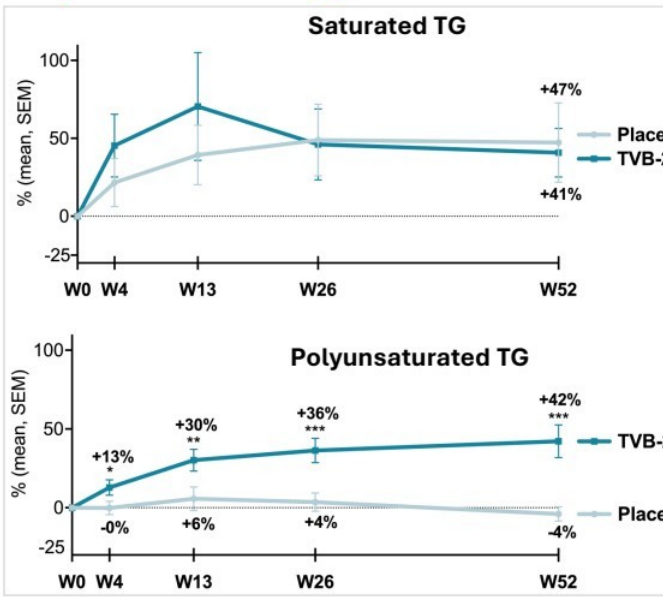
Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. mITT population

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100. Sagimet Biosciences data on file.

Cardiometabolic Health

Denifanstat Decreased LDL-c Levels and Increased Polyunsaturated Triglycerides

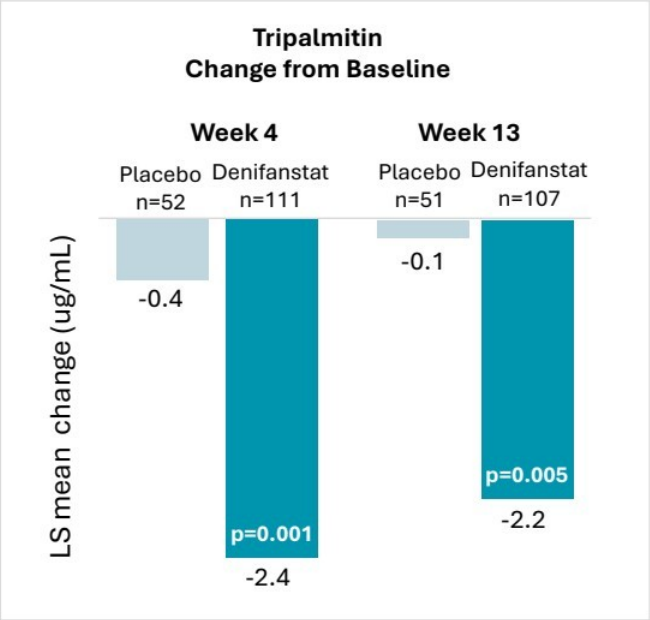




mITT population. Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. *p<0.05, **p<0.01, ***p<0.001. [1]For LDL-c, baseline > 100 mg/dL.

Loomba R, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/Denifanstat_a_fatty_acid_synthase_FASN_inhibitor_shows_significant_fibrosis_improvement_and_MASH_resolution_in_FASCINATE-2_a_Ph2b
Sagimet Biosciences data on file.

Denifanstat Reduced De Novo Lipogenesis



Two sided at the 0.05 significance level, ITT population

Tripalmitin:

- A saturated triglyceride which is a biomarker of DNL inhibition
- Reduced by denifanstat as early as week 4 of treatment

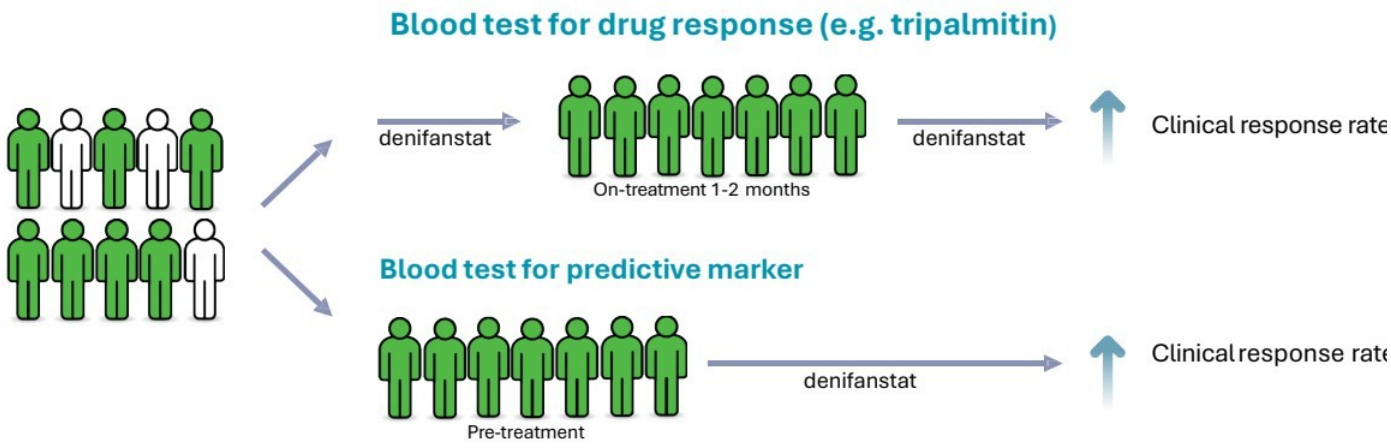
Next steps

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

Loomba R, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/Denifanstat_a_fatty_acid_synthase_FASN_inhibitor_shows_significant_fibrosis_improvement_and_MASH_resolution_in_FASCINATE-2_a_Ph2b
Sagimet Biosciences data on file.

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]



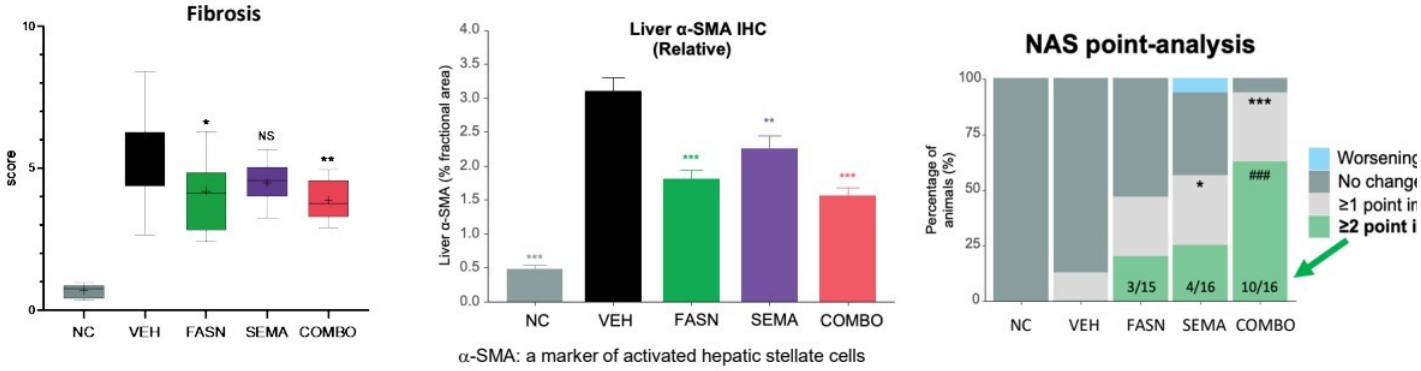
1. Signature has 6 metabolites: ursodeoxycholic acid, DL-2-aminocaprylic acid, sarcosine, glycoursodeoxycholic acid, D(-)-2-aminobutyric acid, PC(0-18:0/22:4). Accuracy 79%, PPV 88%, NPV 63%. Sagimet Biosciences data on file.

Combination Therapy Development Program for MASH

SAGIMET BIOSCIENCES

Combination of FASN Inhibitor and Semaglutide Improved Histological Features in MASH Mouse Model

In a mouse model, combination treatment showed: 1) an additive effect on fibrosis reduction, 2) a direct impact on stellate cells, and 3) a synergistic effect on NAS reduction



α-SMA: a marker of activated hepatic stellate cells

* p <0.05, ** p<0.01, *** p<0.001, ### p<0.001

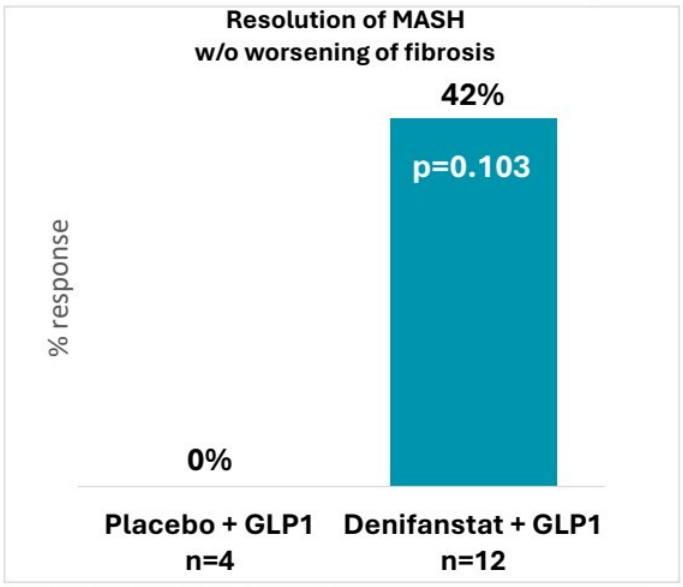
GUBRA DIO MASH mice. PSR images were analyzed by FibroNest (PharmaNest), all scores shown with parenchymal correction

NS: not significant; NC: Normal chow diet control, VEH: MASH vehicle control, FASN: TVB-3664 (FASN inhibitor), SEMA: semaglutide, COMBO: TVB-3664/semaglutide

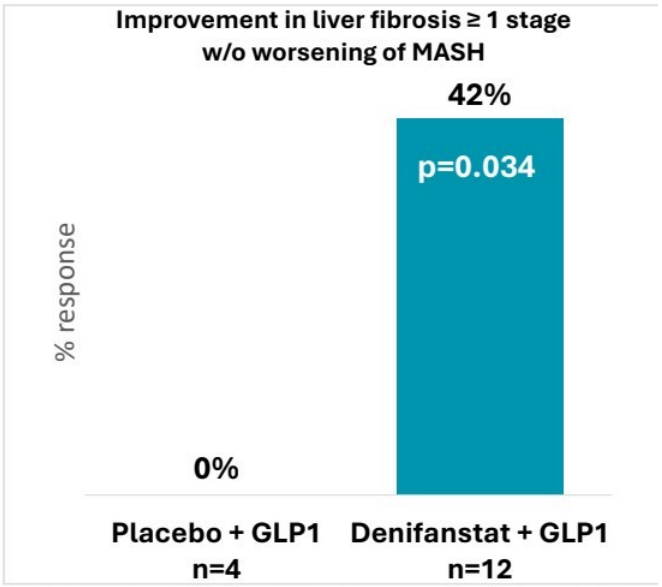
Tsai WW, et al. Presented at: AASLD 2023; November 10-14, 2023; Boston, MA. Abstract 2400-C.

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

FASCINATE-2 Phase 2b - 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

Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100.
Loomba R, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/Denifanstat_a_fatty_acid_synthase_FASN_inhibitor_shows_significant_fibrosis_improvement_and_MASH_resolution_in_FASCINATE-2_a_Ph2b

Mechanism of Action Supports Combination Therapy Opportunity

Potential improved clinical outcome for patients with combination therapy of denifanstat, a fat synthesis inhibitor + a fat oxidizer (THR-beta agonist)

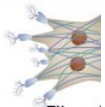
Combination therapy potential:

- Denifanstat MOA complementary to other MOAs – THR-beta, GLPs
- Opportunity for fixed dose combinations with other oral medications

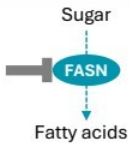
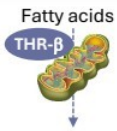
Hypothesis: distinct and complementary mechanisms of the combination lead to synergistic effect

Preclinical combination studies in mouse n showed beneficial impact of FASN inhibitor resmetirom combination on histology and N biomarkers[1]

Liver fat (hepatocyte)



Fibrosi (stellate

Denifanstat Reduces de novo Lipogenesis		**Direct** - decreases de novo lipogenesis	**Direct** – decre fibrogenesis i cells, liver fat lipotoxicity
Resmetirom Increases mitochondrial beta-oxidation		**Direct** - increases fatty acid oxidation and improves mitochondrial function	**Indirect** – dec fibrosis due to decreased live lipotoxicity

MOA- Mechanism of Action

1. Tsai WW, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/2024-EASL-poster-GAN-model-final.pdf
Tsai WW, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/2024-EASL-poster-TNO-model-final.pdf

Potential Benefits of Combination Therapy in Advanced MASH Patients

A combination product could potentially offer an opportunity to serve patient groups with the strongest need of treatment, including those with stage 4 fibrosis

Characteristic	Denifanstat [1]	Resmetirom [2]	Potential Combination
Mechanism	**Direct** – decreases de novo lipogenesis **Direct** – decreases fibrogenesis in stellate cells, liver fat and lipotoxicity	**Direct** – increases fatty acid oxidation **Indirect** – decreases fibrosis due to decreased liver fat and lipotoxicity	Potential synergies in the MOA Note: THR-beta upregulates FA
Formulation	Oral	Oral	Oral
Dosing	Once daily	Once daily	Once daily Fixed-Dose Combination (FD
Clinical Data	Met both primary endpoints in Phase 2b trial with significant reduction in fibrosis	Phase 3 data supported FDA approval for treatment of non-cirrhotic MASH	Potential synergistic effect

Note: These data are placebo-adjusted, derived from different clinical trials at different points in time, with differences in trial design and patient populations. As a result, cross-trial comparisons canno and no head-to-head clinical trials have been conducted.

1. Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100. 2. Harrison SA, et al. *N Engl J Med*. 2024;390(6):497-509.

Clinical Development Program for Denifanstat and Resmetirom Combination

Phase 1 pharmacokinetics (PK) trial for combination of denifanstat and resmetirom completed in December 2025

- Combination of denifanstat and resmetirom was generally well-tolerated over the duration of the study, with no safety signa
- No Serious Adverse Events (SAEs), no clinically significant laboratory results, and no treatment discontinuations

Subject to consultation with regulatory authorities, Phase 2 clinical trial with denifanstat and resmetirom in patie with MASH cirrhosis (F4) planned to initiate in 2H 2026

- Phase 2 proof-of-concept efficacy trial in patients with MASH cirrhosis (F4), for which there are no approved treatments
  - Potential clinical trial design, to be discussed with FDA:
    - 4-5 arms including monotherapy of each agent and up to two combination arms, versus placebo
    - At least 52 weeks combination treatment (up to 96 weeks) with interim readout planned at 52 weeks
    - Main efficacy endpoints: fibrosis improvement in liver biopsies and non-invasive markers of fibrosis
    - Non-invasive tests (NITs) for early readout to evaluate impact of the combination
    - Enrollment estimated between 12 and 18 months

Attractiveness of a Potential Denifanstat/Resmetirom Combination

Denifanstat in MASH

- Denifanstat directly targets the 3 key drivers of MASH (metabolic dysfunction-associated steatohepatiti – liver fat, inflammation, and fibrosis
- Successful outcome of Phase 2b trial; met both primary endpoints with significant reduction in fibrosis

Potential of a Fixed Dose Combination

- Pre-clinical data demonstrated synergistic effect of combination of FASN inhibitor and resmetirom [2]
- Combination of a Phase 3-ready drug candidate with the first drug approved for MASH
- IP for the combination of denifanstat and resmetirom:
  - Application filed 2024; if granted—2044; potential PTE to 2048
- Potential oral, once-daily product
- Potential to address an unmet need in patients with MASH cirrhosis (F4)

Development Program

- Phase 1 PK clinical trial of a combination of denifanstat and resmetirom completed in December 2025
- Phase 2 trial of denifanstat/resmetirom combination in F4 MASH patients is planned to initiate in 2H 20
- Global license agreement with TAPI [3] for innovative forms of resmetirom API for the fixed dose combina program; Sagimet anticipates selecting one of the licensed innovative forms of resmetirom for combination with denifanstat in a fixed dose combination (FDC) tablet for use in a potential Phase 3 tri

1. Loomba R, et al. Lancet Gastroenterol Hepatol. 2024;9(12):1090-1100.
2. Tsai WW, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/2024-EASL-poster-GAN-model-final.pdf
Tsai WW, et al. Presented at: EASL 2024; June 5-8, 2024; Milan, Italy. https://sagimet.com/wp-content/uploads/2024/06/2024-EASL-poster-TNO-model-final.pdf
3. Assia Chemical Industries Ltd. (Assia), doing business as TAPI Technology & API Services (TAPI), a subsidiary of Teva Pharmaceutical Industries Ltd.

FASN Inhibition Offers Potential Benefit in Multiple Indications: Acne

SAGIMET BIOSCIENCES

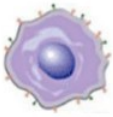
FASN Also Plays a Key Role in Other Diseases With Significant Unmet Need

FASN

Palmitate Lipid building block

Cancer cell

Membrane synthesis, intracellular signaling, protein modification



FASN in cancer

Supports tumor survival

Enables tumor proliferation

Establishes resistance to drugs

Sebocyte

Sebum production

FASN in acne

Increases sebum production

Exacerbates sebum composition

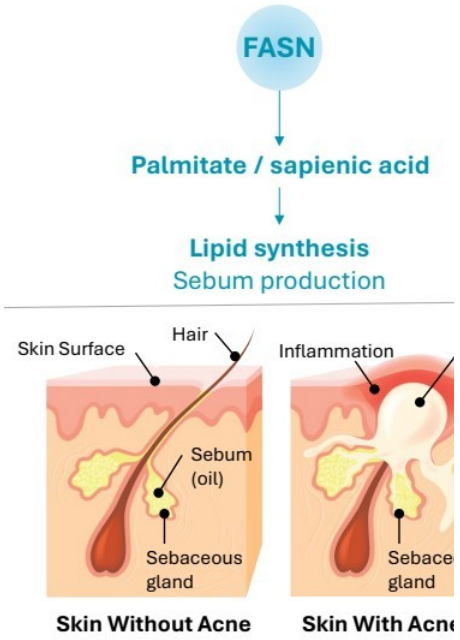
Acne Pathogenesis and Potential Role of FASN Inhibitors

Multifactorial pathogenesis of acne involves 4 key aspects:

- Increased sebum in sebaceous glands (80% of lipids produced through DNL)
- Abnormal or excessive follicular hyper-keratinization
- Accelerated bacterial growth (C. acnes)
- Localized inflammatory response

FASN is an attractive therapeutic target for acne

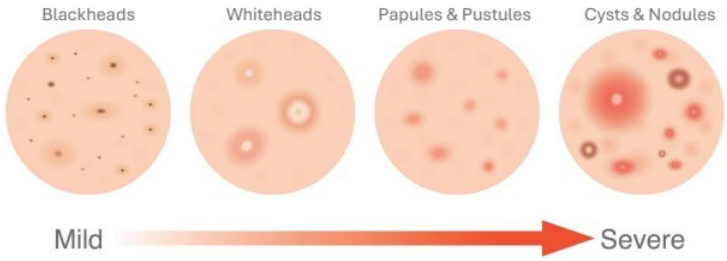
- Denifanstat directly reduced cutaneous (skin) sebum DNL lipids in two Phase 1 studies
- FASN inhibition has potential to reduce inflammation, through decreasing cytokine secretion and Th17 activation[1]



Heng AHS, Chew FT. *Sci Rep*. 2020;10(1):5754.
1. O'Farrell M, et al. *Sci Rep*. 2022;12(1):15661.

Acne Market Overview

Global acne market is expected to reach $17B in next decade[1]



5.1 million US acne patients are treated by dermatologists annually (total US acne market is 50 million people)[2,3]

- Acne is the #1 or #2 patient concern in dermatology offices and 65%+ of patients in dermatology offices have private insurance[4]
- Although acne treatments are currently available, dermatologists are open to new therapies (Seysara® Tablets & Winlevi® Cream)
- There is no cure for acne; due to its pathology, most patients require chronic management and multiple courses for flare control annually

Acne patients visiting a dermatologist are aligned to potential positioning of FASN inhibitor[4]

- 70% of patients presenting to dermatologists have moderate to severe disease[4]
- Approximately 70% of patients have inflammatory lesions, and 16% of patients are post-menopausal women[3]

1. www.expertmarketresearch.com/reports/acne-treatment-market
2. Bickers DR, et al. *J Am Acad Dermatol*. 2006;55(3):490-500. 3. American Academy of Dermatology. Burden of skin disease. 2017. www.aad.org/BSD.
4. Sagimet Biosciences data on file. Market research conducted in July 2024 among 50 dermatologists.

Acne Treatment Algorithm

Disease management involves flare and prevention intervention

Mild Disease	Moderate to Severe Disease	Severe (Cystic) Disease
Treatment includes topical agents used as mono-therapy, combination therapy, or with fixed dosed combination products	**Treatment approach adds oral products on top of the topical agents**	**Severe (cystic) patients are genera managed with isotretinoin (Accutane®)**
Main topical therapy categories • Retinoids • Benzoyl Peroxide • Antibiotics • Clascoterone • Salicylic Acid • Azelaic Acid	Main oral therapy categories: • Antibiotics (tetracyclines, sarecycline) • Hormonal contraceptives • Spironolactone (off-label) • Intralesional corticosteroids	Main therapy categories: • Isotretinoin

- Most acne patients receive skin care routines that include OTC cleansers and moisturizers to address AEs associated wit their treatment

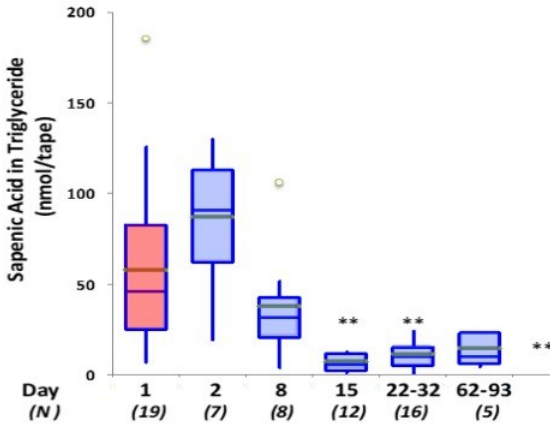
Clinical Data Support Mechanism of Action of a FASN Inhibitor in Acne

In multiple Phase 1 trials, FASN inhibitor demonstrated a decrease in DNL sebum lipids[1-3]

- FASN inhibitor demonstrated a >90% reduction in sebum lipids by day 15[1,2]
- FASN inhibitor maintained the reduced level of sebum lipids through the entire study[1,2]
- FASN inhibitor demonstrated a dose responsive impact on sebum lipids[1,2]

Note: denifanstat dose in this Phase 1 trial in cancer patients is several times higher than 50 mg dose tested in acne and MASH

Phase 1 oncology trial
Sebutape® assessment of cutaneous sebum lip



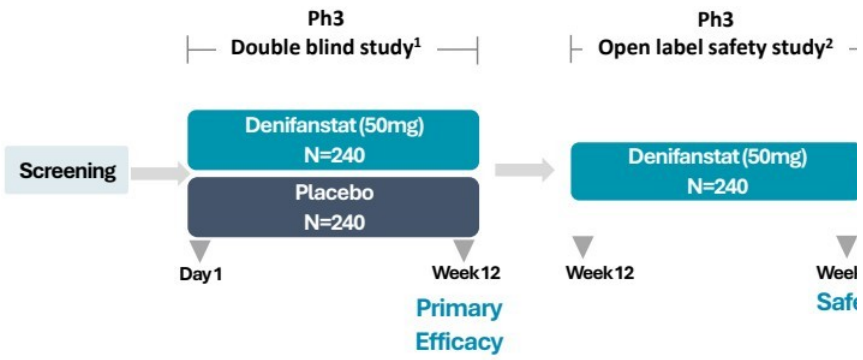
Days on therapy (# of subjects)

1. Duke G, et al. Presented at: EASL 2017; April 19-23, 2017; Amsterdam, The Netherlands. https://sagimet.com/wp-content/uploads/2017/05/3VBIO_EASLposter.pdf. 2. Falchook G, et al. *EClinicalMedicine*. 2021;34:100797.
3. Duke G, et al. Presented at: AASLD 2016; November 11-15, 2016; Boston, MA. https://sagimet.com/wp-content/uploads/2016/11/2016_AASLD_FASN_NASH_36x60_v10.pdf.

Ascletis Acne Phase 3 Clinical Trial Design

Denifanstat Phase 3 in acne

- Moderate to severe acne
- Multi-center placebo controlled
- 1:1 randomization
- Double-blind
- Once daily oral dosing
- 480 patients in China



Primary endpoints at week 12

- % patients who receive IGA success (defined as at least a 2-point reduction in IGA from baseline, and an IGA of 0 or 1 at v
- % change of total lesion counts from baseline
- % change of inflammatory lesion counts from baseline

Key secondary endpoint at week 12

- % change of non-inflammatory lesion counts from baseline

1. ClinicalTrials.gov. NCT06192264. Study ASC40-303. https://clinicaltrials.gov/study/NCT06192264. 2. ClinicalTrials.gov. NCT06248008. Study ASC40-304. https://clinicaltrials.gov/study/NCT06248

Ascletis Acne Phase 3 Clinical Trial: All Primary and Secondary Endpoints M

Baseline Characteristics	50mg denifanstat (n=240)	Placebo (n=240)
Total lesion count	102.2	102.1
Inflammatory lesion count	42.1	43.1
IGA=3 (moderate), %	85.8	85.8
IGA=4 (severe), %	14.2	14.2

Efficacy endpoints [1]	50mg denifanstat (n=240)	Placebo (n=240)	50mg denifanstat (placebo adjusted)	p v
% Treatment success [IGA] [2] (primary endpoint)	33.2	14.6	18.6	<0.
% Change in total lesion count (primary endpoint)	-57.4	-35.4	-22.0	<0.
% Change in inflammatory lesion count (primary endpoint)	-63.5	-43.2	-20.3	<0.
% Change in non-inflammatory lesion count (key secondary endpoint)	-51.9	-28.9	-23.0	<0.
Absolute change in total lesion count (secondary endpoint)	-58.3	-36.2	-22.1	<0.
Absolute change in inflammatory lesion count (secondary endpoint)	-26.6	-18.4	-8.2	<0.

Baseline demographics and efficacy endpoints of 50 mg denifanstat oral, once daily for 12 weeks versus Placebo (Intent-to-treat, ITT analysis change from baseline).

1. The efficacy data are LSMEANs.
2. Treatment success is defined as an Investigator's Global Assessment (IGA) score of 0 (clear) or 1 (almost clear) with at least a 2-point decrease from baseline.

Ascletis Acne Phase 3 Clinical Trial Safety Data

Denifanstat 50mg was generally well tolerated during the 12-week study

Treatment-emergent adverse events (TEAEs):

- TEAE incidence rates were comparable between denifanstat and placebo
- Only two categories of TEAEs had an incidence rate of 5% or more:
  - Dry eye (investigator reported as “dry eye” or “xerophthalmia”) in 10.9% of denifanstat-treated subjects vs 8.0% in th placebo group*
  - Dry skin reported in 6.3% of denifanstat-treated subjects vs 2.9% in the placebo group

Adverse events (AEs):

- All denifanstat-related AEs were mild or moderate
- No denifanstat-related grade 3 or 4 AEs
- No denifanstat-related serious AEs (SAEs)
- No deaths were reported

* The classifications of “dry eye” or “xerophthalmia” were not related to the AE grade.

Ascletis Acne Open Label Phase 3 trial

Denifanstat generally well-tolerated in the open label clinical trial

Treatment-emergent adverse events (TEAEs):

- Only two categories of TEAEs had an incidence rate of 5% or more with dry eye syndrome in 5.5% of denifanstat-treated subjects and dry skin reported in 5.2% of denifanstat-treated subjects

Adverse events (AEs):

- All denifanstat-related AEs were mild or moderate; no denifanstat-related Grade 3 or 4 AEs; no AE-related permanent discontinuations; Grade 1 hair thinning in the study was experienced by only 1 denifanstat-treated patient (which resolved within eight weeks while remaining in study without a change in dose); no deaths were reported

Serious adverse events (SAEs):

- No denifanstat-related SAEs; 2 non-denifanstat-related SAEs (1 breast lump, 1 contusion), both resolved

Efficacy Endpoints (secondary endpoints of the trial) :

- Efficacy endpoints (secondary endpoints of the trial) included the number of subjects with an IGA score decrease by at le points, number of subjects dropping from an IGA score of 3 down to 0 or 1, the percentage reduction in total skin lesion co and the percentage reduction in inflammatory skin lesion count.
- Subjects treated with denifanstat showed improvements in all efficacy endpoints beyond those observed at 12 weeks

Ascletis data on file. Safety and efficacy endpoints of 50 mg denifanstat oral, once daily for 52 weeks versus placebo for 12 weeks and 50mg denifanstat oral once daily for 40 weeks

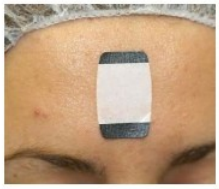
Second FASN Inhibitor TVB-3567 Entered FIH Phase 1

Phase 1 clinical trial initiated June 2025

A double-blind, randomized, placebo-controlled trial to assess the safety, tolerability, pharmacokinetics and pharmacodynamics of single and multiple ascending doses of TVB-3567 in healthy and acne participants

- **Includes sebum analysis as pharmacodynamic readout**

PART	DESIGN	PLANNED # of PARTICIPANTS
A	SAD	~56
B	Food effect	~12
C	MAD	~32
D	MAD/ACNE	~28

Sebumeter	**Sebutape**
	
Quantity of Sebum	Quality* of Sebum

Note: SAD = Single ascending dose. MAD = Multiple ascending dose

Each SAD/MAD cohort planned to include 6 participants on active and 2 on placebo. * Lipidomic analysis with focus on FASN-derived lipids.

ClinicalTrials.gov. NCT06989840. Study SB3567-CLIN-001. https://clinicaltrials.gov/study/NCT06989840.

Potential Clinical Development Program for TVB-3567 in Acne

Phase 1 trial initiated in June 2025

Goal to initiate Phase 2 trial in 2026, subject to consultation with regulatory authorities and outcome of Phase 1 trial

Step 1 - Phase 1 first-in-human pharmacokinetic (PK) clinical trial of TVB-3567 in healthy volunteers

- PK and pharmacodynamics (PD) evaluation to confirm profile
- Assess safety/tolerability
- Confirm potential doses for an acne Phase 2 trial

Step 2 - Phase 2 clinical trial in moderate to severe acne patients

- Upon completion of Phase 1, plan to consult with regulatory authorities regarding Phase 2 trial design, with goal of initiatin Phase 2 trial in 2026
- Phase 2 trial design anticipated to be informed by the results of the Phase 1 trial, expect a 12-week dose ranging study in moderate to severe acne patients with lesion reduction, treatment success (IGA) as endpoints

Attractiveness of FASN Inhibition in Acne

FASN Inhibition in Acne

- Oral FASN inhibitors offer a novel mechanism of action for the potential treatment of moderate t severe acne
- Denifanstat met all primary and secondary endpoints in Phase 3 clinical trial in patients with mo to severe acne vulgaris in China ; Denifanstat was generally well tolerated
- Denifanstat was well-tolerated in Ascletis' open-label Phase 3 clinical trial
- Denifanstat NDA for the treatment of moderate to severe acne accepted by NMPA in December 2

Potential of TVB-3567 in Acne

- Acne market in dermatology is large (>50m people in the US) and aligned to those patients most be prescribed an oral FASN inhibitor
- TVB-3567 IP:
  - Method of use application for TVB-3567 for acne filed 2025; if granted—2046
  - Composition of matter patent—2035; potential PTE to 2038

Development Pathways

- First-in-human Phase 1 clinical trial of TVB-3567 initiated in June 2025 for development in acne
  - Upon completion of TVB-3567 Phase 1, plan to consult with regulatory authorities regardin Phase 2 trial design, with goal of initiating TVB-3567 Phase 2 in 2026
  - Consulted with US FDA at end 2025 on the potential use of Ascletis Phase 3 data for the development of denifanstat in acne (e.g., as one of two registrational trials)

FASN Inhibition Offers Potential Benefit in Multiple Indications: Oncology

SAGIMET BIOSCIENCES

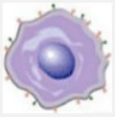
FASN Also Plays a Key Role in Other Diseases With Significant Unmet Need

FASN

Palmitate Lipid building block

Cancer cell

Membrane synthesis, intracellular signaling, protein modification



FASN in cancer

Supports tumor survival

Enables tumor proliferation

Establishes resistance to drugs

Sebocyte

Sebum production

FASN in acne

Increases sebum production

Exacerbates sebum composition

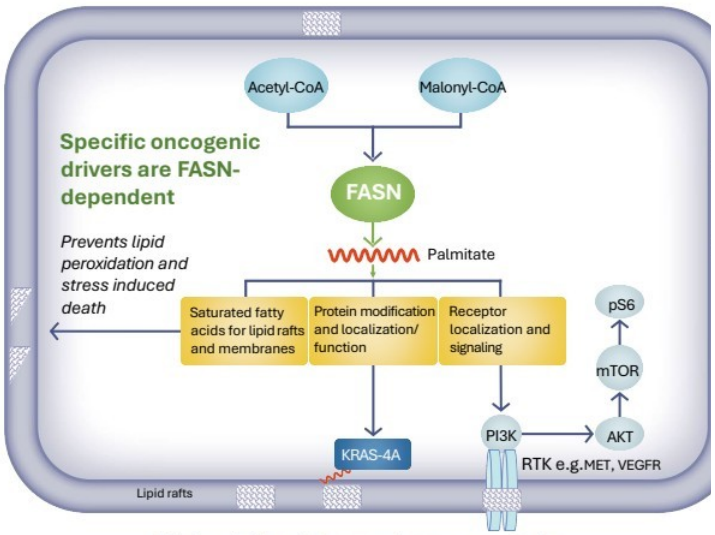
FASN Is Integral to Tumor Cell Proliferation and Survival

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

* Falchook G, et al. *EClinicalMedicine*. 2021;34:100797.

Cancer Program Focuses on 4 FASN-Dependent Tumor Types

Type	Status
Prostate	**Phase 1 ongoing** Investigator Sponsored at Weill Cornell, denifanstat combination with enzalutamide[1] Phase 1 results expected 1H2027
HCC	**Preclinical and translational work completed** Patient selection strategy by bioinformatics on primary samples Positive preclinical combination results[2] Phase 2-ready
NSCLC KRASM	**Preclinical and clinical evidence** Positive preclinical combination with KRAS inhibitor[3] Encouraging monotherapy Phase 1 results with denifanstat[4] Phase 2-ready
GBM	**Phase 2 completed** Positive investigator sponsored Phase 2 results[5] Ascletis announced cessation of China GBM program in August 2025 [6]

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

1. ClinicalTrials.gov. NCT05743621. https://clinicaltrials.gov/study/NCT05743621. 2. Wang H, et al. *Hepatology*. 2022;76(4):951-966. 3. Liu Y, et al. *Lung Cancer*. 2021;153:73-80. 4. O'Farrell M, et a at: AARC 2016; April 16-20, 2016; New Orleans, LA. Abstract LB-214. 5. Kelly W, et al. *Clin Cancer Res*. 2023;29(13):2419-2425. 6. ClinicalTrials.gov. NCT05118776. Study ASC40-301. https://clinicaltrials.gov/study/NCT05118776.

Sagimet at a Glance

Unique MOA: FASN Inhibition

- Our lead molecule, denifanstat, is a novel fatty acid synthase (FASN) inhibitor with a differentiated MOA with the potential to target multiple underserved diseases
- Strong clinical data demonstrates denifanstat's proof of concept across multiple disease states

Denifanstat in MASH

- Denifanstat directly targets the 3 key drivers of MASH (metabolic dysfunction-associated steatohepatiti – liver fat, inflammation, and fibrosis
- Successful outcome of Phase 2b trial; met both primary endpoints with significant reduction in fibrosis
- Pre-clinical data demonstrated synergistic effect of combination of FASN inhibitor and resmetirom
- Phase 1 pharmacokinetics (PK) clinical trial of a combination of denifanstat and resmetirom completed in December 2025, Phase 2 clinical combination trial with denifanstat and resmetirom in patients with MASH cirrhosis (F4) planned to initiate in 2H 2026

TVB-3567 in Acne

- Our follow-on FASN inhibitor, TVB 3567, received Investigational New Drug (IND) clearance in March 2
- First-in-human Phase 1 clinical trial initiated in June 2025 for development of an acne indication