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): October 23, 2023

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 October 23, 2023, Sagimet Biosciences Inc. (the "Company") updated information reflected in a slide presentation, which is attached as Exhibit 99.1 to this Current Report on Form 8-K and is incorporated herein by reference. Representatives of the Company will use the updated presentation in various meetings with investors from time to time.

The information contained in this Item 7.01 (including Exhibit 99.1) is being furnished and shall not be deemed "filed" for purposes of Section 18 of the 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 or the Exchange Act, except as shall be expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Document
99.1	Investor Presentation of Sagimet Biosciences Inc., dated October 23, 2023.
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURE

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

Sagimet Biosciences Inc.

Date: October 23, 2023

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


SAGIMET
BIOSCIENCES
Targeting Metabolic Dysfunction with
Novel Therapies to Treat NASH, Acne and Cancer
October 2023

Forward Looking Statements

- This presentation contains forward-looking statements within the meaning of, and made pursuant to the safe har of, The Private Securities Litigation Reform Act of 1995. All statements contained in this document, other than st historical facts or statements that relate to present facts or current conditions, including but not limited to, state possible or assumed future results of operations, business strategies, research and development plans, regulator market opportunity, competitive position and potential growth opportunities are forward-looking statements. Th involve known and unknown risks, uncertainties and other important factors that may cause our actual results, p achievements to be materially different from any future results, performance or achievements expressed or imp forward-looking statements. In some cases, you can identify forward-looking statements by terms such as "may," "would," "expect," "plan," "anticipate," "could," "intend," "target," "project," "believe," "estimate," "predict," "po "continue" or the negative of these terms or other similar expressions. The forward-looking statements in this pr only predictions. These forward-looking statements speak only as of the date of this presentation and are subject risks, uncertainties and assumptions, some of which cannot be predicted or quantified and some of which are be control, including, among others: the clinical development and therapeutic potential of denifanstat or any other we may develop; our ability to advance drug candidates into and successfully complete clinical trials, including ou Phase 2b clinical trial; our relationship with Ascletis, and the success of its development efforts for denifanstat; th our estimates regarding our capital requirements; and our ability to maintain and successfully enforce adequate i property protection. These and other risks and uncertainties are described more fully in the "Risk Factors" sectio recent filings with the Securities and Exchange Commission and available at www.sec.gov. You should not rely on looking statements as predictions of future events. The events and circumstances reflected in our forward-lookin may not be achieved or occur, and actual results could differ materially from those projected in the forward-look Moreover, we operate in a dynamic industry and economy. New risk factors and uncertainties may emerge from it is not possible for management to predict all risk factors and uncertainties that we may face. Except as require law, we do not plan to publicly update or revise any forward-looking statements contained herein, whether as a r information, future events, changed circumstances or otherwise.

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

Dave Happel
President & CEO

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

George Kemble
Executive Chairman

- AstraZeneca (formerly MedImmune, Aviron): SVP research for biologics and general manager of California operations, VP vaccine research & development for vaccines
- Ph.D. – Stanford University, dept of microbiology & immunology

Eduardo Martins
CMO

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

Anthony Rimac
CFO

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

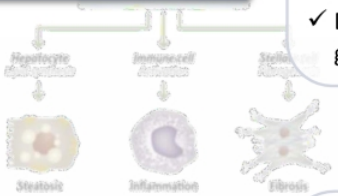
Elizabeth Rozek
General Counsel and CCO

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

Sagimet Investment Highlights

Critical role of FASN enzyme in NASH

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



Denifanstat: FASN inhibitor with compelling clinical data

- ✓ FASCINATE-2 Phase 2b interim data
  - Statistically significant improvements in key biomarkers of NASH: liver fat, inflammation, fibrosis
  - Results consistent with Phase 2a study
  - Strengthen belief in Phase 2b liver biopsy results expected in 1Q 2024

Precision medicine is key differentiator

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

Strong rationale for FASN in acne and cancer

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

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

Strong financial position

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

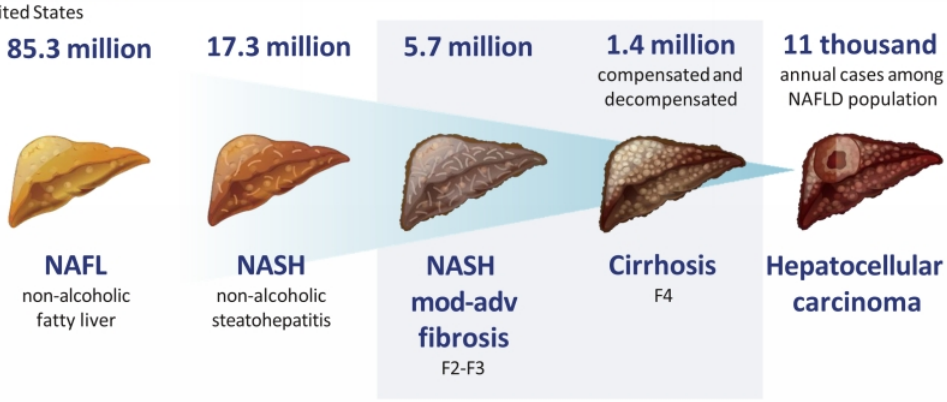
Denifanstat Pipeline of Multiple Indications and Clinical Milest

Therapeutic Area	Indication	Stage of Development	Expected Milest
		Preclinical / Phase 1 / Phase 2 / Phase 3	
Metabolic disease	NASH - F2/F3		• Phase 2b biopsy re
	NASH - cirrhosis		• Phase 1 hepatic im 1Q 2024
Dermatology	Acne*	歌礼 ascletis	• Phase 2 topline res
Oncology	Solid tumors		• Patient selection a dependent tumor
	Recurrent GBM*	歌礼 ascletis	• Phase 3 enrollmen achieved in 3Q 202 planned

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

NASH: A Burgeoning Epidemic

Patients in 2016[1]
United States



Disease challenges

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

Drug development challenges

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

Denifans

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

DNL = de novo lipoge

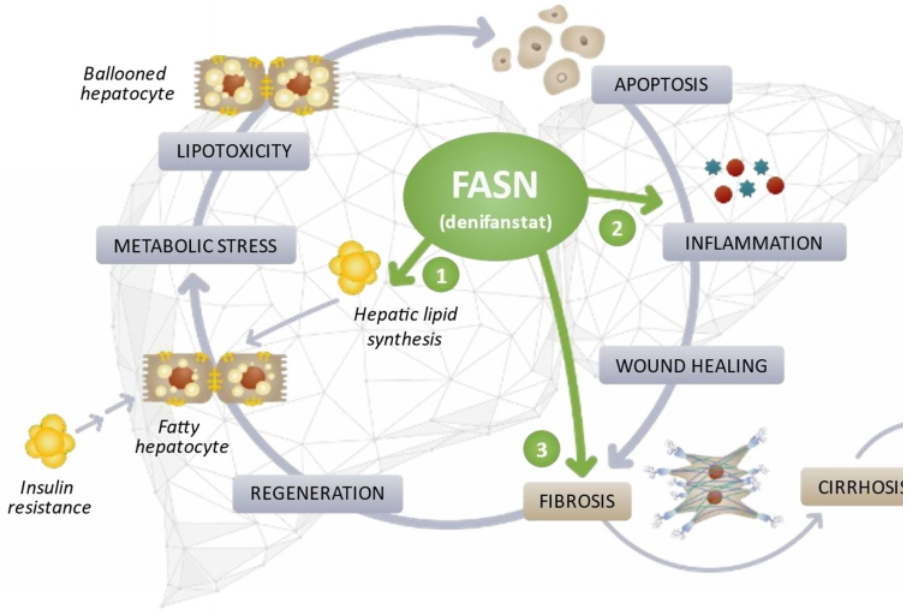
[1] *Estes, et al. 2018;* http://dx.doi.org/10.1016/j.jhep.2018.05.036

Denifanstat in NASH

Denifanstat: Differentiated Mechanism Believed to Target Key Driver

Denifanstat has independent mechanisms designed to:

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



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

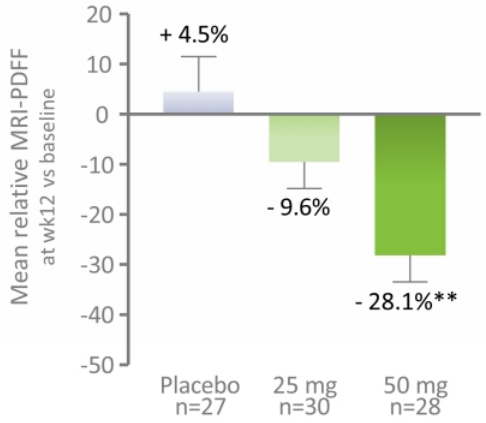
Denifanstat Showed Dose-Dependent Reduction of Liver Fat in FASCINA

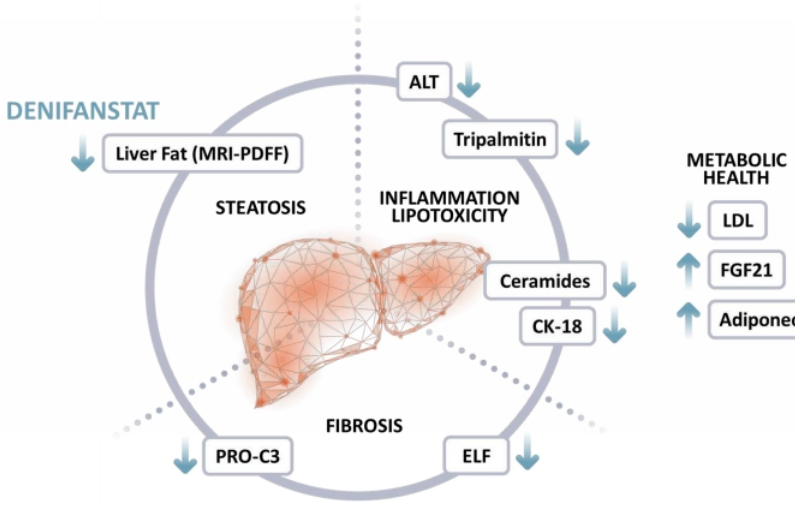
Improved Key Drivers of NASH and Metabolic Health

FASCINATE-1 Phase 2 study[1]

- Dose-finding, global, multicenter, Phase 2 trial
- Oral, once-daily, 12-week dosing
- >8% liver fat and presumed fibrosis
- U.S. and China

FASCINATE-1
Liver Fat Change





[1]*Loomba et al, 2021 Gastroenterology. doi: 10.1053/j.gastro.2021.07.025*
***p<0.005, Mean ±SEM. LSM difference versus placebo for liver fat.*

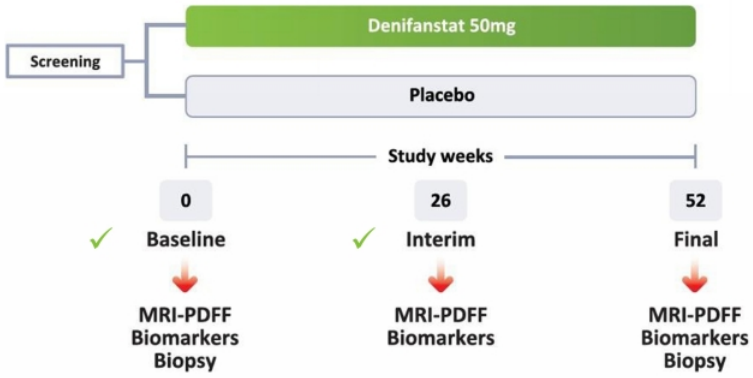
Denifanstat: Well Tolerated at 25/50mg Doses in FASCINATE-1

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

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

Phase 2b Biopsy Trial: Measuring Histological Improvement

FASCINATE-2 Phase 2b trial design



- Biopsy confirmed F2-F3 NASH patients
- 52 weeks, 2:1 50mg or placebo, double-blind
- Fully enrolled: 168 patients in U.S., Canada, and Europe
- Prespecified interim analysis of the first 52 patients with MRI-PDFF >8%

Primary endpoints (biopsy)

- NAS ≥2 points improvement w/o worsenin
  OR
  resolution of NASH w/o worsening of fibro
  - Lead reader of liver biopsies: pa
    Pierre Bedossa MD. PhD.
- Safety

Secondary endpoints

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

Interim Analysis Cohort Represents Target Patient Population

FASCINATE-2 Phase 2b Interim Analysis Demographics			
Mean (SD)	**Placebo (22)**	**Denifanstat (30)**	**Combined**
Age (years)	56.8 (9.4)	56.1 (12.4)	56.4 (11.1)
Female/Male (%)	14 (63.6%) / 8 (36.4%)	17 (56.7%) / 13 (43.3%)	31 (59.6%) / 21 (40.4%)
Not Hispanic or Latino	16 (72.7%)	24 (80.0%)	40 (76.9%)
Weight (kg)	97.8 (21.9)	100.9 (21.2)	99.6 (21.4)
Diabetes (% T2DM)	13 (59.1%)	21 (70.0%)	34 (65.4%)
F2/F3 (%)	12 (54.5%) / 10 (45.5%)	12 (40.0%) / 18 (60.0%)	24 (46.2%) / 28 (53.8%)
MRI-PDFF (%)	21.78 (5.46)	17.46 (6.36)	19.29 (6.32)
Fibroscan (kPa)	10.67 (4.07)	12.29 (7.33)	11.56 (6.04)
ALT (U/L)	69.77 (42.50)	57.14 (27.55)	62.70 (35.11)
AST (U/L)	51.00 (29.87)	44.43 (22.65)	47.32 (26.00)
LDL (mg/dL)	111.37 (40.6)	96.29 (50.27)	102.86 (46.4)
ELF	9.70 (0.76)	9.73 (0.76)	9.72 (0.75)
PRO-C3 cobas® (ng/mL)	35.72 (15.71)	32.54 (11.19)	33.91 (13.28)

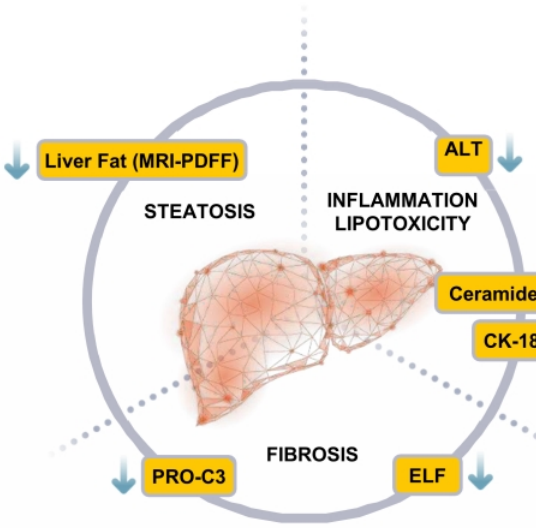
- Typical F2/F3 NAS
- Middle-aged
- High % of diabete
- High liver fat by M
- Elevated liver enz inflammation
- Non-invasive mark consistent with F2

FASCINATE-2 Interim Results Consistent with Comprehensive Positive Readouts from FASCINATE-1

- FASCINATE-2 interim analysis showed consistent improvements in key drivers of NASH as observed in FASCINATE-1

	Mechanism	Biomarker
1	Steatosis	Liver fat (MRI-PDFF)
2	Inflammation/lipotoxicity	ALT, CK-18, ceramides
3	Fibrosis	PRO-C3, ELF

- Improvements observed in multiple biomarkers of 4 metabolic health
  - LDL-cholesterol
  - FGF-21

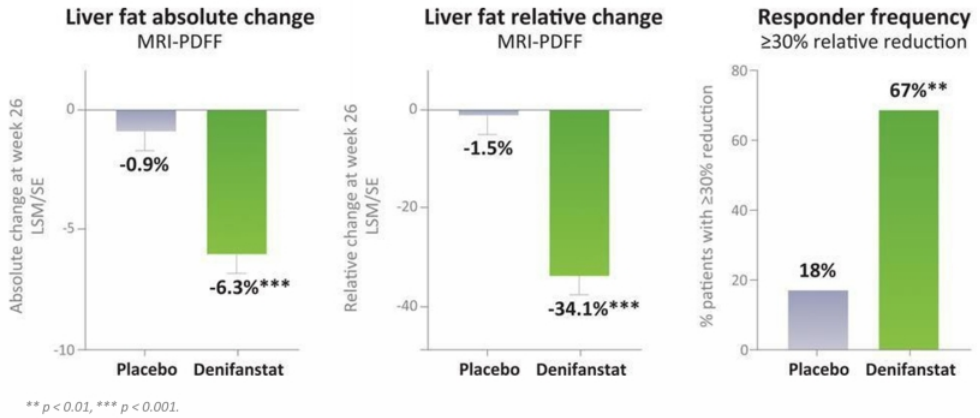


Biomarkers replicated in FASCINATE-2

Denifanstat Decreased Liver Fat

Responders Correlate with Liver Biopsy Improvement

1 Steatosis biomarker – liver fat



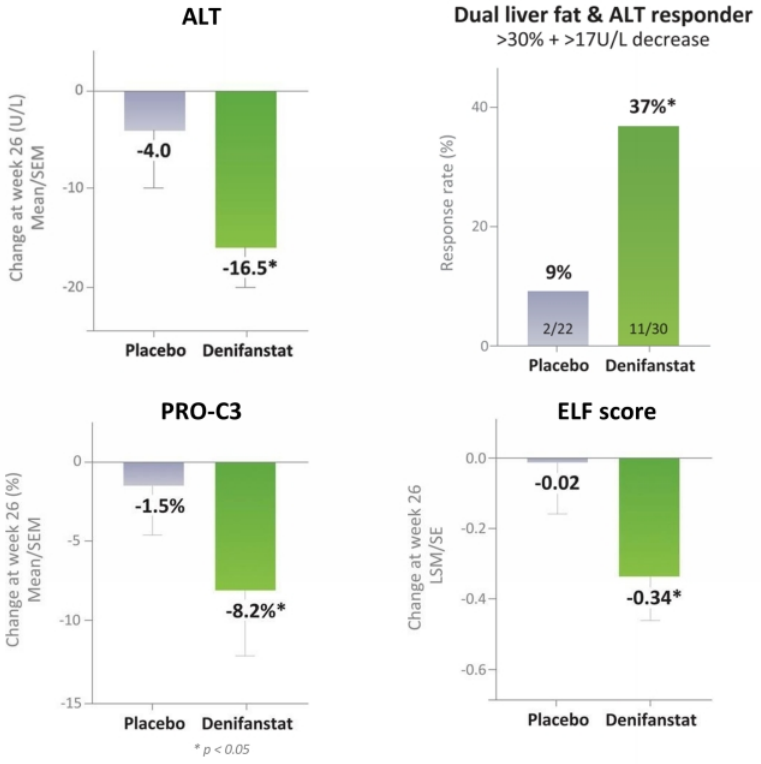
** p < 0.01, *** p < 0.001.

Findings to

- Denifanstat indu statistically signif reduction of liver
- 67% (p<0.001) M response rate
- About half of res decreased liver fa
- A relative reducti ≥30% by MRI-PD shown to correla biopsy response

Denifastat Decreased PRO-C3 and ELF – Suggests Fibrosis Red

2 3 Inflammation and fibrosis biomarkers



* $p < 0.05$

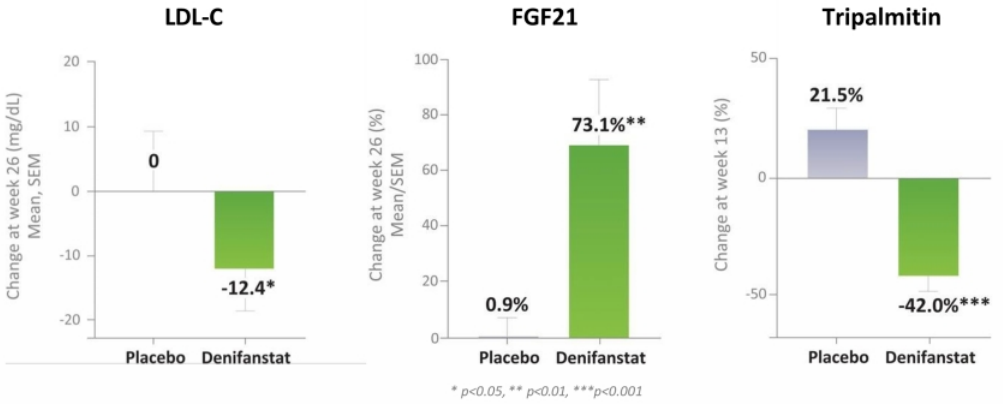
Findings to

- ALT decrease sug decrease in inflar denifanstat
- PRO-C3 decrease decrease of liver denifanstat
- ELF score decrea decrease of liver denifanstat. ELF s prognostic value

Other liver biomarkers con

Denifanstat Improved Markers of Cardiometabolic Health

4 Metabolic health / lipid biomarkers



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

Findings to

- LDL-cholesterol
  denifanstat may
  cardiovascular b
- FGF21 increase
  may induce imp
  insulin sensitivit
- Tripalmitin decre
  denifanstat inhib
  and reduced pal
  synthesis

Denifanstat Passed Planned IDMC Safety Review in FASCINATE

Sagimet is blinded to data

- **All randomized subjects: blinded data set including active and placebo**
- Majority of AEs to date were Grade 1 or 2; no Grade ≥3 drug-related AEs
- A planned safety review of unblinded data from all 168 patients conducted by Independent Data Monitor – no concerns

FASCINATE-2 Phase 2b - Blinded data set	
Treatment Emergent Adverse Event (TEAE) Classification	**N=168 Number of Patients with Event at Stated Grade**
Any TEAE	Gr 1: 115 (68.5%) Gr 2: 69 (41.1%) Gr 3: 10 (6.0%) Gr 4: 1 (0.6%)
TEAE leading to drug/placebo discontinuation	21
Treatment Emergent Serious Adverse Event (SAE)	11 (all unrelated to study treatment)
Drug/placebo-related TEAE	Gr 1: 52 (30.1%) Gr 2: 25 (14.9%)

AE data as of 3 April 2023

NASH Development Program

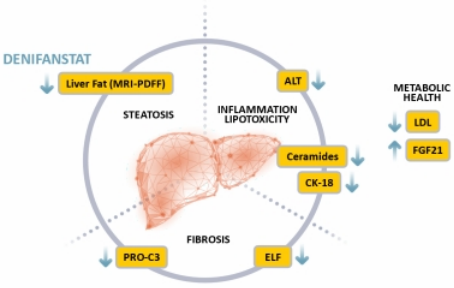
Progression from Phase 2b to Phase 3

Phase 2b – baseline Fibrosis stage

Interim cohort
F2 – 46.2%
F3 – 53.8%

Enrollment completed Sep 2022

Phase 2b – 26 weeks Non-invasive interim



Interim results released Nov 2022

Phase 2b – 52 weeks Histology

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

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

Biopsy results expected 1Q 2024

Ph Fibrosis en

Expect t
2b result
AI patho
to desig
Phase 3

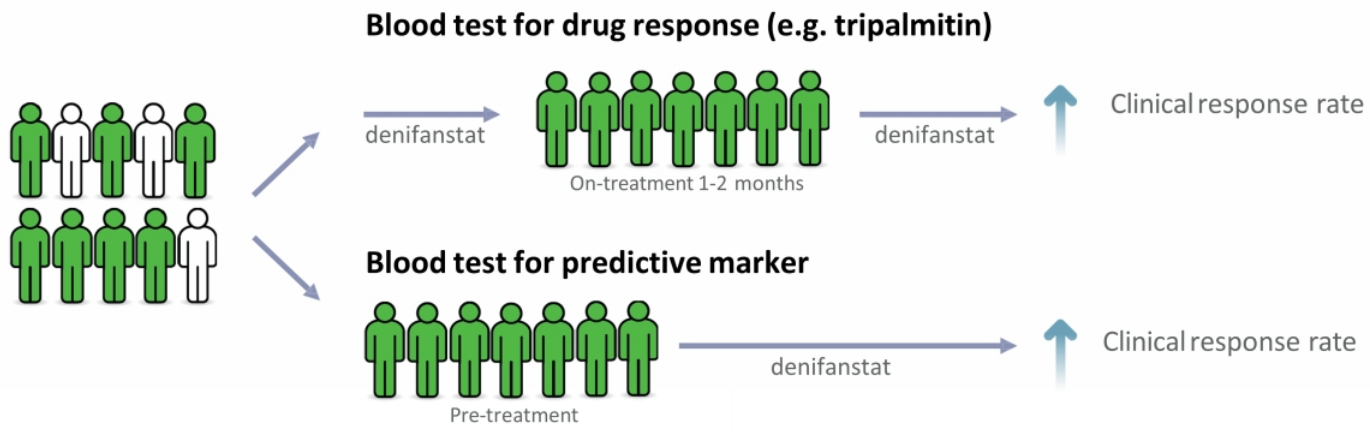
Startup act

We Believe Denifanstat is Differentiated in the Evolving NASH Landsc

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

Precision Medicine: Blood Tests May Lead to Improved Patient Outc

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



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

Strong Monotherapy Opportunity for Denifanstat in NASH

Expansion as backbone of combinations

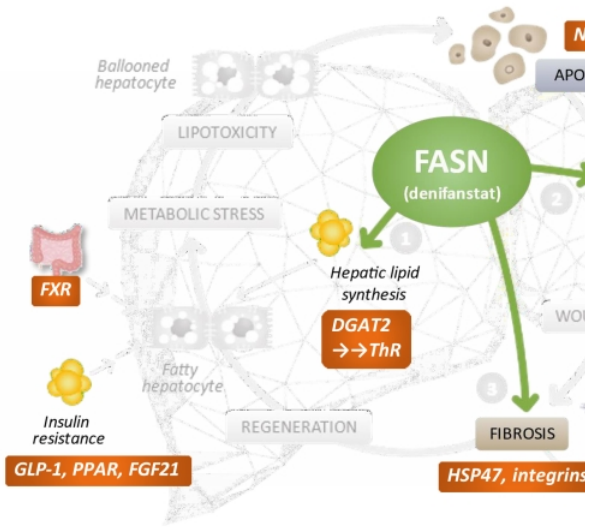
Denifanstat data support success as first line monotherapy

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

Broaden market opportunity through combinations with denifanstat as backbone

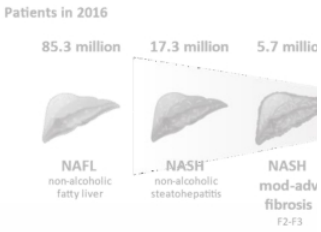
- Denifanstat's potential
  - ✓ Complementary to other mechanisms
  - ✓ Potential for fixed dose combinations with other oral medications
- ✓ Preclinical combination studies ongoing
  - NASH agents: anti-fibrotic, other metabolic agents
  - Co-morbidities: diabetes and other cardiovascular agents

Illustrative potential combo mecl



Additional Expansion Opportunities in NASH

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

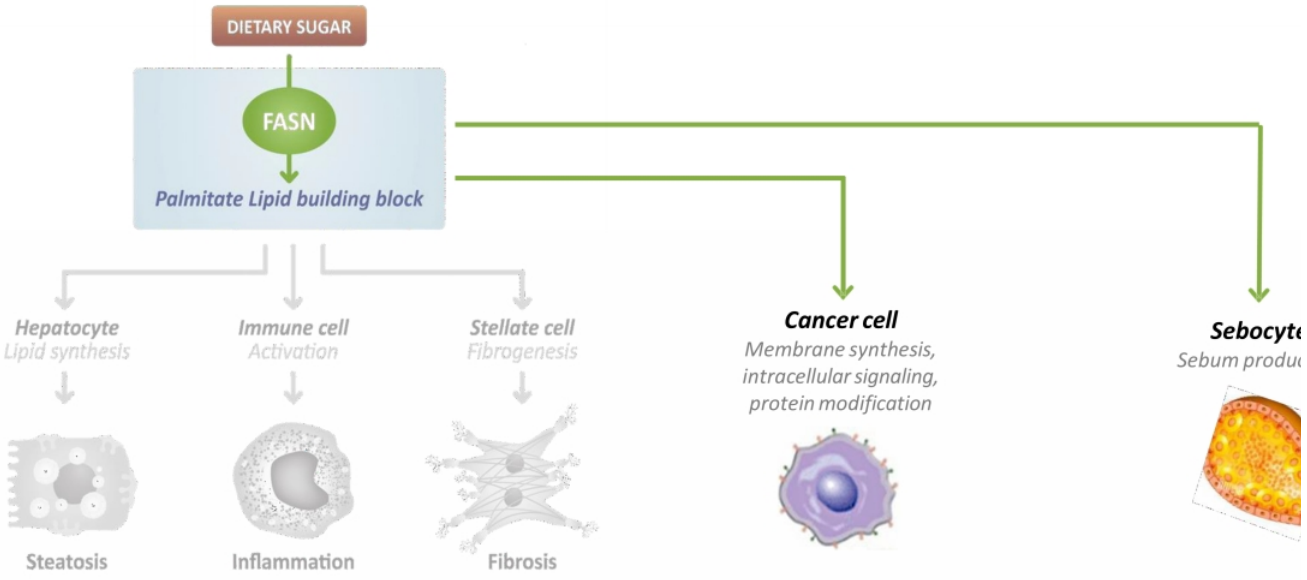


- **Pediatric NASH**
  - 23% of children with NAFLD have NASH at the time of diagnosis
  - Next steps
    - Compile safety data across all denifanstat studies in young adults including FASCINATE-2
    - Nonclinical toxicology study in juvenile animals – plan to initiate in 2024
    - Phase 2 trial in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

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

Other Indications

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



FASN in NASH

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

FASN in cancer

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

FASN in ac

1. Sebum
2. Sebum

DNL Pathway Plays a Role in the Pathogenesis of Acne

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

=> FASN inhibition has potential therapeutic application

Phase 1 – sebum analysis
by Sagimet

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

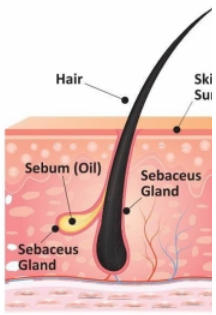
Phase 2 – acne
by Ascletis in China

歌礼 ascletis

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

Positive topline results announced May 2023

- Met primary and secondary endpoints
- Well-tolerated
- Sagimet evaluating clinical development plans for U.S./EU and other major markets



Skin Without Acne

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

FASN is Integral to Tumor Cell Proliferation and Survival

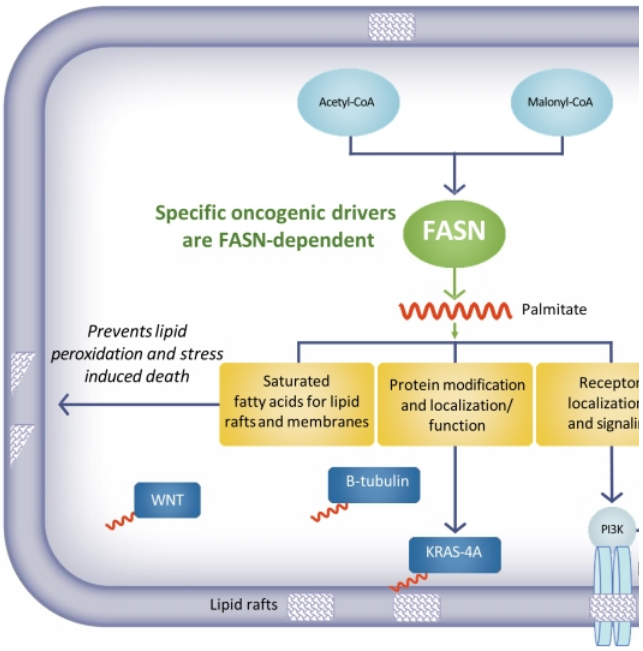
Reprogramed metabolism is one of the hallmarks of cancer

FASN-dependence

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

Completed Phase 1 provides foundation

- 136 patients received denifanstat
- Heavily pretreated Phase 1 population
- Recommended Phase 2 dose defined
- Promising clinical activity consistent with proposed mechanism
  - KRASM NSCLC patients had significantly longer duration on study with denifanstat than KRASWT ($p<0.02$), and 91% KRASM had stable disease



Dietary fatty acids cannot compensa de novo synthesized palmitate

FASN-Dependent Tumor Types Identified that Meet Core Crite

Program focused on 4 selected tumor types

Core criteria

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

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

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

Strong Financial Position and Intellectual Property Portfolio

Financial highlights Nasdaq: SGMT

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

Strong patent estate

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

Denifanstat Pipeline of Multiple Indications and Clinical Milest

Therapeutic Area	Indication	Stage of Development	Expected Milest
		Preclinical / Phase 1 / Phase 2 / Phase 3	
Metabolic disease	NASH - F2/F3		• Phase 2b biopsy re
	NASH - cirrhosis		• Phase 1 hepatic im 1Q 2024
Dermatology	Acne*	歌礼 ascletis	• Phase 2 topline res
Oncology	Solid tumors		• Patient selection a dependent tumor
	Recurrent GBM*	歌礼 ascletis	• Phase 3 enrollmen achieved in 3Q 202 planned

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