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Targeting Metabolic Dysfunction with Novel Therapies to Treat MASH, Acne and Cancer

Conference Call & Webcast on ITT and F3 Patient Population in Phase 2b FASCINATE-2 Clinical Trial of Denifanstat

June 13, 2024

Forward Looking Statements

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

Moreover, we operate in a dynamic industry and economy. New risk factors and uncertainties may emerge from time to time, and it is not possible for management to predict all risk factors and uncertainties that we may face. Except as required by applicable law, we do not plan to publicly update or revise any forward-looking statements contained herein, whether as a result of any new information, future events, changed circumstances or otherwise.

Leadership Team with Proven Development and Commercialization Experience

Dave Happel *President & CEO*

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

Brought multiple innovative healthcare products to the market

Thierry Chauche *CFO*

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

George Kemble *Executive Chairman*

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

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

Liz Rozek *General Counsel*

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

Eduardo Martins *CMO*

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

Led clinical development team of cenicriviroc for MASH

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

HORIZON · AstraZeneca · abbvie · Genentech · proventionbio · NOVARTIS · basilea PHARMACEUTICA · cognoa

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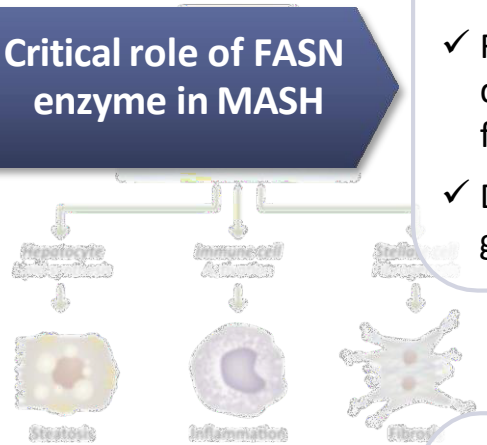
Sagimet Investment Highlights

Critical role of FASN enzyme in MASH

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



Denifanstat: FASN inhibitor with compelling clinical data

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

Precision medicine is key differentiator

- ✓ Blood test confirms drug response
- ✓ Predictive biomarkers identify likely responders
- ✓ Opportunity to personalize treatment and optimize outcomes

Strong rationale for FASN in acne and cancer

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

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

Strong financial position

- ✓ Upsized IPO completed in July 2023 raised $86.2M of net proceeds
- ✓ Follow-on financing completed in January 2024 raised net proceeds of $104.7M
- ✓ Cash, cash equivalents and marketable securities $193.7M as of 03/31/24, expected to fund current operations through 2025

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Development Pipeline: Indications and Clinical Milestones

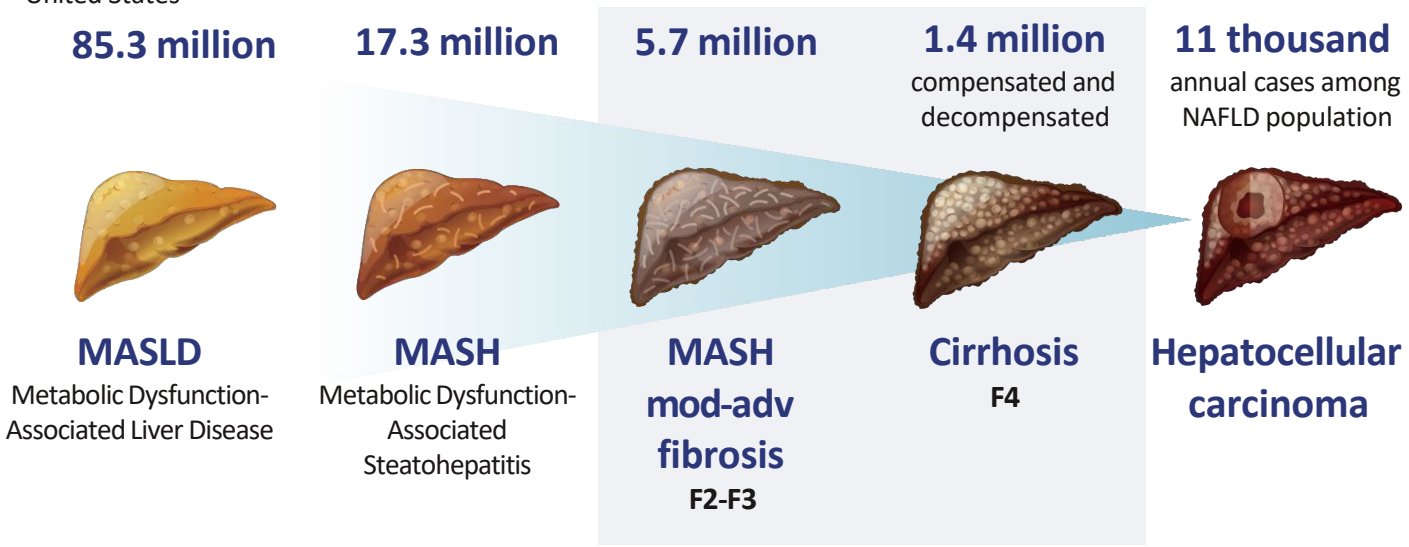
Therapeutic Area	Indication	Stage of Development	Expected Milestone / Status
Metabolic disease	MASH - F2/F3	TVB-2640 (Phase 2b)	• Phase 2b successfully completed; MASH Phase 3 study planned to start 2H 2024
		TVB-2640 (Phase 1)	• Phase 1 hepatic impairment results 1Q 2024
Dermatology	Acne	TVB-3567 (Preclinical)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

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

MASH: A Burgeoning Epidemic

Patients in 2016[1]

United States



Disease challenges

- Only one recently approved drug in U.S., and no approved drugs in Europe
- Complex disease, heterogeneous patient population
- Improving regulatory clarity, but liver biopsy still required

Drug development challenges

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

Denifanstat

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

DNL = de novo lipogenesis

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

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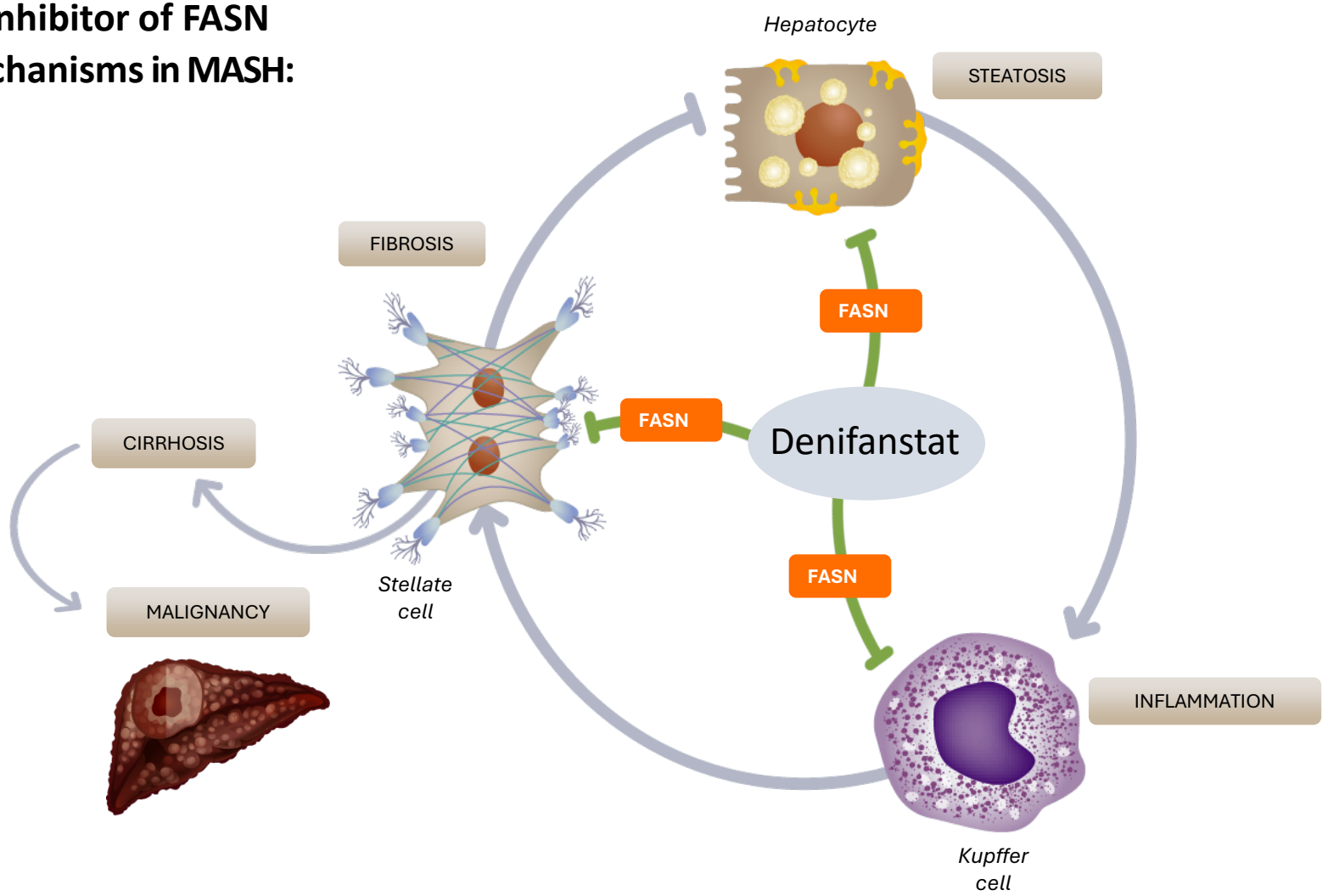
Denifanstat in MASH

FASN Inhibition Addresses Three Independent Mechanisms of MASH Development and Progression

Denifanstat is a specific and potent oral inhibitor of FASN
It functions through three independent mechanisms in MASH:

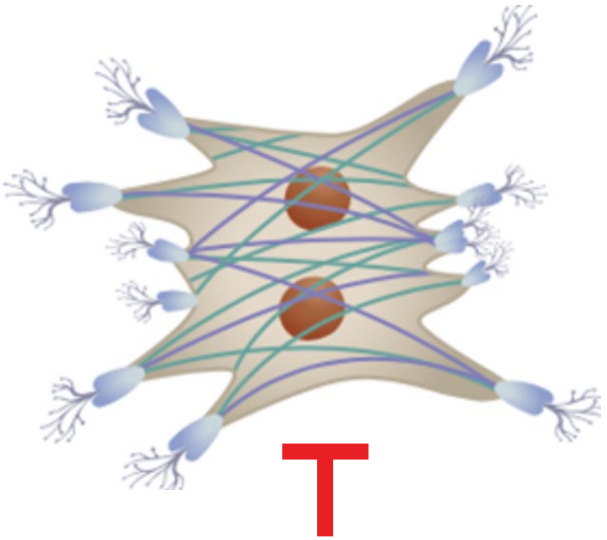
1. Blocks **steatosis** via inhibiting de novo lipogenesis in hepatocytes
2. Reduces **inflammation** via preventing immune cell activation
3. Blunts **fibrosis** via inhibiting stellate cell activation



FASN Inhibition Directly Blocks Human Liver Stellate Cell Function

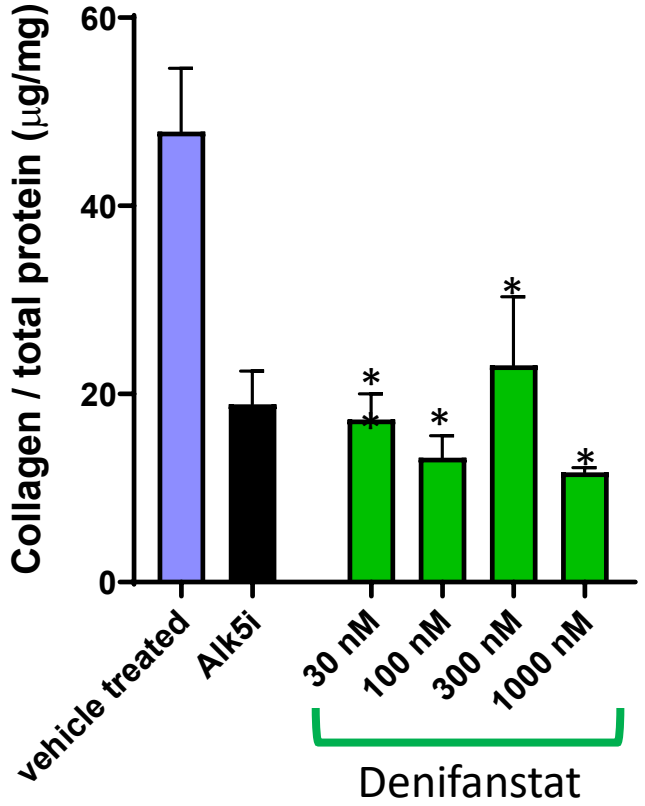
Stellate cell

DNL pathway needed for fibrogenesis



Primary human stellate cells

Denifanstat directly inhibits fibrogenic activity



- *Stimulated by TGF-beta to activate fibrogenesis*
- *Denifanstat similar inhibition to +ve control ALK5 inhibitor*

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

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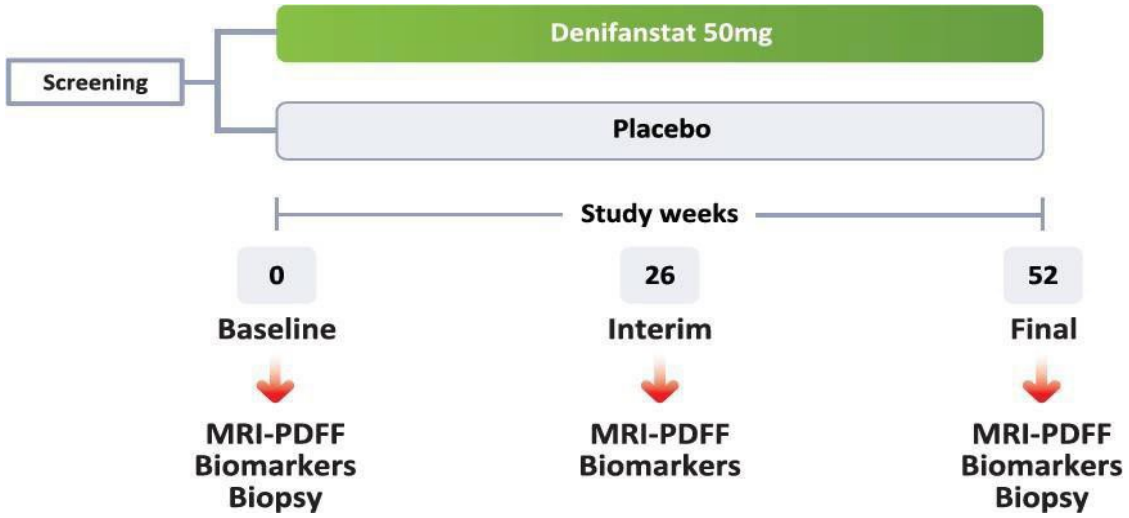
FASCINATE-2 Phase 2b Biopsy Trial Design
Measuring Histological Improvement

FASCINATE-2 Phase 2b

Aim of this trial: Examine safety and efficacy of denifanstat vs placebo in improving fibrosis and NASH resolution after 52 weeks of treatment

- Biopsy confirmed F2-F3 MASH patients
- Randomized 2:1 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

OR

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

Other selected endpoints

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

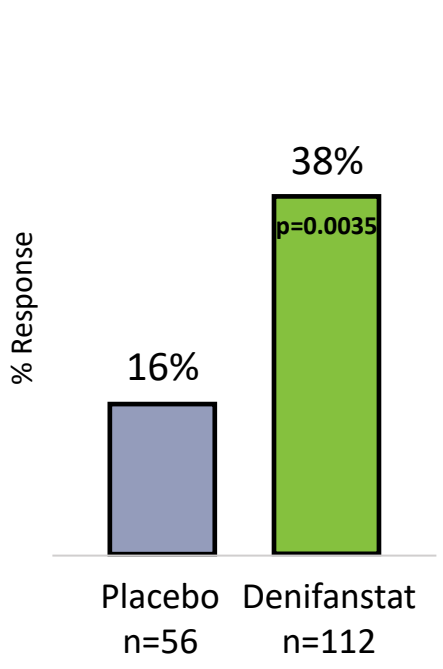
AI: Artificial Intelligence, Bx; biopsy, MRI-PDFF; magnetic resonance imaging derived proton density fat fraction, NAS; NAFLD Activity Score.

FASCINATE-2 Baseline Characteristics
Typical F2/F3 MASH ITT Population

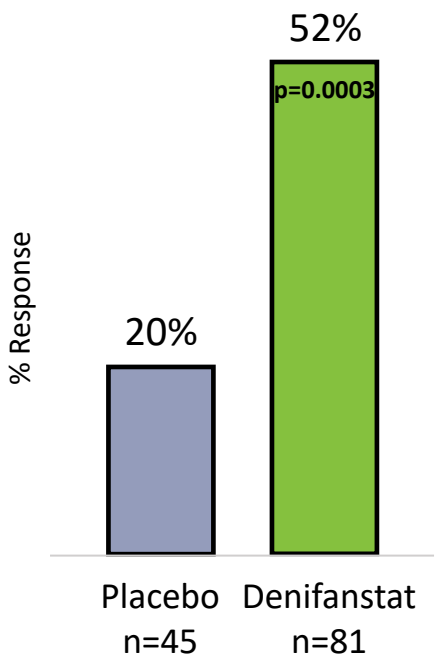
Characteristic	Placebo (n=56)	Denifanstat 50mg (n=112)	Total (n=168)
Age – yr	58·4±11·9	56·3±10·5	57·0±11·0
Male sex – no. (%)	22 (39·3)	46 (41·1)	68 (40·5)
White race – no. (%)	50 (89·3)	100 (89·3)	150 (89·3)
Hispanic or Latino ethnic group – no. (%)	21 (37·5)	34 (30·4)	55 (32·7)
Body mass index	36·2±6·6	34·4±5·8	35·0±6·1
Type 2 diabetes – no. (%)	34 (60·7)	69 (61·6)	103 (61·3)
Alanine aminotransferase – U/liter	64·5±35·4	50·5±25·1	55·2±29·6
Aspartate aminotransferase – U/liter	51·8±30·8	41·9±22·7	45·2±26·0
F2	27 (48·2)	48 (42·9)	75 (44·6)
F3	29 (51·8)	64 (57·1)	93 (55·4)
Liver fat (MRI-PDFF) - %	18·8±6·9	16·8±7·2	17·5±7·2
Liver fat (Fibroscan CAP)	344·9±35·7	336·5±36·4	339·3±36·3
Liver Stiffness - kPa	12·2±4·6	11·2±3·9	11·6±4·2
FAST Score	0·6±0·2	0·6±0·2	0·6±0·2
LDL-cholesterol – mg/dL	103·1±38·9	93·3±37·9	96·5±38·4
Triglycerides – mg/mL	176·6±152·2	170·2±82·9	172·3±110·4

Primary Endpoints: Liver Biopsy
Denifanstat Achieved Statistical Significance at 52 Weeks

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

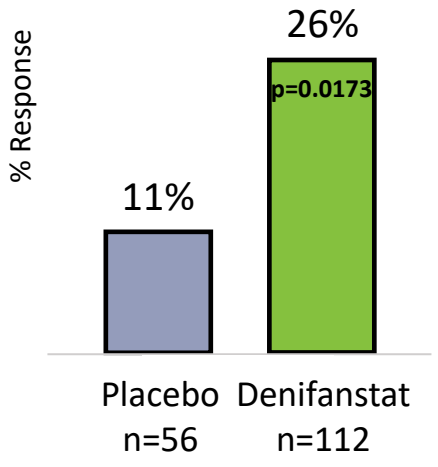


ITT population

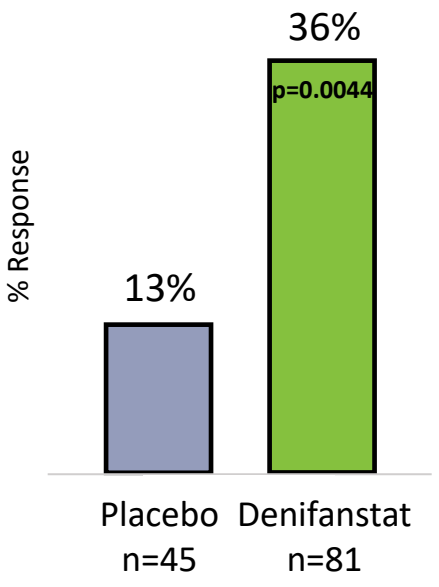


mITT population

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



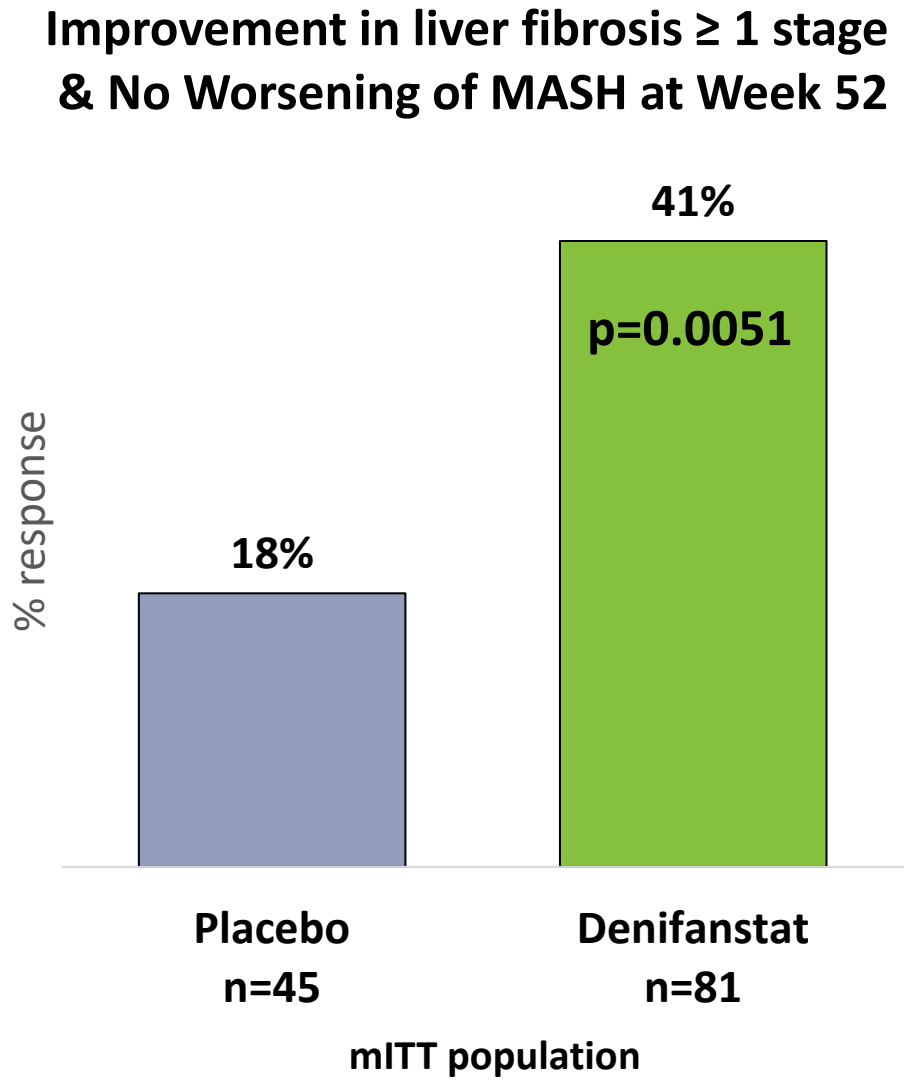
ITT population



mITT population

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

Secondary Endpoint: Liver Fibrosis
Denifanstat Achieved Statistical Significance



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

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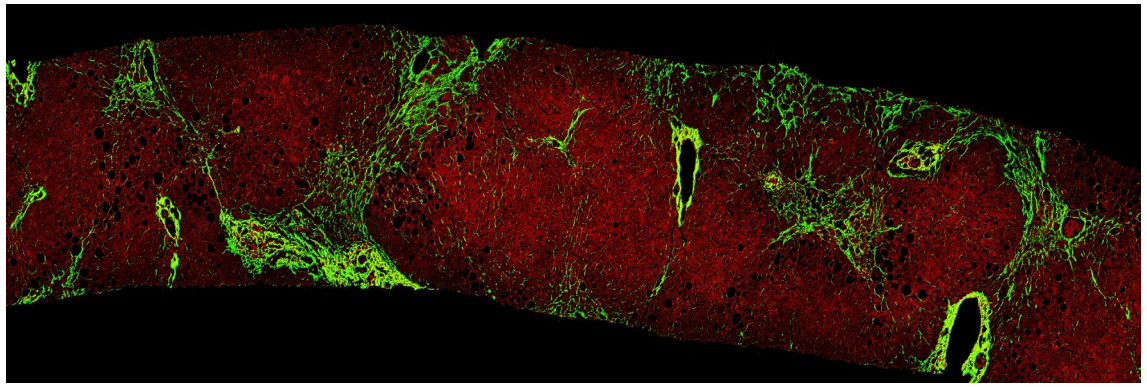
Secondary Endpoints: Liver Fibrosis

Denifanstat Achieved Profound Improvement of Fibrosis

Fibrosis Endpoints	Subgroup	Placebo	Denifanstat	p-value
≥1 stage improvement in fibrosis w/o worsening of MASH	ITT	14%	30%	0.0199
	mITT	18%	41%	0.0051
	F3	13%	49%	0.0032
≥2 stage improvement in fibrosis w/o worsening of MASH	mITT	2%	20%	0.0065
	F3	4%	34%	0.0050
Progression to cirrhosis (F4)	mITT	11%	5%	>0.05

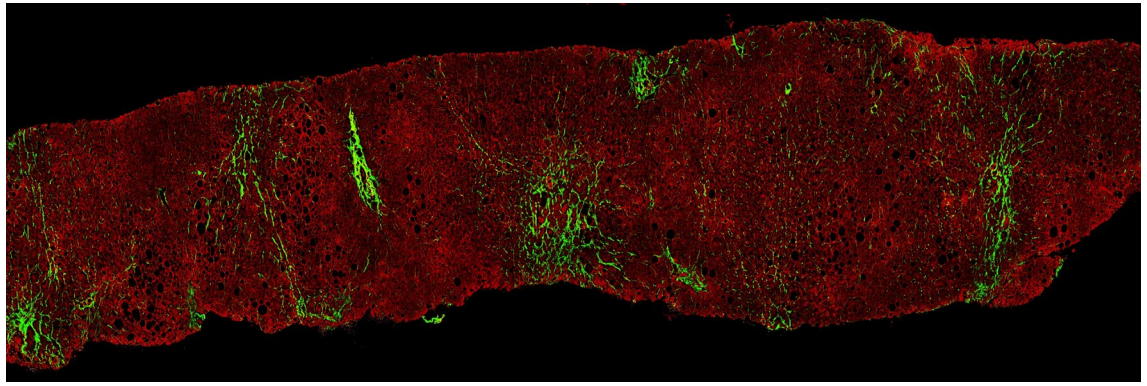
Additional Fibrosis Analysis Using AI-based Digital Pathology
Supporting Evidence that Denifanstat Significantly Reduced Fibrosis

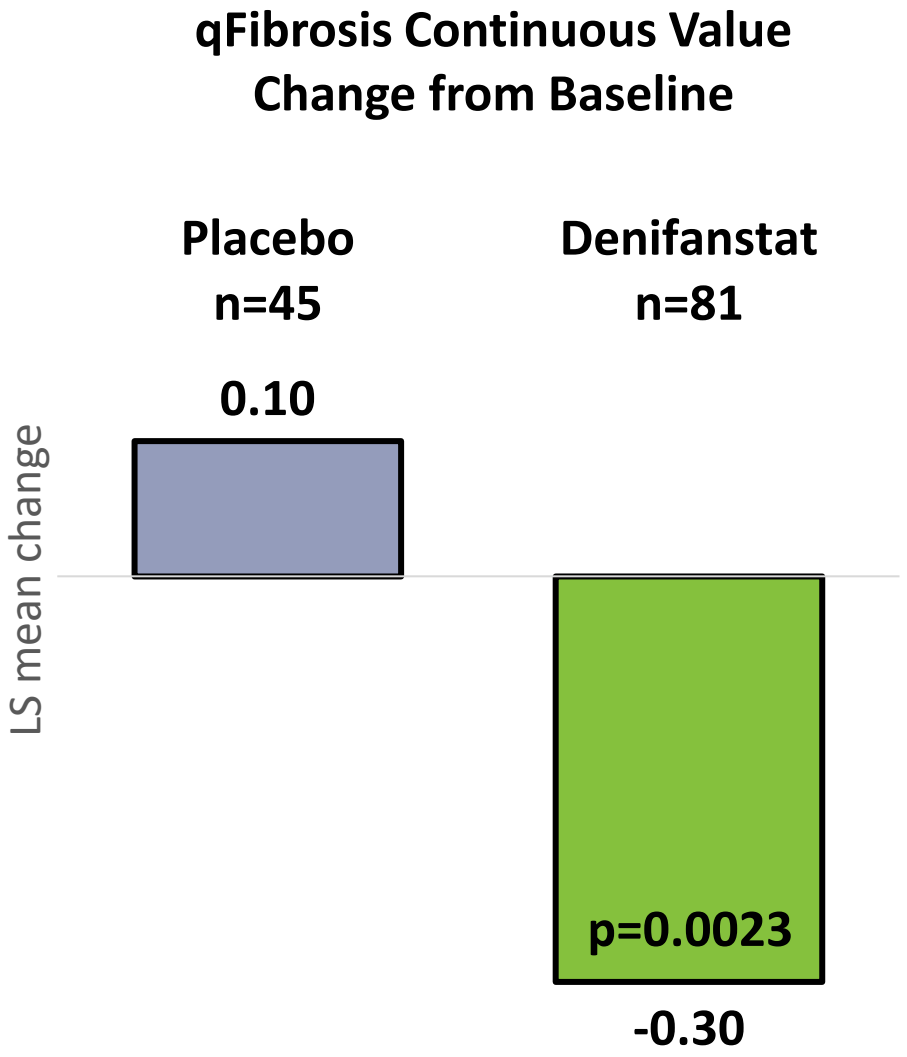
Pre-Treatment Pt A
NASH-CRN Fibrosis stage F3



Denifanstat

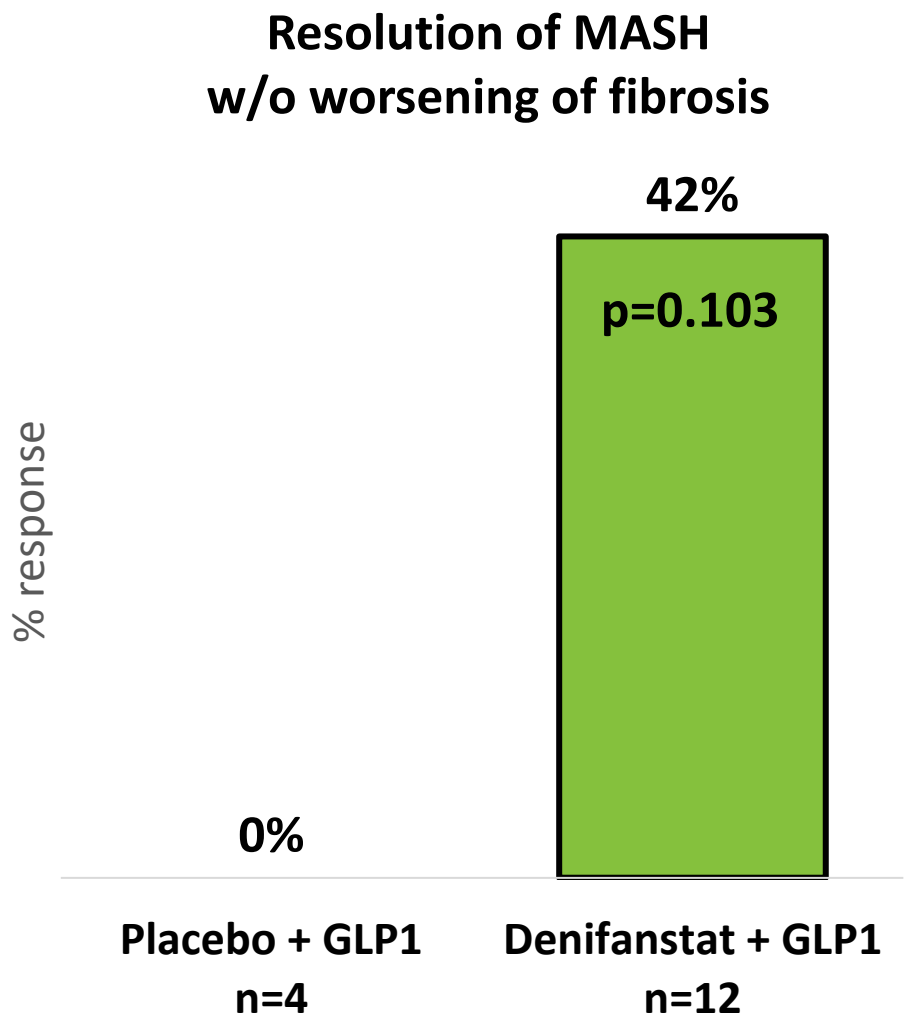
Post-Treatment Pt A
NASH-CRN Fibrosis stage F1

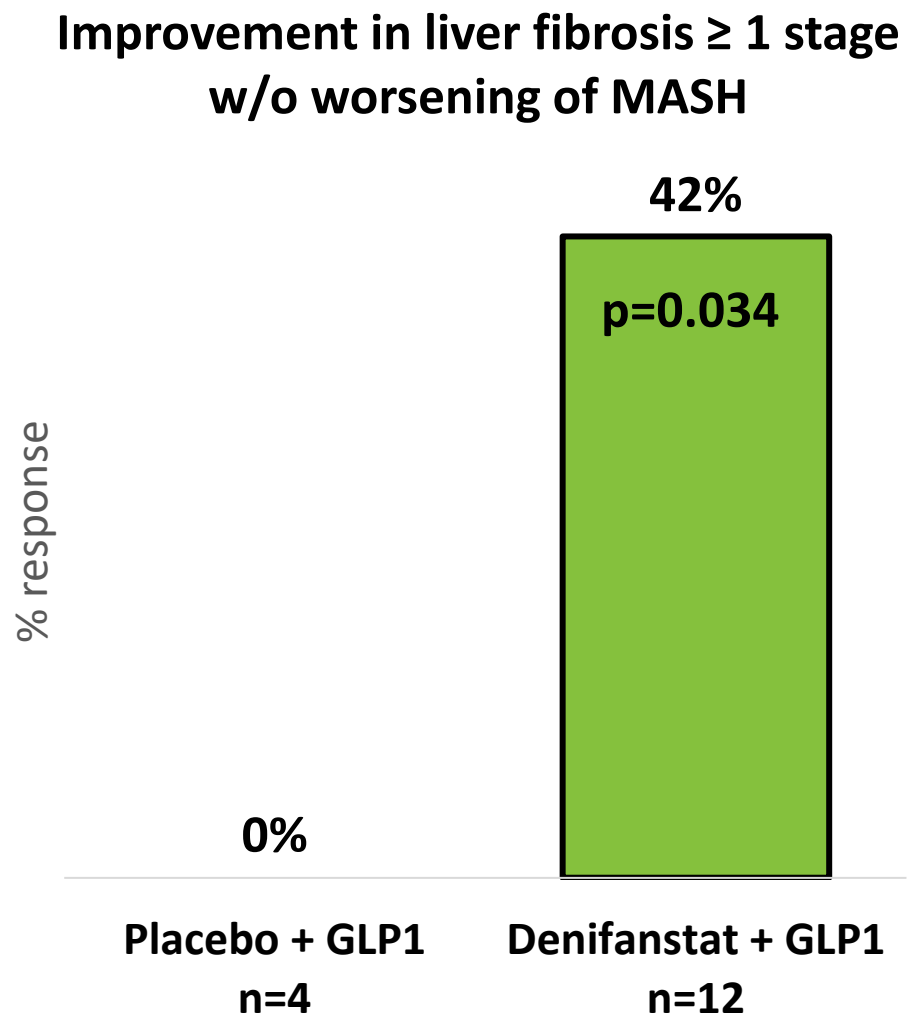




Patient Subset on Stable GLP1-RA at Baseline: Liver Biopsy

Denifanstat Improves 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

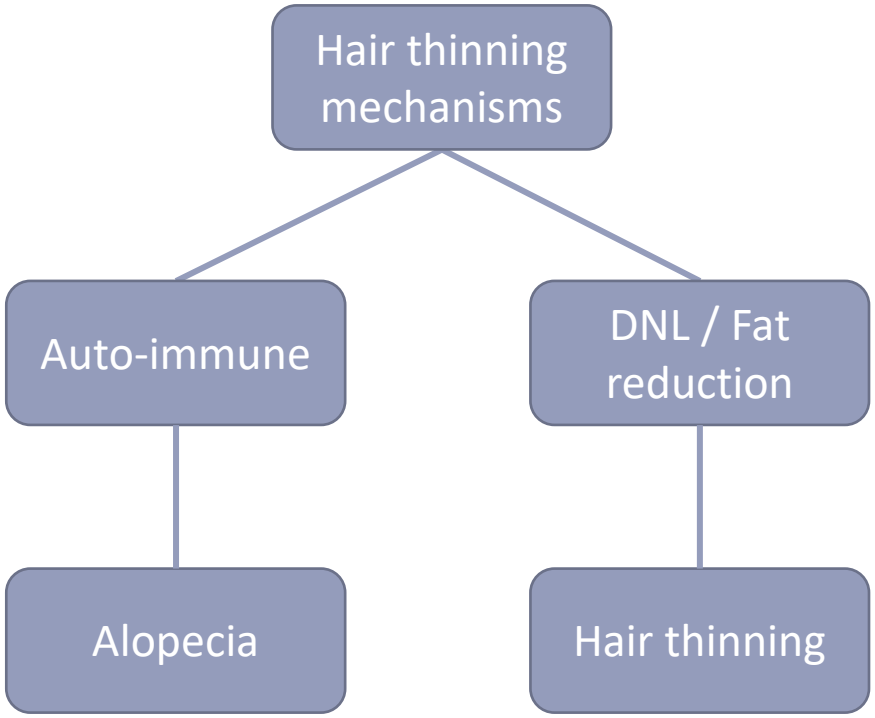
AI digital pathology results also supports fibrosis improvement in patients with GLP1 and denifanstat

FASCINATE-2: Safety
Denifanstat Was Generally Well Tolerated

Event n (%)	Placebo (n=56)	Denifanstat 50mg (n=112)	Total (n=168)
Any adverse event	46 (82.1)	99 (88.4)	145 (86.3)
Adverse event related to denifanstat or placebo	20 (35.7)	51 (45.5)	71 (42.3)
Serious adverse event	3 (5·4)	13 (11·6)	16 (9·5)
TEAE leading to study drug discontinuation	3 (5·4)	22 (19·6)	25 (14·9)
Adverse events affecting ≥ 10% of patients			
COVID-19	6 (10·7)	19 (17·0)	25 (14·9)
Dry eye	8 (14·3)	10 (8·9)	18 (10·7)
Hair thinning	2 (3·6)	21 (18·8)	23 (13·7)

- No DILI signal and no muscle wasting were detected, and GI were comparable to placebo
- AE of hair thinning stabilized with a 2-4 week dose hold; hair thinning was reversible
  - 6% of patients discontinued from the study with hair thinning
    - Consistent with other MASH-related medications
    - In previous clinical studies, <2% of the patients experienced hair thinning at 50mg

Mitigation of On-target AEs



- Hair thinning was reversible and stabilized with a 2-4 week dose hold / reduction
  - Of 8 patients who experienced hair thinning and remained on denifanstat:
    - 5 remained at 50mg and the hair thinning stabilized
    - 3 down titrated to 25mg and hair thinning reversed
- Phase 3 mitigation planned:
  - Encourage patient retention for the expected small subset of patients who experience hair thinning at 50mg with the use of biotin (prevention) and scalp oils
  - Down-titration to 25mg + medication pause for 2-4 weeks when appropriate

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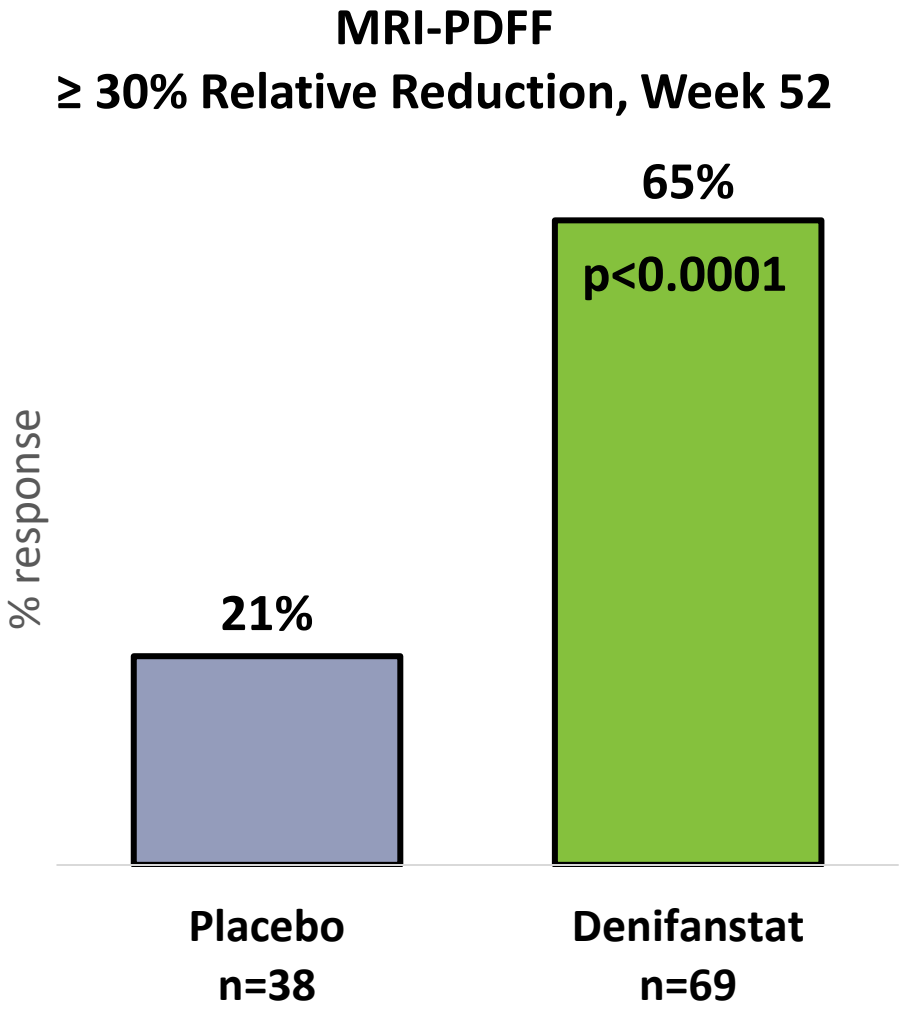
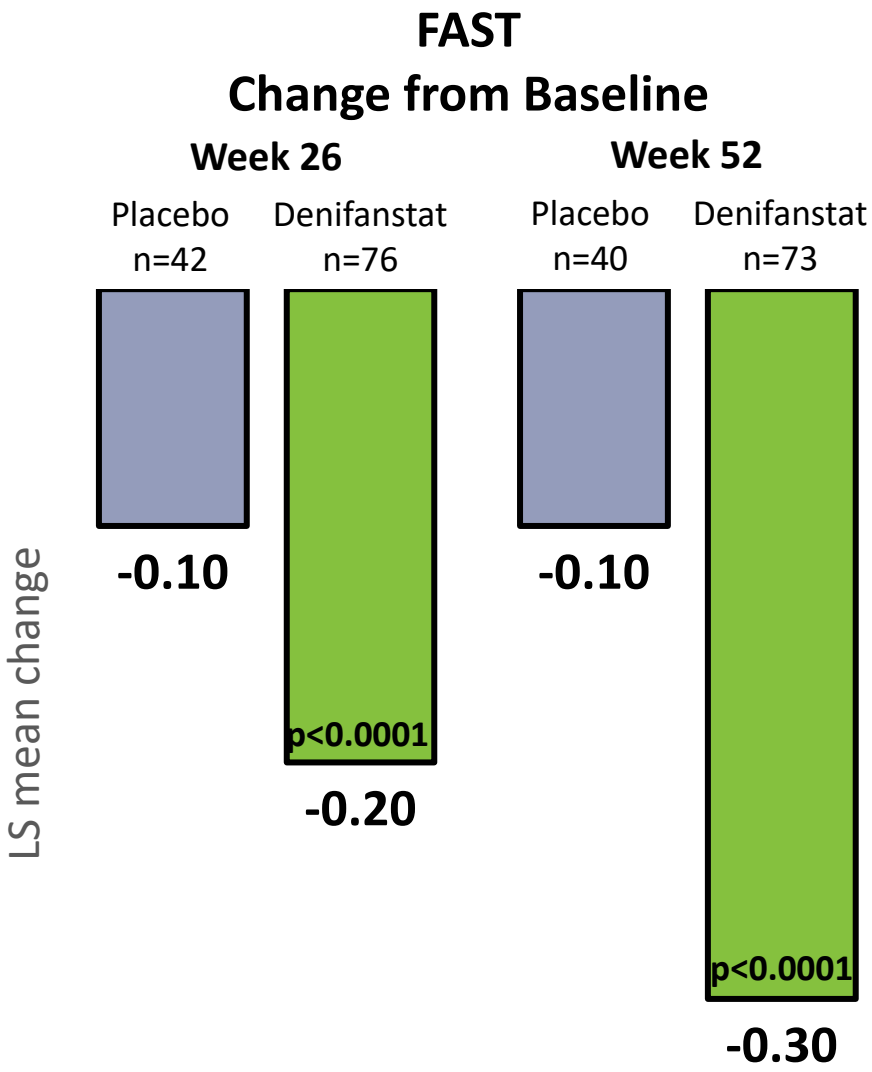
Denifanstat Decreased Liver Fat by MRI-PDFF and Reduced FAST Score

Denifanstat Achieved Statistical Significance

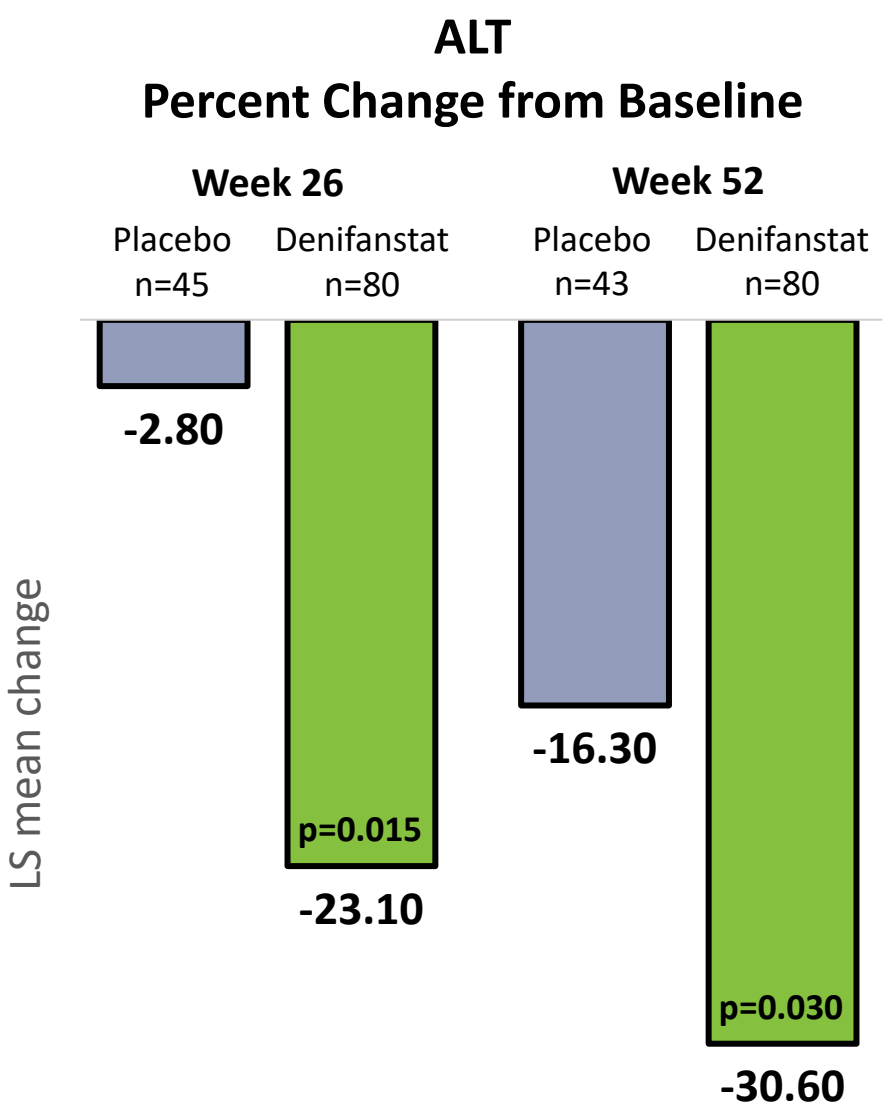


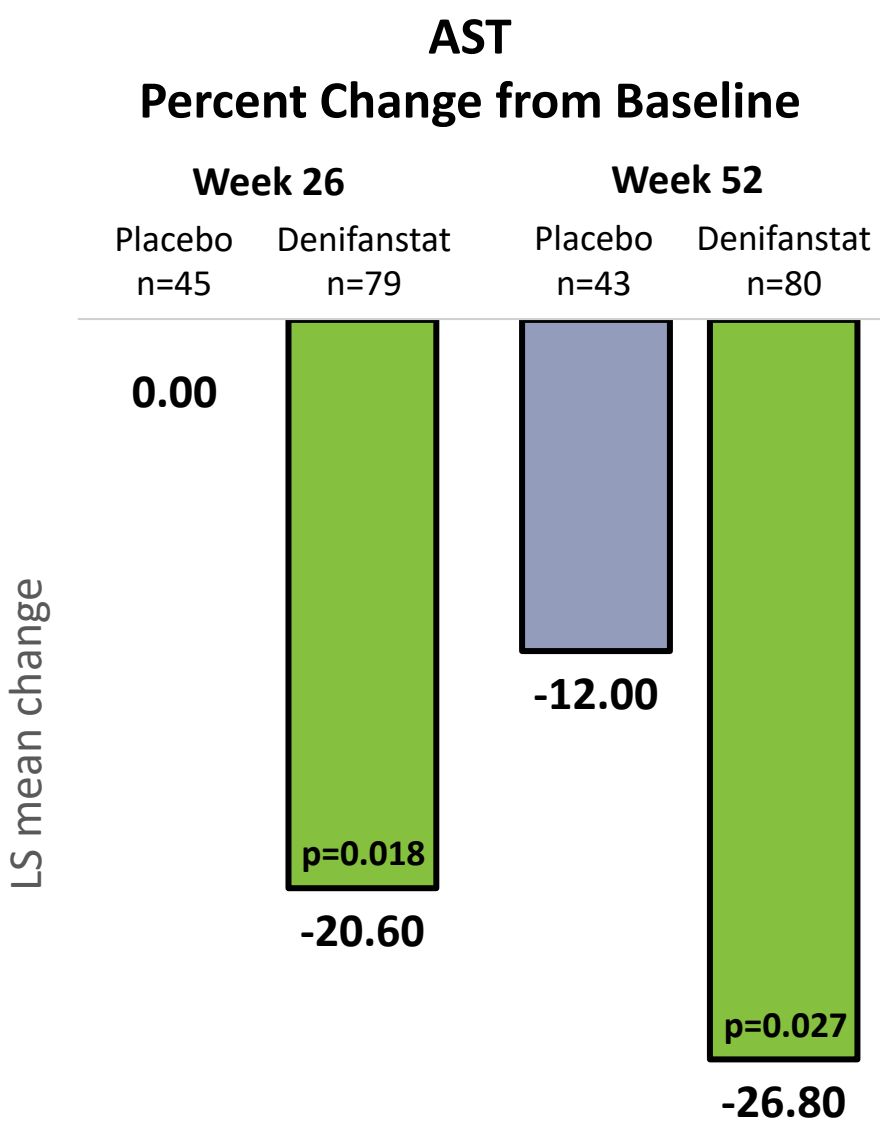


≥30% reduction: Cochran-Mantel-Haenszel Test. Relative reduction: Mixed-effects Model for Repeated Measures . mITT population.

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

Secondary Endpoints: Liver Enzymes
Denifanstat Decreased ALT and AST Levels

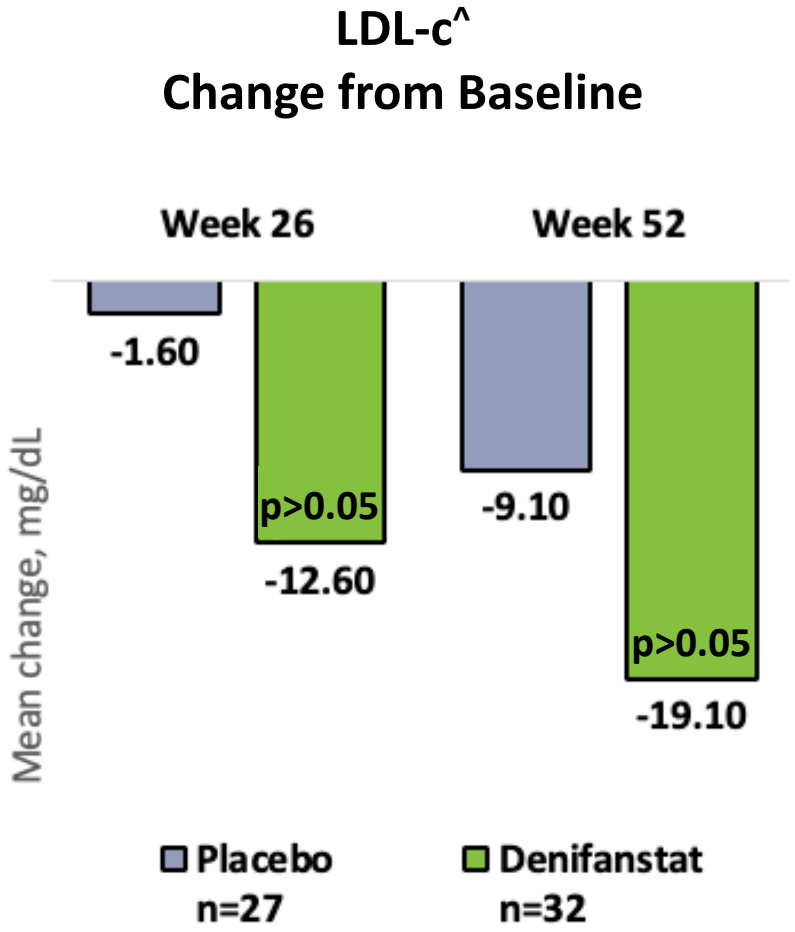


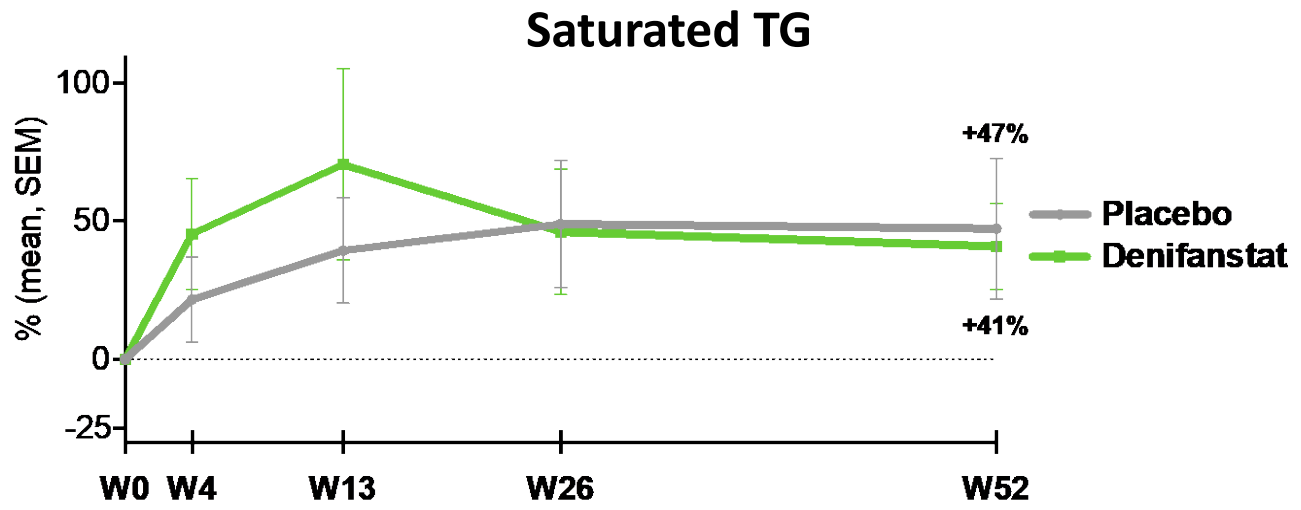


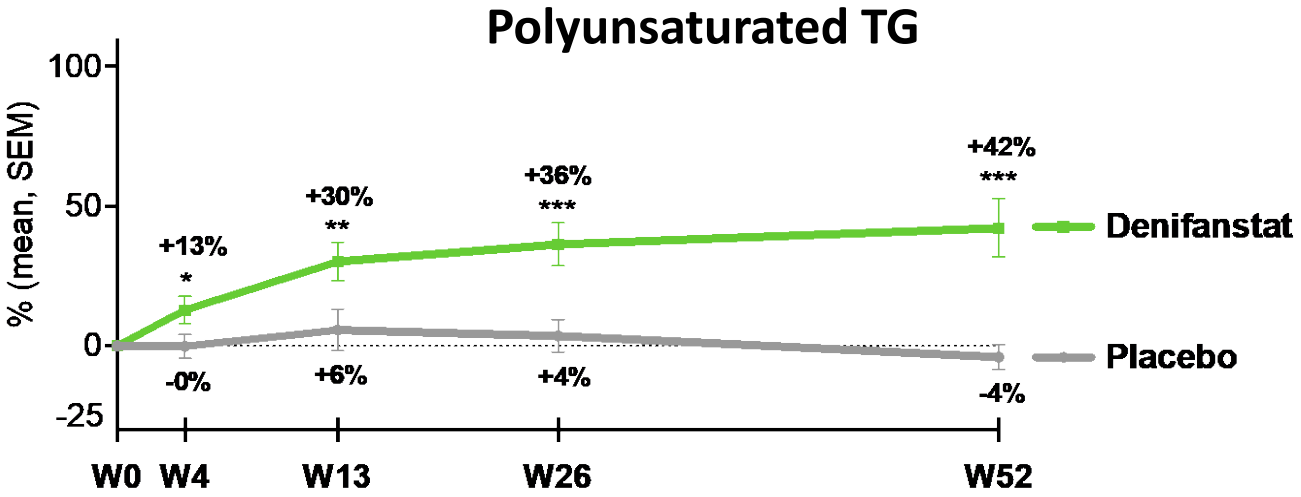
Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. mITT population

Cardiometabolic Health

Denifanstat Decreased LDL-c Levels and Increased Polyunsaturated Triglycerides

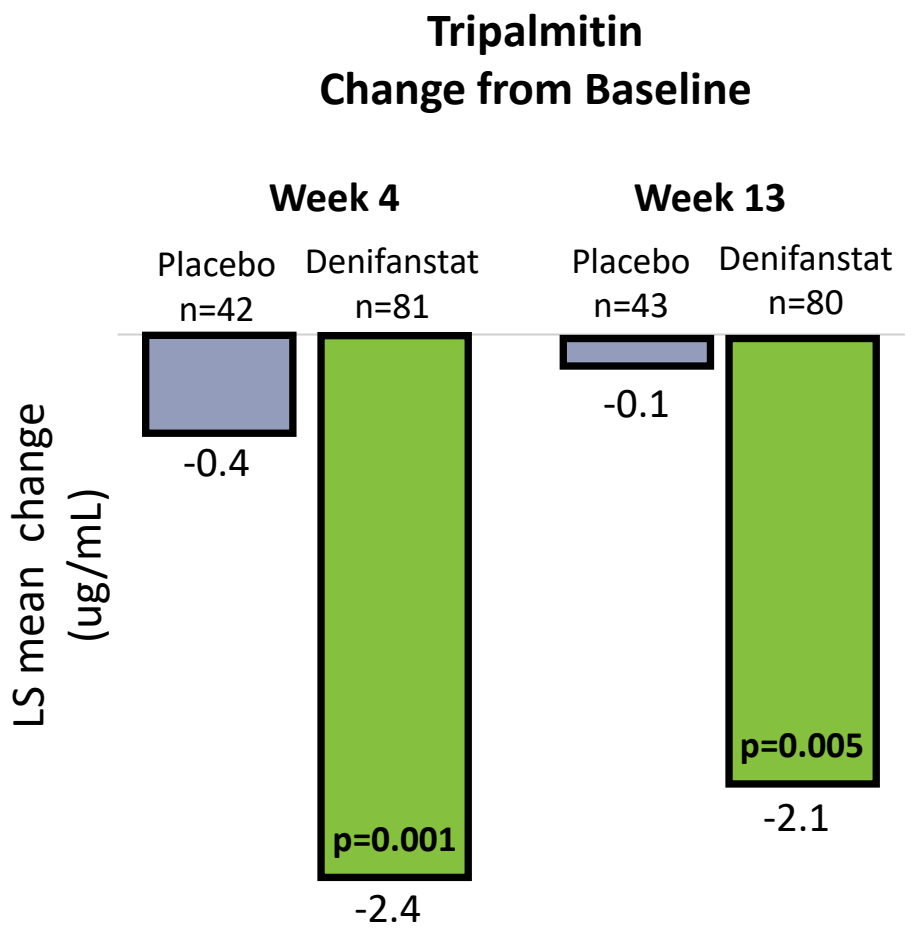






mITT population. ^For LDL-c, baseline > 100 mg/dL. Mixed-effects Model for Repeated Measures – Two sided at the 0.05 significance level. *p<0.05, **p<0.01, ***p<0.001

Denifanstat Rapidly and Robustly Reduces De Novo Lipogenesis



- Tripalmitin, a saturated triglyceride, is a biomarker of DNL inhibition
- Denifanstat rapidly reduced tripalmitin as soon as 4-weeks of treatment
- We plan to continue the development of tripalmitin and additional markers as potential biomarker(s) of treatment response for denifanstat

Denifanstat Fibrosis Improvement in Context

≥1 Stage Improvement in Fibrosis Without Worsening of MASH – in F3 population (placebo adjusted)

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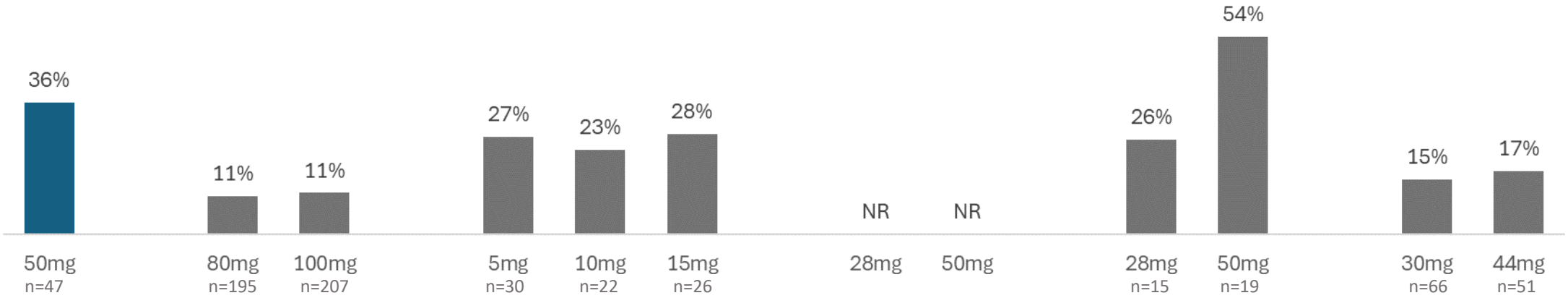
F2-F3 Phase 2b	F1-F3 Phase 3	F2-F3 Phase 2	F2-F3 Phase 2b	F2-F3 Phase 2b
58% F3	62% F3	57% F3	66% F3	65% F3
Single Reader on Completers[1]	Consensus Reading Statistically combined ITT[2]	Consensus Reading Imputed statistics	Consensus Reading on Completers[1]	Consensus Reading on Completers[1]
Denifanstat (oral)[3]	Resmetirom (oral)[4]	Tirzepatide (injection)[5]	Efruxifermin (injection)[6]	Pegozafermin (injection)[7]
52 Week Endpoint	**52 Week Endpoint**	**52 Week Endpoint**	**24 Week Endpoint** / **96 Week Endpoint**	**24 Week Endpoint**



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 cannot be made, and no head-to-head clinical trials have been conducted.

1. Baseline and end-of-study biopsies available
2. Missing biopsies imputed as non-responders
3. Loomba, et al., EASL 2024, Milan Italy (PBO 45 / 23)
4 Harrison, et al. A Phase 3, Randomized, Controlled Trial of Resmetirom in NASH with Liver Fibrosis. NEJM 2024 (PBO 318 /NR)
5. Loomba, et al. Tirzepatide for Metabolic Dysfunction–Associated Steatohepatitis with Liver Fibrosis. NEJM 2024 (PBO 48 / 31)
6. Harrison, et al., Safety and Efficacy of once-weekly efruxifermin versus placebo in non-alcoholic steatohepatitis (HARMONY): a mulicentre, randomised, double-blind, placebo-controlled, phase 2b trial. Lancet Gastroenterology, 2023. (PBO 41/NR) Corporate press release 2024 akerotx.com (PBO 34 / 22)
7. Loomba, et al. Randomized, Controlled Trial of the FGF21 Analogue Pegozafermin in NASH, NEJM 2023 (PBO 61 / 61)

NR (Not Reported)

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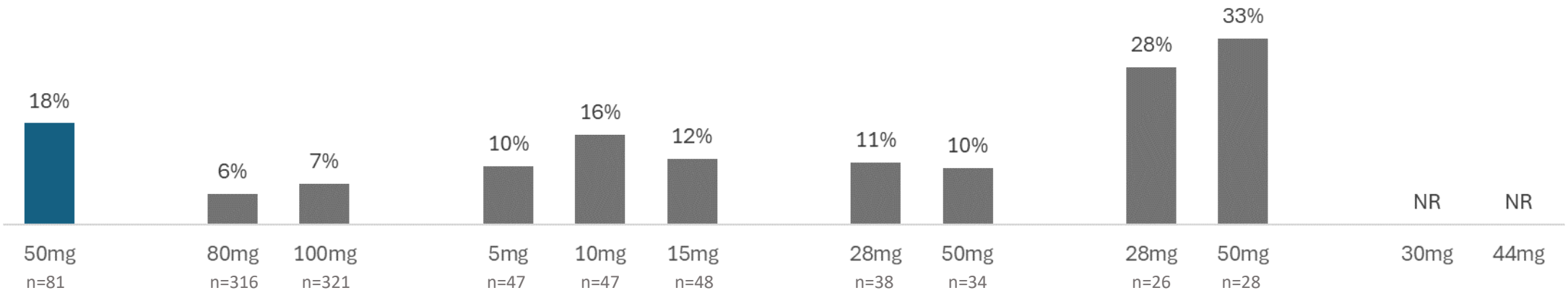
Denifanstat Fibrosis Improvement in Context

≥2 Stage Improvement in Fibrosis Without Worsening of MASH (placebo adjusted)

SAGIMET BIOSCIENCES	Madrigal Pharmaceuticals	Lilly	akero	89bio
F2-F3 Phase 2b	F1-F3 Phase 3	F2-F3 Phase 2	F2-F3 Phase 2b	F2-F3 Phase 2b
58% F3	62% F3	57% F3	66% F3	65% F3
Single Reader on Completers[1]	Consensus Reading Statistically combined ITT[2]	Consensus Reading Imputed statistics	Consensus Reading on Completers[1]	Consensus Reading on Completers[1]
Denifanstat (oral)[3]	Resmetirom (oral)[4]	Tirzepatide (injection)[5]	Efruxifermin (injection)[6]	Pegozafermin (injection)[7]
52 Week Endpoint	**52 Week Endpoint**	**52 Week Endpoint**	**24 Week Endpoint** / **96 Week Endpoint**	**24 Week Endpoint**



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 cannot be made, and no head-to-head clinical trials have been conducted.

1. Baseline and end-of-study biopsies available
2. Missing biopsies imputed as non-responders
3. Loomba, et al., EASL 2024, Milan Italy (PBO 45 / 23)
4 Harrison, et al. A Phase 3, Randomized, Controlled Trial of Resmetirom in NASH with Liver Fibrosis. NEJM 2024 (PBO 318 /NR)
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NR (Not Reported)

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EASL Guidelines: Treatment Recommendations Beyond Lifestyle Modification

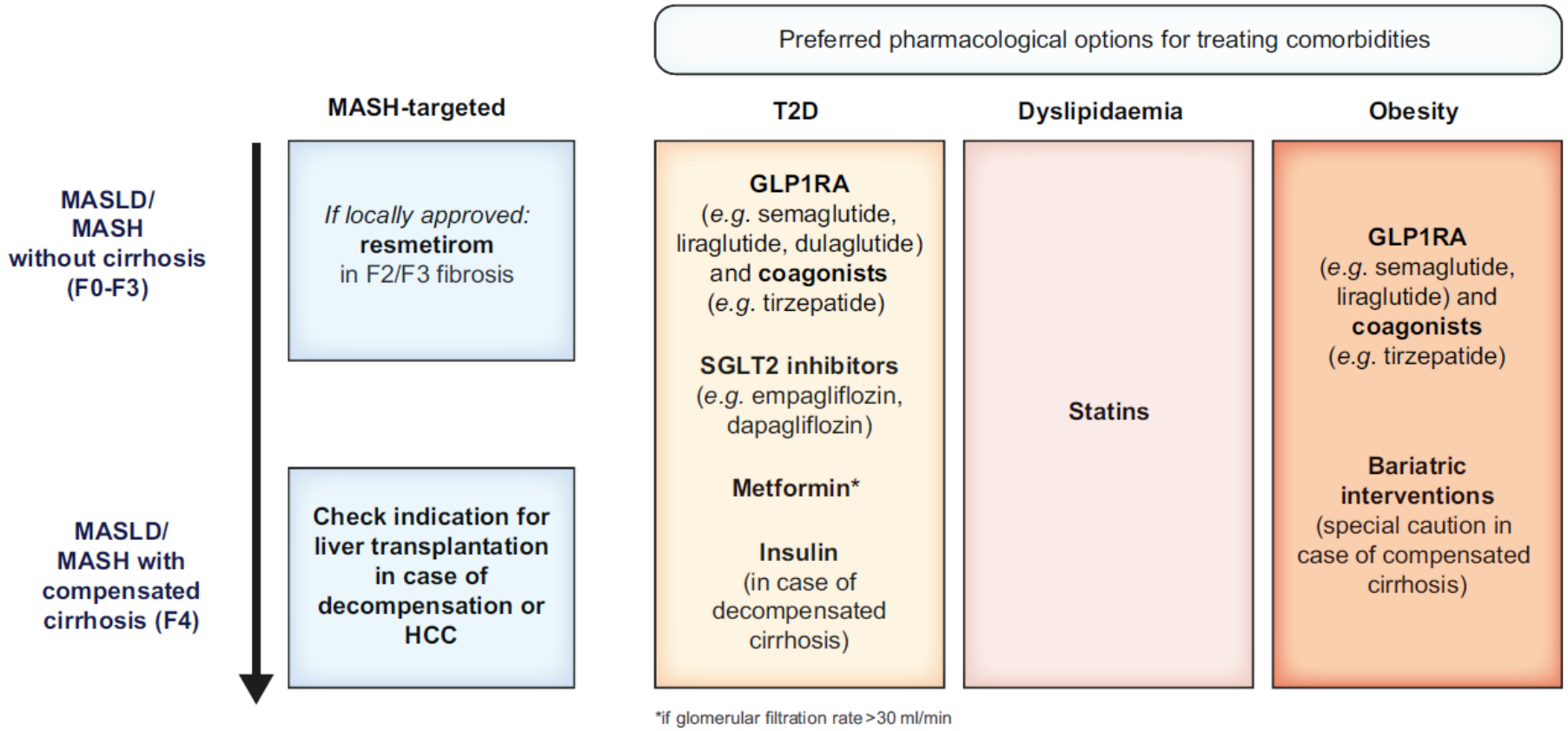
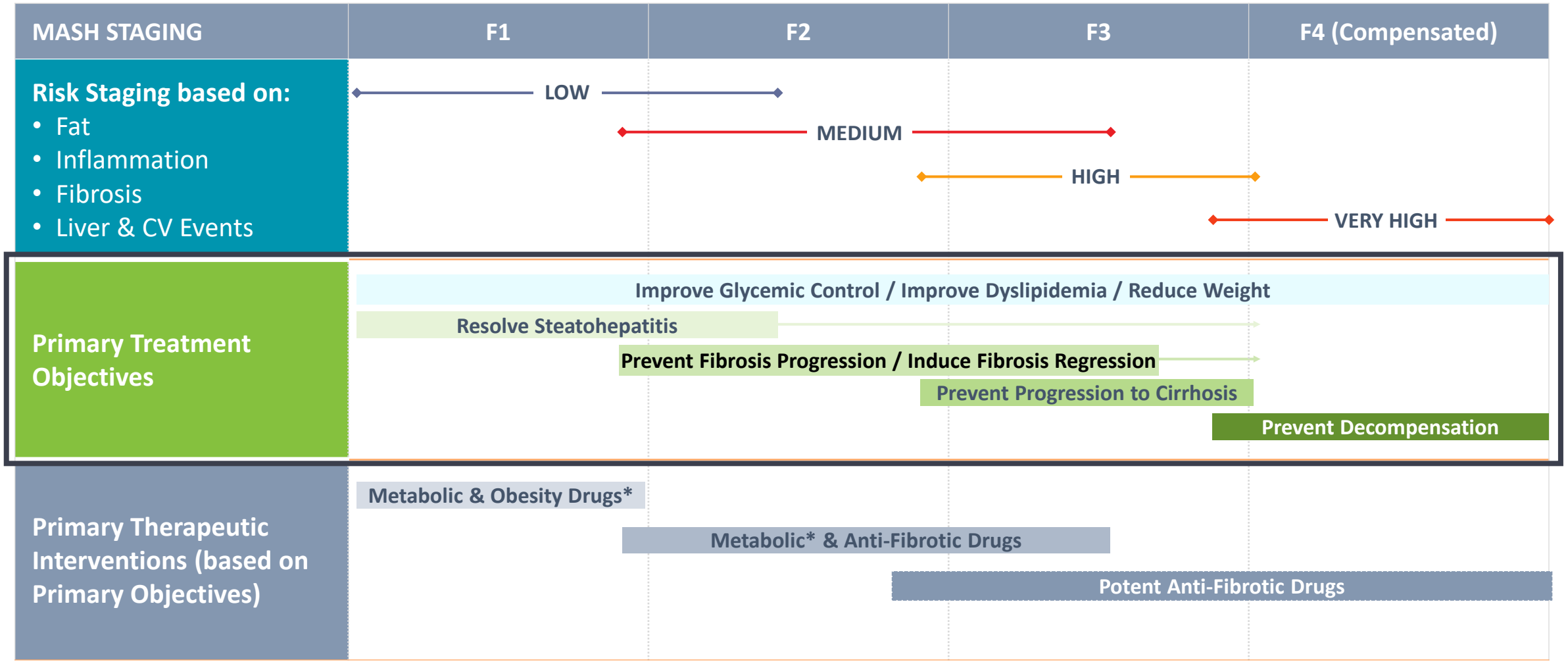


Fig. 4. Treatment recommendations beyond lifestyle modification in MASLD/MASH. The recommended choice of pharmacological treatment options in individuals with MASLD/MASH is dependent on comorbidities and stage of disease. GLP1RA, glucagon-like peptide 1 receptor agonist; HCC, hepatocellular carcinoma; MASH, metabolic dysfunction-associated steatohepatitis; MASLD, metabolic dysfunction-associated steatotic liver disease; SGLT2, sodium-glucose cotransporter 2; T2D, type 2 diabetes.

EASL–EASD–EASO Clinical Practice Guidelines on the management of metabolic dysfunction-associated steatotic liver disease (MASLD); Journal of Hepatology; https://doi.org/10.1016/j.jhep.2024.04.031

Treatment Goal for MASH Across Fibrosis Staging



*Metabolic drugs are anticipated to be background therapy for obesity and type 2 diabetes, until clinical data support use in MASH

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In Summary

- Denifanstat, a fatty acid synthase inhibitor, was better than placebo for both the subpart H approval pathway endpoint(s) including
  - MASH resolution without worsening of fibrosis
  - Fibrosis improvement without worsening of MASH
- Denifanstat delivered clinically meaningful and statistically significant improvements in liver histology
  - Fibrosis regression: 2-stage fibrosis improvement as well as significant improvement in F3 patients
- Improvements in MRI-PDFF, FAST, ALT, AST and LDL were demonstrated
- Tripalmitin is being developed as an early biomarker of target engagement and treatment response
- Denifanstat was generally well tolerated
- Combination of a fat synthesis inhibitor with a fat burner synergistically improves outcomes of disease
- These results support continued clinical development of denifanstat to Phase 3 clinical trials in MASH

MASH Development Program

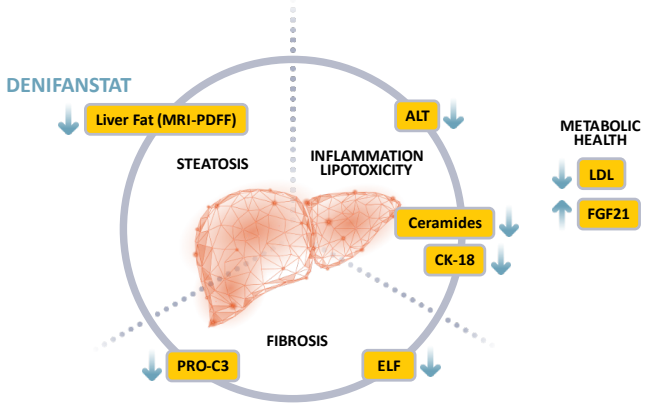
Progression from Phase 2b to Phase 3

Phase 2b – baseline
Fibrosis stage

Interim cohort
F2 – 46.2%
F3 – 53.8%

Enrollment completed Sep 2022

Phase 2b – 26 weeks
Non-invasive interim



Interim results released Nov 2022

Phase 2b – 52 weeks
Histology

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

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

Topline data released Jan 2024

Phase 3
Fibrosis endpoint - human

Using Phase 2b results including AI pathology scores to design and power Phase 3

MASH Phase 3 study planned to start 2H 2024

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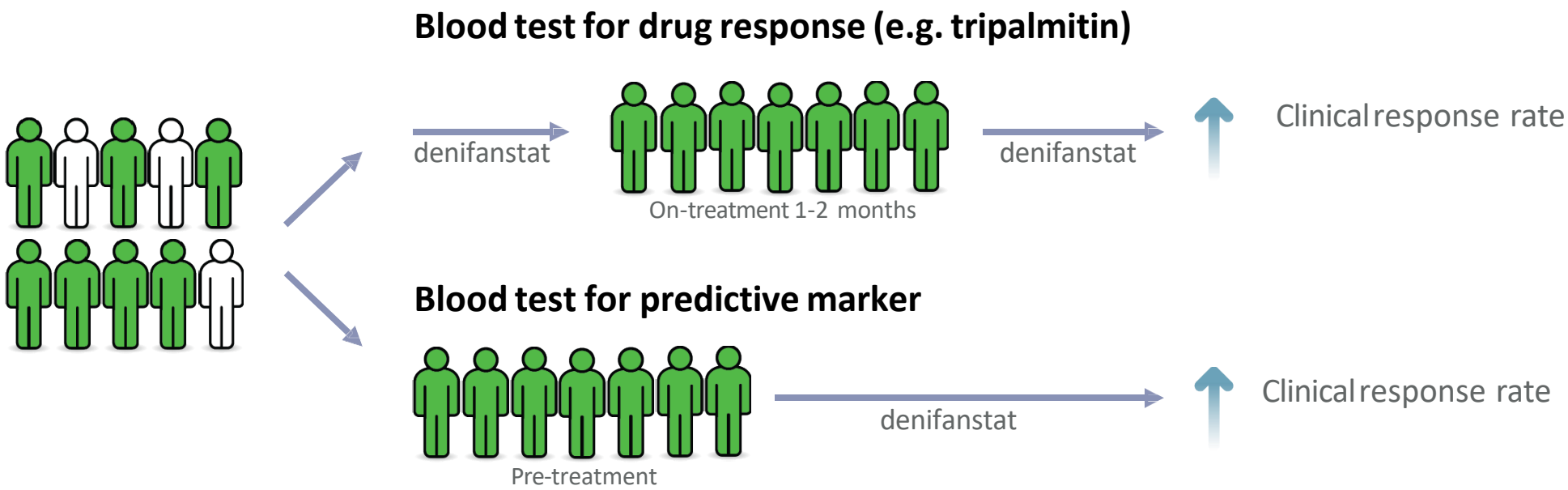
Precision Medicine: Blood Tests May Lead to Improved Patient Outcomes

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



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

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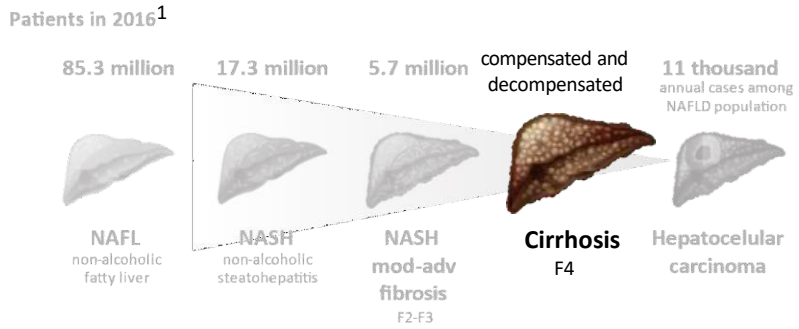
Additional Expansion Opportunities in MASH

- **Compensated cirrhotic patients (MASH F4)**
  - Denifanstat directly targets stellate cells
  - Hepatocytes continue to be functional, and patients frequently have increased liver fat
  - Completed hepatic impairment study supports development in F4 patients
  - Next steps
    - Positive impact on fibrosis in FASCINATE-2
    - Phase 2b/3 trial in MASH-F4



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

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

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Preclinical Combination of FASNi with Resmetirom – 2 Posters at EASL 2024

Complementary mechanisms lead to increased efficacy

Hypothesis: combination of complementary distinct mechanisms can further increase efficacy in MASH

Resmetirom	→	increase liver fat breakdown
FASN inhibitor	→	decrease liver fat synthesis
FASN inhibitor	→	directly inhibit fibrosis by stellate cell

Model 1: LDLr K/O for MASH and cardiovascular

FASNi + Resmetirom
12 week treatment

ALT
AST
LDL
Lipoproteins

Combination showed superior efficacy on liver health and CV biomarkers

Model 2: Diet Induced Obesity model (Gubra)

FASNi + Resmetirom
6 or 12 week treatment

Biomarkers
Liver histology

Includes early timepoint of 6 weeks to test kinetics of combination

Liver histology results were presented for both models

***Regular Poster session**: June 6, Abstract 1326, THU231*
*Combination of fatty acid synthase (FASN) inhibitor and thyroid hormone receptor beta (THRb) agonist, resmetirom, **improved markers of NASH and cardiovascular health in LDL receptor knockout NASH mice.***
Presented by Wen-Wei Tsai et al.

***Late breaker poster session:** June 6, Abstract LB235, THU336.*
*Combination of fatty acid synthase (FASN) inhibitor and thyroid hormone receptor beta (THRb) agonist resmetirom shows **synergistic improvement of NAFLD activity score (NAS) within 6-weeks in diet-induced obese mice with biopsy-confirmed MASH***
Presented by Wen-Wei Tsai et al.

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Secondary Endpoints: Liver Fibrosis

Denifanstat Achieved Profound Improvement of Fibrosis

Fibrosis Endpoints	Subgroup	Placebo	Denifanstat	p-value
≥1 stage improvement in fibrosis w/o worsening of MASH	ITT	14%	30%	0.0199
	mITT	18%	41%	0.0051
	F3	13%	49%	0.0032
≥2 stage improvement in fibrosis w/o worsening of MASH	mITT	2%	20%	0.0065
	F3	4%	34%	0.0050
Progression to cirrhosis (F4)	mITT	11%	5%	>0.05

Q&A Session