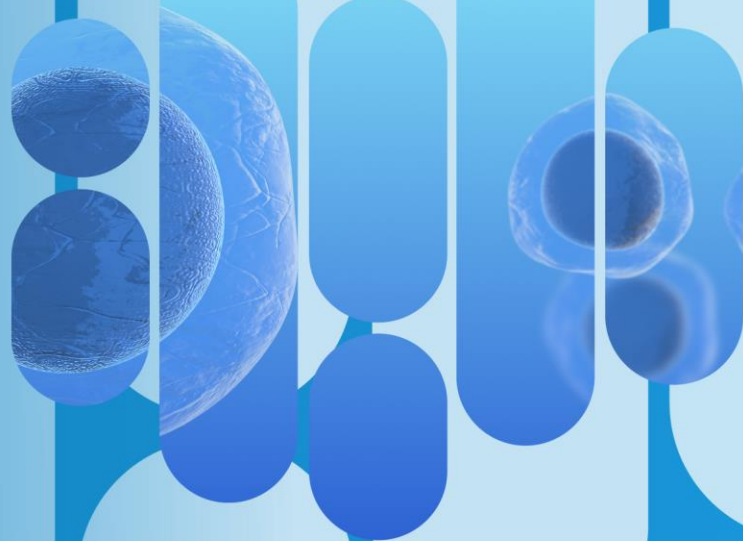


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Nasdaq: ETNB

July 2022



Disclaimer



Cautionary Note Regarding Forward-Looking Statements

This presentation contains “forward-looking statements” within the meaning of the federal securities laws, which statements are subject to substantial risks and uncertainties and are based on estimates and assumptions. Other than statements of historical facts, all statements included in this presentation are forward-looking statements, including statements concerning our plans, objectives, goals, strategies, future events, plans or intentions relating to product candidates, potential market opportunities, estimates of market size, estimates of market growth, the potential clinical benefit, effect on histology and safety and tolerability profile of pegozafermin (formerly BIO89-100), the clinical potential of pegozafermin, potential indications for pegozafermin, the association of clinical data with potential clinical benefit in other indications, the anticipated timing, design, endpoints, and conduct of our ongoing clinical trials for pegozafermin, the timing of anticipated milestones, the timing and likelihood of success, plans and objectives of management for future operations and future results of anticipated product development efforts and our liquidity and capital resources. In some cases, you can identify forward-looking statements by terms such as “may,” “might,” “will,” “objective,” “intend,” “should,” “could,” “can,” “would,” “expect,” “believe,” “design,” “estimate,” “predict,” “potential,” “plan” or the negative of these terms, and similar expressions intended to identify forward-looking statements. These statements involve known and unknown risks, uncertainties and other factors that could cause our actual results to differ materially from the forward-looking statements expressed or implied in this presentation including those described more fully in our most recent Form 10-K and Form 10-Q under the caption “Risk Factors” and elsewhere in such report and in other subsequent disclosure documents filed with the SEC.

We cannot assure you that we will realize the results, benefits or developments that we expect or anticipate or, even if substantially realized, that they will result in the consequences or affect us or our business in the way expected. Forward-looking statements are not historical facts, and reflect our current views with respect to future events. Given the significant uncertainties, you should evaluate all forward-looking statements made in this presentation in the context of these risks and uncertainties and not place undue reliance on these forward-looking statements as predictions of future events. All forward-looking statements in this presentation apply only as of the date made and are expressly qualified in their entirety by the cautionary statements included in this presentation. We disclaim any intent to publicly update or revise any forward-looking statements to reflect subsequent events or circumstances, except as required by law.

We obtained the industry, market and competitive position data used throughout this presentation from our own internal estimates and research, as well as from industry and general publications, and research, surveys and studies conducted by third parties. Internal estimates are derived from publicly available information released by industry analysts and third-party sources, our internal research and our industry experience, and are based on assumptions made by us based on such data and our knowledge of the industry and market, which we believe to be reasonable. In addition, while we believe the industry, market and competitive position data included in this presentation is reliable and based on reasonable assumptions, we have not independently verified any third-party information, and all such data involve risks and uncertainties and are subject to change based on various factors.

Corporate Highlights

Pegozafermin – potential important new cardio-metabolic drug; balanced effect on multiple key endpoints

- Impacts a broad spectrum of cardio and metabolic endpoints
- Robust data in two indications with a favorable safety and tolerability profile
- Potential first-to-market FGF21 analog

Severe Hypertriglyceridemia (SHTG) – Ph. 3 initiation planned in 1H23

- Highly differentiated based on Phase 2 success across multiple endpoints
- Significant opportunity given large, under-served market with limited competition
- Established regulatory pathway allows efficient clinical development

Nonalcoholic Steatohepatitis (NASH) – Ph. 2b enrollment completion in 3Q22; data expected 1Q23

- FGF21 offers greatest promise in this category – addresses multiple facets of the disease
- Pegozafermin has demonstrated compelling results in clinic

Strong cash position with experienced team

- \$200.5M* pro forma cash** as of June 30, 2022
- Track record of developing and commercializing successful drugs and business development at other companies

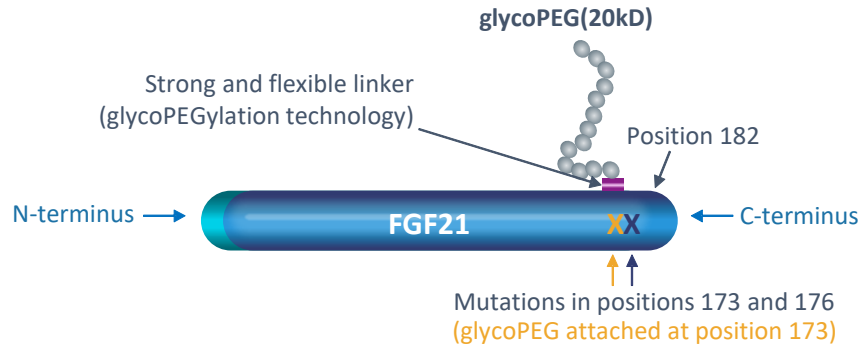
Advancing Pegzofermin in Clinical Development



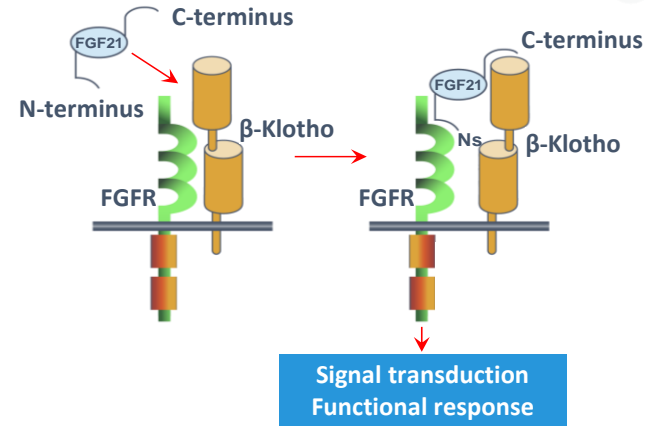
| Indication | Preclinical | Phase 1 | Phase 2 | Phase 3 | Anticipated Milestones |
|-------------|--------------------------|---------|---------|---------|--|
| SHTG | Phase 3 trial - Planning | | | | End of Ph. 2 Meeting – 2H22 Phase 3 trial Initiation – 1H23 |
| NASH | Phase 2b trial | | | | Complete Enrollment – 3Q22 Topline Data – 1Q23 |

ENliven

Pegozafermin Is an FGF21 Analog Optimally Engineered to Balance Efficacy and Long Dosing Interval



* Composition of matter through 2038



- Proprietary glycoPEGylation technology with site-specific mutations
- Increases half-life of native FGF21 (< 2 hours) to 55-100 hours based on single ascending dose study
- Low nanomolar potency against FGF receptors 1c, 2c, 3c, similar to native FGF21
- Composition of Matter patent expiring in 2038

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Opportunity in Severe Hypertriglyceridemia (SHTG)



SHTG Is a Serious Disease in Need of Better Treatment Options



Patients with SHTG have multiple co-morbidities and significant health risks

- Increased Cardiovascular (CV) Risk
 - Increased risk of CV disease, heart failure and ischemic stroke
 - Increased mortality in patients with CHD
- Increased risk of Acute Pancreatitis
- Increased prevalence of Diabetes

“Most patients with severe hypertriglyceridemia have multiple ASCVD risk factors and are at enhanced risk of developing atherosclerotic disease. This risk is conveyed by atherogenic VLDL plus other factors, such as obesity, metabolic syndrome, and hyperglycemia.” [2018 AHA/ACC Guideline For Cholesterol Management]

Average patient in ENTRIGUE study was estimated to have a 23% risk of Major Cardiovascular Events (MACE) over 10 years (per ACC ASCVD risk estimator)

- Average patient: no prior CV event, 54 years old, male, white, BP 131/81, total cholesterol 240, HDL 28, LDL 89, diabetic, non-smoker, taking anti-hypertensive medication, no statins or aspirin. Individual results may vary.

Pegozafermin – Potential Important New Cardio-Metabolic Drug



SHTG represents a large potential opportunity given pegozafermin's profile

- High unmet needs for better therapeutic options to address broader metabolic comorbidities
- Approved therapies are sub-optimal resulting in large uncontrolled or untreated population

ENTRIGUE results demonstrate compelling and differentiated profile in SHTG

- Significant impact on triglycerides, liver fat, and associated metabolic co-morbidities
- Favorable safety and tolerability profile
- Exceeds target product profile defined by literature, KOLs & treating physicians

Results support advancing to Phase 3 – potential first FGF21 analog to market

- Potentially quicker and efficient regulatory path to approval in SHTG
- Phase 3 initiation expected in 1H23; topline results ~ 2 years from study start

ENTRIGUE results increase confidence in overall pegozafermin program, including NASH

Pegozafermin – Unique and Differentiated Profile in SHTG



Efficacy

- Significant reduction in triglycerides across all dose groups – up to 63% (27 mg QW)
 - Results consistent in patients with or without background therapy
 - Result consistent across various subgroups (high/low baseline TGs, Type 2 diabetes status)
 - Significant TG normalization rates
- Significant reduction in liver fat and improvements in liver enzymes and glycemic control markers
- Potent reduction in atherogenic lipids (Non HDL-C) and apolipoproteins (Apo-B)

Safety/Tolerability

- Well tolerated at all doses with low incidence of treatment related adverse events (all Grade 1 or 2)

Proposed Mechanisms of Action for Pegzofermin in SHTG

- **Adipose tissue**

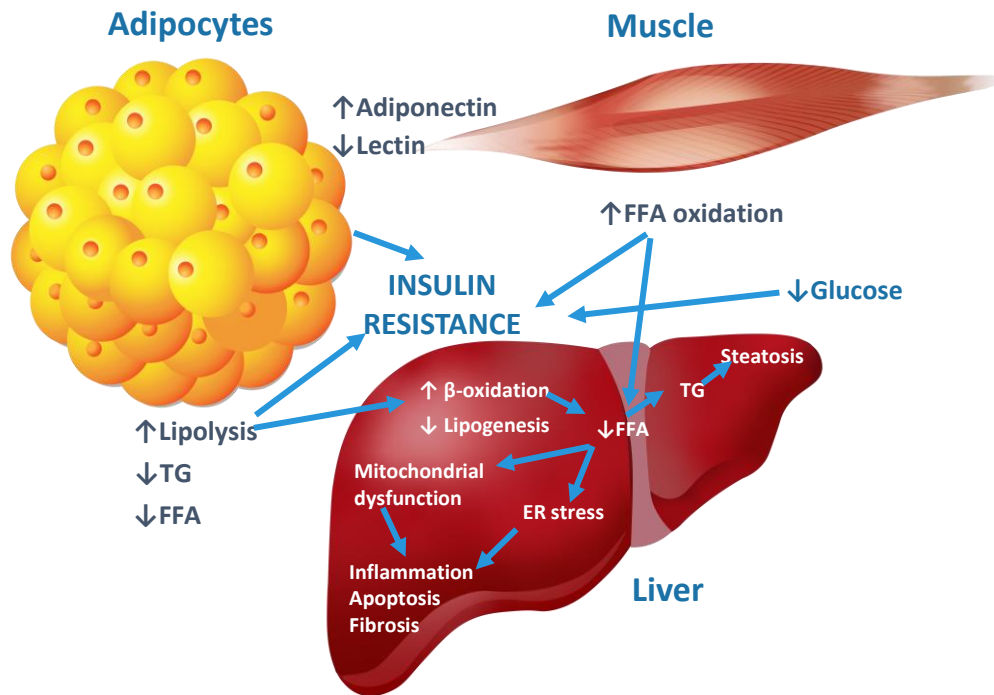
- Decrease lipogenesis and release of FFA
- Improve insulin resistance
- Increase TG uptake
- Increase adiponectin

- **Liver**

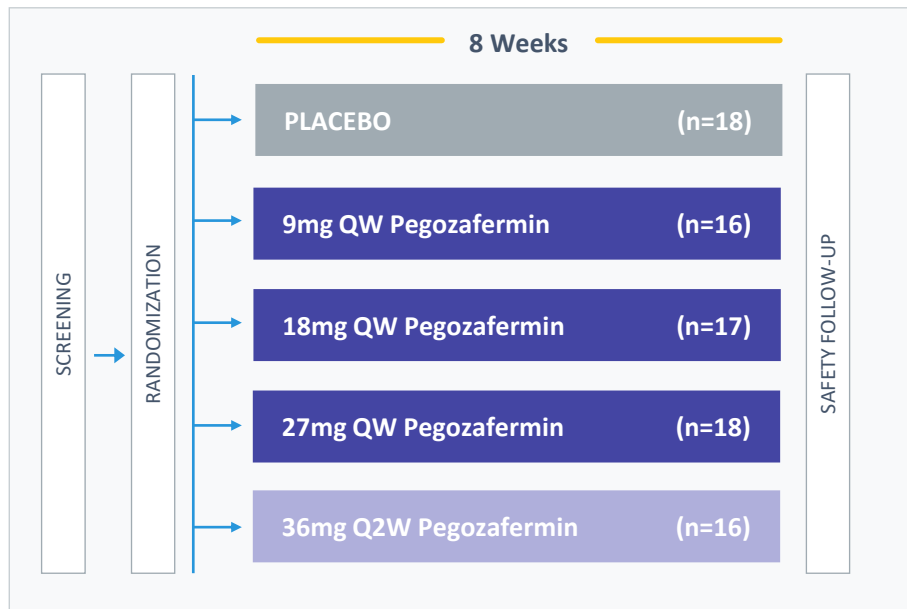
- Increase β -oxidation
- Decrease de novo lipogenesis
- Decrease FFA / TG

- **Muscle**

- Increase FFA oxidation



ENtrigue – Phase 2 SHTG Trial Design



Magnetic Resonance Imaging – Proton Density Fat Fraction
QW, once-weekly; Q2W, once every two weeks.

Safety analysis set, n=85 (patients who received at least 1 dose)

Full analysis set, n=82 (patients with at least 1 post-baseline TG assessment)

MRI analysis set n=23 (patients with baseline and end of treatment MRIs)

KEY INCLUSION CRITERIA

- TG ≥ 500 mg/dL and $\leq 2,000$ mg/dL
- Background therapy: statins and/or prescription fish oil and/or fibrates OR none

PRIMARY ENDPOINT

- Primary endpoint: % Change in TGs from baseline

KEY SECONDARY ENDPOINTS

- Lipids: non-HDL-C, HDL-C, Apo-B
- Liver fat (MRI-PDFF)
- Glycemic control

Baseline Characteristics

Represents an Advanced Population at High Risk for CV Disease



| Parameter Mean or % | Placebo (n=18) | PGZ Pooled (n=67) | PGZ 9mg QW (n=16) | PGZ 18mg QW (n=17) | PGZ 27mg QW (n=18) | PGZ 36mg Q2W (n=16) | Total (n=85) |
|------------------------------|-----------------------|-------------------------|-------------------------|--------------------------|--------------------------|---------------------------|------------------------------|
| Age (years) | 57.5 | 52.7 | 54.6 | 49.2 | 53.9 | 53.1 | 53.7 |
| Male (%) | 66.7 | 77.6 | 68.8 | 82.4 | 72.2 | 87.5 | 75.3 |
| Body Weight (kg) | 98 | 99 | 100 | 97 | 99 | 99 | 99 |
| Type 2 Diabetes (%) | 61.1 | 47.8 | 56.3 | 35.3 | 55.6 | 43.8 | 50.6 |
| TG (mg/dL) | 720 | 736 | 722 | 709 | 680 | 840 | 733 |
| Non-HDL-C (mg/dL) | 220 | 209 | 216 | 203 | 203 | 215 | 211 |
| HDL-C (mg/dL) | 28 | 28 | 31 | 27 | 31 | 25 | 28 |
| LDL-C (mg/dL) | 88 | 89 | 92 | 88 | 97 | 80 | 89 |
| Apo-B (mg/dL) | 116 | 115 | 120 | 115 | 119 | 106 | 115 |
| HbA1c $\geq 6.5\%$ (%) | 38.9 | 44.8 | 56.3 | 35.3 | 50.0 | 37.5 | 43.5 |
| ALT (U/L) | 29.1 | 33.9 | 36.3 | 36.9 | 33.0 | 29.2 | 32.8 |
| Liver Fat Content (%) (n=24) | 16.5 _[n=6] | 21.3 _[n=18] | 19.8 _[n=3] | 18.0 _[n=5] | 22.4 _[n=7] | 25.5 _[n=3] | 20.1_[n=24] |

Baseline Characteristics: Approximately 50% on Background Therapy *Represents Real World Setting*



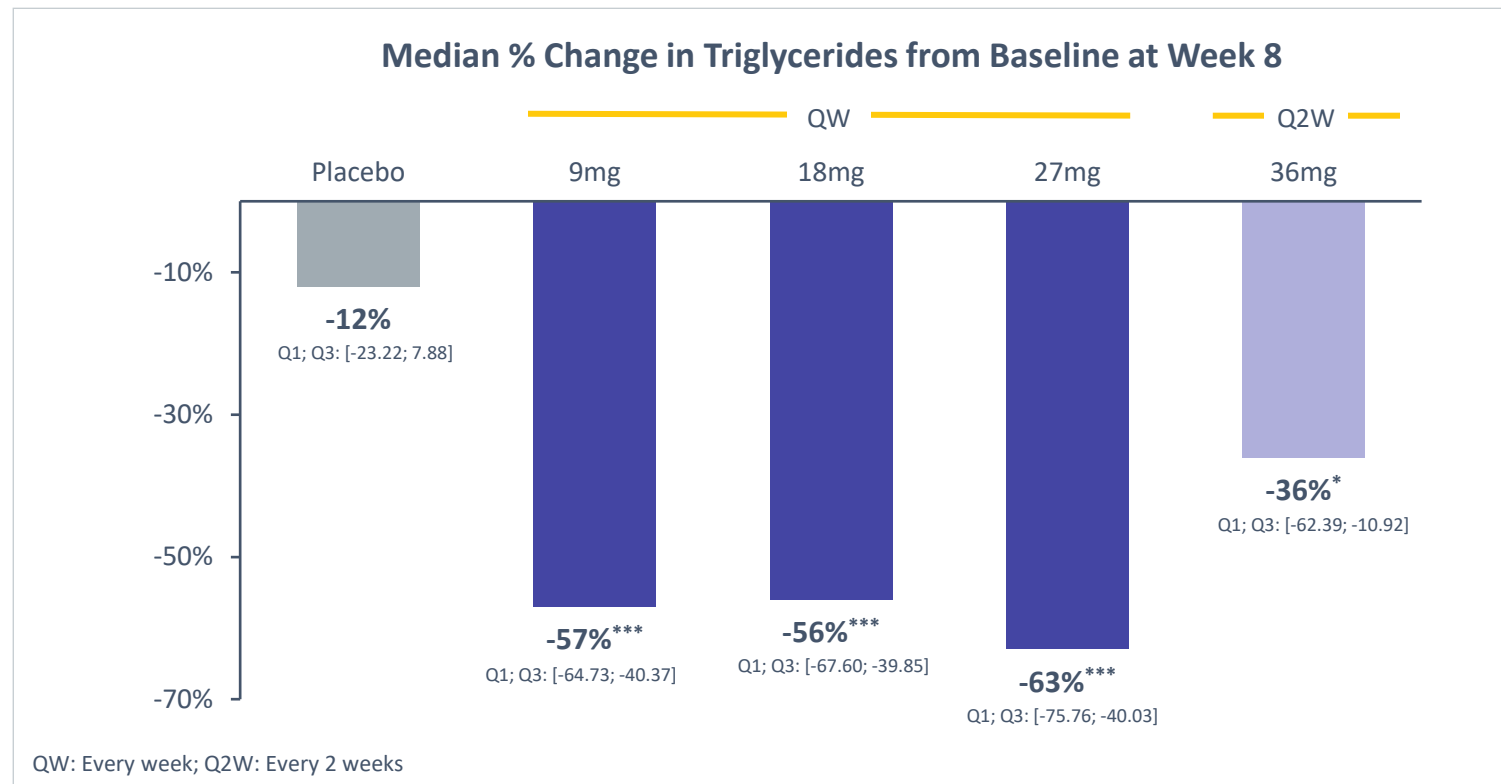
| | Placebo (n=18) | PGZ Pooled (n=67) | PGZ 9mg QW (n=16) | PGZ 18mg QW (n=17) | PGZ 27mg QW (n=18) | PGZ 36mg Q2W (n=16) | Total (n=85) |
|-------------------------------|-------------------|-------------------------|-------------------------|--------------------------|--------------------------|---------------------------|-----------------|
| Any background therapy | 61% | 54% | 50% | 53% | 61% | 50% | 55% |
| Statins/statin combo | 50% | 43% | 38% | 53% | 39% | 44% | 45% |
| Prescription fish oil | 11% | 15% | 6% | 12% | 22% | 19% | 14% |
| Fibrates | 17% | 5% | 0 | 0 | 17% | 0 | 7% |
| Other | 6% | 13% | 13% | 18% | 11% | 13% | 12% |

Patients may be on > 1 lipid-modifying therapy

Background therapy defined as concomitant lipid-modifying therapy

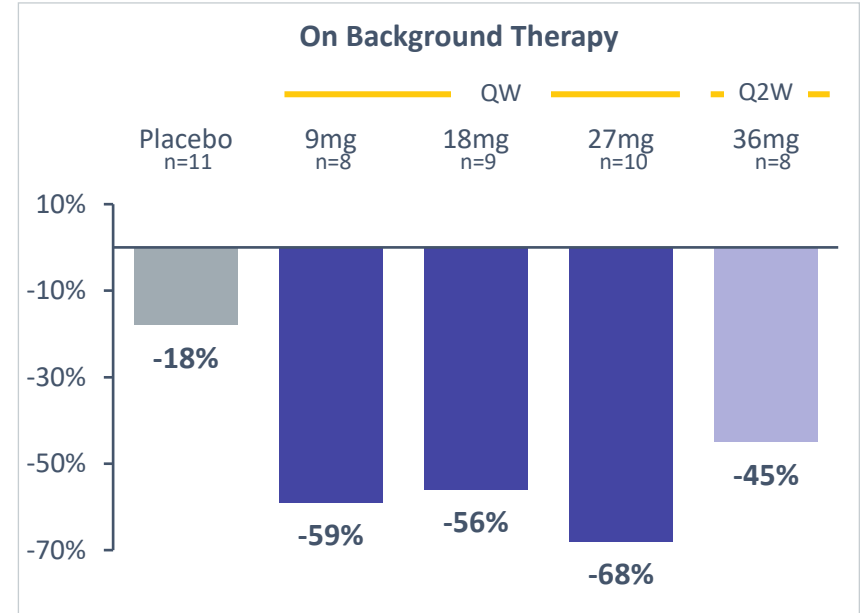
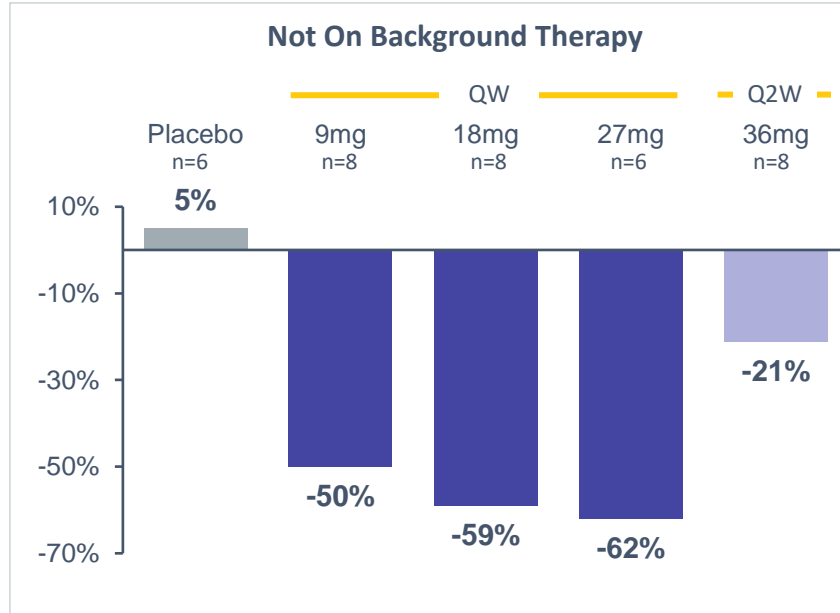
Pegozafermin Significantly Reduces Triglycerides across All Dose Groups

Primary Endpoint



Pegozafermin Shows Significant Decrease in Triglycerides on Top of Background Therapy

Median % Change in Triglycerides from Baseline at Week 8



Results are consistent with data from patients on background therapy of statins or statin combos, prescription fish oils, and fibrates

Pegozafermin Shows Consistent and Significant Benefit in Triglyceride Reduction across All Key Subgroups



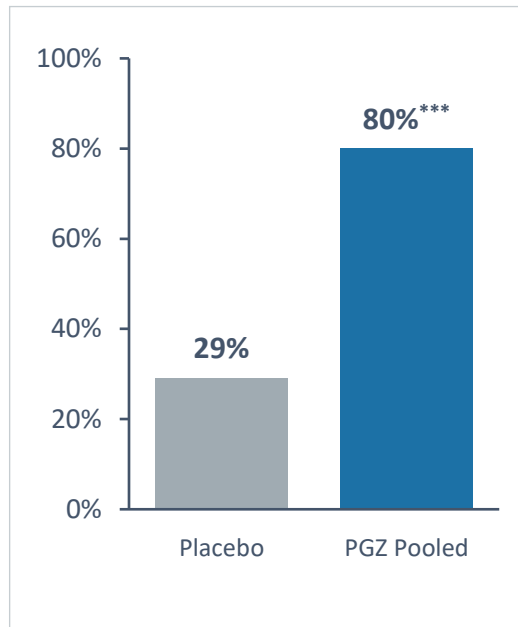
Median % Change in Triglycerides from Baseline at Week 8

| N=(placebo, PGZ pooled) | | Placebo | PGZ pooled |
|-------------------------------|-----------|---------|------------|
| Baseline TG ≥750 mg/dL | | | |
| Yes | (n=4,23) | -20% | -63% |
| No | (n=13,42) | 0% | -52% |
| Type 2 Diabetes | | | |
| Yes | (n=10,31) | -17% | -62% |
| No | (n=7,34) | -8% | -51% |
| Region | | | |
| US | (n=13,43) | -8% | -58% |
| EU | (n=4,22) | -23% | -57% |

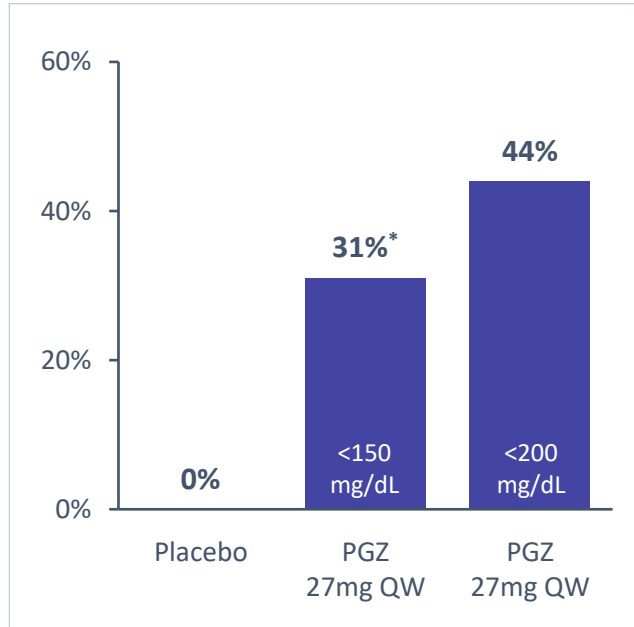
Similar trends were observed in subgroup analyses based on other baseline characteristics

Pegozafermin Shows Significant Decrease in Triglycerides at Different Threshold Levels

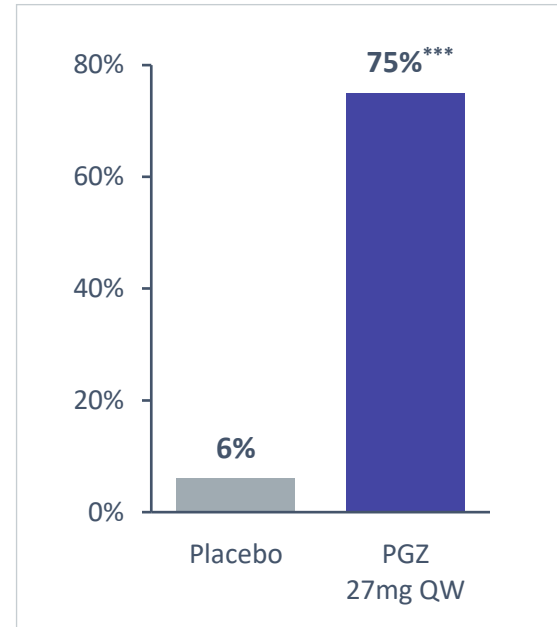
A. Responders (< 500 mg/dL)



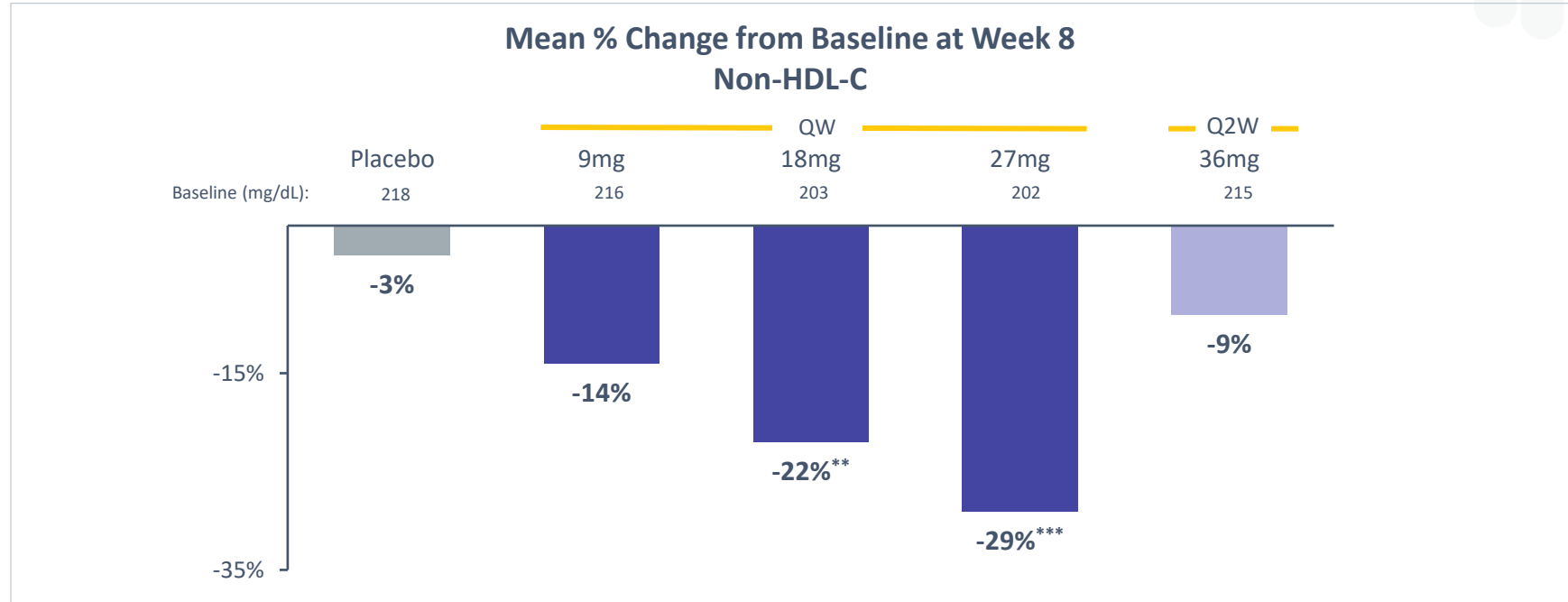
B. TG Normalization (<150mg/dL, <200mg/dL)



C. TG Reduction ≥50% from baseline



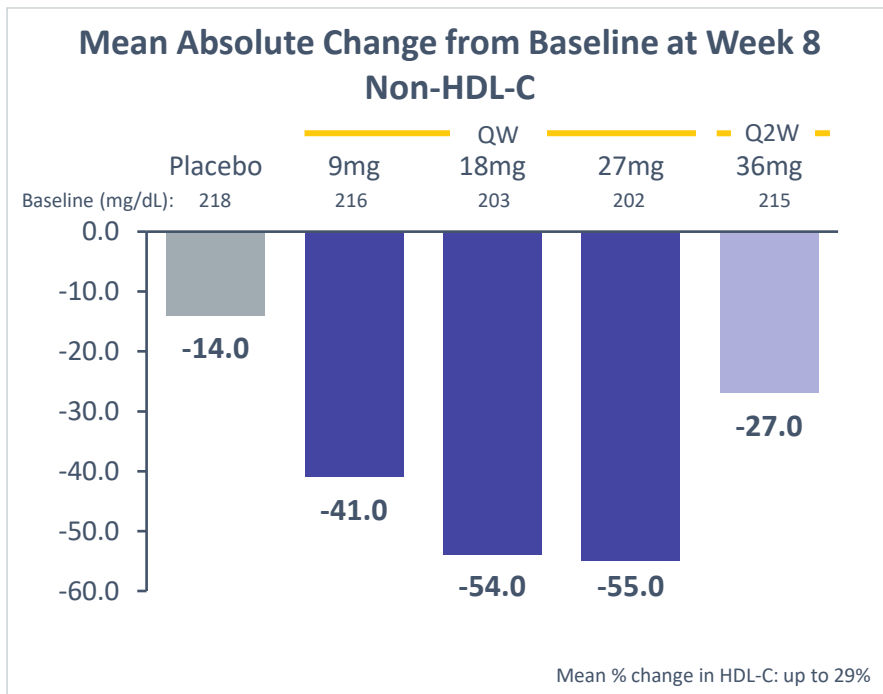
Pegozafermin Demonstrated Clinically Meaningful Improvements in Non-HDL-C – Key Marker of CV Risk



Non-HDL-C measures the cholesterol in atherogenic particles including LDL-C, IDL-C, and VLDL-C and is considered a better measure of CV risk in hypertriglyceridemia [‡]

Pegozafermin Demonstrated Reduction in Absolute Non-HDL-C

Absolute Non-HDL-C Reduction is Associated with MACE Improvement



Non-HDL-C reduction of 55 mg/dL is associated with a 25-30% reduction in MI or CV death

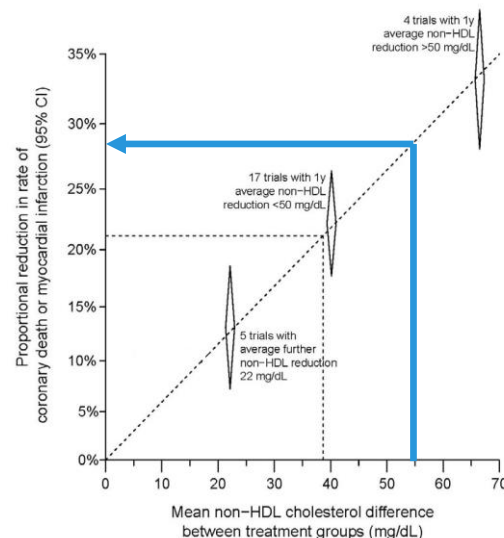
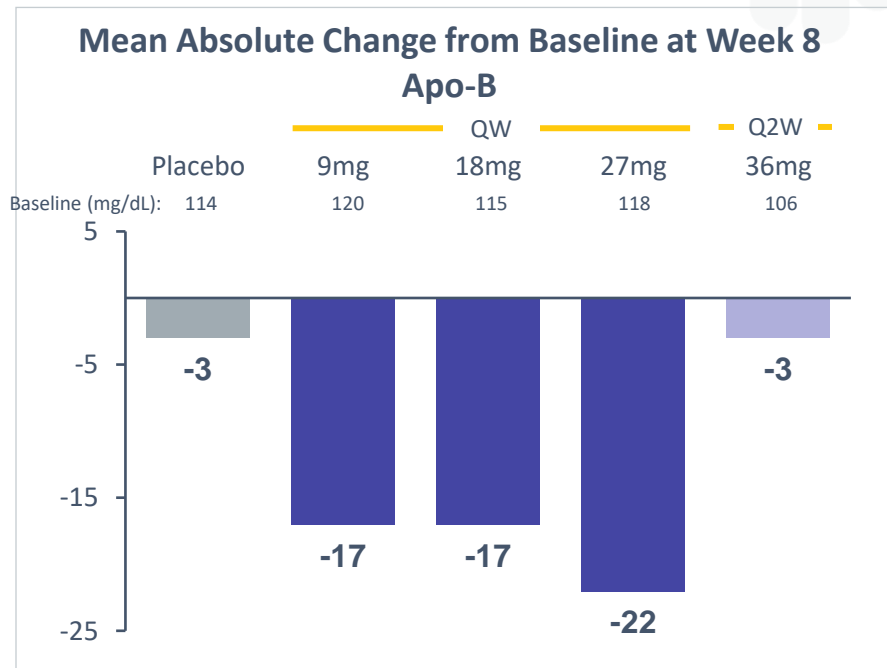
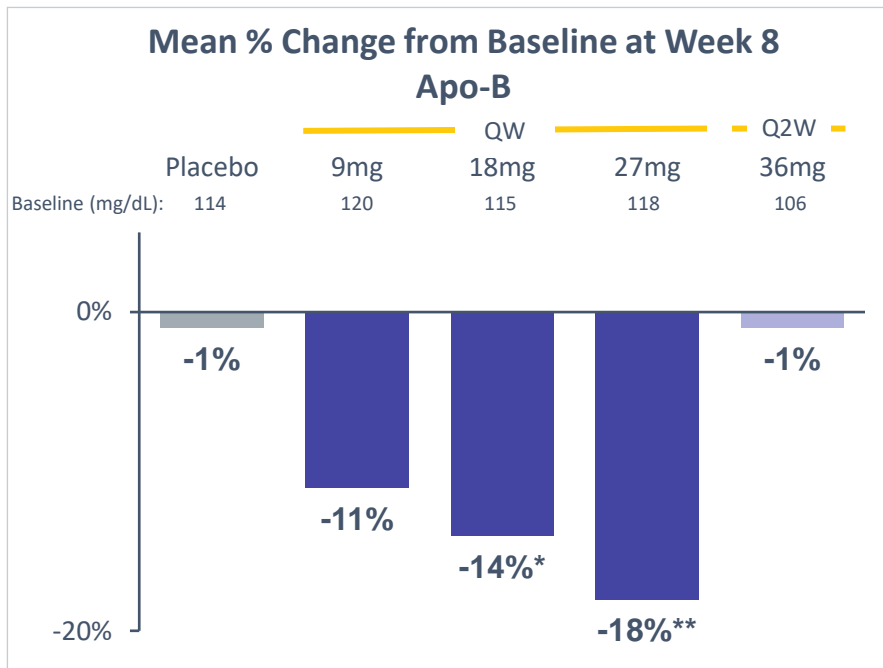


Figure adapted from: The HPS3/TIMI55-REVEAL Collaborative Group, NEJM 2017; 377: 1217-27

Non-HDL-C reduction of 55 mg/dL is associated with a 25-30% reduction in MI or CV death[†]

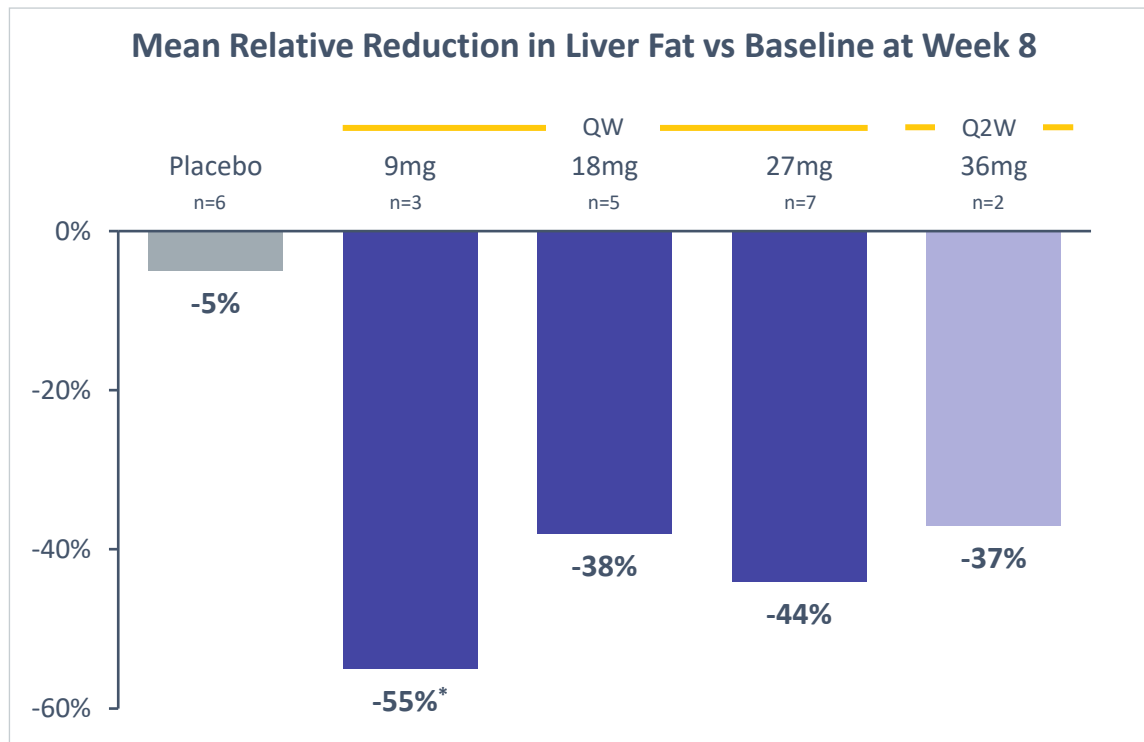
Pegozafermin Demonstrated Clinically Meaningful Improvements in Apo-B – Key Marker of CV Risk



Apo-B is a critical structural protein in atherogenic lipoproteins including LDL-C and is a direct measure of number of atherogenic lipoprotein particles. Apo-B has been proposed as a more accurate indicator of CV risk than LDL-C.¹

Pegozafermin Demonstrated Significant Reduction in Liver Fat

Liver Fat Is an Important Potentiator of CV Risk



Post-hoc analysis of patients with follow-up MRI-PDFF ≤ 21 days from date of last dose in 27mg QW cohort (n=5) demonstrated a 63% mean relative reduction from baseline.

*p < 0.05 vs. placebo

MRI Analysis Set; p value vs placebo based on ANCOVA analysis

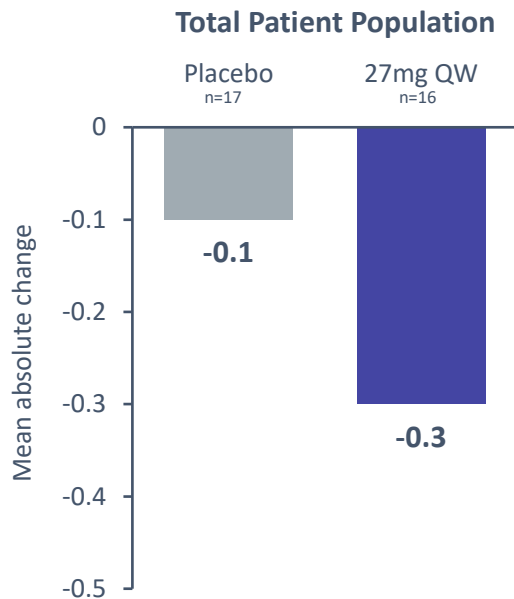
HIGH RESPONDER RATES

- **$\geq 30\%$ Reduction in liver fat:**
88% vs 0% in placebo
- **$\geq 50\%$ Reduction in liver fat:**
41% vs 0% in placebo
- **Normalized liver fat:**
24% vs 0% in placebo

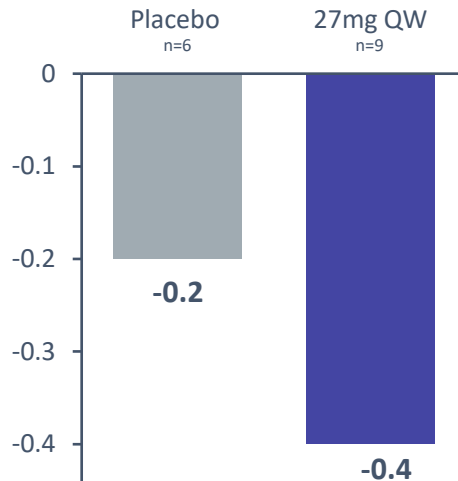
Pegozafermin (n=17) and Placebo (n=6)
Post-hoc analysis of patients with follow-up MRI-PDFF ≤ 21 days from date of last dose (n=14) resulted in 29% of patient with normalized liver fat and 100% and 50% of patients with mean relative reductions of $\geq 30\%$ and $\geq 50\%$ from baseline, respectively.

Pegozafermin Demonstrated Improvement on HbA1c that May Increase with Longer Treatment

Absolute Change in HbA1c at Week 8 in ENTRIGUE



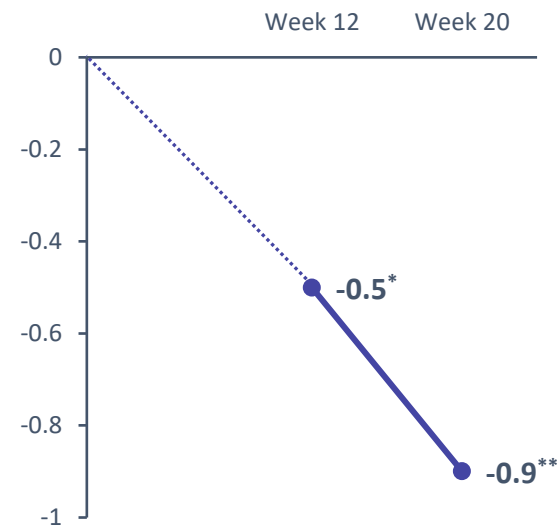
Patients with Baseline HbA1c $\geq 6.5\%$



HbA1c: Mean Baseline 27 mg QW: 7.48%; Week 8: 7.08%

Absolute change in HbA1c in 20-week NASH study

Patients with Baseline HbA1c $\geq 6.5\%$



Study BIO89-100-002; Cohort 7: n=10 at PGZ 27 mg QW

HbA1c: Baseline: 7.3%; Week 20: 6.4%

Patients with baseline HbA1c $\geq 6.5\%$ were on an average of 2 anti-diabetic medications.

*p<0.05; **p<0.01; ***p<0.001. p value for change from baseline based on MMRM analysis

Pegozafermin Was Well Tolerated Across Doses

Safety profile consistent with prior studies



Low Incidence of Treatment-Related AEs in $\geq 7.5\%$ of Pooled Pegozafermin Group

| Preferred Term | Placebo (n=18) | PGZ Pooled (n=67) | PGZ 9mg QW (n=12) | PGZ 18mg QW (n=21) | PGZ 27mg QW (n=18) | PGZ 36mg Q2W (n=16) |
|-------------------------|-------------------|----------------------|----------------------|-----------------------|-----------------------|------------------------|
| Nausea | 0 | 10% | 0% | 5% | 22% | 13% |
| Diarrhea | 0 | 9% | 17% | 5% | 17% | 13% |
| Injection site reaction | 0 | 9% | 8% | 10% | 6% | 13% |

All AEs were Grade 1 or 2; No Grade 3 or higher TEAEs reported. No tremor or transaminase elevation AEs reported.

| | Placebo (n=18) | PGZ Pooled (n=67) | PGZ 9mg QW (n=12) | PGZ 18mg QW (n=21) | PGZ 27mg QW (n=18) | PGZ 36mg Q2W (n=16) |
|---|-------------------|----------------------|----------------------|-----------------------|-----------------------|------------------------|
| Serious adverse event (unrelated) | 0 | 1* | 0 | 0 | 1 | 0 |
| Treatment emergent discontinuations (related/unrelated) | 0 | 2 [^] /2 | 0 | 0 | 2 [^] /2 | 0 |

*Unrelated SAE of Grade 2 hypertension; patient withdrew

[^]Grade 2 abdominal cramps (1) and Grade 2 nausea/vomiting (1)

Safety Analysis Set; patients reported on as treated basis

Pegozafermin has Differentiated Profile Compared with Other TG-Lowering Drugs



| | IN DEVELOPMENT | | APPROVED | | | |
|------------------------|------------------------|----------------------------------|-------------------|------------------------|----------------------------|-------------|
| | Pegozafermin Potential | APOC3 | Fibrates | Prescription Fish Oils | | Statins |
| | | | | Vascepa | Lovaza | |
| Triglyceride reduction | ✓✓✓ | ✓✓✓ | ✓✓ | ✓ | ✓✓ | ✓✓ |
| Liver fat reduction | ✓ | — | Worsens liver fat | — | — | — |
| Insulin sensitizing | ✓ | — | — | — | — | — |
| Cholesterol lowering | ✓ (non-HDL-C) | ✓ (non-HDL-C) | Worsens LDL-C | — | Worsens LDL-C | ✓ |
| Apo-B lowering | ✓ | ✓ | — | ✓ | — | — |
| ALT lowering | ✓ | Transaminase elevations observed | Monitor ALT | — | May require ALT monitoring | Monitor ALT |

For triglyceride reduction: ✓✓✓ = ≥ 60%, ✓✓ = 31%-59%, ✓ = ≤ 30% — No effect/Not reported

Sources: Feingold KR. Triglyceride Lowering Drugs. [Updated 2021 Apr 1]. In: Feingold KR, Anawalt B, Boyce A, et al., editors. Prescribing information. Corporate presentations.
 Note: All data regarding third-party molecules on this slide are based on third-party studies, which are in different stages of development, and not our own.
 Conclusions on this slide are company estimates and are not based on head-to-head results.

Proposed Clinical Development



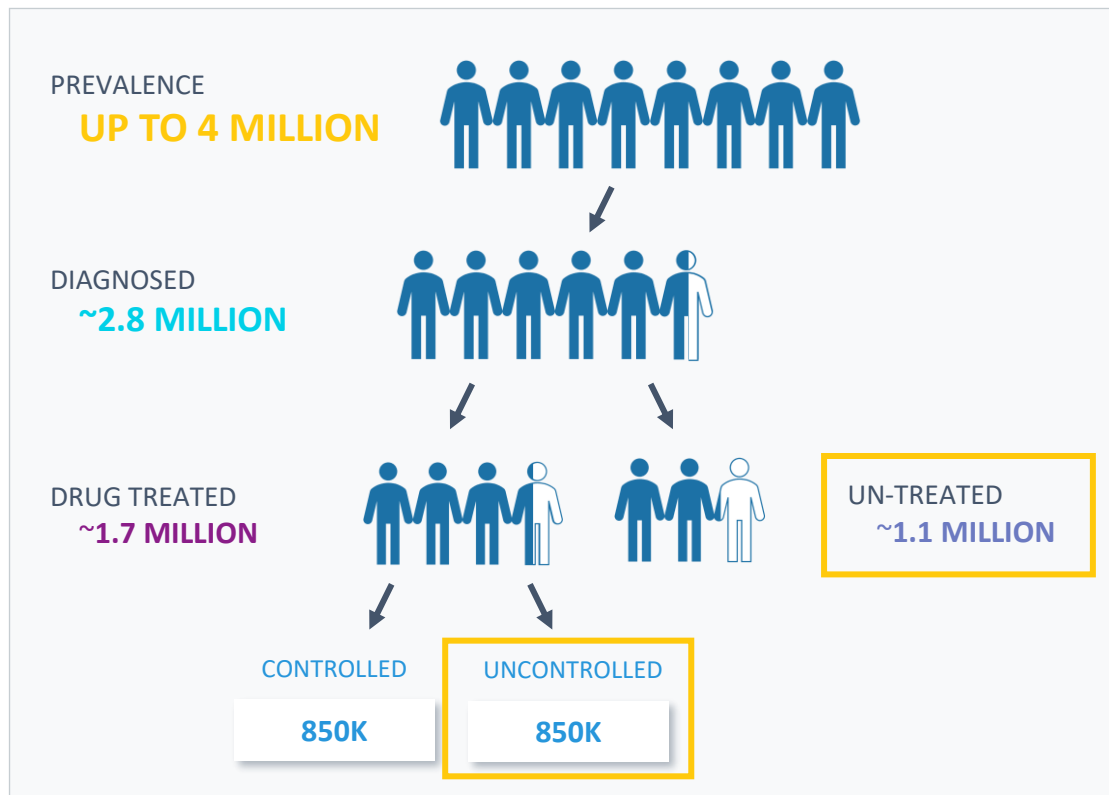
Regulatory

- Seek full approval using endpoint of triglyceride lowering (based on FDA precedent)
- Propose study duration and study size based on prior precedents for approved drugs
- Discuss safety database requirements for approval
- End of Phase 2 meeting planned for 2H22

Clinical Development

- Conduct single pivotal Phase 3 trial in SHTG
 - Trial initiation expected in 1H23; Topline results expected in 1H25
- Consider additional studies to meet safety database requirements per FDA discussions
 - Potential trial(s) in other hypertriglyceridemia patient populations
 - Leverage safety data from NASH studies

SHTG Represents a Large Population with High Unmet Needs



| Co-morbidity | Prevalence in SHTG population |
|-----------------------------|-------------------------------|
| Fatty Liver Disease (NAFLD) | Up to 100% |
| Type 2 diabetes/Prediabetes | Up to 70% |
| Dyslipidemia | Up to 65% |

Pegozafermin profile is unique and compelling to physicians because of potential for metabolic benefits

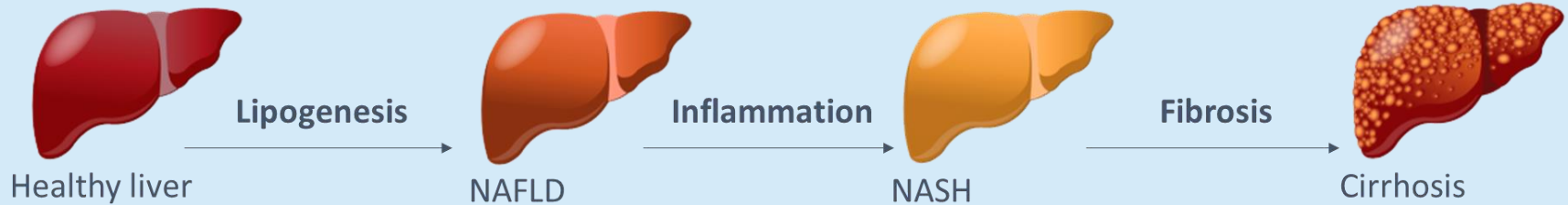
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Opportunity in NASH



NASH is a Serious Liver Condition With Significant Co-Morbidities

Metabolic Dysregulation → Excess Liver Fat Accumulation → Progressive Disease



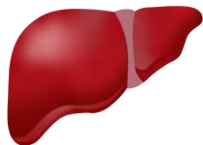
- No treatments currently available
- 16.5 million cases projected to grow to 27 million cases by 2030
- Expected to become the leading cause of liver transplant

| Co-morbidity | Prevalence in NASH population |
|-------------------------------|-------------------------------|
| Hypertriglyceridemia | 83% |
| Obesity | 82% |
| Hyperlipidemia / Dyslipidemia | 72% |
| Metabolic syndrome | 71% |
| Type 2 diabetes | 44% |

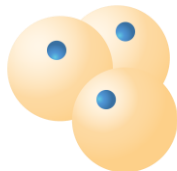
Pegozafermin Has Potential to Be Mainstay of Therapy in Liver and Cardio-Metabolic Disease

↑ Lipid disposal
↑ TG synthesis
↓ LDL synthesis
↑ HDL synthesis
↓ De novo lipogenesis
↑ Insulin sensitivity
↓ Fibrosis
↓ Inflammation

Liver



White adipose tissue

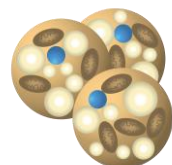
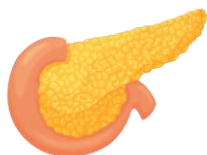


↑ Adiponectin production
↑ TG disposal
↑ FGF21 production
↑ Insulin signaling
↓ Inflammation

Pegozafermin

↑ β cell survival
↑ β cell function
↓ GH signaling
↓ Glucagon

Pancreas



Brown adipose tissue

↑ Glucose Uptake
↑ TG disposal
↑ Thermogenesis

- Endogenous metabolic hormone that regulates energy expenditure and glucose and lipid metabolism
- Reduces liver fat through actions within the liver and adipose tissue
- Impacts liver fibrosis via metabolic pathways and upregulation of adiponectin
- Lowers TG levels by reducing triglyceride production and increasing catabolism

1. Bondurant LD, et al. *Cell Metab.* 2017;25(4):935-944.e4.

2. Schlein C, Talukdar S, Heine M, et al. *Cell Metab.* 2016;23(3):441-4553.

3. Yen CLE, et al. *J Lipid Research.* 2008;49(11): 2283-301.

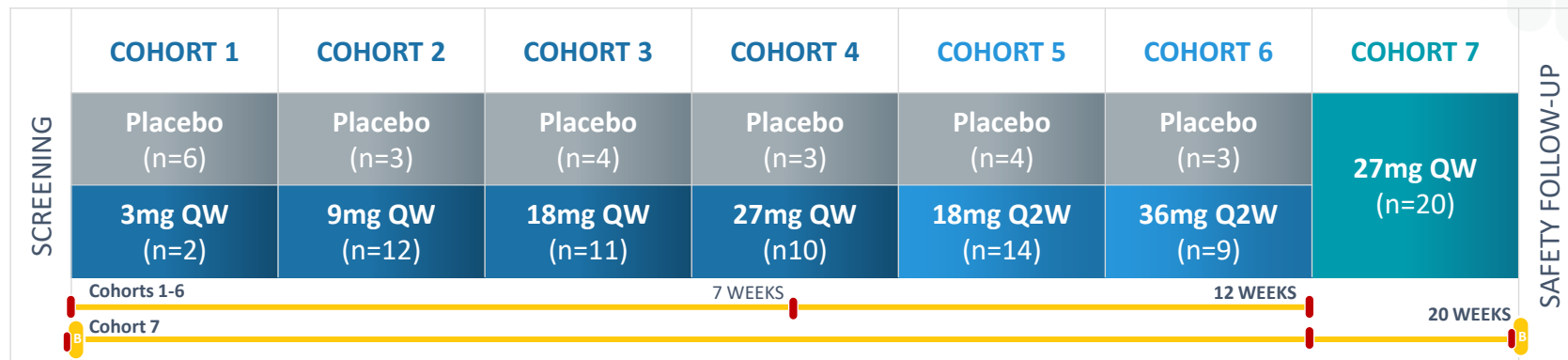
4. Fisher FM, et al. *Genes Dev.* 2012;26(3):271-281.

5. Xu J, et al. *Diabetes.* 2009;58(1):250-259

6. Holland et al, *Cell Metab.* 2013 May 7;17(5):790-7.

7. Vandanmagsar, B., et al., *Cell reports* 2015. 15(8), 1686-1699.

Phase 1b/2a NASH Trial Design



COHORTS 1-6

KEY INCLUSION CRITERIA

- NASH* or phenotypic NASH (PNASH)[#]
- MRI-PDFF $\geq 10\%$

COHORT 7

KEY INCLUSION CRITERIA

- F2-F3 NASH; NAS ≥ 4
- MRI-PDFF $\geq 8\%$

Pegozafermin is delivered subcutaneously

*Patients with biopsy-proven F1-3; [#]Central obesity plus T2DM or evidence of liver injury;
 • Placebo (n=19) combined across cohorts for analysis ; Randomized, pharmacodynamic (PD) and safety analysis set n=81; Study completers n=71; MRI analysis set n=75 (patients with post-baseline MRI)

• Interim data based on Jan 3, 2022 data cut
 • 19/20 (95%) patients completed treatment and had end-of-treatment biopsies; 1 patient discontinued treatment due to withdrawal of consent

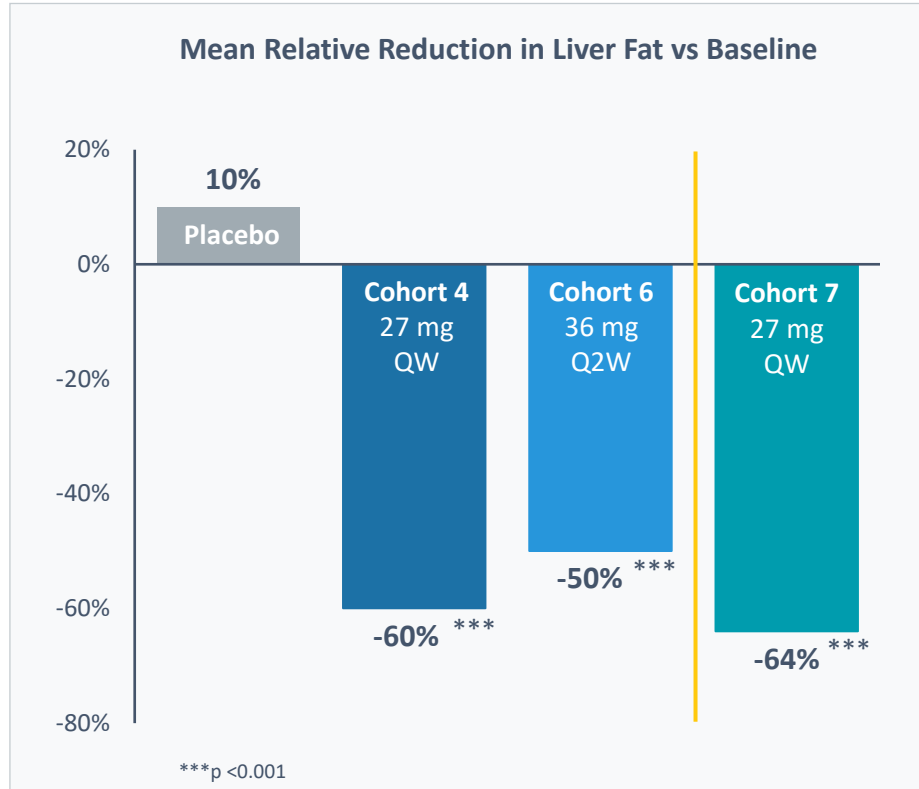
Baseline Characteristics



| Parameter Mean or % | Cohorts 1-6 (n=81) | Cohort 7 (n=20) |
|--------------------------|-----------------------|--------------------|
| Age (years) | 51.9 | 58.4 |
| Female | 61.7% | 75.0% |
| Weight (kg) | 93.6 | 104.6 |
| BMI (kg/m ²) | 34.6 | 37.0 |
| Type 2 Diabetes | 45.7% | 85.0% |
| % F2 / % F3 | NA | 35% / 65% |
| NAS | NA | 5.3 |
| MRI-PDFF (%) | 21.3 | 21.1 |
| ALT (U/L) | 41.5 | 47.1 |
| AST (U/L) | 31.0 | 36.1 |
| Pro-C3 (ng/mL) | 11.9 | 19.3 |
| VCTE (kPA) | 7.3 | 14.3 |
| Triglycerides (mg/dL) | 174.3 | 170.0 |

NA: Not applicable

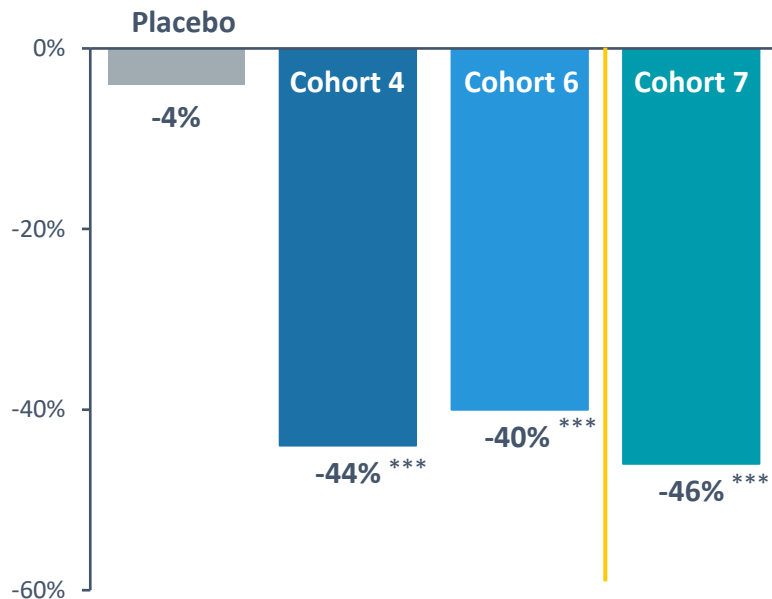
Pegozafermin Demonstrated Robust Liver Fat Reduction with High Responder Rates



| | ≥30% Relative Reduction in Liver Fat | ≥50% Relative Reduction in Liver Fat |
|----------|--------------------------------------|--------------------------------------|
| Cohort 4 | 86% | 71% |
| Cohort 6 | 88% | 50% |
| Cohort 7 | 100% | 78% |

Pegozafermin Significantly Reduced ALT with Greater Reduction Observed in Patients with Elevated Baseline ALT

Mean Percent Change vs Baseline

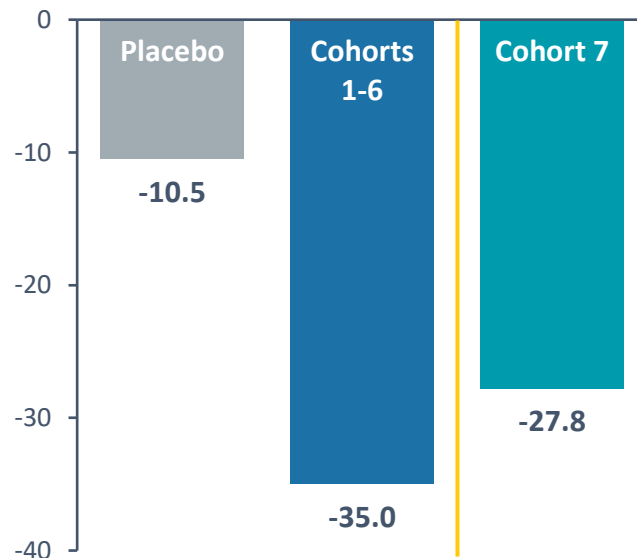


Comparable changes were observed with AST

Cohort 4 and 6: Pre-planned sensitivity analysis; MMRM LS Mean at week 13; *** p<0.001 versus placebo

Cohort 7: p value for change from baseline based on MMRM analysis; Data from week 20; ***p<0.001

ALT – Absolute Change vs Baseline (U/L) Patients with elevated ALT at baseline



71% of pts on pegozafermin had ≥ 17 U/L reduction

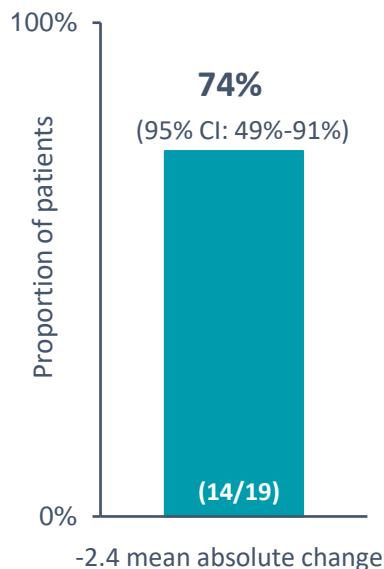
Cohorts 1-6: PD Analysis Set in baseline ALT > 45 U/L; Pre planned sensitivity analysis; MMRM LS Mean at Week 13

Cohort 7: elevated ALT ≥ 30 U/L for women and ≥ 40 U/L for men; Week 20

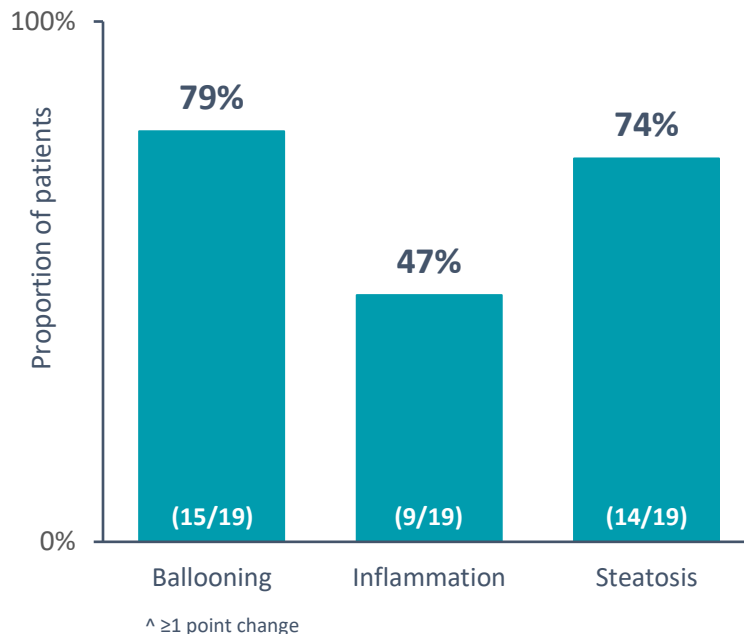
Pegozafermin Robustly Improved NAFLD Activity Score (NAS) and All Components of NAS



≥2pt Improvement in NAS*

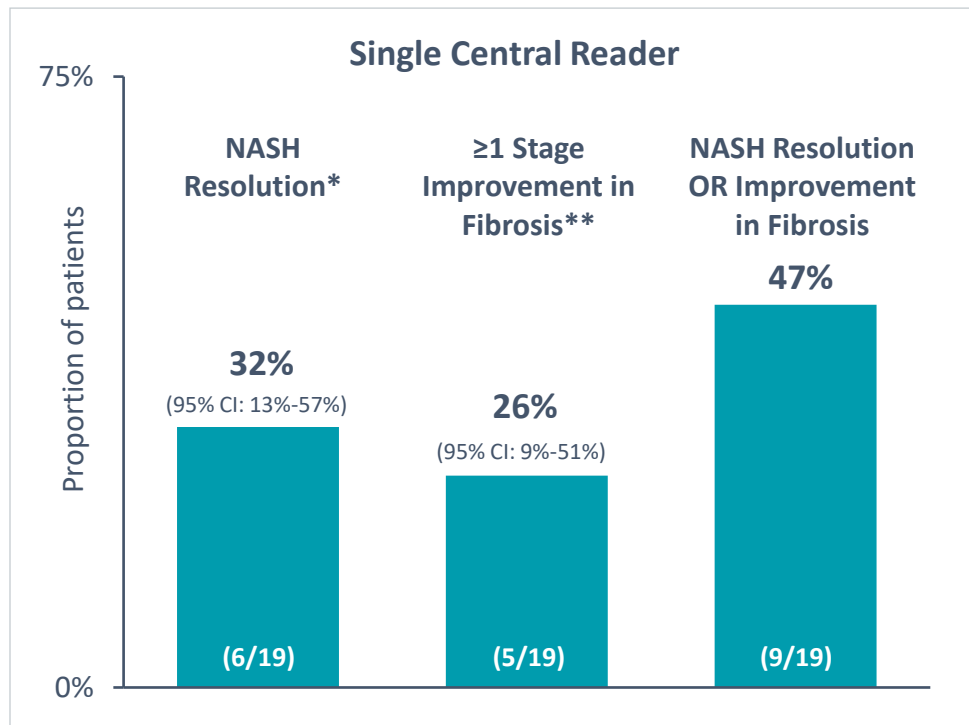


Responder^ Rates by NAS Component



- **63%** of patients had ≥2point improvement in NAS and no worsening of fibrosis* (primary endpoint)
- **100%** of patients had improvement or no change in ballooning and inflammation

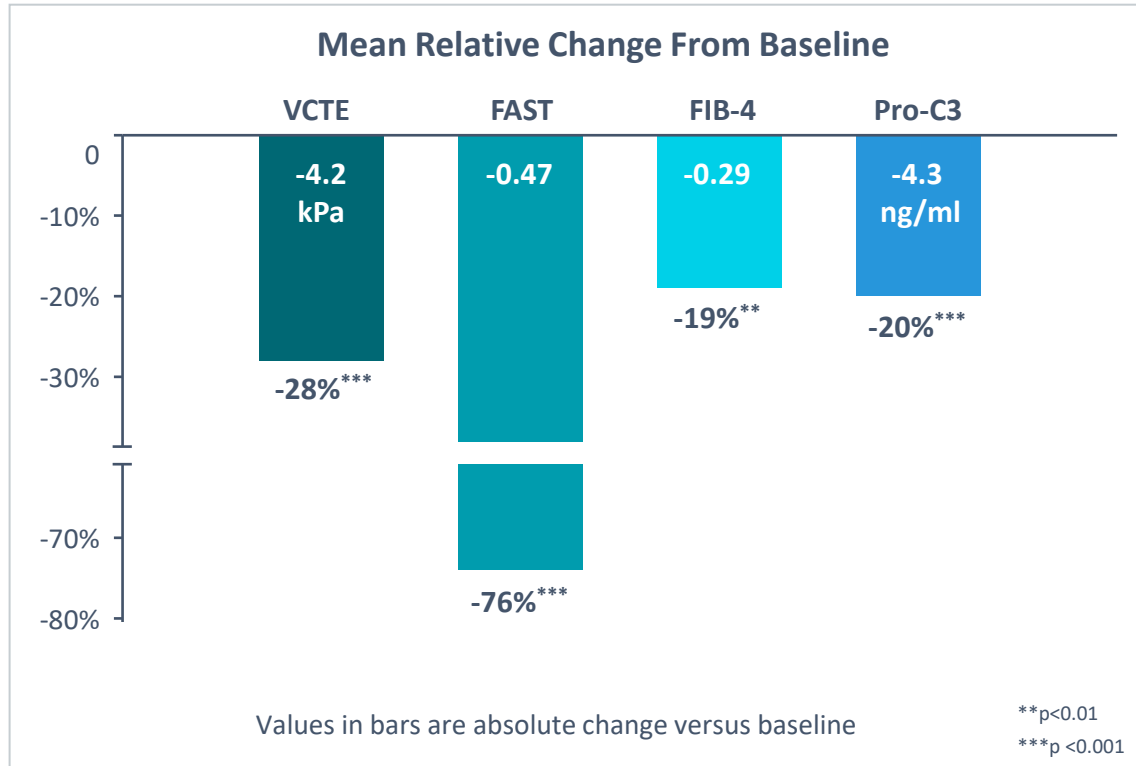
Pegozafermin Demonstrated Clinically Meaningful Changes on Key Histological Efficacy Endpoints



THREE-PANEL READ

- **NASH resolution: up to 47%**
(range: 26-47%)
- **Fibrosis improvement: up to 42%**
(range: 12-42%)
- **2-point NAS improvement: up to 79%**
(range: 68-79%)

Pegzofermin Substantially Improved Scores Across Non-Invasive Tests (NITs) Correlated with Advanced Fibrosis

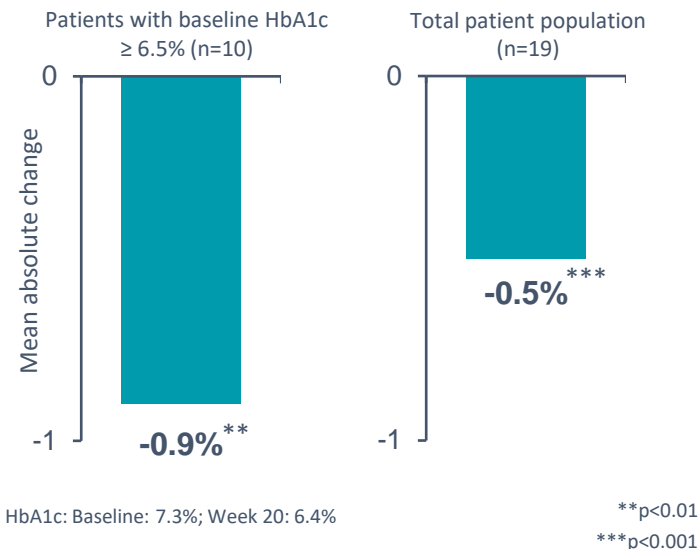


NIT DESCRIPTIONS

- VCTE: Liver stiffness measure using FibroScan®
- FAST score: Liver stiffness (VCTE) and steatosis (CAP) using FibroScan® plus AST; 0-1 scale
- FIB-4 score: Composite serum marker/age measure
- Pro-C3: Collagen deposition serum biomarker

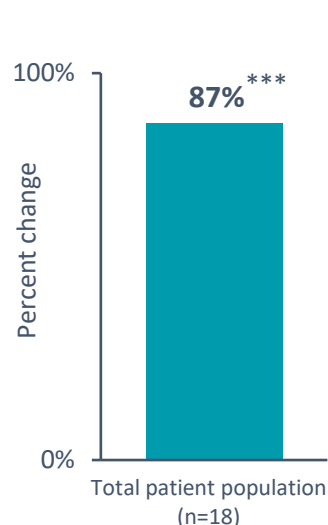
Pegozafermin Demonstrated Clinically Meaningful Improvement on HbA1c and Adiponectin with Notable Body Weight Reduction

Absolute Change in HbA1c



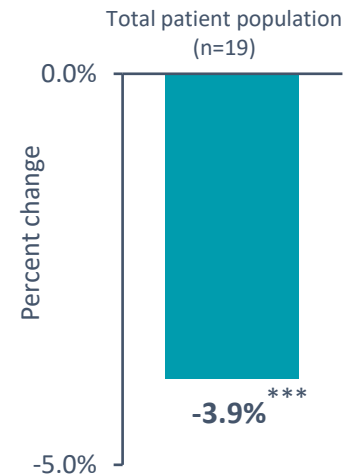
Patients with baseline HbA1c ≥ 6.5% were on an average of 2 anti-diabetic medications

Adiponectin Change



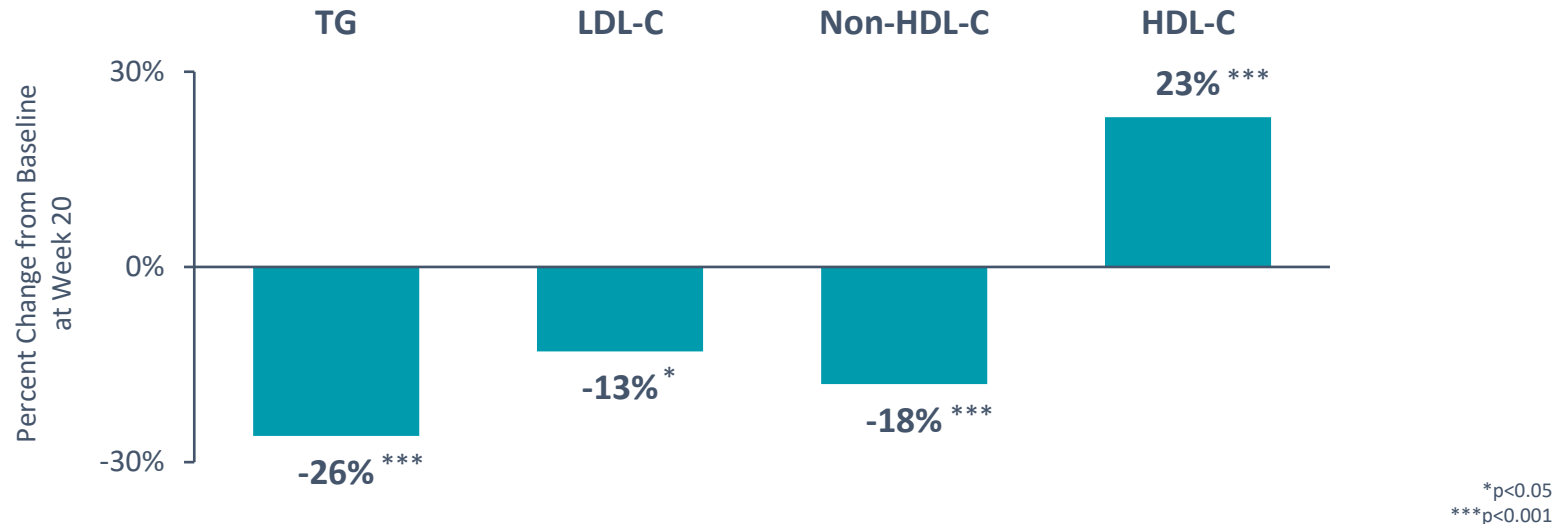
***p<0.001

Weight Change



***p<0.001

Pegozafermin Demonstrated Clinically Meaningful Improvements in Lipid Parameters

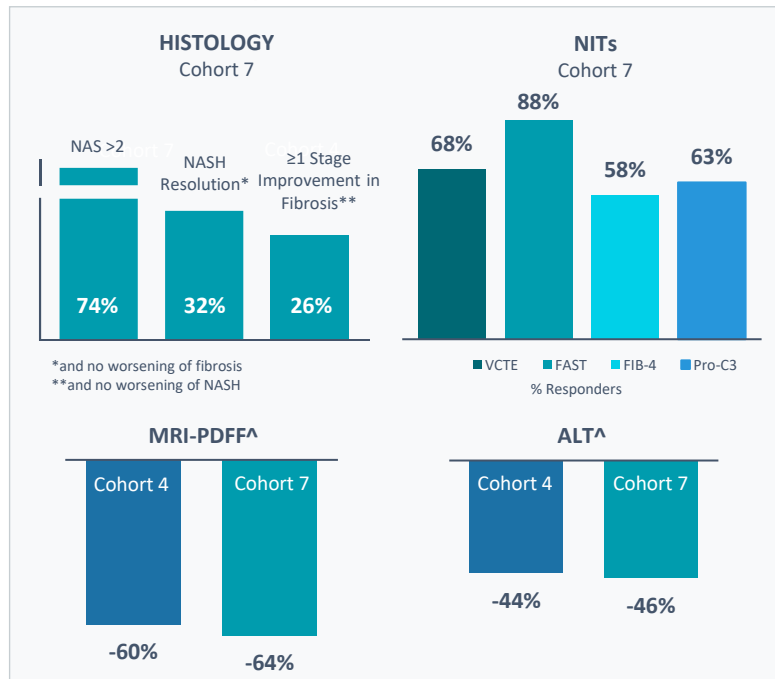


Lipid benefits consistent in the subgroup taking background dyslipidemia treatments (n=12)

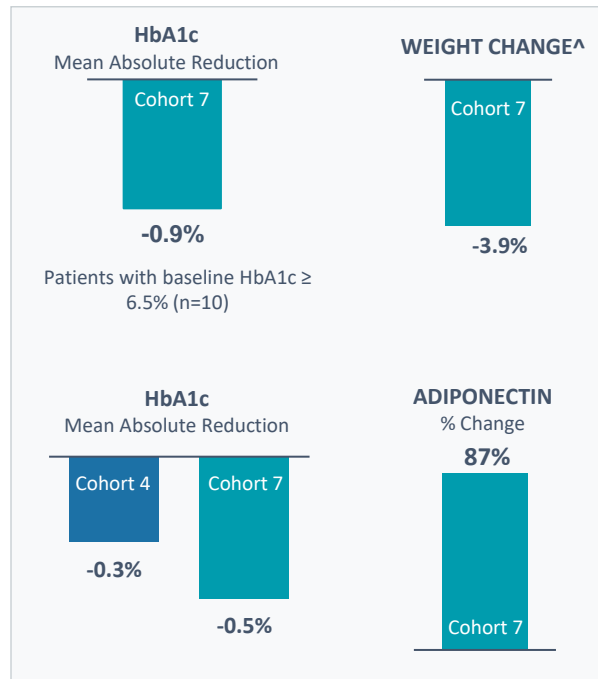
Pegozafermin Improves Many Markers of Liver Health and Co-Morbidities Associated with NASH



LIVER HEALTH



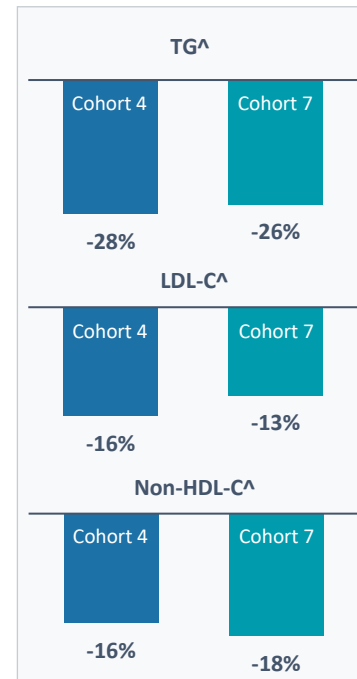
INSULIN RESISTANCE



OBESITY



DYSLIPIDEMA



Pegozafermin - Favorable Safety and Tolerability in Phase 1b/2a Study

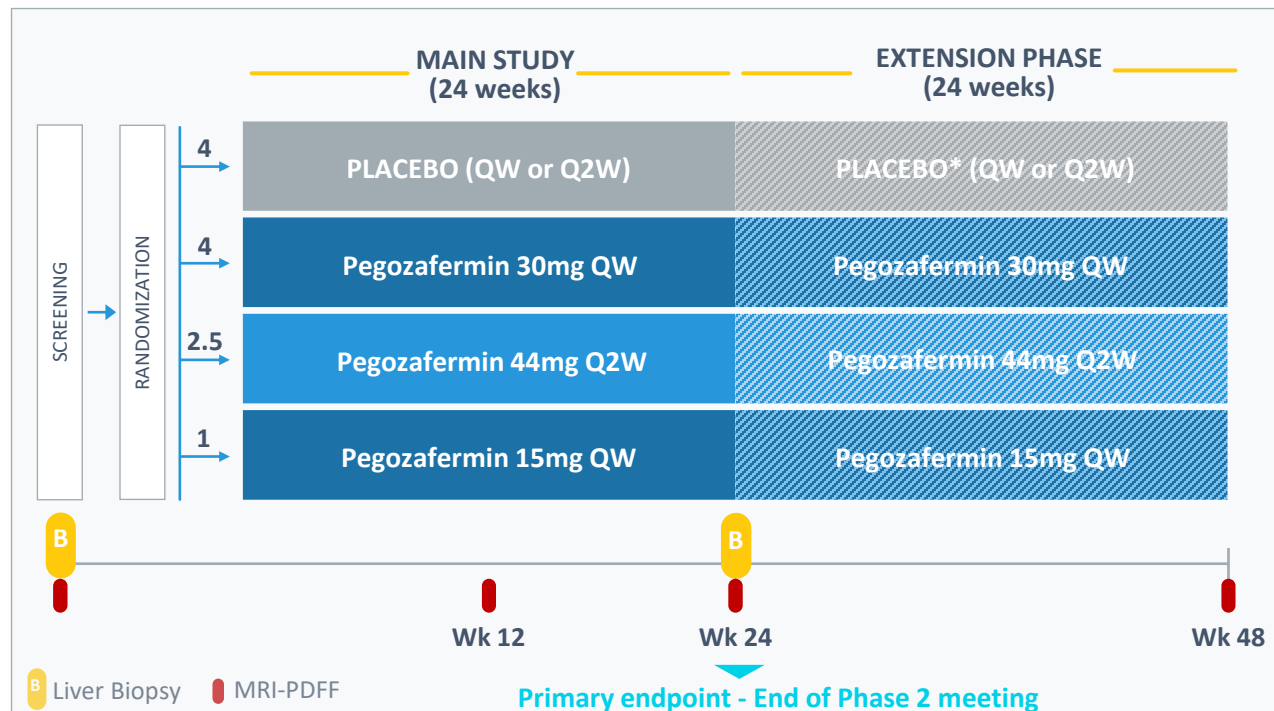
- No treatment-related serious adverse events; only 1 treatment-related discontinuation
- Pooled pegozafermin treatment related Adverse Events (AEs) observed in $\geq 10\%$ of patients were:
 - Increased appetite (13%) vs placebo (0%)
 - Diarrhea (13%) vs placebo (11%)
 - Nausea (12%) vs placebo (11%)
- Most GI AEs were mild and of short duration
- Few mild injection site reactions
- No tremor or hypersensitivity AEs reported
- No adverse effects on blood pressure or heart rate

Comparative Profile of FGF21 Analogs in NASH



| | Pegozafermin | Efruxifermin |
|------------------------------|---|---|
| Structure | <ul style="list-style-type: none"> GlycoPEGylated FGF21 | <ul style="list-style-type: none"> Fc-fused FGF21 |
| Efficacy | <ul style="list-style-type: none"> Significant effect on liver parameters <ul style="list-style-type: none"> Similar effects on multiple non-invasive markers Similar efficacy on NAS >2; other histology endpoints range from lower to equivalent* Robust impact on metabolic parameters – Pegozafermin 27mg potential better glycemic control | |
| Tolerability | <ul style="list-style-type: none"> Well-tolerated at all doses Low incidence of GI events No tremors | <ul style="list-style-type: none"> High frequency and withdrawals from GI events in multiple studies Tremors observed in multiple studies |
| Dosing Frequency | <ul style="list-style-type: none"> Weekly and Every Two-Weeks | <ul style="list-style-type: none"> Weekly |
| Phase 2b Drug Product | <ul style="list-style-type: none"> Liquid | <ul style="list-style-type: none"> Frozen |
| Development Plan | <ul style="list-style-type: none"> Phase 2b (F2/F3) results expected in 1Q23; Larger well-powered Phase 2b | <ul style="list-style-type: none"> Phase 2b (F2/F3) results expected in 3Q22; 3 arms – 128 patients |

Phase 2b (ENLIVEN) NASH Trial Design



KEY INCLUSION CRITERIA

- F2-F3 NASH; NAS ≥ 4

SELECTED KEY ENDPOINTS

- Fibrosis Improvement
- NASH Resolution
- NAS ≥ 2 point improvement
- Composite histology and metabolic endpoints

BIOPSY READING

- 3-panel consensus read – industry best-in-class approach

Planned randomization (~200 patients) – 4 : 4 : 2.5 : 1 (placebo : 30mg QW : 44mg Q2W : 15mg QW)

Financial Position Summary



**Cash, cash equivalents
and short-term investments**

**\$200.5M pro forma cash as of June 30, 2022
that includes estimated net proceeds from
an underwritten public offering of common
stock, pre-funded warrants and warrants**

Corporate Highlights

Pegozafermin – potential important new cardio-metabolic drug; balanced effect on multiple key endpoints

- Impacts a broad spectrum of cardio and metabolic endpoints
- Robust data in two indications with a favorable safety and tolerability profile
- Potential first-to-market FGF21 analog

Severe Hypertriglyceridemia (SHTG) – Ph. 3 initiation planned in 1H23

- Highly differentiated based on Phase 2 success across multiple endpoints
- Significant opportunity given large, under-served market with limited competition
- Established regulatory pathway allows efficient clinical development

Nonalcoholic Steatohepatitis (NASH) – Ph. 2b enrollment completion in 3Q22; data expected 1Q23

- FGF21 offers greatest promise in this category – addresses multiple facets of the disease
- Pegozafermin has demonstrated compelling results in clinic

Strong cash position with experienced team

- \$200.5M* pro forma cash** as of June 30, 2022
- Track record of developing and commercializing successful drugs and business development at other companies

89bio

Appendix



Experienced Management Team Positions 89bio for Success



Rohan Palekar
CEO

CEO, CCO experience
Commercial, strategy,
and R&D experience



Hank Mansbach, MD
CMO

20+ years biopharma and
R&D leadership in clinical
development and medical
affairs



Ryan Martins
CFO

CFO, Strategy/IR,
finance, sell-side
experience



Quoc Le-Nguyen
CTO & Head of Quality

20+ years biopharma and
leadership in technical
operations, product supply,
and quality



Achievements and Milestones



ACHIEVEMENTS

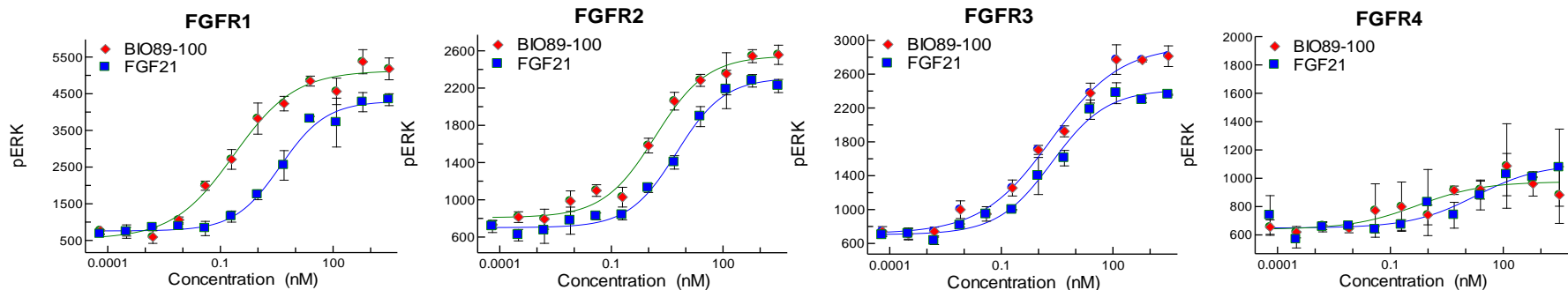
- ✓ Significant progress in the clinic:
5 clinical studies completed or ongoing
in NASH/SHTG
- ✓ Completed key preclinical studies
including long-term tox
- ✓ Scaled up manufacturing at CMO
- ✓ Developed liquid formulation
- ✓ IP through 2038
- ✓ Strong balance sheet



PROGRAM STATUS/MILESTONES

- Complete enrollment in Phase 2b (ENLIVEN)
NASH (F2/F3) trial – 3Q22
- Topline results from Phase 2b (ENLIVEN)
NASH (F2/F3) trial – 1Q23
- Initiate Phase 3 SHTG trial – 1H23

Pegozafermin Exhibits Highly Potent FGF Receptor Agonism

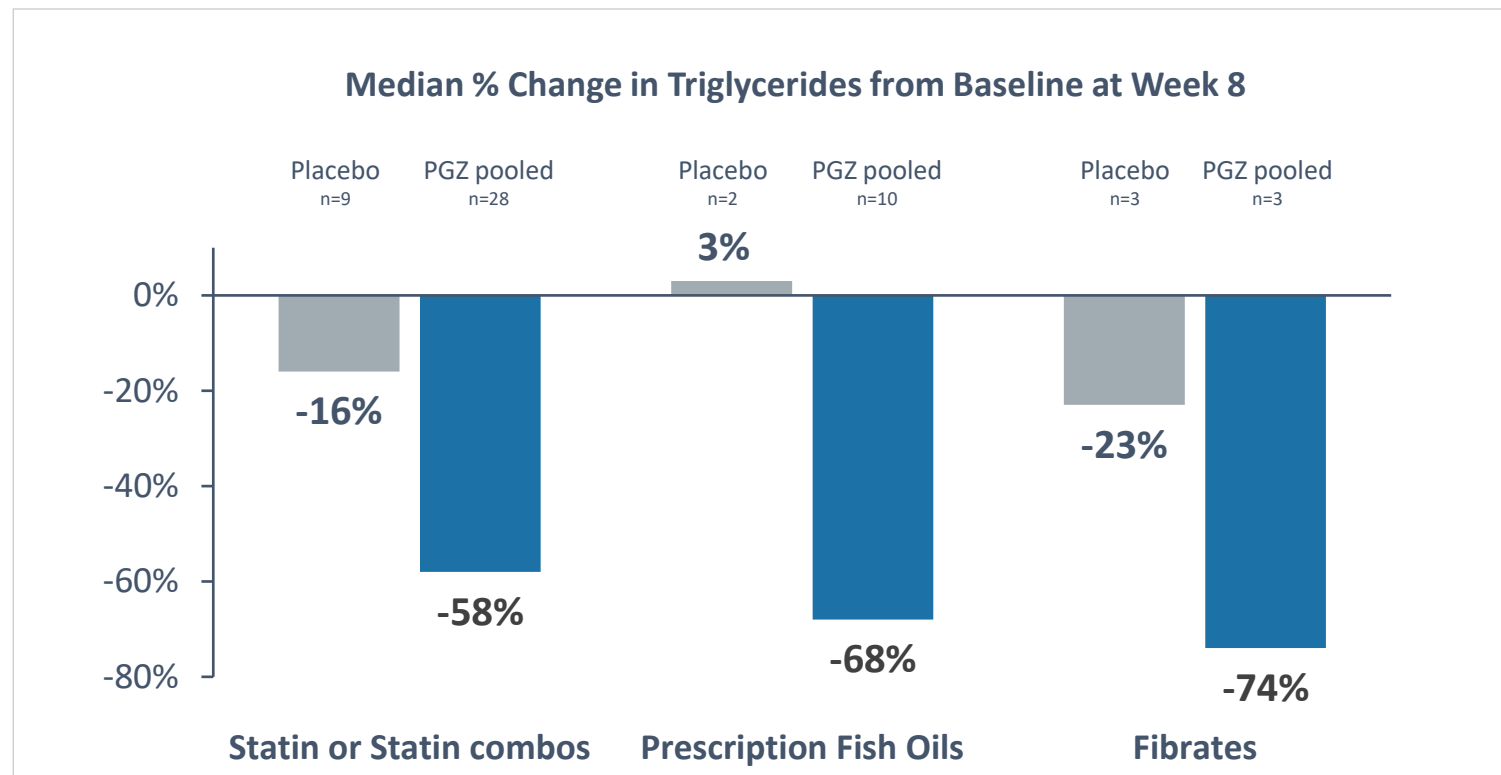


Pegozafermin has the potential to reproduce the beneficial metabolic effects of native FGF21

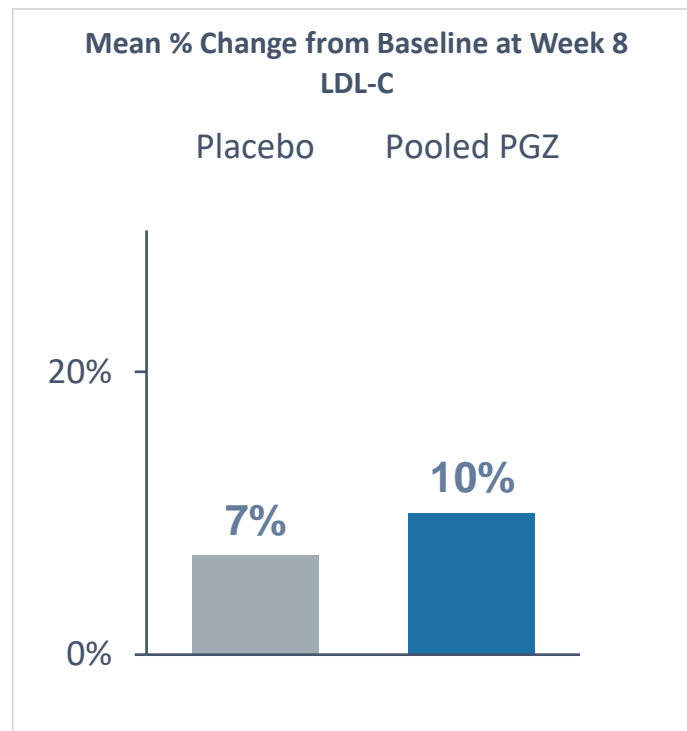
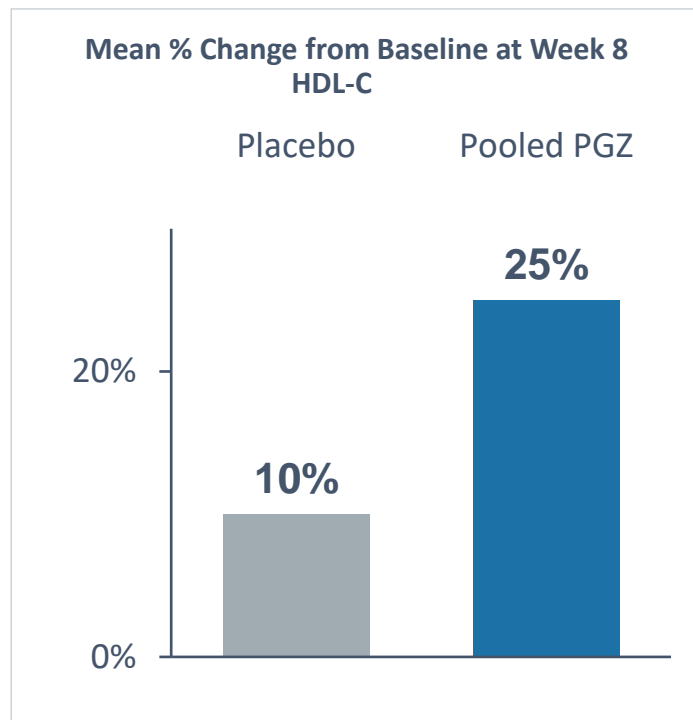
| | FGF21 | Pegozafermin |
|-----------|-----------------------|-----------------------|
| RECEPTOR | EC ₅₀ (nM) | EC ₅₀ (nM) |
| | Mean ± S.D. | Mean ± S.D. |
| KLB | nd | nd |
| KLB/FGFR1 | 4.5 ± 1.0 | 0.3 ± 0.07 |
| KLB/FGFR2 | 4.5 ± 0.9 | 1.1 ± 0.4 |
| KLB/FGFR3 | 1.8 ± 0.3 | 1.2 ± 0.4 |
| KLB/FGFR4 | nd | nd |

nd – not determined; rhFGF19 EC₅₀ at FGFR4 = 1.7 ± 0.4

Pegozafermin Shows Significant Decrease in Triglycerides on Top of Statins, Fish Oils and Fibrates



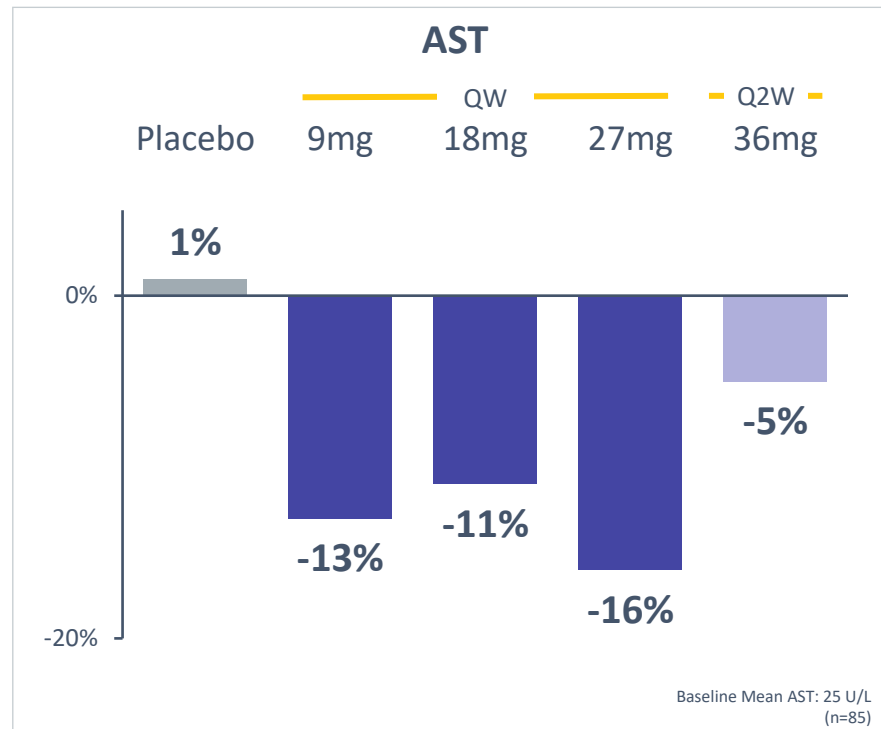
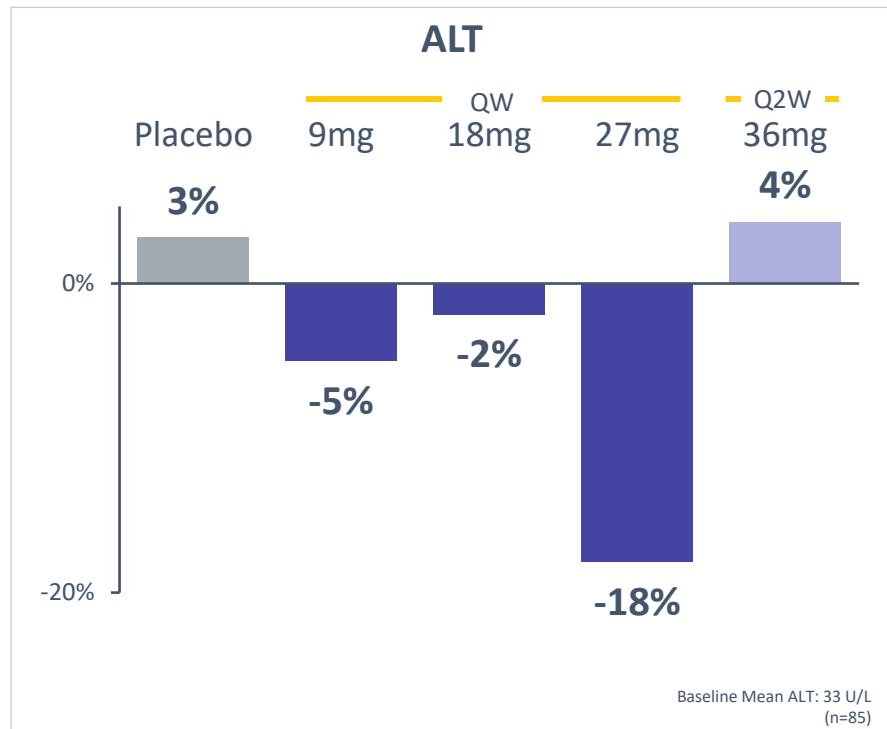
Pegozafermin Demonstrated Clinically Meaningful Improvements in HDL-C and No Change in LDL-C vs. placebo



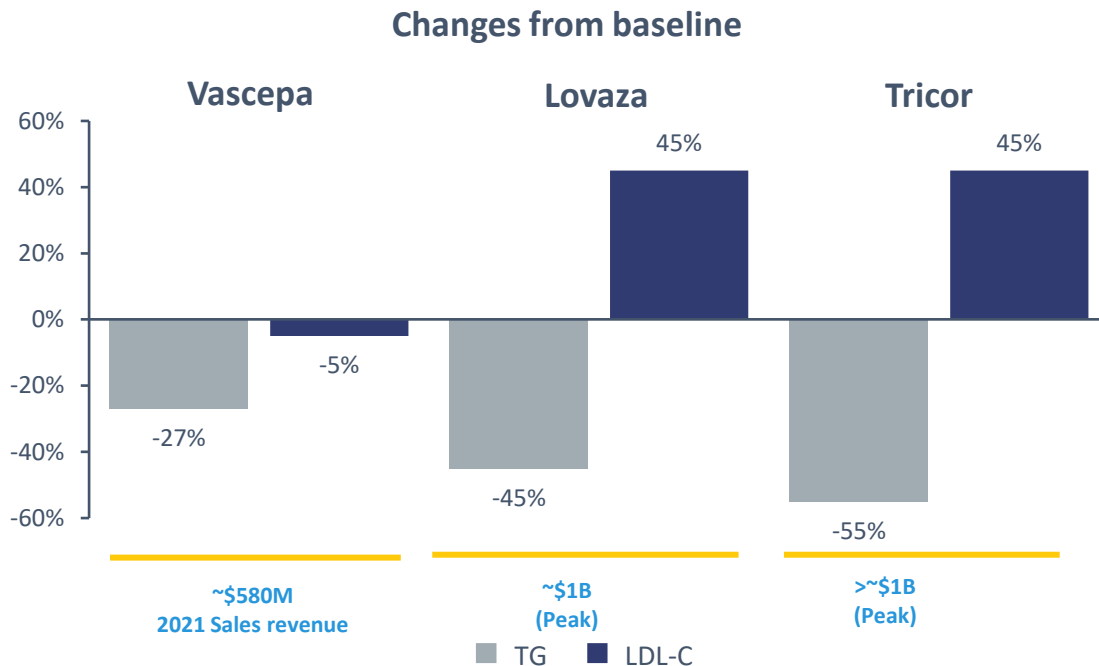
Existing therapies for SHTG increase LDL-C up to 45%

Pegozafermin Improved Liver Function Despite Normal Levels at Baseline

Mean % Change from Baseline at Week 8



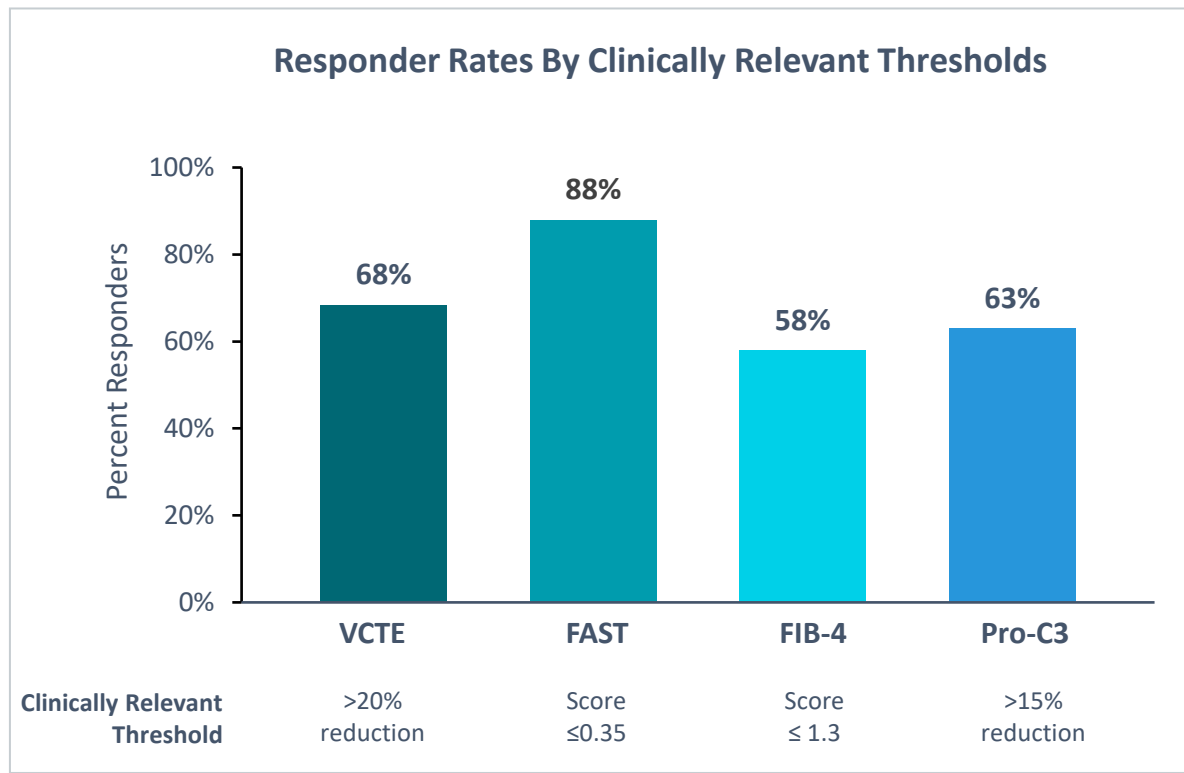
Current Therapies Have Had Commercial Success Despite Less than Ideal Profiles



| SAFETY / TOLERABILITY | | |
|---------------------------|------------------|-------------------------------------|
| FISH OILS | | FIBRATES |
| Vascepa (EPA) | Lovaza (EPA+DHA) | Tricor |
| May prolong bleeding time | | Myopathy, Creatinine increases, DDI |

Lipitor decreases TG by 39% (low dose) and 52% (high dose). Level of sales for hypertriglyceridemia are unknown.

Pegozafermin had High Percentages of Responders Based on Clinically Relevant Thresholds for NITs



VCTE and FAST data exclude one outlier with poor quality measurement

CLINICALLY RELEVANT THRESHOLDS

- VCTE: >20% reduction correlates with fibrosis improvement
- FAST score: Score ≤ 0.35 predicts Fibrosis Stage F0/F1 and NAS <4
- FIB-4 score: Score <1.3 predicts Fibrosis Stage F0/F1
- Pro-C3: >15% reduction correlates with fibrosis improvement

FGF21 Has Potential to be Leading Therapeutic Class in Development for NASH based on Broad Effects

| | FGF21 | FXR | Pan-PPAR* | THR-β | GLP-1 |
|---|------------------------|----------------------|-------------------|----------------------|------------------------------------|
| Robust efficacy with respect to liver pathologies | Liver fat reduction | ✓ | | ✓ | ✓ |
| | Fibrosis improvement | ✓ | ✓ | ✓ | |
| | NASH resolution | ✓ | ✓ | ✓ | ✓ |
| Ability to address underlying co-morbidities | Triglyceride reduction | ✓ | ✓ | ✓ | ✓ |
| | LDL-C improvement | ✓ | Worsens LDL | ✓ | |
| | Glycemic control | ✓ | ✓ | | ✓ |
| Well tolerated at effective dose | Limited Side Effects | ✓ GI effect* | Pruritis LDL ↑ | Weight Gain Edema | Drug-drug interaction GI effect |
| | Dosing frequency | Injectable QW/Q2W | Oral | Oral | Oral |
| | | | | | Injectable |

✓ Effective Unknown/Unchanged/No effect

* for certain agents

Note: Table representative of data published and/or presented on the mid/late-stage clinical programs targeting these mechanisms.
Third party company data taken from publications/publicly available presentations.

Competitor Data – Biomarkers and Non-Invasive Tests



| Parameter Mean or % | Pegozafermin 27mg | Efruxifermin 28mg / 50mg | Ocaliva 25mg | Lanifibranor 1200mg | Resmetirom Ph2b | Semaglutide 0.4mg |
|--|----------------------|---|--------------------|------------------------|--------------------|----------------------|
| MRI-PDFF $\geq 30\%$ | 100% | 100% / 100% | -- | -- | 60% | 70% |
| ALT (% chg. vs. baseline) | -46% | -40% ^E / -50% ^E | -33% | -39% ^{E,C} | -30% ^C | -58% |
| Weight (% chg. vs. baseline) | -3.9% | -0.2% ^C / -2.1% ^C | -2.3% ^C | +2.9% ^C | -0.6% ^C | -13% |
| HbA1c (chg. vs. baseline) | -0.5 -0.9* | -0.1 / -0.4 +0.1** / -0.6** | 0 | -0.4 | +0.04 | -1.15** |
| Pro-C3 (% chg. vs. baseline) | -20% | -32% ^C / -36% ^C | -- | -14% (pooled) | -38% ^E | -- |
| Adiponectin (% chg. vs. baseline) | 88% | 65% / 80% | | | 28% | |
| VCTE kPa (chg. vs. baseline) | -4.6 | -- | -1.2 ^E | -1.0 ^E | -- | -3.0 ^C |

E: value estimated based on presented data, C: calculated

* In patients with HbA1c $\geq 6.5\%$ at baseline

** in patients with Type 2 diabetes at baseline

Competitor Data – Biopsy Based Histology Endpoints

| Parameter Mean or % | Pegozafermin 27mg | Efruxifermin 28mg / 50mg | Ocaliva 25mg | Lanifibranor 1200mg | Resmetirom Ph2b | Semaglutide 0.4mg |
|------------------------------|----------------------|-----------------------------|-----------------|------------------------|--------------------|----------------------|
| %F2/F3 | 100% | 63% / 65% | 100% | 76% | 42% | 68% |
| NAS 2 point reduction | 74% | 77% / 77% | 36% | 64% | 56% | -- |
| -1 point ballooning | 79% | 85% / 85% | 35% | | 57%* | 74% |
| -1 point inflammation | 53% | 69% / 69% | 44% | | 37%* | 38% |
| -1 point steatosis | 68% | | 41% | | 57%* | 63% |
| NASH resolution | 32% [up to 47%**] | 46% / 54% | 12% | 45% | 25% | 59% |
| Fibrosis improvement | 26% [up to 42%**] | 46% / 62% | 23% | 42% | 29% | 43% |

*excludes patients who had ≥5% weight loss

** based on a new analysis of the same histology slides by a panel of an additional three expert liver pathologists

Competitor Data – AE profile in NASH program



| Key Treatment-related GI Adverse Events (> 10%) | Pegozafermin Ph 1b/2a (all doses) | Efruxifermin Ph 2a (28/50 mg) |
|---|--------------------------------------|----------------------------------|
| Diarrhea | 13% | 40% |
| Frequent bowel movements | -- | 13% |
| Nausea | 12% | 26% |
| Vomiting | -- | 18% |
| Increased appetite | 13% | 21% |
| Abdominal pain | -- | 11% |

OTHER IMPORTANT AEs:

- Efruxifermin
 - Tremor
- Ocaliva
 - Pruritis (51%)
- Lanifibranor
 - Weight gain (8%)
 - Peripheral edema (8%)