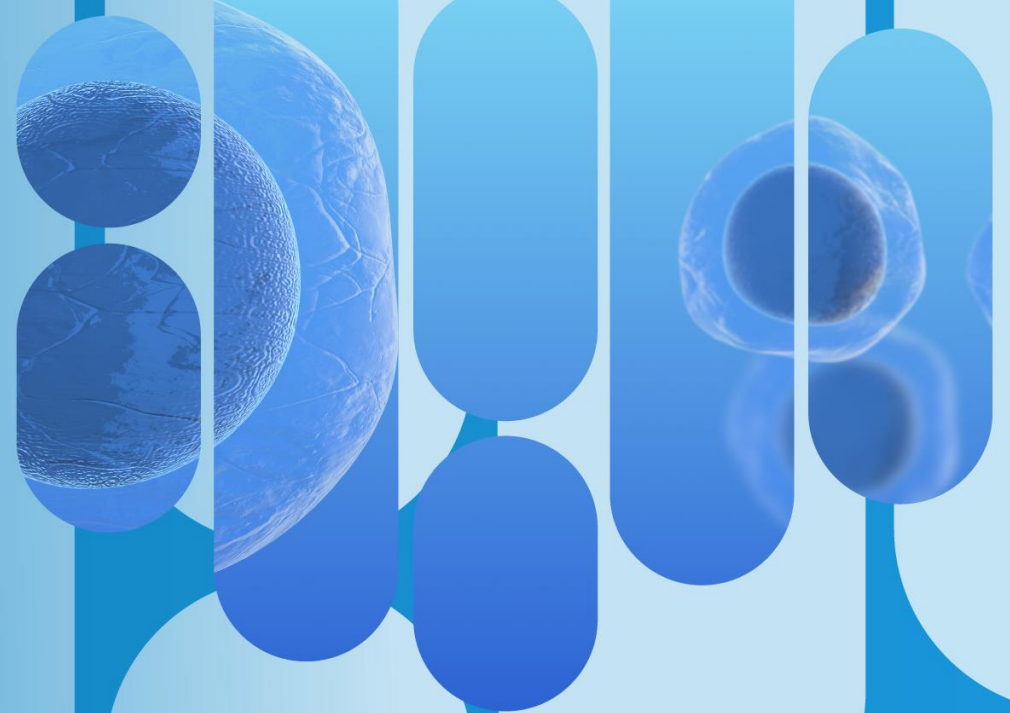


89bio

Webinar on Severe Hypertriglyceridemia

Powerful Science
Meaningful Medicines
Changing Lives



Disclaimer



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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, 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.

Today's Agenda



Introduction	Rohan Palekar
Severe hypertriglyceridemia (SHTG) - current disease state and treatment landscape	Dr. Harold Bays
Pegozafermin program – mechanism of action and clinical development	Dr. Deepak Bhatt
Phase 3 considerations and potential market opportunity	89bio
Q&A	

Speaker Introductions



Harold Bays, MD, FOMA, FTOS, FACC, FACE, FNLA, FASPC

- Dr. Harold Bays is Medical Director and President of the Louisville Metabolic and Atherosclerosis Research Center, Inc. (L-MARC), as well as Your Body Goal.



Deepak L. Bhatt, MD, MPH, FACC, FAHA, FSCAI, FESC

- Dr. Deepak L. Bhatt is Executive Director of Interventional Cardiovascular Programs at Brigham and Women's Health and Professor of Medicine at Harvard Medical School.

Severe Hypertriglyceridemia

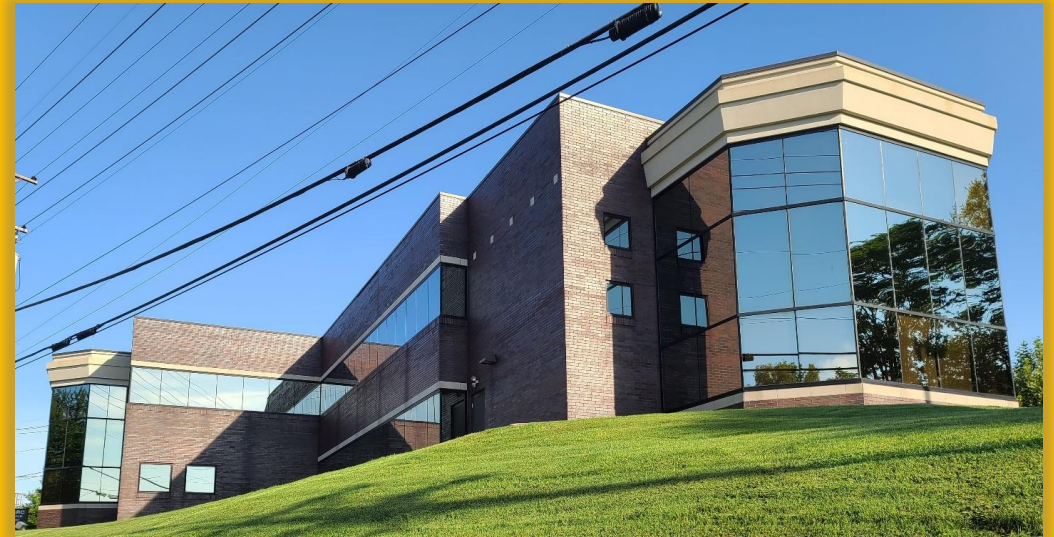
89BIO INVESTOR CALL

Wednesday May 4th 2022

Harold Edward Bays MD, FOMA, FTOS, FACC, FNLA, FASPC

Medical Director / President

Louisville Metabolic and Atherosclerosis Research Center



Disclosures

Neither Dr. Harold Bays, nor his affiliated research center institution & weight management center, own pharmaceutical stocks or patents.

In the past 12 months, Dr. Harold Bays' research site institution has received research grants from 89bio, Akcea, Allergan, Alon Medtech/Epitomee, Amgen, Anji Pharma, AstraZeneca, Axsome, BioHaven, Bionime, Boehringer Ingelheim, CinCor, Civi, CSL Behring, Eli Lilly, Esperion, Evidera, Gan and Lee, Home Access, Lexicon, Matinas, Merck, Metavant, Novartis, NovoNordisk, Pfizer, Regeneron, Sanofi, Satsuma, Selecta, and TIMI.

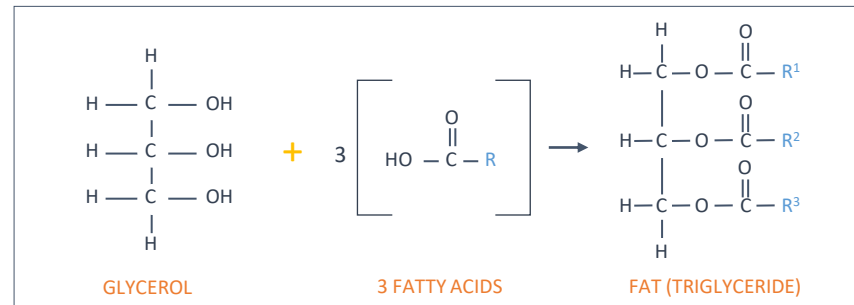
In the past 12 months, Dr. Harold Bays has served as a consultant/advisor for 89bio, Amgen, and Esperion, and speaker for Esperion.

Outline

- Hypertriglyceridemia
 - Role of TG
 - Staging
- Prevalence
- Clinical implications for SHTG
- Causes
- Diagnosis
- Treatment
 - Treatment assessment and initial management
 - ACC SHTG treatment guidelines
- Co-morbidities associated with SHTG
- Unmet need with current pharmacologic therapies
- Next generation therapy – need triglyceride lowering “plus”

Hypertriglyceridemia: Role of TGs

- The purpose of triglycerides is to serve as a storage system and reserve supply of energy
- During low food consumption, TGs can be mobilized and broken down to release energy
- Additional functions:
 - Necessary for membrane structure
 - Insulator (retention of heat)
 - Organ protection
 - Steroid and hormone production
- When production overrides catabolism, TGs are stored in organs outside of adipose tissue (e.g. liver and muscle) and blood levels can start to rise



Hypertriglyceridemia: Staging

- Serum triglyceride (TG) concentration can be stratified as:
 - Normal: <150 mg/dL
 - Borderline high: 150-199 mg/dL
 - ≥High: 200-499 mg/dL
 - **Severe: ≥500 mg/dL**
- An elevated TG level is a target for therapy when severe (≥500 mg/dL)
- **When the TG concentration is severe (≥500 mg/dL, and especially if ≥1000 mg/dL), reducing the concentration to <500 mg/dL to prevent pancreatitis becomes the primary goal of therapy**

Prevalence

Distribution of TG levels in adults ≥ 20 years old:
NHANES 2001-2006

TRIGLYCERIDE LEVEL	ATP-III GUIDELINE CLASSIFICATION	POPULATION ESTIMATE	PERCENTAGE OF POPULATION
< 150 mg/dL	Normal	133,660,634	67.8%
150-200 mg/dL	Borderline high	27,933,114	14.2%
200-<500 mg/dL	High	32,056,089	16.3%
≥500-2,000 mg/dL	SHTG	3,357,214	1.7%
>2,000 mg/dL	SHTG	81,877	0.0%
Total		197,088,927	100.0%

ATP-III, Adult Treatment Panel III; SHTG, severe hypertriglyceridemia

Roughly 3.4 million adults are estimated to have SHTG

Clinical implications for SHTG

CARDIOVASCULAR DISEASE

- Elevated triglyceride levels are independently associated with increased risk of CVD events
- Hypertriglyceridemia is associated with increased mortality in patients with known CHD
- Higher concentrations of non-fasting TGs are associated with higher risk of heart failure

CEREBROVASCULAR EVENTS

- Increased triglyceride levels are associated with an increased risk for ischemic stroke

DIABETES

- Elevated triglycerides predict incident diabetes

PANCREATITIS

- Severely elevated TG levels are associated with an increased risk of pancreatitis (9% vs 14% in patients with HTG and SHTG respectively)

NON-ALCOHOLIC FATTY LIVER DISEASE (NAFLD)

- Up to 100% of patients with SHTG can exhibit hepatic steatosis >5%

Causes*:

- Hereditary
- Acquired

HEREDITARY (GENETIC BASIS):

- Chylomicronemia
- Type Ia hyperlipoproteinemia
- Familial hypertriglyceridemia
- Familial combined hyperlipidemia
- Familial dysbetalipoproteinemia

ACQUIRED

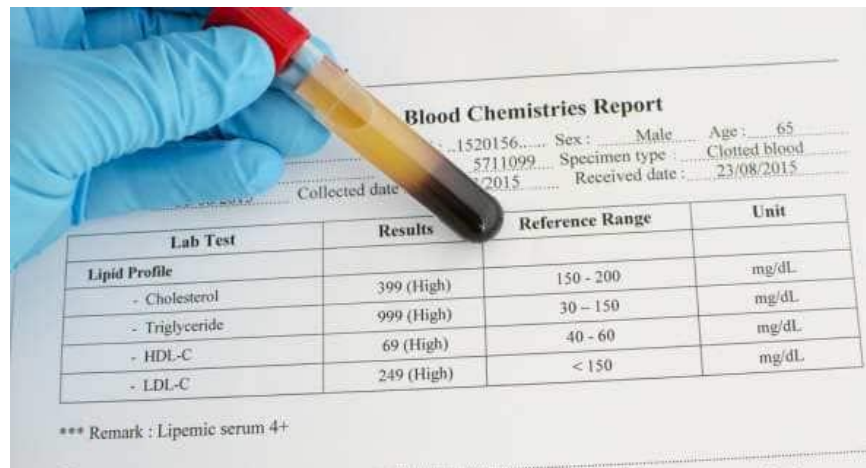
- *Obesity*
- *Metabolic Syndrome*
- *Diabetes mellitus*
- Nephrotic syndrome
- Hypothyroidism
- Pregnancy
- Medications
- Alcohol

*Causes can overlap (acquired and genetic)

Diagnosis: Lipid Panel

CDC RECOMMENDATIONS FOR CHOLESTEROL TESTING

- Every 5 years for people age 20 or older who are at low risk for cardiovascular disease
- More frequently than every 5 years for people with cardiovascular disease risk factors
- The 2018 ACC/AHA Guidelines recommend that cholesterol management be based on a person's lifetime cardiovascular risk
- Every lipid panel tests for triglycerides



ACC SHTG treatment guidelines

- Adults aged ≥ 20 years with TG ≥ 500 mg/dL
 - Rule out secondary causes
 - Optimize diet and lifestyle
 - Optimize glycemic control (in diabetes)
- Adults aged 40-75 years with TG 500-999 mg/dL with 10-year ASCVD* risk $\geq 5\%$, ASCVD or *diabetes mellitus*
 - Initiate or increase intensity of statin therapy and optimize statin adherence
- Persistent fasting TG levels 500-999 mg/dL
 - Emphasize low-fat diet
 - Increase intensity of statin therapy
 - Consider fibrate or prescription omega-3 fatty acids to ***reduce risk of pancreatitis***

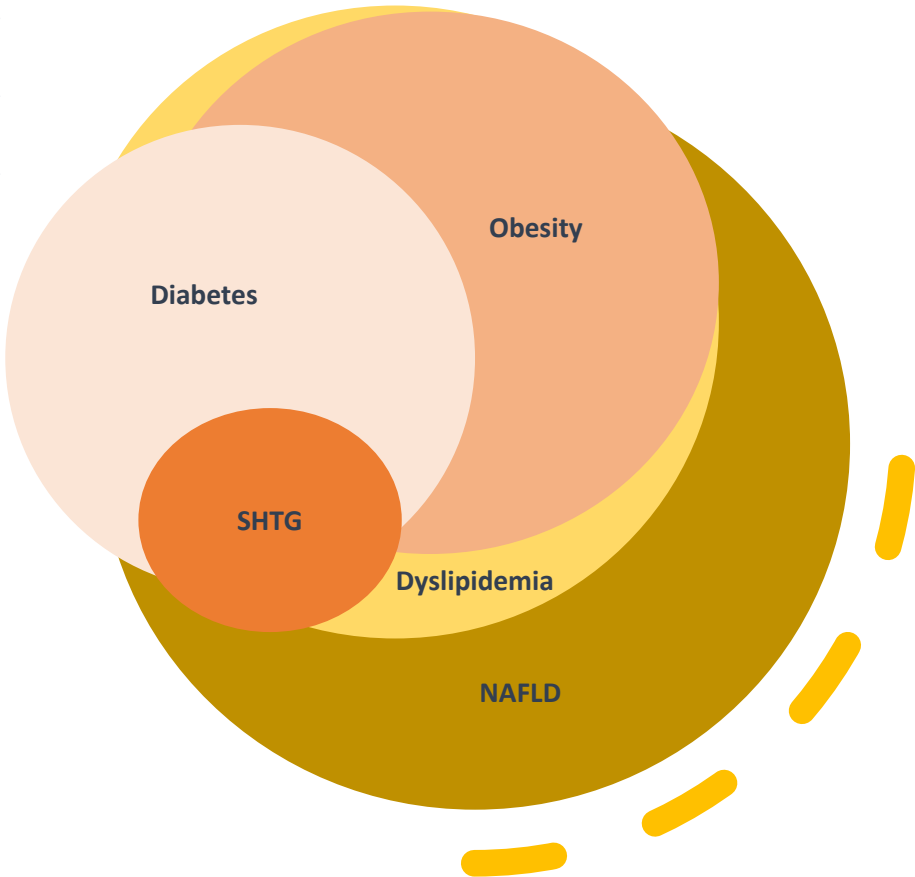
*ASCVD: Atherosclerotic cardiovascular disease

Common co-morbidities associated with SHTG

TREATING CO-MORBIDITIES, IN ADDITION TO LOWERING TG, IS A KEY UNMET NEED

OVERLAPPING CO-MORBIDITIES

Fatty Liver	Up to 100%
Type 2 Diabetes	Up to 70%
High LDL	Up to 65%
Obesity	Up to 45%



Current therapies: MOA

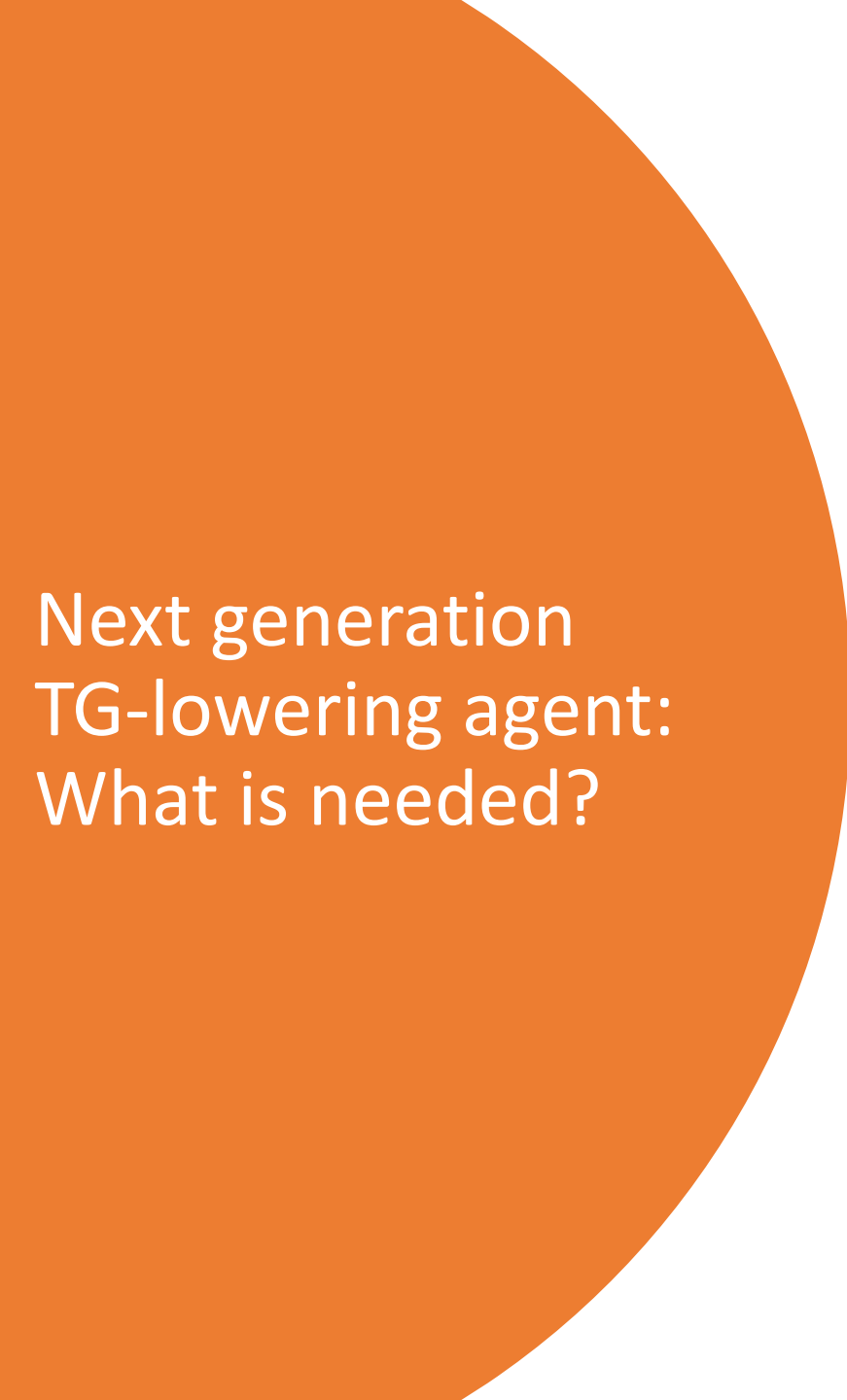
- **Statins** are generally well tolerated. The most important adverse effects are liver and muscle toxicity
- **Fibrate** treatment in patients with SHTG *may raise their LDL-C levels* by increasing intravascular lipolysis of VLDL via LPL, with resultant accumulation of newly formed LDL ('beta-shift' phenomenon)
- **Fish oils** can raise LDL-C
- **Niacin**

Current Therapies Lacking Effect on Co-Morbidities

DRUG	TG	VLDL-C	NON-HDL-C	LDL-C	HDL-C	APO-B	HBA1C	LIVER FAT
Statins	-37, -52	-45, -62	-43, -52	-31, -43	7.5, 22	N/A	↑	-
EPA/DHA	-25, -45	-21, -42	-8, -14	+20, +45	0, 9	-4, +6	-	-
EPA only	-27	-20	-8	-5	-4	-4	-	-
Fibrates	-46, -52	-40, -47	-3, -5	+13, +45	20, 23	-0.4, 3.2	-	-

Data presented as mean % change from baseline
- Unchanged or Inconclusive





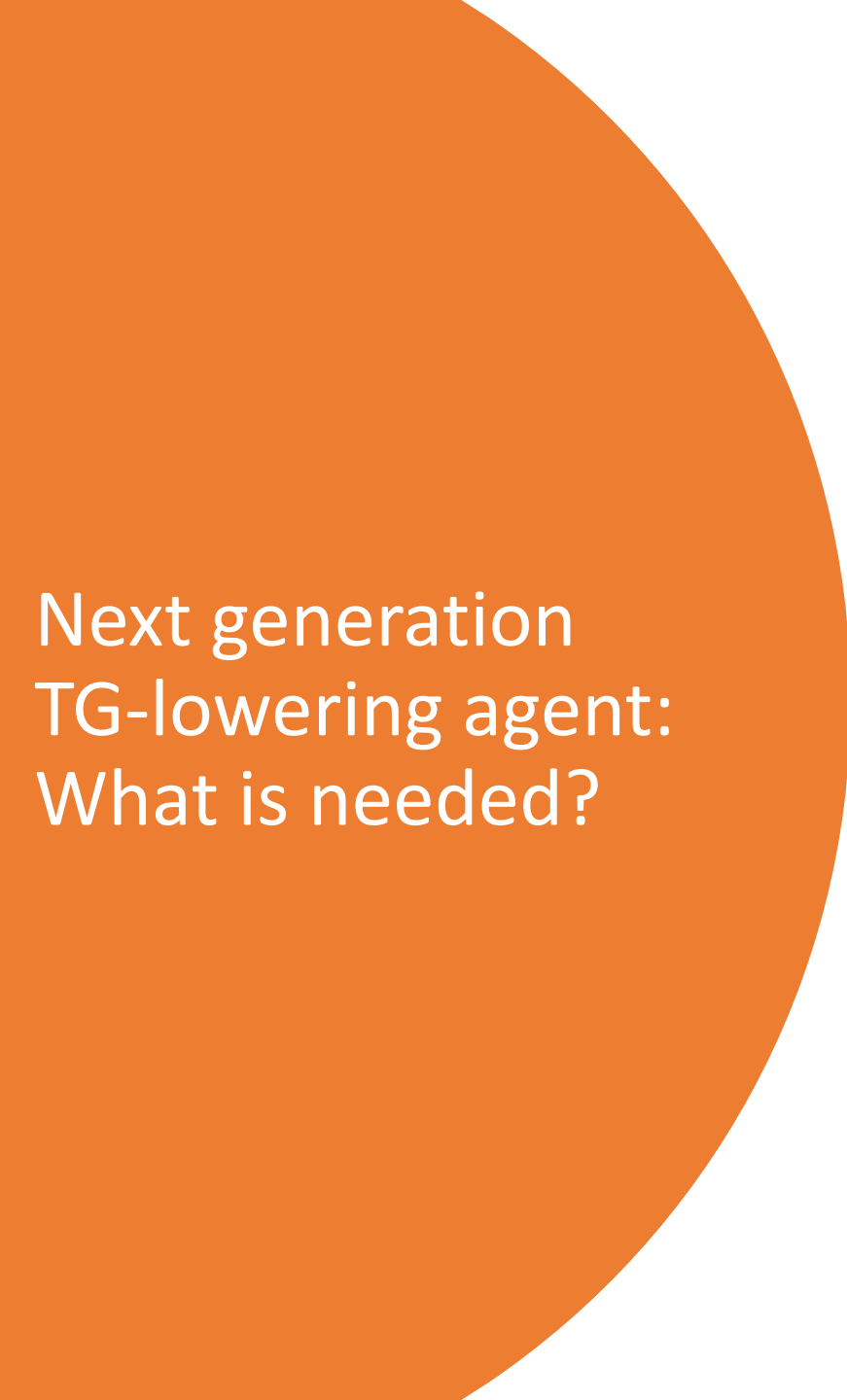
Next generation TG-lowering agent: What is needed?

- Approved for treatment of TG $\geq$ 500 mg/dL
 - Robust TG lowering as adjunct to lifestyle changes and/or add-on therapy “PLUS” favorable effects on:
 - Fatty liver
 - LDL cholesterol
 - Blood sugar
 - Body weight
- 

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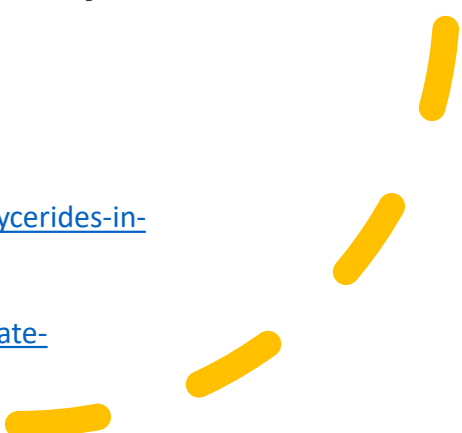
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Next generation TG-lowering agent: What is needed?

- Evinacumab Is an angiopoietin-like protein 3 (ANGPTL3) human monoclonal antibody inhibitor, which by releasing the suppression of, and thus enhances lipoprotein lipase activity, and lowers TG up to 80% in patients with SHTG and history of acute pancreatitis
- Pemafibrate CVOT study (PROMINENT) discontinued April 2022 due to futility

<https://www.news-medical.net/news/20210517/Investigational-drug-reduced-triglycerides-in-patients-with-severe-hypertriglyceridemia-acute-pancreatitis.aspx>

<https://www.prnewswire.com/news-releases/kowa-to-discontinue-k-877-pemafibrate-prominent-cardiovascular-outcomes-study-301520956.html>

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Deepak L. Bhatt, MD, MPH

Executive Director of Interventional Cardiovascular Programs,
Brigham and Women's Hospital
Professor of Medicine, Harvard Medical School



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HARVARD MEDICAL SCHOOL
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Disclosures

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Honoraria: American College of Cardiology (Senior Associate Editor, Clinical Trials and News, ACC.org; Chair, ACC Accreditation Oversight Committee), Arnold and Porter law firm (work related to Sanofi/Bristol-Myers Squibb clopidogrel litigation), Baim Institute for Clinical Research (formerly Harvard Clinical Research Institute; RE-DUAL PCI clinical trial steering committee funded by Boehringer Ingelheim; AEGIS-II executive committee funded by CSL Behring), Belvoir Publications (Editor in Chief, Harvard Heart Letter), Canadian Medical and Surgical Knowledge Translation Research Group (clinical trial steering committees), Cowen and Company, Duke Clinical Research Institute (clinical trial steering committees, including for the PRONOUNCE trial, funded by Ferring Pharmaceuticals), HMP Global (Editor in Chief, Journal of Invasive Cardiology), Journal of the American College of Cardiology (Guest Editor; Associate Editor), K2P (Co-Chair, interdisciplinary curriculum), Level Ex, Medtelligence/ReachMD (CME steering committees), MJH Life Sciences, Oakstone CME (Course Director, Comprehensive Review of Interventional Cardiology), Piper Sandler, Population Health Research Institute (for the COMPASS operations committee, publications committee, steering committee, and USA national co-leader, funded by Bayer), Slack Publications (Chief Medical Editor, Cardiology Today's Intervention), Society of Cardiovascular Patient Care (Secretary/Treasurer), WebMD (CME steering committees), Wiley (steering committee)

Other: Clinical Cardiology (Deputy Editor), NCDR-ACTION Registry Steering Committee (Chair), VA CART Research and Publications Committee (Chair); Research Funding: Abbott, Acesion Pharma, Afimmune, Aker Biomarine, Amarin, Amgen, AstraZeneca, Bayer, Beren, Boehringer Ingelheim, Boston Scientific, Bristol-Myers Squibb, Cardax, CellProthera, Cereno Scientific, Chiesi, CSL Behring, Eisai, Ethicon, Faraday Pharmaceuticals, Ferring Pharmaceuticals, Forest Laboratories, Fractyl, Garmin, HLS Therapeutics, Idorsia, Ironwood, Ischemix, Janssen, Javelin, Lexicon, Lilly, Medtronic, Merck, Moderna, MyoKardia, NirvaMed, Novartis, Novo Nordisk, Owkin, Pfizer, PhaseBio, PLx Pharma, Recardio, Regeneron, Reid Hoffman Foundation, Roche, Sanofi, Stasys, Synaptic, The Medicines Company, 89Bio; Royalties: Elsevier (Editor, Braunwald's Heart Disease); Site Co-Investigator: Abbott, Biotronik, Boston Scientific, CSI, Endotronix, St. Jude Medical (now Abbott), Philips, Svelte; Trustee: American College of Cardiology; Unfunded Research: FlowCo, Takeda

This presentation includes off-label and investigational uses of drugs and devices.

Topics

- Mechanism of Action of current therapies and development candidates
- FGF21: Novel therapeutic option
- Mechanism of Action of pegozafermin
- Review of phase 1b/2a data
- ENTRIGUE trial design
- Baseline data from ENTRIGUE
- Summary

Current therapies and development candidates: Mechanism of action

Statins:

Inhibit HMGCR (3-hydroxy-3-methylglutaryl coenzyme reductase). Although some statins have an indication for hypertriglyceridemia, they *are not indicated for SHTG*

Fibrates:

Peroxisome proliferator-active receptor (PPAR) agonists. Fibrate treatment in patients with SHTG *may raise their LDL-C levels*

Omega-3:

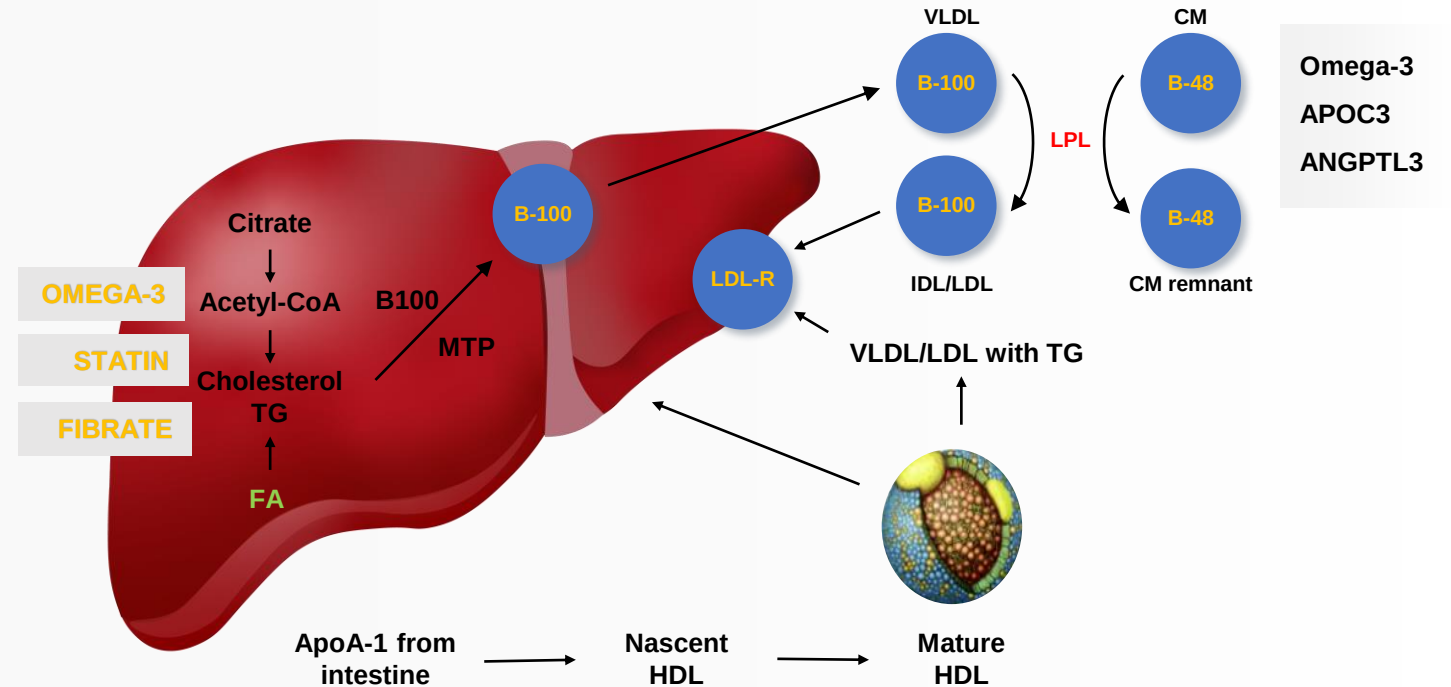
Decreases lipogenesis, increases β -oxidation and plasma LPL. *Associated with increases in LDL*

ANGPTL3 inhibitor:

Evinacumab is a monoclonal antibody inhibitor of ANGPTL3 leading to increased LPL activity. Under investigation for prevention of acute pancreatitis in SHTG

APOC3 inhibitor:

Antisense oligonucleotide and RNAi programs that targets hepatic APOC3 synthesis leading to increased LPL activity; in development for SHTG and FCS

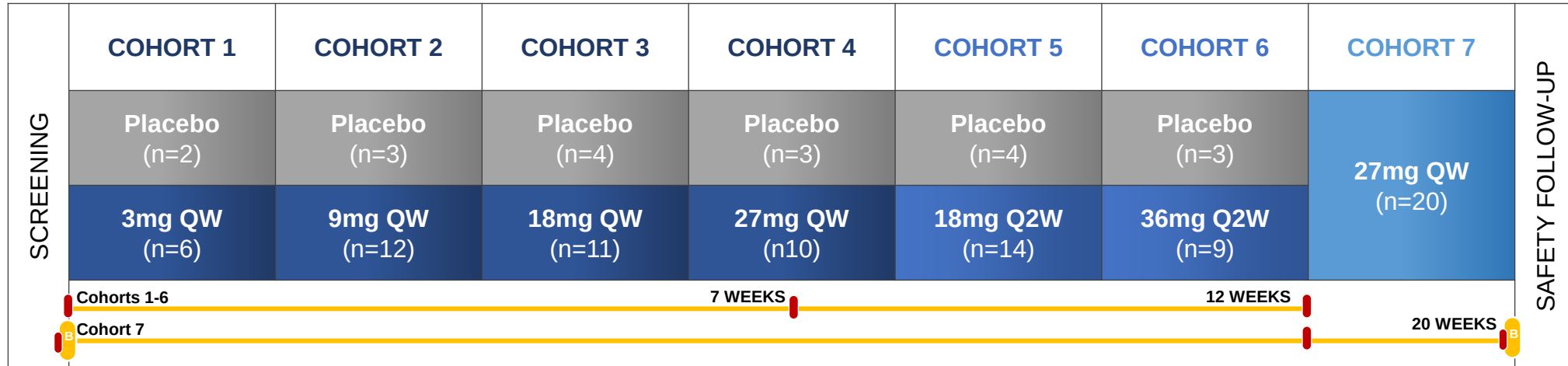


FGF21:

A novel therapeutic option for cardiometabolic disease

- FGF21 was discovered in 2000 and shown to control energy homeostasis by increasing energy expenditure under nutritional stress (starvation, amino acid restriction, ketogenic and high fat diets, simple sugars and ethanol)
- FGF 21 is a metabolic hormone that is synthesized in multiple organs, and has the ability to act on various target tissues (via endocrine, autocrine, or paracrine activity)
- The majority of circulating FGF21 is derived from the liver
- Clinical trial data have demonstrated that FGF21 analogs can:
 - Improve lipid parameters (decrease TG, LDL, Non-HDL, increase HDL)
 - Potently lower liver fat
 - Favorably impact glycemic control
 - Lead to weight reduction
 - Improve liver histology in patients with NASH

Pegozafermin phase 1b/2a NASH trial design – Randomized and open-label cohorts



COHORTS 1-6

KEY INCLUSION CRITERIA

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

- *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)

COHORT 7

KEY INCLUSION CRITERIA

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

- 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

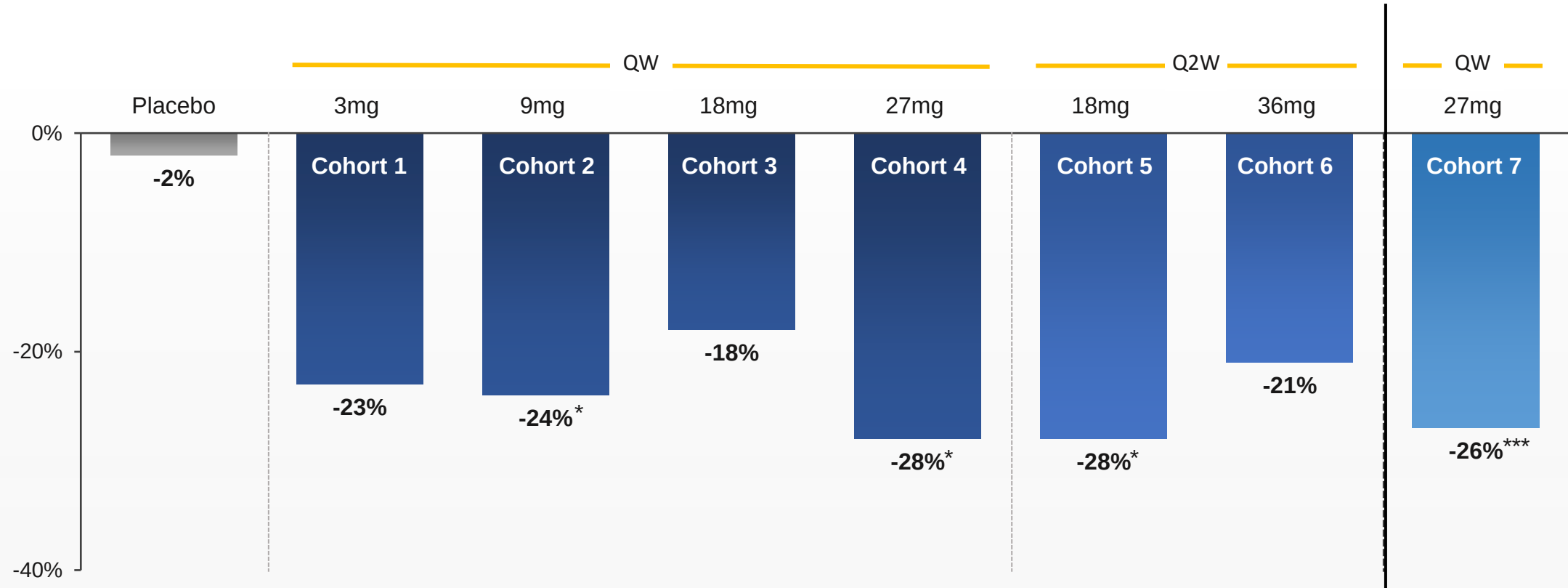
Pegozafermin is delivered subcutaneously

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%
Triglycerides (mg/dL)	174	170
LDL (mg/dL)	114	92
HDL (mg/dL)	43	43
MRI-PDFF (%)	21.3	21.1
ALT (U/L)	41.5	47.1
AST (U/L)	31.0	36.1

Pegozafermin demonstrated clinically meaningful improvements in triglycerides (cohorts 1-7)

Percentage Change from Baseline at Week 13 (Cohorts 1-6) and Week 20 (Cohort 7)

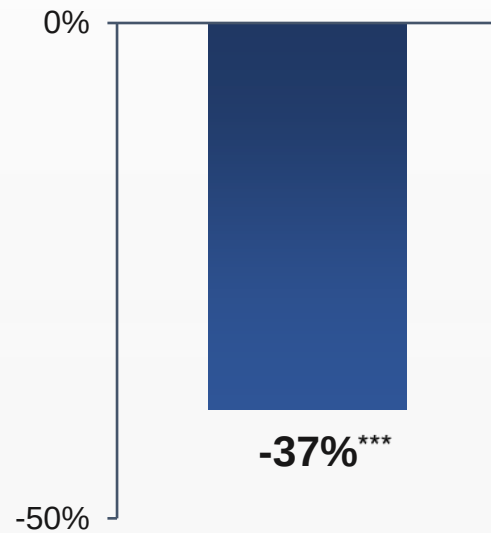


Baseline TGs: Cohorts 1-6: 174 mg/dL; Cohort 7: 170 mg/dL

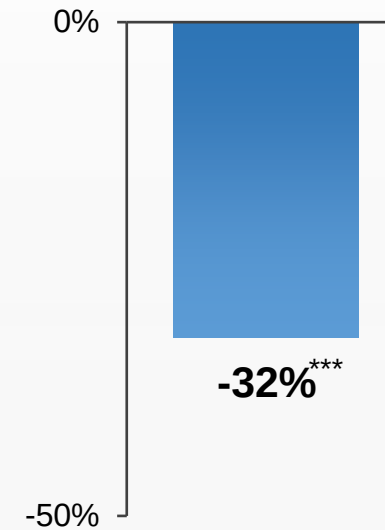
Cohorts 1-6: PD analysis set; MMRM LS Mean; * p<0.05 vs placebo; Data from week 13
Cohort 7: p value for change from baseline based on MMRM analysis; *** p<0.001; Data from week 20

Greater benefit with pegozafermin in patients with higher baseline triglycerides (cohorts 1-7)

Percent Change in TG at Week 13 in Patients with Baseline TG $\geq$ 200 mg/dL - Cohorts 1-6
(n=15, mean=288 mg/dL)



Percent Change in TG at Week 20 in Patients with Baseline TG $\geq$ 150 mg/dL - Cohort 7
(n=11, mean=210 mg/dL)



Cohorts 1-6: PD Analysis Set; MMRM LS Mean; *** p<0.001 vs baseline; Data from week 13
Cohort 7: p value for change from baseline based on MMRM analysis; *** p<0.001; Data from week 20

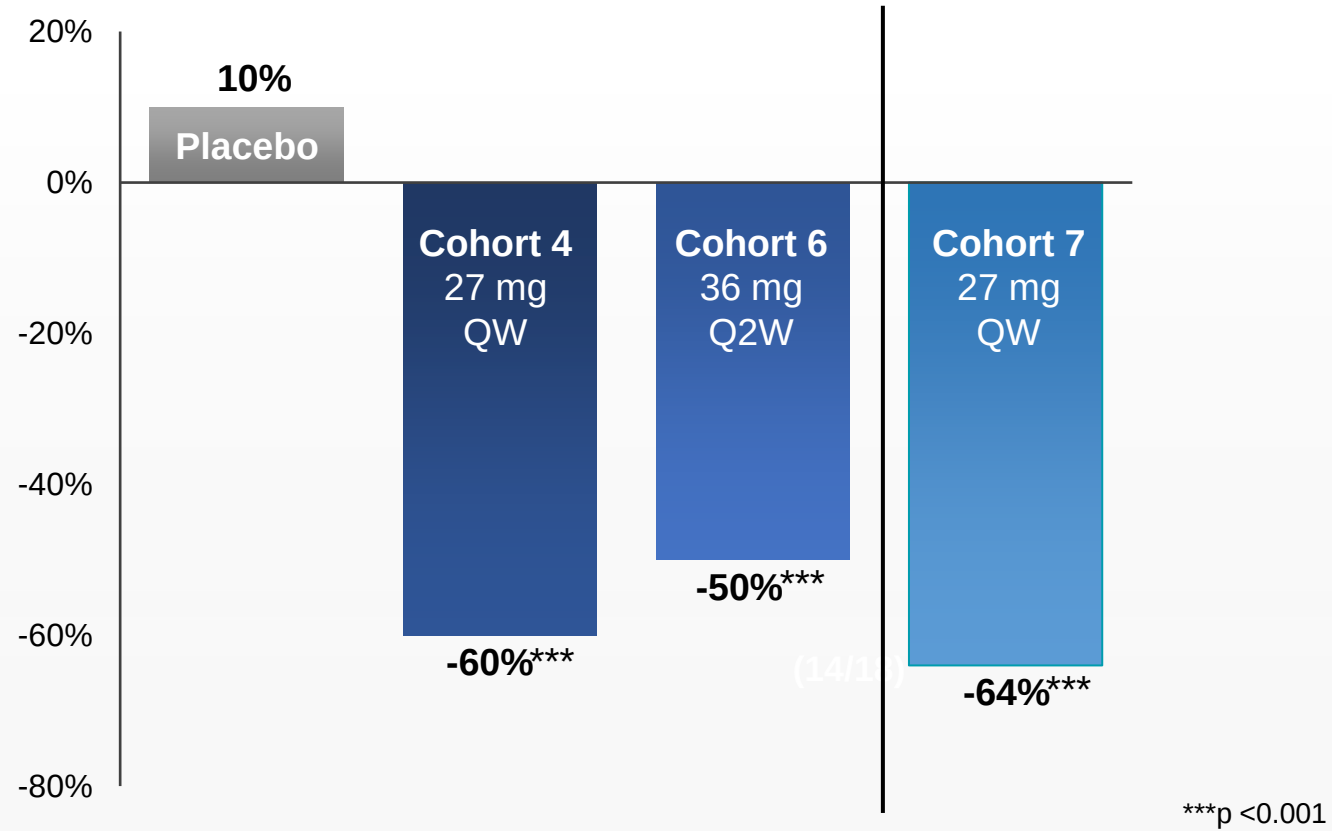
Meaningful lipid improvements relative to baseline in cohort 7 even in patients on background lipid lowering therapies

	TG	LDL-C	HDL-C	Non-HDL-C
All patients (n=19)	- 26%***	- 13%*	+ 23%***	- 18%***
Patients on lipid-lowering therapies (n=12)^	- 28%	- 17%	+ 34%	- 22%

^primarily statin therapy

Pegozafermin demonstrated robust liver fat reduction

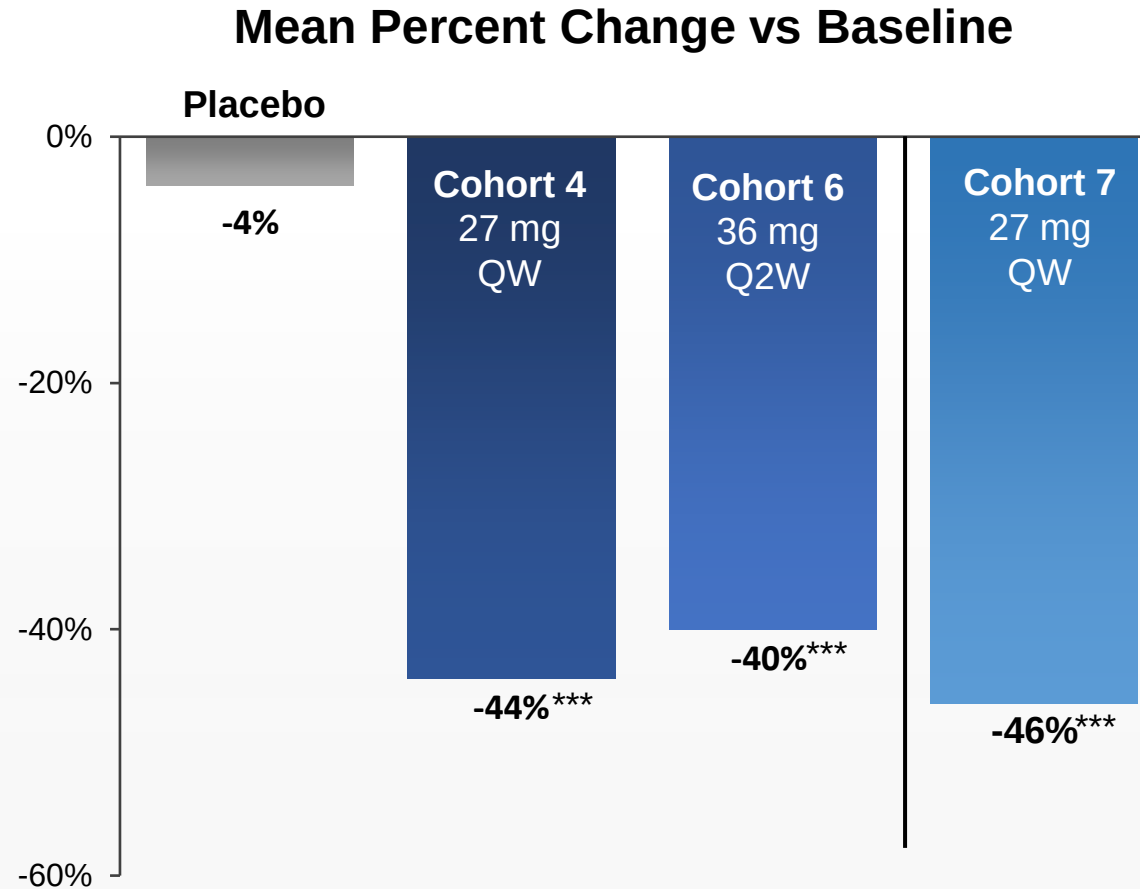
Mean Relative Reduction in Liver Fat vs Baseline



Cohort 4 and Cohort 6: MRI Analysis Set; MMRM LS Mean; p value vs placebo; Data from week 13

Cohort 7: MRI Analysis Set; p value for change from baseline based on MMRM analysis; Data from week 20

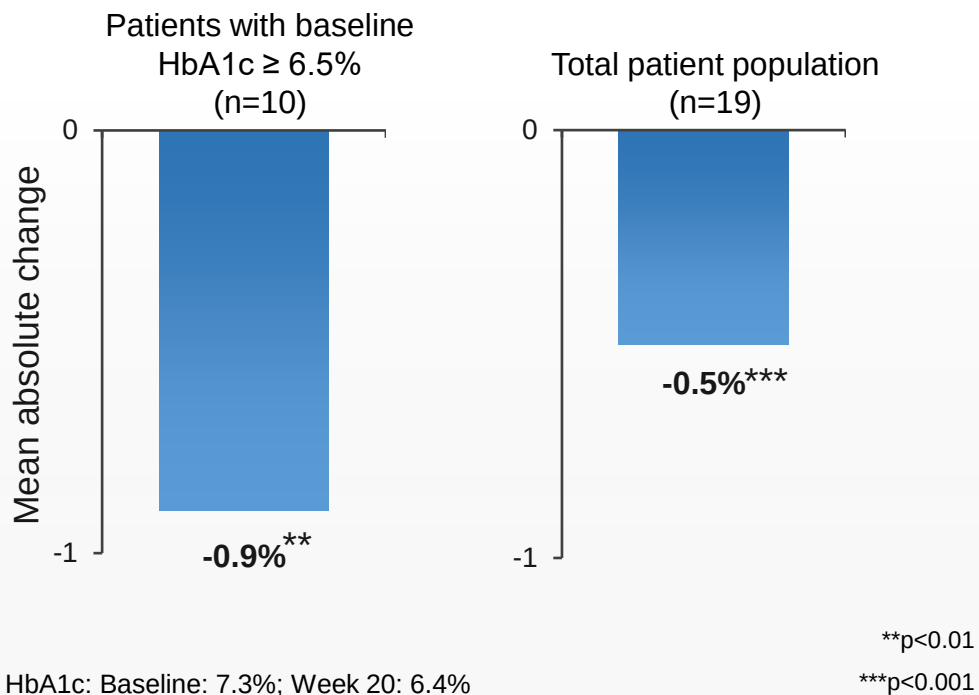
Pegozafermin significantly reduced ALT



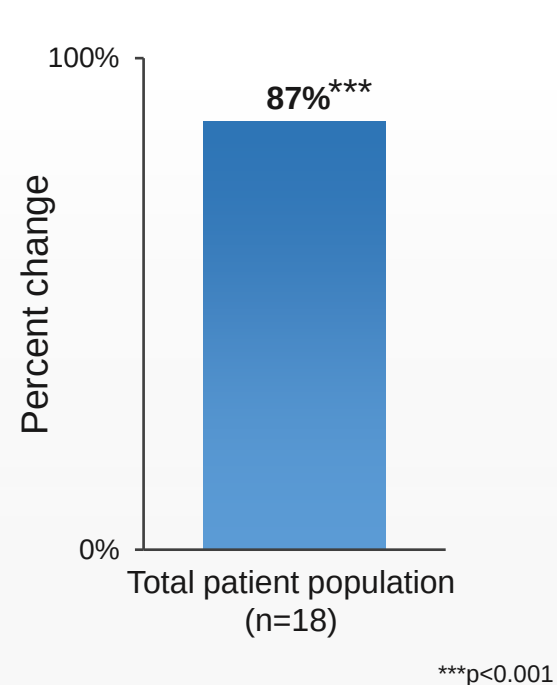
Cohort 4 and 6: Pre-planned sensitivity analysis; MMRM LS Mean at week 13; *** p<0.001 versus placebo
Cohort 7: p value for change from baseline based on MMRM analysis; Data from week 20; ***p<0.001

Pegozafermin demonstrated clinically meaningful improvement on HbA1c and adiponectin with notable body weight reduction

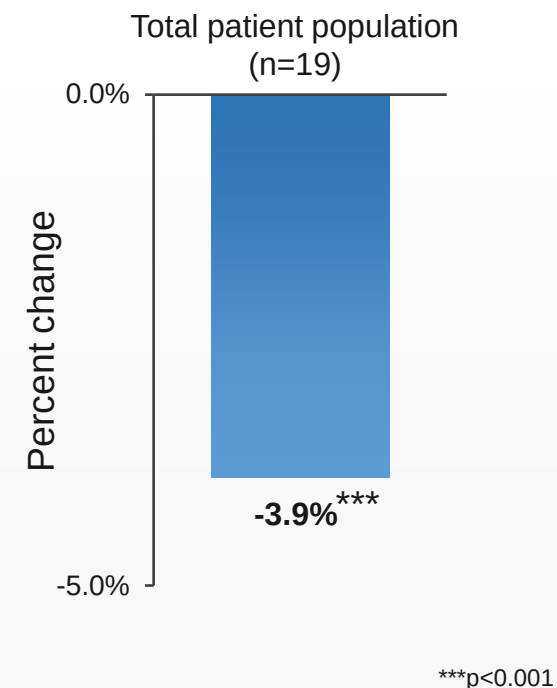
Absolute Change in HbA1c



Adiponectin Change



Weight Change

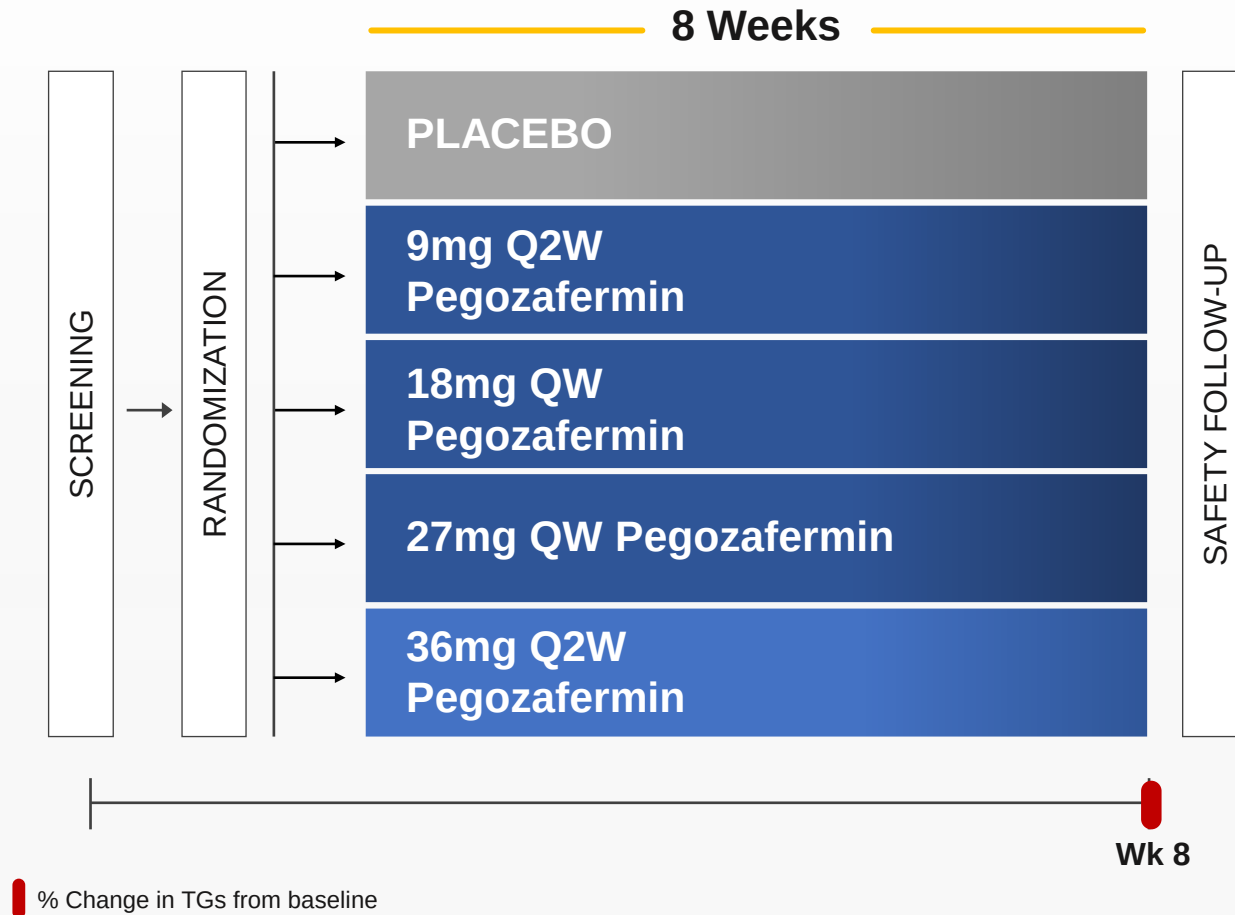


Patients with baseline HbA1c ≥ 6.5% were on an average of 2 anti-diabetic medications

Pegozafermin: Favorable safety and tolerability in phase 1b/2a study (n=83)

- 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

ENTRIGUE – Phase 2 SHTG trial design (n=85)



KEY INCLUSION CRITERIA

- TG ≥ 500 mg/dL and $\leq 2,000$ mg/dL
- Background therapy with prescription fish oil and/or statins and/or fibrates OR not on any background therapy

PRIMARY ENDPOINT

- % Change in fasting TGs from baseline

KEY SECONDARY ENDPOINTS

- Other lipids and metabolic parameters
- Liver fat (MRI-PDFF)

TG qualification process & standardization methodology for ENTRIGUE: Rigorous design to minimize TG variability

- Qualification phase
 - 4 – 6 week lifestyle & diet stabilization period / medication wash-out
 - Followed by 2 week TG qualification period
 - Must have mean of 2 TG levels taken a week apart that average ≥ 500 mg/dL*
 - Additional rules to reduce TG variability for the qualifying values
 - Baseline defined as mean of qualifying TG values
- Standardized methodology for lipid assessment visits
 - Fasting 12 – 14 hours prior to blood draw
 - No alcohol for 48 hours prior to blood draw
- Maintain stable diet, activity level, lipid lowering therapies, and anti-diabetic therapies throughout study
- Patients stratified by background lipid lowering therapy (Y/N) and by TG <750 or ≥ 750 mg/dL

*Up to 10% could qualify with average TG between 475 and 499 mg/dL

Background characteristics of ENTRIGUE population

Consistent with other SHTG studies

High frequency of metabolic comorbidities

Characteristic	Mean blinded baseline data (n=85)*
Age (years)	58
Male	64%
Body weight (kg)	99
BMI (kg/m2)	33
Diabetes	51%
Hypertension	61%
On background lipid lowering therapy	65% (46% statin or statin combo)
TG (mg/dL)	720 (39% TG ≥750 mg/dL)
LDL (mg/dL)	90
HDL (mg/dL)	28
Liver Fat Content (%) (n=24)	20

*based on April 2022 dry run data cut; may not reflect final data; study remains blinded

Patients who could potentially benefit from treatment with pegozafermin

Patients with SHTG (≥ 500 mg/dL)

Patients whose TG level is uncontrolled with current therapy

Patients with co-morbid metabolic disease

Pegozafermin is not approved for commercial use by any health authority



Summary

PEGOZAFERMIN

A POTENTIALLY DIFFERENTIATED TREATMENT FOR SHTG

**Primary effect on
triglycerides**

**Effect on metabolic
co-morbidities**

**Favorable safety
and tolerability
profile**

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Phase 3 Considerations and Potential Market Opportunity



Planning for Success: Pegzofermin Phase 3 Considerations



Well defined regulatory pathway

- Percent reduction in TGs from baseline is the accepted endpoint for full approval
- No cardiovascular outcomes study required for approval in SHTG

Phase 3 precedents for prior SHTG approvals*

- Single Phase 3 trial in SHTG patients with 75-100 patients per arm
- Primary endpoint: Percent reduction in TGs from baseline
- Trial duration: Primary endpoint at 12 weeks

Pegzofermin potential Phase 3 in SHTG patients**

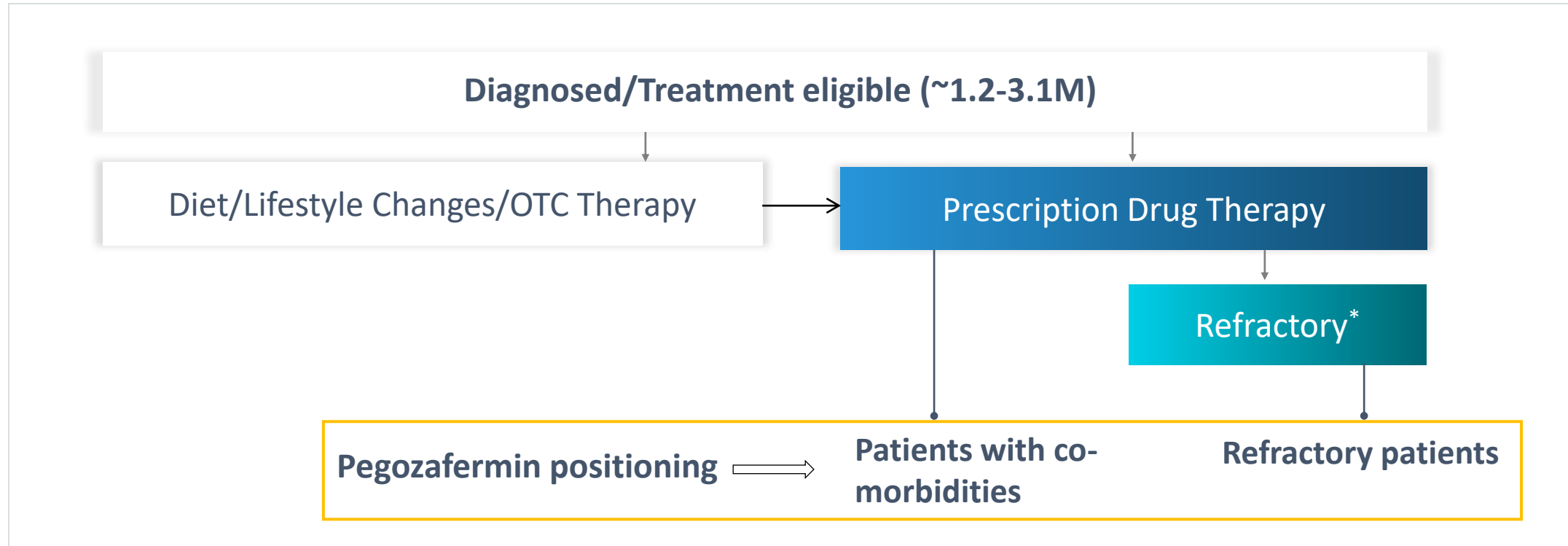
- Add-on therapy in refractory patients or patients not on background therapy
- Trial duration: 12-24 weeks
- Primary endpoint: Percent reduction in TGs from baseline
- Key secondary endpoints: liver fat reduction, glycemic control, weight, other lipids
- Safety database could potentially include trials in other hypertriglyceridemia patient populations

Pegozafermin Positioning

Patients Refractory to Existing Therapies and with Metabolic Co-Morbidities



ESTIMATED US PREVALENCE: 4.0M



ESTIMATED PEGOZAFERMIN TARGET: 400K – 1.0M

Physician Research Suggests Significant Market Opportunity for Pegzofermin



KEY REASONS TO PRESCRIBE

- Triglyceride reduction
- Improvement in other lipids
- Liver fat reduction
- Improvement in HbA1c

COMPETITIVE DIFFERENTIATION

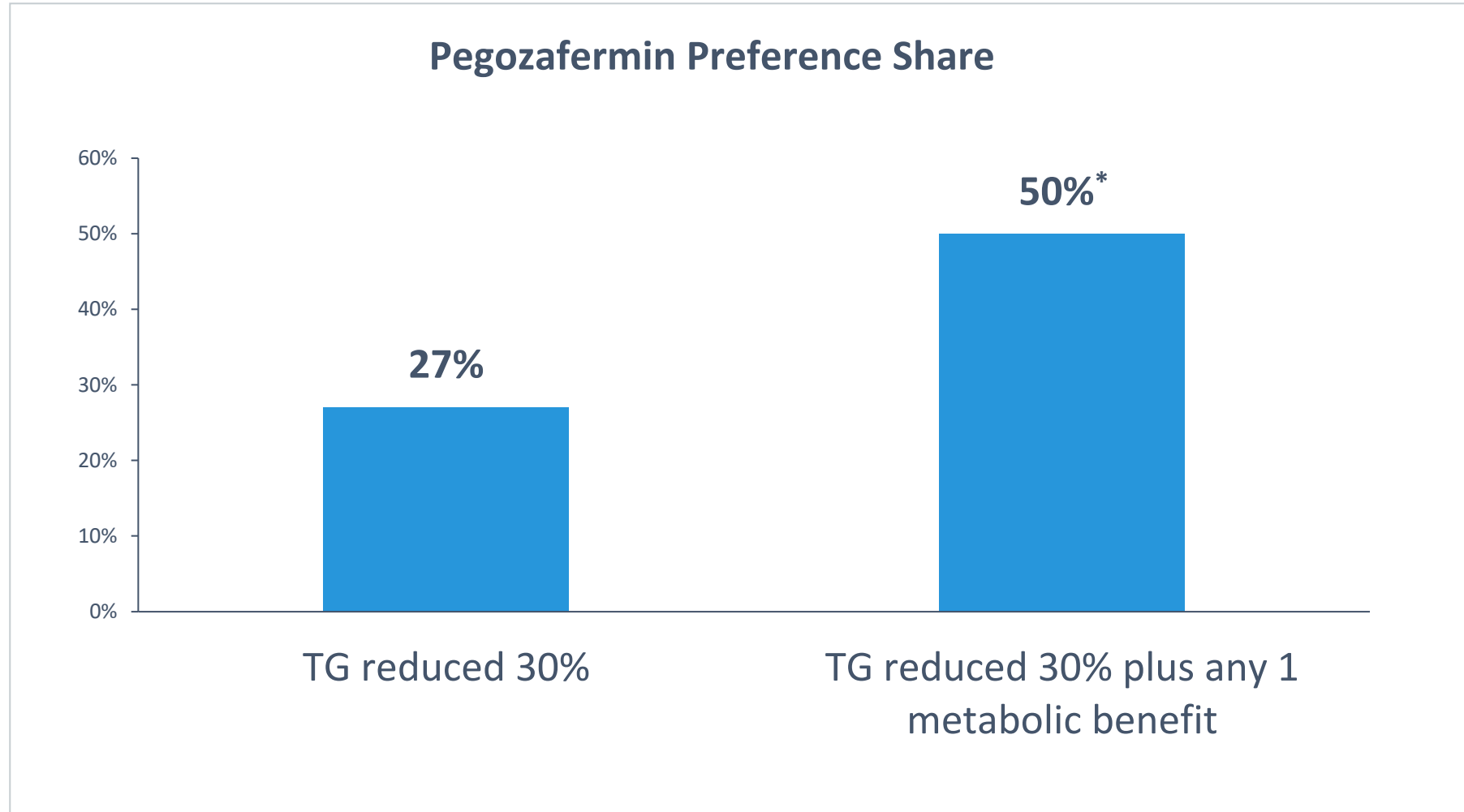
Existing therapies: Preferred to fish oils and fibrates for patients with metabolic co-morbidities

Development programs: Preferred to APOC3 for patients with fatty liver disease and T2DM

EFFICACY THRESHOLD FOR ADOPTION

- 30% triglyceride reduction from baseline in patients uncontrolled on standard-of-care drugs
- Lower threshold for patients with other co-morbidities

Potential for Significant Market Share With Modest Triglyceride Reduction Plus Metabolic Benefits



* Multiple benefits may increase share further

Advancing Pegzofermin in Clinical Development



Indication	Preclinical	Phase 1	Phase 2	Phase 3	Milestones
SHTG			Phase 2 trial	ENtrigue	2Q22
NASH			Phase 2b trial	ENliven	1H23

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Q&A

