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Patients with high LDL-cholesterol; early combination therapy

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Amsterdam, the Netherlands

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ESC CONGRESS 2022
Barcelona & Online

Conflicts of interest

- Dr Kastelein reports consultancies with 89Bio, CiVi Biotech, CSL-Behring, Draupnir Bio, Menarini Ricerce, Madrigal, North Sea Therapeutics, Novartis, Silence Therapeutics, CinCor, Scribe Therapeutics
- Dr Kastelein is acting Chief Medical Officer (CMO) of Staten Biotech and Chief Science Officer (CSO) of NewAmsterdam Pharma

Legal disclaimer

About NewAmsterdam Pharma

NewAmsterdam Pharma is a private clinical-stage biopharmaceutical company whose mission is to improve patient care in populations with metabolic diseases where traditional therapies have not been sufficiently successful or well-tolerated. NewAmsterdam Pharma is investigating obicetrapib, a next-generation oral, low-dose and once-daily CETP inhibitor, as the preferred LDL-C-lowering therapy for high-risk cardiovascular disease ("CVD") patients. Results from NewAmsterdam Pharma's ROSE Phase 2b trial (presented at AHA Scientific Sessions in 2021) included observations that patients receiving obicetrapib 10 mg experienced a median reduction in LDL-C by 51% versus baseline in patients on statin therapy (vs. a 7% reduction in the placebo arm). Based in the Netherlands, NewAmsterdam Pharma was founded in 2019 by the venture capital firm Forbion and John Kastelein, Chief Scientific Officer of the NewAmsterdam Pharma, and closed a $196 million (€160 million) Series A financing in January 2021 led by Forbion, Morningside Ventures and Ascendant BioCapital. In June 2022, NewAmsterdam Pharma entered into an exclusive licensing agreement with the Menarini Group for the commercialization of obicetrapib in Europe, while retaining all rights to commercialize obicetrapib, if approved, in the rest of the world, as well as rights to develop certain forms of obicetrapib for other diseases such as Alzheimer's disease. For more information, please visit: www.newamsterdampharma.com.

About Frazier Lifesciences Acquisition Corporation

Frazier Lifesciences Acquisition Corporation ("FLAC") is blank check company incorporated as a Cayman Islands exempted company in October 2020 for the purpose of effecting a merger, share exchange, asset acquisition, share purchase, reorganization or similar business combination with one or more businesses or entities. FLAC was formed to leverage the extensive experience and track record of its management team with the goal of financing a company that can both develop transformative therapies for patients in need and deliver significant returns to its investors. For more information, please visit: www.frazierlifesciencesacquisition.com.

About Frazier Healthcare Partners

Founded in 1991, Frazier Healthcare Partners is a leading provider of private equity capital to healthcare companies. With more than $8.1 billion total capital raised, Frazier has invested in more than 200 companies with transaction types ranging from buyouts of profitable healthcare companies to venture capital and company creation. Frazier has a philosophy of partnering with strong management teams while leveraging its internal operating resources and network to build exceptional companies. Frazier has offices in Seattle, Washington, and Menlo Park, California, and invests broadly across the U.S., Canada and Europe. For more information, please visit: www.frazierhealthcare.com.

Important Information About the Merger and Where to Find It

In connection with the previously announced business combination between NewAmsterdam Pharma and FLAC, NewAmsterdam Pharma Holding B.V. (the "Company") has filed a registration statement on Form F-4 with the SEC that includes a prospectus with respect to the Company securities to be issued in connection with the business combination and a proxy statement with respect to the shareholder meeting of FLAC to vote on the business combination. **FLAC, the Company and NewAmsterdam Pharma urge its investors, shareholders and other interested persons to read the preliminary proxy statement/prospectus, as well as other documents filed with the SEC, because these documents will contain important information about FLAC, the Company, NewAmsterdam Pharma and the transaction.** After the registration statement is declared effective, the definitive proxy statement/prospectus to be included in the registration statement will be mailed to shareholders of FLAC as of a record date to be established for voting on the proposed business combination. Once available, shareholders of FLAC will also be able to obtain a copy of the Form F-4, including the proxy statement/prospectus, and other documents filed with the SEC without charge, by directing a request to: Frazier Lifesciences Acquisition Corporation, Two Union Square, 601 Union St., Suite 3200, Seattle, WA 98101, Attn: Secretary. The preliminary and definitive proxy statement/prospectus to be included in the registration statement, once available, can also be obtained, without charge, at the SEC's website at www.sec.gov.

Participants in the Solicitation

FLAC, the Company and NewAmsterdam Pharma and their respective directors and executive officers may be deemed to be participants in the solicitation of proxies from FLAC's shareholders in connection with the proposed transaction. Information about the directors and executive officers of FLAC is set forth in FLAC's annual report on Form 10-K filed with the SEC on March 25, 2022 and is available free of charge at the SEC's website at www.sec.gov or by directing a request to: Frazier Lifesciences Acquisition Corporation, Two Union Square, 601 Union St., Suite 3200, Seattle, WA 98101, Attn: Secretary. Information regarding the persons who may, under the rules of the SEC, be deemed participants in the solicitation of FLAC's shareholders in connection with the potential transaction are set forth in the registration statement containing the preliminary proxy statement/prospectus filed with the SEC. These documents can be obtained free of charge from the sources indicated above.

Non-Solicitation

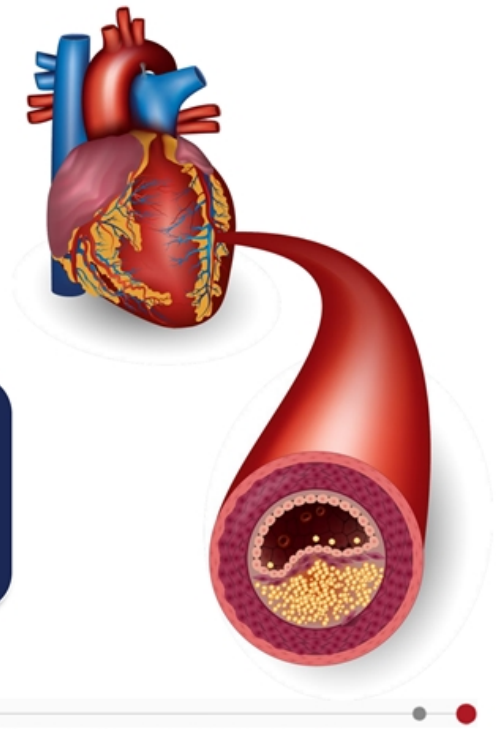
This document is not a proxy statement or solicitation of a proxy, consent or authorization with respect to any securities or in respect of the proposed business combination and shall not constitute an offer to sell or a solicitation of an offer to buy any securities nor shall there be any sale of securities in any state or jurisdiction in which such offer, solicitation, or sale would be unlawful prior to registration or qualification under the securities laws of any such state or jurisdiction. No offer of securities shall be made except by means of a prospectus meeting the requirements of the Securities Act.

Forward-Looking Statements

Certain statements included in this document that are not historical facts are forward-looking statements for purposes of the safe harbor provisions under the United States Private Securities Litigation Reform Act of 1995. Forward looking statements generally are accompanied by words such as "believe," "may," "will," "estimate," "continue," "anticipate," "intend," "expect," "should," "would," "plan," "predict," "potential," "seem," "seek," "future," "outlook" and similar expressions that predict or indicate future events or trends or that are not statements of historical matters. These forward looking statements include, but are not limited to, statements regarding estimates and forecasts of other financial and performance metrics and projections of market opportunity; expectations and timing related to the success, cost and timing of product development activities, including timing of initiation, completion and data readouts for clinical trials and the potential approval of NewAmsterdam Pharma's product candidate; the size and growth potential of the markets for NewAmsterdam Pharma's product candidate; the therapeutic and curative potential of NewAmsterdam Pharma's product candidate; financing and other business milestones; potential benefits of the proposed transactions; and expectations relating to the proposed transactions, including the proceeds of the business combination and NewAmsterdam Pharma's expected cash runway. These statements are based on various assumptions, whether or not identified in this document, and on the current expectations of NewAmsterdam Pharma's, the Company's and FLAC's management and are not predictions of actual performance. These forward looking statements are provided for illustrative purposes only and are not intended to serve as and must not be relied on as a guarantee, an assurance, a prediction, or a definitive statement of fact or probability. Actual events and circumstances are difficult or impossible to predict and may differ from assumptions. Many actual events and circumstances are beyond the control of NewAmsterdam Pharma, the Company and FLAC. These forward looking statements are subject to a number of risks and uncertainties, including changes in domestic and foreign business, market, financial, political, and legal conditions; the inability of the parties to successfully or timely enter into definitive agreements with respect to the proposed transactions or consummate the proposed transactions, including the risk that any regulatory approvals are not obtained, are delayed or are subject to unanticipated conditions (such as any SEC statements or enforcements or other actions relating to special purpose acquisition companies) that could adversely affect NewAmsterdam Pharma or the expected benefits of the proposed transactions, or the risk that the approval of the shareholders of FLAC, the Company or NewAmsterdam Pharma is not obtained; failure to realize the anticipated benefits of the proposed transactions; matters discovered by FLAC, the Company or NewAmsterdam Pharma as they complete their respective due diligence investigations of each other; risks relating to the uncertainty of the projected financial information with respect to NewAmsterdam Pharma and the Company; risks related to the approval of NewAmsterdam Pharma's product candidate and the timing of expected regulatory and business milestones; ability to negotiate definitive contractual arrangements with potential customers; the impact of competitive product candidates; ability to obtain sufficient supply of materials; the impact of COVID 19; global economic and political conditions, including the Russia-Ukraine conflict; the effects of competition on NewAmsterdam Pharma's future business; the amount of redemption requests made by FLAC's public shareholders; and those factors discussed in documents FLAC has filed or will file with the SEC, including the other risks and uncertainties described in the "Risk Factors" section of FLAC's registration statement on Form S-1, as amended (File No. 333-250858), the registration statement filed on Form F-4 in connection with the proposed transactions and other documents filed from time to time. Additional risks related to NewAmsterdam Pharma's business include, but are not limited to: uncertainty regarding outcomes of NewAmsterdam Pharma's ongoing clinical trials, particularly as they relate to regulatory review and potential approval for its product candidate; risks associated with NewAmsterdam Pharma's efforts to commercialize a product candidate; NewAmsterdam Pharma's ability to negotiate and enter into definitive agreements on favorable terms, if at all; the impact of competing product candidates on NewAmsterdam Pharma's business; intellectual property related claims; NewAmsterdam Pharma's ability to attract and retain qualified personnel; ability to continue to source the raw materials for its product candidate. If any of these risks materialize or FLAC's, the Company's or NewAmsterdam Pharma's assumptions prove incorrect, actual results could differ materially from the results implied by these forward-looking statements. There may be additional risks that neither FLAC, the Company nor NewAmsterdam Pharma presently know or that FLAC, the Company and NewAmsterdam Pharma currently believe are immaterial that could also cause actual results to differ from those contained in the forward-looking statements. In addition, forward-looking statements reflect FLAC's, the Company's and NewAmsterdam Pharma's expectations, plans, or forecasts of future events and views as of the date of this document and are qualified in their entirety by reference to the cautionary statements herein. FLAC, the Company and NewAmsterdam Pharma anticipate that subsequent events and developments will cause FLAC's, the Company's and NewAmsterdam Pharma's assessments to change. These forward-looking statements should not be relied upon as representing FLAC's, the Company's and NewAmsterdam Pharma's assessments as of any date subsequent to the date of this document. Accordingly, undue reliance should not be placed upon the forward-looking statements. Neither FLAC, the Company, NewAmsterdam Pharma nor any of their respective affiliates undertake any obligation to update these forward-looking statements, except as required by law.

Elevated levels of LDL-C are the root cause of cardiovascular disease

- Cardiovascular disease (CVD) is the leading cause of death among adults worldwide
- Hyperlipidemia nearly doubles the risk of developing CVD
- Elevated levels of LDL cholesterol (LDL-C) are the root cause of atherosclerosis, the process that leads to CVD



Absolute reduction of LDL-C,
and *duration* of that reduction,
is the *key* to reducing cardiovascular risks

Sources: American Heart Association, CDC, Mayo clinic, Global Health estimates 2016: Deaths by Cause, Age, Sex, by Country and by Regio, 2000-2016, Geneva, WHO; 2018.

How well are we treating our ASCVD patients?

Lack of goal attainment remains a serious problem in ASCVD patients

- US observational studies suggest that in patients with ASCVD, two-thirds to three-quarters of the patients are estimated NOT to be at their LDL-C goal
- A recent observation study across 18 EU countries showed approximately 7 out of 10 patients do NOT achieve their risk-based LDL-C goal, as outlined in the ESC/EAS 2019 guidelines

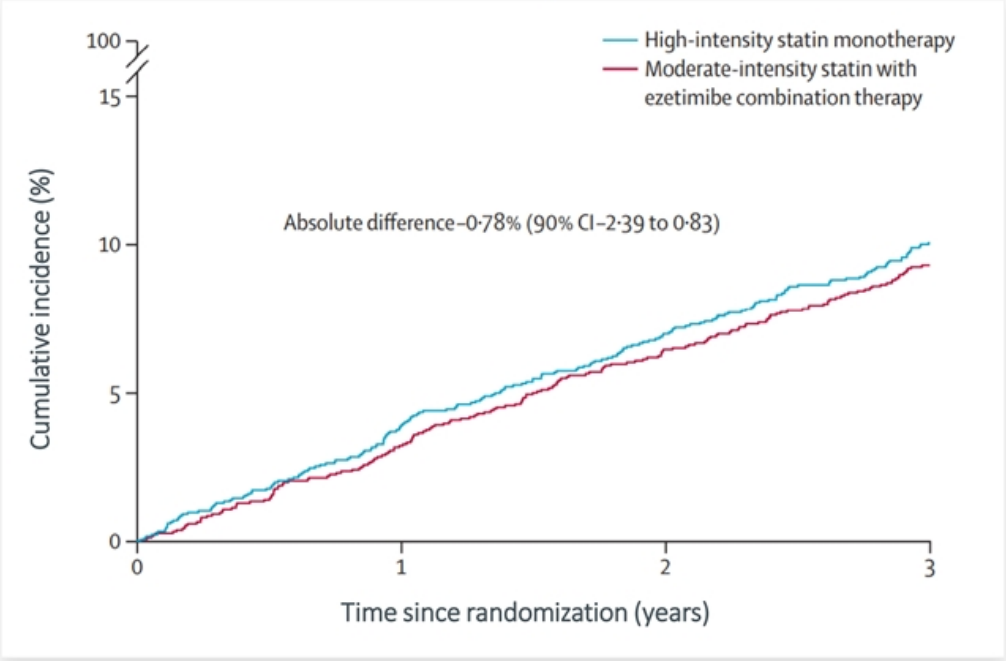
A Klimchak et al, Lipid treatment and goal attainment characteristics among persons with atherosclerotic cardiovascular disease in the United States, American Journal of Preventive Cardiology, Volume 5, March 2021, Pages 100160; Cannon, C.P., et al, for the GOULD Investigators. Use of lipid-lowering therapies over 2 years in GOULD, a registry of patients with atherosclerotic cardiovascular disease in the US. J.A.M.A. Cardiol. 6, 1060-1068 (2021); Ray KK, et. al., DA VINCI study. EU-Wide Cross-Sectional Observational Study of Lipid-Modifying Therapy Use in Secondary and Primary Care: the DA VINCI study. Eur J Prev Cardiol. 2021 Sep 20;28(11):1279-1289. doi: 10.1093/eurjpc/zwaa047. PMID: 33580789.

How can we improve?

Any recent data?

Moderate-intensity statin with ezetimibe observed to be non-inferior and better tolerated than high-intensity statin, suggesting better adherence and less relevance of the MOA for lowering LDL-C

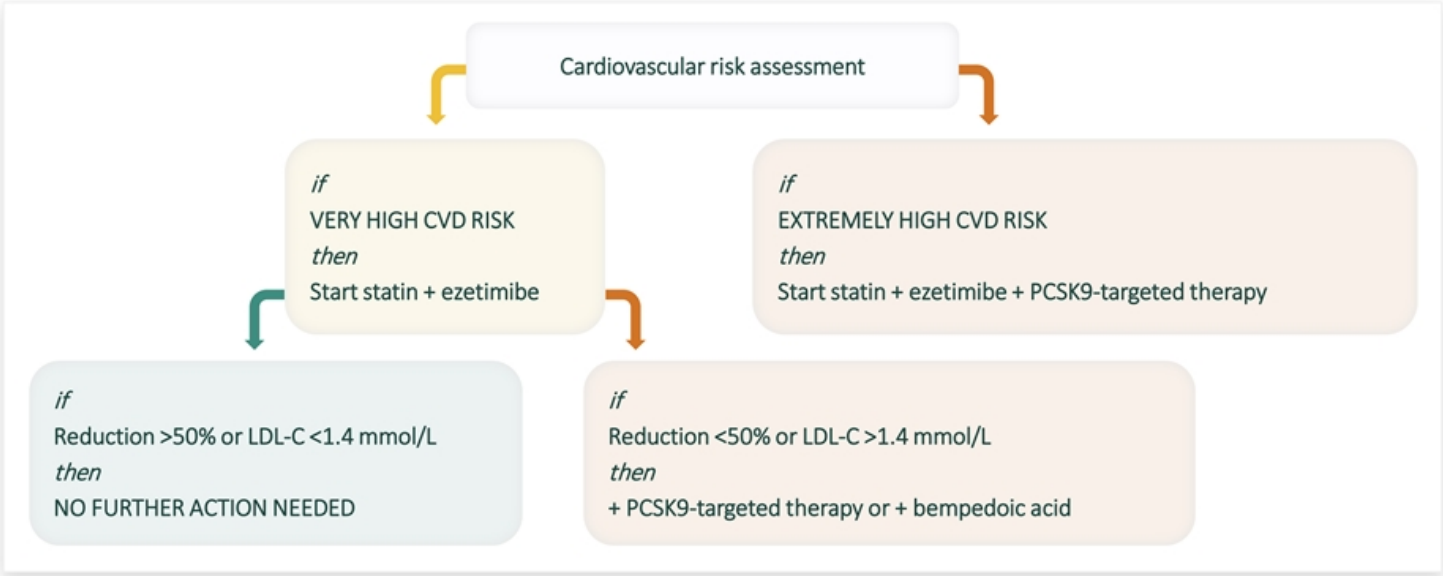
- Primary Endpoint = Composite of cardiovascular death, major cardiovascular event, or non-fatal stroke
- Moderate-intensity statin (>93% rosuva 10mg) with ezetimibe 10mg combination therapy (n=1894)
- High-intensity statin (>90% rosuva 20mg) monotherapy (n=1886)
- At year three, 72% of patient achieved LDL-C < 70mg/dl on the combination vs 58% on HIS, absolute difference 14.8% [95% CI 11.1–18.4] p<0.0001
- Discontinuation or dose reduction of the study drug by intolerance was observed in 88 patients (4.8%) on the combination vs 150 patients (8.2%) on HIS (p<0.0001)



Byeong-Keuk Kimet.al., Long-term efficacy and safety of moderate-intensity statin with ezetimibe combination therapy versus high-intensity statin monotherapy in patients with atherosclerotic cardiovascular disease (RACING): a randomised, open-label, non-inferiority trial, The Lancet, Published:July 18, 2022, DOI:https://doi.org/10.1016/S0140-6736(22)00916-3

Rationale to treat all very high- and extremely high-risk patients with combination therapy as the initial strategy

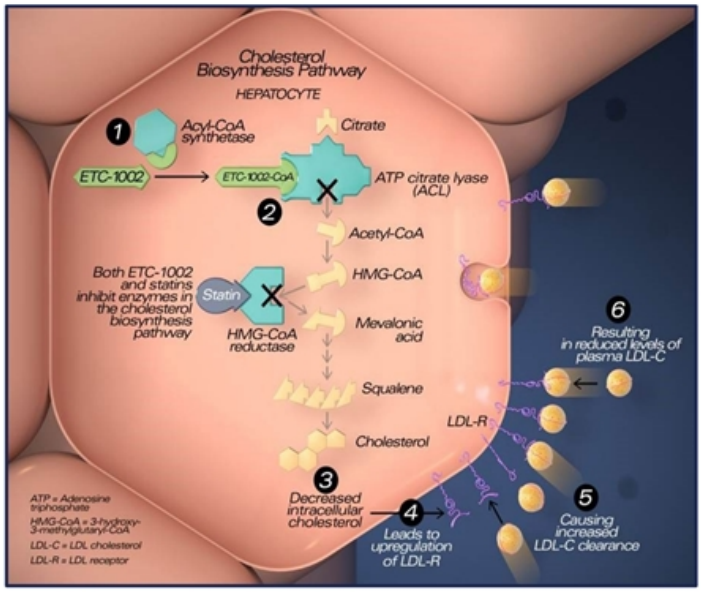
Simplified treatment algorithm for very high-risk patients with ASCVD



Ray, KK, et al., Combination lipid-lowering therapy as first-line strategy in very high-risk patients, European Heart Journal (2022) 43, 830–833

What other options are on the table or under investigation?

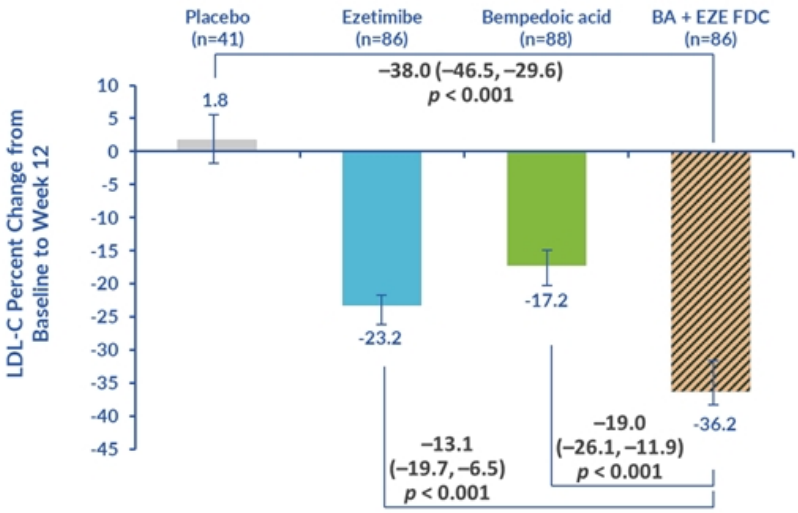
Bempedoic Acid Mechanism Of Action
Converted to ETC-1002-CoA, the Active Form, in the Liver



- Bempedoic acid (BA) acts in the same cholesterol biosynthesis pathway as statins
- BA targets **ATP-Citrate Lyase,** an enzyme upstream of HMG-CoA reductase
- Upregulates LDL receptors and lowers LDL-C
- BA is a prodrug; the specific isozyme (ACSVL1) which converts BA into an active CoA form is not present in skeletal muscle

Source: Pinkosky, S. L., Newton, R. S. et al. (2016). Nature Communications, 7, 13457 doi:10.1038/ncomms13457

Phase 3 Study of Bempedoic Acid or Ezetimibe Alone or in Fixed-dose Combination vs Placebo



BA, bempedoic acid; EZE, ezetimibe; LS, least square.

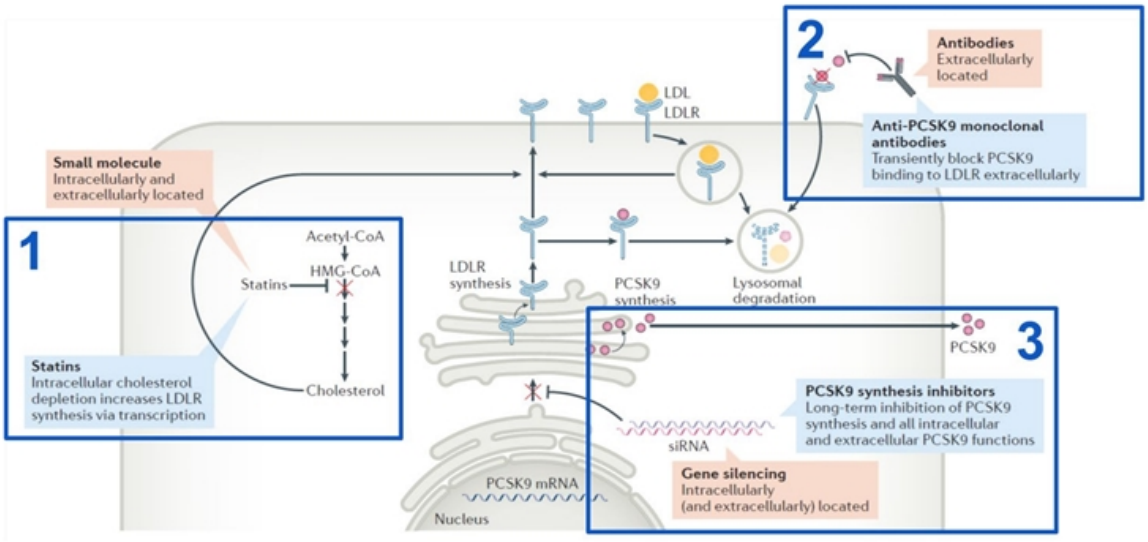
- Patients at high risk due to ASCVD or HeFH with LDL-C ≥100 mg/dL, or multiple CVD risk factors with LDL-C ≥130 mg/dL
- All treatments added to stable background maximally tolerated statin therapy
- No safety concerns

	Non-HDL-C	Apo B
	LS Mean %Δ at 12 weeks	
Placebo	1.8	5.5
EZE	-19.9	-15.3
BA	-14.1	-11.8
BA + EZE	-31.9	-24.6

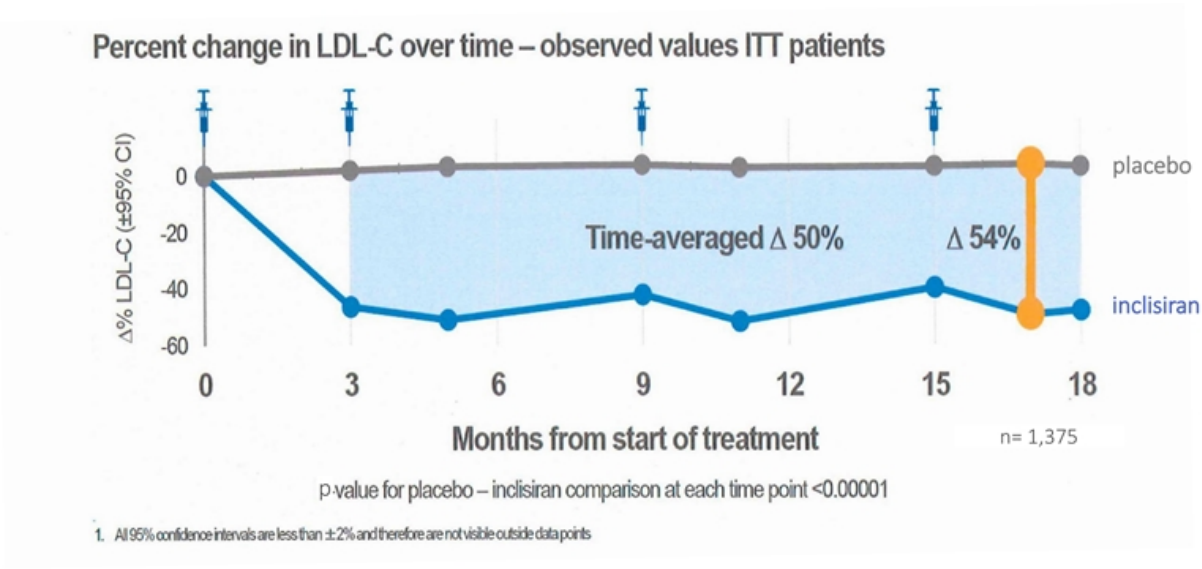
All $p<0.05$ for BA + EZE vs placebo, EZE and BA

Ballantyne CM, et al. Eur J Prev Cardiol. 2020;27:593-603.

Novel therapeutic modalities: siRNA – The third principal approach



ORION-11 Study: Targeting PCSK9 with siRNA



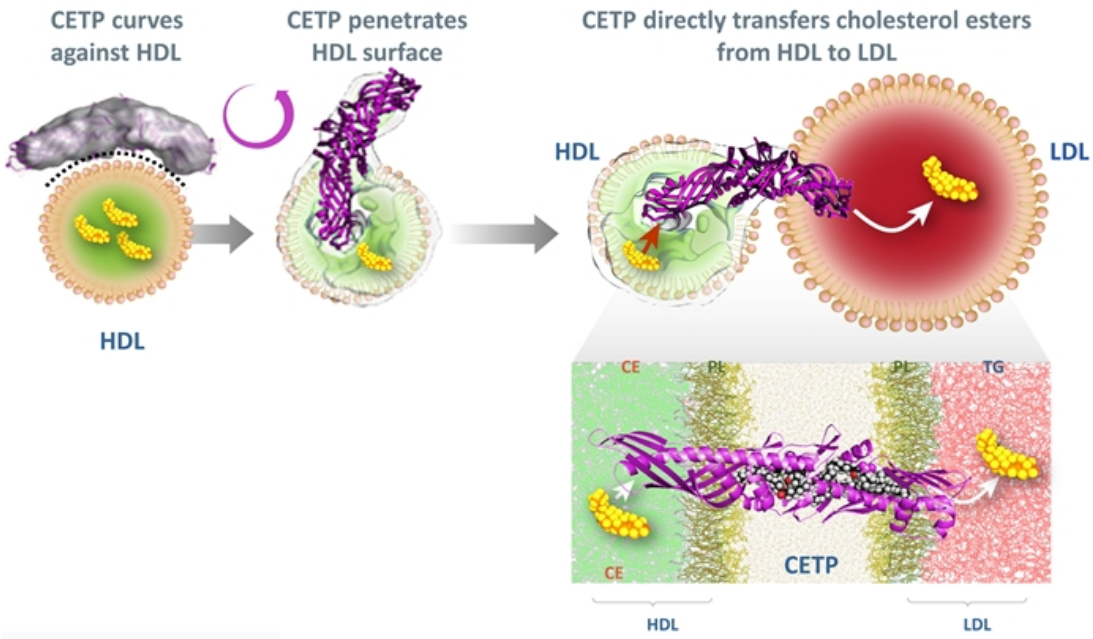
Ray, KK, et al., Two Phase 3 Trials of Inclisiran in Patients with Elevated LDL Cholesterol, April 16, 2020, N Engl J Med 2020; 382:1507-1519, DOI: 10.1056/NEJMoa1912387

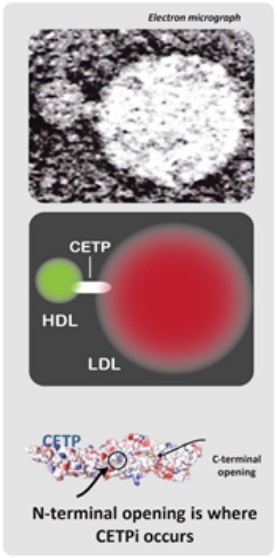
Oral PCSK9 ACTIVE Clinical Pipeline

Compound	MOA/chemistry	Admin.	Phase
CVI-LM001/ CVI Therapeutics	Small molecule modulator of PCSK9 gene expression	oral	Ph 2
NN6435/ Novo	Mimetic peptide inhibitor	SC→ oral	SC: Ph 1 SC oral: Ph 2
MK-0616/ Merck	cyclic Peptide with predecessor Ki = 2-5 pM	oral	Ph 2 DR- to complete Dec '22
AZD-0780/ Astra Zeneca- Dogma	Non-allosteric small molecule inhibitor	oral	Ph 1 (Jun '22- Jun '23)

How does CETP fit in this pathobiology?

CETP activity increases circulating LDL-C levels



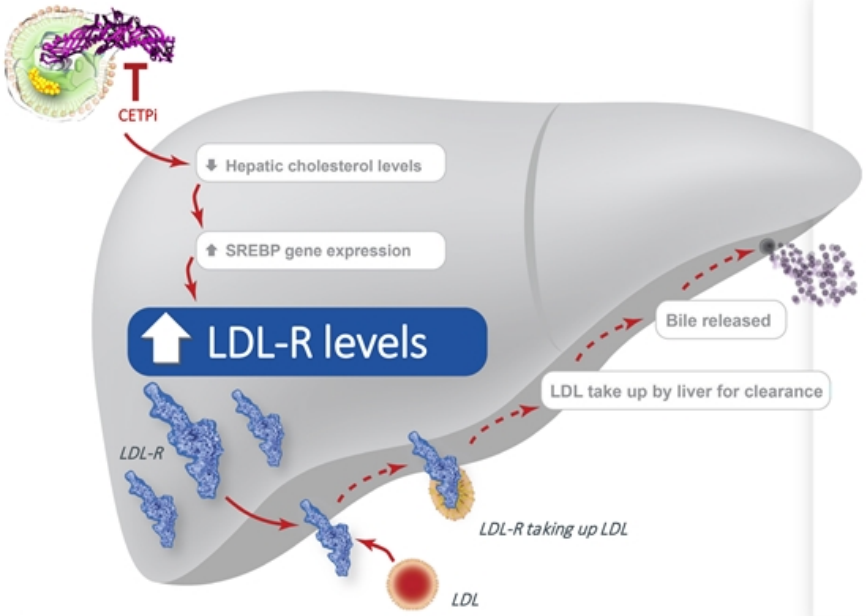


Note: Figures adapted from Meng Zhang, at al., Assessing the mechanisms of cholesteryl ester transfer protein inhibitors, Biochimica et Biophysica Acta (BBA) - Molecular and Cell Biology of Lipids, 1862(12), 2017, 1606-1617, and from Lei D, et al., Insights into the Tunnel Mechanism of Cholesteryl Ester Transfer Protein through All-atom Molecular Dynamics Simulations, J Biol Chem., 2291(27), 2016, 14034-14044.

What are the consequences of CETP inhibition?

CETPi upregulates LDL-R and improves LDL and ApoB clearance through the liver

Same mechanism of action as existing LDL-lowering drugs



Most current LDL-lowering therapies work by promoting LDL receptor upregulation

PCSK9i	STATINS	EZETIMIBE	OBICETRAPIB
Blocks degradation of LDL-R	*Decreases cholesterol synthesis*	*Decreases intestinal cholesterol absorption*	*Designed to block transfer of cholesterol from HDL>LDL*

Decrease in hepatic cholesterol

Increase in hepatic LDL receptors

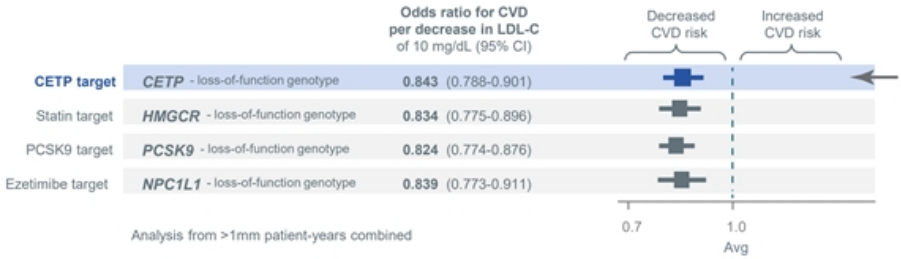
Decrease in plasma LDL levels

Adapted from:
Millar JS, et al., Anacetrapib lowers LDL by increasing ApoB clearance in mildly hypercholesterolemic subjects. J Clin Invest. 2015 Jun;125(6):2510-22. doi: 10.1172/JCI80025.
van der Tuin SJ, et al., Anacetrapib reduces (V)LDL cholesterol by inhibition of CETP activity and reduction of plasma PCSK9. J Lipid Res. 2015 Nov;56(11):2085-93. doi: 10.1194/jlr.M057794

Genomic Support that CETP Inhibition Drives CVD reduction through LDL-C Reduction

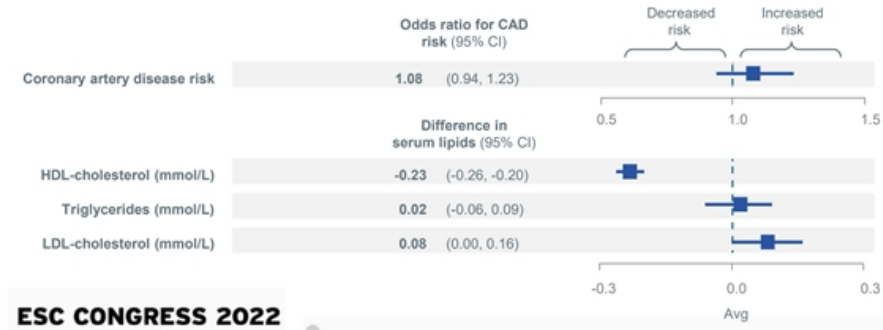
Genetic support that CETPi drives CVD benefit through LDL reduction

Analysis of >1mm patient-years' shows loss-of-function protection equivalent to targets of other LDL-lowering drugs



16% reduction in CVD risk

- A **16% reduction** in CVD risk is observed for every 10 mg/dL decrease in LDL levels
- This is ~equivalent to the effect seen in loss-of-function genotypes for **statins**, **PCSK9 modulators** and **ezetimibe**



More CETP = more CAD risk, less HDL, more LDL and more ApoB

Ference BA et al. JAMA 2017; Blauw et al; Genome-Wide Association Study on Circulating CETP.
Note: CAD means coronary artery disease.

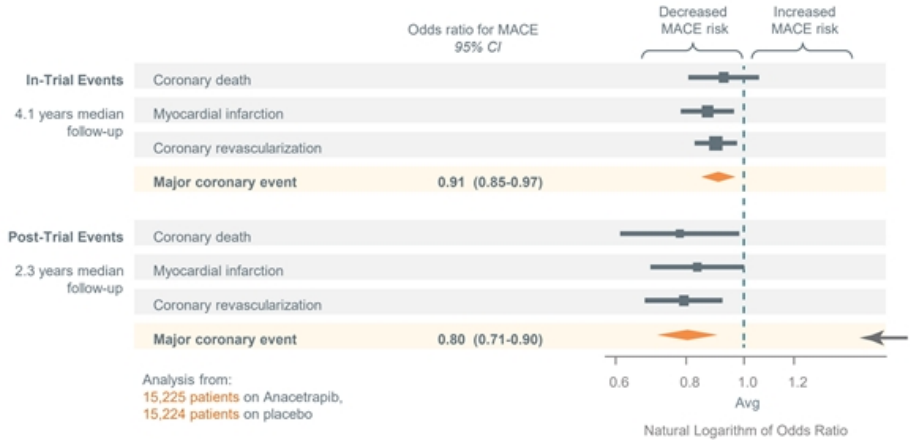
CETP, HDL-C, and the risk of CHD

- Initially, it was not appreciated that loss-of-function mutations in the CETP gene do not only lead to increased HDL-C, but also lower LDL-C, non-HDL and apoB

- We now understand that inhibiting CETP has the potential to lower CHD risk by lowering atherogenic lipids/lipoproteins (e.g. LDL-C and ApoB), NOT by increasing HDL-C

REVEAL supports translation from absolute LDL-C reduction to MACE benefit

A 2.3 year follow-up of the Phase 3 REVEAL study (6.4 years in total) with Anacetrapib showed that CETP inhibition drove a MACE reduction of 20% compared to placebo



Robust, similar decrease of MACE risk observed across all composites of the primary endpoint

MACE reduction of 20% compared to placebo (after 6.4 years of follow up)

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The HPS3/TIMI55–REVEAL Collaborative Group, Effects of Anacetrapib in Patients with Atherosclerotic Vascular Disease, September 28, 2017, N Engl J Med 2017; 377:1217-1227, DOI: 10.1056/NEJMoa1706444

The HPS3/TIMI55-REVEAL Collaborative Group, REVEAL Collaborative Group, Long-term safety and efficacy of anacetrapib in patients with atherosclerotic vascular disease, European Heart Journal, Volume 43, Issue 14, 7 April 2022, Pages 1416–1424, https://doi.org/10.1093/eurheartj/ehab863

Obicetrapib

A Placebo-Controlled, Double-Blind, Randomized, Phase 2 Dose-Finding Study to Evaluate the Effect of Obicetrapib 5 and 10 mg as an Adjunct to High-Intensity Statin Therapy

Stephen J Nicholls, Marc Ditmarsch, John J Kastelein, Scott P Rigby, Douglas Kling, Danielle L Curcio, Nicholas J Alp, Michael H Davidson

ROSE study: Obicetrapib and High-Intensity Statin therapy (HIS)

Objective To evaluate the effect of obicetrapib on top of HIS on LDL-C

Inclusion criteria

- A stable dose of HIS (A 40 / 80; R 20 / 40) 8 weeks prior to screening
- Fasting LDL-C levels >1.8 mmol/L

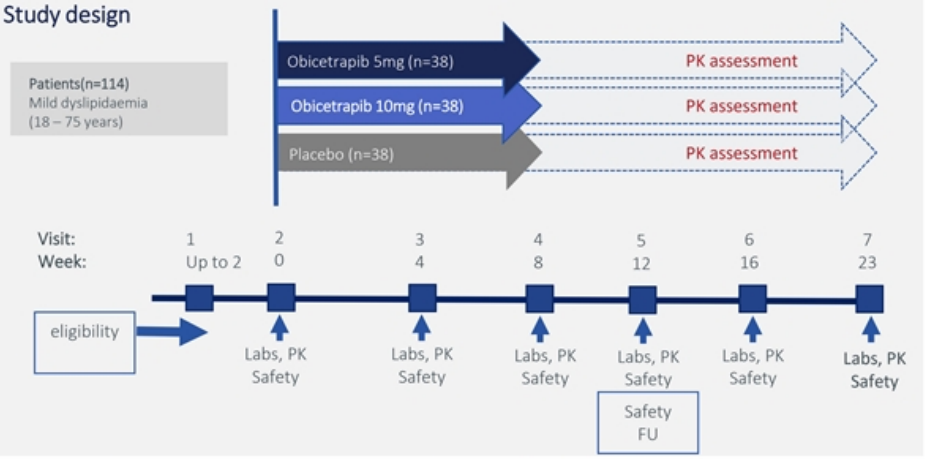
Exclusion criteria

- Current significant CV disease
- Diagnosis of type 1 or type 2 diabetes mellitus;
- Uncontrolled hypertension

Primary efficacy endpoint

- Percent change from baseline in LDL-C compared to the placebo group

Study design



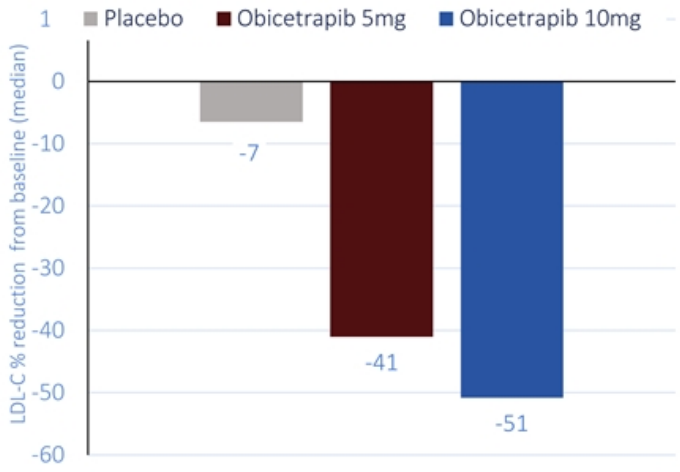
Pre-specified assessment of LDL-C levels by preparative ultra-centrifugation and Friedewald

Baseline characteristics

		Placebo (N=40)	Obicetrapib 5 mg (N=40)	Obicetrapib 10 mg (N=40)
	Mean Age (yrs)	61.3	61.1	62.9
	Female %	52.5	42.5	37.5
	Mean BMI (kg/m^2)	30.2	32.2	30.8
Race %	White	80.0	75.0	75.0
	Black / African American	17.5	25.0	12.5
Statin use (%)	Atorvastatin 40mg	50.0	50.0	62.5
	Atorvastatin 80mg	20.0	25.0	27.5
	Rosuvastatin 20mg	5.0	12.5	7.5
	Rosuvastatin 40mg	25.0	12.5	2.5
Baseline level	LDL-C (mg/dL by PUC)	90.0	95.0	88.0
	HDL-C (mg/dL)	44.5	46.5	44.0
	Lp(a) (nmol/L)	45.3	89.4	29.9

In ROSE Phase 2b clinical trial, obicetrapib demonstrated robust LDL-C lowering as adjunct to high intensity statins

Preparative ultra-centrifugation (PUC) is "gold-standard" for LDL-C quantification



Median (min, max) LDL-C levels (mg/dL) at baseline and EoT

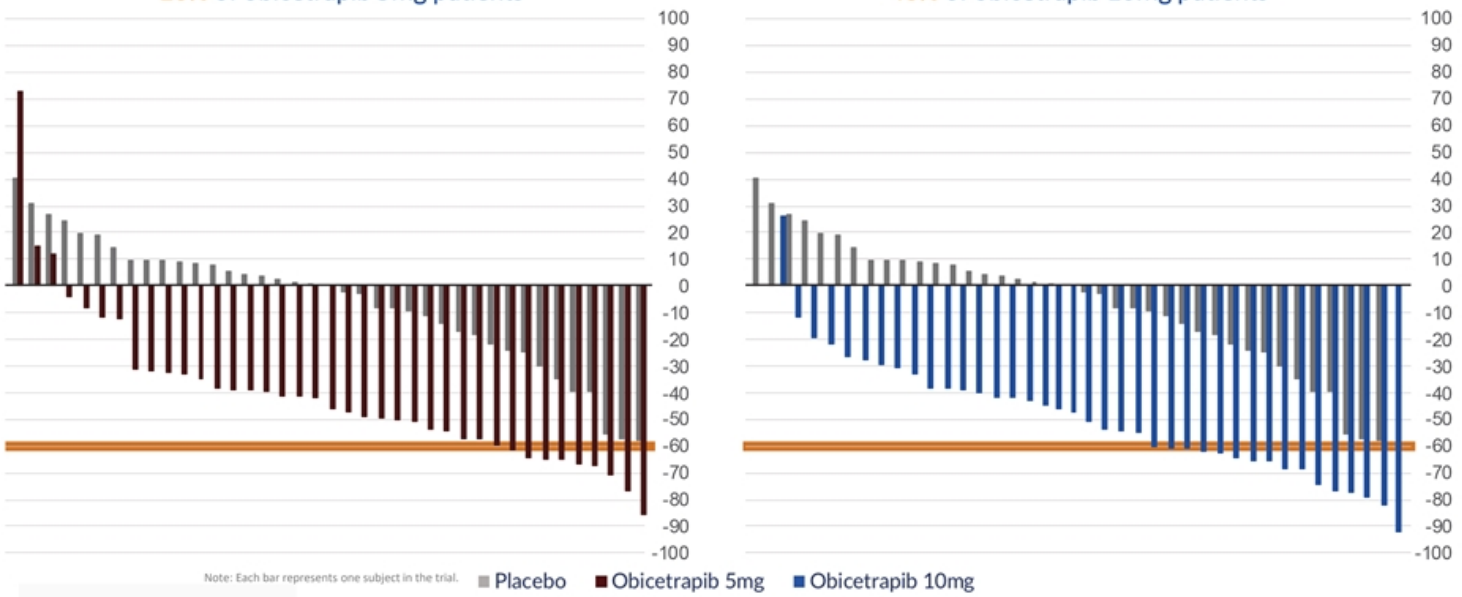
Time	Placebo	Obicetrapib 5mg	Obicetrapib 10mg
Baseline Median	90.0	95.0	88.0
	(63, 204)	(54, 236)	(39, 207)
	N=40	N=39	N=40
EoT Median	86.0	53.0	49.5
	(43, 137)	(13, 126)	(23, 83)
	N=39	N=39	N=40
% Change from Baseline (median)	-6.5	-41.45	-50.75
	(-53.9, 31.6)	(-71.2, 62.3)	(-76.9, 15.6)
% Change from Baseline LS mean (95% CI)	N=39 -4.76 (-11.74, 2.22)	N=38* -37.98 (-44.80, -31.17)	N=40 -44.15 (-50.95, -37.35)
P-value	0.1814	<0.0001	<0.0001

* In obicetrapib 5mg arm, 40 patients were randomized. N-value at end-of-treatment decreased to 38 because one patient was missing an LDL value at baseline and a second patient was missing an LDL value at end-of-treatment reading.

ROSE waterfalls: LDL-C % change from baseline at Day 56

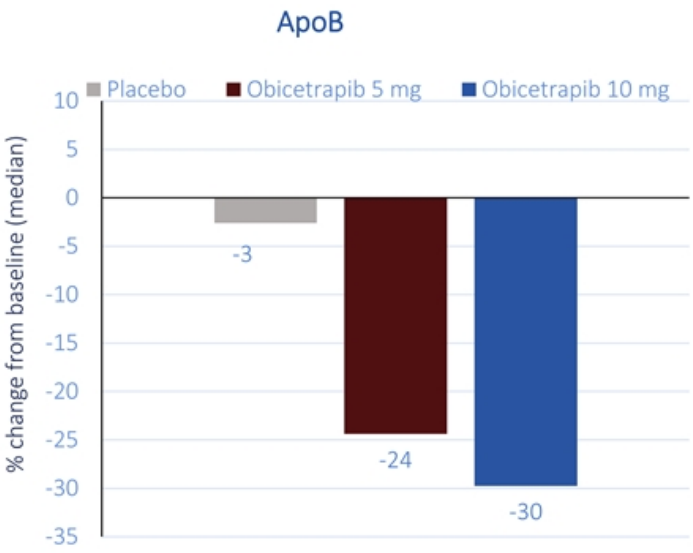
LDL-C reduction of >60% was observed in 20% of obicetrapib 5mg patients

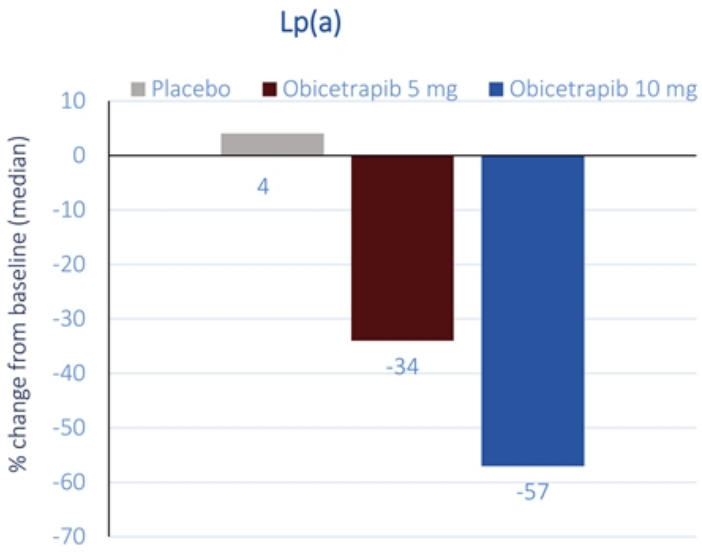
LDL-C reduction of >60% was observed in 40% of obicetrapib 10mg patients



Note: Each bar represents one subject in the trial. Placebo Obicetrapib 5mg Obicetrapib 10mg

ApoB & Lp(a) percent change from baseline





ROSE safety: AEs, SAEs and withdrawals overview

Positive safety profile observed, and no drop-outs due to AEs

	Placebo (N=40)	Obicetrapib 5mg (N=40)	Obicetrapib 10mg (N=40)
AEs (%)			
AEs, total	19 (47.5)	15 (37.5)	8 (20.0)
AEs, related	4 (10.0)	2 (5.0)	1 (2.5)
AEs, severe	1 (2.5)	0	0
SAEs			
SAEs, total	2 (5.0)	0	0
SAEs, related	0	0	0
Deaths	0	0	0
Withdrawals study / medication			
TEAEs leading to discontinuation of study drug	1 (2.5)	0	0
TESAEs leading to discontinuation of study	0	0	0

REVEAL supports translation from absolute LDL reduction to MACE benefit

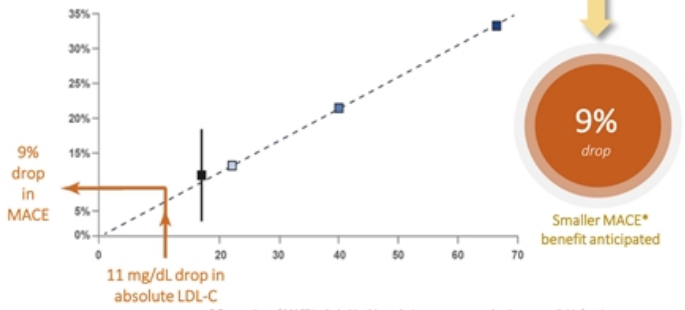
EXPERIENCE: *REVEAL (anacetrapib)*

61 mg/dL × 17% = 11 mg/dL

Low baseline

Drug showed modest LDL-lowering %

Results in SMALL absolute LDL reduction



9% *drop*

Smaller MACE* benefit anticipated

* Composites of MACE included in this analysis were coronary death, myocardial infarction or coronary revascularization.
Source: The HPS3/TIMI55–REVEAL Collaborative Group. N Engl J Med 2017; 377:1217-1227
Cholesterol Treatment Trialists Collaboration. Lancet. 2010 376:1670-81.

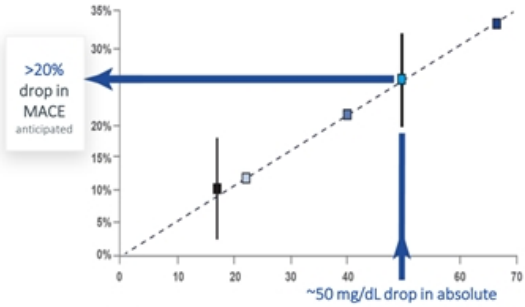
PREDICTION: *PREVAIL (obicetrapib)*

~100 mg/dL(1) × 51% *anticipated* = ~50 mg/dL

Enrich for higher baseline

Stronger expected LDL-lowering %

Expected to result in more absolute LDL reduction



>20% *drop anticipated*

Bigger expected MACE** benefit

Note: Actual results may differ from expectations.
Source: Cholesterol Treatment Trialists Collaboration. Lancet. 2010 376:1670-81 Circulation. 2021;144:e564–e593 17065: Obicetrapib Lowers LDL-C in Patients Taking High Intensity Statins.
(1) Represents estimated average baseline LDL to be enrolled, not entry criteria.
** MACE includes cardiovascular death, myocardial infarction, stroke and non-elective coronary revascularization in adults.

Conclusions

- Driving statins to their highest dose as monotherapy has many downsides, of which failed goal attainment is the worst
- Emerging data support the rationale for the option of combining early with ezetimibe, bempedoic acid, monoclonals against PCSK9 or inclisiran makes the most sense
- Novel agents, such as the CETP-inhibitor obicetrapib and the oral PCSK9 modulators, have shown promising results in clinical trails