

Welcome to Investor Day

August 5, 2026



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Forward Looking Statements

This presentation and the accompanying oral statements made in connection herewith (collectively, this “Presentation”) contains forward-looking statements within the meaning of the United States Private Securities Litigation Reform Act of 1995 and are subject to the “safe harbor” provisions created thereunder. All statements that are not historical facts are hereby identified as forwarding-looking statements for this purposes and include, among others, statements by NewAmsterdam Pharma Company N.V. (“NewAmsterdam”, the “Company”, “we” or “our”) regarding: estimates and forecasts of financial and performance metrics and projections of market opportunity; the Company’s business and strategic plans; 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 the Company’s product candidate; the timing for enrolling patients; the Company’s intention to conduct the PREVAIL interim analysis and expectations regarding the timing of the interim analysis, when the result of the interim analysis may be announced and the potential of PREVAIL to be stopped early for efficacy as a result of the interim analysis; the design and predictive ability of clinical trials; potential predictors of MACE benefit; potential drivers of the blinded event rates observed in PREVAIL; the timing and forums for announcing data; the size and growth potential of the markets for the Company’s product candidate; the Company’s ability to achieve the broad degree of physician adoption and use and market acceptance necessary for commercial success; the therapeutic and curative potential of the Company’s product candidate; financing and other business milestones; the Company’s expected cash runway; and the Company’s plans for commercialization.

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Agenda

Date: August 5, 2026

Corporate Update: Michael Davidson, MD, CEO, NewAmsterdam

- Introduction
 - General Corporate Update
-

Science Update: John Kastelein, MD, PhD, FESC, CSO, NewAmsterdam

- Biomarker predictors of MACE and REVEAL
 - Potential Benefits of HDL
 - REMBRANDT
 - RUBENS
 - CETP and Alzheimer's Disease
-

PREVAIL Update: Michael Davidson, MD, CEO, NewAmsterdam

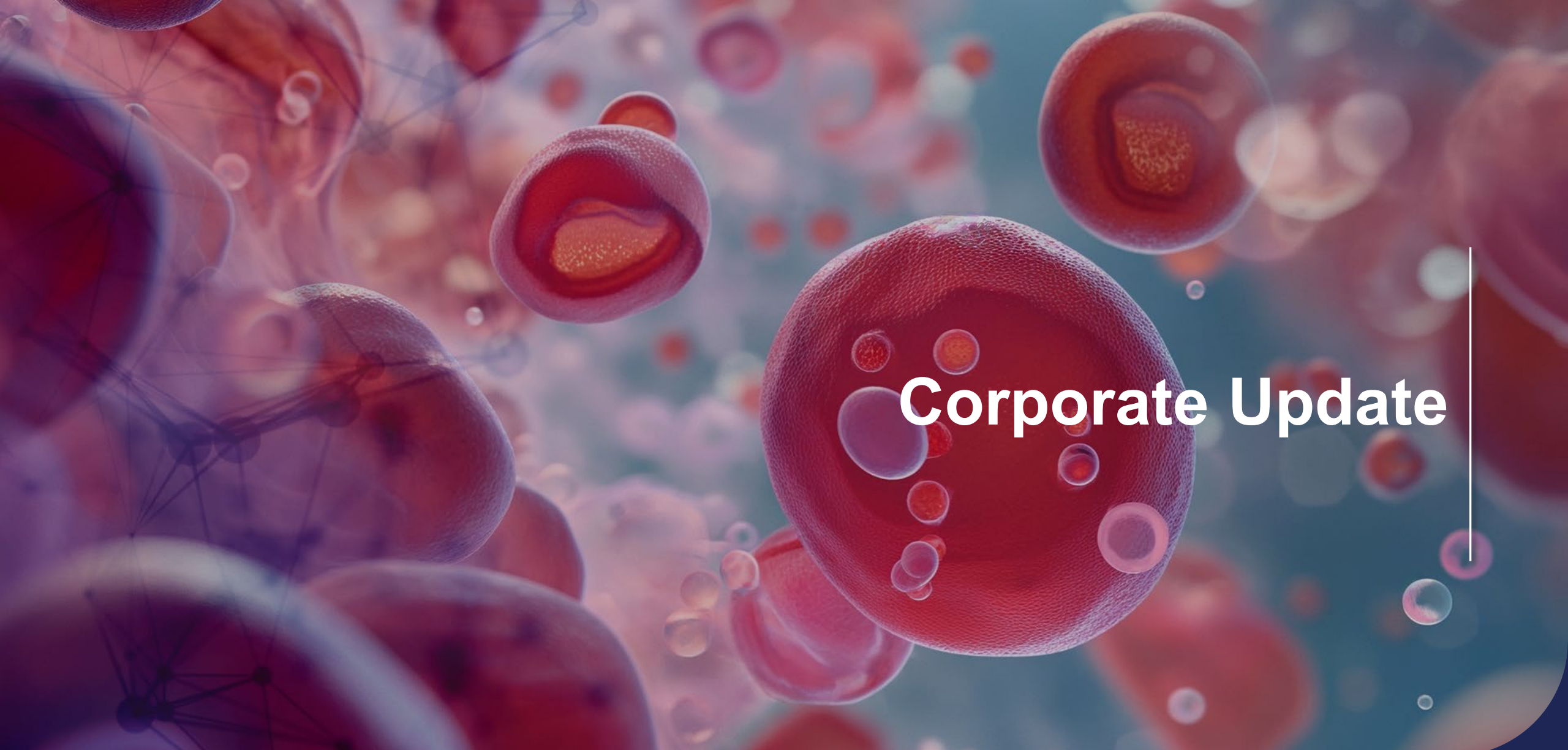
- PREVAIL: Improving Probability of Success
 - Alzheimer's Update
-

Commercial Update: BJ Jones, CCO, NewAmsterdam

- Market Momentum
 - Product Differentiation
 - US Launch Readiness
 - Prelaunch Activities
 - European Launch overview
-

Closing: Michael Davidson, MD, CEO, NewAmsterdam

Q&A



Corporate Update

Our Mission is Clear and Simple



Our mission is to build **a new standard of care** for people living with cardiometabolic disease by challenging convention with courage, translating deep biological insight into meaningful patient impact, and delivering with rigor, precision and purpose. This is how NewAmsterdam is **built to go beyond**.

Moving Forward, Still Looking Ahead



“

I got a wake-up call early in life that I needed to take better care of myself. My family history didn't do me any favors, so, I try to stay as diligent as possible to live a long, healthy life. I'm grateful to my wife, Mary, and my doctors who have helped me change the trajectory of my life. High cholesterol doesn't simply go away, but with the support of my care team and a willingness to consider medication options that may help me, I feel motivated to keep moving forward.

Frank L.

Committed to Prevention, Open to What's to Come

AGE 76 • MALE • CVD, DIABETES &
HYPERTENSION • FHx CVD &
DIABETES
MEDICATIONS: STATIN, EZE •
SWIMMER AND BIKER •
RETIRED ENTERTAINER



The NewAmsterdam Value Proposition



Clinical Expertise: Data driven clinical development increases probability of success



Differentiated Target Product Profile: Phase 3 data demonstrates obicetrapib's potential impact on multiple CVD risk factors



Unmet Need: Significant need for a potent, convenient, well-tolerated low-dose oral therapy



Experienced Leadership and Team: Expanding team comprised of accomplished industry executives



KOL Support: Physician community aware of unmet need and eager to leverage obicetrapib's differentiated profile to achieve desired clinical benefit, if approved



CMC Expertise and Manufacturing Capacity: Preparing to meet anticipated demand for obicetrapib and the fixed dose combination, creating redundancy, expanding capacity and building inventory



Commercial Excellence: Growing team with track record of blockbuster launch success and innovative go-to-market model



Strong Balance Sheet: Cash runway expected to be sufficient to support U.S. commercial launch

Previous Years Were a Time of Groundwork, Goals and Growth



Completed and announced results for BROOKLYN, BROADWAY, and TANDEM Phase 3 studies



Building world-class commercial and MSL functions



Doubled in size (>100) with new hires and offices in Amsterdam, NL, Miami, FL, and Philadelphia metro area



Secured funding to support US commercial launch, if approved, with cash at end of 2Q 2026 of ~\$678M*

2024

2025

2026



Composition of matter IP granted



Announced topline results for BROOKLYN Phase 3 trial



Announced topline results for TANDEM Phase 3 trial



Announced topline results for BROADWAY Phase 3 trial



Announced Alzheimer's topline



Full AD data was presented at AAIC on July 30



EMA submission



CHMP Positive Opinion



European Approvals

* Includes cash and cash equivalents and marketable securities

Lipid Lowering Market Primed for Commercial Success

Observations ¹⁻⁴		Key Takeaways	
1		We have seen LLT market growth each year for the last 5 years	Very large opportunity – even small amounts of penetration can result in significant financial success
2		Non-statin growth continues in the high double digits	Shift towards more aggressive treatment using alternatives / adding to statins
3		Guidelines have shifted to recommend more intense and earlier intervention not only with statins, but with ezetimibe, PCSK9's and bempedoic acid	Governing bodies and KOL's are prioritizing the importance of treating to goal
4		Access to PCSK9's has been increasing, and utilization controls have been decreasing	Payers are willing to cover medications in LLT space if priced appropriately
5		PCSK9 growth continues to accelerate 10 years post launch with 45%+ growth in net sales in each of the last 2 years	Commercial success is being demonstrated and is accelerating
6		FDA labeling changes continue to support broad access and earlier treatment	Labels are less restrictive and support broad usage

1. Based on third party prescription data on file

2. Grundy SM, et al. J Am Coll Cardiol. 2019;73(24):3168-3209.

3. Lloyd-Jones DM, et al. J Am Coll Cardiol. 2022;80(14):1366-1418

4. 2025 ACC/AHA/ACEP/NAEMSP/SCAI Guideline for the Management of Patients With Acute Coronary Syndromes: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines | Circulation

Positive CHMP Opinion Supports Potential Approval in Europe

- Committee for Medicinal Products for Human Use ("CHMP") of the European Medicines Agency ("EMA") adopted positive opinion recommending marketing authorization for **Ubeslo® (obicetrapib 10 mg monotherapy)** and **Evlarco® (10 mg obicetrapib plus 10 mg ezetimibe FDC)** for patients with primary hypercholesterolemia, both HeFH and non-familial or mixed dyslipidemia
- Final decision by European Commission ("EC") has authority to grant centralized marketing authorization applicable across all **27 EU member states**, and **Iceland, Liechtenstein and Norway**



Positive CHMP opinion received in July 2026 based on robust data from the Phase 3 BROADWAY, BROOKLYN and TANDEM trials



European Commission decision expected in 2H26



Partnership with Menarini provides commercial and regulatory infrastructure in EU → under licensing agreement Menarini holds rights for obicetrapib in Europe

NewAmsterdam entitled to tiered double-digit % royalties on net sales and up to an additional €833 million with achievement of various milestones

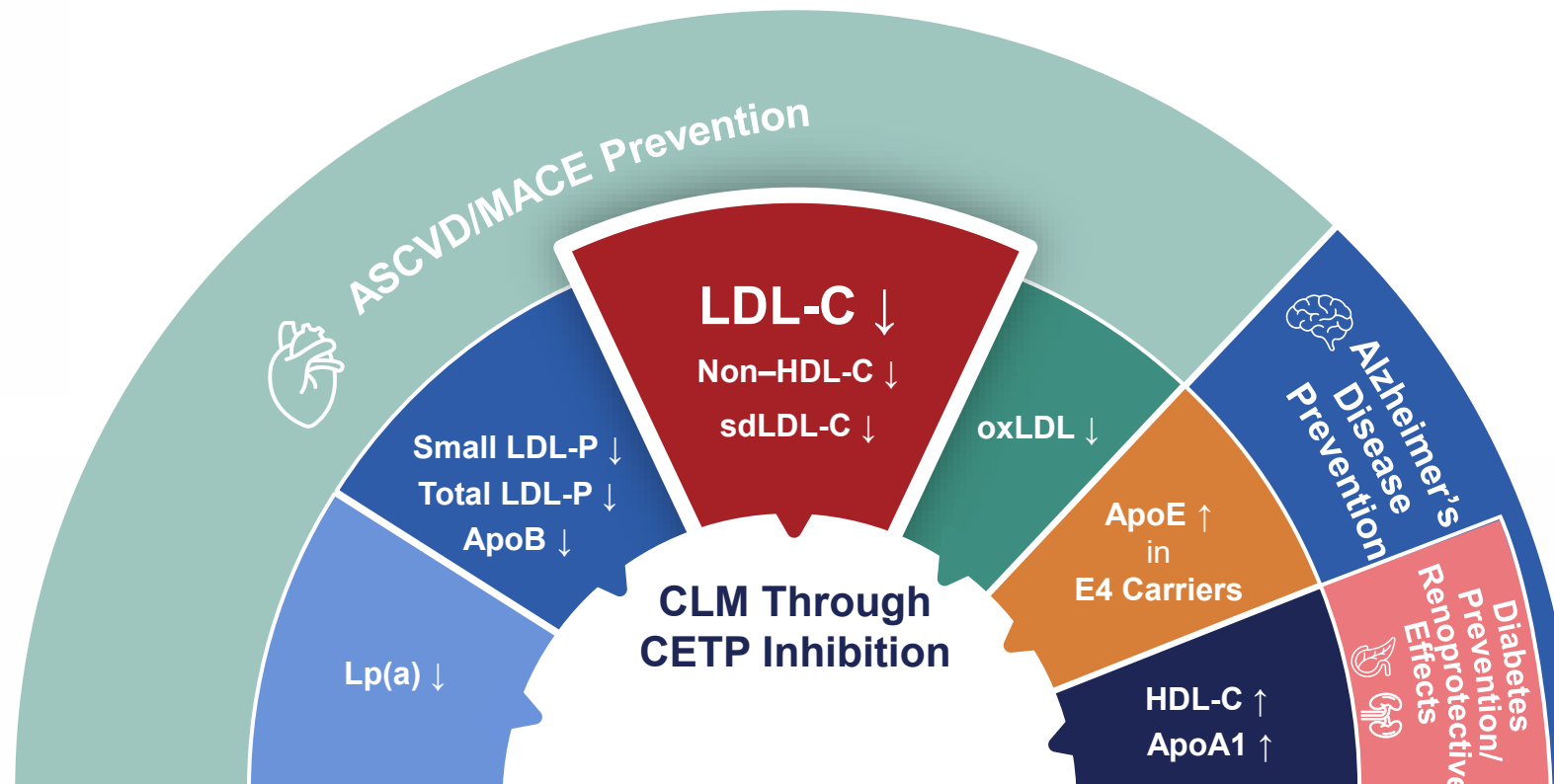


Science Update

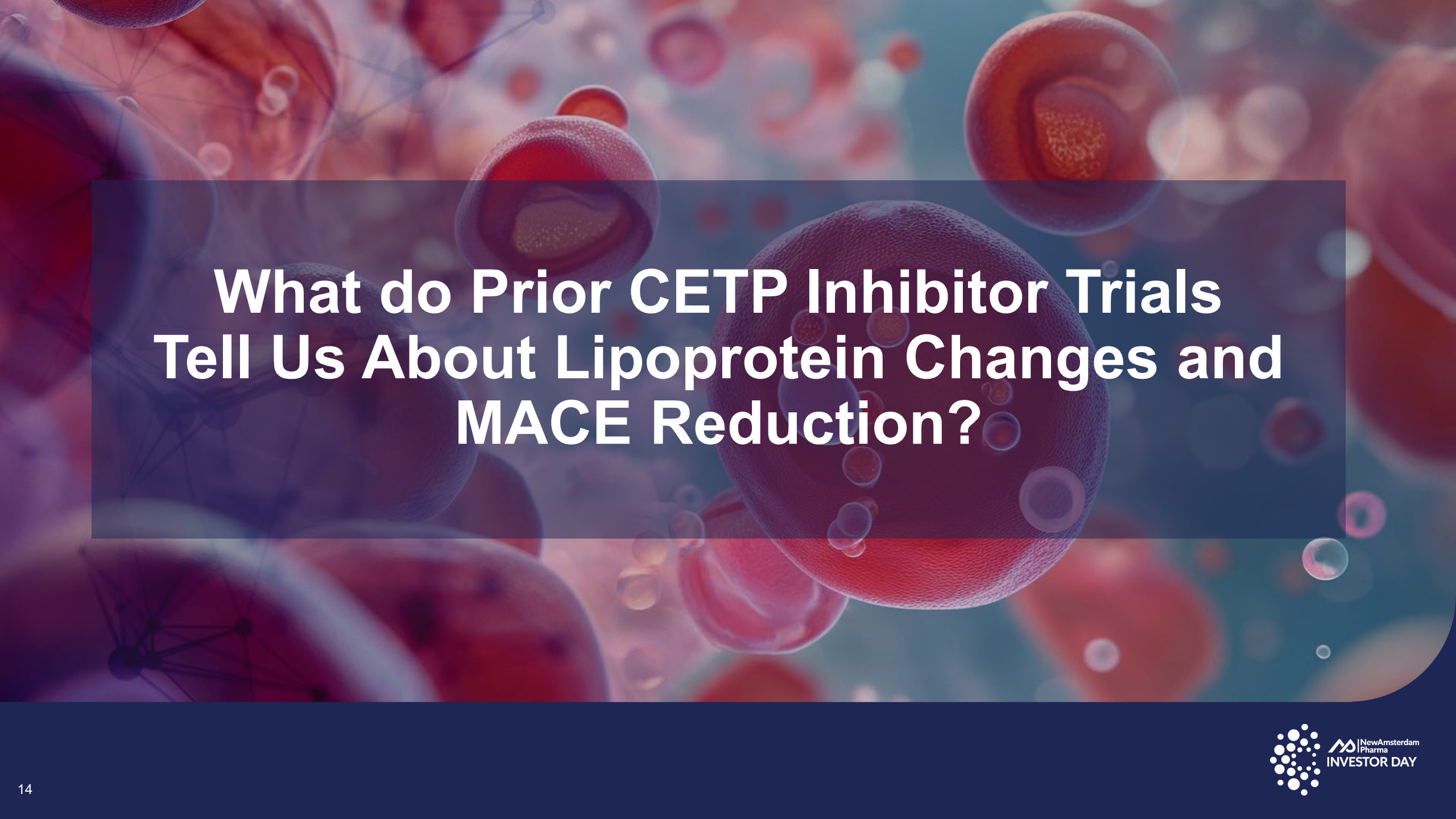


Observed Effects of Obicetrapib on MACE Biomarkers

Obicetrapib Delivers Comprehensive Lipid Modulation Through CETP Inhibition



ApoA1, apolipoprotein A1; ApoE, apolipoprotein E; ASCVD, atherosclerotic cardiovascular disease; CETP, cholesterol ester transfer protein; CVD, cardiovascular disease; LDL-P, low-density lipoprotein particle; MACE, major adverse cardiovascular events; oxLDL, oxidized low-density lipoprotein; sdLDL-C, small dense low-density lipoprotein cholesterol.



What do Prior CETP Inhibitor Trials Tell Us About Lipoprotein Changes and MACE Reduction?

Limitations of Prior CETP Inhibitor CVOTs

TORCETRAPIB¹

Suffered from drug-specific toxicity issue (Pfizer)

DALCETRAPIB²

Drug showed no LDL-lowering efficacy (Roche)

EVACETRAPIB³

Overall mortality benefit ($P = .04$) – but CVOT was too short to demonstrate MACE benefit (Lilly)

ANACETRAPIB⁴

Meaningful MACE benefit observed - but drug accumulated in fat tissue (Merck)

SAFETY

OFF-TARGET TOXICITY, INCREASED BLOOD PRESSURE, ALDOSTERONE (seen early in Phase 2)

Safe & well-tolerated

Safe & well-tolerated

Safe & well-tolerated

Strong safety profile across ~59k patients

LDL-LOWERING POTENCY

NO LDL-LOWERING

~40% CETP inhibition at CVOT dose

Modest LDL-lowering (~17-36%)

~80% CETP inhibition at CVOT dose

Modest LDL-lowering (17%)

~80% CETP inhibition at CVOT dose

We believe that all prior CETPi were developed with a **misguided focus on HDL increase** (rather than LDL decrease) as the primary MoA for CVD risk reduction, leading to **inappropriate compound selection or inappropriate CVOT design**.

CVOT DESIGN (DURATION & BASELINE LDL)

INSUFFICIENT TRIAL DURATION (only 2 years)

Sufficient duration (4.1 years, with 6.3 year follow up)

Baseline LDL too low (60 mg/dL)

COMMERCIAL VIABILITY

COMMERCIALLY UNVIAABLE
High lipophilicity and fat tissue accumulation led to 4+ year half-life

OBICETRAPIB⁵



- ✓ Tolerability profile observed in >4000 patients through Phase 3
- ✓ No concerns seen in biomarker safety data, including blood pressure-associated biomarkers

- ✓ 35-40% LDL lowering observed in phase 3
- ✓ ~50% LDL-lowering observed in fdc phase 3

~97% target coverage

- ✓ Longer trial duration (4 yrs) +
- ✓ High baseline LDL (100 mg/dL)⁽¹⁾

- ✓ Favorable PK/PD profile
- ✓ No accumulation in fat tissue observed

Note: The above trials and data do not represent head-to-head comparisons. Obicetrapib has not been approved for marketing by any regulatory authority. Represents estimated average baseline LDL to be enrolled, not entry criteria. Evacetrapib measured using direct LDL-C assay.

Sources: 1. Barter PJ, et al. N Engl J Med 2007;357:2109-2122; 2. Schwartz GG, et al. N Engl J Med 2012;367:2089-2099; 3. Lincoff AM, et al. N Engl J Med 2017;376:1933-1942; 4. The HPS3/TIMI55-REVEAL Collaborative Group. N Engl J Med 2017;377:1217-1227; 5. Data on file

REVEAL: The Largest CETP Inhibitor Outcomes Trial

30,449¹

patients with ASCVD

Largest CVOT ever conducted¹

(1 tertile is ~PREVAIL-sized)

17%¹

LDL-C reduction

With anacetrapib 100 mg/day on top
of intensive statin therapy led to
9% MACE reduction¹

75-80%

CETP inhibition

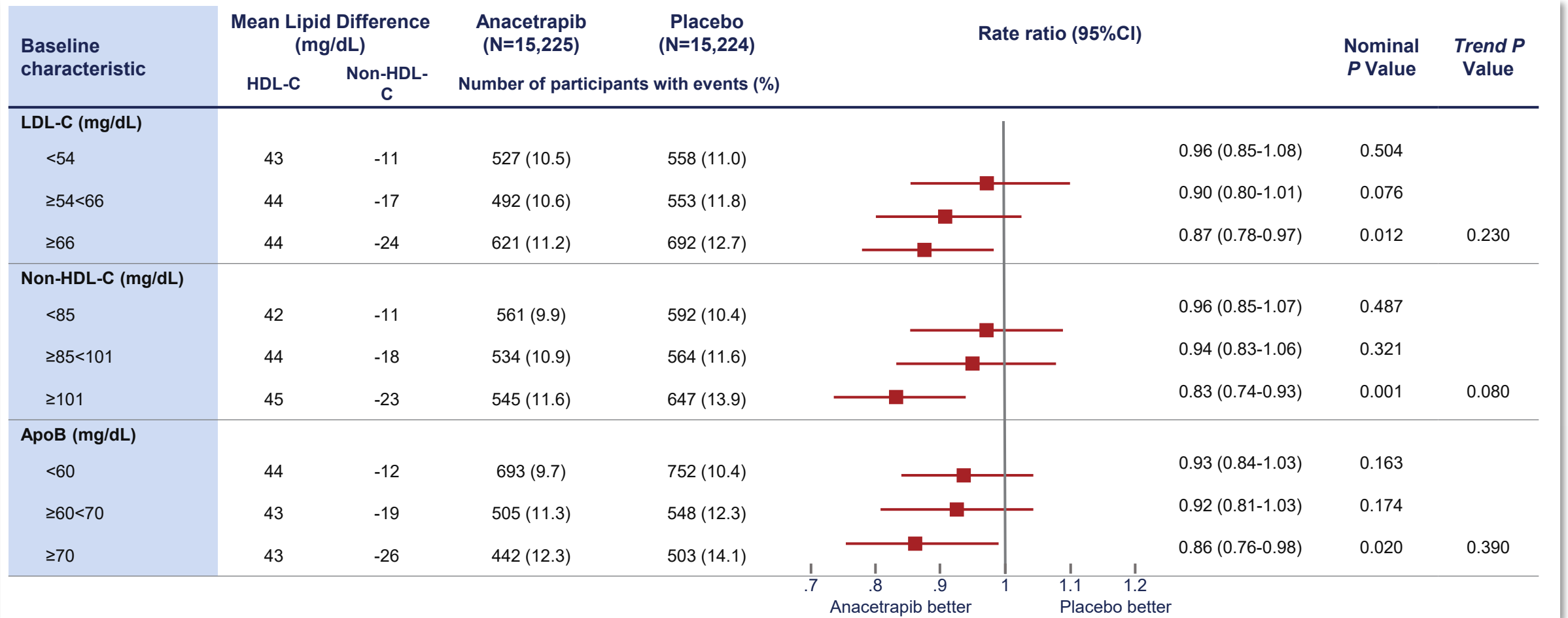
with 100 mg dose²
(Obicetrapib demonstrates 98% inhibition)

Although anacetrapib is a much weaker CETP inhibitor with only modest LDL-C lowering, REVEAL still achieved a statistically significant reduction in MACE validating CETP inhibition on top of statins as a potential route to cardiovascular risk reduction¹

ASCVD, atherosclerotic cardiovascular disease; CETP, cholesteryl ester transfer protein; CVOT, cardiovascular outcomes trial; LDL-C, low-density lipoprotein cholesterol; MACE, major adverse coronary events.

1. HPS3/TIMI55–REVEAL Collaborative Group. *N Engl J Med*. 2017;377(13):1217-1227. 2. Krishna R, et al. *Br J Clin Pharmacol*. 2009;68(4):535-545.

What Did We Observe In REVEAL?



Patients with the highest baseline non-HDL-C showed a ~17% reduction in rate of MACE

ApoB, apolipoprotein B; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol.

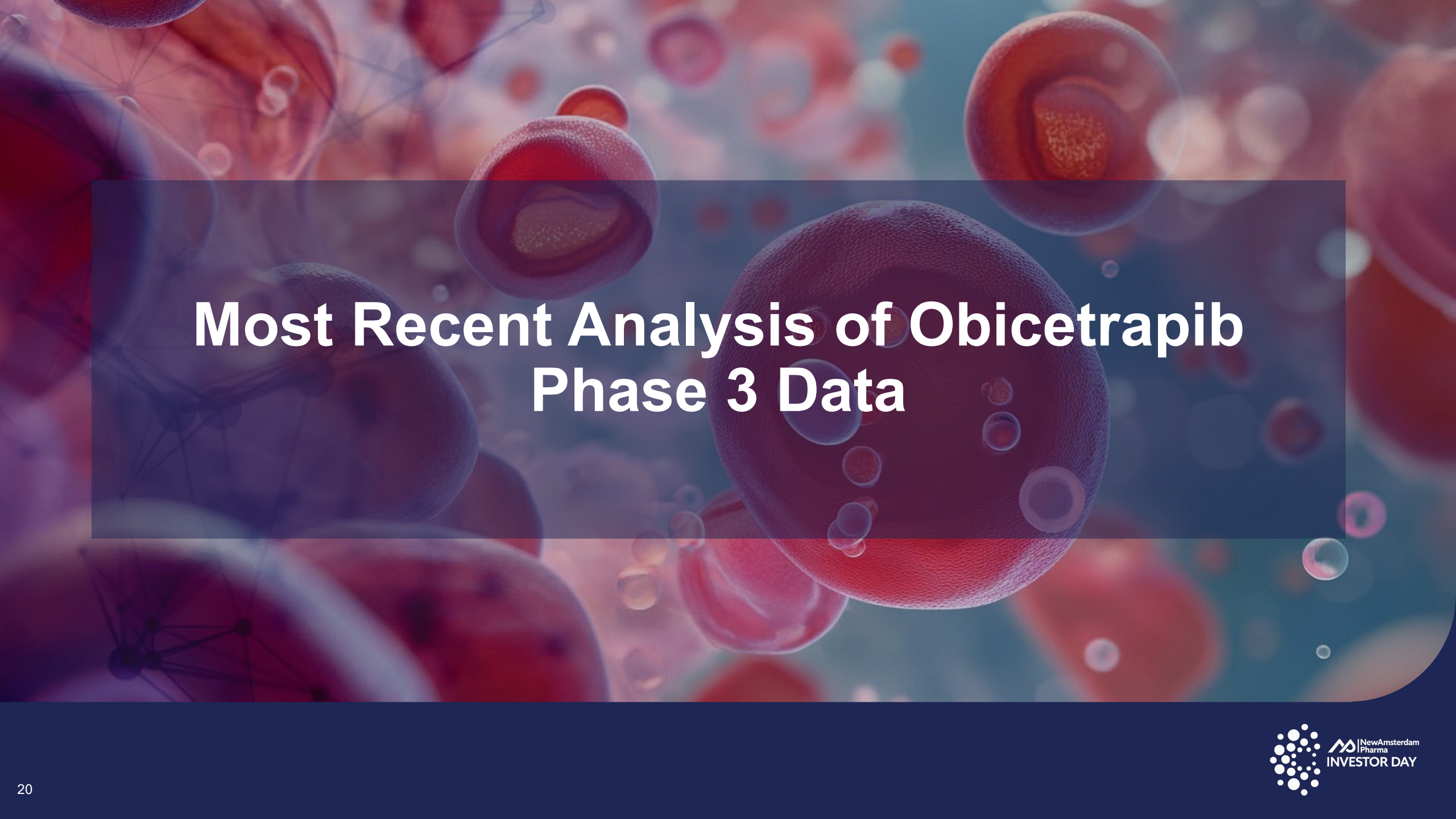
*Major coronary event was defined as a composite of coronary death, myocardial infarction, or coronary revascularization.

HPS3/TIMI55-REVEAL Collaborative Group. *N Engl J Med.* 2017;377(13):1217-1227.

**In REVEAL, Modest CETP and LDL-C
Reduction in 10,000 Patients with Baseline
non-HDL in the Range of PREVAIL
Delivered a 17% reduction in MACE**

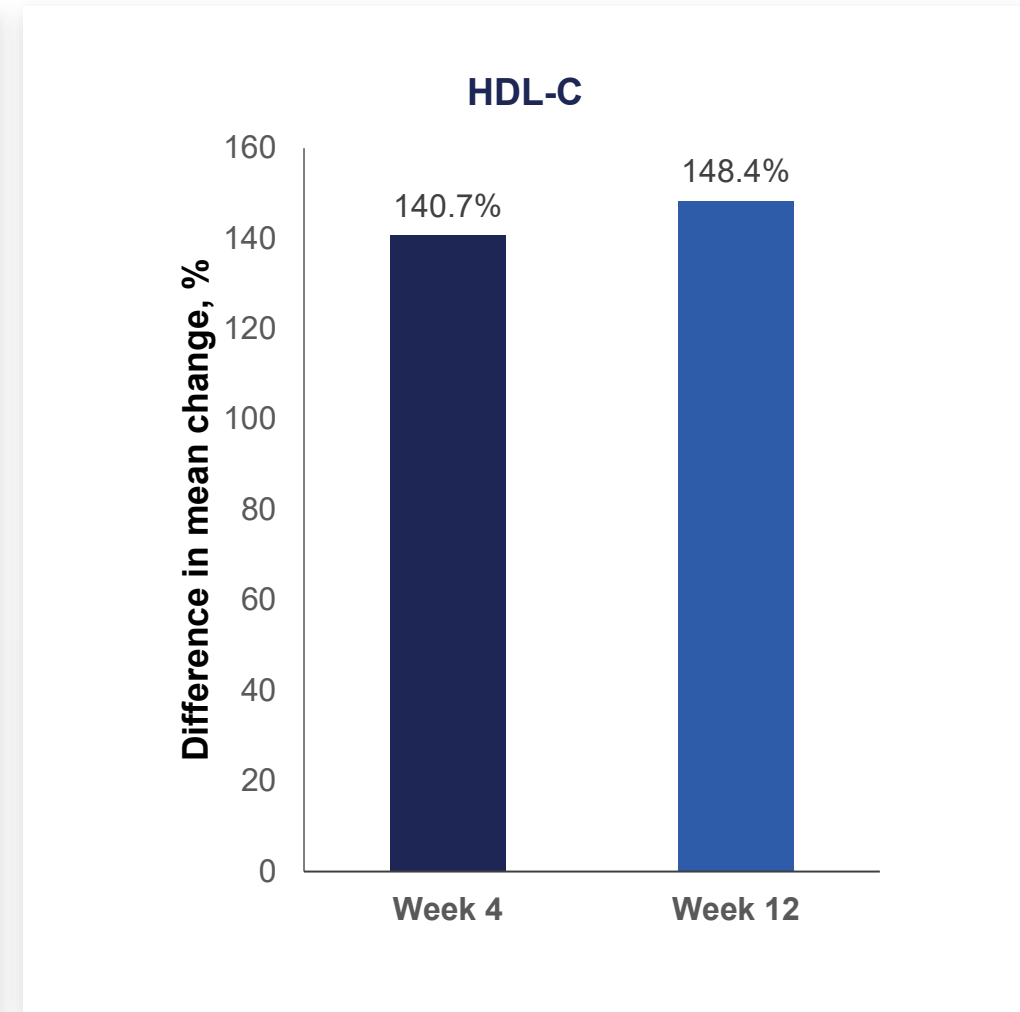
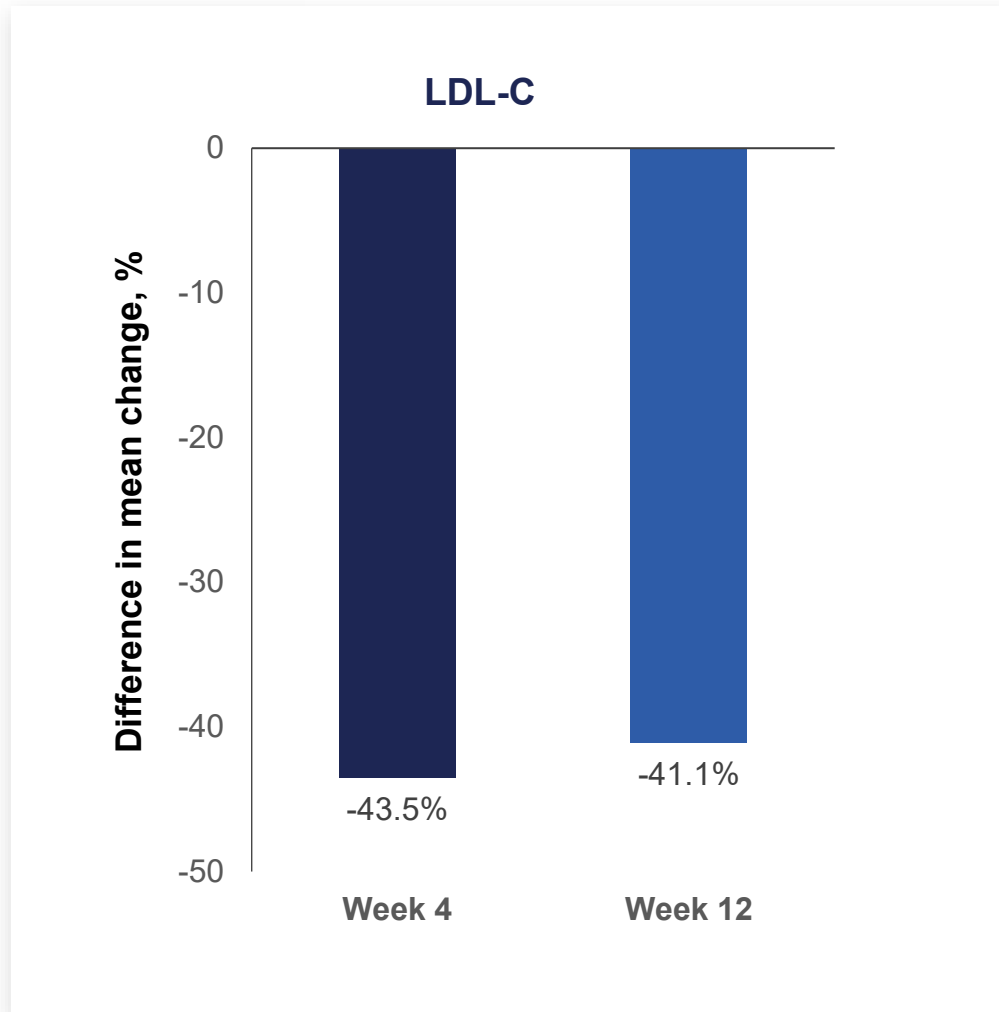


What Have We Observed with Obicetrapib and the FDC Compared to Anacetrapib?

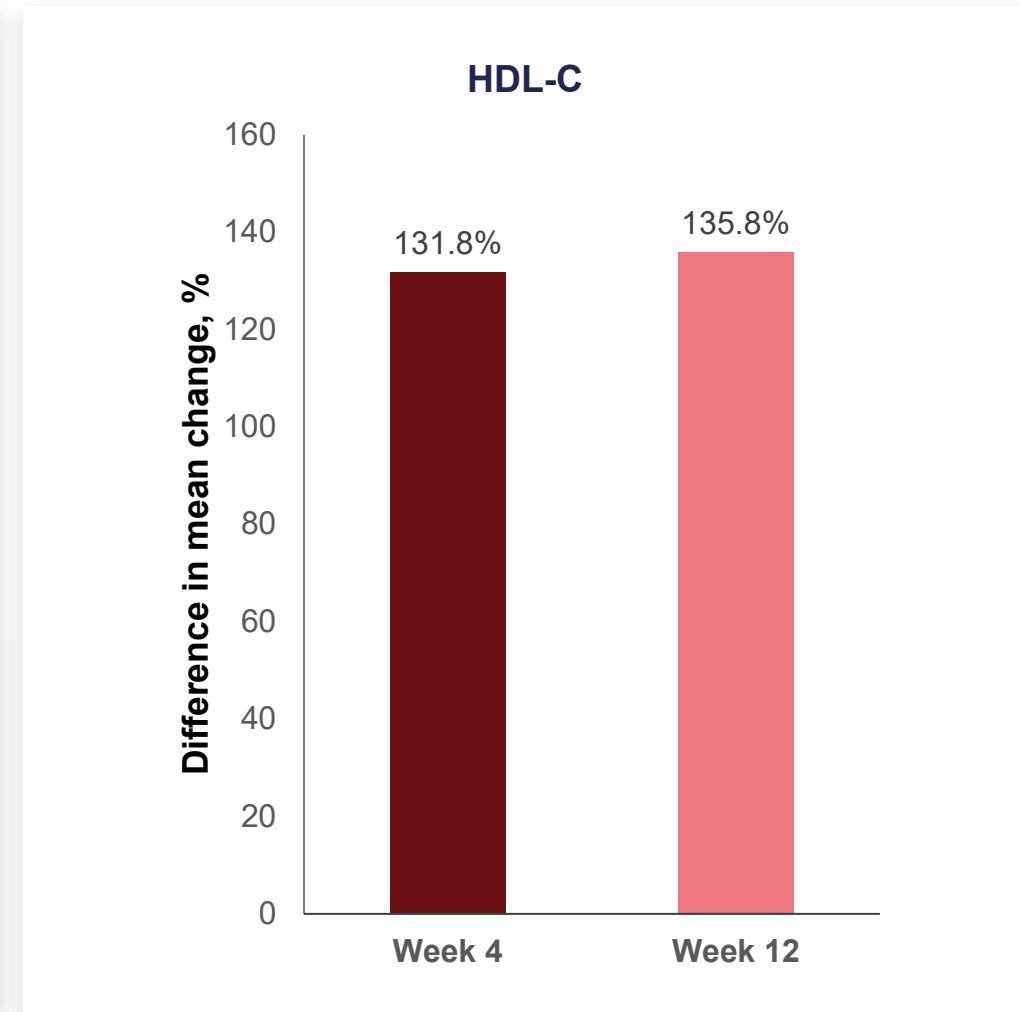
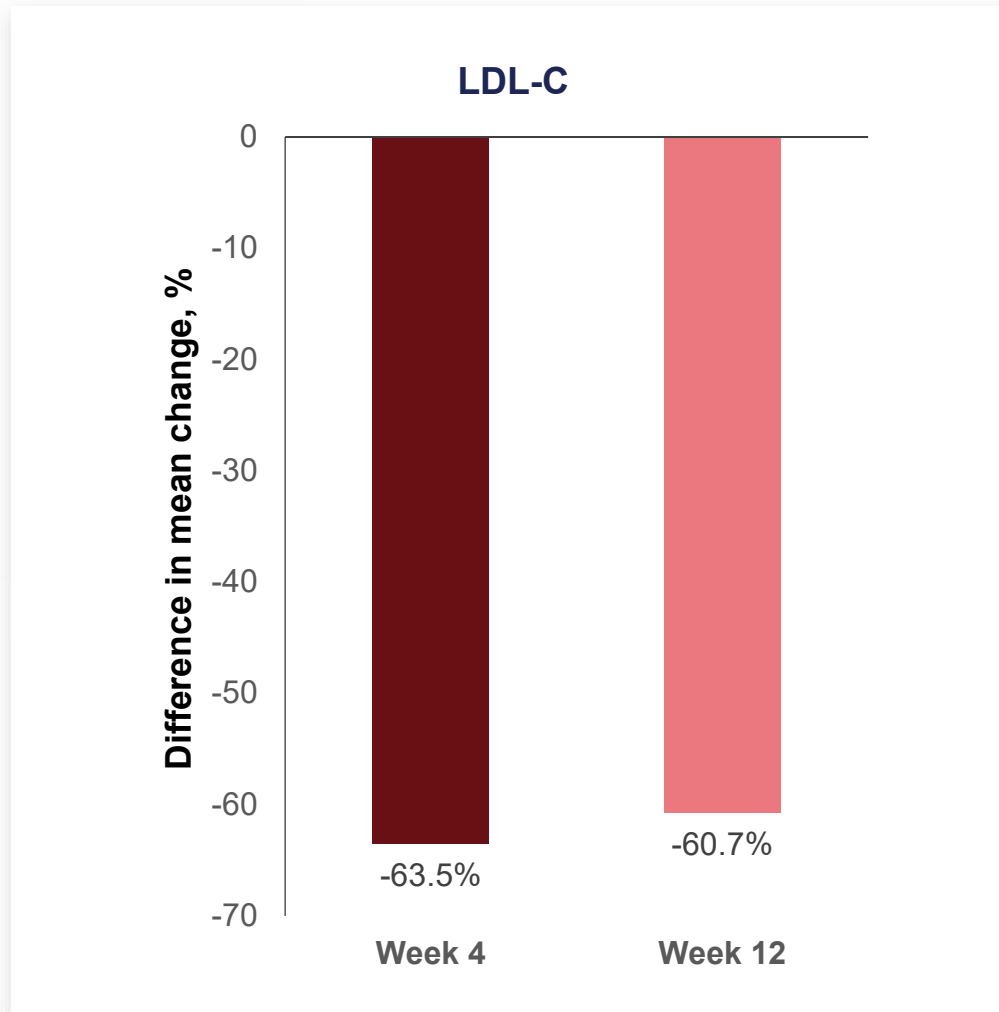


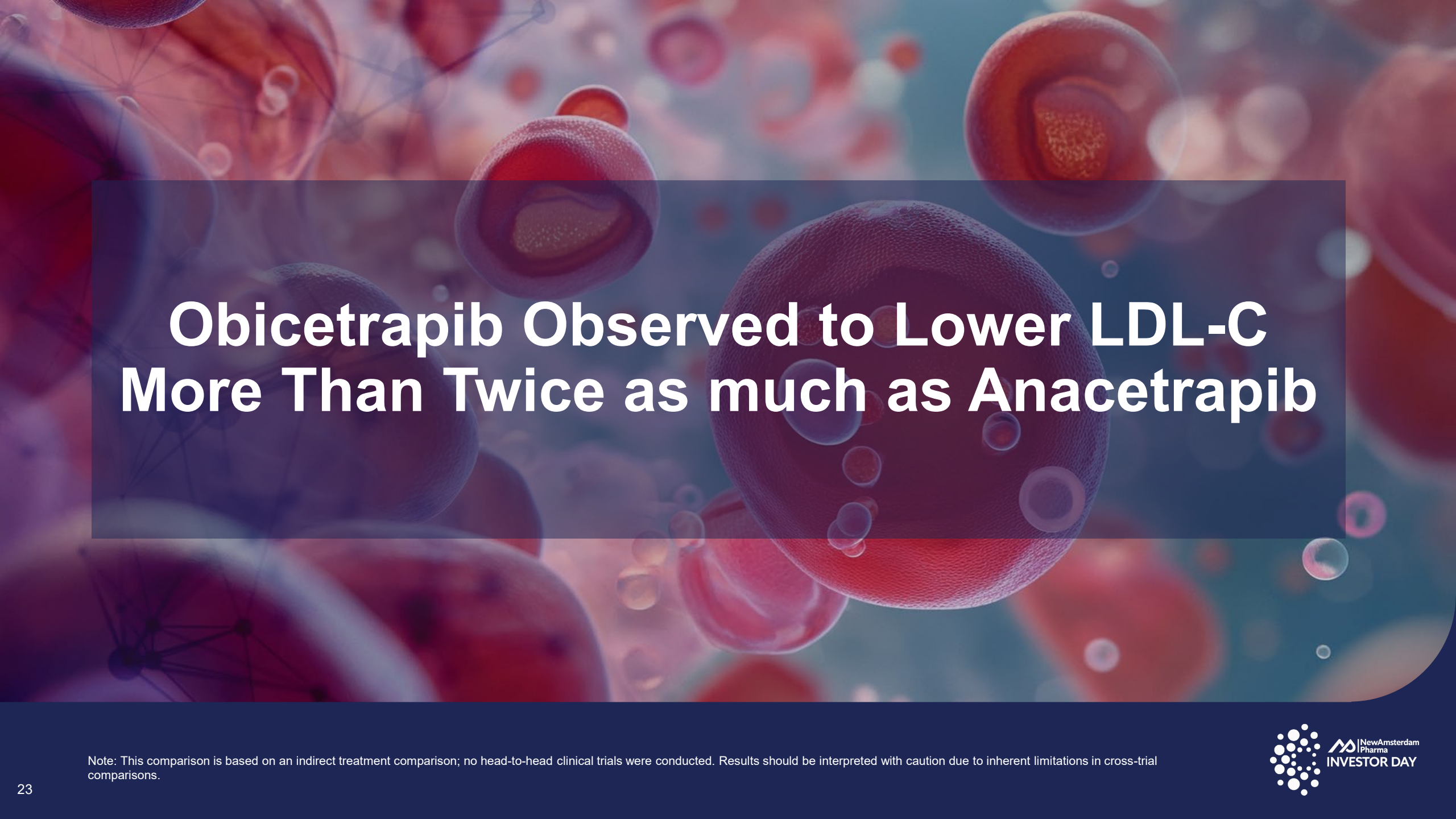
Most Recent Analysis of Obicetrapib Phase 3 Data

PK-Confirmed, Placebo Controlled, On-Treatment Effectiveness of Obicetrapib in Pooled Analysis of BROOKLYN, BROADWAY, and TANDEM



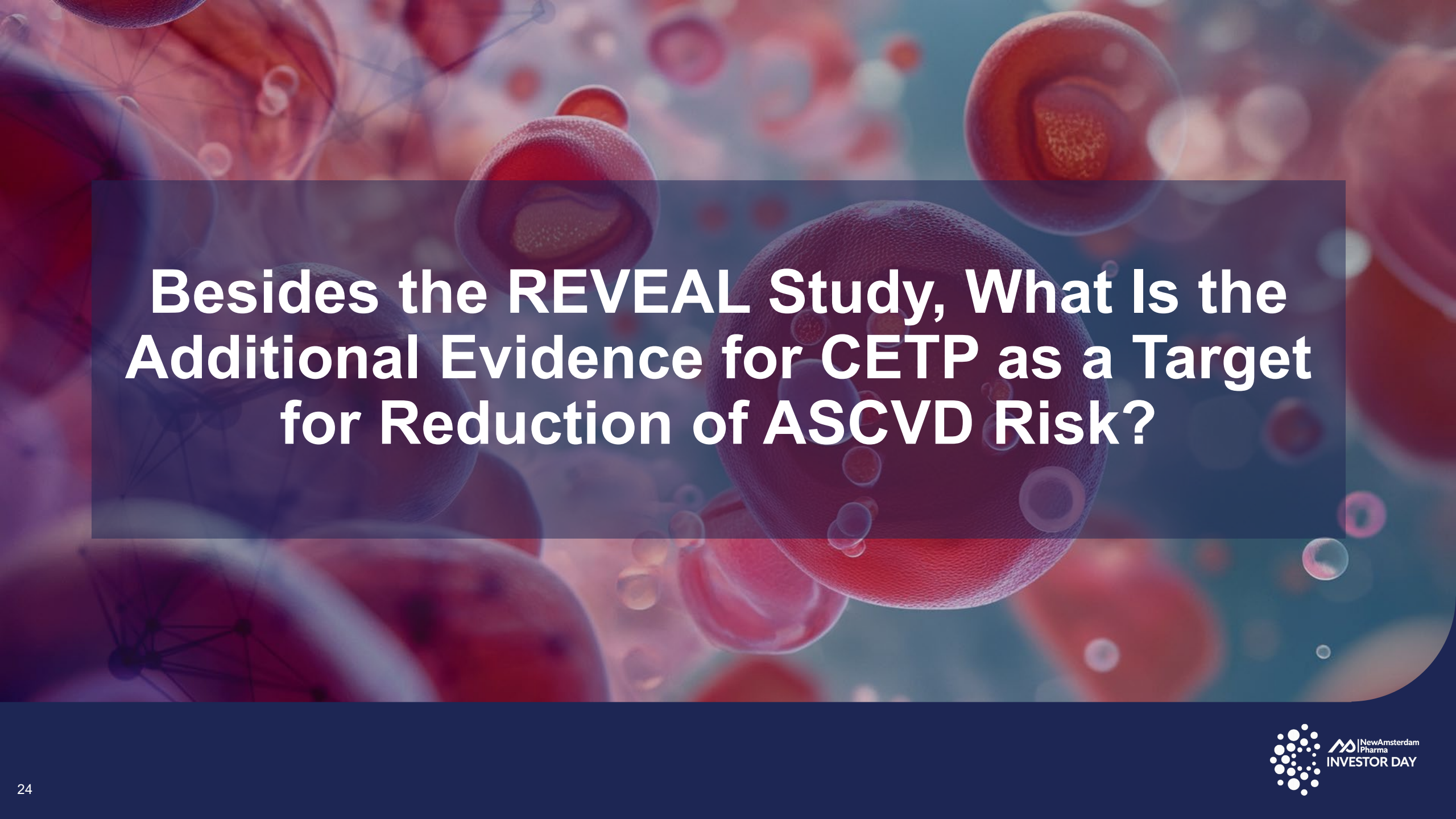
PK-Confirmed, Placebo Controlled, On-Treatment Effectiveness of the Obicetrapib-Ezetimibe Fixed-Dose Combination in Analysis of TANDEM





Obicetrapib Observed to Lower LDL-C More Than Twice as much as Anacetrapib

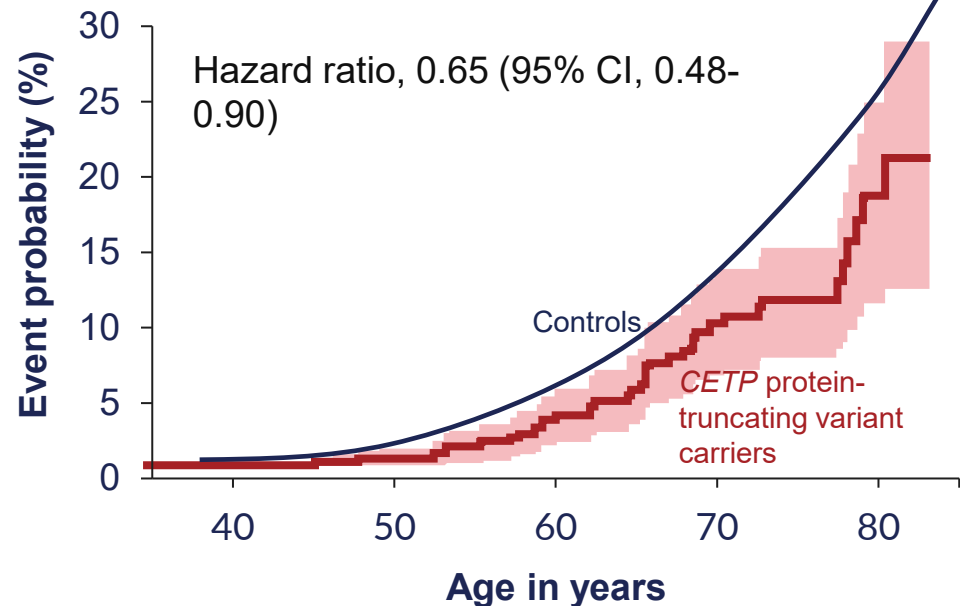
Note: This comparison is based on an indirect treatment comparison; no head-to-head clinical trials were conducted. Results should be interpreted with caution due to inherent limitations in cross-trial comparisons.



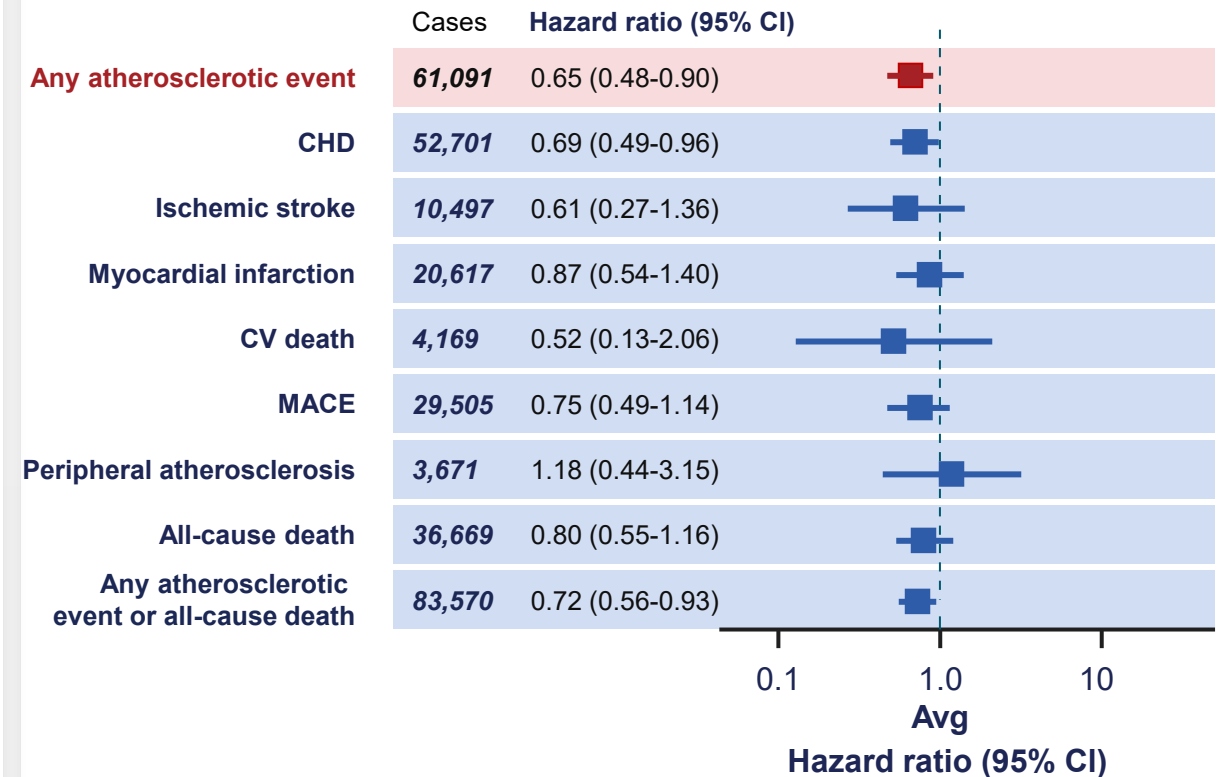
Besides the REVEAL Study, What Is the Additional Evidence for CETP as a Target for Reduction of ASCVD Risk?

In Press: Individuals with 50% Lower CETP Activity Show Lower Risk of CV Events

CETP protein-truncating variant carriers had a lower probability of cumulative CV events compared with controls*



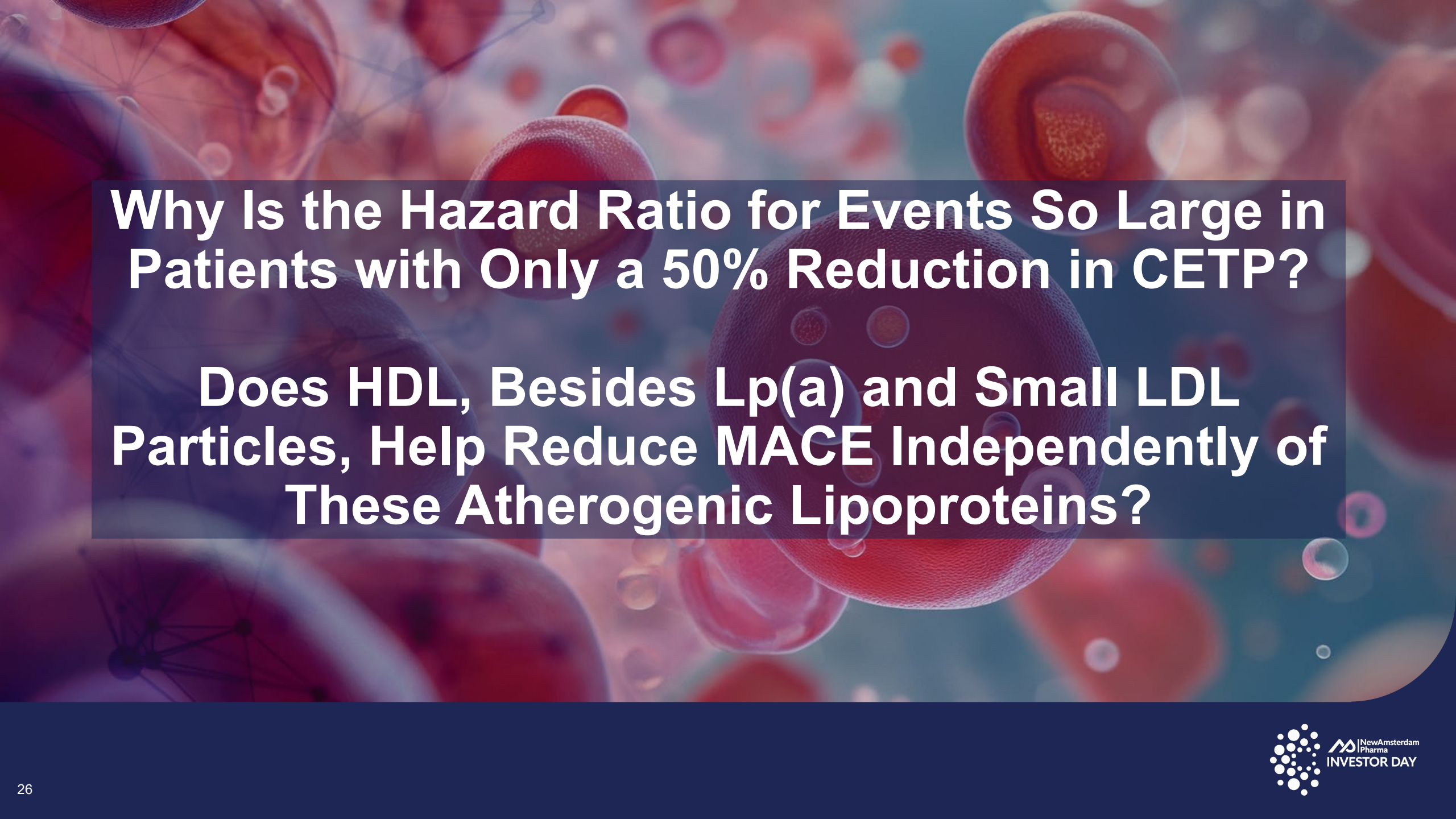
CETP protein-truncating variants were associated with lower risk of any atherosclerotic event†



*In a study of individuals from the UK Biobank, the effects of *CETP* protein-truncating variants on ASCVD risk were determined by analyzing ASCVD events in individuals with *CETP* protein-truncating variant carriers (n=385) and noncarriers (n=409,253). Shaded areas around each curve represent 95% confidence intervals.

†In this analysis, the primary endpoint was any atherosclerotic disease event. All other endpoints were considered secondary endpoints.

ASCVD, atherosclerotic cardiovascular disease; *CETP*, cholesteryl ester transfer protein; CHD, coronary heart disease; CI, confidence interval; CV, cardiovascular; MACE, major adverse cardiovascular events. Landfors F et al. *medRxiv*. 2024. 12.02.24318306



Why Is the Hazard Ratio for Events So Large in Patients with Only a 50% Reduction in CETP?

Does HDL, Besides Lp(a) and Small LDL Particles, Help Reduce MACE Independently of These Atherogenic Lipoproteins?



Let's Look at Genetic Background First: Are Genetics Supportive of Clinical Data?



When Did We Discover That Humans with Low CETP Activity Do Better Than The Rest of Us?

CETP Loss of Function and the Ashkenazi Jewish Longevity Project

Louise Levy, 'supercentenarian' subject of longevity study among Ashkenazi Jews, dies at 112

BY ANDREW SILOW-CARROLL JULY 28, 2023 3:22 PM



Louise Levy was born in 1910 and grew up in Cleveland and New York City; Levy often ascribed her longevity to a daily glass of red wine and a low-cholesterol diet.

The Low CETP Activity Gene Variant Is Associated with Exceptional Longevity and the Healthy Aging Phenotype

CETP I405V genotype and lipoprotein characteristic and plasma CETP levels in families with exceptional longevity vs control

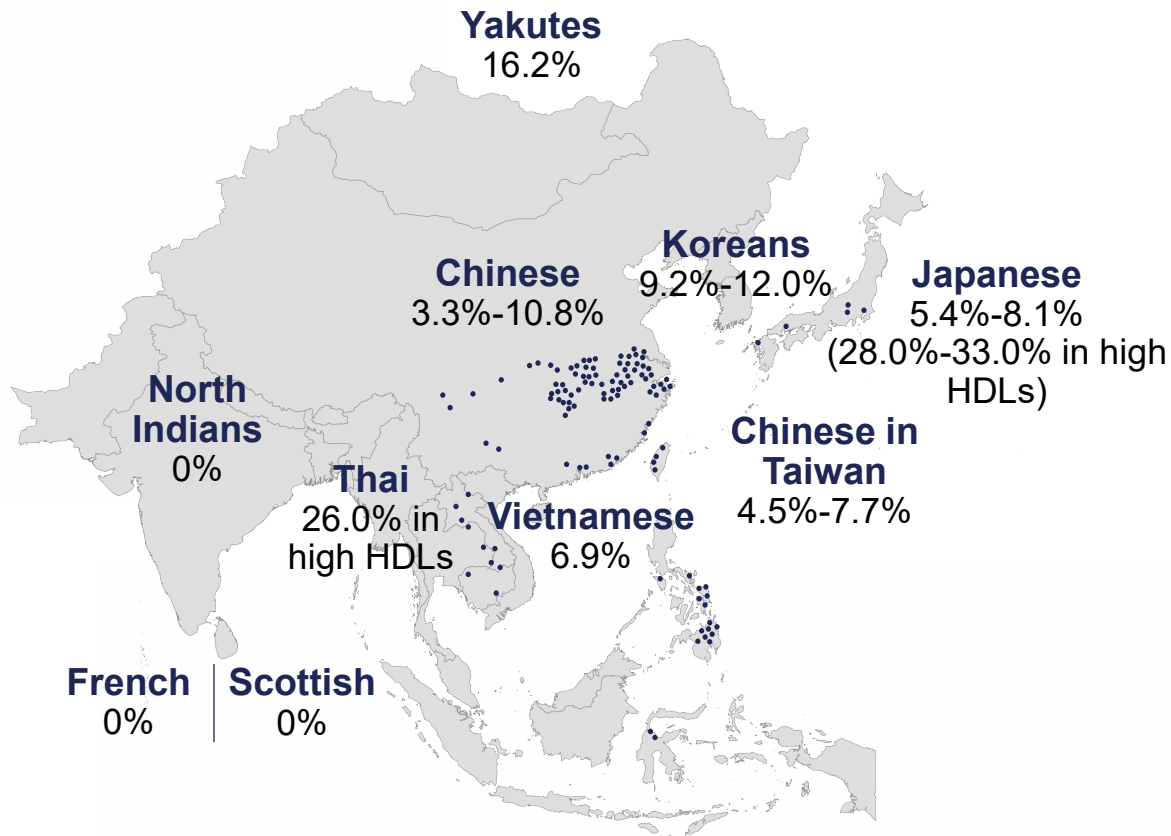
Variable	CETP I405V Genotype VV	CETP I405V Genotype II	P value (VV vs II Genotypes)
HDL			
Concentration, mg/dL	57	55	0.53
Large particle size, % of total	56	60	0.10
Particle size, nm	9.28	9.07	0.02
LDL			
Concentration, mg/dL	114	123	0.16
Large particle size, % of total	67	56	0.02
Particle size, nm	21.29	20.88	0.002
CETP concentration, mg/mL	1.65	1.99	<0.001

Individuals with exceptional longevity had significantly higher frequency of low CETP activity mutations



Where Do We Find Individuals with No CETP Activity at All and Do They Live Longer Too?

CETP Deficiency Resulting in High HDL-C Levels Is Common in Regions With Endemic Schistosomiasis and May Confer Protection



CETP deficiency,* resulting in high HDL-C levels, is common in populations from regions with endemic schistosomiasis

Egg embryonation of *Schistosoma japonicum* in the liver requires plasma HDL, and this process is impaired with CETP deficiency

CETP inhibition is hypothesized to be a viable treatment strategy in schistosomiasis

CETP, cholesteryl ester transfer protein; HDL, high-density lipoprotein; HDL-C, high-density lipoprotein cholesterol.
*D442G mutation prevalence listed as percentage in each ethnic general population.
Yokoyama S et al. *J Biomed Res.* 2015;29(3):176-188.



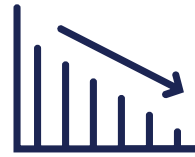
What Do These Individuals Look Like Besides Having Protection Against Parasites?

Characteristics of Individuals With CETP Deficiency



Lipid Profiles¹

- >100% increase in HDL
- 40% decreases in LDL-C and ApoB
- 40% decrease in Lp(a)
- No small LDL particles



Exceptional Longevity²

in individuals homozygous for the I405V allele (associated with lower CETP protein levels)



Lower ASCVD Risk³

CETP protein-truncating variant carriers have a lower probability of cumulative CV events



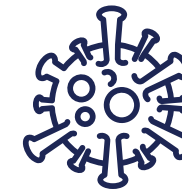
Lower AD Risk⁴

Individuals with 1 or 2 LoF CETP genes have decreased risk of dementia and AD



Lower Risk of AMD^{5,6}

- No AMD in patients with CETP deficiency

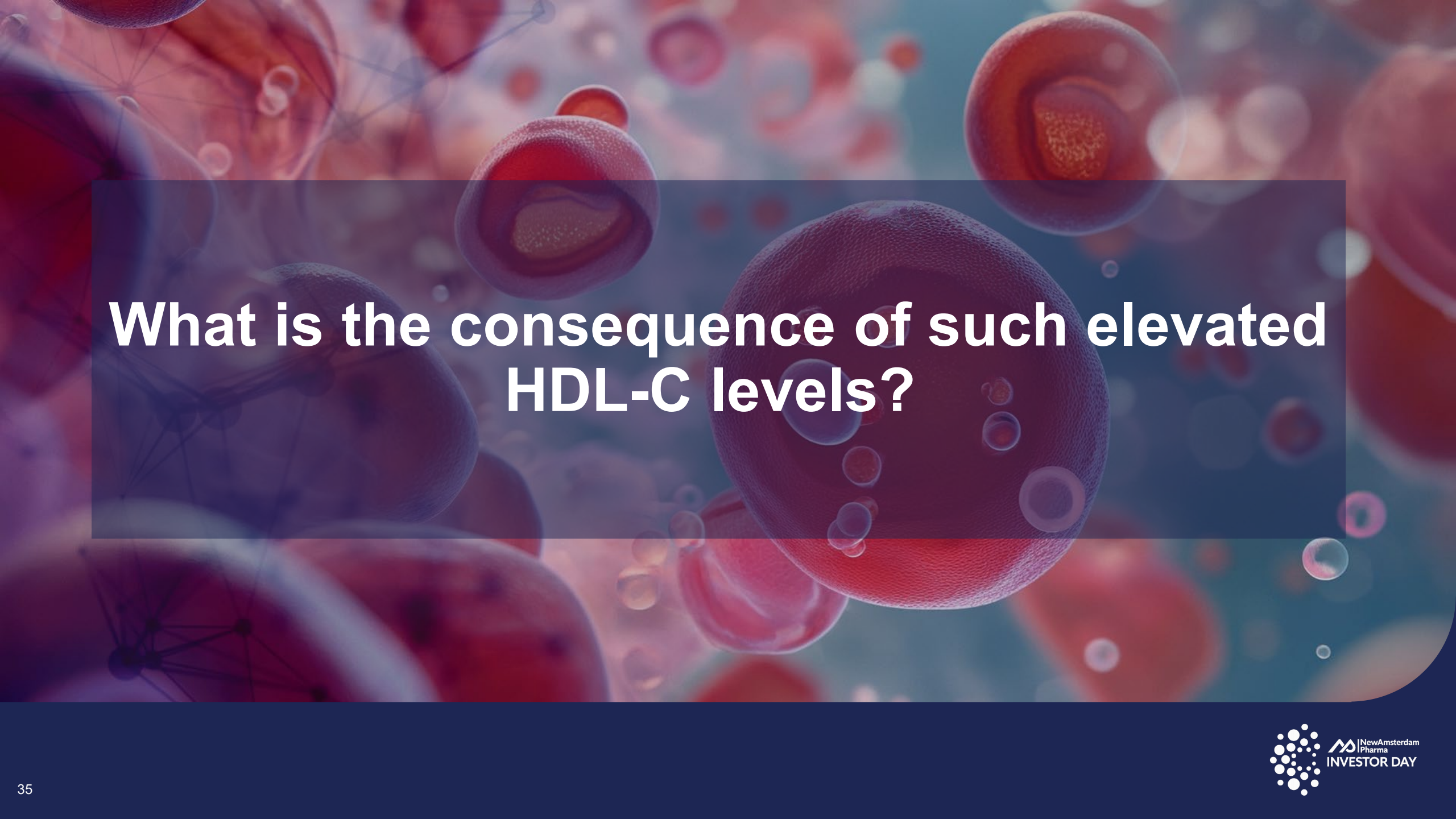


Lower Risk of Sepsis Mortality⁷

Genetically proxied CETP inhibition was associated with lower risk of sepsis-related critical care admission and death

AD, Alzheimer's disease; AMD, age-related macular degeneration; ApoB, apolipoprotein B; ASCVD, atherosclerotic cardiovascular disease; CETP, cholesteryl ester transfer protein; CV, cardiovascular; HDL, high-density lipoprotein; LoF, loss of function; Lp(a), lipoprotein (a).

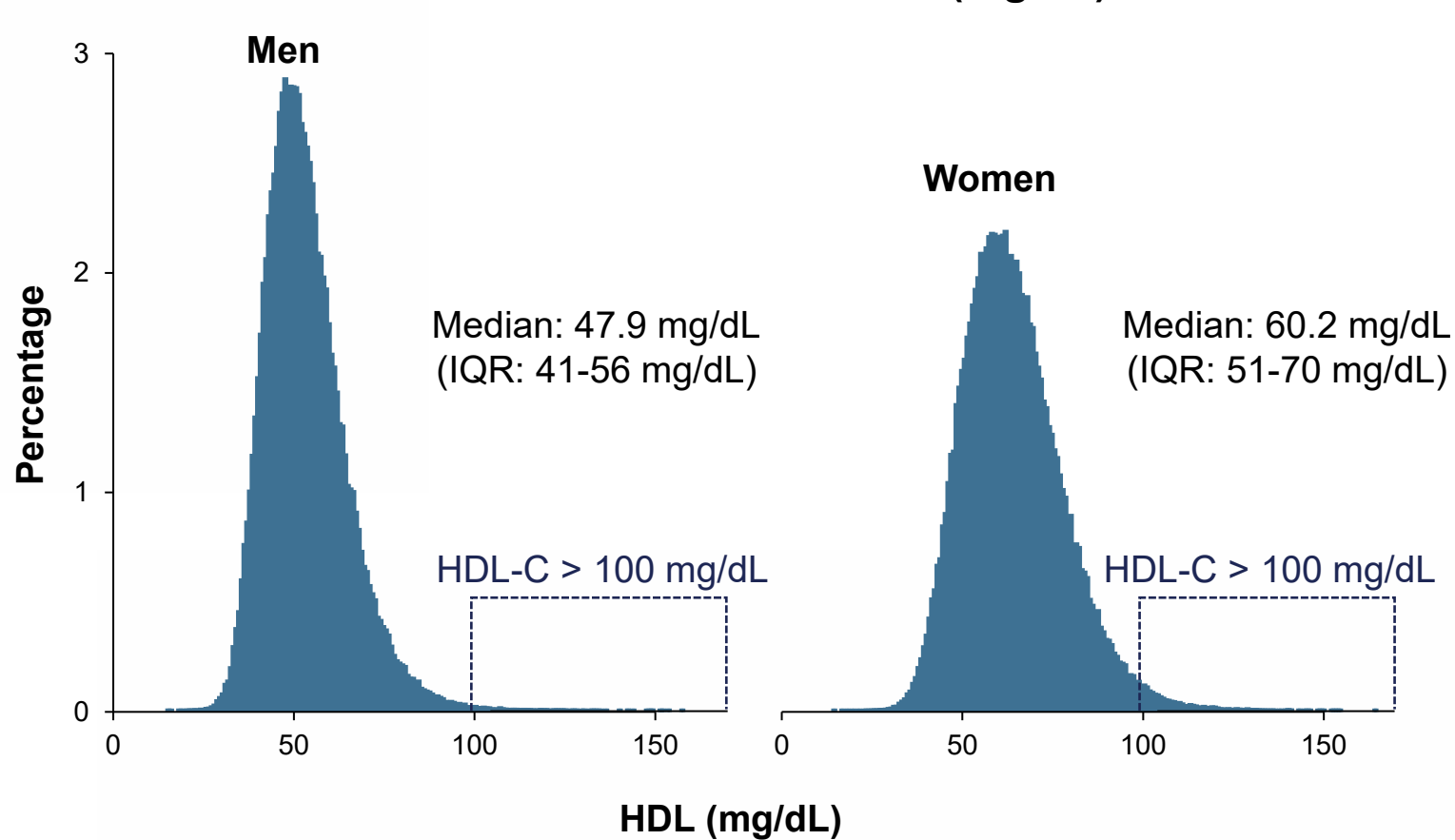
1. Tall AR et al. *Arterioscler Thromb Vasc Biol.* 2007;27(2):257-260. 2. Barzilai N et al. *JAMA.* 2003;290(15):2030-2040. 3. Landfors F et al. *medRxiv.* 2024. 12.02.24318306. 4. Sanders AE et al. *JAMA.* 2010;303(2):150-158. 5. Yu C et al. *J Am Heart Assoc.* 2023;12(21):e031459. 6. Mehta N et al. *Pharmacol Res.* 2023;197:106972. 7. Li Q et al. *World J Emerg Med.* 2025;16(3):256-261.



What is the consequence of such elevated HDL-C levels?

Extremely High Levels of HDL-C* Are Very Uncommon in the General Population

Distribution of HDL-C (mg/dL)



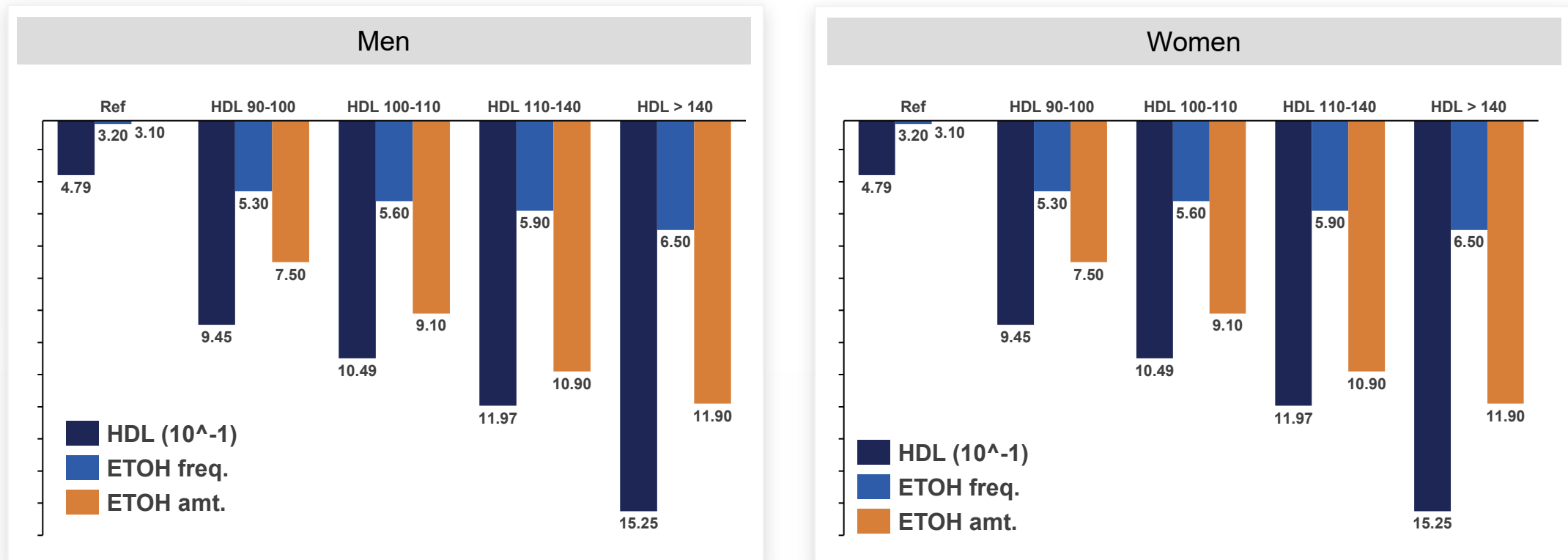
Among 207,741 men in the UK Biobank, 428 (0.5%) had HDL-C levels >100 mg/dL

Among 239,129 women, 3,295 (2%) had HDL-C levels >100 mg/dL

*Reference groups were participants with HDL-C levels between the 25th and 75th percentiles: median (IQR) HDL-C was 47.9 (41-56) mg/dL for men, and 60.2 (51-70) mg/dL for women.
IQR, interquartile range; UK, United Kingdom.
Ference BA et al. ESC 2026. Abstract 86367.

Participants With Extremely High HDL-C Had Progressively Higher Alcohol Use

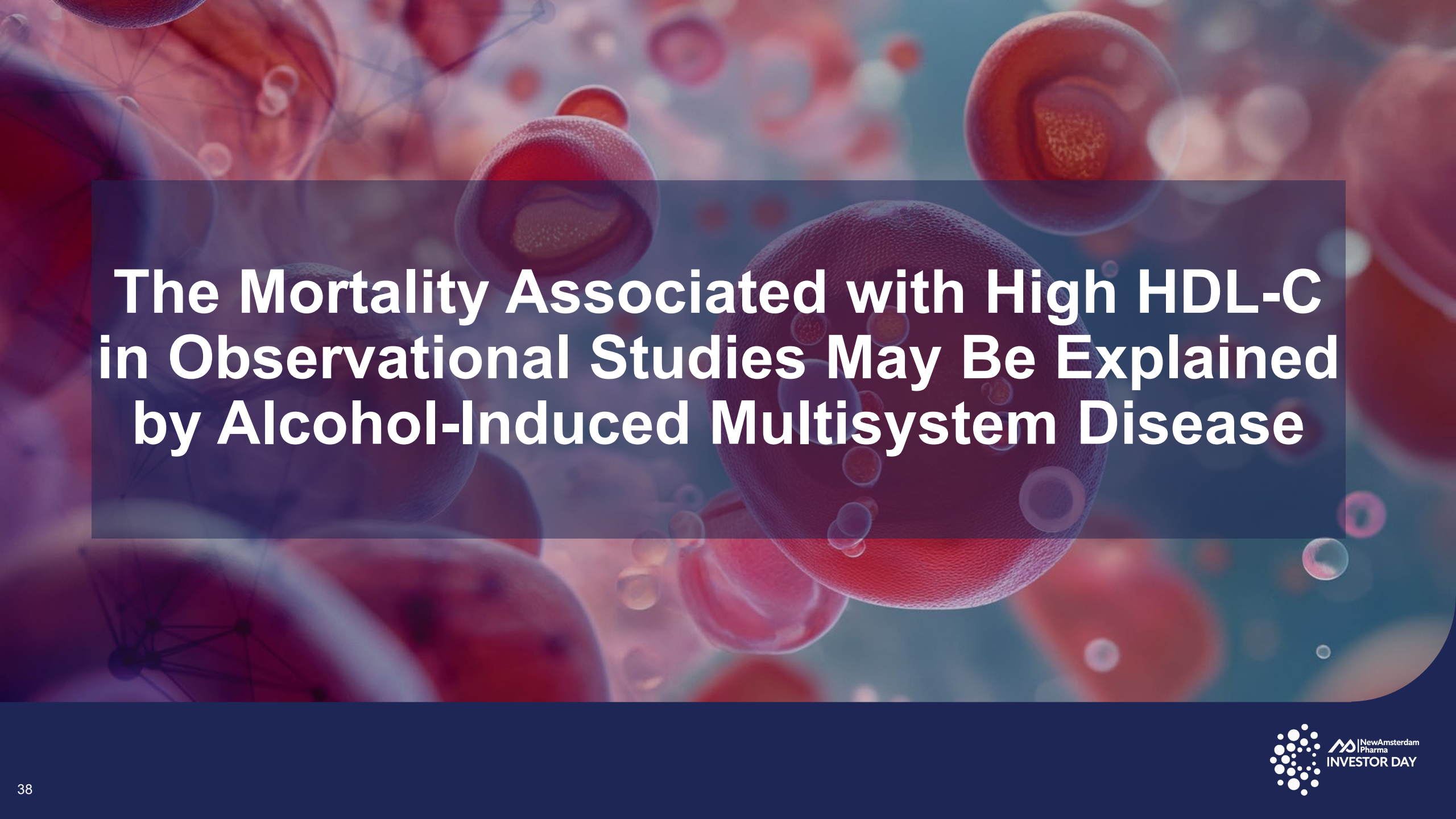
There Is a Stepwise Increase in Alcohol Consumption and Alcohol-Related Comorbidities Among Participants Who Have HDL-C Levels Above 90 mg/dL



Participants with high HDL-C had higher alcohol use, alcohol frequency, GGT, SBP, and heart rate; and lower BMI, weight, and ApoB.

Characteristics of severe alcohol-induced multisystem morbidity.

*Reference groups were participants with HDL-C levels between the 25th and 75th percentiles: median (IQR) HDL-C was 47.9 (41-56) mg/dL for men, and 60.2 (51-70) mg/dL for women. Individual participant data from a study of 429,892 participants enrolled in UK Biobank who had HDL-C measured at baseline. ApoB, apolipoprotein B; BMI, body mass index; ETOH, alcohol; freq, frequency; GGT, gamma-glutamyl transferase; ref, reference group; SBP, systolic blood pressure. Ference BA et al. ESC 2026. Abstract 86367.

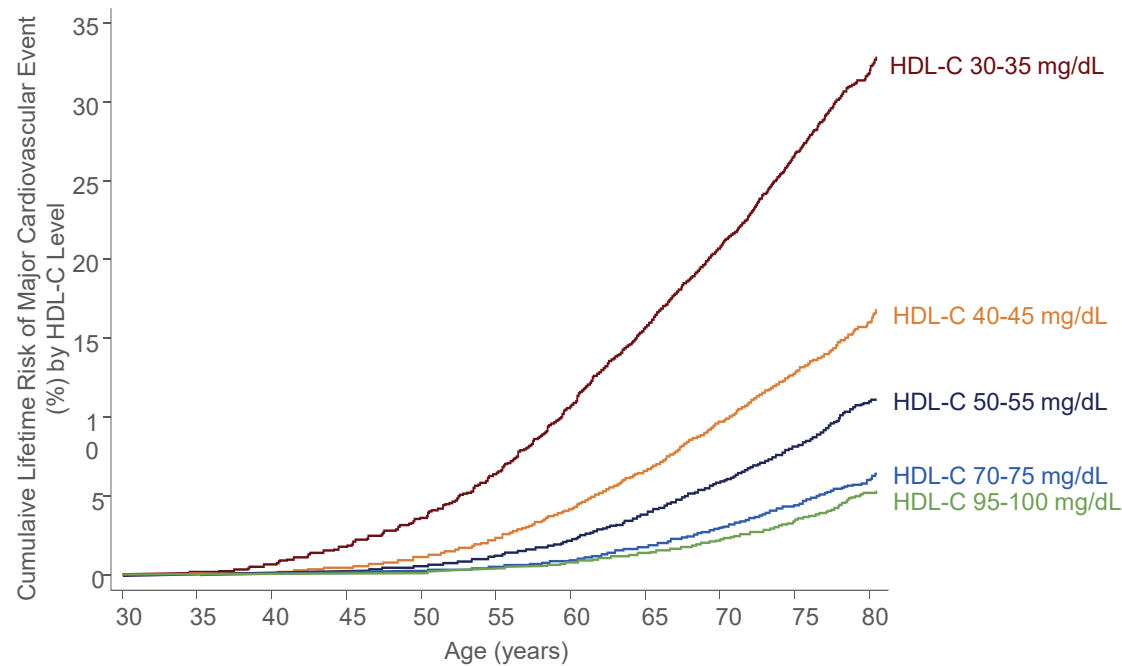


The Mortality Associated with High HDL-C in Observational Studies May Be Explained by Alcohol-Induced Multisystem Disease

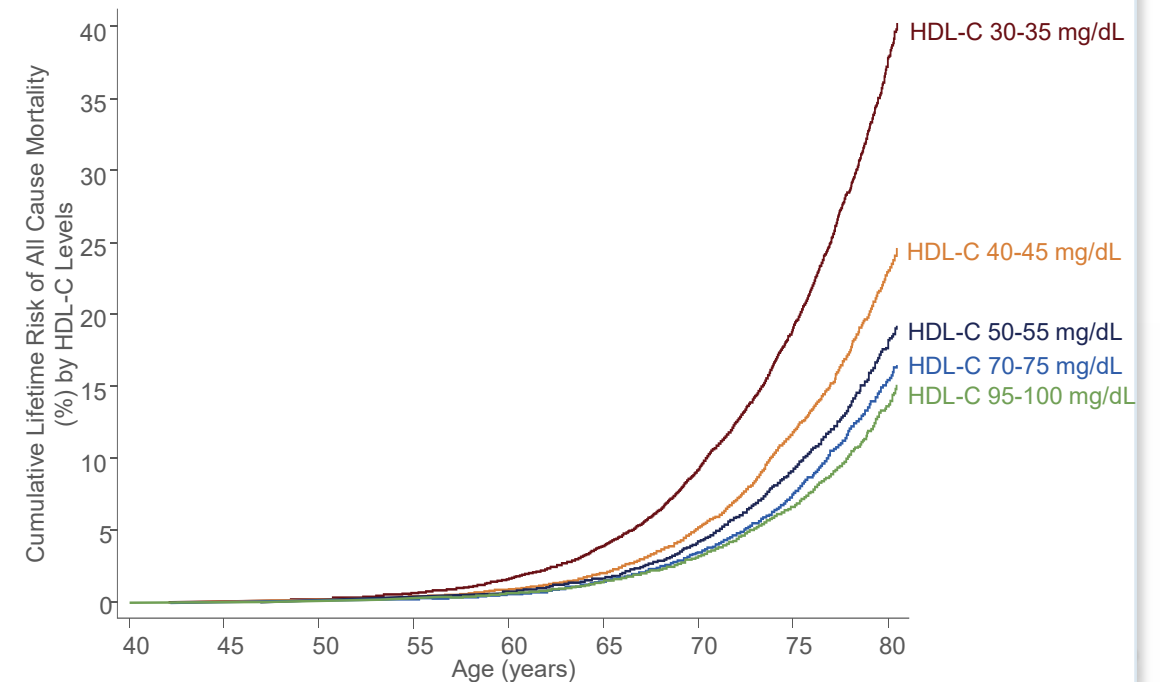
In Fact: HDL-C Levels May be Associated With Decreased Cumulative Lifetime Risk for MACE and All Cause Mortality

ESC CONGRESS
2026 MUNICH

UK Biobank Participants: N=447,651; 29,876 Major Cardiovascular Events; includes both events (and age at event) occurring before and after enrolment to estimate lifetime risk of major coronary events by HDL-c level



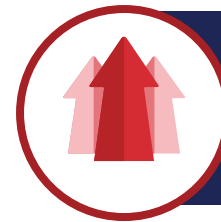
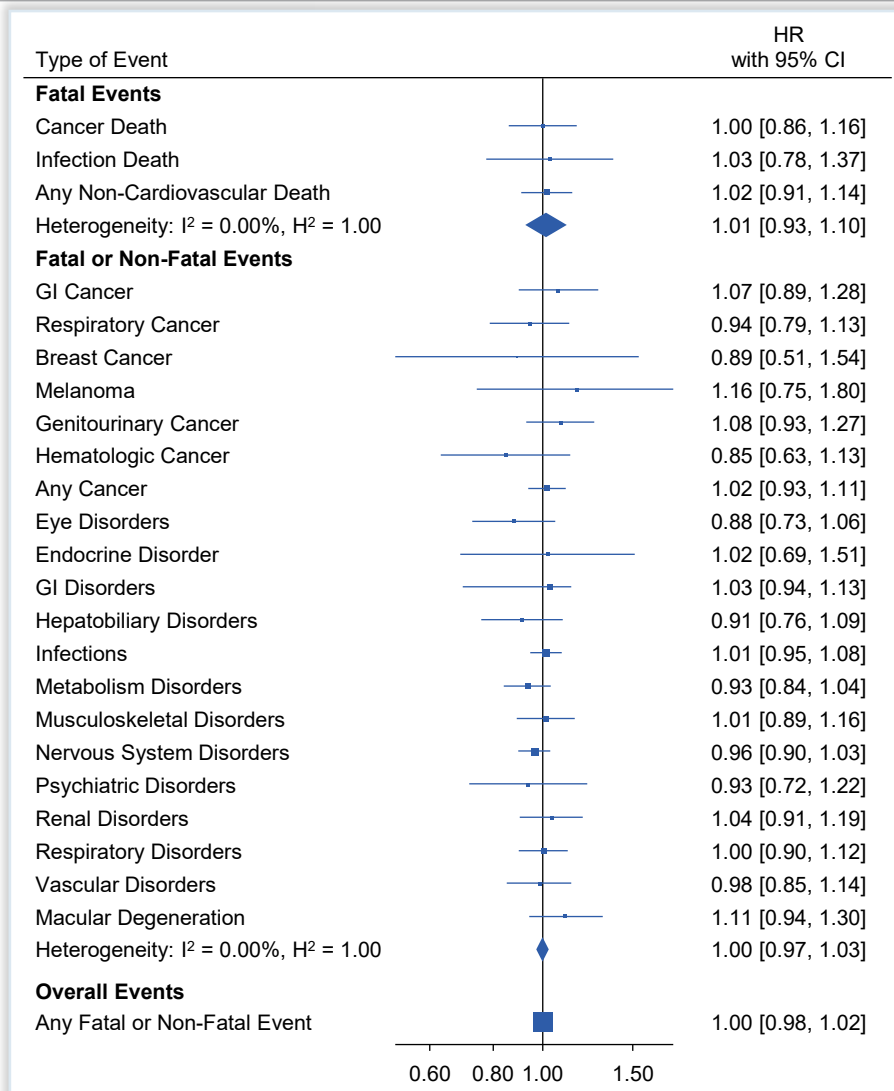
UK Biobank Participants: N=447,651; 31,215 Deaths (any fatal event); Median follow-Up 12.5 years





What is the Safety of a 100% Increase in HDL-C ?

Safety of HDL Increases With CETP Inhibition Were Observed With Anacetrapib in the REVEAL Trial



In REVEAL, anacetrapib increased HDL-C by 104% relative to placebo



This led to no increase in any fatal or non-fatal event in over 30,000 patients



Obicetrapib Clinical Trials Update: Going with the Evidence

Obicetrapib Clinical Pipeline Update

Obicetrapib is being studied in three clinical trials that are designed to demonstrate benefits beyond LDL-C lowering



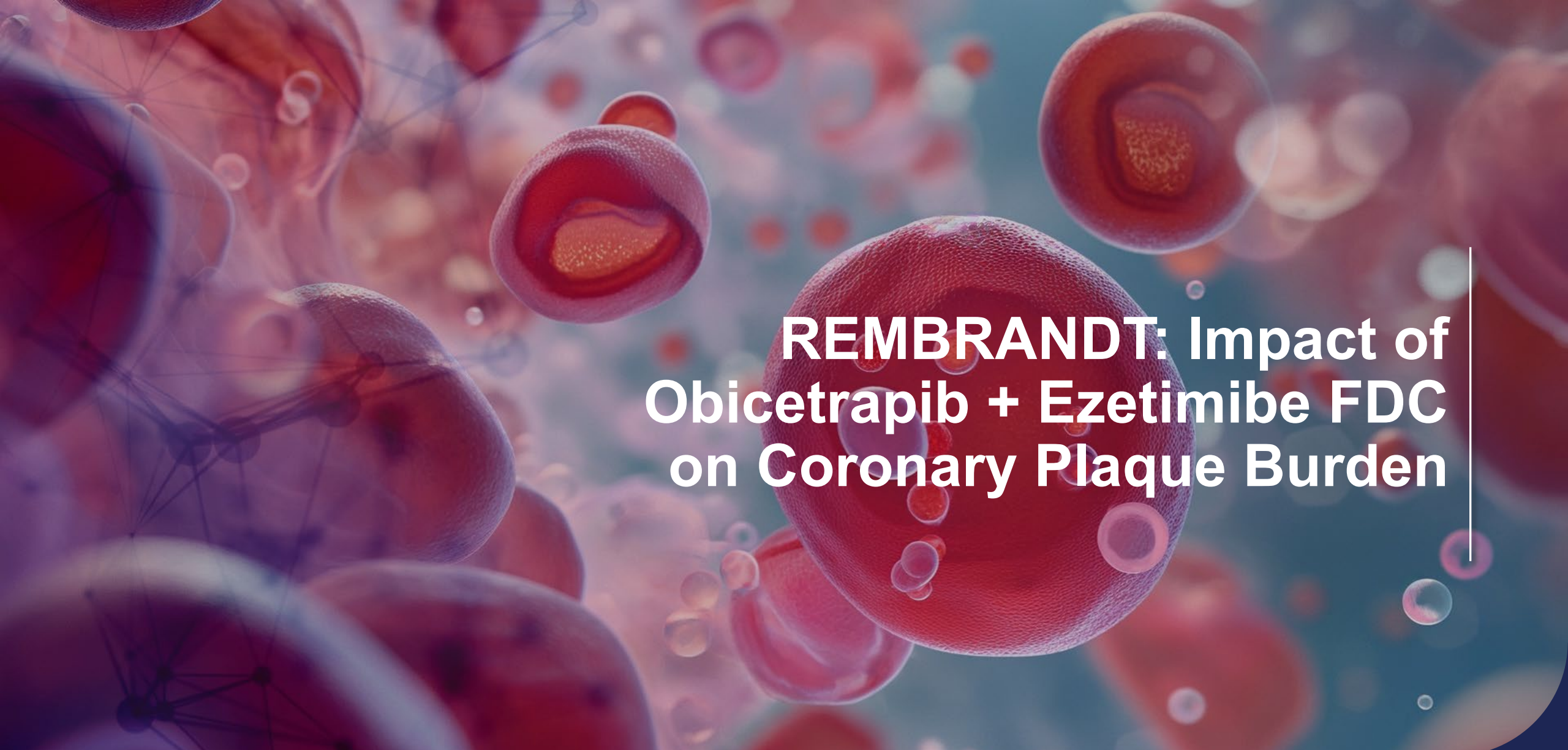
Phase 3 study to evaluate the effect of obicetrapib + ezetimibe FDC on LDL-C lowering in patients with **metabolic syndrome and/or T2D**



Phase 3 CCTA study to evaluate the effect of obicetrapib + ezetimibe FDC on **coronary plaque burden** in patients with **ASCVD**



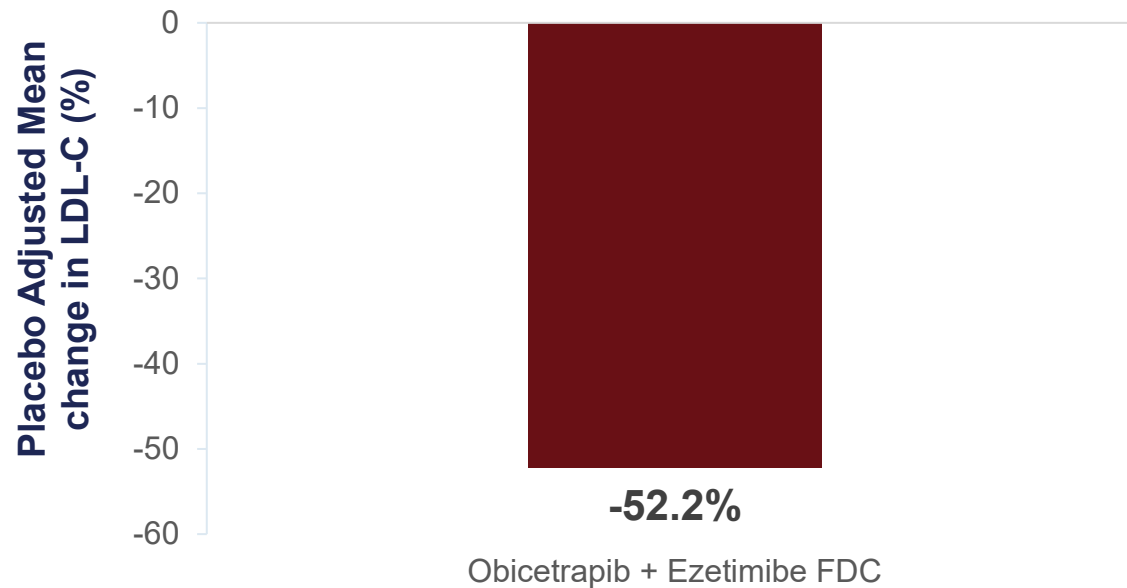
Phase 2b study to evaluate the effect of obicetrapib on Alzheimer's disease biomarkers in high-risk APOE4 carriers with **preclinical AD**



REMBRANDT: Impact of Obicetrapib + Ezetimibe FDC on Coronary Plaque Burden

Obicetrapib + Ezetimibe FDC Delivers PCSK9i – Like* LDL-C Lowering Without Compromising Safety

Obicetrapib + Ezetimibe FDC Demonstrates Robust Reductions in LDL-C



Obicetrapib + Ezetimibe FDC Is Well Tolerated

	Any AE	Serious AE
FDC: Obi + Eze	51%	3%
Obicetrapib (mono)	54%	6%
Ezetimibe (mono)	53%	7%
Placebo	37%	4%

FDC, fixed-dose combination; LDL-C low-density lipoprotein cholesterol; PCSK9, proprotein convertase subtilisin/kexin type 9.

*-PCSK9 label efficacy of 50-60%
Sarraju A, et al. *Lancet*. 2025;405(10491):1757-1768.

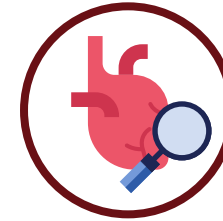
PREVAIL Will Settle Efficacy and Safety — But What About the Plaque?



PREVAIL

The decisive efficacy & safety read-out

OBICETRAPIB MONOTHERAPY

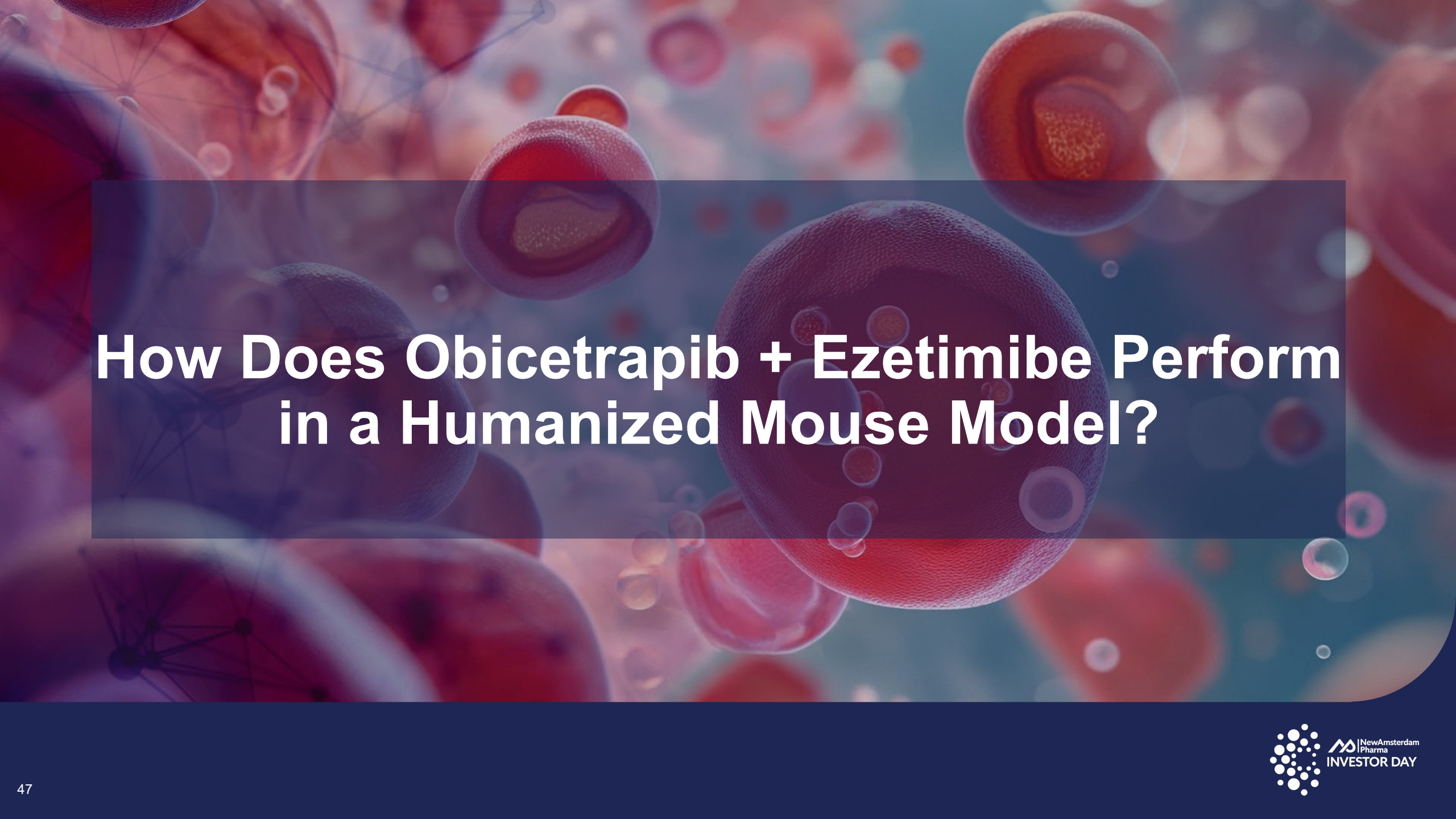


The Plaque Question

How do atherosclerotic plaques behave following treatment with obicetrapib + ezetimibe FDC?

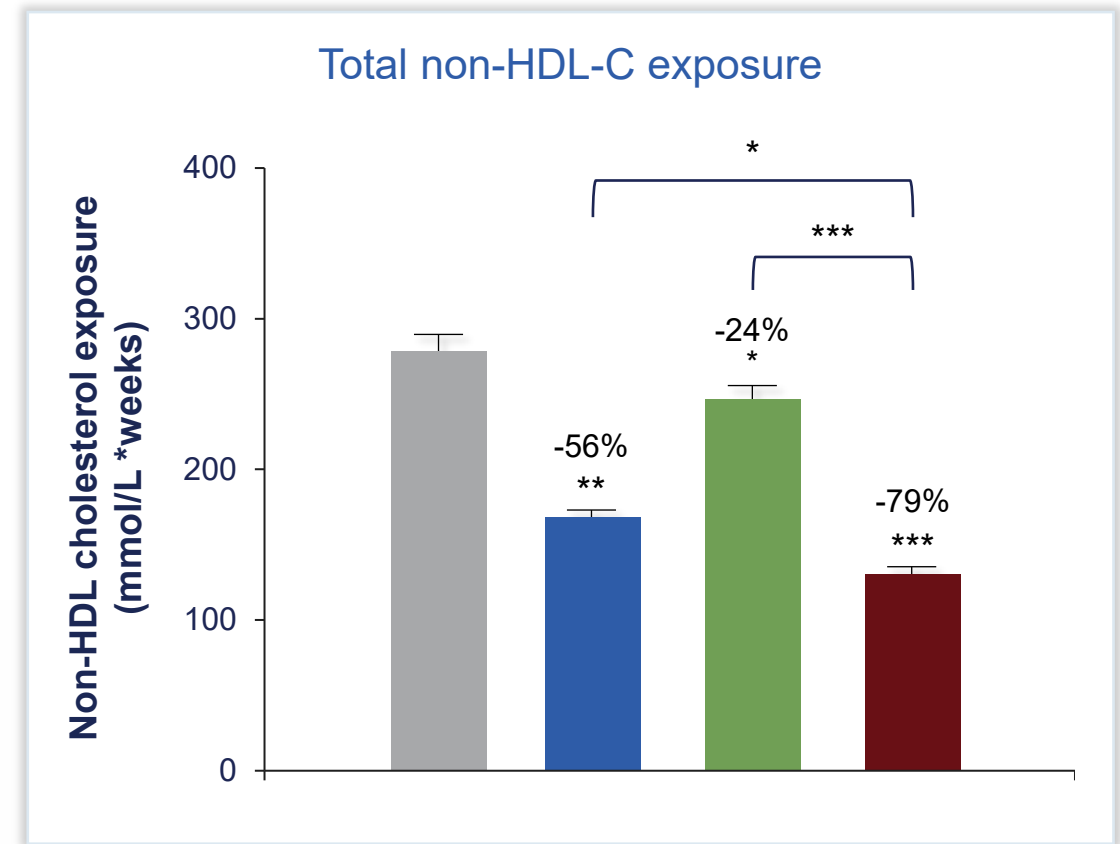
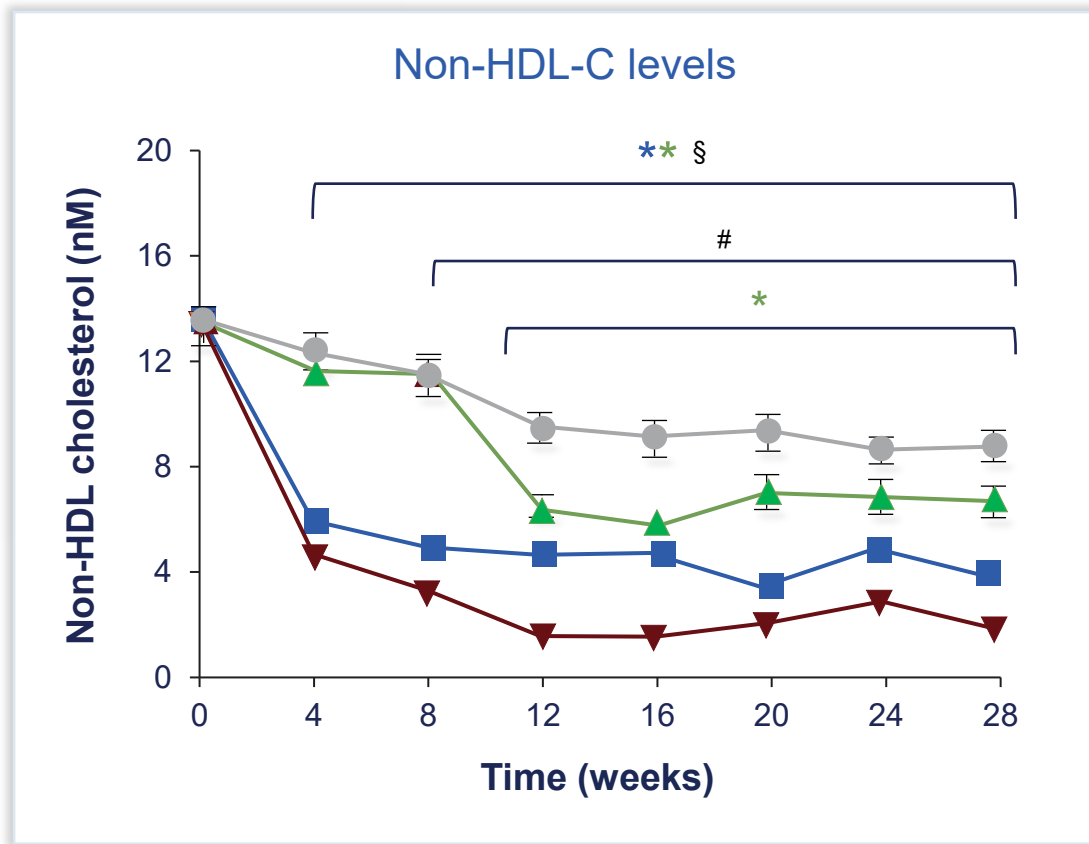
OBICETRAPIB + EZETIMIBE FDC

PREVAIL delivers the decisive read-out — but atherosclerotic plaque behavior under the fixed-dose combination remains to be shown.



How Does Obicetrapib + Ezetimibe Perform in a Humanized Mouse Model?

Obicetrapib plus Ezetimibe Robustly Decreases Non-HDL Cholesterol in Humanized Mouse Model



■ Control ■ Obicetrapib ■ Ezetimibe ■ Obicetrapib + Ezetimibe

*(blue) $P < 0.05$ Obicetrapib vs control. *(red) $P < 0.05$ ezetimibe vs control. *(yellow) $P < 0.05$ ezetimibe vs control. * $P < 0.05$. ** $P < 0.01$. *** $P < 0.001$.

$P < 0.05$ obicetrapib vs obicetrapib + ezetimibe. § $P < 0.05$ ezetimibe vs obicetrapib + ezetimibe.

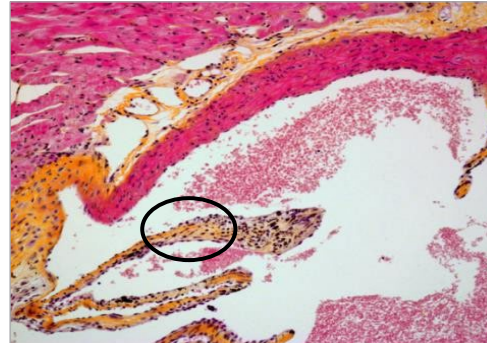
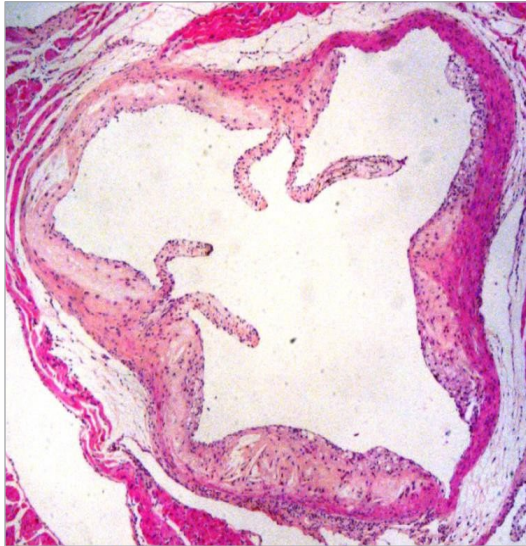
Apo, apolipoprotein; CETP, cholesteryl ester transfer protein; HDL, high-density lipoprotein; HDL-C, high-density lipoprotein cholesterol; LDL, low-density lipoprotein; VLDL, very-low-density lipoprotein.

Inia JA et al. *J Clin Lipidol.* 2025;19(3S):e111.

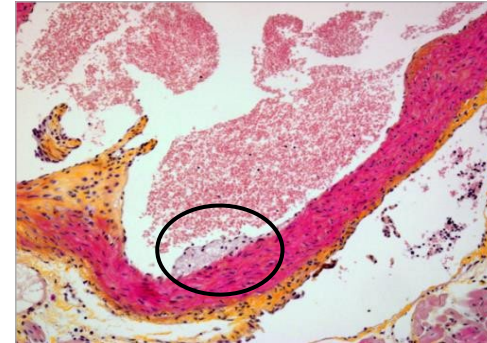


How Did Atherosclerotic Plaques Respond to These Changes in Lipids?

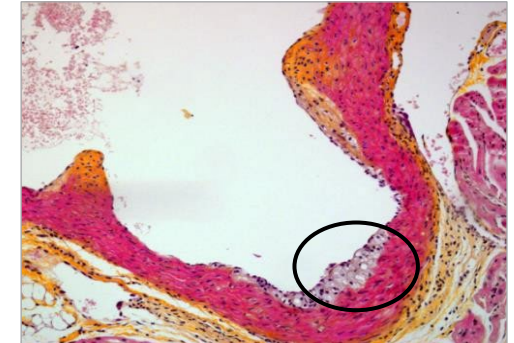
Atherosclerotic Lesions Remain Main Contributor to Risk for Heart Attack and Stroke



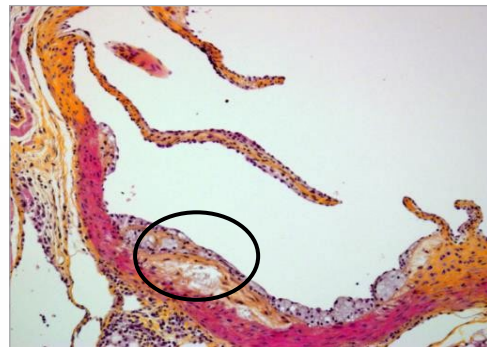
Type I – Early fatty streak
Up to 10 foam cells in the intima



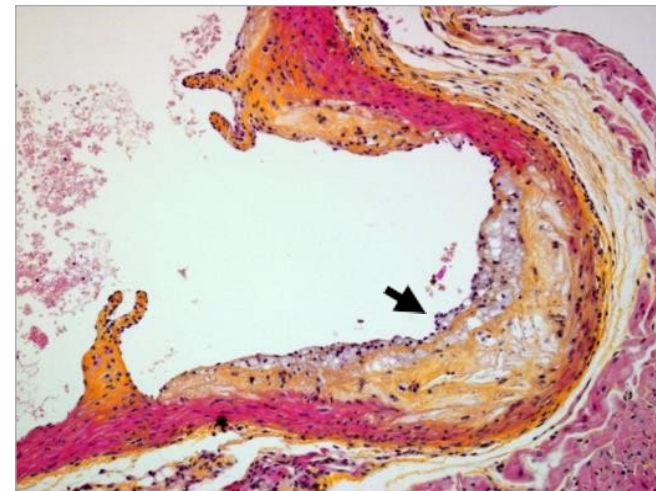
Type II – Regular fatty streak
Over 10 foam cells present in the intima



Type III – Mild plaque
Extension of foam cells into the media with presence of a fibrotic cap



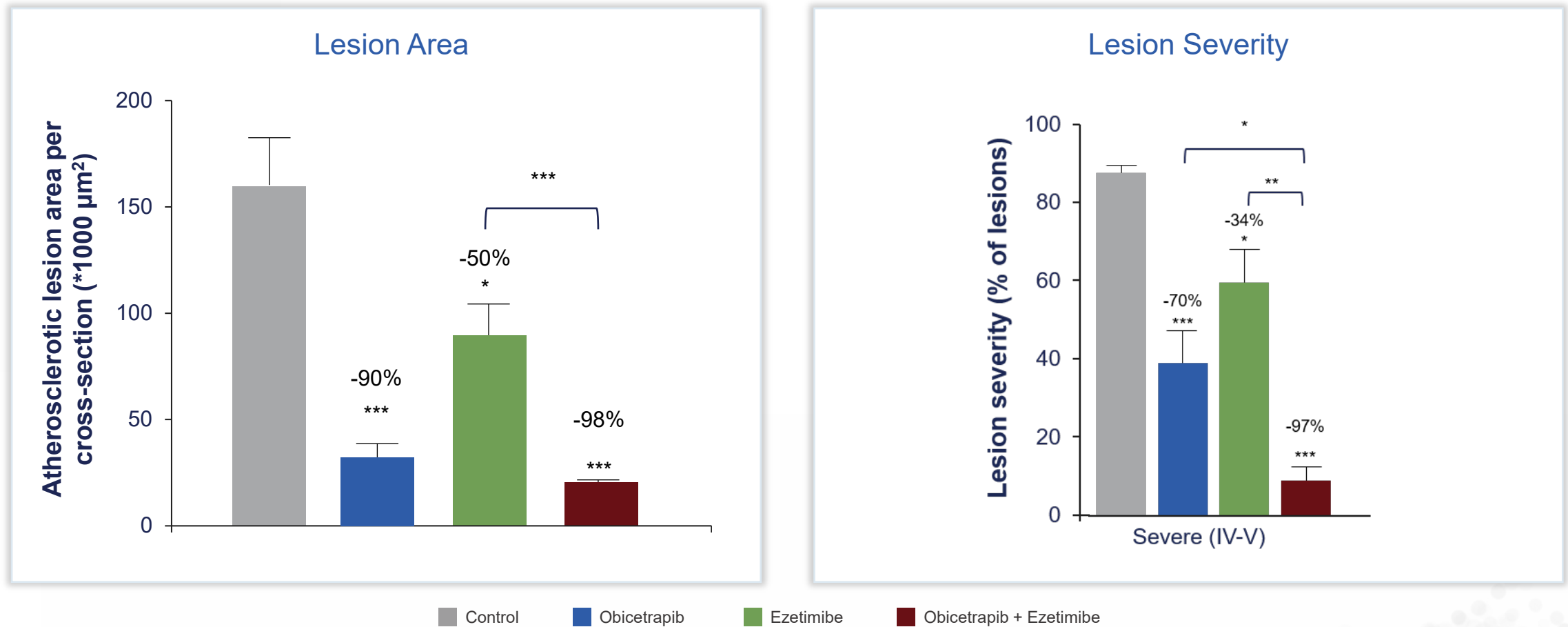
Type IV – Moderate plaque
A more progressive lesion with affected media, without loss of architecture and fibrosis of the media



Type V – Severe plaque

- Media is severely damaged with broken internal elastic lamina
- Cholesterol clefts and/or crystals, calcium mineralization and necrosis are often visible

Obicetrapib plus Ezetimibe Decreased Surface Area of Severe Lesions in the Aortic Root by 98%

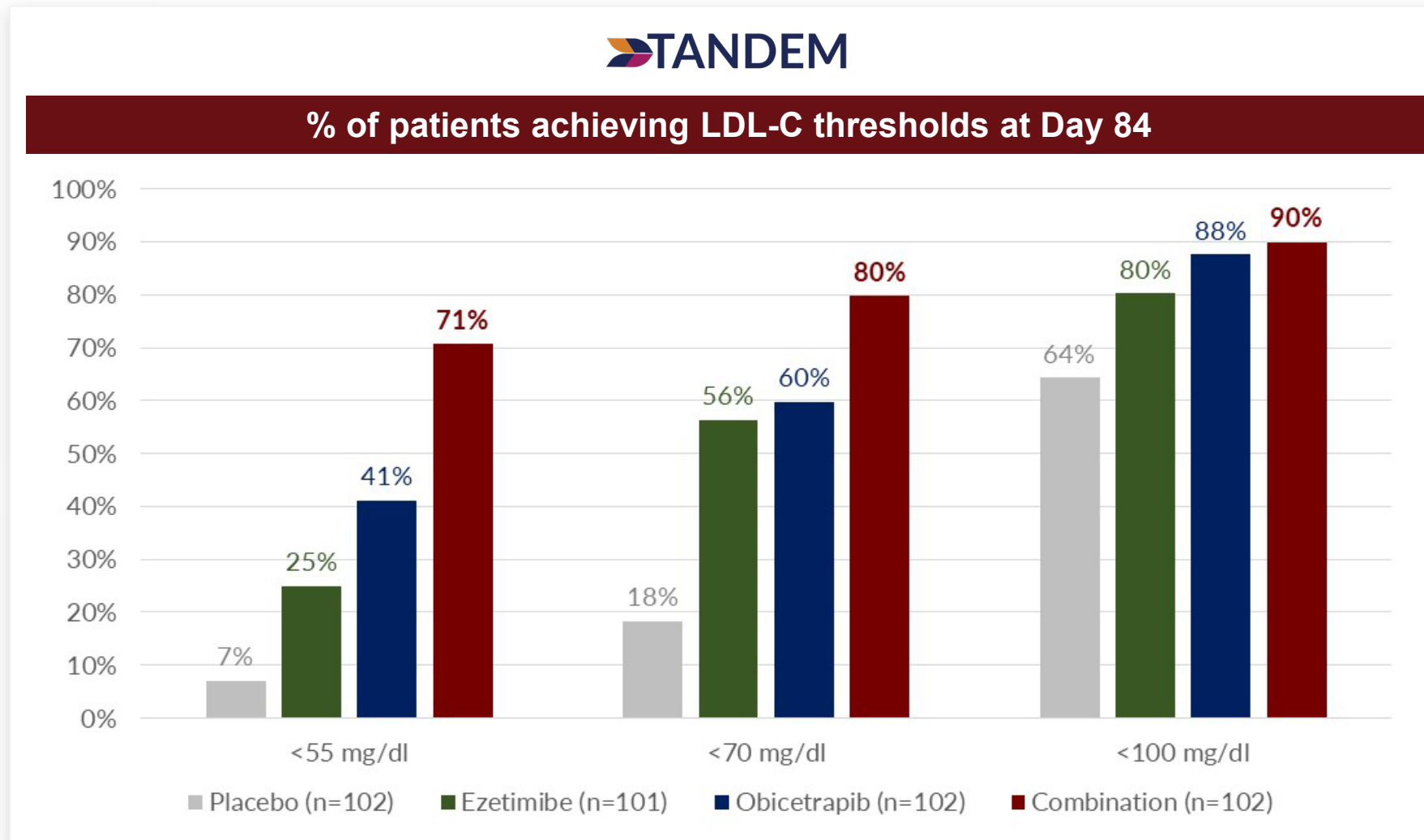


* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.
Inia JA et al. *J Clin Lipidol*. 2025;19(3S):e111.



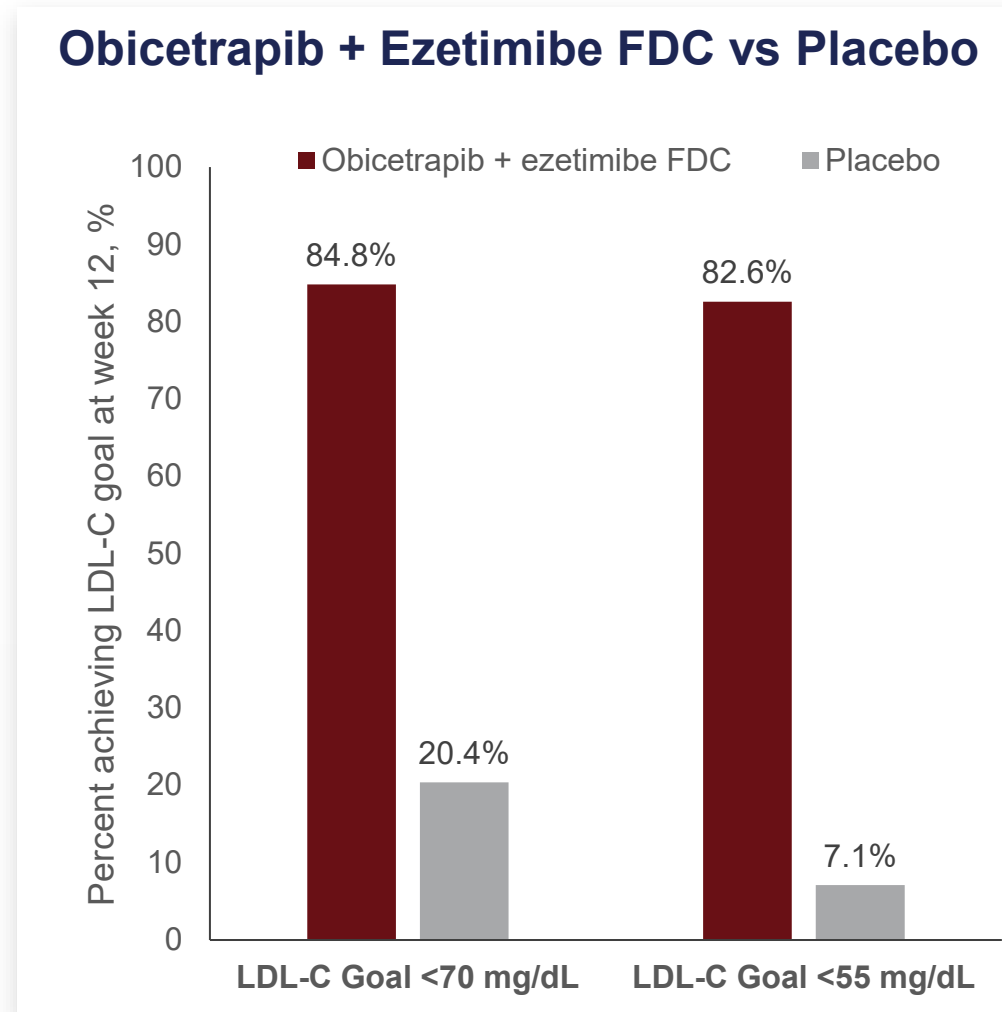
Now Back to Humans....

Over 70% of Patients on Obicetrapib + Ezetimibe Achieved LDL-C <55 mg/dL



Source: Data on file. LDL-C, low-density lipoprotein cholesterol.

PK-Confirmed On-Treatment LDL-C Goal Attainment Was High For Obicetrapib Monotherapy and Obicetrapib + Ezetimibe FDC



REMBRANDT

Effect of FDC Obicetrapib/Ezetimibe on Top of Maximally Tolerated Lipid-Modifying Therapy on Coronary Plaque Characteristics in Participants with or at High Risk for ASCVD

Main inclusion criteria

- Age > 45 years
- ASCVD
 - Imaging evidence (CTA / Angio) of vascular disease.
 - Having established ASCVD (MI, Stroke, revasc, PAD)
- Non calcified total plaque volume >75mm³
- Screening LDL-C level ≥ 70 mg/dL
- On maximally tolerated lipid-modifying therapy

Main exclusion criteria

- Poorly controlled diabetes (HbA1c >10%)
- Hypertension
- Congestive heart failure
- PCI/stroke/MI within 3 months
- eGFR < 45mL/min
- Ezetimibe use within 30 days

Primary endpoint

- % Change total NCPV in ALL major coronary arteries

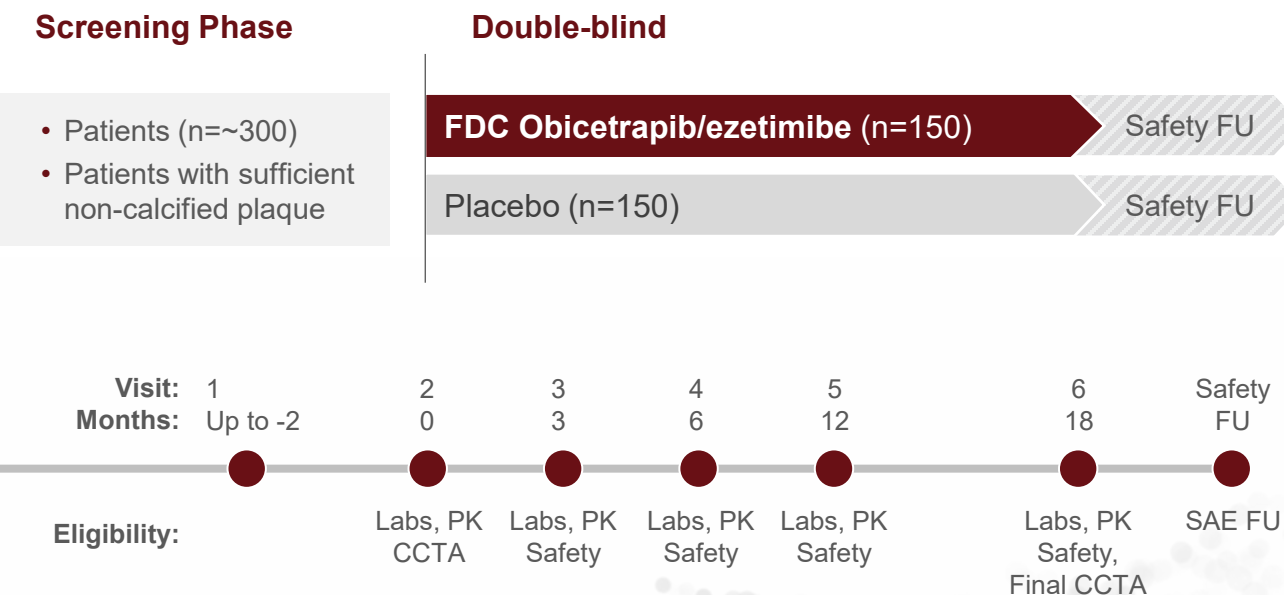
Secondary endpoint

- Absolute change NCPV in ALL major coronary arteries
- % change in LDL-C
- % change NCPVMD
- Absolute change NCPVMD
- Change in FAI

Rationale: Despite reducing the progression of coronary atherosclerosis, statin therapy does not fully address residual CV risk. Adding the FDC obicetrapib/ezetimibe to statin therapy will further reduce the progression of coronary atherosclerosis in ASCVD patients with high residually risk

Objective: To evaluate the effect of FDC obicetrapib/ezetimibe 10 mg FDC daily on total non-calcified coronary atherosclerotic plaque volume at 18 months

Study design: Randomized, double-blind, placebo-controlled



REMBRANDT: CT Angiography Study Designed to Demonstrate Obicetrapib + Ezetimibe FDC's Anti-Atherosclerotic

1

Show LDL-C reduction yields benefit on underlying atherosclerosis

2

Demonstrate FDC regresses coronary plaque volume

3

Link FDC benefit directly to plaque improvement

4

Confirm efficacy and safety in high-risk patients



Increased utilization of CTA for diagnosis of cardiovascular disease



Positive traction with practicing cardiologists



Designed to demonstrate clinical benefit of FDC

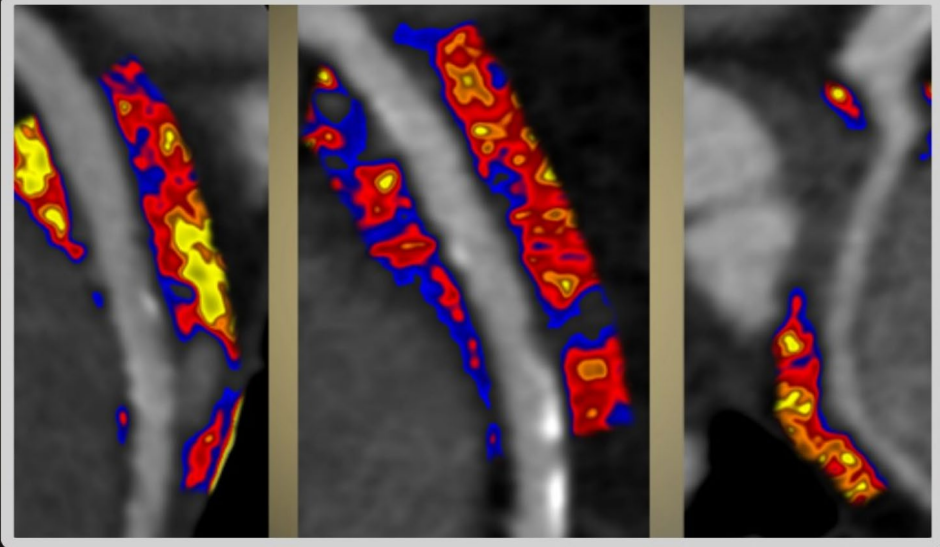


May support labeling and promotion

Recent FDA Approval of New CCTA Imaging Technology

“CaRi-Heart has received U.S. FDA De Novo authorization, becoming the first and only technology authorized in the U.S. to detect and quantify coronary inflammation from routine coronary CT angiography (CCTA).”

 **CARISTO'S CARI-HEART® CORONARY INFLAMMATION TECHNOLOGY AUTHORIZED BY U.S. FDA**



CARISTO BRINGS CORONARY INFLAMMATION DETECTION TO U.S. CARDIOVASCULAR CARE

REMBRANDT Is Fully Enrolled and Tracking to a Readout^{1,2}



ENROLLMENT COMPLETE

323

patients enrolled (vs.
~300 target)

50

sites across 7 countries

18 mo

double-blind treatment
period per patient

2027

estimated trial
completion



First Patient Randomized

May 2024

Trial Completion

YE2027 Est.

Last Patient Randomized

March 2026

Fully enrolled, on schedule, and on track to deliver the next major proof point for the FDC franchise

1. NewAmsterdam Pharma corporate updates (Jan & May 2026); 2. ClinicalTrials.gov NCT06305559.

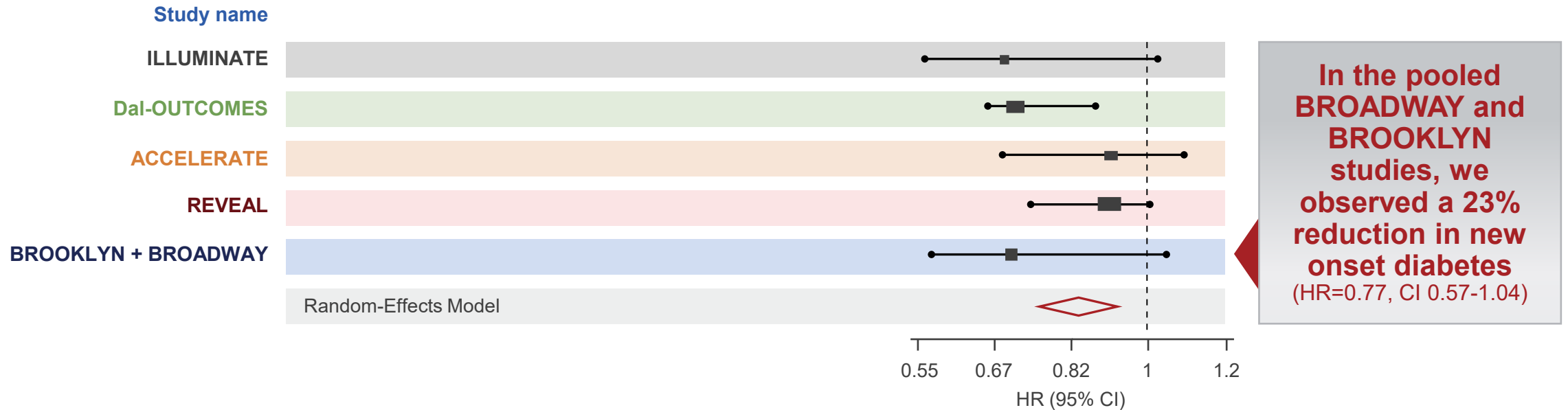


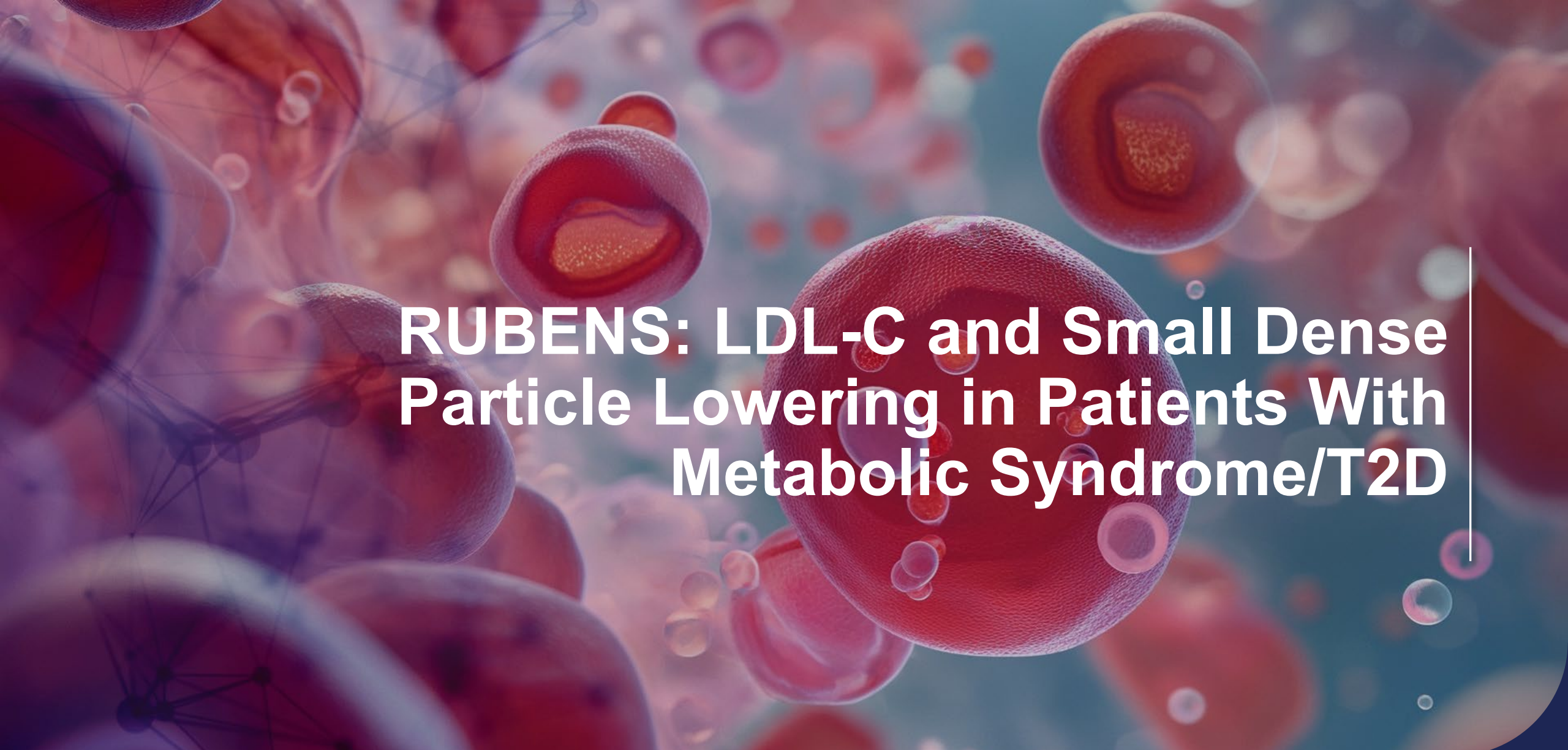


From Plaque to Small LDL Particles and Diabetes

CETP Inhibition Has the Potential to Reverse Diabetes Risk, Potentially Addressing Patient Concerns About High Dose Statins

This is consistent with the benefits observed in other CETP trials that have shown 17% reduction in new onset diabetes (HR=0.83, CI 0.77-0.90)



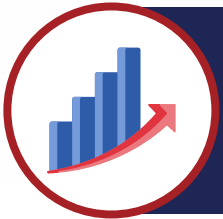


RUBENS: LDL-C and Small Dense Particle Lowering in Patients With Metabolic Syndrome/T2D

LDL-C Lowering in Patients with Obesity, Metabolic Syndrome, and Type 2 Diabetes: The Sweet Spot for Obicetrapib



Shared patient population: patients with obesity, metabolic syndrome, and T2D are frequently prescribed statins

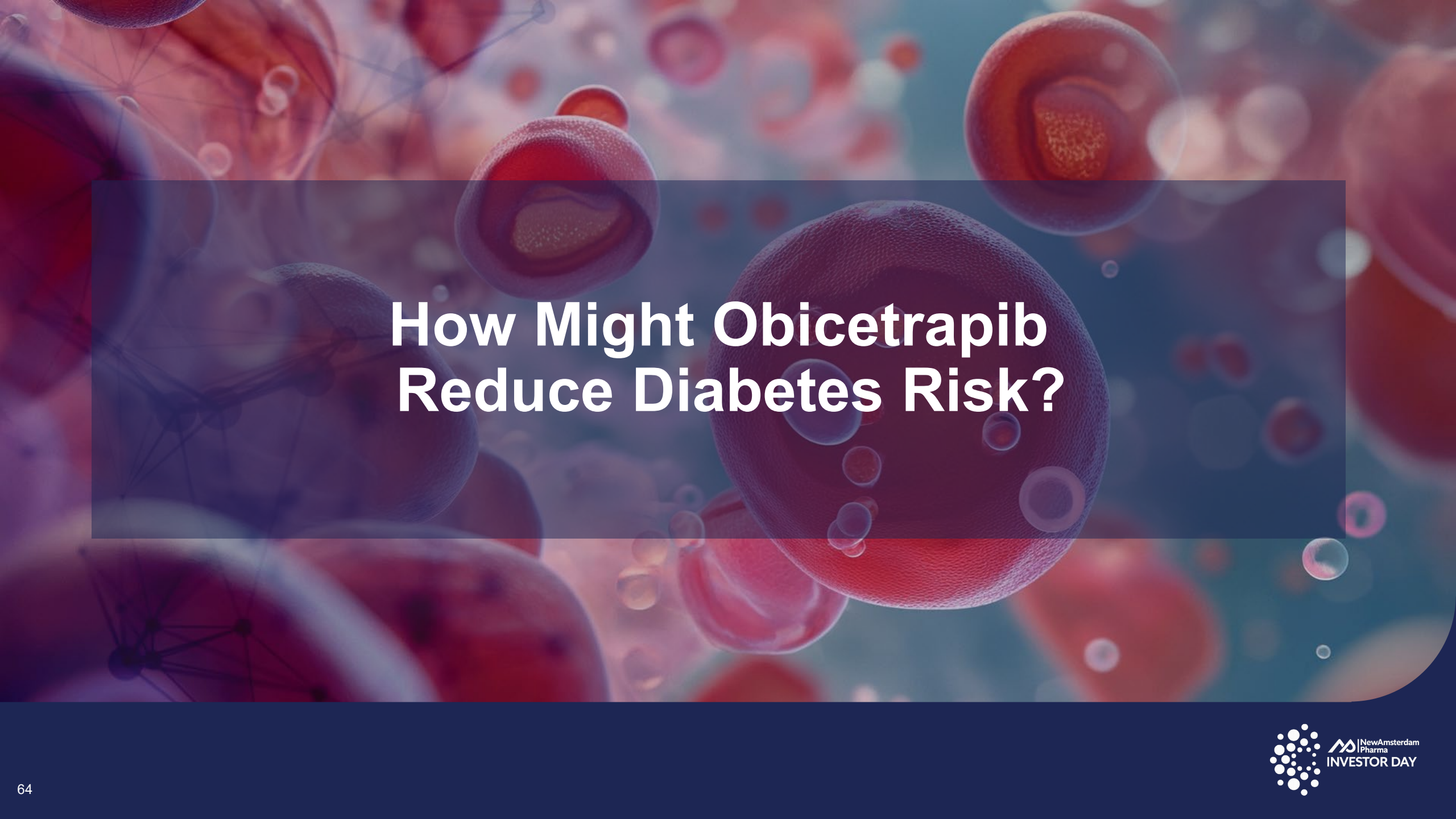


Statins cut both ways: statins increase diabetes risk and preferentially reduce large LDL particles, leaving relatively more small LDL-Ps



Obicetrapib fills the gap: obicetrapib lowers the NoD risk and dramatically reduces small LDL-Ps, potentially being the ideal complement to statin therapy

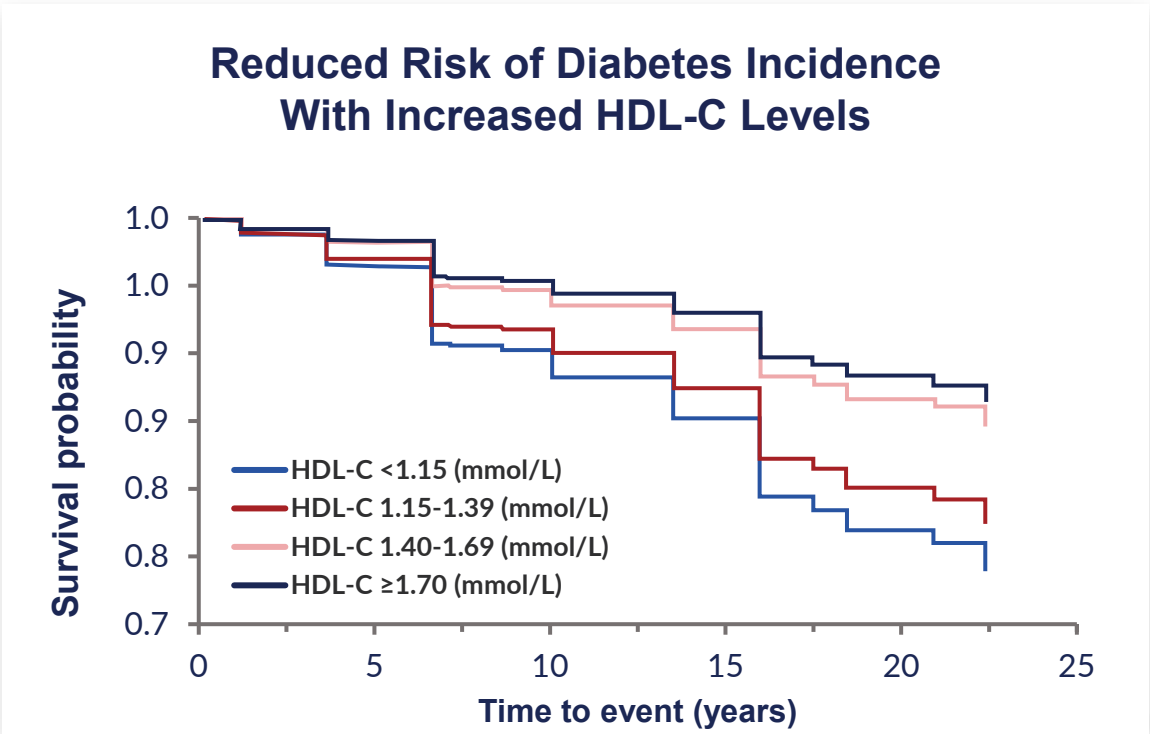
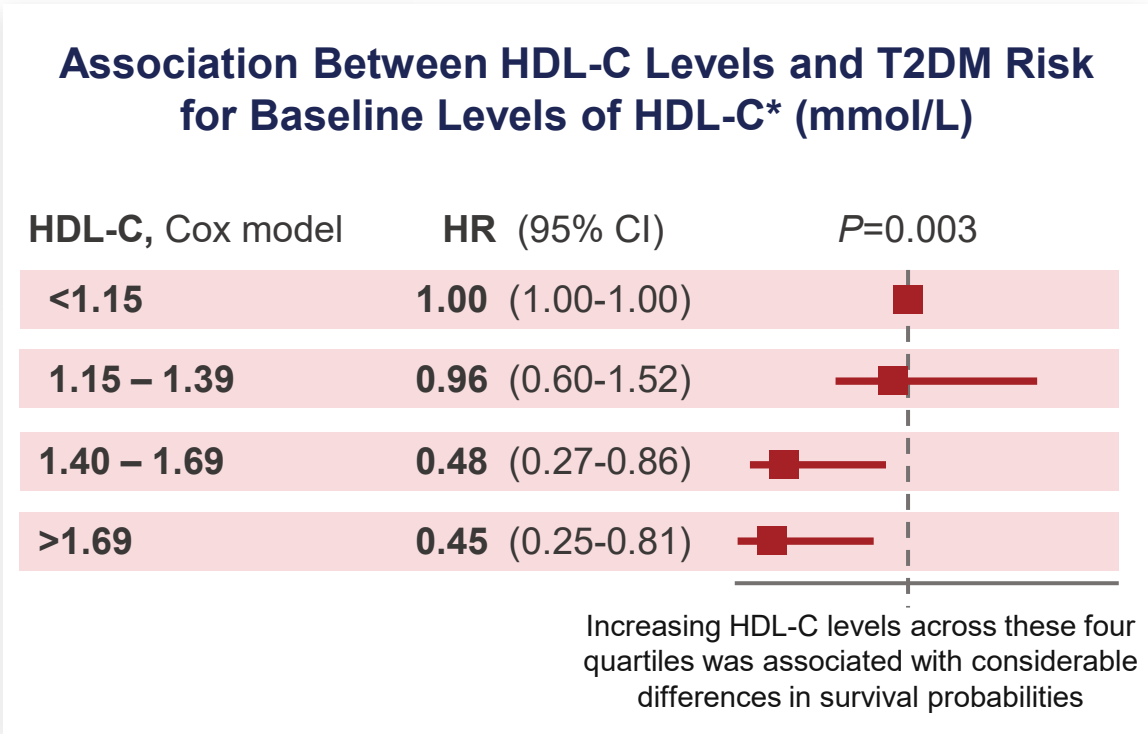
1. Laakso M, Silva LF. *Front Endocrinol (Lausanne)*. 2023;14:1239335. 2. Dugani SB et al. *JAMA Cardiol*. 2016;1(2):136-145. 3. Toth PP et al. *Am J Cardiol*. 2018;121(3):308-314. 4. Ray KK et al. *J Clin Lipidol*. 2025;19(3S):e116-e117. 5. Data on File. NewAmsterdam Pharma. 6. Ballantyne CM et al. *J Clin Lipidol*. 2023;17(4):491-503. 7. Kastelein JJP et al. *Curr Atheroscler Rep*. 2024;26(2):35-44. 8. Nicholls SJ et al. Safety and efficacy of obicetrapib in patients with heterozygous familial hypercholesterolemia: the BROOKLYN trial [manuscript in preparation]. 9. Sarraju A et al. Fixed-dose combination of obicetrapib and ezetimibe for LDL cholesterol reduction [manuscript in preparation].



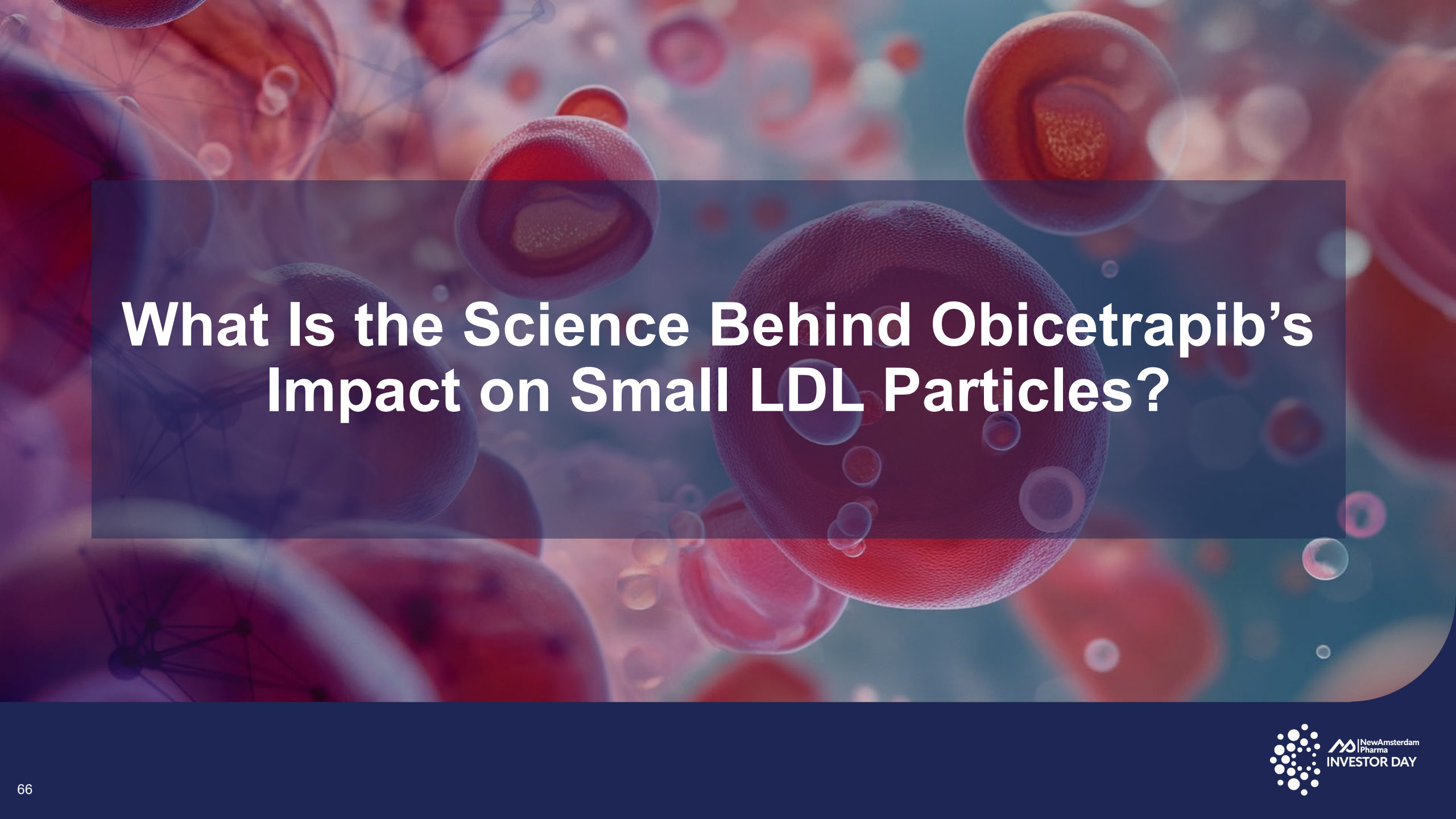
How Might Obicetrapib Reduce Diabetes Risk?

Increase in HDL-C Observed to Reduce Risk of Developing Type 2 Diabetes

- Dose-response relationship observed between HDL-C and the risk of new-onset diabetes
- Multivariate predictive models show robust negative correlation between HDL-C and risk of future T2DM incidence
- 25-year follow-up data also show that increased HDL-C correlates with diminished T2DM risk, suggesting that HDL-C levels may influence glucose metabolism

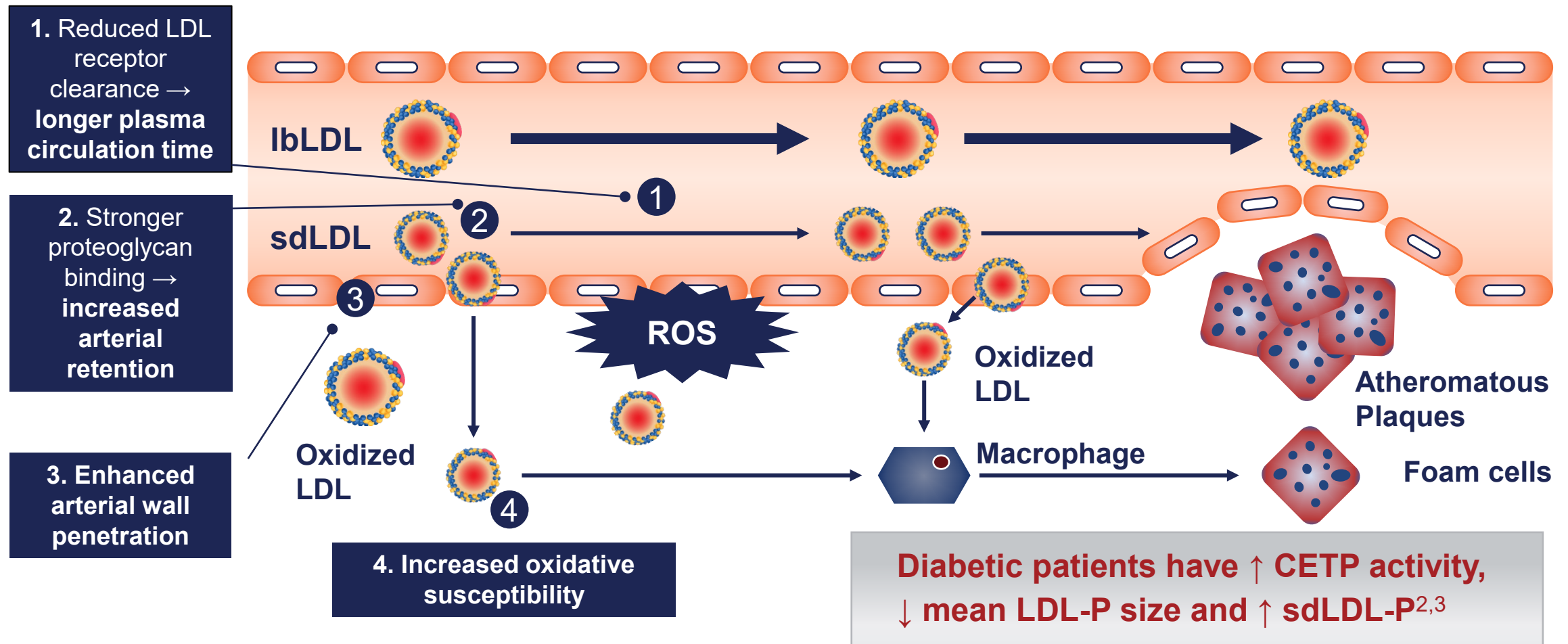


*1 mmol/L HDL=38.67 mg/dL.
CI, confidence interval; HDL-C, high-density lipoprotein cholesterol; HR, hazard ratio; T2DM, type 2 diabetes mellitus.
Cao X et al. *Lipids Health Dis.* 2021;20(1):71.



What Is the Science Behind Obicetrapib's Impact on Small LDL Particles?

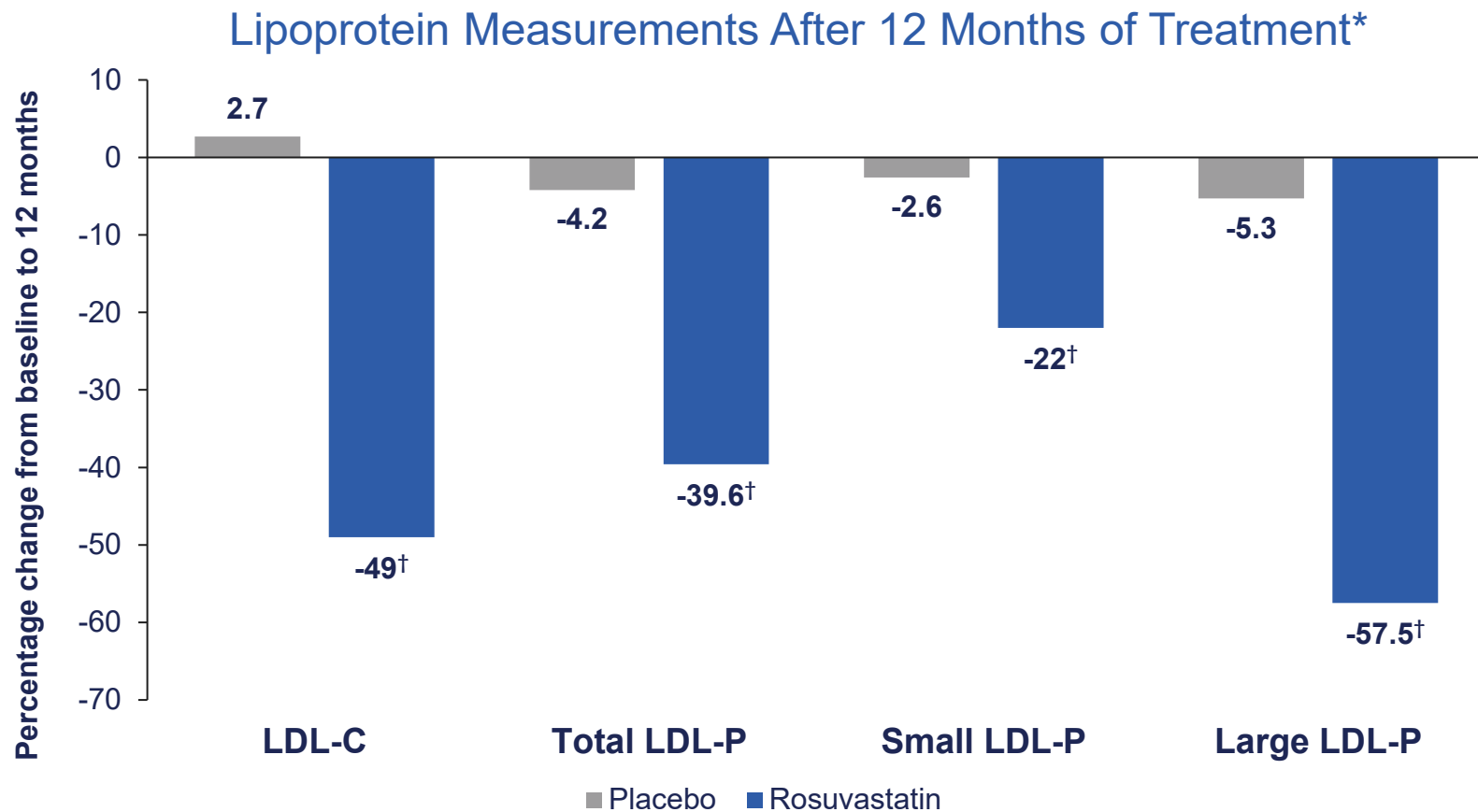
Insulin Resistance-Induced Dyslipidemia is Characterized by Increased sdLDL, Which are More Atherogenic Due to Multiple Mechanisms



IbLDL, large buoyant LDL; LDL-P, LDL particle; ROS, reactive oxygen species.

1. Hirano T. *J Diabetes Investig.* 2025;16(3):370-383. 2. He Q et al. *Sci Rep.* 2025;15(1):19500. 3. Siewert S et al. *J Diabetes Investig.* 2015;6(1):67-77.

Statins Decrease Large LDL-P Concentration to a Greater Extent Than Small LDL-P



Rosuvastatin treatment altered the LDL lipoprotein subclass distribution, resulting in smaller mean sizes (-1.5%; 95% CI, -3.7% to 0.5%)

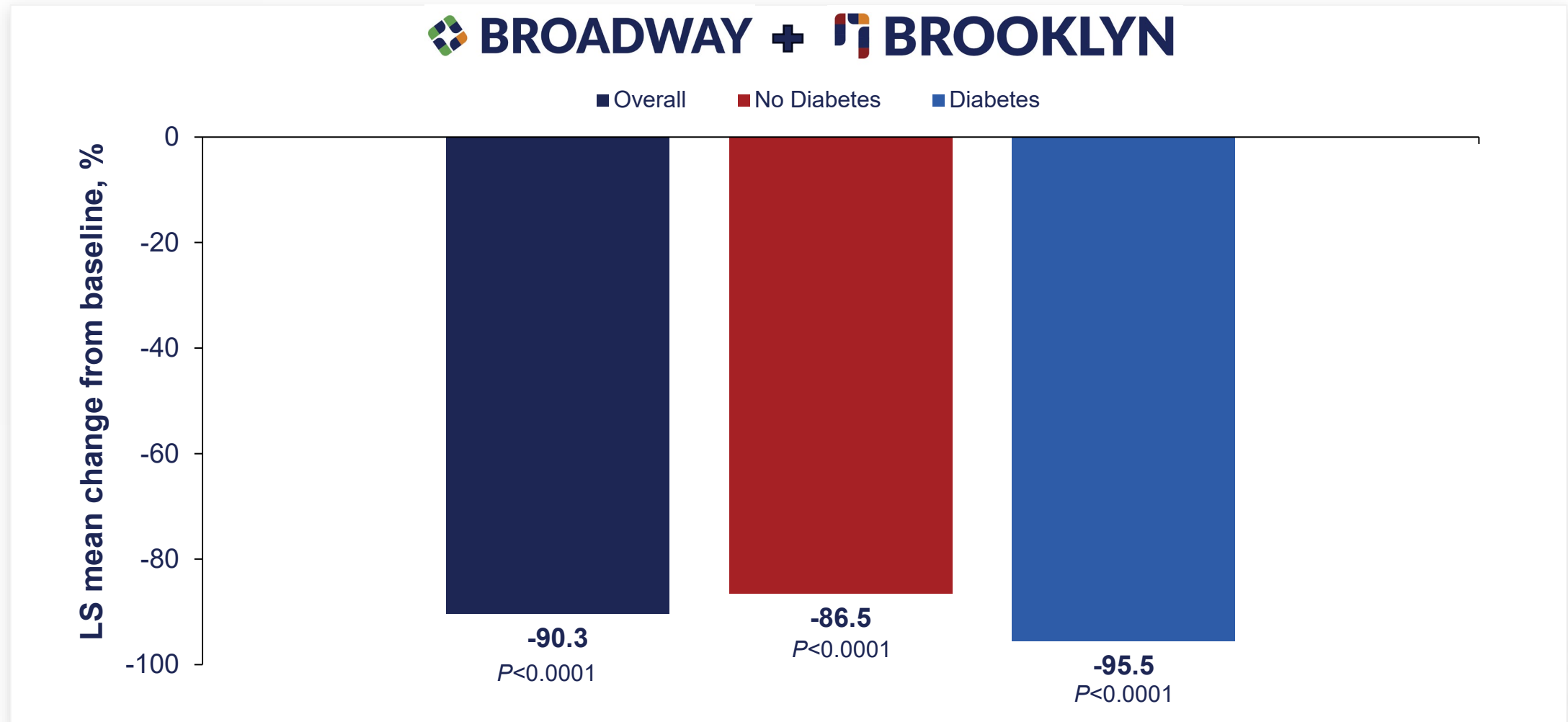
*Includes 9180 individuals with baseline and 12-month measurements.

[†]Differences in change between groups were $P < 0.001$ for all (calculated using the Wilcoxon rank sum test).

CI, confidence interval; LDL, low-density lipoprotein; LDL-P, low-density lipoprotein particle concentration.

Dugani SB et al. *JAMA Cardiol.* 2016;1(2):136-145.

Consistent ~90% Reduction in Small LDL Particles Observed with Obicetrapib in a Pooled Analysis



LDL, low-density lipoprotein; LS, least squares.
Davidson MH et al. *Circulation*. 2025;152(Suppl 3):Abstract 4368890.

**RUBENS Designed to Test the LDL-C
Lowering Effects of Obicetrapib and
Obicetrapib + Ezetimibe FDC in Diabetic
Patient Population**

T2D/Metabolic Syndrome Study

Objective: To evaluate the effect of obicetrapib and the FDC on LDL-C in patients with T2D / Met Syn

Study design: Randomized, double-blind, placebo-controlled

Main Inclusion Criteria:

- ≥ 18 years
- T2D / Metabolic Syndrome
- $\text{LDL} \geq 100 \text{ mg/dL}$
- $\text{TG} \geq 150 < 400 \text{ mg/dL}$
- Guideline recommended LLT

Main Exclusion Criteria:

- Clinical ASCVD
- HoFH
- Uncontrolled hypertension

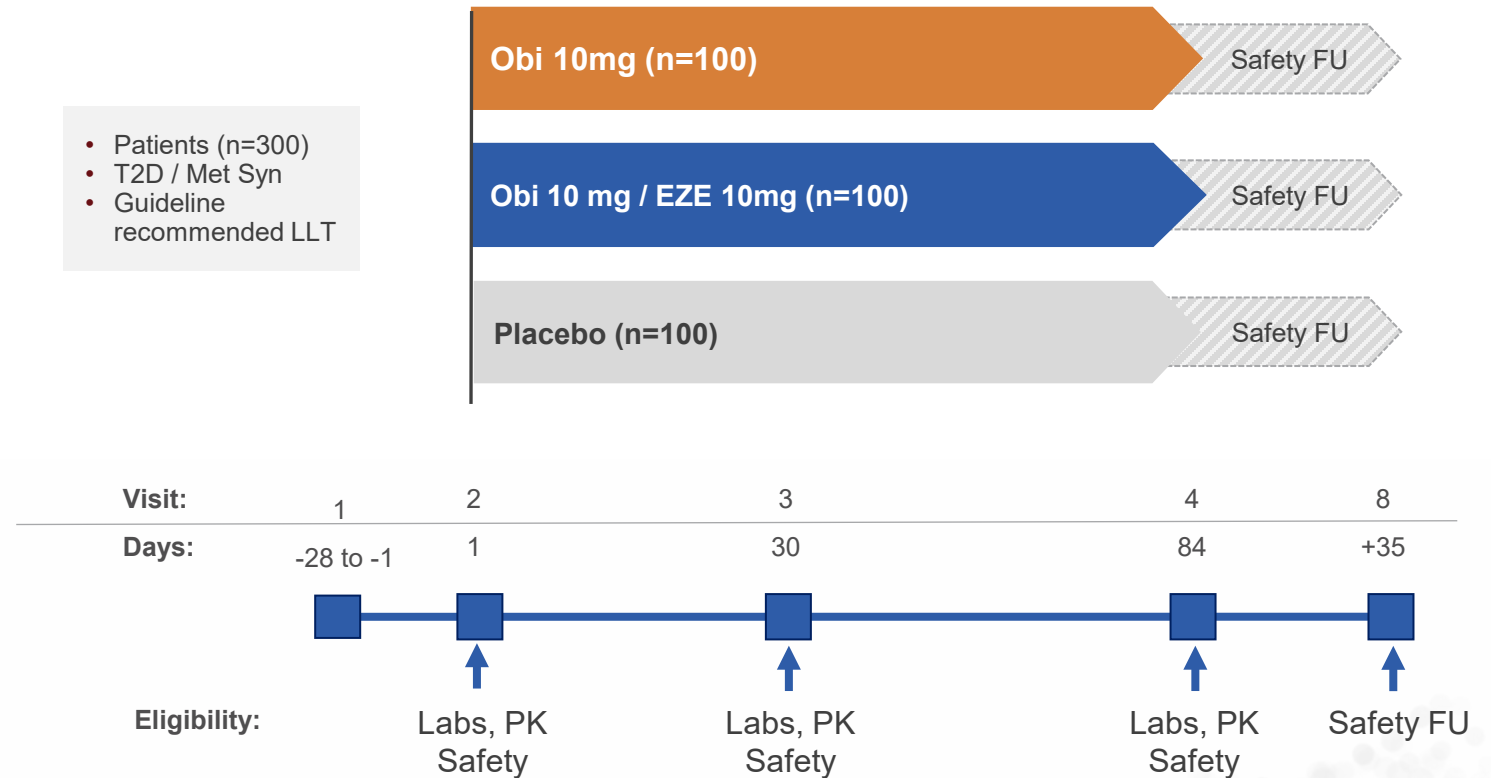
Primary efficacy endpoint

- % LS mean change from BL to Day 84 in LDL-C for obicetrapib & FDC using Martin-Hopkins

Key secondary efficacy endpoint

- LDL-C Goal attainment
- Lp(a), LDL-P, sdLDL

- Patients (n=300)
- T2D / Met Syn
- Guideline recommended LLT



RUBENS Is Designed to Provide Insight into LDL-C and Small Particle Lowering in the Diabetic Population without ASCVD



1

Show obicetrapib and FDC robustly lower LDL-C in T2D/metabolic syndrome patients

2

Demonstrate reduction of atherogenic sdLDL particles and Lp(a)

3

Establish CETP inhibition preserves glycemic control in patients on statin therapy

4

Confirm efficacy and safety in high-risk patients without ASCVD



Large, underserved population: T2D/metabolic syndrome patients not at LDL-C goal despite statin therapy



Positive traction with endocrinologists and diabetes specialists



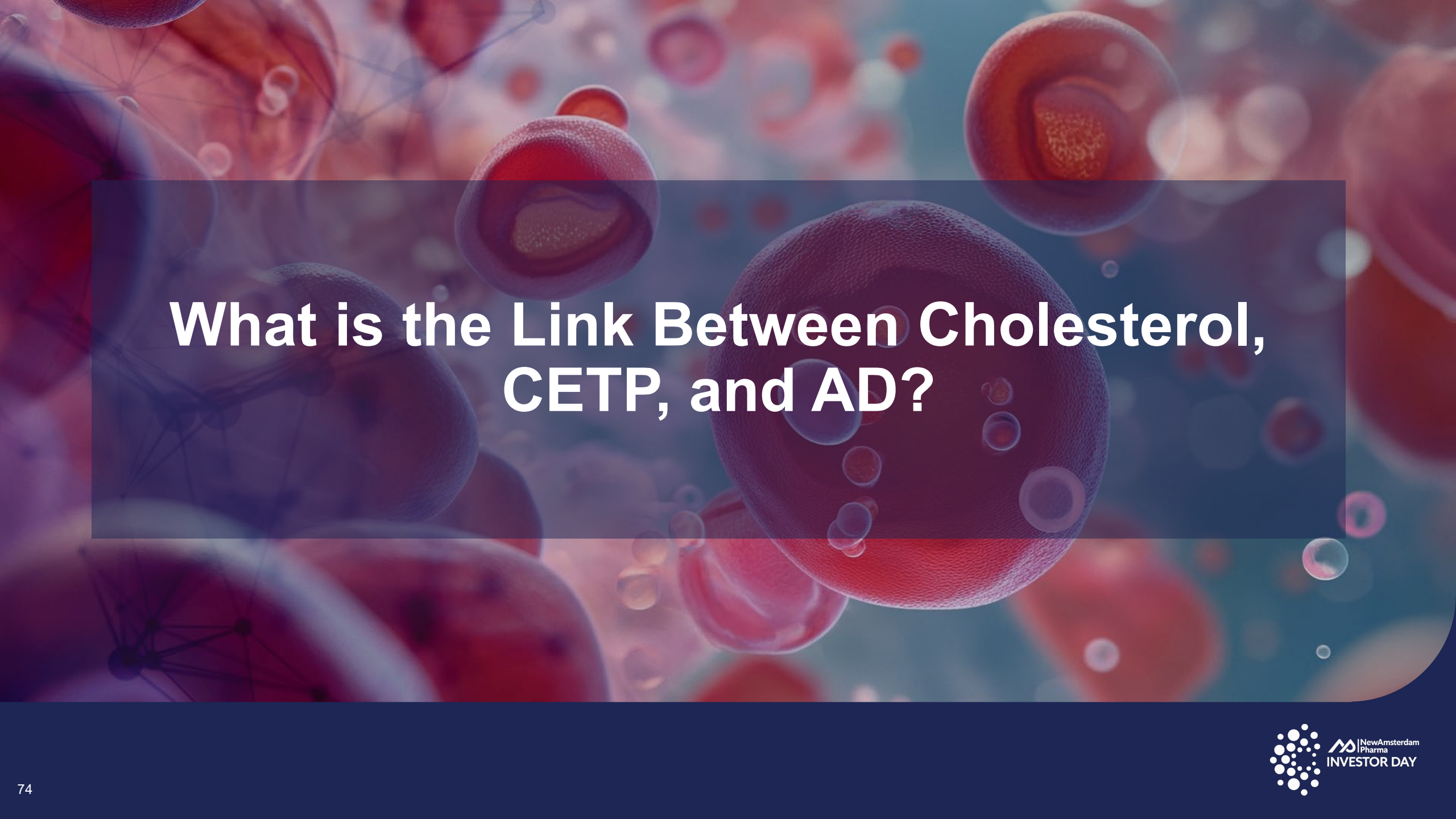
Designed to evaluate changes in LDL-C, sdLDL & Lp(a)



May support LDL-C lowering in a T2D/metabolic syndrome population in the label



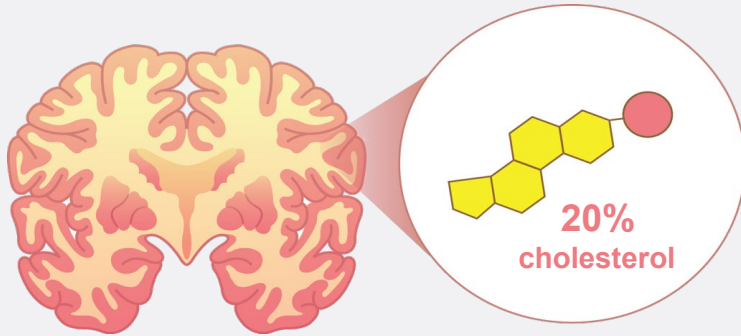
Moving from the Heart to the Brain...



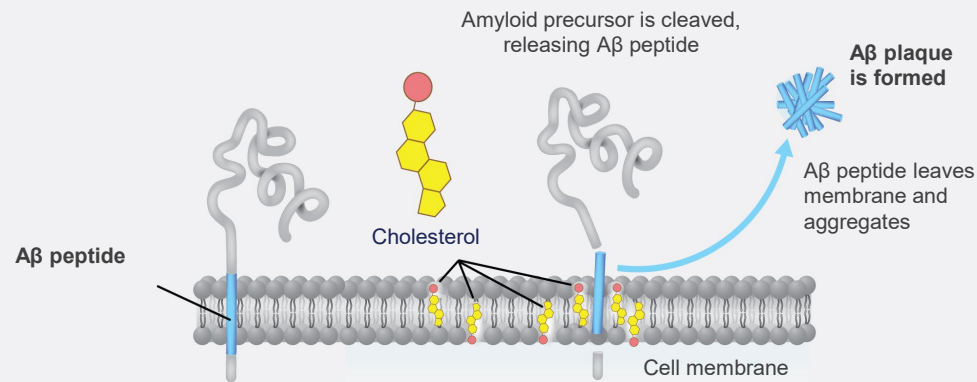
What is the Link Between Cholesterol, CETP, and AD?

Elevated Cholesterol Levels in the Brain Precede Formation of Amyloid-Beta (A β) Plaques

The brain is the most cholesterol-rich organ; it contains only 2% body mass but has 20% of the body's cholesterol



A β plaque formation inside AD brain cells



During early development, oligodendrocytes synthesize large quantities of cholesterol for myelination

In adults, when myelination is complete, glial cells and, to a lesser extent, neurons account for the steady-state production of cholesterol

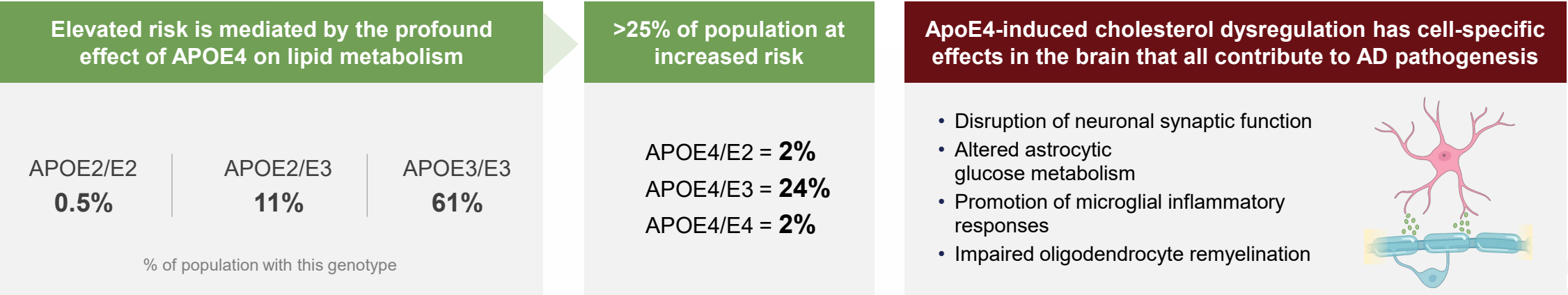
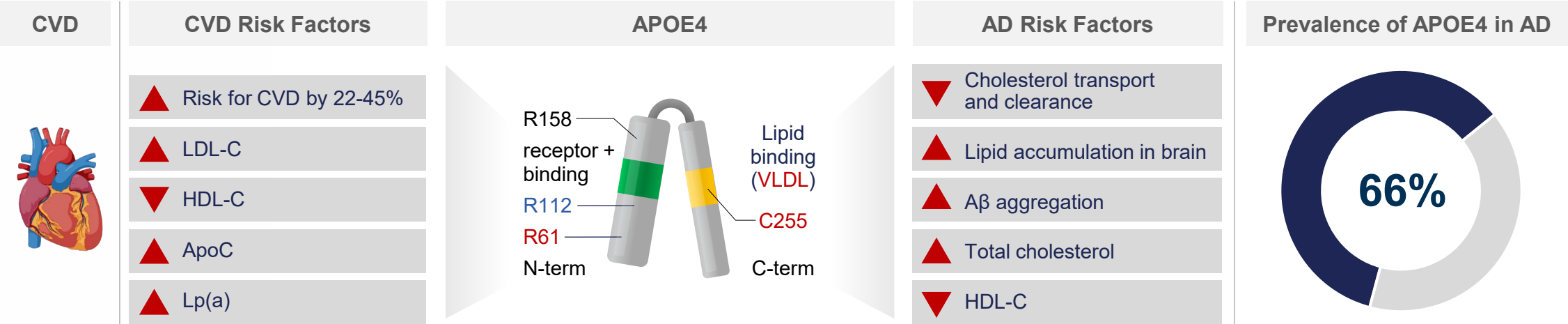
The brain uses a unique mechanism for cholesterol recycling and redistribution

- Involves an ApoE-mediated lipoprotein pathway unique to the CNS whereby cholesterol is turned over
- Excess oxysterols are ultimately delivered to the liver for secretion into the bile

Cholesterol itself does not exit the brain but instead is converted to a metabolite, 24S-hydroxycholesterol

- 24S-hydroxycholesterol is a proxy for excess cholesterol that will be measured in our phase 2a clinical trial

ApoE4 is a Risk Factor for Both CVD and Alzheimer's Disease¹⁻⁵

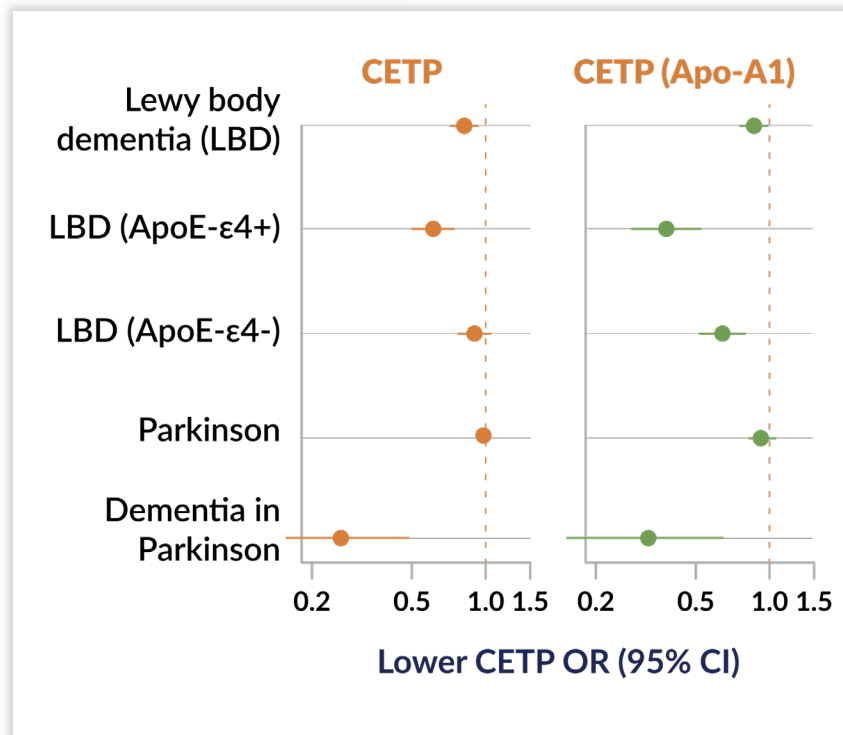


Aβ, amyloid β; AD, Alzheimer's disease; Apo, apolipoprotein; CVD, cardiovascular disease; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; Lp(a), lipoprotein(a). 1. Abondio P et al. *Genes (Basel)*. 2019;10(3):222. 2. Feringa FM et al. *Front Aging Neurosci*. 2021;13:690372. 3. Hottman DA et al. *Neurobiol Dis*. 2014;72(A):22-36. 4. Jeong W et al. *Mol Cells*. 2019;42(11):739-746. 5. Williams HC et al. *Neurobiol Dis*. 2020;136:104742. 6. Li Y et al. *BMC Cardiovasc Disord*. 2024;24(1):353. 7. Fernandez CG et al. *Front Aging Neurosci*. 2019;11:14. 8. Moriarty PM et al. *Arterioscler Thromb Vasc Biol*. 2017;37(3):580-588. 9. Xu M et al. *Biomed Res Int*. 2016;2016:3912175. 10. Mattsson N et al. *Alzheimers Dement*. 2018;14(7):913-924.

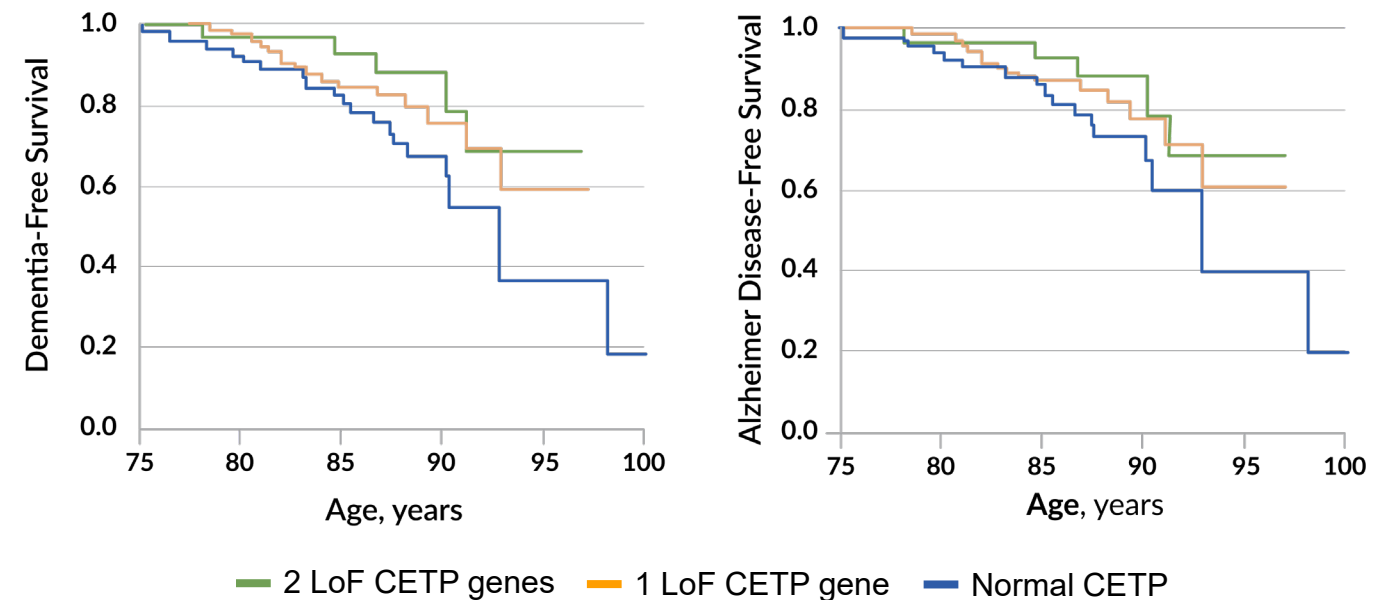
CETP Loss-of-Function (LoF) Genotype May be Associated with Slower Memory Decline and Lower AD Risk

 CETP's potential involvement in CNS cholesterol homeostasis is supported by genomic data¹

 CETP LoF may be associated with lower CETP activity & a corresponding increase in HDL levels²

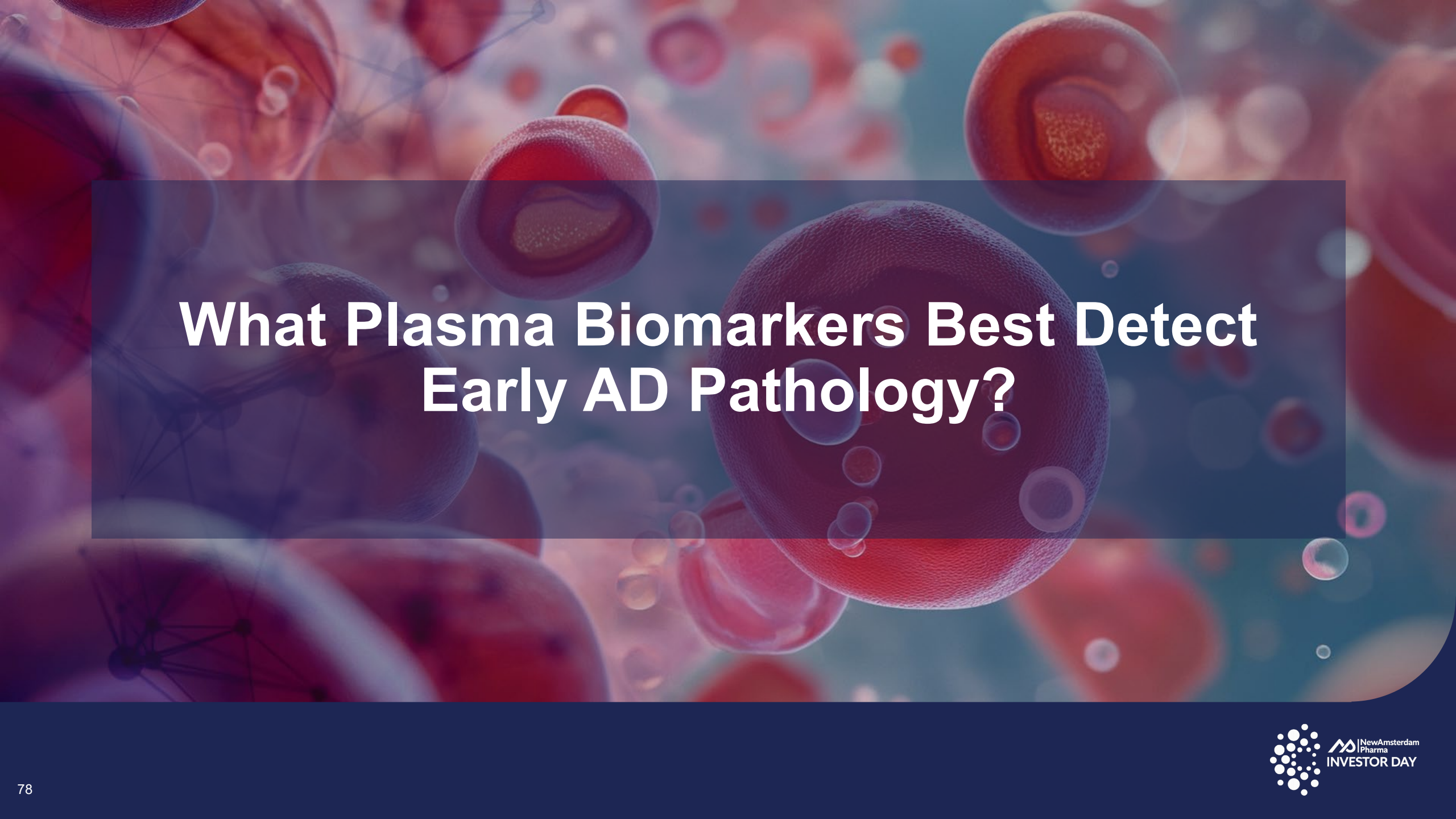


Incidence of Dementia and Alzheimer's Disease by CETP Genotype Group²



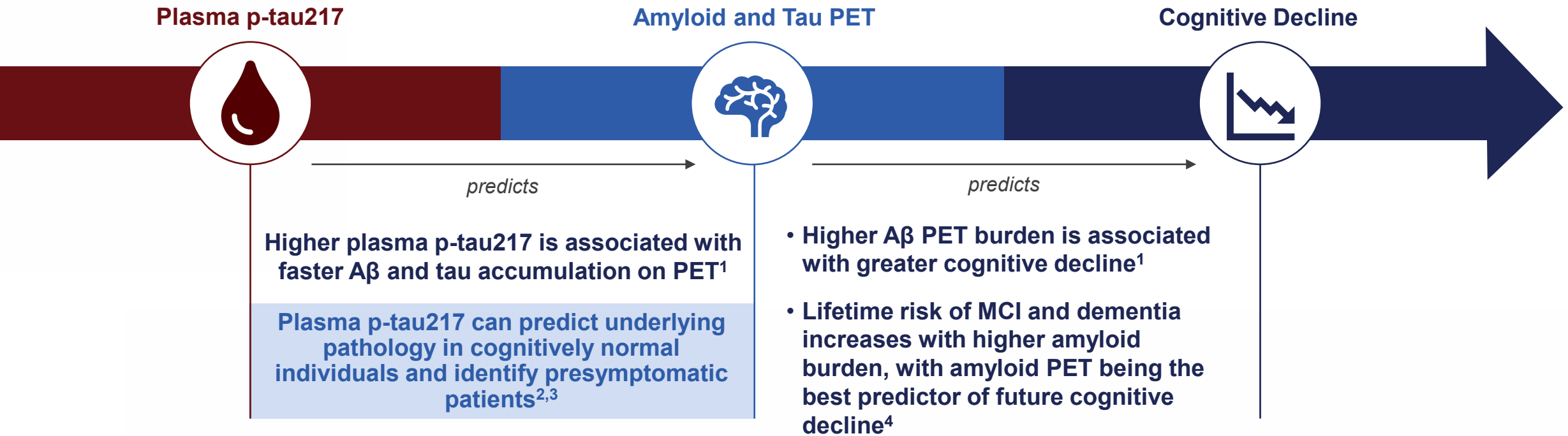
AD, Alzheimer's disease; CETP, cholesteryl ester transfer protein; CNS, central nervous system; HDL, high-density lipoprotein.

1. Schmidt AF et al. *Alzheimers Res Ther.* 2024;16(1):228. 2. Sanders AE et al. *JAMA.* 2010;303(2):150-158.



What Plasma Biomarkers Best Detect Early AD Pathology?

Evidence Supports Plasma p-tau217 as a Predictive Biomarker of Amyloid PET, Which Is Predictive of Cognitive Decline



MCI, mild cognitive impairment; PET, positron emission tomography.

1. Yang HS et al. *Nat Commun.* 2026;17(1):3188. 2. Jonaitis EM et al. *Brain Commun.* 2023;5(2):fcad057. 3. Schindler SE et al. *Alzheimers Dement.* 2024;20(11):8074-8096.

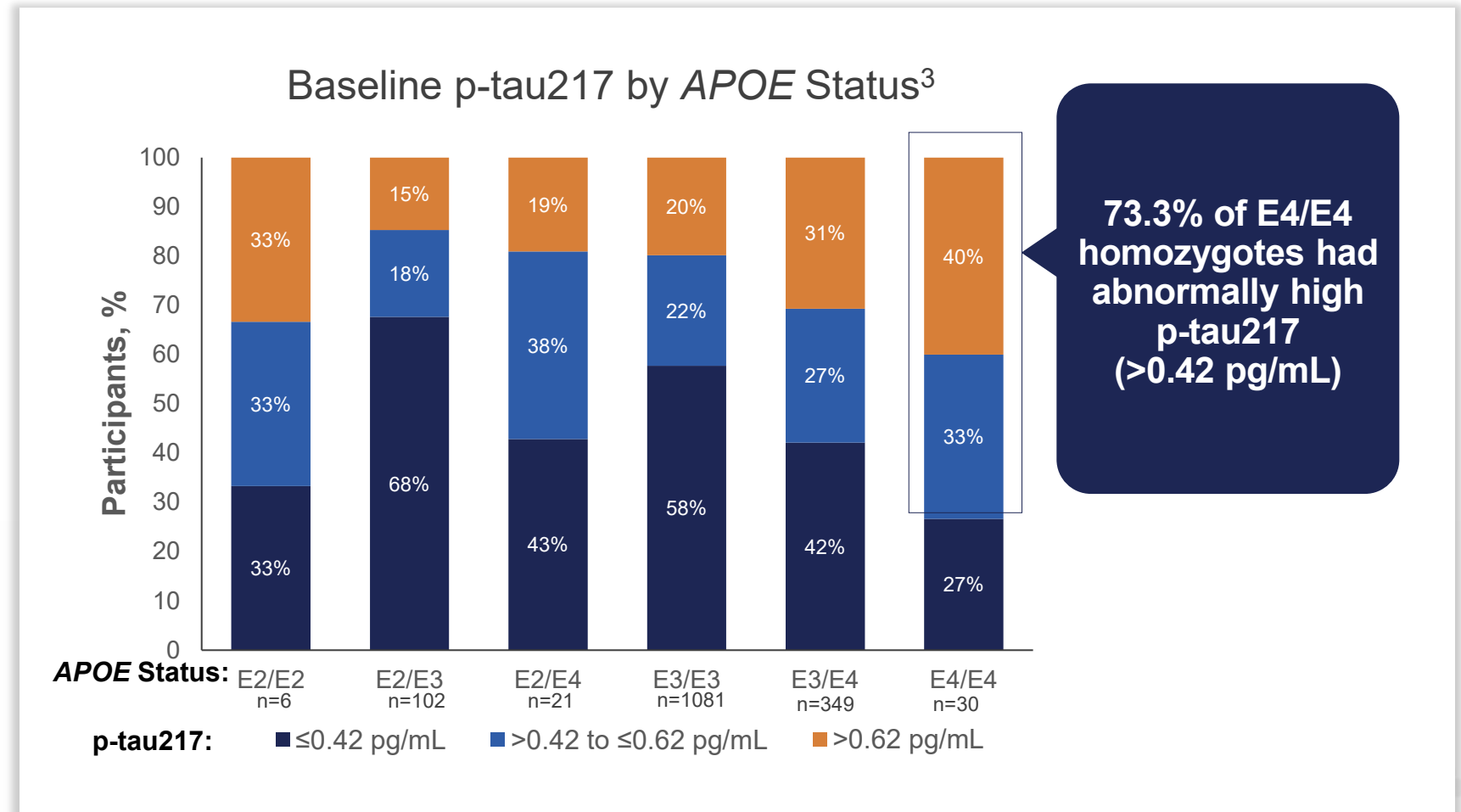
4. Jack CR et al. *Lancet Neurol.* 2025;24(12).

What Did we Discover in the BROADWAY Data?

Patients With ASCVD Carry Undiagnosed AD Pathology, as Measured by p-tau217 Levels¹⁻³

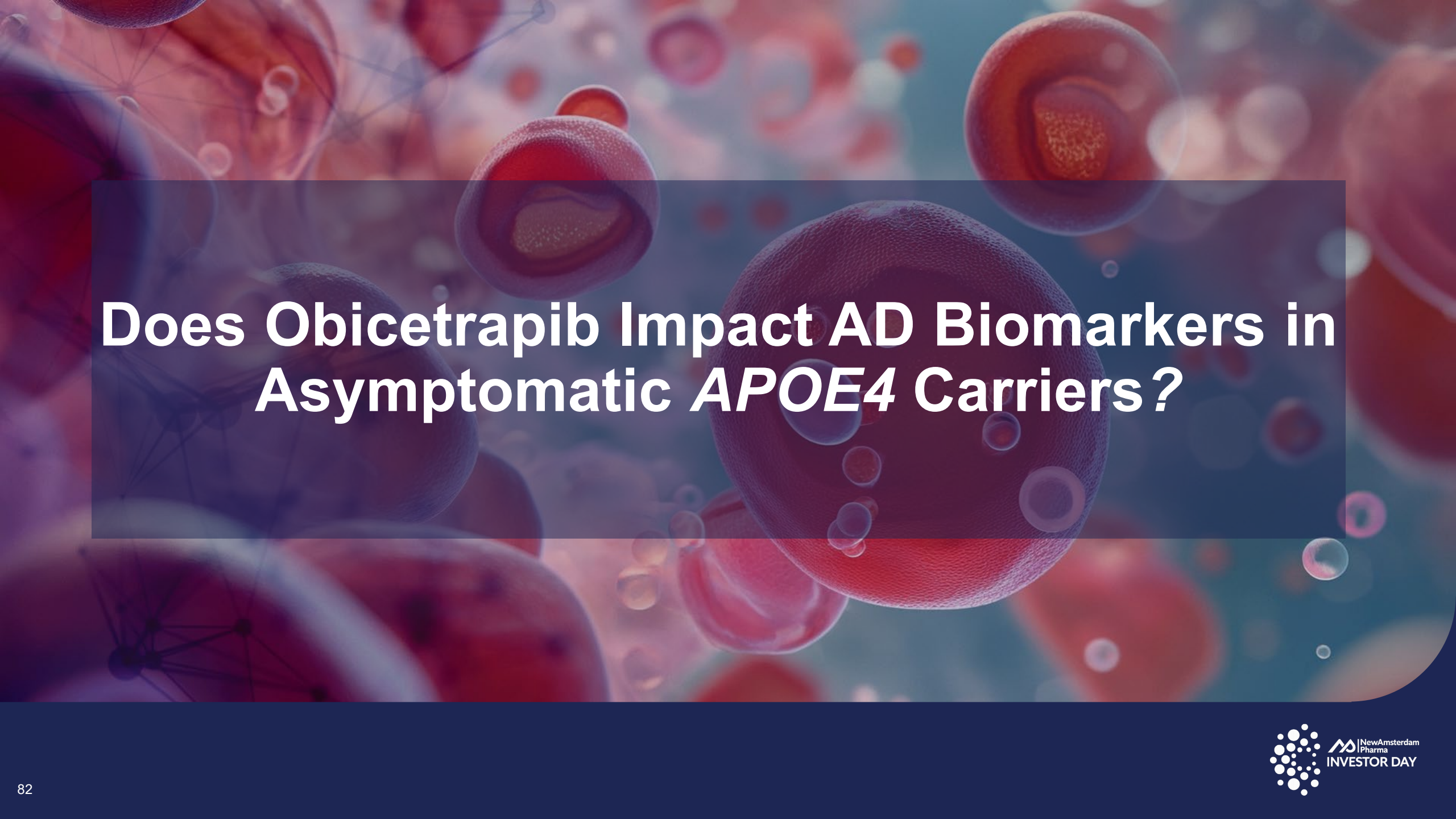


In the BROADWAY AD Biomarker Substudy of 1535 adults with ASCVD, nearly half (45.9%, n=730) had abnormally high p-tau217 levels (>0.42 pg/mL), indicating preclinical AD pathology³



AD, Alzheimer's disease; APOE, apolipoprotein E; ASCVD, atherosclerotic cardiovascular disease.

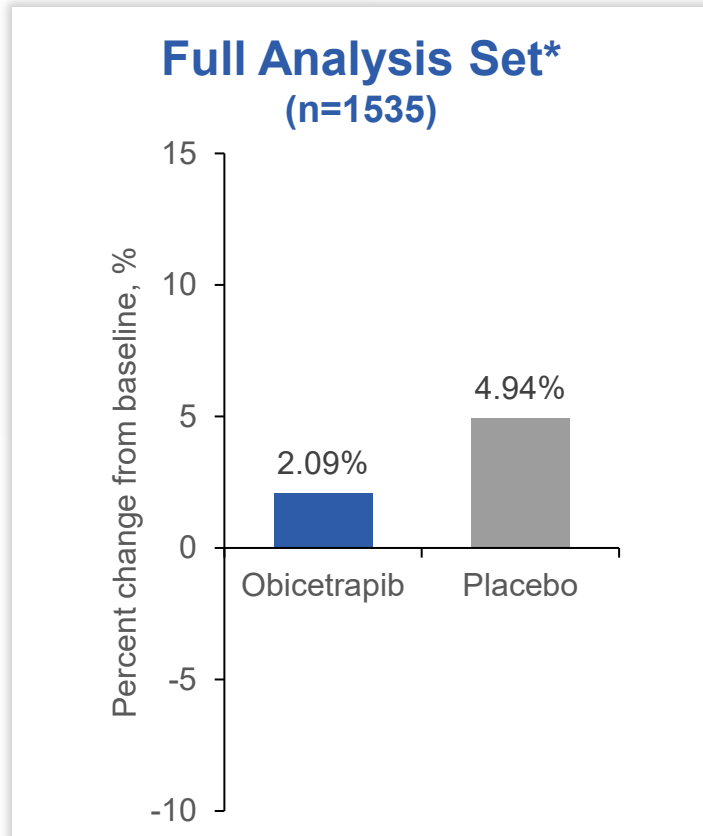
1. van Nieuwkerk AC et al. *Stroke*. 2023;54(8):2181-2191. 2. French SR et al. *Alzheimers Dement*. 2025;21(8):e70565. 3. Kastelein JJP et al. Poster presented at the Alzheimer's Association International Conference 2026. July 12-15, 2026. London, UK.



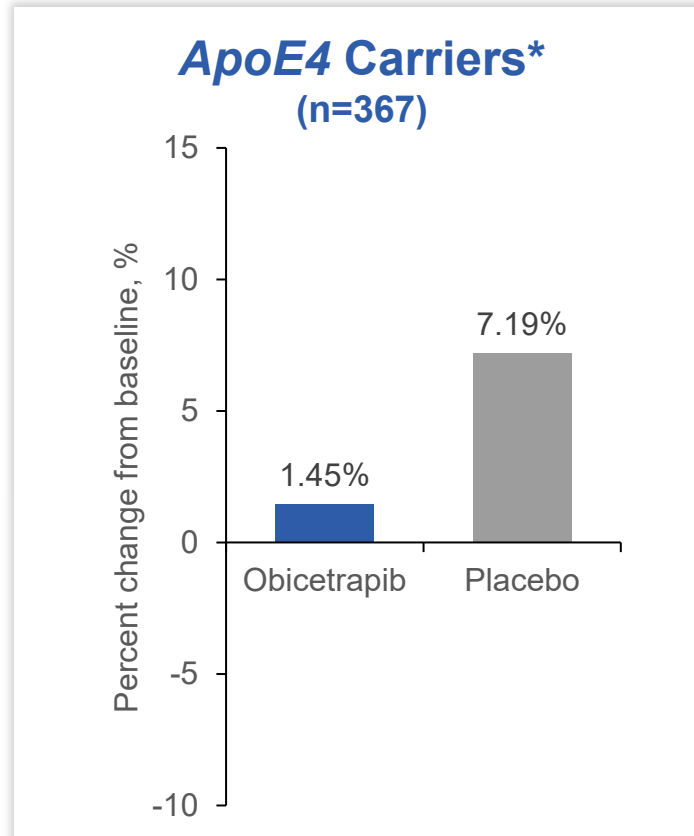
Does Obicetrapib Impact AD Biomarkers in Asymptomatic *APOE4* Carriers?

Significant Reduction in p-tau217 Progression Over 12 Months Observed with Greater Reductions as ApoE4 Risk Factors Increase

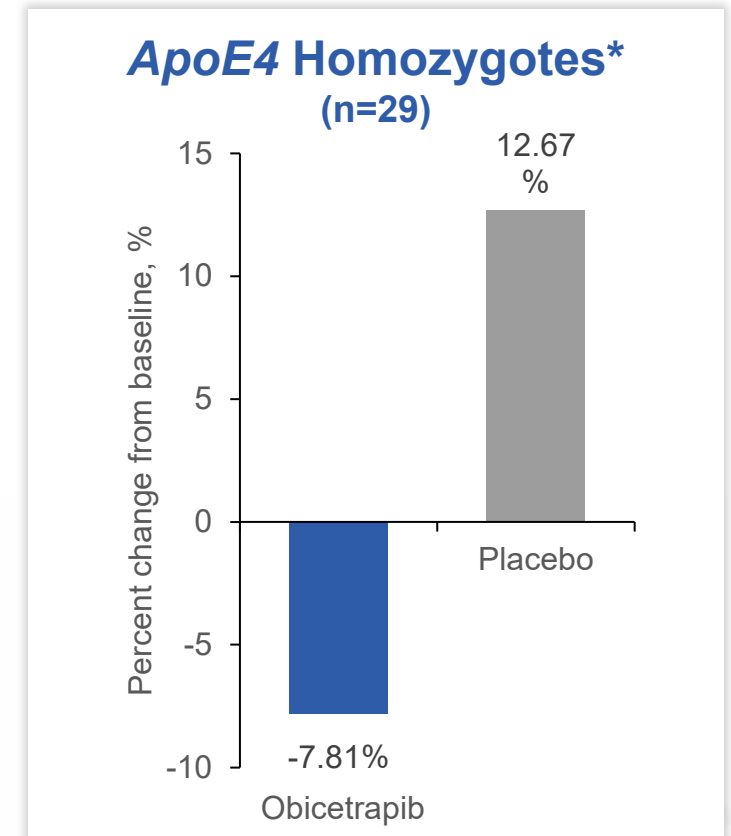
2.84% benefit between obicetrapib and placebo ($P=0.025$)



5.74% treatment benefit in ApoE4 carriers ($P=0.022$)



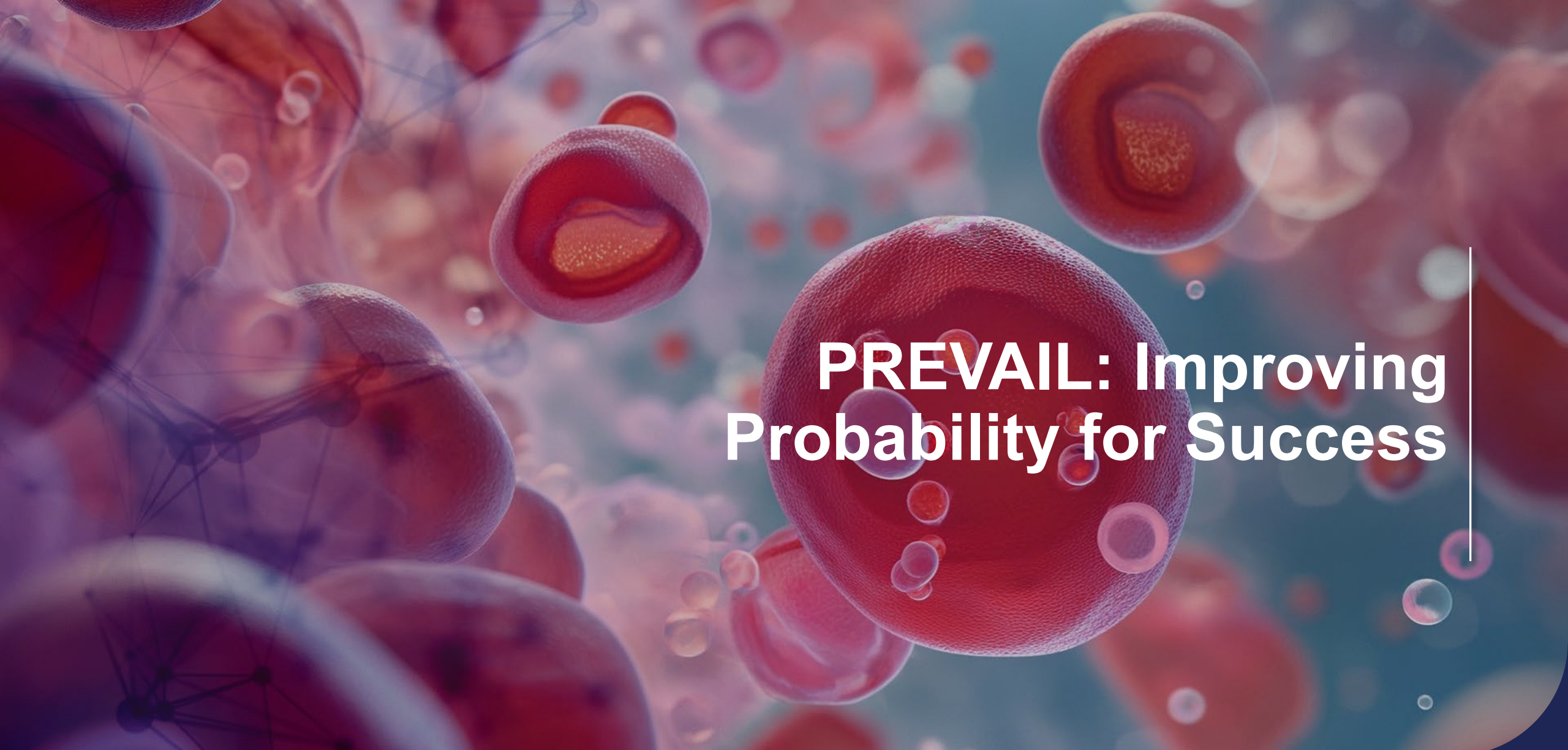
20.5% treatment benefit in ApoE4 homozygotes ($P=0.010$)



*Results reflect adjustment for mean-centered baseline p-tau217 and mean-centered age. Participants with known ApoE status (based on phenotypic analysis), baseline p-tau217 above the lower limit of quantification (LLQ), and available end-of-study p-tau217 were included in the current analyses.

ApoE, apolipoprotein E; p-tau, phosphorylated tau. ApoE4 carriers defined as ApoE3/E4 or ApoE4/E4, based on phenotypic analysis

Davidson MH et al. 2025. Journal of Prevention of Alzheimer's Disease



PREVAIL: Improving Probability for Success

Setting Up PREVAIL for Success

How has our knowledge **MATURED?**

PREVAIL Initiated 2021

- Private Company
- Limited Funding

BROADWAY

- 21% Observed Reduction in MACE

TODAY

- Public Company
- Guideline Updates
- CHMP Recommendation
- \$683M in cash

2021

2022

2023

2024

2025

2026

PREVAIL Designed to Apply Lessons Learned from Previous CVOTs to Demonstrate Obicetrapib's Full Benefit



Greater LDL-C lowering activity anticipated
Targeting higher baseline LDL-C patients



Higher *absolute* LDL-C reduction expected to lead to greater MACE benefit



Longer duration of follow up
Targeting higher-risk patient population



Maximizes opportunity for MACE reduction



Differentiated secondary endpoints

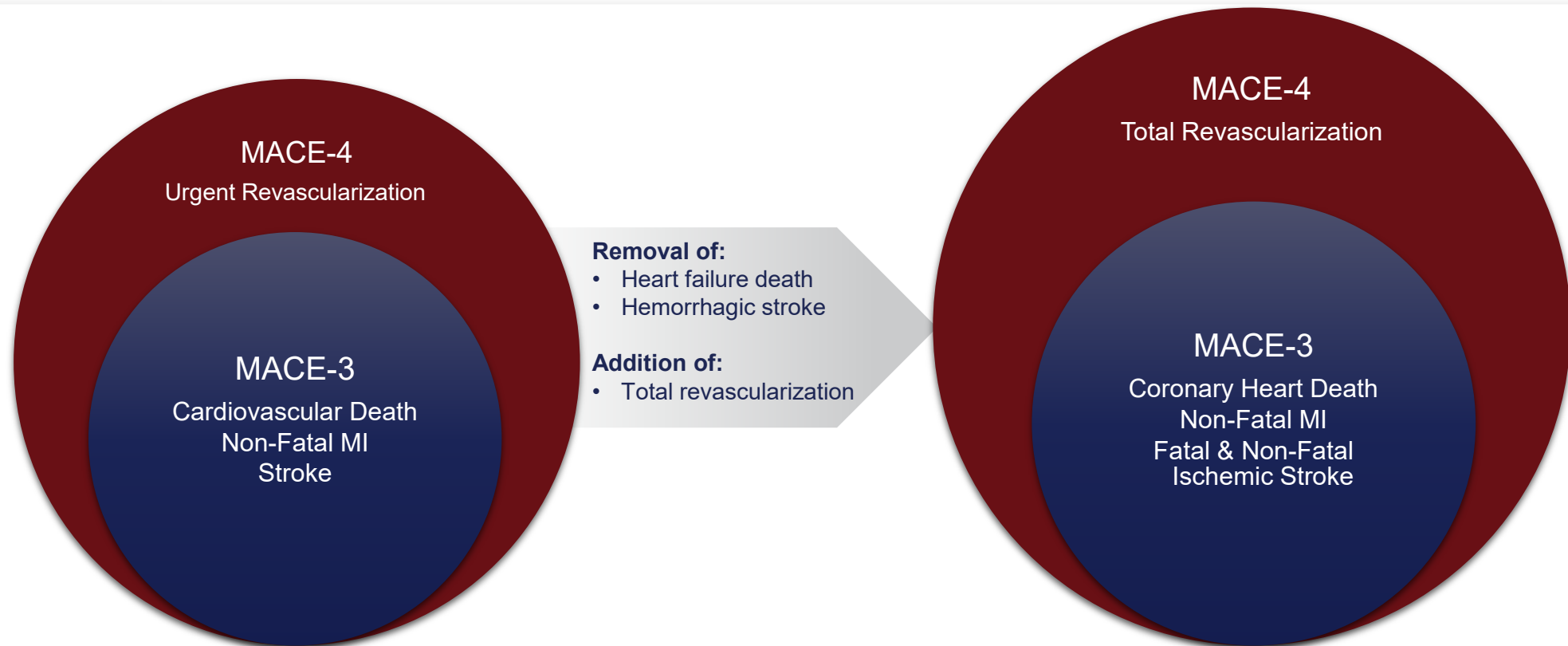


Potentially enhanced commercial profile vs. other LDL-C lowering agents

Primary endpoint selection

Coronary heart disease (CHD) death, non-fatal MI, fatal and non-fatal ischemic stroke, coronary revascularization

Changes in MACE-3/MACE-4 in-line with CTT Definition and Focused on LDL-C Modifiable Endpoints



MACE-4: Urgent/non-elective coronary revascularization updated to total coronary revascularization, given minimal difference in MACE reduction between the two definitions; but by utilizing total coronary revascularization the event number and overall study power increases

PREVAIL Probability of Success and Study Power Increased

	PREVAIL at Initiation	At Interim Analysis	At end of 2027
Estimated Study Completion	YE 2026/1Q 2027	YE 2026/1Q 2027	YE 2027
Primary Event Number	~950	>950	>>950
Median Follow Up	~3.5 years	~3.5 years	~4.5 years
Minimum Follow Up	~2.5 years	~2.5 years	~3.5 years
Expected Time to Majority of MACE benefit ⁽¹⁾	2.0-2.5 years	2.0-2.5 years	2.0-2.5 years

PREVAIL study extension to the end of 2027 driven by slowing event rates plus desire to power the primary MACE-4 endpoint down to 15% through the addition of an additional one year of event accrual

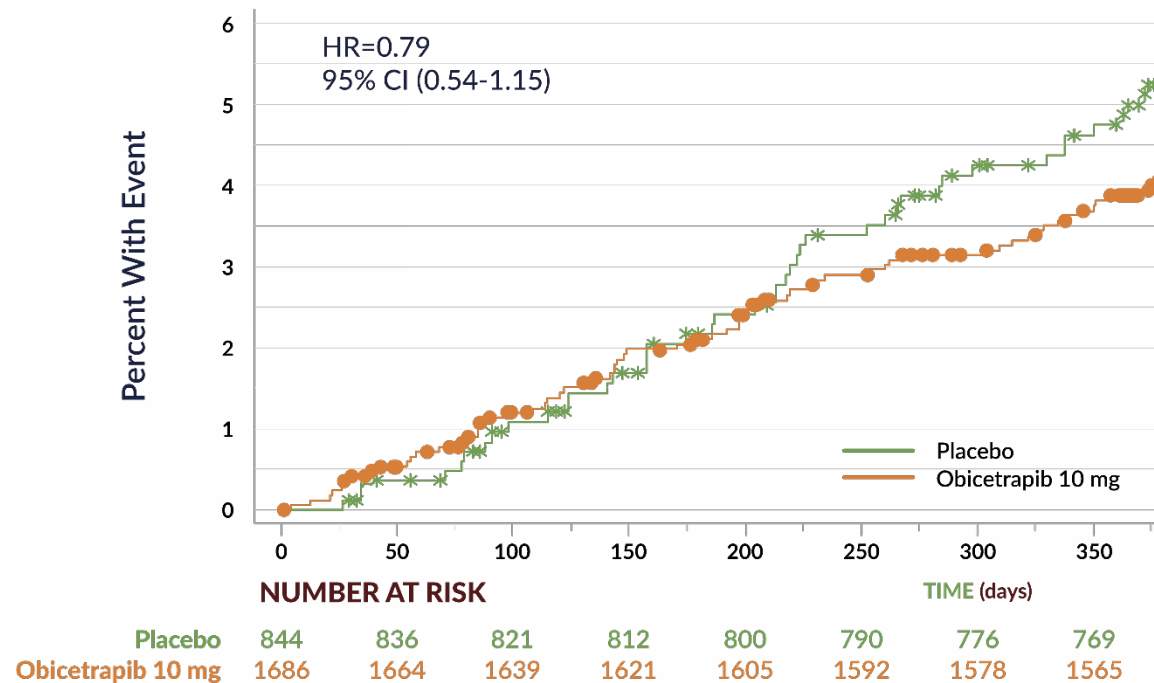
Interim analysis allows the opportunity to enter the market a year earlier, without jeopardizing efficacy or needed alpha for primary endpoint

1. Based on internal analysis

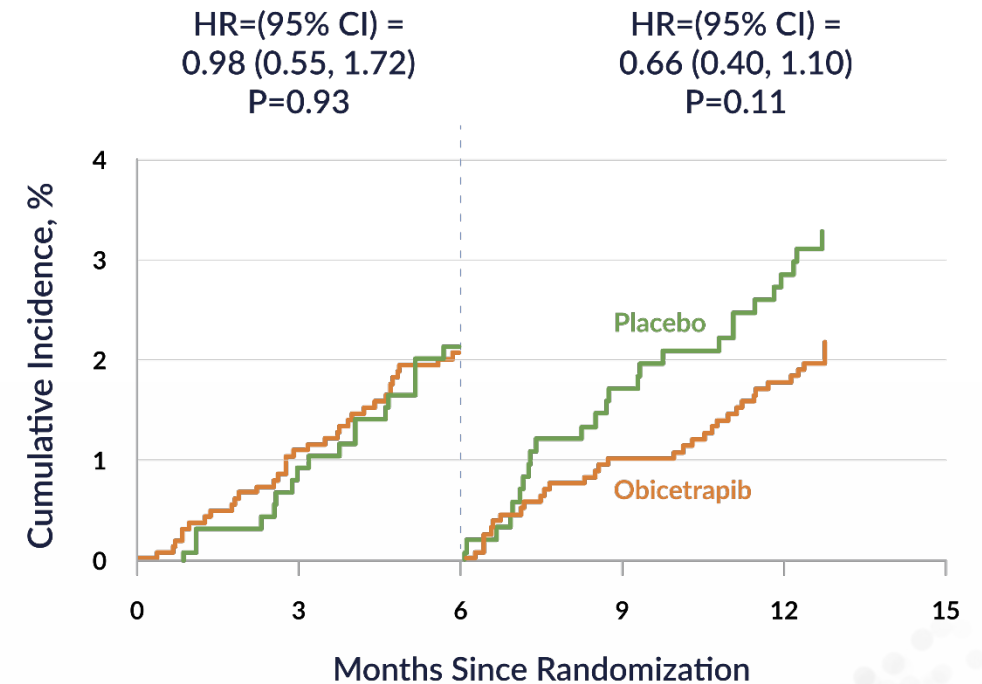
Kaplan-Meier Curve Separates at Day 200 Driven by Attenuation of MACE-4 Events in the Obicetrapib Arm



Kaplan Meier Curve



Landmark Analysis: Censored at 6 months



4-point MACE: CHD death, Non-fatal myocardial infarction, non-fatal stroke, coronary revascularization

While not powered to detect a MACE-4 benefit, we observed positive MACE-4 data as part of our review of the exploratory endpoint. BROADWAY was not powered to measure MACE-4 benefit and the MACE-4 data presented below may not be predictive of the MACE data we observe in PREVAIL, which has been designed to measure MACE benefit.



MACE Benefit Observed Across Multiple Components After 1 Year of Treatment

BROADWAY MACE Data⁽¹⁾

	Placebo N = 844	Obicetrapib N = 1686	Hazard Ratio	95% CI
All-cause mortality – no. (%)	12 (1.4)	19 (1.1)	0.83	(0.40-1.71)
First 4-point MACE – no. (%)	44 (5.2)	70 (4.2)	0.79	(0.54-1.15)
Coronary heart death – no. (%)	5 (0.6)	8 (0.5)	0.80	(0.26-2.44)
Nonfatal MI – no. (%)	11 (1.3)	20 (1.2)	0.91	(0.44-1.90)
Stroke – no. (%)	5 (0.6)	14 (0.8)	1.39	(0.50-3.86)
Coronary revascularization – no. (%)	34 (4.0)	49 (2.9)	0.72	(0.46-1.11)

4-point MACE: CHD death, Non-fatal myocardial infarction, non-fatal stroke, coronary revascularization

(1) MACE was evaluated in BROADWAY as an exploratory endpoint. There are limitations on the interpretation of MACE data derived from both BROOKLYN and BROADWAY studies given they were not designed or powered to assess MACE. MACE data presented above may not be predictive of the MACE data we observe in PREVAIL, which has been designed to measure MACE benefit.

Stroke Benefit Typically Delayed; Contributing in Later Years

Year/Outcome	Events (% p.a.)		RR (CI) per 1 mmol/L reduction in LDL-C	
	Statin / more	Control / less		
YEAR 0–1				
Major coronary event	1441 (1.7)	1727 (2.1)		0.84 (0.76 – 0.92)
Coronary revascularization	1886 (2.3)	2150 (2.6)		0.88 (0.80 – 0.97)
Stroke	557 (0.7)	588 (0.7)		0.96 (0.82 – 1.12)
Major vascular event	3497 (4.3)	3952 (4.8)		0.88 (0.84 – 0.93)
YEAR 1–2				
Major coronary event	1009 (1.3)	1256 (1.6)		0.78 (0.70 – 0.86)
Coronary revascularization	997 (1.3)	1282 (1.7)		0.75 (0.67 – 0.84)
Stroke	445 (0.6)	573 (0.7)		0.77 (0.66 – 0.91)
Major vascular event	2112 (2.8)	2645 (3.6)		0.77 (0.73 – 0.82)

p<0.0001

p<0.0001

Meta-analysis of 21 CVOTs demonstrate delayed stroke benefit through LDL-C reductions

LDL-C Studies: Generally Shown to be Predictive of CVOT Outcomes

Concordance results for lipid lowering drugs evaluated

Drug	Sample Size		MACE EVENTS (1st occurrence)				CVOT ¹ MACE HR (95% CI) Non-CVOT MACE RR (95% CI)	Concordance
	N-active drug	N-comparator	Events-Drug	Events-Comparator	% Event Rate-Drug	% Event Rate-Comparator		
Bempedoic Acid-CVOT	6992	6978	819	927	11.71	13.28	0.87 (0.79–0.96)	Concordant
Non-CVOT	2424	1197	117	75	4.83	6.27	0.77 (0.58–1.02)	
Evolocumab-CVOT	13,784	13,780	1344	1563	9.75	11.34	0.85 (0.79–0.92)	Concordant
Non-CVOT	2976	1489	28	30	0.94	2.01	0.47 (0.28–0.78)	
Alirocumab-CVOT	9462	9462	903	1052	9.54	11.12	0.85 (0.78–0.93)	Concordant
Non-CVOT	3182	1792	52	33	1.63	1.84	0.89 (0.57–1.37)	
Bococizumab-CVOT	13,720	13,718	352	397	3.16	3.59	0.88 (0.76–1.02)	Concordant
Non-CVOT	2377	2058	57	55	2.5	2.7	0.90 (0.62–1.29)	

100% of Non-CVOT studies results were concordant with the CVOT outcome*

BROADWAY Designed to be a Predictor of PREVAIL

BROADWAY

1° endpoint – week 12

N = 2532

Obicetrapib 10 mg (2:1 randomization)

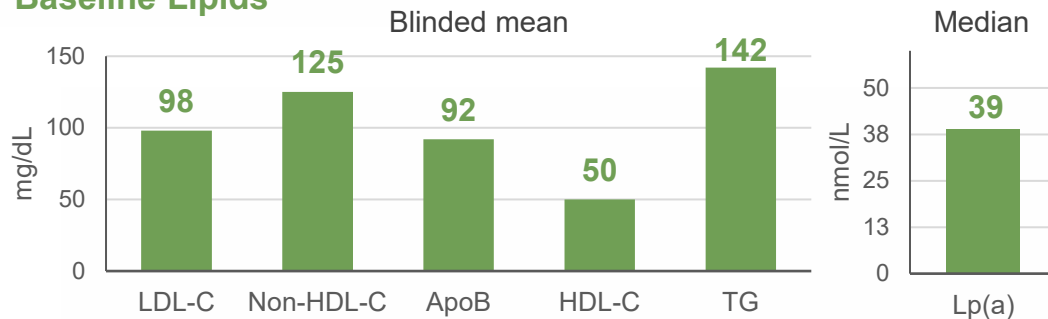
Placebo

13-months

Key Inclusion Criteria

- ASCVD or HeFH
- LDL-C ≥ 100 mg/dL
- LDL-C ≥ 55 mg/dL w/risk factors, or
- Maximally tolerated lipid lowering therapy

Baseline Lipids



Baseline Lipid Modifying Therapy

- Any statin: 91%
- High intensity statin: 65%
- Ezetimibe: 26%
- PCSK9i: 4%
- SGLT2i: 11%
- GLP-1: 6%

PREVAIL

LDL-C endpoint

N = 9541

Obicetrapib 10 mg (1:1 randomization)

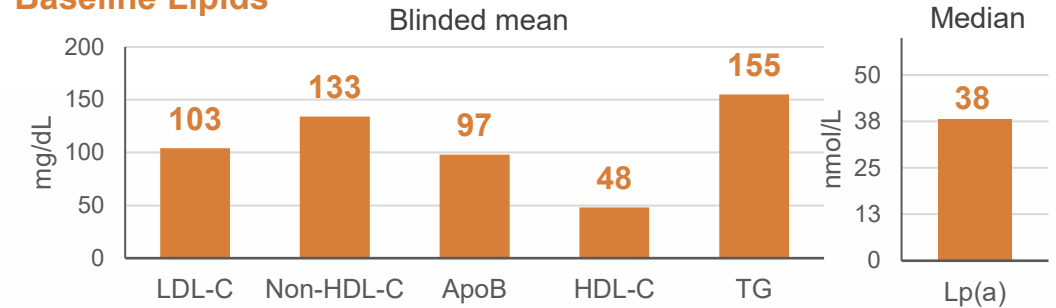
Placebo

54-months

Key Inclusion Criteria

- ASCVD
- LDL-C ≥ 100 mg/dL
- LDL-C ≥ 55 mg/dL w/risk factors, or
- Maximally tolerated lipid lowering therapy

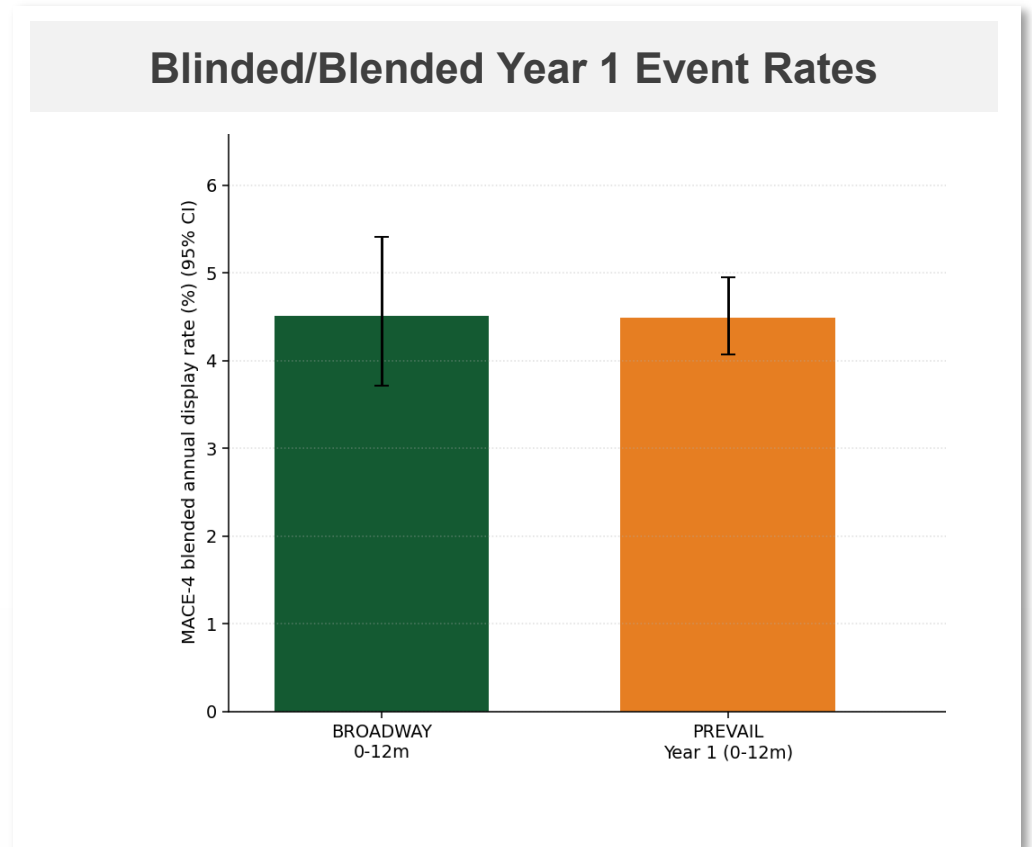
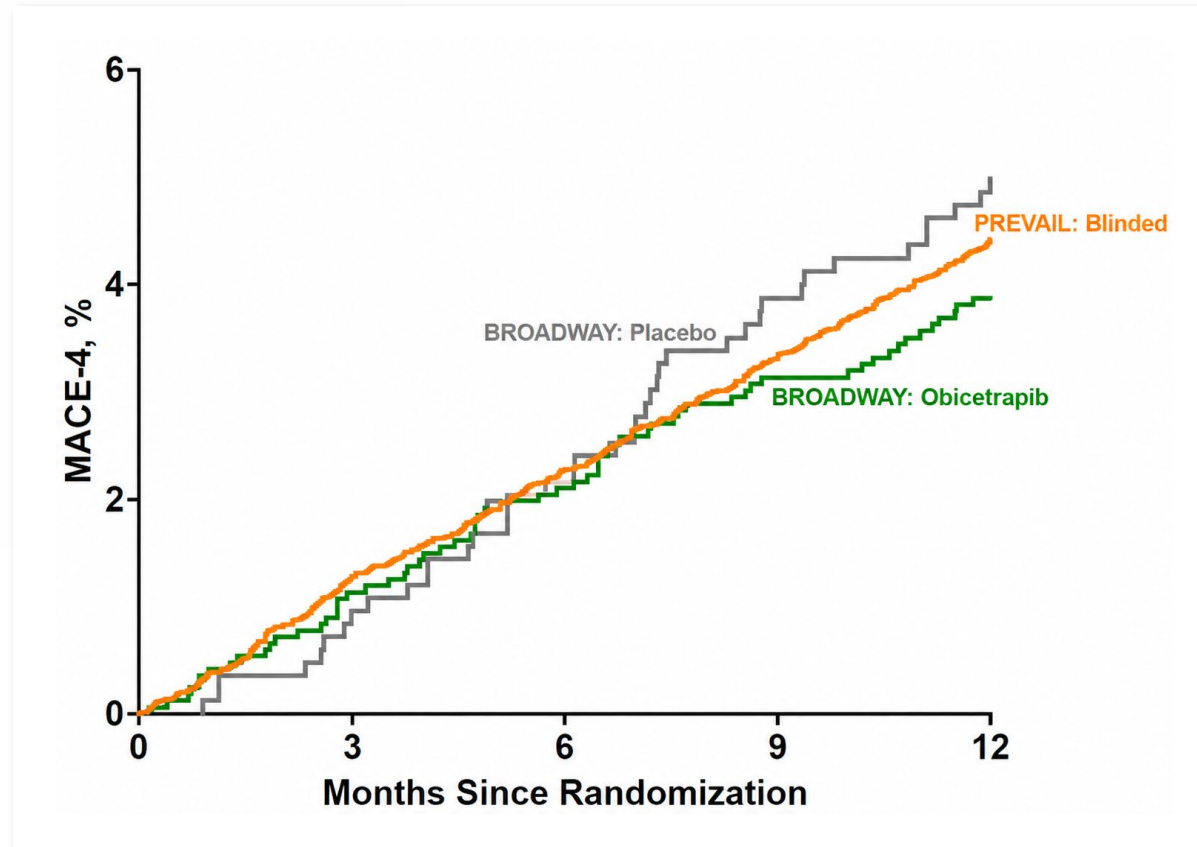
Baseline Lipids



Baseline Lipid Modifying Therapy

- Any statin: >90%
- High intensity statin: 74%
- Ezetimibe: 26%
- PCSK9i: 2%
- SGLT2i: 14%
- GLP-1: 6%

BROADWAY and PREVAIL Year 1 Event Rates - Showing Similar MACE Rates in the Study Populations



4-point MACE: CHD death, Non-fatal myocardial infarction, non-fatal stroke, coronary revascularization

(1) While not powered to detect a MACE benefit, we observed positive MACE-4 data as part of our review of the exploratory endpoint. BROADWAY was not powered to measure MACE benefit and the MACE data presented below may not be predictive of the MACE data we observe in PREVAIL, which has been designed to measure MACE benefit.

PREVAIL data is preliminary and subject to change as MACE events continue to be identified and adjudicated (including with respect to periods presented above). Additionally, Year 1 and Year 2 MACE event trends may not be indicative of future trends.

PREVAIL has a higher or comparable baseline risk vs comparator trials but blinded event rates are trending lower

PREVAIL Blinded Rates vs Contemporary Secondary Prevention CVOT Placebo Rates (2017–2025)

Trial	Year	Median Follow Up (Yr) ²	Age	LDL (mg/dL) ³	Diabetes	Prior MI	Calc. MACE-4 Event Rates ¹	Calc. MACE-3 Event Rates ¹
CLEAR SP Cohort	2023	3.4	65	139	46%	30%	4.60%	3.20%
SOUL	2025	4.1	66	72	100%	100%	3.90%	3.35%
SELECT	2023	3.3 ³	62	78	0%	68%	3.68%	2.41%
FOURIER	2017	2.2	63	92	37%	81%	5.14%	3.36%
ODYSSEY	2018	2.8	59	87	29%	19%	5.57%	3.74%
Average		3.2	63	88	42%	60%	4.58%	3.21%
 PREVAIL	2026	2.78	65	103	49%	50%		
 BROADWAY	2023	1	66	97	38%	57%	5.20%	2.50%

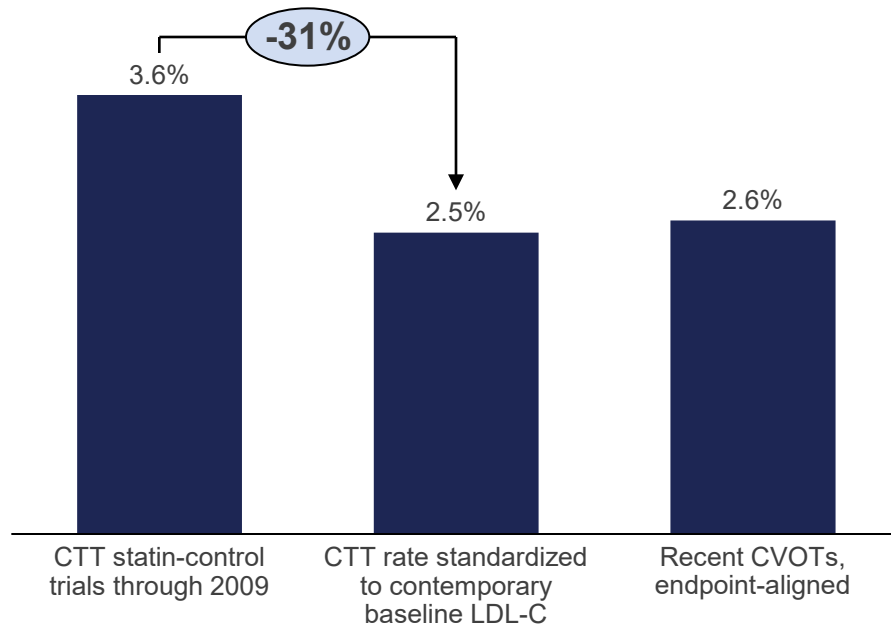
Source: Internal analysis | 1. Sayed et al. International Journal of Cardiology 406 (2024) 132074 2. McGuire et al. N Engl J Med 2025;392:2001-12. 3. Lincoff et al. N Engl J Med 2023;389:2221-32. 4. Sabatine et al. N Engl J Med 2017;376:1713-22. 5. Schwartz et al. N Engl J Med 2018;379:2097-107.

Note: 1. Internal analysis based on annualized placebo rates for comparator trials calculated based on available data; MACE-4 was 4 pt CTT (3pt MACE + total coronary revasc; if not available: then 3 pt + ischemic driven revasc (IDR) or 3 pt + coronary/cerebral revasc (CCR) . 2. Approximate 3. Median values except for PREVAIL, BROADWAY, CLEAR

Results should be interpreted with caution due to inherent limitations in cross-trial comparisons.

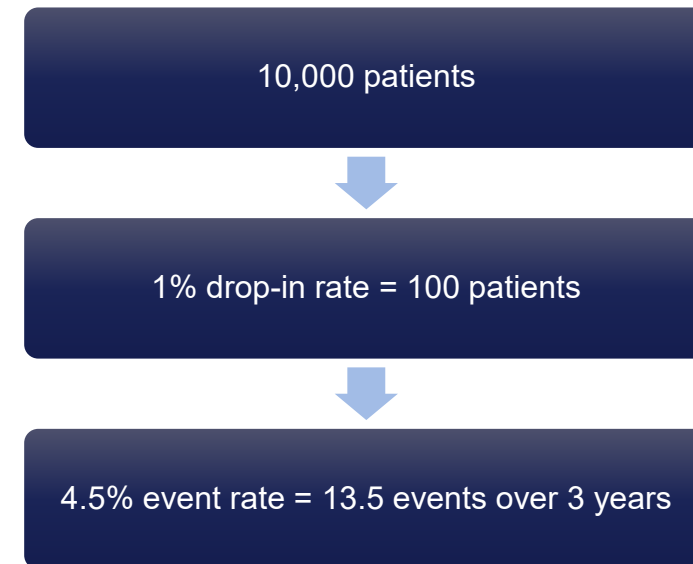
Analysis Suggests Event Rates in CVOT Trials Have Remained Stable When Adjusting for Baseline LDL-C; Impact of Drop-In's Minimal

Has standard of care been reducing event rates?



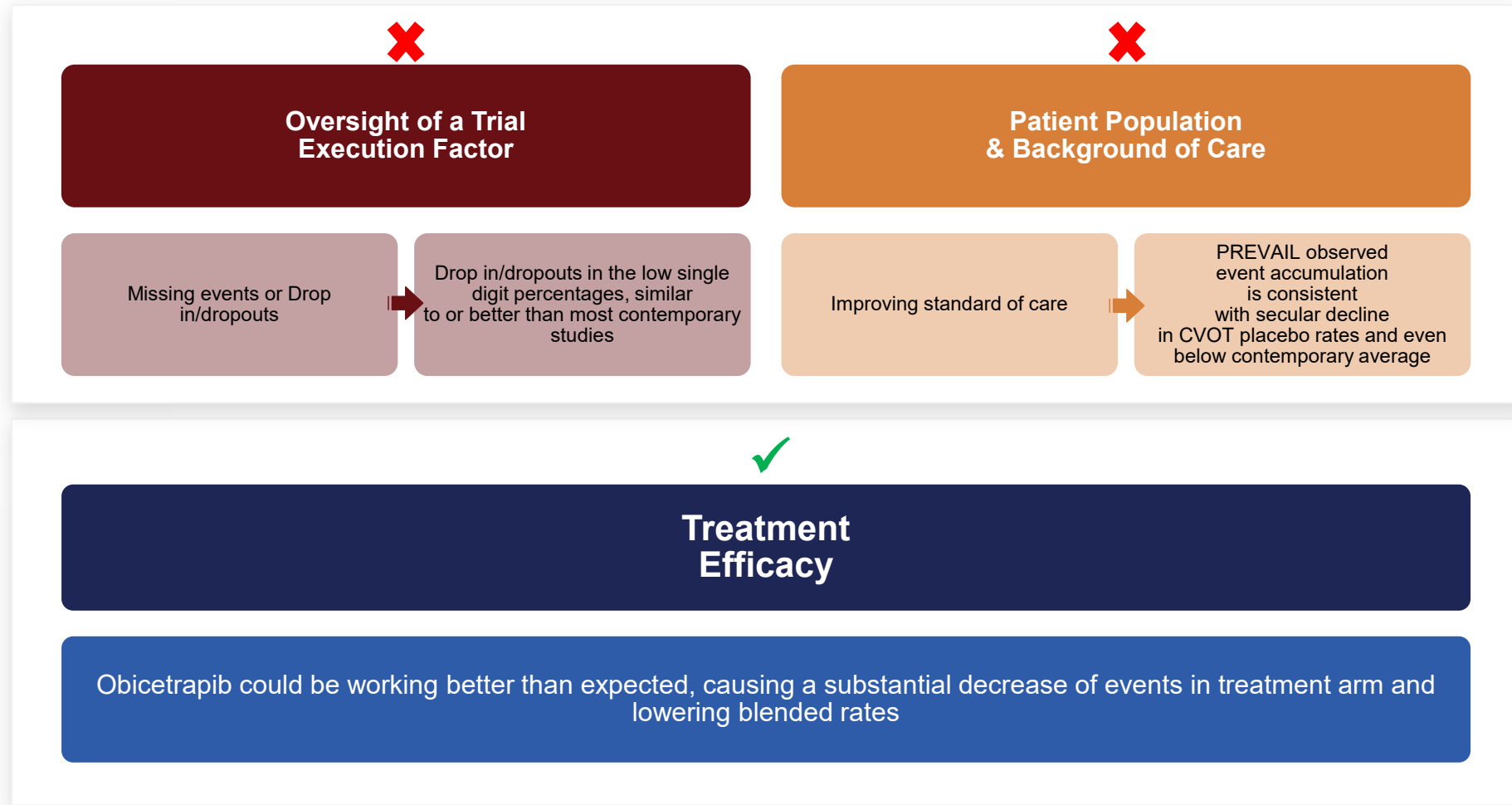
Although novel drug usage is increasing (GLP1, SGLT2, PCSK9, etc.) lower event rates in LDL-C based CVOTs are explainable by lower baseline LDL-C

What effect could drops-in have?



A 1% drop-in rate of a therapy that provides a 20% MACE benefit would only impact event rates by 0.009%, from 4.500% to 4.491%

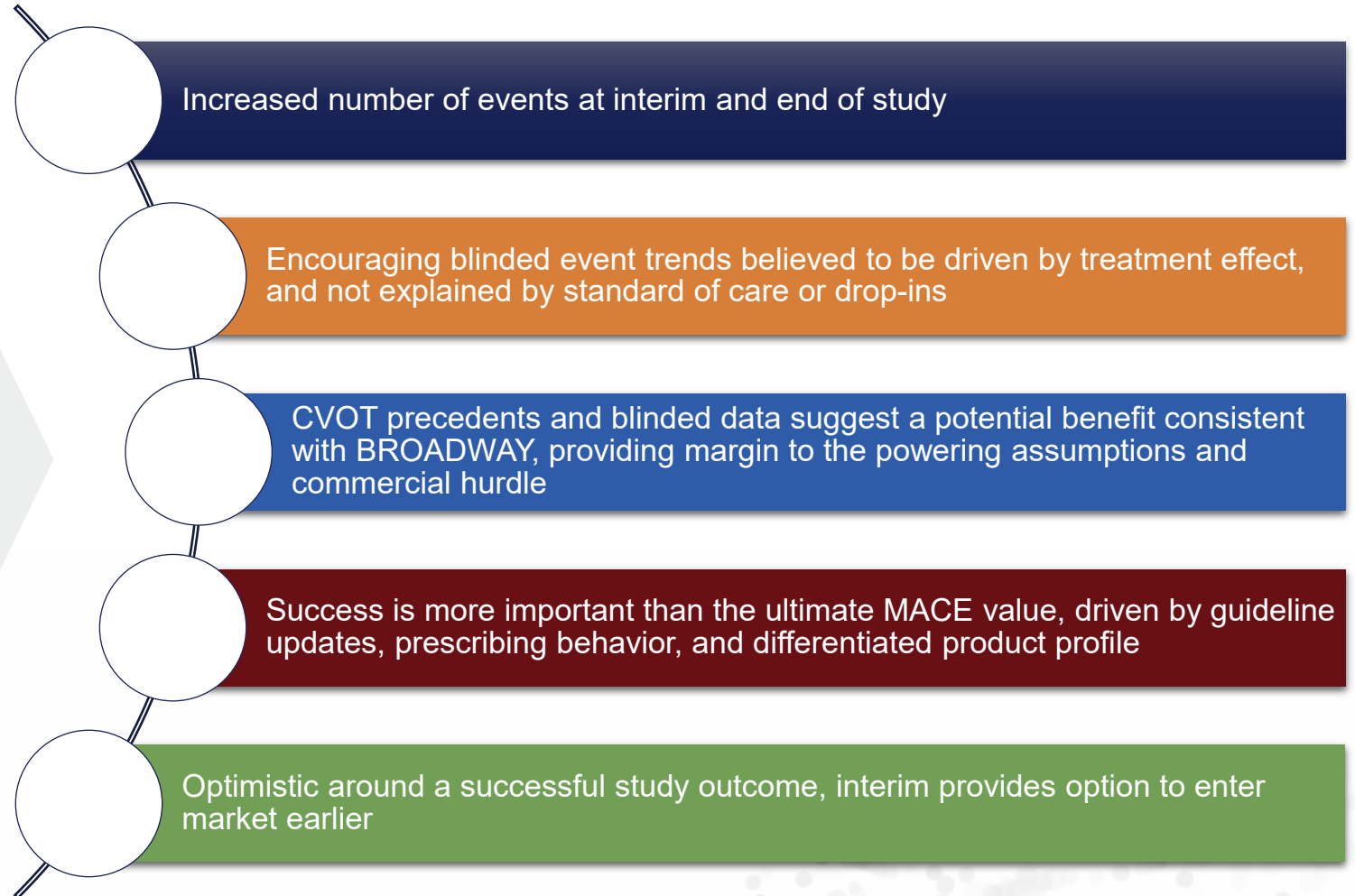
Treatment Effect Believed to be Likely Explanation for Lower Event Rates

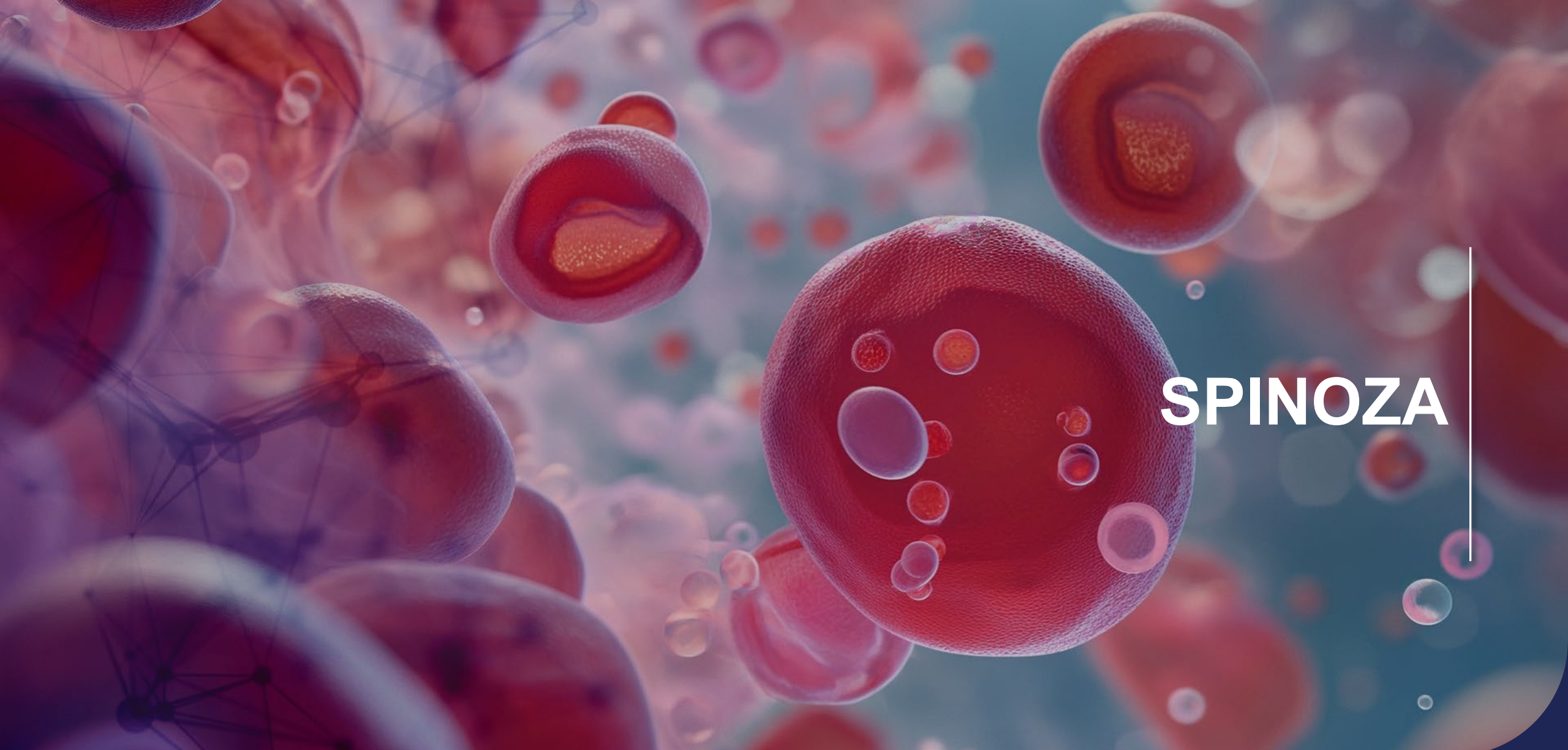


PREVAIL Design Changes Increase Probability of Success



The Study Should
Not Fail the Drug





SPINOZA

A Future Worth Getting Ahead Of

AGE 55 • FEMALE • APOE4 HOMOZYGOTE • FHx ALZHEIMER'S
MEDICATIONS: STATIN • TRIATHLETE & MOUNTAINEER •
PhD, BIOTECH EXECUTIVE



ADVOCATE

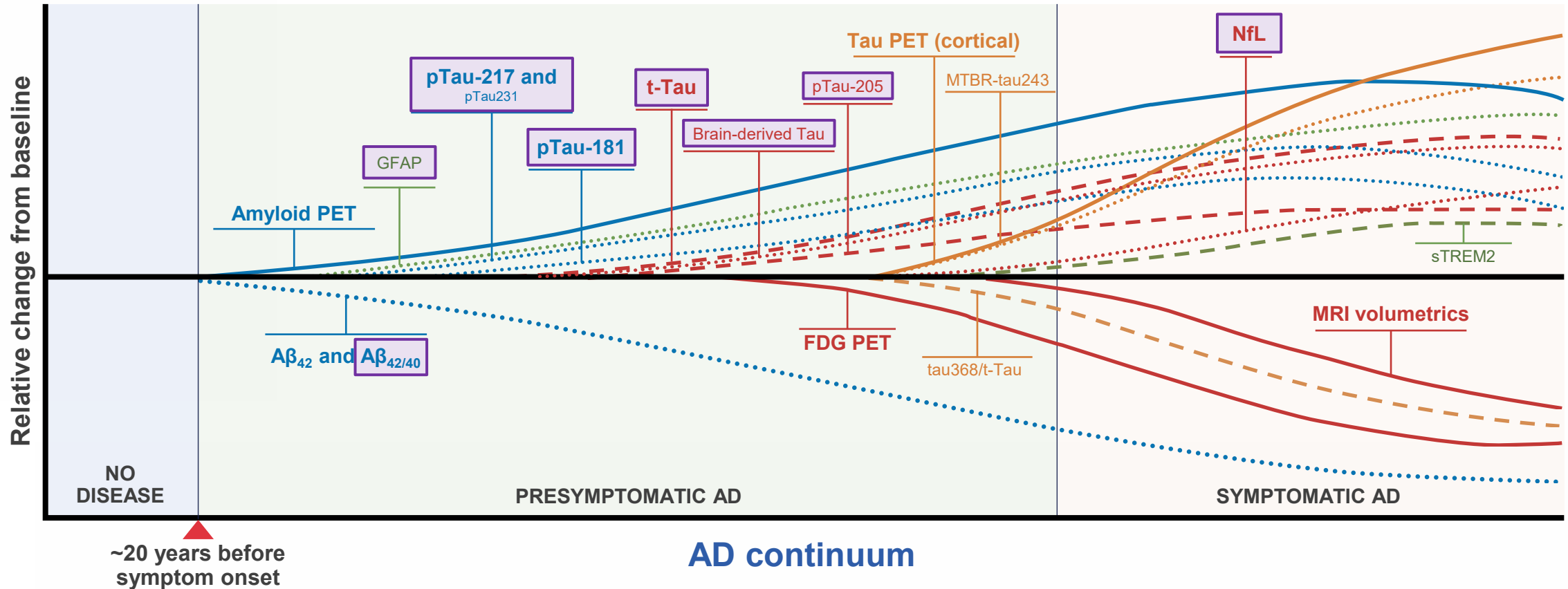
“

I watched Alzheimer's take so much from both my mother and now my father. Living with two copies of APOE4, I can't help but fear that my daughters or I will face the same future. I'm healthy today, but waiting for symptoms is not enough. We need treatment options that enable earlier intervention.

Wendy N.

Living Fully, Still Waiting for Prevention

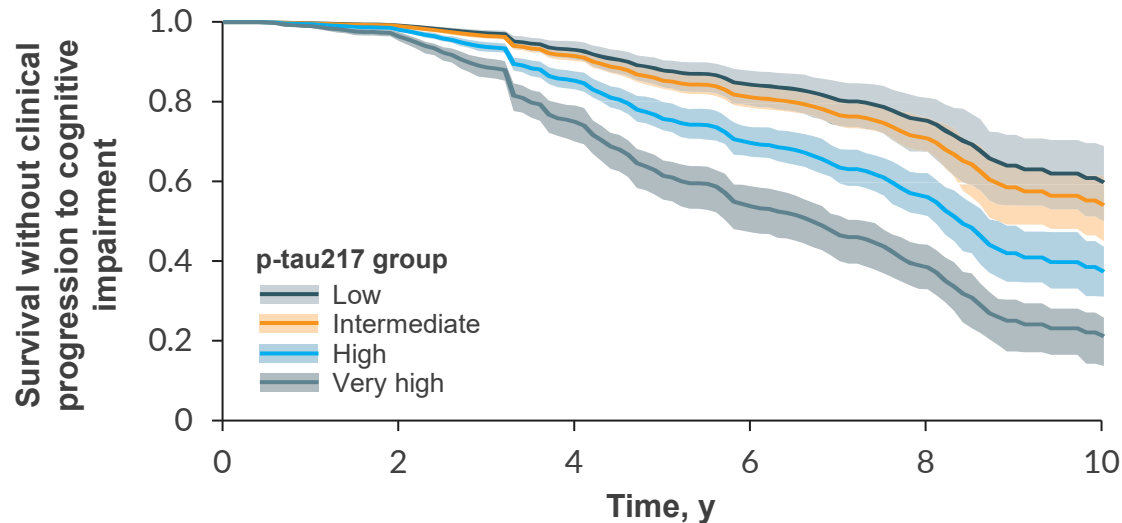
Alzheimer's Disease is Being Redefined as a Biological Process That Begins 20 Years Before Cognitive Decline



TYPES OF BIOMARKER	CLOSEST PATHOLOGIC CORRELATION	STAGE OF DEVELOPMENT	Biomarkers measured as endpoints in obicetrapib AD studies
— Imaging - - - CSF CSF and plasma	Amyloid plaques Neurofibrillary tangles Neuroinflammation Neurodegeneration	CLINICALLY APPROVED Investigational	Biomarkers measured as endpoints in obicetrapib AD studies

Higher Plasma p-tau217 Levels Are Associated With Greater Risk of Progression to Cognitive Impairment in Unimpaired Adults*

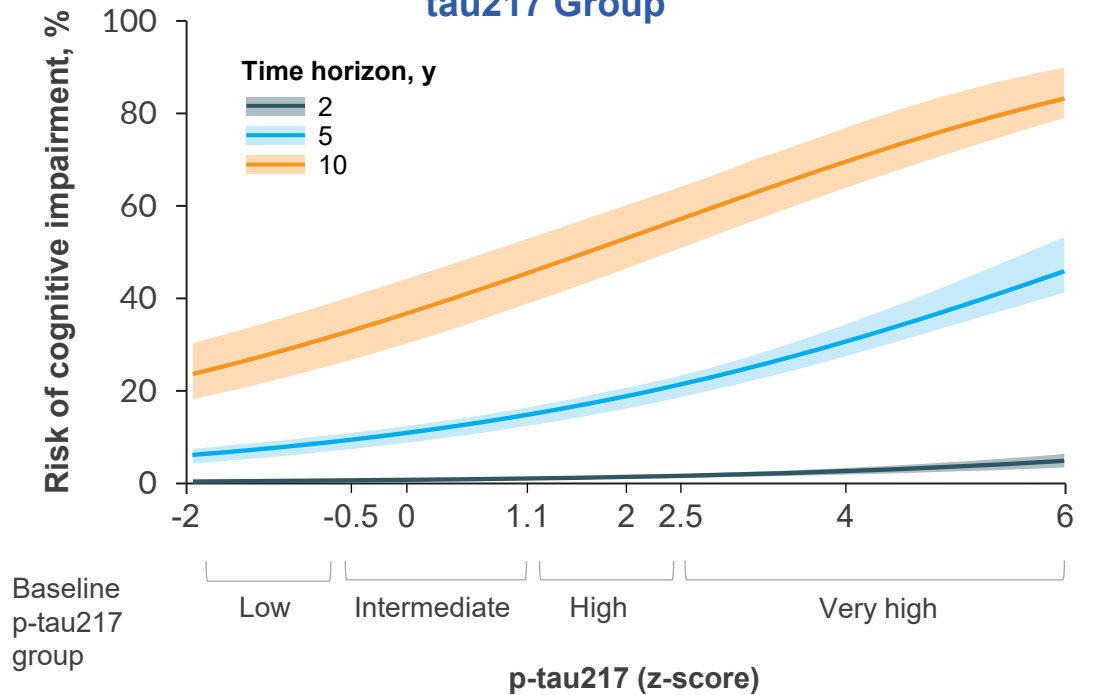
Survival Free of Cognitive Impairment Over Time, by Baseline Plasma p-tau217 Group



No. of participants at risk

Low	516	451	296	197	53	19
Intermediate	1087	983	625	404	139	65
High	598	520	358	195	49	16
Very high	483	425	285	143	27	11

High and Very High Levels of p-tau217 Are Associated With the Greatest Risk of Progression Relative to the Low p-tau217 Group



An increase of 1 SD in p-tau217 was associated with an
HR for progression to cognitive impairment of 1.38 (95% CI, 1.30-1.46; $P < 0.001$)

*Study included 2684 cognitively unimpaired older adults with baseline plasma p-tau217 and A β PET imaging and longitudinal clinical follow-up from multiple well-characterized observational and clinical trial cohorts. Cognitive impairment was defined as a within-cohort diagnosis of mild cognitive impairment, dementia, or 2 consecutive global Clinical Dementia Rating scores of 0.5 or greater. A β , amyloid beta; CI, confidence interval; HR, hazard ratio; PET, positron emission tomography; SD, standard deviation; y, years. Buckley RF et al. *JAMA*. 2026:e2612556. Epub ahead of print. doi:10.1001/jama.2026.12556

SPINOZA



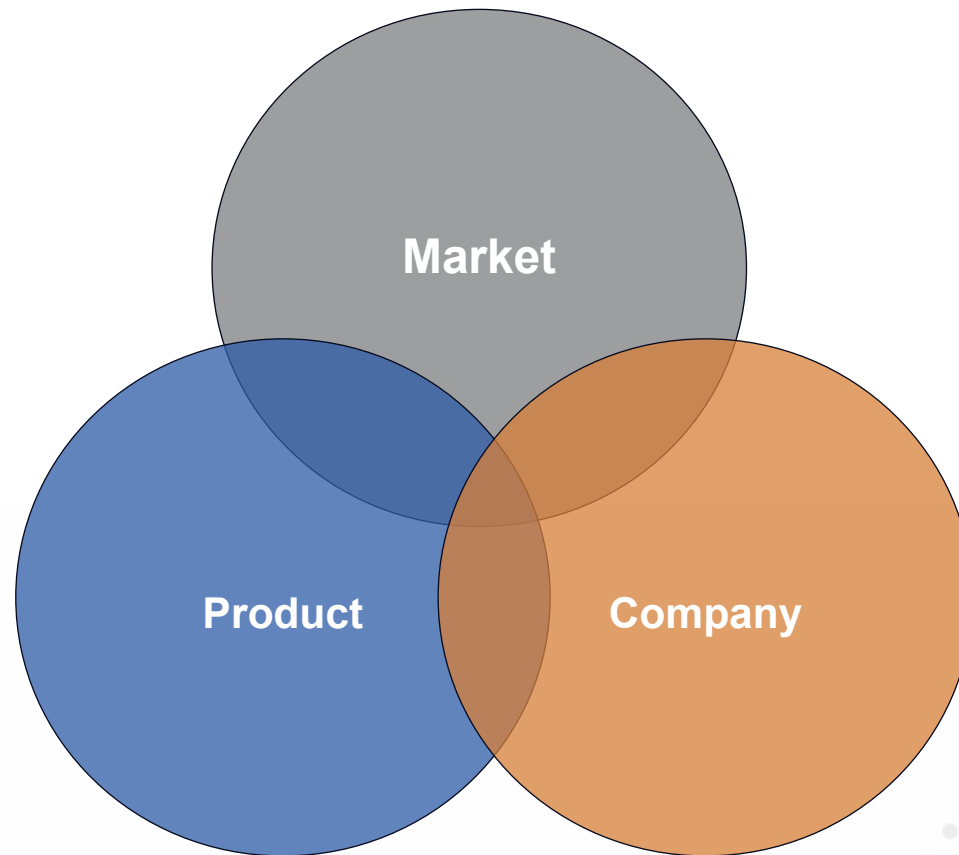


Commercial Update

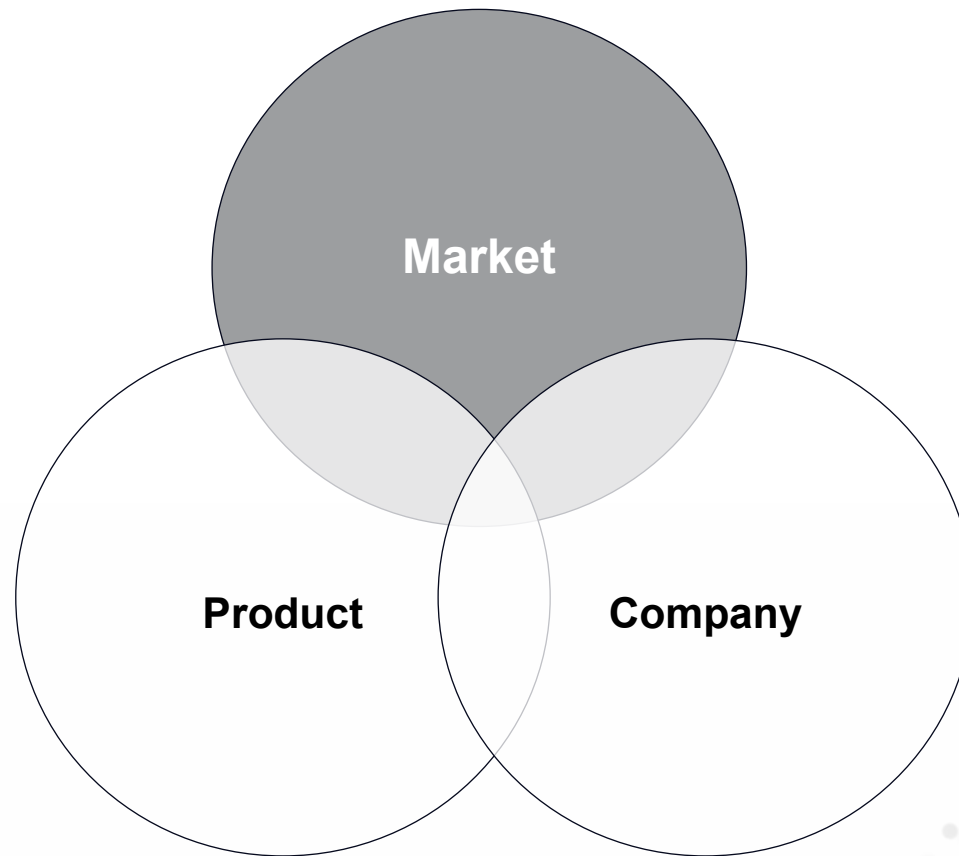
Agenda

- **US Launch Preparations**
- **European Launch Update**

US Launch Preparations



Preparing The Market



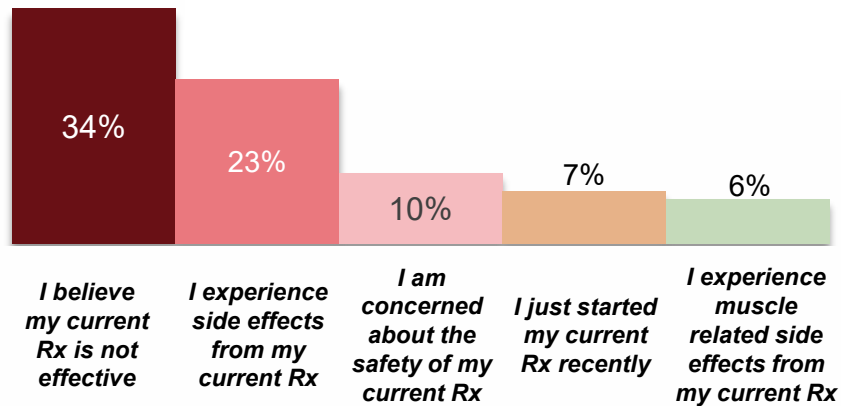
Patient Satisfaction With Current Therapies Is Low



2 out of 3 Patients
are not fully satisfied with their hypercholesterolemia treatment

What is the main reason you were not fully satisfied with your treatments for high cholesterol (hypercholesterolemia)?

Top 5 Reasons



Lack of Efficacy

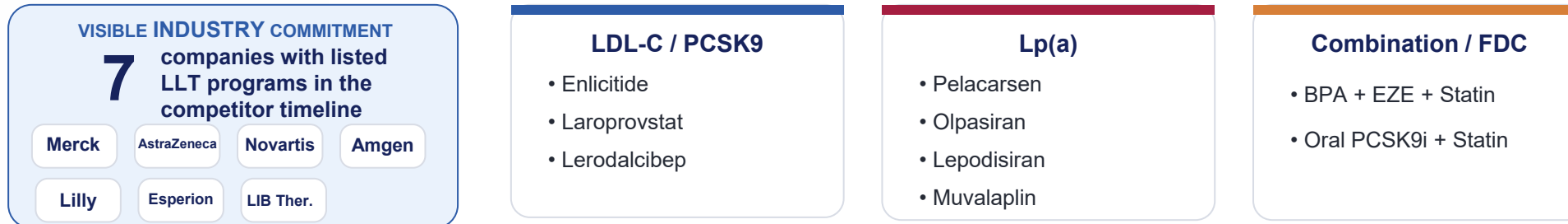
"Frustration. Because some of the best advice I would give someone to lower their cholesterol, I follow. And my cholesterol, is still very high."
- Patient

Safety/Tolerability

"Every statin I've ever tried makes muscles in your body just ache."
- Patient

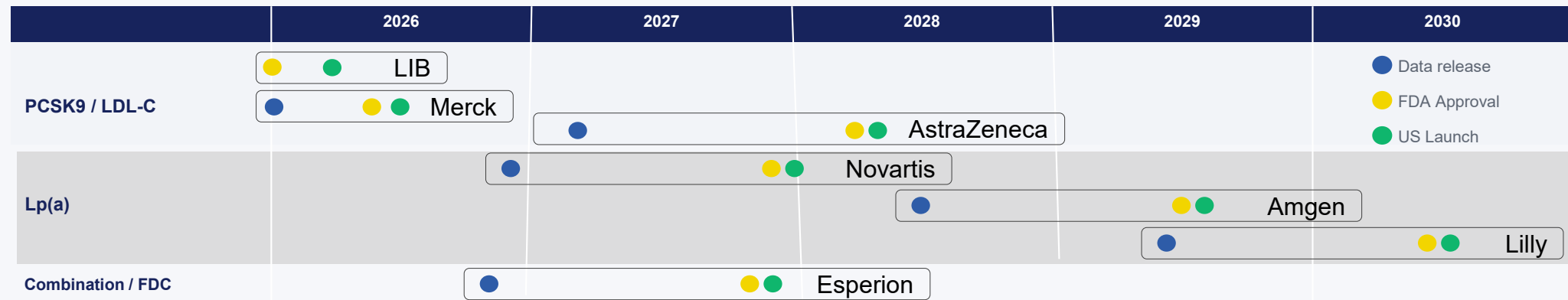
Unmet Need Is Driving Increased Innovation

Multiple companies are advancing LDL-C, Lp(a), and combination programs, reinforcing broad recognition that the LLT market continues to need new patient solutions



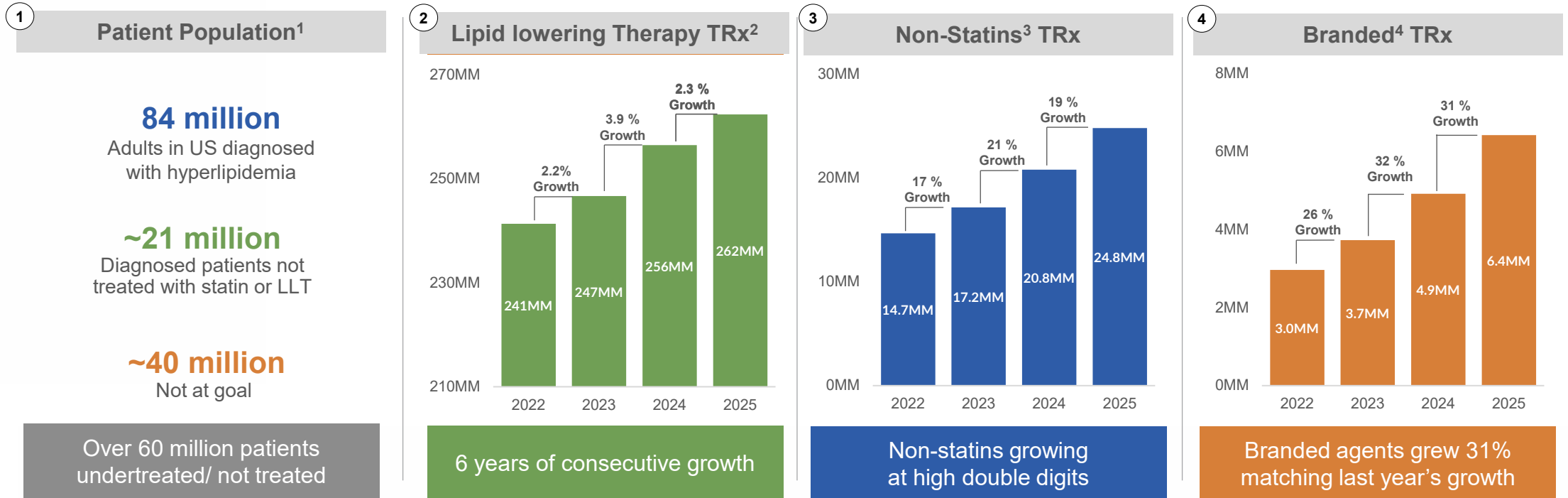
Lipid management pipeline momentum, 2026–2030

Visible timelines include launch, filing, approval, data release, and outcomes readout activity across multiple mechanisms



Industry investment across LDL-C, Lp(a), and combination approaches reinforces broad conviction that lipid management remains a large, active, and underpenetrated cardiovascular market

Market Dynamics: Growth of Non-Statin (+19%) and Branded (+31%) Volume Continue to Grow Rapidly Year Over Year



Market Growth of 2.3% in 2025, adds 6 million additional TRx's, of which 1.5 million are branded TRx's

Source: Based on third party prescription data on file

1. Forian Healthcare Closed Claims and EHR Data (2022-2024)

2. All Lipid Lowering therapies: Statins, Ezetimibe and combinations; PCSK9 and BPA;

3. Non-Statins : Ezetimibe and combinations; PCSK9 and BPA;

4. Branded: PCSK9 and BPA;

Recent Updates to Guidelines Advocate for More Aggressive Intervention

Guidance for patients with very high-risk ASCVD receiving maximally tolerated statin therapy

2018

2019

2020

2021

2022

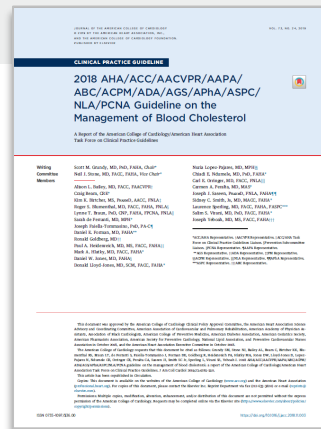
2023

2024

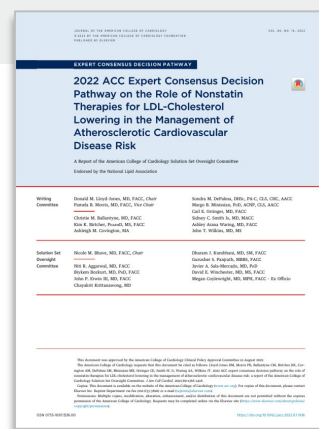
2025

2026

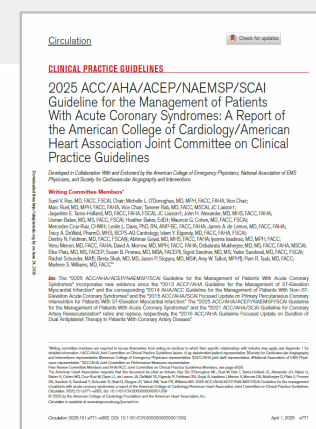
LDL-C threshold of **70 mg/dL** for the addition of nonstatin therapy¹



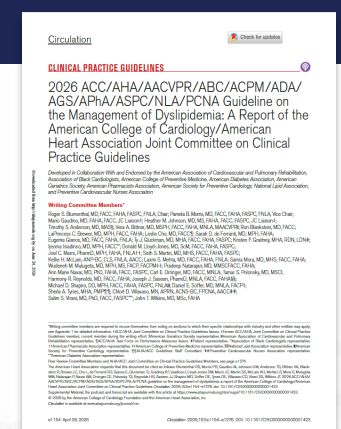
LDL-C threshold of **55 mg/dL** for the addition of nonstatin therapy²



LDL-C threshold of **55 mg/dL** for the addition of nonstatin therapy among hospitalized patients with ACS³



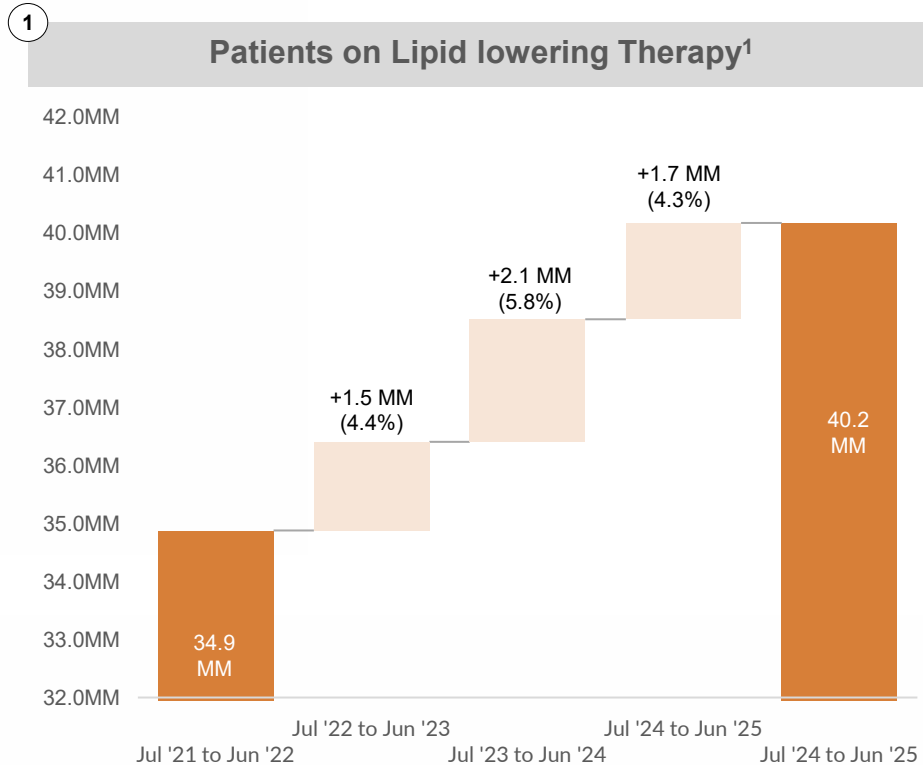
Formal LDL-C goal of <55 mg/dL to guide the addition and intensification of nonstatin therapy⁴



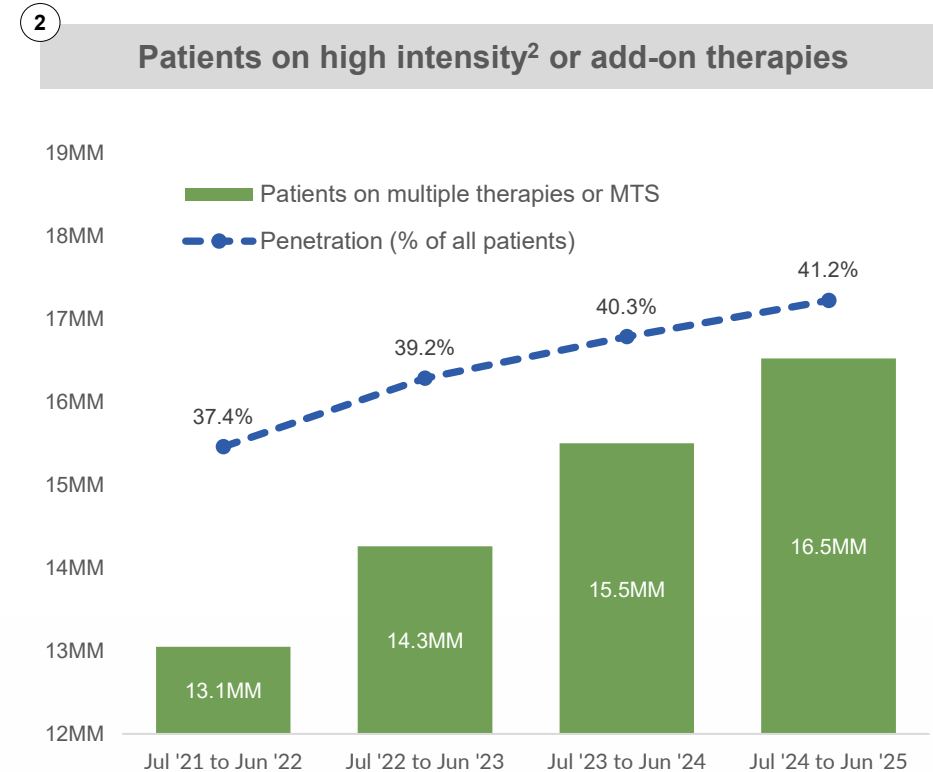
NEW!

1. Grundy SM et al. *J Am Coll Cardiol*. 2019;73(24):e285-e350.
2. Lloyd-Jones DM et al. *J Am Coll Cardiol*. 2022;80(14):1366-1418.
3. Rao SV et al. *Circulation*. 2025; 151(13):e771-e862.
4. Blumenthal RS et al. *Circulation*. 2026;153(17):e1154-e1276.

Resulting In More Patients Treated and Greater Focus On Goal Attainment



Greater than 5 MM treated patients added in the last 3 years



More aggressive treatment over time

Source: Forian data from Jul '21 to Jun '25

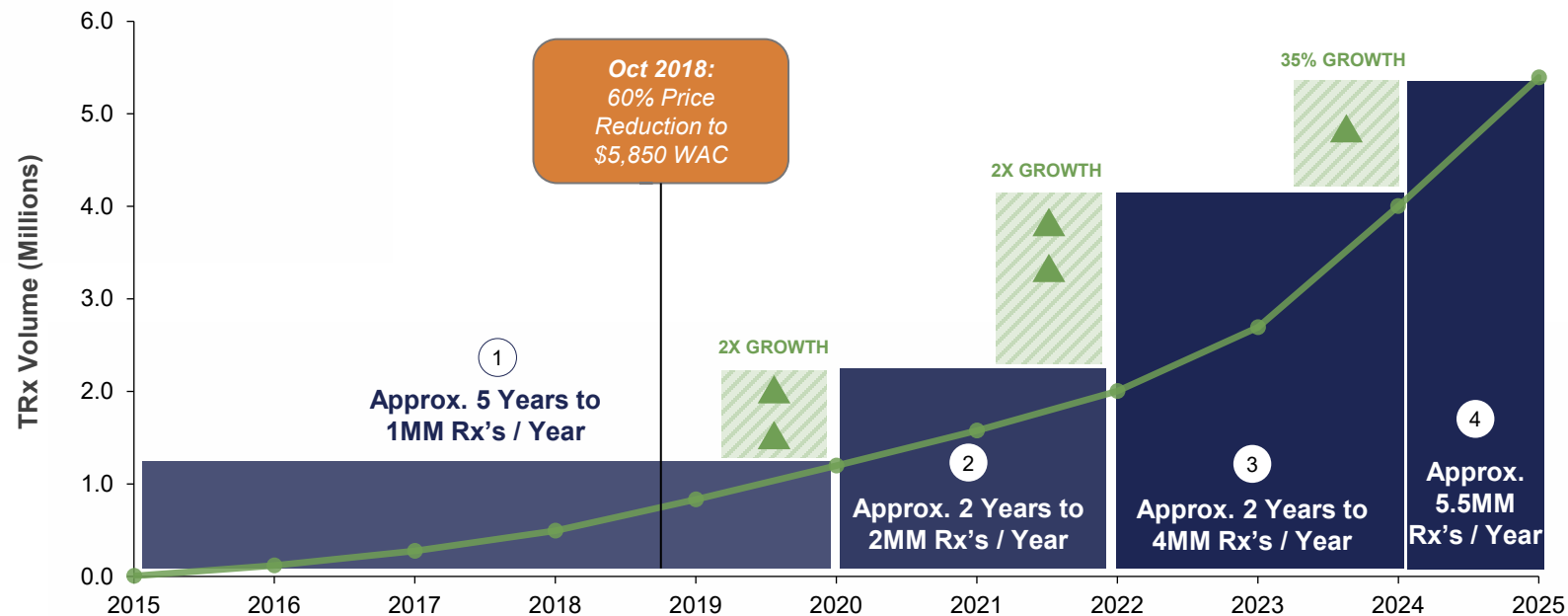
¹All Lipid Lowering therapies: Statins, Ezetimibe and combinations; PCSK9 and BPA

²Atorvastatin 40 & 80 mg, Simvastatin 80 mg, Rosuvastatin 20 & 40 mg

... Leading to Accelerated Brand Growth

Accelerating growth is evidence of the demand for additional options in the LLT space

Repatha® volume continues to be on track to double every 2 years

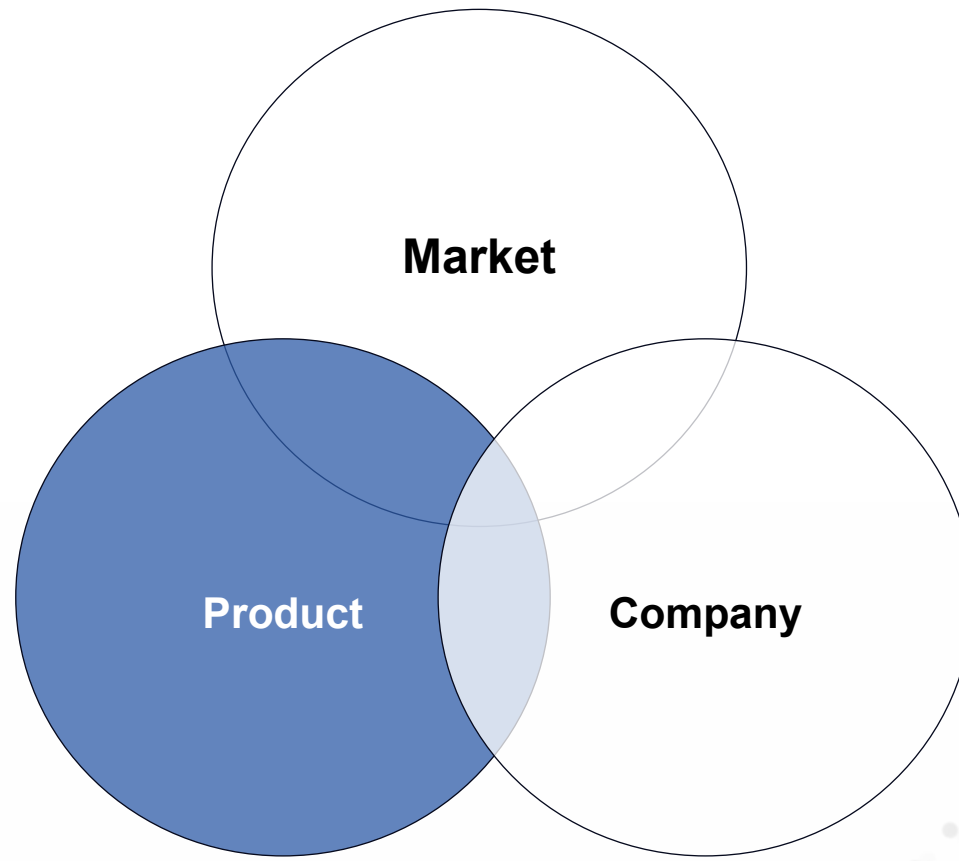


2025 worldwide revenue of
\$3 Billion

US revenues of
\$1.7 Billion

WW PCSK9 class is approaching
\$5 Billion

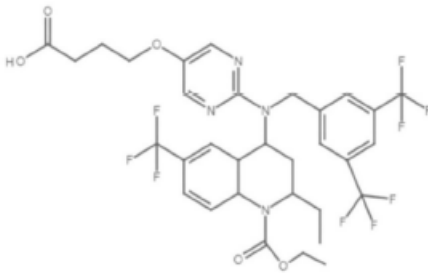
Preparing The Product



Evidence Supporting the Potential of Obicetrapib's Portfolio is Grounded in Comprehensive, Outcomes-Driven Clinical Program

1

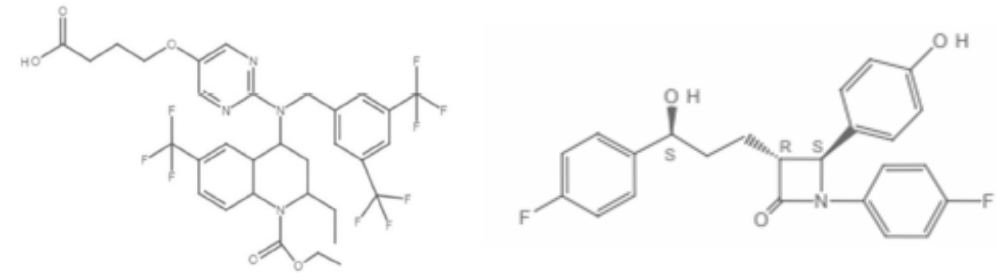
Obicetrapib



&

2

Obicetrapib + Ezetimibe



PHASE 2

TULIP

ROSE

ROSE2

JAPAN

BROOKLYN

BROADWAY

PHASE 3

TANDEM

PREVAIL*

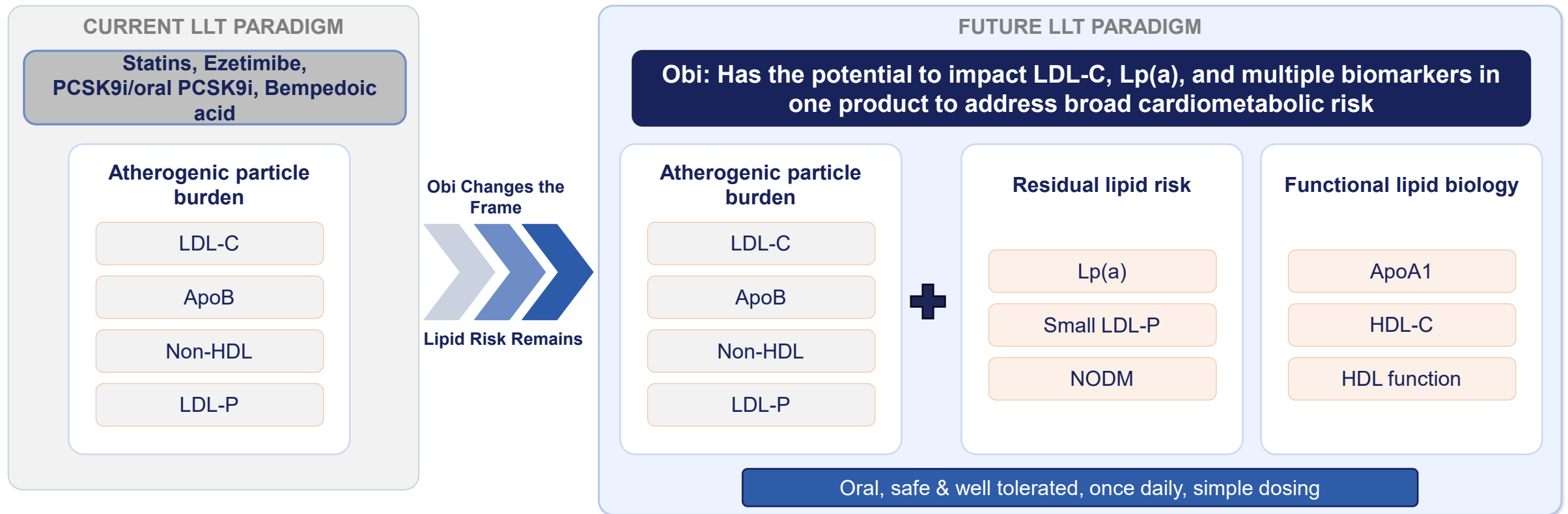
REMBRANDT*

VINCENT*

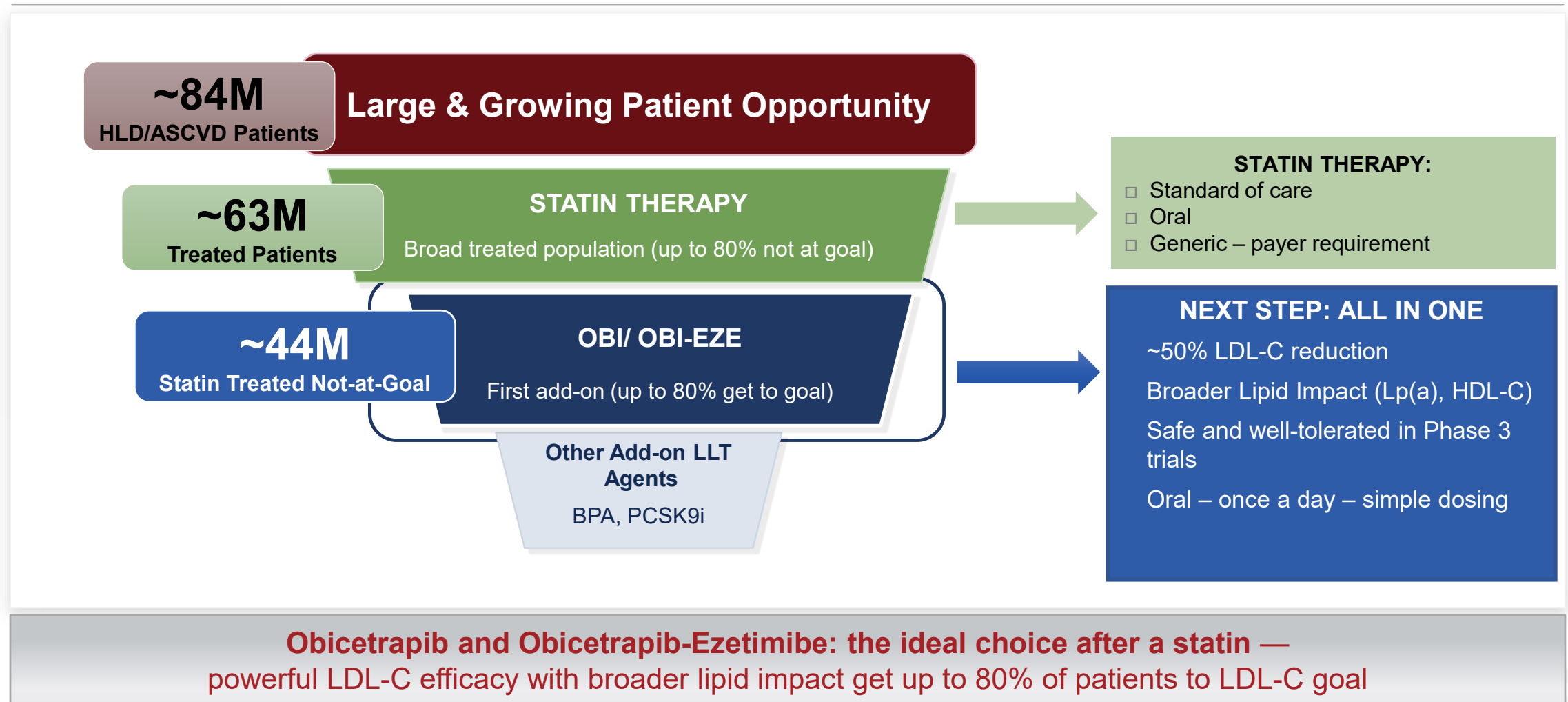
RUBENS*

Obi Delivers What LDL-Only Therapies Were Never Designed to Offer

Obi/ Obi-Eze redefines what physicians expect by addressing a broader set of lipid risk markers beyond LDL-C alone



Obi Redefines Post-Statin Therapy For >60 Million Patients Not At Goal, Supported by a Differentiated LDL-C+ Profile

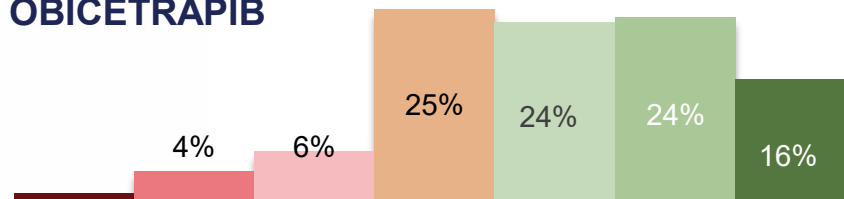


What Are Customers Saying About Obi and Obi-Eze?

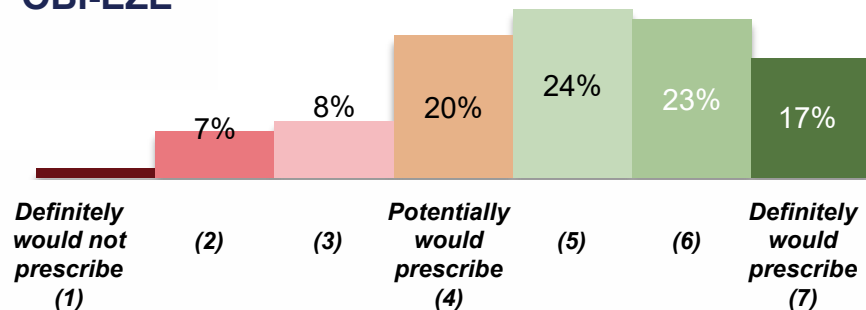


How likely are you to Rx **obi** and **obi-eze** in the first 6 months after they become available?

OBICETRAPIB



OBI-EZE



2 out of 3 HCP's

indicate a propensity to prescribe Obi & Obi-Eze within the first 6 months

Broad Efficacy*

Safety/Tolerability

“Now we have something that is even more effective at getting you to goal with minimal side effects, and it's safe to take with your current medications and, you know, just reducing your overall risk.”

- PCP

Convenience / Oral RoA

“I would just say it's a novel product, you know, once daily, oral, easy to use that targets those harder to fix types of cholesterol, like the Lp(a) and ApoB and boost the HDL, which leads to better cardiovascular outcomes.”

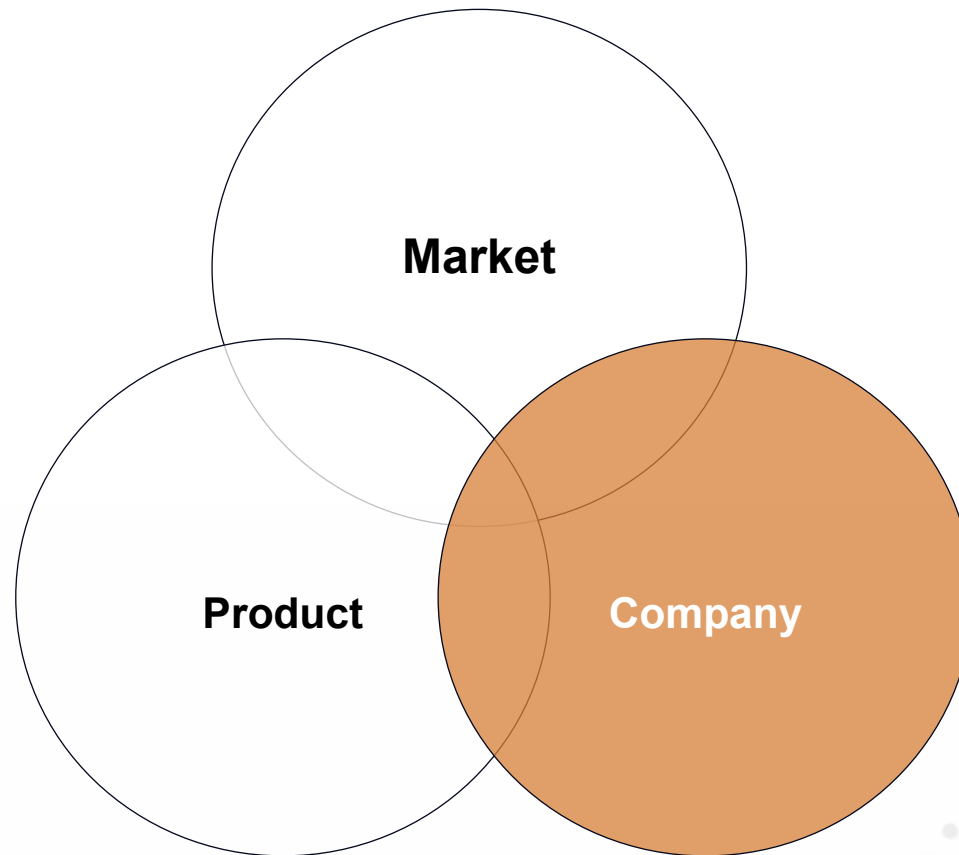
- PCP

Magnolia June 2026 HCP Quant Research, N=250 HCP Quantitative Respondents.

Obicetrapib and obi-eze target product profile described as a once daily tablet for a small molecule CETP inhibitor (or CETP + Ezetimibe for FDC) taken with or without food; with safety and efficacy profile reflecting BROADWAY, TANDEM and BROOKLYN Phase 3 results (including LDL-C, ApoB, Non-HDL-C, HDL-C, Lp(a), 4-point MACE).

*Source: Patient Physician Disconnect Research, April 2026.

Preparing The Company



Launch Success Requires A Great Asset, But Talent Is the Key To Unlocking Market Opportunity

NewAmsterdam Pharma Launch Leadership Team



BJ Jones

Chief Commercial Officer



Steven Albers

Executive Vice President,
Market Access &
Public Affairs



Michael Austwick

Executive Vice President,
Global Commercial



Chris Deluzio

Executive Vice President,
Sales & Enterprise
Operations



Serina Fischer

Executive Vice President,
Global Marketing



Debra Horner

Executive Director,
Operational Excellence



Andy Hsieh

Vice President,
Medical Affairs



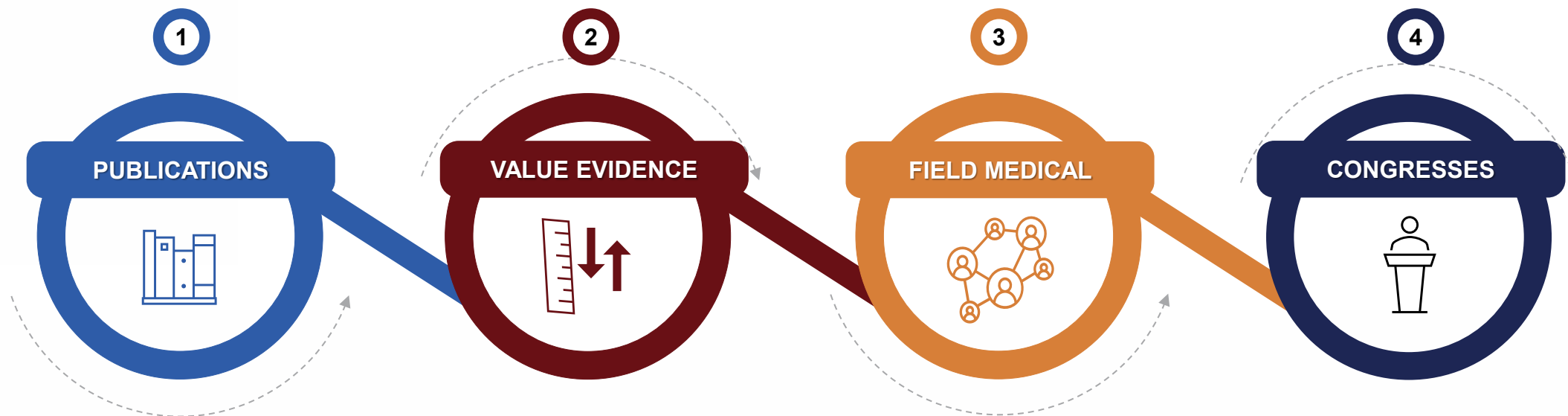
Sanjay Keshav

Vice President, Insights &
Analytics



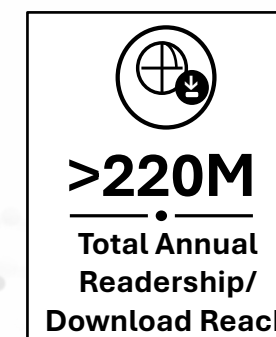
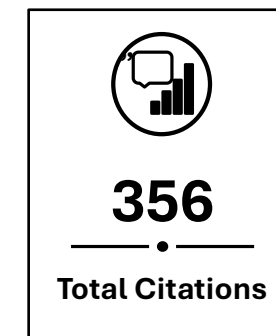
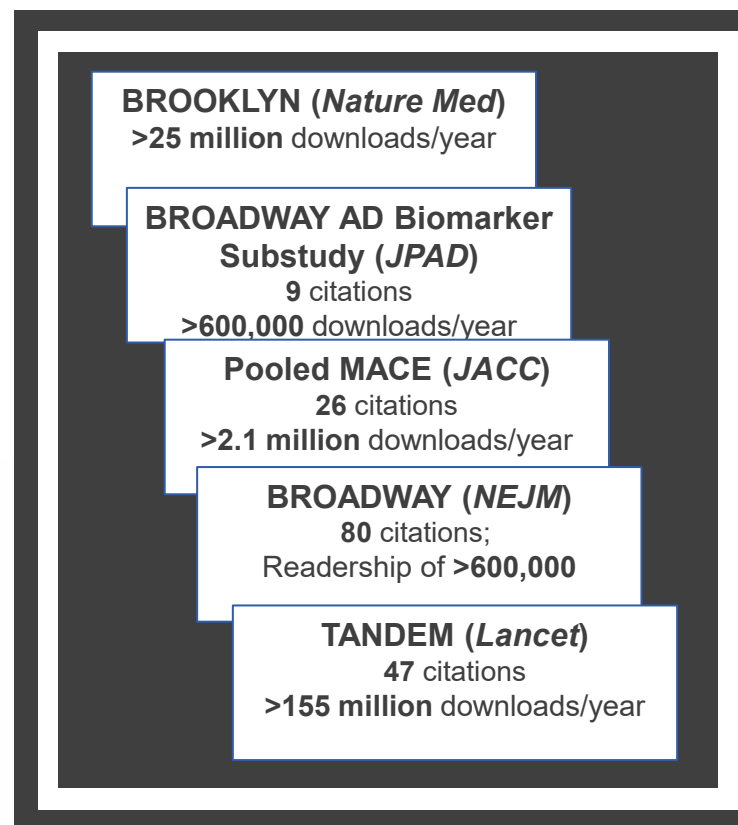
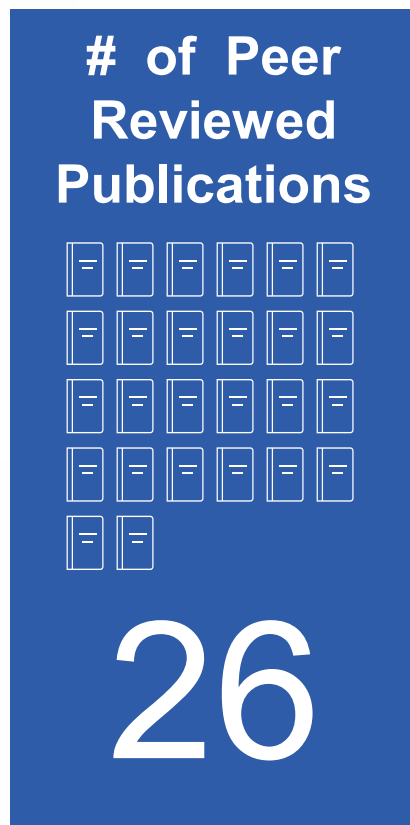
Already Deployed: Medical Affairs Education

Investment in Obicetrapib's scientific platform, engagement with the clinical community, and building value evidence are underway



In Market | Publications

Most Influential Journals in Cardiovascular Medicine, Reaching Hundreds of Millions of Readers



In Market | Value Evidence

Health economic value narrative grounded in clinical benefit and economic value



Milestones completed



Phase 3
(BROADWAY/TANDEM) and
Phase 2 (ROSE) CEAs
**ISPOR 2024 and 2026
posters**



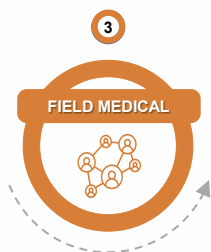
US value dossier



RWE unmet need and
resource use
**2026 NLA posters and
JME publication**

In Market | Field Medical

Built to engage institutions, leaders, and decision-makers shaping cardiovascular practice



Deployed MSL team has over 260 years of combined medical affairs experience!



AT THE INSTITUTION

1,700+

Institutions engaged

Engaged with the academic medical centers and health systems that shape practice



WITH PAYER MEDICAL

12

payer organizations

Pre-launch scientific engagement with the medical teams to inform and educate on unmet need



WITH SCIENTIFIC LEADERS

6000+

Engagements

Sustained scientific dialogue with leading cardiovascular and lipidology KOLs



AT CONGRESSES

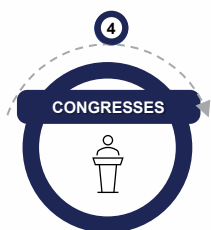
95+

Scientific presentations

Active scientific presence at every major cardiovascular and lipid society meeting

In Market | Congress Presence

Scientific dialogue spanning North America, Europe, and Asia

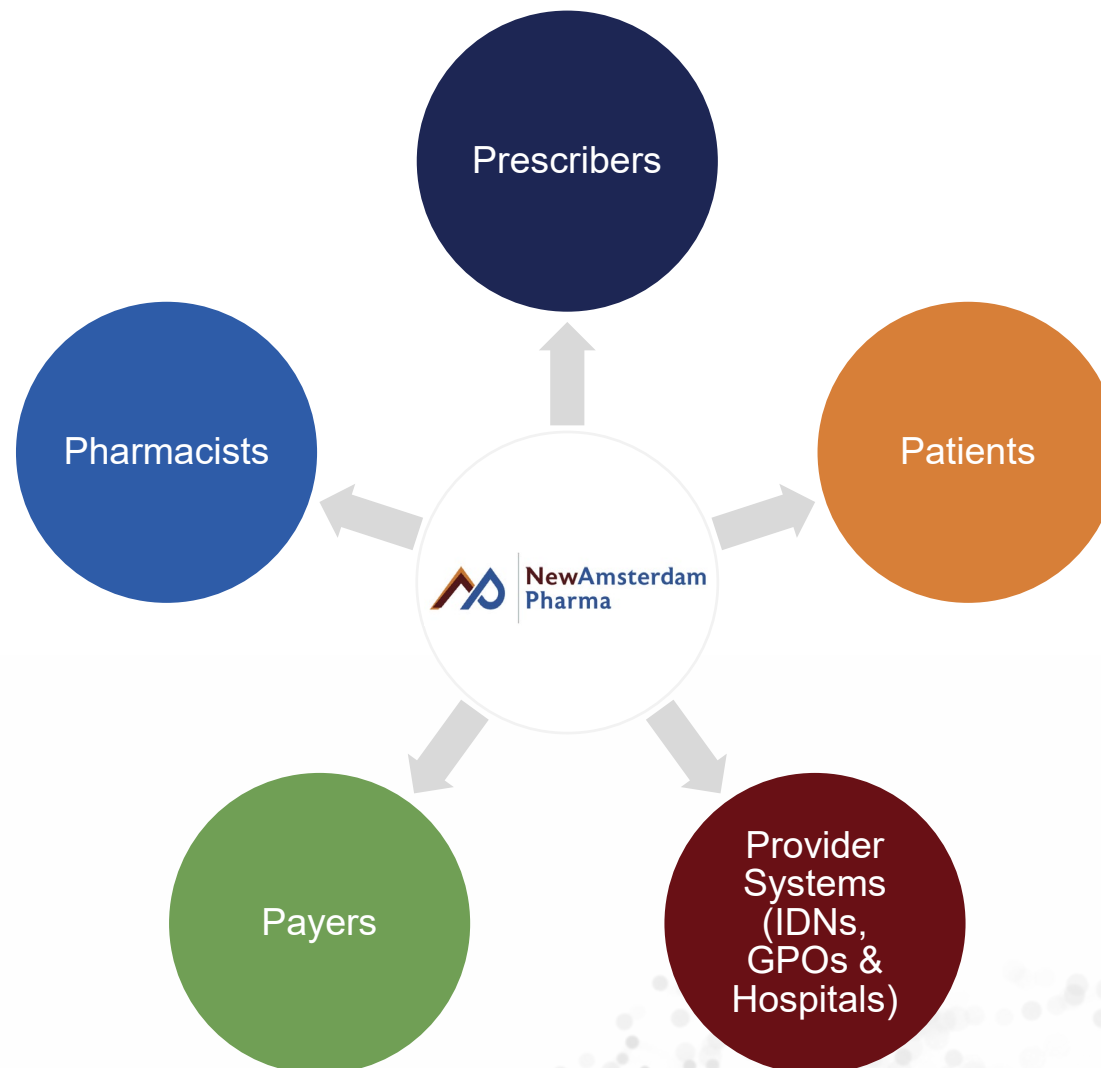


CARDIOVASCULAR SOCIETIES			
ACC American College of Cardiology <i>United States</i>	AHA American Heart Association <i>United States</i>	ESC European Society of Cardiology <i>Europe</i>	ASPC American Society for Preventive Cardiology <i>United States</i>
LIPID AND ATHEROSCLEROSIS SOCIETIES			
NLA National Lipid Association <i>United States</i>	EAS European Atherosclerosis Society <i>Europe</i>	JAS Japan Atherosclerosis Society <i>Japan</i>	

2024 17 presentations — • —
2025 40 presentations — • —
2026 39 presentations & counting — • —

Coming Soon | Branded Launch!

NAMS is a customer-centric organization focused on bringing transformational therapy and a seamless experience to each of its key customer groups



Agenda

- US Launch Preparations
- European Launch Update



Obicetrapib

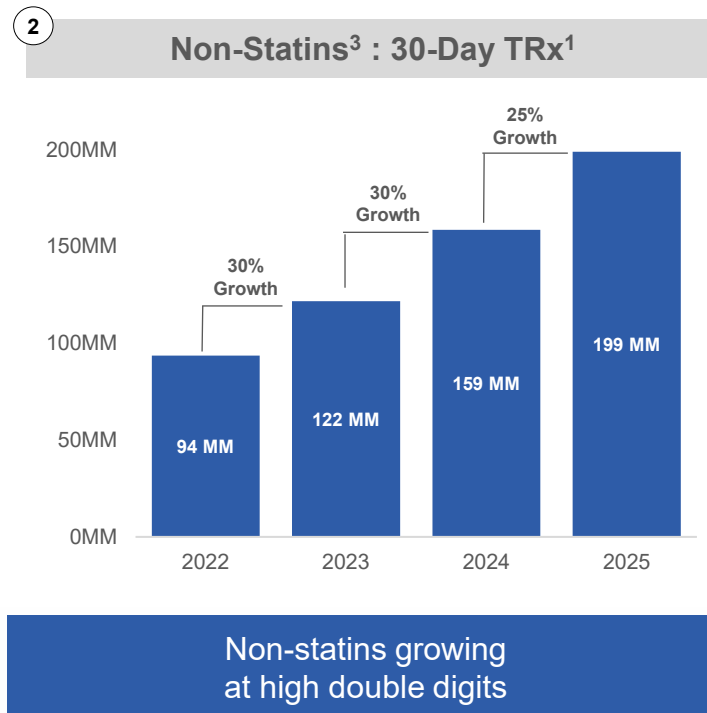
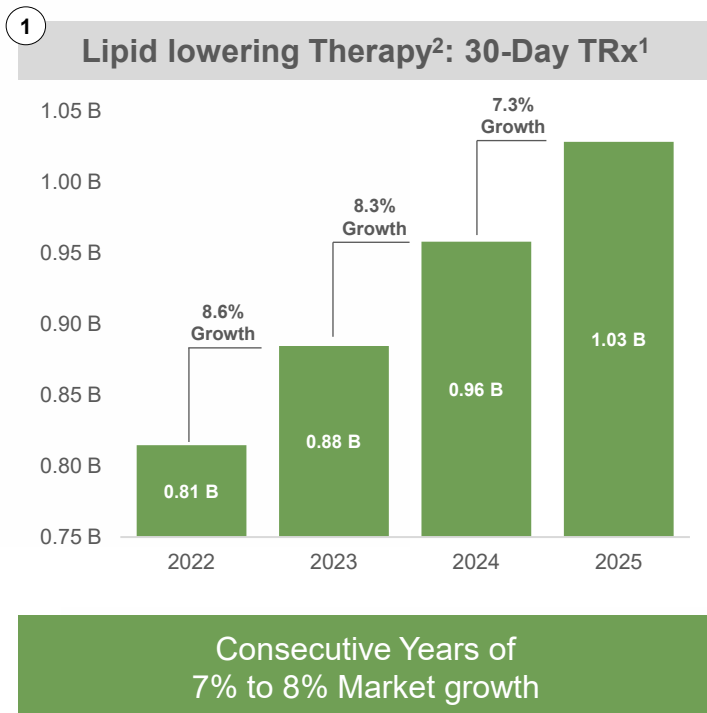
Positive CHMP
Opinion Received



NewAmsterdam
Pharma



EU Lipid Lowering Therapy Market is Large and Growing Rapidly

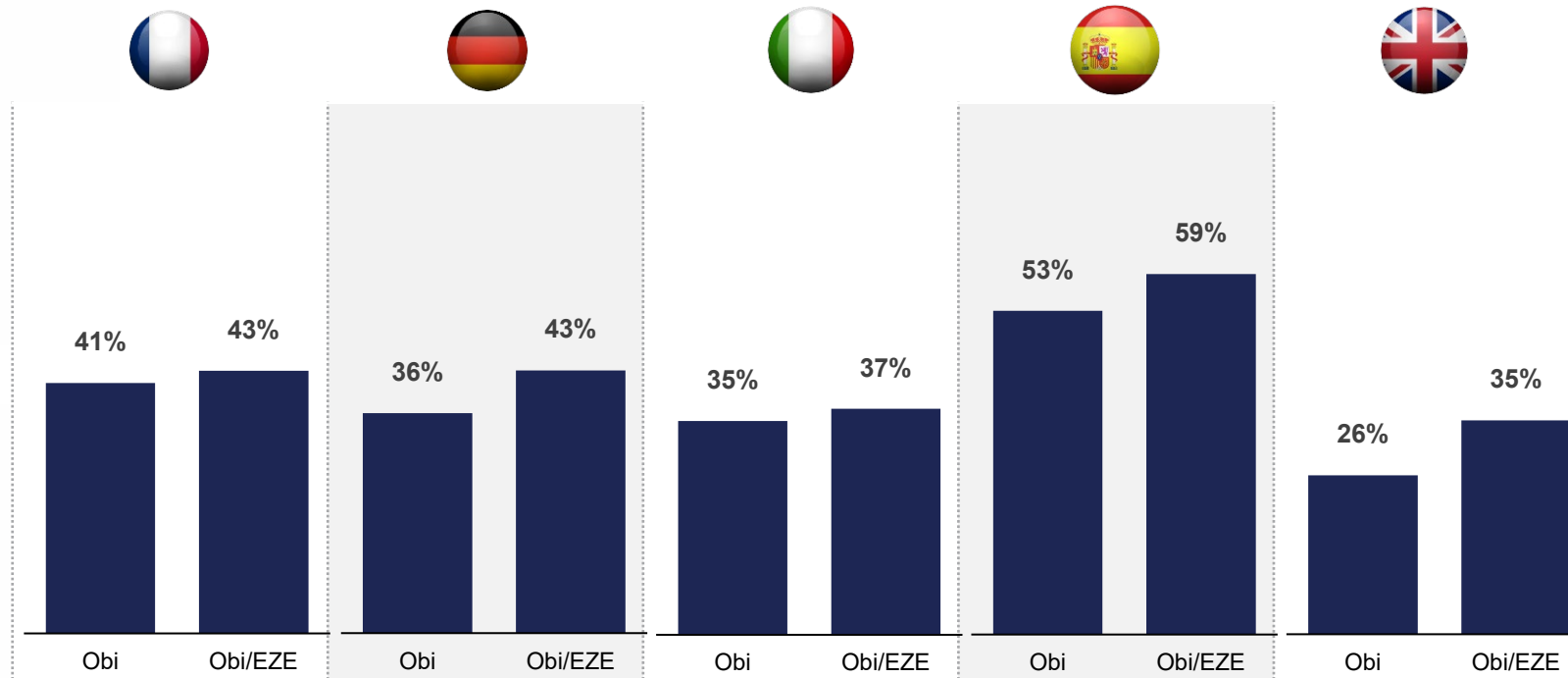


Market Growth of 7.3% in 2025, adds 70 million additional TRx's, of which 40 million are Non-Statin TRx's

Source: IQVIA MIDAS
1. 30 Day TRx: Using Standard units, 30-day Rx has been estimated based on unit usage in 30 days
2. All Lipid Lowering therapies: Statins, Ezetimibe and combinations; PCSK9 and BPA;
3. Non-Statins : Ezetimibe and its combinations; PCSK9 and BPA;
4. Branded: PCSK9 and BPA;

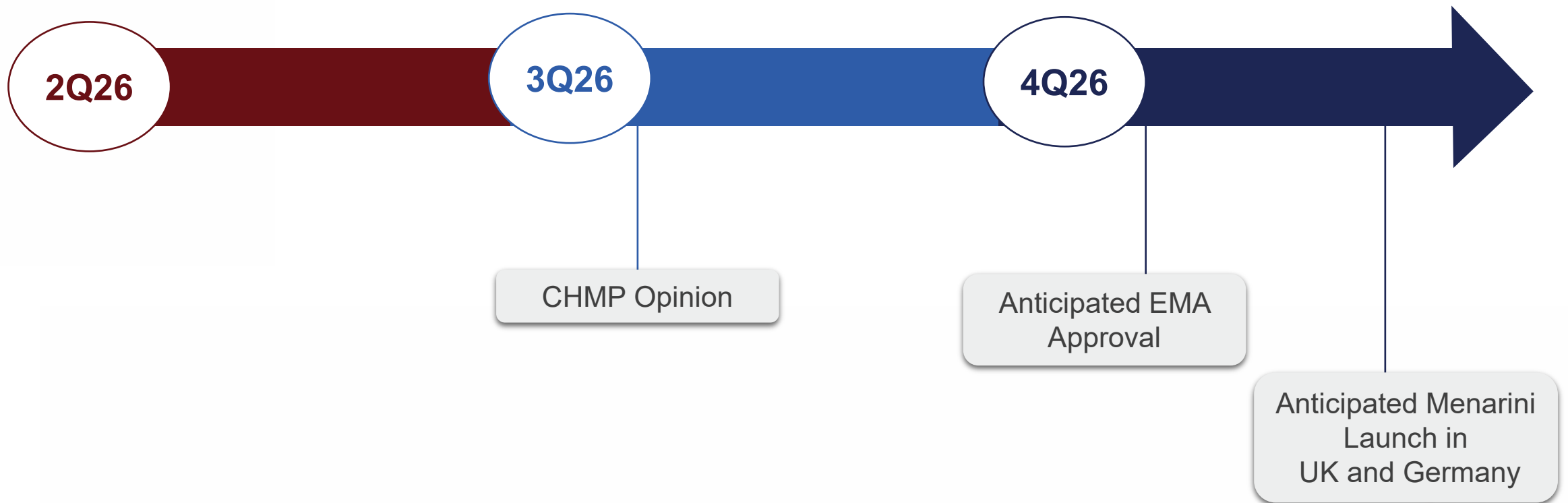
Early Demand for Obicetrapib Franchise Across EU5 Markets

Likelihood to Rx Obi/ Obi-Eze to At Least One Patient in the First 6 months After They Become Available
(7-point likelihood to Rx scale; % HCPs selecting 6 or 7)



Source: Magnolia March 2025 HCP Demand Study, US: N=252 HCP Quantitative Respondents; EU5 Countries: N=100 HCP Quantitative Respondents In Each Market (500 Total).

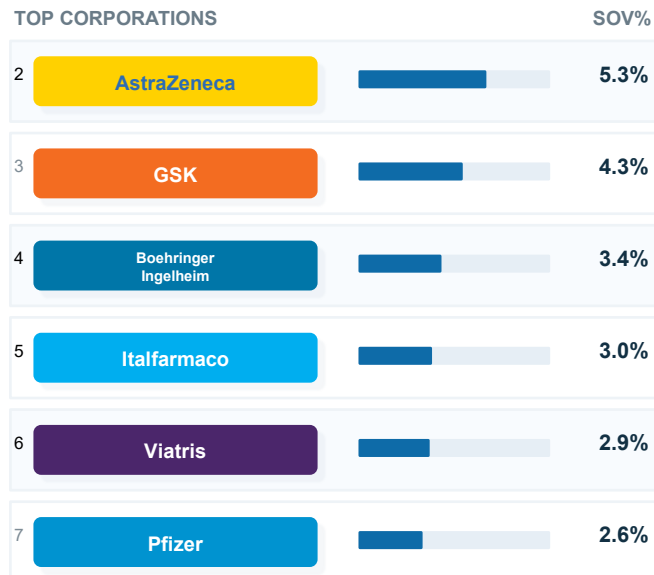
EU Regulatory and Launch Timeline



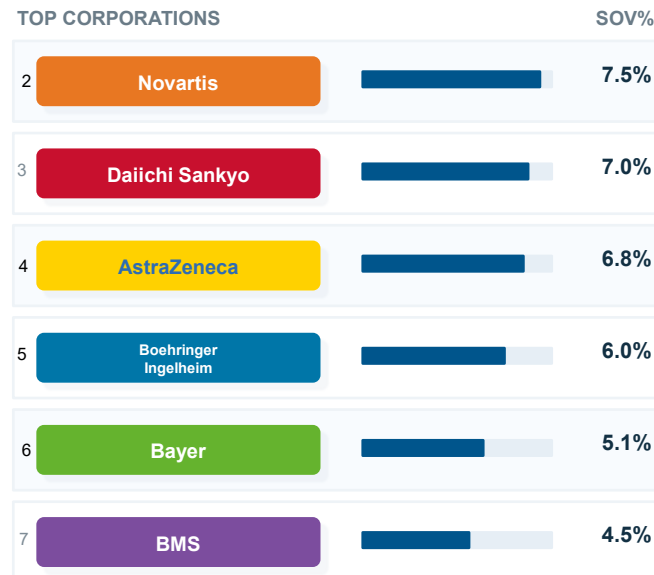
Menarini: Well-positioned to Launch Obi Driven by a Strong CV Heritage, Leading Share of Voice, and Significant Local Presence



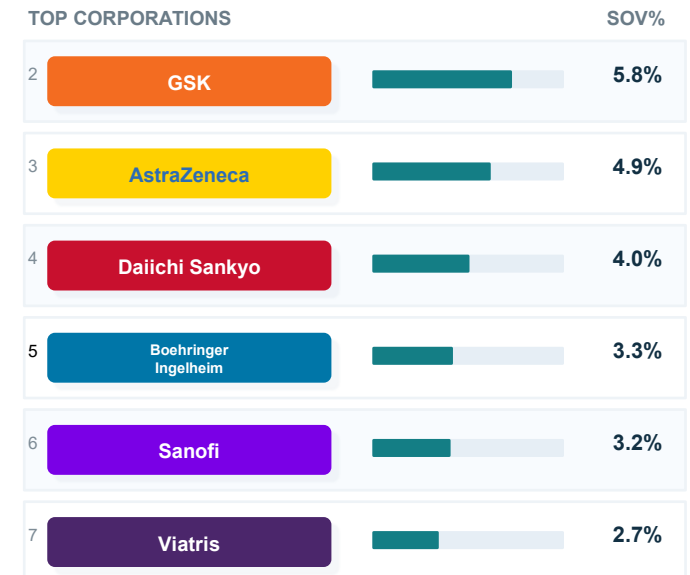
GPs



Cardiologists



Internists



Summary: If Approved, Obi/ Obi-Eze Can Redefine Post-Statins Treatment By Getting Patients to LDL-C Goal, While Mitigating Residual Lipid Risk



Significant unmet need is driving innovation in the cardiomatabolic space.



>60M addressable market for Obi franchise in US alone (~21M untreated/ ~40M not at goal)



Potential to be first LLT to launch with **3 successful Phase 3 studies** (BROOKLYN / BROADWAY / TANDEM) and available outcomes data (PREVAIL)



Observed **powerful LDL-C efficacy**, with differentiated benefit on Lp(a), Small LDL-P, and rate of NODM



Simple, oral, once daily, low dose option with a reported safety/tolerability profile **comparable to placebo**

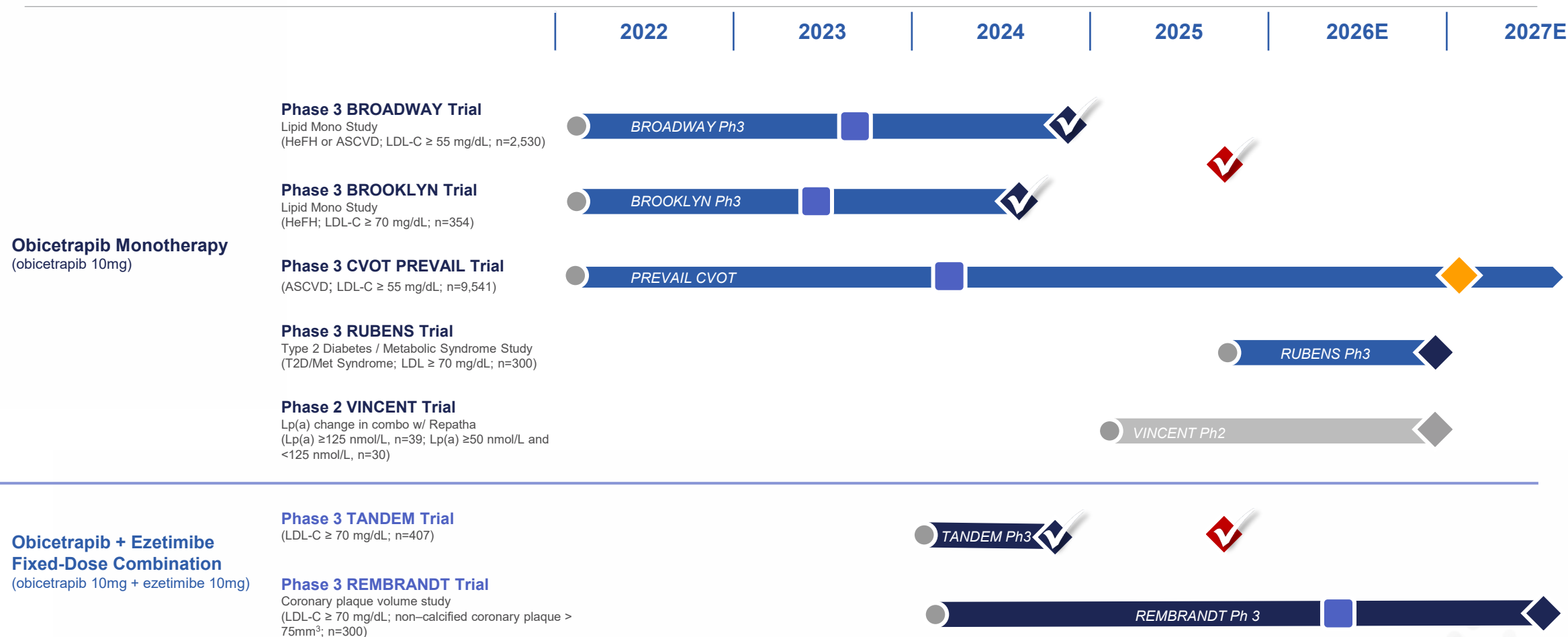


Medical Affairs education and engagement underway, with **industry-leading launch team** on board



Concluding Remarks

Multiple Data Readouts to Drive Future Momentum



*MAAs were filed and accepted for review by EMA, UK and Switzerland regulatory authorities

Note: Other than as noted, the pipeline represents trials that are currently ongoing. 2026 and beyond are projections and subject to inherent limitations. Actual results may differ from expectations.

The NewAmsterdam Value Proposition



Clinical Expertise: Data driven clinical development increases probability of success



Differentiated Target Product Profile: Phase 3 data demonstrates obicetrapib's potential impact on multiple CVD risk factors



Unmet Need: Significant need for a potent, convenient, well-tolerated low-dose oral therapy



Experienced Leadership and Team: Expanding team comprised of accomplished industry executives



KOL Support: Physician community aware of unmet need and eager to leverage obicetrapib's differentiated profile to achieve desired clinical benefit, if approved



CMC Expertise and Manufacturing Capacity: Preparing to meet anticipated demand for obicetrapib and the fixed dose combination, creating redundancy, expanding capacity and building inventory



Commercial Excellence: Growing team with track record of blockbuster launch success and innovative go-to-market model



Strong Balance Sheet: Cash runway expected to be sufficient to support U.S. commercial launch

What to Expect in Next 12-18 Months with Potential Milestones Hitting as Momentum Continues to Build



RUBENS

RUBENS Phase 3

LDL-C, Lp(a), Safety, in Type 2
Diabetics



PREVAIL

PREVAIL CVOT

Interim Analysis



REMBRANDT

REMBRANDT Phase 3

NCPV, LDL-C, Lp(a), Safety
Enrollment Complete



SPINOZA
STUDY

SPINOZA Phase 2b trial

Alzheimer's Disease

Tailwinds Driving the Market



Recent guideline and
label expansions



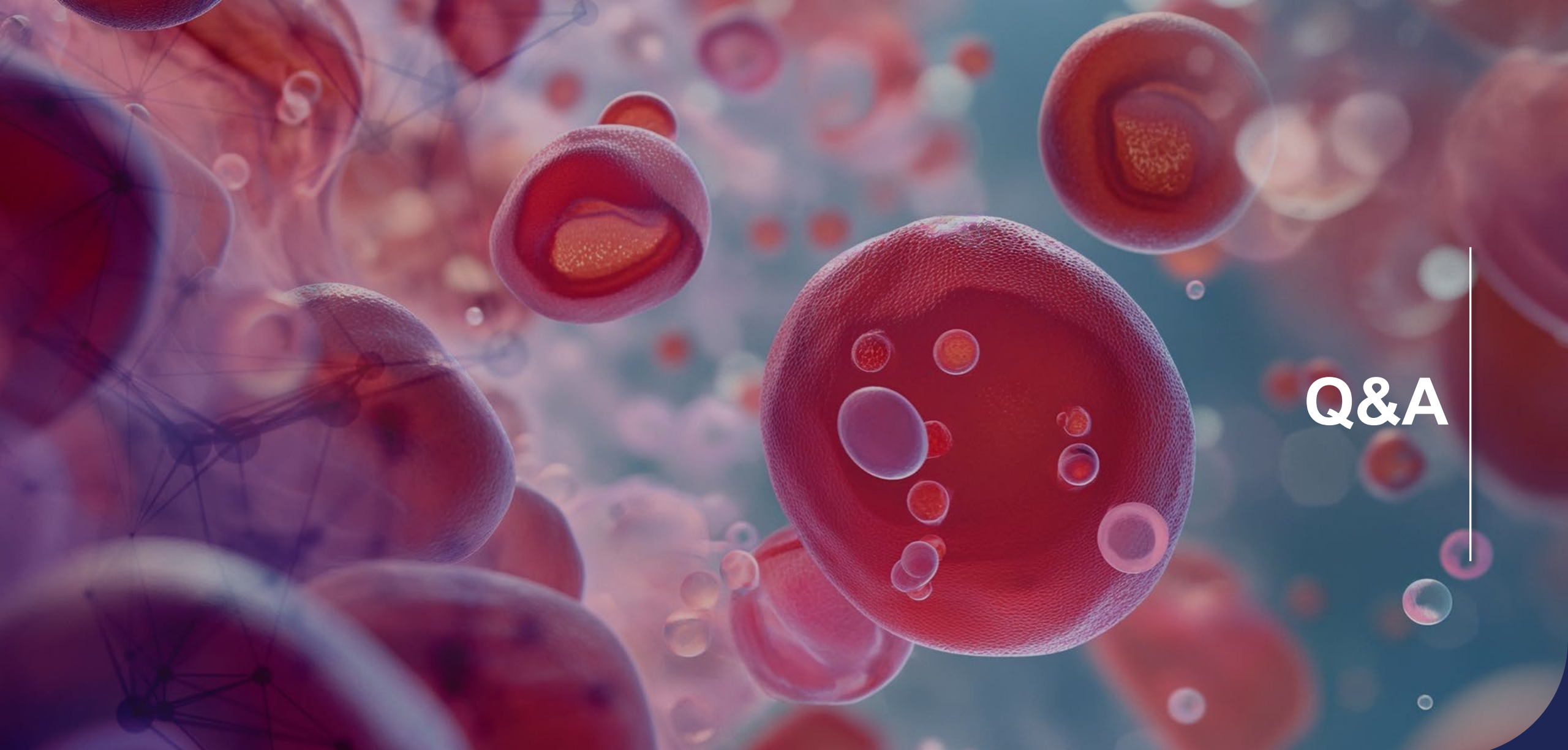
Increased treatment aimed
at hitting LDL-C goals



Lp(a) gaining momentum
as target, testing added
to guidelines



Accelerated adoption of
treatments adjunct to statins



Q&A

Thank You



NewAmsterdam
Pharma

Appendix: Prior CVOT Baseline Characteristics

Table 1. Characteristics of the Patients at Baseline.*		
Characteristics	Evolucumab (N=13,784)	Placebo (N=13,780)
Age — yr	62.5±9.1	62.5±8.9
Male sex — no. (%)	10,397 (75.4)	10,398 (75.5)
White race — no. (%)†	11,748 (85.2)	11,710 (85.0)
Weight — kg	85.0±17.3	85.5±17.4
Region		
North America	2,287 (16.6)	2,284 (16.6)
Europe	8,666 (62.9)	8,669 (62.9)
Latin America	913 (6.6)	910 (6.6)
Asia Pacific and South Africa	1,918 (13.9)	1,917 (13.9)
Type of atherosclerosis‡		
Myocardial infarction — no. (%)	11,145 (80.9)	11,206 (81.3)
Median time from most recent previous myocardial infarction (IQR) — yr	3.4 (1.0–7.4)	3.3 (0.9–7.7)
Nonhemorrhagic stroke	2686 (19.5)	2651 (19.2)
Median time from most recent previous stroke (IQR) — yr	3.2 (1.1–7.1)	3.3 (1.1–7.3)
Peripheral artery disease — no. (%)	1,858 (13.5)	1,784 (12.9)
Cardiovascular risk factors		
Hypertension — no./total no. (%)	11,045/13,784 (80.1)	11,039/13,779 (80.1)
Diabetes mellitus — no. (%)	5,054 (36.7)	5,027 (36.5)
Current cigarette use — no./total no. (%)	3854/13,783 (28.0)	3923/13,779 (28.5)
Statin use — no. (%)§		
High intensity	9,585 (69.5)	9,518 (69.1)
Moderate intensity	4,161 (30.2)	4,231 (30.7)
Low intensity, unknown intensity, or no data	38 (0.3)	31 (0.2)
Ezetimibe — no. (%)	726 (5.3)	714 (5.2)
Other cardiovascular medications — no./total no. (%)		
Aspirin, P2Y ₁₂ inhibitor, or both	12,766/13,772 (92.7)	12,666/13,767 (92.0)
Beta-blocker	10,441/13,772 (75.8)	10,374/13,767 (75.4)
ACE inhibitor or ARB, aldosterone antagonist, or both	10,803/13,772 (78.4)	10,730/13,767 (77.9)
Median lipid measures (IQR)		
LDL cholesterol — mg/dl	92 (80–109)	92 (80–109)
Total cholesterol — mg/dl	168 (151–188)	168 (151–189)
HDL cholesterol — mg/dl	44 (37–53)	44 (37–53)
Triglycerides — mg/dl	134 (101–183)	133 (99–181)
Lipoprotein(a) — nmol/liter	37 (13–166)	37 (13–164)

Table 1. Demographic and Baseline Characteristics of the Patients.*		
Characteristic	Alirocumab (N=9462)	Placebo (N=9462)
Age — yr	58.5±9.3	58.6±9.4
Female sex — no. (%)	2390 (25.3)	2372 (25.1)
Race — no. (%)†		
White	7500 (79.3)	7524 (79.5)
Asian	1251 (13.2)	1247 (13.2)
Black	235 (2.5)	238 (2.5)
Other	475 (5.0)	451 (4.8)
Region of enrollment — no. (%)		
Central and Eastern Europe	2719 (28.7)	2718 (28.7)
Western Europe	2084 (22.0)	2091 (22.1)
Canada or United States	1435 (15.2)	1436 (15.2)
Latin America	1293 (13.7)	1295 (13.7)
Asia	1150 (12.2)	1143 (12.1)
Rest of world	781 (8.3)	779 (8.2)
Medical history before index acute coronary syndrome — no. (%)		
Hypertension	6205 (65.6)	6044 (63.9)
Diabetes mellitus	2693 (28.5)	2751 (29.1)
Current tobacco smoker	2282 (24.1)	2278 (24.1)
Family history of premature coronary heart disease	3408 (36.0)	3365 (35.6)
Myocardial infarction	1790 (18.9)	1843 (19.5)
Percutaneous coronary intervention	1626 (17.2)	1615 (17.1)
Coronary-artery bypass grafting	521 (5.5)	526 (5.6)
Stroke	306 (3.2)	305 (3.2)
Peripheral artery disease	373 (3.9)	386 (4.1)
Congestive heart failure	1365 (14.4)	1449 (15.3)
Index acute coronary syndrome — no. (%)		
ST-segment elevation myocardial infarction	3301 (34.9)	3235 (34.2)
Non-ST-segment elevation myocardial infarction	4574 (48.3)	4601 (48.6)
Unstable angina	1568 (16.6)	1614 (17.1)
Missing data	19 (<0.1)	12 (<0.1)
Percutaneous coronary intervention or coronary-artery bypass grafting for index acute coronary syndrome — no. (%)	6798 (71.8)	6878 (72.7)
Median time from index acute coronary syndrome to randomization (IQR) — mo	2.6 (1.7–4.4)	2.6 (1.7–4.3)
Body-mass index‡	28.5±4.9	28.5±4.8

Source: Internal analysis | 1. Sayed et al. International Journal of Cardiology 406 (2024) 132074 2.McGuire et al. N Engl J Med 2025;392:2001-12. 3.Lincoff et al. N Engl J Med 2023;389:2221-32. 4. Sabatine et al. N Engl J Med 2017;376:1713-22. 5. Schwartz et al. N Engl J Med 2018;379:2097-107.

Appendix: Prior CVOT Baseline Characteristics

Table 1. Demographic and Baseline Patient Characteristics in the Intention-to-Treat Population.*

Characteristic	Bempedoic Acid (N = 6992)	Placebo (N = 6978)
Age		
Mean — yr	65.5±9.0	65.5±8.9
Distribution — no. (%)		
<65 yr	2859 (40.9)	2907 (41.7)
≥65 to <75 yr	3070 (43.9)	3027 (43.4)
≥75 yr	1063 (15.2)	1044 (15.0)
Female sex — no. (%)	3361 (48.1)	3379 (48.4)
White race — no. (%)†	6397 (91.5)	6335 (90.8)
Hispanic or Latinx — no. (%)†	1190 (17.0)	1143 (16.4)
Body-mass index‡	29.9±5.2	30.0±5.2
LDL cholesterol		
Mean value — mg/dl	139.0±34.9	139.0±35.2
Distribution — no. (%)		
<130 mg/dl	3074 (44.0)	3089 (44.3)
≥130 to <160 mg/dl	2213 (31.7)	2250 (32.2)
≥160 mg/dl	1705 (24.4)	1639 (23.5)
HDL cholesterol — mg/dl	49.6±13.3	49.4±13.3
Non-HDL cholesterol — mg/dl	173.8±39.5	173.9±40.2
Total cholesterol — mg/dl	223.5±40.6	223.3±41.1
Median triglycerides (IQR) — mg/dl	159.5 (118.0–216.5)	158.5 (118.0–215.0)
Median high-sensitivity CRP (IQR) — mg/liter	2.3 (1.2–4.5)	2.3 (1.2–4.5)
Estimated GFR — no. (%)		
≥90 ml/min/1.73 m ²	1216 (17.4)	1233 (17.7)
≥60 to <90 ml/min/1.73 m ²	4322 (61.8)	4282 (61.4)
≥30 to <60 ml/min/1.73 m ²	1437 (20.6)	1444 (20.7)
Cardiovascular risk category — no. (%)		
Primary prevention	2100 (30.0)	2106 (30.2)
Secondary prevention	4892 (70.0)	4872 (69.8)
Coronary artery disease	3574 (51.1)	3536 (50.7)
Peripheral arterial disease	794 (11.4)	830 (11.9)
Cerebrovascular atherosclerotic disease	1027 (14.7)	1040 (14.9)
Glycemic status — no. (%)		
Diabetes§	3144 (45.0)	3229 (46.3)
Inadequately controlled diabetes¶	1356 (19.4)	1369 (19.6)
Statin use — no. (%)	1601 (22.9)	1573 (22.5)
Ezetimibe use — no. (%)	803 (11.5)	809 (11.6)

Source: Internal analysis | 1. Sayed et al. International Journal of Cardiology 406 (2024) 132074 2.McGuire et al. N Engl J Med 2025;392:2001-12. 3.Lincoff et al. N Engl J Med 2023;389:2221-32. 4. Sabatine et al. N Engl J Med 2017;376:1713-22. 5. Schwartz et al. N Engl J Med 2018;379:2097-107.

Appendix: Prior CVOT Baseline Characteristics

Table 1. Baseline Characteristics of the Patients.*

Characteristic	Semaglutide (N= 8803)	Placebo (N= 8801)
Age — yr	61.6±8.9	61.6±8.8
Male sex — no. (%)	6355 (72.2)	6377 (72.5)
Race or ethnic group — no. (%)†		
White	7387 (83.9)	7404 (84.1)
Asian	720 (8.2)	727 (8.3)
Black	348 (4.0)	323 (3.7)
Other	253 (2.9)	273 (3.1)
Hispanic or Latino	914 (10.4)	908 (10.3)
Body weight — kg	96.5±17.5	96.8±17.8
BMI‡	33.3±5.0	33.4±5.0
Waist circumference — cm	111.3±13.1	111.4±13.1
Glycated hemoglobin level — %	5.78±0.34	5.78±0.33
Distribution — no. (%)		
<5.7%	2925 (33.2)	2980 (33.9)
≥5.7%	5877 (66.8)	5819 (66.1)
Median high-sensitivity CRP level (IQR) — mg/liter	1.87 (0.89–4.18)	1.80 (0.86–4.06)
Cardiovascular inclusion criteria — no. (%)		
Myocardial infarction only	5962 (67.7)	5944 (67.5)
Stroke only	1578 (17.9)	1556 (17.7)
Peripheral arterial disease only	376 (4.3)	401 (4.6)
Two or more inclusion criteria	718 (8.2)	719 (8.2)
Other§	169 (1.9)	181 (2.1)
eGFR — ml/min/1.73 m²	82.4±17.5	82.5±17.3
Median lipid level (IQR) — mg/dl		
Total cholesterol	153 (131–182)	153 (131–183)
HDL cholesterol	44 (37–52)	44 (37–52)
LDL cholesterol	78 (61–102)	78 (61–102)
Triglycerides	134 (99–188)	135 (100–190)
Systolic blood pressure — mm Hg	131.0±15.6	130.9±15.3
Diastolic blood pressure — mm Hg	79.4±10.0	79.2±9.9
Pulse — beats/min	68.9±10.6	68.6±10.7
EQ-5D-5L index score¶	0.88±0.15	0.88±0.15
EQ-5D-VAS score¶	77.15±15.63	77.15±15.73

Table 1. Characteristics of the Participants at Baseline.*

Characteristic	Oral Semaglutide (N= 4825)	Placebo (N= 4825)
Age — yr	66.1±7.6	66.1±7.5
Female sex — no. (%)	1376 (28.5)	1414 (29.3)
Race or ethnic group — no. (%)†		
White	3327 (69.0)	3321 (68.8)
Black	124 (2.6)	128 (2.7)
Asian	1134 (23.5)	1121 (23.2)
American Indian or Alaska Native	7 (0.1)	12 (0.2)
Native Hawaiian or Pacific Islander	4 (<0.1)	5 (0.1)
Other	185 (3.8)	192 (4.0)
Not reported	44 (0.9)	46 (1.0)
Hispanic or Latino ethnic group — no. (%)†	674 (14.0)	706 (14.6)
Body weight — kg	87.5±19.1	88.3±19.6
Body-mass index‡	31.0±5.7	31.2±5.9
Glycated hemoglobin level — mmol/mol	63.6±12.6	63.5±12.3
Glycated hemoglobin level — %	8.0±1.2	8.0±1.1
Median duration of diabetes (IQR) — yr	14.7 (9.0–20.8)	14.6 (8.9–20.8)
History of cardiovascular or kidney disease — no. (%)§		
Cardiovascular disease only	2730 (56.6)	2738 (56.7)
Chronic kidney disease only	632 (13.1)	609 (12.6)
Both cardiovascular and chronic kidney disease	1303 (27.0)	1317 (27.3)
Hypertension — no. (%)	4378 (90.7)	4381 (90.8)
Current smoking — no. (%)	545 (11.3)	584 (12.1)
Systolic blood pressure — mm Hg	134.6±16.3	134.7±16.4
Diastolic blood pressure — mm Hg	76.6±10.1	76.7±10.1
Pulse — beats/min	72.8±11.1	72.9±11.4
Median high-sensitivity C-reactive protein level (IQR) — mg/liter	2.0 (0.9–4.3)	2.0 (0.9–4.5)
eGFR — ml/min/1.73 m²¶	74.0±22.6	73.6±22.6

Source: Internal analysis | 1. Sayed et al. International Journal of Cardiology 406 (2024) 132074 2.McGuire et al. N Engl J Med 2025;392:2001-12. 3.Lincoff et al. N Engl J Med 2023;389:2221-32. 4. Sabatine et al. N Engl J Med 2017;376:1713-22. 5. Schwartz et al. N Engl J Med 2018;379:2097-107.