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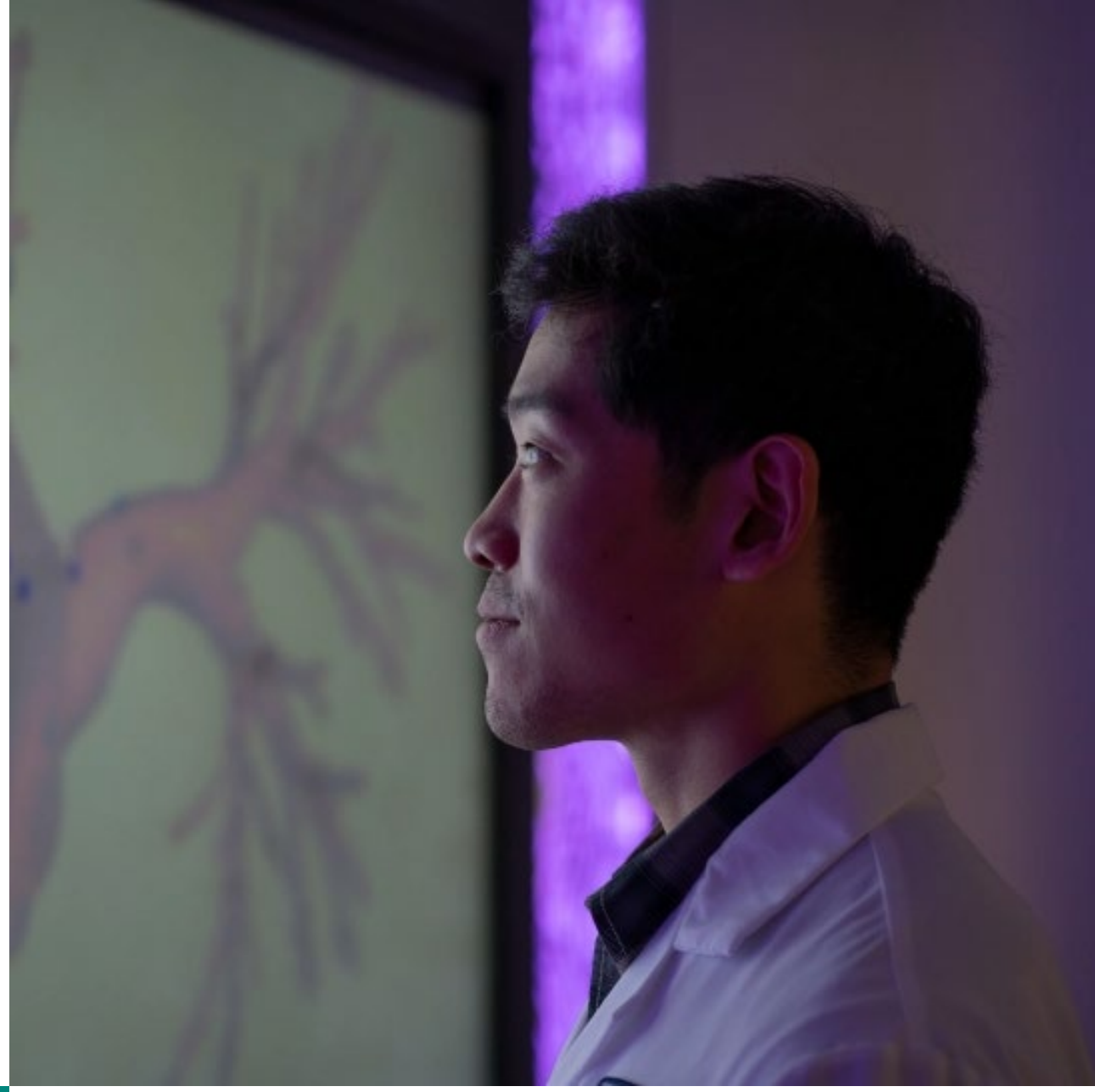
# Merck ACC.23/WCC Investor Event

March 6, 2023



# Forward-looking statement of Merck & Co., Inc., Rahway, N.J., USA

This presentation of Merck & Co., Inc., Rahway, N.J., USA (the "company") includes "forward-looking statements" within the meaning of the safe harbor provisions of the U.S. Private Securities Litigation Reform Act of 1995. These statements are based upon the current beliefs and expectations of the company's management and are subject to significant risks and uncertainties. There can be no guarantees with respect to pipeline candidates that the candidates will receive the necessary regulatory approvals or that they will prove to be commercially successful. If underlying assumptions prove inaccurate or risks or uncertainties materialize, actual results may differ materially from those set forth in the forward-looking statements.

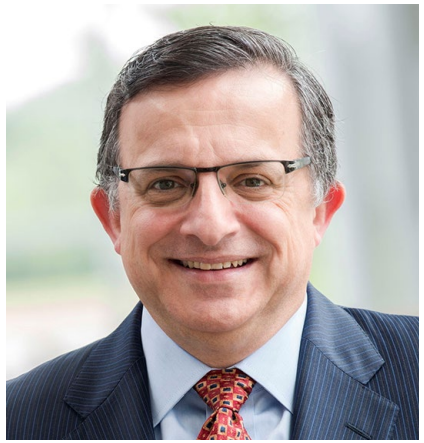
Risks and uncertainties include but are not limited to, general industry conditions and competition; general economic factors, including interest rate and currency exchange rate fluctuations; the impact of the global outbreak of novel coronavirus disease (COVID-19); the impact of pharmaceutical industry regulation and health care legislation in the United States and internationally; global trends toward health care cost containment; technological advances, new products and patents attained by competitors; challenges inherent in new product development, including obtaining regulatory approval; the company's ability to accurately predict future market conditions; manufacturing difficulties or delays; financial instability of international economies and sovereign risk; dependence on the effectiveness of the company's patents and other protections for innovative products; and the exposure to litigation, including patent litigation, and/or regulatory actions.

The company undertakes no obligation to publicly update any forward-looking statement, whether as a result of new information, future events or otherwise. Additional factors that could cause results to differ materially from those described in the forward-looking statements can be found in the company's Annual Report on Form 10-K for the year ended December 31, 2022 and the company's other filings with the Securities and Exchange Commission (SEC) available at the SEC's Internet site (www.sec.gov).

# Presenters

**Dr. Dean Li**
President,
Merck Research Laboratories

**Dr. Joerg Koglin**
VP,
Global Clinical Development, CV & Respiratory



**Dr. Eliav Barr**
SVP,
Head of Global Clinical Development & Chief Medical Officer

# Agenda

- **Opening |** Dean Li
- **ACC.23/WCC Data Highlights |** Joerg Koglin
- **Closing Remarks |** Eliav Barr
- **Q&A |** All with Chirfi Guindo and Jannie Oosthuizen

# Focusing on areas of greatest unmet need for patients

**Cardiovascular disease is the #1 cause of global mortality[1]**

## Pulmonary Arterial Hypertension


5-year mortality for patients with PAH[2]

- Existing therapies do not target the cellular level changes that contribute to PAH pathophysiology

## Atherosclerosis


of all cardiovascular disease deaths attributed to ASCVD[3]

- Large proportion of patients do not reach goals for LDL-C despite existing lipid-lowering therapies

## Thrombosis


Higher risk of CV death in patients with ESRD[4]

- Patients with ESRD have high-risk of thrombotic disease AND bleeding
- Patients with ESRD often excluded from clinical trials

## Heart Failure


5-year mortality for patients with heart failure[5]

- Symptoms of patients with chronic heart failure continue to worsen over time on current therapies

1. IHME, Global Burden of Disease, Number of deaths by cause, World, 2019 (ourworldindata.org); 2. Benza et al, *Chest* 2012; 142(2):448-456; 3. World Health Organization. Cardiovascular diseases (CVDs). https://www.who.int/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds). [Last accessed October 2021; 4. Nephrol Dial Transplant (2018) 33: iii28–iii34 doi: 10.1093/ndt/gfy17; 5. Benjamin EJ, et al. Circulation. 2018:e67-e492; 3. Benza et al, Chest 2012; 142(2):448-456

# Making significant progress across broad pipeline over the last year

**Scientific expertise and exceptional clinical execution**

## Pulmonary Hypertension

- **Sotatercept**:
  - Successfully completed Phase 3 STELLAR trial as an add-on to stable background PAH therapy
  - Continue to advance HYPERION, ZENITH, SOTERIA and CADENCE trials
- **MK-5475**
  - Phase 2/3 INSIGNIA-PAH trial enrollment progressing
  - Initiated Phase 2a INSIGNIA-PH-COPD POC trial

## Atherosclerosis

- **MK-0616**
  - Successfully completed Phase 2 dose-finding study in hypercholesterolemia
  - Planning for Phase 3

## Thrombosis

- **MK-2060**
  - Completed screening for Phase 2 trial in patients with ESRD receiving hemodialysis via an AVG

## Heart Failure

- **Verquvo**:
  - Exceeded 50% of target enrollment in Phase 3 VICTOR trial of patients with CHF and reduced ejection fraction who have not had a recent worsening heart failure event[1]

Further details provided in subsequent slides.

1. Worsening heart failure in this context means a recent hospitalization for heart failure or need for outpatient intravenous (IV) diuretic

# Compelling clinical data presented at ACC.23/WCC

**Sotatercept**

- **6MWD** +40.8 m[1]
- **PVR** -26.7%[2] vs placebo +2.8%
- **NT-proBNP** -62.6%[2] vs placebo +16.2%
- **TTCW** 84%[3] Risk reduction

- Met **primary** and **8 out of 9 secondary** endpoints
- **Potentially transformational** option

**MK-0616**[4]

- **LDL-C** up to 60.9% reduction
- **ApoB** up to 51.8% reduction
- **Non-HDL-C** up to 55.8% reduction
- **LDL goal** up to 90.8% attainment

- **LDL-C reduction and safety profile** in line with injectables
- Transitioned from Phase 1 to Phase 3 in **less than 18 months**

1. Hodges-Lehmann location shift from placebo estimate 2. Median, observed data only (no imputation). 3. TTCW is time to clinical worsening or death; HR of 0.16 (95% CI: 0.08 to 0.35) 4.MK-0616 results shown are at highest dose studied (30mg). LDL-C reduction is primary endpoint (95%CI); ApoB reduction, Non-HDL-C reduction, and LDL goal attainment were secondary endpoints (not multiplicity controlled).

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# ACC.23.WCC Data Highlights Sotatercept -

## an activin signaling inhibitor being studied for the treatment of adults with PAH

# Significant unmet need for people with PAH

## PAH is a rare, rapidly progressive and fatal disease

1-year mortality **15.3%**[1]

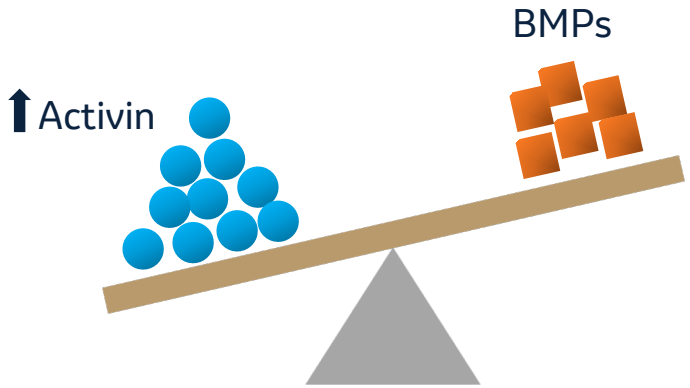
5-year mortality **43.0%**[1]

- **~40,000** people are living with PAH in the United States[2]
- **Mortality is high despite** advances in treatment, including the availability of upfront combination vasodilator therapy
- Current standard of care treats the symptoms of PAH, **does not target the underlying disease pathophysiology**

1. Benza et al, *Chest* 2012; 142(2):448-456 2. Kirson et al. 2011 https://pubmed.ncbi.nlm.nih.gov/21793646/

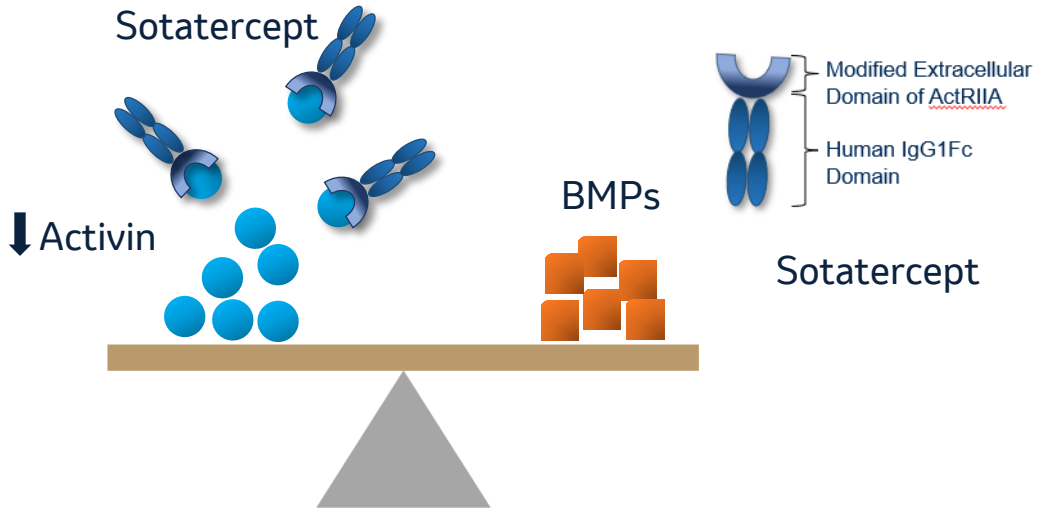
# Sotatercept rebalanced pro- and anti-proliferative signaling to modulate vascular proliferation in pre-clinical models

## Pulmonary arterial hypertension




**Increased vascular proliferation**

## Proposed mechanism of action



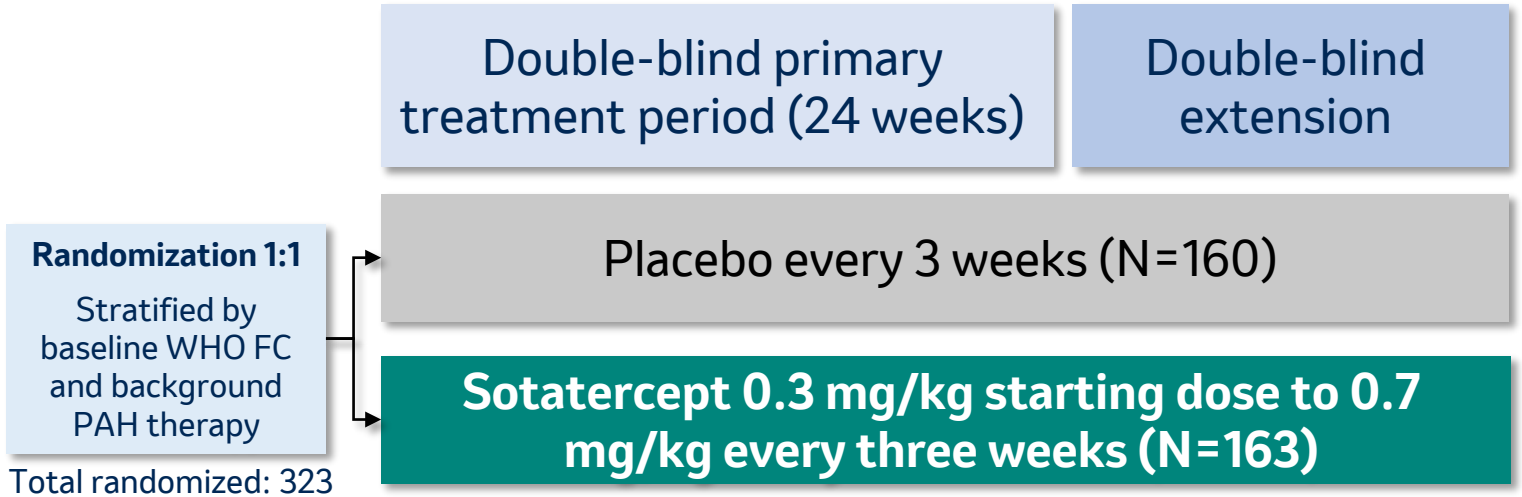

**Rebalanced toward vascular homeostasis**

- **Novel biologic** in treatment of PAH **targeting the underlying disease** pathophysiology
- Potential first in-class **activin signaling inhibitor** that is designed to **modulate vascular proliferation**

# STELLAR Phase 3 patient inclusion criteria and randomization

**Inclusion Criteria:**

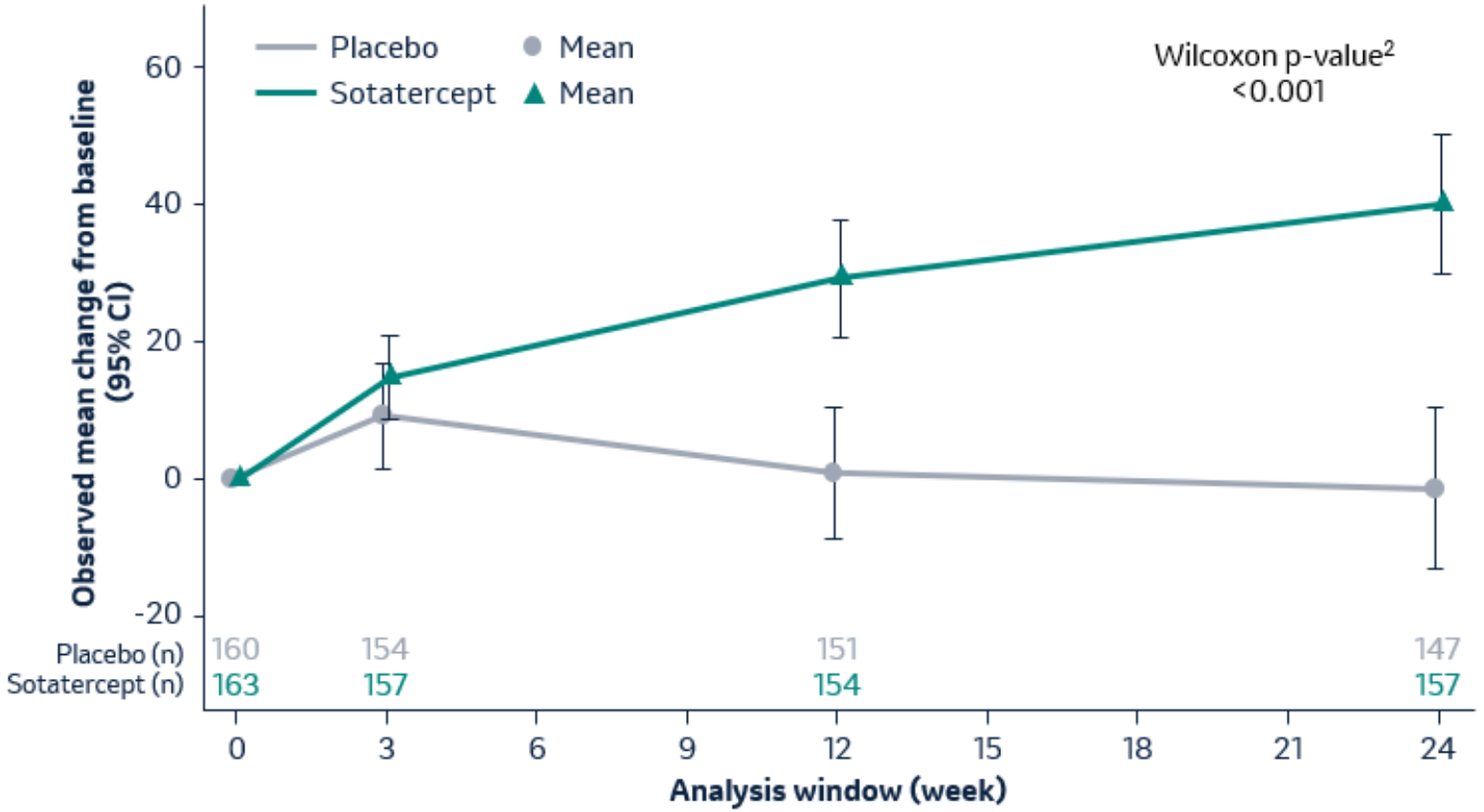
- WHO Group 1 PAH
- WHO FC II or III
- Adult ≥18 years
- Baseline PVR ≥400 dynes·sec·cm$^{-5}$ and PCWP or LVEDP ≤15 mmHg
- 6MWD 150 - 500 meters
- Stable treatment with background PAH therapy*




*Background PAH therapy included monotherapy, double therapy or triple therapy with one or more of the following:
An endothelin receptor antagonist, a phosphodiesterase-5 inhibitor, a soluble guanylate cyclase stimulator, and/or a prostacyclin (including intravenous).

# STELLAR: sotatercept significantly improved 6-minute walk distance at week 24

**Primary endpoint:** Change from baseline in 6MWD at week 24[1]




Statistically significant and clinically meaningful **improvement in 6MWD by 40.8 meters**[3]

1. The chart shows the observed mean change from baseline (95% CI). 2. From the aligned rank stratified Wilcoxon test with randomization factors as strata. 3. 40.8 meters is the pre-specified analysis of the Hodges-Lehmann location shift (95% CI: 27.5 to 54.1) represents the location shift from placebo estimate (median of the differences in change from baseline at week 24 [sotatercept vs. placebo]).

# Sotatercept delivered statistically significant improvement across 8 of 9 secondary endpoints

- ✓ **Multicomponent Improvement**[1]
- ✓ **Pulmonary Vascular Resistance**
- ✓ **NT-proBNP**
- ✓ WHO FC
- ✓ **TTCW or death**
- ✓ French low-risk-score
- ✓ PAH-SYMPACT® Physical Impacts
- ✓ PAH-SYMPACT® Cardiopulmonary
- PAH-SYMPACT® Cognitive/Emotional

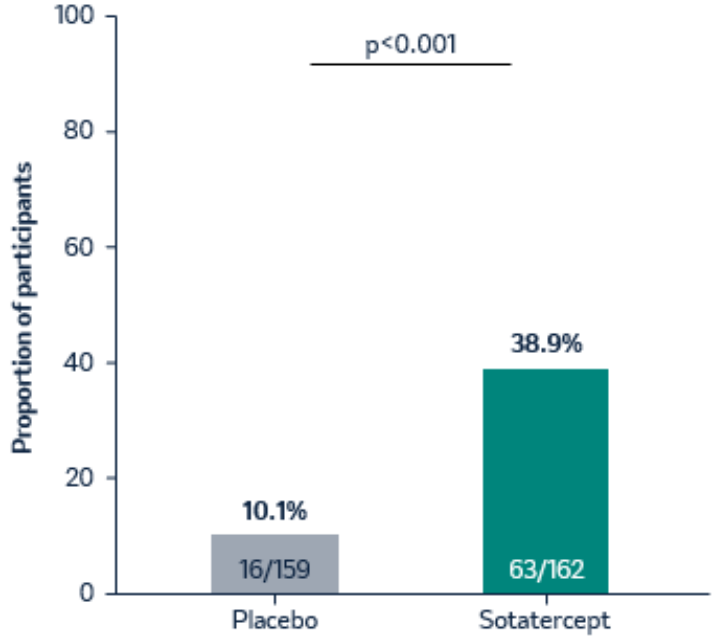


Achieved **significant clinical benefit** across hemodynamics, PAH disease severity, disease biomarkers, risk scores and patient-reported outcomes

1. Multicomponent improvement endpoint consists of improvement in 6MWD [increase of ≥ 30 meters] and improvement in NT-proBNP level [decrease of ≥ 30%] or maintenance/achievement of NT-proBNP level < 300 pg per milliliter and improvement in WHO functional class [shift from class III to II or I, or class II to I] or maintenance of class II; NT-proBNP: N-terminal pro-B-type natriuretic peptide; TTCW is time to clinical worsening or death.

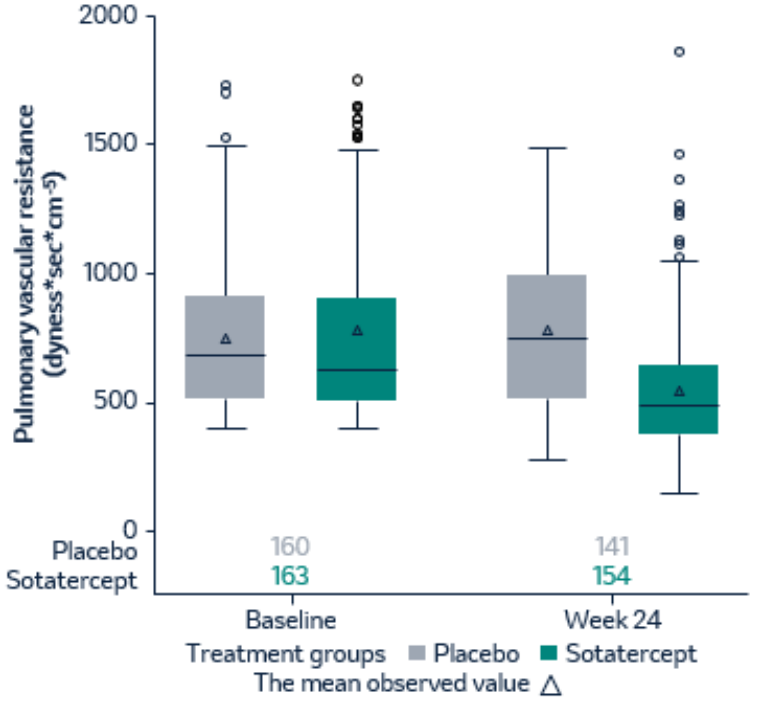
# Sotatercept demonstrated profound effect across secondary endpoints

## Multicomponent Improvement[1]



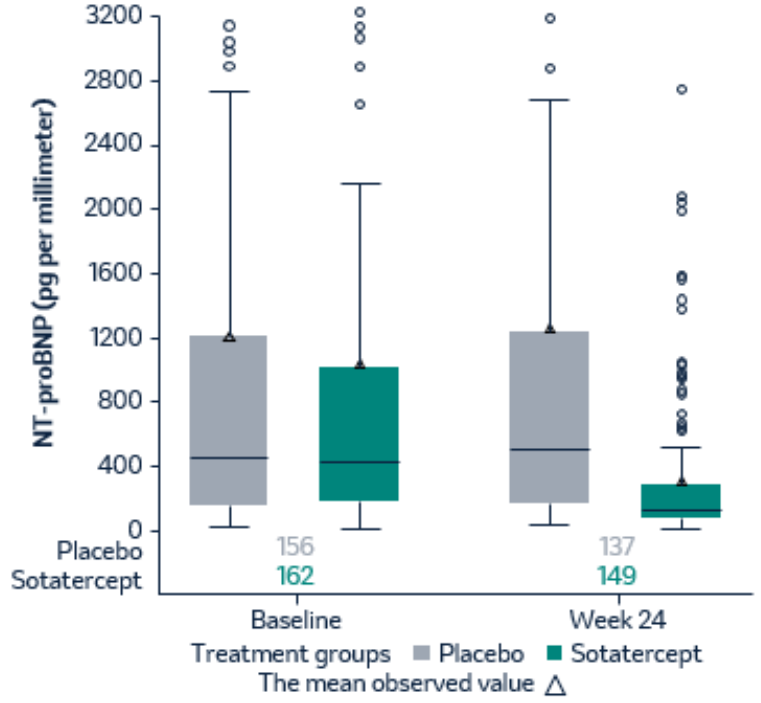

First intervention to demonstrate improvement on MCI composite endpoint at week 24

## Pulmonary Vascular Resistance




Profound reduction in PVR (-234.6 dyn·sec·cm$^{-5}$)

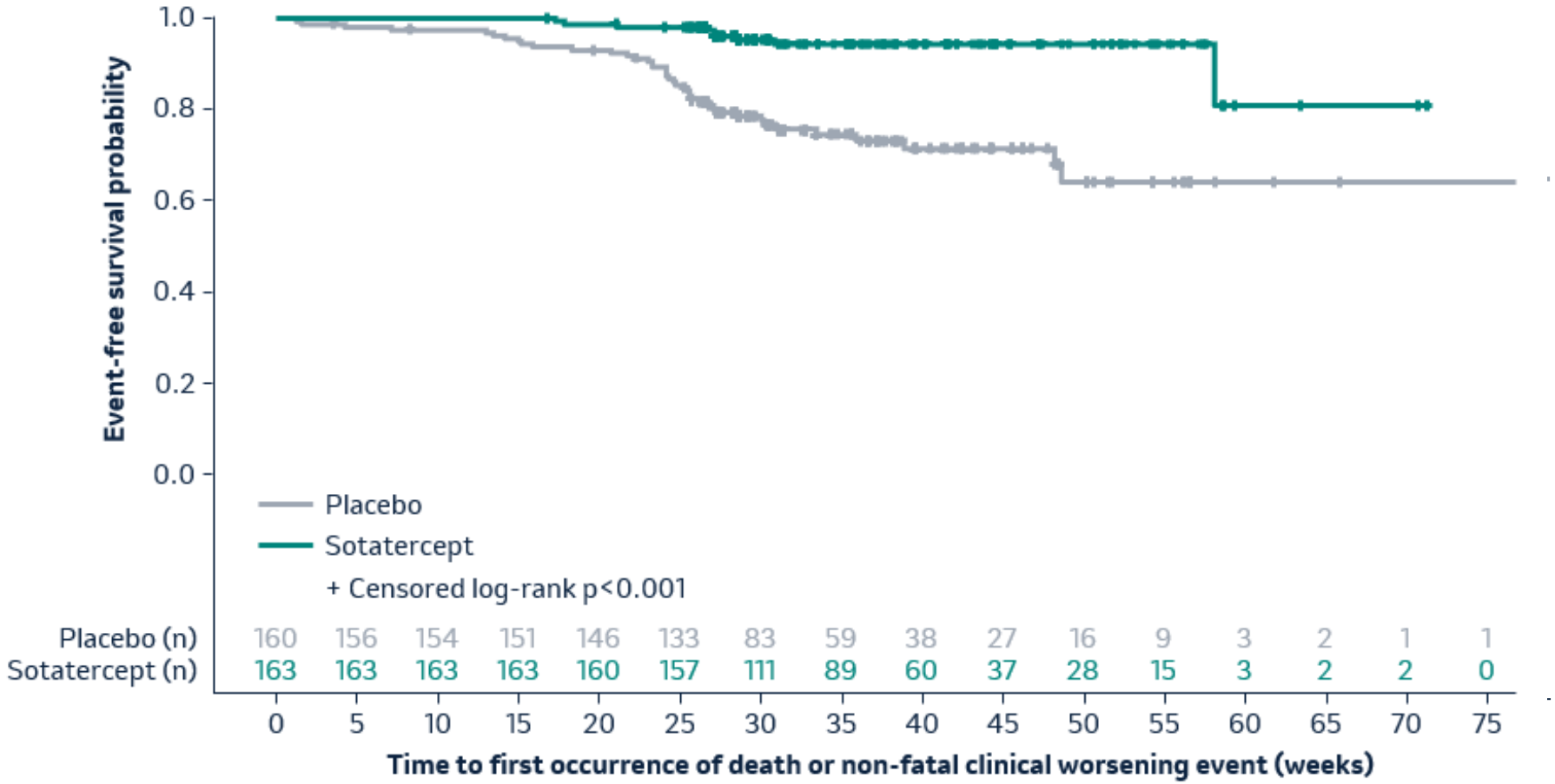
## NT-proBNP




Pronounced impact on right ventricular strain (-441.6 pg/ml)

1. Multicomponent improvement endpoint consists of improvement in 6MWD [increase of ≥ 30 meters] and improvement in NT-proBNP level [decrease of ≥ 30%] or maintenance/achievement of NT-proBNP level < 300 pg per milliliter and improvement in WHO functional class [shift from class III to II or I, or class II to I] or maintenance of class II;
NT-proBNP: N-terminal pro-B-type natriuretic peptide; TTCW is time to clinical worsening or death.

# STELLAR showed significant reduction in risk of death or clinical worsening events




After a median follow-up of 32.7 weeks across the treatment groups, **hazard ratio was 0.16** in the sotatercept group as compared to the placebo group[1]

1. Dates and times of reported adverse events were used by the adjudication committee to determine death or first non-fatal clinical worsening event. Patients could have more than one assessment for their first occurrence of non-fatal clinical worsening event or death. A single patient could have more than one non-fatal clinical worsening event but was only counted once for the time to event analysis. Hazard ratio of 0.16 (95% CI: 0.08 to 0.35)

# Sotatercept demonstrated numerically consistent effect across main components of clinical worsening

| | Placebo (N=160) | Sotatercept (N=163) |
|---|---|---|
| **Patients who died or experienced at least one clinical worsening event** | 26.3% | **5.5%** |
| **Assessment of first occurrence of death or non-fatal clinical worsening event[1]:** | | |
| Death as first event | 3.8% | **1.2%** |
| PAH-related hospitalization (≥24 hours) | 4.4% | **0** |
| Deterioration of PAH | 9.4% | **2.5%** |
| Need to initiate rescue therapy or to increase dose of infusion prostacyclin 10% or more | 10.6% | **1.2%** |
| Worsening-related listing for lung or heart-lung transplant | 0.6% | **0.6%** |
| Need for atrial septostomy | 0 | **0** |

1. Dates and times of reported adverse events were used by the adjudication committee to determine death or first non-fatal clinical worsening event. Patients could have more than one assessment for their first occurrence of non-fatal clinical worsening event or death. A single patient could have more than one non-fatal clinical worsening event but was only counted once for the time to event analysis. The study was not powered to demonstrate superiority across individual categories

# Sotatercept was generally well-tolerated

- AE profile for sotatercept generally **consistent with previous studies**
  - Increases in reported hemoglobin levels were manageable and not associated with treatment discontinuations
- **99% of patients received the higher dose** of 0.7 mg/kg during the study period
  - Of the 163 patients, 145 patients had no dose reductions or dose delays throughout the 24-week study period

| Number of patients with any | Placebo (N=160) | Sotatercept (N=163) |
|---|---|---|
| **Treatment-Emergent adverse events (TEAEs)** | 91.9% | 90.8% |
| **TEAEs related to treatment** | 26.9% | 47.2% |
| **TEAEs leading to treatment discontinuation** | 6.9% | 3.7% |
| **TEAEs leading to death** | 4.4% | 1.2% |
| **Serious TEAEs** | 27.5% | 22.1% |
| **Serious TEAEs related to treatment** | 1.3% | 1.8% |
| **TEAEs of interest**[1] | 45.0% | 59.5% |
| Bleeding events | 15.6% | 31.9% |
| Telangiectasia | 3.8% | 14.1% |
| Increased hemoglobin (increased hematocrit/RBC count) | 0 | 6.1% |
| Thrombocytopenia | 3.1% | 8.6% |
| Increased blood pressure | 0.6% | 4.3% |

Data cut-off date August 26, 2022.
1. TEAE of interest (bleeding events, cardiac events, embryo-fetal toxicity, hepatic toxicity, immunogenicity, increased blood pressure, increased hemoglobin, leukopenia, neutropenia, renal toxicity, suppression of follicle stimulating hormone, thrombocytopenia, thromboembolic events) and special interest (telangiectasia) were predefined parameters that were monitored to assess the overall safety profile of sotatercept. Only those TEAEs in which the point estimate(s) for the between-group differences excluded zero are shown in this table.
A TEAE has a start date on or after the first dose of treatment and up to 56 days after the last dose of treatment.

# Sotatercept is a potentially transformational treatment for PAH

## 6MWD

**+40.8 m**[1]

## PVR

**-26.7%**[2]

vs placebo +2.8%

## NT-proBNP

**-62.6%**[2]

vs placebo +16.2%

## TTCW

**84%**[3]

Risk reduction

1. Hodges-Lehmann location shift from placebo estimate. 2. Median, observed data only (no imputation). 3. TTCW is time to clinical worsening or death; HR of 0.16 (95% CI: 0.08 to 0.35)

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# ACC.23.WCC Data Highlights MK-0616 –

## an oral PCSK9 inhibitor for the treatment of hypercholesterolemia

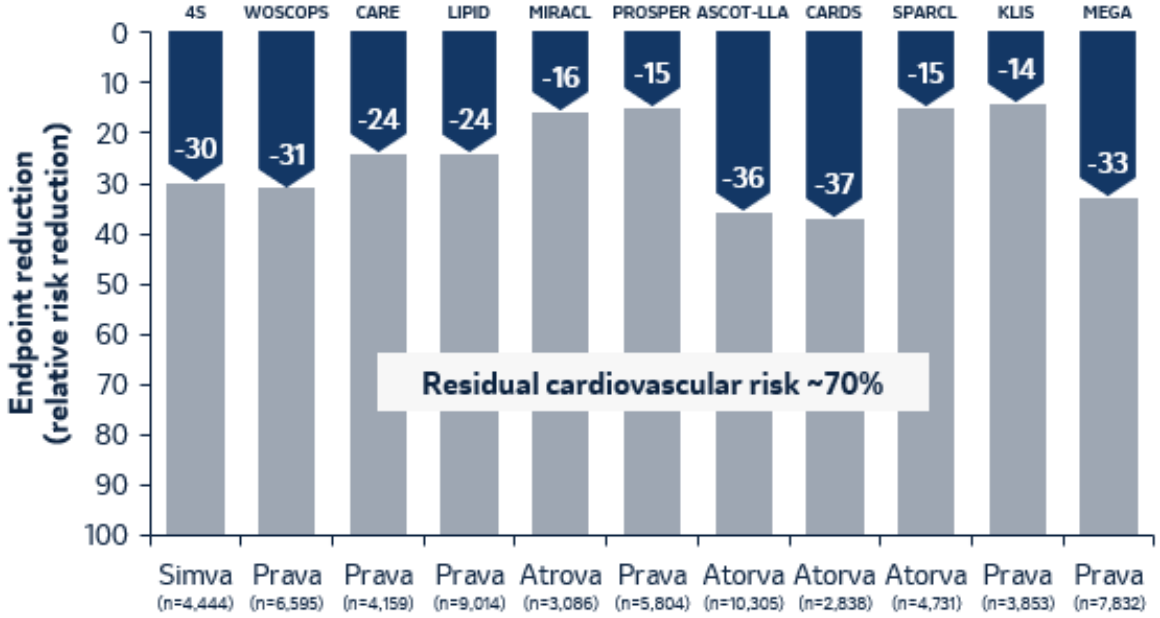
# Elevated LDL-C levels lead to ASCVD – a leading cause of mortality globally

## Atherosclerosis


of all cardiovascular disease deaths attributed to ASCVD[1]

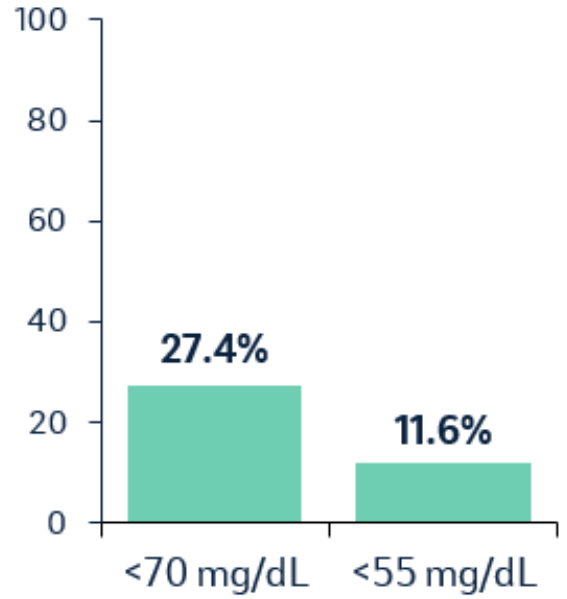
Large proportion of patients do not reach goals for LDL-C despite existing lipid-lowering therapies

## Residual cardiovascular risk




Even with current treatment options, the **residual CV risk remains high** if LDL-C is elevated[2]

## Patients at LDL-C goal




**Less than 30%** of treated patients with ASCVD achieve guideline recommended LDL-C reductions[3]

1. World Health Organization. Cardiovascular diseases (CVDs). https://www.who.int/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds). [Last accessed October 2021] 2. Chart derived from https://www.sciencedirect.com/science/article/pii/S0140673605673941?via% 3. Gu et.al., 2022 (analysis of 2019 Truven data) American Journal of Preventive Cardiology; Cannon et al

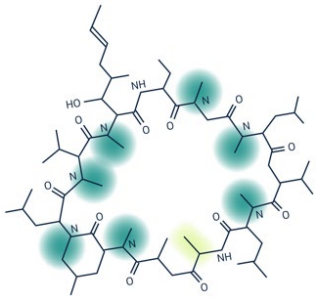
# Our goal is to create the most effective LDL-C lowering pill that is accessible to a broad range of patients

## PCSK9 pathway

- Genetic evidence **supports PCSK9 as a validated therapeutic target** in hypercholesterolemia
  - PCSK9 loss-of-function mutants: lower LDL-C, lower CV risk
  - PCSK9 gain-of-function mutants: higher LDL-C, higher CV risk
- Three injectable PCSK9 inhibitors on the market show **~50–60% LDL-C reduction** with limited real world clinical use
- Currently **no marketed oral PCSK9 inhibitors**

## MK-0616

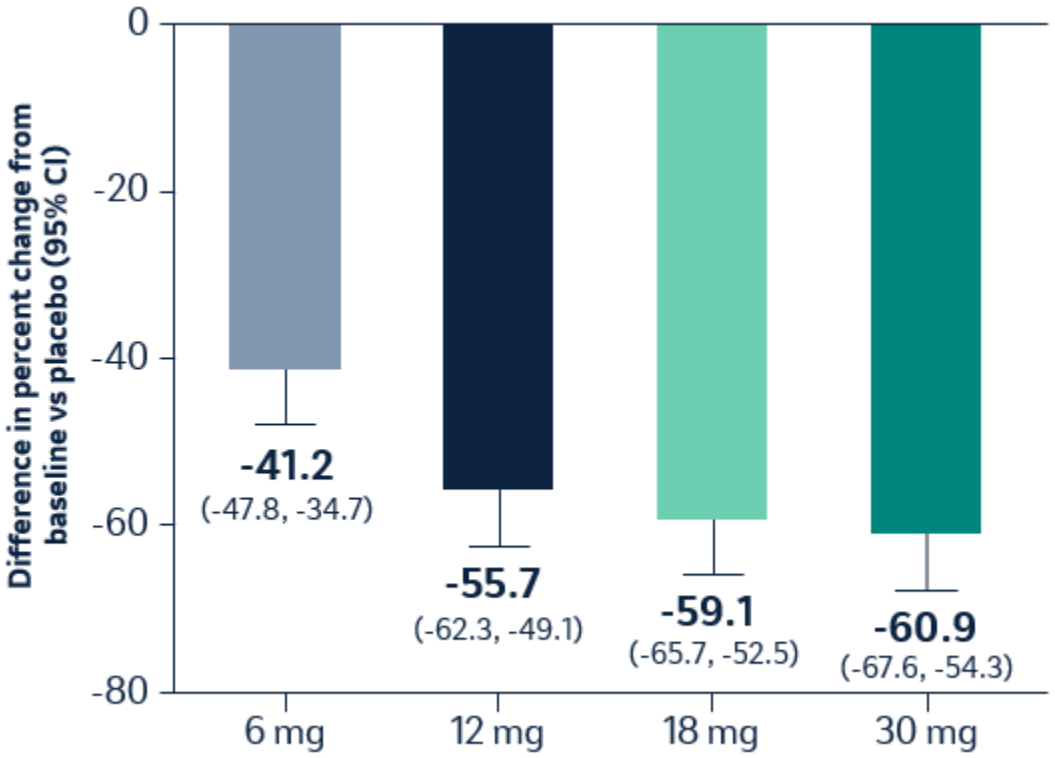
- **Novel macrocyclic peptide platform** offers "mAb-like" potency/selectivity at 1/100$^{th}$ of the molecular weight enabling targeting of "difficult-to-drug" proteins



Cyclic Peptide

- Binds to PCSK9 enzyme with a similar mechanism of action as the marketed mAbs but **administered orally once daily**
- Defined path toward providing a therapy that could achieve **broad global access**

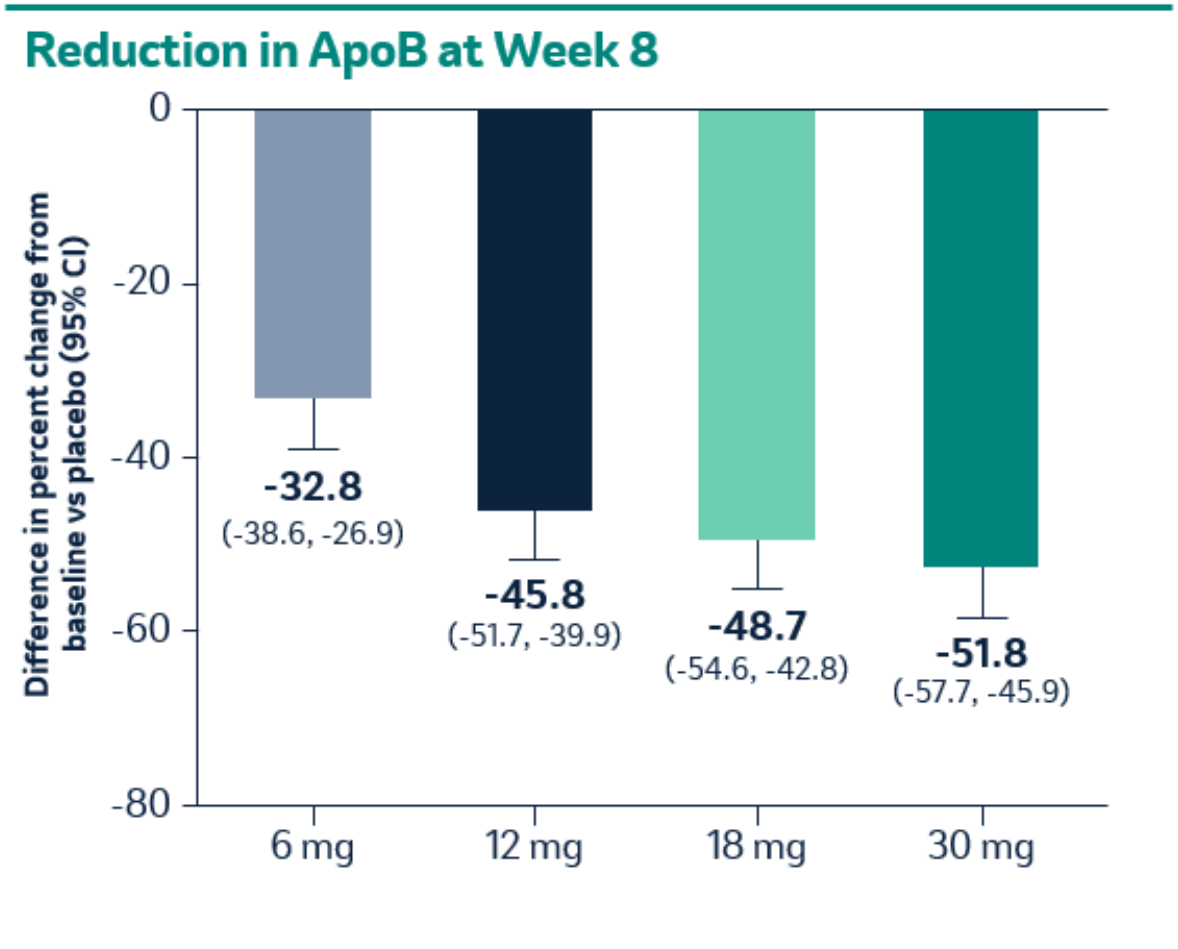
# Phase 2b trial showed significant reductions in LDL-C of up to 60.9%

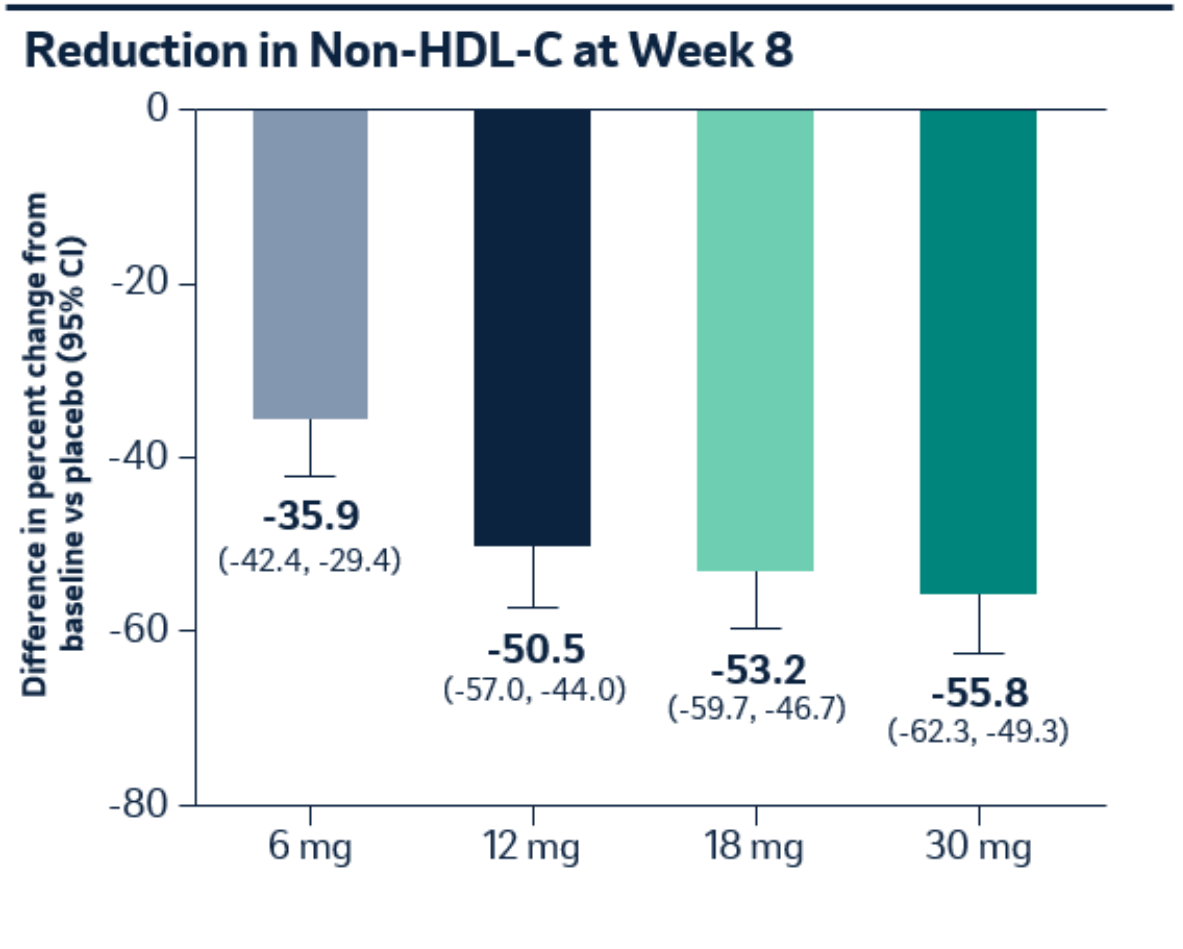



- **LDL-C reduction up to 60.9%** from baseline at week 8 compared to placebo
  - Statistically significant across all dose groups
- **Persistent response** over the 8-week treatment period
- Results generally **consistent across prespecified subgroups**
- **LDL-C reduction in line** with injectable PCSK9 inhibitors

Efficacy Population: All participants who received ≥1 dose, had ≥1 observation for the analysis endpoint, and had baseline data for those analyses that require baseline data. Participants were receiving a range of lipid-lowering therapies from no statin to high-intensity statin.

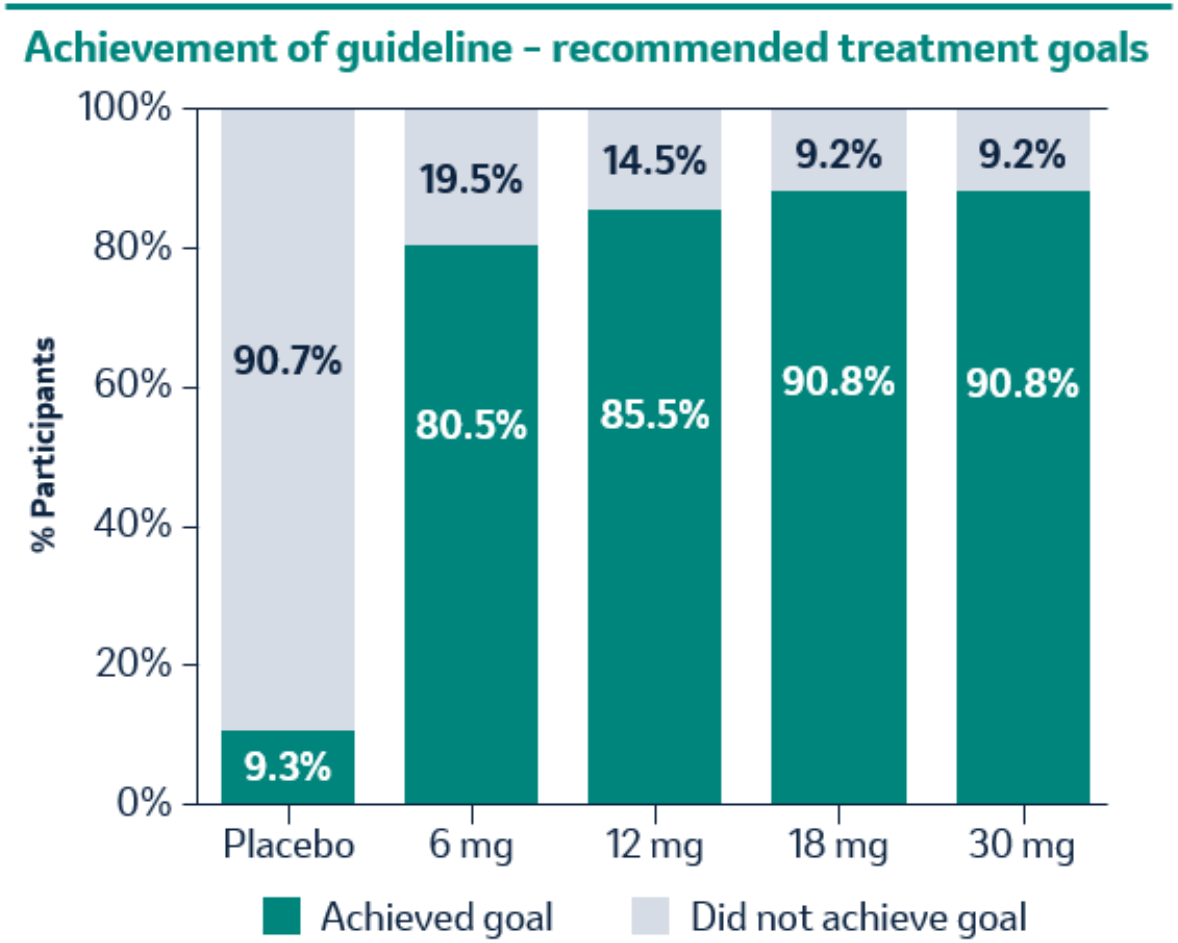
# Phase 2b study demonstrated strong results on key secondary endpoints




Efficacy Population: All participants who received ≥1 dose, had ≥1 observation for the analysis endpoint, and had baseline data for those analyses that require baseline data. Secondary endpoints not multiplicity controlled. Participants were receiving a range of lipid-lowering therapies from no statin to high-intensity statin.

# Vast majority of patients achieved LDL-C goals in Phase 2b study




- **Up to 90.8%** of participants **achieved goal** on MK-0616 at highest dose studied
  - Protocol defined LDL-C goals:
    - LDL-C <70 mg/dL (<1.81 mmol/L) in participants with clinical ASCVD
    - LDL-C <100 mg/dL (<2.59 mmol/L) in participants with high/intermediate ASCVD risk
    - LDL-C <130 mg/dL (<3.37 mmol/L) in participants with borderline ASCVD risk

Secondary endpoints not multiplicity controlled. Participants were receiving a range of lipid-lowering therapies from no statin to high-intensity statin.

# MK-0616 demonstrated a favorable safety and tolerability profile

- Over the 16-week study, including the 8-week treatment period and the 8-week safety follow-up period, **proportion of participants with AEs similar in all arms**
- Overall, there was no change in incidence of AEs based on dose and **no overall trends in AEs** across treatment groups

| | Placebo (n=75) | MK-0616 6 mg (n=77) | MK-0616 12 mg (n=76) | MK-0616 18 mg (n=76) | MK-0616 30 mg (n=76) |
|---|---|---|---|---|---|
| **≥1 Adverse Event (AE)** | 44.0% | 44.2% | 39.5% | 43.4% | 42.1% |
| **Discontinued intervention due to an AE** | 1.3% | 2.6% | 0.0% | 2.6% | 2.6% |
| **Serious AEs** | 0.0% | 1.3% | 3.9% | 2.6% | 2.6% |
| **Moderate or severe AEs** | 14.7% | 13.0% | 11.8% | 13.2% | 15.8% |
| **Study intervention-related AEs**[1] | 10.7% | 7.8% | 14.5% | 14.5% | 10.5% |
| **Deaths**[2] | 0.0% | 0.0% | 0.0% | 1.3% | 0.0% |
| **Most common AEs, n(%)** | | | | | |
| COVID-19 | 9.3% | 7.8% | 2.6% | 6.6% | 5.3% |
| Diarrhea | 1.3% | 6.5% | 1.3% | 3.9% | 1.3% |
| Dyspepsia | 1.3% | 1.3% | 6.6% | 3.9% | 0.0% |

1. Deemed by the investigator to be possibly, probably, or definitely related to study medication. 2. There was one death that was the result of a traffic accident.

# MK-0616 effect size is similar to injectable PCSK9 inhibitors

## LDL-C

up to
**60.9%**
reduction

## ApoB

up to
**51.8%**
reduction

## Non-HDL-C

up to
**55.8%**
reduction

## LDL-C goal achievement

up to
**90.8%**
attainment

MK-0616 results shown are at highest dose studied.

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# Closing Remarks

# Sotatercept demonstrated profound clinical improvement for patients

**First in-class** activin signaling inhibitor

Potential **new class of treatments** designed to modulate vascular proliferation and target **underlying disease** pathophysiology

**Sotatercept** has the potential to profoundly **change the treatment** of PAH

Offered clinical benefits **across sub-groups**[1] **of background therapy** or disease severity

Generally well-tolerated with safety profile consistent with prior studies

1. Sub-group summary provided in Appendix.

# Ambition to develop best-in-class portfolio to treat pulmonary hypertension

## Sotatercept

- **Expand** across the PAH patient journey
- **Deepen** impact with additional outcomes data
- **Extend** to patients with PH secondary to left heart disease

## MK-5475 (inhaled sGC stimulator)

- Potential **first** pulmonary selective vasodilator
- Potential **best-in-class** benefit/ risk profile
- **Ease of use**
- **Extend** to patients with PH secondary to COPD

## Pulmonary hypertension

### WHO Group 1
Pulmonary arterial hypertension

**Sotatercept**

STELLAR

HYPERION

ZENITH

**MK-5475**

INSIGNIA PAH

Phase 2/3 Study of an Inhaled sGC Stimulator in PAH

### WHO Group 2
PH due to left-sided heart disease

**Sotatercept**

CADENCE

### WHO Group 3
PH due to lung disease or hypoxia

**MK-5475**

INSIGNIA PH-COPD

A Study of the Efficacy and Safety of MK-5475 (an Inhaled sGC Stimulator) in Adults With PH-COPD

# Results from Phase 2 study for MK-0616 support further development into Phase 3

Potential **first oral** PCSK9 inhibitor

Phase 2 data demonstrate lipid lowering **in line with injectables**

**MK-0616** has potential to **improve LDL-C goal attainment** and allow for **lipid lowering reduction**

Goal to ensure **broader global access**

Phase 2 data demonstrate **favorable tolerability profile**

# Results from Phase 2 study for MK-0616 provide a defined path toward a globally accessible treatment option

## LDL-C Reduction Indication

- Lipid study in broad patient population (including secondary prevention and intermediate to high-risk primary prevention) for initial lipid indication
- Separate lipid study focused in HeFH patients

## CV Risk Reduction Indication

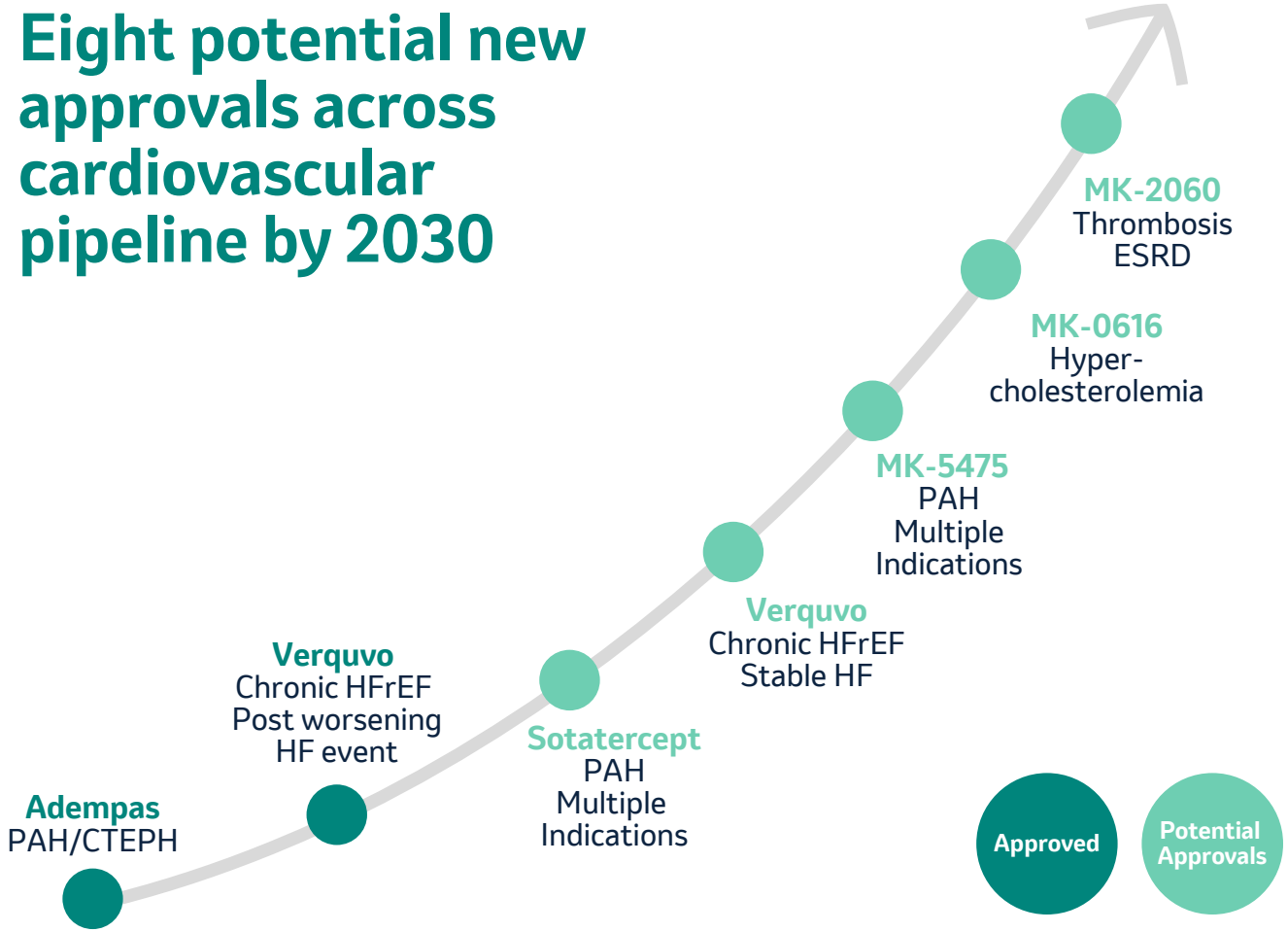
- Broaden claim and target population through CVOT to start in parallel to lipid studies

## Supportive Studies

- To establish MK-0616 as the preferred add-on to statins and support guideline positioning and implementation

# Cardiovascular portfolio well positioned for growth into the next decade

## Eight potential new approvals across cardiovascular pipeline by 2030



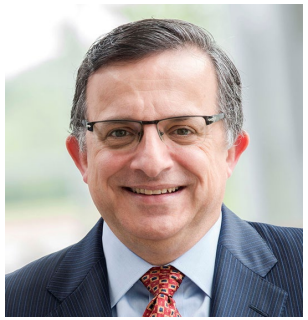

## CV Pipeline Potential[1]

**>$10billion**
**in peak revenue approaching mid-2030s**

1. Includes the expanded indication for Verquvo plus all indications where studies are underway or being planned for clinical assets MK-7962, MK-5475, MK-2060, MK-0616.

# Q&A

**Dr. Dean Li**
President,
Merck Research
Laboratories



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**Peter Dannenbaum**
VP, Investor Relations

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# Appendix

# Acronyms

**6MWD** = 6 Minute Walking Distance
**AE** = Adverse Event
**ApoB** = Apolipoprotein
**ASCVD** = Atherosclerotic Cardiovascular Disease
**ASE** = Asymptotic Standard Error
**AVG** = Arteriovenous Graft
**BMP** = Bone Morphogenetic Protein
**CHF** = Congestive Heart Failure
**CI** = Confidence Interval
**COPD** = Chronic Obstructive Pulmonary Disease
**CTEPH** =Chronic Thromboembolic Pulmonary Hypertension
**CV** = Cardiovascular
**CVOT** = Cardiovascular outcomes study
**ESRD** = End Stage Renal Disease
**FC** = Functional Class
**HeFH** = Heterozygous familial hypercholesterolemia
**HFrEF** = Heart Failure with Reduced Ejection Fraction
**HF** = Heart Failure
**LDL-C** = Low-Density Lipoprotein Cholesterol
**LVEDP** = Left Ventricular End-Diastolic Pressure
**mAb** = monoclonal antibody
**MCI** = Multicomponent Improvement
**Non-HDL-C =** Non-High-Density Lipoprotein Cholesterol
**NT-proBNP** = N-Terminal-pro hormone B-type Natriuretic Peptide
**PAH** = Pulmonary Arterial Hypertension
**PCWP** = Pulmonary Capillary Wedge Pressure
**PH =** Pulmonary Hypertension
**POC** = Proof of Concept
**PVR** = Pulmonary Vascular Resistance
**sGC** = Soluble Guanylate Cyclase
**TEAE** = Treatment Emergent Adverse Event
**TTCW** = Time to Clinical Worsening