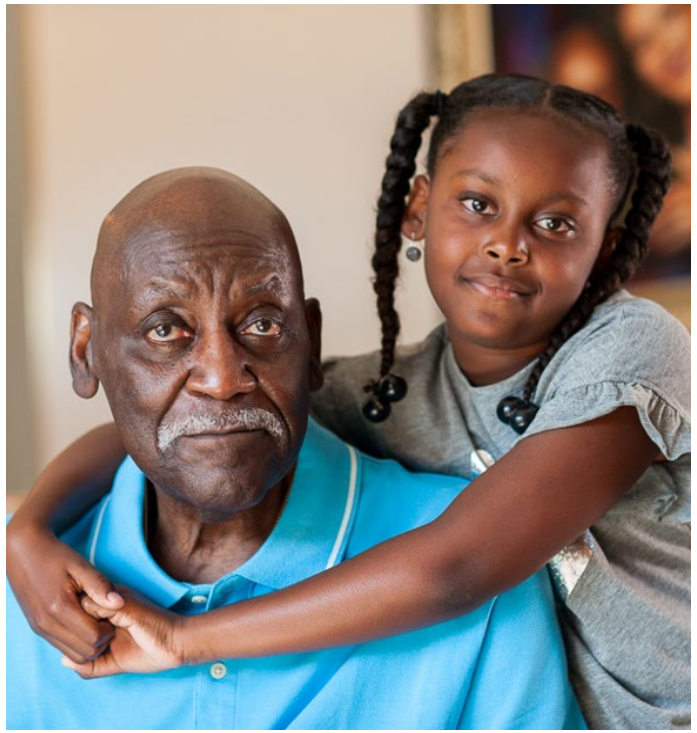
bridgebio

hope through
rigorous science

ATTRibute-CM Phase 3 Topline Results

July 17, 2023



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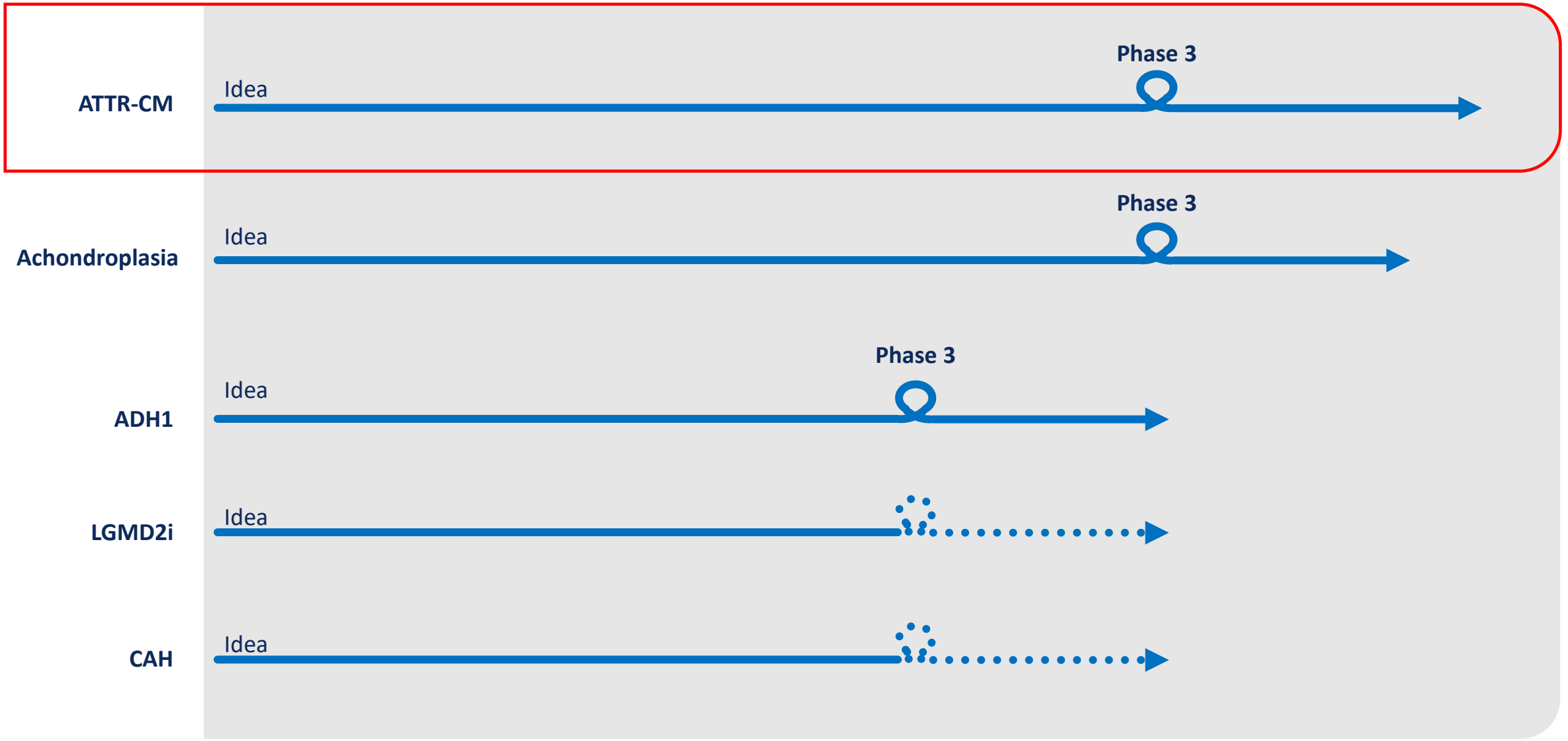
Discussion topics

1	**Introduction & Overview**	Neil Kumar, PhD Chief Executive Officer
2	**ATTRibute-CM Phase 3 Topline Results**	Jonathan Fox, MD, PhD Chief Medical Officer, Cardiorenal
3	**Next Steps**	Uma Sinha, PhD Chief Scientific Officer
4	**Commercial Launch Plans**	Matt Outten, MBA Chief Commercial Officer
5	**Q&A Session**	

(acoramidis for ATTR-CM)

A sincere THANK YOU to patients and families, advocates, physicians, clinical research staff, and collaborating research partners

Program context



Acoramidis was designed to achieve maximal stabilization and preserve native TTR

Design Objectives	Rationale
1 **Maximize TTR stabilization/minimize toxic monomer**	• Strong genotype/phenotype correlation between TTR instability and disease severity[1] • Dose-dependent improvements in both TTR stabilization and clinical outcomes demonstrated by tafamidis in ATTR-CM[2] • Extent of TTR stabilization or knockdown associated with degree of clinical benefit in ATTR-PN[3-6]
2 **Preserve circulating native TTR**	• TTR has been highly conserved throughout evolution[7] • TTR is an abundant plasma protein with relatively rapid turnover requiring sustained metabolic energy expenditure

We plan to enter the ATTR-CM market with acoramidis, a next generation, more potent TTR stabilizer

TTR = Transthyretin; ATTR-CM = TTR amyloid cardiomyopathy.

[1]Hammarstrom, P et al., PNAS. 2002;99:16427-16432. [2]Damy, T., et al., Eur J Heart Fail. 2021;23(2):277-285. [3]Coelho, T. et al., Neurology. 2012;79:785–792. [4]Berk, JL et al , JAMA. 2013;310:2658-2667. [5]Adams, DA. et al., N Engl J Med. 2018;379:11-21. [6]Benson, M.D., et al., N Engl J Med. 2018;379:22-31. [7]Richardson SJ, et al. Front Endocrinol. 2015;5:1-9.

Acoramidis is a next generation stabilizer that employs multiple strategies to maximize potency

Disease mechanism

Native TTR circulates in blood as a tetramer

~130 known destabilizing mutations

Protective T119M mutation

Dissociation into monomers initiates pathogenesis

Monomers aggregate, causing disease

Therapeutic hypothesis

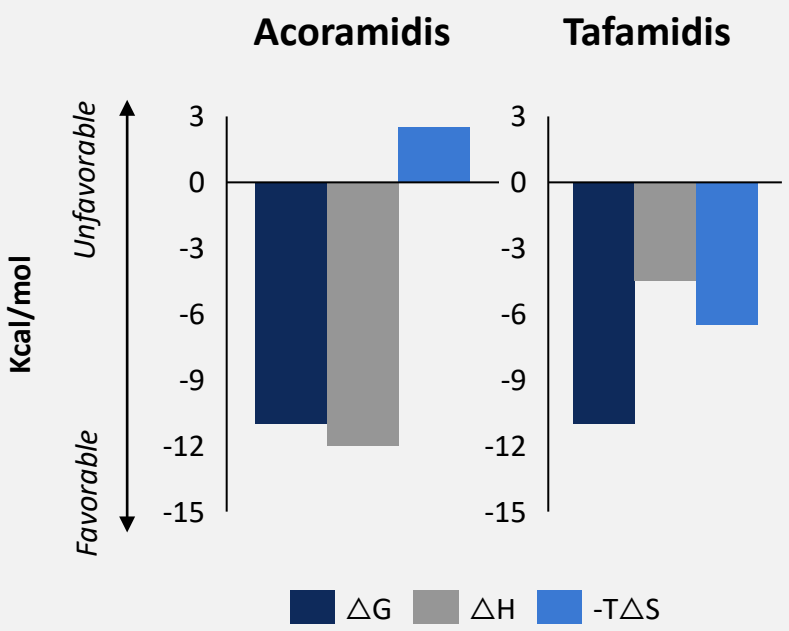
Acoramidis was designed to mimic protective T119M mutation. Acoramidis sees more target (superior free fraction), binds more target (superior kd2), and glues the target together stronger (enthalpic binding mode).[1,2]

Acoramidis is an investigational molecule. The safety and efficacy have not been established by regulatory authorities.

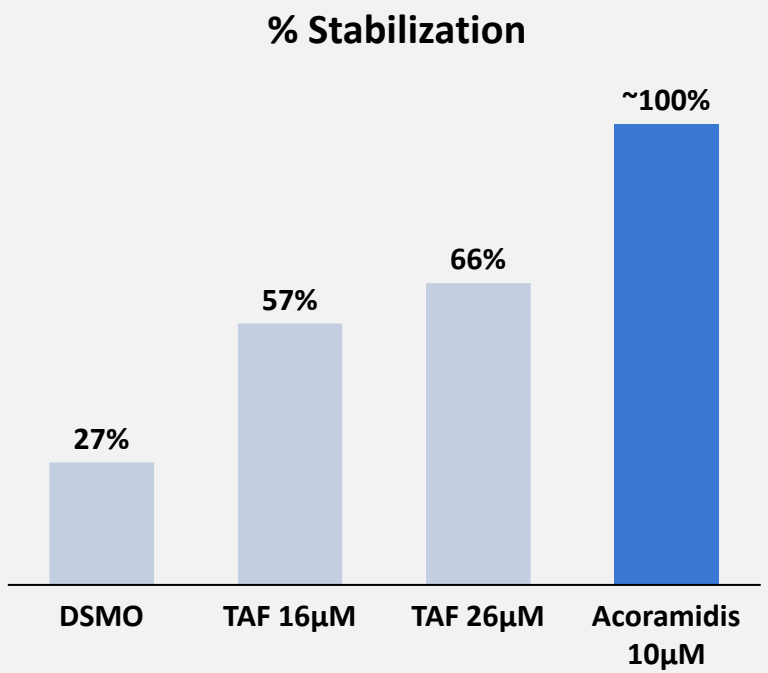
[1]Data on File. [2]Miller, M. et al. J Med Chem. 2018;61:7862-7876.

Data supporting more potent TTR stabilization

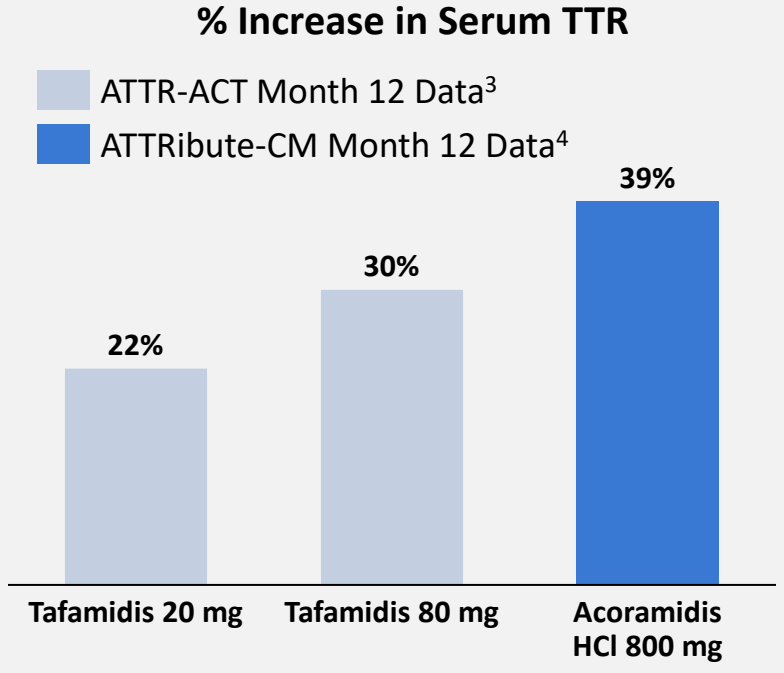
Superior Binding to TTR in vitro[1]
facilitated by enthalpic interactions



Near-Complete TTR Stabilization[2]
at target trough clinical concentrations



Rapid, durable increases in serum TTR
an in vivo marker of native tetramer stability



[1]Miller, M. et al. J Med Chem. 2018;61:7862-7876. [2]Ji, A.X., et al. American Heart Association Scientific Sessions, 2019. [3]Estimated from Damy, T., et al., Eur J Heart Fail. 2021;23(2):277-285. [4]BridgeBio Part A press release, December 27, 2021.
Note: Direct cross-study comparisons may suggest misleading similarities or differences. The values shown are directional and do not report robust comparative analysis.

Unprecedented and consistent benefit on survival and morbidity

Best Case Target Clinical Profile	Outcome Observed
Achieve statistical significance on primary endpoint: p-value <0.04	✓ Primary endpoint met (p<0.0001)
Unprecedented survival: Highest ever 30-month survival rate on drug (>80%) with clinically meaningful separation from placebo	✓ 81% 30-month survival on acoramidis ✓ 6.4% absolute & 25% relative risk reduction compared to placebo
Best-in-class CVH data: Profound reduction in event rates	✓ 50% relative risk reduction for cumulative frequency of CVH (p<0.0001)
Win Ratio better than 1.7: Significant impact on mortality and morbidity	✓ Win Ratio = 1.8
Best-in-class treatment effect on serum biomarkers: NT-proBNP, serum TTR, TTR stabilization	✓ Clinically and statistically significant (p<0.0001) benefit on NT-proBNP and serum TTR; sustained impact on TTR stabilization

CVH = Cardiovascular-related hospitalization; NT-proBNP = N-terminal prohormone of brain natriuretic peptide.

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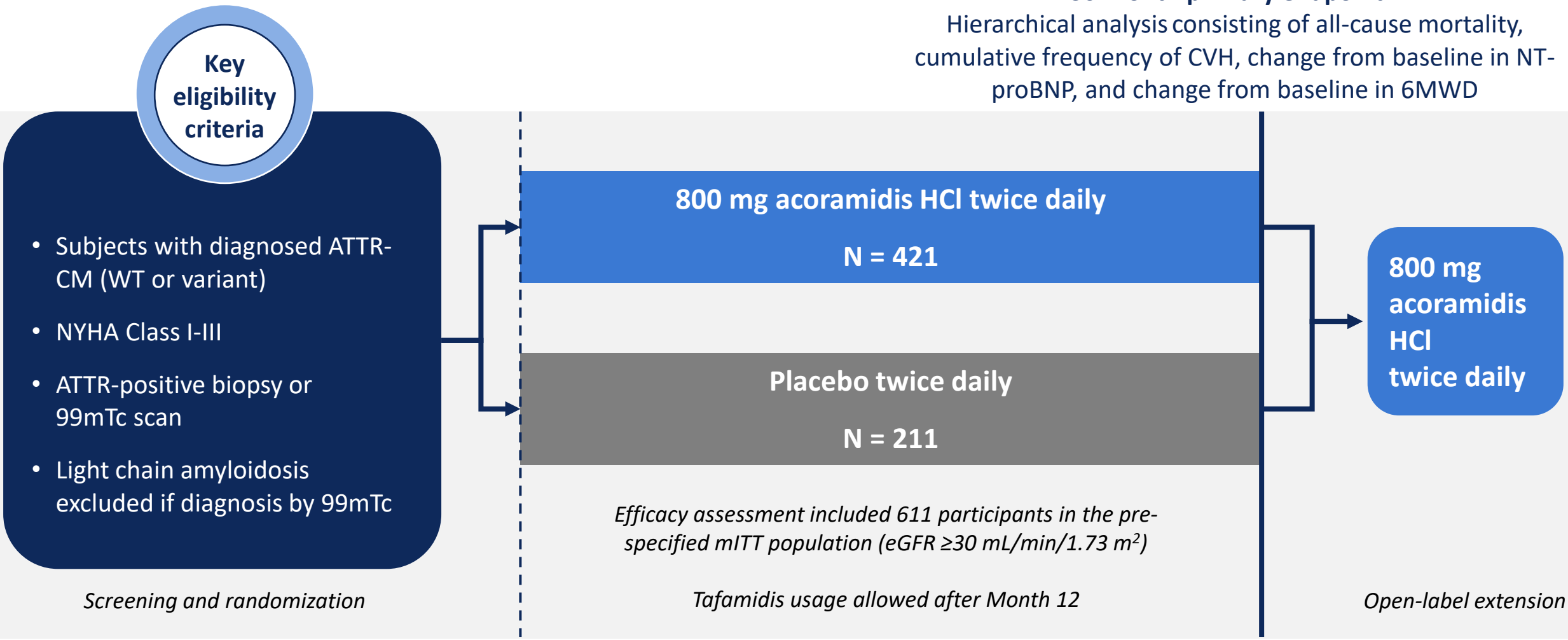
ATTRibute-CM study design[1,2]



6MWD = Six-minute walk distance; NYHA = New York Heart Association; 99mTc = Technetium labeled pyrophosphate (PYP) or bisphosphonate (e.g., DPD); mITT = Modified intent-to-treat. eGFR = Estimated glomerular filtration rate.

[1]ClinicalTrials.gov identifier: NCT03860935. [2]Gillmore JD et al. Circulation. 2019;140(1):14214. Oral poster presented at AHA. [3]Primary analysis assessed using the Finkelstein-Schoenfeld method.

Highly statistically significant result achieved on primary and select secondary endpoints

Primary endpoint[1]	p-value
Hierarchical analysis consisting of: • All-cause mortality[2] • Cumulative frequency of CVH • Change from baseline in NT-proBNP • Change from baseline in 6MWD	p<0.0001
Win Ratio	1.8

58% of ties broken by first two components of Win Ratio analysis

Select secondary endpoints	p-value
Cumulative frequency of CVH[3]	p<0.0001
Change from baseline in 6MWD[4]	p<0.0001
Change from baseline in KCCQ-OS[4]	p<0.0001
Change from baseline in serum TTR[4]	p<0.0001
Change from baseline in NT-proBNP[5]	p<0.0001
All-cause mortality[2,6]	p=0.057

KCCQ-OS = Kansas City cardiomyopathy questionnaire overall summary score.

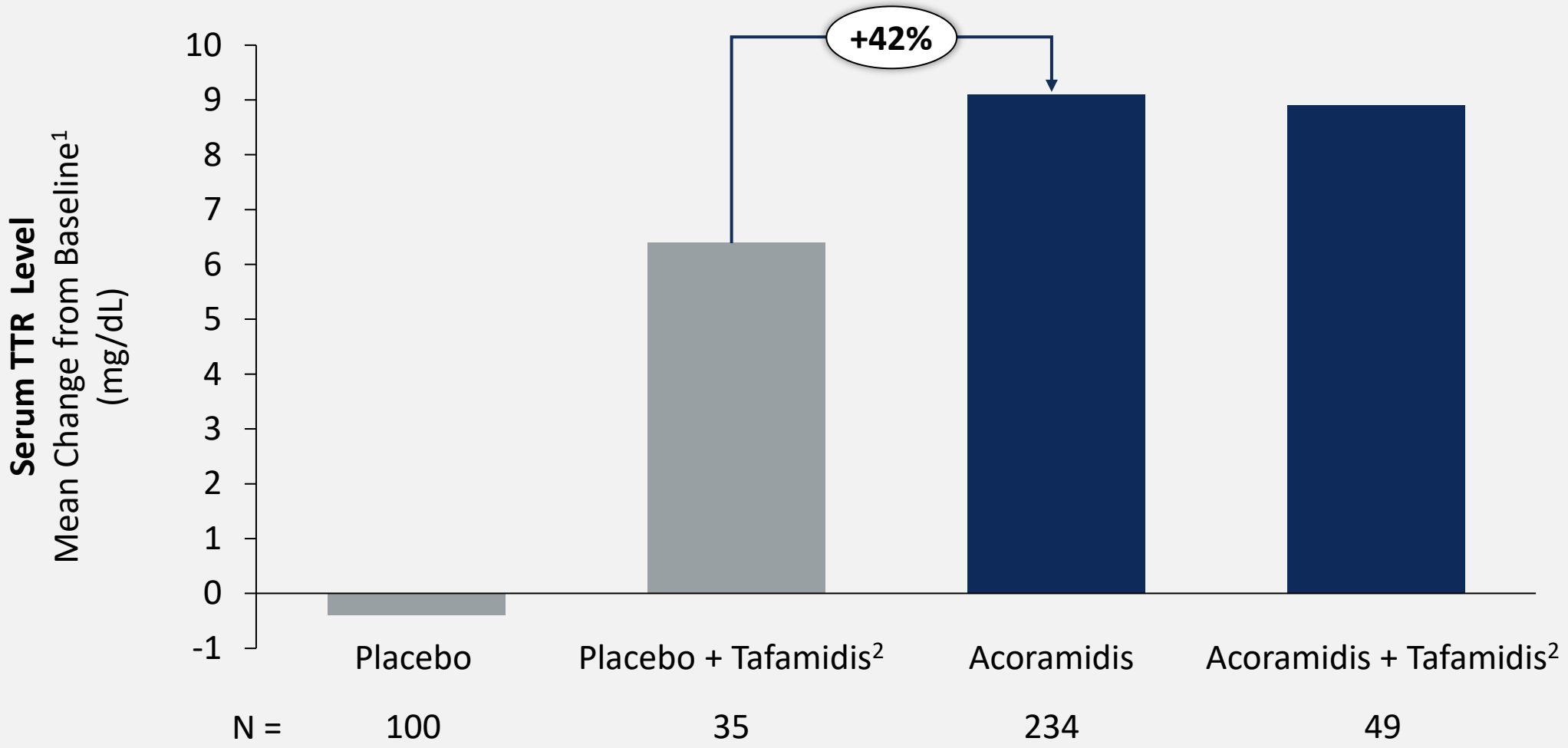
[1]Primary analysis assessed using the Finkelstein-Schoenfeld method. [2]Heart transplant and implantation of cardiac mechanical assistance device were treated as death for this analysis. [3]Negative binomial regression with treatment group, stratification factors and the offset term is used to analyze the cumulative frequency of adjudicated CV-related hospitalization.[4]Least squares mean difference change from baseline at 30 months. [5]Ratio of adjusted geometric mean fold change from baseline at 30 months. [6]Assessed by Cochran-Mantel-Haenszel test; p=0.15 as assessed by Cox Proportional Hazard Model.

No safety signals of potential clinical concern identified

Subjects with one or more event(s)	Acoramidis N=421 N (%)	Placebo N=211 N (%)
Any treatment-emergent adverse events (TEAEs)	413 (98.1%)	206 (97.6%)
TEAE with fatal outcome	60 (14.3%)	36 (17.1%)
TEAE leading to hospitalization	212 (50.4%)	128 (60.7%)
TEAE leading to study drug discontinuation	39 (9.3%)	18 (8.5%)
Any treatment-emergent serious adverse events (SAEs)	230 (54.6%)	137 (64.9%)
Treatment-emergent SAEs leading to study drug discontinuation	21 (5.0%)	15 (7.1%)
Severe TEAEs[1]	157 (37.3%)	96 (45.5%)

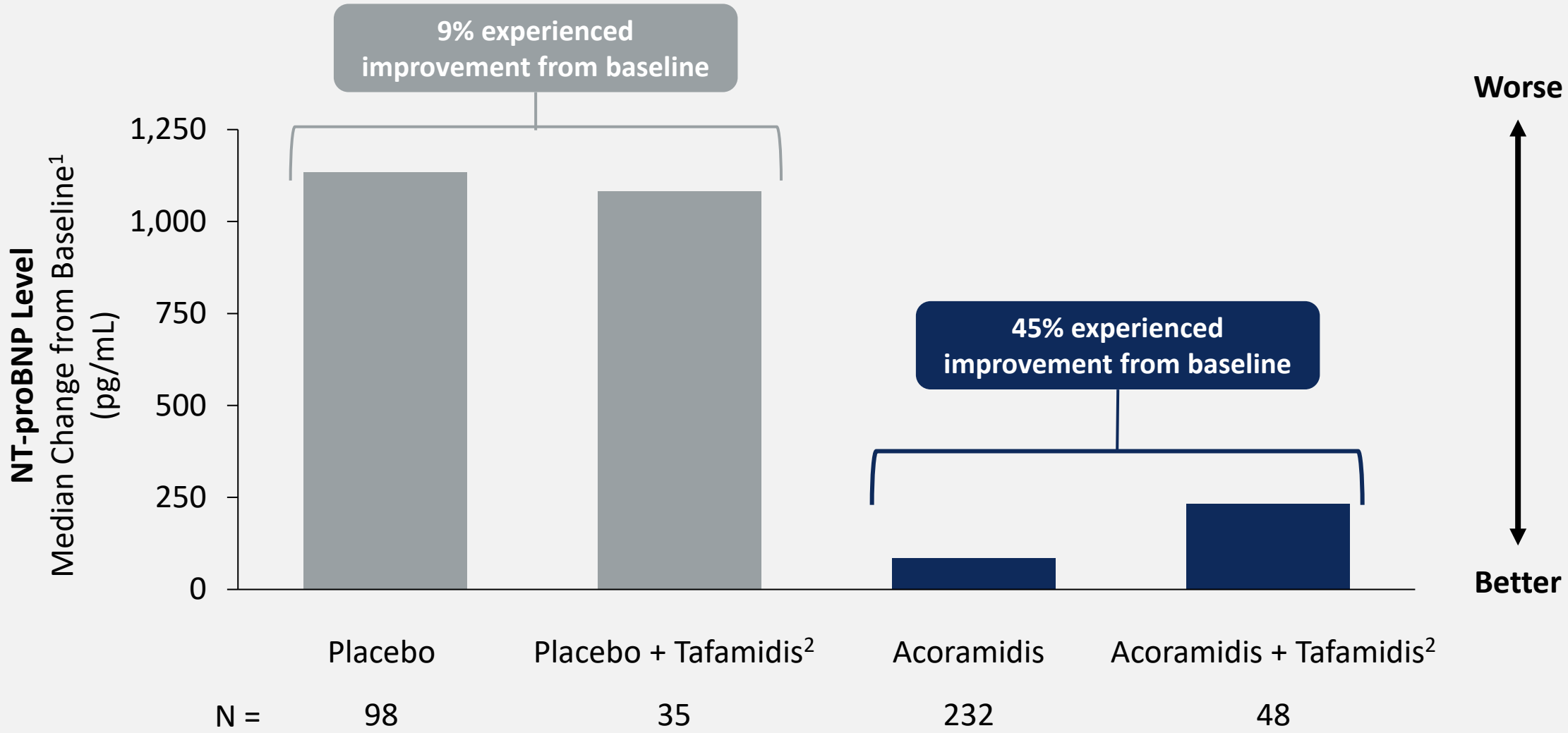
All Adverse Events (AEs) occurring during the treatment period are considered treatment-emergent adverse events (TEAEs). Serious Adverse Event (SAE) meets seriousness criteria.
[1]Severity as assessed by the investigator.

Exploratory post hoc analysis: serum TTR levels



[1]Mean change from baseline in serum TTR at Month 30 in mITT population. [2]Mean exposure on tafamidis = 11 months in mITT population.

Exploratory post hoc analysis: median NT-proBNP



[1]Median change from baseline in NT-proBNP at Month 30 in mITT population. [2]Mean exposure on tafamidis = 11 months in mITT population.

Summary of topline results

Acoramidis was observed to consistently outperform placebo on survival and established measures of ATTR-CM morbidity

- Unprecedented **30-month survival of >80%** for a targeted intervention in ATTR-CM
- Achieved primary endpoint with highly statistically significant result with **Win Ratio of 1.8**
- **6.4% ARR & 25% RRR** in all-cause mortality
- **50% RRR** for cumulative frequency of CVH
- Well-tolerated with no safety signals of potential clinical concern

ARR = Absolute risk reduction. RRR = Relative risk reduction.

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First regulatory submission planned for year-end 2023

Present ATTRibute-CM Primary Results
European Society of Cardiology 2023
August 27th, 2023

File New Drug Application (NDA) with FDA
End of 2023

Submit additional regulatory filings (EMA & others)
2024

Execute lifecycle management
Initiate primary prevention study (ACT-EARLY)
2024

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Commercial launch plans

We have a world-class commercial team and we are prepared to go to market

- 20+ FTEs (Pharmacyclics, Vertex, and Schering Plough alumni) and a distinctive commercial advisory board, inclusive of Fred Hassan, Jennifer Cook and Jim Robinson
- Have initiated discussions with key partners (payers and distributors) to bring this drug to patients

Our goal is to continue working closely with current and future partners to bring this next generation stabilizer to as broad a patient and provider community as possible

- Access
- Global reach

More details on commercial execution to come

ATTRibute-CM Phase 3 Topline Results

Q&A Session