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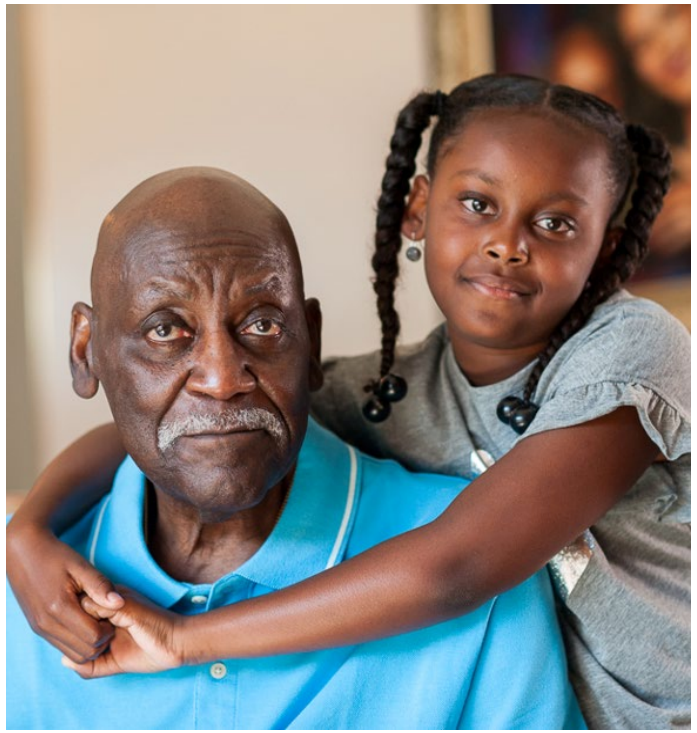
Detailed Results of the ATTRibute-CM Phase 3 Study

August 28, 2023



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Discussion topics

1. Molecular hypothesis
2. ATTRibute-CM Phase 3 results
3. Context for clinical findings
4. Next steps
5. Q&A session

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

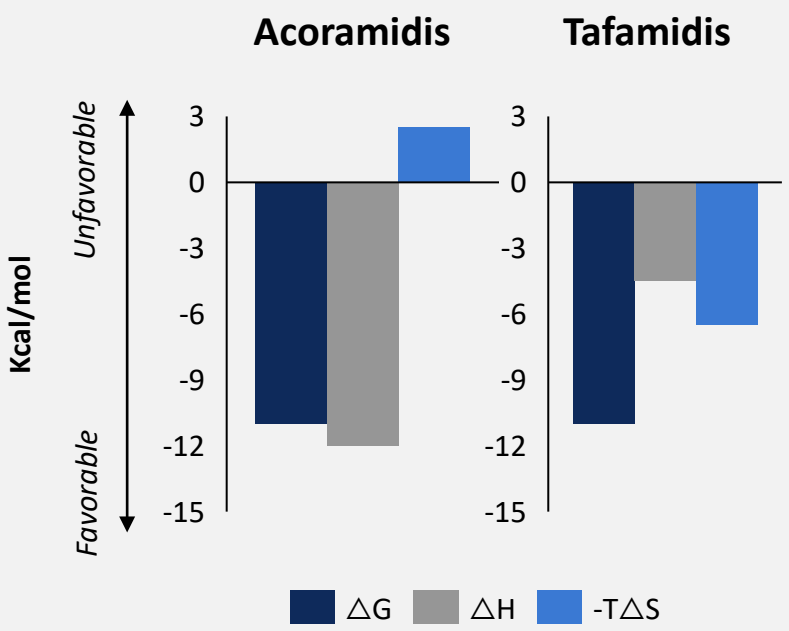
Acoramidis was designed to mimic protective T119M mutation. Acoramidis sees more target (superior free fraction), binds more target (superior kd2), and glues the target together stronger (enthalpic binding mode).[1,2] Phase 3 results confirm differential stabilization via effects on serum TTR.

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

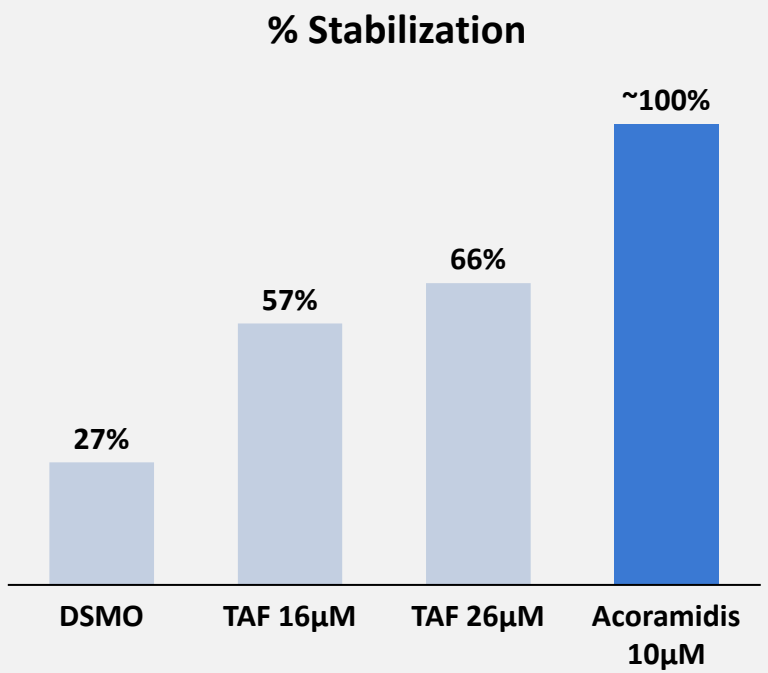
[1]Data on File. [2]Miller, M. et al. J Med Chem. 2018;61:7862-7876.

Data supporting more potent TTR stabilization

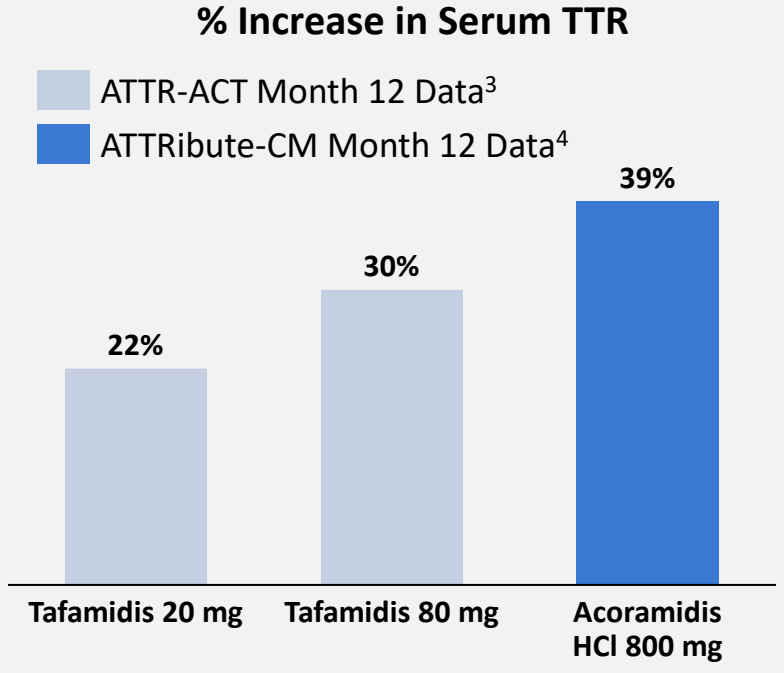
Superior Binding to TTR in vitro[1]
facilitated by enthalpic interactions



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

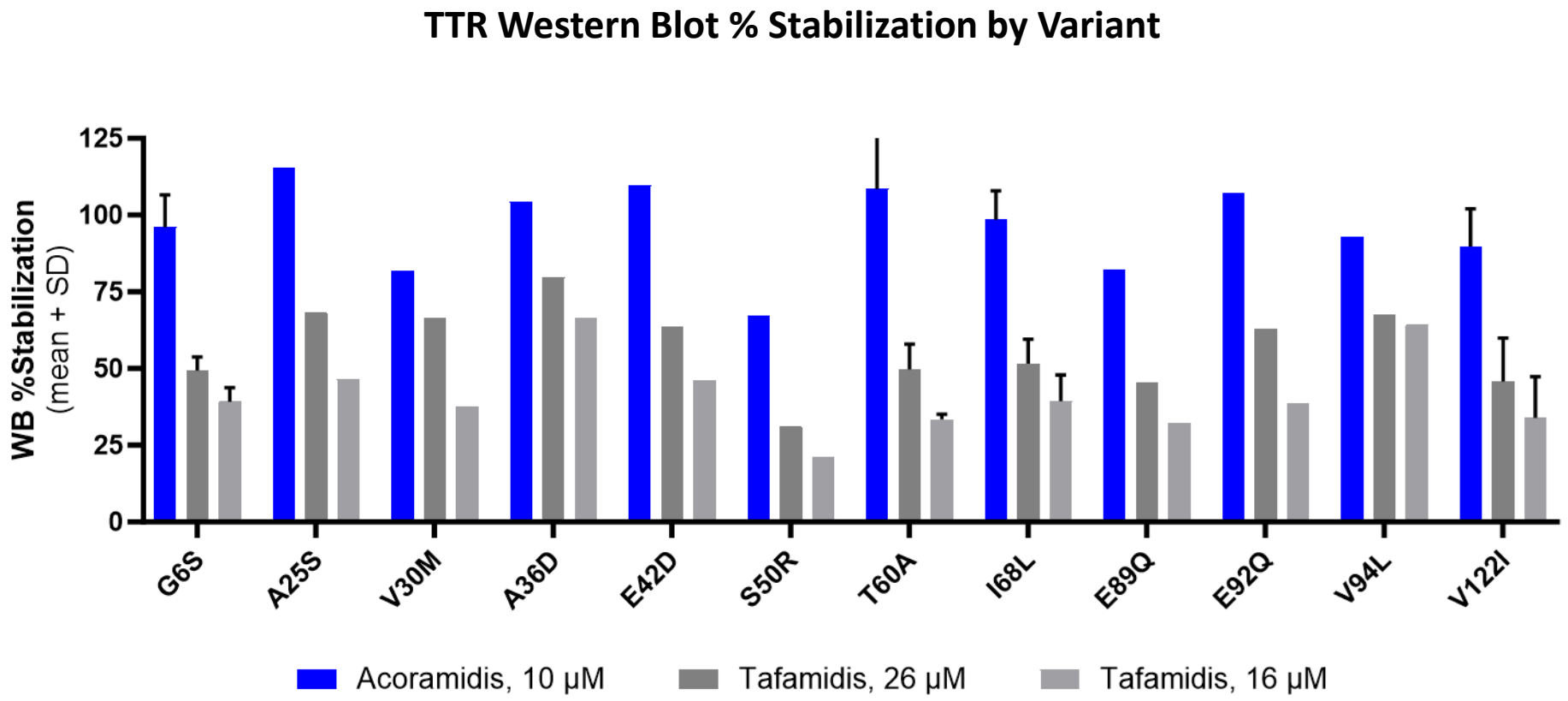


Rapid, durable increases in serum TTR
an in vivo marker of native tetramer stability



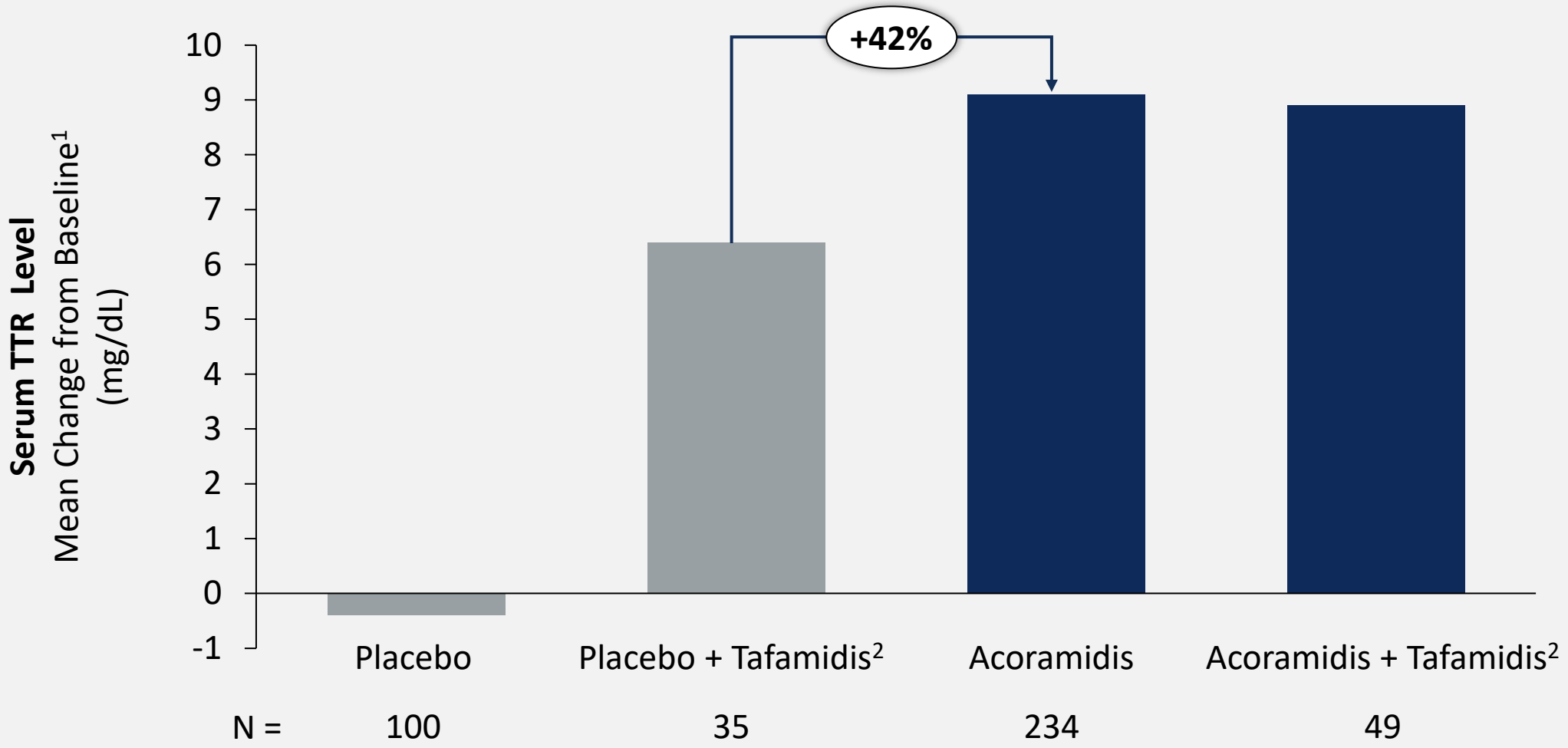
[1]Miller, M. et al. J Med Chem. 2018;61:7862-7876. [2]Ji, A.X., et al. American Heart Association Scientific Sessions, 2019. [3]Estimated from Damy, T., et al., Eur J Heart Fail. 2021;23(2):277-285. [4]BridgeBio Part A press release, December 27, 2021.
Note: Direct cross-study comparisons may suggest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

Acoramidis demonstrated pan-variant TTR stabilization to a greater extent than tafamidis



Source: Ji A. X. et al., ESC 2023: "Acoramidis Produces Near-Complete TTR Stabilization in Blood Samples from Patients with Variant Transthyretin Amyloidosis that is Greater than that Achieved with Tafamidis"
Note: SD shown for measurements with two or more samples

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.

Discussion topics

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2. **ATTRibute-CM Phase 3 results**
3. Context for clinical findings
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Efficacy and Safety of Acoramidis in Transthyretin Amyloid Cardiomyopathy

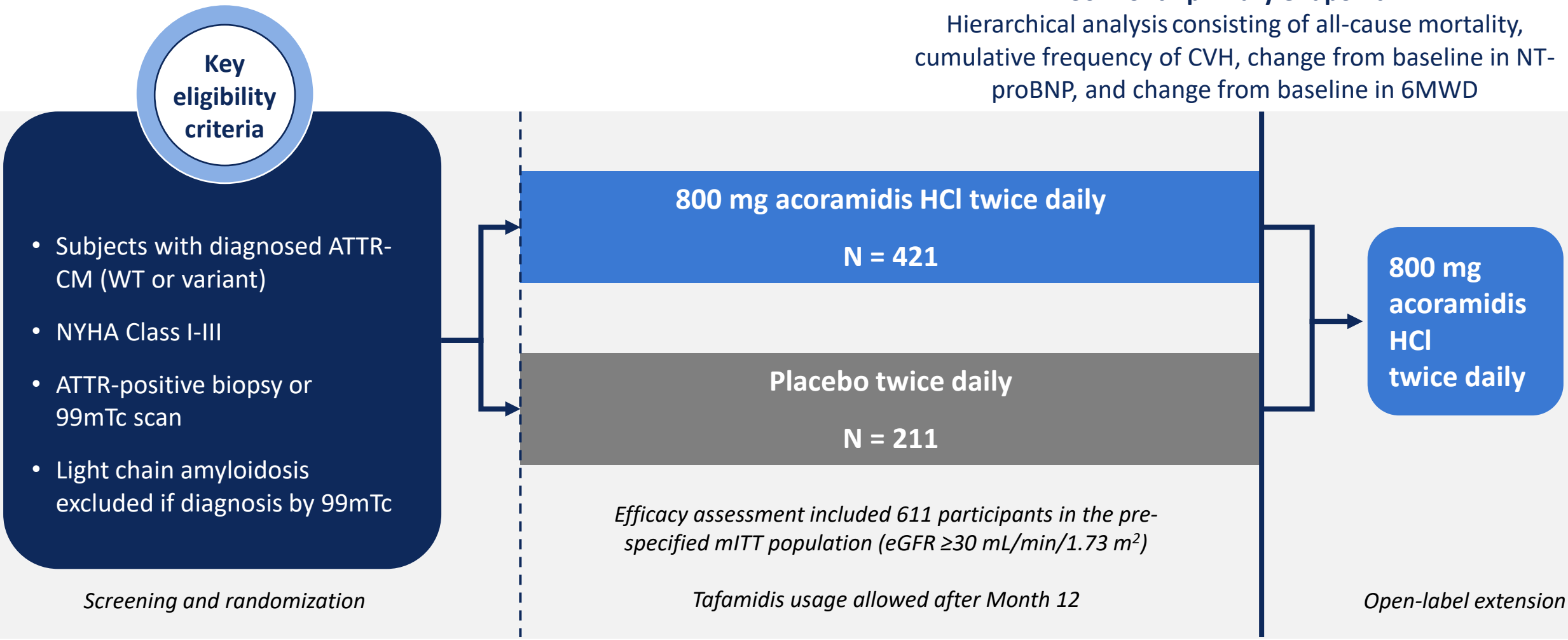
Results of the ATTRibute-CM Trial

Julian D. Gillmore,[1] Daniel P. Judge,[2] Francesco Cappelli,[3] Marianna Fontana,[1] Pablo Garcia-Pavia,[4,5,6] Simon Gibbs,[7] Martha Grogan,[8] Mazen Hanna,[9] James Hoffman,[10] Ahmad Masri,[11] Mathew S. Maurer,[12] Jose Nativi-Nicolau,[13] Laura Obici,[14] Frank Rockhold,[15, 16] Keyur B. Shah,[17] Prem Soman,[18] Jyotsna Garg,[15] Karen Chiswell,[15] Haolin Xu,[15] Xiaofan Cao,[19] Ted Lystig,[19] Uma Sinha,[19] and Jonathan C. Fox[19]

[1]National Amyloidosis Centre, Division of Medicine, University College London, Royal Free Hospital, London, UK; [2] The Medical University of South Carolina, Charleston, SC, USA, [3]Tuscan Regional Amyloidosis Centre, Careggi University Hospital, Florence, Italy;[4]Heart Failure and Inherited Cardiac Diseases Unit, Department of Cardiology, Hospital Universitario Puerta der Hierro Majadahonda, CIBERCV, Manuel de Falla 2, 28222 Madrid, Spain; [5]Universidad Francisco de Vitoria (UFV), Pozuelo de Alarcon, Spain; [6]European Reference Network for Rare, Low Prevalence and Complex Diseases of the Heart-ERN GUARD-Heart; [7]The Victorian and Tasmanian Amyloidosis Service, Department of Haematology, Monash University Eastern health Clinical School, Box Hill, Victoria, Australia; [8]Department of Cardiovascular Diseases, Mayo Clinic, Rochester, MN, USA; [9] Department of Cardiovascular Medicine, Cleveland Clinic, Cleveland, OH, USA; [10] Sylvester Comprehensive Cancer Center, University of Miami, Miami, FL, USA; [11]Cardiac Amyloidosis Program, Knight Cardiovascular Institute, Oregon Health and Science University, Portland, OR, USA; [12]Cardiac Amyloidosis Program, Division of Cardiology, Columbia College of Physicians and Surgeons, New York NY, USA; [13] Amyloidosis Program, Department of Transplant,, Mayo Clinic, Jacksonville, FL, USA; [14]Amyloidosis Research and Treatment Center, IRCCS Fondazione Policlinico San Matteo, Pavia, Italy; [15]Duke Clinical Research Institute, Durham, NC, USA; [16]Duke University Medical Center, Durham, NC, USA; [17]The Pauley Heart Center, Virginia Commonwealth University, Richmond, VA, USA; [18]Divison of Cardiology, University of Pittsburgh Medical Center, Pittsburgh, PA, USA; [19]BridgeBio Pharma, San Francisco, CA, USA

ATTRibute-CM study design[1,2]

30-month primary endpoint[3]:
Hierarchical analysis consisting of all-cause mortality, cumulative frequency of CVH, change from baseline in NT-proBNP, and change from baseline in 6MWD



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

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.

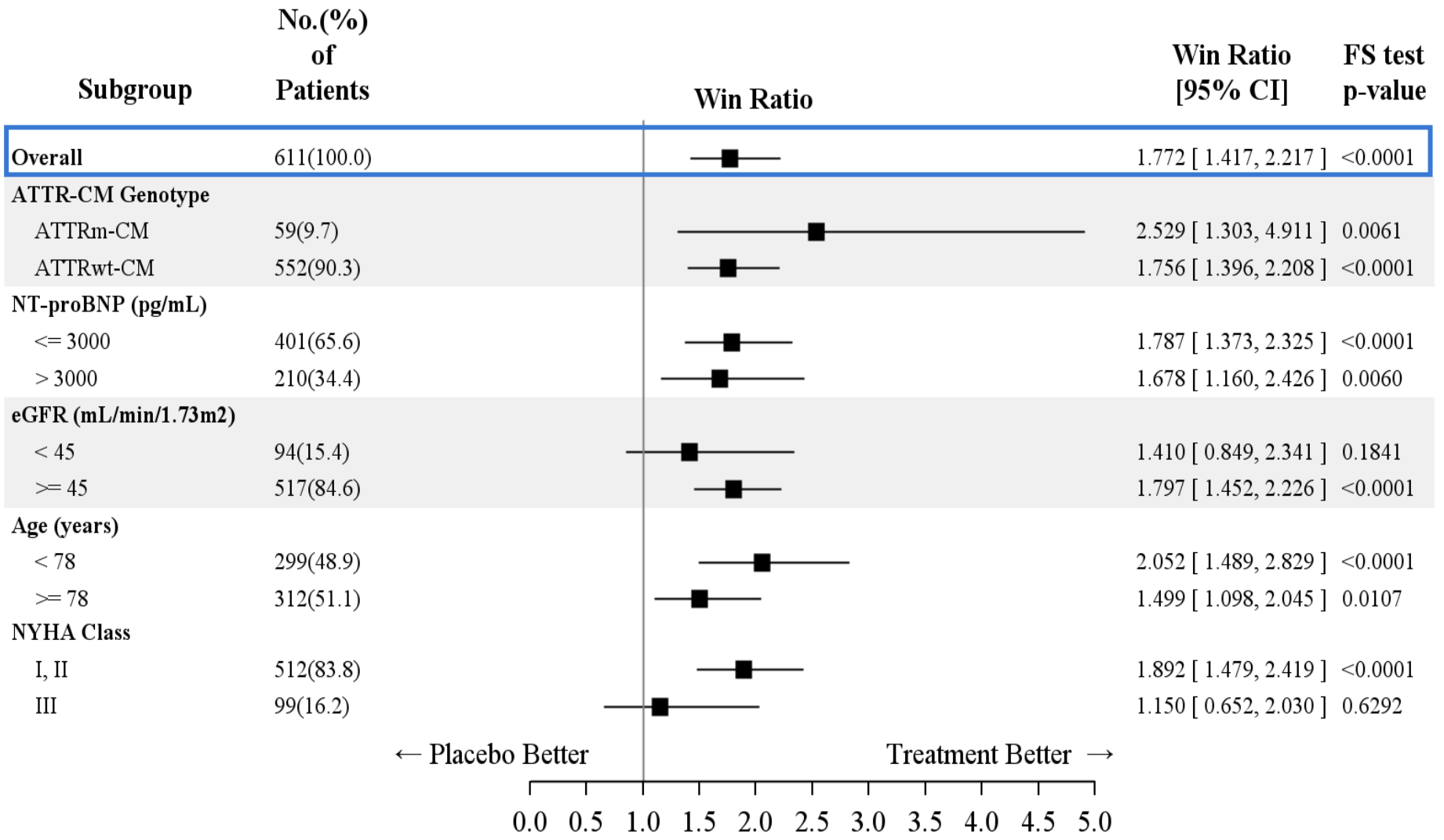
ATTRibute-CM: Baseline Demographic Characteristics

Characteristic	Acoramidis (N=421)	Placebo (N=211)
Age (years), mean (SD)	77.4 (6.5)	77.1 (6.8)
Male sex, n (%)	384 (91.2)	186 (88.2)
ATTRwt-CM, n(%)	380 (90.3)	191 (90.5)
NT-proBNP (pg/mL), median (IQR)	2326 (1332, 4019)	2306 (1128, 3754)
eGFR (mL/min/1.73m^2), mean (SD)	60.9 (18.2)	61.0 (18.7)
TTR (mg/dL), mean (SD)	23.2 (5.6)	23.6 (6.1)
KCCQ-OS, mean (SD)	71.5 (19.4)	70.3 (20.5)
6MWD (m), mean (SD)	361.2 (103.7)	348.4 (93.6)
Concomitant tafamidis use, n (%)*	61 (14.5)	46 (21.8)

ATTRwt-CM = transthyretin amyloidosis wild-type cardiomyopathy; NT-proBNP = N-terminal pro-B-type natriuretic peptide; TTR = transthyretin; KCCQ-OS = Kansas City Cardiomyopathy Questionnaire Overall Summary Score.

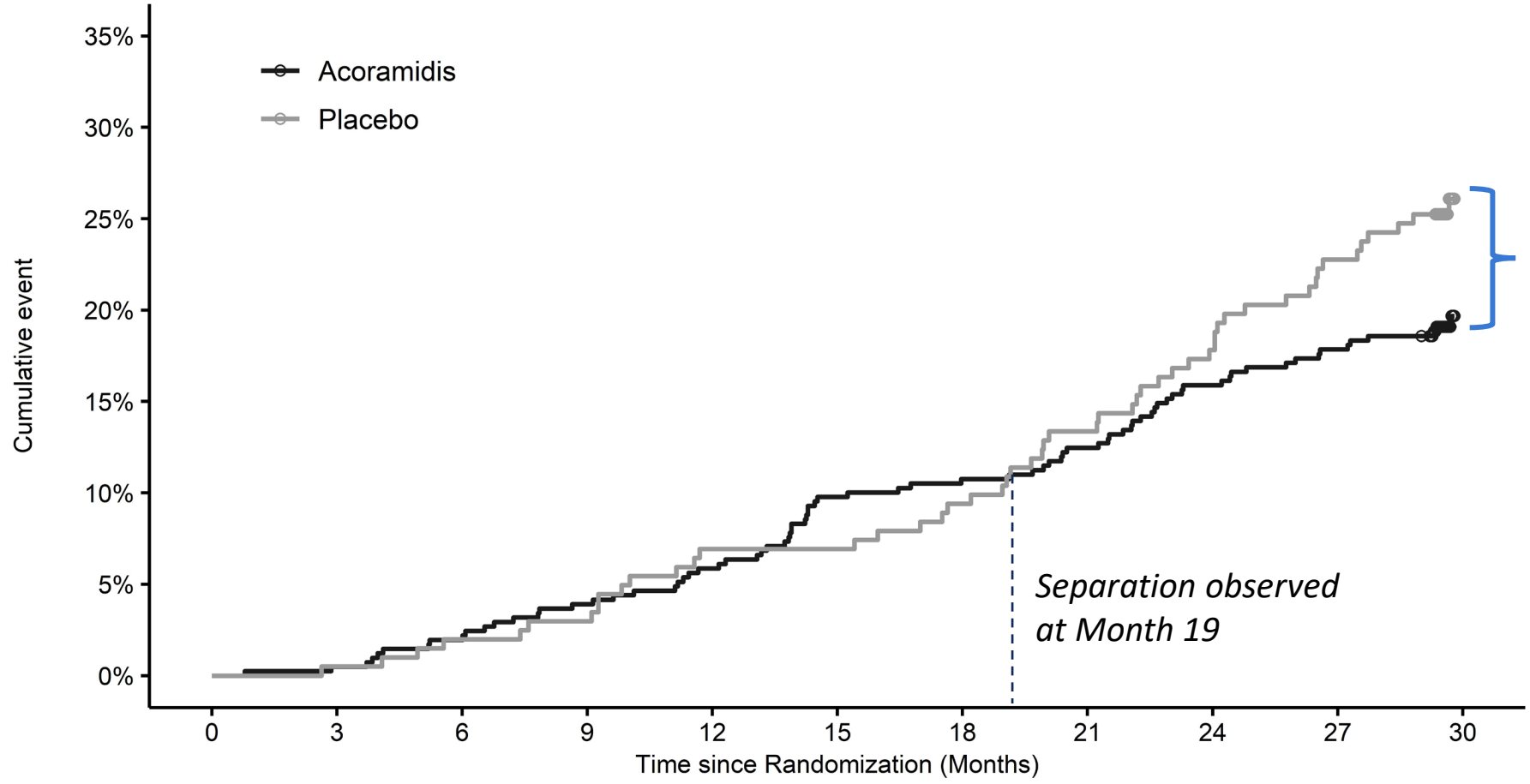
*Tafamidis usage allowed after Month 12.

ATTRibute-CM: Primary Outcome Overall and by Subgroups



FS = Finkelstein-Schoenfeld; CI = Confidence interval.

ATTRibute-CM: All-Cause Mortality

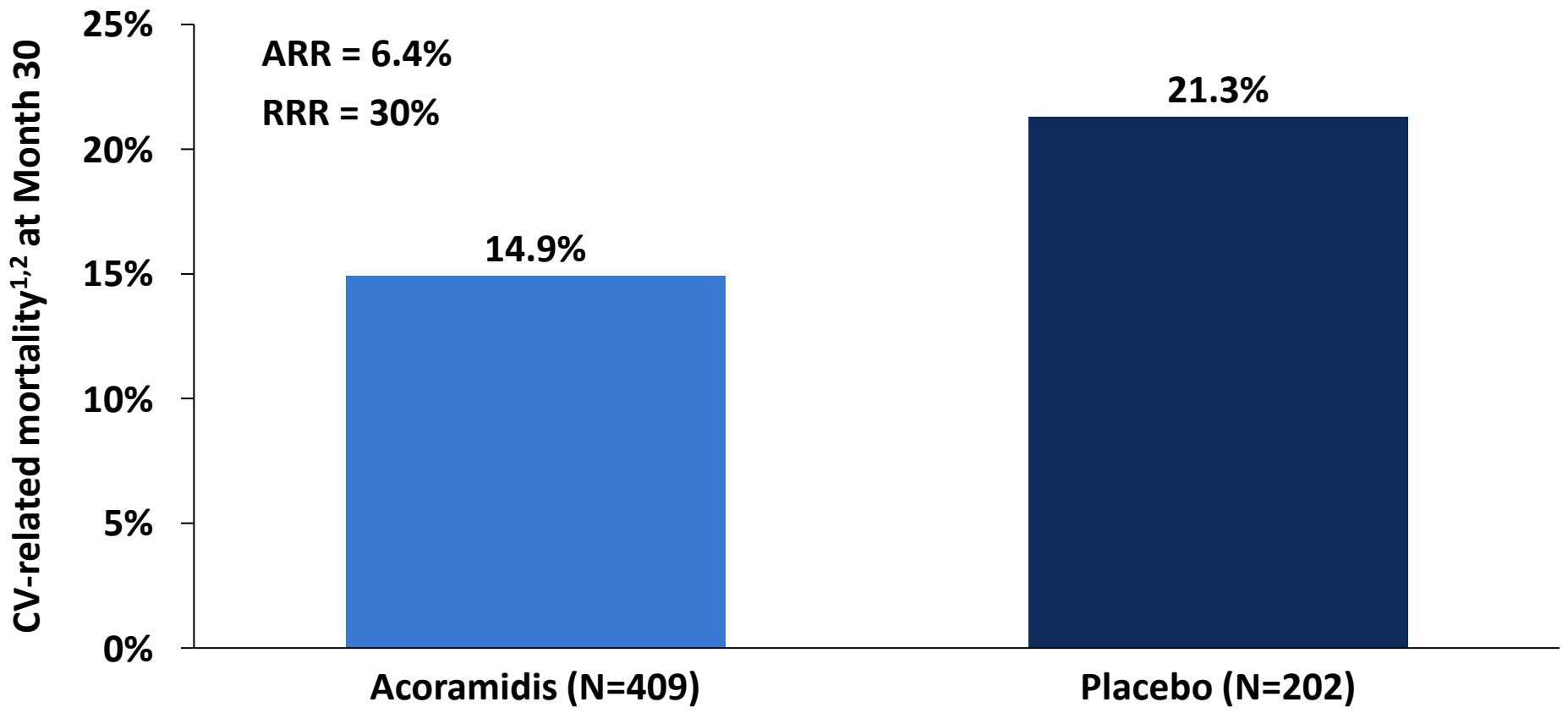


Number at risk (number of events)

Acoramidis	409 (0)	407 (2)	401 (8)	393 (16)	385 (24)	369 (40)	365 (44)	358 (51)	344 (65)	336 (73)	0 (79)
Placebo	202 (0)	201 (1)	198 (4)	196 (6)	188 (14)	188 (14)	183 (19)	175 (27)	166 (36)	156 (46)	0 (52)

ARR = Absolute risk reduction; RRR = Relative risk reduction.
All-cause mortality includes heart transplant, implantation of cardiac mechanical assist device, and all-cause death.

ATTRibute-CM: Cardiovascular-Related Mortality



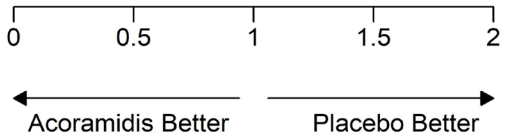
CV-related = Cardiovascular-related.

[1]Heart transplant and implantation of cardiac mechanical assistance device (CMAD) were treated as death for this analysis. N =1 heart transplant & N = 1 CMAD implantation in placebo group.

[2]CV-related mortality includes all adjudicated CV-related and undetermined cause of death.

ATTRibute-CM: Frequency of CVH; p<0.0001 on overall analysis

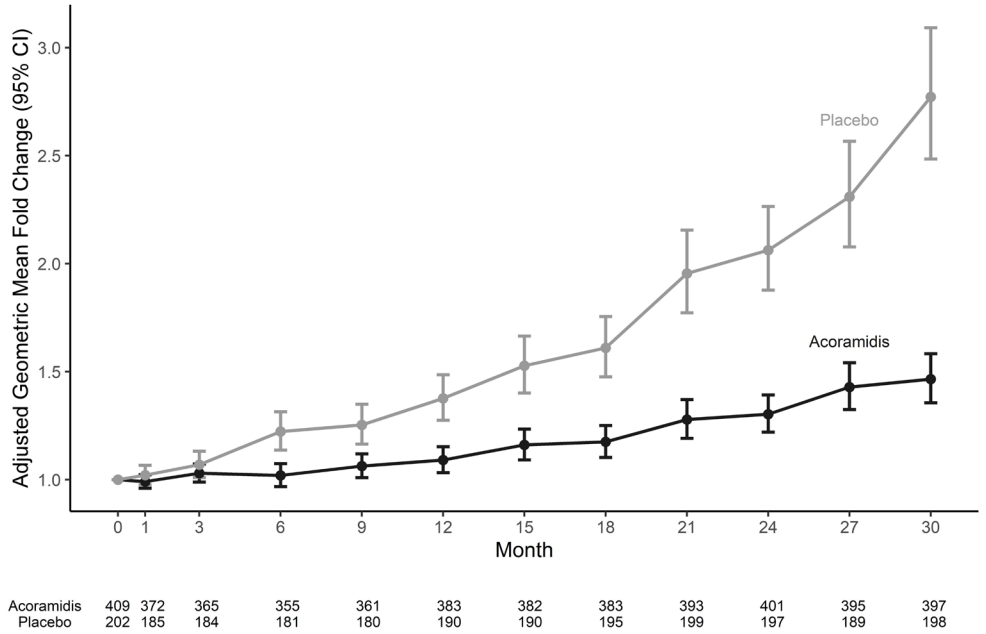
Subgroup	No. of Patients	Relative Risk [95% CI]
Overall	611(100.0)	0.496 [0.355, 0.695]
ATTR-CM Genotype		
ATTRm-CM	59(9.7)	0.377 [0.139, 1.027]
ATTRwt-CM	552(90.3)	0.514 [0.360, 0.734]
NT-proBNP (pg/mL)		
<= 3000	401(65.6)	0.456 [0.299, 0.695]
> 3000	210(34.4)	0.576 [0.330, 1.003]
eGFR (mL/min/1.73m2)		
< 45	94(15.4)	0.594 [0.250, 1.415]
>= 45	517(84.6)	0.481 [0.334, 0.692]
Age (years)		
< 78	299(48.9)	0.437 [0.275, 0.696]
>= 78	312(51.1)	0.576 [0.353, 0.940]
NYHA Class		
I, II	512(83.8)	0.447 [0.310, 0.645]
III	99(16.2)	0.721 [0.313, 1.660]



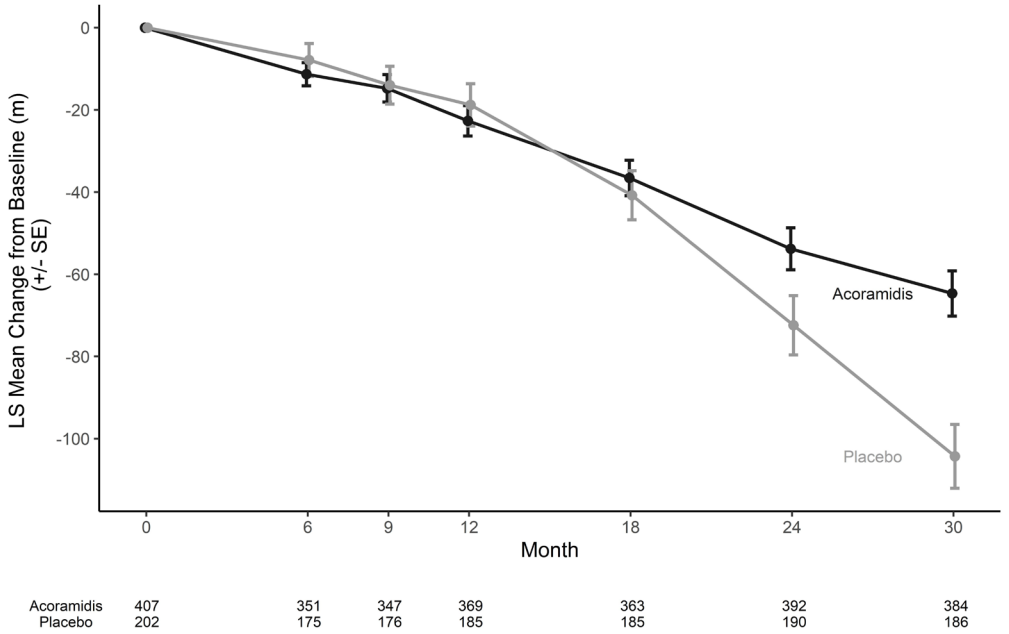
Negative binomial regression with treatment group, stratification factors, and subgroup of interest was used to analyze the cumulative frequency of adjudicated CV-related hospitalization.

ATTRibute-CM: Change from Baseline in NT-proBNP & 6MWD

Change from Baseline in NT-proBNP[1]



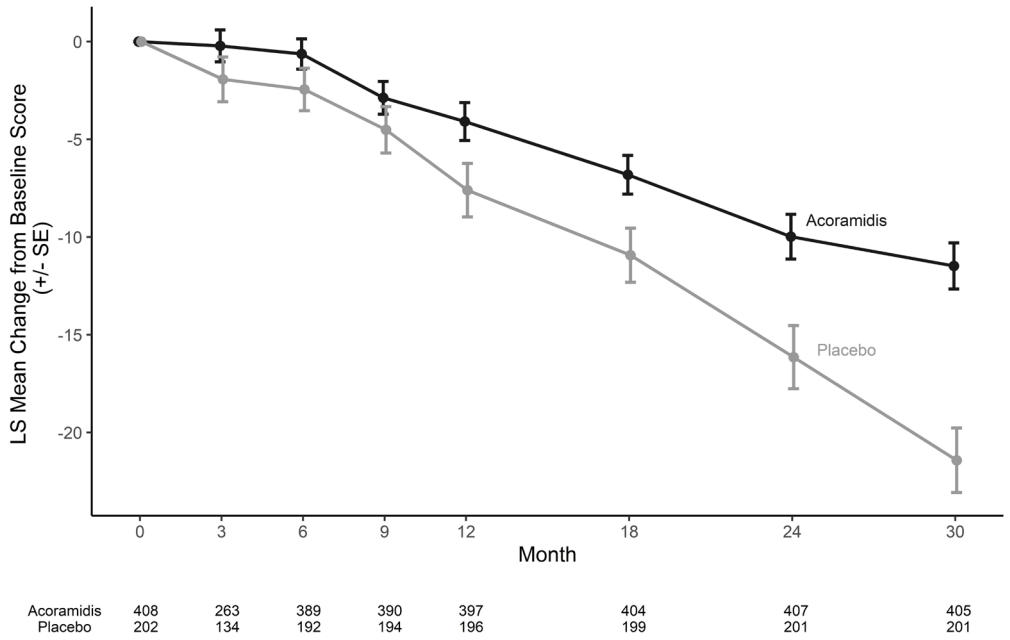
Change from Baseline in 6MWD[1]



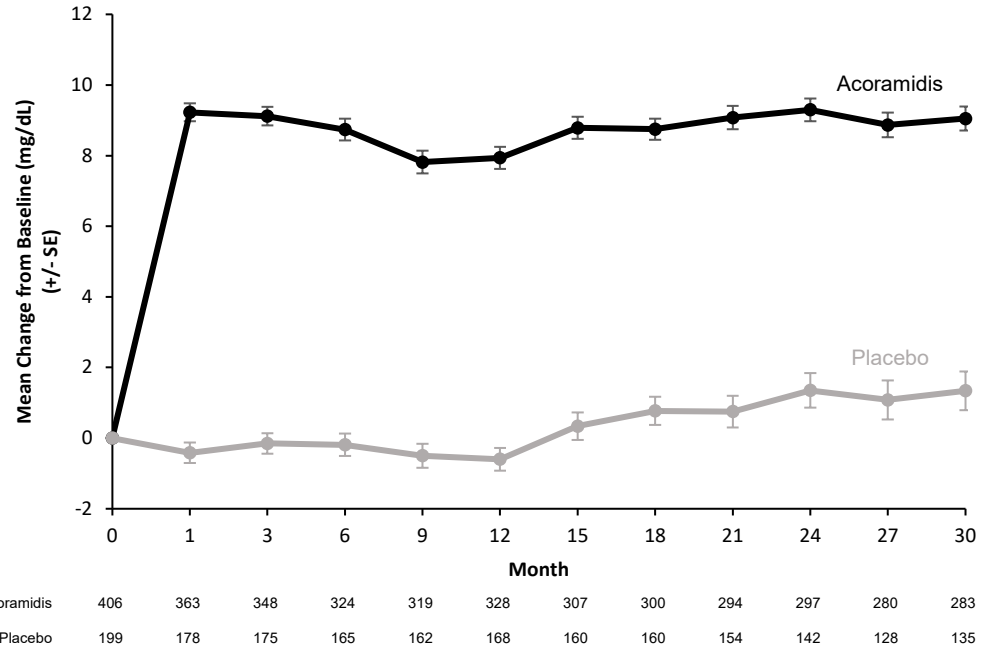
[1]Analyzed using mixed effects model with repeated measures. Missing measurements due to early discontinuation imputed using the Jump to Reference method. Missing measurements due to death performed by sampling with replacement from bottom 5% of observed values.

ATTRibute-CM: Change from Baseline in KCCQ-OS & Serum TTR

Change from Baseline in KCCQ-OS[1]



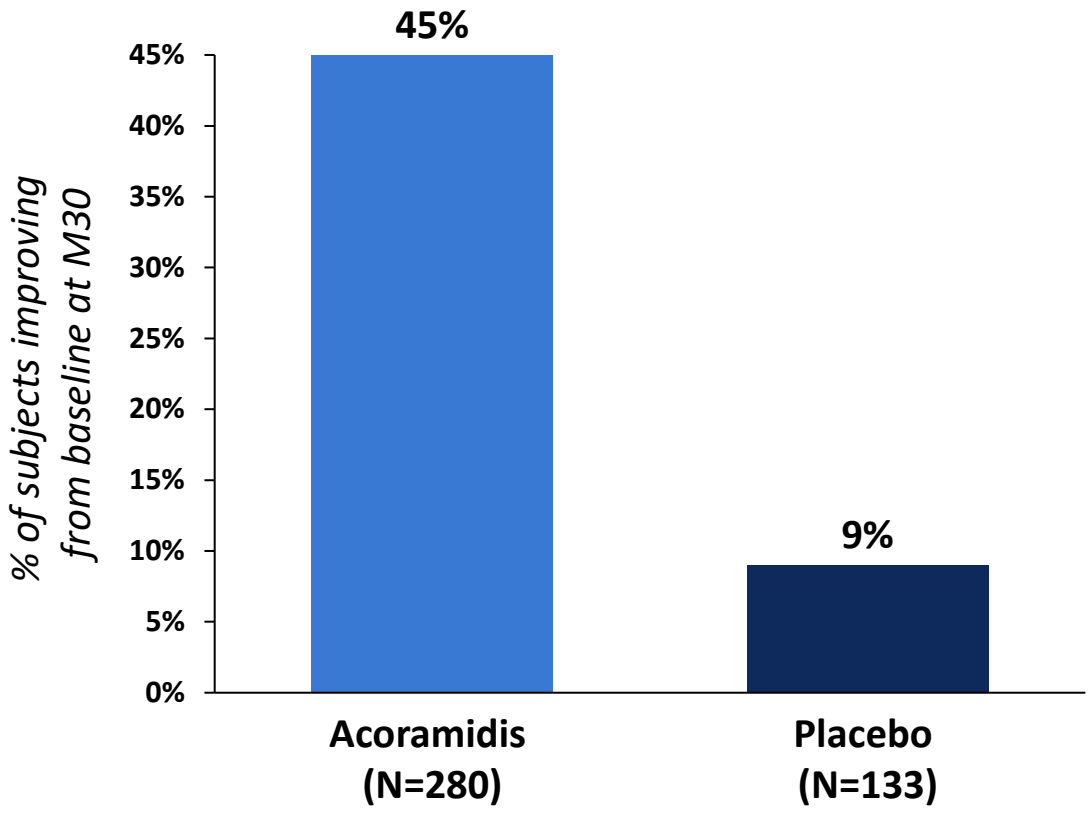
Change from Baseline in Serum TTR[2]



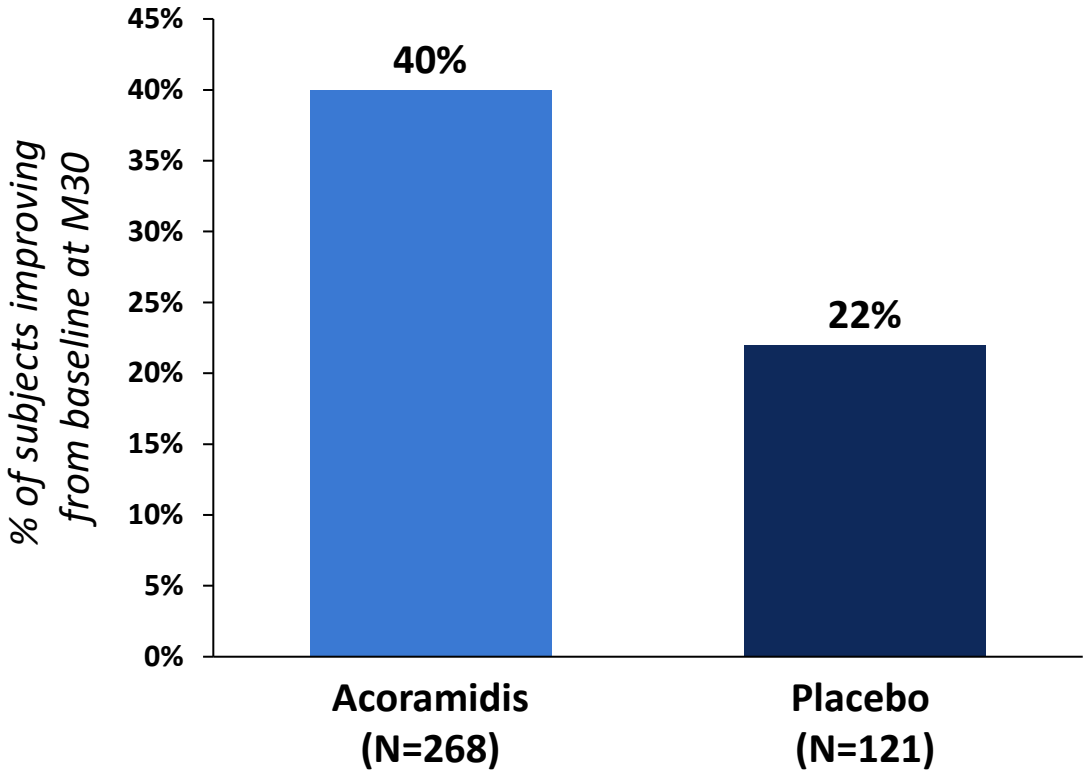
[1]Analyzed using mixed effects model with repeated measures. Missing measurements due to early discontinuation imputed using the Jump to Reference method. Missing measurements due to death performed by sampling with replacement from bottom 5% of observed values. [2]Observed measurements without any imputation. No adjustment was made for early discontinuation for any reason, including death.

ATTRibute-CM: Improvements in Disease Measures

Improvement from baseline in NT-proBNP



Improvement from baseline in 6MWD



mITT population. Improvement is defined as <0 pg/mL change from baseline to month 30 for NT-proBNP; >0 meter change from baseline to month 30 for 6MWD. In both cases, among subjects with both baseline and month 30 values.

ATTRibute-CM: Patient Safety

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%)

Acoramidis was generally well-tolerated with no findings of potential clinical concern

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.

ATTRibute-CM: Conclusions

- Primary endpoint analysis (Finkelstein-Schoenfeld hierarchy of ACM, CVH, NT-proBNP, 6MWD) highly statistically significant
  - Win ratio 1.8; $p<0.0001$; 58% of win ratio ties broken by ACM + CVH
- Consistent treatment effect across secondary endpoints
  - Better preservation of functional capacity (6MWD) and QoL (KCCQ-OS)
  - Reduced progressive increase in NT-proBNP; 45% of patients improved
- 81% survival rate on acoramidis approaches survival rate in age-matched US database (~85%)[1,2]
- 0.29 mean annual CVH frequency on acoramidis approaches annual hospitalization rate observed in broader US Medicare population (~0.26)[3]
- Reassuring safety profile

[1]ssa.gov. [2]Miller et al., Am J Card 2021; 148:146-150. [3]US Department of Health & Human Services, Jan 2018.

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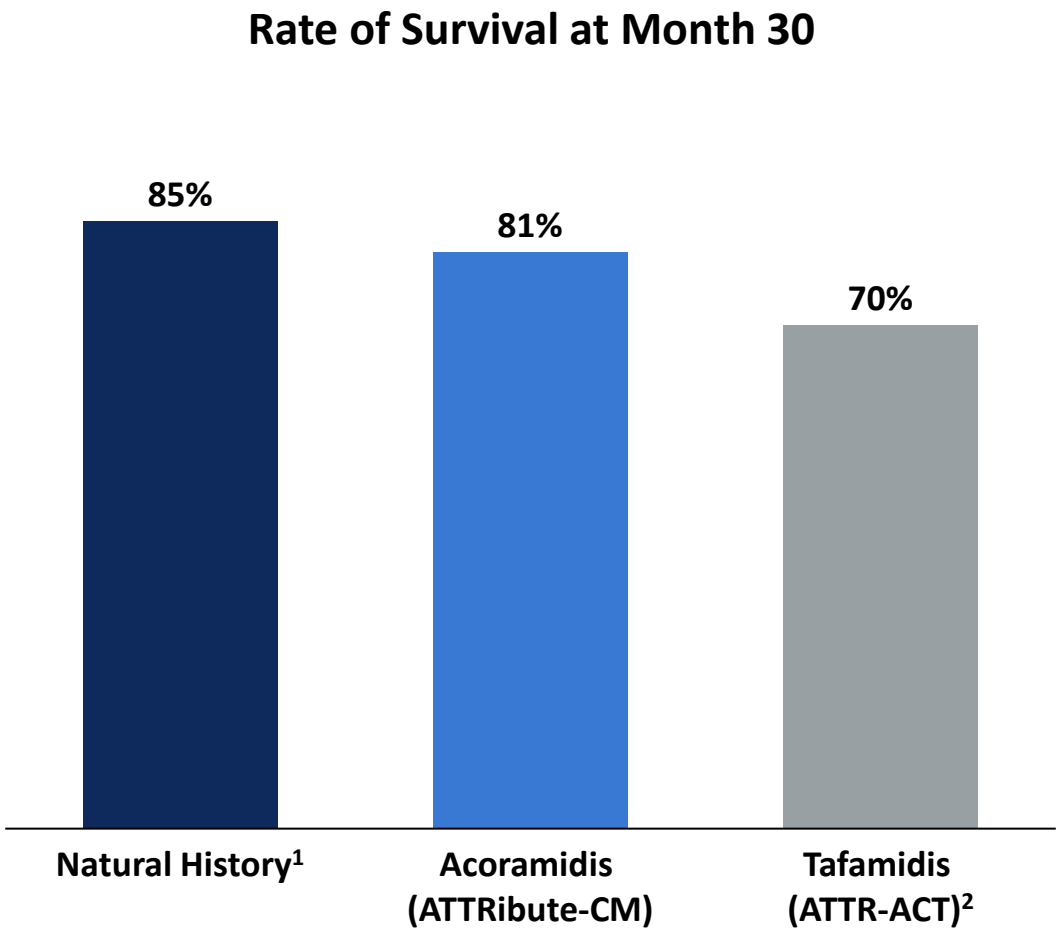
Surviving more and going to the hospital less

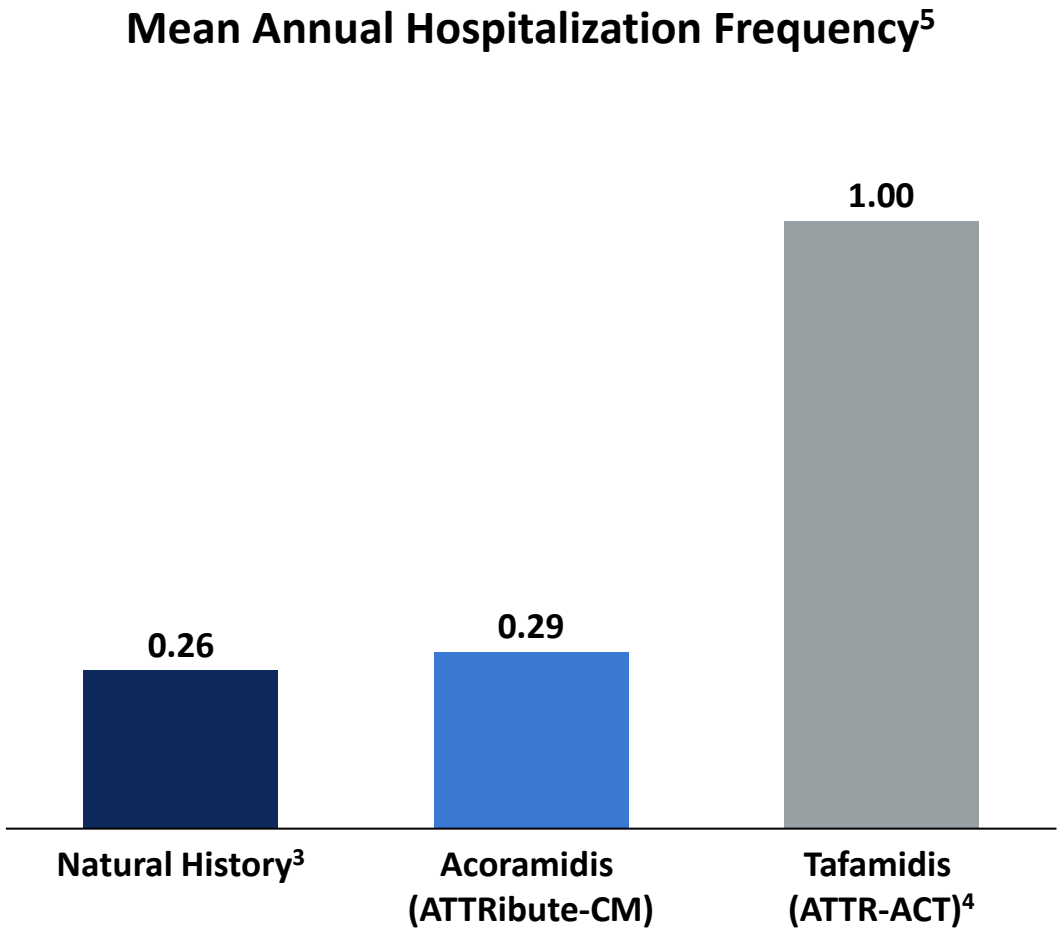
A **Dramatic risk reduction**

B **Biomarker & functional improvement**

C **Connecting the dots between extent of TTR stabilization and outcomes**

A Observed effect of acoramidis approaches rates of mortality and hospitalization in similarly aged US cohorts



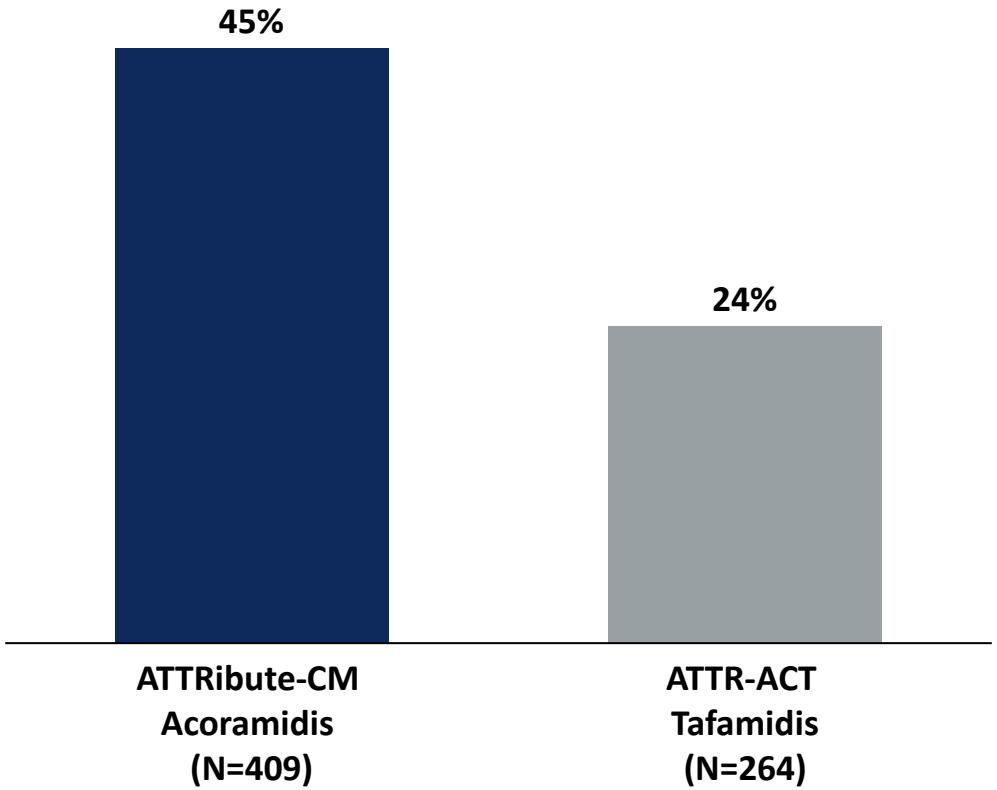


Note: Direct cross-study comparisons may suggest misleading similarities or differences. The values shown are directional and do not report robust comparative analysis.
[1]ssa.gov [2]Maurer M., et al., NEJM 2018; 379:1007-1016. [3]US Department of Health & Human Services 2018. [4]Tafamidis NDA Statistical Review, Table 6.
[5]Natural history reflects US Medicare non-neonatal, non-maternal inpatient stays. ATTRibute-CM and ATTR-ACT data reflect cardiovascular hospitalizations.

B >40% of participants experienced improvement in laboratory and functional measures of disease progression on acoramidis

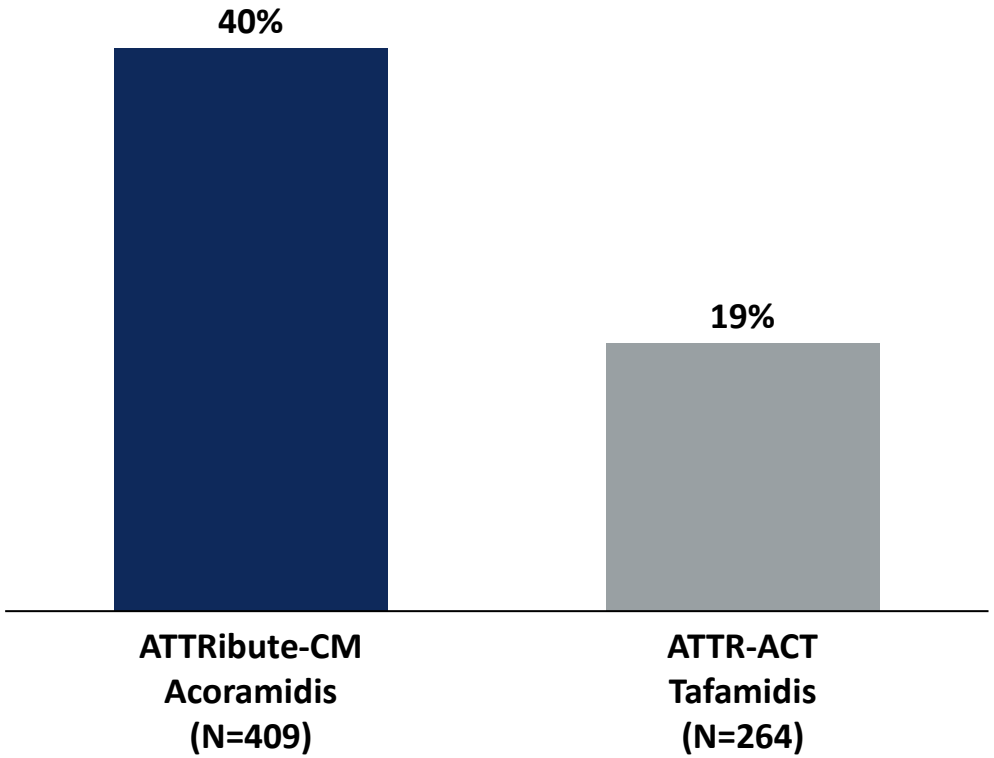
NT-proBNP (pg/mL)

% improvement from baseline at M30



6MWT (m)

% improvement from baseline at M30



Note: Direct cross-study comparisons may suggest misleading similarities or differences. The values shown are directional and do not report robust comparative analysis. N represents number of patients at baseline. ATTRibute-CM data reflects mITT population. Improvement is defined as <0 pg/mL change from baseline to month 30 for NT-proBNP; >0 meter change from baseline to month 30 for 6MWD. In both cases, among subjects with both baseline and month 30 values.

Source: Hanna M. et al, JACC: Adv 2022; 1(5):100148. Garcia-Pavia, P. et al., Eur Jour of Heart Fail., 2021, 23(6):895-905.

C Molecular hypothesis for a second generation TTR stabilizer translated to observed benefit on measures of disease progression

1

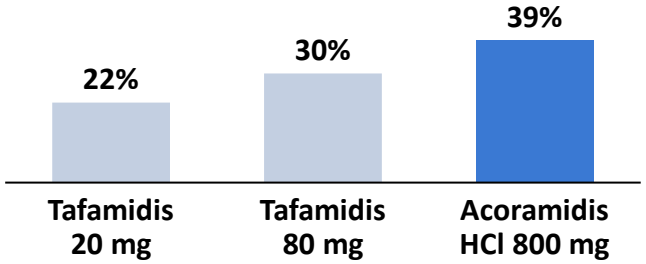
Acoramidis was designed to mimic the protective T119M mutation

Therapeutic hypothesis

2

Increases in TTR stabilization result in higher relative change in serum TTR

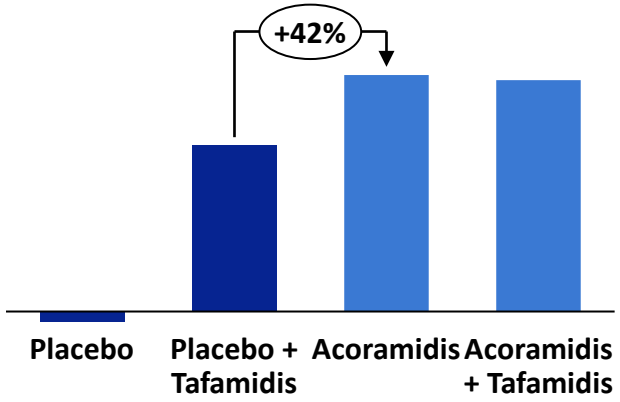
% Increase in Serum TTR at M12[1]



3

Near-complete TTR stabilization results in higher serum TTR levels on acoramidis

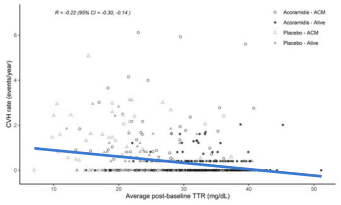
Serum TTR level at M30[2,3]



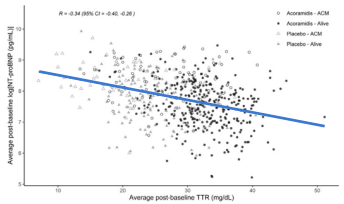
4

Higher serum TTR concentrations correlated with benefit on morbidity and quality of life with high statistical significance (correlation coefficient p<0.0001)

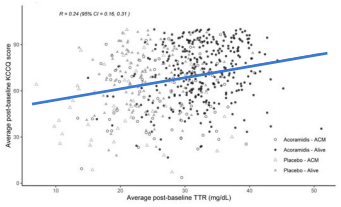
CVH Rate



NT-proBNP



KCCQ



[1]Estimated from Damy, T., et al., Eur J Heart Fail. 2021;23(2):277-285. [2]Mean change from baseline in serum TTR at Month 30 in mITT population. [3]Mean exposure on tafamidis = 11 months in mITT population.

Discussion topics

1. Molecular hypothesis
2. ATTRibute-CM Phase 3 results
3. Context for clinical findings
4. **Next steps**
5. Q&A session

First regulatory submission planned for year-end 2023

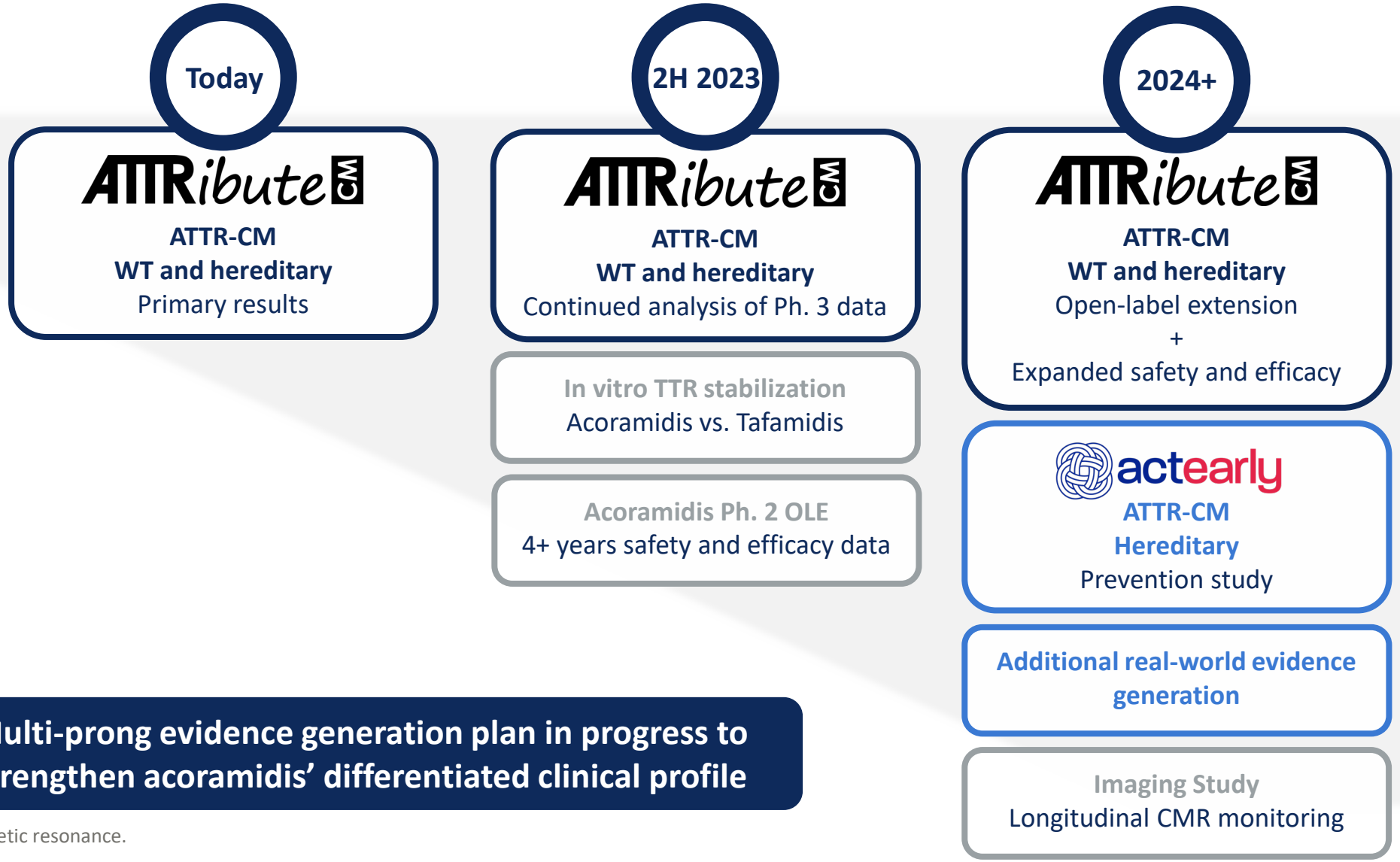
Present ATTRibute-CM Primary Results
European Society of Cardiology 2023
August 27th, 2023

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

Ongoing and planned studies of acoramidis aim to expand evidence base and addressable patient population



Multi-prong evidence generation plan in progress to strengthen acoramidis' differentiated clinical profile

CMR = Cardiac magnetic resonance.

Discussion topics

1. Molecular hypothesis on stabilization
2. ATTRibute-CM Phase 3 results
3. Context for clinical findings
4. Next steps
5. **Q&A session**