Investor Day

HCM Spotlight: Advancing a Next-Generation Cardiac Myosin Inhibitor

November 6, 2026

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Agenda

Today's program and speakers

PROGRAM

01 Introduction to Braveheart Bio

02 HCM Disease Overview

03 Obstructive HCM 52-week Extension Results

04 BHB-1893 Mechanistic Differentiation

05 nHCM Phase 2 Diastolic Data

06 Development Strategy & Future Catalysts

07 Q&A

SPEAKERS



Travis Murdoch, M.D., FRCP(C)
Chief Executive Officer, Braveheart Bio



Marc Evanchik, M.S.
Chief Scientific Officer, Braveheart Bio



Mariko Harper, M.D., MS, FACC
Medical Director, The Hypertrophic Cardiomyopathy Center, Virginia Mason Franciscan Health



Anjali Owens, M.D.
Medical Director of the Center for Inherited Cardiac Disease and an Associate Professor of Medicine in the Perelman School of Medicine at UPenn



Xueyi Wu, M.D.
Fuwai Hospital, Chinese Academy of Medical Sciences and Peking Union Medical College

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Introduction to Braveheart Bio

Travis Murdoch, M.D., FRCP(C)
President, Chief Executive Officer

Braveheart is developing a differentiated CMI for hypertrophic cardiomyopathy patients

BHB-1893 is being developed as a next generation CMI

Validated mechanism, differentiated profile

Cardiac myosin inhibition is clinically and commercially validated with 2 approved CMIs

BHB-1893 is engineered for rapid onset, predictable PK, and a shallow LVEF dose-response curve

Compelling Phase 2 oHCM & nHCM data

oHCM: 86% complete gradient response in Phase 2†; corroborated in 52-week OLE (n=42)

nHCM Phase 2 data suggested greater improvements in diastolic function, biomarkers, clinical endpoints at early time point (12 weeks)

Wide therapeutic index

Low rate of LVEF events, with early data suggesting differentiated efficacy

Consistent PK, with balanced hepatic and renal clearance; no major DDIs identified

Ease of use

89% of BID patients managed on 40 or 60 mg in Phase 2 oHCM study

Supports potential for a simplified, low-monitoring dose regimen

Two large, underserved indications

oHCM and nHCM represent ~700K–960K U.S. patients with significant unmet need

No approved therapy exists for nHCM

Strong financial position

Well-capitalized following IPO to execute global Phase 3 studies in oHCM and nHCM

Experienced leadership team with history of public-company entry and strategic exits

OLE: open-label extension; PK: pharmacokinetic; DDI: drug-drug interaction.
† Group 2 (40-60-80 mg BID) baseline to Week 12 (N=14).

Advancing into global Phase 3 development in both oHCM and nHCM

oHCM studies:

- ***LIONHEART-HCM*** *– enrolling Phase 3, head-to-head vs metoprolol with a single titration step (40→60 mg BID).*
- ***Hengrui Phase 3****– fully enrolled, HRS-1893 on top of SoC in China with topline expected in 1H 2027*
- ***TRUEHEART-HCM*** *– fixed 40 mg BID on top of SoC; further evidence supporting fixed-dose*

nHCM Phase 3: ***initiating 1H 2027 (end-of-Phase 2 meeting complete)***

Program	Phase 1	Phase 2	Phase 3	Status
Obstructive HCM	LIONHEART-HCM (NCT07755904) vs. metoprolol			**Actively enrolling Interim analysis, 2H 2027**
	Ongoing (NCT07021976)			**Enrollment complete Topline results, 1H 2027**
	TRUEHEART-HCM fixed dose			**Trial initiation, 1H 2027**
Non-obstructive HCM	NOBLEHEART-HCM vs. placebo			**Trial initiation, 1H 2027**
HFpEF	Ongoing (NCT07269717)			**Actively enrolling**

Participants from global studies will be candidates for long term extension study

HEARTSONG-LTE

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HFpEF: heart failure with preserved ejection fraction; LTE: long-term extension; SoC: standard of care

Today's Focus

Advancing BHB-1893 as a potential preferred treatment option in HCM



Rapid onset, predictable PK/PD profile with potential minimal LVEF cost

Potential to move beyond symptoms to disease modification

Reproduced robust Ph2 12w data in OLE with deepening clinical activity on exercise capacity and symptoms observed at 52w

Meaningful diastolic improvements in nHCM Ph2 at 12 weeks

Targeting the simplest and most effective dosing paradigm in HCM

Minimal or no titration may enable broader prescriber base

HCM Disease Overview

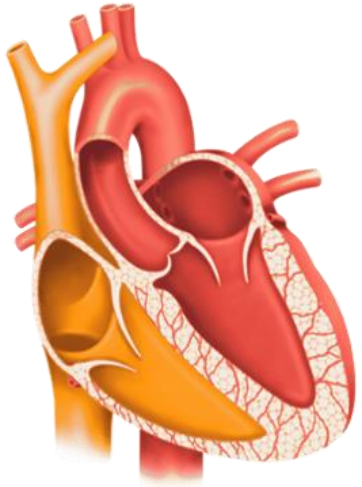
Mariko Harper, M.D.,
Medical Director, HCM Program, VMFH

HCM is the most common inherited cardiac disease with significant unmet need

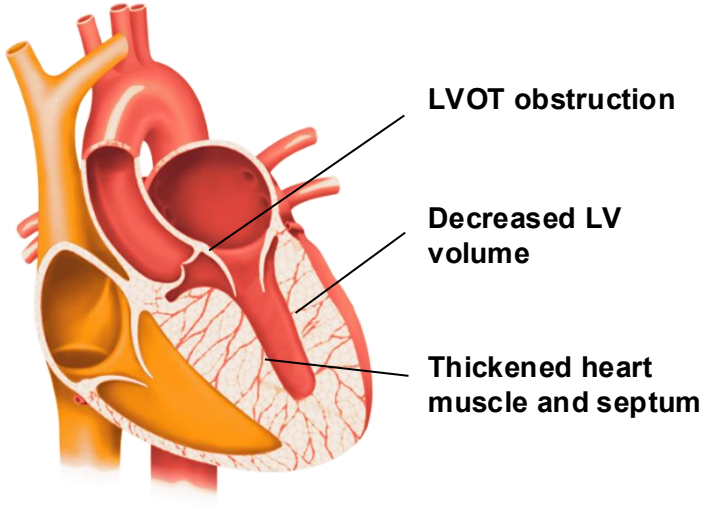
HCM is defined by **asymmetrical left ventricular hypertrophy**, with end-diastolic wall thickness ≥15 mm (or ≥13 mm in adults with a family history or positive genetic test), in the absence of another identifiable cause of LV hypertrophy.[1]

HCM is classified into **obstructive and non-obstructive HCM** based on the presence or absence of LVOT obstruction.[1]

Healthy Heart

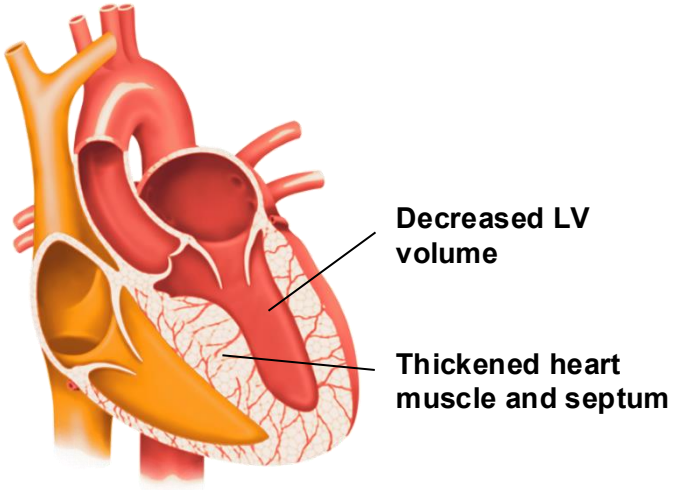


oHCM Heart



Dynamic LVOT obstruction (resting or provocable LVOT-G ≥30 mmHg), often **driven by systolic anterior motion of the mitral valve and septal hypertrophy**[1]

nHCM Heart



Highly heterogenous with the **absence of LVOT obstruction** yet **diastolic dysfunction** and **elevated cardiac biomarkers** persist[1,2]

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1. Ommen SR, et al. Circulation. 2024. 2. Maron MS, et al. J Am Coll Cardiol. 2016.
HCM: hypertrophic cardiomyopathy; LV: left ventricle; LVOT: left ventricular outflow tract; LVOT-G: LVOT gradient; nHCM: nonobstructive HCM; oHCM: obstructive HCM.

HCM is a serious and lifelong progressive cardiovascular disease

Underlying Disease Burden

Diastolic dysfunction

Impaired myocardial relaxation → ↑ LV stiffness, ↑ LV filling pressure, ↑ LA

Ongoing cardiac damage

Chronic myocardial stress → cardiac damage → elevated troponin

Myocardial injury & fibrosis

Long-term myocardial injury → progressive fibrosis/scarring

Common Symptoms



Shortness of breath



Chest pain



Exertional fatigue



Syncope

Resulting Risks



Atrial fibrillation



Heart failure



Stroke



Sudden cardiac death

Impact on US population

~700K–960K
US patients with HCM

~1 in 345
General-population prevalence

~38%
Annual hospitalization rate* (all-cause)

Lifelong
Chronic; ongoing therapy

Source: Bick AG, et al. Am J Hum Genet. 2012; Butzner M, et al. JACC Adv. 2026; Marian AJ. Circ Res. 2021; Maron BJ, et al. Circulation. 1995; Maron BJ, et al. J Am Coll Cardiol. 2022; Maron MS, et al. J Am Coll Cardiol. 2016; Ommen SR, et al. Circulation. 2024; Semsarian C, et al. J Am Coll Cardiol. 2015; Sequeira V, et al. FEBS Lett. 2019; Spudich JA. Pflugers Arch. 2019; Zaiser E, et al. J Patient Rep Outcomes. 2020; Company filings.

**symptomatic oHCM patients. ATP: adenosine triphosphate; LVOT: left ventricular outflow tract; LVOT-G: LVOT gradient; nHCM: nonobstructive HCM; oHCM: obstructive HCM.*

Cardiac myosin inhibitors target the root cause of disease in oHCM

First-line therapies

Target symptoms

Beta blockers, CCBs, SCBs – limited HCM trial evidence[1-3]

Septal reduction

Surgical intervention

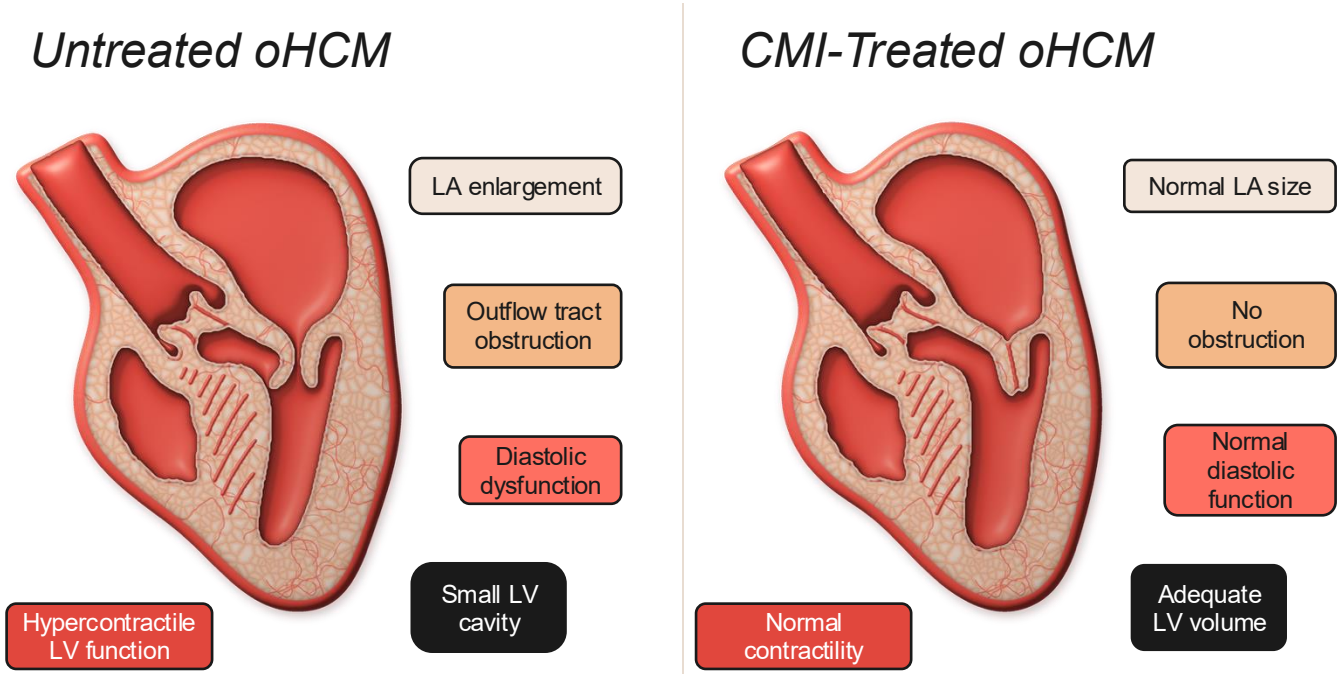
Recommended for drug-refractory population in oHCM – requires highly experienced centers[1,3,4]

CMIs

Disease targeting

Guideline directed use to address underlying pathophysiology of oHCM[5,6]

CMI Mechanism of Action[7]



CMIs have improved management of symptomatic oHCM, with **demonstrated improvements in exercise capacity and patient symptoms**.[5,6]

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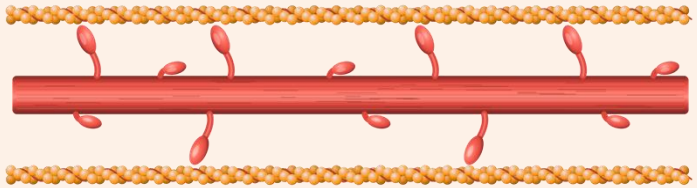
Source: 1. Ommen SR, et al. Circulation. 2024. 2. Palandri C, et al. Drugs. 2022. 3. Arbelo E, et al. Eur Heart J. 2023. 4. Batzner A, et al. Dtsch Arztebl Int. 2019. 5. Olivotto I, et al. Lancet. 2020. 6. Maron MS, et al. N Engl J Med. 2024. 7. Ostrominski JW, et al. JACC Heart Fail. 2023.
CCB: calcium channel blockers; E/e': mitral inflow velocity/average (lateral and septal) mitral annular velocity; LA: left atrial; LV: left ventricle; oHCM: obstructive HCM; SAM: systolic anterior motion.

CMIs directly target HCM pathophysiology by reducing the number of force generating cross-bridges

Effect of Cardiac Myosin Inhibitors on Hypertrophic Cardiomyopathy

Normal Sarcomere

Normal number of myosin-actin cross-bridges

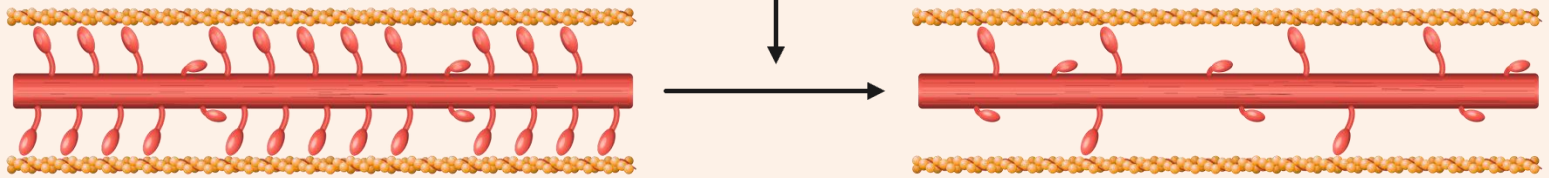


Hypertrophic Cardiomyopathy Sarcomere

Increased number of cross-bridges

Cardiac Myosin Inhibition

Decreased number of cross-bridges



HCM is a disease of the sarcomere

Resulting in abnormal acti-myosin cross-bridging[1-3]

HCM sarcomeres have excess cross-bridges

Which result in increased contractility[1-6,12]

CMIs decrease cross-bridges and reduce hypercontractility

As a result, normalizing the myosin availability to bind actin[1,2,6-8,10,12]

Figure adapted from Braunwald E. N Engl J Med. 2025. 1. Marian AJ. Circ Res. 2021. 2. Spudich JA. Pflugers Arch. 2019. 3. Sequeira V, et al. FEBS Lett. 2019. 4. Ommen SR, et al. Circulation. 2024. 5. Palandri C, et al. Drugs. 2022. 6. Arbelo E, et al. Eur Heart J. 2023. 7. Batzner A, et al. Dtsch Arztebl Int. 2019. 8. Olivotto I, et al. Lancet. 2020. 9. Maron MS, et al. N Engl J Med. 2024. 10. Ostrominski JW, et al. JACC Heart Fail. 2023. 11. Braunwald E. N Engl J Med. 2025. 12. Lim J, Kim HK. J Cardiovasc Imaging. 2025.

CMIs have improved oHCM care, but significant unmet needs remain with potential for greater efficacy and convenience

First generation CMIs



Proof of mechanism

CMI modulation of cross-bridges



Reduction of gradient

LVOT-G response in ~50-60%



Improved feel & function

↑ pVO_2 · KCCQ · NYHA



Established market

Validated mechanism in HCM

Opportunity for next-generation CMI



Non-responders

Large percentage of patients not well served



"LVEF cost"

Caps gradient benefit



Long titration/monitoring

Months to reach effective dose



Long half-life

Slow onset & reversal

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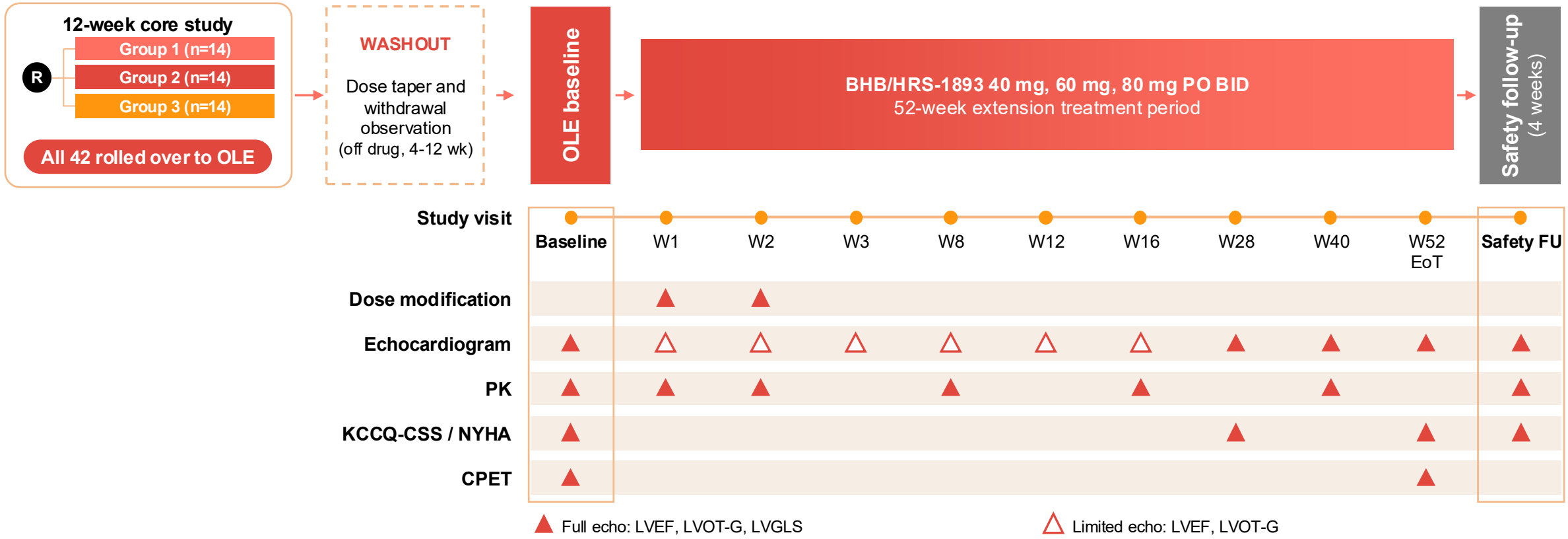
Source: Olivotto et al., Lancet 2020. Tian et al., JAMA Cardiol. 2023. Maron MS et al., N Engl J Med 2024. Garcia-Pavia et al., N Engl J Med 2025.

Obstructive HCM 52-week Extension Results

Travis Murdoch, M.D., FRCP(C)
President, Chief Executive Officer

Every Phase 2 oHCM patient continued onto the 52-week OLE, where responses deepened with longer treatment

Adult patients with symptomatic oHCM who had resting LVOT-G ≥50 mmHg (or resting LVOT-G ≥30 mmHg and Valsalva LVOT-G ≥50 mmHg), LVEF ≥60%, and NYHA Class II-III were enrolled



Database lock: June 2026

BID, twice daily; CPET, cardiopulmonary exercise test; EoT, end of treatment; FU, follow-up; KCCQ-CSS, Kansas City Cardiomyopathy Questionnaire Clinical Summary Score; LV-FS, left ventricular fractional shortening; LV-GLS, left ventricular global longitudinal strain; LVEF, left ventricular ejection fraction; LVOT-G, left ventricular outflow tract gradient; LVOT-VTI, LVOT velocity time integral; NYHA, New York Heart Association; OLE, open-label extension; PK, pharmacokinetics; PO, oral administration; R, randomization; W, week.

After washout from the core treatment period, Valsalva LVOT-G and pVO_2 returned to pre-treatment levels

	40-60-80 BID (N=14)	Core treatment baseline (N=42)	OLE Baseline (N=42)
Age, years, mean (SD)	51.5 (10.4)	51.5 (11.4)	-
Male, n (%)	9 (64.3)	26 (61.9)	-
Background therapy, n (%)			
Beta blockers	12 (85.7)	38 (90.5)	-
Calcium channel blockers	1 (7.1)	2 (4.8)	-
History of Atrial Fibrillation	2 (14.3)	2 (4.8)	-
Valsalva LVOT-G, mmHg, mean (SD)	107.2 (41.7)	100.4 (34.9)	115.1 (44.3)
Resting LVOT-G, mmHg, mean (SD)	80.3 (33.4)	72.5 (25.5)	93.3 (41.5)
LVEF, %	69.3 (3.5)	69.9 (3.3)	71.5 (4.1)
pVO_2, mL/kg/min, mean (SD)	17.5 (3.2)	18.4 (3.5)	17.1 (4.1)
NYHA class, n (%)			
Class I	-	-	2 (4.8)
Class II	14 (100.0)	40 (95.2)	38 (90.5)
Class III	0 (0.0)	2 (4.8)	2 (4.8)
KCCQ-CSS, mean (SD)	86.0 (12.9)	81.7 (13.45)	77.2 (15.1)

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Data are shown as mean (standard deviation) unless otherwise specified. BID, twice daily; KCCQ-CSS, Kansas City Cardiomyopathy Questionnaire Clinical Summary Score; LVEF, left ventricular ejection fraction; LVOT-G, left ventricular outflow tract gradient; NYHA, New York Heart Association; OLE, open-label extension; pVO2, peak oxygen uptake; SD, standard deviation.

52-week Phase 2 OLE results highlight BHB-1893's promising clinical profile



DEPTH

Rapid responses that deepen with sustained treatment

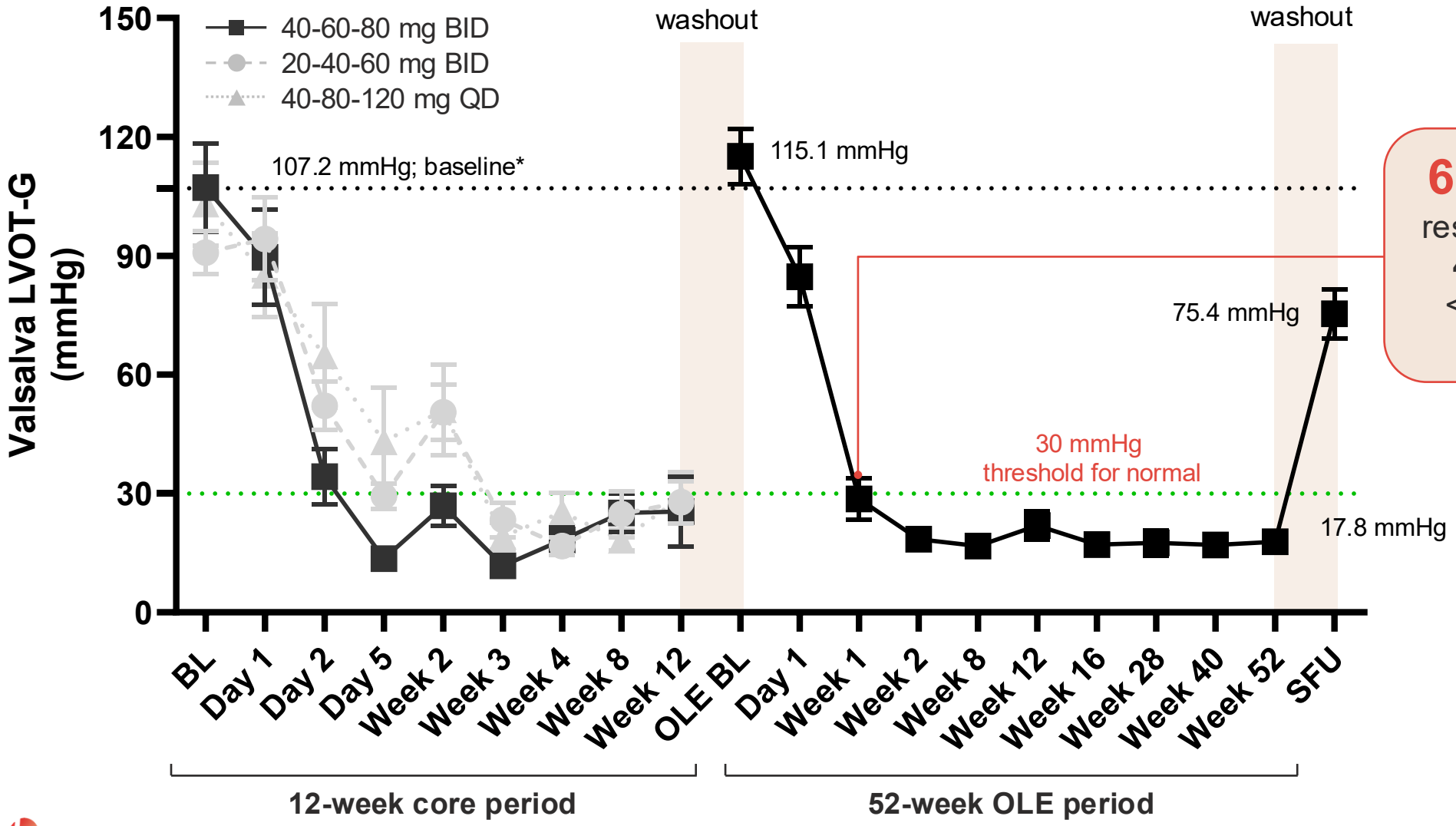
TARGETING

Consistent with altering the pathophysiology of disease

DOSING

Effective starting dose for the majority of patients

Rapid and deep reduction maintained through Week 52

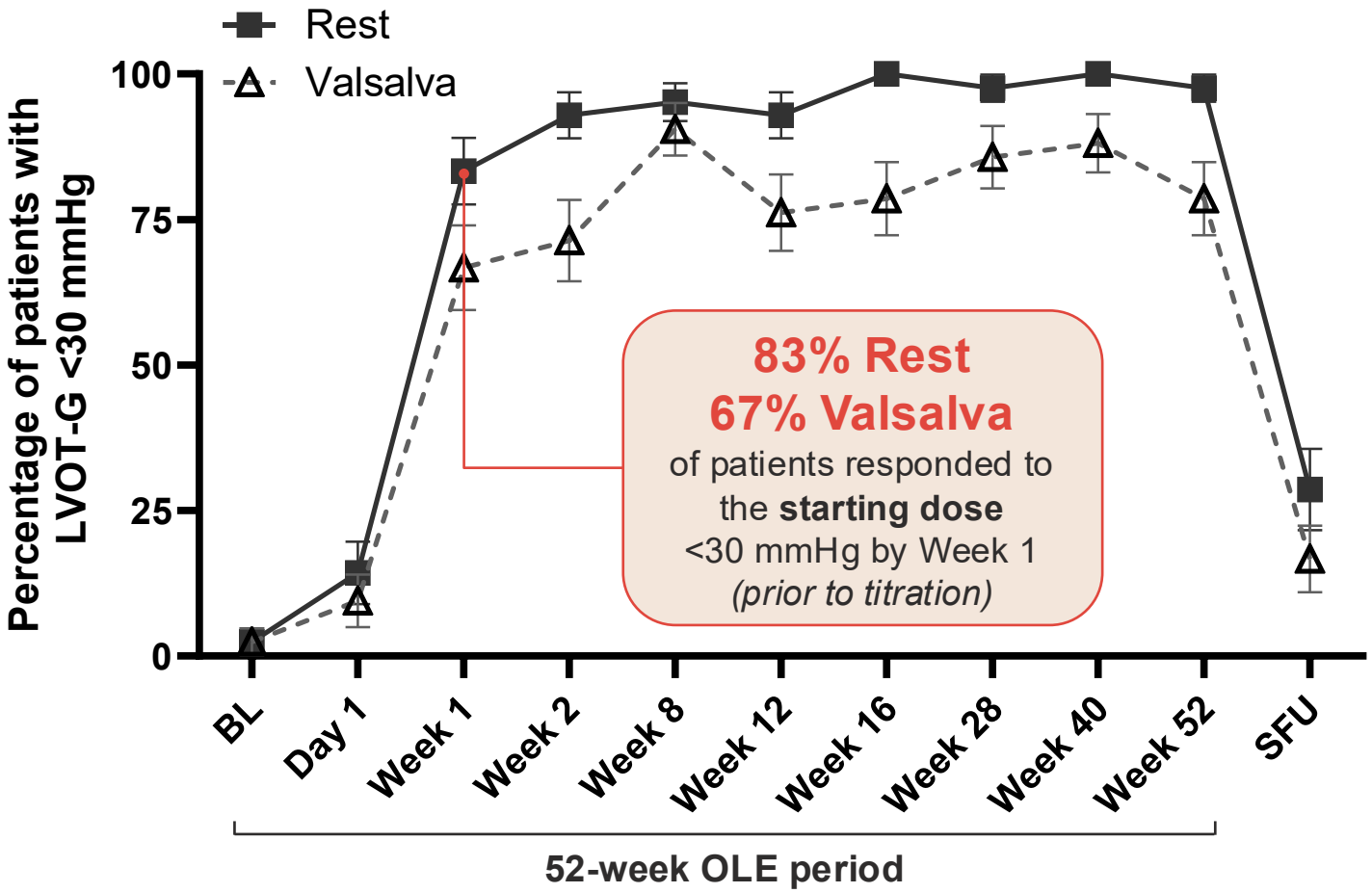


67% of patients
responded to the planned
40 mg starting dose
<30 mmHg by Week 1
(prior to titration)

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**Group 2. BID, twice daily; BL, baseline; LVOT-G, left ventricular outflow tract gradient; OLE, open-label extension; QD, once daily; SFU, safety follow-up.*

90% of patients achieved a Valsalva LVOT-G <30 mmHg during 52 weeks of treatment



- Rapid normalization of gradient observed at both rest and Valsalva
- Responders seen as early as Day 1

Database lock: June 2026
BL, baseline; LVOT-G, left ventricular outflow tract gradient; OLE, open-label extension; SFU, safety follow-up.

Rapid and deep reduction observed, with partial return to baseline after 52 weeks of treatment

Week 12 (Core)

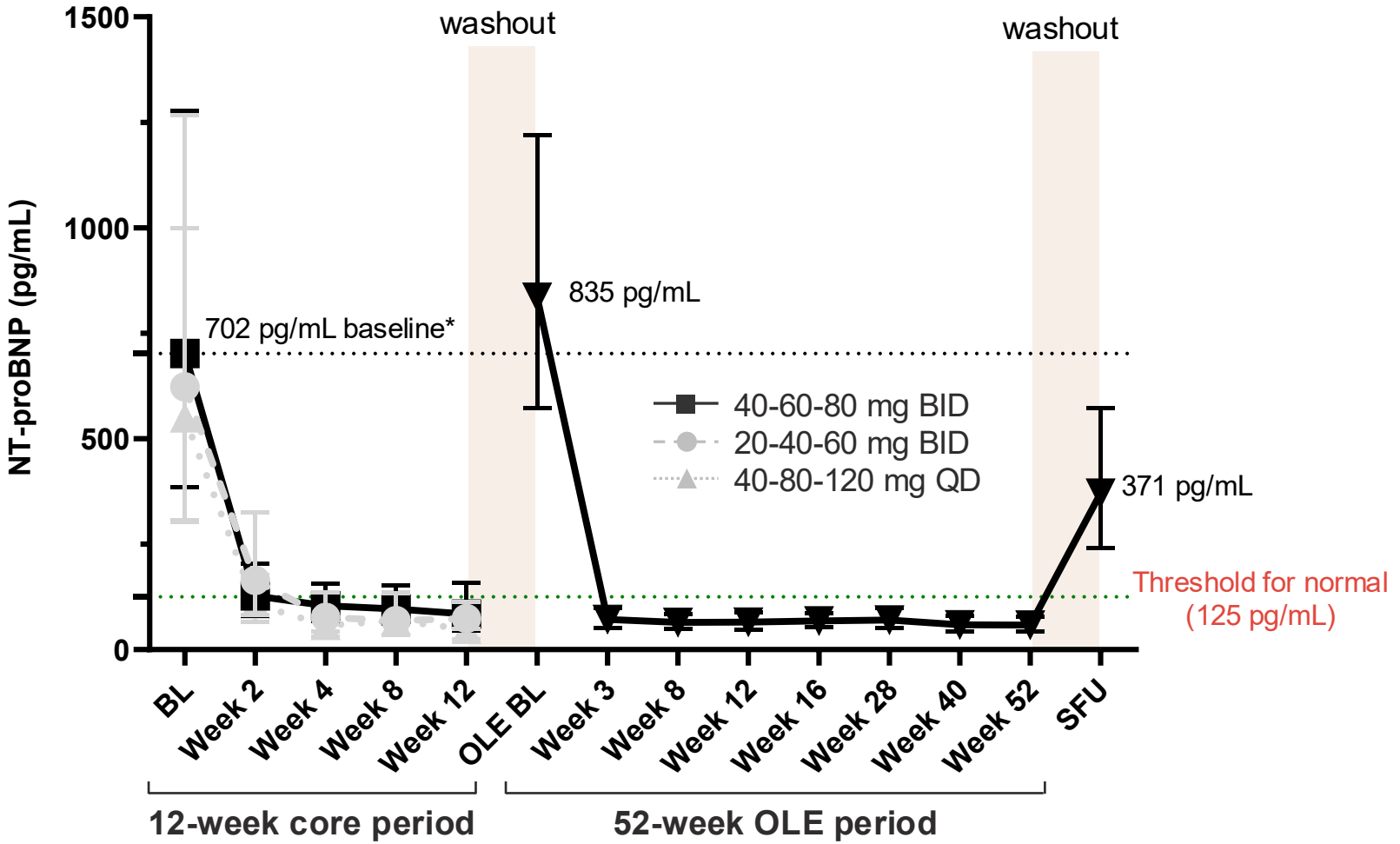
78.8% reduction

76.2% of patients normalized (<125 pg/mL)

Week 52 (OLE)

87.5% reduction

78.6% of patients normalized (<125 pg/mL)

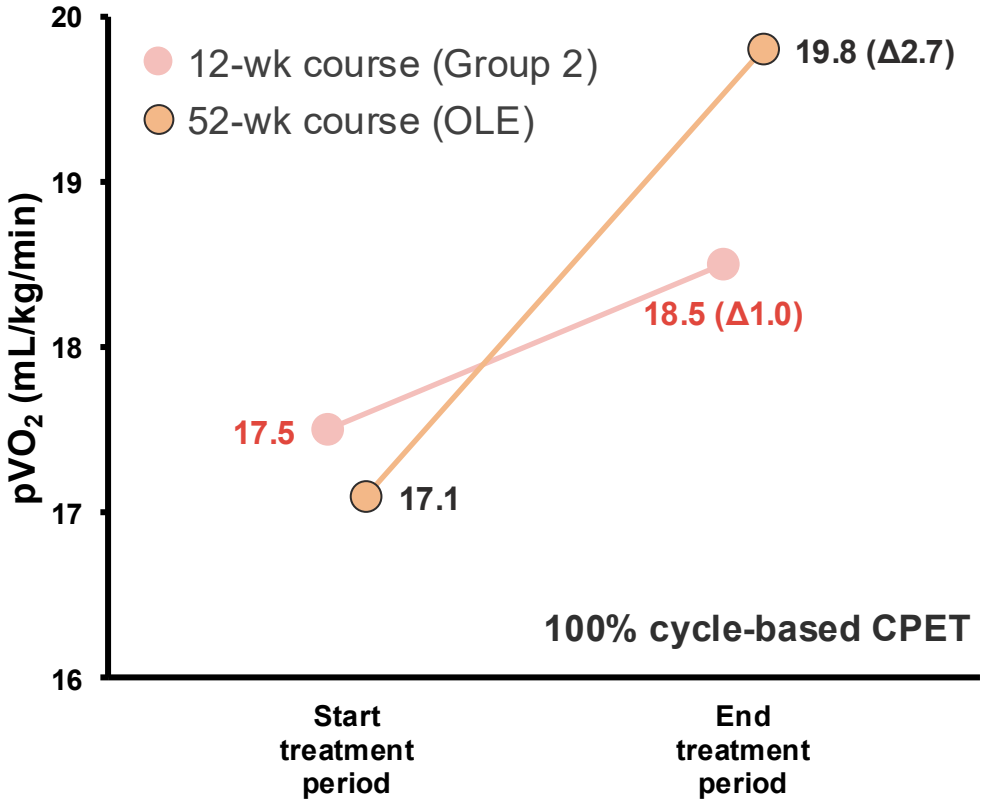


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**Group 2. BID, twice daily; BL, baseline; NT-proBNP, N-terminal pro-B-type natriuretic peptide; OLE, open-label extension; QD, once daily; SFU, safety follow-up.*

CPET improved further by Week 52 in both pVO_2 and VE/VCO2



70% of patients
improved pVO_2 by ≥1.5 mL/kg/min

50% of patients
improved pVO_2 by ≥3.0 mL/kg/min

−4.1
VE/VCO2 improvement at Week 52

Early pVO_2 improvements at Week 12 deepened by Week 52

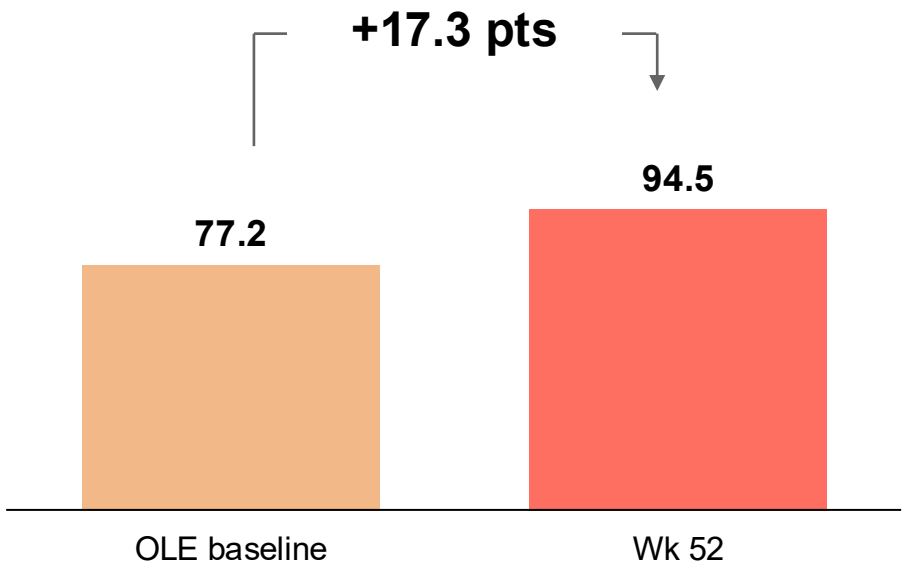
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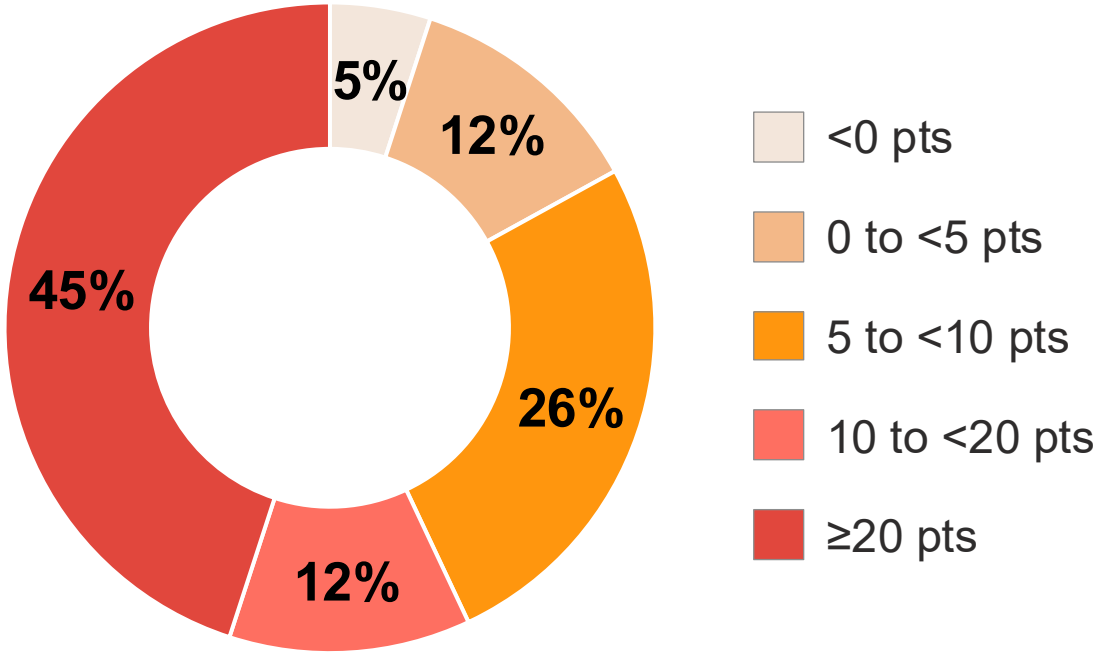
Database lock: June 2026
12 wk = Group 2 (40-60-80 mg BID) baseline to Week 12 (N=14); 52 wk = 40-60-80 mg BID OLE baseline to Week 52 (N=40 with OLE BL CPET available). BID, twice daily; CPET, cardiopulmonary exercise test; OLE, open-label extension; pVO2, peak oxygen uptake; VE/VCO2, minute ventilation to carbon dioxide output ratio; Wk, week.

Significant improvement in KCCQ with large proportion of patients ≥20-point improvement at week 52

+17.3
Point improvement in KCCQ-CSS

45%
of patients with ≥20 pt improvement at Week 52



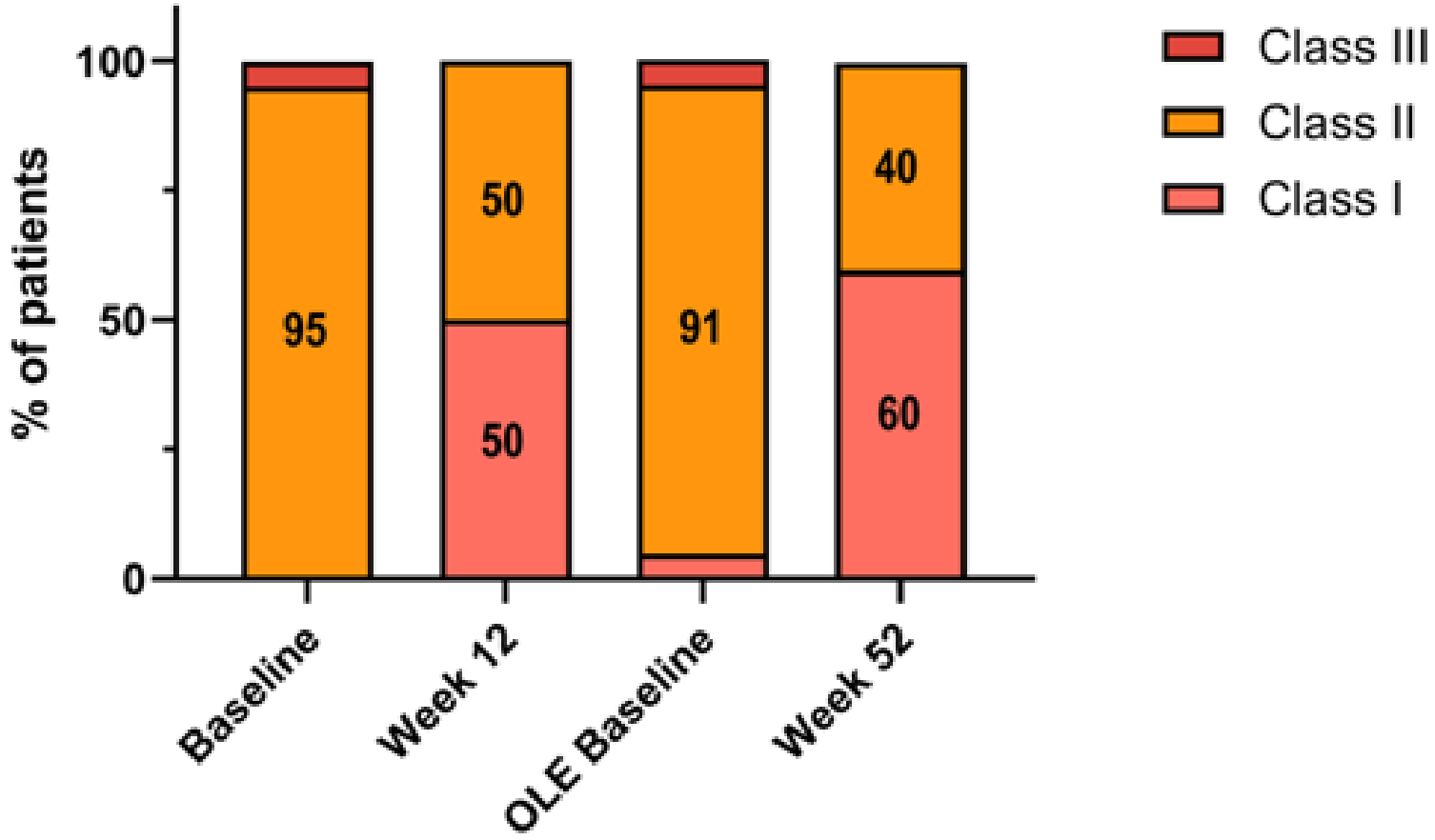


Database lock: June 2026
N=42, KCCQ-CSS values are means. KCCQ, Kansas City Cardiomyopathy Questionnaire; KCCQ-CSS, KCCQ Clinical Summary Score; OLE, open-label extension; Wk, week.

60% of patients reached NYHA Class I by Week 52

NYHA improvement
seen at Week 12
continued at Week 52

55%
improved by ≥1 NYHA class at 52 weeks



Database lock: June 2026
NYHA, New York Heart Association; OLE, open-label extension.

52-week Phase 2 OLE results highlight BHB-1893's promising clinical profile

DEPTH

Rapid responses that deepen with sustained treatment

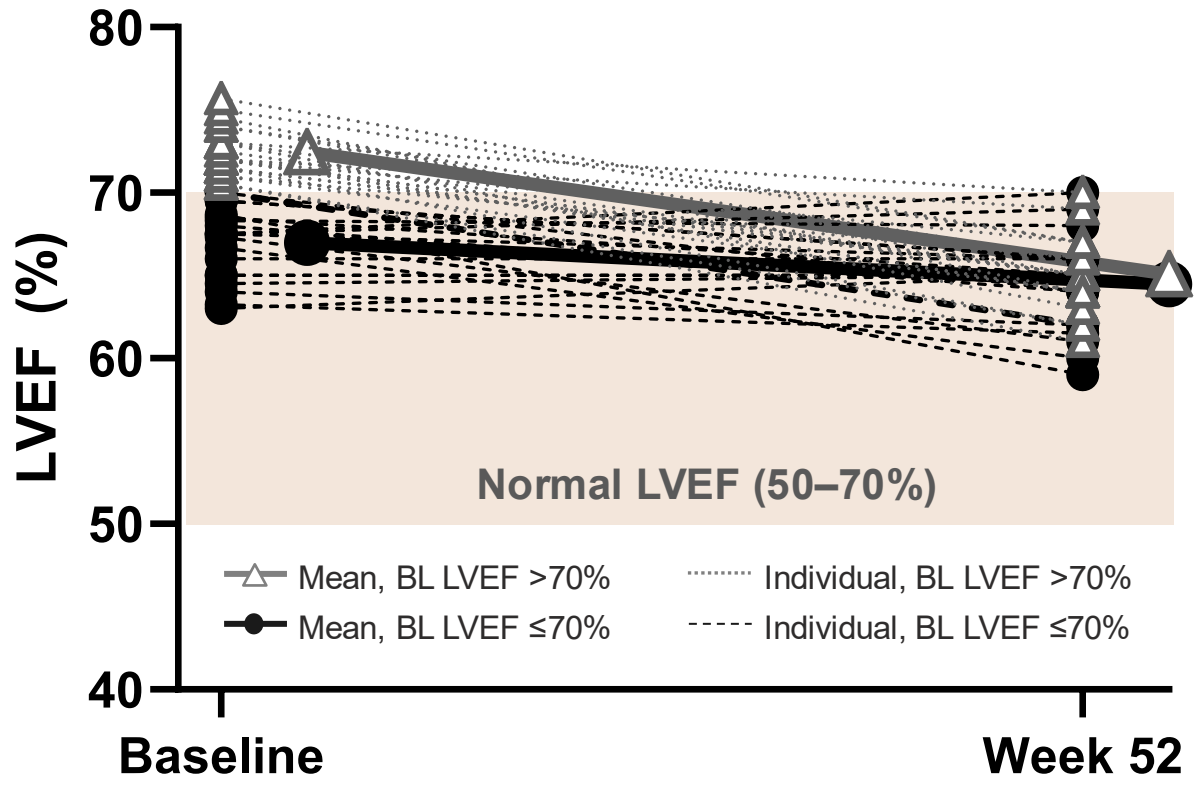
TARGETING

Consistent with altering the pathophysiology of disease

DOSING

Effective starting dose for the majority of patients

BHB-1893 moved LVEF into the normal range from a hypercontractile baseline



Baseline LVEF >70%

73% → **65%**

BL Wk 52

Baseline LVEF ≤70%

67% → **64%**

BL Wk 52

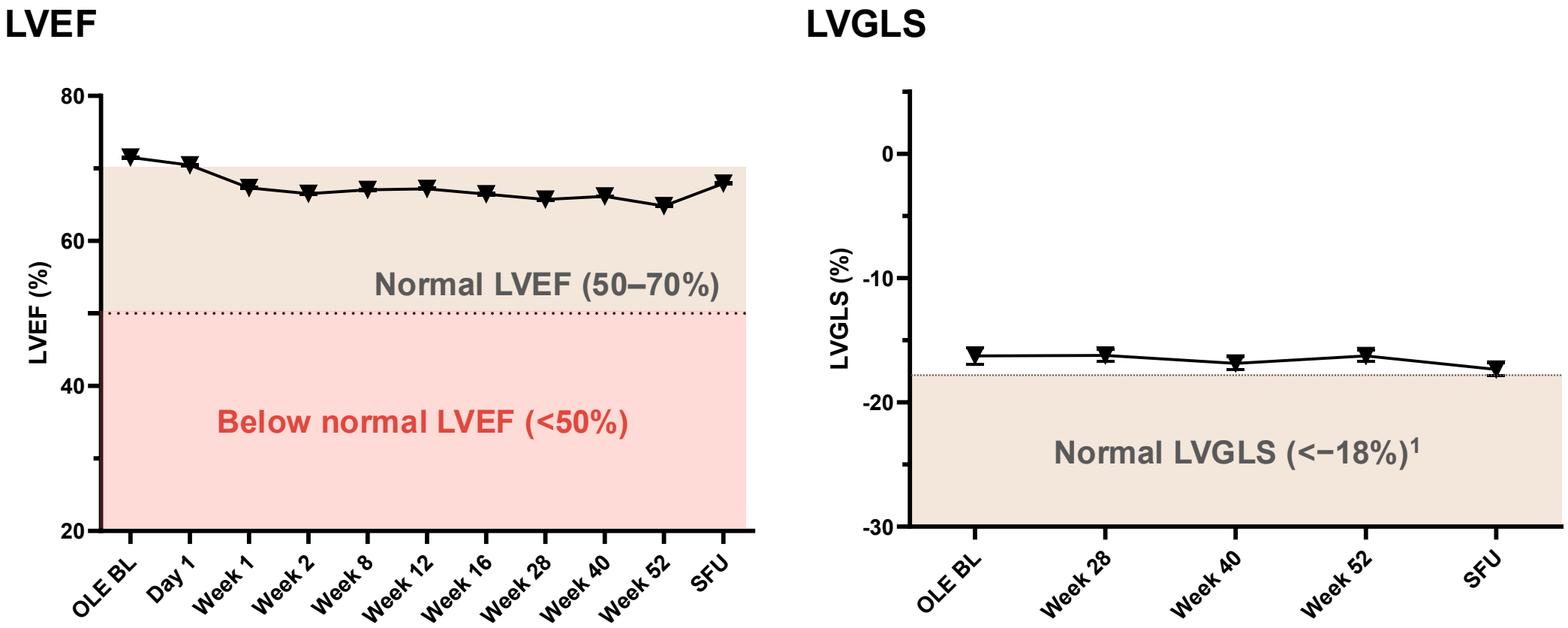
Two patients with transient LVEF <50% (48% and 49% site-measured), both resumed on lower dose

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BL, baseline; LVEF, left ventricular ejection fraction; Wk, week.

LVEF changes coincided with stable LVGLS suggesting maintained systolic function



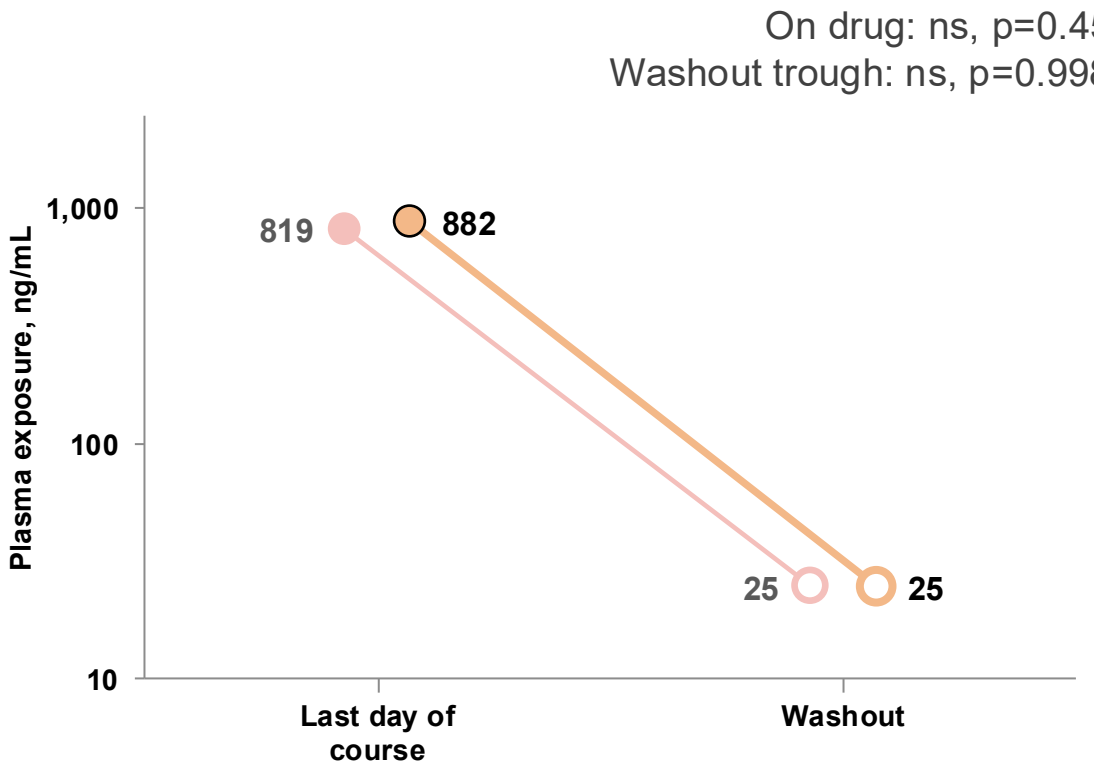
Database lock: June 2026

1. ASE/EACVI. Clinical applications of strain echocardiography: a clinical consensus statement. J Am Soc Echocardiogr. 2025.

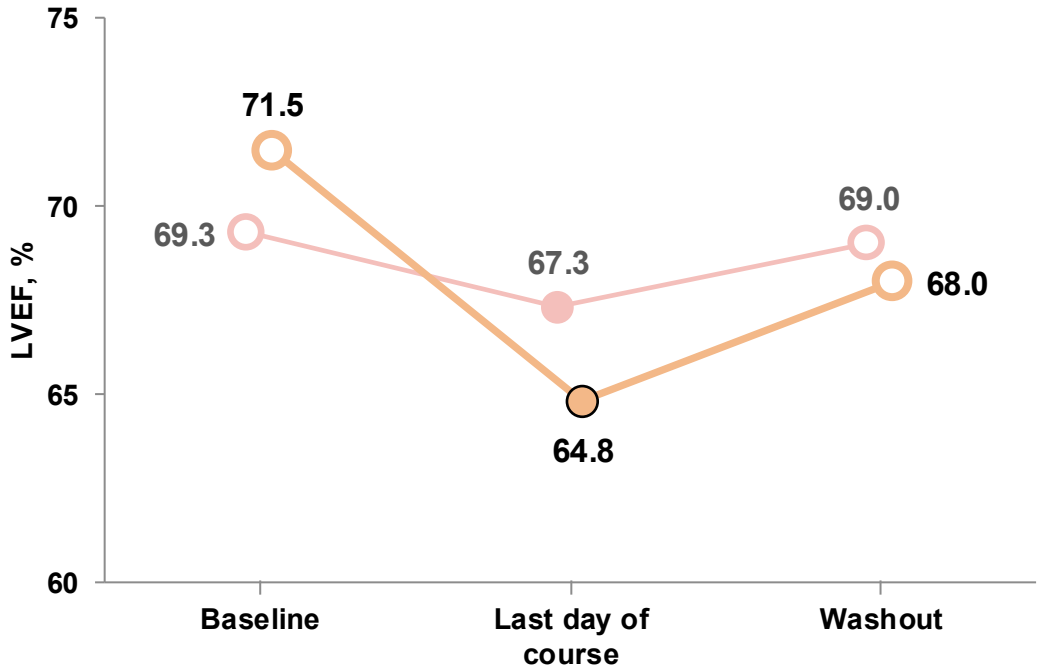
BL, baseline; LVEF, left ventricular ejection fraction; LVGLS, left ventricular global longitudinal strain; OLE, open-label extension; SFU, safety follow-up.

Drug was cleared during washout after both courses; LVEF returned to baseline

Plasma drug exposure by course



LVEF by course



12-wk course (Group 2) 52-wk course (pooled OLE)

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A: Welch t-tests from summary stats: Group 2 Wk 12 (n=14) vs pooled OLE Wk 40 (n=42); OLE Wk 1 (n=39) vs SFU (n=41). Trough SEM 18.1 and 9.8. B: Mean LVEF; filled markers on drug, open markers off drug. Baseline: Group 2 (n=14) and OLE baseline (n=42), as in baseline characteristics.
LVEF, left ventricular ejection fraction; ns, not significant; OLE, open-label extension; SEM, standard error of the mean; SFU, safety follow-up; Wk, week.

Partial return to baseline after 52 weeks, consistent with reverse remodeling

LVOT-G and NT-proBNP after washout

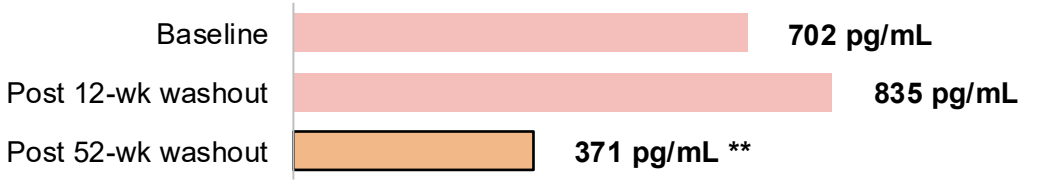
Valsalva LVOT-G



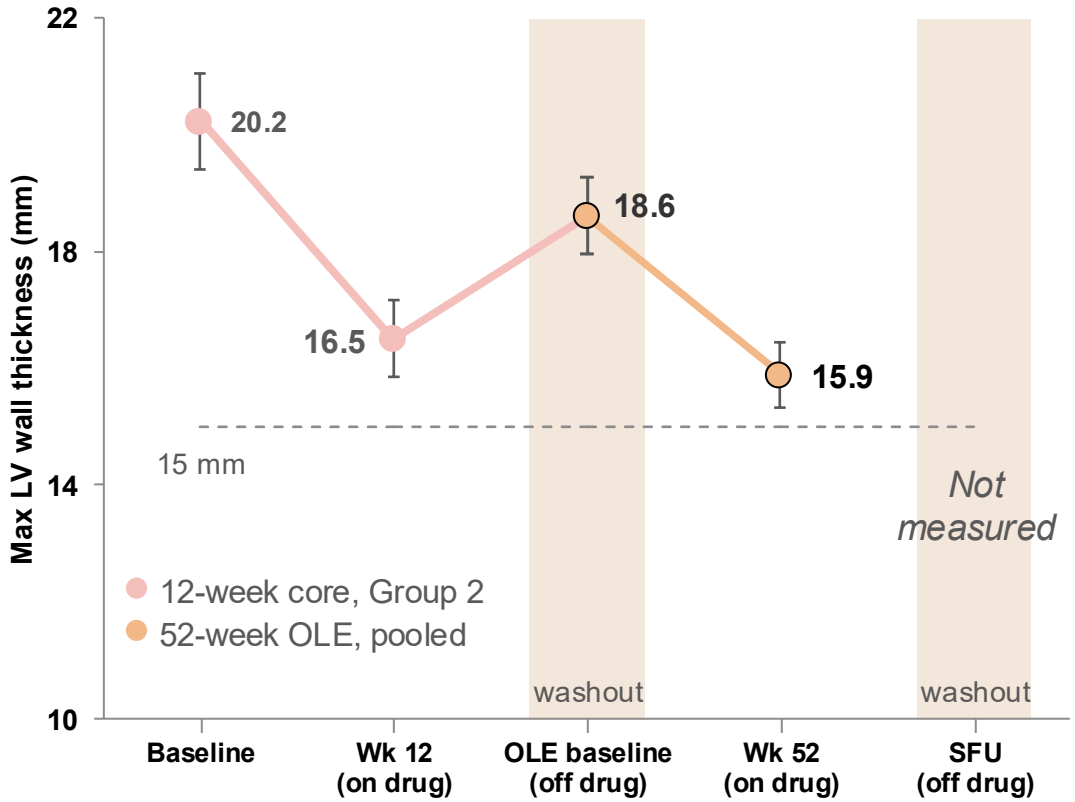
Rest LVOT-G



NT-proBNP



Max LV wall thickness over time



Database lock: June 2026

*C: Group means. Baseline, Group 2 core baseline; post 12-wk washout, OLE baseline; post 52-wk washout, SFU. **p<0.01, ***p<0.001, post 12-wk vs post 52-wk washout (Welch test; NT-proBNP on log scale); exploratory, unadjusted. D: Group means ± SEM. n: Group 2 baseline 13, Wk 12 12; pooled OLE baseline 38, Wk 52 36. No wall thickness measured at SFU. LV, left ventricular; LVOT-G, left ventricular outflow tract gradient; NT-proBNP, N-terminal pro-B-type natriuretic peptide; OLE, open-label extension; SEM, standard error of the mean; SFU, safety follow-up; Wk, week.*

BHB-1893 treatment was generally well-tolerated with no on-treatment severe or serious AEs

n (%)	12-Week Core Period (n = 42)	52-Week Extension Period (n = 42)
Any TEAEs	**34 (81.0)**	**38 (90.5)**
Mild	31 (73.8)	33 (78.6)
Moderate	3 (7.1)	4 (9.5)
Severe	0	1 (2.4) [a]
Any TRAEs	**12 (28.6)**	**12 (28.6)**
Mild	12 (28.6)	12 (28.6)
Moderate	0	0
Severe	0	0
Any TEAE leading to permanent study drug discontinuation	0	0
Any TEAE leading to study drug interruption and/or dose reduction	0	2 (4.8)
Any Serious TEAE	0	1 (2.4) [b]
AESI: LVEF <50%	0	2 (4.8)

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[a] Severe TEAE of platelets decreased occurred during washout. [b] Serious TEAE of chest discomfort occurred during washout. AE, adverse event; AESI, adverse event of special interest; LVEF, left ventricular ejection fraction; TEAE, treatment-emergent adverse event; TRAE, treatment-related adverse event.

52-week Phase 2 OLE results highlight BHB-1893's promising clinical profile

DEPTH

Rapid responses that deepen with sustained treatment

TARGETING

Consistent with altering the pathophysiology of disease



DOSING

Effective starting dose for the majority of patients

Simplified dosing opportunity: majority of patients maintained at 40mg starting dose

Last Dose Level	Week 12 BID cohorts 1 & 2 (n=28)	Week 52 OLE 40-60-80 BID (n=42)
20 mg BID	2 (7.1%)	2 (4.8%)
40 mg BID	**17 (60.7%)**	**24 (57.1%)**
60 mg BID	8 (28.6%)	8 (19.0%)
80 mg BID	1 (3.6%)	8 (19.0%)

Limited titration required given robust gradient response seen with 40mg dose at Week 1

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Database lock: June 2026
BID, twice daily; OLE, open-label extension.

oHCM phase 2 data demonstrated potential for best-in-disease profile for BHB-1893 as a next-generation CMI

Potential simplest dosing regimen

- Majority of patients maintained at starting dose throughout study
- 67% of patients achieved Valsava LVOT-G <30 mmHg within 1 week (~83% rest LVOT-G <30 mmHg); high response rate prior to titration supports *potential for fixed dose approach*

New evidence of potential best-in-disease profile

- Meaningful pVO_2 improvement of 2.7 mL/kg/min at 52 weeks (all cycle-based CPET)
- 90% of patients achieved Valsalva LVOT-G <30 mmHg during 52 weeks of treatment

Support for mechanistic differentiation

- Minimal reduction in systolic function required to achieve therapeutic benefic
- New data suggests potential for reverse modeling with longer time on treatment; consistent with findings in nHCM

Demonstration of consistent clinical profile

- Reproduced rapidity and depth of response in both 12-week core treatment period of 52-week OLE
- Deeper response seen with time on measures of feel and function, and biomarkers

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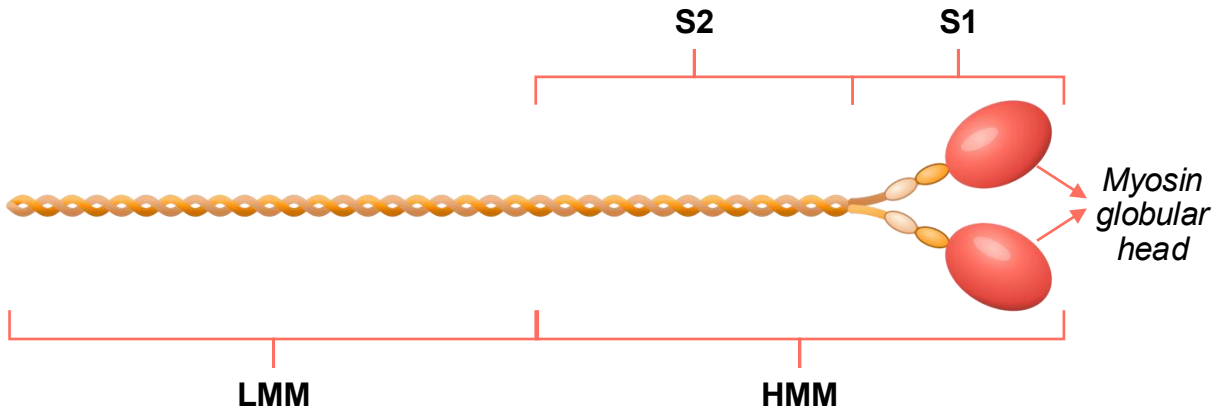
OLE: open-label extension; PK: pharmacokinetic; DDI: drug-drug interaction.

BHB-1893 Mechanistic Differentiation

Marc Evanchik, M.S.
Chief Scientific Officer

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BHB-1893 is a next-generation CMI with a potential best-in-class profile



- BHB-1893 is a next-generation, selective CMI shown to reduce actin-myosin crossbridge formation, **addressing the underlying pathophysiology of HCM**[1,2]
- BHB-1893 is designed to bind directly to the myosin motor head subfragment S1 and slow early systolic contraction[3]

PK/PD Profile[3]

- **Short effective half-life of ~12 hours**
- **Rapid onset of action and rapid reversibility** of PK and PD effects
- **Shallow exposure-response for LVEF**
- **Predictable PK across populations**, with multipathway metabolism and no significant DDIs anticipated
- Passive **elimination primarily through renal clearance** of parent

BHB-1893's mechanism of action reduced excess cross-bridge formation, with the potential to improve myocardial relaxation and diastolic function, reduce hyperdynamic contractility, and improve functional capacity and symptoms in HCM.[1,2,4,5]

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Source: 1. Wang L, et al. ACC 2026. 2. Marian AJ. Circ Res. 2021. 3. Company filings. 4. Wang L, et al. Heart Failure 2026. 5. Spudich JA. Pflügers Arch. 2019.
ATPase: adenosine triphosphatase; DDI: drug-drug interaction; HCM: hypertrophic cardiomyopathy; HMM: heavy meromyosin; LMM: light meromyosin; LVEF: left ventricular ejection fraction; PD: pharmacodynamics; PK: pharmacokinetics; S: subfragment.

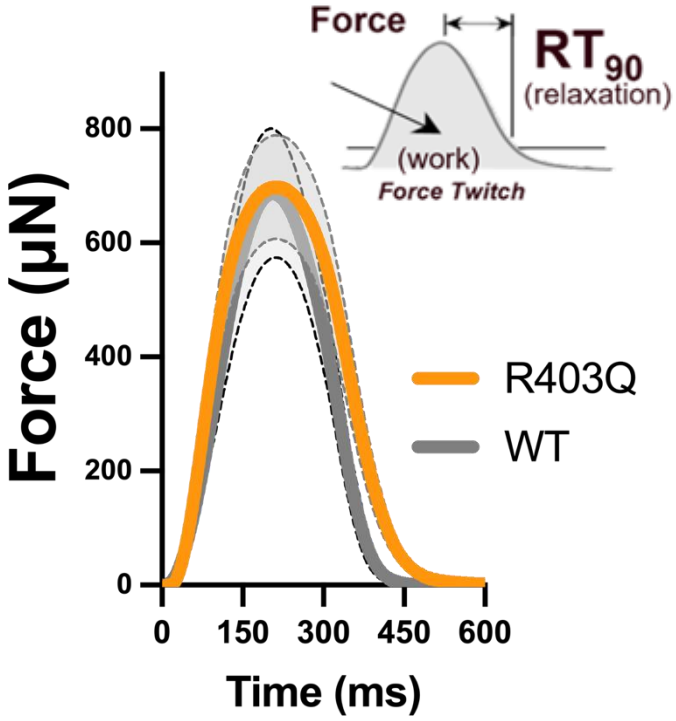
HCM engineered heart tissue (EHT) systems are robust models for assessing contractility and impact on relaxation

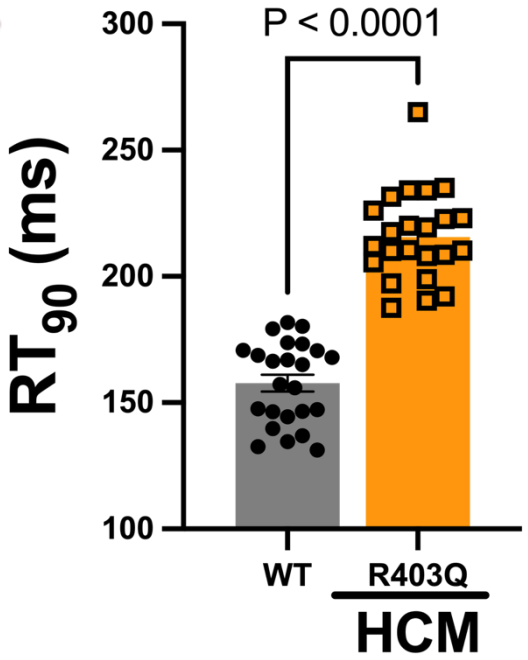
Slower twitch decay
Same force; R403Q relaxes later

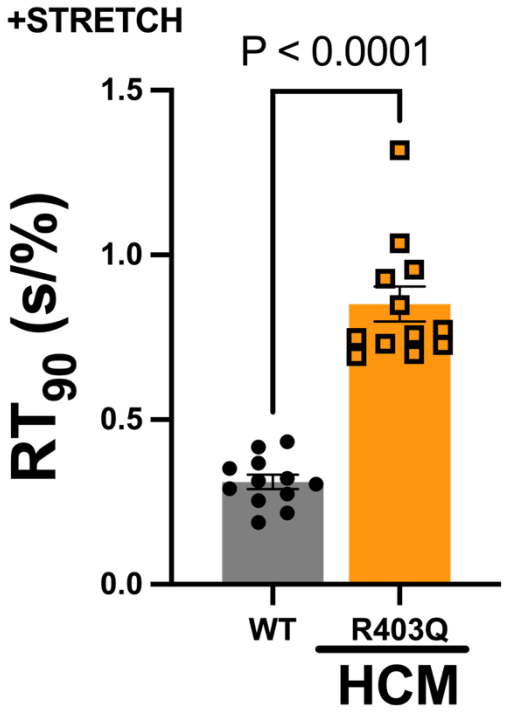
HCM tissues relax slower
RT90 is longer in HCM than WT

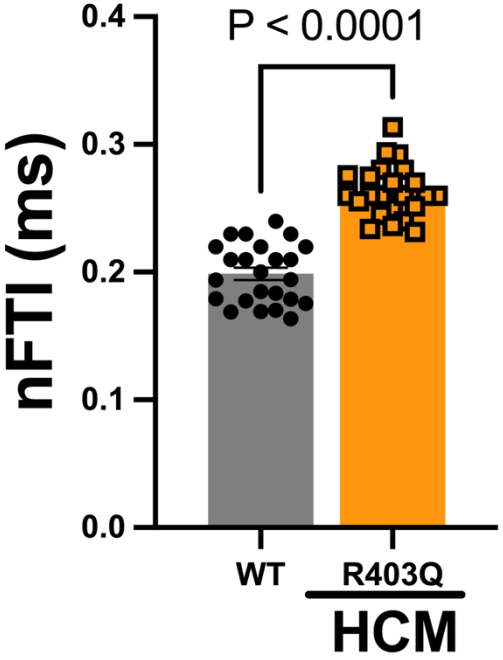
Stretch reveals the defect
Stretch mimics higher filling load

More non-productive work
Matched force – higher nFTI in HCM



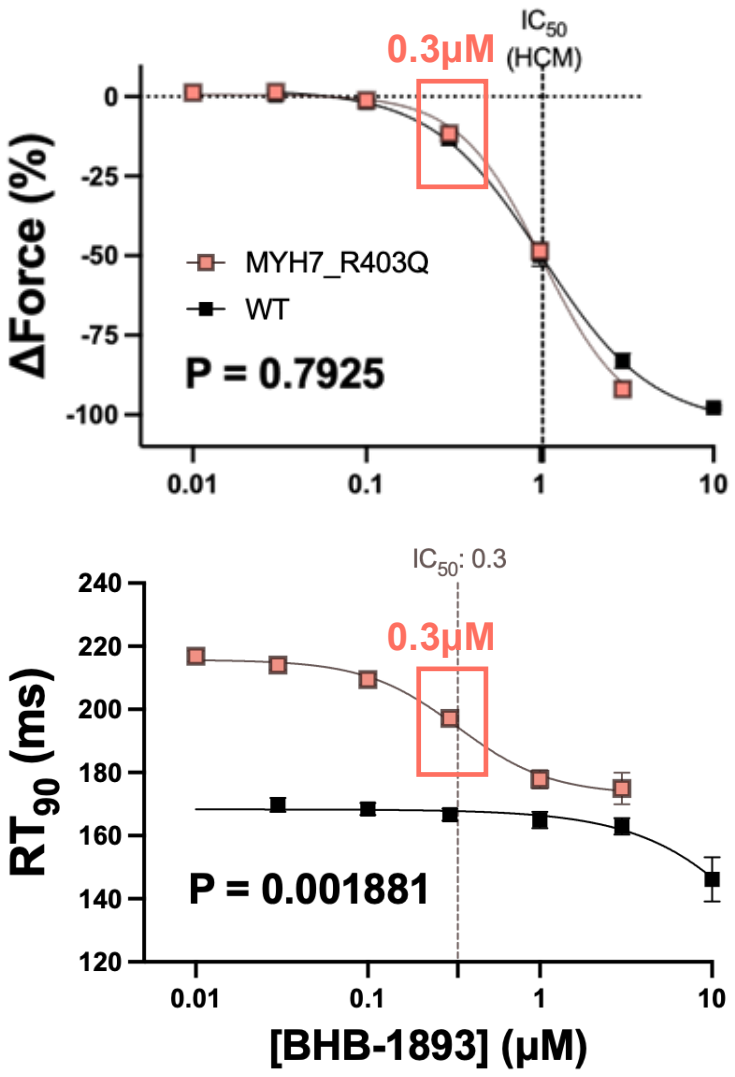






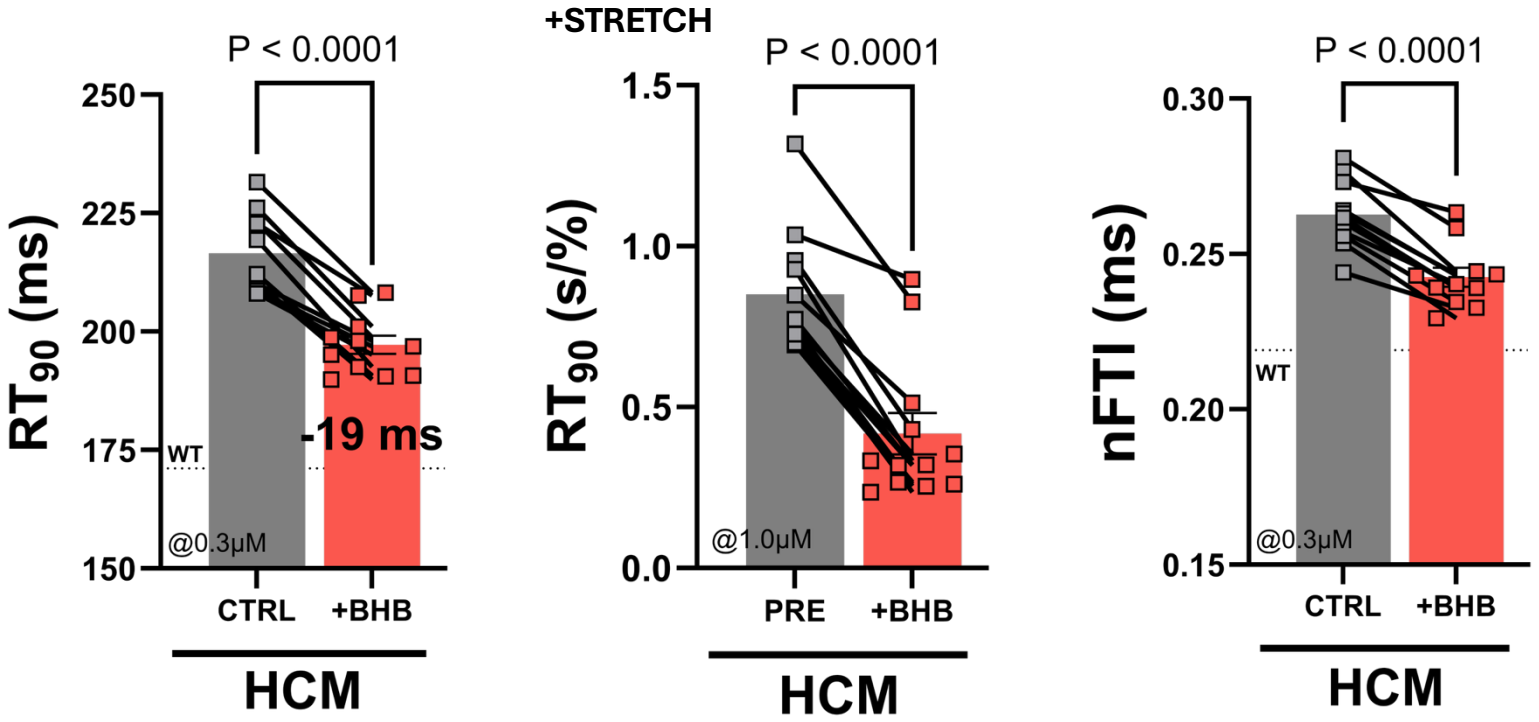
Twitch traces are mean (solid) ± 95% CI (dotted). Bar data are mean ± SEM. EHT, engineered heart tissue; HCM, hypertrophic cardiomyopathy; WT, wild type; RT90, time from peak force to 90% relaxation; nFTI, normalized force-time integral.

BHB-1893 rescued relaxation performance in HCM



Preserved Twitch Force

Negligible (~10%) peak force reduction at 0.3 μM in both WT and HCM, suggesting predictable LVEF changes



Shortened relaxation in HCM

Rescued stress response (+STRETCH)

Reduced non-productive work

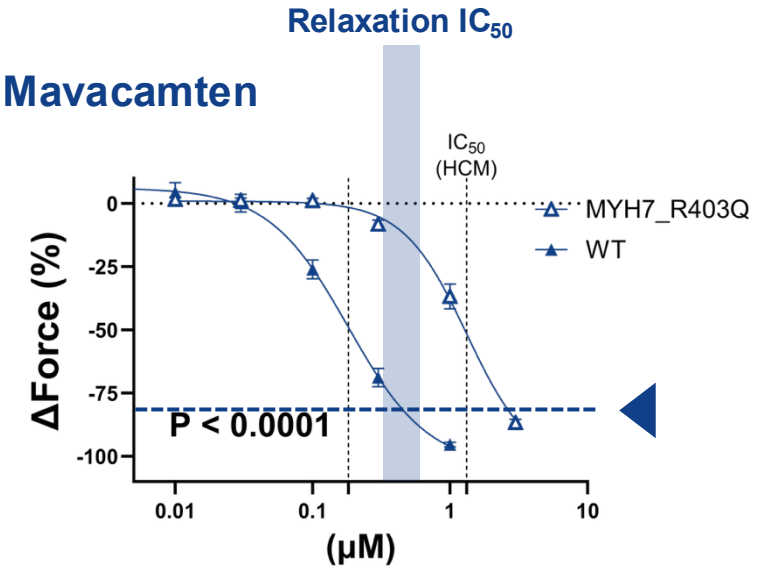
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Data are mean ± SEM. BHB, BHB-1893; HCM, hypertrophic cardiomyopathy; IC_{50}, half-maximal inhibitory concentration; LVEF, left ventricular ejection fraction; RT_{90}, time to 90% relaxation; SEM, standard error of the mean; WT, wild type.

BHB-1893's pharmacology supports early relaxation and predictable impact on contractile force

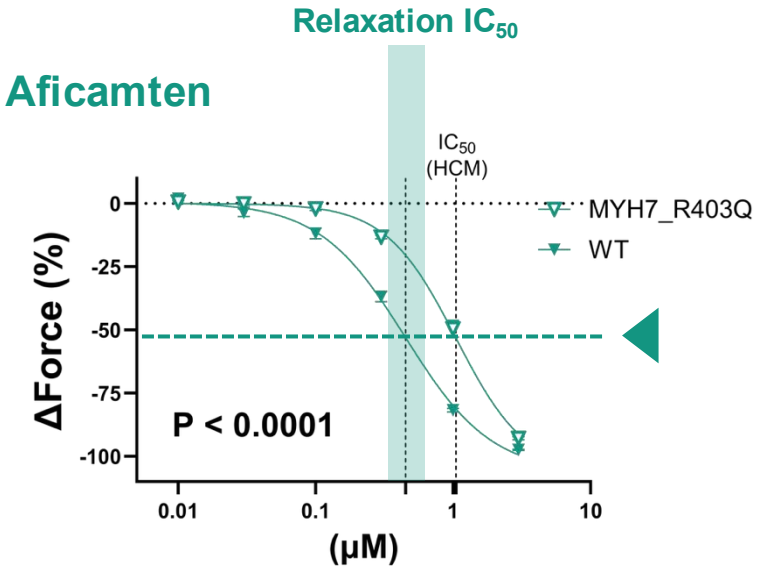
Contraction

Mavacamten



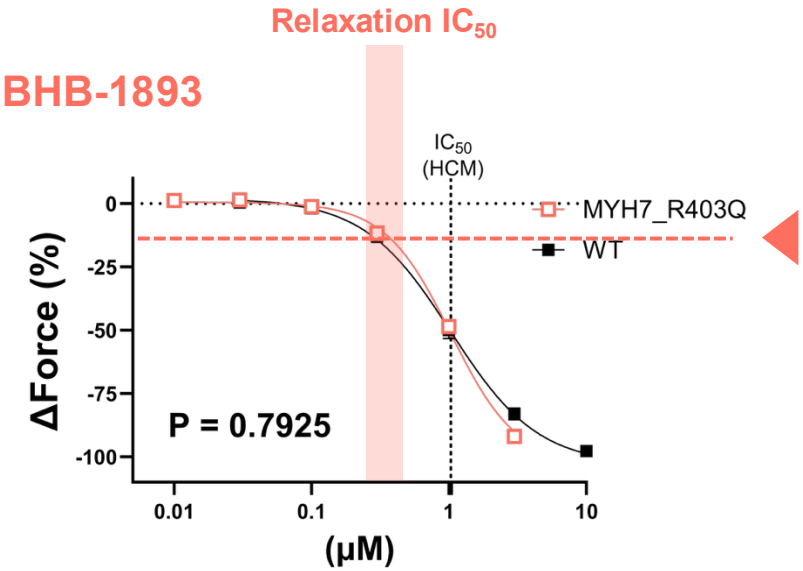
>75% inhibition of normal function[1]

Aficamten



~50% inhibition of normal function[1]

BHB-1893



~10% inhibition of normal function[1]

BHB-1893 differentiating features

- Tight control of aberrant myosin heads – minimal difference in impact on contractility between WT and mutant tissue
- Ability to achieve desired diastolic benefit with reduced systolic cost compared to first-generation CMIs

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1. Inhibition estimates based on IC50 concentrations for RT90

nHCM Phase 2 Diastolic Data

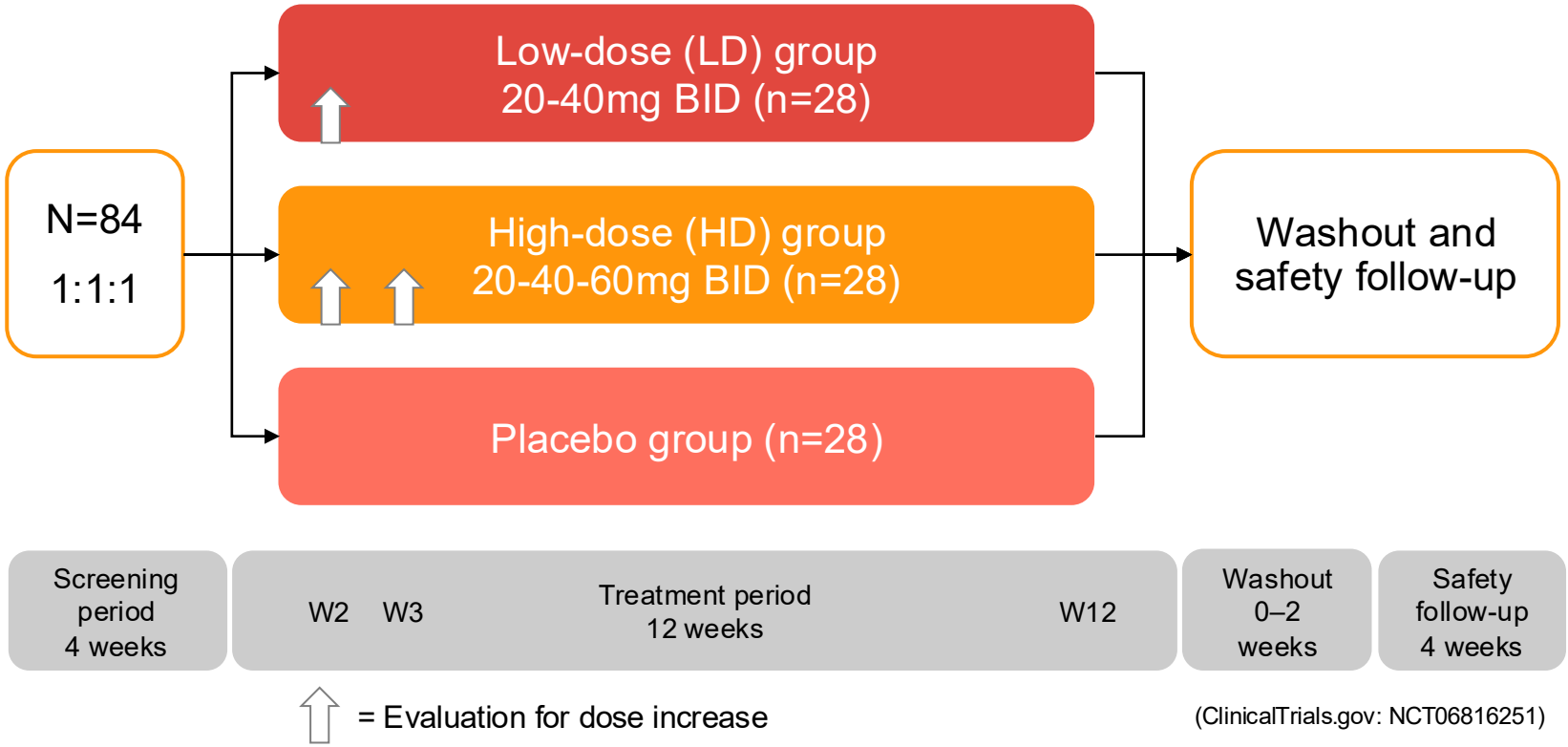
Anjali Owens, M.D.,
Associate Professor, Cardiovascular Medicine, Penn Medicine

Phase 2 placebo-controlled trial in nHCM patients over 12 weeks

Heart Failure & World Congress on Acute Heart Failure 2026

This study enrolled adult nHCM patients with: LVEF ≥60%, NYHA class II-III, resting and Valsalva LVOT-G <30 mmHg at screening, maximal wall thickness ≥15 mm or ≥13 mm with family history of HCM, and KCCQ-CSS ≥30 to ≤85.

Design



(ClinicalTrials.gov: NCT06816251)

Key Endpoints

Primary endpoint: Safety; LVEF < 50%

Biomarkers: NT-proBNP, cardiac troponin I

Feel and function: KCCQ-CSS, pVO_2

Echo: Diastolic, structural

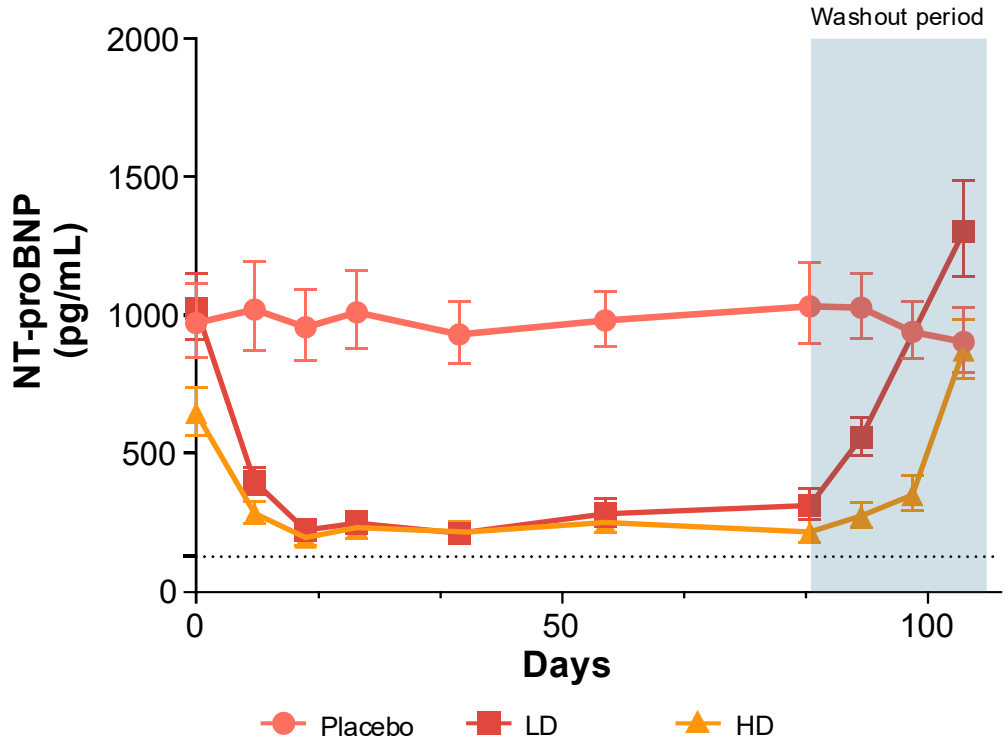
Dose Titration Criteria

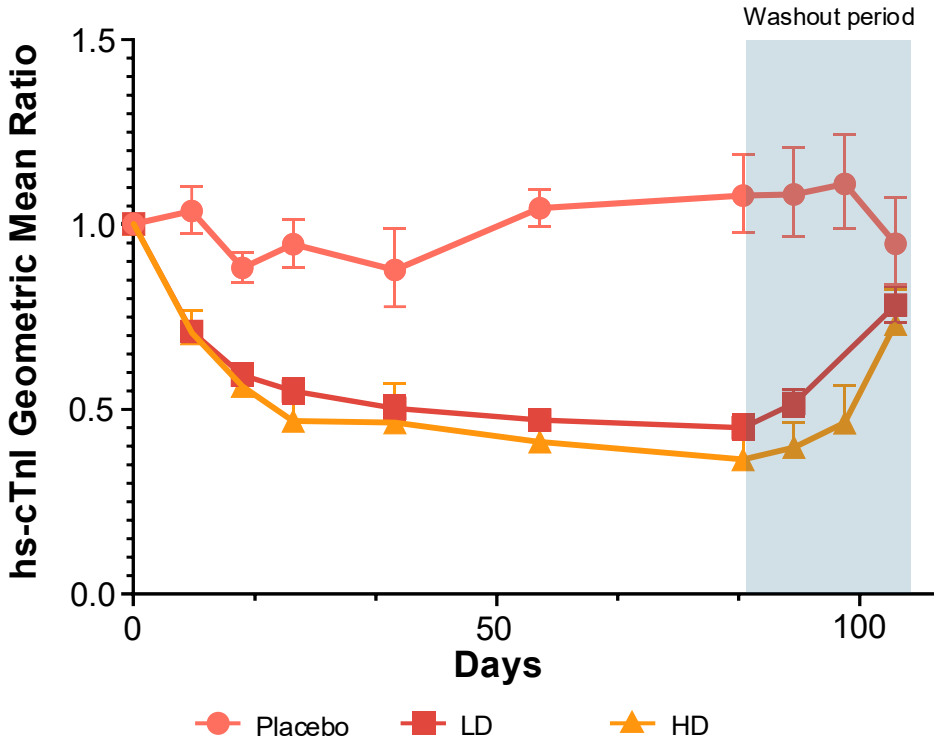
LVEF (%)	Adjustment
≥55%	Increase Dose
<55% and ≥50%	Maintain Dose
<50% and ≥45%	Decrease Dose
<45%	Drug Interrupted

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nHCM: non obstructive hypertrophic cardiomyopathy; LVOT-G: left ventricular outflow tract gradient; LVEF: left ventricular ejectionfraction; KCCQ-CSS: Kansas City Cardiomyopathy Questionnaire-Clinical Summary Score; BID: twice daily; QD: once daily; OLE: Open Label Extension. Phase 2 study was sponsored by Hengrui in China.

Rapid and deep response on key cardiac biomarkers through 12-week treatment period rapidly reversed after washout





68% decrease from baseline in the HD group

60% decrease from baseline in the HD group

42% of patients at 60 mg below 125 pg/mL[a]

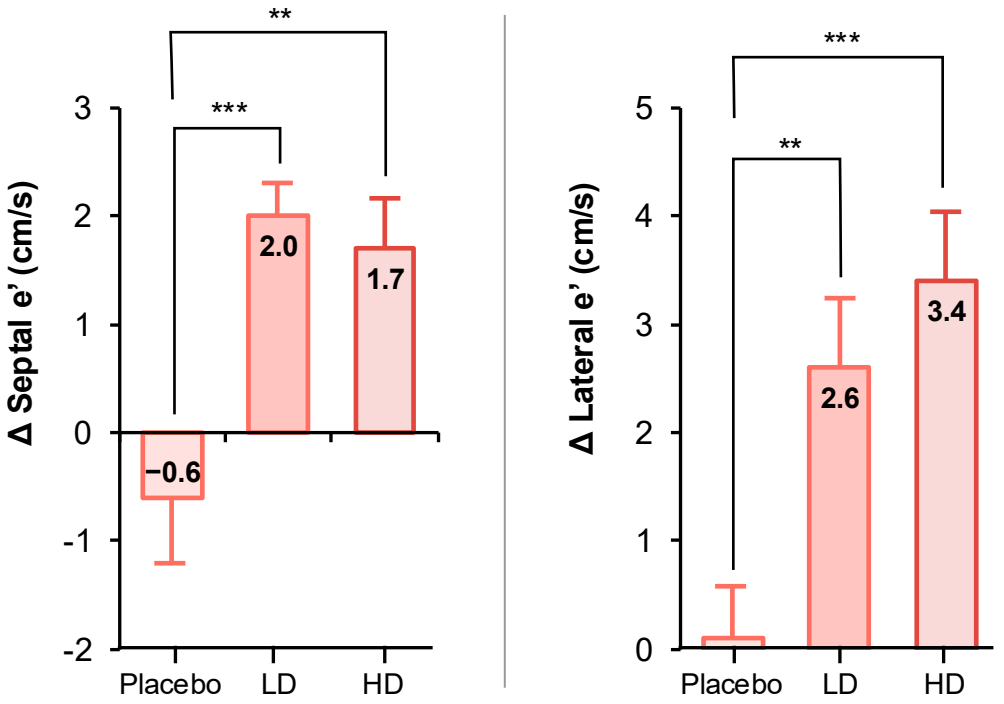
Geometric mean and SE; n ≥27 (Day 0 to Day 84). LD group: 20-40 mg BID; HD group: 20-40-60 mg BID. BID: twice daily; HD: high-dose; hs-cTnI: high-sensitivity cardiac troponin I; LD: low-dose; NT-proBNP: N-terminal pro-B-type natriuretic peptide.

[a] 125 pg/mL upper limit of normal for individuals aged <75 years and cutoff for ruling out heart failure (2022 AHA/ACC/HFSA guidelines).

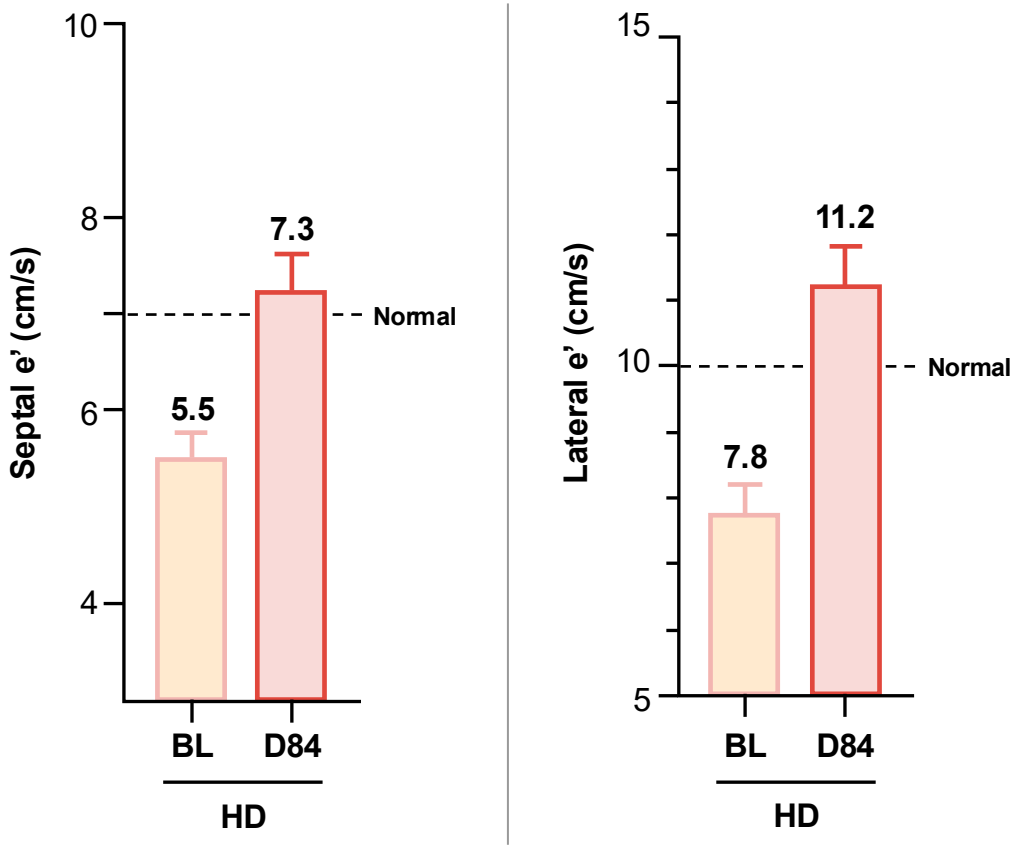
Source: Wang L. et al., Heart Failure 2026.

Diastolic function improved with normalization of e' velocities at 12 weeks

Changes in Early Diastolic Relaxation (12w)

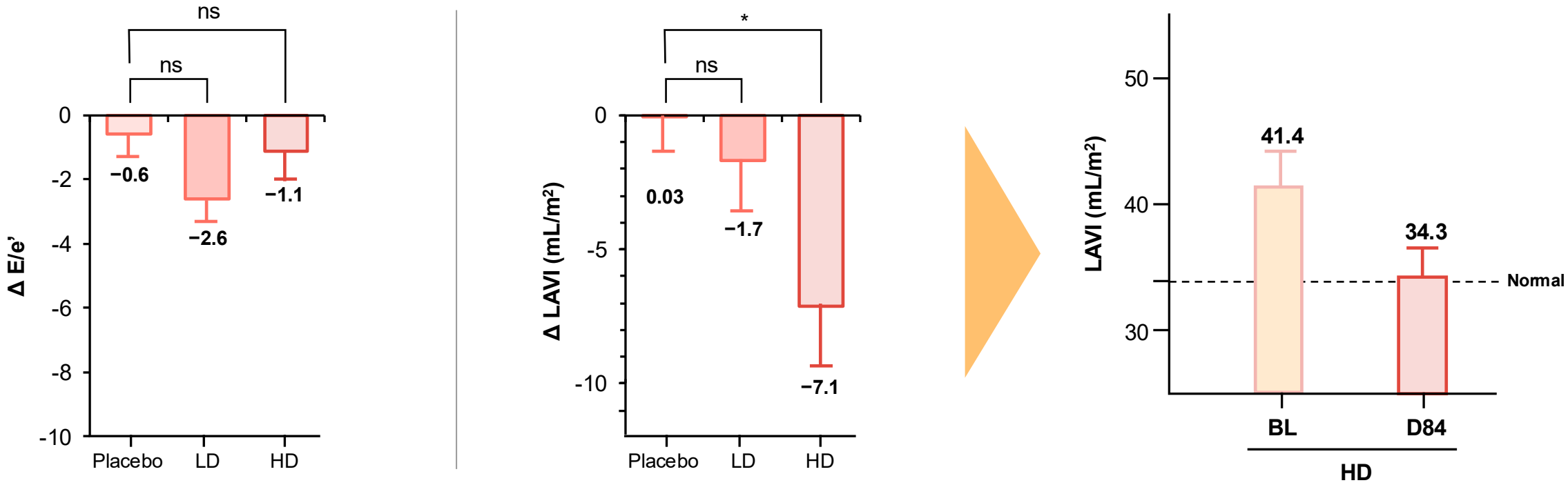


Velocities Moved From Abnormal Toward Normal (12w)



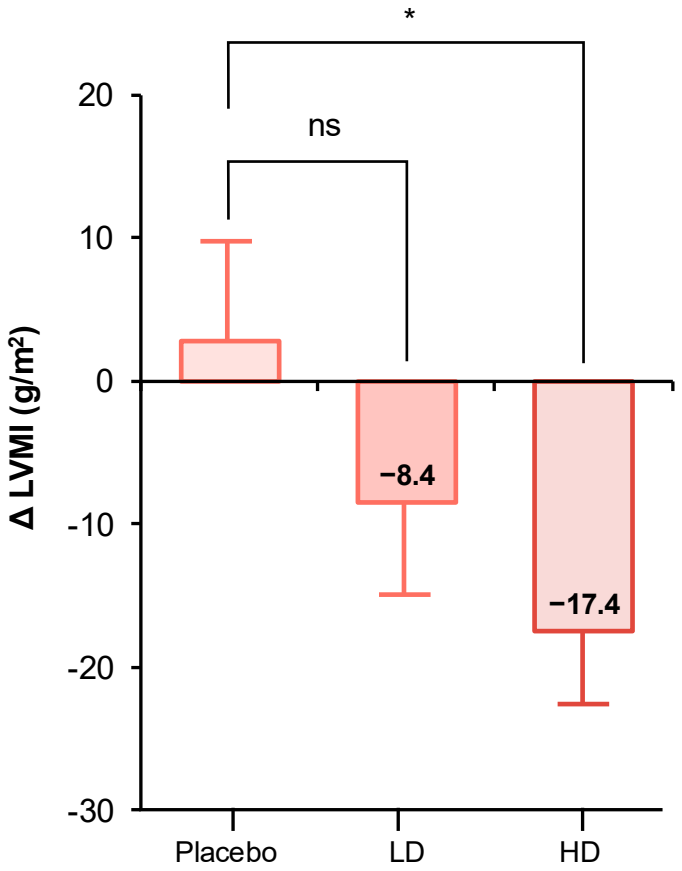
*Data are presented as mean (SEM); **P<.01; ***P<.001. LD group: 20-40 mg BID; HD group: 20-40-60 mg BID.*
Source: Wang L. et al., Heart Failure 2026.
BID: twice daily; BL: baseline; D: day; e': early diastolic (lateral and septal) mitral annular velocity; HD: high-dose; LD: low-dose.

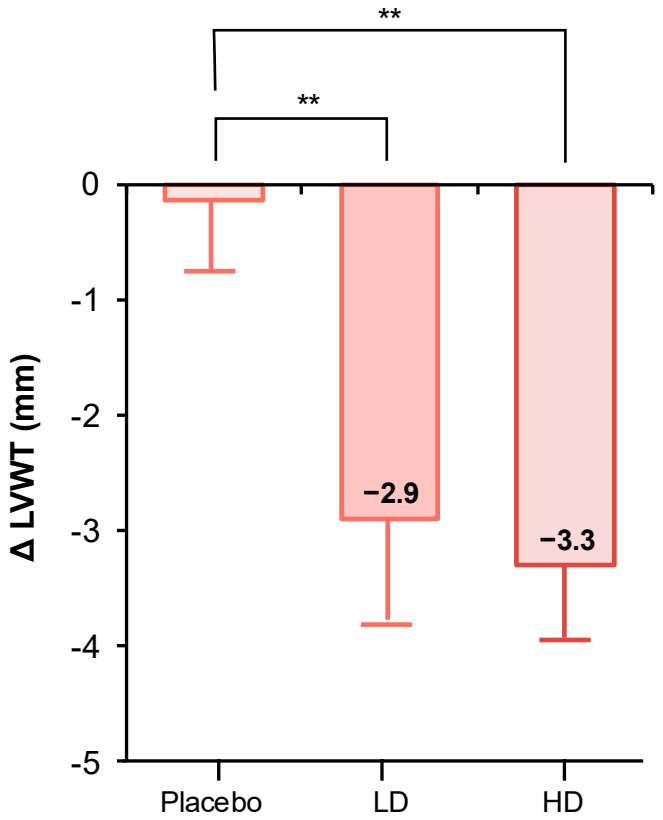
Reduced chronic left atrial pressure overload consistent with hemodynamic benefit and potential normalization of LAVI at 12 weeks



*Data are presented as mean (SEM); *P<.05. LD group: 20-40 mg BID; HD group: 20-40-60 mg BID.*
Source: Wang L. et al., Heart Failure 2026.
BL: baseline; E/e': early transmitral inflow velocity/early diastolic (lateral and septal) mitral annular velocity, an estimate of left ventricular filling pressure; HD: high-dose; LAVI: left atrial volume index; LD: low-dose; ns, not significant.

Significant reductions in left ventricular mass index and wall thickness observed in the first 12 weeks





*Data are presented as mean (SEM); *P <.05; **P <.01. LD group: 20-40 mg BID; HD group: 20-40-60 mg BID.*
Source: 1. Wang L. et al., Heart Failure 2026. 2. Company filings.
BID: twice daily; HD: high-dose; LD: low-dose; LVMI: left ventricular mass index; LVWT: left ventricular wall thickness; ns, not significant.

Safety – overview of AEs

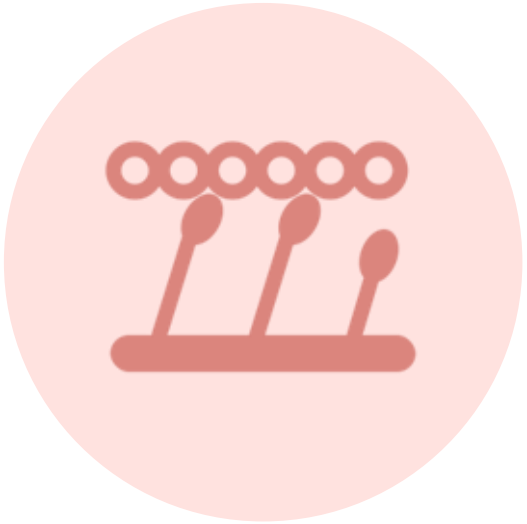
- There were no serious AEs or AEs leading to treatment interruption, discontinuation, or death[1]
- 4 participants experienced LVEF values below 50% (ranging from LVEF 46% to 49%), all of which returned to ≥50% at subsequent visits following dose reductions[1]
- No patients were discontinued from BHB-1893 for a low LVEF[1]
- Safety and tolerability were generally consistent with prior studies of BHB-1893[1-3]

	Placebo (n = 28)	BHB-1893 LD (n = 28)	BHB-1893 HD (n = 28)	BHB-1893 Total (N = 56)
Any TEAEs	19 (67.9)	23 (82.1)	24 (85.7)	47 (83.9)
Mild	17 (60.7)	18 (64.3)	18 (64.3)	36 (64.3)
Moderate	2 (7.1)	5 (17.9)	6 (21.4)	11 (19.6)
Severe	0	0	0	0
Any TEAE leading to permanent discontinuation	0	0	0	0
Any TEAE leading to study drug interruption	1 (3.6)	0	0	0
Any serious TEAE[a]	1 (3.6)	2 (7.1)	1 (3.6)	3 (5.4)
LVEF <50%	0	1 (3.6)	3 (10.7)	4 (7.1)

LD group: 20-40 mg BID; HD group: 20-40-60 mg BID. [a] LD: cellulitis (n = 1), otolithiasis (n = 1); HD: atrial fibrillation (n = 1); placebo: atrial tachycardia (n = 1).
Source: 1. Wang L. et al., Heart Failure 2026. 2. Wang L, et al. ACC 2026. 3. Kang L, et al. ESC Congress 2025.
AE: adverse event; EF: ejection fraction; HD: high dose; LD: low dose; LVEF: left ventricular EF; TEAE: treatment-emergent AE.

BHB-1893 emerging mechanistic data is consistent with the diastolic benefits observed in the clinic

Mechanistic validation of CMIs in disease



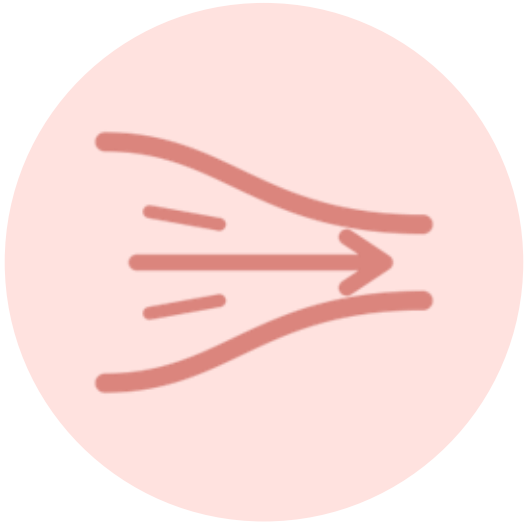
Continued support for the role of excess myosin cross-bridges in driving diastolic dysfunction

Benefits with minimal systolic cost



BHB-1893 demonstrated unique ability to improve diastolic function while preserving systolic performance

Improvements independent of gradient



nHCM Ph2 data showed diastolic improvements occurring independent of gradient relief

BHB-1893 Development Strategy

Travis Murdoch, M.D., FRCP(C)
President, Chief Executive Officer

BHB-1893's profile may enable seamless and immediate adoption across a wide range of HCM patients and physicians

Potential best-in-disease profile

Rapid and deep effects
in oHCM & nHCM

Clinical activity deepened
Through 52 weeks in oHCM

Favorable safety profile
Observed to date

Potential differentiated mechanistic profile

Improved **diastolic function**

Benefits **with minimal systolic cost**

Meaningful activity **across both forms of HCM**

Aim to optimize cardiology practices

Simplified dosing
with minimal/no titration

Higher capacity
to manage more patients

Broad access
for patients who need therapy

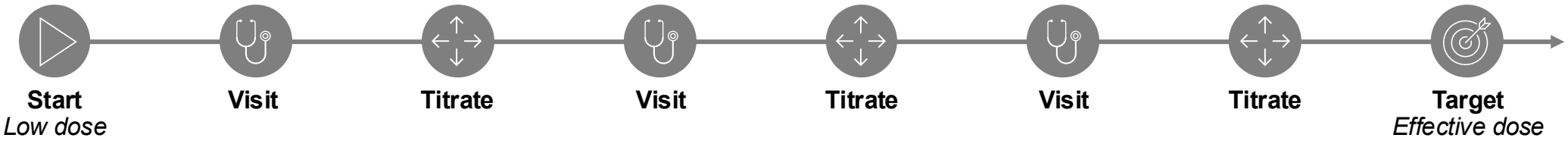
May expand the addressable HCM population to reach more patients in need of therapy

Simplifying Dosing

We are developing BHB-1893 to have a simple dosing approach with immediate clinical activity and minimal or no titration

Illustrative

First-generation CMIs today



Multiple months and several patient visits often required prior to reaching effective dose

BHB-1893 development goal



Maximize simplicity by eliminating titration and starting at effective dose

BHB-1893's emerging profile has the potential to reduce burden and expand utilization beyond HCM specialty centers

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Our oHCM development plan provides opportunities to simplify dosing and expand access while optimizing efficacy

Planned studies

Titration at week 1 & 2

Observed in Ph2

20%–30% single-step titration

57-60%

Patients remain on starting dose

One step titration at week 4

LIONHEART-HCM

20%–40% single-step titration

60-80%

Potential for large proportion of patients to remain on starting dose*

Opportunity to optimize efficacy for patients with residual gradient

Fixed dose

TRUEHEART-HCM

100%

Fixed dose

Potential to broaden access and enable simple patient starts

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* *Illustrative example based on longer time to titration in LIONHEART compared to prior Ph2 studies*

Illustrative

TRUEHEART-HCM : oHCM Ph2b study with fixed dose design

Planned dosing

- Patients receive **fixed dose of 40mg BID**
- No titration visits over the course of the study

Study overview

- **Population:** Symptomatic oHCM on top of standard of care
- **Geography:** Initiate in US, with potential to broaden globally

Status

- **Received supportive FDA feedback** on fixed dose approach
- Protocol being finalized to enable **1H2027 initiation**

BID, twice daily

Broad clinical development plan to advance treatment of HCM

HRS-1893-301
Hengrui Phase 3
oHCM

- Symptomatic oHCM, on top of SoC in China
- Potential weekly titration 40, 60, 80 mg BID
- **Topline results 1H 2027**

LIONHEART-HCM
BHB-1893-301 · Ph3
oHCM

- Symptomatic oHCM, H2H vs beta blocker
- Potential single titration at Week 4
- **Interim analysis 2H 2027**

TRUEHEART-HCM
BHB-1893-201 · Ph2b
oHCM

- Symptomatic oHCM, on top of SoC
- Fixed dose 40mg BID
- **Trial initiation 1H 2027**

NOBLEHEART-HCM
BHB-1893-302 · Ph3
nHCM

- Symptomatic nHCM
- Potential single titration at Week 4
- **Trial initiation 1H 2027**

Participants from global studies will be candidates for long-term extension study

HEARTSONG-LTE

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CPET, cardiopulmonary exercise test; BL, baseline; DB, double-blind; KCCQ-CSS, Kansas City Cardiomyopathy Questionnaire clinical summary score; LTE, long-term extension; LVEF, left ventricular ejection fraction; LVOT-G, LV outflow tract gradient; n/oHCM, non-obstructive/obstructive HCM; pVO_2, peak oxygen uptake; SOC, standard of care

Anticipated Milestones

Multiple catalysts over the next 12+ months, leading to planned NDA filing for BHB-1893

Planned Data Readouts 3

1H27	HRS-1893-301 oHCM Phase 3 topline data
1H27	nHCM Phase 2 OLE 52-week data
2H27	LIONHEART-HCM oHCM interim analysis

Trial Initiations 2

1H27	NOBLEHEART-HCM nHCM
1H27	TRUEHEART-HCM oHCM fixed dose

Planned oHCM NDA filing

LIONHEART-HCM interim analysis
+
HRS-1893-301 final analysis

FDA acknowledged HRS-1893-301 plus the LIONHEART-HCM interim analysis may support future NDA submission

ADJUSTED CASH BALANCE – AS OF JUNE 30, 2026

$527M*

PROJECTED CASH RUNWAY INTO

2029

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** As adjusted cash balance includes June 30, 2026 cash and equivalents of $122.8M and IPO net proceeds of $404.5M closed in August, 2026*

Q&A