Kardigan:
Heart health, reimagined

July 2026

Kardigan™

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Powered to build the precision cardiovascular company of the future

SEASONED LEADERSHIP TEAM

LARGE, UNDERSERVED CV MARKET

3 LATE-STAGE PROGRAMS TARGETING HIGH UNMET NEED

CAPITAL-EFFICIENT CV MODEL

TRANSFORMATIVE TECHNOLOGY WITH PROLAIO™

BLUE-CHIP INVESTOR SUPPORT

CV = cardiovascular

Cardiovascular disease has seen limited R&D despite unmet need

~30%
Annual U.S. deaths caused by CVD

+$400B
Annual CVD spend - Expected to 3x in 3 decades

$1.8T
Expected CVD spend by 2050

5-15K
Patients per trial with broad populations & MACE endpoints

7%
Phase 3 trials for CVD[1]

5%
Drugs in development for CVD across industry pipeline[2]

CVD = cardiovascular disease, MACE = major adverse cardiovascular events

Sources: American Heart Association, Evaluate Pharma | 1 CV drugs represented only 7% of phase 3 clinical trials (2012) 2 Publicly traded U.S. companies with $500M to $5B market caps

Kardigan is building the precision cardiovascular company of the future

Our approach

Reimagining CV drug discovery and development through biology, data, and analytics

- **Targeting the root cause of disease**
- **Studying well-defined patient populations**
- **Harnessing proprietary, transformative technology**
- **Developing and commercializing differently**

Led by former MyoKardia leadership team, backed by leading healthcare investors

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Co-founder, Chief Executive Officer and Board Chair

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Jay Edelberg, MD, PhD
Co-founder and Chief Medical Officer

MYOKARDIA
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Bob McDowell, PhD
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Chief People Officer

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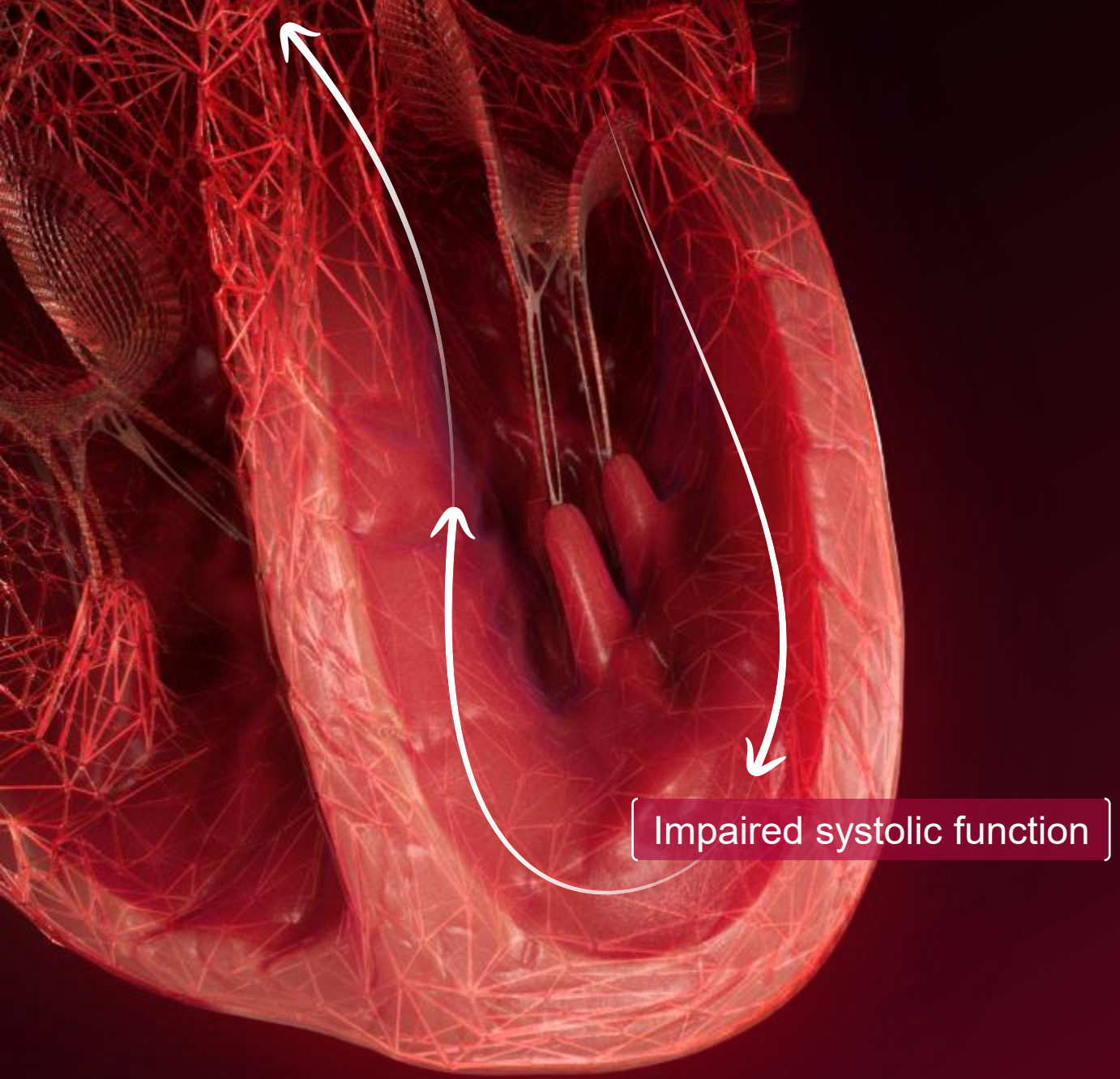
Powered to deliver precision cardiology at scale

Program	Indication	Phase 1	Phase 2	Phase 3	Upcoming milestones
Danicamtiv (Dani) Direct myosin activator	Genetic dilated cardiomyopathy (DCM)	kinship DCM			Phase 2b/3: Topline data 1H 2027
Ataciguat (ATA) Soluble guanylate cyclase activator	Moderate calcific aortic valve stenosis (CAVS)	KATALYST-AV			Phase 2b: 24-week interim data 1H 2027
Tonlamarsen (TLA) Angiotensinogen ASO	Acute severe hypertension (ASH)	KARDINAL ASH			Phase 2b: Topline data in 1H 2027

prolaio™ **proprietary platform advantage supports precision cardiology**



Danicamtiv in genetic dilated cardiomyopathy (DCM)

Danicamtiv: designed to address underlying defects of genetic DCM

WELL-DEFINED PATIENT POP

150-200K
diagnosed primary genetic/familial DCM patients (US)[1]

- DCM is the leading cause of heart transplants[2]
- No approved therapies for DCM
- Genotype+ DCM associated with worse outcomes[3]
- 40-50K sarcomeric patients (majority are MYH7 or TTNtv)

TARGETING ROOT CAUSE

Myosin Activator
restores myosin motor function

- Genetic DCM is caused by myosin dysfunction which impacts heart contractility
- Danicamtiv stabilizes myosin in the force-generating state to restore contractility and cardiac output

CLINICAL RESULTS

Improved Cardiac Function
in Phase 2a trial in DCM

- Improvement in contractility in both left atrium and left ventricle[4]
- Cardiac echo endpoints are known to correlate with functional improvement (pVO_2)[5]
- No clinically meaningful troponin elevations[4]

UPCOMING CATALYST

1H 2027
Phase 2b data readout

- Currently enrolling ~80 genetic DCM patients in Phase 2b
- Evaluating effect of danicamtiv on improving cardiac function and symptoms
- Danicamtiv administered orally as 50mg BID

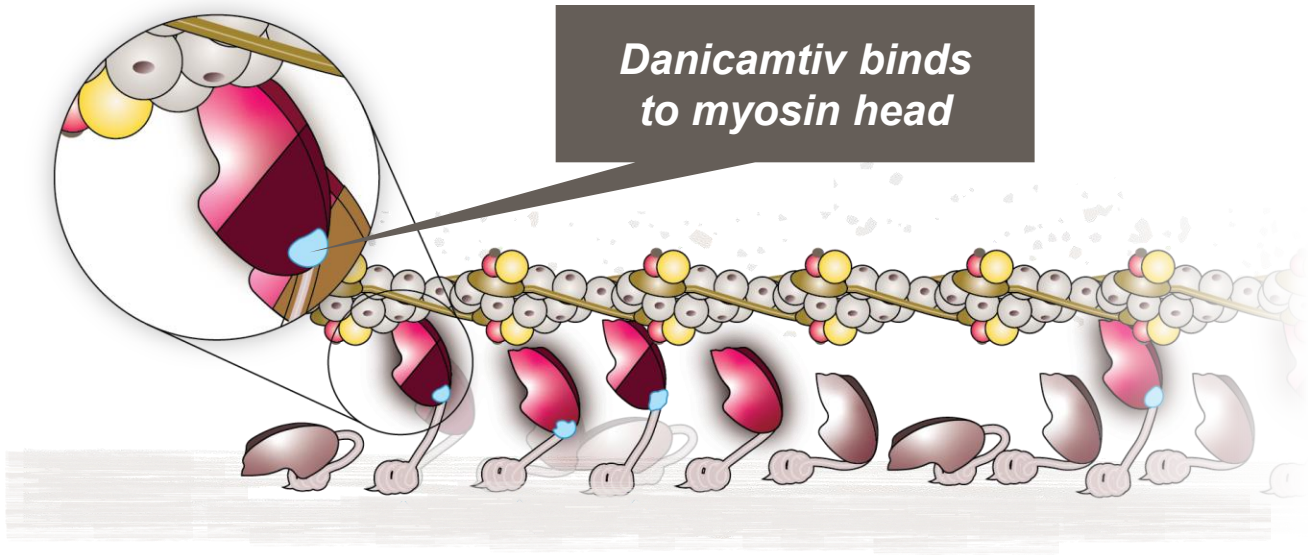
DCM = dilated cardiomyopathy | MYH7 = cardiac myosin heavy chain 7 | TTNtv = titin-truncating variants | pVO_2 = peak oxygen consumption | LA = left atrium | LV = left ventricle | BID = twice daily

1. Epidemiology Trinity market research and Company estimates: TTNtv: ~128-155k patients | MYH7: ~22-45k patients 2. Lakdawala NK, et al. Circ: Arrhythmia and Electrophysiology 2012;6.1:228-273,
3. Escobar-Lopez, et. Al.. J AM Coll Cardiol 2021;78:1682-99, 4. Lakdawala NK, et al. J Am Coll Cardiol. 2025;86(25): 2598–612; 5. Hasselberg NE, et al. Eur Heart J Cardiovasc Imaging. 2015;16(2):217-224

Danicamtiv targets the underlying cause of genetic DCM – stabilizing myosin to restore myocardial function

Myosin motor number and function are **key to myocardial function**

- Inactive “OFF” myosin
- Active “ON” myosin



Danicamtiv is designed to stabilize myosin by allowing recruitment from the “OFF" state to the “ON" state:

- **Restoring working myosin availability**
- **Enhancing the function of normal and defective myosin** in both the ventricle and atrium
- **Counteracting the deleterious effects of TTN variants** on myosin recruitment

Potentially preventing heart failure progression

DCM = dilated cardiomyopathy | TTN = titin

Source: Shen, S. et al. Journal of the American Heart Association (2021), 10(12)

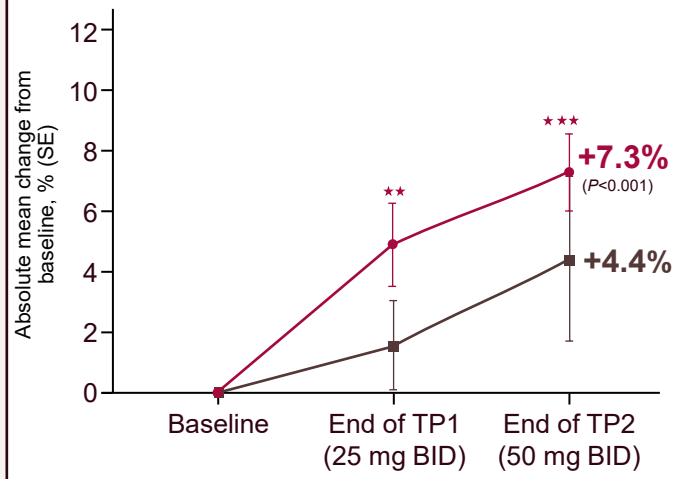
Improved cardiac function was observed following administration of danicamtiv in participants with sarcomeric DCM

Phase 2a Trial – Part A[1]

DCM Cohort: Sarcomeric: MYH7 + TTN | Other Causes

CHANGE FROM BASELINE TO 2 WEEKS

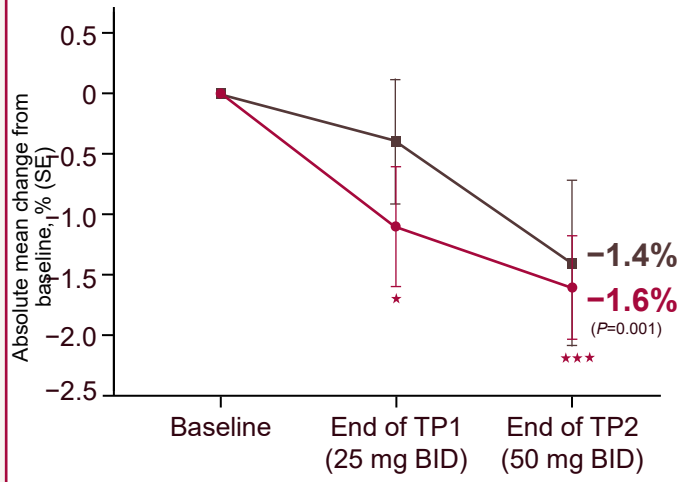
Left ventricular ejection fraction (LVEF)



KEY TAKEAWAYS

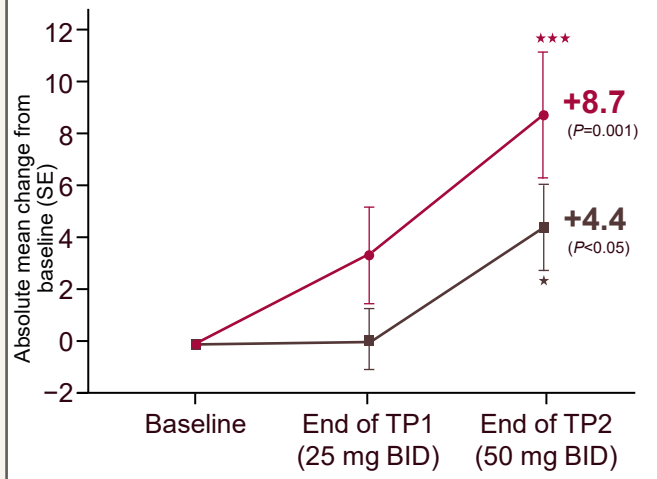
- Improved left ventricular function was observed in the MYH7 and TTN cohorts following administration of danicamtiv

Left ventricular global longitudinal strain (LVGLS)



- Danicamtiv treatment was associated with improved LVGLS
- Reductions in LVGLS correlate with improvement in pVO_2 (a measure of exercise capacity)[2]

Left atrial function index (LAFI)



- Danicamtiv treatment was generally associated with improved atrial function as well as ventricular function

In Part B, an open-label extension, directionally similar improvements at Week 12 were observed, supporting further investigation of danicamtiv for the treatment of genetic DCM

Other causes cohort includes participants with non-MYH7 and non-TTN or non-genetic DCM. *P<0.05, **P<0.01, ***P≤0.001 (P-values are derived from a Student's t-test). DCM = dilated cardiomyopathy | SE = standard error | TP = treatment period

1. Lakdawala NK, et al. J Am Coll Cardiol. 2025;86(25):2598–612; 2. Hasselberg NE, et al. Eur Heart J Cardiovasc Imaging. 2015;16(2):217-224

Danicamtiv was well tolerated in DCM patients

Asset	Phase 2a Study in Primary DCM – Part A	Phase 2a Study in Primary DCM – Part B
Study Population	Primary DCM (Sarcomeric/Other causes)	
Sample Size	n = 41	n = 19
Treatment Arms	danicamtiv 10 mg (n-2) danicamtiv 25 mg (n=41) danicamtiv 50 mg (n=39)	danicamtiv 25 mg (n-1) danicamtiv 50 mg (n=9) danicamtiv 75 mg (n=12)
Overall Safety Findings	Danicamtiv was well tolerated and there were no drug-related AEs leading to discontinuation An asymptomatic increase in cardiac troponin without any clinical signs of ischemia was detected in 3 participants in the Other causes cohort	Danicamtiv was well tolerated, with no new safety signals, Observed safety events were consistent with those seen in DCM patients An asymptomatic increase in cardiac troponin without any clinical signs of ischemia was detected in 2 participants
Deaths	None	None
Serious Adverse Events	No SAEs	5 participants reported SAEs; SAEs in one participant led to discontinuation of study drug; these were considered not study drug-related by the Sponsor

Danicamtiv was well tolerated; 265 individuals have been exposed to danicamtiv across 10 completed clinical trials

DCM = dilated cardiomyopathy

1. Lakdawala NK, et al. J Am Coll Cardiol. 2025;86(25):2598–612;

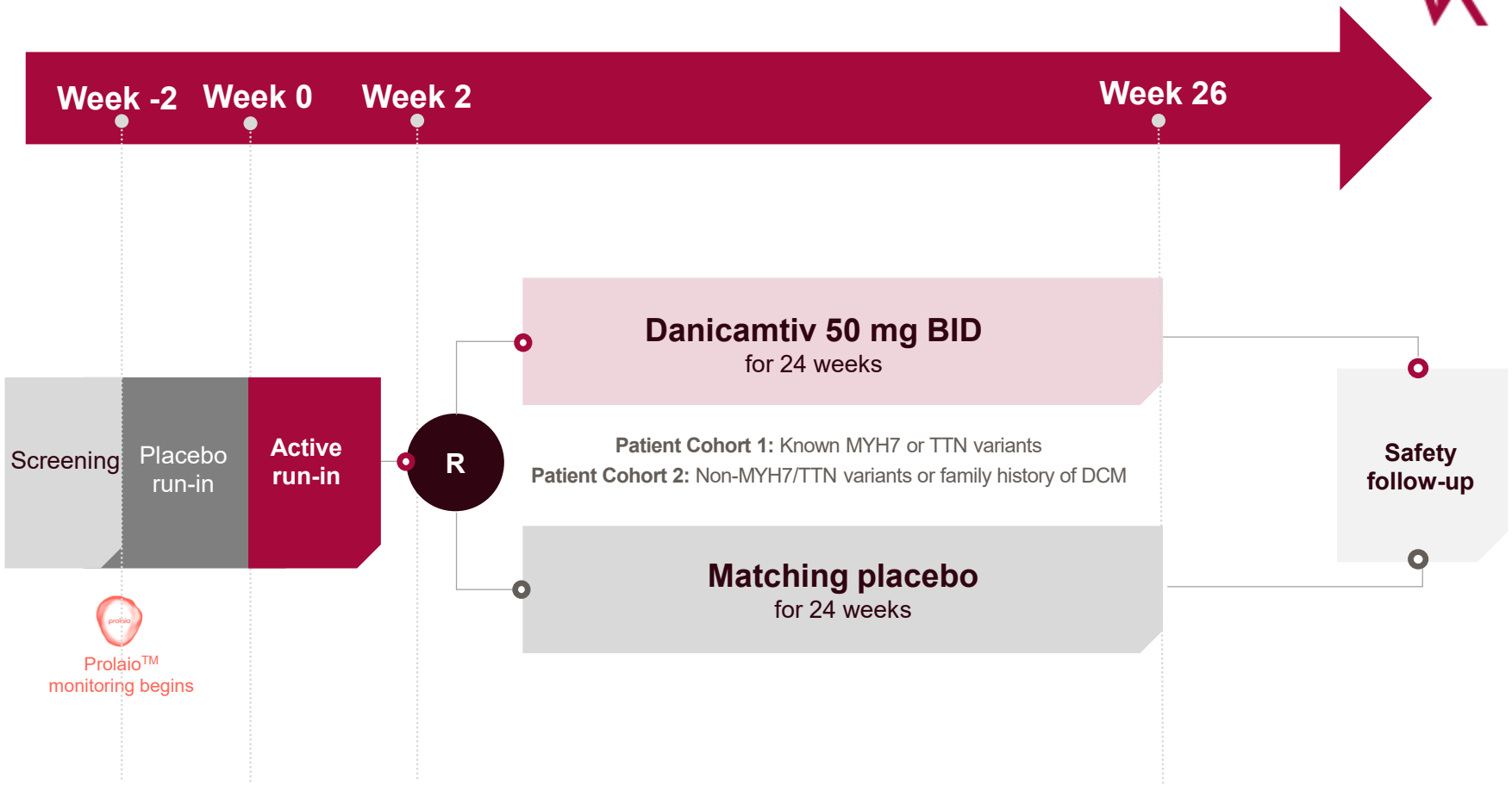
Phase 2b/3 adaptive study in genetic DCM

Key study inclusion criteria:

- Primary DCM, NYHA class II-IV
- Known DCM-associated genetic variant, or family history of DCM
- On stable GDMT for ≥ 4 weeks

Key endpoints:

- **Phase 2b Primary Endpoint:** Change in LAFI, baseline to Week 26 ***(~80 participants)***
- **Phase 3 Registrational Endpoint:** Change in pVO_2, baseline to Week 26 ***(~250 participants)***



Upcoming Catalyst: Phase 2b expected 1H 2027

DCM = dilated cardiomyopathy | LAFI = left atrial functional index | BID = twice daily | R = randomization | MYH7 = cardiac myosin heavy chain 7 | TTN = Titin | pVO2 = peak oxygen consumption

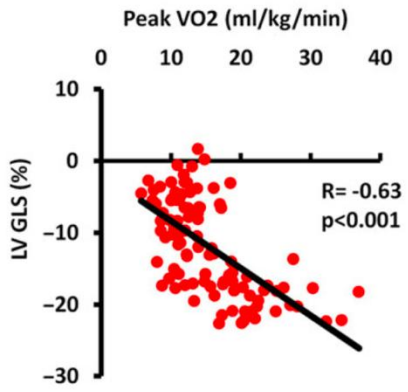
Source: Trial Reference: https://clinicaltrials.gov/study/NCT07210723

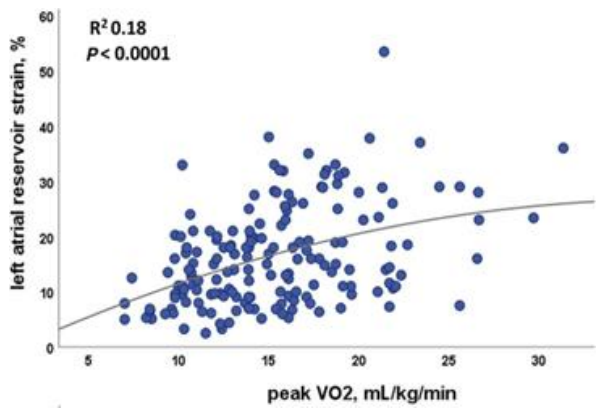
Correlation between echo parameters and pVO_2 derisks clinical development

Phase 2a data

- Part A data showed rapid improvements in function in both the LA and LV after just two weeks
- Part B data showed re-exposure was associated with directionally similar improvements in LVEF, LVGLS and LAFI; early (2-week) gains correlated with late (12-week) improvements
- Potential to see similar improvements in cardiac function with limited troponin elevation in the ongoing Phase 2b study

pVO_2 correlated with LV function (GLS) and LA function and filling parameters





- Reduced LA function was associated with worse outcomes and higher mortality[1]
- Measures of LVGLS, LA function, and size/volume correlate with pVO_2 in HF and in DCM[2]

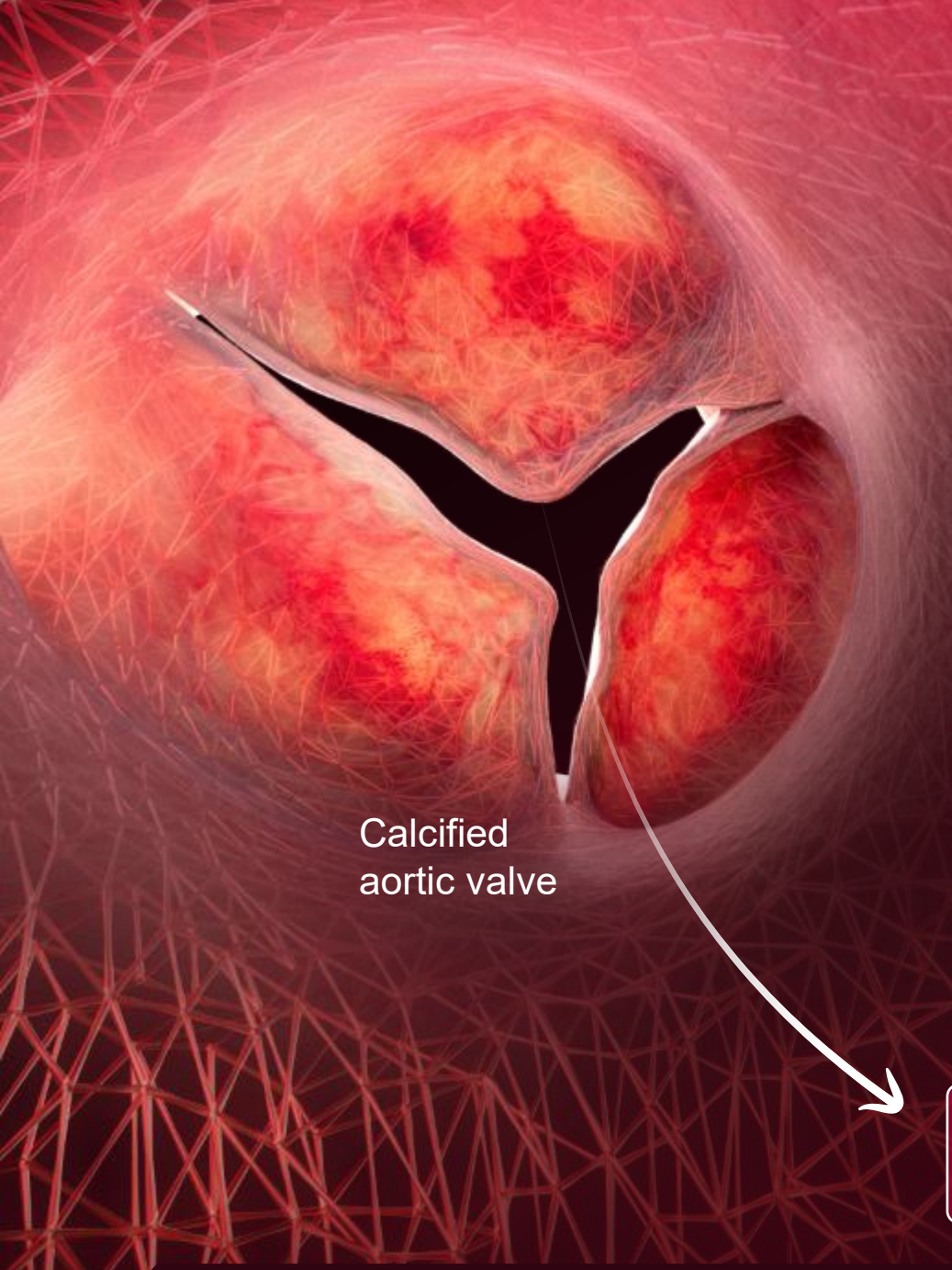
Phase 2b topline data: change in LAFI & pVO_2, baseline to Week 26 (~80 participants)

- Improved LA function correlates with pVO_2 improvement, supporting a path to clinical benefit
- Will continue to monitor totality of evidence across all echo parameters to confirm our development path

DCM = dilated cardiomyopathy | HF = heart failure | LVEF = left ventricular ejection fraction | LVGLS = left ventricular global longitudinal strain | LAFI = left atrial function index

Source: Correlation of Peak VO2 and LVGLS (left chart): Hasselberg NE, et al. Eur Heart J Cardiovasc Imaging. 2015;16(2):217-224, Correlation of Left Atrial Reservoir Strain and peak VO2 (right chart): Maffeis, et. al. ESC Heart Failure (2022) | 1 Shamekhi, J., et al. Clin Res Cardiol 111, 944–954 (2022) | 2 Terzi, et. Al ., International Heart Journal (2005) 46(1):123-31

Ataciguat in moderate calcific aortic valve stenosis (CAVS)



Impaired blood flow

Ataciguat: a novel therapeutic for moderate CAVS

WELL-DEFINED PATIENT POP

~1M
moderate CAVS patients (US)

- No treatments for CAVS: only “watchful waiting”
- 50% 2-year survival rate without intervention[1-3]
- 11-24% 1-year mortality rates post-TAVR[4]

TARGETING ROOT CAUSE

Novel sGC activator
is positioned to restore sGC function to reduce Ca^{2+} deposition

- Calcium accumulation in the aortic valve drives disease progression
- ATA restores sGC activity to directly reduce calcium deposition
- Works without systemic vascular effects (e.g., hypotension)

CLINICAL RESULTS

Slowed AVC progression & improved cardiac function
by >50% in Phase 2 trial

- Improvements in calcification translated to meaningful cardiac benefit (as measured by LVEF, CO, LVMI, E/e’)[5]
- Cardiac benefit is known to translate to functional improvement (as measured by pVO_2)[6]

UPCOMING CATALYST

1H 2027
Phase 2b data readout

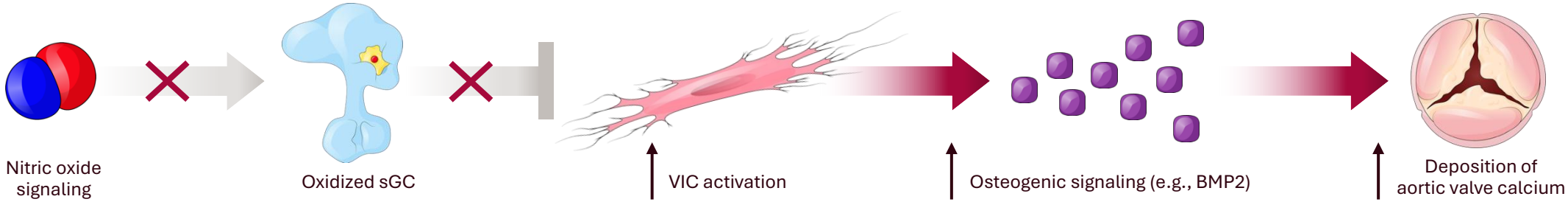
- Currently enrolling ~130 moderate CAVS patients in Phase 2b
- Evaluating effect of ATA on slowing progression of valvular dysfunction

Moderate is defined as aortic valve area (AVA) >1.0 and <1.5 cm1 with mean gradient 20-40 mmHg4,5 | CAVS = calcific aortic valve stenosis | TAVR = transcatheter aortic valve replacement | sGC = aoluble guanylate cyclase | AVC = aortic valve calcium | LVEF = left ventricular ejection fraction | CO = cardiac output | LVMI = left ventricular mass index, E/e’ = ratio early mitral inflow velocity to mitral annular diastolic velocity (measure of diastolic function)

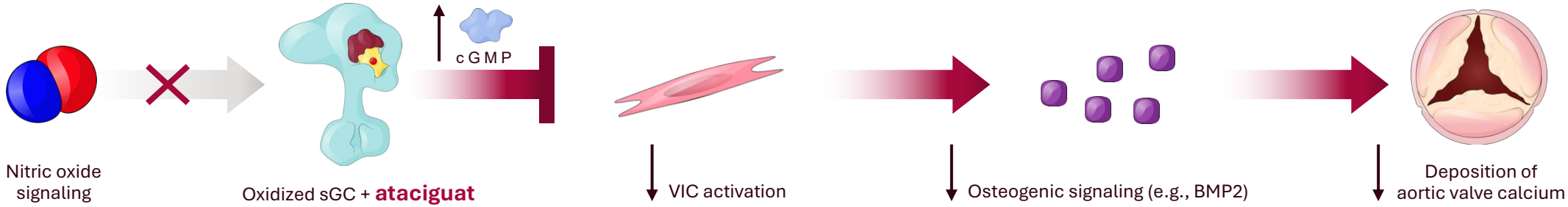
Source: Epidemiology and definitions from Eur Heart Journal | 1 Peeters F, et al. Eur Heart J. 2018;39(28):2618–2624 | 2 Otto CM, et al. JAMA. 2024;332(22):2265–2274 | 3 Genereux P, et al. J Am Coll Cardiol. 2023;82(22):2101–2109 | 4 Strange JE, et. al. Int J Cardiol Heart Vasc. 2022 Nov 30;43:101157 | 5 Swank, et al. Circ Heart Fail. 2012 Jul 6;5(5):579–585. | 6 Tastet L, et al. J Am Heart Assoc. 2024;13(15):e035605.

Ataciguat: restoring sGC function to reduce AVC deposition

Diseased aortic valve[1–8] Oxidized sGC inactivates NO pathway causing calcification



Ataciguat in the diseased aortic valve[1–6] Ataciguat reactivates sGC to restore the NO pathway, reducing calcification



Ataciguat is a unique sGC activator designed to preferentially target valvular oxidized sGC without inducing systemic vascular effects such as hypotension[2,3]

NO = nitric oxide | VIC = valve interstitial cell | BPM2 = bone morphogenetic protein 2| AVC = aortic valve calcium

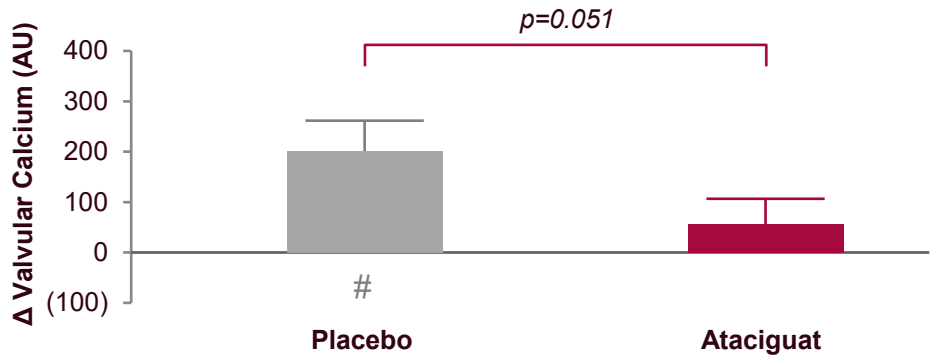
1. Miller JD, et al. J Am Coll Cardiol. 2008;52(10):843–850. 2. Zhou Z, et al. Am J Physiol Heart Circ Physiol. 2008;295(4):H1763-H1771. 3. Zhang B, et al. Circulation. 2025;151(13):913–930. 4. Stasch J-P, et al. Circulation. 2011;123(20):2263–2273. 5. Gould ST, et al. Biomaterials. 2014;35(11):3596–3606. 6. Phua K, et al. Theranostics. 2022;12(11):5189–5203. 7. Fan I, et al. Biomedicine & Pharmacotherapy. 2024;178:117143. 8. Driscoll K, et al. Circ Res. 2021;128(9):1344–1370.

Ataciguat treatment was correlated with reduced progression of calcium and aortic valve area

Phase 2 Trial

Aortic Valve Calcium (AVC)

CHANGE FROM BASELINE TO 6 MONTHS

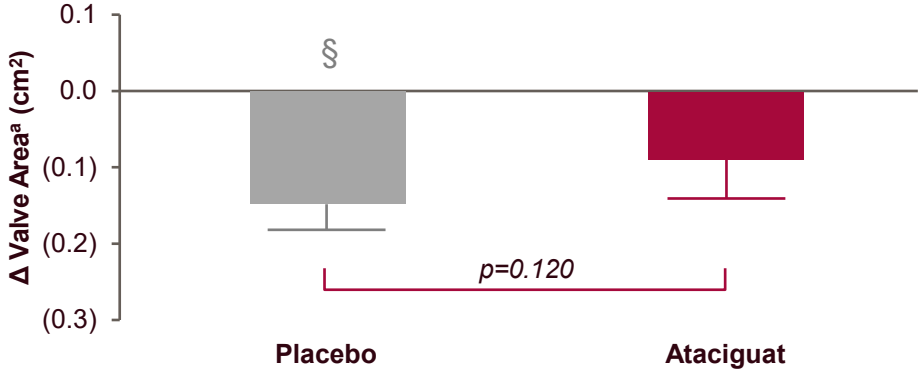


	Placebo	Ataciguat
N	11	12
Baseline values	1208	1055

KEY TAKEAWAYS

- Ataciguat treatment was correlated with slowed progression of AVC by >50%[1]

Aortic Valve Area (AVA)



	Placebo	Ataciguat
N	11	12
Baseline values	1.33	1.36

- Early signs of ATA being correlated with slowing AVA progression

AVC and AVA data is represented as mean and SD

Source: Trial Reference: https://clinicaltrials.gov/study/NCT07001800 | 1 Zhang B, et al. Circulation. 2026;151(13):913–930 [a]By continuity equation. #, change from baseline *P*=0.004; §, change from baseline *P*=0.015

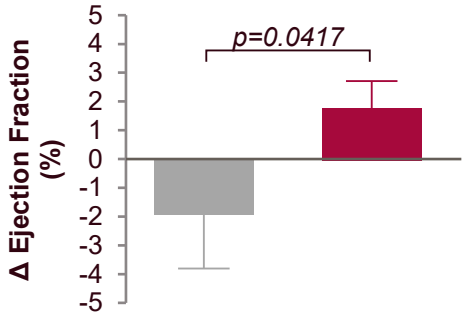
Ataciguat has the potential to markedly improve cardiac structure and function

Phase 2 Trial

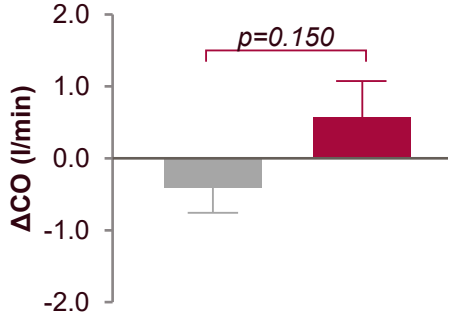
Measures of cardiac function

CHANGE FROM BASELINE TO 6 MONTHS

LVEF[1]



CARDIAC OUTPUT[2]



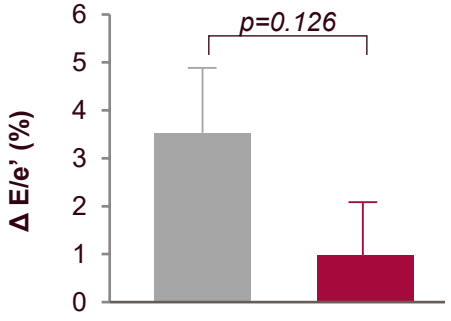
	Placebo	Ataciguat	Placebo	Ataciguat
N	11	12	11	10
Baseline values	64%	65%	6.3	6.3

KEY TAKEAWAYS

- At 6 months, ATA was shown to improve LVEF and CO relative to placebo[1,2]
- Participants with the least increase in AVC generally had the largest increase in CO, and improvements in CO were more frequent in those treated with ataciguat compared with placebo[2]

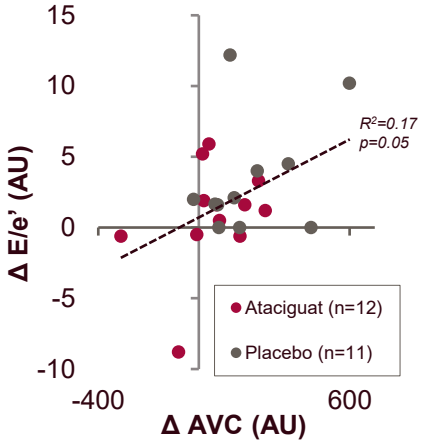
Changes in diastolic dysfunction

E/e'[1]



	Placebo	Ataciguat
N	11	12

E/e' VS AVC[1]



- Ataciguat was observed to slow worsening of diastolic function (E/e'), and increases in AVC correlated with worsening E/e'

Measures of Cardiac Function and Δ E/e' vs E/e' data is represented as mean and SD. E/e' vs AVC data is represented in mean change; AVC = aortic valve calcium | CO = cardiac output | LVEF = left ventricle ejection fraction | AU = arbitrary units | AVC = aortic valve calcium | E/e' = ratio early mitral inflow velocity to mitral annular diastolic velocity (measure of diastolic functoin) | AVACE = aortic valve area by continuity equation | LVMI = left ventricle mass index |

Source: Trial Reference: https://clinicaltrials.gov/study/NCT07001800 | 1 Zhang et al, Circulation. 2026. | 2 Data on file

Ataciguat was generally safe and well tolerated across clinical trials, with AE types and severity similar to placebo and across all dose regimens

Asset	Phase 1b Safety & Tolerability Study in CAVS (13-005387)	Phase 2 Efficacy Study in CAVS (14-006469)
Study Population	mild to moderate AS	moderate AS
Sample Size	N = 36	N = 23
Treatment Arms	ataciguat 100 mg (n=10) ataciguat 200 mg (n=9) matching placebo (n=17)	ataciguat 200 mg (n=12) matching placebo (n=11)
Overall Safety Findings	Ataciguat was found to be safe and well tolerated with no evidence of symptomatic or asymptomatic hypotension or orthostatic intolerance*	
Deaths	None	None
Serious Adverse Events	Two SAEs were reported in the ataciguat arm: Dizziness and Hematuria, both of which were recovered/resolved	One SAE (elevated liver enzymes) was reported in the ataciguat arm, which recovered/resolved

Overall, 996 individuals have been exposed to ataciguat across 22 completed clinical trials. In participants with mild to moderate CAVS, ataciguat was well tolerated with no significant impact to SBP over 6 months of treatment."

CAVS = calcific aortic valve stenosis

*In Phase 1b assessed with both tilt table testing and a controlled transition from a seated to standing position

Kardigan, Inc. (2025) Ataciguat (HMR1766) Investigator's Brochure, Edition 3. Zhang, B., et al. Circulation, 2025. 151(13): p. 913–930

Phase 2b trial in moderate CAVS

KATALYST-AV

Key study inclusion criteria:

- Adults ≥50 years with moderate CAVS
- AVA of ≥1.0 cm to ≤1.50 cm[1]
- AVC 300-1200 AU (women) or 300-2000 AU (men)

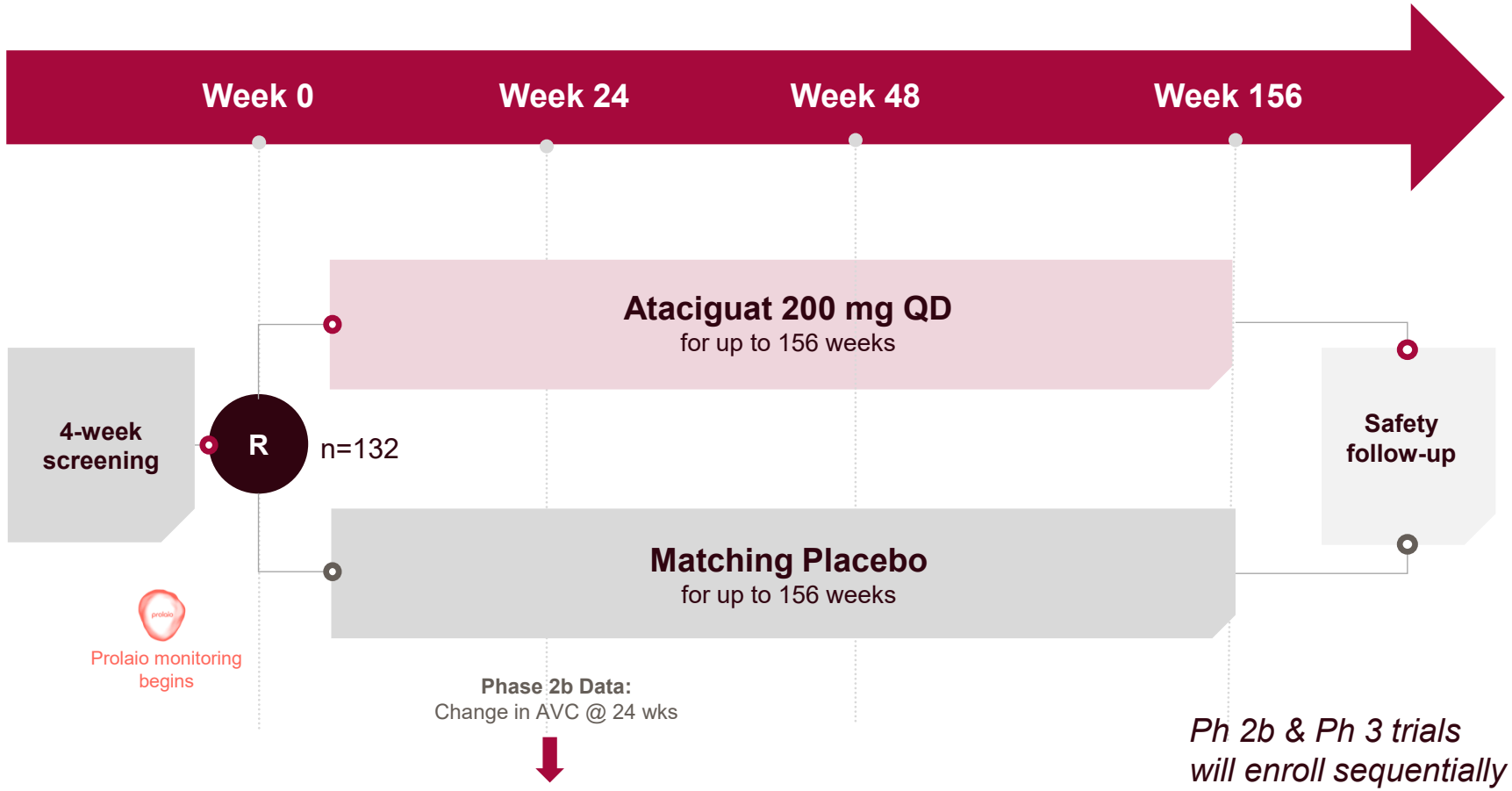
Key endpoints:

Primary Analysis (Week 24):

- Change in AVC

Secondary Analysis (Week 48):

- Correlation between change in AVC and VO_2 (pVO_2 and eVO_2)
- Change in VO_2



Upcoming catalyst: Phase 2b 24-week data readout expected 1H 2027

CAVS = calcific aortic valve stenosis | AVA = Aortic valve area | AVC = Aortic valve calcium | AU = arbitrary units | pVO2 = peak oxygen consumption

Source: Trial Reference: https://clinicaltrials.gov/study/NCT07001800

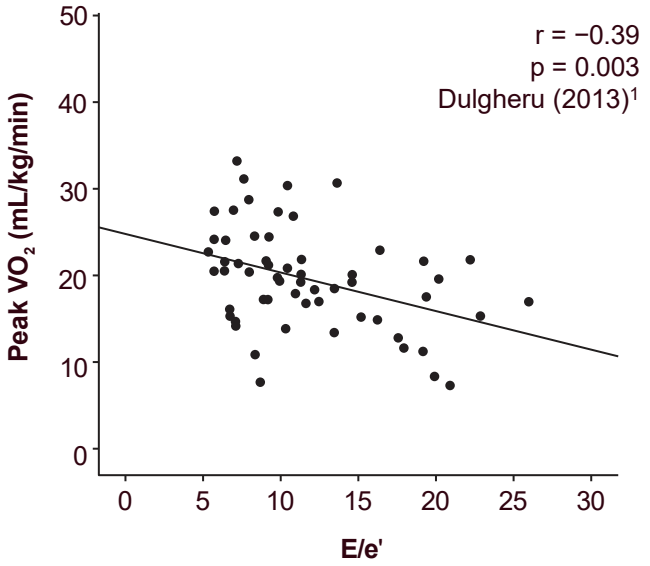
1 Peeters F, et al. Eur Heart J. 2018;39(28):2618–2624

Phase 2b 24-week AVC data derisks further clinical development

What we know now

Cardiopulmonary function correlates to cardiac filling:

pVO_2 correlates with E/e'



Higher E/e' = lower pVO_2.
The relationship is confirmed in CAVS[1]

The mechanistic bridge

Ataciguat (ATA) is designed to slow AVC, which drives the pathway to preserved exercise capacity:

ATA potential to slow AVC

Designed to slow progression of aortic stenosis, as observed in the Phase 2 trial

↓

↓ AVC → ↓ E/e'

Slowing AVC can improve diastolic function, as observed in the Phase 2 trial

24-week ΔAVC confirms whether ATA is correlated with moving the mechanism

What it potentially signals

If 24-week AVC slowing is confirmed, the 48-week topline in the Phase 3 trial is supported:

Change in pVO_2

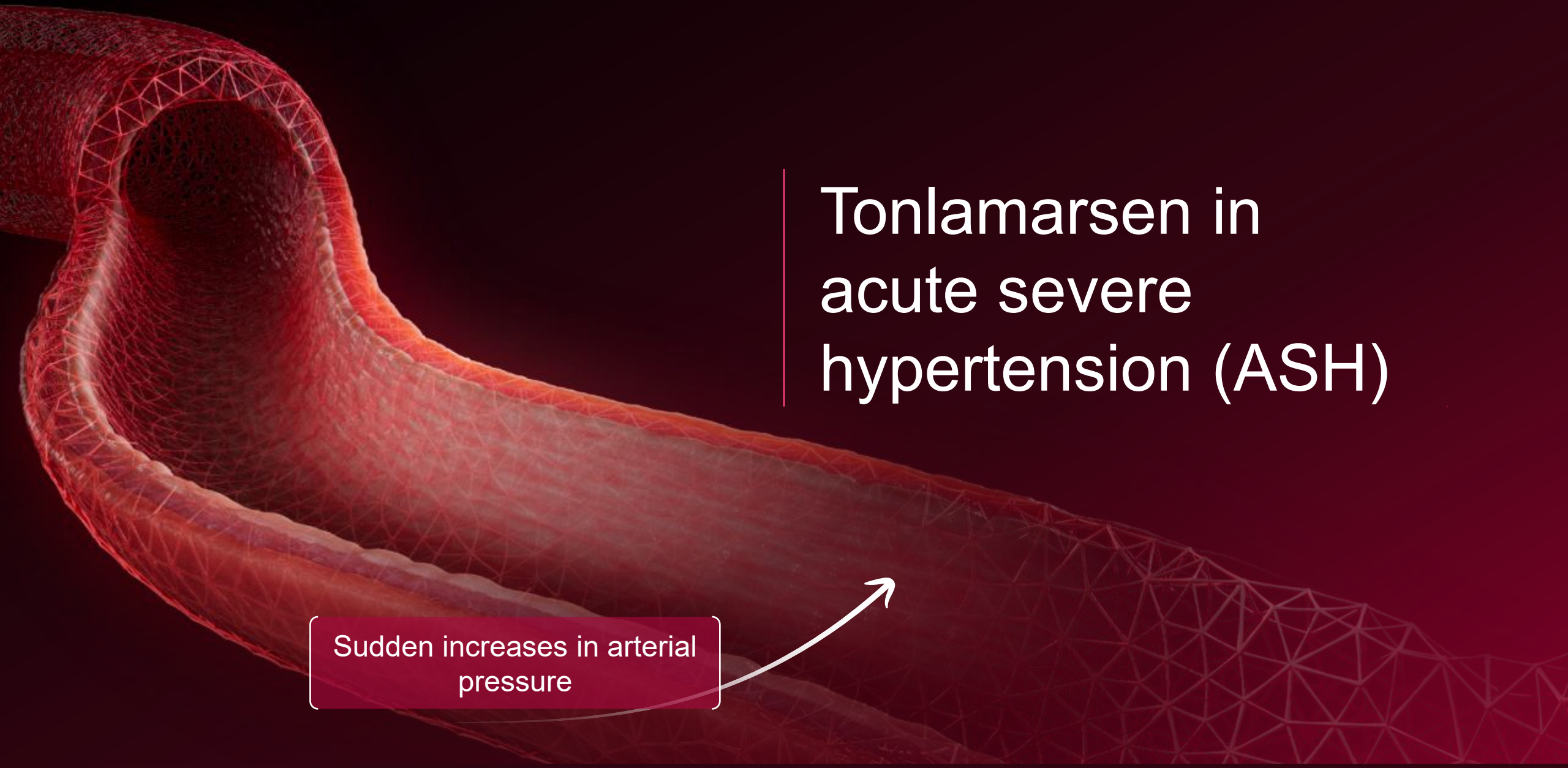
primary registrational endpoint

Mechanism confirmed at 24 weeks → potential for both structural and functional benefit at 48 weeks

AVC = Aortic Valve Calcium | AVA = Aortic Valve Area | E/e' = diastolic filling pressure ratio | pVO_2 = Peak Oxygen Uptake | Phase 2b Part A ΔAVC data expected 1H 2027 |

1. Dulgheru et al 2013.

Tonlamarsen in acute severe hypertension (ASH)



Tonlamarsen: a targeted approach addressing ASH

WELL-DEFINED PATIENT POP

~2M
ASH hospitalizations / year (US)[1]

- No treatments for post-hospital ASH
- Characterized by SBP surges >180 mmHg
- ~35% readmitted within 90 days[2]
- Significant comorbidities: 20% with heart failure & 10-30% with renal disease[3]

TARGETING ROOT CAUSE

AGT modulator
designed to reduce hepatic AGT to break the stress-BP cycle

- Cortisol (stress) spikes lead to AGT increases and SBP surges
- TLA modulates hepatic AGT, lowering BP
- TLA reduced AGT by 67%
- Durable AGT suppression prevents recurrent BP surges

CLINICAL RESULTS

Reduced BP & BP surges
in Phase 2 trial

- TLA reduced BP by 8.9 mmHg in highest-risk patients
- Monthly dosing of TLA was correlated with reduced BP surges of >150 mmHg by 28%
- TLA safety profile in Phase 2 trial supports use in ASH

UPCOMING CATALYST

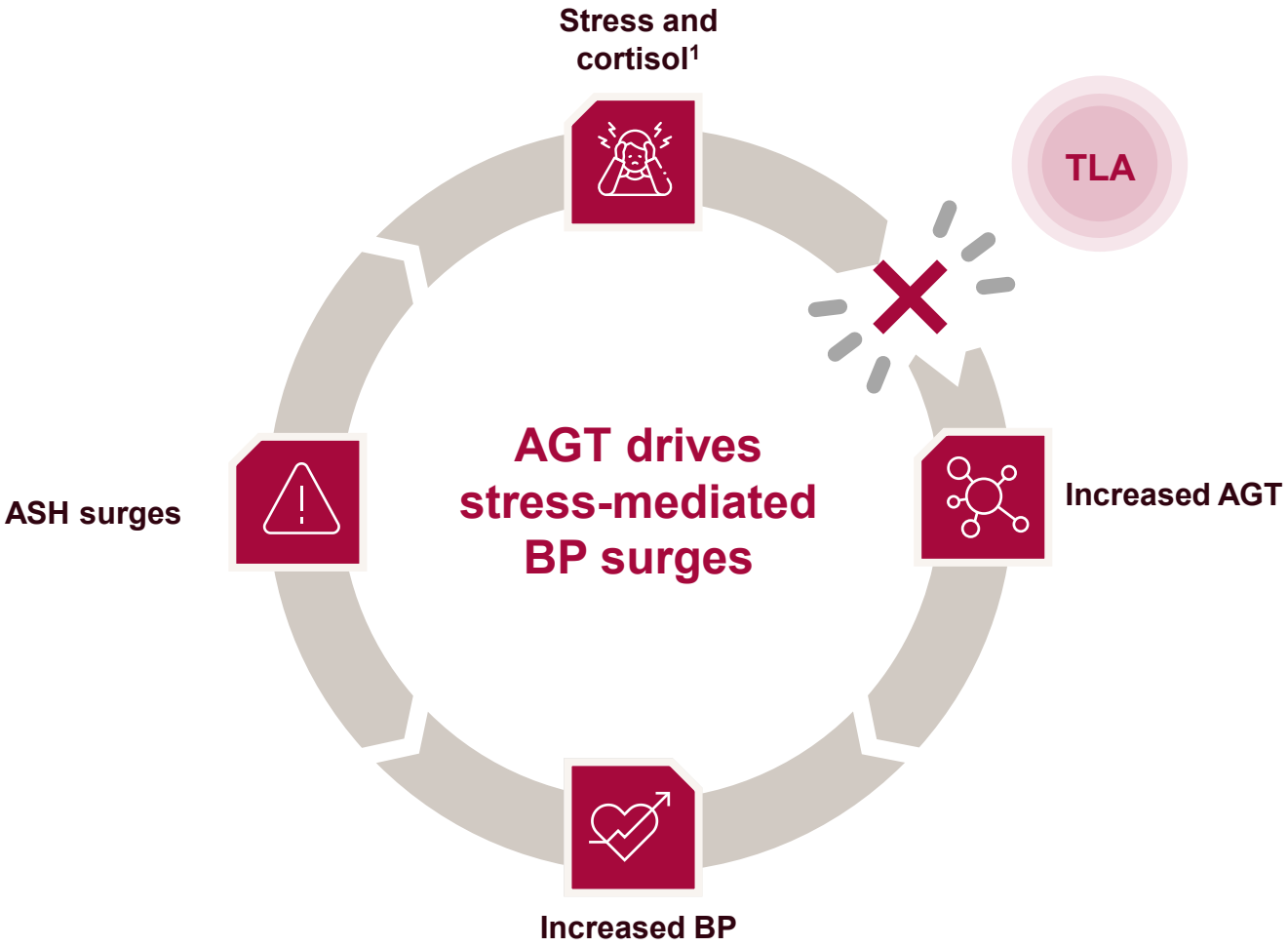
1H 2027
Phase 2b data readout

- Phase 2b study in ~100 hospitalized ASH patients underway
- Evaluating effect of TLA on reductions in BP, BP surges, and hospital readmission rates

ASH = acute severe hypertension | AGT = angiotensinogen | SBP = systolic blood pressure | BP = blood pressure

1. Epidemiology: Scivida market research. N Engl J Med 2019;381:1843-1852 DOI: 10.1056/NEJMcp190111, 2. Gore, et. al. Am Heart J 2010 160:521-527.el; 3. Katz JN et al. Am Heart J 2009; 158:599-606.e1., Siddiqi TN et al. J Am Heart Assoc. 2023;12:e029355.

Stress-driven biology supports angiotensinogen (AGT) as a target in acute severe hypertension (ASH)



AGT expression tightly correlates with BP elevation under stress

Liver-targeted AGT suppression attenuates stress-induced BP increases

AGT modulation normalizes BP without complete pathway shutdown

27% of patients with resistant hypertension have hypercortisolism[2]

AGT = angiotensinogen | BP = blood pressure | TLA = tonlamarsen

1. Geist et al. ACC 2026. 2. DL Bhatt, et al. Prevalence And Clinical Impact Of Hypercortisolism In Individuals With Resistant Hypertension: Primary Results From The Momentum Study (ACC 2026)

Treatment with tonlamarsen was correlated with robust AGT modulation and durable reduction in oSBP out to Week 20

Phase 2 Trial in uHTN

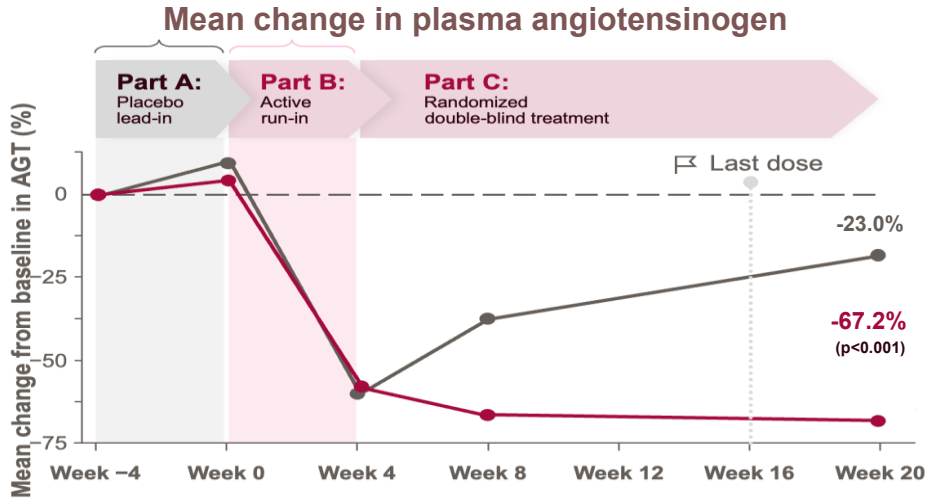
Tonlamarsen (5 doses) *Tonlamarsen (1 dose)*

CHANGE FROM BASELINE

KEY TAKEAWAYS

Measures of target engagement

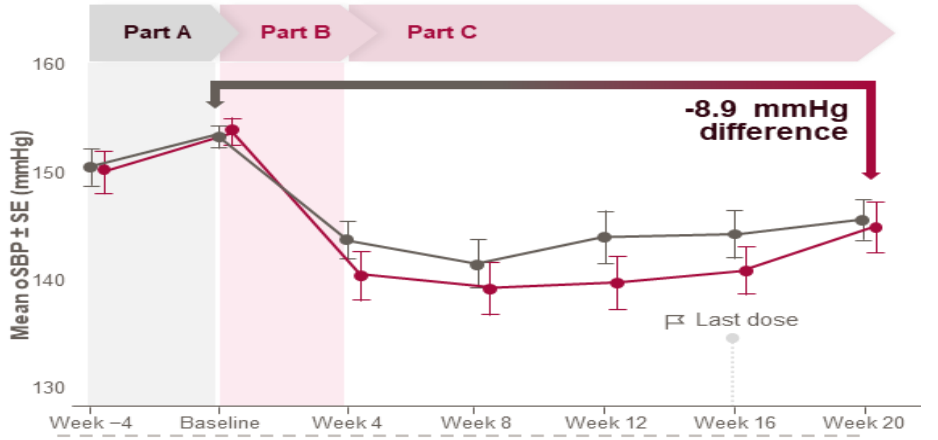
Mean change in plasma angiotensinogen



- AGT reduced by 67% for 5-dose TLA treatment arm versus 23% for single-dose arm (p<0.001) at Week 20.

Changes in SBP of high burden patients

Patients with baseline oSBP > median



- Patients with the highest SBP at baseline (>150mmHg oSBP) had a -8.9 mmHg reduction in oSBP at Week 20 in both arms.

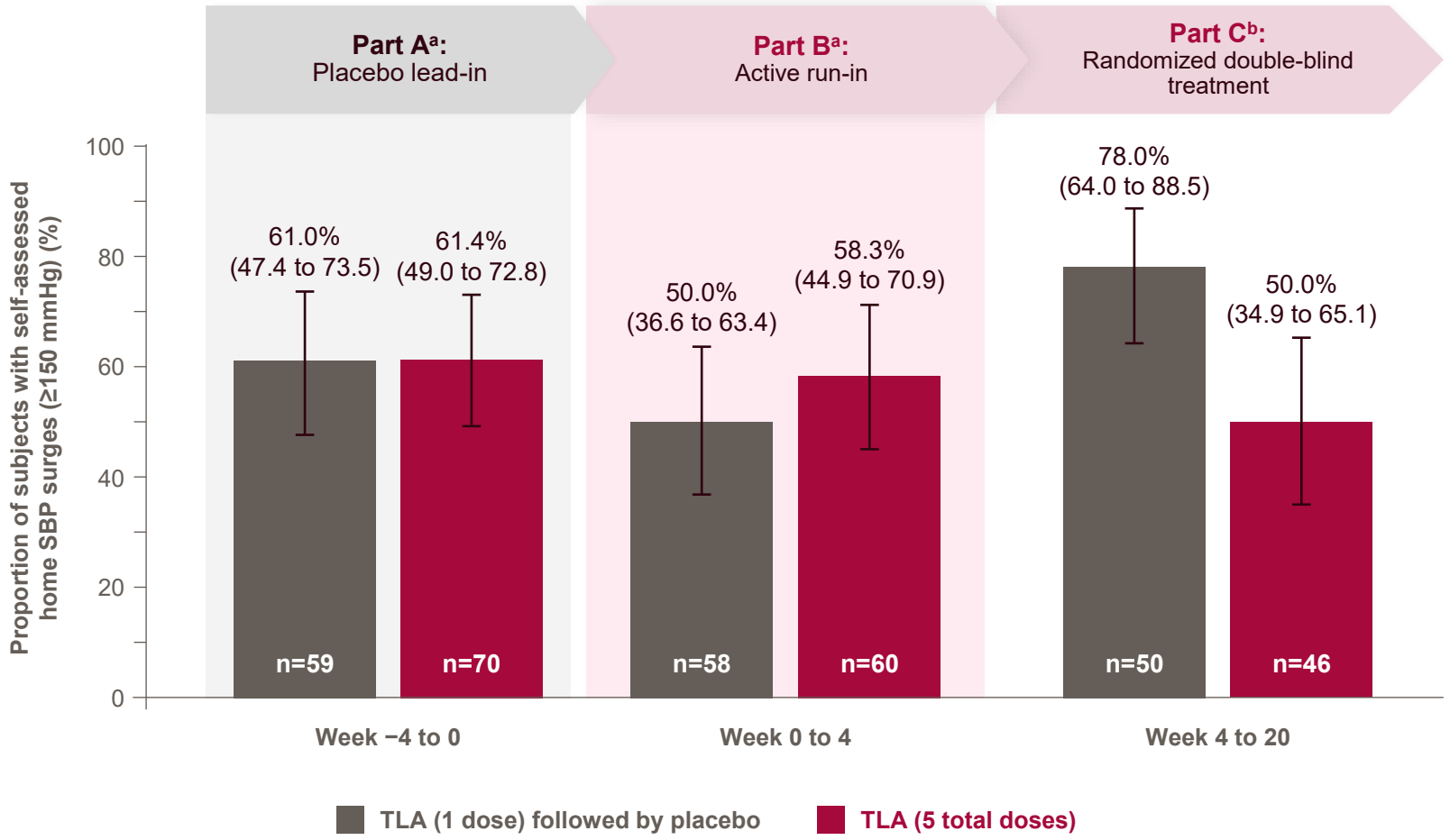
uHTN = uncontrolled hypertension | hSBP = home systolic blood pressure | oSBP = office systolic blood pressure | AGT = angiotensinogen | SBP = systolic blood pressure | BP = blood pressure

1. Geist et al. ACC 2026. | a Minimum 19 daily averages for Parts A/B. b Minimum 75 daily averages for Part C. Error bars represent 95% Clopper-Pearson exact confidence intervals.

hSBP surges (≥150 mmHg) were reduced and sustained in 5-dose arm

- Acute severe hypertension (ASH) is characterized by BP surges that drive recurrent hospitalizations and risk of end-organ damage
- **Continued tonlamarsen (TLA) dosing (5 doses) resulted in greater and more sustained reductions in hSBP surges of ≥150 mmHg during Part C, supporting durability**
- Repeat monthly doses of TLA reduced BP surges of ≥150 mmHg by 28% during Part C

Self-assessed (Prolaio) home SBP surges (≥150 mmHg)



hSBP = home systolic blood pressure

[a]Minimum 19 daily averages for Parts A/B. [b]Minimum 75 daily averages for Part C. Error bars represent 95% Clopper-Pearson exact confidence intervals.

Tonlamarsen was well tolerated: no treatment-related hyperkalemia or hypotension

Tonlamarsen safety profile supports use in high-risk populations

	Tonlamarsen (1 Dose)	Tonlamarsen (5 Total Doses)
Any serious AE	2%	5%
Hypotension[a]	0%	1%
Serum potassium >5.5 mmol/L	2%	0%
Decrease of >30% in eGFR	3%	3%
Injection site reactions	3% (all) 1% (≥ Grade 2)	19% (all) 2% (≥ Grade 2)

Common AEs with current and in-development HTN agents

AE = adverse event | eGFR = estimated glomerular filtration rate | HTN = hypertension

[a]Hypotension was not treatment-related per investigator and there was no treatment interruption

Phase 2b trial in acute severe hypertension (ASH)

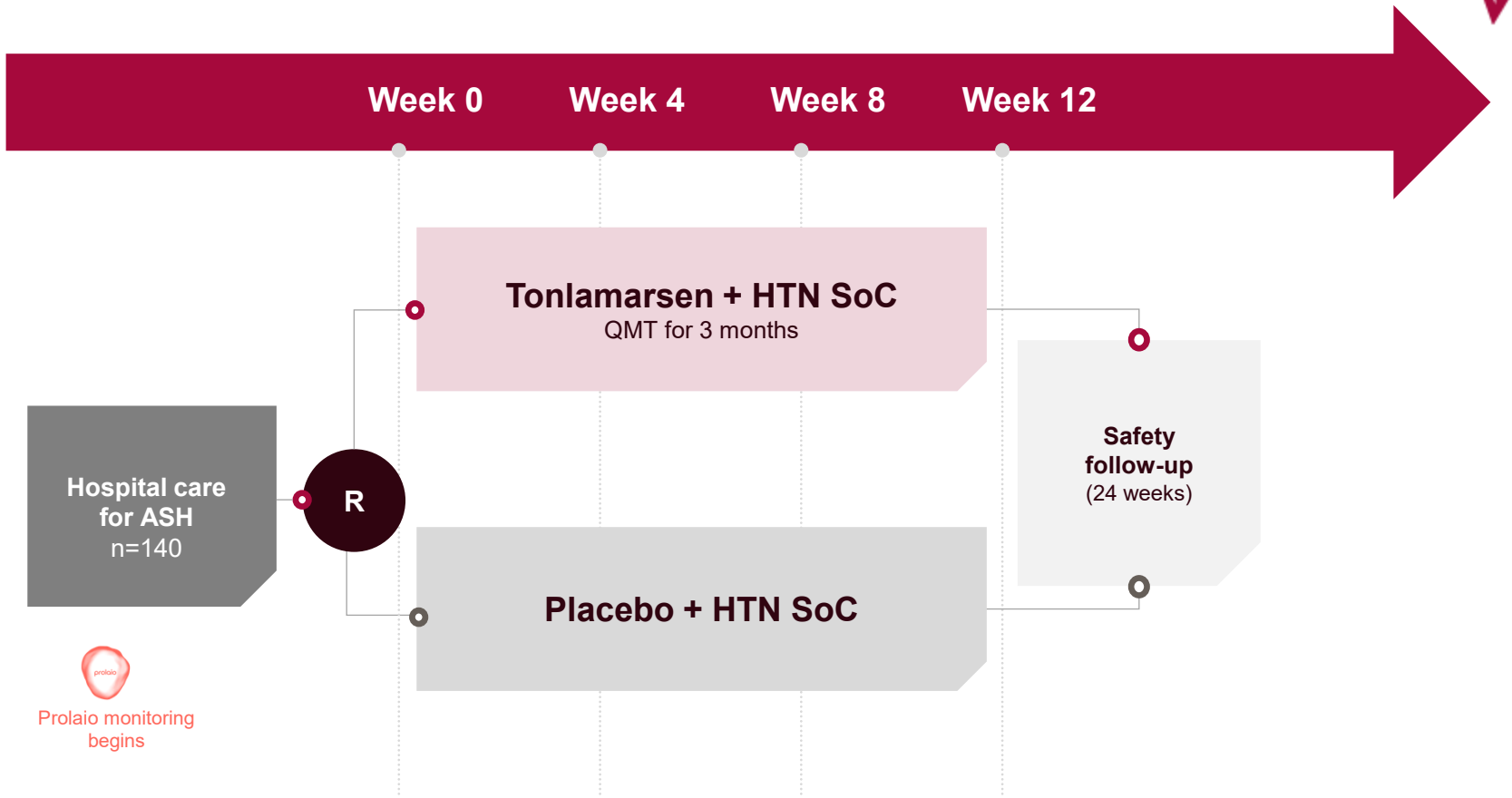
KARDINAL ASH

Key study inclusion criteria:

- ASH patients hospitalized with any SBP ≥180 mmHg and/or DBP ≥110 mmHg
- Enrollment within 7 days post-discharge with SBP ≥145 mmHg

Key endpoints:

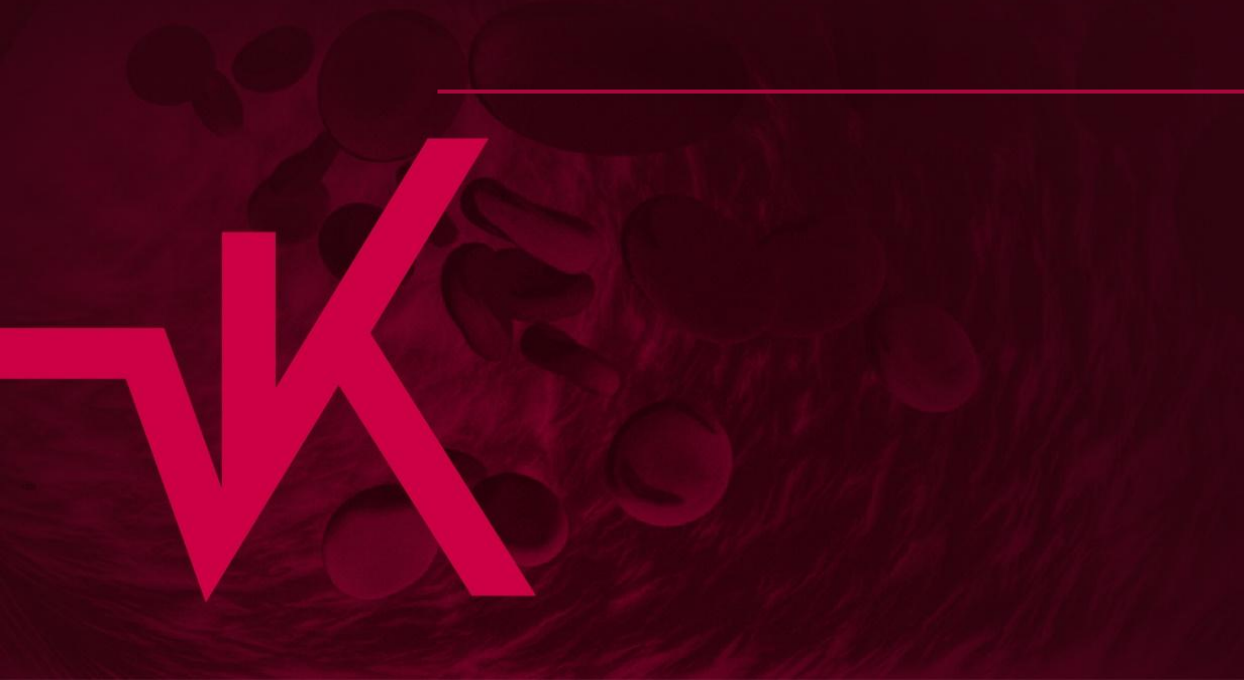
- Measures of BP (oSBP, hSBP), BP surges
- Measures of safety, rescue therapy, readmissions, etc.



Upcoming Catalyst: Phase 2b data readout expected 1H 2027

BP = blood pressure | DBP = diastolic blood pressure | hSBP = home systolic blood pressure | HTN = hypertension | oSBP = office systolic blood pressure | QMT = once monthly treatment | SBP = systolic blood pressure | SoC = standard of care

Harnessing technology



Reimagining clinical trials with Prolaio

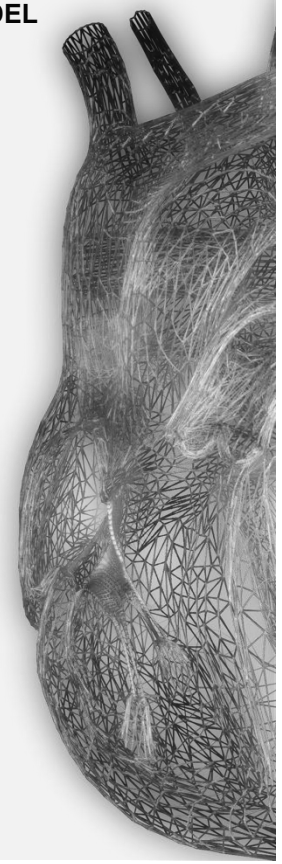
TRADITIONAL CVD CLINICAL TRIAL MODEL

Episodic in-clinic data collection with limited visibility into treatment effects

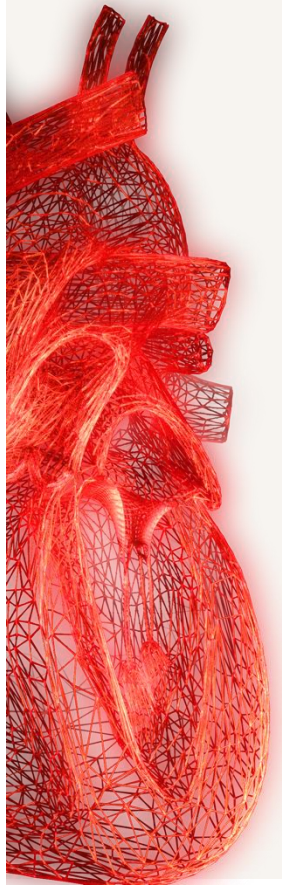
Intermittent, often sparse data points reduce signal and impair assessment

White coat effect can impair accurate assessment of treatment effect

Patient burden of retrospective recall can reduce compliance and engagement



prolaio™



Longitudinal data collection providing enhanced visibility into patient physiology

Real-world data at scale rather than singular snapshots within a clinical setting

Near real-time visibility allows insight into treatment response and safety

Reduced patient burden eliminates unnecessary travel of retrospective recall

Prolaio is our proprietary data and analytics platform, which includes Class I and Class II FDA-regulated devices, including 510(k)-cleared algorithms. Enabling continuous, physiological data collection across all clinical trials.

Note: subject to regulatory approval

Harnessing proprietary, transformative technology with Prolaio

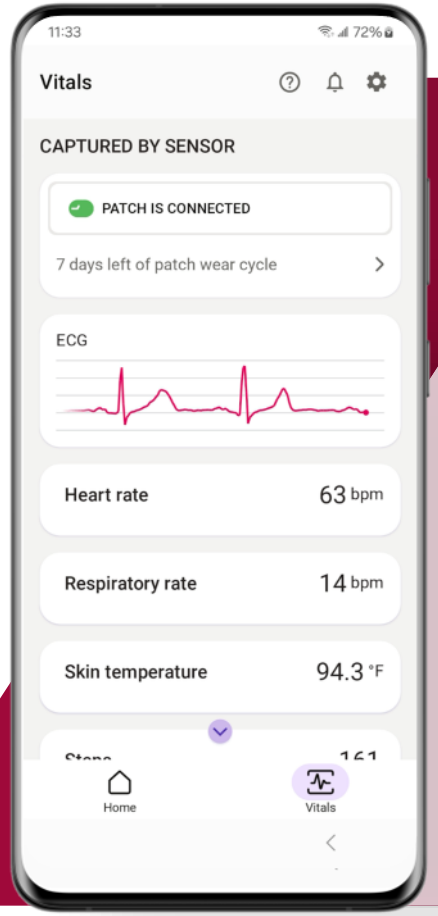
Prolaio brings together real-world data from multiple inputs—like at-home wearables, paired with EHR data—into a continuous stream

prolaio

4M+
hours of
real-time patient data

20
Patents issued

7
FDA-cleared
algorithms



Benefits today:

- ✓ Longitudinal data collection
- ✓ Real-world data at scale
- ✓ Near real-time visibility
- ✓ Rapid, personalized signal detection for faster decisions

Future benefits in development:

- ✓ Digital clinical endpoints
- ✓ Decentralized trials
- ✓ Increased statistical power for smaller clinical trials
- ✓ Increased probability of success

eVO2 = estimated VO2 | EHR = electronic health record

Note: subject to regulatory approval

What’s next

Multiple high-value clinical milestones expected across all three late-stage programs in next 12-18 months

Program	U.S. Patients	Trial	Upcoming Catalysts	
DANICAMTIV	~150-200K PATIENTS[1]	kinship DCM	Phase 2b: 6-month topline data	1H 2027
ATACIGUAT	~1M PATIENTS[2]	KATALYST-AV	Phase 2b: 24-week interim data	1H 2027
			Phase 2b: 48-week topline data	2H 2027
TONLAMARSEN	2M+ PATIENTS[3]	KARDINAL ASH	Phase 2b: 12-week topline data	1H 2027

Upsized IPO generated $460 million in gross proceeds; in addition to $287 million on hand as of March 31, 2026, total cash, cash equivalents, and investments expected to fund operations into 2028

1 Lakdawala et al, Circ Arrhythm Electrophysiol. 2013;6:228-237 | 2 Eur Heart Journal | 3 Epidemiology: Scivida market research. N Engl J Med 2019;381:1843-1852 DOI: 10.1056/NEJMcp190111

Thank you