Corporate Presentation

September 2026

Forward-looking statement

This presentation contains forward-looking statements within the meaning of Section 27A of the Securities Act and Section 21E of the Securities Exchange Act of 1934, as amended, that are based on our management's beliefs and assumptions and on information currently available to our management. Forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified. All statements other than statements of historical facts contained in this presentation, including statements regarding business strategy, plans and 2026 milestones; the clinical, therapeutic and market potential of and expectations regarding our product candidates, platforms and proprietary capabilities; clinical development plans for TN-201, TN-401 and TN-301, preclinical efforts and timelines; clinical outcomes, which may materially change as patient enrollment continues or more patient data become available; availability and content of data from MyPEAK™-1 and RIDGE™-1; and plans to pursue regulatory alignment on pivotal trial plans for TN-201 and TN-401, as well as statements regarding industry trends, are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as "purpose," "focus," "plan," "potential," "may," "future," "anticipated," "objective," "expected," or the negative of these terms or other similar expressions. We have based these forward-looking statements largely on our current expectations and projections about future events and trends that we believe may affect our financial condition, results of operations, business strategy and financial needs.

These forward-looking statements are subject to time, risks, uncertainties and assumptions described in our filings with the SEC, including, but not limited to the section titled "Risk Factors" in our Form 10Q for the quarter ended June 30, 2026, and other documents we have filed, or will file with the SEC. These filings, once filed, are or will be available on the SEC website at www.sec.gov. Such risks include, among other things: the availability of data at the referenced times; the timing of the initiation, progress, completion and potential results of our clinical trials and preclinical studies; our ability to advance product candidates into, and successfully complete, clinical trials and preclinical studies; the potential for clinical trials of our product candidates to differ from preclinical, preliminary, interim or expected results; the timing or likelihood of regulatory filings and approvals; the potential for the FDA or other regulatory agencies to conclude at any time that our product candidates may not have an appropriate risk/benefit profile; our estimates of the number of patients who suffer from the diseases we are targeting and the number of patients that may enroll in our clinical trials; our ability to successfully manufacture and supply our product candidates for preclinical studies, clinical trials and for commercial use, if approved; our ability to commercialize our product candidates, if approved; future strategic arrangements and/or collaborations and the potential benefits of such arrangements and/or collaborations; our estimates regarding expenses, capital requirements and needs for financing, and our ability to obtain capital; our ability to retain the continued service of our key personnel and to identify, hire and retain additional qualified professionals; our ability to obtain and maintain intellectual property protection for our platforms, programs and product candidates; our ability to contract with third-party suppliers and manufacturers and their ability to perform adequately; the pricing, coverage and reimbursement of our product candidates, if approved; and developments relating to our competitors and our industry, including competing product candidates and therapies; general economic and market conditions; and other risks. These risks are not exhaustive. New risk factors emerge from time to time, and it is not possible for our management to predict all risk factors, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in, or implied by, any forward-looking statements. You should not rely upon forward-looking statements as predictions of future events. Although we believe that the expectations reflected in the forward-looking statements are reasonable, we cannot guarantee future results, levels of activity, performance or achievements. In light of the significant uncertainties in these forward-looking statements, you should not regard these statements as a representation or warranty by us or any other person that we will achieve our objectives and plans in any specified time frame or at all. The forward-looking statements made in this presentation relate only to events as of the date on which such statements are made. Except as required by law, we undertake no obligation to update publicly any forward-looking statements for any reason after the date of this presentation. This presentation also contains estimates and other statistical data made by independent parties and by us relating to market size and growth and other data about our industry. This data involves a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. In addition, projections, assumptions, and estimates of our future performance and the future performance of the markets in which we operate are necessarily subject to a high degree of uncertainty and risk.

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TENAYA™
CELEBRATING 10 YEARS

Leading the Precision Medicine Revolution in Cardiovascular Disease

- Founded to transform the treatment of heart disease with therapeutics that target the genetic drivers of disease
- **100% focused on heart disease;** pursuing severe orphan/genetic as well as prevalent conditions
- Decade long leadership has led to multiple novel therapeutic candidates and a modality agnostic drug discovery engine that “follows the science”

In 2026, vision becomes reality

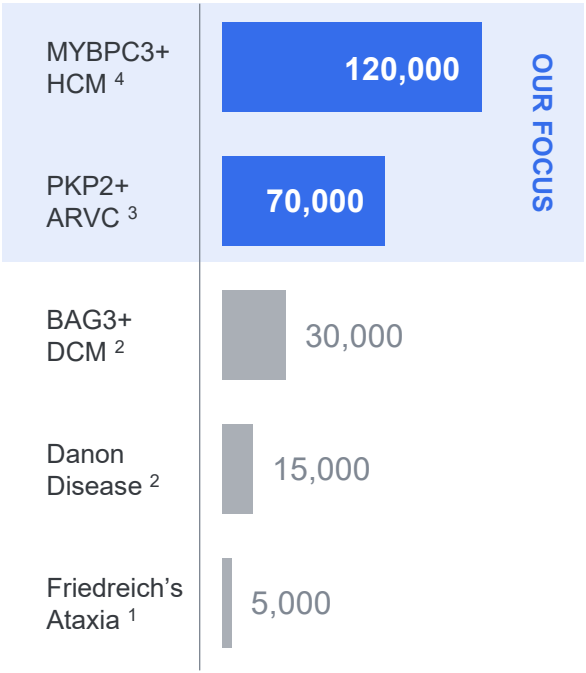
- Proof-of-concept data has the potential to establish registrational pathway for multiple gene therapy programs
- Looking to advance small molecule “pipeline in a target” in rare and prevalent cardiac and cardiac adjacent indications
- Pursuing industry partnerships to expand reach and impact of programs and capabilities

IMMEDIATE OPPORTUNITY:

Advancing TN-201 and TN-401 towards pivotal studies

Large market potential in rare disease setting

Addressable populations in the U.S.



Early evidence of clinically meaningful benefit

TN-201 improved multiple measures of disease (biomarkers, hypertrophy, symptom relief, function)

TN-401 achieved meaningful reductions in measures of electrical instability

Multiple de-risking catalysts in 2026

- Biopsy evidence of transduction, translation and expression
- Decrease in key markers of disease

TN-201

- Enrollment complete
- One-year Cohort 2 data and Cohort 1 durability at two years (Q4)

TN-401

- Enrollment complete
- One-year data for Cohort 2 and durability data for Cohort 1 (Q4)

Regulatory progress across both programs

TN-201: ODD, Fast Track, RPDD, RDEP, PRIME

TN-401: RMAT, ODD, Fast Track, PRIME

Engaging with regulators on pivotal paths; Updates planned for Q4'26

1. Lexeo presentation, Oct 2025
2. Rocket Pharma presentation, Sep 2025
3. Sedaghat-Hemedani, et al., *Clin Res Cardiol*. 2018
4. Groeneweg, et al, *Circ Cardiovasc Gen 2015* & McKenna, et al, *Nature Rev Cardio* 2021

RMAT = Regenerative Medicine Advanced therapy
ODD = Orphan drug designation (FDA)
RPDD = Rare pediatric drug designation (FDA)
RDEP = Rare disease evidence principles (FDA)
PRIME: PRIority Medicines designation (EMA)

NEXT OPPORTUNITY:

Advancing TN-301 towards clinical proof-of-activity to unlock “pipeline-in-a-pill” potential

Broad utility demonstrated by Tenaya and others

• HFpEF
• (Genetic) DCM

• PH-HFpEF
• PAH

DMD / DMD-CM
(+ other MDs)

Mechanism of HDAC6 inhibition well understood

DECREASES
- Inflammation
- Oxidative stress
- Fibrosis
- Metabolic dysregulation

INCREASES
- Autophagy
- Lipid metabolism
- Protein quality control
- Mitochondrial metabolism

Safety established as foundation for future investigation

Phase 1 trial in healthy volunteers complete

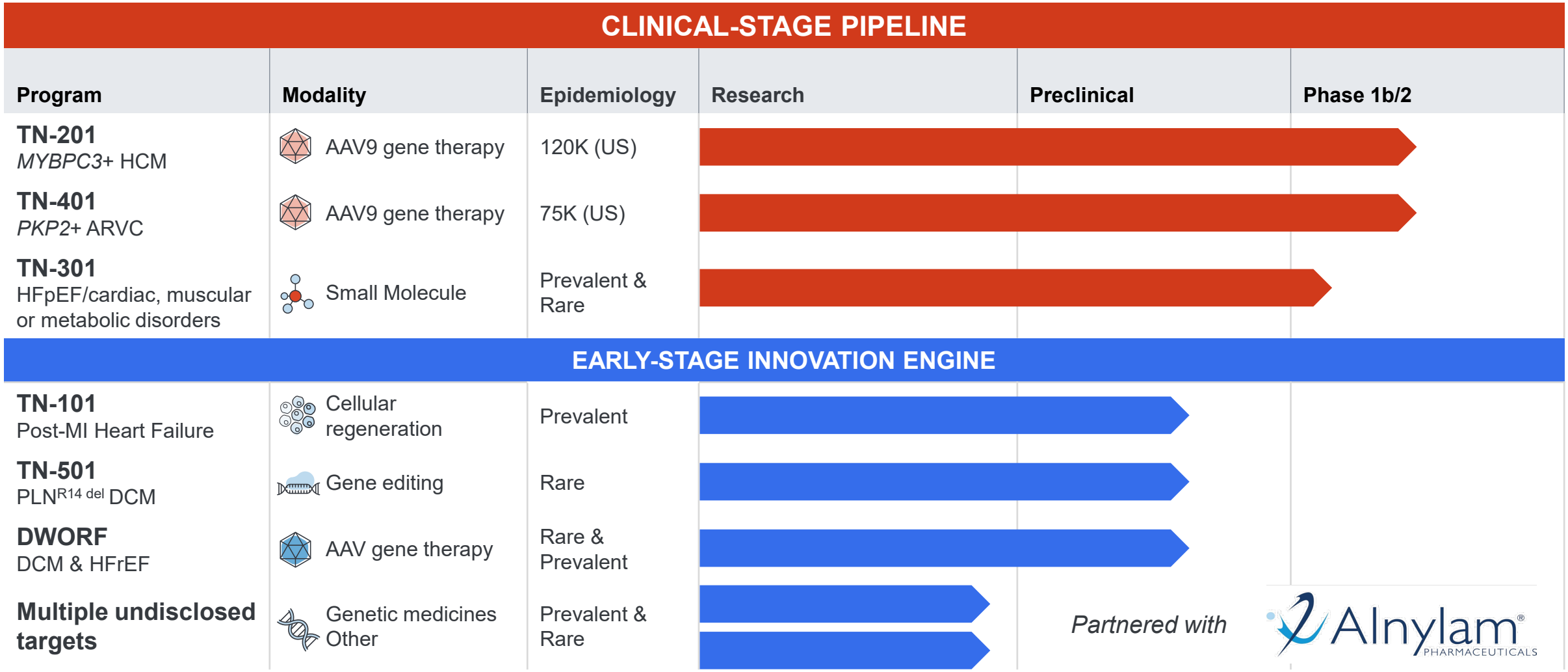
- AEs were mild, similar in profile to placebo across wide range of doses
- Predictable PK supports once-daily dosing

Advancing toward Phase 2 trial in 2H’27 to establish proof-of-activity

- Enabling work being conducted within current cash guidance

HDAC6 = Histone deacetylase 6
HFpEF = heart failure with preserved ejection fraction
DCM = Dilated cardiomyopathy
PAH = Pulmonary Arterial Hypertension
PH = Pulmonary Hypertension
DMD = Duchenne muscular dystrophy &
DMD-CM = DMD cardiomyopathy
MDs = Muscular dystrophies
AEs = adverse events
PK = Pharmacokinetics

Diverse clinical stage programs supported by innovation engine



MYBPC3 = Myosin binding protein C3 (gene)
HCM = hypertrophic cardiomyopathy
AAV9 = adeno-associated virus, serotype 9

PKP2 = Plakophilin 2 (gene)
ARVC = arrhythmogenic right ventricular cardiomyopathy
MI = myocardial infarction

PLN = phosolambam. R14del refers to the specific mutation
DCM = dilated cardiomyopathy
HFrEF = heart failure with reduced ejection fraction

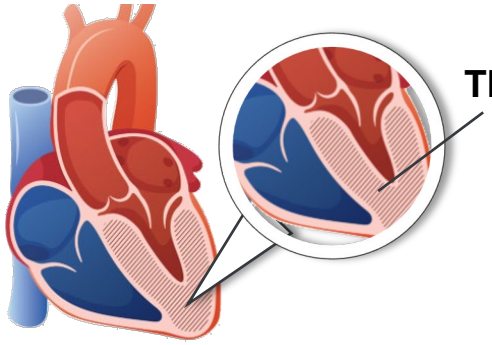
TENAYA™ THERAPEUTICS

TN-201 for *MYBPC3*-associated HCM

MYBPC3-associated HCM is estimated to affect 120,000 people in the U.S. alone[1]

A severe and progressive autosomal dominant condition affecting adults, teens, children and infants

HCM HEART



- Significant functional impairment
- Social and psychological impacts
- Symptoms include shortness of breath, fainting, chest pain, fatigue, palpitations, arrhythmias
- Elevated risk of sudden cardiac death and heart failure

GABE | AGE 10
Living with *MYBPC3*+ HCM

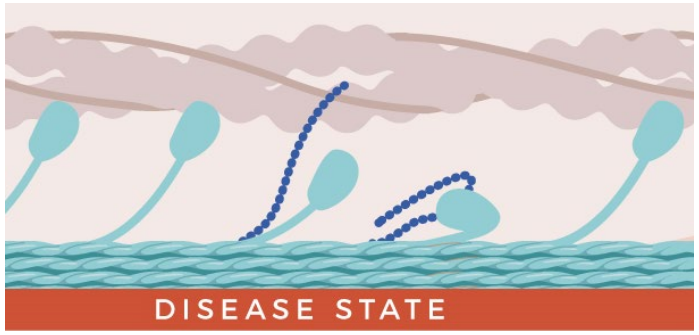
~57% of identified genetic variants underlying familial HCM are *MYBPC3* mutations[2]

>30% of genetic variants underlying childhood-onset HCM are *MYBPC3* mutations[3]

1. Sedaghat-Hemedani, et al., *Clin Res Cardiol* 2018
2. Ho, et al, *Circulation* 2018
3. Marston, et al, *Eur Heart Jrnl* 2021

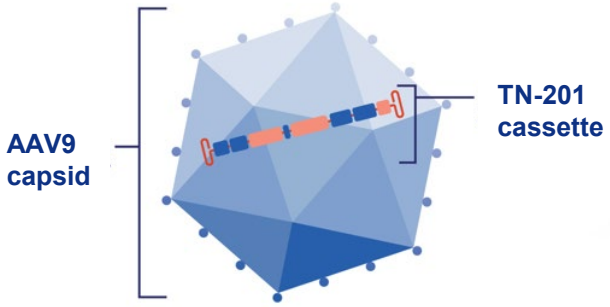
TN-201 is the first gene therapy being developed for MYBPC3-associated HCM[1]

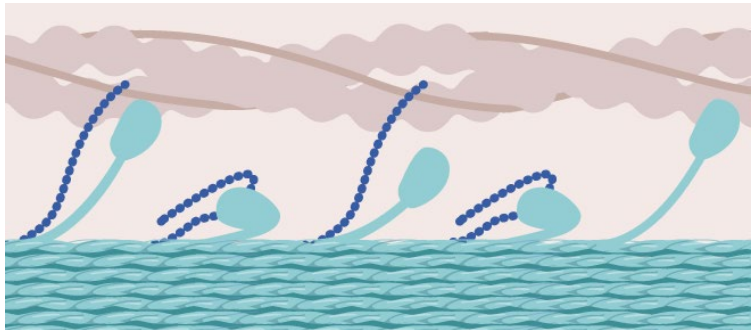
MYBPC3 Pathophysiology



Heterozygous mutations in the *MYBPC3* gene lead to significantly **lower levels of MyBP-C protein** and dysregulated cardiac contractility[1]

TN-201 Mechanism of Action





TN-201 delivers a full-length, functional copy of the *MYBPC3* gene to cardiomyocytes using the most widely dosed capsid, AAV9[2]

Expression of the *MYBPC3* gene increases MyBP-C protein levels and is expected to restore sarcomeric function, reduce hypertrophy, and halt disease progression[3]

MyBP-C protein present in sarcomere

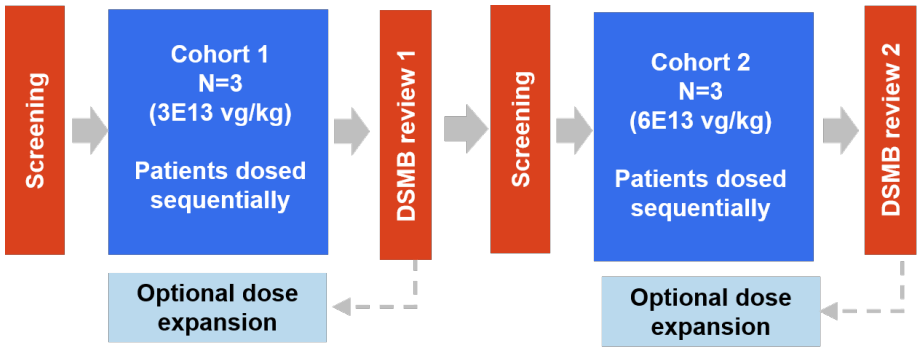
MyBP-C = Myosin-binding protein C

1. O'Leary, et al., *J Mol Cell Cardiol* 2019
2. Byrne, et al., *Mol Ther* 2025
3. Greer-Short, et al., *Nat Commun* 2025

MyPEAK-1 at-a-glance: Seeking directional consistency across multiple parameters

MyPEAK-1 Phase 1b/2 clinical trial

Designed to assess safety, tolerability and dose



MyPEAK-1 patients have greater symptom burden at baseline than average HCM patients

- All patients had severe hypertrophy
- All were symptomatic
- Majority previously underwent myectomy
- 7 patients dosed as of May data cut
- 6 evaluable for molecular and clinical assessments; 3 from each dose cohort

Positive interim results[1] show evidence of clinical benefit

Multiple parameters of disease improving across all six evaluable patients

- Hypertrophy decreased from baseline suggesting cardiac remodeling
- Biomarkers of cardiac injury and strain decreased or remained stable

- All evaluable patients saw measurable improvements in symptom burden
- Higher dose cohort showed early signs of functional improvements

- TN-201 gene therapy is reaching heart cells and being expressed

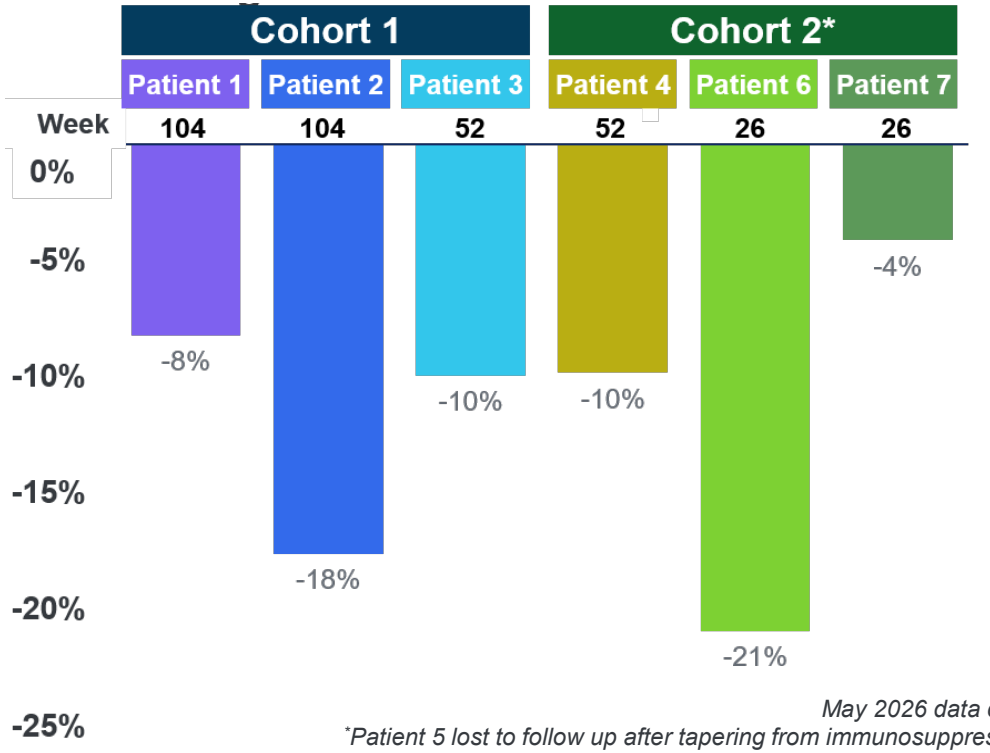
- TN-201 has been well tolerated at both doses
- No cardiotoxicities

1. May 2026 data cut reported June 2026

Decreases in hypertrophy suggest cardiac remodeling and compare favorably with CMIs

All MyPEAK-1 patients achieved reductions in LVMI

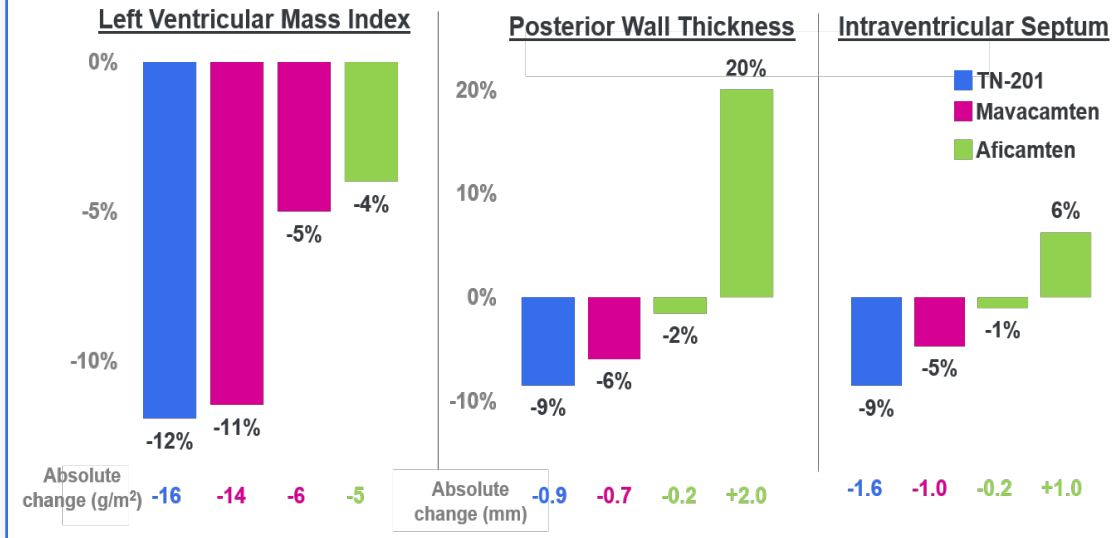
Left ventricular mass index
Change from baseline to most recent visit (g/m²)



May 2026 data cut off
**Patient 5 lost to follow up after tapering from immunosuppressives*

TN-201 decreases across multiple measures of hypertrophy vs. CMIs*

Relative change from baseline to most recent visit *[1-5]



These trials are not head-to-head and caution should be used in drawing any conclusions from these comparisons. Comparisons with peer programs are not intended to indicate likelihood of TN-201 clinical benefit.

1. Desai, et al., *Circ* 2025
2. Hegde, et al., *JACC* 2021
3. Desai, et al., *JACC* 2025
4. Hegde, et al., *JACC* 2024
5. Masri, et al, Circulation 2026

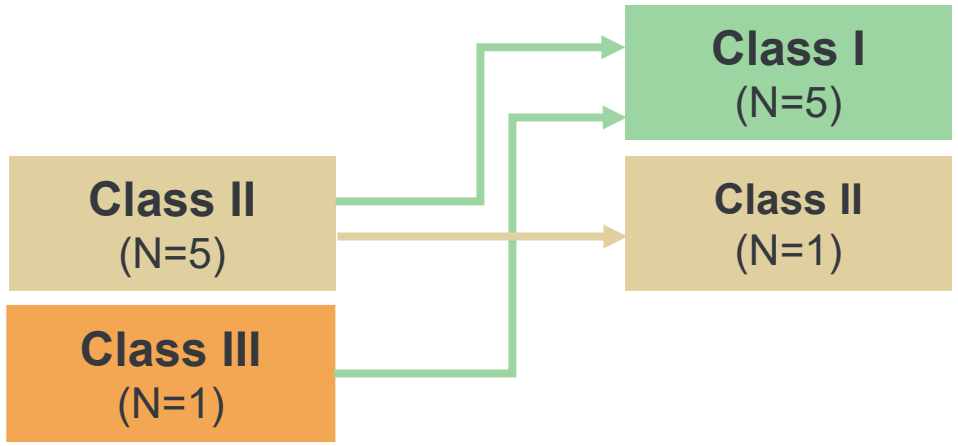
CMI = cardiac myosin inhibitors
LVMI = left ventricular mass index

*Mavacamten readouts: oHCM (VALOR) at 128 weeks; nHCM (MAVERICK) at 48 weeks
*Aficamten readouts: oHCM (SEQUOIA) at 24 weeks; nHCM (ACACIA) at 36 weeks
LVMI was not reported from ACACIA. Posterior and septal wall thickness were not reported from MAVERICK

Symptom burden reduced in all patients by at least one measure following TN-201 treatment

83% of MyPEAK-1 patients achieved NYHA class I (asymptomatic) status

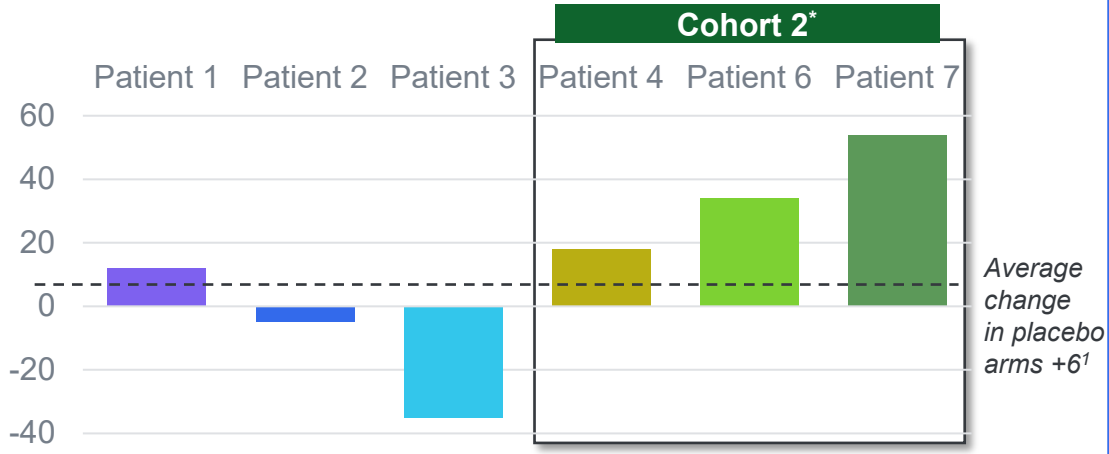
New York Heart Association Classification change from baseline to most recent visit



- Changes in NYHA class coincided with improvements in other parameters

67% achieved meaningful improvements in KCCQ Clinical Summary Score[2]

KCCQ-CSS change from baseline to MRV



- Cohort 2 patients' scores improved by an average of +36 points and are now in the "good to excellent" range (75-100 points)

May 2026 data cut off
**Patient 5 lost to follow up after tapering from immunosuppressives*

NYHA = New York Heart Association
KCCQ = Kansas City Cardiomyopathy Questionnaire
MRV = most recent visit

1. Averaged from mavacamten studies (MAVERICK, VALOR, EXPLORER, ODYSSEY) and aficamten studies (ACACIA, SEQUOIA) published data)
2, Spertus, et al, JACC State-of-the-art review 2020

Functional improvements observed for a majority of Cohort 2 patients

Two of three of Cohort 2 patients achieved improvements in at least one of two validated measurements of exercise capacity

Six-minute walk test from baseline to MRV
Measured in meters

	Baseline	MRV	Difference
Patient 4	500	755	+255
Patient 6	420	390	-30
Patient 7	247	297	+50

Change in peakVO2 from baseline to MRV
Measured as mL/kg/min

	Baseline	MRV	Difference
Patient 4	16.0	18.4	+2.4 (15%)
Patient 6	13.3	Not yet evaluated	
Patient 7	13.8		

- All improvements exceeded the minimal clinically important difference for both assessments[1,2]
- Cohort 1 performance may be confounded by preexisting functional limitations and/or relatively longer duration and higher dose of immunosuppression

May 2026 data cut off

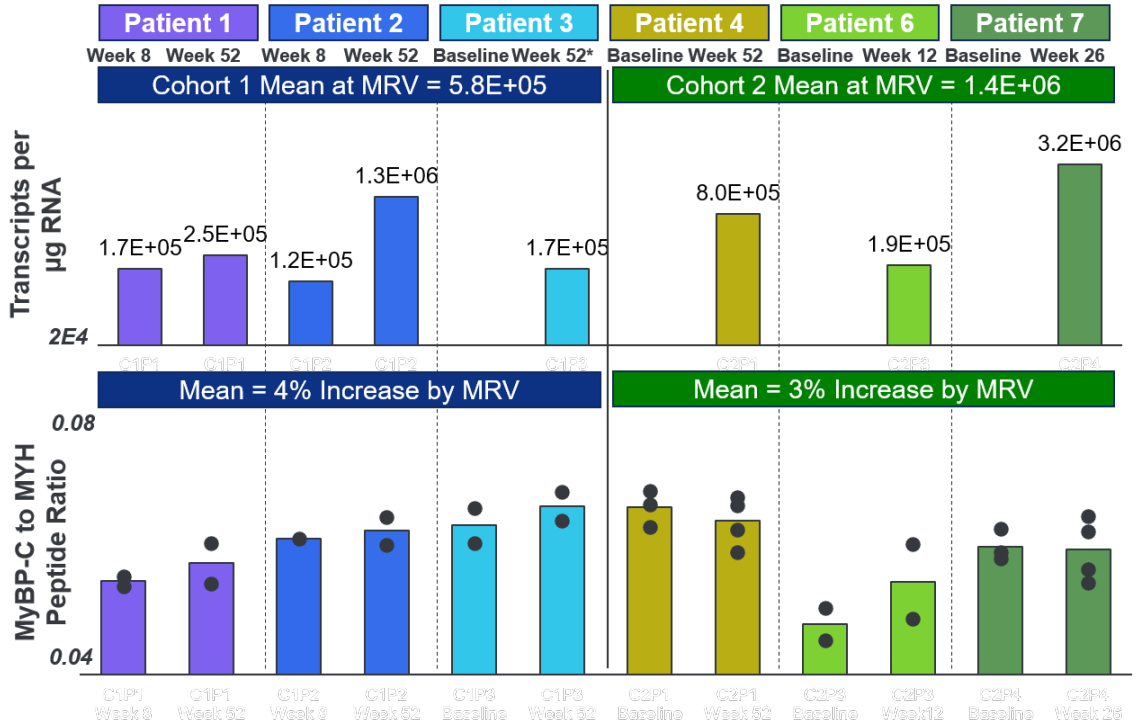
**Patient 5 lost to follow up after tapering from immunosuppressives*

6MWD = 6-minute walk distance
pVO_2 = Peak oxygen uptake

[1]MCID= >30m; Shoemaker, et al., *Cardiopulm Phys Ther J* 2013
[2] MCID=6% increase; Swank, et al., *Circ Heart Fail* 2014

TN-201 is being expressed in cardiomyocytes and is generally well tolerated

Totality of biopsy data show TN-201 is achieving transduction and expression



TN-201 has been well tolerated at both doses

AEs associated with TN-201 treatment were mild, transient and/or reversible

- No clinical TMA
- No need for complement inhibitors
- No signs of cardiotoxicity

The most common AE related to TN-201 were reversible, asymptomatic liver enzyme elevations

TN-201-related AEs	Cohort 1* (3E13 vg/kg; n=3)				Cohort 2* (6E13 vg/kg; n=3)			
	Gr 1	Gr 2	Gr 3	Total	Gr 1	Gr 2	Gr 3	Total
Liver enzyme elevations	1	1	1	**3**	1	-	-	**1**
Platelet reduction/ thrombocytopenia	-	-	-	**0**	2	-	-	**2**
Complement elevation	-	-	-	**0**	2	-	-	**2**

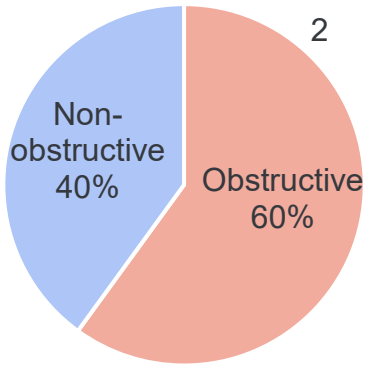
May 2026 data cut off
**Patient 5 lost to follow up after tapering from immunosuppressives*

TN-201 intended to treat the full spectrum of *MYBPC3*-associated HCM

Addressable HCM populations in the U.S.

HCM prevalence is growing driven by increasing diagnosis and aging population

All HCM
700,000 – 1M [1]



[2]

Patients with **obstructive disease** may be eligible for treatment with cardiac myosin inhibitors (CMIs)

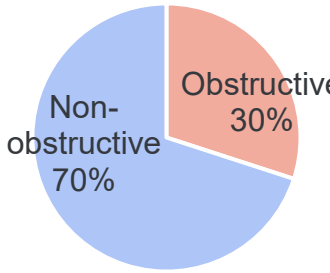
For those with **non-obstructive HCM** there are currently no approved treatments.

CMIs and cardiac sarcomeric modulators are in late-stage development

Total TN-201 potential opportunity

MYBPC3 variants account for 20% of all HCM cases[3]

MYBPC3+ HCM
140,000 – 200,000



MyPEAK-1 is evaluating TN-201 in adults with *MYBPC3*+ HCM

Those <18yo suffer the greatest unmet need

~3,000[4] diagnosed and currently <18yo

Pediatrics represent an uncontested opportunity with potentially faster path to approval

MyClimb Natural History Study

MyCLIMB natural history study has enrolled >220 volunteers; may serve as run-in study and control arm for future pediatric pivotal trial

1. Butzner, et al; JACC Advances 2026 and Semsarian, et al; JACC 2015
2. Estimates vary due in part to lack of diagnosis. This is the most widely accepted breakdown: AHA: Hypertrophic Cardiomyopathy
3. Sedaghat-Hamedani, et al; Clin Research Cardiol; 2018
4. SHaRe registry; Data on file

Children with *MYBPC3*+ disease are at heightened risk for heart failure, arrhythmias, hospitalizations and death[1]

The unmet need in pediatric patients

>90% of patients in MyClimb have non-obstructive HCM[1]

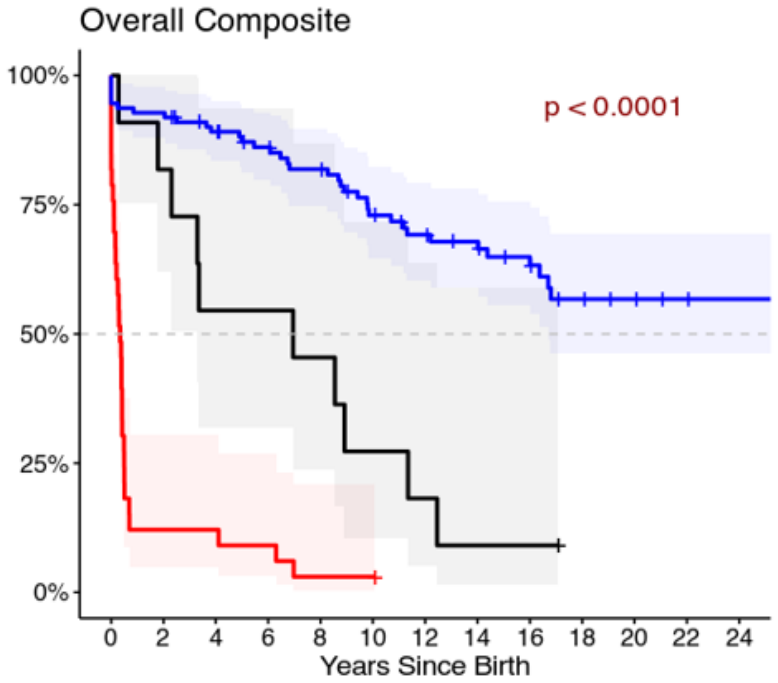
36% more likely to develop life-threatening ventricular arrhythmias[2]

0 treatment options targeting the underlying cause of disease or capable of slowing progression

TN-201 granted FDA **Rare Pediatric Disease Designation** for the treatment of *MYBPC3*-associated HCM in children, adolescents, and young adults

Genotype status impacts outcomes[1]

MyClimb Natural History Study



HOMOZYGOUS:
85% die or require transplant before age 1

COMPOUND HETEROZYGOUS:
64% experienced heart-failure related hospitalizations before age 10; 27% required transplant or died

HETEROZYGOUS:
Median age of diagnosis was 6.5 years. Significant burden of cardiomyopathy including potentially fatal arrhythmias and hospitalization

1. Brugada, et al, ESC 2025
2. Marston, et al, *Eur Heart Journal* 2021

TENAYA™ THERAPEUTICS

TN-401 for *PKP2*-associated ARVC

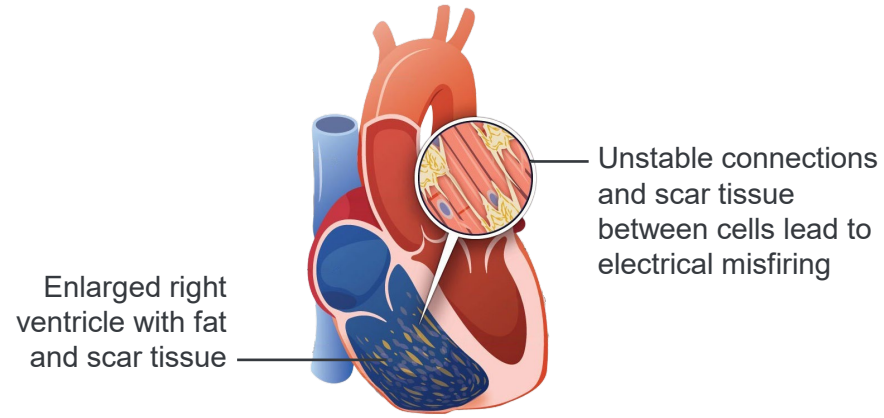
CONFIDENTIAL

PKP2-associated ARVC is estimated to affect >70,000 people in the U.S.[1]

ARVC is a severe and progressive genetic heart disease lacking therapeutic treatment options

TRACY | AGE 45
Living with *PKP2*+ ARVC

ARVC HEART



- Early symptoms include palpitations, lightheadedness, fainting[1]
- Significant impact on quality of life due to arrhythmias, ICD shocks and restrictions on physical exertion[1]

>15% of heart-related deaths in patients < 35 are due to ARVC[1]

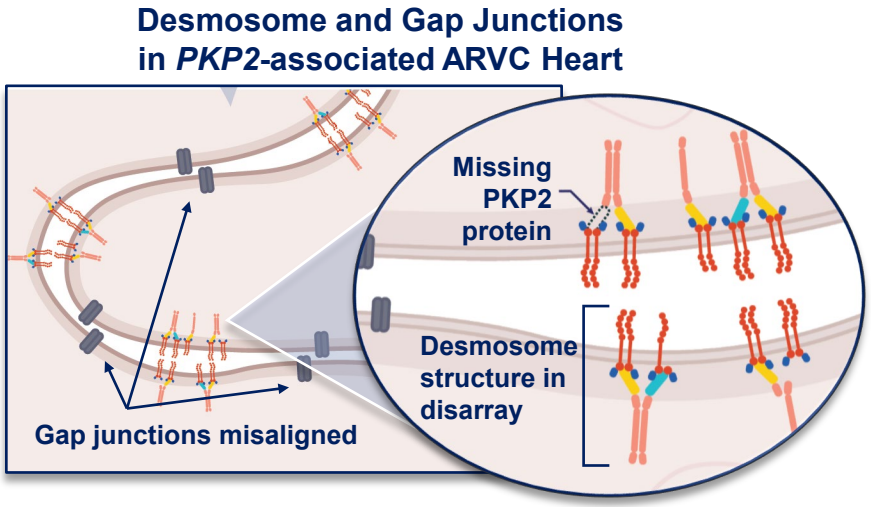
~40% of those diagnosed with ARVC carry pathogenic PKP2 mutations[2]

90% have >500 PVCs/day, despite standard of care[3]

1. Sedaghat-Hemedani, et al., *Clin Res Cardiol* 2018
2. Ho, et al, *Circulation* 2018
3. Marston, et al, *Eur Heart Jrnl* 2021

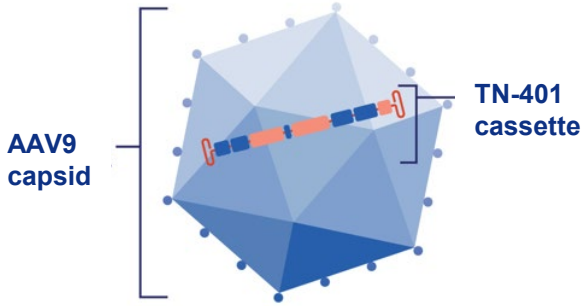
TN-401 gene therapy for PKP2-associated ARVC

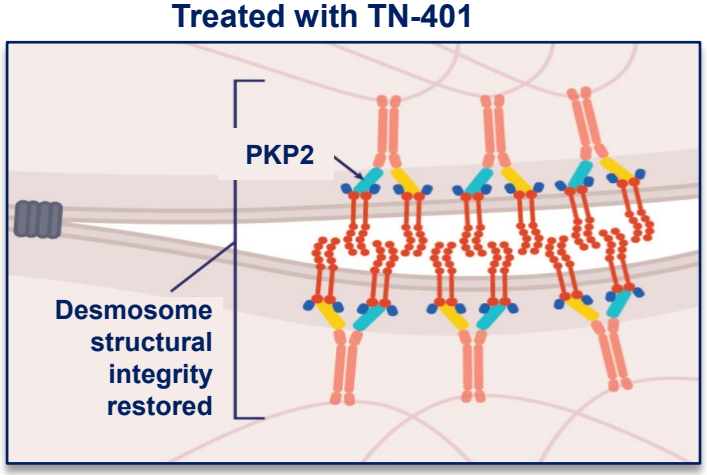
PKP2+ Pathophysiology



- **Mutations of the *PKP2* gene lead to lower levels of PKP2 protein**[1] resulting in
  - Weakened cell-to-cell adhesion
  - Abnormal electrical activity

TN-401 Mechanism of Action





TN-401 delivers a full-length *PKP2* gene to cardiomyocytes via an AAV9 capsid to target the underlying genetic cause of disease

An increase in PKP2 protein levels is expected to restore desmosome function, with the potential to halt disease progression, reverse symptoms, improve patient quality of life and prevent SCA

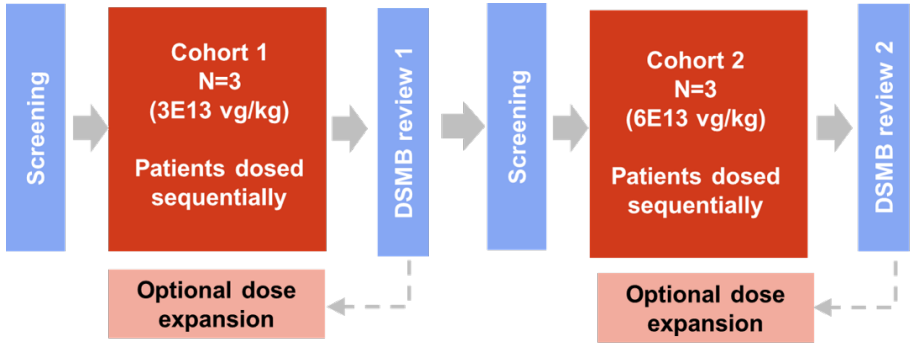
AAV9 = Adeno-associated virus, serotype 9
SCA = Sudden cardiac arrest

1. van Tintelen, et al, Circulation 2006

RIDGE-1 at-a-glance:
Aiming to reduce arrhythmic events

RIDGE-1 Phase 1/2 clinical trial

Designed to assess safety, tolerability and dose



RIDGE-1 patients have greater arrhythmia burden vs. typical population in natural history

- ICDs required for management of ventricular events
- PVCs > 500/day despite background medications
- 6 patients dosed as of May data cut
- 6 evaluable for molecular and clinical assessments as of April 2026 data cut; 3 from each dose cohort

Strong early evidence of improved electrical stability in all patients

- All evaluable patients saw measurable improvements in arrhythmia burden
- Measures of RV dysfunction remain stable

- Dramatic decreases observed in daily PVC counts and NSVTs

- TN-401 gene therapy is reaching heart cells and being expressed

- TN-401 has been well tolerated at both doses
- No cardiotoxicities

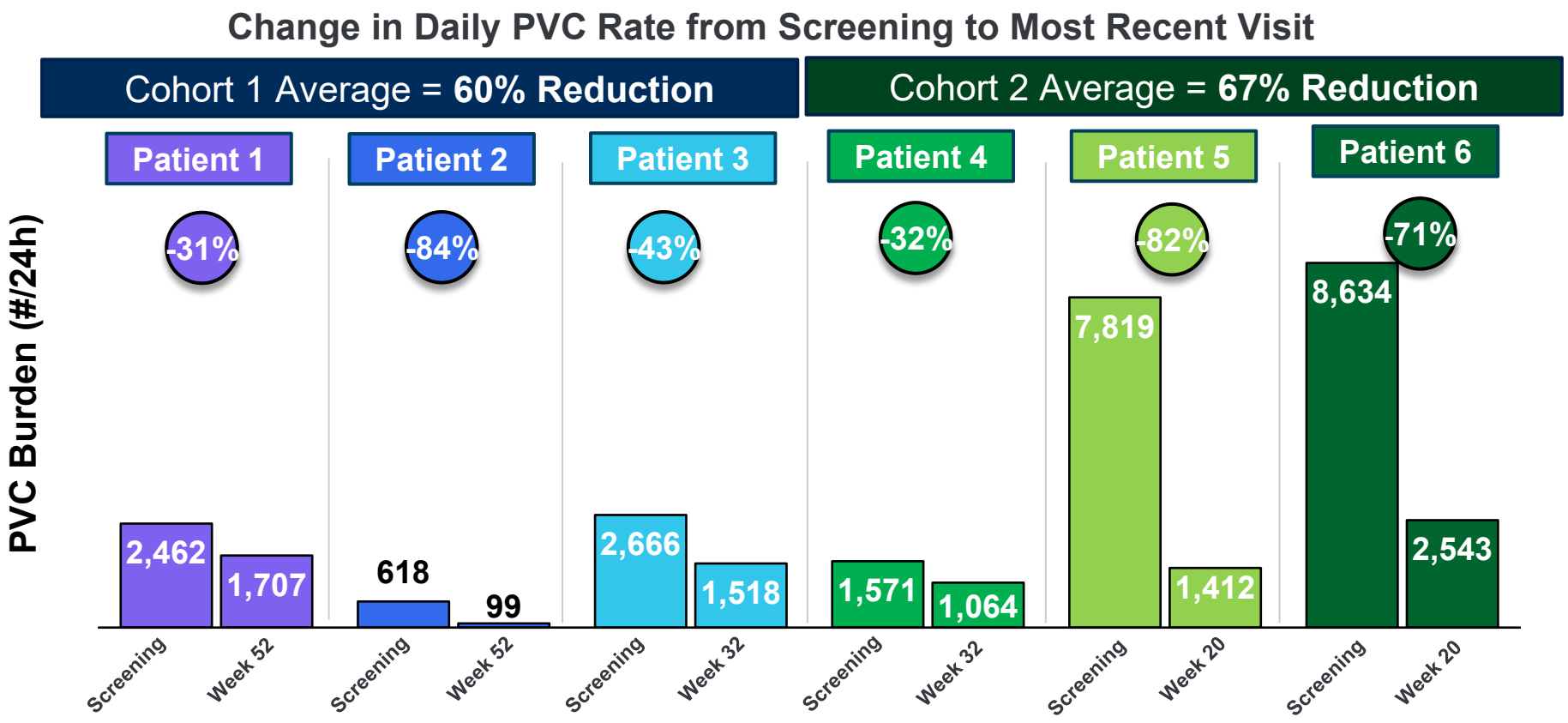
RIDGE-1 interim results supported RMAT designation from FDA

- Previously granted Fast Track and Orphan Drug designations from FDA and PRIME from EMA

PVC = Premature ventricular contractions
NSVT = Non-sustained ventricular tachycardia
ICD = implantable cardio defibrillator
DSMB = Data safety and monitoring board

Consistent reductions in PVC burden achieved in all patients post-TN-401

64% mean reduction in daily PVC count across all patients as measured by continuous ECG over 7-day period



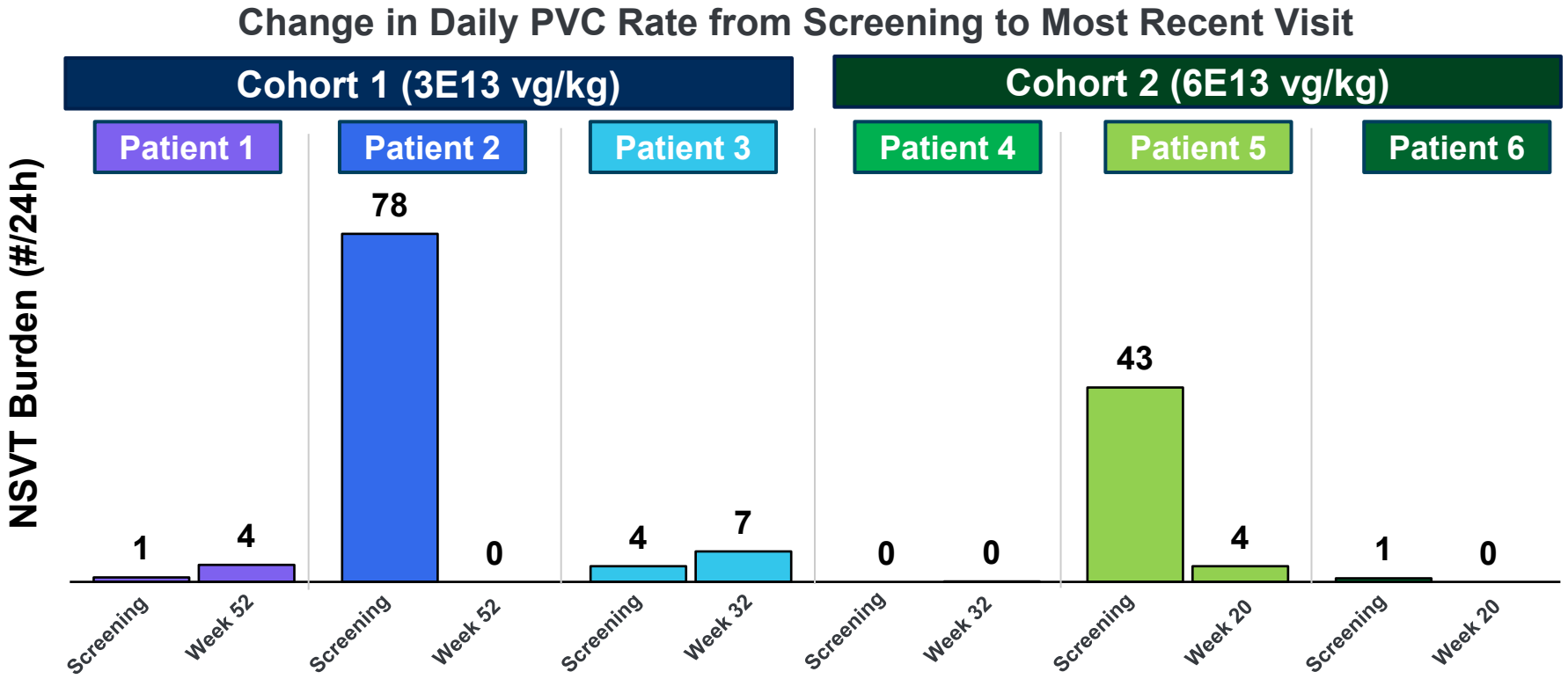
- PVCs are a known risk factor for more dangerous ventricular arrhythmias
- PVC reductions for Patient 1 and 2 remain durable up to 1 year
- Cohort 2 reductions more dramatic and rapid, potentially a dose effect

Data as of April 2026 data cut
Average change: geometric mean percent change from screening

ECG = electrocardiogram

Patients with high NSVT burden had dramatic reductions over time

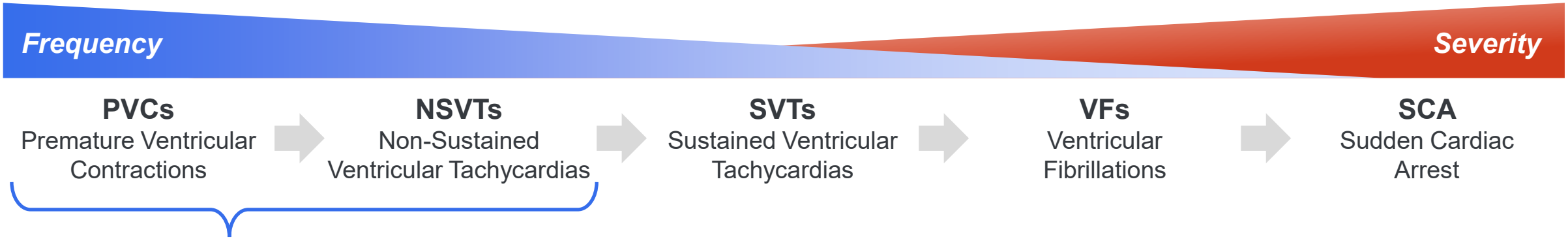
Patients 2 and 5 saw major declines in daily NSVT burden, while other patients maintained low and stable rates



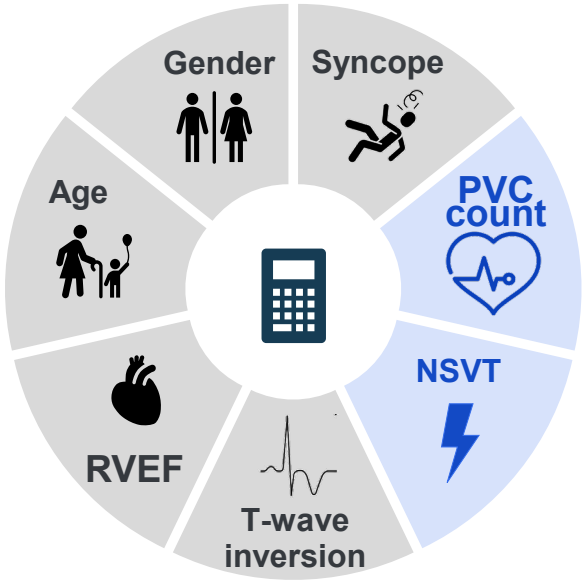
- NSVT burden assessed by 7-day continuous ECG
- All patients are stable across ECG measures, including T-wave inversions and QRS durations

Data as of April 2026 data cut

PVC and NSVT burden are key indicators of electrical instability and risk of life-threatening events

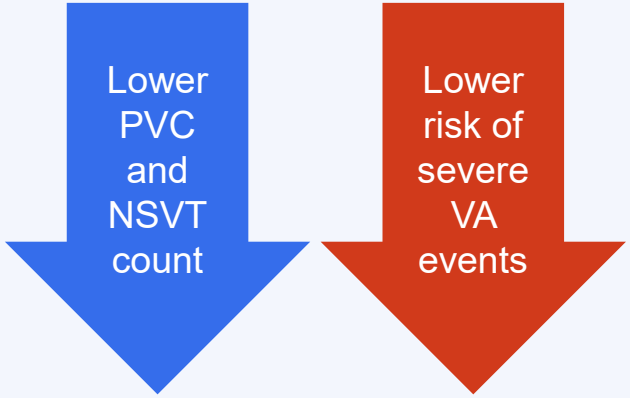


ARVC Risk Stratification Calculator



- PVCs are hallmark of *PKP2*+ ARVC and indicate electrical instability[1]
- Higher PVC counts are a recognized clinical predictor of higher 5-year risk of life-threatening VAs[2]
- PVC burden utilized as risk assessment tool for ICD placement[3]
- Recent KOL paper endorses PVCs as an appropriate clinical endpoint for pivotal studies vs. other measures[4]

Goal of TN-401 Gene Therapy



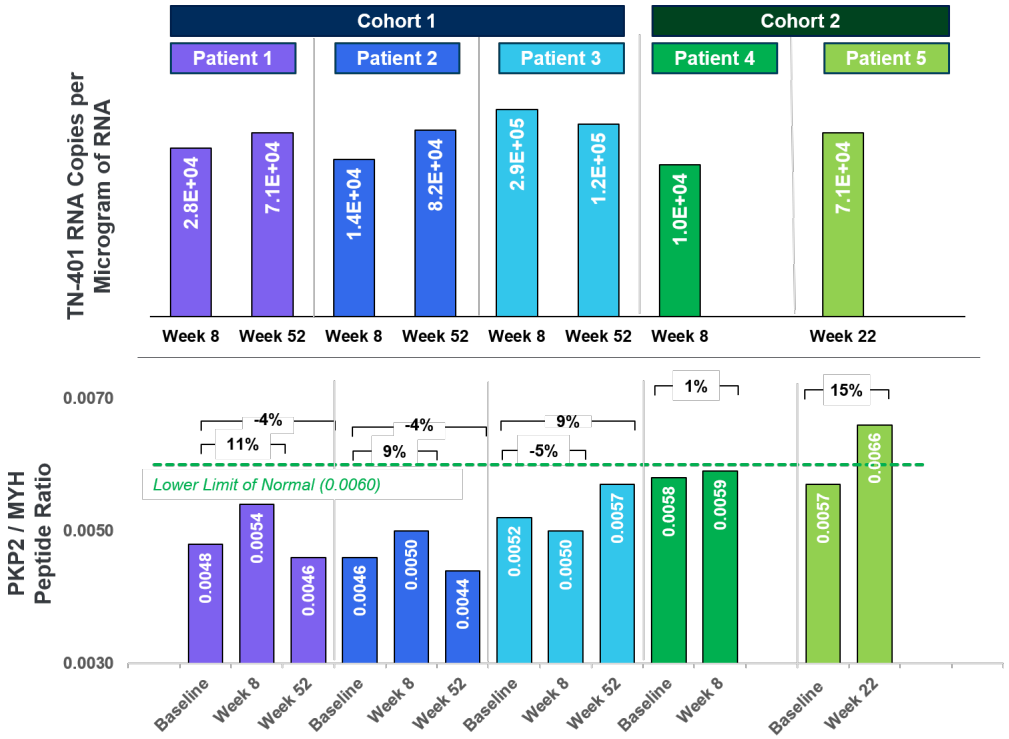
SVT = sustained ventricular tachycardia
RVEF = Right ventricular ejection fraction

1. Calkins, et al, HRS 2025
2. Carrick, et al, Circ Arrhythm and Electrophysiol 2022
3. https://arvcrisk.com/
4. Gasperetti, et al,; Heart Rhythm; 2026

PKP2 is being expressed in cells and TN-401 is generally well tolerated at both doses

Biopsy data support TN-401 pharmacodynamic effect

- Mean TN-401 DNA levels were 3.4 vg/dg (Cohort 1) and 3.8 vg/dg (Cohort 2)



TN-401 well tolerated at both doses

Majority of TN-401-related AEs were mild, asymptomatic and manageable

- No clinical thrombotic microangiopathy
- No sustained VT, VF or ICD therapy related to TN-401; no other cardiotoxicities observed

Relevant Adverse Event	Cohort 1* (3E13 vg/kg; n=3)				Cohort 2 (6E13 vg/kg; n=3)			
	Gr 1	Gr 2	Gr 3	Total	Gr 1	Gr 2	Gr 3	Total
Liver enzyme elevation	3	-	-	3	1	-	1**	2**
Troponin elevation	2	-	-	2	1	-	-	1
Thrombocytopenia†	-	-	-	0	-	-	1**	1**

*Previously disclosed, no new AEs since December 2025 release
**Event attributed to or associated with immunosuppression medication error
†Unrelated to TN-401

April 2026 data cut
**Patient 6 DNA analysis not completed by April 2026 data cut*

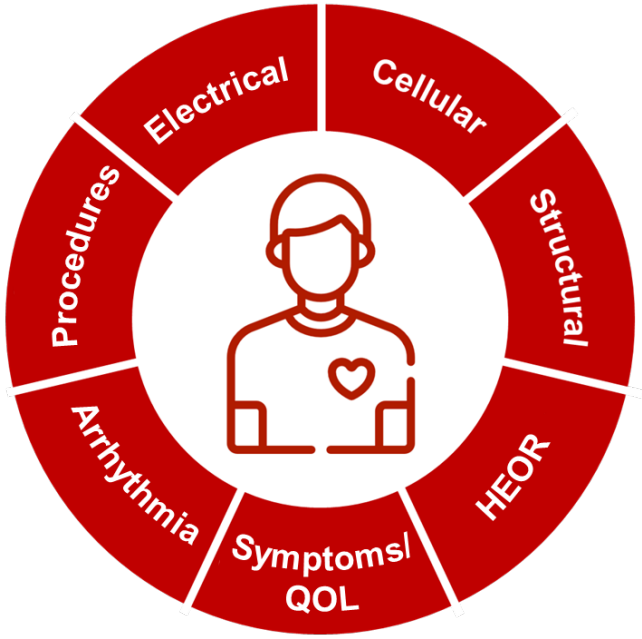
RIDGE™ the largest natural history study of PKP2+ ARVC in the world

Largest Ever PKP2+ ARVC Natural History Study…

RIDGE™

>185 Patients	**>2,500** Years of Follow-Up
21 Sites	**6** Countries

…Collecting Data Across Domains…



…Directly Feeding RIDGE-1 Trial

RIDGE™-1

- All RIDGE-1 participants to date were identified through RIDGE
- RIDGE informed RIDGE-1 design, including eligibility criteria and endpoints
- Complements trial results by demonstrating natural history without gene therapy
- Supports discussions with health authorities about potential approvable endpoints, pivotal trial design

~70% of RIDGE patients eligible for RIDGE-1

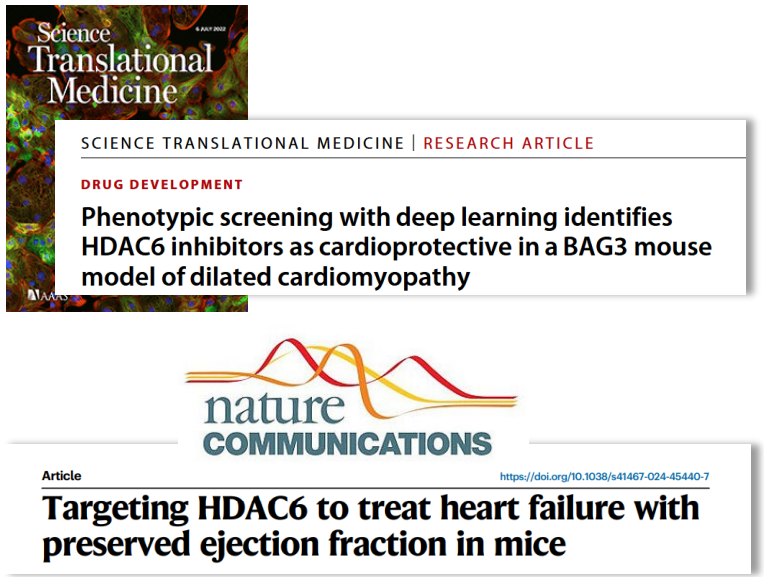
TENAYA™ THERAPEUTICS

TN-301 HDAC6 Inhibitor

Growing conviction in TN-301 opportunity supported by external preclinical and clinical data

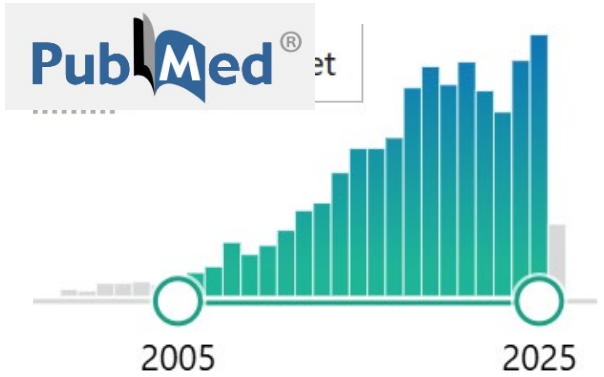
Tenaya has extensively studied HDAC6 inhibition

Preclinical validation achieved in models of HFpEF, DCM, PAH and DMD



HDAC6 inhibition multi-modal MOA externally validated

HDAC6 inhibition efficacy in multiple disease models – including cardiac & cardiac adjacencies – confirm Tenaya insights



>1200 articles

Clinical results with pan-HDAC inhibition de-risks TN-301

Duvyzat™ (givinostat)

- Givinostat significantly slowed disease progression in ambulatory DMD patients (4SC, NSAA)
- MOA attributed to anti-inflammatory effects of HDAC inhibition
- Approved in US & EU for patients age >6 years old
- Robust commercial uptake, including use on top of μ–dystrophin therapies

TENAYA THERAPEUTICS

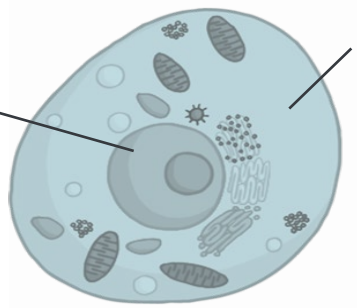
MOA = Mechanism of action
4SC = 4 Stair Climb
NSAA = North Star Ambulatory Assessment

Tenaya's HDAC6 inhibitors offer distinct advantages

HDACs are enzymes that regulate cellular processes and gene expression

HDACs

- HDACs 1-3, 5, 8 and 11 act inside the cell nucleus
- HDACs 4, 7 and 9 shuttle between
- nucleus and cytoplasm



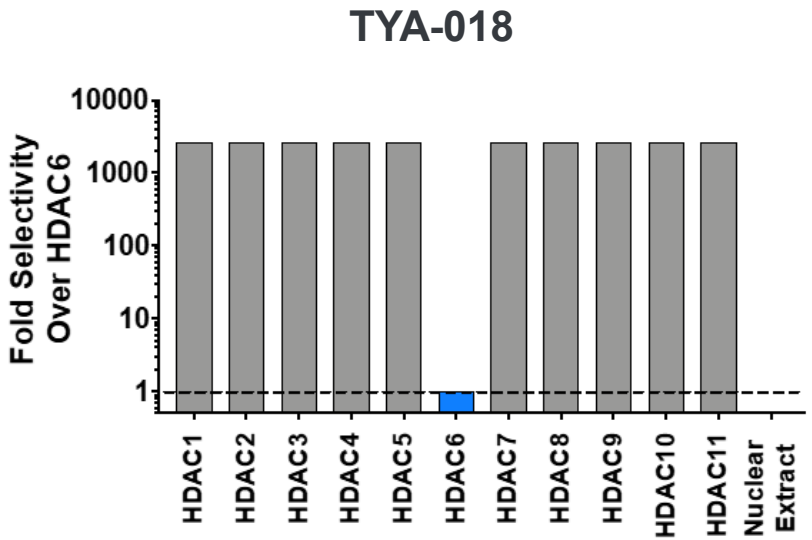
HDAC6

HDAC6 is a cytoplasmic enzyme that acts on proteins to modulate **c**yto-skeleton structure, protein quality control and cellular stress responses

HDAC6 inhibition improves

- Cytoskeletal / microtubule cell mobility (i.e., cellular trafficking and transport)
- Autophagy (i.e., cellular clean up)
- Inflammation
- Oxidative stress
- Metabolic regulation
- Fibrosis

Tenaya's HDAC6 inhibitors have ≥3500x selectivity for HDAC6 vs. other HDACs

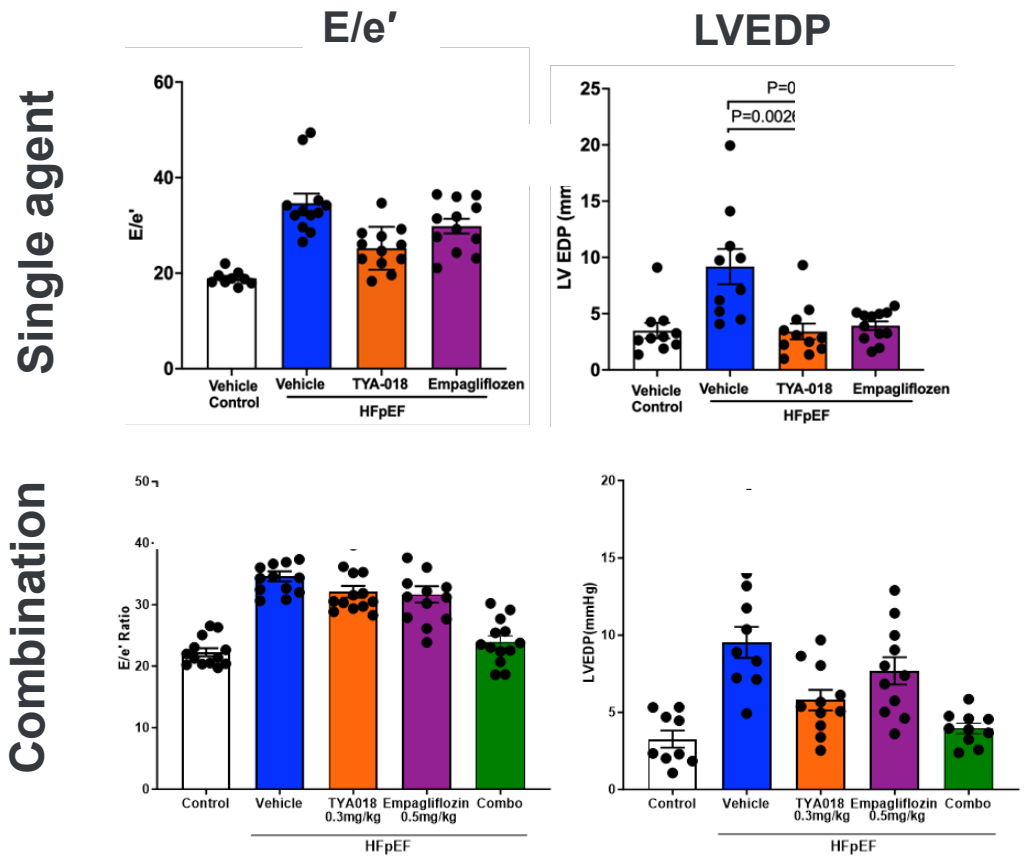


Selectivity provides potential safety advantages vs pan-HDAC inhibitors and allows for higher dosing to maximize efficacy

Hsp90 = heat shock protein 90
TYA-018 = preclinical surrogate structurally and functionally similar to TN-301

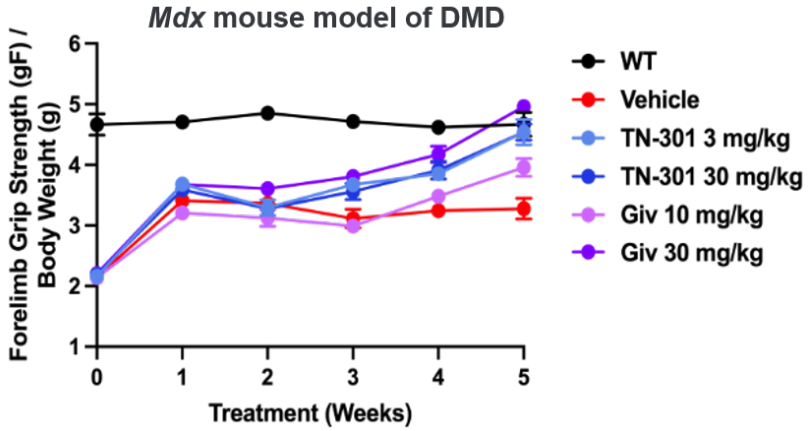
TN-301 demonstrates superior activity vs. commercially available therapeutics in validated disease models

HFpEF: Tn-301 improves diastole alone or in combination with SGLT2 inhibitors



DMD: Potential to improve skeletal and cardiac muscle deficits with greater tolerability

TN-301 restores grip strength to WT levels; outperforms givinostat



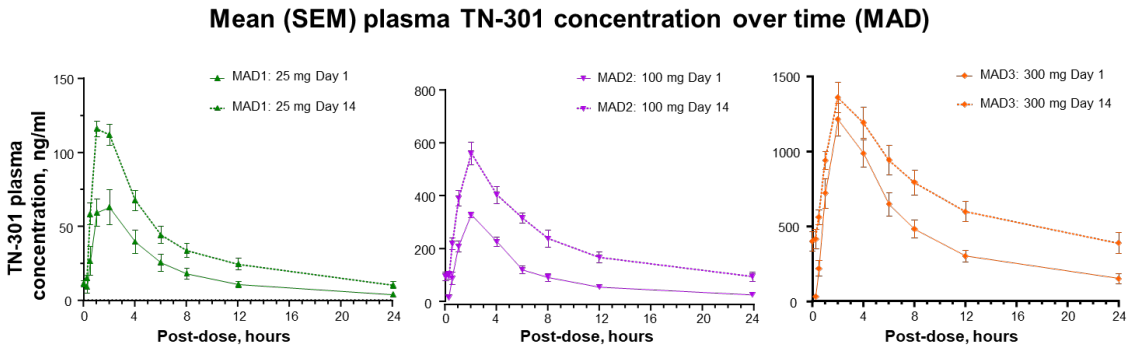
- TN-301 corrected key drivers of DMD cardiomyopathy (e.g., Ca+ handling, mitochondrial respiration) in engineered heart tissues
- Givinostat did not improve these measures

TN-301 Phase 1 trial results in healthy participants

TN-301 was well tolerated at all doses tested; AEs reported were mild and occurred with similar frequency to those in placebo arm

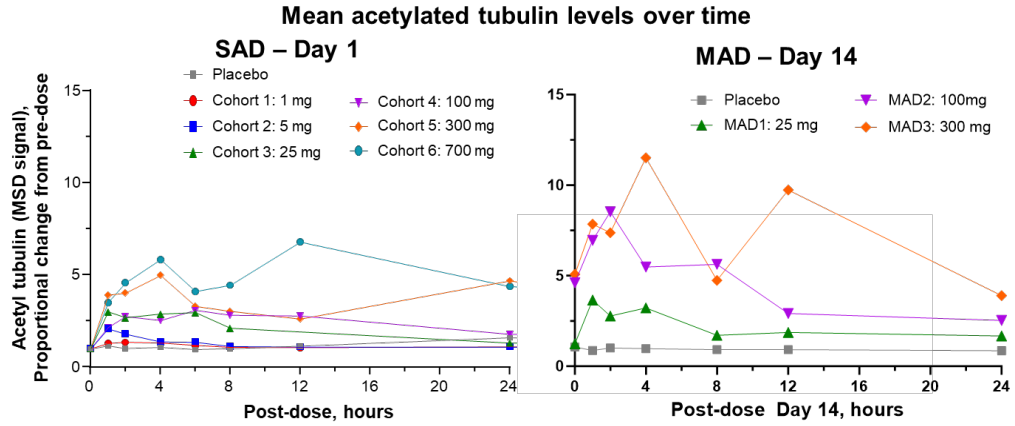
- Phase 1 evaluated single-ascending doses of 1mg–700mg and multiple ascending doses of 25mg, 100mg and 300mg for 14 days
- Most common AEs reported were GI-related
- No thrombocytopenia, no QT prolongation risk, and no histone modification

Pharmacokinetics and half-life support once-daily dosing



Plasma exposure increased proportionally with TN-301 dose across ranges evaluated

Selectivity for HDAC6 inhibition and target engagement demonstrated at low doses



Variability (SEM) in acetylated tubulin levels ranged from 0.038 to 1.410 (SAD results); and 0.067 to 4.050 (MAD results)

Increasing TN-301 exposure correlated with PD effect

AEs = Adverse events
GI = gastrointestinal
MAD = multiple ascending dose
SAD = single ascending dose
PD = pharmacodynamic

TENAYA™ THERAPEUTICS

Key Milestones & Financials

CONFIDENTIAL

Anticipated program milestones

TN-201 for *MYBPC3*-associated HCM

- Reported Cohort 1 & 2 MyPEAK-1 data
- Competed enrollment in MyPEAK-1

- **Q4'26:** Share longer-term results from MyPEAK-1
- **Q4'26:** Provide update on pivotal trial plan discussions with regulators

TN-401 for *PKP2*-associated ARVC

- Presented interim Cohort 1 & 2 RIDGE-1 data
- Competed enrollment in RIDGE-1

- **Q4'26:** Share longer-term results from RIDGE-1
- **Q4'26:** Provide update on pivotal trial plan discussions with regulators

TN-301 HFpEF/cardiac, muscular or metabolic disorders

- Presented preclinical data in DMD models vs. givinostat

- **Q4'26:** Announce development plans
- **2H'27:** Initiate Phase 2 proof-of-activity trial

TENAYA™ THERAPEUTICS

Cash and shares outstanding

$78.1M cash as of June 30, 2026[1] **Expected runway through Q3 2027**		**Number of shares (M)**
Common stock[2]	Shares outstanding	224.2
March 2025 warrants[3]	Exercise price of $0.80 per share, expire March 2030	75.0
December 2025 warrants[4]	Exercise price of $1.50 per share, expire December 2030	50.0
Common stock and warrants[2,3,4]	**Total outstanding**	**349.3**

[1]Reflects cash, cash equivalents, and marketable securities as of June 30, 2026; [2]Shares outstanding as of 8/5/2026; [3]Part of the March 5, 2025 underwritten public offering; [4]Part of the December 15, 2025 underwritten public offering.
Note: Total outstanding shares plus warrants disregards beneficial ownership limitations that may limit the ability of certain holders of warrants to convert into common stock

Thank you