Scaling new heights in the fight against heart disease

March 2024

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 2024 strategic priorities; 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; availability and content of data from MyPeak™-1; the timing of initial dosing for RIDGE™-1; targeted populations for clinical trials and treatments; the sufficiency of Tenaya's cash runway to fund operations, 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," "expected," "plan," "potential," "may," "objective," 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 a number of risks, uncertainties and assumptions described in our filings with the SEC, including, but not limited to the section titled "Risk Factors" in our Annual Report on Form 10-K for the fiscal year ended December 31, 2023, and other documents we have, or will file with the SEC. These filings, filed, are 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; 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.

Our purpose: To transform and extend lives through
the discovery, development and delivery of potentially curative therapies
that target the underlying causes of heart disease.

TENAYA™

Tenaya is well-positioned to lead innovation in precision medicines for heart disease

1 Singular focus on the heart

Leadership expertise in cardiology, gene therapy, and rare diseases

3
clinical-stage programs

TN-201 for *MYBPC3*-associated HCM
TN-401 for *PKP2*-associated ARVC
TN-301 for HFpEF

9 early-stage programs for rare / prevalent conditions

Funding into 2H
2025

1B+ Novel AAV capsids and regulatory elements screened

1000L cGMP AAV manufacturing scale

MYBPC3 = Myosin-binding protein C3 | HCM = Hypertrophic cardiomyopathy | PKP2 = Plakophilin-2 | ARVC = Arrhythmogenic right ventricular cardiomyopathy
HFpEF = Heart failure with preserved ejection fraction | AAV = Adeno-associated virus | cGMP = certified Good Manufacturing Practice

Genetic medicines for heart disease are on the cusp of a revolution

Increasing clinical and regulatory validation across the sector for precision heart medicines and for AAV gene therapies

2 recent approvals for precision heart medications

Vyndamax™ CAMZYOS™

FDA

Guidance supports **smaller pivotal studies** **"feel & function" endpoints** in heart failure (1)

> 100 genes associated with cardiomyopathies, clinical guidelines recommend genetic testing in the U.S. and Europe

ESC

7 approvals for potentially curative AAV gene therapies for diseases of the eye, brain, liver, & muscle (3)

> 5500 of patients in > 50 countries have received AAV gene therapies (4)

Gene therapies for rare disease have **2x-3.5x higher likelihood of approval vs other modalities** and more opportunities for expedited review (1, 2)

Early proof-of-concept data reported for multiple AAV gene therapies for genetically-driven cardiomyopathies

rocket pharma

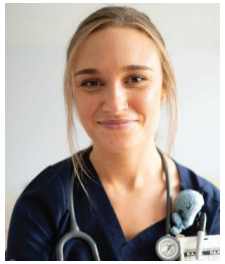
4DMT

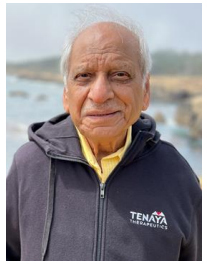
LEXEO therapeutics

TENAYA THERAPEUTICS

1. FDA Draft Guidance for heart failure; for cell and gene therapies
2. Alliance for Regenerative Medicine
3. FDA; EMA
4. Novartis; Kuzmin et al., Nat Rev Drug Discov 2021

Modality-agnostic drug discovery that aims to address the underlying problem

	HCM	ARVC	HFpEF	DCM	MI	HFrEF	DCM
Underlying problem	*MYBPC3* mutation	*PKP2* mutation	Multi-factorial	*PLN*^{R14del} mutation	Loss of heart cells	Decrease in SERCA2a	Unknown mutation(s)
Tenaya approach	Gene transfer	Gene transfer	Small molecule	Gene editing	Cellular regeneration	DWORF addition	Target ID via AI-enabled screens of heart cells
Tenaya programs	TN-201	TN-401	TN-301	← Preclinical efforts →			

MI = myocardial infarction | PLN delR14 = phosolambam deletion of arginine at amino acid residue 14 | SERCA2a = sarcoplasmic reticulum (SR) calcium-ATPase
DWORF= dwarf open reading frame | DCM = dilated cardiomyopathy | MI = myocardial infarction | AI = artificial intelligence

Deep and diverse pipeline addressing rare and prevalent indications

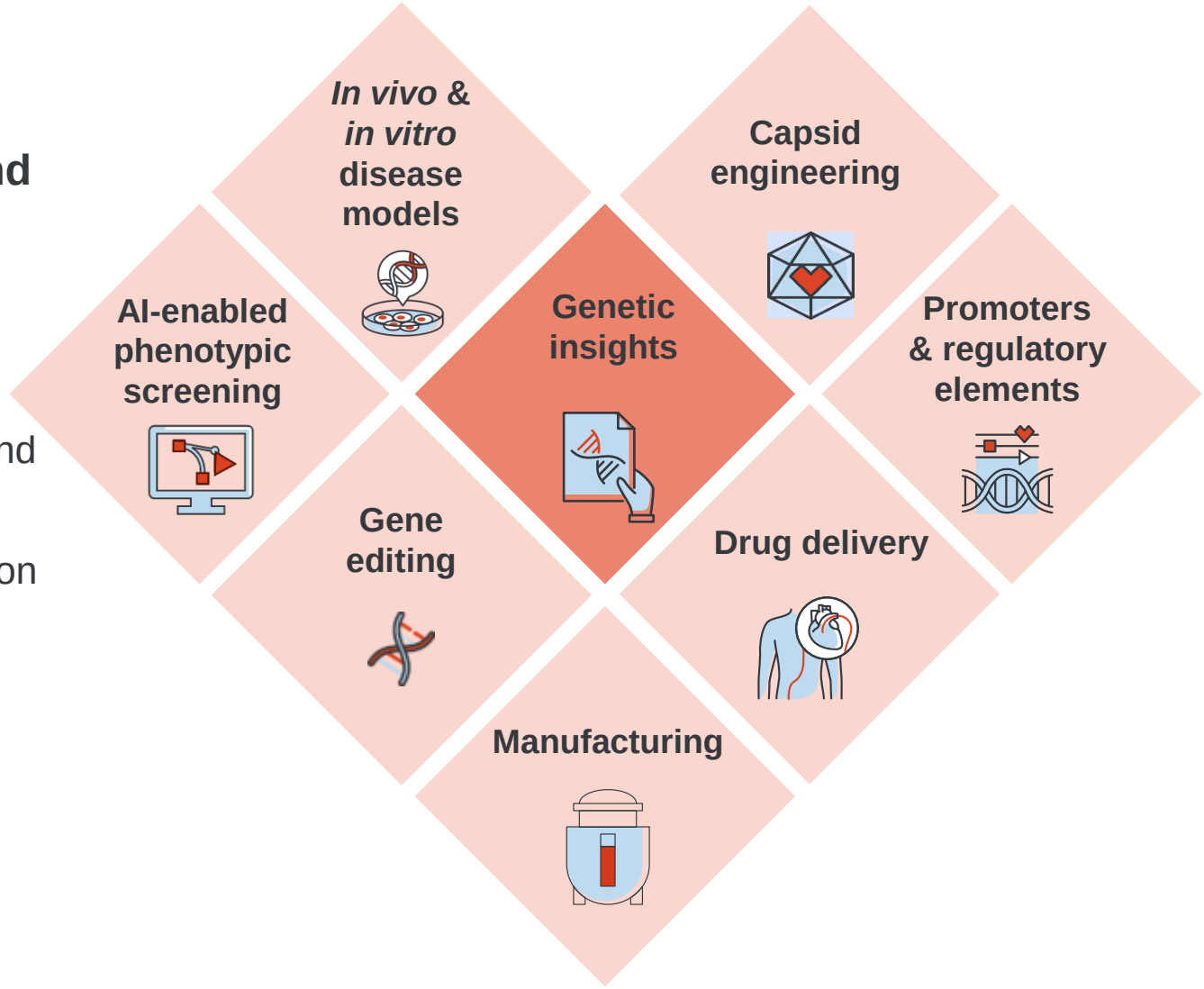
Program	Modality	U.S. Prevalence	Preclinical	Phase 1	Phase 2/3
Clinical-Stage Programs					
TN-201 for *MYBPC3*+ HCM	AAV9 gene therapy	> 120K[1]			
TN-401 for *PKP2*+ ARVC	AAV9 gene therapy	> 70K[2]			
TN-301 for HFpEF	Small molecule	> 3M[3]			
Research-Stage Programs					
DWORF for DCM & HFrEF	AAV gene therapy	Prevalent			
Post-MI Heart Failure	Cellular regeneration	Prevalent			
PLN^{R14del} DCM	Cas9 gene editing	Rare			
Undisclosed targets	Gene therapy Gene editing Gene silencing	Rare and Prevalent			

1. Sedaghat-Hemadani, et al., Clin Res Cardiol 2017
2. Groeneweg, et al, Circ Cardiovasc Gen 2015 & McKenna, et al, Nature Rev Cardio 2021
3. Abovich, et al, Am J Prev Cardio 2023

Integrated internal capabilities power modality-agnostic drug discovery engine

Target Discovery and Validation

- ✓ Deep insight
- ✓ Rapid design iterations
- ✓ Encouraging efficacy and safety signals
- ✓ Human genetic validation



Design, production, and delivery of genetic medicines

- ✓ Targeted delivery
- ✓ Robust expression
- ✓ Better product profiles
- ✓ Growing IP portfolio

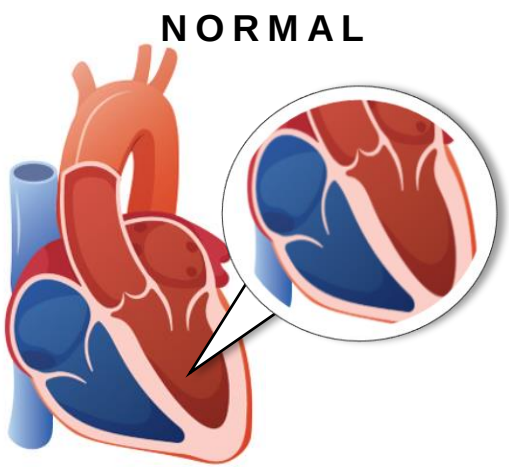
TENAYA THERAPEUTICS

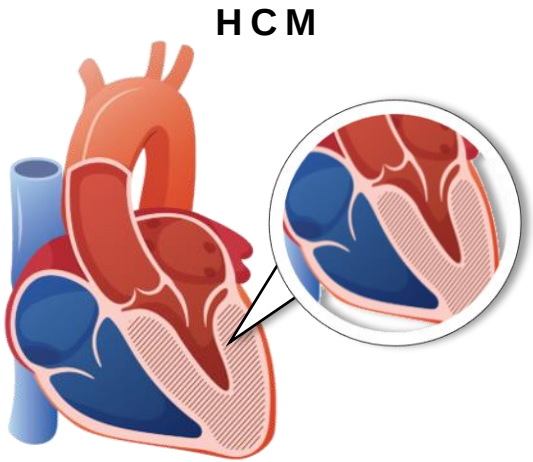
TN-201 gene therapy for *MYBPC3*-associated HCM

MYBPC3+ HCM is a severe and progressive genetic heart disease estimated to affect > 120,000 patients in the US alone

- Hallmark thickening of left ventricle (hypertrophy) due to excessive contraction
- Shortness of breath, fainting, chest pain, fatigue, palpitations and arrhythmias
- Typical age of onset is mid-40s
- Younger onset correlates with higher risk of morbidity and mortality (1)

- Significant functional impairment
- Social and psychological impacts
- Elevated risk of sudden cardiac death and heart failure
- Disease severity is higher when due to pathogenic sarcomeric gene mutations (1)



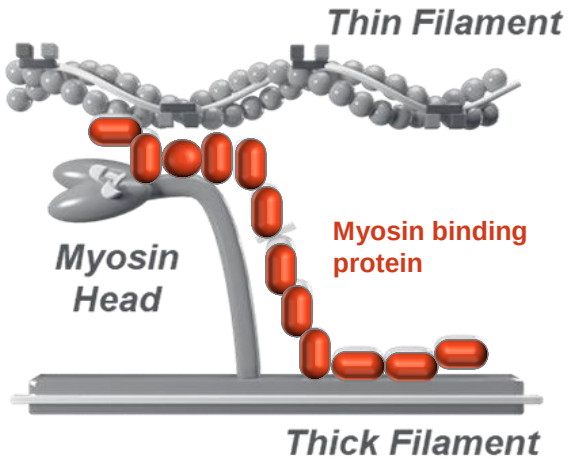


1. Ho, et al, Circulation 2018

TN-201 is the first gene therapy being developed for *MYBPC3*-associated HCM(1)

Underlying problem

- Mutations of the *MYBPC3* gene lead to lower levels (60%-70%) of myosin-binding protein C (MyBP-C)
- Less MyBP-C protein results in abnormal heart function



Tenaya Approach

- Designed to deliver a working *MYBPC3* gene to targeted cells
- Produce functional myosin binding protein to replace what is missing
- Utilizes AAV9 capsid proven to target human cardiomyocytes in clinical studies
- Novel promoter enables robust expression in cardiomyocytes

Key Advantages

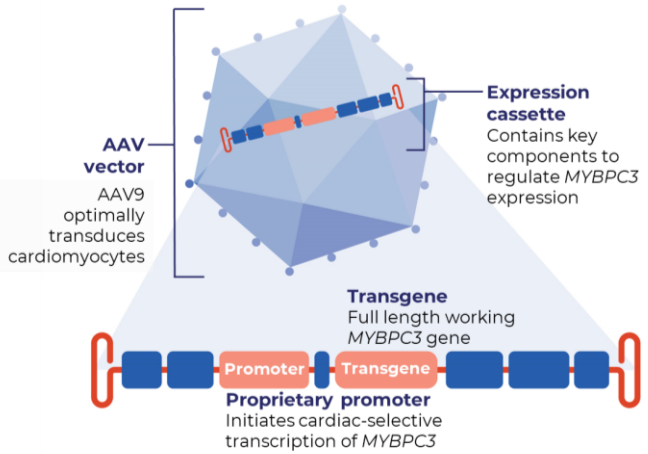
- Targets the underlying genetic cause of disease in a way that no approved treatment or surgical procedure can
- Potential product profile:
  - Single dose with durable effect
  - Restore protein levels
  - Halt disease progression
  - Improve heart function
  - Reverse disease symptoms
  - Improve quality of life

MyBP-C is an essential structural protein in the sarcomere of each cardiomyocyte required for normal heart contractions

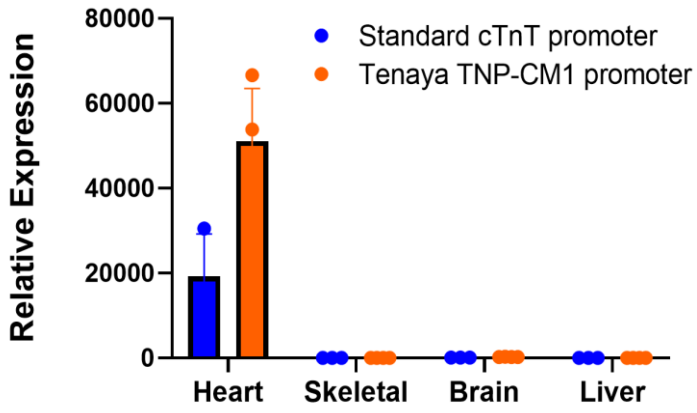
1. Based on publicly available information

TN-201 gene therapy construct has been designed to maximize safety and efficacy

TN-201 proprietary promoter and cassette design enables selective and robust gene expression in relevant heart cells

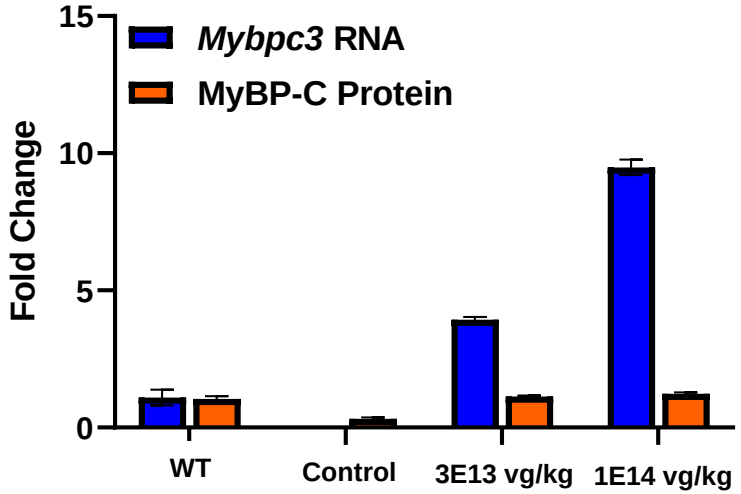


***In vivo* comparison with standard promoter (in WT mice)**



100% WT levels of MYBPC3 protein achieved in severe disease model with no protein overexpression

***In vivo* comparison (WT vs. *Mybpc3* KO mice)**



Unpublished; Tenaya Data on File

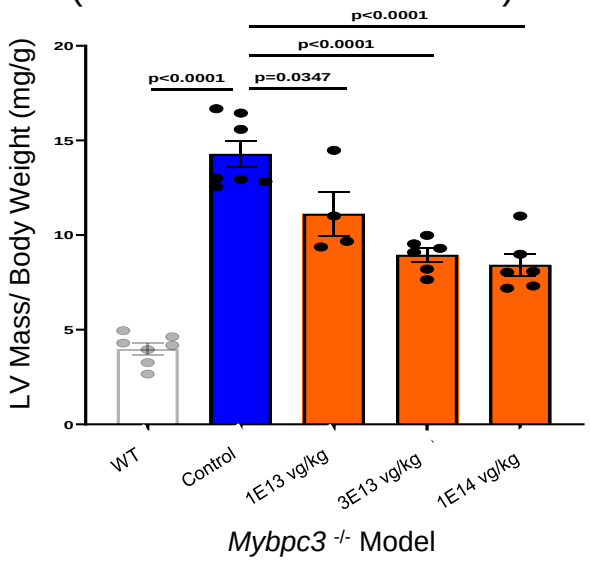
WT = wild type | cTnT = cardiac troponin (measure of cardiac injury) | RNA = ribonucleic acid
KO = knock out | MyBP-C = Myosin binding protein C (protein) | vg/kg = vector genome per kilogram

TN-201 preclinical evidence for disease reversal, improved heart function, increased survival

Single 3E13 vg/kg dose in *MYBPC3* KO mouse model results in reduction in hypertrophy, durable improvement in cardiac function and extended survival

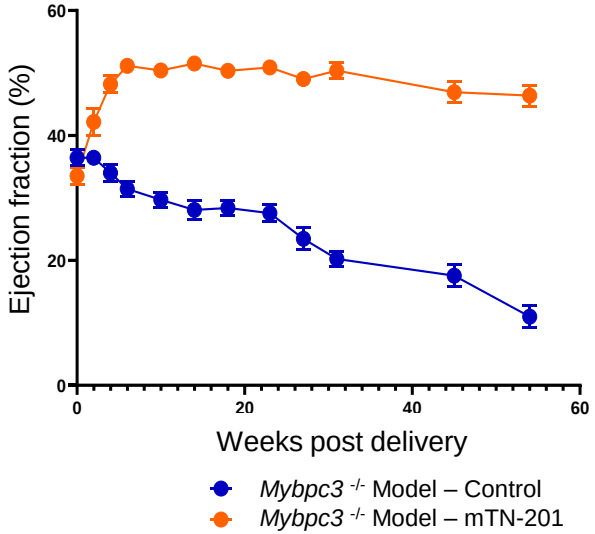
↓ Heart Mass

(8 Months Post-Treatment)



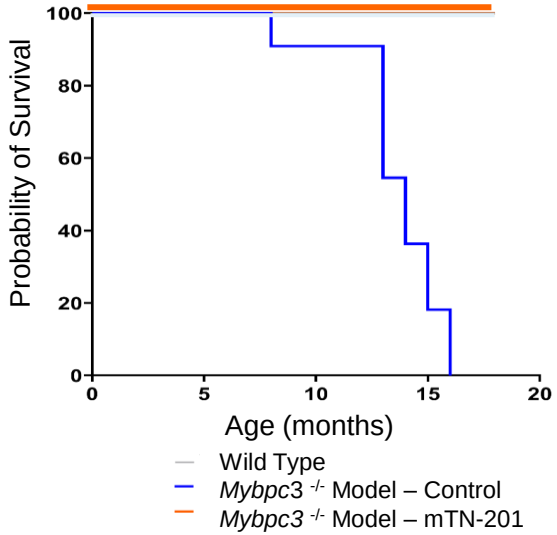
↑ Heart Function

(13 Months Post-Treatment)



↑ Survival

(18 Months Post-Treatment)



mTN-201 = mouse analog | LV = left ventricular

Lombardi, et al, ASGCT 2021

MyPeak-1 Phase 1b clinical study

Patient dosing initiated

Study Objectives

- Safety, tolerability, dose-finding and pharmacodynamics

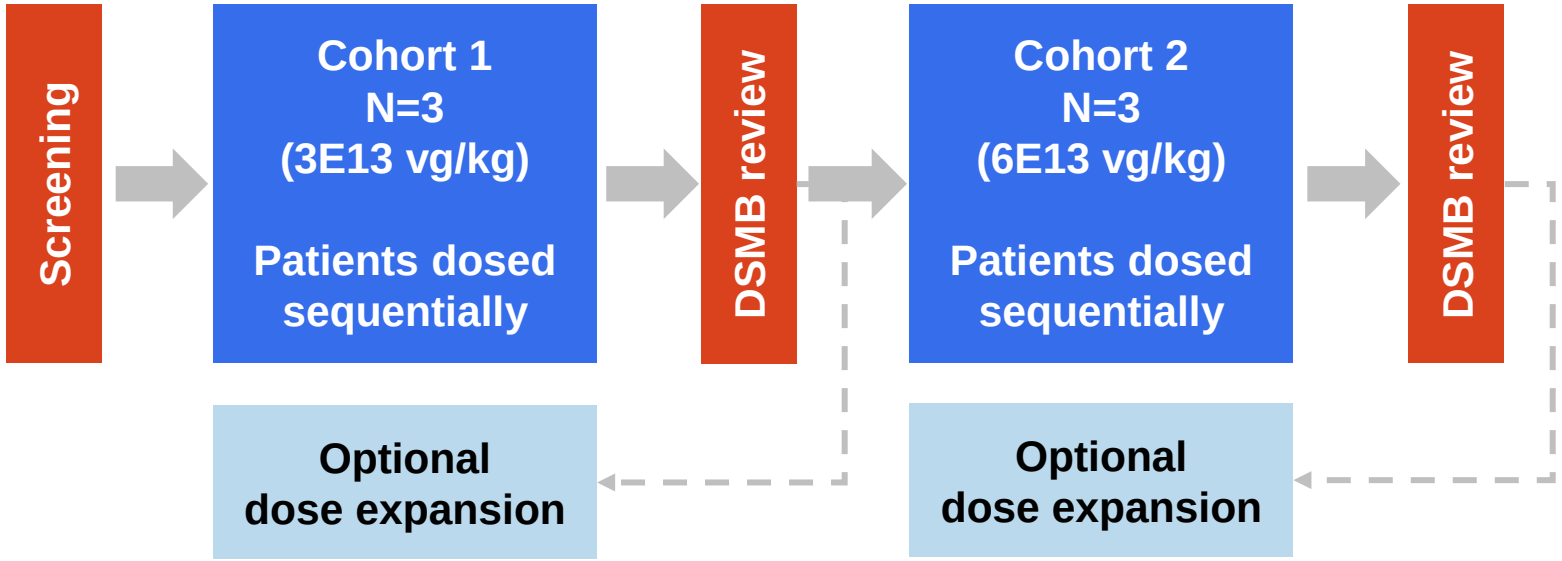
Eligibility

- Non-obstructive HCM
- *MYBPC3* mutation carriers
- Symptomatic (NYHA Class II or III)
- Adults (age 18-65)
- NT-proBNP ≥ 300pg/ml
- ICD present
- Low AAV9 neutralizing antibodies
  - Seroprevalence study initiated at MyPeak-1 sites in 2022

Design

Open-label, multi-center dose-escalation and dose-expansion study

- Preventive immunosuppressive regimen + close safety monitoring
- Regular assessments for safety, PK and PD
- 5-year safety and efficacy follow-up

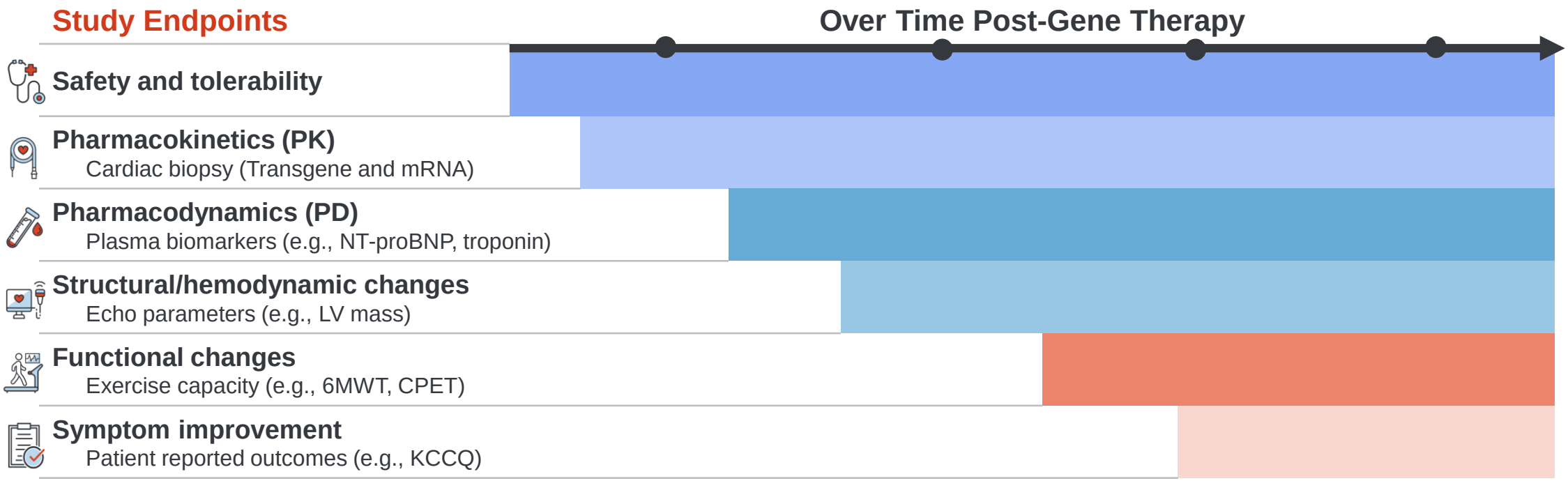


NYHA: New York Heart Association | NT-proBNP = B-type Natriuretic Peptide (measure of cardiac wall stress)
ICD = Implantable cardioverter defibrillator | PK = pharmacokinetics | PD = pharmacodynamics | DSMB: Data Safety Monitoring Board

MyPeak-1 Phase 1b clinical study

Initial data expected 2H 2024

First data readout expected to include (at least) safety, PK and PD
Seeking directional consistency across multiple parameters over time



mRNA = messenger ribonucleic acid | CPET = Cardiopulmonary exercise testing | 6MWT = 6-minute walk test | KCCQ = Kansas City Cardiomyopathy Questionnaire

Why TN-201 is positioned for success in 2024 and beyond

Tenaya's robust TN-201 preclinical package and MyPeak-1 clinical study design supports safety and efficacy doses as low as 3E13 vg/kg

Clinical cardiomyopathy experience across different modalities informs and de-risks TN-201 development

Preclinical*

- ✓ AAV9 broad distribution across heart
- ✓ Cardiomyocyte-specific expression
- ✓ High RNA & protein expression
- ✓ Improvements in
  - Hypercontractility
  - LV mass and thickness
  - Fibrosis
  - ECG parameters
  - Ejection fraction
  - Diastolic dysfunction
  - Survival

Clinical

- ✓ Starting dose associated with near maximal preclinical efficacy
- ✓ Data rich study
- ✓ Strong safety guardrails
- ✓ Multiple sites activated
- ✓ High patient engagement in seroprevalence study

- ✓ AAV9 transduction & durable expression [4]
- ✓ Improvements in
  - Circulating biomarkers [1,2,4,6]
  - LV mass & wall thickness [1,2,4,6]
  - Contractility [1,2,5]
  - Heart function [1,2,4]
  - NYHA class [1,2,4]
  - Exercise capacity [1,2,3,5]
  - Quality of life [1,2,4,5]
- ✓ Approval using feel & functions endpoints [1]
- ✓ Potential for accelerated approval [4]

(1) Bristol Myers Squibb™ (2) Cytokinetics® (3) Imbria (4) rocket pharma (5) 4DMT (6) LEXEO therapeutics

TENAYA THERAPEUTICS

* Tenaya data on file and presented

Plan to explore TN-201 in the full spectrum of patient presentation caused by MYBPC3 mutations

Infants | **Peds** | **Adolescents** | **Adults**

Homozygotes
(0% MyBP-C)

Heterozygotes
(≤ 60% - 70% MyBP-C)

Non-Obstructive Phenotype (70%)

Obstructive Phenotype (30%)

MyClimb Natural History Study

NYHA II NYHA III with ICDs

TENAYA · GENE THERAPY ™ MY PEAK-1

MyClimb natural history study will increase knowledge of pediatric HCM; may serve as a control for future pivotal study

MyPeak-1 relatively homogenous initial adult study population, plan for future expansion

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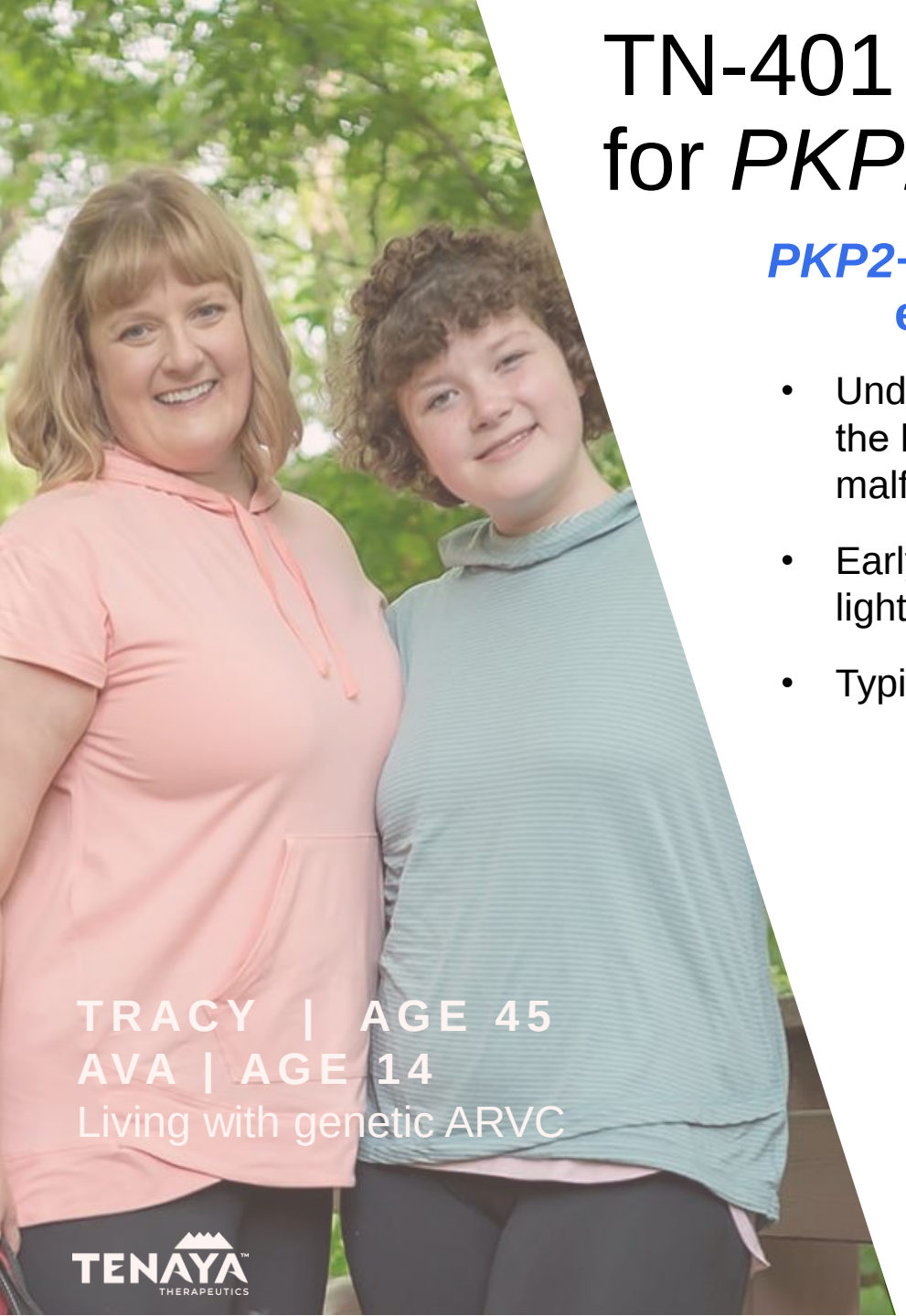


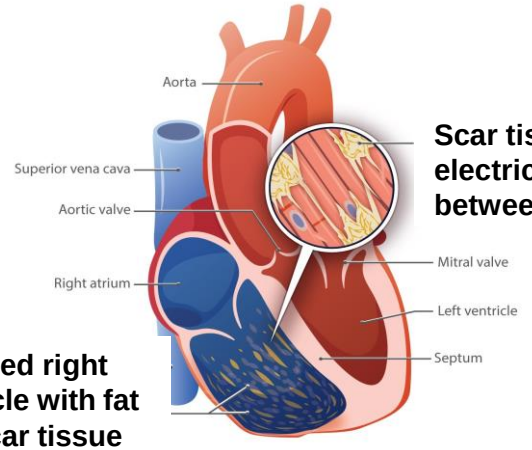
TN-401 gene therapy for *PKP2*-associated ARVC

PKP2+ ARVC is a severe and progressive genetic heart disease estimated to affect >70,000 patients in the U.S. alone

- Underdiagnosed disease in which the heart's electrical signaling malfunctions
- Early symptoms include palpitations, lightheadedness, fainting (1)
- Typically diagnosed before age 40
- 23% of ARVC patients present with sudden cardiac death
- Sudden cardiac arrest often first manifestation of disease (2)
- Significant impact on quality of life due to arrhythmias, ICD shocks and restrictions on physical exertion (3)

NORMAL

ARVC



Scar tissue leads to electrical misfiring between cells

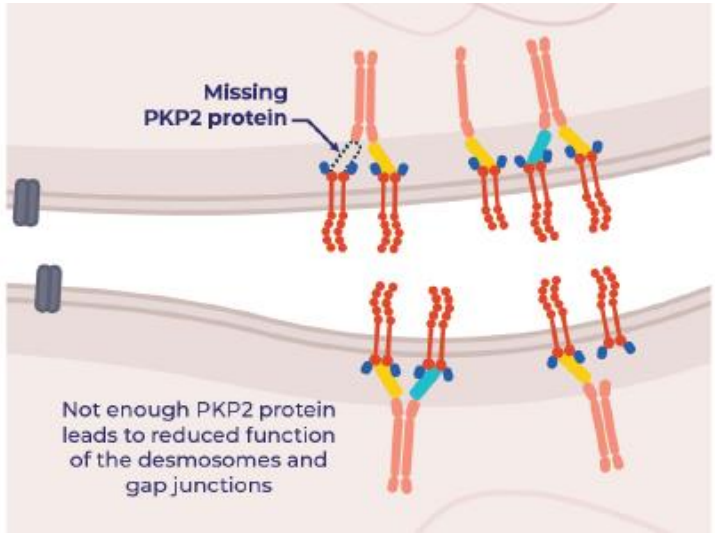
Enlarged right ventricle with fat and scar tissue

1. Dalal, et al, Circ, 2005
2. Hemida, et al, Eur J Heart Failure, 2018
3. SADS Foundation EL-PFDD for ARVC

TN-401 gene therapy for *PKP2*-associated ARVC

Underlying problem

- Mutations of the *PKP2* gene lead to lower levels of Plakophilin-2 (PKP2) protein[1]
- Less PKP2 protein results in abnormal heart function



Tenaya Approach

- Designed to deliver working *PKP2* gene to targeted cells
- Produce functional PKP2 protein to replace what is missing
- Utilizes AAV9 capsid proven to target human cardiomyocytes in clinical studies
- Cardiac-specific promoter enables robust expression in cardiomyocytes

PKP2 is an essential structural protein in the desmosome complex connecting every cardiomyocyte in the heart that supports proper electrical and mechanical signaling and overall tissue strength

Key Advantages

- Targets the underlying genetic cause of disease in a way that no approved treatment or surgical procedure can
- Potential product profile:
  - Single dose with durable effect
  - Restore protein levels
  - Halt disease progression
  - Improve heart function
  - Reverse disease symptoms
  - Improve quality of life

TENAYA THERAPEUTICS

1. McKenna, et al, Nature Rev Cardio 2021

TN-401 use of AAV9 capsid comes with robust validation from preclinical efficacy and clinical studies

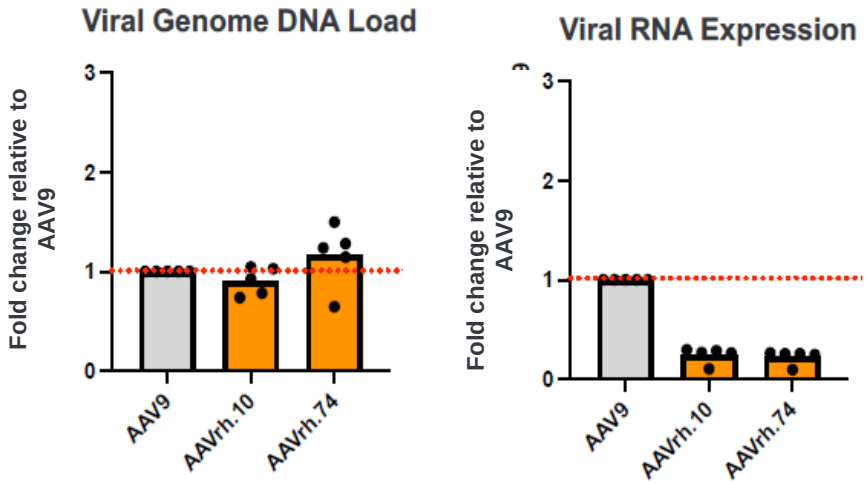
AAV9 Advantages

- ✓ **Most well-established safety record of any capsid,** has been used in >3000 patients in ~50 countries w/ >8 years follow-up (1)
- ✓ **Only capsid with validation from human hearts** for biodistribution, transduction, and durable gene expression (2)
- ✓ **Proven to achieve significantly higher gene expression in cardiomyocytes** vs other capsids in several preclinical studies (3)
- ✓ **Most extensive body of preclinical evidence in *PKP2* disease models** from three independently published studies (5,6,7)
- ✓ **Outperformed AAVrh74 in a head-to-head preclinical comparison in *PKP2* models** plus in other disease models (3,4)
- ✓ **Only capsid endemic to humans being used in *PKP2* clinical studies** as compared to AAVrh74 and AAVrh10 discovered in rhesus monkeys (8)

Head-to-head comparison of capsid performance illustrates difference between cardiac transduction vs expression

Transduction
(multiple heart cell types)

Expression
(cardiomyocytes only)

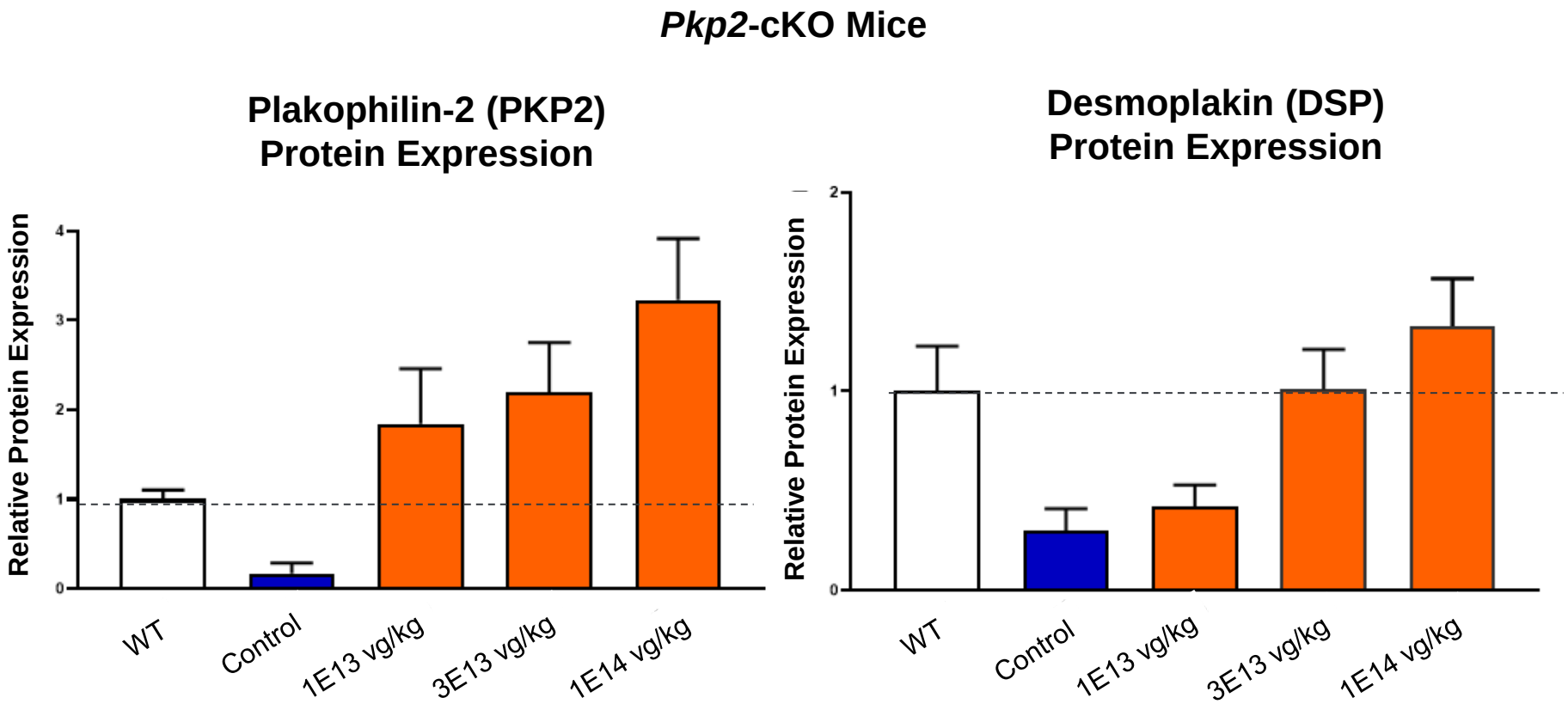


***In vivo* comparison in WT mice using cardiomyocyte-specific promoter**

Ze, et al. ASGCT 2023

1. Novartis March 2023 PR
2. Rocket corporate presntation
3. Tenaya data on file
4. Herzog, et al, US PTO US 2022/0168446 A1
5. 5 Bradford et al, Nature CV Res, 2023
6 Kyriakopoulou et al, Nature CV Res, 2023
7 (In press)Yang, et al, Communications Medicine
AAVrh = Adeno-associated virus rhesus isolate
8 Wang Nat Rev Drug Discov. 2019

AAV9-based gene therapy restores PKP2 protein to normal levels at low doses



Yang, et al, ASGCT 2022

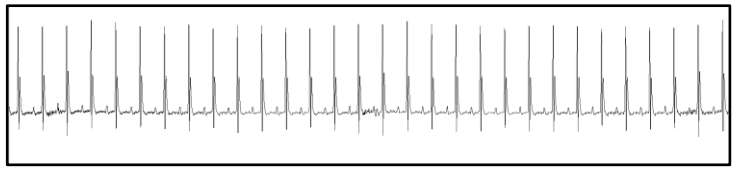
TN-401 preclinical evidence includes arrhythmia prevention, disease modification, survival benefit

Single 3E13 vg/kg dose in cardiac-specific PKP2 knock-out mouse model reverses hallmarks of disease

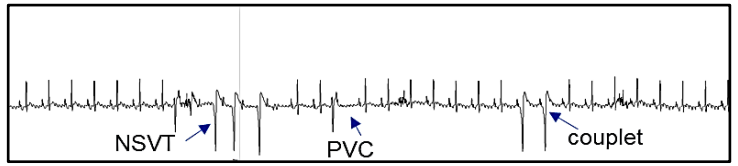
Extends survival ~6 months past control

↓ Arrhythmia

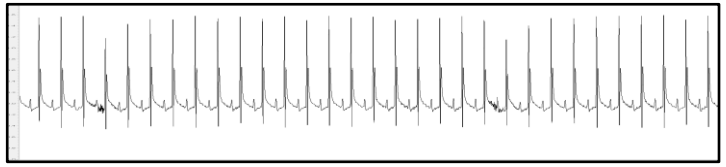
WT: Normal Sinus Rhythm



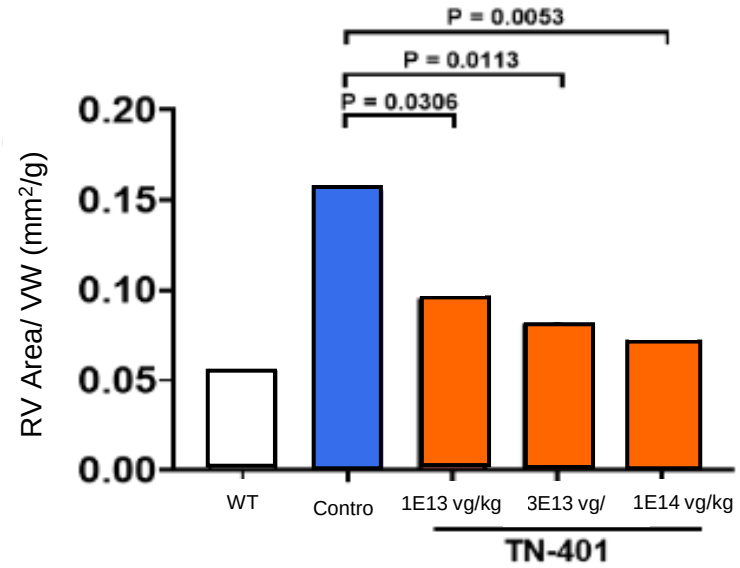
Untreated Control: Abnormal Ventricular Beats



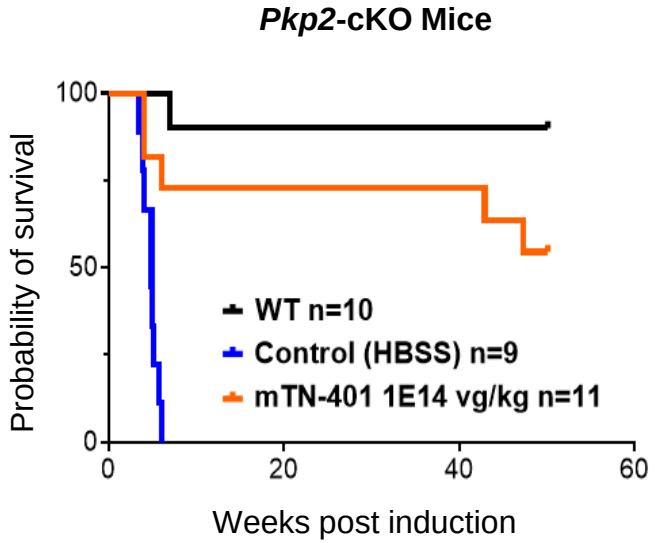
Gene Therapy: Normal Sinus Rhythm



↓ RV Enlargement



↑ Survival



Yang, et al, ASGCT 2022

RIDGE™-1 Phase 1b clinical trial for *PKP2*-associated ARVC

IND cleared; Patient dosing to begin 2H 2024

Study Objectives

- Safety, tolerability, dose-finding and pharmacodynamics

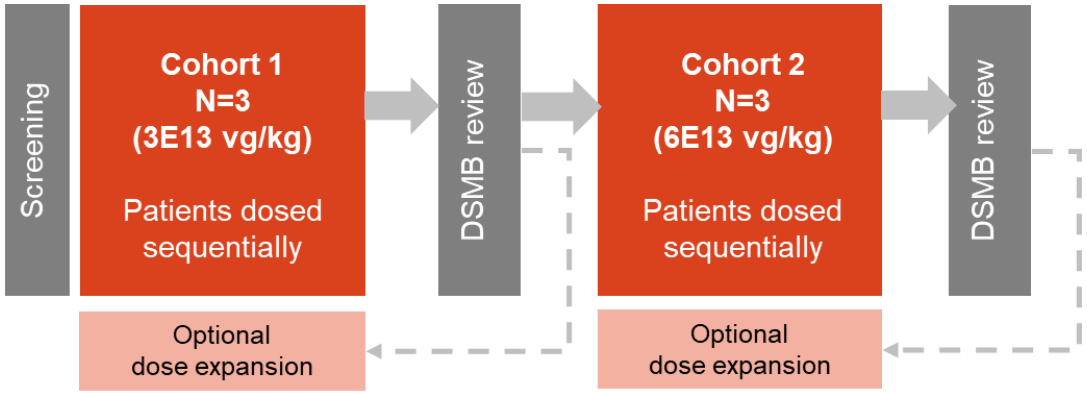
Eligibility

- ARVC diagnosis
- *PKP2* mutation carriers
- Adults (age 18-65)
- ICD present
- Mean PVCs >500 per 24 hours
- LVEF > 50%
- NYHA class I-III
- Low AAV9 titer

Design

Open-label, multi-center dose-escalation and dose-expansion study

- Preventive immunosuppressive regimen + close safety monitoring
- Regular assessments for safety, PK and PD
- 5-year safety and efficacy follow-up



Endpoints

- **Safety and tolerability**
- **Pharmacokinetics (PK)**
  - Transgene and mRNA via cardiac biopsies (baseline, 8wk & 52wk)
- **Pharmacodynamics (PD)**
  - Changes in PVC & NSVT
- **Exploratory efficacy endpoints**
  - Frequency of ICD shocks
  - Frequency of VTs
  - Imaging biomarkers (structural/hemodynamic changes by echo)
  - Plasma biomarkers
  - Patient reported outcomes

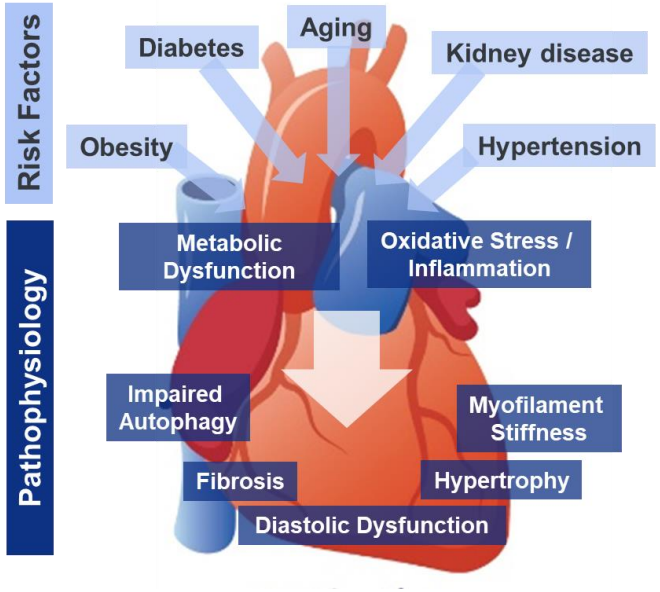
IND = Investigational New Drug application | ICD = implantable cardio defibrillator | LVEF = left ventricular ejection fraction
PVCs = premature ventricular contractions NSVT = non-sustained ventricular tachycardia

TN-301 small molecule HDAC6 inhibitor for HFpEF

HFpEF remains the largest unmet need in heart disease estimated to affect >3M in the U.S. alone (1, 2)

- Characterized by diastolic dysfunction driven by stiffening of heart ventricles
- Initial presentation includes shortness of breath, edema, fatigue, coughing, wheezing, dizziness
- Co-morbidities include obesity, metabolic syndrome, diabetes hypertension, atrial fibrillation, pulmonary disease, and renal dysfunction (3)
- 75% of people hospitalized with HFpEF die within 5 years (4)

- Multiple contributing risk factors resulting in complex pathophysiology



HDAC6 = histone deacetylase 6

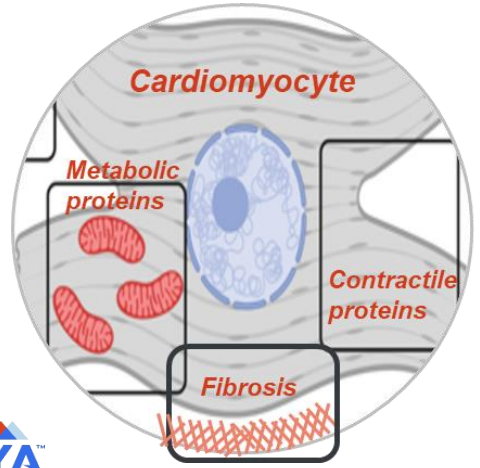
1. Cilia et al, Am J Lifestyle Med 2019
2. Tsao, et al Circulation 2023
3. Van Heerebeek, et al. Neth Heart Jour 2016
4.Shah, et al, JACC 2017

TN-301 small molecule HDAC6 inhibitor for HFpEF

Phase 1 complete; Optimally suited for development by/with a partner

About HFpEF

- Disease driven by multi-factorial processes involving many cell types and cellular structures:
  - Inside heart (cardiomyocytes, fibroblasts, mitochondria, sarcomeres, arterial walls)
  - Outside heart (systemic inflammation, oxidative stress, metabolic dysregulation)



Tenaya Approach

- TN-301 designed to specifically inhibit HDAC6 in the cytoplasm of heart cells
- HDAC6 inhibitor multi-modal MOA addresses diverse HFpEF pathophysiological processes
- Preclinical evidence of robust reversal of heart abnormalities (e.g., stiffness, hypertrophy) and systemic benefits (e.g., inflammation, glucose tolerance, insulin sensitivity, autophagy)

HDAC6 is a cytoplasmic enzyme that regulates diverse cellular processes in many different types of cells of the body

Key Advantages

- TN-301 high selectivity (1000x fold) for HDAC6 provides safety advantage vs partially selective HDAC6 inhibitors
- Pleiotropic effect has potential to address HFpEF co-morbidities (“pipeline in a pill”)
- TN-301 MOA is orthogonal to other heart medicines (e.g. SGLT2 inhibitors) and may yield additive benefits
- PD marker of target engagement conveniently measurable in human plasma
- Small molecule cost of goods appropriate for large indications

MOA = mechanism of action | SGLT2 =sodium glucose cotransporter-2

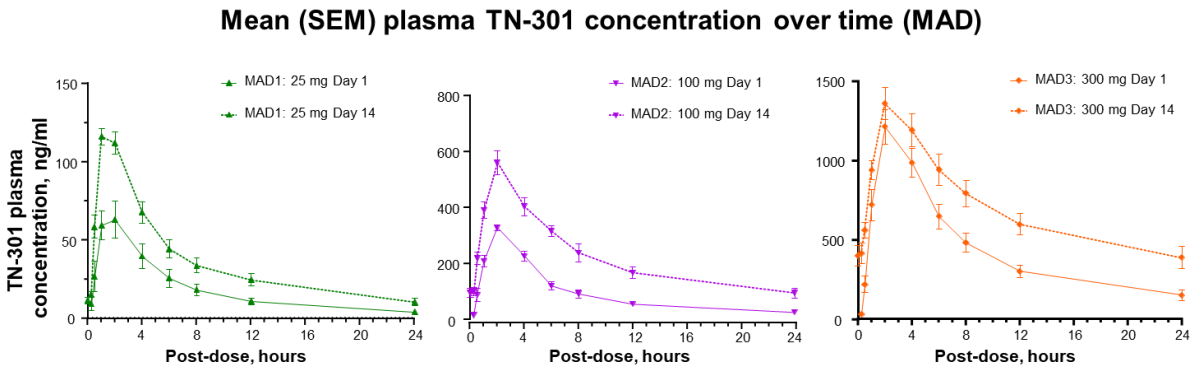
Completed Phase 1 trial of TN-301 in healthy participants

TN-301 was generally well tolerated across broad dose ranges

- Single-ascending doses (SAD) of 1mg – 700mg
- Multiple ascending doses (MAD) of 25mg, 100 mg and 300 mg for 14 days
- Most AEs observed were GI related; occurred with similar frequency in placebo group and did not increase in association with dose

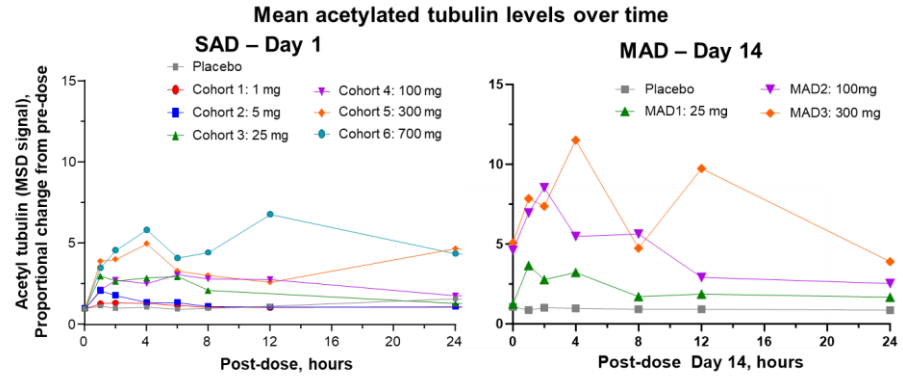
PK and half-life support once-daily dosing

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



Target engagement (as measured by tubulin acetylation) seen at low doses

Increasing TN-301 exposure correlated with PD effect



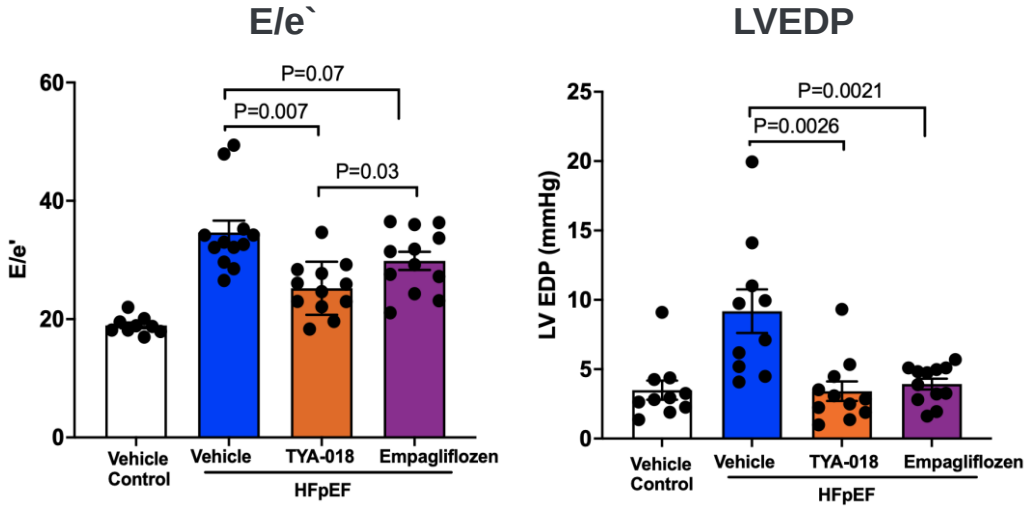
Variability (SEM) in acetylated tubulin levels ranged from 0.038 to 1.410 (SAD results); and 0.067 to 4.050 (MAD results)

AEs = Adverse events | GI = gastrointestinal

HDAC6 inhibitor demonstrates preclinical potential for use as single-agent or in combination with SGLT2 inhibitor

HDAC6 vs. SGLT2 inhibition

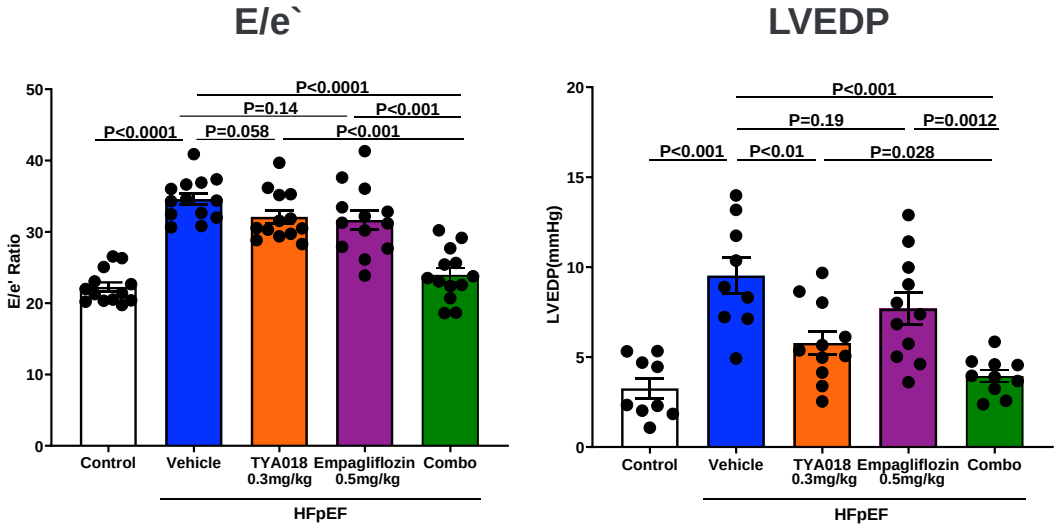
↔ Comparable efficacy as a single agent



Yang, et al; ESC-HF 2022

HDAC6 + SGLT2 inhibition

✚ Additive efficacy in combination



Farshidfar, et al; HFSA 2023

HDAC6 inhibitor demonstrates greater impact vs SGLT2 inhibtor on improving metabolism, oxidative stress and inflammation, key mechanisms involved in HFpEF pathophysiology

E/e' = measure of diastolic function LVEDP = left ventricular end diastolic pressure

TENAYA™ THERAPEUTICS

Scientific Approach and Capabilities

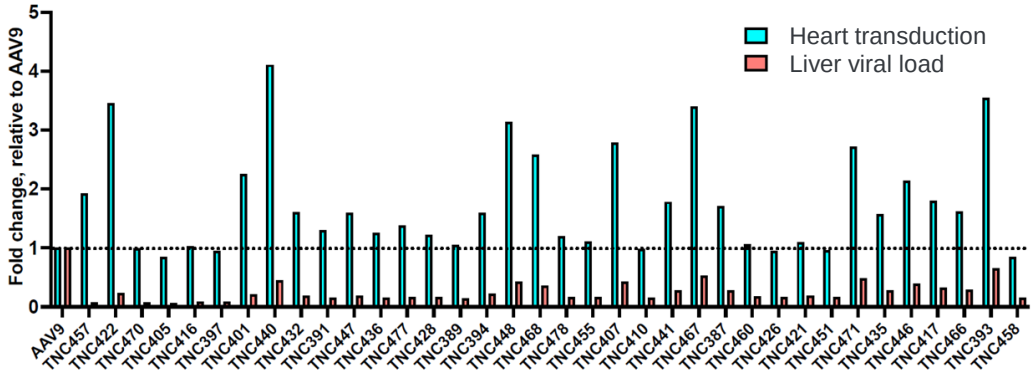
Next generation AAV capsid engineering efforts aimed at enhanced efficacy and safety

Focused AAV Screening Efforts Using Multiple Strategies

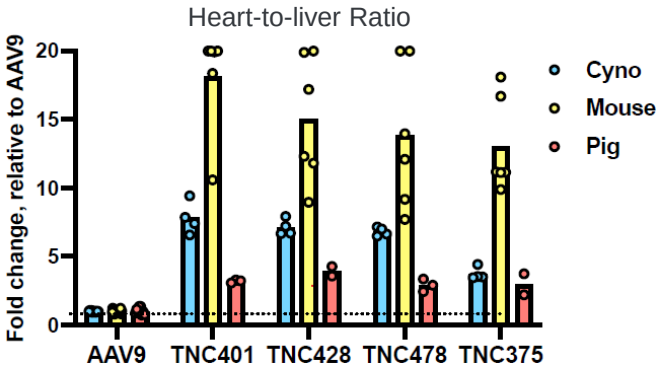
- Screened > 1B variants from ~30 diverse libraries
- Validated *in silico, in vitro* and *in vivo* (4 species)
- Multiple criteria
  - ↑ heart transduction
  - ↓ liver transduction
  - ↔ antigenicity
  - ↔ manufacturability

Novel AAV Capsids for Heart that **Outperform** Parental Vectors

2nd generation novel capsids demonstrate reduced trafficking to the liver in NHPs vs. AAV9



Top novel capsids show improved heart-to-liver ratio across 3 species



Chen, et al; ESGCT 2022

2nd Generation Capsid Characteristics

- ☑ Superior heart transduction → may lead to **more efficacious therapy**
- ☑ Superior liver de-targeting → may **improve the safety profile**
- ☑ Superior NAb evasion → may enable treatment of a **greater number of patients**

NHP = Non-Human Primate | NAb = neutralizing antibodies

End-to-end gene therapy manufacturing capabilities built to scale from early research to drug approvals

Manufacturing Technology Development Center (non-GMP)

Process Development

- Development of science to improve process and product attributes

→ IP filed on technologies to boost AAV drug product potency and yield

Analytical Development

- Development of assays to support drug substance and product release

→ Potency assays accepted by FDA for both TN-201 and TN-401

Genetic Medicines Manufacturing Center (cGMP)

- Modular design enables future expansion for commercial launch

→ Sufficient Phase 1b clinical supply available for TN-201 and TN-401

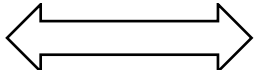
Starting materials

Shake flask

50L

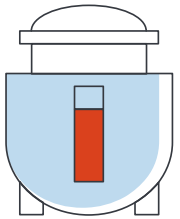
200L

Seamless transition



Continuous learning

200L



1000L

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2024 Strategic Priorities

2024 strategic priorities

Drive clinical-stage gene therapy programs towards initial data readouts

TN-201

FY – Enroll patients in MyPeak-1
2H – Release initial MyPeak-1 data

TN-401

2H – Initiate patient dosing in RIDGE-1

Research & Manufacturing

FY – Present data from early-stage research efforts and platform enhancement innovations

$104.5M as of Q4 2023 + $46.5M in net financing proceeds
Planned operations funded into 2H 2025

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Thank you