# UNITED STATES
# SECURITIES AND EXCHANGE COMMISSION
**WASHINGTON, D.C. 20549**

# FORM 8-K

**CURRENT REPORT**
**Pursuant to Section 13 or 15(d)**
**of the Securities Exchange Act of 1934**

**Date of Report (Date of earliest event reported):**
**December 11, 2025**

# Tenaya Therapeutics, Inc.
**(Exact name of registrant as specified in its charter)**

| Delaware | 001-40656 | 81-3789973 |
|---|---|---|
| (State or other jurisdiction of incorporation) | (Commission File Number) | (IRS Employer Identification No.) |

**171 Oyster Point Boulevard, Suite 500**
**South San Francisco, CA 94080**
**(Address of principal executive offices, including zip code)**

**(650) 825-6990**
**(Registrant's telephone number, including area code)**

**Not Applicable**
**(Former name or former address, if changed since last report)**

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

| Title of each class | Trading Symbol(s) | Name of exchange on which registered |
|---|---|---|
| Common Stock, par value $0.0001 per share | TNYA | Nasdaq Global Select Market |

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

**Item 7.01 Regulation FD Disclosure.**

On December 11, 2025, Tenaya Therapeutics, Inc. (the "Company") issued a press release announcing that the U.S. Food and Drug Administration ("FDA") has removed the clinical hold on its MyPEAK™-1 clinical trial of TN-201 gene therapy. The Company also issued a press release on December 11, 2025 announcing interim data from the first cohort of patients in the RIDGE™-1 clinical trial of TN-401 gene therapy. The press releases are attached hereto as Exhibits 99.1 and 99.2 to this Current Report on Form 8-K and are incorporated herein by reference.

The information in this Item 7.01, including Exhibits 99.1 and 99.2 attached hereto, shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference in any filing under the Securities Act of 1933, as amended, except as expressly set forth by specific reference in such filing.

**Item 8.01 Other Events.**

Following proactive correspondence with the FDA relating to future development plans for TN-201, on November 7, 2025, the Company announced that the FDA placed MyPEAK-1 on clinical hold requesting an amendment to the protocol primarily to standardize activities related to patient monitoring and management of the immunosuppression regimen across trial sites. The Company worked swiftly and collaboratively with the FDA to resolve the clinical hold and has received official notification from the FDA that the clinical hold has been removed. The Company intends to resume dosing once the protocol changes have been implemented at trial sites and expects to present two-year Cohort 1 and one-year Cohort 2 data in the first half of 2026. The Company does not expect this action to impact data milestones or development timelines for TN-201.

RIDGE™-1 is the Company's Phase 1b multi-center, open-label clinical trial, designed to assess the safety, tolerability and efficacy of a one-time intravenous infusion of TN-401. In December 2025, the Company presented interim data from RIDGE-1, including safety, biopsy and arrhythmia results as of the October 2025 data cut off for three patients enrolled in Cohort 1, with follow-up ranging from Week 20 to Week 40. TN-401 was generally well tolerated and no dose-limiting toxicities were observed. Adverse events ("AEs") were generally mild, asymptomatic and manageable and a majority of the AEs were deemed unrelated to TN-401. Among the AEs related to TN-401, there was a Grade 1 incidence of elevated troponin levels categorized as a serious AE due to inpatient monitoring. There were no incidents of thrombotic microangiopathy or cardiotoxicities observed and no arrhythmias associated with TN-401 occurred. To date, none of the Cohort 1 patients have experienced an implantable cardioverter defibrillator ("ICD") shock post treatment. All Cohort 1 patients have tapered off prophylactic immunosuppressive medicines. No new serious AEs related to TN-401 have been reported in Cohort 2.

Serial biopsies taken at baseline and Week 8 post dose for Patients 1 and 2 provided consistent evidence that TN-401 transduced to the heart, with vector copy number ("VCN") per genome of 3.4 and 5, respectively. VCN data for Patient 3 was not available as of the data cut off. At Week 8, TN-401 mRNA expression ranged from $1.4x10^4$ - $2.9x10^5$ copies per microgram of RNA for the three patients in Cohort 1, serving as confirmation of early and robust expression across all three patients. Post-treatment protein levels of *PKP2* increased significantly in Patients 1 and 2 by a mean of 10% from baseline to Week 8 based on rigorous methods to measure protein increases using liquid chromatography–mass spectrometry normalized to myosin heavy chain, a motor protein in the sarcomere found exclusively in cardiomyocytes. Change from baseline in *PKP2* protein levels for Patient 3 appeared slightly lower than baseline despite having the highest levels of TN-401 mRNA expression across Cohort 1. This confounding result falls within the standard deviation of these methods and may be due to the inherent variability in sampling biopsies. A second post-dose biopsy will be collected and analyzed from Week 52 per protocol.

Two of three patients experienced significant and clinically meaningful improvements in electrical instability, as measured by seven-day ambulatory monitoring of premature ventricular contractions ("PVCs"). Patient 1 experienced a decrease in PVCs by 46% as of their most recent (Week 40) visit, while Patient 2 experienced a decrease in PVCs of 89% as of their most recent (Week 32) visit.

Non-sustained ventricular tachycardias ("NSVTs") burden was eliminated or stable after treatment with TN-401 after six months post-dose. Patient 1 had a low NSVT count at baseline, which remained low at their most recent visit (Week 40). Patient 2 also had a substantial NSVT burden of 78 counts per 24 hour period at baseline that dropped all the way to zero and remained stable by Week 32. Meaningful changes in PVCs or NSVTs were not expected nor observed for Patient 3 as of the data cut-off, which was less than six months following treatment with TN-401. Other measures of clinical response including QRS duration, T wave inversions, heart function and New York Heart Association class were in the normal range or remained stable for all three Cohort 1 patients during the post-dose follow-up period. The Company expects to present one-year Cohort 1 data and early Cohort 2 data in the first half of 2026.

***Forward-Looking Statements***

This Current Report on Form 8-K contains forward-looking statements as that term is defined in Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended. Statements in this report that are not purely historical are forward-looking statements. Words such as "will," "anticipated," "believe," "look forward," "potential," and similar expressions are intended to identify forward-looking statements. Such forward-looking statements include, among other things, the planned timing to resume dosing for MyPEAK-1; the planned timing to report additional data from MyPEAK-1; the planned timing to report additional data from RIDGE-1; the clinical, therapeutic and commercial potential of, and expectations regarding TN-401; the value of additional RIDGE-1 data to inform the potential of TN-401; the inferences regarding *PKP2* protein and mRNA expression; and statements regarding the continued development TN-401 and TN-401 clinical outcomes, which may materially change as patient enrollment continues or more patient data become available. The forward-looking statements contained herein are based upon the Company's current expectations and involve assumptions that may never materialize or may prove to be incorrect. These forward-looking statements are neither promises nor guarantees and are subject to a variety of risks and uncertainties, including but not limited to: availability of RIDGE-1 data at the referenced time; the timing and progress of RIDGE-1; the potential failure of TN-401 to demonstrate safety and/or efficacy in clinical testing; the potential for any RIDGE-1 clinical trial results to differ from preclinical, interim, preliminary or expected results; the Company's ability to enroll and maintain patients in clinical trials, including RIDGE-1; risks associated with the process of discovering, developing and commercializing drugs that are safe and effective for use as human therapeutics and operating as an early stage company; the Company's continuing compliance with applicable legal and regulatory requirements; the Company's ability to raise any additional funding it will need to continue to pursue its product development plans; the Company's reliance on third parties; the Company's manufacturing, commercialization and marketing capabilities and strategy; the loss of key scientific or management personnel; competition in the industry in which the Company operates; the Company's ability to obtain and maintain intellectual property protection for its product candidates; general economic and market conditions; and other risks. Information regarding the foregoing and additional risks may be found in the section titled "Risk Factors" in the Company's Quarterly Report on Form 10-Q for the fiscal quarter ended September 30, 2025 and other documents that the Company files from time to time with the Securities and Exchange Commission. These forward-looking statements are made as of the date of this report, and the Company assumes no obligation to update or revise any forward-looking statements, whether as a result of new information, future events or otherwise, except as required by law.

**Item 9.01 Financial Statements and Exhibits.**

(d) Exhibits.

| Exhibit No. | Description. |
|---|---|
| 99.1 | Press Release dated December 11, 2025. |
| 99.2 | Press Release dated December 11, 2025. |
| 104 | Cover Page Interactive Data File (embedded within the Inline XBRL document) |

**SIGNATURES**

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

**TENAYA THERAPEUTICS, INC.**

By: /s/ Jennifer Drimmer Rokovich
Jennifer Drimmer Rokovich
General Counsel and Secretary

Date: December 11, 2025

**Tenaya Therapeutics Announces Rapid Resolution and Lifting of Clinical Hold for MyPEAK-1™ Phase 1b/2a Clinical Trial of TN-201 Gene Therapy**

*MyPEAK-1 Protocol Amendments Agreed Upon with FDA; Tenaya Implementing Changes with Sites*

**SOUTH SAN FRANCISCO, Calif., December 11, 2025 —** Tenaya Therapeutics, Inc. (NASDAQ: TNYA), a clinical-stage biotechnology company with a mission to discover, develop and deliver potentially curative therapies that address the underlying causes of heart disease, today announced it has received official notification from the U.S. Food and Drug Administration (FDA) that the clinical hold on the MyPEAK-1™ Phase 1b/2a clinical trial of TN-201 has been removed. All concerns raised by the FDA related to the clinical hold have been addressed. TN-201 is being developed for the potential treatment of Myosin Binding Protein C3 (*MYBPC3*)-associated hypertrophic cardiomyopathy (HCM), a condition caused by insufficient levels of myosin-binding protein C (MyBP-C).

Tenaya is currently implementing amendments to the study protocol in collaboration with MyPEAK-1 clinical sites after which the company plans to resume dosing. The protocol changes standardize practices adopted in the trial to optimize patient monitoring and management of the immunosuppressive regimen. The immunosuppression regimen of prophylactic prednisone and sirolimus remains unchanged. The protocol amendment formalizes the company's learnings from the timing and dosing of these agents, which enabled shorter durations and lower cumulative doses of these immunosuppressants between cohorts, despite the higher TN-201 dose. These findings, as well as data from Cohort 1 patients at ≥52-weeks of follow-up and available data for Cohort 2 patients at 12- and 26-weeks, were recently featured in a late-breaker presentation at the American Heart Association Scientific Sessions with a simultaneous publication in *Cardiovascular Research*.

To date, TN-201 has been generally well tolerated and the MyPEAK-1 data and safety monitoring board (DSMB) endorsed continued enrollment of the trial following a review of all available safety data this summer. There have been no new safety findings of concern since. The company does not expect the hold to impact data milestones or development timelines.

**About the MyPEAK-1 Phase 1b/2a Clinical Trial**

The MyPEAK-1 Phase 1b/2a clinical trial (Clinicaltrials.gov ID: NCT05836259) is a multi-center, open-label, dose-escalating (3E13 vg/kg and 6E13 vg/kg) study of symptomatic adults (up to 24) who have been diagnosed with *MYBPC3*-associated HCM. MyPEAK-1 is designed to assess the safety, tolerability and clinical efficacy of a one-time intravenous infusion of TN-201 gene replacement therapy. MyPEAK-1 has tested doses of 3E13 vg/kg and 6E13 vg/kg in two cohorts of three patients each and is enrolling additional *MYBPC3*-positive adults with either the nonobstructive or obstructive form of HCM in dose expansion cohorts.

To learn more about gene therapy for HCM and participation in the MyPEAK-1 study, please visit HCMStudies.com.

**About *MYBPC3*-Associated Hypertrophic Cardiomyopathy**

Variants in the Myosin Binding Protein C3 (*MYBPC3*) gene are the most common genetic cause of hypertrophic cardiomyopathy (HCM), accounting for approximately 20% of the overall HCM population, or 120,000 patients, in the United States alone. *MYBPC3*-associated HCM is a severe and progressive condition affecting adults, teens, children and infants. Mutations of the *MYBPC3* gene result in insufficient expression of a protein, called MyBP-C, needed to regulate heart contraction. The heart becomes hypercontractile and the left ventricle thickens, resulting in symptoms such as chest pain, shortness of breath, palpitations and fainting. Patients whose disease is caused by *MYBPC3* mutations are more likely than those with non-genetic forms of HCM to experience earlier disease onset and have high rates of serious outcomes, including heart failure symptoms, arrhythmias, stroke and sudden cardiac arrest or death. There are currently no approved therapeutics that address the underlying genetic cause of HCM.

**About TN-201**

TN-201 is an adeno-associated virus serotype 9 (AAV9)-based gene therapy designed address the underlying cause of *MYBPC3*-associated HCM by delivering a working *MYBPC3* gene to heart muscle cells via a single intravenous infusion and thereby increasing insufficient MyBP-C protein levels with the aim of halting or even reversing disease after a single dose. The U.S. Food and Drug Administration has granted TN-201 Fast Track, Orphan Drug and Rare Pediatric Drug Designations. TN-201 has also received orphan medicinal product designation from the European Commission.

**About Tenaya Therapeutics**

Tenaya Therapeutics is a clinical-stage biotechnology company committed to a bold mission: to discover, develop and deliver potentially curative therapies that address the underlying drivers of heart disease. Tenaya's pipeline includes clinical-stage candidates TN-201, a gene therapy for *MYBPC3*-associated hypertrophic cardiomyopathy (HCM) and TN-401, a gene therapy for *PKP2*-associated arrhythmogenic right ventricular cardiomyopathy (ARVC). Tenaya has employed a suite of integrated internal capabilities, including modality agnostic target validation, capsid engineering and manufacturing, to generate a portfolio of novel medicines based on genetic insights, including TN-301, a clinical-stage small molecule HDAC6 inhibitor for the potential treatment of heart failure and related cardio/muscular disease, and multiple early-stage programs in preclinical development aimed at the treatment of both rare genetic disorders and more prevalent heart conditions. For more information, visit www.tenayatherapeutics.com.

**Forward Looking Statements**

This press release contains forward-looking statements as that term is defined in Section 27A of the Securities Act of 1933 and Section 21E of the Securities Exchange Act of 1934. Statements in this press release that are not purely historical are forward-looking statements. Words such as "expect," and similar expressions are intended to identify forward-looking statements. Such forward-looking statements include, among other things, Tenaya's expectation that the clinical hold will not impact data milestones or development timelines. The forward-looking statements contained herein are based upon Tenaya's current expectations and involve assumptions that may never materialize or may prove to be incorrect. These forward-looking statements are neither promises nor guarantees and are subject to a variety of risks and uncertainties, including but not limited to: Tenaya's ability to successfully implement protocol changes for MyPEAK-1; the potential progress of MyPEAK-1; the potential failure of TN-201 to demonstrate safety and/or efficacy in clinical testing; the potential for any MyPEAK-1 clinical trial results to differ from preclinical, interim, preliminary or expected results; the potential for the FDA and/or other regulatory agencies to conclude at any time that TN-201 may not have an appropriate risk/benefit profile; Tenaya's ability to enroll and maintain patients in MyPEAK-1; risks associated with the process of discovering, developing and commercializing drugs that are safe and effective for use as human therapeutics and operating as an early stage company; Tenaya's continuing compliance with applicable legal and regulatory requirements; Tenaya's ability to raise any additional funding it will need to continue to pursue its product development plans; Tenaya's reliance on third parties; Tenaya's manufacturing, commercialization and marketing capabilities and strategy; the loss of key scientific or management personnel; competition in the industry in which Tenaya operates; Tenaya's ability to obtain and maintain intellectual property protection for its product candidates; general economic and market conditions; and other risks. Information regarding the foregoing and additional risks may be found in the section titled "Risk Factors" in Tenaya's Quarterly Report on Form 10-Q for the fiscal quarter ended September 30, 2025, and other documents that Tenaya files from time to time with the Securities and Exchange Commission. These forward-looking statements are made as of the date of this press release, and Tenaya assumes no obligation to update or revise any forward-looking statements, whether as a result of new information, future events or otherwise, except as required by law.

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**Tenaya Therapeutics Reports Positive Interim Data from Cohort 1 of RIDGE™-1 Phase 1b/2 Clinical Trial of TN-401 Gene Therapy for Adults with *PKP2*-associated ARVC**

*TN-401 was Well Tolerated at 3E13 vg/kg dose*

*Robust Transduction and Demonstrated Increases in PKP2 Protein Levels in First Two Patients at Week 8*

*Clinically Meaningful Reductions in Arrhythmia Burden Observed in First Two Patients with More Than Six Months of Follow-Up*

*Tenaya Management to Host a Conference Call Thursday, December 11, 2025 at 5:00 p.m. ET to Review Preliminary Results*

**South San Francisco, Calif., December 11, 2025 –** Tenaya Therapeutics, Inc. (NASDAQ: TNYA), a clinical-stage biotechnology company with a mission to discover, develop and deliver potentially curative therapies that address the underlying causes of heart disease, today announced interim data from the ongoing RIDGE™-1 Phase 1b/2 clinical trial of TN-401 gene therapy for the potential treatment of adults with arrhythmogenic right ventricular cardiomyopathy (ARVC), a form of arrhythmogenic cardiomyopathy (ACM) that primarily impacts the right ventricle, caused by mutations in the plakophilin-2 gene, *PKP2*.

Data reported today include safety, biopsy and arrhythmia results from three patients who received TN-401 at a dose of 3E13 vg/kg. Patient follow-up at the time of data cut off ranged from 20-40 weeks post-dose. TN-401 was well tolerated, increased PKP2 protein expression from baseline in two of three patients and demonstrated evidence of meaningful improvements in arrhythmia burden as measured by premature ventricular contractions (PVCs) and non-sustained ventricular tachycardias (NSVTs) for those patients with greater than six months follow up.

“We are excited by the strength of the data for TN-401 at this relatively early timepoint in the RIDGE-1 trial. Less than a year after dosing, initial data indicate a promising safety profile, consistent transduction of the gene therapy in cardiomyocytes and RNA and protein expression, and meaningful reductions in PVCs and NSVTs, well-established risk factors for dangerous sustained arrhythmias,” said Whit Tingley, M.D., Ph.D., Tenaya’s Chief Medical Officer. “These findings are an important milestone in TN-401’s development and we’re eager to build on this momentum as we continue monitoring patients in Cohort 1 and Cohort 2.”

*PKP2* gene mutations result in insufficient levels of critical proteins needed to maintain the structural integrity and cell-to-cell signaling of heart muscle cells. TN-401 gene replacement therapy is designed to address the underlying cause of disease by delivering a functional *PKP2* gene into heart muscle cells using an adeno associated virus serotype 9 (AAV9) capsid. The RIDGE-1 clinical trial designed to assess the safety, tolerability and activity of a one-time dose of TN-401 at two dose levels, 3E13 vg/kg and 6E13 vg/kg. To date, Tenaya has dosed three patients at the 3E13 vg/kg dose level (Cohort 1). Initial results reported today focus on interim data for Cohort 1 as of an October 2025 data cut off. Key findings include:

- **TN-401 was well tolerated at the 3E13 vg/kg dose and no dose-limiting toxicities were observed**
    - Adverse events (AEs) were generally mild, asymptomatic and manageable and deemed unrelated to TN-401 treatment.

- Among those AEs related to treatment, there was a transient, asymptomatic Grade 1 event of elevated cardiac troponin levels categorized as a serious AE (SAE) only due to inpatient monitoring
- No incidents of thrombotic microangiopathy (TMA) or cardiotoxicities were observed.
- To date, no implantable cardioverter-defibrillator (ICD) shocks or arrhythmias associated with TN-401 have occurred.
- All patients have tapered off immunosuppressive medicines.

Enrollment and dosing of three patients at the 6E13 vg/kg dose (Cohort 2) is complete with no new SAEs related to TN-401 reported in the cohort to date.

- **Biopsies demonstrate robust transduction and expression detected in all patients within first eight weeks**
  - Patients 1 and 2 provided consistent evidence that TN-401 transduced the heart (3.4 vg/dg and 5.0 vg/dg). A biopsy analysis of TN-401 DNA for Patient 3 was not available at the time of data cut off.
  - Consistently high TN-401 mRNA expression ranging from 1.4x104—2.9x105 copies per microgram of RNA were detected for all three patients in Cohort 1 as early as eight weeks.
  - Post-treatment protein levels of PKP2 increased significantly in Patients 1 and 2 by a mean of 10% from baseline to Week 8
    - These data are based on rigorous methods to measure protein increases using liquid chromatography–mass spectrometry (LC-MS) normalized to myosin heavy chain, a motor protein in the sarcomere found exclusively in cardiomyocytes.
    - Change from baseline in PKP2 protein levels for Patient 3 fell within the standard deviation of these methods despite having the highest levels of TN-401 mRNA expression across Cohort 1, which may be due to the inherent variability in sampling biopsies. A second post-dose biopsy will be collected and analyzed from Week 52 per protocol.
  - The changes in PKP2 protein levels were also apparent using multiplexed immunofluorescent imaging, which provided visual evidence of protein level increases and colocalization of other proteins associated with intracellular stability and electrical signaling.
- **Clinically meaningful improvements in electrical instability were observed in the first two patients with greater than 6 months follow-up after TN-401 dosing**
  - Patient 1 PVC counts decreased by 46% as of their most recent (Week 40) visit, while Patient 2 PVC counts decreased by 89% by their most recent visit (Week 32).
  - Patient 2 also had a substantial NSVT burden of 78 counts per 24-hour period at baseline that dropped all the way to zero and remained stable by Week 32. Patient 1 had low NSVT count at baseline which remained low at Week 40.
  - For Patient 3, meaningful changes in PVCs or NSVTs were not expected nor observed as of the data cut off, which was less than six months following treatment with TN-401.
  - Other measures of clinical response including QRS duration, T wave inversions, heart function, and New York Heart Association (NYHA) class were in the normal range or remained stable for all three Cohort 1 patients during the post-dose follow-up period.

“We extend our heartfelt gratitude to the patients and investigators involved in the RIDGE-1 clinical trial and our RIDGE natural history study of *PKP2*-associated ARVC. Their continued support and dedication have been instrumental in achieving the substantial progress we’ve made thus far,” continued Dr. Tingley.

**Webcast Conference Call**

Tenaya management will host a conference call on Thursday, December 11, 2025, at 5:00 pm ET/2:00 pm PT to discuss the TN-401 data shared today. The webcast conference call, including an accompanying slide presentation, can be accessed from the Investor section on the “Events and Presentations” page of the Tenaya website at www.tenayathera.com.

**About *PKP2*-Associated ARVC**

*Plakophilin-2* (*PKP2*) mutations are the most common genetic cause of arrhythmogenic right ventricular cardiomyopathy (ARVC, also known as arrhythmogenic cardiomyopathy or ACM), occurring in approximately 40 percent of the overall ARVC population. The prevalence of *PKP2*-associated ARVC is estimated at more than 70,000 people in the U.S. alone.

In *PKP2*-associated ARVC, mutations of the *PKP2* gene results in insufficient expression of a protein needed for the proper functioning of the desmosomal complex that maintains physical connections and electrical signaling between heart muscle cells. As the desmosome structure degrades, cardiac muscle cells are replaced by fibrofatty tissue and electrical pulses in the heart become unstable, resulting in irregular heart rhythms. ARVC symptoms include arrhythmias, palpitations, lightheadedness, dizziness and fainting. It is typically diagnosed before age 40, and sudden cardiac arrest due to life-threatening ventricular arrhythmias is frequently the first manifestation of disease. Current treatments include anti-arrhythmic medications, implantable cardioverter-defibrillators (ICDs) and ablation procedures, which do not address the underlying genetic cause of disease.

**About TN-401 Gene Therapy and the RIDGE-1 Clinical Trial**

TN-401 is an investigational AAV9-based gene therapy being developed for the treatment of ARVC due to mutations in the *PKP2* gene. AAV9 was selected as the vector for delivery of Tenaya's *PKP2* gene therapy based on its extensive clinical and commercial safety record and demonstrated ability to target heart muscle cells. TN-401 has received Orphan Drug and Fast Track Designations from the U.S. Food and Drug Administration. Tenaya's development of TN-401 is supported in part by a grant from the California Institute of Regenerative Medicines (CIRM).

The RIDGE-1 Phase 1b/2 clinical trial of TN-401 in patients with *PKP2*-associated ARVC is a multi-center, open-label, dose escalation study being conducted in the U.S. and UK. RIDGE-1 is intended to assess the safety, tolerability and preliminary clinical efficacy of a one-time intravenous infusion of TN-401. RIDGE-1 will seek to enroll up to fifteen adults who have been diagnosed with *PKP2*-associated ARVC, have an ICD and are at increased risk for arrhythmias as determined by premature ventricular count (PVC) during screening.

To learn more about gene therapy for ARVC and the RIDGE-1 clinical trial, please visit ARVCstudies.com or ClinicalTrials.gov (NCT06228924).

**About Tenaya Therapeutics**

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and maintain patients in clinical trials, including RIDGE-1; risks associated with the process of discovering, developing and commercializing drugs that are safe and effective for use as human therapeutics and operating as an early stage company; Tenaya's continuing compliance with applicable legal and regulatory requirements; Tenaya's ability to raise any additional funding it will need to continue to pursue its product development plans; Tenaya's reliance on third parties; Tenaya's manufacturing, commercialization and marketing capabilities and strategy; the loss of key scientific or management personnel; competition in the industry in which Tenaya operates; Tenaya's ability to obtain and maintain intellectual property protection for its product candidates; general economic and market conditions; and other risks. Information regarding the foregoing and additional risks may be found in the section titled "Risk Factors" in Tenaya's Quarterly Report on Form 10-Q for the fiscal quarter ended September 30, 2025 and other documents that Tenaya files from time to time with the Securities and Exchange Commission. These forward-looking statements are made as of the date of this press release, and Tenaya assumes no obligation to update or revise any forward-looking statements, whether as a result of new information, future events or otherwise, except as required by law.

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