NEWS RELEASE

Cytokinetics Announces Four Presentations at the American College of Cardiology Annual Scientific Session & Expo

2026-03-16

New Analyses from Three Trials in Obstructive HCM Support Findings from Previously Published Data on MYQORZO™ (aficamten)

SOUTH SAN FRANCISCO, Calif., March 16, 2026 (GLOBE NEWSWIRE) -- Cytokinetics, Incorporated (Nasdaq: CYTK) today announced four presentations related to MYQORZO™ *(aficamten)* at the American College of Cardiology (ACC) Annual Scientific Session & Expo taking place March 28–30, 2026 in New Orleans, LA. Recently approved for the treatment of adults with symptomatic obstructive hypertrophic cardiomyopathy (oHCM) by the U.S. Food and Drug Administration, European Commission, and the China National Medical Products Administration, MYQORZO is an allosteric and reversible inhibitor of cardiac myosin motor activity. In patients with oHCM, myosin inhibition with MYQORZO reduces cardiac contractility and consequently, left ventricular outflow tract (LVOT) obstruction.

“We are pleased to contribute further evidence that we believe will help physicians and patients with clinical decision making,” said Stephen Heitner, M.D., Senior Vice President, Clinical Research and Development. “The new data can help empower evidence-based choices of therapy for patients living with symptomatic oHCM, showing that *aficamten* improves exercise capacity, when compared to placebo or the beta blocker, metoprolol, while maintaining its safety profile.”

Scientific Presentations Include:

Evaluation of *Aficamten* or Beta-Blocker Monotherapy Versus Placebo in Patients with Obstructive Hypertrophic Cardiomyopathy: A Pooled Analysis of SEQUOIA-HCM and MAPLE-HCM (1186-09)
Moderated Poster Presentation, March 30,2026, 11:00 AM-12:00 PM CT, E Hall
P. Christian Schulze, M.D., Ph.D., Chair of the Department of Medicine and Division of Cardiology, Angiology and Intensive Medical Care at the University Hospital Jena

A new analysis based on combined data from SEQUOIA-HCM (**S**afety, **E**fficacy, and Quantitative **U**nderstanding of **O**bstruction **I**mpact of ***A**ficamten* in **HCM**) and MAPLE-HCM (***M**etoprolol* vs ***A**ficamten* in **P**atients with **L**VOT Obstruction on **E**xercise Capacity in HCM) evaluated *aficamten* or

metoprolol monotherapy versus placebo. Patients (n=371) were pooled into three groups: *aficamten* as monotherapy, *metoprolol* as monotherapy, and placebo without background beta-blockers. For patients with symptomatic oHCM, treatment with beta-blocker monotherapy was no different compared to placebo across many clinically relevant outcomes (exercise capacity, symptoms, cardiac biomarkers and Valsalva gradient). Conversely, *aficamten* as monotherapy was superior to both *metoprolol* and placebo, consistent with previously published[1] findings and supporting the emerging role of *aficamten* in the treatment of symptomatic oHCM.

Clinical Implications Associated with Temporary Treatment Interruption and Reinitiation of *Aficamten* Therapy in Obstructive Hypertrophic Cardiomyopathy (906-11)
Oral Presentation, March 29, 2026, 9:30 AM-10:30 AM CT, Hall E
Martin Maron, M.D., Director, Hypertrophic Cardiomyopathy Center, Lahey Hospital & Medical Center; and National Principal Investigator of SEQUOIA-HCM

This new analysis evaluated data from SEQUOIA-HCM and FOREST-HCM (**F**ollow-up, **O**pen-Label, **R**esearch **E**valuation of **S**ustained **T**reatment with *Aficamten* in **HCM**) related to the safety of interrupting treatment with *aficamten.* The analysis, which included 182 participants, evaluated the impact of discontinuing treatment with *aficamten* for a four-week washout period after assessment of the primary endpoint at 24-weeks as occurred per protocol in SEQUOIA-HCM. Most of these patients subsequently reinitiated treatment in the open-label extension, FOREST-HCM. The analyses showed that washout of *aficamten* was not associated with increased risk of cardiac adverse events or rebound compared to placebo. One (0.7%) patient in the *aficamten* arm experienced an adverse event of mild worsening heart failure due to an acute drop in hemoglobin. Three patients experienced recurrence of HCM symptoms of moderate severity after discontinuing *aficamten* consistent with the loss of therapeutic drug effect during washout. Importantly, reinitiation of *aficamten* yielded a favorable therapeutic response, suggesting *aficamten* can be safely discontinued if treatment interruptions are required for non-cardiac medical reasons, such as surgery or cancer treatment.

***Aficamten* Versus Metoprolol in Patients with Hypertension and Obstructive Hypertrophic Cardiomyopathy (1395-221)**
Poster Presentation, March 28, 2026, 2PM-3PM CT, Hall E
Ahmad Masri, M.D., MS, Director of the Hypertrophic Cardiomyopathy Center at Oregon Health & Science University

A new analysis of MAPLE-HCM evaluated the efficacy and safety of treatment with *aficamten* vs. *metoprolol* in patients with hypertension at baseline, which is common in patients with oHCM. In this analysis of 175 patients, 50% had a history of hypertension, and 81% were treated with a beta-blocker and/or calcium channel blocker before randomization in MAPLE-HCM. While blood pressure increased slightly with *aficamten* (+3.6±14.0 mmHg) compared to a decrease with *metoprolol*, (−8.3±18.2 mmHg), the analysis showed there was no statistically significant difference in the rates of uncontrolled hypertension, defined as systolic blood pressure (SBP) > 140 mmHg or diastolic blood pressure (DBP) > 90 mmHg emergent during treatment (34% vs. 25% in the *aficamten* and metoprolol arms respectively; p=0.38). *Aficamten* was equally effective and displayed a similar safety profile irrespective of hypertension history. These findings suggest *aficamten* is suitable as therapy independent of hypertension history and given the decrease in LVOT gradient during treatment with *aficamten*, may allow for greater optionality in the choice of antihypertensive agents.

Electrocardiographic Changes and Associations with Echocardiographic Changes in Patients with Symptomatic Obstructive Hypertrophic Cardiomyopathy: Insights from the SEQUOIA-HCM Trial (1514-243)
Poster Presentation, March 29, 2026, 2PM-3PM CT, Hall E
Alberto Foà, M.D., Ph.D., Cardiologist, Brigham and Women's Hospital, Harvard Medical School; Heart

Failure and Transplant Unit - IRCCS Azienda Ospedaliero-Universitaria di Bologna

A new analysis of SEQUOIA-HCM examined electrocardiographic (ECG) changes in patients with symptomatic oHCM following 24 weeks of treatment with *aficamten* or placebo. The results showed that, compared to placebo, *aficamten* decreased the presence of ST segment changes (adjusted odds ratio 0.23; 95% CI 0.11, 0.46; p<0.001) and left ventricular hypertrophy (LVH) strain pattern (adjusted OR 0.15; 95% CI 0.06, 0.41; p<0.001) evident on the baseline ECG. The effect *aficamten* on LVOT gradient was similar regardless of baseline LVH strain pattern.

About MYQORZO® (*aficamten*)

MYQORZO® (*aficamten*) is a cardiac myosin inhibitor approved in the U.S., China and European Union for the treatment of symptomatic obstructive hypertrophic cardiomyopathy (oHCM). In patients with oHCM, myosin inhibition with MYQORZO reduces cardiac contractility and consequently, left ventricular outflow tract (LVOT) obstruction. MYQORZO was engineered to achieve a predictable exposure response, rapid onset of action and reversibility.[2]

Aficamten is also under clinical investigation in ACACIA-HCM, a Phase 3 trial in patients with non-obstructive HCM (nHCM) and CEDAR-HCM, in a pediatric population with oHCM. *Aficamten* has not been deemed safe or effective for use in either of these patient populations. In addition, *aficamten* is being studied in FOREST-HCM, an open-label extension clinical study.

INDICATIONS AND USAGE

MYQORZO is indicated for the treatment of adults with symptomatic oHCM to improve functional capacity and symptoms.

IMPORTANT SAFETY INFORMATION

WARNING: RISK OF HEART FAILURE

MYQORZO reduces left ventricular ejection fraction (LVEF) and can cause heart failure due to systolic dysfunction.

Echocardiogram assessments are required prior to and during treatment with MYQORZO to monitor for systolic dysfunction. Initiation of MYQORZO in patients with LVEF <55% is not recommended. Decrease the dose of MYQORZO if LVEF is <50% and ≥40%. Interrupt the dose of MYQORZO if LVEF <40% or if the patient experiences heart failure symptoms or worsening clinical status due to systolic dysfunction.

Because of the risk of heart failure due to systolic dysfunction, MYQORZO is available only through a restricted program under a Risk Evaluation and Mitigation Strategy (REMS) called the MYQORZO REMS Program.

CONTRAINDICATIONS

MYQORZO is contraindicated with concomitant use of rifampin.

WARNING AND PRECAUTIONS

Heart Failure

MYQORZO reduces cardiac contractility, which can reduce LVEF and cause heart failure.
Patients who experience a serious intercurrent illness (eg, serious infection) or arrhythmia (eg, new or uncontrolled atrial fibrillation) may be at greater risk of developing systolic dysfunction and heart failure.

Assess patients' clinical status and LVEF prior to and during treatment and adjust the MYQORZO dose accordingly. New or worsening arrhythmia, dyspnea, chest pain, fatigue, leg edema, or elevations in N-terminal pro-B-type natriuretic peptide may be signs and symptoms of heart failure.

Initiation of MYQORZO in patients with LVEF <55% is not recommended.

MYQORZO REMS Program

MYQORZO is available only through a restricted program called the MYQORZO REMS Program, because of the risk of heart failure due to systolic dysfunction.

Notable requirements of the MYQORZO REMS Program include:

- Prescribers must be certified by enrolling in the MYQORZO REMS Program
- Patients must enroll in the MYQORZO REMS Program and comply with ongoing monitoring requirements
- Pharmacies must be certified by enrolling in the MYQORZO REMS Program and must only dispense to patients who are authorized to receive MYQORZO
- Wholesalers and distributors must only distribute to certified pharmacies

Further information is available at www.MYQORZOREMS.com, or at 1-844-285-7367.

Cytochrome P450 Interactions Leading to Heart Failure or Loss of Effectiveness

MYQORZO is metabolized primarily by CYP2C9, and to a lesser extent by CYP3A, CYP2D6, and CYP2C19 enzymes. Initiation of medications that inhibit multiple P450 pathways of MYQORZO elimination (eg, fluconazole, voriconazole, or fluvoxamine) or strong CYP2C9 inhibitors, and discontinuation of moderate-to-strong CYP3A inducers may lead to increased blood concentrations of *aficamten* and increase the risk of heart failure due to systolic dysfunction. Conversely, initiation of medications that induce P450 pathways of MYQORZO (eg, rifampin, moderate-to-strong CYP3A inducers) may lead to decreased blood concentrations of *aficamten* and potential loss of effectiveness. Assess LVEF 2 to 8 weeks after initiation of such inhibitors or after discontinuation of such inducers and adjust the dose of MYQORZO accordingly.

Advise patients of the potential for drug interactions. Advise patients to inform their healthcare provider of all concomitant medications prior to and during MYQORZO treatment.

ADVERSE REACTIONS

Hypertension (8% vs 2%) was the only adverse reaction occurring in >5% of patients and more commonly on MYQORZO than on placebo in the pivotal trial.

Please see full Prescribing Information, including Boxed WARNING and Medication Guide.

About Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is a disease in which the heart muscle becomes abnormally thick. HCM can be obstructive, when thickened muscle blocks blood flow, or non-obstructive, when blood flow is not blocked but heart function is still affected. In obstructive HCM, the thickening of cardiac muscle leads to the inside of the left ventricle becoming smaller, stiffer and less able to relax and fill with blood. Ultimately, HCM limits the heart's pumping function, leading to reduced exercise capacity and a variety of symptoms.

HCM is the most common monogenic inherited cardiovascular disorder, with well over 300,000 patients diagnosed in the U.S.[3] However, there are an estimated 400,000-800,000 additional patients who remain undiagnosed.[4,5,6] Approximately half of patients with HCM have obstructive HCM (oHCM) and half have non-obstructive HCM (nHCM).[3]

People with HCM are at high risk of also developing cardiovascular complications including atrial fibrillation, stroke and mitral valve disease.[7] People with HCM are at risk for potentially fatal ventricular arrhythmias and it is one of the leading causes of sudden cardiac death in younger people or athletes.[8] A subset of patients with HCM are at high risk of progressive disease leading to dilated cardiomyopathy and heart failure necessitating cardiac transplantation.

About Cytokinetics

Cytokinetics is a specialty cardiovascular biopharmaceutical company, building on its over 25 years of pioneering scientific innovations in muscle biology, and advancing a pipeline of potential new medicines for patients suffering from diseases of cardiac muscle dysfunction. Cytokinetics' MYQORZO™ (*aficamten*) is a cardiac myosin inhibitor approved in the U.S., Europe and China for the treatment of adults with symptomatic obstructive hypertrophic cardiomyopathy (oHCM). *Aficamten* is also being studied for the potential treatment of non-obstructive HCM. Cytokinetics is also developing *omecamtiv mecarbil*, an investigational cardiac myosin activator for the potential treatment of patients with heart failure with severely reduced ejection fraction and *ulacamten*, an investigational cardiac myosin inhibitor for the potential treatment of heart failure with preserved ejection fraction, while continuing pre-clinical research and development in muscle biology.

For additional information about Cytokinetics, visit www.cytokinetics.com and follow us on X, LinkedIn, Facebook and YouTube.

Forward-Looking Statements

This press release contains forward-looking statements for purposes of the Private Securities Litigation Reform Act of 1995 (the "Act"). Cytokinetics disclaims any intent or obligation to update these forward-looking statements and claims the protection of the Act's safe harbor for forward-looking statements. Examples of such statements include, but are not limited to, statements, express or implied, related to Cytokinetics' research and development activities; clinical trial initiation, design, enrollment, conduct, progress, continuation, completion, timing and results; regulatory submissions, review processes, approval timing and outcomes, including with respect to supplemental applications and approvals in jurisdictions outside the United States; the scope, expansion, modification, durability or continuation of labeling and promotional claims; commercial readiness, launch timing, market access and reimbursement; anticipated patient, prescriber and payer adoption; expectations regarding market opportunity, growth and market share; pipeline development and expansion into additional indications

or geographies; access to and use of capital; and Cytokinetics' business strategy, objectives and future plans. Such statements are based on management's current expectations and assumptions; however, actual results may differ materially due to various risks and uncertainties, including, but not limited to, uncertainties inherent in drug development and commercialization; the timing, conduct and outcomes of clinical trials; regulatory review and approval processes in the United States and other jurisdictions; differences in regulatory requirements, labeling, market access or promotional restrictions across jurisdictions; the ability to obtain, expand, maintain or continue desired labeling, promotional claims or commercial positioning for approved products; potential legal, intellectual property or regulatory constraints affecting commercialization and marketing claims; patient and prescriber acceptance of MYQORZO as compared to alternative therapies; the availability and terms of reimbursement from commercial and government payers; manufacturing, supply and distribution risks; competition; and the availability of sufficient capital to execute Cytokinetics' business plans. These forward-looking statements speak only as of the date they are made, and Cytokinetics undertakes no obligation to subsequently update any such statement, except as required by law. For further information regarding these and other risks related to Cytokinetics' business, investors should consult Cytokinetics' filings with the Securities and Exchange Commission (the "SEC").

Contact:
Cytokinetics
Diane Weiser
Senior Vice President, Corporate Affairs
(415) 290-7757

References

1. Maron, MS, et al. *Aficamten* for Symptomatic Obstructive Hypertrophic Cardiomyopathy. N Engl J Med. doi:10.1056/NEJMoa2401424
2. Hartman JJ, Hwee DT, Roebrt-Paganin J, et al. *Aficamten* is a small-molecule cardiac myosin inhibitor designed to treat hypertrophic cardiomyopathy. *Nat Cardiovasc Res.* 2024;3(8) :1003-1016. doi:10.1038/s44161-024-00505-0
3. Butzner M, et al. Epidemiology of Hypertrophic Cardiomyopathy in the United States From 2016 to 2023. JACC Adv. 2026. 2026;5(2):102552. doi:10.1016/j.jacadv.2025.102552
4. CVrg: Heart Failure 2020-2029, p 44; Maron et al. 2013 doi:10.1016/S0140-6736(12)60397-3; Maron et al 2018 10.1056/NEJMra1710575
5. Symphony Health 2016-2021 Patient Claims Data DoF;
6. Maron MS, Hellawell JL, Lucove JC, Farzaneh-Far R, Olivotto I. Occurrence of Clinically Diagnosed Hypertrophic Cardiomyopathy in the United States. Am J Cardiol. 2016; 15;117(10):1651-1654.
7. Gersh, B.J., Maron, B.J., Bonow, R.O., Dearani, J.A., Fifer, M.A., Link, M.S., et al. 2011 ACCF/AHA guidelines for the diagnosis and treatment of hypertrophic cardiomyopathy. A report of the American College of Cardiology Foundation/American Heart Association Task Force on practice guidelines. Journal of the American College of Cardiology and Circulation, 58, e212-260.
8. Hong Y, Su WW, Li X. Risk factors of sudden cardiac death in hypertrophic cardiomyopathy. Current Opinion in Cardiology. 2022 Jan 1;37(1):15-21

Source: Cytokinetics, Incorporated