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): April 2, 2022

Cytokinetics, Incorporated
(Exact Name of Registrant as Specified in Charter)

Delaware	**000-50633**	**94-3291317**
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(I.R.S. Employer Identification Number)

350 Oyster Point Boulevard, South San Francisco, California 94080
(Address of Principal Executive Offices) (Zip Code)

(650) 624-3000

(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:

☐ 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 each exchange on which registered
Common Stock, par value $0.001	**CYTK**	**The Nasdaq Stock Market LLC**

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. ☐

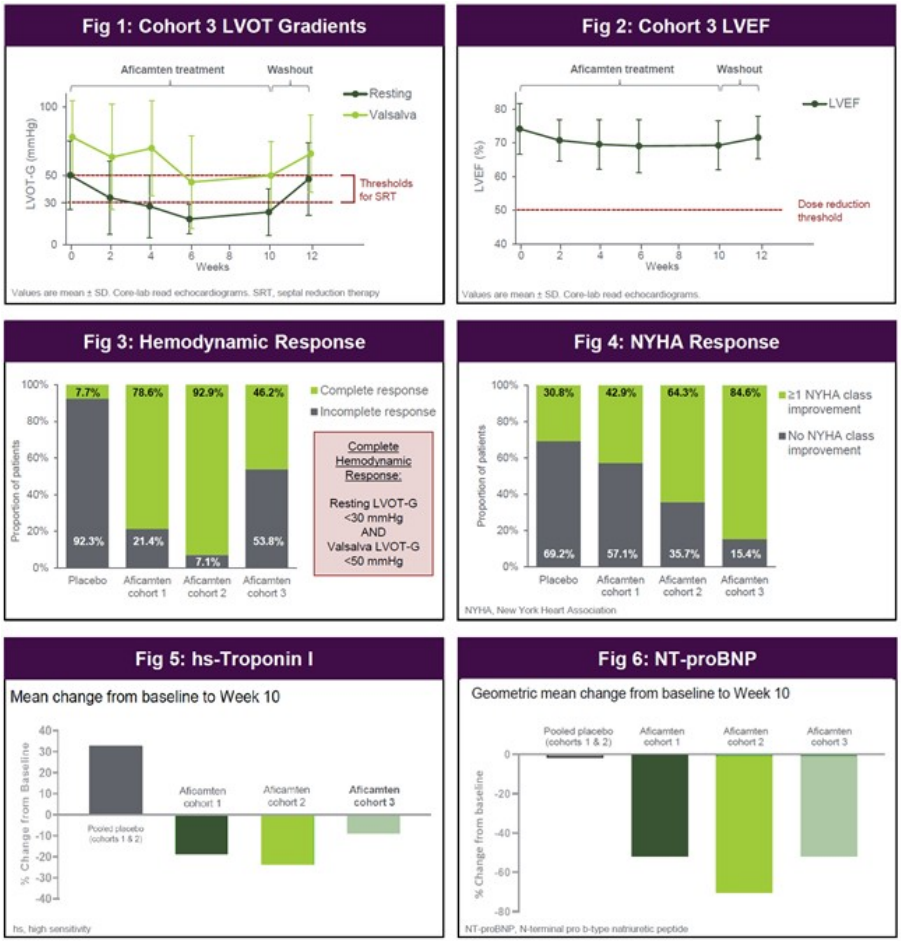
Item 8.01. Other Events.

On April 2, 2022, Cytokinetics, Incorporated (the "Company" or "Cytokinetics") announced that the full results from Cohort 3 of REDWOOD-HCM (**R**andomized **E**valuation of **D**osing **W**ith CK-274 in **O**bstructive **O**utflow **D**isease in **HCM**), the Phase 2 clinical trial of *aficamten* in patients with obstructive hypertrophic cardiomyopathy (HCM), were presented in a Moderated Poster Session by Anjali T. Owens, M.D., Medical Director, Center for Inherited Cardiac Disease, Assistant Professor of Medicine, University of Pennsylvania, at the American College of Cardiology 71st Annual Scientific Session (ACC.22).

Cohort 3 of REDWOOD-HCM enrolled thirteen patients with symptomatic obstructive HCM and a resting or post-Valsalva left ventricular outflow tract gradient (LVOT-G) of ≥50 mmHg whose background therapy included *disopyramide* and, in the majority (11 out of 13 patients), a beta-adrenergic blocker. These patients remained symptomatic despite use of *disopyramide* and represent a group of patients resistant to available medical therapies. All patients received up to three escalating doses of *aficamten* once daily (5, 10, 15 mg), titrated based on echocardiographic guidance. The doses of *aficamten* employed were the same as those used in Cohort 1 of REDWOOD-HCM. Overall treatment duration was 10 weeks with a 4-week follow up period after the last dose. All patients completed the study on treatment.

Patients in Cohort 3 demonstrated a substantial reduction in the mean (± SD) resting LVOT-G (from 50 ± 25 at baseline to 24 ± 17 mmHg at Week 10) and Valsalva LVOT-G (from 78 ± 27 to 50 ± 25 mmHg) (Figure 1). For the resting LVOT-G, the least square mean difference (± SE) for the change from baseline to Week 10 was -28 ± 3.2 mmHg ($p < 0.0001$) and for the Valsalva LVOT-G was -27 ± 5.9 mmHg ($p = 0.0002$). The relief of obstruction was accompanied by a modest reduction in left ventricular ejection fraction (LVEF) (from 74 ± 7% at baseline to 69 ± 7% at Week 10) (Figure 2). For LVEF, the least square mean difference (± SE) for the change from baseline to Week 10 was -4.8 ± 1.9% ($p = 0.018$). There were no patients who experienced a reduction in LVEF below the prespecified safety threshold of 50%.

Treatment with *aficamten* resulted in 6 of the 13 patients (46%) experiencing a complete hemodynamic response by Week 10 (Figure 3), with the remaining 7 (54%) still eligible for dose escalation to the highest dose of *aficamten* (20 mg) employed in SEQUOIA-HCM, the Phase 3 trial. Eleven of 13 patients (85%) experienced improvement in NYHA class by at least one class (Figure 4). In addition to hemodynamic and functional capacity improvements, patients also experienced a significant improvement in NT-proBNP and trended to lower hs-troponin I (Figures 5-6). The safety and tolerability of *aficamten* were consistent with prior experience in REDWOOD-HCM with no dose interruptions or treatment discontinuations and no serious adverse events. Coadministration of *aficamten* along with *disopyramide* and beta- blockers or calcium-channel blockers did not result in any significant electrocardiographic changes including in the QT-interval, or in blood pressure or heart rate.



About REDWOOD-HCM

REDWOOD-HCM HCM (**R**andomized **E**valuation of **D**osing **W**ith CK-274 in **O**bstructive **O**utflow **D**isease in **HCM**) is a Phase 2, multi-center, randomized, placebo-controlled, double-blind, dose finding clinical trial of *aficamten* in patients with symptomatic obstructive HCM (oHCM). In Cohorts 1 and 2, patients continued taking background medications exclusive of *disopyramide*. Results from Cohorts 1 and 2 showed that treatment with *aficamten* for 10 weeks resulted in statistically significant reductions from baseline compared to placebo in the average resting left ventricular outflow tract pressure gradient (LVOT-G) and the average post-Valsalva LVOT-G. A large majority of patients treated with *aficamten* achieved the target goal of treatment, defined as resting gradient <30 mmHg and post-Valsalva gradient <50 mmHg at Week 10, compared to placebo. Patients treated with *aficamten* also saw improvements in heart failure symptoms and reductions in NT-proBNP, a biomarker of cardiac wall stress. Treatment with *aficamten* in REDWOOD-HCM was generally well tolerated and the incidence of adverse events on *aficamten* was similar to that of placebo. No serious adverse events were attributed to *aficamten*, and no treatment interruptions occurred on *aficamten*. Cohort 4 of REDWOOD-HCM is currently enrolling, in an open label fashion, patients with symptomatic non-obstructive HCM receiving background medical therapy. The primary objective is to determine the safety and tolerability of *aficamten* in patients with non-obstructive HCM.

About *Aficamten*

Aficamten is an investigational selective, small molecule cardiac myosin inhibitor discovered following an extensive chemical optimization program that was conducted with careful attention to therapeutic index and pharmacokinetic properties and as may translate into next-in-class potential in clinical development. *Aficamten* was designed to reduce the number of active actin-myosin cross bridges during each cardiac cycle and consequently suppress the myocardial hypercontractility that is associated with hypertrophic cardiomyopathy (HCM). In preclinical models, *aficamten* reduced myocardial contractility by binding directly to cardiac myosin at a distinct and selective allosteric binding site, thereby preventing myosin from entering a force producing state. The development program for *aficamten* is assessing its potential as a treatment that improves exercise capacity and relieves symptoms in patients with HCM as well as its long-term effects on cardiac structure and function. *Aficamten* received Breakthrough Therapy Designation for the treatment of symptomatic obstructive HCM from the U.S. Food & Drug Administration (FDA) as well as the National Medical Products Administration (NMPA) in China.

About Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is a disease in which the heart muscle (myocardium) becomes abnormally thick (hypertrophied). The thickening of cardiac muscle leads to the inside of the left ventricle becoming smaller and stiffer, and thus the ventricle becomes less able to relax and fill with blood. This ultimately limits the heart’s pumping function, resulting in symptoms including chest pain, dizziness, shortness of breath, or fainting during physical activity. A subset of patients with HCM are at high risk of progressive disease which can lead to atrial fibrillation, stroke and death due to arrhythmias. There are no FDA approved medical treatments that directly address the hypercontractility that underlies HCM.

About Cytokinetics

Cytokinetics is a late-stage biopharmaceutical company focused on discovering, developing and commercializing first-in-class muscle activators and next-in-class muscle inhibitors as potential treatments for debilitating diseases in which muscle performance is compromised. As a leader in muscle biology and the mechanics of muscle performance, the company is developing small molecule drug candidates specifically engineered to impact muscle function and contractility. Cytokinetics is readying for the potential commercialization of *omecamtiv mecarbil*, its cardiac muscle activator, following positive results from GALACTIC-HF, a large, international Phase 3 clinical trial in patients with heart failure. Cytokinetics is also developing *aficamten*, a next-generation cardiac myosin inhibitor, currently the subject of SEQUOIA-HCM, the Phase 3 clinical trial of *aficamten* in patients with symptomatic obstructive hypertrophic cardiomyopathy (HCM). *Aficamten* is also being evaluated in non-obstructive HCM in Cohort 4 of the Phase 2 clinical trial, REDWOOD-HCM. Cytokinetics is also developing *reldesemtiv*, an investigational fast skeletal muscle troponin activator, currently the subject of COURAGE-ALS, a Phase 3 clinical trial in patients with amyotrophic lateral sclerosis (ALS). Cytokinetics continues its over 20-year history of pioneering innovation in muscle biology and related pharmacology focused to diseases of muscle dysfunction and conditions of muscle weakness.

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 relating to any of our other clinical trials, including our ability to fully enroll Cohort 4 of REDWOOD-HCM, statements relating to the potential benefits of *aficamten* for patients with obstructive hypertrophic cardiomyopathy or any of our other drug candidates. Such statements are based on management's current expectations, but actual results may differ materially due to various risks and uncertainties, including, but not limited to, potential difficulties or delays in the development, testing, regulatory approvals for trial commencement, progression or product sale or manufacturing, or production of Cytokinetics' drug candidates that could slow or prevent clinical development or product approval; Cytokinetics' drug candidates may have adverse side effects or inadequate therapeutic efficacy; the FDA or foreign regulatory agencies may delay or limit Cytokinetics' ability to conduct clinical trials; Cytokinetics may be unable to obtain or maintain patent or trade secret protection for its intellectual property; standards of care may change, rendering Cytokinetics' drug candidates obsolete; and competitive products or alternative therapies may be developed by others for the treatment of indications Cytokinetics' drug candidates and potential drug candidates may target. For further information regarding these and other risks related to Cytokinetics' business, investors should consult Cytokinetics' filings with the Securities and Exchange Commission.

SIGNATURE

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

CYTOKINETICS, INCORPORATED

Date: April 4, 2022

By: /s/ Ching Jaw
Ching Jaw
Senior Vice President, Chief Financial Officer