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): June 2, 2011

Cytokinetics, Incorporated

(Exact name of registrant as specified in its charter)

Delaware	000-50633	94-3291317
(State or other jurisdiction of incorporation)	(Commission File Number)	(I.R.S. Employer Identification No.)
280 East Grand Avenue, South San Francisco, California		94080
(Address of principal executive offices)		(Zip Code)

Registrant's telephone number, including area code: (650) 624 - 3000

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))

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Item 8.01 Other Events.

On June 2, 2011, Cytokinetics, Incorporated (the "Company") issued a press release announcing the successful completion of its Phase IIa "Evidence of Effect" clinical trial of CK-2017357, a fast skeletal muscle activator, in patients with claudication associated with peripheral artery disease, in connection with a poster presentation at the Society for Vascular Medicine's 2011 Annual Meeting being held June 2-4 in Boston, MA. In addition, a second poster presentation highlighted the potential role of a novel functional endpoint, bilateral heel raise testing, in clinical trials in patients with peripheral artery disease. CK-2017357 is the lead drug candidate from the Company's skeletal muscle contractility program. A copy of the press release is filed as Exhibit 99.1 to this Current Report on Form 8-K, and is incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

d) Exhibits.

The following Exhibit is filed as part of this Current Report on Form 8-K:

Exhibit No. Description

99.1 Press Release, dated June 2, 2011.

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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.

Cytokinetics, Incorporated

June 2, 2011

By: Sharon Barbari

Name: Sharon Barbari
Title: Executive Vice President, Finance and Chief Financial Officer

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Exhibit No.	Description
99.1	Press Release, dated June 2, 2011.

Contact:
Christopher S. Keenan
Director, Investor Relations
(650) 624-3000

CYTOKINETICS ANNOUNCES SUCCESSFUL COMPLETION OF PHASE IIA "EVIDENCE OF EFFECT" CLINICAL TRIAL OF CK-2017357, IN PATIENTS WITH CLAUDICATION ASSOCIATED WITH PERIPHERAL ARTERY DISEASE

Clinical Trial Demonstrates Translation of Mechanism of Action of Novel Drug Candidate in 2nd Disease Population

South San Francisco, CA, June 2, 2011 – Cytokinetics, Incorporated (Nasdaq: CYTK) announced the successful completion of its Phase IIa "Evidence of Effect" clinical trial of CK-2017357, a fast skeletal muscle activator, in patients with claudication associated with peripheral artery disease in connection with a poster presentation today at the 22nd Annual Sessions of the Society for Vascular Medicine being held June 2-4 in Boston, MA. In addition, a second poster presentation today highlighted the potential role of a novel functional endpoint, bilateral heel raise testing, in clinical trials in patients with claudication associated with peripheral artery disease. CK-2017357 is the lead drug candidate from the company's skeletal muscle contractility program.

Phase IIa Evidence of Effect Clinical Trial Presentation

A poster titled "A Phase II, Double-Blind, Randomized, Placebo-Controlled, Three-Way Crossover Pharmacokinetic and Pharmacodynamic Study of CK-2017357, a Fast Skeletal Muscle Troponin Activator, in Patients with Claudication" was presented by Alan Hirsch, MD, Cardiovascular Division and Lillehei Heart Institute, University of Minnesota, on June 2, 2011.

This poster summarized the results of a recently completed Phase IIa Evidence of Effect clinical trial in patients with calf muscle claudication associated with peripheral artery disease. The authors concluded that CK-2017357 increased calf muscle performance in these patients as evidenced by an increase in work achieved during bilateral heel raise testing, a performance measure in this patient population developed for this trial and presented by Dr. Hirsch in a second poster described below. The authors also concluded that increases in calf muscle performance and adverse events appear related to both increasing dose and to the CK-2017357 plasma concentration.

"This clinical trial successfully translated the mechanism of fast skeletal muscle activation with CK-2017357 to an improvement in human skeletal muscle function, as evidenced by the ability of these patients with claudication due to peripheral artery disease to do more calf muscle work before intolerable claudication or limiting fatigue," stated William R. Hiatt, MD, Principal Investigator of this clinical trial, President, CPC Clinical Research and Professor of Cardiovascular Research, University of Colorado School of Medicine. "The increases in calf muscle performance, evidenced during bilateral heel raise testing, were observed after only a single dose of CK-2017357. Further studies are warranted to refine the choice of dose and duration of treatment to minimize systemic adverse effects while still retaining the potential clinical benefit."

"We designed this clinical trial to develop hypotheses that could inform the further development of our fast skeletal muscle troponin activators and therefore we are pleased that it produced pharmacodynamic results that are encouraging and consistent with data generated in our preclinical and Phase I studies," stated Andrew A. Wolff, MD, FACC, Cytokinetics' Senior Vice President of Clinical Research and Development and Chief Medical Officer. "We now have clinically relevant hypotheses regarding CK-2017357 for the possible treatment of claudication associated with peripheral artery disease that we believe warrant further exploration, much as was the case with our previously announced clinical trial of this same drug candidate in patients with amyotrophic lateral sclerosis."

Phase IIa Clinical Trial Design

This Phase IIa Evidence of Effect clinical trial was a double-blind, randomized, placebo-controlled, three-way crossover, pharmacokinetic and pharmacodynamic study of CK-2017357 in male and female patients with symptoms of claudication associated with peripheral artery disease. The primary objective of this trial was to demonstrate an effect of single doses of CK-2017357 on measures of skeletal muscle function and fatigability in patients with peripheral artery disease and claudication. The secondary objectives of this clinical trial were to evaluate and characterize the relationship, if any, between the doses and plasma concentrations of CK-2017357 and its pharmacodynamic effects, and to evaluate the safety and tolerability of CK-2017357 administered as single doses to these patients. Accordingly, in this hypothesis-generating trial, multiple pharmacodynamic assessments were made without specifying a single primary pharmacodynamic endpoint.

A total of 61 patients were enrolled in this three-period crossover trial; in random order and with a 6 to 10 day wash-out period between each of the three treatments, 56 patients received a single dose of CK-2017357 at 375 mg, 33 received a single dose of CK-2017357 at 750 mg, and 57 received a single dose of matching placebo. As was previously announced, the protocol was amended after 33 patients were dosed so that the patients enrolled subsequently received a maximum dose of 500 mg (n = 27). Maximum plasma concentrations of CK-2017357 were generally achieved between 3 and 6 hours after dosing, which is when most assessments were made.

Bilateral heel raise testing was performed before each dose of study drug, and 3 hours and 6 hours after each dose. Six measurements were made during bilateral heel raise testing: time, number of repetitions, and work performed to claudication onset, as well as time, number of repetitions, and total work performed to intolerable claudication or calf muscle fatigue, when the test was ended. Work performed during each repetition was calculated by multiplying the maximal heel elevation achieved during that repetition (measured by an instrument attached to the patient's ankle called an electrogoniometer) by the patient's weight; work to the end of testing was the sum of work performed during all repetitions performed during the test. In addition, a six-minute walk test was conducted at 4 hours after dosing, in which the distance walked by the patient during a six minute period was measured.

Phase IIa Clinical Trial Results

The authors concluded that there was an overall trend for CK-2017357 to increase the parameters of calf muscle function as assessed by bilateral heel raise testing. In general, these increases were most evident after the 750 mg dose and at the highest plasma concentrations achieved during the trial. In addition, they were more prominent at 6 hours than at 3 hours, on measurements taken at the end of testing than at the onset of claudication, and on work performed than on time or number of repetitions. For example, at 6 hours after dosing, the change from baseline in work performed to the end of testing increased by a median value of 5.7 kg-m on placebo, 14.2 kg-m on 375 mg of CK-2017357, 20.2 kg-m on 500 mg of CK-2017357, and 34.1 kg-m on 750 mg of CK-2017357 ($p = 0.097$, 0.181, and 0.002 for 375, 500, and 750 mg of CK-2017357 versus placebo, respectively). In addition, the overall relationship between CK-2017357 dose and work performed to the end of testing achieved conventional statistical significance ($p = 0.007$).

Increases in work performed to the end of testing were related not only to dose but also to the plasma concentration of CK-2017357 measured at the time of the test. At the highest concentrations observed in the trial (>14 mg/mL, n = 37 observations), the change from baseline in work performed to the end of testing increased by an average of 24.4 kg-m versus placebo ($p = 0.007$). In the concentration ranges from 0 to 7 ng/mL (n = 50 observations), >7 to 10 ng/mL (n = 72 observations), and >10 to 14 ng/mL (n = 45 observations), the average increases versus placebo were 9.5 kg-m ($p = 0.247$), 5.4 kg-m ($p = 0.440$), and 16.6 kg-m ($p = 0.047$), respectively. In addition, the relationship between total work performed to the end of testing and the plasma concentration of CK-2017357 also achieved conventional statistical significance ($p = 0.005$).

In contrast to the increases in calf muscle performance observed during bilateral heel raise testing, there were dose- and plasma concentration-related decreases in the six-minute walking distances. From a mean baseline six-minute walk distance of 1079 feet, after CK-2017357 doses of 375 mg , 500 mg, and 750 mg, the average six-minute walk distances declined versus placebo by 23.8 feet ($p = 0.147$), 34.1 feet ($p = 0.120$), and 106.6 feet ($p < 0.001$), respectively, and the relationship of these declines in six-minute walk distance to dose was also statistically significant ($p < 0.001$). Similar decreases in the six-minute walk distance were also observed in relation to increasing plasma concentrations of CK-2017357 ($p < 0.001$).

The authors also concluded that the single doses of CK-2017357 administered in this study appeared safe and generally well-tolerated by the patients in this trial. A substantial majority of the adverse events reported in the study were deemed mild or moderate and were consistent with those observed in prior studies with CK-2017357; however, the percentage of patients reporting severe adverse events during the trial increased with the dose of CK-2017357. The most commonly reported adverse event was dizziness, which was observed in 80% of the patients in the trial and was dose-related. Severe dizziness was reported by more patients than was any other severe adverse event; all four patients who reported severe dizziness did so after receiving the 750 mg dose. The authors speculated that dizziness and other dose-related adverse events may have negatively impacted walking distance but not heel raise testing because walking is a more complex functional task, requiring integration of numerous abilities in addition to calf muscle performance (vision, balance, and coordination, for example). As previously reported, two patients experienced serious adverse events after treatment with CK-2017357 at 750 mg, which were judged to have been related to treatment with the study drug. Both patients required hospitalization but recovered fully without specific therapy. A third patient was hospitalized for severe worsening of cholecystitis following his second dose of study drug at the 500 mg level but investigators deemed that this was unrelated to study drug. The authors concluded that CK-2017357 merits further study and the potential next steps may include studies to explore whether the adverse events observed, such as dizziness, could abate with repeated dosing, alternate dosing regimens, and/or gradual dose titration.

Bilateral Heel Raise Poster Presentation

A second poster titled “Bilateral Heel Raise Test: A Novel Functional Endpoint for Early Stage Clinical Trials in Peripheral Artery Disease (PAD),” was also displayed and presented by Dr. Hirsch on Thursday, June 2, 2011. This poster described in more detail the bilateral heel raise test that was used in the Evidence of Effect trial reported above and evaluated its utility as a functional assessment in patients with calf muscle claudication. The parameters assessed from a single bilateral concentric heel raise test demonstrated modest reliability across baseline measurements during a 3-week period in patients with peripheral artery disease and claudication. Intra-class correlations by patient for time, number of repetitions, and work to the end of testing were 79%, 76%, and 75%, respectively. The authors concluded that bilateral heel raises performed according to a specified-protocol consistently elicited claudication in patients with a history of claudication associated with peripheral artery disease and thus may be a functionally relevant, easy-to-deploy, and cost-effective measure of calf muscle endurance and fatigue in patients with claudication due to peripheral artery disease. Consequently, the use of the heel raise test in early phase “proof of concept” clinical trials may facilitate demonstration of clinical efficacy in patients with claudication without requiring treadmill procedures. Comparison of bilateral heel raise tests with standardized treadmill testing will be needed to establish this test as a surrogate endpoint that parallels the current gold standard for functional exercise testing in the peripheral artery disease population.

Development Status of CK-2017357

CK-2017357 is currently the subject of a clinical trials program that includes two completed Phase I studies in healthy volunteers and a completed Phase IIa Evidence of Effect clinical trial in patients with amyotrophic lateral sclerosis (ALS), all of which have been previously reported. In addition, a Phase IIa Evidence of Effect clinical trial in patients with myasthenia gravis is ongoing.

In the Phase IIa Evidence of Effect clinical trial in patients with ALS, the investigators concluded that the single doses of CK-2017357 evaluated appeared safe and generally well-tolerated by the patients in this trial. In addition, the investigators concluded that both patients and investigators perceived a positive change in the patients’ overall status, in a dose-dependent fashion, at 6 hours after dosing with CK-2017357, based on a Global Assessment in which the patient and the investigator each independently assessed their status compared to just before dosing. Furthermore, there was a clear relationship between improvements in Global Assessments and the CK-2017357 plasma concentration. The investigators proposed that improvements in the patients’ and investigators’ Global Assessments may have resulted from a decrease in the fatigability of their muscles, as evidenced by data from a test of sub-maximal hand-grip fatigability. Data from this clinical trial demonstrated a dose-related trend to increase the maximum volume of air patients could inhale and exhale in one minute (Maximum Voluntary Ventilation) at both 6 and 24 hours after 500 mg of CK-2017357.

CK-2017357 has been granted orphan-drug status by the United States Food and Drug Administration for the potential treatment of ALS. Cytokinetics anticipates initiating a Phase IIa multiple-dose, safety, tolerability, and pharmacokinetic clinical trial of CK-2017357 in patients with ALS by mid-year 2011.

Background on Cytokinetics’ Skeletal Muscle Contractility Program

CK-2017357, a fast skeletal muscle troponin activator, is the lead drug candidate from the company’s skeletal muscle contractility program. CK-2017357 selectively activates the fast skeletal muscle troponin complex by increasing its sensitivity to calcium, leading to an increase in skeletal muscle force. This mechanism of action has demonstrated encouraging pharmacological activity in preclinical models that may relate to the potential treatment of diseases associated with aging, muscle wasting or neuromuscular dysfunction. Skeletal muscle contractility is driven by the sarcomere, the fundamental unit of skeletal muscle contraction. The sarcomere is a highly ordered cytoskeletal structure composed of skeletal muscle myosin, the cytoskeletal motor that is directly responsible for converting chemical energy into mechanical force, as well as actin, and a set of regulatory proteins, troponins and tropomyosin, which make the actin-myosin interaction dependent on changes in intracellular calcium levels. Cytokinetics’ skeletal muscle contractility program is focused to the discovery and development of small molecule skeletal sarcomere activators and leverages Cytokinetics’ expertise developed in its ongoing discovery and development of cardiac sarcomere activators, including the cardiac myosin activator *omecamtiv mecarbil*, now in clinical development as a potential treatment for heart failure. Skeletal sarcomere activators have demonstrated pharmacological activity in preclinical models that may lead to new therapeutic options for diseases associated with aging, muscle wasting and neuromuscular dysfunction. The clinical effects of muscle wasting, fatigue and loss of mobility can range from decreased quality of life to, in some instances, life-threatening complications. By directly improving skeletal muscle function, a small molecule activator of the skeletal sarcomere may potentially enhance physical performance and quality of life in aging patients.

About CPC Clinical Research

Founded in 1989 by the University of Colorado, CPC Clinical Research is an academically led, not-for-profit, clinical research organization offering Phase 1

— 4 clinical research management services to sponsors around the world. CPC specializes in innovative cardiovascular research including a proprietary program, the Endpoint Quality Intervention Program (EQuIP), that helps reduce variability in the collection of endpoint data, allowing more definitive answers to the study objectives. CPC is affiliated with the University Of Colorado Denver School Of Medicine, Denver Health and the National Jewish Health, within the University of Colorado.

About Cytokinetics

Cytokinetics is a clinical-stage biopharmaceutical company focused on the discovery and development of novel small molecule therapeutics that modulate muscle function for the potential treatment of serious diseases and medical conditions. Cytokinetics' lead drug candidate from its cardiac muscle contractility program, *omecamtiv mecarbil* (formerly CK-1827452), is in clinical development for the potential treatment of heart failure. Amgen Inc. holds an exclusive license worldwide (excluding Japan) to develop and commercialize *omecamtiv mecarbil* and related compounds, subject to Cytokinetics' specified development and commercialization participation rights. Cytokinetics is independently developing CK-2017357, a skeletal muscle activator, as a potential treatment for diseases and conditions associated with aging, muscle wasting or neuromuscular dysfunction. CK-2017357 is currently the subject of a Phase IIa clinical trials program and has been granted orphan-drug designation by the U.S. Food and Drug Administration for the potential treatment of amyotrophic lateral sclerosis, a debilitating disease of neuromuscular impairment in which CK-2017357 demonstrated potentially clinically relevant pharmacodynamic effects in a Phase IIa trial. Cytokinetics is also conducting research and non-clinical development of compounds that inhibit smooth muscle contractility and which may be useful as potential treatments for diseases and conditions associated with excessive smooth muscle contraction, such as bronchoconstriction associated with asthma and chronic obstructive pulmonary disorder (COPD). In addition, prior Cytokinetics' research generated three anti-cancer drug candidates that have progressed into clinical development: *ispinesib*, SB-743921 and GSK-923295. All of these drug candidates and potential drug candidates have arisen from Cytokinetics' research activities and are directed towards the cytoskeleton. The cytoskeleton is a complex biological infrastructure that plays a fundamental role within every human cell. Additional information about Cytokinetics can be obtained at www.cytokinetics.com

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 Cytokinetics' and its partners' research and development activities, including plans for and the initiation, conduct, design and results of clinical trials for CK-2017357, the significance and utility of clinical trial results for CK-2017357 and the utility of the bilateral heel raise test as a functional assessment of claudication; and the properties and potential benefits of CK-2017357 and Cytokinetics' other drug candidates and potential drug candidates, including CK-2017357's potential utility in the treatment of patients with ALS or claudication associated with peripheral artery disease. 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, including risks that current and past results of clinical trials or preclinical studies may not be indicative of future clinical trials results, patient enrollment for or conduct of clinical trials may be difficult or delayed, Cytokinetics' drug candidates may have adverse side effects or inadequate therapeutic efficacy, the U.S. Food and Drug Administration (FDA) or foreign regulatory agencies may delay or limit Cytokinetics' or its partners' ability to conduct clinical trials, the FDA may not grant CK-2017357 orphan drug market exclusivity even if it is approved for marketing, and Cytokinetics may be unable to obtain or maintain patent or trade secret protection for its intellectual property; Amgen's decisions with respect to the design, initiation, conduct, timing and continuation of development activities for omecamtiv mecarbil; Cytokinetics may incur unanticipated research and development and other costs or be unable to obtain additional financing necessary to conduct development of its products on acceptable terms, if at all; Cytokinetics may be unable to enter into future collaboration agreements for its drug candidates and programs on acceptable terms, if at all; standards of care may change, rendering Cytokinetics' drug candidates obsolete; competitive products or alternative therapies may be developed by others for the treatment of indications Cytokinetics' drug candidates and potential drug candidates may target; and risks and uncertainties relating to the timing and receipt of payments from its partners, including milestones and royalties on future potential product sales under Cytokinetics' collaboration agreements with such partners. For further information regarding these and other risks related to Cytokinetics' business, investors should consult Cytokinetics' filings with the Securities and Exchange Commission.