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 18, 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 April 18, 2011, Cytokinetics, Incorporated issued a press release announcing that clinical data from its recently completed Phase IIa "Evidence of Effect" (EoE) clinical trial of CK-2017357 in patients with amyotrophic lateral sclerosis (ALS), also known as Lou Gehrig's disease, were presented in the 2011 Clinical Trials Session at the 63rd Annual Meeting of the American Academy of Neurology in Honolulu, Hawaii on Friday, April 15th.

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.

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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

April 18, 2011

By: Sharon Barbari

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

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Exhibit Index

Exhibit No.	Description
99.1	Press Release, dated April 18, 2011

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

CYTOKINETICS ANNOUNCES PRESENTATION OF DATA FROM A PHASE IIA CLINICAL TRIAL OF CK-2017357 IN PATIENTS WITH AMYOTROPHIC LATERAL SCLEROSIS AT THE 63RD ANNUAL MEETING OF THE AMERICAN ACADEMY OF NEUROLOGY

Presentation Included Additional Results Arising from Recent Analyses Relating to Muscle Strength at 24 Hours and Concentration-Response Relationships

South San Francisco, CA, April 18, 2011 – Cytokinetics, Incorporated (Nasdaq: CYTK) announced that clinical data from its recently completed Phase IIa "Evidence of Effect" (EoE) clinical trial of CK-2017357 in patients with amyotrophic lateral sclerosis (ALS), also known as Lou Gehrig's disease, were presented in the 2011 Clinical Trials Session at the 63rd Annual Meeting of the American Academy of Neurology in Honolulu, Hawaii on Friday, April 15th.

A presentation titled, "A Phase 2A, Double-Blind, Randomized, Placebo-Controlled, Single-Dose, Crossover Study of the Selective Fast Skeletal Muscle Troponin Activator, CK-2017357, in Patients with ALS," was made by Jeremy M. Shefner, MD, PhD, Professor and Chair, Department of Neurology, Upstate Medical University, State University of New York. CK-2017357 is the lead drug candidate from the company's skeletal muscle contractility program.

Data from this trial were initially presented at the 21st International Symposium on ALS and Motor Neurone Diseases in December of last year. Additional results presented at this meeting relate to the effects of CK-2017357 on the strength of selected muscle groups, as well as concentration-response relationships for key outcome measures. More recent analyses showed that, at 24 hours after each of the 250 and 500 mg doses of CK-2017357, dose-related increases in the change from the Day 1 baseline in percent predicted muscle strength achieved nominal statistical significance for elbow flexion ($p = 0.005$ and $p = 0.0004$, respectively), shoulder flexion ($p = 0.008$ and $p = 0.002$, respectively) and knee extension ($p = 0.003$ and $p = 0.001$, respectively). In addition, a statistically significant relationship to the CK-2017357 plasma concentration was observed for improvements in both the patients' and investigators' Global Assessments of Change ($p = 0.03$ and $p = 0.01$, respectively), and in the Sniff Inspiratory Pressure ($p = 0.03$).

"I am honored to have the opportunity to present these data to my fellow neurologists at the American Academy of Neurology's 63rd Annual Meeting," said Dr. Shefner. "The additional results presented at this meeting, together with the data presented late last year, suggest that a skeletal muscle activator such as CK-2017357 may offer a novel approach to improving muscle function in patients suffering from amyotrophic lateral sclerosis. All of these results support further study of this class of compounds to evaluate the potential for sustained functional benefit in patients with ALS."

"We are pleased that data from this Phase IIa clinical trial were selected for presentation in the Clinical Trials Session at this prestigious clinical conference," stated Andrew A. Wolff, MD, FACC, Cytokinetics' Senior Vice President of Clinical Research and Development and Chief Medical Officer. "Importantly, this trial has now generated clinically relevant hypotheses that warrant further exploration in larger and longer proof-of-concept clinical trials in patients with ALS."

Phase IIa Clinical Trial Results

In this Phase IIa clinical trial, a single 250 mg dose of CK-2017357, a single 500 mg dose of CK-2017357, and a single matching dose of placebo were each administered once, in a double-blind fashion and in random order, at least 6 days apart to male and female ALS patients. The maximum CK-2017357 plasma concentration generally was achieved between 3 and 6 hours after dosing, which is when most assessments were made; some were also repeated at 24 hours after dosing.

Dr. Shefner reported that both patients and investigators perceived a positive change in the patients' overall status at 6 hours after dosing with CK-2017357, based on a Global Assessment in which the patient and the investigator each independently assessed whether the patient was "better," "same," or "worse" compared to just before dosing on that day. At 6 hours after receiving 500 mg of CK-2017357, 29 of 65 patients assessed themselves as "better," compared to 18 of 63 on placebo ($p = 0.04$). Similarly, at that time point, the investigators assessed only 8 of 63 patients on placebo as "better," compared with 15 of 62 after 250 mg of CK-2017357 ($p = 0.06$) and 20 of 65 after 500 mg of CK-2017357 ($p = 0.004$). Conventional nominal statistical significance for the dose response for these improvements in Global Assessments at 6 hours after dosing was approached for the patients' assessments ($p = 0.07$) and achieved for the investigators' ($p = 0.01$).

Furthermore, there was a clear relationship between improvements in Global Assessments and the CK-2017357 plasma concentration. Patients were more than twice as likely to assess themselves as "better" when their plasma concentrations were above 9 mcg/mL than on placebo (odds ratio 2.45; $p = 0.01$), while the investigators were almost four times more likely to assess their patients as "better" when the patients' plasma concentrations were in that range (odds ratio 3.81; $p = 0.0007$). In addition, the relationship between the likelihood of a "better" assessment and the CK-2017357 plasma concentration was nominally statistically significant both for the patients' assessments ($p = 0.03$) and for the investigators' ($p = 0.01$).

Dr. Shefner proposed that these improvements in the patients' and investigators' Global Assessments may have resulted from a decrease in muscle fatigability of their muscles, as evidenced by data from a test of sub-maximal hand-grip fatigability. For this measurement, each patient was asked to maintain a sub-maximal target force set at 30% of his or her individually measured maximal handgrip force for as long as possible (up to 2 minutes). The times when the patient's hand-grip force fell below 100%, 90%, 80%, 70% and 60% of this target were then recorded. Both hands were studied, and the weaker hands were analyzed separately from the stronger hands.

At 6 hours after dosing, dose-related trends to prolong the times patients could maintain the grip force in the weaker hand above 80%, 70%, and 60% of the target force were apparent. At 6 hours after the 500 mg dose of CK-2017357, these prolongations in the weaker hand approached nominal statistical significance for the times grip force could be maintained above 70% and 60% of the target force (7.8 seconds [$p = 0.08$] and 7.2 seconds [$p = 0.06$] versus placebo, respectively). Similarly, the dose response for these prolongations in the patients' ability to maintain sub-maximal grip force in the weaker hand at 6 hours after dosing also approached nominal statistical significance for the times grip force could be maintained above 70% and 60% of the target force ($p = 0.08$ and $p = 0.06$, respectively).

In analyses not presented last December, muscle strength improved slightly in some muscle groups, but not others. At 24 hours after each of the 250 and 500 mg doses, dose-related increases in the change from the Day 1 baseline in percent predicted muscle strength achieved nominal statistical significance for elbow flexion ($p = 0.005$ and $p = 0.0004$, respectively), shoulder flexion ($p = 0.008$ and $p = 0.002$, respectively) and knee extension ($p = 0.003$ and $p = 0.001$, respectively).

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) which achieved nominal statistical significance at both 6 and 24 hours after 500 mg of CK-2017357 (3.5 liters [p = 0.05] and 4.2 liters [p = 0.02] versus placebo, respectively). Trends to increase the patients' force of inhalation, a measure of pulmonary function known as the Sniff Inspiratory Pressure, were also observed, and approached nominal statistical significance at 6 hours after the 500 mg dose of CK-2017375 (2.84 cm H_2O versus placebo; p = 0.10). In addition, the relationship between increases in Sniff Inspiratory Pressure and increasing CK-2017375 plasma concentrations achieved nominal statistical significance (p = 0.03).

Finally, the investigators also concluded that these single doses of CK-2017357 were safe and generally well-tolerated by the patients in this trial. There were no serious adverse events that were judged to have been related to treatment with the study drug. Most adverse events were classified by the investigators as mild, including dizziness, the most commonly-reported and most clearly dose-related adverse event. All reports of dizziness by patients receiving 250 mg of CK-2017357 were classified as mild, as were 88% of those reported by patients receiving 500 mg of CK-2017357. The other complaints of dizziness by patients receiving 500 mg of CK-2017357 were classified as moderate; none were determined to be severe.

Phase IIa Clinical Trial Design

This Phase IIa Evidence of Effect (EOE) clinical trial was a double-blind, randomized, placebo-controlled, three-period crossover, pharmacokinetic and pharmacodynamic study of CK-2017357 in male and female patients with ALS.

The primary objective of this trial was to evaluate the pharmacodynamic effects of CK-2017357 on measures of skeletal muscle function or fatigability in patients with ALS. Accordingly, in this hypothesis-generating trial, multiple pharmacodynamic assessments were made without specifying a single primary pharmacodynamic endpoint. These assessments included various measures of maximum voluntary muscle strength, development of fatigue at maximum and sub-maximum voluntary muscle contraction, and pulmonary function, measured at baseline, and at 3, 6 and 24 hours post-dosing after each of two single doses of CK-2017357 and placebo. The secondary objectives of this clinical trial were to evaluate the relationship between the plasma concentration of CK-2017357 and its pharmacodynamic effects, to evaluate the safety and tolerability of the two single doses of CK-2017357 administered orally to patients with ALS, and to evaluate the effects of CK-2017357 on patient- and investigator-determined global functional assessments.

Single doses of placebo and of CK-2017357, at each of 250 mg and 500 mg, were each administered once, in a double-blind fashion and in random order, at least 6 days apart. A total of 67 patients with ALS underwent multiple quantitative measures of muscle strength, including hand held dynamometry of 18 muscle groups, vital capacity, sniff inspiratory pressure, maximum voluntary ventilation, and hand grip. In addition, novel measures of endurance were developed, including maintenance of 30% and 70% of maximal handgrip force and duration of shoulder flexion. A functional assessment tool was developed. Testing was performed at baseline, 3, 6, and 24 hours after dosing. Given that pharmacodynamic effects in healthy volunteers were noted 3-7 hours after receiving single doses of CK-2017357 at 250 mg and 500mg, it was felt appropriate to evaluate functional changes at these times.

Development Status of CK-2017357

In 2010, Cytokinetics announced data from two Phase I clinical trials evaluating CK-2017357. The first trial was a two-part, single-dose trial. Part A of this trial was designed to assess the safety, tolerability and pharmacokinetic profile of increasing single doses of this drug candidate in healthy male volunteers and to determine its maximum-tolerated dose and associated plasma concentrations. The maximum-tolerated single dose of CK-2017357 in Part A of the trial was 2000 mg. Part B of this trial was designed to assess the pharmacodynamic effects, versus placebo, of CK-2017357 on skeletal muscle function after single oral doses of 250, 500 and 1000 mg, and to assess the relationship of the effects observed to the associated plasma concentrations of CK-2017357, also in healthy male volunteers. In Part B, CK-2017357 produced concentration-dependent, statistically significant increases versus placebo in the force developed by the tibialis anterior muscle. In both Part A and Part B, CK-2017357 was well-tolerated and no serious adverse events were reported.

The second trial was a multiple-dose, Phase I clinical trial of CK-2017357 designed to investigate the safety, tolerability and pharmacokinetic profile of CK-2017357 after multiple oral doses to steady state in healthy male volunteers. This trial evaluated doses that produced plasma concentrations in the range associated with pharmacodynamic activity in Part B of the single-dose Phase I clinical trial. At steady state, both the maximum plasma concentration and the area under the CK-2017357 plasma concentration versus time curve from before dosing until 24 hours after dosing were generally dose-proportional. In general, systemic exposure to CK-2017357 in this trial was high and inter-subject variability was low. In addition, these multiple-dose regimens of CK-2017357 were well-tolerated, and no serious adverse events were reported.

CK-2017357 is currently the subject of a Phase IIa clinical trials program and has been granted orphan-drug designation by the United States Food and Drug Administration for the potential treatment of ALS. Cytokinetics is conducting two ongoing Phase IIa EoE clinical trials, one in patients with claudication associated with peripheral artery disease and another in patients with myasthenia gravis. Cytokinetics has also initiated a Phase I drug-drug interaction study of CK-2017357 administered orally to healthy volunteers, intended to evaluate the effects of CK-2017357 on the pharmacokinetics of *riluzole* and other drugs and the pharmacokinetics of CK-2017357 when administered after a meal and when fasting.

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 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. Cytokinetics is also conducting 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 systemic hypertension or bronchoconstriction. 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 the conduct, design and results of clinical trials for CK-2017357, the significance and utility of clinical trial results for CK-2017357, 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. 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; 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.