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): January 4, 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 January 4, 2011, Cytokinetics, Incorporated issued a press release announcing that the company has opened enrollment in a Phase IIa "Evidence of Effect" (EoE) clinical trial of CK-2017357 in patients with generalized myasthenia gravis (MG). CK-2017357 is a fast skeletal muscle troponin activator and is the lead drug candidate that has emerged from the company's skeletal muscle contractility program. CK-2017357 selectively activates the fast skeletal muscle troponin complex and increases its sensitivity to calcium, which increases skeletal muscle force in response to neuronal input and also delays the onset and reduces the degree of muscle fatigue. In July 2010, Cytokinetics was awarded a grant in the amount of $2.8 million by the National Institute of Neurological Disorders and Stroke to support research and development of CK-2017357 in MG.

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 January 4, 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

January 4, 2011

By: /s/ 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 January 4, 2011

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

CYTOKINETICS ANNOUNCES OPENING OF A PHASE IIA "EVIDENCE OF EFFECT" CLINICAL TRIAL OF CK-2017357 IN PATIENTS WITH MYASTHENIA GRAVIS

South San Francisco, CA, January 4, 2011 – Cytokinetics, Incorporated (Nasdaq:CYTK) announced today that the company has opened enrollment in a Phase IIa "Evidence of Effect" (EoE) clinical trial of CK-2017357 in patients with generalized myasthenia gravis (MG). CK-2017357 is a fast skeletal muscle troponin activator and is the lead drug candidate that has emerged from the company's skeletal muscle contractility program. CK-2017357 selectively activates the fast skeletal muscle troponin complex and increases its sensitivity to calcium, which increases skeletal muscle force in response to neuronal input and also delays the onset and reduces the degree of muscle fatigue. In July 2010, Cytokinetics was awarded a grant in the amount of $2.8 million by the National Institute of Neurological Disorders and Stroke to support research and development of CK-2017357 in MG.

This Phase IIa EOE clinical trial is a double-blind, randomized, three-period crossover, placebo-controlled, pharmacokinetic (PK) and pharmacodynamic (PD) study of CK-2017357 in patients with generalized MG. At least 36 and up to 78 patients may be enrolled at approximately 15 study centers in the United States. Patients enrolled in the trial will receive single oral doses of placebo, 250 mg, and 500 mg of CK-2017357 in random order. A wash-out period of at least 7 days (to a maximum of 10 days) will be employed between the individual doses for each patient.

The primary objective of this hypothesis-generating clinical trial is to assess the effects of CK-2017357 on measures of muscle strength, muscle fatigue and pulmonary function utilizing the standardized Quantitative MG score, Manual Muscle Test, and MG Composite score. The secondary objectives of this clinical trial are to evaluate and characterize the relationship, if any, between the doses and plasma concentrations of CK-2017357 and its PD effects; to evaluate the safety and tolerability of CK-2017357 administered as single doses to patients with MG; and to evaluate the effect of CK-2017357 on investigator and patient determined global functional assessment and the Modified MG Symptom Score.

"This hypothesis-generating clinical trial is designed to seek evidence of a pharmacodynamic effect of CK-2017357 to increase skeletal muscle performance or to delay the onset and reduce the magnitude of muscle fatigue in patients with generalized myasthenia gravis. Based on non-clinical and clinical results generated to date, we believe that we may be able to demonstrate potentially clinically relevant pharmacodynamic effects of CK-2017357, even after a single dose," stated Fady Malik, MD, PhD, FACC, Cytokinetics' Vice President of Biology and Therapeutics. "Recent data presented from a similarly-designed Phase IIa clinical trial in patients with amyotrophic lateral sclerosis (ALS) provided encouraging signals of potential efficacy in a disease in which neuromuscular signaling is impaired, thereby underscoring a possible role for this drug candidate in patients with other neuromuscular conditions such as myasthenia gravis."

Background on Myasthenia Gravis

Myasthenia gravis is a progressive, chronic, autoimmune neuromuscular disease that commonly strikes people between the ages of 40 and 70 and afflicts between 50,000 and 85,000 people in the United States. Approximately 13,600 new cases of myasthenia gravis are diagnosed each year. Myasthenia gravis is a disease in which the immune system attacks the body's own tissues. The attack occurs at the junction between nerve and muscle and targets either the acetylcholine receptor, the part of a muscle cell that receives signals from a nerve cell or muscle-specific kinase, a protein that helps organize acetylcholine receptors on the muscle cell. The cause of myasthenia gravis is unclear. Researchers suspect viruses or bacteria might trigger the autoimmune response; the thymus gland also sometimes seems to play a role in the disease. The symptoms include fatigue and weakness of voluntary muscles, partial paralysis of eye movements, double vision, droopy eyelids, and weakness and fatigue in neck and jaws with problems in chewing, swallowing and holding up the head; this weakness can become more generalized. As myasthenia gravis progresses, weakness fluctuates each day but tends to spread and progress over the course of a few years, especially if untreated.

Development Status of CK-2017357

Recently, Cytokinetics announced data from its Phase IIa EoE clinical trial of CK-2017357 in patients with amyotrophic lateral sclerosis (ALS). 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. The investigators concluded that these single doses of CK-2017357 were 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 whether the patient was "better", "same", or "worse" compared to just before dosing on that day. Furthermore, there was a clear relationship between improvements in Global Assessments and the CK-2017357 plasma concentration. The investigators proposed that these 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. The investigators also proposed that trends toward increases in parameters of pulmonary function may underlie the improvements in the patients' and investigators' global assessments. 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. Trends to increase the patients' force of inhalation, a measure of pulmonary function known as the Sniff Inspiratory Pressure, were also observed at 6 hours after the 500 mg dose of CK-2017375. In addition, there was a relationship between increases in Sniff Inspiratory Pressure and increasing CK-2017375 plasma concentrations.

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 an ongoing Phase IIa EoE clinical trial in patients with claudication associated with peripheral artery disease and has opened enrollment of a Phase IIa EoE clinical trial in patients with MG. In July 2010, Cytokinetics was awarded a grant in the amount of $2.8 million by the National Institute of Neurological Disorders and Stroke to support research and development of CK-2017357 in MG. The majority of the MG development program costs are funded by the blank grant of $2.8 million provided by National Institute of Neurological Disorders and Stroke.

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 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, the potential size of markets 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 or MG. 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 exclusivity in ALS 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; funding from the National Institute of Neurological Disorders and Stroke may not be available in future periods; 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.

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