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): October 1, 2010

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 October 1, 2010, Cytokinetics, Incorporated issued a press release announcing that two abstracts relating to CK-2017357 were presented at the 2010 Annual Aging Muscle Symposium, held September 30 – October 1, 2010 in San Francisco, California.

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

October 1, 2010

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 October 1, 2010

Contact:

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

CYTOKINETICS ANNOUNCES PRESENTATION OF CLINICAL AND NON-CLINICAL DATA RELATING TO CK-2017357 AT THE 2010 ANNUAL AGING MUSCLE SYMPOSIUM

Results Provide Supportive Evidence for Ongoing Phase IIa Evidence of Effect Clinical Trials

South San Francisco, CA, October 1, 2010 – Cytokinetics, Incorporated (Nasdaq: CYTK) announced today that two abstracts relating to CK-2017357 were presented at the 2010 Annual Aging Muscle Symposium, held September 30 – October 1, 2010 in San Francisco, California.

"We are pleased to present these data in this forum as CK-2017357, and other activators of fast skeletal muscle, may have broad applicability as potential treatment for diseases and conditions associated with aging, muscle wasting or neuromuscular dysfunction," stated Fady Malik, MD, PhD, FACC, Cytokinetics' Vice President of Biology and Therapeutics. "Based on the Phase I clinical data supporting the translation of CK-2017357's mechanism of action into humans and the preclinical data presented at this meeting, Cytokinetics is conducting a Phase IIa Evidence of Effect clinical trial to test the hypothesis that CK-2017357 can delay the onset of muscle fatigue in patients with symptoms of claudication due to peripheral artery disease. This trial may provide insight into the potential role for activators of fast skeletal muscle in an aging population."

Presentations at the Annual Aging Muscle Symposium

The oral presentation titled "CK-2017357, a Novel Activator of Fast Skeletal Muscle, Increases Isometric Force Evoked by Electrical Stimulation of the Anterior Tibialis Muscle in Healthy Male Subjects" was presented yesterday by Richard Hansen, Ph.D., Cytokinetics, Inc., South San Francisco, CA. This presentation presented data from Part B of Cytokinetics' first-time-in-humans Phase I clinical trial of CK-2017357. The objective of Part B of this trial was to determine the change in the force-frequency profile of the tibialis anterior muscle and its relationship to the CK-2017357 plasma concentration after oral administration of CK-2017357 to healthy male volunteers. The authors concluded that CK-2017357 significantly increased the mean placebo-corrected normalized peak force produced in response to transcutaneous electrical stimulation of the tibialis anterior muscles of healthy volunteers in a dose-, concentration-, and frequency-dependent manner. The authors concluded that the mechanism of action of CK-2017357, as observed in preclinical models, can be translated into statistically significant and potentially clinically important increases in skeletal muscle performance in healthy volunteers. These results support further evaluation of CK-2017357 in neuromuscular diseases, such as amyotrophic lateral sclerosis (ALS), and other diseases or conditions associated with muscle weakness or fatigue, such as claudication associated with peripheral artery disease.

The poster presentation titled "The Fast Skeletal Troponin Activator, CK-2017357, Reduces Muscle Fatigue in an *in situ* Model of Vascular Insufficiency" is on display and will be presented later today by Aaron Hinken, Ph.D. of Cytokinetics, Inc. The objective of this study was to evaluate the effects of CK-2017357 on the time to fatigue in native skeletal muscle preparations *in vitro*, and in skeletal muscle *in situ* where blood supply and nervous input to the muscle was left intact. In addition, the time to fatigue *in situ* was examined after the blood supply was limited by occlusion of the femoral artery. The authors concluded that CK-2017357 increased the calcium-sensitivity of force production in skinned fast skeletal muscle, increased sub-maximal force developed in isolated fast skeletal muscle *in vitro*, and increased the time to overall fatigue after repetitive stimulation. Moreover, CK-2017357 increased the time to muscle fatigue *in situ* when blood supply was restricted by femoral artery ligation. The authors concluded that CK-2017357 ameliorated the increase in fatigability induced by vascular insufficiency *in situ* in a rodent model of claudication. These data are consistent with the mechanism of action of CK-2017357. These findings suggest that sensitization of the fast skeletal muscle troponin complex to calcium, as mediated by CK-2017357, has the potential to ameliorate muscle dysfunction induced by peripheral vascular insufficiency, such as exists in claudication.

Development Status of CK-2017357

CK-2017357 is 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 currently conducting two Phase IIa Evidence of Effect clinical trials: one in patients with ALS and one in patients with claudication associated with peripheral artery disease. In addition, the National Institute of Neurological Disorders and Stroke awarded the company a grant to support research and development of CK-2017357 directed to the potential treatment of myasthenia gravis.

Background on Peripheral Artery Disease and Claudication

Claudication is a symptomatic complication associated with peripheral artery disease (PAD). Claudication associated with PAD results primarily from atherosclerosis of arteries in the lower extremities. During walking or other exercise, patients suffering from claudication experience fatigue, aching, cramping, and burning pain in the legs due to impaired blood circulation and chronic changes in muscle metabolism. As many as 3 million people in the United States, including 5% of the population over age 70, are estimated to be affected by claudication due to PAD.

Background on Amyotrophic Lateral Sclerosis

Amyotrophic lateral sclerosis (ALS) is a progressive neurodegenerative disease that afflicts 20,000 to 30,000 people in the United States. Approximately 5,600 new cases of ALS are diagnosed each year. The average life expectancy of an ALS patient is approximately three to five years and only 10% of patients survive for more than 10 years. Death is usually due to respiratory failure because of diminished strength in the skeletal muscles responsible for breathing. Few treatment options exist for these patients, resulting in a high unmet need for new therapeutic options to address the symptoms and modify the disease progression of this grievous illness.

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' research and development activities, including the conduct, design and results of clinical trials and preclinical studies, the significance and utility of clinical trial and preclinical study results, and the properties and potential benefits of CK-2017357 and Cytokinetics' other drug candidates and potential 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, 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 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; the availability of funds under the National Institute of Neurological Disorders and Stroke grant is not assured; 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.