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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):

December 7, 2009

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 December 7, 2009, Cytokinetics, Incorporated issued a press release announcing that a poster and oral presentation summarizing non-clinical data regarding CK-2017357, a fast skeletal troponin activator, were presented at the 5th Cachexia Conference, organized by the Society on Cachexia and Wasting Disorders, being held December 5-8, 2009 in Barcelona, Spain. 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 December 7, 2009.

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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

*December 7, 2009*

*By: Sharon Barbari*

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

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Exhibit Index

<u>Exhibit No.</u>	<u>Description</u>
99.1	Press release, dated December 7, 2009

Contacts:

Cytokinetics, Incorporated  
Christopher S. Keenan (Investors and Media)  
Director, Investor Relations  
(650) 624-3000

**CYTOKINETICS PRESENTS NON-CLINICAL DATA  
RELATING TO CK-2017357  
AT THE 5TH CACHEXIA CONFERENCE**

*Results Support Therapeutic Hypotheses  
for Novel Mechanism Skeletal Muscle Activator Now Advancing Towards Phase II Clinical Trials*

**South San Francisco, CA, December 7, 2009** – Cytokinetics, Incorporated (Nasdaq: CYTK) announced today that both a poster and an oral presentation summarizing non-clinical data regarding CK-2017357, a novel fast skeletal troponin activator, were presented at the 5th Cachexia Conference, organized by the Society on Cachexia and Wasting Disorders, being held December 5-8, 2009 in Barcelona, Spain. CK-2017357 is currently the subject of two ongoing Phase I clinical trials.

“As a result of our focused R&D activities directed to the biology of muscle contractility, we have been able to identify and characterize compounds that activate the skeletal muscle sarcomere, such as CK-2017357, which may have treatment applications in syndromes such as cachexia, as well as other diseases and medical conditions associated with aging, muscle wasting or fatigue and neuromuscular dysfunction,” stated Fady Malik, MD, PhD, FACC, Cytokinetics’ Vice President of Biology and Therapeutics. “We are pleased to have had an opportunity to share these encouraging data at this leading scientific forum on cachexia and look forward to our planned advancement of CK-2017357 into Evidence of Effect studies in disease populations in 2010.”

**Oral Presentation at 5<sup>th</sup> Cachexia Conference**

An oral presentation titled “Direct Activation of the Skeletal Sarcomere by the Troponin Activator, CK-2017357, a Novel Approach to Improving Skeletal Muscle Function” was presented by Fady Malik, MD, PhD, FACC, Vice President, Biology and Therapeutics, Cytokinetics, Inc., South San Francisco, California on December 7, 2009. The presentation highlighted the therapeutic hypothesis underlying Cytokinetics’ skeletal muscle contractility drug discovery and development program. Following a description of the mechanism of action of CK-2017357, namely activation of the fast skeletal troponin complex resulting in sensitization of the sarcomere to calcium, the presentation described three basic pharmacological consequences of this mechanism of action. The presentation highlighted that in non-clinical studies, CK-2017357 and related skeletal troponin activators amplified the response to motor neuron input, increased muscle power and slowed the development of muscle fatigue. In addition, the presentation summarized non-clinical data indicating that CK-2017357 delayed the onset of muscle fatigue in a model of vascular insufficiency and increase muscle performance in a model of muscle endurance. Finally, the presentation reviewed the current development plan for CK-2017357 including a discussion of the two ongoing Phase I clinical trials of CK-2017357 in healthy volunteers designed to assess the safety, tolerability and pharmacokinetics of single doses and multiple doses, respectively, as well as planned Phase II Evidence of Effect clinical trials in patients with neuromuscular disease or medical conditions associated with muscle wasting or fatigue, several of which are expected to be conducted in 2010.

**Poster Presentation at 5<sup>th</sup> Cachexia Conference**

A poster presentation titled “The Fast Skeletal Troponin Activator, CK-2017357, Increases Skeletal Muscle Force *in-vitro* and *in-situ*” was presented on December 6, 2009 – December 7, 2009 at the 5<sup>th</sup> Cachexia Conference. The objective of the study was to evaluate whether CK-2017357 changes force development in native skeletal muscle preparations *in vitro*, using skinned and living skeletal muscle fibers, and *in situ*, where nerve and blood supply are left intact. The authors demonstrated that CK-2017357 increased sub-maximal force development of skinned fast skeletal muscle *in vitro*. Similar findings were observed in human skinned fast skeletal muscle fibers. In addition, compound specificity for fast skeletal muscle fibers was demonstrated, as skinned slow skeletal muscle fibers were approximately ten-fold less responsive to CK-2017357 than skinned fast skeletal muscle fibers. CK-2017357 did not appear to activate cardiac muscle fibers. CK-2017357 increased the force development in living muscle fibers at sub-maximal stimulation frequencies. *In situ*, CK-2017357 increased sub-maximal force development in a predominantly fast skeletal fiber muscle (extensor digitorum longus). Finally, CK-2017357 was shown to reduce isometric fatigue in living skeletal muscle *in vitro*. This effect is hypothesized to be linked to the reduced stimulation frequency required to elicit the same starting force in treated muscle.

The authors concluded that these data support a proposed mechanism of action of CK-2017357 through calcium sensitization of the fast skeletal muscle troponin complex. In skinned muscle fibers, CK-2017357 increased the sensitivity of skeletal muscle to exogenously added calcium. In living muscle fibers, CK-2017357 increased the sensitivity of skeletal muscle to the frequency of electrical stimulation which results in calcium release within the muscle. In each case, the result was an increase in muscle force development at sub-maximal muscle activation, where muscle normally operates. The ability to generate the same force at lower frequencies of nerve input may result in a resistance to fatigue. These findings may translate into functional improvements in skeletal muscle performance and efficiency in conditions marked by muscle weakness by improving the extent of muscle fiber recruitment during physical activity and reducing the rate at which muscle fibers fatigue.

**Development Status of CK-2017357**

In June 2009, Cytokinetics announced that it had initiated a first-time-in-humans Phase I clinical trial of CK-2017357 in healthy male volunteers. The ongoing first part, or “Part A,” of this trial is designed to assess the safety, tolerability, and pharmacokinetic profile of this drug candidate and to determine its maximum tolerated dose and plasma concentration. In November 2009, Cytokinetics announced that the company had initiated the second part, or “Part B,” of this clinical trial. Part B is designed to evaluate the pharmacodynamic effect of single doses that have been tolerated to date in Part A of this trial. In November 2009, the company also announced the initiation of a second Phase I clinical trial to investigate the safety, tolerability and pharmacokinetic profile of CK-2017357 after multiple oral doses to steady state in healthy male volunteers.

**About Cytokinetics**

Cytokinetics is a clinical-stage biopharmaceutical company focused on the discovery and development of 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, *omecantiv mecarbil* (formerly CK-1827452), is in Phase II clinical development for the potential treatment of heart failure. Amgen Inc. holds an exclusive license worldwide (excluding Japan) to develop and commercialize *omecantiv 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 in Phase I clinical development. 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 such as systemic hypertension, pulmonary arterial hypertension or bronchoconstriction. In addition, prior Cytokinetics' research generated three anti-cancer drug candidates in Phase I clinical development: *ispinesib*, SB-743921 and GSK-923295. Cytokinetics is seeking a partner for *ispinesib* and SB-743921. GSK-923295 is being developed by GlaxoSmithKline in collaboration with Cytokinetics. 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](http://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 results of non-clinical data relating to CK-2017357, the significance and utility of such results and the planned initiation and conduct of Evidence of Effect clinical trials for CK-2017357, 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, and Cytokinetics may be unable to obtain or maintain patent or trade secret protection for its intellectual property; Amgen's and GlaxoSmithKline's decisions with respect to the design, initiation, conduct, timing and continuation of development activities for omecamtiv mecarbil and GSK-923295, respectively; 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; others may introduce products or alternative therapies 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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