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): November 17, 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 November 17, 2009, Cytokinetics, Incorporated issued a press release announcing that three abstracts summarizing non-clinical data regarding its smooth muscle contractility program were presented at the 2009 Scientific Sessions of the American Heart Association in Orlando, Florida. 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 November 17, 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

November 17, 2009

By: /s/ Sharon Barbari

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

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Exhibit Index

Exhibit No.	Description
99.1	Press release, dated November 17, 2009

Contacts:

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

**CYTOKINETICS PRESENTS NON-CLINICAL DATA
FROM ITS SMOOTH MUSCLE CONTRACTILITY PROGRAM
AT THE 2009 SCIENTIFIC SESSIONS OF THE AMERICAN HEART ASSOCIATION**

***Data Supports Hypothesis that Inhibition of Smooth Muscle Myosin
May Represent a Novel Approach to Treat Patients with Systemic or Pulmonary Hypertension***

South San Francisco, CA, November 17, 2009 – Cytokinetics, Incorporated (Nasdaq: CYTK) announced today that three abstracts summarizing non-clinical data regarding its smooth muscle contractility program were presented at the 2009 Scientific Sessions of the American Heart Association in Orlando, Florida.

"Our smooth muscle contractility program leverages our expertise in the contractile apparatus of muscle systems, which has generated clinical stage drug candidates in both our cardiac and skeletal muscle programs," stated David J. Morgans, PhD Cytokinetics' Executive Vice President of Preclinical Research and Development. "We are pleased to share these three presentations relating to our novel mechanism approach for modulation of smooth muscle contractility. We believe that these data demonstrate the potential of smooth muscle myosin inhibitors for the treatment of patients with diseases that may cause significant morbidity, such as refractory systemic hypertension and pulmonary arterial hypertension."

Oral Presentations

An oral presentation titled "Inhibition of Smooth Muscle Myosin, a Novel Anti-Hypertensive Strategy" was presented on Monday, November 16, 2009 by Fady Malik, MD, PhD, FACC, Vice President, Biology and Therapeutics, Cytokinetics, Inc., South San Francisco, California. This presentation summarized non-clinical research evaluating smooth muscle myosin inhibitors in models of hypertension. The authors concluded that smooth muscle myosin inhibition reduced vascular resistance and lowered blood pressure in a canine model of hypertension. In addition, the pattern of regional vasodilation is different for a specific smooth muscle myosin inhibitor, CK-2018448, as compared to the calcium channel blocker, *amlodipine*. For CK-2018448, blood flow to the kidney increased without change in blood flow to the limb. In contrast, for *amlodipine*, the largest increases in blood flow were found in the limb. The authors concluded that the smooth muscle myosin inhibitor and the calcium channel blocker elicited different patterns of regional vasodilation. In addition, the renal vasodilation induced by smooth muscle myosin inhibition could have salutary effects in conditions accompanied by renal insufficiency such as hypertension, acute heart failure, and acute renal failure due to an interruption of adequate blood flow to the kidney.

An oral presentation titled "A Direct Inhibitor of Smooth Muscle Myosin as a Novel Therapeutic Approach for the Treatment of Pulmonary Artery Hypertension" was presented on Tuesday, November 17, 2009 by Malarvannan Pannirselvam, M.V.Sc., Ph.D., Scientist, Cytokinetics, Inc., South San Francisco, California. This presentation summarized a non-clinical study designed to test the hypothesis that direct inhibition of smooth muscle myosin should provide a novel and effective means of relaxing vascular smooth muscle. In this study, the authors concluded that CK-2018571, a smooth muscle myosin inhibitor, selectively inhibited the ATPase activity of smooth muscle myosin and relaxed contracted arteriovascular tissue rings as a consequence of direct inhibition of smooth muscle myosin. In addition, CK-2018571 relaxed pulmonary arterial rings from MCT-PAH rats suggesting its potential use as a vasodilator in pulmonary arterial hypertension. Finally, an active pro-drug of CK-2018571, CK-2019165, decreased the elevated right ventricular systolic pressure in two animal models of pulmonary arterial hypertension. The authors concluded that these data together support the hypothesis that direct inhibition of smooth muscle myosin could be a novel therapeutic approach for the treatment of pulmonary arterial hypertension.

Poster Presentation

A poster presentation titled "Inhibition of Smooth Muscle Myosin, a Novel Therapeutic Approach for Pulmonary Hypertension" was presented on Tuesday, November 17, 2009 by David Ho, MD, CV Dynamics and the Department of Cell Biology and Molecular Medicine and Cardiovascular Research Institute, New Jersey Medical School, University of Medicine and Dentistry of New Jersey, Newark, NJ from 9:30 AM – 11:00 AM Eastern Time. This poster summarized a non-clinical study designed to establish a porcine model of acute pulmonary arterial hypertension, by either hypoxia or administration of a thromboxane analog, and to examine the extent to which the inhibition of smooth muscle myosin by CK-2019165 administered either intravenously or by inhalation is able to ameliorate pulmonary hypertension in chronically instrumented pigs. The authors concluded that CK-2019165, delivered intravenously at a dose of 4 mg/kg, reduced ($p<0.01$) the increased pulmonary vascular resistance in both hypoxia and thromboxane models, respectively. Mean arterial pressure fell modestly and heart rate rose slightly in the conscious state. In addition, in the hypoxia model, CK-2019165 was delivered via inhalation and the increased pulmonary vascular resistance fell similarly while mean arterial pressure and heart rate were unchanged. The maximum extent of vasodilation was similar to that produced with sodium nitroprusside. The authors concluded that inhibition of smooth muscle myosin may be a novel therapeutic approach to the treatment of pulmonary arterial hypertension.

Background on Cytokinetics Smooth Muscle Contractility Program

In January 2009, Cytokinetics announced the selection of a small molecule inhibitor of smooth muscle myosin for development. Cytokinetics' smooth muscle research program is directed to smooth muscle myosin, the motor protein responsible for the contraction of the smooth muscle cells that surround airways in the lungs and the blood vessels that control blood pressure. By inhibiting the function of the myosin motor central to the contraction of smooth muscle, these potent small molecules directly lead to the relaxation of contracted smooth muscle. Cytokinetics' smooth myosin inhibitors have demonstrated encouraging pharmacological activity in preclinical models that may relate to uses for the potential treatment of diseases such as systemic hypertension, pulmonary arterial hypertension, asthma and chronic obstructive pulmonary disease (COPD). Cytokinetics continues to progress smooth muscle myosin inhibitors in non-clinical development activities.

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, *omecamtiv 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 *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 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 and GSK-923295 is being developed under Cytokinetics' collaboration with GlaxoSmithKline. 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 Safe Harbor for forward-looking statements contained in the Act. Examples of such statements include, but are not limited to, statements relating to the initiation, scope, design, conduct and results of Cytokinetics' research and development programs, including the significance of results of non-clinical studies relating to Cytokinetics' smooth muscle myosin inhibitors, and the potential benefits of Cytokinetics' drug candidates and potential drug candidates, including the potential utility of its smooth muscle myosin inhibitors for the treatment of pulmonary arterial hypertension, systemic hypertension and other diseases. 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; 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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