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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 9, 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 9, 2009, Cytokinetics, Incorporated issued a press release announcing that it has agreed with GlaxoSmithKline (GSK) to terminate their collaboration and license agreement effective, February 28, 2010. As a result, all rights for GSK-923295, an inhibitor of centromere-associated protein E (CENP-E), will revert to Cytokinetics effective February 28, 2010. GSK remains responsible for all activities and costs associated with completing and reporting on the ongoing Phase I clinical trial of GSK-923295 in advanced, refractory solid tumor patients.

A copy of the press release is being 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 Exhibits are filed as part of this Current Report on Form 8-K:

Exhibit No. Description

99.1 Press Release, dated December 9, 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 9, 2009

By: /s/ 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 9, 2009

Contacts:

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

**CYTOKINETICS AND GLAXOSMITHKLINE AGREE  
TO TERMINATE COLLABORATION AND LICENSE AGREEMENT DIRECTED TO ONCOLOGY**

***Decision Consistent with Cytokinetics' Corporate Strategy to Focus  
Research and Development on Muscle Biology***

**South San Francisco, CA, December 9, 2009** – Cytokinetics, Incorporated (Nasdaq: CYTK) announced that it has agreed with GlaxoSmithKline (GSK) to terminate their collaboration and license agreement, effective February 28, 2010. As a result, all rights for GSK-923295, an inhibitor of centromere-associated protein E (CENP-E), will revert to Cytokinetics effective February 28, 2010. GSK remains responsible for all activities and costs associated with completing and reporting on the ongoing Phase I clinical trial of GSK-923295 in advanced, refractory solid tumor patients.

The decision to terminate the collaboration agreement with GSK reinforces Cytokinetics' corporate commitment to focus its internal research and development to muscle function and related therapeutic applications. Previously, Cytokinetics had negotiated for the return of all rights to the kinesin spindle protein (KSP) inhibitors, SB-743921 and *ispinesib* (SB-715992), that had also been initially developed in collaboration with GSK. Each of these compounds has demonstrated favorable tolerability and clinical activity in one or more clinical trials. Cytokinetics is now seeking to license its portfolio of these three novel mechanism anti-mitotic drug candidates so that they can be advanced in further clinical trials.

"In our industry and especially in these challenging times, it is increasingly important to remain focused and execute on a core business strategy. At Cytokinetics, we believe our best opportunities are rooted in our multiple programs directed to the biology of muscle function. Today's announcement, combined with our company's previously disclosed decisions to discontinue oncology research and phase out related development activities and spending, is further evidence of our commitment to this strategy," stated Robert I. Blum, Cytokinetics' President and Chief Executive Officer. "We appreciate the opportunity we have had to collaborate with GlaxoSmithKline over the last eight years and we look forward to the possibility of advancing these novel oncology drug candidates into next stages of clinical development with another partner."

"Our collaboration with GlaxoSmithKline has been productive in validating the potential of mitotic kinesin inhibitors in both solid and hematologic malignancies," stated Andrew A. Wolff, MD, FACC, Cytokinetics' Senior Vice President of Clinical Research and Development and Chief Medical Officer. "We are encouraged by both the tolerability profiles and the clinical activity demonstrated by our three novel drug candidates in the treatment of a broad array of tumor types."

**Development Status of GSK-923295**

In October 2008, GSK reported interim data from a Phase I dose-escalation and pharmacokinetic study of GSK-923295 in adult patients with solid tumors. The primary objective of this trial is to determine the maximum-tolerated dose (MTD), dose-limiting toxicity (DLT), safety and pharmacokinetics of GSK-923295 in advanced, refractory solid tumors. In this clinical trial, the authors concluded that GSK-923295 was well-tolerated at doses evaluated to date, which ranged from 10-105 mg/m<sup>2</sup>. Of the adverse events observed, nausea and fatigue (all less than or equal to grade 2) were the most frequent non-hematological toxicities, and anemia (all less than or equal to grade 2) was the most frequent hematological toxicity. In addition, no neurotoxicity was observed. The MTD had not been reached but one DLT was observed in the form of reversible aspartate aminotransferase (AST) elevation. Finally, the authors concluded that the plasma pharmacokinetics of GSK-923295 observed were dose-proportional and exhibited low intra-patient and modest inter-patient variability.

**Development Status of SB-743921**

In December 2009, at the American Society of Hematology Annual Meeting, Cytokinetics reported data from the Phase I portion of a Phase I/II clinical trial of SB-743921 designed to evaluate the safety, tolerability, pharmacodynamics and pharmacokinetic profile of this novel inhibitor of KSP administered as a one-hour intravenous infusion on days 1 and 15 of a 28-day schedule, and to assess the potential efficacy and the MTD of SB-743921 administered on this schedule in patients with Hodgkin or non-Hodgkin lymphoma. The authors concluded that the MTD of SB-743921 given on this schedule with G-CSF support was 9 mg/m<sup>2</sup>. The main DLTs of SB-743921 on this schedule with G-CSF support were thrombocytopenia and neutropenia. The authors noted that a greater dose-density was achieved with SB-743921 given on a once every two week schedule without prophylactic G-CSF (i.e., 6 mg/m<sup>2</sup> = 0.43 mg/m<sup>2</sup>/day) than a once every 21 days schedule without prophylactic G-CSF (i.e., 4 mg/m<sup>2</sup> = 0.19 mg/m<sup>2</sup>/day). Dose-density with G-CSF on the once every two week schedule was equal to 0.64 mg/m<sup>2</sup>. Grade 3 or 4 toxicities other than myelosuppression were infrequent; in particular, there was no evidence of neuropathy or alopecia greater than Grade 1. An efficacy signal was observed at doses at or above 6 mg/m<sup>2</sup> in Hodgkin lymphoma patients. Of the 55 patients evaluable for efficacy, four partial responses (three patients with Hodgkin lymphoma and one with indolent non-Hodgkin lymphoma) were observed. The duration of the response in the patients with a partial response was between 8 weeks and 28 weeks. Best response as a percentage reduction in the sum of the product of diameters for the dominant lesion ranged from 53% to 71%.

**Development Status of *Ispinesib***

In June 2009, at the American Society of Clinical Oncology, Cytokinetics presented interim results from the Phase I portion of its Phase I/II clinical trial of *ispinesib*, an inhibitor of KSP. These data demonstrated that *ispinesib* was well-tolerated when administered as a 1-hour intravenous infusion on days 1 and 15 of a 28-day cycle, with the most frequent adverse event being neutropenia. At that time, it was announced that one Response Evaluation Criteria in Solid Tumors (RECIST)-confirmed partial response (PR) with duration of 6 months had been reported. Also, two additional patients had a reduction in tumor burden of 30% or more as their best response. Finally, 10 additional patients experienced stable disease without reaching PR criteria; four of these responses lasted 4 months or longer. The authors also noted that protocol-defined DLTs of Grade 3 ALT/AST increases with a questionable relationship to *ispinesib* were observed in two out of seven patients treated at the 14 mg/m<sup>2</sup> dose level. The protocol has been amended to permit additional dose escalation beyond the 14 mg/m<sup>2</sup> dose level.

**Background on Mitotic Kinesin Inhibitors**

Since their introduction over 40 years ago, anti-mitotic drugs (taxanes and vinca alkaloids) have advanced the treatment of cancer and are commonly used for the treatment of several tumor types. However, these drugs have demonstrated limited treatment benefit against certain cancers. In addition, they target

tubulin, a cytoskeletal protein involved not only in mitosis and cell proliferation, but also in other important cellular functions. Inhibition of these other cellular functions produces dose-limiting toxicities such as peripheral neuropathy, an impairment of peripheral nervous system function. Neuropathies are thought to result when these drugs interfere with the dynamics of microtubule filaments that are responsible for the long-distance transport of important cellular components within nerve cells.

Mitotic kinesins are essential to mitosis, and, unlike tubulin, are not believed to be present in non-dividing cells. Cytokinetics believes that drugs that inhibit CENP-E, KSP and other mitotic kinesins may represent the next generation of anti-mitotic cancer drugs by arresting mitosis and cell proliferation without impacting unrelated, normal cellular functions, thereby avoiding many of the toxicities commonly experienced by patients treated with existing anti-mitotic drugs.

CENP-E plays an essential role in chromosome movement during early mitosis and integrates mitotic spindle mechanics with regulators of the mitotic checkpoint; hence CENP-E is directly involved in coupling the mechanics of mitosis with the mitotic checkpoint signaling machinery, regulating cell-cycle transition from metaphase to anaphase. CENP-E is also essential for prometaphase chromosome movements that contribute to metaphase chromosome alignment. These processes are essential to cell proliferation. CENP-E is expressed exclusively in proliferating cells and is abundant during mitosis; it is absent from non-proliferating cells, including neurons. Inhibition of CENP-E induces cell cycle arrest in mitosis with bipolar mitotic spindles and misaligned chromosomes leading to subsequent programmed cell death, or apoptosis.

KSP is a mitotic kinesin which acts at the earliest stage of spindle formation. Early in mitosis, during prophase, KSP forces the emerging spindle poles to move apart, driving formation of a bipolar spindle and enabling chromosome segregation into two resultant daughter cells. KSP is not expressed in neurons and has only one known function, to drive spindle pole separation during mitosis. Inhibition of KSP motor function prevents formation of a bipolar spindle. KSP inhibition results in cell cycle arrest in mitosis with a characteristic monopolar spindle on which chromosomes are arrayed. In cancer cells, duplicated chromosomes remain attached to this monopolar spindle in a persistent state of cell cycle arrest, resulting in apoptosis.

### **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*, 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](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 the reversion of rights to GSK-923295 to Cytokinetics; Cytokinetics' plans to seek a partner for its oncology drug candidates and the advancement of those drug candidates in clinical development by such partner; Cytokinetics' and its partners' research and development activities, including clinical trial results and the significance and utility of such results; the utility of Cytokinetics' strategic focus on the biology of muscle function; and the properties and potential benefits of Cytokinetics' mitotic kinesin inhibitors and 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; 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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