Cyclacel Pharmaceuticals, Inc.
Form S-3/A
February 12, 2007
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As filed with the Securities and Exchange Commission on February 12, 2007

Registration No. 333-140034

UNITED STATES
SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

Pre-Effective Amendment No. 2

FORM S-3/A

REGISTRATION STATEMENT
UNDER THE SECURITIES ACT OF 1933

CYCLACEL PHARMACEUTICALS, INC.

(Exact name of registrant as specified in its charter)

Delaware	91-1707622
(State or other jurisdiction of incorporation or organization)	(I.R.S. Employer Identification Number)

200 Connell Drive, Suite 1500
Berkeley Heights, NJ 07922
(908) 517-7330

(Address, including zip code, and telephone number, including area code, of registrant's principal executive offices)

Spiro Rombotis
Chief Executive Officer
Cyclacel Pharmaceuticals, Inc.
200 Connell Drive, Suite 1500
Berkeley Heights, NJ 07922
(908) 517-7330

(Name, address, including zip code, and telephone number, including area code, of agent for service)

With a copy to:
Joel I. Papernik, Esq.
Mintz, Levin, Cohn, Ferris, Glovsky and Popeo, P.C.
The Chrysler Center
666 Third Avenue
New York, New York 10017
(212) 935-3000

Approximate date of commencement of proposed sale to the public: From time to time after this Registration Statement becomes effective.

If the only securities being registered on this Form are being offered pursuant to dividend or interest reinvestment plans, please check the following box.

If any of the securities being registered on this Form are to be offered on a delayed or continuous basis pursuant to Rule 415 under the Securities Act of 1933, other than securities offered only in connection with dividend or interest reinvestment plans, check the following box.

If this Form is filed to register additional securities for an offering pursuant to Rule 462(b) under the Securities Act, please check the following box and list the Securities Act registration statement number of the earlier effective registration statement for the same offering.

If this Form is a post-effective amendment filed pursuant to Rule 462(c) under the Securities Act, please check the following box and list the Securities Act registration statement number of the earlier effective registration statement for the same offering.

If this Form is a registration statement pursuant to General Instruction I.D. or a post-effective amendment thereto that shall become effective upon filing with the Commission pursuant to Rule 462(e) under the Securities Act, check the following box.

If this Form is a post-effective amendment to a registration statement filed pursuant to General Instruction I.D. filed to register additional securities or additional classes of securities pursuant to Rule 413(b) under the Securities Act, check the following box.

CALCULATION OF REGISTRATION FEE

Title of each Class of Securities to be Registered[1]	Proposed Maximum Aggregate Offering Price[2][3]	Amount of Registration Fee[4]
Common Stock, $0.001 par value per share	(5)	(5)
Preferred Stock, $0.001 par value per share	(5)	(5)
Warrants	(5)	(5)
Debt Securities	(5)	(5)
Total	$75,000,000	$8,025*

* Previously Paid

(1)

There are being registered hereunder such indeterminate number of shares of common stock, such indeterminate number of shares of preferred stock, such indeterminate number of warrants to purchase common stock, and such indeterminate number of debt securities as shall have an aggregate initial offering price not to exceed $75,000,000. If any debt securities are issued at an original issue discount, then the offering price of such debt securities shall be in such greater principal amount as shall result in an aggregate initial offering price not to exceed $75,000,000, less the aggregate dollar amount of all securities previously issued hereunder. Any securities registered hereunder may be sold separately or as units with other securities registered hereunder. The securities registered also include such indeterminate amounts and numbers of common stock as may be issued upon conversion of preferred stock or pursuant to the antidilution provisions of any such securities. The securities registered also include such indeterminate amounts and numbers of common stock as may be issued upon exercise of warrants or pursuant to the antidilution provisions of any such securities. The securities registered also include such indeterminate amounts and numbers of common stock and debt securities as may be issued upon conversion of or exchange for debt securities that provide for conversion or exchange, upon exercise of warrants or pursuant to the anti-dilution provisions of any such securities.

(2) In United States dollars or the equivalent thereof in any other currency, currency unit or units, or composite currency or currencies.

(3) The proposed maximum per unit and aggregate offering prices per class of security will be determined from time to time by the Registrant in connection with the issuance by the Registrant of the securities registered hereunder.

(4) Estimated solely for purposes of determining the registration fee pursuant to Rule 457(o) under the Securities Act of 1933, as amended.

(5) Not required to be included in accordance with General Instruction II.D of Form S-3.

The Registrant hereby amends this Registration Statement on such date or dates as may be necessary to delay its effective date until the company shall file a further amendment which specifically states that this Registration Statement shall thereafter become effective in accordance with Section 8(a) of the Securities Act of 1933 or until the Registration Statement shall become effective on such date as the Commission, acting pursuant to said Section 8(a), shall determine.

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The information in this prospectus is not complete and may be changed. We may not sell these securities until the registration statement filed with the Securities and Exchange Commission is effective. This prospectus is not an offer to sell these securities and is not soliciting an offer to buy these securities in any state where the offer or sale is not permitted.

SUBJECT TO COMPLETION, DATED FEBRUARY 12, 2007

PROSPECTUS

CYCLACEL PHARMACEUTICALS, INC.

$75,000,000

COMMON STOCK

PREFERRED STOCK

WARRANTS

DEBT SECURITIES

We may, from time to time, issue up to $75,000,000 aggregate principal amount of common stock, preferred stock, warrants and/or debt securities. We will specify in an accompanying prospectus supplement the terms of the securities. We may sell these securities to or through underwriters and also to other purchasers or through agents. We will set forth the names of any underwriters or agents in the accompanying prospectus supplement.

Our common stock is quoted on the Nasdaq Global Market under the symbol ‘‘CYCC.’’ On February 9, 2007, the last reported sale price of our common stock was $8.12 per share. Our preferred stock is quoted on the Nasdaq Capital Market under the symbol ‘‘CYCCP.’’ On February 9, 2007, the last reported sale price of our preferred stock was $5.30 per share.

Investing in our securities involves risks.
See ‘‘Risk Factors’’ on page 7.

Neither the Securities and Exchange Commission nor any state securities commission has approved or disapproved of these securities or passed upon the accuracy or adequacy of this prospectus. Any representation to the contrary is a criminal offense.

This prospectus may not be used to consummate sales of securities unless it is accompanied by a prospectus supplement.

The date of this prospectus is February , 2007.

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ABOUT THIS PROSPECTUS

This prospectus is part of a registration statement that we filed with the Securities and Exchange Commission, or SEC, utilizing a ‘‘shelf’’ registration process. Under this shelf process, we may sell any combination of the securities described in this prospectus in one or more offerings up to a total dollar amount of $75,000,000. We have provided to you in this prospectus a general description of the securities we may offer. Each time we sell securities, we will provide a prospectus supplement that will contain specific information about the terms of that offering. In any applicable prospectus supplements, we may add to, update or change any of the information contained in this prospectus.

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PROSPECTUS SUMMARY

The following is only a summary. We urge you to read the entire prospectus, including the more detailed consolidated financial statements, notes to the consolidated financial statements and other information included herein or incorporated by reference from our other filings with the SEC. Investing in our securities involves risks. Therefore, please carefully consider the information provided under the heading ‘‘Risk Factors’’ starting on page 7.

Our Business

We are a development-stage biopharmaceutical company dedicated to the discovery, development and eventual commercialization of novel, mechanism-targeted drugs to treat human cancers and other serious disorders. Our core area of expertise is in cell cycle biology, or the processes by which cells divide and multiply. We focus primarily on the discovery and development of orally available anticancer agents that target the cell cycle with the aim of slowing the progression or shrinking the size of tumors, and enhancing quality of life and improving survival rates of cancer patients. We have been focused on the cell cycle since our inception. We were founded in 1996 by Professor Sir David Lane, a recognized leader in the field of tumor suppressor biology who discovered the p53 protein, which operates as one of the body’s own anticancer ‘‘drugs’’ by inhibiting cell cycle targets. In 1999, we were joined by Professor David Glover, a recognized leader in the mechanism of mitosis or cell division who discovered, among other cell cycle targets, the mitotic kinases, Polo and Aurora, enzymes that act in the mitosis phase of the cell cycle. Our expertise in cell cycle biology is at the center of our business strategy.

We are generating several families of anticancer drugs that act on the cell cycle including cyclin dependent kinase (CDK) and Aurora kinase inhibitors. Although a number of pharmaceutical and biotechnology companies are currently attempting to develop CDK inhibitor drugs, we believe that our lead drug candidate, seliciclib, is the only orally available CDK inhibitor drug candidate currently in Phase II trials.

We are advancing three of our anticancer drug candidates, seliciclib, sapacitabine and CYC116 through in-house research and development activities. In addition we are progressing further novel drug series, principally for cancer, which are at earlier stages. Taken together, our pipeline covers all four phases of the cell cycle, which we believe will improve the chances of successfully developing and commercializing novel drugs that work on their own or in combination with approved conventional chemotherapies or with other targeted drugs to treat human cancers.

Our lead drug candidate, seliciclib, is a novel, first-in-class, orally available, CDK inhibitor. The compound selectively inhibits a spectrum of enzyme targets – CDK2/E, CDK2/A, CDK7 and CDK9 – that are central to the process of cell division and cell cycle control. Preclinical studies have shown that the drug works by inducing cell apoptosis, or cell suicide, in multiple phases of the cell cycle. To date, seliciclib has been evaluated in approximately 240 patients in several Phase I and II uncontrolled studies and has shown early signs of anti-cancer activity.

We have completed two Phase I trials that enrolled 24 healthy volunteers and three Phase I trials that enrolled a total of 84 cancer patients testing different doses and schedules. The primary toxicities observed were of a non-hematological nature including asthenia or weakness, elevation of liver enzymes, hypokalemia or decreased potassium levels, nausea and vomiting and elevation in creatinine. Although these trials were designed to test safety rather than efficacy of seliciclib given alone as monotherapy in patients with solid tumors who failed multiple previous treatments, several of these patients appeared to have benefited from seliciclib treatment.

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Seliciclib was shown in a further Phase I study sponsored and conducted by independent investigators to have clinical antitumor activity in patients with nasopharyngeal cancer, measured as a decrease in the size of primary tumor and involved lymph nodes, as well as an increase in tumor cell deaths by biomarker analyses. Four Phase II trials have been conducted in cancer patients to evaluate the tolerability and antitumor activities of seliciclib alone or in combination with standard chemotherapies used in the treatment of advanced non-small cell lung cancer, or NSCLC, or breast cancer. Interim data from two Phase II open-label studies of a total of 54 patients with NSCLC, suggest that seliciclib treatment did not aggravate the known toxicities of standard first and second-line chemotherapies nor appear to cause unexpected toxicities, although these trials were not designed to provide statistically significant comparisons. The combination of seliciclib with standard dose of capecitabine was not well tolerated in patients with advanced breast cancer. The Phase II open-label trials of seliciclib have been closed and we expect to report final data within the first quarter of 2007.

Based on our observations of tolerability and antitumor activity of seliciclib in the clinical trials conducted to date, the oral availability of seliciclib, the recommendation of a NSCLC expert panel, and regulatory and marketing considerations, seliciclib is currently being evaluated in the APPRAISE trial, a Phase IIb randomized double-blinded study to evaluate the safety and efficacy of the drug as a third line treatment in patients with NSCLC. The trial, which is expected to enroll approximately 200 patients, is using a randomized discontinuation trial design. We have retained worldwide rights to commercialize seliciclib.

Our second drug candidate, sapacitabine, is an orally available prodrug of CNDAC, which is a novel nucleoside analog, or a compound with a structure similar to a nucleoside. A prodrug is a compound that has a therapeutic effect after it is metabolized within the body. CNDAC has a significantly longer residence time in the blood when it is produced in the body through metabolism of sapacitabine than when it is given directly. Sapacitabine acts through a dual mechanism whereby the compound interferes with DNA synthesis by causing single-strand DNA breaks and induces arrest of the cell division cycle at G2 phase. A number of nucleoside drugs, such as gemcitabine, or Gemzar®;

Eli Lilly, are in wide use as conventional chemotherapies. Both sapacitabine and its major metabolite, CNDAC, have demonstrated potent anti-tumor activity in both blood and solid tumors in preclinical studies. In a liver metastatic mouse model, sapacitabine was shown to be superior to gemcitabine or 5-FU, two widely used nucleoside analogs, in delaying the onset and growth of liver metastasis.

Two Phase I studies of sapacitabine have been completed in the United States by Sankyo, from which we in-licensed sapacitabine, evaluating 87 patients in refractory solid tumors. A Phase Ib dose escalation clinical trial is currently in progress in the United States for the treatment of patients with refractory solid tumors or lymphomas. Preliminary results from this study were reported at the 18th EORTC-NCI-AACR Molecular Targets and Cancer Therapeutics meeting in November 2006. The primary objective of the study was to evaluate the safety profile of sapacitabine administered twice daily for 14 consecutive days or 7 consecutive days every 21 days. Of the 37 treated patients, 28 received the drug twice daily for 14 days and 9 received the drug twice daily for 7 days. The dose-limiting toxicity was reversible myelosuppression. One patient treated at the maximum tolerated dose died of candida sepsis in the setting of grade 4 neutropenia and thrombocytopenia. Non-hematological toxicities were mostly mild to moderate. The best response by investigator assessment was stable disease in 13 patients with five with NSCLC, two with breast cancer, two with ovarian cancer and one each with colorectal cancer, adenocarcinoma of unknown primary, gastrointestinal stroma tumor and parotid acinar carcinoma. The primary toxicity was reversible myelosuppression.

Sapacitabine is also currently being evaluated in a Phase I clinical trial in advanced leukemias and myelodysplastic syndromes, or MDS. The Phase I study is being conducted by Dr. Hagop Kantarjian, Professor of Medicine and Chairman of the Leukemia Department at M.D. Anderson Cancer Center in Houston, Texas. The study's primary objective is to determine the maximum tolerated dose, or MTD, of sapacitabine administered twice daily, or b.i.d., by mouth for

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seven consecutive days every 21 days. As of November 2006, 26 patients were enrolled and 25 patients have received at least one dose of sapacitabine. Preliminary interim data are available on 22 patients, of which nine had de novo acute myelogenous leukemia, or AML; seven had AML preceded by MDS; three had MDS-refractory anemia with excess blasts, or MDS-RAEB; and one each had treatment-related AML, acute lymphocytic leukemia, or ALL and chronic lymphocytic leukemia or CLL. The median age is 62 ranging from 39 to 91. Twenty-one patients received prior chemotherapy and one elderly patient (aged 91) did not receive any prior chemotherapy. The median number of prior chemotherapy regimens is two, ranging from one to four. Fifteen patients were previously treated with Ara-C-containing regimens of which nine had de novo AML and six had AML preceded by MDS. Six patients were previously treated with decitabine of which three had MDS-RAEB, and one each had de novo AML, AML preceded by MDS, and treatment-related AML. One patient treated at the dose level of 275 mg b.i.d. experienced a dose limiting toxicity, or DLT consisting of Grade 3 diarrhea and Grade 3 neutropenic colitis, which resolved after cessation of dosing and medical treatment. No DLTs were reported in the remaining five patients treated at 275 mg b.i.d. Dose escalation continues and the MTD has not been reached at the dose level of 325 mg b.i.d., which is approximately four times the recommended Phase II dose for solid tumor patients. To date, the best response to sapacitabine was reduction in bone marrow blast counts to 5% or less, which was observed in seven patients of which three had de novo AML, two had AML preceded by MDS, and two had MDS-RAEB. We expect to start Phase II evaluation of sapacitabine in 2007. We have retained worldwide rights to commercialize sapacitabine with the exception of Japan where Sankyo has a right of first refusal to market the drug under terms to be negotiated.

We have selected CYC116 as a lead development candidate from our Aurora kinase inhibitor program. In this program, several compounds have demonstrated efficacy by oral administration in hematological and solid tumor models with a mechanism consistent with inhibition of the target. We submitted in December 2006 an Investigational New Drug, or IND application, with the Food and Drug Administration, or FDA, to begin clinical trials of CYC116, an orally-active inhibitor of Aurora kinases A & B and VEGFR2, for the treatment of cancer. Aurora kinases are a family of serine/threonine protein kinases that are only expressed in actively dividing cells and are crucial for the process of cell division, or mitosis. These proteins, which have been found to be over-expressed in many types of cancer, have generated significant scientific and commercial interest as cancer drug targets. Aurora kinases were discovered by Professor David Glover, Chief Scientist of Cyclacel's Polgen Division. VEGFR2 is a receptor protein that is part of an important and validated pathway in angiogenesis, or blood vessel formation. We have retained worldwide rights to commercialize CYC116.

In our development programs, we have been an early adopter in the use of biomarker analysis to help evaluate whether our drug candidates are having their intended effect through their assumed mechanisms. Biomarkers are proteins or other substances whose presence in the blood can serve as an indicator or marker of diseases. Biomarker data from early clinical trials may also enable us to design subsequent trials more efficiently and to monitor patient compliance with trial protocols. We believe that in the longer term biomarkers may allow the selection of patients more likely to respond to its drugs for clinical trial and marketing purposes and increase the benefit to patients.

Our approach to drug discovery and development relies on proprietary genomic technology to identify gene targets, which are then progressed by means of structure-based design techniques through to the development stage. This approach is exemplified by our Aurora kinase and Plk, or Polo-like kinase, inhibitor programs. Fundamentally, this approach to drug discovery and design aims to improve our ability to select promising drug targets in the early stages of the process so as to decrease compound attrition rates during the later, more expensive stages of drug development. We devote more resources initially to enrich the target selection process, so that we focus our efforts on targets that have a higher probability of yielding successful drug candidates. To this end, we have assembled an integrated suite of sophisticated discovery and design technologies, together with highly skilled personnel.

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Our corporate headquarters are located at 200 Connell Drive, Suite 1500, Berkeley Heights, NJ 07922; telephone number (908) 517-7330, where our medical and regulatory functions are also located. Our primary research facility is located in Dundee, Scotland which is the center of our structure-based drug design and development programs. A second research facility is located in Cambridge, England and is home to our Polgen division, which is focused on discovering the function of new cancer genes and validating their use as potential druggable targets.

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RISK FACTORS

The following factors should be considered carefully in evaluating whether to purchase shares of Cyclacel common stock. These factors should be considered in conjunction with any other information included or incorporated by reference herein, including in conjunction with forward-looking statements made herein. See ‘‘Where You Can Find More Information’’ on page 47.

RISKS RELATED TO OUR BUSINESS

We are at an early stage of development as a company and we do not have, and may never have, any products that generate revenues.

We are at an early stage of development as a company and have a limited operating history on which to evaluate our business and prospects. Since beginning operations in 1997, we have not generated any product revenues. We currently have no products for sale and we cannot guarantee that we will ever have any marketable products. We must demonstrate that our drug candidates satisfy rigorous standards of safety and efficacy for their intended uses before the Food and Drug Administration, or FDA, and other regulatory authorities in the United States, the European Union and elsewhere. Significant additional research, preclinical testing and clinical testing is required before we can file applications with the FDA or other regulatory authorities for premarket approval of our drug candidates. In addition, to compete effectively, our drugs must be easy to administer, cost-effective and economical to manufacture on a commercial scale. We may not achieve any of these objectives. Seliciclib and sapacitabine, our most advanced drug candidates for the treatment of cancer, are currently our only drug candidates in clinical trials and we cannot be certain that the clinical development of these or any other drug candidates in preclinical testing or clinical development will be successful, that they will receive the regulatory approvals required to commercialize them or that any of our other research and drug discovery programs will yield a drug candidate suitable for investigation through clinical trials. Our commercial revenues, if any, will be derived from sales of drugs that we do not expect to become marketable for several years, if at all.

We have a history of operating losses and we may never become profitable. Our stock is a highly speculative investment.

We have incurred operating losses in each year since beginning operations in 1997 due to costs incurred in connection with our research and development activities and general and administrative costs associated with our operations, and we may never achieve profitability. As of September 30, 2006, our accumulated deficit was $132.7 million. Our net loss from inception through September 30, 2006 was $170.9 million. Our initial drug candidates are in the early stages of clinical testing and we must conduct significant additional clinical trials before we can seek the regulatory approvals necessary to begin commercial sales of our drugs. We expect to incur continued losses for several years, as we continue our research and development of our initial drug candidates, seek regulatory approvals and commercialize any approved drugs. If our initial drug candidates are unsuccessful in clinical trials or we are unable to obtain regulatory approvals, or if our drugs are unsuccessful in the market, we will not be profitable. If we fail to become and remain profitable, or if we are unable to fund our continuing losses, you could lose all or part of your investment.

We will need to raise substantial additional capital to fund our operations and if we fail to obtain additional funding, we may be unable to complete the development and commercialization of our drug candidates or continue our research and development programs.

We have funded all of our operations and capital expenditures with proceeds from the issuance of public equity securities, private placements of our securities, interest on investments, government grants and research and development tax credits. In order to conduct the lengthy and expensive research, preclinical testing and clinical trials necessary to complete the development and marketing of our drug candidates, we will require substantial additional funds. Based on our current operating plans, we expect our existing resources to be sufficient to fund our planned operations for at least the next 12 months. To meet these financing requirements, we may raise funds through public or private

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equity offerings, debt financings or strategic alliances. Raising additional funds by issuing equity or convertible debt securities will cause our shareholders to experience substantial dilution in their ownership interests and new investors may have rights superior to the rights of our other stockholders. Raising additional funds through debt financing, if available, may involve covenants that restrict our business activities and options. To the extent that we raise additional funds through collaborations and licensing arrangements, we may have to relinquish valuable rights to our drug discovery and other technologies, research programs or drug candidates, or grant licenses on terms that may not be favorable to us. Additional funding may not be available to us on favorable terms, or at all. If we are unable to obtain additional funds, we may be forced to delay or terminate our clinical trials and the development and marketing of our drug candidates.

Clinical trials are expensive, time consuming and subject to delay.

Clinical trials are expensive and complex, can take many years and have uncertain outcomes. We estimate that clinical trials of our most advanced drug candidates will continue for several years, but may take significantly longer to complete. The designs used in some of our trials have not been used widely by other pharmaceutical companies. Failure can occur at any stage of the testing and we may experience numerous unforeseen events during, or as a result of, the clinical trial process that could delay or prevent commercialization of our current or future drug candidates, including but not limited to:

- delays in securing clinical investigators or trial sites for our clinical trials;
- delays in obtaining institutional review board, or IRB, and other regulatory approvals to commence a clinical trial;
- slower than anticipated rates of patient recruitment and enrollment, or reaching the targeted number of patients;
- negative or inconclusive results from clinical trials;
- unforeseen safety issues;
- uncertain dosing issues;
- introduction of new therapies or changes in standards of practice or regulatory guidance that render our clinical trial endpoints or the targeting of our proposed indications obsolete;
- inability to monitor patients adequately during or after treatment or problems with investigator or patient compliance with the trial protocols;
- inability to replicate in large controlled studies safety and efficacy data obtained from a limited number of patients in uncontrolled trials; and
- inability or unwillingness of medical investigators to follow our clinical protocols.

If we suffer any significant delays, setbacks or negative results in, or termination of, our clinical trials, we may be unable to continue development of our drug candidates or generate revenue and our development costs could increase significantly.

Adverse events have been observed in our clinical trials and may force us to stop development of our product candidates or prevent regulatory approval of our product candidates.

Adverse or inconclusive results from our clinical trials may substantially delay, or halt entirely, any further development of our drug candidates. Many companies have failed to demonstrate the safety or effectiveness of drug

candidates in later stage clinical trials notwithstanding favorable results in early stage clinical trials. Previously unforeseen and unacceptable side effects could interrupt, delay or halt clinical trials of our drug candidates and could result in the FDA or other regulatory authorities denying approval of our drug candidates. We will need to demonstrate safety and efficacy for specific indications of use, and monitor safety and compliance with clinical trial protocols throughout the development process. To date, long-term safety and efficacy has not been

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demonstrated in clinical trials for any of our drug candidates. Toxicity and ‘‘severe adverse effects’’ as defined in trial protocols have been noted in preclinical and clinical trials involving certain of our drug candidates. For example, elevations of liver enzymes and decrease in potassium levels have been observed in some patients receiving our lead drug candidate, seliciclib and neutropenia was observed in patients receiving sapacitabine. In addition, we may pursue clinical trials for seliciclib in more than one indication. There is a risk that severe toxicity observed in a trial for one indication could result in the delay or suspension of all trials involving the same drug candidate. We are currently conducting Phase IIb clinical trials to test the safety and efficacy of seliciclib in the treatment of non small cell lung cancer. Independent investigators are conducting Phase I clinical trials to test the safety of sapacitabine in patients with advanced cancers. If these trials or any future trials are unsuccessful, our business and reputation could be harmed and our share price could be negatively affected.

Even if we believe the data collected from clinical trials of our drug candidates are promising with respect to safety and efficacy, such data may not be deemed sufficient by regulatory authorities to warrant product approval. Clinical data can be interpreted in different ways. Regulatory officials could interpret such data in different ways than we do which could delay, limit or prevent regulatory approval. The FDA, other regulatory authorities or we may suspend or terminate clinical trials at any time. Any failure or significant delay in completing clinical trials for our drug candidates, or in receiving regulatory approval for the commercialization of our drug candidates, may severely harm our business and reputation.

If our understanding of the role played by CDKs or Aurora kinases in regulating the cell cycle is incorrect, this may hinder pursuit of our clinical and regulatory strategy.

We have programs to develop small molecule inhibitors of cyclin dependent kinases (CDK) and Aurora kinases. Our lead drug candidate, seliciclib, is a CDK inhibitor, and CYC116 is an Aurora kinase inhibitor, based on our understanding of CDK and Aurora kinase inhibitors. Although a number of pharmaceutical and biotechnology companies are attempting to develop CDK or Aurora inhibitor drugs for the treatment of cancer, no CDK or Aurora kinase inhibitor has yet reached the market. Our seliciclib program relies on our understanding of the interaction of CDKs with other cellular mechanisms that regulate key stages of cell growth. If our understanding of the role played by CDKs or Aurora kinase inhibitors in regulating the cell cycle is incorrect, our lead drug and CYC116 may fail to produce therapeutically relevant results, hindering our ability to pursue our clinical and regulatory strategy.

If we fail to enter into and maintain successful strategic alliances for our drug candidates, we may have to reduce or delay our drug candidate development