Cyclacel Pharmaceuticals, Inc.
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Registration No. 333-187801

PROSPECTUS SUPPLEMENT

(To Prospectus Dated April 22, 2013)

Up to $8,350,000

Common Stock

We have entered into a Controlled Equity Offering[SM] sales agreement, dated July 10, 2015, with Cantor Fitzgerald & Co., relating to shares of our common stock offered by this prospectus supplement and the accompanying prospectus. In accordance with the terms of the sales agreement, we may offer and sell shares of our common stock having an aggregate offering price of up to $8,350,000 from time to time on or after the date hereof, pursuant to this prospectus supplement through Cantor Fitzgerald & Co., acting as agent.

Our common stock is listed on The NASDAQ Global Market under the symbol “CCYC.” On July 8, 2015, the last reported sale price of our common stock on The NASDAQ Global Market was $0.72 per share.

Sales of our common stock, if any, under this prospectus supplement and the accompanying prospectus may be made in sales deemed to be “at-the-market” equity offerings as defined in Rule 415 promulgated under the Securities Act ofs 1933, as amended, or the Securities Act, including sales made directly on or through the NASDAQ Global Market, the existing trading market for our common stock, sales made to or through a market maker other than on an exchange or otherwise, in negotiated transactions at market prices prevailing at the time of sale or at prices related to such prevailing market prices, and/or any other method permitted by law, including in privately negotiated transactions. Cantor Fitzgerald & Co. will act as sales agent on a best efforts basis and use commercially reasonable efforts to sell on our behalf all of the shares of common stock requested to be sold by us, consistent with its normal trading and sales practices, on mutually agreed terms between Cantor Fitzgerald & Co. and us. There is no arrangement for funds to be received in any escrow, trust or similar arrangement.

Cantor Fitzgerald & Co. will be entitled to compensation at a fixed commission rate of 3.0% of the gross sales price per share sold. In connection with the sale of our common stock on our behalf, Cantor Fitzgerald & Co. will be deemed to be an "underwriter" within the meaning of the Securities Act and the compensation of Cantor Fitzgerald & Co. will be deemed to be underwriting commissions or discounts.

The aggregate market value of our outstanding shares of common stock held by non-affiliates was $25,830,731.70 based on 34,702,909 shares of common stock outstanding, as of the date of this prospectus, of which 28,700,813 shares were held by non-affiliates, and a per share price of $0.90 based on the closing sale price of our common stock on the NASDAQ Global Market on June 23, 2015. Under the registration statement to which this prospectus supplement forms a part, we may not sell our securities in a primary offering with a value exceeding one-third of our public float in any 12-month period (unless our public float rises to $75.0 million or more). During the prior 12 month calendar period that ends on, and includes, this prospectus supplement, we have sold securities having an aggregate market value of approximately $220,100 pursuant to General Instruction I.B.6 of Form S-3. Accordingly, we may sell up to $8,350,000 in shares of common stock hereunder.

Investing in our securities involves a high degree of risk. Before making an investment decision, please read "Risk Factors" beginning on page S-13 of this prospectus supplement, page 13 of the accompanying prospectus and in the documents incorporated by reference into this prospectus supplement and the accompanying prospectus.

Neither the Securities and Exchange Commission nor any state securities commission has approved or disapproved of these securities or determined if this prospectus supplement or the accompanying prospectus is truthful or complete. Any representation to the contrary is a criminal offense.

The date of this prospectus supplement is July 10, 2015

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ABOUT THIS PROSPECTUS SUPPLEMENT

This prospectus supplement and the accompanying prospectus are part of a "shelf" registration statement on Form S-3 (File No. 333-187801) that we filed with the Securities and Exchange Commission (SEC) on April 8, 2013 and that, as amended, was declared effective on April 22, 2013.

This document is in two parts. The first part is this prospectus supplement, which describes the terms of this offering and also adds to and updates information contained in the accompanying prospectus and the documents incorporated by reference into this prospectus supplement and the accompanying prospectus. The second part is the accompanying prospectus, which gives more general information about the shares of common stock and other securities we may offer from time to time under our shelf registration statement, some of which does not apply to the securities offered by this prospectus supplement. To the extent there is a conflict between the information contained in this prospectus supplement, on the one hand, and the information contained in the accompanying prospectus or any document incorporated by reference herein or therein, on the other hand, you should rely on the information in this prospectus supplement.

You should read this prospectus supplement, the accompanying prospectus, the documents incorporated by reference in this prospectus supplement and the accompanying prospectus and any free writing prospectus that we have authorized for use in connection with this offering before making an investment decision. You should also read and consider the information in the documents referred to in the sections of this prospectus supplement entitled "Where You Can Find More Information" and "Incorporation of Documents by Reference."

You should rely only on the information contained or incorporated by reference in this prospectus supplement, the accompanying prospectus and any free writing prospectus that we have authorized for use in connection with this offering. We have not authorized anyone to provide you with different information. If anyone provides you with different or inconsistent information, you should not rely on it.

We are not making an offer to sell the securities covered by this prospectus supplement in any jurisdiction where the offer or sale is not permitted.

The information appearing in this prospectus supplement, the accompanying prospectus, the documents incorporated by reference in this prospectus supplement and the accompanying prospectus and any free writing prospectus that we have authorized for use in connection with this offering is accurate only as of its respective date, regardless of the time of delivery of the respective document or of any sale of securities covered by this prospectus supplement. You should not assume that the information contained in or incorporated by reference in this prospectus supplement or the accompanying prospectus, or in any free writing prospectus that we have authorized for use in connection with this

offering, is accurate as of any date other than the respective dates thereof.

In this prospectus supplement, “we,” “us,” “our,” “the company” and “Cyclacel” refer to Cyclacel Pharmaceuticals, Inc., unless the context otherwise requires.

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PROSPECTUS SUPPLEMENT SUMMARY

The items in the following summary are described in more detail later in this prospectus supplement and in the accompanying prospectus. This summary provides an overview of selected information and does not contain all the information you should consider before investing in our common stock. Therefore, you should read the entire prospectus supplement, the accompanying prospectus and any free writing prospectus that we have authorized for use in connection with this offering carefully, including the "Risk Factors" section and other documents or information included or incorporated by reference in this prospectus supplement and the accompanying prospectus before making any investment decision.

Recent Developments

Deficiency and Compliance Notices from The NASDAQ Stock Market

On February 2, 2015, we received a written notification from The NASDAQ Stock Market LLC indicating that we were not in compliance with NASDAQ Listing Rule 5450(a)(1) because the minimum bid price of our shares of common stock was below $1.00 per share for the previous 30 consecutive business days. Pursuant to the NASDAQ Listing Rule 5810(c)(3)(A), we have been granted a 180-calendar day compliance period, or until August 3, 2015, to regain compliance with the minimum bid price requirement. During the compliance period, our shares of common stock will continue to be listed and traded on The NASDAQ Global Market. To regain compliance, the closing bid price of our shares of common stock must meet or exceed $1.00 per share for at least ten consecutive business days during this 180-day grace period. If we are not in compliance by August 3, 2015, we may be afforded a second 180-calendar day grace period if we transfer the listing of our shares of common stock to The NASDAQ Capital Market. To qualify, we would be required to meet the continued listing requirement for market value of publicly held shares and all other initial listing standards for The NASDAQ Capital Market, except for the minimum bid price. In addition, we would be required to notify NASDAQ of our intent to cure the minimum bid price deficiency by effecting a reverse stock split, if necessary.

If we do not regain compliance within the allotted compliance period(s), including any extensions that may be granted by NASDAQ, NASDAQ will provide notice that our shares of common stock will be subject to delisting. We would then be entitled to appeal NASDAQ's determination to a NASDAQ Hearings Panel and request a hearing.

We intend to consider available options to resolve the noncompliance with the minimum bid price requirement. No determination regarding our response has been made at this time. There can be no assurance that we will be able to regain compliance with the minimum bid price requirement or will otherwise be in compliance with other NASDAQ listing criteria.

Equity Transactions

On March 9, 2015, we completed a public offering of 10,000,000 shares of our common stock, at a price to the public of $1.00 per share, for proceeds, net of certain fees and expenses, of approximately $9.2 million.

On July 8, 2015, we sold 314,424 shares of our common stock under our purchase agreement with Aspire Capital Fund, LLC, or Aspire, for proceeds of approximately $220,100. All of the available shares under the purchase agreement have now been sold and the purchase agreement has terminated according to its terms.

Preferred Stock Dividend

On May 22, 2015, the Board of the Company declared a quarterly cash dividend in the amount of $0.15 per share on the Company's 6% Convertible Exchangeable Preferred Stock ("Preferred Stock"). The cash dividend will be payable on August 1, 2015 to the holders of record of the Preferred Stock as of the close of business on July 17, 2015.

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Licensing & Supply Agreement Regarding Development of Seliciclib in Cystic Fibrosis

On June 29, 2015, we announced the execution of a collaboration, licensing and supply agreement with ManRos Therapeutics SA, or ManRos, for the exclusive development and commercialization of our oral seliciclib capsules by ManRos as a treatment for cystic fibrosis, or CF. Among other terms of the agreement, ManRos licensed rights to our proprietary clinical data to enable clinical development of seliciclib for CF indications. The agreement provides for our supply of seliciclib investigational product for initial and later stage clinical trials of seliciclib in CF and technical assistance related to our know-how to facilitate these trials. We will receive an up-front payment, milestone payments and tiered royalties, if seliciclib is commercialized for the treatment of CF.

Our Business

General

We are a pioneer company in the field of cell cycle biology with a vision to improve patient healthcare with orally available innovative medicines. Our goal is to develop and commercialize small molecule drugs that target the various phases of cell cycle control for the treatment of cancer and other serious diseases, particularly those of high unmet medical need.

Our strategy is to build a diversified biopharmaceutical business focused in hematology and oncology based on a development pipeline of novel drug candidates. Substantially all efforts of the Company to date have been devoted to performing research and development, conducting clinical trials, developing and acquiring intellectual property, raising capital and recruiting and training personnel.

Drug Candidates

The cell cycle, the biological process by which cells progress and divide, lies at the heart of cancer. In normal cells, the cell cycle is controlled by a complex series of signaling pathways by which a cell grows, replicates its DNA and divides. This process also includes mechanisms to ensure errors are corrected, and if not, the cells commit suicide or apoptosis. In cancer, as a result of genetic mutations, this regulatory process malfunctions, resulting in uncontrolled cell proliferation.

We have generated several families of anticancer drugs that act on the cell cycle including sapacitabine, seliciclib and CYC065. We believe that these drug candidates are differentiated in that they are orally-available and interact with unique target profiles and mechanisms and have the potential to treat multiple cancer indications.

Our lead candidate, sapacitabine, is a novel, orally-available nucleoside analog. A number of nucleoside drugs, such as gemcitabine and cytarabine, also known as Ara-C, both generic drugs, 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 and fluorouracil, or 5-FU, two widely used nucleoside analogs, in delaying the onset and growth of liver metastasis. We hold the worldwide rights to commercialize sapacitabine, except for Japan, for which Daiichi Sankyo Co., Ltd., or Daiichi Sankyo, has a right of first negotiation.

The U.S. Food and Drug Administration, or FDA, and the European Medicines Agency, or EMA, have designated sapacitabine as an orphan drug for the treatment of both Acute Myeloid Leukemia, or AML, and Myelodysplastic Syndromes, or MDS.

We are currently evaluating sapacitabine in a Phase 3 study being conducted under a Special Protocol Assessment, or SPA, with the FDA for the front-line treatment of AML in the elderly. We are also exploring sapacitabine in Phase 2 studies for MDS, non-small cell lung cancer, or NSCLC, and chronic lymphocytic leukemia, or CLL and in a Phase 1 study in solid tumors in combination with seliciclib, another of our drug candidates. Sapacitabine has been evaluated in approximately 1,000 patients to date.

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In our second development program we are evaluating cyclin dependent kinase, or CDK, inhibitors. CDKs are involved in cancer cell growth, metastatic spread and DNA damage repair. Seliciclib, our lead CDK inhibitor, selectively inhibits a spectrum of enzyme targets — CDK2, CDK5, CDK7 and CDK9 — that are central to the process of c division and cell cycle control. In breast and lung tumors overexpression of cyclin E is associated with poor prognosis and drug resistance. Resistant breast and lung tumor cell lines overexpressing cyclin E are resensitized to apoptotic cell killing by seliciclib. NSCLC cell lines with Ras-activating mutations, such as KRAS and NRAS, have been found to be sensitive to seliciclib-induced apoptosis. Seliciclib will also be evaluated in investigator-sponsored trials, or ISTs, including a study to treat rheumatoid arthritis, or RA, supported by an approximately $1.5 million grant from the UK's Medical Research Council. Enabled by the clinical development experience in solid tumors, investigators believe that seliciclib's mechanism of action and oral administration route may be of benefit in treating patients with RA. To date, seliciclib has been evaluated in approximately 450 patients in several Phase 1 and 2 studies and has shown signs of anti-cancer activity. We have retained worldwide rights to commercialize seliciclib. Seliciclib has completed a Phase 2B randomized study in third-line NSCLC and is currently undergoing a study in solid tumors in combination with our own drug candidate, sapacitabine.

Our second generation CDK inhibitor, CYC065, is a highly selective inhibitor of CDKs targeting CDK2 and CDK9 enzymes. CYC065 has increased anti-proliferative potency and improved pharmaceutical properties compared to seliciclib. Independent investigators have reported that CYC065 reverses resistance in breast cancer cells that have become resistant to trastuzumab. CYC065 has also shown activity against leukemia cells, including those with mixed lineage leukemia rearrangements, or MLLr, and was also shown to be effective against uterine cancer cells including those resistant to chemotherapy. Investigational new drug, or IND, — enabling studies with CYC065 were completed, supported by a $1.9 million grant from the Biomedical Catalyst, a United Kingdom government program, and we have received FDA clearance for the first-in-human Phase 1 study of CYC065.

In addition to these development programs, in our polo-like kinase, or PLK, inhibitor program, we have discovered CYC140 and other potent and selective small molecule inhibitors of PLK1, a kinase active during cell division, targeting the mitotic phase of the cell cycle. PLK was discovered by Professor David Glover, our Chief Scientist. We are progressing the IND-directed preclinical development of CYC140, supported by an approximately $3.5 million grant from the Biomedical Catalyst of the United Kingdom.

We currently retain virtually all marketing rights worldwide to the compounds associated with our drug programs. To optimize our commercial return, we intend to enter into selected partnering arrangements.

Lead Development Programs

Our pipeline and expertise in cell cycle biology

Our core area of expertise is in cell cycle biology and we focus primarily on the 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 the quality of life and improving survival rates of cancer patients.

We have retained rights to commercialize our clinical development candidates and our business strategy is to enter into selective partnership arrangements with these programs.

Oncology Development Programs

We have generated several families of anticancer drugs that act on the cell cycle, including nucleoside analogs, CDK inhibitors, PLK inhibitors and Aurora Kinase/vascular endothelial growth factor, or AK/VEGFR2, inhibitors. In our development programs, we have been an early adopter of biomarker analysis to help evaluate whether our drug candidates are having their intended effect through their assumed mechanisms at different doses and schedules. 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. For example, we reported that sapacitabine efficacy is enhanced in tumor cells that are defective in homologous recombination DNA repair. In another example, we reported that sensitivity to our PLK1 inhibitor CYC140 correlated with the status of tumor suppressor protein p53, in a panel of esophageal cancer cell lines, which could be used as a predictive biomarker in clinical trials to identify

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responders. We believe that in the longer term biomarkers may allow the selection of patients more likely to respond to our drugs in clinical trials and increase the benefit to patients.

Although a number of pharmaceutical and biotechnology companies are currently attempting to develop nucleoside analogs, CDK inhibitors, PLK inhibitors and AK and/or VEGFR inhibitor drugs, we believe that our drug candidates, are differentiated in that they are orally-available and demonstrate unique target profiles and mechanisms. For example, we believe that our sapacitabine is the only orally-available nucleoside analog presently being tested in Phase 3 trials in previously untreated AML and in Phase 2 for high risk MDS.

Market opportunity in hematology

Cancer remains a major life-threatening disease in the United States with approximately 600,000 Americans expected to die of cancer and approximately 1.7 million new cases of cancer diagnosed every year.

AML is a cancer of the blood cells that progresses rapidly and if not treated, could be fatal in a few months. AML is generally a disease of older people and is uncommon before the age of 40. The average age of a patient with AML is about 67 years. According to the American Cancer Society approximately 52,000 cases of leukemia are diagnosed annually in the United States of which about 19,000 are classified as AML of which about half are elderly aged 70 years or older. Nearly 9,000 deaths are caused by this cancer each year in the United States. A review of The University of Texas MD Anderson Cancer Center's historical experience with front-line intensive induction chemotherapy for AML patients aged 70 years or older demonstrated that while 45% achieved a complete remission, median overall survival was only 4.6 months and was associated with a 4-week death rate of 26% and an 8-week death rate of 36%.

MDS is a family of clonal myeloid neoplasms, or malignancies of the blood, caused by the failure of blood cells in the bone marrow to develop into mature cells. Patients with MDS typically suffer from bone marrow failure and cytopenias, or reduced counts of platelets, red and white blood cells. The exact incidence and prevalence of MDS are unknown because it can go undiagnosed and a national survey canvassing both hospitals and office practitioners has not been completed. Some estimates place MDS incidence at 15,000 to 20,000 new cases each year in the US alone with some authors estimating incidence as high as 46,000. Literature suggests that there is a rising incidence of MDS as the age of the population increases with the majority of patients aged above 60 years. Patients currently receive hypomethylating agents as first-line treatment. There is no approved therapy for second-line treatment.

Sapacitabine

Sapacitabine, previously known as CYC682, is an orally-available nucleoside analog. Both sapacitabine and CNDAC, its major metabolite, have demonstrated potent anti-tumor activity in preclinical studies. 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 novel mechanism whereby the compound interferes with DNA synthesis through the incorporation of CNDAC into DNA during replication or repair, triggering a beta-elimination reaction and leading to the formation of SSBs, which can activate the G2 checkpoint transcription coupled nucleotide excision repair, or TC-NER. During subsequent rounds of replication, SSBs are converted to double-strand breaks, or DSBs; these can be repaired by the homologous recombination repair, or HRR, pathway, or, if unrepaired, result in cell death.

We are currently exploring sapacitabine in both hematological cancers and solid tumors. Approximately 1,000 patients have received sapacitabine in Phase 1, 2 and 3 studies.

Hematological Cancers

SEAMLESS, randomized Phase 3, pivotal trial of sapacitabine in elderly patients with AML

The SEAMLESS study is being conducted under an SPA agreement that Cyclacel reached with the FDA.

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The study is chaired by Hagop M. Kantarjian, M.D., Chairman and Professor, Department of Leukemia, The University of Texas MD Anderson Cancer Center. SEAMLESS is a multicenter, randomized, Phase 3 study of sapacitabine as a front-line treatment in approximately 485 elderly patients aged 70 years or older with newly diagnosed AML who are not candidates for or have refused intensive induction chemotherapy. In SEAMLESS an investigational arm of oral sapacitabine administered in alternating cycles with intravenous decitabine is compared with a control arm of intravenous decitabine administered alone. The primary efficacy endpoint is overall survival. SEAMLESS completed enrollment in December 2014 with approximately 110 centers participating from the United States and Europe. Also in December 2014, the Data Safety Monitoring Board, or DSMB, conducted a planned interim analysis for futility after 247 events, or patient deaths, and the final safety review of 470 randomized patients. The DSMB found no safety concerns. However, the planned futility boundary has been crossed and the DSMB determined that, based on available interim data, it would be unlikely for the study to reach statistically significant improvement in survival. The DSMB saw no reasons why patients should discontinue treatment on their assigned arm and recommended that recruited patients stay on treatment.

The interim analysis for futility is primarily driven by the events within the first 6 months of patients entering into the trial. Of 247 events in SEAMLESS, 173 (70%) have occurred in the first 6 months. This means that the survival curves beyond 6 months are poorly estimated at this time. Furthermore, follow up of European patients is significantly shorter than that of U.S. patients as the study opened for European accrual in April 2014. It is important to have complete follow up of all patients to ensure that a potential treatment effect beyond 6 months is not missed.

We remain blinded and, in accordance with the DSMB's recommendations, will follow-up patients as per the study protocol until the prespecified 424 events have been observed. This is estimated to occur between the second half of 2015 and the first half of 2016. In parallel to the follow-up of enrolled patients, we also plan to submit a Pediatric Investigation Plan, or PIP, to the European Medicines Agency, or EMA. A marketing authorization application, or MAA, is valid only if it includes the results of all studies performed in accordance with an agreed upon PIP, or a decision of the EMA granting a deferral or waiver of those studies. Depending on the final data, we may meet with regulatory authorities in Europe and the U.S. to discuss registration submissions for sapacitabine for the AML indication.

Pilot/Lead-in study of sapacitabine in elderly patients with AML

Results from a single-arm, multicenter, Phase 1/2 clinical trial examining the safety and efficacy of oral sapacitabine administered sequentially with intravenous decitabine, the same regimen as in the investigational arm of SEAMLESS, were reported during a poster session at the 2012 American Society of Hematology, or ASH, Annual Meeting. Forty-six patients were treated with alternating cycles of sapacitabine and decitabine. Median age was 77 years (range 70-90). Thirty-three patients (72%) were 75 years or older. Median overall survival was 238 days, or approximately 8 months. The number of patients still alive at 3 months was 38 (83%), at 6 months 30 (65%), at 12 months 16 (35%) and at 18 months 12 (26%). Sixteen patients (35%) survived 1 year or longer. Among 33 patients who were 75 years or older, median overall survival was 263 days, or approximately 9 months, and one-year survival was 36%. Nineteen patients (41%) responded with 10 complete responses (CRs), 4 partial responses (PRs) and 5 major hematological

improvements (HIs). Median time to response was 2 cycles, i.e., one cycle of decitabine and one cycle of sapacitabine (range 1-10). Twenty-seven patients (59%) received 5 or more cycles of treatment. Two dose-limiting toxicities (DLT) were observed (lung infection/sepsis, typhlitis). Thirty-day mortality from all causes was 4%. Sixty-day mortality from all causes was 13% with one death from typhlitis considered to be possibly related to decitabine by investigator assessment.

Phase 2 randomized study of sapacitabine in patients with previously untreated or first relapse AML

SEAMLESS builds on promising one year survival observed in elderly patients with AML enrolled in a Phase 2 study of single agent sapacitabine. In December 2007, we initiated a multicenter, randomized Phase 2 clinical trial of oral sapacitabine in 60 elderly patients with AML aged 70 years or older who were previously untreated or in first relapse. The Phase 2 study, led by Dr. Kantarjian, had a primary endpoint of one year survival and randomized patients to one of three dosing schedules of sapacitabine. Secondary objectives were to assess complete remission, or CR, partial remission, or PR, duration of CR or CRp, or major hematological improvement and their corresponding durations, transfusion requirements, number of hospitalized days and safety. The study used a

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selection design with the objective of identifying a dosing schedule among three different arms, A. 200 mg twice daily for seven days every 3-4 weeks, B. 300 mg twice daily for seven days every 3-4 weeks, and C. 400 mg twice daily for three days per week for two weeks every 3-4 weeks, which would produce a better one year survival rate in the event that all three dosing schedules were active.

In November 2012, the results from the Phase 2 study were published in The Lancet Oncology, demonstrating the safety and efficacy of sapacitabine in this patient population. Between December 27, 2007 and April 21, 2009, a total of 105 patients were enrolled and treated in the Phase 2 study. Their median age was 77 years with a range of 70-91 years. The group was comprised of a randomized cohort of 60 patients and an expanded, non-randomly assigned cohort enrolling a further 45 patients. Of the 105 patients, 86 were previously untreated and 19 in first relapse. Approximately 50% of patients had AML de novo and 50% had AML preceded by antecedent hematological disorder, or AHD, such as MDS or myeloproliferative disease, or treatment-related AML. All but one enrolled patients had intermediate or unfavorable cytogenetics. The randomized cohort of patients was assigned to one of three dosing schedules: 200 mg twice a day for 7 days (Arm A); 300 mg twice a day for 7 days (Arm B); and 400 mg twice a day for 3 days each week for 2 weeks (Arm C). All schedules were given in 28 day cycles. The 3-day dosing schedule in Arm C was selected for further clinical development in elderly patients with untreated AML. This decision was based on the schedule's overall efficacy profile, which included a one-year survival rate of 30%, median overall survival of 213 days and durable complete remissions, or CRs, in 25% of patients. The median overall survival of patients from all arms who achieved CR was 525 days (95% C.I. 192-798). The most common grade 3-4 adverse events regardless of causality were anemia, neutropenia, thrombocytopenia, febrile neutropenia and pneumonia. Seven deaths were thought to be probably or possibly related to sapacitabine treatment.

Randomized Phase 2 clinical trial in older patients with MDS as a second-line treatment

In September 2008, we advanced sapacitabine into an open-label, multi-center, randomized Phase 2 trial as a second-line treatment in patients aged 60 or older with intermediate-2 or high-risk MDS after treatment failure of front-line hypomethylating agents, such as azacitidine and/or decitabine. The Phase 2 study randomized 63 patients aged 60 years or older with MDS of intermediate-2 (n=52) or high-risk (n=11) classification by the International Prognostic Scoring System, or IPSS, at study entry to receive sapacitabine every 4 weeks on one of 3 dosing schedules: 200 mg twice daily for 7 days (Arm G), 300 mg once daily for 7 days (Arm H), or 100 mg once daily for 5 days per week for 2 weeks (Arm I). The primary efficacy endpoint of the study is one-year survival with the objective of identifying a dosing schedule that produces a better one-year survival rate in the event that all three dosing schedules are active. All patients in the study progressed after receiving azacitidine, decitabine, or both agents. Secondary objectives are to assess the number of patients who have achieved CR or CRp, PR, hematological improvement and their corresponding durations, transfusion requirements, number of hospitalization days and safety.

In December 2013 at the 2013 American Society of Hematology, or ASH, Meeting and Exposition, we announced primary endpoint data from the ongoing, open-label, multicenter, randomized Phase 2 trial of oral sapacitabine capsules in older patients with myelodysplastic syndromes after treatment failure of front-line hypomethylating agents, such as azacitidine and/or decitabine. The median overall survival for each arm was approximately 9.7 months

for Arm G, 9.7 months for Arm H, and 7.6 months for Arm I. The median overall survival for all three arms was approximately 8.6 months. One-year survival was 38% for Arm G, 24% for Arm H, and 33% for Arm I. Nine patients had responded (2 CRs, 2 CRp, and 5 major HIs): 19% for Arm G, 10% for Arm H and 14% for Arm I and the time to response was one to four cycles. Median number of cycles was three with a range of one to over 23 and 30 patients received four or more cycles.

Additionally, 23 patients achieved stable disease lasting longer than 16 weeks. The 30 day mortality from all causes was 5% in each of the three arms and ten patients, or approximately 16%, were still alive.

Median survival after treatment failure of front-line hypomethylating agents, such as azacitidine and/or decitabine, for patients with intermediate-2 or high- risk disease per IPSS, is reported in the literature to range between 5.6 and 4.3 months. Patients with high-risk IPSS scores also have a high probability of experiencing transformation of their MDS into AML, an aggressive form of blood cancer with typically poor survival.

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We have recently completed enrollment of a patient cohort in an additional part of the ongoing MDS Phase 2 study in order to evaluate better dosing regimens. We will follow-up with these additional Phase 2 patients until mature survival data become available. In parallel, we have conducted a feasibility analysis in preparation for a Phase 2b randomized controlled trial, or RCT, of sapacitabine in this patient population, which indicated that our proposed study design is feasible.

Solid Tumors

Phase 2 clinical trial in patients with NSCLC

We are also evaluating sapacitabine in a Phase 2, single arm, multicenter, clinical trial in patients with NSCLC who have had at least one prior chemotherapy. This study builds on the observation of prolonged stable disease of four months or longer experienced by heavily pretreated NSCLC patients enrolled in two Phase 1 studies of sapacitabine. The multicenter Phase 2 trial is led by Philip D. Bonomi, M.D., at Rush University Medical Center, Chicago. The primary objective of the study is to evaluate the rate of response and stable disease in patients with previously treated NSCLC. Secondary objectives are to assess progression-free survival, duration of response, duration of stable disease, one year survival, overall survival and safety.

Sixty-two patients have been treated with two dosing schedules, either twice daily or once a day. In the twice daily schedule 15 patients were treated with escalating doses. The recommended Phase 2 dose was reached at 75 mg twice daily for 5 days per week for 2 weeks every 3 weeks. Among 12 patients treated at this recommended Phase 2 dose, 4 achieved stable disease. All 4 responders had at least 2 prior therapies and have been discontinued from the study. Responders received an average of 7 treatment cycles.

In the once daily schedule 45 patients were treated with escalating doses. The recommended Phase 2 dose was reached at 250 mg once daily dosing level for 5 days per week for 2 weeks every 3 weeks. Among 25 patients treated with daily doses ranging from 100 mg to 175 mg, two patients achieved PR and 10 stable disease. The two PR responders had 3 or 4 prior therapies, respectively, and one remains on study. Among the 10 stable disease responders, 9 had at least 2 prior therapies and 2 remain on study. Responders received an average of 10 treatment cycles. The study is closed to accrual.

Phase 1 clinical trial of sapacitabine and seliciclib in patients with advanced cancers

In an ongoing Phase 1, single-arm, dose escalation study, sapacitabine and seliciclib are administered sequentially in patients with incurable advanced solid tumors unresponsive to conventional treatment or for which no effective therapy exists. Sapacitabine is dosed twice daily for 7 days (Day 1-7) and seliciclib twice daily for 3 days (Day 8-11) for three week cycles. At least 3 patients were enrolled at each escalating dose level. The first tumor imaging study is conducted after 2 cycles of treatment and every 3 cycles thereafter. The primary objective of the study is to determine the maximum tolerated dose, or MTD, and recommended Phase 2 dosing schedule of sapacitabine and seliciclib administered sequentially. The secondary objective is to evaluate the antitumor activity of sequential treatment and to explore the pharmacodynamic effect of this treatment in skin and peripheral blood mononuclear cells. At the 2013 American Society of Cancer Research Annual Meeting we reported that of 38 patients with incurable solid tumors and adequate organ function enrolled in the Phase 1 study, 16 were found to be BRCA mutation carriers. Four patients with BRCA-deficient pancreatic, breast or ovarian cancers had confirmed partial responses to the drug regimen. Based on available follow-up to date, three patients are experiencing durable partial responses, with the longest lasting more than 78 weeks. Researchers observed stable disease of 12 weeks or more in eight additional patients, including two patients with ovarian and breast cancers who carried BRCA mutations and whose stable disease lasted 64 and 21 weeks, respectively. Sapacitabine was administered twice daily for seven days followed by seliciclib twice daily for three days. The maximum tolerated doses were 50 mg sapacitabine twice daily and 1,200 mg seliciclib twice daily. Dose-limiting toxicities included reversible transaminase elevations and neutropenia. Adverse events were mild to moderate in intensity. Results of skin biopsies after treatment showed a 2.3-fold increase in DNA damage induced by sapacitabine, as measured by gamma-H2AX immunohistochemistry. Additional DNA damage occurred after treatment with seliciclib with a 0.58-fold further increase in gamma-H2AX staining.

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BRCA1 and BRCA2, or breast cancer susceptibility genes, are tumor suppressor genes that help ensure the stability of DNA, the cell's genetic material, and help prevent uncontrolled cell growth. Genetic testing for BRCA-status is routinely available. BRCA mutation has been linked to predisposition to breast and ovarian cancer. According to the US National Cancer Institute, during her life time a woman has a 60% chance of developing breast cancer and 15-40% chance of developing ovarian cancer if she inherits a harmful BRCA mutation. These risks are 5 times and over 10 times more likely than for women without the mutation, respectively.

Orphan Designation

European Union

During May 2008, we received designation from the EMA for sapacitabine as an orphan medicine in two separate indications: AML and MDS. The EMA's Committee for Orphan Medicinal Products, or COMP, adopted a positive opinion on our application to designate sapacitabine as an orphan medicinal product for the indications of AML and MDS. The objective of European orphan medicines legislation is to stimulate research and development of medicinal products for rare diseases by providing incentives to industry. An orphan designation in the European Union confers a range of benefits to sponsor companies including market exclusivity for a period of 10 years, EMA scientific advice on protocol development, direct access to the centralized procedure for review of marketing authorizations, EMA fee reductions and eligibility for grant support from European agencies.

United States

In June 2010, we announced that the FDA granted orphan drug designation to our sapacitabine product candidate for the treatment of both AML and MDS. An orphan designation in the United States confers a range of benefits to sponsor companies, including market exclusivity for a period of seven years from the date of drug approval, the opportunity to apply for grant funding from the United States government to defray costs of clinical trial expenses, tax credits for clinical research expenses and a potential waiver of the FDA's application user fee. Orphan status is granted by the FDA to promote the development of new drug therapies for the treatment of diseases that affect fewer than 200,000 individuals in the United States.

Seliciclib

Although our current clinical development priorities are focused on sapacitabine only, our second drug candidate, seliciclib, is a novel, orally-available, CDK inhibitor. The compound selectively inhibits a spectrum of enzyme

targets, CDK2, -7 and -9 that are central to the process of cell division and cell cycle control. The target profile of seliciclib is differentiated from the published target profile of other CDK inhibitors. Its selectivity is differentiated by recent publications by independent investigators which showed that seliciclib (i) is more active against NSCLC cells with K-Ras or N-Ras mutations than those with wild type Ras and (ii) overcomes resistance to letrozole in breast cancer cells caused by a particular form of cyclin E in complex with CDK2. 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 450 patients in several Phase 1 and 2 studies and has shown signs of anti-cancer activity.

Phase 2 clinical trial in patients with NSCLC

Four Phase 2 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 NSCLC and also breast cancer. Interim data from two Phase 2 open-label studies of a total of 52 patients with NSCLC, suggests that seliciclib treatment neither aggravated the known toxicities of standard first and second-line chemotherapies nor appeared to cause unexpected toxicities, although these trials were not designed to provide statistically significant comparison.

In December 2010, we announced topline results from APPRAISE, our Phase 2b, randomized discontinuation, double-blinded, placebo-controlled, study of oral seliciclib capsules as a third line or later treatment in patients with NSCLC. APPRAISE was led by Chandra P. Belani, M.D. at Milton S. Hershey Medical Center, Penn State

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University. Topline results, after unblinding the treatment assignment among randomized patients, showed that there was no difference between the seliciclib and placebo arms in terms of progression free survival, or PFS (48 versus 53 days respectively). However, an increase in median overall survival, or OS, was observed favoring the seliciclib arm over the placebo arm (388 versus 218 days respectively). A total of 187 patients from 21 centers in the United States were entered in the study after having progressed on at least two prior therapeutic regimens for their NSCLC. Of these, 53 (28%) were randomized, 27 on seliciclib and 26 on placebo. Forty-five out of 53 randomized patients (85%) received 3 or more prior therapies and 45 out of 53 randomized patients (85%) previously received at least one EGFR inhibitor drug, with 22 on seliciclib and 23 on placebo. Fourteen patients were crossed-over to the seliciclib arm after their cancer progressed while they were receiving placebo. Study data demonstrated seliciclib to be safe at the administered dose.

Published preclinical work indicated that K-Ras mutational status, cyclin D1 and cyclin E1 protein levels correlated strongly with tumor sensitivity to seliciclib. In order to explore this possible molecular rationale for the difference in OS, we retrospectively collected and analyzed available biopsy samples from APPRAISE patients who granted informed consent. As only 30 patient samples were available from 152 consenting APPRAISE patients, results of the retrospective analysis were insufficient to allow meaningful correlation. A new prospectively designed study is required to test the hypothesis that these biomarkers can predict therapeutic effect of seliciclib in patients with advanced stage NSCLC.

Phase 2 clinical trials in patients with NPC

In November 2007, we commenced a Phase 2 multicenter, international, study of oral seliciclib as a single agent in patients with nasopharyngeal cancer, or NPC. The primary objective is to evaluate 6-month progression free survival, or PFS, of two dosing schedules of seliciclib in approximately 75 patients with previously treated NPC. Secondary objectives are OS, response rate, response duration, safety and tolerability. The first part of the study is designed to confirm safety and tolerability of 400 mg twice a day for four days per week or 800 mg once a day for four days per week of seliciclib. It is open to approximately 12 to 24 patients with advanced solid tumors as well as patients with NPC. The second part of the study, which is dependent on clinical data from the lead-in phase and available resources to fund the study, is designed to detect major differences between the two dosing schedules of seliciclib and a placebo group in terms of 6-month PFS in approximately 51 patients.

In May 2009, at the American Society of Clinical Oncology, or ASCO, annual meeting, we reported interim data from the lead-in portion of the Phase 2 study which demonstrated that oral seliciclib could be safely administered in two dosing schedules which were well tolerated and met the criteria for proceeding to the randomized stage of the study. Seliciclib treatment resulted in prolonged stable disease in 70% of previously-treated NPC patients, including 3 with stable disease lasting longer than 8 months, suggesting seliciclib inhibits tumor growth in NPC. The data support further clinical development of oral seliciclib in NPC.

CYC065

CYC065 is a highly-selective, orally-available, 2nd generation inhibitor of CDK2 and -9. These CDK enzymes play pivotal roles in cancer cell growth, metastatic spread and DNA damage repair. Pharmacological inhibition of CDK2 and CDK9 has been shown to have potent anticancer effects in certain tumor types resistant to established treatments. CYC065 causes apoptotic cell death of cancer cells at sub-micromolar concentrations. Antitumor efficacy has been achieved in vivo with once a day oral dosing at well tolerated doses. Translational biology supports the development of CYC065 as a stratified medicine for solid tumors as well as orphan diseases including adult and pediatric leukemias. Published preclinical studies show that CYC065 has the potential for development in AML, multiple myeloma, chronic lymphocytic leukemia and drug-resistant breast cancer.

CYC065 is mechanistically similar to Cyclacel's first generation CDK inhibitor, seliciclib, but with significantly improved potency in vitro and in vivo. CYC065 causes proportionally greater CDK9 inhibition, leading to improved efficacy in hematological malignancies and more prolonged down regulation of MCL-1, a biomarker of cell survival. CYC065 has improved metabolic stability and improved efficacy and dose potency compared with seliciclib. CYC065's physicochemical properties enable dosing by oral or intravenous routes. In April 2015, we presented CYC065 preclinical data at the American Association for Cancer Research (AACR) Annual Meeting 2015. Preclinical data demonstrated that CYC065 inhibits key cancer and leukemia survival mechanisms and causes

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death by apoptosis in cancer cells. We believe CYC065 is effective against AML, and in particular, AML with genetic abnormalities such as MLL rearrangements (MLL-r), which confer a poor prognosis. Potent anticancer activity of CYC065 was demonstrated in vivo in AML xenograft models resulting in over 90% inhibition of tumor growth.CYC065 was also shown to be effective against uterine cancer cells including those resistant to chemotherapy and was especially potent in uterine cancer cells in which cyclin E, the partner protein of CDK2, was amplified or overexpressed. In each case CYC065 showed synergy with available anticancer agents.

We received clearance by the FDA of an IND submission for CYC065 and plan to initiate Phase 1 clinical trials in patients with advanced solid tumors and lymphomas following institutional review board approval. The IND - directed preclinical development of CYC065 was supported by a grant award of approximately $1.9 million from the Biomedical Catalyst of the United Kingdom government.

PLK inhibitors

In our PLK inhibitor program we have discovered potent and selective small molecule inhibitors of PLK1, a kinase active during cell division, targeting the mitotic phase of the cell cycle. At the 2012 Annual Meeting of the American Association of Cancer Research, or AACR, we reported that one of these compounds, CYC140, was selected for further preclinical development. In a panel of esophageal cancer cell lines, sensitivity to CYC140 correlated with p53 status. Esophageal cell lines lacking functional p53 showed the greatest sensitivity to CYC140. Short drug exposure times demonstrated differential sensitivity between cancerous esophageal cells versus control, outlining the potential broad therapeutic index for CYC140 in treating esophageal cancers, and in particular those with non-functional p53. Status of p53 could be used as a predictive biomarker in clinical trials to identify responders. PLK was discovered by Professor David Glover, our Chief Scientist. We have received a grant award of approximately $3.7 million from the Biomedical Catalyst of the United Kingdom government to complete IND-directed preclinical development of CYC140.

Aurora kinase inhibitors

Aurora kinases, or AK, are a family of serine/threonine protein kinases discovered by Professor David Glover, our Chief Scientist, which 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. VEGFR2 is a receptor protein that plays a key regulatory role in the angiogenesis pathway, or blood vessel formation. VEGFR is targeted by recently approved drugs such as bevacizumab and sorafenib indicated for the treatment of several solid cancers, such as breast, colorectal, kidney, liver and lung.

At the Annual Meeting of the AACR 2012 we reported that collaborators tested the activity of CYC3, our novel Aurora Kinase A specific inhibitor, in pancreatic cancer cell lines. The collaborators reported that CYC3 suppresses pancreatic cancer cell growth, inducing mitotic arrest and apoptosis. CYC3 was also shown to act synergistically against pancreatic cancer cell lines in combination with paclitaxel at a 10-fold lower dose resulting in comparable anti-proliferative activity to standard paclitaxel dosing. As myelosuppression is associated with paclitaxel administration, the CYC3/low-dose paclitaxel combination was compared with high-dose paclitaxel in an in vitro granulocyte and macrophage assay in which the CYC3/low-dose paclitaxel combination displayed less myelotoxicity. The collaborators reported that the combination merits further investigation and has the potential for improved therapeutic index in vivo. In June 2007, we initiated and completed a multicenter Phase 1 pharmacologic clinical trial of CYC116, an orally-available inhibitor of Aurora kinase A and B and VEGFR2, in patients with advanced solid tumors. We have retained worldwide rights to commercialize CYC116 and our other Aurora kinase inhibitors.

Non-oncology Programs

Preclinical results from several independent investigators suggest that cell cycle inhibitors such as seliciclib and its backup molecules arrest the progress of the cell cycle and may have therapeutic benefit in the treatment of patients with autoimmune and inflammatory diseases as well as in diseases characterized by uncontrolled cell proliferation. Published data indicate potential benefit in glomerulonephritis, graft-versus-host disease, idiopathic pulmonary fibrosis, lupus nephritis, polycystic kidney disease and rheumatoid arthritis.

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In this regard, we are supporting investigator sponsored trials, or ISTs, evaluating seliciclib in endocrinologic and inflammatory indications in patients who have failed prior treatments. Specifically an IST for which clinicians at Cedars-Sinai, Los Angeles, were awarded a grant from The National Institute of Diabetes and Digestive and Kidney Diseases to evaluate seliciclib as a potential therapy for Cushing's disease and a European IST for rheumatoid arthritis, which is also being supported by an approximately $1.5 million grant from the United Kingdom's Medical Research Council. Seliciclib may work for RA by targeting proliferating fibroblasts, a different type of approach than conventional RA therapies. As with all ISTs, we do not control the timing or conduct of such studies and will report updates as the investigators may notify us from time to time.

We have entered into a collaboration, licensing and supply agreement with ManRos Therapeutics SA, or ManRos, for the exclusive development and commercialization of our oral seliciclib capsules by ManRos as a treatment for cystic fibrosis, or CF. Among other terms of the agreement, ManRos licensed rights to our proprietary clinical data to enable clinical development of seliciclib for CF indications. We will receive an up-front payment, milestone payments and tiered royalties, if seliciclib is commercialized for the treatment of CF.

Corporate Information

Our corporate headquarters are located at 200 Connell Drive, Suite 1500, Berkeley Heights, New Jersey, 07922, and our telephone number is (908) 517-7330. This is also where our marketing, medical and regulatory functions are located. Our research facility is located in Dundee, Scotland, which is also the center of our translational work and development programs. Our Internet address is *www.cyclacel.com*. The information on our website is not a part of, and should not be construed as being incorporated by reference into, this prospectus supplement or the accompanying prospectus.

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THE OFFERING

Common stock offered by us	Shares of our common stock having an aggregate offering price of up to $8,350,000.
Common stock to be outstanding after this offering	Up to 46,300,131 shares (as more fully described in the notes following this table), assuming sales of 11,597,222 shares of our common stock in this offering at an offering price of $0.72 per share, which was the last reported sale price of our common stock on the NASDAQ Global Market on July 8, 2015. The actual number of shares issued will vary depending on the sales price under this offering.
Manner of offering	"At-the-market" offering that may be made from time to time through our sales agent, Cantor Fitzgerald & Co. See "Plan of Distribution" on page S-19.
Use of Proceeds	We intend to use the net proceeds from this offering, if any, for our operations, for working capital and other general corporate purposes. See "Use of Proceeds" on page S-16.
Risk Factors	You should read the "Risk Factors" section of this prospectus supplement and in the documents incorporated by reference in this prospectus supplement for a discussion of factors to consider before deciding to purchase shares of our common stock.
NASDAQ Global Market symbol	CYCC

The number of common stock to be outstanding immediately after this offering as shown above is based on 34,702,909 shares of common stock outstanding as of July 8, 2015, but does not include the following:

- 1,336,954 shares of common stock issuable upon the exercise of outstanding options at a weighted-average exercise price of $10.96 per share;
- 3,395,000 shares of common stock available for issuance under our 2015 Equity Incentive Plan;
- 1,138,630 shares of common stock issuable upon the exercise of outstanding warrants at a weighted-average exercise price of $11.57 per share; and
- 20,381 shares of common stock, subject to adjustment, that are issuable upon the conversion of 335,273 shares of convertible preferred stock that are issued and outstanding.

Unless otherwise stated, all information contained in this prospectus supplement reflects an assumed public offering price of $0.72 per share, which was the last reported sale price of our common stock on the NASDAQ Global Market on July 8, 2015.

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RISK FACTORS

You should consider carefully the risks described below and discussed under the section captioned "Risk Factors" contained in our annual report on Form 10-K for the year ended December 31, 2014 and in our subsequent quarterly reports on Form 10-Q and annual reports on Form 10-K, as updated by our subsequent filings under the Securities Exchange Act of 1934, as amended, or the Exchange Act, each of which is incorporated by reference in this prospectus supplement and the accompanying prospectus in their entirety, together with other information in this prospectus supplement, the accompanying prospectus and the information and documents incorporated by reference in this prospectus supplement and the accompanying prospectus, and any free writing prospectus that we have authorized for use in connection with this offering before you make a decision to invest in our common stock. If any of the following events actually occur, our business, operating results, prospects or financial condition could be materially and adversely affected. This could cause the trading price of our common stock to decline and you may lose all or part of your investment. The risks described below are not the only ones that we face. Additional risks not presently known to us or that we currently deem immaterial may also affect our business operations.

Risks Related to This Offering

Our management will have broad discretion over the use of any net proceeds from this offering, you may not agree with how we use the proceeds, and the proceeds may not be invested successfully.

Our management will have broad discretion as to the use of any net proceeds from this offering and could use them for purposes other than those contemplated at the time of this offering. Accordingly, you will be relying on the judgment of our management with regard to the use of any proceeds from the sale of shares of common stock in this offering, and you will not have the opportunity, as part of your investment decision, to assess whether the proceeds are being used appropriately. It is possible that the proceeds will be invested in a way that does not yield a favorable, or any, return for Cyclacel.

Our shareholders may experience significant dilution as a result of future equity offerings or issuances and exercise of outstanding options and warrants.

In order to raise additional capital or pursue strategic transactions, we may in the future offer, issue or sell additional shares of common stock or other securities convertible into or exchangeable for shares of our common stock. We cannot assure you that we will be able to sell shares or other securities in any other transaction at a price per share or that have an exercise price or conversion price per shares that is equal to or greater than the price for the securities purchased by investors in this offering, and investors purchasing shares or other securities in the future could have rights superior to existing shareholders. The price per share at which we sell or issue additional shares of common

stock or other securities convertible into or exchangeable for our common stock future transactions may be higher or lower than such price.

Sales of a significant number of shares of common stock in the public markets, or the perception that such sales could occur, could depress the market price of our shares of common stock.

Sales of a substantial number of shares of common stock in the public markets could depress the market price of our shares of common stock and impair our ability to raise capital through the sale of additional equity securities. We cannot predict the effect that future sales of our shares of common stock would have on the market price of our shares of common stock.

We do not intend to pay any cash dividends on our common stock in the foreseeable future and, therefore, any return on your investment in our common stock must come from increases in the fair market value and trading price of our common stock.

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We do not intend to pay any cash dividends on our common stock in the foreseeable future and, therefore, any return on your investment in our common stock must come from increases in the fair market value and trading price of our common stock.

The shares of common stock offered under this prospectus supplement and the accompanying prospectus may be sold in "at-the-market" offerings, and investors who buy shares at different times will likely pay different prices.

Investors who purchase shares under this prospectus supplement and the accompanying prospectus at different times will likely pay different prices, and so may experience different outcomes in their investment results. We will have discretion, subject to market demand, to vary the timing, prices, and numbers of shares sold, and there is no minimum or maximum sales price. Investors may experience declines in the value of their shares as a result of share sales made at prices lower than the prices they paid.

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CAUTIONARY STATEMENT REGARDING FORWARD-LOOKING STATEMENTS

This prospectus supplement and the accompanying prospectus and the documents we have filed with the SEC that are incorporated by reference into this prospectus supplement and the accompanying prospectus contain forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, as amended, or the Securities Act, and Section 21E of the Securities Exchange Act of 1934, as amended, or the Exchange Act. Forward-looking statements reflect our current view about future plans, intentions or expectations. These forward-looking statements may be included herein or incorporated by reference in this prospectus and include, in particular, statements about our plans, strategies and prospects and may be identified by terminology such as “may,” “goal,” “strategy,” “target,” “likely,” “could,” “s “will,” “should,” “expect,” “plan,” “intend,” “anticipate,” “believe,” “estimate,” “aim,” “potential,” or “continue” or the negati terms or other comparable terminology. These forward-looking statements are subject to risks, uncertainties and assumptions about us. Although we believe that our plans, intentions and expectations are reasonable, we may not achieve our plans, intentions or expectations.

Important factors that could cause actual results to differ materially from the forward-looking statements we make in this prospectus supplement and accompanying prospectus are set forth in this prospectus under the caption “Risk Factors”, and in the reports we have filed or will file with the SEC and which are incorporated by reference herein, including statements under the caption “Risk Factors” and “Forward-Looking Statements” in such reports. All forward-looking statements attributable to us or persons acting on our behalf are expressly qualified in their entirety by the cautionary statements in this prospectus under the caption “Risk Factors”, and in the reports we have filed or will file with the SEC and which are incorporated by reference herein, including statements under the caption “Risk Factors” and “Forward-Looking Statements” in such reports, in which we have disclosed the material risks related to our business. These forward-looking statements involve risks and uncertainties, and the cautionary statements identify important factors that could cause actual results to differ materially from those predicted in any forward-looking statements. We undertake no obligation to update any of the forward-looking statements after the date of this prospectus supplement to conform those statements to reflect the occurrence of unanticipated events, except as required by applicable law. You should read this prospectus supplement and the accompanying prospectus and the documents incorporated by reference completely and with the understanding that our actual future results, levels of activity, performance and achievements may be materially different from what we expect. We qualify all of our forward-looking statements by these cautionary statements.

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USE OF PROCEEDS

We cannot assure you that we will receive any proceeds in connection with the shares common stock offered pursuant to this prospectus supplement and the accompanying prospectus. We intend to use any net proceeds from the sale of common stock under this prospectus supplement and the accompanying prospectus for our operations, working capital and other general corporate purposes. As a result, our management will retain broad discretion in the allocation and use of any net proceeds. Pending use of any net proceeds, we would expect to invest any proceeds in a variety of capital preservation instruments, including short-term, investment grade, interest bearing instruments.

DIVIDEND POLICY

We have never declared nor paid any cash dividends on our common stock and do not currently anticipate declaring or paying any cash dividends on our outstanding shares of common stock in the foreseeable future. We are, however, required to make or accrue quarterly dividend payments on our shares of convertible preferred stock. Except for dividends that may be paid on the shares of convertible preferred stock, we currently intend to retain all of our future earnings, if any, to finance operations. Any future determination relating to our dividend policy will be made at the discretion of our Board of Directors and will depend on a number of factors, including future earnings, capital requirements, financial conditions, future prospects, contractual restrictions and other factors that our Board of Directors may deem relevant.

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PLAN OF DISTRIBUTION

We have entered into a Controlled Equity Offering[SM] sales agreement with Cantor Fitzgerald & Co., or Cantor, under which we may issue and sell shares of our common stock from time to time through Cantor acting as agent. Pursuant to this prospectus supplement, we may issue and sell shares of our common stock having an aggregate gross sales price of up to $8,350,000 from time to time through Cantor acting as agent. The sales agreement will be filed as an exhibit to a current report on Form 8-K filed under the Exchange Act and incorporated by reference in this prospectus supplement.

Upon delivery of a placement notice and subject to the terms and conditions of the sales agreement, Cantor may sell our common stock by any method permitted by law deemed to be an "at-the-market" offering as defined in Rule 415 promulgated under the Securities Act, including sales made directly on the NASDAQ Global Market, on any other existing trading market for our common stock or to or through a market maker. Cantor may also sell our common stock by any other method permitted by law, including in privately negotiated transactions. We may instruct Cantor not to sell common stock if the sales cannot be effected at or above the price designated by us from time to time. We or Cantor may suspend the offering of common stock upon notice and subject to other conditions.

We will pay Cantor commissions, in cash, for its services in acting as agent in the sale of our common stock. Cantor will be entitled to compensation at a fixed commission rate of 3.0% of the gross sales price per share sold. Because there is no minimum offering amount required as a condition to close this offering, the actual total public offering amount, commissions and proceeds to us, if any, are not determinable at this time. We have also agreed to reimburse Cantor for certain specified expenses, including the fees and disbursements of its legal counsel in an amount not to exceed $50,000. We estimate that the total expenses for the offering, excluding compensation and reimbursements payable to Cantor under the terms of the sales agreement, will be approximately $140,000.

Settlement for sales of common stock will occur on the third business day following the date on which any sales are made, or on some other date that is agreed upon by us and Cantor in connection with a particular transaction, in return for payment of the net proceeds to us. Sales of our common stock as contemplated in this prospectus will be settled through the facilities of The Depository Trust Company or by such other means as we and Cantor may agree upon. There is no arrangement for funds to be received in an escrow, trust or similar arrangement.

Cantor will use its commercially reasonable efforts, consistent with its sales and trading practices, to solicit offers to purchase the common stock shares under the terms and subject to the conditions set forth in the sales agreement. In connection with the sale of the common stock on our behalf, Cantor will be deemed to be an "underwriter" within the meaning of the Securities Act and the compensation of Cantor will be deemed to be underwriting commissions or discounts. We have agreed to provide indemnification and contribution to Cantor against certain civil liabilities, including liabilities under the Securities Act.

The offering of our common stock pursuant to the sales agreement will terminate upon the termination of the sales agreement as permitted therein. We and Cantor may each terminate the sales agreement at any time upon ten days' prior notice.

Cantor and its affiliates may in the future provide various investment banking, commercial banking and other financial services for us and our affiliates, for which services they may in the future receive customary fees. To the extent required by Regulation M, Cantor will not engage in any market making activities involving our common stock while the offering is ongoing under this prospectus supplement.

This prospectus supplement and the accompanying prospectus in electronic format may be made available on a website maintained by Cantor and Cantor may distribute this prospectus supplement and the accompanying prospectus electronically.