UNITED STATES
SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): April 19, 2020

CYCLACEL PHARMACEUTICALS, INC.

(Exact name of registrant as specified in its charter)

Delaware	**000-50626**	**91-1707622**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

200 Connell Drive, Suite 1500
Berkeley Heights, NJ 07922
(Address of principal executive offices and zip code)
Registrant's telephone number, including area code: (908) 517-7330

(Former Name or Former Address, if Changed Since Last Report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (*see* General Instruction A.2. below):

☐ **Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)**

☐ **Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)**

☐ **Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))**

☐ **Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))**

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter). ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 1.01 Entry into a Material Definitive Agreement.

On April 19, 2020, Cyclacel Pharmaceuticals, Inc. (the "**Company**") entered into a Material Transfer Agreement (the "**MTA**") with The University of Edinburgh ("**Edinburgh**"). The main objective of the MTA is to study fadraciclib (CYC065) and seliciclib (CYC202 or R-roscovitine), the Company's clinical stage CDK2/9 inhibitors, as potential early treatments for the inflammatory response observed in patients with COVID-19 disease.

Under the terms of the MTA, the parties will assess the Company's medicines mentioned above for their suitability for use in safety and experimental medicine studies in COVID-19 patients (the "**Evaluation**"). The Evaluation is part of a broader project ("**STOPCOVID**") studying the inflammatory pathways that lead directly to COVID-19 lung injury. STOPCOVID is supported by a £2 million (approximately $2.5 million) grant from LifeArc, a medical research charity. Edinburgh is seeking further funding.

The MTA shall remain in effect for the duration of the Evaluation. Additionally, each of the Company and Edinburgh may terminate the MTA if the other party commits a breach of its obligations thereunder.

The foregoing summary of the MTA does not purport to be complete and is qualified in its entirety by reference to the MTA, which will be filed as an exhibit to the Company's quarterly report on Form 10-Q for the quarter ending June 30, 2020.

Item 8.01 Other Events.

On April 20, 2020, the Company issued a press release announcing that the Company had entered into the MTA described in Item 1.01 above. A copy of the press release is attached hereto as Exhibit 99.1 and is incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	Description
99.1	Press Release of Cyclacel Pharmaceuticals, Inc., dated April 20, 2020.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the Registrant has duly caused this report to be signed on its behalf by the undersigned thereunto duly authorized.

CYCLACEL PHARMACEUTICALS, INC.

By: /s/ Paul McBarron
Name: Paul McBarron
Title: Executive Vice President—Finance, Chief Financial Officer and Chief Operating Officer

Date: April 20, 2020

Exhibit 99.1

Cyclacel Pharmaceuticals, Inc.

P R E S S R E L E A S E

CYCLACEL PHARMACEUTICALS ANNOUNCES COLLABORATION WITH THE UNIVERSITY OF EDINBURGH TO STUDY ITS CDK INHIBITORS TO REDUCE RUNAWAY INFLAMMATION IN COVID-19 DISEASE

-Evaluation of Fadraciclib and Seliciclib to Promote Apoptosis of Inflammatory Neutrophils in the Setting of COVID-19 Lung Injury-

Berkeley Heights, NJ and Dundee, UK, April 20, 2020 - Cyclacel Pharmaceuticals, Inc. (NASDAQ: CYCC, NASDAQ: CYCCP; "Cyclacel" or the "Company") a biopharmaceutical company developing innovative medicines today announced that it entered into an agreement with the University of Edinburgh to study fadraciclib (CYC065) and seliciclib (CYC202 or R-roscovitine), its clinical stage CDK2/9 inhibitors, as potential early treatments for the inflammatory response observed in patients with COVID-19 disease.

The parties will assess Cyclacel's medicines above for their suitability for use in safety and experimental medicine studies in COVID-19 patients. This evaluation is part of a broader project ("STOPCOVID") studying the inflammatory pathways that lead directly to COVID-19 lung injury, drawing upon more than 30 years of experience from the University of Edinburgh's Centre for Inflammation Research. STOPCOVID is supported by a £2 million (approximately $2.5 million) grant from LifeArc and the University is seeking further funding.

"We are eager to evaluate the potential role of Cyclacel's CDK inhibitors as enablers of inflammatory neutrophil apoptosis," said Professor Kev Dhaliwal, STOPCOVID lead and Consultant in Respiratory Medicine at The University of Edinburgh. "Clinical data from international studies suggest that an early peripheral blood neutrophil response is associated with a poor outcome in COVID-19. If we can stop the inflammatory cascade early, we may be able to prevent or delay the severity of COVID-19 induced inflammation and the need for assisted ventilation in affected patients."

Previously published research from The University of Edinburgh and other investigators have found that CDK inhibitors, including seliciclib, help resolve undesirable inflammation by promoting apoptosis of inflammatory neutrophils. CDK inhibitors were shown to reduce levels of the anti-apoptotic protein Mcl-1 and inhibit transcription of interleukin-6 (IL-6), both of which are believed to be drivers of the overactive systemic inflammatory response severely damaging the lungs of symptomatic COVID-19 patients.

"Our published research showed that the CDK2/9 inhibitor seliciclib, induced human neutrophil apoptosis possibly by suppressing levels of the Mcl-1 anti-apoptotic protein and augmented resolution of inflammation in 'neutrophil dominant' models," said Prof. Adriano G. Rossi, Chair of Respiratory and Inflammation Pharmacology and Deputy Director of the Centre for Inflammation Research. "We are interested in extending these observations with fadraciclib, a second generation CDK inhibitor, as part of our STOPCOVID project and possibly translating our findings to benefit COVID-19 patients."

"We are excited to be contributing fadraciclib and seliciclib to the global effort to combat COVID-19," said Spiro Rombotis, President and Chief Executive Officer of Cyclacel. "As previously reported, our two investigational CDK inhibitors suppress Mcl-1 in patients with proliferative diseases. Seliciclib has also been shown to efficiently suppress IL-6 transcription, a presumptive contributor to COVID-19 cytokine storm. Seliciclib is active in models of lung injury, pleurisy and rheumatoid arthritis and is undergoing clinical investigation in patients with rheumatoid arthritis. We are looking forward to working with The University of Edinburgh team and are humbled by the possibility of helping COVID-19 patients in need."

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About COVID-19 Respiratory Distress & Cytokine Storm

Most COVID-19 infected patients experience mild respiratory problems. Some hospitalized patients require mechanical ventilation because of severe hypoxia and acute lung injury which may lead to rapid decline of respiratory function and death. This condition, often called acute respiratory distress syndrome (ARDS), is thought to be associated with a systemic inflammatory response of the immune system (cytokine storm). Clinical correlates of mortality from an analysis of 150 Chinese patients include old age, sepsis, elevated d-dimer, and increased inflammatory parameters including IL-6 and CRP (C-reactive protein).

About Anti-IL-6 Antibodies as Potential Treatment for COVID-19

IL-6 is produced, along with other cytokines, by the immune system in response to COVID-19 infection and contributes to antiviral defense through the stimulation of a local and general immune response. Once triggered continued synthesis of IL-6 has pathological consequences through chronic inflammation. Evidence points to an association of peak levels of IL-6 with the severity of respiratory symptoms. Humanized anti- IL-6 receptor antibodies, including tocilizumab and sarilumab, have been proposed as potential treatments to attenuate the overactive immune response and have begun evaluation in COVID-19 patients (NCT04317092 and NCT04327388).

Published interim data from a small, uncontrolled Chinese study suggest that IL-6 may be driving the inflammatory immune response that causes ARDS in seriously ill COVID-19 patients. The study showed that treatment with tocilizumab, a humanized anti IL-6 receptor antibody, in 15 of the 20 patients on study resulted in reduced need for oxygen support and improved CRP levels.

About CDK Inhibitors as Potential Treatment for COVID-19

The objective of treating COVID-19 patients with transcriptionally-active cyclin- dependent kinase (CDK) inhibitors is to dampen the overactive immune response in which activated inflammatory neutrophils may contribute. Neutrophil survival is promoted by Mcl-1 expression. Rossi et al showed that seliciclib induced Mcl-1 downregulation and apoptosis of inflammatory neutrophils in models of acute lung injury, arthritis and pleurisy. Raje et al showed that seliciclib inhibited transcription and secretion of IL-6 triggered by multiple myeloma cells adhering to bone marrow stromal cells. Further it was shown that seliciclib was more effective than anti-IL-6 antibody at suppressing Mcl-1 expression.

About the TRAFIC Clinical Study of Seliciclib in Patients with Rheumatoid Arthritis

Seliciclib is being tested in patients with refractory rheumatoid arthritis (RA) in an investigator-sponsored study ('TRAFIC') led by Prof. J. Isaacs and colleagues at Newcastle University and five other UK hospitals. TRAFIC is evaluating seliciclib's potential to help patients with RA by means of reducing proliferation of synovial fibroblasts. As previously reported the TRAFIC Independent Data Monitoring Committee recommended that the study should continue to its second stage which is currently enrolling.

About Cyclacel Pharmaceuticals, Inc.

Cyclacel Pharmaceuticals is a clinical-stage biopharmaceutical company developing innovative cancer medicines based on cell cycle, transcriptional regulation and DNA damage response biology. The transcriptional regulation program is evaluating fadraciclib as a single agent in solid tumors and in combination with venetoclax in patients with relapsed or refractory AML/MDS and CLL. The DNA damage response program is evaluating an oral combination of sapacitabine and venetoclax in patients with relapsed or refractory AML/MDS. An IST is evaluating an oral combination of sapacitabine and olaparib in patients with BRCA mutant breast cancer. The anti-mitotic program is evaluating CYC140, a PLK1 inhibitor, in advanced leukemias/MDS patients. Cyclacel's strategy is to build a diversified biopharmaceutical business focused in hematology and oncology based on a pipeline of novel drug candidates. For additional information, please visit www.cyclacel.com.

Forward-looking Statements

This news release contains certain forward-looking statements that involve risks and uncertainties that could cause actual results to be materially different from historical results or from any future results expressed or implied by such forward-looking statements. Such forward-looking statements include statements regarding, among other things, the efficacy, safety and intended utilization of Cyclacel's product candidates, the conduct and results of future clinical trials, plans regarding regulatory filings, future research and clinical trials and plans regarding partnering activities. Factors that may cause actual results to differ materially include the risk that product candidates that appeared promising in early research and clinical trials do not demonstrate safety and/or efficacy in larger-scale or later clinical trials, trials may have difficulty enrolling, Cyclacel may not obtain approval to market its product candidates, the risks associated with reliance on outside financing to meet capital requirements, and the risks associated with reliance on collaborative partners for further clinical trials, development and commercialization of product candidates. You are urged to consider statements that include the words "may," "will," "would," "could," "should," "believes," "estimates," "projects," "potential," "expects," "plans," "anticipates," "intends," "continues," "forecast," "designed," "goal," or the negative of those words or other comparable words to be uncertain and forward-looking. For a further list and description of the risks and uncertainties the Company faces, please refer to our most recent Annual Report on Form 10-K and other periodic and other filings we file with the Securities and Exchange Commission and are available at www.sec.gov. Such forward-looking statements are current only as of the date they are made, and we assume no obligation to update any forward-looking statements, whether as a result of new information, future events or otherwise.

References

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6. Lucas CD, et al, Mucosal Immunol, 2014 Jul; 7(4): 857–868.
7. Dzhagalov I, et al, Blood, 2007 Feb 15; 109(4): 1620–1626.

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