UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT
PURSUANT TO SECTION 13 OR 15(d)
OF THE SECURITIES EXCHANGE ACT OF 1934

Date of Report (Date of earliest event reported): November 13, 2019

DECIPHERA PHARMACEUTICALS, INC.
(Exact name of registrant as specified in its charter)

Delaware	**001-38219**	**30-1003521**
(State or other jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification Number)**

200 Smith Street **Waltham, MA**	**02451**
(Address of registrant's principal executive office)	**(Zip code)**

(781) 209-6400
(Registrant's telephone number, including area code)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 203.425)

☐ 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 or Rule 12b-2 of the Securities Exchange Act of 1934.

Emerging growth company ☒

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. ☒

Title of each class	Trading Symbol	Name of exchange on which registered
Common Stock, $0.01 Par Value	**DCPH**	**The Nasdaq Global Select Market**

Item 7.01. Regulation FD Disclosure.

On November 13, 2019, Deciphera Pharmaceuticals, Inc., or the Company, issued a press release announcing preliminary data from its ongoing Phase 1 study of DCC-3014, an oral inhibitor of CSF1R, including data in patients with diffuse-type tenosynovial giant cell tumor. The data were presented on November 13, 2019 (November 14, 2019 local time) in a poster session at the Connective Tissue Oncology Society (CTOS) 2019 Annual Meeting being held November 13-16, 2019 in Tokyo, Japan. A copy of the press release is furnished as Exhibit 99.1 to this Current Report on Form 8-K, and a copy of the presentation is furnished as Exhibit 99.2 to this Current Report on Form 8-K.

The furnishing of the attached press release and presentation is not an admission as to the materiality of any information therein. The information contained in the press release and the presentation is summary information that is intended to be considered in the context of more complete information included in the Company's filings with the U.S. Securities and Exchange Commission, or the SEC, and other public announcements that the Company has made and may make from time to time by press release or otherwise. The Company undertakes no duty or obligation to update or revise the information contained in this report, although it may do so from time to time as its management believes is appropriate. Any such updating may be made through the filing of other reports or documents with the SEC, through press releases or through other public disclosures. For important information about forward looking statements, see the "Cautionary Note Regarding Forward-Looking Statements" section of the press release in Exhibit 99.1 attached hereto.

The information in this Item 7.01 of this Current Report on Form 8-K and Exhibit 99.1 and Exhibit 99.2 attached hereto shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that section or Sections 11 and 12(a)(2) of the Securities Act of 1933, as amended. The information contained in this Item 7.01 and in the press release attached as Exhibit 99.1 and in the presentation attached as Exhibit 99.2 to this Current Report shall not be incorporated by reference into any filing with the SEC made by the Company, whether made before or after the date hereof, regardless of any general incorporation language in such filing, except as expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description
99.1	Press Release issued by Deciphera Pharmaceuticals, Inc. on November 13, 2019, furnished herewith.
99.2	Presentation from November 13, 2019, furnished herewith.

SIGNATURES

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

Date November 13, 2019

DECIPHERA PHARMACEUTICALS, INC.

By: /s/ Steven L. Hoerter
Steven L. Hoerter
President and Chief Executive Officer

Deciphera Pharmaceuticals, Inc. to Present Data from DCC-3014 and Ripretinib Programs at the Connective Tissue Oncology Society (CTOS) 2019 Annual Meeting

- Preliminary Anti-tumor Activity Observed in Initial Diffuse-type TGCT Patients Treated with DCC-3014 in Ongoing Phase 1 Study -

- DCC-3014 was Generally Well Tolerated with No Reported Grade 3 or Higher TEAEs in Initial Diffuse-Type TGCT Patients -

- Encore Presentation of Results from the INVICTUS Pivotal Phase 3 Study of Ripretinib in Advanced GIST to be Featured in Oral Presentation Session -

Waltham, MA – November 13, 2019 – Deciphera Pharmaceuticals, Inc. (NASDAQ:DCPH) today announced preliminary data from the ongoing Phase 1 study of DCC-3014, an oral inhibitor of CSF1R, including initial data in diffuse-type tenosynovial giant cell tumor (TGCT) patients as well as an encore presentation of the INVICTUS pivotal Phase 3 study of ripretinib, a broad-spectrum KIT and PDGFRα inhibitor, in patients with advanced gastrointestinal stromal tumors (GIST). Results from these programs will be presented at the Connective Tissue Oncology Society (CTOS) 2019 Annual Meeting being held November 13-16 in Tokyo, Japan.

“We are excited to share preliminary data from the initial TGCT patients enrolled in the ongoing Phase 1 study of DCC-3014. While this program in TGCT is in its early stages, we are encouraged by the preliminary evidence of anti-tumor activity and emerging tolerability profile,” said Matthew L. Sherman, M.D., Executive Vice President and Chief Medical Officer of Deciphera. “We plan to continue to enroll TGCT patients to further explore the potential of DCC-3014, with the goal of making a meaningful impact on disease progression and, importantly, quality of life for patients with TGCT.”

Preliminary Data from DCC-3014 in Initial TGCT Patients

The Company’s Phase 1 study of DCC-3014 was designed to evaluate the safety, pharmacokinetics, and pharmacodynamics of multiple doses of DCC-3014 in patients with advanced solid tumors and TGCT. Tumor reductions from baseline were determined by investigator assessment by Response Evaluation Criteria in Solid Tumors (RECIST) version 1.1. The CTOS presentation highlights preliminary results from the initial three TGCT patients enrolled in the dose-escalation portion of the Phase 1 study. Safety, pharmacokinetic, and pharmacodynamic data were analyzed as of September 10, 2019, with additional anti-tumor activity data reported as of November 8, 2019.

- All three patients with diffuse-type TGCT treated as of the data analyses dates showed preliminary anti-tumor activity.
    - As of their first tumor assessment at Cycle 3 Day 1, tumor reductions from baseline of 48%, 25% and 24%, respectively, were observed.
    - One patient had a confirmed partial response, which has been sustained for nine months and is ongoing as of the most recent investigator report, with a tumor reduction from baseline of 84% as of Cycle 10 Day 1.
- Symptomatic improvements in mobility and reduced pain, as reported by the investigator, were observed.
- These patients were enrolled in Cohort 5 (30 mg loading dose daily for 5 days followed by a maintenance dose of 30 mg twice a week).

- DCC-3014 was generally well-tolerated, with no grade 3 or higher treatment-emergent adverse events (TEAEs) observed.
- Two patients remained on study as of the November data analyses date. One patient discontinued in Cycle 4 due to relocation outside of the U.S.
- Dose-escalation evaluation is ongoing to determine the recommended Phase 2 dose for advanced solid tumors and diffuse-type TGCT.

Results from the INVICTUS Pivotal Phase 3 Study of Ripretinib

An encore presentation of results from the INVICTUS pivotal Phase 3 study of ripretinib in advanced GIST will be featured during an oral presentation session. INVICTUS is a randomized (2:1), double-blind, placebo-controlled, international, multicenter study to evaluate the safety, tolerability, and efficacy of ripretinib compared to placebo in 129 patients with advanced GIST whose previous therapies have included at least imatinib, sunitinib, and regorafenib. As previously reported, the study achieved the primary endpoint of improved progression free survival (PFS) compared to placebo in patients with fourth-line and fourth-line plus GIST, as determined by blinded independent central radiologic review using modified RECIST version 1.1.

Based on the positive INVICTUS data, the Company expects to submit an NDA to the U.S. Food and Drug Administration (FDA) for ripretinib for the treatment of patients with advanced GIST who have prior treatment with imatinib, sunitinib and regorafenib in the first quarter of 2020.

Presentation Details

Poster Presentation:

Poster Title: Phase 1 study of DCC-3014 to assess the safety, tolerability, pharmacokinetics, and pharmacodynamics, in patients with malignant solid and diffuse-type tenosynovial giant cell tumor
Author: Breelyn Wilky, M.D., Associate Professor, Department of Medicine, Division of Medical Oncology, University of Colorado Cancer Center, University of Colorado School of Medicine
Poster Viewing Reception Date and Time: Thursday, November 14, 2019, 5:30 – 6:30 PM JST
Location: 3rd Floor, Hilton Tokyo Hotel
Abstract Number: 3241734

Oral Presentation:

Poster Title: INVICTUS: A Phase 3, interventional, double-blind, placebo-controlled study to assess the safety and efficacy of ripretinib (DCC-2618) in patients with advanced gastrointestinal stromal tumors (GIST) who have received treatment with prior anticancer therapies (NCT03353753)
Session Title: GIST
Author: Jean-Yves Blay, M.D., General Director, Centre Léon Bérard and Université Claude Bernard Lyon 1, Lyon, France
Presentation Date and Time: Friday, November 15, 2019, 1:00- 1:12 PM JST
Location: Kiku Ballroom, Hilton Tokyo Hotel
Abstract Number: 3254072

A copy of each presentation is available at www.deciphera.com/science/presentation-publications/.

About DCC-3014

DCC-3014 is an investigational, orally administered, potent and highly selective inhibitor of CSF1R. DCC-3014 was designed using the Company's proprietary switch control kinase inhibitor platform to selectively bind to the CSF1R switch pocket. DCC-3014 has greater than 100-fold selectivity for CSF1R over other closely related kinases and has an even greater selectivity for CSF1R over approximately 300 other human kinases. CSF1R controls the differentiation and function of macrophages including tumor-associated macrophages (TAMs) whose density within certain tumors including cancers of the breast, cervix, pancreas, bladder and brain, as well as tenosynovial giant cell tumors (TGCT), correlates with poor prognosis. Tumors induce TAMs to suppress a natural immune response mediated by cytotoxic T-cells, a type of lymphocyte that would otherwise eradicate the tumor; a process known as macrophage checkpoints. Through inhibition of CSF1R, DCC-3014 has in preclinical studies demonstrated potent macrophage checkpoint inhibition as both a single agent and in combination with PD1 inhibitors. DCC-3014 is currently being evaluated in a Phase 1 clinical study. For more information about the clinical trial design please visit www.clinicaltrials.gov (NCT03069469).

About Ripretinib

Ripretinib is an investigational tyrosine kinase switch control inhibitor that was engineered to broadly inhibit KIT and PDGFRα mutated kinases by using a unique dual mechanism of action that regulates the kinase switch pocket and activation loop. Ripretinib is currently in clinical development for the treatment of KIT and/or PDGFRα-driven cancers, including gastrointestinal stromal tumors, or GIST, systemic mastocytosis, or SM, and other cancers. Ripretinib inhibits initiating and secondary KIT mutations in exons 9, 11, 13, 14, 17, and 18, involved in GIST, as well as the primary D816V exon 17 mutation involved in SM. Ripretinib also inhibits primary PDGFRα mutations in exons 12, 14 and 18, including the exon 18 D842V mutation, involved in a subset of GIST. In June 2019, the U.S. FDA granted Fast Track Designation to ripretinib for the treatment of patients with advanced GIST who have received prior treatment with imatinib, sunitinib and regorafenib. In October 2019, FDA granted Breakthrough Therapy Designation (BTD) for ripretinib for the treatment of patients with advanced GIST who have received prior treatment with imatinib, sunitinib and regorafenib. For more information about the Company's clinical trials with ripretinib, please visit www.clinicaltrials.gov.

Deciphera Pharmaceuticals has an exclusive license agreement with Zai Lab (Shanghai) Co., Ltd. for the development and commercialization of ripretinib in Greater China (Mainland China, Hong Kong, Macau and Taiwan). Deciphera Pharmaceuticals retains development and commercial rights for ripretinib in the rest of the world.

About Deciphera Pharmaceuticals

Deciphera Pharmaceuticals is a clinical-stage biopharmaceutical company focused on improving the lives of cancer patients by addressing key mechanisms of drug resistance that limit the rate and/or durability of response to existing cancer therapies. Our small molecule product candidates are directed against an important family of enzymes called kinases, known to be directly involved in the growth and spread of many cancers. We use our deep understanding of kinase biology together with a proprietary chemistry library to purposefully design compounds that maintain kinases in a "switched off" or inactivated conformation. These investigational therapies comprise tumor-targeted agents designed to address therapeutic resistance causing mutations and immuno-targeted agents designed to control the activation of immunokinases that suppress critical immune system regulators, and agents designed to inhibit reprogramming of cancer cell metabolism. We have used our platform to develop a diverse pipeline of tumor-targeted, immuno-targeted, and metabolism-targeted product candidates designed to improve

outcomes for patients with cancer by improving the quality, rate and/or durability of their responses to treatment.

About Deciphera Pharmaceuticals

Deciphera Pharmaceuticals is a clinical-stage biopharmaceutical company focused on improving the lives of cancer patients by addressing key mechanisms of drug resistance that limit the rate and/or durability of response to existing cancer therapies. Our small molecule product candidates are directed against an important family of enzymes called kinases, known to be directly involved in the growth and spread of many cancers. We use our deep understanding of kinase biology together with a proprietary chemistry library to purposefully design compounds that maintain kinases in a "switched off" or inactivated conformation. These investigational therapies comprise tumor-targeted agents designed to address therapeutic resistance causing mutations and immuno-targeted agents designed to control the activation of immunokinases that suppress critical immune system regulators, and agents designed to inhibit reprogramming of cancer cell metabolism. We have used our platform to develop a diverse pipeline of tumor-targeted, immuno-targeted, and metabolism-targeted product candidates designed to improve outcomes for patients with cancer by improving the quality, rate and/or durability of their responses to treatment.

Cautionary Note Regarding Forward-Looking Statements

This press release contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, as amended, including, without limitation, statements regarding our expectations regarding our ongoing Phase 1 study of DCC-3014, our plans to continue to enroll TGCT patients in this study, the potential benefits of DCC-3014 in patients with TGCT and other cancers, our planned potential NDA submission with FDA for ripretinib for patients with advanced GIST and the timing of such filing, the breakthrough therapy designation of ripretinib for patients with advanced GIST, and the potential of our pipeline product candidates to improve the lives of patients with cancer. The words "may," "will," "could," "would," "should," "expect," "plan," "anticipate," "intend," "believe," "estimate," "predict," "project," "potential," "continue," "target" and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Any forward-looking statements in this press release are based on management's current expectations and beliefs and are subject to a number of risks, uncertainties and important factors that may cause actual events or results to differ materially from those expressed or implied by any forward-looking statements contained in this press release, including, without limitation, risks and uncertainties related to the delay of any current or planned clinical studies or the development of our product candidates, including ripretinib, our ability to successfully demonstrate the efficacy and safety of our product candidates including in later-stage studies, the preclinical and clinical results for our product candidates, which may not support further development of such product candidates, the possibility that results experienced in early, preliminary, top-line or initial data, including preliminary data from initial TGCT patients in our Phase 1 study of DCC-3014, may not be indicative of the results experienced in final data, our ability to timely complete and prepare the information required for and file an NDA for ripretinib, the fact that receipt of a breakthrough therapy designation for a product candidate, such as ripretinib, may not result in us receiving any of the benefits of such designation, our ability to manage and our reliance on third parties such as our third party drug substance and drug product contract manufacturers, actions of regulatory agencies, any or all of which may affect the initiation, timing and progress of clinical studies and the timing of and our ability to obtain regulatory approval, if at all, and make our investigational drugs available to patients, and other risks identified in our SEC filings, including our Quarterly Report on Form 10-Q for the quarter ended September 30, 2019, and subsequent filings with the SEC. We caution you not to place undue reliance on any forward-looking statements, which speak only as of the date they are made. We disclaim any obligation to publicly update or revise any such

statements to reflect any change in expectations or in events, conditions or circumstances on which any such statements may be based, or that may affect the likelihood that actual results will differ from those set forth in the forward-looking statements. Any forward-looking statements contained in this press release represent our views only as of the date hereof and should not be relied upon as representing its views as of any subsequent date. We explicitly disclaim any obligation to update any forward-looking statements.

Contacts:
Investor Relations:
Jen Robinson
Deciphera Pharmaceuticals, Inc
jrobinson@deciphera.com
781-906-1112

Media:
David Rosen
Argot Partners
David.Rosen@argotpartners.com
212-600-1902

Phase 1 study of DCC-3014 to assess the safety, tolerability, pharmacokinetics, and pharmacodynamics in patients with malignant solid tumors and diffuse-type tenosynovial giant cell tumor

Breelyn Wilky[1], Matthew Taylor[2], Todd Bauer[3], Stephen Leong[1], Gege Tan[4], Cynthia Leary[4], Xiaoyan Li[4], Keisuke Kuida[4], Rodrigo Ruiz Soto[4], Lara E. Davis[2]

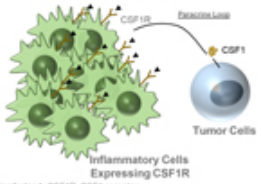
[1]Division of Medical Oncology, University of Colorado, Denver, CO, USA; [2]Knight Cancer Institute, Oregon Health & Science University, Portland, OR, USA; [3]Sarah Cannon Research Institute and Tennessee Oncology, Nashville, TN, USA; [4]Deciphera Pharmaceuticals, LLC, Waltham, MA, USA;

Abstract: 3241734

INTRODUCTION

- Colony stimulating factor 1 receptor (CSF1R) is a receptor tyrosine kinase that is implicated in the recruitment and survival of tumor-associated macrophages (TAMs) through a paracrine interaction with tumor cells in the tumor microenvironment[1,2]
  - CSF1R has 2 known ligands; CSF1 (also known as macrophage-CSF) and interleukin 34 (IL-34)[3]
- Tenosynovial giant cell tumor (TGCT; formerly known as pigmented villonodular synovitis [PVNS] or giant cell tumor of the tendon sheath) is a rare disease arising from the joint synovium, bursa, and tendon sheath caused by translocation in CSF1 gene resulting in overexpression of CSF1 and recruitment of CSF1R-positive inflammatory cells into the lesion (**Figure 1**)[4]
  - TGCT presents either as localized (a single, well-defined nodule) or diffuse-type with multiple nodules that are more aggressive

Figure 1. Role of CSF1R in development of TGCT



CSF1, colony stimulating factor 1; CSF1R, CSF1 receptor

- DCC-3014 is an orally administered, potent, and selective inhibitor of CSF1R that was engineered to bind as a switch control inhibitor of CSF1R and inhibit kinase activity[5]
  - DCC-3014 potently inhibits CSF1R signaling in cellular assays, as well as blocks macrophage-mediated tumor cell migration, osteoclast differentiation, and proliferation of a CSF1R-dependent cell line
  - DCC-3014 is designed to inhibit macrophages that contribute to, or are the source of, tumor development and dissemination
- DCC-3014 exhibits >100-fold selectivity for CSF1R relative to closely-related kinases, including FLT3, KIT, and PDGFRα/β, and >1,000-fold selectivity vs other kinases[5]
- An ongoing phase 1 study (NCT03069469) was initiated to evaluate the safety, preliminary antitumor activity, pharmacokinetics (PK) and pharmacodynamics (PD) of DCC-3014 in advanced solid tumors, including diffuse-type TGCT

METHODS

- This is a phase 1 multicenter, open-label, single arm study of DCC-3014 in advanced solid tumors including diffuse-type TGCT
- The study consists of two parts:
  - Part 1 (dose escalation) will determine the recommended phase 2 dose (RP2D) and the maximum tolerated dose (MTD) using a 3+3 dose escalation design with a minimum of 3 patients enrolled at each dose level cohort; starting at a dose of 10 mg once daily (**Tables 1–3**)
    - Loading doses used in Cohort 2 and subsequent cohorts were based on PK profiles observed in Cohort 1
  - Part 2 (dose expansion) will evaluate the safety, tolerability, preliminary antitumor activity, PK, and PD in two expansion cohorts: advanced solid tumors and diffuse-type TGCT

Table 1. Dose cohorts in Part 1 (3+3 dose escalation)

	Loading doses	Dose
Cohort 1	None	10 mg QD
Cohort 2	10 mg QD x 5 days	10 mg twice a week
Cohort 3	20 mg QD x 5 days	20 mg once a week
Cohort 4	20 mg QD x 5 days	20 mg twice a week
Cohort 5	30 mg QD x 5 days	30 mg twice a week
Cohort 6	40 mg QD x 5 days	40 mg twice a week
Cohort 7	50 mg QD x 3 days	20 mg QD

QD, once daily.

Table 2. Key inclusion and exclusion criteria for Part 1

Inclusion criteria
≥18 years old
Tumors with known contribution of macrophages or phagocytes
Symptomatic diffuse-type TGCT patients for which surgical resection is not an option
Exclusion criteria
Prior anticancer therapy or other investigational therapy ≤14 days or <28 days if half-life longer than 3 days
Unresolved toxicity according to NCI-CTCAE, >grade 1 or baseline, from previous therapy
Known active CNS metastases
History or presence of clinically relevant cardiovascular abnormalities
Major surgery within 2 weeks of first dose

NCI-CTCAE, National Cancer Institute common terminology criteria for adverse events; NYHA, New York Heart Association; TGCT, tenosynovial giant cell tumor.

Table 3. Study endpoints

Primary endpoints
Safety and tolerability (including occurrence of DLTs and incidence of TEAEs*)
RP2D/MTD
Pharmacokinetics (including T_{max}, C_{max}, C_{trough}, AUC, $t_{1/2}$)

RESULTS

Safety

- Among treatment-emergent adverse events (TEAEs) occurring in ≥10% of patients (regardless of relatedness), most events were grade 1 or 2 (**Table 5**)
  - Grade ≥3 related TEAEs occurred in 4 patients (grade 3 aspartate aminotransferase [AST] increased, grade 4 lipase increased, grade 3 amylase increased, and grade 3 colitis)
  - No grade ≥3 TEAEs in diffuse-type TGCT patients
- Serious adverse events (SAEs) were reported in 17 malignant solid tumor patients; none of which were related to DCC-3014
  - No SAEs were reported in diffuse-type TGCT patients

Table 5. Common (≥10%) TEAEs regardless of relatedness

Preferred term	Advanced solid tumor total n = 36		Diffuse-type TGCT n = 3		Total (All patients) n = 39	
	All	*≥G3*	*All*	*≥G3*	*All*	*≥G3*
Constipation	13 (36.1)	0	1 (33.3)	0	14 (35.9)	0
Vomiting	12 (33.3)	2 (5.6)	1 (33.3)	0	13 (33.3)	2 (5.1)
Diarrhea	10 (27.8)	0	1 (33.3)	0	11 (28.2)	0
Nausea	10 (27.8)	0	1 (33.3)	0	11 (28.2)	0
Fatigue	8 (22.2)	2 (5.6)	2 (66.7)	0	10 (25.6)	2 (5.1)
Decreased appetite	9 (25)	1 (2.8)	0	0	9 (23.1)	1 (2.6)
Dyspnea	8 (22.2)	0	1 (33.3)	0	9 (23.1)	0
Abdominal pain	7 (19.4)	3 (8.3)	1 (33.3)	0	8 (20.5)	3 (7.7)
AST increased	5 (13.9)	1 (2.8)[a]	3 (100)	0	8 (20.5)	1 (2.6)
Dehydration	7 (19.4)	0	0	0	7 (17.9)	0
Pyrexia	6 (16.7)	0	1 (33.3)	0	7 (17.9)	0
Arthralgia	5 (13.9)	1 (2.8)	1 (33.3)	0	6 (15.4)	1 (2.6)
Back pain	5 (13.9)	0	1 (33.3)	0	6 (15.4)	0
Blood CPK increase	4 (11.1)	0	2 (66.7)	0	6 (15.4)	0
Anemia	5 (13.9)	1 (2.8)	0	0	5 (12.8)	1 (2.6)
Asthenia	5 (13.9)	0	0	0	5 (12.8)	0
Cough	4 (11.1)	0	1 (33.3)	0	5 (12.8)	0
Headache	3 (8.3)	1 (2.8)	2 (66.7)	0	5 (12.8)	1 (2.6)
Pain in extremity	5 (13.9)	0	0	0	5 (12.8)	0
Periorbital edema	4 (11.1)	0	1 (33.3)	0	5 (12.8)	0
Urinary tract infection	4 (11.1)	0	1 (33.3)	0	5 (12.8)	0
Abdominal distension	4 (11.1)	0	0	0	4 (10.3)	0
Depression	4 (11.1)	0	0	0	4 (10.3)	0
Dyspepsia	4 (11.1)	0	0	0	4 (10.3)	0
Hypokalemia	4 (11.1)	1 (2.8)	0	0	4 (10.3)	1 (2.6)
Insomnia	4 (11.1)	0	0	0	4 (10.3)	0
Edema peripheral	4 (11.1)	0	0	0	4 (10.3)	0
Pain	3 (8.3)	2 (5.6)	1 (33.3)	0	4 (10.3)	2 (5.1)

*Grade 2 by the central laboratory assessment.
AST, aspartate aminotransferase; CPK, creatine phosphokinase; G, grade; TGCT, tenosynovial giant cell tumor.

- There were 2 dose-limiting toxicities (DLTs) in the first cohort (10 mg QD): grade 4 lipase increased and grade 3 hypocalcemia
  - Both DLTs could be explained by the mechanism of action of DCC-3014; therefore, any grade of asymptomatic serum enzyme elevation and grade 3 hypocalcemia were excluded from DLTs for evaluation of subsequent cohorts
  - Dose density of Cohort 2 (the total amount of DCC-3014 given in Cycle 1) was lowered from that of Cohort 1 (**Table 1**), and then subsequently increased
- No further DLTs were reported in the other cohorts

Laboratory results

- Increases in alanine transaminase (ALT) and AST are considered as an on-target mechanism of action of DCC-3014
  - Grade 1 AST elevations were observed in 84% of patients
  - Grade 2 AST elevations were seen in 8% of patients
  - Grade 1 ALT elevations were seen in 29% of patients
  - Asymptomatic and mostly not reported as AEs
  - Similar increases have been reported with other anti-CSF1R therapies[2]
- No bilirubin elevations were observed by treatment with DCC-3014

Pharmacokinetics and pharmacodynamics

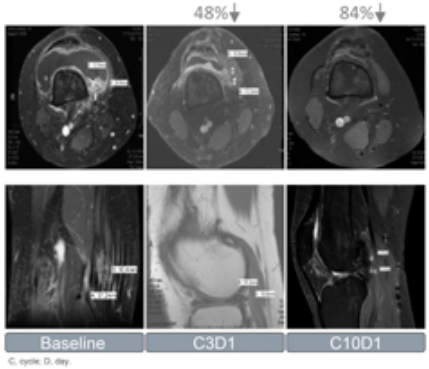
- DCC-3014 exposure is consistent between diffuse-type TGCT and solid tumor patients
- DCC-3014 treatment caused a dose-related rise in plasma CSF1 and IL-34 and a reduction of CD16+ monocytes in diffuse-type TGCT

Case studies

- Symptomatic improvements are based on descriptive notes obtained from investigators
- Adverse events are summarized in **Table 5**
- Changes in tumor size from baseline by investigator assessment per RECIST version 1.1 are summarized in **Figure 3**

Patient 1

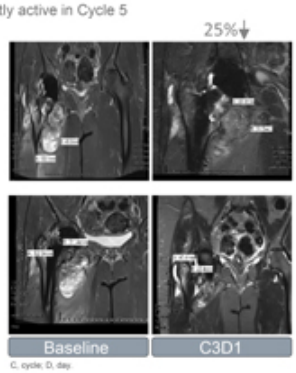
- 24-year-old female patient diagnosed with diffuse-type TGCT (right posterior knee) in Jun 2016
- Prior surgeries: synovectomies/mass resections in Jun 2016, Jul 2016, and Dec 2017
- Recurrence/progression on MRI by Dec 2018
- Enrolled in Feb 2019 in Cohort 5
- Symptom improvement/tumor assessment on the study
  - Taking Mobic and Percocet daily at baseline with inadequate pain control
    - On C10D1, taking Percocet only as needed approximately once a week
  - Improved pain and swelling, effusion nearly resolved in the first cycle
- Change in tumor size: 48%, 61%, 75%, and 84% decreases from baseline (C3D1, C5D1, C7D1, and C10D1 scan, respectively) per RECIST
- Currently active in Cycle 10



C, cycle; D, day.

Patient 2

- 57-year-old female patient diagnosed with diffuse-type TGCT (right hip) in 2014
- Prior surgeries: resection (May 2014), synovectomy (Aug 2015 and Aug 2016), total hip replacement (Aug 2016), hip revision and resection (Aug 2018), cryoablation (May 2019)
- Recurrent disease on MRI (Feb 2019)
- Enrolled in July 2019 in Cohort 5
- Symptom improvement/tumor assessment on the study
  - Pain improved, walking 1 mile, increased range of motion, and less stiffness
  - Change in tumor size: 25% decrease from baseline on C3D1 scan per RECIST
- Currently active in Cycle 5



C, cycle; D, day.

Patient 3

- 28-year-old male patient diagnosed with diffuse-type TGCT (left knee) in Jan 2016 after several years of pain
- Prior surgery: resection and posterior synovectomy (Jan 2016)
- Pain, swelling, and stiffness recurred due to disease progression not long after surgery
- Enrolled in Mar 2019 in Cohort 5
- Symptom improvement/tumor assessment on the study
  - Rapid symptom improvement, with less pain and swelling and improved range of motion after the first cycle

Relevant exploratory endpoints

- Pharmacodynamics
  - Levels of CSF1/IL-34 in plasma
  - Levels of circulating CD16+ monocytes in blood by flow cytometry
  - Macrophage content and/or polarization in tumor
- Tumor response assessment by RECIST version 1.1

[a]Adverse events graded by NCI-CTCAE, Version 4.03.
AUC, area under the curve; C_{max}, maximum concentration; CSF1, colony stimulating factor; C_{trough}, trough concentration; DLT, dose limiting toxicities; IL-34, interleukin 34; MTD, maximum tolerated dose; NCI-CTCAE, National Cancer Institute common terminology criteria for adverse events; RP2D, recommended phase 2 dose; RECIST, response evaluation criteria in solid tumors; TEAE, treatment emergent adverse events; $t_{1/2}$, half-life; T_{max}, time of C_{max}.

Types of tumors

- As of September 10, 2019, 39 patients were enrolled (**Table 4**), including 3 patients with diffuse-type TGCT in Cohort 5

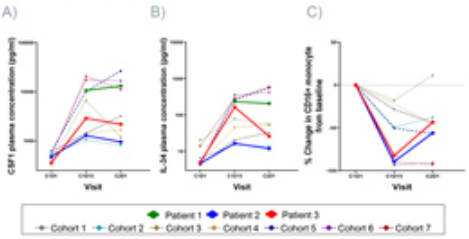
Table 4. Tumor type

	Total (n = 39)
Colorectal cancer	8 (21)
Pancreatic cancer	5 (13)
Ovarian cancer	4 (10)
Diffuse-type TGCT	3 (8)
Prostate	3 (8)
Other[a]	16 (41)

All values n (%).
[a]Leiomyosarcoma (2), liver cancer (2), uterine cancer (2), anal cancer, breast cancer, chondrosarcoma, endometrial, gastroesophageal junction, melanoma, uveal melanoma, synovial sarcoma, non-small cell lung cancer, thymus. TGCT, tenosynovial giant cell tumor.

patients (Figure 2)

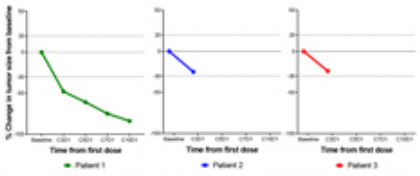
Figure 2. Changes in levels of circulating A) CSF1 and B) IL-34 in plasma and C) changes in levels of whole blood CD16+ monocytes



A and B: Levels of CSF1 and IL-34 in plasma were determined by standard ELISA. Plasma samples were collected from patients on Cycle 1 Day 1, Cycle 1 Day 15, and Cycle 2 Day 1. C: Levels of CD16+ monocytes were assessed by flow cytometry. Whole blood samples were collected from patients on Cycle 1 Day 1, Cycle 1 Day 15, and Cycle 2 Day 1.
C, cycle; CSF1, colony stimulating factor 1; D, day; IL-34, interleukin 34.

- Able to play basketball with no pain
- Change in tumor size: 24% decrease from baseline on C3D1 scan per RECIST
- Discontinued in Cycle 4 due to relocation to the outside of US
- Patient did not consent for inclusion of MRI images in publication

Figure 3. Changes from baseline in tumor size assessed per RECIST version 1.1



Dashed lines denote 30% decrease and 20% increase in tumor size threshold for partial response and progressive disease, respectively, per RECIST version 1.1
C, cycle; D, day; RECIST, response evaluation criteria in solid tumors.

Conclusion

- In this phase 1 study, DCC-3014 was generally well tolerated in patients with malignant solid tumors and diffuse-type TGCT
- All 3 patients with diffuse-type TGCT treated with DCC-3014 to date showed rapid, preliminary anti-tumor activity by cycle 3
  - One patient had a confirmed partial response by cycle 3; sustained for 9 months and ongoing as of last investigator report
- Symptomatic improvements in mobility and reduced pain were observed in all 3 diffuse-type TGCT patients
- Exposure to DCC-3014 was consistent between malignant solid tumor and diffuse-type TGCT patients and associated with an increase in plasma CSF1 and IL-34 in plasma, and a rapid, sustained reduction of CD16+ monocytes in peripheral blood
- Dose-escalation evaluation is ongoing to determine the recommended phase 2 dose for advanced solid tumors and diffuse-type TGCT
- These results are encouraging and support further evaluation of DCC-3014 in diffuse-type TGCT

Presented at the Connective Tissue Oncology Society (CTOS) Annual Meeting; Tokyo, Japan; November 13–16, 2019

Acknowledgments

This study was sponsored by Deciphera Pharmaceuticals, LLC. Medical writing and editorial support were provided by Nicole Seneca, PhD; and Stefan Kolata, PhD, of AlphaBioCom, LLC (King of Prussia, PA).

References

1) Lin Y, et al. *J Hematol Oncol.* 2019; 12:76; **2)** Cannarile MA, et al. *J Immunother Cancer.* 2017; 5:53; **3)** Dron N, et al. *J Leukoc Biol.* 2010; 87:745-747; **4)** Gouin F, et al. Orthop Traumatol Surg Res. 2017; 103:591-597; **5)** Smith BD, et al. AACR Annual Meeting; April 16–20, 2016; New Orleans, LA.