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

FORM 8-K

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

Date of report (Date of earliest event reported): **December 5, 2016**

TG Therapeutics, Inc.
(Exact Name of Registrant as Specified in Charter)

Delaware	**001-32639**	**36-3898269**
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(IRS Employer Identification No.)

2 Gansevoort Street, 9th Floor
New York, New York 10014
(Address of Principal Executive Offices)

(212) 554-4484
(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.
☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act.
☐ Pre-commencement communications pursuant to Rule 14d-2b under the Exchange Act.
☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act.

Item 8.01. Other Events.

On December 5, 2016, TG Therapeutics, Inc. issued press releases announcing certain data regarding preclinical and clinical studies of TGR-1202 at the 58th American Society of Hematology (ASH) annual meeting in San Diego, CA. Copies of the press releases are being filed as Exhibits 99.1 and 99.2 and incorporated in this Item by reference.

Item 9.01 Financial Statements And Exhibits.

(d) Exhibits.

99.1 Press Release, dated December 5, 2016.

99.2 Press Release, dated December 5, 2016

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.

TG Therapeutics, Inc.
(Registrant)

Date: December 5, 2016

By: /s/ Sean A. Power
Sean A. Power
Chief Financial Officer

INDEX TO EXHIBITS

Exhibit Number	**Description**
99.1.	Press Release, dated December 5, 2016
99.2	Press Release, dated December 5, 2016

Exhibit 99.1

TG Therapeutics, Inc. Announces Preclinical Data Presentations for TGR-1202 at the 58th American Society of Hematology Annual Meeting

Preclinical work may offer rationale for the differentiated activity and safety effects of TGR-1202

SAN DIEGO, CA (December 5, 2016) - TG Therapeutics, Inc. (NASDAQ:TGTX), announced the presentation yesterday of two preclinical data sets, one oral presentation and one poster presentation, for TGR-1202, the Company's once-daily PI3K delta inhibitor, at the 58th American Society of Hematology (ASH) annual meeting in San Diego, California.

Michael S. Weiss, the Company's Executive Chairman and Interim Chief Executive Officer, stated, "We want to thank the teams at Columbia and Moffitt for their extensive laboratory work on TGR-1202 to better understand the mechanism of action and impact on the immune system. The preclinical data they have generated helps to better explain and perhaps offer a rationale for the differentiated safety profile seen with TGR-1202 as compared to earlier generation PI3K delta inhibitors. We believe these preclinical findings along with the robust safety and efficacy data we have observed in the clinic, support our belief that TGR-1202 is a differentiated best in class PI3K delta inhibitor. We look forward to continuing our research collaborations with Columbia and Moffitt and to presenting updated safety and efficacy data for TGR-1202 to further confirm its unique profile."

"Dr. Deng's presentation today has really begun to shed some long-needed light on the important differences among the PI3K delta inhibitors. His work has identified that a novel kinase important in the PI3K pathway, CK-1epsilon, is uniquely inhibited by TGR-1202, which may explain the drug's effects on c-Myc. These chemical differences may also help to explain the important immunologic differences in the safety profiles of these agents," stated Dr. Owen A. O'Connor, Professor of Medicine and Experimental Therapeutics, Director Lymphoid Malignancies at Columbia Presbyterian Medical Center.

The following summarizes the oral presentation and poster presentation which occurred yesterday:

***Oral Presentation:* Silencing c-Myc Translation as a Therapeutic Strategy through Targeting PI3K Delta and CK1 Epsilon in Hematological Malignancies (Abstract Number 291)**

This oral presentation includes data from the manuscript titled, "Silencing c-Myc Translation as a Therapeutic Strategy through Targeting PI3K Delta and CK1 Epsilon in Hematological Malignancies," which was recently published in *Blood*, the Journal of the American Society of Hematology. The presentation was delivered by Changchung Deng, MD, PhD of Columbia Presbyterian Medical Center and included the following highlights:

- TGR-1202 and carfilzomib, but not combinations of other drugs in the same classes, synergistically inhibit c-Myc translation and c-Myc dependent gene transcription, by potently inhibiting phosphorylation of 4E-BP1;
- TGR-1202 and carfilzomib synergistically induce apoptosis in lymphoma cells through targeting c-Myc, whereas the other combinations did not;
- TGR-1202, but not idelalisib or duvelisib, was found to uniquely inhibit casein kinase-1 (CK1) epsilon; and
- Based on this extensive preclinical work, the Company recently announced the launch of a Phase 1/2 study to evaluate the safety and efficacy of TGR-1202 in combination with carfilzomib, in patients with relapsed or refractory lymphoma.

Poster Presentation: ***Modulation of T Cell Compartment in a Preclinical CLL Murine Model By a Selective PI3K Delta Inhibitor, TGR-1202 (Abstract Number 3236)***

This poster presentation included preclinical data describing the differential regulation of human T-cells by TGR-1202 in a preclinical CLL murine model. Highlights from this poster include:

- Both TGR-1202 and duvelisib oral administration demonstrated comparable efficacy by reducing CLL burden over time in leukemic mice;
- TGR-1202 and duvelisib both targeted the T cell population *in vivo*, however:
- TGR-1202 relatively maintained the number of Tregs and Th17 cells and expression of functional markers on Tregs compared to duvelisib treatment *in vivo* and *ex vivo*; and
- Duvelisib resulted in greater disruption of Treg/Th17 ratio compared to TGR-1202 *in vivo*, which may have implications for occurrence of autoimmune-like organ toxicity.

PRESENTATION DETAILS:

Copies of the above referenced presentations are available on the Company's website at www.tgtherapeutics.com, located on the Publications page.

TG THERAPEUTICS INVESTOR & ANALYST EVENT DETAILS:

TG Therapeutics will also host an investor and analyst reception on Monday, December 5th, 2016 beginning at 8:00pm PT. The event will take place at the Marriott Gaslamp, in San Diego, California, in the Presidio AB Ballroom.

NOTE: *This event will be webcast live and will be available on the Events page, located within the Investors & Media section of the Company's website at www.tgtherapeutics.com, as well as archived for future review. This event will also be broadcast via conference call. In order to access the conference line, please call 1-877-407-8029 (U.S.), 1-201-689-8029 (outside the U.S.), and reference Conference Title: TG Therapeutics 2016 Investor & Analyst Event.*

ABOUT TG THERAPEUTICS, INC.

TG Therapeutics is a biopharmaceutical company focused on the acquisition, development and commercialization of novel treatments for B-cell malignancies and autoimmune diseases. Currently, the company is developing two therapies targeting hematological malignancies and autoimmune diseases. TG-1101 (ublituximab) is a novel, glycoengineered monoclonal antibody that targets a specific and unique epitope on the CD20 antigen found on mature B-lymphocytes. TG Therapeutics is also developing TGR-1202, an orally available PI3K delta inhibitor. The delta isoform of PI3K is strongly expressed in cells of hematopoietic origin and is believed to be important in the proliferation and survival of B-lymphocytes. Both TG-1101 and TGR-1202 are in clinical development for patients with hematologic malignancies, with TG-1101 recently entering clinical development for autoimmune disorders. The Company also has pre-clinical programs to develop IRAK4 inhibitors, BET inhibitors, and anti-PD-L1 and anti-GITR antibodies. TG Therapeutics is headquartered in New York City.

Cautionary Statement

Some of the statements included in this press release, particularly those with respect to anticipating future clinical trials, the timing of commencing or completing such trials and business prospects for TG-1101, TGR-1202, the IRAK4 inhibitor program, the BET inhibitor program, and the anti-PD-L1 and anti-GITR antibodies may be forward-looking statements that involve a number of risks and uncertainties. For those statements, we claim the protection of the safe harbor for forward-looking statements contained in the Private Securities Litigation Reform Act of 1995. Among the factors that could cause our actual results to differ materially are the following: our ability to successfully and cost-effectively complete preclinical and clinical trials for TG-1101, TGR-1202, the IRAK4 inhibitor program, the BET inhibitor program, and the anti-PD-L1 and anti-GITR antibodies; the risk that early preclinical and clinical results that supported our decision to move forward with TG-1101, TGR-1202, the IRAK4 inhibitor program, the BET inhibitor program, and the anti-PD-L1 and anti-GITR antibodies will not be reproduced in additional patients or in future studies; the risk that trends observed which underlie certain assumptions of future performance of TGR-1202 will not continue, the risk that TGR-1202 will not produce satisfactory safety and efficacy results to warrant further development following the completion of the current Phase 1 study; the risk that the combination of TG-1101 and TGR-1202, referred to as TG-1303, will not prove to be a safe and efficacious backbone for triple and quad combination therapies; the risk that the data (both safety and efficacy) from future clinical trials will not coincide with the data produced from prior preclinical and clinical trials; the risk that trials will take longer to enroll than expected; our ability to achieve the milestones we project over the next year; our ability to manage our cash in line with our projections, and other risk factors identified from time to time in our reports filed with the Securities and Exchange Commission. Any forward-looking statements set forth in this press release speak only as of the date of this press release. We do not undertake to update any of these forward-looking statements to reflect events or circumstances that occur after the date hereof. This press release and prior releases are available at www.tgtherapeutics.com. The information found on our website is not incorporated by reference into this press release and is included for reference purposes only.

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Exhibit 99.2

TG Therapeutics, Inc. Announces Oral Data Presentation for TGR-1202 in Combination with Ibrutinib in Patients with Relapsed or Refractory CLL or MCL at the 58th American Society of Hematology Annual Meeting

Combination of TGR-1202 plus ibrutinib is well-tolerated with no Grade 3/4 transaminitis, pneumonitis, diarrhea, or colitis observed, with patients approaching two years on therapy

88% ORR in CLL patients, including 1 CR, and 73% ORR in MCL patients treated with the combination

SAN DIEGO, CA (December 5, 2016) - TG Therapeutics, Inc. (NASDAQ:TGTX), today announced the presentation of combination data from a Phase 1b study evaluating TGR-1202, the Company's once-daily PI3K delta inhibitor, in combination with ibrutinib, the oral Bruton's tyrosine kinase (BTK). This study is being run in collaboration with the Blood Cancer Research Partnership (BCRP) and Dana-Farber Cancer Institute (DFCI), Boston, MA. Data from this trial were presented today by the Principal Investigator, Matthew S. Davids, MD, of Dana-Farber Cancer Institute, during an oral session at the 58th American Society of Hematology (ASH) annual meeting in San Diego, CA.

Michael S. Weiss, the Company's Executive Chairman and Interim CEO commenting on the data said, "We and our investigators continue to be impressed with the ease of combining TGR-1202 with other novel agents. Here Dr. Davids demonstrated that the double blockade of the BCR pathway with dual oral once-daily inhibitors can be performed safely and conveniently, and derives high response rates in patients with both CLL and MCL. This all oral approach offers a unique and highly active alternative for patients who do not want to receive infused therapies but still want to advance their treatment beyond single agent ibrutinib, which may offer multiple benefits over single agent therapy. With one bone marrow confirmed complete response (CR) and 5 additional deep responses nearing radiographic CR out of the 17 evaluable CLL patients, we and our investigators believe we are seeing activity beyond what one might expect from either of these agents alone. We want to thank Dr. Davids and his collaborators at DFCI and the Leukemia & Lymphoma Society for providing support for this important investigator driven research and we look forward to follow-up data in the future from this study and data from our own triple therapy of TG-1101 plus TGR-1202 plus ibrutinib, which we are targeting for presentation next year."

The following summarizes the key highlights from this oral presentation which occurred today:

Oral Presentation:* *TGR-1202 in Combination with Ibrutinib in Patients with Relapsed or Refractory CLL or MCL: Preliminary Results of a Multicenter Phase I/Ib Study (Abstract Number 641)

This oral presentation includes data from patients with relapsed or refractory chronic lymphocytic leukemia (CLL) or mantle cell lymphoma (MCL) treated with TGR-1202 in combination with Ibrutinib. 31 patients were evaluable for safety (18 CLL patients and 13 MCL patients), of which 28 patients were available for efficacy (17 CLL patients and 11 MCL patients.) CLL patients had a median of 1.5 prior lines of therapy (range 1-6), with 2 patients receiving prior ibrutinib and 4 receiving prior PI3K inhibitors. MCL patients had a median of 3 prior lines of therapy (range 2-5), also with 2 patients receiving prior ibrutinib.

Highlights from this oral presentation include:

- 88% (15 of 17) Overall Response Rate (ORR) (including Complete Response (CR), Partial Response (PR), and Partial Response with lymphocytosis (PR-L)) in patients with CLL, with 1 patient achieving a bone marrow confirmed CR and 5 patients with a >80% nodal reduction, nearing radiographic CR
- 1 year progression free survival (PFS) and overall survival (OS) for CLL is 94% (n=17), with the longest patient on study approaching two years
- 73% (8/11) ORR in patients with MCL, with clinical benefit observed in two additional patients
- 1 year PFS and OS for MCL is 37% and 52%, respectively (n=11)
- The combination appears well tolerated across all patients with no grade 3/4 transaminitis (liver toxicity), diarrhea, colitis or pneumonitis observed

PRESENTATION DETAILS:

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