December 5, 2016

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, Dec. 05, 2016 (GLOBE NEWSWIRE) -- 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.
TGTX - G

CONTACT:

Jenna Bosco

Vice President, Investor Relations

TG Therapeutics, Inc.

Telephone: 212.554.4351

Email: ir@tgtxinc.com

Source: TG Therapeutics, Inc.

News Provided by Acquire Media