December 7, 2015

TG Therapeutics, Inc. Announces Oral Presentation of Novel Pre-Clinical Combinations With TGR-1202 by Investigators at Columbia University at the 57th American Society of Hematology Annual Meeting

TGR-1202 Combinations Uniquely Able to Modulate c-Myc Activity, With Significant Potential in the Treatment of Diffuse-Large B-Cell Lymphoma and Other Malignancies

ORLANDO, Fla, Dec. 07, 2015 (GLOBE NEWSWIRE) -- TG Therapeutics, Inc. (Nasdaq:TGTX), today announced the presentation of pre-clinical data describing the synergy of the Company's next generation PI3K-delta inhibitor, TGR-1202, with proteasome inhibitors in various hematologic cell lines and patient donor cells. The oral presentation was delivered by Changchun Deng, MD, PhD, Assistant Professor, Center of Lymphoid Malignancies, Columbia University Medical Center at the 57th American Society of Hematology (ASH) Annual Meeting and Exposition being held at the Orange County Convention Center in Orlando, FL.

Combination data was generated using TGR-1202, the PI3k-delta inhibitor idelalisib, and the proteasome inhibitors carfilzomib and bortezomib. Data revealed that the combination of TGR-1202 and carfilzomib was uniquely synergistic as compared to any other combination of a PI3K-delta inhibitor and proteasome inhibitor, including the combination of idelalisib and cafilzomib and idelalisib and bortezomib. These data were generated as part of a large pre-clinical research collaboration with the Center for Lymphoid Malignancies, whereby the activity and mechanism of action of TGR-1202 is being studied in a variety of *in-vitro* and *in-vivo* models.

Presently there are no agents approved that specifically target c-Myc, an oncogene often found constitutively active in a variety of cancers, including Diffuse Large B-Cell Lymphoma, and has recently been the target of a class of drugs knows as BET (bromodomain and extraterminal domain family) inhibitors. The combination of TGR-1202 and carfilzomib was found to potently inhibit cap dependent translation of c-Myc in all cell lines tested, including DLBCL, mantle cell lymphoma, multiple myeloma, T-cell lymphoma, and CLL cells. In these cell lines, inhibition of c-Myc expression resulted in increased caspase 3/7 activity and complete cleavage of PARP, both mechanisms of apoptosis. Importantly, the combination was not found to be cytotoxic when evaluated on healthy patient lymphocytes indicating the specificity towards malignant cells. As a result of these data, the combination of TGR-1202 and carfilzomib is intended to be studied in a Phase I/II clinical trial to be led by investigators at Columbia University Medical Center.

Commenting on the data, Owen A. O'Connor, MD, PhD, Professor of Medicine and Experimental Therapeutics, and Director of the Center for Lymphoid Malignancies at Columbia University Medical Center stated, "The development of agents that have the ability to inhibit the expression or activity of c-Myc, a key driver in a large variety of hematologic and solid-tumor malignancies, has long been an area of focused research which to date has yielded modest results. The potential for this unique combination is far reaching, and begins to explain the differentiated pharmacologic profile demonstrated by TGR-1202 in patients. We look forward to continuing to elucidate the mechanisms for TGR-1202's unique tolerability and efficacy, as well as evaluating this combination in patients in our upcoming Phase I/II study."

Michael S. Weiss, the Company's Executive Chairman and Interim CEO stated, "TGR-1202 has demonstrated strong activity with a differentiated safety and tolerability profile in patients across a variety of clinical trials, and we are eager to explore and understand the mechanisms that contribute to TGR-1202's potential best-in-class attributes. We thank the investigators at Columbia University, especially Dr. Deng and Dr. O'Connor, for all their efforts on this important research program."

PRESENTATION DETAILS

A copy of the slides used for the oral presentation is available on the Company's website at www.tgtherapeutics.com, located on the Publications Page, within the Pipeline section.

TG THERAPEUTICS INVESTOR & ANALYST EVENT DETAILS

TG Therapeutics will also host a reception on Monday, December 7th, 2015 beginning at 7:45pm ET, with featured presentations beginning promptly at 8:00pm ET. The event will take place at the Hyatt Regency Orlando in the Bayhill 17/18 Room. 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 2015 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. 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, alone and in combination with each other (when combined referred to as "TG-1303"), are in clinical development for patients with hematologic malignancies. The Company also has a pre-clinical program to develop IRAK4 inhibitors, as well as an antibody research program to develop 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 possible success of those trials and business prospects for TG-1101, TGR-1202, TG-1303, the IRAK4 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 pre-clinical and clinical trials for TG-1101, TGR-1202, TG-1303, the IRAK4 inhibitor program and the anti-PD-L1 and anti-GITR antibodies; the risk that early pre-clinical and clinical results particularly pre-clinical combinations with TGR-1202 that supported our decision to move forward with TG-1101, TGR-1202, TG-1303, the IRAK4 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 and TG-1303 will not continue, the risk that TGR-1202 or TG-1303 will not produce satisfactory safety and efficacy results to warrant further development following the completion of the current Phase 1 studies; the risk that the combination of 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 pre-clinical 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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