FOR IMMEDIATE RELEASE

TRILLIUM TO PROVIDE UPDATE ON CD47 PROGRAM AT THE 2016 AMERICAN ASSOCIATION FOR CANCER RESEARCH ANNUAL MEETING

Toronto, Ontario, April 14, 2016 – Trillium Therapeutics Inc. (Nasdaq:TRIL; TSX: TR) an immuno-oncology company developing innovative therapies for the treatment of cancer, today announced it will be providing an update on its SIRPaFc immune checkpoint inhibitor program, targeting the CD47 protein, at the 107th Annual Meeting of the American Association for Cancer Research. The meeting will be held April 16-20, 2016 in New Orleans, LA. Details of the poster presentation, entitled “SIRPaFc, a CD47-Blocking Cancer Immunotherapeutic, Triggers Phagocytosis of Lymphoma Cells by Both Classically (M1) and Alternatively (M2) Activated Macrophages”, are listed below:

Date: Monday April 18, 2016  
Time: 1:00 pm – 5:00 pm (CT)  
Session Category: Immunology  
Session Title: Immune Checkpoints 1  
Abstract #: 2345  
Presenter: Dr. Natasja Nielsen Viller  
Location: Section 26

The company will present data demonstrating that its SIRPaFc fusion protein, which targets the CD47 “do not eat” signal, promotes the phagocytosis of lymphoma cells by diverse types of macrophages. The studies also assess the impact of macrophage polarizing agents on drug activity and delineate the role of different Fc gamma receptors in promoting tumor cell killing by SIRPaFc.

“Macrophages are heterogeneous and certain types, notably M2s, are often implicated in tumor progression,” commented Trillium’s Chief Scientific Officer, Dr. Robert Uger. “Our data indicate that TTI-621, our CD47-blocking decoy receptor, enables all macrophage subsets tested, including M2s, to kill tumor cells. These results suggest that TTI-621 is able to convert otherwise pro-tumor macrophages into efficient anti-tumor effector cells. Rather
than ablating M2 macrophages in the tumor microenvironment, these data support using TTI-621 to unleash their tumoricidal function.”

**About Trillium Therapeutics:**
Trillium Therapeutics Inc. is a clinical stage immuno-oncology company developing innovative therapies for the treatment of cancer. The Company’s lead program, SIRPaFc, is a fusion protein that consists of the CD47-binding domain of human SIRPa linked to the Fc region of a human immunoglobulin. It is designed to act as a soluble decoy receptor, preventing CD47 from delivering its inhibitory (“do not eat”) signal. Neutralization of the inhibitory CD47 signal enables the activation of macrophage anti-tumor effects by pro-phagocytic (“eat”) signals. A Phase 1 clinical trial ([NCT02663518](https://clinicaltrials.gov/ct2/show/NCT02663518)) evaluating SIRPaFc (TTI-621) is ongoing. Trillium also has a proprietary medicinal chemistry platform, using unique fluorine chemistry, which permits the creation of new chemical entities from validated drugs and drug candidates with improved pharmacological properties. Stemming from this platform, the Company’s most advanced preclinical program is an orally-available bromodomain inhibitor, followed by an epidermal growth factor receptor antagonist with increased uptake in the brain. In addition, a number of compounds directed at undisclosed immuno-oncology targets are currently in the discovery phase.

For more information visit: [www.trilliumtherapeutics.com](http://www.trilliumtherapeutics.com)

**Caution Regarding Forward-Looking Information:**
This press release may contain forward-looking statements, which reflect Trillium’s current expectation regarding future results, events or developments, including our belief that TTI-621 is able to convert pro-tumor macrophages into efficient anti-tumor effector cells. These forward-looking statements involve risks and uncertainties that may cause actual results, events or developments to be materially different from any future results, events or developments expressed or implied by such forward-looking statements. Such risks and uncertainties are described in the company’s ongoing quarterly and annual reporting. Except as required by applicable securities laws, Trillium undertakes no obligation to publicly update or revise any forward-looking statements, whether as a result of new information, future events or otherwise.

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