This presentation may contain forward-looking statements, which reflect Trillium's current expectation regarding future events. 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 factors include, but are not limited to, Trillium's ability to obtain financing to advance the products in its development portfolio; changing market conditions; the successful and timely completion of pre-clinical and clinical studies; the establishment of corporate alliances; the impact of competitive products and pricing; new product development risks; uncertainties related to the regulatory approval process or the ability to obtain drug product in sufficient quantity or at standards acceptable to health regulatory authorities to complete clinical trials or to meet commercial demand; and other risks detailed from time to time in Trillium's ongoing quarterly and annual reporting. Forward-looking statements are made only as of the date of this presentation and 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.
Investment Highlights

- Immuno-oncology company developing a next generation checkpoint inhibitor, engaging multiple arms of the immune system
- Lead programs targets CD47, a “do not eat” signal tumor cells exploit to escape destruction by the immune system
- TTI-621 is being evaluated in two clinical studies (intravenous and intratumoral administration)
- Multiple anti-tumor responses observed in both trials
- Proprietary fluorine-based medicinal chemistry platform generating a pipeline of pre-clinical oncology assets
Trillium Pipeline: A Clinical Focus on CD47 Blockade with a Platform for Generating New Oncology Assets

<table>
<thead>
<tr>
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<th>Discovery</th>
<th>Preclinical</th>
<th>Phase 1</th>
<th>Phase 2</th>
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<tbody>
<tr>
<td>SIRPαFc (CD47 Blockade – Monotherapy &amp; Combinations)</td>
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<td>Hematologic malignancies + select solid tumors</td>
<td>Solid tumors + mycosis fungoides</td>
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<td>TTI-621 (Systemic)</td>
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<td>TTI-621 (Intratumoral)</td>
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<td>Solid tumors + mycosis fungoides</td>
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<td>TTI-622</td>
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<td>Combination therapy</td>
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<td>Fluorine Medicinal Chemistry Platform</td>
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<td>EGFR Inhibitor</td>
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<td>High brain penetration for glioblastoma &amp; brain metastases</td>
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<td>Undisclosed IO Targets</td>
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Many Tumor Cells Use the CD47 “Do Not Eat” Signal to Inhibit Macrophage Phagocytosis

CD47 delivers an inhibitory “do not eat” signal to macrophages through SIRPα

Many hematologic and solid tumors express high levels of CD47

High CD47 expression often correlates with aggressive disease and poor clinical outcomes
TTI-621: A Dual Function SIRPαFc Decoy Receptor that is Differentiated from Other CD47 Blocking Agents

CD47 binding domain of human SIRPα

Blocks the DO NOT EAT signal from CD47 but does not bind CD47+ red blood cells

Human IgG1 Fc

Delivers an EAT signal to macrophages through FcγRs; differentiated from IgG4 antibodies

TTI-621 is a dual function decoy receptor that blocks the suppressive CD47 signal while engaging activating Fc receptors
TTI-621 Has the Properties of an Ideal CD47 Blocking Agent

☑ Contains IgG1 Fc to deliver an activating “eat” signal to enhance monotherapy activity

☑ Minimizes likelihood of anemia

☑ Avoids the RBC antigen sink

☑ No interference with transfusion medicine testing

☑ Contains non-mutated sequences to reduce risk of immunogenicity
TTI-621 Activates the Innate and Adaptive Immune Systems

CD47 inhibits macrophage phagocytosis

SIRPαFc binds to CD47

SIRPαFc triggers activating FcγRs on macrophages

Phagocytosis leads to antigen presentation and activation of the adaptive immune system

©Audra Geras
Multicenter, open-label phase 1 study in patients with relapsed/refractory mycosis fungoides or other percutaneously accessible solid tumors (NCT02890368)

Direct intratumoral injection is employed to enhance both local and systemic anti-tumor activity

TTI-621 is being evaluated as monotherapy and in combination with:
- Anti-PD-1/PD-L1
- IFN-α
- Talimogene laherparepvec (T-vec)
- Radiation therapy

Monotherapy data (N=18) presented at ASH 2017:
- Intratumoral injections were very well tolerated
- Rapid responses observed in heavily pre-treated mycosis fungoides patients
Intratumoral TTI-621 Monotherapy is Very Well Tolerated and Results in Rapid Tumor Regression in MF Patients

- No dose-limiting toxicity observed following direct intratumoral injections of TTI-621

- A rapid reduction in CAILS scores, which measures local lesion responses, was observed in 9/10 mycosis fungoides patients and a reduction in circulating leukemic Sézary cells was observed in 3/3 patients

An 85 year old man with Stage IIB mycosis fungoides with large cell transformation in whom 4 prior systemic therapies, PUVA and radiation therapy failed received a single 10 mg injection of TTI-621 delivered directly into the proximal lesion on the left foot. By week 4, both proximal and distal lesions demonstrated considerable improvement.
Further Examples of Rapid Tumor Regression in MF Patients

A 72 year old man with Stage IIB mycosis fungoides with large cell transformation in whom prior topical therapy had failed received a **single 1 mg injection** of TTI-621 delivered directly into the lesion on the dorsal surface of the left foot. The injected lesion became edematous by day 3 and demonstrated steady and continued resolution to a loco-regional complete response over subsequent weeks.

A 61 year old man with Stage IIB mycosis fungoides in whom 3 prior systemic therapies and radiation therapy failed received **six 10 mg injections** (M/W/F X 2) of TTI-621 delivered directly into the anterior tumor lesion denoted by “I” at Baseline. At week 1, confluent, loco-regional tumor masses had resolved and this clinical improvement was maintained at week 3.
Intravenous Administration Study (TTI-621-01)

- Multicenter, open-label phase 1 study in patients with relapsed/refractory heme malignancies and solid tumors (NCT02663518)
- TTI-621 is being evaluated as monotherapy and in combination with:
  - Rituximab in CD20+ lymphoma
  - Anti-PD-1 in Hodgkin lymphoma
- Patients receive weekly IV infusions starting at 0.2 mg/kg with subsequent dose intensification to 0.5 mg/kg
- Dose escalation data (N=18) reported at ASH 2016 and expansion phase data (N=89) reported at ASH 2017:
  - Weekly IV infusions are well tolerated
  - Objective responses observed in patients with heavily pre-treated, relapsed/refractory DLBCL
Weekly Infusions of TTI-621 are Well Tolerated in Over 100 Treated Patients

- Manageable infusion-related reactions
- Attenuated thrombocytopenia after the first dose
- Stable hemoglobin levels, consistent with lack of RBC binding by TTI-621
- Preliminary experience indicates that patients can be safely dose intensified beyond 0.2 mg/kg

Percentage change (pre- to 4 hours post dose) in platelets and hemoglobin at weeks 1, 2 and 3
Multiple Patients with Relapsed/Refractory DLBCL Achieved Objective Responses Following Systemic TTI-621

- Systemic TTI-621, particularly in combination with rituximab, resulted in objective responses in 5/18 evaluable patients with heavily pre-treated DLBCL
- Several other patients experienced prolonged progression-free intervals

62-year old male with DLBCL who had received 8 prior therapies, including 5 prior CD20-based regimens, received 0.1 mg/kg TTI-621 and rituximab. Pseudoprogression was noted at Week 4 and a complete metabolic response (CMR) was observed at Week 12.
TTI-621 – Broad Clinical Effort with a Focus on Emerging Signals of Clinical Activity in T Cell Lymphoma

**Intratumoral Trial (TTI-621-02):**
- Further characterize the efficacy of repeat intratumoral TTI-621 monotherapy in patients with CTCL
- Probe the biological effects of TTI-621 on the tumor microenvironment following intratumoral administration
- Explore combinations with other immunomodulatory therapies

**Intravenous Trial (TTI-621-01):**
- Expand enrollment of T cell lymphoma patients, including CTCL and PTCL in separate cohorts
- Mandated dose intensification to increase exposure
- Explore combinations with monoclonal antibodies
Expanding our CD47 Pipeline with TTI-622

- TTI-621 is the most potent SIRPαFc format
- TTI-622 is less likely to deplete platelets, enabling higher exposures
- Both agents may have unique combination opportunities
- Both agents are differentiated from antibodies by a lack of binding to human erythrocytes
- IND submitted in Q4/17; clinical study to begin in H1/18
Trillium’s Innovative Chemistry Platform – Creating Differentiated New Medicines with Fluorine

- Approximately 25% of all marketed drugs contain fluorine
- Block sites of metabolism to increase half-life and reduce toxicity
- Electronegativity alters chemical properties to improve binding & potency
- Lipophilicity improves oral absorption and blood-brain-barrier (BBB) penetration

Innovative proprietary chemistry allows access to an unprecedented class of novel fluorinated molecules
Approved EGFR Inhibitors Exhibit Poor CNS Penetration

- Epidermal growth factor receptor (EGFR) is a receptor tyrosine kinase involved in the pathogenesis of many cancers.
- Multiple lines of evidence indicate that EGFR is a target in brain cancer, but none of the EGFR-targeted drugs on the market are FDA approved for CNS tumors.
- Clinical testing of EGFR inhibitors in brain cancers has yielded mixed results.
- Poor blood-brain barrier (BBB) penetration (<5%) limits efficacy of existing agents.
- There is an urgent unmet medical need for novel BBB-penetrant EGFR inhibitors.

EGFR-targeted drugs approved by FDA

- HNSCC: head and neck squamous cell carcinoma
- Lung: Erlotinib, Gefitinib, Afatinib, Osimertinib
- Pancreatic: Erlotinib
- Colorectal: Cetuximab, Panitumumab

HNSCC: head and neck squamous cell carcinoma
TTI-2341 as a Potential Best-in-class Brain-penetrant Covalent EGFR Inhibitor

- TTI-2341 is a novel covalent inhibitor of wild type and mutant EGFR developed with Trillium’s proprietary fluorine chemistry

- Compared to the benchmark compounds, TTI-2341 exhibits:
  - Similar \textit{in vitro} potency to EGFR mutant targets
  - Superior \textit{in vitro} ADME properties
  - Greater oral bioavailability and systemic exposure
  - Enhanced BBB penetration and free brain exposure

- TTI-2341 is amenable to radiolabeling with $^{18}$F for use as a PET imaging probe

- Future development steps have been identified, IND submission can be achieved in 12-15 months
## Capitalization and Intellectual Property

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<tr>
<th><strong>SHARES OUTSTANDING</strong></th>
<th>19.3M Common &amp; Preferred</th>
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<tbody>
<tr>
<td><strong>CASH AND MARKETABLE SECURITIES</strong></td>
<td>$81.8M CAD as of December 31, 2017</td>
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<tr>
<td><strong>INSTITUTIONAL OWNERSHIP</strong></td>
<td>~70%</td>
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</tbody>
</table>
| **INTELLECTUAL PROPERTY** | • Two SIRPαFc patent families covering method of use and composition of matter through 2030 and 2033  
• Eleven patent families covering fluorine small molecule therapeutics and medical uses through 2035 and beyond |
Trillium Therapeutics Inc. (NASDAQ/TSX:TRIL) is an immuno-oncology company dedicated to the discovery and development of novel and innovative cancer therapies.