**Strong Cancer Immunotherapy Pipeline**

- Comprehensive portfolio of adenosine receptor antagonists
- Differentiated programs vs. competition (best-in-class / first-in-class)
- Large market potential

**Partnered with $100bn + Company**

- Exclusive worldwide rights acquired from the TATA Group, a $100bn + company with deep R&D expertise

**Strong Management Team**

- Founders each have 20+ years of R&D, healthcare investment, capital markets, and pharma deal-making experience
- Supported by highly experienced Scientific Advisory Board

**Multiple Inflection Points**

- **Major Value Inflection Points Near & Mid Term**
  - Filing of IND for 3 drugs within 6-8 months
  - Clinical data in multiple cancer indications within 18 months

- **Low Development Risk**
  - Validated pathway in multiple diseases
  - Strong efficacy and extensive safety data generated for IND filing
  - Mode of action and clinical efficacy successfully evidenced for adenosine receptor antagonism

- **High Valuation for Comparators**
  - Comparator companies valued between $100-$400 million
  - Tarus positioned favorably against competition with differentiated assets

- **Partnering Opportunity for Non Cancer Indications**
  - Upside potential from out-licensing non-cancer applications
  - Tarus has generated extensive data in Parkinson’s, Lung Fibrosis, Sickle Cell Disease and IBD (Ulcerative Colitis)

**Partnered with $100bn + Company**

- Exclusive worldwide rights acquired from the TATA Group, a $100bn + company with deep R&D expertise
Adenosine is a key suppressor of immune cells in the tumor microenvironment (TME)

Extracellular adenosine elicits a major immuno-suppressive signal through adenosine A2A and A2B receptors, thwarting anti-tumor immunity

Adenosine’s role in immune suppression is corroborated by observations that the TME has significantly elevated concentrations (100-500 fold) of extracellular adenosine

A critical mechanism of cancer immune evasion is the generation of high levels of immunosuppressive adenosine via the purinergic pathway within the tumor microenvironment

Extracellular adenosine has a marked dampening effect on the immune response, suppressing effector cell function and stabilizing immunosuppressive regulatory cells

Upon engagement of the A2A or A2B receptors, adenosine triggers increased adenylyl cyclase activity with concomitant increases in intracellular cAMP resulting in profound dampening of the immune response – a fundamental mechanism of cancer immune evasion

Accordingly, it is a high priority target for immunotherapeutic intervention

Targeting Adenosine Receptors

A Breakthrough Approach for Cancer Immunotherapy

• Selective inhibition of adenosine receptors can markedly enhance anti-tumor immunity

Adenosine A2A and A2B receptors mediate profound tumor resistance

• Over-expression of A2AR and A2BR leads to poor prognosis in multiple cancers

• Genetic ablation of A2AR and A2BR leads to spontaneous regression of established tumors
### Tarus Strongly Positioned with the Most Comprehensive Portfolio of Adenosine Receptor Antagonists in the Industry

<table>
<thead>
<tr>
<th>Company</th>
<th>Lead programs</th>
<th>Target</th>
<th>Status</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tarus</td>
<td>TT-10, TT-4, TT-3, TT-53</td>
<td>A2AR, A2BR, A2BR-Gut, Dual</td>
<td>IND-enabling</td>
<td></td>
</tr>
<tr>
<td>Arcus</td>
<td>AB928</td>
<td>Dual</td>
<td>Phase Ib</td>
<td>NASDAQ listed (RCUS)</td>
</tr>
<tr>
<td>Corvus</td>
<td>CPI-444</td>
<td>A2AR</td>
<td>Phase II</td>
<td>NASDAQ listed (CRVS)</td>
</tr>
<tr>
<td>iTeos</td>
<td>EOS100850</td>
<td>A2AR</td>
<td>Phase I</td>
<td>$125M Series B (April ’20)</td>
</tr>
<tr>
<td>AdoRx</td>
<td>A2AR Antagonist</td>
<td>A2AR</td>
<td>Discovery</td>
<td>$10M Series A (June’18)</td>
</tr>
<tr>
<td>AZN/Heptares</td>
<td>AZD4635</td>
<td>A2AR</td>
<td>Phase Ib</td>
<td>$500M+ deal value</td>
</tr>
<tr>
<td>Merck/Domain</td>
<td>AR Inhibitors</td>
<td>AR</td>
<td>Discovery</td>
<td>$290M+ deal value</td>
</tr>
<tr>
<td>Novartis/ Palobiofarma</td>
<td>PBF-509</td>
<td>A2AR</td>
<td>Phase II</td>
<td>$15M upfront plus milestones &amp; royalties</td>
</tr>
</tbody>
</table>
## Oncology Programs

<table>
<thead>
<tr>
<th>Program</th>
<th>Indication</th>
<th>IND-enabling</th>
<th>Phase 1a/b</th>
<th>Phases 2/3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>TT-10</strong> A2AR Inhibitor</td>
<td>Solid tumors</td>
<td>IND-filing 4Q’20</td>
<td>1Q’21</td>
<td></td>
</tr>
<tr>
<td><strong>TT-4</strong> A2BR Inhibitor</td>
<td>Solid tumors</td>
<td>IND-filing 4Q’20</td>
<td>1Q’21</td>
<td></td>
</tr>
<tr>
<td><strong>TT-3</strong> Gut-restricted A2BR Inhibitor</td>
<td>Colorectal I, GI tumors</td>
<td>IND-filing 4Q’20</td>
<td>1Q’21</td>
<td></td>
</tr>
<tr>
<td><strong>TT-53</strong> A2AR/A2BR Dual Inhibitor</td>
<td>Solid tumors</td>
<td>IND-filing 1Q’21</td>
<td>2Q’21</td>
<td></td>
</tr>
</tbody>
</table>

## Non-oncology Programs

<table>
<thead>
<tr>
<th>Program</th>
<th>Indication</th>
<th>IND-enabling</th>
<th>Phase 1a/b</th>
<th>Phases 2/3</th>
</tr>
</thead>
<tbody>
<tr>
<td>A2AR Inhibitors</td>
<td>Parkinson’s</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A2BR Inhibitors</td>
<td>Sickle Cell Disease, Lung Fibrosis, Asthma</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gut-restricted A2BR Inhibitors</td>
<td>Ulcerative Colitis</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Best-in-Class & First-in-Class Adenosine Receptor Antagonists

**TT-10**
A2AR Inhibitor
Highly Potent and Selective Oral Inhibitor of A2AR
Significant Therapeutic Potential as Single Agent and Combination Therapy

**TT-4, TT-3**
A2BR Inhibitor
Highly Potent and Selective Oral Inhibitor of A2BR
Significant Therapeutic Potential as Single Agent and Combination Therapy

**TT-53**
Dual Inhibitor A2AR & A2BR
TT-53 is an equipotent lead compound with low nanomolar/picomolar activity against both A2AR and A2BR
TT-10

A2AR INHIBITOR

Best-In-Class
<table>
<thead>
<tr>
<th><strong>TT-10 A2AR INHIBITOR</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Most potent (sub nanomolar) and highly selective oral A2AR inhibitor</td>
</tr>
<tr>
<td>Long receptor occupancy, even at high extracellular adenosine concentrations; QD dosing</td>
</tr>
<tr>
<td>Efficacy and safety established in preclinical PoC studies in syngeneic breast and colon cancer models</td>
</tr>
<tr>
<td>No safety issues in mouse, rat and dog toxicity studies</td>
</tr>
<tr>
<td>Patents issued in US, Europe, Japan</td>
</tr>
<tr>
<td>IND filing Q4 2020</td>
</tr>
</tbody>
</table>
TT-10 Inhibits A2AR with Picomolar Potency and High Selectivity over other receptor subtypes

<table>
<thead>
<tr>
<th>Receptor</th>
<th>Ki (nm)</th>
<th>Selectivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>A2A</td>
<td>0.065</td>
<td>1</td>
</tr>
<tr>
<td>A1</td>
<td>10,000</td>
<td>&gt;10,000</td>
</tr>
<tr>
<td>A2B</td>
<td>100</td>
<td>&gt;1,400</td>
</tr>
<tr>
<td>A3</td>
<td>10,000</td>
<td>&gt;10,000</td>
</tr>
</tbody>
</table>

Picomolar potency is critical for inhibition of the high-affinity A2AR receptor

TT-10 potency is a key differentiating factor vs. competition – given high concentrations of adenosine in the TME

Superior selectivity against other adenosine receptors predicts better safety profile vs. other agents

Functional antagonism (change in intracellular cAMP induced by agonist) IC50 0.40nM
**TT-10 Profile is Highly Differentiated vs. Competition**

<table>
<thead>
<tr>
<th>Company</th>
<th>Drug</th>
<th>Affinity (Ki nm)</th>
<th>Selectivity against A2AR</th>
</tr>
</thead>
<tbody>
<tr>
<td>TARUS</td>
<td>TT-10</td>
<td>0.68</td>
<td>700 2000 10,000</td>
</tr>
<tr>
<td>Corvus</td>
<td>CPI-444</td>
<td>3.52</td>
<td>54 531 693</td>
</tr>
<tr>
<td>AZN/Heptares</td>
<td>AZD4635</td>
<td>1.58</td>
<td>0.1 na na</td>
</tr>
<tr>
<td>Juno/Redox Tx</td>
<td>Vipadenant</td>
<td>1.3</td>
<td>48 52 773</td>
</tr>
<tr>
<td>Kyowa Hakko</td>
<td>Istradefylline</td>
<td>36</td>
<td>79 50 83</td>
</tr>
</tbody>
</table>

**TT-10 binds A2A receptor with very high affinity and selectivity**
**Prolonged Receptor Occupancy**

**TT-10**

- **Prolonged pharmacodynamic (PD) effect of 10+ hours**
- **Critical differentiator in the presence of high ligand concentration in TME**
- **Predicts once/day (QD) dosing**

**Extended Resident Time vs. Other A2AR Antagonists**

- TT-10 (6nM)
- Preladenant (60nM)
- DMSO
- Tozadenant (200nM)
- MRS-1191 (300nM) (negative control)
Efficacy of TT-10 and TT-4 as Monotherapy was Evaluated in Established Immunotherapy Resistant Murine Models

4T1 Syngeneic Breast Cancer Model

- Highly malignant and poorly immunogenic model which resembles advanced breast cancer in humans
- Refractive to most immune-stimulation therapies
- Highly Metastatic

CT-26 Syngeneic Colon Cancer Model

- Immunotherapy resistant mouse model
- CD73 positive

Efficacy of TT-10 & TT-4 was directly compared with anti-PD-1 antibody
TT-10 Effect on Tumor Volume

Breast Cancer (TNBC)

4T1 Syngeneic Mouse Model

48% Reduction in tumor growth vs. control (p<0.05)

29% Reduction vs. anti-PD-1 antibody (p<0.05)

12 mice/group (female BALB/c)
TT-10: 1mg/kg PO, BID; Anti-PD-1 MAb: 200ug/mouse IP (day 8, 11, 14, 18)
Effect of TT-10 statistically significant vs. control
Effect of TT-10 statistically significant vs. anti-PD-1
TT-10 Significantly Reduces Lung Metastasis

4T1 Syngeneic Breast Cancer Mouse Model

65% Reduction in lung metastasis vs. control
p<0.05

40% Reduction vs. anti-PD-1 antibody
p<0.05
Colon Cancer

CT-26 Syngeneic Colon Cancer Mouse Model

Effect on Tumor Volume

12 mice/group (female BALB/c)
TT-10: 1mg/kg PO, BID; Anti-PD-1 MAb: 200ug/mouse IP (day 8, 11, 14, 18)
Effect of TT-10 statistically significant vs. control

54% Reduction in tumor growth vs. control (p<0.05)
27% Reduction vs. anti-PD-1 antibody
TT-4
A2BR INHIBITOR
<table>
<thead>
<tr>
<th>TT-4</th>
<th>A2BR INHIBITOR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Novel, First-in-Class, highly potent &amp; selective oral A2BR inhibitor</td>
<td>Hypoxia-inducible, promotes metastasis in breast, bladder, colon and other solid tumors</td>
</tr>
<tr>
<td>No safety issues in mouse, rat and dog toxicity studies</td>
<td>Efficacy and safety established in preclinical PoC studies in syngeneic breast and colon cancer models</td>
</tr>
<tr>
<td>Patents issued in US, Europe and Japan</td>
<td>IND filing in Q4 2020</td>
</tr>
</tbody>
</table>
TT-4 Inhibits A2BR with High Potency and Selectivity

Functional antagonism of receptor

<table>
<thead>
<tr>
<th>Receptor</th>
<th>Ki (nm)</th>
<th>Selectivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>A2B</td>
<td>13</td>
<td>1</td>
</tr>
<tr>
<td>A1</td>
<td>300</td>
<td>23</td>
</tr>
<tr>
<td>A2A</td>
<td>1800</td>
<td>138</td>
</tr>
<tr>
<td>A3</td>
<td>60,000</td>
<td>&gt;4000</td>
</tr>
</tbody>
</table>

High selectivity against other adenosine receptors predicts wide safety window

Binding affinity

<table>
<thead>
<tr>
<th>Receptor</th>
<th>Ki (nm)</th>
<th>Selectivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>A2B</td>
<td>9</td>
<td>1</td>
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<tr>
<td>A1</td>
<td>&gt;30,000</td>
<td>&gt;400</td>
</tr>
<tr>
<td>A2A</td>
<td>&gt;10,000</td>
<td>&gt;150</td>
</tr>
<tr>
<td>A3</td>
<td>&gt;30,000</td>
<td>&gt;400</td>
</tr>
</tbody>
</table>
**TT-4 Effect on Tumor Volume**

- 46% Reduction in tumor growth vs. control (p<0.05)
- 26% Reduction vs. anti-PD-1 antibody (p<0.05)

**Breast Cancer**

4T1 Syngeneic Mouse Model

- 12 mice/group (female BALB/c)
- GN-4: 3mg/kg PO, BID; Anti-PD-1 MAb: 200ug/mouse IP (day 8, 11, 14, 18)
- Effect of TT-4 statistically significant vs. control
- Effect of TT-4 statistically significant vs. anti-PD-1
TT-4 Significantly Reduces Lung Metastasis

4T1 Syngeneic Breast Cancer Mouse Model

80% Reduction in lung metastasis vs. control

64% Reduction vs. anti-PD-1 antibody
Novel Formulation

TT-3
GUT RETENTIVE A2BR INHIBITOR
TT-3
First in Class

Novel Target Agent for Gastro-Intestinal Cancers

Gut-restricted distribution of TT-3 offers a unique opportunity to attain superior therapeutic window in GI cancers

• Adenosine 2B receptors are consistently upregulated in colorectal cancer (2/3rd of adenocarcinomas)

• Hypoxia induces A2B expression

• A2BR antagonists inhibit colon cancer cell proliferation

IND READY

No safety issues in animal studies

Patents granted in US, Europe and Japan
A2BR

Over Expression Promotes Colon Carcinoma Growth

A2BR is induced by hypoxia

A2BR is over-expressed in colon adenocarcinoma

Cell proliferation (A570 - 650 nm)

LOVO

A2BR Inhibition Impedes Colon Cancer Growth

Human Pathology 41 (2010)
TT-53

A2AR & A2BR DUAL INHIBITOR

Best-In-Class
TT-53

Dual Inhibitor:

Targeting Both A2AR and A2B

With A Single Molecule

A2AR and A2BR promote tumor resistance via independent mechanisms.

A2AR inhibition potentiates anti-tumor effects by enhancing effector functions of cytotoxic lymphocytes and preventing the recruitment of immunosuppressive cells in the TME.

By contrast, A2BR antagonism affects tumor-intrinsic and host mediated pathways – such as myeloid cells / tumor-associated macrophages (TAMs).
TT-53 Shows Synergistic Inhibition of Adenosine Mediated Immunosuppression

Highly potent dual A2AR / A2BR antagonists with low nanomolar/ picomolar activity identified

Lead compound TT-53 is equipotent against both receptor subtypes

- Suitable ADME and pharmacokinetic profiles
- No liability for hERG, CYP inhibition, or hepatocyte instability
- Preclinical PoC studies in animals to be initiated

TT-53 shows synergistic inhibition of cAMP signaling – the main pathway for adenosine-mediated immunosuppression

Compound series covered by two issued patents
Clinical Indications

TT-10 (A2AR)
Renal
Lung
Prostate

TT-3 (A2BR Gut)
Colorectal
Gastric

TT-4 (A2BR)
TNBC
Bladder

TT-53 (Dual)
Renal, Lung, TNBC,
Bladder, Ovarian, Prostate

Single agent and in combination with ICP’s and Chemo
Robust platform of differentiated immuno-oncology assets with high therapeutic value

TT-10: Best-In-Class A2AR Inhibitor
TT-4: First-In-Class A2BR Inhibitor
TT-3: Unique gut-restricted A2BR inhibitor for GI cancers
TT-53: Dual Inhibitor with Highest Potency and Selectivity
Single Agent Activity Against Immunotherapy Resistant Cancers
Higher Efficacy vs. anti-PD-1 MAb

Together, the company’s assets represent a high value proposition for addressing unmet need with a large market potential
This presentation contains forward-looking statements that involve substantial risks and uncertainties. All statements, other than statements of historical facts, contained in this presentation, including statements regarding our strategy, future operations, future financial position, future revenues, projected costs, prospects, plans and objectives of management, are forward-looking statements within the meaning of the US Private Securities Litigation Reform Act of 1995, as amended. We may not actually achieve the plans, intentions or expectations disclosed in our forward-looking statements, and you should not place undue reliance on our forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward looking statements we make. These forward-looking statements include, among other things, statements about our plans to develop, out-license or commercialize our product candidates, the timing of availability of data, the potential effectiveness of our drug candidates, our ability to translate research into future clinical success, our anticipated milestones, our expectations regarding the sufficiency of our financial resources to fund operations and our ability to enter into third party collaborations. Any forward-looking statement contained in this presentation reflects Tarus Therapeutics’ views as of the date of this presentation with respect to future events and Tarus Therapeutics assumes no obligation to publicly update or revise these forward-looking statements for any reason, or to update the reasons actual results could differ materially from those anticipated in these forward-looking statements, even if new information becomes available in the future, except as otherwise required by applicable law.

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