Forward-Looking Statements

Except for statements of historical fact, the statements in this presentation are forward-looking statements, including, but not limited to, statements regarding the future development of our proprietary Engineered Toxin Body (ETB) technology; statements relating to the potential lifting of the partial clinical hold on our MT-3724 clinical trials; statements relating to the development of the MT-3724, MT-5111, TAK-169, and MT-6402, and our preclinical pipeline; our utilization of a next-generation ETB scaffold that has been designed to reduce or eliminate the propensity for innate immunity, including CLS, and to reduce the propensity for aggregation; our plans to enter the clinic with multiple candidates; our expected receipt of clinical data; our future cash needs; and statements relating to the outcome of our collaborations as they relate to our ETB platform; whether our collaborators will exercise their options and our receipt of future development, regulatory and sales milestones and royalty payments. These statements constitute "forward-looking statements" within the meaning of Section 27A of the Securities Act and Section 21E of the Securities Exchange Act and are usually identified by the use of words such as "anticipates," "believes," "estimates," "expects," "intends," "may," "plans," "projects," "seeks," "should," "will," and variations of such words or similar expressions. These forward-looking statements reflect our current views about our plans, intentions, expectations, strategies and prospects, which are based on the information currently available to us and on assumptions we have made. Although we believe that our plans, intentions, expectations, strategies and prospects as reflected in or suggested by those forward-looking statements are reasonable, we can give no assurance that the plans, intentions, expectations or strategies will be attained or achieved. Furthermore, actual results may differ materially from those described in the forward-looking statements and will be affected by a variety of risks and factors that are beyond our control. These statements involve risks and uncertainties that can cause actual results to differ materially from those in such forward-looking statements. Important factors that may cause actual results to differ materially from the results discussed in the forward-looking statements include risks and uncertainties, including (1) our failure to secure and maintain relationships with collaborators; (2) risks relating to clinical trials and other uncertainties of product candidate development; (3) our ability to successfully resolve the partial clinical hold with regard to MT-3724; (4) risks relating to the commercialization, if any, of our proposed product candidates (such as marketing, regulatory, product liability, supply, competition, and other risks); (5) dependence on the efforts of third parties including our strategic partners; (6) dependence on intellectual property; and (7) risks from global pandemics including COVID-19. Further information regarding these and other risks is included under the heading "Risk Factors" in our filings with the Securities and Exchange Commission available from the SEC's website (www.sec.gov). Existing and prospective investors are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date hereof. These forward looking statements reflect management's current views and we do not undertake to update any of these forward-looking statements to reflect a change in events or circumstances that occur after the date of this presentation except as required by law.
MTEM: Developing Novel Therapeutics With a Unique Platform

**Unique MOA**
Engineered Toxin Bodies (ETBs) have the specificity of an antibody, can induce their own internalization, and are designed to act through a potent and unique mechanism of action: ribosomal destruction.

**Advancing Pipeline**
POC with 1st-Gen ETB demonstrating forced internalization, clinical activity and generally favorable tolerability profile. Two 2nd-Gen ETBs in clinic with improved activity and tolerability. FPI with 3rd-Gen ETB targeting PD-L1 in 1H21.

**Known Targets for Early Signs of Tolerability and Response**
ETBs against validated targets can provide evidence of tolerability and response as early as Phase 1.

**Global Partners**

**Future Opportunities**
ETB platform provides continued pipeline opportunities via partnerships and internal development. Next-Gen ETBs in preclinical development against targets including CTLA-4, SLAMF-7, CD45.

**Strong Cash Position**
Current cash funds operations through 2022\(^1\) without additional business development.

\(^1\)Including the upfront payment from our recently announced collaboration agreement with BMS.
ETBs: Novel Mechanisms of Action in Oncology

ETBs use an antibody domain for targeting genetically fused to a de-immunized SLTA

- ETBs can be made to bind any extracellular target
- ETBs retain the SLTA-mediated:
  - Internalization (even against non-internalizing targets)
  - Routing to the cytosol
  - Enzymatic and irreversible destruction of ribosome

Iterative improvements made to ETB scaffold

- Clinical validation of forced internalization, tolerability and activity with 1st-Gen ETB (MT-3724)
- 2nd-Gen ETBs have been engineered to have:
  - Increased potency
  - Decreased adaptive immunity
  - Decreased innate immunity via reduced TLR4 affinity
  - Reduced propensity for aggregation
- 3rd-Gen ETBs have all the properties of the 2nd-Gen and can specifically alter the immunophenotype of tumor cells via antigen seeding technology (AST)
MT-3724
1st-Gen ETB Targeting CD20 for Lymphomas
MT-3724: 1\textsuperscript{st}-Gen CD20-Targeted ETB

**CD20 Targeted**
Single-chain variable fragment (scFv) with specificity to CD20

**Forced Target Internalization**
First agent to demonstrate efficient internalization against CD20, a non-internalizing receptor

**Wild-Type Payload**
Wild-type Shiga-like toxin A-subunit payload. Retains TLR4 interaction

**Other Properties**
Fusion protein; no linker chemistry. Short serum half-life; irreversible intra-cellular effects.

**Novel Enzymatic Payload**
Primary mechanism of cell-kill is enzymatic ribosome destruction

**MT-3724**
1\textsuperscript{st}-Gen ETB
- Not de-immunized
- Less potent than next-gen ETBs
- Propensity for aggregation
MT-3724: Activity Demonstrated in DLBCL

Phase 1/1b study conducted in heavily pretreated B-cell lymphoma patients

- Median age of 65
- Median of 4 prior NHL therapies; median of 2 prior anti-CD20 Mabs

Deep and prolonged dose-dependent B-cell depletion observed

Favorable tolerability profile

- Maximum tolerated dose (MTD) established at 50 µg/kg
  - Dose cohorts of 5, 10, 20, 50, 75, and 100 µg/kg evaluated
- Dose-limiting toxicities (DLTs) were non-life threatening grade 2/3 events including grade 2 capillary leak syndrome (CLS) which resolved upon cessation of dosing; CLS did not recur upon re-challenge at lower doses in our Phase 1 trial

High serum levels of Rituxan® (RTX) inhibits MT-3724 activity

- 0/6 response rate
- Patients screened out for high RTX in ongoing studies

Patients evaluable for efficacy in phase I (n=25)

- DLBCL or Mixed DLBCL/FL (n=19)
- Low serum RTX levels (n=13)

- 2 Complete Responses (CR)
- 1 Complete Metabolic Response (CMR)
- 2 Partial Responses (PR)
- 3 Stable Disease (SD) (49%, 47% tumor reduction)
- 5 Progressive Disease (PD)

38% Objective Response Rate (ORR)a

60% ORR at MTD: 2 CRs, 1 PR, 2 PD
MT-3724: Update on Partial Clinical Hold

- One subject death occurred on 20-Oct-2020 in the MT-3724 monotherapy study due to grade 5 capillary leak syndrome (CLS)
  - Subject was a seventh line DLBCL patient (ex-US site) who had rapidly progressed through three lines of therapy (including a first-generation CAR-T) in the six months prior to MT-3724 dosing
  - Subject experienced grade 2 CLS after two doses of MT-3724 which had resolved prior to resuming dosing

- First grade 5 CLS event across all MT-3724 studies
  - All prior occurrences of CLS across all MT-3724 studies were grade 2 or below

- Elevated $C_{\text{max}}$ exposure observed in 5 of the last 7 subjects dosed on MT-3724 monotherapy study
  - Higher than expected pharmacokinetic data (beyond pharmacokinetic projections) observed in these 5 subjects
  - All five subjects received drug from the same single lot of MT-3724 drug product
  - $C_{\text{max}}$ exposures observed in these five subjects were higher than has been observed in any other subjects or studies with MT-3724
  - Elevated $C_{\text{max}}$ exposures have not been observed with 2nd generation ETB programs

- Investigation ongoing and MT-3724 studies put on partial clinical hold by the FDA on Nov 4, 2020
  - No new enrollment; patients on drug and benefiting will continue to receive MT-3724; lot in question held until investigation is complete

- Other MTEM studies continue and are not affected
  - TAK169, MT-5111 and planned MT-6402
2nd-Generation ETBs
Increased Potency; Better Tolerability; Reduced Aggregation
2\textsuperscript{nd}-gen ETBs Designed to Have Improved Potency and Reduced Toxicity

- **First-gen ETB**
  - Wildtype SLTA payload
  - NHP Tox: HNSTD < 150 ug/kg
  - In-vitro potency (IC\textsubscript{50}): ~sub-nanomolar

- **Next-gen ETBs**
  - Proprietary De-immunized SLTA payload
  - NHP Tox: HNSTD > 500 ug/kg
  - In-vitro potency (IC\textsubscript{50}): ~pico to sub-picomolar

- MT-3724 utilizes wild-type SLTA payload with intact innate receptor interaction (TLR4)
- Suboptimal structure orientation
- ETBs utilize next-gen SLTA payload de-immunized to reduce adaptive and innate immunogenicity
- ETB structural modifications (orientation) greatly improve potency (more efficient intracellular routing)
2nd-gen ETBs Designed to Have Reduced Propensity for Aggregation

**MT-3724 (1st-Gen ETB)**

1st-gen ETB scaffold design exhibits propensity to form higher order aggregates via covalent and non-covalent interactions

- 15-mer linker in scFv can create dimers and monomers
- Free cysteine on SLTA scaffold used to create dimers via oxidation step during purification

**2nd-Gen ETBs**

2nd-Gen ETB scaffold designs prevents the creation of multiple species and covalent aggregation

- 25-mer linker in scFv used to force monomers (MT-5111)
- 5-mer linker used to create diabodies (TAK-169 and MT-6402)
- No free cysteine in Next-Gen SLTA scaffold
- MT-6402, 3rd-gen ETB, utilizes immunodominant 9-mer CMV peptide (hydrophobic) fused to C-terminus for Antigen Seeding
Purity (SE-UPLC): 1\textsuperscript{st}-gen ETB (MT-3724) vs 2\textsuperscript{nd}-gen ETB (MT-5111)

MT-3724 (1\textsuperscript{st} gen) scaffold exhibits propensity to form higher order aggregates via covalent and non-covalent interactions

MT-5111 (2\textsuperscript{nd}-gen ETB)

2nd-gen scaffold prevents the creation of multiple species and covalent aggregation

Lot AG3545
Covalent dimer: 75%
Non-covalent dimer: 10%
HMW: 12%
LMW: 3%

Extended HMW shoulder

Monomer: 97%
HMW: 2%
LMW: 1%
2nd-Generation ETBs
MT-5111 / HER2
MT-5111: A 2nd-Generation HER-Targeted ETB

**HER2 TARGETED**
Single-chain variable fragment (scFv) with specificity to HER2. Binds a distinct epitope from trastuzumab/pertuzumab

**SMALL SIZE FOR BETTER PENETRATION**
55 kDa versus ~145 kDa for Mabs/ADCs

**DEIMMUNIZED PAYLOAD**
De-immunized SLTA payload for reduced innate and adaptive response. Reduced TLR4 interaction to minimize innate triggering (CLS)

**OTHER PROPERTIES**
Fusion protein; no linker chemistry. Short serum half-life; irreversible intra-cellular effects

**NOVEL ENZYMATIC PAYLOAD**
Primary mechanism of cell-kill - enzymatic ribosome destruction. pM potency against HER2+ cells

**MT-5111: 2nd-Generation ETB**
HER2 binding antibody fragment
DI SLTA Payload
MT-5111: Clinical Development

- **Dose Escalation**
  - Cohorts of 0.5, 1.0, 2.0, 3.0, 4.5, and 6.75 mcg/kg
    - (predicted tx range ≥5 mcg/kg)

  - **Continued Dose Escalation**
    - Cohorts of 10.0 mcg/kg and above in HER2+ non-MBC pts

  - **Expansion in MBC**
    - Initial dosing at 10 mcg/kg with continued dose escalation

  - **Expansion in HER2+ non-MBC tumor types**
    - Gastric and other HER2+ tumors

  - **1H21**

  - **When MTD reached or rec. Ph. 2 dose determined**

- **Tumor types enrolled:**
  - MBC (n=6; all ≤3 μg/kg); biliary (n=6); pancreatic (n=2); one colon and one GE junction
  - 1 pt w/ MBC (1 μg/kg) w/ unmeasurable disease by RECIST remained on tx for 10 cycles w/ SD, had 3 sub-cm lesions that disappeared at cycle 8
  - No DLTs or cardiotoxicity have been observed to date
  - 6.75 mcg/kg cohort on-going

2021 Corporate Presentation
2nd-Generation ETBs

TAK-169 / CD38
**TAK-169: A 2nd-Generation CD38-Targeted ETB**

**CD38 Targeted**
Single-chain variable fragment (scFv) with specificity to CD38. Binds in the presence of daratumumab.

**Forced Receptor Internalization**
Efficient internalization against CD38, a poorly-internalizing receptor.

**Deimmunized Payload**
De-immunized SLTA payload for reduced innate and adaptive response. Reduced TLR4 interaction to minimize innate triggering (CLS).

**Novel Enzymatic Payload**
Primary mechanism of cell-kill - enzymatic ribosome destruction. pM potency against CD38+ cells.

**Other Properties**
Fusion protein; no linker chemistry. Short serum half-life; irreversible intra-cellular effects.

**TAK-169: 2nd-Generation ETB**
CD38 binding antibody fragment
DI SLTA Payload
# TAK-169: 2nd-Gen ETB Targeting CD38

<table>
<thead>
<tr>
<th>CD38-targeting Agents</th>
<th>CD38 is a poorly-internalizing receptor central to disease in multiple myeloma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mab: Darzalex</td>
<td>TAK-169 efficiently internalizes and destroys low- or high-expressing CD38</td>
</tr>
<tr>
<td>Engineered Toxin Body: TAK-169</td>
<td>TAK-169 activity is retained in the presence of daratumumab in preclinical models</td>
</tr>
<tr>
<td>MOA</td>
<td>TAK-169 active in patient samples (including dara-refractory)</td>
</tr>
<tr>
<td>Indirect CDC cell kill</td>
<td>TAK-169 has shown activity in xenograph models when dosed weekly or bi-weekly</td>
</tr>
<tr>
<td>Direct cell kill (enzymatic ribosome inactivation)</td>
<td>Reduced ADA and innate response (de-immunized STLA scaffold)</td>
</tr>
<tr>
<td>CD38 target interaction</td>
<td>HNSTD of 750 mcg/kg in NHPs (150 mcg/kg for MT-3724)</td>
</tr>
<tr>
<td>Binding</td>
<td>Phase I in rel/ref myeloma patients with weekly dosing started at 50 mcg/kg</td>
</tr>
<tr>
<td>Binding and internalization</td>
<td>CD55/59 upregulation in failures, inhibiting immune response</td>
</tr>
<tr>
<td>Mechanism of resistance</td>
<td>Not applicable</td>
</tr>
</tbody>
</table>

**MOA**
- Indirect CDC cell kill
- Direct cell kill (enzymatic ribosome inactivation)
3rd-Gen ETBs / Novel Approach to IO
2nd-Gen Scaffold + antigen seeding
Direct cell-kill of PD-L1+ tumor and immune cells and alteration of the tumor immunophenotype

- ETBs localize to ER/cytosol to “seed” tumors with foreign non-self antigens

- Antigen cleaved intracellularly and presented on cell surface in context with MHC-I

- Delivery of pp65 CMV antigen
  - Mediate native CMV-specific T cell response to tumor
  - Large existing population infected with CMV
  - CMV-specific T-cells undergo “memory inflation” in response to persistent reactivation of CMV – less prone to exhaustion
  - Large reservoirs of CMV-specific T-cells with significant proportion specific to pp65 CMV epitope

- Fundamental alteration of immunophenotype on tumor with foreign viral antigens to redirect T-cell response

- FPI expected in 1H21

MT-6402: PD-L1 Targeting 3rd-gen ETB

- CMV pp65 peptide antigen
- scFv targeting PD-L1
- De-immunized SLTA
MT-6402: Preclinical Potent Activity Against PD-L1+ Tumor Cells

Potent effect on PD-L1+ tumor cells: Direct Cell-kill against PD-L1+ tumors through two diverse MOAs

• Ribosomal destruction is independent of tumor microenvironment
• Novel, potent MOA not previously used against PD-L1+ tumor types

Unprecedented alteration of tumor immunophenotype
Strong evidence that CMV-specific T-cells are present in tumor microenvironments (Rosato et al, Nature Comm 2019)

MOA1 (ribosomal destruction)

MOA1 + MOA2 (AST)
MT-6402: Potent Activity Against PD-L1+ Immune Cells in the TME

Potent effect on PD-L1+ immune cells in NHP model

PD effects observed in NHP model with MT-6402 have not been seen with checkpoint antibodies

MT-6402 depletion of immune cells and Lymphocyte activation is associated with immune checkpoint inhibitor inflammatory signature

(A) MT-6402 or the SLTA-inactive ETB control were dosed weekly for 4 weeks in non-human primates. (B,C) Immune cell subsets were evaluated from circulation by flow cytometry. (D) Serum cytokine responses were evaluated across two independent studies in NHP – data is displayed as percent of responder for study 1, n=2 NHP and study 2 n=8 NHP (MT-6402) and n=5 (inactive variant). Data reflects induction of cytokines any time after dose 3 in the studies.

MT-6402 is designed to deplete PD-L1 positive IC and TC in the TME. Targeting of IC in patients has shown clinical benefit (1,2) and combination ICI treatment can lead to immune activation and immune related adverse events (irAEs) observed predominantly with combination immune checkpoint inhibitors in clinical settings and are associated with beneficial therapeutic response (3,4).

1,2 – myocarditis and dermatitis are common immune-related adverse events (irAEs) observed predominantly with combination immune checkpoint inhibitors in clinical settings and are associated with beneficial therapeutic response (3,4).
# ETBs and IO: PD-L1 ETB Moving to Clinic; Potential New IO Targets in the Works

<table>
<thead>
<tr>
<th>Potent effect on PD-L1+ tumor cells and immune cells</th>
<th>Exploration of additional IO targets where ETB approach may provide substantial differentiation</th>
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</thead>
<tbody>
<tr>
<td>- Direct cell-kill on tumor cells through ribosomal destruction (MOA1) independent of tumor microenvironment</td>
<td>- CTLA4 lead development work underway; IND filing expected in 2021</td>
</tr>
<tr>
<td>- Novel alteration of cancer cell immunophenotype for pre-existing, synaptic T-cell recognition of tumor (MOA2)</td>
<td>- Potential safety and efficacy benefits around direct cell-kill of CTLA4+ T cells vs blocking</td>
</tr>
<tr>
<td>- Early in vivo and in vitro data suggest potent activity on PD-L1 immune cells and activation of immune system</td>
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Engineered Toxin Bodies
A Robust Pipeline with Clinical Data in 2021
## Robust Clinical Pipeline Supporting Value of Drug Candidates and Platform

<table>
<thead>
<tr>
<th>Program (Target)</th>
<th>Indication/Phase</th>
<th>1Q21</th>
<th>2Q21</th>
<th>3Q21</th>
<th>4Q21</th>
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<tbody>
<tr>
<td>MT-3724 (CD20)</td>
<td>NHL/Ph. 2</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td><strong>Potential resolution of</strong></td>
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<td></td>
<td></td>
<td><strong>partial clinical hold</strong></td>
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<tr>
<td>MT-5111 (HER2)</td>
<td>Solid tumors/Ph. 1</td>
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<tr>
<td></td>
<td></td>
<td><strong>Phase 1 dose escalation data update</strong></td>
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<td><strong>Initiation of breast cancer exp. cohort</strong></td>
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<tr>
<td>MT-6402 (PD-L1 + AST)</td>
<td>Solid tumors/Ph. 1 in 2Q</td>
<td></td>
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</tr>
<tr>
<td>TAK-169 (CD38)</td>
<td>Multiple myeloma/ Ph. 1</td>
<td></td>
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<tr>
<td>Pipeline (CTLA-4, SLAMF-7, CD45)</td>
<td>Various/Preclinical</td>
<td></td>
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<tr>
<td>Partnerships + New Bus Dev</td>
<td>Takeda preclin multi-target (TBD)</td>
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<td></td>
<td>Vertex preclin. multi-target (Myeloablation)</td>
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</tbody>
</table>

- **1Q21**: Potential resolution of partial clinical hold
- **2Q21**: Decision regarding MT-3724 Ph. 2 studies vs. acceleration of next-gen CD20 ETB
- **3Q21**: Completion of dose esc. initiation of gastric exp. cohort
- **4Q21**: Potential interim dose exp. data

**Notes:**
- Potential interim dose exp. data
- Read on platform in solid tumor
- Potential interim dose esc. data
- Preclinical data presentations
- CTLA-4 ETB IND filing
Bristol Myers Squibb Collaboration

On 2/11/21, MTEM announced a worldwide strategic research collaboration with Bristol Myers Squibb to discover and develop multiple novel therapies designed for specific oncology targets, utilizing MTEM’s next generation ETB platform.

MTEM will conduct research activities for the discovery of next generation ETBs for multiple targets, of which the first target has been selected by Bristol Myers Squibb.

Bristol Myers Squibb will have the option to obtain an exclusive worldwide license to develop and commercialize ETBs directed to each selected target. Following the exercise of the option, Bristol Myers Squibb would be solely responsible for developing and commercializing the licensed ETBs.

Collaboration Economics

<table>
<thead>
<tr>
<th>Payment Type</th>
<th>Amount</th>
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</thead>
<tbody>
<tr>
<td>Upfront payment</td>
<td>$70 million</td>
</tr>
<tr>
<td>Near term and development and regulatory milestone payments</td>
<td>Up to $875 million</td>
</tr>
<tr>
<td>Sales-based milestone payments</td>
<td>Up to $450 million</td>
</tr>
<tr>
<td>Tiered royalties</td>
<td>Ranging from mid-single digits up to mid-teens (as percentages of calendar year net sales, subject to certain reductions)</td>
</tr>
</tbody>
</table>
## Summary of MTEM Collaborations

<table>
<thead>
<tr>
<th></th>
<th>BMS</th>
<th>Vertex</th>
<th>Takeda</th>
<th>Takeda</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Year</strong></td>
<td>2021</td>
<td>2019</td>
<td>2018</td>
<td>2017</td>
</tr>
<tr>
<td><strong>Scope</strong></td>
<td>Multi-target</td>
<td>Multi-target</td>
<td>CD38</td>
<td>Multi-target</td>
</tr>
<tr>
<td><strong>Upfront cash (M)</strong></td>
<td>$70</td>
<td>$23</td>
<td>$30</td>
<td>$5</td>
</tr>
<tr>
<td><strong>Equity component (M)</strong></td>
<td>$-</td>
<td>$15</td>
<td>$-</td>
<td>$20</td>
</tr>
<tr>
<td><strong>Option/milestone payment opportunity (M)</strong></td>
<td>$1,325</td>
<td>$522</td>
<td>$633</td>
<td>$572</td>
</tr>
<tr>
<td><strong>Royalties (%)</strong></td>
<td>Mid-single digit to mid-teens</td>
<td>Mid-single digit</td>
<td>Low double-digits to low-twenties</td>
<td>Mid-single to low double-digits</td>
</tr>
</tbody>
</table>