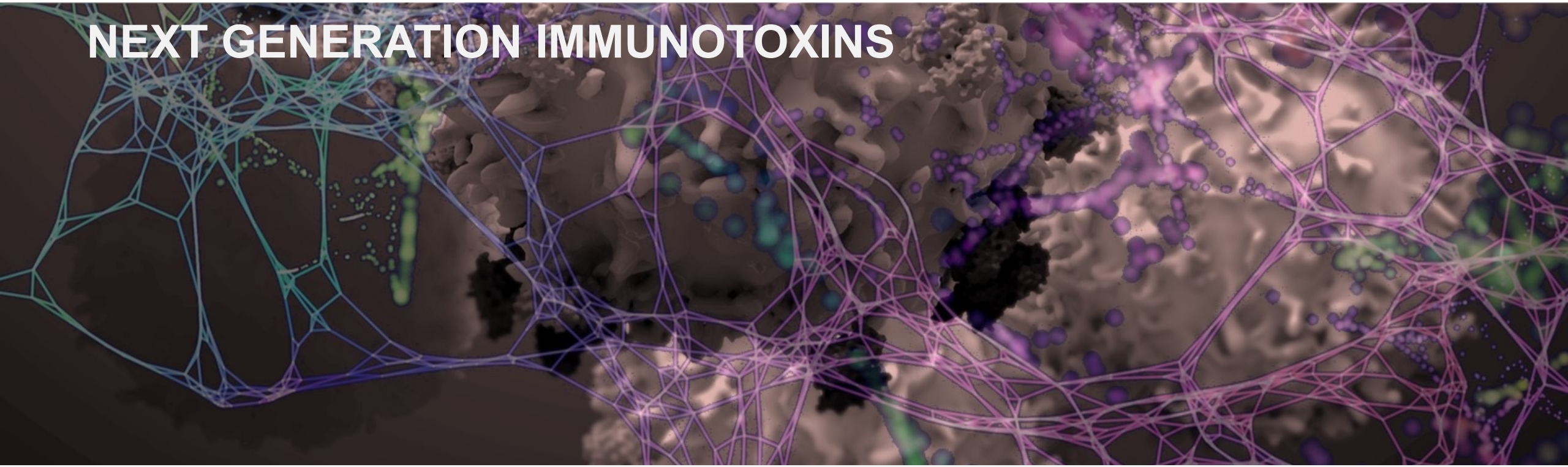
MOLECULAR TEMPLATES

MOLECULAR TEMPLATES

NEXT GENERATION IMMUNOTOXINS



Corporate Presentation

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 development of MT-6402, MT-5111, MT-0169, and MT-8421 and our next generation ETBs and preclinical pipeline; statements regarding the safety or potential efficacy of our drug or biologic candidates, including the anticipated benefits of our next-generation ETBs; our belief that our proprietary ETB technology provides for a differentiated mechanism of action that may address some of the limitations associated with currently available cancer therapeutics; statements regarding expected demand and opportunities for certain targets; expected program milestones; the timing, progress and results of pre-clinical studies and clinical trials for our drug or biologic candidates or any future candidates; the timing or likelihood of regulatory filings, including expected timing for submission and approval of various IND applications; the expected participated and presentation at upcoming conferences; our expected receipt of clinical data; the expected timing for providing updates on our pipeline, including MT-6402, MT-5111, MT-0169 and MT-8421, and our earlier stage pipeline of ETBs; our future cash needs; the length of time for which our cash resources are expected to be sufficient; and statements relating to the outcome of our collaborations as they relate to our ETB platform. 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 drug or biologic candidate development; (3) risks relating to the commercialization, if any, of our proposed drug or biologic candidates (such as marketing, regulatory, product liability, supply, competition, and other risks); (4) dependence on the efforts of third parties including our strategic partners; (5) dependence on intellectual property; and (6) 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.

Developing Novel Oncology Therapies with Differentiated Technology Platform

Unique MOA and I/O approach

- Unique biological MOA of: (i) forced cellular internalization once bound to target and (ii) potent direct cell kill via enzymatic ribosome destruction
- Delivery of viral antigens to redirect resident T-cell response to tumor (Antigen Seeding)
- Emerging data supporting innovative immuno-oncology approach to directly modify immunosuppressive tumor microenvironment

Robust Preclinical Pipeline and Key Partnerships

- TROP-2, TIGIT, SLAMF-7
- Collaboration with pharma partner to discover and develop products containing ETBs directed to multiple targets

Key 2022 Program Milestones

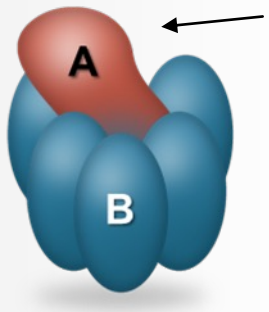
- MT-6402 novel I/O MOA Phase 1 data
- MT-5111 initial Phase 1 breast cancer expansion
- MT-0169 initial Phase 1 myeloma data
- CTLA-4 program IND filing

Strong Balance Sheet

- Operations funded to the end of 2023

The Engineered Toxin Body (ETB) Platform:
Targeted Biologics with Novel MOAs

Wild-type Shiga-Like Toxin (SLT) Biology



Catalytical active A-subunit (SLT-A)

CD77 binding B-subunit

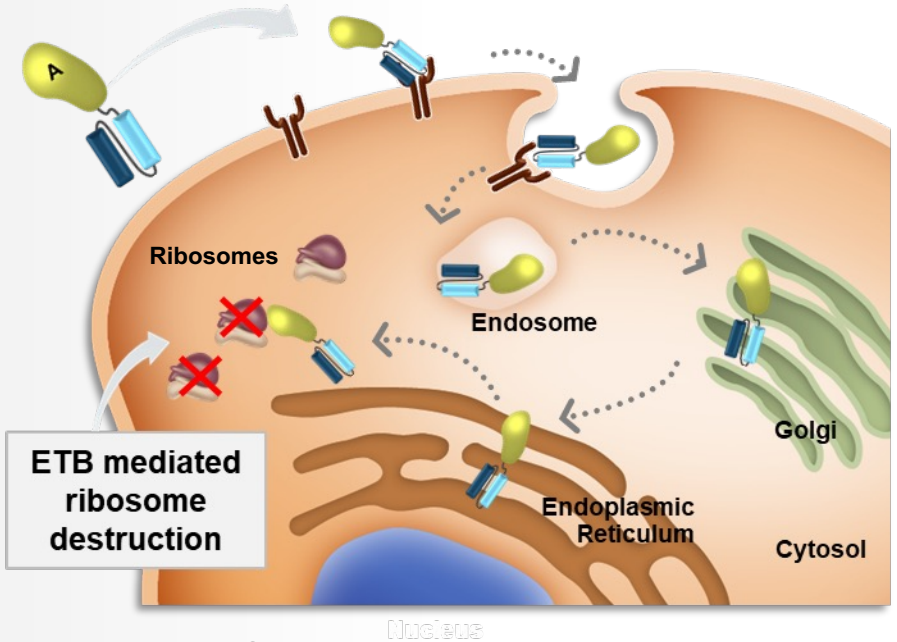
- Binds CD77 (non-internalizing GSL) and mediates its own internalization
- A-subunit mediates translocation to cytosol an enzymatically destroys ribosome

Engineered Toxin Body (ETB) Platform

Proprietarily de-immunized SLTA

Targeting via Antibody Domain

ETBs Retain Intrinsic Biology of SLT-A Subunit

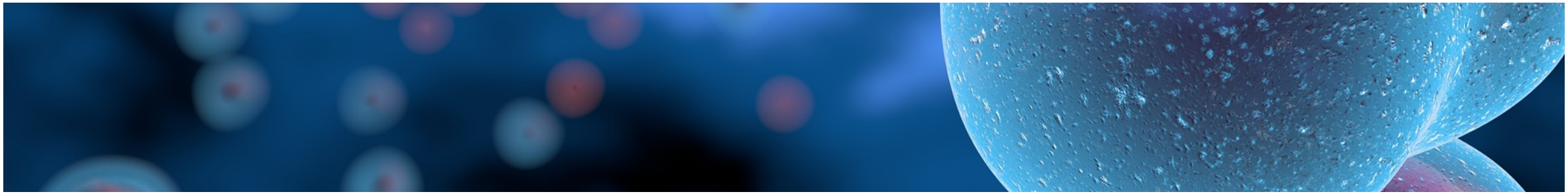


- ETBs specifically bind target and induce their own internalization through SLT-A payload
- Endosomal escape and routing to cytosol
- Potent enzymatic destruction of ribosomes

MOLECULAR TEMPLATES

I/O APPROACH: MT-6402

DIRECT PD-L1 TARGETED CELL-KILL AND ANTIGEN SEEDING



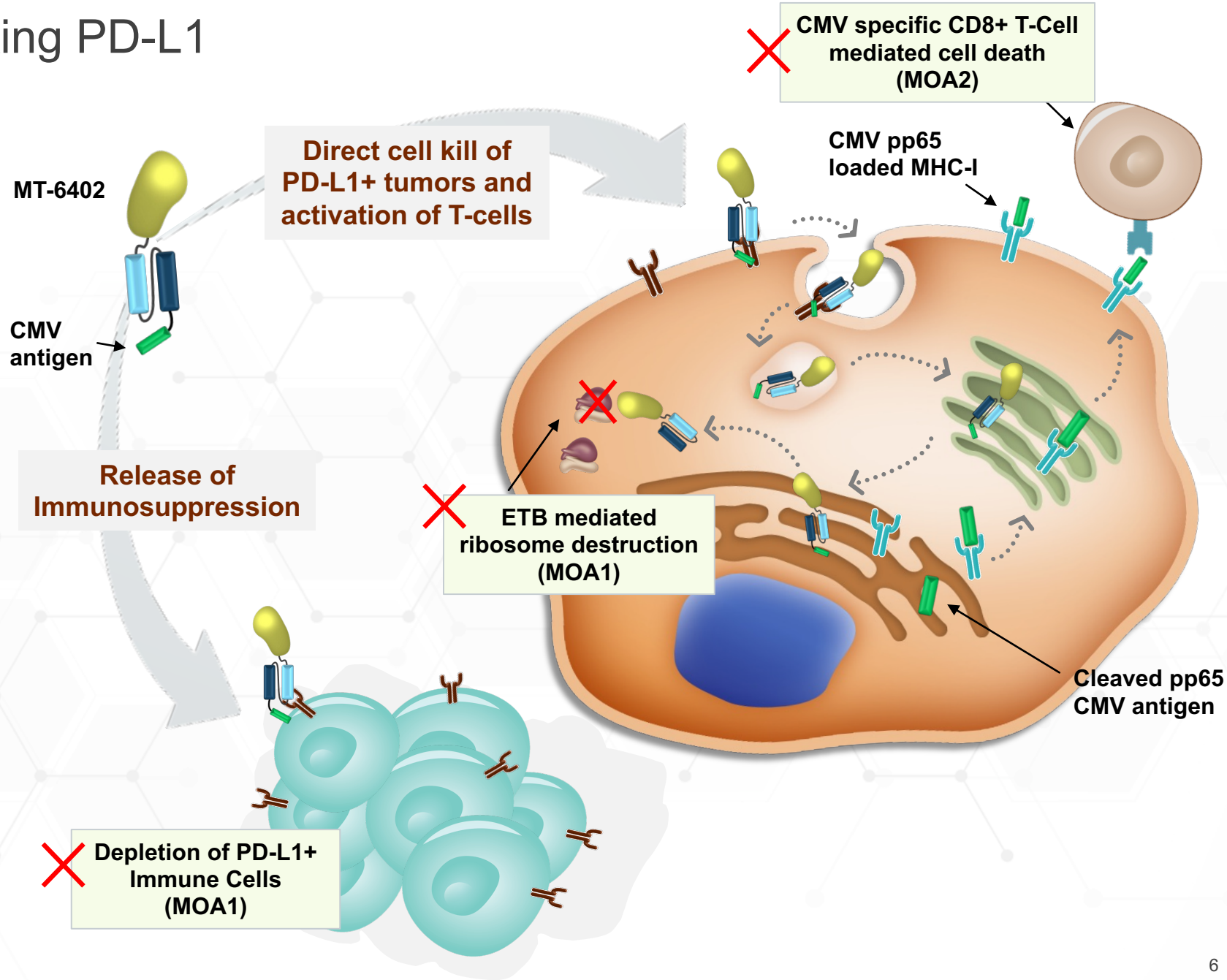
MT-6402: Dual MOA targeting PD-L1

Destroys PD-L1 Expressing Cells – MOA1

- Potent direct cell-kill (pM) against PD-L1+ tumor cells
- Potent direct cell-kill (pM) against PD-L1+ immune cells to activate the immune system

Antigen Seeding Technology (AST) – MOA2

- Delivers pp65 CMV antigen for cell surface expression in context with MHC-I
- Mediates native CMV-specific T-cell response to tumor
- For CMV infected, large reservoirs of CMV-specific T-cells



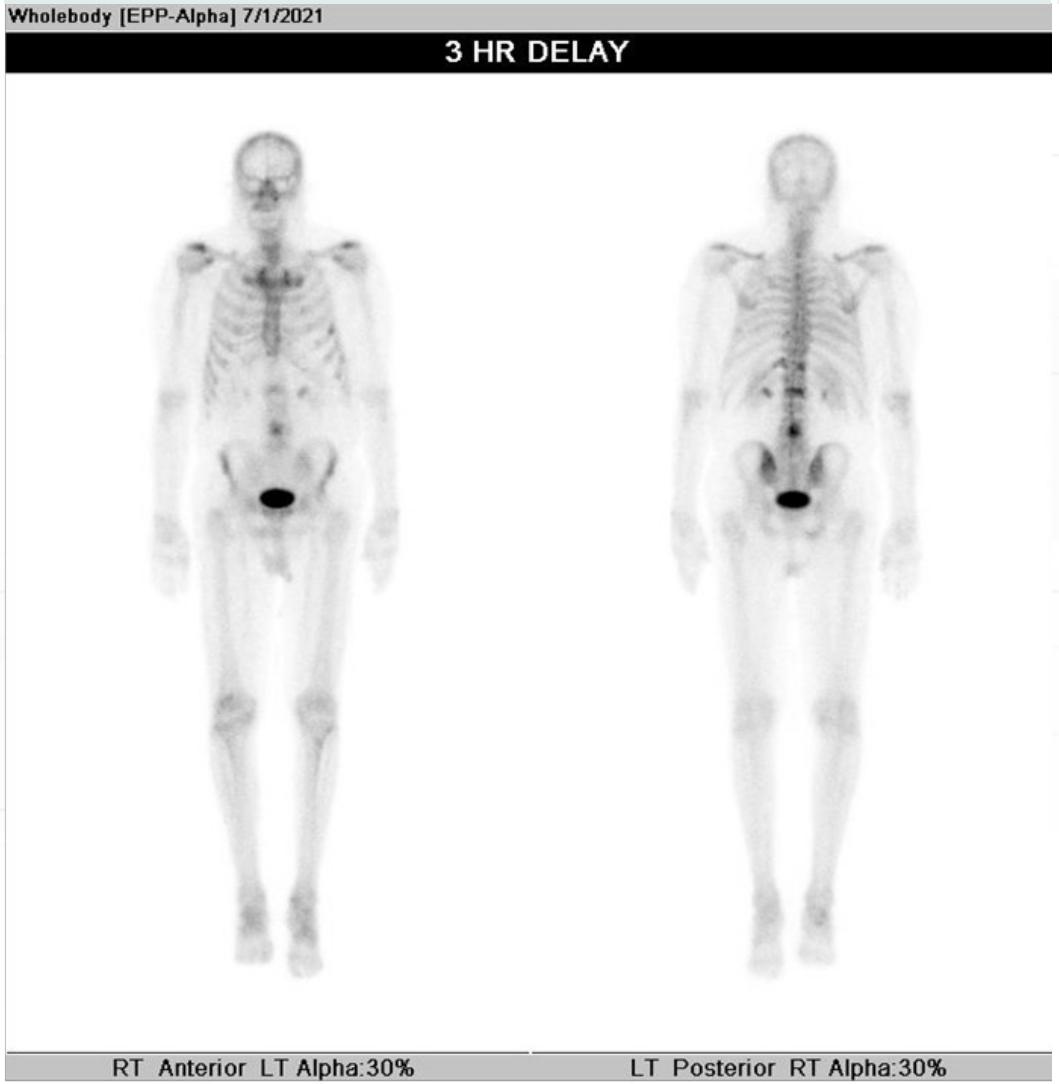
MT-6402 Phase I Study – Clinical Overview

- **Cohort 1 (16 mcg/kg) completed with 6 patients treated**
  - No DLTs; first two patients experienced grade 2 immune-related adverse events (CRS and IRR)
  - First patient (1008-001 NSCLC with bone metastases) had evidence of tumor reduction
    - 3 of the patient's 4 bone lesions resolved and remaining lesion showed decrease uptake
    - At C8, patient had disease progression with increased uptake in bone lesions on bone scan
- **Cohort 2 (24 mcg/kg) completed with 6 patients treated**
  - The first patient developed extensive body rash beginning on C1D4, reaching Grade 2 on C1D6
    - Rash responded quickly to oral steroids; re-challenged at the same dose
  - No other clinically relevant TEAEs
- **Immune-related AEs, although rare with PD(L)1 antibodies, correlate with better outcomes**
- **Cohort 3 (32 mcg/kg) enrolling**
- **To date, 3 patients who are CMV+/HLA-A2+ have been treated (AST engaged patients)**

Subject 1008-001 (16 mcg/kg – AST Engaged): Resolution/Decrease of Osseous Lesions

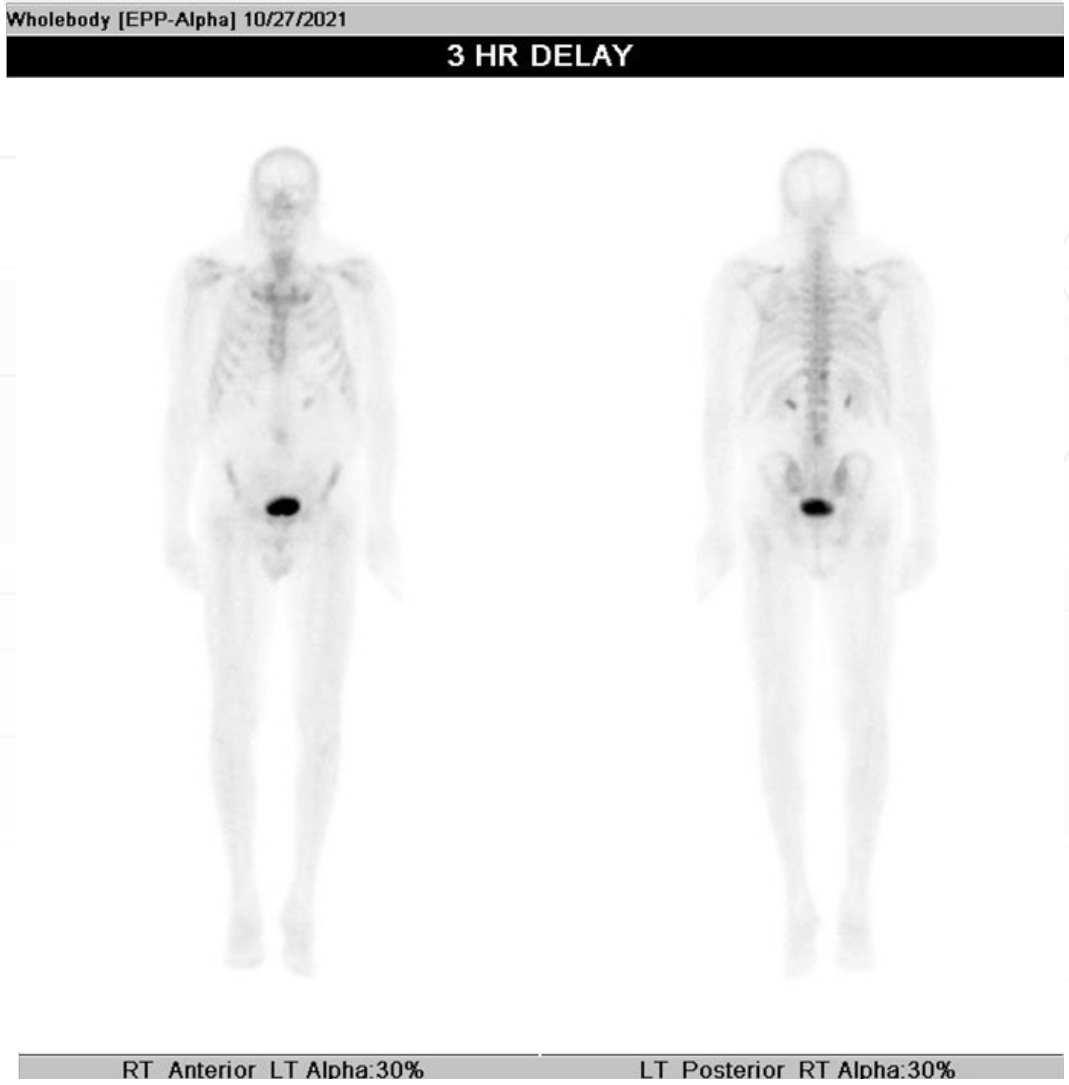
Jul 01, 2021

Metastatic uptake: L1, T11, left 11th rib, left 5th rib, right ischial tuberosity



Oct 27, 2021

Interval decrease of T11, L1 has mostly resolved, left 5th rib resolved, left 11th rib resolved



Cytokine and Phenotypic Changes Observed in All Patients and Differentially in AST-Engaged Patients

Similar effects not observed with PDL-1 monoclonal antibodies

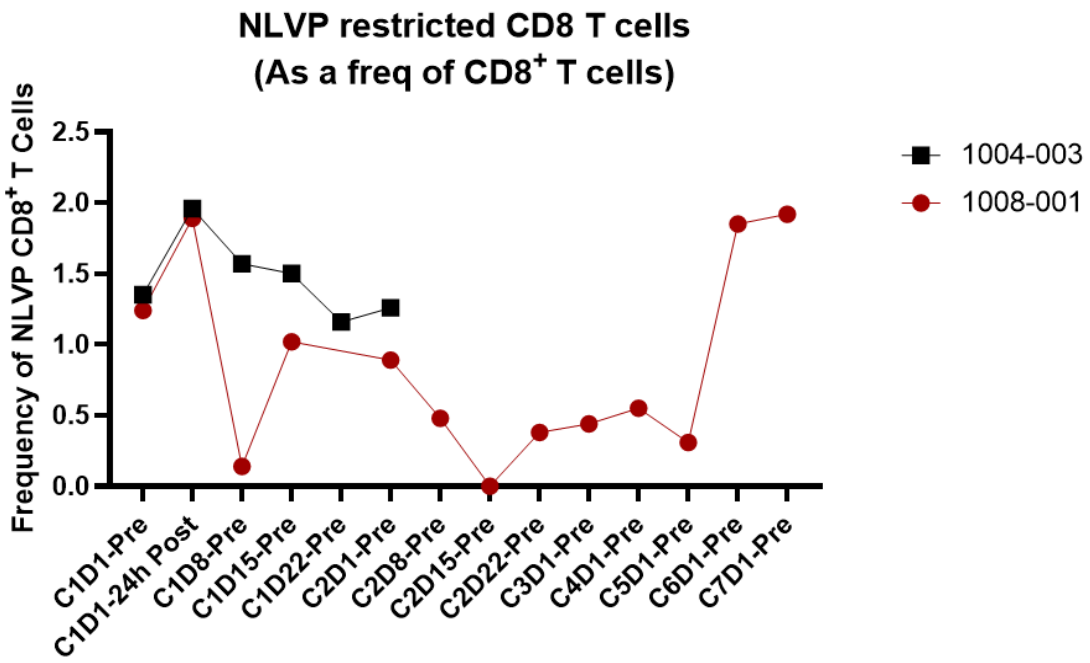
	Cytokines*	PBMC Changes	
All Patients (Irrespective of HLA or CMV status)	▲ CCL2/MCP-1	▼ Monocytes ▼ MDSC	**Movement of myeloid cells to tissues and/or direct cell kill**
	▲ IL-2	▲ Tbet+ CD8 T cells ▲ NK cells	**Global T cell activation due to checkpoint break**
AST-Engaged Only (HLA:A2, CMV+)	▲ TNFa ▲ IL-10	▲ Peripheral CD8 T cells ▲ Peripheral NLVP T cells (initial expansion)	**Checkpoint break plus AST driven activation of CMV specific T cells**
	▲ IP-10/CXCL10	▼ Peripheral NLVP T cells (after first dose) ▼ Peripheral dendritic cells	**Migration of DCs and CMV specific T cells from periphery**

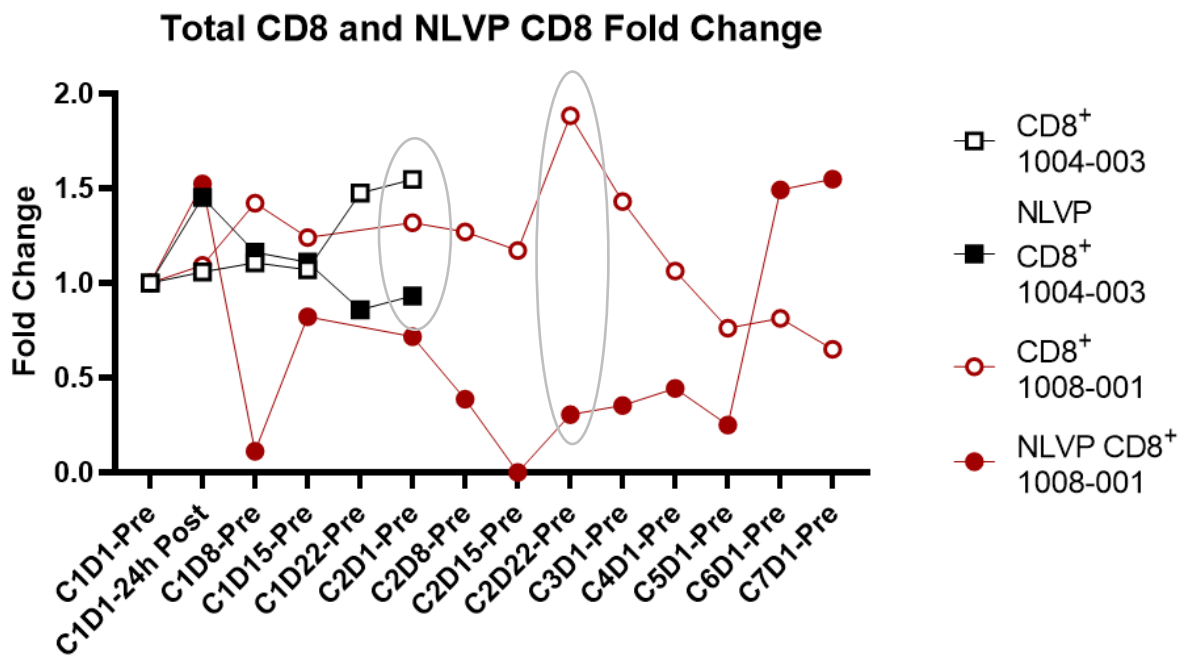
** Listed cytokines are not comprehensive; panel contains 24 total cytokines, some of which were below limit of detection, some were unchanged, and others did not show trends*

MT-6402 Summary - Implications of Observed PD Effects

- **Reduction in peripheral PD-L1+ monocytes, MDSCs, and Tregs is a unique effect in the immune checkpoint inhibitor landscape**
  - High levels of immune cells associated with poor likelihood of response to PD-1 inhibitors
  - PD effects observed independent of HLA status; similar PD effects not observed with other PD-L1 agents
  - Possibility of alterations to tumor microenvironment conducive to re-awakening of anti-tumor immune responses
- **Extravasation of CMV-specific T cells from the periphery indicates evidence of clinical proof of principle for Antigen Seeding Technology (AST)**
  - Possibility of re-direction of resident cytotoxic CMV-T cells toward tumor tissues
  - Combination of loss of immunosuppressive cells (“releasing the brakes”) with re-direction of CMV-T cells (“putting on the gas”) could provide additivity in anti-tumor responses
  - Pre-treatment and on-treatment biopsies are planned for dose expansion cohorts
- **Early but compelling rationale for combination of MT-6402 with a PD-1 inhibitor**
  - Can MT-6402 remove a mechanism of resistance to PD-1 checkpoint therapy?

CMV-Specific T-Cells Leave the Periphery in AST-Engaged Subjects



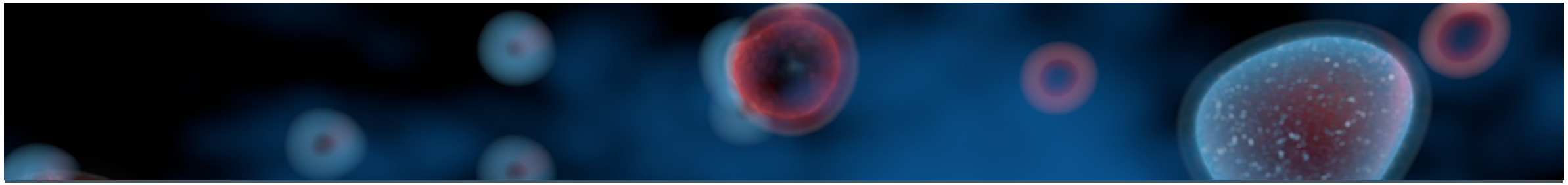


- **In HLA:A2 and CMV+ patients (AST-engaged), 1008-001 and 1004-003, initial (24hrs) increase of CMV specific T cells (peripheral presentation of antigen)**
- **Initial expansion followed by reduction of CMV specific CD8 T cells (likely extravasation to tumor or secondary tissues)**
- **CMV-T cells are stable in all non AST-engaged subjects (non-HLA:A2, CMV+/-)**
- **General CD8 T cells (non-CMV specific) increase, indicating a likely general T cell expansion due to checkpoint break**

MOLECULAR TEMPLATES

ANTI-CTLA-4 ETB

DIFFERENTIATED I/O APPROACH TARGETING CTLA-4

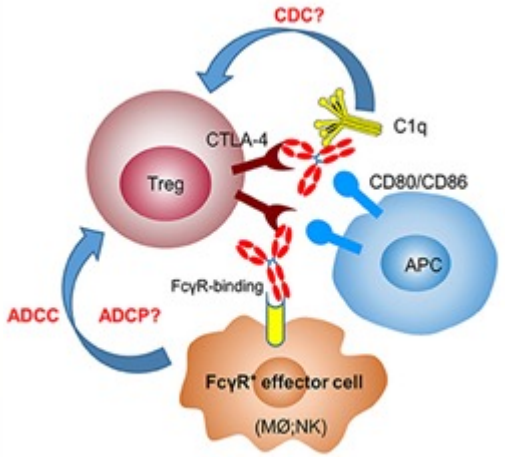


CTLA-4 targeting ETB IND filing expected in 2H22

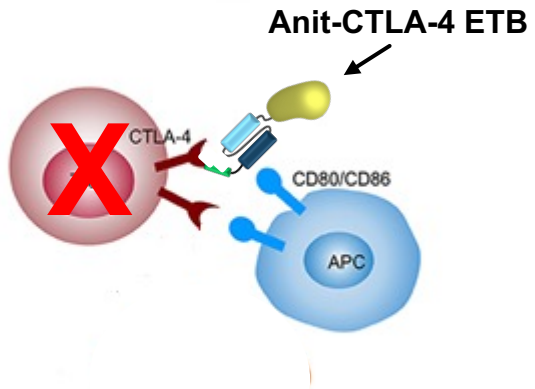
Need to clear CTLA-4+ Tregs from TME to allow for immune surveillance of tumor

- Tregs in the TME express high levels of CTL-4 and can drive immunosuppressive tumor microenvironment
- CTLA-4 targeting ETB has a potent mechanism of CTLA-4 targeted cell-kill that may not be subject to immunosuppression
- ETB approach is designed to avoid CD8 T-cell activation to prevent autoimmune toxicity

Existing antibody therapies to CTLA-4 may not mediate efficient cell kill given general immunosuppression in the TME



Mab mediated clearance dependent on effector cell function which is inhibited by TME



ETB CTLA-4 targeted cell-kill not dependent on effector cell or TME

ETBs Represent a Differentiated Modality to Target CTLA-4

MT-8421: CTLA-4 Targeting Agent Designed to Overcome Mab Limitations

Potently destroys CTLA-4+ T-regs via enzymatic ribosome destruction

Murine models and clinical data suggest destruction of CTLA-4 Tregs and not CTLA-4 blockade is necessary for CTLA-4 mediated efficacy

MOA independent of TME

Inability of antibodies to destroy Tregs in patient tumors is likely due to inhibition of effector cells function in TME; effector-cell independent kill mechanism is necessary

Preferential activity on TME resident T-regs

Potency of MT-8421 cell-kill correlates with CTLA-4 expression levels allowing for preferential cell-kill of Tregs in TME; lack of blockade effect may reduce peripheral autoimmune toxicity

MT-8421 IND Timeline

MT-8421: CTLA-4 Targeting Agent Designed to Overcome Mab Limitations

Potently destroys CTLA-4+ T-regs via enzymatic ribosome destruction

MOA independent of TME

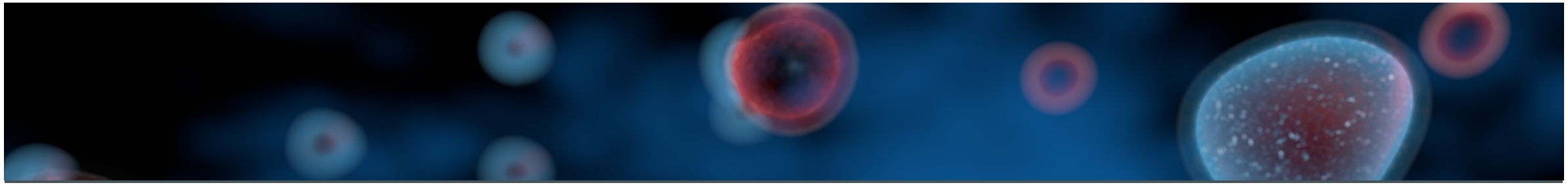
Preferential activity on TME resident T-regs

- **IND filing estimated second half of 2022**
- **Phase 1 FPD estimated 2023**

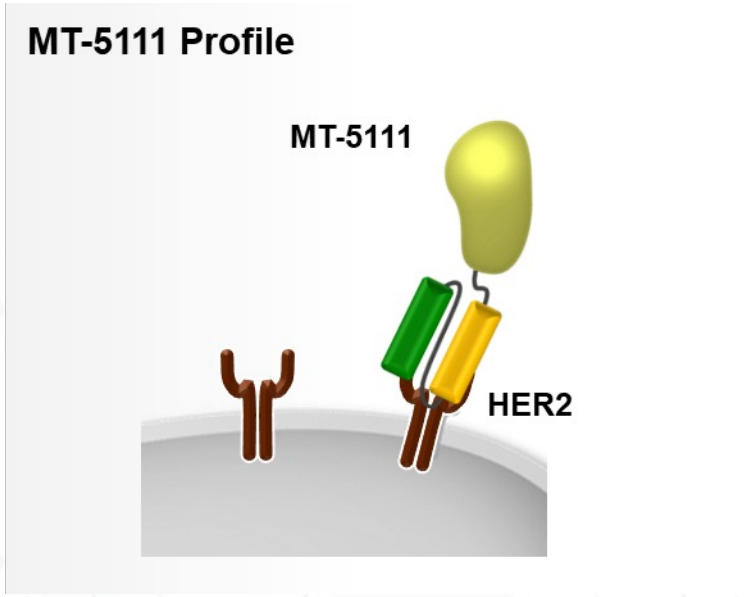
Potency of MT-8421 cell-kill correlates with CTLA-4 expression levels allowing for preferential cell-kill of Tregs in TME; lack of blockade effect may reduce peripheral autoimmune toxicity

SOLID TUMOR: MT-5111

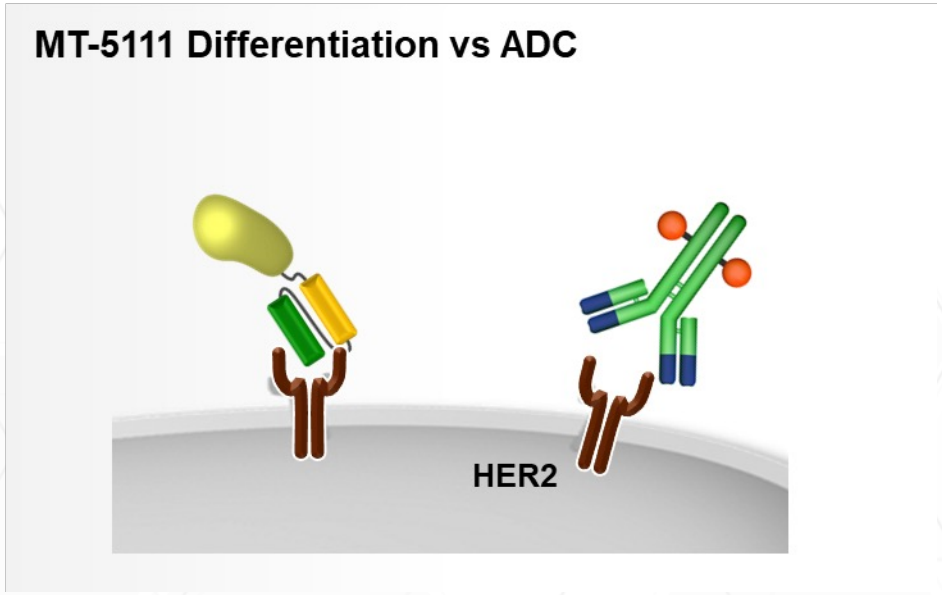
ANTI-HER2 ETB



MT-5111: Anti-HER2 ETB with Differentiated Binding and MOA profile



- Binds HER2 at a distinct epitope from trastuzumab and pertuzumab
- De-immunized Shiga-Like Toxin (SLT-A) Payload
- Reduced TLR4 interaction to minimize innate triggering (CLS)
- Potent direct cell kill (pM) against HER2 expressing cancer cells



- Potent MOA of direct cell kill that does not appear subject to resistance mechanism to Mabs or ADC chemo payloads
- Distinct HER epitope binding allows for combination potential with Mabs and ADCs
- Smaller size (monomer – 55kDa) may allow for improved tumor penetration
- No conjugation chemistry involved to improve manufacturability

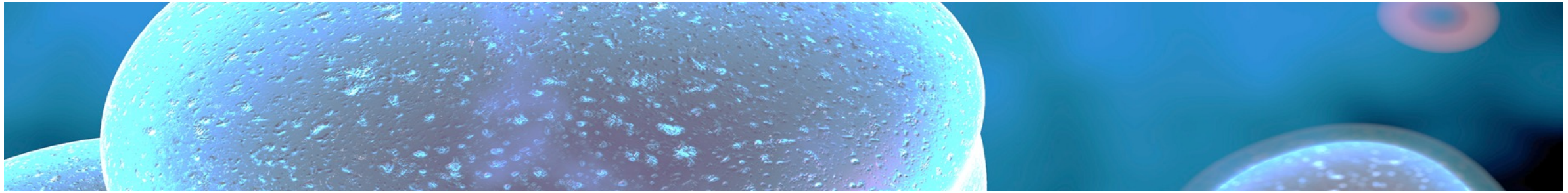
MT-5111: Dose Levels at 10 mcg/kg or Higher Likely Needed for Activity

- **27 patients dosed with no DLTs or CLS observed to date - dose escalation continues**
- **Dose level at 13 mcg/kg and breast cancer dose expansion cohort (10 mcg/kg) currently enrolling with >20 sites open**

MT-5111 Cmax (ng/mL) Across Dose Cohorts (0.5 to 10.0 mcg/kg)

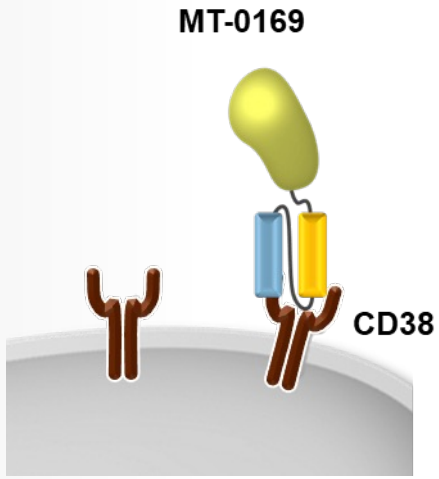
HEMATOLOGICAL MALIGNANCIES: MT-0169

ANTI-CD38 ETB



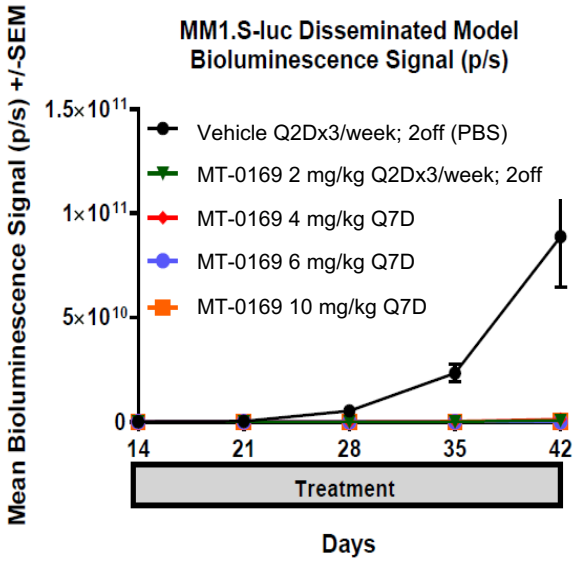
MT-0169: New Modality Targeting CD38

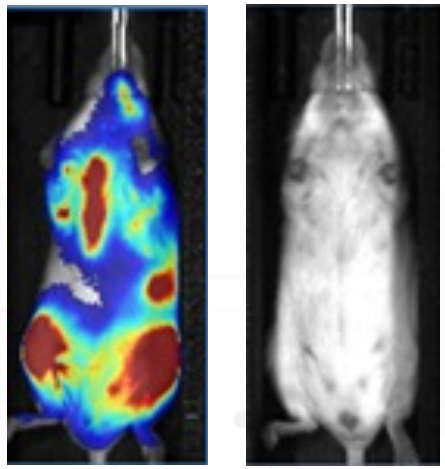
MT-0169 Profile



- Binds CD38 with high specificity and potency retained even in presence of daratumumab (overlapping epitope)
- De-immunized Shiga-Like Toxin (SLT-A) Payload
- Reduced TLR4 interaction to minimize innate triggering (CLS)
- Potent direct cell kill against CD38 expressing cancer cells (sub pM – most potent ETB created to date)

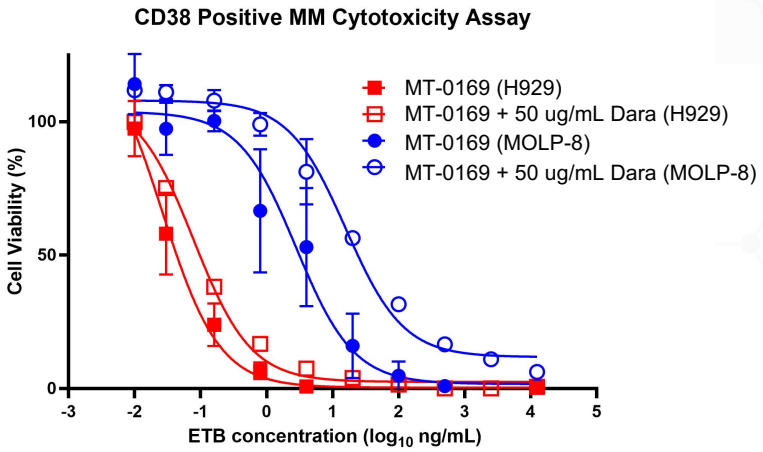
Potent Antitumor Activity in Various Xenograft Models[1]

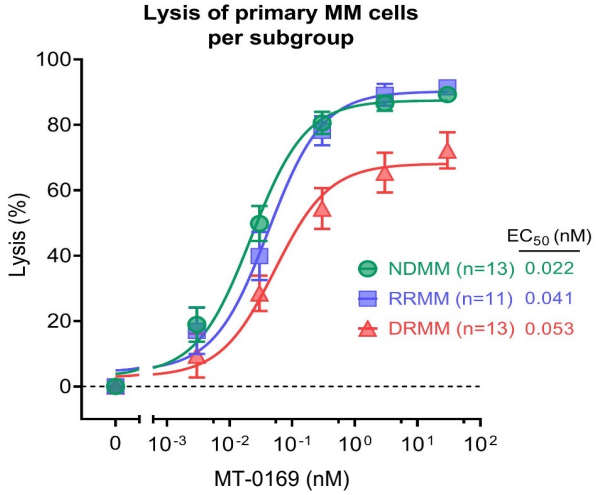




Activity in Presence of Dara and in Dara Refractory Primary MM Cells[1]

1. 2019 AACR Abstract 2384





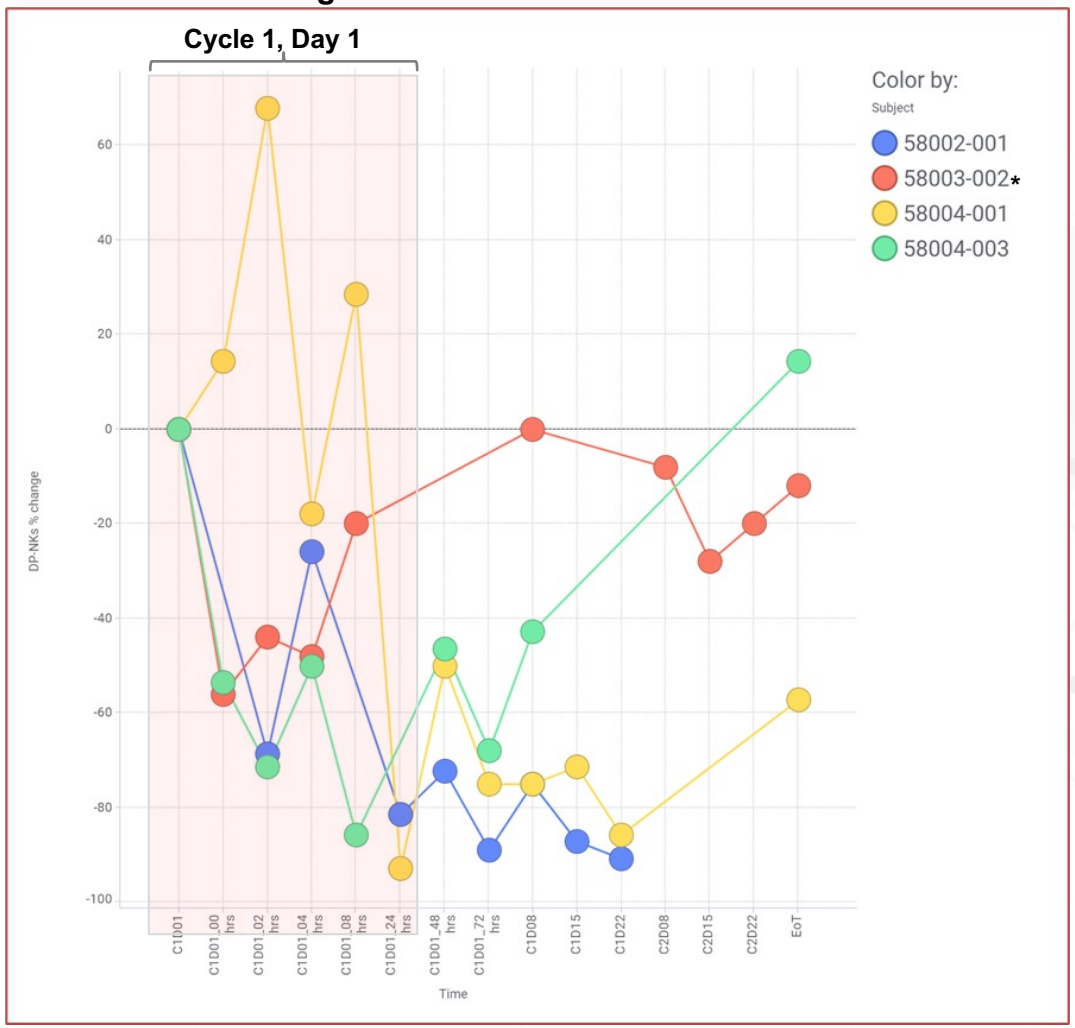
MT-0169: PD Activity Observed in Phase 1 and Safety Signals

- **Five pts treated to date with two pts with cardiac AEs that meet criteria for DLT**
  - Evaluation of both AEs triggered by asymptomatic elevations in high-sensitivity troponin
  - The first DLT was an asymptomatic, rapidly reversible episode of myocarditis that did not require treatment
  - The second DLT was an asymptomatic, nonischemic cardiomyopathy that also resolved within 1-2 months
  - Similar clinically relevant cardiac AEs not seen with any other ETB at any dose in clinic

- **DLTs may be due to MT-0169 activity against low CD38-expression in cardiac endothelium**
  - Starting dose of 50 mcg/kg is highest starting dose for any ETB
  - Rapidity and depth of CD38+ NK cell depletion in humans was unanticipated by NHP data
    - Lack of NK cells can drive eosinophilic infiltration in experimental model of myocarditis
  - Cardiotox not seen with CD38 Mabs likely due to low CD38 MFI and limited ability for effector function (ADCC, CDC) in tissue

- **The patient with cardiomyopathy had evidence of symptomatic benefit (reduction in myeloma bone pain)**
  - Anti-tumor effects can take longer than PD effects due to proliferative nature of malignancy

NK Cell – Fold Change over Time



**Patient 58003-002 had low CD38 expression on NK cells*

Corporate Summary

- **Clinical data milestones expected across three ETB programs in 2022**
  - MT-6402 demonstrating early signs of clinical benefit and pharmacodynamic activity
  - MT-5111 drug exposure levels now in therapeutic range
  - MT-0169 shows potent pharmacodynamic activity; moving forward with dose reduction
- **Potential for initiation of combination studies across programs by early 2023**
- **New IND for CTLA4 ETB (differentiated I/O approach to CTLA-4) at YE22**
  - Expect one new ETB IND each year
- **Ongoing pharma partnership**
- **Cash runway to end of 2023**

Advancing Wholly Owned Clinical Pipeline with Novel MOAs

ETB PROGRAM	TARGET	MOA	INDICATION
MT-6402	PD-L1	• Direct cell kill • Antigen Seeding (CMV antigen)	Solid Tumors
MT-5111	HER2	Direct cell-kill	Breast Cancer
MT-0169	CD38	Direct cell-kill	Multiple Myeloma
ETB Candidate	CTLA-4	Direct cell-kill	Solid Tumors
ETB Candidate	TIGIT	Direct cell-kill	Solid Tumors
ETB Candidate	TROP-2	• Direct cell kill • Antigen Seeding (CMV antigen)	Solid Tumors
ETB Candidate	SLAMF-7	Direct cell-kill	Multiple Myeloma

- **Relevant data updates on pipeline expected throughout 2022**
- **Data presentations expected at ASCO (Jun), SITC (Nov), SABC (Dec), and ASH (Dec)**