UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): January 7, 2021

Molecular Templates, Inc.
(Exact name of registrant as specified in its charter)

Delaware	001-32979	94-3409596
(State or other jurisdiction of incorporation)	(Commission File Number)	(IRS Employer Identification No.)

9301 Amberglen Blvd, Suite 100
Austin, TX 78729
(Address of principal executive offices and zip code)

Registrant's telephone number, including area code: (512) 869-1555

(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligations of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, $0.001 Par Value Per Share	**MTEM**	**The Nasdaq Capital Market**

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01: Regulation FD Disclosure.

Attached hereto as Exhibit 99.1 and incorporated by reference herein is the January 2021 Corporate Presentation of Molecular Templates, Inc., which is being provided in connection with upcoming investor conferences.

The information set forth under this "Item 7.01. Regulation FD Disclosure," including Exhibit 99.1 attached hereto, shall not be deemed "filed" for any purpose, and shall not be deemed incorporated by reference into any filing under the Securities Act of 1933, as amended, or the Securities Exchange Act of 1934, as amended, regardless of any general incorporation language in any such filing except as shall be expressly set forth by specific reference in such filing.

Item 9.01 Financial Statements and Exhibits

(d) *Exhibits.*

Exhibit 99.1	Molecular Templates, Inc. Corporate Presentation, Dated January 2021.
Exhibit 104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the Registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Molecular Templates, Inc.

Dated: January 7, 2021

By: /s/ Eric E. Poma, Ph.D.
Name: Eric E. Poma, Ph.D.
Title: Chief Executive Officer

Exhibit 99.1

MOLECULAR TEMPLATES

Corporate Presentation I January 2021

(NASDAQ: MTEM)

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; and our future cash needs. 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 act through a potent and unique mechanism of action: ribosomal destruction

Global Partners

Takeda: CD38 co-development, multi-target collaboration, equity investment. **Vertex:** Multi-target collaboration around myeloablation, equity investment

Advancing Pipeline

POC with 1st-Gen ETB demonstrating forced internalization, efficacy, and safety. Two 2nd-Gen ETBs in clinic with improved efficacy and safety. FPI with 3rd-Gen ETB targeting PD-L1 in 1H21.

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

Known Targets for Early Signs of Safety & Efficacy

ETBs against validated targets can provide evidence of safety and response as early as Phase 1

Strong Cash Position

Current cash funds operations into 2H22 without additional business development

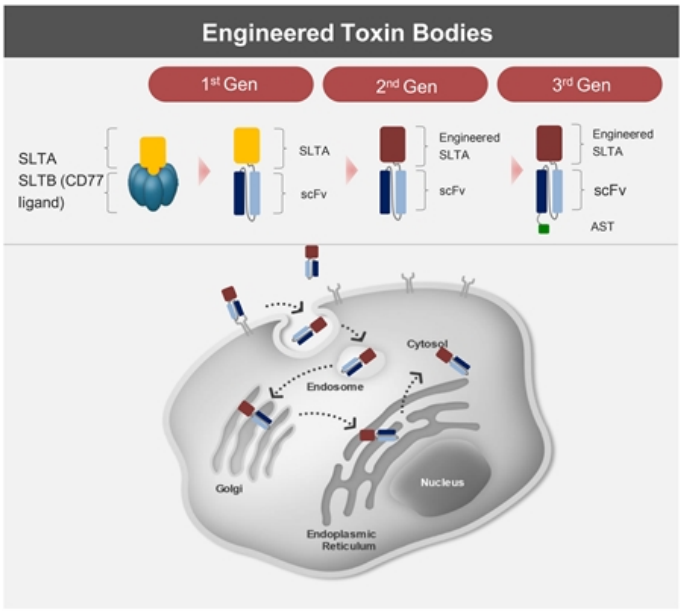
ETBs: Novel Mechanisms of Action in Oncology

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, safety, and efficacy 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: 1st-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

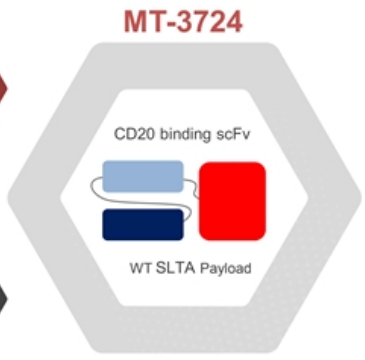
Wild-type Shiga-like toxin A-subunit payload. Retains TLR4 interaction

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



1st-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

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

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

MD Anderson Cancer Center · Memorial Sloan Kettering Cancer Center · UNC School of Medicine · University of Arizona Cancer Center

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

Median number of prior therapies in responders was 3; responses seen in R-CHOP refractory pts. Includes 2 stable disease patients who had 49% and 47% tumor reductions

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_{max} exposure observed in last 5 of 6 subjects dosed on MT-3724 monotherapy study**
  - Higher than expected pharmacokinetic (beyond pharmacokinetic projections) observed in these 5 subjects
  - All five subjects received drug from the same single lot of MT-3724 drug product
  - C_{max} exposures observed in these five subjects were higher than has been observed in any other subjects or studies with MT-3724
  - Elevated C_{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 Safety; Reduced Aggregation

2nd-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_{50}): ~sub-nanomolar

- MT-3724 utilizes wild-type SLTA payload with intact innate receptor interaction (TLR4)
- Suboptimal structure orientation

Next-gen ETBs

Proprietary De-immunized SLTA payload

NHP Tox: HNSTD > 500 ug/kg

In-vitro potency (IC_{50}): ~pico to sub-picomolar

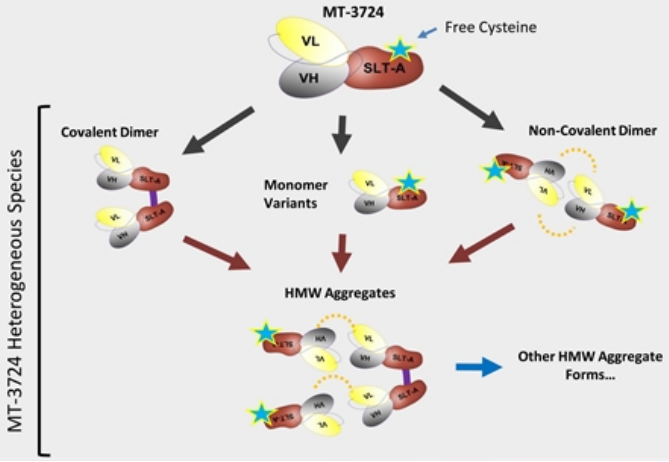
- ETBs utilize next-gen SLTA payload de-immunized to reduce adaptive and innate immunogenicity
- ETB structural modifications (orientation) greatly improve potency (more efficient intracellular routing)

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

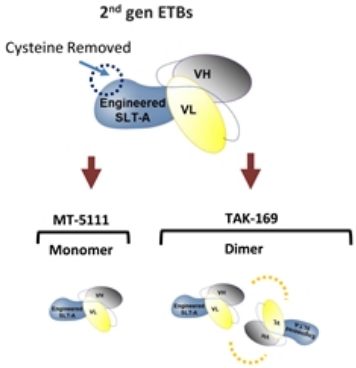
- 15-mer linker in scFv can create dimers and monomers
- Free cysteine on SLTA scaffold used to create dimers via oxidation step during purification



2nd-Gen ETBs

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

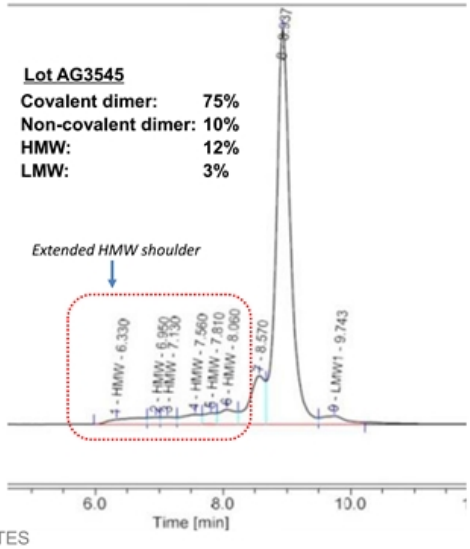
- 25-mer linker in scFv used to force monomers (MT-5111)
- 5-mer linker used to create diabodies (TAK-169 and MT-6402)
- No free cysteine in Next-Gen SLTA scaffold
- MT-6402, 3rd-gen ETB, utilizes immunodominant 9-mer CMV peptide (hydrophobic) fused to C-terminus for Antigen Seeding



Purity (SE-UPLC): 1st-gen ETB (MT-3724) vs 2nd-gen ETB (MT-5111)

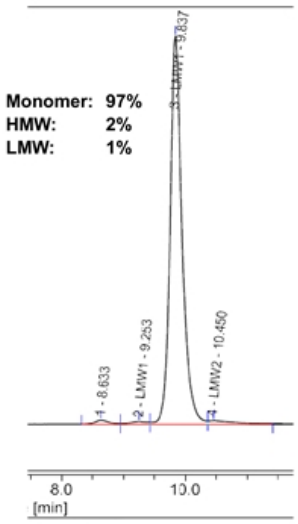
MT-3724 (1st gen) scaffold exhibits propensity to form higher order aggregates via covalent and non-covalent interactions

MT-3724 (1st gen ETB)



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

MT-5111 (2nd-gen ETB)



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)

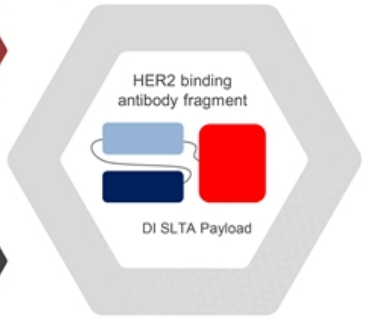
NOVEL ENZYMATIC PAYLOAD

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

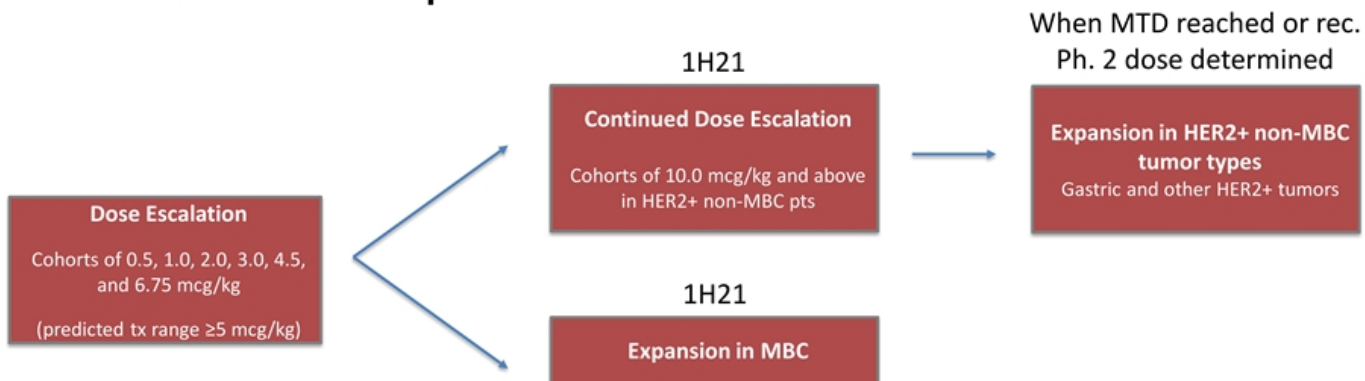
OTHER PROPERTIES

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

MT-5111:
2nd-Generation ETB



MT-5111: Clinical Development



- 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

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)

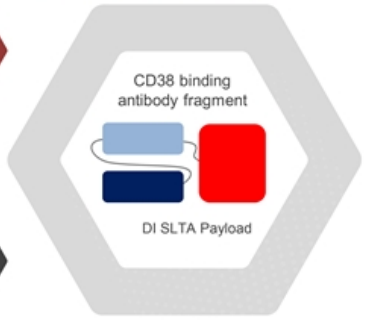
NOVEL ENZYMATIC PAYLOAD

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



TAK-169: 2nd-Gen ETB Targeting CD38

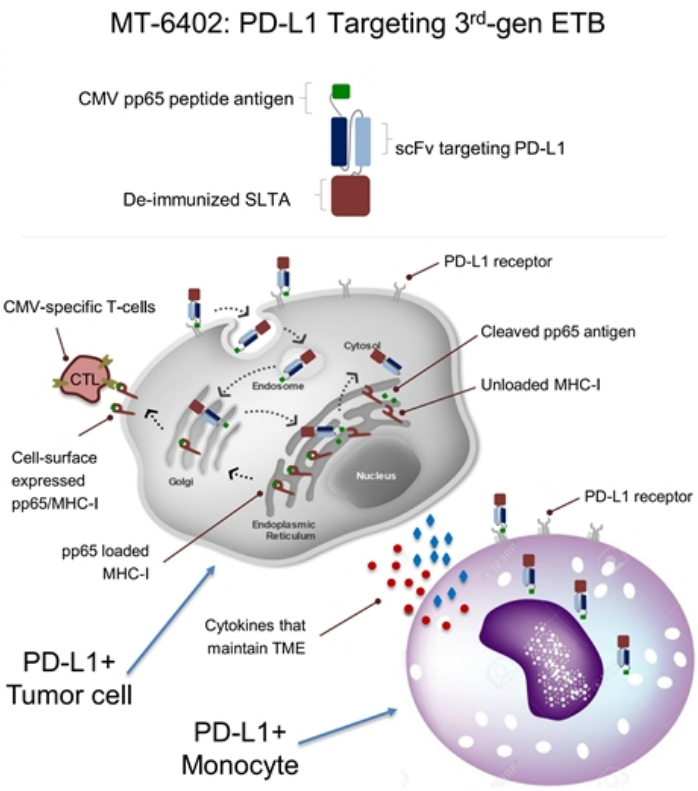
CD38-targeting Agents		
	Mab: Darzalex	Engineered Toxin Body: TAK-169
		
MOA	Indirect CDC cell kill	Direct cell kill (enzymatic ribosome inactivation)
CD38 target interaction	Binding	Binding and internalization
Limitations	CD55/59 upregulation in failures, inhibiting immune response	None identified

- CD38 is a poorly-internalizing receptor central to disease in multiple myeloma
- TAK-169 efficiently internalizes and destroys low- or high-expressing CD38
- TAK-169 activity is retained in the presence of daratumumab in preclinical models
- TAK-169 active in patient samples (including dara-refractory)
- TAK-169 has shown activity in xenograph models when dosed weekly or bi-weekly
- Reduced ADA and innate response (de-immunized STLA scaffold)
- HNSTD of 750 mcg/kg in NHPs (150 mcg/kg for MT-3724)
- Phase I in rel/ref myeloma patients with weekly dosing started at 50 mcg/kg


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

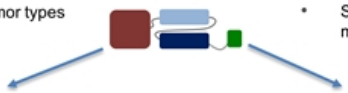
- ETBs localize to ER/cytosol to “seed” tumors with foreign non-self antigens
- Antigen cleaved intracellularly and presented on cell surface in context with MHC-I
- Delivery of pp65 CMV antigen
  - Mediate native CMV-specific T cell response to tumor
  - Large existing population infected with CMV
  - 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**



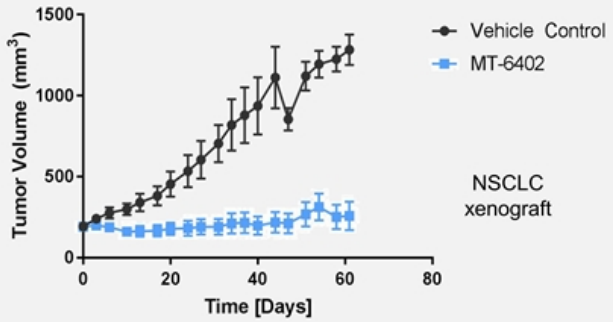
MT-6402: Potent Activity Against PD-L1+ Tumor Cells

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

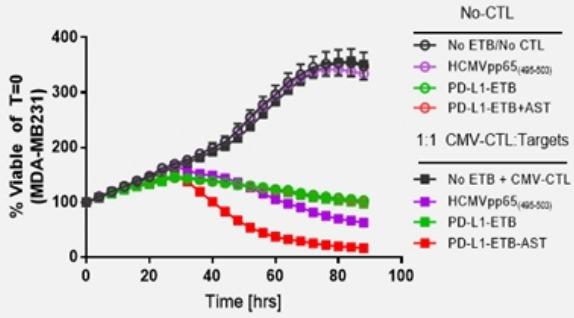
- Ribosomal destruction is independent of tumor microenvironment
- Novel, potent MOA not previously used against PD-L1+ tumor types
- Unprecedented alteration of tumor immunophenotype
- Strong evidence that CMV-specific T-cells are present in tumor microenvironments (Rosato et al, Nature Comm 2019)



MOA1 (ribosomal destruction)

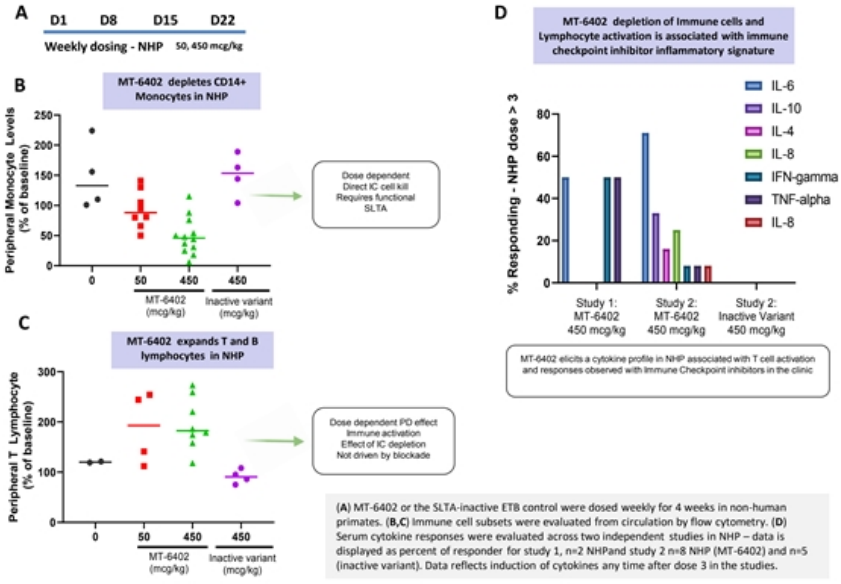


MOA1 + MOA2 (AST)



MT-6402: Potent Activity Against PD-L1+ Immune Cells in the TME

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



(**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 NHPand 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.

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

Group	Dosing	Immune Effects	Pharmacodynamic Response
MT-6402	50 mcg/kg	↑Monocyte depletion ↑T cell, B cell, Eosinophil expansion increased with sequential dosing	Minimal Troponin-I release; no significant cardiac findings; evidence of skin inflammation[1]
MT-6402	450 mcg/kg	↑↑Monocyte depletion ↑↑↑T cell. B cell, NK, Eosinophil expansion; increased with sequential dosing Cytokine induction IL-2, IFN-γ, TNF-α, IL-6, IL-10	Troponin-I release associated with CD3+ T cardiac infiltration minimal monocyte and B ell infiltration[2]; resolving with dose cessation evidence of skin inflammation[1]
SLTA-inactive	450 mcg/kg	No remarkable findings	No Remarkable findings

1,2 – myocarditis and dermatitis are common immune-related adverse events (irAE) observed predominantly with combination immune checkpoint inhibitors in clinical settings and are associated with beneficial therapeutic response (3,4).

(E) 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 (irAE) in NHP and humans correlative to favorable response (3,4). Targeting cells for depletion with MT-6402 may lead to unique benefits in the clinic not observed with antibody block which mediates activity through steric block alone. **(F)** MT-6402 stimulates immune activation and a pharmacodynamic and irAE profile consistent with combination but not single agent checkpoint inhibitors in NHP (3,4). Reposes are dose dependent and mediated by STLA direct cell kill and not steric inhibition as the inactive control does not trigger immune activation or PD response *in vivo*. ***MT-6402 represents a unique therapeutic approach with the ability for potent responses in humans as a single agent therapy.***

ETBs and IO: PD-L1 ETB Moving to Clinic; New IO Targets in the Works

Potent effect on PD-L1+ tumor cells and immune cells

- Direct cell-kill on tumor cells through ribosomal destruction (MOA1) independent of tumor microenvironment
- Novel alteration of cancer cell immunophenotype for pre-existing, synaptic T-cell recognition of tumor (MOA2)
- Early in vivo and in vitro data suggest potent activity on PD-L1 immune cells and activation of immune system

Exploration of additional IO targets where ETB approach may provide substantial differentiation

- CTLA4 lead development work underway; IND filing expected in 2021
- Potential safety and efficacy benefits around direct cell-kill of CTLA4+ T cells vs blocking

Engineered Toxin Bodies

A Robust Pipeline with Clinical Data in 2021

Robust Clinical Pipeline Driving Value of Drug Candidates and Platform