Molecular Templates

Corporate Presentation
December 2018

Forward-Looking Statements

Except for statements of historical fact, the statements in this presentation are forward-looking statements, including statements regarding the future development of our proprietary Engineered Toxin Body (ETB) technology; the future development of evofosfamide; our milestones; 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 our failure to secure and maintain relationships with collaborators; (2) risks relating to clinical trials and other uncertainties of product candidate development; (3) risks relating to the commercialization, if any, of our proposed product candidates (such as marketing, regulatory, product liability, supply, competition, and other risks); (4) dependence on the efforts of third parties including our strategic partners; and (5) dependence on intellectual property. 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.

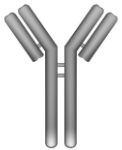
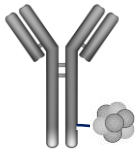
MTEM is Developing a Pipeline of Novel Oncology Therapies

- Engineered Toxin Bodies (ETB) act through a potent and unique mechanism of action
- Lead program MT-3724 has demonstrated single agent responses in advanced DLBCL
- Three new ETBs targeting CD38, HER2 and PD-L1 expected to enter the clinic in next 12 months
- Development of ETBs against validated targets includes evaluation of safety and responses in Phase I
- Growing Takeda relationship: CD38 co-development partnership, multi-target collaboration, equity investment
- ETB platform technology provides continued pipeline expansion and enables rapid drug discovery through partnerships and internal development

Engineered Toxin Bodies

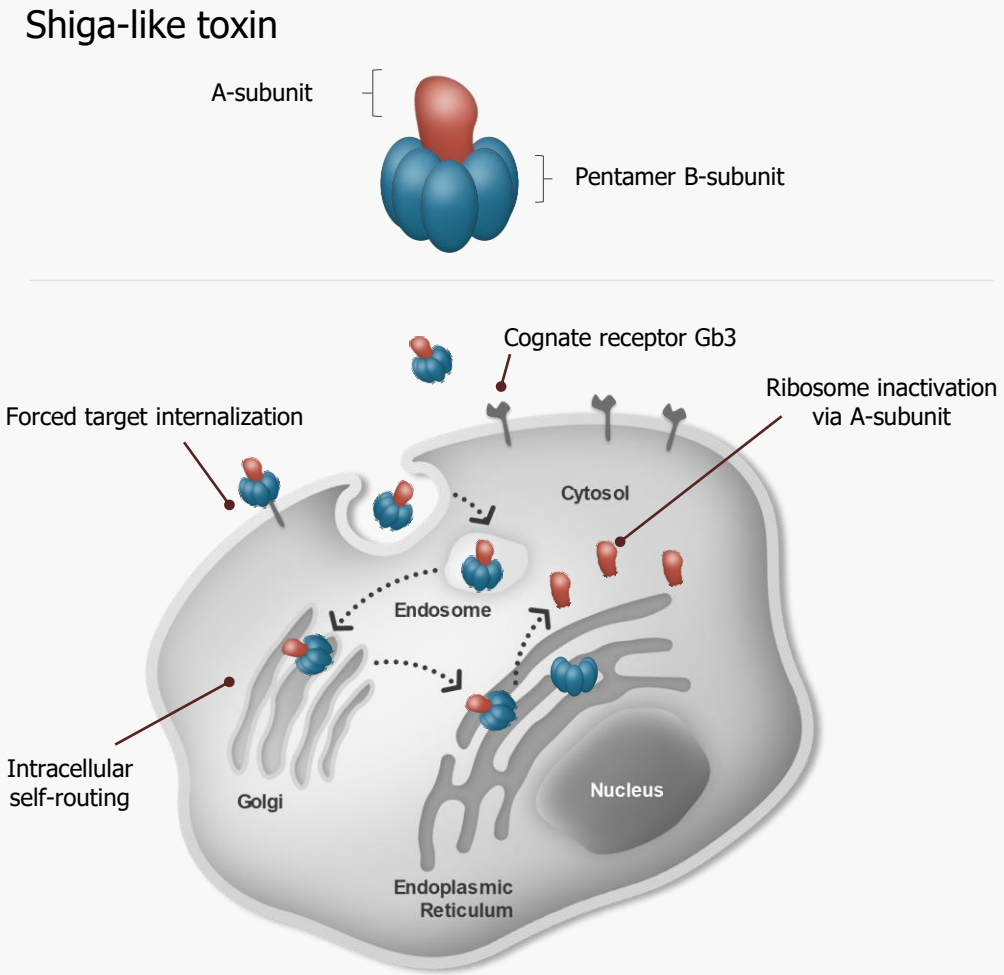
Technology Overview

Evolution of Antibody-Based Therapeutic Platform: ETBs Have Unique Biological Properties

	Monoclonal antibody	ADC	Engineered Toxin Body
			
MOA	Indirect cell-killing: ADCC/CDC	Direct cell kill: Chemo-based	Direct cell-kill: Ribosomal destruction
Receptor target internalization required	No	Yes	No: Forced internalization
Intracellular routing	No	No	Yes: Payload delivery

Engineered Toxin Bodies: Leveraging the Unique Biology of Shiga Toxin

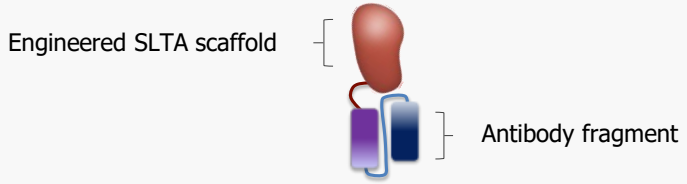
- Shiga-like toxin (SLT) produced by certain E. coli strains made of two parts:
  - A-subunit: catalytic activity
  - B-subunit pentamer: binds cognate receptor Gb3, a non-internalizing glycosphingolipid
- Shiga-like toxin subunit A (SLTA) has evolved to retain biological activity inside the cell:
  - Forced receptor internalization – clathrin mediated
  - Intracellular self-routing – endosomal escape to TGN, ER, and cytosol
  - Ribosome-inactivation – enzymatically and permanently depurinates 28S rRNA

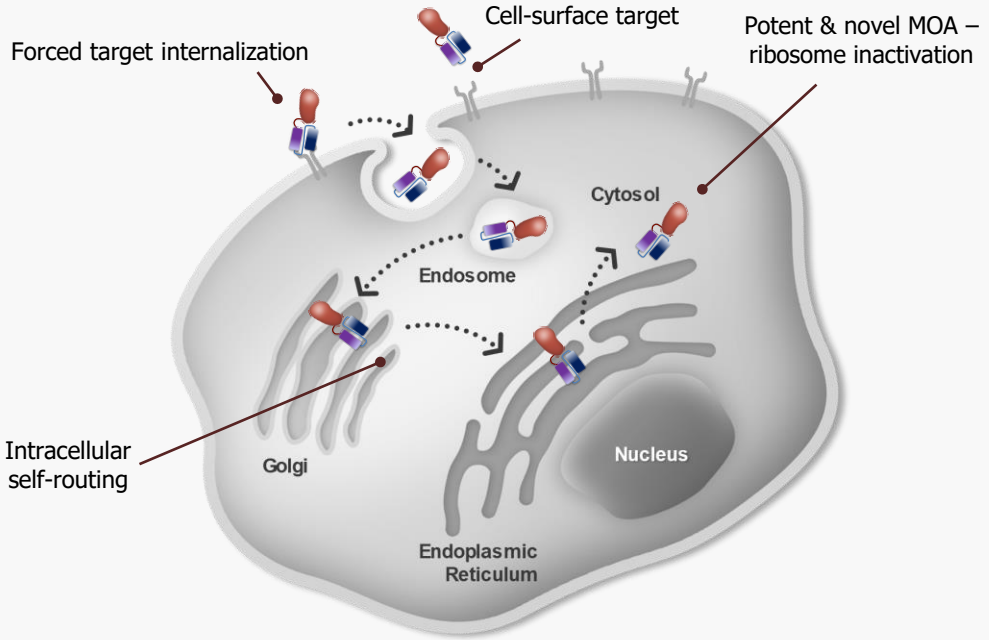


Engineered Toxin Bodies: Novel Modes of Action

- Targeted therapies derived from an engineered Shiga-like toxin A-subunit (SLTA) scaffold with novel MOAs
  - Antibody-like target specificity
  - Forced target internalization
  - Intracellular routing
  - Enzymatic ribosome inactivation – unique mode of killing

Engineered Toxin Bodies

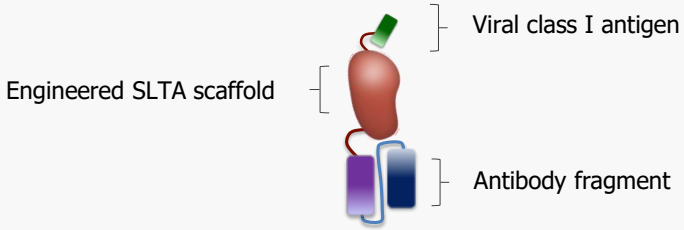


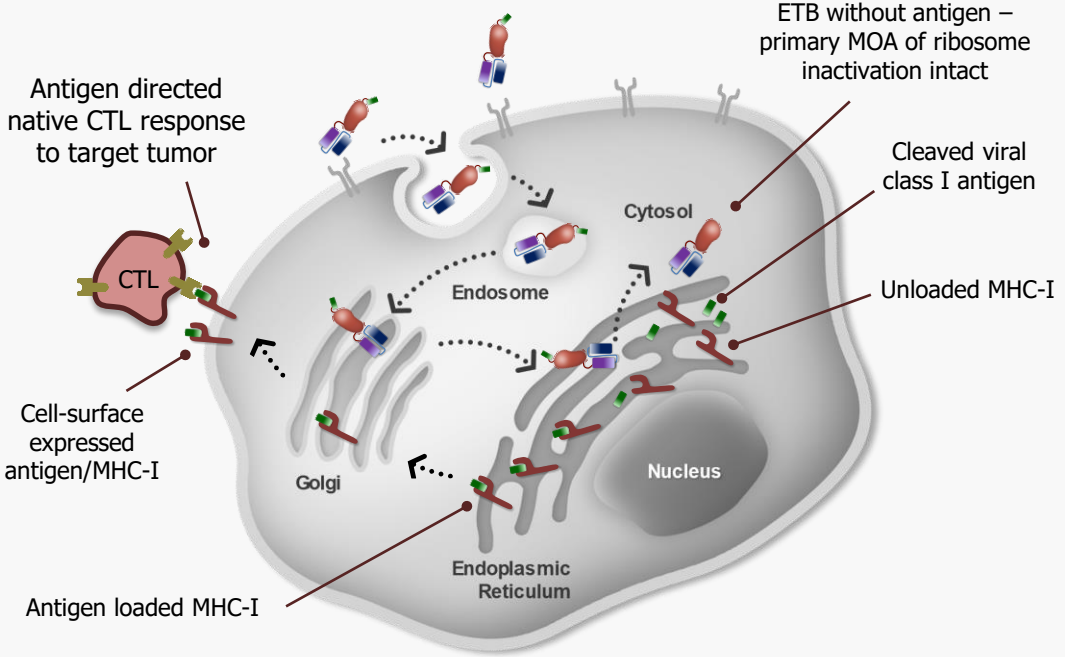


Antigen Seeding: Novel I-O Approach Leveraging ETB biology

- ETB delivery of viral class I antigens inside target tumor
- Antigen processed and presented on cell surface loaded on MHC-I
- Re-directed native immunity (antigen specific CTLs) to target tumor
- Dual MOAs of action potential – ribosome inactivation cell-kill intact

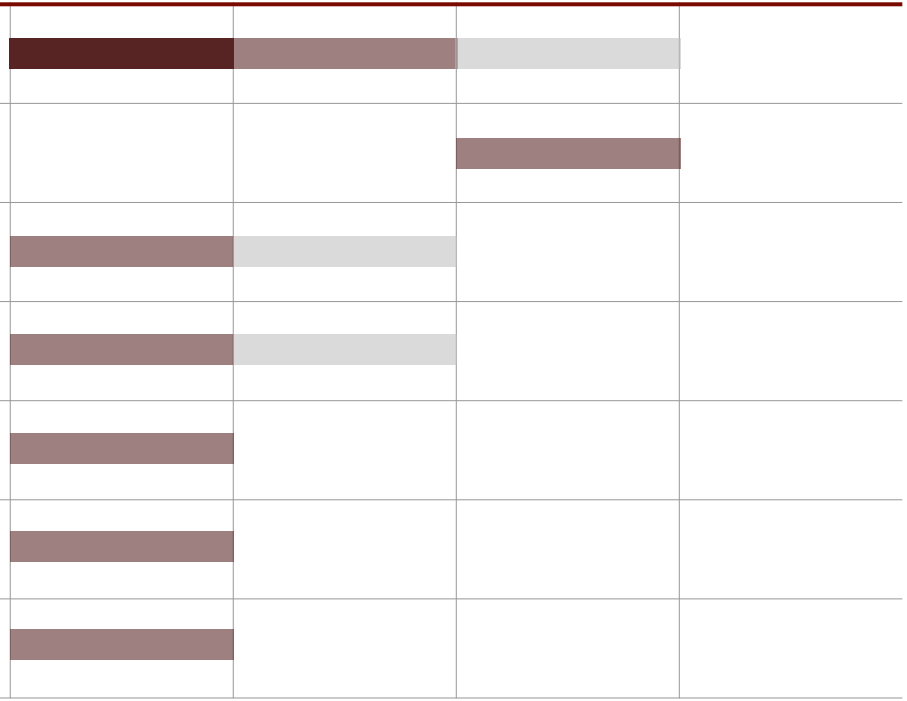
Engineered Toxin Bodies





Oncology Pipeline with Novel MOAs Driven by ETB Platform

Program	Partner	Indication (Target)
MT-3724	MolTem	DLBCL[a] monotherapy (CD20)
	MolTem	DLBCL combination (CD20)
MT-5111	MolTem	Multiple – solid tumors (HER2)
TAK-169	Takeda Millennium	Multiple Myeloma (CD38)
PD-L1 ETB (antigen seeding)	MolTem	Multiple – solid tumors (PD-L1)
Takeda Target 1	Takeda Millennium	Undisclosed
Takeda Target 2	Takeda Millennium	Undisclosed



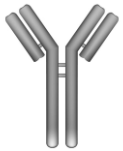
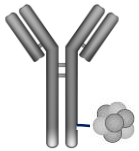
a) Potential for Phase 2 to be pivotal in relapsed/refractory setting as monotherapy

MT-3724

ETB Targeting CD20 for Lymphomas

MT-3724 – a Novel MOA to CD20

CD20 targeting agents

	Monoclonal antibody (Rituxan)	RIT (Bexxar, Zevalin)	Engineered Toxin Body (MT-3724)
			
MOA	ADCC/CDC	Direct cell kill (radioisotope)	Direct cell-kill (enzymatic ribosome inactivation)
CD20 target interaction	Binding	Binding	Binding and internalization

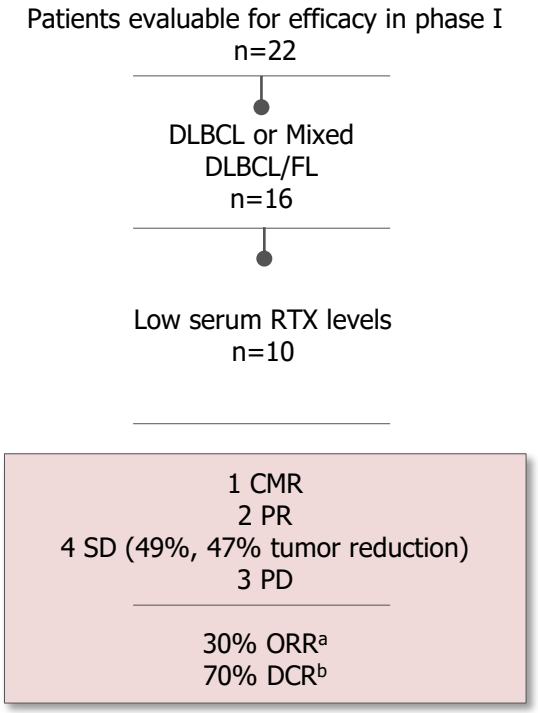
- CD20 central to disease in lymphomas
- CD20 is a non-internalizing receptor
- RITs have shown comparable to favorable activity over naked Mabs
- Mabs only available MOA targeting CD20
- MT-3724 kills via a novel MOA – forced internalization against CD20 and enzymatic and permanent protein synthesis inhibition

Phase 1 Study Conducted in Heavily Pretreated NHL Patients

Characteristics (n=24)[a]		
Age (mean, range, years)		69 (34-78)
NHL subtype (n, %)	DLBCL (diffuse large B-cell lymphoma)	15 (63%)
	FL (follicular lymphoma)	4 (16%)
	Mixed histology (DLBCL and FL)	3 (13%)
	MCL (mantle cell lymphoma)	2 (8%)
Prior NHL treatments (median)		**5**
Rituxan serum levels[b] (n)	Low/undetectable	16
	High (5-116x higher than MT-3724)	8

a) Clinical sites include: MD Anderson, Memorial Sloan Kettering, University of North Carolina, and University of Arizona
b) Patient serum levels of RTX (rituximab) at first dosing of MT-3724; RTX and MT-3724 compete for CD20 binding; RTX dosed in gram/dose vs MT-3724 dosed in mg/dose. Low/undetectable determined by assay (cutoff of <500 ng/mL)

Promising Signals of Activity Demonstrated in DLBCL



- Promising signals of efficacy in heavily pretreated DLBCL patients
  - Deep and prolonged dose-dependent B-cell depletion observed
- High levels of serum RTX inhibits MT-3724 activity
  - Patients screened out for high RTX in ongoing phase 1b study (0/6 response rate)
- Favorable tolerability profile
  - MTD established at 50 mcg/kg
  - DLTs were non-life threatening grade 2/3 events consistent with capillary leak syndrome and resolved upon cessation of dosing

a) All responses were in RCHOP relapse/refractory patients with median prior therapies of 5
b) Includes 2 stable disease patients who had 49% and 47% tumor reductions

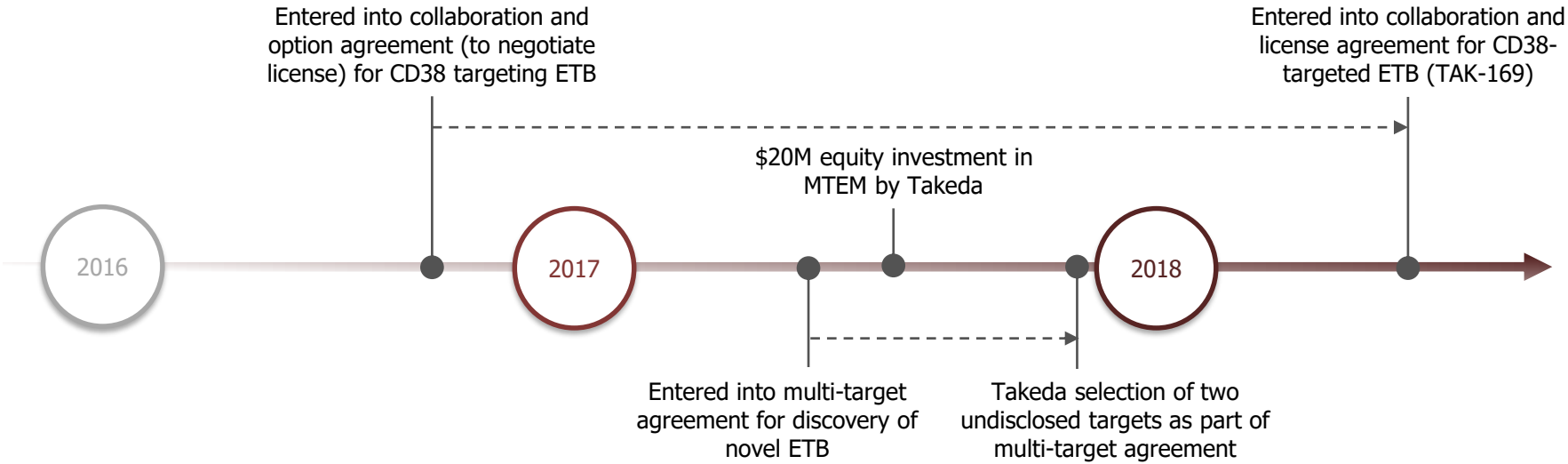
MT-3724 DLBCL Development Progressing and Expanding

Study	Indication	Status	Next Milestone
Monotherapy			
Phase 1b expansion	R-R DLBCL (2+ lines of therapy)	In-Progress	Transition to Phase 2
Phase 2	R-R DLBCL (2+ lines of therapy)	Planning	› Initiate 1Q19 › Potential to be pivotal study
Combination			
Phase 2 – GEMOX/MT-3724	R-R DLBCL (2nd line SCT ineligible)	In-Progress	Start enrollment in 4Q18
Phase 2 – Revlimid/MT-3724	R-R DLBCL (2nd line SCT ineligible)	Planning	Initiate in 1Q19

Takeda Collaboration and Partnership

ETB Platform Technology Facilitates Efficient Discovery of Novel ETBs

Collaborations with Takeda for CD38 ETBs and Two Undisclosed Targets



Takeda Multi-Target Collaboration Provides Capital and Pipeline Expansion

June 2017 agreement for MTEM to use Takeda antibodies to create ETBs against two undisclosed targets designated by Takeda that were not originally part of MTEM's pipeline

	Total for Two Targets
Upfront fee + program plans for designated targets	$5M
Exercise of option to license ETBs	$25M
Contingency fees for replacement of targets	$10M
R&D and commercial milestones	$547M
Tiered royalties	Mid-single to low-double digits

Collaboration for Co-Development of CD38 ETBs

- MTEM and Takeda conducted a thorough (18+ month) evaluation of ETBs created with Takeda antibodies as well as MTEM's MT-4019
- TAK-169 selected as lead compound:
  - Highly potent ETB
  - Favorable tolerability profile observed in NHPs
  - Clinical data in 2019

Takeda/MTEM Co-development Deal

	Terms
Upfront fee	$30.0M
Potential R&D and commercial milestones	$632.5M ($337.5M if no co-dev)
Tiered royalties	Double-digits up to low twenties (high single digits to low teens if no co-dev)
Co-development	50/50 cost share

TAK-169 Targeting CD38

MOA	Ribosome inactivation: highly potent
TAK-169 target	› CD38 is a poorly internalizing receptor target (not appropriate for ADC) › Lead ETB efficiently internalizes against CD38
Indication / opportunity	› Daratumumab failures retain CD38 expression but show CD55/59 upregulation (complement inhibiting protein) as means of Mab MOA escape › Post-daratumumab failures and in combo with standards of care
TAK-169 ETB Summary	› Potent activity against CD38 low or high expressing cells › Reduced ADA and innate response (DI SLTA scaffold)

Additional Pipeline Programs

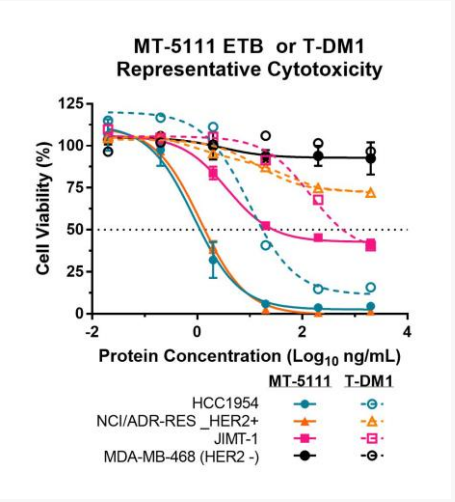
ETBs Targeting CD38, HER2, PD-L1

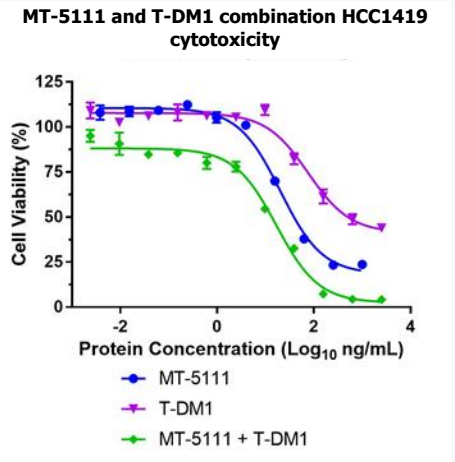
ETB Platform Technology Evolution Driven by Optimized Scaffold and New MOAs

Program	Ribosome Inactivation	Forced receptor internalization	De-immunized	Antigen seeding
MT-3724 (CD20)				
TAK-169 (CD38)				-- Not added -- T-cell engagement in myelomas not validated
MT-5111 (HER2)		-- Not applicable – HER2 self-internalizes		-- Not added – HER2 upregulation downregulates MHC-I
PD-L1 ETB				

MT-5111 Targeting HER2

MOA	Ribosome inactivation: picomolar IC_{50}
HER2 target	› HER2 persists after failure of existing modalities (Mab, TKI, ADC) › MT-5111 binds an epitope separate from trastuzumab
Indication / opportunity	› Ribosome inactivation represents a wholly distinct MOA versus antibodies, TKIs, and ADCs › Potential to combine through all lines of therapy given distinct binding epitope of MT-5111
Summary	› More potent than T-DM1 (Kadcyla) in *in vitro* assay › Active in T-DM1 refractory cell lines › Potentially additive or synergistic with T-DM1 › Reduced ADA and innate response (DI SLTA scaffold)





PD-L1 ETB Employs Two Novel MOAs

MOA	› Ribosome inactivation: picomolar IC_{50} › Antigen seeding (pp65 CMV peptide)
PD-L1 target	› PD-L1 central target on tumors that persists after Mab failures › PD-L1 is a poorly internalizing receptor (not appropriate for ADCs) › PD-L1 ETB efficiently internalizes against PD-L1
Indication / opportunity	› Various solid tumors via novel MOA of ribosome inactivation and novel immuno-oncology approach (antigen seeding)
Summary	› Ribosome inactivation mediated cell-kill not dependent on tumor microenvironment › PD-L1 ETB delivers pp65 CMV antigen inside the target cell and mediates pp65/MHC-I cell surface expression in an HLA:A02 restricted manner to direct potent CMV-specific CTL response

Antigen seeding is a novel I-O approach

Therapeutic Strategy	Targeting MOA	T Cell Requirements
Bispecific	T cell and Tumor	Polyclonal T Cell engagement May be restricted by local exhausted TIL
CAR-T	Tumor	Exogenous engineering required Difficult in vivo expansion
ETB antigen seeding	Tumor	Co-opts Endogenous memory Surveilling and expansion competent

Upcoming Milestones and Financial Summary

Important Expected Near-Term Milestones

	Timing	Milestone
MT-3724	4Q18	Start of enrollment in Phase 2 GEMOX combination study in DLBCL
	1Q19	Initiation of Phase 2 Revlimid combination study
	1Q19	Initiation of Phase 2 monotherapy study (potential pivotal)
	2019	Initial results from the Phase 2 combination studies
	2H19	Potential interim results for Phase 2 monotherapy study
Pipeline	1Q19	File IND for HER2 program
	2019	File IND for TAK-169 (CD38)
	2H19	File IND for PD-L1 program
	2H19	Preliminary results for HER2 Phase 1 study
Business Development	2018-2019	Continued progress with Takeda partnerships
	2018-2019	Potential new research partnerships

Financial Highlights

- ~$175M Market Capitalization, ~200,000 average daily trading volume
- 36.5 shares outstanding (44.0 fully diluted) as of 3Q18 10Q
- ~$109M in cash ($79M at 9/30/18 + $30M Takeda upfront) provides runway into 2021
- Top shareholders include:

Longitude CAPITAL
BVF PARTNERS L.P.
CAMCapital™ CAXTON ALTERNATIVE MANAGEMENT
SANTÉ VENTURES
PERCEPTIVE ADVISORS
Takeda

- Non-dilutive funding from CPRIT grants (State of Texas) and Takeda partnership
- Potential for additional non-dilutive funding through additional partnerships