NONCLINICAL DEVELOPMENT OF PROBODY DRUG CONJUGATES

Jennifer Richardson, Ph.D.
Vice President, Pharmacology & Toxicology

25th Annual Biotech Symposium
September 9-11, 2019

Forward Looking Statements

This presentation may contain projections and other forward-looking statements regarding future events. All statements other than statements of historical facts contained in this presentation, including statements regarding our future financial condition, technology platform, development strategy, prospective products, preclinical and clinical pipeline and milestones, regulatory objectives, expected payments from and outcomes of collaborations, and likelihood of success, are forward-looking statements. Such statements are predictions only and involve known and unknown risks, uncertainties and other important factors that may cause our actual results, performance or achievements to be materially different from any future results, performance or achievements expressed or implied by the forward-looking statements. These risks and uncertainties include, among others, the costs, timing and results of preclinical studies and clinical trials and other development activities; the uncertainties inherent in the initiation and enrollment of clinical trials; expectations of expanding on-going clinical trials; availability and timing of data from clinical trials; the unpredictability of the duration and results of regulatory review; market acceptance for approved products and innovative therapeutic treatments; competition; the potential not to receive partnership milestone, profit sharing or royalty payments; the possible impairment of, inability to obtain and costs of obtaining intellectual property rights; and possible safety or efficacy concerns, general business, financial and accounting risks and litigation. Because forward-looking statements are inherently subject to risks and uncertainties, some of which cannot be predicted or quantified and some of which are beyond our control, you should not rely on these forward-looking statements as predictions of future events. More information concerning us and such risks and uncertainties is available on our website and in our press releases and in our public filings with the U.S. Securities and Exchange Commission. We are providing this information as of its date and do not undertake any obligation to update or revise it, whether as a result of new information, future events or circumstances or otherwise. Additional information may be available in press releases or other public announcements and public filings made after the date of this presentation.

This presentation concerns products that have not yet been approved for marketing by the U.S. Food and Drug Administration (FDA). No representation is made as to their safety or effectiveness for the purposes for which they are being investigated.

Outline

- Overview of Probody™ technology
- CX-2009: a Probody Drug Conjugate (PDC) targeting CD166 (ALCAM)
  - Target rationale
  - Nonclinical safety and PK
  - Clinical study update
- CX-2029: a PDC targeting CD71 (transferrin receptor)
  - Target rationale
  - Nonclinical safety

Probody Therapeutics are Protease-activatable Antibody Prodrugs

ON TARGET TOXICITY HAS LIMITED THE DEVELOPMENT OF POTENTIALLY ATTRACTIVE ANTIBODY THERAPEUTICS

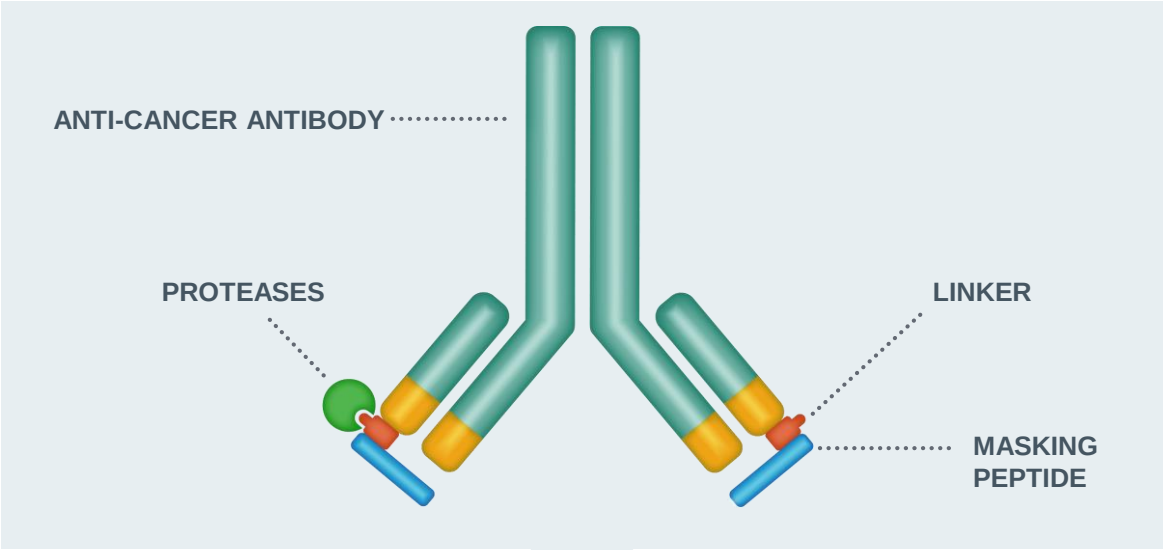
- Anti-EphA2 ADC, MEDI-547
- Anti-CD44v6 ADC, bivatuzumab mertansine

CYTOMX PROBODY PLATFORM IS DESIGNED TO LOCALIZE TARGET BINDING TO TUMOR

- Maintaining potency
- Reducing side effects
- Enabling new target opportunities

PROBODY PLATFORM IS APPLICABLE ACROSS MULTIPLE MODALITIES

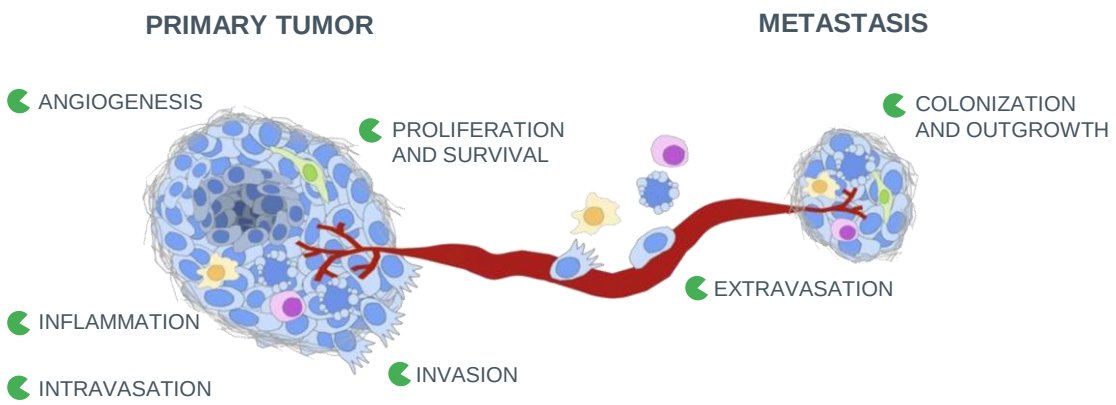
- Antibodies
- Antibody drug conjugates
- T cell engaging bispecific antibodies
- Chimeric antigen receptors, cytokines



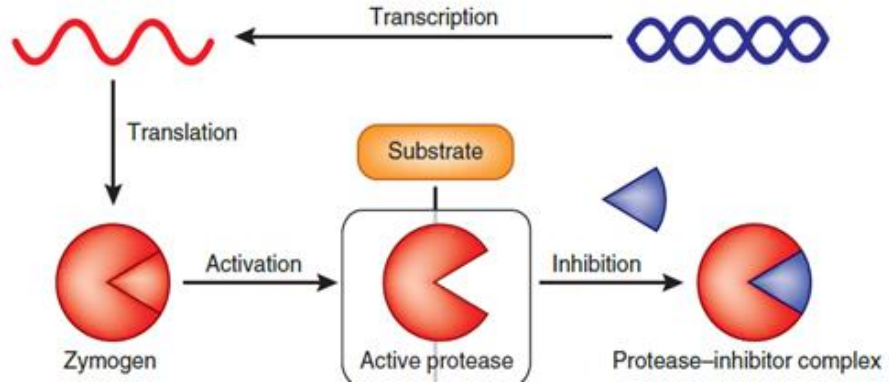
CytomX Therapeutics

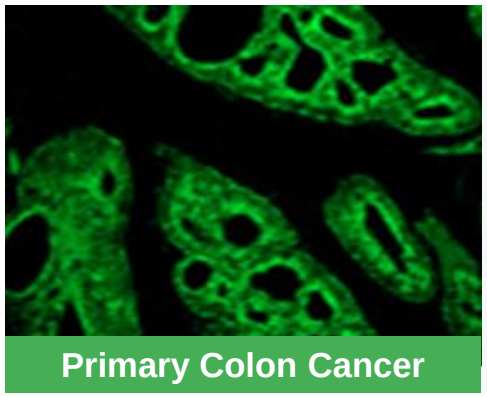
Activated Proteases are Prevalent in Tumors but Not in Healthy Tissue

UPREGULATED PROTEASE ACTIVITY IS A HALLMARK OF ALL CANCERS[1]



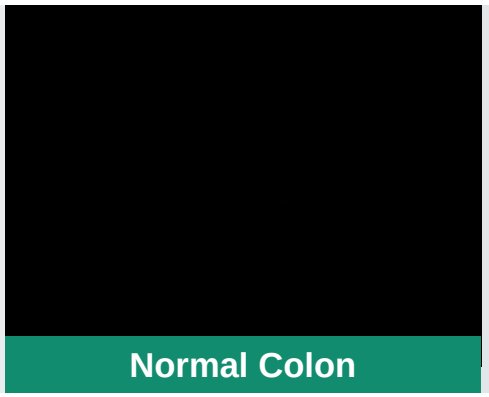
PROTEASE ACTIVITY IS TIGHTLY CONTROLLED IN HEALTHY TISSUES[2]





Primary Colon Cancer

Metastatic Colon Cancer



Normal Colon

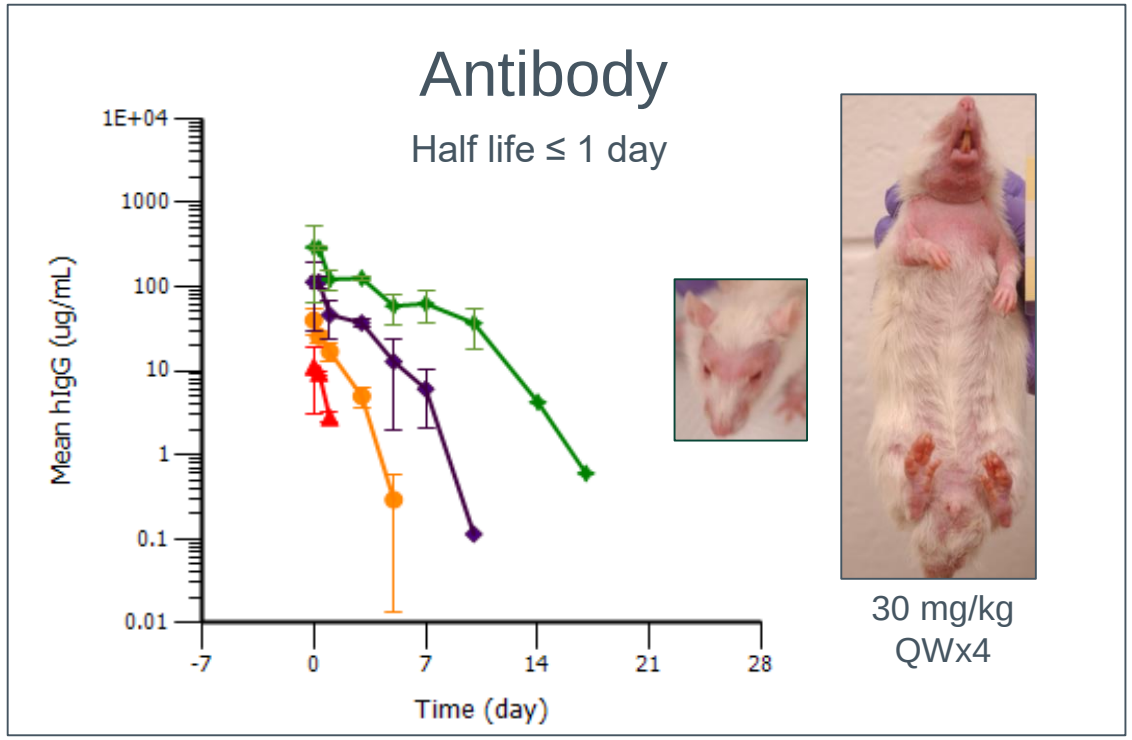
IMAGING OF ACTIVE PROTEASE[3]

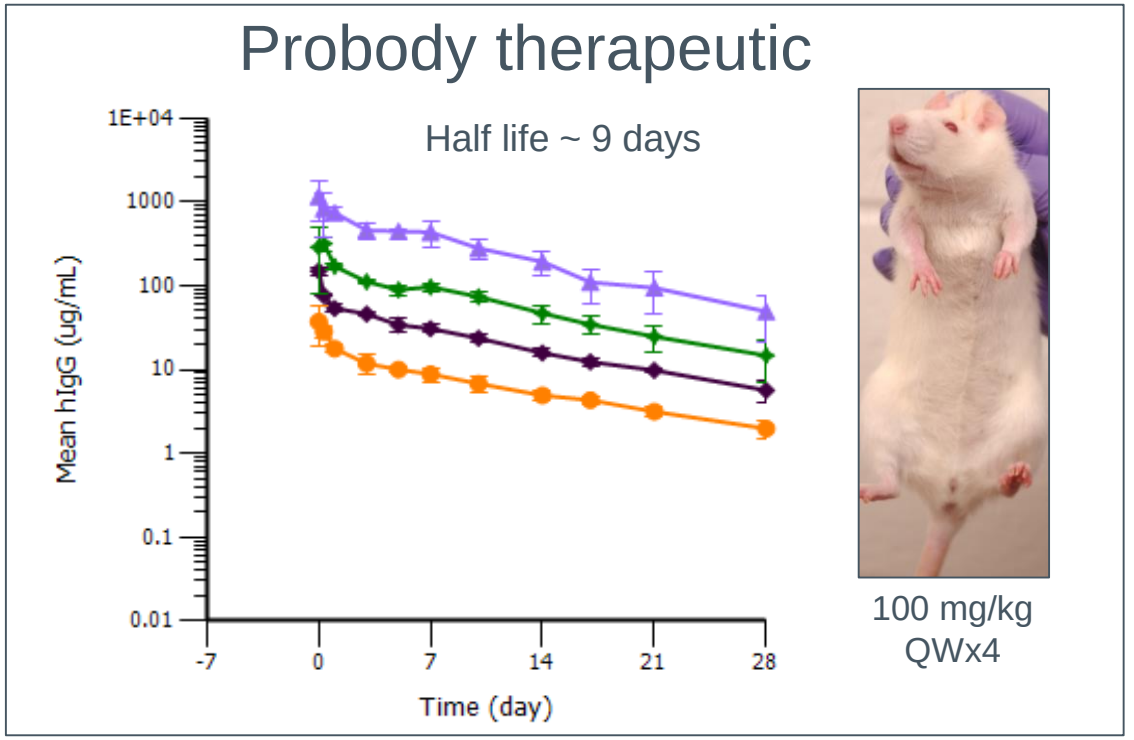
1. Sevenich, et. al. Gene & Dev., 2014; 2. Deu, et.al., Nature Struct Mol Biol 2012; 3. Matriptase: LeBeau, et al., PNAS 2013

CytomX Therapeutics

Antibody Masking Reduces Target Engagement in Healthy Tissues

Anti-Jagged Probody therapeutic (Tx) avoids TMDD and skin toxicity associated with anti-Jagged antibody in rats



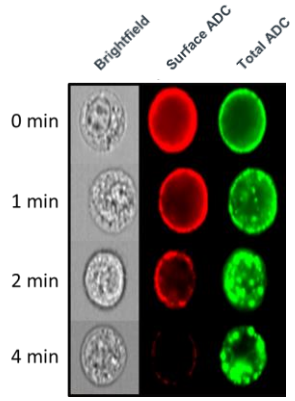


1 mg/kg 3 mg/kg 10 mg/kg 30 mg/kg 100 mg/kg

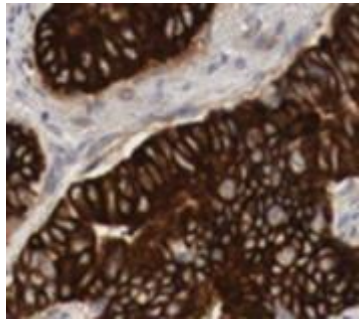
CytomX Therapeutics

The Probody Platform Potentially Enables an Attractive Class of ADC Target

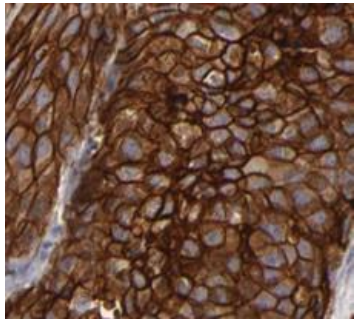
Efficiently Internalizing



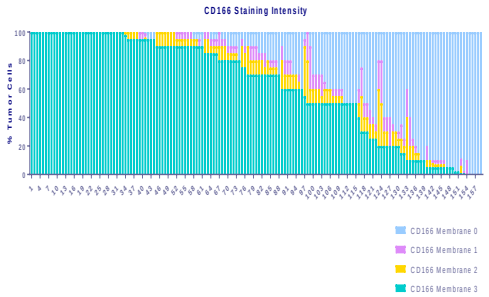
High Membrane expression



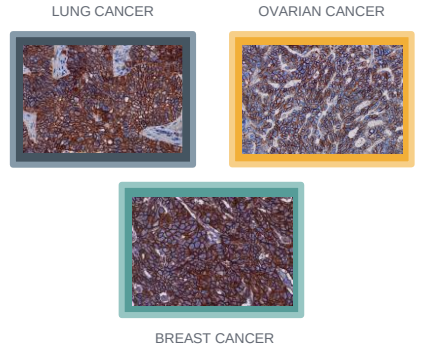
Uniform Tumor Expression



Majority of Patients Express at High Level



Highly Expressed in Multiple Common Cancers



These targets are typically expressed highly in normal tissues = not suitable for traditional ADC

CytomX Therapeutics

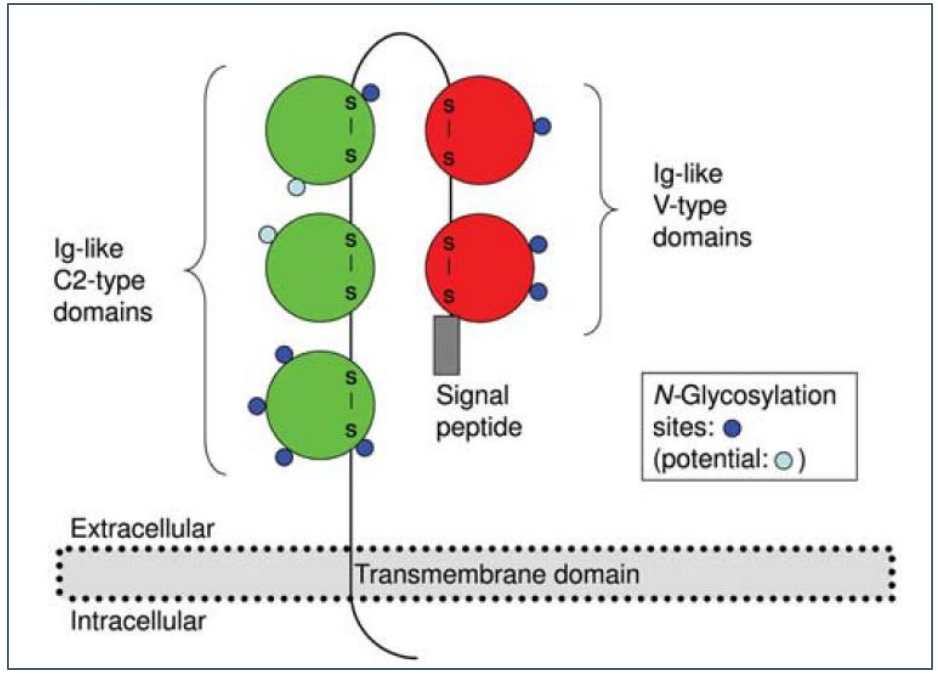
CX-2009:
A Probody Drug Conjugate (PDC) Targeting CD166

CD166 (ALCAM)

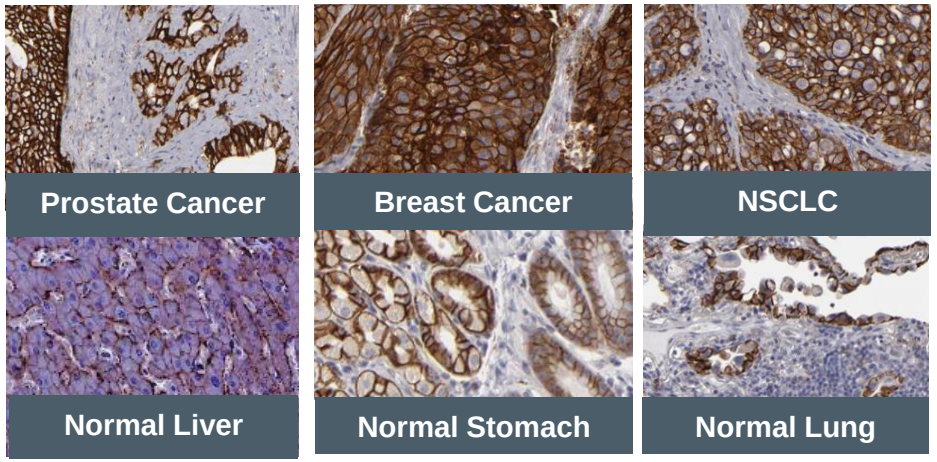
- Member of the immunoglobulin superfamily
- Role in cell adhesion, migration, T cell activation

- Efficiently internalizes cytotoxic payload
- Homogeneous, high expression in multiple cancers
- Also broadly expressed in normal tissues
  - By IHC, expression observed in every tissue tested except skeletal muscle
  - Strong to moderate staining of liver, kidney, lung, pancreas, stomach, small intestine, and colon
  - Staining pattern similar in cyno and human

- High risk target for a traditional ADC

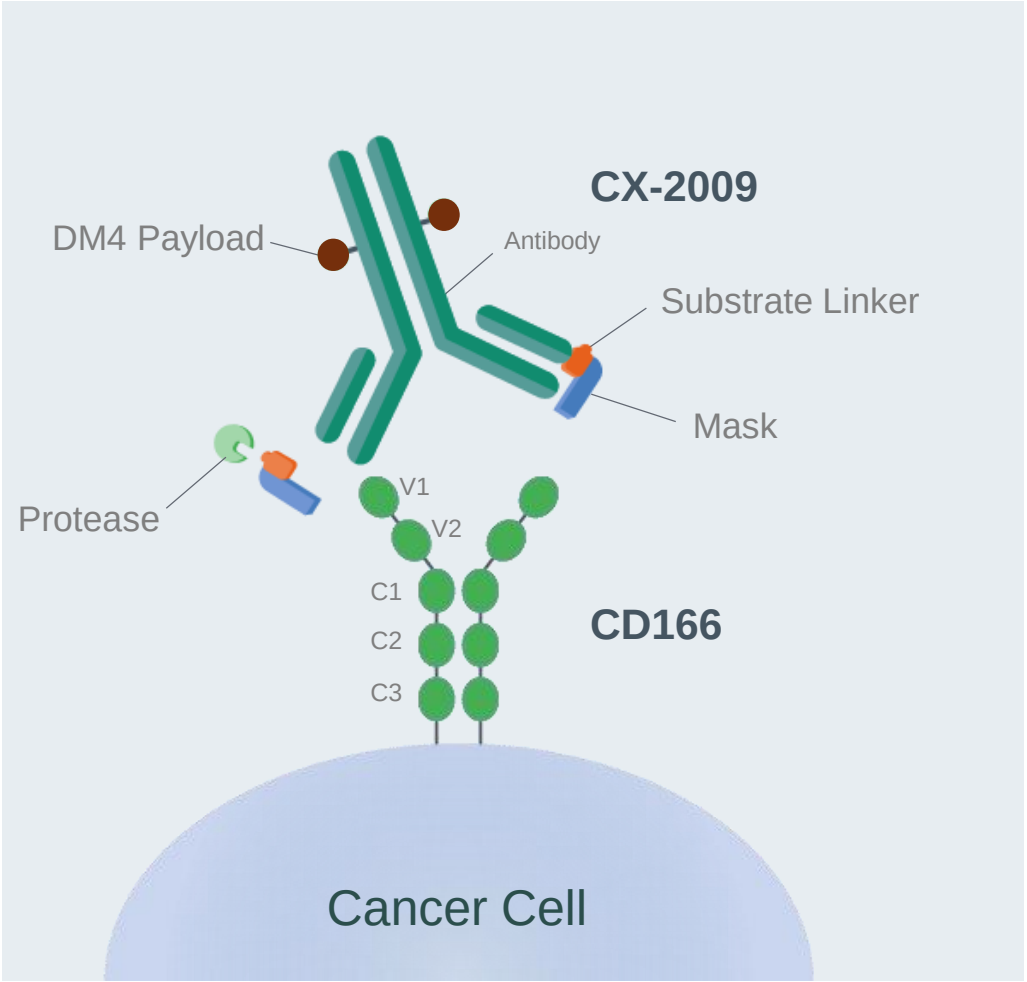


Weidle, Cancer Genomics Proteomics 7: 231-244 (2010)



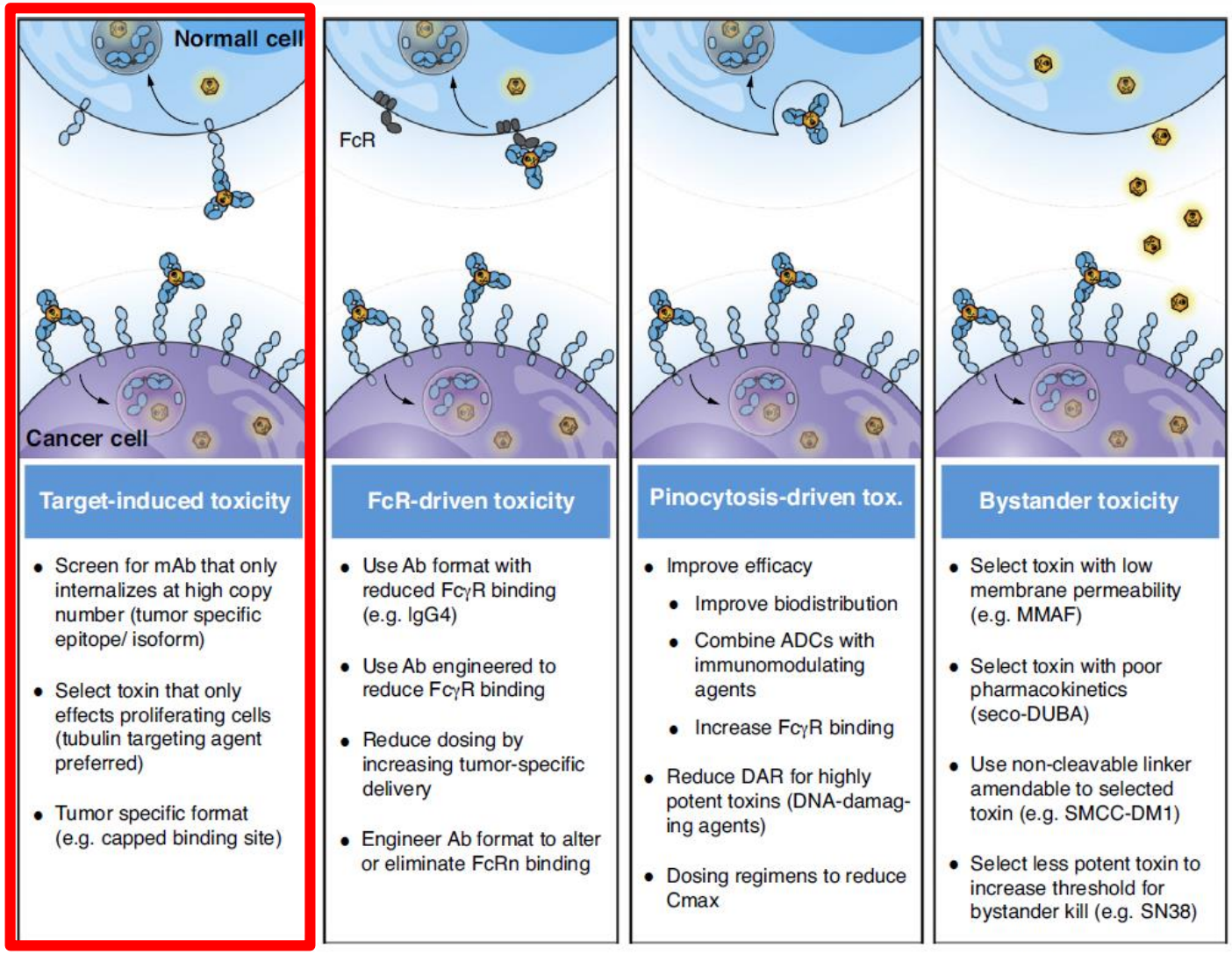
CX-2009: a Probody Drug Conjugate (PDC) targeting CD166

- Masked form of a proprietary anti-CD166 IgG1 antibody
- Linker-payload: SPDB-DM4 (DAR~3.5)
- Microtubule inhibitor (maytansinoid)
  - Well characterized off-target toxicities
    - Ocular toxicity
    - Peripheral neuropathy
- Linker-payload and conjugation technology licensed from ImmunoGen



Masking has Potential to Reduce On-Target Toxicity of Antibody Drug Conjugates

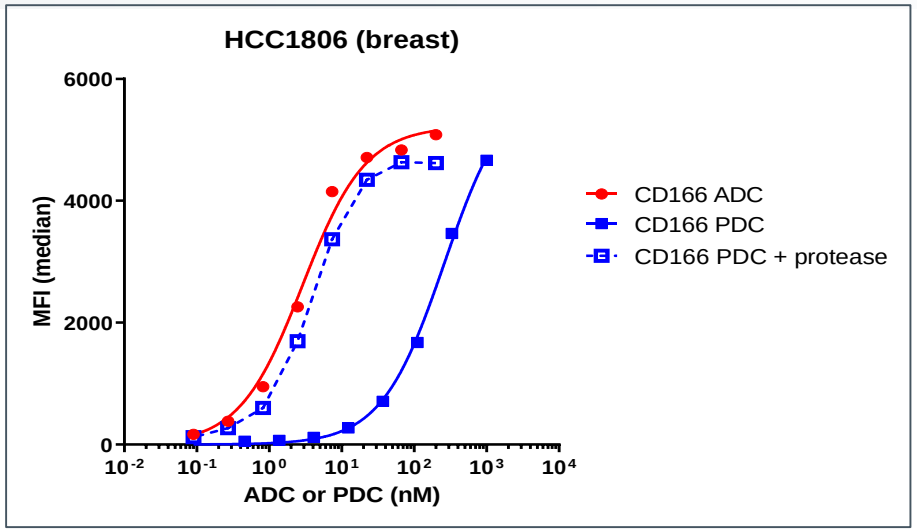
- Can create or widen a therapeutic window where on-target toxicity is dose limiting
- **No impact on off-target toxicities**



De Goeij and Lambert, 2016.Curr Opin Immunol 40:14-23

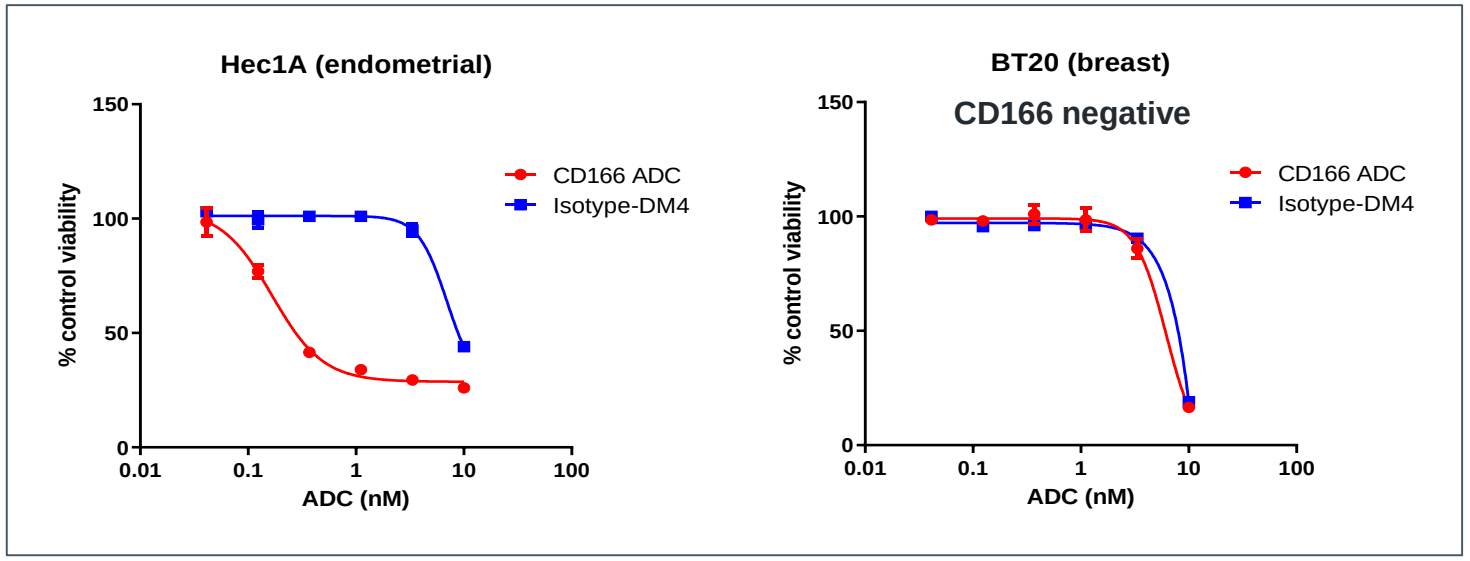
In Vitro Characterization of CX-2009

Binding

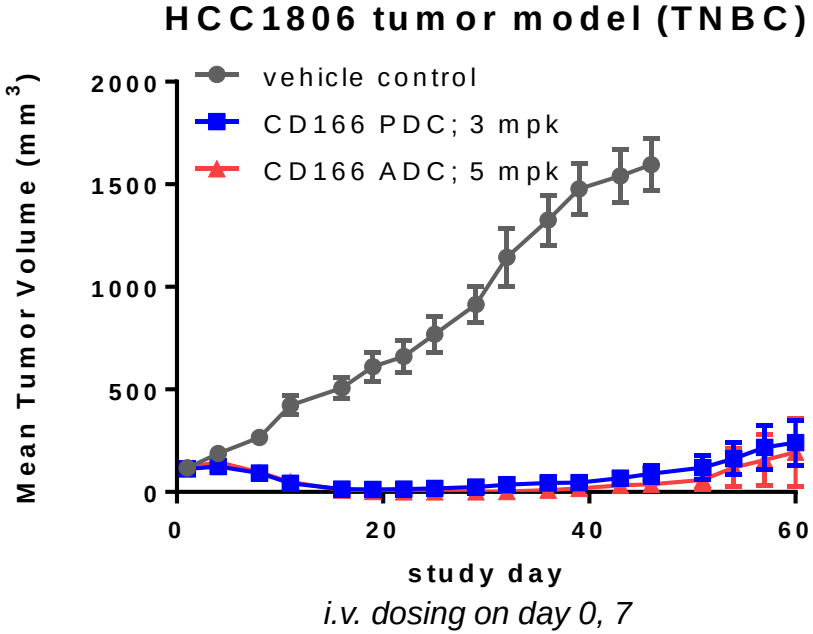


- Mask reduces binding to CD166
- In vitro protease activation of PDC (CX-2009) restores binding activity
- CD166 ADC shows potent and selective in vitro cytotoxicity

Cytotoxicity

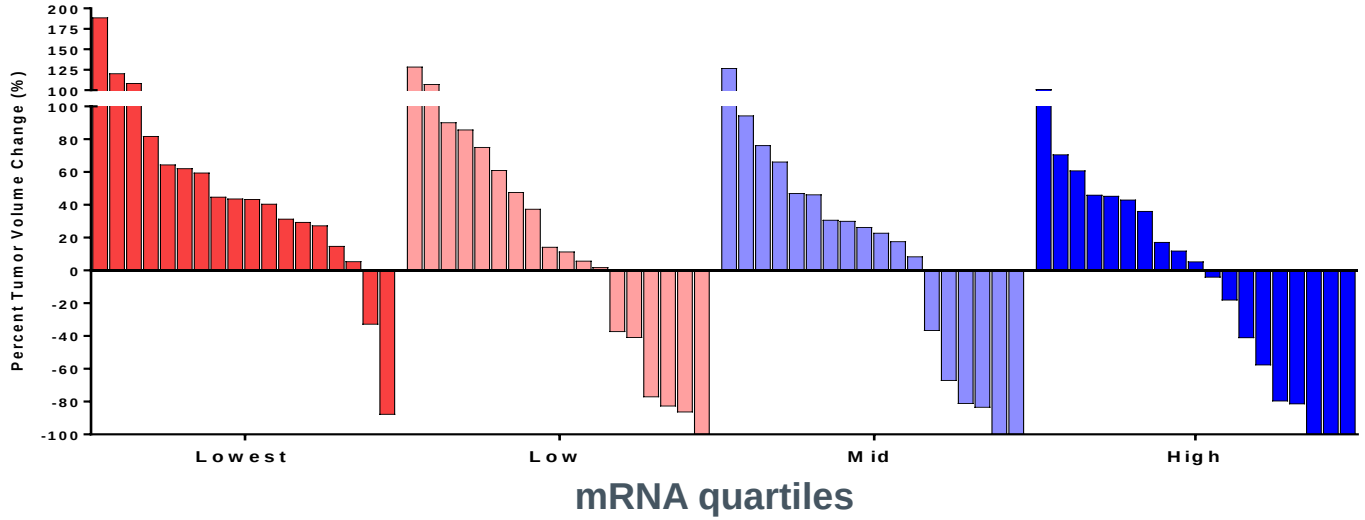


CX-2009 is Highly Active in Preclinical Tumor Models



i.v. dosing on day 0, 7

Activity in unselected PDX models is associated with high CD166 mRNA expression levels



Mouse clinical trial format, IV dosing 5 mg/kg q2wX3

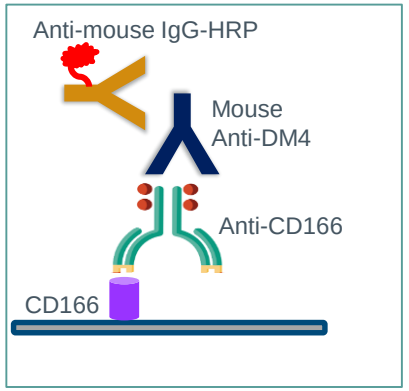
Liu, B, AACR Poster 2019

Nonclinical Safety Studies Supporting FIH Clinical Study of CX-2009

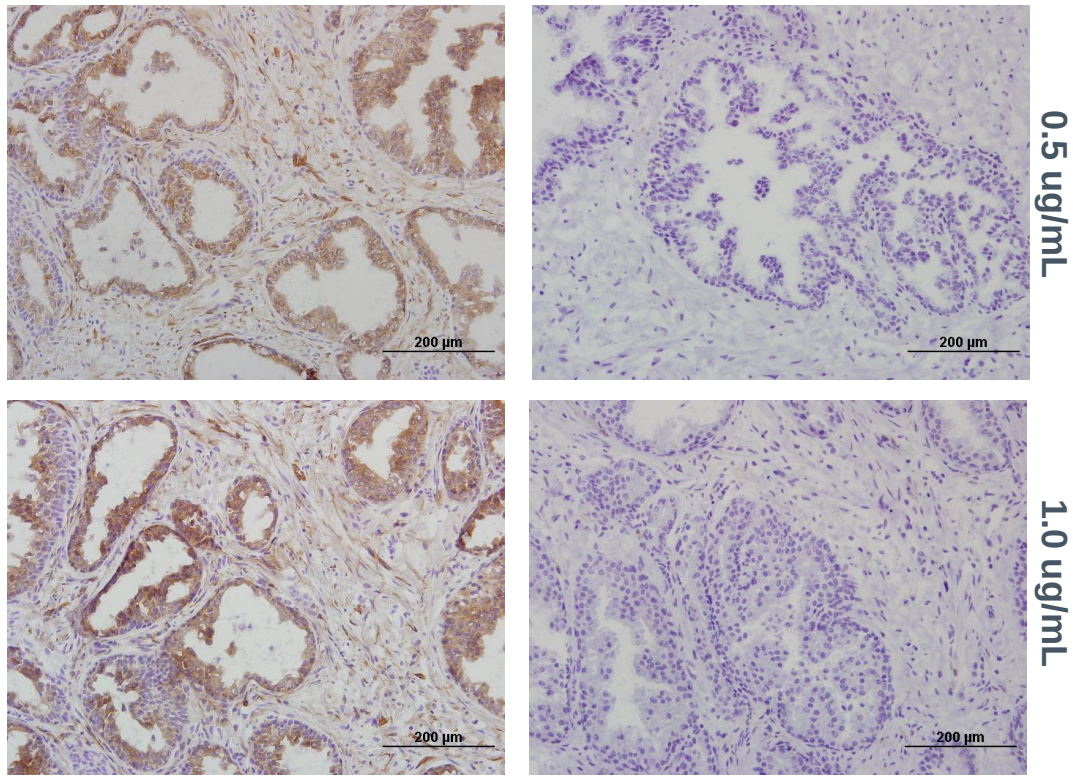
- Human tissue cross reactivity study (GLP)
- Repeat dose toxicity study of CX-2009 in cynomolgus monkeys (GLP)
- Supporting studies

Tissue Cross Reactivity Study of CX-2009 (GLP)

- 38 frozen human adult tissues were stained with
  - CX-2009
  - CD166 ADC
  - Control ADC
- Staining was performed at two concentrations, determined to be optimal and high for CD166 ADC
- Control tissues used for assay optimization were prostate (+) and heart (-)
- Assay format:

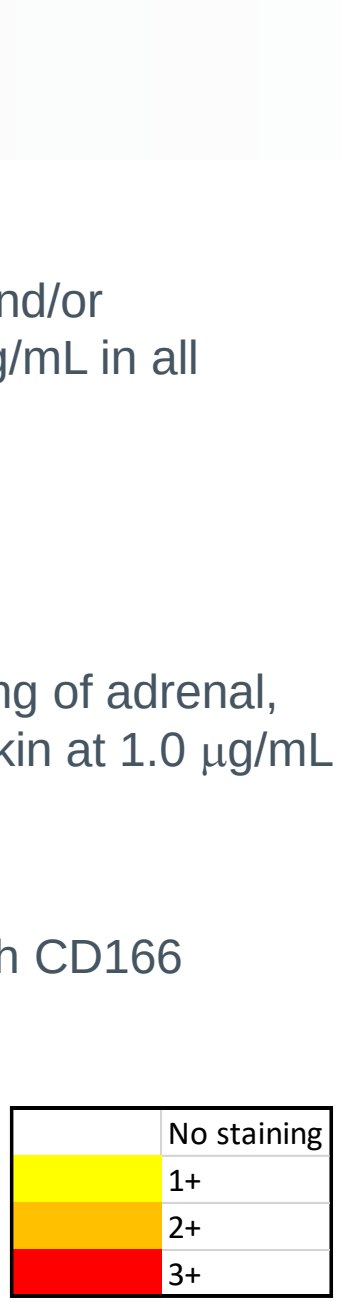


Positive Control Tissue: Human Prostate



Limited Tissue Binding of CX-2009

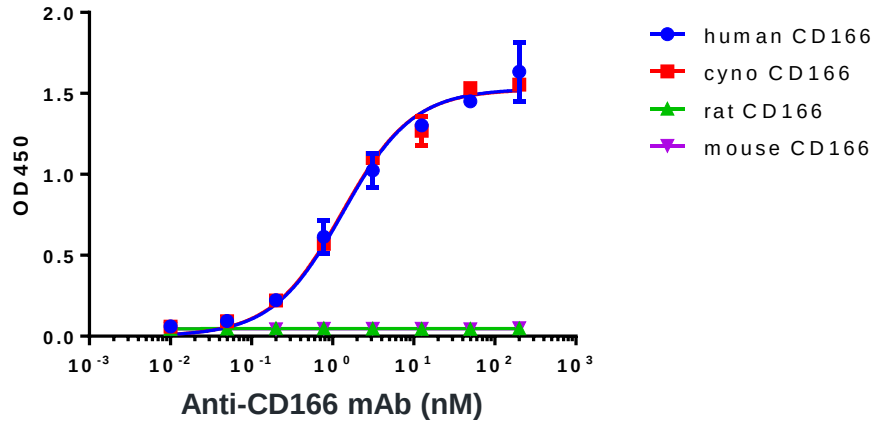
- CD166 ADC
  - Minimal (1+) to moderate (3+) intensity membrane and/or cytoplasmic staining observed at both 0.5 and 1.0 μg/mL in all tissues examined *except for* blood and bone marrow
- CX-2009
  - No binding to any tissue at the optimal concentration
  - Minimal (1+) to mild (2+) intensity cytoplasmic staining of adrenal, fallopian tube, lung, parathyroid, parotid gland and skin at 1.0 μg/mL
- Conclusions
  - Breadth and pattern of ADC binding is consistent with CD166 distribution (based on IHC data)
  - No tissues where PDC bound but ADC did not
  - CX-2009 is effectively masked



CytomX Therapeutics

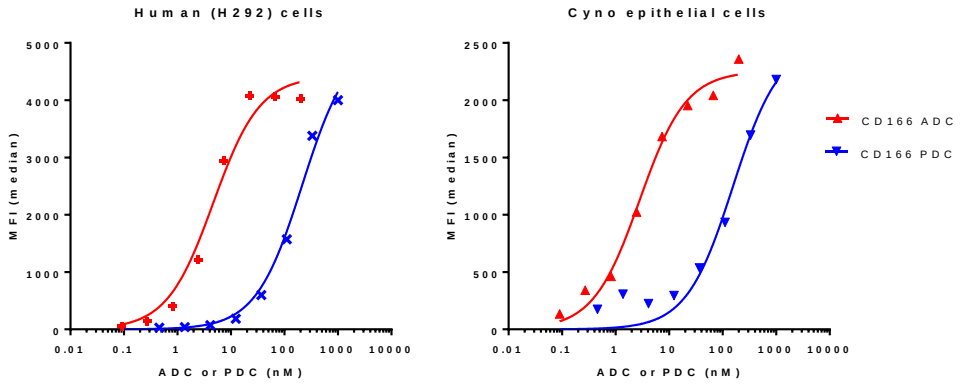
Considerations for Toxicity Species Selection

- Is binding affinity of activated Probody Tx or parental antibody to [species] and human target comparable?
- Is binding affinity of intact Probody Tx to [species] and human target similar?
- Is target expression similar in [species] and human tissues?
- Do I need to conduct my tox study in a tumor-bearing model?
- Are the proteases I care about conserved between [species] and human?



% identity (aa) to human CD166

Human	Cyno	Rat	mouse
100	98.6	93.4	93.2



Molecule	Apparent Kd (nM) Human	Apparent Kd (nM) Cyno
CD166 ADC	4.7	2.8
CD166 PDC	215	156
Ratio	46	56

2-Dose GLP Toxicity Study of CX-2009 in Cynomolgus Monkeys

Test Article	Dose (mg/kg)	Dose Day	Route	Main Nx (D26)	Recovery Nx (D43)
Vehicle	0	1, 22	IV bolus	3/3	2/2
CX-2009	5	1, 22	IV bolus	3/3	2/2
CX-2009	10	1, 22	IV bolus	3/3	2/2
CX-2009	15	1, 22	IV bolus	3/3	2/2

- Standard toxicity endpoints, safety pharmacology, toxicokinetics, immunogenicity

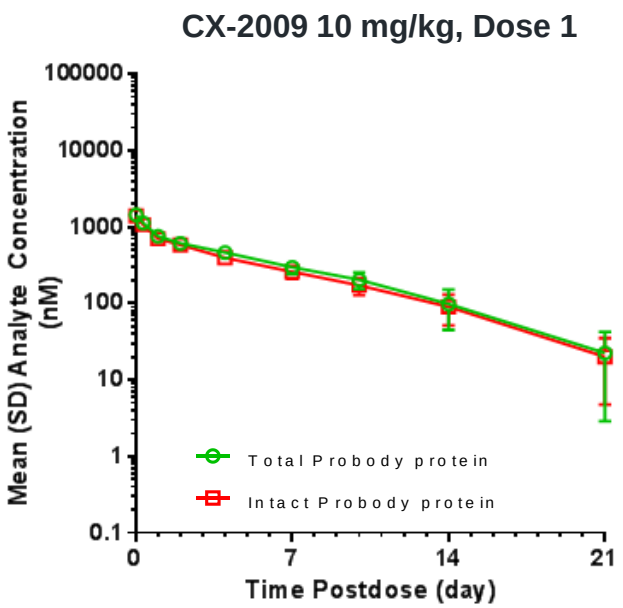
- 5 analytes selected for exposure analysis
  - Total Probody protein (intact + activated)
  - Intact Probody protein
  - Total Probody protein-conjugated DM4

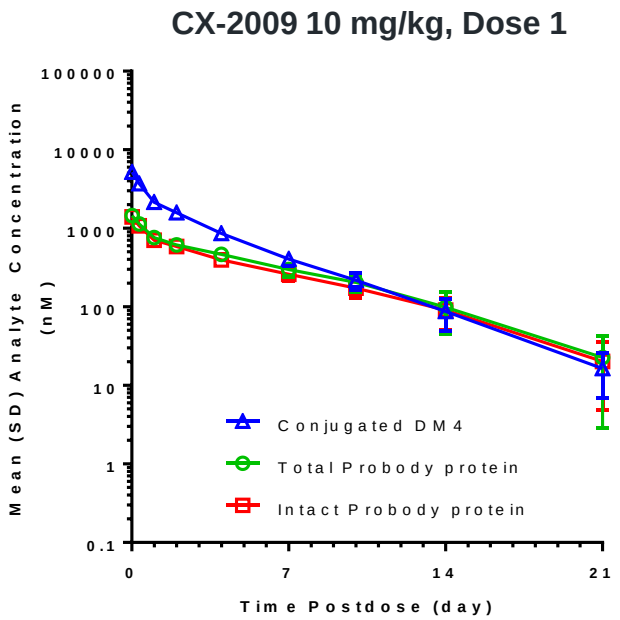
  } Affinity capture + LC-MS/MS

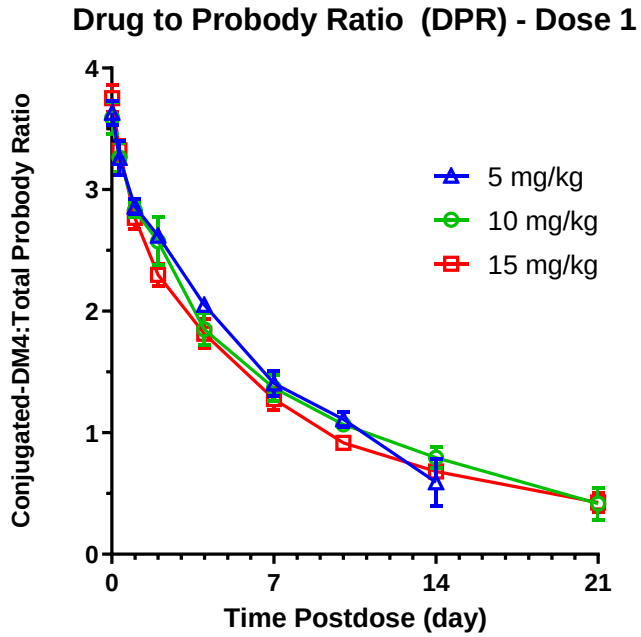
  - Unconjugated DM4
  - S-methyl-DM4 (DM4-Me)

  } LC-MS/MS

CX-2009 Circulates Predominantly as the Intact Species in Cynomolgus Monkeys







- Most of the CX-2009 exposure was due to Intact CX-2009 (84% to 99% based on mean $AUC_{0\text{-}21Day}$)
- Deconjugation occurs in a predictable manner
- Free DM4 and DM4-Me levels were low at all CX-2009 dose levels
- CX-2009 exposure increased in a dose proportional manner from 5 to 15 mg/kg

GLP Toxicity Study In-Life Findings

- Clinical observations
  - Hunched posture and decreased activity at 15 mg/kg; 2 females euthanized on Days 11 and 12
  - Skin changes (black, red, dry, ulcerative or flaking) at ≥ 5 mg/kg; severity/incidence increasing with dose
  - Transient infusion-type reactions

- Ophthalmic observations (Day 24)
  - Diffuse corneal pigmentation at ≥ 10 mg/kg, which did not resolve after a 3-week recovery

- Hematology
  - Transient decreases in most leukocyte subsets at ≥ 5 mg/kg
    - Marked neutropenia in some high dose animals
  - Decreased red blood cell mass + transient decrease in reticulocytes at ≥ 10 mg/kg

- Clinical chemistry and coagulation
  - Acute phase response (decreased albumin and increased globulins and/or fibrinogen) at 15 mg/kg
  - Increased alkaline phosphatase at 15 mg/kg

- Electrocardiology, respiration rate and neurological examination – no CX-2009 related changes

Histopathology Findings at Terminal Necropsy

- Neurons (≥ 5 mg/kg, non-reversing)
  - Axonal degeneration in spinal cord, dorsal root ganglion and sciatic nerve

- Hematologic tissues (≥10 mg/kg, reversible)
  - Bone marrow: decreased erythroid and myeloid cellularity
  - Lymph nodes, spleen, thymus, GALT: decreased lymphoid cellularity

- Epithelial tissues (≥10 mg/kg, reversible)
  - Corneal epithelium: Single cell necrosis and increased mitoses
  - Tongue: epidermal single cell necrosis, increased mitoses; neutrophil infiltrate
  - Skin: epidermal single cell necrosis, increased mitoses, hyperplasia; hyperkeratosis, epidermal/dermal ulceration, inflammation

- Pancreas (15 mg/kg, reversible)
  - Secretory (zymogen granule) depletion in exocrine pancreas

Similar findings in two 15 mg/kg animals euthanized on days 11/12

GLP Toxicity Study Summary

- CX-2009 was tolerated by cynomolgus monkeys at ≤ 10 mg/kg (2 doses, 3-week interval)
- TK analysis confirmed adequate exposure of CX-2009
- Findings in nerves, hematologic tissues and epithelial tissues are similar to those reported for other maytansinoid ADCs and are consistent with the activity of DM4
- No clear evidence of ‘on-target’ toxicity
- First human study of CX-2009 began in May 2017 with a starting dose of 0.25 mg/kg
  - 1/6 the lowest dose tested, adjusted for body surface area

PROCLAIM™ CX-2009

Phase 1 Dose Escalation and Next Steps

PHASE 1 DOSE ESCALATION

A: DOSE ESCALATION IN 7 TUMOR TYPES: 0.25-10 mg/kg

Advanced metastatic disease

A2: BIOPSY REQUIRED: 4–10 mg/kg

Advanced metastatic disease, CD166+++

Ongoing Dose Finding

COHORT EXPANSION

B: MULTIPLE CD166+++ TUMOR TYPES

Initiation: 2019

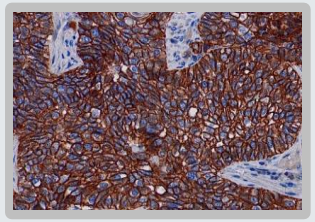
Dose refinement in CD166+++ patients

Enrollment completed | Enrollment ongoing | Trial not initiated

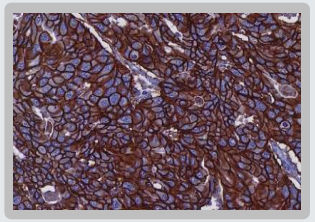
SEVEN TUMOR TYPES IN MONOTHERAPY DOSE ESCALATION ARM:

- Breast cancer
- Castration-resistant prostate cancer
- Cholangiocarcinoma
- Endometrial cancer
- Head and neck cancer
- Non-small cell lung cancer
- Ovarian cancer

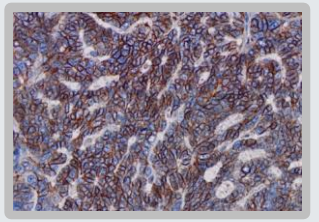
LUNG CANCER



BREAST CANCER



OVARIAN CANCER

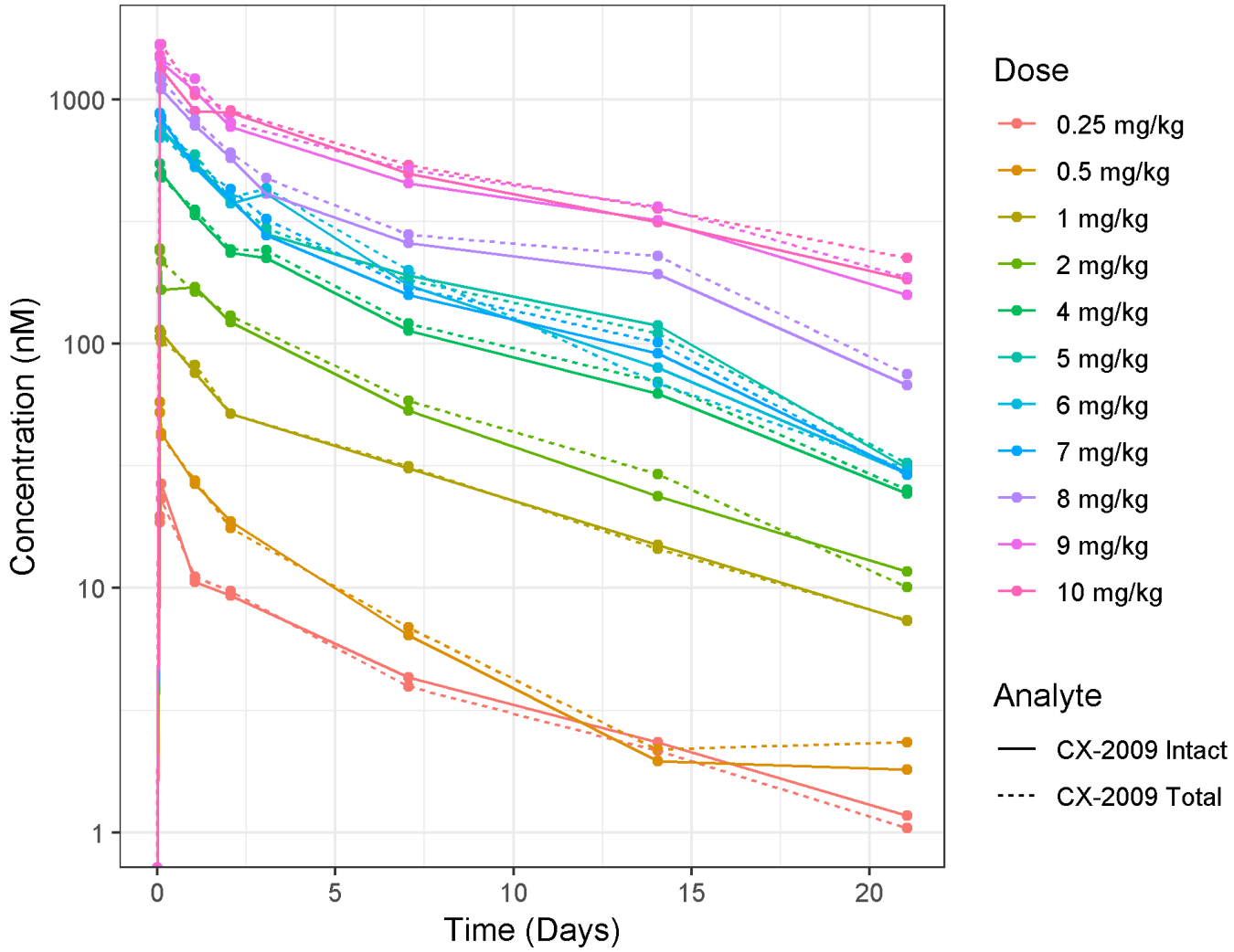


CytomX Therapeutics

PROCLAIM CX-2009

Phase 1 Dose Escalation: CX-2009 Remains Effectively Masked in the Circulation of Cancer Patients

- Single-dose CX-2009 PK data suggest that CX-2009 circulates predominantly as the intact prodrug species
- Consistent with prior findings for CX-072



CytomX Therapeutics

PROCLAIM CX-2009

CX-2009 Clinical Safety is Consistent with that of other DM4-ADCs

Not indicative of CD166-related toxicity

Table 4. Most Common TRAEs Leading to Permanent Discontinuation of Study Treatment

TRAE, n (%)	TRAEs Leading to Discontinuation (n=9)
Keratitis	6 (67)
Vision blurred	1 (11)
Peripheral neuropathy[a]	1 (11)
Nausea[b]	1 (11)

[a]Patient (4–5 mg/kg) had baseline neuropathy.
[b]Patient (6–7 mg/kg) had tumor-related small bowel obstruction.

Table 5. Most Common Grade 3+ TRAEs (≥2%; All Cohorts)

	CX-2009 Dose (mg/kg)					
TRAE, n (%)	<4 (n=10)	4–5 (n=19)	6–7 (n=18)	8–9 (n=23)	10 (n=8)	All Cohorts (n=78)
Keratitis	0	1 (5)	0	4 (17)[a]	1 (13)	6 (8)
Increased AST	0	0	0	1 (4)	3 (38)	4 (5)
Increased ALT	0	0	0	1 (4)	2 (25)	3 (4)
Nausea	0	0	1 (6)	2 (9)	1 (13)	4 (5)
Hyponatremia	0	0	2 (11)	1 (4)	0	3 (4)
Anemia	0	1 (5)	1 (6)	0	0	2 (3)
Fatigue	0	1 (5)	0	0	1 (13)	2 (3)
Peripheral sensory neuropathy	0	1 (5)	1 (6)	0	0	2 (3)
Vomiting	0	0	1 (6)	1 (4)	0	2 (3)

TRAEs, treatment-related adverse events; AST, aspartate aminotransferase; ALT, alanine aminotransferase.
[a]Including one patient with grade 4 keratitis.

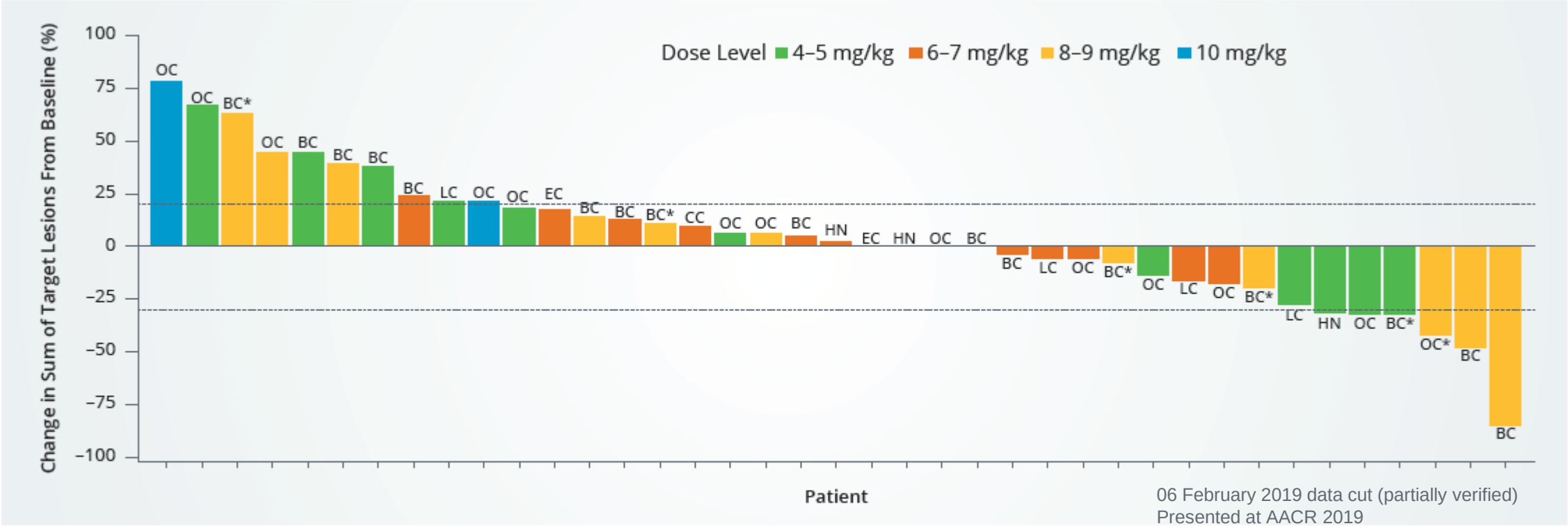
06 February 2019 data cut (partially verified)
Presented at AACR 2019

CytomX Therapeutics

PROCLAIM
CX-2009

CX-2009 is showing early evidence of biological activity in multiple cancer types at dose levels ≥ 4 mg/kg

Figure 3. Best Percent Change in Sum of Target Lesion Dimensions From Baseline (N=39)[a]



06 February 2019 data cut (partially verified)
Presented at AACR 2019

*Denotes patient considered to be on treatment, as no end-of-treatment date listed in database as of data cut-off date.
[a]CX-2009 4- to 10-mg/kg dose levels; response-evaluable population with post-baseline disease assessments.
Patients (n=9) who are evaluable for efficacy but are not presented in the figure as the magnitude of tumor burden change was not in the database at the time of data cut-off. Patients (n=2) with non-measurable disease at baseline who are evaluable for efficacy are not included in the figure. Patients (n=3) with one evaluable post-baseline tumor scan <7 weeks from treatment start assessed as stable disease will be considered to have best overall response of *not evaluable*.
BC=breast carcinoma; LC=non-small cell lung carcinoma; OC=epithelial ovarian carcinoma; EC=endometrial carcinoma; HN=head and neck squamous cell carcinoma; CC=cholangiocarcinoma.

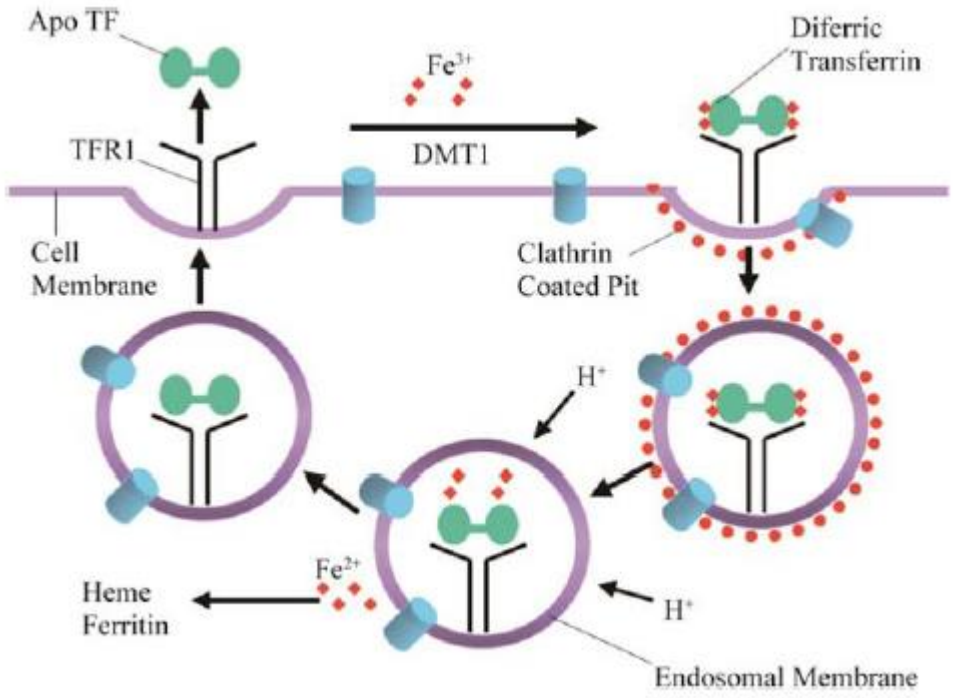
CX-2009 Summary and Conclusions

- CX-2009 is a first-in class protease-activatable PDC targeting a highly expressed antigen, CD166
- CX-2009 shows potent activity in mouse tumor models, response rate correlating with CD166 expression
- A cynomolgus monkey GLP toxicity study revealed no clear evidence of on-target toxicity
  - Target tissues (epithelial, hematologic, neuronal) are consistent with known toxicities of DM4
- In a FIH Phase 1 study, CX-2009 is generally well tolerated with early evidence of biological activity in multiple cancer types over a wide range doses (4–10 mg/kg) in a heavily pretreated population
- Treatment-related adverse events observed in the clinic are consistent with known target tissues of DM4
  - DM4-associated ocular toxicity is the most common grade 3+ TRAE, leading to dose delays and early discontinuation in some patients.
- In both cynomolgus monkeys and cancer patients, CX-2009 circulates predominantly as the intact species

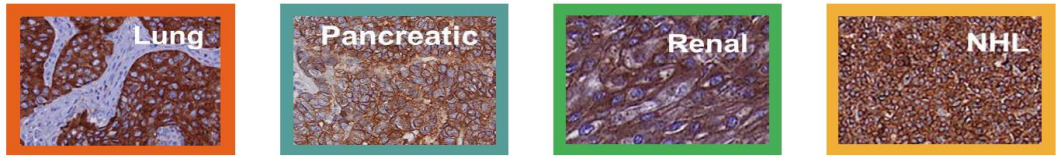
CX-2029:
A Probody Drug Conjugate (PDC) Targeting CD71, Transferrin Receptor

CD71 (TfR1) Transferrin Receptor

- Transmembrane glycoprotein that efficiently internalizes iron-bound transferrin
- Highly expressed in malignant cells
- ‘Professional’ internalizer
- Also expressed in healthy tissues with high iron requirement, notably
  - Dividing cells
  - Erythrocyte precursors
- Difficult to drug with current ADC technology



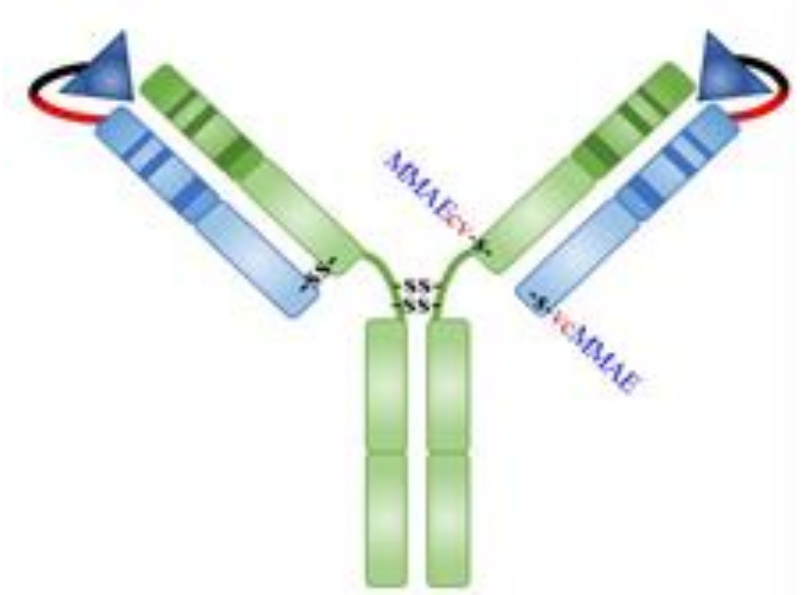
Elliott & Head, J Cancer Therapy, 3: 278-311 (2012)



CX-2029: CD71 Targeting PDC

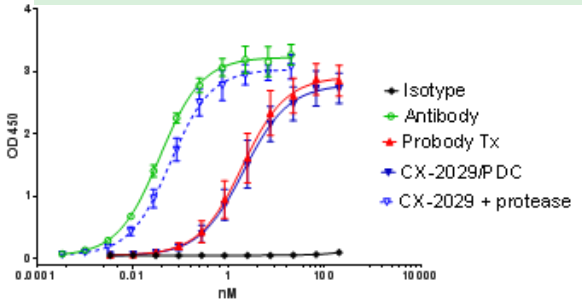
- Masked form of a proprietary anti-CD71 antibody (humanized IgG1)

- Linker-payload: val-cit-MMAE (DAR 2, purified)
  - Microtubule inhibitor
  - Well characterized toxicity profile
    - Peripheral neuropathy (human)
    - Neutropenia

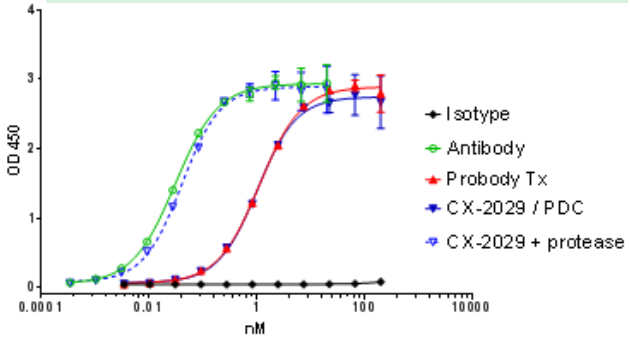


CX-2029 is Highly Active in CDX and PDX Tumor Models in Mice

Human CD71



Cynomolgus CD71

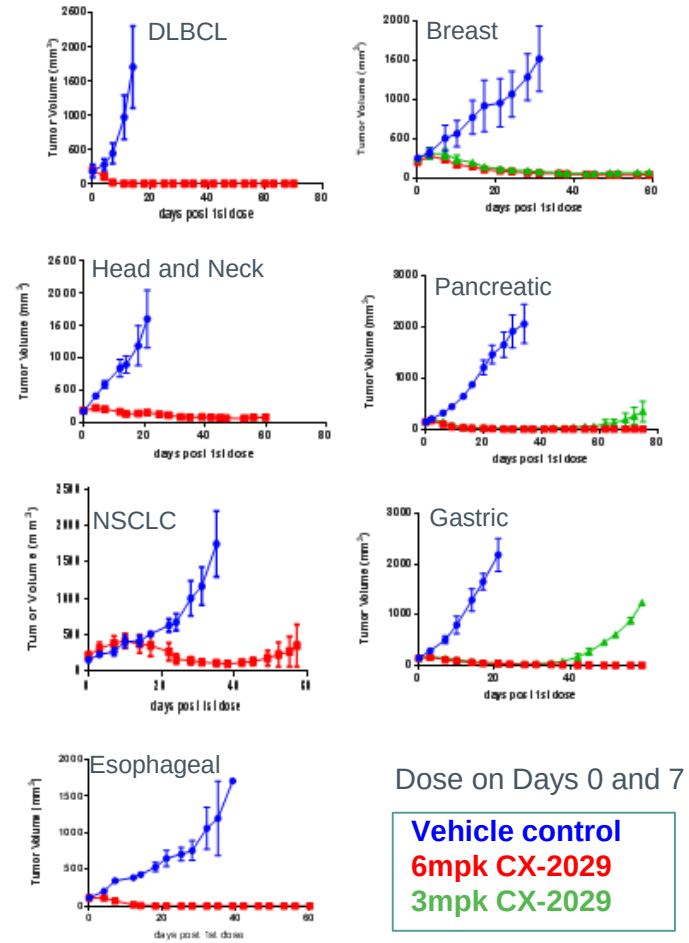


CDX - cell line derived xenograft
PDX - patient derived xenograft

- Parental anti-CD71 antibody binds equivalently to human and monkey CD71 (ELISA)
- Probody therapeutic shows reduced binding to CD71
- Protease activation of PDC restores binding activity
- Broad, potent activity in mouse tumor models

Model Type	Regressions or Stasis
CDX (unselected)	15/21 (71%)
PDX (high expressing)	30/36 (83%)

Representative PDX Models



Dose on Days 0 and 7

Vehicle control
6mpk CX-2029
3mpk CX-2029

CytomX Therapeutics abbvie

Dose Range Finding Study of CX-2029 and CD71 ADC (both DAR2)

Test Article*	Dose (mg/kg)	Dose Day	# Animals	Necropsy Day	Neutrophils*
Vehicle	NA	1, 22	3	29	4,693 ± 2,284
CX-2029 (PDC)	6	1, 22	3	29	347 ± 169
CX-2029 (PDC)	12	1, 22	3	29	87 ± 90
CX-2029 (PDC)	18	1	3	11 (unsc), 15 (unsc), 22	20 ± 10
CX-2030 (ADC)	6	1	1	10 (unsc)	20 (D10)
CX-2030 (ADC)	2	1	1	9 (FD)	70 (D7)
CX-2030 (ADC)	0.6	1, 22	2	29	280 ± 42

*Average Neutrophil count +/- SD (per ul) on Day 11 or as indicated

- Masking improved tolerability: CX-2029 was tolerated at a 10-fold higher dose than CX-2030
  - Hunched posture, decreased activity, and body temperature elevation at non-tolerated dose levels
  - Decreased cellularity in bone marrow and lymphoid tissues, with corresponding reductions in red blood cell mass and all leukocyte populations
  - Mortality attributed to secondary infection

CX-2029 Summary and Conclusions

- CX-2029 is a protease-activatable PDC, targeting the transferrin receptor CD71
- Robust efficacy is observed in mouse CDX and PDX models representing multiple cancer indications
- In cynomolgus monkeys, CX-2029 is tolerated at a 10-fold higher dose than the corresponding ADC
  - HNSTD 6 mg/kg for PDC vs 0.6 mg/kg for ADC
  - Hematologic toxicity is dose limiting
- CX-2029 FIH trial was initiated in June 2018 (NCT03543813)

Overall Summary and Conclusions

- Probody technology offers a promising approach to avoidance of on-target toxicity of ADCs
  - New target space: antigens with broad / high normal tissue expression
- Masking cannot reduce off-target toxicity
  - Coupling with advancements in linker/payload technology may maximize therapeutic window
- IND-enabling studies are similar to those for a conventional mAb or ADC
  - Additional considerations for species selection - mask strength
- Bioanalysis is more complex than for antibody therapeutics
  - Additional analytes (intact and activated proteins)
  - Novel approaches to assess Probody therapeutic activation in disease tissue

Acknowledgments

CX-2009

Pharm/tox:
Annie Yang
Amy DuPage
Michael Krimm
Luc Desnoyers
Jennifer Richardson
W. Michael Kavanaugh

Bioanalysis:
Laura Serwer
Hong Lu
William Mylott Jr.

Clinical Development:
Matthias Will
Sreeni Yalamanchili
Pratigya Gautam
Annie Weaver
Rachel Li

Clinical Pharmacology:
Mark Stroh

Protein Sciences:
Yuanhui Huang
Andrew Jang
Eric Ureno
Shouchun Liu

CMC:
Eric Ureno
Adam Miller
Sarah Patrick
Shanti Duvur
Claus Krebber
Sridhar Viswanathan
Marc Besman

ImmunoGen:
Rachael Susser
Godphrey Amphlett
Sonia Connaughton
Michelle Palmer
Gillian Payne

CX-2029

Pharm/tox:
Shweta Singh
Laura Serwer
Tracy Henriques
Amy DuPage
Luc Desnoyers
Michael Krimm
Ken Wong

Protein Sciences:
Shouchun Liu
Yuanhui Huang
Andrew Jang

Conjugation:
Niharika Chauhan
Fritz Buchanan
Matthew Ravn
Rob Leanna

CMC:
Eric Ureno
Adam Miller
Sarah Patrick
Shanti Duvur
Claus Krebber
Sridhar Viswanathan

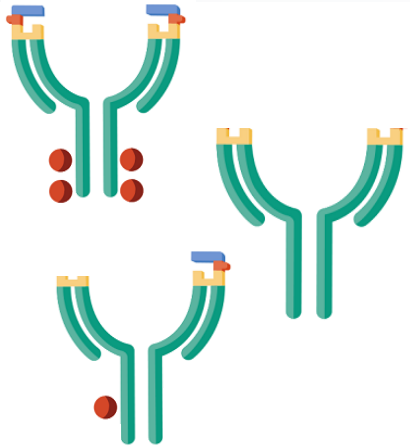
Research leadership:
Susan Morgan-Lappe
W. Michael Kavanaugh

Clinical Investigators
Patients and families

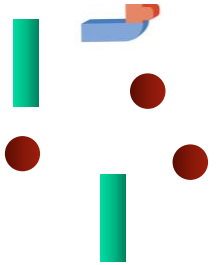
Hybrid Affinity Capture + LC-MS/MS Assay for CX-2009

Cynomolgus monkey plasma

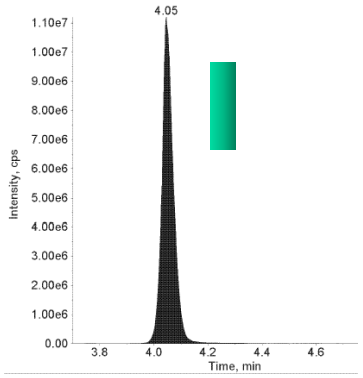
Affinity capture (protein A)



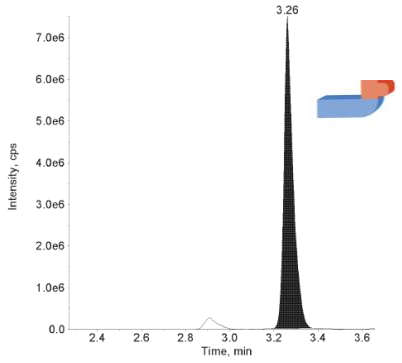
Denaturation
reduction
alkylation
trypsin digestion



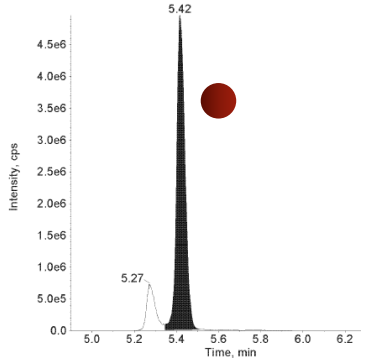
LC-MS/MS



Non-prodomain peptide
(Total Probody protein)

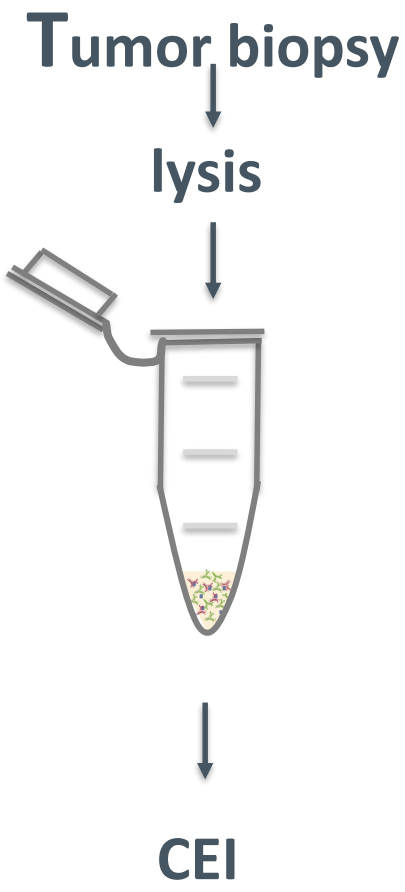


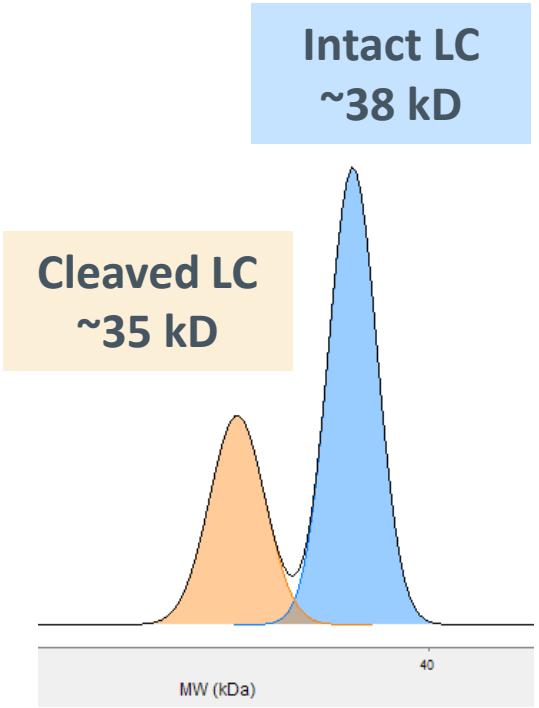
Prodomain peptide
(Intact Probody protein)



DM4
(Conjugated DM4)

Capillary Electrophoresis Immunoassay (CEI) for the Quantification of Cleaved and Intact Light Chains from a Probody Therapeutic in Patient Tumor Biopsies

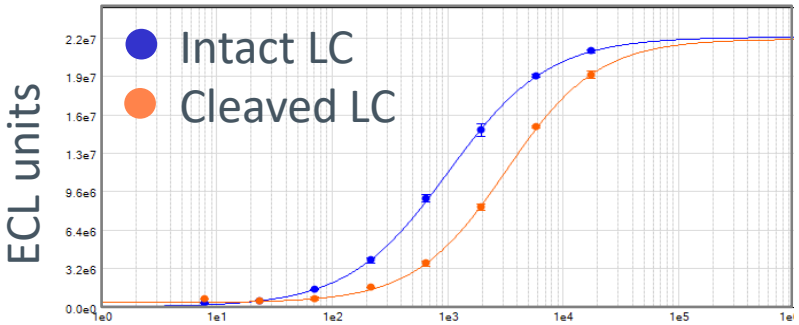




Intact & cleaved CX-072 LC are detected using a custom anti-idiotypic antibody*

* Data shown are for illustration only

Standard curve

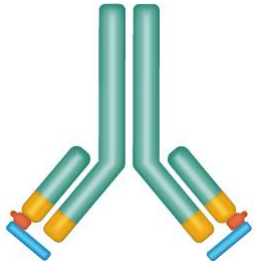
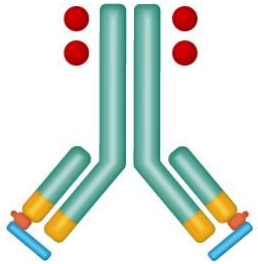
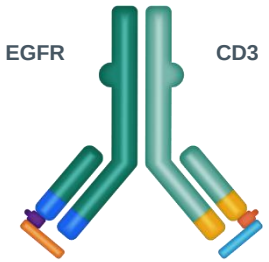
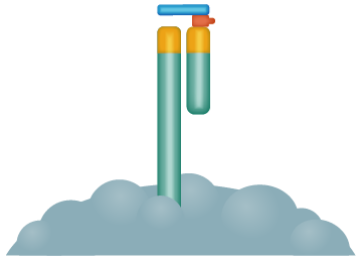
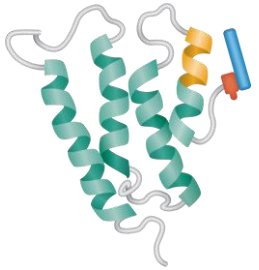


CX-072 LC, ng/ml

Interpolate tumor samples vs. standard curve to quantify levels of intact and active (cleaved) LC in sample

CytomX Therapeutics

Probody Platform is Applicable Across Multiple Modalities

ANTIBODIES	ANTIBODY DRUG CONJUGATES	T-CELL BISPECIFICS	CARS	CYTOKINES
				
PD-L1 (CX-072) CTLA-4 (BMS-986249) PD-1 (CX-188)	CD166 (CX-2009) CD71 (CX-2029) Multiple undisclosed targets	EGFR-CD3 Multiple undisclosed targets	Discovery Stage	Discovery Stage

Plasma Concentrations of Unconjugated DM4 and DM4-Me are Low