Harnessing the power of the immune system to improve lives and eradicate cancer

Nasdaq: BOLT

May 2026

BOLT BIOTHERAPEUTICS

Disclaimer

This presentation contains forward-looking statements that involve substantial risks and uncertainties. All statements other than statements of historical facts contained in this presentation, including statements regarding Bolt Biotherapeutics, Inc. (the "Company," "we," "us," or "our")'s future financial condition, ability to achieve upcoming milestones for our product candidates, the timing of our clinical trials, and the success and results of our pipeline programs and partnerships, are forward-looking statements. In some cases, you can identify forward-looking statements by terminology such as "anticipate," "believe," "continue," "could," "estimate," "expect," "intend," "may," "plan," "potentially" "predict," "should," "will" or the negative of these terms or other similar expressions. We have based these forward-looking statements largely on our current expectations and projections about future events and trends that we believe may affect our financial condition, results of operations, business strategy, and financial needs. These forward-looking statements are subject to a number of risks, uncertainties and assumptions, including, among other things: the advancement and success of our clinical trial for BDC-4182, the viability of the Boltbody™ ISAC platform, the anti-tumor potency, safety and tolerability, and characteristics of our product candidates, the success, cost and timing of our product development activities and clinical trials; our expectations about the timing of achieving regulatory approval and the cost of our development programs; our ability to obtain funding for our operations, including funding necessary to complete further development and commercialization of our product candidates; our ability to fund our clinical programs and the sufficiency of our cash, cash equivalents, and marketable securities to fund operations through key milestones and the achievement of key milestones; the commercialization of our product candidates, if approved; our plans to research, develop, and commercialize our product candidates; our ability to attract collaborators with development, regulatory and commercialization expertise; future agreements with third parties in connection with the commercialization of our product candidates; the success of our current collaborations with third parties, including our collaborations with Genmab A/S and Toray Industries, Inc.; the achievement of milestone payments or any tiered royalties related to our collaborations; the size and growth potential of the markets for our product candidates, and our ability to serve those markets; the rate and degree of market acceptance of our product candidates; and regulatory developments in the United States and foreign countries. These risks are not exhaustive. For a detailed discussion of the risk factors that could affect our actual results, please refer to the risk factors identified in our SEC reports, including, but not limited to our Annual Report on Form 10-K for the year ended December 31, 2025. New risk factors emerge from time to time and it is not possible for our management to predict all risk factors, nor can we assess the impact of all factors on our business or the extent to which any factor, or combination of factors, may cause actual results to differ materially from those contained in, or implied by, any forward- looking statements. You should not rely upon forward-looking statements as predictions of future events. Although we believe that the expectations reflected in the forward-looking statements are reasonable, we cannot guarantee future results, levels of activity, performance or achievements. Except as required by law, we undertake no obligation to update publicly any forward-looking statements for any reason after the date of this presentation.

In addition, statements that "we believe" and similar statements reflect our beliefs and opinions on the relevant subject. These statements are based upon information available to us as of the date of this presentation, and while we believe such information forms a reasonable basis for such statements, such information may be limited or incomplete, and our statements should not be read to indicate that we have conducted an exhaustive inquiry into, or review of, all potentially available relevant information. These statements are inherently uncertain and investors are cautioned not to unduly rely upon these statements.

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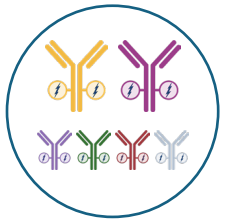
BOLT is Pioneering a New Approach to Cancer Treatment

BDC-4182 will provide proof-of-concept for the ISAC approach

BDC-4182 (claudin 18.2 ISAC) Phase 1/2 clinical trial ongoing

- First-in-class, best-in-class Immune Stimulating Antibody Conjugate (ISAC)
- Initial clinical data from >10 patients at therapeutically relevant dose levels expected in 3Q26



ISAC proof-of-concept should have a substantial multiplier effect

- ISACs have potential to combine the power of ADCs with the durability of T cell engagers
- Industry is hungry for ADC payloads beyond Topo1 to overcome resistance issues
- Pipeline of Boltbody™ ISACs (CEA, PD-L1, Genmab & Toray collaboration programs)

Operating runway into 2027[1]

- BDC-4182 initial clinical data (3Q26) expected to be a key inflection point

[1] Cash, cash equivalents, and marketable securities balance of $23.9 million as of 3/31/2026

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Boltbody™ ISAC Approach Improves on ADCs and TCEs

	Durability	Bystander	Immune Activation	Killing Mechanism	Resistance
Boltbody™ ISACs	High	High	High	T cells + Phagocytes	-
Naked mAbs	Low/Med	-	Low	ADCC & CDC	Low antigen expression
T cell Engagers	Med	Low/Med	Med	T cells	Exhaustion & Exclusion
ADCs	Low	Med	Low/Med	Chemotherapy	Efflux & Payload Resistance

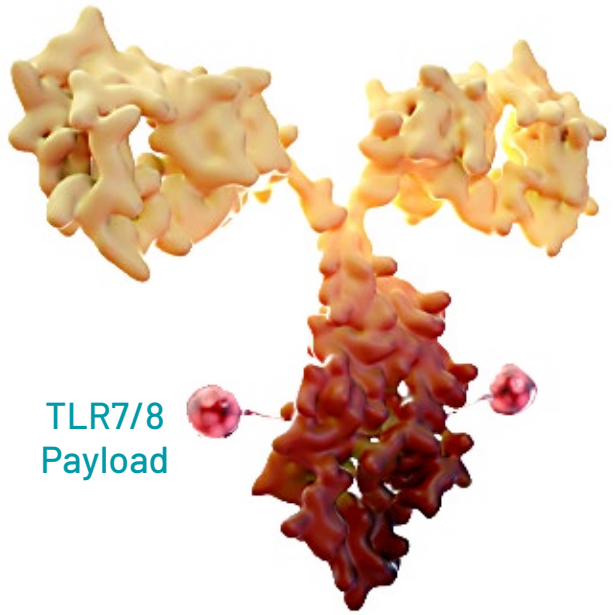
Lower — Higher

Bolt's ISACs broadly activate the immune system to enable durable responses and killing of antigen-negative tumors

BDC-4182: Claudin 18.2 Boltbody™ ISAC Program

Phase 1/2 clinical trial ongoing, initial results expected 3Q26

BDC-4182



Next-Generation Claudin 18.2 ISAC

BDC-4182 Opportunity

- Phase 1/2 clinical trial ongoing for best-in-class claudin 18.2 ISAC
- Claudin 18.2 is a clinically validated target in gastric cancer
- This is a large market with significant unmet need

Key Attributes

- ISAC mechanism combines power of ADCs with durability of TCEs
- Immunological memory should improve durability
- Activity in low-antigen-density tumors could expand the market

Preclinical Differentiation in Competitive Claudin 18.2 Landscape

- Superior efficacy versus TOPO1 and MMAE-ADCs in multiple experiments
- Epitope spreading to also kill tumors that no longer express claudin 18.2
- Potential for better tolerability than other claudin-18.2-targeting agents

Preclinical data published in Fu CL et al. SITC 2025 and Fu CL et al. SITC 2024

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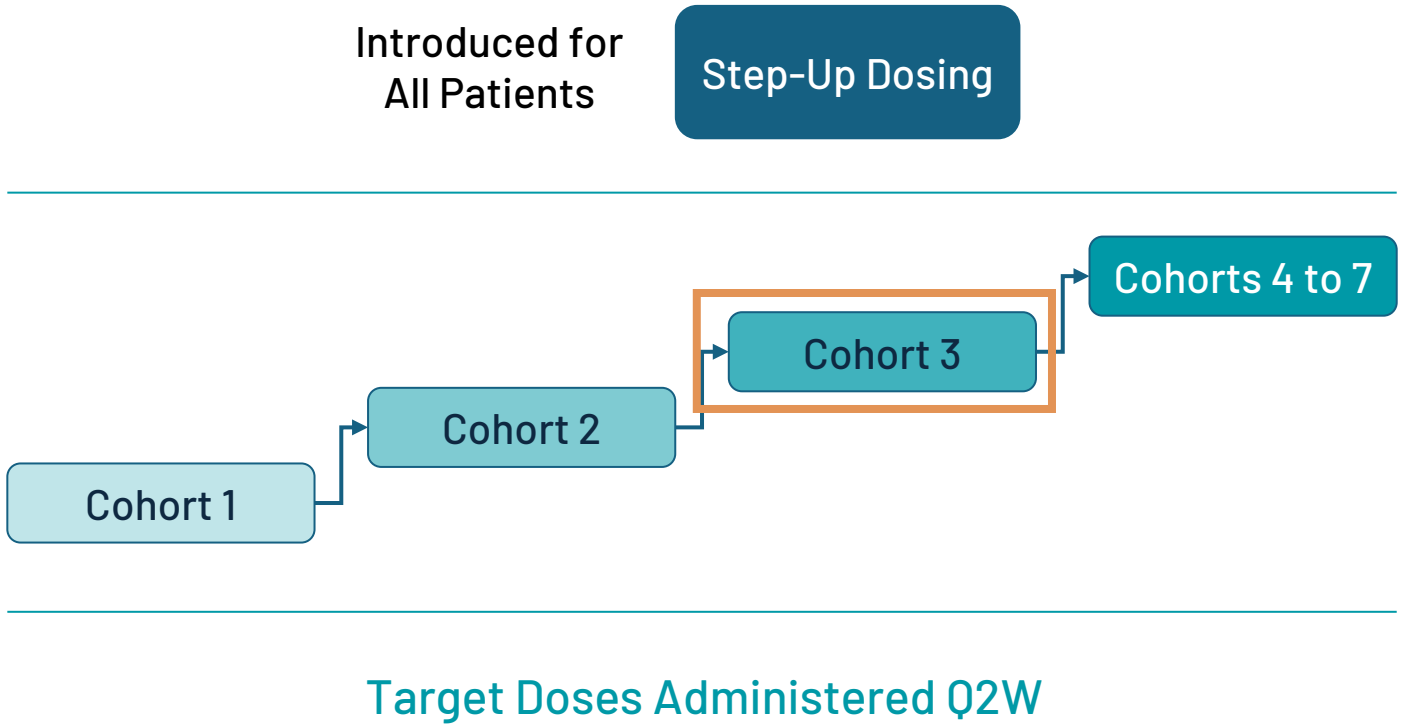
BDC-4182 Phase 1/2 Clinical Trial in 2L+ Gastric or Gastroesophageal Cancer

Dose Escalation with Cycle 1 Step-Up Dosing



- Open for enrollment in Australia, South Korea, and Taiwan (NCT06921837)
- CLDN18.2 IHC 2+ ≥ 1%

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Gastric Cancer Opportunity

May 2026

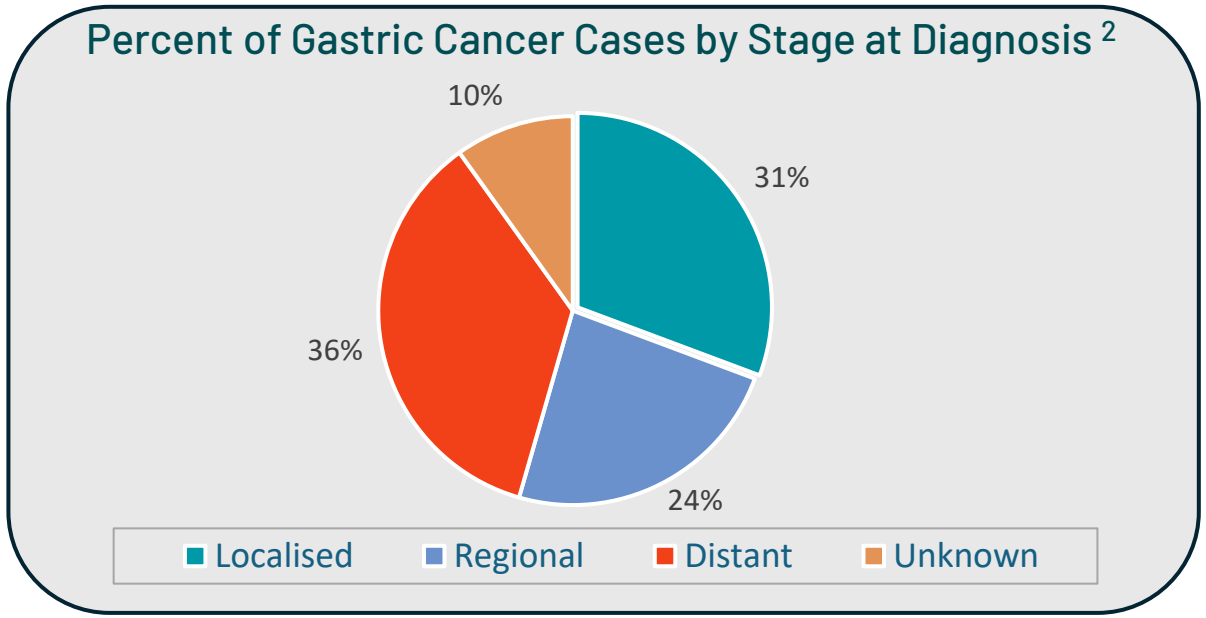
Gastric Cancer is Common and Difficult to Treat

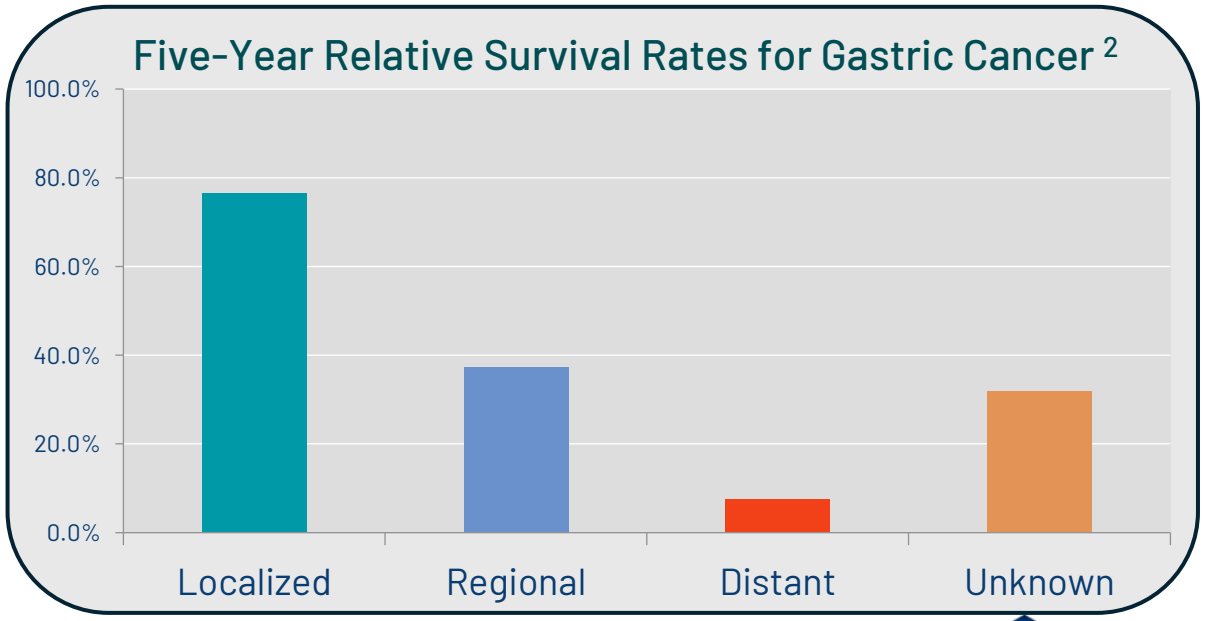
Gastric cancer is the 5th most common malignancy worldwide, with one million new cases annually[1]

- More than 30,000 new cases and 10,000 deaths per year In the US [1,2]

More than 50% of patients have advanced, incurable disease upon diagnosis[3,4]

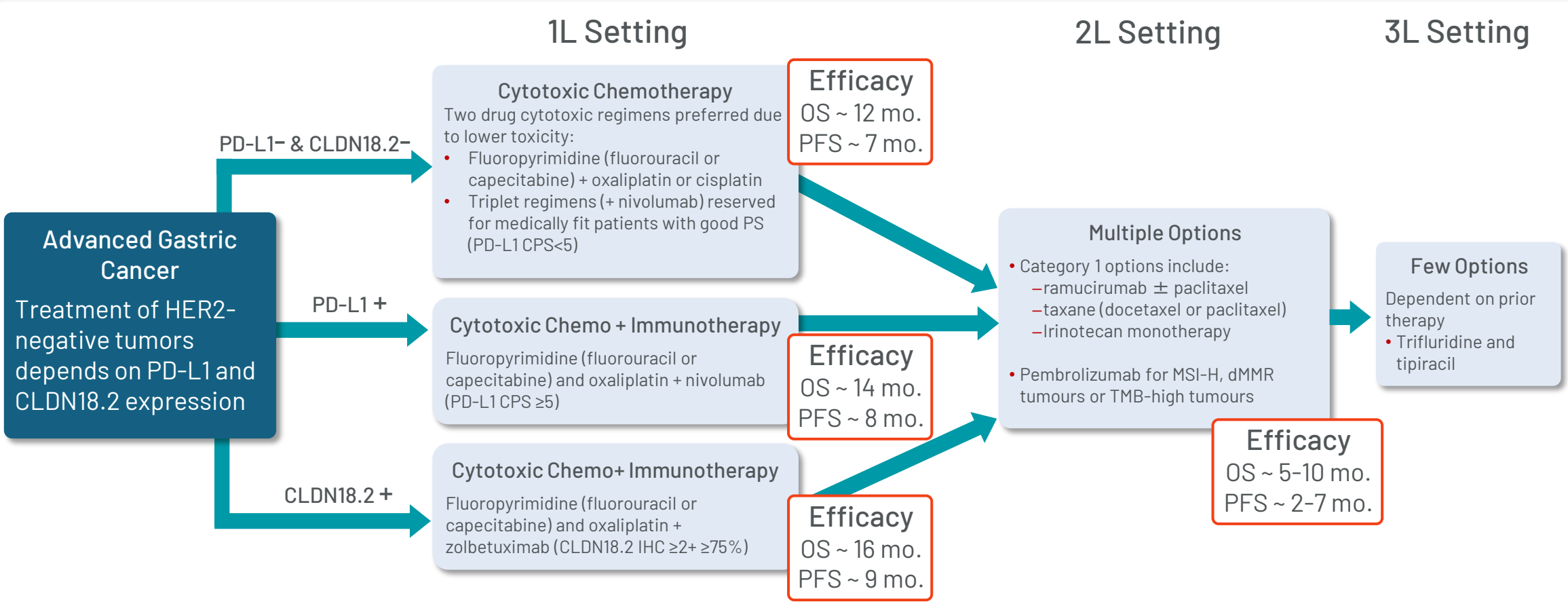
- Gastric adenocarcinomas typically develop slowly with early changes rarely causing symptoms
- Only 3 of 10 patients have localized disease at diagnosis





Patients with Metastatic/Unresectable Gastric Cancer have Few Options

Average progression-free survival in 2L setting is 2-7 months



Source: NCCN Guidelines Gastric Cancer, Version 2. 2025. Abbreviations: FOLFOX – Fluorouracil (generally with Leucovarin) + oxaliplatin; CAPOX – capecitabine + oxaliplatin. MSI-H – Microsatellite instability-high; dMMR – DNA mismatch repair; TMB – Tumour mutational burden. Note: zolbetuximab efficacy based on final pooled analysis; 2L efficacy based on ramucirumab monotherapy and combination data

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Claudin 18.2: Validated Target with Opportunity to Improve on Zolbetuximab

BDC-4182 has potential to improve depth and duration of response in large market

CLDN18.2 Highly Expressed in Gastric Cancer

Tumor Type	Prevalence
Gastric	>50%
Esophageal	>50%
Pancreatic	>50%
Ovarian	>10%
NSCLC	>4%

Zolbetuximab Validates the Target

Endpoint	Zolbe + Chemo	Chemo (alone)
OS (mo)	14.4 - 18.2	12.2 - 15.5
PFS (mo)	8.2 - 10.6	6.8 - 8.7
ORR	32 - 40%	31 - 40%

Clear Survival Improvements
in CLDN-18.2-high patients

Large Near-term Market Opportunity

Gastric Cancer TAM:
~$7.8 Billion

- US, EU, Japan, China
- 1L-3L Opportunity
- Expansion into pancreatic/ other tumor types

Expression data: Shitara et. al. ASCO, Gastric cancer: official journal of the International Gastric Cancer Association and the Japanese Gastric Cancer Association (2024), AstraZeneca Epidemiology Data 2022
Clinical data: Tureci, Annals of Oncology 2019 – 4 PRs noted in 43 patients with >50% CLDN18.2 expression cut-off, all of which had > 70% which subgroup was 29 patients. Zolbe alone was 9-14% ORR
TAM: Adapted from Globe Life Science report to Bolt January 2025. Includes first, second, and third line therapy for US, UK, France, Germany, Italy, Spain, Japan and China.

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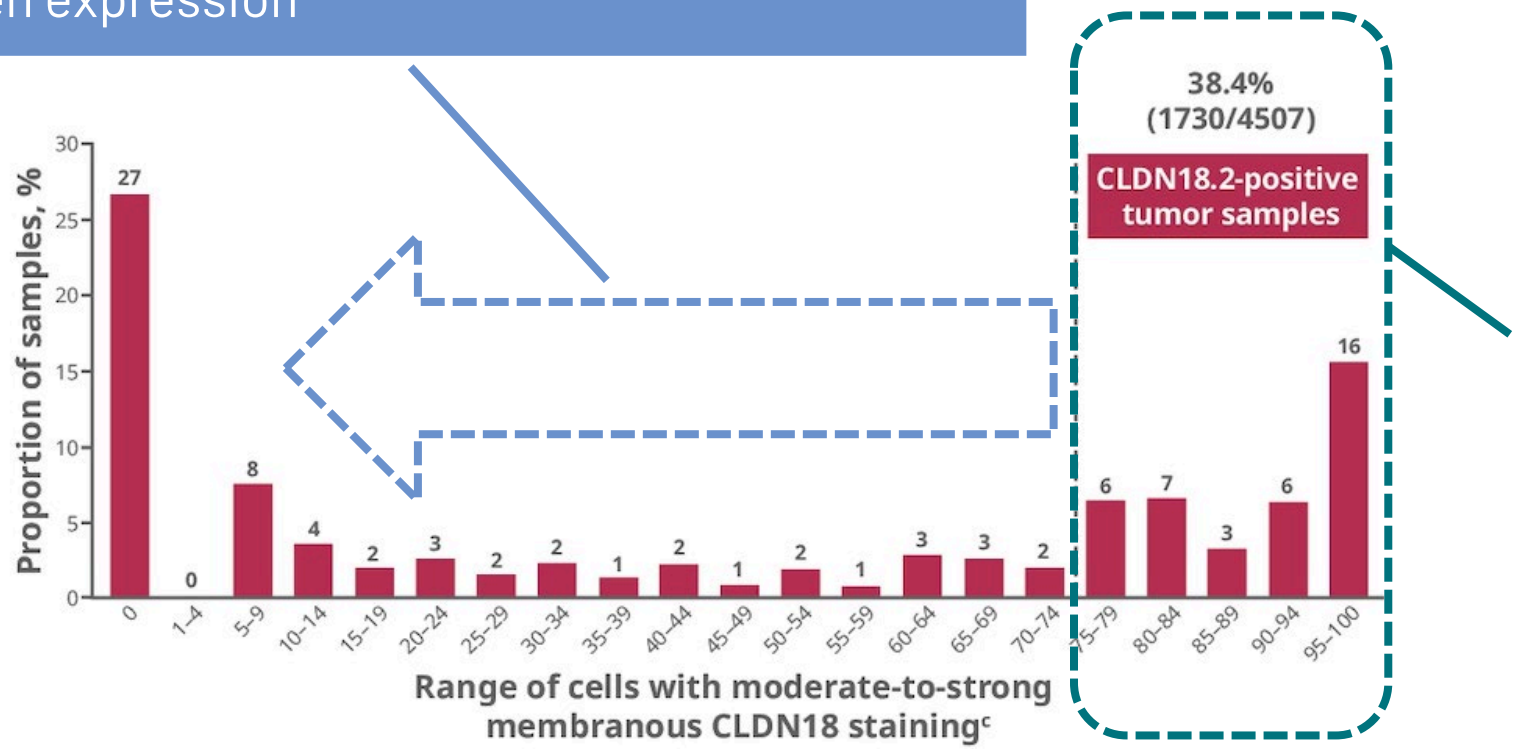
BDC-4182 Has the Potential to Double Current Claudin 18.2 Market

BDC-4182 has the potential to expand the market AND to outperform other claudin 18.2-directed therapies in tumors with lower antigen expression



Vyloy® (zolbetuximab) is approved for use in tumors with ≥75% IHC2+, ~40% of the market

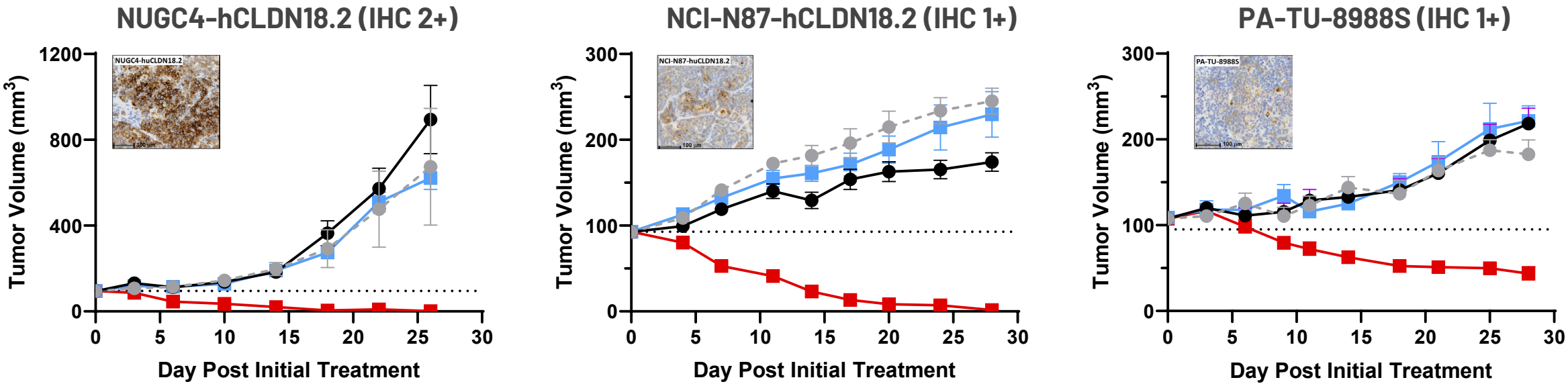
Preclinical data published in Fu CL et al. SITC 2025

BDC-4182 Preclinical Data

May 2026

Support for Potential to Expand the Market to Lower Antigen Levels

Efficacy demonstrated in low-antigen preclinical models with no adaptive immune system

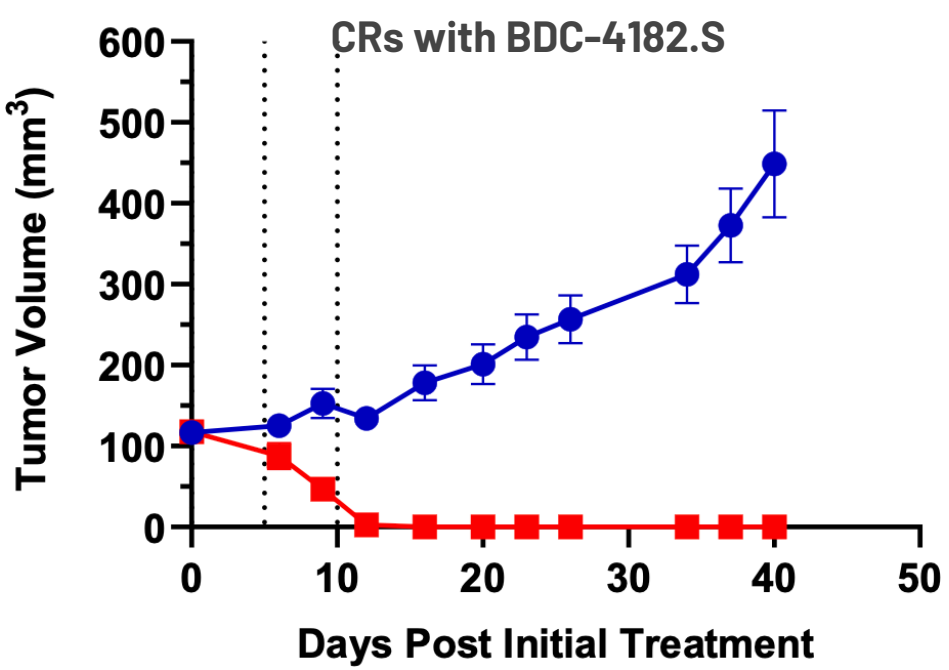


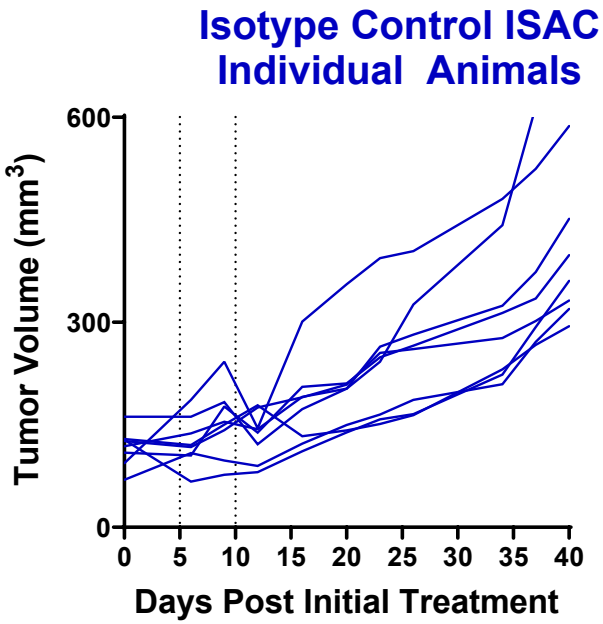
	IHC Score	BDC-4182.S (TGI)	Tumor Free Mice
NUGC4-hCLDN18.2	2+	100%	8 out of 8
NCI-N87-hCLDN18.2	1+	99%	6 out of 8
PA-TU-8988S	1+	76%	None

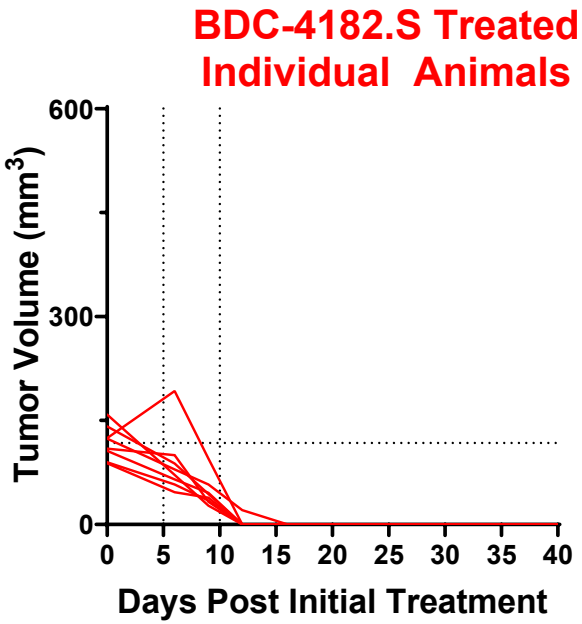
Adapted from Fu CL et al., SITC 2024.
SCID/beige mice bearing the indicated tumors were treated with BDC-4182.S (BDC-4182 surrogate) at 5 mg/kg BIW (4 total doses, n=8 per group). Tumor growth inhibition (TGI) was measured on day 26 or 28 post initial treatment The number of tumor free animals was recorded on the final day of the study. NUGC4 and NCI-N87 are gastric cancer cell lines, engineered here to express claudin 18.2. PA-TU-8988S is a pancreatic cancer cell line that endogenously expresses claudin 18.2.

BDC-4182.S Elicits Complete Regression in Immunologically Cold KPC Model

Complete Responses in Cold KRAS^{mut} P53^{mut} PDAC Tumor-bearing Mice







Isotype Control ISAC BDC-4182.S

Adapted from Fu CL et al. SITC 2024
C57BL/6 mice bearing KPC tumors (IHC 3+ CLDN18.2) were treated via IP with the indicated test articles at 5 mg/kg when tumors reached ~100 mm3. Animals received three doses in total (dashed lines). Data are shown as mean with SEM from n=8 female mice per group.

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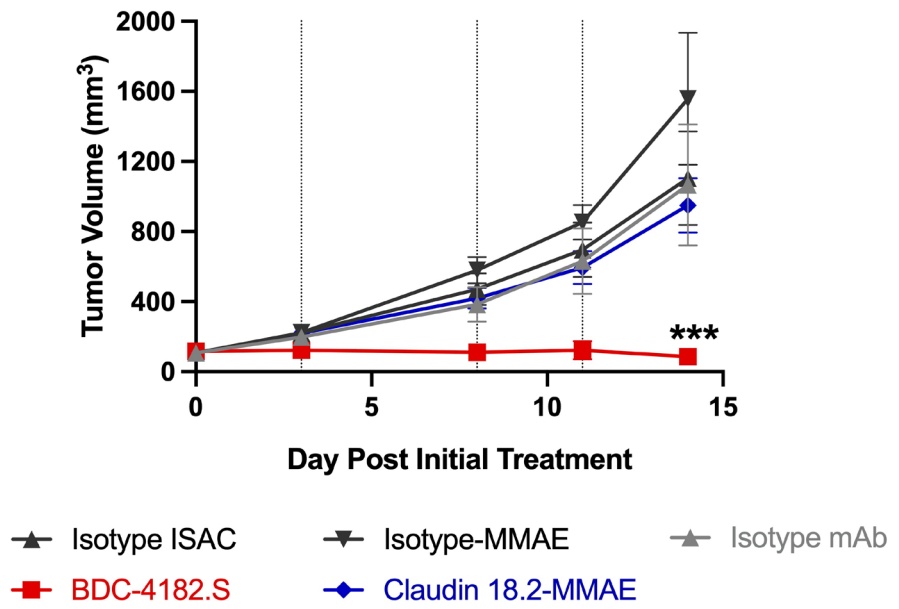
BDC-4182 Activity Superior to MMAE and TOPO1 ADCs in IHC1+ Syngeneic Model

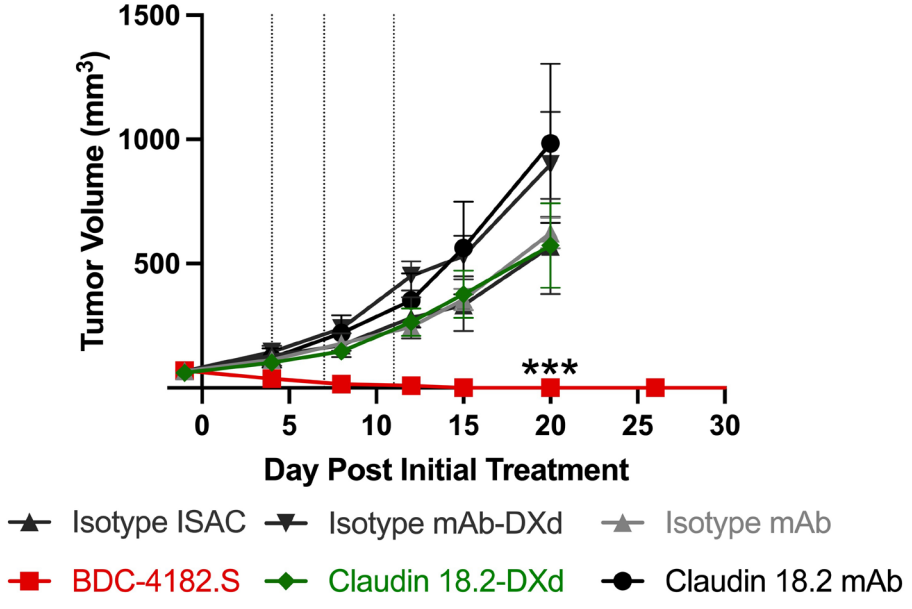
Superior to MMAE ADC

Limited ADC Efficacy in IHC1+ Model



Superior to TOPO1 (DXd) ADC

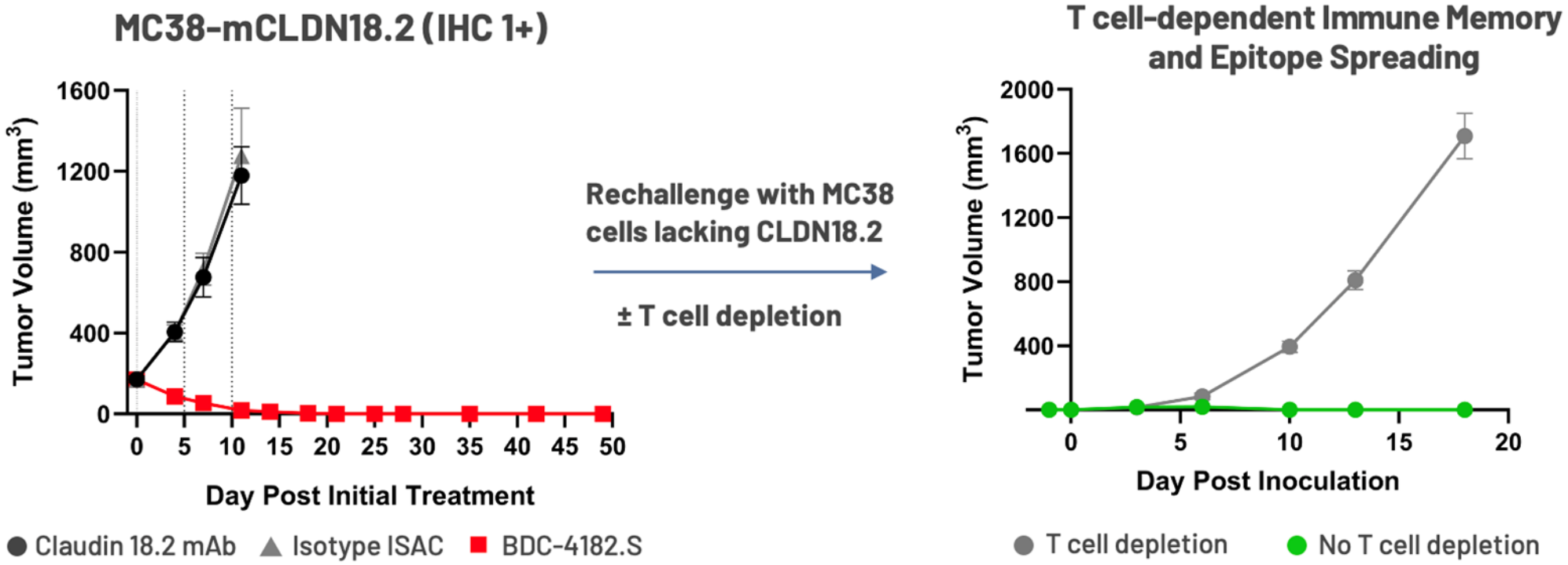
Limited ADC Efficacy in IHC1+ Model



Adapted from Fu CL et al., SITC 2024
C57BL/6 mice bearing MC38-mCLDN18.2 tumors (IHC 1+ by H-score) were administered 5 mg/kg of the indicated test article biweekly for 4 doses (dashed lines; n=6 to 8 mice/group)

BDC-4182 Has Curative Potential

BDC-4182.S Induces Immunological Memory and Epitope Spreading



Adapted from Fu CL et al. SITC 2025

BDC-4182.S treated mice (5 mg/kg, q5dx3) with complete tumor regression for > 28 days following their last treatment were rechallenged with MC38-mCLDN18.2 cells (IHC1+ by H-score) on their right flank and parental MC38 cells on the left flank in the presence of absence of CD4 & CD8 T cell depletion (n=4 mice/group). Mice remained tumor free when challenged with MC38-mCLDN18.2 cells in the absence of T cell depletion (not shown).

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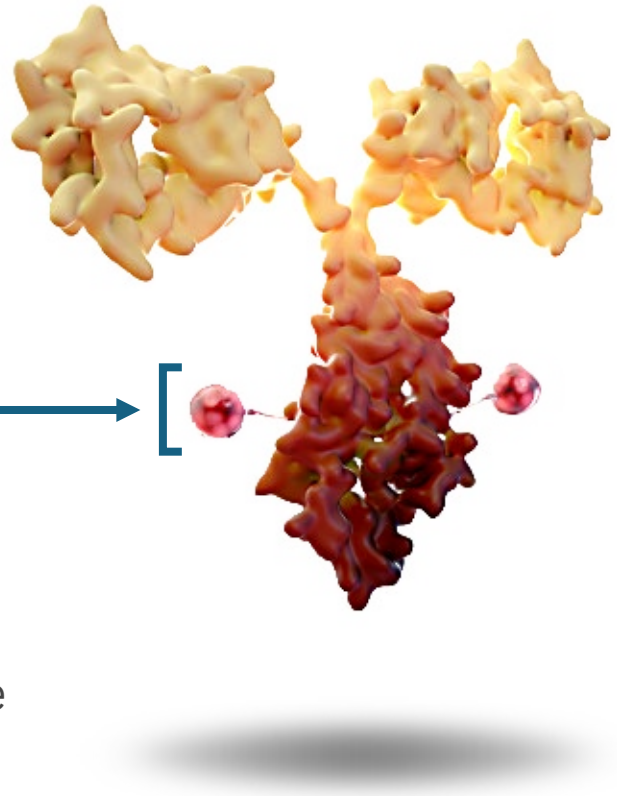
Pioneering Boltbody™ ISAC Platform

May 2026

Pioneering a New Class of Immuno-oncology Products:
Immune-stimulating Antibody Conjugates (ISACs)

Boltbody™ ISAC



Tumor-targeting Antibody

- Geo-locates ISAC to antigen on surface of a tumor cell
- Active Fc region drives antibody-dependent cellular phagocytosis (ADCP)

Immune-stimulating Linker-payload

- Potent stimulator of the innate immune system
- Non-cleavable linker
- Cell membrane impermeable

Ackerman SE, et al. Nature Cancer. 2021;2:18–33.

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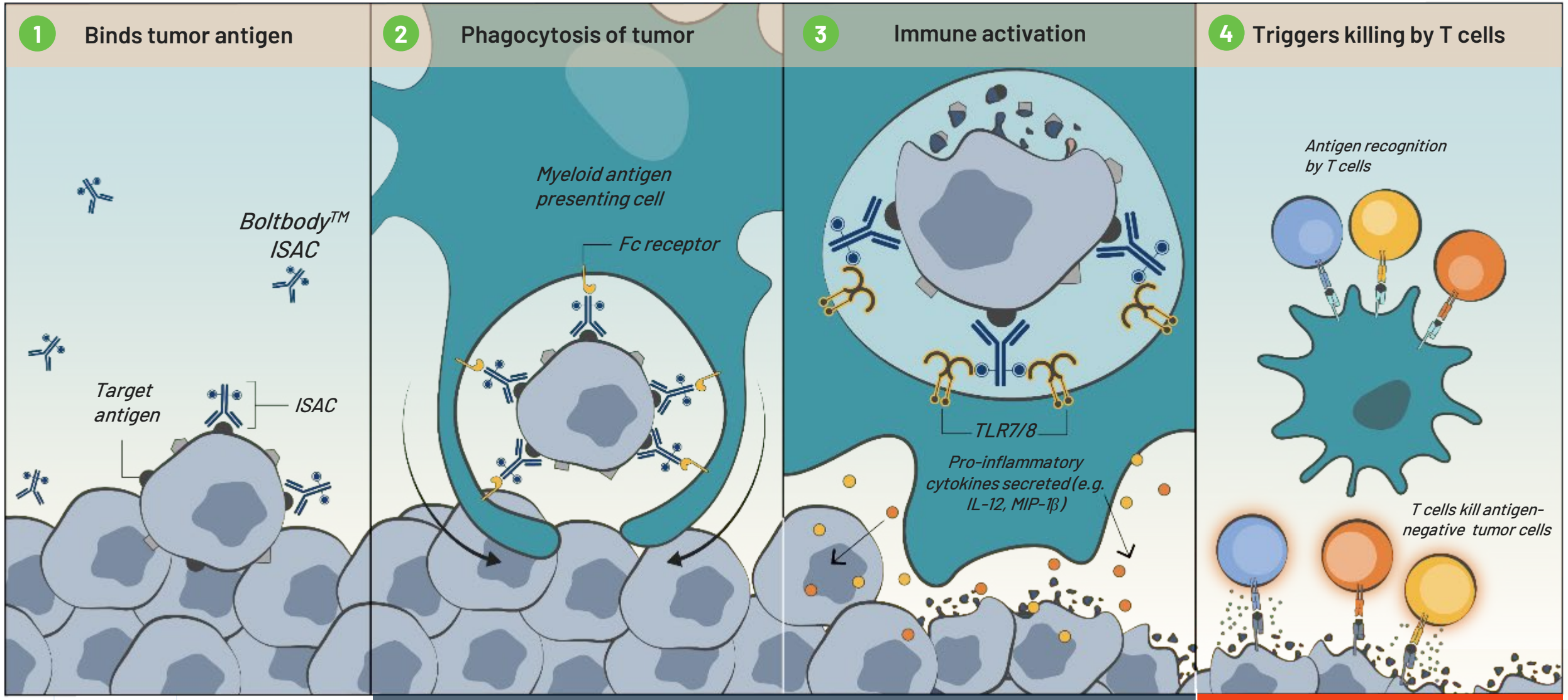
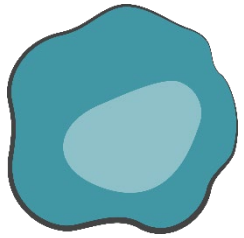
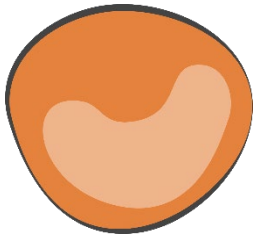
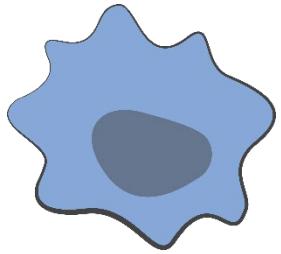
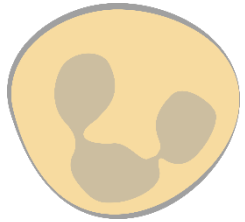
Boltbody™ ISAC Mechanism of Action

Broad activation of the immune system to enable durable responses and bystander killing of antigen-negative tumors



Why Target Toll-like Receptors 7 and 8?

- TLRs are receptors that recognize specific foreign patterns/signatures (e.g. viral, bacterial, fungal)
- TLR7 and TLR8 are expressed intracellularly in the phagolysosome in a variety of immune cells:

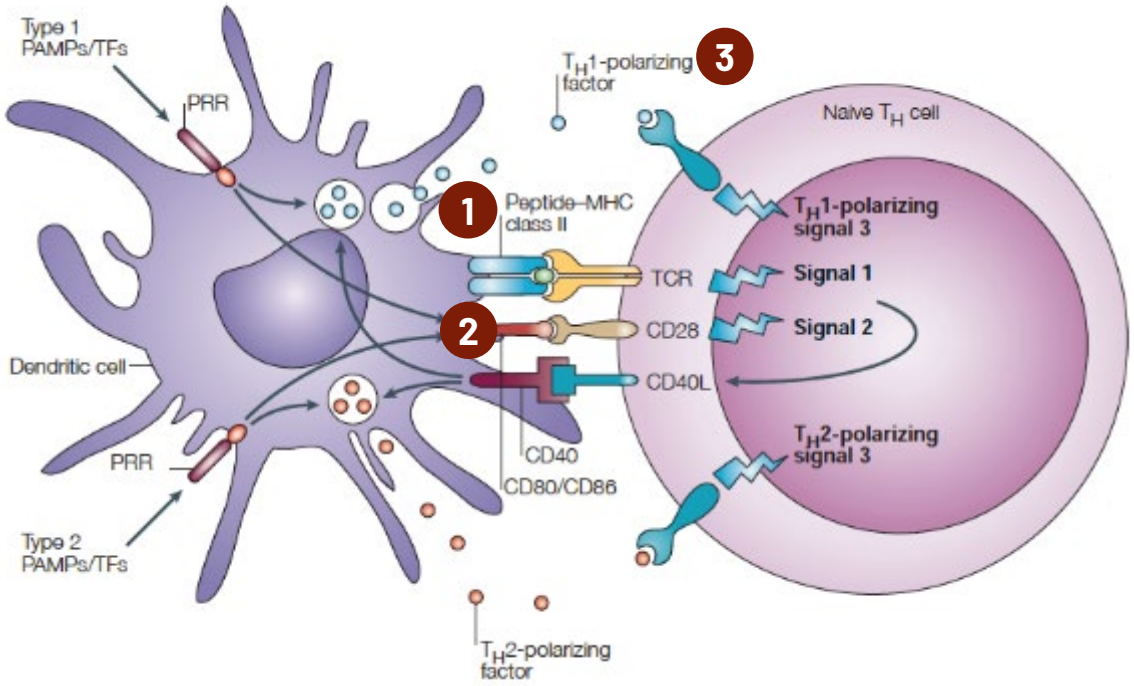
TLR7	TLR7/8			TLR8
				
Plasmacytoid Dendritic Cells	Conventional Dendritic Cells	Monocytes	Macrophages	Neutrophils

Goal of TLR7 and TLR8 stimulation is anti-tumor activity

Stimulation produces IFNα	Stimulation produces cytokines such as TNFα and IL-12p70 and chemokines such as MIP-1β (recruits more myeloid cells) & IP-10 (recruits more T cells)

ISACs Deliver Three Different Activation Signals to T cells

T cell engagers only deliver one



1. Signal 1: Antigen Specificity
2. Signal 2: Co-stimulation
3. Signal 3: T Cell Polarization (e.g. IL-12p70)

Adapted from Kapsenberg, M.L. et al. (2003) Nat Rev Immunol. 3(12):984-93

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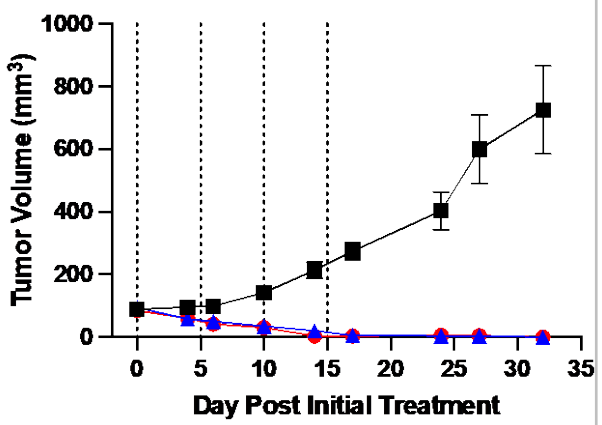
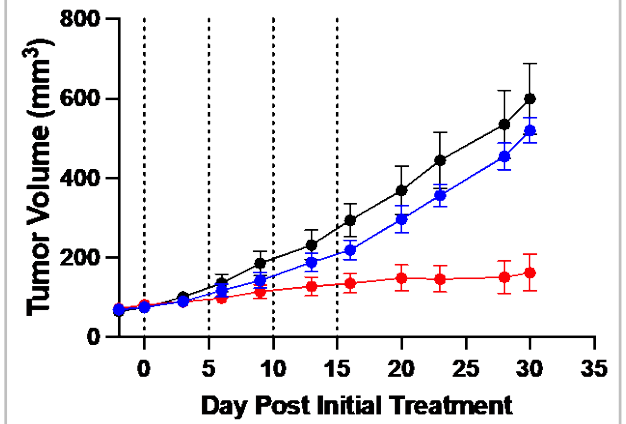
Next-generation ISACs are Dramatically Better than First-generation ISACs

Across multiple tumor antigens with varying expression levels

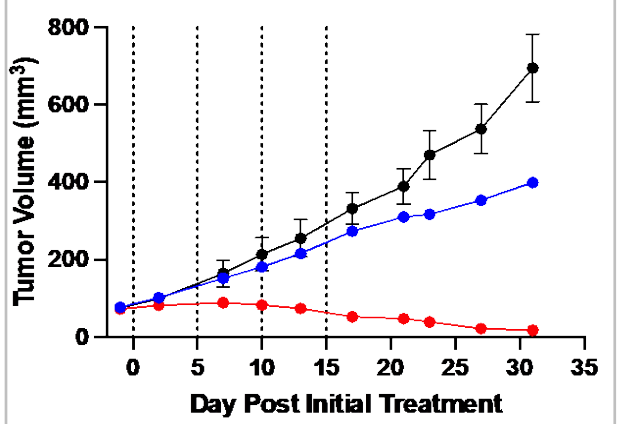
HER2
HCC1954 Model >500K Molecules/Cell



HER2
JIMT-1 Model ~25K Molecules/Cell

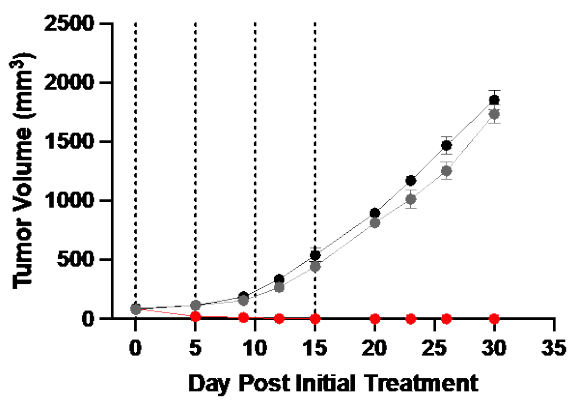


TROP2
JIMT-1 Model ~50K Molecules/Cell



CEA
HPAF-II Model ≥60K Molecules/Cell

Not Shown: 17.4% TGI for First-Gen ISAC in separate study



Naked mAb Control **First-generation ISAC** **Next-generation ISAC** **Isotype mAb**

Adapted from Ptacek J et al. SITC 2024
Mice bearing tumors with the indicated cell line were treated with 5 mg/kg or 2.5 mg/kg Q5D x 4 of the indicated tumor-targeting naked mAb, tumor-targeting ISAC or isotype mAb.

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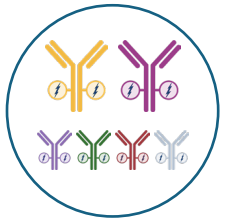
- Initial clinical data should provide proof-of-concept for broader ISAC approach



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Thank you