Bolt Biotherapeutics

BDC-3042 Phase 1 Results & Corporate Update

May 12, 2025

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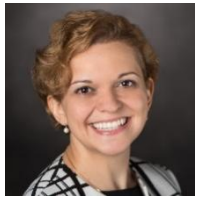
Introduction

Sarah Nemec

SVP Finance & Principal Accounting Officer

Presenters for Bolt Biotherapeutics

	Sarah Nemec SVP Finance & Principal Accounting Officer
	Willie Quinn President & CEO
	Justin Kenkel, Ph.D. Senior Principal Scientist

	Ecaterina Dumbrava, M.D. Associate Professor Investigational Cancer Therapeutics MD Anderson Cancer Center BDC-3042 Investigator
	Grant Yonehiro Chief Operating Officer & Chief Business Officer
	Jakob Dupont, M.D. Board Director & Senior Clinical Advisor

Additional team members available for Q&A

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Overview

Willie Quinn

President & CEO

Bolt Biotherapeutics Overview

- Mission: *Harnessing the power of the immune system to improve lives and eradicate cancer*
- Finished 1Q25 with $58.0 million cash[1]
  - Operating runway to mid-2026
- BDC-3042, first-in-class dectin-2 agonist antibody
  - Announced Phase 1 results at AACR in April
  - Running a partnering process
- BDC-4182, next-generation Boltbody™ ISAC
  - Phase 1 trial opened for enrollment in April
- Genmab collaboration continues to add value
  - 2 product candidates have advanced into development
  - Research and development on additional programs continues

[1] Cash, cash equivalents, and marketable securities balance of $58.0 million as of 3/31/2025

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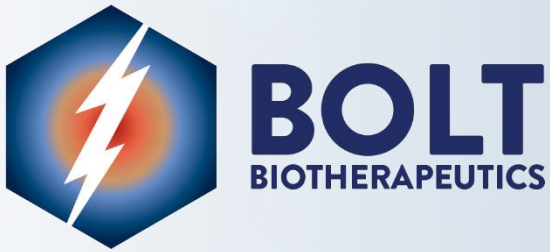
Agenda

• BDC-3042 rationale & preclinical development	Justin Kenkel, Ph.D.
• BDC-3042 Phase 1 clinical results	Ecaterina Dumbrava, M.D.
• BDC-3042 partnering	Grant Yonehiro
• BDC-4182 clinical development plan	Jakob Dupont, M.D.
• Conclusion	Willie Quinn



BDC-3042 Rationale & Preclinical Development

Justin Kenkel, Ph.D.

Senior Principal Scientist

BDC-3042 First-in-Class Agonist Antibody Program

Emerging Anti-Tumor Efficacy Demonstrated in Phase I Dose-Escalation Study

BDC-3042

Dectin-2-targeting agonist antibody

Powerful and Selective I/O Target: Dectin-2

- Expressed by tumor-associated macrophages (TAMs)
- Dectin-2 agonism activates TAMs & elicits anti-tumor response
- Anti-PD-1 therapy upregulates Dectin-2 expression in the tumor microenvironment[1]

Dose-Escalation Study Supports Further Development

- Safe and well tolerated, supporting combination strategies
- Biologically active, with evidence of target engagement & immunostimulatory effects consistent with preclinical studies
- 20-day half-life enabling convenient dosing regimens
- Promising signs of antitumor activity as a single agent in non-small cell lung cancer, in the post-checkpoint inhibitor setting, and at the highest dose tested

Data cut-off April 7, 2025. Dumbrava et al., AACR 2025

[1]Based on published transcriptomic data assessing human tumor biopsies before & after treatment with anti-PD-1 mAbs (Cindy Yang et al., Nat Commun 2021; Bi et al., Cancer Cell 2021; Chen et al., Cancer Discov 2016)

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First-in-Class Opportunity with High Specificity to Target TAMs

Dectin-2: Immune-activating pattern recognition receptor with favorable expression profile[1,2]

- Restricted to myeloid cells
  - Tumor-associated macrophages
  - Monocytes, dendritic cells (cDC2)
  - Alveolar macrophages, other tissue-resident populations
  - Low granulocyte expression
- Dectin-2 agonism activates & reprograms TAMs leading to:
  - Enhanced $CD8^+$ T cell responses
  - Complete tumor regression
  - Induction of immunological memory[3,4]

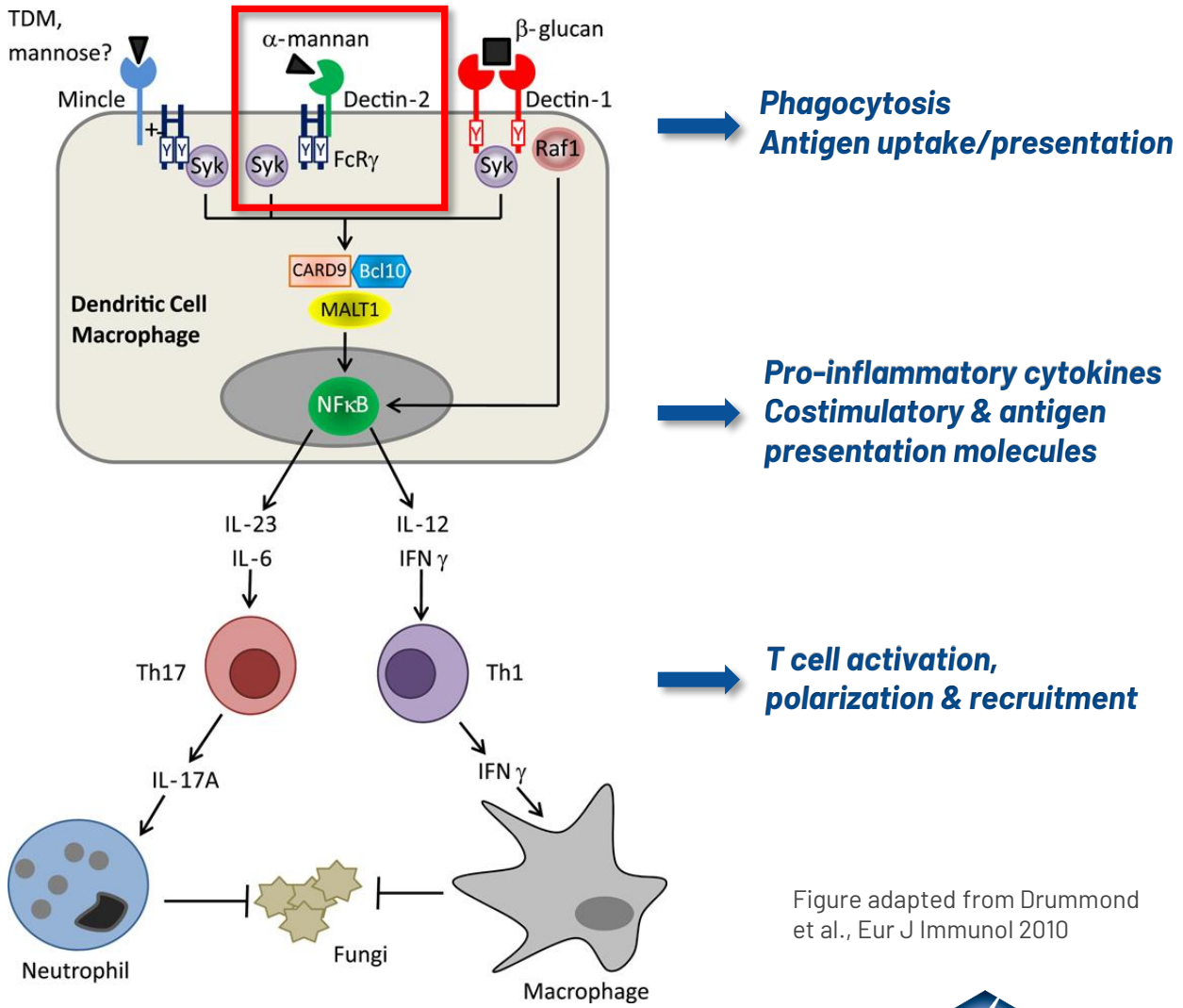
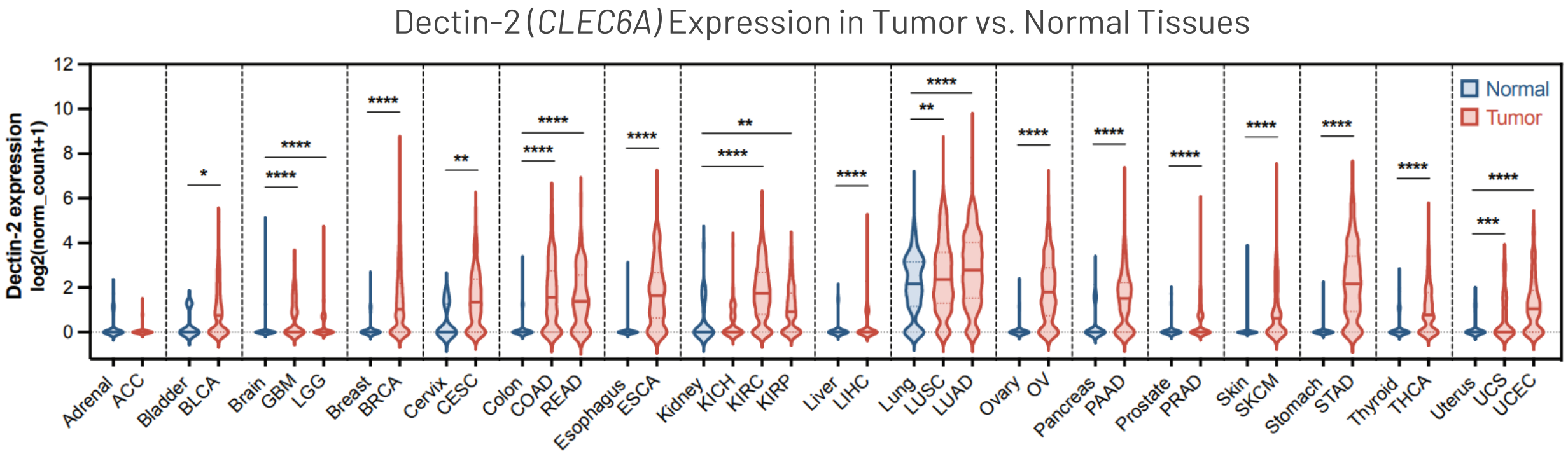


Figure adapted from Drummond et al., Eur J Immunol 2010

[1]Taylor et al., Eur J Immunol 2005; [2]Kerscher et al., Int Immunol 2013; [3]Kenkel et al., SITC 2021; [4]Kenkel et al., AACR 2023

Dectin-2 Gene Expression is Elevated Across Most Solid Tumor Types

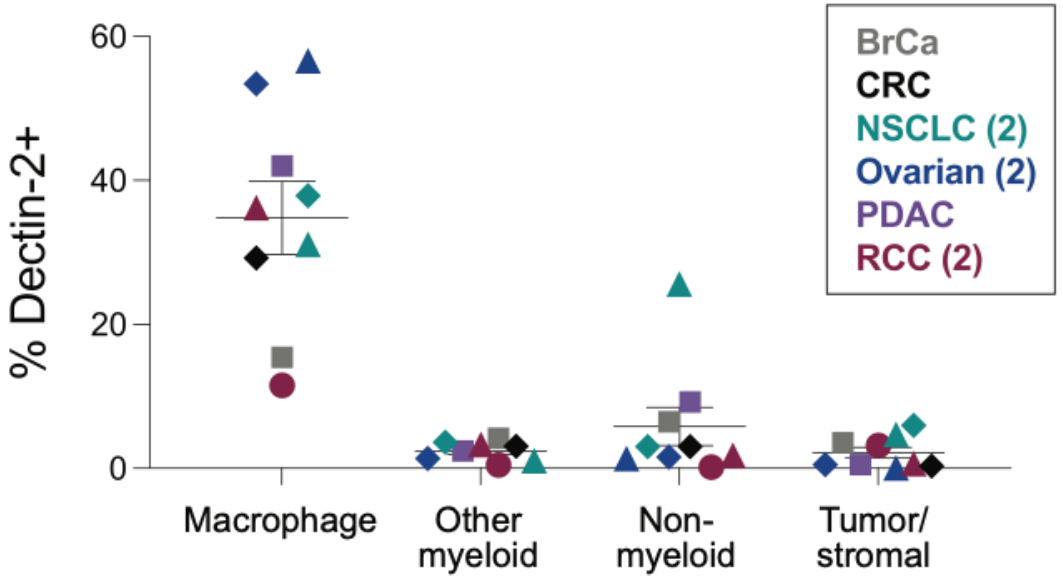
Potential market opportunity exceeds $10 billion in initial target indications



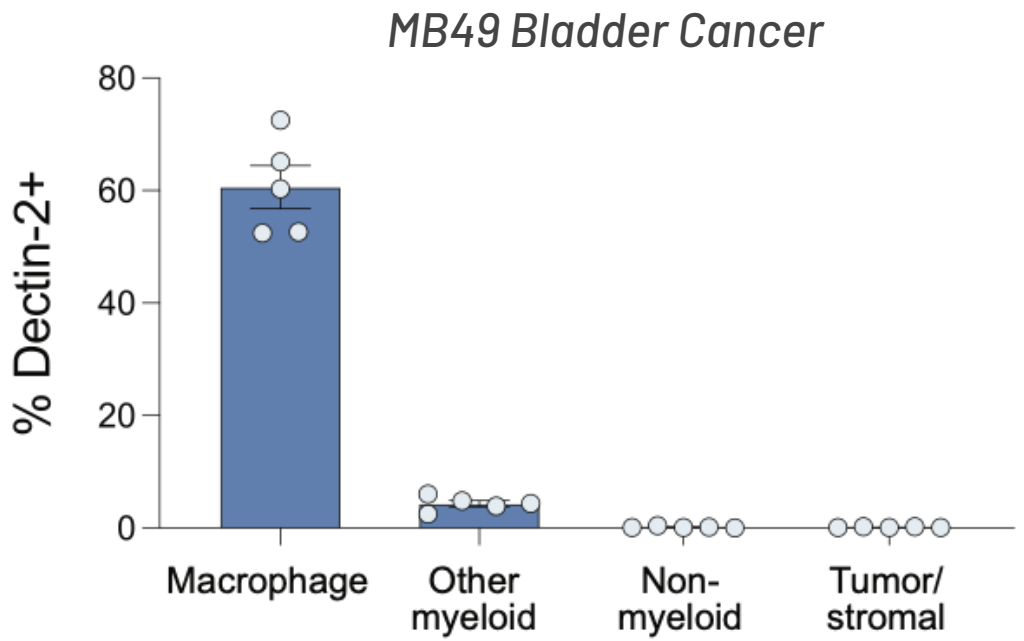
Dectin-2/CLEC6A mRNA expression in human tissue samples from the TCGA (tumor) and GTEX (normal) datasets (accessed Oct. 2019).
Kenkel JA, et al. Cancer Research. 2023;83(suppl 7):2964.

Dectin-2 Is Selectively Expressed by Tumor-Associated Macrophages (TAMs)

Dectin-2 Expression in Human Tumors



Dectin-2 Expression in Syngeneic Mouse Model



Kenkel JA, et al., SITC Poster (2021)

Human and murine tumor samples were processed into single cell suspensions and analyzed by flow cytometry. Dectin-2 expression on the indicated cell subsets was evaluated using commercially available anti-Dectin-2 antibodies.

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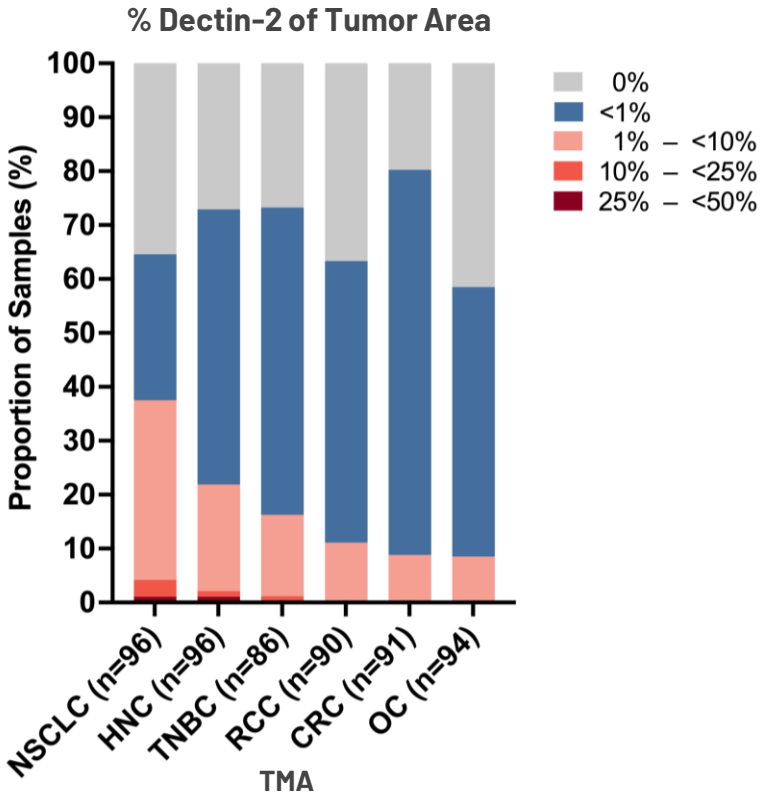
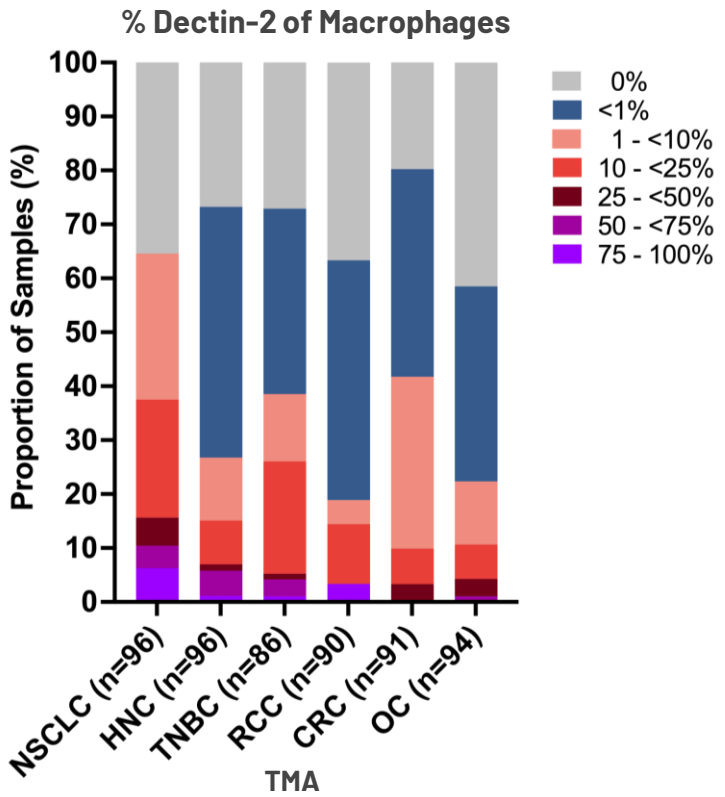
Dectin-2 Expression Confirmed by IHC in NSCLC and Additional Tumor Types

Dectin-2 IHC performed on whole tissue sections & tissue microarrays comprising over 600 samples

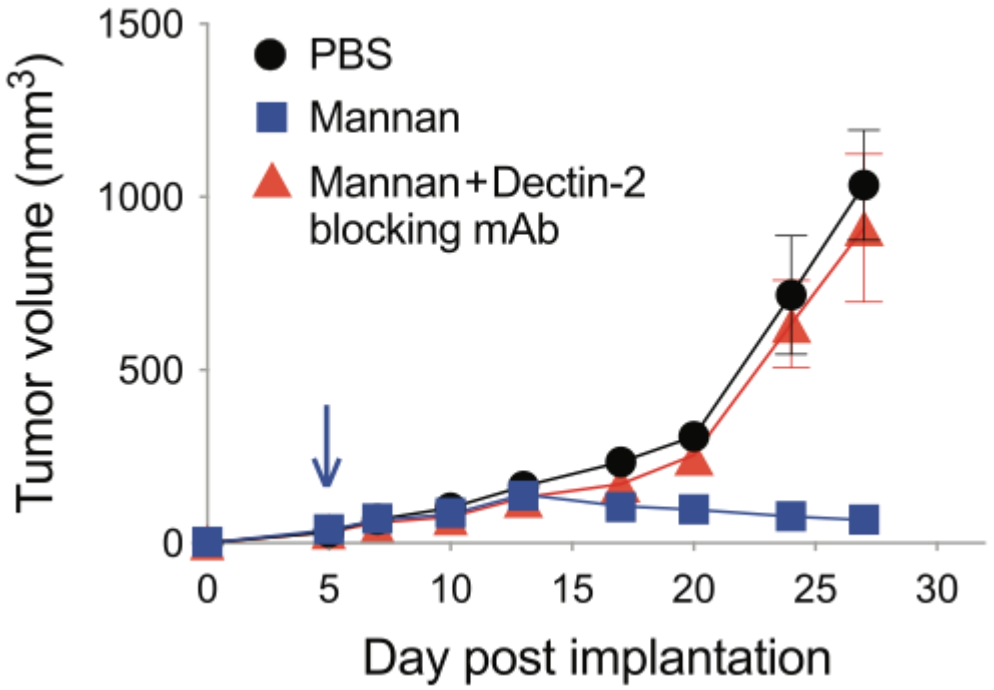
- Dectin-2 expression was observed in all whole tissue sections tested (n=137, ~20 samples per indication)
- TMAs have much smaller tissue area, often with limited stroma, and a subset lacked expression
- In NSCLC, ~40% of TMA samples and 50% of whole tissue sections had ≥1% tumor area positive for Dectin-2



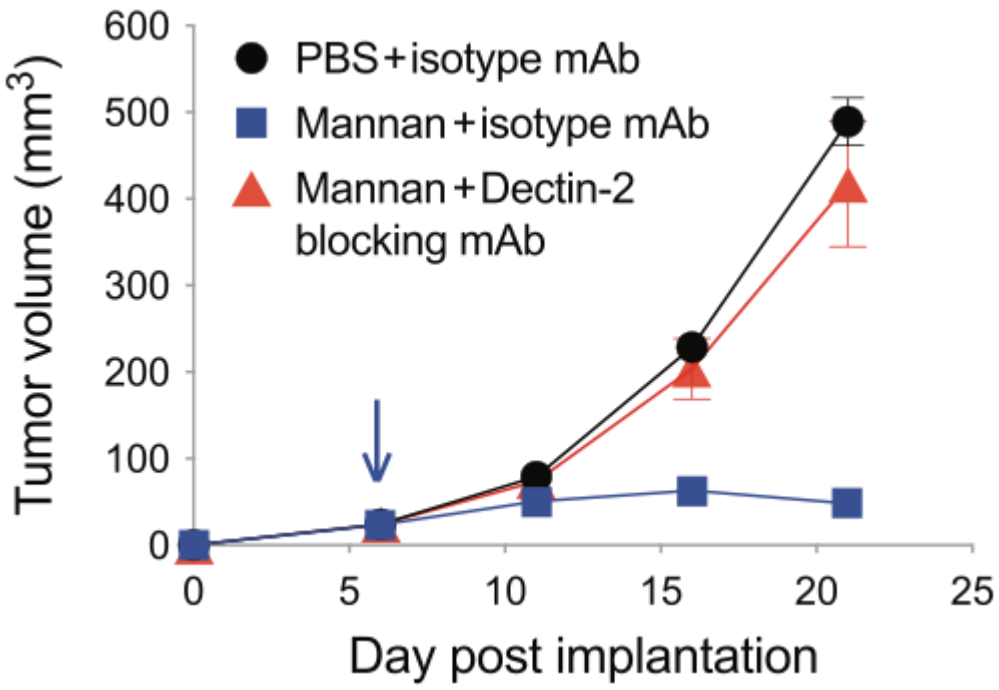


BDC-3042 Surrogate (Mannan) Elicits Tumor Regression in Multiple Syngeneic Models

MB49 Bladder Cancer



LMP Pancreatic Cancer



Kenkel JA, et al., SITC Poster (2021)

Tumor-bearing mice were treated systemically with surrogate Dectin-2 agonist (*S. cerevisiae* mannan; 12.5 mg/kg IV Q2D). Groups of mice were pretreated with Dectin-2-blocking or isotype control antibodies. Blue arrows indicate day of treatment initiation.

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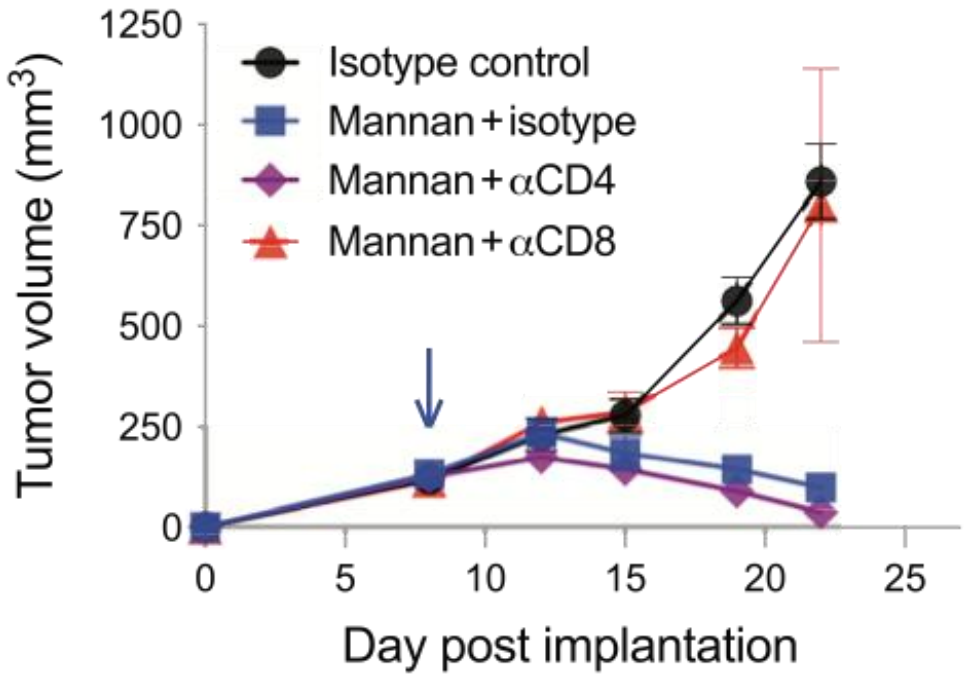
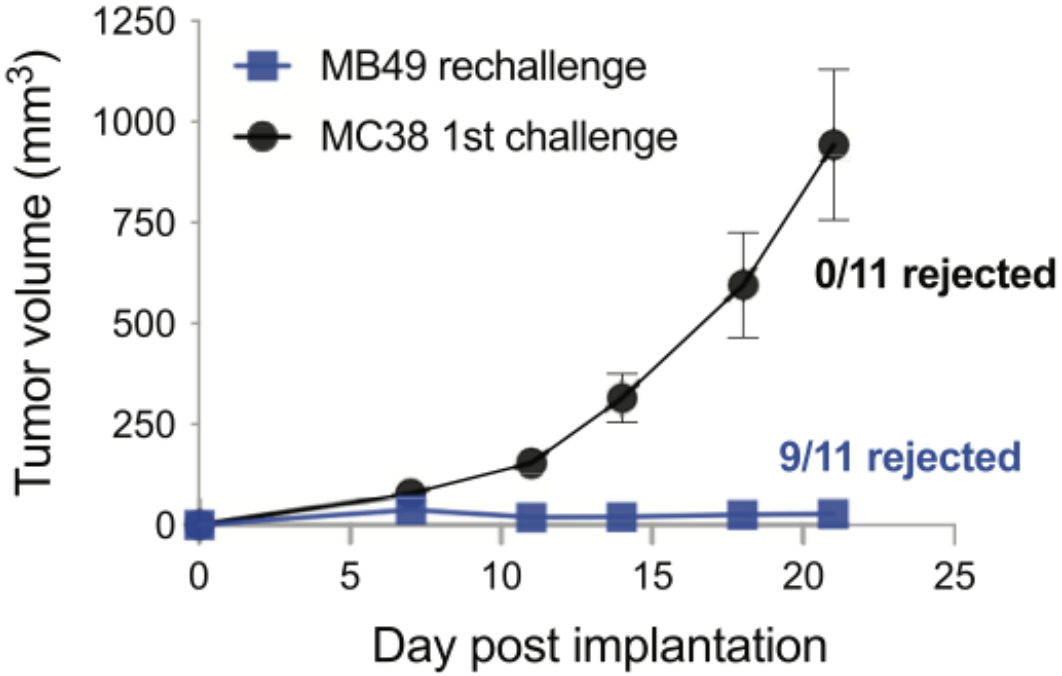
BDC-3042.S Elicits CD8⁺ T Cell-Dependent Antitumor Activity & Immunological Memory

MB49 T Cell Depletion Study



Tumor Rechallenge Study



Kenkel JA, et al., SITC Poster (2021)

MB49 tumor-bearing mice were treated systemically with mannan plus T cell-depleting or isotype control antibodies. Mice that experienced complete tumor regression were rechallenged with MB49 and an unrelated tumor cell line (MC38) on opposite flanks.

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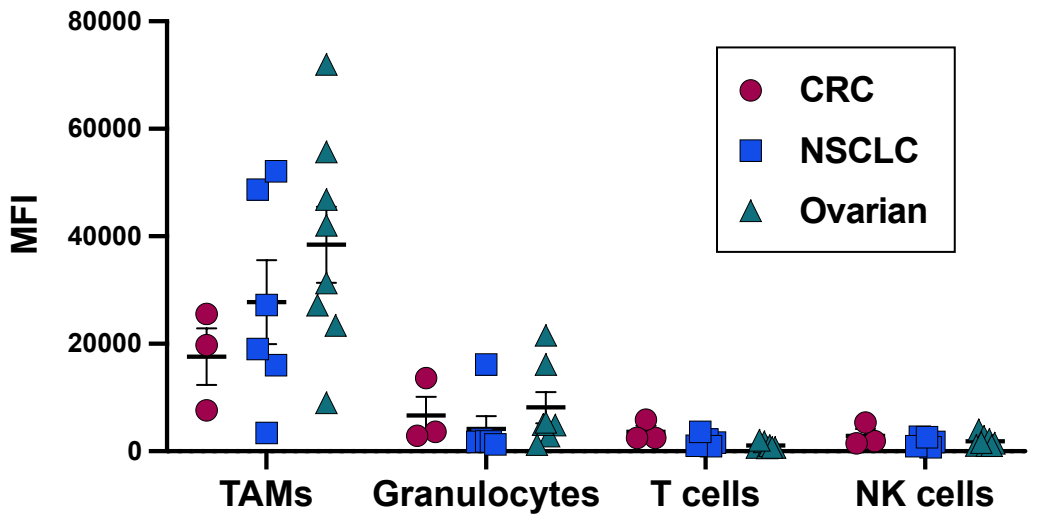
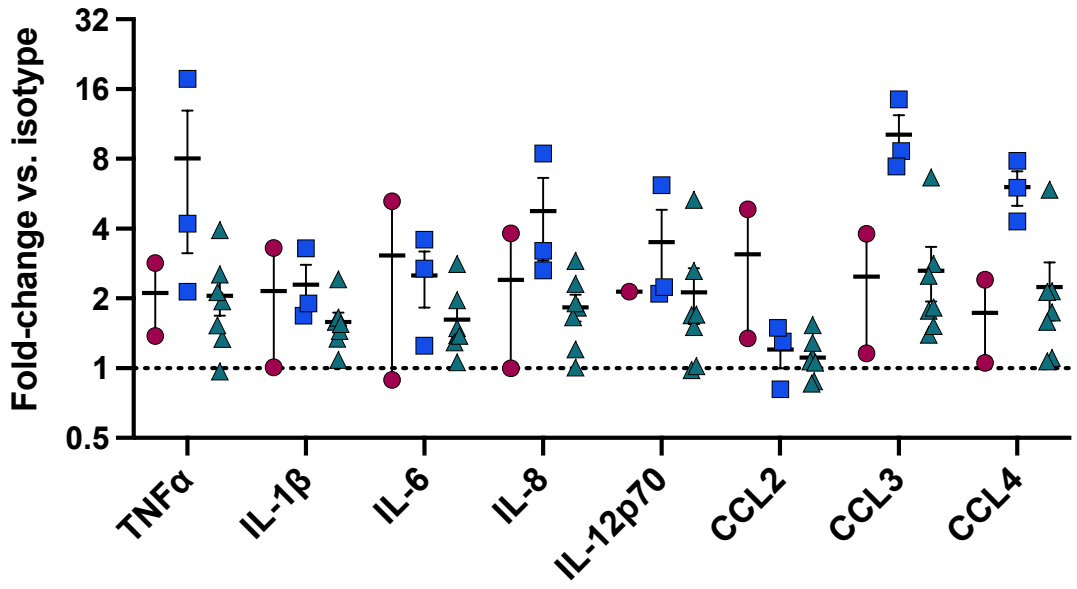
BDC-3042 Binds to TAMs & Elicits Cytokine Responses from Human Tumor Samples

BDC-3042 Binding to Tumor-Infiltrating Leukocytes



BDC-3042 Mediated Cytokine/Chemokine Secretion



Kenkel JA, et al., AACR Poster (2023)

BDC-3042 binding to immune cell subsets from dissociated human tumor samples was assessed by flow cytometry. A subset of the samples were stimulated overnight with BDC-3042 or an isotype control mAb followed by cytokine analysis.

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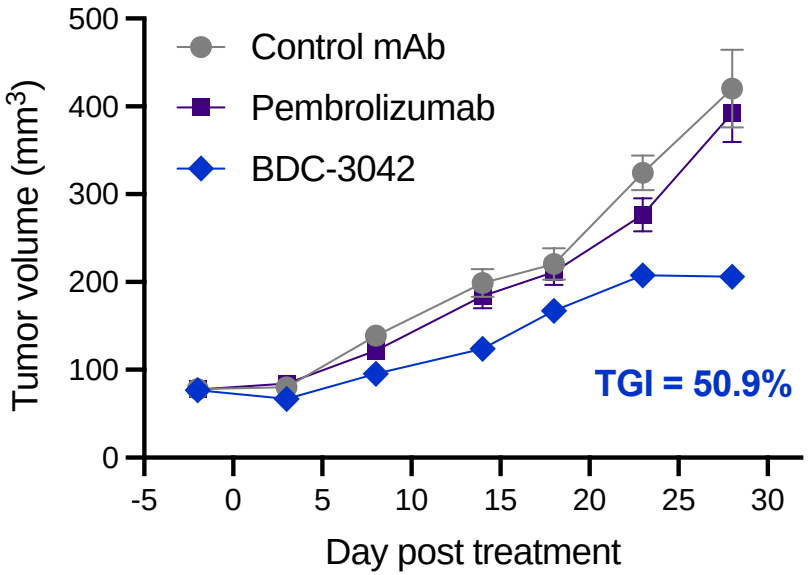
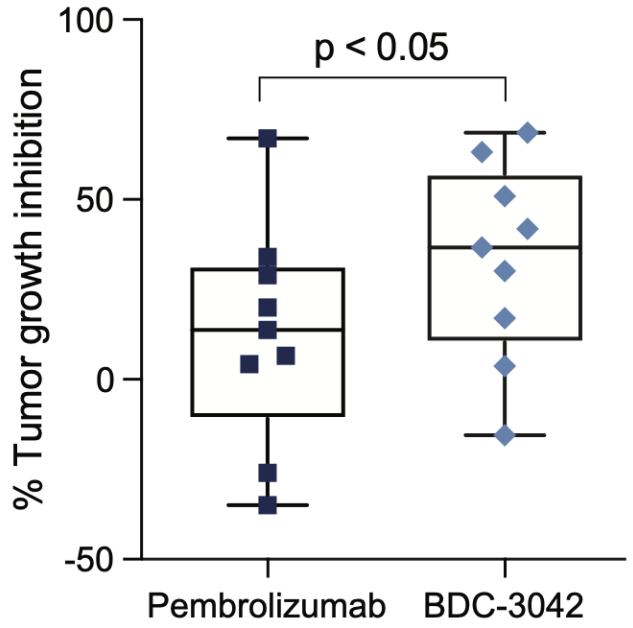
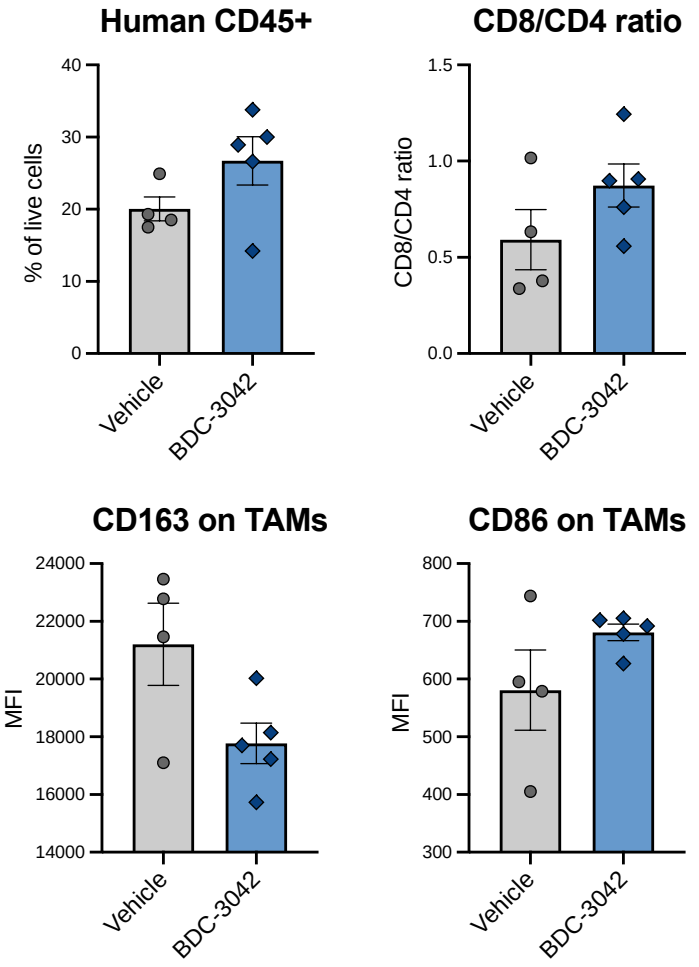
BDC-3042 Mediates Antitumor Activity in MDA-MB-231 Humanized Mouse Model

Greater Tumor Growth Inhibition with BDC-3042 vs. Pembrolizumab







Kenkel JA, et al., SITC Poster (2022)

(Left) huNOG-EXL mice from a single CD34+ HSC donor were implanted with bilateral MDA-MB-231 tumors (n=4-5 mice per group) and treated with the indicated test article at 1 mg/kg via IP administration (Q5D x 6). (Middle) Tumor growth inhibition (TGI) was calculated across 9 HSC donor cohorts for BDC-3042 or pembrolizumab as compared to an isotype control antibody. (Right) Tumors were analyzed by flow cytometry 48 hours after the second dose of vehicle or BDC-3042.

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BDC-3042 Phase 1 Clinical Results

Ecaterina Dumbrava, M.D.

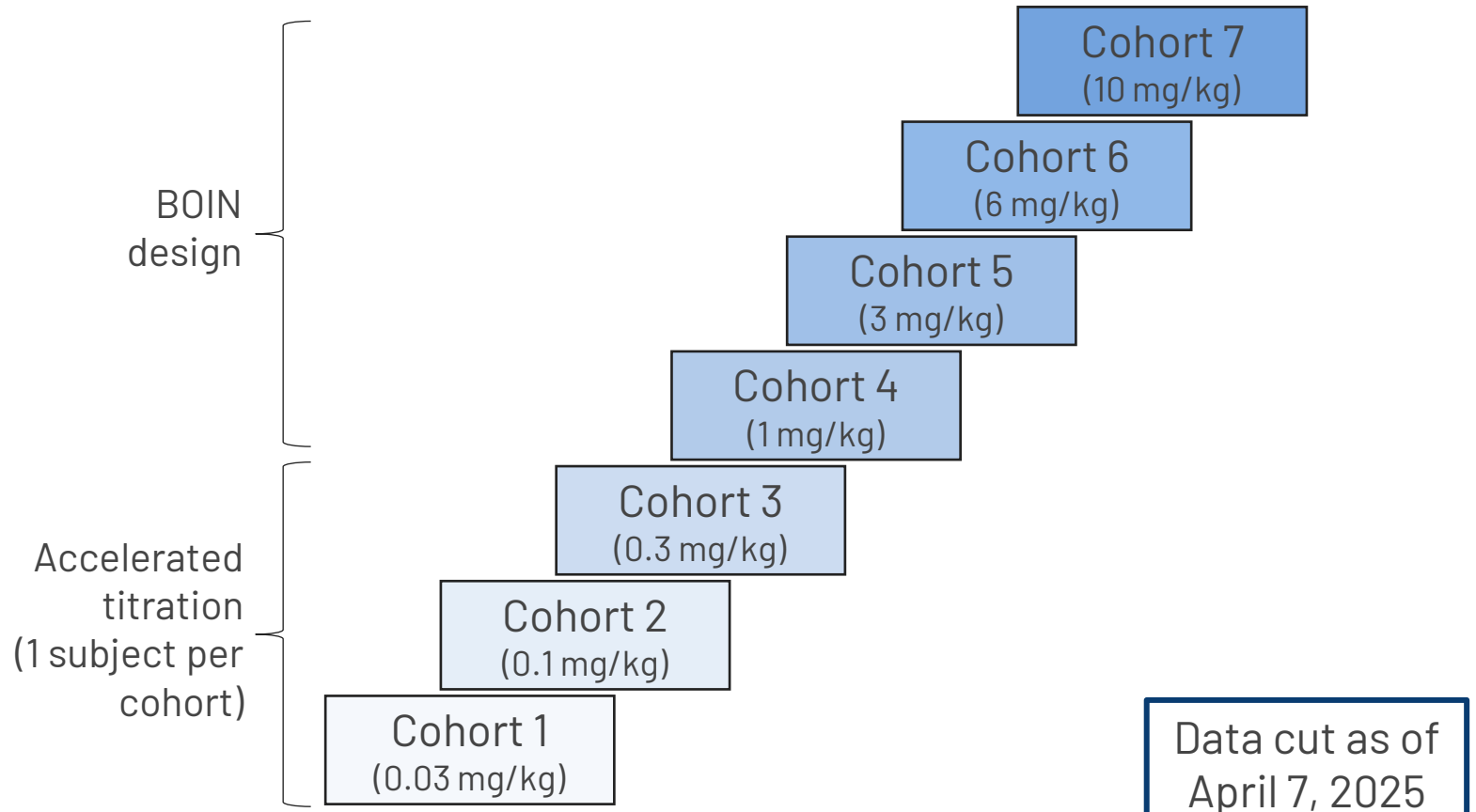
BDC-3042 Investigator

Associate Professor, Investigational Cancer Therapeutics

MD Anderson Cancer Center

BDC-3042 Phase 1 Clinical Trial in Various Solid Tumor Types

Dose Escalation Schema



Tumor Type	N (%)
CRC	8 (47%)
NSCLC	3 (18%)
ccRCC	2 (12%)
Ovarian	2 (12%)
Melanoma (Uveal)	1 (6%)
TNBC	1 (6%)
H&N	0 (0%)

Enrolled Subjects	17
Subjects on Treatment	2 (NSCLC)

Dumbrava et al., AACR 2025

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Demographics

Heterogeneous & heavily pretreated population (n=17) including 8 CRC patients

	Cohort 1 0.03 mg/kg N=1	Cohort 2 0.1 mg/kg N=1	Cohort 3 0.3 mg/kg N=1	Cohort 4 1 mg/kg N=4	Cohort 5 3 mg/kg N=4	Cohort 6 6 mg/kg N=3	Cohort 7 10 mg/kg N=3	**Total N=17**
Mean age, years (range)	61.0 (61, 61)	53.0 (53, 53)	78.0 (78, 78)	62.0 (52, 83)	55.8 (51, 63)	68.7 (59, 75)	68.0 (65, 72)	63.1 (51, 83)
Sex, n (%) **Female** **Male**	 0 1 (100%)	 1 (100%) 0	 1 (100%) 0	 2 (50%) 2 (50%)	 1 (25%) 3 (75%)	 1 (33.3%) 2 (66.7%)	 2 (66.7%) 1 (33.3%)	 8 (47.1%) 9 (52.9%)
Prior lines of therapies, Mean (range)	5 (5, 5)	2 (2, 2)	4 (4, 4)	4.8 (3, 8)	3.3 (2, 5)	6.0 (4, 8)	4 (4, 4)	4.3 (2, 8)
Prior immune therapy, n (%)	0 (0%)	0 (0%)	1 (100%)	1 (25%)	0 (0%)	2 (66.7%)	3 (100%)	7 (41.2%)
Tumor types, n (%): **Colorectal** **NSCLC** **Ovarian** **ccRCC** **TNBC** **Uveal Melanoma**	 1 (100%) 0 (0%) 0 (0%) 0 (0%) 0 (0%) 0 (0%)	 1 (100%) 0 (0%) 0 (0%) 0 (0%) 0 (0%) 0 (0%)	 0 (0%) 1 (100%) 0 (0%) 0 (0%) 0 (0%) 0 (0%)	 2 (50%) 0(0%) 1 (25%) 1 (25%) 0 (0%) 0 (0%)	 3 (75%) 0 (0%) 1 (25%) 0 (0%) 0 (0%) 0 (0%)	 1 (33.3%) 0 (0%) 1 (33.3%) 1 (33.3%) 0 (0%) 0 (0%)	 0 (0%) 2 (66.7%) 0 (0%) 0 (0%) 0 (0%) 1 (33.3%)	 8 (47.1%) 3 (17.6%) 2 (11.8%) 2 (11.8%) 1 (5.9%) 1 (5.9%)

Dumbrava et al., AACR 2025

Safety Summary

BDC-3042 has been well tolerated up to 10 mg/kg q2w

- No DLTs observed up to 10 mg/kg q2w
- No drug-related SAEs
- No grade 4 or 5 drug-related AEs
- One drug-related infusion related reaction (grade 1)
- Most frequent drug-related AEs were fatigue (12%), flatulence (12%), and nausea (12%)
- No drug-related treatment discontinuations
- No overarching trends identified in safety profile

Well tolerated at all doses, supporting combination strategies

Dumbrava et al., AACR 2025

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Summary of Treatment-Related TEAEs

	Cohort 1 0.03 mg/kg N=1	Cohort 2 0.1 mg/kg N=1	Cohort 3 0.3 mg/kg N=1	Cohort 4 1 mg/kg N=4	Cohort 5 3 mg/kg N=4	Cohort 6 6 mg/kg N=3	Cohort 7 10 mg/kg N=3	**Total N=17**
All grades (%)	0	1 (100%)	1 (100%)	2 (50%)	2 (50%)	1 (33.3%)	1 (33.3%)	8 (47.1%)
Grade ≥ 3 (%)	0	0	0	2 (50%)	0	0	0	2 (11.8 %)
Serious adverse events (%)	0	0	0	0	0	0	0	0
Leading to treatment discontinuation	0	0	0	0	0	0	0	0
Leading to treatment interruption	0	0	0	0	0	0	0	0
Leading to death	0	0	0	0	0	0	0	0

Related Grade 3 TEAEs: increased amylase/lipase and muscle weakness (Cohort 4)

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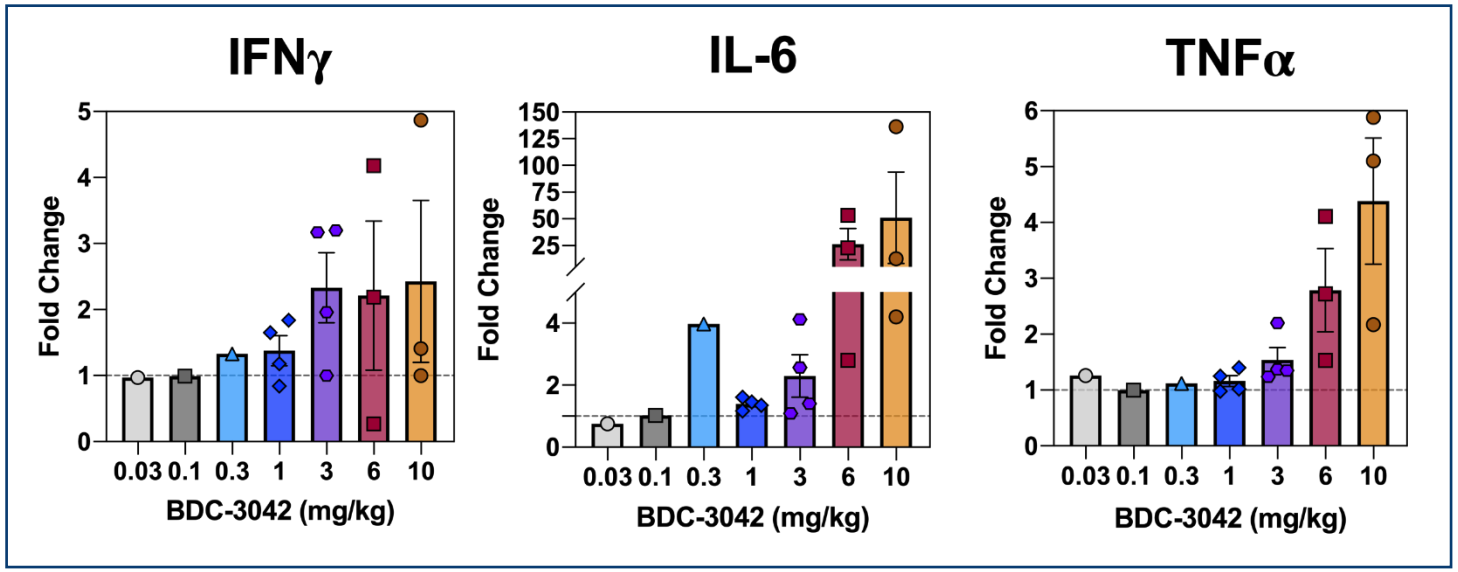
BDC-3042 Elicits Dose-Dependent Increases in Cytokines & Chemokines

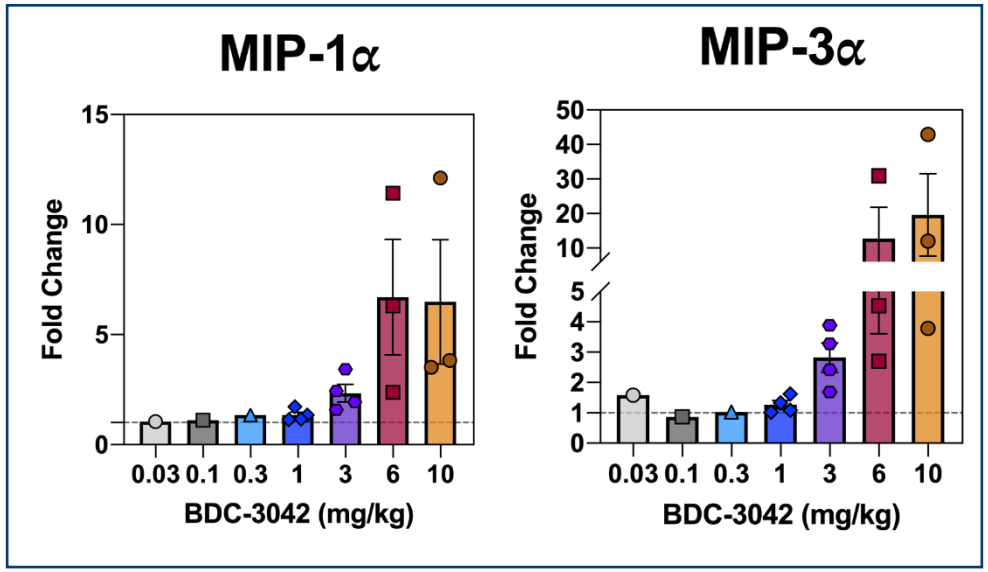
BDC-3042 induces dose-dependent increases in proinflammatory cytokines & chemokines

- Peripheral cytokine response is consistent with preclinical studies and suggests activation of both myeloid cells (IL-6, TNFα, MIPs) and T/NK cells (IFNγ)

Cytokines



Chemokines



Serum Cytokines & Chemokines Post 1st Dose

Serum cytokine levels at 4h (TNFα, IL-6, MIP-1α, MIP-3α) or 1d (IFNγ) following the first cycle of BDC-3042. Fold-changes were calculated relative to C1D1 pre-dose levels. Individual data points are shown with mean and SEM.

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Biomarker Responses in Clinical Trial Consistent with Preclinical Models

Comparison of Biomarkers Associated with Efficacy in Preclinical Models

BDC-3042 elicits cytokine response similar to profile associated with efficacy in preclinical models

- Doses of 3 mg/kg and above induce cytokine levels comparable to those seen in preclinical studies

		Biomarker	Preclinical[1]		BDC-3042 FIH Trial[4]				
			Syngeneic Mouse Model[2]	Humanized Mouse Model[3]	0.3 mg/kg (n = 1)	1 mg/kg (n = 4)	3 mg/kg (n = 4)	6 mg/kg (n = 3)	10 mg/kg (n = 3)
Myeloid Activation & Leukocyte Recruitment	Cytokines	IL-6	11.3x	2.9x	4.0x	1.4x	2.3x	26.4x	51.1x
		TNFα	1.9x	1.2x	1.1x	1.2x	1.5x	2.8x	4.4x
	Chemokines	CCL3 (MIP-1α)	~4x	1.7x	1.3x	1.3x	2.3x	6.7x	6.5x
		CCL4 (MIP-1β)	4.9x	1.8x	1.1x	1.1x	1.8x	4.3x	5.9x
T/NK Cell Activation	Cytokines	IFNγ	1.8x	2.1x	1.3x	1.4x	2.3x	2.2x	2.4x

[1]Fold-change calculated using group average biomarker levels with BDC-3042 or surrogate agonist relative to vehicle at same time point. [2]MB49 tumor-bearing mice (murine bladder cancer cell line) treated with surrogate Dectin-2 agonist mannan (10 mg/kg; response at 3h post 1st dose, or post 3rd dose for IFNγ). [3]CD34+ HSC-engrafted humanized mice (n=5/group) derived from an efficacy responsive donor bearing MDA-MB-231 TNBC tumors (treated with vehicle or BDC-3042 at 0.3 mg/kg; response at 6h post 1st dose, or 24h post 3rd dose for IFNγ). [4]Fold-change in biomarker levels 4h (or 1d for IFNγ) following 1st dose of BDC-3042 calculated relative to baseline values in each subject. Group averages are shown for multi-subject cohorts.

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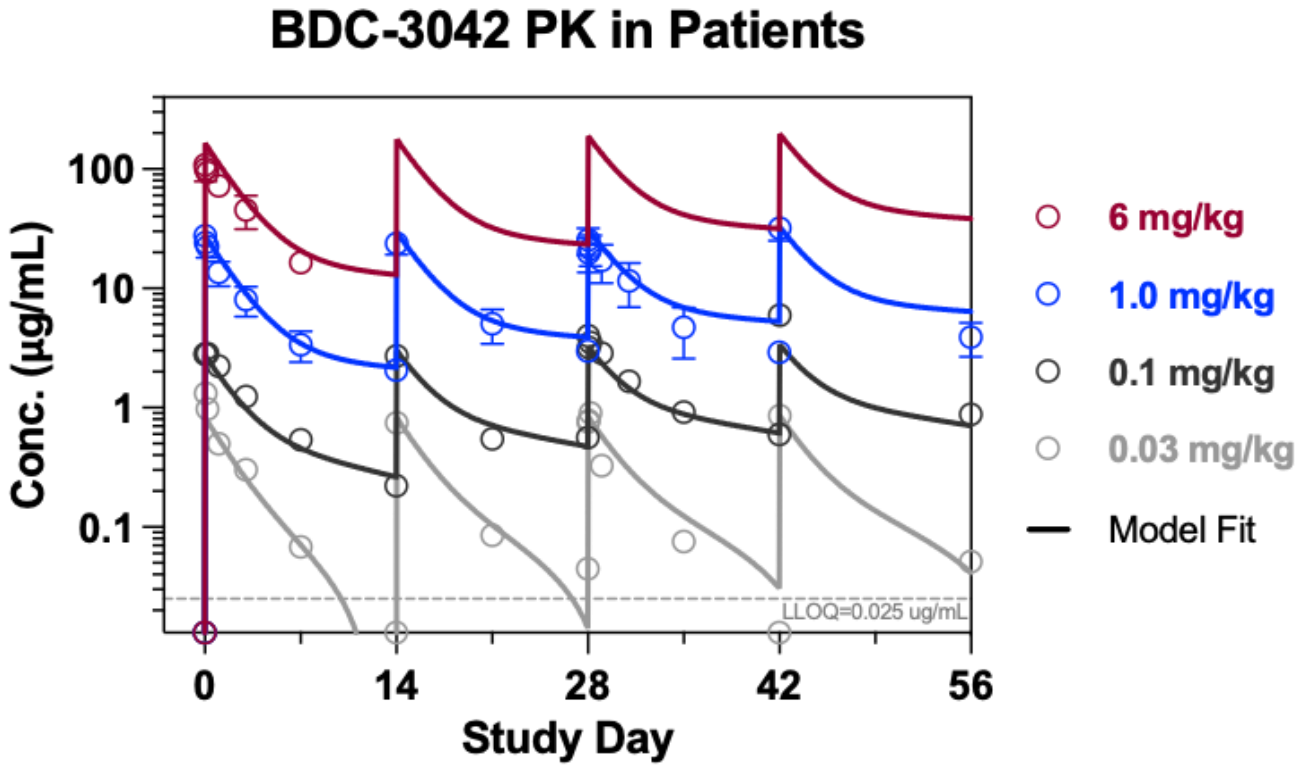
Favorable PK Profile Conducive to Q2W, Q3W, or Q4W Dosing

Typical mAb PK characteristics

- Clearance in linear dose range: 4.4 mL/day/kg
- Half-life in linear dose range: 20 days

Drug accumulation at ≥ 0.1 mg/kg Q2W

No evidence of ADA formation to date



* All data from the 7 cohorts were analyzed by compartmental modeling simultaneously; selected cohorts are shown here for visual clarity

Dumbrava et al., AACR 2025

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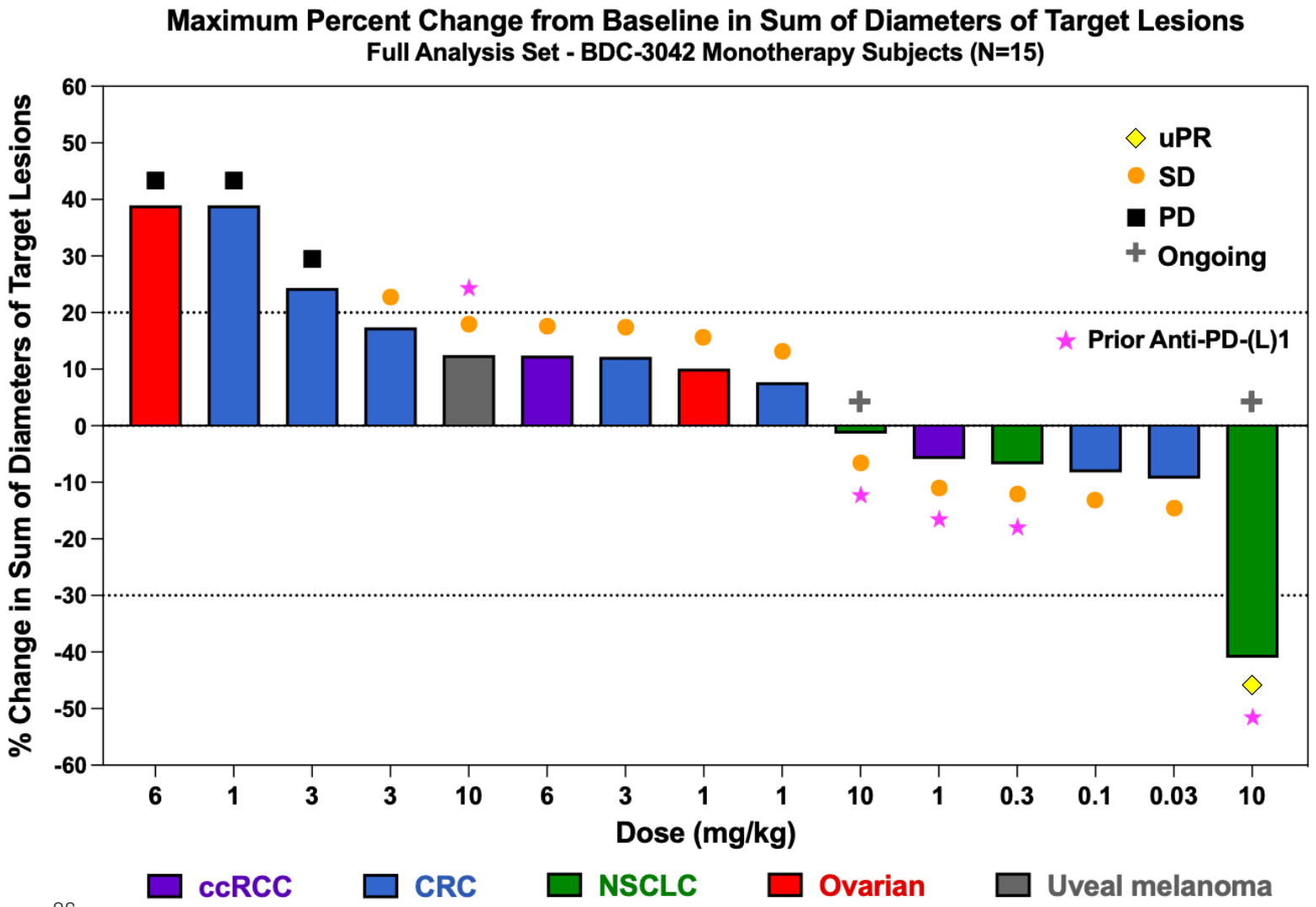
Promising Signs of Efficacy in NSCLC and Post PD-(L)1 Patients



- 3/3 (100%) NSCLC patients had a uPR or SD with some reduction in tumor size
- 4/5 (80%) patients previously treated with PD-(L)1 blockers showed reduction in tumor size[1]
- 12 out of 15 (80%) evaluable patients had SD or better as their best response[2]
- 2/3 patients at 10 mg/kg show tumor reduction and remain on study

[1]Treatment with anti-PD-(L)1 agent within 12 months of enrollment
[2]Two patients were not evaluable (off study prior to initial scan)

Dumbrava et al., AACR 2025. April 7, 2025 data cut-off.

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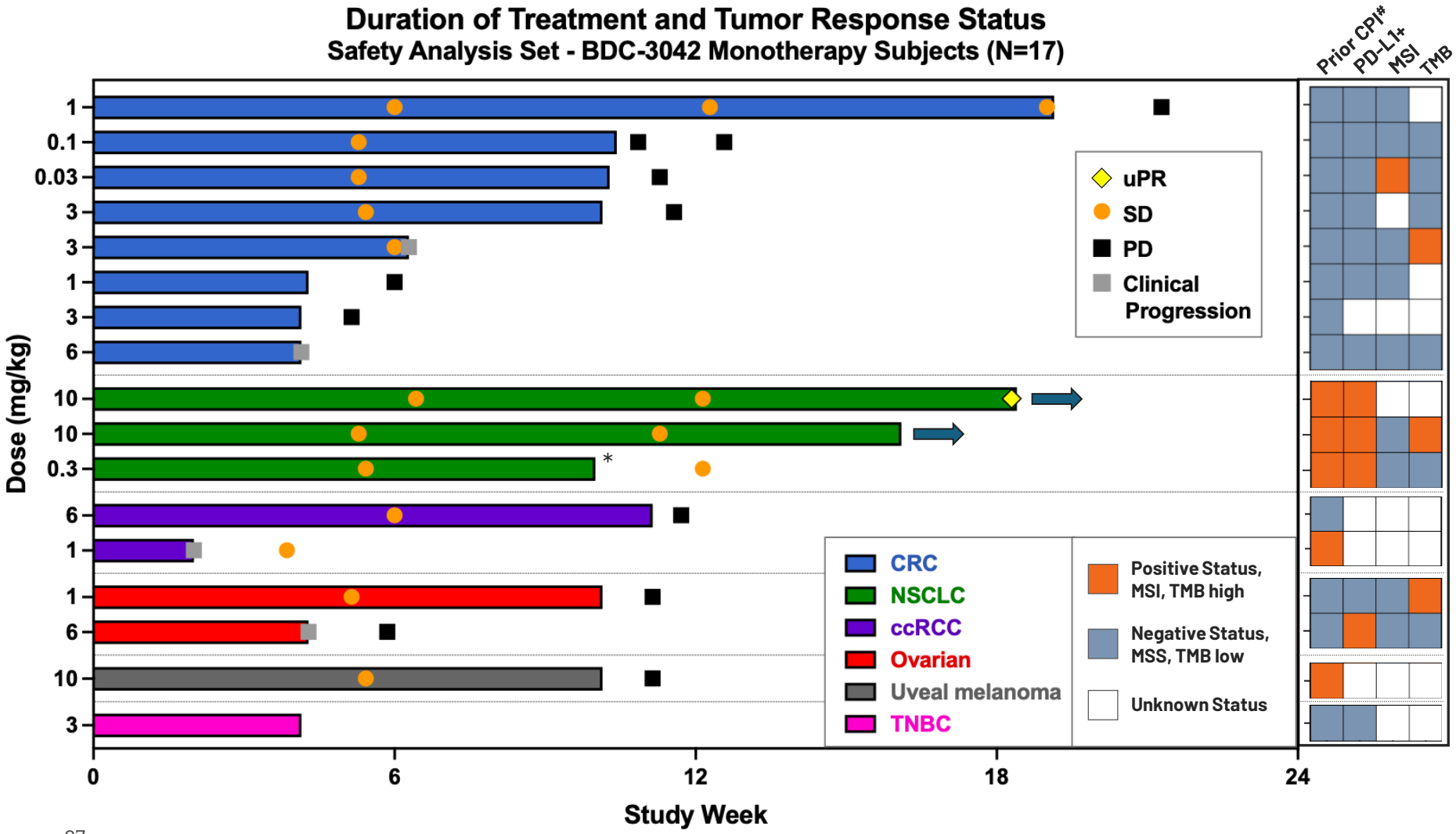
Emerging Signs of Disease Control in NSCLC Patients



- 5/6 CRC pts with data available were MSS
- All NSCLC pts had multiple (≥2) prior lines of αPD-(L)1 therapy

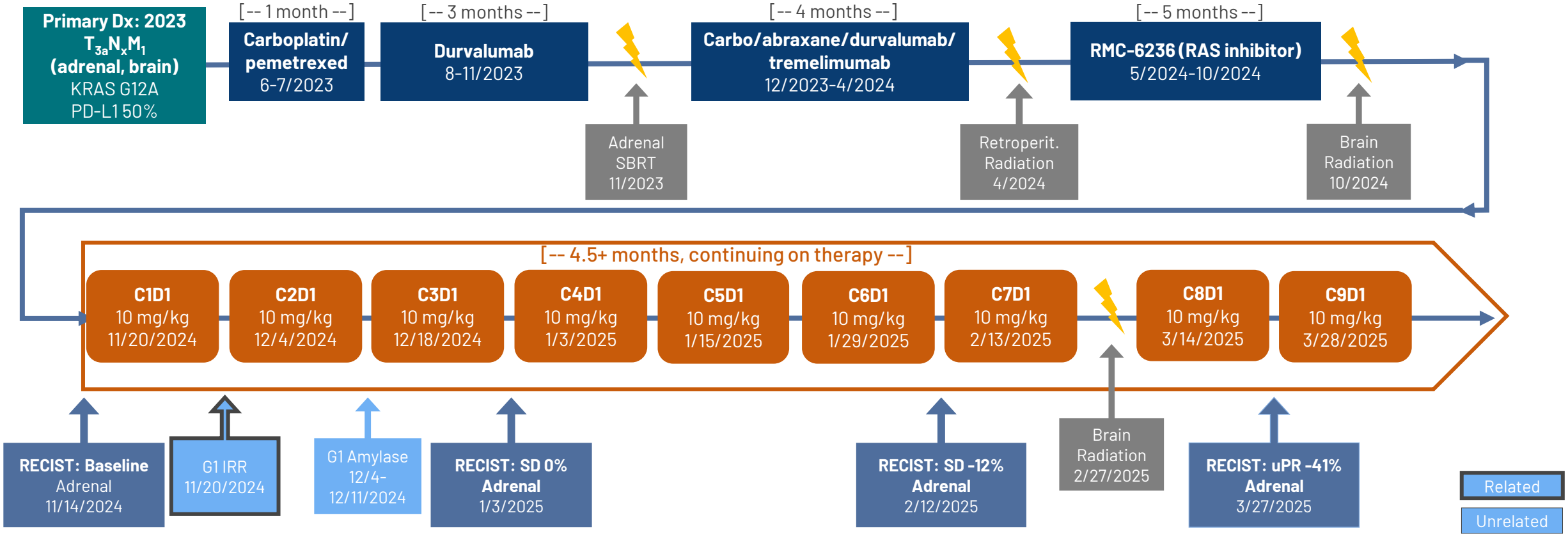
#Prior tx with anti-PD-(L)1 within 12 mo

*0.3 mg/kg NSCLC pt had SD @ 12 wks but came off treatment due to unrelated AE

Dumbrava et al., AACR 2025. April 7, 2025 data cut-off.

Just Confirmed PR in Patient with uPR at the time of the AACR Poster

Cohort 7 (10.0 mg/kg), NSCLC, Continuing BDC-3042 Therapy



BDC-3042 Monotherapy Dose-Escalation Results Summary
Well Tolerated with Promising Signs of Efficacy

Well tolerated up to 10 mg/kg Q2W
- Across all dose levels in a heterogeneous and heavily pretreated population
- Potential for combination with a broad range of therapies

Early clinical support for Mechanism of Action
- Demonstration of target engagement
- Peripheral biomarker responses indicative of innate and adaptive immune activation
- Dose-dependent cytokine responses & modulation of immune populations consistent with preclinical studies

Favorable PK profile
- Opportunity for more convenient dosing—less frequent and aligned with combination agents
- Potential for enhanced PD/efficacy with alternative regimens

Promising signs of efficacy in NSCLC and post-immunotherapy setting
- PR (unconfirmed) in 1 of 3 NSCLC patients (-41% at 18 wks)
- Signs of disease control (SD ≥12 wks) in 3 of 3 NSCLC patients
- Both NSCLC patients in 10 mg/kg cohort remain on study (>16 wks)
- Tumor reductions in 3/3 NSCLC pts, and 4/5 pts post anti-PD-(L)1 therapy

Dumbrava et al., AACR 2025

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BDC-3042 Partnering

Grant Yonehiro

Chief Business Officer

BDC-3042 Partnering

Seek partner to rapidly development and optimize commercialization of BDC-3042

- Open to various structures to maximize value to both parties

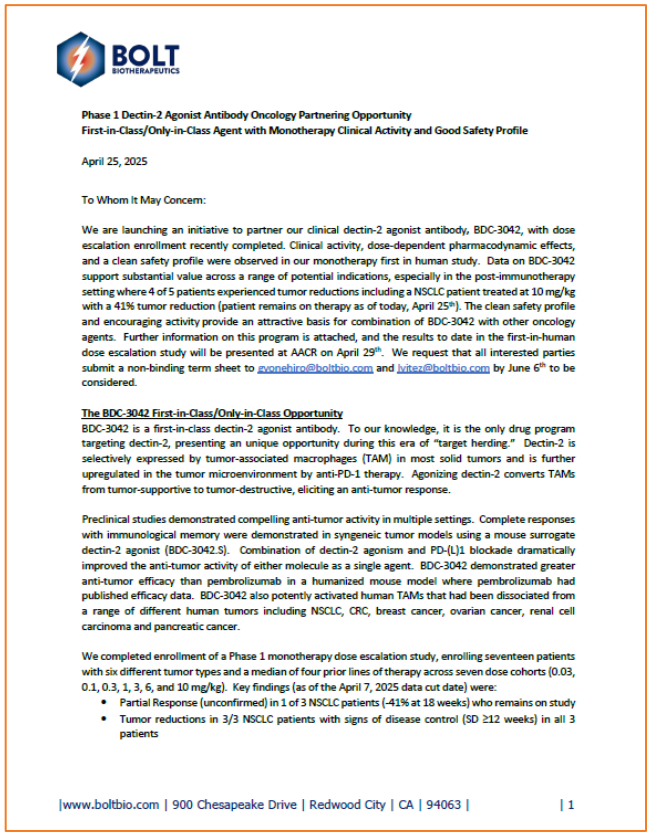
Partnering packet sent to prospective partners with AACR disclosure (April 25)

Confidential & non-confidential discussions ongoing

Non-binding term sheet requested by June 6

- Submit via email to gyonehiro@boltbio.com & lvitez@boltbio.com

First-in-Class/Only-in-class Partnering Opportunity



BOLT BIOTHERAPEUTICS

Phase 1 Dectin-2 Agonist Antibody Oncology Partnering Opportunity
First-in-Class/Only-in-Class Agent with Monotherapy Clinical Activity and Good Safety Profile

April 25, 2025

To Whom It May Concern:

We are launching an initiative to partner our clinical dectin-2 agonist antibody, BDC-3042, with dose escalation enrollment recently completed. Clinical activity, dose-dependent pharmacodynamic effects, and a clean safety profile were observed in our monotherapy first in human study. Data on BDC-3042 support substantial value across a range of potential indications, especially in the post-immunotherapy setting where 4 of 5 patients experienced tumor reductions including a NSCLC patient treated at 10 mg/kg with a 41% tumor reduction (patient remains on therapy as of today, April 25th). The clean safety profile and encouraging activity provide an attractive basis for combination of BDC-3042 with other oncology agents. Further information on this program is attached, and the results to date in the first-in-human dose escalation study will be presented at AACR on April 29th. We request that all interested parties submit a non-binding term sheet to gyonehiro@boltbio.com and lvitez@boltbio.com by June 6th to be considered.

The BDC-3042 First-in-Class/Only-in-Class Opportunity

BDC-3042 is a first-in-class dectin-2 agonist antibody. To our knowledge, it is the only drug program targeting dectin-2, presenting an unique opportunity during this era of "target herding." Dectin-2 is selectively expressed by tumor-associated macrophages (TAM) in most solid tumors and is further upregulated in the tumor microenvironment by anti-PD-1 therapy. Agonizing dectin-2 converts TAMs from tumor-supportive to tumor-destructive, eliciting an anti-tumor response.

Preclinical studies demonstrated compelling anti-tumor activity in multiple settings. Complete responses with immunological memory were demonstrated in syngeneic tumor models using a mouse surrogate dectin-2 agonist (BDC-3042.S). Combination of dectin-2 agonism and PD-(L)1 blockade dramatically improved the anti-tumor activity of either molecule as a single agent. BDC-3042 demonstrated greater anti-tumor efficacy than pembrolizumab in a humanized mouse model where pembrolizumab had published efficacy data. BDC-3042 also potently activated human TAMs that had been dissociated from a range of different human tumors including NSCLC, CRC, breast cancer, ovarian cancer, renal cell carcinoma and pancreatic cancer.

We completed enrollment of a Phase 1 monotherapy dose escalation study, enrolling seventeen patients with six different tumor types and a median of four prior lines of therapy across seven dose cohorts (0.03, 0.1, 0.3, 1, 3, 6, and 10 mg/kg). Key findings (as of the April 7, 2025 data cut date) were:

- Partial Response (unconfirmed) in 1 of 3 NSCLC patients (-41% at 18 weeks) who remains on study
- Tumor reductions in 3/3 NSCLC patients with signs of disease control (SD ≥12 weeks) in all 3 patients

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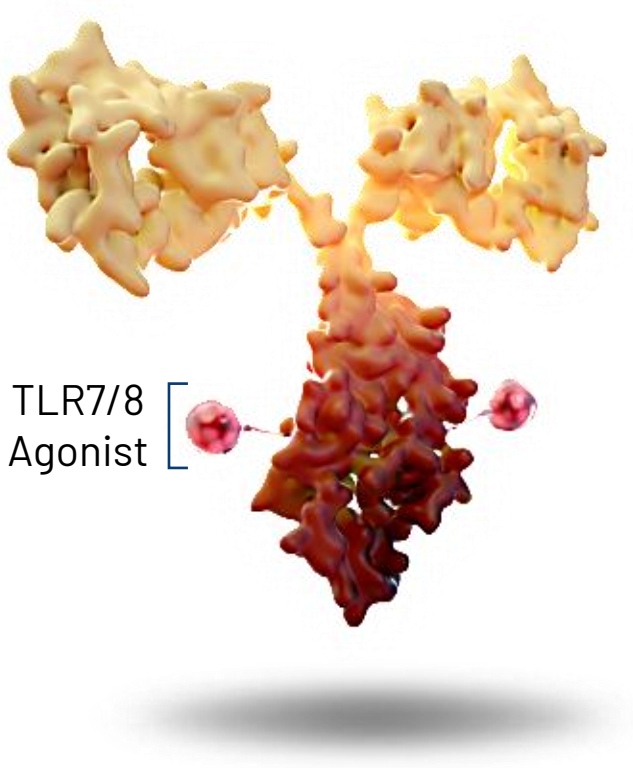
BDC-4182 Update

Jakob Dupont, M.D.

Senior Clinical Advisor & Director

BDC-4182: Claudin 18.2 Boltbody™ ISAC Program

BDC-4182



Opportunity

- Claudin 18.2 is a clinically validated target with a multi-billion-dollar market opportunity
- BDC-4182 success would validate next-generation ISAC platform

Best-in-Class Target Product Profile

- Expands the market to include tumors with lower antigen expression
- Superior efficacy to AZD0901 & TOP1-based ADCs in syngeneic models
- Curative potential (demonstrated durable immunological memory with epitope spreading)
- Safe to combine (toxicology profile and MoA support the ability to combine with SoC)

Key Updates

- Trial open for enrollment

On Track for Key Inflection Points

- 2Q 2025 – Expect first patient to be treated this quarter
- 1H 2026 – Expect preliminary efficacy evaluation from dose escalation study

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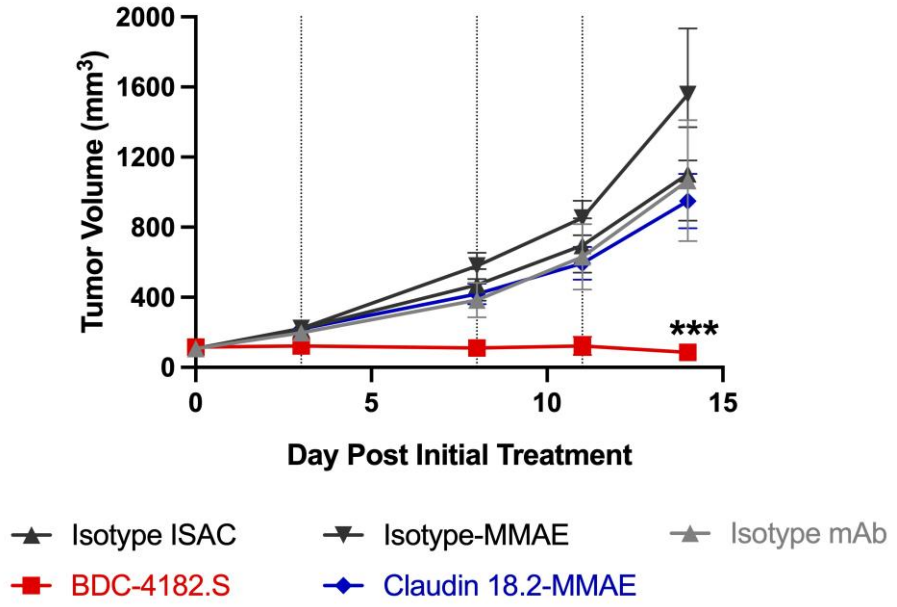
BDC-4182 Activity Superior to MMAE and TOPO1 ADCs in IHC1+ Syngeneic Model

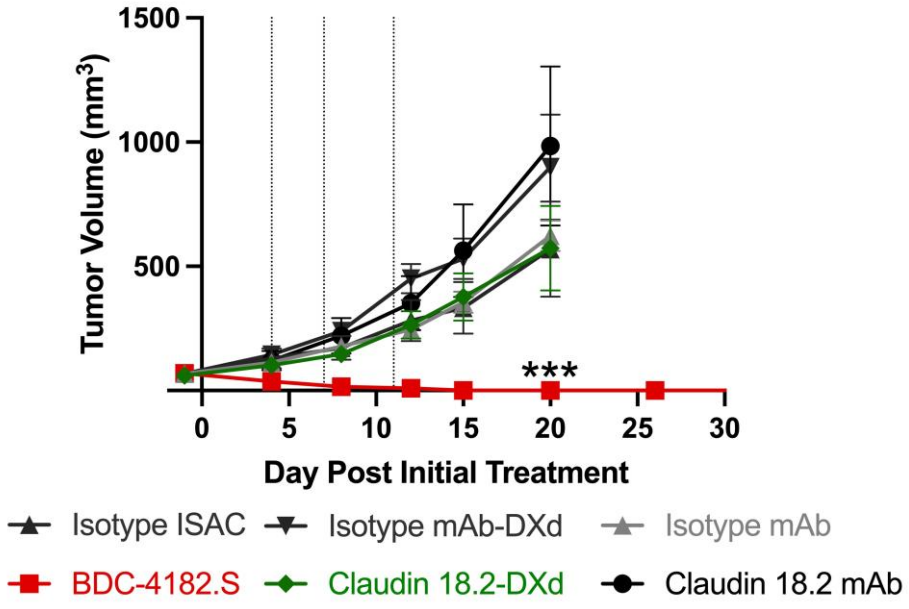
Superior to MMAE ADC

Limited ADC Efficacy in IHC1+ Model



Superior to TOPO1 (DXd) ADC

Limited ADC Efficacy in IHC1+ Model

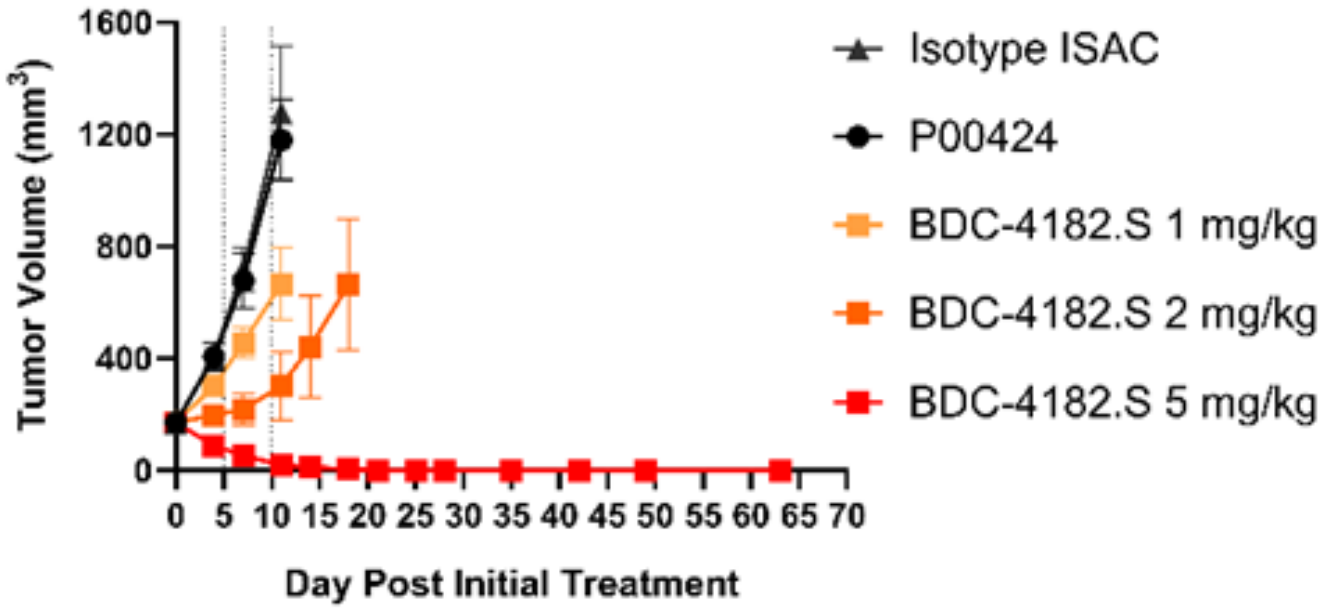


Adapted from Fu CL et al., SITC 2024

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)

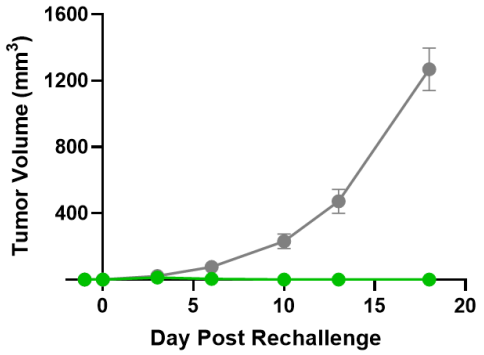
BDC-4182 Has Curative Potential
BDC-4182.S Induces Immunological Memory and Epitope Spreading

MC-38-mCLDN18.2 (IHC 1+)



Immunological Memory
MC38 tumor expressing Claudin 18.2 (IHC 1+)

T cells Prevent Tumor Regrowth

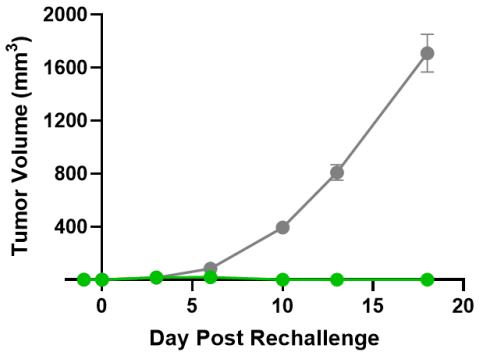


T cell depletion

No T cell depletion

Epitope Spreading
MC38 tumor lacking Claudin 18.2 expression

Claudin 18.2 Expression Not Required



T cell depletion

No T cell depletion

Adapted from Kim SITC 2023

C57BL/6 mice bearing MC38-mCLDN18.2 tumors were treated via IP injection with the indicated test articles every fifth day for a total of three doses (q5dx3)(dashed lines) at the indicated dose levels. Mice with no detectable tumor following treatment with BDC-4182.S were observed for 26 days to ensure no regrowth occurred. These mice (N=4) were rechallenged with MC38 cells expressing CLDN18.2 (75K molecules/cell) or parental MC38 with or without T cell depletion.

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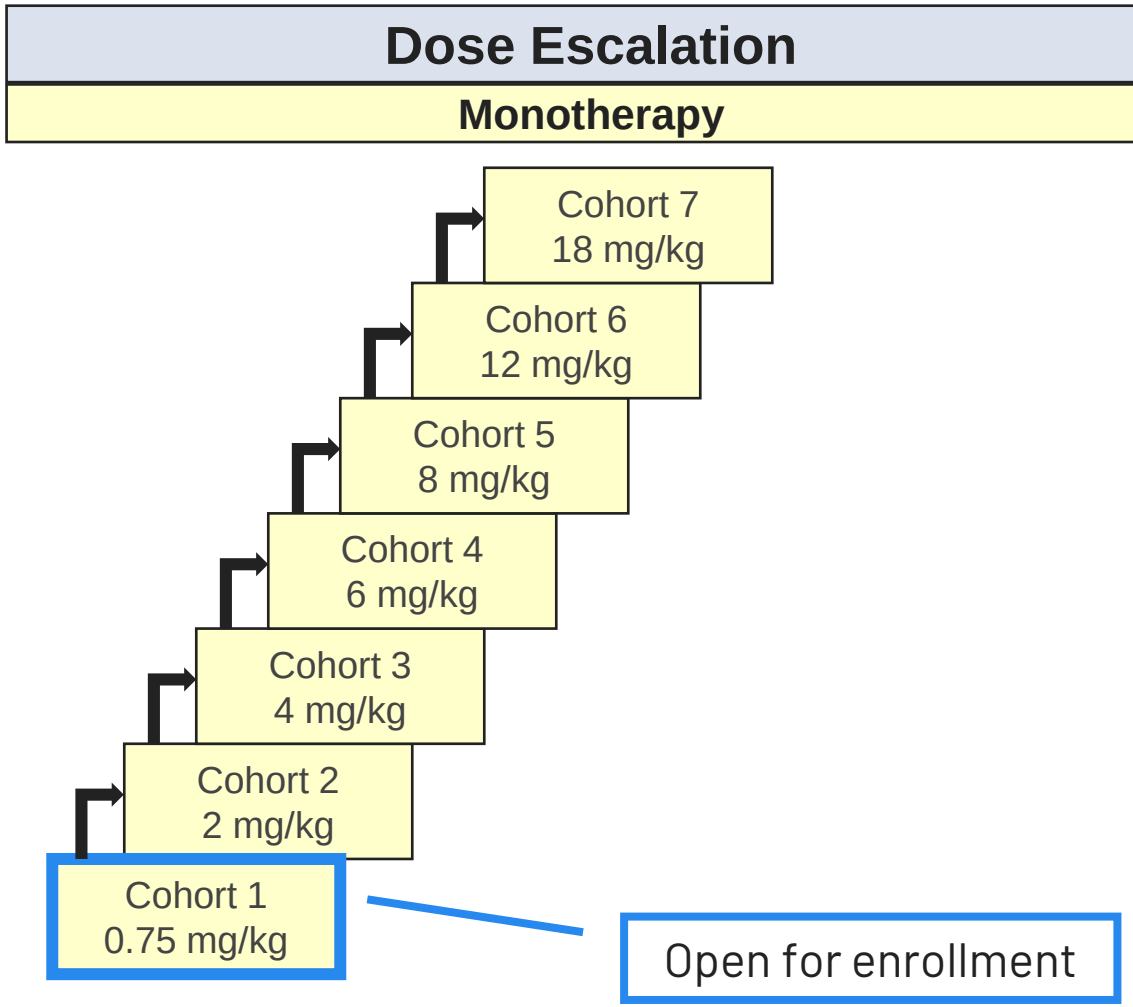
BDC-4182 Clinical Trial Design



Recruiting

A First-in-Human Study Using BDC-4182 as a Single Agent in Advanced Gastric and Gastroesophageal Cancer

ClinicalTrials.gov ID NCT06921837

Sponsor Bolt Biotherapeutics, Inc.

- Initial sites are in Australia
- Enrolling patients with claudin 18.2-expressing tumors

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Conclusion

Willie Quinn

President & CEO

Focused Oncology Pipeline

Portfolio of proprietary and partner-funded programs addressing significant unmet needs

Program (Target)	Indications	Preclinical	Phase 1	Phase 2	Phase 3	Upcoming Milestones
BDC-3042 (Dectin-2 agonist mAb)	Non-small Cell Lung Cancer & Other Solid Tumors		Dose-escalation study — Partnering			Partner selection
BDC-4182 (Claudin 18.2 ISAC)	Gastric Cancer		Open for enrollment			First patient enrolled 2Q 2025
CEA ISAC	Colorectal Cancer, Non-small Cell Lung Cancer	Lead selected				
PD-L1 ISAC	Solid Tumors Resistant to Checkpoint Inhibitors					

Boltbody™ ISAC Collaborations

Genmab	Funds 3 bispecific Boltbody ISACs through early clinical development
TORAY (Caprin-1 ISAC)	Funds Boltbody ISAC targeting Caprin-1 through early clinical development

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Concluding Thoughts

BDC-4182 (Claudin 18.2 Boltbody™ ISAC)

- Next-gen ISAC targeting gastric & gastroesophageal cancers
- Clinical trial initiating in 2Q 2025

BDC-3042 (Dectin-2 Agonist Antibody)

- First-in-class antibody with monotherapy anti-tumor activity
- Reported results at AACR & initiated partner process

Efficient drug development

- Existing cash[1] funds key milestones & operations to mid-2026
- Collaborations fund themselves & provide future upside

ISAC = Immune-stimulating antibody conjugate

[1] Cash, cash equivalents, and marketable securities balance of $58 million as of 3/31/2025

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Thank you.

Nasdaq: BOLT

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

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Questions?

Nasdaq: BOLT

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