Leveraging the power of the innate and adaptive immune systems to address key unmet needs in cancer

November 2021

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, results of operations, business strategy and plans, and objectives of management for future operations, as well as statements regarding industry trends, 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 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 into 2023; the impact of the COVID-19 pandemic on our operations; the commercialization of our product candidates, if approved; our plans to research, develop and commercialize our product candidates; our ability to obtain, maintain, expand, protect and enforce our intellectual property rights; our ability to operate our business without infringing, misappropriating or otherwise violating the intellectual property rights of third parties; 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 Bristol-Myers Squibb Company, Innovent Biologics, Inc., 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; regulatory developments in the United States and foreign countries; our ability to contract with third-party suppliers and manufacturers and their ability to perform adequately; the success of competing therapies that are or may become available; and our ability to attract and retain key scientific or management personnel. 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, 2020. 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.

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BOLT BIOTHERAPEUTICS

Progress in Our Pioneering Journey

First-in-class Boltbody™ ISAC: BDC-1001

- Ongoing Phase 1/2 in patients with HER2-expressing solid tumors
- BDC-1001 was well tolerated in the first 20 patients with early signs of clinical activity including a PR and changes in biomarkers (data as of 1/29/21)
- Upcoming data update on total of ~50 patients at ESMO I/O (December 2021)

Broadly Enabling Platform Technology

- Expertise in antibody selection and immune-stimulating linker-payloads
- Preclinical data demonstrates ISAC activity with diverse tumor-targeting antibodies
- Growing pipeline of proprietary and partnered programs

Cash on Hand Achieves Key Milestones

- Cash of $295.5 million[1] expected to fund operations through 2023
- Funded through key milestones for BDC-1001 & BDC-2034

[1] Proforma Cash, cash equivalents, and marketable securities as of September 30, 2021: $290.5 million balance plus $5 million Innovent receivable

BOLT BIOTHERAPEUTICS

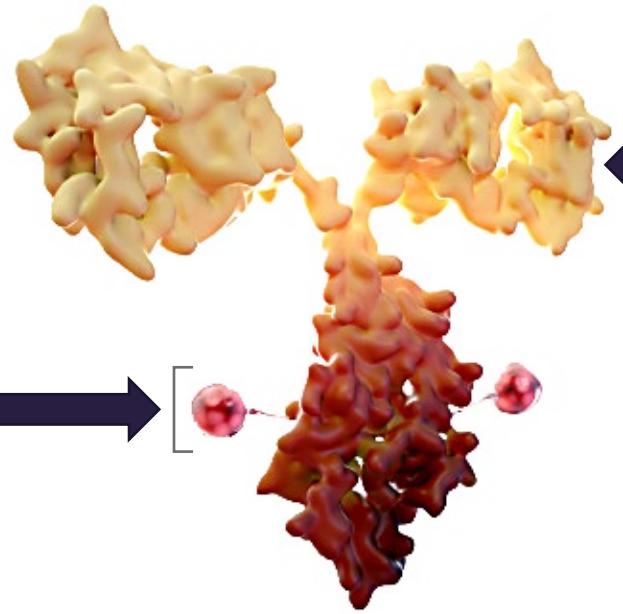
Pioneering a New Class of Immuno-oncology Products

Immune-stimulating Antibody Conjugates (ISACs)

Boltbody™ ISAC

Immune-stimulating Linker-payload

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



Tumor-targeting Antibody

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

BOLT BIOTHERAPEUTICS

Robust Pipeline of Boltbody™ ISACs and Myeloid Modulator

	Candidate	Target Antigen	Indications	Preclinical	Phase 1	Phase 2	Phase 3	Bolt Commercial Rights
Boltbody ISACs	**BDC-1001**	HER2	• HER2+ Breast Cancer • HER2 Low Breast Cancer • HER2+ Gastric Cancer • Other HER2+ Cancers	Ongoing Phase 1/2 Trial				Global
	BDC-2034	CEA	• NSCLC • CRC • Pancreatic Cancer • Breast Cancer					Global
	PD-L1 Program	PD-L1	Checkpoint Refractory Tumors • NSCLC & SCLC • CRC • Breast Cancer					Global
Agonist Antibody	**Myeloid Modulator**	Dectin-2*	Tumors with: • KRAS mutations • TP53 mutations					Global

*Dectin-2 (also known as TAM1)

BOLT BIOTHERAPEUTICS

Growing Our Pipeline Through Strategic Collaborations

Innovent

Fully integrated biopharma with large antibody library and strong presence in Greater China

- Innovent funds 3 Boltbody ISACs through early clinical development
- Bolt has option to co-develop & commercialize 2 candidates in certain regions
  - Bolt received $5M upfront; possible future equity investment of up to $10M, plus milestones and royalties

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Genmab

Innovative leader in antibody & bispecific development for oncology

- Genmab funds 3 bispecific Boltbody ISACs through early clinical development
- Bolt has option to co-develop & commercialize 1 candidate in certain regions
  - Bolt received $25M upfront; eligible for up to $285M in milestones + tiered royalty per program exclusively developed & commercialized by Genmab

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TORAY

Global leader in innovative technologies, conducting research in cancer immunotherapeutics

- Toray funds Boltbody ISAC for specific & novel target through end of Phase 1
- Global co-development/co-commercialization

BOLT BIOTHERAPEUTICS

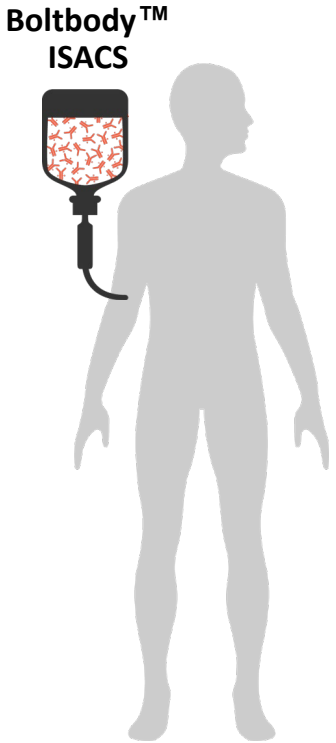
Boltbody™ ISAC Mechanism Targets the Innate Immune System

Spreads to Adaptive Immune System for Optimal Anti-tumor Response

Innate Immune Response → **Adaptive Immune Response**

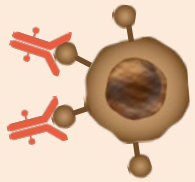
Myeloid Cells Kill Tumor Cells via ADCP

Engages T Cell-driven Tumor Killing



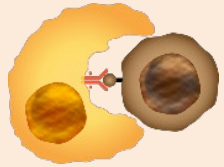
TUMOR MICROENVIRONMENT

① Tumor Antigen Recognition



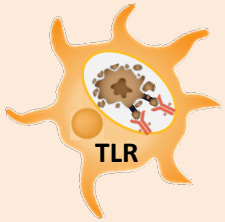
Antigen Expression
- High, medium, & low

② FcR-Dependent Phagocytosis



Myeloid Antigen-Presenting Cells
- Monocytes
- Macrophages
- pDCs and cDCs

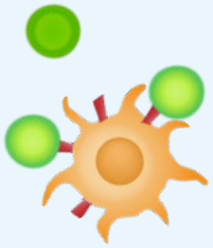
③ TLR-Mediated Activation



Activated Myeloid Cells
- Chemokine & cytokine secretion
- Enhanced antigen presentation

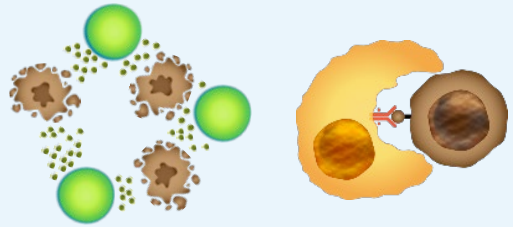
TUMOR-DRAINING LYMPH NODES

④ T cell Priming & Expansion



TUMOR MICROENVIRONMENT

⑤ T Cell Killing of Tumor Cells



Result: An Immune-"Hot" Tumor
- Chemokines attract immune effector cells
- Cytokines lower immune activation threshold
- Increases myeloid APC phagocytosis
- Activated T cells migrate to tumor


Boltbody™ ISAC

Tumor Antigen


Tumor Cell


Dying Tumor Cell


Myeloid APC


T Cell


Activated T Cell


MHC-Tumor Peptide Complex


Granzymes, IFNγ, TNFα

BOLT BIOTHERAPEUTICS

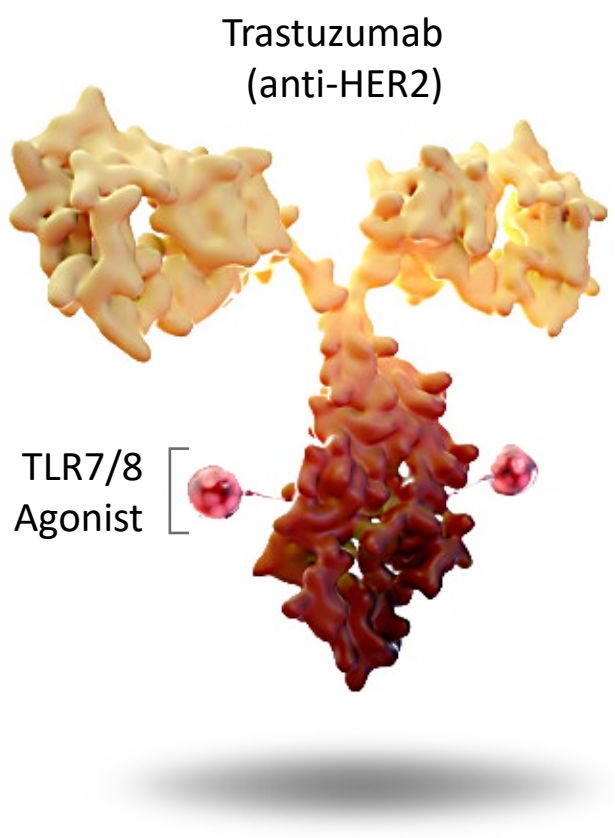
BDC-1001

HER2-Directed Boltbody™ ISAC

BDC-1001: Generating Proof of Mechanism for Our Boltbody ISAC Approach

Treatment of HER2-Expressing Solid Tumors

BDC-1001



Trastuzumab biosimilar (anti-HER2) conjugated to a proprietary TLR7/8 agonist via a non-cleavable linker

Early Clinical Proof of Concept Achieved in Phase 1/2 Trial

- 20 patients treated through January 29, 2021
- Well-tolerated: no DLTs, no drug-related SAEs
- Promising signs of clinical activity: stable disease & tumor volume reductions, including a PR by RECIST 1.1
- Pharmacodynamic biomarkers consistent with MOA

Compelling preclinical anti-tumor activity in large tumors with immunological memory, and clean NHP toxicology profile

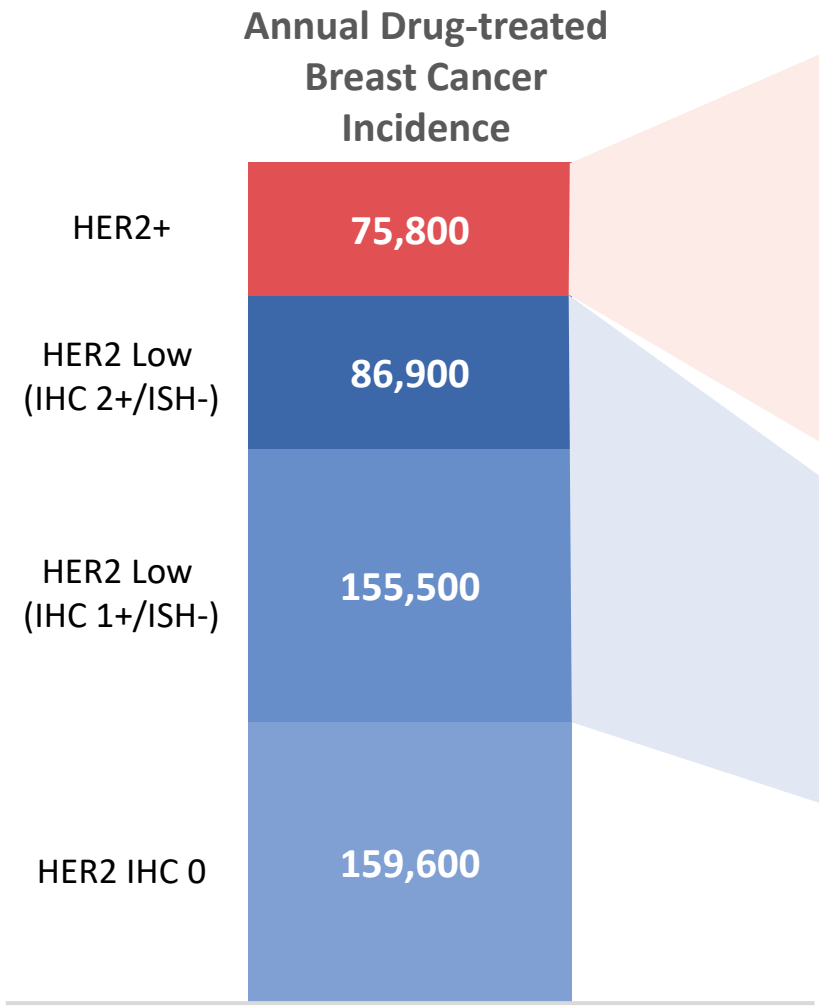
Expected Milestones

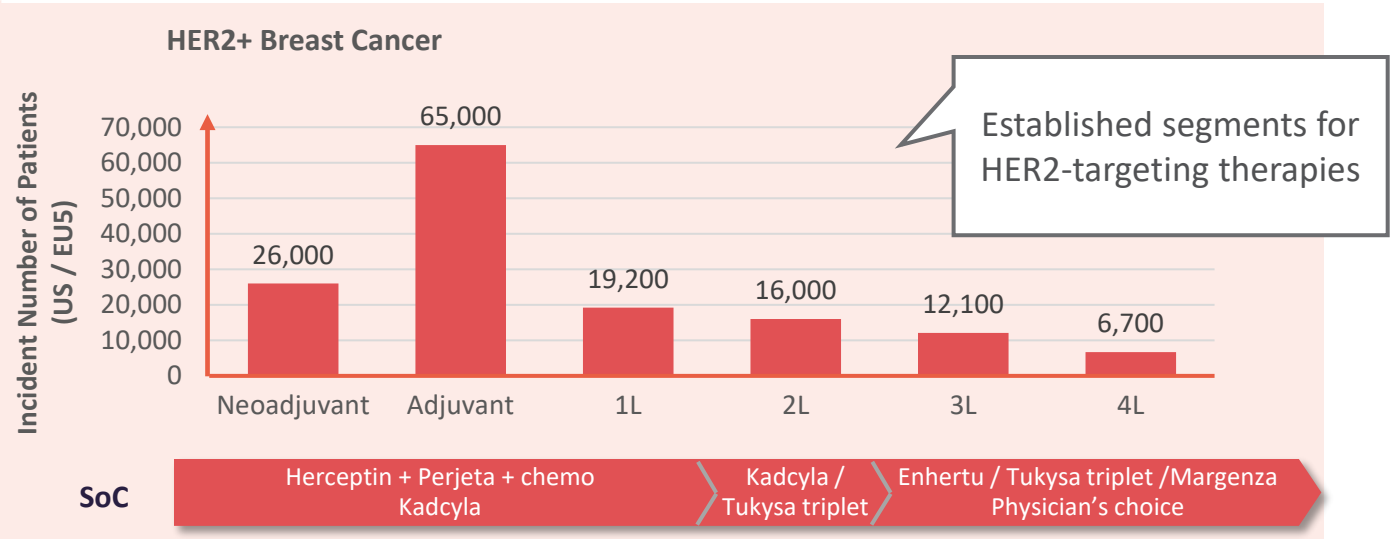
- 4Q21: Initiate combination trial with anti-PD-1
- 2022: Complete monotherapy dose escalation
- 2022: Initiate monotherapy Phase 2 dose expansions

BOLT BIOTHERAPEUTICS

HER2+ Breast Cancer: Large Initial Market with Significant Unmet Needs

Additional Opportunities in HER2-Low & Other HER2-Expressing Cancers



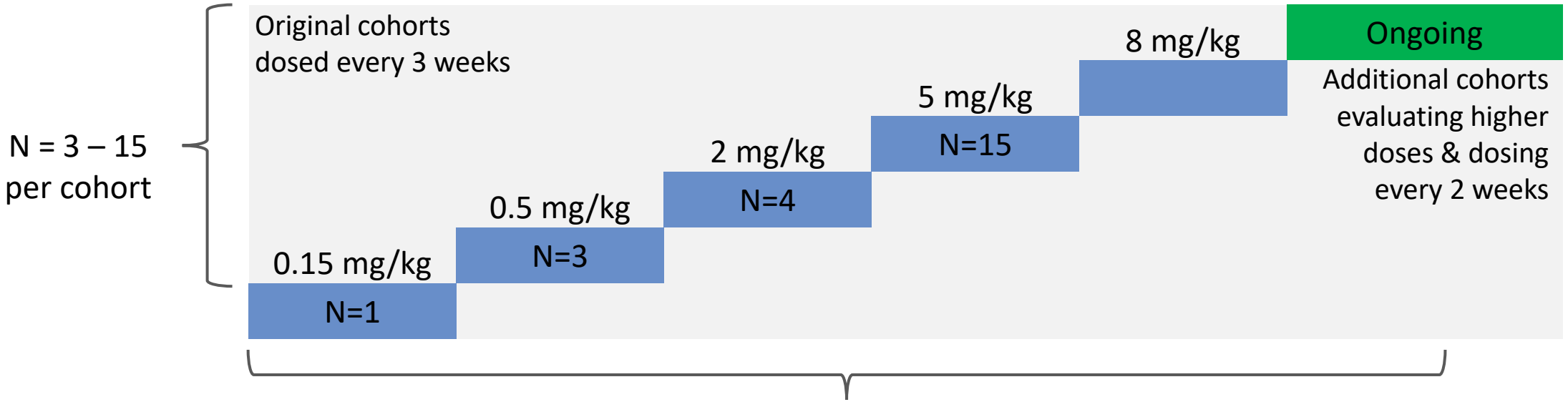


Significant HER2-low Opportunity
No approved HER2-targeting therapy

Numbers for US + EU4 + UK. Source: Company estimates; Globe Life Sciences research commissioned by Bolt; patient numbers are from 2018 Roche Potential Target Patient Population Appendix with additional segmentation based upon HER2 segmentation from Arch Pathol Lab Med (2014).

BOLT BIOTHERAPEUTICS

BDC-1001 Monotherapy Dose Escalation in Ongoing Phase 1/2

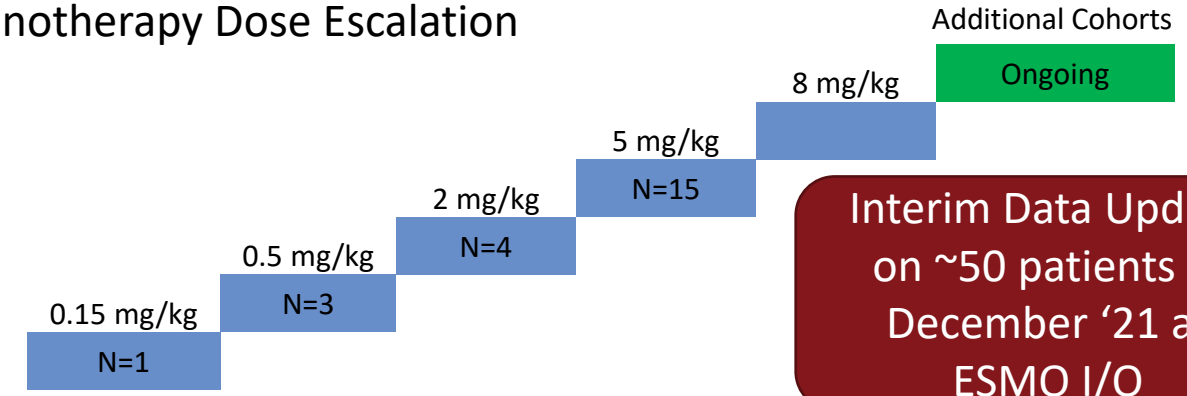


Will present interim data update on ~50 patients
in December at ESMO Immuno-Oncology Congress 2021

Primary Endpoints	Safety, Dose Selection
Other Endpoints	PK, preliminary anti-tumor activity, biomarkers to explore proof of mechanism
Eligibility	Any HER2-expressing solid cancer: • HER2 IHC2+/3+ or • HER2-amplified

BDC-1001 Phase 1/2 Trial Design

Part 1
Monotherapy Dose Escalation



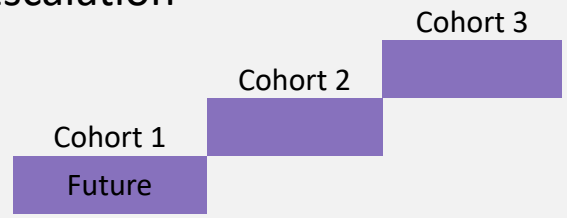
Interim Data Update on ~50 patients in December '21 at ESMO I/O

Part 3
Phase 2 Monotherapy Dose Expansion

- Multiple defined cohorts
  - Tumor types (e.g. colorectal, breast)
  - HER2 expression
- Two-stage design
  - Expand to 30 with positive signal

Part 2
Combination Dose Escalation

Starting in December '21



Opdivo® (nivolumab) Anti-PD1 Antibody Supplied through Collaboration with Bristol Myers Squibb

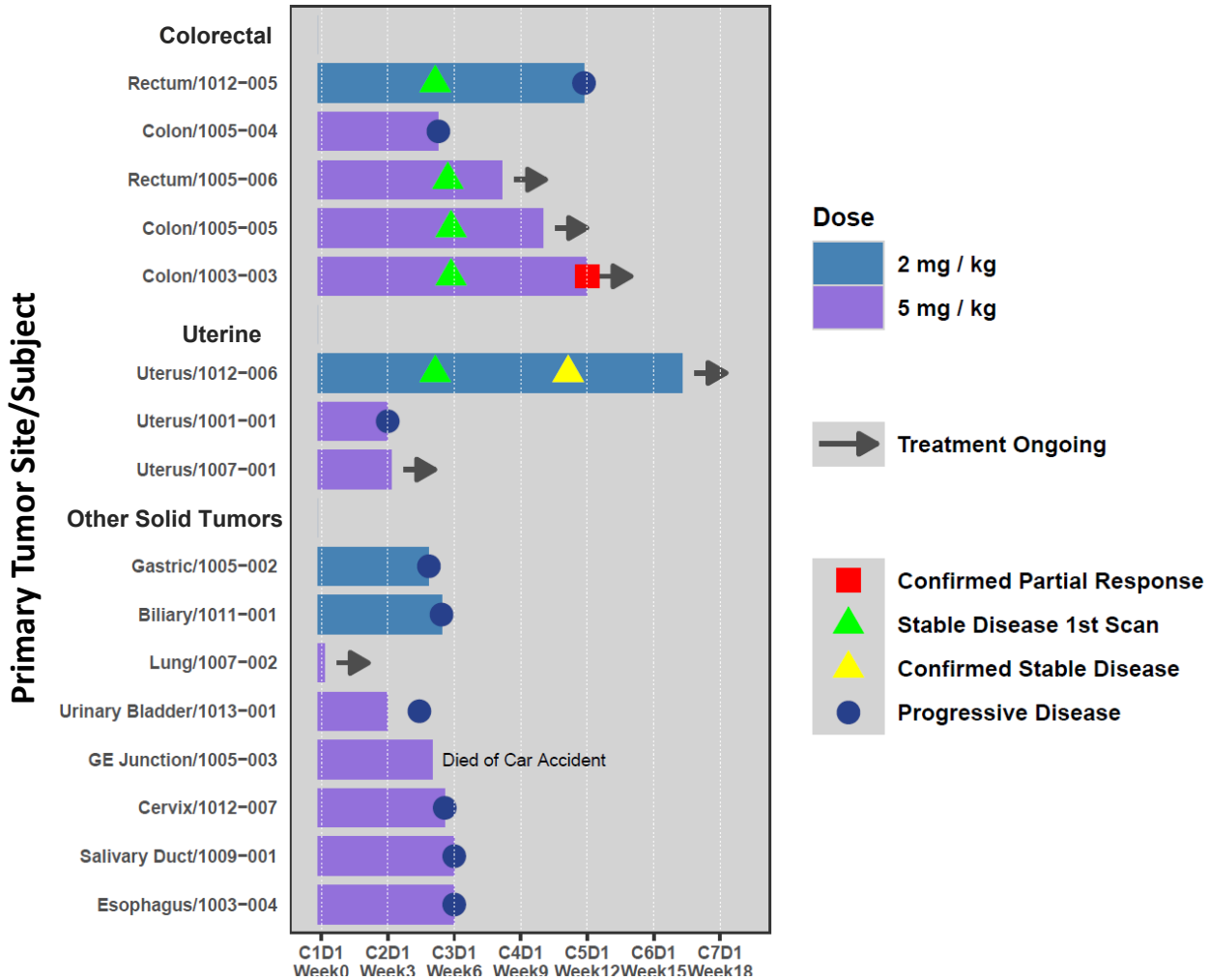
Part 4
Phase 2 Combination Dose Expansion

- Multiple defined cohorts
  - Tumor types (e.g. colorectal, breast)
  - HER2 expression
- Two-stage design
  - Expand to 30 with positive signal

BOLT BIOTHERAPEUTICS

Preliminary BDC-1001 Clinical Results Demonstrate Promising Clinical Activity

2 mg/kg and 5 mg/kg Cohorts



BDC-1001

- Patients with advanced refractory tumors, progression documented upon enrollment
- No DLTs or drug-related SAEs
- Reduction in tumor volume, including a PR by RECIST 1.1 and stable disease observed in patients with MSS tumors
- Plasma biomarkers consistent with mechanism of action
  - Myeloid cell activation: MCP-1, MIP1α, IP-10
  - TLR stimulation: TNFα

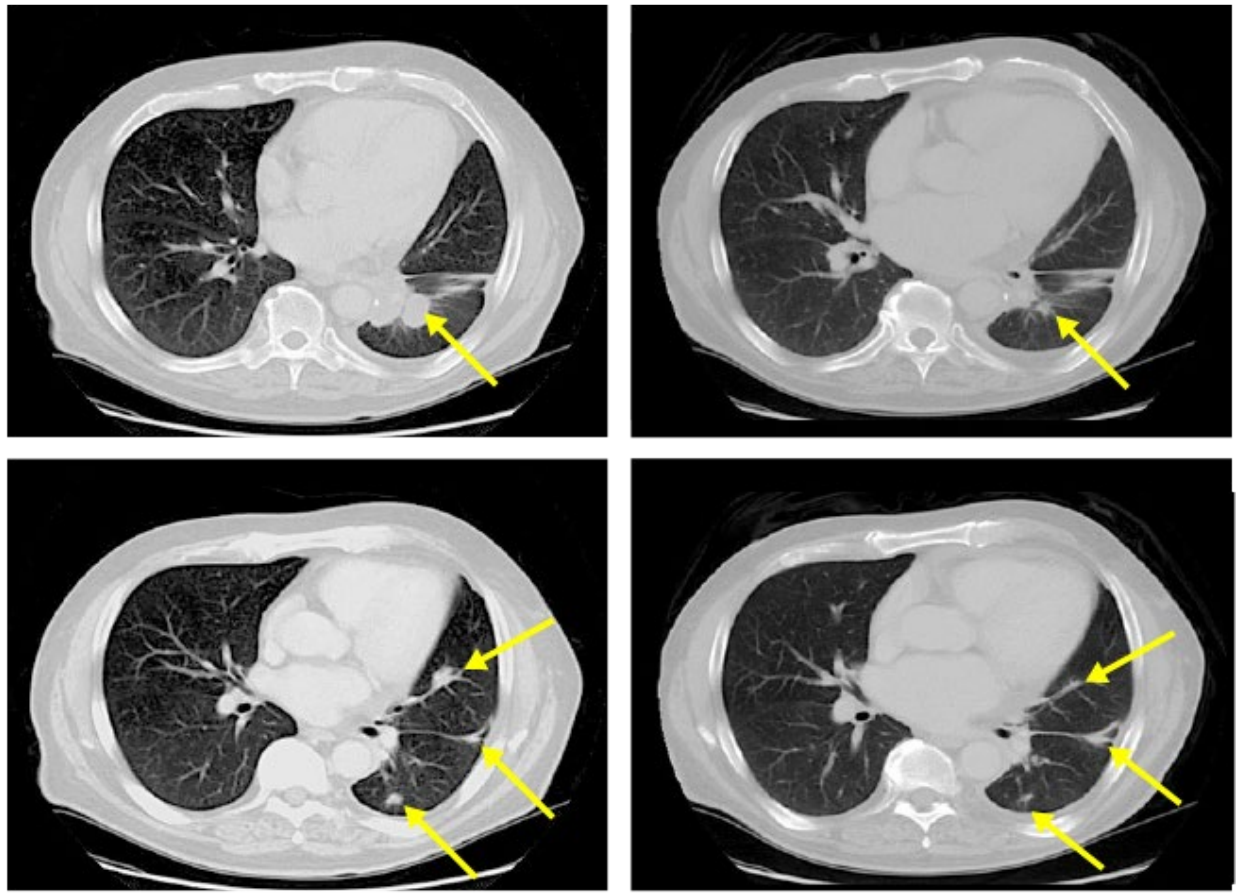
ASCO 2021; data as of January 29, 2021

Confirmed Partial Response (PR)

39% Reduction in Tumor Lesions in Patient with MSS Colon Cancer

Prior to BDC-1001 Treatment

12 weeks on BDC-1001 Treatment



66-year-old male (patient 1003-003) with progressive adenocarcinoma of the colon, metastatic to lungs

- **Tumor progression after multiple prior therapies**, including chemotherapy, radiation therapy, and PD-1 inhibitor
- Tumor HER2+ (IHC3+, amplified FMI); microsatellite stable, KRASwt

39% reduction of the sum of the longest diameters of all four measurable lesions after cycle 4

ASCO 2021; data as of January 29, 2021

BOLT BIOTHERAPEUTICS

Three Additional Patients with Evidence of Disease Control

All MSS Tumors with Progressive Disease and Visceral Metastases

2 mg/kg Cohort	**1012-006 – patient with confirmed stable disease**	• 84-year-old female with endometrial cancer (lung metastases) • Multiple prior therapies including Herceptin + Perjeta • Treated with 6 cycles of BDC-1001 to date, remains on study
5 mg/kg Cohort	**1005-005 – patient with stable disease**	• 71-year-old female with metastatic colorectal cancer (lung and liver metastases) • Multiple prior therapies including Herceptin • Treated with 3 cycles of BDC-1001 to date, remains on study
	1005-006 - patient with stable disease	• 73-year-old female with metastatic colorectal cancer (lung metastases) • Multiple prior treatments including Enhertu and the anti-HER2 bispecific ZW25 • Treated with 3 cycles of BDC-1001, remains on study

ASCO 2021; data as of January 29, 2021

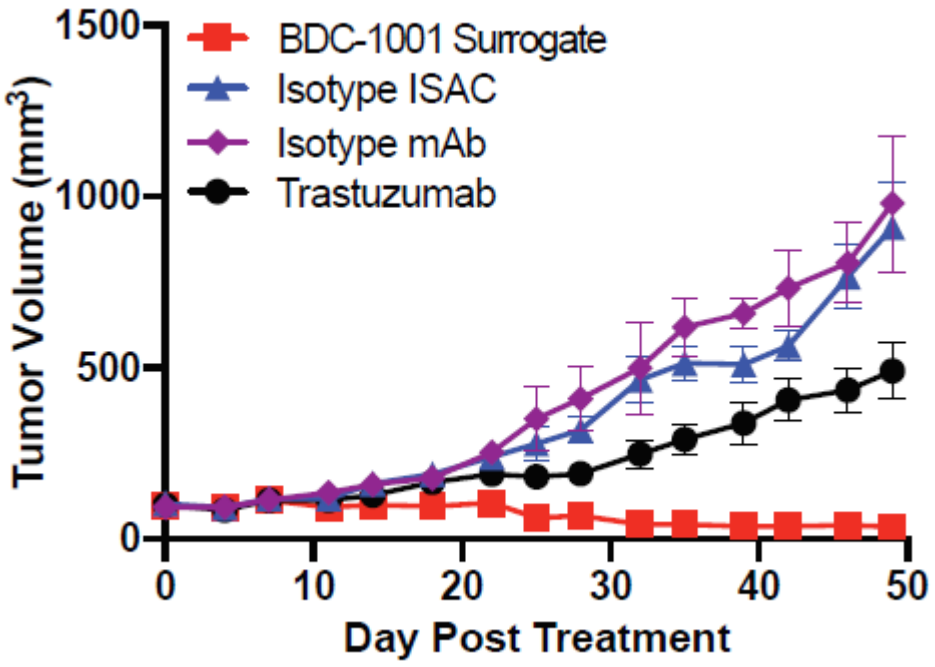
BOLT BIOTHERAPEUTICS

“Three-Factor Authentication” Provides For Broad Safety Window

Support for ADCP-driven Mechanism in a Model without T Cells

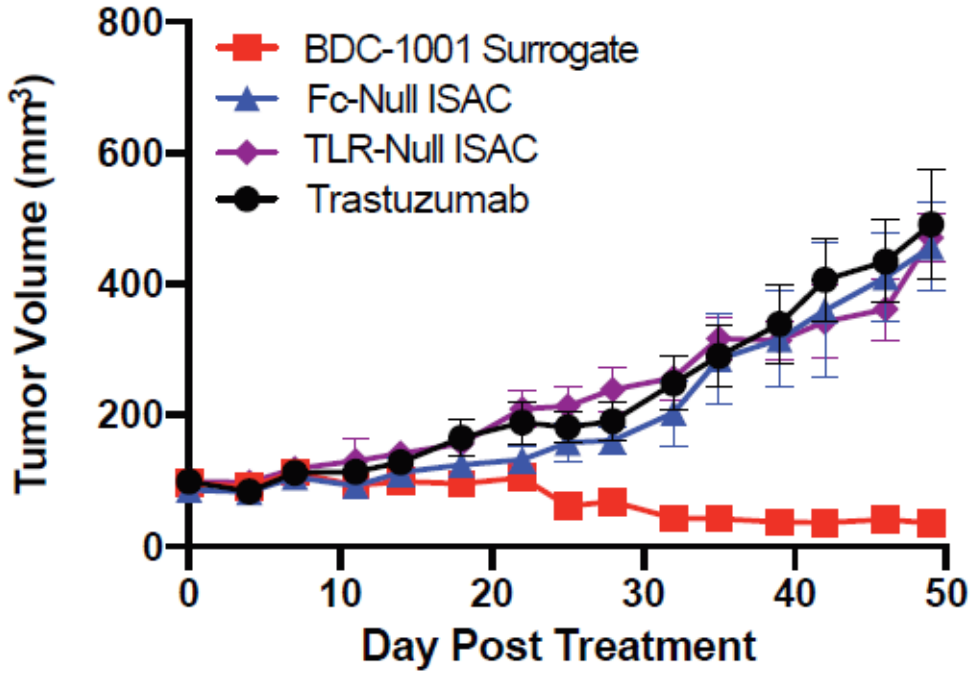
Activity dependent on
1) Tumor antigen recognition

Mice implanted with HER2-expressing HCC1954 cell line



Activity dependent on both
2) FcR engagement & 3) TLR agonism

Mice implanted with HER2-expressing HCC1954 cell line



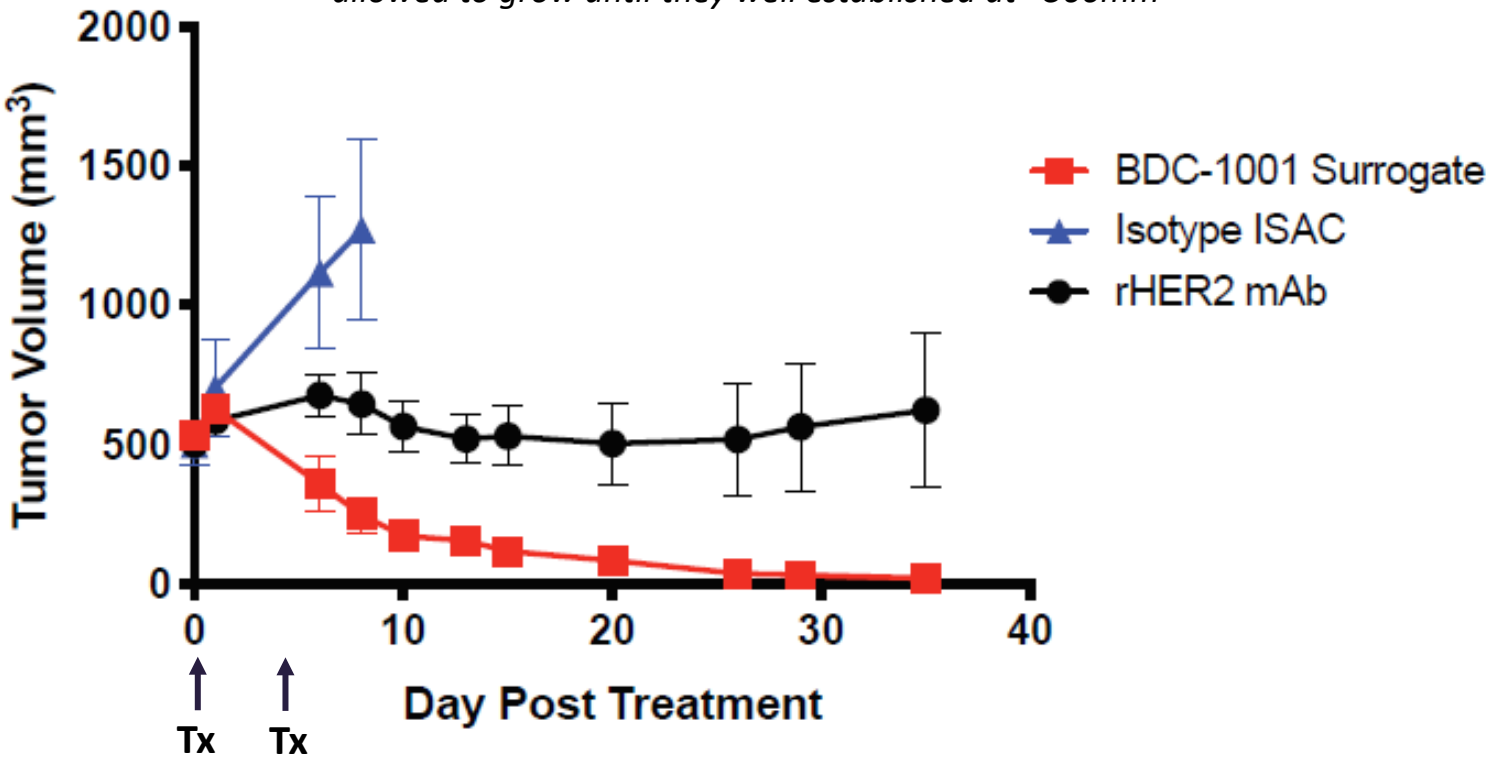
Ackerman SE, et al., *Nature Cancer* (2020)
Note: NSG mice were dosed systemically with 5 mg/kg every 5 days through day 25. Data are shown as mean ± SEM with 5 mice per group.

BOLT BIOTHERAPEUTICS

Eliminated Large, Immunologically Cold & Well-established Tumors

Syngeneic tumor models add the adaptive immune system, allowing for exploration of the full potential for the Boltbody™ ISAC mechanism

Mice implanted with MMC tumor cell line; tumors were allowed to grow until they well established at ~500mm³

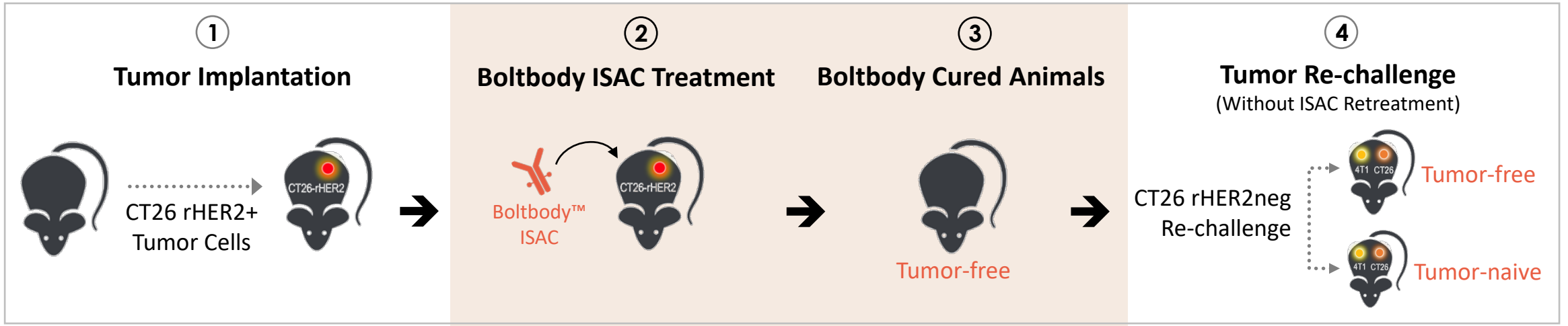


Ackerman SE, et al., *Nature Cancer* (2020)

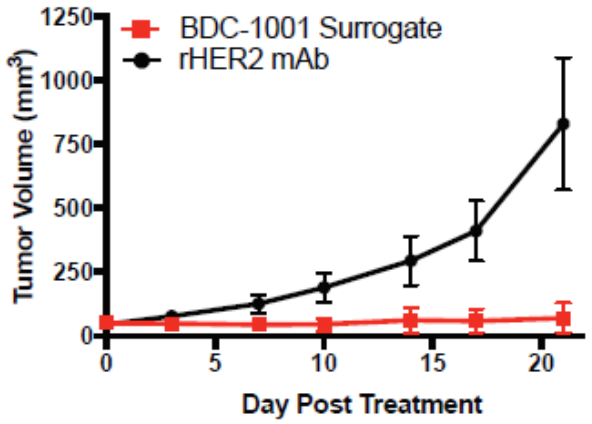
Note: FVB Erbb2 transgenic mice were dosed systemically with 5 mg/kg on days 0 and 5. Data are shown as mean ± SEM with 4-7 mice per group.

BOLT BIOTHERAPEUTICS

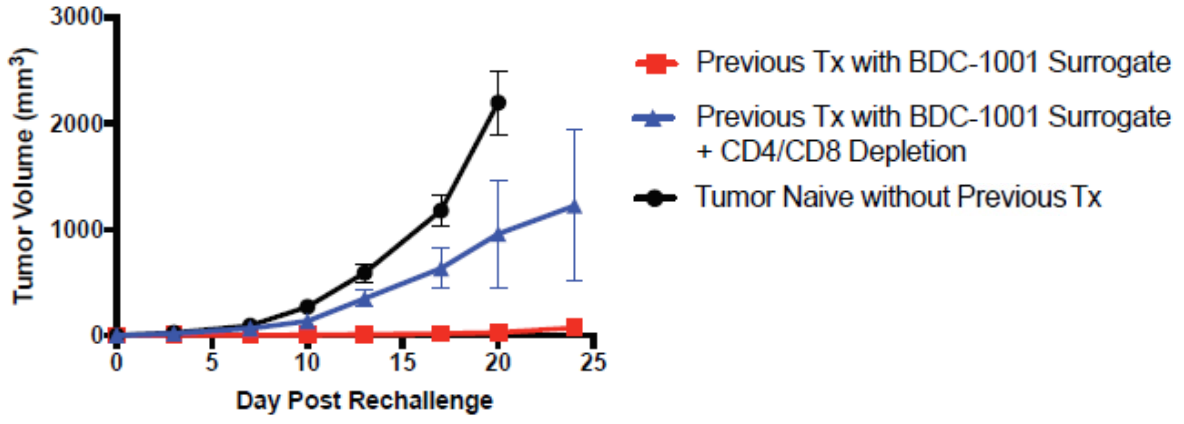
Broad Retraining of Immune System to Recognize Neoantigens with Epitope Spreading



Initial CT26-rHER2 Treatment



Re-challenge with CT26 Tumors without rHER2



Ackerman SE, et al., *Nature Cancer* (2020)

Note: Balb/c mice were dosed systemically with 10 mg/kg every 5 days through day 25. Mice that eliminated their tumors for >21 days after the last treatment with BDC-1001 surrogate or tumor naïve mice were challenged with CT26 tumor cells without rHER2 expression and 4T1 tumor cells. Data are shown as mean ± SEM with 3-8 mice per group.

BOLT BIOTHERAPEUTICS

BOLT BIOTHERAPEUTICS

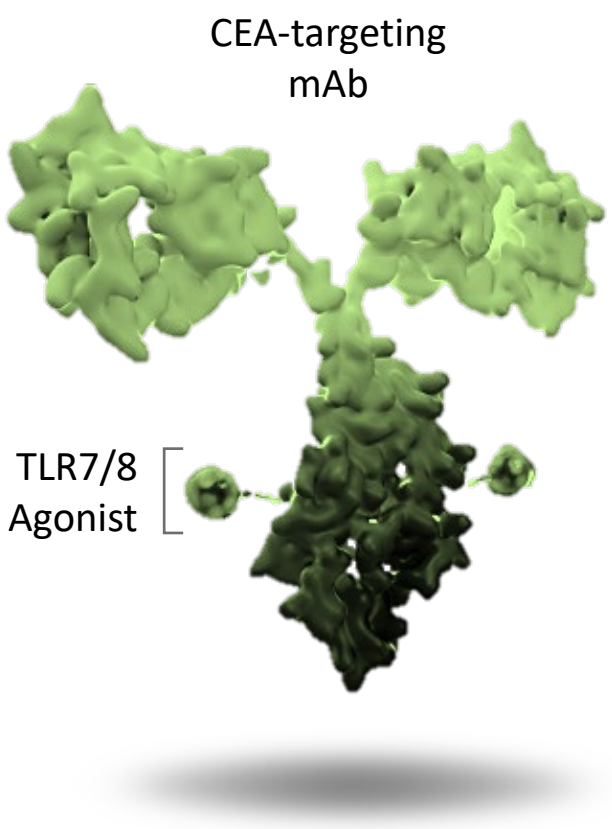
Pipeline Overview

- **BDC-2034 CEA-Directed Boltbody ISAC**
- **PD-L1 Boltbody ISAC Program**
- **Dectin-2 Agonist Antibody Program**

BDC-2034: Extending the Boltbody™ ISAC Platform to

Address Significant Unmet Needs in CEA-Expressing Solid Tumors

BDC-2034



CEA-targeting mAb conjugated to a proprietary TLR7/8 agonist via a non-cleavable linker

- Unique opportunity to target “cold” tumors that express CEA
- Slow internalizing tumor antigen results in longer residence time

Preclinical Proof of Concept Achieved

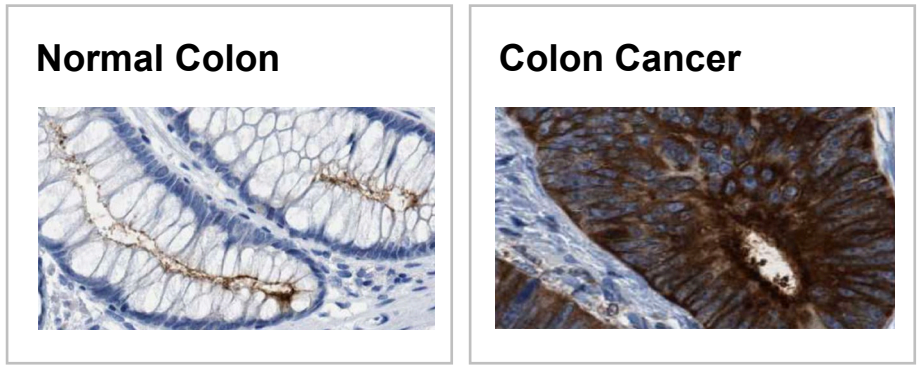
- Robust activation of human myeloid APCs
- Potent inducer of antibody-dependent cellular phagocytosis
- Anti-tumor activity in immunologically “cold” models of pancreatic cancer

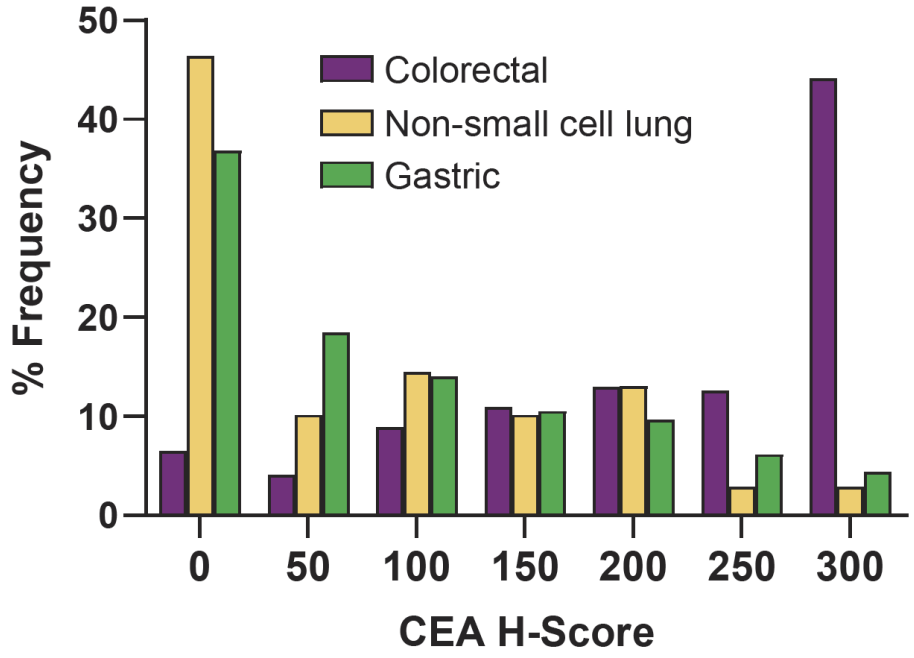
Status

- Entered IND-enabling studies
- Expecting Phase 1 initiation in 2022

BOLT BIOTHERAPEUTICS

CEA Profile Provides a Favorable Opportunity for ISAC Targeting





CEA (CEACAM5) is a cell-surface glycoprotein

- Slowly internalizing: 60% remains on cell surface after 5 hours

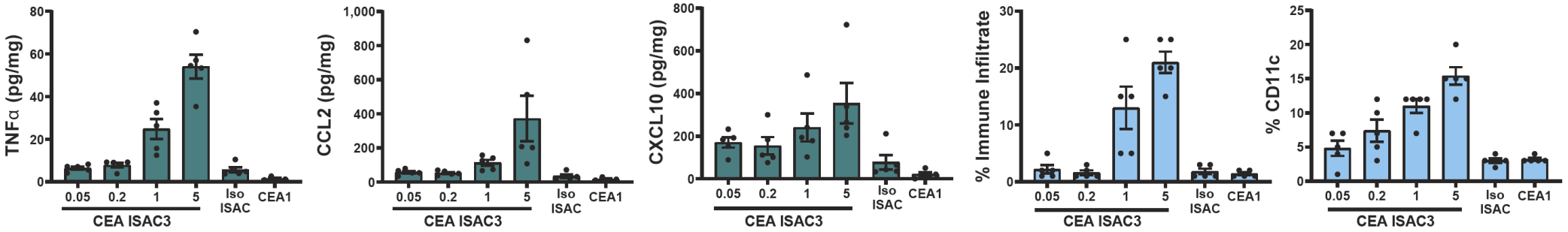
CEA is Highly Expressed in Select Cancers

- Colorectal Cancer: >90% CEA+
  - Universal myeloid immune cell infiltration
  - Low T-cell infiltrate except in MSI-H tumors
- Gastric/GEJ Cancer: >50% CEA+
- Non-small Cell Lung Cancer: >50% CEA+

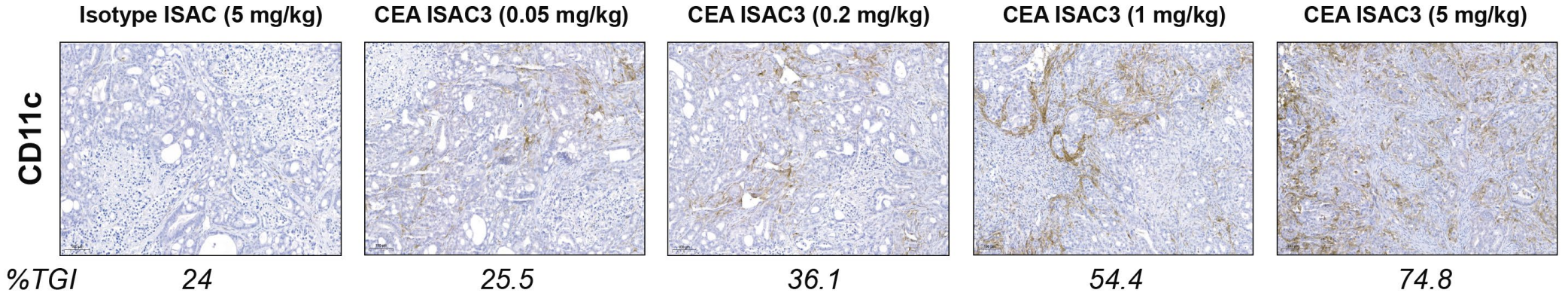
Representative CEA IHC images from Human Protein Atlas;
Mallet WG, et al., SITC Poster (2021)

CEA ISAC Promotes CEA-dependent Cytokine Secretion and Immune Cell Infiltrate in HPAF-II Xenograft Tumors

Cytokine Secretion and Immune Infiltrate Increases with Dose of CEA ISAC



Myeloid Cell Infiltrate Increases with Dose of CEA ISAC, Together with Tumor Growth Inhibition

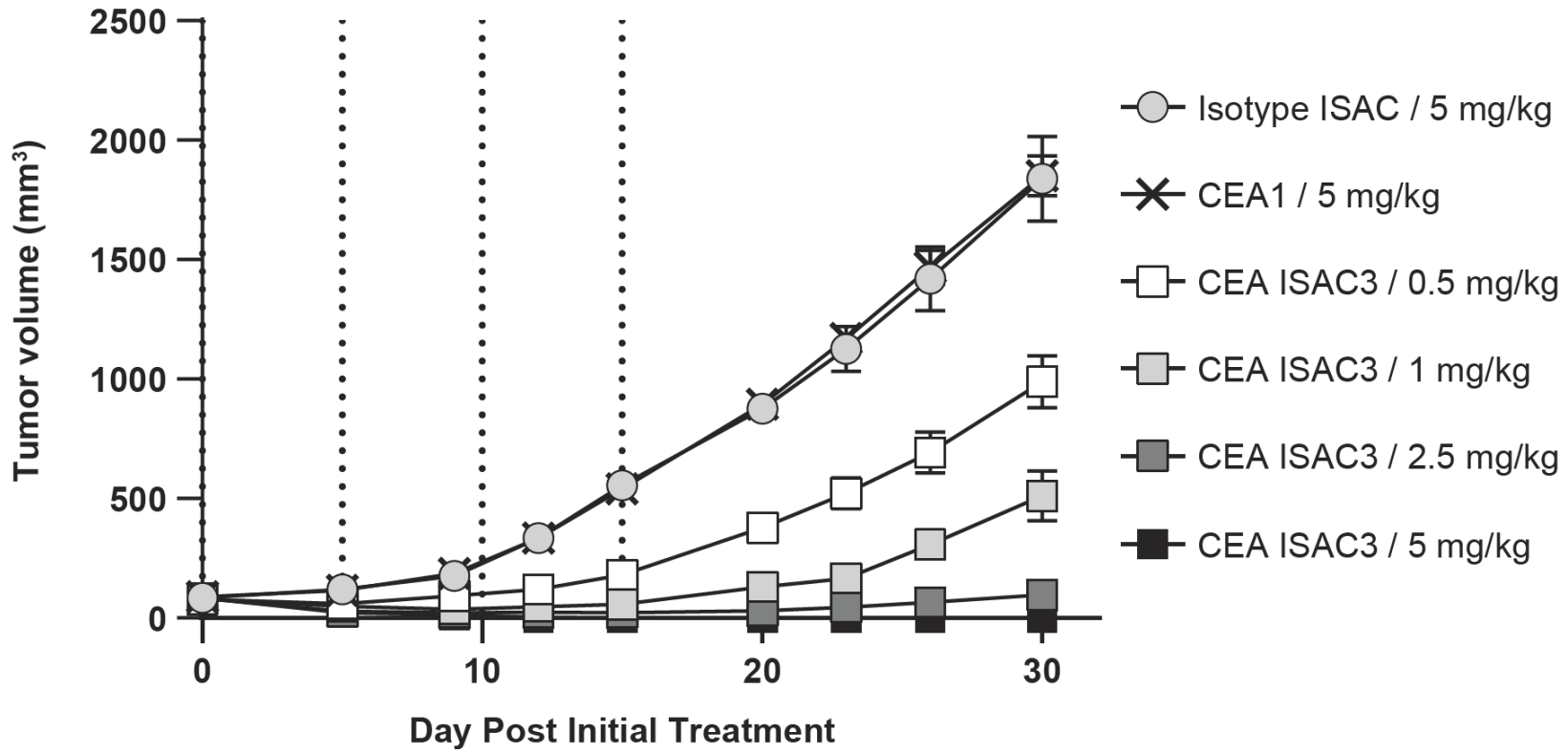


Mallet WG, et al., SITC Poster (2021)

BOLT BIOTHERAPEUTICS

CEA ISAC Demonstrates Robust Anti-tumor Activity in CEA-Expressing Pancreatic Cancer Model

CEA ISAC anti-tumor activity correlated with cytokine secretion and immune infiltration in a dose-dependent manner



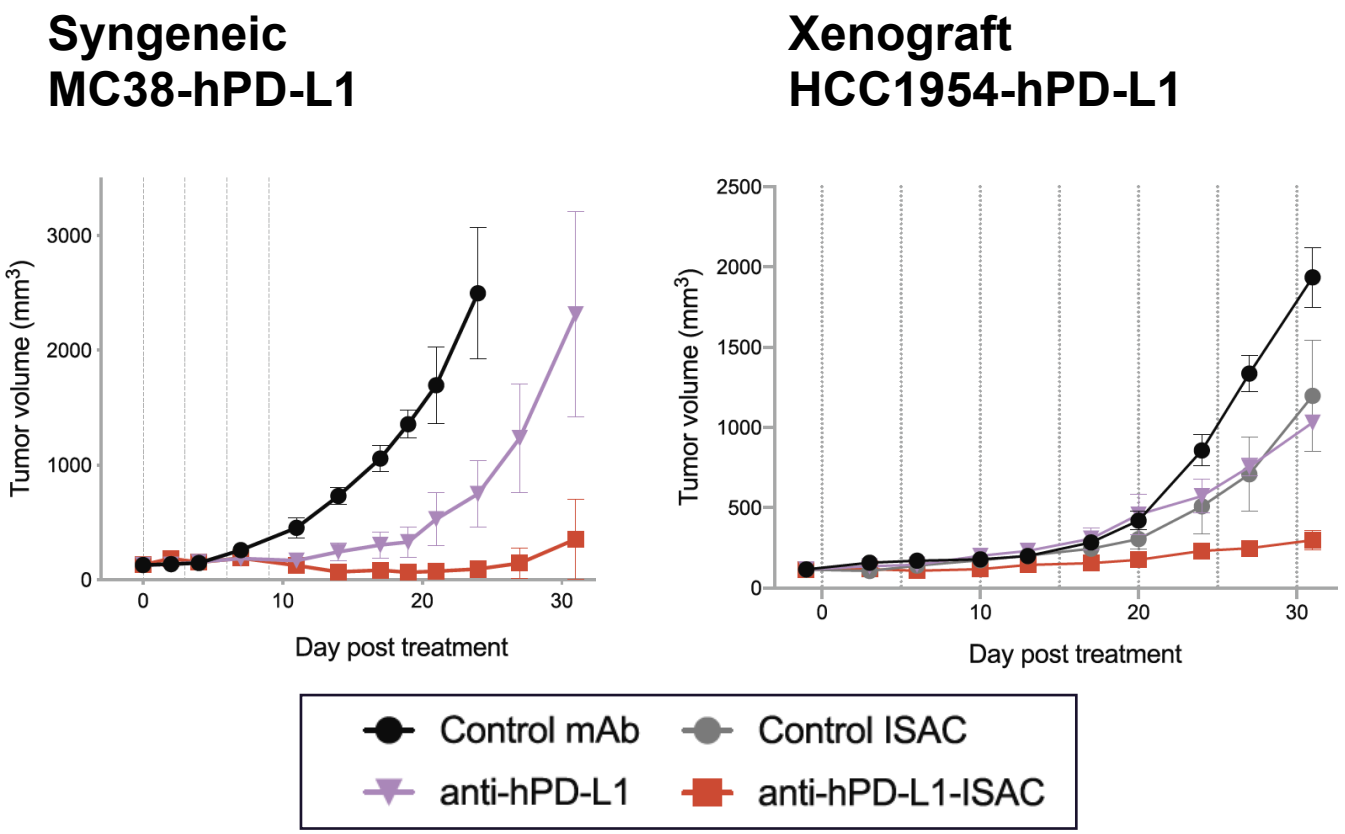
Mallet WG, et al., SITC Poster (2021)

SCID/beige mice were dosed systemically with 5 mg/kg every 5 days through day 15. Data are shown as mean ± SEM with 5 mice per group.

BOLT BIOTHERAPEUTICS

PD-L1 Boltbody ISAC Demonstrates Improved Anti-tumor Activity Relative to PD-L1 Antibody in Multiple Tumor Models

PD-L1 ISAC displayed in vivo anti-tumor activity in Xenograft and Syngeneic Tumor Models

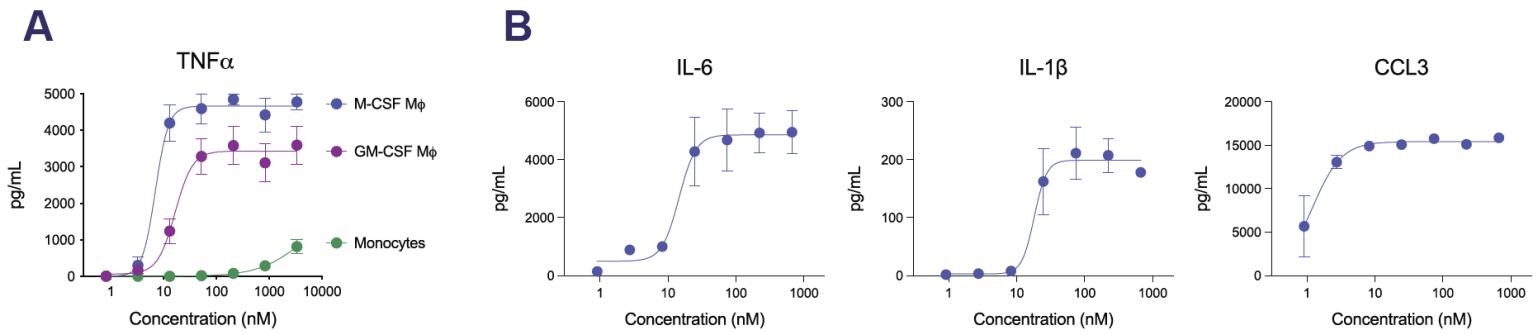


- PD-L1 Boltbody ISAC program focuses on tumors that are nonresponsive or become refractory to immune checkpoint blockade
- Adds PD-L1 checkpoint blockade to the usual ISAC mechanism
- PD-L1 is expressed by both solid tumor and myeloid cells
- We have identified PD-L1-targeting mAbs with the desired ADCP activity & ability to block the PD-L1/PD-1 axis

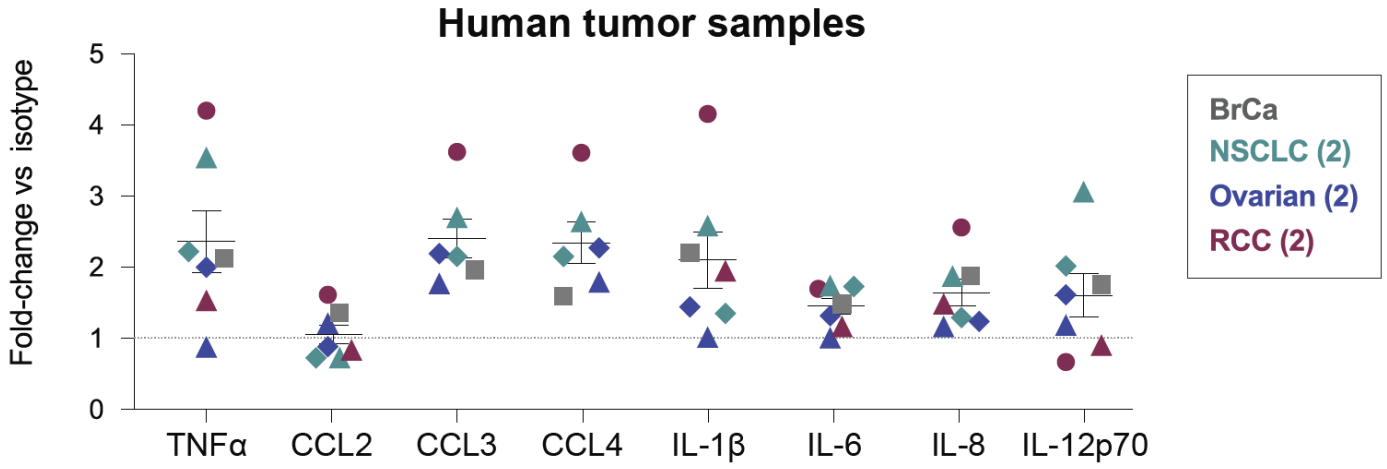
Kenkel JA, et al., SITC Poster (2021)
Note: C57BL/6J mice were dosed systemically with 5 mg/kg every 3 days through day 9. Data are shown as mean ± SEM with 4 mice per group.

Dectin-2 Myeloid Modulator Shows Potential for Anti-tumor Activity by Reprogramming Tumor-supportive Macrophages

Dectin-2 agonist mAb potently activates human macrophages



Dectin-2 agonist mAb activates primary human TAMs ex-vivo



- Dectin-2 is selectively expressed on tumor-supportive macrophages in a range of human cancers
- Dectin-2 agonism results in the production of pro-inflammatory cytokines more consistent with the characteristics of tumor-destructive myeloid cells
- Dectin-2 agonism can mediate tumor regression in syngenic models
- KRAS and TP53 mutations may upregulate Dectin-2 on tumor-associated myeloid cells

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

BOLT BIOTHERAPEUTICS

Summary

Experienced Team, Proven Track Record in Drug Discovery and Development

Randall Schatzman, Ph.D.
Chief Executive Officer

ALDER BIOPHARMACEUTICALS
CELLTECH
Roche

William Quinn
Chief Financial Officer

sunesis
Jazz Pharmaceuticals
BULLET BIOTECHNOLOGY, INC.

Edith A. Perez, M.D.
Chief Medical Officer

MAYO CLINIC
Genentech

Grant Yonehiro
Chief Business Officer

maxygen
astellas
PERSEID therapeutics

David Dornan, Ph.D.
Chief Scientific Officer

Genentech
GILEAD

Track Record of Successful Drug Development

ALECENSA
Cytovene®
EVENITY
GAZYVA
Herceptin
Kadcyla
PERJETA
POLIVY
Rituxan
TECENTRIQ
Valcyte
VENCLEXTA
vyepti

BOLT BIOTHERAPEUTICS

Pioneering a New Class of Immuno-oncology Products

First-in-class Boltbody™ ISAC: BDC-1001	Broadly Enabling Platform Technology	Cash on Hand Achieves Key Milestones
		

Upcoming Milestones

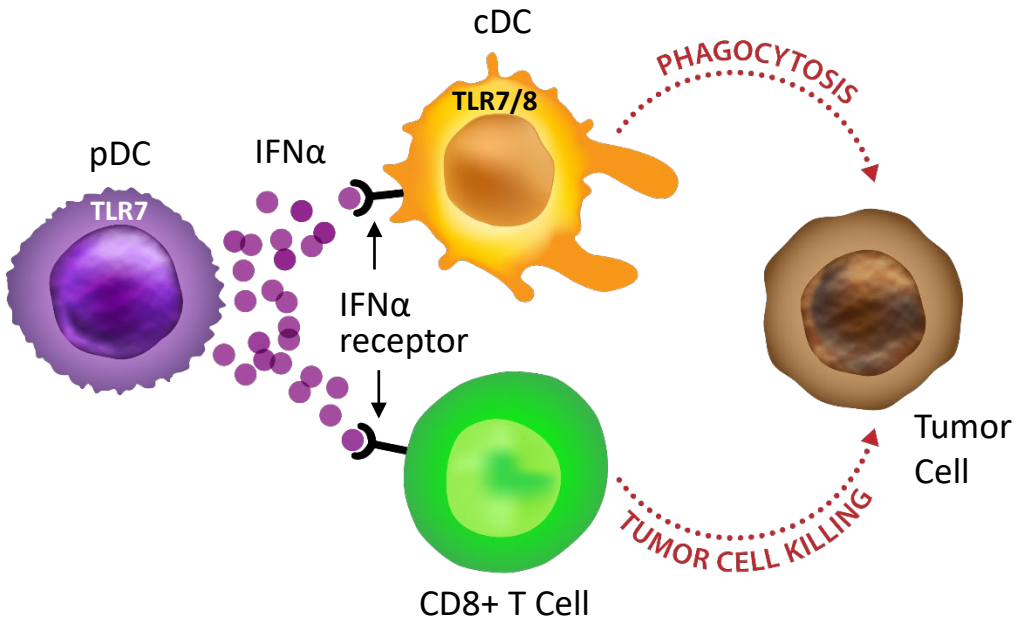
- 4Q21: Interim data on BDC-1001 monotherapy dose escalation
- 4Q21: Initiate BDC-1001 + anti-PD-1 combination dose escalation
- 2022: Initiate BDC-1001 Phase 2 monotherapy dose expansions
- 2022: Initiate BDC-2034 first-in-human clinical trial

BOLT BIOTHERAPEUTICS

Thank You

Dual TLR7 & TLR8 Agonism Optimizes Productive Anti-tumor Immune Response

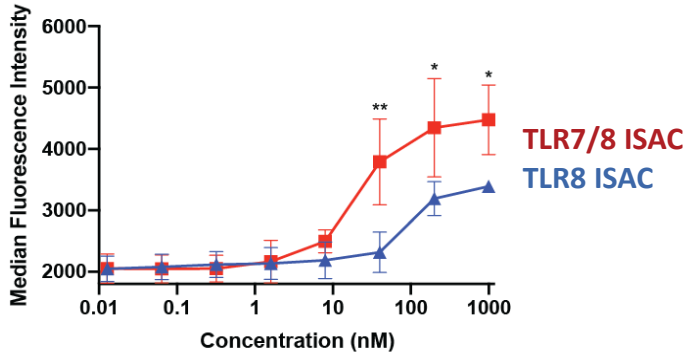
TLR7/8 dual agonist provides an amplification of the immune response



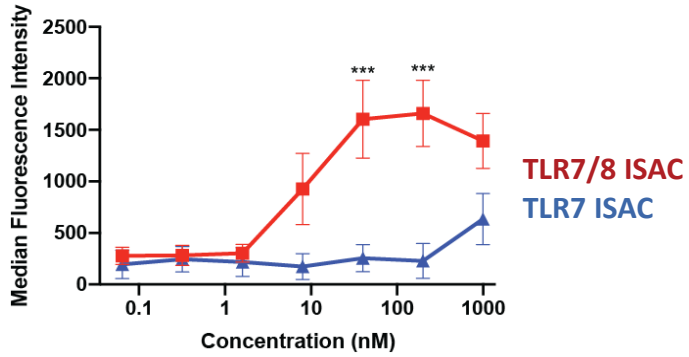
TLR7/8 dual agonist results in enhanced myeloid activation

Human myeloid APCs were co-cultured with CD20+ tumor cells and rituximab ISACs or rituximab for 18 hours.

Expt #1: **TLR7/8 ISAC vs. TLR8 ISAC (CD86)**



Expt #2: **TLR7/8 ISAC vs. TLR7 ISAC (CD86)**

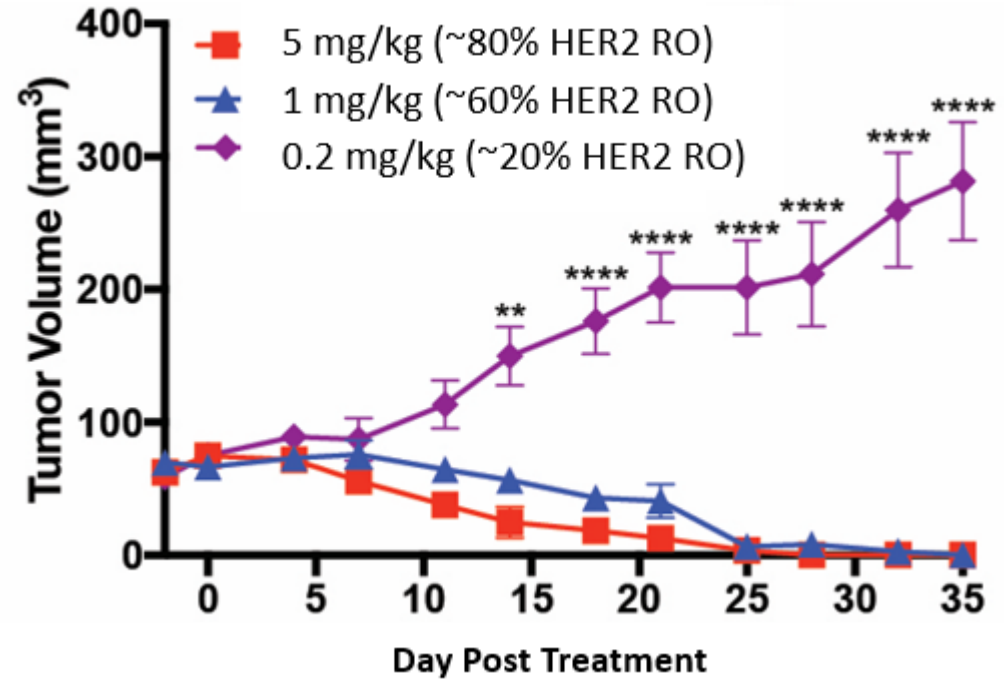


Receptor Occupancy Threshold Triggers ADCP, Eliminating Tumors

Further Support for ADCP-driven Mechanism in a Model Without T Cells

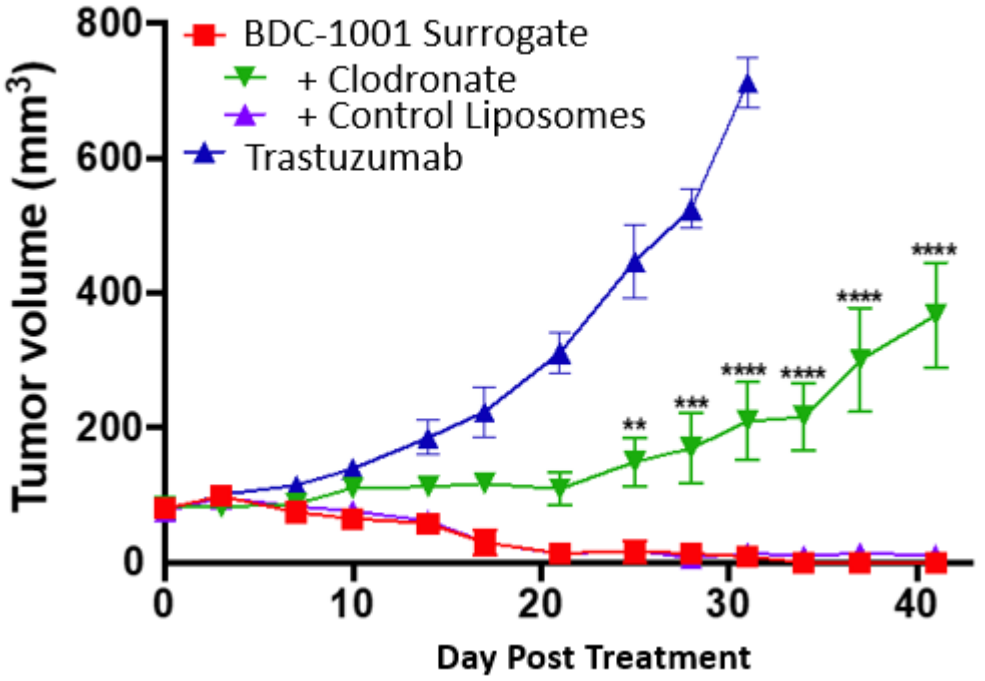
Activity dependent on ~60% receptor occupancy (RO)

Mice implanted with HER2-expressing HCC1954 cell line



Activity dependent on presence of phagocytes

Mice implanted with HER2-expressing HCC1954 cell line



AACR 2021

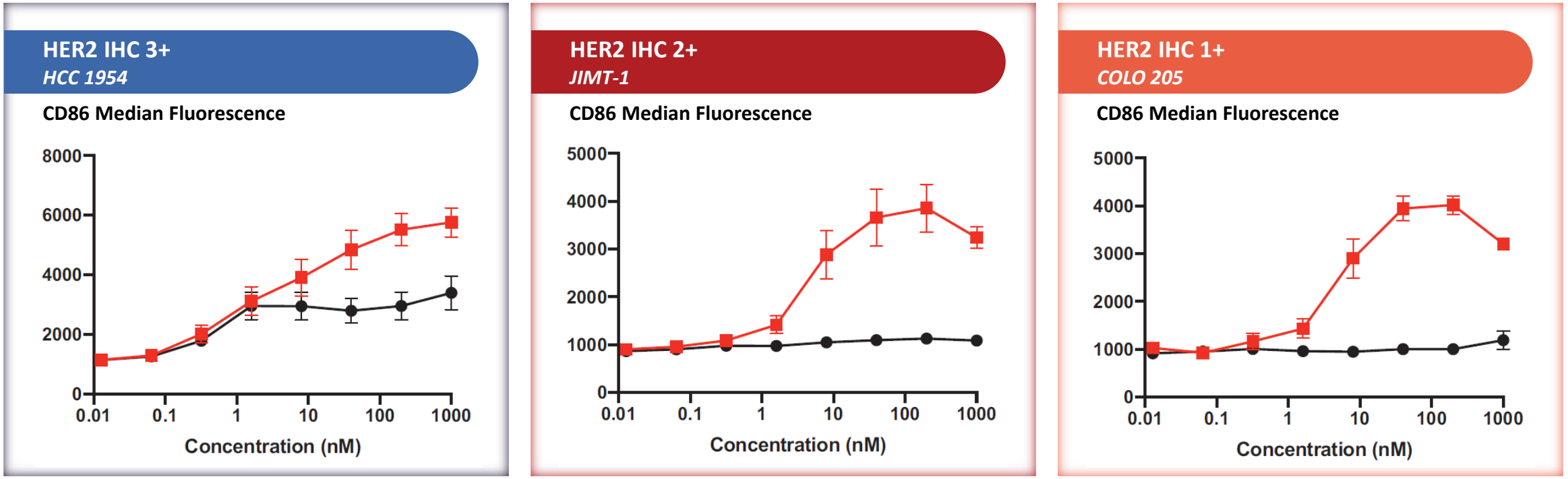
Note: NSG mice were dosed systemically with 5 mg/kg every 5 days through day 25 on left, and through day 10 on right. Data are shown as mean ± SEM with 5 mice per group.

BOLT BIOTHERAPEUTICS

Potential to Treat Tumors with Lower Levels of HER2 Expression

Opportunity to Address Larger Patient Population than Current HER2-Targeted Therapies

BDC-1001 activated human myeloid APCs to a similar extent when co-cultured with tumor cell lines expressing high (IHC3+) or lower levels of HER2 (IHC2+ or IHC1+)



Similar increased expressions of CD40 and TNFα secretion were also observed, each of which are indicative of a robust myeloid activation response

Note: Pooled myeloid APCs were incubated with the indicated cancer cell line and trastuzumab or BDC-1001. Median fluorescence intensity of CD86 is shown. Data are shown as mean ± SEM from 3 experiments with 18 donors.

BOLT BIOTHERAPEUTICS

Preclinical Tolerability Suggests Wide Therapeutic Window

Well Tolerated: No Findings

SPECIES CHOICE

- Single species toxicology program in cynomolgus monkeys
- BDC-1001 activates myeloid cells in both NHPs and humans
- Trastuzumab is cross-reactive between NHP and human HER2

SUMMARY

- 10, 30 or 90 mg/kg of BDC-1001 dosed weekly for a total of 4 administrations, n=7 per group
- No gross or histopathological findings
- No adverse effects at any dose level tested
- No treatment-related changes in systemic cytokines
- No interstitial lung disease
- No observed adverse effect level (NOAEL) for BDC-1001 was determined to be 90 mg/kg, the highest dose tested

BOLT BIOTHERAPEUTICS

BDC-1001 Safe and Well Tolerated in First 20 Patients

No DLTs, No Drug-related SAEs, All Patients Completed Safety Evaluation Period

	All TEAEs (N=20)		Treatment-related TEAEs (N=20)	
Preferred Term, n (%)	**All Grades***	**≥ Grade 3**	**All Grades**	**≥ Grade 3**
Fatigue	8 (40%)	1 (5%)	2 (10%)	0
Arthralgia	4 (20%)	0	2 (10%)	0
Infusion-related reaction	4 (20%)	0	4 (20%)	0
Pyrexia	4 (20%)	0	2 (10%)	0
Nausea	3 (15%)	1 (5%)	1 (5%)	0
Abdominal pain	3 (15%)	1 (5%)	1 (5%)	0
Dyspnea	3 (15%)	1 (5%)	0	0
Aspartate aminotransferase increased	3 (15%)	0	1 (5%)	0
Diarrhea	3 (15%)	0	3 (15%)	0
Urinary tract infection	3 (15%)	1 (5%)	0	0
Vomiting	3 (15%)	1 (5%)	0	0

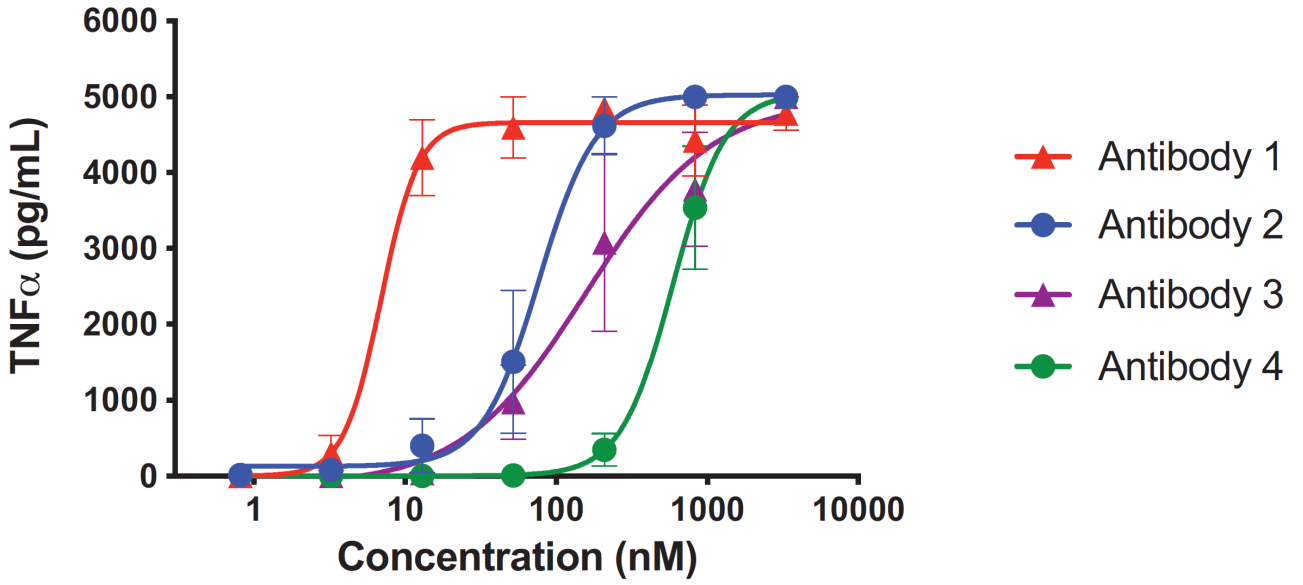
** Occurring >10% in all patients*

ASCO 2021; data as of January 29, 2021

BOLT BIOTHERAPEUTICS

Dectin-2 Myeloid Modulator Shows Potential to Reprogram Tumor-supportive Macrophages

Capacity of Dectin-2 binding mAbs to enhance TNFα secretion from tumor-supportive macrophages



- We have identified mAbs (Antibodies 1-4) capable of binding to and agonizing a novel cell surface protein, Dectin-2, on tumor-supportive macrophages
- Dectin-2 agonism results in the production of pro-inflammatory cytokines more consistent with the characteristics of tumor-destructive myeloid cells
- KRAS and TP53 mutations may upregulate Dectin-2 on tumor-associated myeloid cells
- Potential avenue to develop precision medicine with an immune modulator

Note: TNFα secretion by human M-CSF differentiated macrophages stimulated with Dectin-2 binding mAbs for 20 hours. Data are shown as mean ± SEM with 5 donors.

BOLT BIOTHERAPEUTICS