Collaboration Propels Innovation

May 2025

Forward-Looking Statements

Ichnos Glenmark Innovation ("IGI") is an alliance between Glenmark Pharmaceuticals Limited ("GPL") and IGI Inc. ("IGI Inc") for the purpose of collaborating with each other on the discovery and development of new molecules by leveraging on each other capabilities to achieve synergies around developing innovative pharmaceutical products. These materials have been prepared by IGI solely for informational purposes and are strictly confidential and may not be taken away, reproduced, or redistributed to any other person.

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OUR MISSION

"To provide curative therapies that extend and improve lives."

OUR VISION

"We dare to imagine a world where cure is possible."

Clinical-Stage Biotechnology Company at the Forefront of Innovation in Oncology

Fully Integrated Biotech

- Core capabilities in biologics
- Global footprint: U.S., Switzerland and India
- Shifting to outsourced biologics manufacturing

Biologics Discovery Engine

- Proprietary protein engineering platform (BEAT®)

Robust Pipeline

- Clinical stage pipeline in Oncology
- Engaging different types of immune cells
- 2 Alliances

BEAT®: Bispecific Engagement by Antibodies based on the TCR

Highly Experienced Leadership Team

LEADERSHIP TEAM

Cyril Konto, M.D.
President and Chief Executive Officer

Lida Pacaud, M.D.
Chief Medical Officer

Mario Perro, Ph.D.
Head of Biologics Research

Roberto Giovannini, Ph.D.
Chief Process & Manufacturing Officer

Dean Thomas, LLM
General Counsel

Sebastien Chenuet, Ph.D.
Head of Business Development

Karishma Sipahimalani, Ph.D.
Head of Human Resources

PREVIOUS EXPERIENCE

Pfizer
Bristol Myers Squibb
Allogene
Roche
LEGEND BIOTECH
NOVARTIS
sanofi
Debiopharm
Boehringer Ingelheim
Glenmark
A new way for a new world

BY THE NUMBERS

100+
Years combined experience in biotech and pharmaceuticals

30+
Products developed or launched

40+
Mergers, acquisitions, IPOs and other transactions

Accomplished Board of Directors With Track Record of Success

Glenn Saldanha
Chairman & Managing Director
Glenmark Pharmaceuticals Limited

Alind Sharma
Global CHRO of Glenmark
Glenmark Pharmaceuticals Limited

V S Mani
Global CFO
Glenmark Pharmaceuticals Limited

Lawrence Olanoff, M.D., Ph.D.
Former President and COO
Forest Laboratories

Dennis Purcell
Founder of Aisling Capital and Former Senior Managing Partner

Cyril Konto, M.D.
President and Chief Executive Officer
Ichnos Glenmark Innovation

Meet The Scientific Advisory Board

Adam Cohen, M.D.
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Wolf Hervé Fridman, M.D., Ph.D.
Professor Emeritus of Immunology, at Université Paris Cité Medical School, France

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Drug Discovery Scientist, Pharma Executive, Cancer Research Horizons

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Professor of Medicine, Deputy Division Head of Hematologic Malignancies at Memorial Sloan Kettering Cancer Center, USA

Philippe Moreau, M.D., Ph.D.
Professor of Clinical Hematology at the University Hospital of Nantes at the Medical University of Nantes in France

Lawrence Olanoff, M.D., Ph.D.
Adjunct Assistant Professor and Special Advisor to the President for Corporate Relations at the Medical University of South Carolina, USA

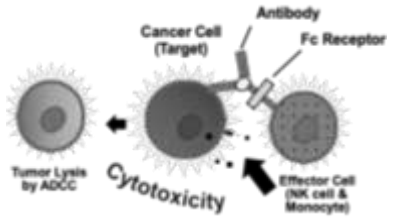
Eugene Zhukovsky, Ph.D.
Manager and Partner, ZM Scientific, Switzerland

IGI's Roadmap

CY24	CY25	CY26
✓ Formation of IGI		
✓ Clinical Proof-of-Concept for ISB 1442 and/or ISB 2001	ISB 2001 Licensing	IPO Capital Raise

Multispecific antibodies and Small Molecule (SM) Modulators are Complementary and Will Drive the Next Wave of Innovation in Oncology

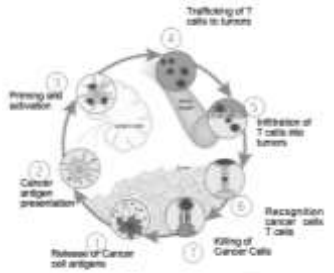
1990s



TARGETED THERAPIES

FC Function-based Tumor Killing

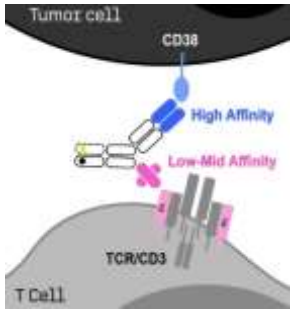
2010s



IMMUNO-ONCOLOGY

Checkpoint and Innate Immunity Modulators

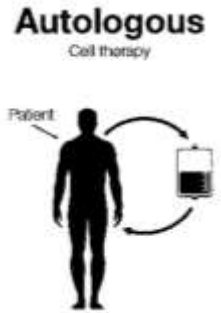
2014



BISPECIFIC ANTIBODIES

CD3 T Cell Engagers

2017



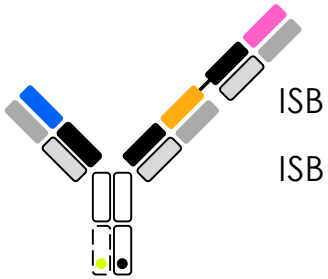
CAR-T CELLS

Engineered T Cells

Next Wave

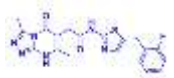
MULTISPECIFICS AND SMALL MOLECULE IMMUNOMODULATORS

Targeting simultaneously multiple cell surface antigens on cancer and immune cells while modulating their intracellular pathways.



ISB 1442

ISB 2001



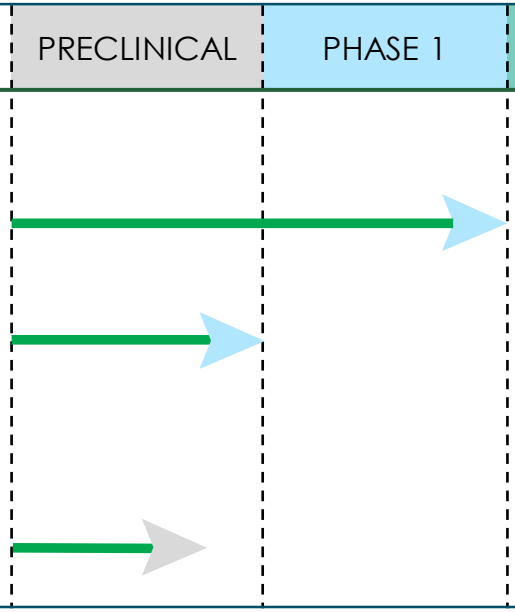
GRC 65327

Sudhakar A., J Cancer Sci Ther., 2009. DOI
Chen DS et al. Immunity. 2013 DOI, with permission from Elsevier

PIPELINE

Oncology-Focused Pipeline to Drive Long-Term Value Growth

ASSET	DESCRIPTION	INDICATION	STATUS
CLINICAL ASSETS			
ISB 2001	BCMA x CD38 x CD3 TREAT ™ trispecific **T-Cell *Engager***	Multiple Myeloma	PHASE 1 ORPHAN DRUG
GRC 65327	Cbl-b Inhibitor **Small Molecule**	Solid Tumors	PRE-CLINICAL
CANDIDATES			
ISB 2301	IMMUNITE ™ **NK-Cell Engager**	Solid Tumors	DISCOVERY



Strategic Partnerships Outside of Oncology to Maximize Pipeline Value

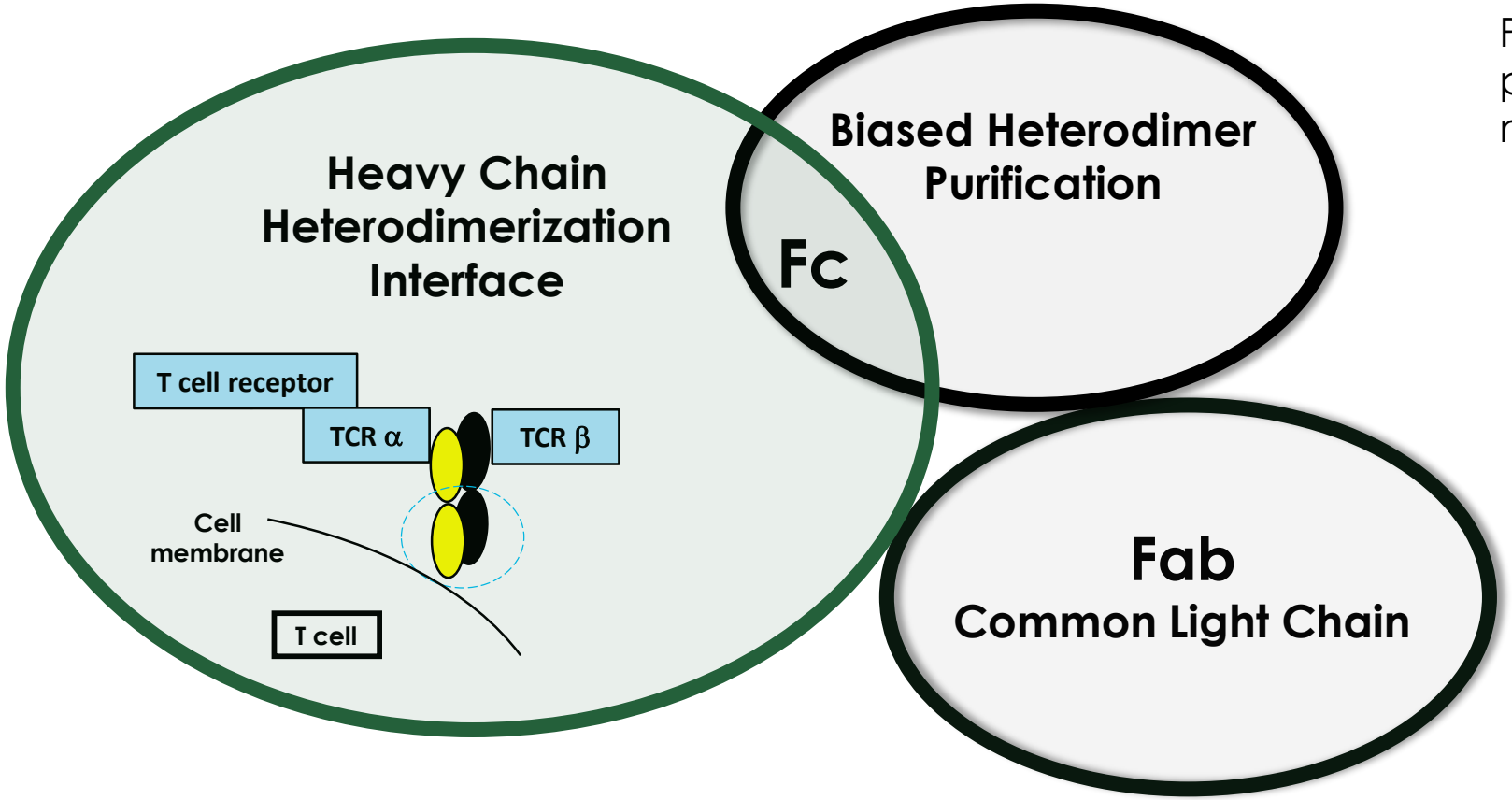
PRODUCTS	DESCRIPTION	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	STATUS
Licensed to	astria	$320 million for upfront payment, development, regulatory and sales milestone payments, plus tiered royalties on global sales				
Telazorlimab ISB 830-X8/STR-310	OX40 antagonist **Monoclonal Antibody**	Atopic Dermatitis →	→	→		SUCCESSFUL PHASE 2B PHASE 1a
Licensed to	almirall feel the science	€20.8 million for upfront payment. Plus development, regulatory and sales milestone payments, and tiered royalties on global sales				
ISB 880/ALM27134	IL-1RAP antagonist **Monoclonal Antibody**	Hidradenitis Suppurativa →	→			PHASE 1

Partnering-Ready Asset to Accelerate Short-Term Value Creation

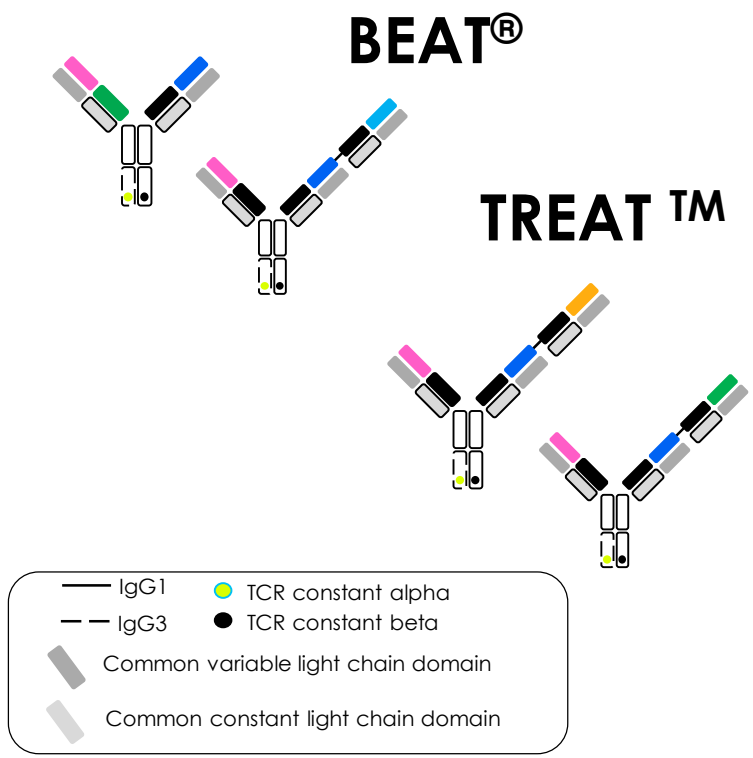
ASSET	DESCRIPTION	INDICATION	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	STATUS
CLINICAL ASSETS							
ISB 1442	CD38 biparatopic x CD47 BEAT® ***Myeloid-Cell Engager***	Multiple Myeloma		→			PHASE 1 ORPHAN DRUG

BEAT® Platform

BEAT® Combines TCR Interface-Based Heavy Chain Pairing and Universal Light Chain to Streamline Multispecific Antibodies Generation

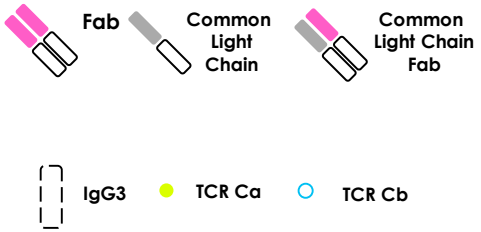


Proprietary plug-and-play modular platform enables a plurality of multispecific configurations

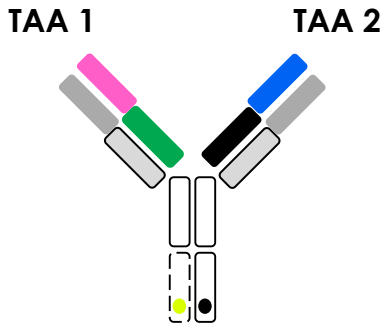


TREAT ™: TRispecific Engagement by Antibodies based on the TCR
TCR α/β: T-cell receptor α and β subunits; Fc: fragment crystallizable; Fab: fragment antigen-binding; Ig: immunoglobulin

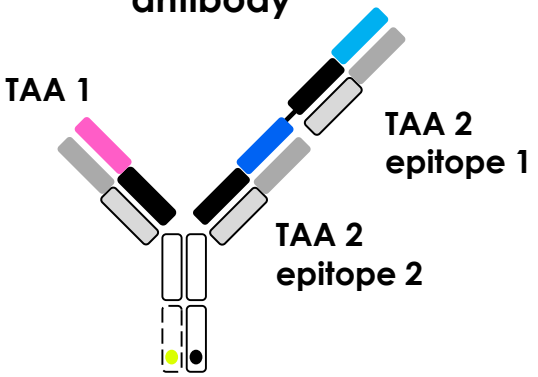
BEAT® is a Clinically Proven Platform Enabling the Design and Production of Immune Cell Engagers with High Developability Properties



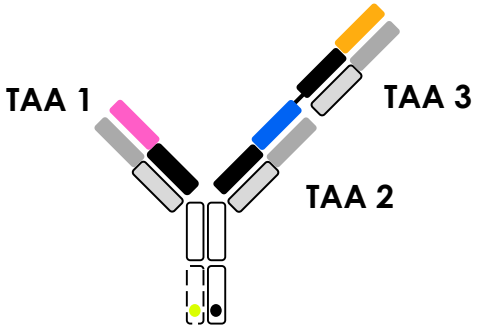
BEAT® (1+1) bispecific antibody



BEAT® (2+1) bispecific biparatopic antibody



TREAT ™ trispecific antibody



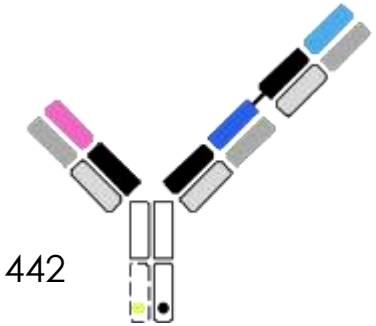
HPLC-Size Exclusion (% monomer)	98.2	98.4	98.0
LC-Mass Spectrometry (% purity)	99.9	99.0	100
Titer CHO (g/L)	2.5	11*	10*
Solubility without formulation (PBS, mg/ml)	≥ 50	≥ 50	≥ 50

* High cell density seeding (process intensification) at proof-of-concept stage, demonstrated in 3L bioreactors

Expression using optimized vector system led to the detection of 94% (LC-Mass Spectrometry) heterodimer in the cell culture supernatant prior to purification

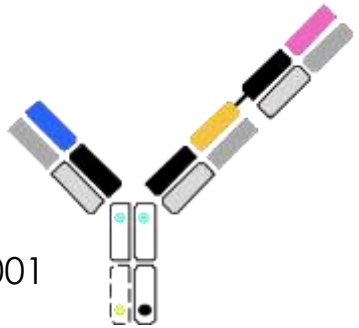
Multispecific Antibodies Using Clinically Proven BEAT® platform are Tailored to Specific Biological Functions

BEAT® (2 + 1)



ISB 1442

TREAT ™ TRISPECIFIC



ISB 2001

Enables design and development of bi/multispecific antibodies that unlock new biology (e.g., T cell, NK cells, macrophage engagers) by optimizing:

- Affinity: low-medium-high combinations
- Epitope: target/test several epitopes
- Architecture: avidity, immune synapse size
- Fc function: T cell: silent; non T cell: active – enhanced
- Improved druggability and developability – rapid engineering

Platform welcome partnerships to:

- Establish collaboration leveraging our BEAT technology, discovery and development capabilities
- Create new opportunities in therapeutic areas within oncology, autoimmune diseases and beyond
- Collaborate through discovery and license agreements, co-development or company creation.

MHC: Major histocompatibility complex, CDC: Complement-Dependent Cytotoxicity
ADCC: Antibody-Dependent Cell-mediated Cytotoxicity

ISB 2001

ISB 2001 – Executive Summary

- First-in-class trispecific BCMAxCD38xCD3 antibody, developed in relapsed/refractory multiple myeloma.
- Phase 1 first-in-human study of ISB 2001 for the treatment of relapsed/refractory multiple myeloma is currently ongoing in the US, Australia and India (Clinicaltrials.gov identifier: NCT05862012).
- Preliminary results from the phase 1 dose escalation (ongoing) showed:
  - ISB 2001 is well tolerated with no dose limiting toxicities up to 1200 µg/kg, low grade cytokine release syndrome, no neurological Adverse Events or ICANs, low infection and hematological toxicity rates, no Adverse Events leading to discontinuation.
  - Early, deep and sustained responses were observed across effective dose levels (DL3 to DL7) with antimyeloma activity from 50 µg/kg (MRD negative sCR) and higher.
  - Overall Response rate (ORR) was 83% (22% Complete response (CR) or better, 50% Very Good Partial Response (VGPR) and 11% Partial Response (PR). The ORR was 75 % in patients pretreated with CAR-T or bispecific T cell engagers and 90 % in patients who had not been treated with T-cell directed therapies.
- Pre-clinical data[1] showed potential for ISB 2001 to induce enhanced cytotoxicity relative to teclistamab against MM expressing variable levels of BCMA and CD38, mimicking natural tumor heterogeneity.
- Granted orphan drug designation (ODD) by the U.S. Food and Drug Administration (FDA).
- ASH 2024 Oral Presentation: First results of a Phase 1, First-in-Human, Dose Escalation Study of ISB 2001, a BCMAxCD38xCD3 Targeting Trispecific Antibody in Patients with Relapsed/Refractory Multiple Myeloma (RRMM)

[1]*Carretero-Iglesia L. et al. Nature Cancer 2024 DOI*
[2]*Abstract #1026, Monday, December 9, 2024, at 5:45 PM*

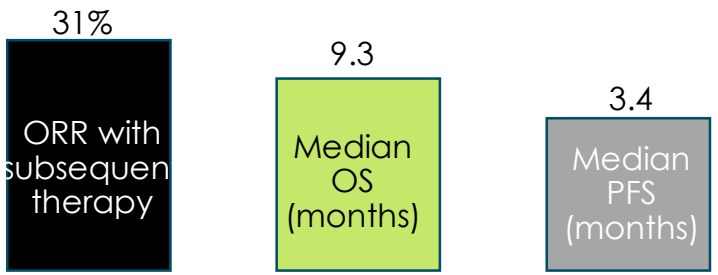
ISB 2001 Clinical Positioning Addresses Unmet Needs in Multiple Myeloma, Overcomes Limitations of Select Therapies

HIGH UNMET NEED AND LARGE MARKET

Global multiple myeloma cases annually[1]

160000

Low Responses for Triple Refractory Patients[2]



LIMITATIONS OF SELECT THERAPIES

- Decreased CD38 expression limits efficacy of CD38- targeted therapies[3]
- Resistance to Complement Dependent Cytotoxicity
- Few options following failure of BCMA-targeted therapies

[1] *Ludwig H. et al, Oncologist 2020 DOI* [2] *Gandhi UH et al. Leukemia 2019 DOI*

[3] *Saltarella I. et al., Cells 2020 DOI*

ISB 2001: A Testament to IGI R&D Excellence
Recognized by International Peer-Reviewed Journals and Conferences

2022	March Clinical Candidate Selection	2023	November First-in-Human	2024	June Proof-of Concept

Oral Presentation (pre-clinical)[1]

Oral Presentation

Oral Presentation (pre-clinical)[2]

Manuscript[3]

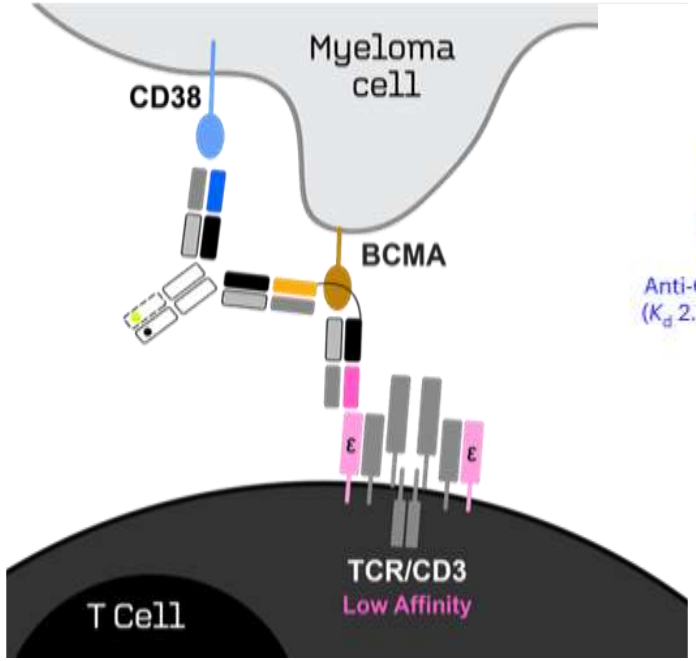
Oral Presentation (clinical)

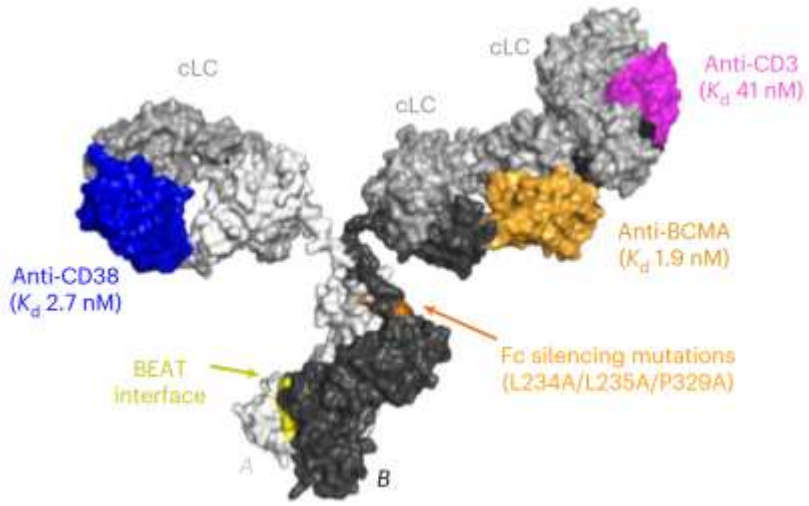
Commentary by Paul Parren[4]
"Antibody avidity meets multiple myeloma"

[1] Pihlgren M. et al. Blood (2022) DOI; [2] Carretero L. et al Cancer Research (2024) DOI
[3] Carretero-Iglesia L. et al. Nature Cancer (2024) DOI [4] Ruuls S. et al. Nature Cancer (2024) DOI

ISB 2001 (BCMAxCD38xCD3): First TREAT ™ Trispecific Antibody for Relapsed/Refractory Multiple Myeloma

ISB 2001 (BCMAxCD38x CD3) trispecific antibody	TREAT ™

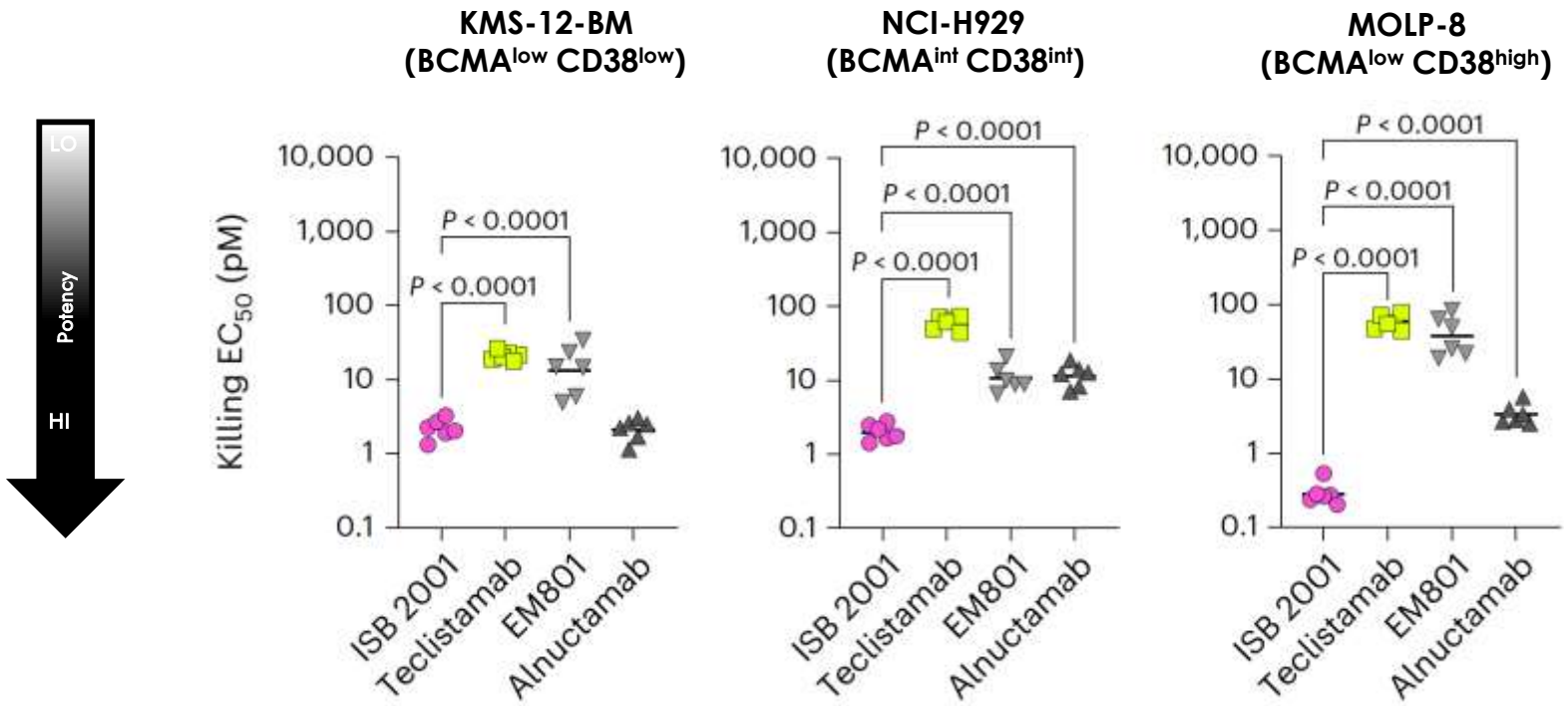




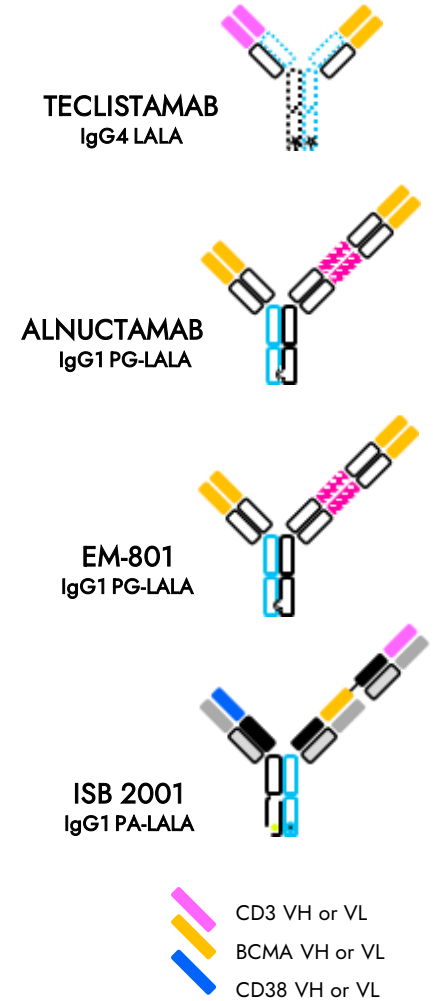
Key Attributes

- Three proprietary fragment antigen-binding arms: CD3ε on T cells; BCMA and CD38 on Multiple Myeloma cells
- Heterodimerisation based on the BEAT platform in a TREAT™ format
- Fab domains derived from synthetic phage display library with common light chain (Vκ3-15 + IgκJ1)
- Increased binding specificity to Multiple Myeloma cells due to enhanced avidity-based binding of anti-BCMA and anti-CD38 Fab domains
- FDA/HREC clearance and a first-in-human study started in November 2023
- Granted Orphan Drug Designation by FDA

ISB 2001 Designed to Mediate Potent MM Cell Killing via Dual Targeting Avidity-Driven Tumor Binding



Expression sABC	CD38 expression (sABC)	BCMA expression (sABC)	Clinical case modelling
KMS-12-BM	**LOW** 28000	**LOW** 9000	Post treatment with daratumumab + teclistamab
NCI-H929	**MID** 85000	**MID** 52000	Newly Diagnosed or post IMIDs + PI
MOLP-8	**HIGH** 512000	**VERY LOW** 3200	Post BCMA targeted therapy

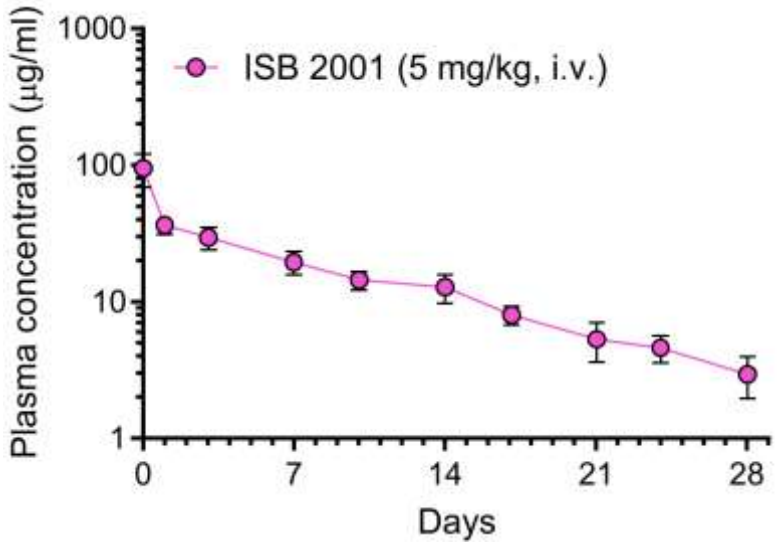


NQ: Not Quantifiable DU: Dummy (irrelevant binder)

Carretero-Iglesia L. et al. Nature Cancer (2024)

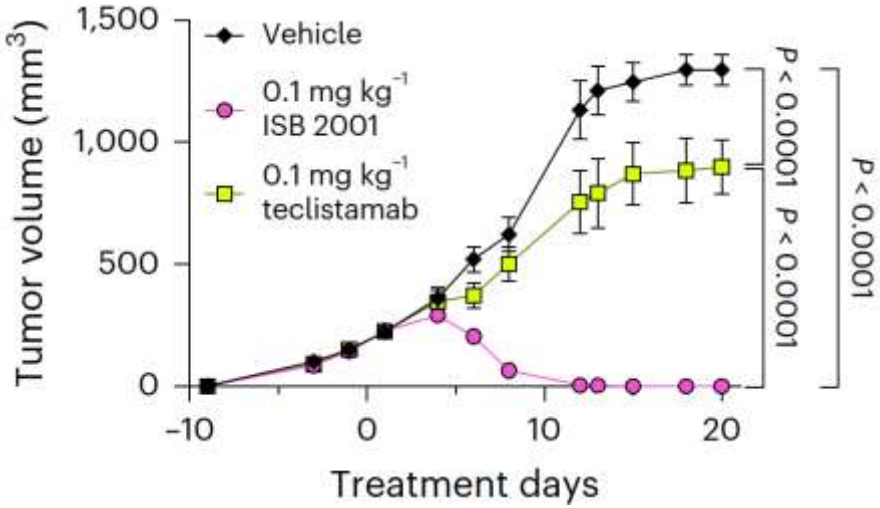
ISB 2001 Exhibits Desirable PK and shows 100% Complete Responses In Vivo in a BCMA^{low} CD38^{low} Multiple Myeloma Model

ISB 2001 Half-Life in Tg32 (huFcRn Tg) Mice



Molecule	Half-Life (days)	Cmax (µg/ml)	AUC (µg.days/ml)
ISB 2001	**7.6** ± 0.9	**95** ± 26	**417** ± 75

Efficacy in NSG-PBMC transfer Mouse Model (KMS-12-BM)

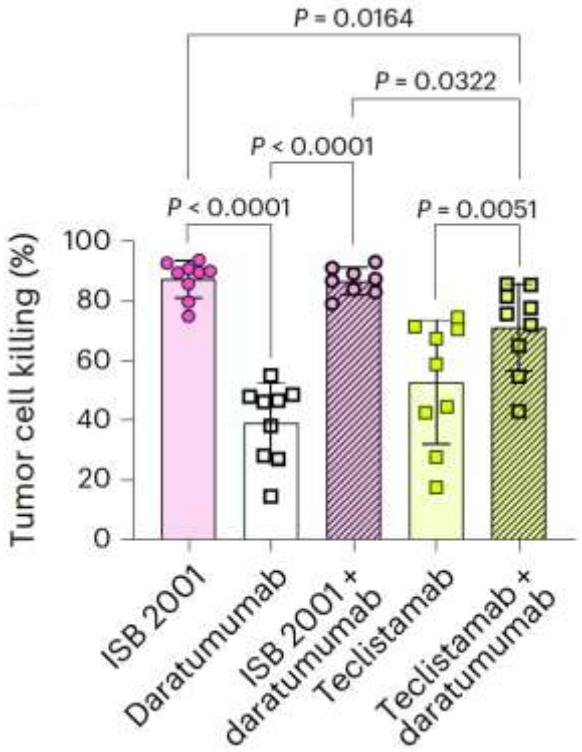


Treatment	Complete Response
Teclistamab	**0%** (0/8 mice)
ISB 2001	**100%** (8/8 mice)

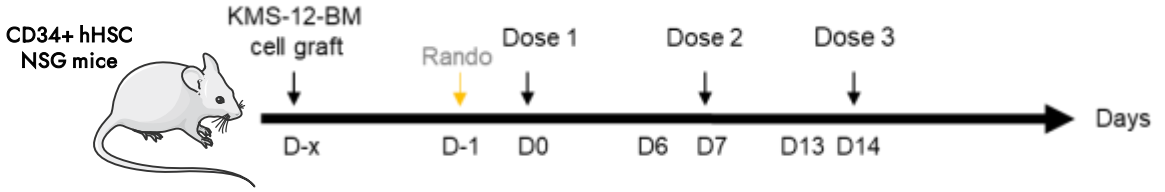
Carretero-Iglesia L. et al. Nature Cancer (2024) DOI

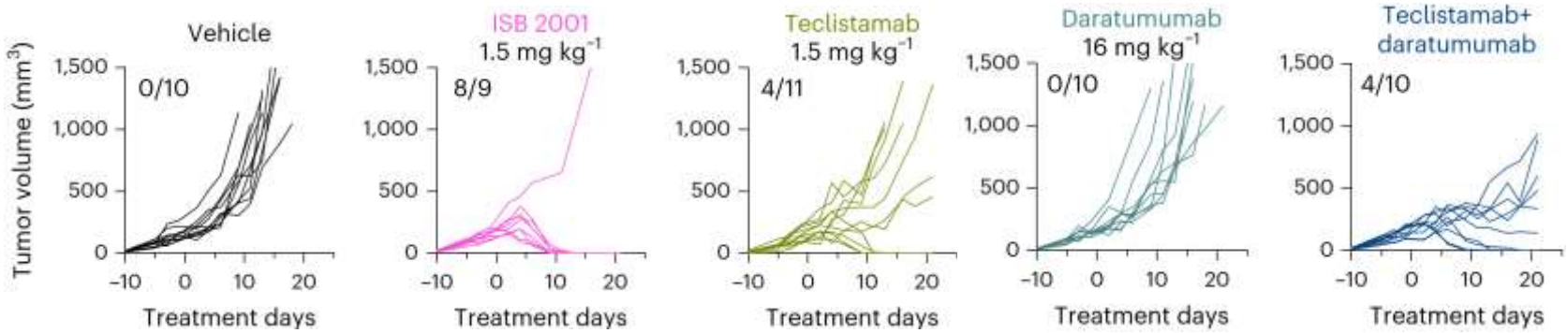
ISB 2001 Enhances Anti-Tumour Activity In Vitro and In Vivo Compared to BCMA and CD38 targeted therapies alone or in Combination

ISB 2001 is significantly more potent than Teclistamab + Daratumumab combination



Paired one-way ANOVA followed by Tukey's multiple comparisons test



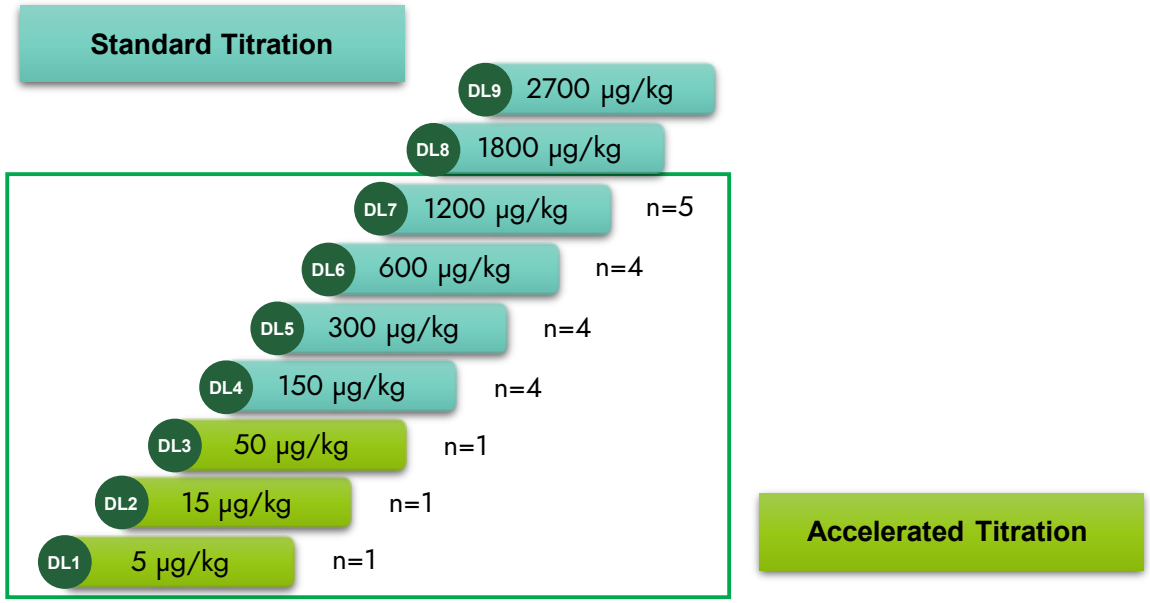


Treatment	Complete response	% of cured mice	2-way ANOVA vs ISB 2001
Vehicle	0/10	0 %	****
ISB2001	8/9	89 %	N.A.
Teclistamab	3/11	27 %	****
Daratumumab	0/9	0 %	****
Teclistamab + Daratumumab	3/10	30 %	****

Carretero-Iglesia L. et al. Nature Cancer (2024) DOI

ISB 2001-101: Phase 1 Dose-Escalation/Expansion Study

On-going Part 1 : Dose Escalation (n ≈ 40)



In dose escalation, ISB 2001 is administered subcutaneously (SC) once weekly (q1w) in 28-day cycles, starting with 2 step-up doses on Days 1 and 4, followed by the full target dose from Day 8 onwards.
Backfill to each DL allowed.

Key Eligibility Criteria:

- R/R MM, after a CD38 antibody, IMiDs, PIs, not candidates for regimens known to provide benefit
- Failed 3 or more prior lines of therapies
- Prior CAR-Ts and/or bispecifics allowed, prior BCMA-targeted agents allowed

Primary Objectives:

- Assess safety and tolerability
- Determine MTD/RP2D

Secondary Objectives:

- PK, immunogenicity
- Preliminary clinical activity by IMWG

Status:

- As of 1-Oct-2024, 20 subjects dosed in Australia and US in DL1 to DL7
- Dose-expansion part 2 will test at least 2 putative Phase 2 doses and dosing schedule to establish Recommended Phase 2 Dose

BCMA, B-cell maturation antigen; CAR, Chimeric antigen receptor; MTD, Maximum tolerated dose; RP2D, recommended phase 2 dose; IMWG, International Myeloma Working Group; PK, pharmacokinetics; DL dose level. ClinicalTrials.gov identifier NCT05862012
Oral Presentation, ASH Dec 2024

ISB 2001-101: Demographics and Disease Characteristics

Characteristic	Total (N=20)
Gender	
Female, n (%)	8 (40)
Median Age, range (years)	66 (52; 80)
Race, n (%)	
Black or African American	1 (5)
White	16 (84)
Other	2 (11)
Ethnicity, n (%)	
Not Hispanic or Latino	19 (95)
ECOG performance status, n (%)	
0	15 (75)
1	5 (25)
Lytic Bone Disease, n (%)	15 (75)
Extramedullary Disease, n (%)	6 (30)
Revised ISS, n (%)	
I	11 (55)
II	5 (25)
III	1 (5)
Cytogenetics available, n (%)	12 (60)
High risk cytogenetics	5 (42)
Bone Marrow Myeloma/Plasma cells ≥ 30%, n (%)	5 (25)

Characteristic	Total (N=20)
Median number of lines of previous therapy (range)	6 (3; 11)
Previous therapy exposure, n (%)	
Triple-exposed	20 (100)
Triple-refractory	5 (25)
Penta-exposed	14 (70)
Penta-refractory	2 (10)
Refractory to last line of therapy	13 (65)
ASCT	19 (95)
Anti-BCMA CAR-T	2 (10)
Bispecifics	9 (45)
BCMA	1 (5)
FcRH5	6 (30)
GPRC5D	4 (20)
Anti-BCMA ADC	5 (25)

High risk cytogenetics defined as presence of del(17), del(1p), t(14:16), t(14:20), t(4:14) or 1q amp. ECOG, Eastern Cooperative Oncology Group; ISS, International Staging System; ASCT, Autologous stem cell transplantation; FcRH5, Fc receptor-like protein 5; GPRC5D, G-protein coupled receptor family C group 5 member D; ADC, antibody-drug conjugate. Oral Presentation, ASH Dec 2024

ISB 2001: Clinically Manageable Safety Profile

Summary of TEAEs (N=20)	
n (%)	**Total**
Any AE Drug-related	20 (100) 20 (100)
Serious AE Drug-related	14 (70) 7 (35)
Grade 3-4 AE Drug-related	18 (90) 12 (60)
Grade 5 AE	0
AE leading to treatment discontinuation	0
Dose Limiting Toxicity	0

n, Number of patients ; TEAE, Treatment Emergent Adverse Events

Oral Presentation, ASH Dec 2024

ISB 2001: Low Rate of Hematologic and Infection Drug-Related TEAEs

Drug-Related Hematologic TEAEs (N=20)			
AEs, n (%)	All	Grade 3	Grade 4
Any Related Hematologic TEAEs	12 (60)	6 (30)	3 (15)
Anaemia	1 (5)	1 (5)	0
Lymphocyte count decreased	2 (10)	1 (5)	0
Neutropenia	7 (35)	3 (15)	3 (15)
Thrombocytopenia	8 (40)	2 (10)	0

Drug-Related Infections (N=20)			
AEs, n (%)	All	Grade 3	Grade 4
Any Related Infections	9 (45)	3 (15)	0
Lower respiratory tract infection	3 (15)	2 (10)	0
COVID-19	2 (10)	0	0
Upper respiratory tract infection	2 (10)	0	0
Cytomegalovirus viraemia	1 (5)	0	0
Pneumonia	1 (5)	1 (5)	0
Sinusitis	1 (5)	0	0

ISB 2001: Low Rates of Severe TCE-Related Adverse Events

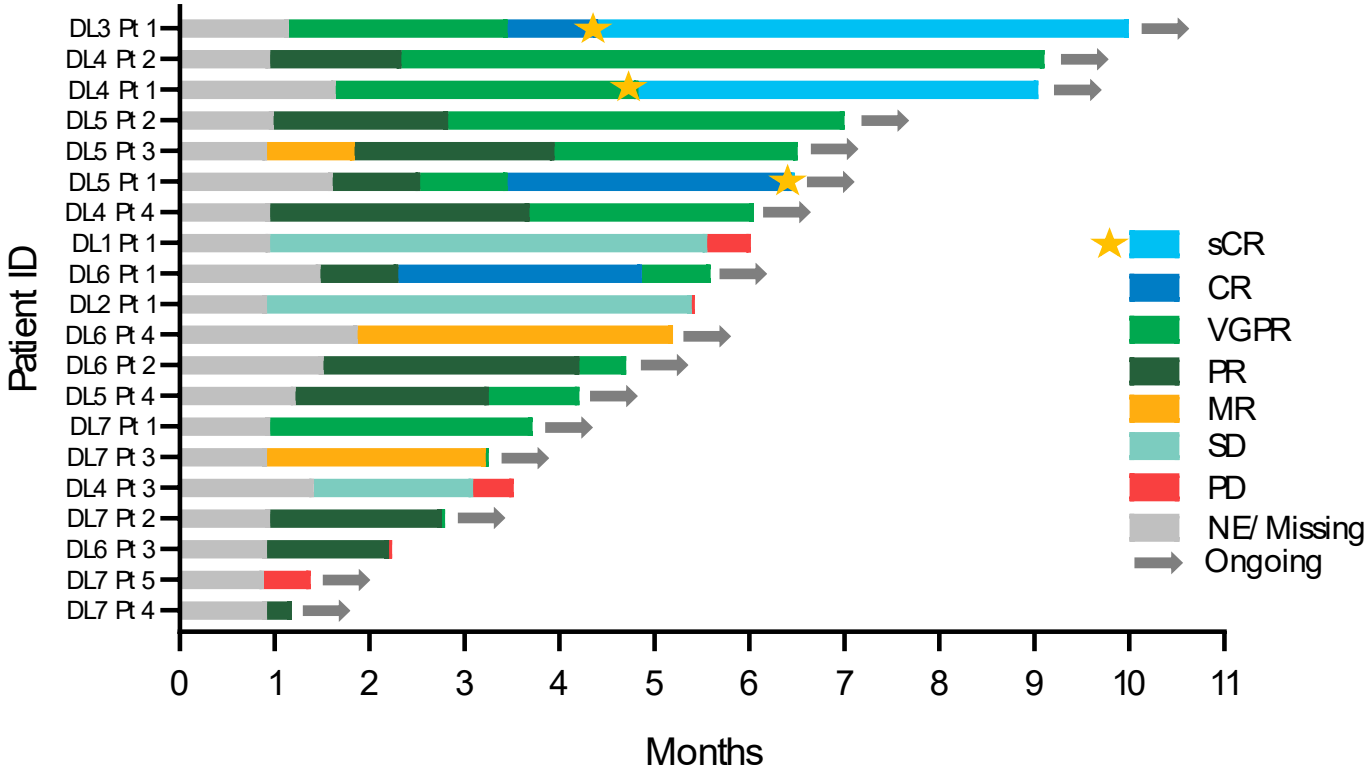
Non-Hematologic Drug-Related TEAEs (≥ 15%, N=20)			
AEs, n (%)	All	Grade 3	Grade 4
Any Related Non-Hematologic TEAEs	20 (100)	3 (15)	0
Cytokine release syndrome	15 (75)	0	0
Injection site reaction	12 (60)	0	0
Alanine aminotransferase increased	5 (25)	0	0
Aspartate aminotransferase increased	4 (20)	1 (5)	0
Fatigue	3 (15)	0	0
Gamma-glutamyltransferase increased	3 (15)	0	0
Nausea	3 (15)	0	0

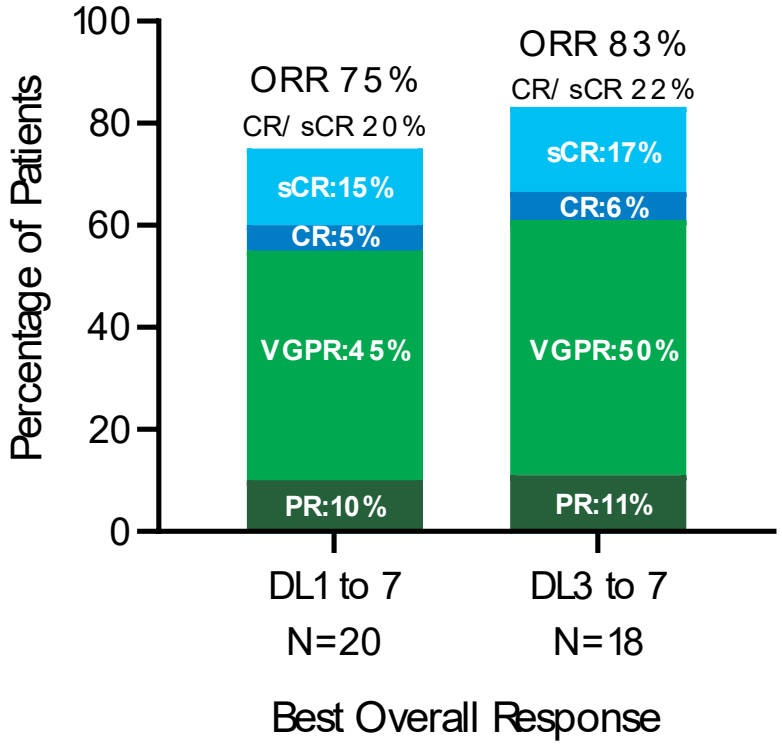
- No ICANS
- CRS events mostly G1 limited to first administration of ISB 2001
- All CRS reported were G1 except 2 cases reported as G2, one on Day 1 and second case on Day 118 (confounded by COVID-19)
- Median time to CRS: 3 (1;118) days
- Median Duration of CRS: 2 (1;8) days
- 2 subjects received Dexamethasone, and 7 subjects received Tocilizumab

ICANS, Immune effector cell-associated neurotoxicity syndrome; CRS, Cytokine Release Syndrome

Oral Presentation, ASH Dec 2024

ISB 2001: Deep and Lasting Responses Observed at ≥ 50 µg/kg



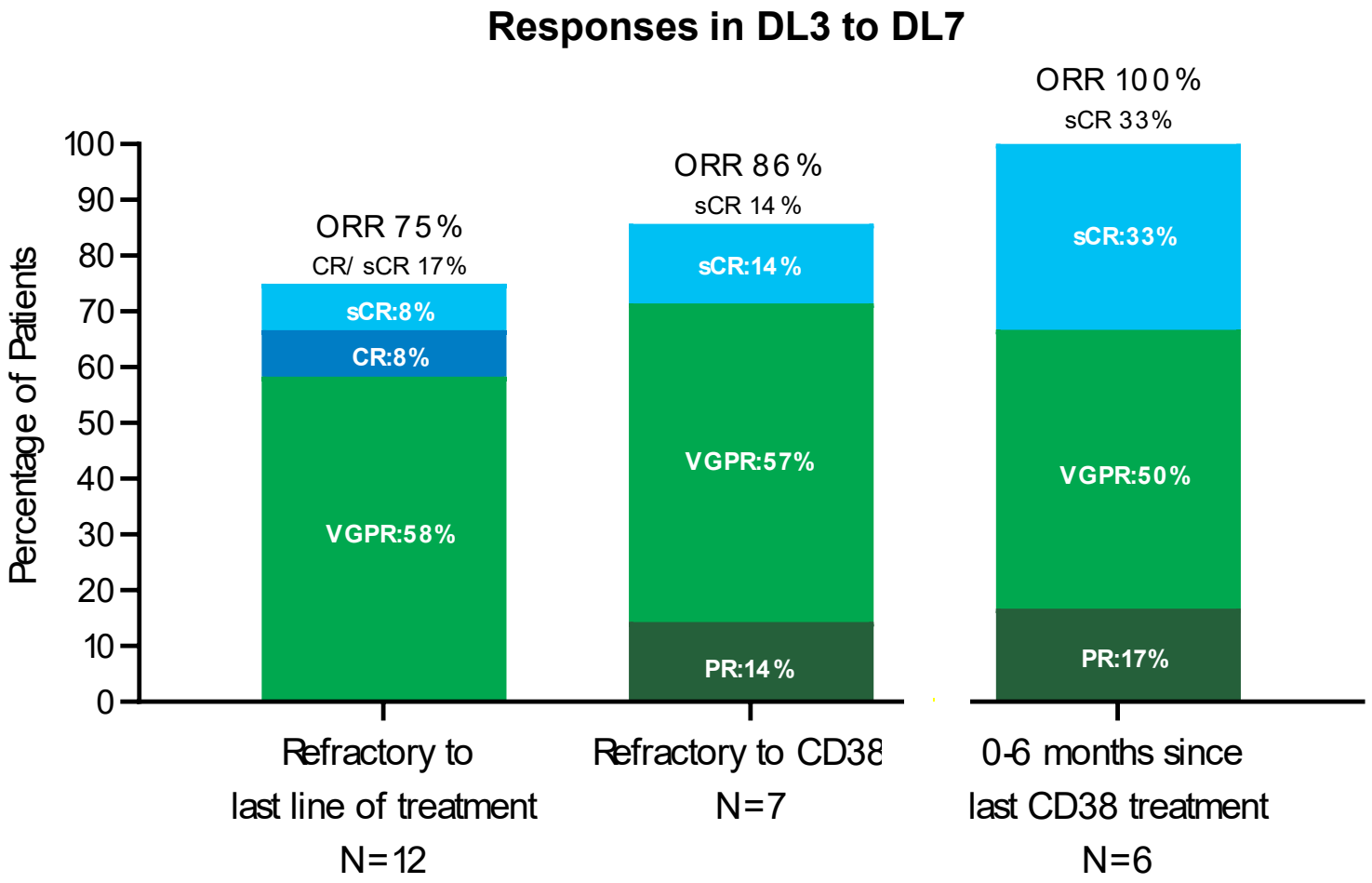


Median follow up 6 months (range: 2-10)

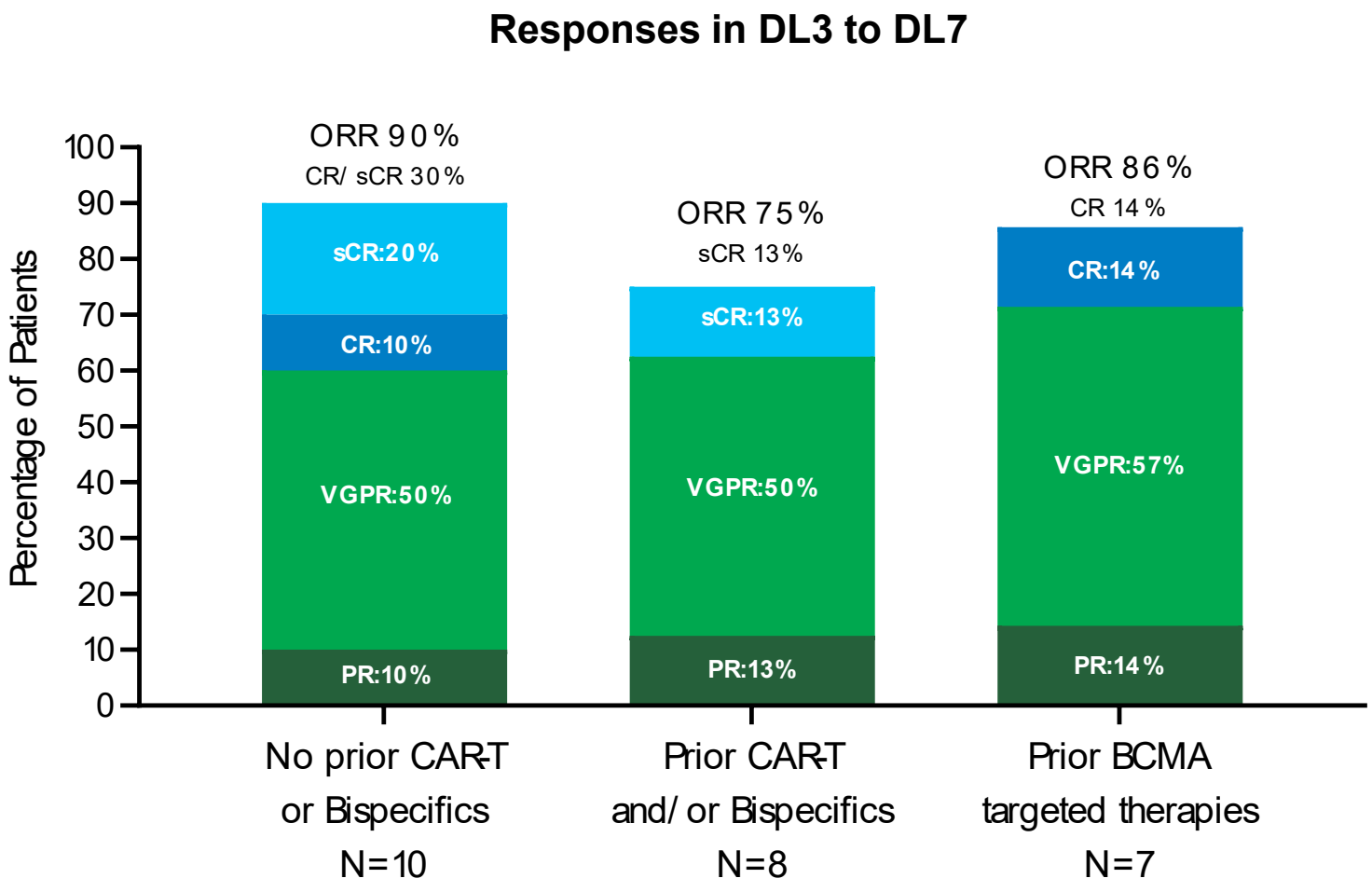
First objective response observed at DL3 (sCR, MRD negative at 10^{-5} level)

Median time to first response was 36 days (range: 29-57)

ISB 2001: High Response Rates In Patients Refractory to Last Line of Therapy, Refractory or Recently Failing CD38 Therapies



ISB 2001: High Response Rates in Patients With or Without Prior BCMA targeted and/or T Cell Directed Therapies*



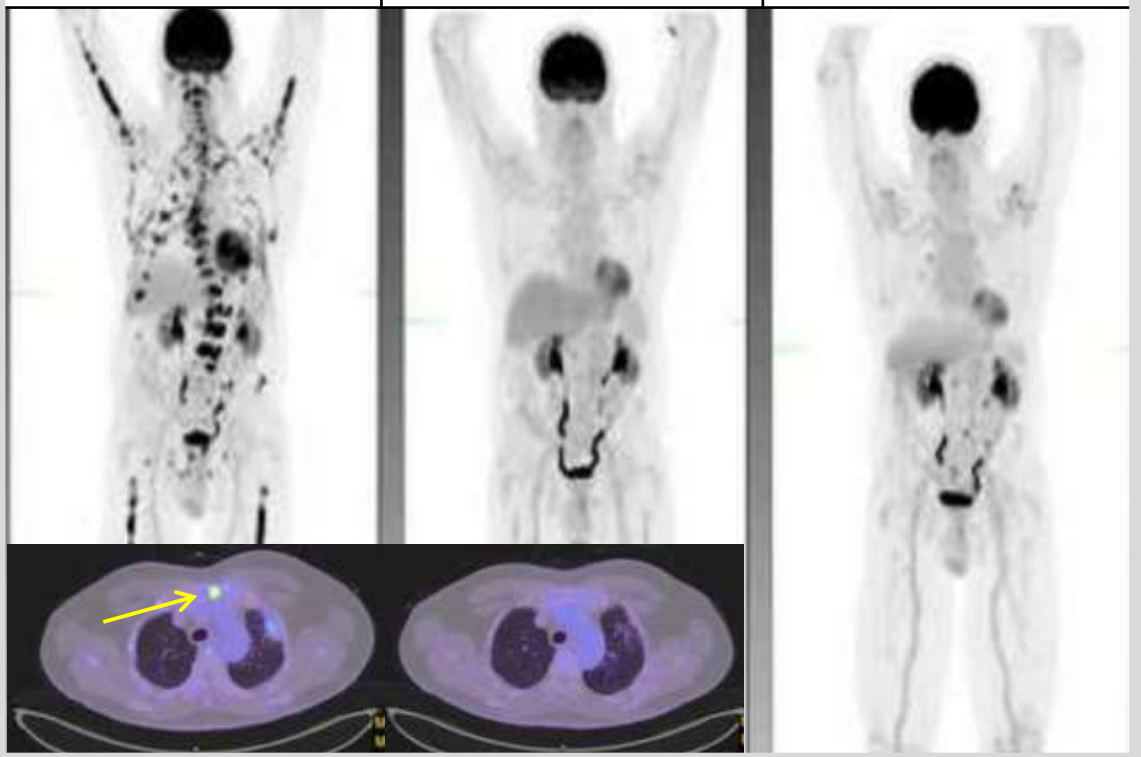
* T cell directed therapies include CD3 bispecifics and CAR T cell therapies

Oral Presentation, ASH Dec 2024

ISB 2001: Rapid and Sustained Responses by PET-CT

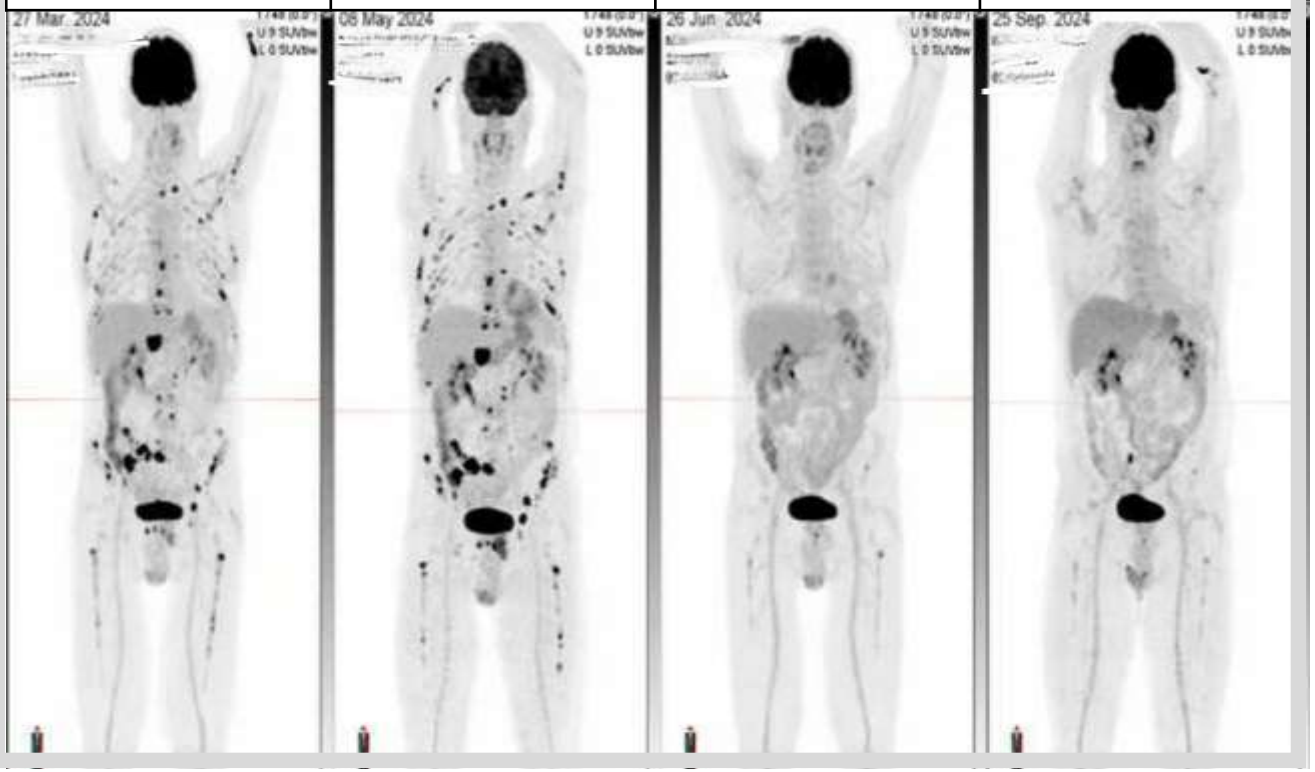
Patient 1 (300 µg/kg): 4 Prior lines

Baseline	After 1 Cycle (PR)	After 6 Cycles (VGPR)

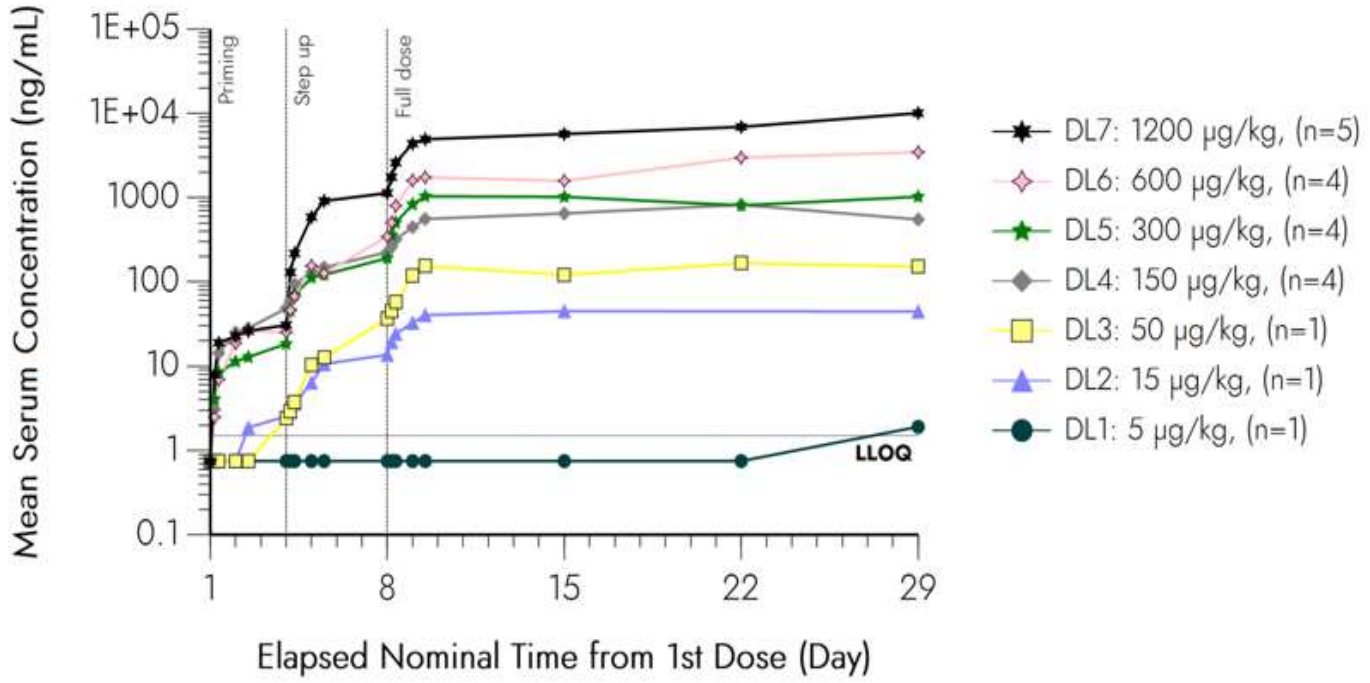


Patient 2 (300 µg/kg): 3 Prior Lines Including T Cell Directed Therapy (Forimtamig)

Baseline	After 1 Cycle (PR)	After 3 Cycles (VGPR)	After 5 Cycles (CR)



Dose-Proportional PK Profile and Long Half-Life Supporting Less-Frequent Dosing Schedule for ISB 2001



Slow absorption, consistent with expected PK profile of SC administration

Near dose proportional increase in serum exposures observed from DL2 to DL7

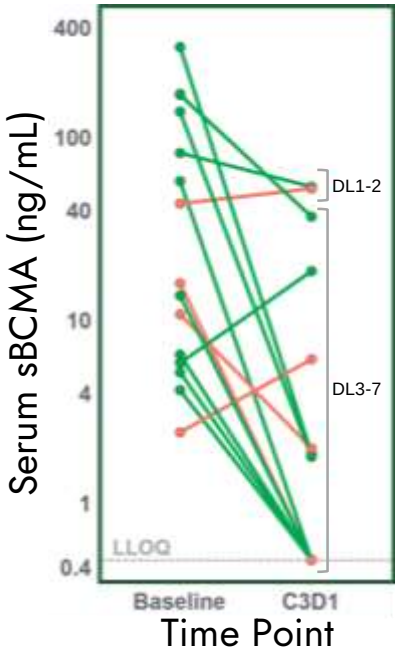
Preliminary half-life of over 10 days

In total 2 out of 20 evaluable patients (10%), one each in DL1 and DL4, showed positive ADA

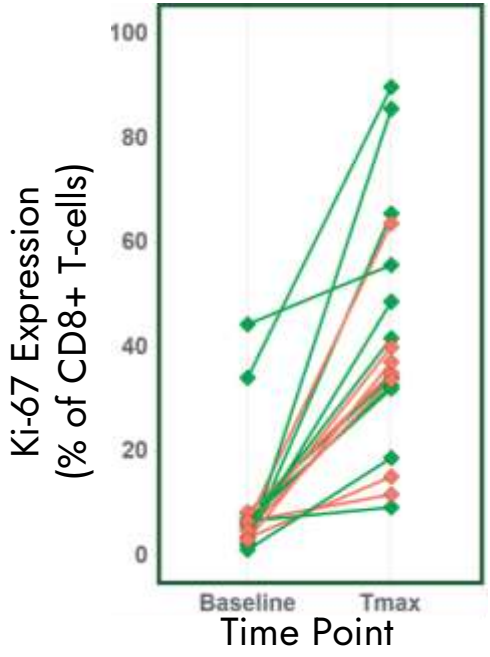
LLOQ: Lower Limit of Quantitation; SC: Subcutaneous; ADA: Anti-Drug Antibody

T-Cell Activation and Reduction of Soluble BCMA After ISB 2001

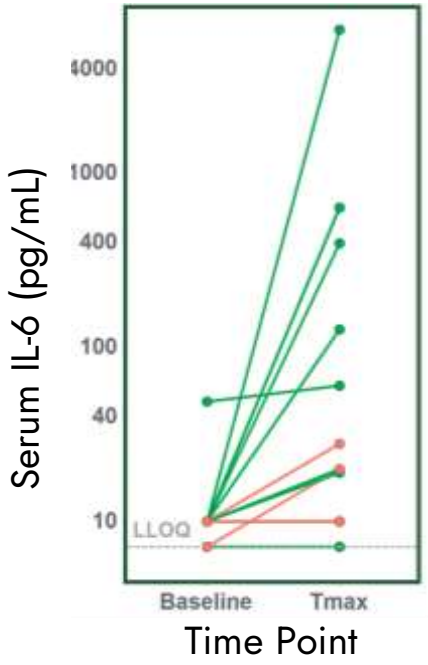
Reduced Serum sBCMA



Increased T Cell Proliferation (Ki-67)



Increased Serum IL-6



Prior CAR-T and/or Bispecifics

Yes
No

Reduction in serum soluble BCMA levels within 1-2 treatment cycles in responding subjects

ISB 2001 induced T cell proliferation and activation (Ki-67, PD-1) in CD4+ and CD8+ cells

Mild increases in serum cytokines observed in most subjects, constant with mild clinical CRS profile

Effects observed in patients previously treated with a CAR-T and/or bispecifics

Early Clinical Results of ISB 2001 Novel TREAT ™ Trispecific

Safety:

- No DLTs up to 1200 µg/kg weekly dosing.
- Mild CRS and injection site reactions, no ICANS.
- Low infection and hematological toxicity rates.

Early and sustained responses were observed across effective dose levels:

- Anti-myeloma activity From 50 µg/kg (MRD-negative sCR) and higher
- 83% ORR overall (22% CR or better, 50% VGPR, 11% PR),
- 90% and 75% ORR in CAR-T/bispecific-naïve or pretreated patients, 86% with prior BCMA therapy, 86% in CD38-refractory patients.

PK and Translational:

- Dose-proportional PK with long half-life supports less-frequent dosing.
- T cell activation observed at effective doses.

Next Steps:

- Escalation continues to 2700 µg/kg, followed by dose-expansion to establish RP2D and best dosing schedule.

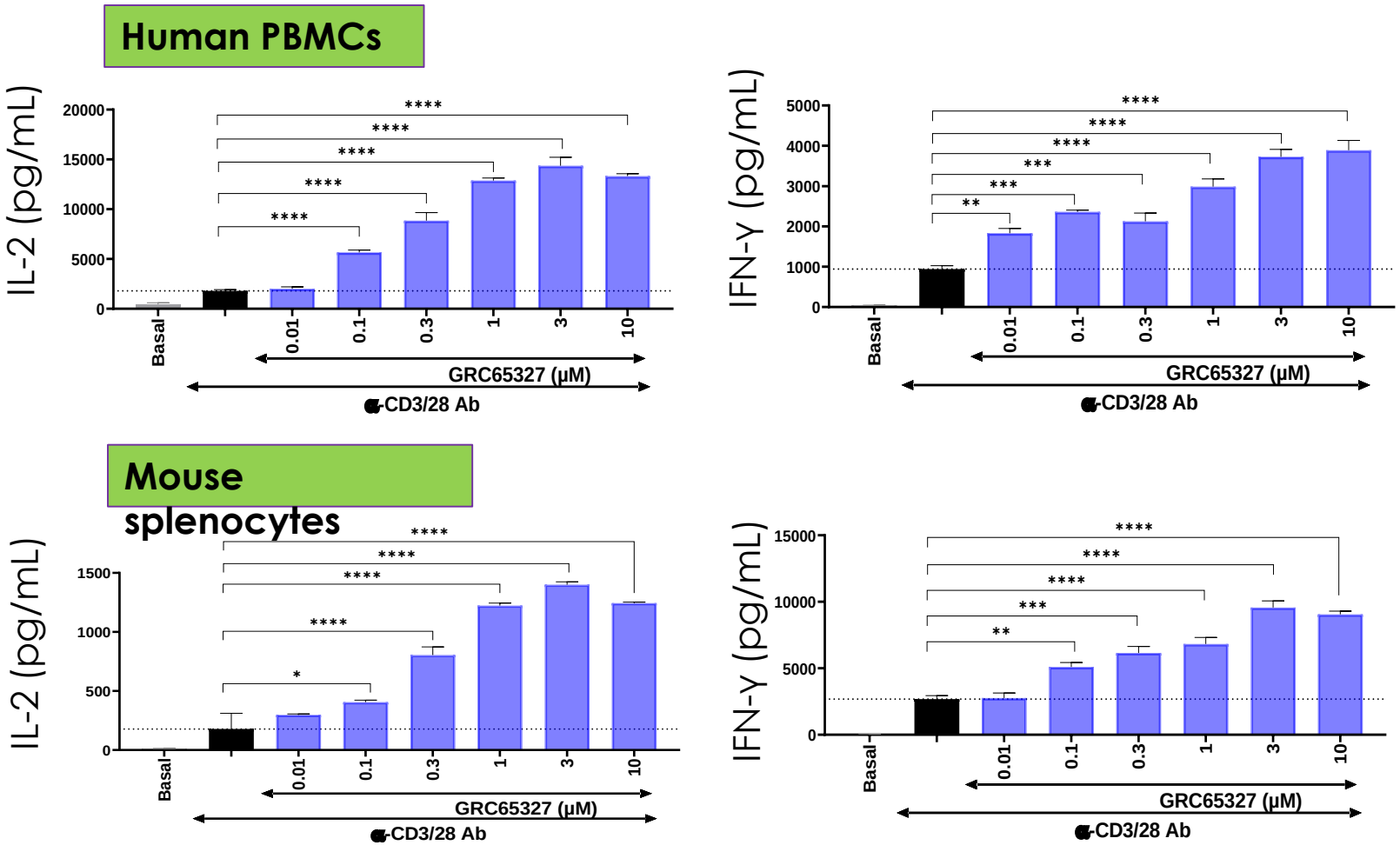
GRC 65327

GRC 65327 – Executive Summary

- Selective, small molecule, orally available, Cbl-b inhibitor, phase I ready for solid tumor indications.
- Demonstrated nM Cbl-b activity, >20-fold selectivity, potentiation of IL-2 and IFN-γ and T cells proliferation.
- Robust immunomodulatory activity by reversing CD28 low T-cell exhaustion and Tumor cells killing
- Significant tumor growth inhibition as a monotherapy and in combination with anti-PD1, while also inducing durable complete responses associated with memory immune responses.
- An increased cellularity in mesenteric lymph nodes, a tissue immune response was noted at very low exposures (AUC ~1500 ng.h/mL) in a 1-month GLP monkey toxicology study.
- FIH based on theoretical HNSTD in dogs as 10 mg BID (20 mg total dose/day)
- IND submission to DCGI completed in October 2024

Choudhary et al., SITC 2024, poster presentation; DOI

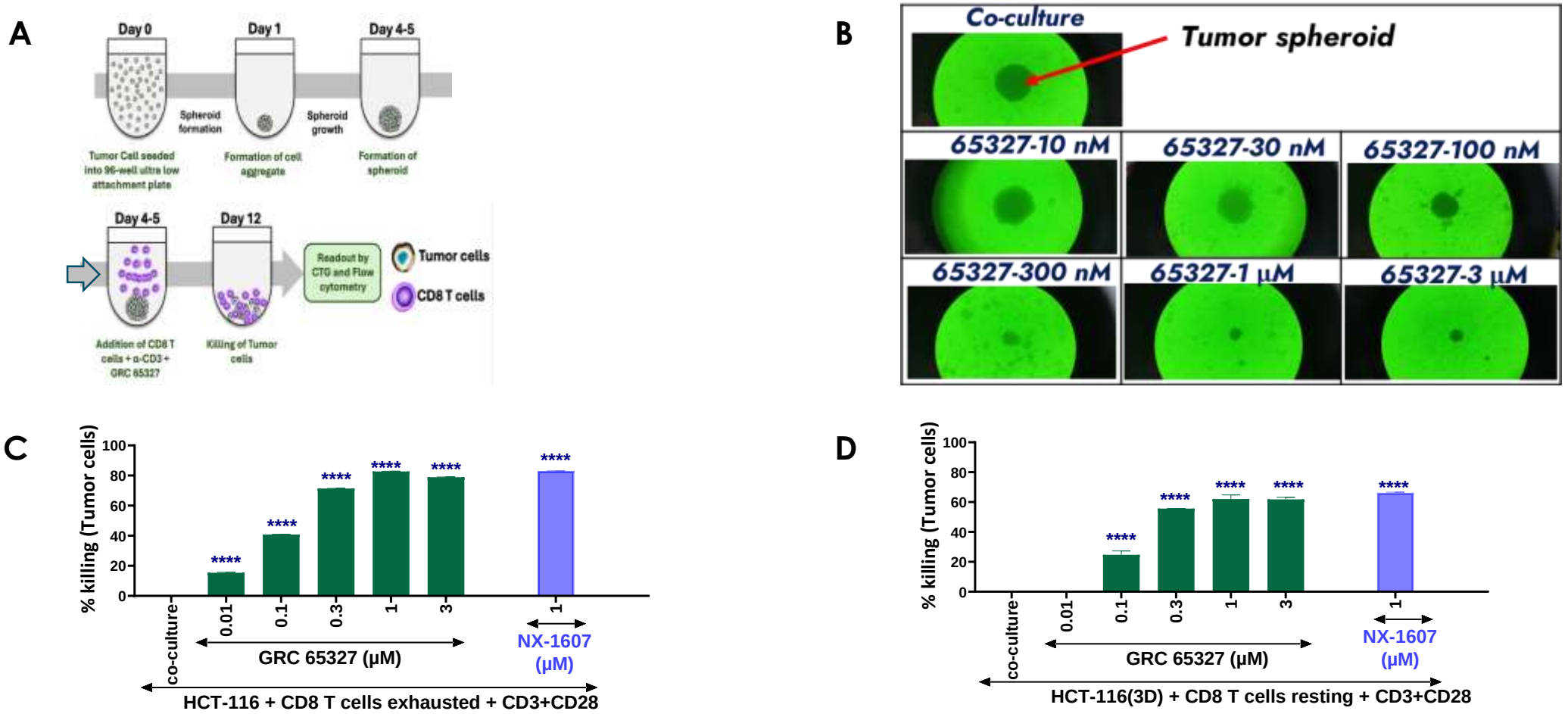
GRC 65327 Demonstrates Potent Immune-Stimulatory Activity



Human PBMCs (upper panel) & mouse splenocytes (lower panel) were treated with GRC 65327 and stimulated with anti-CD3 and anti-CD28 antibodies; cytokine release in supernatant was detected by sandwich ELISA. Statistical significance of differences was evaluated by Dunnett's multiple comparison test. * p< 0.05, ** p< 0.01, *** p< 0.001, **** p< 0.0001

Choudhary et al., SITC 2024, poster presentation; DOI

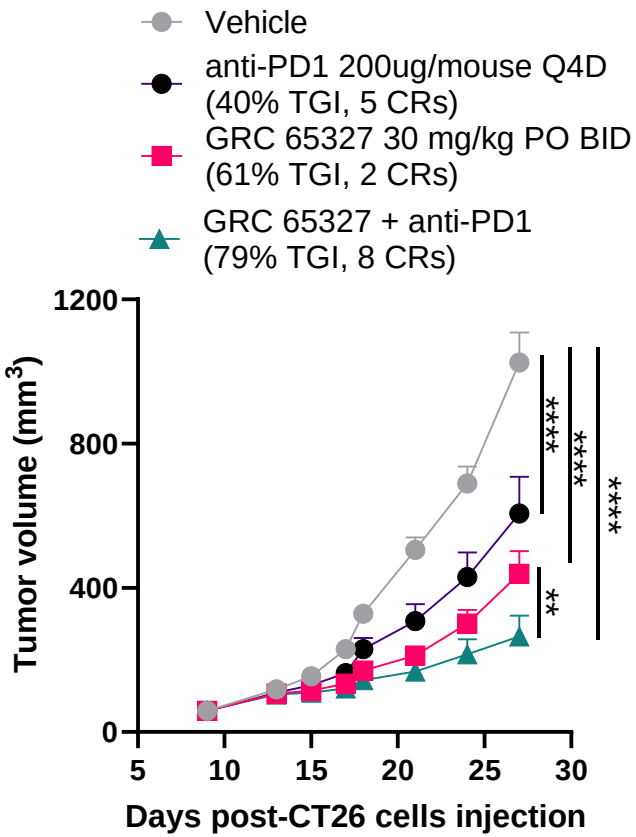
GRC 65327 Facilitates Robust Immune-Mediated Tumor Cell Killing



Human purified resting T cells and exhausted T cells were co-cultured with HCT116 spheroids in the presence of GRC 65327 and anti-CD3 and anti-CD28 antibodies stimulation (A). Microscopic images of spheroid – CD8 T cells co-culture with different concentrations of GRC 65327 (B). Percent tumor cell killing mediated by exhausted CD8 T-cells (C) and resting T-cells (D). Statistical significance of differences was evaluated by Dunnett's multiple comparison test. **** p< 0.0001

Choudhary et al., SITC 2024, poster presentation; DOI

GRC 65327 Enhances Anti-Tumor Immune Response as a Single Agent and in Combination with Anti-PD1 in the CT26 Tumor Model



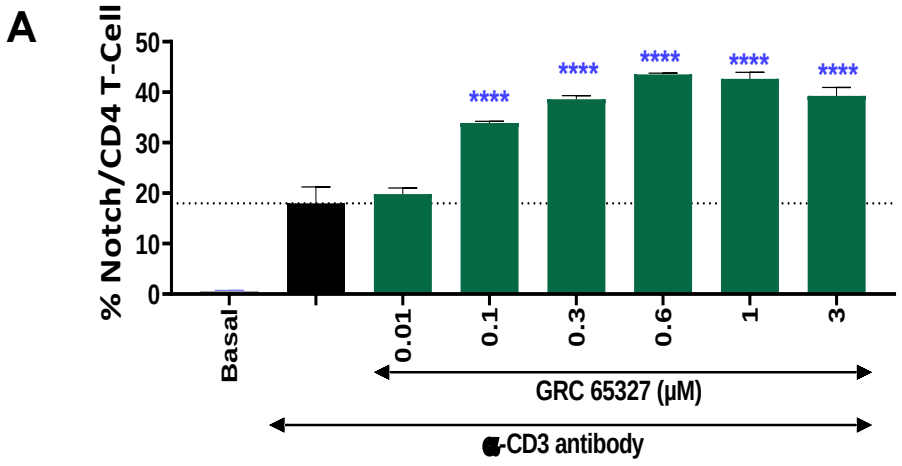
Statistics: 2-Way ANOVA followed by Bonferroni test
***p<0.01, ***p<0.001 ****p<0.0001*

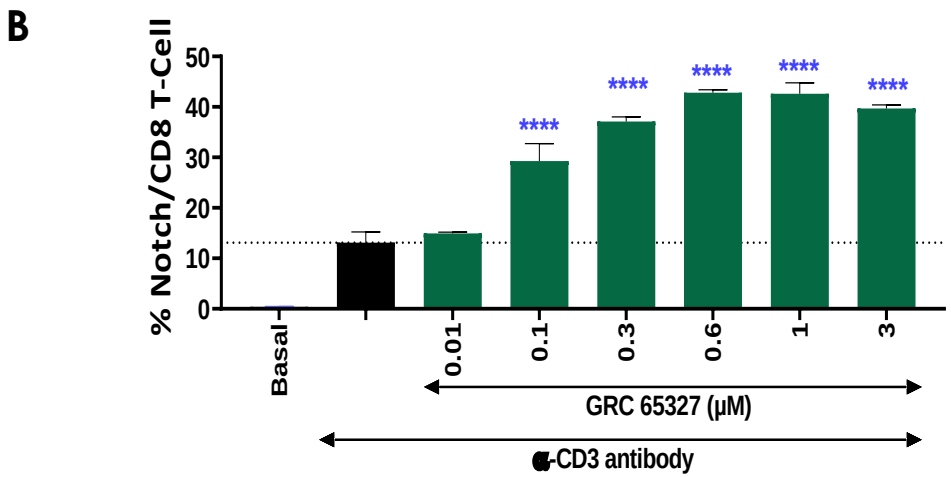
- 0.1 million CT26 cells were implanted subcutaneously into female BALB/c mice
- Animals were randomized when tumor volume reached ~50 mm^3
- Doses: GRC 65327 dosed PO twice daily at 30 mg/kg, anti-PD1 antibody dosed IP BIW at 200µg/mouse.

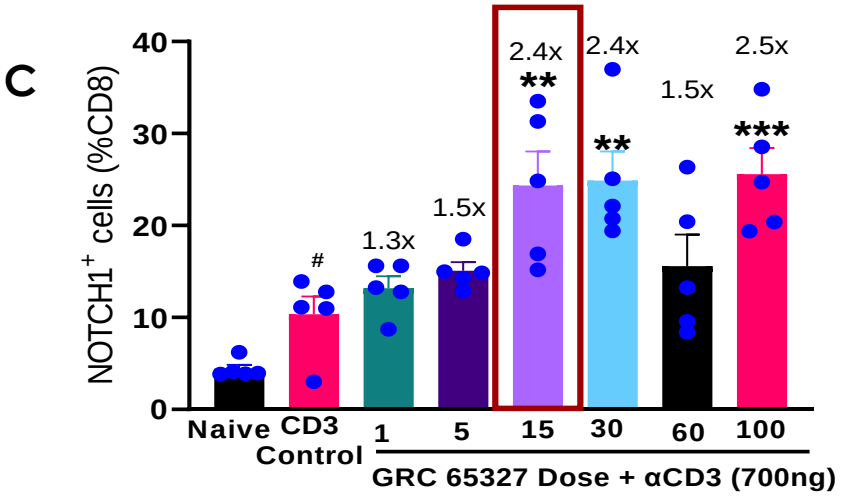
Effective as a monotherapy, GRC 65327 achieved 7-9 complete responses in combination with anti-PD1

Choudhary et al., SITC 2024, poster presentation; DOI

GRC 65327 Demonstrates Ability To Shape TME Via Biomarker Modulation







Human PBMCs were pre-treated with GRC 65327, followed by stimulation with anti-CD3 antibody. Surface expression of Notch1 on CD4 (A) and CD8 T-cells (B). Mice were treated orally with GRC 65327 followed by anti-CD3 antibody IP. Spleen was harvested to measure modulation of Notch1 on CD8 T-cells post 24 h dosing (C). Statistical significance of differences was evaluated by Dunnett's multiple comparison test. **p<0.01, ***p<0.001, ****p<0.0001

Choudhary et al., SITC 2024, poster presentation; DOI

Accomplishments

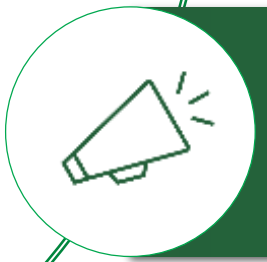
ISB 2001 Trispecific T Cell Engager: *Achieved Clinical Proof-of-Concept*

ISB 1442 Myeloid-Cell Engager: *Program Discontinued*

GRC 65327 Cbl-b Inhibitor: *IND Submission Completed with DCGI in India*

Alliance Formation: *Partnership between Ichnos Sciences and Glenmark, Establishing IGI*

New Biologics Manufacturing Strategy: *Shifting from In-House Production to Specialized CDMOs*



ASH24 Annual Meeting: *First Presentation of Clinical Data with ISB 2001*

Thank You!
Together, Let's Accelerate the Cure for Cancer

ICHNOS GLENMARK INNOVATION
Collaboration propels innovation