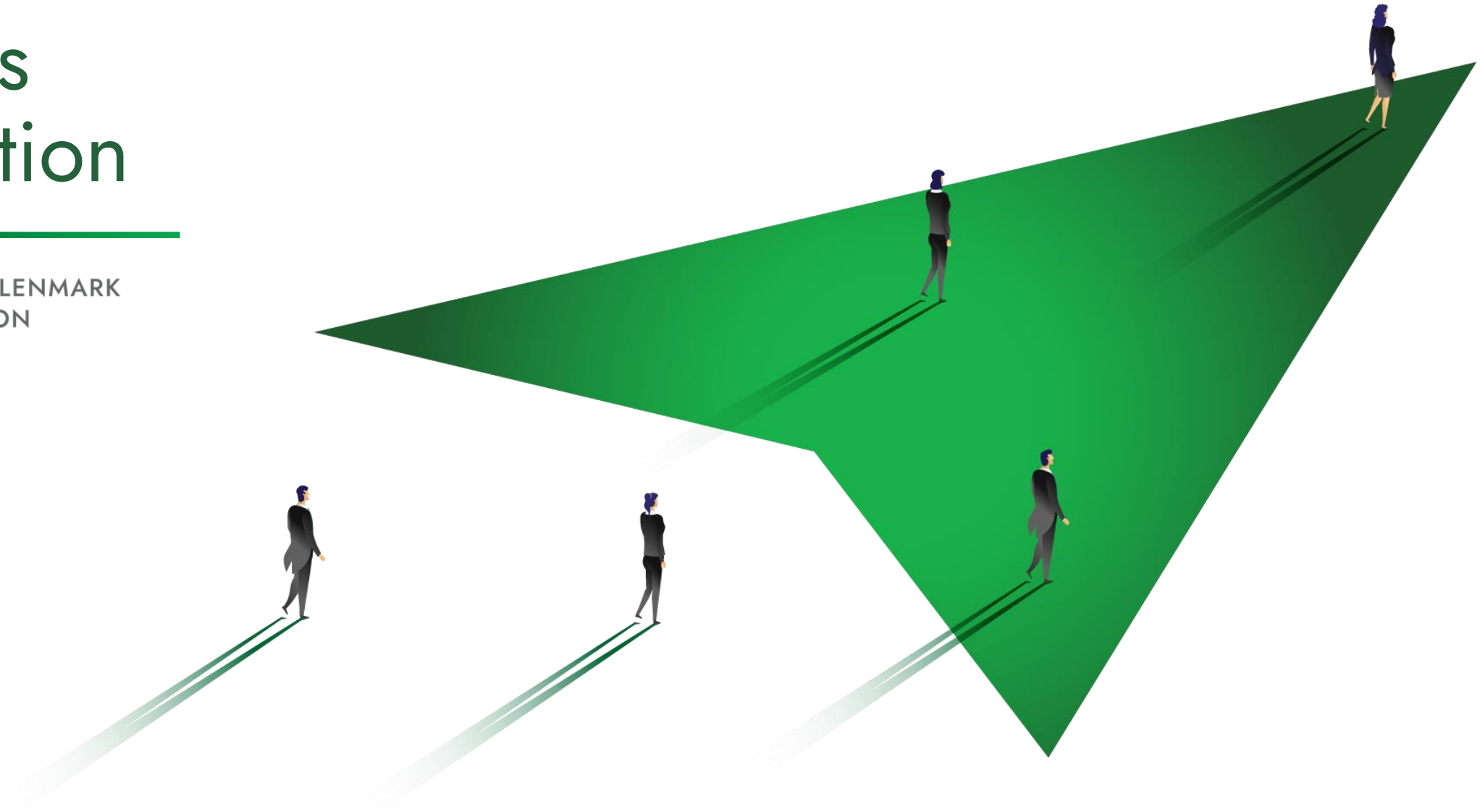
Collaboration propels innovation

ICHNOS GLENMARK INNOVATION

Corporate Presentation
March, 2024



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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 and small molecules: discovery, antibody engineering, clinical development
- Global footprint: U.S., Switzerland and India
- Ongoing discussions for divesting the biologics manufacturing plant

Complementary Biologics and Small Molecules Discovery engines

- Proprietary protein engineering platform (BEAT®) allowing maximal flexibility and manufacturability of full length multispecific antibodies
- Structure-based SMs and degrader discovery platform powered by computational chemistry to design molecular entities with desired binding mode, functional activity, selectivity and degradation
- Dual-pronged research strategy based on multispecific immune cell engager antibodies that can engage multiple targets on cell surface and small molecules modulating intracellular pathways in cancer or immune cells

Robust Pipeline

- Clinical stage pipeline in Oncology
- 6 initial programs targeting hematologic malignancies and solid tumors
- Engaging different types of immune cells
- Open to asset-level partnerships with global immuno-oncology leaders
- Auto-immune programs led by Alliance Partners

BEAT®: Bispecific Engagement by Antibodies based on the TCR

Highly Experienced Leadership Team

LEADERSHIP

CYRIL KONTO, M.D.
President and Chief Executive Officer

LIDA PACAUD, M.D.
Chief Medical Officer

MARIO PERRO, Ph.D.
Head of Biologics Research

NAGARAJ GOWDA
Head of Small Molecule Research

DEAN THOMAS, J.D.
General Counsel

SEBASTIEN CHENUET, Ph.D.
Head of Business Development

EVA YUEN
Head of Finance

KARISHMA SIPAHIMALANI, Ph.D.
Head of Human Resources

PREVIOUS EXPERIENCE

Pfizer
Allogene Therapeutics
Bristol Myers Squibb
Roche
Legend Biotech
Novartis
sanofi
Aurigene Oncology — Conquering Cancer
Debiopharm
Glenmark — A new way for a new world

BY THE NUMBERS

110+
Years combined experience in biotech and pharmaceuticals

30+
Products developed or launched

40+
Mergers, acquisitions, IPOs and other transactions

Accomplished Governance Board With Track Record of Success

GLENN SALDANHA
Chairman and Managing Director,
Glenmark Pharmaceuticals Limited

CYRIL KONTO, M.D.
President and Chief Executive Officer
Ichnos Glenmark Innovation

DAVID LUBNER
Former CFO of Ra Pharma

DENNIS PURCELL
Founder of Aisling Capital and
Former Senior Managing Partner

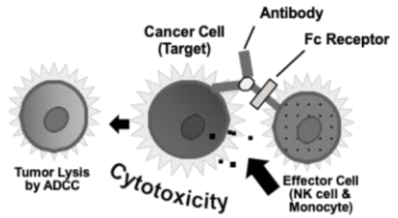
LAWRENCE OLANOFF, M.D., Ph.D.
Former President and COO of
Forest Laboratories

V S MANI
Global CFO of Glenmark Pharmaceuticals Limited

ALIND SHARMA
Global CHRO of Glenmark Pharmaceuticals Limited

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

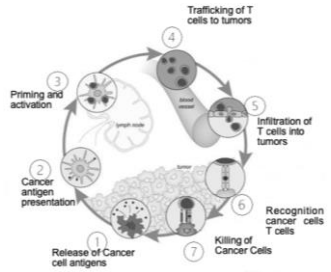
1990s



TARGETED THERAPIES

Fc Function-based Tumor Killing

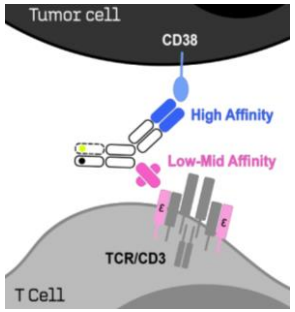
2010s



IMMUNO-ONCOLOGY

Checkpoint and Innate Immunity Modulators

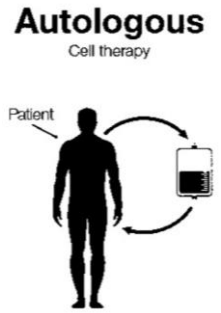
2014



BISPECIFIC ANTIBODIES

CD3 T-Cell Engagers

2017



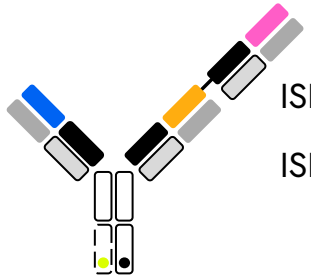
CAR-T CELLS

Engineered T-Cells

Next Wave

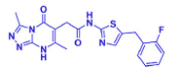
MULTISPECIFICS AND SM MODULATORS

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



ISB 1442

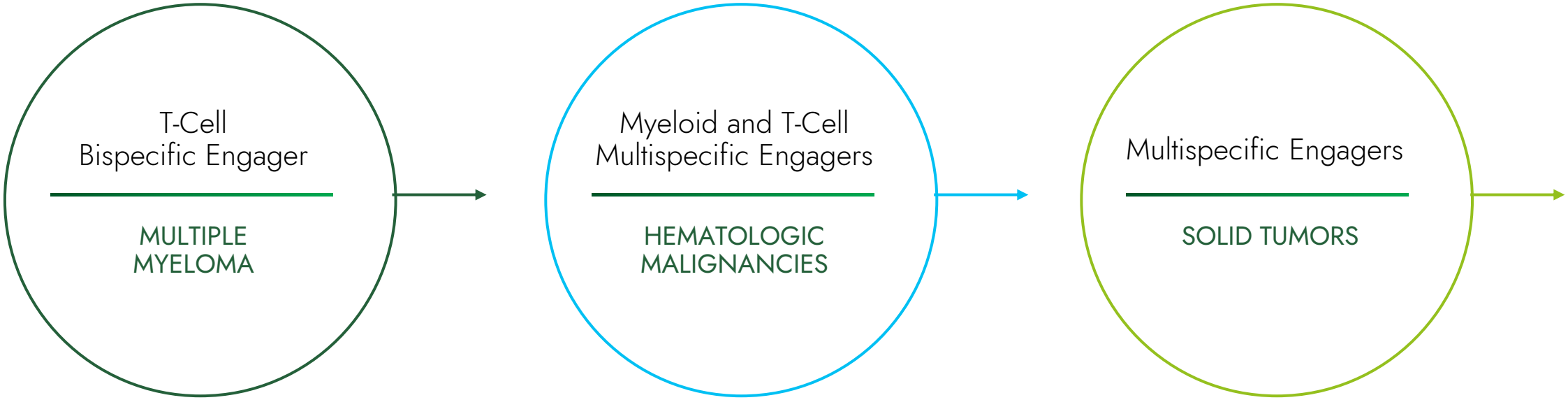
ISB 2001



GRC 65327

IRAK-M

Multispecifics: Strategy Starts with a Validated Target in Multiple Myeloma, hen Expands



ISB 1342, a CD38 X CD3 BEAT® bispecific antibody

- ISB 1442, a first-in-class CD38 X CD 47 2+1 Biparatopic BEAT® bispecific antibody
- ISB 2001, BCMA X CD38 X CD3 TREAT™ trispecific antibody

ISB 2301, BEAT® NK-Cell Engaging Multispecific Platform

BEAT®: Bispecific Engagement by Antibodies based on the TCR
TREAT™: Trispecific Engagement by Antibodies based on the TCR

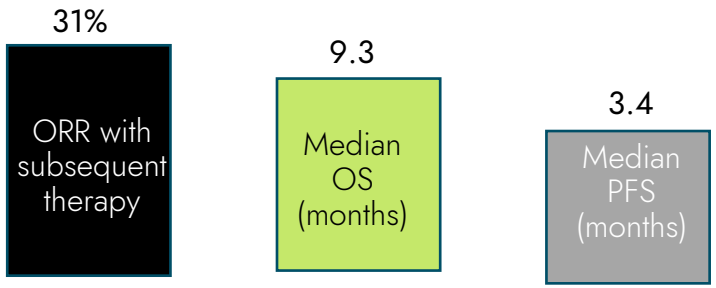
Portfolio Addresses Unmet Needs in Multiple Myeloma, Overcomes Limitations of Select Therapies

HIGH UNMET NEED AND LARGE MARKET

160000

Global multiple myeloma cases annually[1]

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
- CD47-targeted therapies not suitable as single agents
- Few options following failure of BCMA-targeted therapies

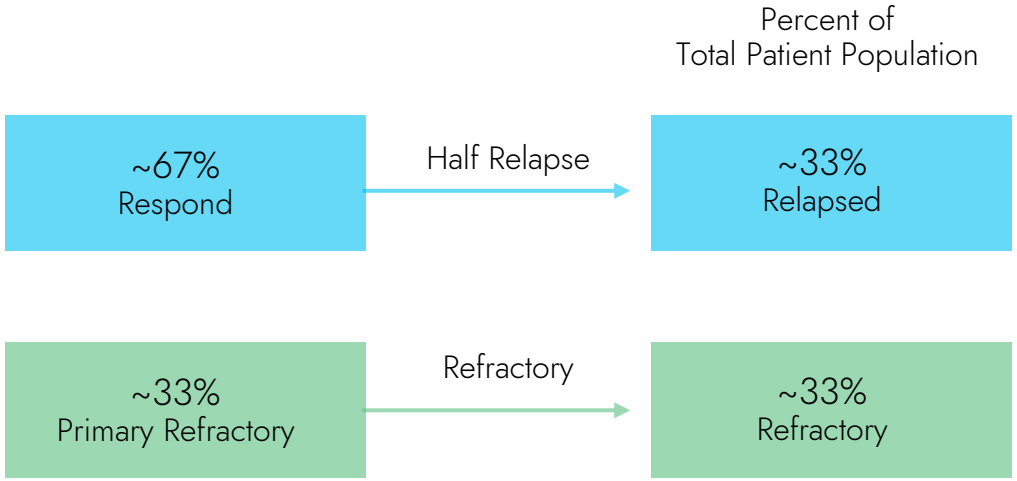
1 Oncologist. 2020 September;25(9):e1406-e1413 | 2 Gandhi UH et al. Leukemia 2019; 33: 2266–75.
3 Saltarella I. et al. CELLS 2020 Following Proteasome inhibitors, Immunomodulators, and Anti-CD38 therapies.

ISB 1442 Addresses Unmet Needs in Acute Myeloid Leukemia, Overcomes Limitations of Current and Developing Therapies

HIGH UNMET NEED AND LARGE MARKET (2020)

41000+ New AML patients in the U.S., France, Germany, Italy, Spain, UK and Japan[1]

High rates of resistance and recurrence[2]



LIMITATION OF CURRENT AND DEVELOPING THERAPIES

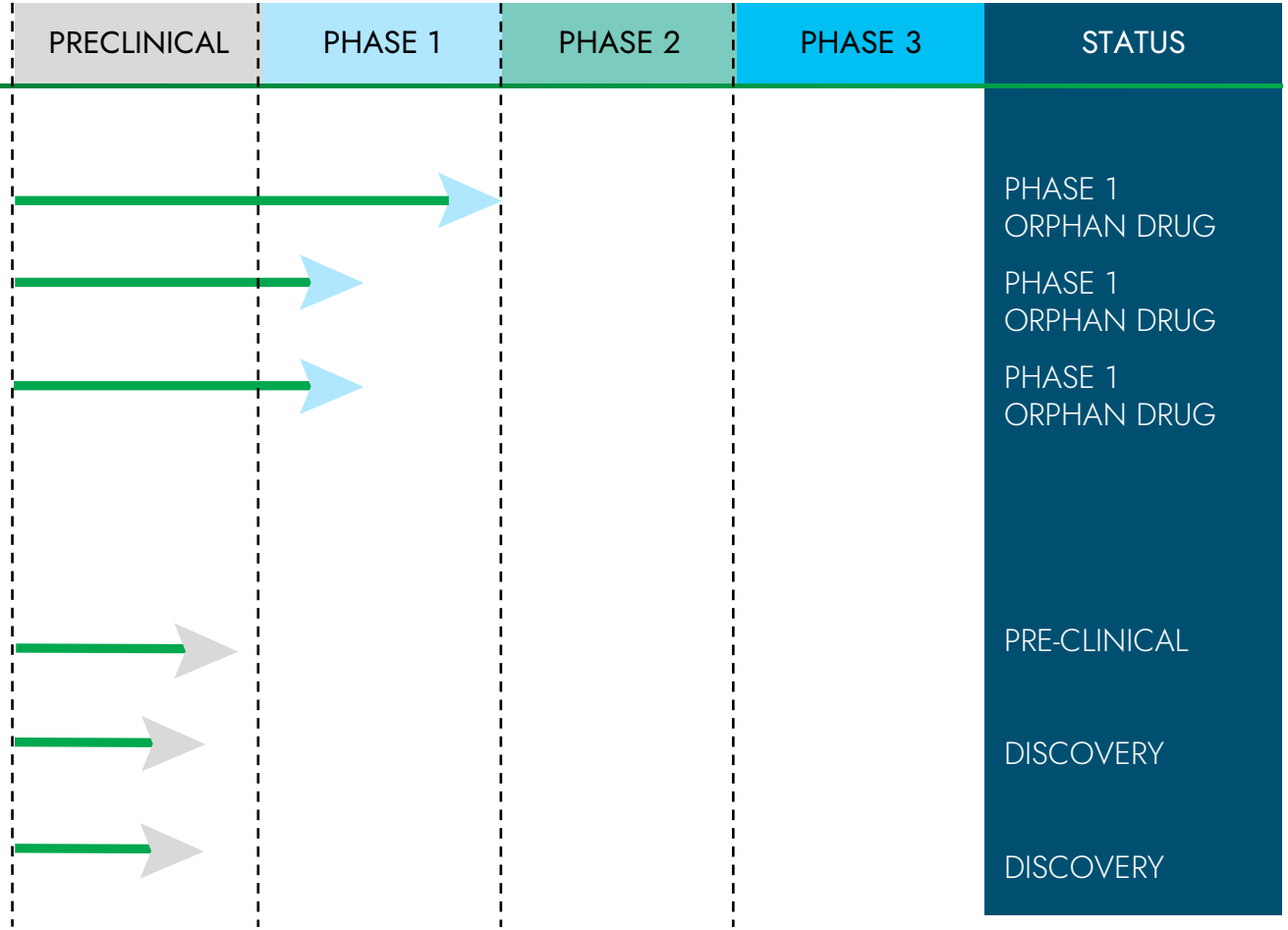
- Few late stage, emerging agents for AML
- Standard of care for R/R AML lacks desired efficacy and durability
- Multiple, persistent and/or emerging mechanisms of resistance may be overcome by co-targeting CD38 and CD47

1 DelveInsight, "Acute Myeloid Leukemia (AML) - Market Insight, Epidemiology and Market Forecast – 2032"
2 American Cancer Society, "Treatment Response Rates for Acute Myeloid Leukemia (AML)"

Pipeline

Diversity of Immune Cell Engagement and Indications Across Hematologic and Solid Tumors

ASSET	DESCRIPTION	INDICATION	STATUS
CLINICAL ASSETS			
ISB 1342*	CD38 x CD3 BEAT™ bispecific antibody	Multiple Myeloma	PHASE 1 ORPHAN DRUG
ISB 1442	CD38 x CD47 BEAT™ bispecific antibody	Multiple Myeloma; AML planned	PHASE 1 ORPHAN DRUG
ISB 2001	BCMA x CD38 x CD3 TREAT™ trispecific antibody	Multiple Myeloma	PHASE 1 ORPHAN DRUG
CANDIDATES			
GRC 65327	Cbl-b Inhibitor	Solid Tumors	PRE-CLINICAL
ISB 2301	IMMUNITE NK-cell engager	Solid Tumors	DISCOVERY
IRAK-M	Interleukin-1 receptor associated Kinase-3 inhibitor	Solid Tumors	DISCOVERY



** Open for partnership*

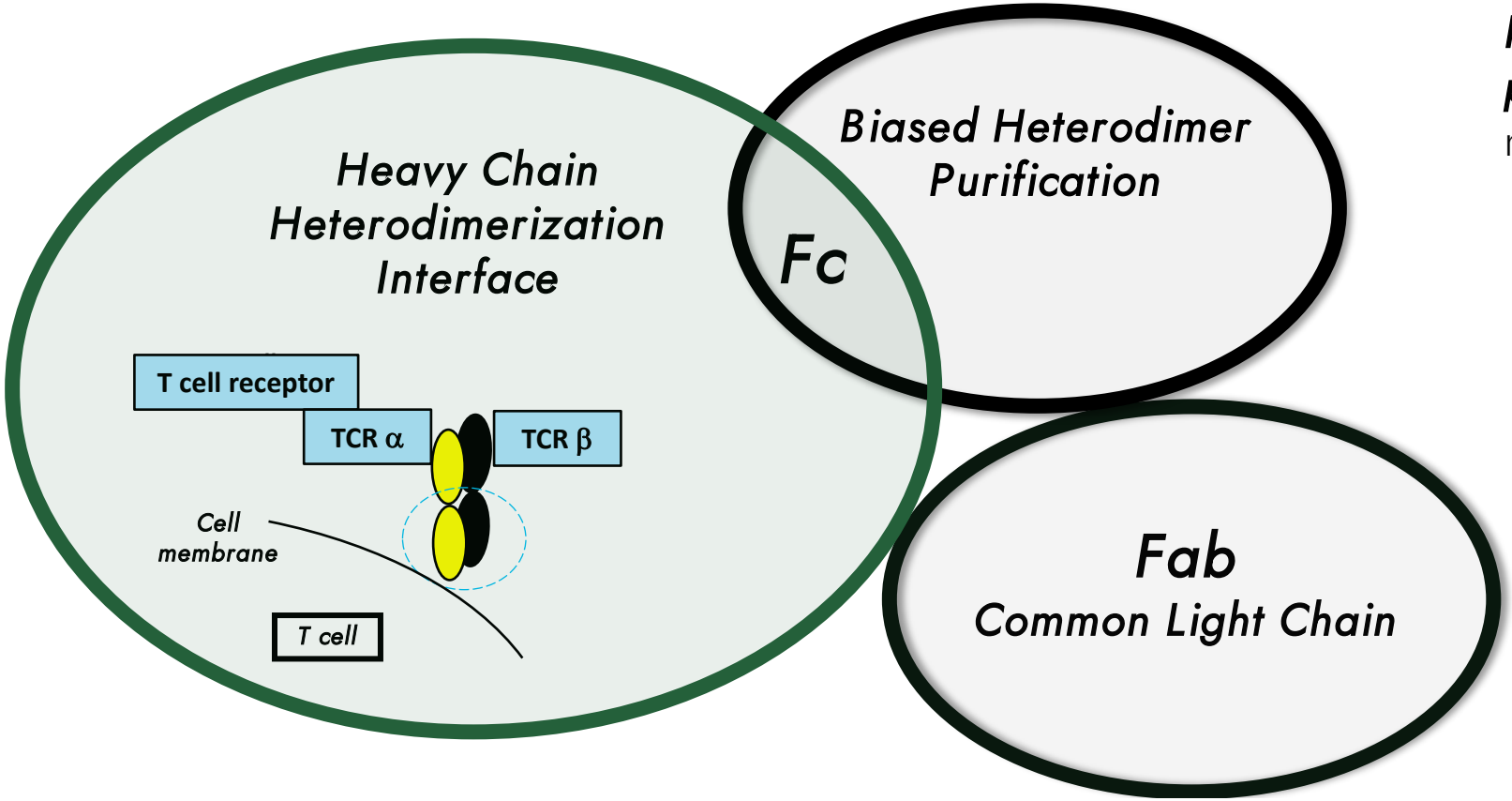
Out-Licensing Autoimmune Disease Programs to Enable Greater Focus on Oncology Clinical Studies

PRODUCTS	TARGET	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3	STATUS
Licensed to astria THERAPEUTICS	$320 million for upfront payment, development, regulatory and sales milestone payments, plus tiered royalties on global sales					
Telazorlimab and ISB 830-X8	OX40 antagonist monoclonal antibody	Atopic Dermatitis →				SUCCESSFUL PHASE 2B*
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	Autoimmune Disease →				PHASE 1

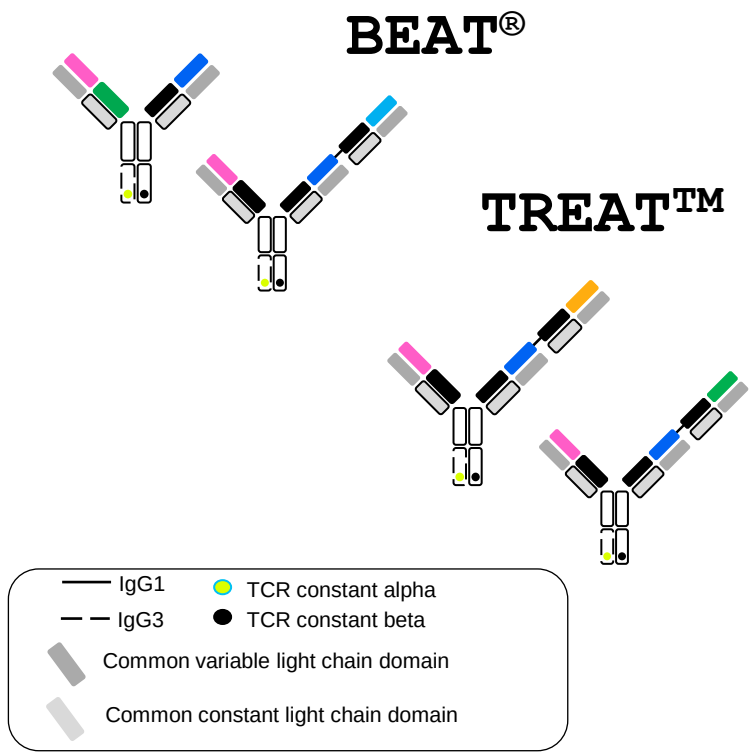
* *A US IND for rheumatoid arthritis and other autoimmune indications is active*

BEAT® Platform

BEAT® Combines TCR Interface-Based Hc Pairing and Common Lc to Streamline the Creation and Development of Multispecific Antibodies



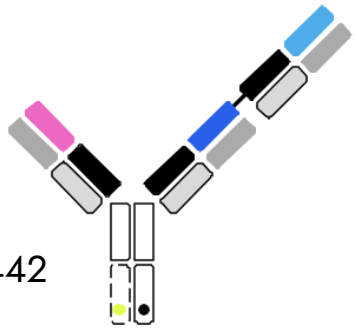
Proprietary plug-and-play modular platform enables a plurality of multispecific configurations



BEAT®: Bispecific Engagement by Antibodies based on the TCR
TREAT™: Trispecific Engagement by Antibodies based on the TCR

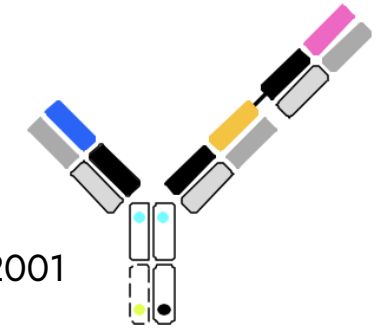
BEAT® Enables Production of Multispecific Antibodies with Competitive Developability Properties

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 1342

ISB 1342 - Executive Summary

- Phase I, first-in-class, bispecific CD38xCD3 antibody developed in RRMM

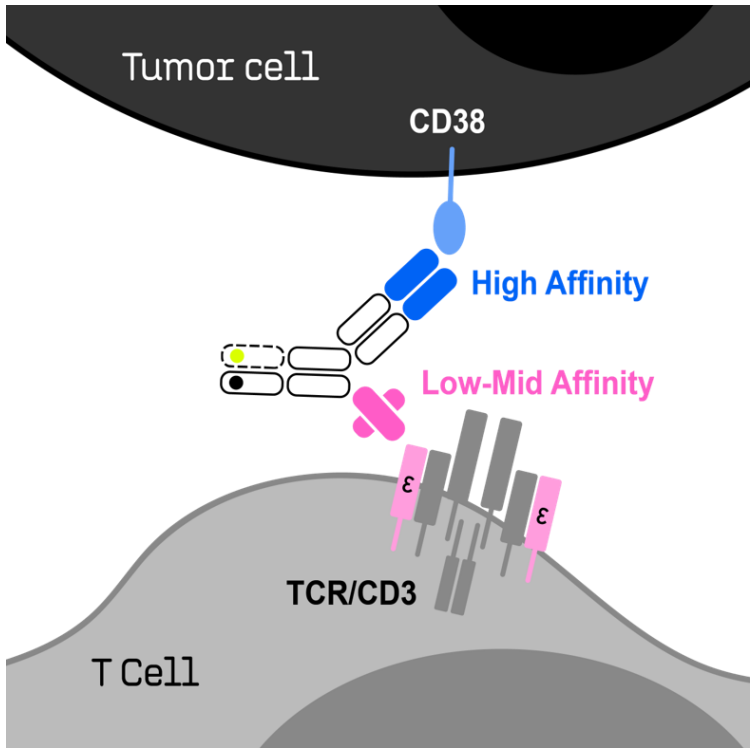
- Designed to overcome Daratumumab resistance mediated by low CD38-expressing tumor cells and resistance to CDC and ADCC:
  + ISB 1342 demonstrated cytotoxic potency and tumor growth inhibition superior to daratumumab
  + ISB 1342 demonstrated killing of multiple myeloma cells resistant to Daratumumab
  + ISB 1342 does not compete with Daratumumab for the same binding epitope on CD38 thus not requiring a wash out period in clinic

- ISB 1342-101 Study in Triple Refractory Patients with R/R MM demonstrated a favorable safety profile and preliminary anti-myeloma effect in a heavily pre-treated patient population

- Available for partnerships due to pipeline strategic reprioritization

ISB 1342 (CD38 x CD3) Bispecific Antibody: Potential First-in-Class Therapy in Relapsed/Refractory Multiple Myeloma

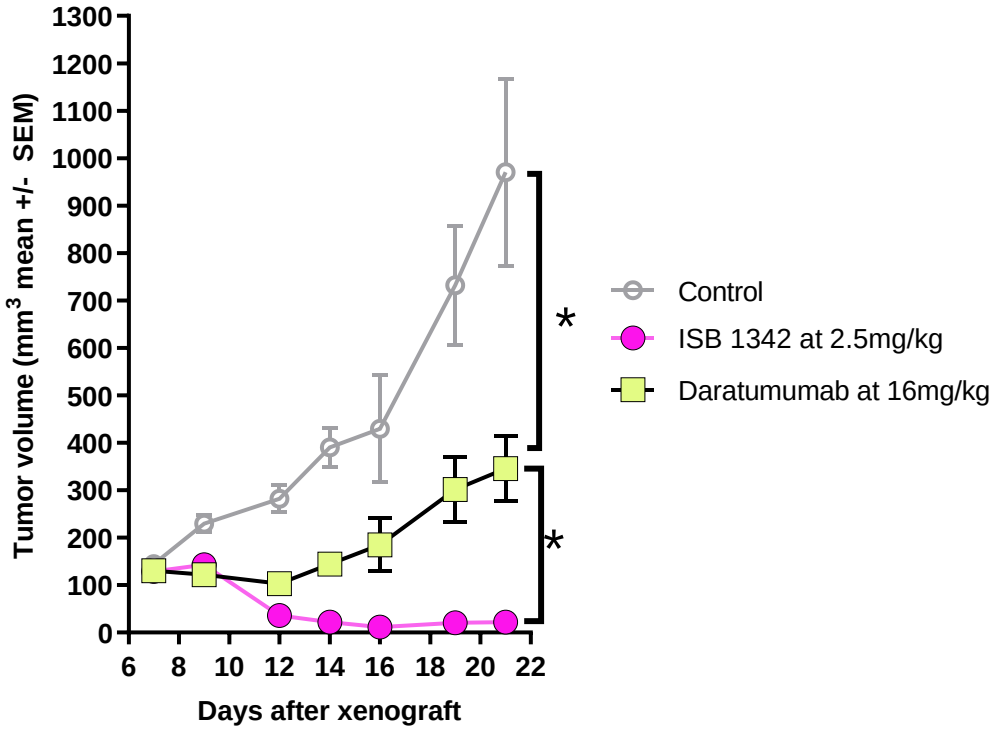
ISB 1342 (CD38 X CD3)
bispecific antibody

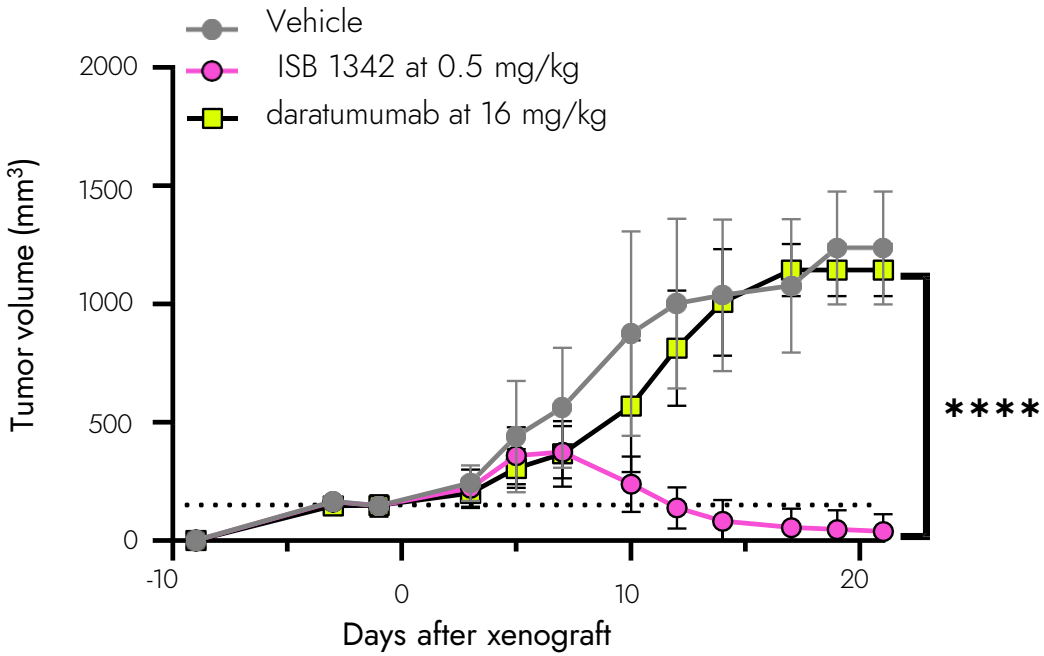


KEY ATTRIBUTES

- CD38 is expressed on the surface of multiple myeloma cells and is a clinically validated target
- ISB 1342 is a bispecific antibody that redirects T cells to kill CD38-expressing tumor cells in MHC-antigen-independent manner
- ISB 1342 binds to a proprietary anti-CD38 epitope, which is different from that of daratumumab
- ISB 1342 is designed to overcome:
  + Daratumumab resistance by killing low CD38-expressing tumor cells
  + Resistance to CDC and ADCC mediated by daratumumab
- ISB 1342 was Granted Orphan Drug Designation for Multiple Myeloma by U.S. FDA

ISB 1342 Effectively Controls Tumor Growth *In Vivo* and demonstrates tumor growth inhibition superior to daratumumab

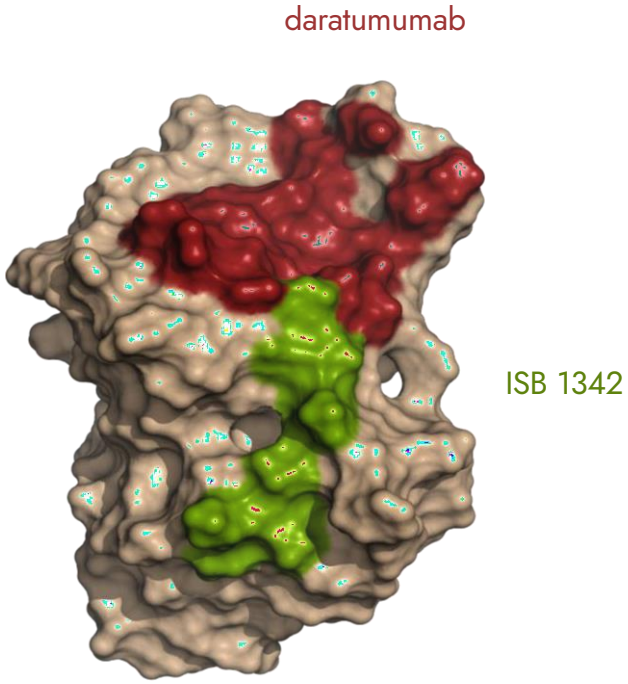




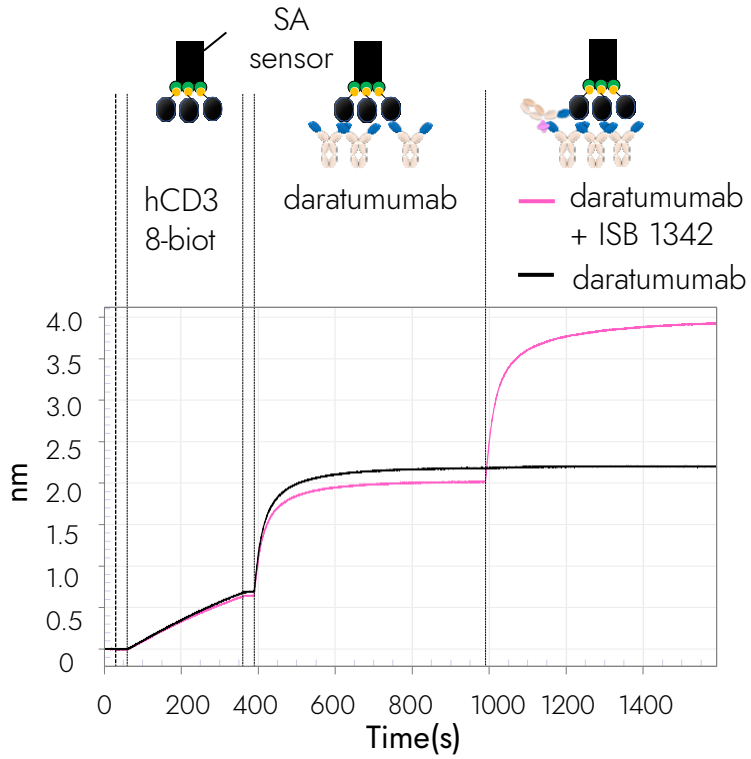
NOD-SCID mice were xenografted subcutaneously with human peripheral blood mononuclear cells and Daudi cells. ISB 1342 or daratumumab were injected intravenously weekly when tumor reached 100 mm³ and tumor growth monitored over two weeks ***(left panel).*** *(Mann Whitney test). * = p<0.05*

ISB 1342 retains cytotoxic potency in the presence of Daratumumab, which obviates the need for a wash out period in clinic

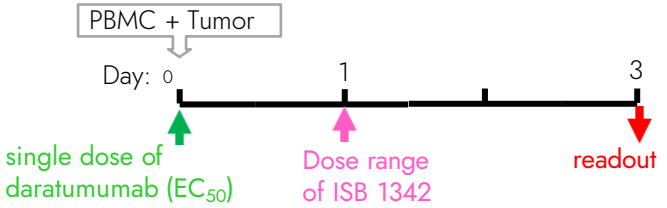
Epitope mapping of daratumumab and ISB 1342 on CD38 demonstrates distinct epitope binding sites

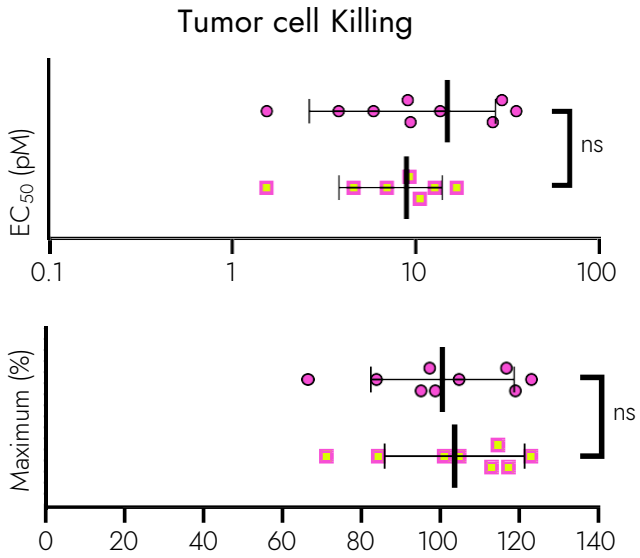


ISB 1342 does not compete with daratumumab and can engage CD38 prebound by daratumumab



ISB 1342 Cytotoxicity of NCI-H929 MM cell line is not impacted by pre-treatment of daratumumab

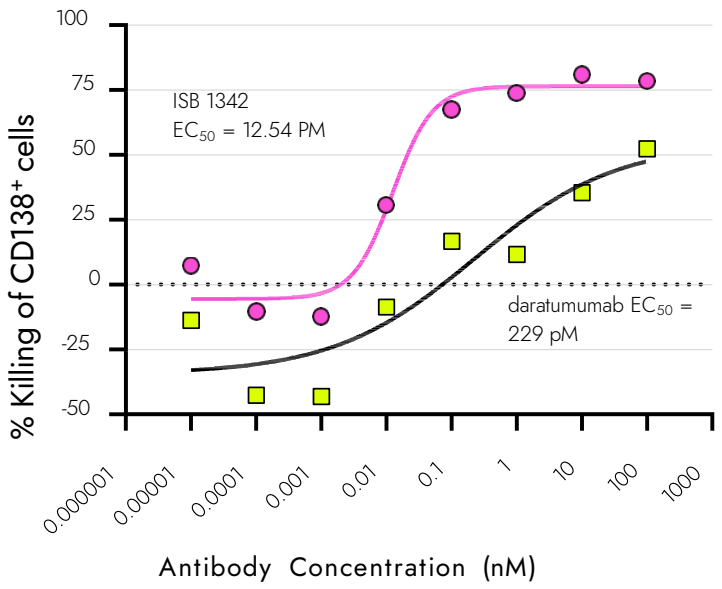




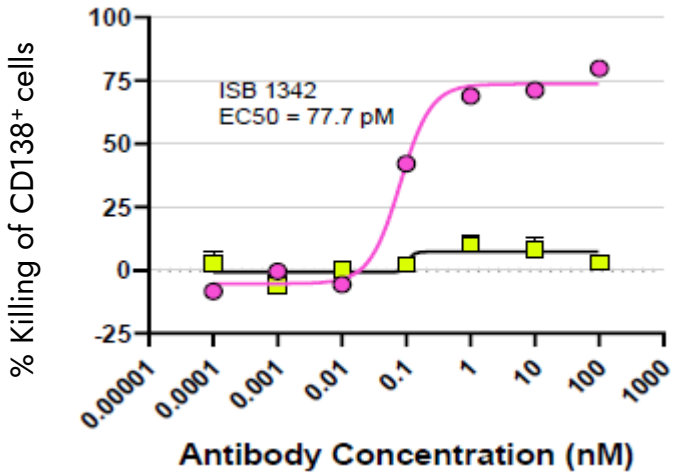
Pouleau B. et al. Blood 2023

ISB 1342 Demonstrates Superior Cytotoxic Potency relative to Daratumumab in Several CD38+ patient-derived hematological malignancies

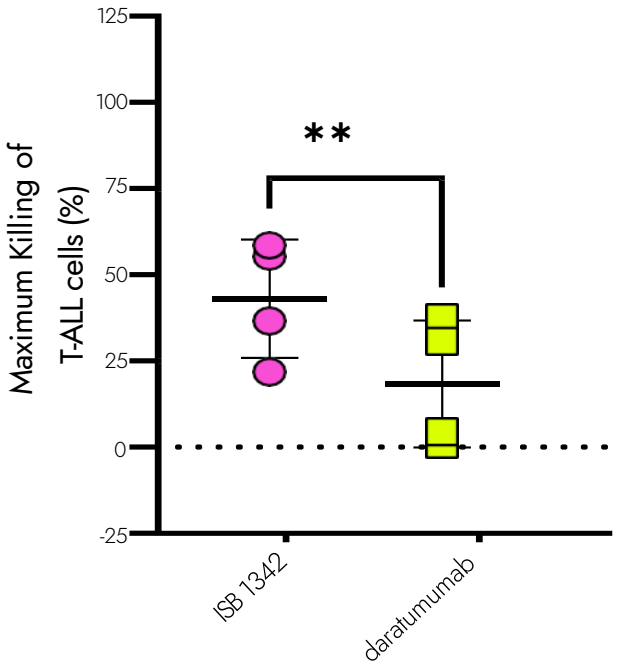
Waldenstrom Macroglobulinemia
dara-naive



Plasma Cell Leukaemia
dara-naive



T-Acute Lymphocytic Leukaemia

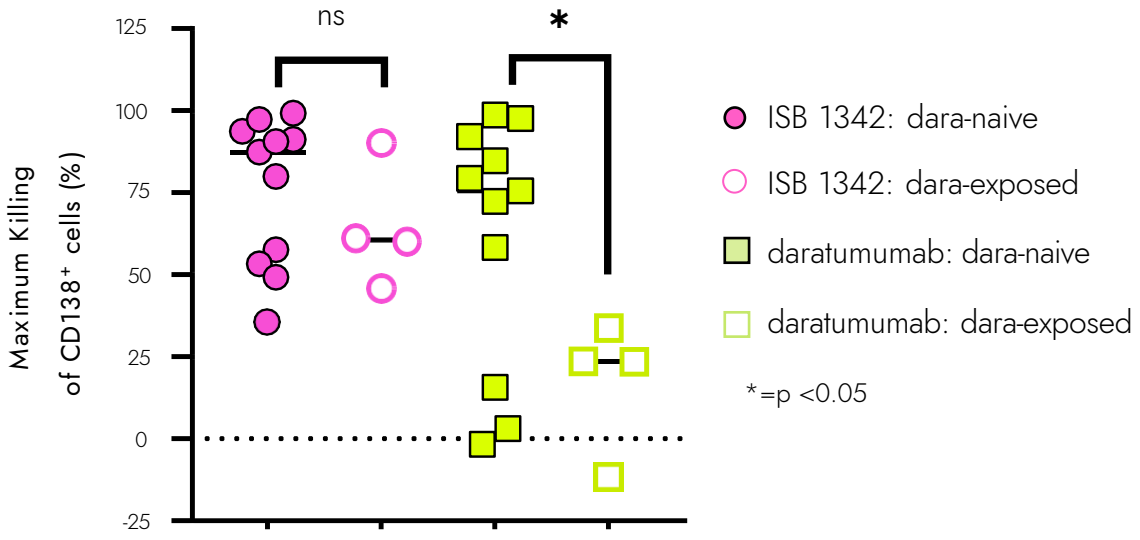


ISB 1342 Kills Multiple Myeloma Cells That Are Resistant to Daratumumab

Many patients relapse when they become resistant to anti-CD38 therapies (e.g., daratumumab).

ISB1342 was designed to be active ***regardless of CD38 expression***.

- **Demonstrated Efficacy:**
  - In vivo – ISB 1342 induced complete tumor eradication in mice models
  - In vitro – ISB 1342 maintains high potency to kill tumor cells in bone marrow samples from patients previously treated with daratumumab (figure at right)
- **Demonstrated Safety:**
  - Acceptable toxicology profile in cynomolgus monkeys



Maximal cytotoxicity of CD138+ tumor cells with ISB 1342 (10-100 nM) or daratumumab (100 nM) in samples from dara-naïve patients (filled symbols) vs dara-exposed (open symbols). Dots represent individual samples, and data are mean ± SD compared using 1-way ANOVA followed by Dunnett multiple comparison analysis to daratumumab on dara-naïve samples.

Results suggest ISB 1342 may be an option for patients whose multiple myeloma is resistant to anti-CD38 therapies.

ISB 1342-101 Clinical Trial Design

ISB 1342-101 Study in Triple Refractory Patients with R/R MM

Part 1

3+3 Dose Escalation
Enrolling Patients at 16 µg/kg IV

- Steroids premedication
- Step up dosing

Enrollment Closed
29 patients enrolled in biweekly dosing cohorts
52 patients enrolled in weekly dosing cohorts

Primary endpoint: MTD

NCT03309111

Clinical Proof-of-Concept for BEAT®

Further Development

Part 2 Dose Expansion

- Pivotal single arm Phase 2 study in R/R MM, monotherapy in 4th line

Other Studies

- Phase 2 combination studies
- Phase 2 in other CD38-expressing hematologic malignancies

Phase 3 Confirmatory Studies

Primary endpoint: ORR

ISB 1342-101 Clinical Trial

- First-in-human, open-label study, including a dose escalation part (Part 1), and a disease-specific expansion part (Part 2) in subjects with relapsed and/or refractory multiple myeloma (RRMM) who received prior proteasome inhibitors (PIs), immunomodulators (IMiDs) and CD38.

- Primary objective to assess the safety profile of ISB-1342 and determine the maximal tolerated dose (MTD) and/or recommended phase 2 dose (RP2D).

- Data from earlier cohorts were previously reported at the ASH Annual Meeting 2022 (Blood (2022) 140 (Supplement 1): 7264–7266).

- Here we report findings from the Q1W dose-escalation portion from Cohorts 108, 109 and 110 (4, 8 and 16 µg/kg, respectively, as a full dose on Day 8, preceded by a priming dose on Day 1 for each dose level).

Data extraction was performed on 10/23/23 (ongoing database)

Adverse Events of Special Interest

- CRS occurred in 16/28 (57%) subjects; all were low grade (25% Grade 1, 28% Grade 2, no Grade 3 or higher). CRS generally started between days 1-2, resolved within 4.75 days. Five of 16 subjects had more then one episode of CRS. Tocilizumab was used in 8/28 (28.6%) subjects.

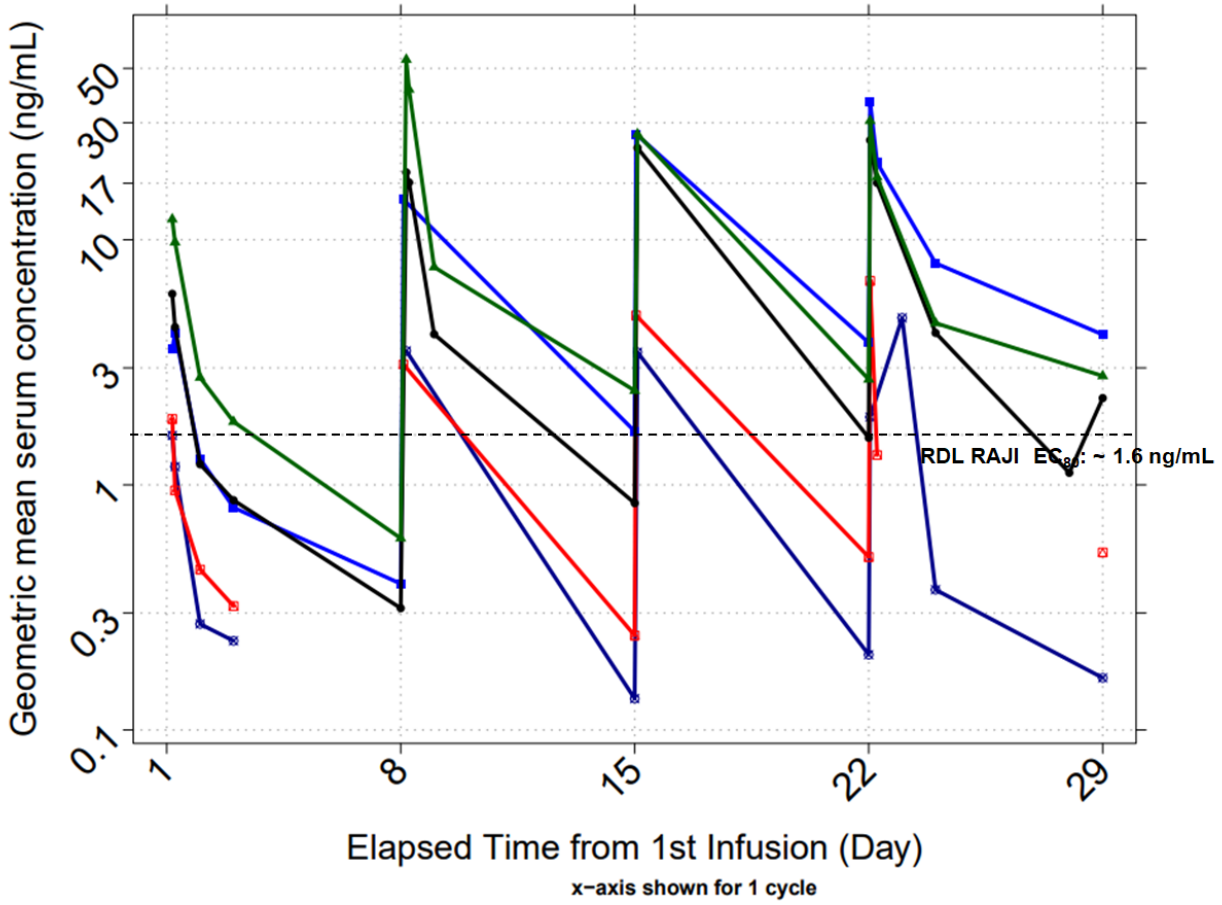
- Injection site reactions were observed in 7/8 (87%) subjects with subcutaneous (SC) administration; all were grade 1 or 2. Two subjects had skin biopsies showing neutrophilic dermatoses.

- 2/28 had related infection (one grade 2 herpes virus reactivation, one grade 4 sepsis, both resolved).

- Grade 3-4 neutropenia was observed in 11/28 (39%) of patients in Cohorts 109 and 110 IV (expected range for TCE). Neutropenia risk did not translate into the infectious risk.

- No immune effector cell associated neurotoxicity syndrome (ICANS) observed.

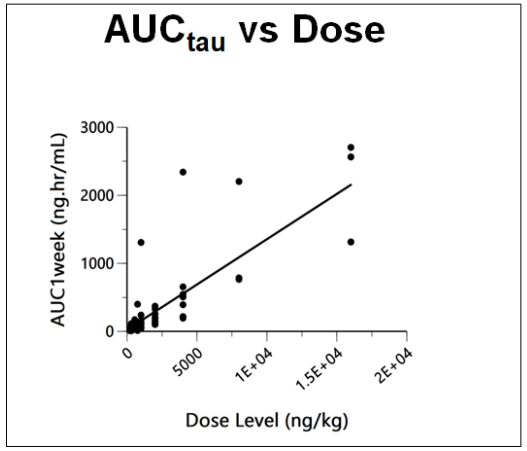
PK Profile with Q1W IV Dosing Regimen

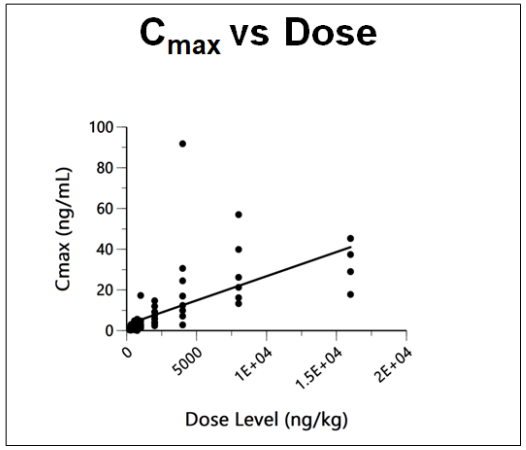


In general, the C_{max} was achieved at the end of the infusion followed by a bi-exponential decline of serum concentrations

- The Q1W PK profile support weekly dosing regimen, maintained higher C_{trough} with lower fluctuations relative to Q2W regimen
- Limited data from SC injection suggested slow absorption and lower C_{max} than IV

Dose Linear Increase in Exposures





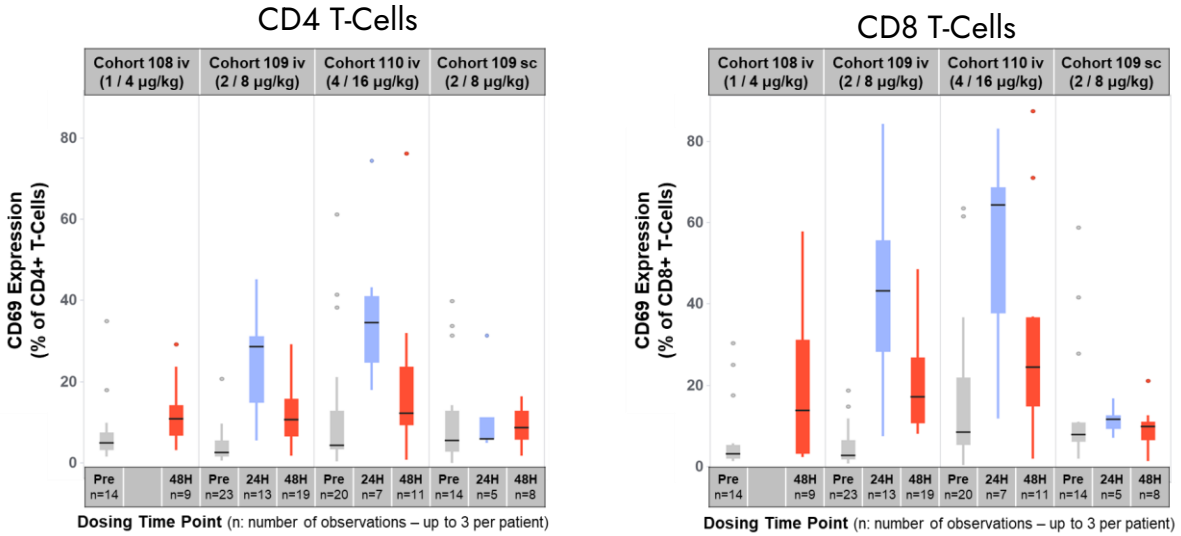
ISB 1342 Q1W Summary Immunogenicity Profile

ISB 1342-101	Total Subjects (Evaluable)	Treatment Emergent ADA	Treatment Boosted ADA	ADA Titers	ADA Positive at Baseline only	Neutralizing Ab	Persistent
Q1W	48	12/48 (25%)	1/48 (2.1%)	10-655360	None	13/48 (27.1%)	12/48 (25%)

- In the Q1W regimen, 12 out of 48 (25%) evaluable subjects tested positive for ADA with titers ranging from 10 to 655360
- ADA were neutralizing in nature in all 12 subjects
- High ADA titers appeared to influence the serum exposures

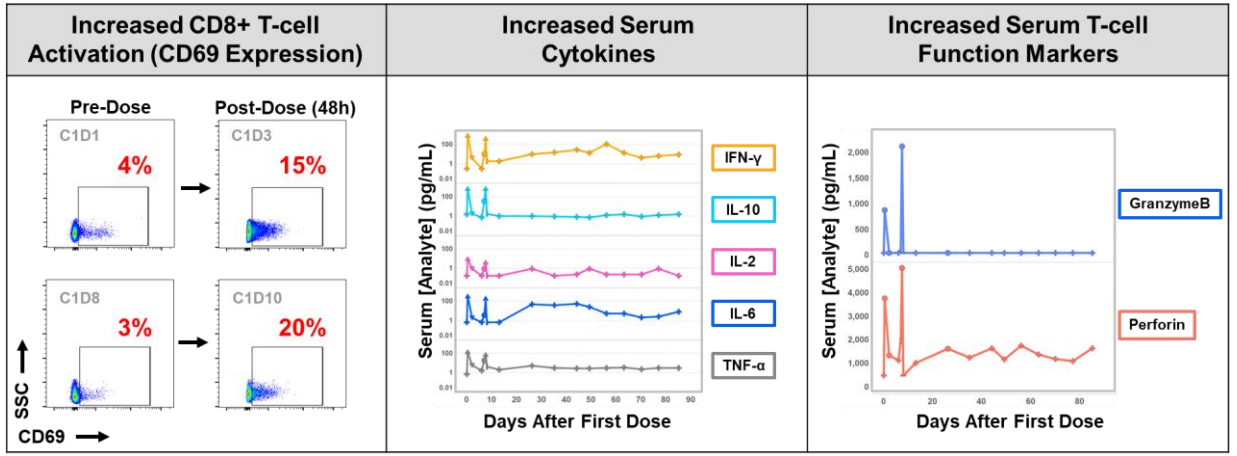
Biomarker Profile

T-Cell Activation Measured by Expression of CD69 in T-cells Following Treatment with ISB 1342



- T-cell expression of CD69 was assessed by flow cytometry of peripheral blood samples collected during Cycle 1 from patients in Cohorts 108-110 (target dose levels 4-16 µmg/kg).
- Comparison of CD69 expression frequencies indicate increased T-cell activation levels at 24-48h post-dose compared to the pre-dose samples.
- Increased levels of T-cell activation were observed at higher dose levels, and this effect was reduced in patients receiving ISB 1342 subcutaneously compared to patients treated intravenously.

Biomarker Changes Associated with Response in a Patient Treated with ISB 1342 (Cohort 109 IV)



- Transient increases in several T-cell activation-related biomarkers were consistently observed following ISB 1342 dosing at target dose levels 4-16 µg/kg.
- Increased expression of CD69 on T-cells was determined by flow cytometry analysis of peripheral blood (left panel); circulating cytokine levels (center panel, assessed by Meso Scale Discovery /MSD immunoassay) and T-cell functional markers, granzyme B and perforin (right panel, assessed by ELISA) were measured in serum, indicating transient increases associated with T-cell activation.
- Pre and post dosing samples were drawn at various time points within 48h following the first 2 doses. All other time points shown were collected prior to dosing.

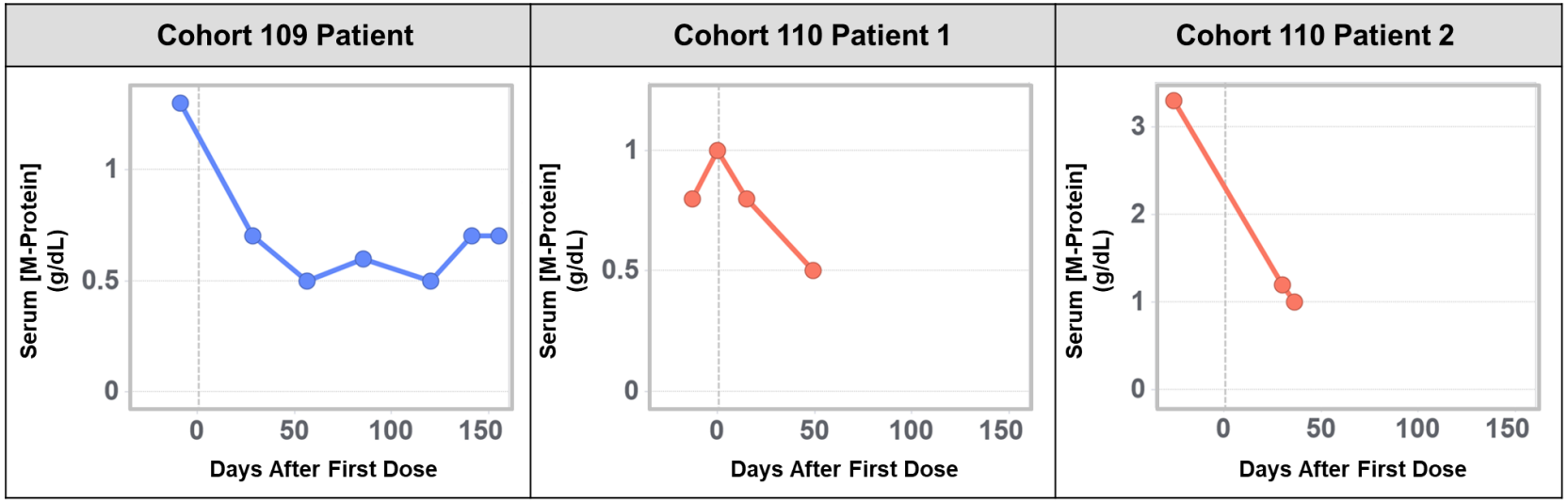
Clinical Responses

Best Overall Response	*Cohort 108 IV 4µg/kg (N=4)*	*Cohort 109 IV 8µg/kg (N=10)*	*Cohort 109 SC 8µg/kg (N=8)*	*Cohort 110 IV 16µg/kg (N=6)*
PR	0	1 (10%)	0	2 (33.3%)*
MR	0	0	0	1 (16.7%)
SD	2 (50%)	3 (30%)	3 (37.5%)	1 (16.7%)
PD	2 (50%)	5 (50%)	2 (25%)	2 (33.3%)
NE	0	1 (10%)	1 (12.5%)	0
Missing	0	0	2 (25%)	0

^Data extract update from 5th December, 2023
**1 PR assessment based on central laboratory data*
NE: not evaluated

Clinical Responses

Reduced Serum M-Protein Levels in Three Patients Treated with ISB 1342 (IV Cohorts)



Patients by Cohort	Anti- CD38	Last dose of anti- CD38	ISB 1342 C1D1
Cohort 110 Patient 2	Isatuximab	21-Jan-2022	10-Oct-2023
Cohort 109 patient	Daratumumab	04-Jan-2023	08-Mar-2023
Cohort 110 Patient 1	Daratumumab	04-May-2023	03-Jul-2023

- The efficacy signals observed in Cohorts 108 -110 are consistent with the lower end of the predicted efficacy dose range (5.0 to 50 ug/kg) by quantitative systems pharmacology (QSP) based on preclinical data.

Conclusions of the phase I dose escalation study

- Treatment with ISB 1342 was well tolerated at the evaluated weekly 4, 8 and 16 µg/kg dose levels.
- The observed CRS events were mild to moderate and manageable with supportive care. No new safety signal identified.
- Serum M-protein decreased indicating PR has been seen in 3 subjects (based on ongoing dataset analysis).
- Further dose-escalation (to 32 and 64 µg/kg) is warranted based on manageable safety profile, anti-myeloma activity observed, and supported by PK profile as well as T-cell activation biomarkers with current dose of 16 µg/kg IV.
- Trials have paused enrolment due to strategic prioritisation. Program is open for partnership for further development.

ISB 1442

ISB 1442 - Executive Summary

- Phase I, first-in-class, bispecific and biparatopic CD38xCD47 antibody developed in RRMM and AML

- Novel approach for the treatment of CD38+ tumors by co-targeting CD38 and CD47 with a 2+1 biparatopic bispecific antibody, showing:
  + Triple Mechanism of Tumor Killing: ADCP, ADCC, CDC
  + Higher cytotoxic potency relative to daratumumab in $CD38^{high/low}$ *in vitro* tumor models
  + High cytotoxic potency in AML *ex vivo*

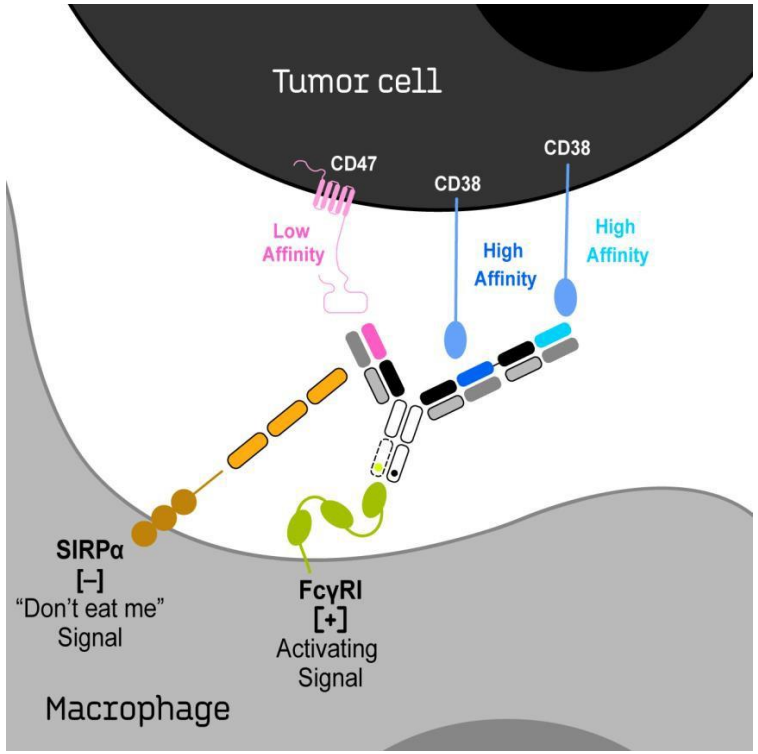
- Barely detectable on-target off-tumor binding compared to anti-CD47 mAb (5F9) as per molecular design

- Phase 1 dose-finding study of ISB 1442 in RRMM opened in the U.S., Australia and India. Favorable safety profile shown as of April data cut-off and clinical proof-of-mechanism demonstrated in patients

- Phase 1 dose-finding study in relapsed/refractory AML planned in 2024

ISB 1442 Triple Mechanism of Tumor-Cell Killing Driven by Enhanced ADCP, ADCC and CDC

ISB 1442 (CD38 x CD47)
biparatopic bispecific antibody



KEY ATTRIBUTES

- Dual binding to CD38 and CD47 epitopes, increasing avidity relative to daratumumab
- Two Fab regions drive binding to distinct CD38 epitopes that don't compete functionally with daratumumab
- One arm blocks CD47-SIRPa binding on tumor cells to enhance ADCP
  - + CD47 is over-expressed by hematologic tumors and associated with worse prognosis
  - + Enhanced phagocytosis by blocking CD47 and increasing activation signaling through FcγR binding
  - + Reduced potential for antigen sink with lower-affinity Fab binding to CD47 expressed on healthy cells
- Potent ADCC, CDC and ADCP based on optimized affinity, architecture/avidity and enhanced Fc function
- Optimized tolerability with low potential for adverse effects on red blood cells such as hemagglutination, platelet aggregation
- ISB 1442 was granted Orphan Drug Designation for Multiple Myeloma by the U.S. FDA

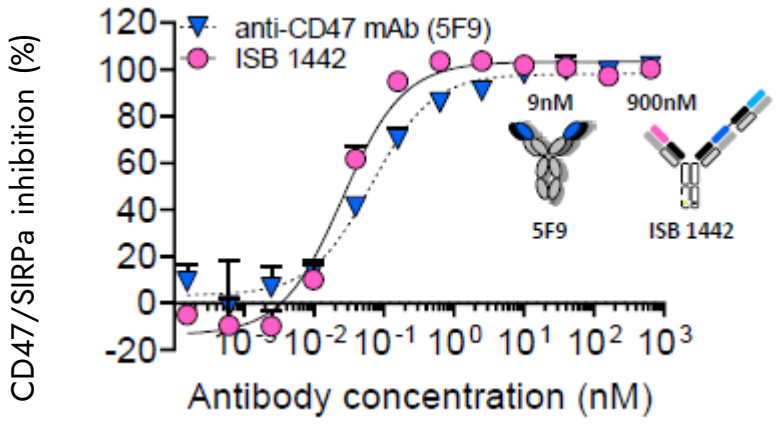
ADCP: Antibody-Dependent Cellular Phagocytosis
ADCC: Antibody-Dependent Cell Cytotoxicity
CDC: Complement-Dependent Cytotoxicity

ISB 1442 Blocks CD47/SIRPα Interactions & Induces Enhanced Phagocytosis of CD38 Low-Expressing Tumor Cells vs. Daratumumab

Blocks CD47/SIRPα Interactions

CD38^{high}(Daudi)

CD38	CD47
180,000	60,000



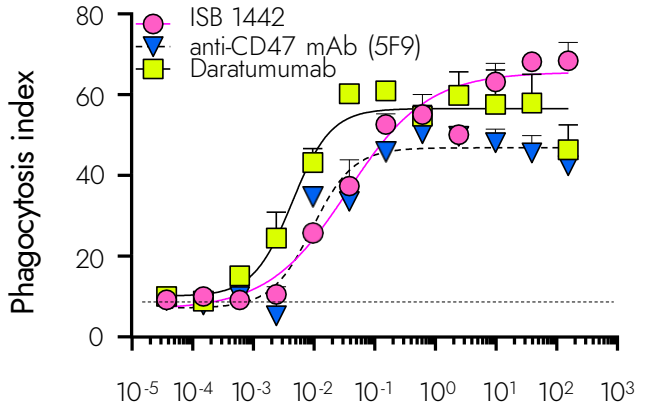
Higher Phagocytosis Relative to Daratumumab in CD38^{low}

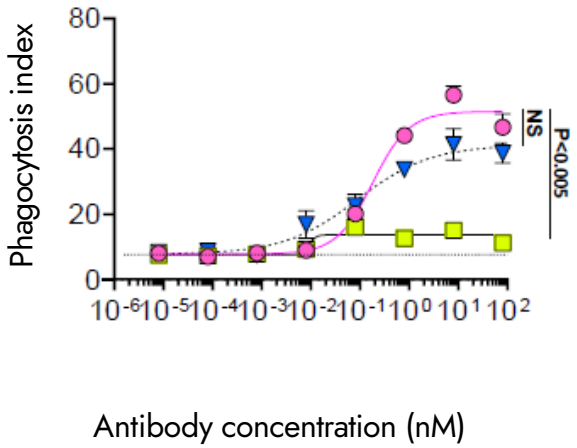
CD38^{high}(Daudi)

CD38	CD47
180,000	60,000

CD38^{low}(KMS-12-BM)

CD38	CD47
5,000	210,000





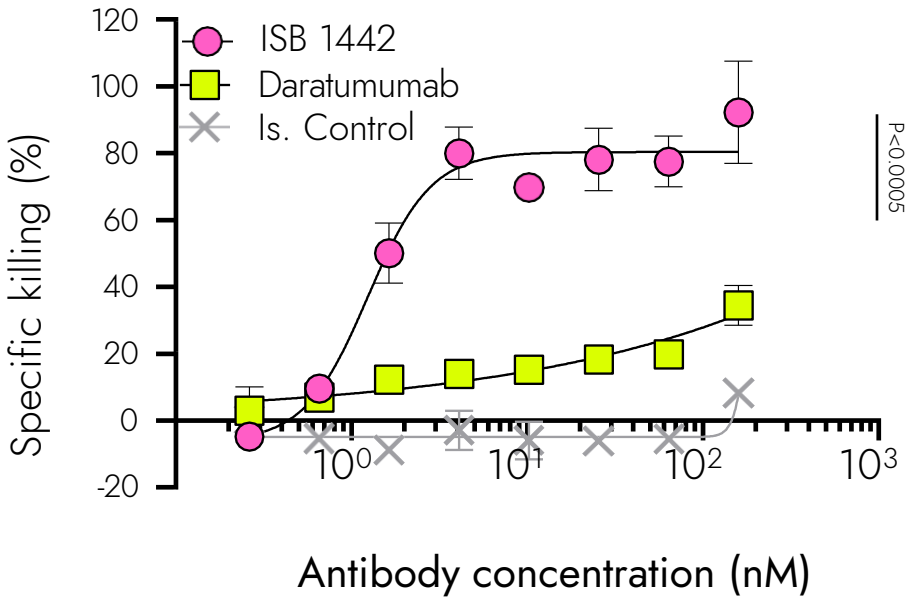
Statistics: Tukey's multiple comparison test.

ISB 1442 Shows Higher Killing Potency Across a Broad Range of CD38 Expression Levels Relative to Daratumumab

Higher Complement Dependent Cell Cytotoxicity (CDC) Than Daratumumab

CD38^{high}(Daudi)

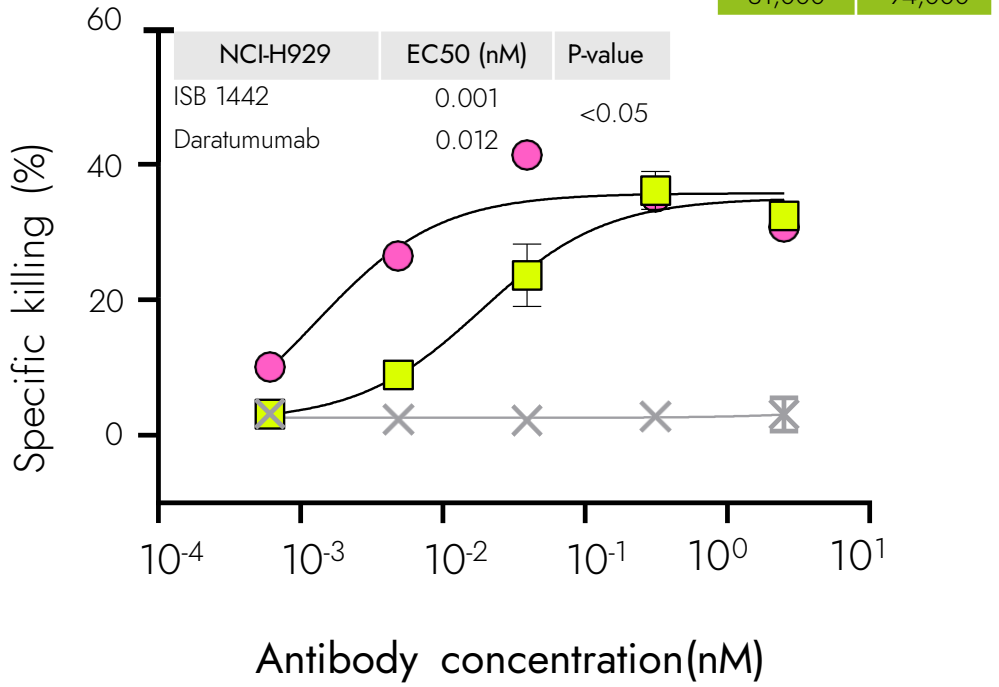
CD38	CD47
180,000	60,000



Higher Potency Than Daratumumab in CD38^{low} Antibody Dependent Cell Cytotoxicity (ADCC)

CD38$^{low\text{-}int}$ (NCI-H929)

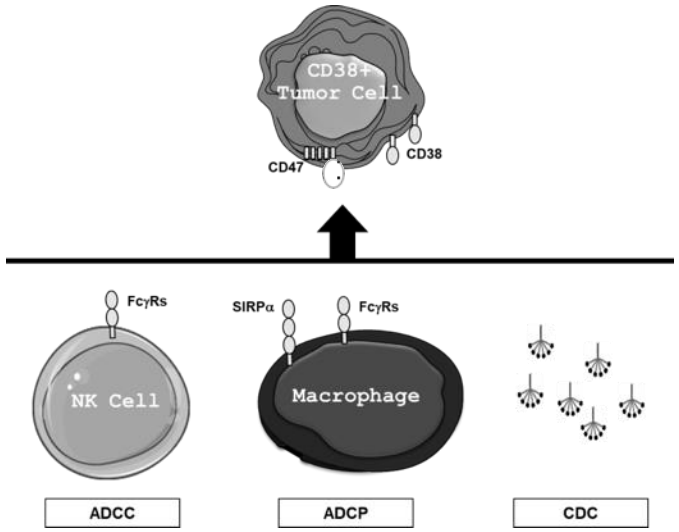
CD38	CD47
31,000	94,000



Statistics: Tukey's multiple comparison test.
EC50: Half Maximal Effective Concentration

ISB 1442 Shows Superior Tumor Cell Killing Compared to 5F9 and Daratumumab, as Monotherapies or in Combination

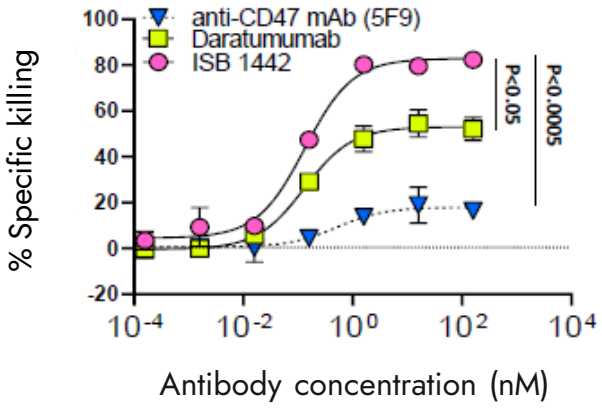
Multiple Mode of Action of Killing (MMoAK)



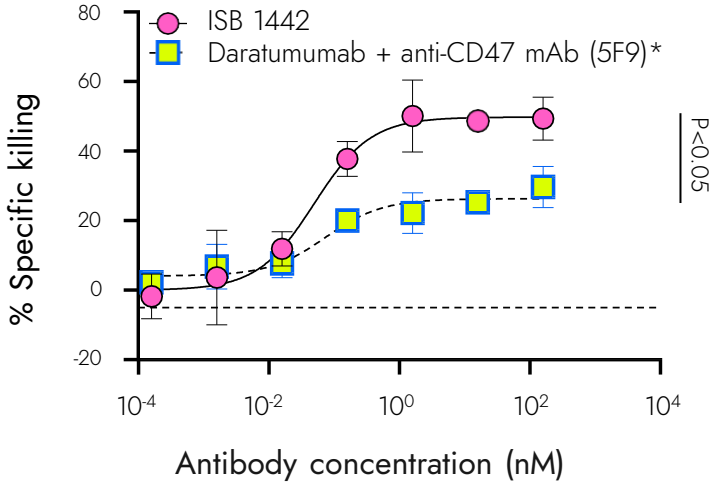
CD38$^{low\text{-}int}$ (NCI-H929)

CD38	CD47
31,000	94,000

ISB 1442 Compared to Anti-CD47 (5F9) and Daratumumab Monotherapy



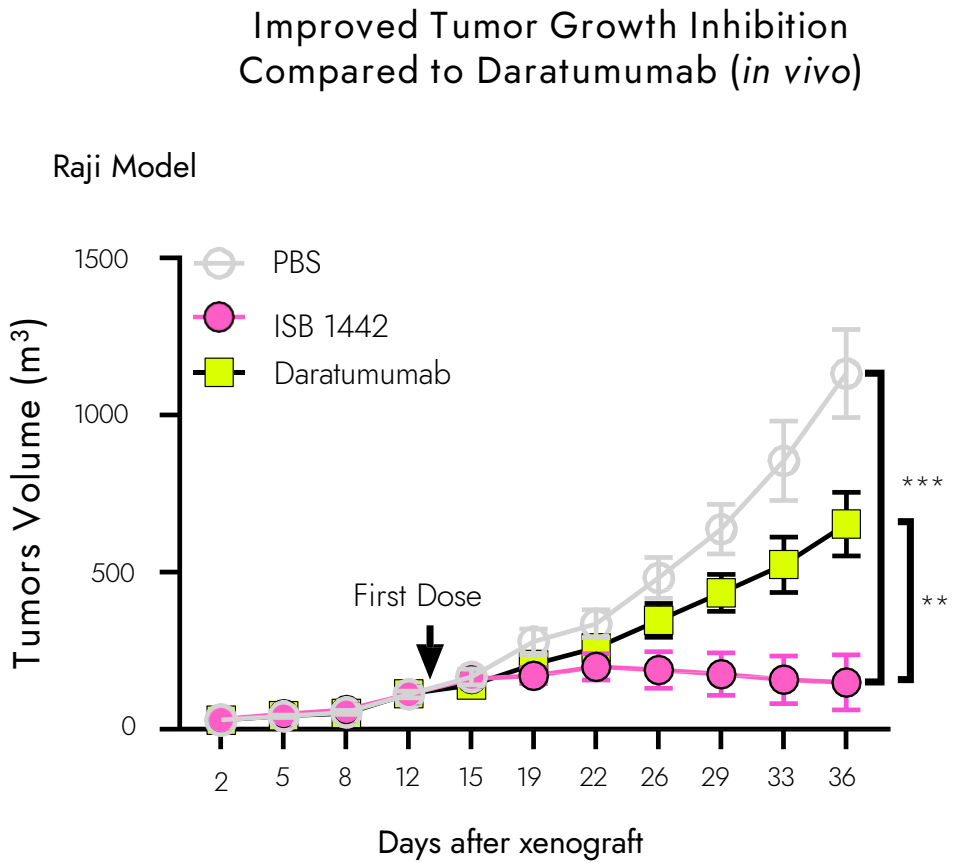
ISB 1442 Compared to Combination of Anti-CD47 (5F9) and Daratumumab



- MMoAK assay shown above is run in the presence of irrelevant competing IgGs from serum, more closely representing the environment in patient serum
- The enhanced Fc in ISB 1442 activates macrophages regardless of the presence of competing IgGs, resulting in greater monotherapy efficacy than anti-CD47 (5F9)

Statistics: Tukey's multiple comparison test.

ISB 1442 Shows Higher Tumor Growth Inhibition *In Vivo vs.* Daratumumab

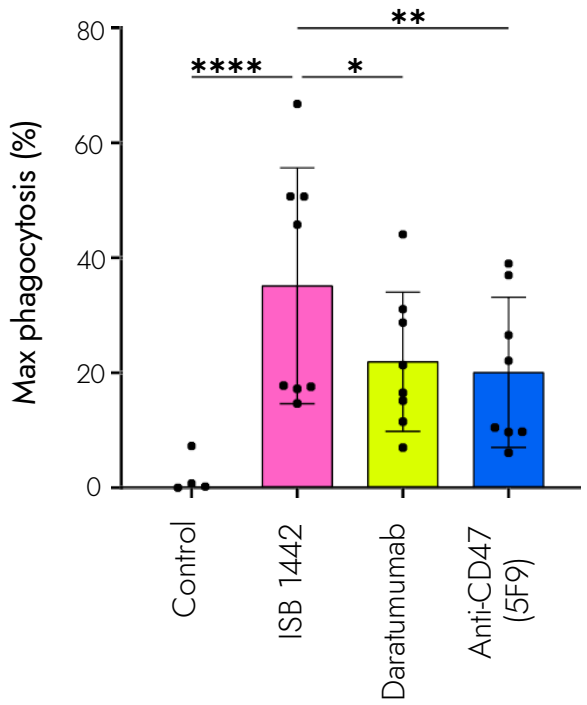


- 10 million Raji cells were implanted subcutaneously into CB17/SCID mice
- Animals were randomized when tumor volume reached ~100 mm^3
- Doses: ISB 1442 and 5F9 (anti CD47) dosed IV QW at 10 mg/kg, daratumumab dosed IV BIW at 16 mg/kg

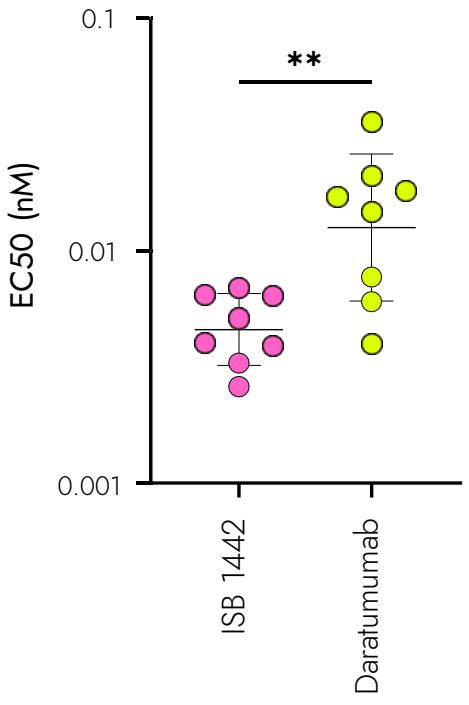
*Statistics: 1-way ANOVA w. Tukey post hoc testing ** p<0.01; *** p<0.001*

ISB 1442 Shows Higher Cytotoxic Potency Against AML Cell Lines Compared to Anti-CD38 and Anti-CD47 Mono-Targeting Antibodies

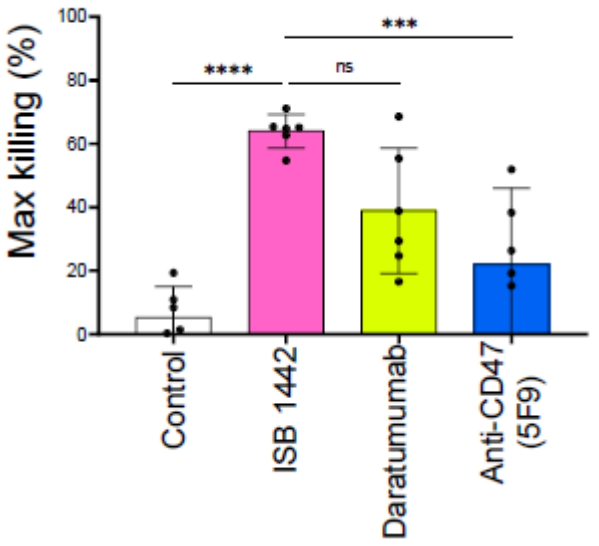
Antibody Dependent Cell Phagocytosis (ADCP)



Antibody Dependent Cell Cytotoxicity (ADCC)



Multiple Mechanisms Of Action Of Killing (MMoAK)

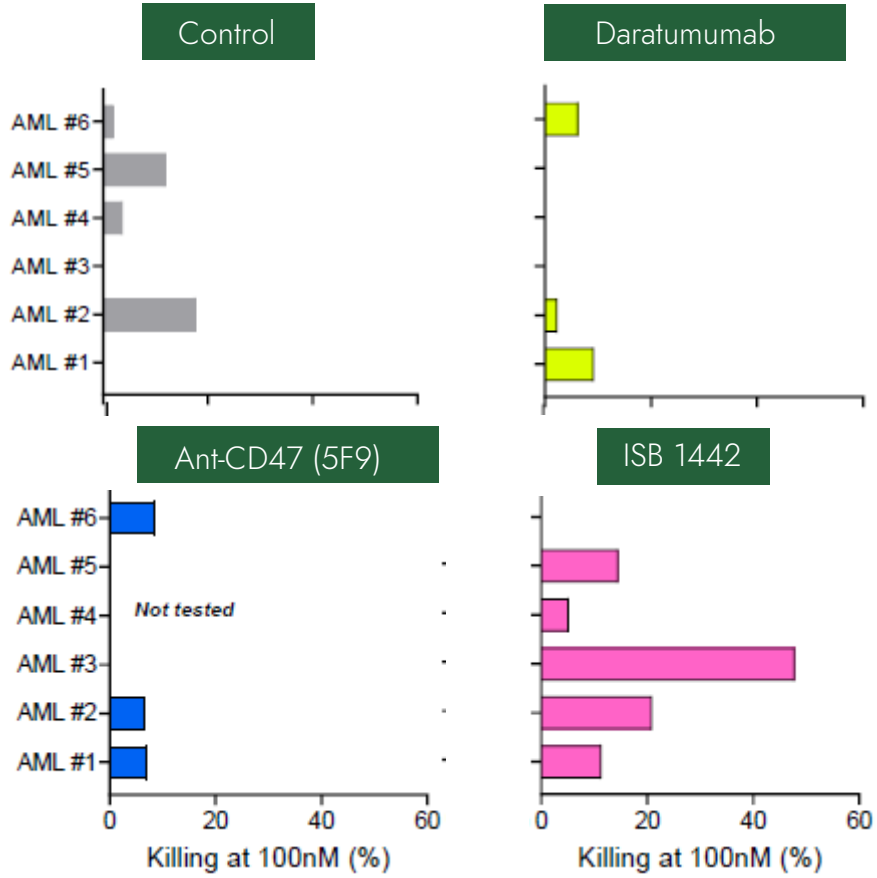


P < 0.05
** P < 0.01
*** P < 0.001
**** P < 0.0001

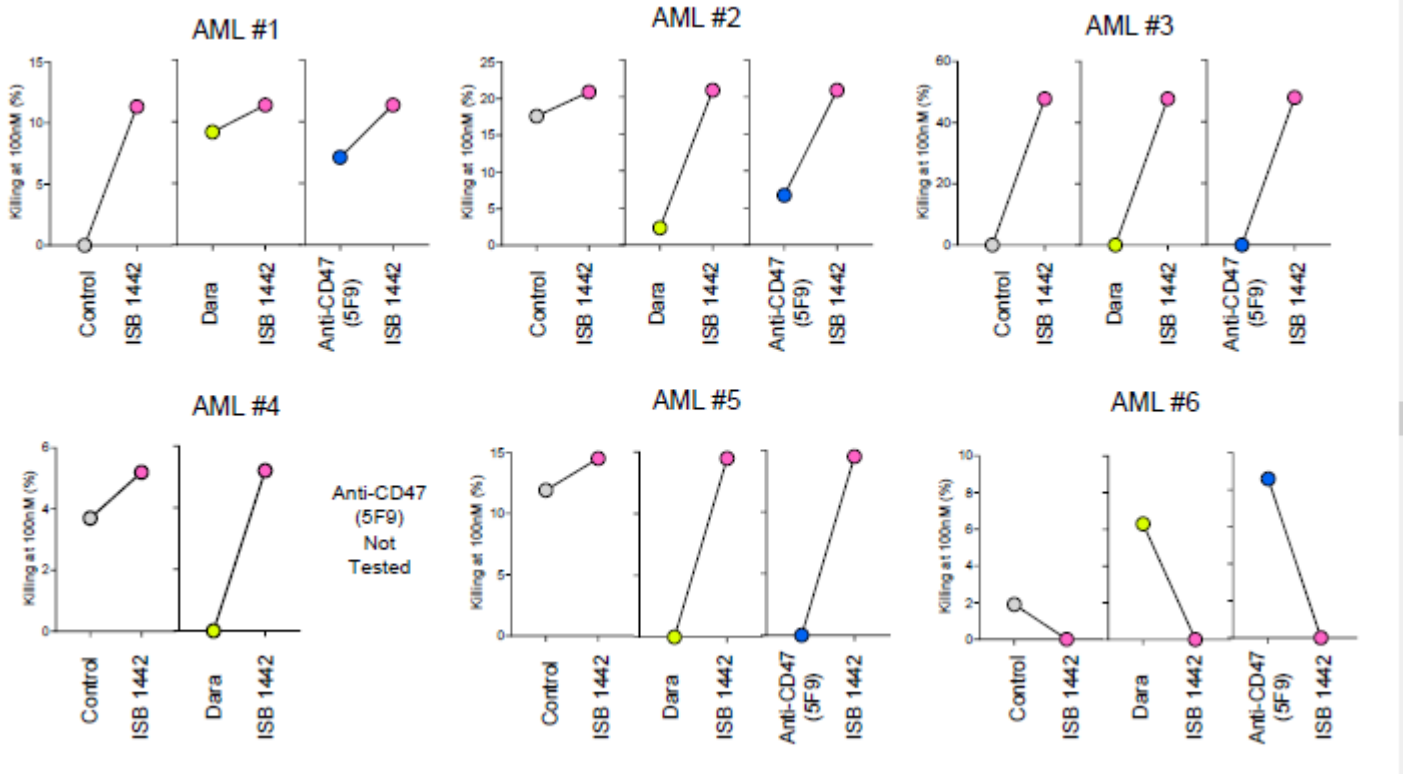
Statistics: Not Significant (NS) AML: Acute Myeloid Leukemia
Cell used: MOLM-13 ($CD38^{int}$ $CD47^{low}$)
Presented at American Society of Hematology 2022 Annual Meeting

ISB 1442 Showed More Frequent Killing of AML Blasts and a Trend Towards Higher Cytotoxic Potency as Compared to Monospecific Anti-CD38 Daratumumab or Anti-CD47 (5F9) Ex Vivo

Killing of AML Blasts from Patients' Bone Marrow Aspirates



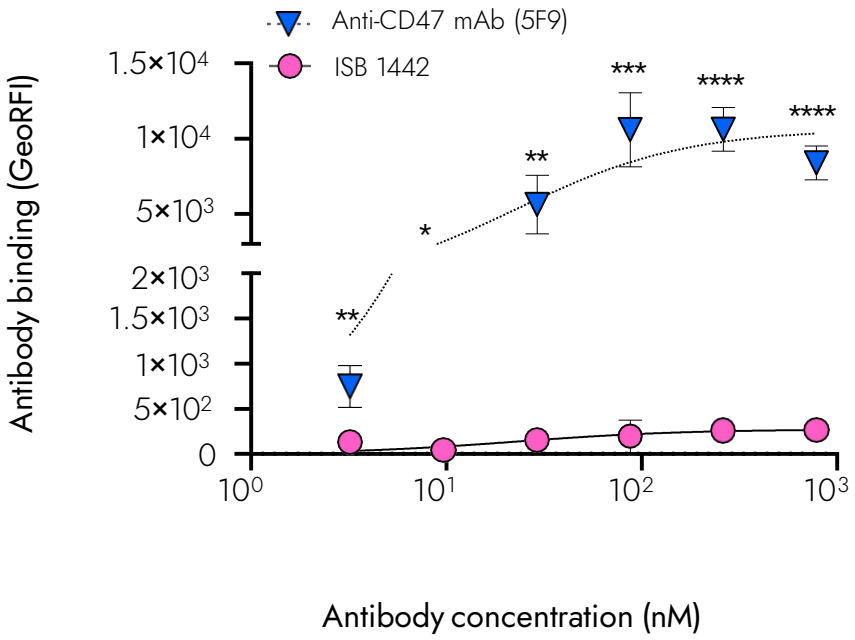
Potency Compared to Anti-CD38 or Anti-CD47 Monospecifics



DARA: Daratumumab
Presented at American Society of Hematology 2022 Annual Meeting

ISB 1442 Only Binds to CD47 After Engaging CD38, Reducing On- Target, Off-Tumor Depletion of Red Blood Cells

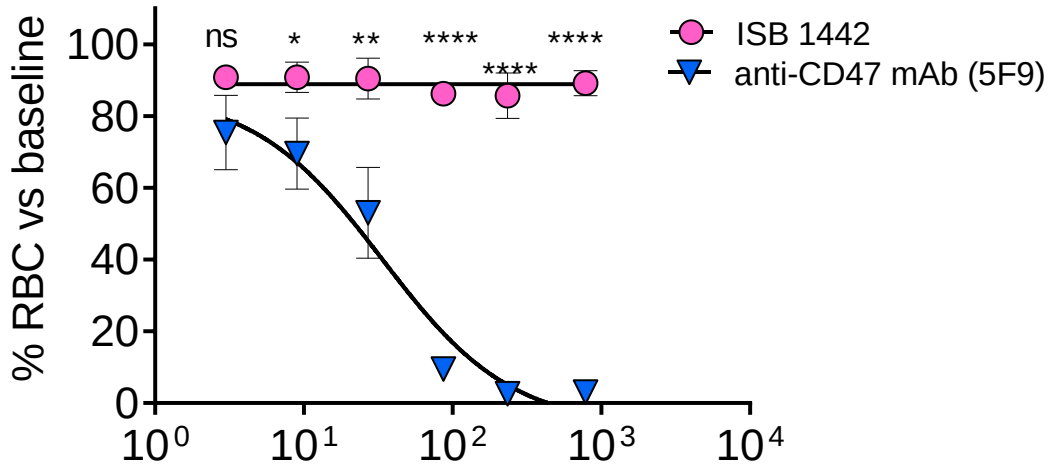
Reduced Binding to RBC



Antibody concentration (nM)

Statistics: 2-way ANOVA with multiple comparisons

Minimal RBC Depletion



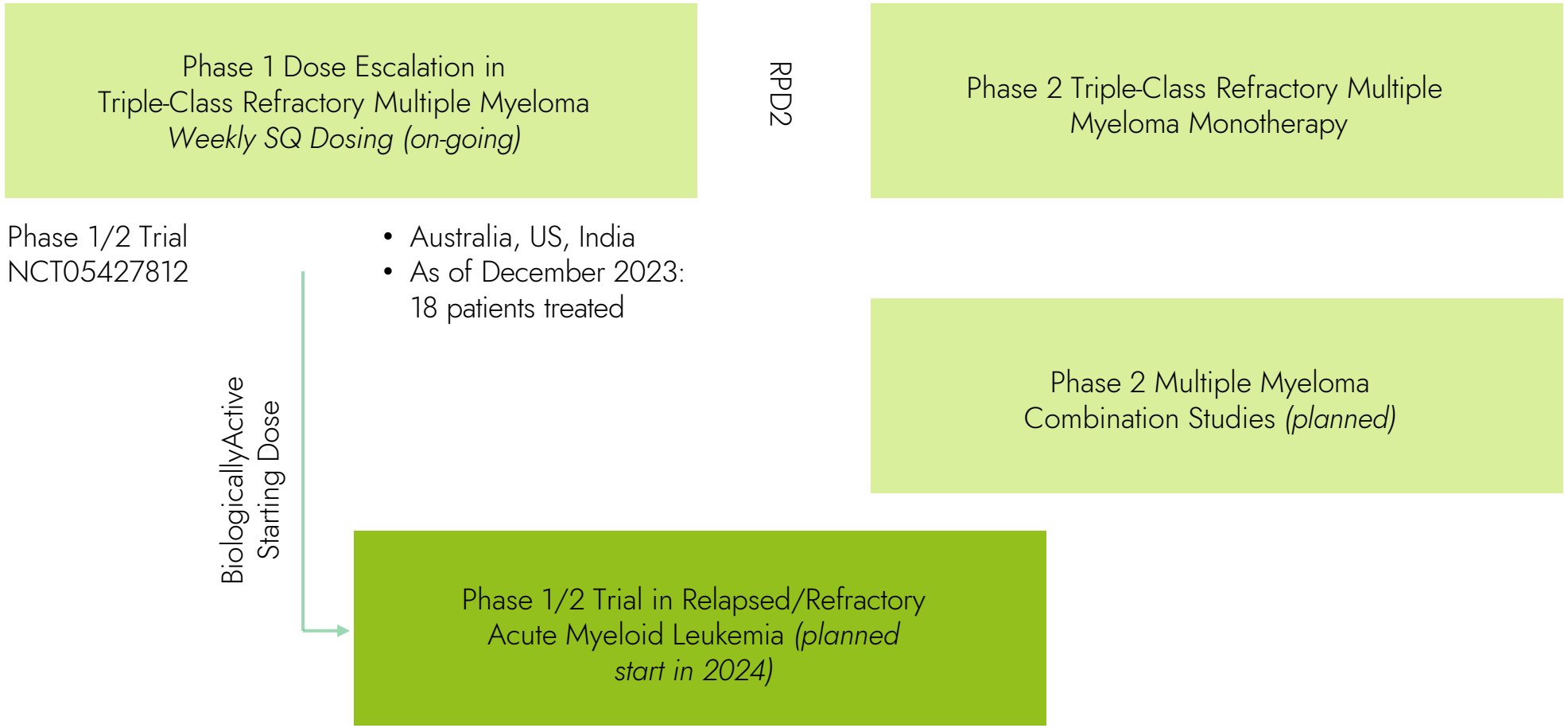
Antibody concentration (nM)

Statistics: 2-way ANOVA with multiple comparisons

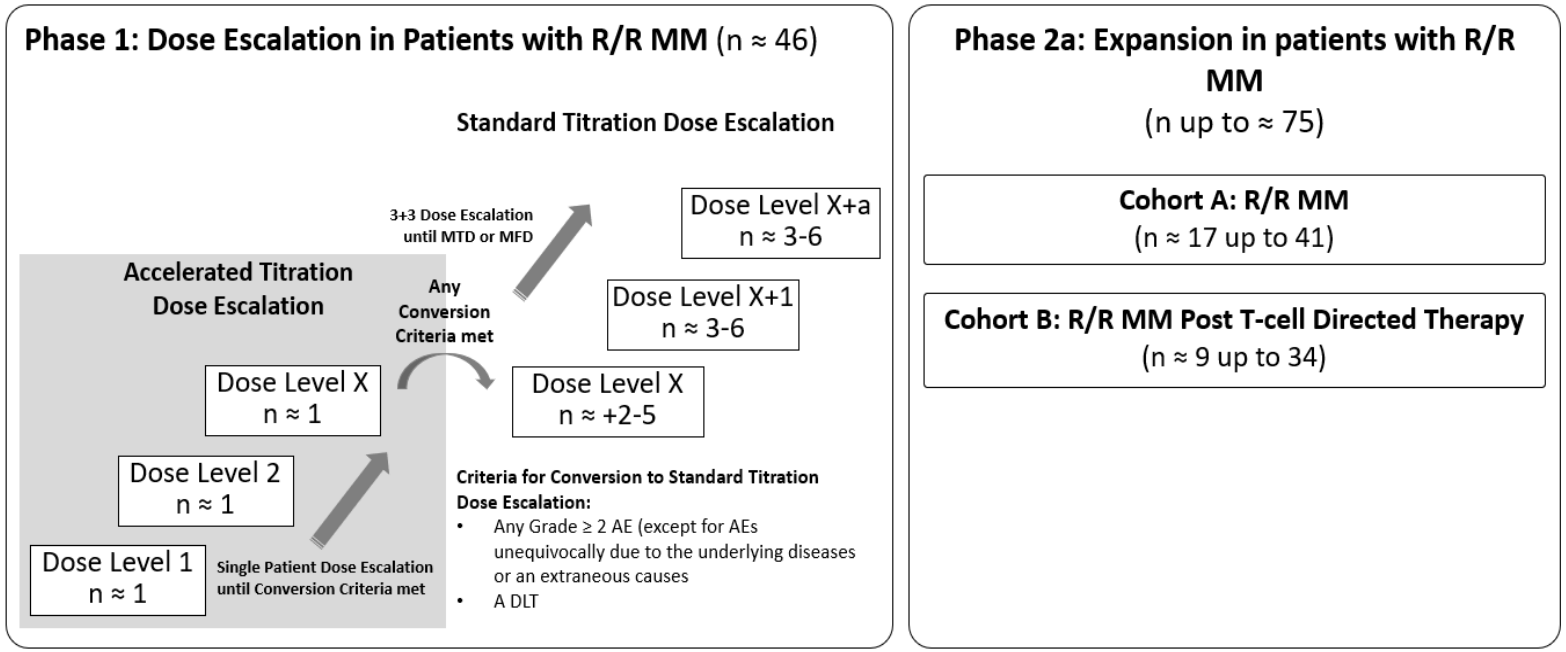
*ns p≥ 0.05; * p<0.05; ** p<0.01; *** p<0.001; **** p<0.0001*

Statistics: Not Significant (NS) AML: Acute Myeloid Leukemia
Cell used: MOLM-13 ($CD38^{int}$ $CD47^{low}$)
Presented at American Society of Hematology 2022 Annual Meeting

ISB 1442 Clinical Development



ISB 1442-101 Clinical Trial Design



- a = the numerical value that leads to the next higher dose level in the standard titration design. n = number of subjects.. X = The dose level in the accelerated titration design at which the conversion criteria is met. DLT = dose-limiting toxicity; MFD = maximum feasible dose; MM = multiple myeloma; MTD = maximum tolerated dose; RP2D = recommended phase 2 dose; R/R = relapsed or refractory.
- *According to Project Optimus, 2 putative recommended phase 2 doses will be tested. Dose levels to be determined.

Key Patient Eligibility Criteria:

- R/R MM with measurable disease after a CD38 antibody, IMiDs, PIs, and who must not be candidates for regimens known to provide clinical benefit
- Failed 3 or more prior lines of therapies

Primary Objectives:

- Assess safety, tolerability
- Determine MTD/RP2D

Secondary Objectives:

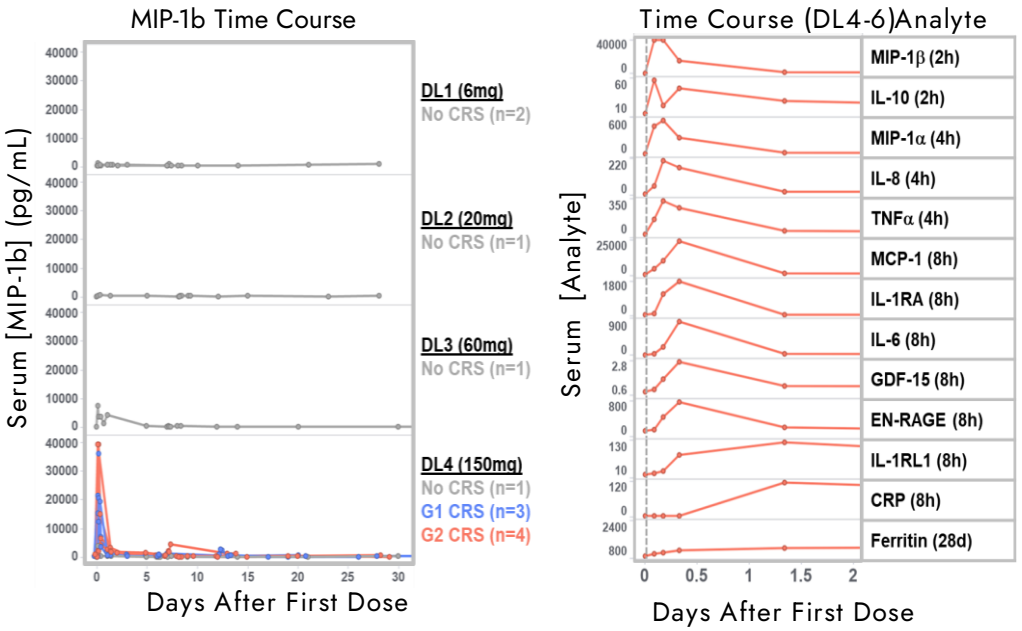
- PK, immunogenicity
- Assess preliminary clinical activity of ISB 1442

Exploratory Objectives:

- Assess biomarkers and their correlation with clinical activity, safety, and other clinical endpoints of interest
- Assess minimal residual disease (MRD) when indicated

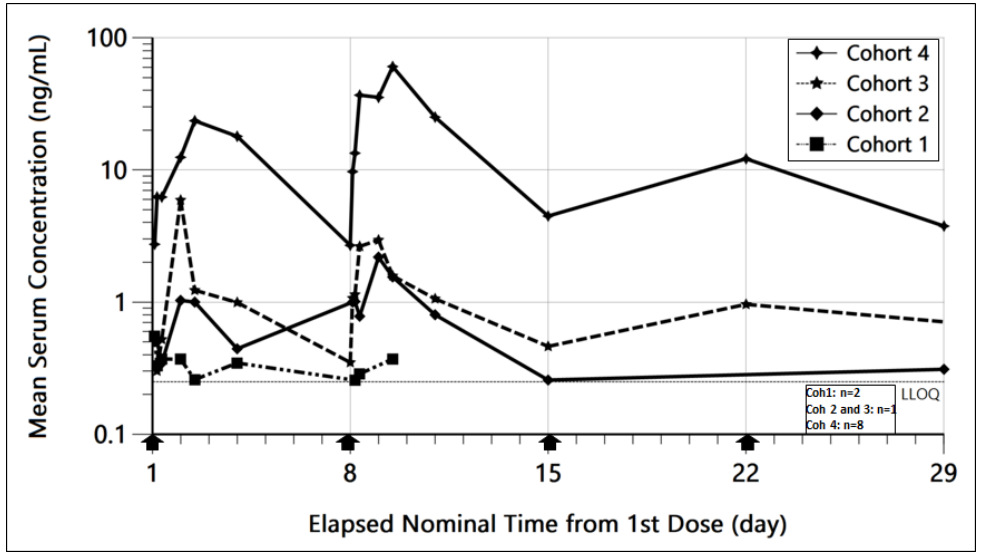
Biomarkers and Pharmacokinetics

Dose-Dependence and Time Course of Soluble Biomarker Changes Following Treatment with ISB 1442



- Transient increases in soluble biomarkers were predominantly observed following the first dose of ISB 1442, with greater increases at dose level 4 (150 mg) compared to lower dose levels, and in patients exhibiting CRS-like symptoms (left panel).
- The time course of biomarker changes during the first 48h after dosing is shown for one patient (DL4-ptatient 6), demonstrating the different timing of peak levels for several serum biomarkers after dosing within this patient (right panel).

Pharmacokinetics



- Mean ISB 1442 serum concentration versus time profile from Cohort 1 (6 mg SC), Cohort 2 (20 mg SC), Cohort 3 (60 mg SC) and Cohort 4 (150 mg SC) are shown.
- Rich PK samples collected after C1D1 and C1D8 doses, followed by pre-dose samples for the remaining dose occasions.
- Super-Proportional Increases in ISB 1442 Serum exposures (Cmax and AUC) most evident between cohorts 3 to 4.
- Slow absorption of ISB 1442 after SC injection with Tmax achieved mostly on the 2nd day of dosing.

Clinical Responses as of December 2023

Best Overall Response	DL1(6mg) N=2	DL2(20mg) N=1	DL3(60mg) N=5	DL4(150mg) N=9	DL5(300mg) N=1
Stable disease (SD)	0	0	1 (33%)	3 (33%)	0
Progressive Disease (PD)	1 (50%)	1 (100%)	1 (33%)	4 (44%)	0
Not Evaluable (NE)	1 (50%)	0	0	0	0
Missing	0	0	1 (33%)	2 (22%)	1 (100%)

- Based on in-vitro modeling, current DL4 mean ISB 1442 concentration is in the EC50 range. EC90 range could potentially be achieved with DL 6 to 7 dose (450-600mg).

Data extract update from 6th December, 2023

Preliminary Results of the Ongoing Phase I Study

- Overall, treatment with ISB 1442 is well tolerated.
- CRS events observed at DL4 (150mg) were of low grade (1 or 2) and mostly resolved within one day.
- No risk of neurotoxicity, hemolytic anemia or infections have been observed to date.
- Soluble biomarker changes, with increased macrophage-related markers, are among the first changes observed supporting proof of mechanism.
- Dose escalation is ongoing with participants enrolling in DL5 (300 mg).
- Phase 1 dose-finding study in relapsed/refractory AML planned in late 2024/early 2025.

ISB 2001

ISB 2001 - Executive Summary

- Phase I, first-in-class trispecific BCMAxCD38xCD3 antibody, developed in RRMM

- Pre-clinical data show potential for ISB 2001 to induce enhanced cytotoxicity relative to clinical benchmarks against MM expressing variable levels of BCMA and CD38, mimicking natural tumor heterogeneity.
  - + Increased killing of tumor cells compared to teclistamab and alnuctamab
  - + Higher potency when compared to the combination of daratumumab and teclistamab
  - + Superior cytotoxicity over teclistamab in ex vivo assays in patient bone marrow aspirates
  - + Superior efficacy over teclistamab in in vivo models demonstrating 100% complete response

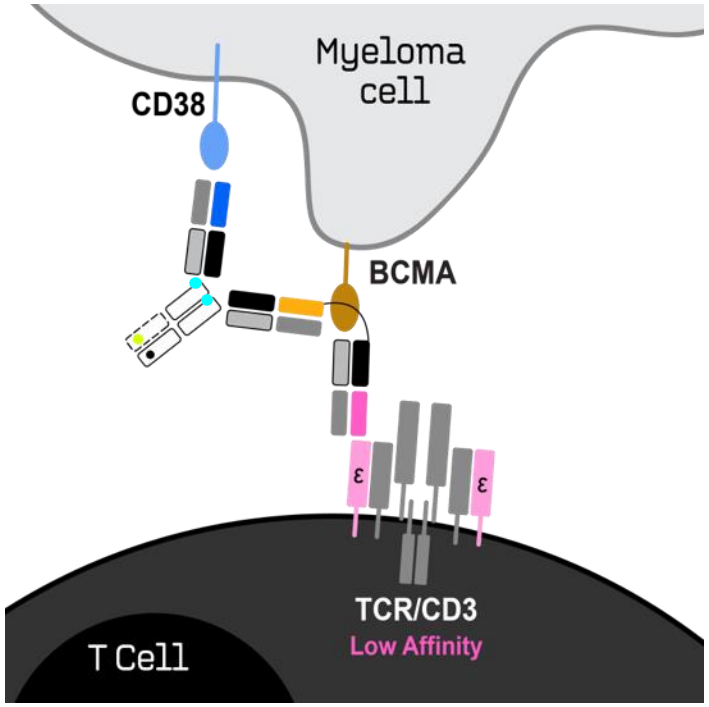
- An innovative quantitative systems pharmacology model supports an optimal first-in-human dose, eliminating use of primates and reducing patient exposure to sub-efficacious doses of therapies.

- Phase 1 first-in-human study of ISB 2001 for the treatment of relapsed/refractory multiple myeloma is currently ongoing in the US and Australia (Clinicaltrials.gov identifier: NCT05862012).

- Granted orphan drug designation (ODD) by the U.S. Food and Drug Administration (FDA)

ISB 2001 is First TREAT™ Trispecific Antibody For Relapsed/Refractory Multiple Myeloma

ISB 2001 (BCMA x CD38 x CD3) trispecific antibody

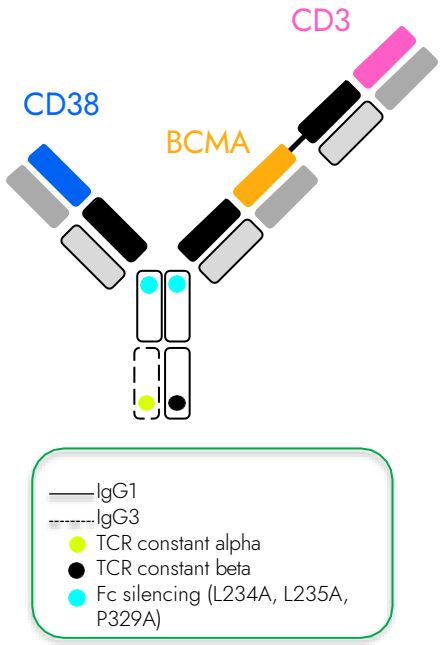


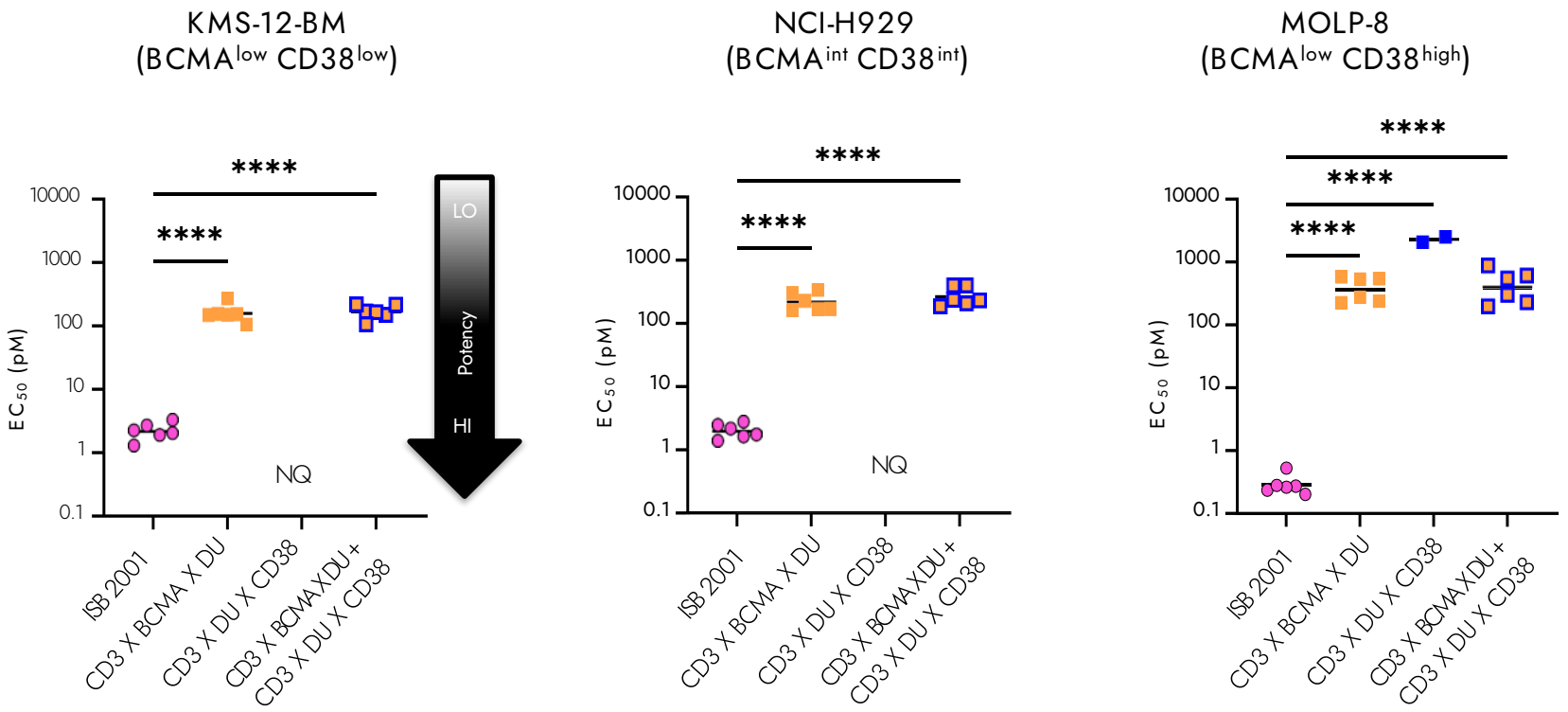
KEY ATTRIBUTES

- BCMA and CD38 are expressed on the surface of multiple myeloma cells and are clinically validated targets.
- ISB 2001 combines three proprietary Fab arms binding to CD3ε on T-cells, and to BCMA and CD38 on myeloma cells.
- In vitro studies showed increased tumor killing potency of ISB 2001 compared to all tested antibodies, including currently approved and investigational multiple myeloma therapies.
- In vivo studies in multiple myeloma models also show superior potency of ISB 2001 relative to antibodies for the treatment of multiple myeloma.
- ISB 2001 redirects CD3+ T lymphocytes to kill tumor cells expressing from low to high levels of both BCMA and CD38.
- With two different tumor-associated antigens, ISB 2001 is expected to be more resistant to antigen escape associated with treatment of MM patients.
- Ichnos received authorizations from HREC in Australia and the U.S. FDA to initiate a Phase 1 first-in-human study of ISB 2001 for the treatment of MM and was granted ODD by the U.S. FDA for the same indication.

TREAT™: Trispecific Engagement by Antibodies based on the TCR
MHC: Major histocompatibility complex, CDC: Complement-Dependent Cytotoxicity ADCC: Antibody-Dependent Cell-mediated Cytotoxicity

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





Expression sABC	CD38 mean	BCMA mean
KMS-12-BM	28,000	9,000
NCI-H929	85,000	52,000
MOLP-8	512,000	3,200

****= p <0.0001

NQ: Not Quantifiable. DU: Dummy (irrelevant binder)
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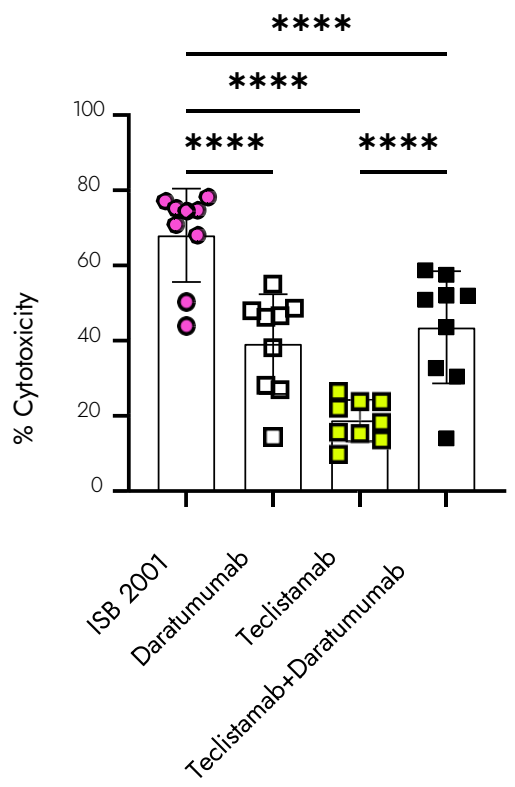
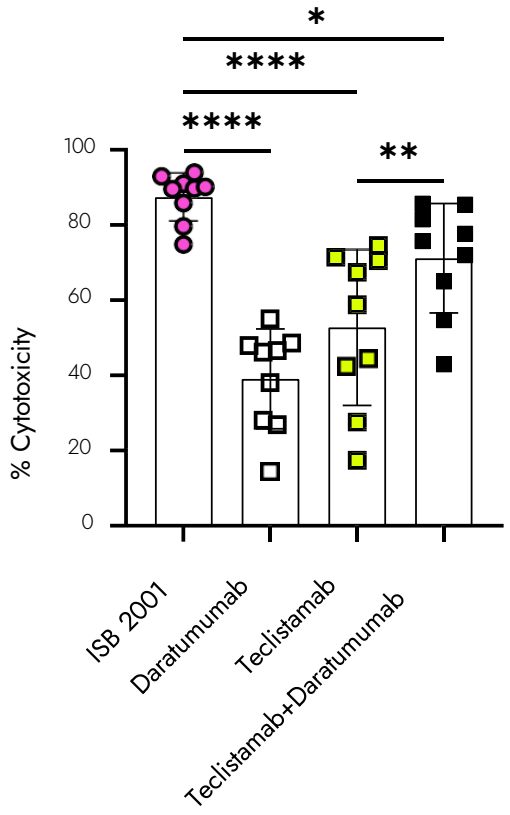
ISB 2001 Potency is Superior to the Combination of Teclistamab + Daratumumab

ISB 2001 or Teclistamab at 10 pM
Daratumumab at 100 nM



ISB 2001 or Teclistamab at 100 pM
Daratumumab at 100 nM



- Multiple Mode of Action Killing assay of tumor cell line KMS-12-BM
- The tumor cells were incubated for 48 hours with human PBMCs from healthy donors at a 5:1 effector to target ratio
- 10 pM or 100 pM of ISB2001, teclistamab in the presence or absence of 100 nM of daratumumab were added to the culture
- The percentage of cytotoxicity is displayed

*=p <0.05
**=p <0.01
****= p <0.0001

Paired one-way ANOVA followed by Tukey's multiple comparisons test
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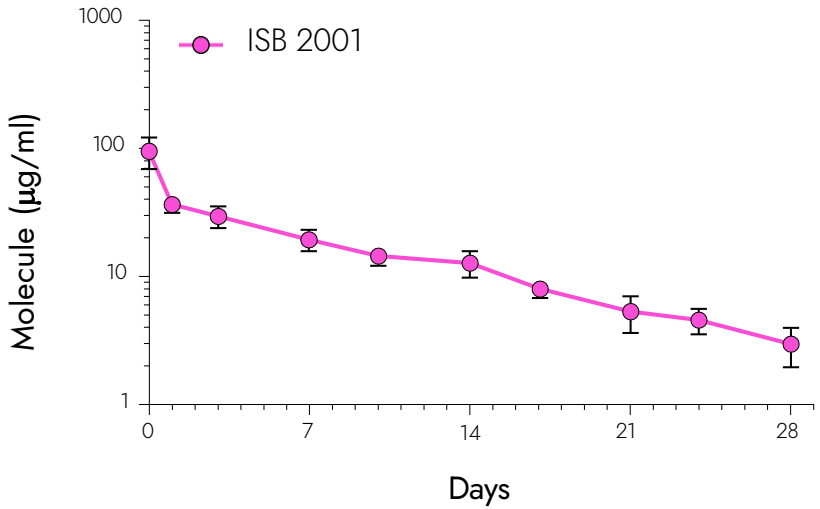
ISB 2001 Exhibits Desirable PK and shows 100% Complete Responses *In Vivo* in a BCMA^{low} CD38^{low} MM Model

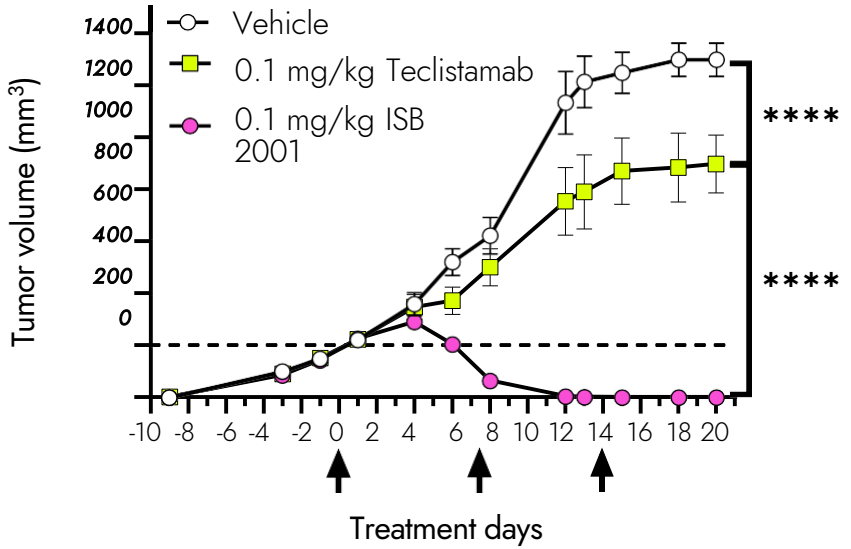
ISB 2001 Half-Life in Tg32 (huFcRn Tg) Mice

Pharmacokinetic 5 mg/kg single dose i.v.



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

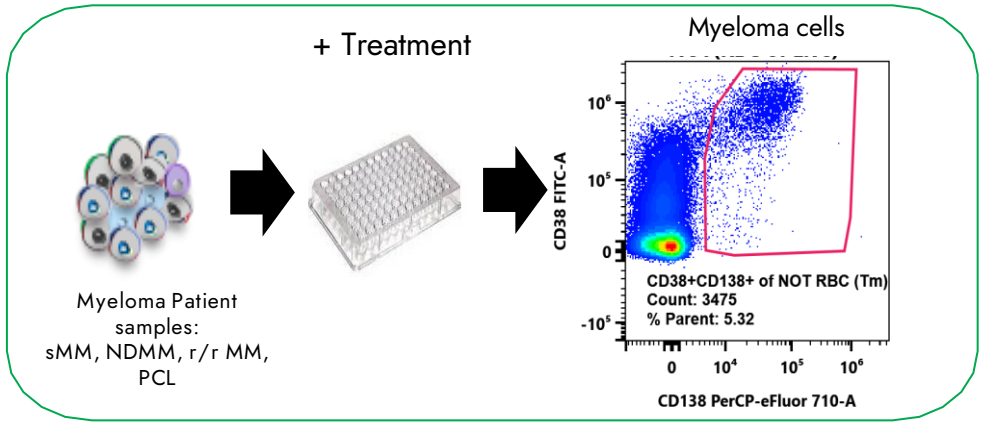
Efficacy in NSG-PBMC transfer Mouse Model (KMS-12-BM)

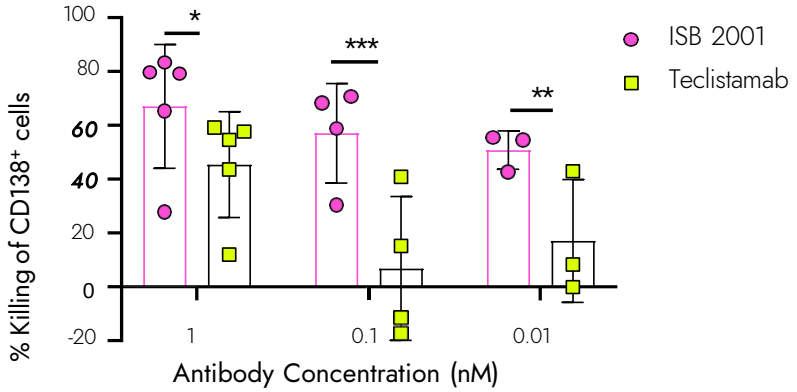


Treatment	Complete Response
Teclistamab	0% (0/8 mice)
ISB 2001	100% (8/8 mice)

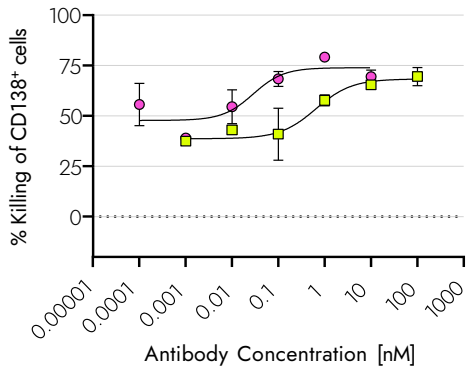
**=p <0.01
***= p <0.001
****= p <0.0001

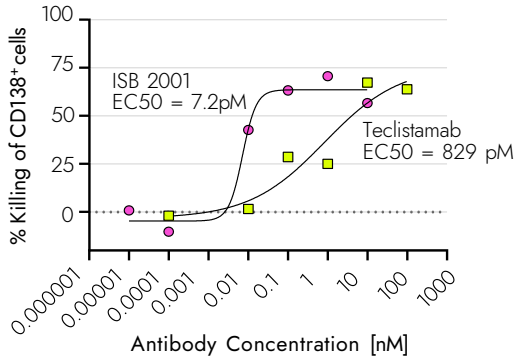
ISB 2001 Exhibits Higher Cytotoxic Potency *Ex Vivo* Compared to Teclistamab on MM Patient Samples

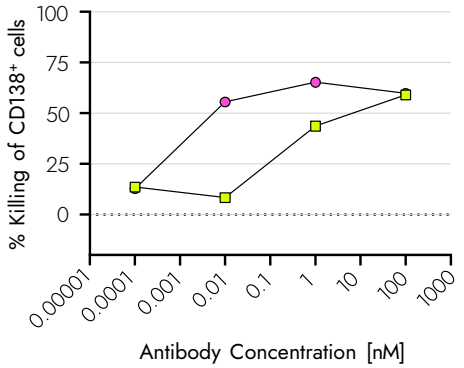


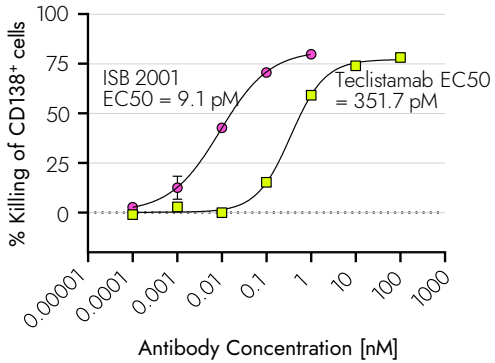


Smoldering MM → Newly Diagnosed MM → r/r MM → Plasma Cell Leukemia









*=p <0.05
**=p <0.01
***= p <0.001

sMM: Smoldering Multiple Myeloma *NDMM: Newly Diagnosed Multiple Myeloma*
r/r MM: Relapsed Refractory Multiple Myeloma *PCL: Plasma Cell Leukemia*

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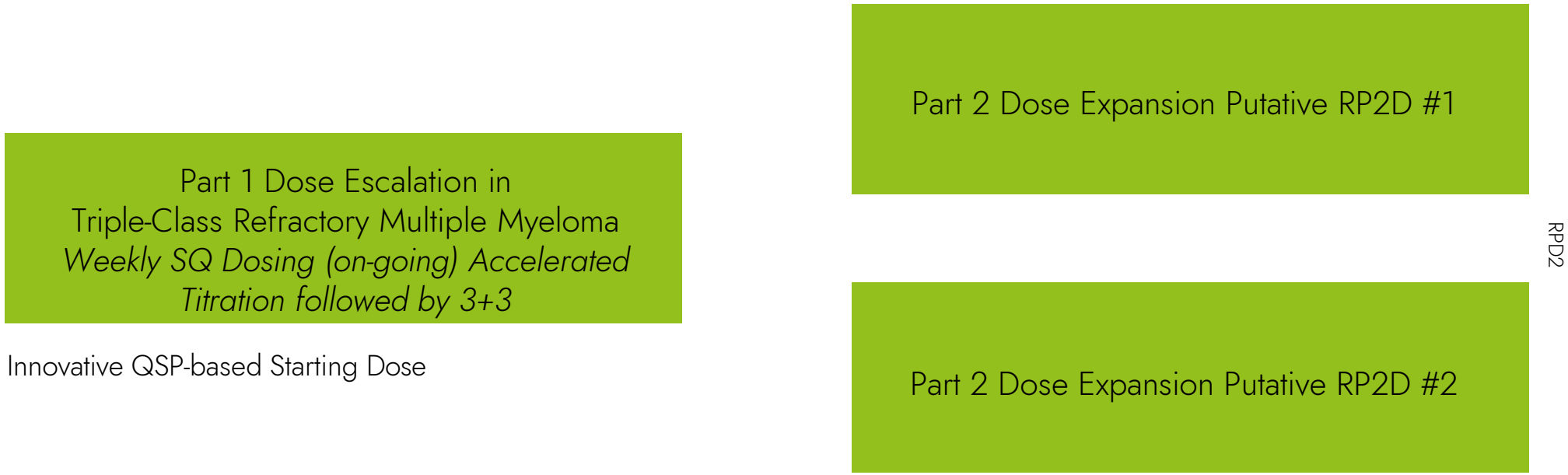
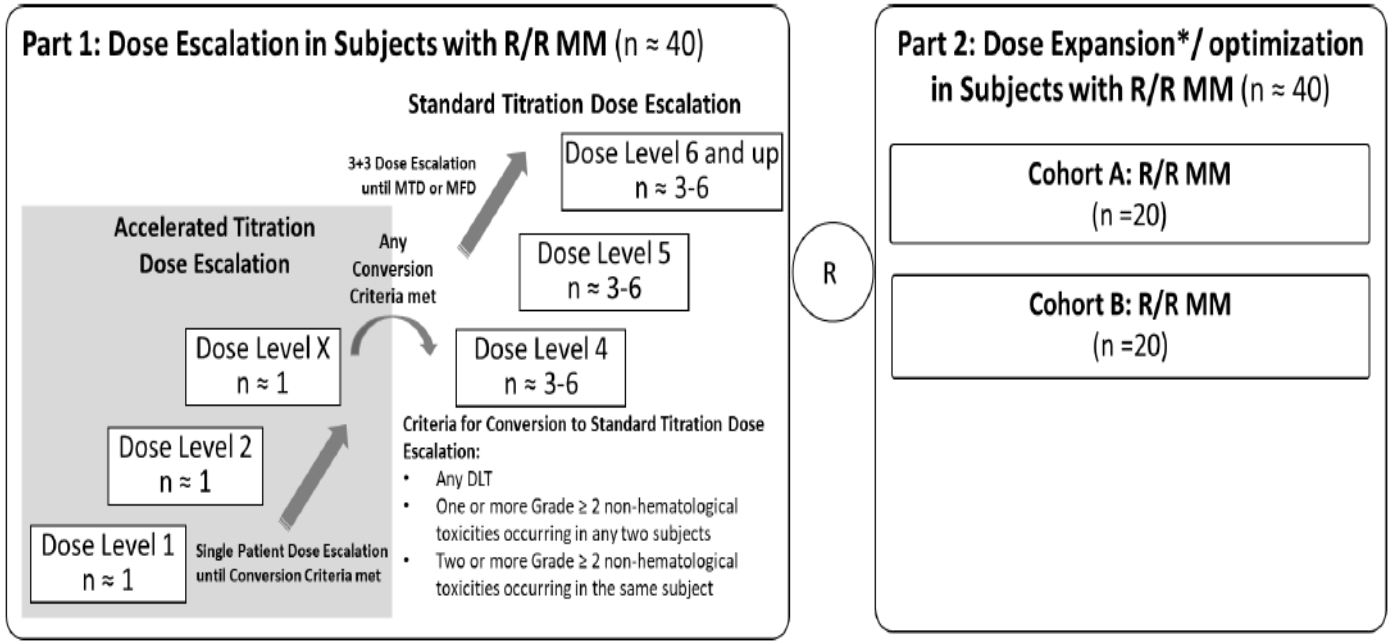
ISB 2001 Clinical Development



- Phase 1, First-in-Human, Multicenter, Open-Label, Dose Escalation and Dose-Expansion Study of Single-Agent ISB 2001 in Subjects with Relapsed/Refractory Multiple Myeloma (R/R, MM)
- Approved by the U.S. FDA on 12 April 2023, Australian HREC on 5 April 2023 and granted Orphan Drug Designation for the treatment of multiple myeloma by the U.S. FDA on 28 June 2023.
- Study is open to accrual, First patient dosed on November 1, 2023

QSP: Quantitative Systems Pharmacology (in-silico) RP2D: Recommended Phase 2 Dose

ISB 2001-101 Clinical Trial Design



- a = the numerical value that leads to the next higher dose level in the standard titration design. n = number of subjects.. X = The dose level in the accelerated titration design at which the conversion criteria is met. DLT = dose-limiting toxicity; MFD = maximum feasible dose; MM = multiple myeloma; MTD = maximum tolerated dose; RP2D = recommended phase 2 dose; R/R = relapsed or refractory.
- *According to Project Optimus, 2 putative recommended phase 2 doses will be tested. Dose levels to be determined.

Key Patient Eligibility Criteria:

- R/R MM with measurable disease after a CD38 antibody, IMiDs, PIs, and who must not be candidates for regimens known to provide clinical benefit
- Failed 3 or more prior lines of therapies

Primary Objectives:

- Assess safety, tolerability
- Determine MTD/RP2D

Secondary Objectives:

- PK, immunogenicity
- Assess preliminary clinical activity of ISB 2001

Exploratory Objectives:

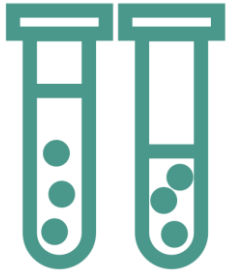
- Assess biomarkers and their correlation with clinical activity, safety, and other clinical endpoints of interest
- Assess minimal residual disease (MRD) when indicated

1Cohen, A. D. et al. J. Clin. Invest. 129, 2210–2221 (2019).
2 Nijhof, I. S. et al. Blood 128, 12 (2016).

Preliminary Results of the Ongoing Phase I Study

- Overall, treatment with ISB 2001 is well tolerated. No DLT observed.
- Low grade CRS events observed in 2/6 subjects (one Gr1 and one gr2). Injection site reactions observed in 3/6 subjects (all gr 1).
- No risk of neurotoxicity, cytopenias, or infections have been observed to date.
- All 6 subjects are ongoing on treatment (longest is 7 cycles).
- Dose escalation is ongoing with participants enrolling in DL5 (300 mg/kg).
- High interest from investigators/recruitment.

Company Summary



- 3 Phase 1 Assets in Multiple Myeloma
- Phase 1 Trial in R/R AML with ISB 1442 Planned in mid-2024
- Phase 1 Trial in Solid Tumors with Cbl-b Inhibitor GRC 65327 planned in early 2025

- Standardized Process Development and Manufacturing Operations Achieving High-Titer Yield for BEAT® Multispecifics.

- Out-licensing of All Non-Oncology Assets Completed
- Divestiture of the process development and manufacturing plant in progress

- Privately-Held Biotech Company with Plan for IPO in the medium-term

Thank you!
Let's collaborate to accelerate cancer cure

ICHNOS GLENMARK INNOVATION

Collaboration propels innovation