FROM NOW ON

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Forward-Looking Statements

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Ichnos Sciences Is a Clinical-Stage Biotechnology Company at the Forefront of Innovation in Oncology

Fully Integrated Biotech

- Global footprint: U.S. and Switzerland
- Fully owned by Glenmark, with plans to expand the investor base following achievement of clinical proof-of-concept with BEAT® platform*, anticipated in early 2022
- Accomplished management team with proven track record
- Core capabilities in biologics (discovery, antibody engineering, CMC, clinical development)

Deep and Broad Pipeline

- Focus on immune cell engagers/modulators
- Disease-centric
- Broad first-wave multispecific oncology pipeline with five programs, including a clinical-stage T-cell engager in multiple myeloma (ISB 1342)
- Beyond oncology, pipeline of potential first-in-class therapeutics addressing autoimmune diseases available to out-license

Novel BEAT® Platform

- Proprietary BEAT® antibody engineering platform represents the discovery engine to sustain innovation and drive long-term growth:
  - + Next-generation multispecific immune cell engager/modulator antibodies that can engage multiple targets simultaneously

*BEAT®: **B**ispecific **E**ngagement by **A**ntibodies based on the **T**-cell receptor
All years are calendar years

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Ichnos: Highly Experienced Biotech Leadership Team

Management

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

ERIC J. FELDMAN, M.D.
Chief Medical Officer

ROBERTO GIOVANNINI, Ph.D.
Chief Process and Manufacturing Officer

GRACE MAGUIRE, MBA
Head of Communications and Corporate Affairs

MICHAEL D. PRICE
Chief Financial Officer

Board of Directors

GLENN SALDANHA
Chairman & Managing Director, Glenmark Pharmaceuticals

DAVID LUBNER
Non-Executive Director

V S MANI
Board Member & Global CFO of GPL

LAWRENCE OLANOFF, M.D., Ph.D.
Non-Executive Director

JAYARAM PHILKANA
President & Global CHRO of GPL

DENNIS PURCELL
Non-Executive Director

SONIA QUARATINO, M.D., Ph.D.
Non-Executive Director

Ichnos Oncology Pipeline - First Wave Focuses on T-Cell Engagers and Innate Modulators - Differentiated and Potentially First-in-Class Assets

Candidate	Target	Preclinical	Phase 1	Phase 2	Phase 3	Status
ISB 1342	CD38 x CD3 BEAT® 1.0 bispecific antibody	Relapsed/Refractory Multiple Myeloma				Phase 1
ISB 1442	CD38 x CD47 BEAT® 2.0 bispecific antibody	Relapsed/Refractory Multiple Myeloma				IND-Enabling Studies
ISB 2001	TREAT™ trispecific antibody	Hematologic Malignancies				Discovery
ISB 2004	BEAT® 2.0 bispecific antibody	Hematologic Malignancies/ Solid Tumors				Discovery
ISB 2005	TREAT™ trispecific antibody	Hematologic Malignancies				Discovery

*BEAT®: **B**ispecific **E**ngagement by **A**ntibodies based on the **T**-cell receptor*
*TREAT™: **Tr**ispecific **E**ngagement by **A**ntibodies based on the **T**-cell receptor*

Note: Assets that were previously identified as GBR XXXX are now identified as ISB XXXX.

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Ichnos is considering potential partnership

Ichnos to Out-License Assets in Autoimmune Disease

Candidate	Target	Preclinical	Phase 1	Phase 2	Phase 3	Status
ISB 830 (telazorlimab)	OX40 antagonist monoclonal antibody	Atopic Dermatitis (AD)*				Phase 2b completed. Primary efficacy endpoint (EASI score, % change, baseline to Week 16) met at two highest doses tested.†
ISB 880	IL-1RAP antagonist monoclonal antibody	Autoimmune Disease				IND-Enabling Studies Completed

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

†Numerical improvements were seen for the two higher dose arms (300 mg and 600 mg q 2 weeks) of telazorlimab compared to placebo in the secondary endpoints of EASI-75 and Investigator Global Assessment, but the differences were generally not statistically significantly different from placebo.

Note: Assets that were previously identified as GBR XXXX are now identified as ISB XXXX

BEAT® Platform

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Ichnos BEAT® Is Among the Most Innovative Multispecific Platforms

1st Generation

Fragment based Formats

Genetic fusion of 2 antibody fragments - scFvs, with or without Fc

- Limited target druggability and developability
- Not suited for multispecific antibodies

2nd Generation

Fab-Fragment based Formats

Genetic fusion of a Fab and an antibody fragment - scFv or dAb, with Fc

- Improved druggability and developability – Rapid engineering
- Not optimal for multispecific antibodies

Ichnos BEAT® 1.0 Platform – Proprietary heavy chain technology – Assemble light chains via scFv formatting

3rd Generation

Fab-Fab based Formats

Mimic the natural antibody format

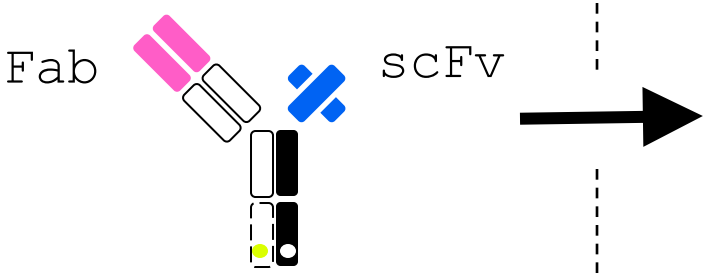
- Optimal druggability and developability – Rapid engineering
- Best suited for multispecific antibodies

Ichnos BEAT® 2.0 Platform – Assemble light chains via common light chain Fab technique

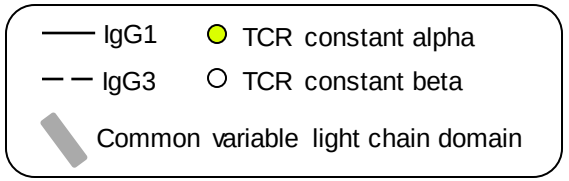
Domain antibody (dAb) is an antibody fragment consisting of a single monomeric variable antibody domain that is able to bind an antigen; Antigen-binding fragment (Fab) is the natural region on an antibody that binds to an antigen. It is composed of one constant and one variable domain of each of the heavy and the light chain; Fragment crystallizable (Fc) region is the tail region of an antibody that interacts with cell surface receptors called Fc receptors, including the neonatal Fc receptor, a key determinant in maintaining and prolonging antibody plasma half-life; Single chain fragment variable (scFv) is a genetic fusion of the heavy and light chain variable domains of an antibody that is able to bind an antigen

Ichnos BEAT® Platform Delivers Optimized and Readily Developable Multispecific Antibodies

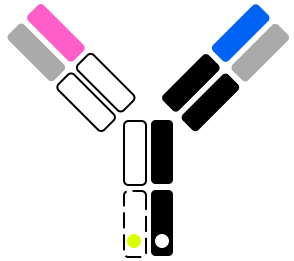
BEAT® 1.0



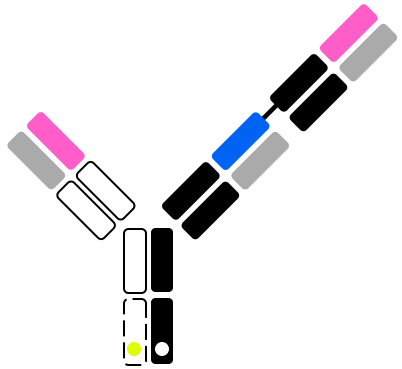
- Versatile and quick to engineer
- Not suited for trispecific format



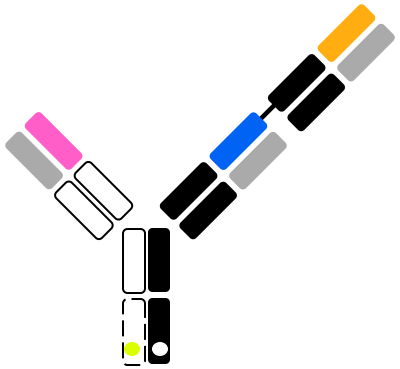
BEAT® 2.0 (1+1)



BEAT® 2.0 (2+1)



TRISPECIFIC (TREAT™)



Enables deep exploration of the bi/multispecific design space to optimize drug candidates and unlock new biology (e.g., T cell, NK cells, macrophage engagers) by testing:

+ **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*

BEAT®: Bispecific Engagement by Antibodies based on the T-cell receptor
TREAT™: Trispecific Engagement by Antibodies based on the T-cell receptor

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Capabilities Extending From Antibody Engineering Through Manufacturing Provide Competitive Advantage

Process and Analytical Development and GMP-certified Manufacturing Facility

Advantages

- Dedicated to BEAT® 2.0 platform
- Fully integrated with Ichnos Discovery
- Continuous process improvement with potential IP extension
- Better control on manufacturing timelines by avoiding lead time associated with Contract Manufacturing Organizations
- Competitive timelines to IND filings

Oncology Compounds

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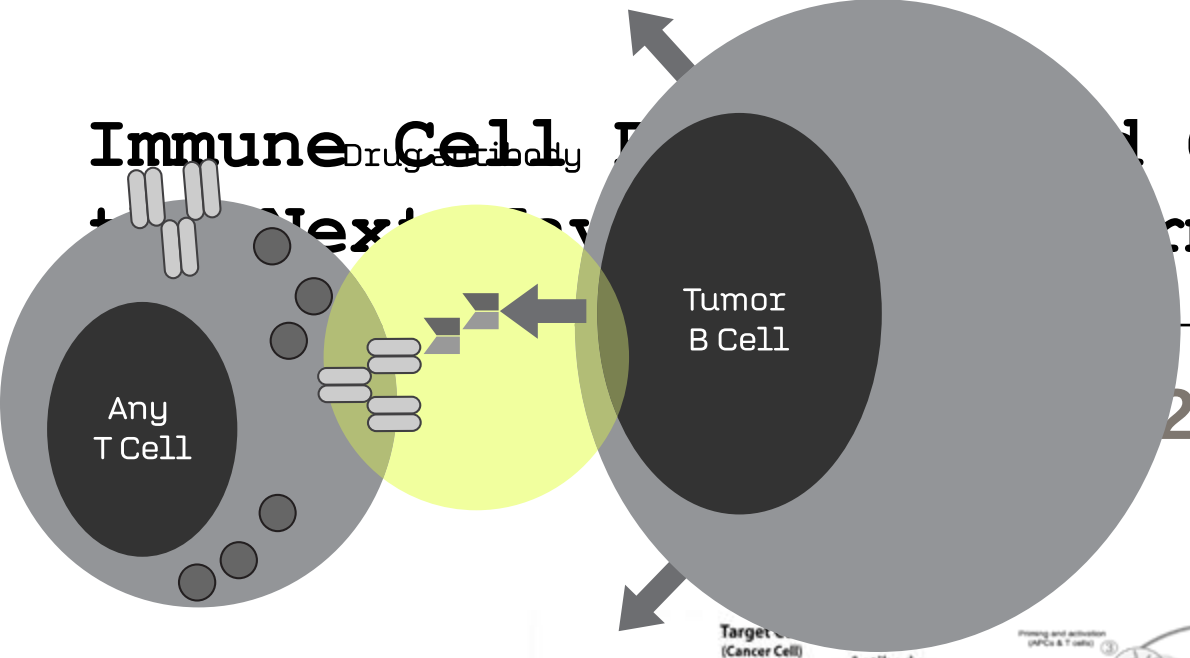
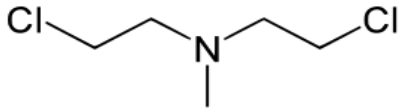
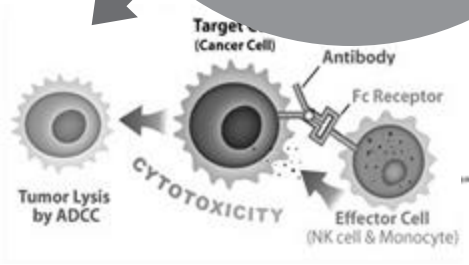
Immune Cell [illegible] Cell Therapies Are Emerging as [illegible] rmative Medicines in Oncology

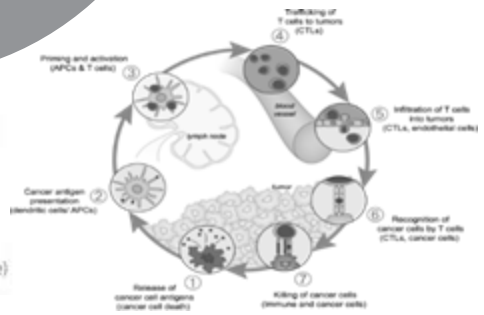


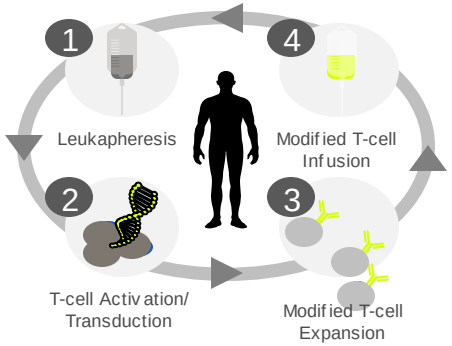
2010s

Present









Chemotherapy
Indiscriminate – kills healthy and cancer cells

Targeted Therapies
Target receptor/molecular oncogenic drivers

Immuno-Oncology
Checkpoint and innate immunity modulators

Cell Therapies
Re-engineered T cells

Immune Cell Engagers
Bispecific antibodies
Multispecific antibodies

CAR: Chimeric Antigen Receptor
Sudhakar A. *J Cancer Sci Ther.* 2009;1:1-4;
Checkpoint inhibitors image: Reprinted from *Immunity*, Vol 39, Chen DS, Mellman I. Oncology Meets Immunology: The Cancer-Immunity Cycle, Pages 1-10, Copyright 2013, with permission from Elsevier.

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ISB 1342

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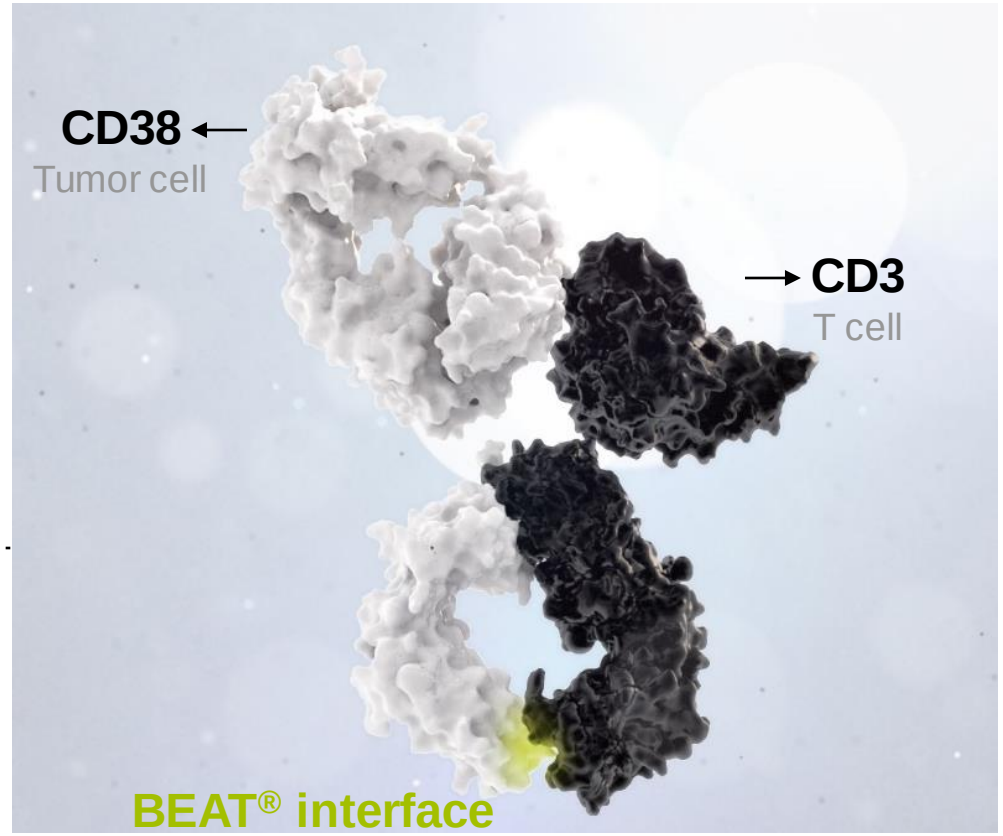
ISB 1342 (CD38 x CD3) Bispecific Antibody: Potential First-in-Class Therapy in Relapsed/Refractory Multiple Myeloma

Key Attributes

- CD38 is expressed on the surface of multiple myeloma cells and is a validated target
- ISB 1342 is a bispecific antibody that redirects T lymphocytes 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 or isatuximab
- ISB 1342 is designed to overcome:
  - + Daratumumab resistance by killing low CD38-expressing tumor cells
  - + Resistance to CDC and ADCC mediated by daratumumab

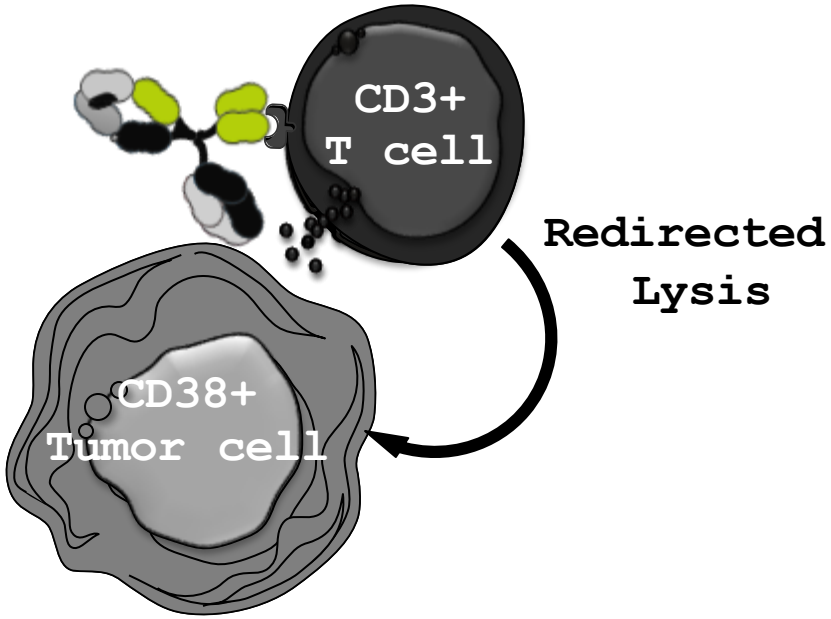
- Granted orphan drug designation in 2019
- Phase 1 dose escalation and expansion study, including biweekly and weekly dosing, is ongoing
- Clinical proof-of-concept anticipated in early 2022

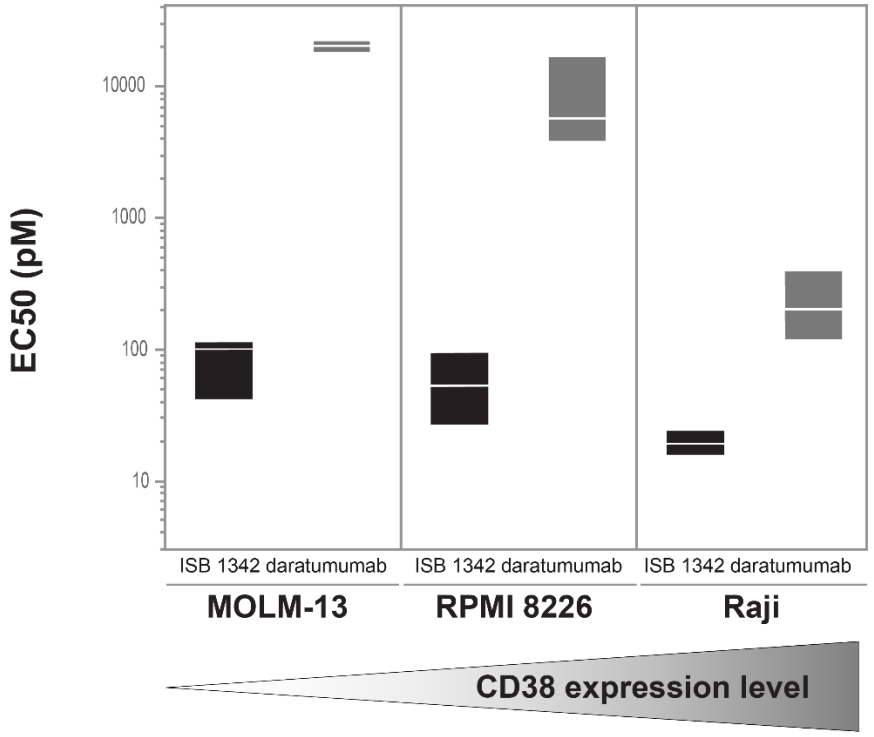
ISB 1342 (CD38 x CD3) bispecific antibody | BEAT® 1.0



MHC: Major histocompatibility complex, CDC: Complement-Dependent Cytotoxicity
ADCC: Antibody-Dependent Cell-mediated Cytotoxicity
All years are calendar years

ISB 1342 Induces More Potent Redirected Lysis Against Various CD38-Expressing Tumor Cells Compared to Daratumumab In Vitro





By co-engaging TCR/CD3ε on T lymphocytes and CD38 on tumor cells, ISB 1342 induces the formation of an immunological synapse between T cells and tumor cells and the redirected lysis of tumor cells (left panel). The potency of daratumumab and ISB 1342 to kill in vitro tumor cells expressing low, intermediate, and high levels of CD38 was compared in a Multiple Mode of Action Killing Assay that combines ADCC, CDC, and redirected cell lysis (right panel).

Journal of Clinical Oncology 39, no. 15_suppl (May 20, 2021) 8044-8044
Clinical significance unknown.
ADCC: Antibody-Dependent Cell-mediated Cytotoxicity; CDC: Complement-Dependent Cytotoxicity

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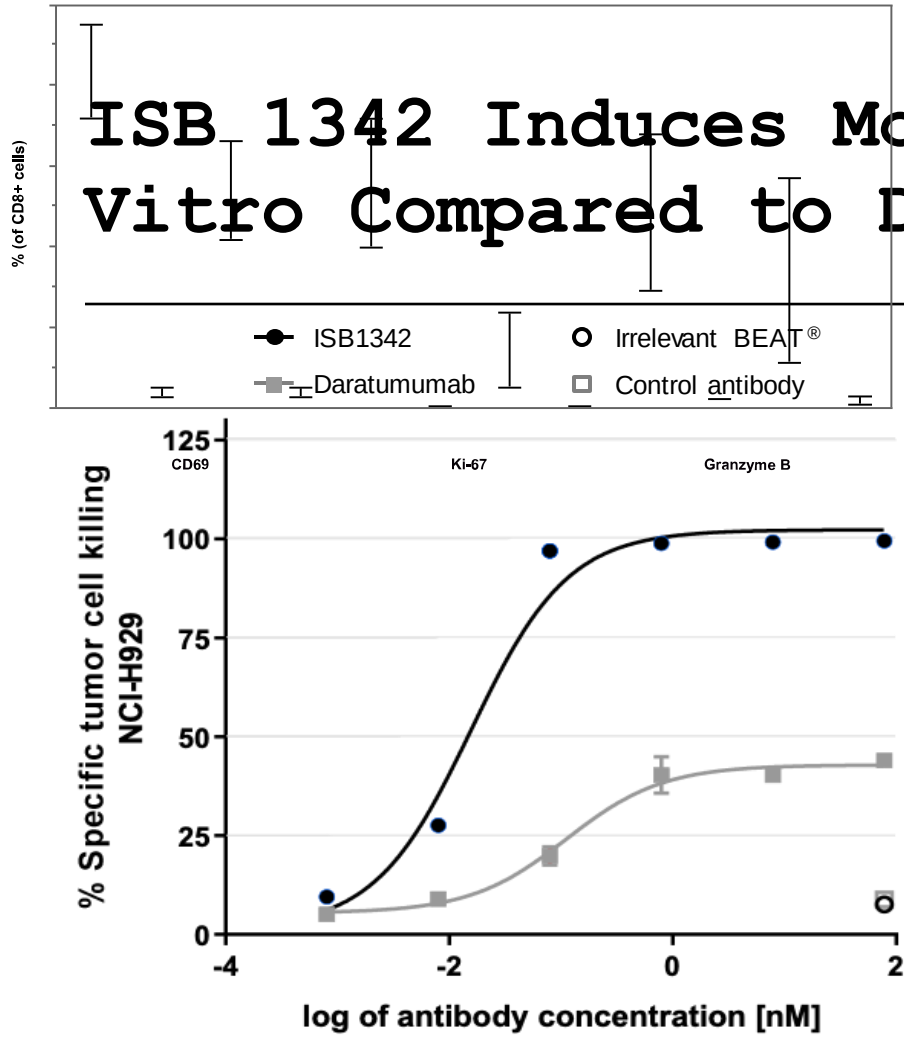
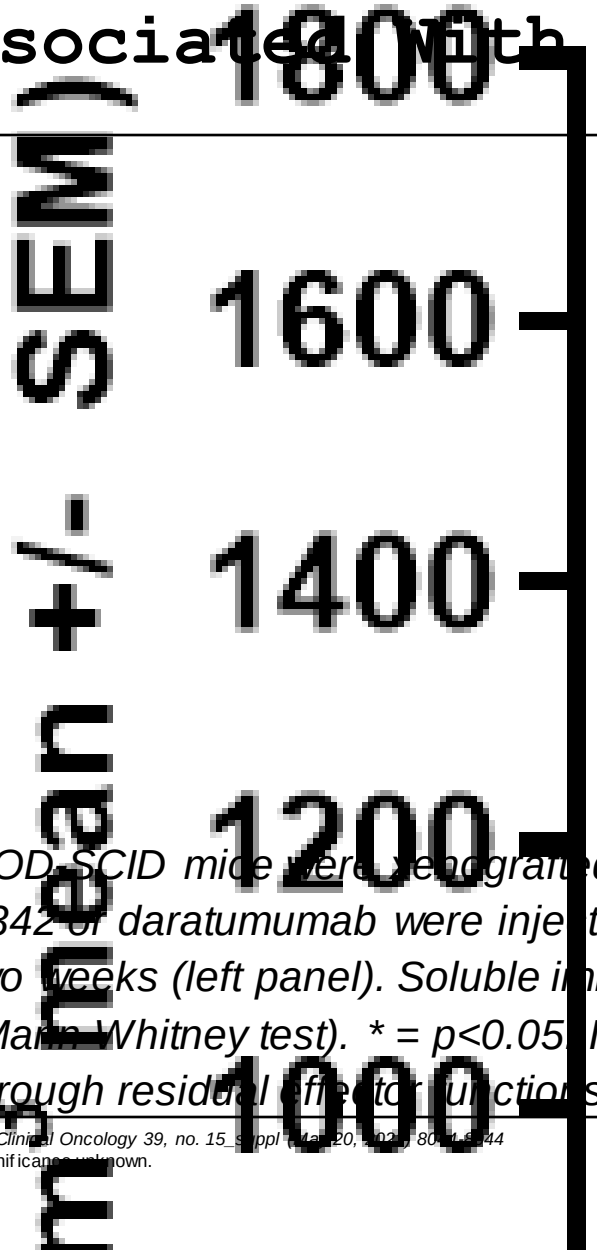
ISB 1342 Induces More Potent Tumor Cell Killing In Vitro Compared to Daratumumab



Potency of ISB 1342 and daratumumab to kill NCI-H929 cells (CD38 intermediate tumor cells) in vitro in a Multiple Mode of Action Killing Assay (left panel). Specific tumor cell killing was measured at t=48h; isotype control antibody and irrelevant BEAT ® with dummy CD3 and CD38 binders were used as negative controls. T-cell activation in response to maximum dose of ISB 1342 and daratumumab was measured in the same assay at t=48h (right panel).

Journal of Clinical Oncology 39, no. 15_suppl (May 20, 2021) 8044-8044
Clinical significance unknown.

ISB 1342 Effectively Controls Tumor Growth In Vivo Associated With Production of Cytolytic Markers



*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 mm^3 and tumor growth monitored over two weeks (left panel). Soluble immune factors were quantified in dissociated tumors one-week post-treatment (right panel). (Mann-Whitney test). * = p<0.05. ISB 1342_CD3DU (made of irrelevant CD3 binder) partially controls tumour growth through residual effector functions.*

Journal of Clinical Oncology 39, no. 15_suppl (May 20, 2021) 8044-8044
Clinical significance unknown.

ISB 1442

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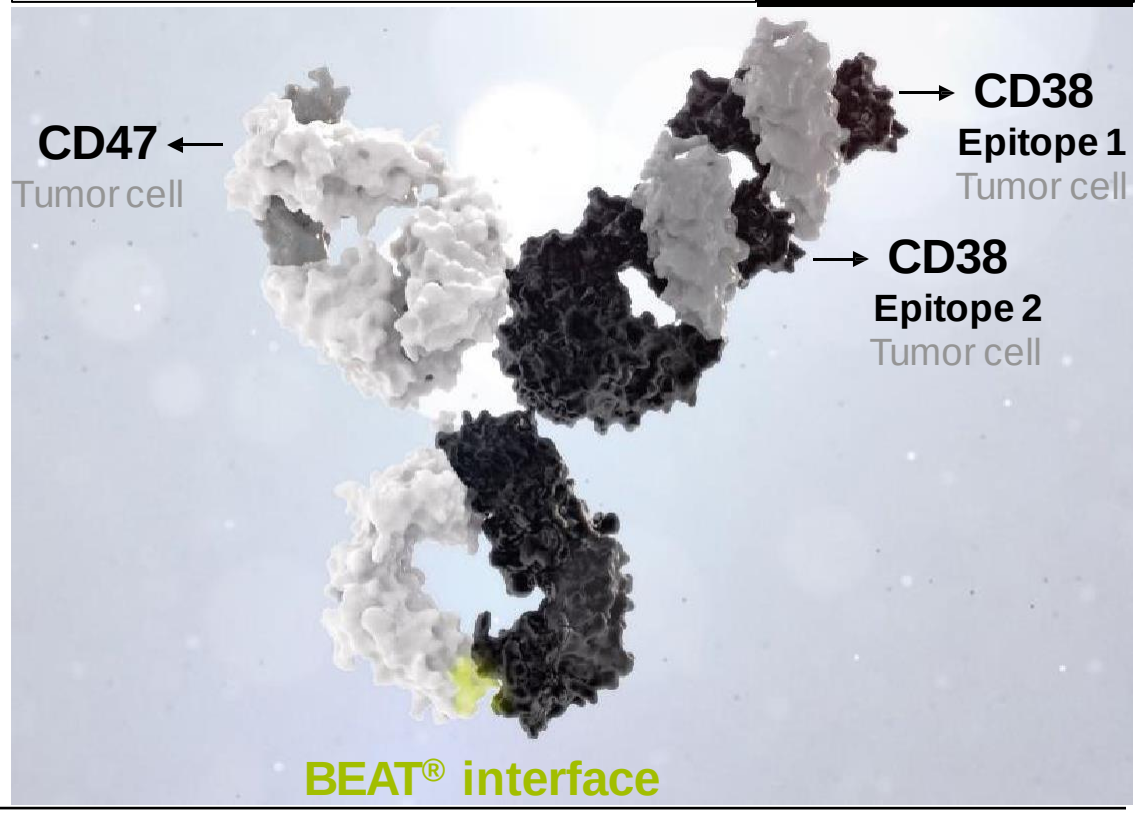
ISB 1442 (CD38 X CD47) BEAT® 2.0: Potential First-in-Class Therapy in Relapsed/Refractory Multiple Myeloma

Key Attributes

- Redirects myeloid cells to kill CD38+ tumors
- Blocks CD47 inhibitory activity to enhance myeloid-mediated killing of CD38-expressing tumor cells
- Biparatopic 2+1 bispecific antibody combines 2 proprietary anti-CD38 binding arms targeting 2 different regions on CD38 with antagonistic anti-CD47 arm, equivalent to a trispecific antibody
- Potent Antibody-Dependent Cell-mediated Cytotoxicity (ADCC) and Complement-Dependent Cytotoxicity (CDC) based on optimized affinity, epitope, architecture, and Fc engineering
- Avoids antigen sink: CD47 is widely expressed (including on red blood cells and platelets)
- Optimized tolerability: hemagglutination, platelet aggregation
- US IND targeted for 1Q 2022 in multiple myeloma

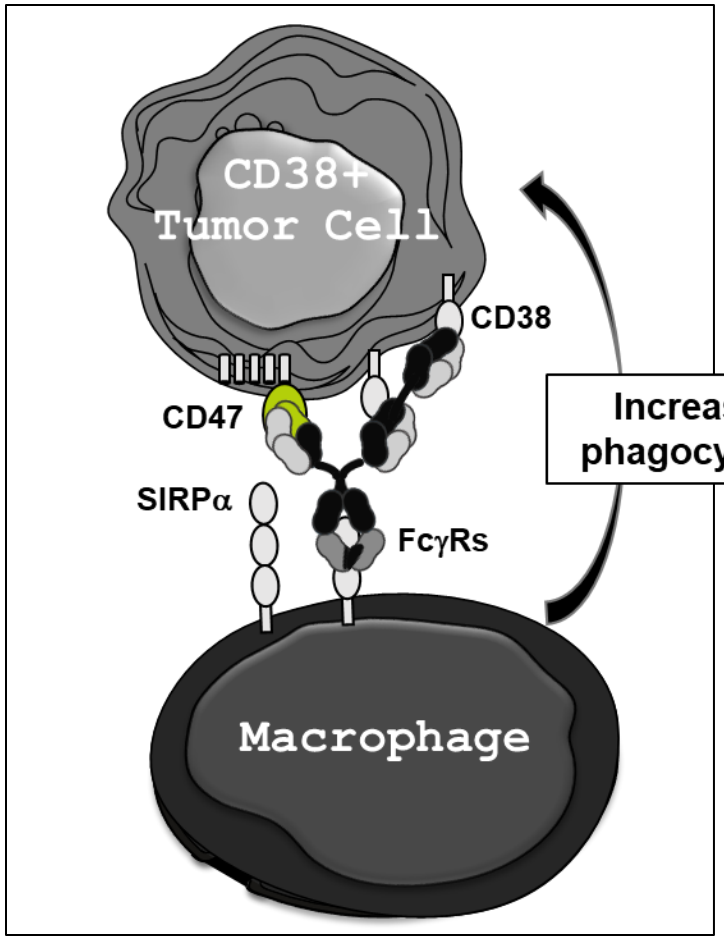
ISB 1442 (CD38 x CD47)
2+1 bispecific antibody

BEAT® 2.0



*BEAT®: **B**ispecific **E**ngagement of **A**ntibodies based on the **T**-cell receptor*
All years are calendar years

ISB 1442 (CD38 X CD47) BEAT® 2.0 – Redirect Myeloid Cell-Mediated Killing of CD38-Expressing Tumor Cells in Multiple Myeloma



CD47

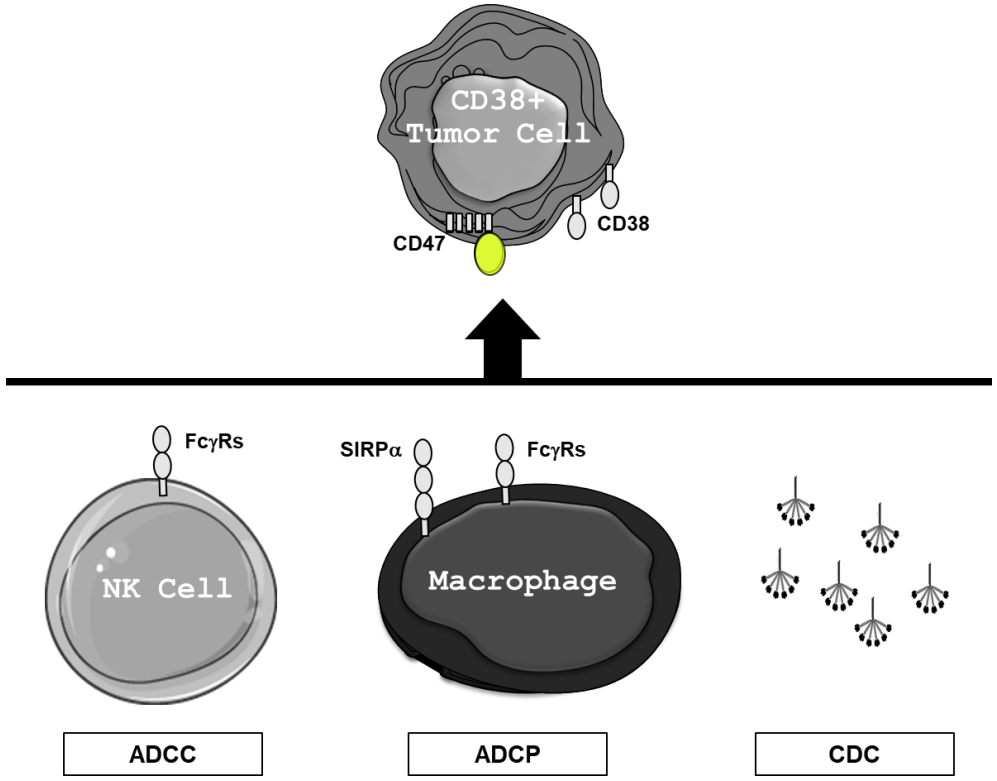
- "Don't eat me" signal: CD47 inhibits phagocytosis through interaction with Signal Regulatory Protein alpha (SIRPα) expressed on phagocytes
- CD47 plays a broad role in cancer immune evasion
- CD47 is over-expressed by hematological and solid tumors
- Increased expression of CD47 correlates with a worse prognosis in several hematological malignancies

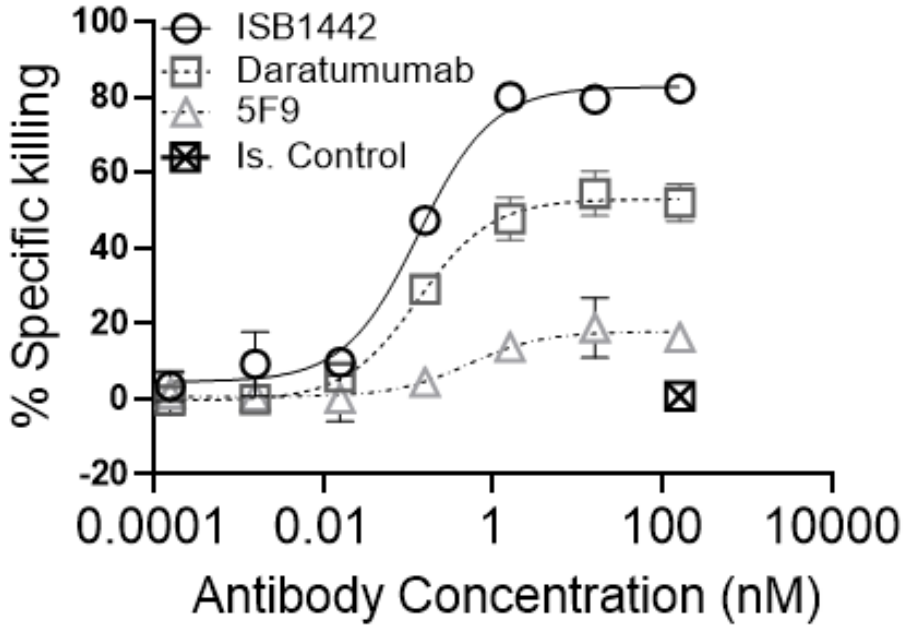
CD38

- CD38 is a clinically approved target in multiple myeloma (MM)
- Resistance to daratumumab in MM is associated with decreased CD38 surface expression and up-regulated CD47 expression on tumor cells

*BEAT® : **B**ispecific **E**ngagement of **A**ntibodies based on the **T**-cell receptor*

ISB 1442 (CD38 X CD47) Clinical Candidate Shows a Higher Killing Potency of CD38-Expressing Tumor Cells Than Clinical Benchmarks In Vitro



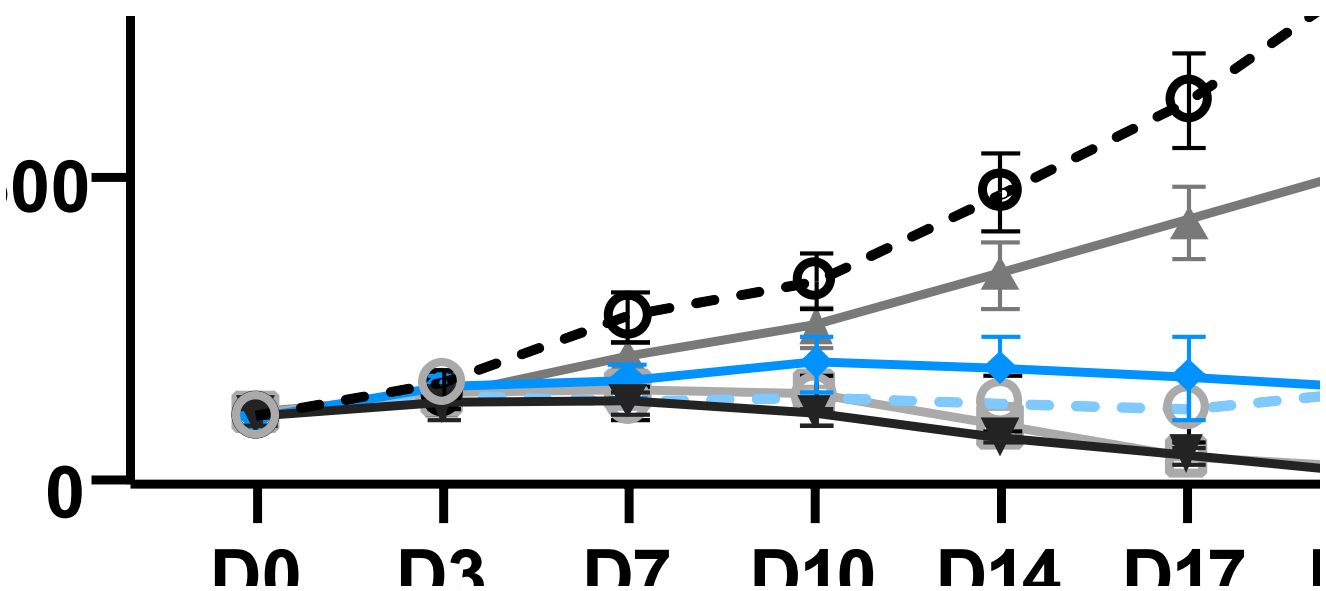


Assay: Multiple Mode of Action Killing assay performed with NCI-H929 tumor cells (CD38 low-int expressing cells)

Assay combines ADCC and CDC to enable direct comparison of ISB 1442 with 5F9 (ADCP) and daratumumab (ADCC, CDC); 5F9 is magrolimab from Forty Seven, Inc.

Data on file. Clinical significance unknown.
ADCC: Antibody-Dependent Cellular Cytotoxicity; ADCP: Antibody-Dependent Cellular Phagocytosis;
CDC: Complement-Dependent Cytotoxicity

ISB 1442 and Single Arm Controls Show Similar Efficacy to anti-CD47 Comparator 5F9 in Raji Preclinical Model



CB17/SCID mice were implanted subcutaneously with 10 million Raji cells and stratified into groups when tumor volume reached ~100 mm³. Graph shows mean +/- SEM of tumor volume for 5 mice. Statistical analysis conducted by 2-way ANOVA w. Geisser-Greenhouse correction.

* 5F9 (magrolimab) dosed weekly at 1

Daratumumab dosed bi-weekly at 1

*Denotes statistically significant difference from ISB 1442

* *ISB 1442 shows superior efficacy to daratumumab. Single arm controls show substantial efficacy at this high dose. Strong efficacy with anti-CD47 blocker 5F9 is expected in this model because anti-tumor immunity is driven by innate effectors with a strong bias to the CD47-SIRP1α axis.*

Data on file. Clinical significance unknown.

Available for Out-License:
Autoimmune Biologics

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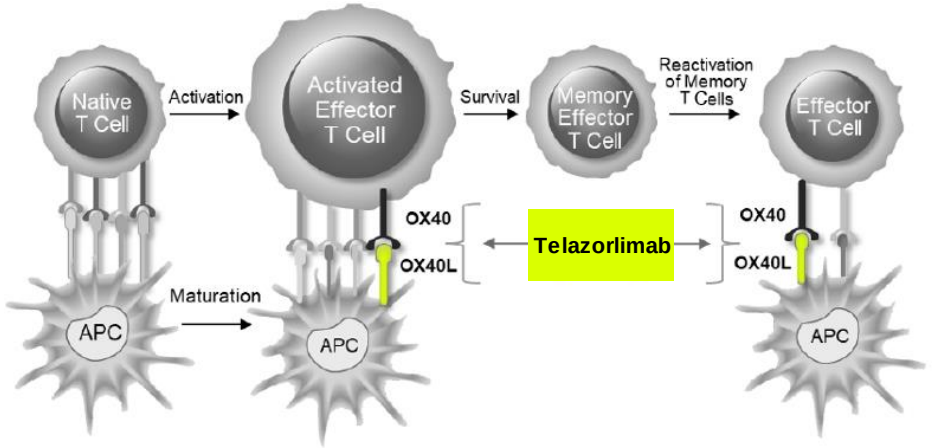
OX40 Antagonist Telazorlimab(ISB 830)Showed Efficacy in Phase 2b Study in Atopic Dermatitis (AD)

Study Results and Status:

- Telazorlimab met the primary endpoint of EASI score, % change from baseline to Week 16 at the two highest doses (vs. placebo)[1]
  - + Improvement was also observed for the two higher doses versus placebo in secondary endpoints, EASI-75[2] and IGA[3] (generally not statistically significant)
  - + Clinical efficacy continued to improve after Week 16, with maximal impact achieved several weeks later
  - + Reduction in AD disease activity was maintained after discontinuation of telazorlimab, through three months of follow-up
- Telazorlimab was safe in this study, and the most commonly reported adverse events (>5%) were atopic dermatitis, nasopharyngitis, upper respiratory tract infection, and headache[4]
- A US IND to conduct studies of telazorlimab in autoimmune diseases, including rheumatoid arthritis (RA), is active and Ichnos plans to out-license this asset for further development

Signaling through OX40 increases disease activity by enhancing T effector activation and production of proinflammatory cytokines



- OX40 (CD134) is a key T-cell costimulatory molecule involved in autoimmunity and inflammation
- Telazorlimab binds to OX40 on the surface of activated T cells, preventing T effector proliferation and cytokine production

EASI: Eczema Area and Severity Index; IGA: Investigator Global Assessment

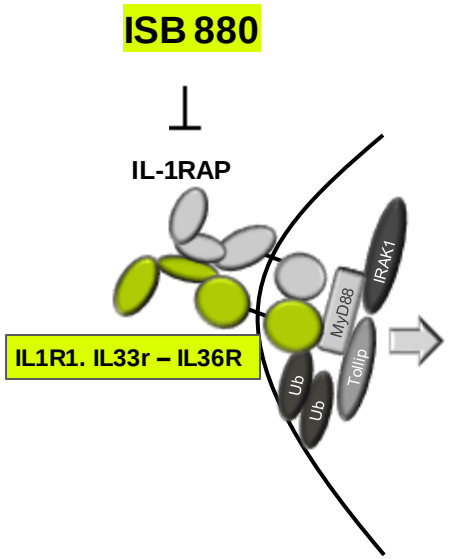
[1] 2021 Society for Investigative Dermatology Virtual Meeting
[2] Proportion of patients with ≥75% improvement in EASI score from baseline to Week 16
[3] Proportion of patients with IGA of clear or almost clear (0 or 1) and ≥2 point reduction from baseline at Week 16
[4] One death observed due to pre-existing hypertension in the telazorlimab group- Investigator reported this as unrelated to study drug

ISB 880 (Anti-human IL1RAP Antagonist mAb): Blocks Multiple Disease Drivers (IL1R, IL36R, and IL33R) in Inflammatory Diseases and Oncology

Key Attributes

- Fully human, high affinity (Kd ~250 pM) antagonist mAb against human IL1RAP (human IgG1 isotype, silenced Fc function)
- Blocks signaling of 3 key disease drivers IL1R, IL36R, and IL33R and downstream inflammatory response
- Potential to deliver superior and sustained clinical efficacy in broad disease indications
- US IND targeted for 2H 2021 in autoimmune disease
- Available for out-license

Disease Pathophysiology



- Epithelial barrier dysfunction
- Inflammation
- Fibrosis
- Tissue remodeling

Potential Indications

- Inflammatory Diseases
- Oncology

All years are calendar years

Ichnos Sciences Is a Clinical-Stage Biotechnology Company at the Forefront of Innovation in Oncology

Fully Integrated Biotech

- Global footprint: U.S. and Switzerland
- Fully owned by Glenmark, with plans to expand the investor base following achievement of clinical proof-of-concept with BEAT® platform*, anticipated in early 2022
- Accomplished management team with proven track record
- Core capabilities in biologics (discovery, antibody engineering, CMC, clinical development)

Deep and Broad Pipeline

- Focus on immune cell engagers/modulators
- Disease-centric
- Broad first-wave multispecific oncology pipeline with five programs, including a clinical-stage T-cell engager in multiple myeloma (ISB 1342)
- Beyond oncology, pipeline of potential first-in-class therapeutics addressing autoimmune diseases available to out-license

Novel BEAT® Platform

- Proprietary BEAT® antibody engineering platform represents the discovery engine to sustain innovation and drive long-term growth:
  + Next-generation multispecific immune cell engager/modulator antibodies that can engage multiple targets simultaneously

*BEAT®: **B**ispecific **E**ngagement by **A**ntibodies based on the **T**-cell receptor
All years are calendar years

...ichnos...

FROM NOW ON

...ichnos...

Thank you

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