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 in 2021 and beyond
- 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 seven programs, including two clinical-stage T cell engager assets in multiple myeloma (ISB 1342) and metastatic HER2+ breast cancer (ISB 1302)
- Beyond oncology, pipeline of potential first-in-class therapeutics addressing autoimmune disease and pain: 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

Ichnos: Highly Experienced Biotech Leadership Team

Management

ALESSANDRO RIVA, M.D.★
Chief Executive Officer

MARTIN WILSON
General Counsel

KALA SUBRAMANIAN,Ph.D.
Chief of Staff, Head of Strategic Development, Business Development & Licensing

GABRIELA GRUIA, M.D.*
Chief Development Officer

M. LAMINE MBOW, Ph.D.
Head of NBE, Discovery Research

NEELUFAR MOZAFFARIAN,M.D.,Ph.D.
Head of Clinical Sciences, Autoimmune Disease, and Pain

ROBERTO GIOVANNINI, Ph.D.
Head of CMC Development

STEPHANE CHERIX
Head of Finance

ISABEL CARMONA
Chief Human Resources Officer

GRACE MAGUIRE, MBA
Head of Communications and Corporate Affairs

* Recently retired. Consulting until a Chief Medical Officer is hired.

Board of Directors

Glenn Saldanha
Chairman & Managing Director, Glenmark Pharmaceuticals

Bernard Munos
Non-Executive Director

Dennis Purcell
Non-Executive Director

V S Mani
Board Member & Global CFO of GPL

Jayaram Philkana
President & Global CHRO of GPL

Marcela Maus, M.D., Ph.D.
Non-Executive Director

David Lubner
Non-Executive Director

Lawrence Olanoff, M.D., Ph.D.
Non-Executive Director

★ Also a member of Board of Directors

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

Candidate	Target	Preclinical	Phase 1	Phase 2	Phase 3	Status
ISB 1342	CD38 x CD3 BEAT® bispecific antibody	Relapsed/Refractory Multiple Myeloma				Phase 1 Enrolling
ISB 1908	CD38 x CD3 BEAT® bispecific antibody	Relapsed/Refractory Multiple Myeloma				Pre-IND
ISB 1909	BEAT® T cell engager bispecific antibody	Undisclosed				Discovery
ISB 1442	CD38 x CD47 BEAT® bispecific antibody	Hematologic Malignancies				Pre-IND
ISB 2004	BEAT® bispecific antibody	Undisclosed				Discovery
ISB 2001	BEAT® trispecific antibody	Undisclosed				Discovery
ISB 1302	HER2 x CD3 BEAT® bispecific antibody	Metastatic HER2+ Breast Cancer				Phase 1 Enrolling

Ichnos is considering potential partnership

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

Ichnos to Out-License Small Molecules and Assets in Autoimmune Disease

Candidate	Target	Preclinical	Phase 1	Phase 2	Phase 3	Status
Oncology						
ISC XXXXX	HPK1 inhibitor small molecule	Undisclosed				IND-Enabling Activities
Autoimmune Disease						
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 Activities
Pain						
ISC 17536	TPRA1 antagonist small molecule	Diabetic Peripheral Neuropathy (DPN) Phase 2a Proof of Concept (PoC)				Completed preclinical toxicology study and formulation study in healthy volunteers†

*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. A US IND for Rheumatoid Arthritis and other autoimmune indications is active.

†A Phase 2a PoC study in patients with painful DPN was previously completed. The primary endpoint was not met for the overall study population, but a statistically significant reduction in pain was seen in a prespecified subgroup of patients with preserved small nerve fiber function. During a Type C meeting with FDA in March 2020, agreement was reached regarding the nonclinical plan to enable a randomized, double-blind, placebo-controlled, Phase 2b, dose-range-finding study for painful DPN. Preclinical toxicology studies and a formulation study in healthy volunteers were recently completed. All rights for ISC 17536 are in the process of being transferred to Glenmark, Ichnos' parent company. Out-licensing responsibility will also be assumed by Glenmark.

Note: Assets that were previously identified as GBR and GRC are now identified as ISB (for biologics) and ISC (for small molecules), respectively.

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

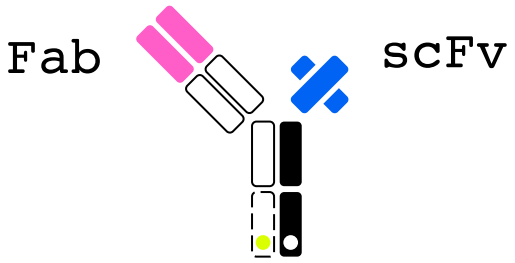
- Optimal druggability and developability – Rapid engineering
- Best suited for multispecific antibodies

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

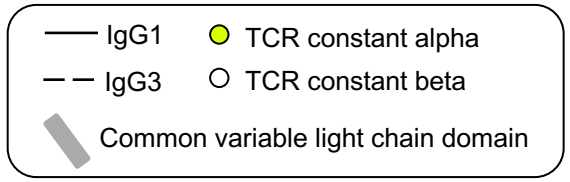
Domain antibody (dAb) is an antibody fragment consisting of a single monomeric variable antibody domain that is able to bind an antigen; Antigen-binding fragment (Fab) is the natural region on an antibody that binds to an antigen. It is composed of one constant and one variable domain of each of the 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

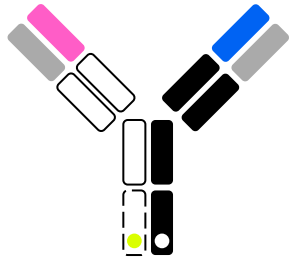
BEAT® 1.0



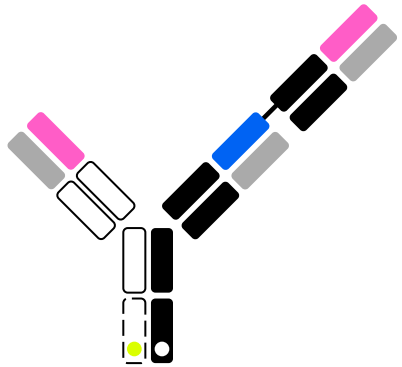
- Versatile and quick to engineer
- Not suited for trispecific format



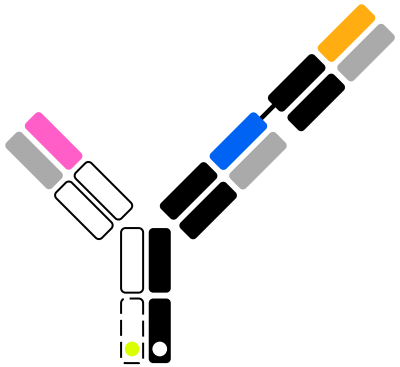
BEAT® 2.0 (1+1)



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

Oncology Compounds in Clinical Development

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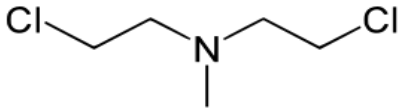
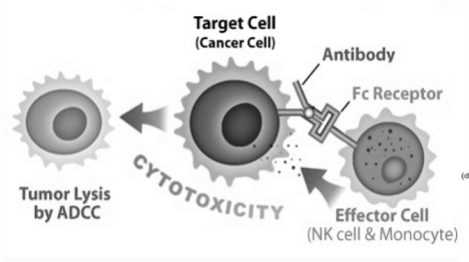
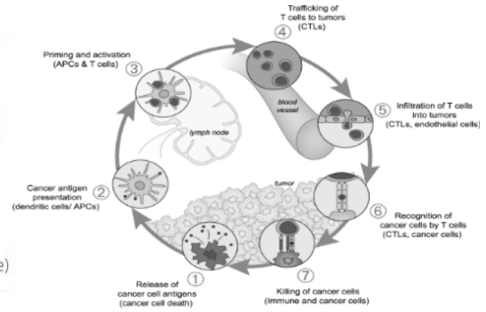
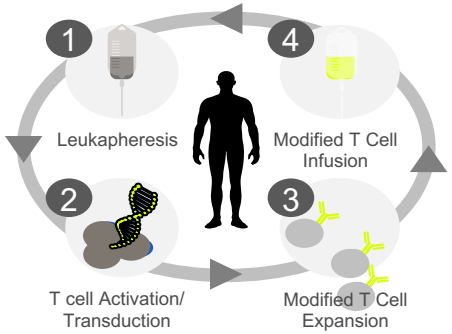
Immune Cell Engagers and Cell Therapies Are Emerging as the Next Wave of Transformative Medicines in Oncology

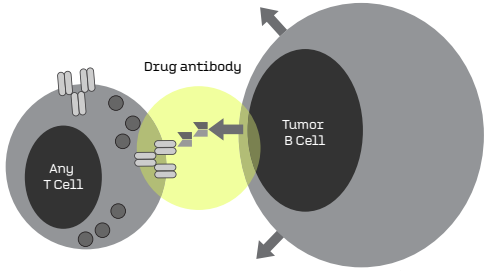
1940s 1990s 2010s Present











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.

ISB 1342

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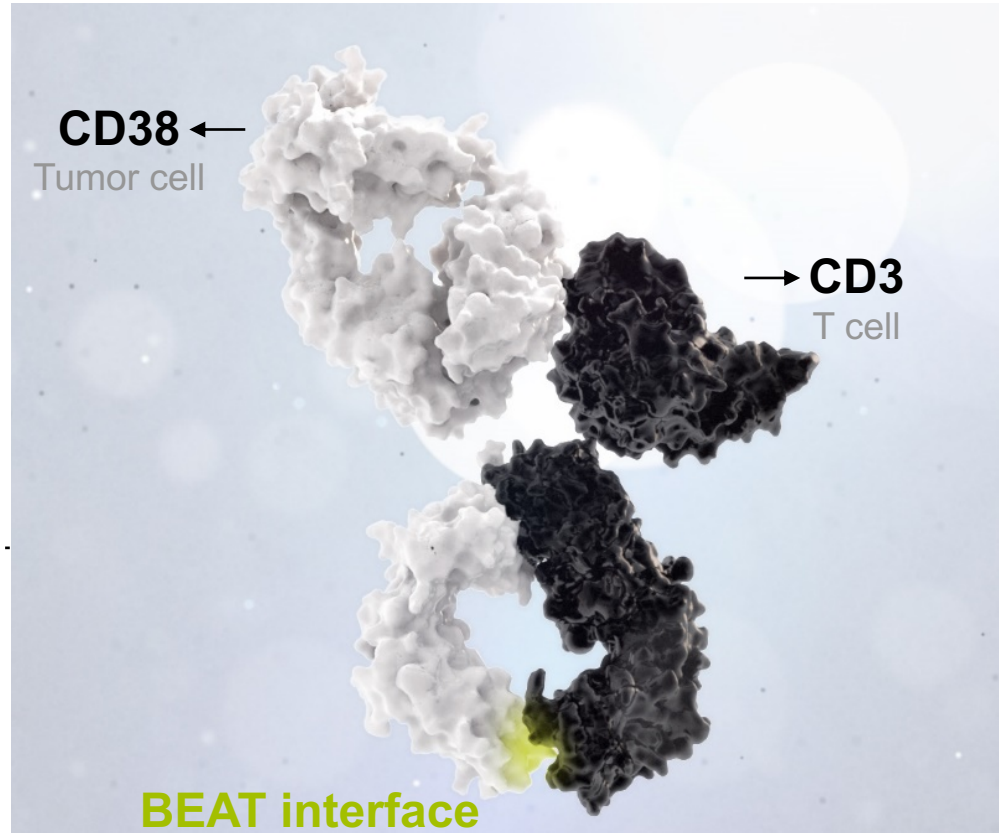
ISB 1342(CD38 x CD3)Bispecific Antibody: Potential First-in-Class Therapy in Relapsed/Refractory Multiple Myeloma

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

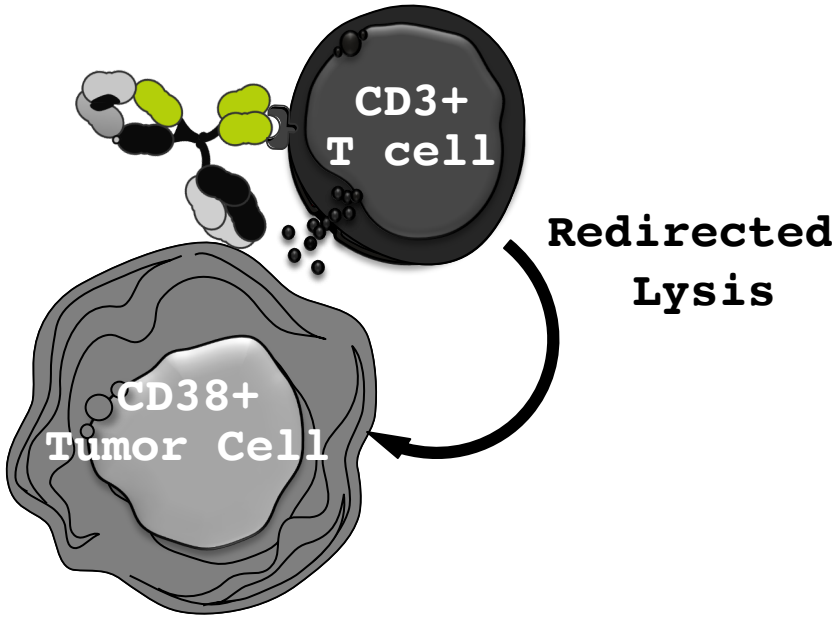
- Granted orphan drug designation in 2019
- Phase 1 dose escalation and expansion study, including weekly dosing, is ongoing

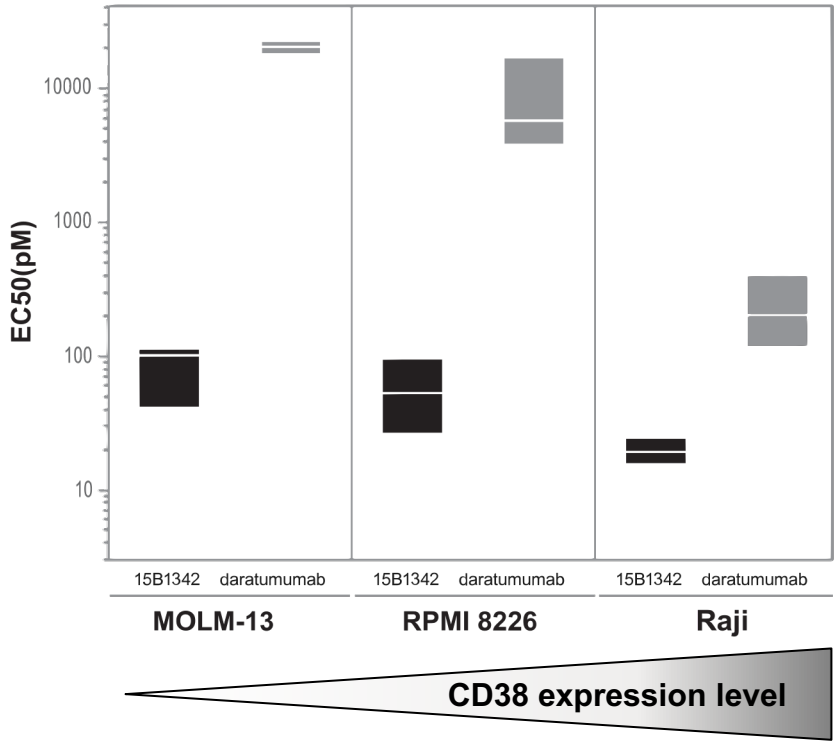
ISB 1342 (CD38 x CD3) bispecific antibody | **BEAT® 1.0**



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

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

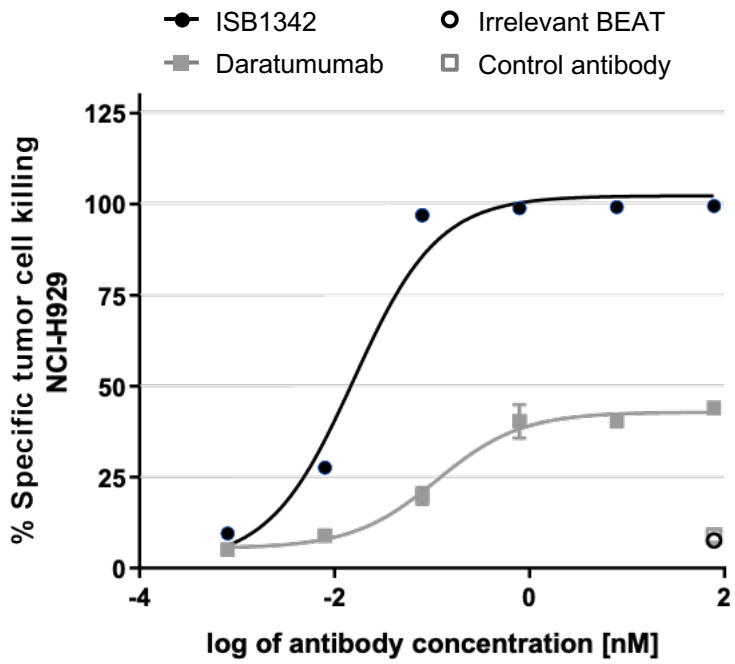


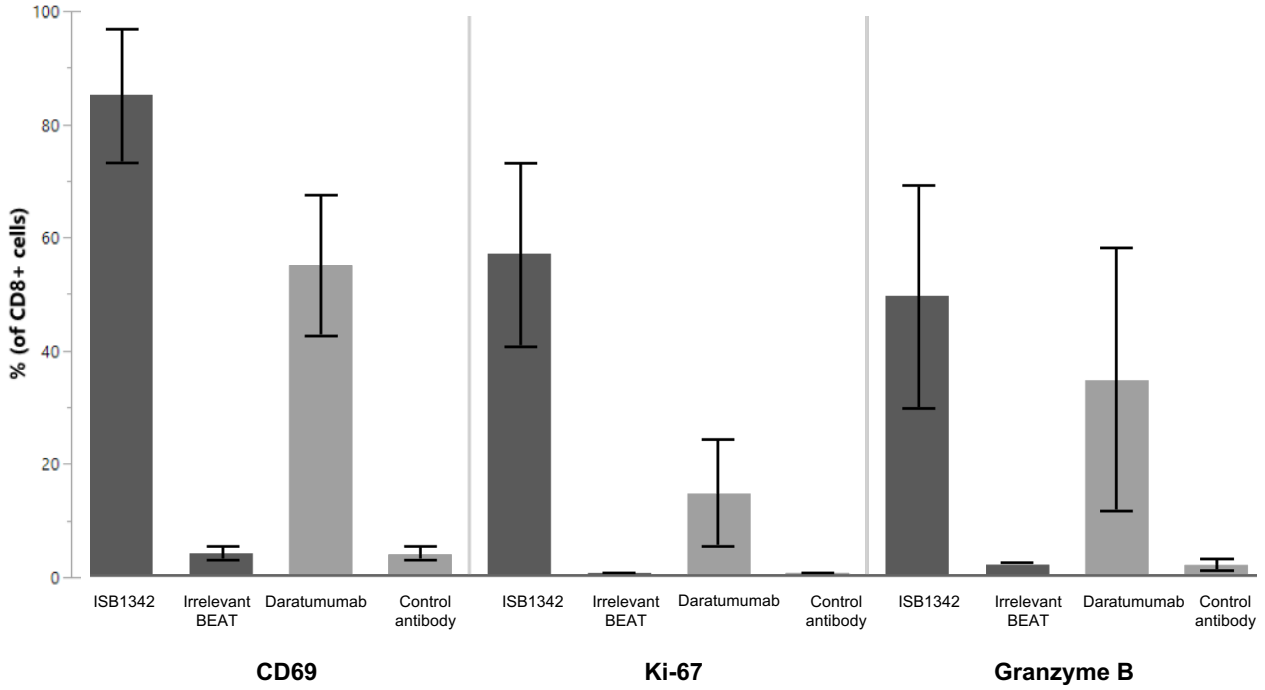


By co-engaging TCR/CD3ε on T lymphocytes and CD38 on tumor cells, ISB 1342 induces the formation of an immunological synapse between T cells and tumor cells and the redirected lysis of tumor cells (left panel). The potency of daratumumab and ISB 1342 to kill in vitro tumor cells expressing low, intermediate, and high levels of CD38 was compared in a multiple mode of action killing assay that combines Antibody-Dependent Cell-mediated Cytotoxicity (ADCC), Complement-Dependent Cytotoxicity (CDC), and redirected cell lysis (right panel).

Data on file. Clinical significance unknown.

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

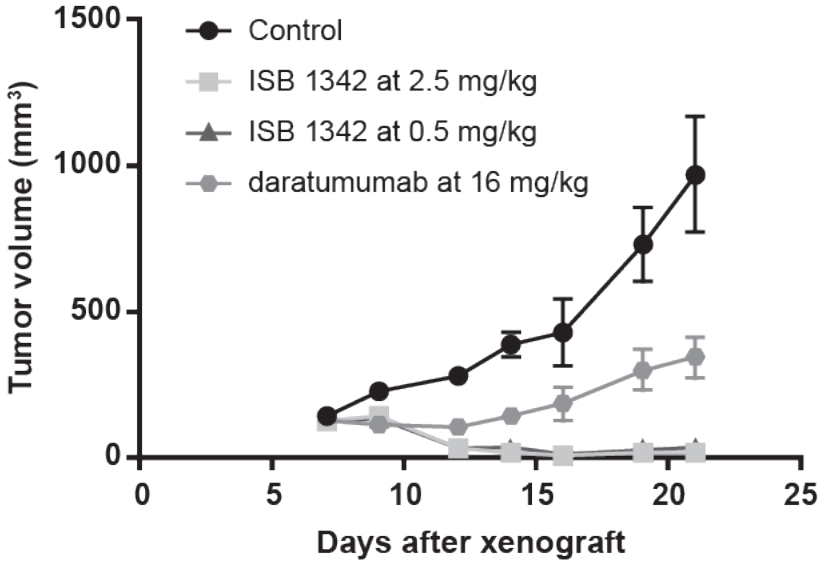


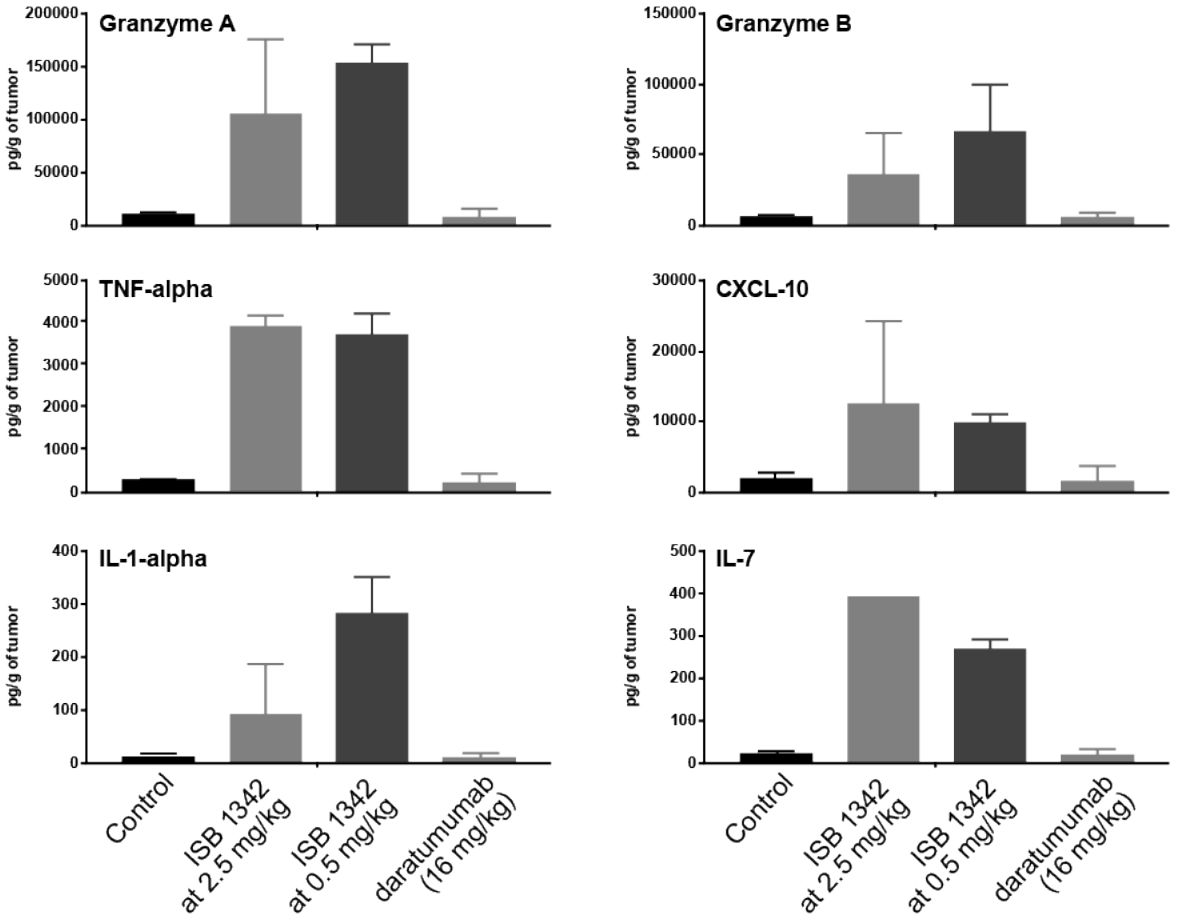


Potency of ISB 1342 and daratumumab to kill NCI-H929 cells (CD38 intermediate tumor cells) in vitro in a multiple mode of action killing assay (left panel). Specific tumor cell killing was measured at t=48h, isotype control antibody and irrelevant BEAT with dummy CD3 and CD38 binders were used as a negative 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).

Data on file. 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 mm³ and tumor growth monitored over two weeks (left panel). Soluble immune factors were quantified in dissociated tumors one week post-treatment (right panel).

Data on file. Clinical significance unknown.

ISB 1302

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ISB 1302(HER2 x CD3): Bispecific Antibody Positioned to Address Unmet Medical Needs in Metastatic HER2+ Breast Cancer

Key Attributes

- HER2 is expressed on the surface of numerous cancers, including breast cancer, and is the target of several highly effective therapies
- ISB 1302 is designed to redirect T cell mediated killing of HER2-expressing tumor cells in MHC-antigen independent manner
- ISB 1302 binds to the same HER2 epitope as trastuzumab
- ISB 1302 has demonstrated effective tumor killing in preclinical models. In vivo model to evaluate the impact of ISB 1302 on trastuzumab-resistant tumor lines is under development

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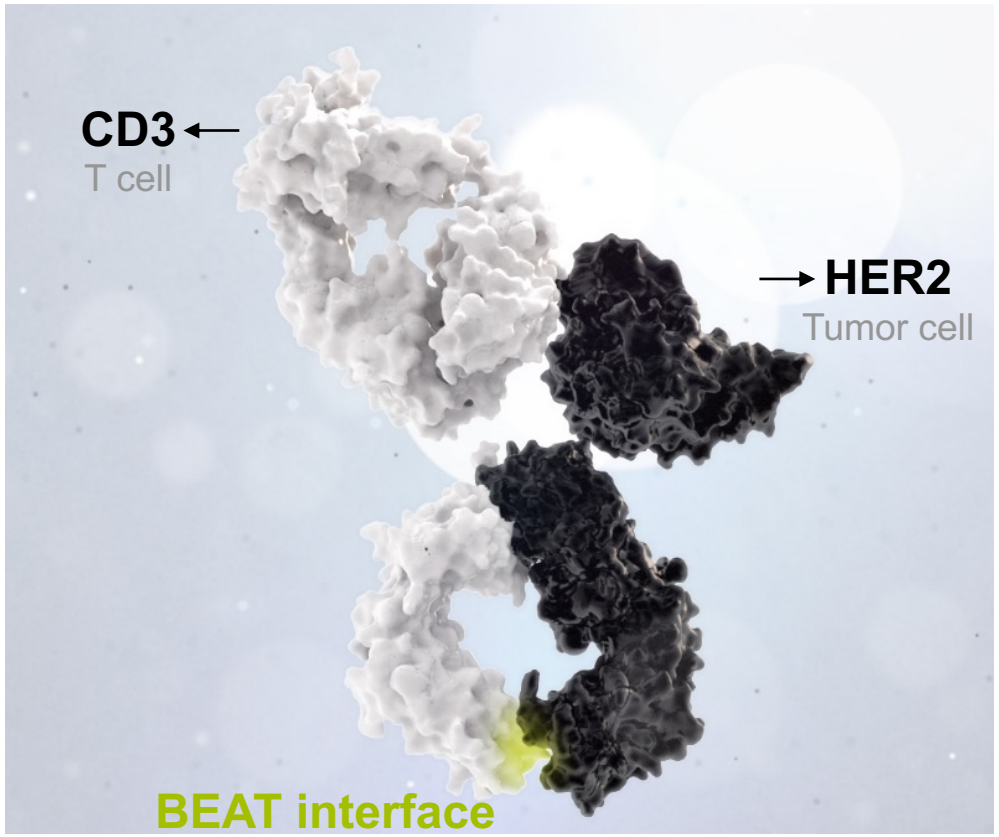
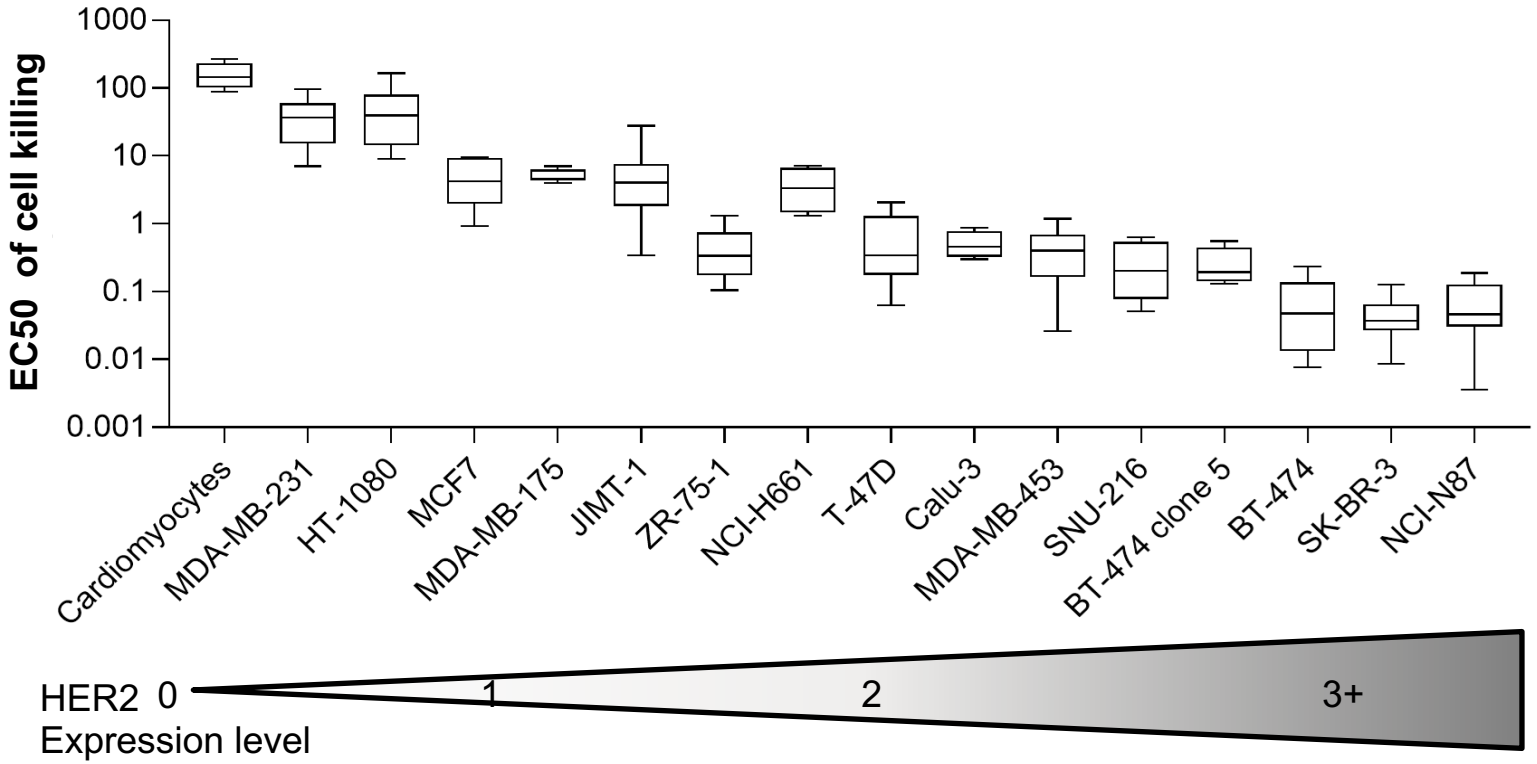
- Ongoing phase 1 trial in patients with HER2 metastatic breast cancer; dose escalation and expansion including weekly and biweekly dosing

ISB 1302 (HER2 x CD3) bispecific antibody | **BEAT® 1.0**



MHC: major histocompatibility complex

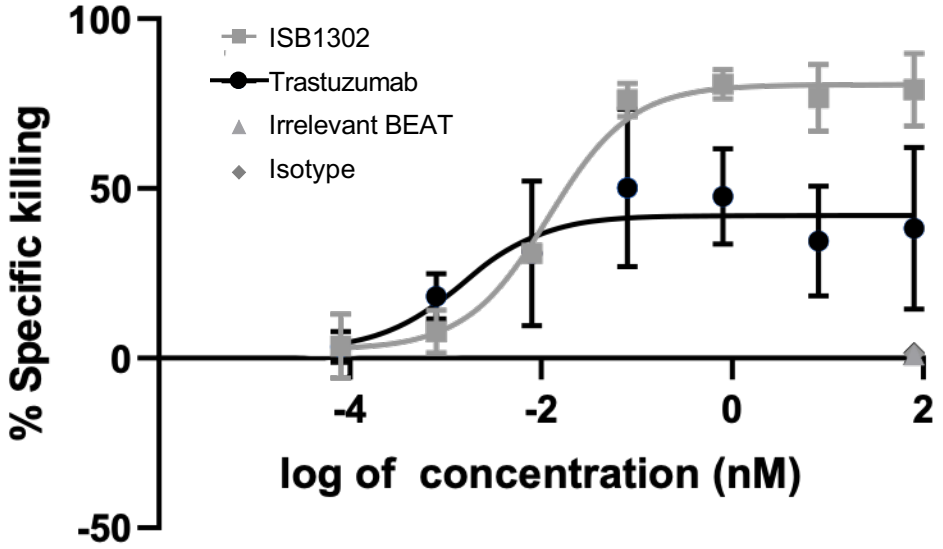
ISB 1302 Killing Potency In Vitro Is Associated With the Level of HER2 Expression on Target Cells

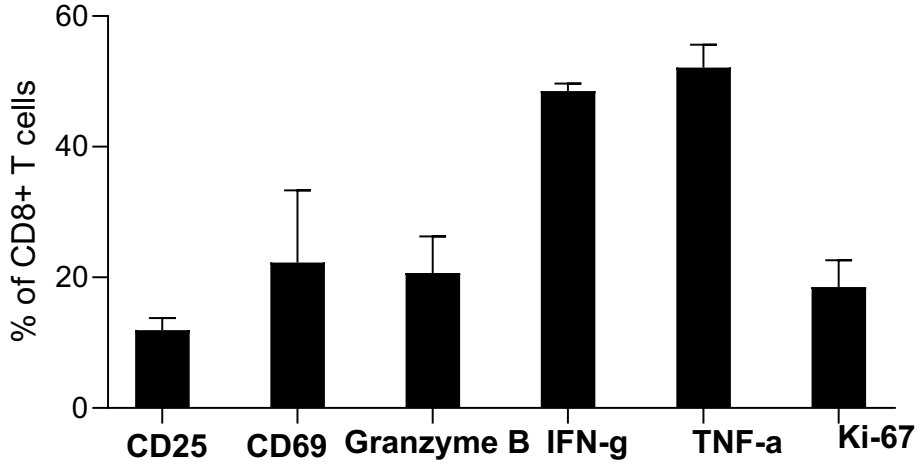


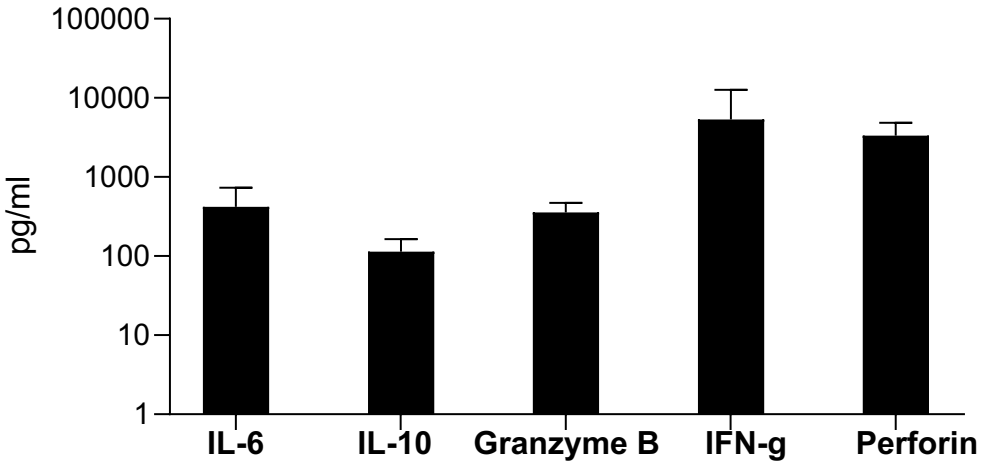
Redirected cell lysis assay performed in the presence of peripheral mononuclear cells and cardiomyocytes or various HER2-expressing tumor cells. EC50 of specific killing at t=72h is shown for each cell type.

Data on file. Clinical significance unknown.

ISB 1302 Kills Trastuzumab-Resistant Breast Cancer Tumor Cells In Vitro



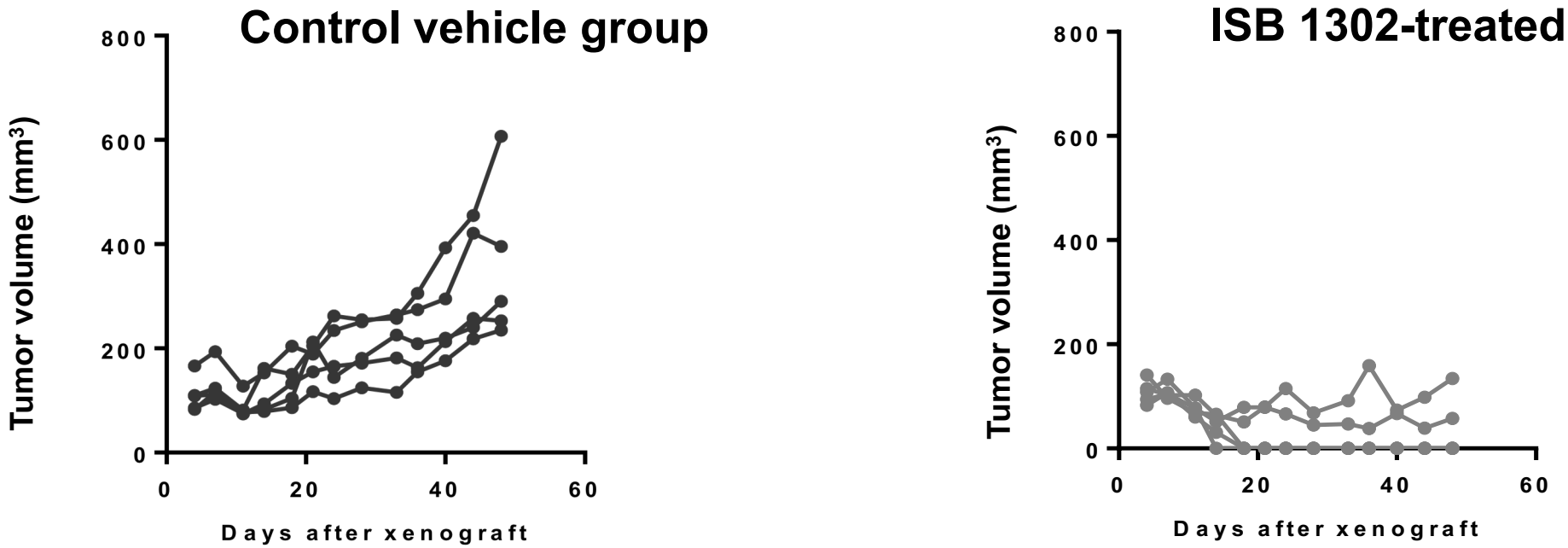




Redirected tumor cell lysis assay performed with peripheral blood mononuclear cells, BT-474.5 trastuzumab-resistant breast tumor cells, and increasing concentrations of ISB 1302 or trastuzumab (t=72h, left panel). Isotype control antibody and irrelevant BEAT with dummy CD3 and HER2 binders were used as negative controls. T cell activation (top right panel) and release of soluble cytokines and cytotoxic markers (bottom right panel) in response to maximum dose of ISB 1302 were quantified at t=72h.

Data on file. Clinical significance unknown.

ISB 1302 Efficiently Controls Tumor Growth In Vivo



NOD-SCID mice were xenografted subcutaneously with human peripheral blood mononuclear cells and NCI-N-87 cells. ISB 1302 was injected intravenously weekly at 0.05 mg/kg when tumor reached 100-200 mm^3. Tumor growth was monitored over 50 days (n=5 mice for control and treated group). One representative experiment out of two, with two distinct PBMC donors, is shown.

Data on file. Clinical significance unknown.

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 in 2021 and beyond
- 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 seven programs, including two clinical-stage T cell engager assets in multiple myeloma (ISB 1342) and metastatic HER2+ breast cancer (ISB 1302)
- Beyond oncology, pipeline of potential first-in-class therapeutics addressing autoimmune disease and pain: 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

Available for Out-License: Autoimmune, Pain & Oncology Small Molecules

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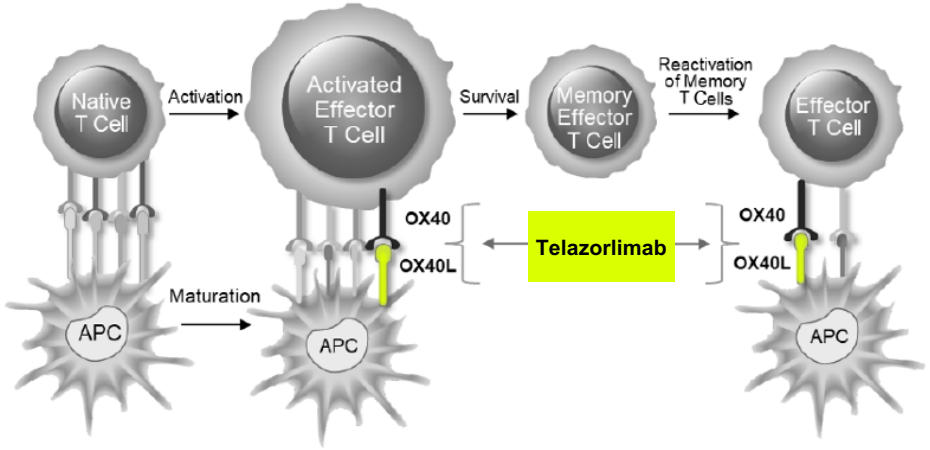
Telazorlimab (ISB 830): Potential First-in-Class OX40 Antagonist for Autoimmune Diseases

Key Attributes

- 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
- Results from Phase 2b study in moderate-to-severe atopic dermatitis
  - + Efficacy: The primary endpoint of EASI score, % change from baseline to Week 16, was achieved for the two highest doses tested (300 mg and 600 mg q 2 weeks) versus placebo. Numerical improvements were seen for the two higher-dose arms of telazorlimab compared to placebo in the secondary endpoints of EASI-75[1] and Investigator Global Assessment,[2] but the differences were generally not statistically significantly different from placebo
  - + Safety: The most commonly reported adverse events for telazorlimab (>5%) were: atopic dermatitis, nasopharyngitis, upper respiratory tract infection, and headache. There was one death due to pre-existing hypertension in a patient in the telazorlimab group. This was reported as unrelated to study drug by the investigator

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



- A US IND to conduct studies of telazorlimab in additional autoimmune diseases, including Rheumatoid Arthritis (RA), is active and Ichnos plans to out-license this asset for further development.

EASI: Eczema Area and Severity Index
1 Proportion of patients with ≥75% improvement in EASI score from baseline to Week 16
2 Proportion of patients with Investigator Global Assessment of clear or almost clear (0 or 1) and ≥2 point reduction from baseline at Week 16

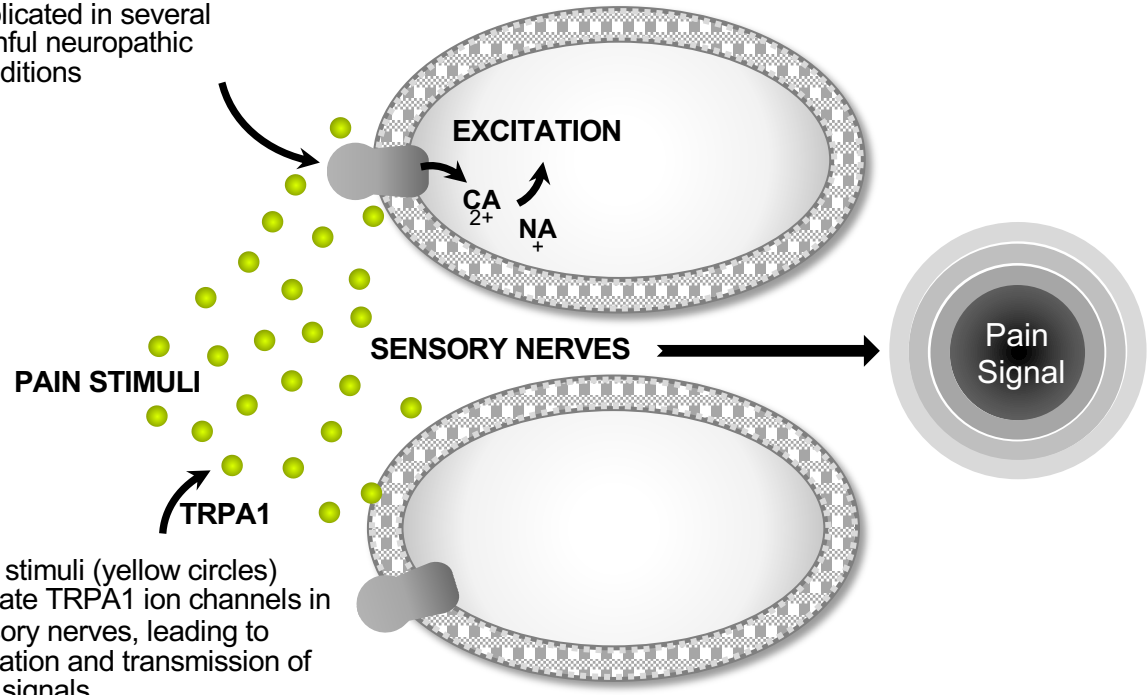
ISC 17536: Novel Transient Receptor Potential Ankyrin 1(TRPA1) Antagonist for Painful Diabetic Peripheral Neuropathy(DPN)

Key Attributes

- TRPA1 (transient receptor potential ankyrin 1) is a key ion channel responsible for pain sensation by small nerve fibers throughout the body
- ISC 17536 is a selective antagonist of TRPA1
- Completed a randomized, placebo-controlled, dose-range-finding phase 2a proof-of-concept trial in patients with painful diabetic peripheral neuropathy:
  - + primary endpoint was not met in the overall study population
  - + clinically and statistically significant reduction in pain was achieved in a prespecified subpopulation of patients with preserved small nerve fiber function
- All rights and oversight of future development of ISC 17536 are being transferred to Ichnos' parent company, Glenmark. Future out-licensing activities for the product will be conducted by Glenmark Business Development.

ISC 17536: An Oral, TRPA1 Antagonist

- TRPA1 is a receptor on small nerve fibers
- Implicated in several painful neuropathic conditions



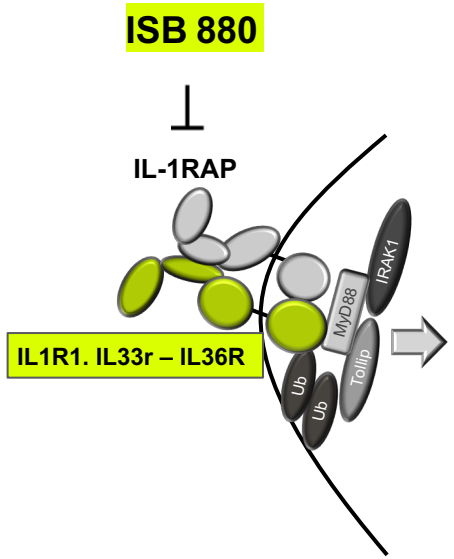
Pain stimuli (yellow circles) activate TRPA1 ion channels in sensory nerves, leading to excitation and transmission of pain signals

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, LaLa mutated Fc)
- 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

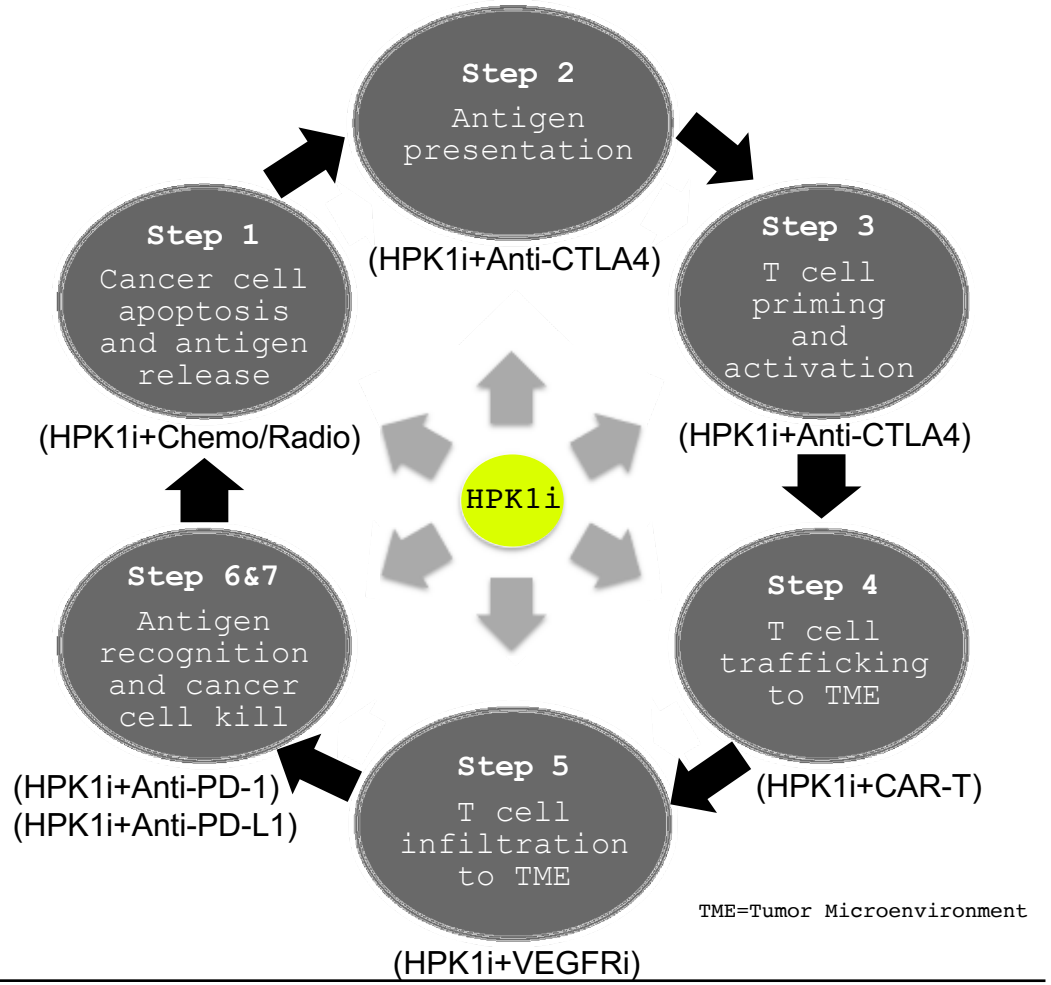
Potential Indications

- Inflammatory Diseases
- Oncology

ISC XXXXX: HPK1 Inhibition Role in Cancer Immune Cycle

Key Attributes

- Hematopoietic Progenitor Kinase 1 (HPK1, MAP4k1)[1,2] Inhibitor is a negative regulator of TCR and BCR[3]
- Immunosuppressive mediators of PGE2 and adenosine regulate HPK1 activation[3]
- In vivo anti-tumor activity by HPK1 gene deletion, kinase dead HPK1 and small molecule inhibitors has been demonstrated in multiple immunogenic syngeneic tumor models[3,4]
- Enhanced anti-tumor efficacy by combining HPK1 inhibition with checkpoint inhibitors (CPIs) like anti-PD-1, anti-PD-L1, or anti-CTLA4 antibodies[4]
- HPK1 is a target for immuno-oncology treatment in cancers responsive or non-responsive to current CPIs



TME=Tumor Microenvironment

1. F.Kiefer et al., *The EMBO Journal* 1996
2. Hu et al., *Genes and Development* 1996
3. Sawasdikosol and Burakoff. *eLife* 2020;9:e55122
4. AACR Annual Meeting Virtual Meeting II June 22-24 2020

FROM NOW ON

...ichnos...

Thank you

...ichnos...