UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT

Pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934

Date of Report (date of earliest event reported): **April 8, 2025**

iBio, Inc.

(Exact name of registrant as specified in charter)

Delaware
(State or other jurisdiction of incorporation)

001-35023	**26-2797813**
(Commission File Number)	*(IRS Employer Identification No.)*

11750 Sorrento Valley Road, Suite 200
San Diego, California 92121
(Address of principal executive offices and zip code)

(979) 446-0027
(Registrant's telephone number including area code)

N/A
(Former Name and Former Address)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12(b) under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, $0.001 par value per share	IBIO	The Nasdaq Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by checkmark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01. Regulation FD Disclosure.

iBio, Inc. (the "Company") has updated its corporate presentation. A copy of the updated corporate presentation is furnished as Exhibit 99.1 to this Current Report on Form 8-K.

The information in this Item 7.01 and in the corporate presentation attached as Exhibit 99.1 to this Current Report on Form 8-K shall not be deemed to be "filed" for purposes of Section 18 of the Securities Act of 1934, as amended, or otherwise subject to the liabilities of that section or Sections 11 and 12(a)(2) of the Securities Act of 1933, as amended. The information contained in this Item 7.01 and in the corporate presentation attached as Exhibit 99.1 to this Current Report on Form 8-K shall not be incorporated by reference into any filing with the Securities and Exchange Commission made by the Company, whether made before or after the date hereof, regardless of any general incorporation language in such filing.

The corporate presentation attached as Exhibit 99.1 to this Current Report on Form 8-K includes "safe harbor" language pursuant to the Private Securities Litigation Reform Act of 1995, as amended, indicating that certain statements contained therein are "forward-looking" rather than historical.

The Company undertakes no duty or obligation to update or revise the information contained in this Current Report on Form 8-K, although it may do so from time to time if its management believes it is appropriate. Any such updating may be made through the filing of other reports or documents with the Securities and Exchange Commission, through press releases or through other public disclosures.

Item 8.01. Other Events.

The Company has updated its corporate presentation, a copy of which is attached as Exhibit 99.1 to this Current Report on Form 8-K, for use in meetings with investors, analysts and others. The information on slides 16, 17, 18, 19, 20, 25, and 26 of Exhibit 99.1 is incorporated by reference herein.

The non-human primate data from a non-human primate study of IBIO-600, the Company's long-acting anti-myostatin antibody, showed extended half-life and muscle growth. The results indicate IBIO-600 promoted a dose-dependent increase in lean mass and a reduction in fat mass from baseline values. Standard PK calculations indicated the half-life of IBIO-600 in non-human primates was 40 to 52 days. Non-human primate pharmacokinetics data suggests IBIO-600, a potentially best-in-class long-acting anti-myostatin antibody, could have a human half-life as long as 130 days.

Preclinical data for a first-in-class Activin E antibody disclosed in January, showed that the antibody effectively blocks Activin E signaling in human adipocytes and is currently being evaluated in an exploratory study with obese mice, both as a monotherapy with bi-weekly dosing and in combination with semaglutide dosed daily. After only two weeks of dosing, monotherapy resulted in fat-selective weight loss of approximately 4%, with a significant 18% reduction in total body fat compared to placebo. Notably, when combined with semaglutide, the Activin E antibody demonstrated a strong synergistic effect, enhancing total weight loss by an additional 9% beyond GLP-1 therapy alone, leading to an overall weight reduction of 34%. This combination also resulted in a remarkable 72% reduction in body fat over the treatment period, as measured by DEXA scans.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit No.	**Description**
99.1	Corporate Presentation of iBio, Inc., dated April 2025
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURES

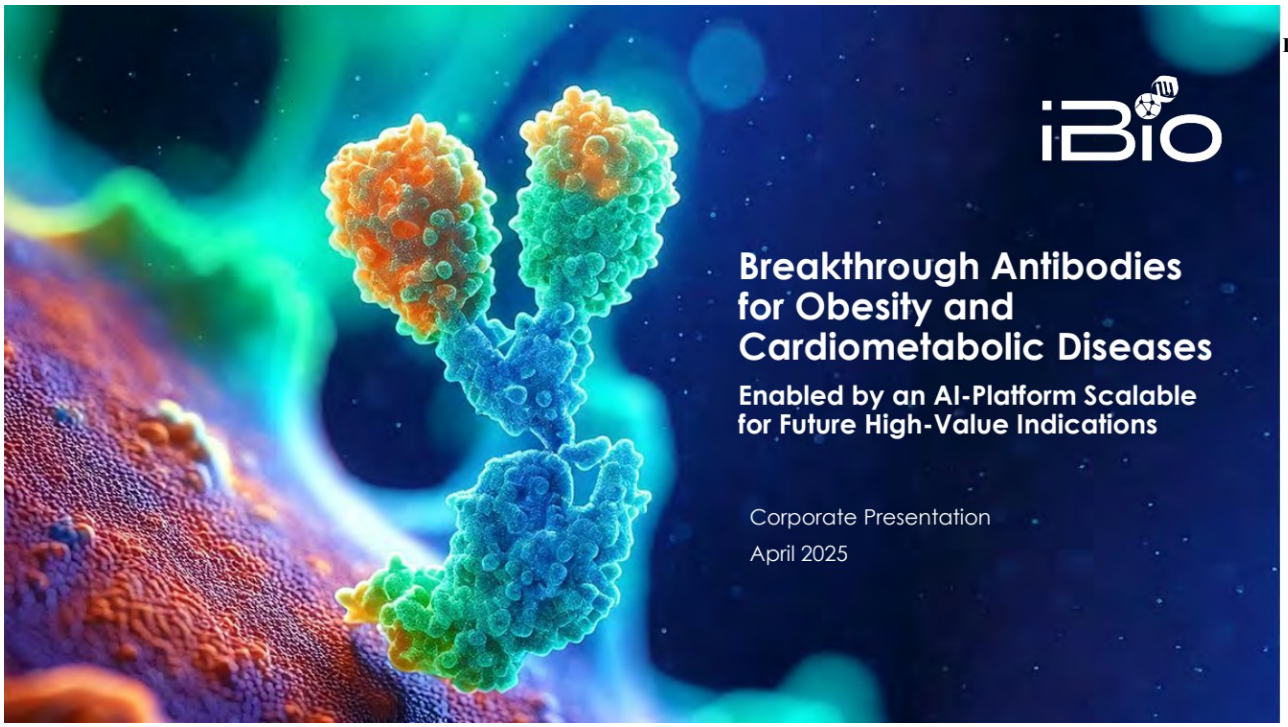
Pursuant to the requirements of the Securities Exchange Act of 1934, as amended, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Date: April 8, 2025

IBIO, INC.

By: /s/ Marc A. Banjak
Name: Marc A. Banjak
Title: Chief Legal Officer

Exhibit 99.1

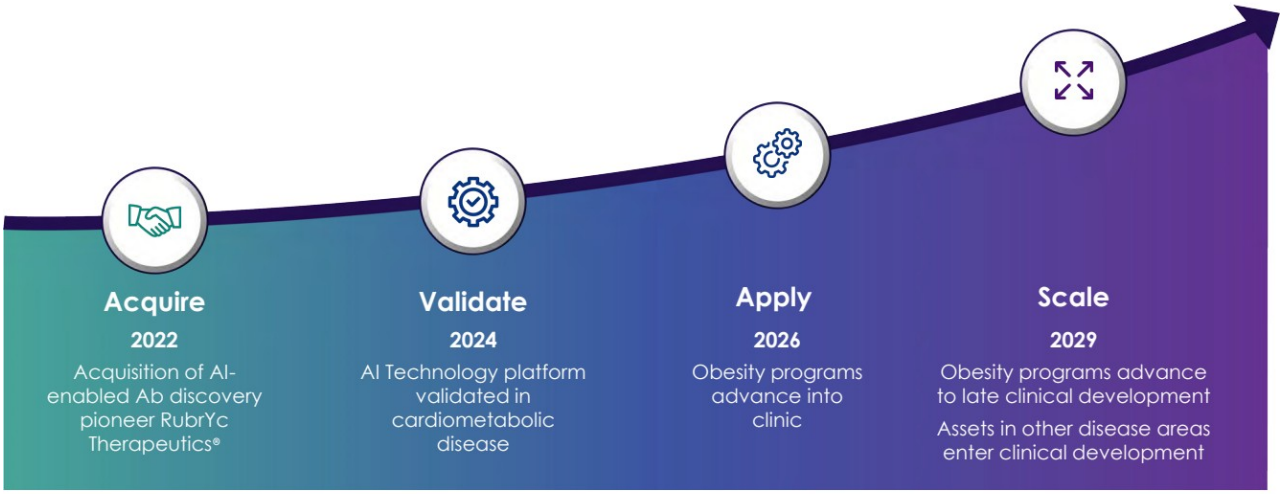

iBio
Breakthrough Antibodies for Obesity and Cardiometabolic Diseases
Enabled by an AI-Platform Scalable for Future High-Value Indications
Corporate Presentation
April 2025

Forward looking statements

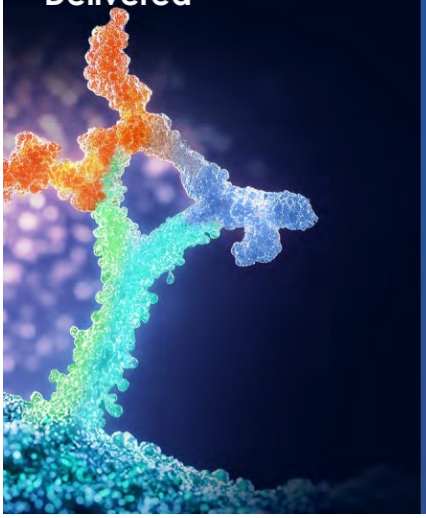
Certain statements in this presentation constitute "forward-looking statements" within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. Words such as "may," "might," "will," "should," "believe," "expect," "anticipate," "estimate," "continue," "predict," "forecast," "project," "plan," "intend" or similar expressions, or statements regarding intent, belief, or current expectations, are forward-looking statements. These forward-looking statements are based upon current estimates and includes statements regarding near term catalysts. While iBio, Inc., a Delaware corporation (including its consolidated subsidiaries, "iBio," the "Company," "we," "us" or "our") believes these forward-looking statements are reasonable, undue reliance should not be placed on any such forward-looking statements, which are based on information available to us on the date of this presentation. These forward-looking statements are subject to various risks and uncertainties, many of which are difficult to predict that could cause actual results to differ materially from current expectations and assumptions from those set forth or implied by any forward-looking statements. Important factors that could cause actual results to differ materially from current expectations include, among others, the Company's ability to obtain regulatory approvals for commercialization of its product candidates, or to comply with ongoing regulatory requirements, regulatory limitations relating to its ability to promote or commercialize its product candidates for specific indications, acceptance of its product candidates in the marketplace and the successful development, marketing or sale of products, its ability to attain license agreements, the continued maintenance and growth of its patent estate, its ability to establish and maintain collaborations, its ability to obtain or maintain the capital or grants necessary to fund its research and development activities, competition, its ability to retain its key employees or maintain its NYSE American listing, and the other factors discussed in the Company's most recent Annual Report on Form 10-K and the Company's subsequent filings with the SEC, including subsequent periodic reports on Forms 10-Q and 8-K. The information in this presentation is provided only as of today, and we undertake no obligation to update any forward-looking statements contained in this presentation on account of new information, future events, or otherwise, except as required by law.

An Antibody (Ab) Discovery Platform for Cardiometabolic Disease and Beyond

Advancing Antibodies Against Difficult Targets With an Integrated and Validated AI Technology Stack



iBio's AI-Driven Antibody Discovery Platform Has Delivered



Corporate Highlights

- AI-driven antibody discovery Platform including patented ***Epitope steering, StableHu™, EngageTx™, and ShieldTx®***
- Platform has delivered Development Candidates in as little as 7 months
- 11 active programs:

5 Cardiometabolic/Obesity programs - 3 of which are partnered - demonstrating the value of our approach

6 in-house pre-clinical programs in immuno-oncology

Near Term Catalyst

2025

- Long-acting anti-Myostatin program; IND by Q4 '25/Q1'26

2026

- Ph 1 trial for long-acting anti-Myostatin program initiated by 2H '26
- Additional IND by 2H '26

Any Epitope on Any Drug Target

AI Epitope Engineering and Antibody Optimization Engines unlock challenging target classes

iBio's Discovery Engine

We use our Tech Stack to generate new IP against **hard-to-drug targets** – from **idea to Development Candidate** in **7 months**

iBio's Proprietary AI Technology Platform

- **Multi-layer technology platform** addresses multiple challenges in Ab discovery
- **Patented Epitope Steering** technology
- **Single-step Ab** StableHu x Mammalian Display
- **Masked** (ShieldTx) Antibodies
- **T-cell engager panel** (EngageTx)

AI-guided precision hits that are epitope class agnostic

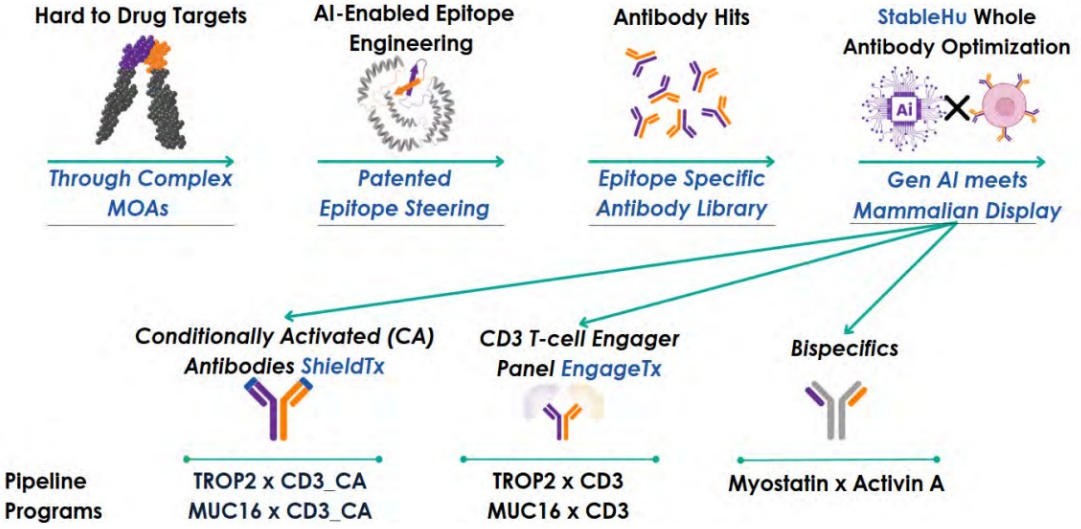
- Selectively targets **functional epitopes**
- Epitopes with **complex modes of action**
- Unlocks **novel target** classes
- Accelerates discovery of Ab against **validated targets**

Generative AI meets mammalian display: Ab optimization in 3 weeks

- Gen AI creates **mammalian display** libraries with phage-like diversity
- Single-shot **multidimensional lead optimization**
- Compatible with **multi-specific** antibody formats
- Antibody **format agnostic**

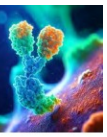
iBio's AI Tech Stack is a Fully Integrated Solution for Antibody Discovery

Enables and accelerates Antibody Discovery & Development Against Hard-to-Drug Targets




Harnessing the Power of
Our Platform to Address
Challenges in Cardio-
metabolic Disease

iBio's Rapidly Advancing High Value Obesity and Cardiometabolic Pipeline



Therapeutic Area	Program	Early Discovery	Late Discovery	Lead Optimization	IND-Enabling
Cardio-metabolic	**IBIO-600** Myostatin (obesity)				
	Myostatin x Activin A (obesity/ potentially PH-HFpEF)				
	Activin E (obesity)				
	Target 3 (obesity)			Partnered with ASTRALBIO	
	Target 4 (obesity)				

Current Treatments in Obesity Fall Short

Significant Unmet Medical Need for Innovation to Improve Overall Metabolic Health and Function

iBio's AI-enabled platform is addressing the challenges of current anti-obesity medicines

The Harvard Gazette

Losing fat is good, but losing muscle isn't

Researchers call for makers of new anti-obesity drugs to study results of body composition in addition to weight loss

nature biotechnology

News | Published: 05 March 2024

After obesity drugs' success, companies rush to preserve skeletal muscle

BioSpace

What's Next for Obesity Therapeutics? Higher Quality Weight Loss

Mercer

GLP- 1 discontinuation affirms need for holistic weight-loss plan

The Next Generation

Potential Avenues:

- **Preservation of muscle mass** during GLP-1 agonist induced weight loss
- **Improved fat burning** and prevention of dyslipidemia
- Improved cardiac function and **treatment of cardiovascular disease** related to metabolic syndrome

A Clear Strategy to Create a High-Value Pipeline of Differentiated Products

A prime opportunity exists for GLP-1 complementary therapeutic approaches

Address Challenges With Current GLP-1 Drugs

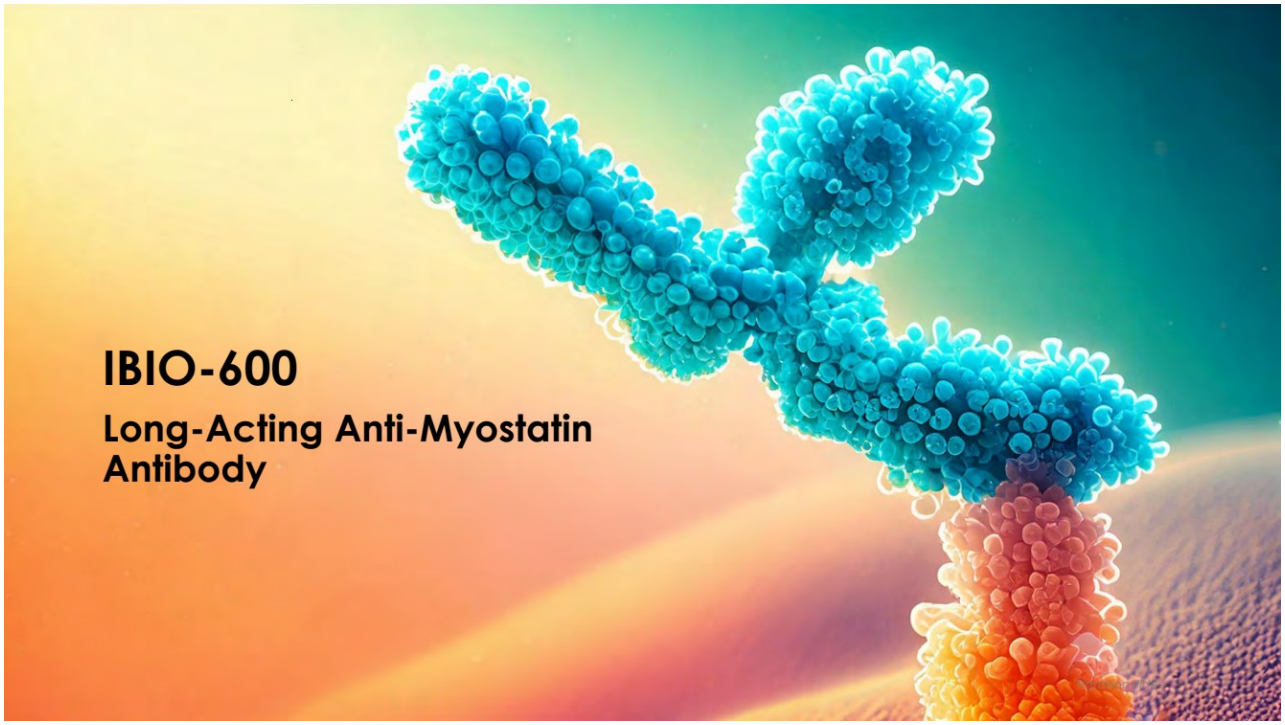
- **Muscle** mass **loss**
- **Side effects** leading to discontinuation
- Inconvenient **dosing frequency**
- Room for **high quality** weight loss

Focus on Highly Validated Targets

- **Preserve** and **build** muscle mass
- **Fat-specific** weight reduction
- Targeting both sides of the equation, **calorie intake** and **energy expenditure**

iBio's Platform Fuels a High-Value Pipeline

- Tackling **complex**, hard to drug targets
- Optimizing **function** and **developability** simultaneously
- Rapidly optimizing **multi-specifics**


IBIO-600
Long-Acting Anti-Myostatin
Antibody

Myostatin Antagonism

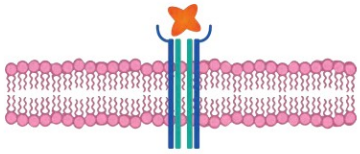
Enhancing the Quality of Weight Loss by Maintaining Muscle Mass During and After Weight Loss with GLP-1s

We are developing Myostatin inhibitors to **preserve and increase muscle mass**, **complementary to** treatment with **GLP-1 drugs**

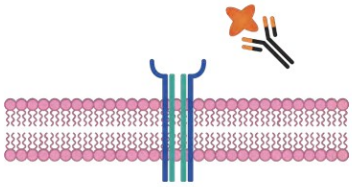
Why We Target Myostatin

- **Incretin drugs** reduce caloric intake, causing **weight loss in both fat and muscle**
- Myostatin is a **highly validated key negative regulator** of muscle mass[1]
- Inhibition of Myostatin function drives significant **muscle growth without** apparent **adverse health effects**
- Beyond its effects on muscle, Myostatin plays a role in the **regulation of total body fat mass**[2]

Binding of Myostatin to cells leads to **muscle atrophy**



Blocking of Myostatin leads to **muscle growth**



1. Schuelke M. (2004). New England Journal of Medicine 350(2682–2688).
2. Deng, B. (2017). Nutrition and Metabolism, 14(29).

IBIO-600: A Long-Acting First-in-Class Anti-Myostatin Antibody

First Anti-Myostatin Antibody With a Target Product Profile Specifically Tailored for an Obese Patient Population

IBIO-600

Long-Acting Anti-Myostatin Antibody

First-in-class innovation: First Myostatin therapy tailored for large, chronic disease populations

Convenient Dosing: Half-life extension anticipated to support dosing every 2-3 months

Broad Potential: Opportunities for expansion into sarcopenia, frailty, and other age-related disorders

Highly Developable: Resistant to various stress conditions, improved expression, high thermostability[1]

Target product profile characteristics for obese patients

- Well-tolerated for long-term use
- Infrequent subcutaneous self-administration

AI-enabled CDR design

- Rapidly generates novel IP
- Large library of novel lead molecules

Single-shot multi-dimensional lead optimization

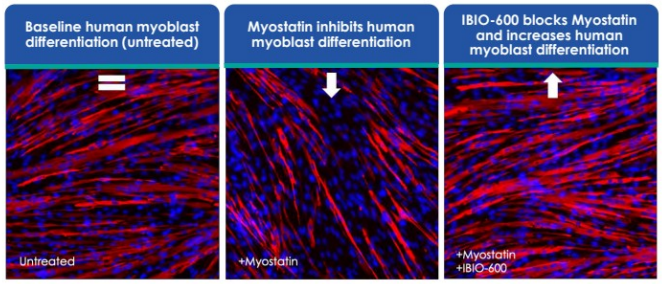
- Optimized for affinity, half-life and manufacturability

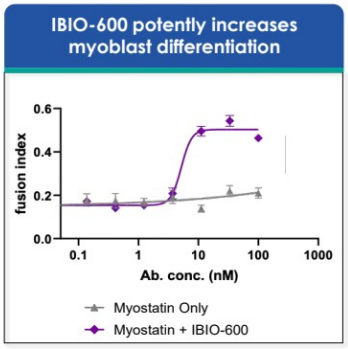
1. Data on file

IBIO-600 Increases Muscle Differentiation in Primary Human Myoblast Cells

Red indicates muscle cell growth and development (as measured by myoblast differentiation)

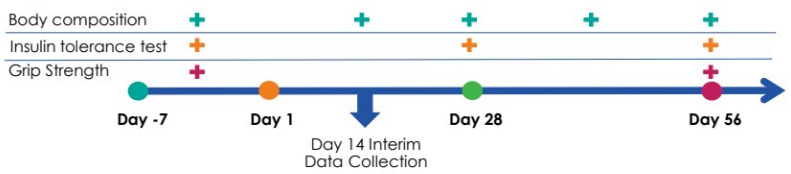
The **human Myoblast differentiation** model is **highly predictive** of **muscle growth** in humans





Interim Data: IBIO-600 Preserves Muscle Mass in GLP-1 Treated Diet Induced Obesity Mice

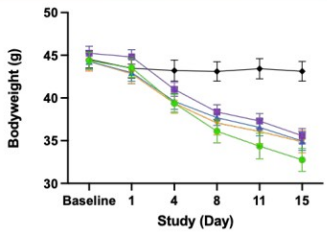
Study Design



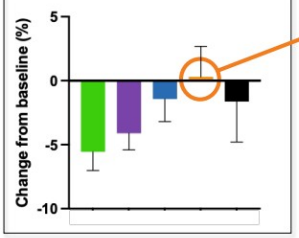
Interim Data

- Sema only
- Sema + 1mg/kg IBIO-600
- Sema + 3mg/kg IBIO-600
- Sema + 10mg/kg IBIO-600
- No treatment

Body weight loss up to day 14



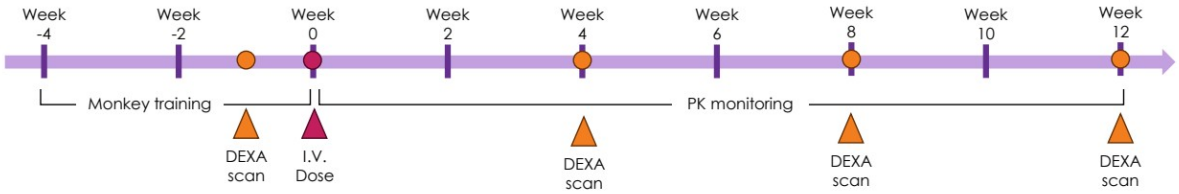
Lean mass change on Day 14



Sema + 10 mg/kg IBIO-600

In this study the murine surrogate antibody of IBIO-600 was used

IBIO-600 Pharmacokinetics (PK) Study in Non-Human Primates (NHPs)



Study details

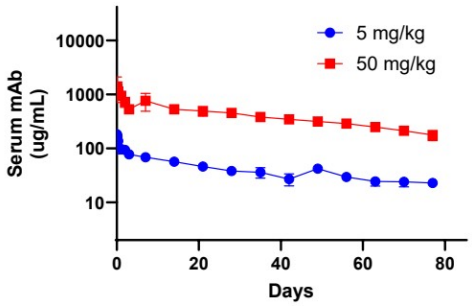
- Obese, aged NHPs
- Performed at Kunming Biomed International (KBI)
- Material produced transiently by Wuxi Biologics

Study design

- N=3 per group
- 2 arms, single I.V. dose
  - 5mg/kg
  - 50 mg/kg
- DEXA scan for body composition every 4 weeks
- Periodic PK sampling

IBIO-600 Fc Engineering Drives Extended Half-Life in Obese NHPs

12 Week Pharmacokinetics Data[1]



IBIO-600 Fc engineering results in enhanced FcRn binding

Clone	Fc	Fold increase over standard IgG
IBIO-600 FAB	Standard IgG4	**1.0**
IBIO-600	Engineered IgG4	**16.5**

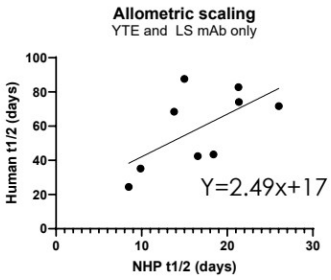
IBIO-600 demonstrates extended half-life in NHPs

Dose	$t_{1/2}$ (days)
5 mg/kg	**52.4**
50 mg/kg	**40.7**

1. Linear elimination phase used to estimate half-life with simple linear model

Allometric Scaling Suggests Meaningfully Extended Half-Life for IBIO-600 in Humans

Allometric scaling model for half-life extended antibodies[1]



Generic allometric scaling model for antibodies[2]

$$T_{1/2Human} = T_{1/2NHP} \times \left[\frac{\text{Human Body Weight}}{\text{NHP Body Weight}}\right]^{0.15}$$

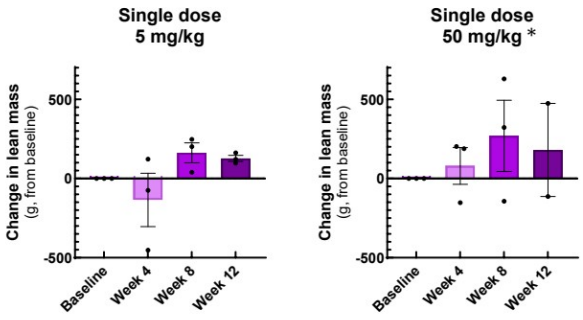
Measured NHP and predicted human half-life of IBIO-600

Dose	NHP $t_{1/2}$ (actual)	Human $t_{1/2}$ (predicted)[1,2]
5 mg/kg	52.4	**74-130 days**
50 mg/kg	40.7	**57-101 days**

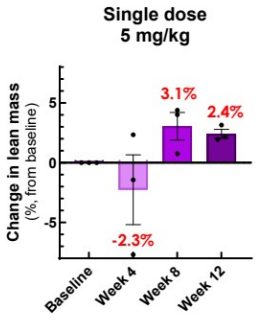
1 https://pmc.ncbi.nlm.nih.gov/articles/PMC9709760/#CR37
2 https://www.jstage.jst.go.jp/article/bpb/43/5/43_b19-01042/_html/-char/en

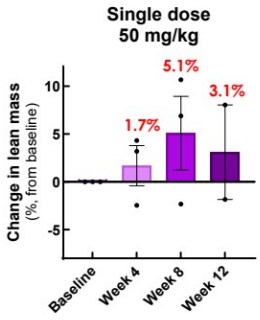
Lean Mass Peaks at 8 Weeks Remains Elevated at 12 Weeks After a Single IBIO-600 i.v. Injection

Lean Mass Increase in Grams

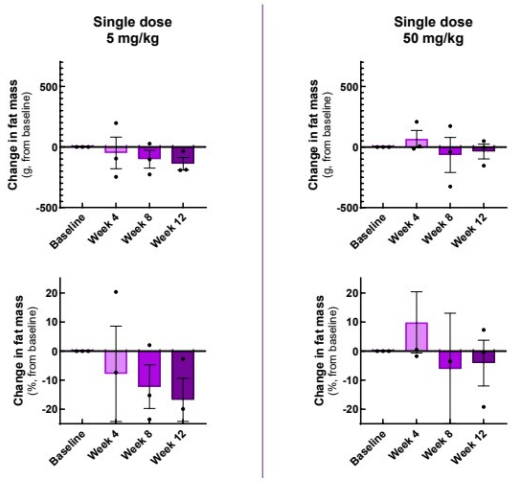


Lean Mass Increase in Percent

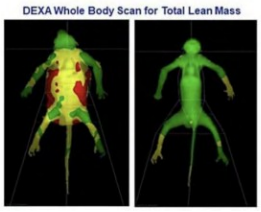


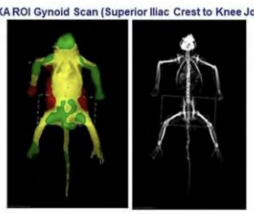


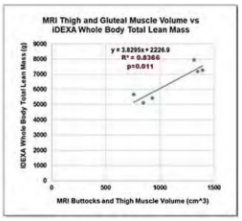
Fat Mass between Thigh and Abdomen is Reduced After a Single I.V. Dose of IBIO-600

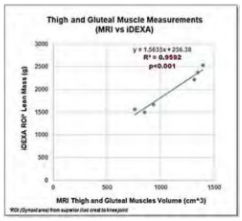


Region of Interest (ROI) DEXA Analysis of Gluteal and Thigh Regions Correlates Better with MRI Data[1]

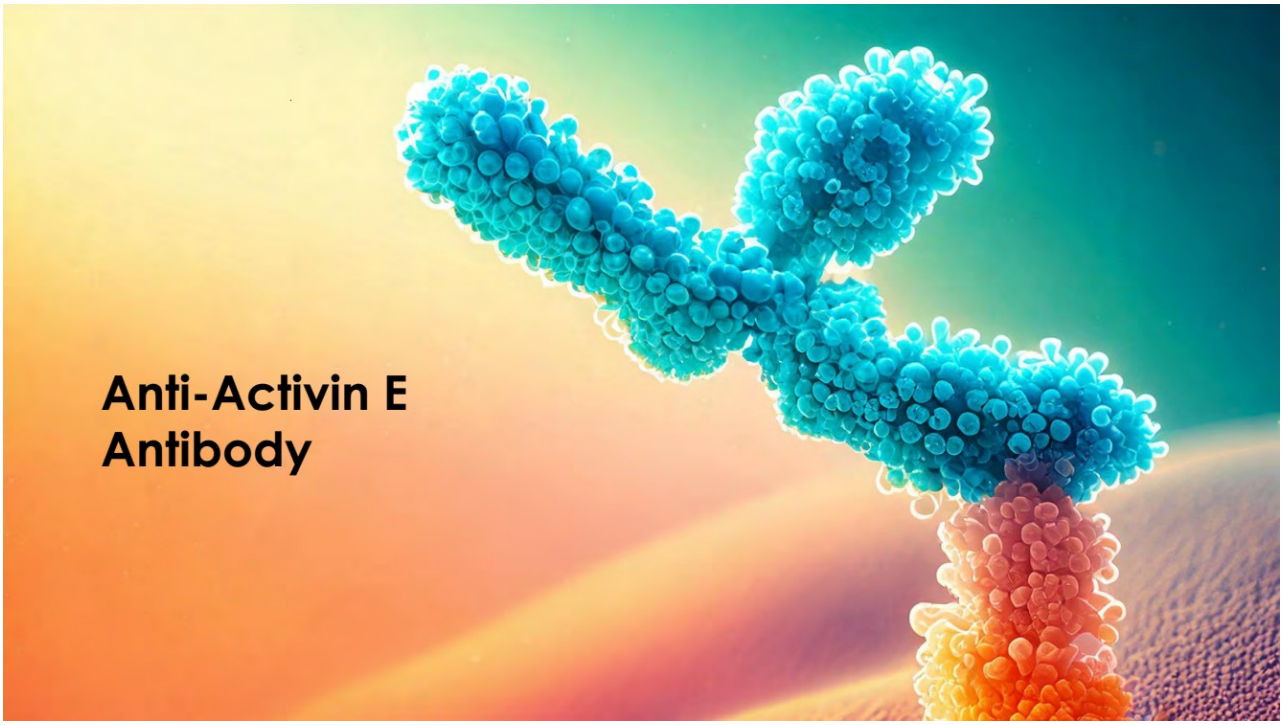








1. Data on file


Anti-Activin E
Antibody

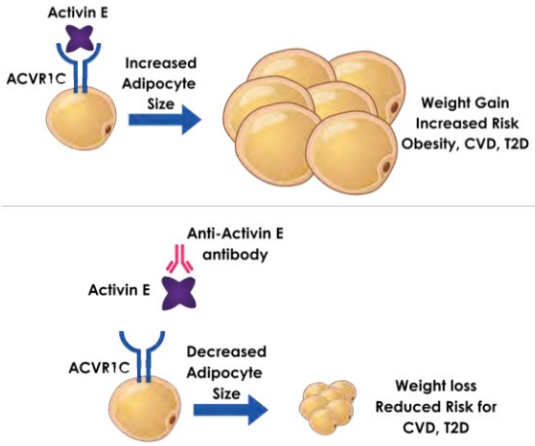
Activin E Antagonism

Attractive Fat-Specific Weight Loss Mechanism with Excellent Compatibility for Bi-Specific Pairing with Anti-Myostatin

We are developing **inhibitors of Activin E** to **promote fat-specific weight loss**, either as a standalone drug or as a bi-specific antibody with Myostatin.

Why We Target Activin E

- Activin E is a Hepatokine, produced in the liver and a member of the TGFβ family
- Activin E and its receptor are highly genetically validated
- Genetic loss of function decreases adiposity and risk for Diabetes / Cardiovascular Disease (CVD)
- **2 RNA targeting molecules provide preclinical pharmacological validation**
- Challenge to produce active recombinant Activin E until recently has proven to be extremely difficult for antibody discovery



iBio's AI-Enabled Epitope Steering Engine Bypasses Recombinant Activin E, Creating Functional Antibodies Directly from the Target Sequence

Activin E Antibody

Innovative AI solution: Epitope steering engine overcame the challenge of full-length Activin E unavailability, creating a first-in-class antibody targeting Activin E

Convenient Dosing: Half-life extension potentially enables dosing every 2-3 months

Versatile Combinability: Easily integrates with other TGFβ family targets into bi-specific antibodies, offering a potential alternative to incretin drugs (fat-specific weight loss with increase in muscle mass)

AI epitope engineering breaks barrier to discovery

- First-in-class functional antibody for Activin E

AI-enabled CDR design

- Rapidly generates novel IP
- Large library of novel lead molecules

Single-shot multi-dimensional lead optimization

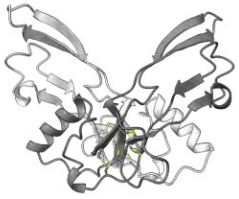
- Optimized for affinity, half-life and manufacturability

iBio's AI Engineered Epitope Engine Delivers a First-in-Class Functional Activin E Antibody

We have **uniquely solved an industry-wide problem** with our proprietary epitope engineering engine to create functional Activin E antibodies

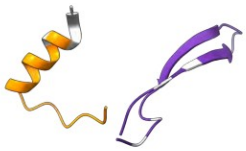
Challenge

Active, native recombinant **Activin E unavailable until recently**



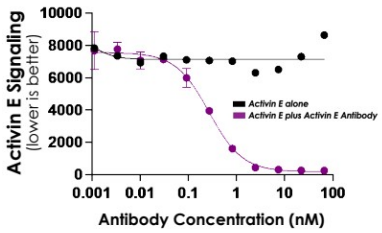
iBio's Solution

Create Engineered Epitopes to guide Antibodies against full-length Activin E



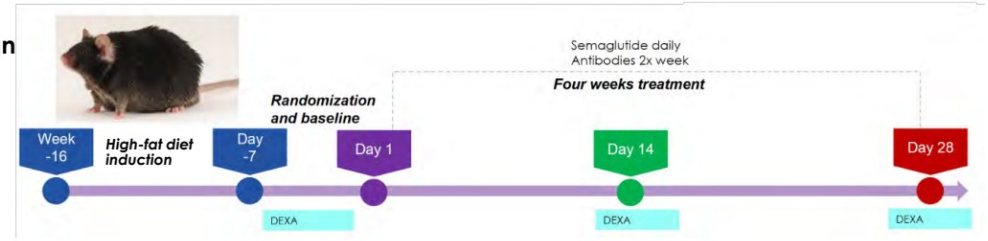
Breakthrough

First-in-Class **Activin E Antibody Blocks** Activin E **Signaling**



Preclinical Study: Activin E Antibody in Combination With GLP-1 in Diet-Induced Obese Mice

Study Design



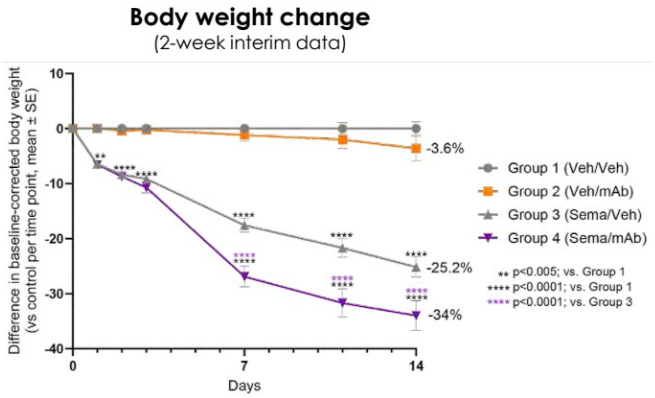
Treatment Arms

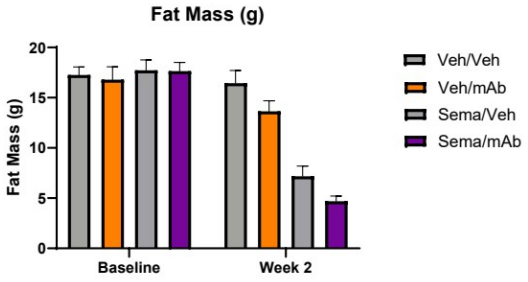
- Group 1: Vehicle / Vehicle
- Group 2: Vehicle / iBio Activin E mAb
- Group 3: Semaglutide / Vehicle
- Group 4: Semaglutide / iBio Activin E mAb

Study Details

- Activin E mAb: Mouse IgG1, human VH and VL
- Semaglutide: 40ug/kg (mimics human dose), daily
- iBio Activin E mAb: 10mg/kg, 2x/week
- Body composition (DEXA) and multiple terminal endpoints
- Ten mice per group

2 Week Interim Data: Activin E Antibody Alone and in Combination with GLP-1Causes Fat-Specific Weight Loss






Anti-Myostatin x Activin A
Bispecific Antibody

Combined Myostatin and Activin A Antagonism

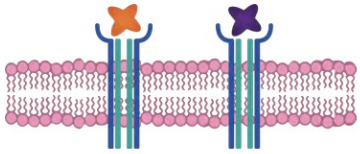
Synergistic Effect on Muscle Growth and Potential Treatment for Pulmonary Hypertension (PH) in Heart Failure With Preserved Ejection Fraction (HFpEF)

For obesity, we are developing bi-specific **co-inhibitors of Myostatin and Activin A** to **enhance muscle growth** and **improve quality of weight loss** during and after treatment with incretin drugs

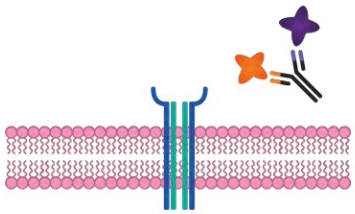
Why Myostatin & Activin A

- Myostatin and Activin A are **key negative regulators** of muscle mass
- Both are members of the TGFβ superfamily
- Activin A mechanism is **pharmacologically validated**[1, 2]
- **Combined** Activin A and Myostatin **inhibition**, **causes** more **pronounced muscle growth**[3]
- Myostatin and Activin A inhibition are **key features for treating PH-HFpEF**

Binding of Myostatin and Activin A to cells leads to **muscle atrophy**



Simultaneous blocking of Myostatin and Activin A leads to **muscle growth**



1.Villanueva, J. et al. Am J Cardiovasc Drugs (2024).
2.US20220119514A1, Regeneron corporate slides
3.Latres, E. et al. Nat Commun 8, 15153 (2017).

A Long-Acting First-in-Class Anti-Myostatin x Activin A Bispecific Antibody

Myostatin x Activin A Bi-specific

First-in-class innovation: Myostatin x Activin A bispecific antibody with unique therapeutic potential

Convenient Dosing: Half-life extension potentially enables dosing every 2-3 months

Optimize Potency: Higher-valency antibody format might increase potency and reduce dose

Potential Advantage: May avoid BMP* inhibition, minimizing bleeding risks associated with ligand traps

Target product profile for obese and potentially Ph-HFpEF patients

- Well-tolerated for long-term use
- Infrequent subcutaneous self-administration

AI-enabled CDR design

- Generates novel IP
- Large library of novel lead molecules

Single-shot multi-dimensional lead optimization

- Bi-specific optimized for affinity, half-life and manufacturability

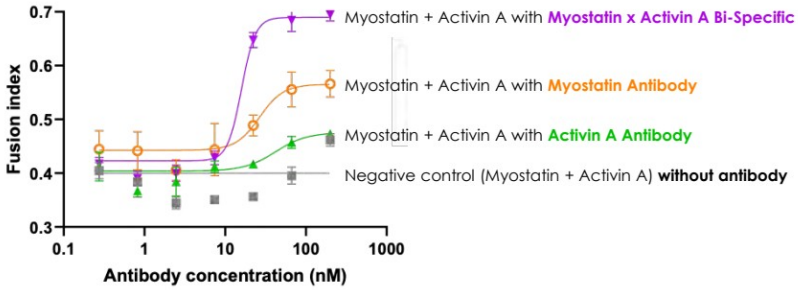
*Bone Morphogenetic Proteins

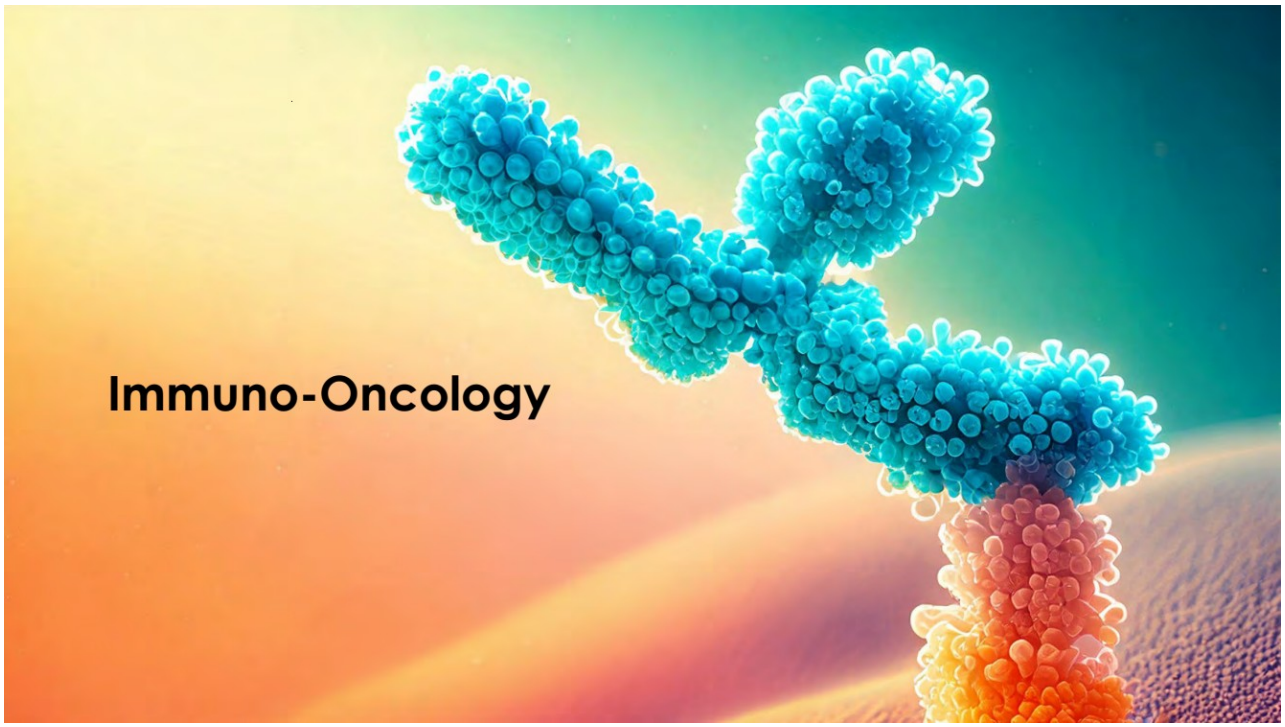
iBio's Myostatin and Activin A Bi-Specific Targets Both Key Negative Muscle Regulators, Synergistically Increasing Muscle Mass

In Vitro Data

Only a Myostatin x Activin A bi-specific antibody fully blocks both muscle growth suppressors, enabling optimal growth, while single-target antibodies fall short

Increased muscle fusion index in human muscle stem cells is a surrogate of muscle growth




Immuno-Oncology

Beyond Cardiometabolic – Driving Value Within Our Oncology Pipeline

Program	MoA	Potential Indications	Early Discovery	Late Discovery	Lead Optimization	IND-Enabling	Highlights
IBIO-101	Treg depletion, IL-2 sparing	Solid tumors, orphan indications					Synergistic efficacy with checkpoint inhibitors
CCR8	Tumor-infiltrating Treg depletion	Solid tumors					Highly selective vs. closely related GPCRs
Trop-2 x CD3 ShieldTx EngageTx	Tumor-protease activated T cell engager	Solid tumors					ShieldTx technology enables masking; delivery as pro-drug activated in TME*
MUC16 x CD3 ShieldTx EngageTx	Tumor-protease activated T cell engager	Ovarian and pancreatic cancer					Binds membrane-proximal epitope, distinct from Regeneron MUC16xCD3
EGFRvIII	ADCC-enhanced Fc	Glioblastoma					Highly selective for EGFRvIII over EGFR
Target 5	Protein Complex Stabilization	Solid tumors					Innovative mechanism of action locking protein complex in inactive form

*Tumor Micro Environment

A Leadership Team with Deep Industry Experience

Martin Brenner, DVM, Ph.D.
CEO & CSO

PFENEX
Lilly
MERCK
AstraZeneca
Pfizer
Recursion

Felipe Duran
CFO

LUPIN
teva
noven

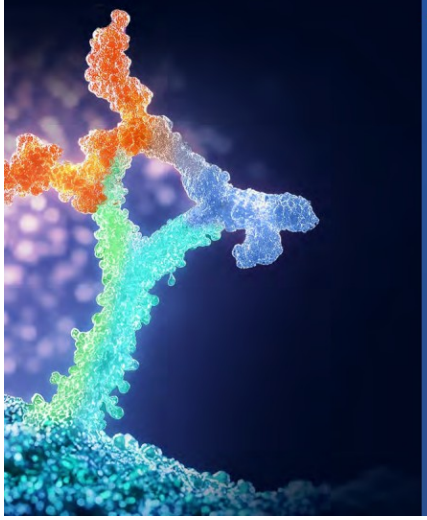
Marc Banjak
CLO

istari
Dova
ICON
B BRAUN
PPD

Kristi Sarno
Senior VP BD

HOMOLOGY
PFENEX
Latham
Crucell

Executive Summary

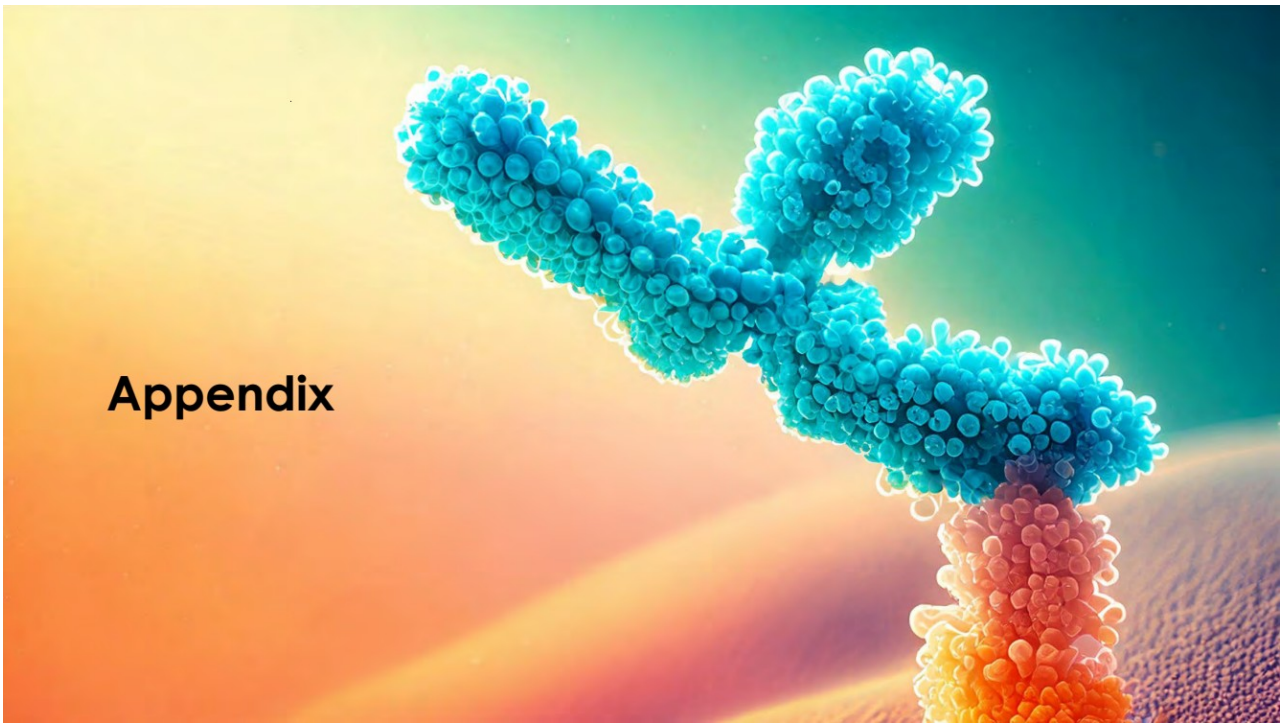


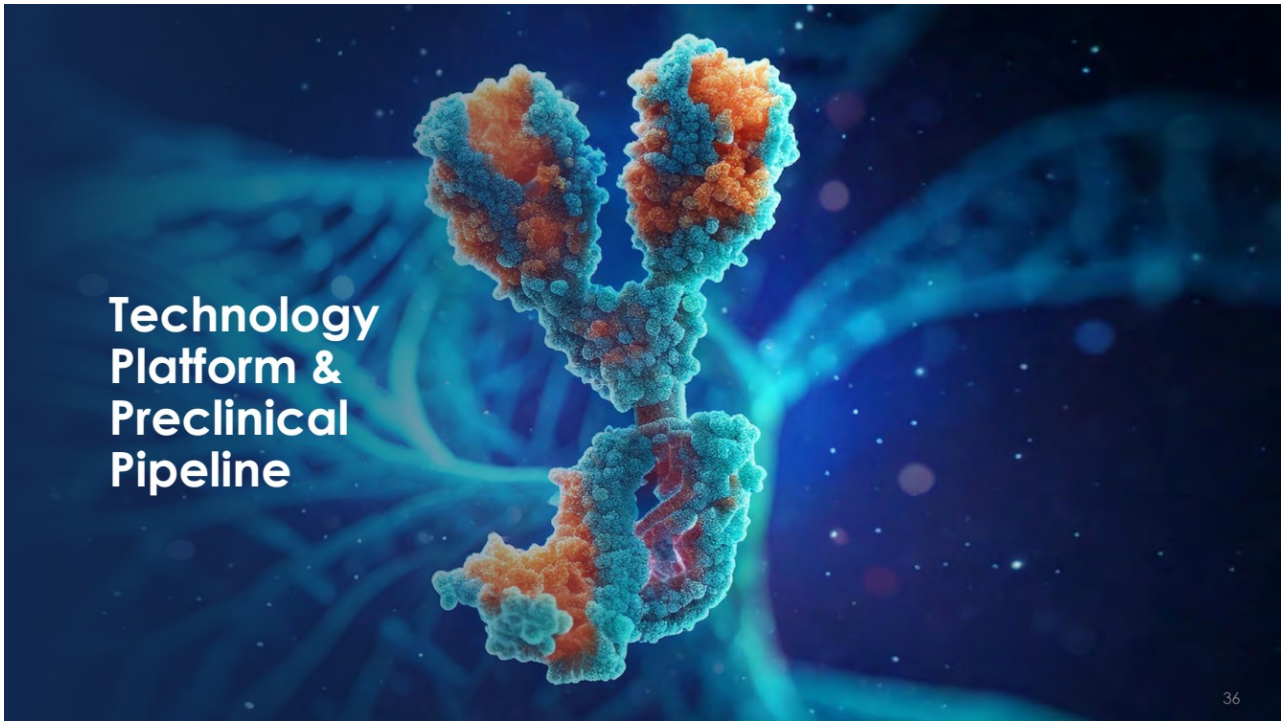
Corporate Highlights

- Patented AI-driven Discovery Tech Stack which can:
  - Rapidly advance a highly developable pre-clinical pipeline
  - Solve hard-to-drug problems
  - Pipeline of cardio/obesity rapidly progressing
  - Pipeline of immuno-oncology molecules ready for strategic partners

Financial Highlights

- $7.2M in Cash and Restricted cash as of Dec 31, 2024
- 10.07M shares outstanding as of Feb 11, 2025


Appendix


Technology
Platform &
Preclinical
Pipeline
36


Technology
Stack
37

iBio's Tech Stack Aims to Solve Major Challenges in Antibody Discovery & Development

Epitope Steering

Unlocking Novel Biology

Pursuit of Elusive Targets
GPCRs, Ion Channels, Protein Complexes

Complex modalities
Agonistic Antibodies, Cell Activators, Protein Complex Stabilizers

Proprietary Naïve mAb Library

Improved Speed and Developability

Fully human Ab
Reduced immunogenicity risk by clinically validated Ab frameworks

Speed
Rapid hit ID vs immunization campaigns

Improved Developability
Known sequence liabilities eliminated

StableHu & Mammalian Display

Library Diversity
ML tools create focused diversity with smaller library size

Speed
Simultaneous, Multi-Dimensional Optimization

Improved Developability
Mammalian Display with production cell lines exclusively yields expressible clones

Optimized Antibody Leads

Reduced Lead-Optimization Time
Optimization in less than **4 weeks**

Minimized Developability Risk
Mammalian Display in Manufacturing Cell Line

Potential for Improved Safety
Selective "on-tissue" action of masked antibodies

First in Class Antibodies and / or Best in Class Antibodies

iBio's Tech Stack Addresses Immuno-oncology Discovery and Development Challenges

EngageTx

2nd Gen T-cell Engager Panel

Sequence Diversity
Increased humanness and broad CD3 activity for optimized pairing with antigen arms

Hu-Cyno Cross Reactivity
Risk reduction via cyno monkey toxicity study compatibility

Range of Cytokine Release
Tailored cytokine release for expanded therapeutic window

ShieldTx

Greater Safety With Tissue Specificity

Seamlessly Integrated Ab Masking
Engineered epitopes serve dual purpose for raising and masking of Abs

Flexibility in Candidate Selection
Simultaneous co-optimization of Ab, mask and linker provides maximized flexibility in candidate selection

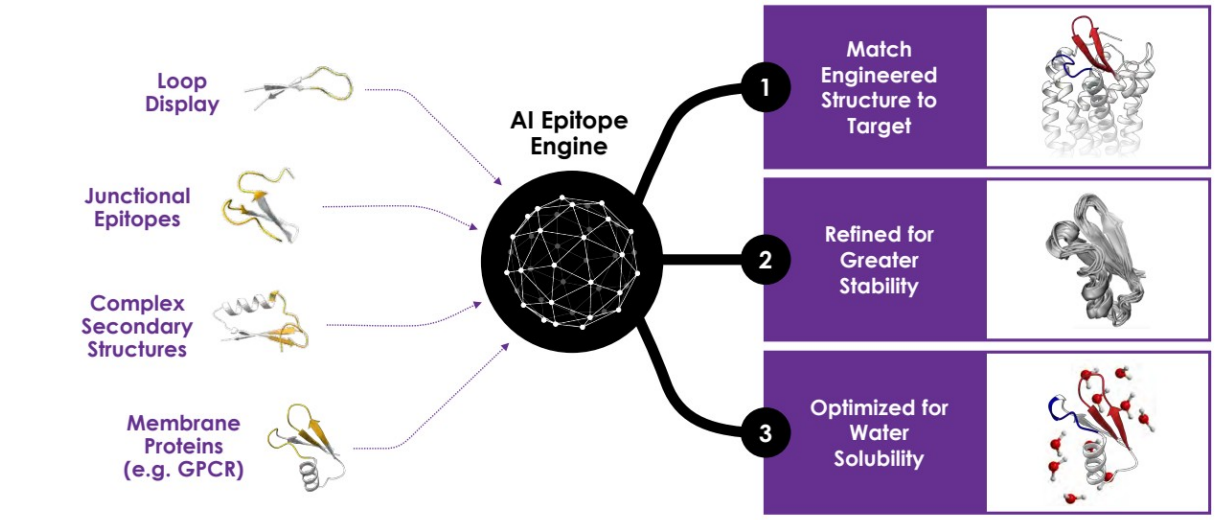
Enhanced Efficacy and Safety of I/O Antibody Leads

Finely tuned T-cell engagement
Adjustable T-cell engagement to fit any tumor target engager

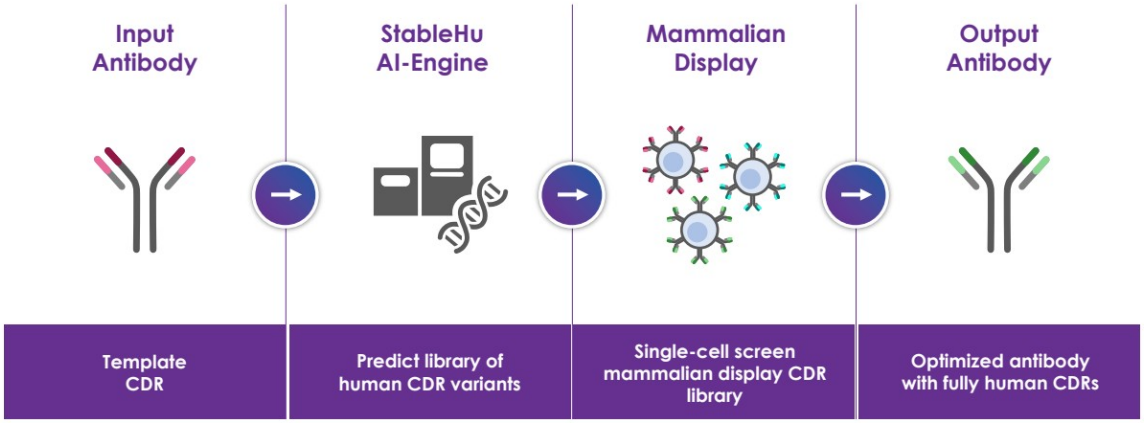
Improved safety prediction
Cyno cross reactivity allows for better preclinical safety assessment

Improved Safety Profile
Tissue selective action through "smart", conditionally activated, antibodies

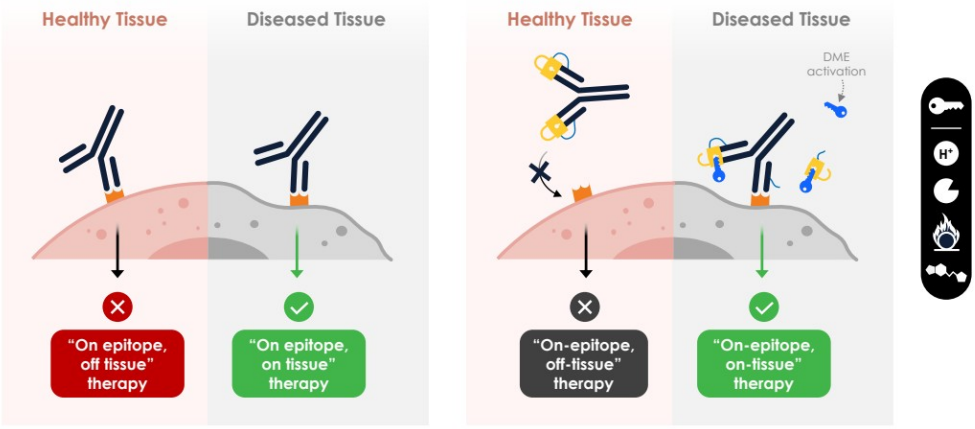
Unlocking High-Value Drug Targets: AI-Engineered Epitopes are Generalizable to a Broad Set of Complex Structural Drug Binding Sites



Accelerate Success: StableHu Antibody Optimization & Mammalian Display Screening Propel Faster, Cost-Effective Antibody Development



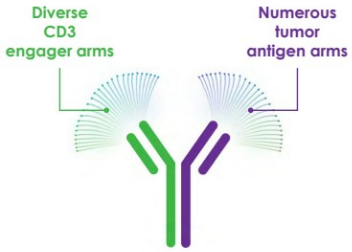
"Smart" Antibodies: ShieldTx Conditionally Activated Antibodies Strive to Improve Safety by Selectively Targeting Diseased but not Healthy Tissue



EngageTx, a CD3-Based T-Cell Engager Panel, Addresses 3 Key Challenges: Cytokine Release, NHP Cross-Reactivity and Immunogenicity Risk

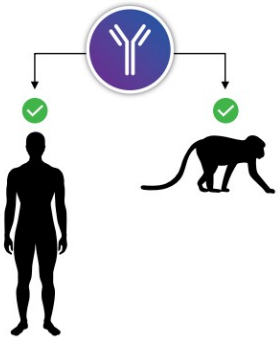
1 Sequence Diversity

Increased humanness and broad CD3 activity for optimized paring with tumor antigen arms



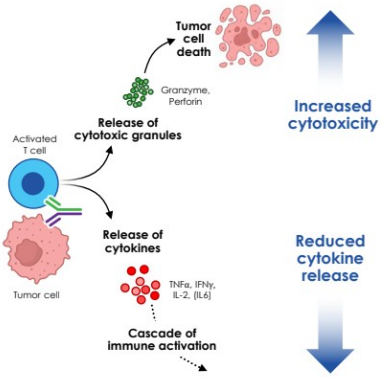
2 Hu-Cyno Cross-Reactivity

Risk reduction via cyno monkey toxicity study compatibility

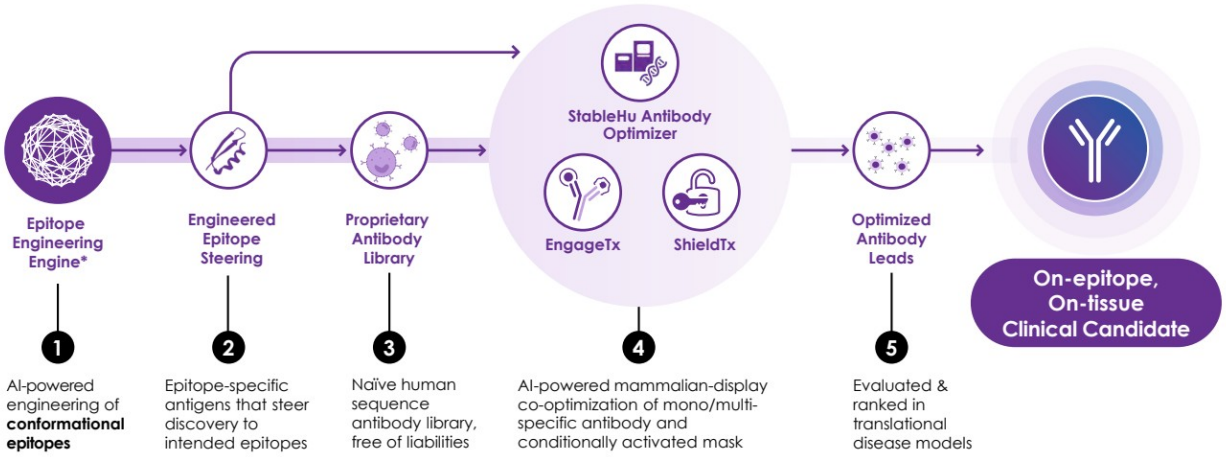


3 Range of Cytokine Release

Tailored cytokine release for expanded therapeutic window



iBio's Platform Tackles Discovery Challenges for the Next Era of Antibodies



* U.S. Patent No. 11,545,238 (issued January 3, 2023)


IBIO-101
IL-2 Sparing Anti-CD25
45

IBIO-101 for Regulatory T-Cell (T_{reg}) Depletion

Target Mechanism

Depletion of immunosuppressive T_{regs} via antibody dependent cellular cytotoxicity (ADCC), without disrupting activation of effector T-cells (T_{effs}) in the tumor microenvironment

Potential Indications

- Solid tumors
- Hairy cell leukemia
- Relapsed mult. myeloma
- Lymphoma
- Head & neck cancer

Differentiation / Opportunity

- IL-2 sparing anti-CD25 antibodies enables depletion of T_{regs} without affecting T_{effs}
- Fast-follower to Roche's RG6292 clinical molecule

Recent Transactions & Milestones

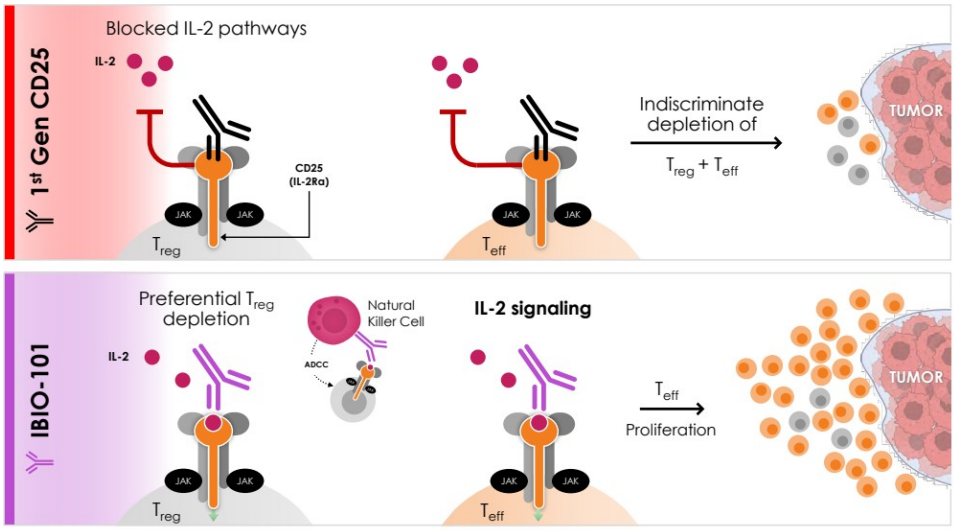
PRECLINICAL TESTING | IND | CLINICAL TESTING | CLINICAL PROOF OF CONCEPT

***Roche / Tusk Therapeutics (*Sep 2018*)**
$81M upfront,
$677M milestones

****Roche RG6292 Ph1 Data (*Apr 2023*)**
Well-tolerated: manageable safety profile
Confirmed MOA: Reduced intratumoral T_{regs}
Efficacy: 29% stable disease (45% PD-L1 combo)

**Roche acquisition of Tusk Therapeutics completed for €70M upfront, acquiring worldwide rights to anti-CD25 program. Values converted to dollars as reported in public press releases*
***Data presented by Roche at AACR 2023*

IBIO-101 Reduces Tumor Growth in Preclinical Studies by Selectively Depleting Immunosuppressive T_{regs} without Affecting Cancer Killing T_{effs}



1st gen CD25 mAbs depleted immuno-suppressive T_{reg} and immuno-stimulatory T_{eff}

Limited efficacy

2nd gen IBIO-101 selectively targets T_{regs} without blocking IL-2 signaling to T_{effs}

Strong preclinical anti-tumor response

Data on file. Treg = Regulatory T Cells; Teff = Effector T Cells; ADCC = Antibody Dependent Cellular Cytotoxicity

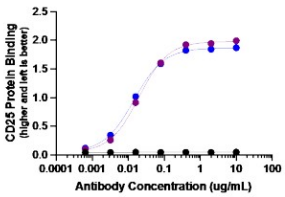
IBIO-101 Selectively Depletes Tregs

IBIO-101 potently binds recombinant CD25

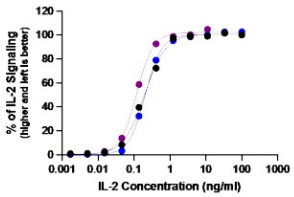
while preserving IL-2 signaling

which leads to T_{reg} depletion

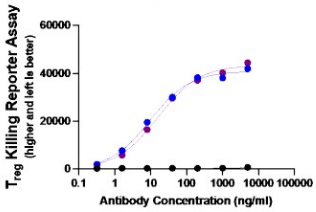
while sparing T_{effs}



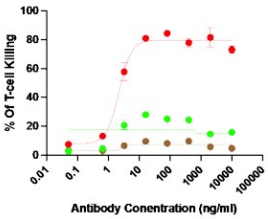
- Negative control, EC_{50} = no binding
- IBIO-101, EC_{50}= 16.4 ng/ml
- RG6292 (Roche), EC_{50}= 24.7 ng/ml



- IL2, EC_{50}= 0.11 ng/ml
- IBIO-101, EC_{50}= 0.17 ng/ml
- RG6292, EC_{50} = 0.14 ng/ml



- Negative control, EC_{50} = no cell killing
- IBIO-101, EC_{50}= 4.7 ng/ml
- RG6292, EC_{50} = 18.6 ng/ml

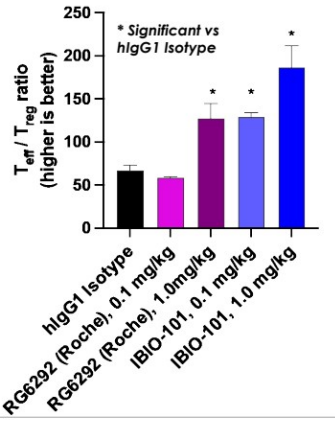


- T_{reg} killing, EC_{50}= 7.09 ng/ml
- Activated $CD4^+$ T_{eff} killing, EC_{50}= no activity
- Activated $CD8^+$ T_{eff} killing, EC_{50}= no activity

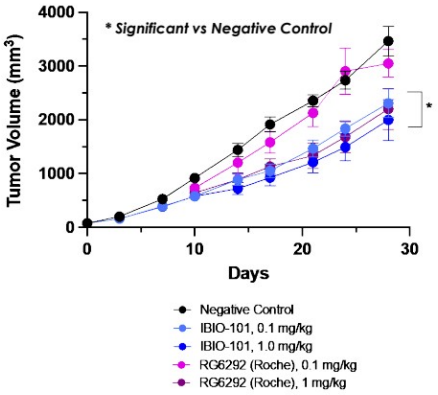
RG6292 is Roche's monoclonal antibody that targets CD25 (IL-2Rα). IBIO-101 data on file.

IBIO-101 Increases in T_{eff}/T_{reg} Ratio in Preclinical Studies Inhibiting Tumor Growth

Potently increases T-eff/T-reg ratio[1]



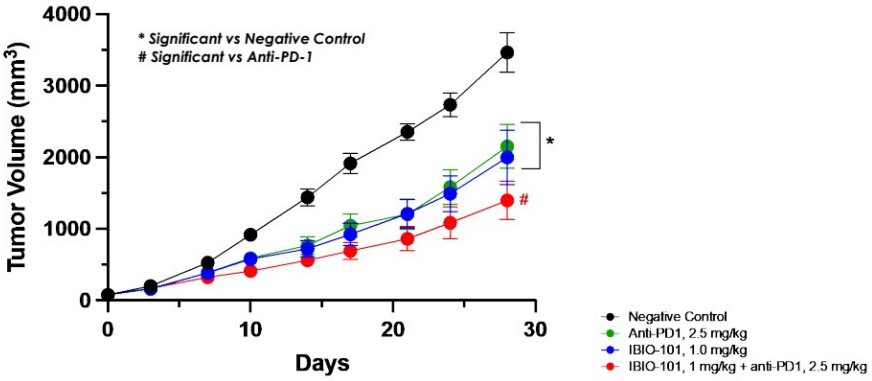
Tumor growth inhibition correlates with T-eff/T-reg ratio



[1]hCD25 animal model - Data on file.

IBIO-101 in Combination With a Checkpoint Inhibitor Shows Greater Efficacy

IBIO-101 + PD-1 Checkpoint Inhibitor In PreClinical Studies Enhances Tumor Suppression



*hCD25 animal model - Data on file.


Anti-CCR8
High ADCC Anti-CCR8
for the Depletion of
T-regulatory Cells
51

CCR8 for Tumor-Infiltrating T_{reg} Depletion

Target Mechanism

Tumor-infiltrating Tregs highly express CCR8. iBio program targets depletion of highly immunosuppressive CCR8+ Tregs in tumor microenvironment via an ADCC mechanism.

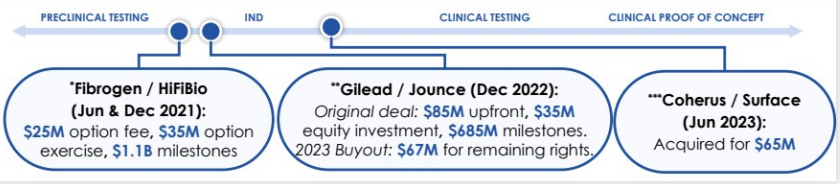
Potential Indications

- Broadly applicable in solid tumors
- Prospective combination therapy

Differentiation / Opportunity

- Selective binding to CCR8 over its close homolog, CCR4

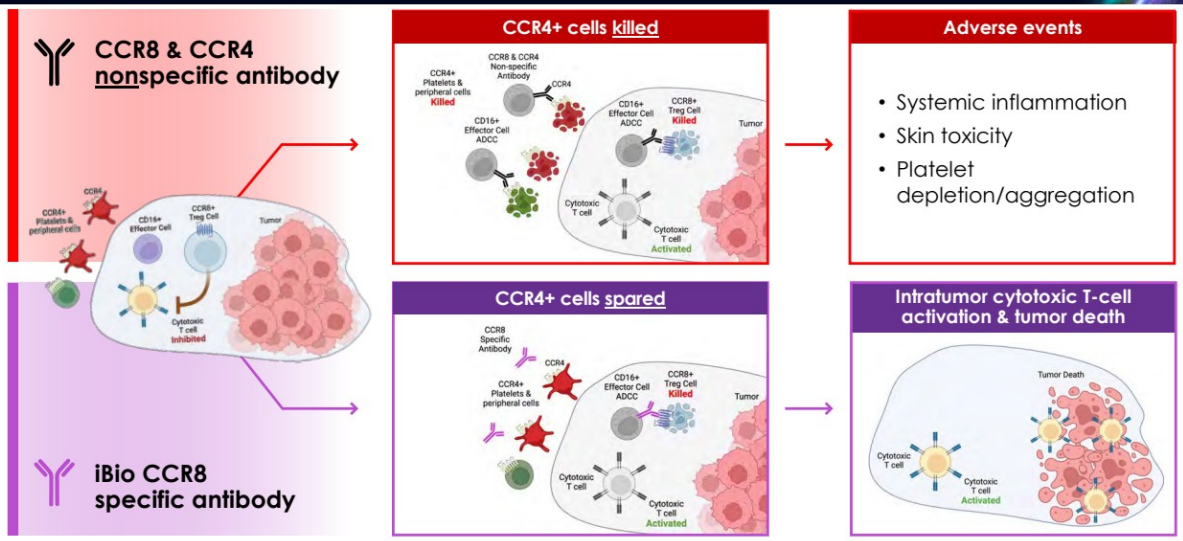
Recent Transactions & Milestones



*Fibrogen / HiFiBio: Fibrogen purchased option to multiple programs in June 2021, then exercised the option for excl. license to CCR8 program in Dec. 2021.
**Gilead / Jounce: Exclusive worldwide license to anti-CCR8 antibody.
*** Coherus / Surface Oncology: acquisition, announced in June 2023, adds two clinical assets, including a phase 2 anti-IL-27 and a phase 1/2 anti-CCR8 for oncology.

CCR8+ T_{reg} Cells Are Tumor Infiltrating and Highly Immunosuppressive

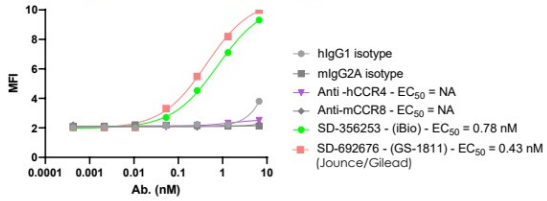
Depletion of CCR8+ Treg cells has potential to evoke potent tumor immunity



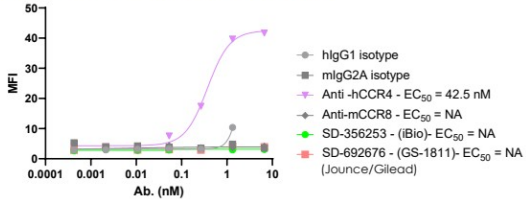
Afucosylated Anti-CCR8 Antibody Exhibits High Specificity, CCL1 Antagonism and CCR8-Specific Cell Killing

High Specificity CCR8 Cell Binding

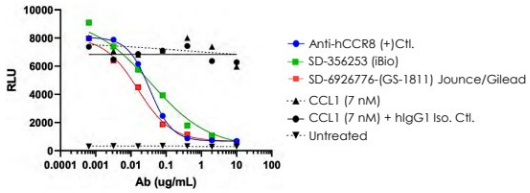
Potent binding to CCR8 overexpressing cells



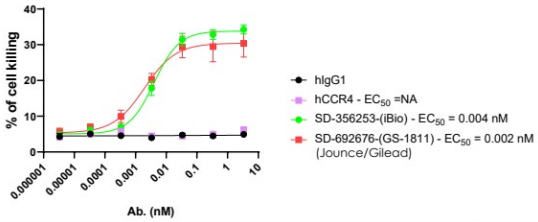
No binding to CCR4 overexpressing cells



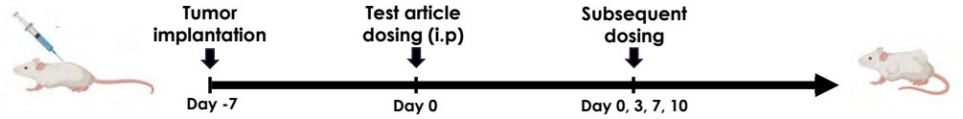
CCR8-CCL1 Antagonism

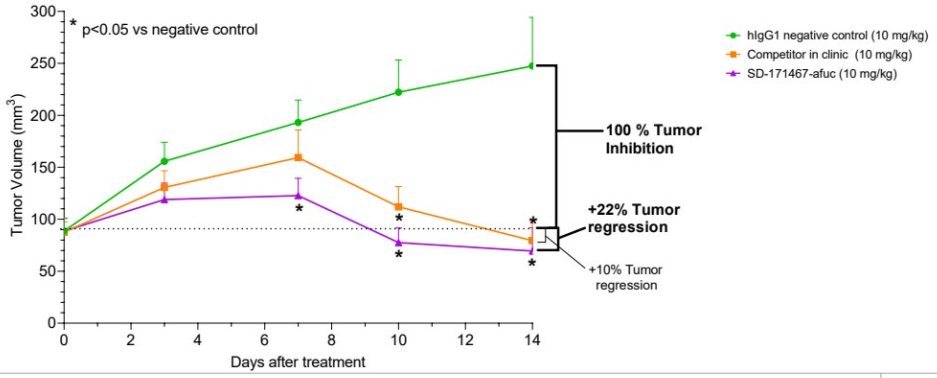


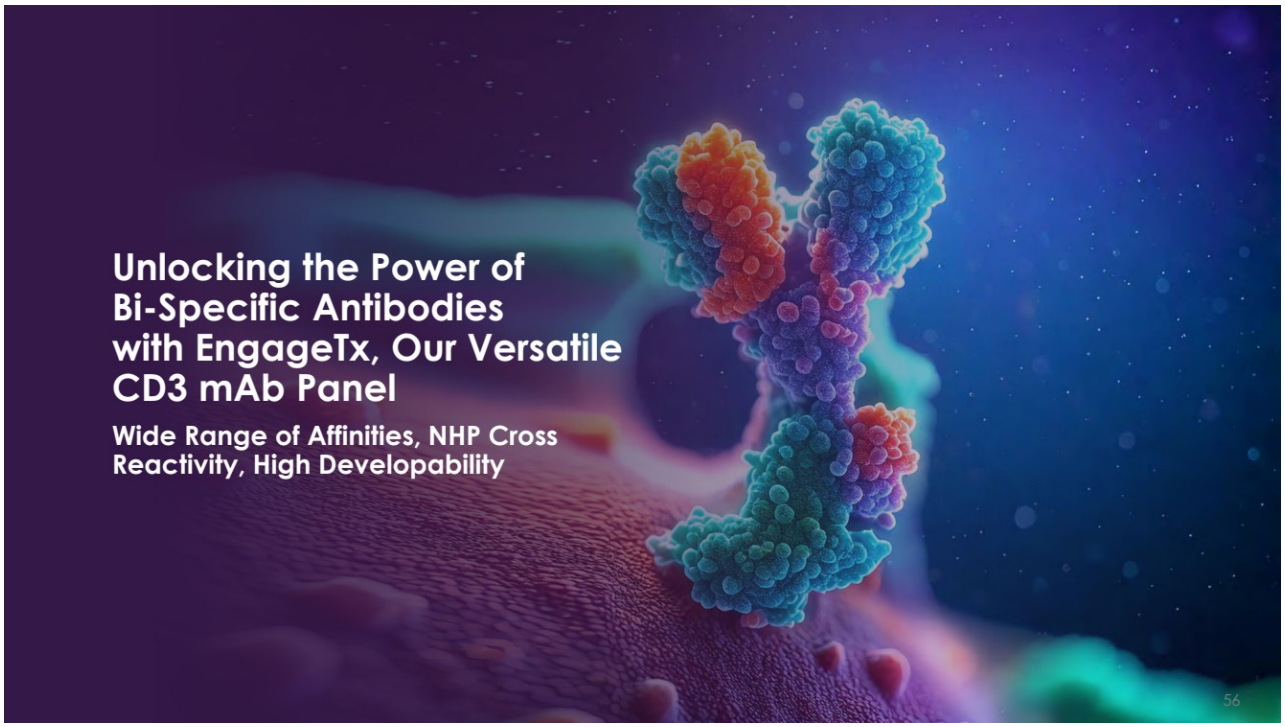
PBMC-Induced CCR8 Cell Killing



iBio's CCR8-Specific High ADCC Antibody Induces Tumor Regression in a Transgenic Human CCR8 Mouse Model






Unlocking the Power of Bi-Specific Antibodies with EngageTx, Our Versatile CD3 mAb Panel
Wide Range of Affinities, NHP Cross Reactivity, High Developability
56

Next Generation Anti-CD3 T Cell Engagers

Target Mechanism

T-cell-redirecting bispecific antibodies are a new therapeutic class that simultaneously targets CD3 on T cells and tumor antigens, inducing T cell mediated tumor cell killing

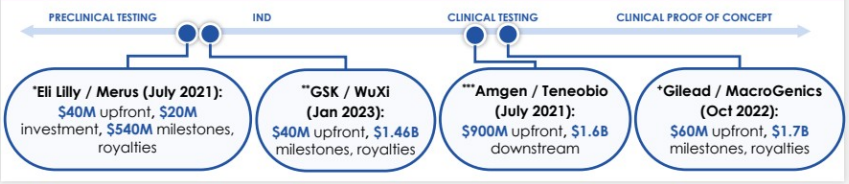
Potential Indications

- Broad solid tumor potential
- Expands therapeutic options across programs

Differentiation / Opportunity

- Range of T cell activation for diverse tumor antigens
- Cyno-tox study compatibility
- StableHu optimized sequence reduces downstream risks

Recent Transactions & Milestones



*Eli Lilly / Merus: Fibrogen Research collaboration using Merus' proprietary platform to develop up to three CD3-engaging T-cell re-directing bispecific antibody therapies.
** GSK WuXi: License of WuXi's preclinical CD3 bi-specific, plus 3 earlier stage programs
***Amgen / Teneobio: Teneobio was developing a heavy-chain only platform as well as its CD3 engager technology, TNB-585, the lead program, was in phase 1.
+ Gilead / MacroGenics: Gilead granted option to MGD024, a phase 1 CD3 bi-specific, plus collaboration on two additional research programs.

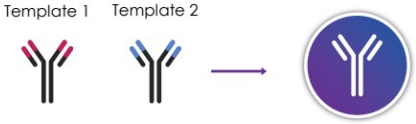
Dual Approaches to a Diverse Panel of Anti-CD3 Antibodies

AI Discovery Engine

Structural-Epitope Immunization & Screening

AI-Engineered Immunogens → Epitope & T Cell Immunization → Epitope & CD3 Screen

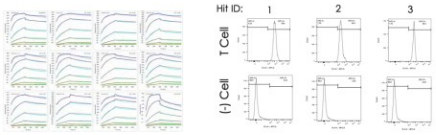
StableHu Optimizer



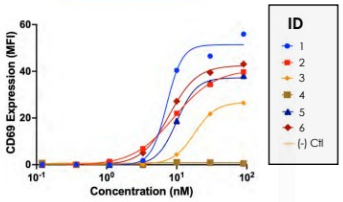
2 Template Antibodies → Optimized Antibodies

SCREEN

Hu/Cyno CD3 & T Cell Binding



T Cell Activation

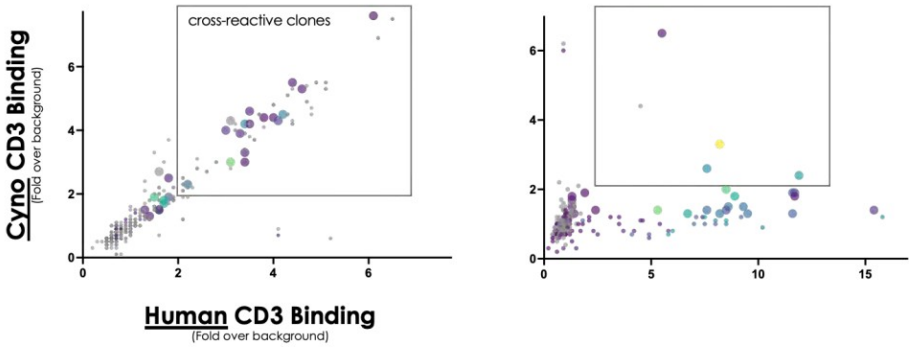


Libraries and Screens Discover Hu-Cyno CD3 Cross-Reactive Antibodies

Library Screen:

StableHu **Mammalian-Display**

Epitope-Steered **Immunization**



Human T Cell **Binding**

High

Medium

Low

Selected Hit

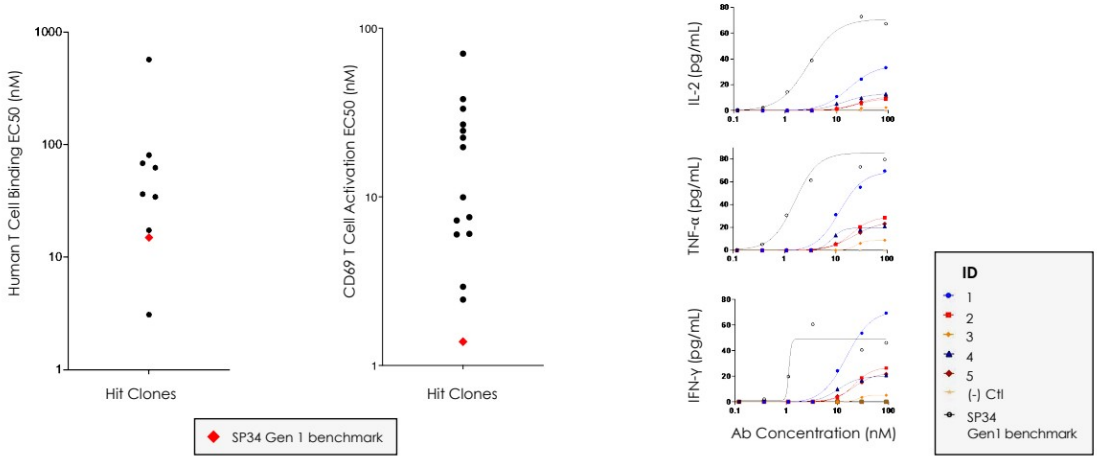
Not Selected

EngageTx is Selected for a Diversity of T Cell Binding and Activation

T Cell Assay: **Binding** | **Activation** | **Cytokines**

EC50: 3 – 570 nM

EC50: 2.5 – 70 nM




ShieldTx
Antibody masking technology for delivering on-epitope, on-tissue clinical candidates with enhanced safety and developability
61

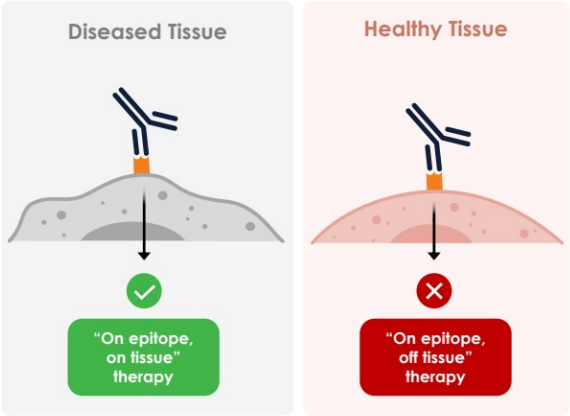
On-Target-Off-Tissue Side Effects Severely Limit The Potential of Existing And Future Antibodies

"(...) targeting antibody delivery to selected organs and tissues (...) represents a major unmet challenge that if ultimately solved may rewrite medical textbooks" - Paul J. Carter and Arvind Rajpal, Cell, 2022.

Even exquisitely specific antibodies fail in clinical trials by doing exactly what they are asked to do – hit the target. The problem often lies in the target being also expressed on *healthy* tissue.

Many potential targets remain unexplored as a drug target for fear of on-epitope off-tissue side effects.

The challenge: how do we achieve disease tissue specificity while avoiding healthy tissue expressing the same epitope?

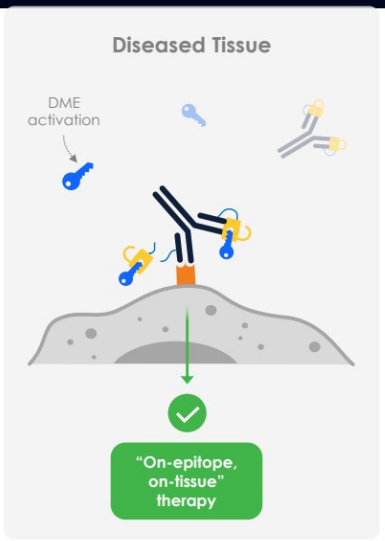


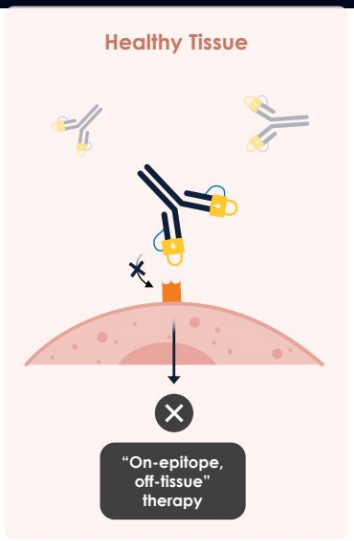
Our Engineered Epitopes Provide an Integrated Solution for Identifying And Subsequently Masking Antibodies

Antibodies are activated by the removal of the mask in the diseased tissue.

Masks can be removed by tumor-specific enzymes, pH, redox state, and disease-specific metabolites.

The technology can be employed for other indications i.e. inflammatory and auto-immune diseases.





Antibodies remain inactive in healthy tissue

Masked Antibodies are a Proven Concept and iBio's Platform has the Potential to Solve Key Remaining Challenges

	THE PROBLEM	OUR SOLUTION
1 **Discovery process**	*Separate* antibody and mask discovery process is inefficient	*Co-discovery* of epitope-steered antibody and mask is more efficient
2 **Masking performance**	Separate discovery processes does *not co-evolve* an optimal antibody, mask, linker combination	*Co-evolution* of libraries of antibody, mask and linker for maximized effectiveness of masking and unmasking
3 **Developability**	Antibody + mask + linker combinations *not screened* for high developability in production cell lines	Mammalian-display libraries of antibody, mask and linker combinations *screened for developability* in production CHO cell lines
4 **Immunogenicity**	*Random* peptide or anti-idiotype masks increase masked antibody immunogenicity risk	*Engineered epitope masks* are designed with intention to maximize the natural sequence of the epitope and minimize immunogenicity


Anti-Trop-2 x CD3
Bi-Specific Antibody against Tumor-Specific Trop-2 Cancer Cells
65

Trop-2 x CD3 Bi-Specific Antibody Potentially for Head & Neck and Other Cancer

Target Mechanism

Select killing cancer cells that up-regulate Trop-2 expression while improving safety margin in reducing cytokine release syndrome (CRS)

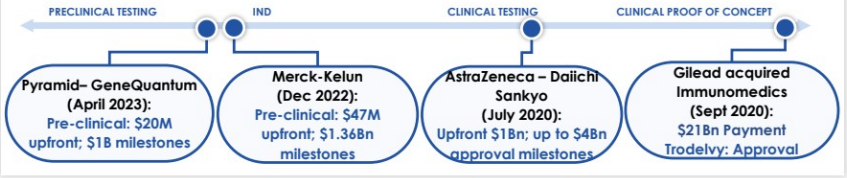
Potential Indications

- Head & neck cancer
- Lung cancer
- Ovarian cancer
- Breast cancer
- Pancreatic cancer

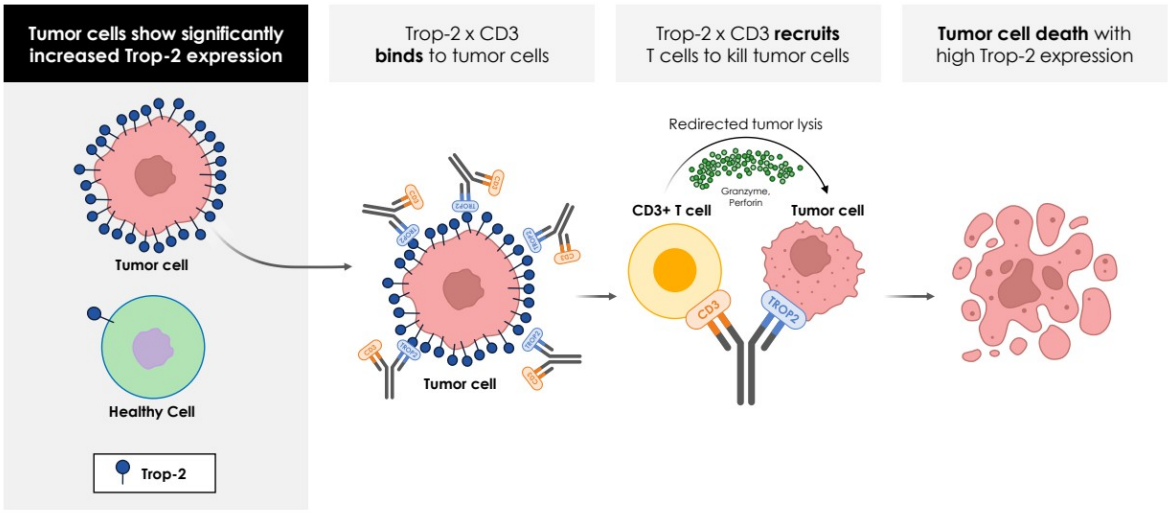
Differentiation / Opportunity

- Novel Trop-2 epitope with extreme high affinity to target
- Trop-2 binder with mouse/cyno/human cross reactive enables early safety profile optimization
- Optimal iBio CD3 engager with low CRS and cyno/human cross reactive

Recent Trop-2 ADC Transactions & Milestones

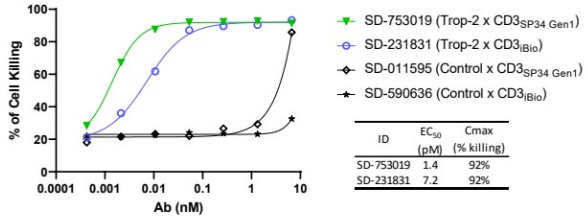


Trop-2 x CD3 Bi-Specific Antibody Selective Target Overexpress Trop-2 Cancer Cells



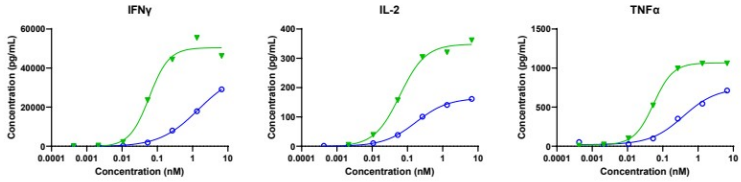
iBio's Trop-2 x CD3 Bi-Specific Antibody Potently Kills Tumor Cells with Low Cytokine Release

Potent Cancer Cell Killing

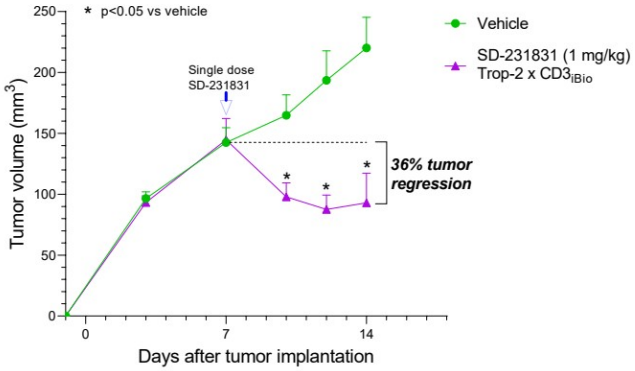


ID	EC_{50} (pM)	Cmax (% killing)
SD-753019	1.4	92%
SD-231831	7.2	92%

Minimal Cytokine Release



A Single Dose of iBio's Bispecific Trop-2 x CD3 Antibody Induces Tumor Regression in a Humanized Mouse Cancer Model




Conditionally Activated Anti-MUC16 x CD3 Bispecific Antibodies Targeting the Non-Shed MUC16 Region
Leveraging iBio's Epitope Steering, ShieldTx, and EngageTx Technologies
70

MUC16 Potentially for Ovarian and Other Cancers

Target Mechanism

Bind a membrane-proximal MUC16 epitope

Membrane-proximal binding avoids epitope elimination by tumors

Bind a non-glycosylated epitope to avoid altered glycosylation on tumors

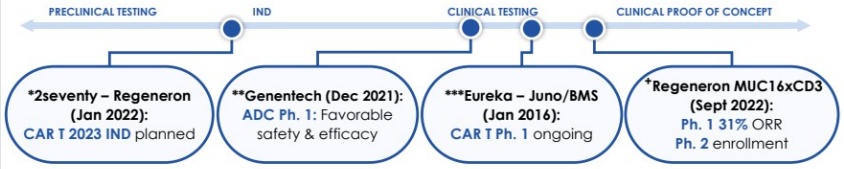
Potential Indications

- Ovarian
- Uterine
- Pancreatic

Differentiation / Opportunity

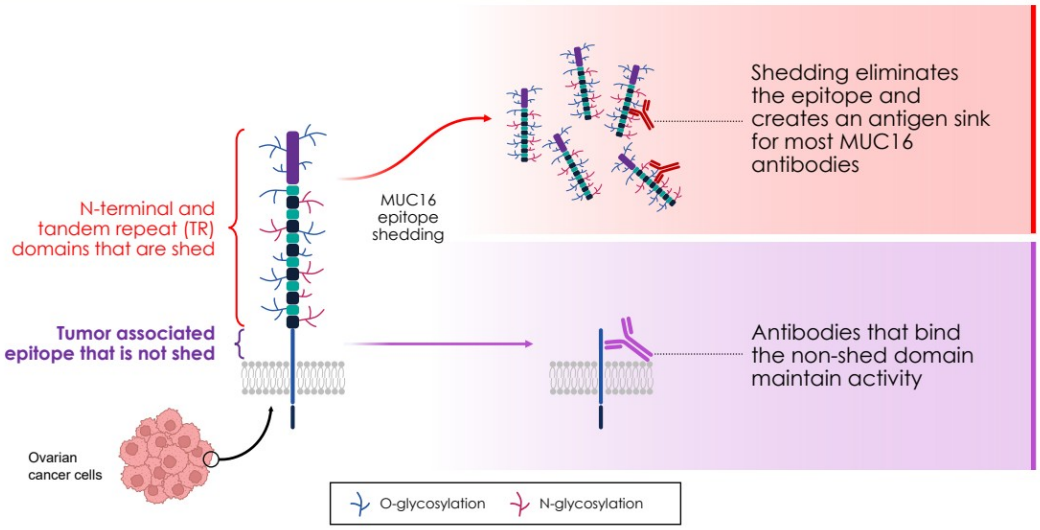
- MUC16 epitope avoids primary modes of tumor evasion
- Enabling modalities: T Cell engager, ADC, CAR-T

Recent Transactions & Milestones

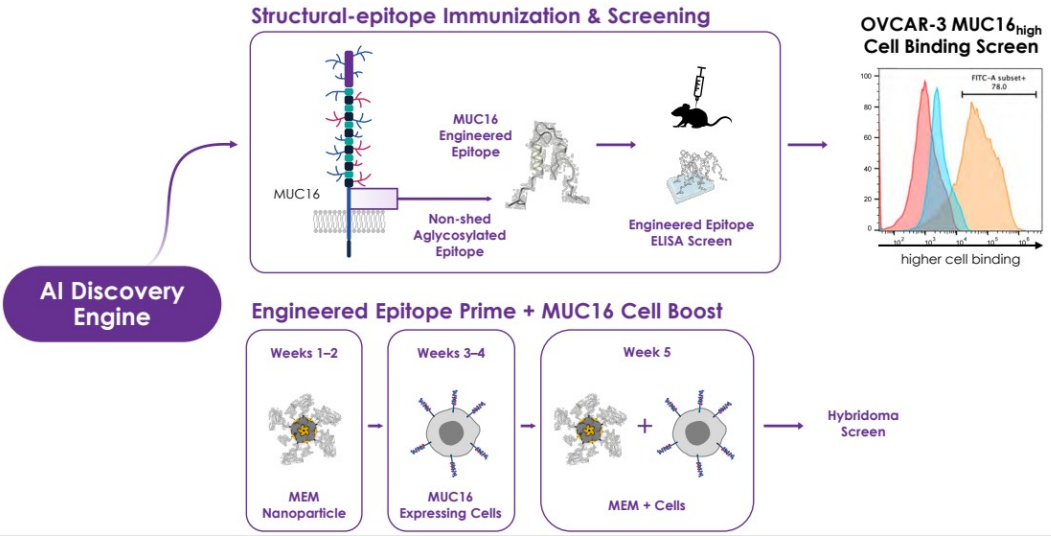


*Regeneron, 2seventy name the target of their first solid tumor CAR-T, aim for 2023 IND
** Liu et al., An open-label phase I dose-escalation study of the safety and pharmacokinetics of DMUC4064A in patients with platinum-resistant ovarian cancer
***Eureka Therapeutics Announces Exclusive License Agreement between Memorial Sloan Kettering Cancer Center and Juno Therapeutics for Use of a Novel, Fully-Human MUC16 Binder in CAR T Cell Immunotherapy
+Novel Regeneron Bispecific Antibodies Show Encouraging Anti-Tumor Activity in Two Advanced Solid Tumors

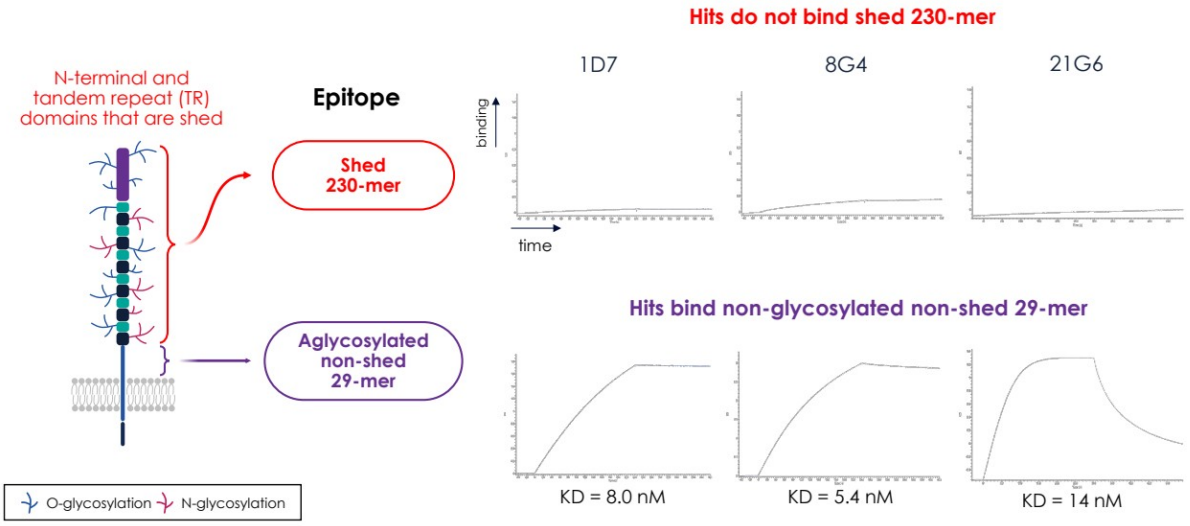
MUC16 Is Overexpressed and Shed by Tumor Cells



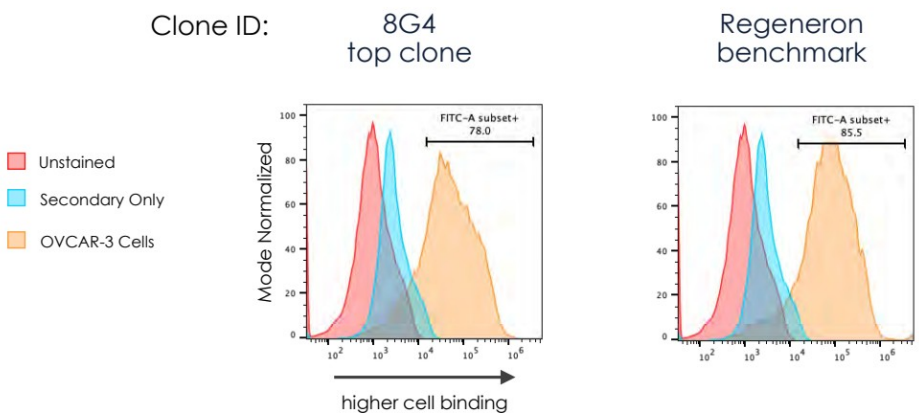
Immunizations Were Steered to a MUC16 Epitope that Avoids Epitope Shedding



Top Three Hit Clones Bind the Non-Glycosylated MUC16 Epitope Closest to the Membrane



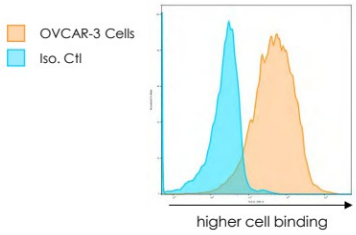
Top MUC16 Clone 8G4 Binds OVCAR-3 Cells Comparable to Regeneron Benchmark



8G4 Clone Maintains OVCAR-3 Cell and MUC16 Epitope Binding in a Fully Human Framework

8G4 with fully human framework reduces immunogenicity risk

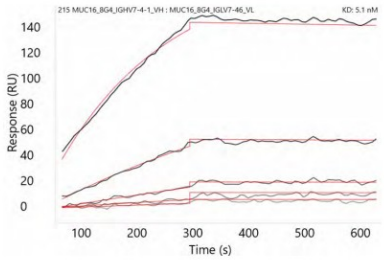
Cell binding



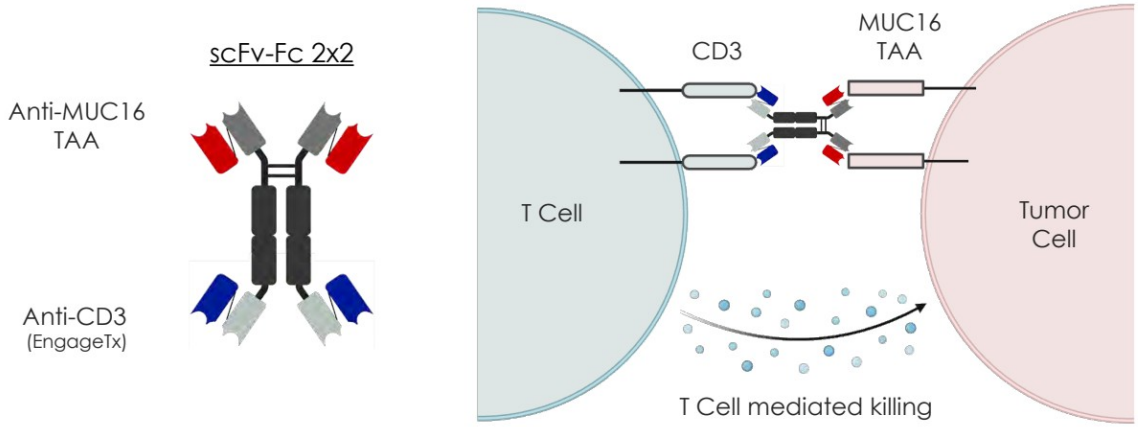
Glycosylated MUC16 membrane-proximal epitope SPR:

KD = 5.1 nM

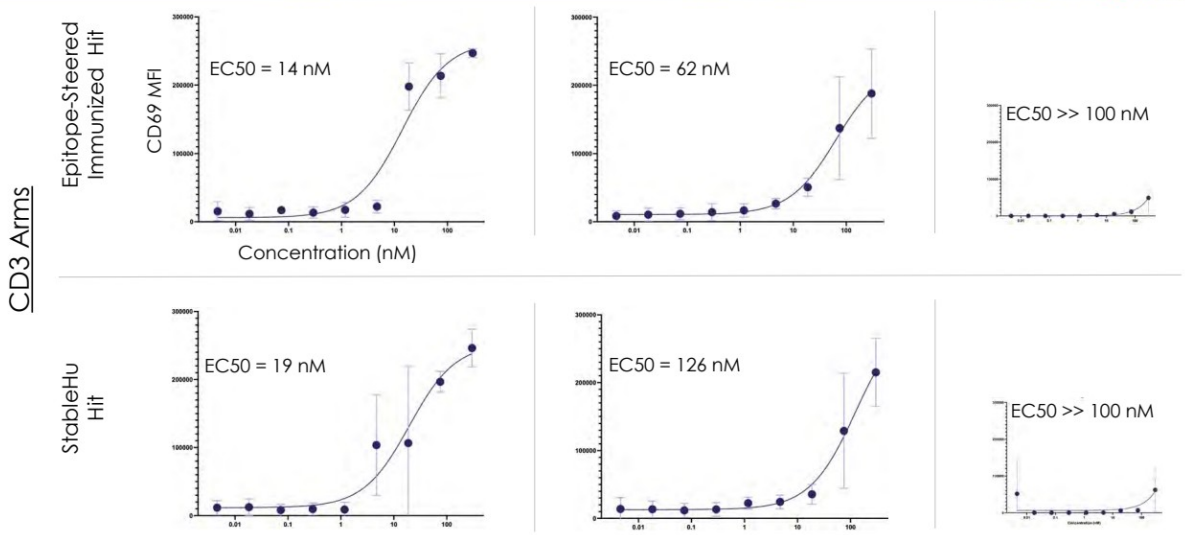
Epitope binding



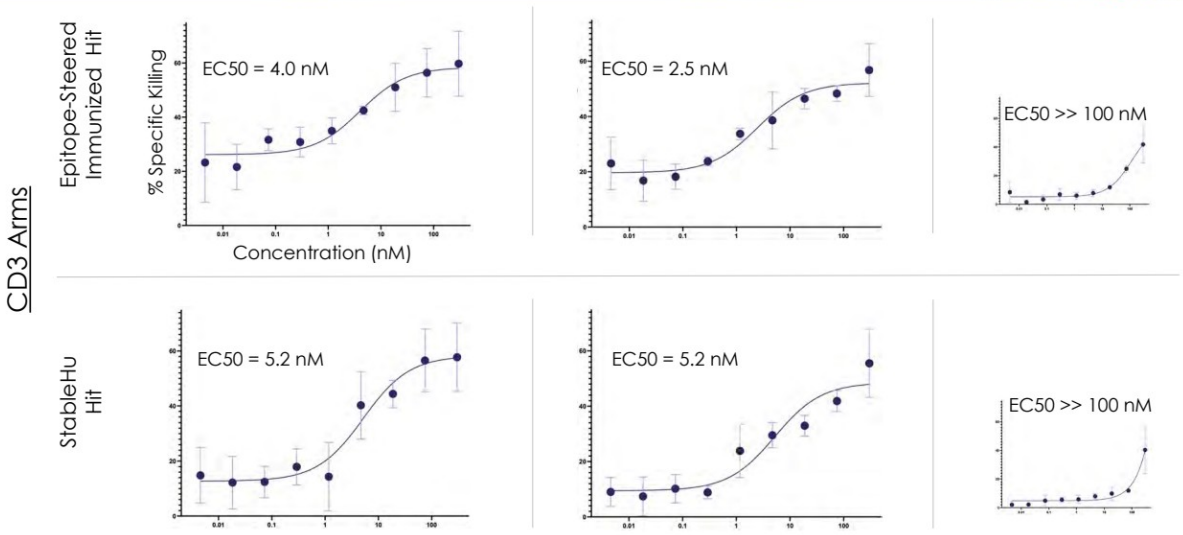
Efficient Expression with 2x2 Format: Anti-CD3 x MUC16 Bispecific T-Cell Engagers



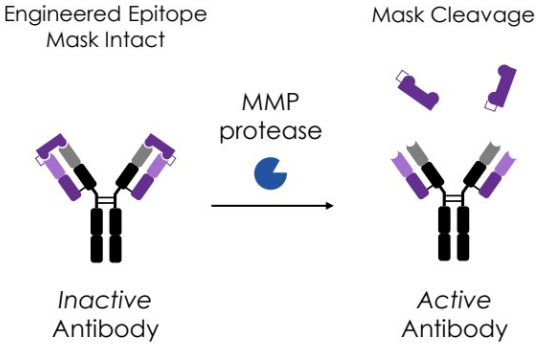
2X2 Anti-CD3 X MUC16 T Cell Engagers Stimulate T Cells in Donor PBMCs

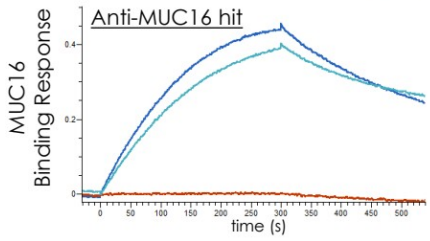


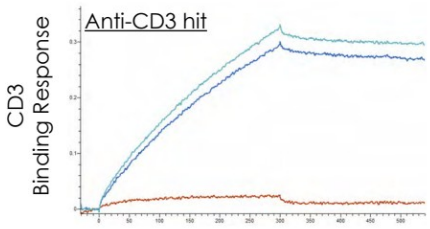
2X2 Anti-CD3 X MUC16 T Cell Engagers Kill OVCAR-3 Ovarian Cancer Cells

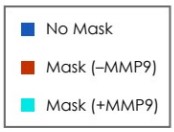


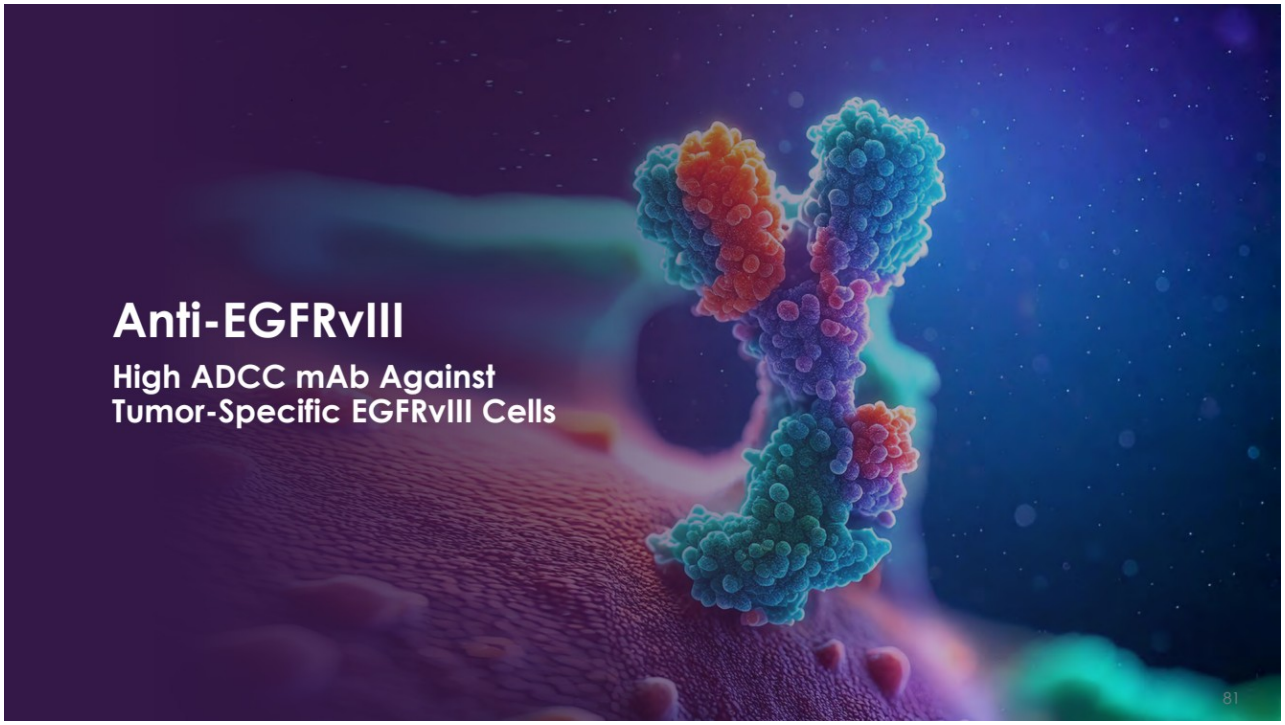
ShieldTx Engineered Epitope Mask Conditionally Activates MUC16 and CD3 Hits










Anti-EGFRvIII
High ADCC mAb Against
Tumor-Specific EGFRvIII Cells
81

EGFRvIII Potentially for Glioblastoma and Other Cancers

Target Mechanism

Binding a tumor-specific mutation of EGFR variant III with an afucosylated antibody for high ADCC.

EGFRvIII is constantly "switched on" which can lead to the development of a range of different cancers.

Potential Indications

- Glioblastoma
- Head & neck cancer
- Non-small cell lung cancer

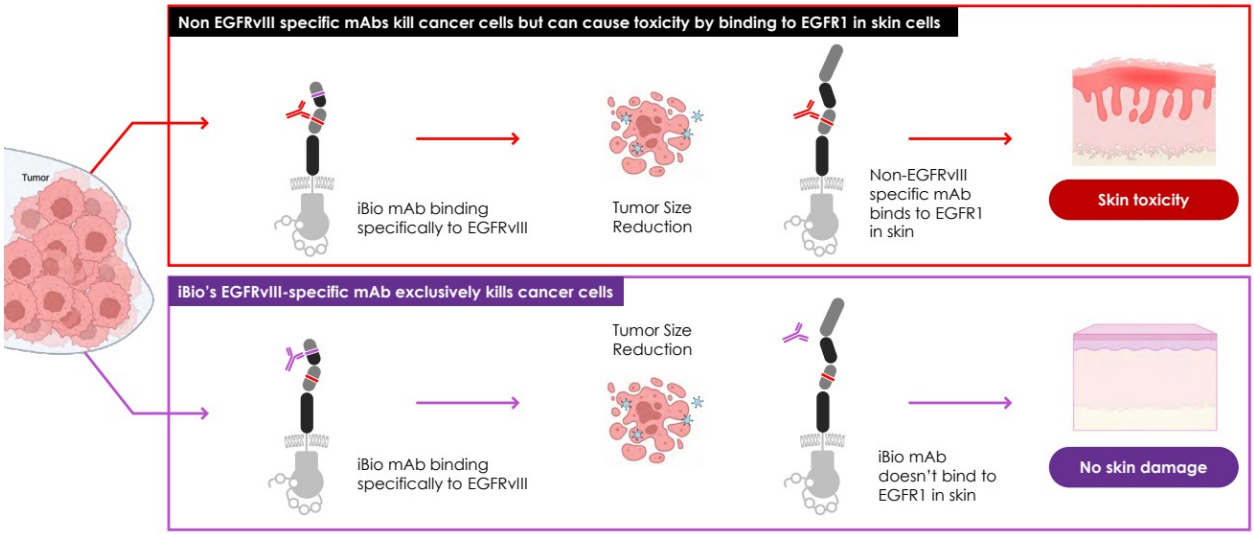
Differentiation / Opportunity

- Novel EGFRvIII high ADCC mechanism, potentially further reducing toxicity & expanding therapeutic window
- Other enabling modalities: T Cell engager, ADC, CAR-T

Recent Transactions & Milestones

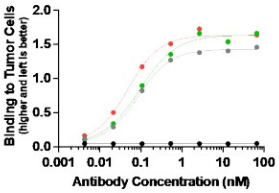
** Pierre Fabre / Scorpion: Scorpion licensed two preclinical-stage programs to Pierre Fabre which are targeted to specific EGFR mutations in lung cancer.*
***Seagen transaction with LAVA Therapeutics was an exclusive license to LAVA-1223 (EGFR program), plus additional projects using Lava's platform.*
****Taiho transaction to acquire Cullinan Oncology's subsidiary, Cullinan Pearl, which has worldwide rights outside of Japan to CLN-081/TAS6417 (EGFR mutant mAb).*

iBio's Anti-EGFRvIII mAbs Selectively Kill EGFRvIII-Positive Tumor Cells and Not EGFR1-Expressing Cells in Healthy Tissues

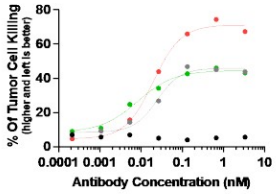


iBio's EGFRvIII-Selective mAbs Kill Tumor Cells without Affecting Healthy Cells

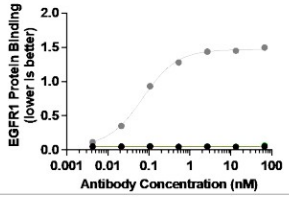
iBio EGFRvIII mAbs bind recombinant EGFRvIII →



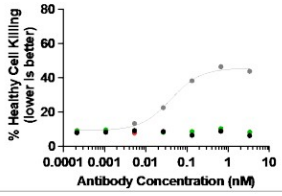
which leads to tumor cell killing



but not binding wild-type EGFR1 →

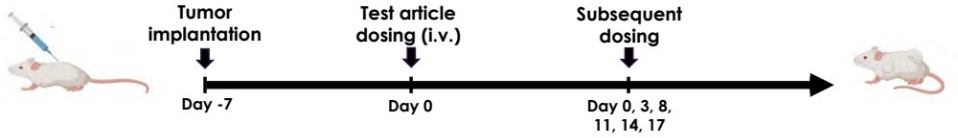


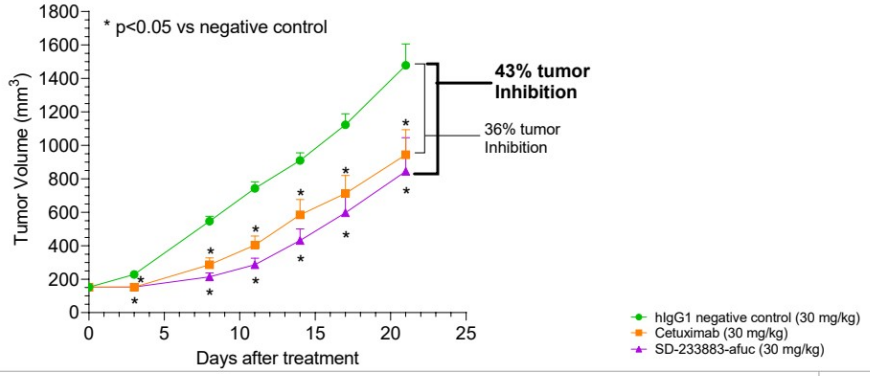
and thus not affecting healthy cells



- Negative control, EC_{50} = no binding
- Cetuximab, EC_{50} = 0.018 nM
- SD-233883, EC_{50} = 0.008 nM
- SD-710726, EC_{50} = 0.020 nM

iBio's EGFRvIII-Specific High-ADCC Antibody Inhibits Tumor Growth in an EGFRvIII Tumor Xenograft Mouse Model






Market-Tested Potential in Immuno-Oncology
Competitor Early-Stage Deals Signal Promising Opportunities
86

Market-Tested Potential: Immuno-Oncology Early-Stage Deals

Pre-2020

SEP 2018
CD25
Roche / Tusk Therapeutics*:
$81M upfront,
$677M milestones

SEP 2020
CCR8
Gilead / Jounce*:
$85M upfront,
$35M equity investment,
$685M milestones

JUL 2020
TROP-2
AstraZeneca / Daiichi*:
$1B upfront (some deferred),
$5B milestones

SEP 2020
TROP-2
Gilead / Immunomedics*:
acquired for **$21B**

2021

FEB 2021
PD-1 agonist
Merck / Pandion*:
acquired for **$1.85B**

JUN & DEC 2021
CCR8
Fibrogen / HiFiBio+:
$25M option fee,
$35M option exercise,
$1.1B milestones

JUL 2021
CD3
Eli Lilly / Merus+:
$40M upfront, **$20M** investment
$540M milestones

JUL 2021
CD3
Amgen / Teneobio*:
$900M upfront,
$1.6B milestones

DEC 2021
ShieldTx
Sanofi / Amunix*
acquired for **$1B**, **$225M** milestones

2022

MAY 2022
EGFRvIII
Taiho / Cullinan Oncology+:
$275M upfront, **$130M** milestones

AUG 2022
PD-1 agonist
Gilead / Mirobio*:
acquired for **$405M**

SEP 2022
EGFRvIII
Seagen / LAVA Therapeutics+:
$50M upfront, **$650M** milestones

OCT 2022
CD3
Gilead / MacroGenics+:
$60M upfront, **$1.7B** milestones

NOV 2022
ShieldTx
Regeneron / Cytomx+
$30M upfront, **$2B** milestones

2023

JAN 2023
CD3
GSK / WuXi Biologics+:
$40M upfront,
$1.46B milestones

JAN 2023
CCR8
Gilead / Jounce+:
$67M for remaining stake in CCR8 program

APR 2023
TROP-2
BioNTech / Duality Biologics+:
$170M upfront, **$1.5B** milestones

APR 2023
EGFRvIII
Pierre Fabre / Scorpion+:
$65M upfront,
$553M milestones

JUN 2023
CCR8
Coherus / Surface Oncology*:
acquired for **$65M**

2024

DEC 2023
CD25
Neoleukin / Neurogene*:
all-stock transaction

JAN 2024
TROP-2
Biohaven / Pyramid*:
acquired for **$55M**

MAR 2024
TROP-2
Merck / Harpoon*:
acquired for **$680M**

APR 2024
MUC16
Regeneron / 2Seventy Bio:
multi-asset purchase for
$5M upfront w/milestone