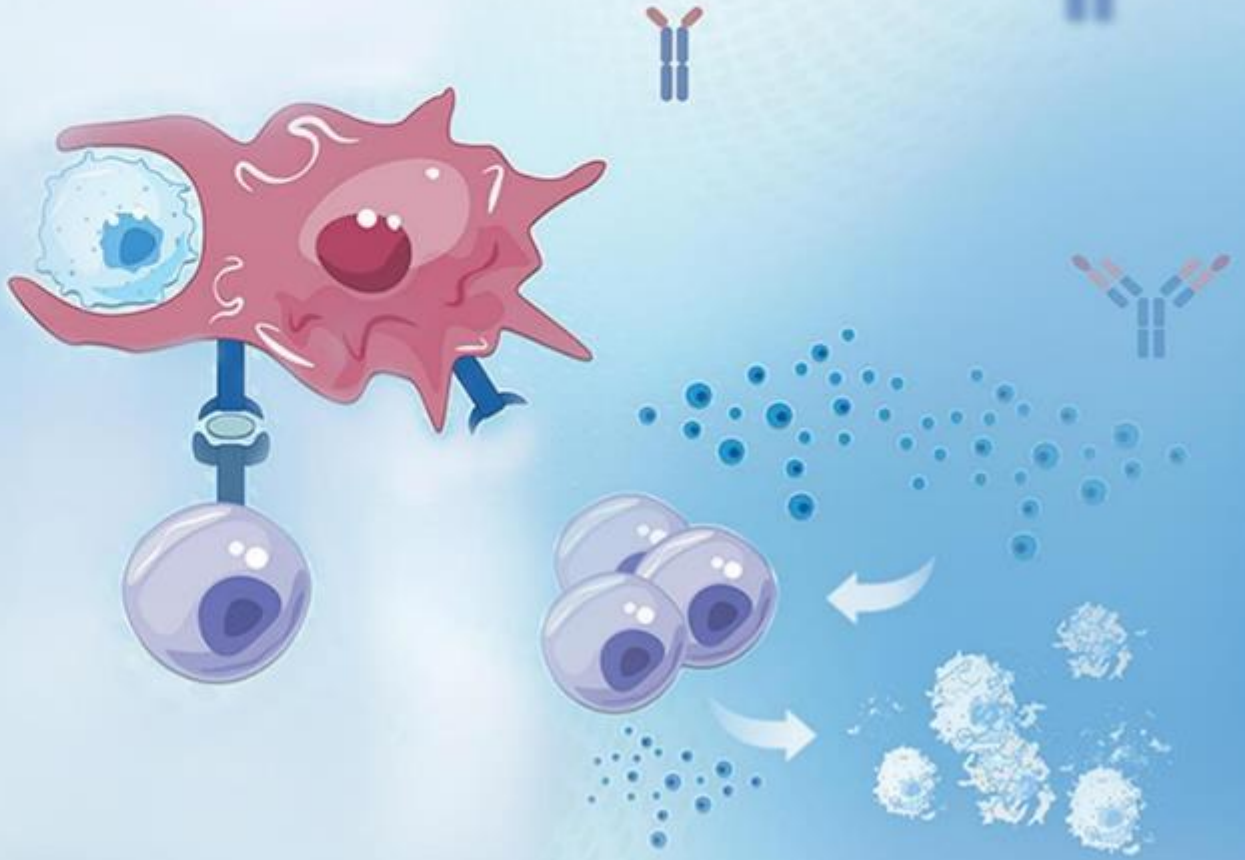
宜明昂科
ImmuneOnco

Corporate Presentation

March 2024



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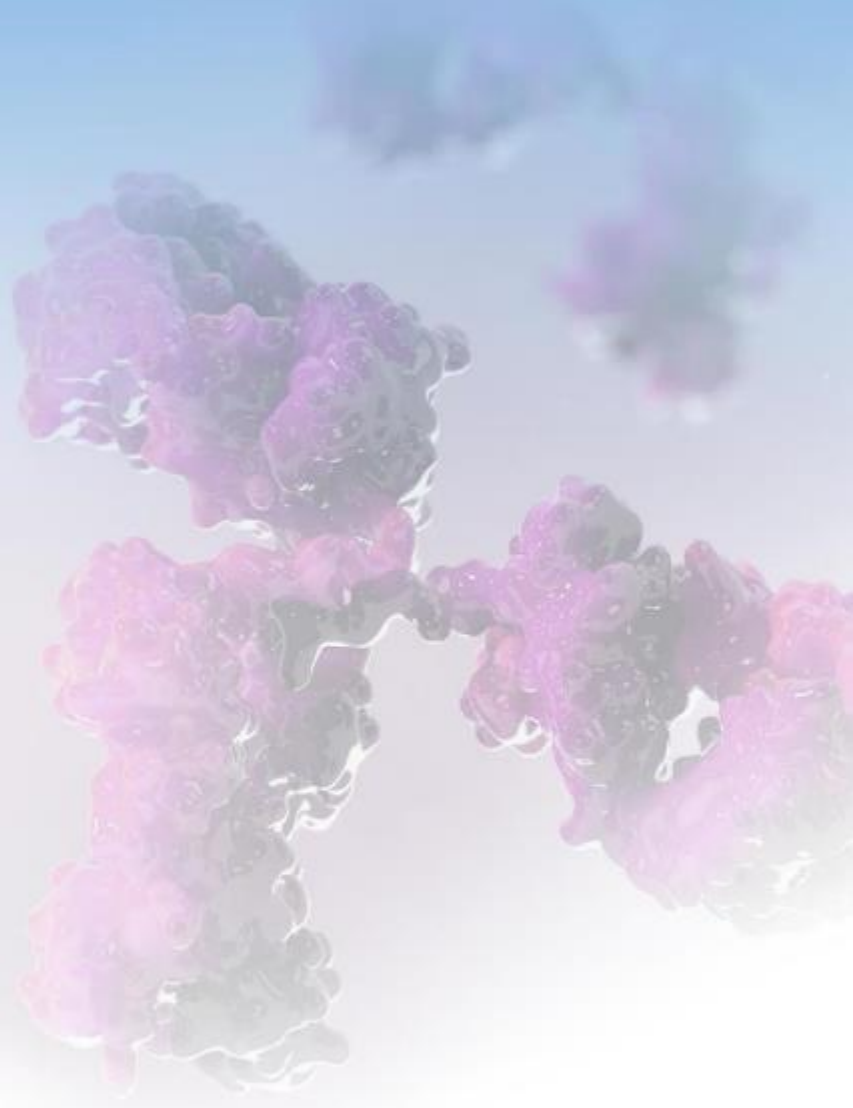
SECTION 1

Company Overview



Mission:

“TO DEVELOP BEST-IN-CLASS AND/OR FIRST-IN-CLASS ANTI-CANCER DRUGS FOR CANCER PATIENTS AROUND THE WORLD”

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Key Milestones

- Steady team with **10+** years coordination
- **24** IND approvals from the NMPA and the FDA
- **33** issued patents, **1** allowed patent applications,
- **20** pending patent applications
- **10** ongoing clinical programs, **3** IND/IND-enabling stage programs

Pipeline

2015-2020

- 2015: ImmuneOnco was incorporated in the PRC
- 2019: The first patient of the Phase I clinical trial for **IMM01** was enrolled
- 2019: IND approval for **IMM0306** from NMPA
- 2020: Established the **pilot production line** with 200L GE single-use mammalian cell bioreactors
- 2020: IND approval for **IMM2510** from NMPA

2021

IMM01:
- IND approval by NMPA for the Phase Ib/II clinical trial of **IMM01's combination with each of azacitidine and inetetamab**
- Phase II initiation for **IMM01 monotherapy**

IMM0306:
- IND approval by FDA

IMM2902:
- IND approval by NMPA and FDA

IMM27M:
- IND approval by NMPA

2022-2023

IMM01:
- Phase Ib/II clinical trial initiation for **IMM01's combination with azacitidine** and **dosed the first patient**
- Phase II trial initiation in China for **IMM01's combination with tislelizumab**

IMM2520:
- IND approved by NMPA and FDA and dosed the first patient for the Phase I clinical trial in China

IMM40H:
- IND approved by NMPA and FDA

IMM2902:
- Phase I clinical trial dosed their respective first patient in China and US; received Fast Track Designation from FDA

IMM2510:
- IND approved by NMPA for IMM2510 and chemo combo's Phase Ib/II trial as well as the Phase Ib/II trial for IMM2510 and IMM27M's combination

IMM0306:
- Phase Ib/IIa trial initiation in China for **IMM0306's combination with lenalidomide** and dosed its first patient

2015 | 2016 | 2017 | 2018 | 2019 | 2020 | 2021 | 2022 | 2023

Financing

2017: Series Pre-A, RMB30 MM	2018: Series A, RMB90 MM	2020: Series Pre-B, RMB40 MM	2020: Series B, RMB240 MM	2021: Series B+, US$65 MM	2022: Series C, US$87.5 MM	2023: IPO, US$43 MM

Key Investors

Lilly Asia Ventures 礼来亚洲基金 | LYFE CAPITAL | 龙磐投资 LAPAM CAPITAL | 上海科创基金 Shanghai Sci-Tech Innovation Center Capital | 阳光保险集团 Sunshine Insurance Group | 南京星健睿赢 | 荣昌股权投资

Total amount of fund raised: ~$255MM

Management team

Wenzhi Tian, MD, EMBA
Founder, Chairman & CEO

- ***30+*** years academic and industrial experience in the field of immuno-oncology
- ***24*** IND approvals from the NMPA and the FDA
- ***27*** issued patents, ***21*** patent applications, and ***30+*** scientific publications

Qiying Lu, MD
CMO, SVP

Frank Xiaodong Gan, Pharm.D.
SVP, Clinical Development

Zikai Xiong, PhD
SVP, BD

Mr. Ruliang Zhang
Deputy General Manager
SVP, CMC & Registration

Song Li, BA, MS
VP, R&D

Mei Guan, BS, MS
Secretary of the Board

Integrated proprietary R&D platform

Efficiently Conduct Screening and Druggability Analysis → **Optimize Molecule Structure Design** → **Cost-effectively Manufacture High-quality Drug In-house** → **Next-generation Innovative Oncology Therapies**


Target Selection and Validation


Drug Discovery


High-throughput Screening


Molecule Design


Preclinical Studies


CMC and IND-enabling Capabilities


Clinical Development

Integrated in-house R&D Platform

Advanced Hybridoma Technology	High-throughput Screening	Proprietary mAb-Trap Bispecific Platform	Strong Immunoassay and Bioassay Technology	Efficient Cell Line Development	Robust CMC and Manufacturing Capacity
• Efficiently identify and improve antibody fragments with higher specificity, affinity and other best-suited properties		• Allowing for favorable binding affinity with tumor targets while preserving IgG1 Fc effector function • Ease of manufacturing, product stability, higher tier and protein yield			• Our stable R&D, CMC and regulatory affairs teams with 62 members

Pilot manufacturing: 200L/250L bioreactors

Global Rights

- **24** IND approvals from the NMPA and the FDA
- **33** Issued patents
- **1** Allowed patent applications
- **20** Pending patent applications

Comprehensive Pipeline Covering Oncology and non-Oncology Therapeutic Areas

Program [1]	Target (Modality)	Indication(s)	Discovery	Preclinical	IND/IND-Enabling	Phase Ia/I	Phase Ib/II	Phase III/ Pivotal	Current Status / Upcoming Milestone	Commercial Rights
IMM01										
IMM01 + Azacitidine	CD47 (SIRPα-Fc fusion protein)	MDS, AML, CMML[2]	China (NMPA)						Phase Ib/II commenced in January 2022; expect to complete Phase II and initiate CDE discussion in Q1 2024	Global
IMM01 + Tislelizumab	CD47+PD-1	cHL[3], Solid tumor	China (NMPA)						Phase Ib/II commenced in May 2022; communicated with CDE on Phase III trial design in January 2024	Global
IMM0306 Monotherapy	CD47xCD20 (Bispecific)	R/R FL&MZL	China (NMPA)						Phase II trial commenced in Q2 2023	Global
IMM0306 + Lenalidomide	CD47xCD20 (Bispecific)	B-NHL	China (NMPA)						Phase Ib/IIa commenced in June 2023 in China	Global
IMM2510 Monotherapy	VEGFxPD-L1 (Bispecific)	STS	China (NMPA)						Phase Ib/II commenced in November 2023 in China	Global
IMM2510 + Chemo	VEGFxPD-L1 (Bispecific)	1L TNBC, 1L NSCLC	China (NMPA)						IND approved in China in November 2023	Global
IMM2510 + IMM27M	VEGFxPD-L1 (Bispecific) + CTLA-4	2L HCC, TNBC	China (NMPA)						IND approved in China in October 2023	Global
IMM27M	CTLA-4 ADCC+ (mAb)	Solid tumors	China (NMPA)						Phase I completed in September 2023 in China and RP2D was identified as 5mg/kg	Global
IMM2902	CD47xHER2 (Bispecific)	HER2-positive and low-expressing solid tumors	China (NMPA), US (FDA)						Phase Ia commenced in February 2022 in China and in June 2022 in the U.S.	Global
IMM2520	CD47xPD-L1 (Bispecific)	Solid tumors	China (NMPA), US (FDA)						IND approved in China and the U.S. in Q4 2022; Phase I commenced in China in March 2023 and 5th cohort ongoing	Global
IMM47	CD24 (mAb)	Solid tumors	China (NMPA), US (FDA)						IND approved in China and the U.S. in October and December in 2023; Phase I commenced in September 2023 in Australia	Global
IMM40H	CD70 (mAb)	Liquid/Solid tumors	China (NMPA), US (FDA)						IND approved in China and the U.S. in August 2022	Global
IMM4701	CD24xCD47 (Bispecific)	Solid tumors	China (NMPA), US (FDA)						IND-enabling	Global
IMC-002 (IMM0306)	CD47xCD20 (Bispecific)	Undisclosed							Filed IND application with the NMPA in March 2024	Global
IMC-001 (IMM01)	CD47 (SIRPα-Fc fusion protein)	Undisclosed							IND-enabling	Global
IMC-003 (IMM72)	ActRIIA (Fc-fusion protein)	PAH, Undisclosed							IND-enabling in one year	Global
IMC-004 (IMM7211)	ActRIIA x Non-disclosed (Bispecific)	Undisclosed							IND-enabling in one and a half year	Global

Innate Immunity Targets | **Innate and Adaptive Immunity Targets** | **Adaptive Immunity Targets** | **Autoimmune and metabolic disease**

Source: Company Data

Notes:

(1) All of the Company's clinical- and IND-stage drug candidates are classified as Category 1 innovative drugs, and preclinical- and discovery-stage drug candidates are expected to be classified as Category 1 innovative drugs, in accordance with relevant laws and regulation in China

(2) The cohort-expansion trials of this combination are mainly designed to target the first-line treatment of higher-risk MDS (patients who fall into higher-risk group categories in the original or revised International Prognostic Scoring System), unfit AML (individuals of older age with AML who are considered not eligible for intensive treatment approaches), and CMML. On November 8, 2023, the combination therapy of IMM01 and Azacitidine was granted the orphan-drug designation by the FDA for the treatment of CMML

(3) This combination of IMM01 and tislelizumab targets all subtypes of cHL

Highlights of Projects at Clinical Stages

IMM01
(SIRPα-Fc)

- **IMM01 + Azacitidine, Ph II** (CMML, MDS, AML)
- Best response
  - ✓ **1L CMML : ORR 84.6%** (1)
  - ✓ **1L MDS: ORR 89.3%** (1)
- **IMM01 + Tislelizumab, Ph II** (solid tumors and lymphoma)
- **PD-1 failed R/R cHL: ORR 66.7%**
- Issued patents in China, the U.S., Japan and Europe

IMM0306
(CD47xCD20 mAb-Trap)

- **IMM0306 + Lenalidomide, Ph Ib/IIa** (FL, MZL)
- Best response: **1CR, 4PRs and 1SD out of 7 evaluable patients.**
- No DLT observed at 2mg/kg
- Issued patents in China, the U.S., Japan and Europe
- Potentials in other indications SLE, RA, MS can also be explored

IMM2510
(VEGFxPD-L1 mAb-Trap)

- **Ph II** (Monotherapy, STS)
- **IND approved for Ph Ib/II:**
  + Chemo: 1L TNBC, NSCLC
  + 27M: 2L HCC, TNBC
- No DLT at 20mg/kg
- Best response**: 3 PRs,** including two NSCLC patients and one thymus adenosquamous carcinoma patient； **4SDs with tumor shrinkage of over 15%**
- Issued patents in China, the U.S. and Japan

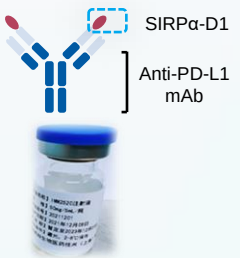
IMM27M
(CTLA-4, ADCC-enhanced IgG1)

- **Ph I**
- RP2D: 5mg/kg
- Best response: **2PRs, 3SDs with tumor shrinkage**
- Patent application planning

IMM2902
(CD47xHER2 mAb-Trap)

- **Ph I** (4.0mg/kg finished, higher dose ongoing)
- Phase I in China and the U.S.; Fast Track Designation from the FDA
- Issued patents in the U.S., Japan and Europe

IMM2520
(CD47xPD-L1 mAb-Trap)



- **Ph I** (FPI in 2023)
- Issued patents in China, the U.S. and Japan

IMM47
(CD24, ADCC-enhanced IgG1)

- **Ph I** (FPI in Australia in 2023)
- Issued patents in China, the U.S. and Japan

Notes:
1. Patients treated for over 6 months

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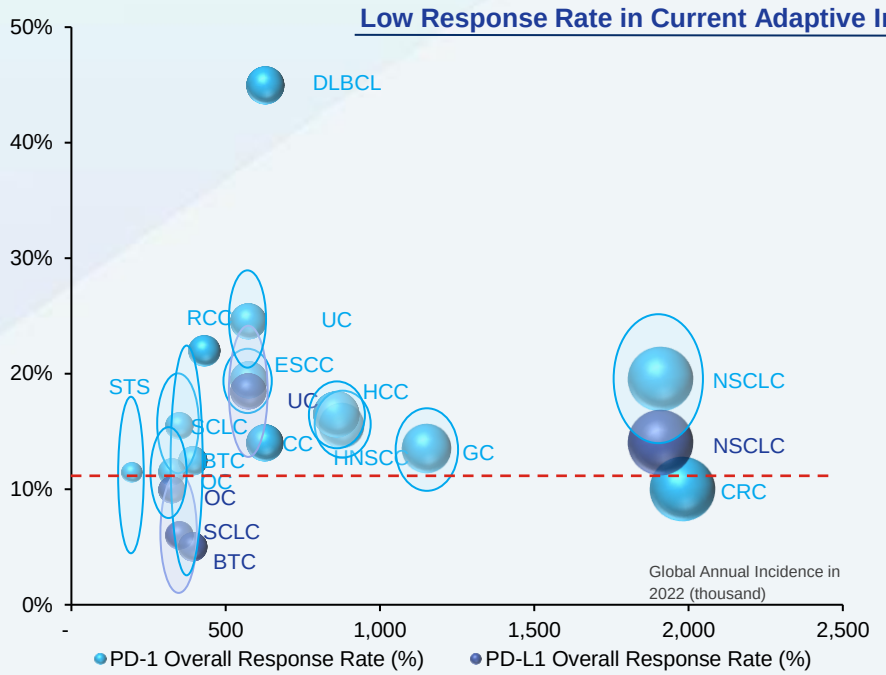
SECTION 2

Our Approach

Breaking Drug Resistance and Broaden Benefit

Although PD-1/PD-L1 Inhibitors Have Been Hugely Successful with Approvals in Almost All Major Tumor Indications, Its Monotherapy Response Rates Are Generally Below 20%



The current approved immunotherapies primarily target T-cell immune checkpoints, including **PD-1/PD-L1, CTLA-4 and LAG-3**

Only about **10% to 25%** of patients across almost all major cancer types respond to PD-1/PD-L1 inhibitor monotherapy

In 2035, the global immuno-oncology therapy market is projected to reach **US$340.4 billion**, accounting for **over 50%** of the total global oncology market

PD-1/PD-L1 Inhibitor Monotherapy

Low response rates[2] across almost all major cancer indications

	NSCLC	SCLC	CRC	GC	HNSCC	HCC	ESCC	BTC	RCC	OC	CC	UC	STS	DLBCL
PD-1	19-20%	12-19%	<10%	13-14%	13-16%	16-17%	19-20%	3-22%	22%	8-15%	14%	20-29%	5-18%	45%
PD-L1	14%	2-10%						5%		10%		13-24%		

Notes:
1. Bubble size indicates annual incidence of diseases. (1) The response rates are based on the latest label from FDA and NMPA except for CRC, GC, SCLC, OC, BTC and STS, which are based on the published clinical results. (2) Only monotherapy clinical results are listed. (3) Results of adjuvant therapy are excluded. Results may vary from different cancer sub-types or clinical trials. (4) The clinical results listed are from general cancer population regardless of PD-L1 expression, except for the ORR of CC, which is restricted in PD-L1 positive population (combined positive score (CPS)≥1).

Source: Frost & Sullivan

Breaking Drug Resistance and Broaden Benefit

Research Has Shown PD-1/PD-L1 Inhibitors Are Only Expected to be Effective in Hot Tumors, Corresponding to its Limited Monotherapy Response Rates

Type I

Lack of TILs in TME

Innate immune activation to induce inflammation and attract adaptive immune cells

Type II

Overregulation of activated TILs

Type III

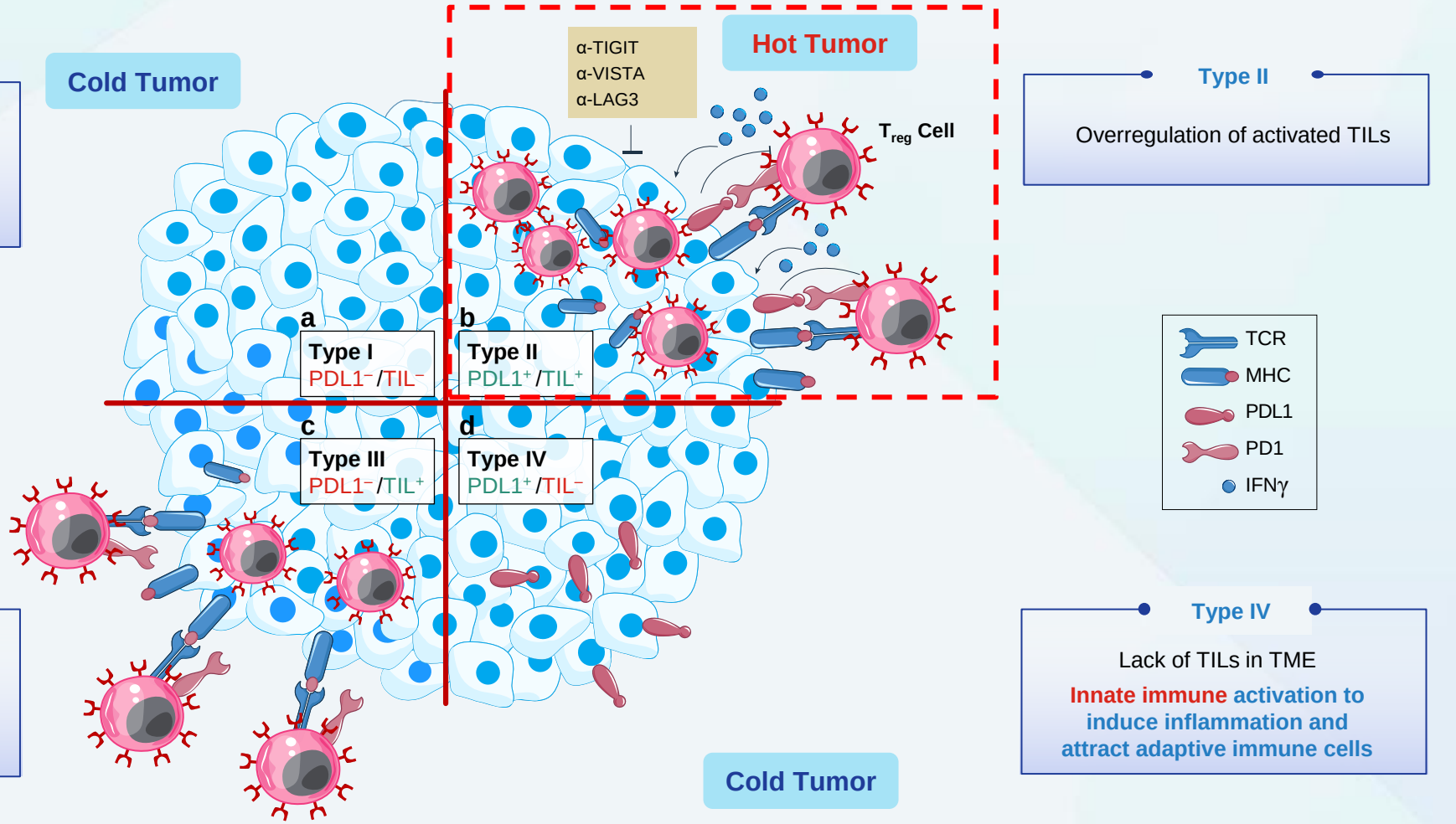
Dysfunctional TILs activation

Activation of antigen specific T cells through antigen presenting cells

Type IV

Lack of TILs in TME

Innate immune activation to induce inflammation and attract adaptive immune cells



Source: Frost & Sullivan, literature review

Breaking Drug Resistance and Broaden Benefit

The Activation of Innate Immune Cells is Able to Attract T Cells into Tumor Microenvironment, Turning Cold Tumors to Hot Tumors, Significantly Improve the Response Rates of PD-1/PD-L1 Inhibitors

Overview of Innate and Adaptive Immune Systems

	Innate Immunity			Adaptive Immunity	
Activation Process	First line of defense, short response time, no need for antigen priming			Antigen priming required	
Key Immune Cell Type	Macrophage	NK cell	DC	T cell	B cell
Tumor Tissue Distribution[1]	20%-50%	5%-10%	3%-10%	10%-30%	3%-40%
Major Immune Checkpoint(s)	CD47/SIRPα, CD24/Siglec-10, PSGL-1, EP4	KIR family, CD94-NKG2A, CD24/Siglec-10, TIGIT, EP4	PD-1/PD-L1, CD47/SIRPα, EP4	PD-1/PD-L1, CTLA-4, LAG-3, TIM-3, TIGIT	CD40/CD40L, CD19, CD22
Major Immune Functions	• Macrophage-mediated phagocytosis • Attracting T cells to the TME • Antigen presentation • Trogocytosis	• NK cell-mediated cytolysis via the secretion of perforin and granzymes • Activating of T cells, macrophages and DCs through release of cytokines	• Attracting T cells to the TME • Antigen presentation	• T-cell mediated killing of tumor cell via exocytosis of cytotoxic granules (perforin, granzymes) and secretion of antitumor cytokines	• Antibody production • Cytokine secretion

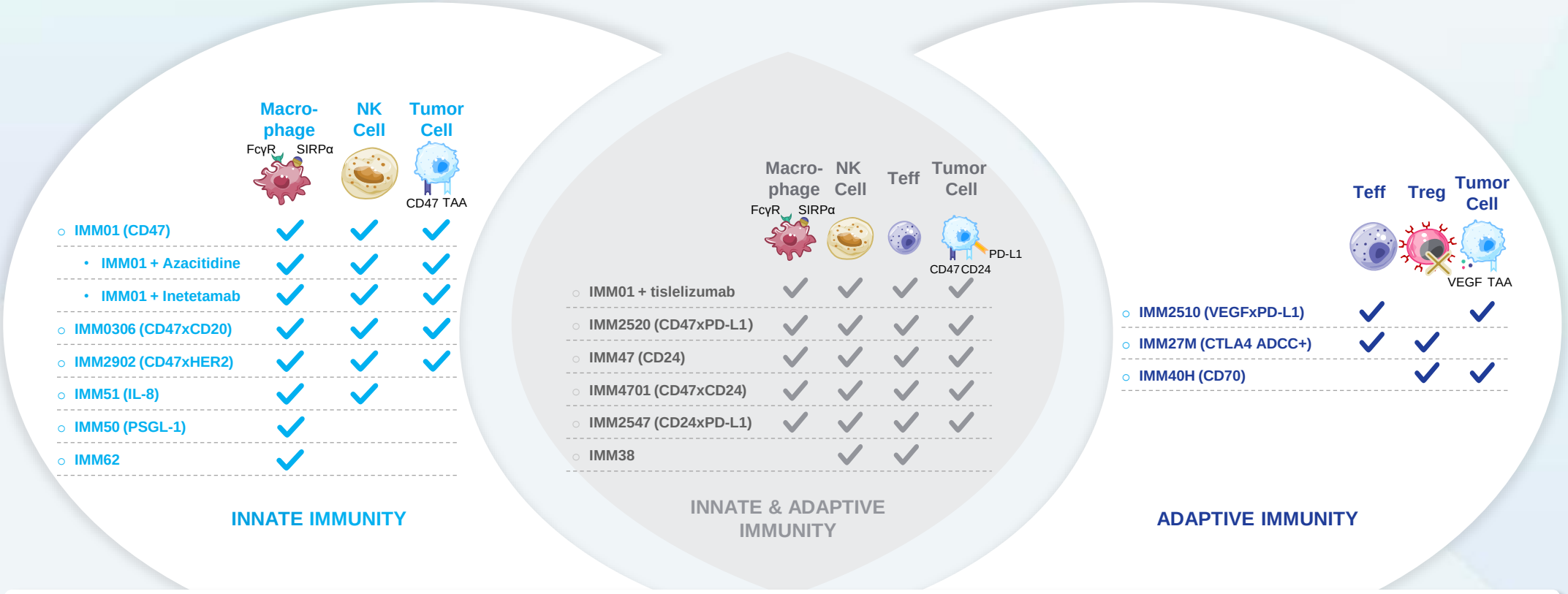
1 Attract T cells to the TME
2 Activate T cells through antigen presentation and release of cytokines

→ **Turning cold tumor into hot tumor**

Notes:
1. The tumor tissue distribution is the proportion of certain immune cells in different tumor tissues

Breaking Drug Resistance and Broaden Benefit

Our Pipeline Harnessing Both Innate and Adaptive Immunities



The Company stands out as one of the few biotechnology companies globally adopting a systematic therapeutic approach to harness both the innate and adaptive immune systems

Breaking Drug Resistance and Broaden Benefit

Deep and broad innate immunity-based portfolio targeting a wide range of solid and hematologic tumors to address critical unmet medical needs

CD47

CD47 is a critical macrophage checkpoint that plays a broad role in cancer immune evasion across many cancer types

4 drug candidates in clinical stage

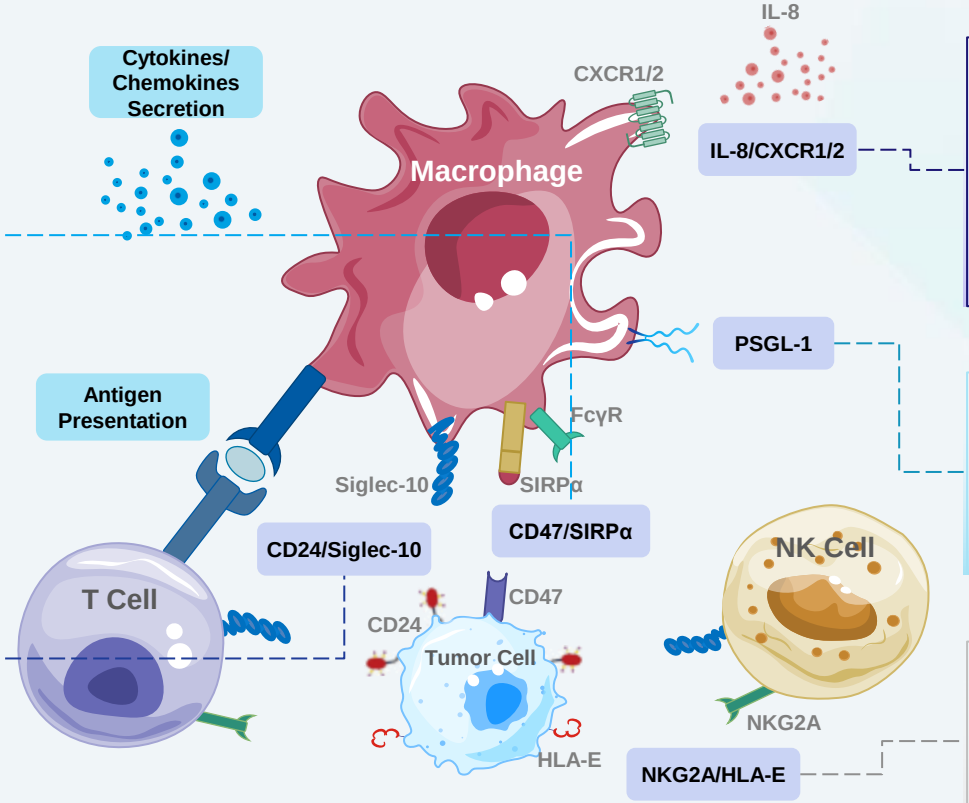
Numerous other discovery- and preclinical-stage molecules

CD24

CD24 is a promising innate immune checkpoint widely expressed on numerous types of solid tumor cells

2 drug candidates in IND-enabling-stage and CMC stage

Multiple other discovery- and preclinical-stage bispecific molecules



IL-8

IL-8 is a chemokine that mediates the inflammatory process and functions as a significant regulatory factor within the TME

We are evaluating **IMM51**, an IL-8 mAb, in a number of preclinical studies

PSGL-1

We are investigating PSGL-1, an adhesion molecule expressed by macrophage and many other hematopoietic cells

NKG2A

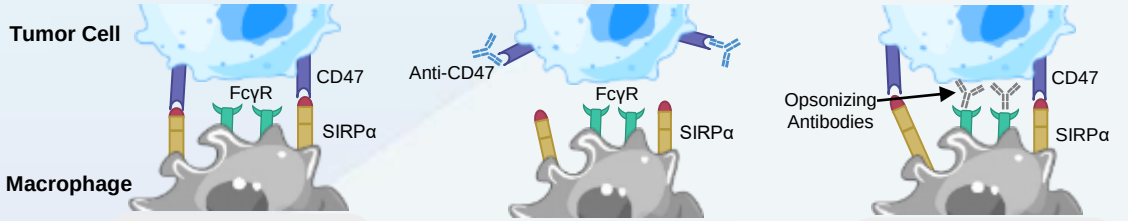
We are exploring NKG2A, an inhibitory cell surface molecule mainly expressed on both NK cells and CD8+ T cells

Source: Company, literature review, Frost & Sullivan

Breaking Drug Resistance and Broaden Benefit

Scientifically and structurally differentiated molecule design based on our “drug-by-design (DbD)” concept to achieve potent efficacy and favorable safety

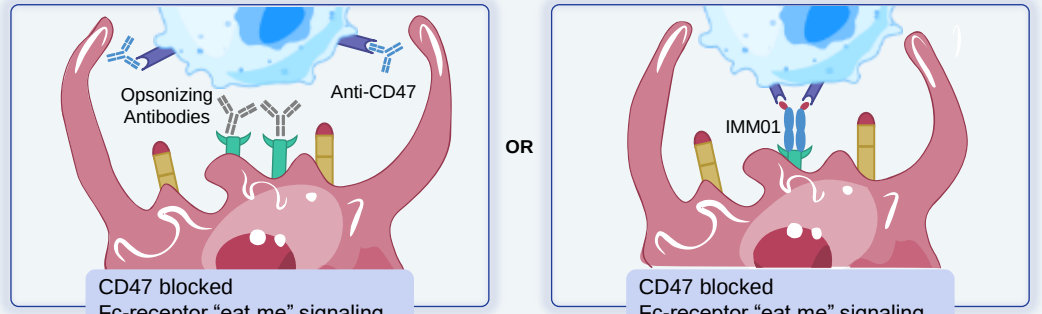
Mechanism of Action in the CD47-SIRPα Signaling Pathway



CD47 “don’t eat me” signaling
No “eat me” signaling
Poor macrophage activation

CD47 blocked
No “eat me” signaling
Limited macrophage activation

CD47 “don’t eat me” signaling
Fc-receptor “eat me” signaling
Limited macrophage activation



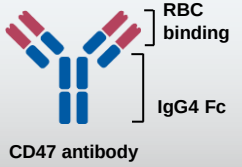
CD47 blocked
Fc-receptor “eat me” signaling
Full macrophage activation

CD47 blocked
Fc-receptor “eat me” signaling
Full macrophage activation

Guided by the DbD concept, our **differentiated molecule design** tackles scientific barriers presented by the selected immune checkpoints, leading to an **optimized safety and efficacy profile**

How Our Differentiated Design Improves Safety and Efficacy

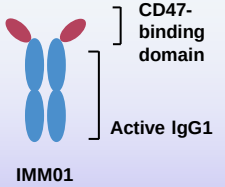
The safety concerns around CD47 pose considerable challenge



CD47 antibodies inevitably bind with RBCs, generating issues including **severe blood toxicity**, **antigenic sink**, and **decreased potency**

Most CD47 antibodies resort to an **IgG4 Fc with weak FcγR engagement**, unable to deliver the “eat me” signal

Our differentiated design allows full macrophage activation



Engineered CD47-binding domain with **no RBC binding** *in vitro;* Modification of deglycosylation of the binding domain **mitigates immunogenicity**

No RBC binding enables usage of **potent IgG1 Fc**

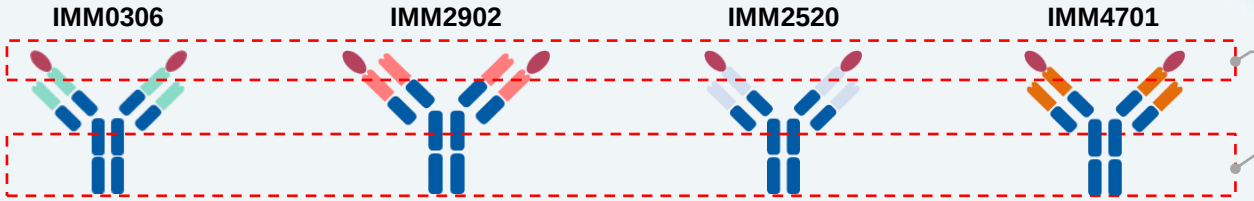
Breaking Drug Resistance and Broaden Benefit

Scientifically and structurally differentiated molecule design based on our “drug-by-design (DbD)” concept to achieve potent efficacy and favorable safety (cont’d)

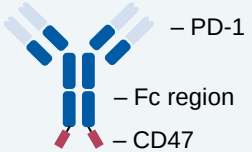
How Our Differentiated Design Improves Safety and Efficacy – CD47-based Bispecific Molecules

The structure of our bispecific molecules was **deliberately designed through a series of rigorous studies and tests guided by our “DbD” concept** on various aspects, including synergy between targets, tailored molecule structure, expected dosing level, stability, and ease of manufacturing

We developed our CD47-based bispecific molecules leveraging our **mAb-Trap platform** – all having symmetrical structure with the same engineered CD47-binding fragment used in IMM01



HX009 (Hans Bio)



Certain molecules connect the CD47-binding domain to the Fc end, which could **interfere with CD47-binding epitope located at the N-terminal of SIRPα fragment**, and further **disrupt immune activation resulted from Fc-FcγR engagement**

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SECTION 3

Oncology Program Overview

Our differentiated approach to developing therapies targeting CD47-based and other promising innate and adaptive immune checkpoints

Our Approach to Seize Market Opportunities

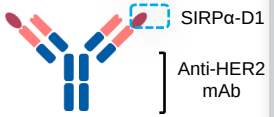
Innate Immunity

IMM01

- **IMM01** (CD47)
  - **IMM01 + Azacitidine**
  - **IMM01 + Inetetamab**
- **IMM0306** (CD47xCD20)

IMM0306

IMM2902



- **IMM2902** (CD47xHER2)
- **IMM51** (IL-8)
- **IMM50**
- **IMM62**

Innate & Adaptive Immunity

IMM2520

- **IMM01 + tislelizumab**
- **IMM2520** (CD47xPD-L1)
- **IMM47** (CD24)

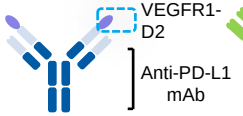
IMM47

IMM4701

- **IMM4701** (CD47xCD24)
- **IMM2547** (CD24xPD-L1)
- **IMM38**

Adaptive Immunity

IMM2510



IMM27M

IMM40H

- **IMM2510** (VEGFxPD-L1)
- **IMM27M** (CTLA4 ADCC+)
- **IMM40H** (CD70)

Wide Range of Indications with Unmet Need

Achieve Better Therapeutic Efficacy and Safety Profiles

Notes:
ADCC refers to antibody-dependent cellular cytotoxicity; NSCLC refers to non-small cell lung cancer; SCLC refers to small cell lung cancer; HNSCC refers to head and neck squamous cell carcinoma; HCC refers to hepatocellular carcinoma; CRC refers to colorectal cancer; GC refers to gastric cancer; OC refers to ovarian cancer; ESCC refers to esophageal squamous cell carcinoma; UC refers to urothelial carcinoma; B-NHL refers to B-cell non-Hodgkin lymphoma; AML refers to acute myeloid leukemia; MDS refers to myelodysplastic syndrome; CMML refers to chronic myelomonocytic leukemia

CD47-Targeted Drug Development

Deep understanding, scientific thinking, and sophisticated molecular designing will set the basis for differentiated CD47-targeted drug development

Challenges for CD47-Targeted Drug Development

- Feb 2022: Partial clinical holds on Magrolimab clinical trials
- Aug 2022: AbbVie discontinues clinical trial of anti-CD47 mAb
- Jan 2023: Arch Oncology gives up on CD47
- Jul 2023: Gilead discontinued Phase III study of magrolimab plus azacitidine in Higher-Risk MDS
- Feb 2024: Gilead discontinued Phase III study of magrolimab in AML

FDA Puts Clinical Hold on Trials Assessing Magrolimab/Azacitidine Combo in AML/MDS

Feb 2, 2022
Hayley Virgil

Due to an imbalance of investigator-reported unexpected adverse reactions, the FDA placed a partial clinical hold on all trials examining the combination of magrolimab and azacitidine in acute myeloid leukemia and myelodysplastic syndrome.

A partial clinical hold has been placed by the FDA on studies assessing the use of magrolimab and azacitidine in acute myeloid leukemia (AML) and myelodysplastic syndrome (MDS), according to a press release from developer Gilead Sciences.

The hold was implemented because of a notable imbalance of investigator-reported unexpected adverse reactions across different study arms. The partial hold is going into effect globally and will apply to any trials utilizing the combination until further data are gleaned, although no clear patterns in adverse reactions or novel safety signals have been observed.

AbbVie to discontinue phase 1 trial for I-Mab's anti-CD47 therapy for treatment of cancers

Aug. 16, 2022 5:57 PM ET | **I-Mab (IMAB)** | ABBV | By: **Anuron Mitra**, SA News Editor | 3 Comments

narvikk/iStock via Getty Images

Chinese biotech I-Mab (NASDAQ:IMAB) on Tuesday said its U.S. partner AbbVie (ABBV) would discontinue a phase 1b study evaluating a combination treatment including its anti-CD47 antibody therapy lemzoparlimab for two types of cancers.

Scoop: Roche-backed startup gives up on CD47, lays off all employees

Kyle LaHucik
Associate Editor

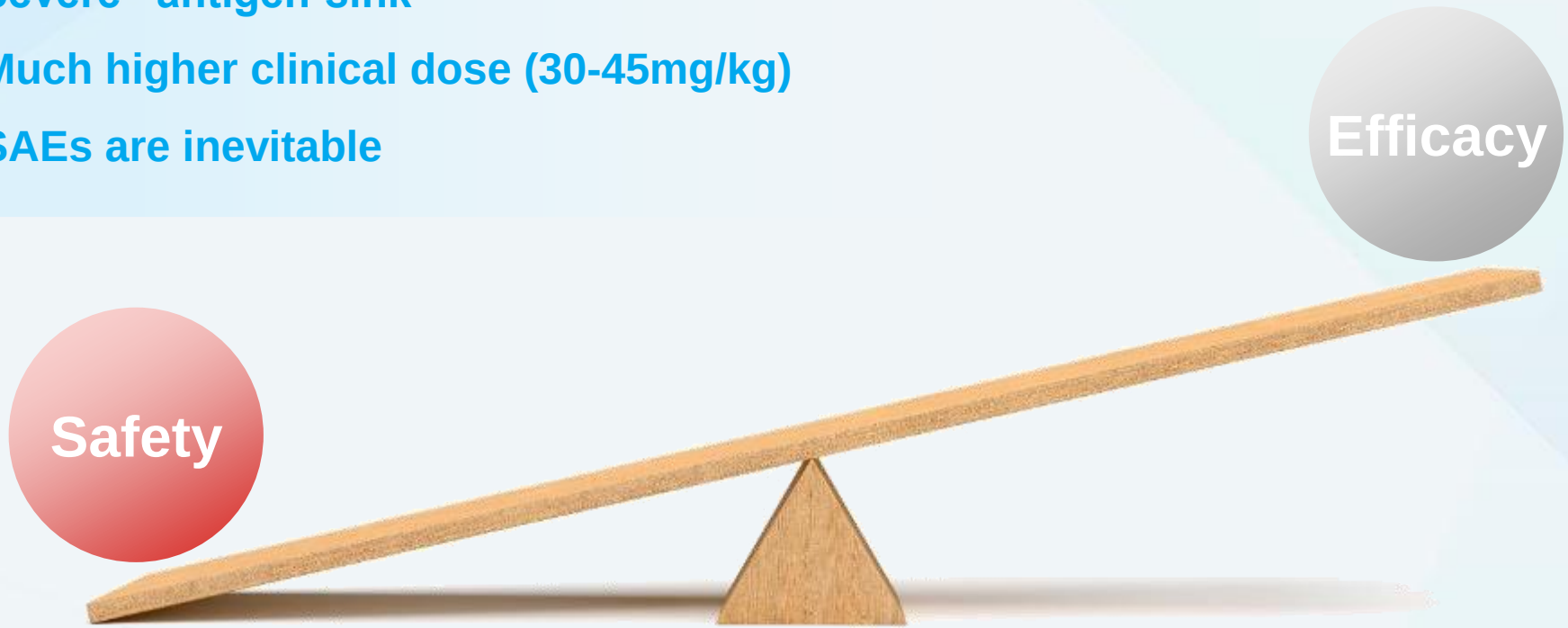
Arch Oncology ended its work on developing an anti-CD47 antibody and most employees have left the company, *Endpoints News* has learned.

The Brisbane, CA, and St. Louis biotech scrapped clinical development of the antibody, dubbed AO-176, according to an automatic reply email from a former clinical operations director.

Challenges for CD47-Targeted Drug Development (cont'd)

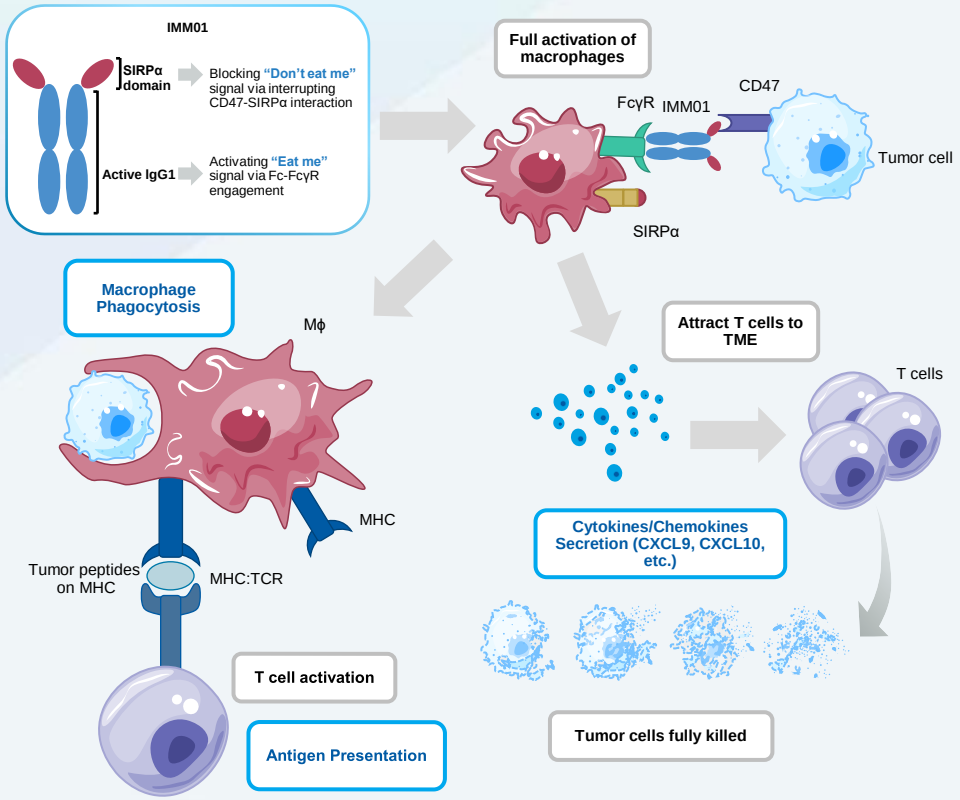
CD47 Antibody

- Target affinity is too high
- Severe "antigen-sink"
- Much higher clinical dose (30-45mg/kg)
- SAEs are inevitable



Our Differentiated Approaches

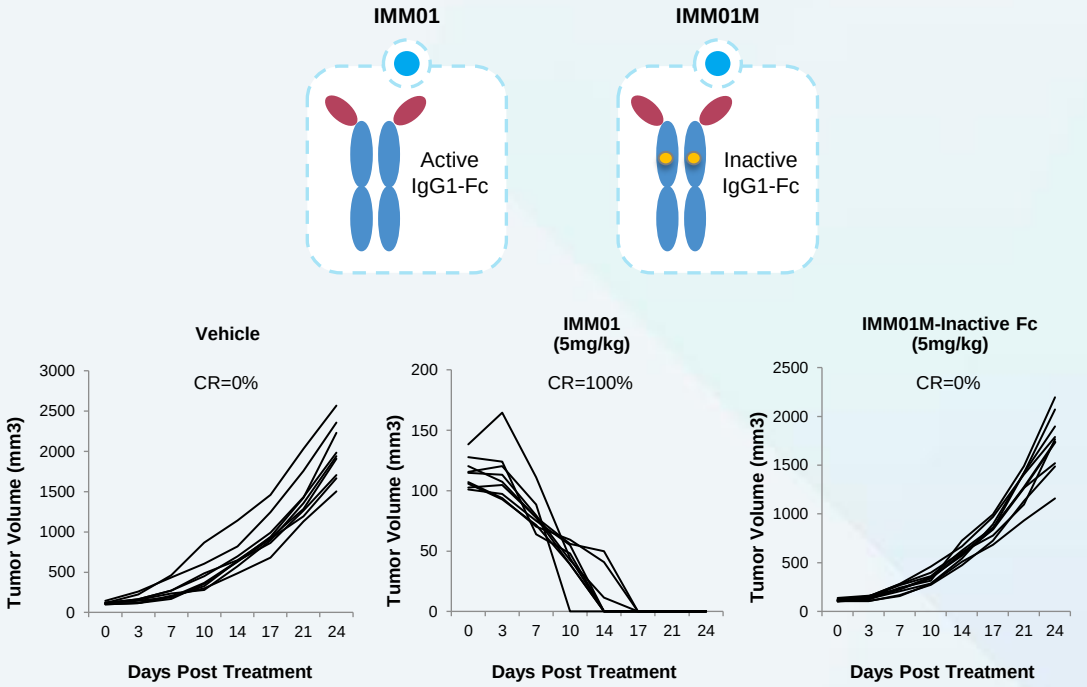
Overview and Competitive Advantage of IMM01



Notes:
MHC refers to major histocompatibility complex

Source: Company Data

In Vivo Efficacy of IMM01 is Dependent on Effective Fc Function (HL-60 xenograft model)

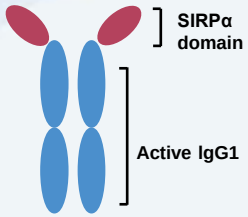


Notes: IMM01M has an engineered mutant inactive IgG1 Fc.

Our Differentiated Approaches (cont'd)

Overview and Competitive Advantage of IMM01

Overview

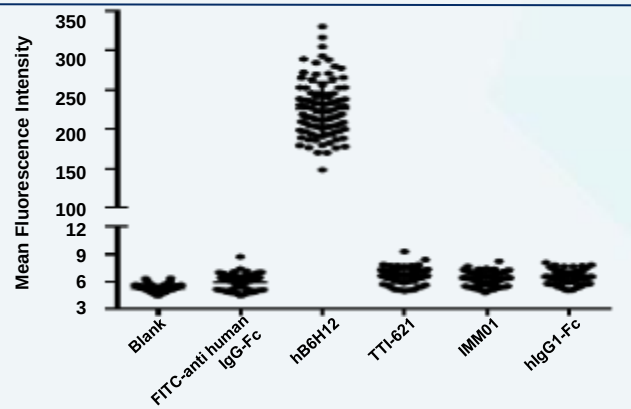


- First SIRPα-Fc fusion protein to enter the clinical stage in China
- Improved safety profile with engineered CD47-binding domain of human SIRPα to avoid human RBC binding
- Designed to fully activate macrophages through a dual mechanism
- Encouraging single-agent efficacy at relatively low dose, with CR observed

Source: Company Data

Competitive Advantage of IMM01 Monotherapy - Safety

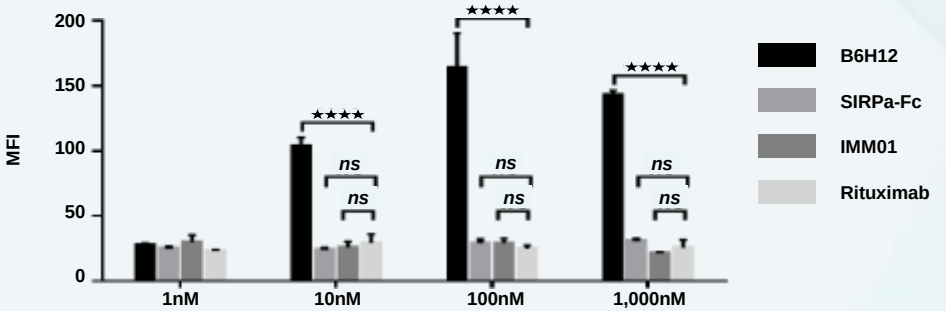
Human RBC Binding Analysis of IMM01



Donor Characteristics (n=100)
Male (n=62)
Female (n=38)
Type A blood group (n=29)
Type B blood group (n=31)
Type AB blood group (n=10)
Type O blood group (n=30)
hB6H12: 500nM
TTI-621: 5000nM
IMM01: 5000nM
hIgG1-Fc: 5000nM

Notes: B6H12 is a CD47-based antibody that serves as the control.

Phagocytosis Against Human RBC

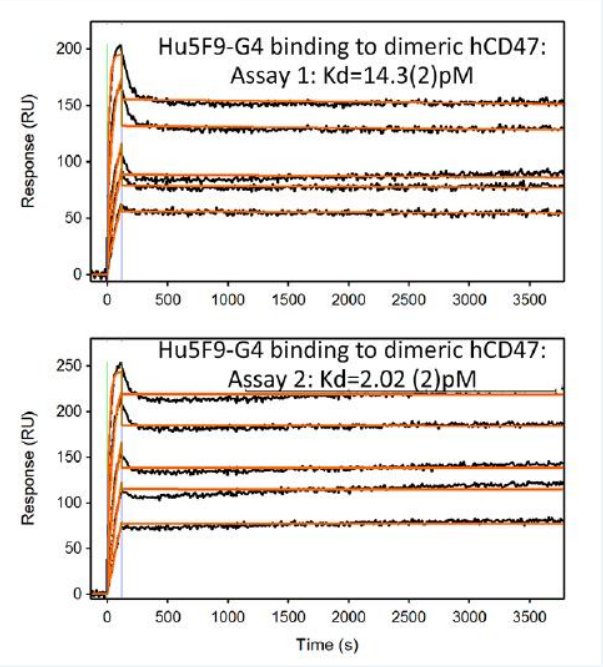


Notes: B6H12 is a CD47-based antibody that serves as the control.

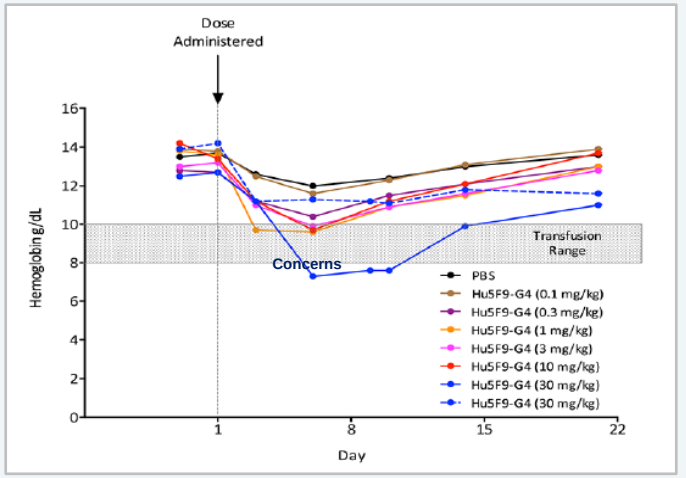
Our Differentiated Approaches (cont'd)

Magrolimab Has Very High Target Affinity and RBC Binding Activity

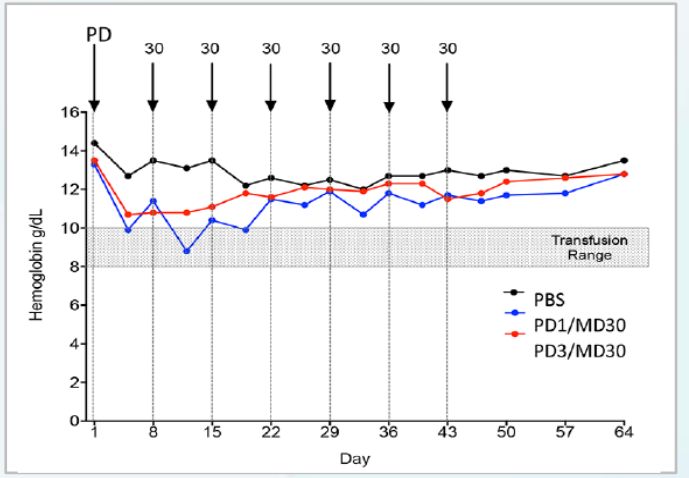
Target affinity assay



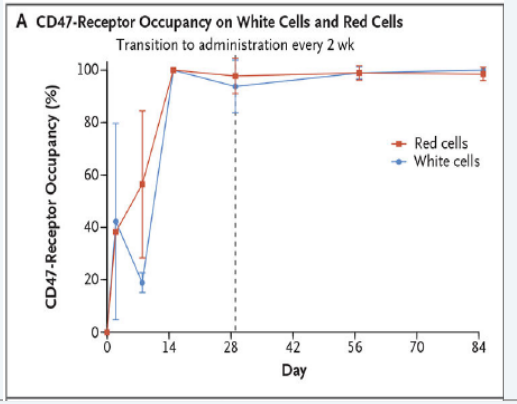
Without priming dose



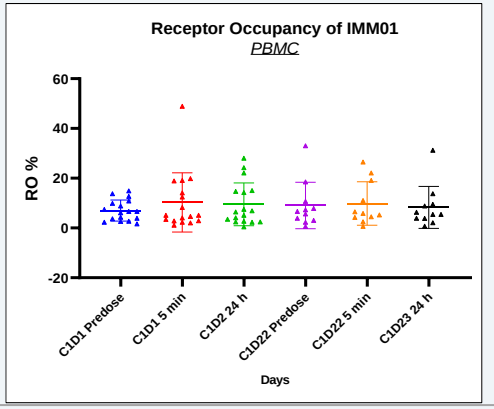
With priming dose (1mpk, 3mpk)



Magrolimab Receptor Occupancy (RO)



IMM01 Receptor Occupancy (RO)



Magrolimab: KD = 2-14.3pM

Timdarpacept (IMM01): KD = ~3nM

Source: Liu et al. PLoS One. 2015 Sep 21;10(9):e0137345.

Concerns

- **Bind to RBC, leading to hemolysis**
- **Too high target affinity, causing severe "antigen sink"**
- **High clinical dose is required due to the "antigen sink"**
- **High clinical dose will inevitably results in severe adverse event (SAE)**

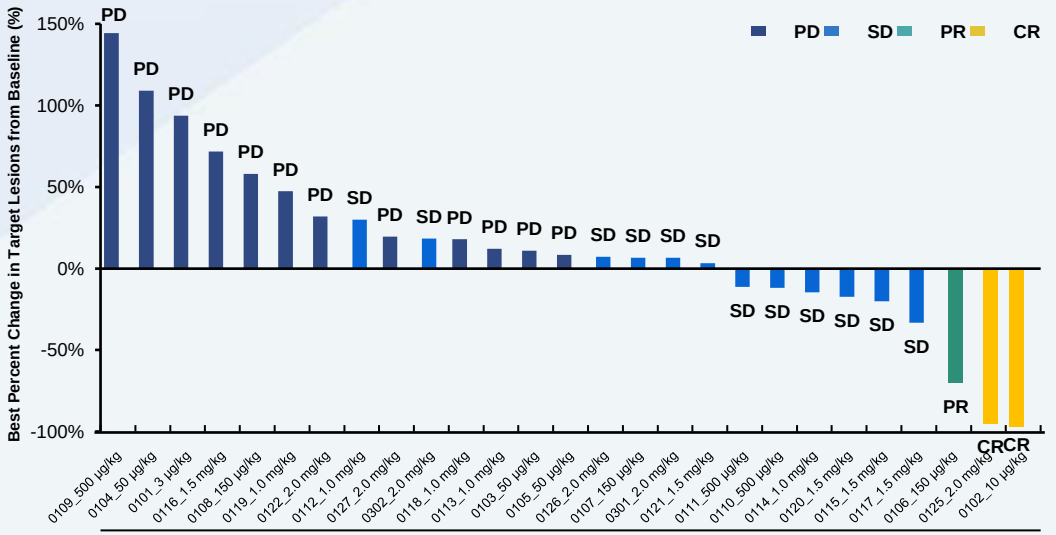
Our Differentiated Approaches (cont'd)

Phase I Clinical Trial Results of IMM01 Monotherapy

One of the only two companies to have observed **CR in monotherapy** clinical trials with a **well tolerated safety profile**

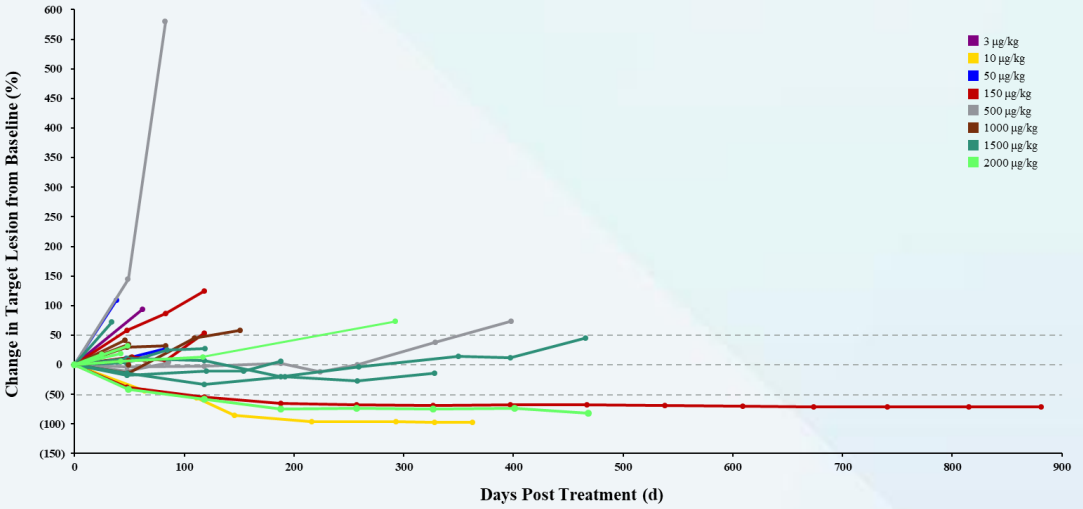
Potent Antitumor Activity and Encouraging Preliminary Clinical Efficacy

Response Observed in Patients Treated with IMM01 Monotherapy



Note: The colors of bars represent the best overall changes in size of target tumor lesions among 27 evaluable patients in the Phase I monotherapy study
Source: Company Data, as of December 14, 2022

Duration of Response in Patients Treated with IMM01 Monotherapy



Among 27 evaluable patients receiving **0.003 mg/kg** to **2.0 mg/kg** dosage, two patient reached complete response (**2 CRs**), one reached partial response (**1 PR**), and 13 reached stable disease (**13 SDs**) (including **six cases** with **observed substantial tumor shrinkage**)

Source: Company Data

Our Differentiated Approaches (cont'd)

Clinical Trial Results of IMM01 Monotherapy

Safety Results

Majority of TRAE is **grade 1 and 2**

Grade 3 and above TRAE mainly include **Leukopenia, Thrombocytopenia, Anemia, Neutropenia**, with the highest rate of occurrence as **14% (4/29)**

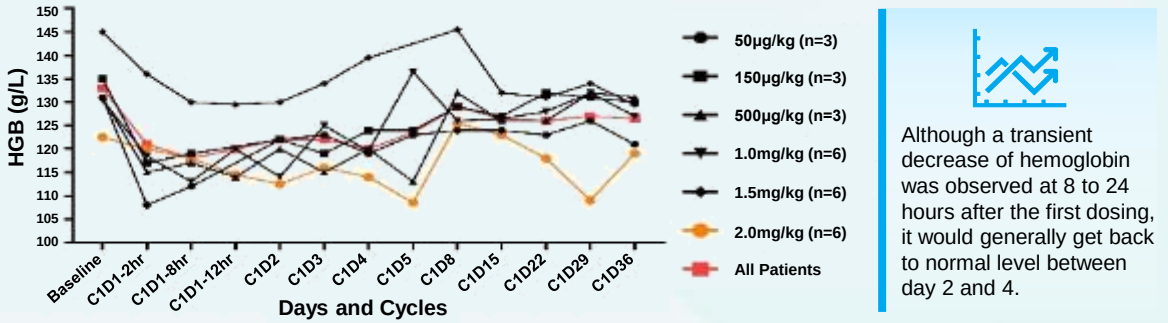
Treatment-related adverse event (n=29)	ALL n (%)	≥Gr 3 n (%)
Positive of Anti erythrocyte antibody	17 (59)	
Leukopenia	16 (55)	2 (7)
Hemolysis	15 (52)	
Infusion related reaction	15 (52)	
Thrombocytopenia	13 (45)	3 (10)
Hypertriglyceridemia	13 (45)	
Anemia	13 (45)	4 (14)
Neutropenia	12 (41)	1 (3)
Neutrocytosis	12 (41)	
Alkaline phosphatase increased	8 (28)	
Leukocytosis	8 (28)	
Hyperbilirubinemia	7 (24)	
Hypercholesteremia	6 (21)	
Fever	5 (17)	
Proteinuria	5 (17)	
ALT increased	4 (14)	
GGT increased	3 (10)	
Hyperuricemia	3 (10)	
Hypothyroidism	3 (10)	
AST increased	4 (14)	

Notes:
1. TRAE above 10% is presented
2. IMM01 is generally safe and well tolerated in 29 patients
3. Majority of TRAEs were grade 1 and 2
4. Grade 3 and above TRAEs mainly include Leukopenia, Thrombocytopenia, Anemia, Neutropenia, with the highest rate of occurrence as 14% (4/29)

Source: Company Data

The impact on hemoglobin or platelet is **transient and insignificant** following the administration of IMM01.

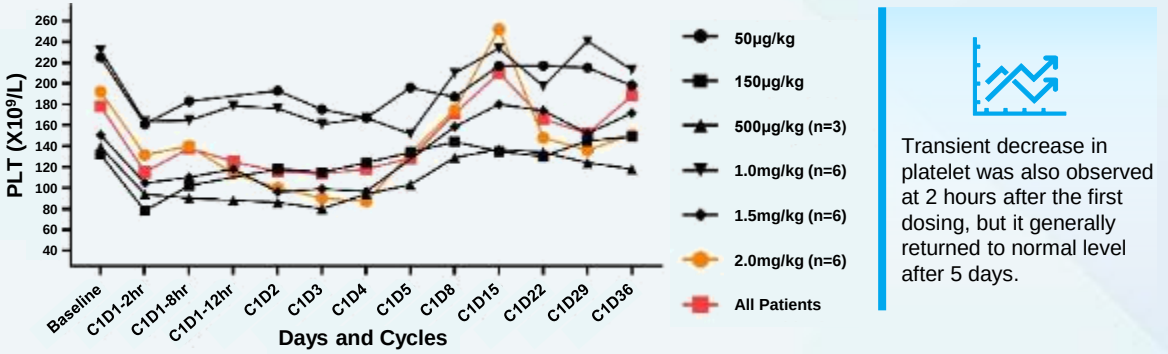
HGB Changes Following Single-dose and Cycle 1 by Cohort



Although a transient decrease of hemoglobin was observed at 8 to 24 hours after the first dosing, it would generally get back to normal level between day 2 and 4.

Note: Dosing days are C1D1, C1D8, C1D15, C1D22, C1D29, C1D36.

PLT Following Single-dose and Cycle 1 by Cohort



Transient decrease in platelet was also observed at 2 hours after the first dosing, but it generally returned to normal level after 5 days.

Note: Dosing days are C1D1, C1D8, C1D15, C1D22, C1D29, C1D36.

IMM01 - Competitive Landscape

One of the only two companies to have observed **CR in monotherapy** clinical trials with a **well tolerated safety profile**

Drug Name	Company	Molecule	Fc isotype	RBC binding	1st in human	Monotherapy CR	Latest Stage
IMM01	ImmuneOnco 宜明昂科	SIRPαFc	IgG1	No	2019.9	Yes	Ph II
Hu5F9 (Magrolimab)	Forty-Seven (Gilead)	mAb	IgG4	Yes	2014.8	No	Ph III (Partial Suspension by the Company)
TTI-621	Trillium Therapeutics (Pfizer)	SIRPαFc	IgG1	No	2016.1	Yes	Ph II (Partial Suspension by the Company)
TTI-622		SIRPαFc	IgG4	No	2018.5	Yes	Ph II
CC-90002	Celgene (BMS)	mAb	IgG4	Yes	2015.2	No	Ph I (Partial Suspension by the Company)
SRF231	Surface Oncology	mAb	IgG4	Yes	2018.4	No	Ph I (Suspension by the Company)
ALX-148 (Evorpacept)	ALX Oncology	SIRPαFc	IgG1 Fc(Inert)	Yes	2017.1	No	Ph II/III
SHR1603	HengRui 恒瑞	mAb	IgG4	Yes	2018.10	No	Ph I (Suspension by the Company)
AO-176	Arch Oncology	mAb	IgG2	Minimal	2019.2	No	Ph I/II (Suspension by the Company)
IBI188 (Letaplimab)	Innovent 信达生物	mAb	IgG4	Yes	2018.11	No	Ph Ib/III (Partial Suspension by the Company)
TJC4 (Lemzoparlimab)	I-Mab 天境生物/ AbbVie	mAb	IgG4	Minimal	2019.5	No	Ph III (Partial Suspension by the Company)
AK117	Akesobio 康方生物	mAb	IgG4	Minimal	2020.4	No	Ph II

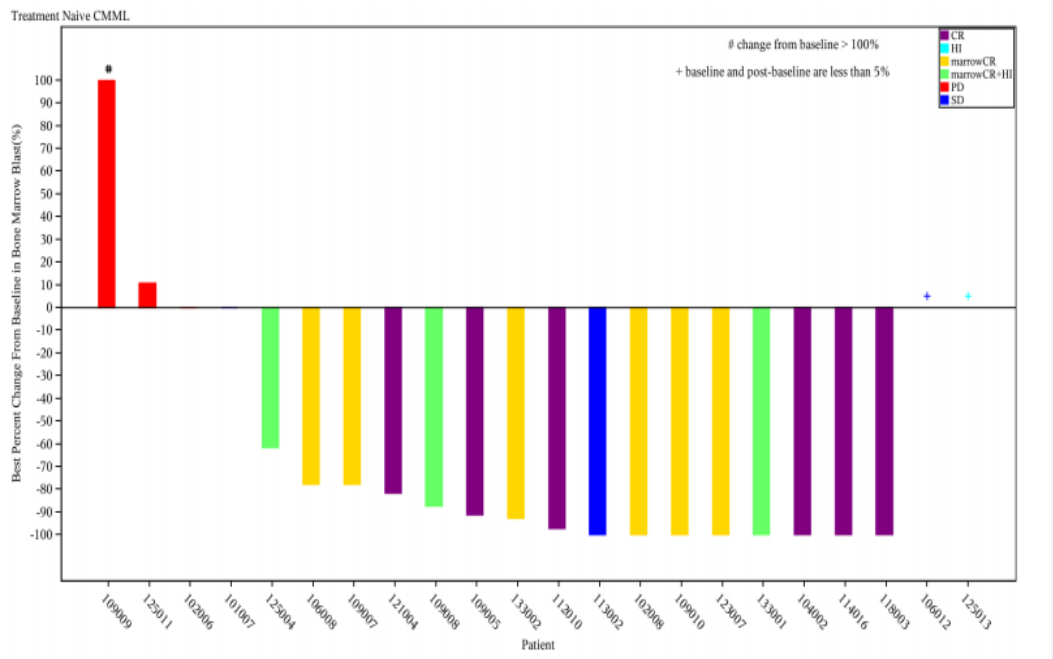
Source: Frost & Sullivan, Official Websites of Relevant Companies

Notes:
1. Clinical data are extracted from official websites of relevant companies, reported clinical trials and published literature
2. Despite a comparison is made here, the key results are not from head-to-head studies
3. "1st in human" refers to the first posted date of the first clinical trial
4. The stage listed here is the latest clinical trial of the drug
5. Partial suspension means not all clinical trials of this drug are suspended, such as monotherapy of CC-90002 which has been suspended but its combination therapy with rituximab has completed
6. For the drugs associated with two companies, the company in the parenthesis is the acquirer
7. The FDA has lifted all of the partial clinical hold placed on several trials evaluating magrolimab, as it determined that, following a comprehensive review of the safety data from each trial, the clinical sponsor had satisfactorily addressed the deficiencies
8. As to the monotherapy CR column, "No" means that no CR was achieved in a completed or suspended clinical trial
9. The clinical trials of drug candidates marked as dark-gray have been suspended

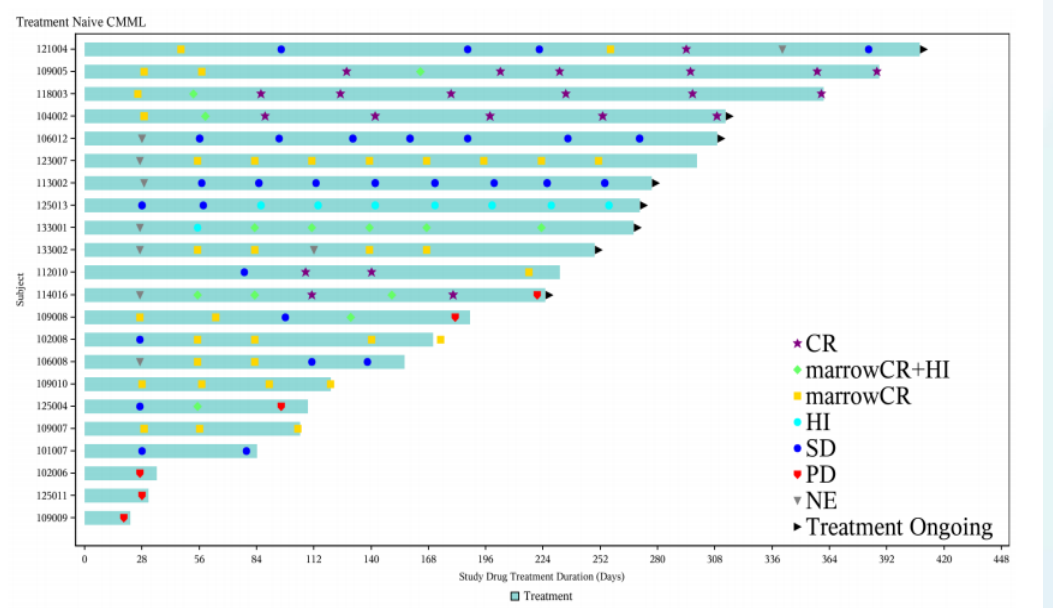
IMM01 + Azacitidine in 1L CMML

Phase II

Best Percent Change from Baseline in the Blast Cells in the Bone Marrow



Duration of Treatment and Response



Best Overall Response, n (%)	1L CMML (N=22)	≥4 months (N=16)	≥6 months (N=13)
ORR	**16 (72.7%)**	**14 (87.5%)**	**11 (84.6%)**
CR	**6 (27.3%)**	**6 (37.5%)**	**6 (46.2%)**
mCR + HI	3 (13.6%)	2 (12.5%)	2 (15.4%)
mCR alone	6 (27.3%)	5 (31.3%)	2 (15.4%)
HI	1 (4.5%)	1 (6.3%)	1 (7.7%)

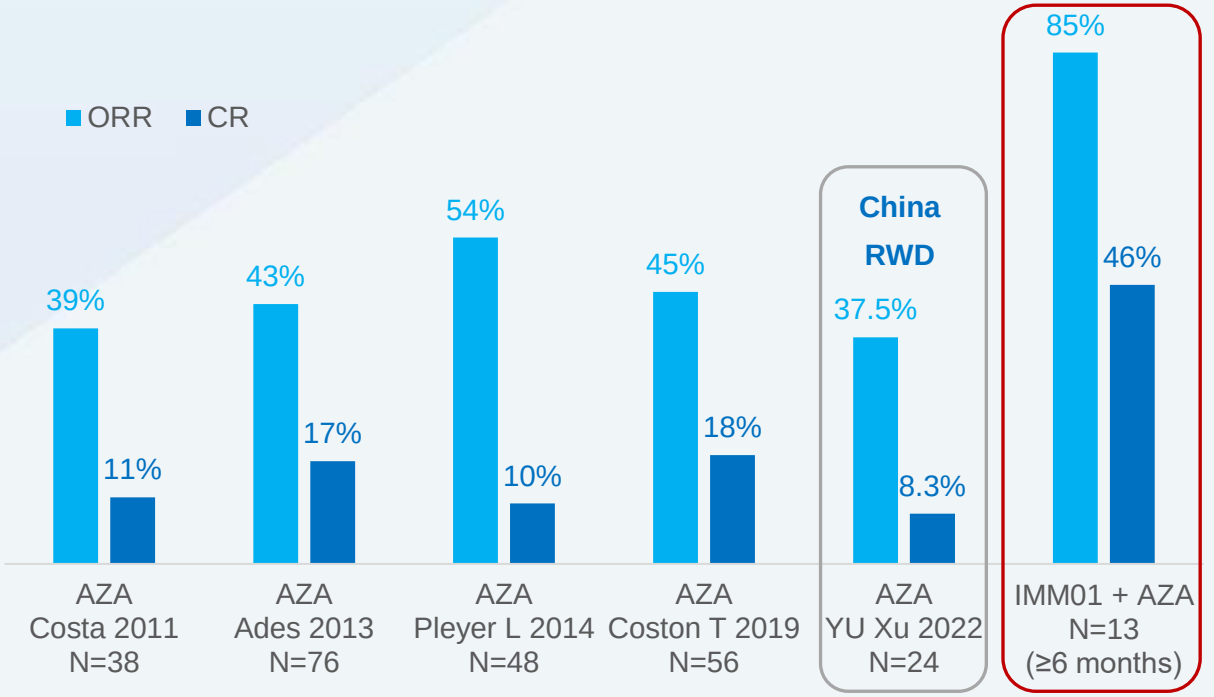
Notes: ORR = Overall Response Rate, CR = Complete Response, mCR = Marrow Complete Response, HI = Hematological Improvement

Source: Company Data; The clinical data is as of December 31st, 2023

IMM01 + Azacitidine in 1L CMML (cont'd)

Comparison in Treating 1L CMML

Response of Major Clinical Studies in CMML



- As indicated by the graph, the ORR and CR rates range from 37% to 54% and 8% to 18% respectively in major clinical trials of azacitidine in CMML based on historical data.
- Particularly, real-world data on efficacy and safety of azacitidine therapy in 24 patients with CMML from a multicenter, retrospective study in **China** published in July 2022 **showed an ORR of 37.5% with a CR rate and a mCR/HI rate of 8.3% and 20.8%, respectively**. In contrast, in our Phase II trial for the combination of IMM01 and azacitidine, among the 13 evaluable patients (≥6 months) with 1L CMML, six reached complete response (**6 CRs**), four reached marrow complete response with two hematological improvement (**2 mCRs + HI and 2 mCRs alone**), and one reached hematological improvement alone (**1 HI alone**), **resulting in an ORR of 84.6% and a CR rate of 46.2%**.

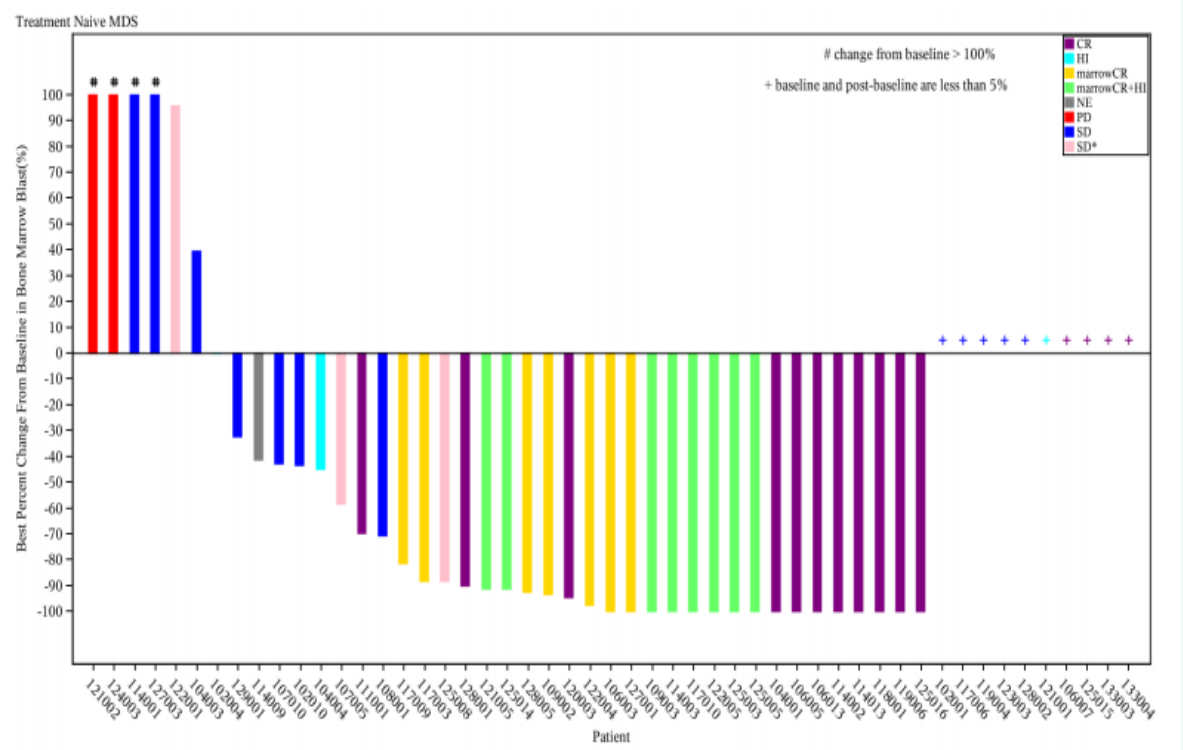
Notes:
1. ORR refers to overall response rate; CR refers to complete response.
2. There were no head-to-head comparison clinical trials conducted between these drugs. The results of clinical trials of a drug cannot be directly compared to that of another drug and may not be representative of the overall data.

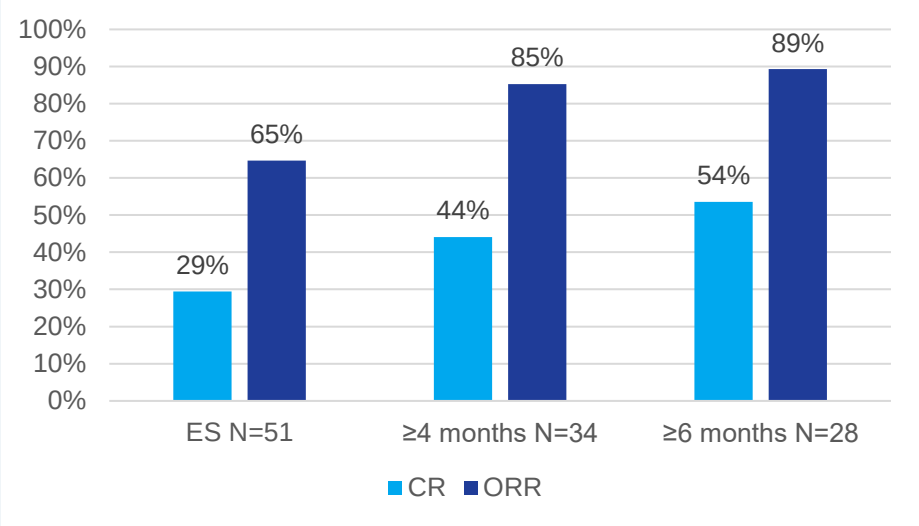
Source: Literature Review; Company Data, the clinical data is as of December 31st, 2023

IMM01 + Azacitidine in 1L MDS

Phase II

Best Percentage Change from Baseline in the Blast Cells in the Bone Marrow (%)





Best Overall Response n (%)	1L MDS (N=51)	≥4 months (N=34)	≥6 months (N=28)
ORR	**33 (64.7%)**	**29 (85.3%)**	**25 (89.3%)**
DCR	**45 (88.2%)**	**34 (100%)**	**28 (100%)**
CR	**15 (29.4%)**	**15 (44.1%)**	**15 (53.6%)**
mCR+HI	8 (15.7%)	7 (20.6%)	5 (17.9%)
mCR alone	7 (13.7%)	4 (11.8%)	3 (10.7%)
HI	3 (5.9%)	3 (8.8%)	2 (7.1%)
SD	12 (23.5%)	5 (14.7%)	3 (10.7%)

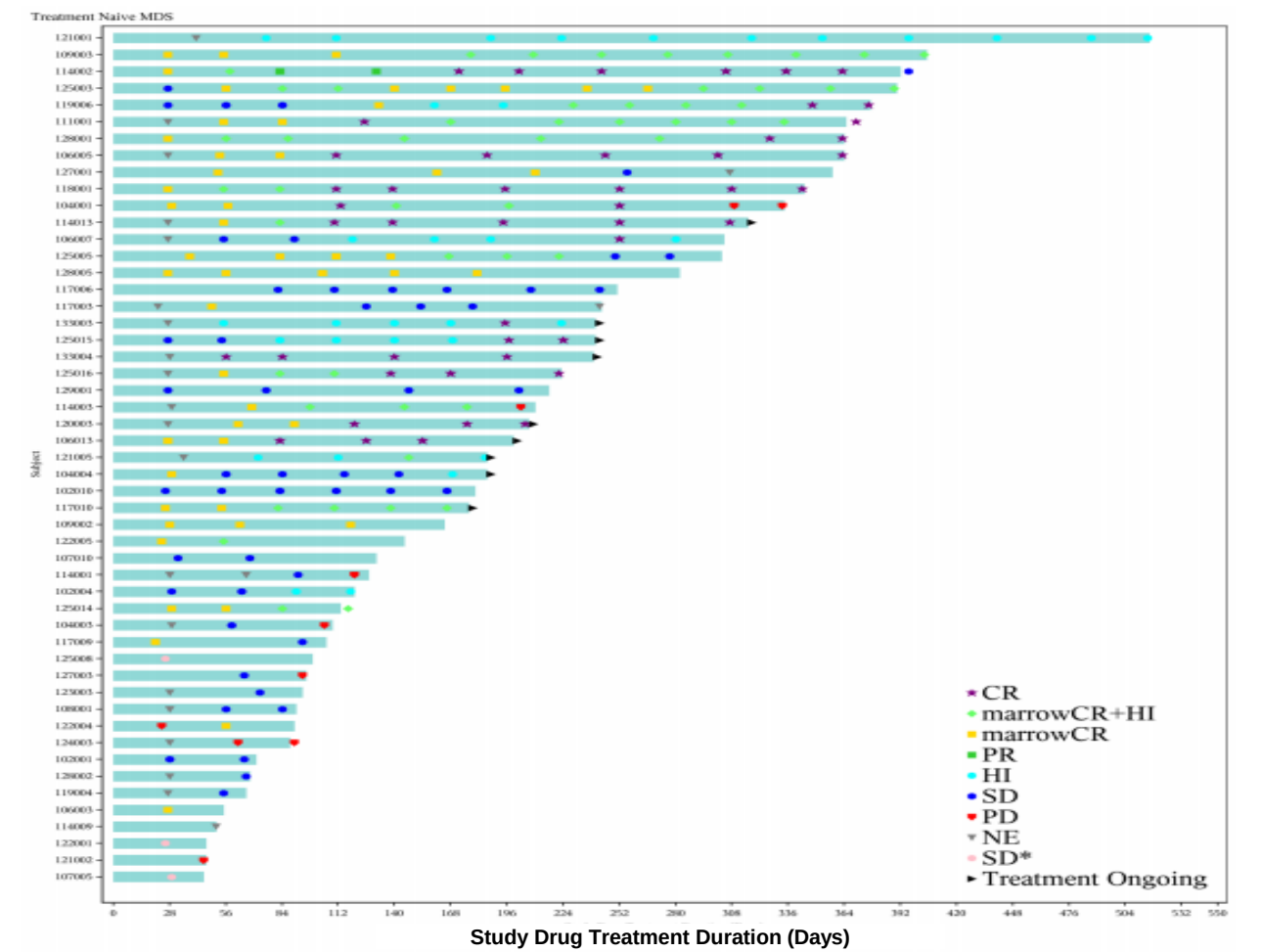
Notes: ORR = Overall Response Rate, CR = Complete Response, mCR = Marrow Complete Response, HI = Hematological Improvement, SD = Stable Disease

Source: Company Data; The clinical data is as of December 31st, 2023

IMM01 + Azacitidine in 1L MDS (cont'd)

Phase II

Duration of Treatment and Response



Notes: ORR = Overall Response Rate, CR = Complete Response, mCR = Marrow Complete Response, HI = Hematological Improvement, SD = Stable Disease

Source: Company Data; The clinical data is as of December 31st, 2023

IMM01 + Azacitidine

Comparison: Safety results

Magrolimab + AZA vs AZA alone

	MDS Ib Magrolimab + AZA (N=95)		AZA-001 MRCT AZA alone (N=175)	
TRAE	**All grades, N(%)**	**≥Grade 3, N(%)**	**All grades, N(%)**	**≥Grade 3, N(%)**
Anemia	49 (51.6%)	**45 (47.4%)**	90 (51.4%)	**24 (13.7%)**
Leukopenia	**28 (29.5%)**	**28 (29.5%)**	**32 (18.2%)**	**26 (14.9%)**
Neutropenia	45 (47.4%)	44 (46.3%)	115 (65.7%)	107 (61.1%)
Febrile neutropenia	**29 (30.5%)**	**27 (28.4%)**	**24 (13.7%)**	**22 (12.6%)**
Thrombocytopenia	52 (54.7%)	44 (46.3%)	122 (69.7%)	102 (58.3%)

Compared to the trial of AZA alone, **significant higher rates of occurrence of some TRAEs** (such as anemia, leukopenia and febrile neutropenia) were observed in the clinical trial for the combination of Magrolimab and AZA.

IMM01 + AZA vs AZA alone

	IMM01-02 Study – MDS Cohort IMM01 + AZA (N=57)		China MDS-002 Single-arm Study AZA alone (N=72)	
TRAE	**All grades, N(%)**	**≥Grade 3, N(%)**	**All grades, N(%)**	**≥Grade 3, N(%)**
Anemia	27 (47.4%)	25 (43.9%)	38 (52.8%)	35 (48.6%)
Neutropenia	38 (66.7%)	38 (66.7%)	48 (66.7%)	48 (66.7%)
Thrombocytopenia	41 (71.9%)	38 (66.7%)	52 (72.2%)	50 (69.4%)

Compared to the trial of AZA alone, **similar rates of occurrence of TRAEs** were observed in our phase II trial for the combination of IMM01 and AZA.

Source: Valeria Santini1 et al, Eur J Haematol. 2010 August ; 85(2): 130–138.
David et al. A.J Clin Oncol ,2023,41:2815-2826;
Company Data; The clinical data is as of December 31st, 2023

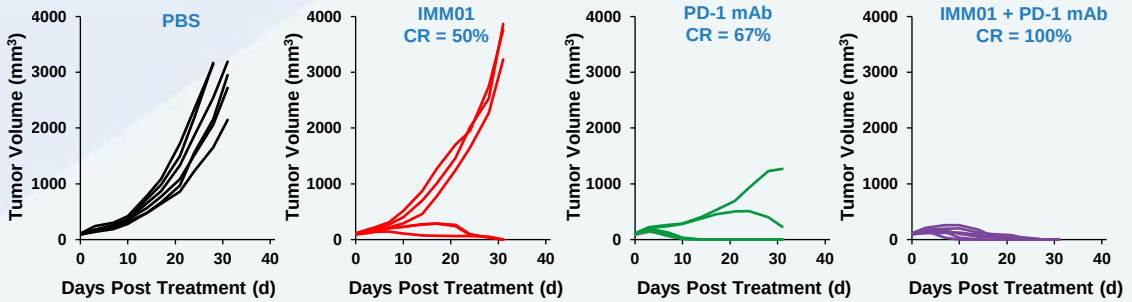
IMM01 + Tislelizumab (PD-1 mAb)

Preclinical Results and Clinical Development Plan

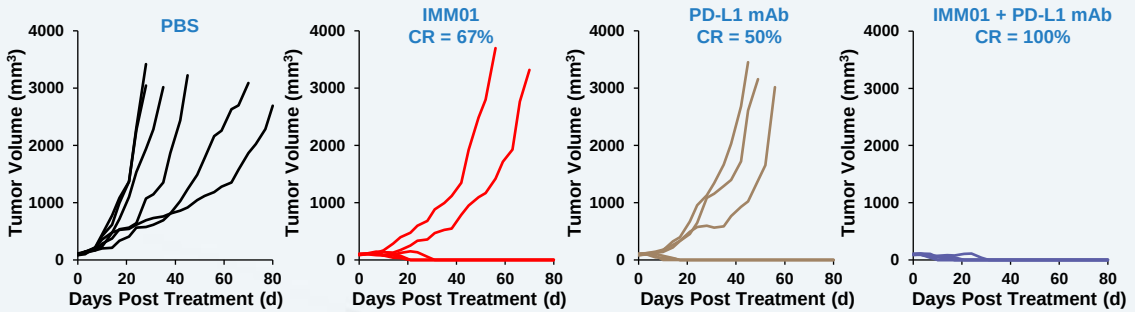
Combination with a PD-1/PD-L1 Antibody

The combination of IMM01 with either a PD-1 or PD-L1 antibody exhibited **encouraging synergistic effects** in our *in vivo* solid tumor efficacy models

Efficacy Study of IMM01 and a PD-1 Antibody in Colon Cancer (CT26) Syngeneic Mouse Model



Efficacy Study of IMM01 and a PD-L1 Antibody in Colon Cancer (CT26) Syngeneic Mouse Model



Notes: 1. Six mice per group were used in this study; 2. The colors of lines represent different groups using different drugs and/or drug candidates
Source: Company Data

Clinical Development Plan

Program	Indications	Clinical trial stage (status)	Trial site	First-patient-in date
IMM01 + tislelizumab	NSCLC, SCLC, HNSCC, other solid tumors, cHL[(1)]	Phase Ib (completed) Phase II (ongoing)	China	May 2022

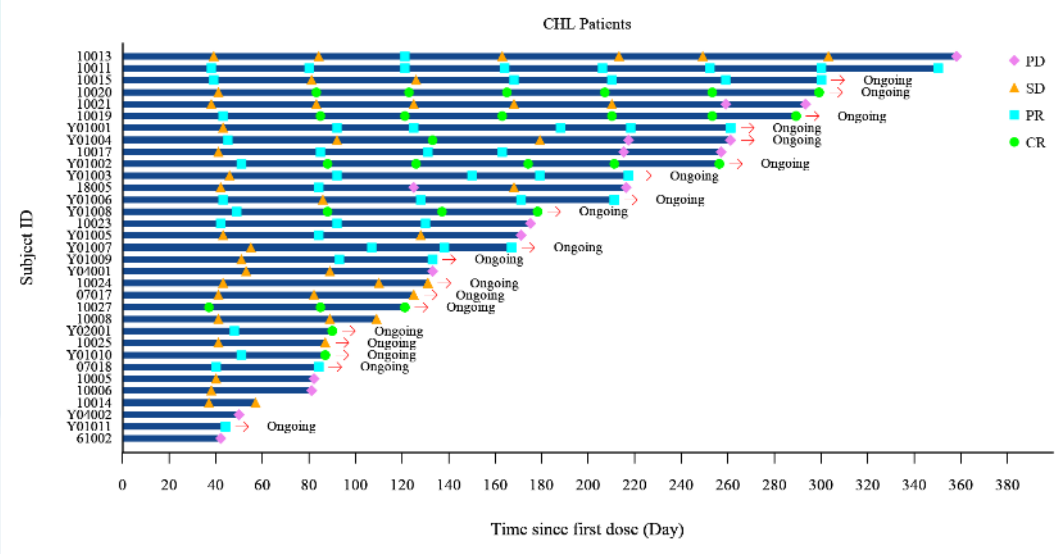
Notes:

We are evaluating this combination therapy in cHL patients who relapsed or progressed after the treatment of PD-1 inhibitors which may allow us to **pursue an accelerated marketing approval** leveraging the results of relatively small sample size studies

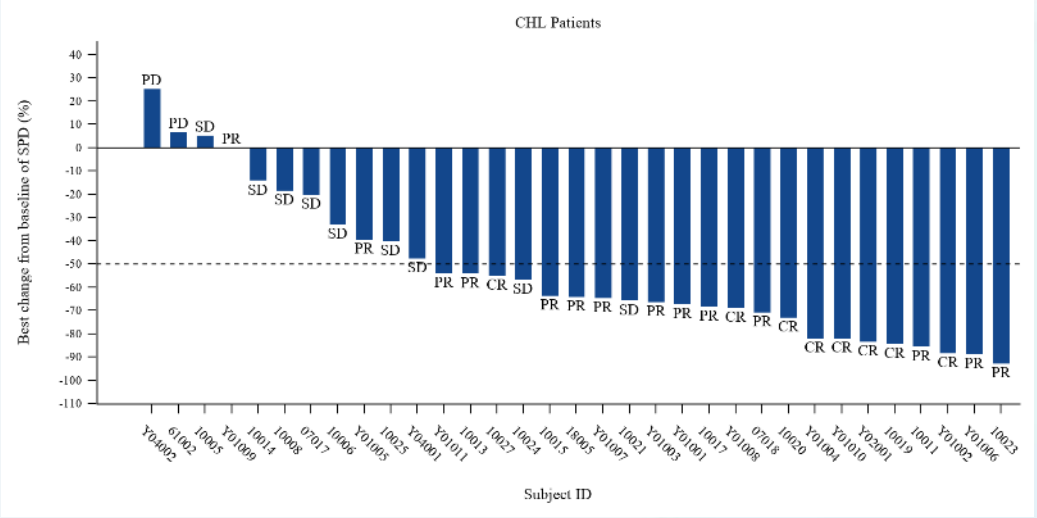
IMM01 + Tislelizumab (PD-1 mAb) (cont'd)

Phase II Efficacy in Prior Anti-PD-1 R/R cHL

Duration of Treatment and Response



Best Percentage Change from Baseline in Target Lesion



Remarkable anti-tumor efficacy

- Of 33 response-evaluable patients as of March 1, 2024, best overall responses were **8 CR, 14 PR and 9 SD, resulting in an ORR of 66.7% and DCR of 93.9%**.

Best Overall Response n (%)	R/R cHL (N=33)
ORR	**22 (66.7)**
DCR	**31 (93.9)**
CR	**8 (24.2)**
PR	**14 (42.4)**
SD	9 (27.3)
PD	2 (6.1)

Source: Company Data; The clinical data is as of March 1st , 2024

IMM01 + Tislelizumab (PD-1 mAb) (cont'd)

Phase II: Superior Efficacy in PD-1 Failed R/R cHL

	Timdarpacept (SIRPα-Fc) + Tislelizumab (PD-1)	Favezelimab (Anti-LAG-3) + Pembrolizumab [1]	Tifcemalimab (Anti-BTLA) + Toripalimab (PD-1) [2]
N	33	34	34
ORR	66.7%	29%	35.3%
CR	24.2%	9%	0%
Status	Phase III expected To initiated in Q1 2024	Phase III of the coformulated two drugs started in Oct 2022	Phase III started in Dec 2023 to treat R/R cHL
Study Geography	China	China + International	China

Source:

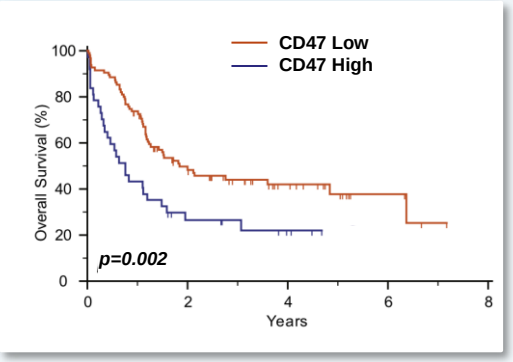
1. Timmerman et al. Blood (2022) 140 (Supplement 1): 768–770.
2. Song et al. Blood (2023) 142 (Supplement 1): 4458.Hodgkin Lymphoma. Histopathologically, 95% of HL cases are classified as cHL."- Momotow et al. J. Clin. Med. 2021, 10(5), 1125

Company Data; The clinical data is as of March 1st , 2024

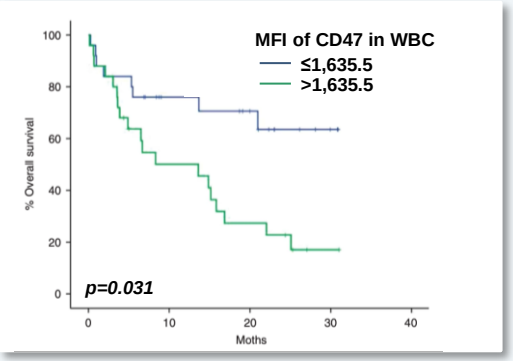
IMM01 - Market Opportunities

Increased CD47 Expression is Correlated with Poor Clinical Outcomes for Various Cancers

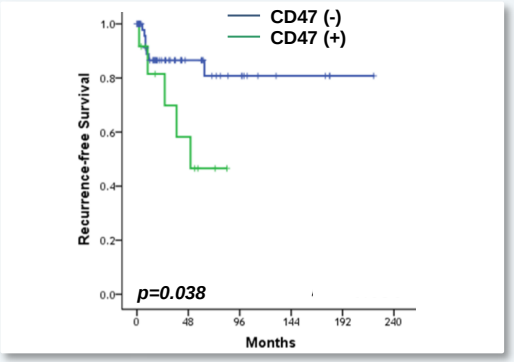
AML[1]



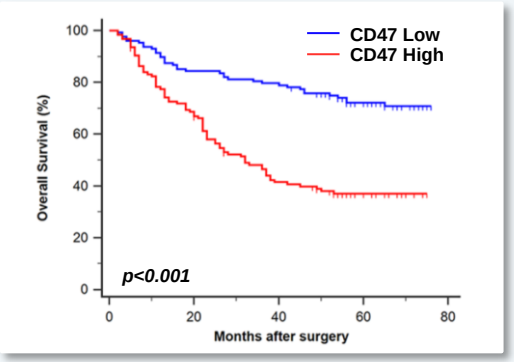
NSCLC[2]



HCC[3]



GC[4]



Note: Diagram HCC illustrates the recurrence survival of patients post surgical resection without any adjuvant therapy

Source:

1. Majeti et al. Cell 138, 286–299, July 24, 2009
2. Barrera et al. Br J Cancer 117, 385–397 (2017)
3. Kim H, et al. J Clin Pathol 2020;0:1–5
4. Shi et al. Cancer Immunol Immunother 70, 1831–1840 (2021)

Strong Potentials of CD47-based Therapies

- ✓ CD47 is overexpressed on the surface of numerous tumor cells, including **NSCLC, SCLC, BC, GC, CRC, HNSCC, HCC, ESCC, BTC, OC, lymphoma, AML, MDS, CMML, MM** and **highly correlated** with **poor prognosis**
- ✓ Therapeutic potential of CD47-targeted agents have been **validated** by accumulating clinical data in treating **both hematologic and solid tumors**, such as **non-Hodgkin lymphoma (NHL), AML, MDS, SCLC, HNSCC, OC and GC**
- ✓ **Unique MoA** of IMM01 could **present strong synergy with PD-1/PD-L1 inhibitors** and **enhance the response rates** of solid tumors to PD-1/PD-L1 treatments

Developing In-house and Own IP and Commercial Rights

1 issued patent in China， ***1*** issued patent in Japan， ***1*** issued patent in the U.S.

1 issued European patent (validated in the ES, CH, DE, FR, GB, IT)

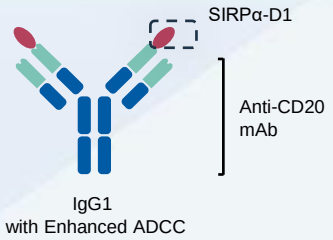
Other patent applications are pending

IMM0306 (CD47×CD20)

1st CD47 and CD20 Dual-targeting Bispecific to Enter into the Clinical Stage Globally

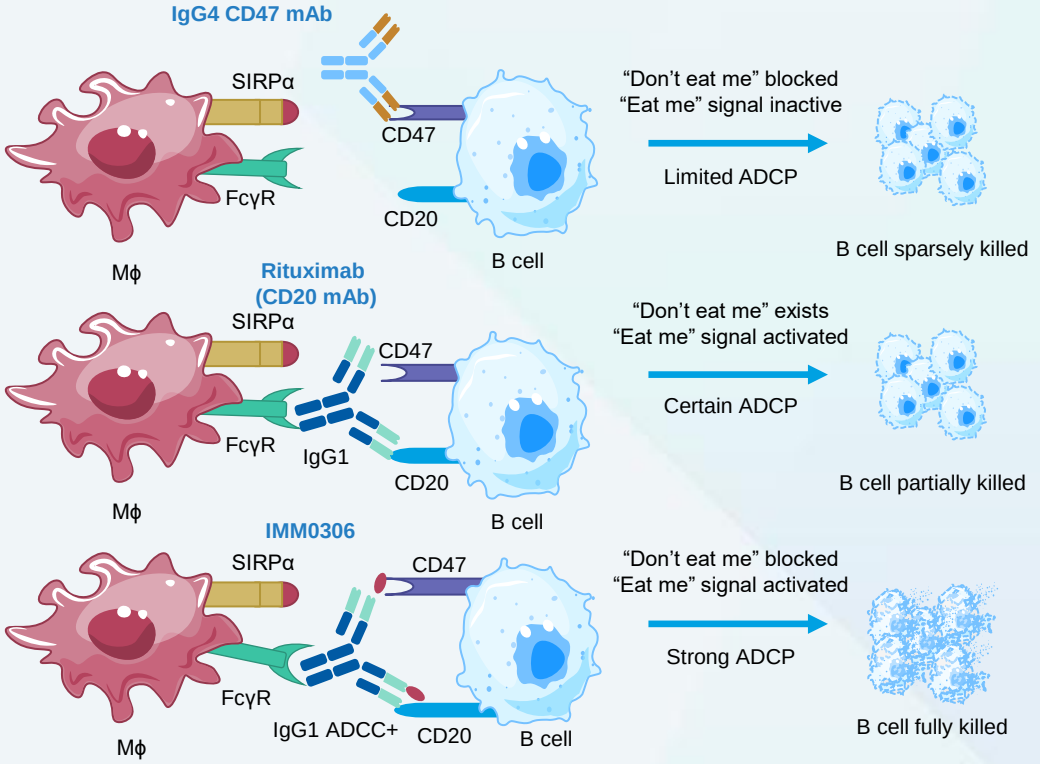
Overview

IMM0306 Molecule Structure



- Full macrophage activation
- Improved ADCP and ADCC activity
- Improved effectiveness for treating patients predominantly expressing FcγRIIIA-158F polymorphism that is less sensitive to CD20 antibody treatment

Mechanism of Action



Market Opportunities and Competition

Unmet needs of R/R B-NHL treatment:

- ✓ CD20 antibody combined with chemotherapy are recommended for 1L & later line treatment
- ✓ However, **approximately 50% of B-NHL patients will eventually relapse**

2 CD47×CD20 bispecific antibodies/fusion proteins under development globally
Among them, IMM0306 is the **1st** to enter into a clinical trial

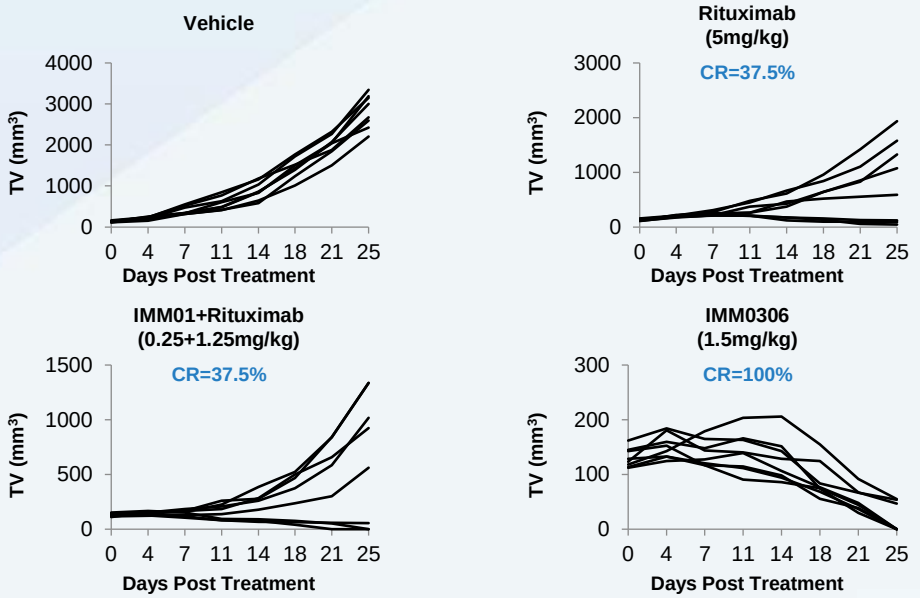
Have great potential in addressing the **unmet needs of R/R B-NHL treatment**

IMM0306 (CD47×CD20) (cont'd)

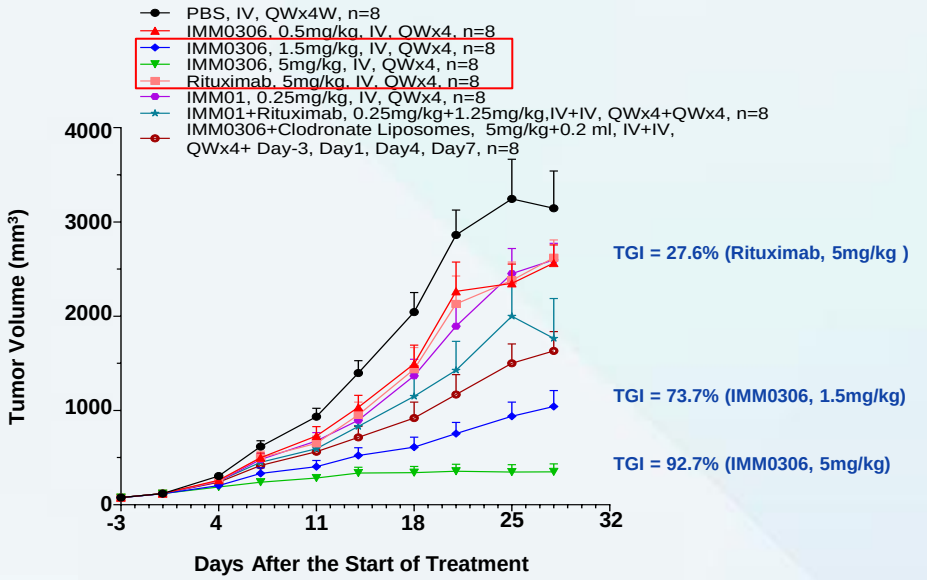
Preclinical Results

In Vivo Efficacy Study

Efficacy Study in Lymphoma (Daudi) Xenograft Mouse Model



Efficacy Study in Lymphoma (Raji) Xenograft Mouse Model



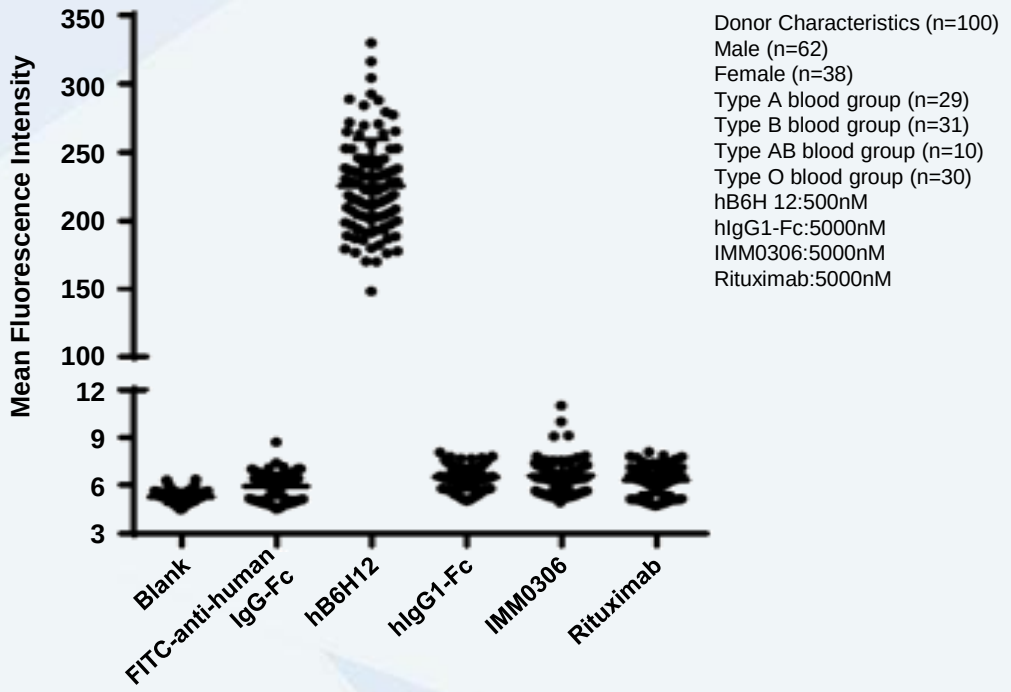
- IMM0306 was more potent than **rituximab (CD20 mAb) monotherapy**, even at a much lower dosing level, and it is more potent than the **combination therapy of IMM01 and rituximab** at a comparable dosing level

IMM0306 (CD47×CD20) (cont'd)

Preclinical Results

Favorable Safety Profile with No Human Red Blood Cell Binding *In Vitro*, with Only Minor Cytokine Storm

Human RBC Binding Analysis of IMM0306



Donor Characteristics (n=100)
Male (n=62)
Female (n=38)
Type A blood group (n=29)
Type B blood group (n=31)
Type AB blood group (n=10)
Type O blood group (n=30)
hB6H 12:500nM
hIgG1-Fc:5000nM
IMM0306:5000nM
Rituximab:5000nM

Does not bind to RBCs in *in vitro* preclinical studies or **cause hemagglutination or hemolysis** in clinical trials

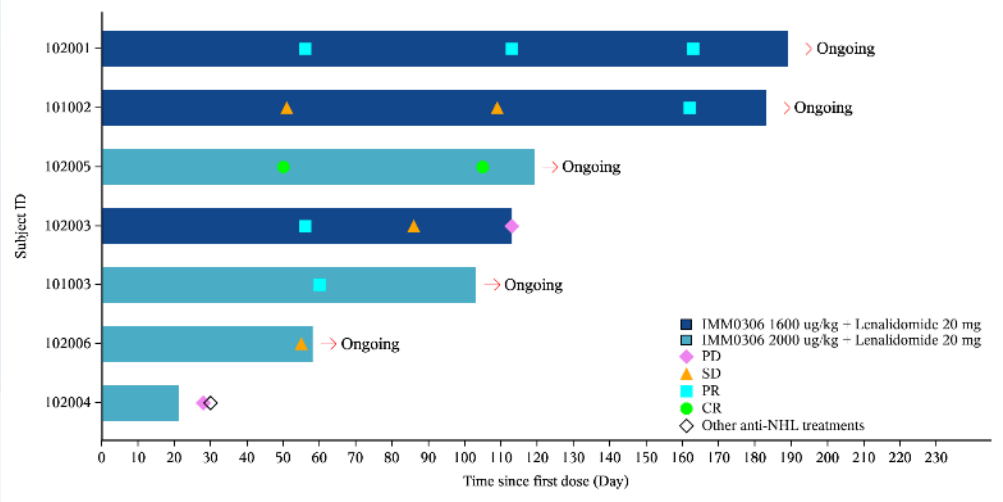
With the higher affinity for CD20 to **minimize "on-target, off-tumor" toxicity**

Only triggers **minor CRS**

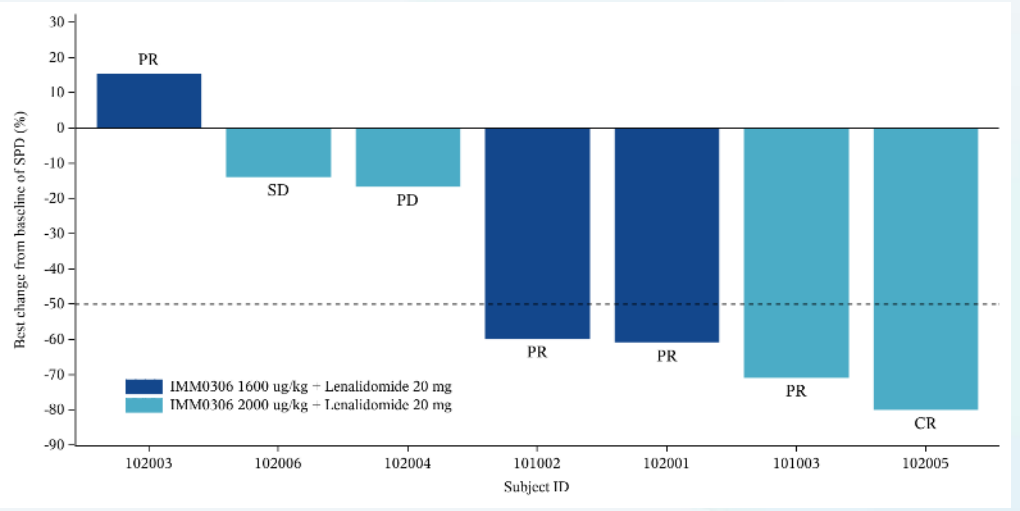
IMM0306 + Lenalidomide (CD47×CD20) (cont'd)

1st CD47 and CD20 Dual-targeting Bispecific to Enter the Clinical Stage Globally

Duration of Treatment and Response



Best Percentage Change from Baseline in Target Lesion



Developing In-house and Own its IP and Commercial Rights

5 issued patents in China, Japan, Europe (validated in the ES, CH, DE, FR, GB, IT) and the U.S.

Other patent applications are pending

Best Overall Response n (%)	Efficacy Evaluable (N=7)
CR	1 (14.3)
PR	4 (57.1)
SD	1 (14.3)
PD	1 (14.3)
ORR	5 (71.4)
DCR	6 (85.7)

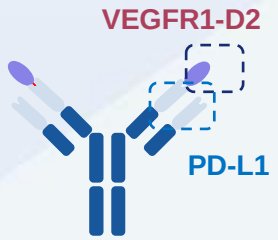
Of 7 response-evaluable patients as of Jan 5, 2024, **The ORR and DCR were 71.4% and 85.7%, respectively.**

IMM2510 (VEGF × PD-L1)

A bsAb with the mAb-Trap Structure Targeting VEGF and PD-L1

IMM2510 - Target Introduction and Molecule Structure

IMM2510 Molecule Structure



IgG1 Fc with Enhanced ADCC

PD-L1

Tumor cells expressing PD-L1 can **bind to PD-1** on the surface of T cells to evade T-cell attacks

VEGF

A dynamic angiogenic factor that is up-regulated in many tumor indications, which contributes to **angiogenesis** and tumor growth

Synergistic effect validated by approved anti-PD-(L)1 and anti-VEGF combo

	PD-L1	VEGF	PD-(L)1 Combo[1]
Molecule	TECENTRIQ, BAVENCIO, IMFINZI	AVASTIN	
IL Treatment	UC, SCLC, NSCLC	CRC, NSCLC	**RCC, HCC, NSCLC**
Others	NSCLC, HNSCC, Melanoma HCC, RCC, UC, HL	GBM, CRC, NSCLC, RCC, OC, CC	**EAC, CC**

IMM2510 - Mechanism of Action

By connecting VEGF and PD-L1, IMM2510 can

Block the PD-(L)1 pathway and thus **activate T cells**, which has demonstrated robust antitumor activities in a broad range of solid tumors

Reduce VEGF-mediated tumor **angiogenesis** and inhibit immune **suppression**, thus promoting the activation of T-cell immune responses

Exert **synergistic antitumor activities** than the combination of a VEGF blocker and a PD-L1 antibody

Equipped with an ADCC-enhanced IgG1 Fc fragment, IMM2510 can

Further activate NK cells and macrophages through strengthened Fc-mediated ADCC/ADCP activities to promote innate and subsequent adaptive immune responses

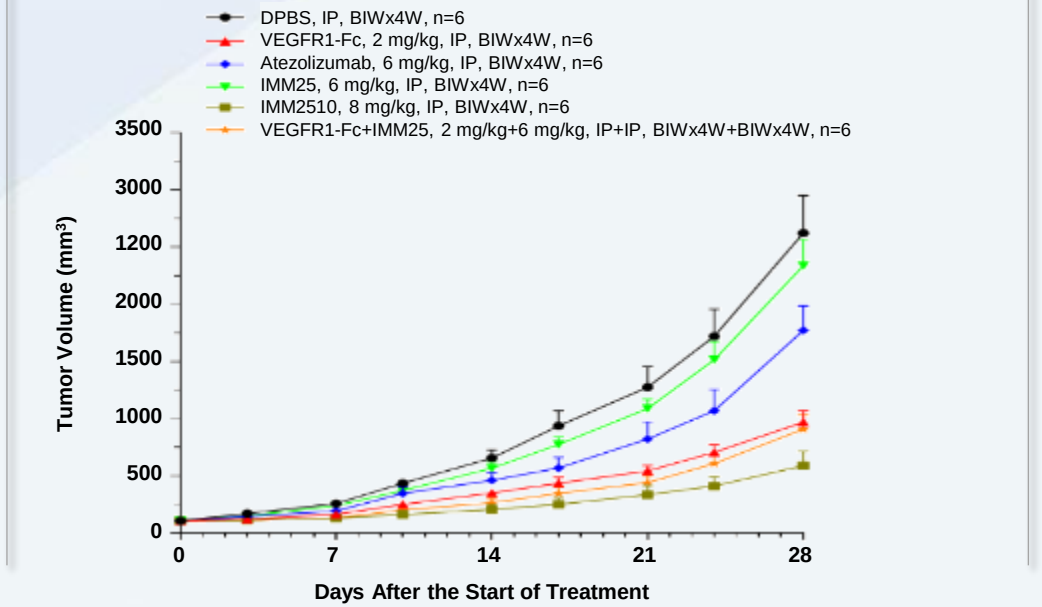
Notes:
1. Approved anti-PD-1/PD-L1 and anti-VEGF combination therapies

IMM2510 (VEGF × PD-L1) (cont'd)

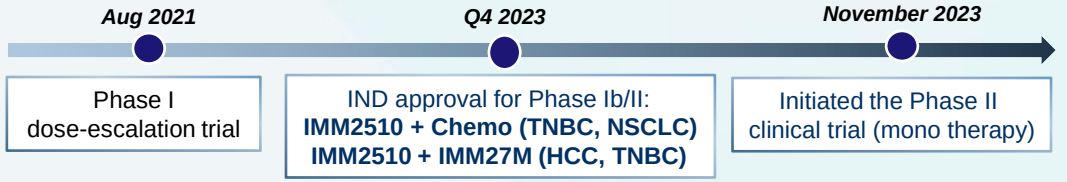
Competitive Advantages

Potent In Vivo Efficacy Compared to the VEGF Or PD-L1 Antibodies used as a Single Agent or in Combination

Efficacy Study in Breast Cancer (MDA-MB-231-Luc) Xenograft Mouse Model



Clinical Development Plan



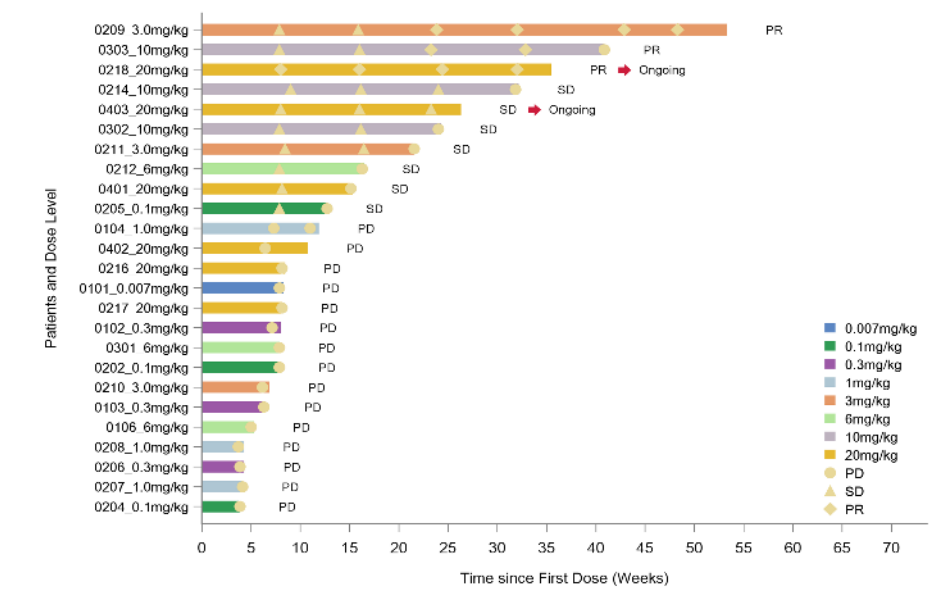
Developing One Owned Patent Family

- 1 issued patent in each of the U.S. and Japan
- 1 issued patent in the PRC
- 1 pending patent application in each of Europe and the U.S.

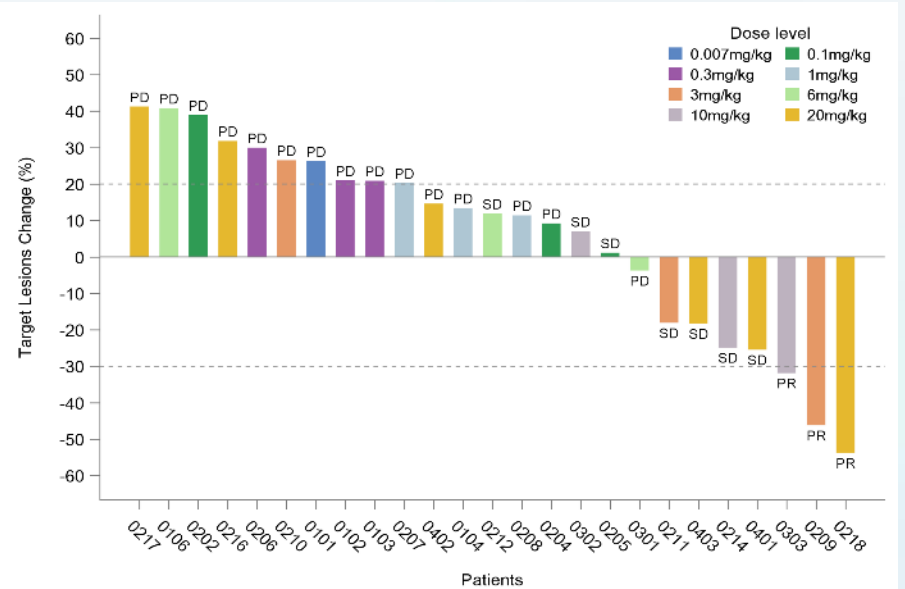
IMM2510 (VEGF × PD-L1) (cont'd)

Phase I: Efficacy Summary in advanced solid tumors

Duration of Treatment and Response



Best Percentage Change from Baseline in Target Lesion



Of 25 response-evaluable patients as of Dec 31, 2023, **3 patients had confirmed PR, and 7 patients achieved SD, with 4 of them observed tumor shrinkage of over 15%**

Source: Company Data; The clinical data is as of December 31st, 2023

IMM27M (CTLA-4 ADCC+)

A CTLA-4 mAb with Enhanced ADCC Activity

IMM27M - Mechanism of Action and Limitations of Approved Molecule

IMM27M Molecule Structure

Engineered IgG1 CTLA-4 mAb with Enhanced ADCC

- **Blocking the interaction** between CTLA-4 and CD80/CD86, and thus enhancing immune responses of T cells to tumor antigens
- Inducing enhanced immune responses targeting CTLA-4 **overexpressed T_{reg} cells**
- Promoting **T_{reg} depletion**, thus improving T-cell antitumor response to kill tumor cells

Currently Approved CTLA-4 Antibody with Unmodified Fc:

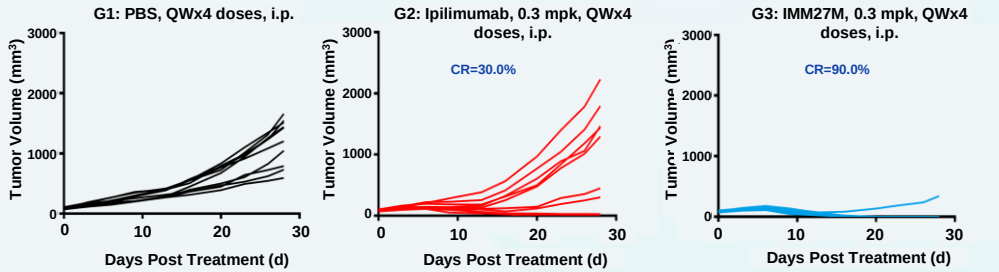
Limited efficacy → High dosage to achieve desirable efficacy → **Serious safety issues**

Clinical Development Plan

Jun 2022	*Mar 2023*	*Sept 2023*
Phase I mono	IMM27M+PD-1 (advanced solid tumors) IND approval for Ph Ib/II	Phase I mono completed and confirmed RP2D

Stronger In Vivo Antitumor Effects and Preliminary Ph1 data

Efficacy Study in Colon Cancer (MC38) Syngeneic Mouse Model



Significantly **stronger antitumor activity** than ipilimumab, **complete tumor remission** even at a dose as low as **0.3 mg/kg** (~0.03 mg/kg human equivalent dose)

Phase I Preliminary Efficacy

- **2 patients had confirmed PR:**
  1 patient with mBC (HR+/HER2+, IO naïve, 6L previous treatments) at 3.0 mg/kg with **best tumor shrinkage 62.5%,** duration of response about 9 months;
  1 patient with mBC (HR+/HER2-, IO naïve, 4L previous treatments) at 5.0 mg/kg with **best tumor shrinkage 41.0%,** duration of response over 4 months;
- **3 patients had SDs with tumor shrinkage:**
  1 patient with metastatic melanoma has achieved tumor shrinkage of 22.9% at 2 mg/kg;
  2 patients with HR positive BCs have achieved tumor shrinkage of 18.5% at 7.5 mg/kg and 10.3% at 5 mg/kg, respectively.

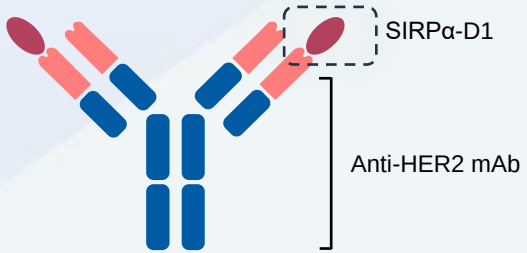
Source: Company Data; The clinical data is as of December 31st, 2023

IMM2902 (CD47×HER2)

The only CD47×HER2 bispecific molecule that has entered into clinical trial globally

Overview

IMM2902

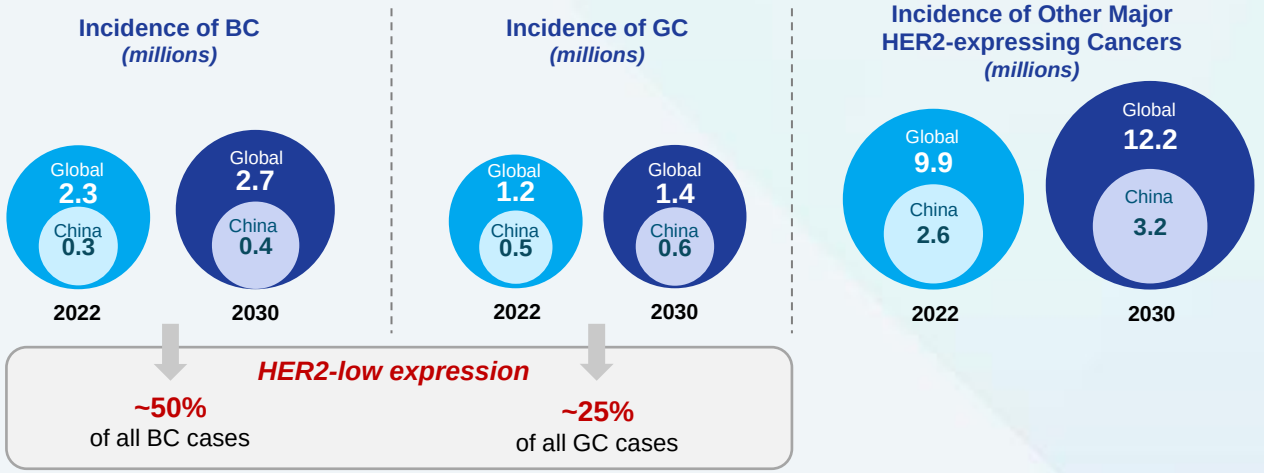


IgG1
with enhanced ADCC

- No RBC binding *in vitro*
- Enhanced ADCP and ADCC activity
- Accelerated HER2 internalization and degradation

Market Opportunities and Competition

HER2 overexpression is prevalent in many major cancer types, such as BC, GC, lung cancer, CRC, EC, BTC, HNSCC and CC, presenting a **huge market potential**



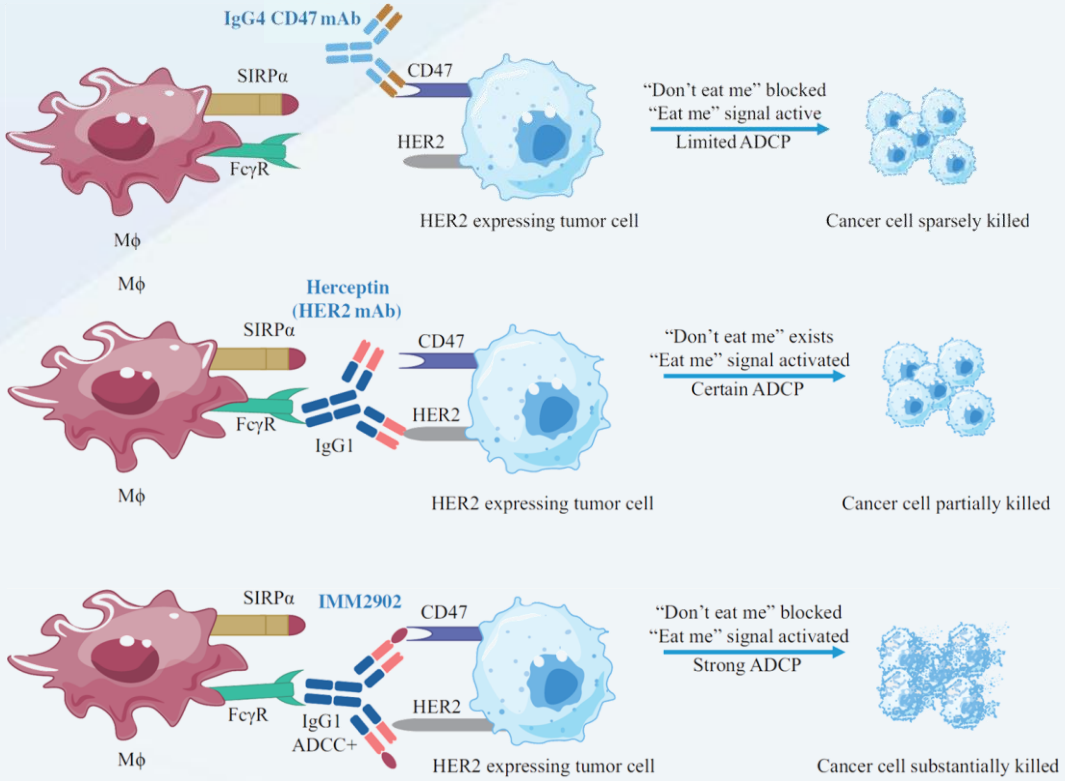
IMM2902 could benefit a large patient population globally

Safer and more efficacious treatment for patients with **HER2-low expressing** solid tumors and those relapsed from trastuzumab treatment

IMM2902 (CD47×HER2) (cont'd)

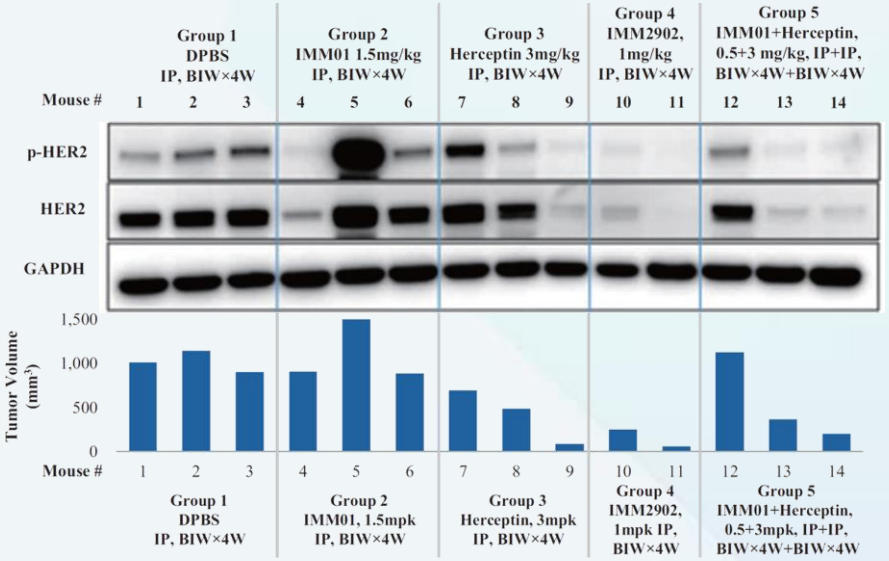
MoA and Competitive Advantages

Mechanism of Action



Enhanced ADCC, ADCP, potentially ADCT, and accelerated HER2 degradation

Expression Analysis of HER2 and p-HER2 by Western Blot (1)



- Preclinical study showed that IMM2902 could **accelerate the endocytosis and degradation of HER2**, resulting in robust tumor suppression
- IMM2902 is also expected to **potentially induce ADCT activity**, another important Fc-induced mechanism observed with amivantamab (a marketed EGFR/c-MET bispecific antibody with IgG1 Fc), which works together with **ADCC and ADCP** to combat tumor cells

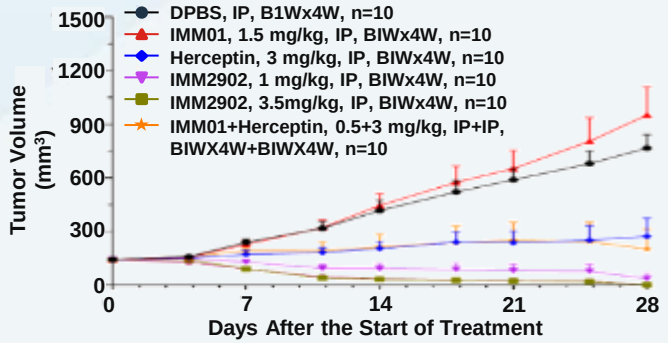
Notes:
1. p-HER2 refers to phospho-HER2, DPBS refers to Dulbecco's Phosphate Buffered Saline, intended to provide a buffer system for maintaining cell culture media in the physiological range of 7.2 to 7.6.

Source: Company data

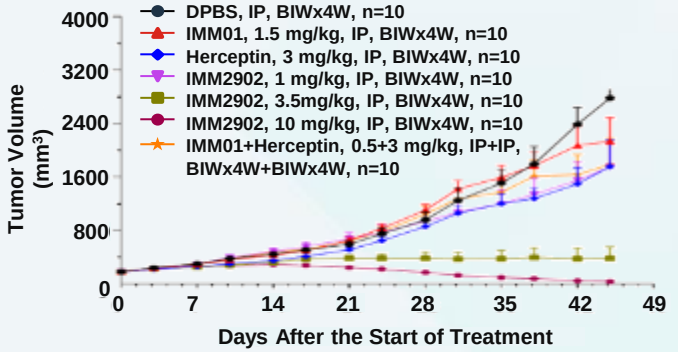
IMM2902 (CD47×HER2) (cont'd)

Preclinical Results – Strong *In Vivo* Antitumor Efficacy

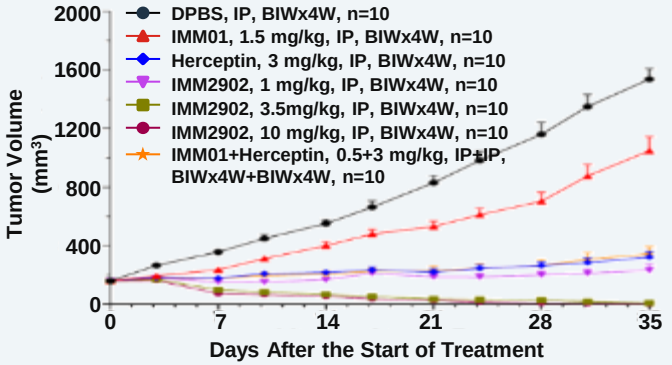
Efficacy Study in Herceptin-sensitive Breast Cancer (BT474) Xenograft Mouse Model



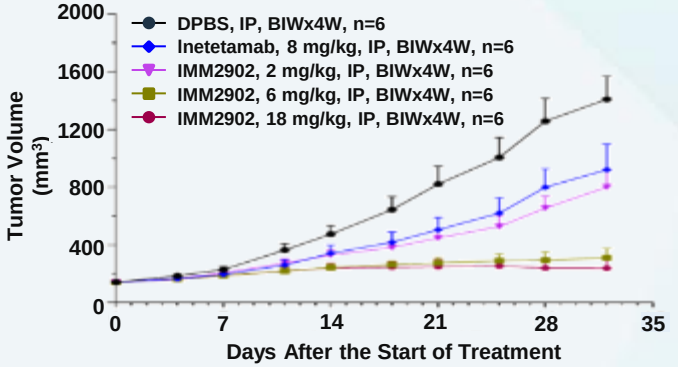
Efficacy Study in Herceptin-resistant Breast Cancer (HCC-1954) Xenograft Mouse Model



Efficacy Study in Herceptin-sensitive Gastric Cancer (NCI-N87) Xenograft Mouse Model



Efficacy Study in HER2-low Expressing Gastric Cancer (SNU-1) Xenograft Mouse Model



- IMM2902 exhibited favorable efficacy in **trastuzumab-sensitive and HER2-low expressing BC and GC models**

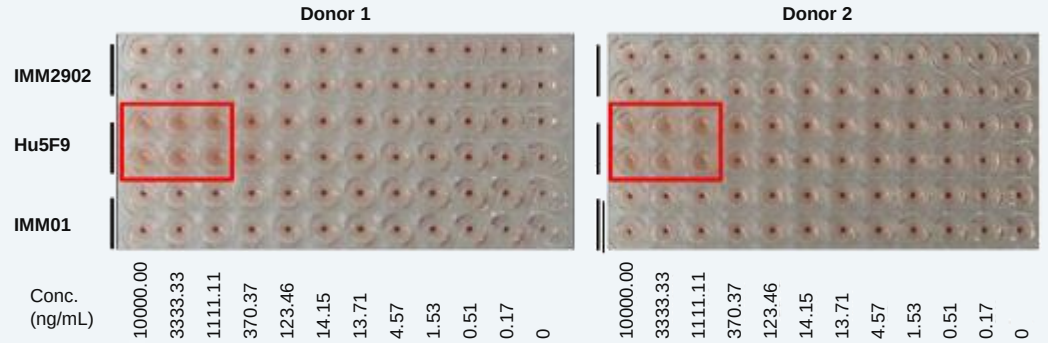
IMM2902 (CD47×HER2) (cont'd)

Preclinical Results and Clinical Development Plan

Favorable safety profile with no human RBC binding *in vitro*

- With an engineered CD47-binding domain, IMM2902 does not bind to human RBCs nor induces hemagglutination *in vitro*
- IMM2902 did not induce hemagglutination even at the concentration as high as 10,000 ng/ml; while magrolimab analog induced obvious hemagglutination at the concentration beyond 370 ng/ml

IMM2902 Does Not Induce Hemagglutination of Human Red Blood Cells (1)



Clinical Development Plan

- Phase I/II trial initiated in China in February 2022
- Evaluated for treatment of advanced HER2-positive and HER2-low expressing solid tumors, including BC, GC, NSCLC and BTC
- Enrolling the seventh cohort

- Phase Ia/Ib trial in the U.S. ongoing, with the first patient dosed in June 2022
- Evaluated for treatment of HER2-positive and HER2-low expressing solid tumors
- Received **Fast Track Designation from the FDA** in July 2022

Developing In-house and Own IP and Commercial Rights

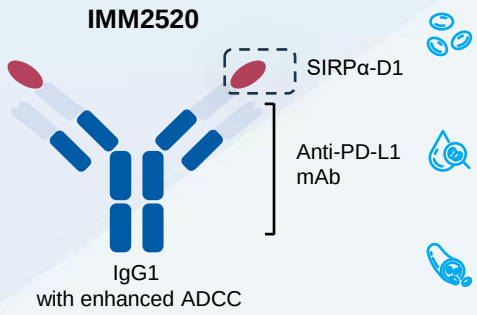
1 issued patent in the U.S. and ***1*** issued patent in Japan
1 allowed European patent application

3 pending patent applications in PRC, the U.S., and Hong Kong

Notes:
1. Magrolimab analog used in this study was replicated by us based on public information
Source: Company data

IMM2520 (CD47×PD-L1)

Overview



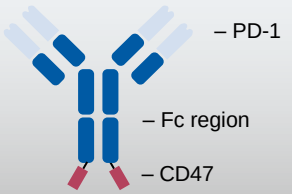
- A CD47 and PD-L1 dual-targeting bispecific molecule for the treatment of solid tumors
- Unique structure to **avoid RBC binding**
- Engineered ADCC-enhanced IgG1 Fc fragment to **fully activate macrophages** and **induce enhanced ADCP and ADCC activity**

Competition Landscape

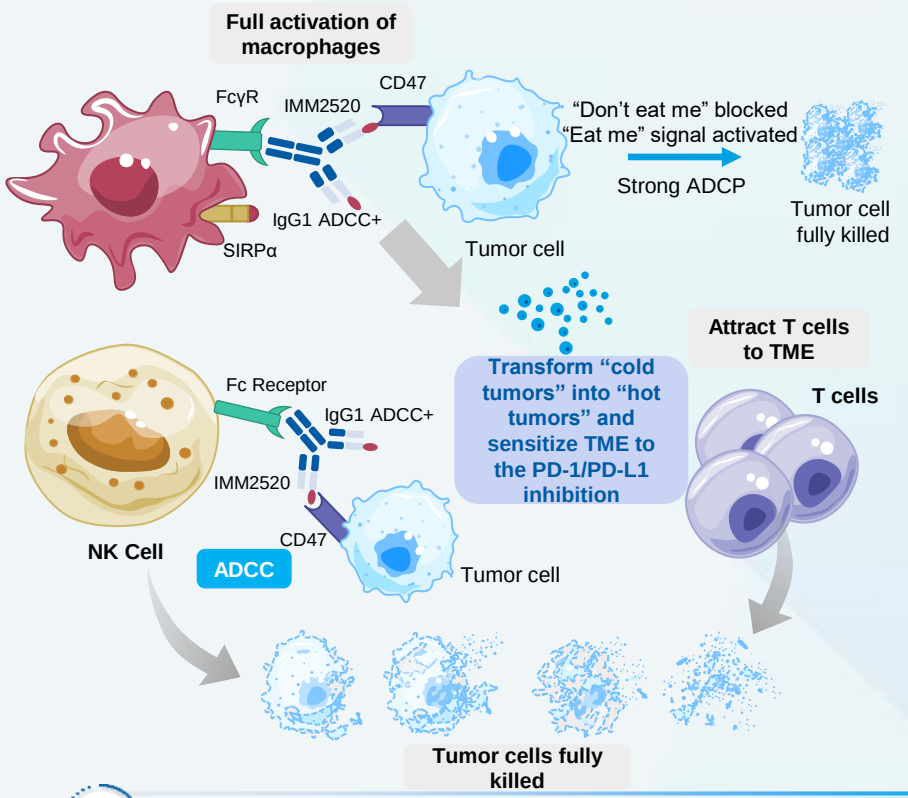
Other 9 CD47 and PD-1/PD-L1 bispecific molecules under clinical development

- ✓ Bispecific molecule formats: certain molecules connect the CD47-binding to the Fc end, which could disrupt immune activation resulted from Fc-FcγR engagement
- ✓ IgG4 Fc: several molecules resort to an IgG4 Fc region with weak FcγR engagement

HX009 (Hans Bio)



Mechanism of Action

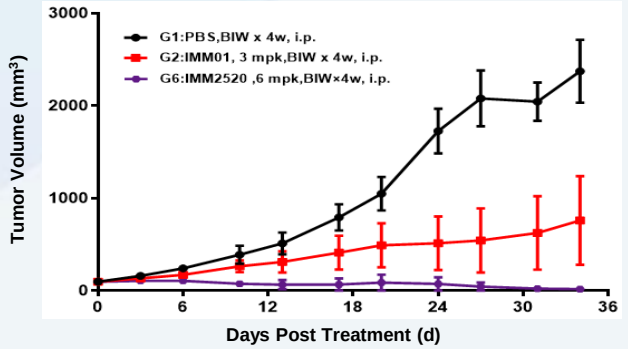


Due to the crosstalk among macrophages, NK cells and T cells, IMM2520 is able to unleash significant synergistic effects

IMM2520 (CD47×PD-L1) (cont'd)

Preclinical Results

Efficacy Study in Colon Cancer (CT26) Mouse Model (1)



Note:
1. IMM2505 is a first-generation CD47 and PD-L1 bispecific molecule internally developed by us; (2) Six mice per group were used in this study
Source: Company data

IMM2520 has also demonstrated **a favorable safety profile**. Its engineered CD47-binding domain shows no binding activity with human RBCs *in vitro*.

Developing In-house and Own its IP and Commercial Rights

- **1** issued patent in Japan
- **1** issued patent in PRC
- **1** issued patent in the U.S.
- Several pending patent applications in Europe, Korea and Brazil

Market Opportunities and Clinical Development Plan

Opportunities

A huge market potential for IMM2520

- ✓ **A wide range of cancer indications with high macrophage infiltration**
- ✓ Only about 10% to 25% of patients across almost all major cancer types respond to PD-1/PD-L1 inhibitor monotherapy, including but not limited to **NSCLC, SCLC, CRC, GC, HNSCC, HCC, ESCC, OC, prostate cancer, and pancreatic cancer**

Clinical Development Plan

Have obtained IND approvals from the NMPA in November 2022 and from the FDA in December 2022; Phase I commenced in China in March 2023.

Phase I Preliminary Efficacy

As of December 31, 2023, we have observed **3 SDs with over 10% tumor shrinkage**:

1 Cervical cancer (1L previous treatments) at 0.1 mg/kg with tumor shrinkage 21.1%;

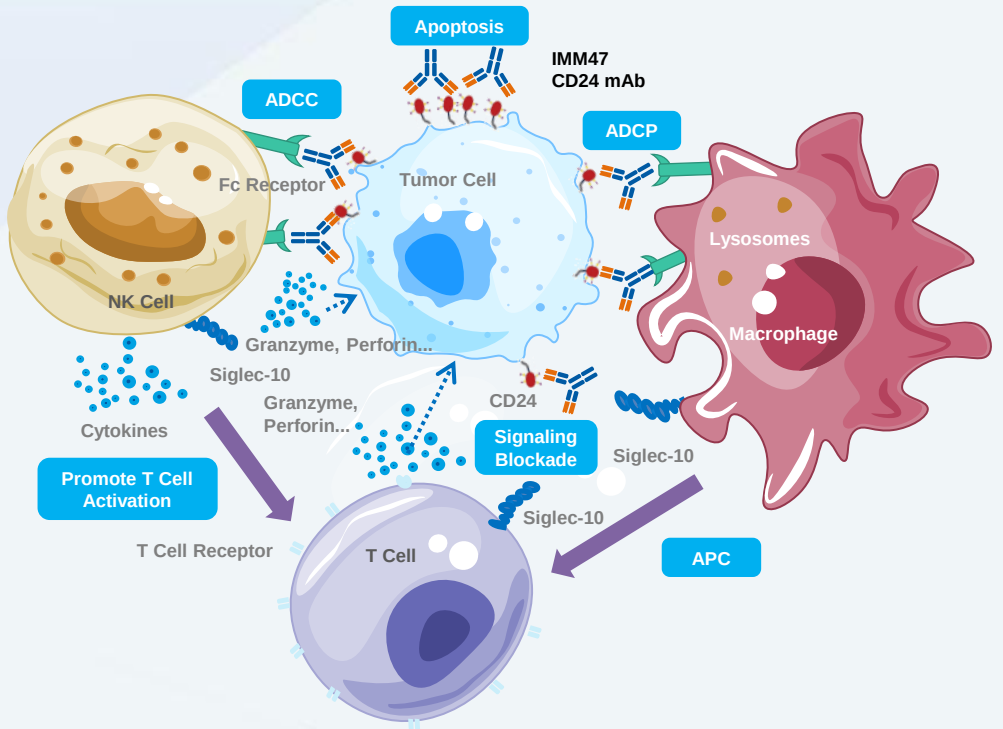
1 SCLC (previous IO failure, 2L previous treatments) at 2.0 mg/kg with tumor shrinkage 19.0% (further -26.3% by Jan 2024);

1 CRC (≥4L previous treatments) at 2.0 mg/kg with tumor shrinkage 11.4%;

IMM47 (CD24)

A Potential Global First-in-Class CD24-Targeted mAb

IMM47 – Molecule Structure and Mechanism of Action



IMM47 Molecule Structure

CD24 mAb
IgG1 Fc with
Enhanced ADCC

Block CD24/Siglec-10 immune inhibitory signaling

Induce ADCC/ADCP

Suppressing the CD24/Siglec-10 inhibitory signals sent to macrophages, NK cells and T cells

Potently activating macrophage and NK cell-immune responses through ADCP and ADCC

Significantly increasing the amount of M1 macrophages in tumor tissues

Activating and promoting T-cell response likely through tumor antigen presentation by activated macrophages to T cells and direct blockade of CD24/Siglec-10 inhibitory signals

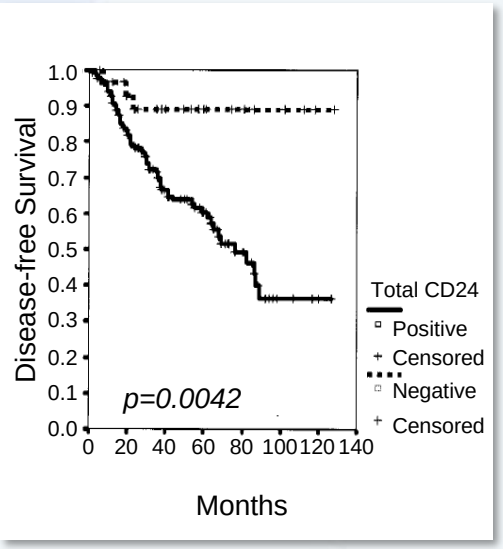
IMM47 (CD24) (cont'd)

CD24 is a Promising Target with Wide Expression and Broad Therapeutic Potential across Various Tumor Types

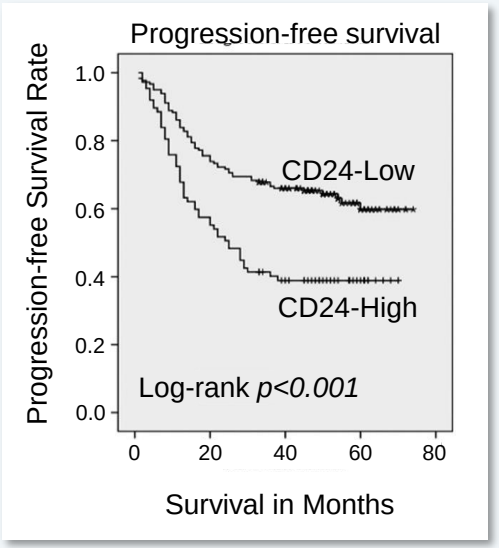
Broadly overexpressed on many types of tumor tissues, including B-cell lymphomas, erythroleukemia, gliomas, SCLC, ESCC, HCC, CCA, PAAD, UC, OC, BC, primary NECs, and PC[1]

Recognized as an important marker for poor prognosis of those cancers, presenting a huge market potential

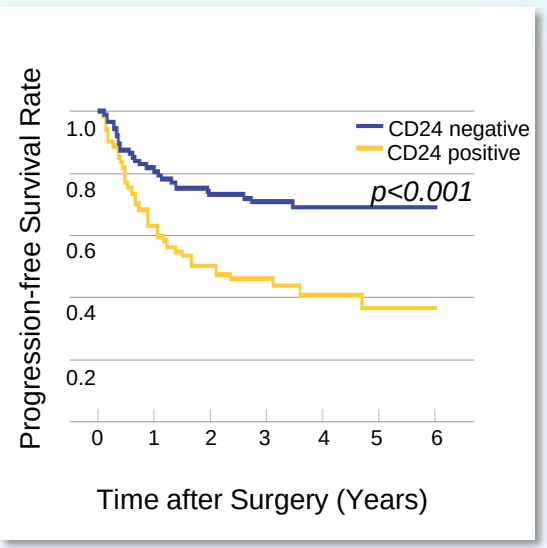
Primary Breast Cancer[2]



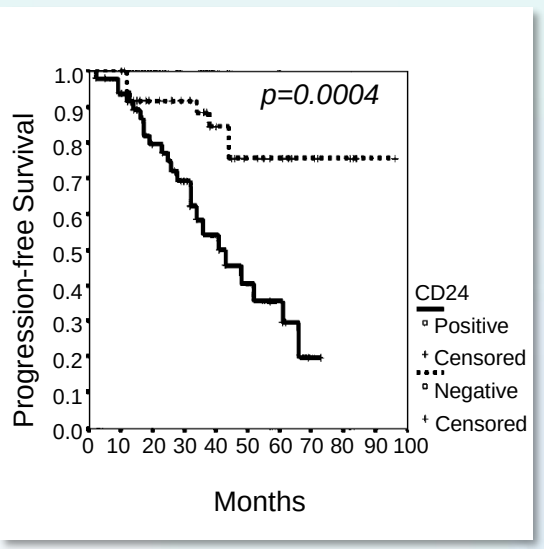
NSCLC[3]



ESCC[4]



Prostate Cancer[5]



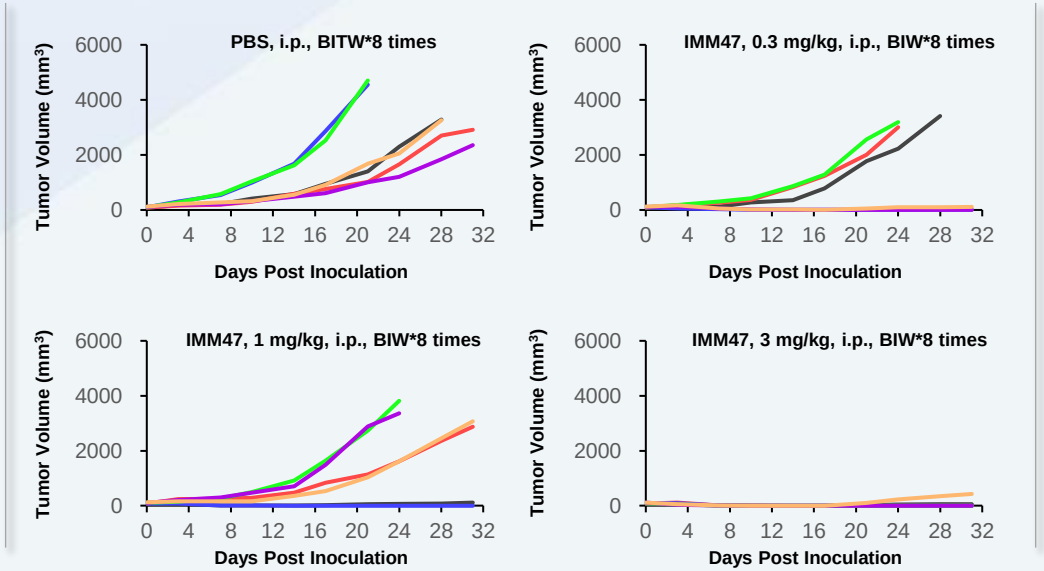
Source:
1. Cellular & Molecular Immunology (2010) 7, 100–103); 2. Clin Cancer Res 2003; 9:4906–4913; 3. J Thorac Oncol. 2010;5: 649–657; 4. Ann Surg Oncol (2009) 16:506–514; 5. The Prostate 58:183 ^192 (2004)

IMM47 (CD24) (cont'd)

Competitive Advantages

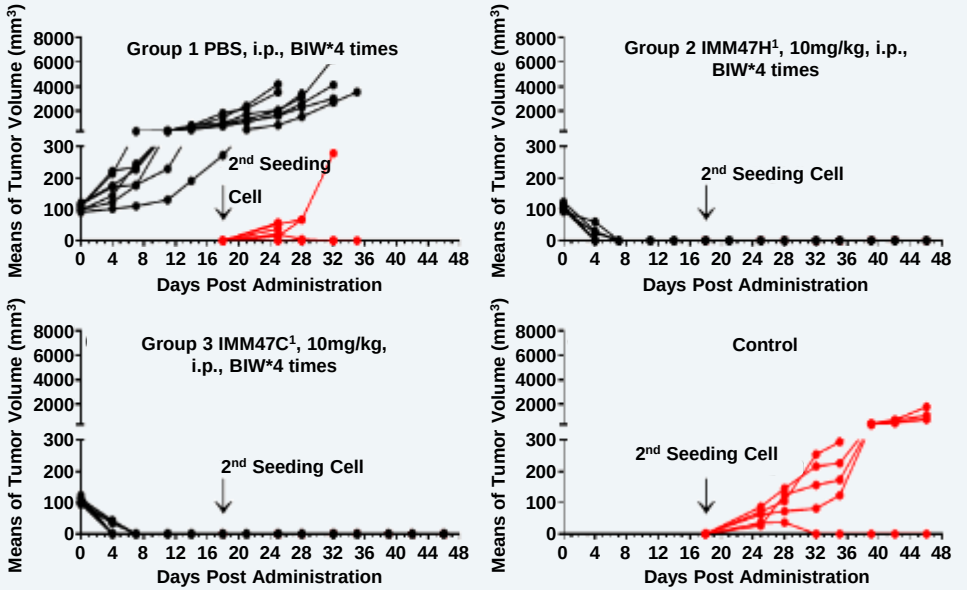
Proof-of-Concept Study in Colon Cancer (MC38-hCD24) Syngeneic Model in hSiglec-10 Tg C57BL/6 Mice

Compelling Tumor Killing Capabilities



- At the dose level of 3.0mg/kg (~0.3kg/mg in human), IMM47 successfully **eradicated subcutaneously inoculated tumor cell** in all six mice after three treatments in colon cancer model

Complete Tumor Eradication



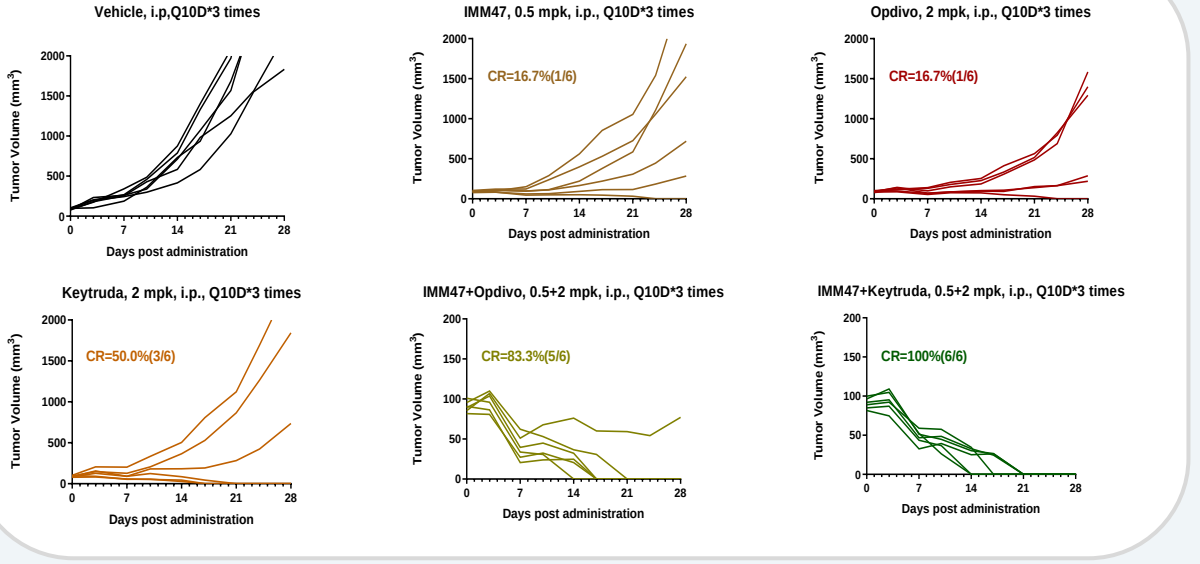
- Robust antitumor activities, leading to complete tumor eradication, with the ability to induce immunological memory against tumor
- Tumor-specific immune response that prevents tumor growth against re-inoculation

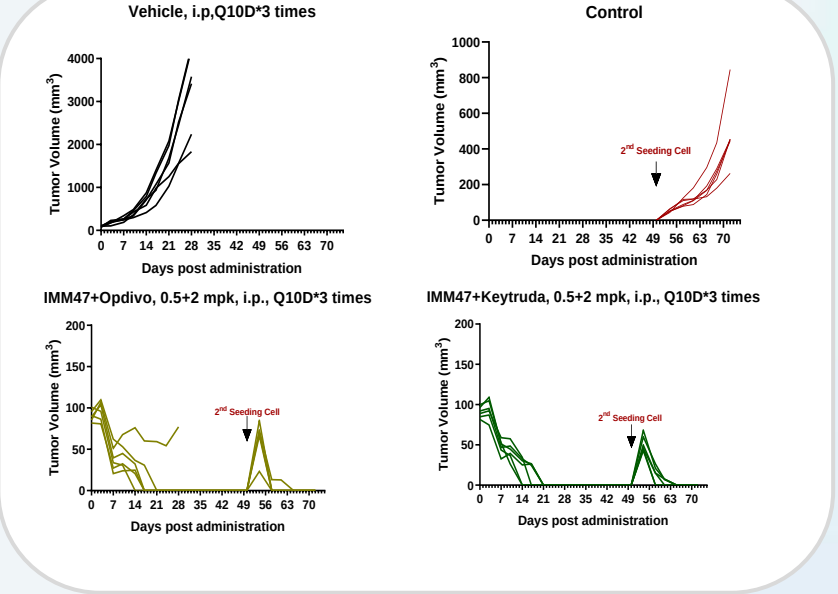
Notes:
1. IMM47C is a previous chimeric version of IMM47 and IMM47H is an earlier fully humanized version of IMM47. IMM47 revealed highly similar *in vitro* efficacy as IMM47C and IMM47H, and was eventually selected for further development

IMM47 (CD24) (cont'd)

Strong synergy between IMM47 and PD-1 mAb

Proof-of-Concept Study in MC38-hCD24/hPD-L1 syngeneic tumor model in hPD-1 Tg C57 BL/6 mice





- In comparison to the vehicle-controlled group, IMM47 (anti-CD24 antibody, 0.5mpk), Opdivo (anti-PD-1 antibody, 2mpk), and Keytruda (anti-PD-1 antibody, 2mpk), all showed significant but similar anti-tumor activity at relatively lower dose;
- While the combination of IMM47 with either Opdivo or Keytruda at comparable dose demonstrates a potent and robust anti-tumor activity, **with complete response rate of 83% and 100% respectively**

- Most intriguingly, upon reinoculation of the same cancer cells into the mice pretreated with IMM47 and anti-PD-1 antibodies, tumor growth was quickly and completely eliminated, **suggesting tumor-specific immune response has been established**

IMM47 (CD24) (cont'd)

Novel Target Development with Only a Handful Contenders, Well-Recognized by Industry Pioneers

IMM47 - Competition Landscape

No approved drug targeting CD24 globally. Only one drug candidate recently receiving IND approval from the FDA for its Phase I clinical trial

Global R&D race with few contenders: only very few reported CD24-targeted mAbs under pre-clinical development for cancer treatment have global first-in-class potential; ImmuneOnco as the only company reported to have been developing CD24-targeted bispecific molecule around the world

High entry barrier: the weak immunogenicity of CD24 due to its small protein core has made the screening and development of antibodies against CD24 highly challenging

Drug Name	Target	Modality	Clinical Stage
IMM47	CD24	mAb	IND Enabling
IMM4701	CD47 x CD24	Bispecific	Preclinical
IMM2547	CD24 x PD-L1	Bispecific	Discovery

宜明昂科 ImmuneOnco

*The **ONLY** company reported to have been developing CD24-targeted bispecific molecules*

Clinical Development Plan

2023

File IND applications with the NMPA and FDA

Sep 2023

Initiated clinical trial first in Australia （FPI）

Recent Catalysts: Validation from Industry Veterans and Pioneers

Pheast Therapeutics

Founders	Latest Financing	Key Financial Investors	Strategic/CVC/ Research Institutes
▪ Dr. Amira Barkal ▪ Dr. Irving Weissman	Series-A: US$76MM (with an estimated valuation of US$304-456MM)	ARCH Venture Partners CATALIO Alexandria Venture Investments	Stanford University

Proprietary Intellectual Property

1 issued patent in the PRC and ***1*** issued patent in Japan

1 allowed patent application in the U.S.

1 pending patent application in Europe

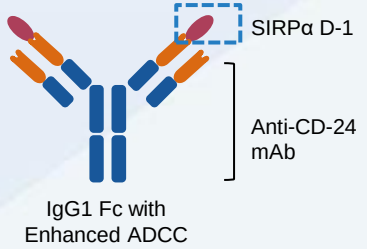
Source: Frost & Sullivan, public information, https://datacommons.technation.io/companies/pheast_llc

IMM4701 (CD47 × CD24)

A Bispecific Molecule Targeting CD24 and CD47 with Global First-in-Class Potential

IMM4701 - Mechanism of Action

IMM4701 Molecule Structure



- Activating key innate and adaptive immune responses
- Enhancing the synergistic crosstalk

IMM4701 - Competitive Advantage

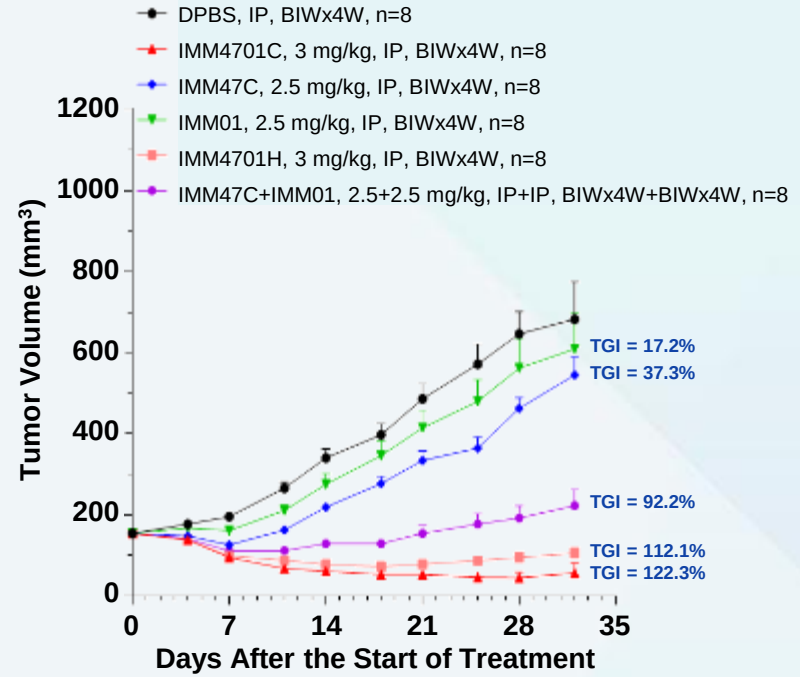
- Undisputable Global Leader
- Carefully Designed Clinical Roadmap
- Potent and Synergistic Immune Activation
- Promising Combination Partner

Developing One Owned Patent Family

- ***3*** issued patents in China, Japan and the U.S.
- ***1*** patent application in Europe

Strong and Robust Antitumor Activities

Efficacy Study in Triple-negative Breast Cancer (MCF-7) Xenograft Mouse Model



Notes:

1. IMM47 revealed highly similar in vitro efficacy as IMM47C (a previous chimeric version of IMM47) and IMM47H (a previous fully humanized version of IMM47), and was eventually selected for the further development. IMM4701, IMM4701C and IMM4701H were developed based on IMM47, IMM47C and IMM47H, respectively
2. IMM2547 is another innovative discovery-stage bsAb targeting CD24 × PD-L1 developed by the Company

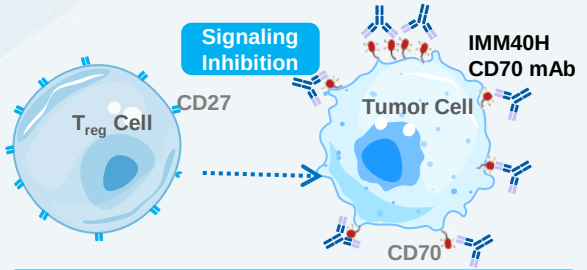
IMM40H (CD70)

A Humanized IgG1 CD70 mAb with Potential to Combo with IMM01

IMM40H – Mechanism of Action

IMM40H Molecule Structure

Humanized IgG1 CD70 mAb with High Target Affinity



Obstructing the activation and proliferation of T_{reg} cells

Inducing immune attacks against tumor cells

IMM40H - Competitive Advantage

Promising adaptive immune checkpoint: IMM40H demonstrated strong synergistic potential when used alongside IMM01

Leading R&D progress worldwide: the Company is one of the first few companies to develop molecules targeting CD70

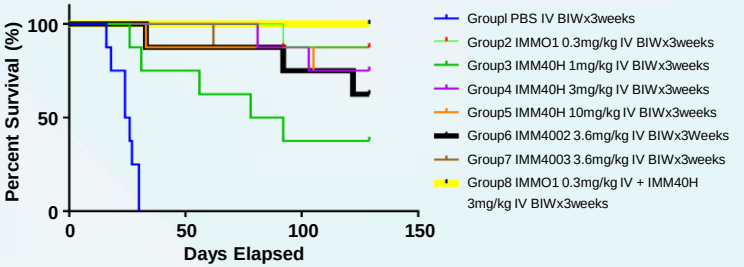
Developing One Owned Patent Family

- ***1*** issued patent the U.S.
- ***1*** issued patent in the PRC
- ***1*** issued patent in Japan
- ***1*** pending patent application in Europe

Potent In *Vivo* Antitumor Effects in Combination with IMM01

Efficacy Study in Lymphoma (Raji) Orthotopic Mouse Model[1]

- IMM40H has exhibited **strong antitumor activity** in the preclinical studies.
- **Strong synergism** between IMM01 and IMM40H has been observed



Other Pre-clinical Benefits of IMM40H

- Stronger CD70-binding affinity than cusatuzumab
- Demonstrated potent ADCC, CDC and ADCP activity
- Favorable safety profile of IMM40H

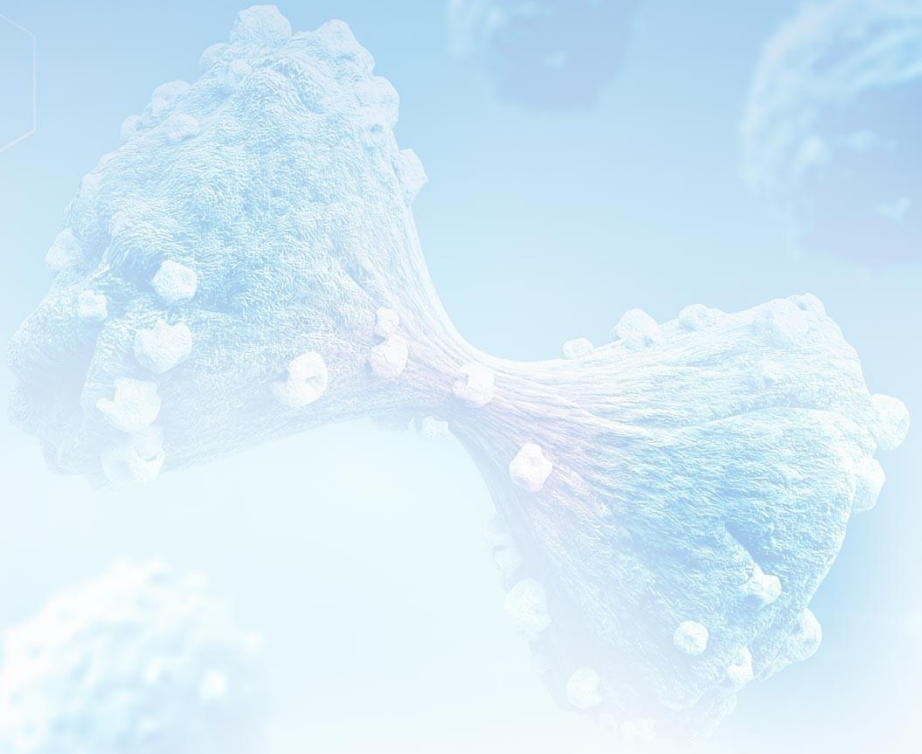
Current Status

IND approved by both NMPA and FDA in August 2022

宜明昂科
ImmuneOnco

SECTION 4

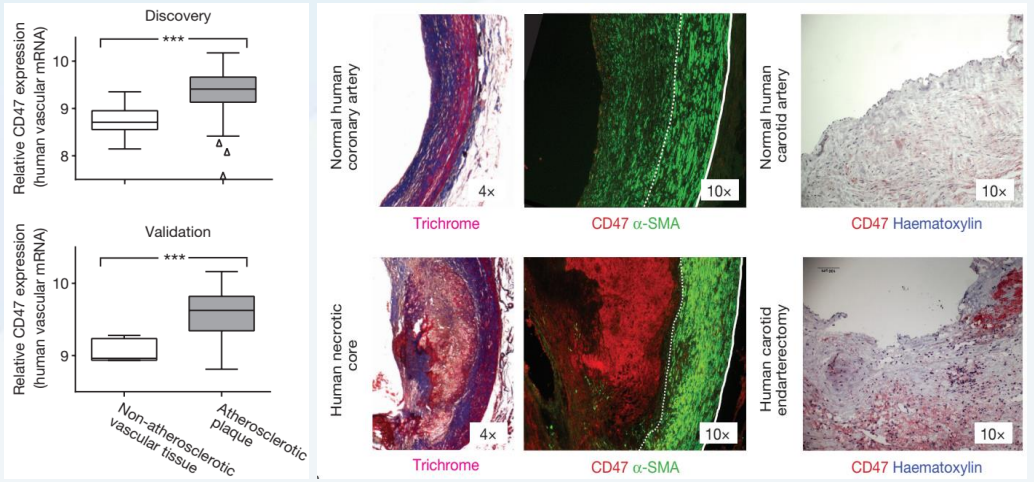
Non-Oncology Pipeline Introduction



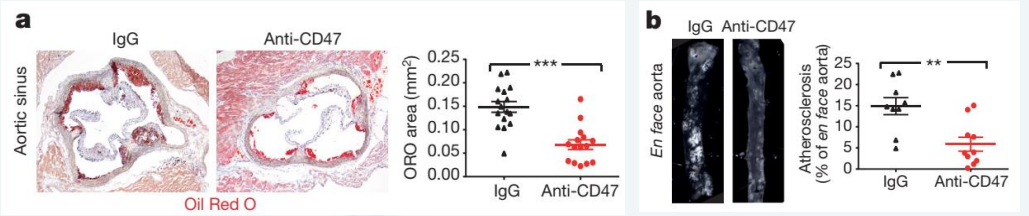
IMC-001 (IMM01, SIRPα-Fc)

Our CD47-targeted IMM01 presents a strong potential in treating atherosclerosis

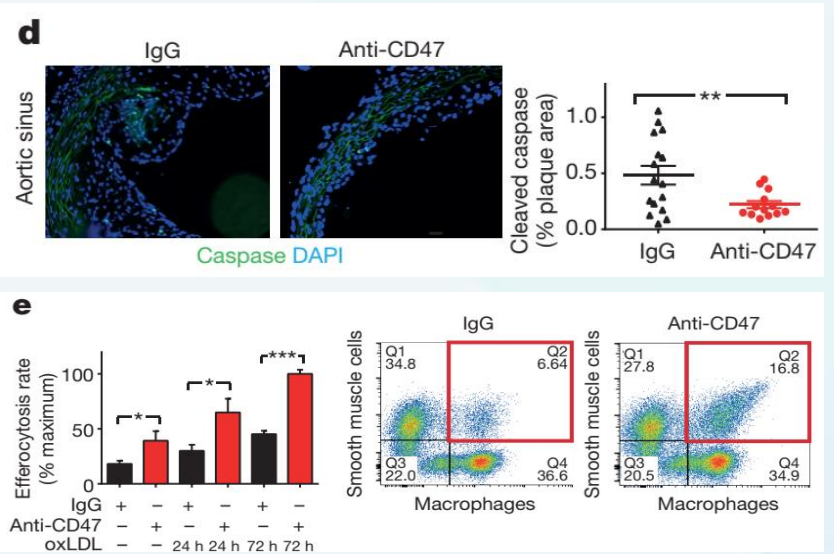
CD47 is highly expressed in human atherosclerotic plaque

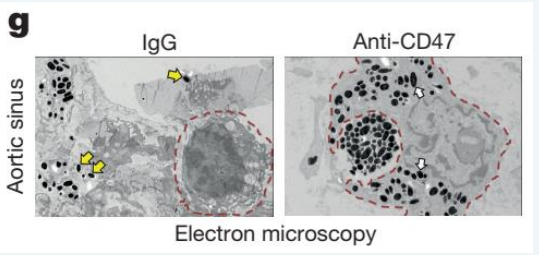


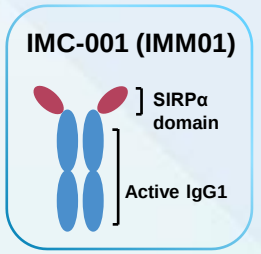
Shrinkage of atherosclerotic plaque was observed in rat model by blocking the CD47/SIRPα signaling pathway



By blocking the CD47 signal, macrophages can phagocytose the atherosclerotic plaque in rat vessel





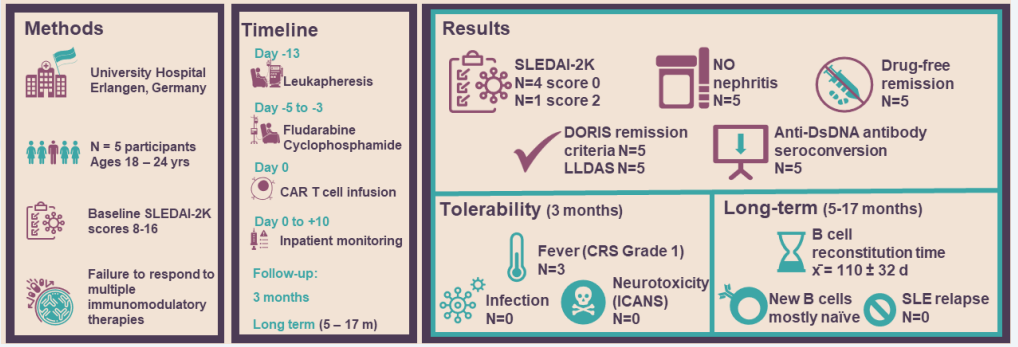


Reference: Yoko Kojima, et al., 86 , Nature, Vol 536, August 2016;

IMC-002 (IMM0306, CD47xCD20/mAb-Trap)

B-cell depletion was observed in IMM0306 clinical studies, presents a strong potential in the treatments of autoimmune diseases

A study conducted by Dr. Georg Schett at the University of Erlangen-Nuremberg showed that **deep depletion of B cells** was observed following CD19+ CAR-T treatment in 15 patients, including 8 with systemic lupus erythematosus (SLE), 3 with idiopathic inflammatory myopathies (IIM), and 4 with multiple sclerosis (MS). All patients achieved drug-free remission after the reappearance of B cells, and there were no relapses reported during the 2-year follow-up period.

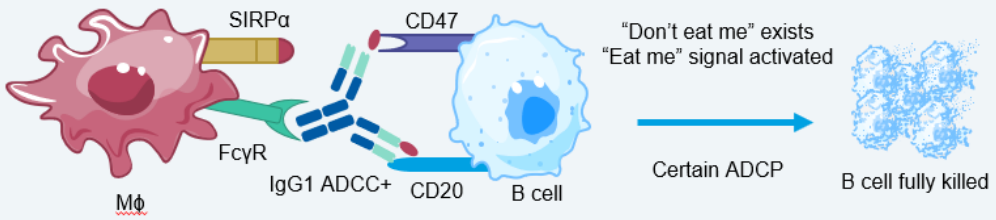


RITUXAN® (rituximab, CD20) was approved by FDA in 2006 for the treatment of **rheumatoid arthritis (RA)**;

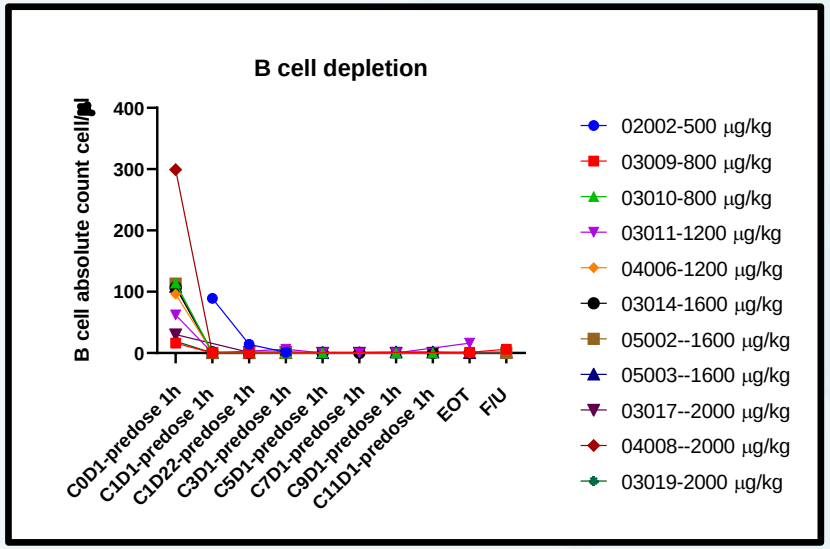
BRIUMVI™(Ublituximab, CD20) was approved by FDA in 2022 for the treatment of **multiple sclerosis (MS)**;

B-cell depletion therapies (BCDTs) are widely used in the treatments of autoimmune diseases.

Mechanism of Action



Upon binding with CD20 and CD47, **IMC-002 is expected to deplete B cells by inducing enhanced ADCC and ADCP activity**



Reference: Dimitrios Mougiakakos, et al., August 5, 2021, N Engl J Med 2021; 385:567-569; Andreas Mackensen, et al. NATURE MEDICINE, VOL 28, Oct. 2022, 2124–2132

IMC-003 (IMM72, ActRIIA/Fc-fusion)

ActRII biology in reducing fat mass while preserving muscle mass

ActRII biology in adipose tissue

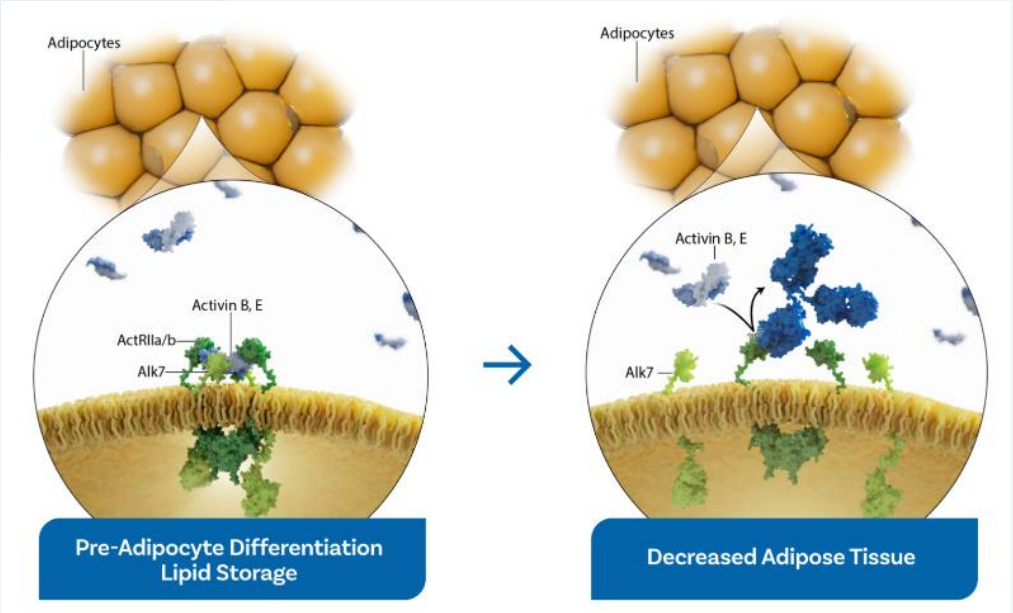
Activin signaling via ActRII receptors directly promotes lipid storage, acting as a key driver of visceral fat accumulation and obesity

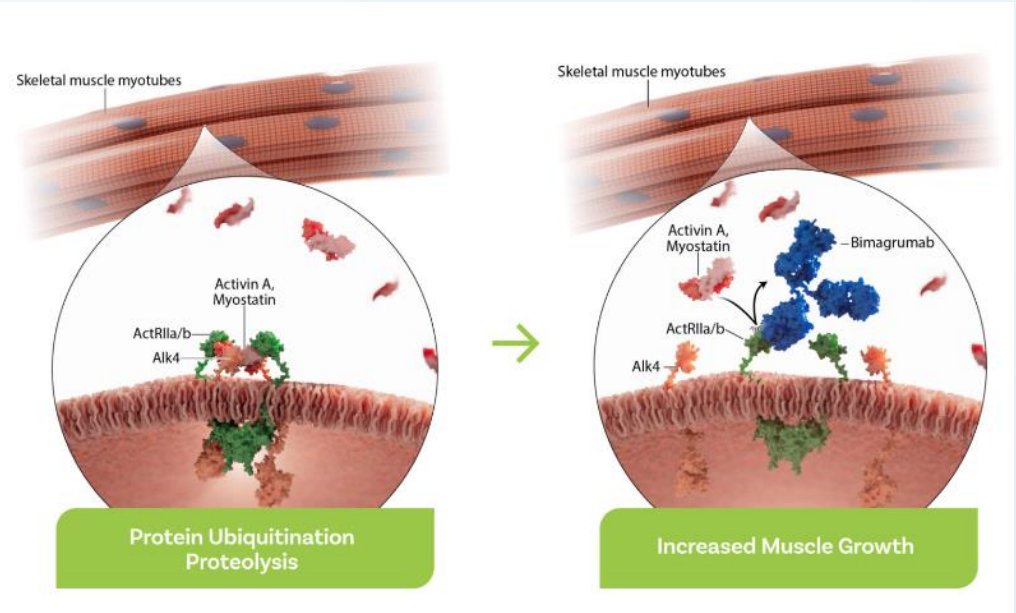
By blocking ActRII signaling in adipose cells, can mobilize and metabolize fat.

ActRII biology in muscle tissue

Signaling via ActRII receptors inhibits muscle growth and promotes atrophy.

Blocking activin signaling in skeletal muscles inhibits this atrophy and can promote increases in muscle mass, helping patients with obesity improve body composition and metabolism while losing fat.





Reference: versanisbio.com

IMC-003 (IMM72, ActRIIA/Fc-fusion)

Recent deal: Eli Lilly completed the acquisition of Versanis Bio in up to $1.925 billion cash

Lilly Completes Acquisition of Versanis Bio

August 14, 2023

INDIANAPOLIS, Aug. 14, 2023 /PRNewswire/ -- Eli Lilly and Company (NYSE: LLY) today announced the successful completion of its acquisition of Versanis Bio. The acquisition expands Lilly's portfolio to include Versanis' lead asset, bimagrumab, which is currently being assessed in a Phase 2b study alone and in combination with semaglutide in adults living with overweight or obesity.

"Combining our current incretin portfolio, including tirzepatide, with activin receptor blockers such as bimagrumab, could be the next major step in innovative treatments for those living with cardiometabolic diseases, like obesity," said Ruth Gimeno, Ph.D., group vice president, diabetes, obesity and cardiometabolic research at Lilly. "The wealth of knowledge that our new colleagues from Versanis will bring to Lilly will propel our research and development efforts forward, ultimately benefiting patients around the world."

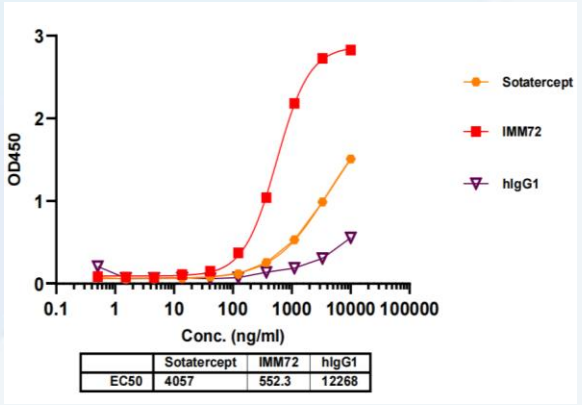
Under the terms of the agreement, Versanis shareholders could receive up to $1.925 billion in cash, inclusive of the upfront payment and subsequent payments upon achievement of certain development and sales milestones.

For Lilly, Kirkland & Ellis LLP is acting as legal counsel. For Versanis, Goodwin Procter LLP is acting as legal counsel, Cooley LLP is advising as to patent matters, and J.P. Morgan and Company is acting as financial advisor.

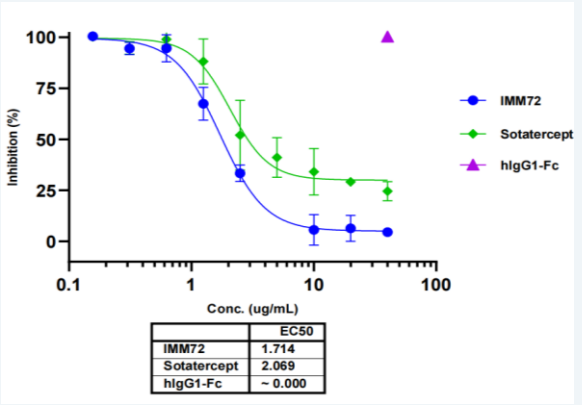
IMC-003 (IMM72, ActRIIA/Fc-fusion)

Preclinical Results

Compared to Sotatercept, IMC-003 has stronger binding and blocking capacity



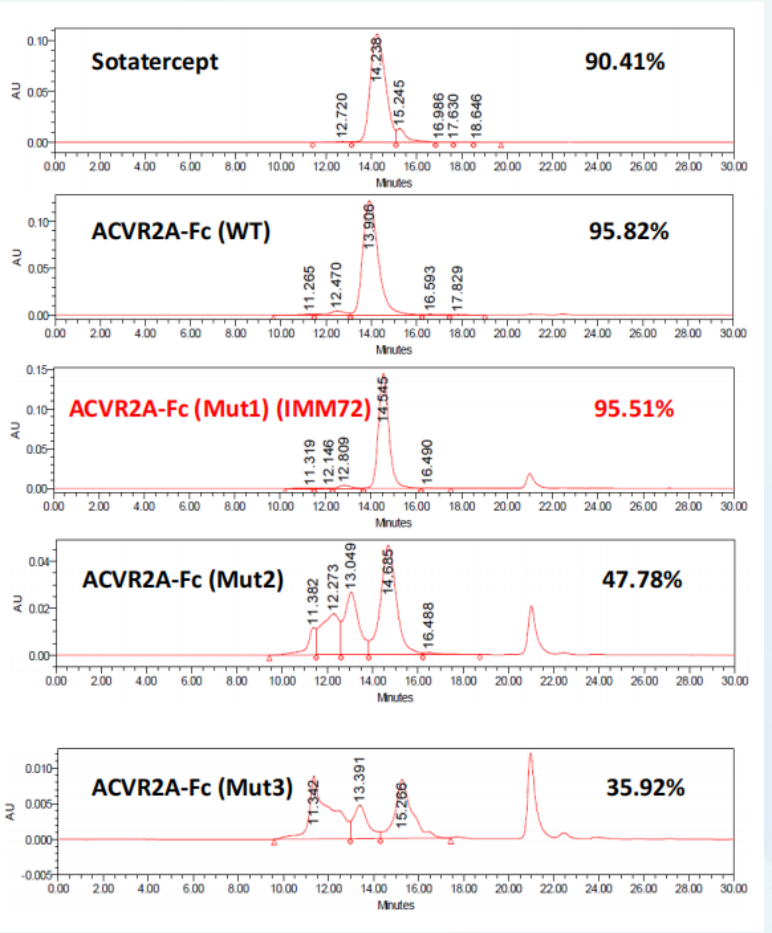
	Sotatercept	IMM72	hIgG1
EC50	4057	552.3	12268



	EC50
IMM72	1.714
Sotatercept	2.069
hIgG1-Fc	~ 0.000

	IMC-003 (IMM72)	Sotatercept
Company	ImmuneOnco (ImmuneCare)	Merck
Molecule Structure	ActRIIA-Fc (Engineered)	ActRIIA-Fc (Wild-type)
Affinity Assay	Similar, but higher response	Similar
Binding Activity	**7x than Sotatercept**	Moderate
Blocking Activity	**Strong**	Moderate
***In Vivo* Efficacy**	**Strong**	Moderate

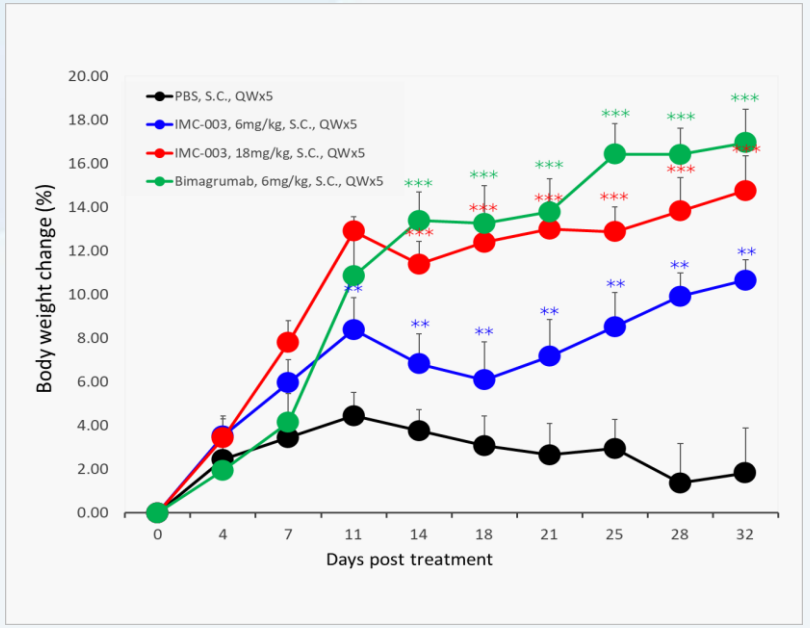
SEC Analysis



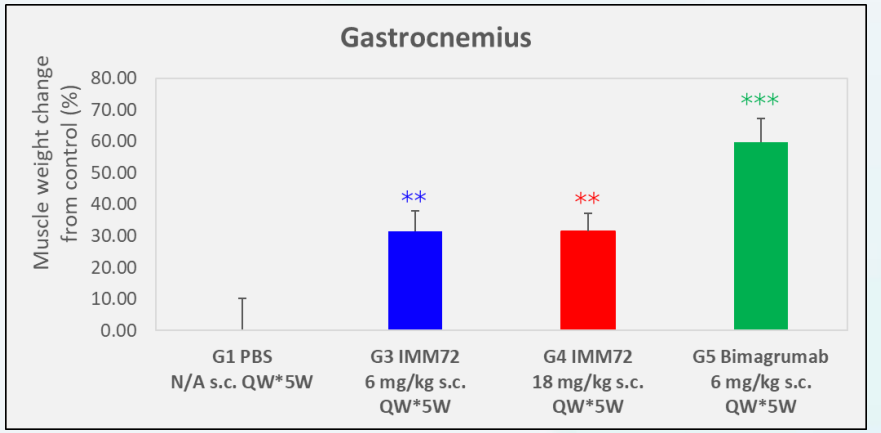
IMC-003 (IMM72, ActRIIA/Fc-fusion)

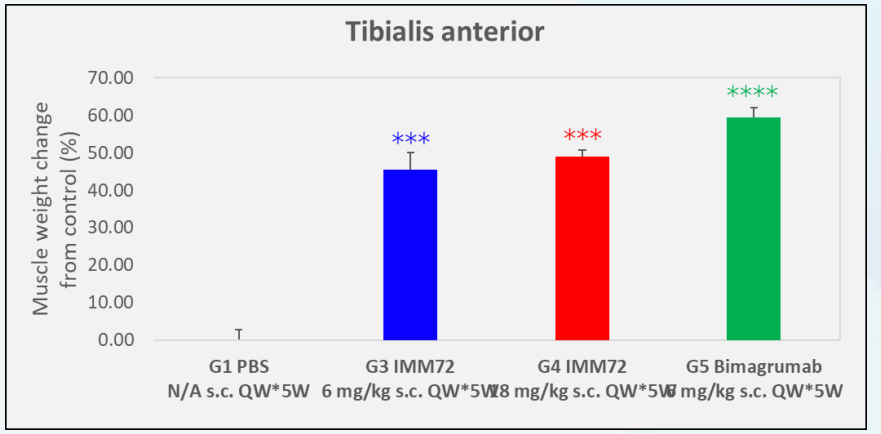
Preclinical Results helps build muscle and lose weight

Body weight increased substantially by IMC-003 treatment



Skeletal muscle increased substantially by IMC-003 treatment

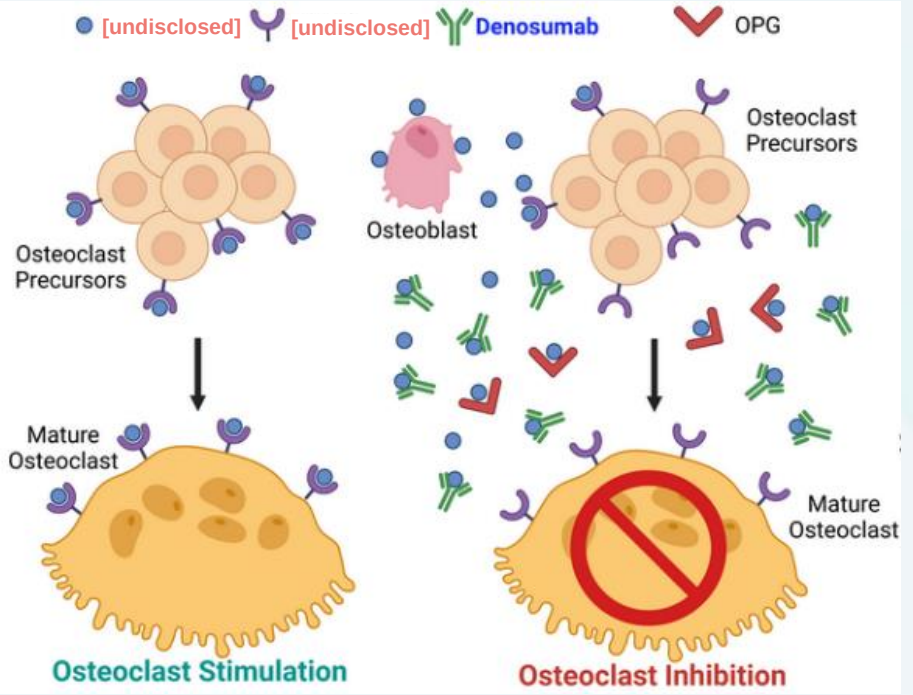


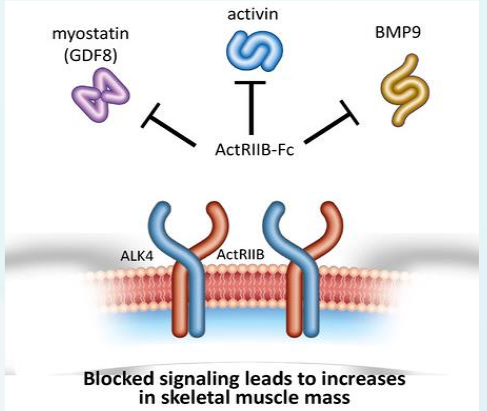


IMC-004 (IMM7211, [undisclosed] x ActRIIA/mAb-Trap)

A Bispecific molecule Targeting [undisclosed] and ActRIIA with Global First-in-Class Potential

- The binding of [undisclosed] to its receptor triggers osteoclast precursors to differentiate into osteoclasts and results in osteoporosis.
- Activin A can stimulate the formation of osteoclasts. **By blocking the Activin A/ActRIIA signaling pathway** can inhibit the formation of osteoclasts and increase the bone density, and also leads to increases in skeletal muscle mass.
- **IMC-004 (IMM7211)** is expected for the better treatments of osteoporosis and skeletal muscle mass decrease, **by blocking both [undisclosed] and Activin A/ActRIIA signaling pathway**.



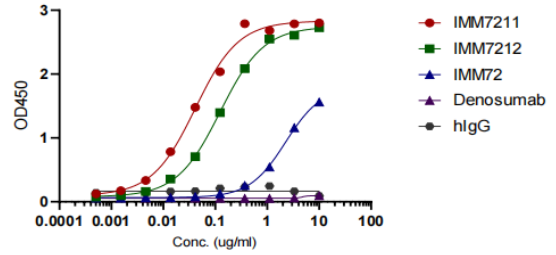


IMC-004 (IMM7211, [undisclosed] x ActRIIA)

Preclinical Results

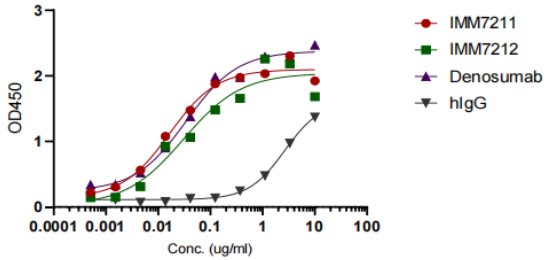
IMC-004 (IMM7211) has stronger binding and blocking capacity than IMM7212 on Activin A and [non-disclosed]; and is similar to Denosumab on [non-disclosed]

20230629 Binding of IMM7211 to Activin A by ELISA



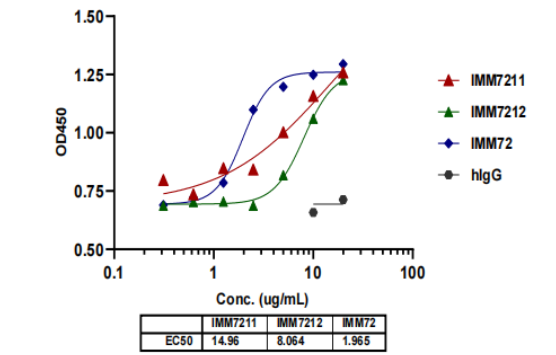
	EC50
IMM7211	0.04139
IMM7212	0.1245
IMM72	2.260

20230629 Binding of IMM7211 to [undisclosed] by ELISA



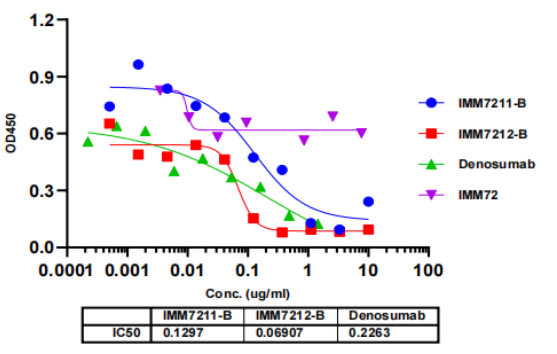
	EC50
IMM7211	0.01644
IMM7212	0.03036
Denosumab	0.03438

20230630 IMM7211 relieves the inhibitory effect of activin A on MPC-11 cell proliferation



	IMM7211	IMM7212	IMM72
EC50	14.96	8.064	1.965

IMM7211/IMM7212 Blocking the Interaction of [undisclosed]



	IMM7211-B	IMM7212-B	Denosumab
IC50	0.1297	0.06907	0.2263