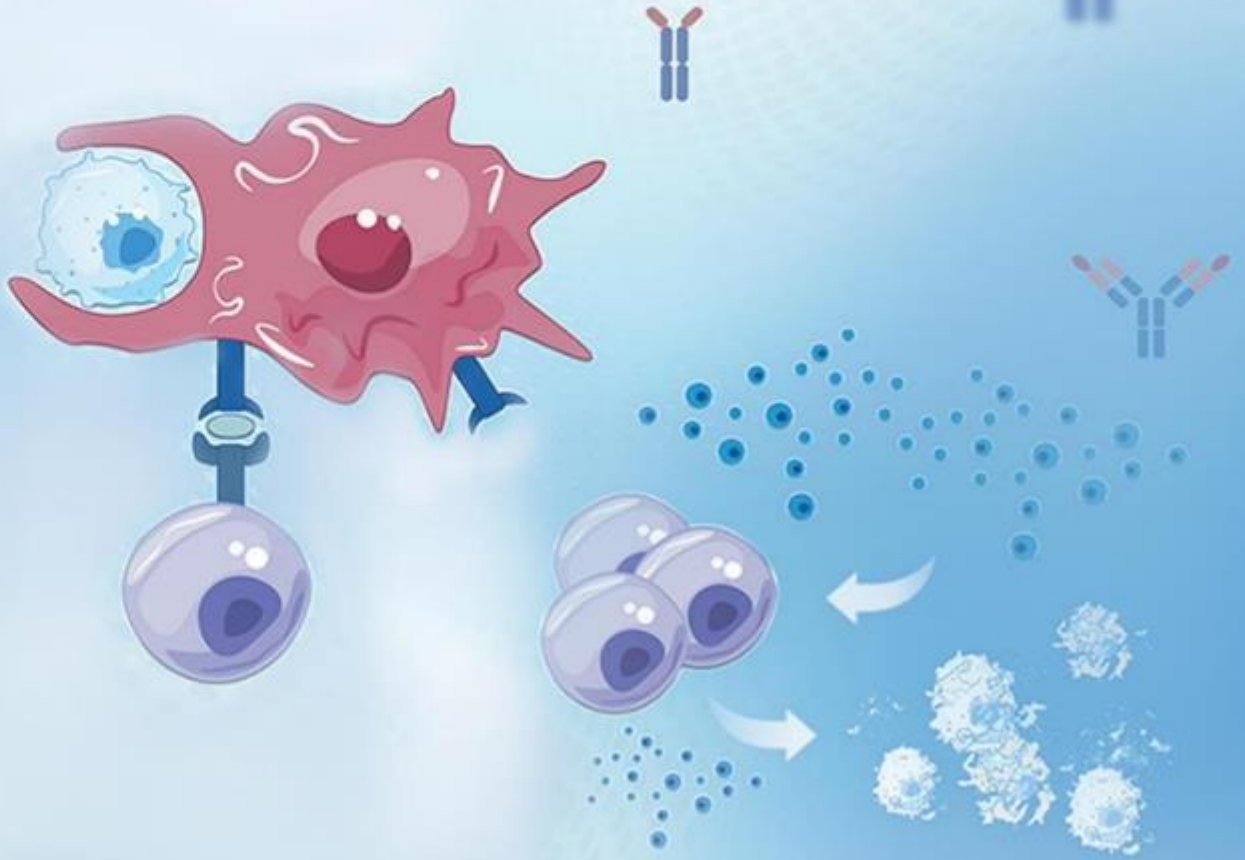
宜明昂科
ImmuneOnco

Corporate Presentation

September 2024



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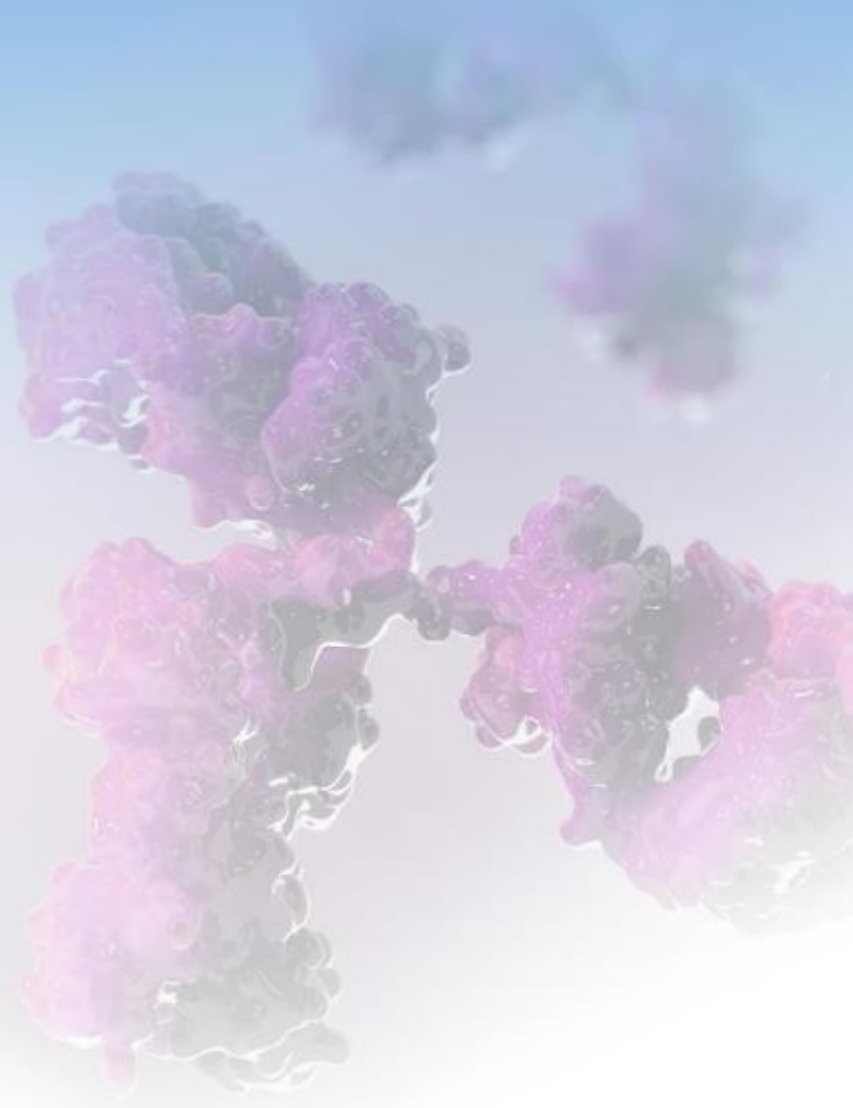
SECTION 1

Company Overview



Key Milestones

- Steady team with **10+** years coordination
- **30** issued patents
- **23** pending patent applications
- **29** IND approvals from the NMPA and the FDA
- **8** ongoing clinical 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 in 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

IMM01:
- Phase II in combination with either PD-1 mAb or azacitidine commenced in China

IMM2902:
- Phase I dosed patients in both China and US

IMM27M:
- Phase I trial patients dosed in China

IMM40H & IMM2520:
- IND approval by NMPA and FDA

2023

IMM01:
- Orphan drug designation in the U.S.

IMM0306:
- Phase Ib/IIa initiation in China in combination with lenalidomide and dosed its first patient

IMM2510:
- Phase I dose escalation LPI and RP2D determined
- IND approved for IMM2510+ chemo and IMM2510+ IMM27M in China
- Phase II monotherapy for R/R STS dosed first patient

IMM27M:
- Phase I dose escalation LPI and RP2D determined in China

IMM47:
- IND approval by NMPA
- Dosed first patient in Australia

2024

IMM01:
- Three phase III clinical trials approved for MDS, CMML and cHL in China
- Phase III IMM01+PD-1 mAb for PD-(L) 1-refractory cHL dosed first patient

IMM0306:
- Phase II of IMM0306+ lenalidomide initiated for advanced R/R FL
- Phase Ib of IMM0306+ lenalidomide for R/R DLBCL dosed first patient
- Two IND approvals in autoimmune field

IMM2510:
- Phase Ib in combination with IMM27M for solid tumors dosed first patient
- Reached a license-out agreement of US$2.1B with Instil Bio

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

宜明昂科 ImmuneOnco

Management team

Wenzhi Tian, MD, EMBA
Founder, Chairman & CEO

- ***30+*** years academic and industrial experience in the field of immuno-oncology
- ***29*** IND approvals from the NMPA and the FDA
- ***30*** issued patents, ***23*** 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

- **29** IND approvals from the NMPA and the FDA
- **30** Issued patents
- **23** 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	Partners	Current Status / Upcoming Milestone	Commercial Rights
IMM01 （Timdarpacept）											
IMM01 + Azacitidine	CD47 (SIRPα-Fc fusion protein)	MDS[2]	China (NMPA)							Received Phase III approval from CDE in May	Global
IMM01 + Azacitidine	CD47 (SIRPα-Fc fusion protein)	CMML	China (NMPA)							Received Phase III approval from CDE in June	Global
IMM01 + Tislelizumab	CD47+PD-1	cHL[3], Solid tumor	China (NMPA)							Received Phase III approval from CDE in April; FPI on July 1, 2024	Global
IMM2510 Monotherapy	VEGFxPD-L1 (Bispecific)	STS, Solid Tumor	China (NMPA)						InstilBio	Phase Ib/II commenced in November 2023 in China	Great China
IMM2510 + Chemo	VEGFxPD-L1 (Bispecific)	1L TNBC, 1L NSCLC	China (NMPA)						InstilBio	IND approved in China in November 2023	Great China
IMM2510 + IMM27M	VEGFxPD-L1 (Bispecific) + CTLA-4	2L HCC, TNBC	China (NMPA)						InstilBio	IND approved in China in October 2023, FPI on July 24, 2024	Great China
IMM27M	CTLA-4 ADCC+ (mAb)	Solid tumors	China (NMPA)						InstilBio	Phase I completed in September 2023 in China and RP2D confirmed	Great China
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
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
IMC-002 (IMM0306)	CD47xCD20 (Bispecific)	SLE, NMOSDs								IND approved in China for the treatment of SLE and NMOSDs in June 2024, expecting to initiate enrollment in Q4, 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 (IMM7211c)	ActRIIA x Non-disclosed (Bispecific)	Undisclosed								IND-enabling in one and a half year	Global

Source: Company Data

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

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

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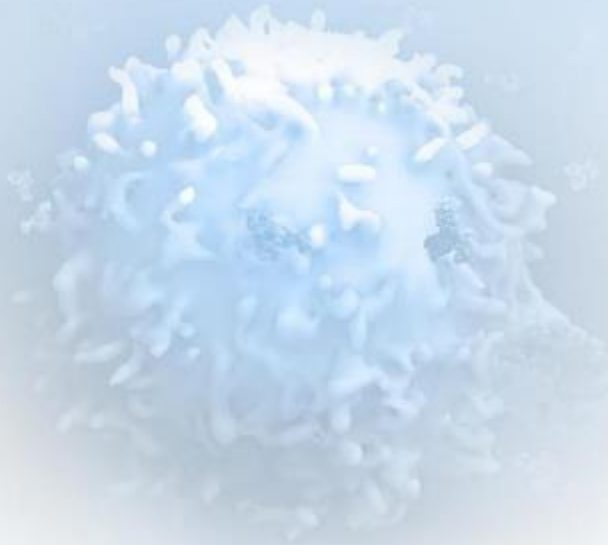
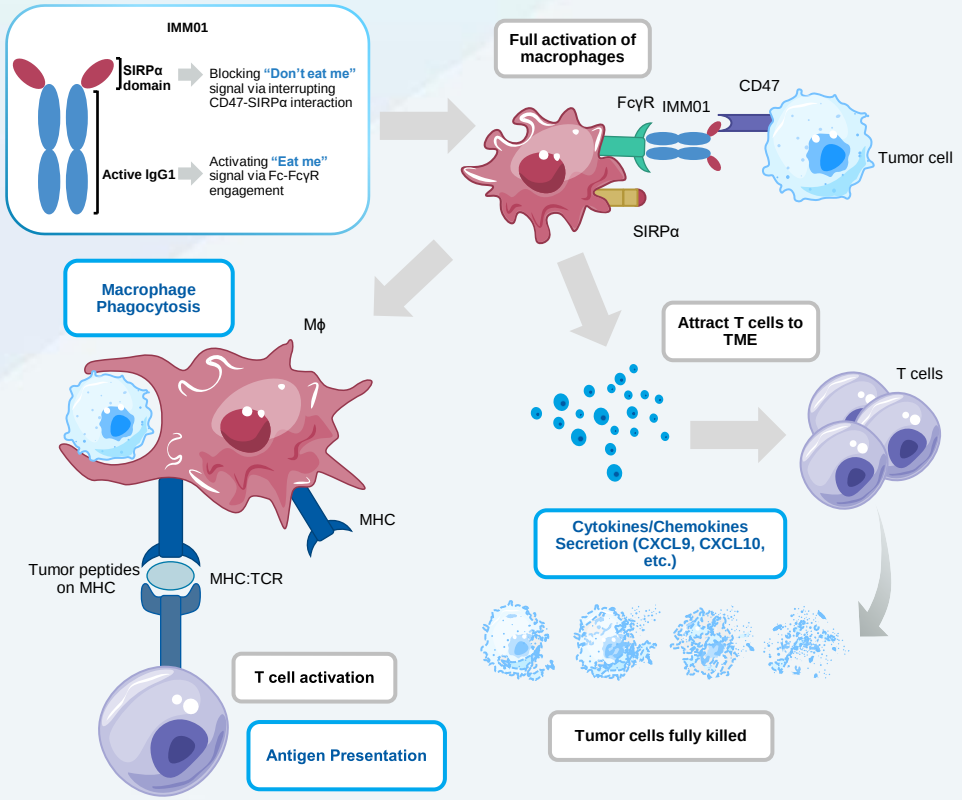
SECTION 2

Major Oncology Programs



IMM01(Timdarpacept)

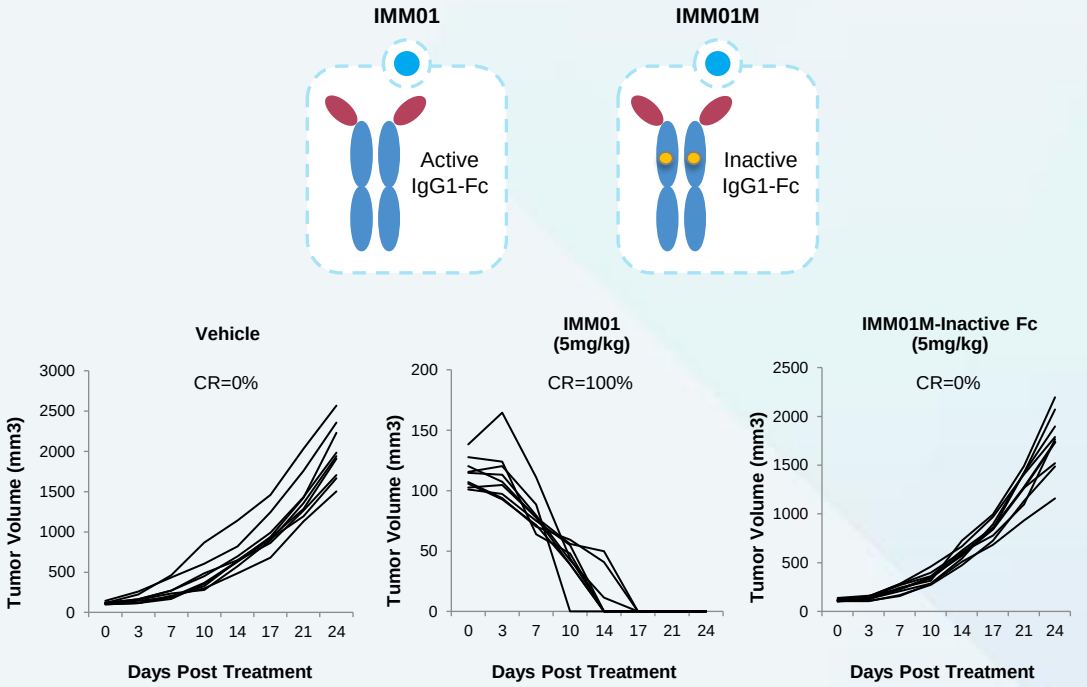
Overview and Competitive Advantage of IMM01(Timdarpacept)



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.

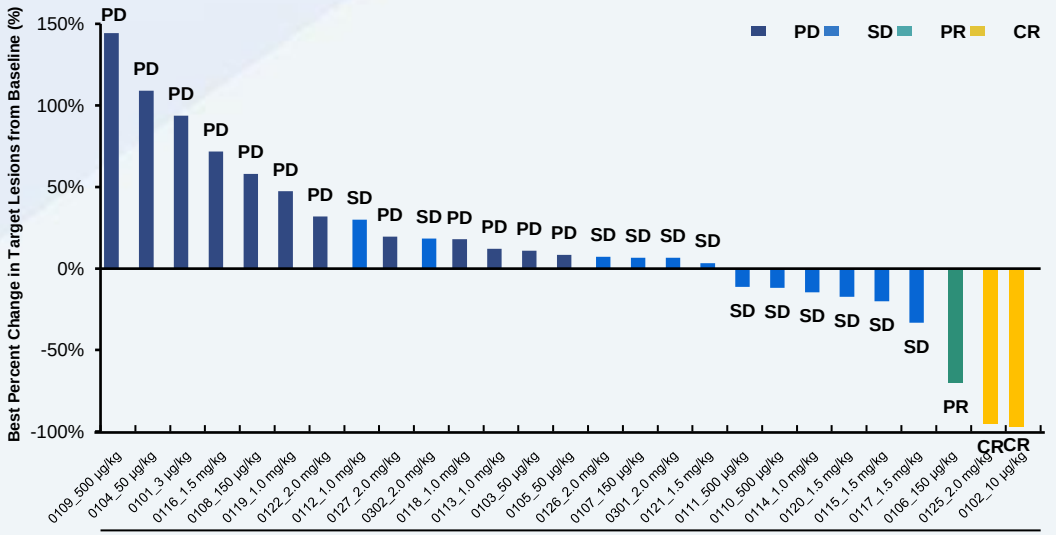
IMM01(Timdarpacept)

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**

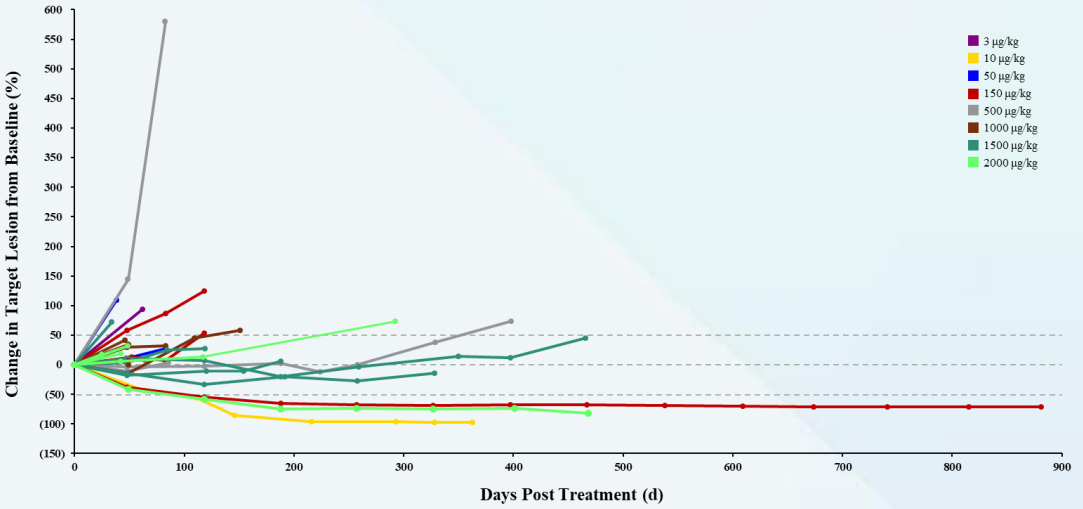
Potent Antitumor Activity and Encouraging Preliminary Clinical Efficacy

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

IMM01(Timdarpacept)

Phase I 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)**

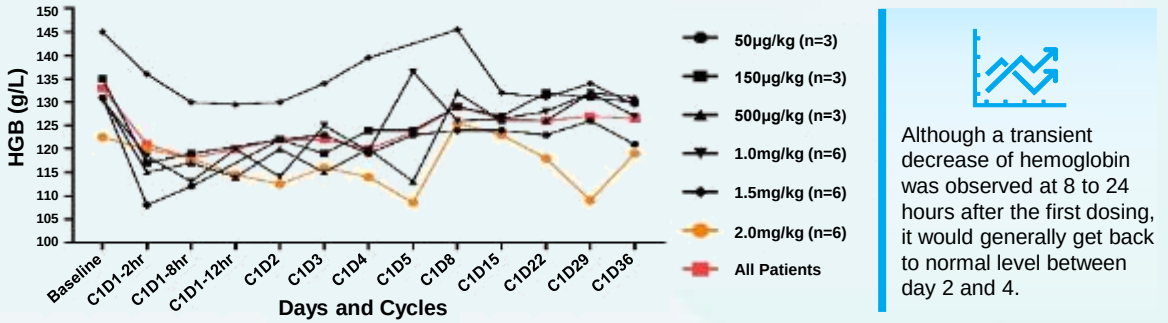
Treatment-related adverse event (n=29)	ALL n (%)	≥Gr 3 n (%)
Positive of Anti erythrocyte antibody	17 (59)	
Leukopenia	16 (55)	2 (7)
Hemolysis	15 (52)	
Infusion related reaction	15 (52)	
Thrombocytopenia	13 (45)	3 (10)
Hypertriglyceridemia	13 (45)	
Anemia	13 (45)	4 (14)
Neutropenia	12 (41)	1 (3)
Neutrocytosis	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.

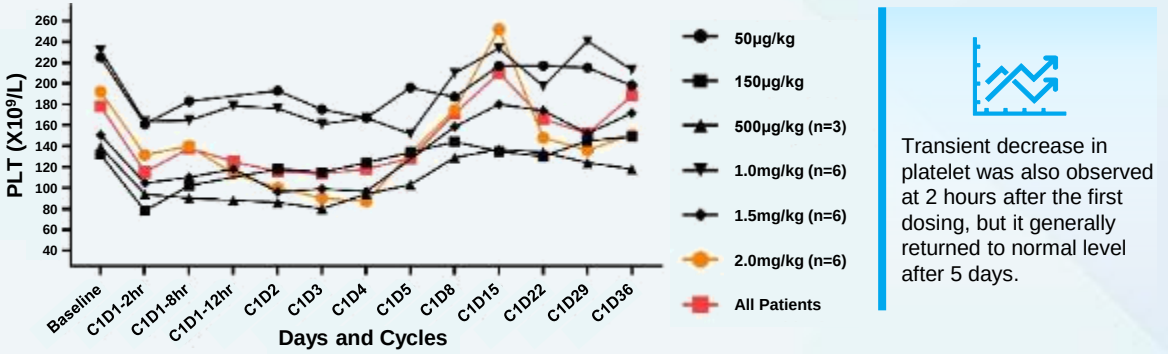
HGB Changes Following Single-dose and Cycle 1 by Cohort



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



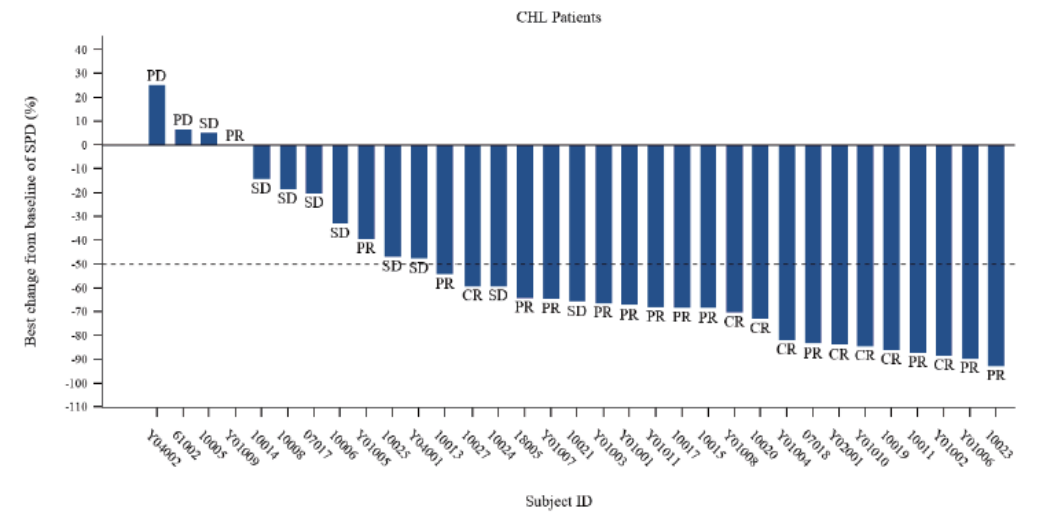
Transient decrease in platelet was also observed at 2 hours after the first dosing, but it generally returned to normal level after 5 days.

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

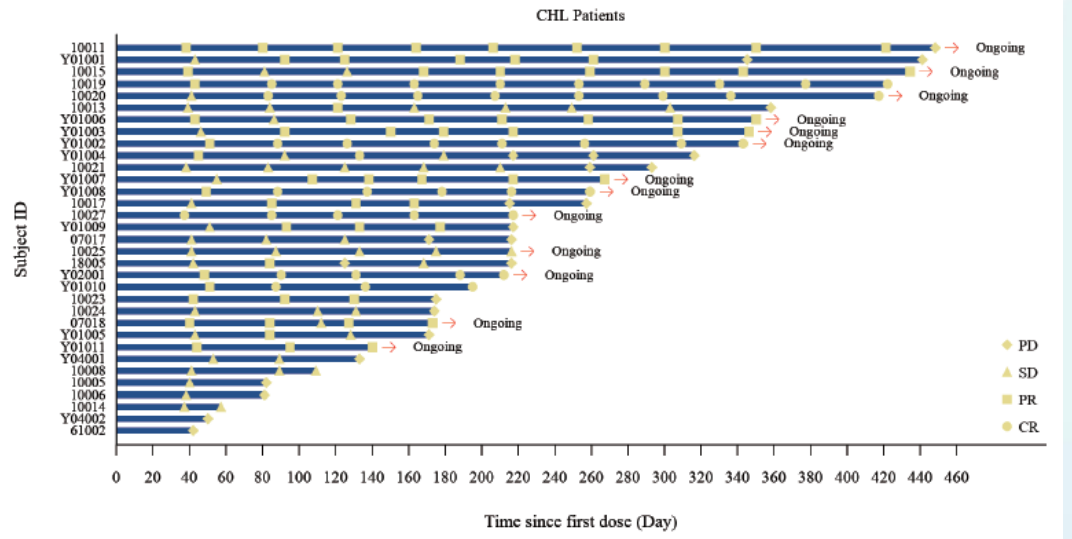
IMM01 (Timdarpacept) + Tislelizumab (PD-1 mAb)

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

Best Percentage Change from Baseline in Target Lesion



Duration of Treatment and Response



Received approval from the NMPA for the protocol of the Phase III clinical trial of IMM01 in combination with tislelizumab versus physician's choice chemotherapy in prior PD-(L) 1-refractory cHL in April 2024. FPI reached on July 1, 2024

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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 June 30th , 2024

IMM01 (Timdarpacept) + Tislelizumab (PD-1 mAb)

Phase II: Superior Efficacy in Anti-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 started in Jul 2024 to treat PD-(L)1 refractory cHL	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

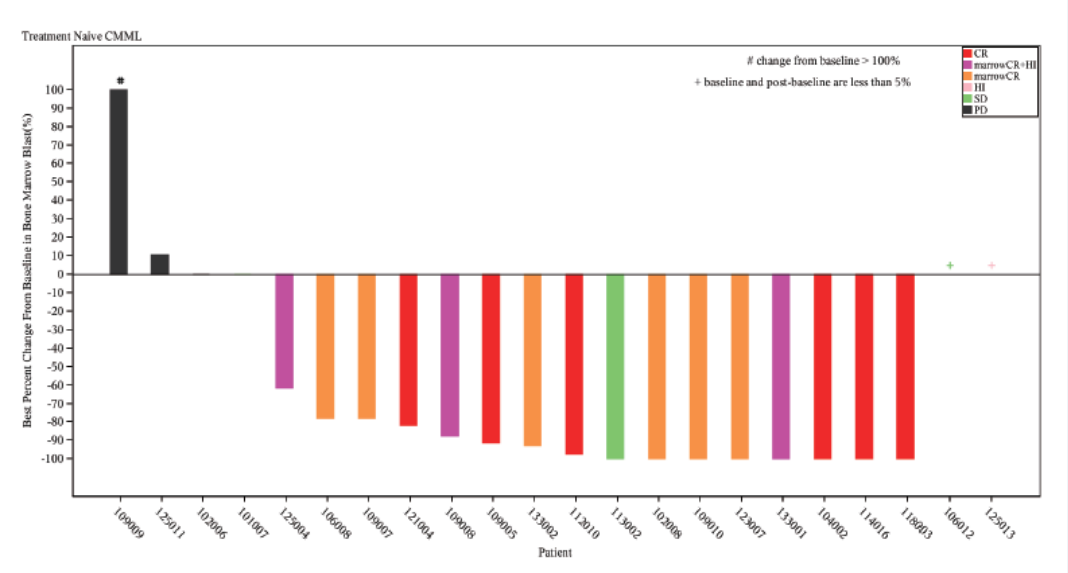
Source:

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

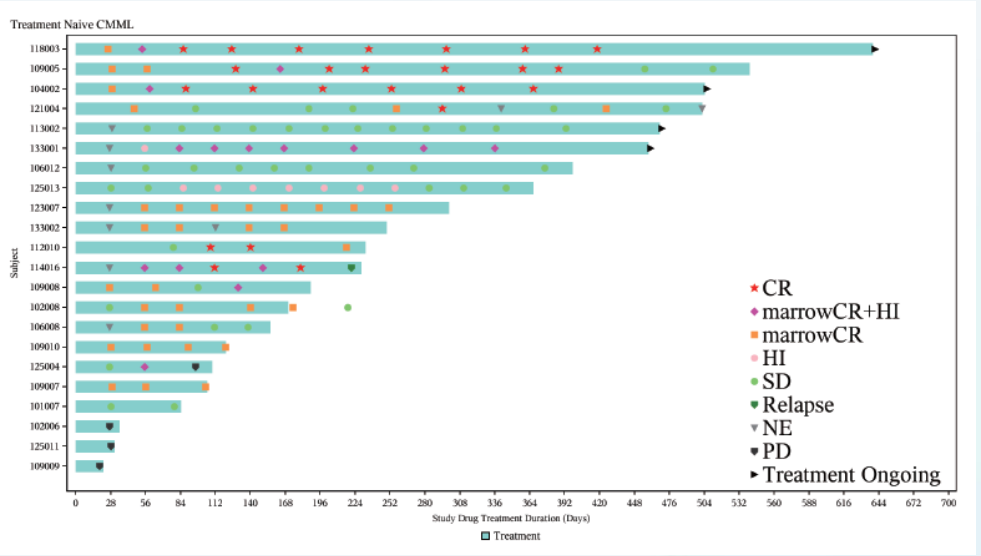
Company Data; The clinical data is as of June 30th , 2024

IMM01 (Timdarpacept) + Azacitidine in 1L CMML (Phase II)

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



Duration of Treatment and Response



Phase III study of IMM01 (Timdarpacept) in combination with azacitidine in patients with newly diagnosed CMML was approved by NMPA in June 2024

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%)

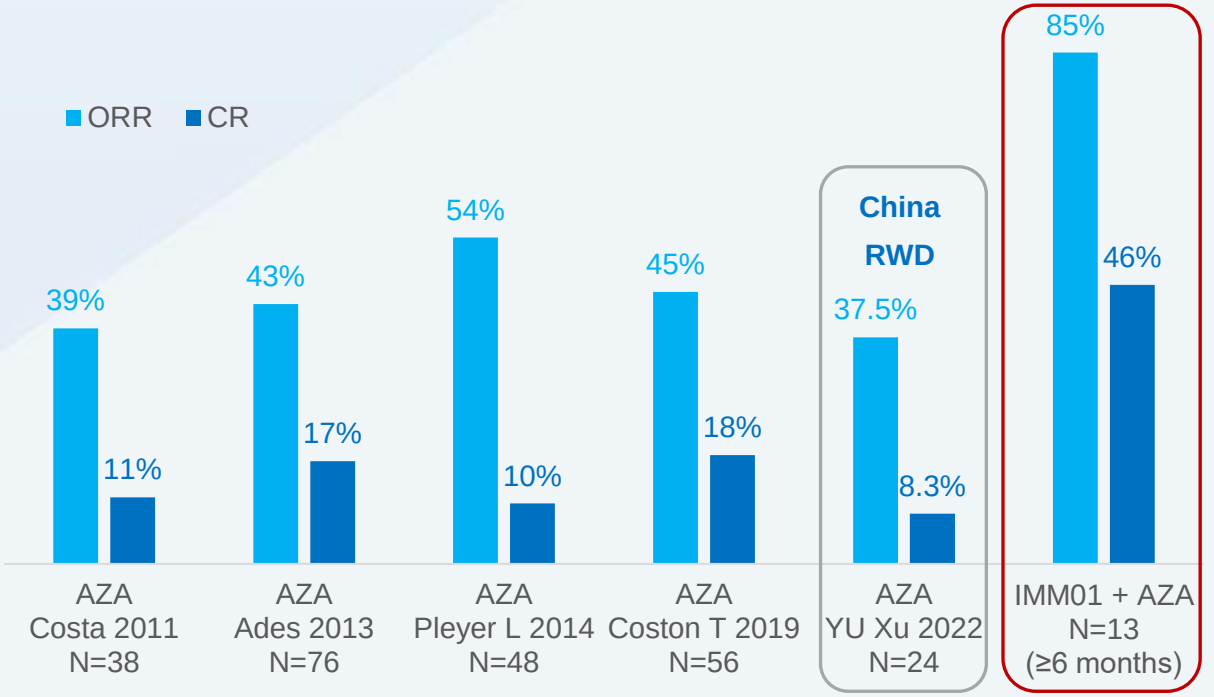
Notes: ORR = Overall Response Rate, CR = Complete Response, mCR = Marrow Complete Response, HI = Hematological Improvement

Source: Company Data; The clinical data is as of June 30th, 2024

IMM01 (Timdarpacept) + Azacitidine in 1L CMML

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%**.

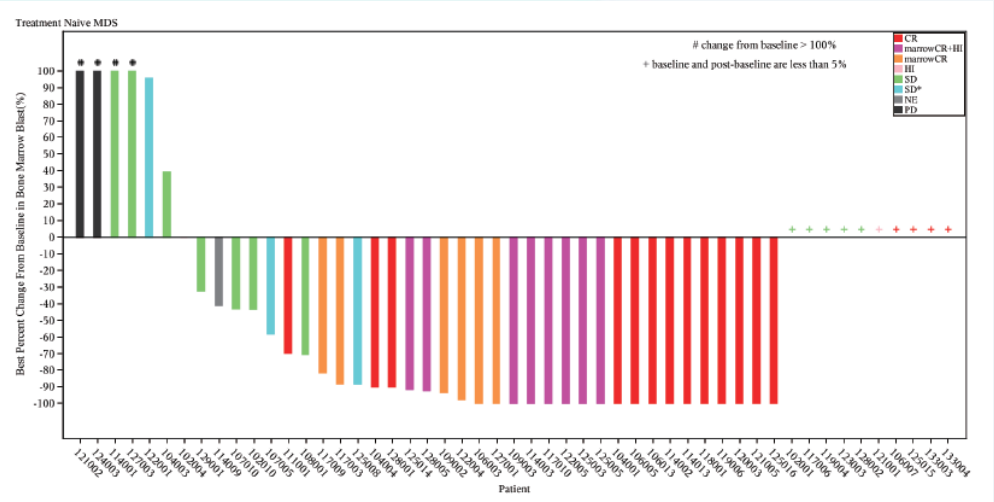
Notes:

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.

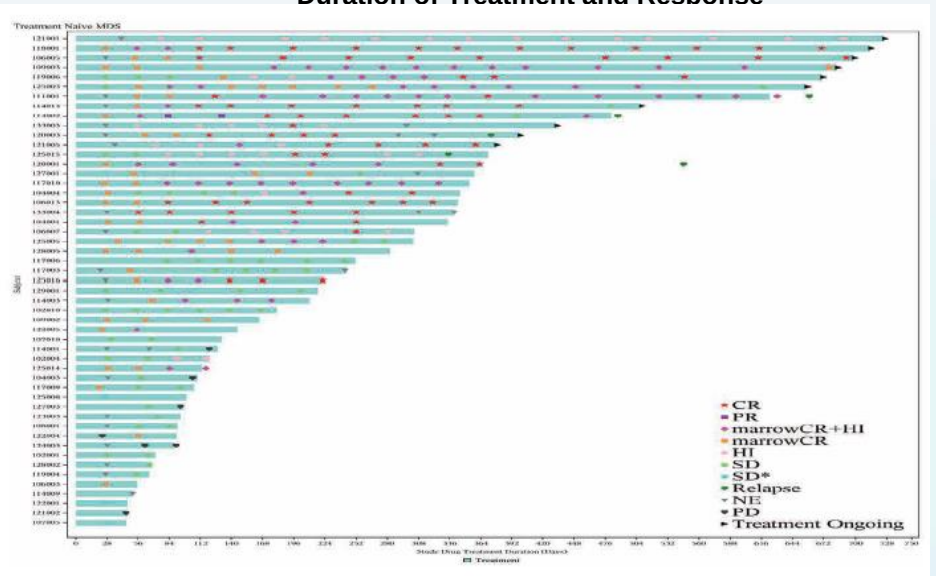
Source: Literature Review; Company Data, the clinical data is as of June 30th, 2024

IMM01 (Timdarpacept) + Azacitidine in 1L MDS (Phase II)

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



Duration of Treatment and Response



Phase III study of IMM01 in combination with azacitidine in patients with newly diagnosed higher-risk MDS was approved by NMPA in May 2024

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Best Overall Response n (%)	1L MDS (N=51)	≥4 months (N=34)	≥6 months (N=29)
ORR	**33 (64.7%)**	**29 (85.3%)**	**26 (89.7%)**
DCR	**45 (88.2%)**	**34 (100%)**	**29 (100%)**
CR	**17 (33.3%)**	**17 (50.0%)**	**17 (58.6%)**
mCR+HI	8 (15.7%)	7 (20.6%)	6 (20.7%)
mCR alone	6 (11.8%)	3 (8.8%)	2 (6.9%)
HI	2 (3.9%)	2 (5.9%)	1 (3.4%)
SD	12 (23.5%)	5 (14.7%)	3 (10.3%)

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 June 30th, 2024

IMM01 (Timdarpacept) + 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.

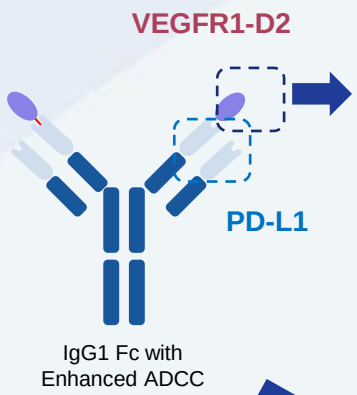
Source: Valeria Santini1 et al, Eur J Haematol. 2010 August ; 85(2): 130–138.
David et al. A.J Clin Oncol ,2023,41:2815-2826;
Company Data; The clinical data is as of June 30th, 2024

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



VEGF

VEGF receptor trap binds multiple VEGF receptor ligands beyond VEGF-A for broader antiangiogenic activity

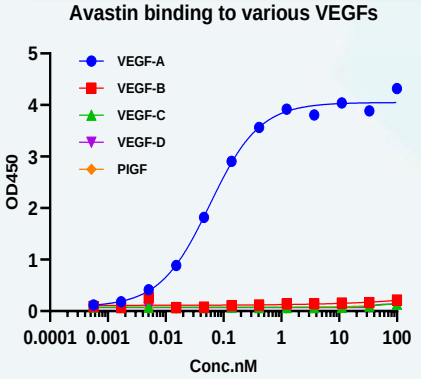
PD-L1

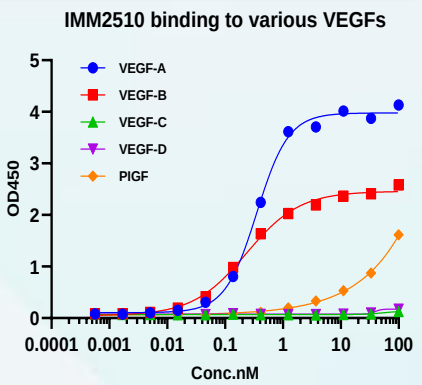
Tumor cells expressing PD-L1 can **bind to PD-1** on the surface of T cells to evade T-cell attacks

ADCC

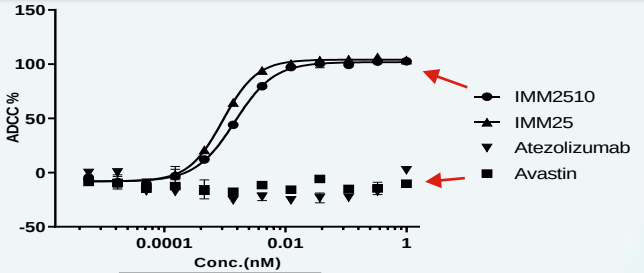
ADCC-enhanced antibody designed to induce direct killing of immunesuppressive PD-L1+ tumor cells

IMM2510 binds multiple VEGF receptor ligands





IMM2510 has enhanced ADCC activity



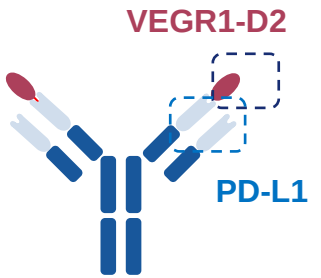
	EC50
IMM2510	0.001461
IMM25	0.0009247

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

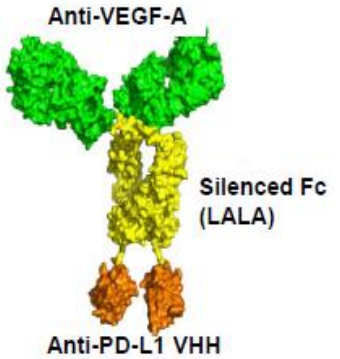
IMM2510 (VEGF × PD-L1)

Key Competitor Landscape

	IMM2510	PM8002	AK112
VEGF binding	VEGF-A, VEGF-B, PLGF	VEGF-A	VEGF-A
PD-1 or PD-L1	PD-L1	PD-L1	PD-1
ADCC	Enhanced ADCC	None	None
Key clinical data	Multiple responses in patients with prior PD-1 treatment in Phase 1a trial	1L NSCLC: 47% ORR 1L TNBC: 79% ORR 2L SCLC: 61% ORR	Superiority over Keytruda® in 1L NSCLC Approved in 2L EGFRm NSCLC



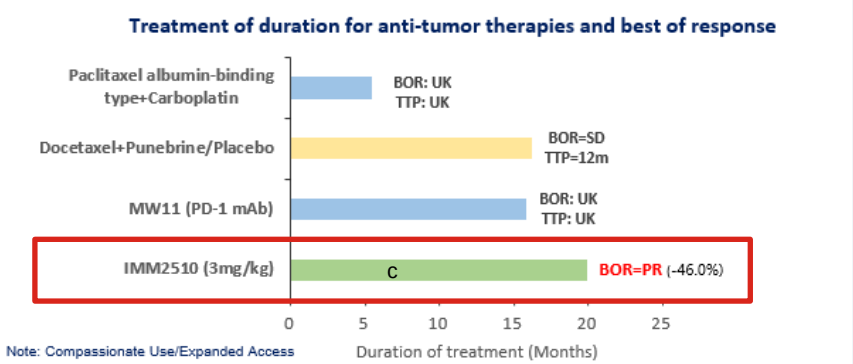
IgG1 Fc with Enhanced ADCC



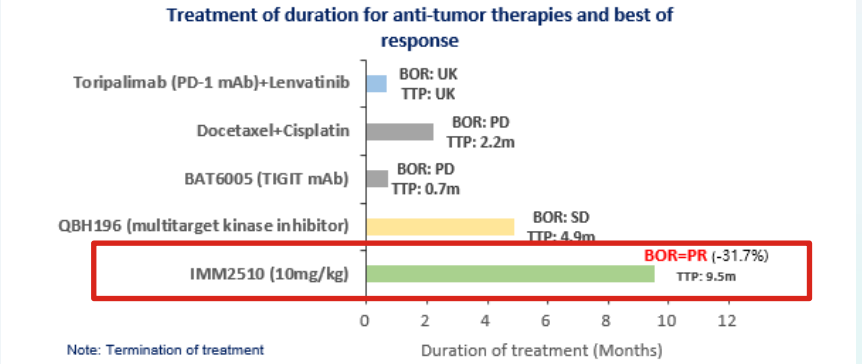
IMM2510 (VEGF × PD-L1)

IMM2510 achieved multiple responses in patients with prior PD-1 inhibitor during dose escalation

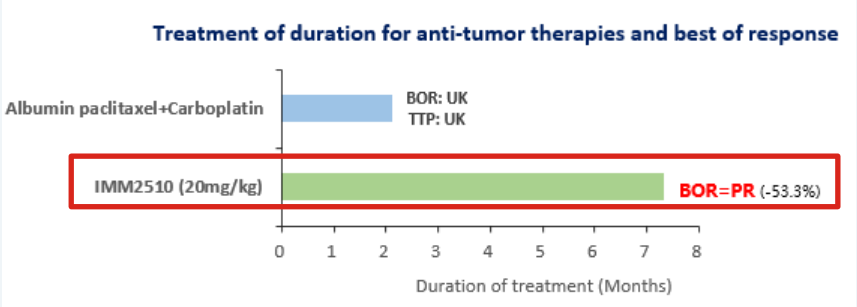
Squamous NSCLC



Squamous NSCLC



Thymus adenosquamous carcinoma



completed dose escalation up to 20 mg/kg Q2W with a manageable safety profile and no observed dose - limiting toxicities (DLT)

IMM2510 (VEGF × PD-L1)

Global Collaboration

On August 1, 2024, we have reached a license and collaboration agreement with SynBioTx Inc., a wholly-owned subsidiary of Instil Bio, Inc. (NASDAQ:TIL) pursuant to which SynBioTx will in-license the global rights (outside the Greater China region) to our proprietary PD-L1xVEGF bispecific molecule IMM2510, as well as our next-generation anti-CTLA-4 antibody (ADCC+) IMM27M.

We will receive an upfront payment and potential near-term payments of up to **US$50 million** as well as potential additional development, regulatory, and commercial milestones payments of up to **US$2.1 billion,** plus single digit to low double-digit percentage royalties on global (outside the Greater China region) net sales.

Developing One Owned Patent Family

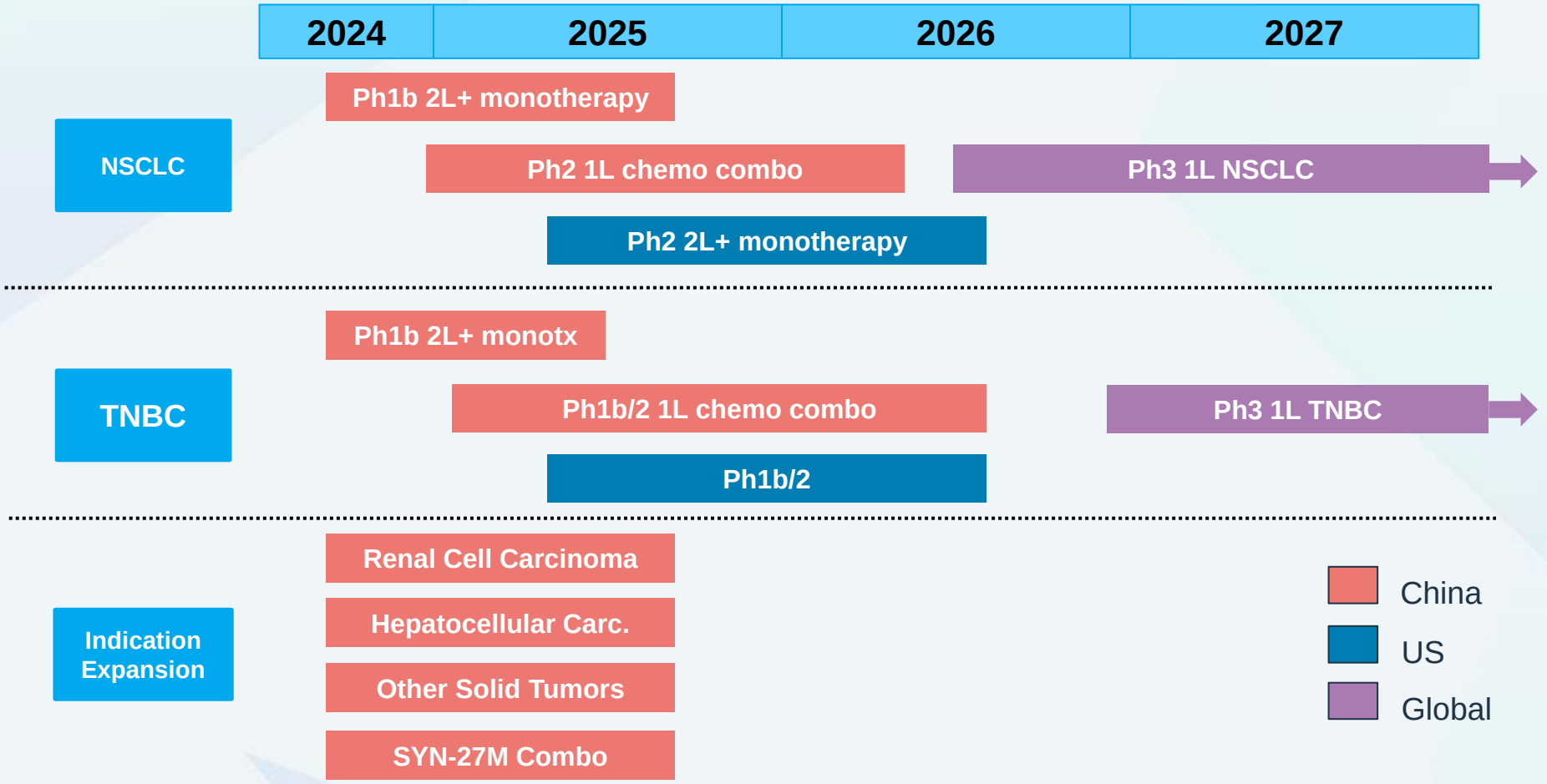
- *1* issued patent in each of the U.S. and Japan;
  1 issued patent in the PRC
- *1* pending patent application in each of Europe and the U.S.

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 (VEGF × PD-L1)

Potential Global IMM2510/ SYN-2510 Development



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:

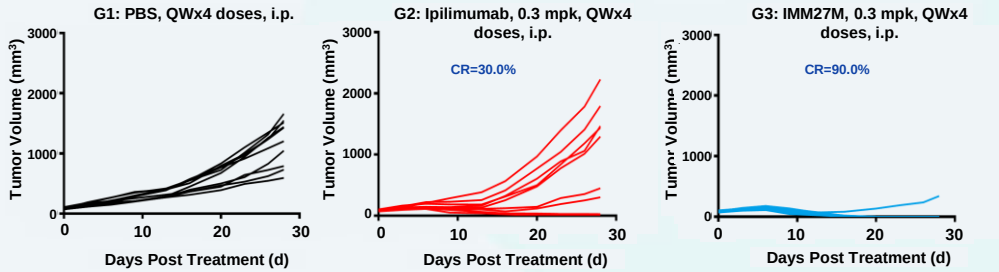
Limited efficacy → High dosage to achieve desirable efficacy → **Serious safety issues**

Clinical Development Plan

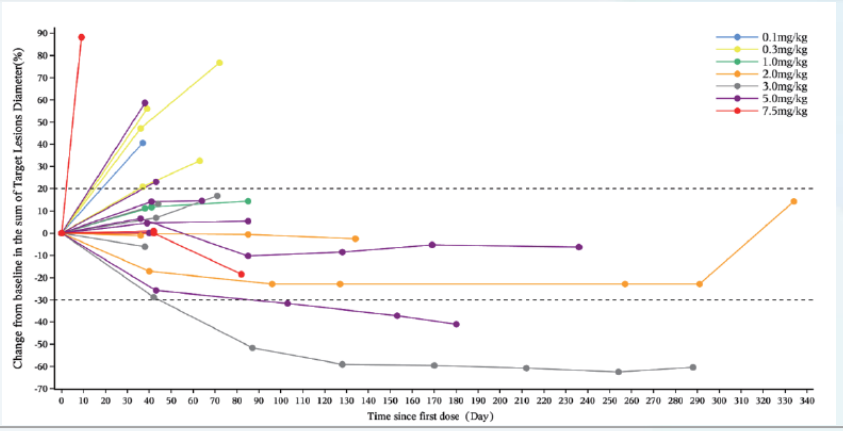
Jun 2022	Mar 2023	Sept 2023	July 2024
Phase I mono	IMM27M+PD-1 (advanced solid tumors) IND approval for Ph Ib/II	Phase I mono completed and confirmed RP2D	IMM2510 + IMM27M FPI

Stronger In Vivo Antitumor Effects and Preliminary Ph1 data

Efficacy Study in Colon Cancer (MC38) Syngeneic Mouse Model



Duration of Treatment and Best Response in Phase I



Preliminary efficacy: **2 confirmed PR and 3 SD** with tumor shrinkage

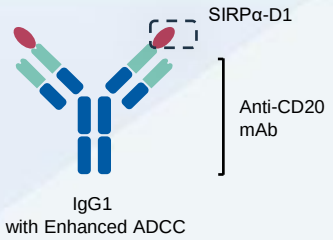
Source: Company Data; The clinical data is as of June 30th, 2024

IMM0306 (CD47×CD20)

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

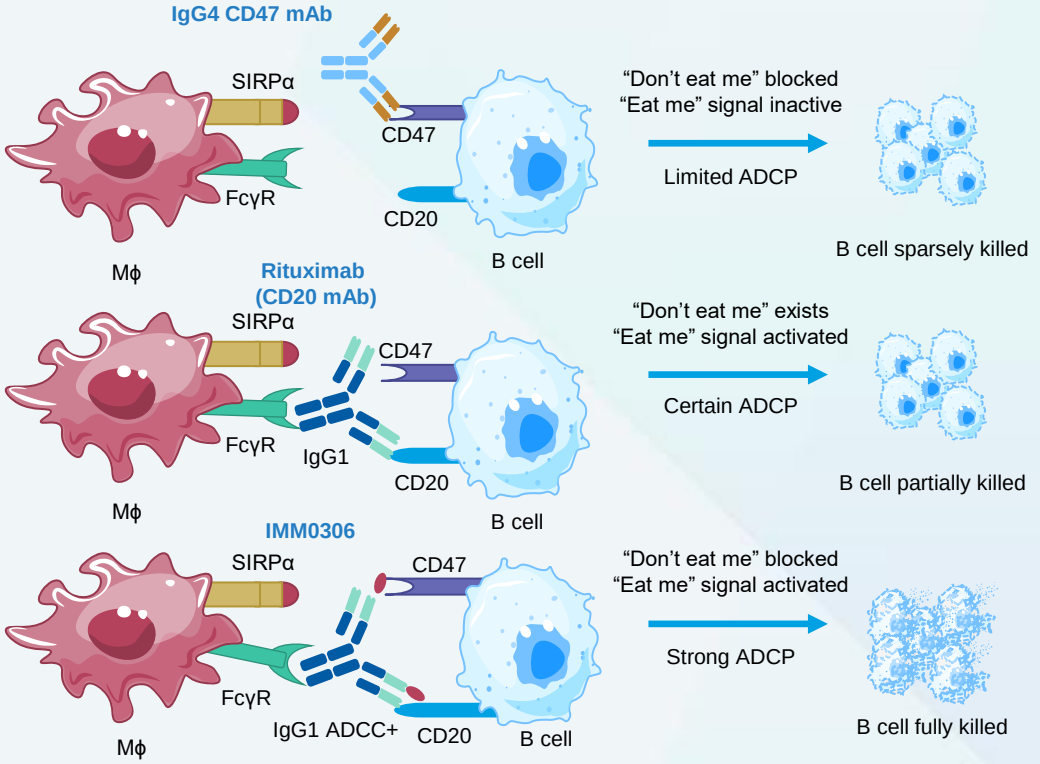
Overview

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**

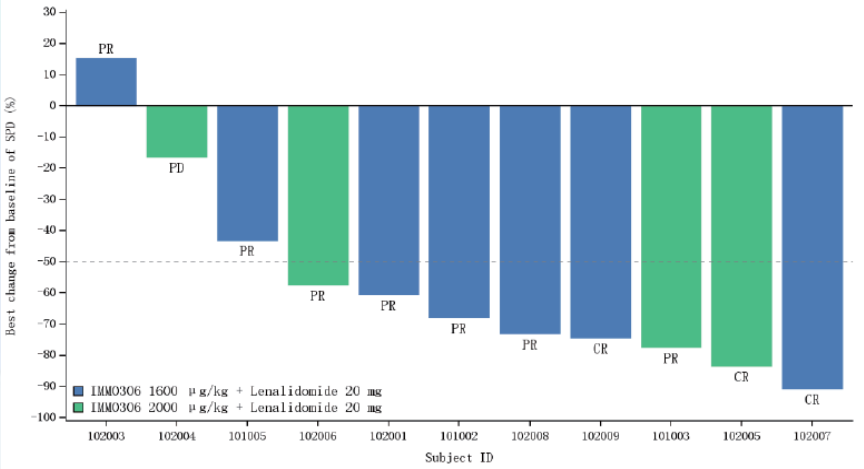
2 CD47×CD20 bispecific antibodies/fusion proteins under development globally
Among them, IMM0306 is the **1st** to enter into a clinical trial

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

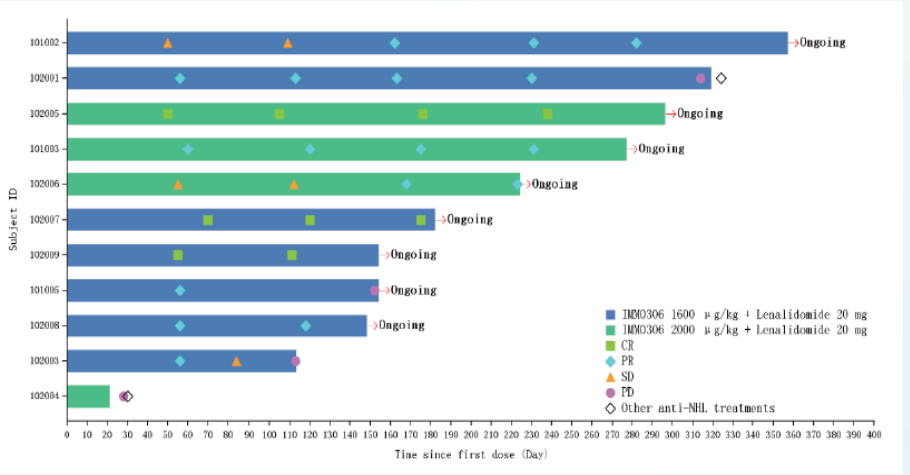
IMM0306 + Lenalidomide (CD47×CD20)

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

Best Percentage Change from Baseline in Target Lesion in Phase Ib



Duration of Treatment and Response in Phase Ib



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.

IMM0306 + Lenalidomide Phase Ib

Best Overall Response n (%)	Efficacy Evaluable (N=11)
ORR	10 (90.9)
CR	3 (27.3)
PR	7 (63.6)
PD	1 (9.1)

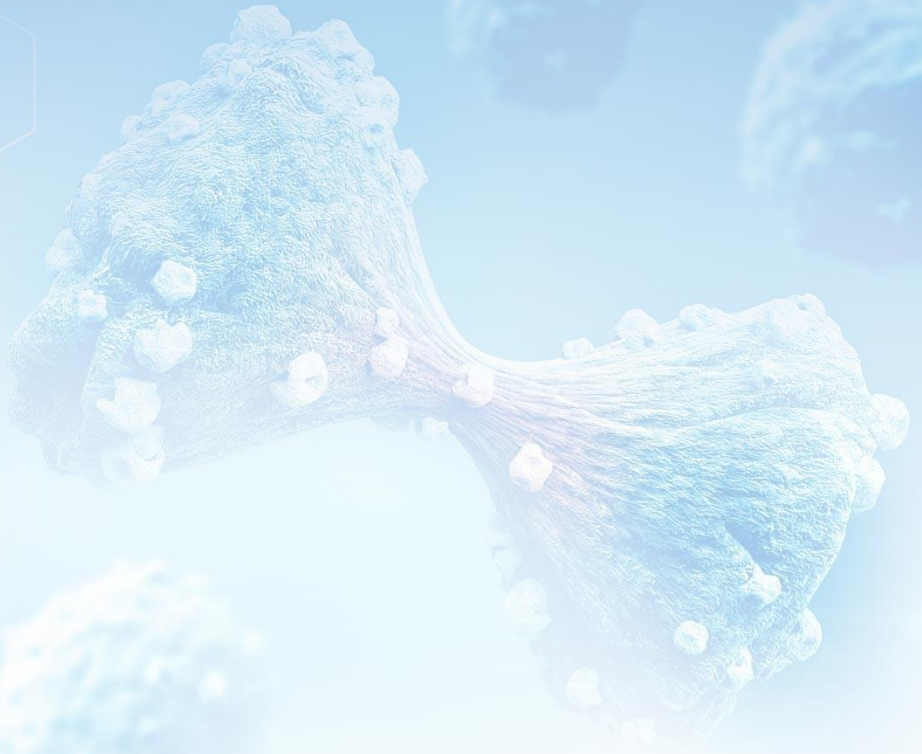
Note: among 6 efficacy-evaluable R/R FL patients in the Phase **IIa trial,** 4 CR and 2 PR were assessed by investigators by Mid July. **The ORR and CRR were 100% and 66.7%,** respectively

Source: Company Data; The clinical data is as of June 30th, 2024

宜明昂科
ImmuneOnco

SECTION 3

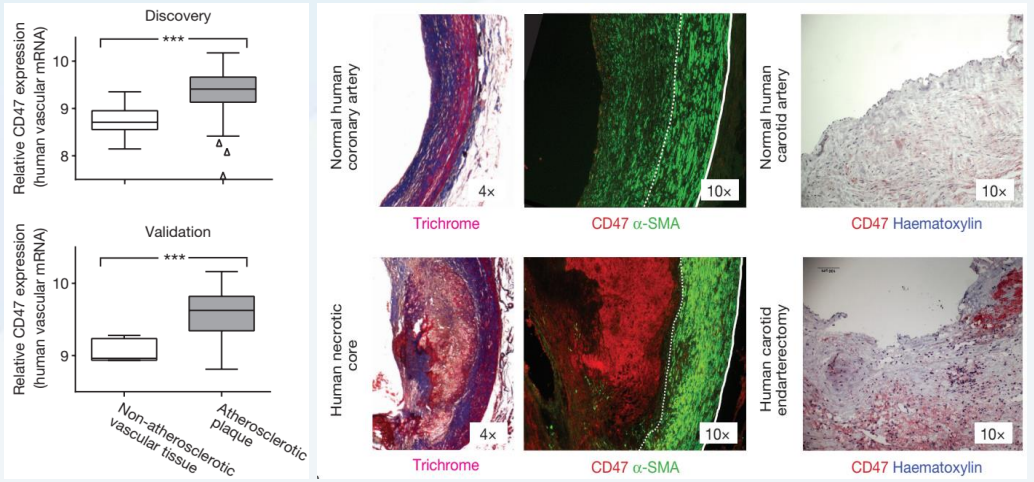
Non-Oncology Programs



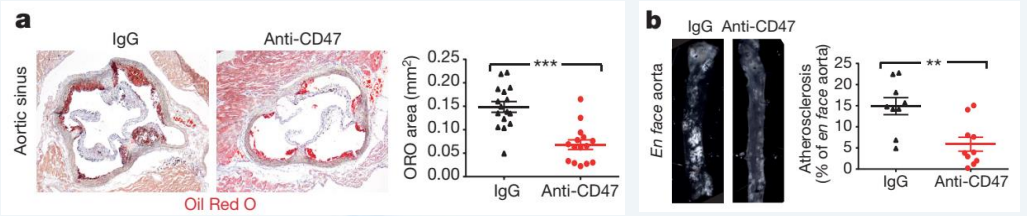
IMC-001 (IMM01, SIRPα-Fc)

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

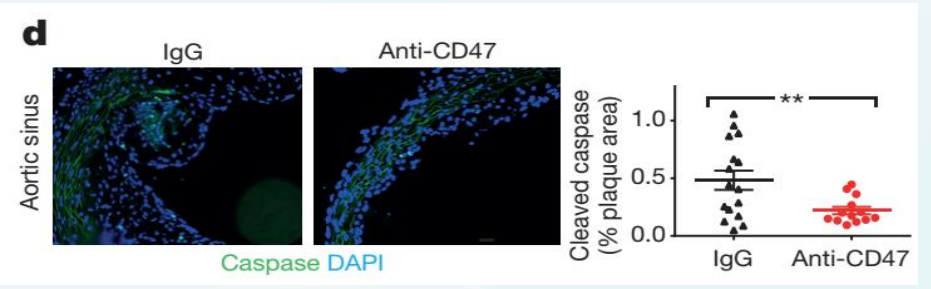
CD47 is highly expressed in human atherosclerotic plaque

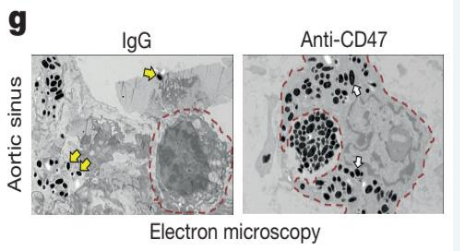


Shrinkage of atherosclerotic plaque was observed in rat model by blocking the CD47/SIRPα signaling pathway



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





BITTERROOTBIO

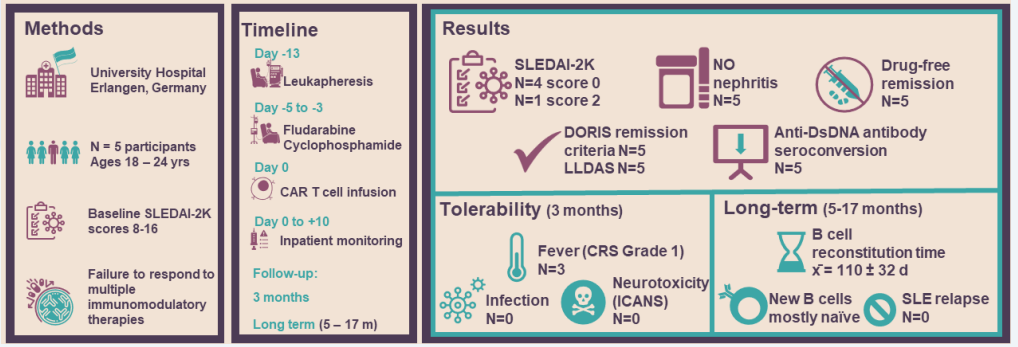
- Bitterroot Bio has one CD47/ SIRRPα that was developed for atherosclerosis
- Bitterroot Bio announced the completion of $145 million round A funding in June 2023
- It was co-founded by Irv Weissmanv, Nick Leeper, John C. Martin and Lou Lange

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.

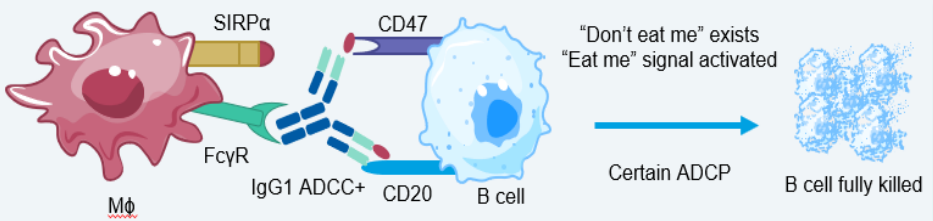


RITUXAN® (rituximab, CD20) was approved by FDA in 2006 for the treatment of **rheumatoid arthritis (RA)**;

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

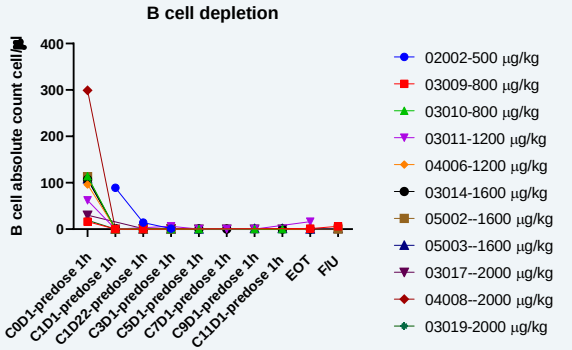
B-cell depletion therapies (BCDTs) are widely used in the treatments of autoimmune diseases.

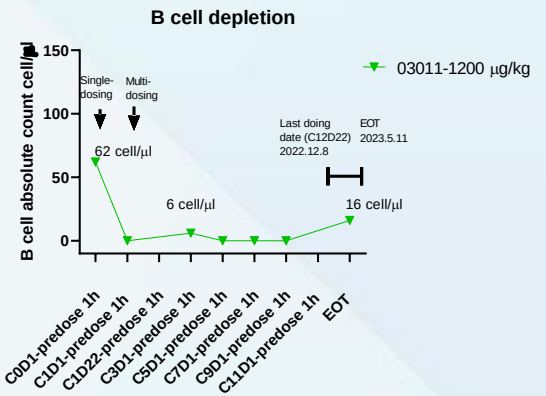
Mechanism of Action



Peripheral B cell depletion by IMM0306 was observed in a wide range of clinical dose

Peripheral B cell depletion by IMM0306 was robust and sustained until three months after treatment withdraw



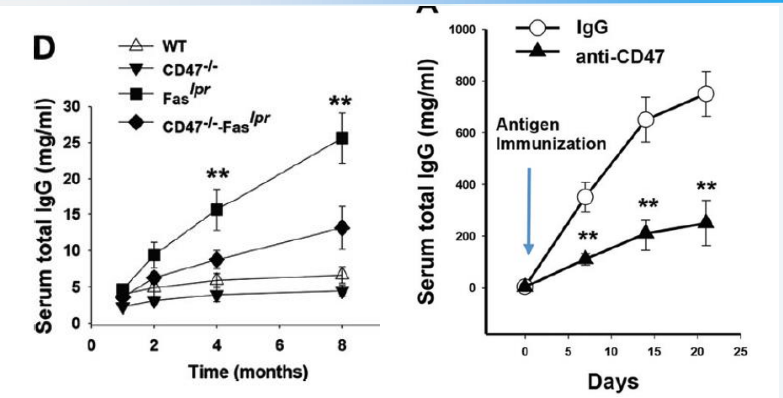


Reference: Dimitrios Mougiakakos, et al., August 5, 2021, N Engl J Med 2021; 385:567-569; Andreas Mackensen, et al. NATURE MEDICINE, VOL 28, Oct. 2022, 2124–2132

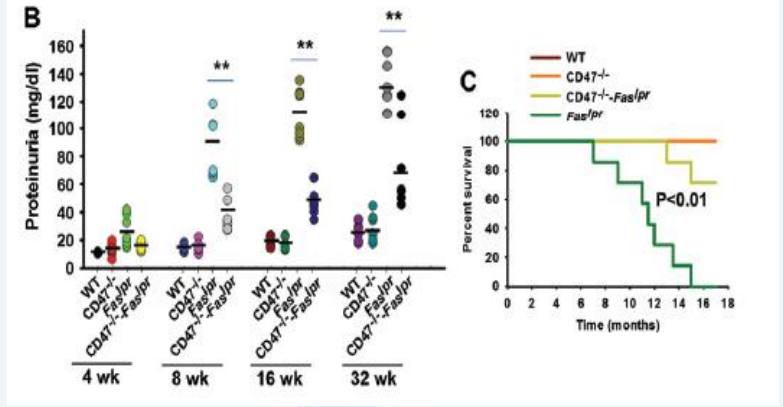
IMC-002 (IMM0306, CD47xCD20/mAb-Trap)

CD47 contributes to pathogenesis of lupus nephritis

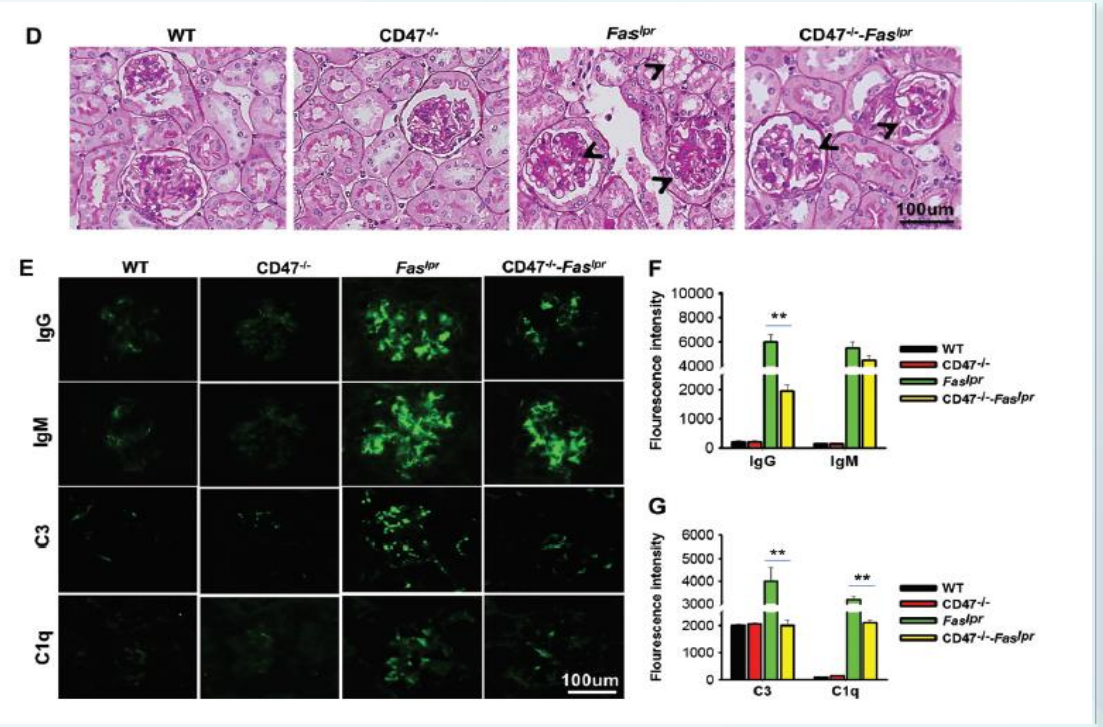
Knockout of CD47 reduces IgG levels and improves lupus nephritis



CD47 knockout in Faslpr LN disease model resulted in decreased proteinuria levels and prolonged survival



CD47 knockout in Faslpr LN disease model resulted in remission of glomerular base membrane thickening and significantly reduced autoantibodies and complement deposition



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

ActRII biology in reducing fat mass while preserving muscle mass

ActRII biology in adipose tissue

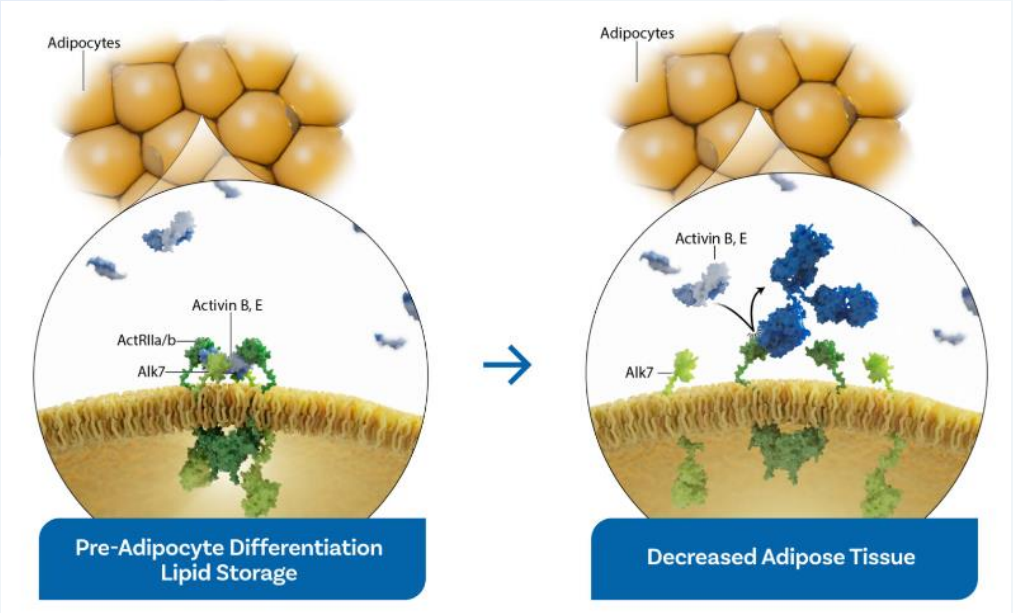
Activin signaling via ActRII receptors directly promotes lipid storage, acting as a key driver of visceral fat accumulation and obesity

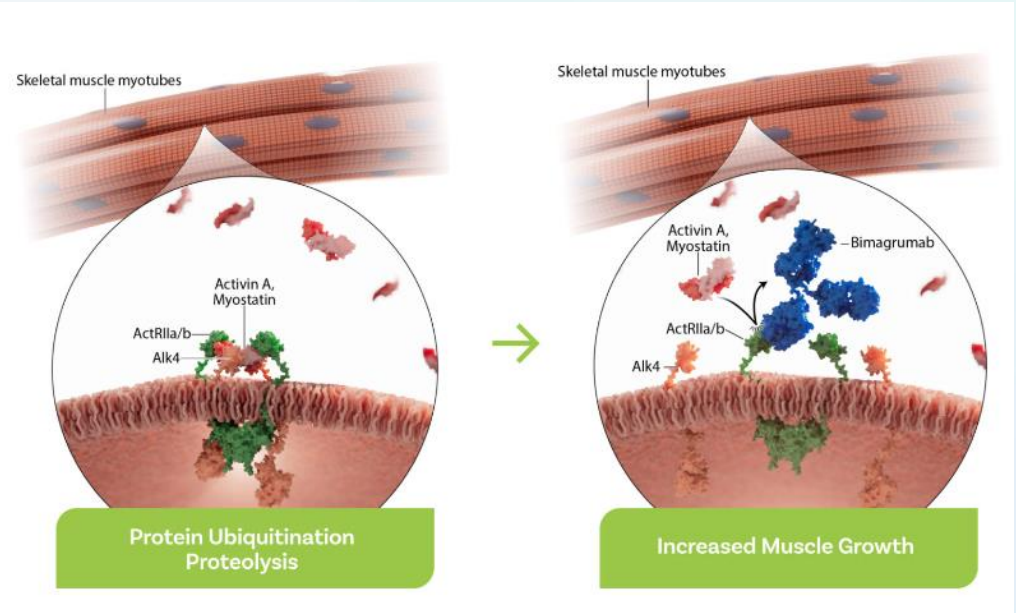
By blocking ActRII signaling in adipose cells, can mobilize and metabolize fat.

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.



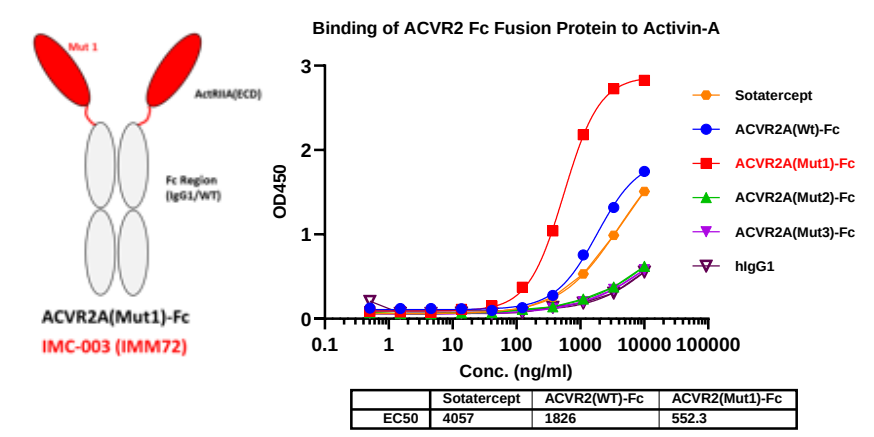


Reference: versanisbio.com

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

Preclinical Results

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

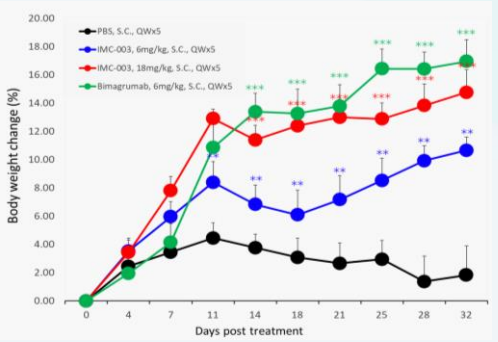


	Sotatercept	ACVR2(WT)-Fc	ACVR2(Mut1)-Fc
EC50	4057	1826	552.3

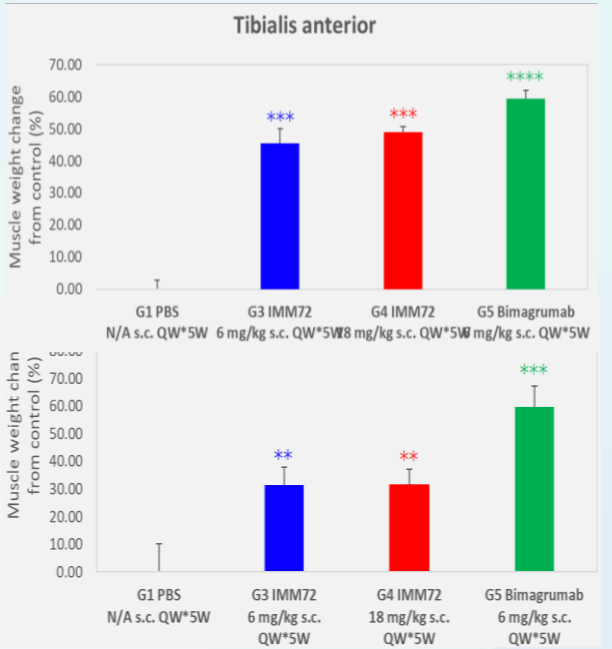
	IMC-003 (IMM72)	Sotatercept
Company	ImmuneOnco	MSD
Structure	ACVR2A-Fc (point mutation)	ACVR2A-Fc
Affinity	Comparable	Comparable
Binding (ELISA)	Stronger (by 7 times)	Medium
Blocking	Stronger	Medium
In vivo efficacy	Stronger	Medium

IMC-003 helps build muscle and lose weight

Body weight increased substantially by IMC-003 treatment



Skeletal muscle increased substantially by IMC-003 treatment



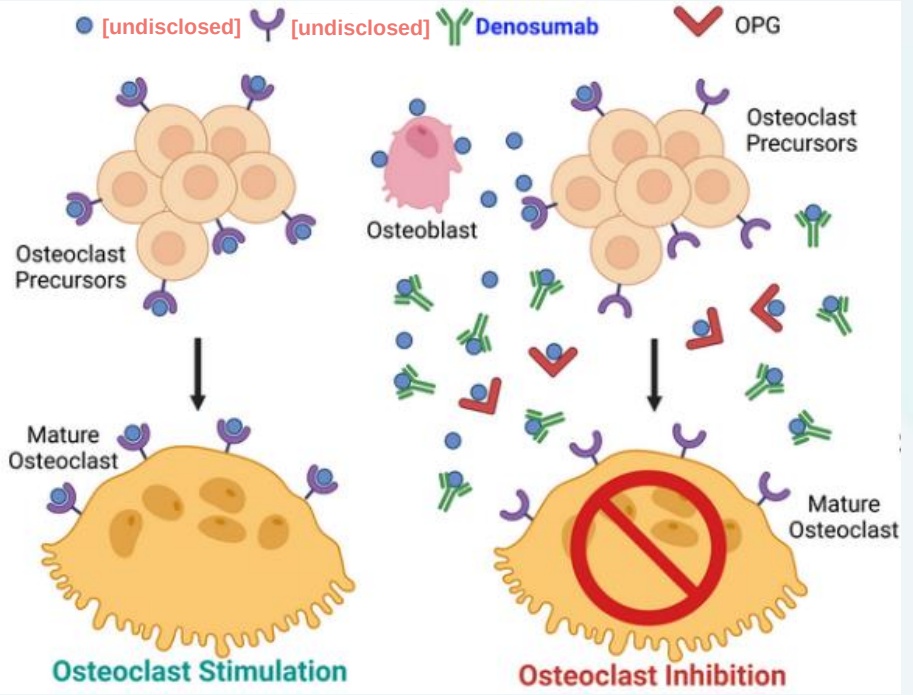
Eli Lilly completed the acquisition of **Versanis Bio in up to $1.925 billion cash** in August 2023.

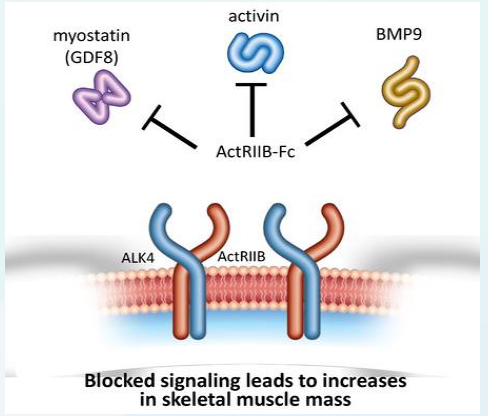
Versanis' lead asset, bimagromab, which was being assessed in a phase IIb study alone and in combination with semaglutide in adults living with overweight or obesity.

IMC-004 (IMM7211c, [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**.



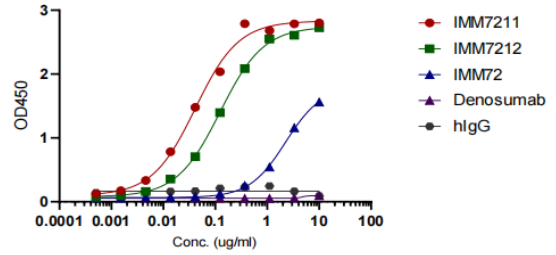


IMC-004 (IMM7211c, [undisclosed] x ActRIIA)

Preclinical Results

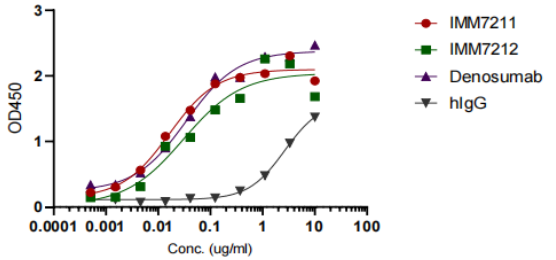
IMC-004 (IMM7211) has stronger binding and blocking capacity than IMM7212 on Activin A and [non-disclosed]; and is similar to Denosumab on [non-disclosed]

20230629 Binding of IMM7211 to Activin A by ELISA



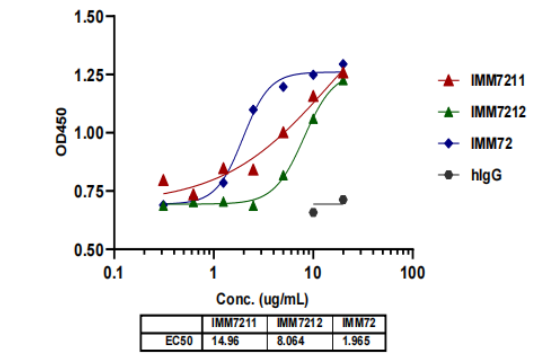
	EC50
IMM7211	0.04139
IMM7212	0.1245
IMM72	2.260

20230629 Binding of IMM7211 to [undisclosed] by ELISA



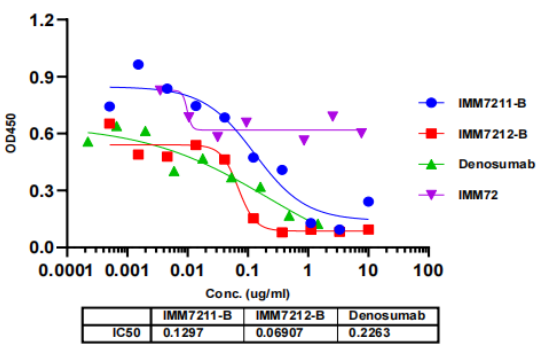
	EC50
IMM7211	0.01644
IMM7212	0.03036
Denosumab	0.03438

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

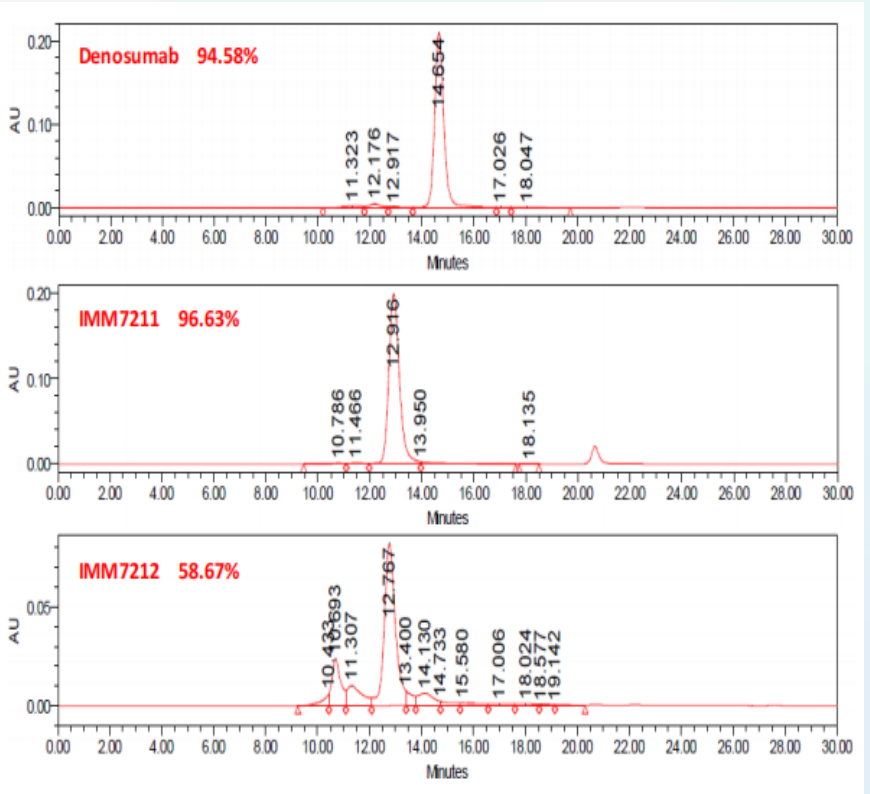


	IMM7211	IMM7212	IMM72
EC50	14.96	8.064	1.965

IMM7211/IMM7212 Blocking the Interaction of [undisclosed]



	IMM7211-B	IMM7212-B	Denosumab
IC50	0.1297	0.06907	0.2263



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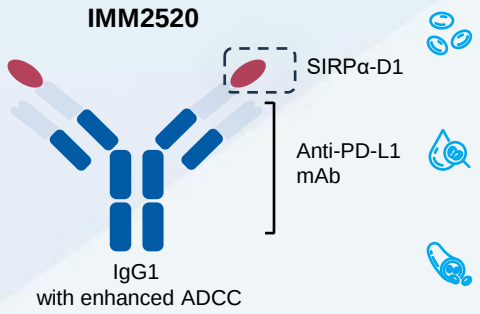
SECTION 4

Other Oncology Programs

IMM2520 (CD47×PD-L1)

Overview



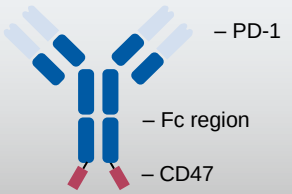
- A CD47 and PD-L1 dual-targeting bispecific molecule for the treatment of solid tumors
- Unique structure to **avoid RBC binding**
- Engineered ADCC-enhanced IgG1 Fc fragment to **fully activate macrophages** and **induce enhanced ADCP and ADCC activity**

Competition Landscape

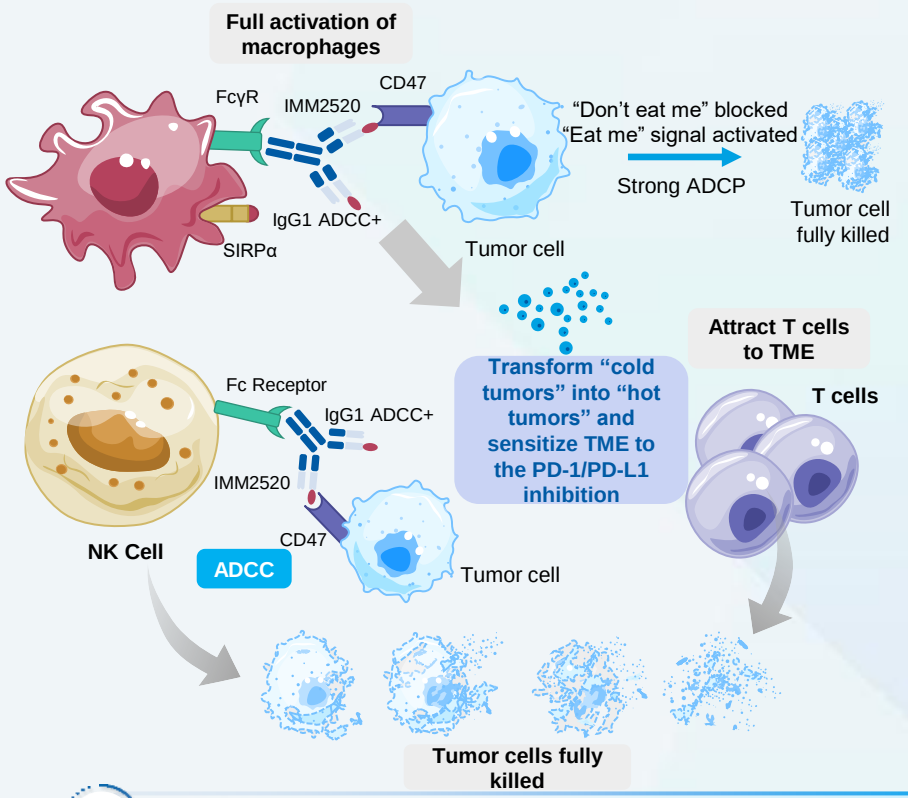
Other 9 CD47 and PD-1/PD-L1 bispecific molecules under clinical development

- ✓ 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

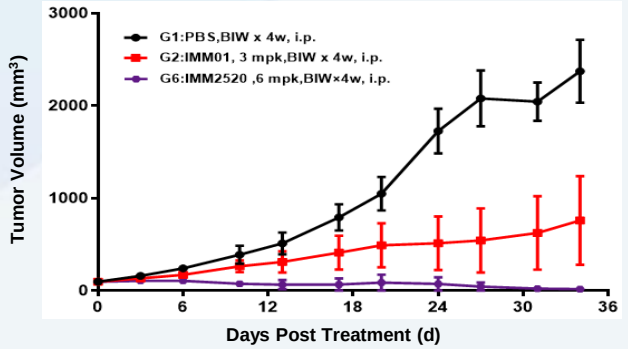


Due to the crosstalk among macrophages, NK cells and T cells, IMM2520 is able to unleash significant synergistic effects

IMM2520 (CD47×PD-L1)

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. Expect to complete this trial in 2024.

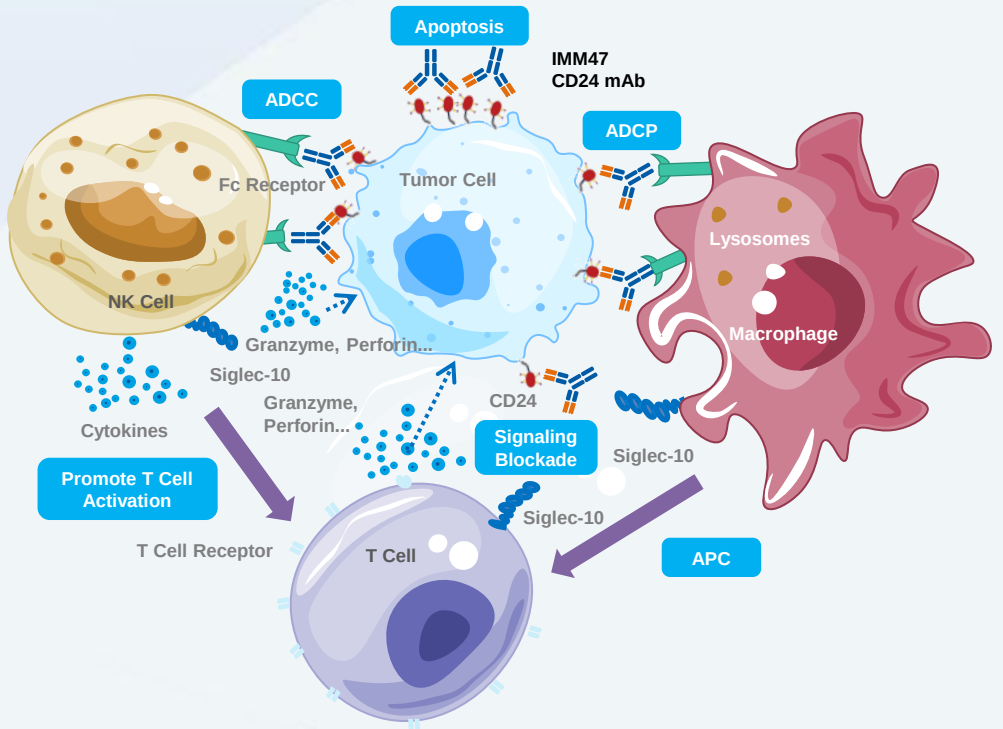
Phase I Preliminary Efficacy

As of June 30, 2024, 24 patients have been enrolled and dosed. The preliminary data has demonstrated that IMM2520 is safe and well tolerated. One PR and two SDs with tumor shrinkage over 10% were achieved.

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)

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

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

3 pending patent application in Europe, Korea and Brazil

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

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APPENDIX：

Our Approach

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, 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

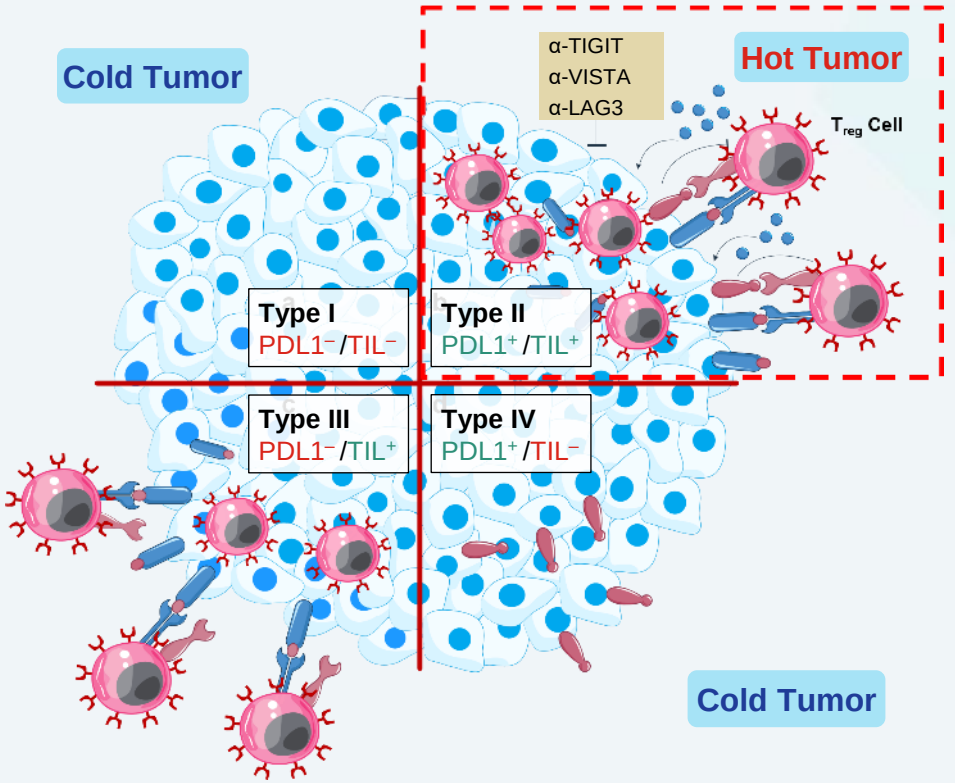
Type I

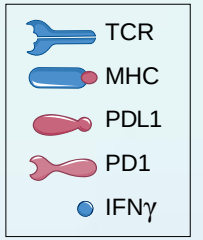
Lack of TILs in TME

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

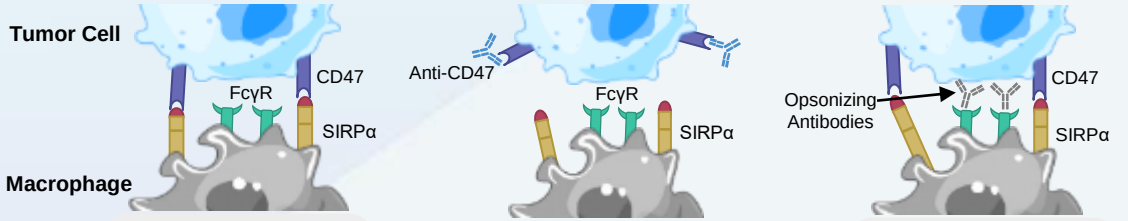
Lack of TILs in TME

Innate immune activation to induce inflammation and attract adaptive immune cells

Source: Frost & Sullivan, literature review

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

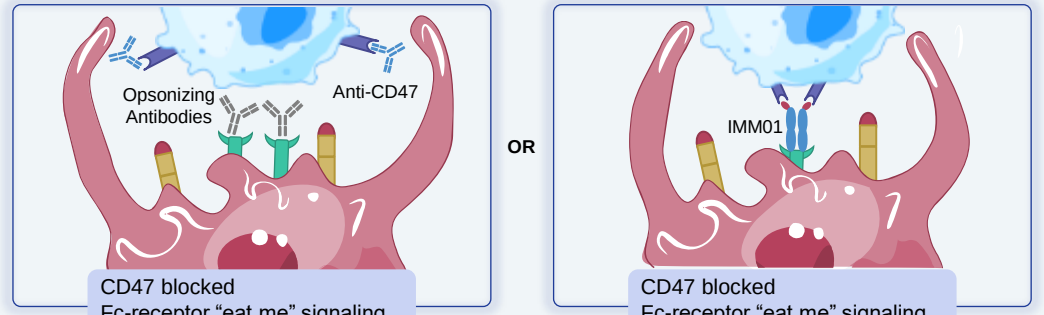
Mechanism of Action in the CD47-SIRPα Signaling Pathway



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



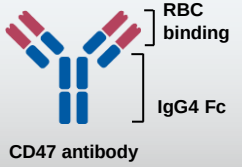
CD47 blocked
Fc-receptor “eat me” signaling
Full macrophage activation

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

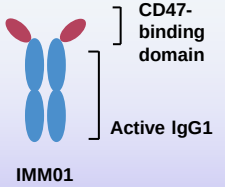
The safety concerns around CD47 pose considerable challenge



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

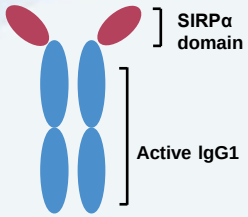


Engineered CD47-binding domain with **no RBC binding** *in vitro;* Modification of deglycosylation of the binding domain **mitigates immunogenicity**

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

Overview and Competitive Advantage of IMM01 (Timdarpacept)

Overview

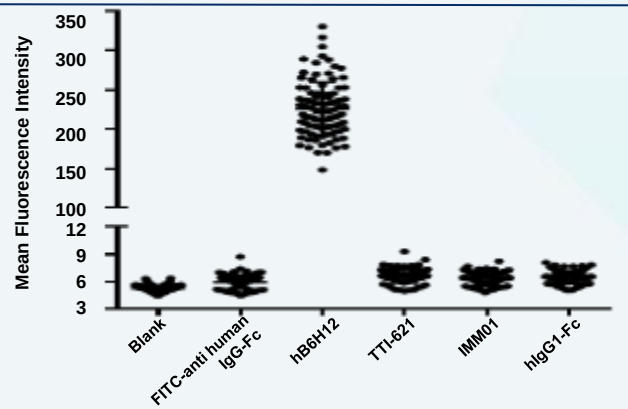


- First SIRPα-Fc fusion protein to enter the clinical stage in China
- Improved safety profile with engineered CD47-binding domain of human SIRPα to avoid human RBC binding
- Designed to fully activate macrophages through a dual mechanism
- Encouraging single-agent efficacy at relatively low dose, with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

Source: Company Data

Competitive Advantage of IMM01 Monotherapy - Safety

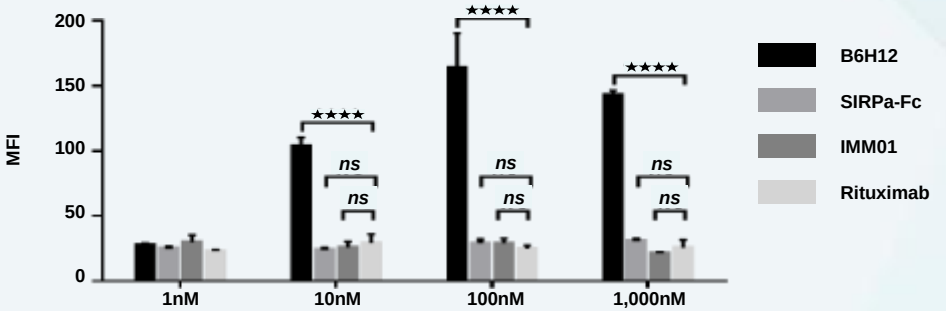
Human RBC Binding Analysis of IMM01



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

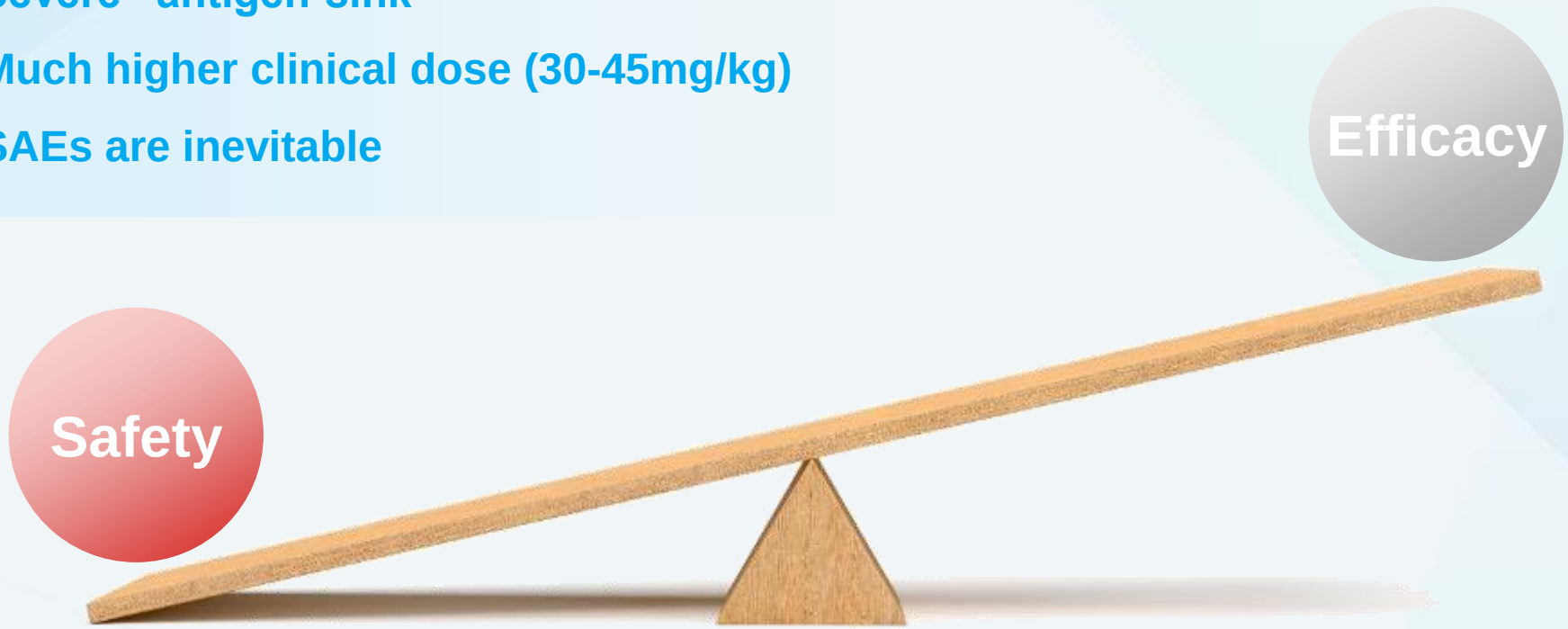


Notes: B6H12 is a CD47-based antibody that serves as the control.

Challenges for CD47-Targeted Drug Development

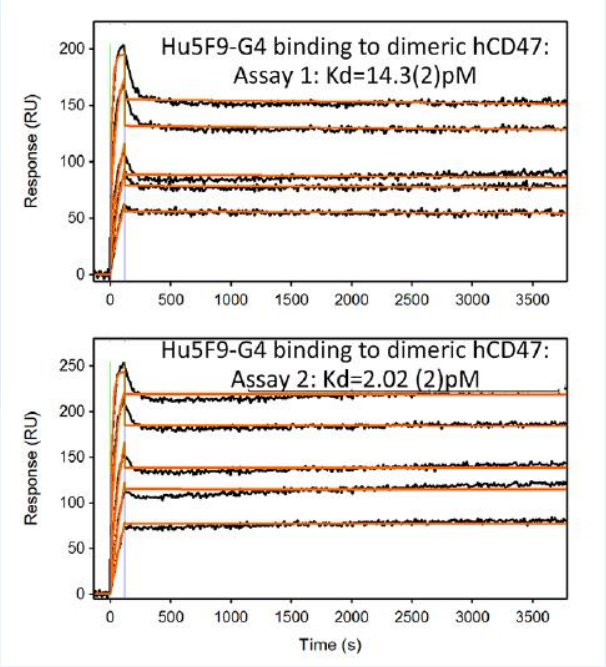
CD47 Antibody

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

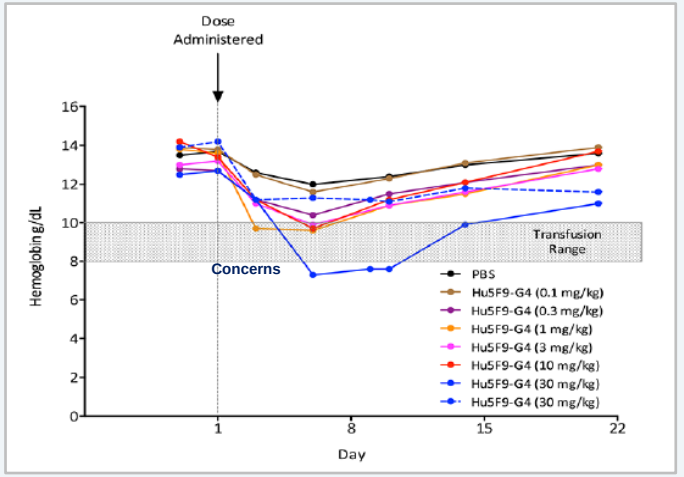


Magrolimab Has Very High Target Affinity and RBC Binding Activity

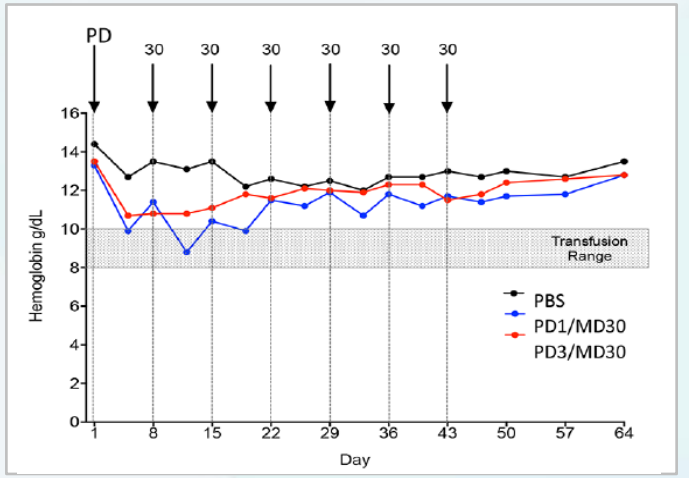
Target affinity assay



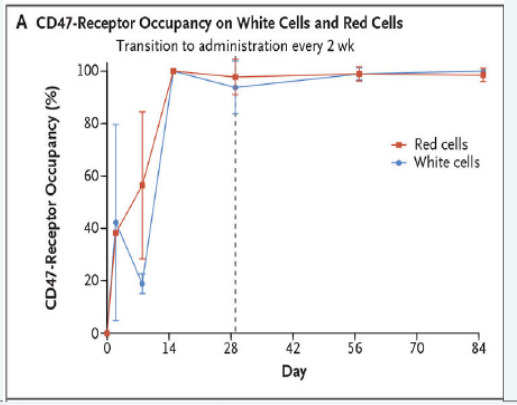
Without priming dose



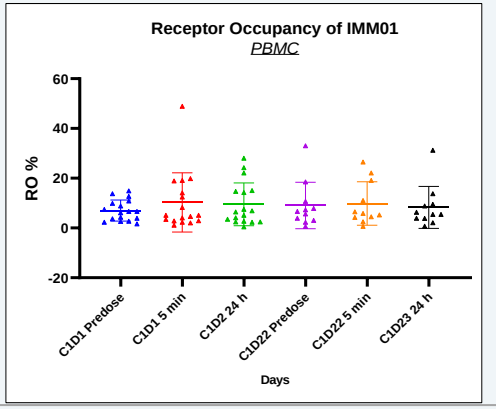
With priming dose (1mpk, 3mpk)



Magrolimab Receptor Occupancy (RO)



IMM01 Receptor Occupancy (RO)



Magrolimab: KD = 2-14.3pM

Timdarpacept (IMM01): KD = ~3nM

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)**

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

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Thank you!