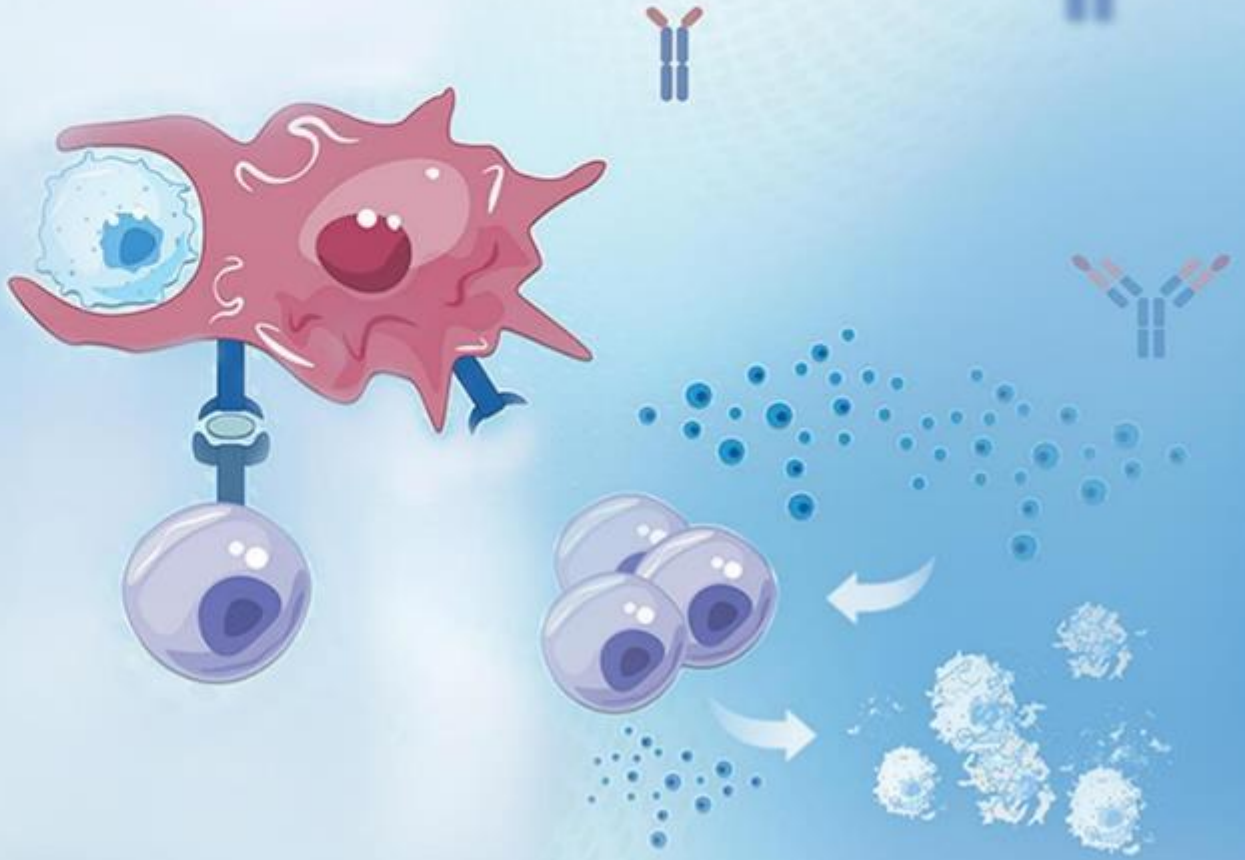
宜明昂科
ImmuneOnco

Corporate Presentation

September 2025



Disclaimer

THIS DOCUMENT OR THE INFORMATION CONTAINED HEREIN IS NOT INTENDED TO AND DOES NOT CONSTITUTE ANY OFFER OR INVITATION, SOLICITATION, COMMITMENT OR ADVERTISEMENT OF ANY OFFER FOR SUBSCRIPTION, PURHCASE OR SALE OF ANY SECURITIES, NOR SHALL ANY PART OF THIS DOCUMENT FORM THE BASIS OF OR BE RELIED ON IN CONNECTION WITH ANY CONTRACT OR COMMITMENT WHATSOEVER.

This document is strictly confidential to the recipient only, and may not be copied, reproduced, redistributed, disseminated, or used or disclosed to any other person, or published, in whole or in part, for any other purpose. This document has been prepared by ImmuneOnco Biopharmaceuticals (Shanghai) Inc. (the "Company") but without further investigation and cannot be warranted as to its accuracy or completeness. Neither the Company, its advisors and representatives nor any of their respective subsidiaries or affiliates have or may have been able to verify independently any or all such information or assumptions made, or there may exist other facts, risks or considerations which might be material concerning the information herein. Accordingly, neither the Company, its advisors and representatives, nor any of their respective subsidiaries or affiliates, nor any of their respective directors, officers, employees or agents, make any representation or warranty, expressed or implied, with respect to the information or assumptions contained in this document or on which this document is based, or that the information or assumptions remains unchanged after the issue of this document, and will not accept any loss, liability or responsibility whatsoever for the accuracy or completeness of the information or assumptions on which this document is based.

This document does not have regard to the specific investment objectives, financial situation or particular needs of any specific persons who may receive this document. This document is not to be relied upon as such or used in substitution for the exercise of independent judgment. The recipient must make its own assessment of the relevance, accuracy and adequacy of the information contained or assumptions made in this document prior to entering into any transaction or investment.

Certain data in this document was obtained from external data sources, and the Company has not verified such data with independent sources. Accordingly, the Company and its advisors and representatives make no representations as to the accuracy or completeness of that data. Such data involves risks and uncertainties and is subject to change based on various factors. The use of registered trademarks, commercial trademarks and logos or photographic materials within this document are exclusively for illustrative purposes and are not meant to violate the rights of the creators and/or applicable intellectual property laws.

Certain statements are set forth in this document with respect to the Company or other events, including but not limited to opinions and forward-looking statements with respect to the future financial condition and results of operations of the Company and certain plans and objects of the management of the Company. Such statements are based on a number of assumptions, including but not limited to the present business strategies of the Company and other matters beyond the control of the Company, such as the political, social, legal and economic environment in which the Company will operate in the future. Such statements are subject to known and unknown risks, uncertainties and other factors which may cause the actual performance or results of operations of the Company to differ materially from such opinions or forward-looking statements or the views, expressed or implied, contained in this document. No reliance should be placed on such statements, which reflect the view of the management of the Company as at the date of this document. Neither the Company nor any of its advisors or representatives shall be obliged in any way to update such opinions or forward-looking statements for any event or circumstances that may occur. In any case, past performance is not necessarily an indication of future results.

This document is for information and reference only and does not constitute or form part of and should not be construed as, an offer to sell or issue or the solicitation of an offer to buy or acquire securities (the "Securities") of the Company in any jurisdiction or an inducement to enter into investment activity nor should it form the basis of, or be relied on in connection with, any contract or commitment or investment decision whatsoever. In particular, this document and the information contained herein are not an offer of the securities for sale in the United States and are not for publication or distribution in the United States. The document is being presented to you on the basis that you have confirmed that you are either (i) a qualified institutional buyer (as defined in Rule 144A under the U.S. Securities Act of 1933, as amended (the "Securities Act")) or (ii) a non-U.S. person (as defined in Regulation S under the Securities Act). This document is not intended for distribution to persons who are not professional investors (as defined in Schedule 1 to the Securities and Futures Ordinance (Chapter 571 of the Laws of Hong Kong)).

THE SECURITIES HAVE NOT BEEN, AND WILL NOT BE, REGISTERED UNDER THE SECURITIES ACT, OR THE SECURITIES LAWS OF ANY STATE OF THE UNITED STATES OR ANY OTHER JURISDICTION AND MAY NOT BE OFFERED OR SOLD WITHIN THE UNITED STATES, EXCEPT IN CERTAIN TRANSACTIONS EXEMPT FROM, OR NOT SUBJECT TO, THE REGISTRATION REQUIREMENTS OF THE SECURITIES ACT. NO PUBLIC OFFERING OF ANY SUCH SECURITIES WILL BE MADE IN THE UNITED STATES OR IN ANY OTHER JURISDICTION WHERE SUCH AN OFFERING IS RESTRICTED OR PROHIBITED.

H1 2025 Highlights and Upcoming Milestones

Timdarpacept (IMM01)

- **ASCO 2025 poster:** in combination with AZA in 1L CMML(PhII) and combination with tislelizumab in anti-PD-1 failed R/R cHL(PhII): updating **mPFS** to **17.8M and 14.7M** respectively
- Ph III trials are advancing for 1L CMML and prior PD-(L)1-refractory cHL
- Approval from CDE for combination with IMM2510 in PhIb/II clinical trials for solid tumors

Palverafusp alfa (IMM2510)

- Published the preliminary data for 1L NSCLC in combination with chemo: **ORR was 62%** (13/21), with an **ORR of 80%** (8/10) for **sq-NSCLC**; the majority of efficacy evaluable patients had only one tumor assessment as of July 1, 2025
- IMM2510 momotherapy for I/O treated advanced sq- NSCLC: **ORR was 35.3%** (6/17), **mPFS was 9.4M** (WCLC 2025 poster)
- Obtained IND approval for R/R solid tumors from the FDA

Amulirafusp alfa IMM0306

- **ASCO 2025 poster:** in combination with LEN for R/R FL(PhII)- **ORR was 88.2%, CRR was 52.9%**
- Published the preliminary data of SLE(PhIb), demonstrating favorable efficacy and safety profile. The percentage of patients with a reduction in SLEDAI-2000 by ≥4 was **87.5%** (7/8) in the 1.2 mg/kg cohort

IMC-003 ActRIIA Fc-fusion

- obtained **IND approval** in June 2025 and initiated patient enrollment in August, making it the fastest progressing innovative molecule with the same target for the treatment of PAH in China, aside from sotatercept

Upcoming Milestones

The data of IMM2510 momotherapy for I/O treated advanced sq- NSCLC will be presented at WCLC 2025

WCLC 2025

The preliminary data of IMM0306 for SLE (PhIb) will be presented at ACR 2025

ACR Convergence Where Rheumatology Meets #ACR25

IMM0306 in combination with LEN for R/R FL(PhII)

IMM2510 in combination with chemo (PhII)

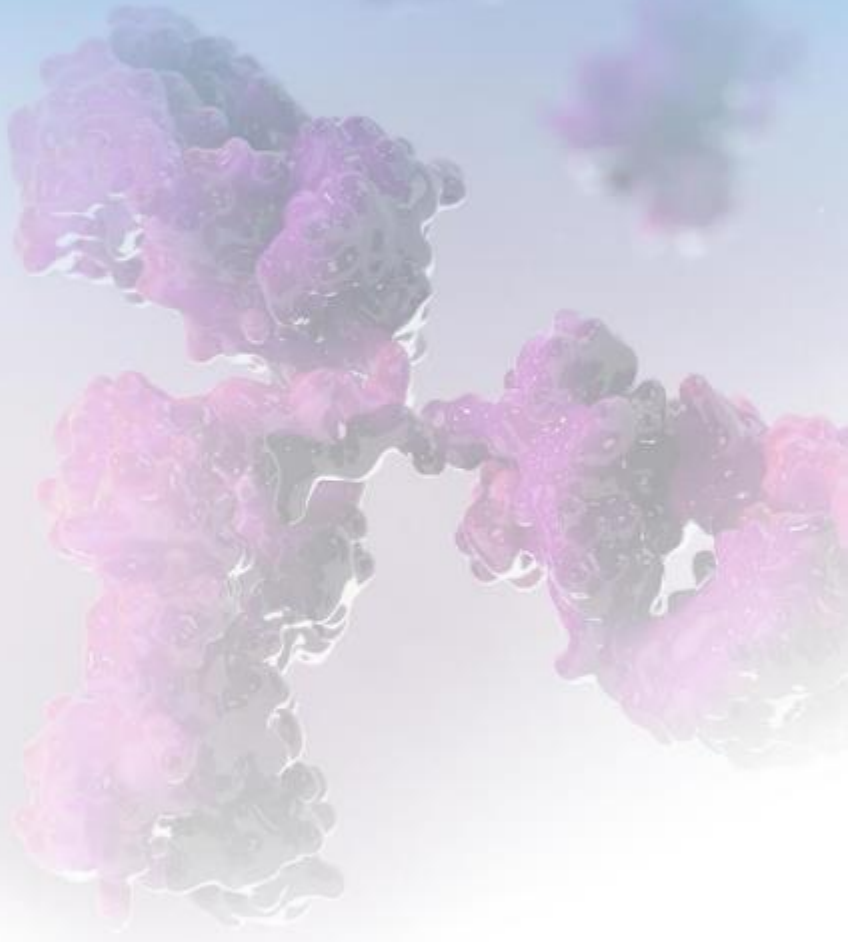
Future international academic conferences….

Table of Contents

宜明昂科
ImmuneOnco

SECTION 1

Company Overview



Key Milestones

- Steady team with **10+** years coordination
- **30** issued patents
- **31** pending patent applications
- **31** IND approvals from the NMPA and the FDA
- **12** 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

2021-2022

IMM01:
- IND approval by NMPA for the Phase Ib/II in with each of azacitidine and inetetamab
- Phase II in combination with either PD-1 mAb or azacitidine commenced in China

IMM0306:
- IND approval by FDA

IMM2902:
- IND approval by NMPA and FDA

IMM27M:
- IND approval by NMPA, FPI

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 cHL & CMML 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
- SLE& NMOSD dosed first patient

IMM2510:
- Phase Ib in combination with IMM27M for solid tumors dosed first patient
- Phase Ib/II in combination with chemo for 1L NSCLC first patient
- Reached a license-out agreement of US$2.1B with Instil Bio

2025

IMM01
- IND approved for IMM01+ IMM2510 for advanced solid tumors in China

IMM2510:
- +chemo 1L NSCLC phaseIb/II FPI
- IND approved by FDA

IMM0306:
- Published preliminary data of SLE, demonstrating favorable efficacy and safety

IMC-003
- IND approved by CDE

2015 2016 2017 2018 2019 2020 2021 2022 2023 2024 2025

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	2024: Placement, US$30 MM

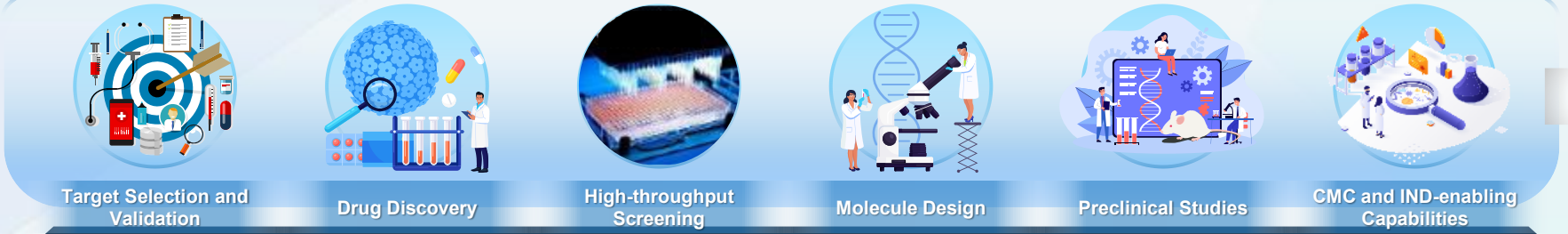
Key Investors

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 · 南京星健睿赢 · 荣昌股权投资

Total amount of fund raised: ~$285MM

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 ~60 members

Global Rights

- **31** IND approvals from the NMPA and the FDA
- **30** Issued patents
- **31** Pending patent applications

Pilot manufacturing: 200L/250L bioreactors

Comprehensive Pipeline Covering Oncology and non-Oncology Therapeutic Areas

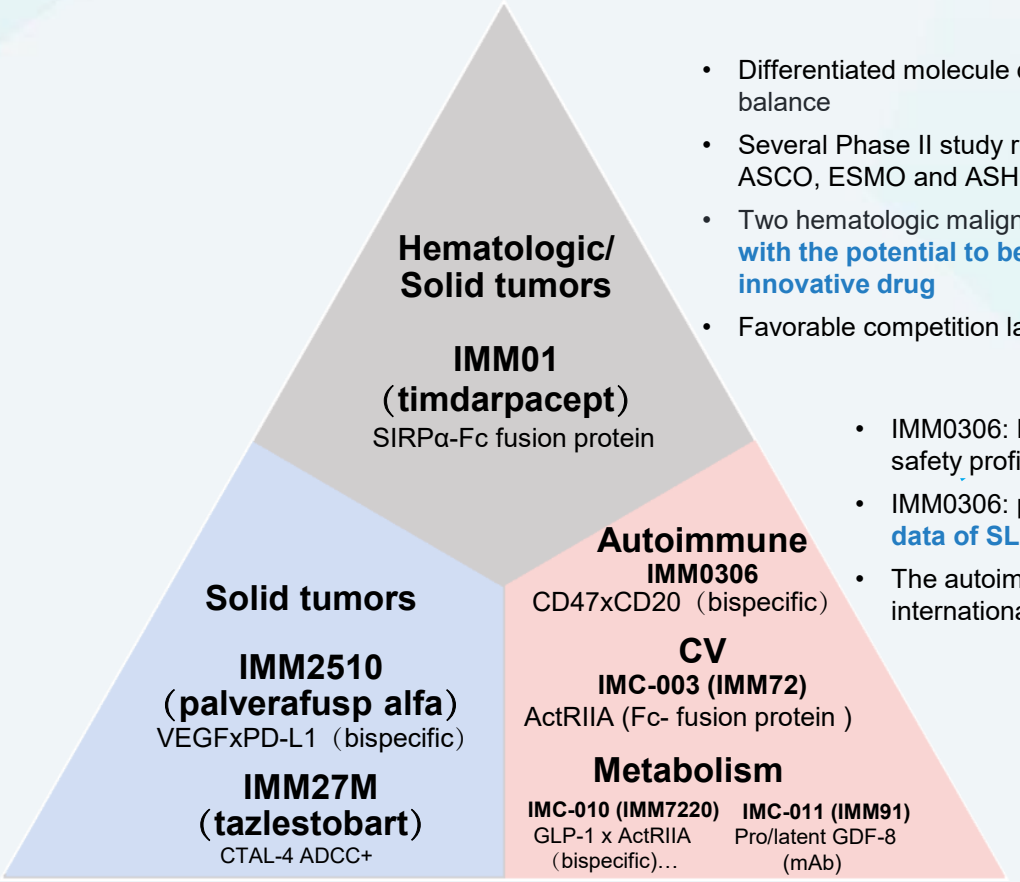
Program [1]	Target (Modality)	Indication(s)	Discovery	Preclinical	IND/IND-Enabling	Phase Ia/I	Phase Ib/II	Phase III/ Pivotal	Partners	Current Status / Upcoming Milestone	Commercial Rights
IMM01 （timdarpacept）											
IMM01 + Azacitidine	CD47 (SIRPα-Fc fusion protein)	MDS[2]					China (NMPA)			Received Phase III approval from CDE in May 2024	Global
IMM01 + Azacitidine	CD47 (SIRPα-Fc fusion protein)	1L CMML					China (NMPA)			Received Phase III approval from CDE in June, FPI in November 2024	Global
IMM01 + Tislelizumab	CD47+PD-1	cHL[3]					China (NMPA)			Received Phase III approval from CDE in April; FPI in July 2024	Global
IMM01 + IMM2510	CD47+VEGFxPD-L1	Solid Tumors					China (NMPA)			Received Phase Ib/II approval from CDE in March 2025	Global
IMM2510 （palverafusp alfa） Monotherapy	VEGFxPD-L1 (Bispecific)	Solid Tumors					China (NMPA)		InstilBio	Phase Ib/II commenced in November 2023 in China	Great China
IMM2510 + Chemo	VEGFxPD-L1 (Bispecific)	1L NSCLC					China (NMPA)		InstilBio	IND approved in China in November 2023, FPI in December 2024	Great China
IMM2510 + Chemo	VEGFxPD-L1 (Bispecific)	1L TNBC					China (NMPA)		InstilBio	IND approved in China in November 2023, FPI in June 2025	Great China
IMM2510 + IMM27M	VEGFxPD-L1 (Bispecific) + CTLA-4	Solid Tumors					China (NMPA)		InstilBio	IND approved in China in October 2023, FPI in July 2024	Great China
IMM27M （tazlestobart）	CTLA-4 ADCC+ (mAb)	Solid tumors					China (NMPA)		InstilBio	Phase Ia completed in September 2023 in China，FPI for Phase Ib HR+ mBC in September 2024	Great China
IMM0306 (amulirafusp alfa) IMM0306 + Lenalidomide	CD47xCD20 (Bispecific)	R/R FL&MZL					China (NMPA)			LPI for FL cohort in December 2024	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	Global
IMM0306 (amulirafusp alfa)	CD47xCD20 (Bispecific)	SLE					China(NMPA)			FPI in October 2024, completed the first and second cohort enrollment in July,2025	Global
		NMOSDs					China(NMPA)			FPI in December 2024, completed patient enrollment for dose escalation	Global
		LN					China(NMPA)			IND approved in China in December 2024	Global
IMM01 （timdarpacept）	CD47 (SIRPα-Fc fusion protein)	Atherosclerosis								IND-enabling	Global
IMC-003 (IMM72)	ActRIIA (Fc-fusion protein)	PAH, Undisclosed					China(NMPA)			IND approved in China in June 2025, FPI in August 2025	Global
IMC-010 (IMM7220)	GLP-1xActRIIA (Bispecific)	Obesity (lose fat and build muscle)								In vivo efficacy study is ongoing	Global
IMC-011 (IMM91)	Pro/latent GDF-8 （mAb）	Obesity (lose fat and build muscle)								IND-enabling	Global

Innate Immunity Targets | Innate and Adaptive Immunity Targets | Adaptive Immunity Targets | CV, autoimmune, 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 prior PD-(L) 1-refractory cHL.

Three Strategic Product Matrices Support Future Growth

We have matured proprietary R&D platform and comprehensive innovation pipeline portfolio



- Differentiated molecule design to achieve optimal safety and efficacy balance
- Several Phase II study results were selected for oral presentation at ASCO, ESMO and ASH
- Two hematologic malignancy indications have advanced to phase III, **with the potential to become the first approved CD47-targeted innovative drug**
- Favorable competition landscape and with enormous market potential

- The Phase III clinical trial data of a peer molecule demonstrated strongly positive results in a head-to-head comparison against Keytruda, proving the immense potential of VEGFxPD-(L)1 bispecific molecules
- The combination therapy with chemo for 1L NSCLC has entered phase Ib/II, with **the preliminary data readout in H2 2025**

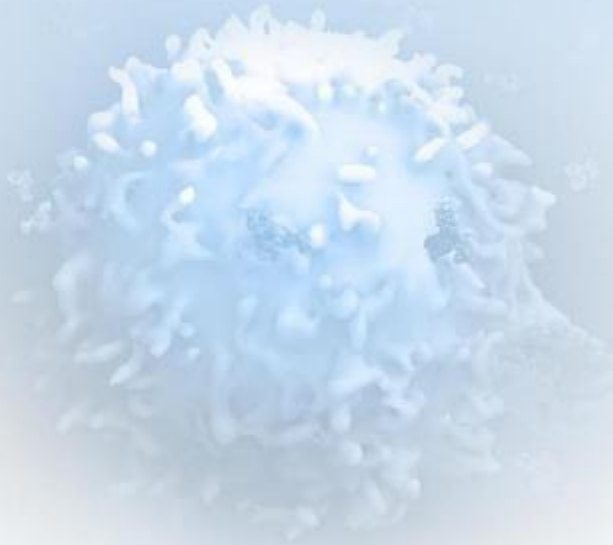
- IMM0306: B-cell depletion without CRS demonstrates a favorable safety profile
- IMM0306: phase Ib/II is ongoing for SLE and MNOSD; **preliminary data of SLE will be presented as poster at ACR 2025**
- The autoimmune disease field has enormous market potential, with international BD collaborations booming

- **IMC-003 has received IND approval in China in June, FPI in August**
- Multiple new-generation candidates targeting GDF-8 ActRIIA/B, **with strategic in-depth layout in obesity (lose fat and build muscle)**

宜明昂科
ImmuneOnco

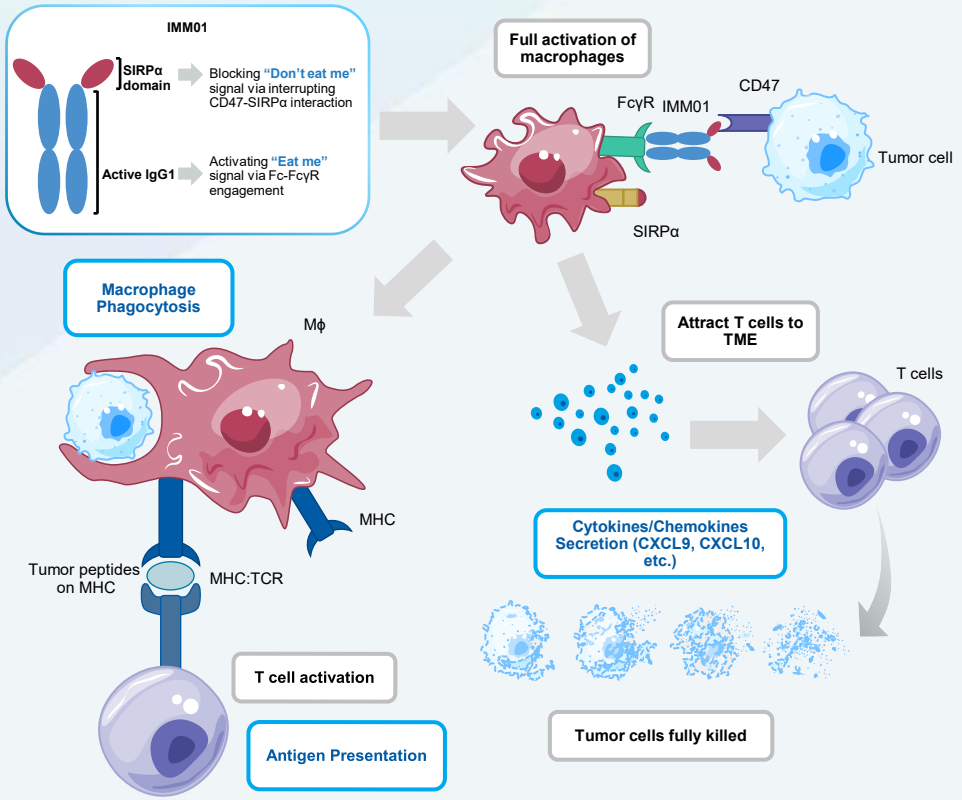
SECTION 2

Major Oncology Programs



Timdarpacept (IMM01)

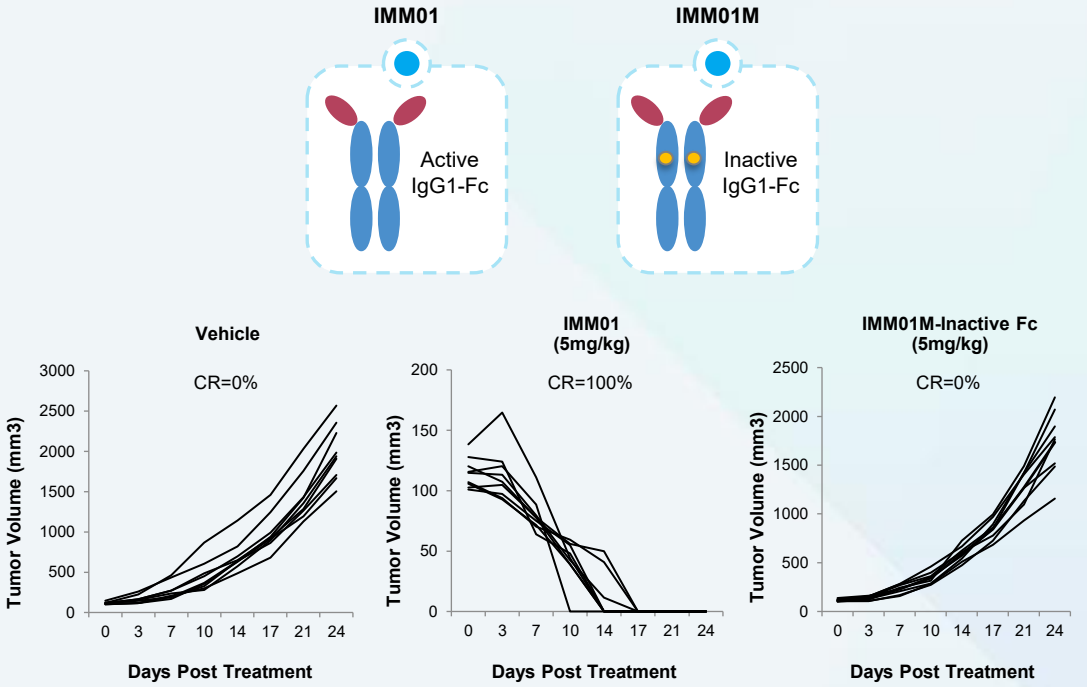
Overview and Competitive Advantage of IMM01(Timdarpacept)



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.

Timdarpacept（IMM01）- Outstanding Ph II Clinical Trial Data

- The Phase II clinical data for the three indications listed below are impressive, having gained oral presentations at top international academic conferences
- We have received phase III clinical approvals for the three indications in China; with **enrollment for prior PD-(L) 1-refractory classical Hodgkin lymphoma (cHL) and newly diagnosed chronic myelomonocytic leukemia (CMML) in 2024.** Timdarpacept holds a leading position globally in the progress of clinical trial for CD47-based therapies

+ Azacitidine in 1L HR-MDS (ph II)[1]

Best Overall Response n (%)	1L MDS (N=51)
ORR	33 (64.7%)
DCR	45 (88.2%)
CR	17 (33.3%)
mCR+HI	8 (15.7%)
mCR	6 (11.8%)
HI	2 (3.9%)
SD	12 (23.5%)

2024 ASCO ANNUAL MEETING ASH

Oral Presentation

+ Azacitidine in 1L CMML (ph II) [1]

Best Overall Response n (%)	1L CMML (N=22)
ORR	16 (72.7%)
CR	6 (27.3%)
mCR + HI	3 (13.6%)
mCR	6 (27.3%)
HI	1 (4.5%)
mPFS	17.8M (95%CI, 5.3-NR)

ESMO Oral Presentation

2025 ASCO ANNUAL MEETING Poster

+ Tislelizumab in Anti-PD-1 Failed R/R cHL(ph II) [2]

Best Overall Response n (%)	R/R cHL (N=33)
ORR	23 (69.7)
DCR	31 (93.9)
CR	8 (24.2)
PR	15 (45.5)
SD	8 (24.3)
PD	2 (6.1)
mPFS	14.7M (95%CI, 7.0-NA)

2024 ASCO ANNUAL MEETING ESMO ASH

Oral Presentation

2025 ASCO ANNUAL MEETING Poster

Source: Company Data; 1. the clinical data is as of Dec 31st, 2024
2. The clinical data is as of Mar 31st, 2025

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

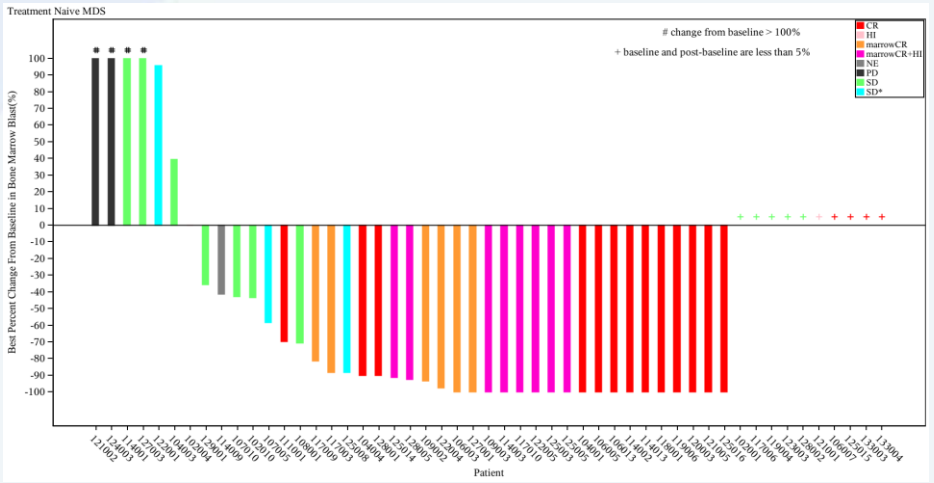
There are two major guidelines recommending the treatment of myelodysplastic syndromes (MDS):

1. The Chinese Guidelines for the Diagnosis and Treatment of Myelodysplastic Syndromes (2019 Edition), formulated by the Chinese Medical Association.

2. The MDS Treatment Guidelines (2023.v1.0), formulated by the National Comprehensive Cancer Network (NCCN).

Hypomethylating agents (HMAs) are the standard treatment for most newly diagnosed patients with higher-risk MDS.

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



Timdarpacept+AZA VS SOC (Non-head-to-head Comparison)

Best Overall Response n (%)	Timdarpacept +AZA N=51	1L HR-MDS SOC in China（AZA）[1] N=72	Systematic literature review and meta-analysis of AZA in 1L HR- MDS patients [2]
ORR	**64.7%**	/	50.0%
CR	**33.3%**	9.7%	16.0%
PR	/	0	6.0%

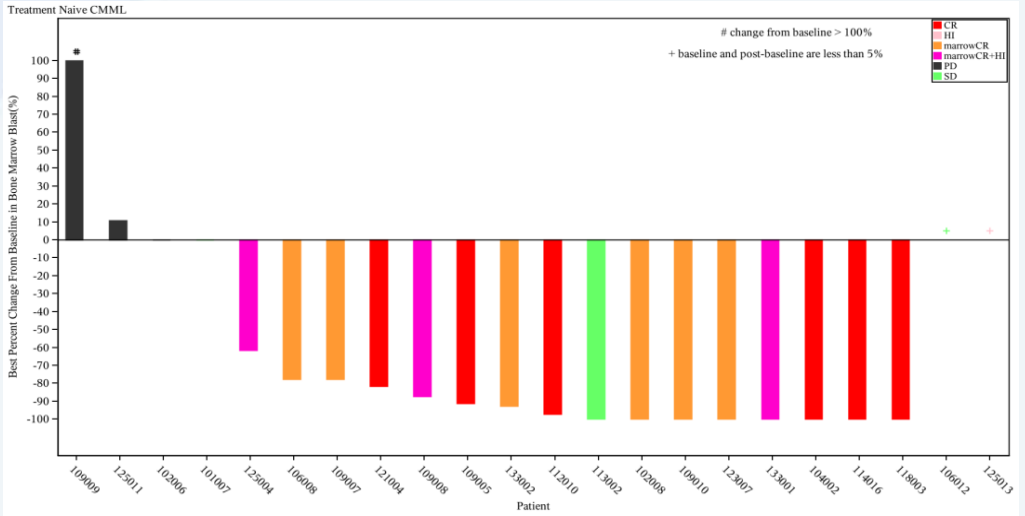
Source: Company Data; the clinical data is as of Dec 31st, 2024

1. Xin Du, et al. Asia Pac J Clin Oncol . 2018;14(3):270-8.
2.Including 16 studies（5 RCT trials, 3 prospective studies and 8 retrospective studies）

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

- The options for treating CMML are very limited, apart from the approval of HMAs for MDS (only a small number of CMML patients have been included in the pivotal studies for MDS).
- Over the past 20 years, regulatory agencies have not approved any new drugs specifically for CMML indications, resulting in a significant unmet clinical need for CMML treatment.

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



Timdarpacept +AZA VS SOC (Non-head-to-head Comparison)

Best Overall Response n (%)	Timdarpacept +AZA 1L CMML (N=22)	A multicenter retrospective study in China（N=24）[1]
ORR	**72.7%**	37.5%
CR	**27.3%**	8.3%
mPFS	17.8M (95%CI, 5.3-NR)	/

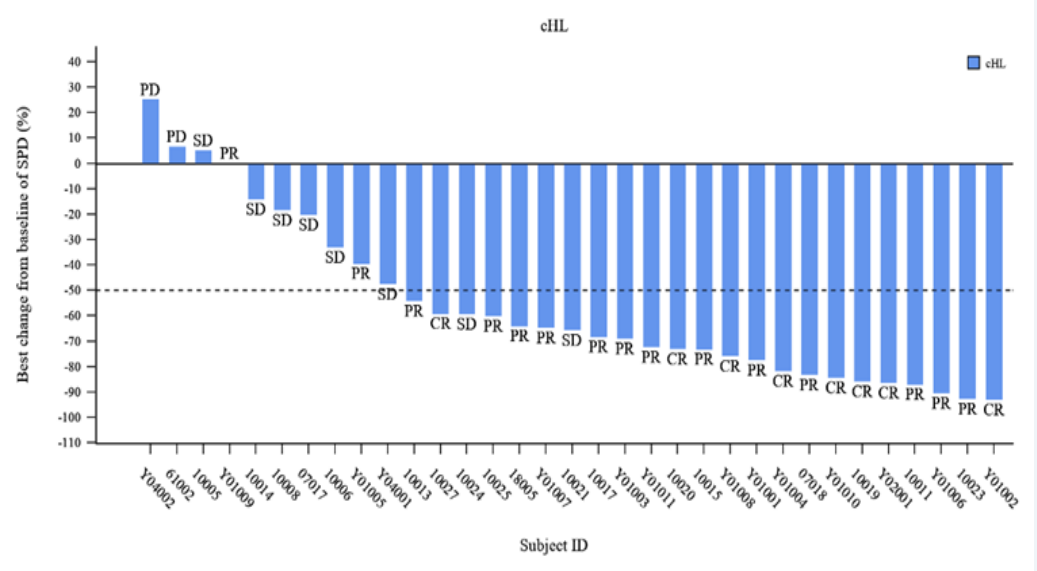
Source: Company Data; the clinical data is as of Dec 31st, 2024

1. YU Xu, 2022

Timdarpacept (IMM01) + Tislelizumab (PD-1 mAb) in Anti-PD-1 Failed R/R cHL (Phase II)

- Anti-PD-1 Failed R/R cHL: there is currently no standard treatment available, neither domestically nor internationally, and treatment options are limited. Clinicians rely on chemotherapy based on their experience or enroll patients in clinical trials, highlighting an urgent clinical need.

Best Percentage Change from Baseline in Target Lesion



Ph II Data Comparison of R/R cHL (Non-head-to-head Comparison)

Best Overall Response (%)	Tislelizumab (SIRPα-Fc)+ Tislelizumab（PD-1） N=33	Favezelimab (Anti-LAG-3) + Pembrolizumab 1 N=34	Tifcemalimab (Anti-BTLA) + Toripalimab (PD-1) [2] N=34
ORR	**69.7%**	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 Stopped in Dec 2024	Phase III started in Dec 2023 to treat R/R cHL
Study Geography	China	China + International	China

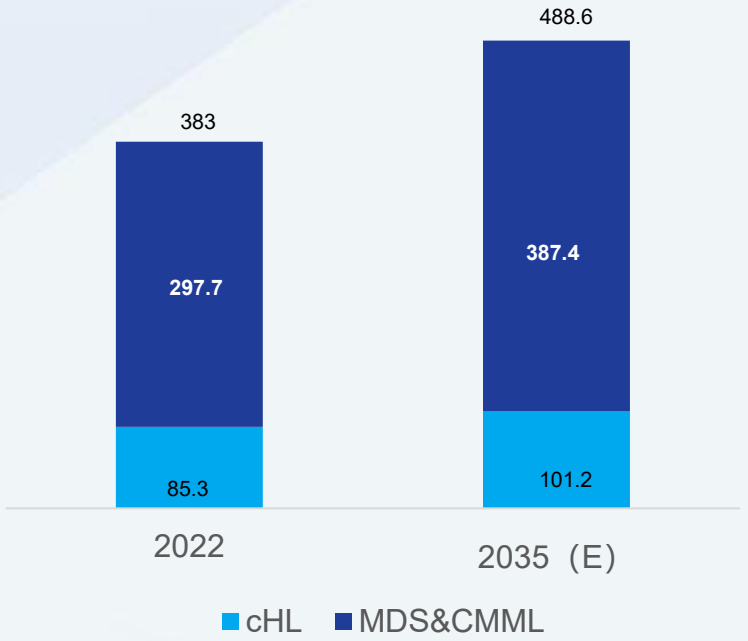
Source: Company Data; the clinical data is as of March 31st, 2025

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

Strong Potentials of CD47-based Therapies

Timdarpacept holds a leading position globally in the progress of clinical trial for blood tumors and has been **approved for combination with IMM2510 in Phase Ib/II clinical trials for solid tumors**. The clinical trial is set to commence soon.

Global Incidence of cHL, MDS&CMML*
Thousand



- 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**
- **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

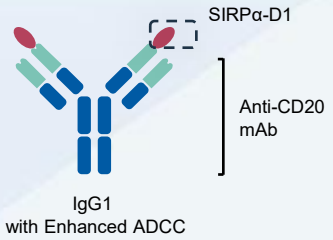
* Source: Frost & Sullivan, Company Prospectus

Amulirafusp alfa (IMM0306) (CD47×CD20)

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

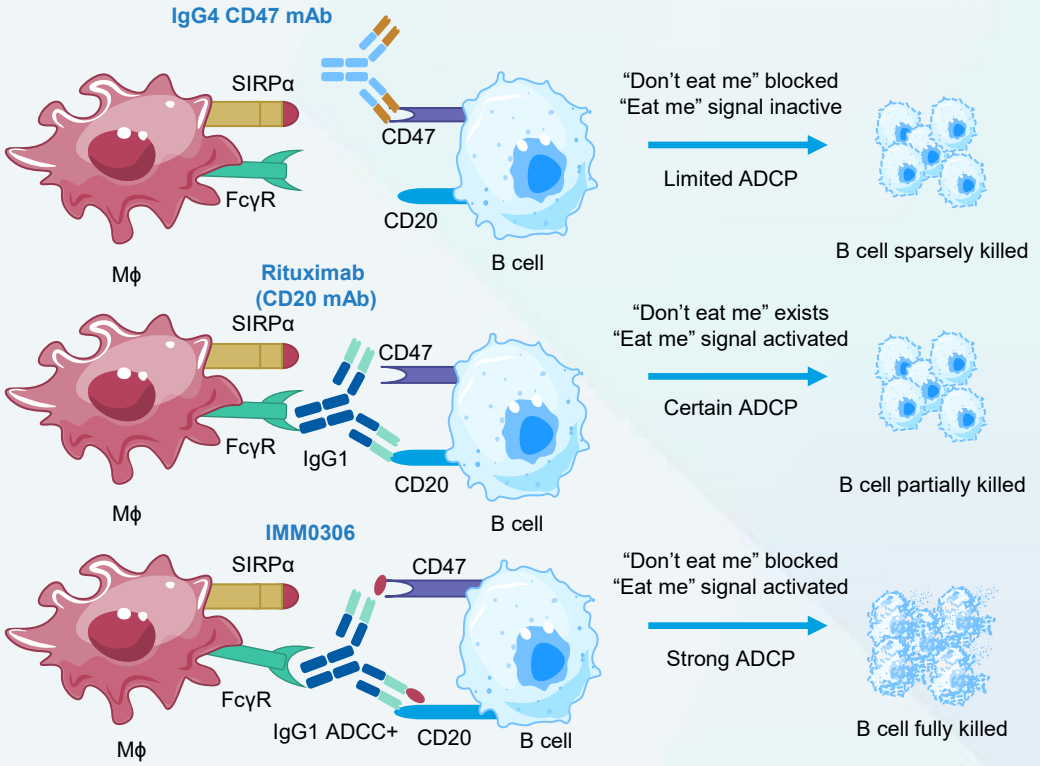
Overview

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**

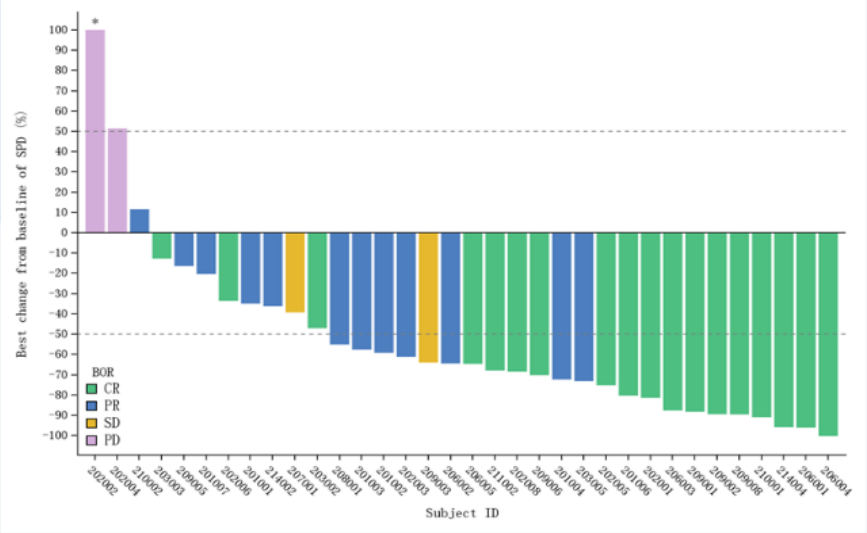
2 CD47×CD20 bispecific antibodies/fusion proteins under development globally
Among them, IMM0306 is the **1st** to enter into a clinical trial

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

Amulirafusp alfa (IMM0306) in Combination with Lenalidomide

- The Phase II data of amulirafusp alfa in combination with lenalidomide in relapsed and refractory follicular lymphoma showed excellent CRR and ORR

Best Percentage Change from Baseline in Target Lesion in Phase II



IMM0306+LEN R/R FL VS R/R FL Data of Other Therapies

	IMM0306+ Len (PhII) N=34	Obinutuzumab +Len [1] N=86	Obinutuzumab+ Bendamustine[2] N=155	Rituximab+ Len (R2) [3] N=147	Tafasitamab+ R2 [4] N=273
ORR	**88.2%**	79.0%	78.7%	80.3%	83.5%
CR	**52.9%**	38.0%	15.5%	34.7%	52.0%
PR	35.3%	41.0%	63.2%	45.6%	31.5%

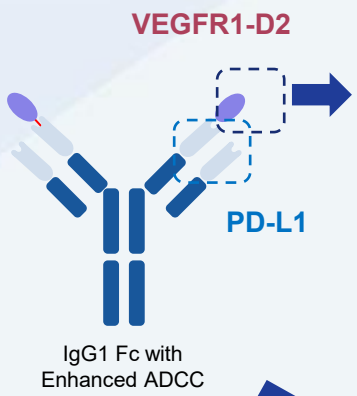
Source: Company Data; the clinical data is as of March 14th, 2025 (ASCO Poster)
1. NCCN2025； Lancet Haematol 2019;6:e429-e437
2. NCCN2025； Lancet Oncol 2016;17:1081-1093； Label
3. NCCN2025； J Clin Oncol 2019;37:1188 1199； Label
4. 2024 ASH data

Palverafusp alfa(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

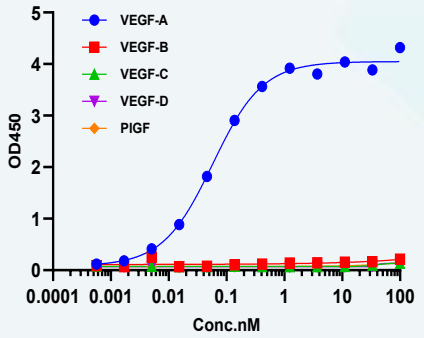
Tumor cells expressing PD-L1 can **bind to PD-1** on the surface of T cells to evade T-cell attacks

ADCC

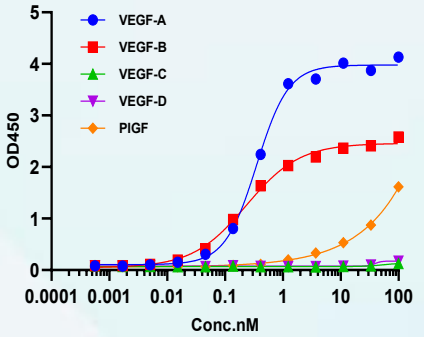
ADCC-enhanced antibody designed to induce direct killing of immunesuppressive PD-L1+ tumor cells

IMM2510 binds multiple VEGF receptor ligands

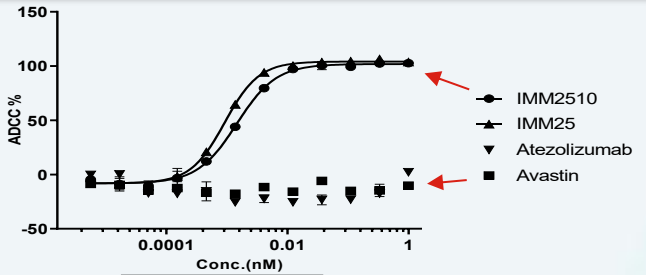
Avastin binding to various VEGFs



IMM2510 binding to various VEGFs



IMM2510 has enhanced ADCC activity



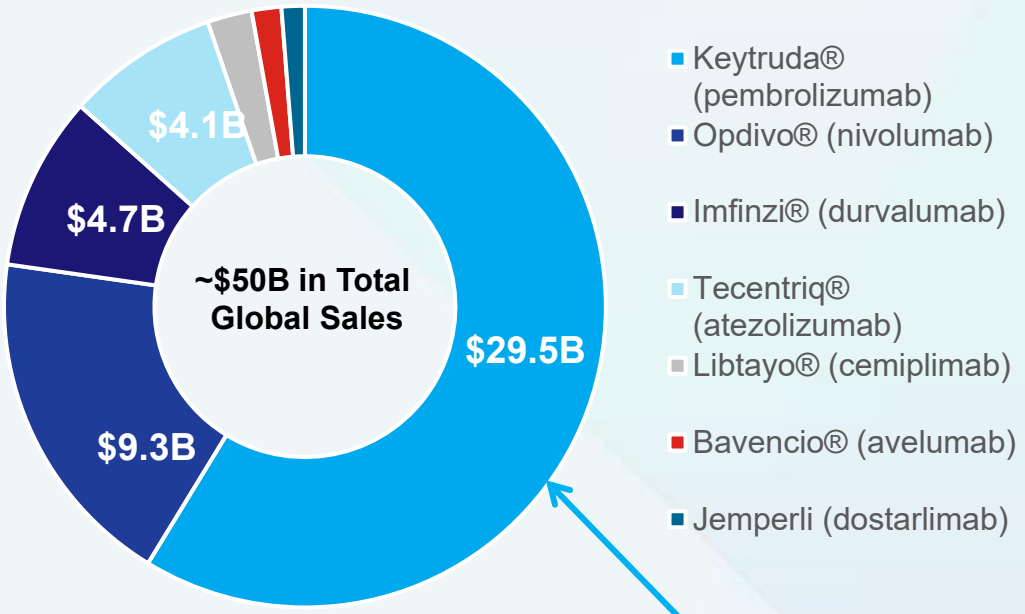
	EC50
IMM2510	0.001461
IMM25	0.0009247

Palverafusp alfa(IMM2510) (VEGF × PD-L1)

IMM2510 targets largest market in oncology: NSCLC

- **NSCLC is the largest market opportunity for Keytruda,** accounting for ~ 1/3 of sales
- **PD-(L)1** inhibitors are estimated to reach ~$90B in global sales in 2028[1]
  - Four PD-(L)1 inhibitors achieved >$4B in sales in 2024[2]
- **VEGF** inhibitor market represents additional opportunity for expansion

2024 Sales of PD-(L)1 Inhibitors[2]



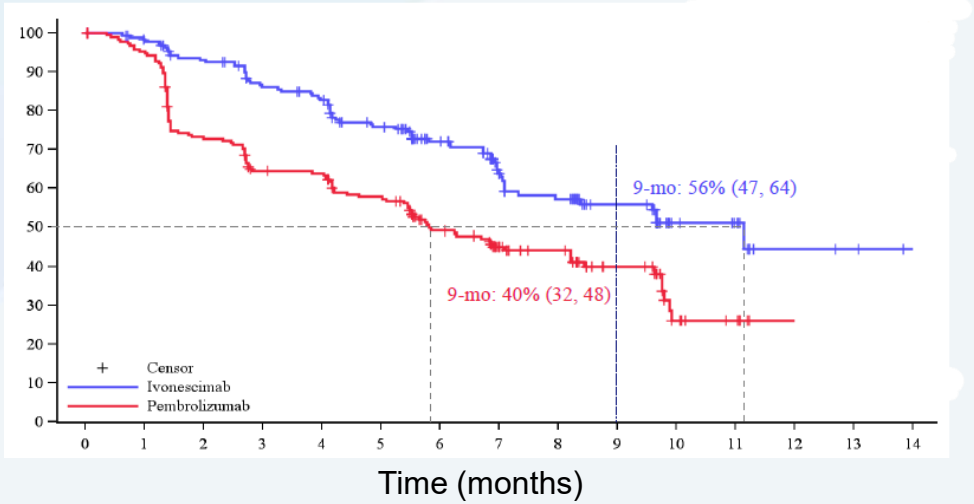
Keytruda® (pembrolizumab) alone represented **$29.5B**, with **~$10B** coming from lung cancer indications.[3]

Notes:
1. IQVIA Institute for Human Data Science, "Global Oncology Trends 2024: Outlook to 2028"
2. Company earnings releases
3. Stifel research report published on March 25, 2024.

Palverafusp alfa(IMM2510) (VEGF × PD-L1)

PD-(L)1xVEGF Bispecifics Outperform Pembrolizumab

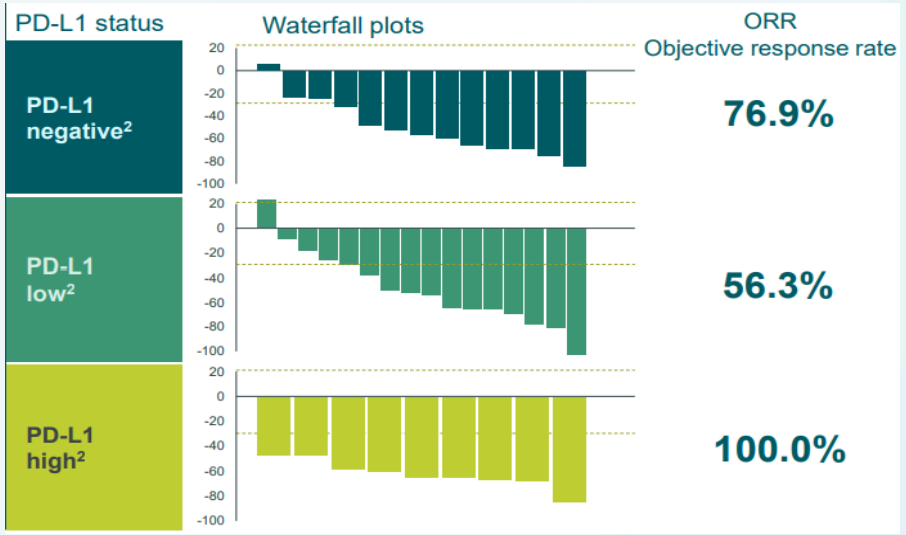
Ivonescimab: HARMONi-2 Trial[1]



In the Phase III HARMONi-2 trial, ivonescimab **showed clinically meaningful improvement over pembrolizumab** in patients with PD-L1-positive NSCLC on PFS (HR:0.51, p<0.0001) and OS (HR:0.777, p=NS).

PD-(L)1xVEGF bispecifics have largely avoided significant VEGF-associated toxicities, including serious bleeding events.

BNT327: TNBC Trial[2]



BNT327 drove clinical benefit irrespective of PD-L1 status in combination with chemotherapy in patients with TNBC, demonstrating that PD-(L)1xVEGF bispecifics **can potentially treat patient populations not currently addressed by existing PD-(L)1 therapies**.

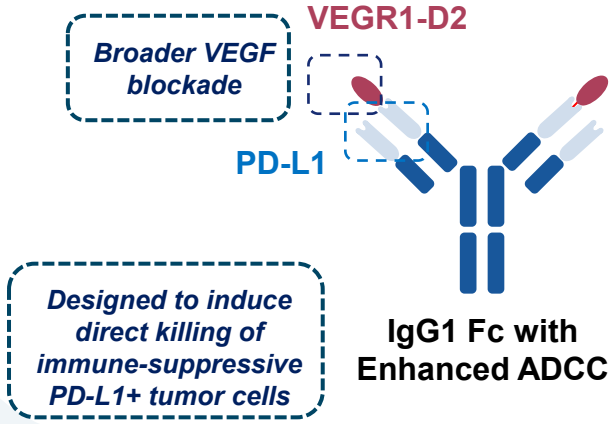
1. Zhou et al. Presented at WCLC 2024.
2. Y. Meng et al. Presented at ESMO 2024.

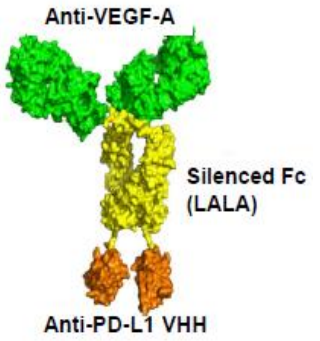
NS = not statistically significant; TNBC: triple-negative breast cancer

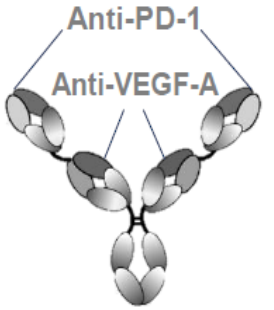
Palverafusp alfa(IMM2510) (VEGF × PD-L1)

Key Competitor Landscape

	IMM2510 (ImmuneOnco / Instil Bio)	PM8002 (BioNTech)	AK112 (Akeso / Summit)	SSGJ-707 (3SBio/ Pfizer)
VEGF binding	VEGF-A, VEGF-B, PlGF	VEGF-A	VEGF-A	VEGF-A
PD-1 or PD-L1	PD-L1	PD-L1	PD-1	PD-1
ADCC	Enhanced ADCC	None	None	None



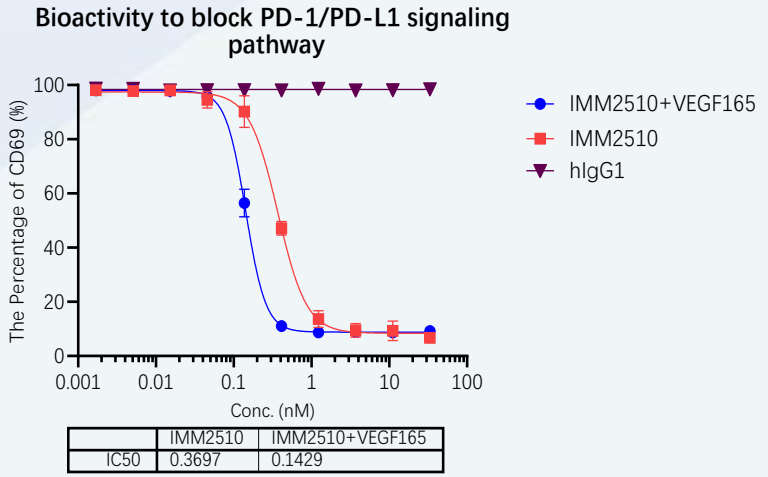




Palverafusp alfa(IMM2510) (VEGF × PD-L1)

IMM2510 demonstrates cooperative binding to PD-L1 *in vitro*

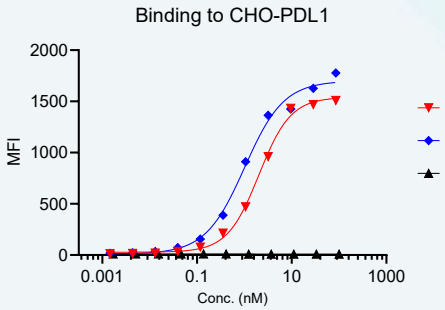
Presence of VEGF enhances PD-1 signaling inhibition by IMM2510



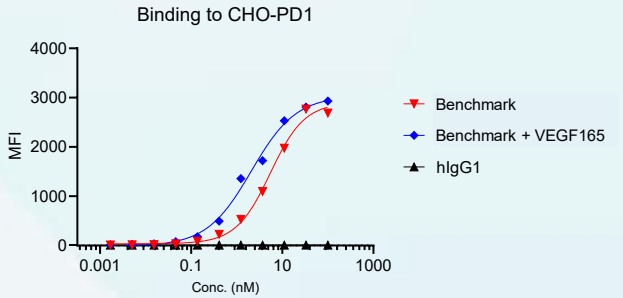
	IMM2510	IMM2510+VEGF165
IC50	0.3697	0.1429

- IMM2510 demonstrates enhanced blockade of PD-1/PD-L1 signaling in the presence of VEGF (cooperative effect)

Binding of IMM2510 and benchmark antibody to CHO-PDL1 and CHO-PD1 cells, respectively, is enhanced with VEGF



	IC50
IMM2510	2.035
IMM2510+VEGF165	1.013



	IC50
Benchmark	5.464
Benchmark +VEGF165	2.094

- Competitor benchmark antibody* and IMM2510 demonstrate similar shift in binding affinity to PD-1 and PD-L1, respectively, in the presence of VEGF

*Competitor benchmark antibody from most clinically advanced PD1xVEGF bispecific program

Palverafusp alfa (IMM2510) (VEGF × PD-L1) + chemo in 1L NSCLC

IMM2510 development strategy prioritizes 1L NSCLC

- We have reported initial preliminary data recently: the ORR is 62% (13/21); notably, in patients with squamous NSCLC, the ORR reached 80% (8/10). The majority of efficacy evaluable patients had only one tumor assessment at data cut-off of July 1st, 2025

- IMM2510's safety profile supports further clinical development, with: No dose-limiting toxicities observed in the 33 safety evaluable patients
  - No treatment-related adverse events (TRAE) leading to dose reduction or death
  - Only one TRAE leading to drug discontinuation
  - Most common Grade 3+ TRAEs were hematologic, with uncommon clinical sequelae

Expect to present updated safety and efficacy data at a future medical conference

Source: Company Data; the clinical data is as of July 1st, 2025. Study is ongoing, data subject to change.

Palverafusp alfa (IMM2510) (VEGF × PD-L1) + chemo in 1L NSCLC

Preliminary Efficacy Data is Comparable to Competitors

	Palverafusp alfa (IMM2510)[1]		Ivonescimab[2]		SSGJ-707[3]	
	1L Non-sq	1L squamous	1L Non-sq	1L squamous	1L Non-sq	1L squamous
Phase	Phase II		Phase II		Phase II	
Data publishing time	July 2025, Company Annoucement		ASCO 2022, 2023		2025 JPM	
Dosage	2510 10 mg/kg Q3W+pemetrexed +carboplatin	2510 10 mg/kg Q3W+paclitaxel + carboplatin	Ivonescimab 10 or 20mg/kgQ3W+pemetrexed +carboplatin	Ivonescimab 10 or 20mg/kgQ3W+paclitaxel + carboplatin	707 10 mg/kg Q3W+pemetrexed +carboplatin	707 10 mg/kg Q3W+paclitaxel + carboplatin
N (efficacy evaluable)	11	10	72	63	12	16
ORR	**46%**	**80%**	**54.2%**	**71.4%**	**58.3%**	**81.3%**

Notes:
1. The majority of efficacy evaluable patients had only one tumor assessment at data cut-off of July 1st, 2025
2. 3. Soochow Securities Research

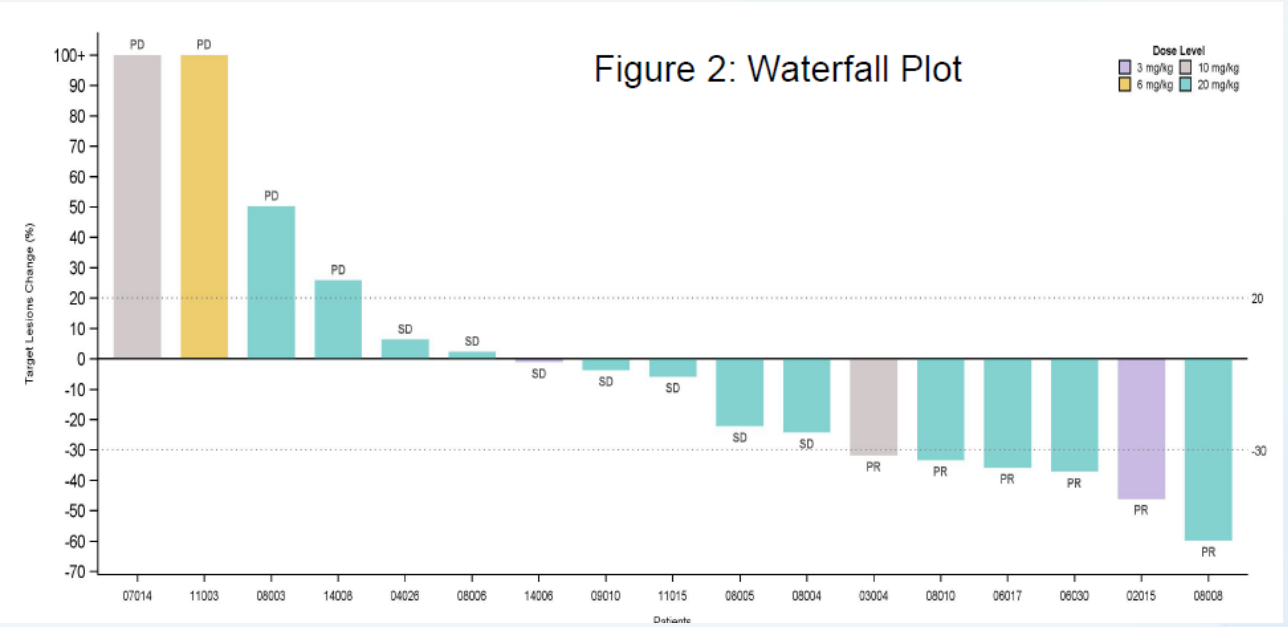
Palverafusp alfa (IMM2510) (VEGF × PD-L1) Monotherapy

The data of IMM2510 momotherapy for I/O treated advanced sq- NSCLC

- ORR was **35.3%** (6/17) and DCR was 76.5% (13/17).
- Median DoR was 7.59 months (95% CI: 4.07–NA); median PFS was **9.4 months** (95% CI: 1.87–NA).
- ORR of docetaxel-resistant population(n=5) was **40%** (2/5)
- IMM2510 momotherapy was well tolerated

WCLC 2025

Best Percentage Change from Baseline in Target Lesion



Based on the above results, a Phase III clinical trial application for IMM2510 in combination or without docetaxel for the treatment of sq-NSCLC after I/O therapy failure is expected to be submitted soon.

Data cut off date Aug 6, 2025. WCLC 2025 poster. Study is ongoing, data subject to change.

Palverafusp alfa (IMM2510) (VEGF × PD-L1) Monotherapy

Efficacy summary of monotherapy for I/O treated advanced sq- NSCLC

Company	ImmuneOnco[1]	Henlius[2]	Innovent [3]	
Product	Palverafusp alfa (IMM2510)	HLX43	IBI363	
Target (Modality)	PD-L1 /VEGF (Bispecific)	PD-L1 ADC	PD-1 /IL-2$^{\alpha}$ (Bispecific)	
Clinical ID	NCT05972460	NCT06115642	NCT05460767	
Patient Population	Late-stage/advanced metastatic sq-NSCLC	Late-stage/advanced metastatic sq-NSCLC	Late-stage/advanced metastatic sq-NSCLC	
N (efficacy evaluable)	17	28	27	30
Prior Lines ≥2	64.7%	73.2%	64.3%	67.7%
I/O treated	100%	89.3%	100%	96.8%
Dosage	3mg/KgQ3W； 6mg/kgQ3W ； 10mg/kgQ3W; 20 mg/kg Q3W	2.0mg/kg Q3W； 2.5 mg/kg Q3W	1mg/Kg Q2W； 1.5mg/Kg Q3W	3mg/Kg Q3W
ORR	35.3%	28.6%	25.9%	36.7%
DcR	76.5%	82.1%	66.7%	90%
mPFS	9.4	Undisclosed	5.5	9.3
mDoR	7.59	Undisclosed	10.2	NR
mOS	NR	Undisclosed	15.3	NR

Note： AK112 combined with docetaxel for the treatment of immune-resistant NSCLC has entered Phase III clinical trials, but efficacy data for AK112 as a monotherapy for immune-resistant NSCLC has not been published.

Source: 1. WCLC 2025 presentation 2.WCLC 2025 presentation ; 3. ASCO 2025 presentation;

Palverafusp alfa (IMM2510) (VEGF × PD-L1) Monotherapy

Safety summary of monotherapy for I/O treated NSCLC

Category	IMM2510 [1] (3mg/kg Q3W； 6mg/kg Q3W ； 10mg/kg Q3W; 20 mg/kg Q3W) Phase I (n=23) [3]	HLX43 [2] Phase I (2.0mg/kg； 2.5 mg/kg) (n=56)[1]	IBI363 [3] Phase I (1mg/kg Q2W ;1.5mg/kg Q3W) (n=62) [2]	IBI363 [3] Phase I (3mg/kg-Q3W) (n=57) [2]
TRAEs	100%	100%	93.5%	96.5%
Grade ≥3 TRAEs	43.5%	46.4%	17.7%	43.9%
Severe TRAEs	17.4%	Undisclosed	21%	40.4%
TRAEs Leading to Treatment Discontinuation	4.3%	8.9%	6.5%	7.0%
irAE	13% (1 Grade 3 rash, 1 Grade 2 elevated bilirubin, 1 Grade1 rash)	21.4% immune-related pneumonitis (14.3%)	**Possible irAEs**: Arthralgia ~40% Rash ~25% Hypothyroidism ~35% Hyperthyroidism ~10% Elevated alanine aminotransferase (ALT) ~15% Elevated aspartate aminotransferase (AST) ~15% Hyperglycemia ~10% Elevated bilirubin ~10%	**Possible irAEs**: Arthralgia ~65% Rash ~55% Hypothyroidism ~40% Hyperthyroidism ~30% Elevated alanine aminotransferase (ALT) ~25% Elevated aspartate aminotransferase (AST) ~20% Hyperglycemia ~20% Elevated bilirubin ~15%

1. IMM2510 momotherapy for I/O treated advanced sq- NSCLC . Among 23 patients, 2 received 3 mg/kg, 1 received 6 mg/kg, 4 received 10 mg/kg, and 16 received 20 mg/kg (RP2D).
2.HLX43 2.0 mg/kg; 2.5 mg/kg, monotherapy for advanced/metastatic NSCLC refractory to SOC
3.IBI363 monotherapy for I/O treated NSCLC, including squamous and non-squamous.

Source: 1. WCLC 2025 presentation 2.WCLC 2025 presentation ; 3. ASCO 2025 presentation;

Palverafusp alfa (IMM2510)(VEGF × PD-L1)

Global Collaboration

On August 1, 2024, we reached a license and collaboration agreement with Axion Bio, Inc. (a wholly-owned subsidiary of Instil Bio (TIL US)) (formerly known as SynBioTx Inc.), pursuant to which Axion 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.

We have received **US$30 million** as of August 26, 2025

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**

Tazlestobart (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

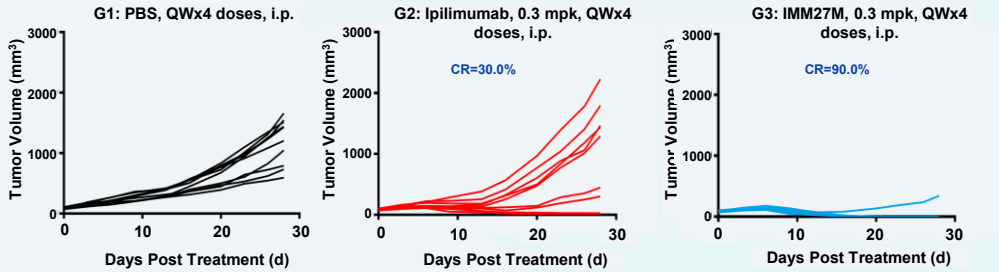
- **Blocking the interaction** between CTLA-4 and CD80/CD86, and thus enhancing immune responses of T cells to tumor antigens
- Inducing enhanced immune responses targeting CTLA-4 **overexpressed T_{reg} cells**
- Promoting **T_{reg} depletion**, thus improving T-cell antitumor response to kill tumor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

Currently Approved CTLA-4 Antibody with Unmodified Fc:

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

Stronger In Vivo Antitumor Effects and Preliminary Ph1 data

Efficacy Study in Colon Cancer (MC38) Syngeneic Mouse Model

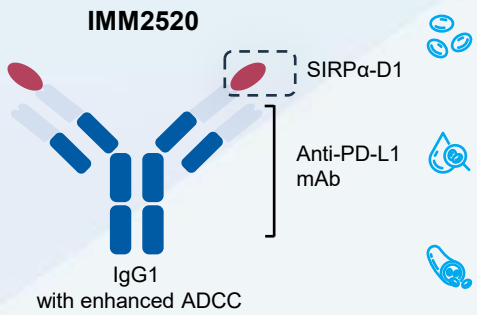


Clinical Development Plan

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

IMM2520 (CD47×PD-L1)

Overview



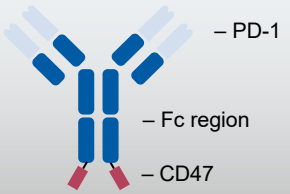
- A CD47 and PD-L1 dual-targeting bispecific molecule for the treatment of solid tumors
- Unique structure to **avoid RBC binding**
- Engineered ADCC-enhanced IgG1 Fc fragment to **fully activate macrophages** and **induce enhanced ADCP and ADCC activity**

Competition Landscape

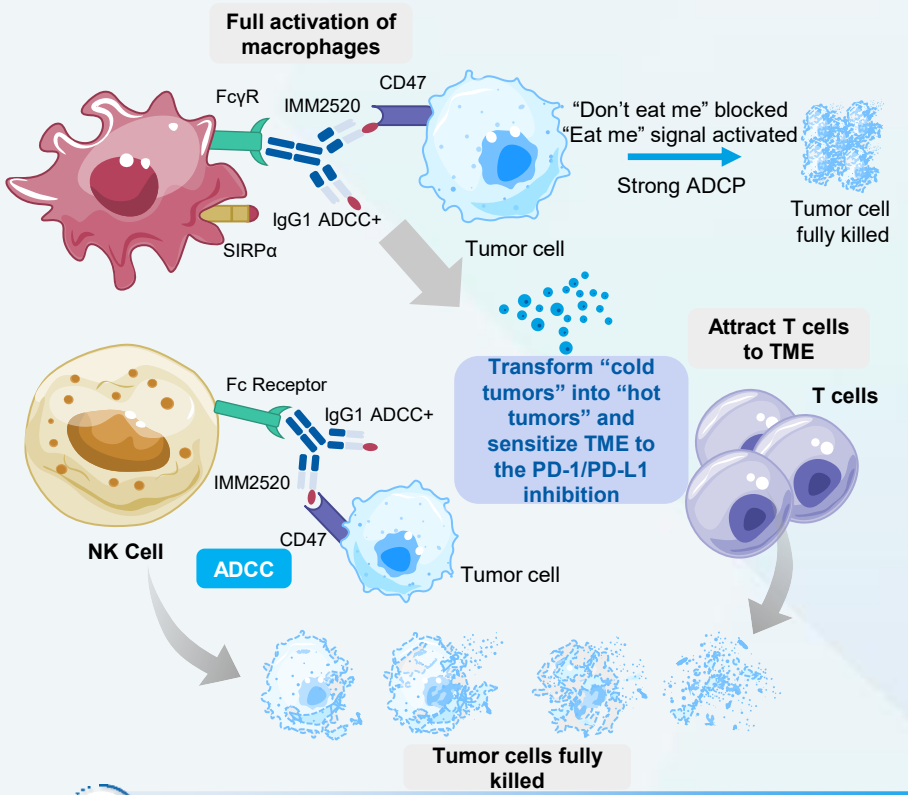
Other 9 CD47 and PD-1/PD-L1 bispecific molecules under clinical development

- ✓ 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

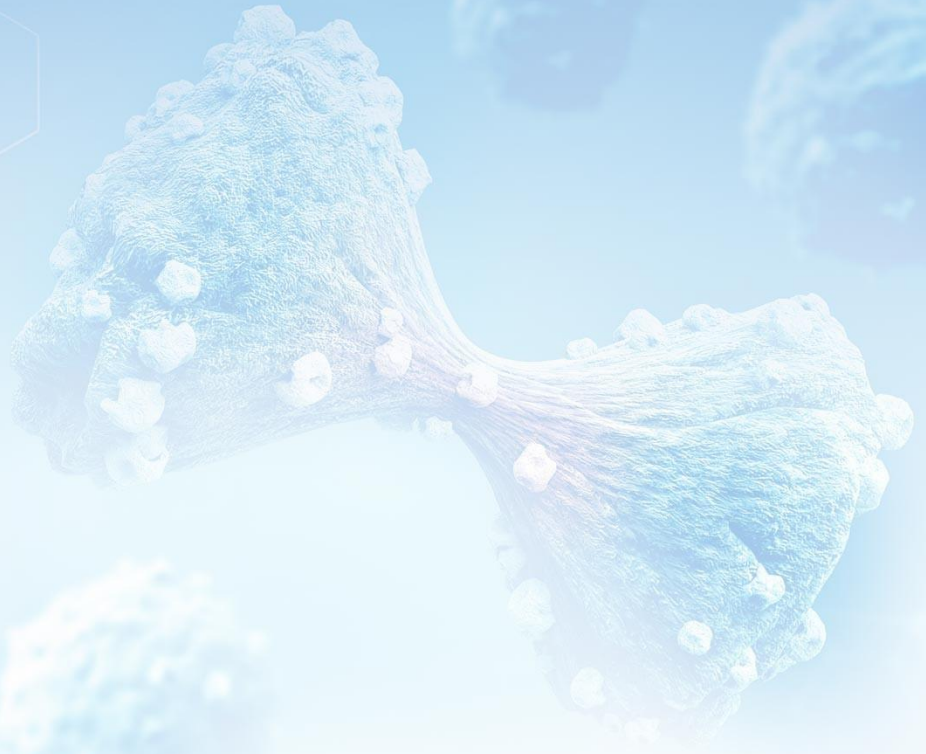


Due to the crosstalk among macrophages, NK cells and T cells, IMM2520 is able to unleash significant synergistic effects

宜明昂科
ImmuneOnco

SECTION 3

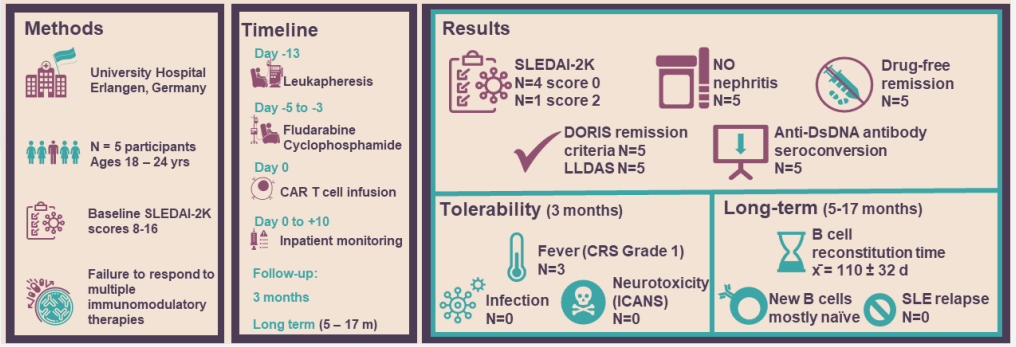
Non-Oncology Programs



Amulirafusp alfa(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.

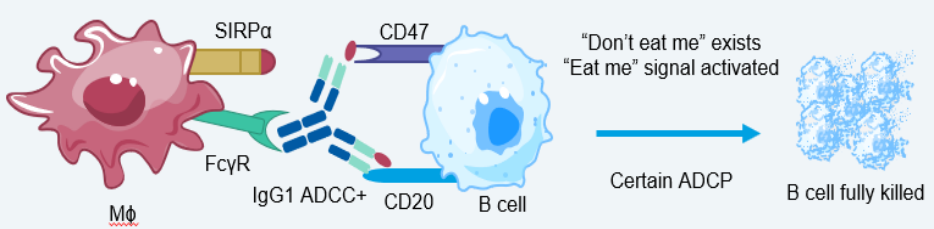


RITUXAN® (rituximab, CD20) was approved by FDA in 2006 for the treatment of **rheumatoid arthritis (RA)**;

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

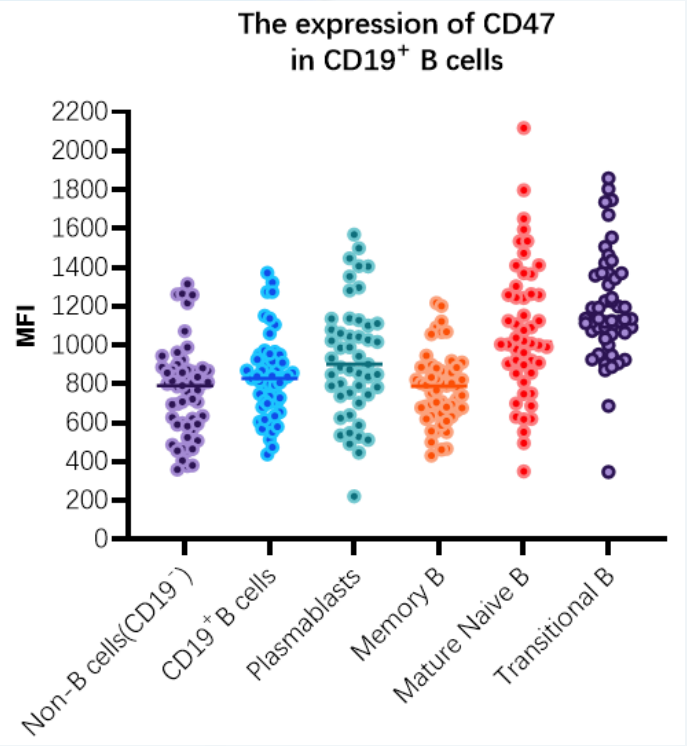


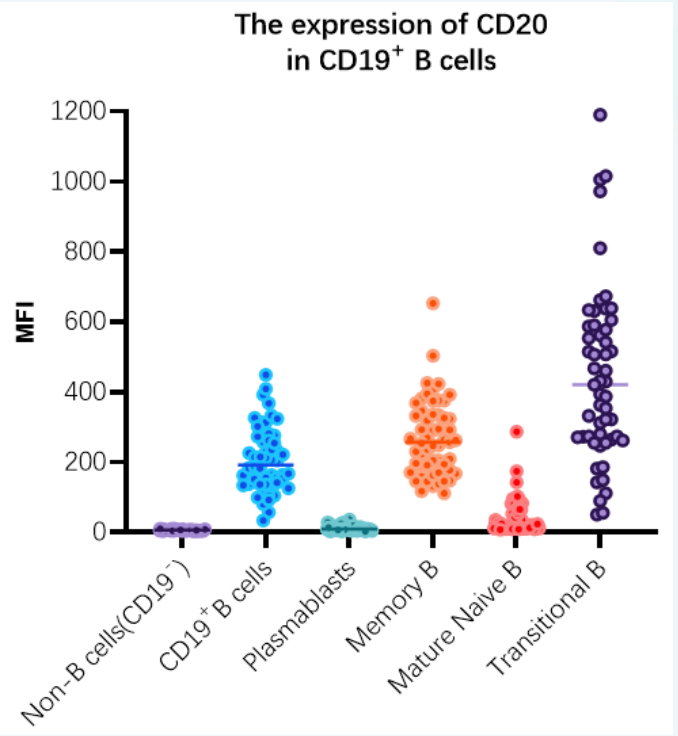
	IMM0306	SIRPα-Fc	CD47 mAb IgG4	Rituximab
ADCP	+++	+++	+	++
ADCC	+++	+	+	++
CDC	++	No	N/A	+++
Induction of hemagglutination	**No**	No	Yes	Not relevant

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

Amulirafusp alfa (IMM0306) (CD47xCD20/mAb-Trap)- Potential in Autoimmune Diseases

Dual Targeting of CD20 and CD47 Enhances Cell Lineage Coverage, Improving Therapeutic Potential





In vitro analysis of SLE patient blood revealed:

- CD47: High expression across B-cell subtypes (including plasmablasts), with no significant variation.
- CD20: Minimal expression in plasmablasts and mature naïve B-cells vs other B-cell subsets.

Amulirafusp alfa (IMM0306) (CD47xCD20/mAb-Trap)- Potential in Autoimmune Diseases

IND Approved in China

Systemic lupus erythematosus (SLE)
Phase Ib

Neuromyelitis optica spectrum disorder (NMOSD)
Phase Ib

Lupus nephritis (LN)
Phase II

IND planned in US & China

Subcutaneous formulation
China: Phase II
US: Phase II

Multiple sclerosis (MS)
China: Phase II
US: Phase Ib/II

Myasthenia gravis (MG)
China: Phase II
US: Phase Ib/II

Amulirafusp alfa (IMM0306) (CD47xCD20/mAb-Trap)- Potential in Autoimmune Diseases

Global SLE Population

7.8 Million [1]

Global MS Population

2.8 Million [2]

Global LN Population

2.7 Million [3]

Global MG Population

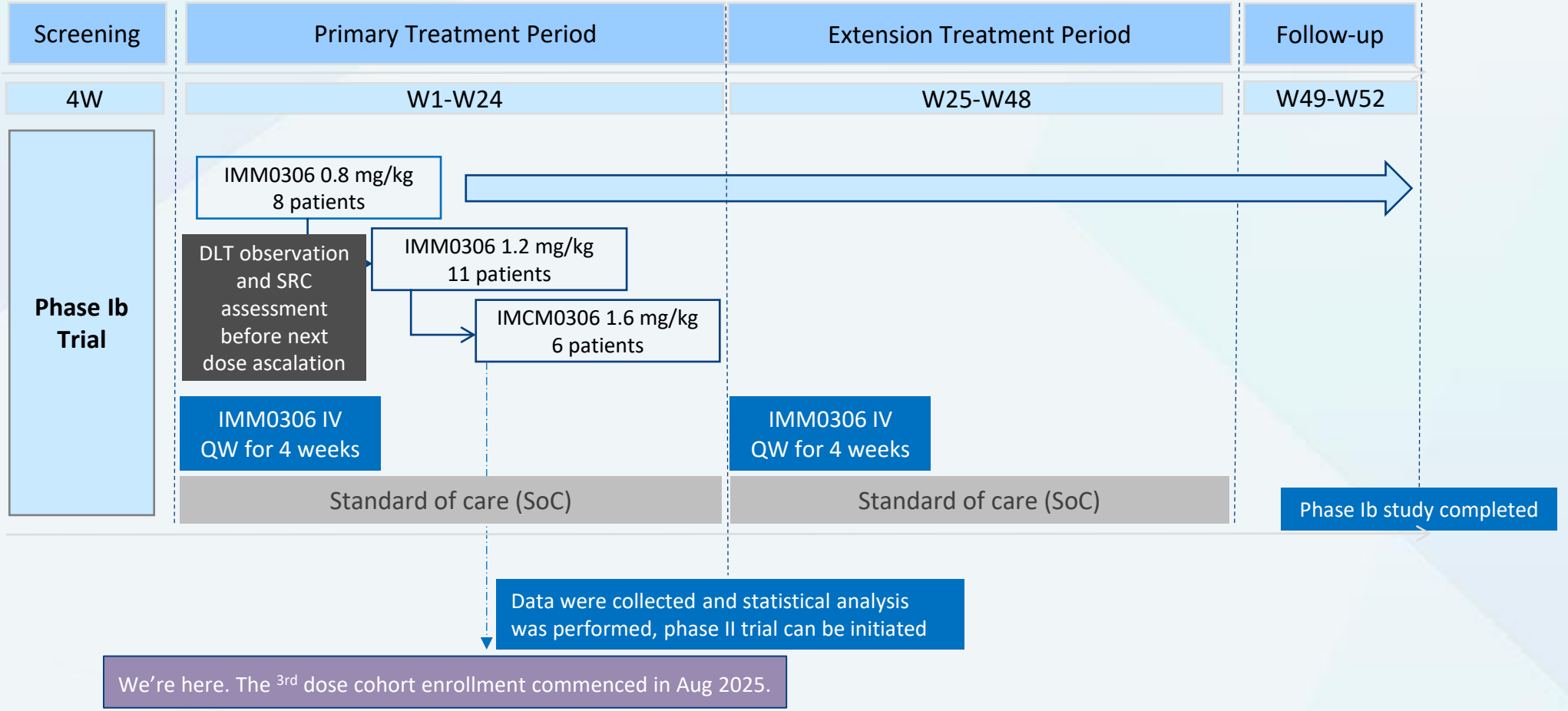
1.09 Million [4]

Global NMOSD Population

0.17 Million [5]

1. Frost & Sullivan, global SLE population in 2020
2. MSIF data
3. Frost & Sullivan, global LN population in 2020
4. Frost & Sullivan, global MG population in 2020
5. Frost & Sullivan, global NMOSD population in 2020

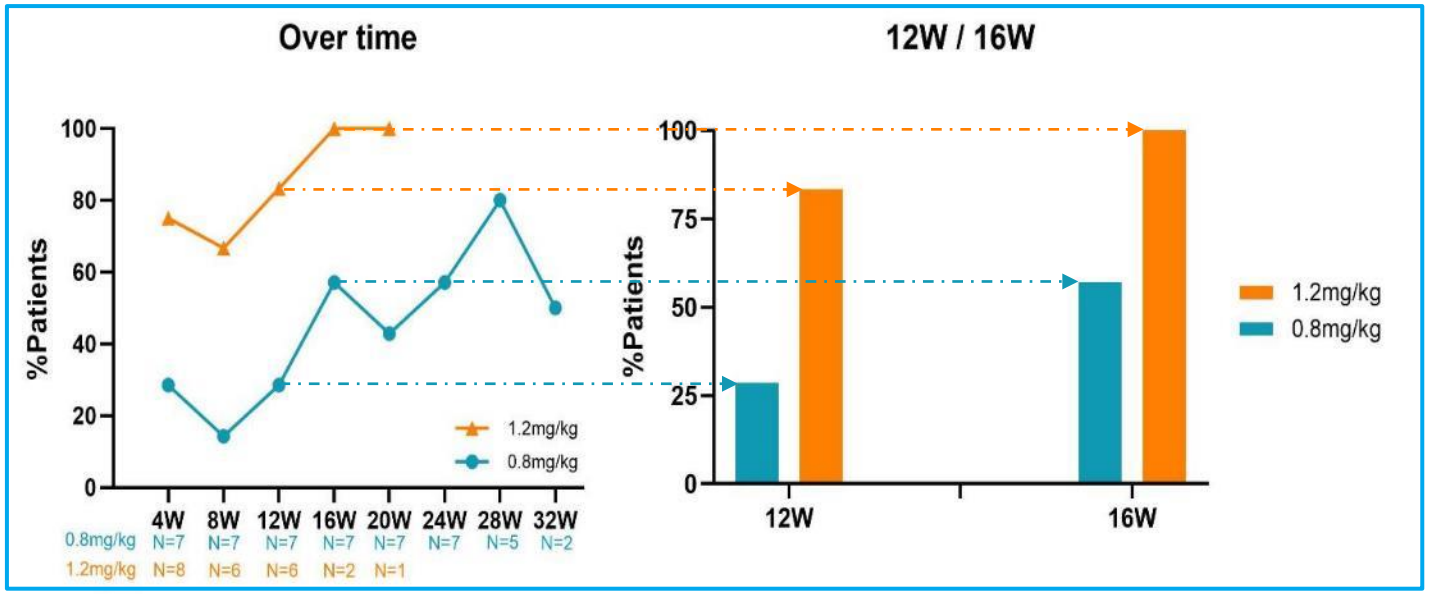
Amulirafusp alfa(IMM0306) (CD47xCD20/mAb-Trap) SLE Phase Ib Trial Design



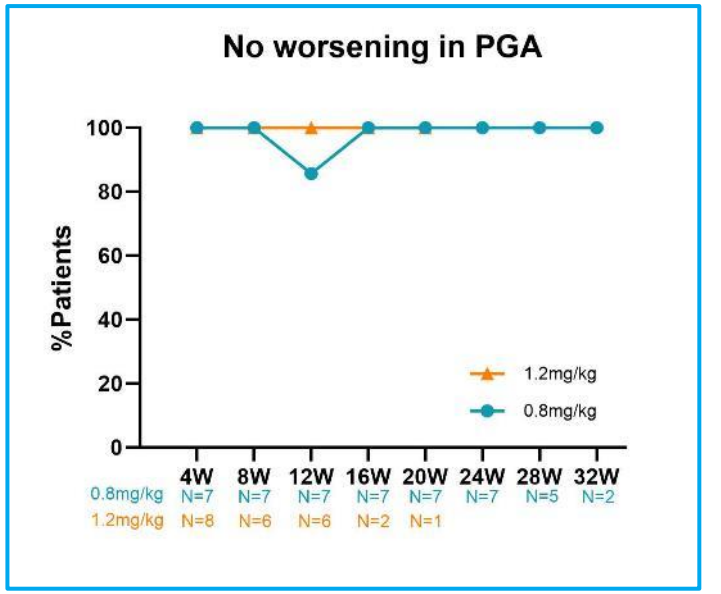
GC: glucocorticoids. QW: Once a week. DLT: dose limiting toxicity. SRC: safety review committee.

Amulirafusp alfa (IMM0306) Shows Rapid, Dose-Dependent SLEDAI-2K Improvement

Proportion of patients with ≥ 4 points reduction from baseline in SLEDAI-2K score



No worsening in PGA

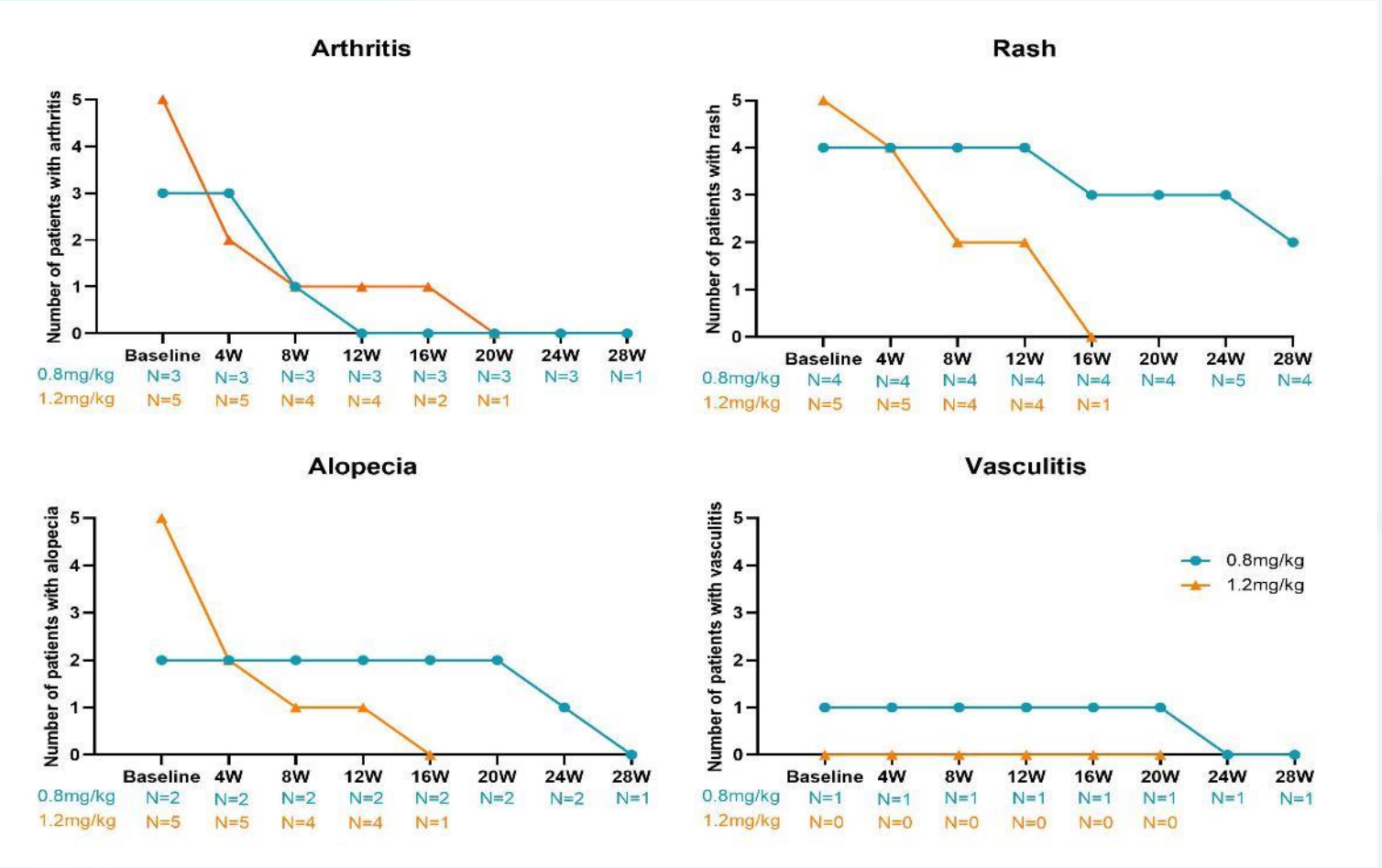


The percentage of patients with a reduction in SLEDAI-2000 by ≥4 was 87.5% (7/8) in the 1.2 mg/kg cohort

Data cut-off date July 1, 2025.

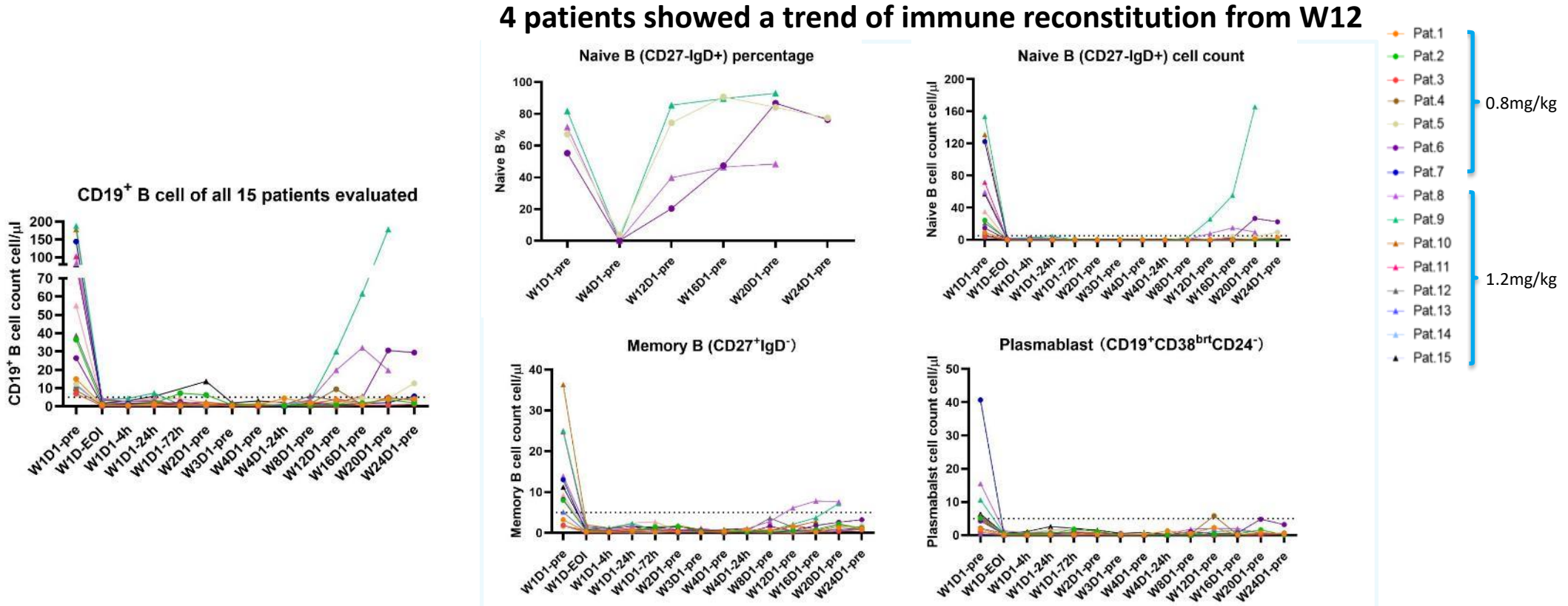
The patients included in the efficacy analysis had completed ≥4 doses and at least one efficacy evaluation (7 patients in 0.8mg/kg cohort, 8 patients in 1.2mg/kg cohort)

Amulirafusp alfa(IMM0306)- Situation of Arthritis, Rash, Alopecia and Vasculitis are Improved



Data cut-off date July 1, 2025.

Amulirafusp alfa(IMM0306)- Efficient and Sustained B-cell Depletion with Immune Reconstitution Observed



In Patients 5, 6, 8, and 9, B-lineage cells rebounded between Weeks 12 and 24. Notably, the reconstituted B-cell pool was predominantly composed of naïve B cells, whereas memory B cells continued to decline, and plasmablasts persisted at very low levels. These findings suggest that IMC-002 treatment led to a reconstitution of B-cell lineages toward a predominantly naïve phenotype.

宜明昂科 ImmuneOnco

Amulirafusp alfa(IMM0306)- Shows Best-in-disease Potential in SLE

	Amulirafusp alfa (IMM0306)	Mosunetuzumab[2]	Telitacicept[3]	Belimumab[4]
Target	CD47xCD20	CD3xCD20	BLyS, APRIL	BLyS
≥4 points reduction from baseline in patients	87.5% (7/8) Week4-20 [1]	66.7% (4/6) Week52	77.8% (49/63) Week48[3.1]	46.5% (127/273) Week52[4.1]
B-cell depletion right after infusion	Yes	n.a.	n.a.	n.a.
Cytokine release syndrome	0	26.7% (4/15)	n.a.	n.a.
Dose step-up	Not required	Required	Not required	Not required
Stage	Phase Ib	Phase Ib	Approved in China	Approved by FDA

n.a. not available
1. 1.2 mg/kg. 2. Chindalore et al. EULAR2025 POS1160. . 2.1 Base line SLEDA! score ≥ 8
3. Wu et al. Ann Rheum Dis 2023;0:1–13. BLyS: B lymphocyte stimulator; APRIL: a proliferation inducing ligand. 3.1 Approved dose (160 mg).
4. Furie et al. Arthritis Rheum. 2011 Dec;63(12):3918-30. 4.1 Approved dose (10mg/kg), base line SLEDAI score ≥ 6.

Amulirafusp alfa(IMM0306)(CD47xCD20/mAb-Trap) – Global Deals in the Area

Since 2024, the global business development of innovative BsAb and TsAb in the autoimmune field has been booming

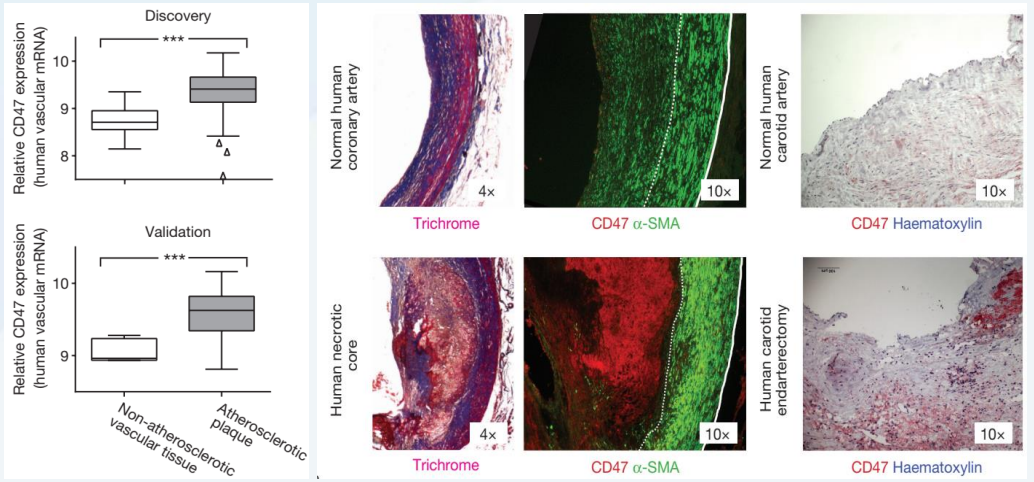
Product	Licenser	Licensee	Upfront and milestone payment	Time	Clinical trial progress
DR-0201 （CD20-directed BsAb MCE [1]）	Dren Bio	Sanofi	Upfront payment of $600 million+ milestone payment of $1.3 billion	2025.3	PhI study in B-NHL patients and is expanding into various autoimmune indications
CN201 (CD3×CD19 BsAb)	Curon Biopharma	MSD	Upfront payment of $700 million+ milestone payment of $600 million	Aug 2024	R/R NHL: PhI; R/R ALL： PhIb/II Autoimmune indications have not yet entered the clinical stage
CMG1A46 (CD3×CD19×CD20 TsAb)	Chimagen Biosciences	GSK	Upfront payment of $300 million+ milestone payment of $550 million	Oct 2024	Hematologic malignancies: PhI/II GSK plans to initiate a PhI trial for lupus in 1H 2025
GB261 (CD20×CD3 BsAb）	Genor Bio	TRC 2004	A double digit million US dollars upfront payment+ up to $443 million in milestone payments	Aug 2024	Completed PhI/II B-NHL（DLBCL&FL） Autoimmune indications have not yet entered the clinical stage
CM336 （BCMA×CD3 BsAb）	Keymed Biosciences	Platina	Upfront and near-term payment of $16 million+ up to $610 million in milestone payments	Nov 2024	R/R MM:PhI/II Platina plans to initiate a PhI trial for the first autoimmune indication in 1H 2025
EMB-06 (BCMA×CD3 BsAb)	EpimAb Biotherapeutics	Vignette Bio	Upfront payment of $60 million in cash and equity +up to $575 million in milestone payments	Sep 2024	R/R MM： PhI/II Autoimmune indications have not yet entered the clinical stage
LBL-051 (CD3 × BCMA ×CD19 TsAb）	Leads Biolabs	Oblenio	Upfront and near-term payment of $35 million +up to $579 million in milestone payments	Nov 2024	IND enabling
HBM7020 (BCMA×CD3)	Harbour Biomed	Otsuka Pharmaceutical	Upfront and near-term payment of $47 million +up to $623 million in milestone payments	June 2025	IND enabling

Source: announcements and news of the above companies

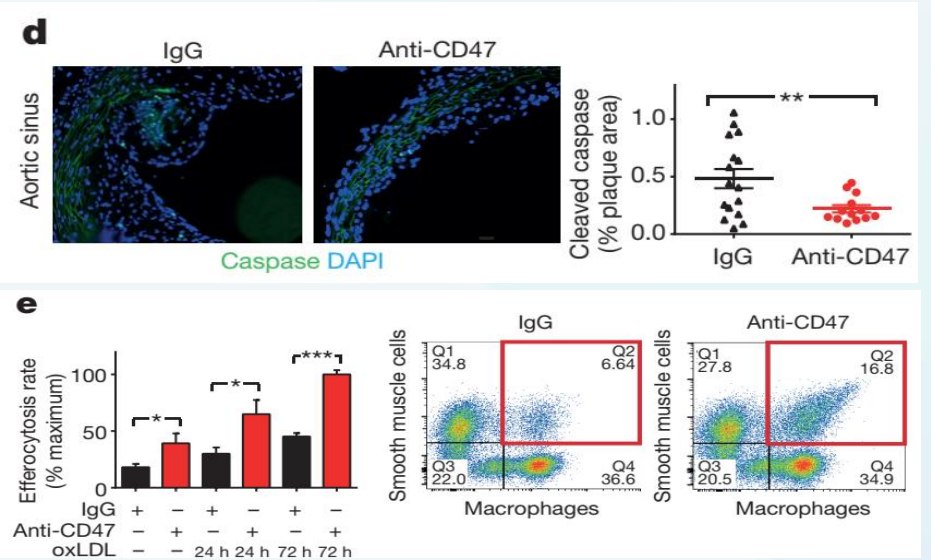
Timdarpacept (IMM01) Has Strong Potentials in treating atherosclerosis

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

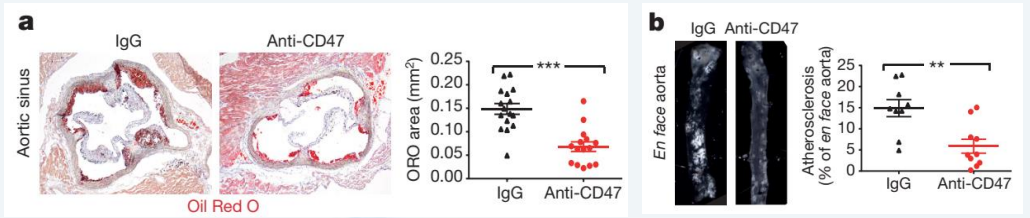
CD47 is highly expressed in human atherosclerotic plaque

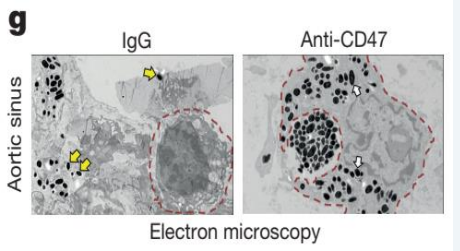


By blocking the CD47 signal, macrophages can phagocytose the atherosclerotic plaque in rat vessel



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





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
- The PhII in Austria initiated in June 2025
- Bitterroot Biowas co-founded by Irv Weissmanv, Nick Leeper, John C. Martin and Lou Lange

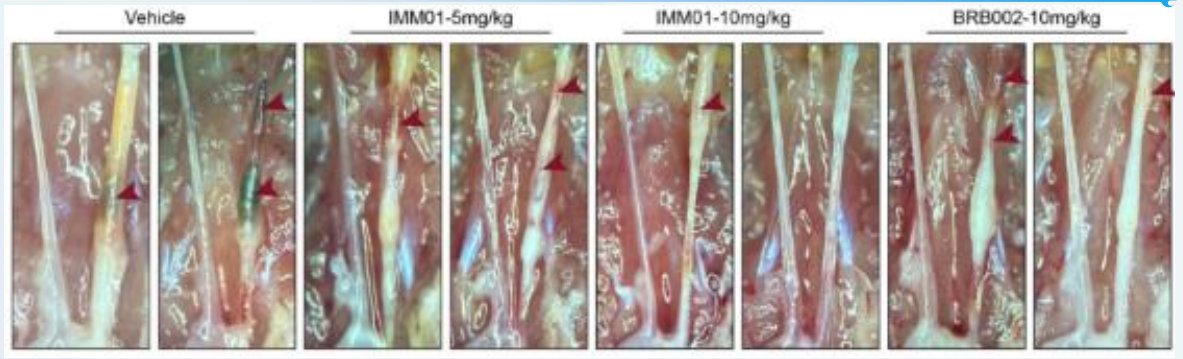
Source: Yoko Kojima, et al., 86 , Nature, Vol 536, Augest 2016;

宜明昂科 ImmuneOnco

)

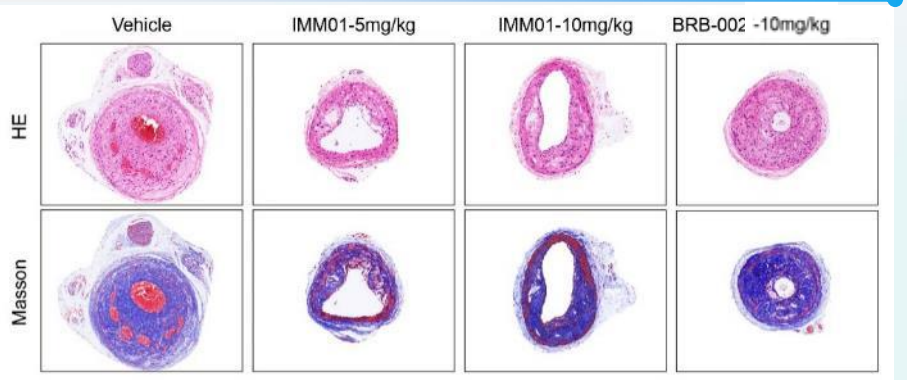
Timdarpacept (IMM01) – Animal study Results Demonstrated Strong Potential in Treating Atherosclerosis

Representative gross pictures of the left common carotid artery

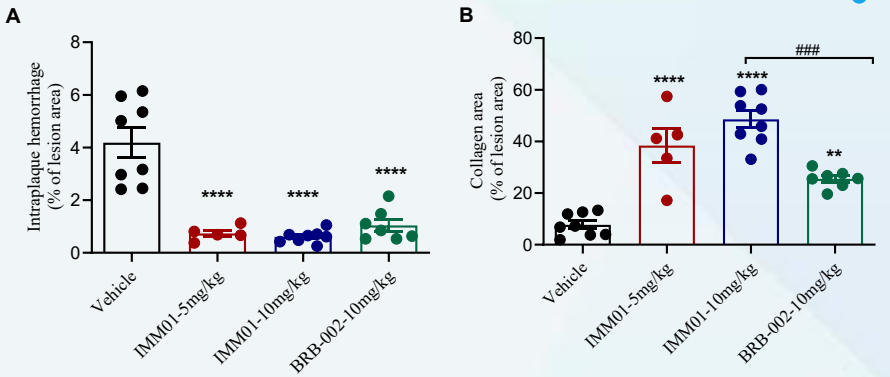


Red arrow: hemorrhage site

Statistically analysis of the results from HE staining (n=5-8/group)



statistically analysis of the results from Massion staining (n=5-8/group)



****p<0.0001, **p<0.01: VS Vehicle; ###p<0.001: IMM01(10mg/kg) VS BRB-002(10mg/kg) (One-way ANOVA)

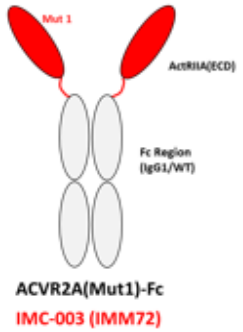
Plaque vulnerability model in a hCD47/hSIRPα apoE$^{-/-}$ mouse:

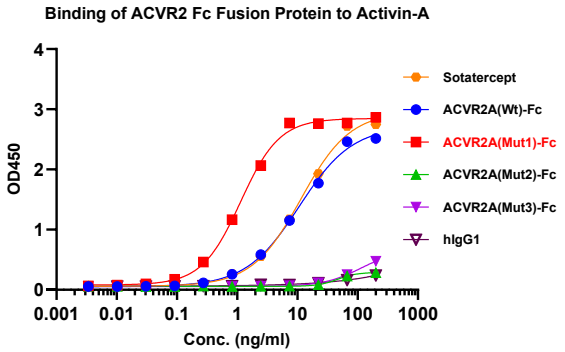
- Compared to vehicle control (model group), IMM01 (5mg/kg, 10 mg/kg) or BRB-002(10 mg/kg) treatment **significantly reduced intraplaque hemorrhage**; and the increased collagen area stabilized the atherosclerotic plaques and **reduced the risk of bleeding.**
- **IMM01 showed better efficacy than BRB-002.**

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

Preclinical Results

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

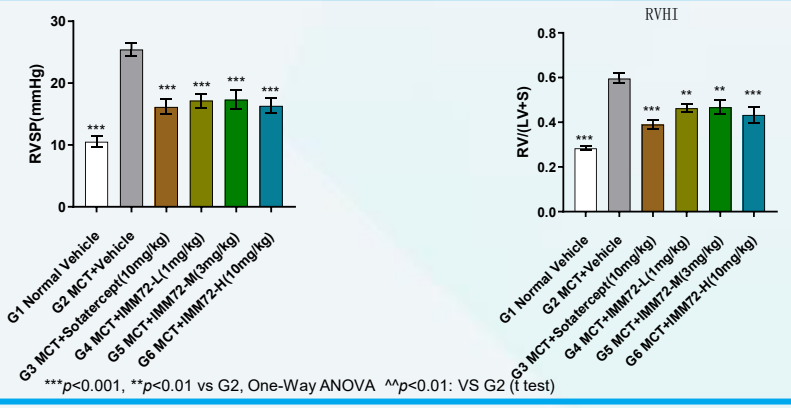




	Sotatercept	ACVR2(WT)-Fc	ACVR2(Mut1)-Fc
EC50	11.43	10.70	1.140

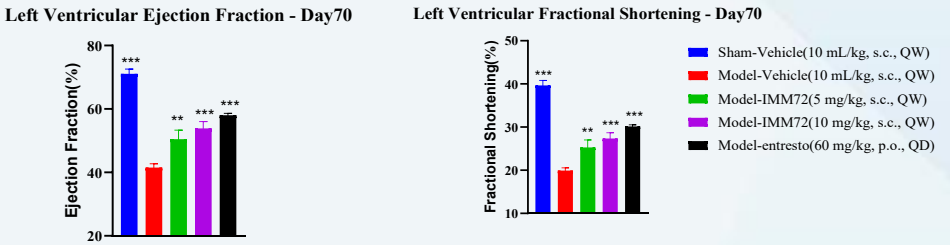
	IMC-003 (IMM72)	Sotatercept
Company	ImmuneOnco	MSD
Structure	ACVR2A-Fc (point mutation)	ACVR2A-Fc
Affinity	Comparable	Comparable
Binding (ELISA)	Stronger (≥7 times)	Medium
Blocking	Stronger	Medium
In vivo efficacy	Stronger	Medium

IMC-003 exhibits good efficacy in MCT induced PAH model



***p<0.001, **p<0.01 vs G2, One-Way ANOVA ^^p<0.01: VS G2 (t test)

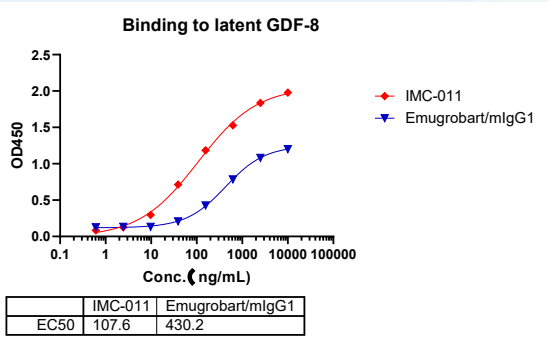
IMC-003 exhibits good efficacy in a TAC model of heart failure



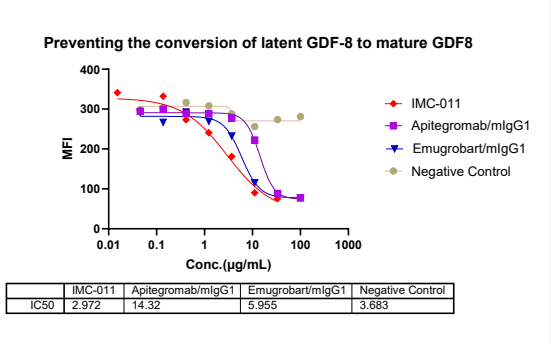
**p<0.01, ***p<0.001: VS G2(One-way ANOVA/Dunnett's)

IMC-011 (IMM91, pro/latent GDF-8 mAb)

IMC-011 shows excellent binding and blocking activity



	IMC-011	Emugrobart/mIgG1
EC50	107.6	430.2



	IMC-011	Apitegromab/mIgG1	Emugrobart/mIgG1	Negative Control
IC50	2.972	14.32	5.955	3.683

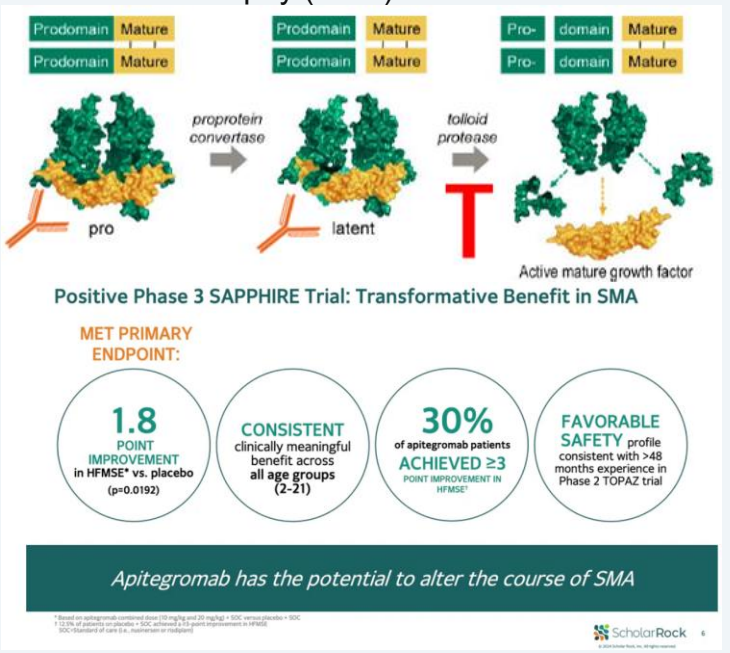
Source:

Scholar Rock Reports Positive Phase 2 EMBRAZE Trial Results Demonstrating Statistically Significant Preservation of Lean Mass with Apitegromab During Tirzepatide-Induced Weight Loss - Scholar Rock, Inc.

https://investors.scholarrock.com/static-files/1917b515-7a43-49f6-a6f2-eb02f52a71c9

Scholar Rock's Apitegromab (SRK-015) has demonstrated druggability

- SRK-015: A fully human antibody that binds to both pro- and latent myostatin
- Positive results from pivotal Phase III SAPPHIRE trial of Apitegromab in spinal muscular atrophy (SMA).



INVESTORS AND MEDIA

Scholar Rock Reports Positive Phase 2 EMBRAZE Trial Results Demonstrating Statistically Significant Preservation of Lean Mass with Apitegromab During Tirzepatide-Induced Weight Loss

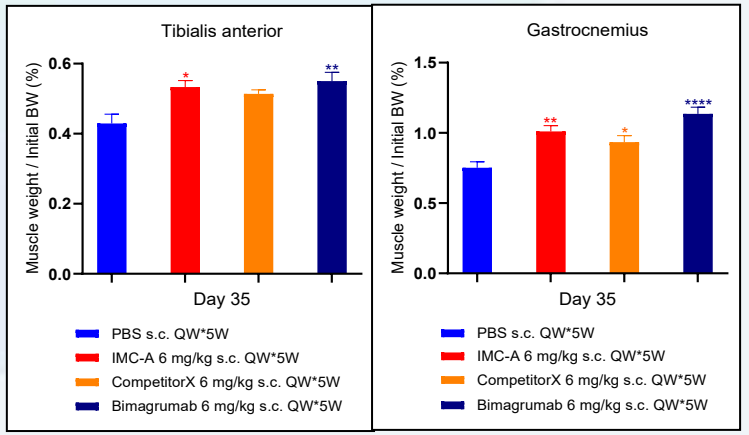
June 18, 2025 PDF Version

	24 Weeks		
	apitegromab 10 mg/kg + tirzepatide (n=43)	placebo + tirzepatide (n=44)	Difference apitegromab vs. placebo
Change in Lean Mass (SE)	-1.6 (0.57) kg -3.4 (1.25) lbs	-3.5 (0.52) kg -7.6 (1.14) lbs	1.9 (0.58) kg 4.2 (1.27) lbs (p=0.001) 54.9% preservation
Total Mass Loss due to Lean Mass Loss (SE) in %	14.6 (3.19) %	30.2 (2.89) %	-15.6 (3.23) %
Change in Fat Mass (SE)	-8.5 (0.85) kg -18.8 (1.87) lbs	-8.0 (0.77) kg -17.7 (1.70) lbs	-0.5 (0.86) kg -1.1 (1.90) lbs
Total Mass Loss due to Fat Mass Loss (SE) in %	85.3 (3.22) %	69.5 (2.93) %	15.8 (3.27) %
Change in body weight (SE) (% change in body weight)	-11.2 (1.21) kg -24.6 (2.65) lbs (-12.3%)	-12.5 (1.09) kg -27.5 (2.41) lbs (-13.4%)	1.3 (1.22) kg 2.9 (2.69) lbs

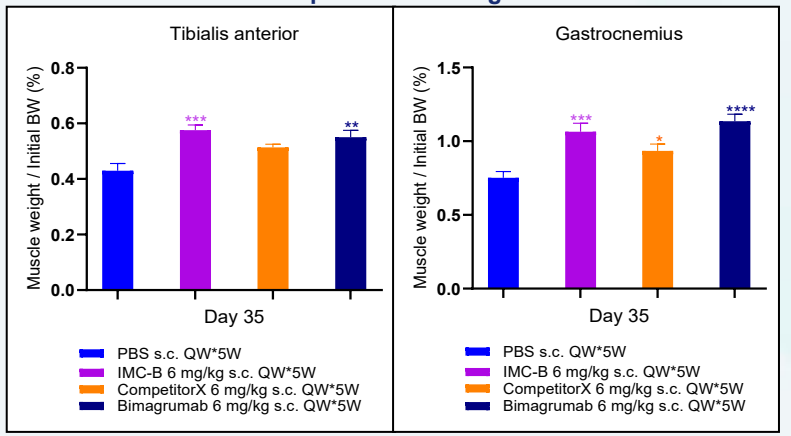
Innovative Molecule Matrix Targeting ActRIIA/B

In CB17-SCID mouse model, our candidates showed significant increase in muscle mass after once-weekly administration for 5 weeks

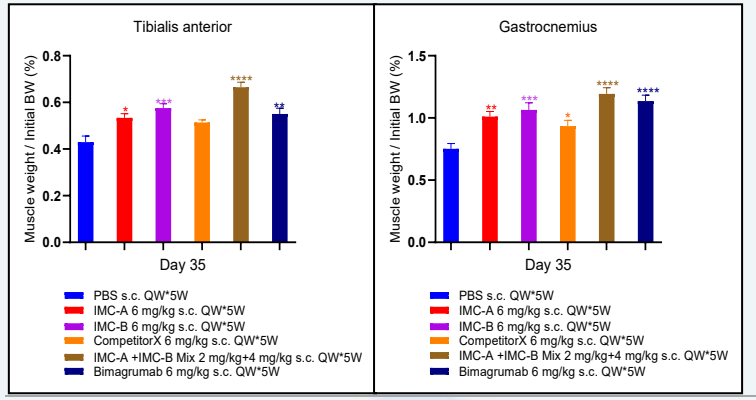
Candidate A's efficacy was better than competitorX



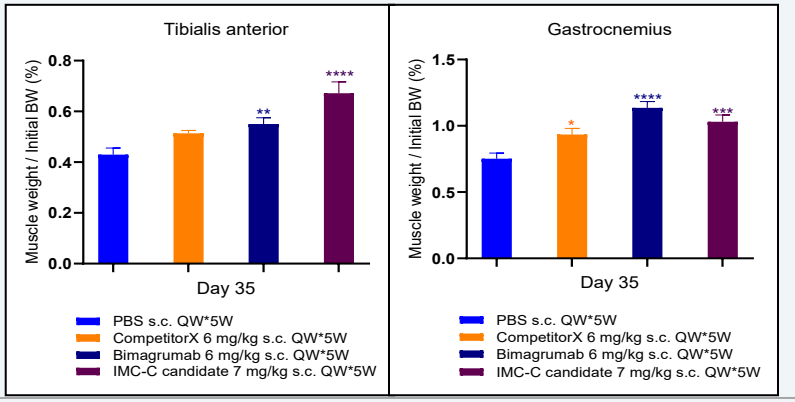
Candidate B's efficacy was superior to competitorX and comparable to bimagrumab



Combo of candidate A and B exhibits the optimal efficacy



Candidate C can significantly increase muscle weight



a, Mean ± SEM; N=6.
b, p value was calculated based on different groups of muscle mass using vehicle group as the control by T-Test. *p<0.05; **p<0.01; ***p<0.001; ****p<0.0001.

*IMC-A, IMC-B, IMC-C represents our candidate A(mAb), candidate B(mAb) and candidate C (*BsAb*) respectively.*

MOA of ActRII mAb

ActRII biology in reducing fat mass while preserving muscle mass

ActRII biology in adipose tissue

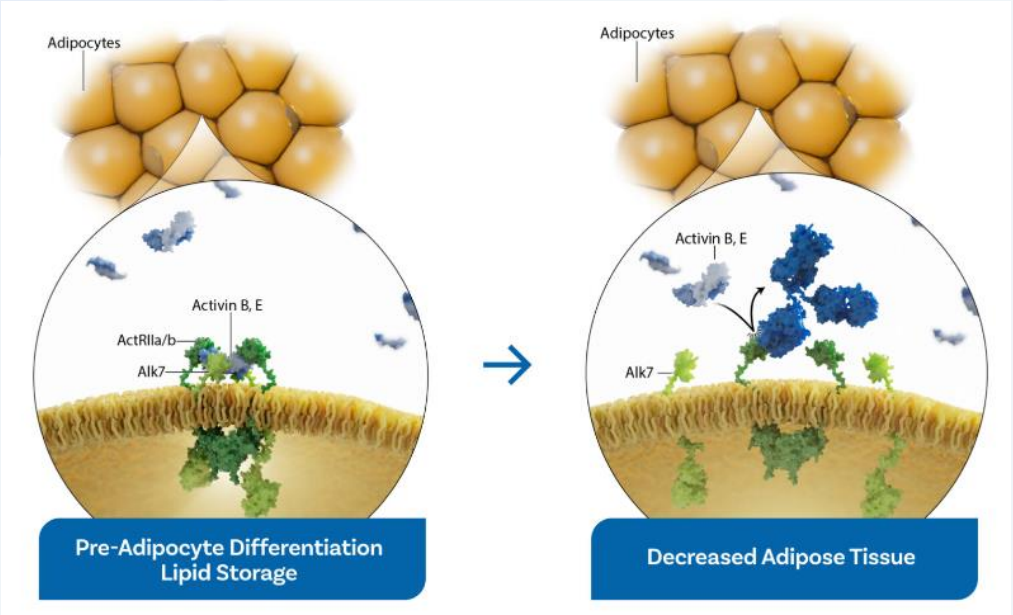
Activin signaling via ActRII receptors directly promotes lipid storage, acting as a key driver of visceral fat accumulation and obesity

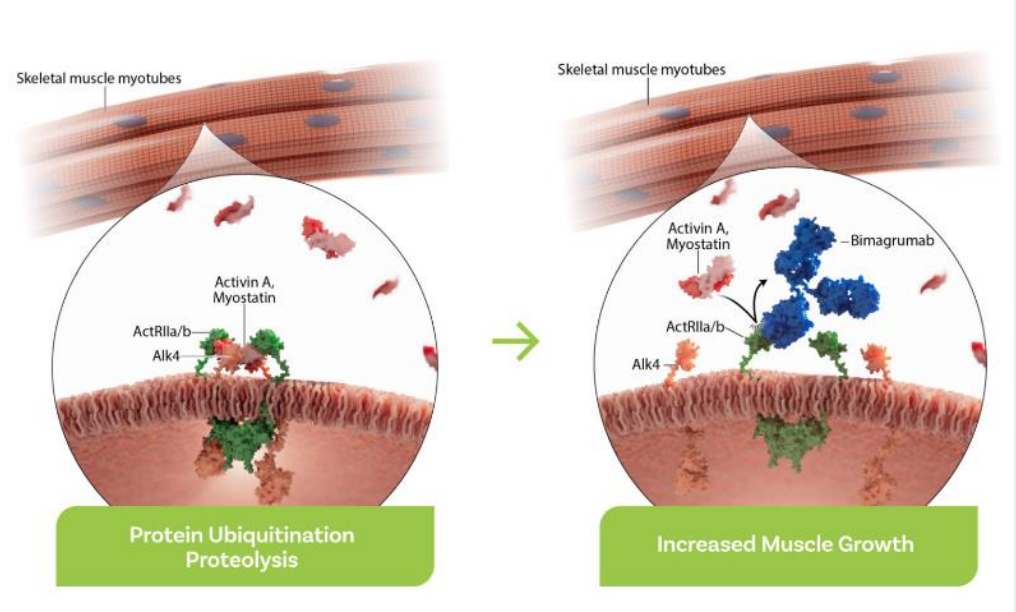
By blocking ActRII signaling in adipose cells, can mobilize and metabolize fat.

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.





Source: : versanisbio.com

宜明昂科
ImmuneOnco

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

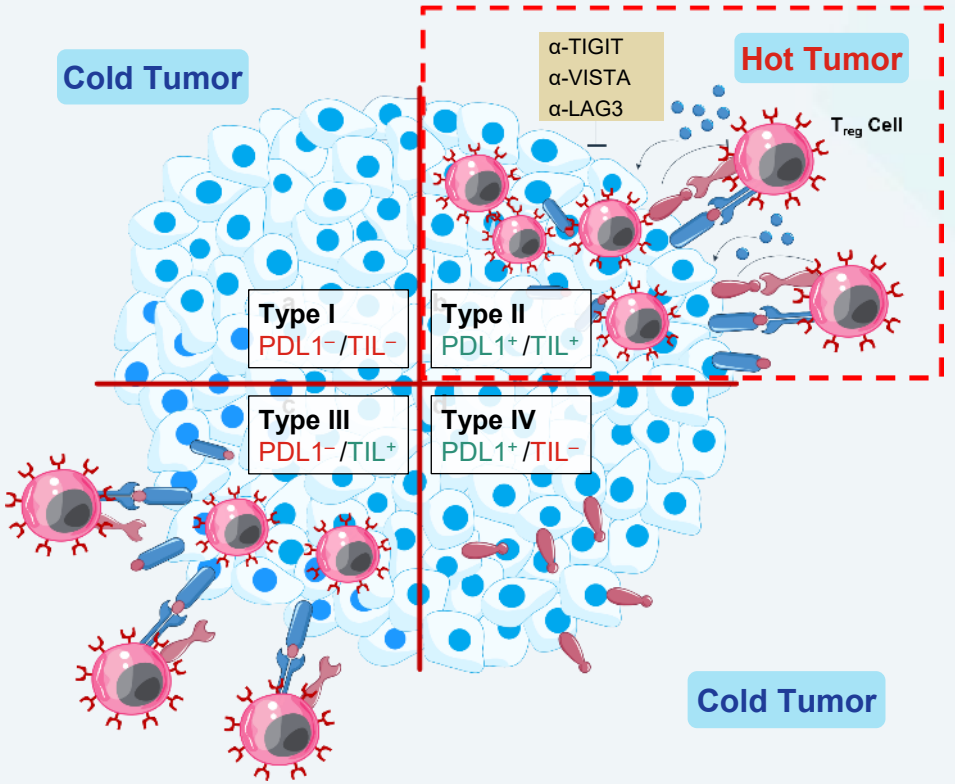
Type I

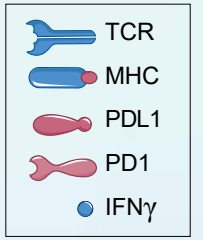
Lack of TILs in TME

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

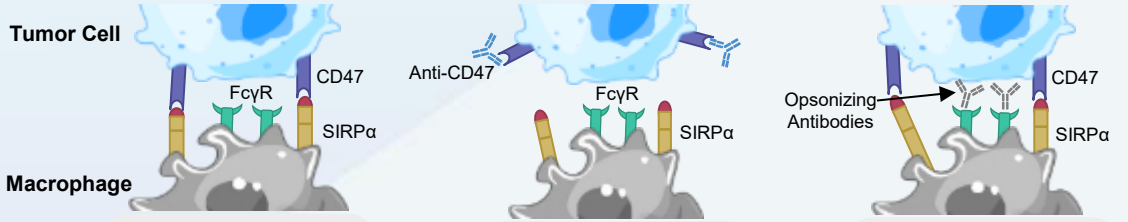
Lack of TILs in TME

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

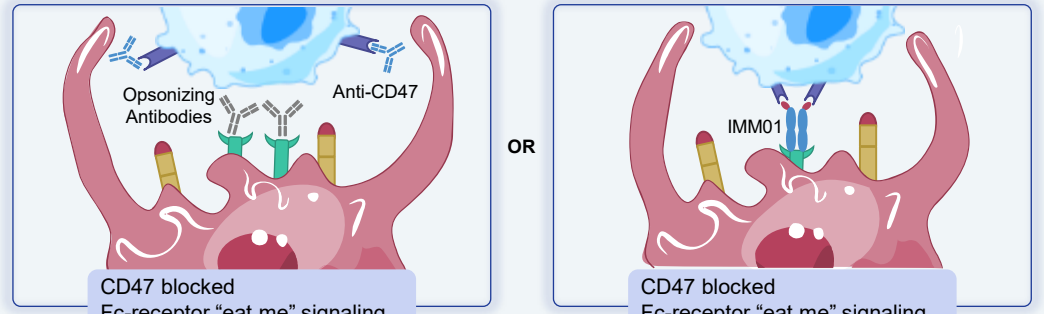
Mechanism of Action in the CD47-SIRPα Signaling Pathway



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



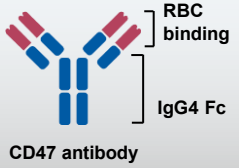
CD47 blocked
Fc-receptor “eat me” signaling
Full macrophage activation

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

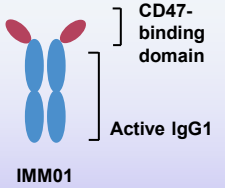
The safety concerns around CD47 pose considerable challenge



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

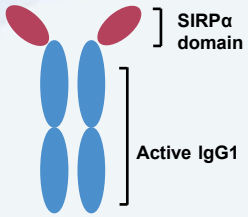


Engineered CD47-binding domain with **no RBC binding** *in vitro;* Modification of deglycosylation of the binding domain **mitigates immunogenicity**

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

Overview and Competitive Advantage of IMM01 (Timdarpacept)

Overview

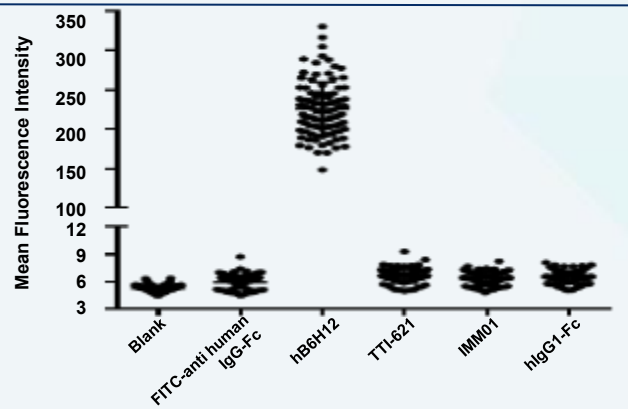


- First SIRPα-Fc fusion protein to enter the clinical stage in China
- Improved safety profile with engineered CD47-binding domain of human SIRPα to avoid human RBC binding
- Designed to fully activate macrophages through a dual mechanism
- Encouraging single-agent efficacy at relatively low dose, with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

Source: Company Data

Competitive Advantage of IMM01 Monotherapy - Safety

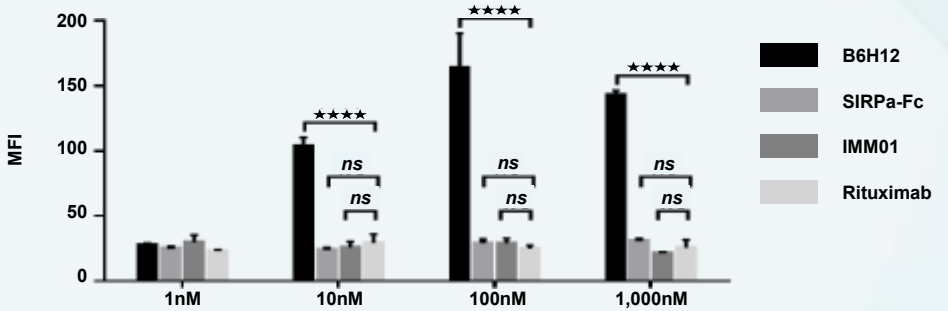
Human RBC Binding Analysis of IMM01



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

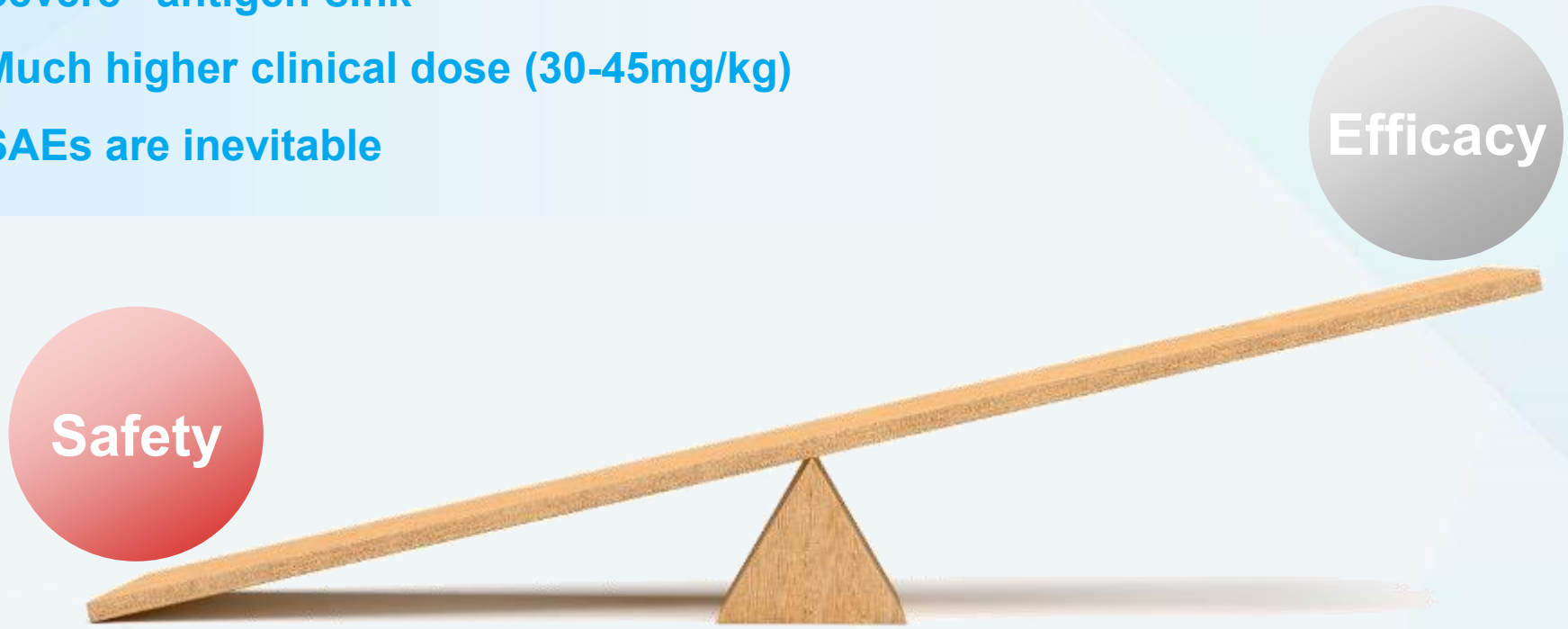


Notes: B6H12 is a CD47-based antibody that serves as the control.

Challenges for CD47-Targeted Drug Development

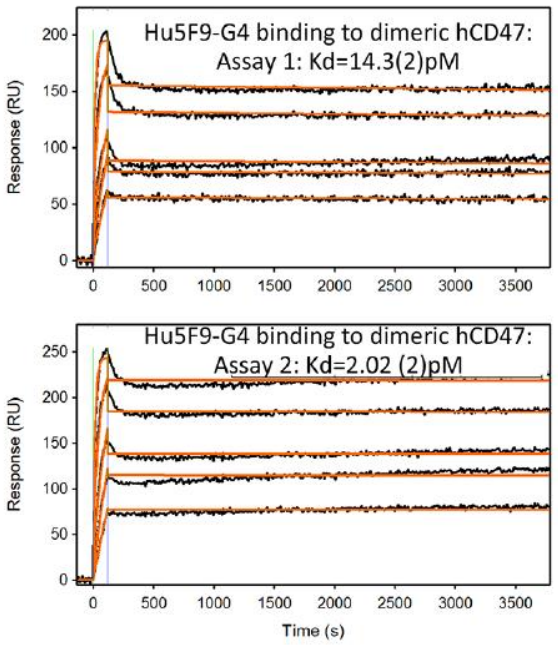
CD47 Antibody

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

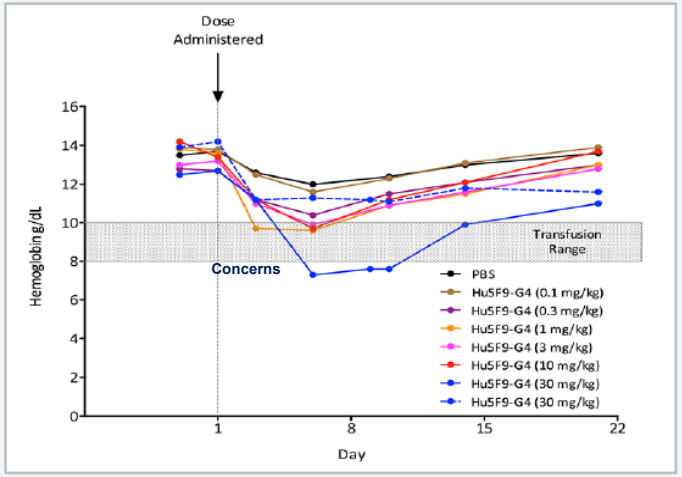


Magrolimab Has Very High Target Affinity and RBC Binding Activity

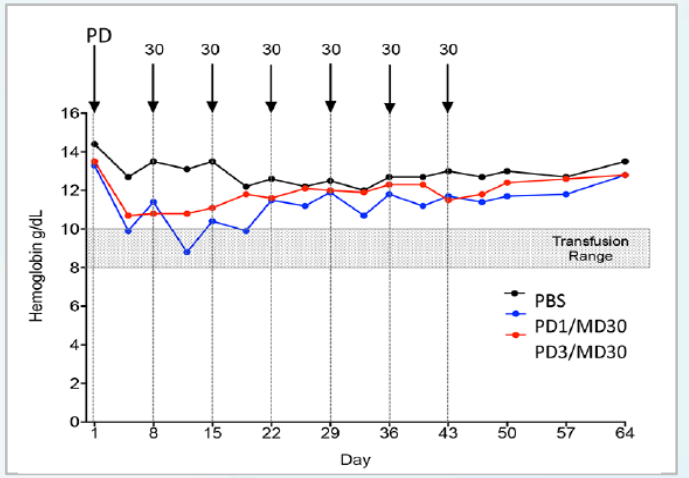
Target affinity assay



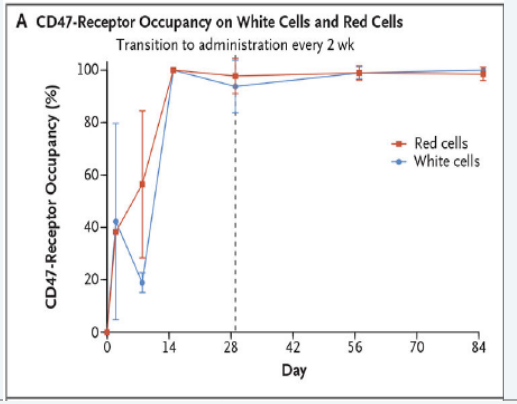
Without priming dose



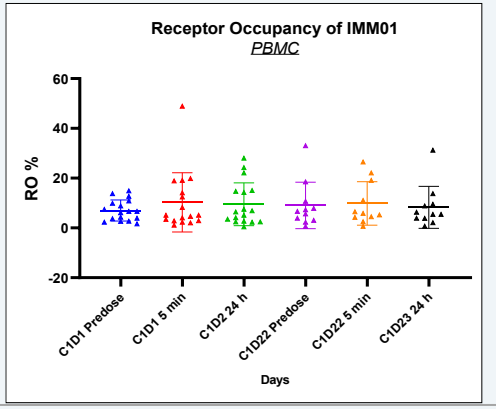
With priming dose (1mpk, 3mpk)



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.

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)**

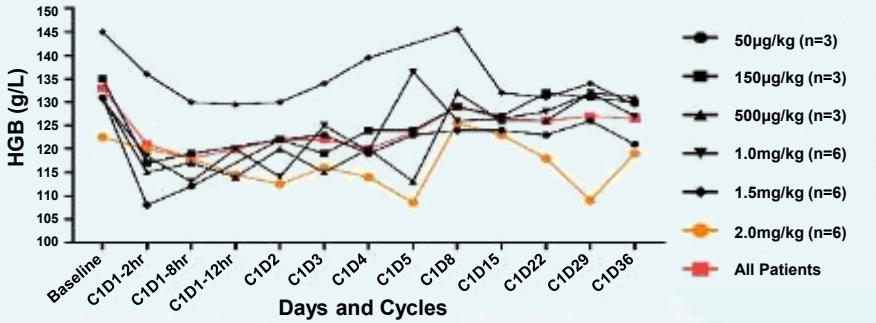
Treatment-related adverse event (n=29)	ALL n (%)	≥Gr 3 n (%)
Positive of Anti erythrocyte antibody	17 (59)	
Leukopenia	16 (55)	2 (7)
Hemolysis	15 (52)	
Infusion related reaction	15 (52)	
Thrombocytopenia	13 (45)	3 (10)
Hypertriglyceridemia	13 (45)	
Anemia	13 (45)	4 (14)
Neutropenia	12 (41)	1 (3)
Neutrocytosis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

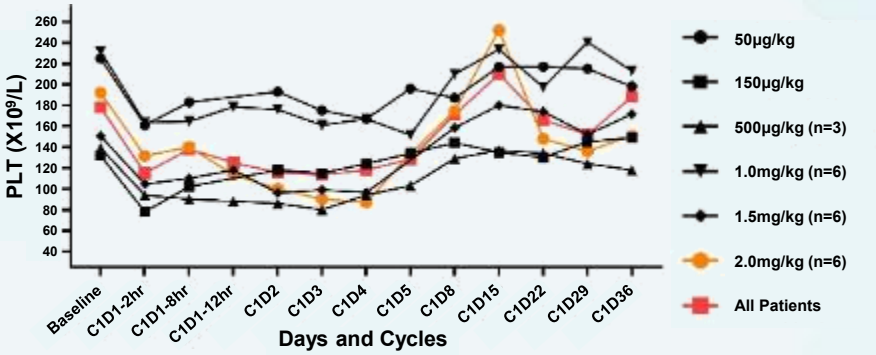
HGB Changes Following Single-dose and Cycle 1 by Cohort



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) + 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 Dec 31st, 2024

宜明昂科
ImmuneOnco

Thank you!