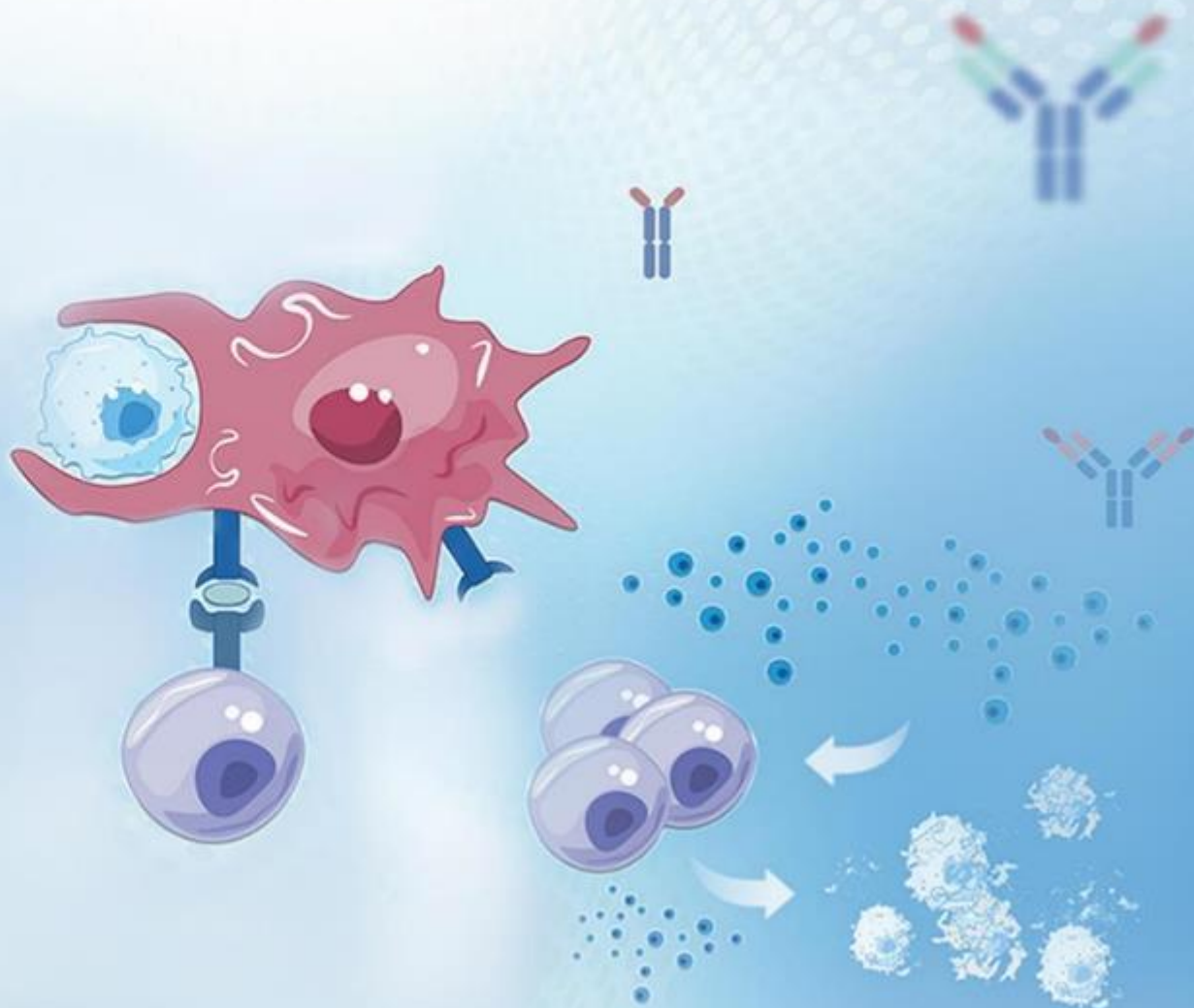




宜明昂科
ImmuneOnco

Corporate Presentation

August 2024



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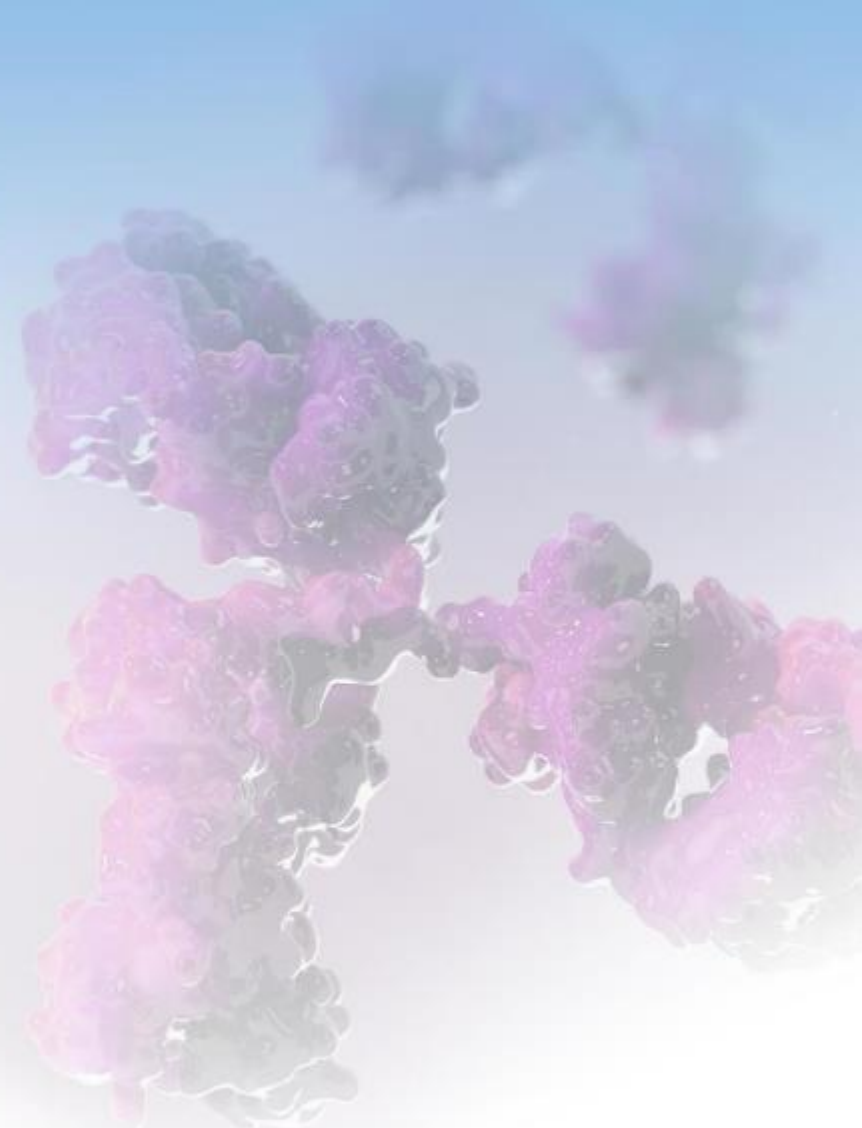
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SECTION 1

Company Overview



Key Milestones



- Steady team with **10+** years coordination



- 29** IND approvals from the NMPA and the FDA



- 30** issued patents
- 23** pending patent applications



- 8** ongoing clinical programs

Pipeline	2015-2020		2021	2022	2023		2024							
	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		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	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	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	IMM01: - Three phase III clinical trials approved for MDS, CMML and cHL in China - Phase III IMM01+PD-1 mAb for R/R 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						
201520162017201820192020202120222023														
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													
 礼来亚洲基金  LYFE 资本基金  龙磐投资 LAPAM CAPITAL  上海科创基金 Shanghai Sci-Tech Innovation Center Capital  阳光保险集团 Sunshine Insurance Group 南京星健睿赢 荣昌股权投资														

Total amount of fund raised: ~\$255MM

Management team



**Wenzhi Tian, MD,
EMBA**
Founder, Chairman &
CEO

 **30+** years academic and industrial experience in the field of immuno-oncology

 **29** IND approvals from the NMPA and the FDA

 **30** issued patents, **23** patent applications, and **30+** scientific publications



Karolinska
Institutet



Weill Cornell
Medicine



North Shore
University Hospital



ImClone Systems



Qiying Lu, MD
CMO, SVP



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

NMSGROUP
ONCOLOGY FORWARD



Zikai Xiong, PhD
SVP, BD



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



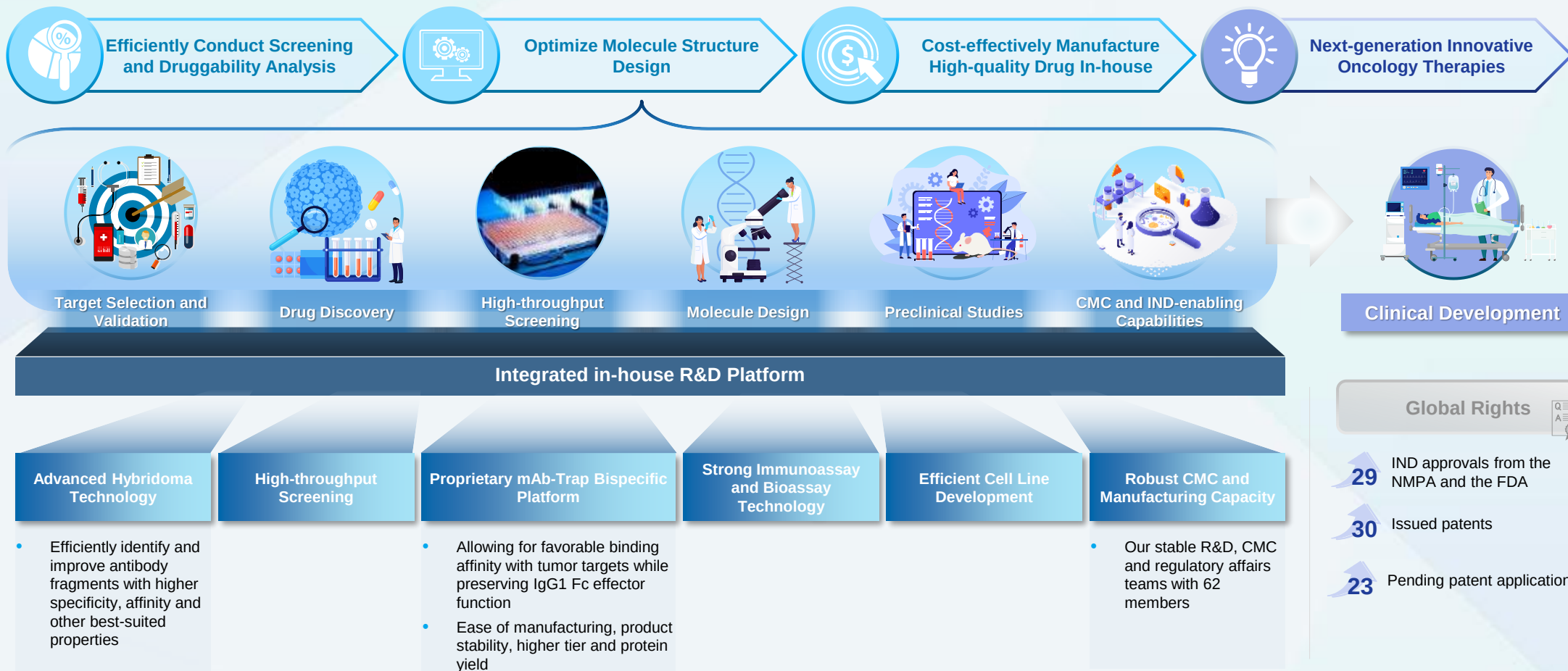
Song Li, BA, MS
VP, R&D



Mei Guan, BS, MS
Secretary of the Board



Integrated proprietary R&D platform



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 (SIRPa-Fc fusion protein)	MDS ⁽²⁾	China (NMPA)							Received Phase III approval from CDE in May	Global
IMM01 + Azacitidine	CD47 (SIRPa-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
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/Ia commenced in June 2023 in China	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
IMM2902	CD47xHER2 (Bispecific)	HER2-positive and low-expressing solid tumors	China (NMPA), US (FDA)							Phase Ia commenced in February 2022 in China and in June 2022 in the U.S.	Global
IMM2520	CD47xPD-L1 (Bispecific)	Solid tumors	China (NMPA), US (FDA)							IND approved in China and the U.S. in Q4 2022; Phase I commenced in China in March 2023 and 5 th cohort ongoing	Global
IMM47	CD24 (mAb)	Solid tumors	China (NMPA), US (FDA)							IND approved in China and the U.S. in October and December in 2023; Phase I commenced in September 2023 in Australia	Global
IMM40H	CD70 (mAb)	Liquid/Solid tumors	China (NMPA), US (FDA)							IND approved in China and the U.S. in August 2022	Global
IMM4701	CD24xCD47 (Bispecific)	Solid tumors	China (NMPA), US (FDA)							IND-enabling	Global
IMC-002 (IMM0306)	CD47xCD20 (Bispecific)	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 (SIRPa-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.



SECTION 2

Our Approach

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

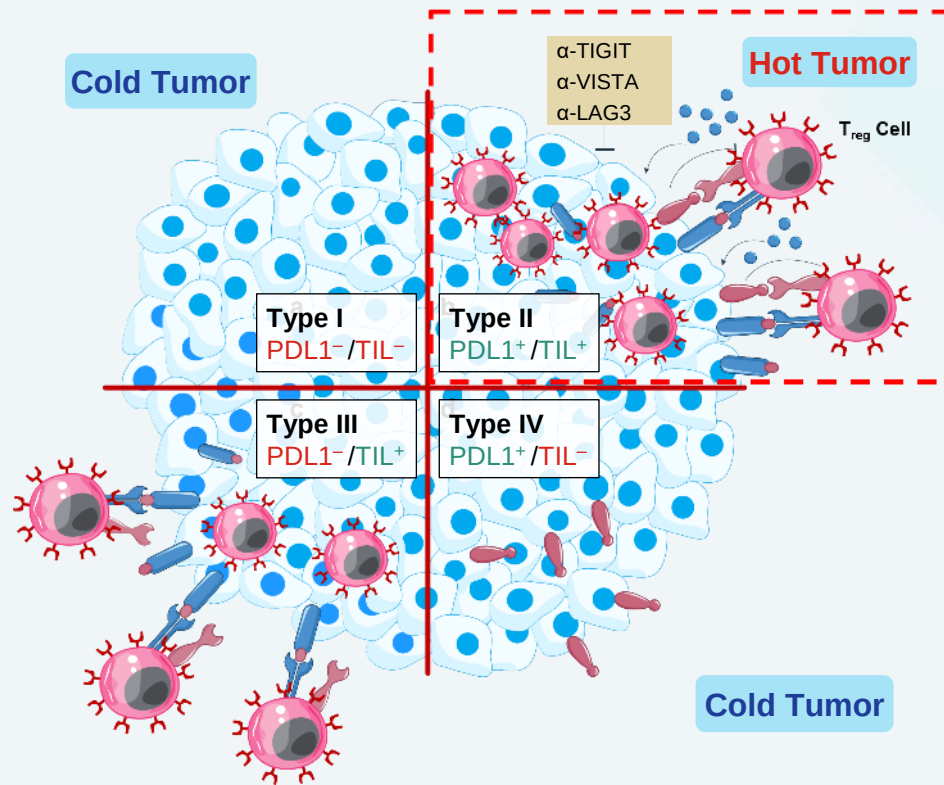
Lack of TILs in TME

Innate immune activation to induce inflammation and attract adaptive immune cells

Type III

Dysfunctional TILs activation

Activation of antigen specific T cells through antigen presenting cells



Type II

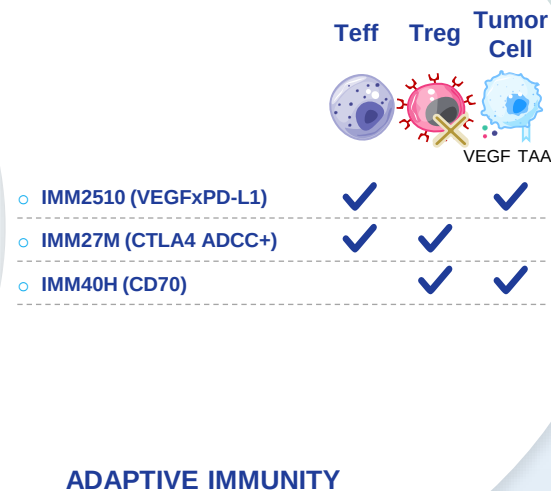
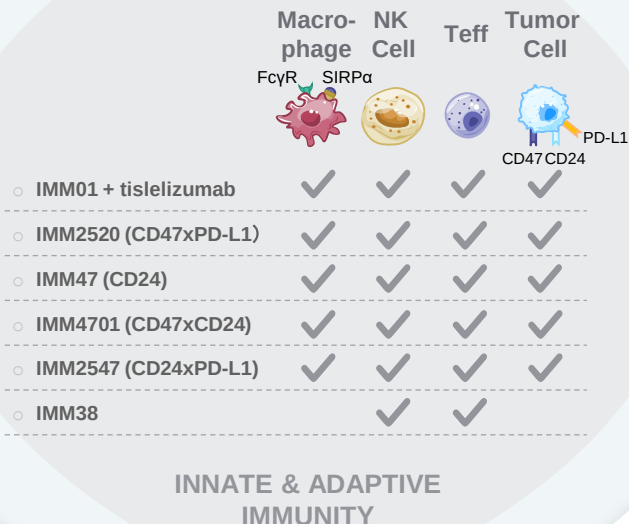
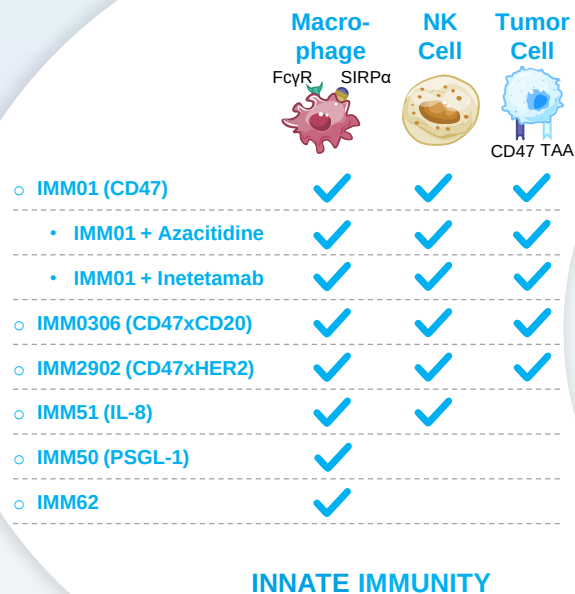
Overregulation of activated TILs

Type IV

Lack of TILs in TME

Innate immune activation to induce inflammation and attract adaptive immune cells

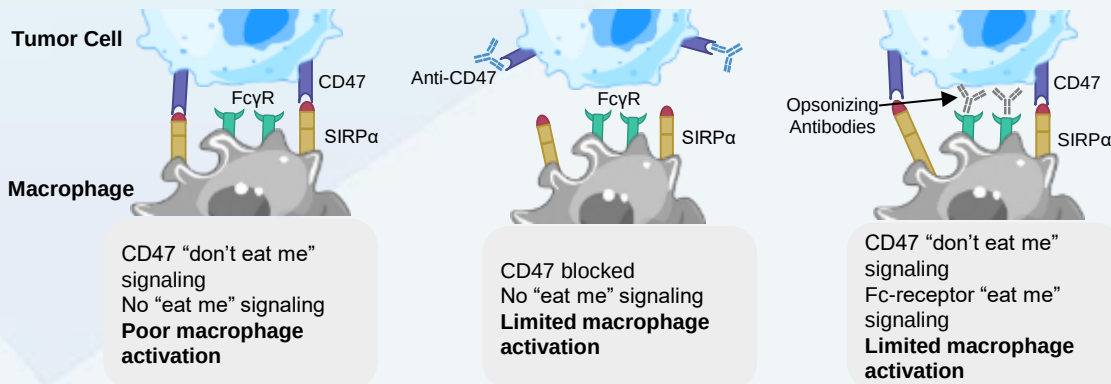
Our Pipeline Harnessing Both Innate and Adaptive Immunities



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

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-SIRP α Signaling Pathway



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

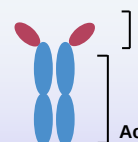


CD47 antibody

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

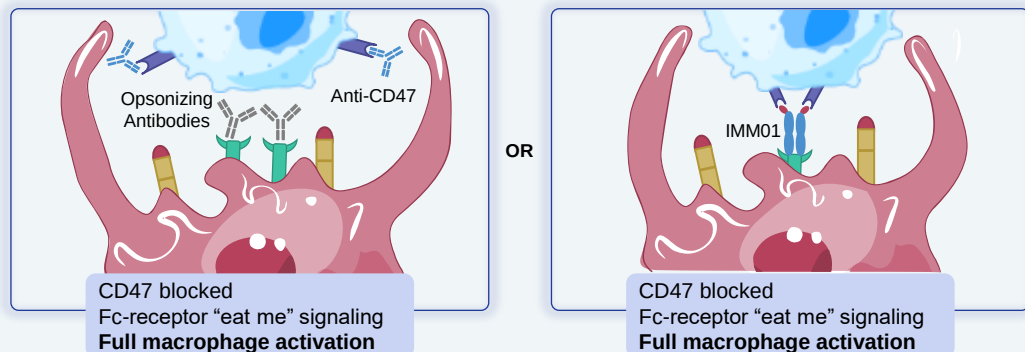
Our differentiated design allows full macrophage activation



IMM01

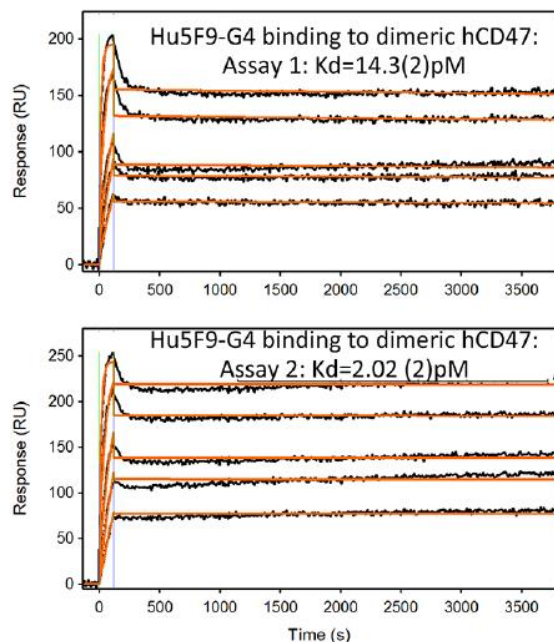
Engineered CD47-binding domain with **no RBC binding in vitro**; Modification of deglycosylation of the binding domain **mitigates immunogenicity**

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



Magrolimab Has Very High Target Affinity and RBC Binding Activity

Target affinity assay

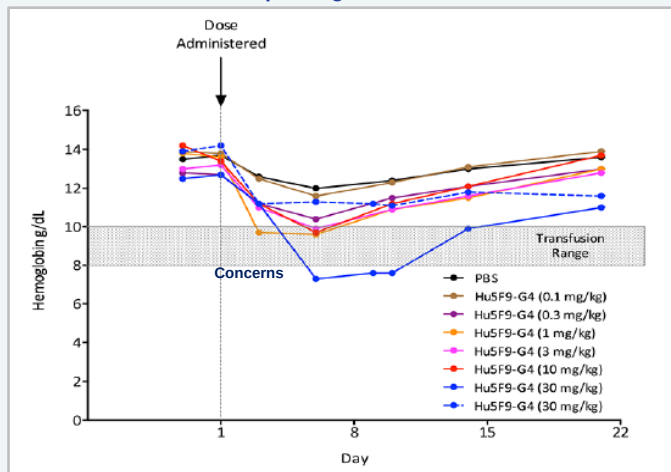


Magrolimab: $K_D = 2\text{-}14.3\text{pM}$

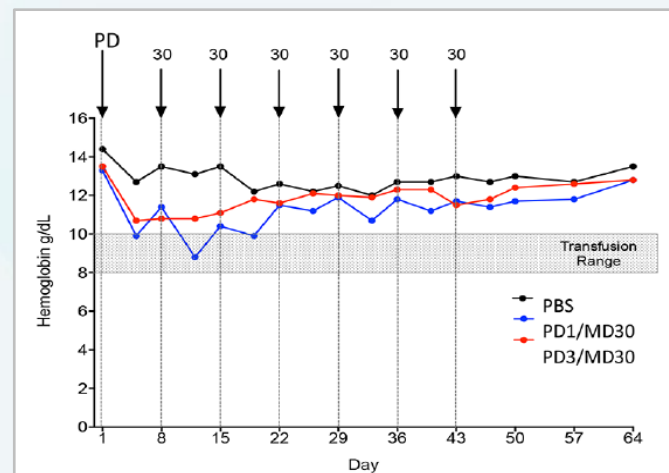
Timdarpaccept (IMM01): $K_D = \sim 3\text{nM}$

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

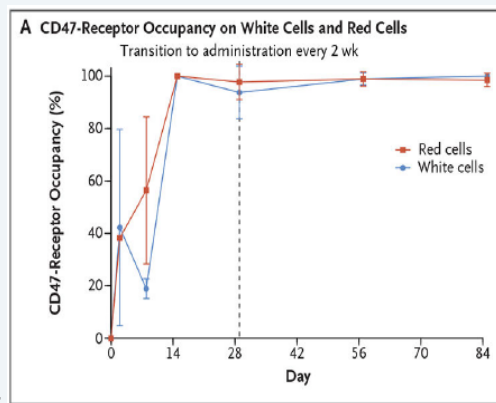
Without priming dose



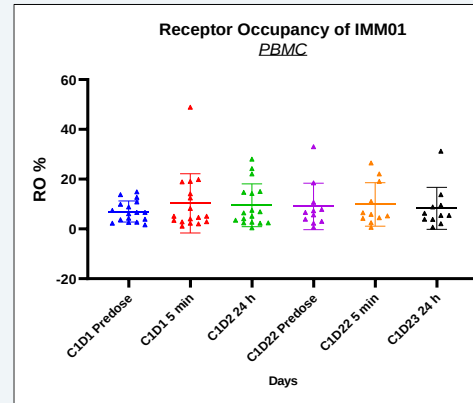
With priming dose (1mpk, 3mpk)



Magrolimab Receptor Occupancy (RO)



IMM01 Receptor Occupancy (RO)



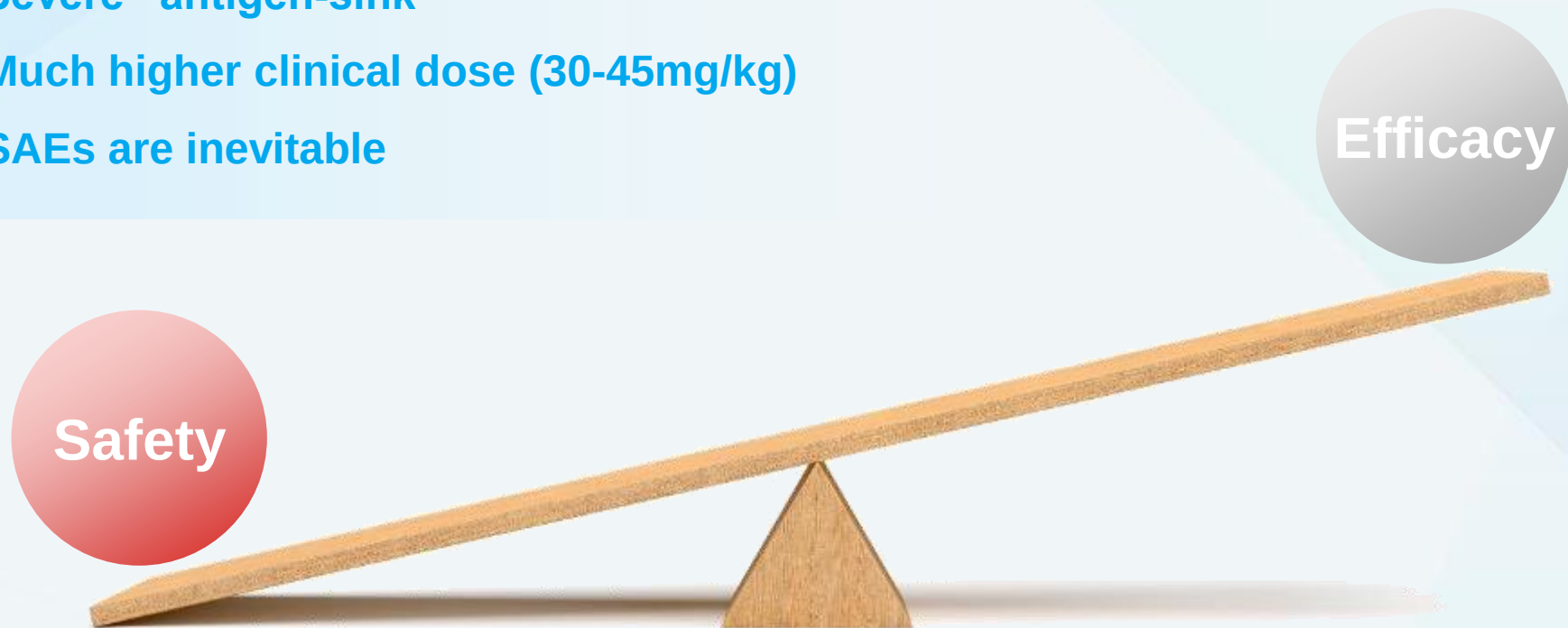
Concerns

- 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)

Challenges for CD47-Targeted Drug Development

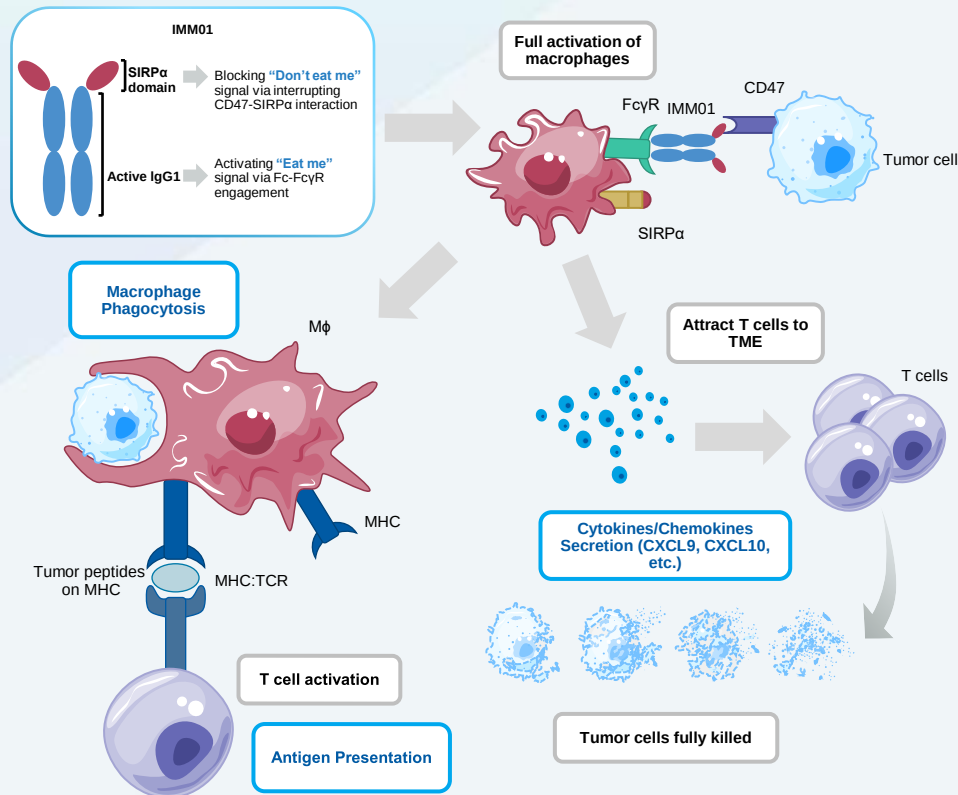
CD47 Antibody

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



Our Differentiated Approaches

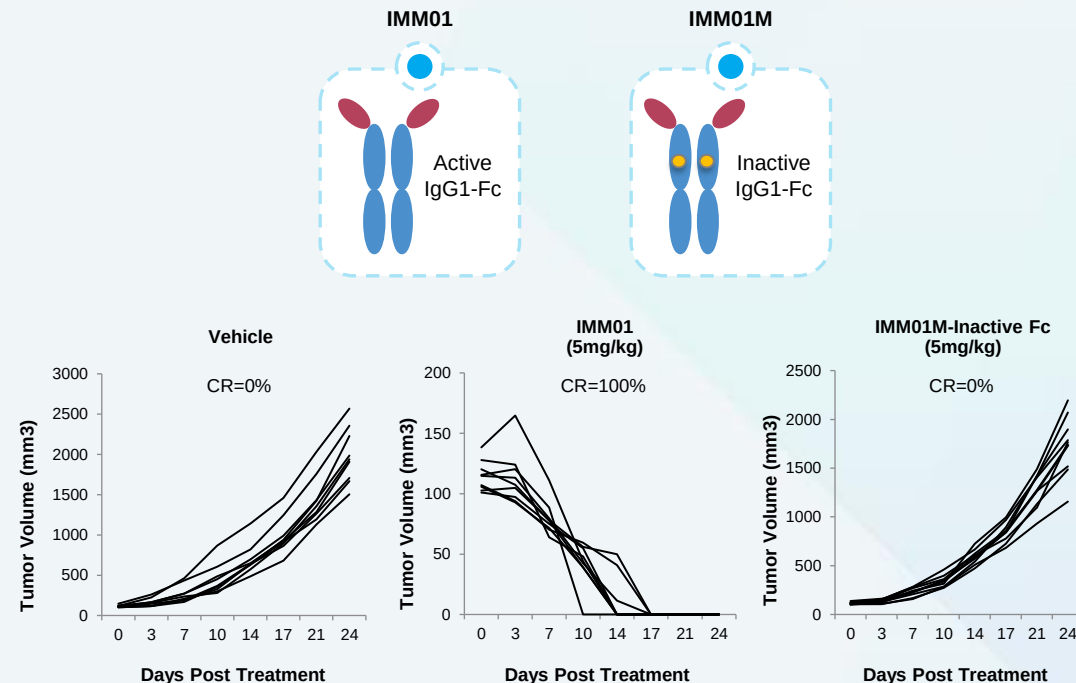
Overview and Competitive Advantage of IMM01(Timdarpacept)



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)

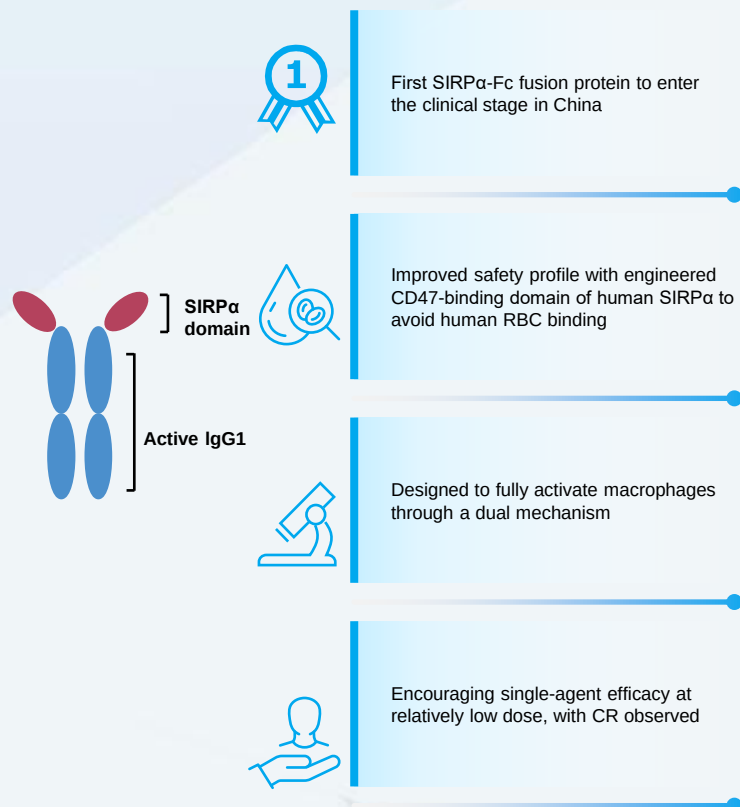


Our Differentiated Approaches (cont'd)

Overview and Competitive Advantage of IMM01 (Timdarpaccept)



Overview

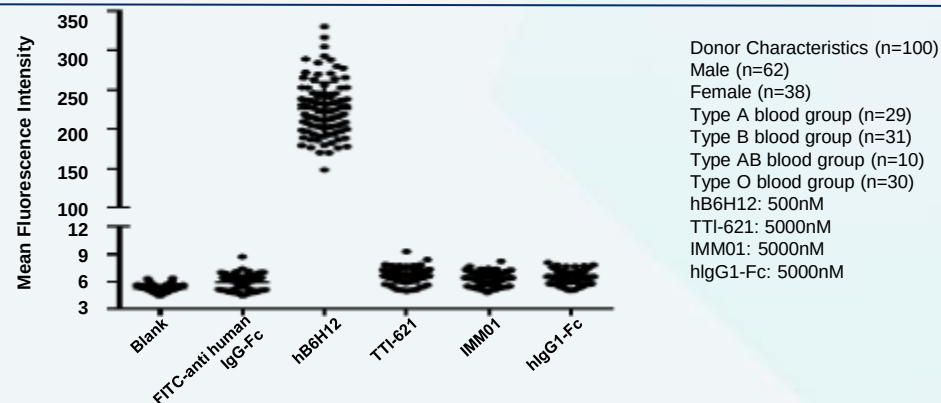


Source: Company Data



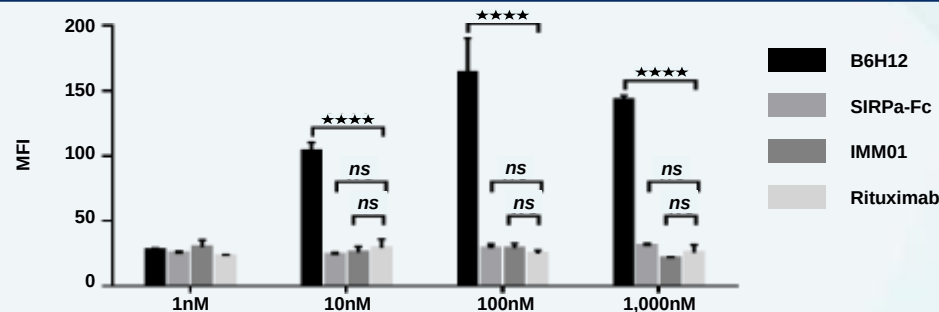
Competitive Advantage of IMM01 Monotherapy - Safety

Human RBC Binding Analysis of IMM01



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

Phagocytosis Against Human RBC



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

Our Differentiated Approaches (cont'd)

Phase I Clinical Trial Results of IMM01 Monotherapy

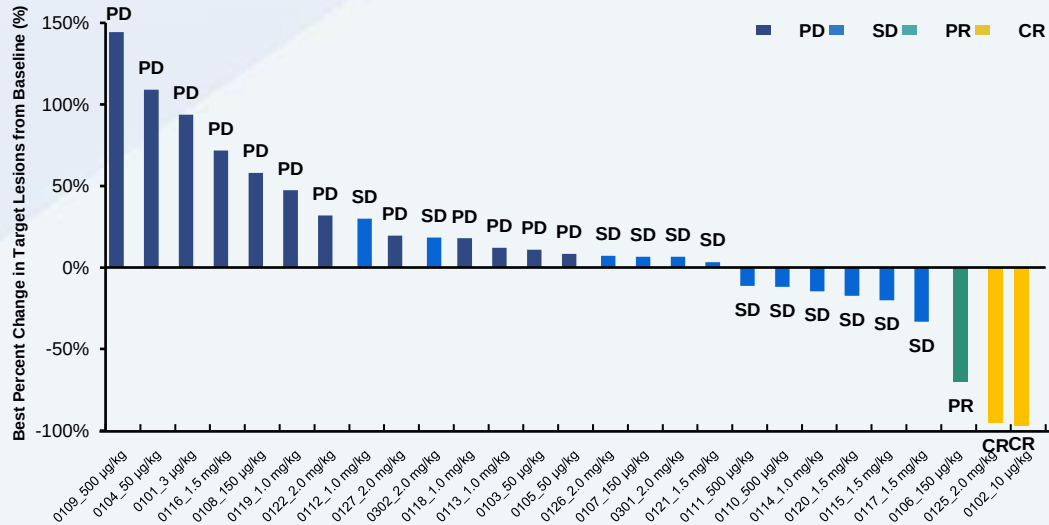


One of the only two companies to have observed **CR in monotherapy** clinical trials with a **well tolerated safety profile**



Potent Antitumor Activity and Encouraging Preliminary Clinical Efficacy

Response Observed in Patients Treated with IMM01 Monotherapy

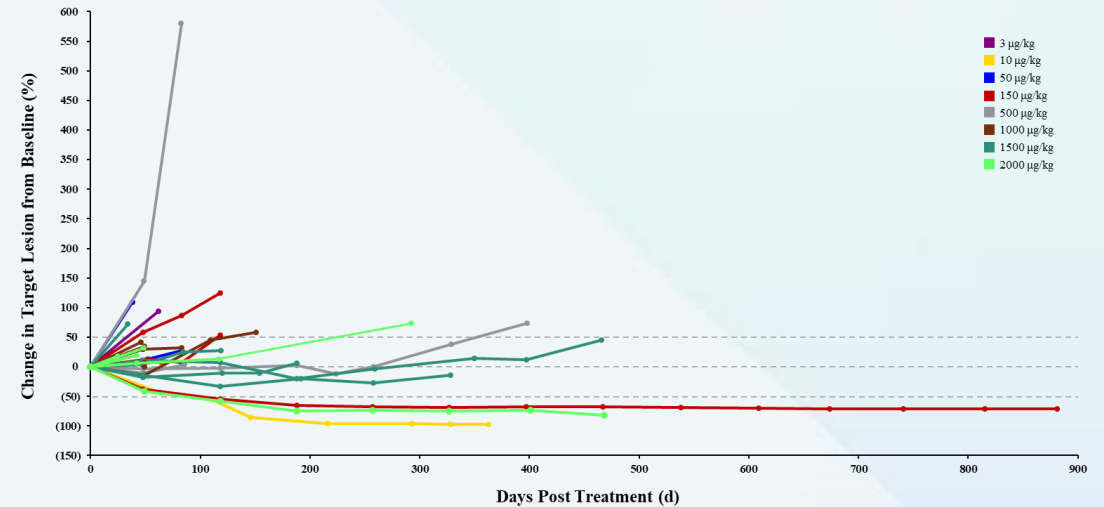


Patients

Note: The colors of bars represent the best overall changes in size of target tumor lesions among 27 evaluable patients in the Phase I monotherapy study

Source: Company Data, as of December 14, 2022

Duration of Response in Patients Treated with IMM01 Monotherapy



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

Source: Company Data

Our Differentiated Approaches (cont'd)

Clinical Trial Results of IMM01 Monotherapy



Safety Results



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



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

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

Notes:

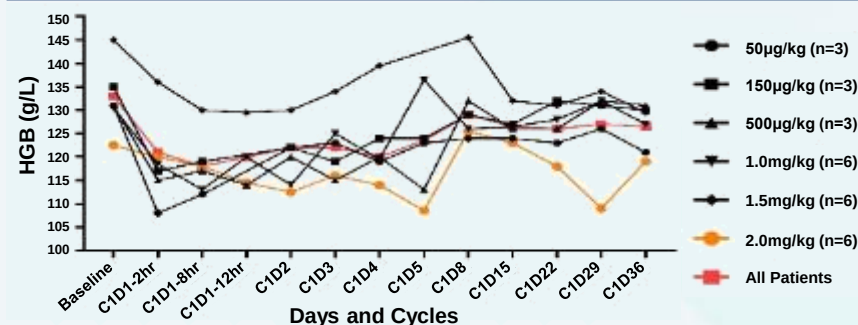
1. TRAE above 10% is presented
2. IMM01 is generally safe and well tolerated in 29 patients
3. Majority of TRAEs were grade 1 and 2
4. Grade 3 and above TRAEs mainly include Leukopenia, Thrombocytopenia, Anemia, Neutropenia, with the highest rate of occurrence as 14% (4/29)

Source: Company Data



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

HGB Changes Following Single-dose and Cycle 1 by Cohort

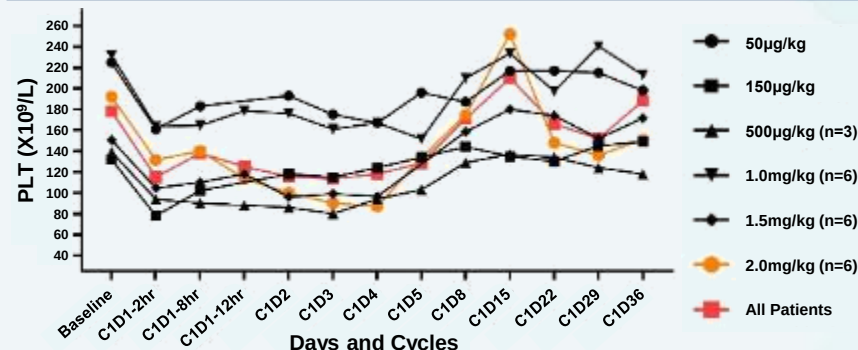


Note: Dosing days are C1D1, C1D8, C1D15, C1D22, C1D29, C1D36.



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.

PLT Following Single-dose and Cycle 1 by Cohort



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



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

Bispecific molecules developed by our mAb-Trap platform to achieve potent efficacy and favorable safety

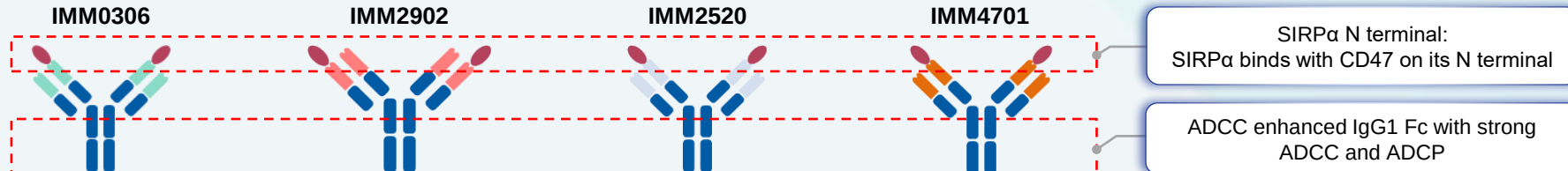
How Our Differentiated Design Improves Safety and Efficacy – CD47-based Bispecific Molecules



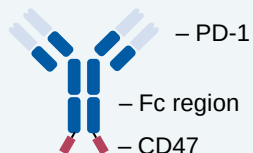
The structure of our bispecific molecules was **deliberately designed through a series of rigorous studies and tests guided by our “DbD” concept** on various aspects, including synergy between targets, tailored molecule structure, expected dosing level, stability, and ease of manufacturing



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



HX009 (Hans Bio)



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



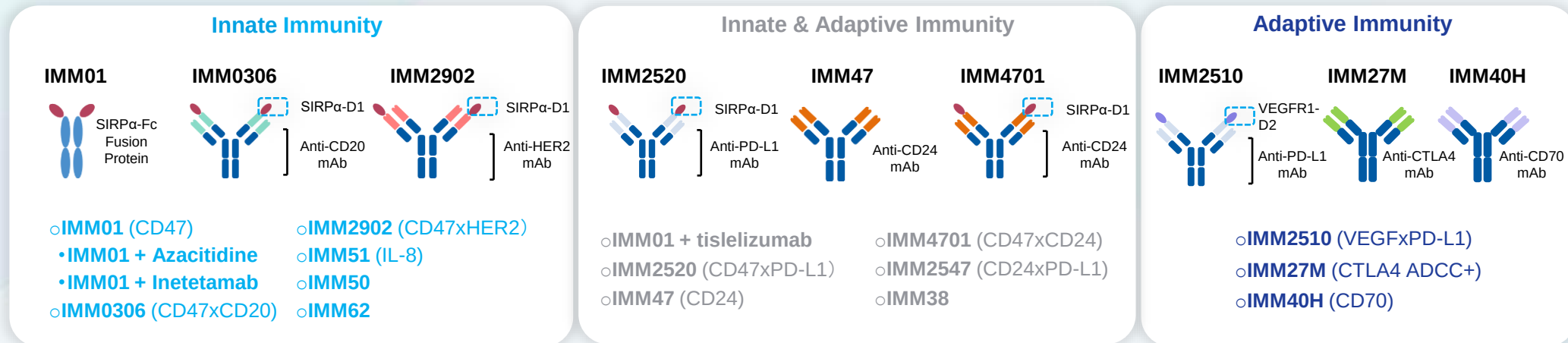
SECTION 3

Major Oncology Programs



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

Our Approach to Seize Market Opportunities



Wide Range of Indications with Unmet Need



Achieve Better Therapeutic Efficacy and Safety Profiles

Notes:

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

IMM01 (Timdarpaccept) - Competitive Landscape



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

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

Notes:

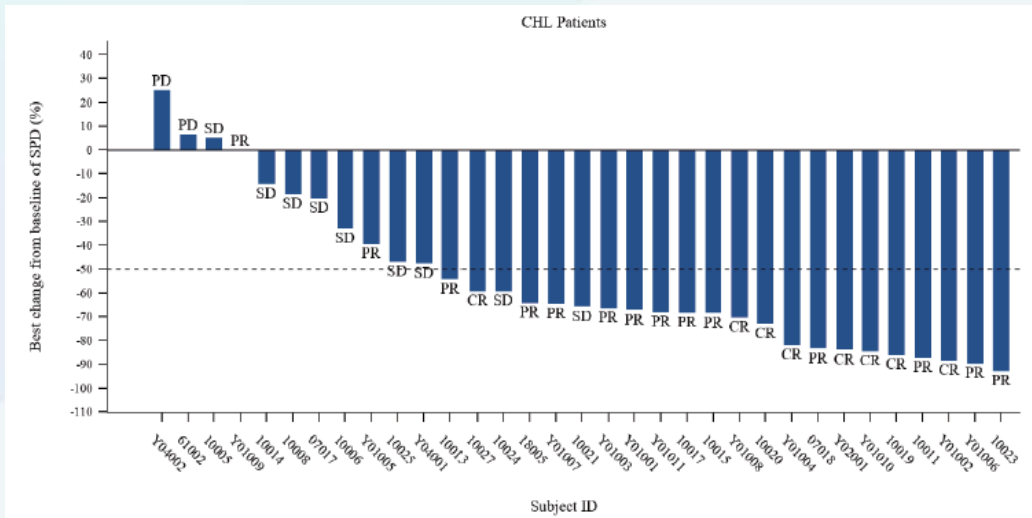
- Clinical data are extracted from official websites of relevant companies, reported clinical trials and published literature
- Despite a comparison is made here, the key results are not from head-to-head studies
- "1st in human" refers to the first posted date of the first clinical trial
- The stage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

Source: Frost & Sullivan, Official Websites of Relevant Companies

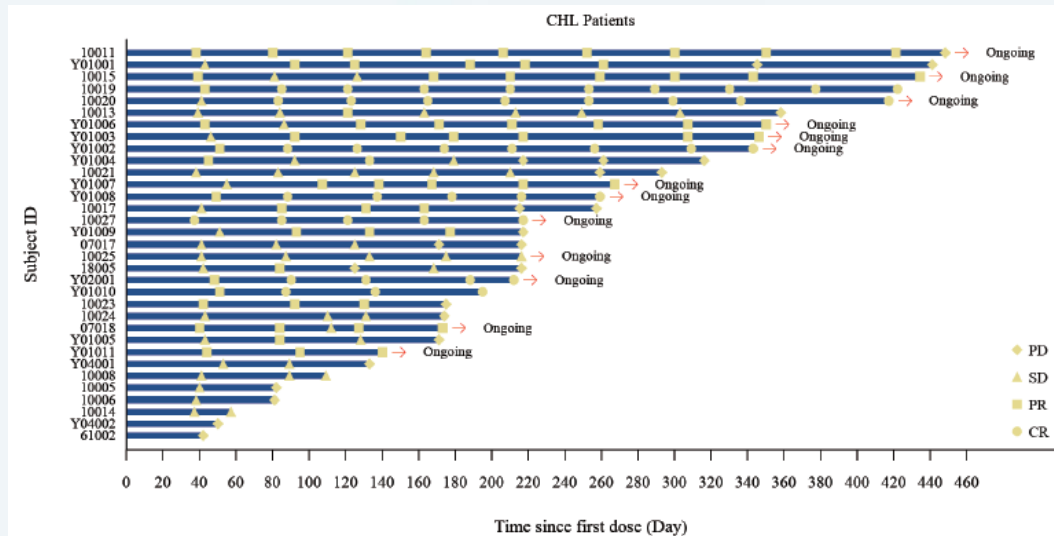
IMM01 (Timdarpaccept) + Tislelizumab (PD-1 mAb)

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

2024 ASCO
ANNUAL MEETING

Oral Presentation

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

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)

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

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

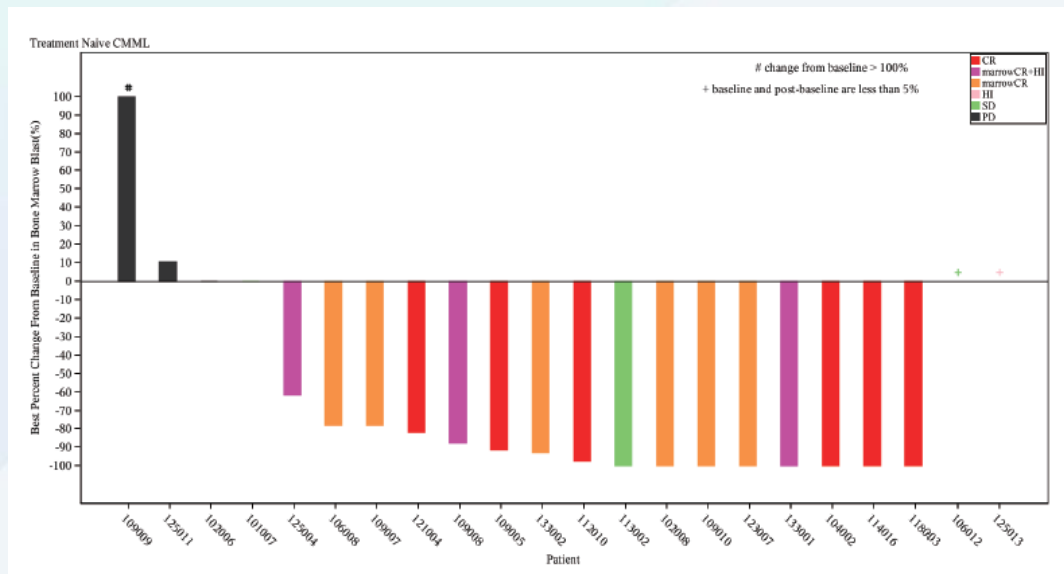
	Timdarpaccept (SIRP α -Fc) + Tislelizumab (PD-1)	Favezelimab (Anti-LAG-3) + Pembrolizumab ¹	Tifcemalimab (Anti-BTLA) + Toripalimab (PD-1) ²
N	33	34	34
ORR	66.7%	29%	35.3%
CR	24.2%	9%	0%
Status	Phase III started in Jul 2024 to treat PD-(L)1 refractory cHL	Phase III of the coformulated two drugs started 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 (Timdarpaccept) + Azacitidine in 1L CMML (Phase II)

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

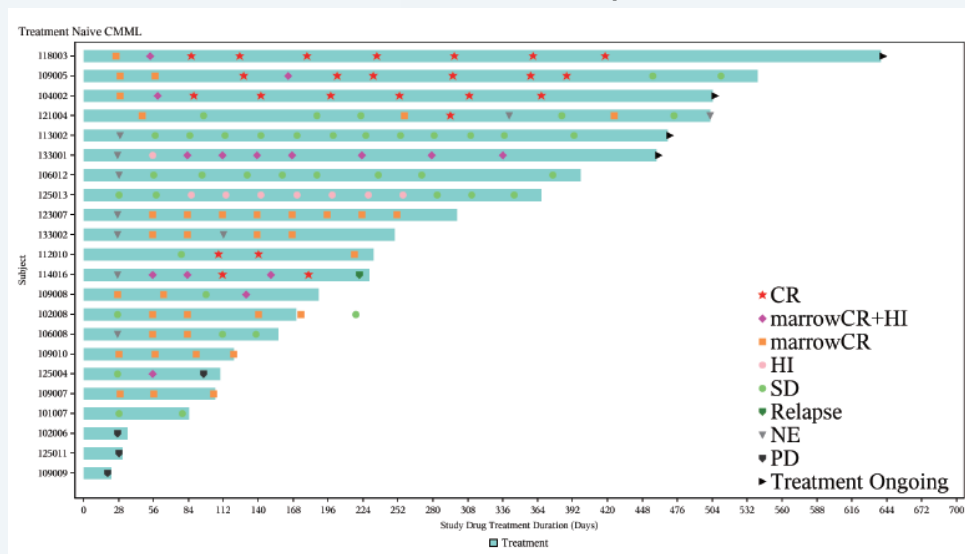


Phase III study of IMM01 (Timdarpaccept) in combination with azacitidine in patients with newly diagnosed CMML was approved by NMPA in June 2024

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

Duration of Treatment and Response

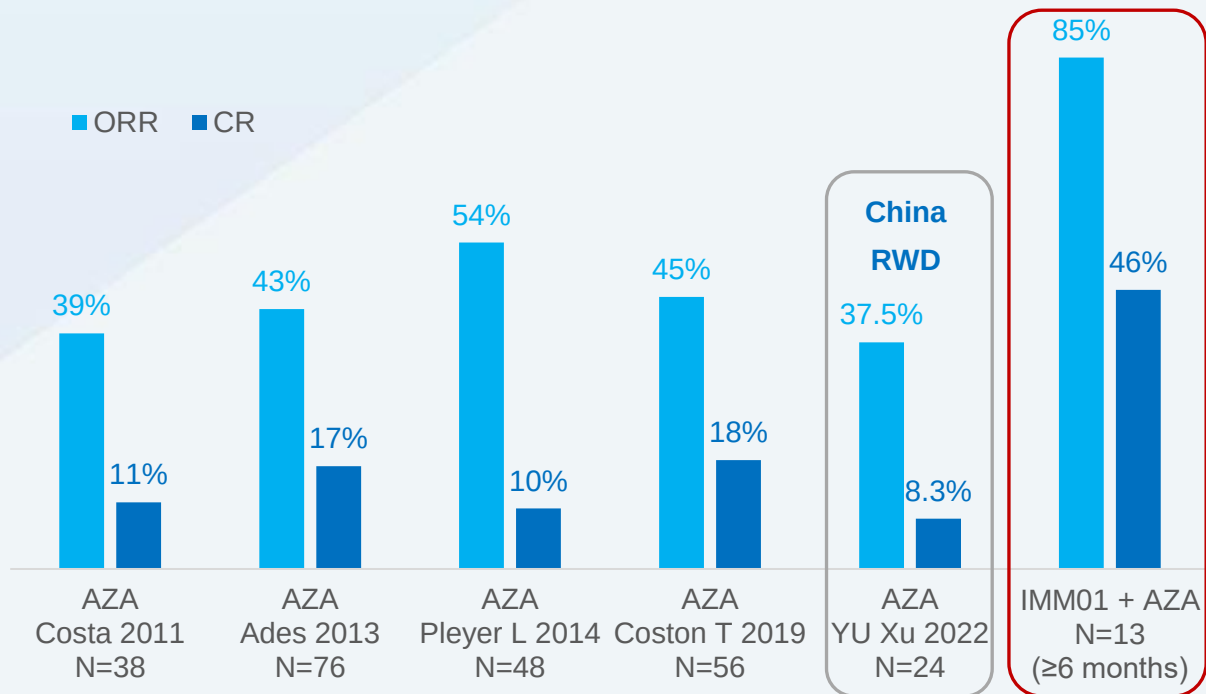


Best Overall Response, n (%)	1L CMML (N=22)	≥4 months (N=16)	≥6 months (N=13)
ORR	16 (72.7%)	14 (87.5%)	11 (84.6%)
CR	6 (27.3%)	6 (37.5%)	6 (46.2%)
mCR + HI	3 (13.6%)	2 (12.5%)	2 (15.4%)
mCR alone	6 (27.3%)	5 (31.3%)	2 (15.4%)
HI	1 (4.5%)	1 (6.3%)	1 (7.7%)

IMM01 (Timdarpaccept) + 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:

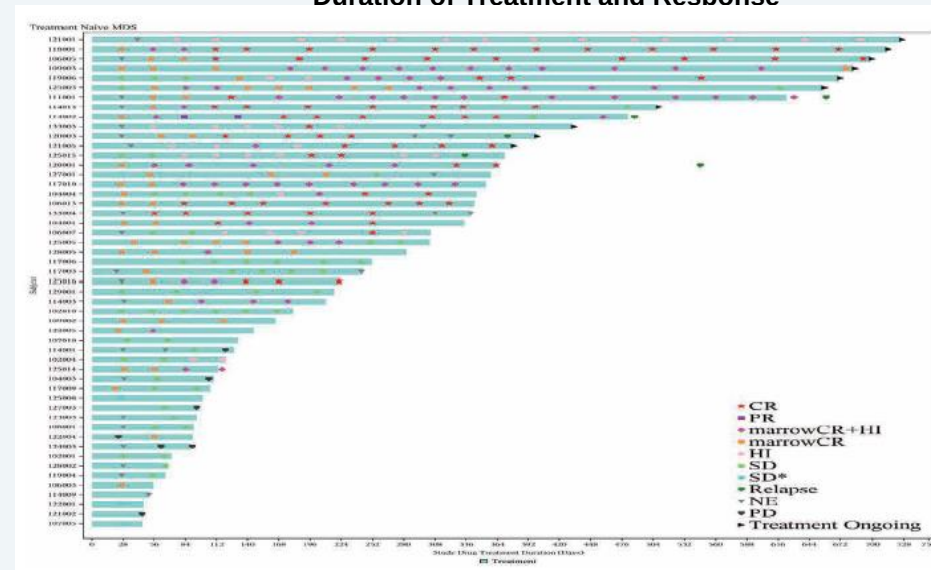
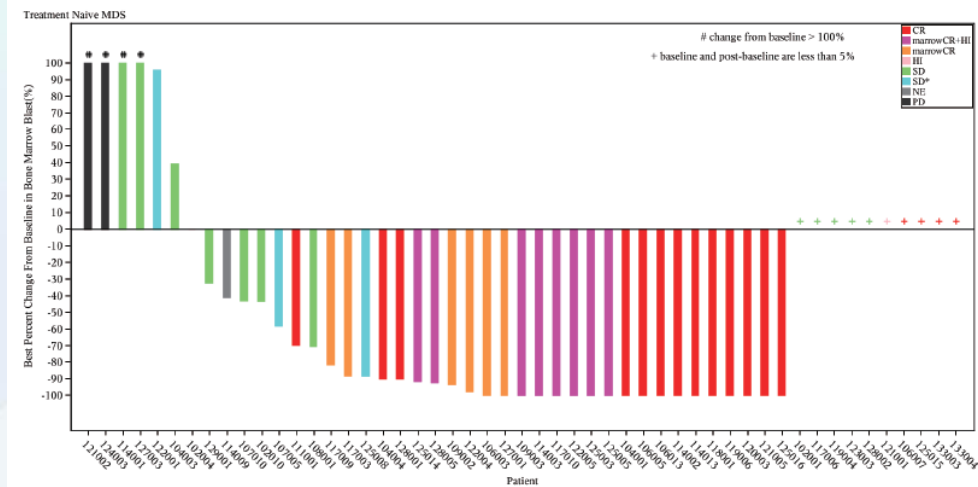
- ORR refers to overall response rate; CR refers to complete response.
- There were no head-to-head comparison clinical trials conducted between these drugs. The results of clinical trials of a drug cannot be directly compared to that of another drug and may not be representative of the overall data.

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

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

Duration of Treatment and Response

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



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

2024 ASCO
ANNUAL MEETING

Oral Presentation

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%)

IMM01 (Timdarpaccept) + Azacitidine

Comparison: Safety results

Magrolimab + AZA vs AZA alone

TRAE	MDS Ib Magrolimab + AZA (N=95)		AZA-001 MRCT AZA alone (N=175)	
	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.

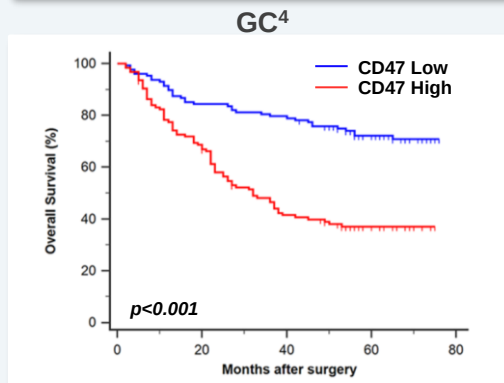
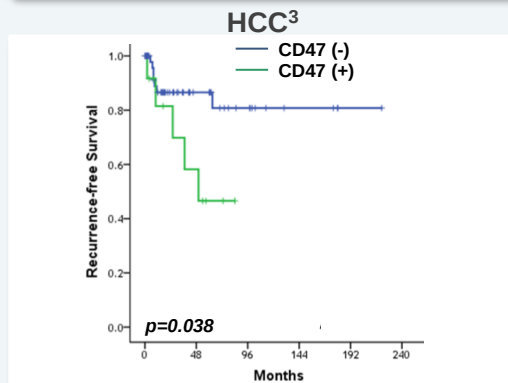
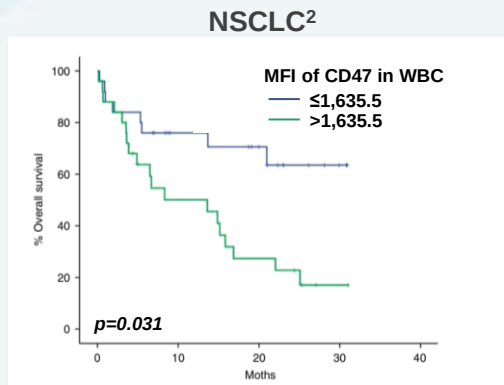
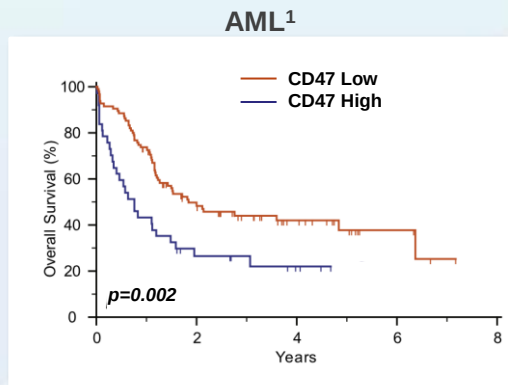
IMM01 + AZA vs AZA alone

TRAE	IMM01-02 Study – MDS Cohort IMM01 + AZA (N=57)		China MDS-002 Single-arm Study AZA alone (N=72)	
	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.

IMM01 (Timdarpaccept) - Market Opportunities

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



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

Source:

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

Strong Potentials of CD47-based Therapies

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

Developing In-house and Own IP and Commercial Rights



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

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

Other patent applications are pending

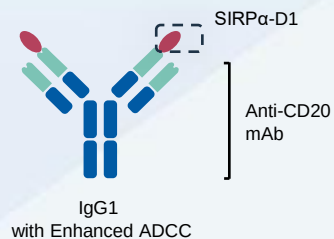
IMM0306 (CD47×CD20)

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



Overview

IMM0306 Molecule Structure



Full macrophage activation

Improved ADCP and ADCC activity

Improved effectiveness for treating patients predominantly expressing FcγRIIIA-158F polymorphism that is less sensitive to CD20 antibody treatment

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**



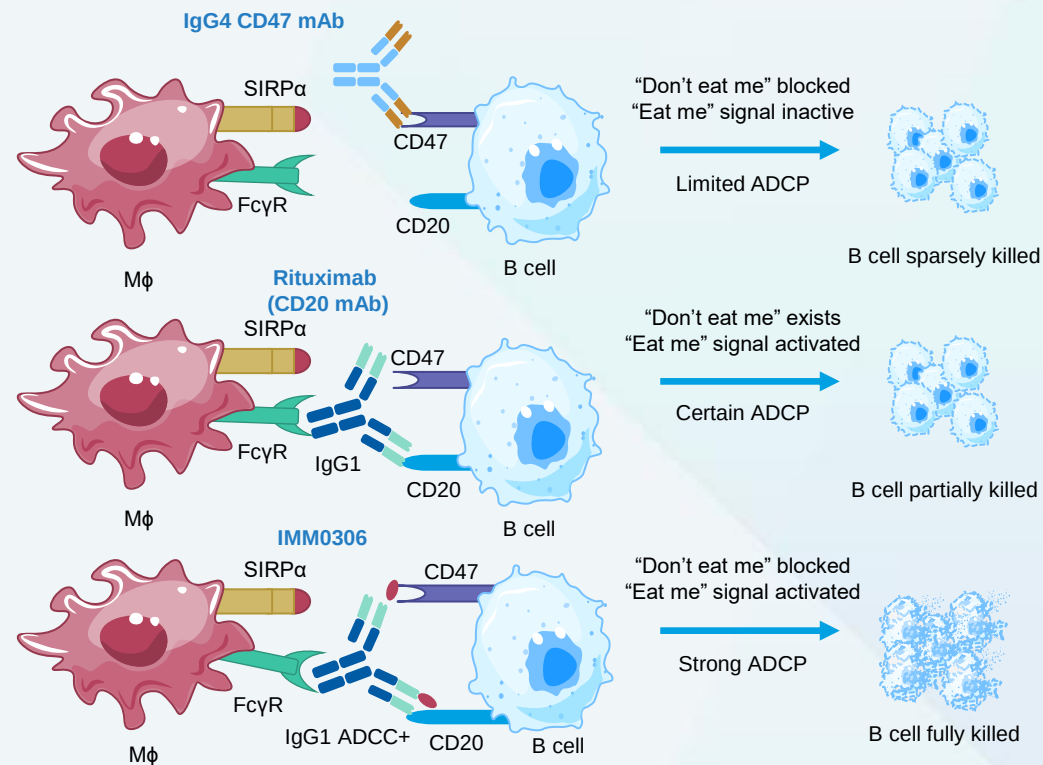
2 CD47×CD20 bispecific antibodies/fusion proteins under development globally
Among them, IMM0306 is the **1st** to enter into a clinical trial



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



Mechanism of Action



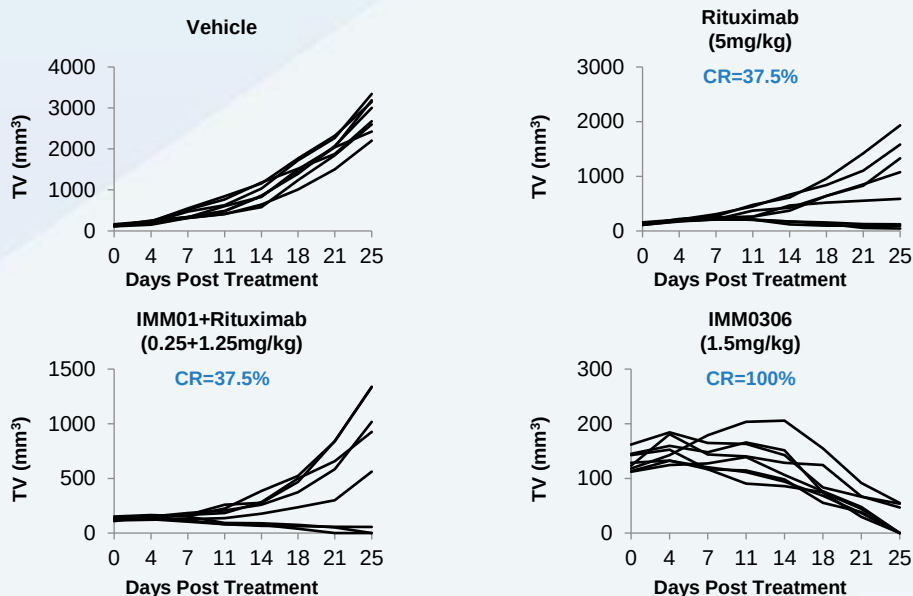
IMM0306 (CD47×CD20) (cont'd)

Preclinical Results

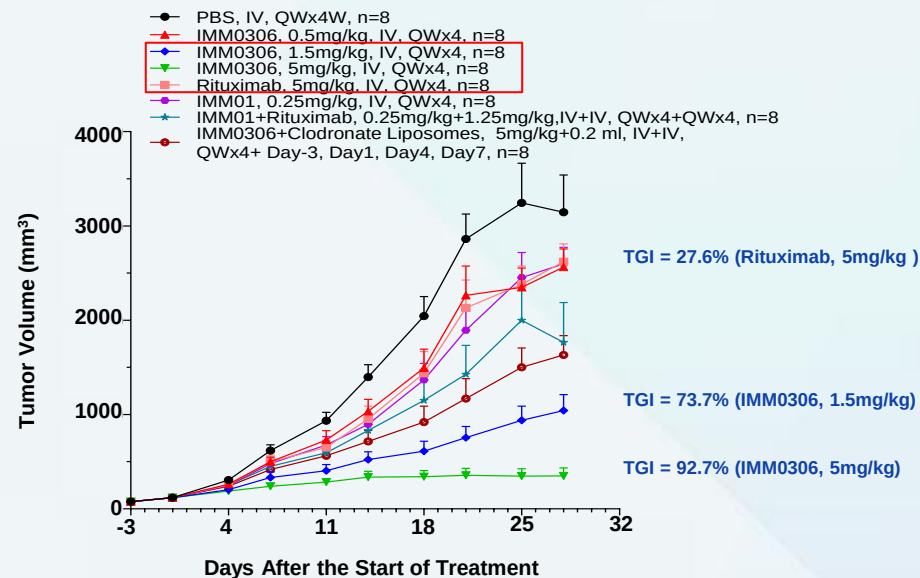


In Vivo Efficacy Study

Efficacy Study in Lymphoma (Daudi) Xenograft Mouse Model



Efficacy Study in Lymphoma (Raji) Xenograft Mouse Model



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

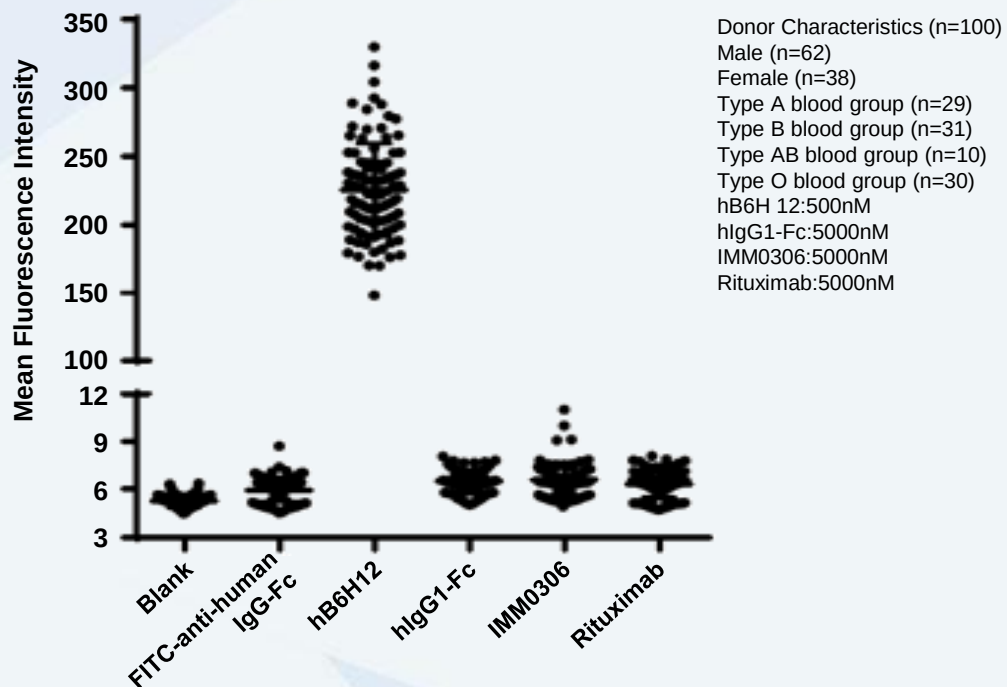
IMM0306 (CD47×CD20) (cont'd)

Preclinical Results



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

Human RBC Binding Analysis of IMM0306



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



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

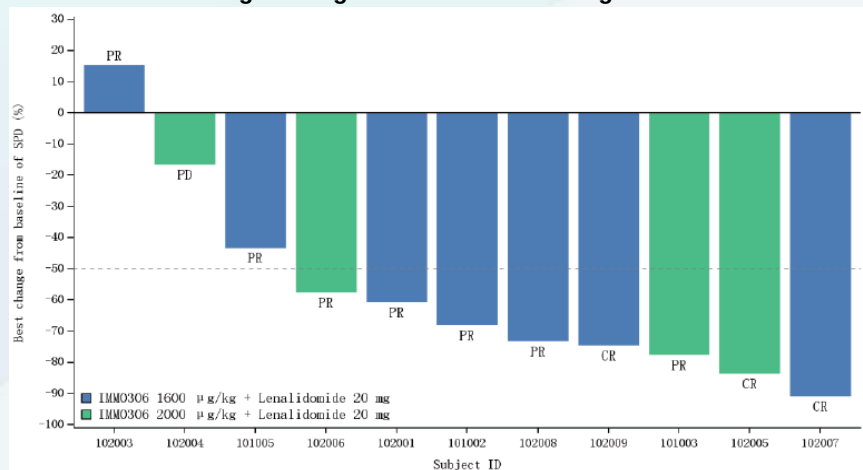


Only triggers **minor CRS**

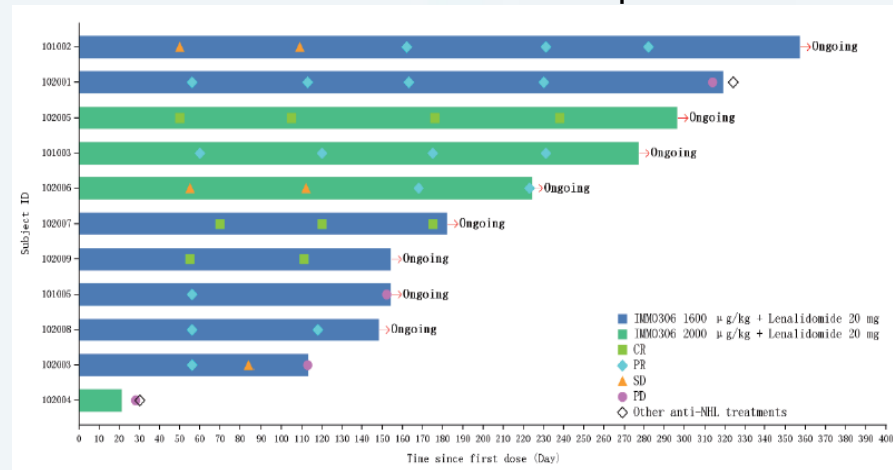
IMM0306 + Lenalidomide (CD47×CD20)

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)

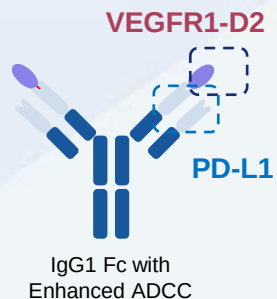
Note: among 6 efficacy-evaluable R/R FL patients in the Phase **Ila** trial, 4 CR and 2 PR were assessed by investigators by Mid July. The ORR and CRR were 100% and 66.7%, respectively

IMM2510 (VEGF × PD-L1)

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

IMM2510 - Target Introduction and Molecule Structure

IMM2510 Molecule Structure



PD-L1

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



VEGF

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



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

	 PD-L1	 VEGF	 PD-(L)1 Combo ¹
Molecule	  		
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

Notes:

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

IMM2510 - Mechanism of Action

By connecting VEGF and PD-L1, IMM2510 can



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



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



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

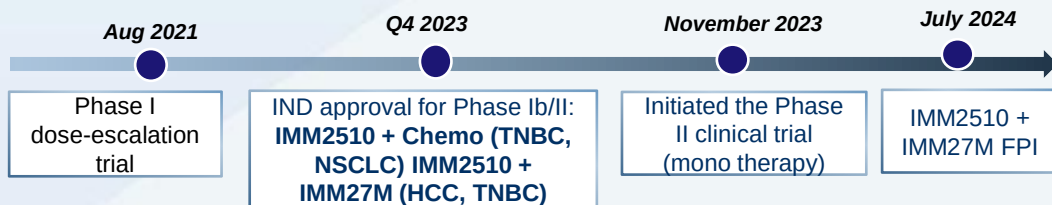


Further activate NK cells and macrophages through strengthened Fc-mediated ADCC/ADCP activities to promote innate and subsequent adaptive immune responses

IMM2510 (VEGF × PD-L1) (cont'd)



Clinical Development Plan



Developing One Owned Patent Family



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



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.

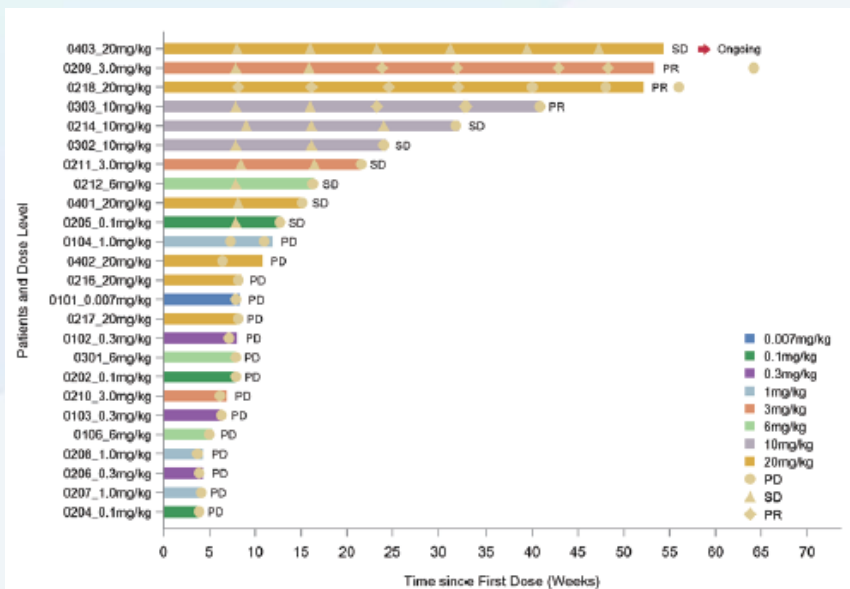
We will receive an upfront payment and potential near-term payments of up to **US\$50 million** as well as potential additional development, regulatory, and commercial milestones payments of up to **US\$2.1 billion**, plus single digit to low double-digit percentage royalties on global (outside the Greater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



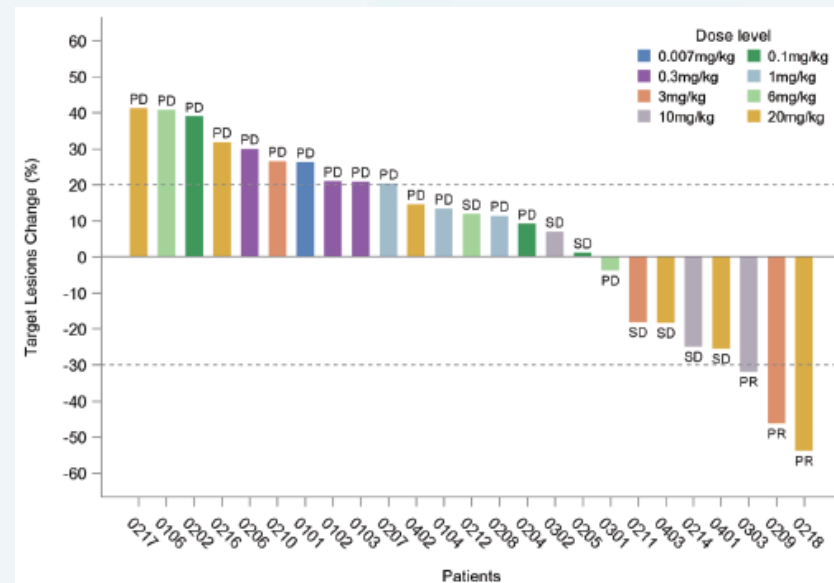
IMM2510 (VEGF × PD-L1) (cont'd)

Phase I: Efficacy Summary in advanced solid tumors

Duration of Treatment and Response



Best Percentage Change from Baseline in Target Lesion



Of 25 response-evaluable patients as of June 30, 2024, **3 patients** had confirmed PR, and **7 patients** achieved SD, with 4 of them observed tumor shrinkage of over 15%

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

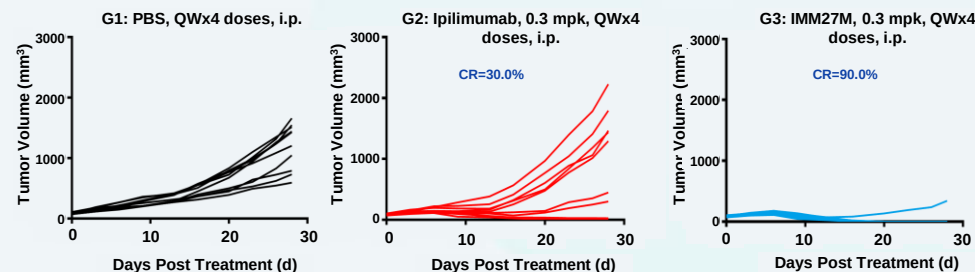
Phase I mono
completed and
confirmed RP2D

IMM2510 +
IMM27M FPI

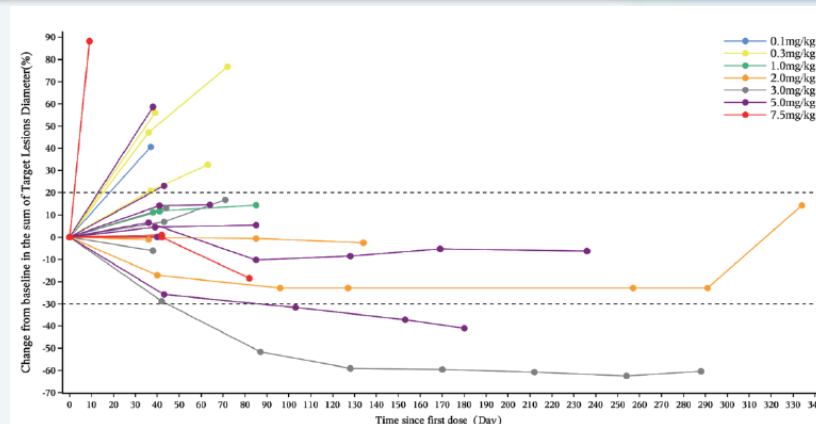


Stronger In Vivo Antitumor Effects and Preliminary Ph1 data

Efficacy Study in Colon Cancer (MC38) Syngeneic Mouse Model



Duration of Treatment and Best Response in Phase I



Preliminary efficacy:

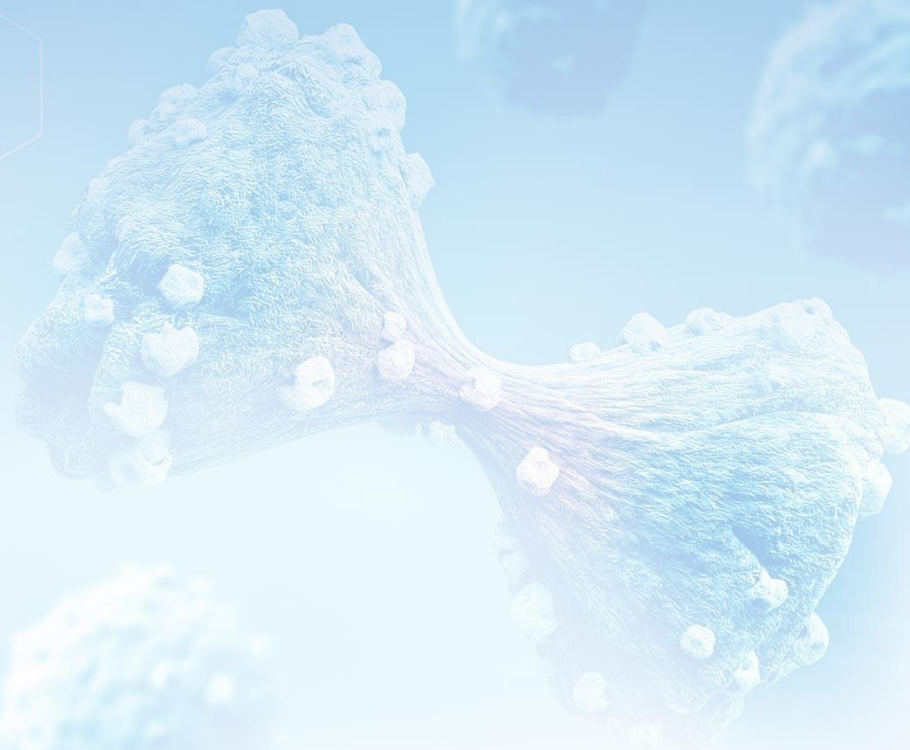
2 confirmed PR
and 3 SD with tumor
shrinkage

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



SECTION 4

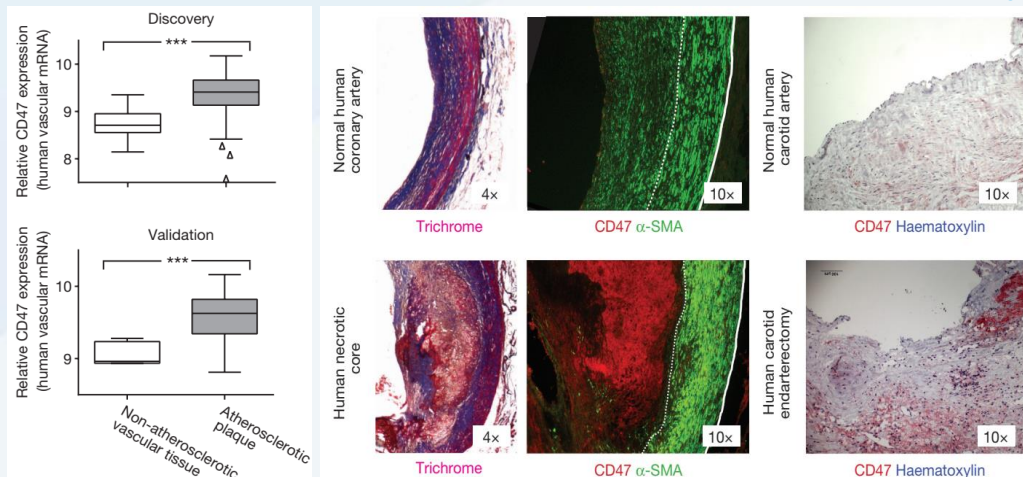
Non-Oncology Programs



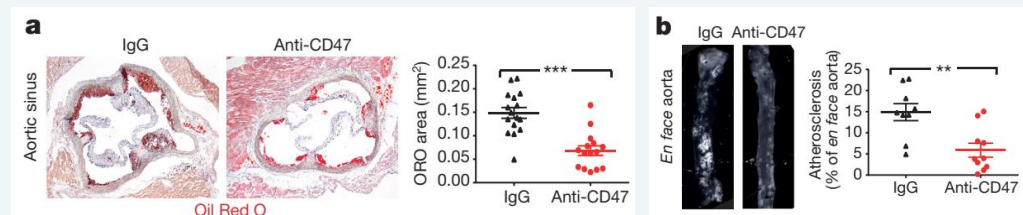
IMC-001 (IMM01, SIRP α -Fc)

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

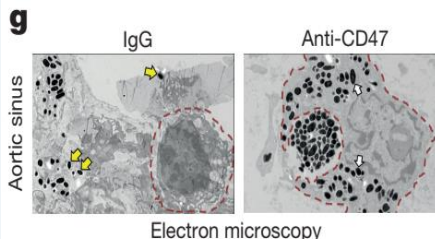
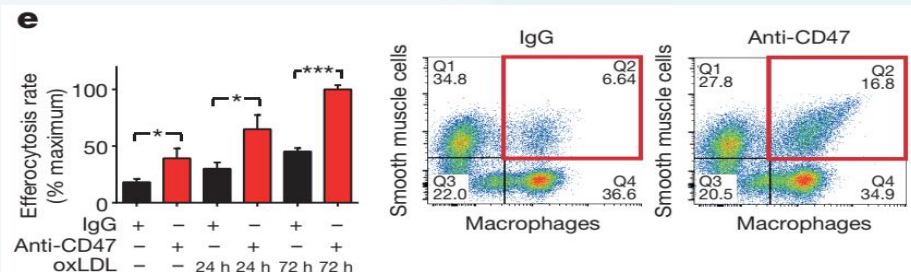
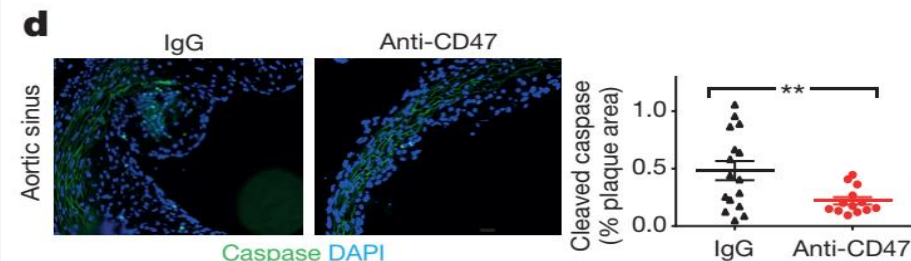
 CD47 is highly expressed in human atherosclerotic plaque



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



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



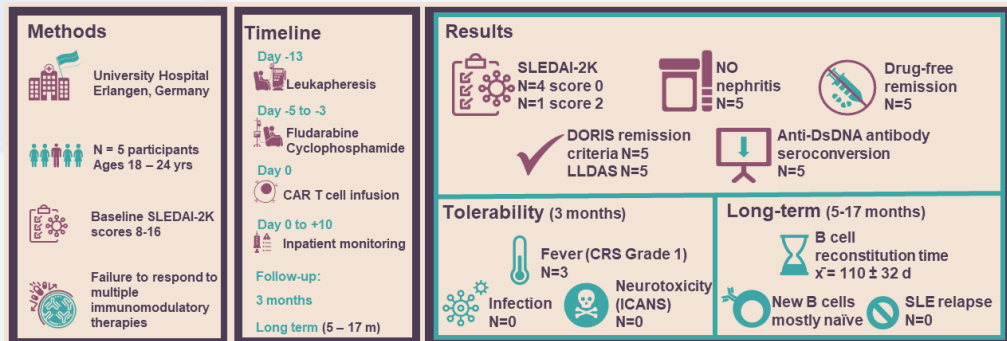
 **BITTERROOT BIO**

- Bitterroot Bio has one CD47/SIRP α 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 Weissman, Nick Leeper, John C. Martin and Lou Lange

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.

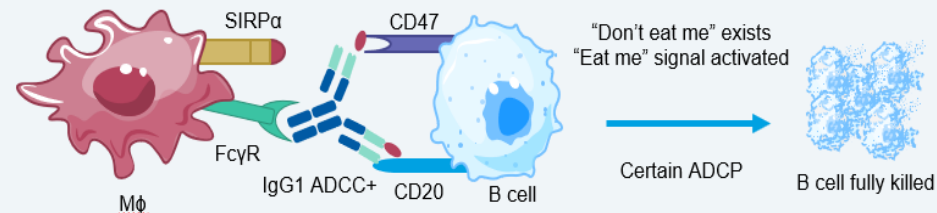


RITUXAN® (rituximab, CD20) was approved by FDA in 2006 for the treatment of **rheumatoid arthritis (RA)**;

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

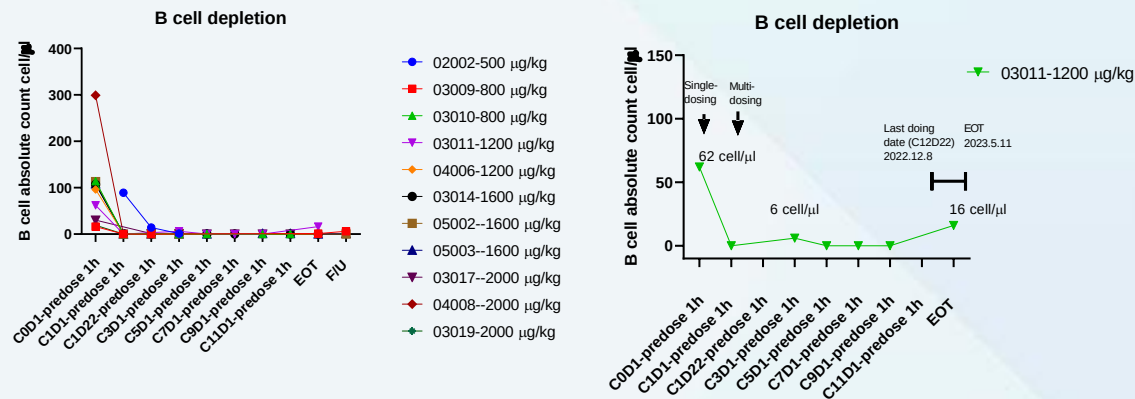
B-cell depletion therapies (BCDTs) are widely used in the treatments of autoimmune diseases.

Mechanism of Action



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

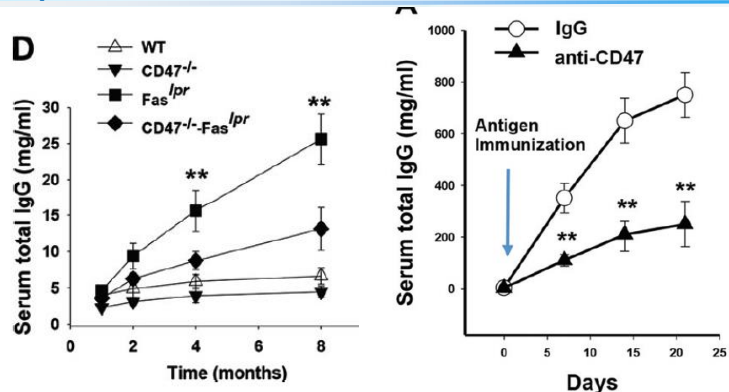
Peripheral B cell depletion by IMM0306 was robust and sustained until three months after treatment withdraw



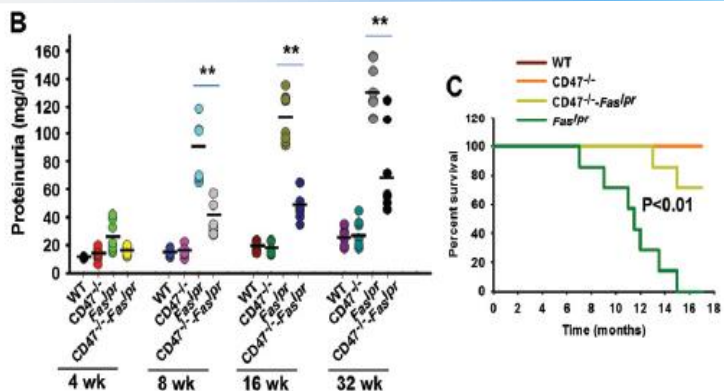
IMC-002 (IMM0306, CD47xCD20/mAb-Trap)

CD47 contributes to pathogenesis of lupus nephritis

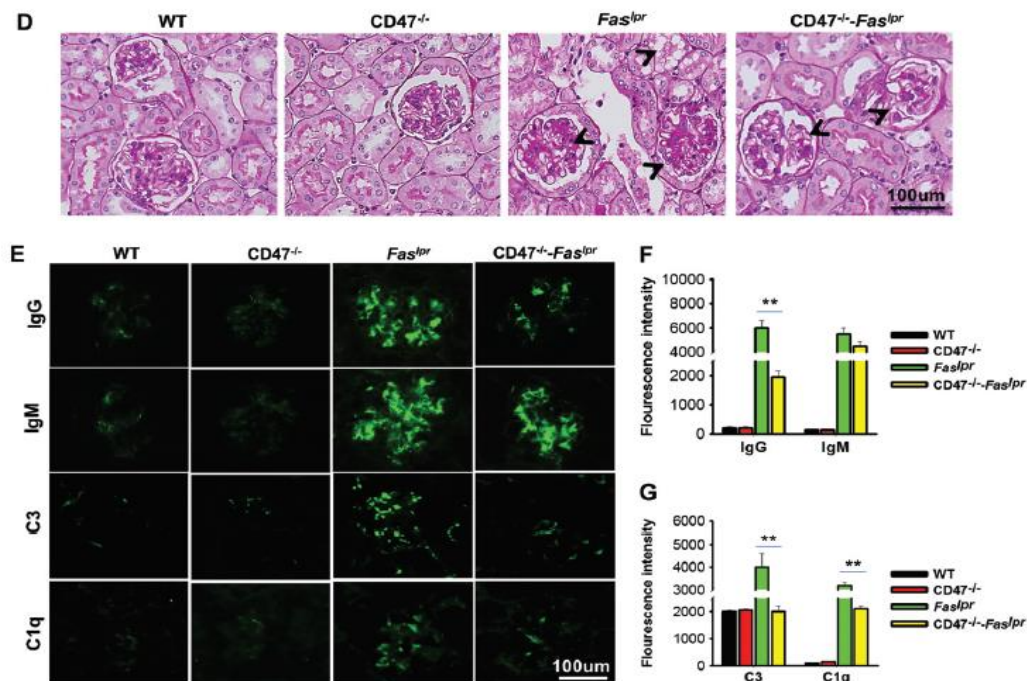
Knockout of CD47 reduces IgG levels and improves lupus nephritis



CD47 knockout in Fas^{lpr} LN disease model resulted in decreased proteinuria levels and prolonged survival



CD47 knockout in Fas^{lpr} 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

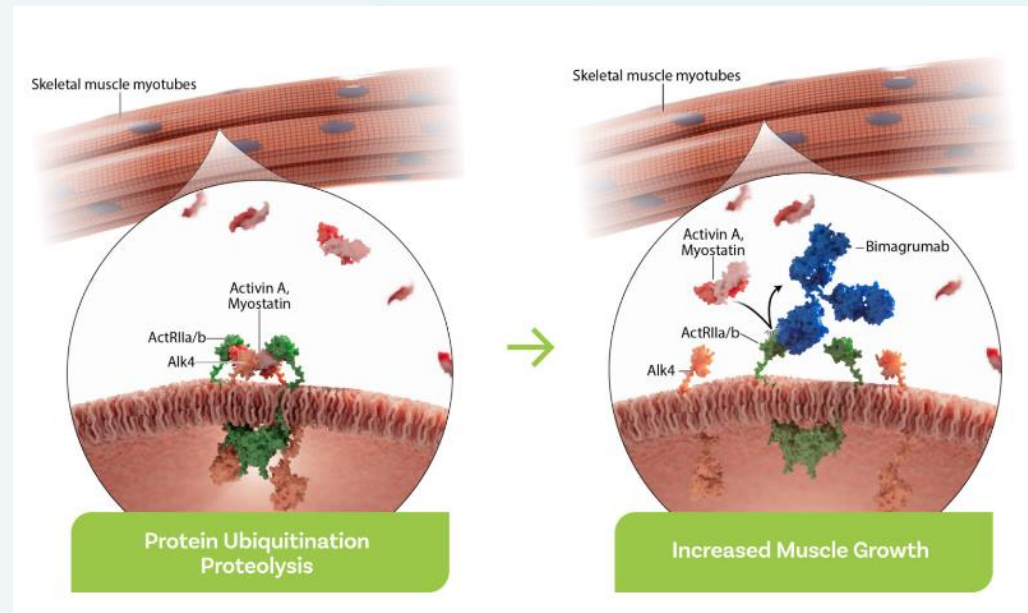
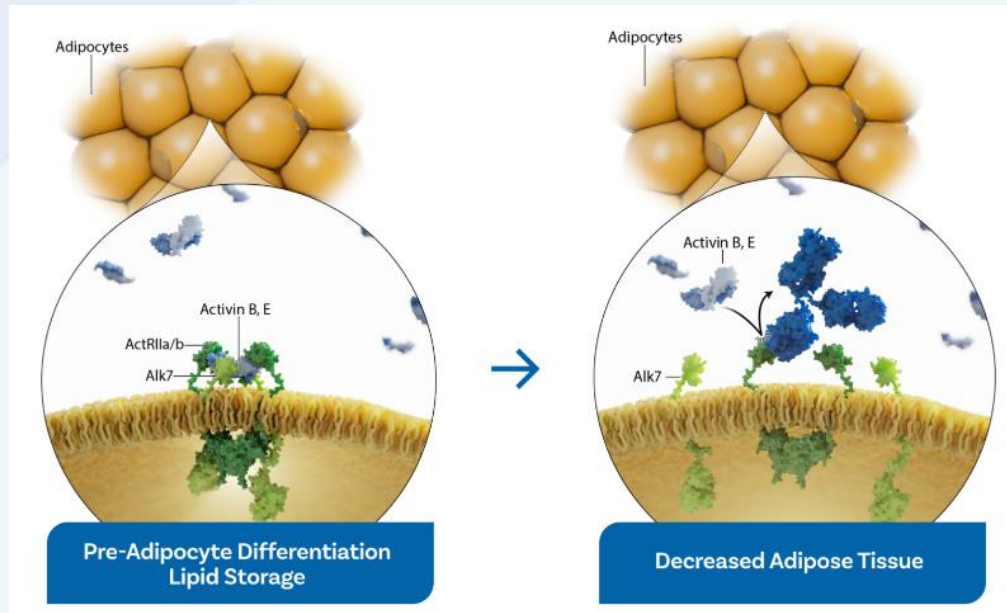

 Activin signaling via ActRII receptors directly promotes lipid storage, acting as a key driver of visceral fat accumulation and obesity

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.

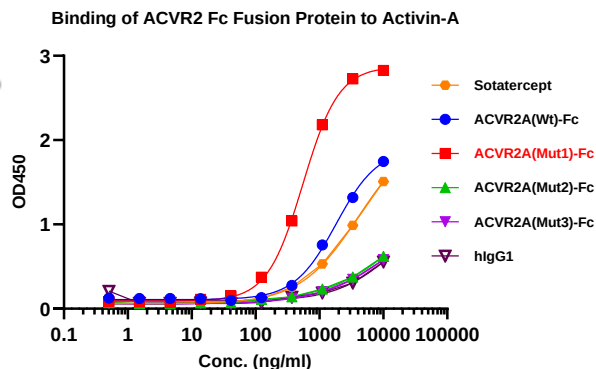
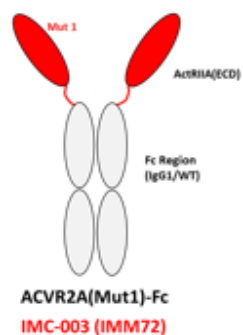


IMC-003 (IMM72, ActRIIA/Fc-fusion)

Preclinical Results



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



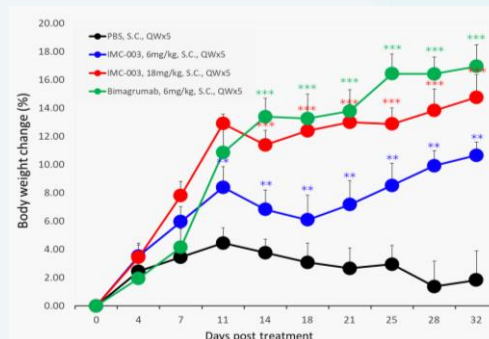
	Sotatercept	ACVR2(WT)-Fc	ACVR2(Mut1)-Fc
EC50	4057	1826	552.3

	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

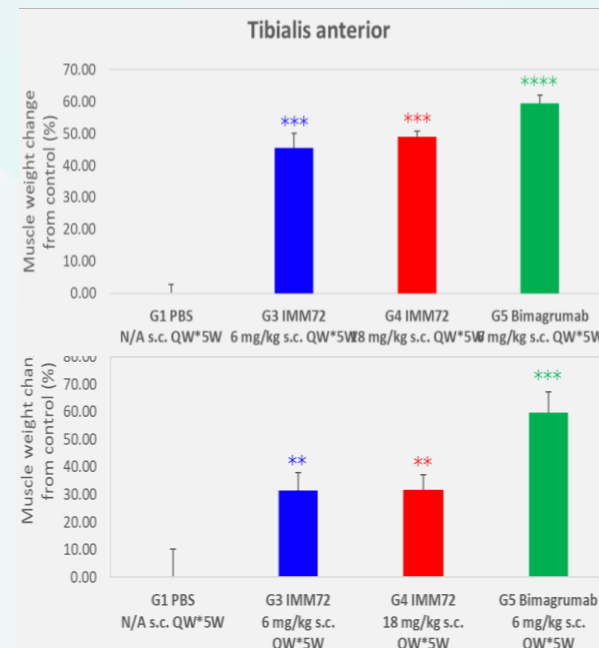


IMC-003 helps build muscle and lose weight

Body weight increased substantially by IMC-003 treatment



Skeletal muscle increased substantially by IMC-003 treatment



Lilly

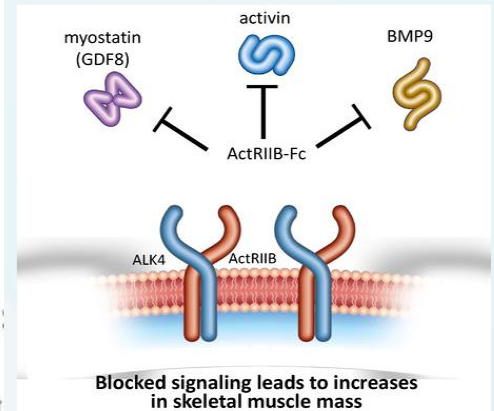
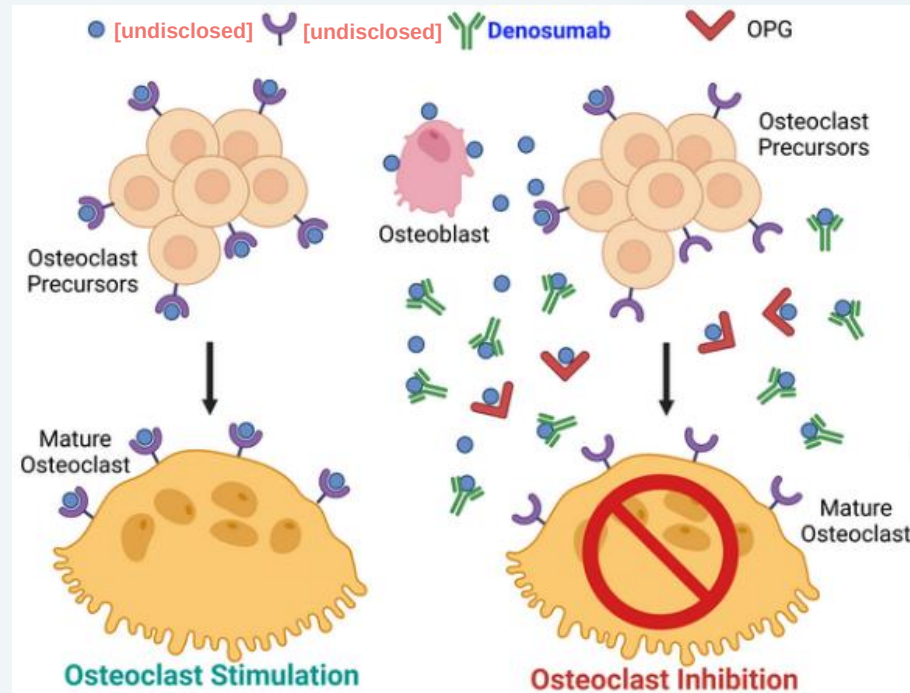
Eli Lilly completed the acquisition of Versanis Bio in up to \$1.925 billion cash in August 2023.

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 (IMM7211c) is expected for the better treatments of osteoporosis and skeletal muscle mass decrease, by blocking both [undisclosed] and Activin A/ActRIIA signaling pathway.

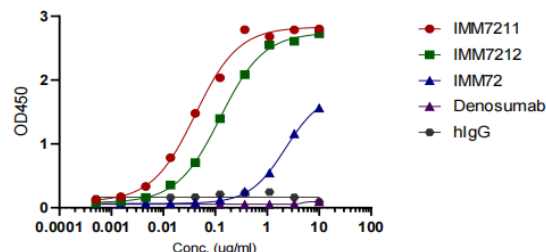


IMC-004 (IMM7211c, [undisclosed] x ActRIIA)

Preclinical Results

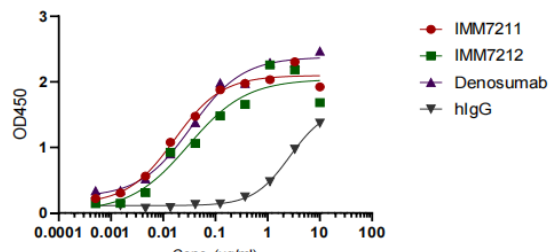

IMC-004 (IMM7211) has stronger binding and blocking capacity than IMM7212 on Activin A and [non-disclosed]; and is similar to Denosumab on [non-disclosed]

20230629 Binding of IMM7211 to Activin A by ELISA



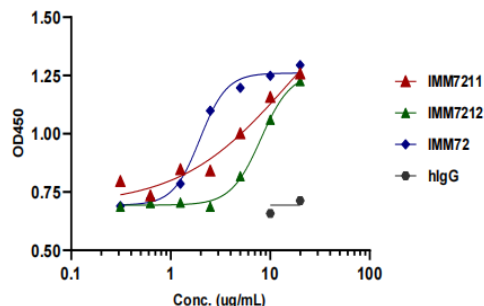
	EC50
IMM7211	0.04139
IMM7212	0.1245
IMM72	2.260

20230629 Binding of IMM7211 to [undisclosed] by ELISA



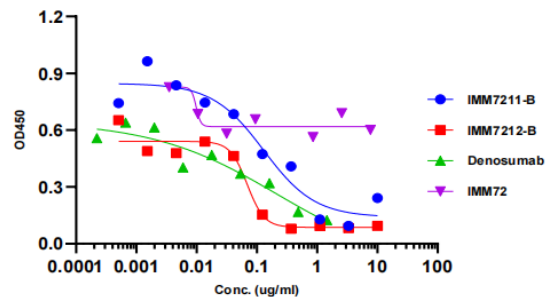
	EC50
IMM7211	0.01644
IMM7212	0.03036
Denosumab	0.03438

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

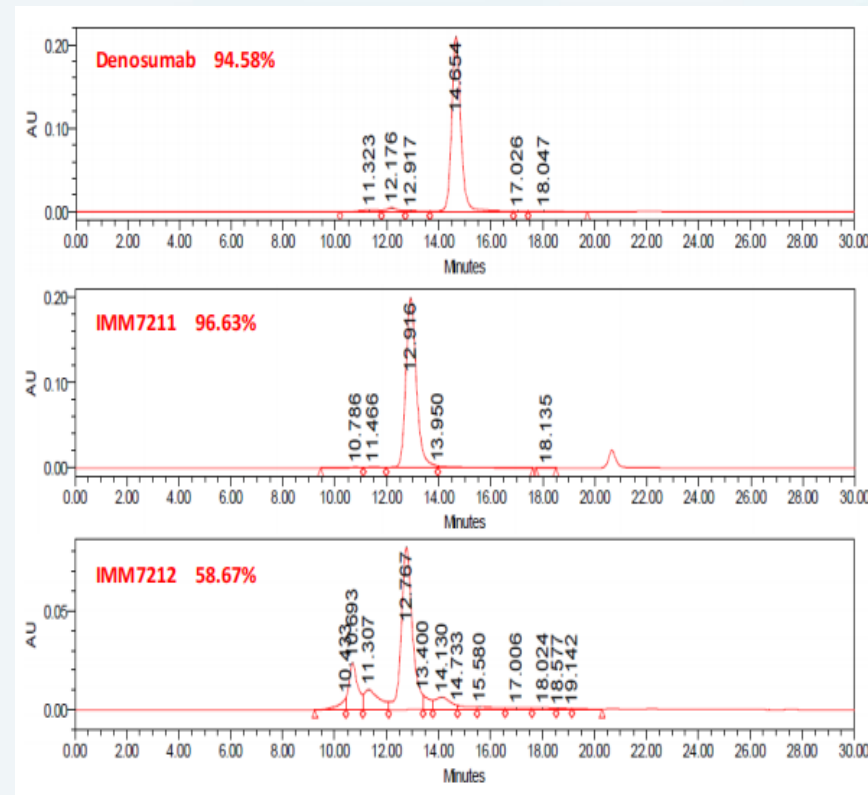


	IMM7211	IMM7212	IMM72
EC50	14.96	8.064	1.965

IMM7211/IMM7212 Blocking the Interaction of [undisclosed]



	IMM7211-B	IMM7212-B	Denosumab
IC50	0.1297	0.06907	0.2263





宜明昂科
ImmuneOnco

SECTION 5

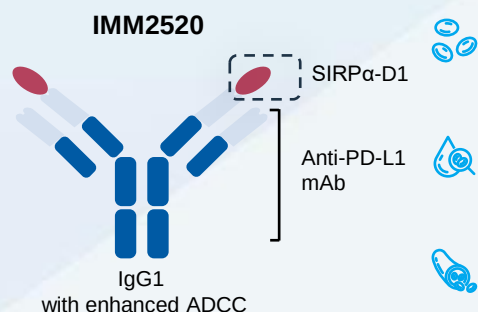
Other Oncology Programs



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 ADCC and ADCC activity**



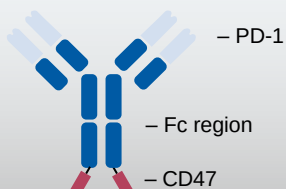
Competition Landscape

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

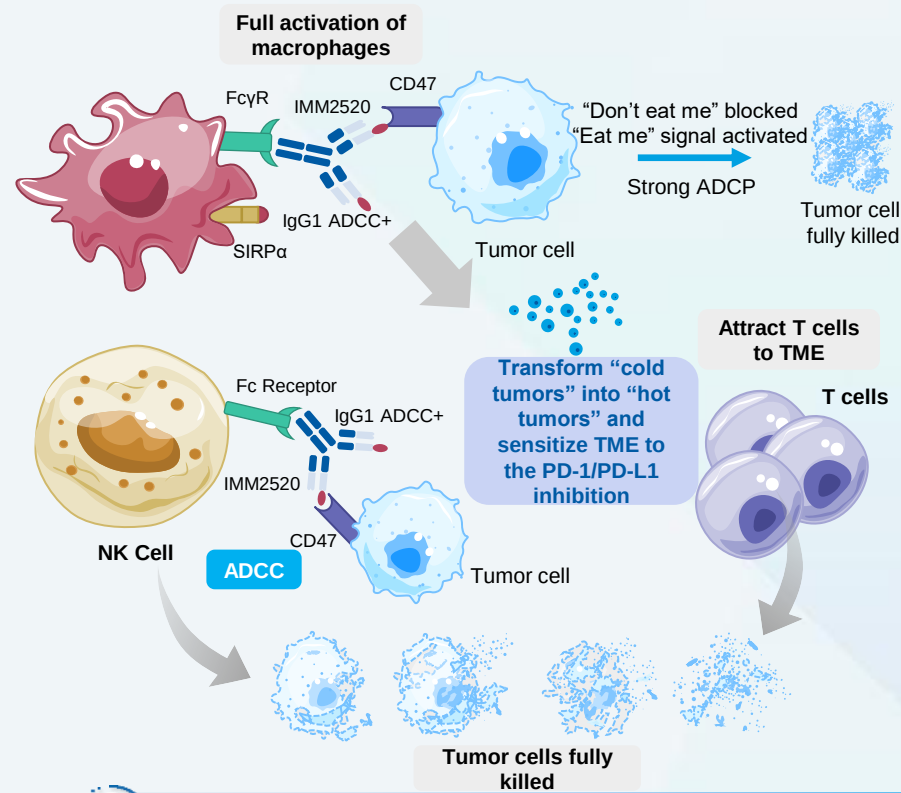


- ✓ Bispecific molecule formats: certain molecules connect the CD47-binding to the Fc end, which could disrupt immune activation resulted from Fc-FcγR engagement
- ✓ IgG4 Fc: several molecules resort to an IgG4 Fc region with weak FcγR engagement

HX009 (Hans Bio)



Mechanism of Action



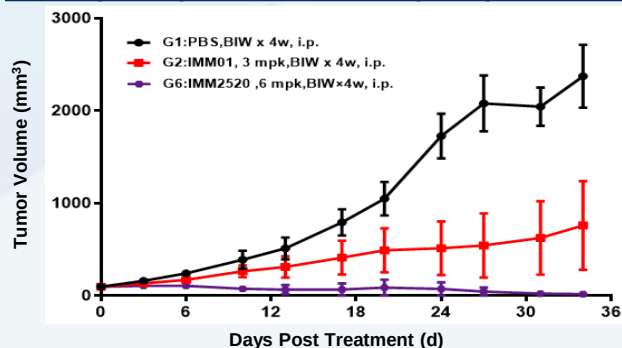
Due to the crosstalk among macrophages, NK cells and T cells, IMM2520 is able to unleash significant synergistic effects

IMM2520 (CD47×PD-L1) (cont'd)



Preclinical Results

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



Note:

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



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

Developing In-house and Own its IP and Commercial Rights



1 issued patent in Japan

1 issued patent in PRC

1 issued patent in the U.S.

Several pending patent applications in Europe, Korea and Brazil



Market Opportunities and Clinical Development Plan

Opportunities

A huge market potential for IMM2520



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

Clinical Development Plan

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

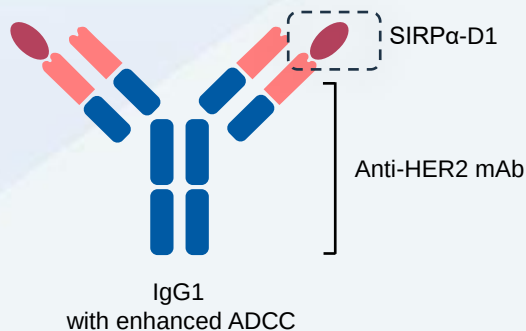
IMM2902 (CD47×HER2)

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



Overview

IMM2902



No RBC binding *in vitro*



Enhanced ADCP and ADCC activity



Accelerated HER2 internalization and degradation

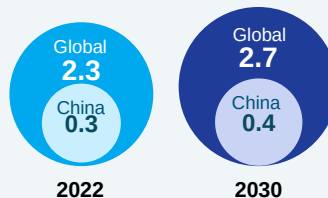


Market Opportunities and Competition

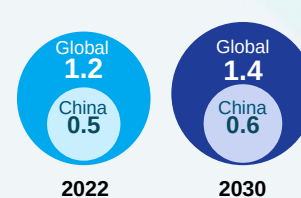


HER2 overexpression is prevalent in many major cancer types, such as BC, GC, lung cancer, CRC, EC, BTC, HNSCC and CC, presenting a **huge market potential**

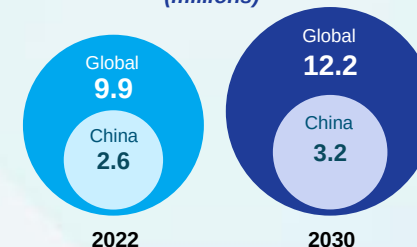
Incidence of BC (millions)



Incidence of GC (millions)



Incidence of Other Major HER2-expressing Cancers (millions)



HER2-low expression

~50%
of all BC cases

~25%
of all GC cases



IMM2902 could benefit a large patient population globally

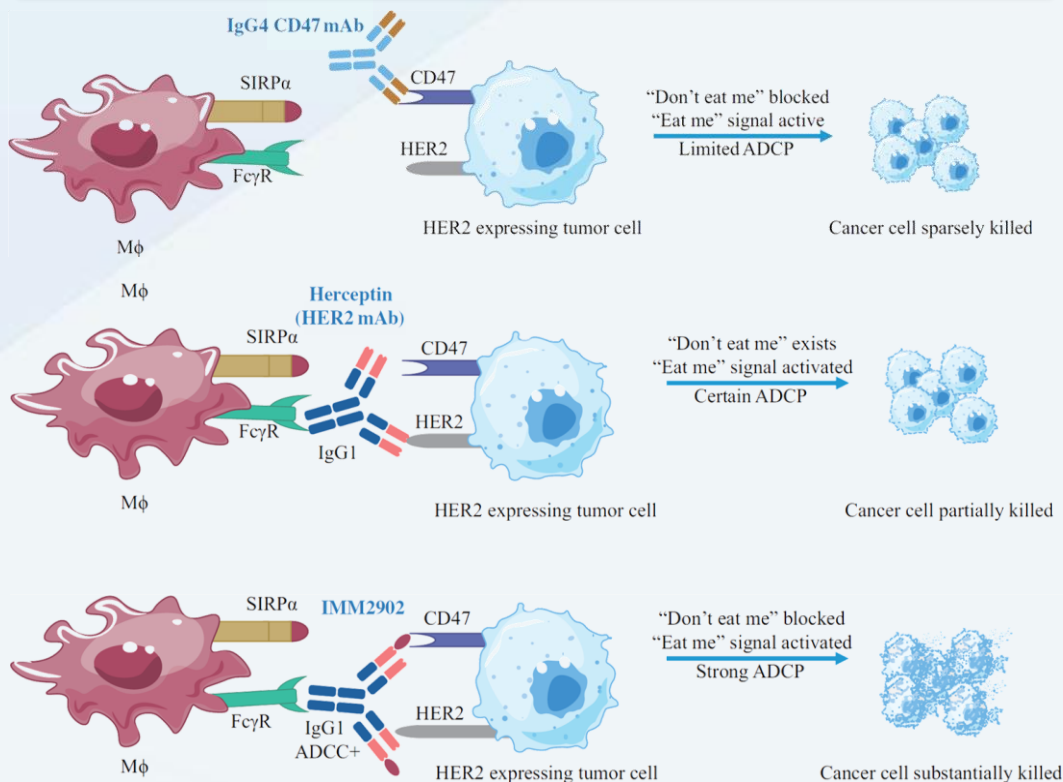
Safer and more efficacious treatment for patients with **HER2-low expressing** solid tumors and those relapsed from trastuzumab treatment

IMM2902 (CD47×HER2) (cont'd)

MoA and Competitive Advantages

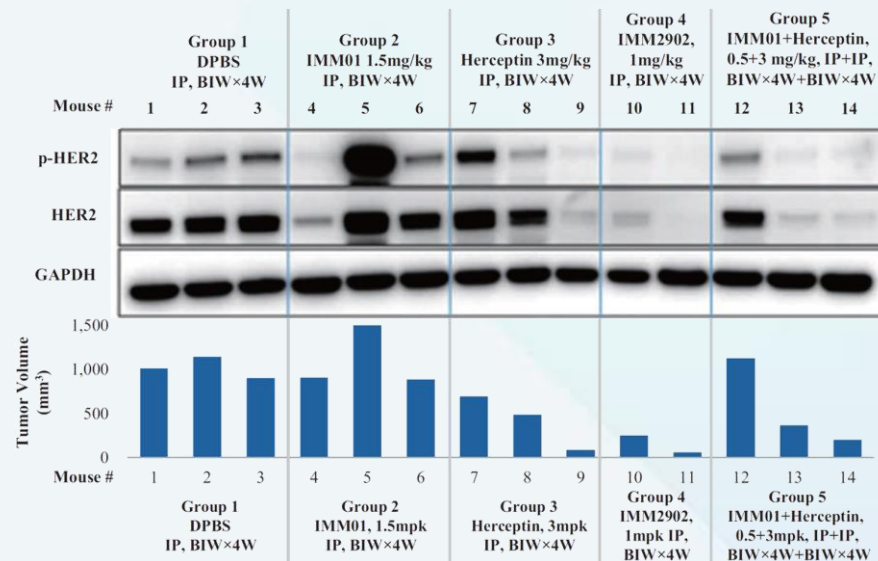


Mechanism of Action



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

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



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

Notes:

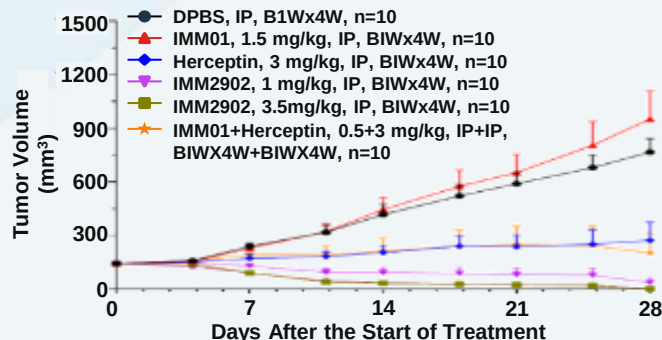
1. p-HER2 refers to phospho-HER2, DPBS refers to Dulbecco's Phosphate Buffered Saline, intended to provide a buffer system for maintaining cell culture media in the physiological range of 7.2 to 7.6.

Source: Company data

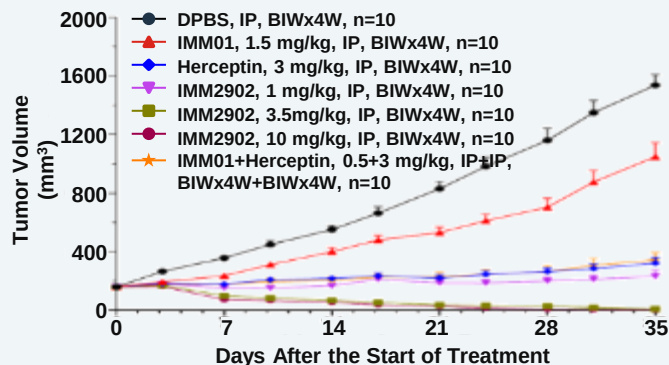
IMM2902 (CD47 $\times$ HER2) (cont'd)

Preclinical Results – Strong *In Vivo* Antitumor Efficacy

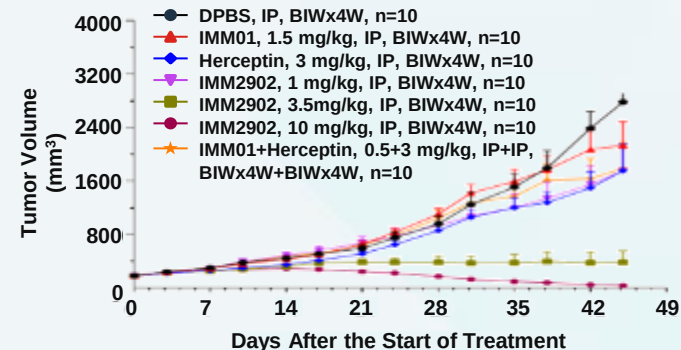
Efficacy Study in Herceptin-sensitive Breast Cancer (BT474) Xenograft Mouse Model



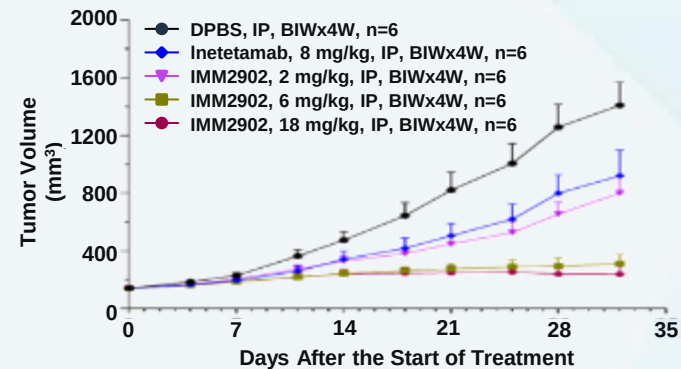
Efficacy Study in Herceptin-sensitive Gastric Cancer (NCI-N87) Xenograft Mouse Model



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



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



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

IMM2902 (CD47×HER2) (cont'd)

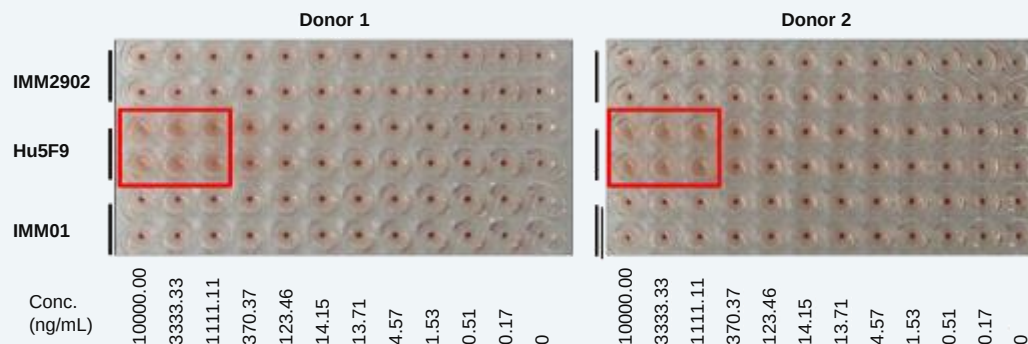
Preclinical Results and Clinical Development Plan



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

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

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



Clinical Development Plan



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



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

Developing In-house and Own IP and Commercial Rights



1 issued patent in the U.S., **1** issued patent in Japan, **1** issued patent in PRC, **1** allowed European patent application (Unitary effect registered & validated in the GB)

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

Notes:

1. Magrolimab analog used in this study was replicated by us based on public information

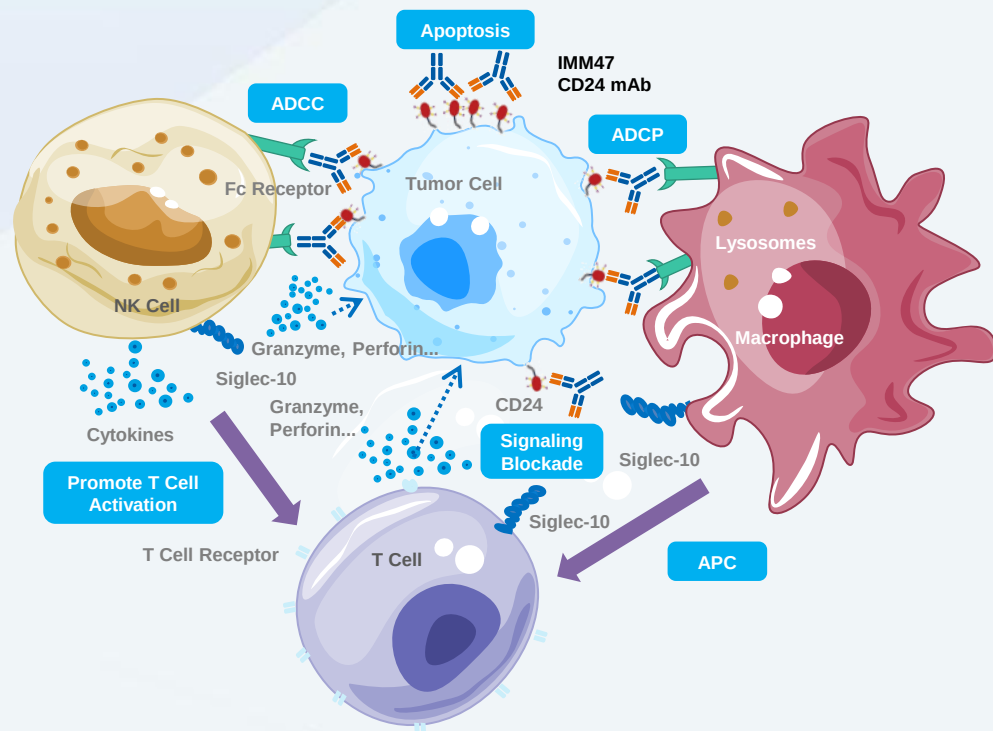
Source: Company data

IMM47 (CD24)

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



IMM47 – Molecule Structure and Mechanism of Action



IMM47 Molecule Structure



CD24 mAb
IgG1 Fc with
Enhanced ADCC

Block CD24/Siglec-10
immune inhibitory signaling

Induce ADCC/ADCP

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

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

Significantly increasing the amount of M1 macrophages in tumor tissues

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

IMM47 (CD24) (cont'd)

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

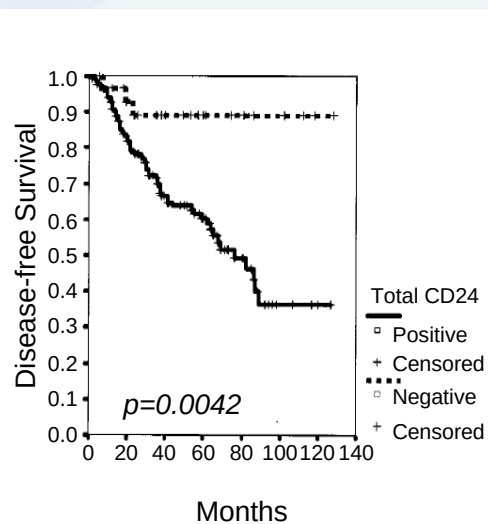


Broadly overexpressed on many types of tumor tissues, including B-cell lymphomas, erythroleukemia, gliomas, SCLC, ESCC, HCC, CCA, PAAD, UC, OC, BC, primary NECs, and PC¹

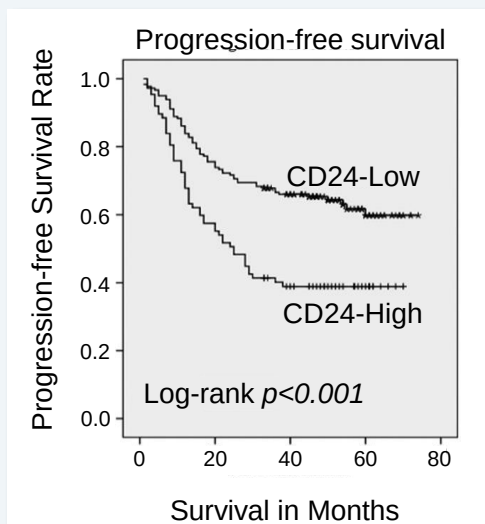


Recognized as an important marker for poor prognosis of those cancers, presenting a huge market potential

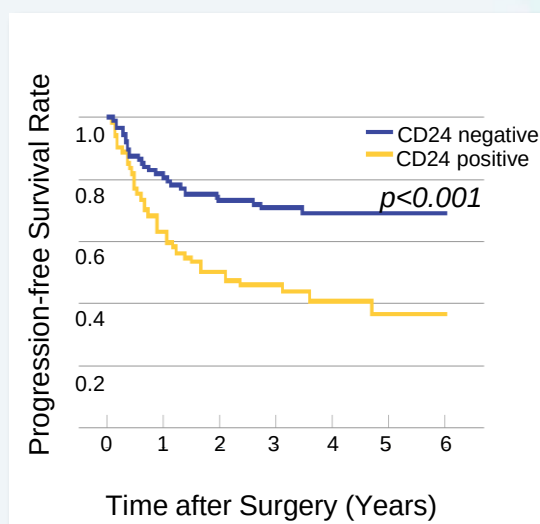
Primary Breast Cancer²



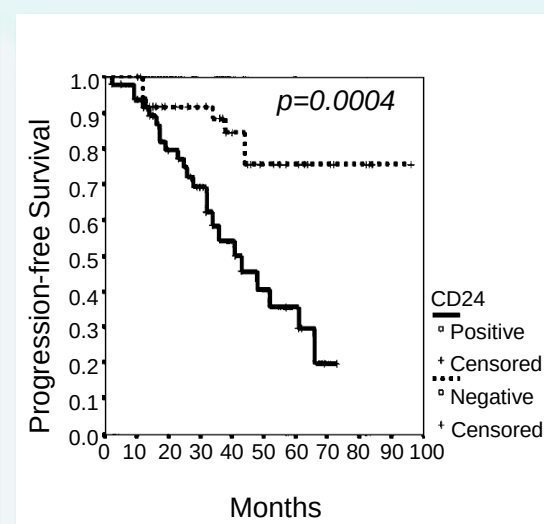
NSCLC³



ESCC⁴



Prostate Cancer⁵



Source:

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

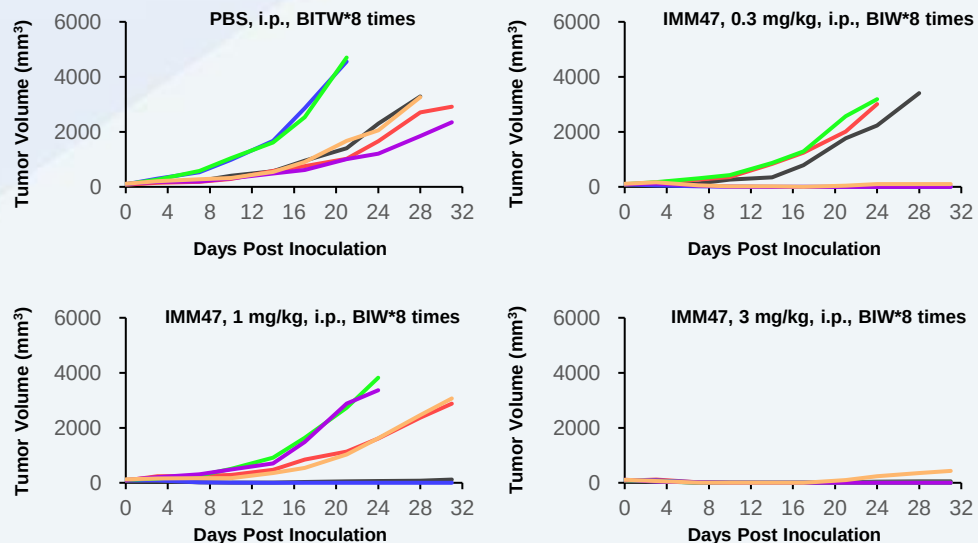
IMM47 (CD24) (cont'd)

Competitive Advantages



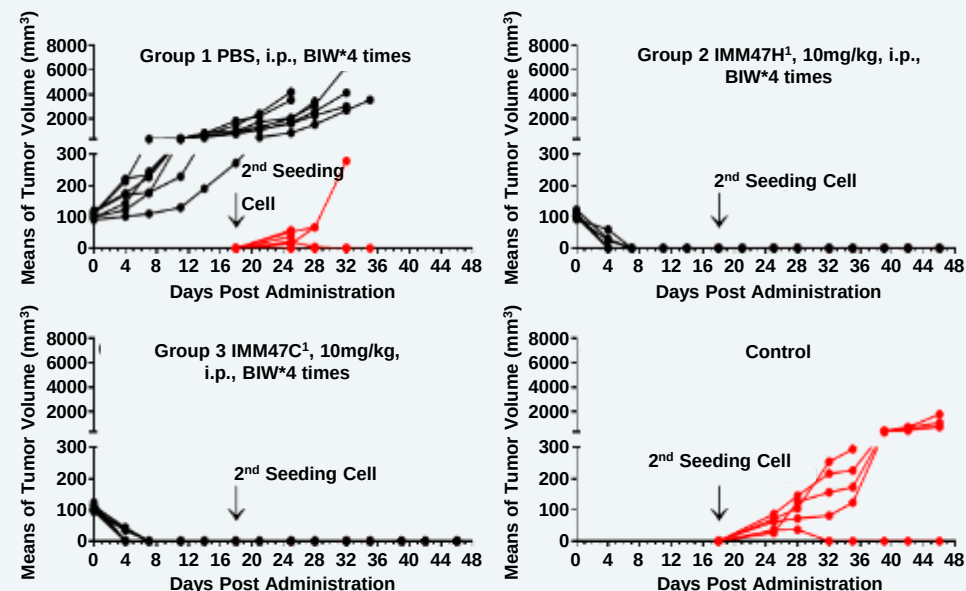
Proof-of-Concept Study in Colon Cancer (MC38-hCD24) Syngeneic Model in hSiglec-10 Tg C57BL/6 Mice

Compelling Tumor Killing Capabilities



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

Complete Tumor Eradication



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

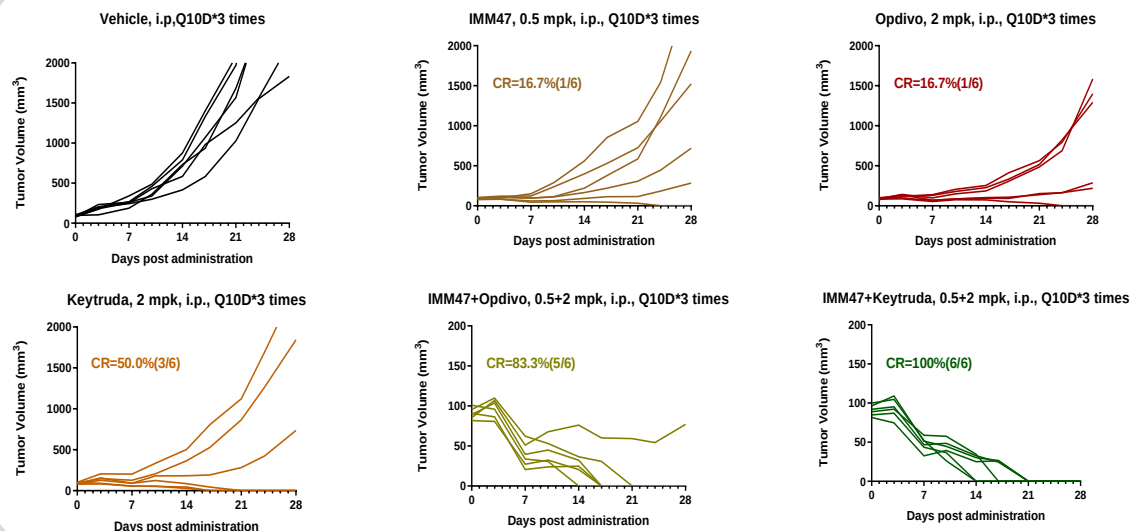
Notes:
1. IMM47C is a previous chimeric version of IMM47 and IMM47H is an earlier fully humanized version of IMM47. IMM47 revealed highly similar *in vitro* efficacy as IMM47C and IMM47H, and was eventually selected for further development

IMM47 (CD24) (cont'd)

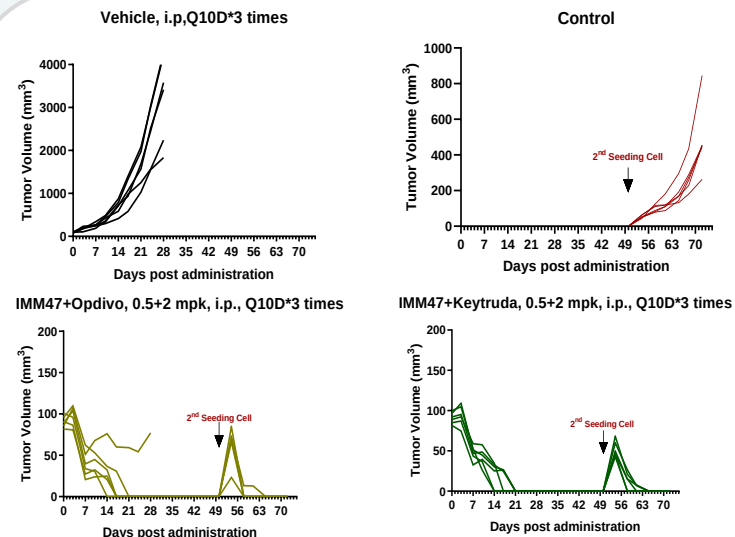
Strong synergy between IMM47 and PD-1 mAb



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



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



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

IMM47 (CD24) (cont'd)

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



IMM47 - Competition Landscape



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



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



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

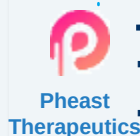
Drug Name	Target	Modality	Clinical Stage
IMM47	CD24	mAb	IND Enabling
IMM4701	CD47 x CD24	Bispecific	Preclinical
IMM2547	CD24 x PD-L1	Bispecific	Discovery



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



Recent Catalysts: Validation from Industry Veterans and Pioneers



Founders

- Dr. Amira Barkal
- Dr. Irving Weissman

Latest Financing

Series-A: US\$76MM
(with an estimated valuation of US\$304-456MM)

Key Financial Investors



Strategic/CVC/ Research Institutes



Clinical Development Plan

2023

File IND applications with the NMPA and FDA

Sep 2023

Initiated clinical trial first in Australia (FPI)

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

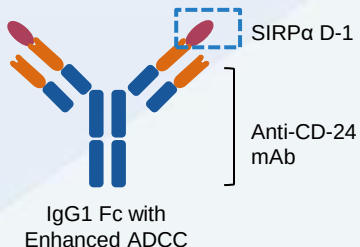
IMM4701 (CD47 × CD24)

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



IMM4701 - Mechanism of Action

IMM4701 Molecule Structure



Activating key innate and adaptive immune responses



Enhancing the synergistic crosstalk



IMM4701 - Competitive Advantage



Undisputable Global Leader



Carefully Designed Clinical Roadmap



Potent and Synergistic Immune Activation



Promising Combination Partner

Developing One Owned Patent Family



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

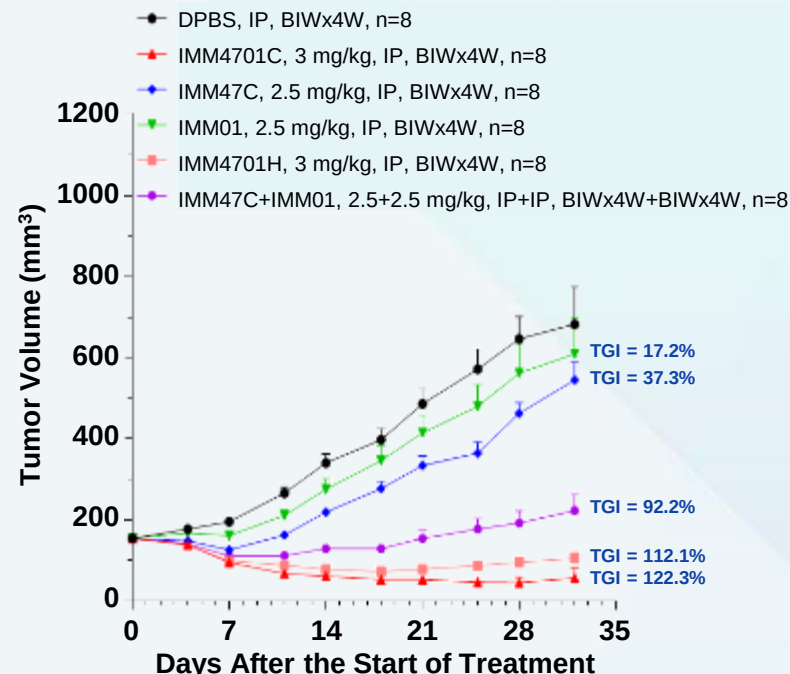
Notes:

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



Strong and Robust Antitumor Activities

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



IMM40H (CD70)

A Humanized IgG1 CD70 mAb with Potential to Combo with IMM01

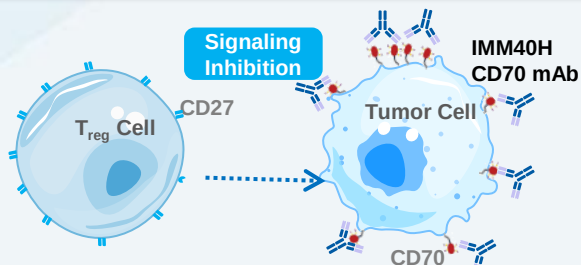


IMM40H – Mechanism of Action

IMM40H Molecule Structure



Humanized IgG1 CD70 mAb with High Target Affinity



Obstructing the activation and proliferation of T_{reg} cells

Inducing immune attacks against tumor cells



IMM40H - Competitive Advantage



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



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

Developing One Owned Patent Family

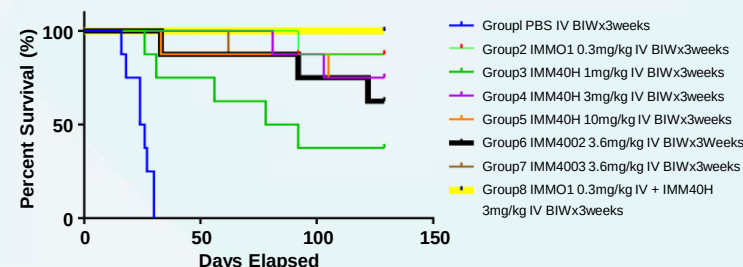
- 1 issued patent the U.S. , 1 issued patent in the PRC
- 1 issued patent in Japan
- 1 pending patent application in Europe



Potent In Vivo Antitumor Effects in Combination with IMM01

Efficacy Study in Lymphoma (Raji) Orthotopic Mouse Model¹

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



Other Pre-clinical Benefits of IMM40H



Stronger CD70-binding affinity than cusatuzumab



Demonstrated potent ADCC, CDC and ADCP activity



Favorable safety profile of IMM40H



Current Status

IND approved by both NMPA and FDA in August 2022



Thank you!

