OVERVIEW

We are a science-driven biotechnology company dedicated to the development of next-generation immuno-oncology therapies. We are one of the few biotechnology companies globally adopting a systematic approach to harness both the innate and adaptive immune systems. Currently approved immunotherapies primarily focus on the adaptive immune system and are often confronted with limited clinical benefits due to low response rates and inevitable drug resistance and/or relapse in many cancer indications. Harnessing both the innate and adaptive immune systems allows us to overcome the limitations of current T-cell-based immunotherapies and address significant unmet medical needs of cancer patients.

We have developed a robust pipeline of over ten innovative drug candidates with eight ongoing clinical programs, anchored by a deep and broad innate-immunity-based asset portfolio. Our pipeline reflects our deep insight into the frontiers of cancer biology and immunology, and our expertise in turning scientific research into promising drug candidates. We continue to advance the drug development targeting innate immune checkpoints in cancer and we believe that the introduction of these novel therapies into the field of cancer immunotherapy will lead to robust and durable treatment responses. Our founder, Dr. Wenzhi Tian, began to explore the therapeutic potential of CD47 blockade in 2010, long before this innate immune checkpoint became widely recognized and clinically validated in the biopharmaceutical industry. Based on our comprehensive understanding of the biology underlying CD47-SIRPα interaction and its potential synergy with other tumor targets and/or immune checkpoints, we have built a differentiated CD47-based portfolio with favorable safety and promising efficacy profiles since our inception in 2015. In addition to CD47, we have selected and validated another promising innate immune checkpoint, CD24, in recent years. Around CD24, we are developing one IND-enabling-stage and several discovery- and preclinical-stage drug candidates, each with the potential to become the first of its class to enter into clinical stage around the world. Moreover, we are also developing drug candidates that target other promising innate and adaptive immune checkpoints, including IL-8, NKG2A and PSGL-1, to maximize the clinical and commercial value of our platform.

Our continuous innovation is driven by an experienced and stable R&D team led by Dr. Tian. Core members of our R&D team have been working with Dr. Tian for over 10 years and possess multi-disciplinary expertise in drug discovery, design and development. Emulating the "Quality-by-Design (QbD)" concept that is intended to improve drug product quality by using analytical and risk-management methodologies, we created the "Drug-by-Design (DbD)" concept that emphasizes the fundamental role of molecule design rationale in the process of large molecule drug development. This concept requires that the structure of every drug molecule be deliberately designed with a sound scientific rationale predicated on target-specific biological functions and validated in preclinical studies. Under the guidance of our "DbD" concept and the leadership of Dr. Tian, we have built a fully-integrated R&D platform. It features our proprietary technologies and know-how (including our mAb-Trap bispecific antibody platform technology) and encompasses all key functionalities throughout the innovative drug development process.

Strictly adhering to the "DbD" concept and leveraging our R&D platform, we have designed and developed a rich pipeline that aims to unlock not only the full power of the largely untapped innate immune system, but also the synergistic potential of harnessing the innate and adaptive immune systems at the same time. The following chart summarizes the development status of our selected drug candidates as of the Latest Practicable Date:

	Program	Target (Modality)	Indication(s)	Discovery	Preclinical	IND/IND-Enabling	Phase Ia/I	Phase Ib/II	Phase III/Pivotal	Current Status / Upcoming Milestone[1]	Commercial Rights
Innate Immunity	**IMM01**	CD47 (SIRPα-Fc fusion protein)									
	IMM01 + Azacitidine		MDS, AML, CMML[2]			China (NMPA)				Phase Ib/II commenced in January 2022; expect to initiate pivotal trial in Q4 2023	Global
	IMM01 + Tislelizumab	CD47+PD-1	cHL, Solid tumor			China (NMPA)				Phase Ib/II commenced in May 2022; expect to initiate pivotal trial in Q3 2024[3]	Global
	IMM01 + Inetetamab	CD47+HER2	HER2-positive solid tumors			China (NMPA)[4]				Phase Ib/II IND approved	Global
	IMM01 + Bortezomib + Dexamethasonum	CD47	MM			China (NMPA)				Phase Ib/IIa IND approved	Global
	IMM0306 Monotherapy	CD47xCD20 (Bispecific)	Indolent B-NHL			China (NMPA), US (FDA)				Phase IIa commenced in March 2023 in China; IND approved in the U.S.	Global
	IMM0306 + Lenalidomide	CD47xCD20 (Bispecific)	B-NHL			China (NMPA)				Phase Ib/IIa IND approved	Global
	IMM2902	CD47xHER2 (Bispecific)	HER2-positive and low-expressing solid tumors			China (NMPA), US (FDA)				Phase I commenced in February 2022 in China and in June 2022 in the U.S.; expect to largely complete Phase Ia trials in China and the U.S. in 2023	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
	IMM47	CD24 (mAb)	Solid tumors			China (NMPA), US (FDA)				IND-enabling; expect to enter into clinical trials in mid-2023	Global
	IMM4701	CD47xCD24 (Bispecific)	Solid tumors							CMC	Global
	IMM2547[5]	CD24xPD-L1 (Bispecific)	Solid tumors							Discovery	Global
	IMM51[5]	IL-8 (mAb)	Solid tumors							Preclinical	Global
	IMM38[5]	Undisclosed	Solid tumors							Preclinical	Global
	IMM50[5]	Undisclosed	Solid tumors							Discovery	Global
	IMM62[5]	Undisclosed	Solid tumors							Discovery	Global
Adaptive Immunity	**IMM2510**	VEGFxPD-L1 (Bispecific)	Solid tumors			China (NMPA)				Phase I commenced in August 2021 and 8th cohort ongoing in China; expect to complete Phase I in mid-2023	Global
	IMM27M	CTLA-4 ADCC+ (mAb)	Solid tumors			China (NMPA)				Phase I commenced in June 2022 in China; expect to complete in mid-2023; IND approved in China for Phase Ib/II trial for its combination with a PD-1 antibody[6]	Global
	IMM40H	CD70 (mAb)	Liquid/Solid tumors			China (NMPA), US (FDA)				IND approved in China and the U.S. in August 2022	Global

★ Core Product ▲ Key Product

Innate Immunity Targets | Innate and Adaptive Immunity Targets | Adaptive Immunity Targets

Notes:

(1) Expected completion date for Phase Ia/I trial refers to the time when RP2D can be determined, and expected completion date for Phase Ib/II trial refers to the time when top-line data is available for regulatory discussions. Follow-up period required would not delay the initiation of the next phase clinical trials, and is thus not considered.

(2) The cohort-expansion trials of this combination are mainly designed to target the first-line treatment of higher-risk MDS (patients who fall into higher-risk group categories in the original or revised International Prognostic Scoring System), unfit AML (individuals of older age with AML who are considered not eligible for intensive treatment approaches), and CMML. Particularly, we plan to seek an accelerated marketing approval through relatively small sample size studies targeting the first-line treatment of CMML, a rare type of disease with highly unmet medical needs.

(3) In July 2022, we obtained the NMPA's consent for adding R/R cHL as an additional expansion cohort into the ongoing combination trial of IMM01 and tislelizumab. We dosed the first patient with R/R cHL in China in January 2023.

*(4) The clinical trial is led and funded by Sunshine Guojian Pharmaceutical (Shanghai) Co., Ltd. ("**Sunshine Guojian**"). As denoted by the dotted line, Sunshine Guojian and us have obtained an IND approval for a Phase Ib/II trial of this combination therapy from the NMPA in August 2021, and therefore the parties can skip the Phase Ia stage and directly initiate a Phase Ib/II trial.*

(5) We will continue to conduct preclinical studies for IMM2547, IMM51, IMM38, IMM50 and IMM62, including cell line development, in vivo studies and further evaluation.

(6) We are currently conducting the Phase I trial for IMM27M monotherapy, and have obtained the IND approval for a Phase Ib/II trial for its combination with a PD-1 antibody.

* *Currently we have several other drug candidates in preclinical stage and plan to further develop these candidates through collaboration, such as IMM2518, a second-generation VEGF×PD-L1 bispecific molecule and IMM5601, a CD47×CD38 bispecific molecule.*

Abbreviations: MDS refers to myelodysplastic syndrome; AML refers to acute myeloid leukemia; CMML refers to chronic myelomonocytic leukemia; MM refers to multiple myeloma; B-NHL refers to B-cell non-Hodgkin lymphoma; cHL refers to classical Hodgkin lymphoma; IND refers to investigational new drug; CMC refers to chemistry, manufacturing, and controls; ADCC refers to antibody-dependent cellular cytotoxicity.

Source: Company Data

Our CD47-based drug candidates include:

- **IMM01**, our Core Product, is a next-generation CD47-targeted molecule. It is the first SIRPα-Fc fusion protein to enter into clinical stage in China. IMM01 designed with IgG1 Fc can fully activate macrophages via a dual mechanism — simultaneously blocking the "don't eat me" signal by disrupting CD47/SIRPα interaction and delivering the "eat me" signal through the engagement of activating Fcγ receptors on macrophages. Furthermore, the CD47-binding domain of IMM01 was specifically engineered to avoid human red blood cell (RBC) binding. With the differentiated molecule design, IMM01 has achieved a favorable safety profile and demonstrated its ability to potently activate macrophages. We are actively evaluating IMM01 in several ongoing and planned clinical trials:

  — **Monotherapy**: We have completed a Phase I dose-escalation study evaluating IMM01 in relapsed or refractory (R/R) lymphoma. IMM01 has demonstrated encouraging results in safety and efficacy in this trial as a single agent. Among 27 evaluable patients receiving 0.003 mg/kg to 2.0 mg/kg IMM01 in the dose-escalation study, two patients achieved complete response (2 CRs), one achieved partial response (1 PR), and 13 achieved stable disease (13 SDs) (including six cases with substantial tumor shrinkage observed). Among the six patients receiving an RP2D dose of 2.0 mg/kg in this monotherapy clinical trial, one achieved complete response (1 CR), and four achieved stable disease (4 SDs), resulting in a disease control rate (DCR) of 83% in these previously heavily pre-treated R/R lymphoma patients. Based on the encouraging results of the Phase I dose-escalation study, we initiated clinical trials evaluating combination therapies of IMM01 and other drugs, including each of azacitidine and tislelizumab.

  According to Frost & Sullivan, among numerous drug developers of CD47-targeted molecules globally, we are one of the only two companies to have observed CR in monotherapy clinical trials with a well tolerated safety profile. With encouraging efficacy and favorable safety in monotherapy clinical trials and robust preclinical data of its combination studies, IMM01 is expected to achieve strong synergistic effects used in combination with other cancer agents, and therefore we plan to prioritize the clinical development of IMM01 for combination use.

  — **Combination of IMM01 and azacitidine:** As validated by multiple publicly reported clinical trials, the combination of CD47-targeted therapies and azacitidine can generate synergistic tumor-killing effects. However, since azacitidine also induces blood toxicity, its combination with CD47 antibodies (which also cause blood toxicity) may lead to exacerbated blood toxicity and serious safety issues. In contrast, based on the initial data from our ongoing Phase Ib/II clinical trial, IMM01 presents strong potential to be a combination partner with azacitidine because of its dual mechanisms and favorable safety profile. IMM01 is also safer than CD47 antibodies partly due to the significantly lower dose required (2.0 mg/kg), as compared to the typical dose of 30.0 to 45.0 mg/kg required for CD47 antibodies.

  We are evaluating the combination of IMM01 and azacitidine for the first-line treatment of higher-risk (HR) myelodysplastic syndromes (MDS), unfit acute myeloid leukemia (AML), and chronic myelomonocytic leukemia (CMML). Upon completion of the Phase Ib trial, we initiated the Phase II trial of this combination mainly for the first-line treatment of HR MDS, unfit AML and CMML in China in June 2022. Interim data as of February 10, 2023 from the Phase Ib/II trial has demonstrated favorable safety and promising efficacy profile. Neither DLT nor hemagglutination was observed among all 12 patients receiving the combination

treatment at all three dose levels of IMM01 (1.0 mg/kg, 1.5 mg/kg, and 2.0 mg/kg) in our Phase Ib trial. Moreover, the interim data obtained from our Phase II trial as of February 10, 2023 has demonstrated that: (i) among the eight evaluable patients with 1L CMML, two reached complete response (2 CRs), six reached marrow complete response (6 mCRs), with one hematological improvement (1 HI, which also achieved mCR), resulting in an overall response rate (ORR) of 100%, and (ii) among the 16 evaluable HR MDS patients who have received at least three cycles of treatment, three achieved CR (3 CRs), nine achieved mCR (9 mCRs), and seven achieved HI (7 HIs, among which 4 also achieved mCR), resulting in an ORR of 93.8%. We expect to commence a pivotal trial in China in the fourth quarter of 2023. In particular, we plan to seek an accelerated marketing approval through relatively small sample size studies targeting the first-line treatment of CMML, a rare type of disease with highly unmet medical needs. Subject to further clinical validation, we plan to file an IND application with the FDA for a Phase II study of this combination treatment.

— **Combination of IMM01 and tislelizumab:** Unlike CD47 antibodies that often employ an IgG4 Fc region, IMM01 is designed with IgG1 Fc that can fully activate macrophages by activating an additional "eat me" signal through Fc-FcγR engagement. Activated macrophages can then secrete certain cytokines and chemokines to recruit T cells to tumor sites, thus effectively converting "cold tumors" (tumors that lack T-cell infiltration) into "hot tumors" that are more responsive to the treatment of PD-1/PD-L1 inhibitors. As macrophages are widely distributed and highly infiltrated in tumor tissues of various cancers, this combination has the potential to treat a broad range of solid tumors. Our preclinical studies have demonstrated promising synergistic antitumor effects for the combination of IMM01 with either PD-1 or PD-L1 inhibitors. We intend to develop the combination therapy of IMM01 and tislelizumab for the treatment of solid tumors that are refractory or relapsed from the standard of care such as PD-1/PD-L1 inhibitors, including among others, non-small cell lung cancer (NSCLC), small cell lung cancer (SCLC), head and neck squamous cell carcinomas (HNSCC) and colorectal cancer (CRC). We are currently evaluating IMM01 and tislelizumab in a Phase II trial in various advanced solid tumors that failed to respond to or relapsed from the standard of care such as PD-1/PD-L1 inhibitors. We dosed the first patient for the Phase Ib trial in May 2022 and initiated the Phase II trial in December 2022. In our Phase Ib trial, a heavily pre-treated NSCLC patient with six lines of prior treatment and refractory to PD-1 inhibitors achieved PR after three cycles of treatment with target lesion shrinkage of 40%. After accumulating more clinical data, we may also further evaluate this combination therapy for the first-line treatment of those solid tumors as well as for the treatment of other cancer indications. We are also evaluating this combination therapy in cHL patients who relapsed or progressed after the treatment of PD-1 inhibitors, which may allow us to pursue an accelerated marketing approval leveraging the results of relatively small sample size studies. In July 2022, we obtained the NMPA's consent for adding R/R cHL as an additional expansion cohort into the ongoing combination trial of IMM01 and tislelizumab. We dosed the first patient with R/R cHL in China in January 2023. We expect to initiate a pivotal trial in the third quarter of 2024.

— **Combination of IMM01 and other drugs:** IMM01 has demonstrated a promising efficacy and safety profile in its Phase I monotherapy trial, which sets the stage for its combination use with other immunotherapies or targeted therapies. We are currently exploring therapeutic potential of IMM01 in combination with various other drugs for a range of cancer indications. We reached a collaboration with Sunshine Guojian, under which Sunshine Guojian will be primarily responsible for

driving and funding the clinical development of the combination treatment of IMM01 and CIPTERBIN® (inetetamab, a HER2 mAb) for HER2-positive solid tumors in mainland China. We are also conducting numerous preclinical studies to evaluate the combination use of IMM01 with other drugs. These combination studies have revealed strong synergistic potential in our mouse models.

Developed on our mAb-Trap platform, our CD47-based bispecific molecules share a common structure: connecting the same engineered CD47-binding domain used in IMM01 to a base antibody with antibody-dependent cellular cytotoxicity (ADCC)-enhanced human IgG1 Fc fragment. This unique structural design with the engineered CD47-binding fragment allows our CD47-based bispecific molecules to avoid RBC binding, thus enabling the adoption of an ADCC-enhanced IgG1 Fc fragment capable of inducing full macrophage activation and much improved antibody-dependent cellular phagocytosis (ADCP) and ADCC activity, which results in stronger antitumor immune responses compared to most IgG4-based CD47 bispecific antibodies. When designing these molecules, we connect the engineered CD47-binding domain to the N-terminal of the heavy chain or light chain of a base antibody against another tumor target rather than to the Fc end, which ensures undisrupted binding to CD47 and preserves the intact Fc region with full immune effector function. Compared to combination therapies, our bispecific molecules targeting the same set of targets have demonstrated stronger antitumor activity at comparable dose levels in our preclinical studies. Our CD47-based bispecific molecules include:

- **IMM0306**, one of our Key Products, is the first CD47×CD20 bispecific molecule globally to enter into clinical stage. IMM0306 has a higher affinity for CD20 than CD47, which enables it to preferentially and simultaneously bind to CD20 and CD47 on malignant B cells rather than CD47-positive normal tissues and further mitigate CD47-related toxicity.

  Our preclinical studies suggest that IMM0306 is more potent than RITUXAN® (rituximab, a 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. We initiated a Phase I trial for IMM0306 in R/R B-cell non-Hodgkin lymphoma (B-NHL) in China in May 2020, of which the preliminary data demonstrated encouraging results in safety and efficacy. According to our initial clinical data as of February 27, 2023, IMM0306 was safe and well tolerated up to 2.0 mg/kg. Among the evaluable patients across four cohorts dosed from 0.8 mg/kg to 2.0 mg/kg, who had relapsed or progressed after receiving rituximab previously, two CRs and five PRs were observed. The only evaluable FL patient at 2.0 mg/kg, who relapsed and progressed after rituximab treatment, has also been confirmed as PR. At 2.0 mg/kg, one patient with primary bone diffuse large B-cell lymphoma (DLBCL) who had four lines of prior treatment has achieved PR with all measurable lesions disappeared after 65 days of treatment. The encouraging clinical results of IMM0306 have provided further validation of our mAb-Trap platform. We commenced a Phase IIa trial for IMM0306 monotherapy for the third- or later-line treatment of FL in March 2023 and plan to seek an accelerated marketing approval through a single-arm trial. We expect to commence pivotal clinical trials in China in the third quarter of 2024. Furthermore, our IND application for the combination of IMM0306 and lenalidomide targeting front-line B-NHL was approved by the NMPA in January 2023, and we are in preparation to commence the Phase Ib trial for this combination in China. We have also received an IND approval for IMM0306 from the FDA in January 2021. With further clinical validation in the Phase I trial in China, we will then decide on our clinical development and collaboration strategy for IMM0306 in the U.S.

- **IMM2902**, one of our Key Products, is currently the only CD47×HER2 bispecific molecule that has entered into clinical stage globally. By simultaneously binding to HER2 and CD47, IMM2902 suppresses tumor cell growth and proliferation through the

blockade of HER2 and CD47/SIRPα inhibitory signals as well as the promotion of HER2 degradation, and further destroys tumor cells through enhanced ADCP, ADCC, and potentially antibody dependent cellular trogocytosis (ADCT). Our preclinical studies demonstrated strong antitumor activities of IMM2902 in a variety of breast and gastric tumor models, including those with HER2-low expression and resistant to HERCEPTIN® (trastuzumab). We are conducting a Phase Ia/Ib clinical trial in China to evaluate IMM2902 in advanced HER2-positive and HER2-low expressing solid tumors, including breast cancer (BC), gastric cancer (GC), NSCLC and biliary tract cancer (BTC), with the first patient dosed in February 2022. IMM2902 was shown to be safe and well tolerated up to 2.0 mg/kg. Dosing is ongoing for higher dose level cohorts. We have also initiated the clinical trial for advanced HER2-positive and HER2-low expressing solid tumors in the U.S. with the first patient dosed in June 2022. We have received the Fast Track Designation from the FDA in July 2022.

- **IMM2520**, one of our Key Products, is a CD47×PD-L1 bispecific molecule for the treatment of solid tumors. By targeting CD47 and PD-L1 on tumor cells and with its functional IgG1 Fc, IMM2520 can simultaneously activate macrophages and T cells to achieve strong synergistic effects and induce long-lasting tumor-specific immune responses. IMM2520 showed encouraging *in vivo* efficacy and safety in several animal models. We have obtained IND approvals for IMM2520 from the NMPA in November 2022 and from the FDA in December 2022, and dosed the first patient for the Phase I clinical trial in China in March 2023. We will primarily focus on the solid tumors that are generally resistant or not sensitive to the currently available immunotherapies, such as CRC, GC, lung cancer and HNSCC, among others.

In addition to CD47, we have selected and validated another promising innate immune checkpoint, CD24. We started the discovery research on CD24 as early as 2019, and have successfully identified lead drug candidates with potent target activity and *in vivo* therapeutic efficacy. Currently, we have one innovative IND-enabling-stage humanized monoclonal antibody (IMM47) and several discovery- and preclinical-stage bispecific molecules, including IMM4701 and IMM2547, targeting this checkpoint. CD24 is widely expressed in numerous types of solid tumors, including BC, NSCLC, CRC, HCC, renal cell carcinoma (RCC), and ovarian cancer (OC), and has been recognized as an important marker for those poor prognosis of these cancers, presenting tremendous clinical potential. However, there is currently no approved or clinical-stage drug candidate targeting CD24 globally, according to Frost & Sullivan.

- **IMM47** is a potentially global first-in-class humanized monoclonal antibody targeting CD24 for cancer treatment. We have successfully screened IMM47 despite the fact that the screening of antibodies against CD24 is highly challenging due to the relatively weak immunogenicity resulting from its small extracellular domain. With a high affinity for CD24 expressed on tumor cells, IMM47 can suppress the immune inhibitory signals sent from CD24/Siglec-10 pathway to macrophages, natural killer (NK) cells and T cells. With the ADCC-enhanced IgG1 Fc, IMM47 can specifically bind to CD24 and potently activate macrophage and NK cell-immune responses through ADCP and ADCC. IMM47 has also been shown to significantly increase the amount of M1 macrophages in tumor tissues in our *in vivo* proof-of-concept studies. It can also activate and promote T-cell response likely through tumor antigen presentation by activated macrophages to T cells and direct blockade of CD24/Siglec-10 inhibitory signals. Our preclinical studies have demonstrated promising efficacy of IMM47. In a colon cancer model, it completely eradicated subcutaneously inoculated tumor cells in all six mice after three doses of 3.0 mg/kg (~0.3 mg/kg human equivalent dose). In addition, IMM47 can establish tumor-specific immune responses that prevent tumor growth even against re-inoculation of tumor cells in mice, demonstrating its capability to further induce T-cell-based adaptive immune activation. We expect to file IND applications for IMM47 for the treatment of solid tumors with the NMPA and the FDA in 2023, and initiate a Phase I

dose-escalation study first in Australia in mid-2023. Initiating a clinical trial in Australia first can help us to begin global clinical trials earlier and accelerate clinical validation of IMM47. Additionally, we believe Australian trial can generate valuable clinical data on ethnically diverse populations, thus enhancing our ability to pursue collaboration opportunities with global pharmaceutical companies.

- **IMM4701** is a bispecific molecule that simultaneously targets CD47 and CD24. It is also developed on our mAb-Trap platform and shares a similar structure as our other CD47-based bispecific molecules. We have observed robust antitumor activity of IMM4701 in various solid tumor models, in which IMM4701 achieved 122% tumor growth inhibition (TGI) at 3.0 mg/kg (~0.3 mg/kg human equivalent dose). Further leveraging the data observed from IMM47, we plan to file IND applications with the NMPA and the FDA subsequently, and further seek collaboration opportunities with global pharmaceutical companies.

We have also been actively evaluating the therapeutic potential of other promising innate immune checkpoints, including IL-8, NKG2A and PSGL-1, and we aim to continue to stay at the forefront of the development of next-generation immunotherapies through scientific innovation.

Our adaptive immunity-based drug candidates include:

- **IMM2510** is a bispecific molecule with a mAb-Trap structure targeting VEGF and PD-L1. IMM2510 can inhibit angiogenesis, leading to tumor shrinkage, and sensitize tumor cells to immune responses, while activating T cells, NK cells, and macrophages via the blockade of PD-L1/PD-1 interaction and the induction of Fc-mediated ADCC/ADCP activity. Our preclinical efficacy studies showed that IMM2510 exerted stronger synergistic antitumor activities than the combination of a VEGF blocker and a PD-L1 antibody. We are currently conducting the Phase I dose-escalation trial for IMM2510 in China in a variety of advanced solid tumors, including, but not limited to, HCC, RCC, GC, NSCLC and soft-tissue sarcomas (STS). Initial clinical results as of February 15, 2023 have shown favorable safety and promising efficacy. IMM2510 was safe and tolerable up to 10.0 mg/kg in patients with advanced solid tumors, and we are currently evaluating patients for 10.0 mg/kg dose cohort. Among the two evaluable NSCLC patients in the trial so far, we have observed PRs in both patients with best tumor shrinkage response of 46% and 35% respectively. We expect to complete this dose-escalation study in mid-2023, and subsequently commence a cohort-expansion study.

- **IMM27M** is a new generation CTLA-4 antibody with enhanced ADCC activity. It can induce potent immune responses targeting CTLA-4 overexpressed immune-suppressive T_{reg} cells and promote T_{reg} depletion from the TME, thus enhancing T-cell antitumor response. Our preclinical studies have demonstrated that IMM27M could induce significantly stronger antitumor activity than YERVOY® (ipilimumab) and it resulted in complete tumor remission even at a dose as low as 0.3 mg/kg at which ipilimumab only exhibited approximately 50% TGI. We have commenced the Phase I clinical trial in solid tumors, with the first patient dosed in June 2022. We had enrolled 15 patients as of February 10, 2023, and we are currently enrolling patients for the sixth cohort of 5.0 mg/kg. The preliminary data demonstrates that IMM27M is safe and well tolerated up to 3.0 mg/kg. We have observed 4 SDs in this trial so far, among whom one patient with breast carcinoma who had six lines of prior treatment has achieved SD with tumor shrinkage of 28.8% at 3.0 mg/kg, and one patient with metastatic melanoma has achieved SD with tumor shrinkage of 22.9% at 2.0 mg/kg. We expect to complete this trial in mid-2023. We received an IND approval from the NMPA for a Phase Ib/II study to evaluate the combination of IMM27M and a PD-1 antibody for the treatment of

advanced solid tumors, such as RCC, NSCLC, GC and thymic carcinoma (TC), in March 2023. We may initiate clinical trials or explore collaboration opportunities for this combination therapy.

- **IMM40H** is a humanized IgG1 CD70 monoclonal antibody with enhanced ADCC activity. It can obstruct the activation and proliferation of T_{reg} cells through the inhibition of CD70/CD27 signaling. Our *in vitro* cell-based assay demonstrated that IMM40H had much stronger CD70-binding affinity than cusatuzumab (a CD70-targeted antibody developed by Argenx and currently in Phase II stage), allowing it to block the interaction of CD70 and CD27 more effectively. Moreover, IMM40H has also shown potent ADCC, complement-dependent cytotoxicity (CDC) and ADCP activity, resulting in strong immune attack on tumor cells and potentially potent therapeutic efficacy. Our preclinical data also suggests a favorable safety profile of IMM40H. According to Frost & Sullivan, CD70 could potentially be an effective therapeutic target for the treatment of CD70-positive tumors, including CD70-positive lymphoma, RCC, NSCLC, HNSCC and OC. We have obtained IND approvals for IMM40H from the NMPA and the FDA in August 2022, and may initiate Phase I clinical studies or pursue potential collaboration opportunities.

As of the Latest Practicable Date, we owned four issued patents and five allowed patent applications in the PRC, six issued patents and two allowed patent applications in the U.S., nine issued patents and two allowed patent applications in other jurisdictions, 18 pending patent applications in different jurisdictions, one PRC patent application filed as a priority application, six PCT patent applications which have entered national phases and four pending PCT patent applications which may enter various contracting states in the future.

We will continue to advance the development of our drug candidates and enrich our pipeline. To fully unleash the clinical and commercial potential of our comprehensive portfolio, we may develop and commercialize multiple other discovery- and preclinical-stage drug candidates by ourselves or through business collaboration, such as out-licensing and co-development arrangements. Led by our visionary scientist founder, Dr. Tian, we have assembled a seasoned management team with a global perspective coupled with industry expertise and a proven track record of translating biological discoveries into efficacious therapies. With the deep scientific knowledge and extensive experience of our management team, we will continue to expand our footprint in major markets worldwide and maximize the clinical and commercial value of our drug candidates.

OUR STRENGTHS

Science-driven biotechnology company with a rich next-generation immuno-oncology pipeline harnessing both the innate and adaptive immune systems

We are one of the few biotechnology companies globally adopting a systematic therapeutic approach to harness both the innate and adaptive immune systems, unleashing their synergistic potential to address the limitations of T-cell-based immunotherapies. Currently approved immuno-oncology therapies promote immune responses primarily through approaches that target T-cell immune checkpoints, exemplified by PD-1/PD-L1, CTLA-4 and LAG3. Although those immunotherapies targeting adaptive immune checkpoints have illuminated the incredible power of the immune system in combatting a wide range of cancers and dramatically changed the landscape of oncology treatment, only about 10% to 25% of cancer patients could benefit from PD-1/PD-L1 inhibitor monotherapy across almost all major cancer types, according to Frost & Sullivan. The response rates to immunotherapies targeting adaptive immune checkpoints are particularly low in "cold tumors," or in non-T cell-inflamed immune-suppressive tumor microenvironment (TME). These limitations have driven a continued search for new immunotherapeutic approaches to improve treatment outcomes. In recent years, emerging research breakthroughs have revealed the power of innate immunity and fueled a wave of next-generation immuno-oncology drugs.

To unlock the strong power of innate immunity and the synergistic potential between the two arms of immune systems, we have long been researching innate immunity to overcome the limitations of currently approved immunotherapies since our inception in 2015. To date, we have built a pipeline of over ten drug candidates that address promising key innate and adaptive immune targets. We believe our pipeline assets have significant clinical potential to treat a broad spectrum of cancer indications. Our selected drug candidates and the immune system and checkpoints they target are illustrated in the diagram below:

Note: Currently we have several other discovery- and preclinical-stage drug candidates and plan to further develop these candidates by ourselves or through collaboration.
Source: Company Data

Compared to adaptive immunity, innate immunity provides immediate non-specific immune responses to a broad array of foreign substances. Major types of innate immune cells, including macrophages, NK cells and dendritic cells (DC), are widely distributed in cancerous tissues. These innate immune cells can induce an instant immune reaction against tumor cells, and can elicit more wholistic and long-lasting immune responses against cancer, working together with adaptive immune system. The following table sets forth an overview and comparison of the key adaptive and innate immune cells in the TME:

	Adaptive Immunity		Innate Immunity		
Activation Process	Antigen priming required		First line of defense, short response time, no need for antigen priming		
Key Immune Cell Type	T cell	B cell	Macrophage	NK cell	DC
Tumor Tissue Distribution[(1)]	10-30%	3%-40%	20-50%	5%-10%	3%-10%
Major Immune Functions	• T-cell mediated killing of tumor cell via exocytosis of cytotoxic granules (perforin, granzymes) and secretion of antitumor cytokines	• Antibody production • Cytokine secretion	• Macrophage-mediated phagocytosis • Attracting T cells to the tumor microenvironment (TME) • Antigen presentation • Trogocytosis	• NK cell-mediated cytolysis via the secretion of perforin and granzymes • Activating of T cells, macrophages and DCs through release of cytokines	• Attracting T cells to the TME • Antigen presentation

Note: The tumor tissue distribution is the proportion of certain immune cells in different tumor tissues.
Source: Frost & Sullivan

By unleashing the power of innate immunity, the combination of immunotherapies targeting innate immune checkpoints and those targeting adaptive immune checkpoints may revolutionize the treatment for many cancer patients. According to Frost & Sullivan, driven in part by the growth of the innate immunotherapy market, the global market size of immuno-oncology therapy is expected to reach US$311.2 billion in 2035, representing over 50% of the then global oncology drug market. Currently, there are no innate immune checkpoint-targeted therapies approved for marketing worldwide, indicating a large untapped market.

To fill the vacuum of immunotherapies targeting innate immune checkpoints and further address critical unmet medical needs, we are committed to the discovery and development of next-generation immunotherapies that harness both powerful arms of the immune system. Underlying our drug discovery and development efforts and guided by our deep understanding of cancer biology and immunology, we uphold an overarching R&D concept that we call "DbD." The "DbD" concept requires us to carefully select and validate promising targets and to deliberately design the structure of every drug molecule based on a sound scientific rationale and preclinical validation. With our scientific expertise and insights into the landscape of current cancer treatments, we constructed a rich product portfolio with highly differentiated molecule design and global first-in-class or best-in-class potential.

Among innate immune checkpoints, CD47 has now been widely recognized and clinically validated as one of the most promising immunotherapeutic targets for the treatment of a wide range of cancers. Our founder, Dr. Wenzhi Tian, started to explore the therapeutic potential of CD47-targeted strategy in oncology as early as 2010, long before it became widely recognized in the biopharmaceutical industry. Since then, Dr. Tian has continued to closely monitor the scientific progress related to this target and has been further convinced of its potential to be a next-generation cancer immunotherapeutic target. Leveraging the fundamental insights into CD47, we started our development efforts on IMM01 since our inception in 2015, which later became the first CD47-targeted SIRPα-Fc fusion protein to enter into clinical stage in China. According to Frost & Sullivan, among numerous drug developers of CD47-targeted molecules globally, we are one of the only two companies to have observed CR in monotherapy clinical trials with a well tolerated safety profile.

Building on the engineered and global IP-protected CD47-binding domain of IMM01 and in adherence to our "DbD" concept, we further designed and developed multiple CD47-based bispecific molecules with ADCC-enhanced IgG1 Fc leveraging our proprietary mAb-Trap platform. Two of our clinical-stage CD47-based bispecific molecules, IMM0306 (CD47×CD20) and IMM2902 (CD47×HER2), are the first bispecific molecules with their respective targets globally to enter clinical trials. The other clinical-stage CD47-based bispecific molecule, IMM2520 (CD47×PD-L1), is also a highly differentiated molecule that demonstrates very promising efficacy targeting solid tumors.

In addition to CD47, we have selected and validated another promising innate immune checkpoint — CD24, through our preclinical proof-of-concept studies. This checkpoint is widely expressed on different types of cancer cells and has shown to be highly correlated to poor prognosis, presenting a robust potential for broad clinical applications. According to Frost & Sullivan, there is currently no approved or clinical-stage drug candidate targeting CD24 globally. The screening of monoclonal antibodies against CD24 is highly challenging due to the relatively weak immunogenicity resulting from its small extracellular domain. Leveraging our deep understanding in immuno-oncology, we started the discovery research on CD24 as early as 2019 and have successfully identified lead drug candidates, IMM47 and IMM4701, with potent target activity and *in vivo* therapeutic efficacy. We are also developing several other discovery- and preclinical-stage bispecific molecules targeting CD24. All of our CD24-based candidates have the global first-in-class potential in the fields of oncology and potentially other diseases. Furthermore, given its ability to stimulate innate immune responses, CD24-targeted therapy is also expected to exhibit great synergistic effects when combined with T-cell-based immune therapies.

With a comprehensive portfolio of drug candidates targeting promising innate immune checkpoints, we believe that we are well-positioned at the forefront of the global immuno-oncology drug market to address the limitations faced by T-cell-based cancer therapeutics and capture immense market opportunities.

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

We have developed in-house one of the deepest and broadest portfolios of innate immune-targeted programs globally. Our well-constructed portfolio is built based on key innate immune targets and pathways critical to a variety of hematologic and solid tumors to address significant unmet medical needs. These portfolio candidates would not only enhance direct tumor-killing activity of innate immune cells, but can also elicit holistic immune responses across the innate and adaptive immune systems that ultimately lead to long-lasting and robust antitumor effects.

Our portfolio features a deep CD47-based pipeline comprising IMM01 (SIRPα-Fc fusion protein), multiple clinical-stage bispecific drug candidates, including IMM0306 (CD47×CD20), IMM2902 (CD47×HER2), IMM2520 (CD47×PD-L1), and numerous other discovery- and preclinical-stage CD47-based bispecific molecules. All of those CD47-based candidates are designed with IgG1 Fc. Furthermore, around CD24, we have developed one IND-enabling-stage drug candidate, IMM47 (CD24 mAb), and several discovery- and preclinical-stage bispecific molecules, including IMM4701 (CD47×CD24) and IMM2547 (CD24×PD-L1). All of those CD24-based candidates have the global first-in-class potential to address enormous market opportunities. In addition, we have expanded our portfolio to target other emerging critical innate immune checkpoints with large clinical and commercial potential, including IL-8, NKG2A and PSGL-1. Guided by our "DbD" concept, all programs are designed with a unique structure that best suit the considerations and requirements for each respective target or target pairings.

CD47

CD47 is a critical macrophage checkpoint that plays a broad role in cancer immune evasion across many cancer types. Research reveals that high expression of CD47 is shown to correlate with aggressive disease and poor prognosis. Tumor cells often express high levels of CD47, which transmit a "don't eat me" signal by binding to SIRPα on the surface of macrophages to evade macrophage destruction. As mounting clinical evidence suggests, targeting the CD47/SIRPα pathway is an effective immune-therapeutic strategy. The CD47/SIRPα pathway has been clinically validated and became one of the most attractive next-generation cancer immunotherapeutic targets, which is believed to potentially rival PD-1/PD-L1 in terms of clinical significance and market size.

According to Frost & Sullivan, the global market size of CD47/SIRPα-targeted therapies is expected to reach US$13.1 billion and US$33.7 billion in 2030 and 2035, respectively. The prospect promised by this new therapy has also been validated by publicly reported clinical data and several multibillion-dollar takeover and licensing transactions backed by leading multinational pharmaceutical companies, including Gilead, Pfizer and AbbVie.

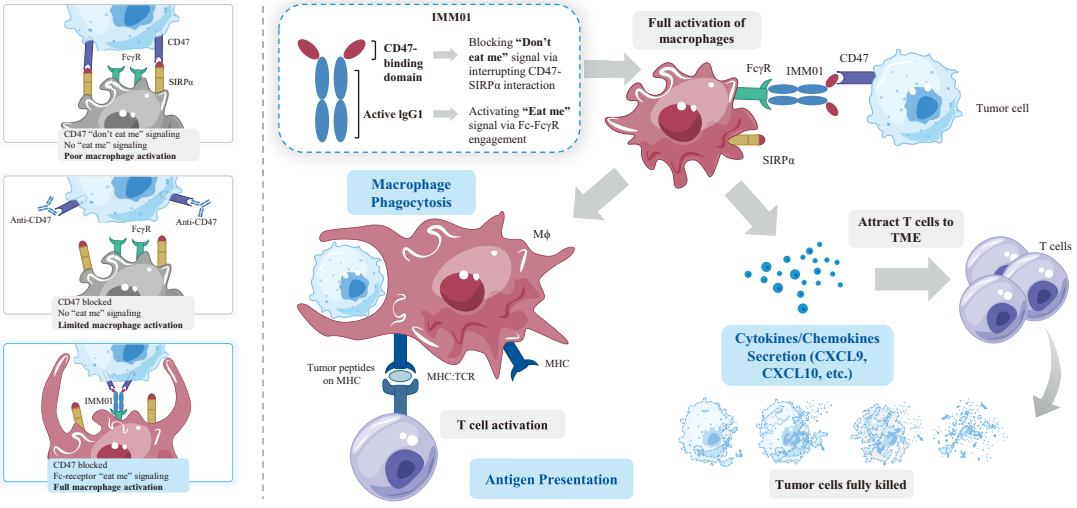
Despite the promising prospect, the successful development of CD47-targeted drugs still needs to overcome various challenges. On the one hand, binding of CD47-targeted agents with blood cells that ubiquitously express CD47, particularly RBCs, generate issues including severe blood toxicity, rapid reduction in drug exposure (or "antigenic sink") and decreased potency. On the other hand, in addition to the blockade of the "don't eat me" signal, full activation of macrophages requires an additional "eat me" signal induced by an IgG1 Fc via engagement with activating FcγR. However, due to the inevitable binding (weak or strong) of CD47 antibodies to RBCs, most of those antibodies resorted to an IgG4 Fc region with weak FcγR engagement to avoid RBC phagocytosis by macrophages, sacrificing their therapeutic efficacy for safety. Even

with IgG4 Fc, the hemagglutination (the clumping of RBCs) as a result of RBC binding of CD47 antibodies still presents substantial safety issues as seen in certain clinical trials. In addition to the clinical suspension seen with CD47 antibodies of Bristol-Myers (Celgene) and Surface Oncology, a recent example is that the FDA placed a partial clinical suspension on studies to evaluate Gilead's magrolimab (a CD47 mAb) in MDS, AML, MM and diffuse large B-cell lymphoma (DLBCL) due to an apparent imbalance in investigator-reported suspected unexpected serious adverse reaction (SUSAR) between study arms observed in trials in early 2022. All of those partial suspensions have been subsequently lifted, as the FDA determined that, following comprehensive review of the safety data from each trial, the clinical sponsor had satisfactorily addressed the deficiencies.

To tackle the safety concerns around CD47, we used an engineered CD47-binding domain of human SIRPα in IMM01, which does not bind to human RBCs in *in vitro* studies. Furthermore, our modification of deglycosylation of the binding domain mitigates the immunogenicity of the molecule. The resulting properties enable IMM01 to reduce blood toxicity and avoid antigenic sink with improved pharmacokinetic (PK) profile. The favorable safety and tolerability of IMM01 allow us to use a more potent IgG1 Fc fragment to fully activate macrophages to achieve exceptional single-agent activity even at one-fifteenth (1/15) the dose level (up to 2.0 mg/kg) of that of CD47 antibodies (typically in the range of 30 to 45 mg/kg). Additionally, IMM01 is also proven not to trigger the T-cell apoptosis that could be induced by certain CD47 antibodies.

The unique structure of IMM01, comprising an engineered CD47-binding domain and an IgG1 Fc fragment, enables it to exert a dual mechanism imperative for full macrophage activation: blocking the "don't eat me" signal while triggering a strong "eat me" signal. The dual mechanism acts to fully activate both macrophages and NK cells. Activated macrophages will not only mediate direct antitumor phagocytosis, but can also release chemokines and cytokines to recruit T cells into the TME, turning "cold tumors" into "hot tumors," further activating T-cell response through antigen presentation. Activated NK cells can on the one hand mediate ADCC against tumor cells and, on the other hand, further promote T-cell differentiation and T-cell response. The following diagram illustrates the mechanism of action of IMM01:

Mechanism of Action of IMM01



Definition: MHC refers to major histocompatibility complex.
Source: Frost & Sullivan, Literature Review, Company Data

Thus far, IMM01 has demonstrated encouraging monotherapy efficacy and good tolerability in our completed Phase I trial targeting lymphoma. With encouraging efficacy and favorable safety in monotherapy clinical trials and robust preclinical data of its combination studies, IMM01 is expected to achieve strong synergistic effects used in combination with other cancer agents.

IMM01 showed strong synergistic antitumor activity when used in combination with T-cell immunotherapies, such as PD-1 and PD-L1 inhibitors, and other cancer agents, such as azacitidine and targeted therapies. When used in combination with IgG4 antibodies, such as most PD-1 inhibitors, IMM01 with the potent IgG1 fragment is able to activate the additionally required "eat me" signal to achieve full macrophage activation and enhanced immune responses. We intend to develop the combination therapy of IMM01 and tislelizumab for the treatment of solid tumors that are refractory or relapsed from the standard of care such as PD-1/PD-L1 inhibitors, including among others, NSCLC, SCLC, HNSCC and CRC. We are currently evaluating IMM01 and tislelizumab in a Phase II trial in various advanced solid tumors that failed to respond to or relapsed from the standard of care such as PD-1/PD-L1 inhibitors. In our Phase Ib trial, a heavily pre-treated NSCLC patient with six lines of prior treatment and refractory to PD-1 inhibitors achieved PR after three cycles of treatment with target lesion shrinkage of 40%. In addition, we are also evaluating this combination therapy in cHL patients who relapsed or progressed after the treatment of PD-1 inhibitors in this Phase Ib/II trial, which may allow us to pursue an accelerated marketing approval leveraging the results of relatively small sample size studies. We dosed the first patient with R/R cHL in January 2023.

In addition, since IMM01 does not bind to RBCs *in vitro* and only a fraction of the dose is required due to its dual mechanisms, it demonstrates encouraging efficacy and is expected to achieve more favorable tolerability than CD47 antibodies, and therefore it could be broadly used in combination therapies for the treatment of a wide range of cancer indications. We have completed the Phase Ib trial and initiated the Phase II trial for the IMM01 combined with azacitidine mainly for the first-line treatment of HR MDS, unfit AML and CMML in China. Interim data from the Phase Ib/II trial has demonstrated a favorable safety profile and promising efficacy profile. Neither DLT nor hemagglutination was observed among all 12 patients receiving the combination treatment at all three dose levels of IMM01 (1.0 mg/kg, 1.5 mg/kg, and 2.0 mg/kg) in our Phase Ib trial. Moreover, the interim data obtained from our Phase II trial as of February 10, 2023 has demonstrated that: (i) among the eight evaluable patients with 1L CMML, two reached CR (2 CRs), six reached mCR (6 mCRs), and one reached HI (1 HI, which also achieved mCR), resulting in an ORR of 100%, and (ii) among the 16 evaluable HR MDS patients who have received at least three cycles of treatment, three achieved CR (3 CRs), nine achieved mCR (9 mCRs), and seven achieved HI (7 HIs, among which 4 also achieved mCR), resulting in an ORR of 93.8%. We plan to seek an accelerated marketing approval through relatively small sample size studies targeting the first-line treatment of CMML, a rare type of disease with highly unmet medical needs.

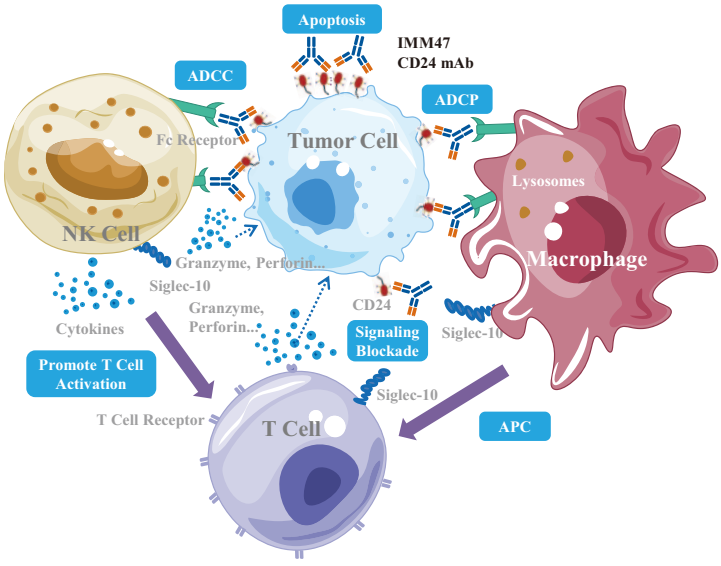
Leveraging our experience in developing IMM01, we designed and are developing numerous CD47-based bispecific molecules that incorporate the same engineered CD47-binding domain as IMM01 and an ADCC-enhanced IgG1 Fc. The engineered CD47-binding domain enables our CD47-based bispecific molecules to avoid RBC binding, thus allowing the adoption of IgG1 Fc capable of inducing full macrophage activation, much enhanced ADCP and ADCC activity, and stronger antitumor immune responses compared to most IgG4-based CD47 bispecific antibodies. Notably, compared to combination therapies of the same targets, those bispecific molecules displayed better *in vivo* efficacy, showing promise to offer improved clinical benefits and affordability.

CD24

CD24 is a promising innate immune checkpoint widely expressed on numerous types of solid tumor cells, including BC, NSCLC, CRC, HCC, RCC and OC, and strongly correlated with poor prognosis. Blocking CD24/Siglec-10 pathway will exert multifaceted activation effects on immune responses against cancer, presenting large clinical and market potential. There is currently no approved or clinical-stage drug candidate targeting CD24 globally, according to Frost & Sullivan.

IMM47 is a humanized monoclonal antibody targeting CD24, which can disrupt inhibitory CD24/Siglec-10 signaling to macrophages, NK cells and T cells. With its ADCC-enhanced IgG1 Fc fragment, IMM47 can potently activate macrophage- and NK cell-mediated immune responses through ADCP and ADCC. IMM47 has also been shown to significantly increase the amount of M1 macrophages in tumor tissues in our *in vivo* proof-of-concept studies. It can also activate and promote T-cell response likely through tumor antigen presentation by activated macrophages to T cells and direct blockade of CD24/Siglec-10 inhibitory signals. We have observed strong antitumor activity of this molecule in our preclinical solid tumor models. The following diagram illustrates the mechanism of action of IMM47:

Mechanism of Action of IMM47



Source: Frost & Sullivan, Literature Review, Company Data

Against CD24, we have subsequently developed several bispecific molecules, among which IMM4701 (CD47×CD24) is at the CMC stage and has demonstrated potent *in vivo* efficacy in a triple-negative breast cancer (TNBC) animal model.

As CD24-targeted therapy is able to activate key innate immune cells, converting non-T cell-inflamed immune-suppressive TME and further promoting T-cell response through the crosstalk between innate and adaptive immune systems, it can also create a strong synergistic potential with other immunotherapies, such as PD-1/PD-L1 inhibitors. In fact, our preclinical studies have shown that the combination of IMM47 and OPDIVO® or KEYTRUDA® can lead to a significant increase in response rates in our mouse model compared to using OPDIVO® or KEYTRUDA® alone. Furthermore, when we reinoculate the same cancer cells into mice pre-treated with IMM47 and PD-1 antibodies, tumor growth could be rapidly and completely eliminated, indicating the establishment of a tumor-specific immune response.

Other innate immune checkpoints

We have also been actively exploring the therapeutic potential of other promising innate immune checkpoints, and we aim to continue to stay at the forefront of the development of next-generation immunotherapies through scientific innovation. For example, to further modulate the TME, we are developing IMM51, a monoclonal antibody that targets IL-8, for the treatment of solid tumors. IL-8 is a chemokine that alters the immune microenvironment by recruiting myeloid derived suppressor cells (MDSC) to the TME, resulting in therapeutic resistance to immune checkpoint inhibitor (ICI) therapy. Thus, blocking IL-8 with IMM51 could potentially sensitize cancer cells to existing immunotherapies. In addition, we are conducting investigations on molecules that target NKG2A and PSGL-1. NKG2A is an inhibitory cell surface molecule mainly expressed on both NK cells and CD8+ T cells. Interaction of NKG2A with HLA-E expressed on tumor cells inhibits the activation of NK cells and T cells, thus NKG2A blocking antibody may activate the cytotoxic activity of effector CD8+ T cells and NK cells. Recent research has revealed that targeting PSGL-1, an adhesion molecule expressed by macrophage and many other hematopoietic cells, can repolarize M2 macrophages to M1-like state, inducing pro-inflammatory cytokine and chemokine production known to be associated with beneficial clinical responses. We will continue to evaluate other promising innate immune checkpoints and enrich our pipeline with novel therapies.

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

Targeting our strategically selected innate and adaptive immune checkpoints, we are committed to designing differentiated molecules that can achieve an optimized safety and efficacy profile. Our strong capabilities in molecule design are underpinned by an experienced scientific team with deep expertise in tumor biology and immunology. Guided by our "DbD" concept, we have designed and developed six clinical-stage, one IND-stage, one IND-enabling-stage and numerous discovery- and preclinical-stage drug candidates, each with differentiated structure and protected by global IP rights. We believe our R&D capabilities and established R&D platforms will enable us to rapidly advance our pipeline candidates towards commercialization, and to continuously discover new generations of immuno-oncology therapies to address critical unmet medical needs.

CD47-based drug candidates

Our portfolio of CD47-based drug candidates includes a SIRPα-Fc fusion protein and multiple bispecific molecules as follows:

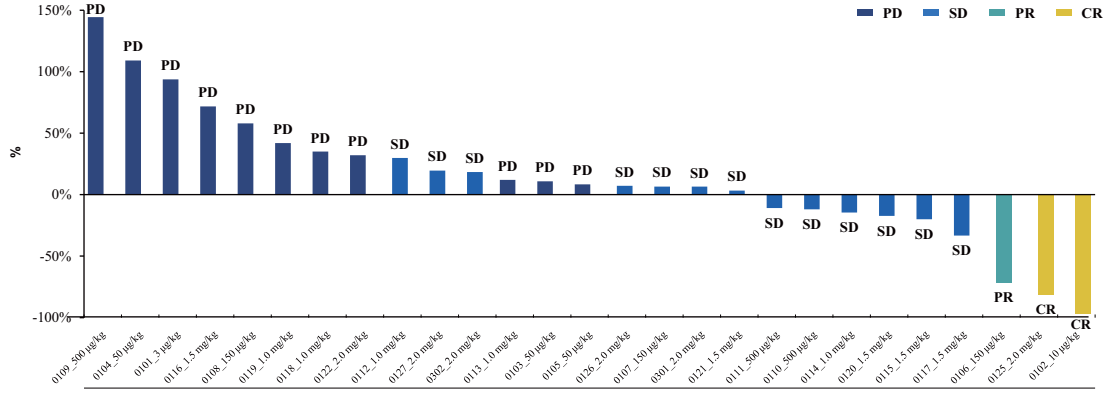
IMM01 (SIRPα-Fc fusion protein)

IMM01 is a next-generation CD47-targeted molecule. It is the first SIRPα-Fc fusion protein to enter into clinical stage in China. IMM01 is being developed for the treatment of various hematologic malignancies and solid tumors in combination with other agents. According to Frost & Sullivan, among numerous drug developers of CD47-targeted molecules globally, we are one of the only two companies to have observed CR in monotherapy clinical trials with a well tolerated safety profile.

IMM01 monotherapy has exhibited good safety and preliminary efficacy in early clinical trials. We have completed the Phase I dose-escalation study of IMM01 in R/R lymphoma patients. In the Phase I clinical trial, IMM01 was well tolerated and safe up to 2.0 mg/kg in patients. The majority of treatment-related adverse events (TRAEs) were Grade 1 and 2. Grade 3 or above TRAEs of IMM01 mainly included leukopenia, thrombocytopenia, anemia and neutropenia, with the highest rate of occurrence at 14% (four out of 29). As of December 14, 2022, clinical results from the Phase I study showed IMM01 monotherapy treatment led to two CRs (2 CRs), one PR (1

PR) and 13 SDs (including six cases with substantial tumor shrinkage observed) among 27 evaluable patients in the Phase I study, while treatments are still ongoing for the patients with PR and CR. In this Phase I monotherapy clinical trial, among the six patients at RP2D dose of 2.0 mg/kg, one reached CR (1 CR), and four reached SDs (4 SDs), with a DCR of 83% in these previously heavily pre-treated R/R lymphoma patients. The diagram below illustrates the best overall changes in size of target tumor lesions.

Best Overall Changes in Size of Target Tumor Lesions



Note: The colors of bars represent the best overall changes in size of target tumor lesions.
Source: Company Data, as of December 14, 2022

We are actively evaluating IMM01 in combination with other drugs in the following clinical trials:

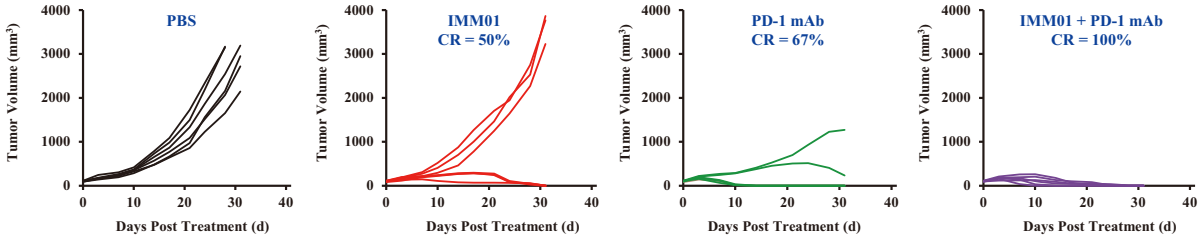
- **Combination of IMM01 and azacitidine:** The therapeutic benefits of CD47-targeted therapies in combination with azacitidine have been validated in multiple clinical trials according to publicly reported data. Our *in vivo* efficacy studies also showed that the combination of IMM01 and azacitidine exhibited favorable safety profiles and strong synergistic antitumor activity. With the dual mechanisms of action, IMM01 has demonstrated a promising efficacy signal at a dose level of 2.0 mg/kg which is much lower than the typical dose required for CD47 antibodies (30.0 to 45.0 mg/kg) when used in combination with azacitidine. IMM01 in combination with azacitidine has exhibited a favorable safety profile due to such lower doses required for treatment and its minimal impact on RBCs. We started to dose patients in a Phase Ib/II trial to evaluate this combination mainly for the first-line treatment of HR MDS, unfit AML and CMML in January 2022. Neither DLT nor hemagglutination was observed among all 12 patients at all three dose levels of 1.0 mg/kg, 1.5 mg/kg, and 2.0 mg/kg in our Phase Ib trial. Moreover, the interim data obtained from our Phase II trial as of February 10, 2023 has demonstrated that: (i) among the eight evaluable patients with 1L CMML, two reached CR (2 CRs), six reached mCR (6 mCRs), and one reached HI (1 HI, which also achieved mCR), resulting in an ORR of 100%, and (ii) among the 16 evaluable HR MDS patients who have received at least three cycles of treatment, three achieved CR (3 CRs), nine achieved mCR (9 mCRs), and seven achieved HI (7 HIs, among which 4 also achieved mCR), resulting in an ORR of 93.8%.

- **Combination of IMM01 and tislelizumab:** The combination of PD-1/PD-L1 inhibitors and CD47-targeted therapies is expected to benefit a large population of patients with solid tumors that have limited response to PD-1/PD-L1 inhibitors. When combined with PD-1 inhibitors that typically consist of IgG4 Fc, IMM01, designed with distinctive IgG1 Fc, is able to deliver the additional "eat me" signal which is for full macrophage

activation, in contrast to IgG4 Fc CD47 antibodies. Activation of macrophages can subsequently remodel the non-T cell-inflamed immune-suppressive TME and convert "cold tumors" into "hot tumors," which significantly enhance the efficacy of PD-1/PD-L1 inhibitors. Additionally, IMM01 significantly inhibits the production of IL-8, which acts as one of the key mediators of resistance to PD-1/PD-L1 inhibitors.

This is supported by the results of our *in vivo* efficacy studies assessing IMM01 in combination with a PD-1/PD-L1 antibody. In a xenograft model in mice, the combination of IMM01 and a PD-1 antibody (tislelizumab) resulted in significantly stronger antitumor effects than the PD-1 antibody used alone in our solid tumor model (see the figure below).

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



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

Based on the positive preclinical data, we are currently evaluating IMM01 and tislelizumab in a Phase II trial in various advanced solid tumors that failed to respond to or relapsed from the standard of care such as PD-1/PD-L1 inhibitors, including among others, NSCLC, SCLC, and HNSCC. We dosed the first patient for the Phase Ib trial in May 2022 and initiated the Phase II trial in December 2022. In our Phase Ib trial, a heavily pre-treated NSCLC patient with six lines of prior treatment and refractory to PD-1 inhibitors achieved PR after three cycles of treatment with target lesion shrinkage of 40%. We expect to initiate a pivotal trial in the third quarter of 2024. After accumulating more clinical data, we may also further evaluate this combination therapy for the first-line treatment of those solid tumors as well as for the treatment of other cancer indications. We are also evaluating this combination therapy in cHL patients who relapsed or progressed after the treatment of PD-1 inhibitors, which may allow us to pursue an accelerated marketing approval leveraging the results of relatively small sample size studies. We dosed the first patient with R/R cHL in China in January 2023.

- **Combination of IMM01 and other drugs:** IMM01 has demonstrated a promising efficacy and safety profile in its Phase I monotherapy trial, which sets the stage for its combination use with other immunotherapies or targeted therapies in our various *in vivo* studies. We are currently exploring the therapeutic potential of IMM01 in combination with other drugs for treating a wide range of cancer indications. We reached a collaboration with Sunshine Guojian, under which Sunshine Guojian will be primarily responsible for driving and funding the clinical development of the combination treatment of IMM01 and inetetamab for HER2-positive solid tumors in mainland China. We are also conducting numerous preclinical studies to evaluate the combination use of IMM01 with other drugs. These combination studies have shown robust synergistic potential in our mouse models.

In addition to the above, targeting unmet medical needs globally, we will strategically explore other promising combination therapies of IMM01 with targeted drugs or immune checkpoint inhibitors to fully unleash the potential of IMM01, based on solid scientific rationale and results generated from preclinical studies.

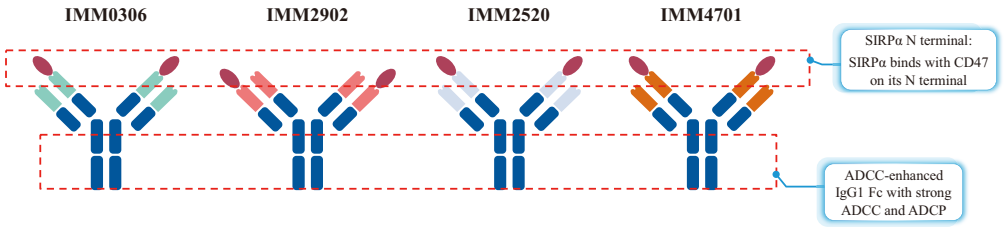
CD47-based bispecific molecules

Based on the validated molecule structure of IMM01, we have subsequently developed multiple CD47-based bispecific molecules leveraging our mAb-Trap platform. These bispecific molecules all have symmetrical structure with the same engineered CD47-binding fragment used in IMM01. 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.

This unique structural design with the engineered CD47-binding fragment allows our CD47-based bispecific molecules to avoid RBC binding, thus enabling the adoption of an ADCC-enhanced IgG1 Fc fragment capable of inducing full macrophage activation and much improved ADCP and ADCC activity, resulting in stronger antitumor immune responses compared to most IgG4-based CD47 bispecific antibodies. Also, all of these bispecific molecules have higher binding affinity with the tumor antigen of the base antibody, attracting them to TME and preferentially binding to CD47 on tumor cells, minimizing "on-target, off-tumor" toxicity.

Studies on the crystal structure of CD47 have revealed that the CD47-binding region of SIRPα is located at its N-terminal. When designing the molecules, we thus connect the CD47-binding domain to the N-terminal of the heavy chain or light chain of a base antibody against another tumor target rather than to the Fc end, as is commonly seen in other CD47-based bispecifics. Our design prevents conformational interference with CD47 binding and preserves the intact Fc region with full immune effector function. The below diagram illustrates the unique structure of our bispecific molecules:

Structure of Our CD47-based Bispecific Molecules



Source: Company Data

Compared to combination therapies against the same targets, our bispecific molecules are more likely to bind with two targets co-expressed on the same cancer cell, which is the prerequisite for the dual-targeting strategy to show synergistic effects. As demonstrated in our preclinical studies, our bispecific molecules can exert more potent antitumor activity than the combination therapies with same targets even at a relatively lower dose level. In addition, the symmetric structure of our bispecific molecules developed on our mAb-Trap platform minimizes mismatch during the production process, allowing for ease of manufacturing, product stability, higher titer and protein yield. In fact, average protein yield for IMM0306, IMM2902, and IMM2520 ranges from 3.8 g/L to 4.6 g/L, much higher than the industry average for bispecific molecules of 1.0 g/L to 3.0 g/L.

As of the Latest Practicable Date, three of those bispecific molecules, *i.e.*, IMM0306 (CD47×CD20), IMM2902 (CD47×HER2) and IMM2520 (CD47×PD-L1), are in clinical stage. We are also developing multiple other discovery- and preclinical-stage CD47-based bispecific molecules, including IMM4701 (CD47×CD24).

IMM0306 (CD47×CD20)

IMM0306 is the first bispecific molecule targeting both CD47 and CD20 to enter into clinical stage globally. IMM0306 can simultaneously bind to CD47 and CD20 expressed on malignant B cells, with a higher affinity for CD20 than CD47. This fine-tuned unbalanced affinity design enhances the specificity of tumor-targeting to mitigate "on-target, off-tumor" toxicity by reducing binding to CD47 in normal tissues. Upon simultaneous binding to its targets, IMM0306 can deplete malignant B cells by activating macrophage-mediated ADCP and inducing NK cell-mediated enhanced ADCC. We are currently developing IMM0306 for the treatment of B-cell lymphoma.

Our IMM0306 has demonstrated stronger antitumor activities as compared to the combination therapy of IMM01 and rituximab or each of them as a single agent in preclinical studies. The robust *in vivo* efficacy of IMM0306 observed in this study suggests its potential to replace rituximab for the treatment of B-cell lymphoma in the first-line setting. Furthermore, our preclinical *in vitro* studies have also demonstrated that IMM0306 had a favorable safety profile as it does not bind to human RBCs or cause hemagglutination.

For IMM0306, we have initiated the Phase I trials for the treatment of R/R B-NHL in China in May 2020. Preliminary data collected from the China trial have suggested a favorable safety and promising efficacy profile. According to our initial clinical data as of February 27, 2023, IMM0306 was safe and well tolerated up to 2.0 mg/kg in patients. Among the evaluable patients across four cohorts dosed from 0.8 mg/kg to 2.0 mg/kg, who had relapsed or progressed after receiving rituximab previously, two CRs and five PRs were observed. The only evaluable FL patient at 2.0 mg/kg who relapsed and progressed after rituximab treatment has also been confirmed as PR. At 2.0 mg/kg, one patient with primary bone DLBCL who had four lines of prior treatment has achieved PR with all measurable lesions disappeared after 65 days of treatment. We commenced a Phase IIa trial for IMM0306 monotherapy for the third- or later-line treatment of FL in March 2023 and plan to seek an accelerated marketing approval through a single-arm trial. The encouraging clinical results of IMM0306 also validated our CD47-based bispecific programs developed with our mAb-Trap platform. We have also received an IND approval for IMM0306 from the FDA in January 2021. With further clinical validation in the Phase I trial in China, we will then decide on our clinical development and collaboration strategy for IMM0306 in the U.S.

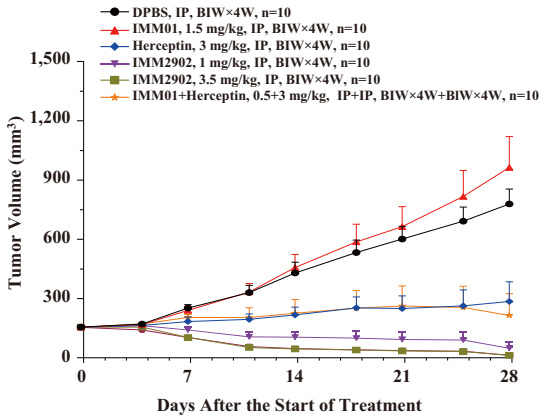
IMM2902 (CD47×HER2)

IMM2902 is the only bispecific molecule targeting both CD47 and HER2 that has entered into clinical trial globally. Similarly, IMM2902 also adopts an ADCC-enhanced IgG1 Fc region to further enhance immune activation. In addition to macrophage activation and enhanced ADCC activity, IMM2902 has been demonstrated to accelerate the degradation of HER2 in preclinical studies and can also potentially induce ADCT similar to RYBREVANT® (amivantamab, an FDA-approved IgG1 Fc EGFR×c-MET). These mechanisms, working together, lead to enhanced tumor killing of IMM2902. We are currently developing IMM2902 for the treatment of HER2-positive and HER2-low expressing solid tumors.

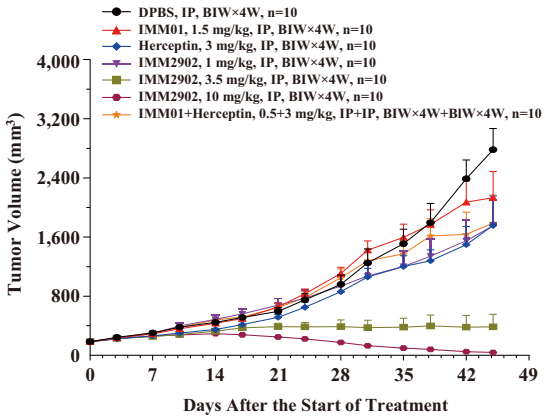
Our preclinical studies revealed strong antitumor activities of IMM2902 in a variety of xenograft models of breast and gastric tumors, including those with HER2-low expression and models resistant to trastuzumab. As shown in the diagrams below, IMM2902 completely eradicated established tumors at 3.5 mg/kg (~0.35 mg/kg human equivalent dose) in a trastuzumab-sensitive

BC and at 10 mg/kg (~1.0 mg/kg human equivalent dose) in a trastuzumab-resistant BC model. Our IMM2902 also exhibited favorable efficacy in trastuzumab-sensitive and HER2-low expressing GC models in our preclinical studies.

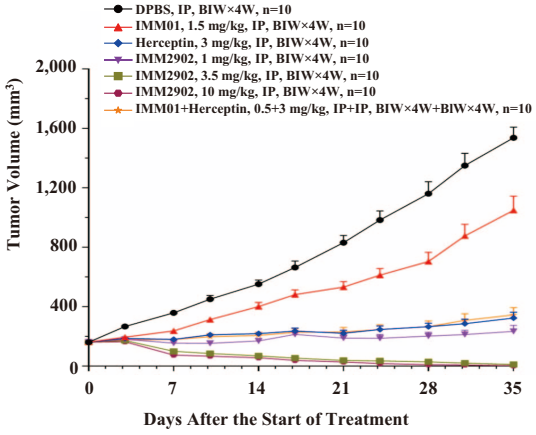
Efficacy Study in Trastuzumab-Sensitive Breast Cancer (BT474) Xenograft Mouse Model



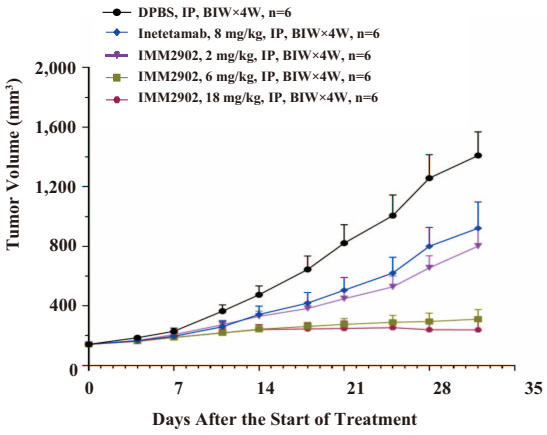
Efficacy Study in Trastuzumab-resistant Breast Cancer (HCC-1954) Xenograft Mouse Model



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



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



Source: Company Data

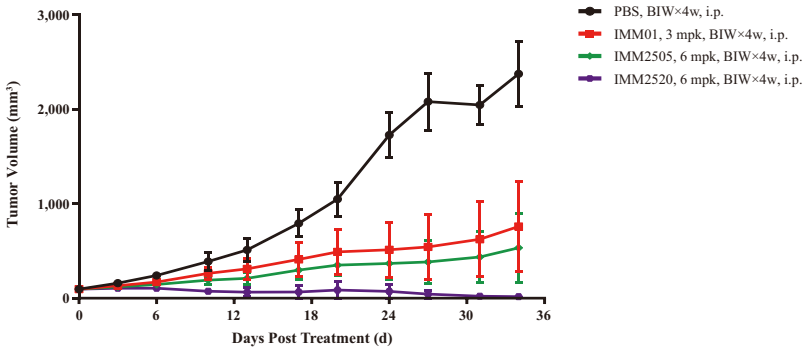
Our preclinical studies have also demonstrated that, compared to magrolimab analog (Hu5F9, a CD47 antibody replicated by us based on public information) that could induce obvious hemagglutination at the concentration beyond 370 ng/ml, IMM2902 does not induce hemagglutination of human red blood cells, even at the concentration as high as 10,000 ng/ml. We have obtained IND approvals for IMM2902 in China and the U.S. for the treatment of advanced HER2-positive and HER2-low expressing solid tumors, including BC, GC, NSCLC and BTC. We dosed the first patient in China in February 2022 and are enrolling the sixth cohort for this dose-escalation study in China. IMM2902 was shown to be safe and well tolerated up to 2.0 mg/kg. Dosing is ongoing for higher dose level cohorts. We have also initiated the clinical trial for advanced HER2-positive and HER2-low expressing solid tumors in the U.S. with the first patient dosed in June 2022. We have received the Fast Track Designation from the FDA in July 2022.

IMM2520 (CD47×PD-L1)

IMM2520 is a bispecific molecule that targets both CD47 and PD-L1 for the treatment of solid tumors. Unlike most other CD47×PD-L1 bispecifics, our engineered CD47-binding fragment allows us to adopt an ADCC-enhanced IgG1 Fc in IMM2520 that is capable of triggering an additionally required "eat me" signal to fully activate macrophages, inducing enhanced ADCP and ADCC activity, mobilizing both innate and adaptive immunities to target tumor cells and improving the clinical response to PD-1/PD-L1 inhibition.

As shown in the below diagram, a syngeneic model in mice demonstrated that IMM2520 led to complete tumor remission at the dose of 6 mg/kg (~0.6 mg/kg human equivalent dose):

Efficacy Study in Colon Cancer (CT26) Syngeneic Mouse Model



Note: IMM2505 is a first-generation CD47 and PD-L1bispecific molecule internally developed by us.
Source: Company Data

In addition, our preclinical toxicity studies of IMM2520 also demonstrated that IMM2520 did not bind to human RBCs. We have obtained IND approvals for IMM2520 from the NMPA in November 2022 and from the FDA in December 2022, and dosed the first patient for the Phase I clinical trial in China in March 2023. We will particularly focus on the solid tumors generally resistant or not sensitive to the currently available immunotherapies, such as CRC, GC, lung cancer and HNSCC, among others.

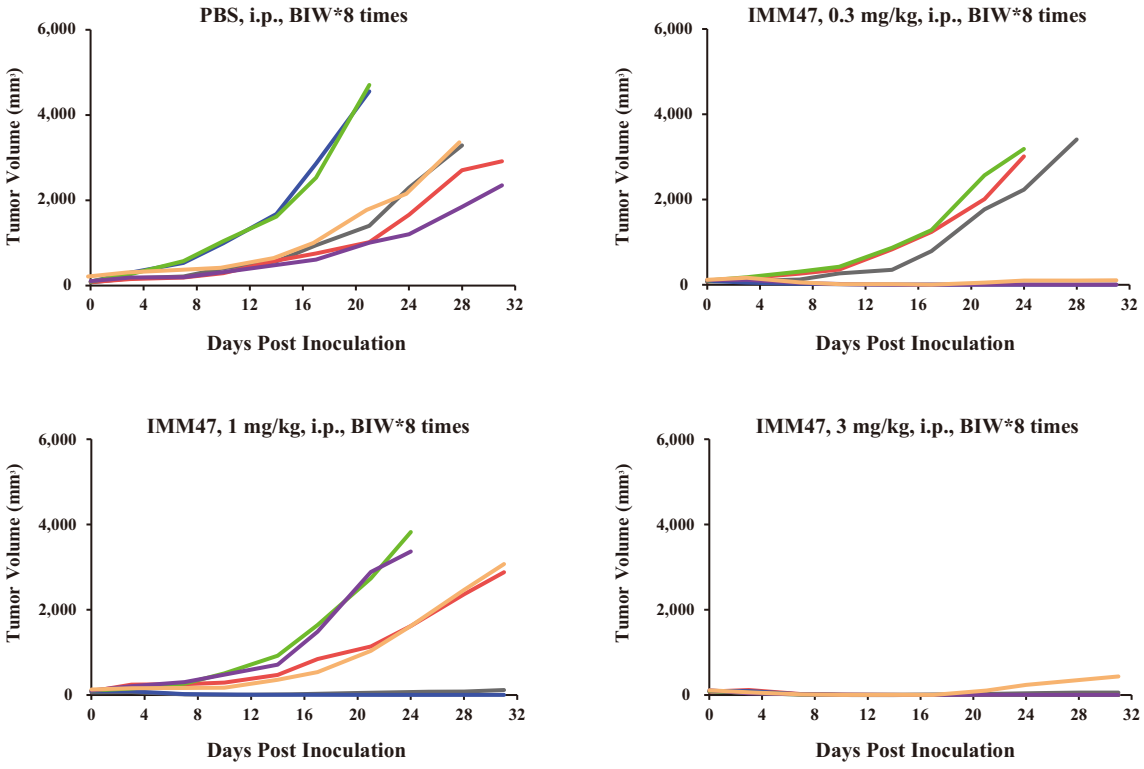
CD24-targeted drug candidates

We started our discovery research on CD24 as early as 2019, and have in-house screened and developed one IND-enabling-stage humanized monoclonal antibody, IMM47 (CD24 mAb), and several discovery- and preclinical-stage bispecific molecules, including IMM4701 (CD24×CD47) and IMM2547 (CD24×PD-L1), all with global first-in-class potential.

IMM47 (CD24 mAb)

IMM47 is a potentially global first-in-class humanized monoclonal antibody targeting CD24 for oncology treatment. According to Frost & Sullivan, there is no approved or clinical stage molecule targeting CD24 worldwide. With its ability to activate integrated innate and adaptive immune responses, IMM47 has shown robust tumor activity in our preclinical studies. In a colon cancer model, IMM47 completely eradicated subcutaneously inoculated tumor cells in all six mice after three doses of 3.0 mg/kg (~0.3 mg/kg human equivalent dose).

Proof-of-Concept Study in Colon Cancer (MC38) Syngeneic Mouse Model



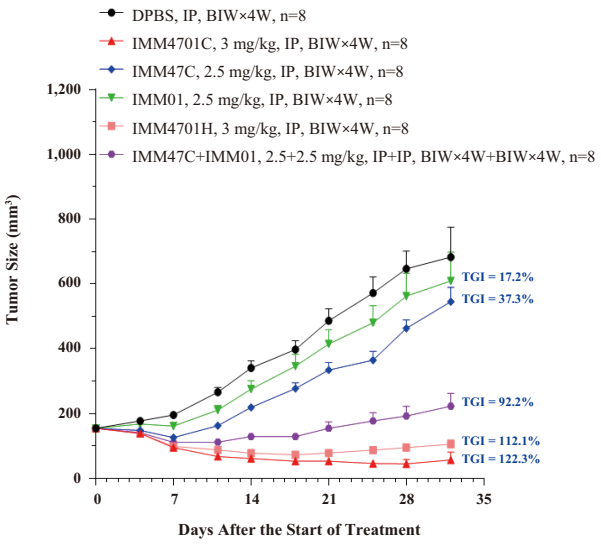
Notes: (1) Six mice per group were used in this study. (2) The colors of lines represent the different responses of the six mice in each group.
Source: Company Data

Our IMM47 has further demonstrated the ability to induce immunological memory against tumors in our *in vivo* studies. Mice treated with IMM47 established tumor-specific immune responses that prevented tumor growth even against re-inoculation of tumor cells, demonstrating IMM47's capability to further induce T-cell-based adaptive immune activation. Furthermore, our preclinical studies have revealed a strong synergistic effect of IMM47 when used in combination with OPDIVO® or KEYTRUDA® as compared to OPDIVO® or KEYTRUDA® monotherapy. For details, please see paragraphs and diagrams under the heading "— Our Innate Immune Checkpoint-targeted Drug Candidates — IMM47 (CD24 mAb)."

IMM4701 (CD47×CD24)

IMM4701 is a bispecific molecule with a mAb-Trap structure targeting CD47 and CD24 for the treatment of solid tumors. The mAb-Trap molecule structure of IMM4701 allows it to exhibit similar advantages in safety and efficacy as other CD47-based bispecific molecules designed by us. By targeting CD24 and CD47 simultaneously, IMM4701 can exert therapeutic effects stronger than the combination of single agents against those two targets. In our preclinical studies, IMM4701C, to which IMM4701 has highly similar *in vitro* efficacy, demonstrated potent *in vivo* antitumor activity. As illustrated in the below diagram, under the MCF-7 xenograft model in severe combined immunodeficiency disease (SCID) mice, IMM4701C resulted in reduced tumor size and exhibited a high potency when administered at 3.0 mg/kg (~0.3 mg/kg human equivalent dose).

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



Note: 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.

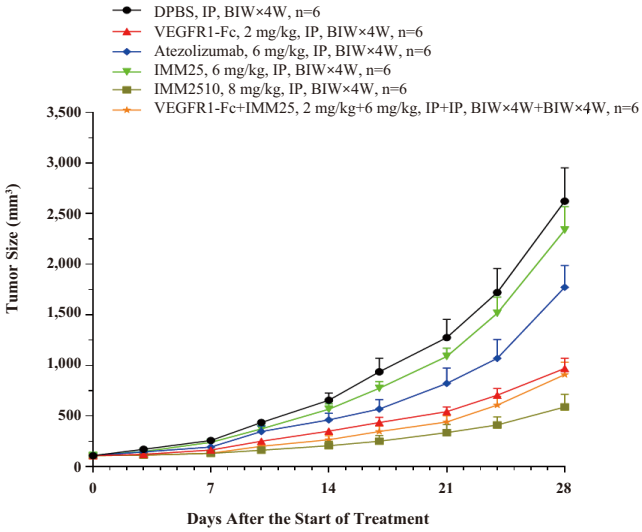
Source: Company Data

Selected adaptive immune checkpoint-targeted drug candidates

IMM2510 (VEGF×PD-L1)

IMM2510 is a bispecific molecule with a mAb-Trap structure targeting VEGF and PD-L1 for the treatment of solid tumors. Both VEGF and PD-L1 are clinically validated targets and have shown strong synergistic effects with the approval of combination therapies targeting these two pathways in many solid tumor indications. With the binding of VEGF and PD-L1, IMM2510 can simultaneously block the PD-L1/PD-1 pathway and the VEGF/VEGFR pathway, activating T-cell tumor killing and at the same time inhibiting tumor angiogenesis and tumor growth. IMM2510 can also activate NK cells and macrophages via IgG1 Fc-mediated ADCC and ADCP activities. As shown in the below diagram, our *in vivo* efficacy studies demonstrated that IMM2510 had a better efficacy profile than the VEGF blockers and PD-L1 antibodies either as a single agent or used in combination.

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



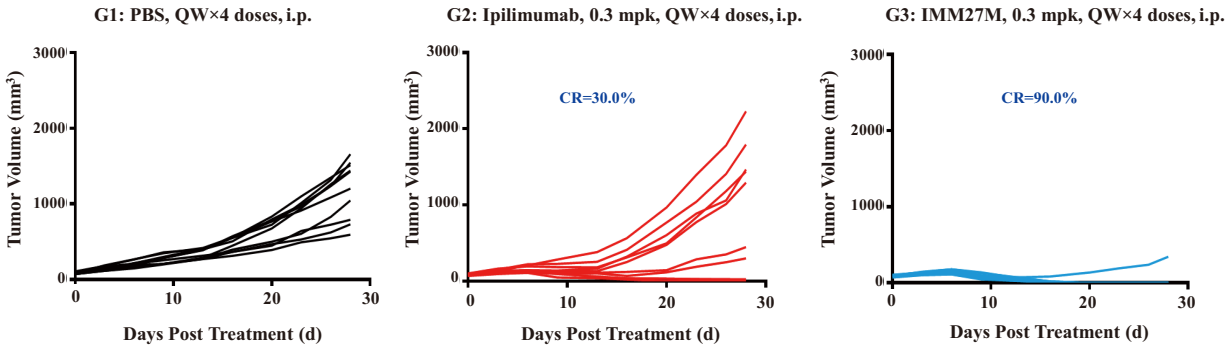
Note: IMM25 is an engineered PD-LI antibody with ADCC enhancement.
Source: Company Data

The initial results of the Phase I clinical trial have demonstrated preliminary efficacy of IMM2510. As of February 15, 2023, it was safe and tolerable up to 10.0 mg/kg in patients with advanced solid tumors. Among the two evaluable NSCLC patients in the trial so far, we have observed PRs in both patients with best tumor shrinkage response of 46% and 35% respectively.

IMM27M (CTLA-4 ADCC-enhanced mAb)

IMM27M is a new generation CTLA-4 antibody with enhanced ADCC activity through genetic-engineering modification. Through enhanced ADCC activities, IMM27M is able to induce enhanced immune responses against CTLA-4 overexpressed T_{reg} cells and promote T_{reg} depletion, thus enhancing T-cell antitumor responses. As illustrated in the diagrams below, our *in vivo* efficacy studies demonstrated that IMM27M could induce a significantly stronger antitumor activity than ipilimumab, and it resulted in complete tumor remission even at a dose as low as 0.3 mg/kg (~0.03 mg/kg human equivalent dose):

Efficacy Study in Colon Cancer (MC38) Syngeneic Mouse Model



Source: Company Data

We have commenced the Phase I clinical trial in solid tumors, with the first patient dosed in June 2022. We had enrolled 15 patients as of February 10, 2023, and we are currently enrolling patients for the sixth cohort of 5.0 mg/kg. The preliminary data demonstrates that IMM27M is safe and well tolerated up to 3.0 mg/kg. We have observed 4 SDs in this trial so far, among whom one patient with breast carcinoma who had six lines of prior treatment has achieved SD with tumor shrinkage of 28.8% at 3.0 mg/kg, and one patient with metastatic melanoma has achieved SD with tumor shrinkage of 22.9% at 2.0 mg/kg. We expect to complete this trial in mid-2023.

Integrated proprietary R&D engine anchored around our deep understanding of tumor immunology, continuously driving the discovery and development of innovative next-generation immunotherapies

We have established an integrated in-house R&D platform that covers target selection and validation, drug discovery, high-throughput screening, molecule design, preclinical studies, CMC and IND-enabling capabilities. Our platform enables us to continuously discover and develop next-generation innovative oncology therapies and move them forward to the clinical stage. The R&D engine includes a proprietary mAb-Trap bispecific platform, advanced hybridoma technology, high-throughput screening, strong immunoassay and bioassay technology, efficient cell line development and antibody production, as well as robust CMC and manufacturing capacity, which allow us to efficiently conduct screening for leading compounds and druggability analysis, cost-effectively manufacture high-quality drug candidates in-house, and provide firm support for our drug development efforts. Our R&D capabilities are anchored by our profound comprehension of biology and our stable R&D, CMC and regulatory affairs teams consisting of 62 members with extensive experience in drug discovery, preclinical research, process development and CMC.

Our integrated R&D platform enables us to effectively select novel targets, optimize molecule structure design and accelerate the drug development process. With proprietary hybridoma technology and know-how, we can efficiently identify and improve antibody fragments with higher specificity, affinity and other best-suited properties. We are currently exploiting the hybridoma technology and high-throughput screening to develop multiple therapeutic monoclonal antibodies, including IMM47, IMM40H, and discovery- and preclinical-stage candidates for several new targets, which are targets with no approved drugs in anywhere of the world. As a testament to our R&D competencies, we have successfully in-house discovered and developed over ten drug candidates with 17 IND approvals from the NMPA and the FDA. As of the Latest Practicable Date, around the globe, we owned 19 issued patents, nine allowed patent applications, 22 pending patent applications and one PRC patent application filed as a priority application, enabling us to tap into the global market and maximize the commercial value of our drug candidates.

Guided by our insights in tumor biology and immunology and our "DbD" concept, we have built the mAb-Trap bispecific platform to effectively facilitate the science-driven drug design and development. This platform enables us to connect engineered tumor target binding domains to the N-terminal of the heavy chain or light chain of respective antibodies, whichever is best suited for the targets we have selected, allowing for favorable binding affinity with tumor targets while preserving IgG1 Fc effector function. A number of bispecific molecules stemmed from this platform and demonstrated potent efficacy, good safety in preclinical studies. Four of those molecules (i.e., IMM0306, IMM2902, IMM2510 and IMM2520) have entered into clinical development stage. Preliminary clinical results of IMM0306 thus far have further validated the advantages of this unique molecule design and our mAb-Trap platform. IMM0306 was safe and well tolerated up to 2.0 mg/kg in patients. Among the evaluable patients across four cohorts dosed from 0.8 mg/kg to 2.0 mg/kg, who had relapsed or progressed after receiving rituximab previously, two CRs and five PRs were observed. The only evaluable FL patient at 2.0 mg/kg who relapsed and progressed after rituximab treatment has also been confirmed as PR. At 2.0 mg/kg, one patient with primary bone DLBCL who had four lines of prior treatment has achieved PR with all measurable lesions disappeared after 65 days of treatment.

In addition, the symmetric structure of these mAb-Trap bispecific molecules, similar to that of native antibodies, allows for ease of manufacturing, product stability, higher tier and protein yield, and makes the CMC process and production by standard antibody manufacturing techniques more feasible. Our mAb-Trap bispecific candidates thus have consistently maintained high production yield in our manufacturing process. For example, the average protein yield for IMM0306, IMM2902, and IMM2520 ranges from 3.8 g/L to 4.6 g/L, much higher than the industry average for bispecific molecules of 1.0 g/L to 3.0 g/L.

Seasoned management team with a track record of drug innovation and clinical development, led by a renowned immunologist founder and backed by blue chip investors

Our founder, chief executive officer and chief scientific officer, Dr. Wenzhi Tian, EMBA, is a renowned expert in cancer immunotherapies. Dr. Tian brought us over 30 years of academic and industrial experience in the field of immuno-oncology. Based on his in-depth understanding of cancer immunology, Dr. Tian has been at the forefront of scientific research and built a proven track record in target validation, molecule design and drug development for innovative immunotherapies. He identified CD47 as a promising immunotherapeutic target and commenced drug research on CD47 starting from 2010, roughly 10 years earlier than the validation of CD47 by clinical data. His deep expertise and foresight in target selection also led to our development of multiple monoclonal antibody and bispecific molecules targeting CD24, another promising checkpoint since 2019, all with global first-in-class potential. A prolific scientist, Dr. Tian invented 13 issued patents, 9 allowed patent applications and 27 patent applications, and published over 30 scientific papers in the area of immunology and CD47 in internationally-recognized journals.

Led by Dr. Tian, we have assembled a leadership team with extensive preclinical and clinical development experience and a proven track record of drug innovation. Core members of our R&D team, led by Mr. Song Li and Mr. Ruliang Zhang, have been working with Dr. Tian for close to 10 years.

Mr. Song Li, our vice president of R&D, has over 10 years of experience in antibody drug discovery and process development. He has led the drug discovery and preclinical development of all our IND-approved drug candidates. Mr. Li possesses solid expertise in lead selection, antibody engineering and optimization, cell line and process development and antibody characterization. Mr. Li holds 11 issued patents, 8 allowed patent applications and 24 patent applications.

Mr. Ruliang Zhang, our deputy general manager and senior vice president, has over 15 years of CMC, quality control, regulatory and project management experience in the biopharmaceutical industry. Mr. Zhang has successfully advanced 8 drug candidates into clinical stage with 17 IND approvals, among which 13 were approved by the NMPA and four were approved by the FDA.

Our team also has other seasoned executives who leverage their extensive experience at leading multinational pharmaceutical companies and top investment banks.

Dr. Qiying Lu, our senior vice president and chief medical officer, has around 20 years of experience in clinical practice and innovative oncology drug development. He brings us valuable long-term experience with multinational pharmaceutical companies, including GlaxoSmithKline, AstraZeneca, and Pfizer. During his tenure at Pfizer, he successfully led the strategy development until regulatory marketing approval of various drug candidates in China, including IBRANCE® and VIZIMPRO®.

Dr. Frank Xiaodong Gan, our senior vice president, has over 25 years of experience in preclinical and clinical development in the academia and biopharmaceutical industry. Dr. Gan has accumulated a wealth of clinical development experience in critical positions over the years at

various prestigious multinational pharmaceutical companies. Dr. Gan led the global clinical development of various drug candidates and played an important role in the successful market launch of numerous products, including CYRAMZA®, BALVERSA®, and JANUVIA®.

Ms. Ziyi Song, our chief financial officer, brings us over 15 years of capital markets experiences gained at global investment banks, combined with solid biomedical sector knowledge developed through her educational background in medical sciences, healthcare-focused investment banking and investment management experiences. Ms. Song has extensive experience in capital market and corporate strategy through executing high-profile capital market transactions, including IPOs, financings, M&As, and healthcare investment.

Dr. Zikai Xiong, Ph.D., our senior vice president of Business Development, has extensive experience in the biotechnology industry, ranging from pharmaceutical giants to startups. During his career, Dr. Xiong held key strategy and business development positions in multinational corporations and biotechnology companies. For further details of our senior management's proven track record and industry experience, please refer to the section headed "Directors, Supervisors and Senior Management" in this document.

We are also backed by multiple global and locally recognized blue-chip institutional investors and healthcare-focused specialized investment funds, including, among others, Lilly Asia Ventures, LYFE Capital, Shanghai Science and Technology Innovation Fund, and RemeGen VC. We have raised approximately US$216 million in capital across 6 series of financings within 6 years, demonstrating the market's strong confidence in our business potential.

OUR STRATEGIES

To advance the development of our drug candidates to unleash their therapeutic potential and address significant unmet medical needs

We have formulated and are implementing a stepwise clinical development strategy that would allow us to thoroughly evaluate the therapeutic potential of our innate and adaptive immunity-based candidates spearheaded by our CD47 portfolio and CD24 candidates, and expand their clinical application, with an aim to ultimately overcome the limitations of the current standard of care and potentially reshape the tumor-treatment paradigm globally.

Leveraging the expertise of our clinical development team, we are rapidly advancing the clinical development of our drug candidates targeting innate and adaptive immune checkpoints to treat a wide array of hematologic malignancies and solid tumors and address significant unmet medical demands.

Treatment of hematologic malignancies

Accumulating clinical evidence has supported the effectiveness of CD47-targeted agents in treating hematologic tumors. We are developing IMM01, IMM0306 and other pipeline candidates for the treatment of hematologic malignancies, such as lymphoma, MDS/CMML and AML, through monotherapy and combination strategies. Our clinical development plans for our programs that target hematologic malignancies are as follows:

- IMM01 in combination with azacitidine: Upon completion of the Phase Ib trial, we initiated a Phase II trial to evaluate the combination therapy of IMM01 and azacitidine mainly for the first-line treatment of HR MDS, unfit AML and CMML in June 2022, from which so far we have observed an encouraging efficacy and safety profile of this combination therapy. We expect to commence a pivotal trial in China in the fourth quarter of 2023. In particular, we plan to seek an accelerated marketing approval through relatively small sample size studies targeting the first-line treatment of CMML,

a rare type of disease with highly unmet medical needs. Subject to further clinical validation, we plan to file an IND application with the FDA for a Phase II study of this combination treatment.

- IMM01 in combination with tislelizumab: We are evaluating this combination therapy in cHL patients who relapsed or progressed after the treatment of PD-1 inhibitors, which may allow us to pursue an accelerated marketing approval leveraging the results of relatively small sample size studies. We dosed the first patient with R/R cHL in China in January 2023.

- IMM0306 (CD47×CD20): We initiated a Phase I trial for IMM0306 in R/R B-NHL in China in May 2020, of which preliminary data showed good safety and promising efficacy. We commenced a Phase IIa trial for IMM0306 monotherapy for the third- or later-line treatment of FL in March 2023 and plan to seek an accelerated marketing approval through a single-arm trial. We expect to commence pivotal clinical trials in China in the third quarter of 2024. Furthermore, our IND application for the combination of IMM0306 and lenalidomide targeting front-line B-NHL was approved by the NMPA in January 2023, and we are in preparation to commence the Phase Ib trial for this combination in China. We have also received an IND approval for IMM0306 from the FDA in January 2021. With further clinical validation in the Phase I trial in China, we will then decide on our clinical development and collaboration strategy for IMM0306 in the U.S.

- IMM40H (CD70 mAb): We have obtained IND approvals for IMM40H from the NMPA and the FDA in August 2022, and may initiate Phase I clinical studies or pursue potential collaboration opportunities.

Treatment of solid tumors

Parallel with the programs targeting hematologic malignancies, we are also actively advancing the development of our pipeline candidates for the treatment of solid tumors, which would allow us to tap into a huge market with a large patient population. The synergies of our selected innate and adaptive immune targets also multiply the combination potential among our pipeline assets for the treatment of a wide range of tumor indications. Our clinical development plans for our programs targeting solid tumors are as follows:

- CD47-targeted drug candidates

  - IMM01 in combination with tislelizumab: We initiated the Phase Ib/II trial to evaluate IMM01 in combination with tislelizumab for the treatment of various advanced solid tumors that failed to respond to or relapsed from the standard of care such as PD-1/PD-L1 inhibitors, including among others, NSCLC, SCLC, HNSCC and CRC in May 2022. After accumulating more clinical data, we may further evaluate this combination therapy for the first-line treatment of those solid tumors as well as for the treatment of other cancer indications. We expect to initiate a pivotal trial in the third quarter of 2024.

  - IMM2902 (CD47×HER2): We initiated a Phase Ia/Ib trial for IMM2902 in advanced HER2-positive and HER2-low expressing solid tumors, including BC, GC, NSCLC and BTC, in China in February 2022. Based on an IND approval for IMM2902 in HER2-positive and HER2-low expressing solid tumors granted by the FDA in August 2021, we have also initiated the clinical trial for advanced HER2-positive and HER2-low expressing solid tumors in the U.S. with the first patient dosed in June 2022. We expect to largely complete the Phase Ia trials in China and the U.S. in 2023.

- IMM2520 (CD47×PD-L1): We have obtained IND approvals for IMM2520 from the NMPA in November 2022 and from the FDA in December 2022, and dosed the first patient for the Phase I clinical trial in China in March 2023. We will particularly focus on the solid tumors generally resistant or not sensitive to the currently available immunotherapies, such as CRC, GC, lung cancer and HNSCC, among others.

- CD24-targeted drug candidates

  - IMM47 (CD24 mAb): We expect to file IND applications for IMM47 with the NMPA and the FDA for the treatment of solid tumors in 2023, and initiate a Phase I dose-escalation study first in Australia in mid-2023. Initiating a clinical trial in Australia first can help us to begin global clinical trials earlier and accelerate clinical validation of IMM47. Additionally, we believe Australian trial can generate valuable clinical data on ethnically diverse populations, thus enhancing our ability to pursue collaboration opportunities with global pharmaceutical companies.

  - IMM4701 (CD47×CD24): Further leveraging the data observed from IMM47, we expect to file IND applications for IMM4701 with the NMPA and the FDA for the treatment of solid tumors, and further seek collaboration opportunities with global pharmaceutical companies.

- Adaptive immune checkpoint-targeted drug candidates

  - IMM2510 (VEGF×PD-L1): We received the IND approval for IMM2510 from the NMPA in December 2020. We commenced the Phase I dose-escalation trial for IMM2510 in China in August 2021 for the treatment of a variety of advanced solid tumors, including but not limited to, HCC, RCC, GC, NSCLC and STS. We expect to complete this dose-escalation study in mid-2023, and subsequently commence a cohort-expansion study.

  - IMM27M (CTLA4 ADCC+): We obtained the IND approval for IMM27M from NMPA in November 2021 have commenced the Phase I clinical trial in solid tumors, with the first patient dosed in June 2022. We expect to complete this trial in mid-2023. We received an IND approval from the NMPA for a Phase Ib/II study to evaluate the combination of IMM27M and a PD-1 antibody for the treatment of advanced solid tumors, such as RCC, NSCLC, GC and TC, in March 2023. We may initiate clinical trials or explore collaboration opportunities for this combination therapy.

  - IMM40H (CD70): We have obtained IND approvals for IMM40H from the NMPA and the FDA in August 2022, and may initiate Phase I clinical studies or pursue potential collaboration opportunities.

Upon obtaining supportive clinical evidence from our ongoing trials in patients with cancers resistant to currently available therapies, we will further advance our clinical trials towards first-line treatment to expand the market share for our drug candidates. To achieve such goal, we plan to conduct head-to-head clinical trials to evaluate our drug candidates against standard-of-care approved for first-line treatment. Clinical use in the first-line setting will open a significant market for our drug candidates due to larger patient populations and a comparatively longer treatment duration.

To expand our global footprint and maximize the clinical and commercial value of our drug candidates through global clinical trials and accretive partnerships

We endeavor to expand our global footprint and develop next-generation immuno-oncology therapies to fully grasp global market opportunities. We have designed a clear overseas clinical development strategy with an initial focus on the U.S. market. We plan to rapidly advance early-stage clinical studies in China, and may subsequently leverage the China data to obtain IND approvals for Phase II clinical studies in the U.S. in order to save the time and costs of clinical development in the overseas market. As many of our drug candidates have global first-in-class potential, we believe we are well-positioned to conduct multi-regional clinical trials to obtain marketing approvals in multiple countries and seek potential collaboration opportunities in the global market. Dr. Frank Xiaodong Gan, an industry expert with notable clinical development experience at multiple prestigious pharmaceutical companies in the U.S., including Merck, Bristol Myers Squibb, Eli Lilly and Janssen, joined us as our Senior Vice President with responsibility to lead the clinical development in the United States. Moreover, leveraging our experienced senior management team's deep-rooted network within the medical community in the U.S., we have collaborated with reputable principal investigators to formulate scientific clinical designs and engaged industry-leading CROs for efficient clinical development. We will continue to strengthen our relationships with these principal investigators and CROs, and actively explore other cooperation opportunities globally. We have obtained IND approvals from the FDA for, among others, IMM0306, IMM2902, IMM2520, and IMM40H. Based on an IND approval granted by the FDA in August 2021 for IMM2902, we have initiated the clinical trial for advanced HER2-positive and HER2-low expressing solid tumors in the U.S. with the first patient dosed in June 2022. With the support of further clinical validation from the China trials, we also plan to file an IND application for a Phase II trial for the combination of IMM01 and azacitidine with the FDA.

To penetrate the global market in a cost-effective and efficient manner, we will also actively seek strategic collaboration opportunities, including licensing arrangements, co-development and/or co-commercialization arrangements to optimize our pipeline structure, expedite the development of our drug candidates, broaden their addressable patient population, and accelerate the penetration in a variety of markets. We have presented preclinical and clinical data of our drug candidates at various international conferences including annual meetings of AACR, ASH and ASCO, to attract the interest of potential strategic partners. With the proven R&D capabilities and the encouraging preclinical and clinical data of our drug candidates, we believe that we are well-positioned to form value-accretive partnerships with renowned global and local pharmaceutical companies. In particular, we are seeking out-licensing and co-commercialization opportunities for our pipeline with partners that possess (i) strong medical and clinical resources to advance our global clinical development; and/or (ii) an established commercialization infrastructure, including a strong local salesforce, a broad distributor network, and a long-standing relationship with commercial insurers, health maintenance organizations, and pharmacy benefit managers.

In addition to our selected pipeline discussed above, leveraging our strong discovery and R&D capability as well as our integrated R&D platform, we have developed multiple drug candidates in discovery and preclinical stage. To fully unleash the value of our comprehensive product pipeline, we will strategically seek out-licensing and other collaboration opportunities for certain drug candidates, such as IMM2518, a second-generation VEGF×PD-L1 bispecific molecule and IMM5601, a CD47×CD38 bispecific molecule, among others, all of which are in preclinical stage. In particular, when seeking potential partners, we expect to reach satisfactory commercial terms with those who possess strong resources and capabilities to advance the clinical development of our drug candidates efficiently. We may also consider strategic collaboration and co-development opportunities with companies that have complementary oncology portfolio with synergistic potential to combine with our drug candidates. We intend to identify and collaborate with the most suitable and resourceful partners, and leverage the complementary capabilities and differentiated expertise of such business partners to maximize the clinical and commercial value of

our drug candidates. Furthermore, to maximize the value of our pipeline, we will carefully assess licensing and other collaboration arrangements in the context of our overall development strategy to prevent potential competition among our drug candidates in the same regions or for the same indications.

To continuously enrich our innovative pipeline through fundamental biological research and translational medicine

The development of innovative cancer therapies requires pioneering foresight in target selection and validation. Leveraging our profound expertise in immunotherapies, we are able to strategically select the targets and effectively design and screen our molecules with a sound scientific rationale and strong validation in preclinical studies. To address the limitations of current immunotherapies, we have established our in-house drug discovery and design capabilities with integrated R&D platform. We believe our comprehensive knowledge in tumor biology and immunology and strong R&D capabilities and technologies serve as the driving force that propels our steady efforts to validate novel targets, improve molecule design and ultimately deliver innovative medicine with clinical potential.

We are determined to continue enriching our pipeline by actively exploring new immuno-oncology mechanisms and translating fundamental biological research into promising drug candidates. We have adopted a systematic approach to research and validate the mechanism of action of novel targets and pathways, assess their clinical significance and global competitive landscape, and screen, develop and design molecules with the best-suited structure and properties, to address unmet medical needs globally. With our methodical approach and integrated R&D platform, we are currently developing multiple therapeutic monoclonal antibodies, including IMM47, IMM40H, and discovery- and preclinical-stage candidates for several new targets, which are targets with no approved drugs in anywhere of the world. Around CD24, validated by us as another promising innate immune checkpoint, we have developed one IND-enabling-stage and multiple discovery- and preclinical-stage candidates which exhibit global first-in-class potential. We have also been actively exploring prospects of other innovative immune checkpoints, such as IL-8, NKG2A and PSGL-1. We will continue to single out and evaluate other promising innate immune checkpoints and enrich our pipeline with novel therapies. We are committed to identifying and validating promising immuno-oncology targets, as well as screening and advancing innovative molecules with the optimum structure for each target. If a novel pathway or target is identified as having combinatorial potential with our drug candidates, we may further explore such potential by building up combination therapies or bispecific molecules.

We will continue to invest in our translational medicine research capabilities to expedite our bench-to-bedside process, which we believe would put us at the forefront of the race to market. Meanwhile, we plan to conduct all-encompassing patient sub-group analyses to identify biomarkers that are predictive of the efficacy of our drug candidates. The biomarkers may assist in identifying patients who will benefit the most from the treatment. The expression level of a common biomarker may signal the effectiveness of our approach across a range of tumor indications. In this way, we would be able to present more precise treatment to a wider group of patients.

To upscale our GMP-compliant manufacturing capacity

We believe a self-sufficient manufacturing capability will grant us many strategic advantages, including improved cost-effectiveness, enhanced quality control, and flexibility in supply chain management. Those advantages are critical for us to grow into the integrated biopharmaceutical company that we envision. Our current pilot production line with the scale of 450L enables us to produce high-quality drugs used in clinical trials in-house for certain drug candidates. We intend to strategically expand our GMP-compliant manufacturing capacity, while improving efficiency and cost-effectiveness. We have already commenced the construction of our new manufacturing facility that occupies a site area of approximately 28.7 thousand square meters in Zhangjiang Science City,

Shanghai. This facility is designed to meet the stringent cGMP standards. We plan to complete the first stage of construction by 2025, which will support clinical and commercial production of our pipeline products. Prior to completion of the construction, we will collaborate with CDMOs and utilize our in-house pilot manufacturing facilities to manufacture our drug candidates for preclinical studies and clinical trials. We will commence the second stage of construction, depending on the schedule of the regulatory approval and sales ramp-up of our drug portfolio in the future.

To enlarge our talent pool to support our continuous growth

We place a high priority on selecting and retaining top talents. To fully support our growth, we will continue to recruit industry-leading R&D, clinical development, and commercialization professionals. We are committed to providing our employees with robust career development and learning opportunities, mentorship from our industry veterans, clear career trajectories, competitive compensation, and a close-knit and supportive work environment.

With more of our drug candidates advancing into the clinical stage, in the near term we will strengthen our clinical development team by attracting talents with extensive experience both in China and globally, to support clinical development and regulatory affairs in our target markets. We believe that, under the guidance of our seasoned and capable clinical development management team, our new team members can make a significant contribution to our clinical development progress. We also plan to expand our translational medicine team acting as an engine to support our continuous innovative drug development by recruiting talented personnel with interdisciplinary backgrounds.

In the longer term, to facilitate our transformation from a biotechnology company to a biopharmaceutical company, we intend to establish a team of dedicated in-house sales staff to execute our commercialization strategy and seek commercialization partnerships with other pharmaceutical industry players. We also plan to build a team of pharmaco-economics experts to develop our competitive pricing strategy, with an aim to facilitate the inclusion of our products into the National Reimbursement Drug List (NRDL) in China as well as the commercial insurance catalogue in the overseas markets. To unleash the market potential of IMM01, we will actively prepare and participate in the price negotiation with the regulators for its inclusion in the NRDL in China, upon obtaining marketing approval. We will also seek its inclusion in commercial insurance catalogue overseas through potential commercialization partners in the global market to further increase its accessibility.

OUR DRUG CANDIDATES

As a science-driven innovative biotechnology company, we have internally developed all of our pipeline candidates by utilizing our proprietary and integrated R&D platforms. Differentiated from companies that are focused primarily on the development of immunotherapies targeting adaptive immune checkpoints, mostly T-cell-based therapeutics, we constructed our pipeline to harness both innate and adaptive arms of immunity to unleash their synergistic potential. Our pipeline is designed to address the limitations of current T-cell-based immunotherapies, such as limited response due to "cold tumors" or non-T cell-inflamed immune-suppressive TME, thereby bringing clinical benefits to patients with a wide range of cancer indications. As of the Latest Practicable Date, we had built up a robust innovative pipeline composed of over ten drug candidates targeting critical innate and adaptive immune pathways, with eight ongoing clinical programs. We own worldwide IP and commercial rights to our pipeline candidates, which allows us to address critical medical needs in the global market.

As of the Latest Practicable Date, we had eight ongoing clinical programs in China and/or the U.S., four IND-stage and one IND-enabling-stage programs, and multiple discovery and preclinical-stage assets. The following chart summarizes the development status of our selected drug candidates as of the Latest Practicable Date:

	Program	Target (Modality)	Indication(s)	Discovery	Preclinical	IND/IND-Enabling	Phase Ia/I	Phase Ib/II	Phase III/Pivotal	Current Status / Upcoming Milestone[1]	Commercial Rights
Innate Immunity	**IMM01** ★	CD47 (SIRPα-Fc fusion protein)									
	IMM01 + Azacitidine		MDS, AML, CMML[2]				China (NMPA)			Phase Ib/II commenced in January 2022; expect to initiate pivotal trial in Q4 2023	Global
	IMM01 + Tislelizumab	CD47+PD-1	cHL, Solid tumor				China (NMPA)			Phase Ib/II commenced in May 2022; expect to initiate pivotal trial in Q3 2024[3]	Global
	IMM01 + Inetetamab	CD47+HER2	HER2-positive solid tumors				China (NMPA)[4]			Phase Ib/II IND approved	Global
	IMM01 + Bortezomib + Dexamethasonum	CD47	MM				China (NMPA)			Phase Ib/IIa IND approved	Global
	IMM0306 ▲ Monotherapy	CD47xCD20 (Bispecific)	Indolent B-NHL				China (NMPA), US (FDA)			Phase IIa commenced in March 2023 in China; IND approved in the U.S.	Global
	IMM0306 + Lenalidomide	CD47xCD20 (Bispecific)	B-NHL				China (NMPA)			Phase Ib/IIa IND approved	Global
	IMM2902 ▲	CD47xHER2 (Bispecific)	HER2-positive and low-expressing solid tumors				China (NMPA), US (FDA)			Phase I commenced in February 2022 in China and in June 2022 in the U.S.; expect to largely complete Phase Ia trials in China and the U.S. in 2023	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
	IMM47	CD24 (mAb)	Solid tumors				China (NMPA), US (FDA)			IND-enabling; expect to enter into clinical trials in mid-2023	Global
	IMM4701	CD47xCD24 (Bispecific)	Solid tumors							CMC	Global
	IMM2547[5]	CD24xPD-L1 (Bispecific)	Solid tumors							Discovery	Global
	IMM51[5]	IL-8 (mAb)	Solid tumors							Preclinical	Global
	IMM38[5]	Undisclosed	Solid tumors							Preclinical	Global
	IMM50[5]	Undisclosed	Solid tumors							Discovery	Global
	IMM62[5]	Undisclosed	Solid tumors							Discovery	Global
Adaptive Immunity	**IMM2510**	VEGFxPD-L1 (Bispecific)	Solid tumors				China (NMPA)			Phase I commenced in August 2021 and 8th cohort ongoing in China; expect to complete Phase I in mid-2023	Global
	IMM27M	CTLA-4 ADCC+ (mAb)	Solid tumors				China (NMPA)			Phase I commenced in June 2022 in China; expect to complete in mid-2023; IND approved in China for Phase Ib/II trial for its combination with a PD-1 antibody[6]	Global
	IMM40H	CD70 (mAb)	Liquid/Solid tumors				China (NMPA), US (FDA)			IND approved in China and the U.S. in August 2022	Global

★ Core Product ▲ Key Product

Innate Immunity Targets | Innate and Adaptive Immunity Targets | Adaptive Immunity Targets

Notes:

(1) Expected completion date for Phase Ia/I trial refers to the time when RP2D can be determined, and expected completion date for Phase Ib/II trial refers to the time when top-line data is available for regulatory discussions. Follow-up period required would not delay the initiation of the next phase clinical trials, and is thus not considered.

(2) The cohort-expansion trials of this combination are mainly designed to target the first-line treatment of higher-risk MDS (patients who fall into higher-risk group categories in the original or revised International Prognostic Scoring System), unfit AML (individuals of older age with AML who are considered not eligible for intensive treatment approaches), and CMML. Particularly, we plan to seek an accelerated marketing approval through relatively small sample size studies targeting the first-line treatment of CMML, a rare type of disease with highly unmet medical needs.

(3) In July 2022, we obtained the NMPA's consent for adding R/R cHL as an additional expansion cohort into the ongoing combination trial of IMM01 and tislelizumab. We dosed the first patient with R/R cHL in China in January 2023.

*(4) The clinical trial is led and funded by Sunshine Guojian Pharmaceutical (Shanghai) Co., Ltd. ("**Sunshine Guojian**"). As denoted by the dotted line, Sunshine Guojian and us have obtained an IND approval for a Phase Ib/II trial of this combination therapy from the NMPA in August 2021, and therefore the parties can skip the Phase Ia stage and directly initiate a Phase Ib/II trial.*

(5) We will continue to conduct preclinical studies for IMM2547, IMM51, IMM38, IMM50 and IMM62, including cell line development, in vivo studies and further evaluation.

(6) We are currently conducting the Phase I trial for IMM27M monotherapy, and have obtained the IND approval for a Phase Ib/II trial for its combination with a PD-1 antibody.

* *Currently we have several other drug candidates in preclinical stage and plan to further develop these candidates through collaboration, such as IMM2518, a second-generation VEGF×PD-L1 bispecific molecule and IMM5601, a CD47×CD38 bispecific molecule.*

Abbreviations: MDS refers to myelodysplastic syndrome; AML refers to acute myeloid leukemia; CMML refers to chronic myelomonocytic leukemia; MM refers to multiple myeloma; B-NHL refers to B-cell non-Hodgkin lymphoma; cHL refers to classical Hodgkin lymphoma; IND refers to investigational new drug; CMC refers to chemistry, manufacturing, and controls; ADCC refers to antibody-dependent cellular cytotoxicity.

Source: Company Data

Our drug candidates are subject to BLA approval by relevant authorities, such as the NMPA in China and the FDA in the U.S., before commercialization in relevant jurisdictions. As of the Latest Practicable Date, we had not received any material concerns, objections or negative statements raised by the NMPA, the FDA or other relevant authorities that we are not able to address in a timely manner. We believe we are on track to advance the development of our discovery- and preclinical-stage as well as clinical-stage drug candidates as described in "— Our Drug Candidates."

OUR INNATE IMMUNE CHECKPOINT-TARGETED DRUG CANDIDATES

To overcome the limitations of T-cell-based immunotherapies, we have strategically designed and built a portfolio consisting of four clinical-stage, one IND-enabling-stage and multiple discovery- and preclinical-stage drug candidates targeting innate immune checkpoints. This portfolio includes: (i) a CD47-targeted fusion protein, IMM01 (SIRPα-Fc), being or to be evaluated in combination with each of azacitidine, tislelizumab (PD-1 mAb), inetetamab (HER2 mAb) and bortezomib/dexamethasonum for the treatment of hematologic and solid tumors, (ii) three CD47-based clinical-stage mAb-Trap bispecific molecules with the ability to achieve enhanced tumor-killing effects via ADCP and ADCC activated through IgG1 Fc effector function, namely IMM0306 (CD47×CD20), IMM2902 (CD47×HER2), and IMM2520 (CD47×PD-L1), as well as multiple preclinical-stage CD47-based bispecific molecules, including IMM4701 (CD47×CD24), (iii) one IND-enabling-stage humanized CD24-targeted monoclonal antibody, IMM47 (CD24 mAb), and several CD24-targeted discovery- and preclinical-stage bispecific molecules, including IMM4701 (CD24×CD47) and IMM2547 (CD24×PD-L1), and (iv) various discovery- and preclinical-stage drug candidates targeting other novel innate immune targets, including IL-8, NKG2A and PSGL-1.

Our Approach

The immuno-oncology therapies present huge clinical and commercial potential. According to Frost & Sullivan, the global immuno-oncology drug market is expected to represent 50.6% of the overall global oncology drug market in 2030. Currently, approved immunotherapies primarily target T-cell immune checkpoints, such as PD-1/PD-L1, CTLA-4, and LAG-3. T-cell immune checkpoint inhibitors have revolutionized the treatment paradigm for many cancer indications in the past decade. However, only about 10% to 25% of patients across almost all major cancer types can benefit from PD-1/PD-L1 monotherapy treatment. The low response rates could be due to lack of T-cell infiltration in "cold tumors," or non-T cell-inflamed immune suppressive TME.

In response to this challenge, we have developed and built a deep portfolio of drug candidates targeting innate immune checkpoints. Major types of innate immune cells (macrophages, NK cells and DCs) widely exist in almost all types of body tissues, including lung, esophagus, stomach, liver, small and large intestines, and serve as the first line of defense against tumor cells. Macrophages, in particular, are widely distributed in a broad range of tumor types, accounting for 20% to 50% of cells in respective tumor tissues, higher than T cells' 10% to 30% tissue distribution. Upon activation, macrophages can ingest other cells and pathogens, including phagocytotic activity against tumor cells. Activated macrophages can release a slew of cytokines and chemokines, such as CXCL9 and CXCL10, to recruit T cells to tumor sites, effectively turning immune-suppressive "cold tumors" into immune-sensitive "hot tumors." They can also present tumor-associated antigens to T cells and elicit tumor-specific adaptive immune responses. Activated NK cells can mediate ADCC against tumor cells and promote T-cell differentiation, thus

further enhancing T cell responses. DCs can also attract T cells into the TME and activate T-cells through antigen presentation. The following table sets forth an overview and comparison of the key adaptive and innate immune cells in the TME:

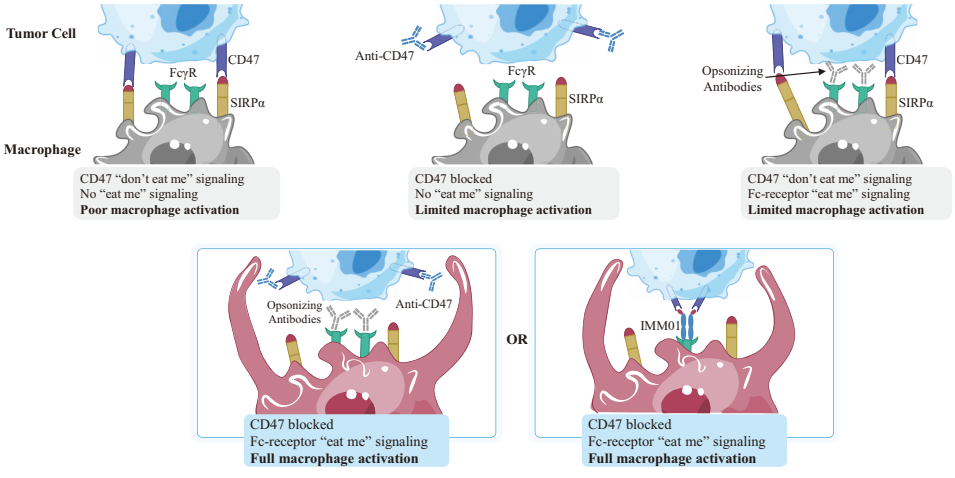
	Adaptive Immunity		Innate Immunity		
Activation Process	Antigen priming required		First line of defense, short response time, no need for antigen priming		
Key Immune Cell Type	T cell	B cell	Macrophage	NK cell	DC
Tumor Tissue Distribution(1)	10-30%	3%-40%	20-50%	5%-10%	3%-10%
Major Immune Functions	• T-cell mediated killing of tumor cell via exocytosis of cytotoxic granules (perforin, granzymes) and secretion of antitumor cytokines	• Antibody production • Cytokine secretion	• Macrophage-mediated phagocytosis • Attracting T cells to the tumor microenvironment (TME) • Antigen presentation • Trogocytosis	• NK cell-mediated cytolysis via the secretion of perforin and granzymes • Activating of T cells, macrophages and DCs through release of cytokines	• Attracting T cells to the TME • Antigen presentation

Note: The tumor tissue distribution is the proportion of certain immune cells in different tumor tissues.
Source: Frost & Sullivan

CD47 has been recognized as a critical macrophage checkpoint that plays a broad role in cancer immune evasion across multiple cancer types. CD47 interacts with SIRPα, an inhibitory receptor expressed on macrophages. By binding to SIPRα, CD47 conveys a "don't eat me" signal to inhibit macrophage-mediated tumor phagocytosis. Upregulating CD47 is a primary mechanism by which tumor cells evade attack by the innate immune systems. High CD47 expression is often correlated with aggressive disease and poor outcomes in a wide range of hematologic and solid tumors. Blocking the CD47-SIPRα axis has been validated in various clinical studies as an effective approach for the development of immunotherapeutics. Supported by mounting clinical evidence, this therapeutic strategy has shown a great potential to treat both hematologic and solid tumors, including lymphoma, MDS, AML, GC, HNSCC and SCLC.

However, research has revealed that to fully activate macrophages, blocking the "don't eat me" signal of CD47-SIPRα axis alone is not enough. Therapeutic agents, either as monotherapy or in combination therapy, must also deliver an activating "eat me" signal to macrophages via Fc-FcγR engagement or other costimulatory pathways. The following diagram illustrates the mechanism of full macrophage activation through both CD47 blockade and the activation of an "eat me" signal:

Dual Mechanisms of Macrophage Activation



Source: Frost & Sullivan, Literature Review

Driven by our understanding of and deep insights into cancer immunology, we have designed and developed IMM01, a SIRPα-Fc fusion protein with an IgG1-Fc region. Unlike IgG4 Fc adopted by most CD47 antibodies, IgG1 Fc used in IMM01 is able to elicit strong ADCP activity mediated by macrophages through efficient engagement with Fcγ receptors. IMM01 can thus exert a dual mechanism to simultaneously (i) block the CD47-SIRPα "don't eat me" pathway, and (ii) activate the "eat me" signal via Fc-FcγR engagement. Given the potent efficacy and favorable safety attributable to its unique molecule design, we have been exploring IMM01's combination potential with other cancer agents, and have designed multiple bispecific molecules that incorporate the engineered CD47-binding domain of IMM01 with an IgG1 Fc.

IMM01 (SIRPα-Fc Fusion Protein) — Our Core Product

IMM01 is a next-generation CD47-targeted molecule that displays favorable safety and encouraging efficacy in clinical studies. IMM01's favorable safety profile demonstrated in clinical trials is attributable to its specifically-engineered CD47-binding domain of human SIRPα, which does not bind to human RBCs. In terms of efficacy, IMM01 designed with IgG1 Fc can fully activate macrophages by delivering the additionally required "eat me" signal, and induce ADCC by activating NK cells. As a result, IMM01 can lead to all-around innate and adaptive immune responses, demonstrated by its encouraging single-agent efficacy even at a relatively low dose. Thus, we are able to establish the RP2D for IMM01 monotherapy at 2.0 mg/kg, much lower than most CD47 antibodies (typically in the range of 30.0 to 45.0 mg/kg). A lower effective dose of IMM01 allows for a better safety profile. With encouraging efficacy and favorable safety in monotherapy clinical trials and robust preclinical data of its combination studies, IMM01 is expected to achieve strong synergistic effects used in combination with other cancer agents.

IMM01 is being developed for the treatment of various hematologic malignancies and solid tumors in combination with other agents. We own the global IP and commercial rights of IMM01. As of the Latest Practicable Date, with respect to IMM01, we owned one patent family, which includes one issued patent in China, one issued patent and two pending patent applications in the U.S., one issued patent in Japan, one allowed EU patent application and one PCT patent application which has entered national phases.

Our founder, Dr. Wenzhi Tian, started to explore the therapeutic potential of CD47-targeted strategy in oncology as early as 2010, long before it became widely recognized in the biopharmaceutical industry. Leveraging the fundamental insights into CD47, we started our development efforts on IMM01 since our inception in 2015, which later became the first CD47-targeted SIRPα-Fc fusion protein to enter into clinical stage in China. We (i) have completed the Phase I dose-escalation study of IMM01 in R/R lymphoma patients, (ii) have completed a Phase Ib trial to evaluate IMM01 in combination with azacitidine for the treatment of R/R MDS and R/R AML, and initiated a Phase II trial mainly for the first-line treatment of HR MDS, unfit AML and CMML in June 2022, and (iii) initiated a Phase Ib clinical trial for the combination of IMM01 and tislelizumab in May 2022 for the treatment of various advanced solid tumors that failed to respond to or relapsed from the standard of care such as PD-1/PD-L1 inhibitors, including among others, NSCLC, SCLC, HNSCC and CRC, and initiated the Phase II trial in December 2022. Furthermore, we are collaborating with Sunshine Guojian to develop a combination therapy of inetetamab and IMM01 for HER2-positive solid tumors in mainland China, for which Sunshine Guojian will drive and fund the clinical development. We are also actively conducting numerous preclinical studies to evaluate the combination potential of IMM01 with other drugs.

Clinical data available thus far showed favorable safety and promising preliminary efficacy of IMM01 as a single agent. According to the safety data from the Phase I dose-escalation study, IMM01 was well tolerated and safe up to the RP2D of 2.0 mg/kg in patients and demonstrated no hemagglutination. As of February 10, 2023, neither hemagglutination nor hemolytic anemia had been observed in its Phase II clinical trial. In terms of efficacy, as of December 14, 2022, among 27 evaluable patients in the Phase I monotherapy clinical study, two patients reached CR (2 CRs), one reached PR (1 PR), and 13 reached stable disease (13 SDs) (including six cases with

substantial tumor shrinkage observed). According to Frost & Sullivan, among numerous drug developers of CD47-targeted molecules globally, we are one of the only two companies to have observed CR in monotherapy clinical trials with a well tolerated safety profile.

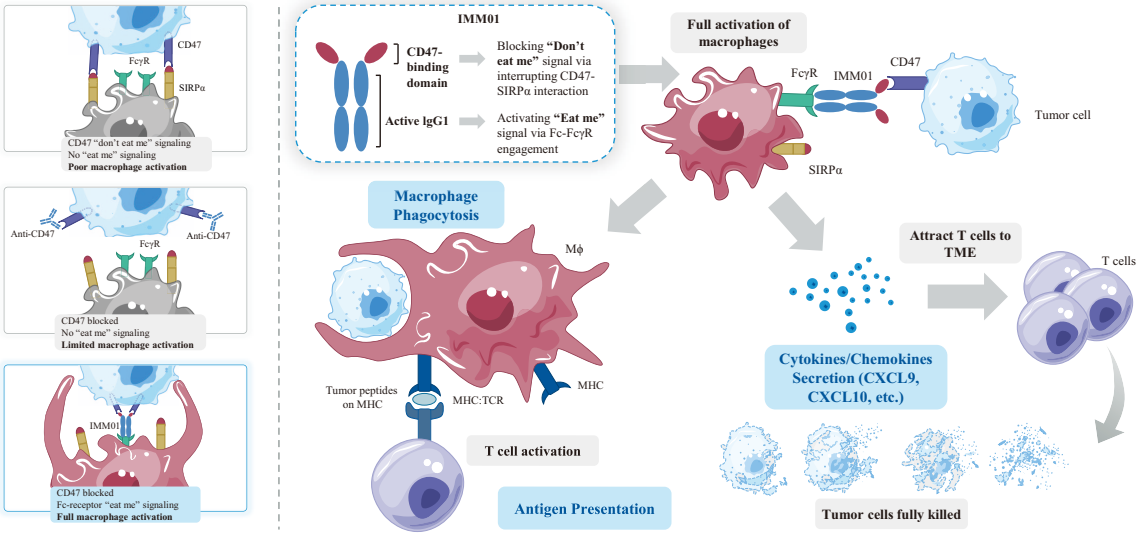
Mechanism of Action

Macrophages are a type of white blood cells that can phagocytose antigens. They are part of the innate immune system that acts as the first line of defense to protect the body from infection. Macrophages are widely distributed in numerous solid tumors with significantly higher tissue penetration than T cells. CD47 is a critical macrophage checkpoint that can transmit a "don't eat me" signal to macrophages by interacting with SIRPα. Thus, the overexpression of CD47 enables tumor cells to evade immune responses via the CD47-SIRPα immune inhibitory pathway.

IMM01 is a SIRPα-Fc fusion protein that consists of an engineered extracellular CD47-binding domain of human SIRPα, linked to the Fc region of human IgG1. IMM01 is designed to fully activate macrophages through a dual mechanism. On one hand, the engineered CD47-binding domain of IMM01 can selectively bind to CD47 overexpressed on tumor cells and prevent CD47 from delivering the "don't eat me" signal to macrophages. This specifically modified CD47-binding domain can avoid binding to human RBCs *in vitro*. The binding specificity of IMM01 minimizes its blood toxicity and allows for a favorable safety with the ability to potently activate macrophages. On the other hand, the engineered human IgG1 Fc of IMM01 can engage Fc receptors on macrophages to deliver a strong "eat me" signal that is essential to full macrophage activation. In addition to direct tumor killing activities, activated macrophages also release a slew of cytokines and chemokines, such as CXCL9 and CXCL10, that can attract and recruit T cells into the TME. Increased T-cell infiltration in solid tumors can turn the non-immune responsive "cold tumors" to immune-sensitive "hot tumors." The activity of macrophages can further increase antigen presentation to T cells, thus leading to enhanced tumor-specific T-cell response. Additionally, the engineered IgG1 Fc can induce ADCC mediated by NK cells, leading to direct tumor-killing effects. Overall, the activation of macrophages and NK cells and their crosstalk with T cells empower all-around immune responses.

The following diagram illustrates the mechanism of action of IMM01:

Mechanism of Action of IMM01



Definition: MHC refers to major histocompatibility complex.
Source: Frost & Sullivan, Company Data, Literature Review

Market Opportunities and Competition

The current approved immunotherapies primarily target T-cell immune checkpoints, including PD-1/PD-L1, CTLA-4 and LAG-3. However, the overall response rates of these T-cell immune checkpoint inhibitors are limited in many major types of cancer. As summarized in the table below, only about 10% to 25% of patients across almost all major cancer types respond to PD-1/PD-L1 inhibitor monotherapy.

Tumor Response Rates to PD-1/PD-L1 Inhibitor Monotherapy

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

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

Definitions: NSCLC refers to non-small cell lung cancer; SCLC refers to small cell lung cancer; CRC refers to colorectal cancer; GC refers to gastric cancer; HNSCC refers to head and neck squamous cell carcinoma; HCC refers to hepatocellular carcinoma; ESCC refers to esophageal squamous cell carcinoma; BTC refers to biliary tract cancer; RCC refers to renal cell carcinoma; OC refers to ovarian cancer; CC refers to cervical cancer; UC refers to urothelial carcinoma; STS refers to soft-tissue sarcomas; DLBCL refers to diffuse large B-cell lymphoma.

Source: Frost & Sullivan

In recent years, mounting research highlights the potential to deploy innate immunity-targeted strategies to overcome the limitations of using only T-cell immunotherapies in cancer treatment. According to Frost & Sullivan, there is significant market potential worldwide and in China for CD47/SIRPα-targeted therapies. The global market size of CD47/SIRPα-targeted therapies is expected to reach US$13.1 billion and US$33.7 billion in 2030 and 2035, respectively. The prospect promised by this new therapy was also validated by several multi-billion dollar take-over transactions of CD47 focused biotechnology companies as well as licensing deals for CD47-targeted agents backed by leading multinational pharmaceutical companies, including Gilead, Pfizer and AbbVie.

We believe CD47-targeted agents in combination with other agents have significant opportunities to fulfill the unmet medical needs of numerous hematologic malignancies and solid tumors in China and worldwide. We are developing the combination of IMM01 and azacitidine for the first-line treatment of HR MDS, unfit AML and CMML. The combination of IMM01 and tislelizumab is being developed for the second-line treatment of NSCLC, SCLC, HNSCC, CRC and other solid tumors, as well as the third-line treatment of cHL. We will also consider moving our current treatment into front-line settings in a stepwise manner at a later stage when promising clinical efficacy has been validated. In addition, the combination of IMM01 and inetetamab is intended to be used in the second- and third-line treatments of HER2 positive solid tumors. According to Frost & Sullivan, the relapse rates of the first-line treatments of advanced NSCLC, SCLC, HNSCC and CRC are approximately 75%, 100%, 50% and 80%, and the overall relapse rate post second-line treatment for advanced cHL is roughly 10%. For patients with advanced HER2 positive solid tumors, almost all of them are expected to relapse after the first-line treatment and proceed to second- and third-line of treatments. Please refer to "Industry Overview — Selected Indications Analysis" for more details on the incidence and prevalence of indications targeted by IMM01 in China and oversea markets.

Hematologic malignancies

CD47 overexpression is widely observed in many hematologic malignancies, including lymphoma, MDS/CMML and AML. It is found to correlate with poor prognosis and reduced overall survival (OS). Over 80% of lymphomas are classified as NHL. The current medical treatments for NHL generally include chemotherapy and targeted therapy. However, approximately 50% of NHL patients will eventually progress to R/R NHL after first-line treatments. Due to the drug resistance and side effects associated with standard treatments, patients with R/R diseases are left with very limited effective treatment options. For classical Hodgkin lymphoma (cHL), PD-1/PD-L1 inhibitors alone or in combination with chemotherapy are mainly recommended. Despite the fact that PD-1/PD-L1 inhibitors have shown good efficacy in R/R cHL, patients who had relapsed or progressed after PD-1/PD-L1 inhibitors are left with very limited treatment options. Additionally, the first-line treatments for MDS/CMML and AML are generally limited to chemotherapy, presenting unmet needs of most patients for highly specific treatment. Please refer to "Industry Overview" for more details on the incidence, treatment paradigm and unmet medical needs for NHL, MDS/CMML and AML. In recent years, the therapeutic potential of CD47-targeted agents in lymphoma, MDS/CMML and AML has been validated by accumulating clinical data. For example, in clinical trials, Gilead's magrolimab in combination with azacitidine has delivered an ORR of 75% and 73% in the first-line treatment of MDS and AML, respectively. However, since both azacitidine and CD47 antibodies also induce blood toxicity, the combination use of these two agents could induce further exacerbated blood toxicity and ultimately lead to serious safety issues. Given its advantages in single-agent efficacy and safety compared to CD47 antibodies, IMM01, when used in combination with azacitidine, has a high potential to fulfill the unmet medical needs of patients with MDS/CMML and AML.

Solid tumors

So far, PD-1/PD-L1 inhibitors have been approved for the treatment of a broad range of cancers worldwide. However, their monotherapy only produces meaningful responses in 10% to 25% patients across almost all major cancer types. The response rates could be particularly low in "cold tumors" with insufficient T-cell infiltration. Moreover, survival benefits of current combination therapies based on PD-1/PD-L1 inhibitors are also limited in many cancer types. For extensive-stage SCLC, metastatic HNSCC and metastatic ESCC, PD-1/PD-L1 inhibitor-based combination therapies only provide an approximately two- to three-month improvement in median overall survival (mOS) compared with chemotherapy alone. Relatively short median progression-free survival (mPFS) is observed with the treatment of PD-1/PD-L1 inhibitor-based combinations in many solid tumors, including metastatic GC (7.7 months), metastatic CRC (8.9 to 10.6 months in the first-line treatment), HCC (4 months), ESCC (6.3 months) and NSCLC (6.4 to 8.8 months in the first-line treatment). Given these limitations of PD-1/PD-L1 inhibitors, there is a clear need for other effective treatment options to improve treatment outcomes for patients. Please refer to "Industry Overview" for more details on the incidence, treatment paradigm and unmet medical needs for the solid tumors.

Our research suggests that IMM01 acts synergistically with PD-1/PD-L1 inhibitors and enhance their activity in solid tumors. Macrophages are widely distributed in a broad range of tumor types, accounting for 20% to 50% of cells in respective tumor tissues, including NSCLC, SCLC, GC, BC, HNSCC, HCC, ESCC, BTC, and OC. As described in the "— Mechanism of Action," IMM01 can fully activate macrophages to promote the T cell immune response, which could potentially enhance the response rates of solid tumors to PD-1/PD-L1 treatments. Thus, combining IMM01 with tislelizumab may be an effective therapeutic approach for treating cold tumors with limited sensitivity to PD-1/PD-L1 inhibition. In our Phase Ib trial evaluating the combination of IMM01 and tislelizumab, a heavily pre-treated NSCLC patient with six lines of prior treatment and refractory to PD-1 inhibitors achieved PR after three cycles of treatment with target lesion shrinkage of 40%.

Competitive landscape

As of the Latest Practicable Date, there were no CD47-targeted therapies approved for marketing in China or the rest of the world. IMM01 is the first SIRPα-Fc fusion protein targeting CD47 to enter into clinical stage in China.

Safety issues have been the primary concern regarding CD47-targeted agents, especially CD47 antibodies. For CD47 antibodies, their inevitable binding with human RBCs and platelets (which ubiquitously express CD47) can lead to severe blood toxicity, such as anemia, thrombocytopenia and hemagglutination. A number of clinical-stage CD47 antibodies have shown RBC binding activity, resulting in severe adverse effects. Gilead's magrolimab (CD47 mAb) is a recent example. The FDA temporarily placed a partial clinical suspension on trials evaluating magrolimab in MDS, AML, MM and DLBCL in early 2022 due to an apparent imbalance in investigator-reported SUSAR between study arms observed in trials in early 2022. All of those partial suspensions have been subsequently lifted, as the FDA determined that, following comprehensive review of the safety data from each trial, the clinical sponsor had satisfactorily addressed the deficiencies. The clinical trials of multiple other CD47 antibodies, including Bristol-Myers (Celgene)'s CC-90002 (CD47 mAb) and Surface Oncology's SRF231 (CD47 mAb), have also been suspended or partially suspended due to safety issues. As compared to CD47 antibodies, with an engineered CD47-binding domain, IMM01 does not bind to human RBCs *in vitro*. It also demonstrated a good safety and tolerability in patients in our Phase I/II clinical trial.

To address the safety concerns, almost all CD47 antibodies have resorted to Fc isotypes with weak Fcγ receptors engagements, such as IgG4 and IgG2. Although such design may reduce the risks of inducing macrophage phagocytosis against healthy blood cells, it leads to weakened immune responses against tumor cells, and thus none of the CD47 antibodies showed single-agent CR in clinical trials. Compared to those CD47 antibodies, IMM01 that incorporates an IgG1 Fc demonstrates enhanced immune effector function and can fully activate macrophages as a single agent. In our Phase I trial of IMM01 monotherapy, promising efficacy signals were observed, including two CRs (2 CRs), one PR (1 PR) and 13 SDs (including six cases with substantial tumor shrinkage observed).

According to Frost & Sullivan, among numerous drug developers of CD47-targeted molecules globally, we are one of the only two companies to have observed CR in monotherapy clinical trials with a well tolerated safety profile. The following table summarizes the information of major clinical-stage CD47-targeted molecules globally:

Competitive Landscape of CD47-targeted Drug Candidates

Drug Name	Company	Molecule	Fc isotype	RBC binding	1st in human	Monotherapy CR	Indication	Latest Stage
Hu5F9 (Magrolimab)	Forty Seven (Gilead)	mAb	IgG4	Yes	2014.8	No	AML, MDS, MM, NHL, HNSCC, TNBC, OC, CRC	Ph III (Suspension Lifted by FDA)
TTI-621	Trillium Therapeutics (Pfizer)	SIRPαFc	IgG1	No	2016.1	Yes	AML, MDS, MM, Lymphoma, Leiomyosarcoma, Solid Tumor	Ph II
TTI-622		SIRPαFc	IgG4	No	2018.5	Yes	AML, MM, Lymphoma, OC	Ph II
CC-90002	Celgene (BMS)	mAb	IgG4	Yes	2015.2	No	AML, MDS, MM, NHL, Solid tumor	Ph I (Partial Suspension by the Company)
SRF231	Surface Oncology	mAb	IgG4	Yes	2018.4	No	Advanced Solid Cancers, Hematologic Cancers	Ph I (Suspension by the Company)
ALX-148 (Evorpacept)	ALX Oncology	SIRPαFc	IgG1 Fc(Inert)	Yes	2017.1	No	AML, MDS, NHL, Solid Tumor	Ph II/III
SHR1603	HengRui 恒瑞	mAb	IgG4	Yes	2018.10	No	Advanced Malignancies, Lymphoma	Ph I (Suspension by the Company)
AO-176	Arch Oncology	mAb	IgG2	Minimal	2019.2	No	MM, GC, NSCLC, HNSCC, OC, Prostate Cancer, Endometrial Carcinoma	Ph I/II (Suspension by the Company)
IBI188 (Letaplimab)	Innovent 信达生物	mAb	IgG4	Yes	2018.11	No	AML, MDS, Lymphoma, Solid Tumor	Ph Ib/III (Partial Suspension by the Company)
TJC4 (Lemzoparlimab)	I-Mab 天境生物 /AbbVie	mAb	IgG4	Minimal	2019.5	No	AML, MDS, MM, CD20 Positive Lymphoma, Advanced Solid Tumor	Ph III (Partial Suspension by the Company)
IMM01	ImmuneOnco 宜明昂科	SIRPαFc	IgG1	No	2019.9	Yes	MDS, AML, CMML, HL, NHL, Solid Tumor	Ph II
AK117	Akesobio 康方生物	mAb	IgG4	Minimal	2020.4	No	AML, MDS, Lymphoma, TNBC, HNSCC, NSCLC, SCLC, OC, CRC, HCC	Ph II

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

Source: Frost & Sullivan, CDE, ClinicalTrials, Company Website, Literature Review

Given its good safety and promising single-agent efficacy, IMM01 could be a more favorable combination partner with many other cancer agents as compared to CD47 antibodies. When used in combination with IgG4 Fc antibodies, such as most PD-1 inhibitors, IMM01 with IgG1 Fc can fully activate macrophages to exert more potent antitumor effects than CD47 antibodies with IgG4 Fc.

Competitive Advantages

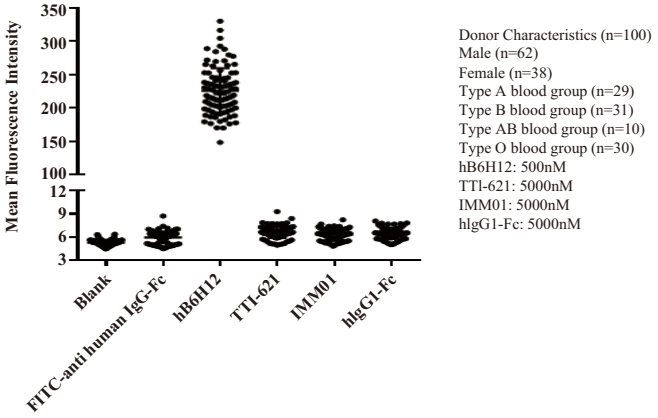
Attributable to its well-designed molecule structure, IMM01 monotherapy has the following competitive strengths:

(1) Favorable safety profile with no observed binding to human RBCs in vitro

Despite the clinical significance of CD47 as a potential backbone innate immune checkpoint, the therapeutic benefits brought by CD47-targeted agents are largely compromised due to their safety issues. The safety issues are mainly resulted from the ubiquitous expression of CD47 in blood cells, such as RBCs and platelets. Thus, treatment with CD47-targeted agents that bind to CD47 on blood cells may greatly reduce the number of circulating RBCs and platelets, leading to severe blood toxicity, such as anemia and thrombocytopenia. Binding with circulating blood cells with high affinity, CD47 antibodies will confront with "antigenic sink" (rapid drug clearance), thereby preventing the agents from reaching tumor tissues. Additionally, CD47 antibodies with IgG4 Fc cannot fully activate macrophages, thus requiring much higher drug dosing, inducing greater toxicity and inflicting a heavier economic burden on patients.

We specifically modified the CD47-binding domain of IMM01 to overcome these limitations. Since CD47 expressed on human RBCs and tumor cells has different glycosylation profiles, the engineered CD47-binding domain selectively binds to CD47 on tumor cells, while not binding to human RBCs *in vitro* without compromising its ability to potently activate macrophages. We assessed the binding affinity of IMM01 with normal cells and tumor cells in various *in vitro* binding assays and cross-reactivity tests. Results of our *in vitro* studies showed that IMM01 generally has much stronger binding affinity for tumor cells than normal tissue cells, and it does not bind with human RBCs *in vitro*. Further, deglycosylation modification to CD47-binding domain also mitigates the immunogenicity of IMM01 and improves its PK profile. The chart below illustrates that IMM01 does not bind to human RBCs *in vitro* as tested in human blood samples obtained from 100 donors with different blood types.

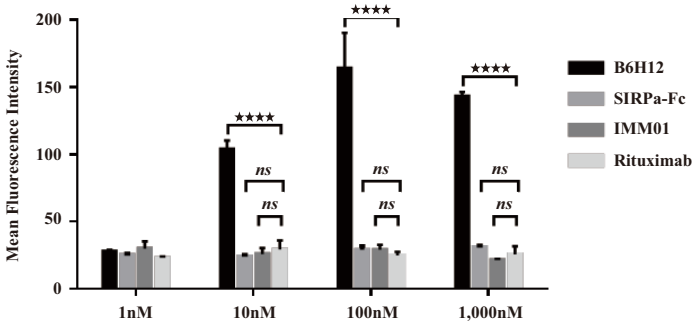
Human RBC Binding Analysis of IMM01



Note: B6H12 is a CD47-based antibody that serves as the control.
Source: Company Data

Furthermore, our preclinical studies revealed that IMM01 did not induce phagocytosis against human RBCs *in vitro* up to 1000 nM, as shown in the following bar chart:

Phagocytosis Against Human RBC



Note: B6H12 is a CD47-based antibody that serves as the control.
Source: Company Data

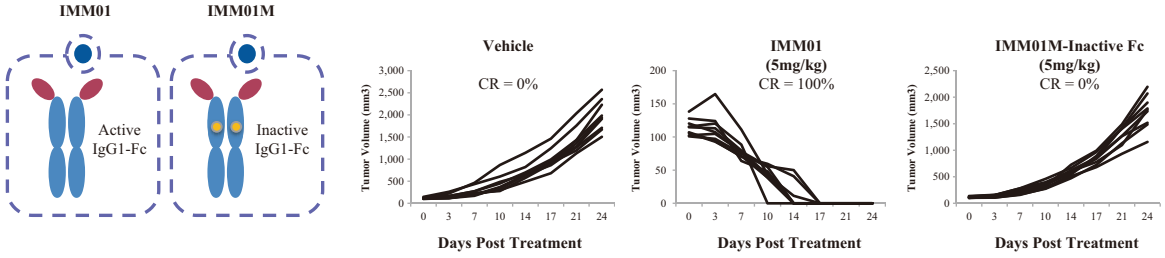
By selectively binding to tumor cells, IMM01 triggers tumor cell-specific phagocytosis by macrophages. In addition, the dual mechanisms of IMM01 enables it to exert antitumor activity at a relatively low dosage. IMM01 monotherapy and its combination therapies can achieve a promising efficacy profile at a lower dose of 2.0 mg/kg, as compared to the typical dose of 30.0 to 45.0 mg/kg required for CD47 antibodies.

The safety profile of IMM01 was further demonstrated in our clinical trials. Our Phase I dose-escalation trial demonstrated that IMM01 monotherapy was well tolerated and safe up to 2.0 mg/kg in patients with R/R lymphoma and showed no hemagglutination. As of February 10, 2023, neither hemagglutination nor hemolytic anemia had been observed in its Phase II clinical trial.

(2) Potent antitumor activity and encouraging preliminary clinical efficacy

As IMM01 shows no *in vitro* binding to human RBCs, it can adopt an IgG1 Fc that assists in full macrophage activation via Fc-FcγR engagement without serious safety concerns. The IgG1 Fc in IMM01 can fully activate macrophages, leading to enhanced ADCP and ADCC activity and strong immune responses. As demonstrated in the charts below, our *in vivo* efficacy studies showed that an active IgG1 Fc is imperative for the stimulation of antitumor activity, as IMM01M with an engineered mutant inactive IgG1 Fc has exhibited very limited efficacy as compared to IMM01.

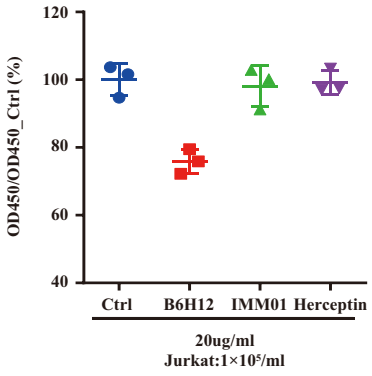
***In Vivo* Efficacy of IMM01 is Dependent on Effective Fc Function (HL-60 xenograft model)**



Note: IMM01M has an engineered mutant inactive IgG1 Fc.
Source: Company Data

In addition, we carefully designed the molecule of IMM01 to avoid triggering T-cell apoptosis. Research revealed that CD47 ligation by certain CD47 antibodies may induce T-cell apoptosis, resulting in T-cell toxicities and compromised T-cell immune response. As illustrated in the chart below, as compared to B6H12, a CD47 antibody, IMM01 does not induce T-cell apoptosis.

IMM01 Does Not Induce T cell Apoptosis

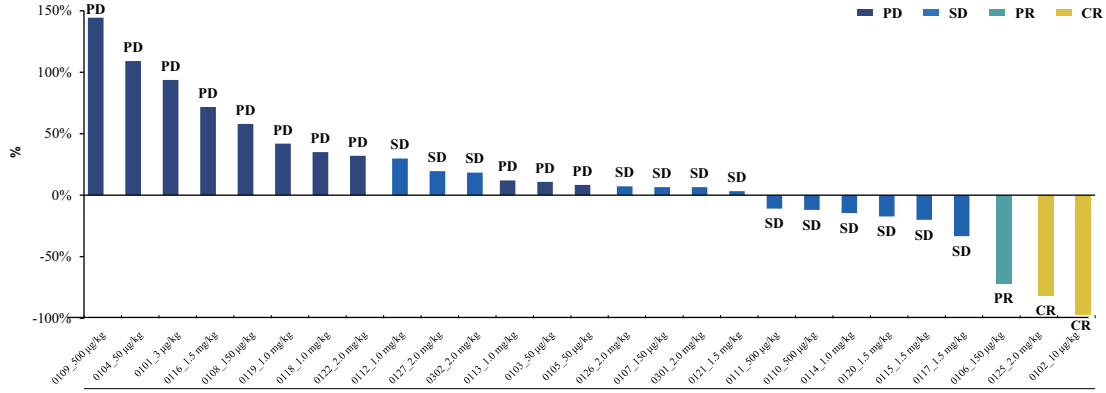


Note: (1) B6H12 is a CD47-based antibody that serves as the control. OD450/OD450 control measures the proportion of live T cells in the respective samples; (2) Three mice per group were used in this study; (3) The colors of graphics represent different groups using different drugs, drug candidates or molecules.

Source: Company Data

The advantages of molecule design of IMM01 has also translated into clinical benefits. In the Phase I dose-escalation study, IMM01 has demonstrated promising single-agent antitumor activities. Among 27 evaluable patients in the Phase I monotherapy clinical study, two CRs (2 CRs), one PR (1 PR) and 13 SDs (including six cases with substantial tumor shrinkage observed) were confirmed. Among the six patients at RP2D dose of 2.0 mg/kg, one reached complete response (1 CR), and four reached stable disease (4 SDs), with a DCR of 83% in these previously heavily pre-treated R/R lymphoma patients. Notably, according to Frost & Sullivan, among numerous drug developers of CD47-targeted molecules globally, we are one of the only two companies to have observed CR in monotherapy clinical trials with a well tolerated safety profile.

Best Overall Changes in Size of Target Tumor Lesions



Note: The colors of bars represent the best overall changes in size of target tumor lesions.

Source: Company Data, as of December 14, 2022

(3) Combination potential with a wide range of cancer therapeutics

The critical role of macrophages in the stimulation of innate immunity and the enhancement of T-cell response provide robust scientific rationale for the development of IMM01 in combination with other immune-mediated agents, given IMM01's dual mechanisms for full macrophage activation. Consistent with the scientific rationale, we have observed further enhanced antitumor activity when combining IMM01 with T-cell immunotherapies, including PD-1/PD-L1 inhibitors, other immunotherapies and targeted therapies in our preclinical studies. With encouraging efficacy and favorable safety in monotherapy clinical trials and robust preclinical data of its combination studies, IMM01 is expected to achieve strong synergistic effects used in combination with other cancer agents.

Combination with azacitidine

The combination of CD47 antibody and azacitidine has been well tested and validated in clinical trials. According to publicly disclosed clinical data, the combination of CD47 antibody and azacitidine was efficacious in treating MDS and AML patients. For example, in clinical trials, Gilead's magrolimab in combination with azacitidine has delivered an ORR of 75% and 73% in the first-line treatment of MDS and AML, respectively. However, when combining with azacitidine which itself can cause blood toxicity such as anemia and thrombocytopenia, CD47 antibodies used at a high dose level may induce combined and exacerbated severe blood toxicity and adverse events, as exemplified by the partial suspension of the trials evaluating magrolimab combined with azacitidine due to an imbalance in investigator-reported SUSAR between study arms, although those partial suspensions have been subsequently lifted, as the FDA determined that, following comprehensive review of the safety data from each trial, the clinical sponsor had satisfactorily addressed the deficiencies.

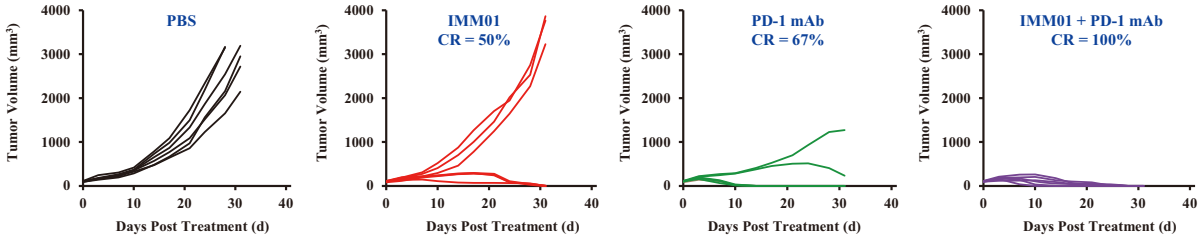
IMM01 has demonstrated a favorable safety profile when used in combination with azacitidine in our clinical trial. Upon completion of the Phase Ib trial, we initiated a Phase II trial to evaluate the safety and efficacy of IMM01 in combination with azacitidine mainly for the first-line treatment of HR MDS, unfit AML and CMML in June 2022. Interim data as of February 10, 2023 from the Phase Ib/II trial has demonstrated a favorable safety and promising efficacy profile. Neither DLT nor hemagglutination was observed among all 12 patients in the combination treatment at all three dose levels of 1.0 mg/kg, 1.5 mg/kg, and 2.0 mg/kg in our Phase Ib trial. This combination therapy has also shown promising efficacy signal at a low dose level (2.0 mg/kg), which is much less than the typical dose of 30.0 to 45.0 mg/kg required for CD47 antibodies. In our Phase II trial, as of February 10, 2023, (i) among the eight evaluable patients with 1L CMML, two reached CR (2 CRs), six reached mCR (6 mCRs), and one reached HI (1 HI, which also achieved mCR), resulting in an ORR of 100%, and (ii) among the 16 evaluable HR MDS patients who have received at least three cycles of treatment, three achieved CR (3 CRs), nine achieved mCR (9 mCRs), and seven achieved HI (7 HIs, among which 4 also achieved mCR), resulting in an ORR of 93.8%. With a much lower required dose, IMM01 can further reduce potential safety risks as observed with other CD47 antibodies.

Combination with tislelizumab

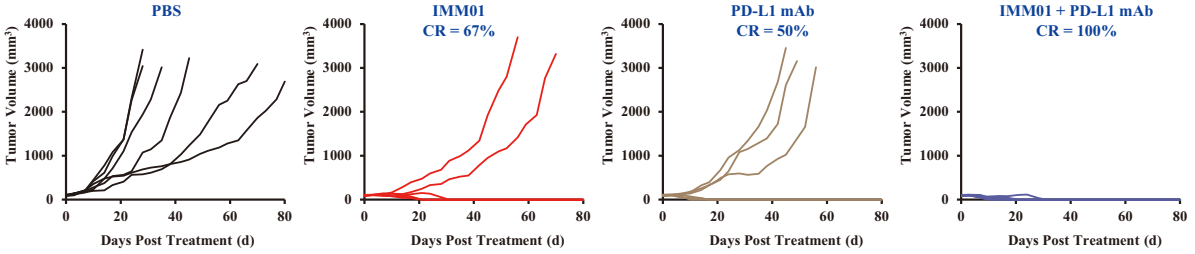
Despite their huge commercial success, PD-1/PD-L1 inhibitors can only reach response rates between 10% to 25% across almost all major cancer types as monotherapy, which are particularly low when targeting "cold tumors" that lack T-cell infiltration. While adding additional T-cell immune checkpoint inhibitors, such as LAG3 antibody, to a PD-1/PD-L1 antibody has shown impressive efficacy in certain cancer types, the benefits of such combinations are still expected to limit to "hot tumors" with substantial T-cell infiltration.

The IgG1 Fc design of IMM01 provides it with a unique advantage for the development of combination therapy with a PD-1 antibody, as compared to CD47 antibodies with IgG4 Fc. As most PD-1 antibodies also consist of IgG4 Fc, those IgG4 Fc-based CD47 antibodies cannot fully activate macrophages when combined with PD-1 antibodies due to the lack of the additionally required "eat me" signal activation. In contrast, IMM01 with IgG1 Fc can fully activate macrophages as a single agent and achieve synergistic effects in combination with PD-1 antibodies. Fully activated macrophages can secrete cytokines and chemokines that recruit T cells into the TME to turn "cold tumors" into "hot tumors," and further enhance T-cell response through antigen presentation, thus maximizing the benefits of the combination therapy. Additionally, IMM01 significantly inhibits the production of IL-8 which acts as one of the key mediators of resistance to PD-1/PD-L1 inhibitors. As shown in the charts below, the combination of IMM01 with either a PD-1 or PD-L1 antibody exhibited encouraging synergistic effects in our *in vivo* solid tumor efficacy models.

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



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



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

In our Phase Ib trial for the combination of IMM01 and tislelizumab, a heavily pre-treated NSCLC patient with six lines of prior treatment and refractory to PD-1 inhibitors achieved PR after three cycles of treatment with target lesion shrinkage of 40%.

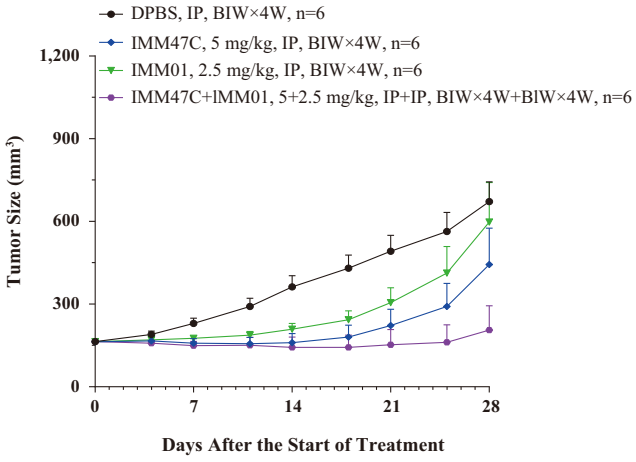
Combination with IMM47

IMM47 is a CD24-targeted humanized monoclonal antibody developed by us and has global first-in-class potential. CD24 is widely expressed in many tumor types and is found to be highly correlated with poor prognosis. Accumulating research has demonstrated that the inhibition on CD24 can prevent tumor cells from delivering inhibitory signals to macrophages, NK cells and T cells. Our preclinical studies have shown a promising efficacy profile of IMM47. For more details on the mechanism of action and preclinical results of IMM47, please refer to "— Our Innate Immune Checkpoint-targeted Drug Candidates — IMM47 (CD24 mAb)." Given IMM01 and

IMM47 both work to stimulate and activate overall innate and adaptive immune systems, the combination of these two candidates is expected to act synergistically to induce more profound antitumor immune responses.

The contemplated synergy has been further demonstrated in our preclinical studies. As shown in the diagram below, the combination treatment of IMM01 with IMM47C (previous generation chimeric version of IMM47) strongly suppressed the tumor growth in a xenograft model of TNBC, and largely outperformed either of the single agents alone at the same dose level:

IMM01 + IMM47C: Triple-negative Breast Cancer (MCF-7) Xenograft Mouse Model



Note: IMM47 revealed highly similar *in vitro* efficacy as IMM47C (previous generation chimeric version of IMM47), and was eventually selected for further development.
Source: Company Data

Combination with inetetamab

Inetetamab, independently developed by Sunshine Guojian, is a HER2 monoclonal antibody which was approved by the NMPA in June 2020 for the treatment of HER2-positive metastatic BC in combination with chemotherapy. We are collaborating with Sunshine Guojian to develop a combination therapy of inetetamab and IMM01 for HER2-positive solid tumors in mainland China (excluding Hong Kong, Macau and Taiwan). We have obtained an IND approval for a Phase Ib/II clinical trial in HER2-positive solid tumors from the NMPA in August 2021. For details, please refer to the paragraph headed "— Collaboration Agreement — Collaboration with Sunshine Guojian."

Combination with other drugs

The combination of IMM01 with other immunotherapies and targeted therapies have all seen promising efficacy in our preclinical studies. We may strategically further develop IMM01-based combination therapies on our own or with collaboration partners. We see great potential to target a wide range of tumor indications with IMM01's combination strategy.

Summary of Clinical Trial Results

IMM01 Monotherapy

We initiated a Phase I/II study of IMM01 monotherapy in September 2019, with Phase I dose-escalation study in R/R lymphoma completed in January 2022. Leveraging the safety and efficacy data and RP2D obtained from the Phase I trial, we obtained the IND approvals for clinical trials evaluating IMM01 and each of azacitidine, tislelizumab, inetetamab, and bortezomib/dexamethasonum. We commenced the Phase II cohort-expansion study in October 2021. Based on increasing data collected from our ongoing clinical trials for IMM01 monotherapy, combination therapies as well as CD47-based bispecific molecules, we continue to adaptively adjust the clinical development strategy for IMM01 in the context of development planning for our entire CD47-based product portfolio. Considering the much enhanced efficacy data observed in IMM01's combination trial targeting MDS and AML, as well as promising efficacy data observed in the clinical trial for IMM0306 (CD47×CD20) in R/R B-NHL, we plan to prioritize our resources on the clinical development of IMM01-based combination therapies as well as CD47-based bispecific assets in order to achieve optimal resource allocation.

Trial Design. The primary objectives of the Phase I monotherapy study were to preliminarily assess safety, tolerability and PK characteristics, and determine the MTD (if any), recommended dose for expansion (RDE) and RP2D of IMM01. Subjects with R/R lymphoma received IMM01 across eight cohorts at 3 μg/kg, 10 μg/kg, 50 μg/kg, 150 μg/kg, 500 μg/kg, 1.0 mg/kg, 1.5 mg/kg, and 2.0 mg/kg, respectively. Dose escalation was performed in the routine accelerated titration design for the 3 μg/kg and 10 μg/kg cohorts, and standard "3+3" design for the 50 μg/kg, 150 μg/kg, 500 μg/kg, 1.0 mg/kg, 1.5 mg/kg, and 2.0 mg/kg cohorts. Each cycle contains four weeks of once-weekly dosing followed by a week's rest after cycle one. We have enrolled a total of 29 subjects in this Phase I study. The primary endpoints for this Phase I study are adverse events, DLT, MTD and RP2D. The secondary endpoints include PK profile, immunogenicity and preliminary efficacy, including ORR, DCR, duration of response (DoR), PFS, and OS. The Phase II cohort-expansion study is designed to further evaluate the safety, PK profile, preliminary efficacy and immunogenicity of IMM01 monotherapy for the treatment of various hematologic malignancies. RDE dosing level is 1.5 mg/kg to 2.0 mg/kg, and RP2D is eventually set at 2.0 mg/kg.

Trial Status. We have completed a Phase I dose-escalation study of IMM01 monotherapy for the treatment of R/R lymphoma in January 2022 with a total of 29 patients enrolled. Phase II cohort-expansion clinical trial of IMM01 monotherapy was initiated in October 2021 and 29 patients with R/R lymphoma have been enrolled. We have discontinued enrolling patients for this Phase II trial since October 2022. Patients with treatment benefit will remain on the trial until their diseases further progress.

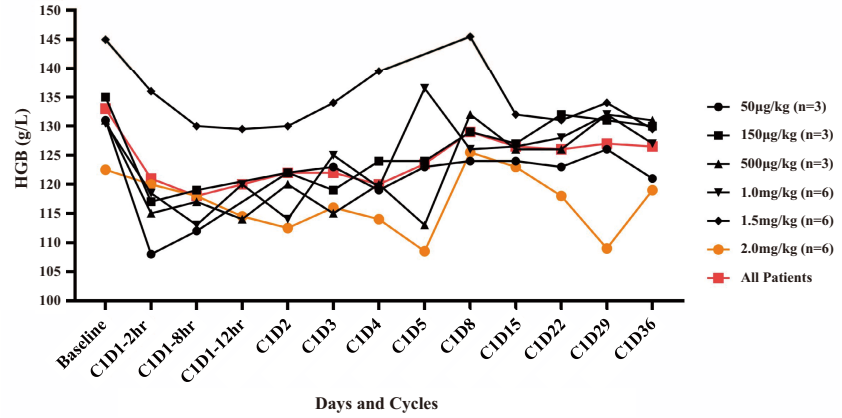
Safety Results. As of August 30, 2022, data obtained from the Phase I study has demonstrated that IMM01 monotherapy was well tolerated and safe up to 2.0 mg/kg. RDE of 1.5 to 2.0 mg/kg and RP2D of 2.0 mg/kg have been determined and used in the Phase II cohort-expansion study. According to the safety data from the Phase I study as of August 30, 2022, only one subject with DLT was observed at 1.5 mg/kg, and MTD was not reached up to 2.0 mg/kg. As illustrated by the following table, the majority of TRAEs observed are Grade 1 and 2. Grade 3 or above TRAEs of IMM01 mainly included leukopenia, thrombocytopenia, anemia and neutropenia, with the highest rate of occurrence at 14% (four out of 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 was generally safe and well tolerated in 29 patients. (3) The majority of TRAEs were grade 1 or 2. (4) Grade 3 and above TRAEs mainly included leukopenia, thrombocytopenia, anemia and 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. As the diagram below illustrates, 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. As of February 10, 2023, neither hemagglutination nor hemolytic anemia had been observed in its Phase II clinical trial.

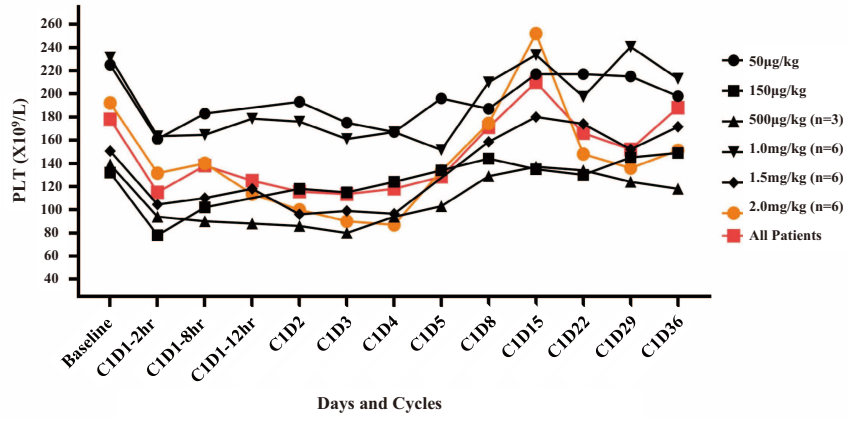
Hemoglobin Changes Following Single-dose Administration in Cycle One by Cohort



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

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

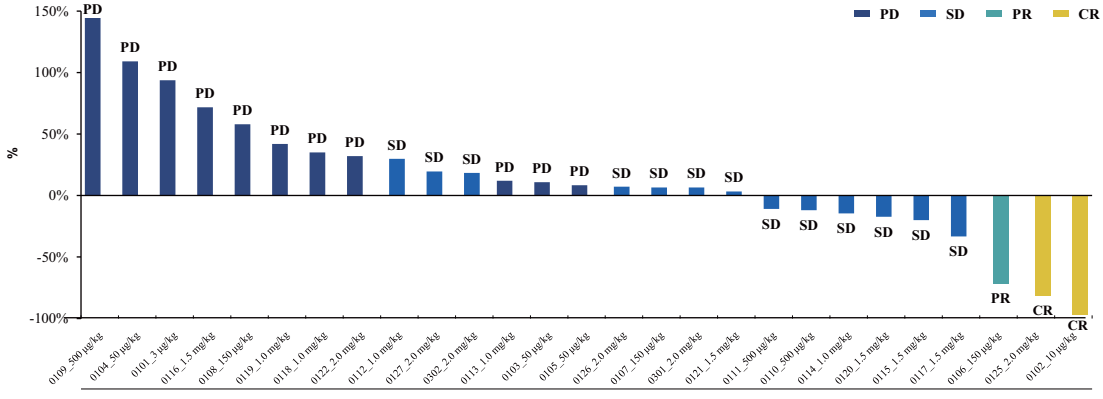
Platelet Changes Following Single-dose Administration in Cycle One by Cohort



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

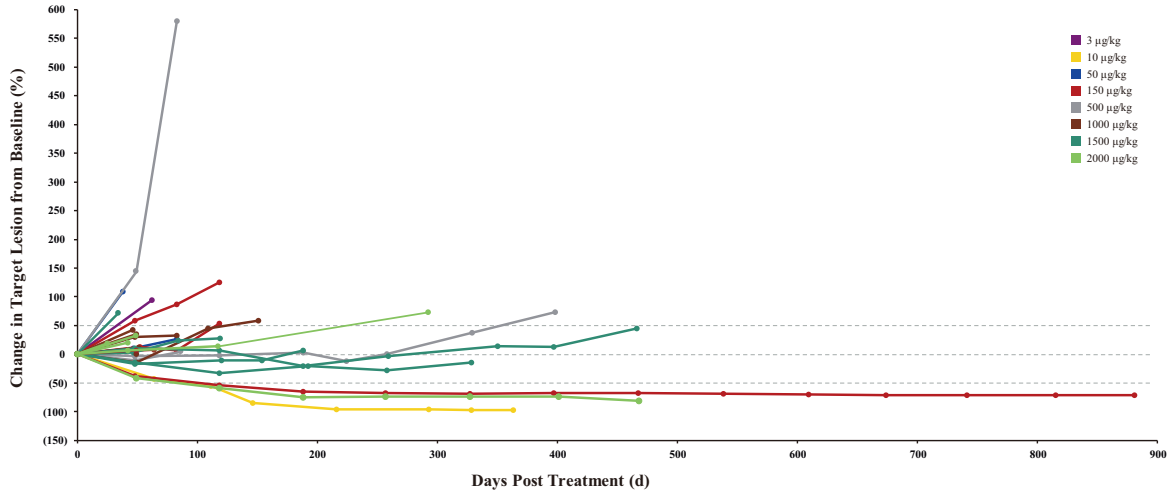
Efficacy Results. As of December 14, 2022, the data obtained from the Phase I dose-escalation study showed a favorable PK/PD profile and preliminary antitumor activity of IMM01 monotherapy: among 27 evaluable patients receiving 0.003 mg/kg to 2.0 mg/kg IMM01 in the Phase I study, two patients reached 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). Among the six patients at RP2D dose of 2.0 mg/kg in this monotherapy clinical trial, one reached complete response (1 CR), and four reached stable disease (4 SDs), with a DCR of 83% in these previously heavily pre-treated R/R lymphoma patients. CR observed in one of the evaluable patients lasted for 4.9 months before it turned into a progressive disease (PD) because of new lesions, and this patient was under continued treatment for another 2.5 months subsequently. Another patient achieved CR after 14 cycles of treatment. Treatments are still ongoing for those benefited patients. The diagrams below illustrate the best overall changes in size of target tumor lesions and duration of response in patients treated with IMM01 monotherapy.

Best Overall Changes in Size of Target Tumor Lesions



Note: The colors of bars represent the best overall changes in size of target tumor lesions.
Source: Company Data, as of December 14, 2022

Duration of Response in Patients Treated with IMM01 Monotherapy



Source: Company Data, as of December 14, 2022

Conclusion. IMM01 monotherapy has exhibited a favorable safety profile, and its preliminary efficacy results have demonstrated encouraging antitumor activities in R/R lymphoma. According to Frost & Sullivan, among numerous drug developers of CD47-targeted molecules globally, we are one of the only two companies to have observed CR in monotherapy clinical trials with a well tolerated safety profile. The encouraging safety and efficacy data of the Phase I trial lays a solid foundation to support the further development of IMM01 in combination therapies as well as the development of CD47-based bispecific molecules.

IMM01 in combination with azacitidine

We initiated a Phase Ib/II study of IMM01 and azacitidine in January 2022, with the Phase Ib trial targeting R/R MDS and R/R AML completed in June 2022. We commenced the Phase II clinical trial mainly for the first-line treatment of HR MDS, unfit AML and CMML in June 2022.

Trial Design. The primary objectives of the Phase Ib study of the combination of IMM01 and azacitidine were to assess its safety and tolerability for the treatment of R/R MDS and R/R AML, and determine the MTD (if any) and RP2D. Subjects received IMM01 and dose of azacitidine across three cohorts at 1.0 mg/kg, 1.5 mg/kg and 2.0 mg/kg of IMM01 and fixed dose of azacitidine (75 mg/m^2/day), respectively. Dose escalation was performed in the standard "3+3" design for these cohorts. Each cycle contains four weeks of once-weekly dosing of IMM01 and injection of azacitidine for seven consecutive days. We have enrolled a total of 12 subjects in this Phase Ib study. The primary endpoints for the Phase Ib study of IMM01 and azacitidine include adverse events, DLT, MTD and RP2D. The secondary endpoints include PK profile and preliminary efficacy, including CR, PR, SD and PD.

The ongoing Phase II cohort-expansion study is designed to further evaluate the safety and efficacy of IMM01 and azacitidine mainly for the first-line treatment of HR MDS, unfit AML and CMML. In the Phase II trial, each cycle contains four weeks of once-weekly dosing of IMM01 and injection of azacitidine for seven consecutive days. The primary endpoints for this Phase II trial include adverse events. The secondary endpoints include PK profile and preliminary efficacy.

Trial Status. We have completed the Phase Ib trial of IMM01 and azacitidine for the treatment of R/R MDS and R/R AML with a total of 12 patients enrolled. Phase II trial of IMM01 in combination of azacitidine was initiated in June 2022.

Safety Results. Neither DLT nor hemagglutination was observed among all 12 patients in the combination treatment at all three dose levels of 1.0 mg/kg, 1.5 mg/kg, and 2.0 mg/kg in the Phase Ib trial. As of February 10, 2023, among MDS patients, neither patient discontinuation due to TRAE nor Grade 3 or higher hemolysis was observed in the Phase II trial. No Grade 3 or higher hemolysis was observed among patients with CMML or AML in the Phase II trial.

Efficacy Results. In the Phase II trial, as of February 10, 2023, among the eight evaluable patients with 1L CMML, two reached CR (2 CRs), six reached mCR (6 mCRs), and one reached HI (1 HI, which also achieved mCR), resulting in an ORR of 100% and a CR rate of 25% after one to five cycles of treatment.

Best Overall Response	1L CMML (N=8)
ORR	8 (100%)
CR	2 (25.0%)
mCR+HI	1 (12.5%)
mCR alone	5 (62.5%)

Notes: (1) The clinical data is as of February 10, 2023. (2) ORR (CR+mCR+HI) refers to overall response rate; CR refers to complete response; mCR refers to marrow complete response; HI refers to hematologic improvement.
Source: Company Data

Among the 16 evaluable HR MDS patients who have received at least three cycles of treatment, three achieved CR (3 CRs), nine achieved mCR (9 mCRs), and seven achieved HI (7 HIs, among which 4 also achieved mCR), resulting in an ORR of 93.8%. Most patients received only one or two cycles of treatment as of February 10, 2023, and their treatment are still ongoing.

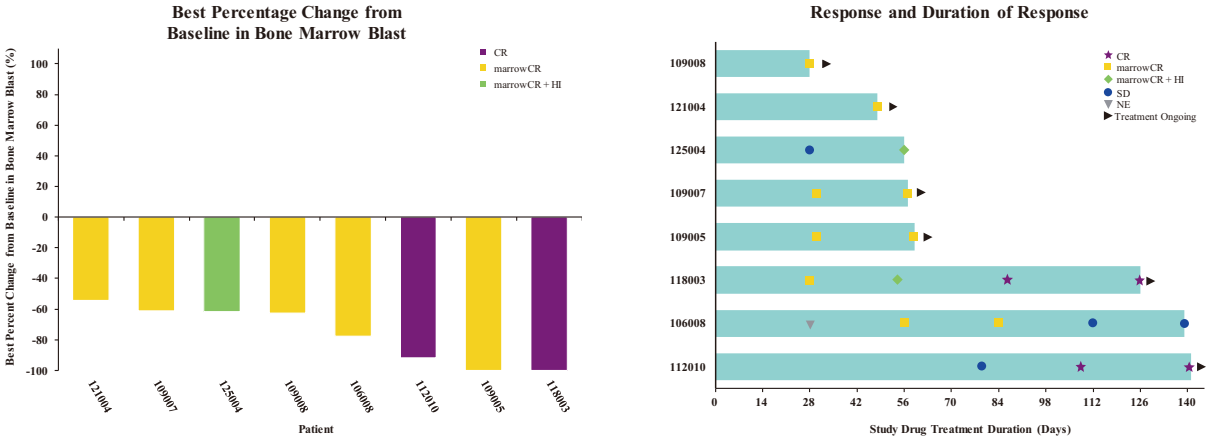
Best Overall Response	Treatment Cycle Since First Dose (ES N=35)	
	≥ 3 cycles (N=16)	≥ 4 cycles (N=13)
ORR	15 (93.8%)	12 (92.3%)
CR	3 (18.8%)	3 (23.1%)
mCR+HI	4 (25.0%)	4 (30.8%)
mCR alone	5 (31.3%)	3 (23.1%)
HI	3 (18.8%)	2 (15.4%)
SD	1 (6.3%)	1 (7.7%)
SD*	0	0
NE	0	0
PD	0	0

Notes: (1) The clinical data is as of February 10, 2023. (2) ORR refers to overall response rate; CR refers to complete response; mCR refers to marrow complete response; HI refers to hematologic improvement; SD refers stable disease; SD* refers to SD not met for over eight weeks; PD refers to progressive disease; NE refers to not evaluable; (3) ES (evaluable analysis set) is defined as subjects with at least one post-baseline tumor assessment.
Source: Company Data

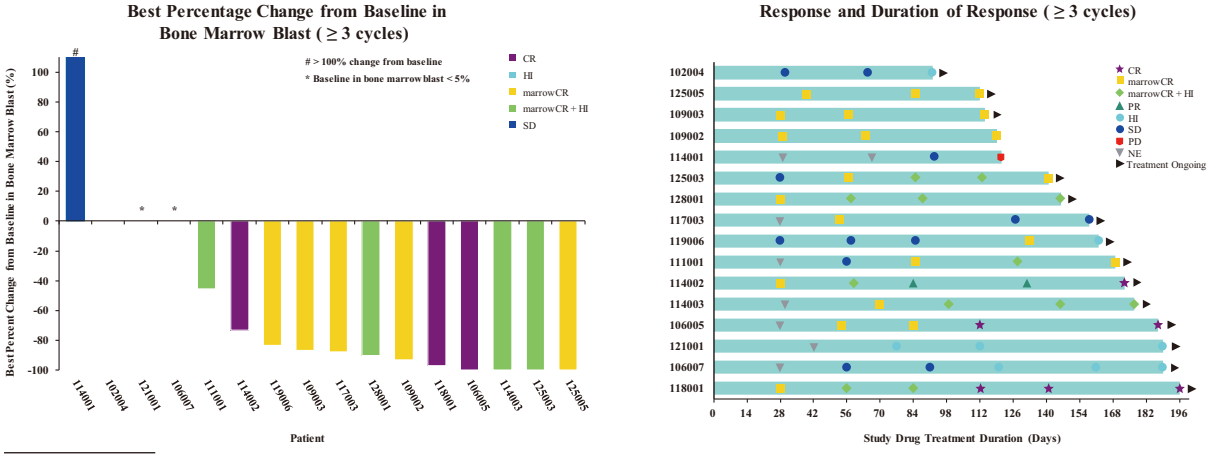
The following diagram illustrates the interim efficacy data of the combination of IMM01 and azacitidine as of February 10, 2023:

Efficacy Data of IMM01 in Combination with Azacitidine

1L CMML



1L MDS



Notes: (1) The clinical data is as of February 10, 2023. (2) ORR refers to overall response rate; CR refers to complete response; MarrowCR refers to marrow complete response; HI refers to hematologic improvement; SD refers stable disease.

Source: Company Data

Conclusion. The clinical data from the Phase Ib/II trial for the combination of IMM01 and azacitidine has demonstrated positive safety and preliminary efficacy profile, and supports continued development of IMM01 in combination with azacitidine.

IMM01 in combination with tislelizumab

We initiated a Phase Ib/II clinical trial to evaluate IMM01 in combination with tislelizumab in May 2022, and initiated the Phase II dose expansion trial in December 2022.

Trial Design. The Phase Ib dose escalation trial is designed to evaluate the safety, MTD/RP2D and preliminary efficacy in advanced solid tumors that failed to respond to or relapsed from the standard of care. Subjects received IMM01 and tislelizumab across three cohorts at 1.0 mg/kg, 1.5 mg/kg and 2.0 mg/kg of IMM01 and fixed dose of tislelizumab (200 mg), respectively. Dose escalation was performed in the standard "3+3" design for these cohorts. Each cycle contains three weeks of once-weekly dosing of IMM01 and injection of tislelizumab for once a cycle. The primary endpoints for the Phase Ib trial of IMM01 and tislelizumab include adverse events, DLTs, MTD and RP2D. The secondary endpoints include PK profile, immunogenicity and preliminary efficacy, including ORR, DoR, PFS, DCR and time to response.

The ongoing Phase II cohort-expansion trial is designed to further evaluate the safety and efficacy of IMM01 and tislelizumab in advanced solid tumors and lymphoma, including NSCLC, SCLC, HNSCC, R/R cHL and others, which failed to respond to or relapsed from the standard of cares including PD-1/PD-L1 inhibitors. In the Phase II trial, each cycle contains three weeks of once-weekly dosing of IMM01 and injection of tislelizumab for once a cycle. The primary endpoint for this Phase II trial is efficacy. The secondary endpoints include safety, tolerability and immunogenicity.

Trial Status. We dosed the first patient for the Phase Ib trial in May 2022. We have enrolled a total of 14 subjects in this Phase Ib trial and completed the enrollment of subjects and observation of DLT for the Phase Ib trial. We determined 2.0 mg/kg as the RP2D of IMM01 in combination with tislelizumab and dosed the first patient for the Phase II trial in December 2022.

Safety Results. As of February 10, 2023, the combination of IMM01 and tislelizumab was shown to be safe and well tolerated at up to 2.0 mg/kg of IMM01.

Efficacy Results. In our Phase Ib trial, a heavily pre-treated NSCLC patient with six lines of prior treatment and refractory to PD-1 inhibitors achieved PR after three cycles of treatment with target lesion shrinkage of 40% and the treatment is still ongoing.

Conclusion. The clinical data from the Phase Ib trial for the combination of IMM01 and tislelizumab has demonstrated positive safety and preliminary efficacy profile, and supports continued development of IMM01 in combination with tislelizumab in the Phase II trial.

Clinical Development Plan

Given the favorable safety and efficacy profile of IMM01 shown in early clinical trials, we plan to further develop IMM01 in combination with azacitidine, tislelizumab and other cancer agents that have the potential to address significant unmet medical needs.

Program	Indications	Clinical trial stage (status)	Trial site	First-patient-in date	(Expected) BLA submission date[1]
IMM01+ azacitidine	MDS, AML, CMML[2]	Phase Ib (completed) Phase II (ongoing)	China	January 2022	Q1 2025[3]
IMM01 + tislelizumab	NSCLC, SCLC, HNSCC, CRC, other solid tumors, cHL[4]	Phase Ib (completed) Phase II (ongoing)	China	May 2022	Q3 2025[5]

Notes: (1) Denotes the date on which we expect to submit the first BLA for each program; (2) We are conducting cohort expansion trials for the first-line treatment of HR MDS, unfit AML and CMML. Particularly, we believe there is possibility that we could seek an accelerated marketing approval through relatively small sample size studies targeting the first-line treatment of CMML, a rare type of disease with highly unmet medical needs; (3) Subject to positive clinical results of the Phase II trial, we plan to commence a pivotal trial in December 2023. We expect to submit the BLA for CMML to the NMPA in the first quarter of 2025, and submit the BLA for MDS/AML in the fourth quarter of 2025; (4) We are evaluating this combination therapy in cHL patients who relapsed or progressed after the treatment of PD-1 inhibitors which may allow us to pursue an accelerated marketing approval leveraging the results of relatively small sample size studies; (5) We expect to submit the BLA for cHL to the NMPA in the third quarter of 2025, and submit the BLA for solid tumors by the end of 2025.

We are currently conducting all of the clinical trials for IMM01 in combination therapies in China, and will actively explore opportunities to obtain accelerated marketing approvals leveraging the results of relatively small sample size studies. A conditional marketing approval achieved through single-arm study design will typically have conditions that require the drug developer to obtain and report additional clinical data after the commercial launch of the approved drug to further confirm its efficacy and safety. The NMPA will grant a full marketing approval if the additional clinical data fulfills the requirements for a normal marketing approval. If our IMM01 in combination therapies is conditionally approved through single-arm trial design for accelerated marketing, we will need to discuss and reach consensus with the NMPA on details of the post-approval research pursuant to the relevant laws in China. To fully unleash the clinical value of IMM01 in a cost-effective and efficient manner, in addition to our internal development, we may also strategically seek out-licensing and other co-development opportunities to conduct clinical development in other jurisdictions.

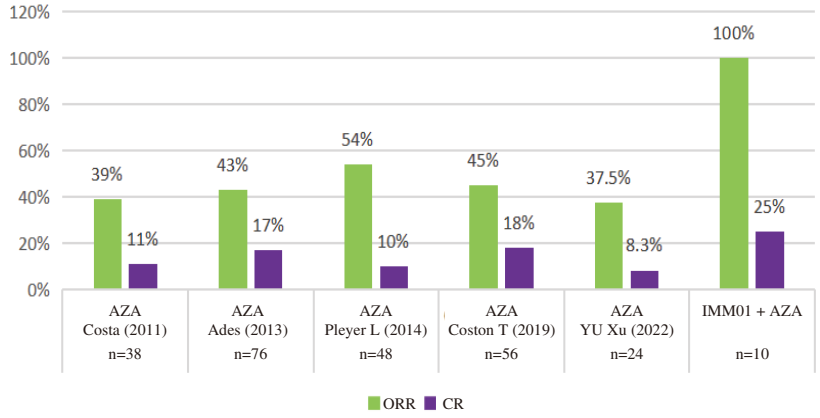
Combination with azacitidine

In the completed Phase Ib trial evaluating IMM01 in combination with azacitidine, we enrolled 12 patients in total, including 9 patients with R/R AML and 3 patients with R/R MDS. Upon completion of the Phase Ib trial, we initiated a Phase II trial to evaluate the safety and efficacy of IMM01 in combination with azacitidine mainly for the first-line treatment of HR MDS, unfit AML and CMML in June 2022. As of February 10, 2023, we have enrolled 78 patients, including 16 patients with treatment-naive AML, 44 patients with treatment-naive MDS, and 10

patients with treatment-naive CMML, as well as 8 patients with R/R MDS/AML. We plan to recruit around 80 to 90 patients in total for this trial. Subject to further clinical validation, we plan to file an IND application with the FDA for a Phase II study of this combination treatment.

Particularly, we believe we could seek an accelerated marketing approval through relatively small sample size studies targeting the first-line treatment of CMML in China, given that CMML is a rare disease whose incidence is around 1 of every 100,000 people in China, and there is a lack of effective treatment for CMML. As indicated by the graph below, 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 eight evaluable patients with 1L CMML, two reached complete response (2 CRs), six reached marrow complete response (6 mCRs), with one hematological improvement (1 HI), resulting in an ORR of 100% and a CR rate of 25%.

Summary of Major Clinical Studies in CMML



Notes: (1) The clinical data is as of February 10, 2023. (2) ORR refers to overall response rate; CR refers to complete response. (3) 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

According to Frost & Sullivan, only few drugs, such as azacitidine, have been approved for the first-line treatment of advanced CMML. However, the initial responses of azacitidine are often limited and short-lived, and very few of other CD47-based drug candidates are being evaluated for CMML in clinical trials. As an innovative drug targeting life-threatening malignancies without effective treatment, this combination therapy could be qualified to apply for an accelerated marketing approval, and the number of patients required for its pivotal trial could be relatively small considering its overall patient population. The Company dosed the first patient with CMML for the Phase II trial in August 2022. Subject to positive clinical results of the Phase II trial, the Company plans to commence a pivotal trial in December 2023 and then file an BLA with the NMPA in the first quarter of 2025.

Combination with tislelizumab

We intend to develop the combination therapy of IMM01 and tislelizumab for the treatment of solid tumors that are refractory or relapsed from the standard of care such as PD-1/PD-L1 inhibitors, including among others, NSCLC, SCLC, HNSCC and CRC, as well as R/R cHL. In February 2022, we obtained the IND approval from the NMPA for Phase Ib/II clinical trial to

evaluate the combination therapy of IMM01 and tislelizumab in solid tumors in China. We have procured Beigene's BAIZE'AN® (tislelizumab) at market price in the open market for our clinical trials, which is in compliance with the relevant laws and regulations and in line with industry practice. We are currently evaluating IMM01 and tislelizumab in a Phase II trial, and we expect to initiate a pivotal trial in the third quarter of 2024. As of February 10, 2023, we have enrolled 10 patients, including four patients with NSCLC, three patients with HNSCC, one patient with SCLC, two patients with cHL for this Phase II trial. After accumulating more clinical data, we may further evaluate this combination therapy for the first-line treatment of those solid tumors as well as for the treatment of other cancer indications.

We are also developing this combination therapy for cHL patients who relapsed or progressed after the treatment of PD-1 inhibitors, which may allow us to pursue an accelerated marketing approval leveraging the results of relatively small sample size studies. According to Frost & Sullivan, currently there is very limited effective treatment for cHL patients who relapsed or progressed post to PD-1 inhibitor treatment, presenting highly unmet medical needs. Given the strong synergistic effects observed in our preclinical studies and preliminary efficacy signal of IMM01 monotherapy shown in clinical trials, we believe this combination therapy has the potential to fulfill the unmet medical needs of those R/R cHL patients. Since none of other CD47-based drug candidates are being evaluated for R/R cHL in clinical trials to date, this combination therapy is well-positioned to pursue an accelerated marketing approval as an innovative therapy targeting R/R cHL if it can demonstrate its therapeutic benefits in the pivotal trial. Since cHL occurs in only 0.57 of every 100,000 people in China and the number of R/R cHL patients is fewer, the patient number required for its pivotal trial could be relatively small. In July 2022, we obtained the NMPA's consent for adding R/R cHL as an additional expansion cohort into the ongoing combination trial of IMM01 and tislelizumab. We dosed the first patient with R/R cHL in China in January 2023.

Combination with other drugs

We are currently exploring the therapeutic benefits of IMM01 in combination with various other drugs for a wide range of cancer indications. We reached a collaboration with Sunshine Guojian, under which Sunshine Guojian will be primarily responsible for driving and funding the clinical development of the combination of IMM01 and inetetamab for HER2-positive solid tumors in mainland China. We and Sunshine Guojian have obtained the IND approval for the Phase Ib/II trial to evaluate this combination therapy. Sunshine Guojian will formulate the detailed clinical plan and lead the clinical development for this combination therapy, and this combination trial has not been commenced as of the Latest Practicable Date as the progress of this clinical program is under the control of Sunshine Guojian based on their internal resource allocation and strategic priority. We are also conducting numerous preclinical studies to evaluate the combination use of IMM01 with other drugs targeting various cancer indications. Multiple combination therapies have shown robust synergistic potential in mouse models.

In addition, we have obtained an IND approval for the Phase Ib/II clinical trial to evaluate the combination of IMM01 with rituximab (a CD20 mAb) for the treatment of R/R B-NHL from the NMPA in August 2021. Since we will place our focus on the development of IMM0306 for this indication, we currently do not plan to initiate any clinical trials for this combination therapy in the near future. We have also obtained an IND approval for the Phase Ib/IIa clinical trial to evaluate the combination of IMM01 with bortezomib and dexamethasonum for the treatment of MM from the NMPA in January 2023. We may seek partnership to further develop this combination therapy.

Licenses, Rights and Obligations

We are internally developing IMM01, and own the global rights to research, develop and commercialize IMM01. We are collaborating with Sunshine Guojian to develop a combination therapy using inetetamab and IMM01 for the treatment of HER2-positive solid tumors in mainland China (excluding Hong Kong, Macau and Taiwan). For details, please refer to the paragraph headed "— Collaboration Agreement — Collaboration with Sunshine Guojian."

Material Communications

We had not received any regulatory agency's concerns or objections to our clinical development plans as of the Latest Practicable Date.

WE MAY NOT BE ABLE TO ULTIMATELY DEVELOP AND MARKET IMM01 SUCCESSFULLY.

CD47-based Bispecific Molecules

Based on the validated molecule structure of IMM01, we have subsequently developed multiple CD47-based bispecific molecules leveraging our mAb-Trap platform. These bispecific molecules all contain the same engineered CD47-binding fragment used in IMM01 and an ADCC-enhanced IgG1 Fc fragment. 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.

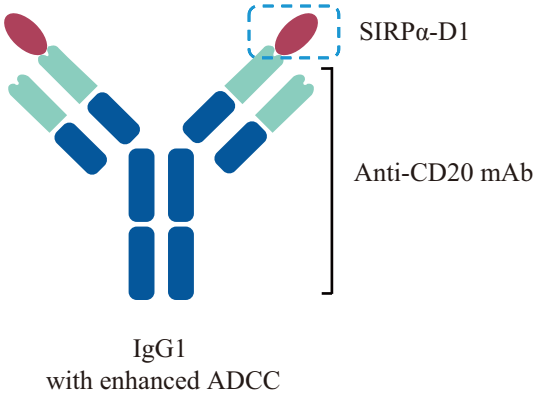
Studies on crystal structure of CD47 have revealed that CD47-binding region of SIRPα is located at its N-terminal. When designing the molecules, we thus connect the CD47-binding domain to the N-terminal of the heavy chain or light chain of a base antibody against another tumor target rather than to the Fc end, as is commonly seen in other CD47 based bispecifics. Our design prevents conformational interference with CD47 binding and preserves the intact Fc region with full immune effector function.

A prerequisite for a combination therapy to exert synergistic effects is that the two agents must simultaneously bind to the same cancer cell. As only a portion of the single agents administered will bind with same cancer cells, a much higher dosing level of each agent will be required to achieve a strong synergistic effect. Comparatively, our bispecific molecules with a higher affinity for a tumor antigen than CD47 are more likely to bind to two targets co-expressed on the same tumor cell, and simultaneously activate immune responses through the ADCC-enhanced IgG1 Fc, allowing for stronger synergistic effects. Our preclinical studies have shown that these bispecific molecules, even at a relatively lower dose level, could have better synergistic effects than the combination therapies of two antibodies targeting respective targets. Further, with the fine-tuned unbalanced binding affinity, our bispecific molecules can preferentially bind to CD47 on tumor cells, minimizing "on-target, off-tumor" toxicity. In addition, the symmetric structure of our bispecific molecules developed on our mAb-Trap platform minimizes mismatch during the production process, allowing for ease of manufacturing, product stability, higher titer and protein yield. In fact, average protein yield for IMM0306, IMM2902, and IMM2520 ranges from 3.8g/L to 4.6g/L, much higher than the industry average for bispecific molecules of 1.0g/L to 3.0g/L.

IMM0306 (CD47×CD20) — Our Key Product

IMM0306 is a bispecific molecule that simultaneously targets both CD47 and CD20 and is the first CD47 and CD20 dual-targeting bispecific to enter into clinical stage globally. The diagram below illustrates the molecule structure of IMM0306:

IMM0306



Source: Company Data

Based on our mAb-Trap platform, we designed the molecule of IMM0306 to consist of the CD47-binding domain of IMM01 and an ADCC-enhanced IgG1 Fc fragment which is capable of inducing full macrophage activation and much improved ADCP and ADCC activity, resulting in strong antitumor immune responses.

In our preclinical studies, IMM0306 elicited stronger *in vivo* antitumor activity compared to rituximab single agent or its combination with IMM01, and showed a favorable safety profile. In May 2020, we initiated a Phase I clinical trial to evaluate IMM0306 in R/R B-NHL in China. The preliminary data from the Phase I clinical trial has demonstrated encouraging efficacy and favorable safety profile of IMM0306. IMM0306 was safe and well tolerated up to 2.0 mg/kg. Among the evaluable patients across four cohorts dosed from 0.8 mg/kg to 2.0 mg/kg, who had relapsed or progressed after receiving rituximab previously, two CRs and five PRs were observed. The only evaluable FL patient at 2.0 mg/kg who relapsed and progressed after rituximab treatment has also been confirmed as PR. At 2.0 mg/kg, one patient with primary bone DLBCL who had four lines of prior treatment has achieved PR with all measurable lesions disappeared after 65 days of treatment. We commenced a Phase IIa trial for IMM0306 monotherapy for the third- or later-line treatment of FL in March 2023, and expect to start pivotal trials in the third quarter of 2024. Furthermore, our IND application for the combination of IMM0306 and lenalidomide targeting front-line B-NHL was approved by the NMPA in January 2023, and we are in preparation to commence the Phase Ib trial for this combination in China. We have also received an IND approval for IMM0306 from the FDA in January 2021. With further clinical validation in the Phase I trial in China, we will then decide on our clinical development and collaboration strategy for IMM0306 in the U.S.

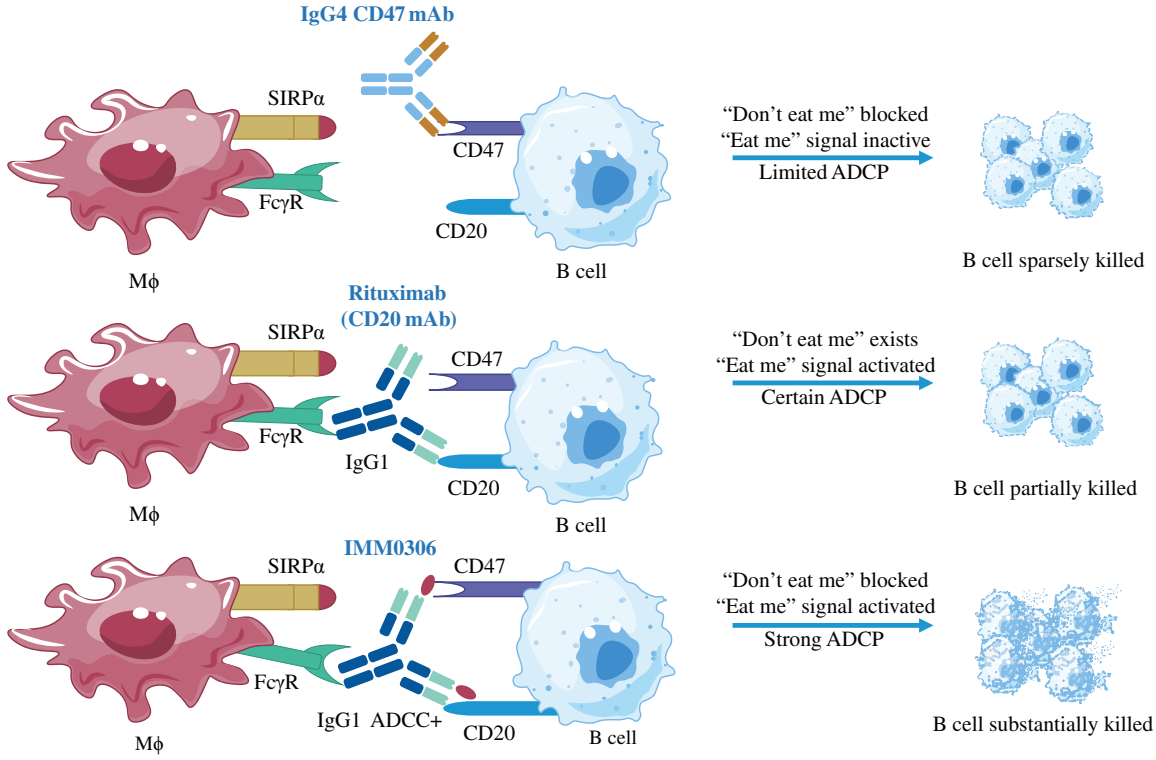
We are developing IMM0306 in-house and own its global IP and commercial rights. As of the Latest Practicable Date, with respect to IMM0306, we owned one patent family, which includes four issued patents in China, Japan and the U.S., one allowed patent application in the EU, and one PCT patent application which has entered national phases.

Mechanism of Action

Upon binding with CD20 and CD47, IMM0306 is expected to deplete malignant B cells by inducing enhanced ADCC and ADCP activity and possibly eliciting subsequent T-cell response, leading to an integrated immune activation. The ADCC-enhanced IgG1 Fc region of IMM0306 could further improve its effectiveness for treating patients predominantly expressing FcγRIIIA-158F polymorphism that is less sensitive to CD20 antibody treatment, according to Frost & Sullivan. Public data have demonstrated synergistic therapeutic benefits of the combination use of CD47-targeted agents and CD20 antibodies, showcasing the advantages of this dual-targeting strategy. However, a prerequisite for synergistic effects in this combination therapy is that these two agents must simultaneously bind to the same cancer cell. As only a portion of the single agents administered will bind with same cancer cells, a much higher dosing level of each agent will be required to achieve a strong synergistic effect. Comparatively, IMM0306, as a bispecific molecule, is more likely to bind to two targets co-expressed on the same cell, and simultaneously activate immune responses through its ADCC-enhanced IgG1 Fc, allowing for stronger synergistic effects even at a relatively lower dose level. To ensure targeting specificity of the molecule, the fine-tuned unbalanced binding affinity enables selective targeting to CD20-positive malignant B cells and mitigates "on-target, off-tumor" toxicity by minimizing inadvertent binding to CD47 on blood cells or other normal tissues.

The following diagrams illustrate the mechanism of action of IMM0306 in comparison to the combination of separate agents targeting CD47 and CD20:

Mechanism of Action of IMM0306 versus Combination of CD47 mAb and CD20 mAb



Source: Company Data

Market Opportunities and Competition

We are currently developing IMM0306 for the treatment of R/R B-NHL. According to Frost & Sullivan, the global and China incidence of NHL was 556.2 thousand and 95.2 thousand in 2021, respectively, and is expected to increase to 670.3 thousand and 117.4 thousand in 2030, respectively. B-NHL patients account for 85% of patients with NHL. According to Frost & Sullivan, approximately 95% of B-NHL express CD20 antigen. CD20 antibody in combination with chemotherapy is the main treatment option covering the first-line and following treatment for B-NHL. However, approximately 50% of NHL patients will eventually experience disease progression to R/R NHL, which remains a challenge with limited effective treatment options. For R/R B-NHL, CD20-targeted therapy is generally associated with limited effectiveness due to drug resistance. As B-NHL is a malignant tumor of lymphatic system which contains numerous immune cells, simultaneously targeting innate and adaptive immunity have great potential in addressing the unmet needs of NHL treatment.

According to Frost & Sullivan, there were two CD47×CD20 bispecific antibodies/fusion proteins under development globally as of the Latest Practicable Date. Among them, IMM0306 is the first one to enter into a clinical trial. In our ongoing Phase I clinical trial, IMM0306 has shown promising efficacy signals in treating patients with R/R B-NHL. Given IMM0306's much more potent *in vivo* efficacy compared to rituximab in our preclinical studies, as well as its encouraging preliminary clinical efficacy data targeting R/R patients previously treated with and progressed after rituximab, we believe that it also has the potential to become a new first-line treatment option for our targeted indications.

Competitive Advantages

We believe IMM0306 has the following competitive advantages:

(1) Potent in vivo antitumor effects at a lower dosing level compared to CD20 antibody as monotherapy or its combination with IMM01

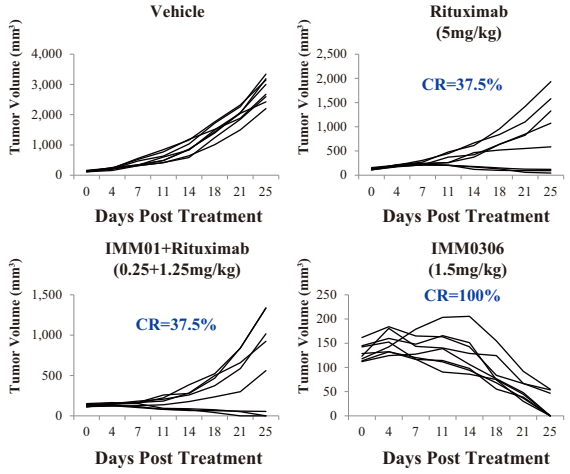
While CD20 antibody used with chemotherapy is currently the main treatment option covering all lines of B-NHL treatment, the potency of CD20 antibody could be hampered by the inhibitory signaling of CD47. By dual-targeting of CD20 and CD47, IMM0306 can lead to stronger antitumor effects than a CD20 antibody through eliciting more integrated immune responses.

Although the combined use of a CD47-targeted agent and a CD20 antibody can also achieve the synergistic activity, the prerequisite for such synergistic activity is that both antibodies bind simultaneously on the same tumor cell. Compared to the combination therapy, IMM0306 is more likely to bind with two targets co-expressed on the same tumor cell, thus achieving stronger synergistic effects at a relatively lower dose level. In addition, the IgG1 Fc of IMM0306 enables the molecule to potentially treat patients with the predominantly expressed less-sensitive FcγRIIIA polymorphism (FcγRIIIA-158F).

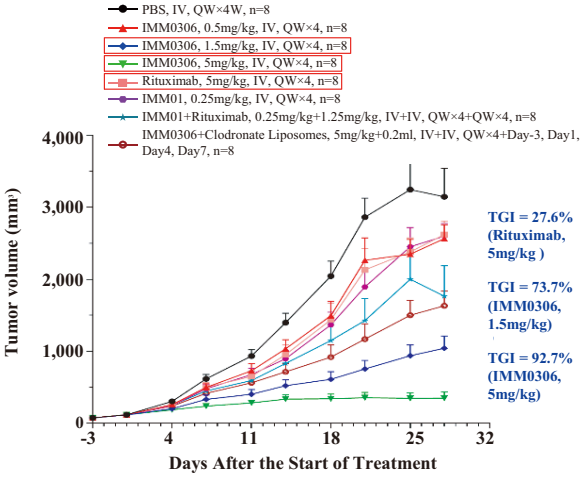
Our *in vivo* efficacy studies have demonstrated that IMM0306 was more potent than rituximab (CD20 mAb) monotherapy, even at a much lower dosing level, and it is more potent than the combination therapy of IMM01 and rituximab at a comparable dosing level. As shown in the diagrams below, under Daudi xenograft model in SCID mice, IMM0306 resulted in complete remission in 100% of mice at 1.5 mg/kg. In the same model, the combination therapy of IMM01 and rituximab at a comparable dosing level only resulted in a complete remission rate of 37.5%, and rituximab monotherapy led to a complete remission rate of 37.5% even at a much higher dose

of 5 mg/kg. Under Raji xenograft model in SCID mice, IMM0306 led to a dose-dependent response with TGI rates of 73.7% and 92.7% at 1.5 mg/kg and 5 mg/kg, respectively. In comparison, at a high dose of 5.0 mg/kg, rituximab only resulted in a much lower TGI rate of 27.6%.

Efficacy Study in Lymphoma (Daudi) Xenograft Mouse Model



Efficacy Study in Lymphoma (Raji) Xenograft Mouse Model



Note: Eight mice per group were used in this study.
Source: Company Data

We dosed the first patient in a Phase I clinical trial in treating R/R B-NHL in China in May 2020, and preliminary results available thus far showed positive efficacy signals. All patients enrolled in this trial had relapsed or progressed after receiving rituximab previously. Among the evaluable patients across four cohorts dosed from 0.8 mg/kg to 2.0 mg/kg, who had relapsed or progressed after receiving rituximab previously, two CRs and five PRs were observed. The only evaluable FL patient at 2.0 mg/kg who relapsed and progressed after rituximab treatment has also been confirmed as PR. At 2.0 mg/kg, one patient with primary bone DLBCL who had four lines of prior treatment has achieved PR with all measurable lesions disappeared after 65 days of treatment.

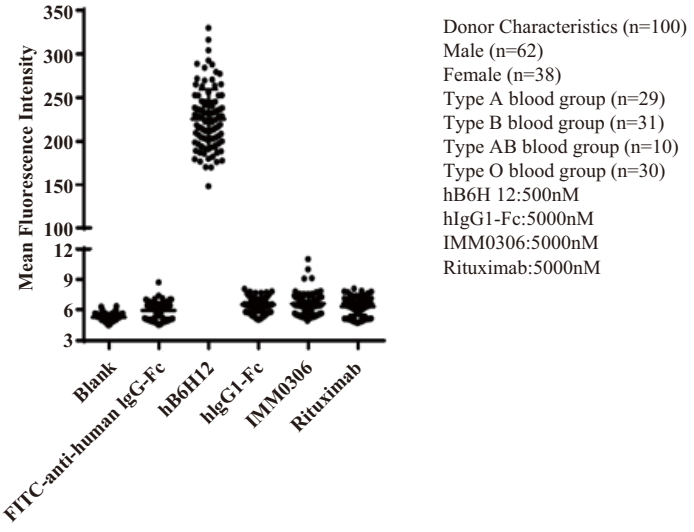
Currently, the first-line treatment of B-cell lymphoma is primarily CD20 antibody (such as rituximab) plus chemotherapy. IMM0306 has revealed promising efficacy targeting patients who had relapsed or progressed after receiving rituximab. Since IMM0306 demonstrates much stronger *in vivo* antitumor activity than rituximab at a lower dose level, we believe IMM0306 has the great potential to become a first-line treatment option for B-cell lymphoma.

(2) Favorable safety profile with no human red blood cell binding in vitro, with only minor cytokine storm

Major concerns regarding the use of CD47-targeted agents are driven by the ubiquitous expression of CD47 in normal tissues, especially on RBCs, which leads to severe blood toxicity and antigenic sink. The safety concerns set up a high technical barrier for the molecule design of CD47-targeted agents. Our IMM0306 does not bind to RBCs in *in vitro* preclinical studies or cause hemagglutination in clinical trials, attributable to the same CD47-binding domain used in IMM01. With the higher affinity for CD20, IMM0306 can preferentially bind to CD20 and CD47 co-expressing tumor cells, thus minimizing "on-target, off-tumor" toxicity.

As shown in the diagram below, based on the blood samples drawn from 100 donors including males and females with different blood types, IMM0306 interacting with human RBCs manifests minimum mean fluorescence intensity as measured by the flow cytometer, demonstrating no binding activity toward human RBCs, while hB6H12, a CD47 antibody, showed significant RBC binding activities.

Human RBC Binding Analysis of IMM0306



Source: Company Data

Moreover, different from T-cell engaging bispecific antibodies, which normally induce serious cytokine release syndrome (CRS), a severe immune reaction in which the body releases too many cytokines within a very short time leading to severe inflammation and potential organ failures, IMM0306 only triggers minor CRS. CRS is one of the main reasons driving dose-limiting toxicities of T-cell engaging bispecific antibodies, mostly CD3-based bispecific antibodies due to their direct activation of T cells, which eventually leads to the termination or suspension of multiple clinical trials for CD3-based bispecifics, including Amgen's AMG673 (CD3×CD33), AMG427 (CD3×FLT3) and AMG701 (CD3×BCMA), Regeneron's odronextamab (CD3×CD20), and Pfizer's elranatamab (CD3×BCMA). Our preliminary clinical data has suggested favorable safety and tolerability profiles of IMM0306. As of February 27, 2022, among 48 patients enrolled in its Phase I clinical trial, no DLT was observed and MTD was not reached. The majority of TRAEs observed are Grade 1 and 2. The most frequent TRAEs were lymphocyte decrease, white blood cell decrease, neutropenia, platelet decrease, anemia and drug-related infusion related reactions.

Clinical Development Plan

We are executing a comprehensive clinical development plan for IMM0306 in China and the U.S. We initiated a Phase I clinical trial of IMM0306 in treating R/R B-NHL in China in May 2020. We commenced a Phase IIa trial for IMM0306 monotherapy for the third- or later-line treatment of FL in March 2023 and plan to seek an accelerated marketing approval through a single-arm trial. We expect to commence pivotal clinical trials in China in the third quarter of 2024. Furthermore, our IND application for the combination of IMM0306 and lenalidomide targeting front-line B-NHL was approved by the NMPA in January 2023, and we are in preparation to commence the Phase Ib trial for this combination in China. We have also received an IND

approval for IMM0306 from the FDA in January 2021. With further clinical validation in the Phase I trial in China, we will then decide on our clinical development and collaboration strategy for IMM0306 in the U.S.

Licenses, Rights and Obligations

We are developing IMM0306 in-house and own the global rights to develop and commercialize IMM0306.

Material Communications

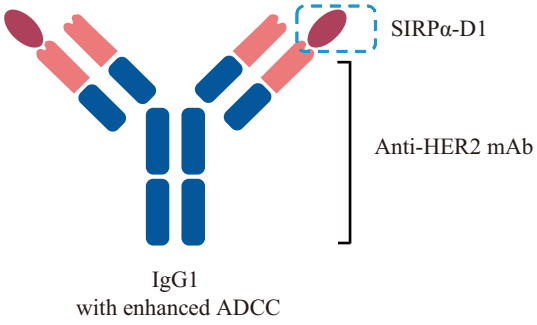
We had not received any regulatory agency's concerns or objections to our clinical development plans as of the Latest Practicable Date.

WE MAY NOT BE ABLE TO ULTIMATELY DEVELOP AND MARKET IMM0306 SUCCESSFULLY.

IMM2902 (CD47×HER2) — Our Key Product

IMM2902 is the only bispecific molecule targeting CD47 and HER2 simultaneously that has entered into clinical trial globally. The following diagram illustrates the structure of IMM2902:

IMM2902



Source: Company Data

With its unique structural design with the engineered CD47-binding fragment connected to the N-terminus of light chains, our IMM2902 shows no RBC binding *in vitro,* and is able to adopt an ADCC-enhanced IgG1 Fc fragment capable of inducing full macrophage activation, enhanced ADCP and ADCC activity, and potent antitumor immune responses. By simultaneously binding to HER2 and CD47, IMM2902 suppresses tumor cell growth and proliferation through the blockade of CD47/SIRPα immune inhibitory signal, enhanced ADCP/ADCC, as well as the induction of accelerated HER2 internalization and degradation. Additionally, the structurally optimized IgG1 Fc could potentially induce ADCT as found with amivantamab.

IMM2902 demonstrated potent antitumor activity in our *in vivo* efficacy models of trastuzumab-sensitive and trastuzumab-resistant HER2-low expressing BC and GC. In addition, it exhibited a favorable safety profile in our preclinical studies. We have initiated the Phase Ia/Ib trial for the treatment of advanced HER2-positive and HER2-low expressing solid tumors, including BC, GC, NSCLC and BTC, in China in February 2022. We have also initiated the clinical trial for advanced HER2-positive and HER2-low expressing solid tumors in the U.S. with the first patient dosed in June 2022. We have received the Fast Track Designation from the FDA in July 2022. We expect to largely complete the Phase Ia trials in China and the U.S. in 2023.

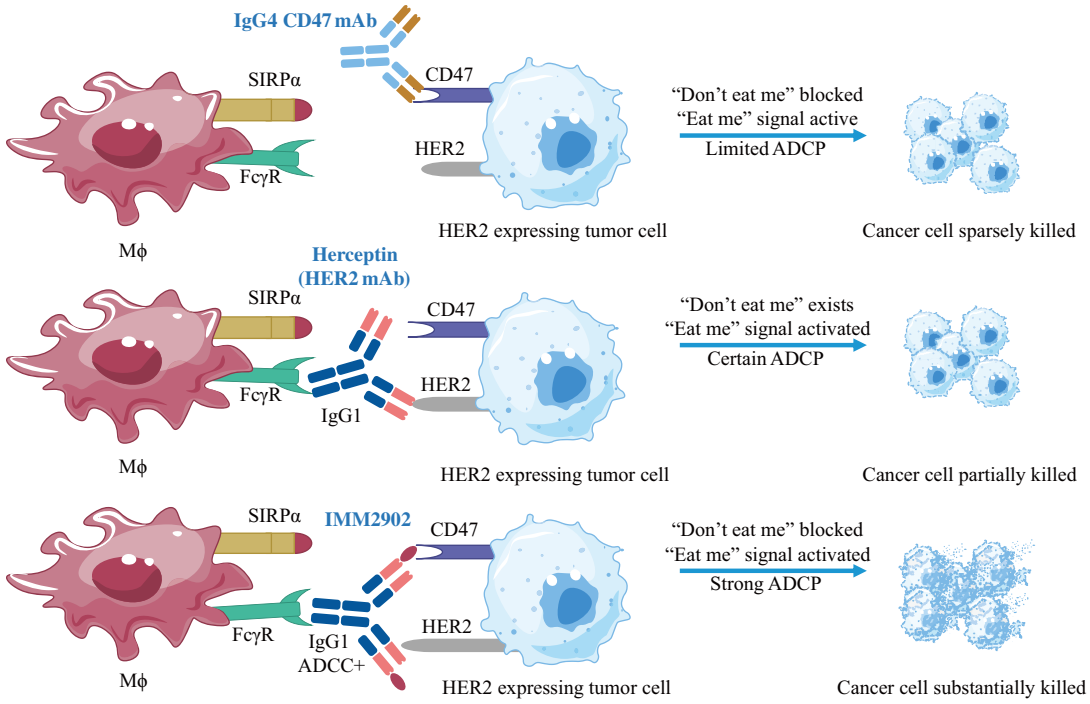
As of the Latest Practicable Date, with respect to IMM2902, we owned one patent family, which includes one issued patent in the U.S., one issued patent in Japan, four pending patent applications in the PRC, the U.S., the EU and Hong Kong, and one PCT patent application which has entered national phases.

Mechanism of Action

HER2 regulates cell proliferation and apoptosis, and amplification of HER2 gene promotes the acceleration of tumor cell growth. With the higher affinity for HER2, IMM2902 can preferentially bind to HER2 and CD47-positive tumor cells, while sparing CD47-expressing normal cells, to minimize "on-target, off-tumor" toxicity. IMM2902 can inhibit the signaling of HER2 pathway, thereby directly suppressing tumor growth and proliferation and leading to cell death. IMM2902 has been shown to accelerate the degradation of HER2, leading to tumor cell apoptosis. Moreover, it can block the "don't eat me" signal via disrupting CD47/SIRPα interaction and also activate the "eat me" signal through Fc-FcγR engagement, thereby fully activating macrophages. The IgG1 Fc fragment is further engineered to enhance ADCC activity, especially benefiting the patient population harbouring the predominantly expressed polymorphism of phenylalanine at 158 amino acid position of the Fcγ receptor IIIA (FcγRIIIA-158F). Additionally, IMM2902 is expected to potentially induce ADCT as found with amivantamab (a marketed EGFR/c-MET bispecific antibody designed with IgG1 Fc), an underappreciated mechanism of action contributing to tumor suppression. With the multi-targeting ability and multifaceted mechanisms against tumor cells, IMM2902 is expected to achieve much stronger antitumor activity at a lower dosing level, as compared to HER2 antibodies or their combination with IMM01, and efficacious even for solid tumors with HER2-low expression.

The following diagram illustrates the mechanisms of action of IMM2902 in comparison to the combination of separate agents targeting CD47 and HER2:

Mechanism of Action of IMM2902 versus Combination of CD47 mAb and HER2 mAb



Source: Company Data

Market Opportunities and Competition

According to Frost & Sullivan, HER2 overexpression is prevalent in many major cancer types, such as BC, GC, lung cancer, CRC, esophageal cancer (EC), BTC, HNSCC and cervical cancer (CC). According to Frost & Sullivan, the incidence of BC reached 2.3 million and 0.3 million worldwide and in China in 2021, respectively, and is expected to increase to 2.7 million and 0.4 million in 2030, respectively. The incidence of GC was 1.1 million and 0.5 million in 2021 globally and in China, respectively, and is expected to increase to 1.4 million and 0.6 million in 2030, respectively. The incidence of other major HER2-expressing cancers was 9.6 million and 2.5 million in 2021 globally and in China, respectively, and 12.0 million and 3.3 million in 2030 globally and in China, respectively.

While HER2 antibodies (such as trastuzumab) have been used as the standard treatment for HER2-positive BC and GC in combination with chemotherapy, around 35% of HER2-positive cancer patients have intrinsic resistance to the standard treatment, and the remaining 65% who respond to the standard treatment will eventually develop acquired resistance to the standard treatment with a median response duration of 12.5 months, resulting in disease progression. Moreover, patients with HER2-low expression who comprise about 50% of all BC cases and over 25% of GC cases do not respond to HER2 antibodies in general. Although HER2 antibody-drug conjugates (ADCs) are shown to be active in certain HER2-low expressing tumors in clinical trials, they are often associated with severe adverse effects, such as interstitial lung disease, and can sometimes be fatal. ADCs, such as Enhertu®, still present limited PFS/OS data targeting patients with certain HER2-expressing solid tumors (including GC and NSCLC), despite a much improved ORR rate. This suggests a clear need to develop novel therapeutics with a better efficacy-safety balance for patients with HER2-low expressing cancers and trastuzumab-resistant cancers.

CD47 and HER2 dual-targeting strategy may provide safer and more efficacious treatment for patients with HER2-low expressing solid tumors and those relapsed from trastuzumab treatment. To date, IMM2902 is the only CD47 and HER2 bispecific molecule that has entered into clinical stage globally. Given its multifaceted mechanisms, IMM2902 has shown potent antitumor activity in HER2-low expressing and trastuzumab-resistant solid tumor models. Thus, IMM2902 has the potential to benefit a large patient population globally, including the even larger market with HER2-low expressing solid tumors and those relapsed after prior trastuzumab treatment.

Competitive Advantages

We believe IMM2902 has the following competitive advantages:

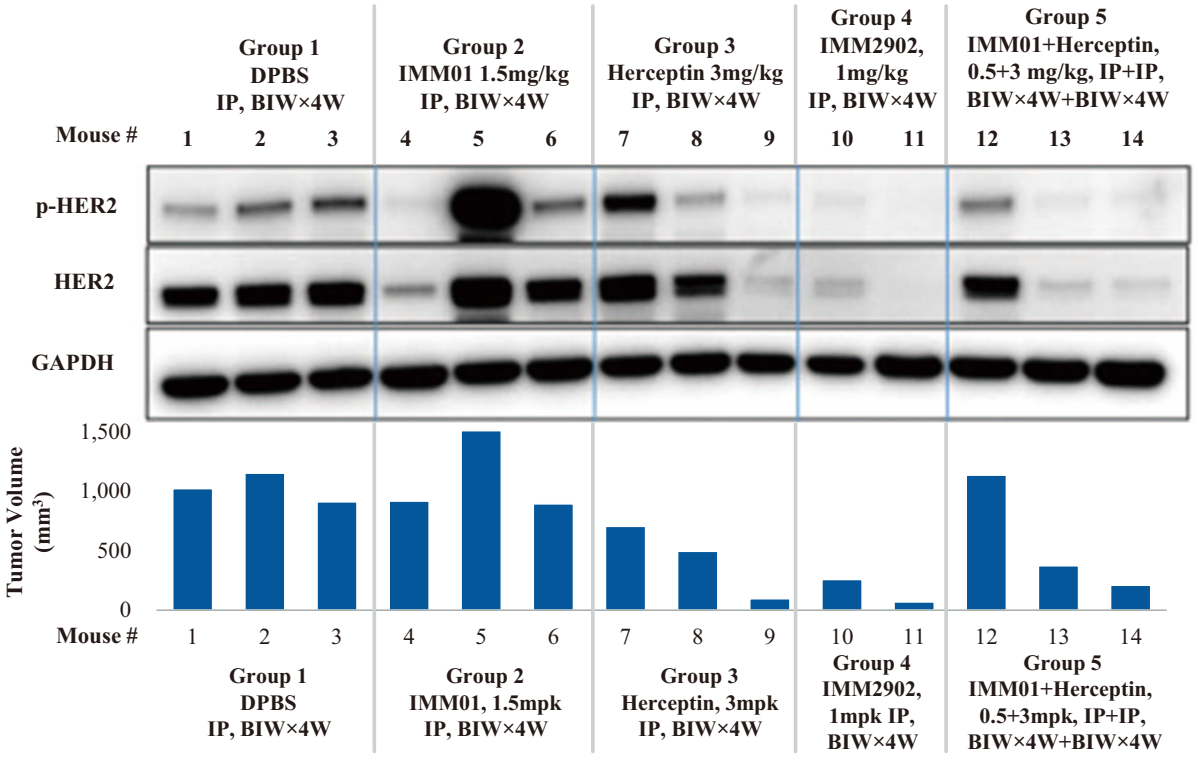
(1) Enhanced ADCC, ADCP, potentially ADCT, and accelerated HER2 degradation

IMM2902 can fully activate macrophages by activating an additional "eat me" signal, leading to phagocytosis against tumor cells, and stronger T-cell response through the secretion of immune modulatory cytokines and chemokines and boosted antigen presentation. In addition, the IgG1 Fc fragment of IMM2902 is further engineered to enhance ADCC activity. IMM2902 is also expected to potentially induce ADCT activity, another important Fc-induced mechanism observed with amivantamab (a marketed EGFR/c-MET bispecific antibody with IgG1 Fc), which works together with ADCC and ADCP to combat tumor cells. Through these mechanisms, IMM2902 can induce all-around innate and adaptive immune responses and potent tumor killing.

Further, our preclinical study showed that IMM2902 could accelerate the endocytosis and degradation of HER2, thereby resulting in robust tumor suppression. We conducted a Western blot analysis on tumor tissues to compare the HER2 protein degradation induced by IMM2902 with IMM01, trastuzumab and their combination treatment. As can be seen from the diagram below, the reduction of HER2 expression has a strong correlation to the shrinkage of tumor size. Notably, in

Group 4 where IMM2902 was administered at a lower dose level of 1 mg/kg (~0.1 mg/kg human equivalent dose) than other study arms, HER2 protein expression significantly decreased due to accelerated degradation induced by IMM2902, and consequently, IMM2902 produced the strongest tumor growth inhibitory activity among all treatment groups.

Expression Analysis of HER2 and p-HER2 by Western Blot



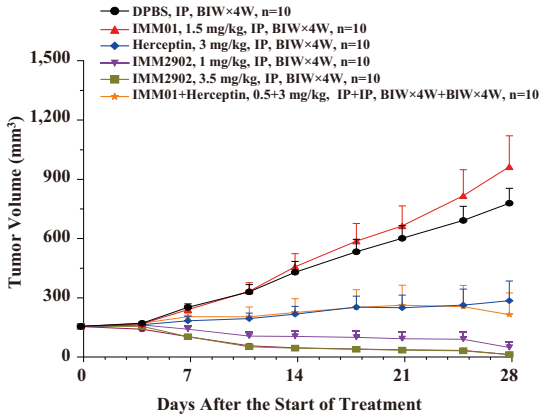
Notes: (1) The data from the Western blot analysis is representative images of the preclinical study. (2) p-HER2 refers to phospho-HER2, DPBS refers to Dulbecco's Phosphate Buffered Saline, intended to provide a buffer system for maintaining cell culture media in the physiological range of 7.2 to 7.6. (3) Ten mice per group were used in this study. (4) While the change in p-HER2 among the treated group is not significant when compared to the control group, the change in constitutive expression of HER2 in IMM2902-treated group is significantly lower than that in the control group. (5) It demonstrated that down-regulation of HER2 is one of the many important mechanisms by which IMM2902 exerts antitumor activity. Similar phenomenon could be referred to amivantamab (an EGFR×MET bispecific antibody) inducing strong *in vivo* antitumor activity via several mechanisms including down-regulation of EGFR and MET expression on tumor cells *(Mol Cancer Ther (2020) 19 (10):2044-2056)*. (6) The study primarily focus on correlations of different variables, which does not necessarily imply a causative relationship.

Source: Company Data

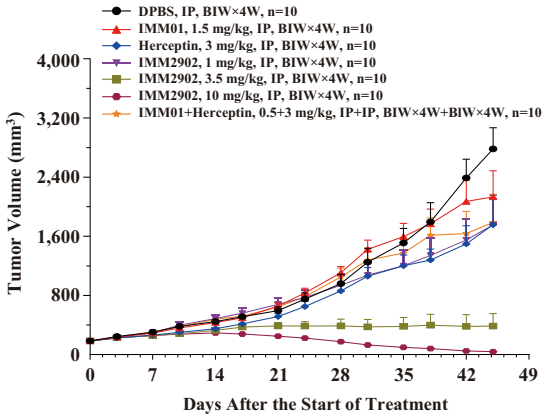
(2) Strong in vivo antitumor efficacy

A series of *in vivo* efficacy studies have been completed by two independent and reputable CROs to evaluate tumor inhibitory effects of IMM2902 in xenograft models that are sensitive or resistant to trastuzumab. These preclinical studies revealed strong antitumor activity of IMM2902 against a variety of breast and gastric tumors. As shown in the panels below, IMM2902 completely eradicated established tumors at 10 mg/kg (~1.0 mg/kg human equivalent dose) in both trastuzumab-sensitive and trastuzumab-resistant BC models. In addition, at equivalent doses, IMM2902 was significantly more efficacious than trastuzumab alone or its combination with IMM01. IMM2902 also exhibited favorable efficacy in trastuzumab-sensitive and HER2-low expressing GC models. These promising preclinical results suggest the potential of IMM2902 to treat cancer patients who have relapsed from initial trastuzumab treatment and to subsequently advance to the first-line setting.

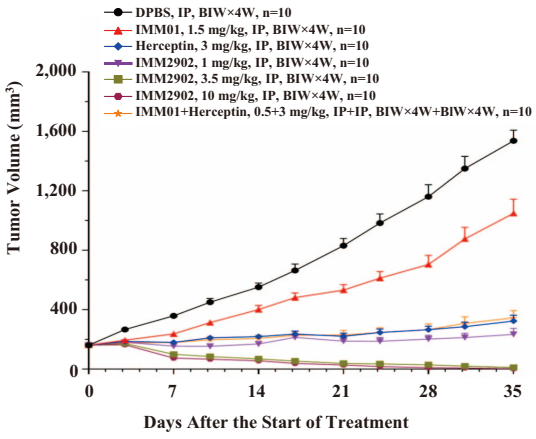
Efficacy Study in Trastuzumab-Sensitive Breast Cancer (BT474) Xenograft Mouse Model



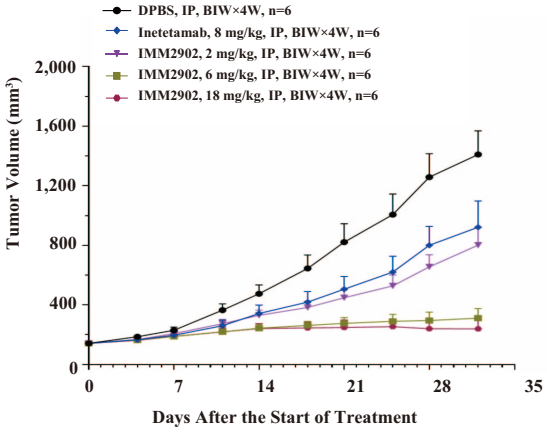
Efficacy Study in Trastuzumab-resistant Breast Cancer (HCC-1954) Xenograft Mouse Model



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



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

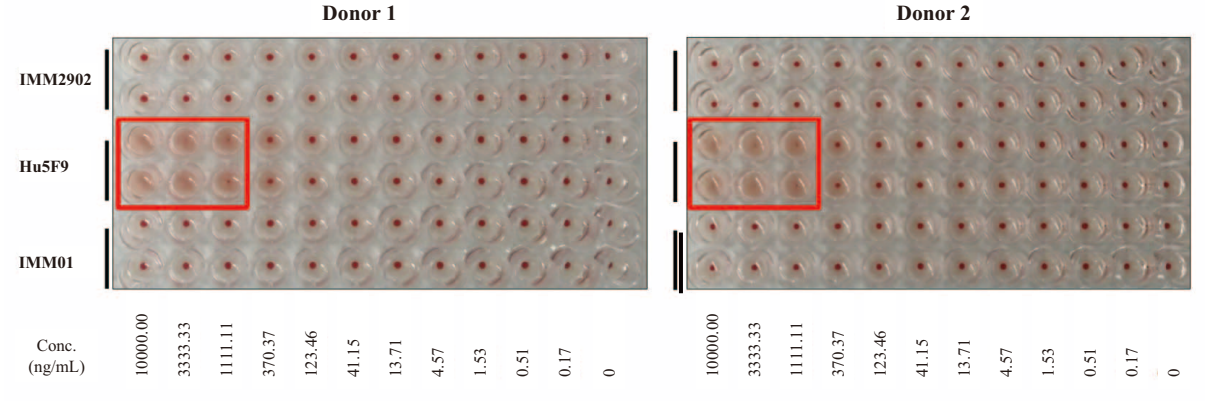


Source: Company Data

(3) 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 (clumping of RBCs) *in vitro*. In our preclinical studies as shown below, while magrolimab analog replicated by us based on public information induced obvious hemagglutination at the concentration beyond 370 ng/ml, IMM2902 did not induce hemagglutination even at the concentration as high as 10,000 ng/ml. In addition, IMM2902 with a higher affinity for HER2 than CD47 can preferentially bind with tumor cells co-expressing HER2 and CD47 rather than CD47-positive normal tissues (including RBCs), which further improves its safety and tolerability.

IMM2902 Does Not Induce Hemagglutination of Human Red Blood Cells



Note: Analog of magrolimab (Hu5F9) used in this study was replicated by an independent biotechnology company based on public information, which may not be exactly identical to magrolimab but can exhibit identical or very similar results in preclinical studies. When a competing drug is not available on the market, it is acceptable and common to use its analog for preclinical evaluation in the industry. Our preclinical study showed that Hu5F9 started to induce obvious hemagglutination at the concentration of 370 ng/ml, and neither IMM2902 nor IMM01 induced hemagglutination at the concentration as high as 10,000 ng/ml. The results of this preclinical study provide important guidance to predict the effects of study drugs in human. If the average blood concentration required for a drug to be effective in a human body is higher than the concentration level that induced hemagglutination *in vitro* (such as 370 ng/ml for Hu5F9 and 10,000 ng/ml for IMM2902 and IMM01), hemagglutination may be induced in human body. As the concentration of a drug in peripheral blood shortly after it is injected will be generally higher than the calculated average blood concentration for a specific dose level, and aging RBCs with poor glycosylation stuck on the walls of blood vessels are more likely to bind with CD47-targeted agents at a lower dose, the dose level that may cause hemagglutination in human could be lower than that observed in this study.

Source: Company Data

Clinical Development Plan

We initiated a Phase Ia/Ib trial for IMM2902 in advanced HER2-positive and HER2-low expressing solid tumors, including BC, GC, NSCLC and BTC, in China in February 2022, and are enrolling the sixth cohort for this dose-escalation study in China. Based on an IND approval for IMM2902 in HER2-positive and HER2-low expressing solid tumors granted by the FDA in August 2021, we have also initiated the clinical trial for advanced HER2-positive and HER2-low expressing solid tumors in the U.S. with the first patient dosed in June 2022. We have received the Fast Track Designation from the FDA in July 2022. We expect to largely complete the Phase Ia trials in China and the U.S. in 2023.

Licenses, Rights and Obligations

We are developing IMM2902 in-house and own the global rights to develop and commercialize IMM2902.

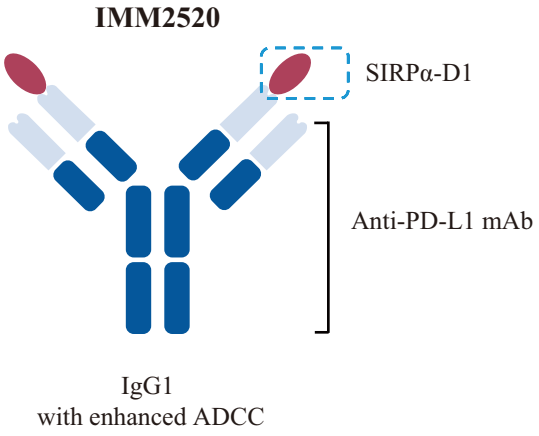
Material Communications

We had not received any regulatory agency's concerns or objections to our clinical development plans as of the Latest Practicable Date.

WE MAY NOT BE ABLE TO ULTIMATELY DEVELOP AND MARKET IMM2902 SUCCESSFULLY.

IMM2520 (CD47×PD-L1) — Our Key Product

IMM2520 is a CD47 and PD-L1 dual-targeting bispecific molecule for the treatment of solid tumors. As shown in the following diagram, IMM2520 consists of a PD-L1 antibody with an engineered ADCC-enhanced IgG1 Fc region, linked to the same CD47-binding domain used in IMM01 at the N-terminus of heavy chains:



Source: Company Data

This unique structure allows our CD47-based bispecific molecules to avoid RBC binding, thus enabling the adoption of an ADCC-enhanced IgG1 Fc fragment to fully activate macrophages and induce enhanced ADCP and ADCC activity, resulting in potent integrated antitumor immune responses. We have obtained IND approvals for IMM2520 from the NMPA in November 2022 and from the FDA in December 2022, and dosed the first patient for the Phase I clinical trial in China in March 2023. We will particularly focus on the solid tumors that are generally resistant or not sensitive to the currently available immunotherapies, such as CRC, GC, lung cancer and HNSCC, among others. As of the Latest Practicable Date, with respect to IMM2520, we owned one patent family, which includes one issued patent in Japan, one allowed patent application in the U.S., one allowed patent application in the PRC, one pending patent application in the EU, and one pending PCT patent application which may enter various contracting states in the future.

Mechanism of Action

CD47 and PD-L1 serve as critical innate and adaptive immune checkpoints, respectively, as these are two key pathways frequently exploited by various cancer cells to escape immune responses. Although PD-1/PD-L1 inhibitors have been approved for the treatment of a broad range of cancers, they only produce limited responses in "cold tumors" or non-T cell-inflamed immune-suppressive TME. With its potent IgG1 Fc, IMM2520 is able to deliver the additionally required "eat me" signal via Fc-FcγR engagement, thus effectively activating macrophages to exert tumor killing activity through multiple integrated mechanisms of action. Fully activated macrophages, on the other hand, are able to transform "cold tumors" into "hot tumors" and sensitize TME to the PD-1/PD-L1 inhibition, showing great synergistic potential with T-cell activation. Moreover, the engineered IgG1 Fc region also induces enhanced ADCC mediated by NK cells, leading to direct tumor-killing effects. Due to the crosstalk among macrophages, NK cells and T cells, IMM2520 is able to unleash significant synergistic effects, fully eliciting all-around innate and adaptive immune responses and leading to profound and durable tumor

killing effects. With the higher affinity for PD-L1, IMM2520 can preferentially bind with PD-L1 and CD47 co-expressing tumor cells, rather than normal cells expressing CD47, thus minimizing "on-target, off-tumor" toxicity.

Market Opportunities and Competition

According to Frost & Sullivan, only about 10% to 25% of cancer patients are responsive to PD-1/PD-L1 inhibitor monotherapy across almost all major types of cancer, due to "cold tumors" or non-T cell-inflamed immune-suppressive TME. The incidence of the cancers for which conditions PD-1/PD-L1 can be used as monotherapy was approximately 1,060.1 thousand and 335.2 thousand in 2021 globally and in China, respectively, and is expected to increase to 1,306.5 thousand and 421.4 thousand in 2030 globally and in China, respectively. Compared to chemotherapy's average ORR of approximately 36% in various cancer indications, the addition of PD-1/PD-L1 inhibitor to chemotherapy can enhance the average ORR by approximately 14% for the treatment of those indications. In general, adding PD-1/PD-L1 inhibitors to other cancer agents (including chemotherapy, targeted therapy and other immunotherapy) can achieve an increase of approximately 16% in the average ORR in various cancers as compared to that of the other cancer agents. Since IMM2520 showed more potent antitumor effects than PD-1/PD-L1 inhibitor monotherapy in preclinical studies, IMM2520 in combination with other agents is expected to achieve improved treatment outcomes than PD-1/PD-L1 inhibitor-based combination therapies. However, macrophages are widely distributed in a broad range of tumor types and account for around 20% to 50% of cells in respective tumor tissues, presenting a huge market potential for our IMM2520. With the capability to activate macrophages and unleash their synergistic effects with T-cell activation response, IMM2520 may benefit patients who are previously not responsive to or have progressed after PD-1/PD-L1 inhibitors, thus capturing the vast worldwide market opportunities. According to Frost & Sullivan, as IMM2520 is expected to provide effective treatment for solid tumors with low response rates to PD-1/PD-L1 inhibitors it has the potential to treat a wide range of cancer indications with high macrophage infiltration, including NSCLC, SCLC, HCC, GC, HNSCC, CRC, ESCC, OC, prostate cancer, and pancreatic cancer.

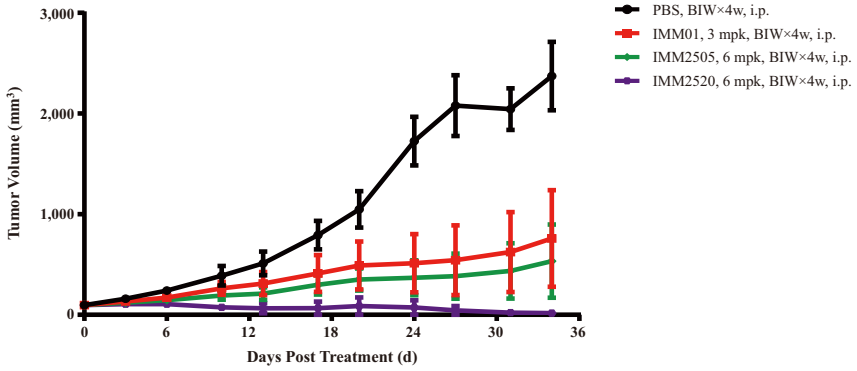
According to Frost & Sullivan, as of the Latest Practicable Date, a total of six CD47 and PD-1/PD-L1 bispecific molecules are under clinical development globally. Among those bispecific molecules, certain molecules connect the SIRPα fragment's N-terminal to the Fc end, which could interfere with CD47-binding epitope also located at its N-terminal, and further disrupt immune activation resulted from Fc-FcγR engagement. Further, due to the inevitable binding of CD47 antibodies to RBCs, several other bispecific molecules resort to an IgG4 Fc region with weak FcγR engagement. In contrast, only very few molecules preserve intact IgG1 Fc region with a better ability to engage Fc receptors and elicit stronger effector functions. IMM2520 adopts an ADCC-enhanced IgG1 Fc region and connects the C-terminal of the CD47-binding fragment to the heavy chain, allowing it to efficiently block CD47/SIRPα binding, and at the same time, activate stronger antitumor activity through potent ADCP and ADCC, thus better sensitizing TME to PD-L1 inhibitors, achieving a stronger synergistic effect.

Competitive Advantages

IMM2520 with an ADCC-enhanced IgG1 Fc can induce full macrophage activation and much improved ADCP/ADCC activity, thus maximizing the synergistic effects and significantly improving treatment outcomes of PD-1/PD-L1 inhibition, which results in stronger antitumor immune responses compared to most IgG4-based CD47 bispecific antibodies.

In addition, IMM2520 can simultaneously bind to the two targets on the same tumor cell to achieve potent synergistic effects, as compared to the combination therapy of CD47 antibodies and PD-1/PD-L1 inhibitors which require a higher dose for similar treatment efficacy. As illustrated in the diagram below, our *in vivo* efficacy studies demonstrated IMM2520's potent antitumor effects.

Efficacy Study in Colon Cancer (CT26) Syngeneic Mouse Model



Notes: (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 is identical to IMM01's and shows no binding activity with human RBCs *in vitro*. In addition, similar to our other CD47-based bispecific molecules, we designed IMM2520 to have a higher affinity for PD-L1 than CD47, allowing it to preferentially bind to PD-L1 and CD47 co-expressing tumor cells, rather than normal cells expressing CD47, thus minimizing "on-target, off-tumor" toxicity.

Clinical Development Plan

We have obtained IND approvals for IMM2520 from the NMPA in November 2022 and from the FDA in December 2022. We dosed the first patient for the Phase I clinical trial targeting a basket of solid tumor indications, with a particular focus on those solid tumors generally resistant or not sensitive to the currently available immunotherapies, such as CRC, GC, lung cancer and HNSCC, among others, in China in March 2023.

Licenses, Rights and Obligations

We are developing IMM2520 in-house and own the global rights to develop and commercialize IMM2520.

Material Communications

We had not received any regulatory agency's concerns or objections to our clinical development plans as of the Latest Practicable Date.

WE MAY NOT BE ABLE TO ULTIMATELY DEVELOP AND MARKET IMM2520 SUCCESSFULLY.

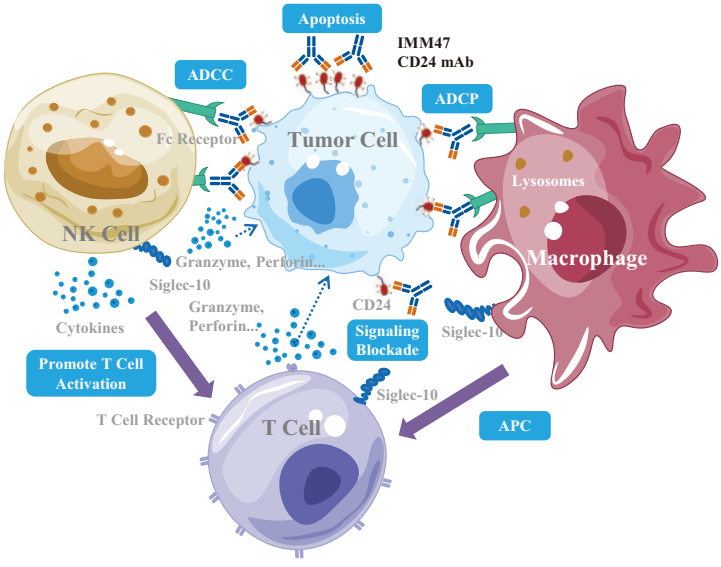
IMM47 (CD24 mAb)

IMM47 is a CD24-targeted humanized antibody we internally screened and developed with global first-in-class potential for the treatment of solid tumors. CD24 is widely expressed in numerous types of solid tumors, including BC, NSCLC, CRC, HCC, RCC and OC, and has been recognized as an important marker for poor prognosis of those cancers, presenting a huge market potential in a broad-spectrum application. According to Frost & Sullivan, there is no approved or

clinical-stage molecule targeting CD24 globally. We started the discovery research on CD24 as early as 2019, and have developed one innovative IND-enabling-stage molecule with potent *in vivo* efficacy, and multiple discovery- and preclinical-stage bispecific molecules. Recently, Pheast Therapeutics, led by Dr. Amira Barkal and Dr. Irving Weissman, the world's pioneer in CD47, revealed their move into the development of cancer therapies targeting CD24, which is expected to stir a new wave of enthusiasm for this novel next-generation immuno-oncology target across the global biopharmaceutical industry. However, the screening of monoclonal antibodies against CD24 is highly challenging due to the relatively weak immunogenicity resulting from its small extracellular domain. We have developed IMM47 and filed multiple patent applications. We expect to submit IND applications for IMM47 with the NMPA and the FDA in 2023, and subsequently initiate a Phase I clinical trial first in Australia in mid-2023 for the treatment of various solid tumor indications. Initiating a clinical trial in Australia first can help us to begin global clinical trials earlier and accelerate clinical validation of IMM47. Additionally, we believe Australian trial can generate valuable clinical data on ethnically diverse populations, thus enhancing our ability to pursue collaboration opportunities with global pharmaceutical companies.

CD24 interacts with its ligand, Siglec-10, on the surface of various immune cells, including macrophages, NK cells, T cells and B cells, leading to immune escape of tumor cells. With a high affinity for CD24, IMM47 is able to suppress the CD24/Siglec-10 inhibitory signals sent to macrophages, NK cells and T cells. With its ADCC-enhanced IgG1 Fc, IMM47 can potently activate macrophage and NK cell-immune responses through ADCP and ADCC. It has also been shown to significantly increase the amount of M1 macrophages in tumor tissues in our *in vivo* proof-of-concept studies. IMM47 can also activate and promote T-cell response likely through tumor antigen presentation by activated macrophages to T cells and direct blockade of CD24/Siglec-10 inhibitory signals. The following diagram illustrates the mechanism of action of IMM47:

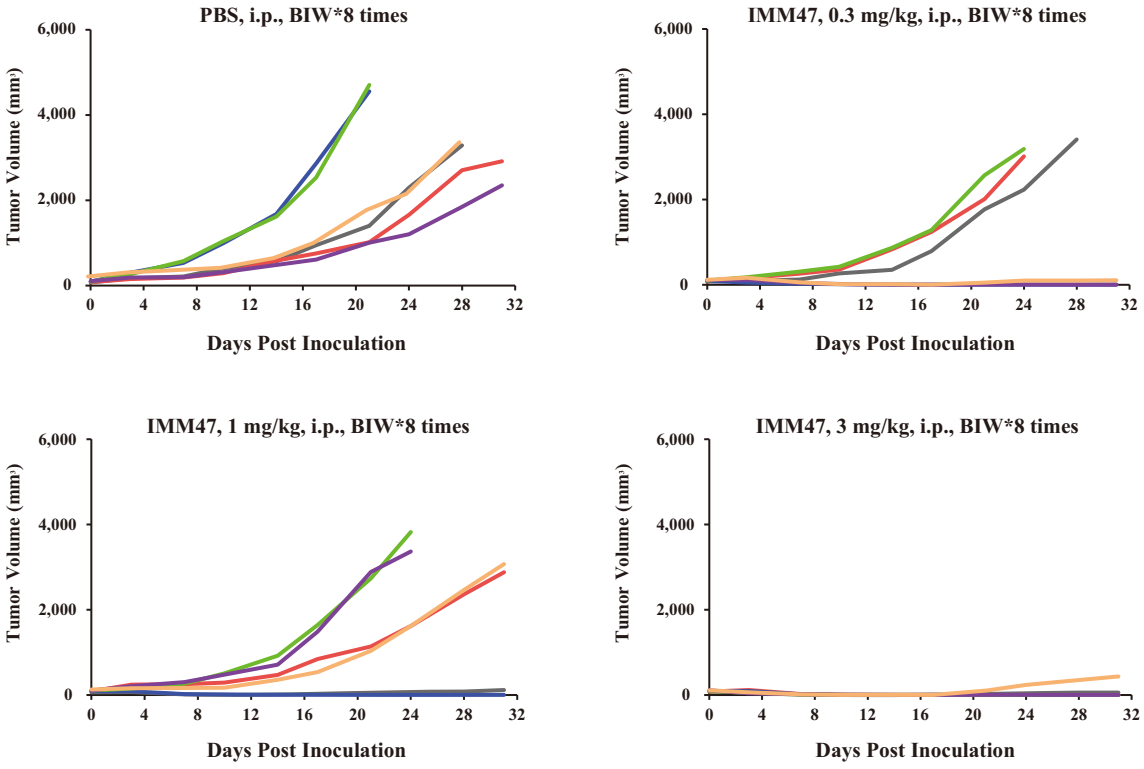
Mechanism of Action of IMM47



Source: Company Data

IMM47 has demonstrated compelling capabilities to kill tumor cells in our preclinical studies as illustrated in the diagram below. At the dose level of 3.0 mg/kg (~0.3 mg/kg human equivalent human dose), IMM47 successfully eradicated subcutaneously inoculated tumor cells in all six mice after three treatments in a colon cancer model, which demonstrated robust antitumor activity of IMM47 as monotherapy in solid tumor models.

Proof-of-Concept Study in Colon Cancer (MC38) Syngeneic Mouse Model

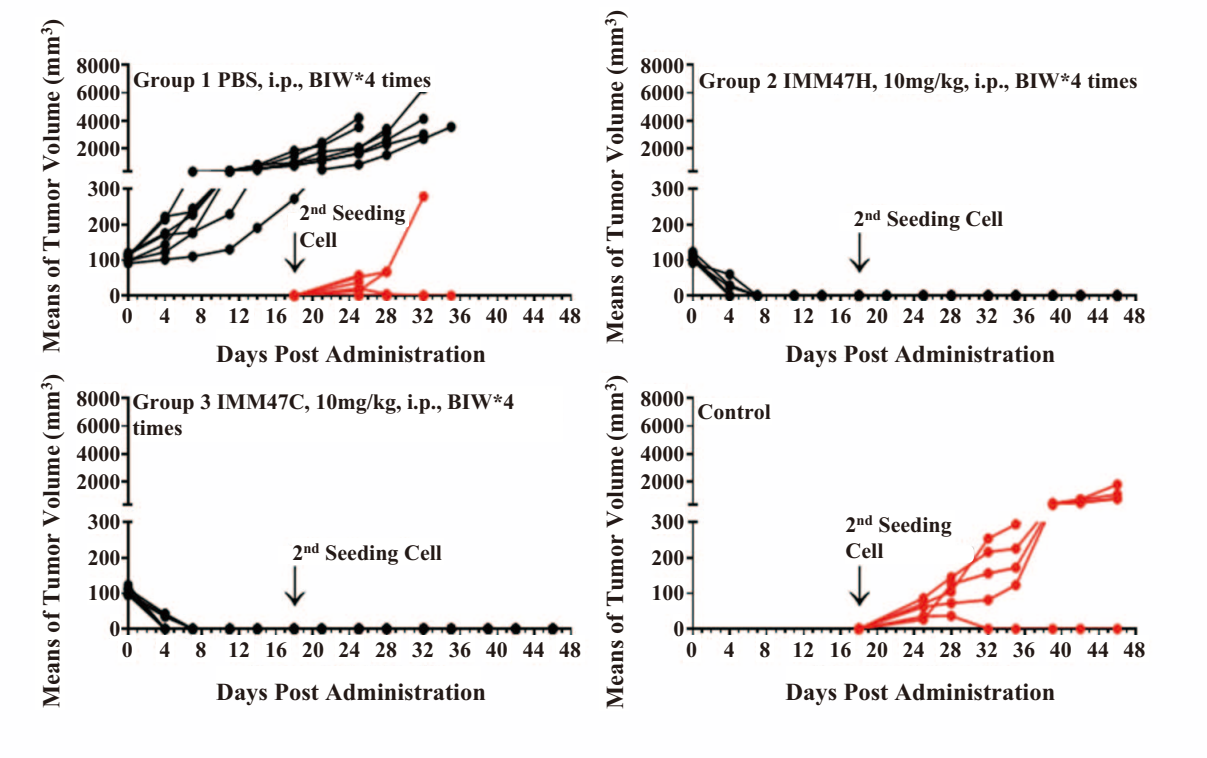


Notes: (1) Six mice per group were used in this study. (2) The colors of lines represent the different responses of the six mice in each group.

Source: Company Data

More intriguingly, IMM47C and IMM47H (both are earlier generations of IMM47) have demonstrated robust antitumor activities, leading to complete tumor eradication, with the ability to induce immunological memory against tumors in our *in vivo* preclinical studies. Mice treated with IMM47C and IMM47H established tumor-specific immune responses that prevented tumor growth even against re-inoculation of tumor cells.

Proof-of-Concept Study in Colon Cancer (MC38) Syngeneic Mouse Model

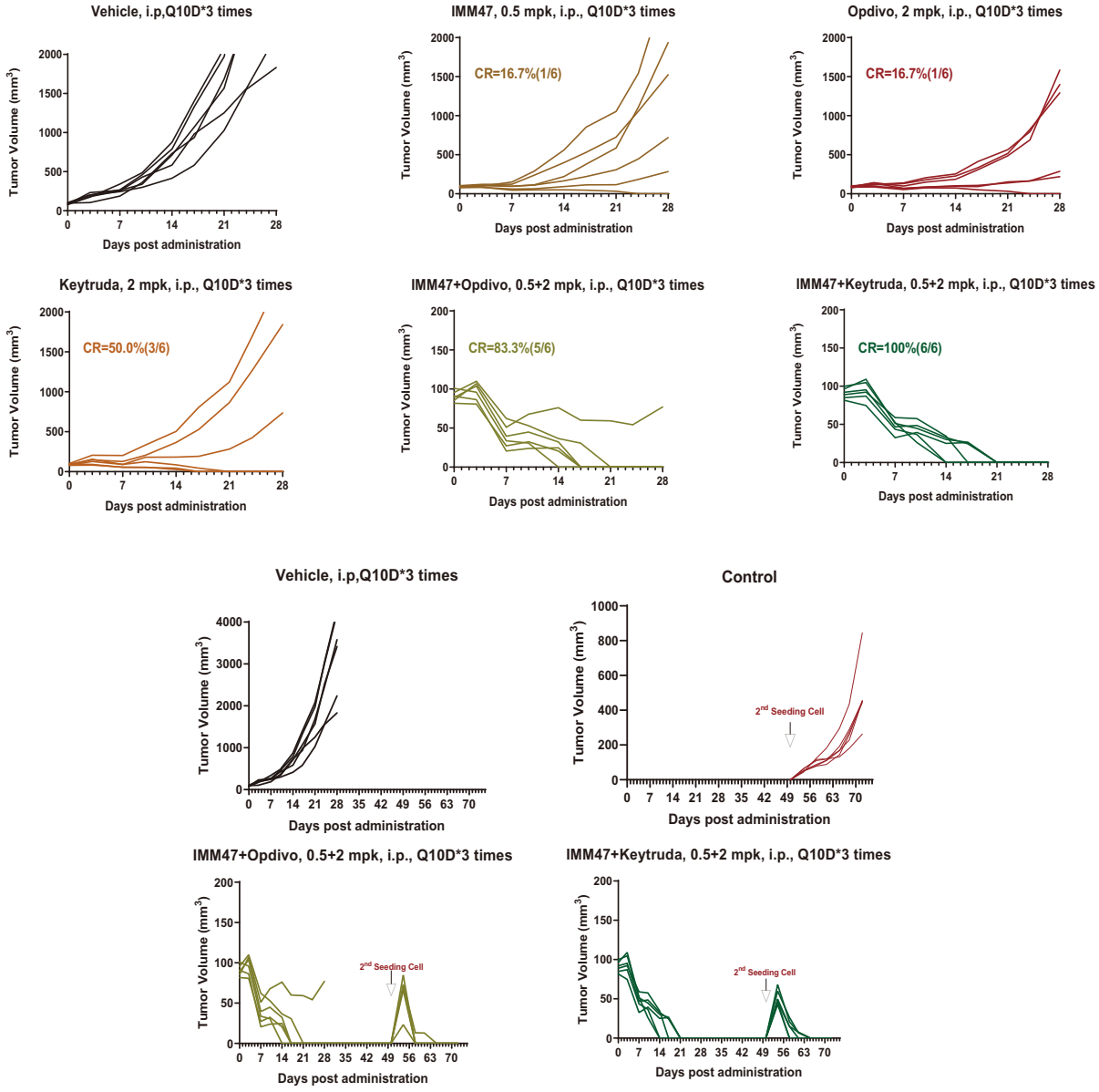


Notes: (1) IMM47C is a previous chimeric version of IMM47 and IMM47H is an earlier fully humanized version of IMM47. IMM47 revealed highly similar *in vitro* efficacy as IMM47C and IMM47H, and was eventually selected for further development; (2) Ten mice per group were used in the first seeding, with seven of the ten subsequently used in second inoculation for group 1, 2 and 3, and five used in the control group for the second seeding; (3) The colors of the lines represent the first and the second seeding respectively.

Source: Company Data

Targeting both innate and adaptive immunity, CD24-targeted drugs present a significant potential in treating a wide range of cancer indications. Given the all-around immune responses stimulated by blocking the CD24/Siglec-10 signaling pathway, they also suggest a strong synergistic potential with other immunotherapies, including PD-1/PD-L1 inhibitors. In fact, as illustrated in the diagrams below, our preclinical studies have shown that the combination of IMM47 and OPDIVO® or KEYTRUDA® can lead to a significant increase in response rates in our mouse model compared to using OPDIVO® or KEYTRUDA® alone. Furthermore, when we reinoculate the same cancer cells into mice pre-treated with IMM47 and PD-1 antibodies, tumor growth could be rapidly and completely eliminated, indicating the establishment of a tumor-specific immune response.

Proof-of-Concept Study in Colon Cancer (MC38) Syngeneic Mouse Model



As there is no approved or clinical-stage molecule targeting CD24 globally according to Frost & Sullivan, all of our CD24-targeted molecules, including CD24-targeted antibody and CD24-based bispecific molecules, are with global first-in-class potential. Although two drug candidates targeting Siglec-10 are currently under clinical development for the treatment of COVID-19, they are designed to bind with Siglec-10 to inhibit cytokine secretion and reduce COVID-19 induced immune over-reaction, exhibiting completely different mechanisms from the CD24-targeted therapies.

As of the Latest Practicable Date, with respect to IMM47, we owned one patent family, which includes one allowed patent application in the PRC, and one pending patent application in each of the U.S., the EU and Japan, and one pending PCT patent application which may enter various contracting states in the future.

WE MAY NOT BE ABLE TO ULTIMATELY DEVELOP AND MARKET IMM47 SUCCESSFULLY.

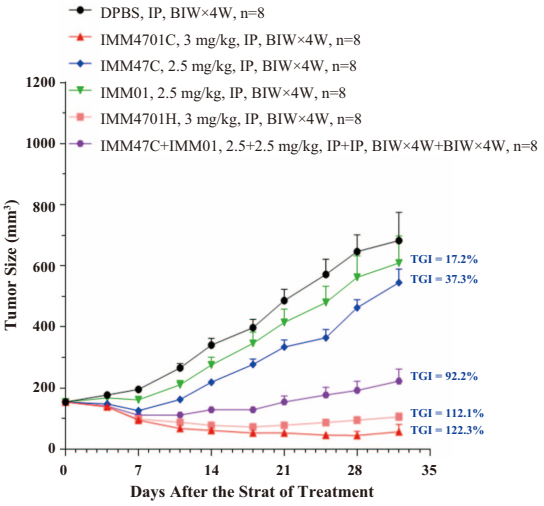
IMM4701 (CD47×CD24)

IMM4701, developed based on our mAb-Trap platform, is a bispecific molecule with the global first-in-class potential that targets both CD47 and CD24 for the treatment of solid tumors. According to Frost & Sullivan, there is no approved or clinical-stage CD24-targeted molecule globally. It has demonstrated promising antitumor activity in our *in vivo* efficacy studies. As of the Latest Practicable Date, with respect to IMM4701, we owned one patent family, which includes one allowed patent application in the PRC, and one patent application in each of the U.S., the EU and Japan, and one pending PCT patent application which may enter various contracting states in the future. Further leveraging the data observed from IMM47, we expect to file the IND applications for IMM4701 with the NMPA and the FDA for the treatment of solid tumors subsequently, and further seek collaboration opportunities with global pharmaceutical companies.

IMM4701 consists of an antibody targeting CD24 and the CD47-binding domain same as IMM01 connected to the N-terminal of the heavy chains, enabling it to adopt an ADCC-enhanced IgG1 Fc region. As simultaneous binding of CD47 and CD24 can activate key innate and adaptive immune responses and enhance the synergistic crosstalk between the two immune systems, IMM4701 demonstrates potent synergistic effects.

Our preclinical studies revealed strong and robust antitumor activities of IMM4701 against solid tumors. As shown in the diagram below, under MCF-7 xenograft TNBC model in SCID mice, IMM4701 resulted in reduced tumor size and exhibited strong potency at a low dose of 3 mg/kg (~0.3 mg/kg human equivalent dose).

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



Note: 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.

Source: Company Data

As discussed above, CD24-targeted molecules present strong potential in treating a wide range of cancer indications. Currently, our IMM4701 is the only reported CD24-targeted bispecific molecule under development for tumor treatment worldwide, which demonstrates the global first-in-class potential. In addition, leveraging the synergistic effects between innate and adaptive immunity, IMM4701 could also be a promising combination partner with PD-1/PD-L1 inhibitors.

WE MAY NOT BE ABLE TO ULTIMATELY DEVELOP AND MARKET IMM4701 SUCCESSFULLY.

IMM51 (IL-8 mAb)

We are developing IMM51, a monoclonal antibody that targets IL-8, for the treatment of solid tumors. IL-8 is a chemokine that mediates the inflammatory process and functions as a significant regulatory factor within the TME. A high level of IL-8 expression correlates with poor prognosis and short survival time of cancer patients. Given the effects of IL-8 signaling on a variety of effectors and downstream targets, suppressing IL-8 signaling may be an effective therapeutic intervention in targeting the TME. By blocking IL-8, IMM51 can potentially suppress tumor progression and metastasis, and sensitize cancer cells to PD-1/PD-L1 inhibition and other treatments. According to Frost & Sullivan, currently there is only one clinical-stage molecule targeting IL-8 worldwide, that is BMS-986253 being evaluated in a Phase I/II trial.

We are evaluating the toxicity and pharmacological effects of IMM51 in a number of *in vitro* and *in vivo* preclinical studies. Our *in vitro* studies have demonstrated IMM51's favorable binding activity and affinity, as well as its strong capability of blocking the binding of IL-8 with CXCR1 and CXCR2 receptors. We plan to continue to conduct preclinical studies to further evaluate IMM51, including *in vivo* studies.

WE MAY NOT BE ABLE TO ULTIMATELY DEVELOP AND MARKET IMM51 SUCCESSFULLY.

OUR ADAPTIVE IMMUNE CHECKPOINT-TARGETED CANDIDATES

We have also established a strong pipeline of multiple adaptive immune checkpoint-targeted drug candidates to capture the promising worldwide market opportunities for immunotherapies targeting adaptive immune checkpoints. These drug candidates have also shown significant promise when used in combination with our innate immune drug candidates. Our adaptive immune checkpoint-targeted candidates mainly include: (i) IMM2510 (VEGF×PD-L1), (ii) IMM27M (CTLA-4 mAb with enhanced ADCC activity), (iii) IMM40H (CD70 mAb), and (iv) multiple drug candidates in the discovery and preclinical stage, including IMM2518, a second-generation VEGF×PD-L1 bispecific molecule.

IMM2510 (VEGF×PD-L1)

IMM2510 is a bispecific molecule with the mAb-Trap structure that targets VEGF and PD-L1 for the treatment of solid tumors. Drugs targeting VEGF and PD-L1, which are clinically validated targets, have demonstrated potent synergistic effects when used in combination. By targeting VEGF and PD-L1, IMM2510 is able to activate T-cell tumor killing activities and simultaneously inhibit tumor angiogenesis and tumor growth. Moreover, IMM2510 can also activate NK cells and macrophages through Fc-mediated ADCC/ADCP activities. With respect to IMM2510, we owned one patent family, which includes one issued patent in the U.S., one issued patent in Japan, one allowed patent application in the PRC, one pending patent application in each of the EU and the U.S., and one PCT patent application which has entered national phases, as of the Latest Practicable Date.

Mechanism of Action

Tumor cells expressing PD-L1 can bind to PD-1 on the surface of T cells to evade T-cell attacks. PD-L1 antibodies could block the PD-1/PD-L1 pathway and thus activate T cells, which has demonstrated robust antitumor activities in a broad range of solid tumors. VEGF, as a dynamic angiogenic factor, is up-regulated in many tumor indications, which contributes to angiogenesis and tumor growth. Inhibiting VEGF can reduce VEGF-mediated tumor angiogenesis and inhibit immune suppression, thus promoting the activation of T-cell immune responses.

The powerful synergistic effect between these two targets has been evidenced by anti-PD-1/PD-L1 and anti-VEGF combinations approved for an array of cancer indications, including RCC, NSCLC, HCC and CC. Anti-PD-1/PD-L1 and anti-VEGF combinations, such as TECENTRIQ® (atezolizumab) and AVASTIN® (bevacizumab), are recommended as first-line treatment for late-stage HCC. We connect VEGFR1-D2 (the second extracellular domain of VEGFR1) to the N-terminal of the heavy chain of a PD-L1 antibody with an ADCC-enhanced IgG1 Fc fragment. Through ADCC-enhanced IgG1 Fc, IMM2510 can further activate NK cells and macrophages through strengthened Fc-mediated ADCC/ADCP activities to promote innate and subsequent adaptive immune responses.

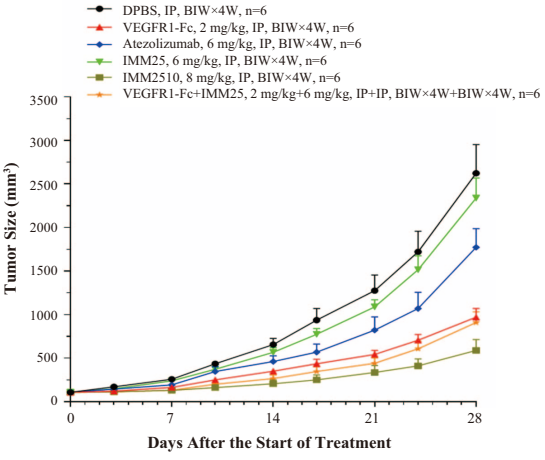
Market Opportunities and Competition

We believe there is a significant market opportunity for IMM2510 as a bispecific molecule targeting PD-L1 and VEGF which combines multiple mechanisms. Currently, PD-1/PD-L1 inhibitors or VEGF blockers have been approved for many cancer indications, and the combination use of these two have also demonstrated robust efficacy in clinical settings for the treatment of a wide range of cancers, indicating huge market opportunities for our IMM2510. For example, PD-1/PD-L1 inhibitors have been approved in BC, HCC, RCC, GC, NSCLC, SCLC, and EC. VEGF blocking agents have been approved in CRC, HCC, NSCLC, GC, RCC, OC and CC. In comparison to the combination therapies, a well-designed bispecific molecule has a competitive edge due to the synergistic effects between the two targets and much lower costs when used as a single agent, having the potential to address the significant market opportunities.

Competitive Advantages

According to Frost & Sullivan, there are currently four bispecific molecules simultaneously targeting VEGF and PD-L1 in the global pipeline, two of which have no active IgG1 Fc fragment with potent effector function. Through angiogenesis inhibition and T-cell activation, IMM2510 with ADCC-enhanced IgG1 Fc can modulate the TME and lead to substantially improved therapeutic efficacy. As illustrated by the below diagram, our *in vivo* efficacy studies showed that IMM2510 had a better efficacy profile than the VEGF or PD-L1 antibodies used as a single agent or in combination. In addition, compared to combination therapies, IMM2510 presents a tremendous competitive advantage with respect to affordability for patients.

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



Source: Company Data

The initial results of the Phase I clinical trial have revealed a promising efficacy signal. Our preliminary clinical data as of February 15, 2023 has demonstrated that IMM2510 was safe and tolerable up to 10.0 mg/kg in patients with advanced solid tumors, and we are currently evaluating patients for 10.0 mg/kg dose cohort. Among the two evaluable NSCLC patients in the trial so far, we have observed PRs in both patients with best tumor shrinkage response of 46% and 35% respectively. Dose escalation is still ongoing.

Clinical Development Plan

We commenced the Phase I dose-escalation trial for IMM2510 in China in August 2021 for the treatment of a variety of advanced solid tumors, including but not limited to, HCC, RCC, GC, NSCLC and STS. We expect to complete the Phase I clinical trial in mid-2023 and initiate the Phase II clinical trial in 2023 in China.

Licenses, Rights and Obligations

We are developing IMM2510 in-house and own the global rights to develop and commercialize IMM2510.

Material Communications

We had not received any regulatory agency's concerns or objections to our clinical development plans as of the Latest Practicable Date.

WE MAY NOT BE ABLE TO ULTIMATELY DEVELOP AND MARKET IMM2510 SUCCESSFULLY.

IMM27M (CTLA-4 ADCC-enhanced mAb)

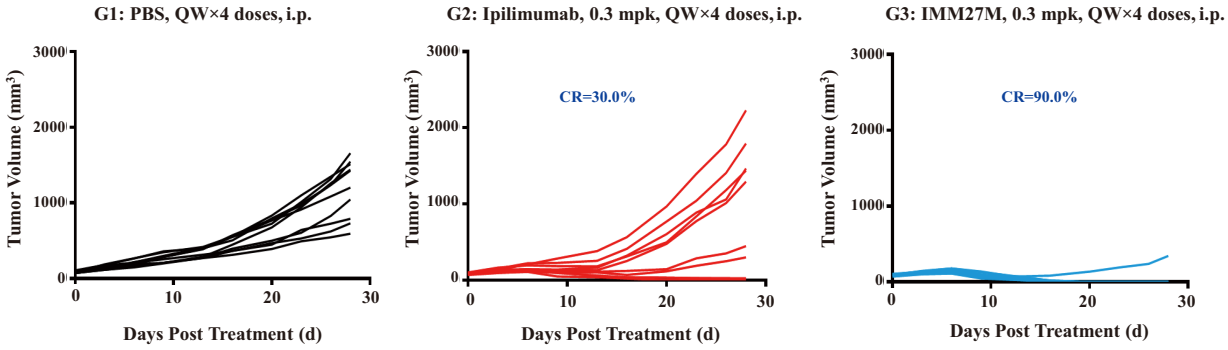
IMM27M is a new generation CTLA-4 antibody with enhanced ADCC activity through genetic engineering modification. We have commenced the Phase I clinical trial targeting solid tumors, with the first patient dosed in June 2022. We expect to complete this trial in mid-2023.

As a protein receptor that can be found on the activated T cells, CTLA-4 can downregulate immune responses by binding to CD80/CD86, its natural ligands found on the surface of antigen presenting cells, delivering inhibitory signal and thus suppressing T-cell immune function. CTLA-4 antibodies can block the interaction between CTLA-4 and CD80/CD86, and thus enhance immune responses of T cells to tumor antigens. Though CTLA-4 is a clinically validated target, so far there is only one approved product globally.

Recent studies on CTLA-4 have further revealed that its key mechanism for tumor suppression is T_{reg} depletion. CTLA-4 antibodies deplete T_{reg} cells in the TME, inducing immune attacks against tumor cells. The currently approved CTLA-4 antibody with unmodified Fc shows limited efficacy, thus requiring a high dosage to achieve desirable efficacy which leads to serious safety issues. We thus designed IMM27M with enhanced ADCC modification through genetic engineering. With augmented ADCC activities, IMM27M is able to induce enhanced immune responses targeting CTLA-4 overexpressed T_{reg} cells and promote T_{reg} depletion, thus improving T-cell antitumor response to kill tumor cells.

As expected, our *in vivo* efficacy studies demonstrated that IMM27M could induce a significantly stronger antitumor activity than ipilimumab and result in complete tumor remission even at a dose as low as 0.3 mg/kg (~0.03 mg/kg human equivalent dose), as illustrated in the diagrams below:

Efficacy Study in Colon Cancer (MC38) Syngeneic Mouse Model



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

We have commenced the Phase I clinical trial targeting solid tumors, with the first patient dosed in June 2022. We had enrolled 15 patients as of February 10, 2023, and we are currently enrolling patients for the sixth cohort of 5.0 mg/kg. The preliminary data demonstrates that IMM27M is safe and well tolerated up to 3.0 mg/kg. We have observed 4 SDs in this trial so far, among whom one patient with breast carcinoma who had six lines of prior treatment has achieved SD with tumor shrinkage of 28.8% at 3.0 mg/kg, and one patient with metastatic melanoma has achieved SD with tumor shrinkage of 22.9% at 2.0 mg/kg. We expect to complete this trial in mid-2023. In addition to its strong efficacy as a monotherapy, IMM27M could be used in combination with PD-1 antibodies targeting a wide range of solid tumor indications, as IMM27M could promote T_{reg} depletion and T-cell activation, inducing overall immune responses to fight tumor cells. Moreover, we received an IND approval from the NMPA for a Phase Ib/II study to evaluate the combination of IMM27M and a PD-1 antibody for the treatment of advanced solid tumors, such as RCC, NSCLC, GC and TC, in March 2023. We may initiate clinical trials or explore collaboration opportunities for this combination therapy.

WE MAY NOT BE ABLE TO ULTIMATELY DEVELOP AND MARKET IMM27M SUCCESSFULLY.

IMM40H (CD70 mAb)

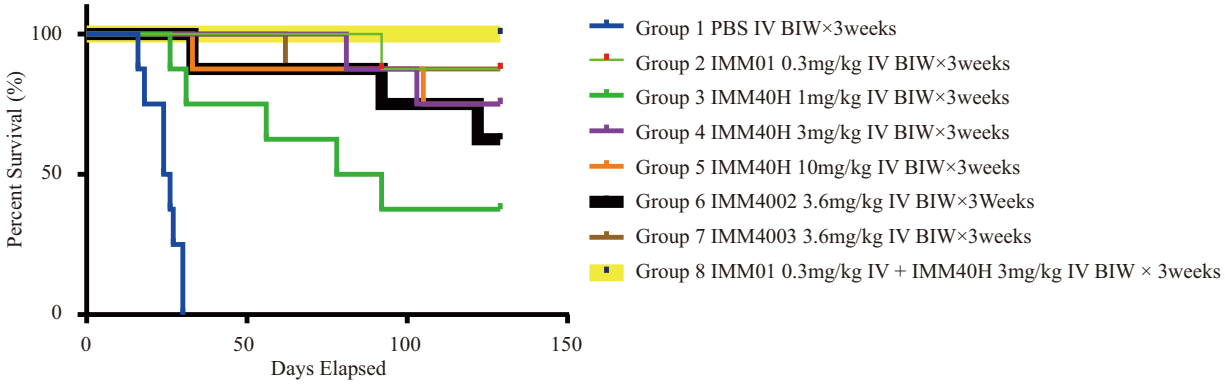
IMM40H is a humanized IgG1 CD70 monoclonal antibody, which demonstrates robust tumor-killing properties and strong synergy when combined with IMM01. CD70 could be targeted for the treatment of liquid and solid tumors. We have obtained IND approvals for IMM40H from the NMPA and the FDA in August 2022, and may initiate Phase I clinical studies or pursue potential collaboration opportunities. We are one of the first few companies to develop molecules targeting CD70 globally. With respect to IMM40H, we owned one patent family, which includes one allowed patent application in the U.S., one allowed patent application in the PRC, one patent application in each of the EU and Japan, and one pending PCT patent application which may enter various contracting states in the future, as of the Latest Practicable Date.

A significant level of CD70 can be detected in various types of tumor tissues and CD27 is expressed on T_{reg} cells. The interaction between CD70 and CD27 can stimulate the proliferation and survival of cancer cells and increase the level of soluble CD27, which is associated with a low survival rate in patients with lymphoma and certain solid tumors. IMM40H can bind with CD70 on

tumor tissues and obstruct the activation and proliferation of T_{reg} cells through the inhibition of CD70-CD27 signaling. As evidenced by *in vitro* cell-based assay, IMM40H has shown a much stronger CD70-binding affinity than cusatuzumab (a CD70-targeted antibody developed by Argenx and currently in Phase II stage), which allows IMM40H to block the interaction of CD70 and CD27 more effectively. Moreover, IMM40H has also demonstrated potent ADCC, CDC and ADCP activity, resulting in strong immune attacks on tumor cells and potentially potent therapeutic efficacy. Our preclinical data also suggests a favorable safety profile of IMM40H. According to Frost & Sullivan, CD70 could potentially be an effective therapeutic target for the treatment of CD70-positive tumors, including CD70-positive lymphoma, RCC, NSCLC, HNSCC and OC.

IMM40H has exhibited strong antitumor activity in our preclinical studies. Additionally, strong synergism between IMM01 and IMM40H has been observed *in vivo*. As shown in the diagram below, the combination therapy of IMM01 and IMM40H has demonstrated strong treatment efficacy:

Efficacy Study in Lymphoma (Raji) Orthotopic Mouse Model



Note: Eight mice per group were used in this study.
Source: Company Data

WE MAY NOT BE ABLE TO ULTIMATELY DEVELOP AND MARKET IMM40H SUCCESSFULLY.

OUR PLATFORM

We have established an integrated platform encompassing three main functions: (i) drug discovery and preclinical development, (ii) CMC and pilot manufacturing, and (iii) clinical development. Leveraging the collaboration among different functional groups, our platform empowers us with robust research and development capabilities, allowing us to efficiently discover and advance the development of next-generation immunotherapies towards commercialization. As a result, we have constructed a comprehensive pipeline consisting of over ten innovative drug candidates targeting both innate and adaptive immune systems, with eight ongoing clinical programs.

Drug Discovery and Preclinical Development

Led by Dr. Tian and Mr. Song Li, our dedicated drug discovery team consisted of 19 experienced and capable members as of the Latest Practicable Date. This team is responsible for, among others, target screening, molecule (including bispecific molecule) design, optimization, validation and development, cell-line development, and lab scale test process development. Dr. Tian and Dr. Deqiang Jing are the inventors of IMM01, our Core Product. Guided by Dr. Tian, and with joint efforts of other members of our drug discovery team, Mr. Song Li (vice president of research and development), Mr. Ruliang Zhang (deputy general manager and senior vice

president), Ms. Wei Zhang (director of up-stream processing), Mr. Xiaoping Tu (director of downstream processing), Ms. Li Zhang (director of quality control), Ms. Fengli Huang (director of quality assurance) and Mr. Dianze Chen (assistant director of research and development), further conducted the preclinical development of IMM01. Dr. Tian and Mr. Song Li are the inventors of all of our key products, IMM0306, IMM2902 and IMM2520, and are also responsible for the preclinical development of those drug candidates together with Mr. Ruliang Zhang, Ms. Wei Zhang, Mr. Xiaoping Tu, Ms. Li Zhang, Ms. Fengli Huang, Mr. Dianze Chen and other members of our drug discovery team.

Our solid drug discovery and preclinical platform includes advanced hybridoma technology, high-throughput screening, strong immunoassay and bioassay technology, and a proprietary bispecific mAb-Trap platform. These integrated platforms allow us to efficiently conduct screening for lead compounds and druggability analysis. Our established preclinical development function enables us to perform studies concerning proof-of-concept *in vivo* efficacy, preclinical PK and pharmacodynamic (PD), and toxicity in animals. Leveraging our strong drug discovery and preclinical development capabilities, we are developing over ten drug candidates at various stages. These in-house developed drug candidates all have the potential to be either first-in-class or best-in-class drugs if successfully advanced to the market:

- *mAb-Trap bispecific platform:* Guided by our insights in tumor biology and immunology and our "DbD" concept, we have built the mAb-Trap bispecific platform, which is best suited for the targets we have selected, to effectively facilitate our science-driven drug design and development. Leveraging this mAb-Trap platform, we have constructed a number of bispecific molecules and four of them (i.e., IMM0306, IMM2902, IMM2510 and IMM2520) have entered into clinical development stage. The bispecific molecules developed based on this platform have a symmetric structure, akin to that of native antibodies, allowing for ease of manufacturing, product stability, higher titer and protein yield. This structure makes the CMC process and production by standard antibody manufacturing techniques more feasible. The average protein yield for IMM0306, IMM2902, and IMM2520 ranges from 3.8g/L to 4.6g/L, much higher than the industry average for bispecific molecules of 1.0g/L to 3.0g/L.

- *hybridoma technology:* With proprietary hybridoma technology and know-how, we can effectively accomplish the immunization of mice with particular target antigens and efficiently identify and optimize antibody fragments with higher specificity, affinity and other required properties for respective targets. We are currently using this hybridoma technology to screen therapeutic monoclonal antibodies for several new targets, for which no drug has yet been approved globally;

- *high-throughput screening:* Utilizing our high-throughput screening technology, we have identified molecules that have desirable characteristics for further cost-efficient development. This allows us to rapidly advance our assets to the preclinical and clinical evaluation stage and accelerate the drug development process. IMM40H and IMM47 are two excellent examples of using our hybridoma technology and high-throughput screening for innovative antibody drug development;

- *immunoassay and bioassay technology:* Our well-established comprehensive immunoassay and bioassay technology includes, among others, an assay of ADCC, CDC, and ADCP, Jurkat-CVR (Chimeric VEGF Receptor) cell line used for bioassay of VEGF/VEGFR-targeted drug development, Jurkat-CPR (Chimeric PD-1 Receptor) cell line used for bioassay of PD-1/PD-L1 antibody drug development, and Jurkat-CSR (Chimeric SIRPα Receptor) cell line used for bioassay of CD47/SIRPα targeted drug development, immunoassay of receptor occupancy, cytokine release assay, antibody-induced receptor internalization and signal transduction assay. These in-house developed technologies allow us to screen drug candidates effectively and precisely.

CMC and Pilot Manufacturing

Our CMC and regulatory affairs team, consisting of 45 members as of the Latest Practicable Date, is responsible for, among other relevant functions, cell line development, upstream and downstream process development, formulation development, analytical method development and validation, and pilot manufacturing. For cell line development, we developed a CHO-K1 host cell line with glutamine synthetase gene knocked out via gene editing. The resulting host cell line, named CHOK1-GSKO, has passed the inspections and audits by qualified third parties, was certified to be compliant with GMP standards, and has been validated for use in multiple clinical programs. We have also developed and optimized the cell line screening techniques which significantly help shorten the time for the development of stable expression cell lines with much higher titers.

We have established substantial pilot manufacturing capabilities with the scale of 450L. With our GE and Thermo Fisher single-use mammalian cell bioreactors, AKTA™ Process protein chromatography purification system, quality analysis platform and quality assurance system in accordance with GMP requirements, we are able to manufacture high-quality drug candidates in-house in an efficient and cost-effective manner.

Considering the benefits of having our own self-sufficient manufacturing facilities, we intend to strategically expand our GMP-compliant manufacturing capacity, while improving efficiency and cost-effectiveness. We have already commenced the construction of our new manufacturing facility occupying a site area of approximately 28.7 thousand square meters in Zhangjiang Science City, Pudong New Area of Shanghai, which is designed to meet the stringent cGMP standards. We plan to complete the first stage of construction by 2025, and plan to commence the second stage of construction depending on the schedule of regulatory approval and sales ramp-up of our drug portfolio in the future. Once completed, the manufacturing facility will provide us with an additional 12,000L manufacturing capacity.

We currently also collaborate with CMOs/CDMOs for the manufacturing of a portion of our drug candidates for preclinical studies and clinical trials. We have adopted procedures to ensure that production qualifications, facilities and processes of CMOs/CDMOs comply with the relevant regulatory requirements and our internal guidelines. We selected our CMOs/CDMOs by carefully reviewing and considering various factors, including their qualifications, expertise, production capacity, geographic proximity, reputation and costs.

Clinical Development

Our capable clinical development function is responsible for clinical trial design and implementation, as well as translational medicine. We also engage CROs and consultants in China and the U.S. to support our clinical trials. We have established long-standing partnerships with hospitals and principal investigators throughout China and the U.S., which enables us to conduct multiple large-scale clinical trials. In addition, our medical function allows us to analyze preclinical and clinical data to guide our clinical strategy, as well as the design and timely adjustments of clinical development plans.

As of the Latest Practicable Date, our clinical development team was comprised of 45 members, among whom 11 hold doctorate degrees or are medical doctors and 12 hold master's degrees. This team is led by Dr. Qiying Lu, who has around 20 years of experience in clinical practice and innovative oncology drug development with multinational pharmaceutical companies and biotechnology companies, including GlaxoSmithKline, AstraZeneca, and Pfizer, and Dr. Frank Xiaodong Gan, who brings us over 25 years of experience in preclinical and clinical development in academia and the biopharmaceutical industry and had led numerous global clinical development of various drug candidates for multinational pharmaceutical companies, including Merck & Co., Bristol Myers Squibb, Eli Lilly and Janssen. During the Track Record Period, the operation of our

clinical programs had been directly managed and driven by our clinical operation directors, supported by our project manager and clinical research associates for each clinical program. We also have a dedicated clinical medical team in charge for formulating trial protocol, reviewing clinical data, as well as adjusting and adapting clinical development plan in a timely manner based on signals and data observed in clinical trials. The clinical operation directors and clinical medical team collectively reported to the vice president of clinical research, prior to the joining of Dr. Lu. Before Dr. Lu joined us, we had completed the Phase I trial of IMM01 monotherapy, and we were continuously advancing a number of clinical trials of our drug candidates in China, including the Phase Ib/II trial of IMM01 in combination with azacitidine, the Phase I trial of IMM0306, the Phase I trial of IMM2902, and the Phase I trial of IMM2510. The leadership of Dr. Lu and Dr. Gan further strengthen the capabilities of our clinical development function, propelling multiple drug candidates into next clinical stage in China and/or the U.S., including the Phase II trial of IMM01 in combination with azacitidine, the Phase Ib/II trial of IMM01 in combination with tislelizumab, the Phase I trial of IMM2902 in the U.S., and the Phase I trials of IMM27M and IMM2520, and the Phase II trial of IMM0306. The leadership team of our clinical development department is generally responsible for the formulation of the clinical strategy and supervision of overall clinical development of our Core Product and Key Products, and our clinical operation directors are responsible for carrying out the execution of respective clinical programs of our drug candidates.

Our strong clinical development team is extensively involved in substantially all stages of our clinical trials, including trial protocol design, selection of investigators and sites, and management of our clinical trial programs. We design protocols and clinical trials in-house to maintain clinical operational excellence. We utilize adaptive clinical trial design to achieve efficiency in drug development processes and potentially accelerate approvals for our drug candidates. Leveraging extensive knowledge and experience in managing clinical trials, our clinical development experts are particularly good at identifying unique therapeutic opportunities for our drug candidates based on the differentiating properties observed in the trials and improving their clinical plans accordingly.

We employ in-house medical research team to monitor treatment response in clinical trials, analyze clinical results, timely adapt clinical trial designs, and potentially discover predictive biomarkers to guide the design and execution of clinical studies. Our medical function allows us to validate mechanisms of action and drug resistance mechanisms, increasing the success rate of our clinical trials.

As is customary in the pharmaceutical industry, we use CROs to conduct and support our preclinical studies and clinical trials under our close supervision and overall management. We have selected CROs weighing various factors, such as their qualifications, expertise, experience, reputation and costs. Our cooperative relationship with CROs is based on specific projects. The preclinical CROs generally provide services related to preclinical toxicity and safety evaluations (such as animal studies), and *in vivo* pharmacology and PK studies under our study design. The clinical CROs mainly provide us with assistance in our conduct of clinical trials, including trial preparation, clinical monitoring, medical monitoring, and project management. We have exploited the CROs' professional expertise to facilitate optimal site selection, timely patient recruitment and efficient conduct of complex clinical trials. We carefully supervise the CROs to ensure that they perform their duties in a manner that complies with our protocols and applicable laws and protects the data integrity.

Below is a summary of the key terms of an agreement we typically enter into with our CROs:

- Services. The CRO provides the high-quality services to us, including the implementation and management of a preclinical or clinical research project as specified in the agreement.
- Term. The CRO is required to perform its services and complete the preclinical or clinical research project within the prescribed time limit set out in each agreement.
- Payments. We are required to make payments to the CRO in accordance with the payment schedule agreed by the parties.
- Intellectual property rights. We own all intellectual property rights arising from the preclinical or clinical research project.
- Risk allocation. Each party should indemnify the other party for losses caused by its fault or gross negligence.

COMMERCIALIZATION

We plan to recruit capable marketing professionals and develop our capabilities of commercialization. As our current pipeline of drug candidates comes to the market, we will build up our commercialization and distribution capabilities as well as seek commercialization partnerships with other pharmaceutical industry players to maximize the reach of our product offering and expedite market acceptance of our products.

COLLABORATION AGREEMENT

Collaboration with Sunshine Guojian

On January 18, 2021, we entered into a joint drug development collaboration agreement with Sunshine Guojian, an innovative biopharmaceutical company in China. Pursuant to this agreement, the parties will collaborate to conduct clinical studies to evaluate the combination therapy of a HER2 monoclonal antibody inetetamab and IMM01 for the treatment of HER2-positive solid tumors in mainland China (excluding Hong Kong, Macau and Taiwan). Given the preclinical effects of the combination of CD47 and HER2 targeted therapies, this collaboration allows us to further expand IMM01's market in a cost-efficient manner by leveraging the funds and resources of Sunshine Guojian. During the Track Record Period and up to the Latest Practical Date, except as disclosed in the paragraphs headed "Directors, Supervisors and Senior Management" in the document and a limited amount of CDMO services provided by Sunshine Guojian, there were not any past or present relationships or dealings (including family, business, employment, trust, financing or otherwise) between the Company and Sunshine Guojian, their respective substantial shareholders, directors or senior management, or any of their respective associates.

Pursuant to the agreement, Sunshine Guojian is responsible for the design of the clinical study protocol, coordination with the CROs and regulatory filings related to each phase of clinical studies. Sunshine Guojian is entitled to determine potential indications for the clinical development of this combination therapy. During the term of this agreement, we will not conduct, or supply drugs for, any clinical study of IMM01 in combination with other HER2 antibodies for the indications selected by Sunshine Guojian in mainland China, unless the collaborated clinical studies of this combination therapy for such selected indication fail. Sunshine Guojian has final decision-making authority with respect to all material matters in relation to the clinical studies, including but not limited to, the preparation and modification of the clinical trial protocols, of this combination therapy for selected indications.

Each party will supply its product for the purpose of clinical studies at its own cost. All costs incurred in the clinical studies in mainland China will be borne by Sunshine Guojian, except for certain costs to be borne by us as provided in the agreement, which include the cost of supplying IMM01, the costs of assigning our own representatives to participate in the clinical development and regulatory communications, and providing related technology support. Upon Sunshine Guojian's request, we may execute clinical studies evaluating this combination therapy with the costs and expenses borne by Sunshine Guojian.

Each party retains ownership of intellectual property rights in its own product. Any new data generated and intellectual property rights (including patents) arising from collaborated clinical studies will be jointly owned by both parties, and can be used free of charge in manufacturing and commercialization activities of each party. If we grant licenses for the use of such new data and intellectual property rights arising from collaborated clinical studies to third parties, we will pay 70% of our gains from the relevant licensing arrangement to Sunshine Guojian, since both parties agree that it is commercially reasonable for Sunshine Guojian to enjoy the majority of fees from the licensing arrangement to the extent related to data and IP generated from the collaborated clinical trials as Sunshine Guojian carries the burden of financing those clinical trials. We retain full rights to commercialize IMM01 worldwide. Except for the aforementioned costs and fees arrangements, there are no upfront, milestone or other payment arrangements under this agreement.

This agreement, unless terminated earlier, will continue until the completion of the clinical studies for this combination therapy. This agreement can be terminated upon (i) mutual consent, or (ii) written notice by either party in the event of the other party's uncured breach. With respect to any dispute that cannot be resolved by negotiation, either party can submit such dispute to binding and final arbitration.

This agreement was negotiated and approved on an arm's length basis and determined based on normal and fair commercial terms considering the therapeutic potential of this combination therapy, the uniqueness of IMM01 and the potential economic gain for each party.

INTELLECTUAL PROPERTY

Our intellectual property is an important component of our business. We rely on a combination of patent and other intellectual property, as well as confidentiality procedures, non-disclosure agreements, employee non-disclosure and invention assignment agreements, and other contractual restrictions to establish and protect our commercially important technologies, inventions and know-how related to our business. While we believe our intellectual property rights and applications in the aggregate are important to our competitive position, no single intellectual property right or application is material to our business as a whole.

As of the Latest Practicable Date, we owned (i) four issued patents and five allowed patent applications in the PRC, (ii) six issued patents and two allowed patent applications in the U.S., (iii) nine issued patents and two allowed patent applications in other jurisdictions, and (iv) 29 patent applications, including two pending PRC patent applications and one PRC patent application filed as a priority application, one pending Hong Kong patent application, six pending U.S. patent applications, six PCT patent applications which have entered into national phases, four pending PCT patent applications which may enter various contracting states in the future, and nine pending applications in other jurisdictions. As reviewed and advised by our legal advisor as to intellectual property laws, material aspects (e.g. constructs, sequences or indications under development) of the Company's Core Product and Key Products can be covered by certain granted patents or pending patent applications in the PRC and the U.S. Furthermore, the Company has allowed or pending patent applications in the PRC and the U.S. to cover material aspects (e.g. constructs, sequences or indications under development) of relevant drug candidates. Please refer to the paragraph headed "Statutory and General Information — Further Information about the Business of our Company — Our Material Intellectual Property Rights" in Appendix VI to this document for further information of our material intellectual property rights.

The following table sets forth the portfolio of patents and patent applications material to our business operations as of the Latest Practicable Date (for each drug candidate, all the counterparts in its related patent family are set forth in the following table):

Drug Candidate	Title of Invention	Application Number	Filing Date[1]	Inventors	Jurisdiction	Status[2]	Grant Date	Estimated Expiration Date
IMM01 (SIRPα-Fc)	Novel Recombinant Bi-functional Fusion Proteins, Preparation and Use Thereof (SIRPαD1-Fc)	201510203619.7	April 24, 2015	Lijuan Liu, Deqiang Jing, Hua Wang[3]	PRC	Granted	January 15, 2019	April 24, 2035
		16/905,262	November 16, 2015	Wenzhi Tian, Deqiang Jing	U.S.	Pending	N/A	N/A
		17/412,445	November 16, 2015		U.S.	Pending	N/A	N/A
		15/566,724	November 16, 2015		U.S.[4]	Granted	October 13, 2020	April 14, 2036
		2017-552177	November 16, 2015		Japan	Granted	March 27, 2020	November 16, 2035
		PCT/CN2015/094739	November 16, 2015		PCT	Entered national phase	N/A	N/A
		15889744.7	November 16, 2015		EU	Allowed	N/A	N/A

Notes:

(1) As to the patent family relating to IMM01, "filing date" of the U.S., EU and Japanese counterparts denotes the filing date of the PCT application in this patent family, which is typically earlier than the actual submission date of each counterpart application in respective jurisdictions and its substantive examination;

(2) "Entered national phase" denotes the status of a PCT patent application that has entered the process whereby an applicant files one or more patent applications in one or more individual jurisdictions or countries of interest. "Allowed" denotes the status of a patent application that has been examined and determined to have met all statutory requirements for patent grant in applicable jurisdictions and for which a notice of allowance has been sent to the applicant, but is still waiting for completion of the procedures for patent grant such as paying the official fees for the patent grant by the applicant and publication of the granted patent by the applicable patent examination authority;

(3) Our IMM01 was discovered, designed and developed by Dr. Deqiang Jing and Dr. Wenzhi Tian, both of whom are currently key R&D personnel of the Company, when Dr. Jing was a consultant at Shanghai Hanyu Biopharmaceuticals Co., Ltd (上海翰謦生物科技有限公司, "**Hanyu**" and Dr. Tian worked at Huabo Biopharm (Shanghai) Co., Ltd. (華博生物醫藥技術(上海)有限公司) ("**Huabo Biopharm**", respectively) in 2014. Dr. Jing, our senior director in the clinical department, was engaged as a consultant by Hanyu in February 2014. Dr. Tian co-founded Huabo Biopharm and served as its general manager from June 2011 to April 2015. For the purpose of developing the product, Hanyu entered into a technology development agreement with Huabo Biopharm in March 2014, under which Huabo Biopharm was engaged to provide CRO-like technical service for the production of two recombinant proteins, HY03M and HY03MM (which are described in the IMM01 patent family), by using the target gene DNA provided by Hanyu, and Hanyu was required to pay a service fee to Huabo Biopharm. As a result, all the products of the CRO-like technical service along with their legal rights shall belong to Hanyu. During the discovery process, Dr. Jing made substantive contributions to, among others, the structure and sequence designs, biological activity analysis, and animal studies of the IMM01 molecule and Dr. Tian made substantive contributions to the related inventions of IMM01 patent family by, among others, providing suggestions on the sequence, vector construction, protein expression, and bio-assay analysis. Subsequently, Hanyu filed a Chinese patent application (No. 201510203619.7) in relation to the target molecule (which was later developed to IMM01) on April 24, 2015. In August 2015, the Company entered into a patent application assignment agreement with Hanyu, pursuant to which all rights in this Chinese patent application and the inventions disclosed therein were transferred from Hanyu to the Company. After the assignment, the Company has obtained the issued Chinese patent as the sole owner and the right to file the patent and patent applications in relation to IMM01 in other jurisdictions as the sole applicant. The Company obtained the full rights to IMM01 based on the assignment agreement, and Hanyu does not retain any rights to IMM01 according to this assignment agreement, as confirmed by the IP legal advisor. Hanyu was established in Shanghai on February 13, 2014 and used to be a start-up biotechnology company. Hanyu was officially deregistered in July 2020. Before its deregistration, Hanyu was owned by Lijuan Liu and Hua Wang, and Lijuan Liu used to be the sole director of Hanyu. After its deregistration, it no longer exists as a legal entity. Huabo Biopharm was established in Shanghai in June 2011. According to public information, Huabo Biopharm is a biotechnology company with multiple clinical-stage assets in the fields of oncology, autoimmune and ophthalmic. It is a wholly-owned subsidiary of Shanghai Hua'aotai Pharmaceutical Co., Ltd., which is in turn a subsidiary of Zhejiang Huahai Pharmaceutical Co., Ltd. (SH stock exchange: 600521) ("**Huahai Pharmaceutical**"). Mr. Song Li (vice president of research and development), Mr. Ruliang Zhang (deputy general manager and senior vice president), and Mr. Zimeng Zhao (supervisor) used to work at Huabo Biopharm. For more details, please refer to "Directors, Supervisors and Senior Management". They have no connection or association with Huabo Biopharm or Huahai Pharmaceutical since their departure. Each of Mr. Song Li and Mr. Ruliang Zhang was granted a small amount of stock options by Huahai Biopharmaceutical in 2015, but they did not exercise those options thus hold no shares or interests in Huabo Biopharm or Huahai Pharmaceutical. Save for the aforementioned employment relationships, during the Track Record Period and up to the Latest Practicable Date, there were not any material past or present relationships or dealings (including family, business, employment, trust, financing or otherwise) between us and Hanyu, Huabo Biopharm, Hanyu and Huabo Biopharm's respective shareholders, directors or senior management, or any of their respective associates.

The initial Chinese patent application filed by Hanyu listed Lijuan Liu, Dr. Deqiang Jing and Hua Wang as inventors. However, as confirmed by Hanyu in supplemental agreements to the assignment agreement, and confirmed in the interview with relevant personnel, Dr. Tian and Dr. Jing are the only inventors that made substantive contributions to the inventions of IMM01. Under the supplemental agreements, Hanyu also confirmed that the Company may list the correct inventors in the U.S. patents and patent applications as well as other foreign patents and patent applications in the patent family which were filed subsequently after the transfer of the patent rights. The Company did not correct the inventorship of the Chinese patent (CN106146670B) since the relevant patent application was already filed at the time of transfer. As advised by JunHe LLP, the intellectual property legal advisor to the Company, the error in inventorship in this Chinese patent would not affect the ownership rights or validity of this Chinese patent since this Chinese patent has been granted and the error in inventorship does not form a legal ground to challenge the validity of a patent under the Chinese patent laws and regulations, and the Company fully owns the intellectual property rights and global commercial rights in relation to IMM01. As of the Latest Practicable Date, the Company has not been involved in any settled, existing or potential legal, arbitral or administrative proceedings, or any dispute or third party claim, in respect of the initial Chinese patent application and later granted as Chinese patent CN106146670B. Furthermore, as the Company has legally obtained full ownership rights to the Chinese patent CN1016146670B as the sole owner pursuant to Hanyu Agreement, even if a third party claims any rights in relation to the Chinese patent CN106146670B, the Company shall not be deprived of the ownership rights to the Chinese patent CN1016146670B to the extent that the Company was a bona fide third party in obtaining the ownership rights to the same. Even if any former employee or agent of Hanyu asserts claims to ownership of any service invention he/she has made contributions to, he/she could only assert such claim to Hanyu under the applicable PRC laws, because Hanyu was the relevant employer or partner who has contractual or employment relationship with him/her. As advised by the Company's PRC legal advisor, JunHe LLP, and the Company's PRC intellectual property legal advisor, JunHe LLP, the risk of having third party claims and listed names associated with Lijuan Liu and Hua Wang in relation to the initial Chinese patent application and later granted as CN106146670B would be remote. Based on the due diligence performed by the Joint Sponsors and having taken into account the factors foregoing, the Joint Sponsors are not aware of any factor that would cause them to cast doubt in any material respect on the above views of the Company's IP Legal Advisors;

(4) The three patent applications in relation to IMM01 in the U.S. were filed and directed to a pharmaceutical composition and fusion proteins with different protection scopes, respectively.

Drug Candidate	Title of Invention	Application Number	Filing Date[1]	Inventors	Jurisdiction	Status[2]	Grant Date	Estimated Expiration Date
IMM0306 (CD47xCD20)	Novel Recombinant Bi-functional Fusion Proteins, Preparation and Application Thereof (CD47/CD20)	201880011334.5	March 15, 2018	Wenzhi Tian, Song Li	PRC	Granted	April 12, 2022	March 15, 2038
		201710151979.6	March 15, 2017		PRC	Granted	October 16, 2020	March 15, 2037
		2019-542396	March 15, 2018		Japan	Granted	April 11, 2022	March 15, 2038
		16/489,360	March 15, 2018		U.S.	Granted	August 9, 2022	December 28, 2038
		PCT/CN2018/079187	March 15, 2018		PCT	Entered national phase	N/A	N/A
		18768501.1	March 15, 2018		EU	Allowed	N/A	N/A
IMM2902 (CD47xHER2)	Recombinant Bifunctional Protein Targeting CD47 and HER2	201980051644.4	August 6, 2019	Wenzhi Tian, Song Li	PRC	Pending	N/A	N/A
		62021034787.3	August 6, 2019		Hong Kong	Pending	N/A	N/A
		PCT/CN2019/099530	August 6, 2019		PCT	Entered National Phase	N/A	N/A
		16/535,075	August 8, 2019		U.S.	Granted	September 27, 2022	August 8, 2039
		19847964.4	August 6, 2019		EU	Pending	N/A	N/A
		2021-506322	August 6, 2019		Japan	Granted	December 12, 2022	August 6, 2039
		17/820,624	August 8, 2019		U.S.	Pending	N/A	N/A
IMM2520 (CD47xPD-L1)	Novel Recombinant Bi-functional Fusion Protein and Preparation and Application Thereof	2021-163660	October 4, 2021	Wenzhi Tian, Song Li	Japan	Granted	April 25, 2022	October 4, 2041
		202111083819.5	September 15, 2021		PRC	Allowed	N/A	N/A
		17/496,051	October 7, 2021		U.S.	Allowed	N/A	N/A
		21199189.8	September 27, 2021		EU	Pending	N/A	N/A
		PCT/CN2022/116312	August 31, 2022		PCT	Pending	N/A	N/A

Drug Candidate	Title of Invention	Application Number	Filing Date[1]	Inventors	Jurisdiction	Status[2]	Grant Date	Estimated Expiration Date
IMM2510 (PD-L1×VEGF)	Recombinant Protein Targeting PD-L1 and VEGF	201980079944.3	December 2, 2019	Wenzhi Tian, Song Li	PRC	Allowed	N/A	N/A
		PCT/CN2019/122446	December 2, 2019		PCT	Entered national phase	N/A	N/A
		16/699,732	December 2, 2019		U.S.	Granted	August 9, 2022	July 16, 2040
		19892300.5	December 2, 2019		EU	Pending	N/A	N/A
		2021-531099	December 2, 2019		Japan	Granted	September 27, 2022	December 2, 2039
		17/737,159	December 2, 2019		U.S.	Pending	N/A	N/A
IMM47 (CD24 mAb)	Antibodies targeting CD24 and their preparation and use	202111195246.5	October 13, 2021	Wenzhi Tian, Song Li, Dianze Chen[5], Huiqin Guo[6]	PRC	Allowed	N/A	N/A
		17/685,530	March 3, 2022		U.S.	Pending	N/A	N/A
		22156295.2	February 11, 2022		EU	Pending	N/A	N/A
		2022-019864	February 10, 2022		Japan	Pending	N/A	N/A
		PCT/CN2022/114945	August 25, 2022		PCT	Pending	N/A	N/A
IMM4701 (CD24×CD47)	Recombinant Protein Targeting CD47 and CD24	202111195248.4	October 13, 2021	Wenzhi Tian, Song Li, Dianze Chen	PRC	Allowed	N/A	N/A
		17/543,033	December 6, 2021		U.S.	Pending	N/A	N/A
		22150987.0	January 11, 2022		EU	Pending	N/A	N/A
		2021-195587	December 1, 2021		Japan	Pending	N/A	N/A
		PCT/CN2022/116315	August 31, 2022		PCT	Pending	N/A	N/A

Notes:

(5) Mr. Dianze Chen served as an assistant director of our Company as of the Latest Practicable Date. He has participated in and made substantive contributions to the screening and validation of our drug candidates, including IMM47, IMM4701 and IMM40H.

(6) Ms. Huiqin Guo served as the head of hybridoma antibody discovery of our Company as of the Latest Practicable Date. She has participated in and made substantive contributions to the screening of various drug candidates, including IMM47 and IMM40H.

Drug Candidate	Title of Invention	Application Number	Filing Date[1]	Inventors	Jurisdiction	Status[2]	Grant Date	Estimated Expiration Date
IMM40H (CD70 mAb)	Antibodies targeting CD70 and their preparation and use	202111191860.4	October 13, 2021	Wenzhi Tian, Song Li, Dianze Chen, Huiqin Guo	PRC	Allowed	N/A	N/A
		2022-015259	February 2, 2022		Japan	Pending	N/A	N/A
		22155661.6	February 8, 2022		EU	Pending	N/A	N/A
		17/685,501	March 3, 2022		U.S.	Allowed	N/A	N/A
		PCT/CN2022/114942	August 25, 2022		PCT	Pending	N/A	N/A

The term of an individual patent may vary based on the jurisdictions in which it is granted. The actual protection afforded by a patent varies on a claim-by-claim and jurisdiction-by-jurisdiction basis and depends upon many factors, including the type of patent, the scope of its coverage, the availability of any patent term extensions or adjustments, the availability of legal remedies in a particular jurisdiction and the validity and enforceability of the patent. We cannot provide any assurance that patents will issue with respect to any of our owned pending patent applications or any such patent applications that may be filed in the future, nor can we provide any assurance that any of our owned issued patents or any such patents that may be issued in the future will be commercially useful in protecting our drug candidates and methods of manufacturing the same. It usually takes about three to five years after a patent application enters into substantive examination for the applicable patent examination authorities to make a final decision on whether a patent should be issued or not. Our legal advisor as to intellectual property laws, JunHe LLP, has checked and reviewed the legal status of the pending patent applications in relation to the Core Product, Key Products and other drug candidates with filed patent applications in the public online databases of the CNIPA, the USPTO, World Intellectual Property Organization ("**WIPO**") and some other public patent databases as well as the information provided by us regarding the pending patent applications. Our legal advisor as to intellectual property laws, JunHe LLP, is not aware of any fact or legal impediment with respect to those pending patent applications that would preclude the issuance of patents with respect to such pending patent applications except that these patent applications remain subject to the examination opinions from the applicable patent examination authorities during the ordinary pendency and examination of such patent applications.

As reviewed and advised by our legal advisor as to intellectual property laws, material aspects (e.g., constructs, sequences or indications under development) of our Core Product can be covered by granted patent and pending patent applications in the PRC and the U.S. For the pending patent applications, as the time required for the substantial review is at the discretion of relevant patent examination authority, we are unable to predict the expected time frame of receiving material updates in relation to the pending patent applications. Given that obtaining issuance of such pending patent applications is not a prerequisite for our future R&D or commercial activities, we do not expect the pending status of patent applications in relation to our respective products would impose barriers on the commercialization of respective products when those products reach commercialization stage. Even if we fail to obtain issuance of any patents that we are applying for, we will still be able to commercialize our drug candidates in the U.S. (unless a legal proceeding has been filed against us and as a result of the legal proceeding an injunction has been issued or a final decision has been rendered by the court which requires us to cease manufacturing and commercializing our products) and the PRC, although without the protection of the relevant intellectual property right offered by patents during respective patent's validity period. Therefore, we believe any failure to obtain issuance of the patent applications we are applying for will not directly hamper our business, financial conditions or results of operations.

However, if any of the patent applications was rejected, we may lack patent protection covering certain key characteristics of our respective products before or during the commercialization of our products. If any of the above circumstances occurs, our business, financial conditions and prospects could be materially and adversely affected. See for more details in the "Risk Factors — Risks Relating to Our Intellectual Property Rights — If we are unable to obtain and maintain adequate patent and other intellectual property protection for our drug candidates throughout the world, or if the scope of such intellectual property rights obtained is not sufficiently broad, our current or any future patents may be challenged and invalidated even after issuance".

We are aware of certain issued patents in the U.S. belonging to third parties that may potentially cover our CD47-based drug candidates and may not expire before our anticipated commercial launch of relevant drug candidates in the U.S. As reviewed and advised by our legal advisor as to intellectual property law, JunHe LLP[1], the scope of the relevant patent claims is too broad and the patent claims are obvious over prior art or lack written description and enablement support, the validity and enforceability of the third-party patents are thus questionable; as a result, if such third parties bring the legal proceedings against us, the risk that we will be determined by courts or other competent authorities in the U.S. to have infringed on such patent rights of the third parties is remote. However, whether a product infringes a patent involves an analysis of complex legal and factual issues, the determination of which is often uncertain, and the burden of proof required to successfully challenge a third-party patent may be high. For details, please refer to the paragraphs headed "Risk Factors — Key Risks Relating to Our Business, Business Operations, Intellectual Property Rights and Financial Prospects — Third parties may initiate legal proceedings alleging that we are infringing, misappropriating or otherwise violating their intellectual property rights, the outcome of which would be uncertain."

In addition, in 2019, we signed a technology transfer agreement with an independent third party, pursuant to which such third party acquired certain rights and interests (including one patent application in China relating to IMM2505) from us to develop and commercialize IMM2505 in China (including Hong Kong, Macau and Taiwan), while we retain the full rights and interests to IMM2505 in the rest of the world. We decided to license out IMM2505 since such out-licensing arrangement can supplement our cash flow to develop our pipeline products. At the time of such transfer, IMM2505 was at early discovery stage. The Chinese patent application of IMM2505 has not been issued, and is currently under the CNIPA's substantive examination. If such patent application of IMM2505 is approved with the currently pending claims, it may potentially cover IMM2520. However, based on the opinion of our legal advisor as to intellectual property law, JunHe LLP, the currently pending claims of the Chinese patent application relating to IMM2505 are too broad and lack inventiveness over prior art, considering (i) bispecific molecules binding to both CD47 and PD-L1 have been disclosed in the prior art; (ii) the amino acid sequence of SIRP extracellular Ig-like domain (which binds to CD47) is known in the prior art; (iii) various PD-L1 antibodies with different amino acid sequences have been disclosed in the prior art; and (iv) the first office action issued by the CNIPA raises novelty or inventiveness rejections on the pending claims. In addition, the issued patents in the U.S. and Japan regarding IMM2505 were granted with claims reciting specific amino acid sequences of the PD-L1 antibody and SIRP extracellular Ig-like domain. Therefore, it is expected that the pending claims of the Chinese patent application of IMM2505 would be narrowed down during prosecution by further limiting the amino acid

[1] JunHe LLP has a registered office in the Silicon Valley of California and has extensive experience in the U.S. patent practice. The JunHe patent team has deep expertise across many aspects of life sciences including biological and small therapeutic compounds and uses thereof, proteomics, genomics, molecular diagnostics, drug discovery tools, chemicals and materials science, and medical devices. The JunHe Patent team has decades of experience in a wide range of U.S. patent related matters including drafting, prosecuting patents at the U.S. Patent and Trademark Office, patent infringement and validity opinions regarding U.S. patents, strategic counseling and due diligence reviews of U.S. patents in M&A deals, capital market offerings, financing and other high-value transactions. In addition, a U.S. based international law firm, Locke Lord LLP, was specifically engaged to conduct analysis of a certain U.S. patent. Locke Lord LLP has substantial patent practice experience in the U.S. in life science industry. Its team combines strong scientific understanding, courtroom experience, regulatory knowledge and industry background in the U.S. Their technical understanding spans pharmaceuticals, molecular genetics, biotechnology, organic, inorganic and industrial chemistry, and biochemistry.

sequences of the PD-L1 antibody portion of IMM2505, similar to our issued patents in the U.S. and Japan. For details, please refer to the paragraphs headed "Risk Factors — Key Risks Relating to Our Business, Business Operations, Intellectual Property Rights and Financial Prospects — If we are unable to obtain and maintain adequate patent and other intellectual property protection for our drug candidates throughout the world, or if the scope of such intellectual property rights obtained is not sufficiently broad, our current or any future patents may be challenged and invalidated even after issuance."

We also own a number of registered trademarks and pending trademark applications. As of the Latest Practicable Date, we had registered trademarks for our Company and our corporate logo in the PRC and Hong Kong and are seeking trademark protection for our Company and our corporate logo in the jurisdictions where available and appropriate.

During the Track Record Period and up to the Latest Practicable Date, (i) we were not involved in any legal, arbitral or administrative proceedings in respect of, and we had not received notice of any material claims of infringement, misappropriation or other violations of third-party intellectual property; and (ii) we were not involved in any proceedings in respect of any intellectual property rights that may be threatened or pending and that may have an influence on the research and development for any of our drug candidates in which we may be a claimant or a respondent.

RAW MATERIALS AND SUPPLIERS

Suppliers

During the Track Record Period, our suppliers primarily consisted of CROs, CMO/CDMOs, and suppliers of equipment, devices and construction services. We select our suppliers by considering their product quality, costs, delivery standards, industry reputation and compliance with relevant regulations and industry standards.

For the years ended December 31, 2022 and 2021, the aggregate purchases attributable to our five largest suppliers amounted to RMB58.1 million and RMB55.9 million, respectively, representing 30.2% and 32.4% of our total purchases, respectively. Purchases attributable to our single largest supplier amounted to RMB16.8 million and RMB17.8 million for the same periods, accounting for 8.7% and 10.3% of our total purchases, respectively. All of our five largest suppliers during the Track Record Period operate their business in the PRC, except for one major supplier in 2022 that operates its business in the U.S. We believe that we maintain strong and stable relationships with our major suppliers.

The following table sets forth details of our five largest suppliers for the year ended December 31, 2022:

Ranking	Supplier	Products/Services purchased	Length of business relationship	Purchase amount	% of total purchase
				(RMB '000)	
1	Supplier A (a CDMO) . . .	Manufacturing services	2 years	16,751	8.7%
2	Supplier B (a construction company)	Construction	2 years	13,514	7.0%
3	Supplier C (a CRO)	R&D services	2 years	10,940	5.7%
4	Supplier D (a CRO)	R&D services	3 years	8,768	4.6%
5	Supplier E (a CRO)	R&D services	2 years	8,125	4.2%
	Total			**58,098**	**30.2%**

The following table sets forth details of our five largest suppliers for the year ended December 31, 2021:

Ranking	Supplier	Products/Services purchased	Length of business relationship	Purchase amount	% of total purchase
				(RMB '000)	
1	Supplier D (a CRO)	R&D services	2 years	17,750	10.3%
2	Supplier A (a CDMO) . . .	Manufacturing services	1 year	12,704	7.4%
3	Supplier F (a laboratory construction company) .	Construction works for office building decoration	3 years	10,415	6.0%
4	Supplier G (a CMO)	Manufacturing services	6 years	7,571	4.4%
5	Supplier H (a supplier of equipment)	Equipment	4 years	7,442	4.3%
	Total			**55,882**	**32.4%**

During the Track Record Period, none of our five largest suppliers was our related parties. None of our Directors or their associates or, to the knowledge of our Directors, any Shareholder with over 5% of the share capital of our Company has any interest in any of our five largest suppliers in the years ended December 31, 2021 and 2022.

Raw Materials

During the Track Record Period, we have procured raw materials for the pilot production of our drug candidates for clinical trials from suppliers in China. The principal raw materials that we used include resin, filtration materials, excipient, among others. We select our suppliers by considering cost and their capability, quality, reputation, delivery and regulatory compliance.

CUSTOMERS

During the Track Record Period, since we had not obtained regulatory approval for the commercial sale of any of our drug candidates, we had not generated any revenue from sales of any drug products. Our revenue was generated from out-licensing fee, sales of cell strain and other products and testing services during the Track Record Period. For further details, please refer to the paragraphs headed "Financial Information — Description of Selected Components of Consolidated Statements of Profit or Loss and Other Comprehensive Income — Revenue."

For the years ended December 31, 2022 and 2021, the aggregate sales to our five largest customers were RMB0.5 million and RMB5.0 million, representing 84.6% and 98.8% of our total sales, respectively. Revenue from our single largest customer accounted for 28.1% and 93.3% of our total sales amount for the same periods, respectively. All of our top five customers during the Track Record Period are independent third parties and located in the PRC.

The following table sets forth details of our five largest customers for the year ended December 31, 2022:

Ranking	Customer	Products/Services sold	Length of business relationship	Sales amount	% of total sales
				(RMB '000)	
1	Customer A (a biotechnology company)	Cell lines, growth medium and technical services	1 year	151	28.1%
2	Customer B (a biotechnology company)	Cell lines and growth medium	2 years	150	27.9%
3	Customer C (a biotechnology company)	Growth medium	4 years	98	18.2%
4	Customer D (a biotechnology company)	Growth medium	4 years	36	6.7%
5	Customer E (a biotechnology company)	Technical services	1 years	20	3.8%
	Total			**456**	**84.6%**

The following table sets forth details of our five largest customers for the year ended December 31, 2021:

Ranking	Customer	Products/Services sold	Length of business relationship	Sales amount	% of total sales
				(RMB '000)	
1	Customer F (a biotechnology company)	Technology license; growth medium	3 years	4,727	93.3%
2	Customer G (a CDMO)	cell strain	1 year	143	2.8%
3	Customer C (a biotechnology company)	Growth medium	3 years	61	1.2%
4	Customer H (a biotechnology company)	Testing services	1 year	38	0.7%
5	Customer I (a biopharmaceutical company)	Testing services	1 year	38	0.7%
	Total			**5,006**	**98.8%**

To the knowledge of our Directors, none of our Directors, their respective associates or any of our Shareholders holding more than 5% of our issued share capital immediately following the completion of the **[REDACTED]** had an interest in any of our customers during the Track Record Period.

COMPETITION

The market for biopharmaceutical industry and immuno-oncology solutions is evolving and highly competitive. While we believe that our research and development capabilities enable us to establish a favorable position in the industry, we encounter competition from international and domestic biopharmaceutical companies, specialty pharmaceutical and biotechnology companies of various sizes, academic institutions and research institutions. For more information on the competitive landscape of our drug candidates, please refer to the paragraphs headed "Industry Overview" and "— Our Drug Candidates."

We believe the primary competitive factors in our markets are identification of promising targets, mechanisms and pathways for drug development, molecule screening and design, efficacy and safety of drug candidates, manufacturing efficiency and commercialization development. We expect the competition will become more intensive in the future as additional players enter into the segments. Any drug candidates that we successfully develop and commercialize will compete with existing drugs or any new drugs that may become available in the future. For potential impact of market competition, see "Risk Factors — Key Risks Relating to Our Business, Business Operations, Intellectual Property Rights and Financial Prospects — We face substantial competition and our competitors may discover, develop or commercialize competing drugs faster or more successfully than we do."

EMPLOYEES

As a biotechnology company, our employees are our valuable resource. We are led by a diverse and talented team of management and experts who seek to understand immuno-oncology therapies' challenges and are dedicated to tackling them. As of the Latest Practicable Date, we had a total of 143 full-time employees, among which 140 were in China, one in Australia and two in the U.S. The following table sets forth a breakdown of our employees categorized by function as of the Latest Practicable Date:

Function	Number	Percentage
R&D	17	11.9%
Clinical Development	44	30.8%
CMC and Regulatory Affairs	45	31.5%
Business Strategy and Corporate Development	12	8.4%
General and Administrative	25	17.5%
Total	**143**	**100.0%**

We also plan to develop our internal sales and marketing team preparing for the commercialization of our drug candidates in the future.

Employment Agreements with key management and R&D staff

We enter into standard labor, confidentiality and non-compete agreements with our employees. The non-compete restricted period typically expires two years after the termination of employment, and we agree to compensate the employees with a certain percentage of their pre-departure salary during the restricted period. For further details regarding the terms of the confidentiality and non-compete and employment agreements with our certain of our senior management, please refer to the section headed "Directors, Supervisors and Senior Management" in this document.

We recruit and retain highly engaged and motivated team players who are driven by our commitment and are excited to contribute to the development of next-generation immuno-oncology therapies leveraging their extensive experience. We believe that we are in a good position to create an equitable, inclusive and diverse workplace while maintaining a good working relationship with our employees. As of the Latest Practicable Date, we had not experienced any major labor disputes.

Training and Development

We offer employees a variety of professional development opportunities and encourage a performance-driven environment. We focus on creating a robust culture to encourage retention and engagement. Given our emphasis on operating a fully-integrated platform for our drug development processes, we attach great importance to internal talent growth. We continually pursue progression opportunities for our staff through various internal and external training and development programs.

Employee Benefits

We are committed to making sure that working conditions throughout our business network are safe and that employees are treated with care and respect. We believe we offer our employees competitive compensation packages, reflecting our stakeholder-centric ethos which we believe leads to sustainable and durable growth. As required by PRC regulations, we participate in various government statutory employee benefit plans, including social insurance, namely pension insurance, medical insurance, unemployment insurance, work-related injury insurance, maternity insurance, and housing funds. We are required under PRC law to make contributions to employee benefit plans at specified percentages of the salaries, bonuses and certain allowances of our employees, up to a maximum amount specified by the local government regulations from time to time.

LAND AND PROPERTIES

Our corporate headquarter is located in Shanghai Municipality, PRC. As of the Latest Practicable Date, we had a land use right to a land parcel located in Pudong New Area with a site area of approximately 28,763.1 sq.m, and a total of approximately 6,180.98 sq.m leased property space as our office premises, and research and development center in the PRC. The relevant lease agreements generally provide a duration of up to 74 months.

The following table sets forth the details of our owned and leased properties as of the Latest Practicable Date:

Usage	Location	GFA (sq.m)	Lease Term
R&D, manufacturing, office	Shanghai	28,763.10	/[(1)]
R&D, manufacturing, marketing, office	Shanghai	2,707.44	May 1, 2019, to July 31, 2024
R&D, office	Shanghai	1,662.58	April 1, 2021, to March 31, 2027
Office	Shanghai	1,441.37	March 1, 2021, to February 29, 2024
Office	Shanghai	403.64	April 1, 2021 to March 31, 2027
Storage, office	Shanghai	369.59	October 1, 2022, to November 30, 2028

Note:

(1) This property is owned by the Company.

According to Chapter 5 of the Listing Rules and section 6(2) of the Companies (Exemption of Companies and Prospectuses from Compliance with Provisions) Notice, this document is exempted from compliance with the requirements of section 342(1)(b) of the Companies (Winding Up and Miscellaneous Provisions) Ordinance in relation to paragraph 34(2) of the Third Schedule to the Companies (Winding Up and Miscellaneous Provisions) Ordinance which require a valuation report with respect to all our interests in land or buildings, for the reason that, as of June 30, 2022, none of our properties has a carrying amount of 15% or more of our consolidated total assets.

As of the Latest Practicable Date, except for the land use right to a land parcel located in Pudong New Area recently granted to us, we do not own any other real property for our operations. Upon expiration of our leases, we will need to negotiate for renewal of the leases or relocate. There are sufficient alternative locations for us to choose from, but we may incur additional costs in relation to the potential relocation. During the Track Record Period, we did not experience any dispute arising out of our leased properties. For details of risks relating to our leased properties, see the section headed "Risk Factors — Other Risks Relating to Our Operations — We are subject to risks associated with leasing space" in this document.

AWARDS AND RECOGNITIONS

We have received various awards and recognitions for our projects and entities. The following table sets forth the selected awards and projects for which we received government grants as of the Latest Practicable Date:

Year of Grant	Award/Recognition	Issuing Authority
2022	Dr. Tian was awarded as "Top 10 Drug Innovation Scientists/Research Teams of the Year"	Securities Times
2022	IMM2902 was awarded as "Drug Innovation and Development of the Year"	Securities Times
2022	IMM01 was award as "2022 Top 10 Innovative Pharmaceutical CHIP Seed Projects"	CHIP Academy
2022	Shanghai Technologically Advanced Small and Medium-sized Enterprise	Shanghai Municipal Commission of Economy and Informatization
2022	Shanghai Pudong New Area Innovative Small and Medium-Sized Enterprise	Shanghai Municipal Commission of Economy and Informatization
2022	Joint Unit of the New Overseas Chinese Training Base	Shanghai Pudong New Area Government Overseas Chinese Affairs Office
2021	Top 100 China Pharmaceutical Innovative Seed Enterprises in 2021	China Pharmaceutical Enterprise Management Association, China Pharmaceutical Biotechnology Association, General Office of the Central Committee of the Chinese Peasants and Workers Democratic Party, Hangzhou Investment Promotion Bureau, Hangzhou Qiantang New Area Management Committee

Year of Grant	Award/Recognition	Issuing Authority
2021	Innovative Startup Award	Shanghai Pudong New Area Government
2020	High-tech Enterprise Certificate	Shanghai Municipal Science and Technology Commission, Shanghai Municipal Finance Bureau, State Administration of Taxation (Shanghai Taxation Bureau)
2019	Top 50 Most Innovative Companies of Chinese Biomedicine in 2019	Shanghai Tuling Biotechnology Co., Ltd. Xingyao Research Institute
2018	Zhangjiang Venture Capital TOP100 Enterprise Honor	"Insight into Zhangjiang" Venture Capital Database
2018	2018 Shanghai Science and Technology Business Incubator 30-year Cutting-edge Start-up Enterprises	Shanghai Science and Technology Entrepreneurship Center
2017	Enterprise Excellence Award	Shanghai Science and Technology Entrepreneurship Center
2017	Excellent Enterprise	Organizing Committee of China Innovation and Entrepreneurship Competition
2016	50 Top Shanghai Start-ups with Most Investment Potential in 2016	Shanghai SME Development Service Center, Shanghai SME listing Promotion Center

ENVIRONMENTAL, SOCIAL, HEALTH AND SAFETY MATTERS

We are committed to environmental protection and promoting corporate social responsibility and best corporate governance practices to develop sustainable value for stakeholders and take up responsibilities as a corporate citizen. We are currently at an early stage of laboratory operations and partially rely on CMO/CDMOs for the manufacturing function and on CROs for animal studies, clinical trials and other activities. As a result, the current nature of our business does not expose us to a substantial risk of environmental, health or work safety matters, and we do not expect the potential risks of such matters will have a material adverse impact on our business operation and financial performance. Our operations in the future, particularly after the completion of construction and commencement of operations of our manufacturing facility in Shanghai, will be subject to numerous environmental, social, health and safety laws and regulations. For a discussion on PRC laws and regulations on environmental protection and work safety, see "Regulations — Regulations relating to Environmental Impact Assessment of Construction Projects."

We are committed to complying with PRC regulatory requirements, preventing and reducing hazards and risks associated with our operation, and ensuring the health and safety of our employees and surrounding communities. We will comply with the environmental, social and governance ("**ESG**") reporting requirements after **[REDACTED]** and the responsibility to publish ESG report on an annual basis in accordance with Appendix 27 to the Listing Rules. We will focus on each of the areas as specified in Appendix 27 to the Listing Rules to analyze and disclose important ESG matters, risk management and the accomplishment of performance objectives, particularly those environmental and social issues that could have a material impact on the sustainability of our operations and that are of interest to our Shareholders. We have adopted company-wide environment, health and safety policies and various systems and procedures relating

to hazardous waste management, wastewater treatment, air pollution control, environmental risk management, emergency response and process safety management. We have also adopted and maintained a series of rules, standard operating procedures and measures to maintain a healthy and safe environment for our employees. We implement safety guidelines setting out information about potential safety hazards and procedures for operating in the manufacturing facilities. We require new employees to participate in safety training to familiarize themselves with the relevant safety rules and procedures. In particular, we invite experts on fire control safety to conduct training sessions and regularly perform emergency evacuation drills to reduce risks associated with potential fire accidents. Also, we have adopted relevant policies and measures to ensure the hygiene of our work environment and the health of our employees. We are endeavored to provide a safe work environment in light of the COVID-19 pandemic, including procurement of epidemic prevention materials and release of work-from-home plan and work resumption plan.

Our Board has established an ESG working group that comprises three centers, including finance center, production and quality center and human resource center. The ESG working group serves as a supportive role to our Board in implementing the agreed ESG policies, targets and strategies; conducting materiality assessments of environmental-related, climate-related, social-related risks; collecting ESG data from different parties while preparing for the ESG report; and continuous monitoring of the implementation of measures. The ESG working group has to prepare a quantitative report with regard to our environmental-related and social-related data on a quarterly basis and prepare a qualitative report with regard to effectiveness of our ESG measures two times a year.

Our Board sets targets for each material key performance indicators ("**KPIs**") in accordance with the disclosure requirements of Appendix 27 to the Listing Rules and other relevant rules and regulations upon **[REDACTED]**. In setting targets for the ESG-related KPIs, our Group has taken into account their respective historical levels for 2021 and 2022 and has considered our future business expansion thoroughly and prudently with a view of balancing business growth and environmental protection to achieve sustainable development. We will also review our KPIs on a yearly basis to ensure that they remain appropriate to our Group. Set forth below are our major KPIs during the Track Record Period:

- *Hazardous waste disposal*. We have monitored our hazardous waste disposal levels on a periodic basis. For 2021 and 2022, our hazardous waste discharge levels were approximately 6.1 tons and 5.6 tons, respectively, and such waste was disposed by qualified third parties.

- *Electricity consumption*. We have monitored our electricity consumption levels and implement measures to improve energy efficiency. For 2021 and 2022, our electricity consumption levels were approximately 2.2 million kWh and 2.7 million kWh, respectively.

- *Water consumption*. We have monitored our water consumption levels and implement measures to promote water conservation. For 2021 and 2022, our water consumption levels were approximately 4,971 tons and 5,068 tons, respectively.

We do not operate in a highly polluting industry, while our operation may involve the use and disposal of hazardous materials and wastes. We contract with qualified third parties for the disposal of hazardous materials and wastes. We require their operational qualifications in accordance with relevant governmental laws and regulations. We establish a regular assessment as to our suppliers' safety performance and strengthen our supervision and management of our suppliers. Our contracted third-party service providers are required under our agreements to comply with all applicable laws.

In addition, we identify our ESG-related KPIs to include fair employment and healthy and safe environment for our employees. We place a high value on diversity in our Company and continuously implement pro-diversity management practices. We are also dedicated to providing fair and equal treatment and career opportunities to all of our employees. We prohibit any form of discrimination based on gender, family origin, disability, religious beliefs or races throughout our recruiting process. By implementing these practices, we aim to cultivate health, wellbeing and work-life balance for all of our employees. We have also adopted and maintained a series of rules, standard operating procedures and measures to maintain a healthy and safe environment for our employees. In particular, we (i) have established various guidelines governing laboratory procedures and the handling, use, storage, treatment and disposal of hazardous materials and wastes to ensure such guidelines are strictly enforced for the disposal of laboratory materials and wastes; (ii) inspect our equipment and facilities regularly to identify and eliminate safety hazards; (iii) provide regular safety awareness training to our employees; (iv) keep health records for all employees and conduct health examinations before, during and after their time at the company, especially for employees engaged in work involving occupational hazards; (v) implement company-wide self-protection policies for employees in light of the COVID-19 outbreaks, including providing face masks and disinfectant to our employees; and (vi) conduct regular fire safety inspections, maintenance of fire-fighting equipment and regular emergency drills.

We believe that we are not susceptible to climate change. Moreover, we consider that potential changes to the regulations in the PRC regarding climate change will not adversely impact our business operations. We will continue to pay attention to risks regarding climate change and formulate emergency plans to safeguard us from climate change and extreme weather conditions, such as hurricane and rainstorms. As of the Latest Practicable Date, we had not experienced any material impact on our business operations or financial performance as a result of climate change or extreme weather conditions.

For 2021 and 2022, we spent approximately RMB665.7 thousand and RMB727.7 thousand, respectively, with respect to environmental and work safety protection. Our PRC Legal Advisor has confirmed that, during the Track Record Period and up to the Latest Practicable Date, we had not been subject to any material claim or penalty in relation to environmental, social, health and safety protection, had not been involved in an accident or fatality and had been in compliance with the relevant PRC laws and regulations in all material aspects.

Our Directors recognize the importance of good corporate governance in protecting the interests of our Shareholders. Our directors and senior management will actively develop our environment, social and governance strategies and targets, and evaluate, determine and address the related risks.

INSURANCE

We maintain insurance policies that we consider to be in line with market practice and adequate for our business to safeguard against risks and unexpected events. Our insurance policies cover adverse events in our clinical trials, and we also maintain property loss insurance. We maintain social insurance for our employees in accordance with relevant PRC laws and regulations. We believe that our insurance coverage is adequate to cover our key assets, facilities, and liabilities.

LICENSES AND PERMITS

Our PRC Legal Advisor has advised that during the Track Record Period and up to the Latest Practicable Date, we have obtained all material licenses, permits, approvals and certificates from the relevant government authorities that are material for the business operations of our Group. Our PRC Legal Advisor also confirmed that during the Track Record Period and up to the Latest Practicable Date, we were not involved in any litigation, arbitration or administrative proceedings pending or, to the best knowledge of our Directors, threatened against us or any of our Directors that could have a material adverse effect on our business, results of operations or financial condition.

The following table sets forth details of selected material licenses and permits obtained by our Group as of the Latest Practicable Date:

License/Permit	Holder	Date of Grant	Expiry Date
Customs Declaration Unit Registration Certificate	our Company	October 18, 2017	long term
Foreign Trade Dealers Filing Receipt	our Company	May 16, 2018	/
Entry and Exit Inspection and Quarantine Declaration Enterprise Filing Receipt	our Company	October 17, 2017	/

LEGAL PROCEEDINGS AND COMPLIANCE

As of the Latest Practicable Date, there was no litigation, arbitration or administrative proceedings pending or threatened against the Company or any of our Directors which could have a material and adverse effect on our financial condition or results of operations. Potential future litigation or any other legal or administrative proceeding, regardless of the merit or outcome, is likely to result in substantial costs, diversion of our resources, and have a negative impact on our reputation and brand image, which in turn, would have negative impact on our business, financial condition, and results of operations. For potential impact of legal or administrative proceedings on us, see "Risk Factors — Other Risks Relating to Our Operations — We may be involved in lawsuits or other legal proceedings, which could adversely affect our business, financial conditions, results of operations and reputation."

We are of the view that, during the Track Record Period and up to the Latest Practicable Date, we had complied, in all material respects, with all relevant laws and regulations in the jurisdictions we operate in, and no material administrative penalties imposed on us had been found that may have a material adverse effect on our Group's business operations.

RISK MANAGEMENT AND INTERNAL CONTROL

We have devoted ourselves to establishing and maintaining risk management and internal control systems consisting of policies and procedures that we consider to be appropriate for our business operations, and we are dedicated to continuously improving these systems.

Risk Management

We are exposed to various risks in our business operations and we recognize that risk management is critical to our success. For more details, please refer to the section headed "Risk Factors" for a discussion of various operational risks and uncertainties we face. We are also exposed to various market risks, in particular, credit, liquidity, interest rate and currency risks that arise in the normal course of our business. Please refer to "Financial Information — Market Risk Disclosure" for a discussion of these market risks.

We have adopted a series of risk management policies which set out a risk management framework to identify, assess, evaluate and monitor key risks associated with our strategic objectives on an ongoing basis. Risks identified by management will be analyzed on the basis of likelihood and impact, and will be properly followed up and mitigated and rectified by our Company and reported to our Directors. Our audit committee, and ultimately our Directors supervise the implementation of our risk management policies.

The following key principles outline our Group's approach to risk management and internal control:

- Our audit committee will oversee the implementation of, as well as evaluate and improve the internal control system, including (i) reviewing the internal control and risk management policies, and making suggestions to improve the same; (ii) discussing with the management and evaluating the effectiveness of the internal control and risk management policies, to ensure the performance by the management of their duties to formulate effective internal control and risk management policies; (iii) studying the material findings in relation to internal control and the relevant measures taken by the management; and (iv) overseeing any potential misconduct of the employees with respect to internal control, and establishing relevant procedures to investigate and handle the complaints of the same and of the internal control of the Company.

- Our Board will be responsible for (i) formulating our risk management policy and reviewing major risk management issues of our Company; (ii) providing guidance on our risk management approach to the relevant teams in our Company; (iii) reviewing the relevant teams' reporting on key risks and providing feedbacks; and (vi) supervising the implementation of our risk management measures by the relevant teams.

- The relevant teams in our Company are responsible for implementing our risk management policy and carrying out our day-to-day risk management practice. In order to formalize risk management across our Company and set a common level of transparency and risk management performance, the relevant teams will (i) gather information about the risks relating to their operation or function; (ii) conduct risk assessments, which include the identification, prioritization, measurement and categorization of all key risks that could potentially affect their objectives; (iii) prepare a risk management report annually for our chief executive officer's review; (iv) continuously monitor the key risks relating to their operation or function; (v) implement appropriate risk responses where necessary; and (vi) develop and maintain an appropriate mechanism to facilitate the application of our risk management framework.

We consider that our Directors and members of our senior management possess the necessary knowledge and experience in providing good corporate governance oversight in connection with risk management and internal control.

Internal Control

Our Board is responsible for establishing our internal control system and reviewing its effectiveness. We have engaged an internal control consultant (the "Internal Control Consultant") to perform certain agreed-upon procedures (the "Internal Control Review") in connection with the internal control during the period from November 1, 2020 to October 30, 2021 of our Company and our major operating subsidiaries in certain aspects, including entity-level controls, Financial reporting and disclosure controls, human resources and payroll management, general controls of IT system and other procedures of our operations. The Internal Control Consultant performed the Internal Control Review in November 2021, identified internal control deficiencies and provided recommendation accordingly. We have adopted the corresponding remediation actions to improve the effectiveness of internal control system. The Internal Control Consultant performed a follow-up review with regard to those actions taken by us and there are no further material findings identified in the process of the follow up Review. As of the Latest Practicable Date, there were no material outstanding issues relating to our Company's internal control.

During the Track Record Period, we regularly reviewed and enhanced our internal control system. Below is a summary of the internal control policies, measures and procedures we have implemented or plan to implement:

- We have adopted various measures and procedures regarding each aspect of our business operation, such as related party transaction, risk management, protection of intellectual property, environmental protection and occupational health and safety. We provide periodic training about these measures and procedures to our employees as part of our employee training program. Our internal audit team conducts audit fieldwork to monitor the implementation of our internal control policies, reports the weakness identified to our management and audit committee and follows up on the rectification actions.

- Our Directors (who are responsible for monitoring the corporate governance of our Group) with help from our legal advisors, will also periodically review our compliance status with all relevant laws and regulations after the **[REDACTED]**.

- We have established an audit committee which, among others, (i) makes recommendations to our Board of Directors on the appointment and removal of external auditors; and (ii) reviews the financial statements and internal control system of our Company.

- We plan to provide various and continuing trainings to update our Directors, senior management, and relevant employees on the latest PRC laws and regulations from time to time with a view to proactively identify any concerns and issues relating to any potential non-compliance.