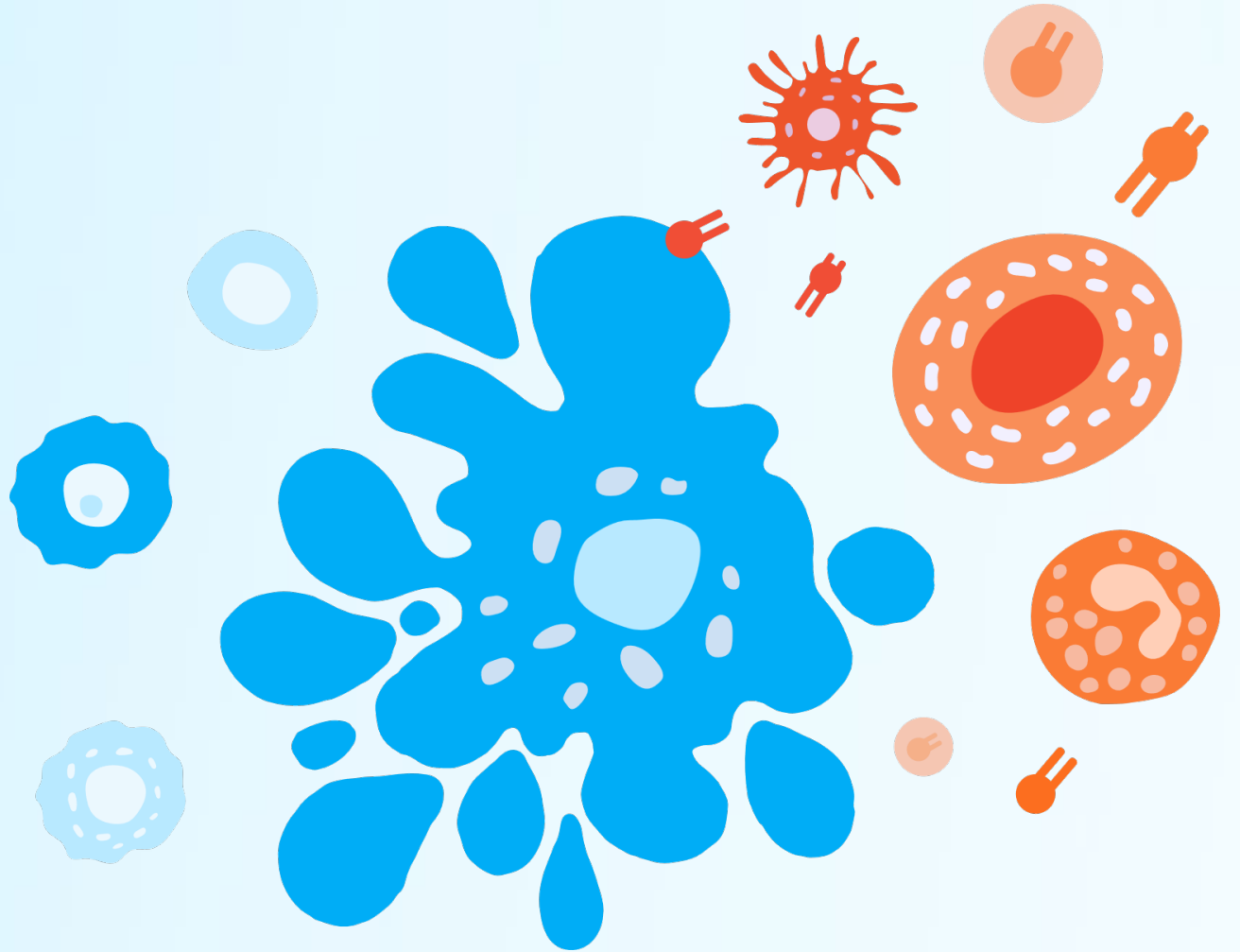




Transforming Potential into Reality

I-Mab Biopharma

November 14, 2024



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Transition to a US-Based Biotech Primarily Complete



Advancing a Differentiated Pipeline

ASSET	PHASE 1	PHASE 2	PHASE 3	MARKET OPPORTUNITY	STATUS/POTENTIAL NEXT STEPS	PARTNERSHIPS
Uilelimab CD73 mAb				1L mNSCLC: Target population of 300k+ patients ²	1H 2025: First patient dosed in pembrolizumab + chemo combination for 1L mNSCLC 2H 2025: Phase 2 PFS data from ongoing TJBio study (China-only) evaluating combination with toripalimab	
Givastomig¹ CLDN18.2 X 4-1BB Bispecific Ab				1L GC, GEJ, EAC: Target population of 100k+ patients ²	Sep-2024: Phase 1 dose expansion monotherapy data presented at ESMO 2024 2H 2025: Phase 1b data in combination with nivolumab + chemo in 1L GC, GEJ, EAC	
Ragistomig/ABL503¹ PD-L1 X 4-1BB Bispecific Ab				Refractory/relapsed cancers: PD-(L)1 progression impacts most patients with metastatic disease ²	May 2024: Phase 1 monotherapy data presented at ASCO 2024	



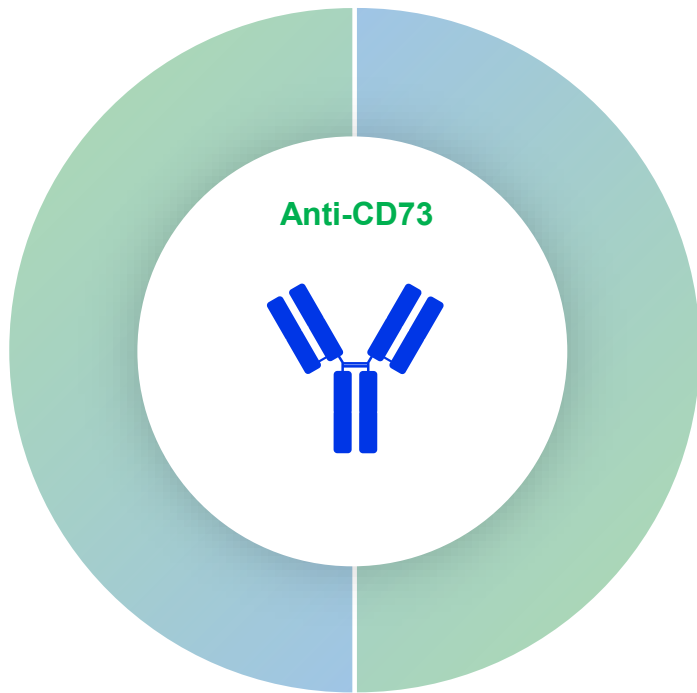
1. Co-developed with ABL Bio (givastomig also known as ABL111, ragistomig also known as ABL503)

2. Global Data Epidemiology Data, Guidehouse legacy research

Notes: CPI = checkpoint inhibitors; mNSCLC = metastatic non-small cell lung cancer; PD-(L)1 refers to inhibitors of PD-L1 or PD-1; Ab = antibody; GC = gastric cancers; GEJ = gastroesophageal junction; EAC = esophageal adenocarcinoma cancer; 1L = first line; ASCO 2024 = the American Society for Clinical Oncology Annual Meeting in 2024; PFS = progression free survival; ESMO 2024 = the European Society for Medical Oncology Annual Meeting in 2024

Uliledlimab (targeting CD73)

Initial development focused on 1L mNSCLC with potential to expand across multiple indications in combination with immune checkpoint inhibitors



CD73 Biology

CD73 is the **rate-limiting enzyme that converts AMP into immunosuppressive adenosine**

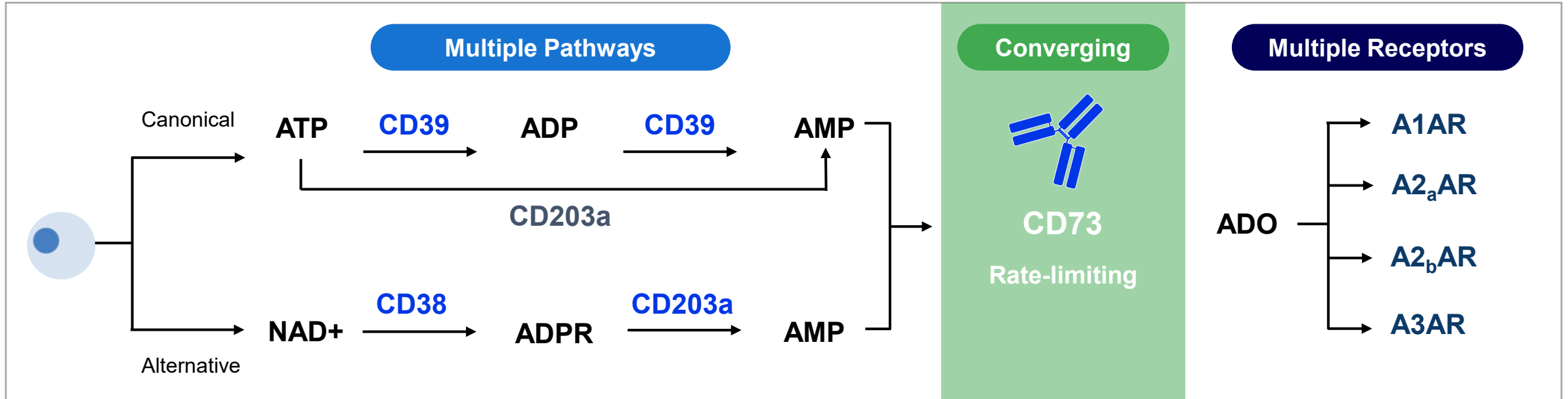
Blocking CD73 activity leads to **complete inhibition of the adenosine pathway**

Key Advantages

Uliledlimab **completely inhibits** CD73 activity and the production of adenosine

Uliledlimab targets CD73 non-competitively **without the “hook effect”**¹

CD73 is the Rate-Limiting Enzyme in the Adenosine Immunosuppression Pathway



All AMP pathways converge at CD73 to generate adenosine

Advantages of targeting CD73 for cancer therapy:

blocking CD73 activity leads to complete inhibition of the adenosine pathway.

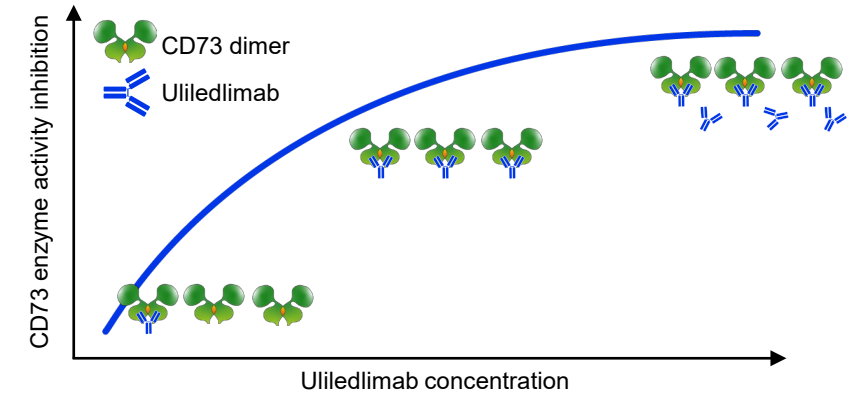
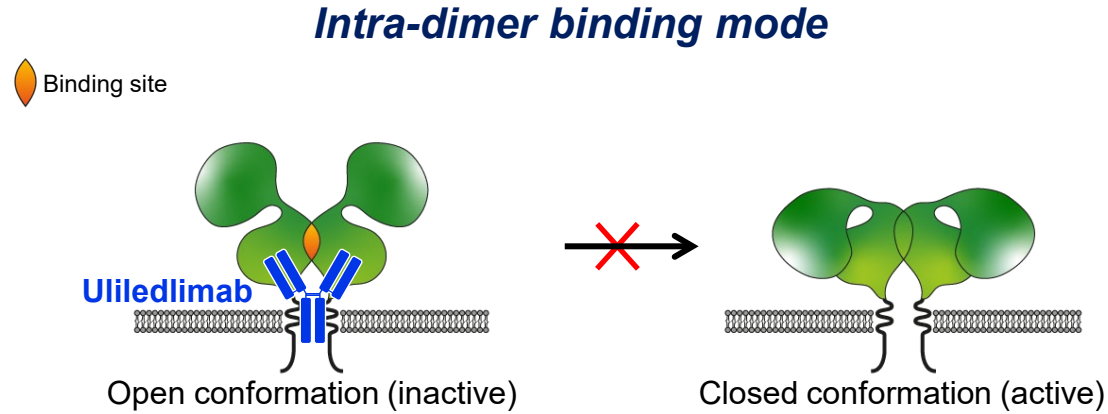
Known potential escape pathways (ATP, cyclic AMP, and nicotinamide adenine dinucleotide through separate biochemical pathways) exist when targeting upstream CD39 or downstream adenosine receptors.

Uiledlimab: A Differentiated CD73 Antibody

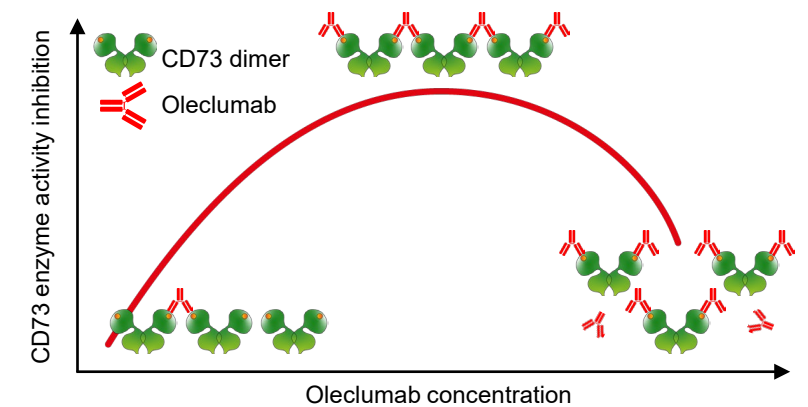
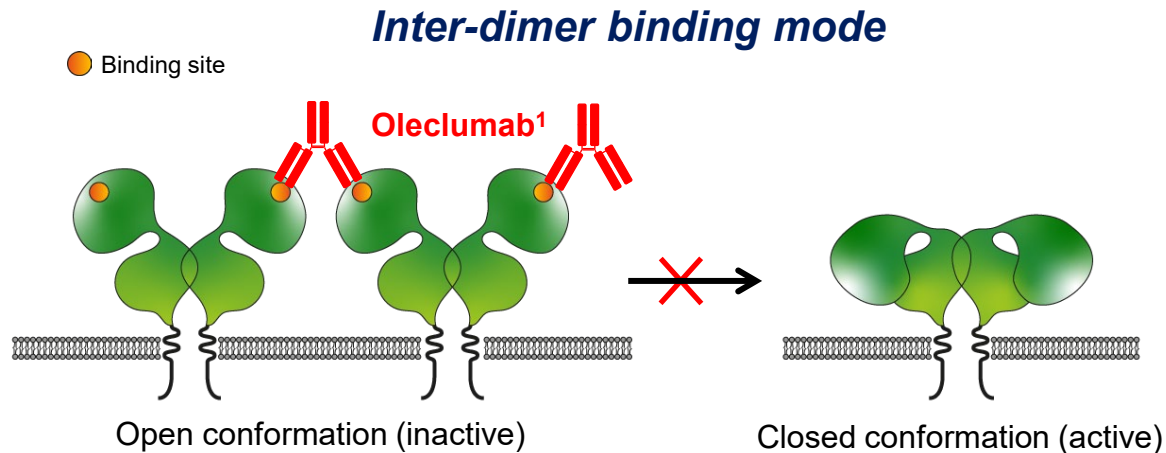
Unique intra-dimer binding through a C-terminus epitope

Dose-dependent CD73 inhibition without the “hook effect”²

Uiledlimab inhibits CD73 by binding to the **C-terminus** and preventing CD73 dimerization

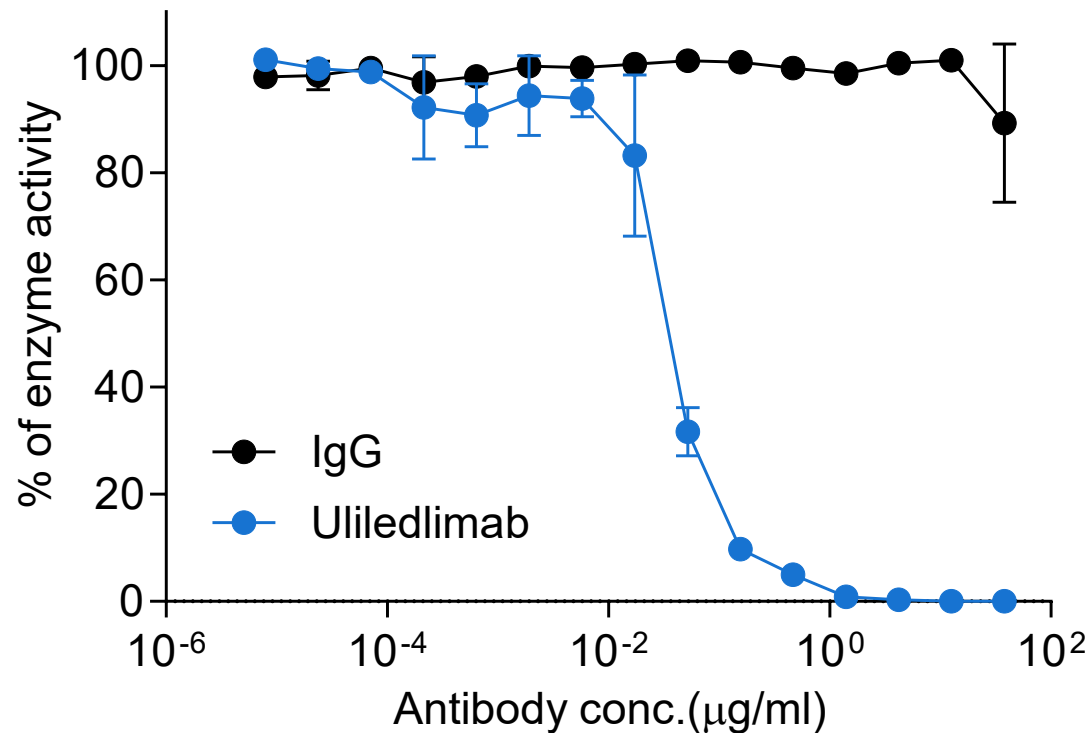


Oleclumab inhibits CD73 by binding to the **N-terminus** and preventing CD73 dimerization

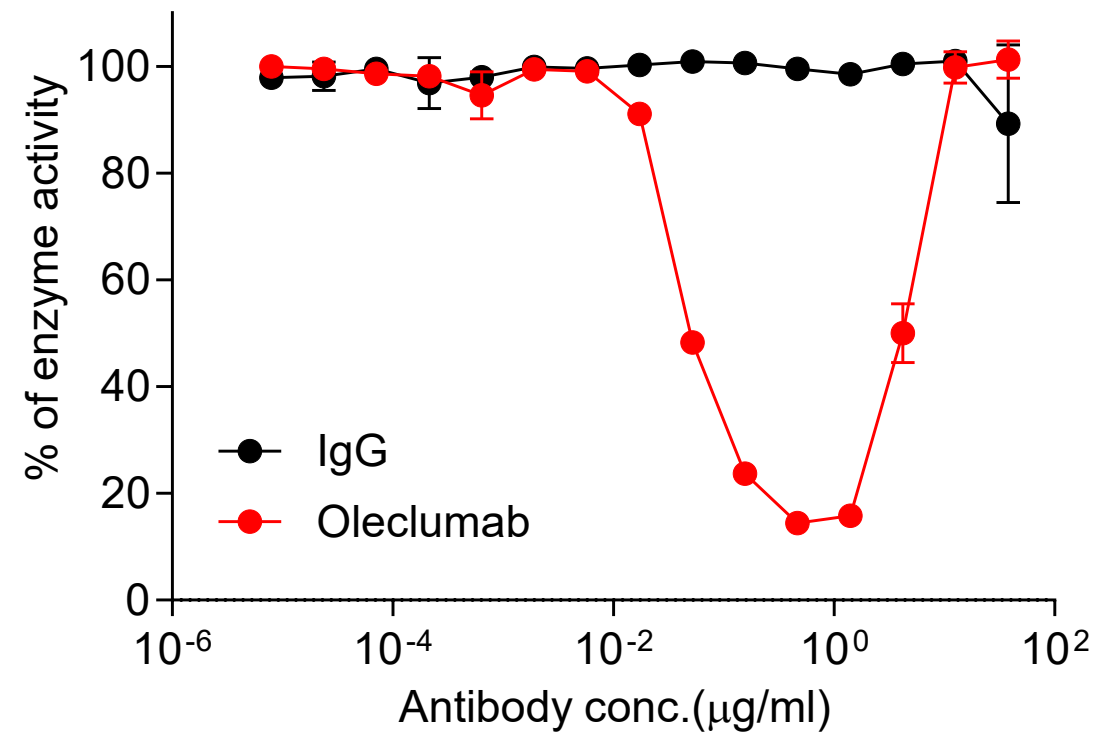


Uliledlimab May Completely Inhibit CD73 Function *in vitro*, Whereas Competitor Antibody Does Not

Complete inhibition by intra-dimer binding mode



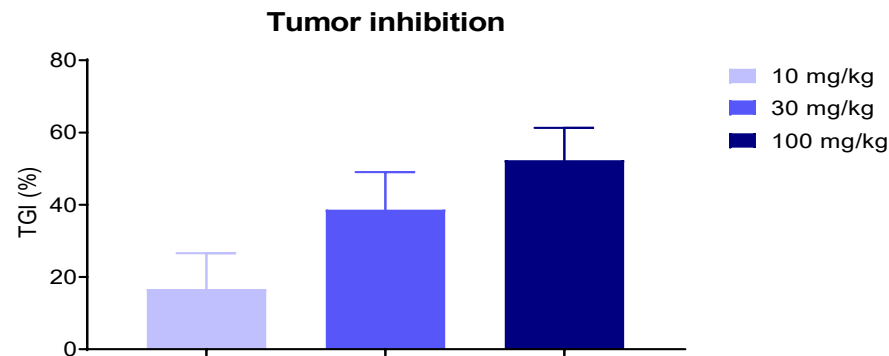
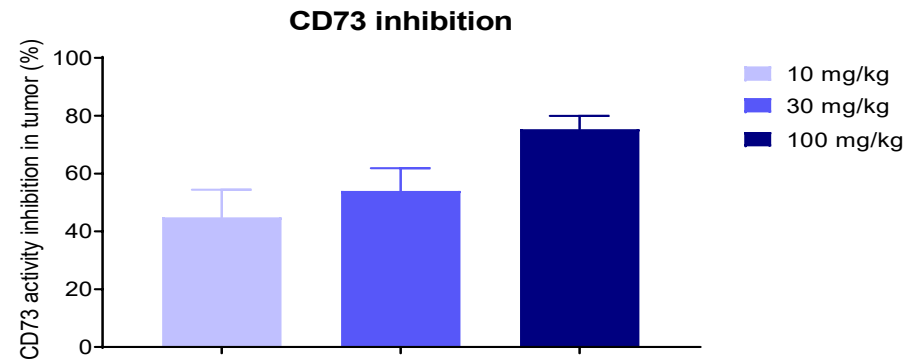
Partial inhibition by inter-dimer binding mode



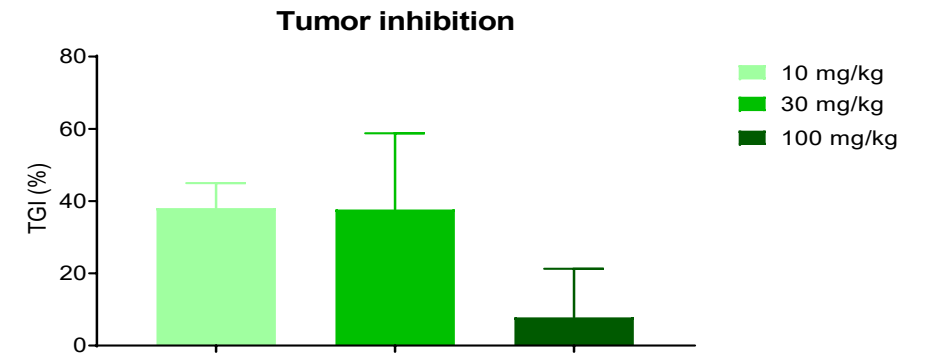
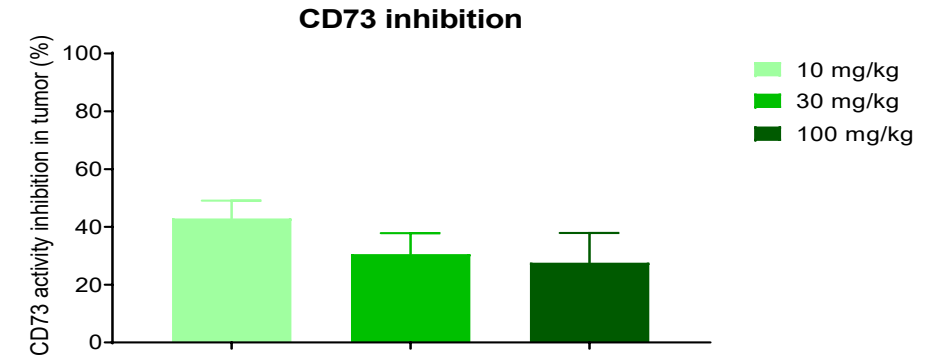
Inhibition of CD73 Activity & Tumor Growth is Dose-Dependent for Uliledlimab

Dose-dependency not observed for oleclumab

Inhibition of CD73 activity and tumor growth *in vivo* by uliledlimab is dose-dependent



Inhibition of CD73 activity and tumor growth *in vivo* is limited by oleclumab's hook effect biology



Uiledlimab + Toripalimab Data Support Patient Selection Based on CD73 Expression and Show Manageable Toxicity

Phase 2 ORR data from front-line NSCLC Cohort*

ORR% (n)	PD-L1 All	PD-L1 \geq 1%
CD73^{High}	53% (10/19)	63% (10/16)
CD73^{Low}	18% (8/45)	20% (5/25)
Pembro (KN-042) PD-L1\geq1%	NA	27% (174/637)

Safety observations for uiledlimab, administered to >200 patients in combination studies with CPIs

Safety profile of combination comparable to CPI monotherapy studies

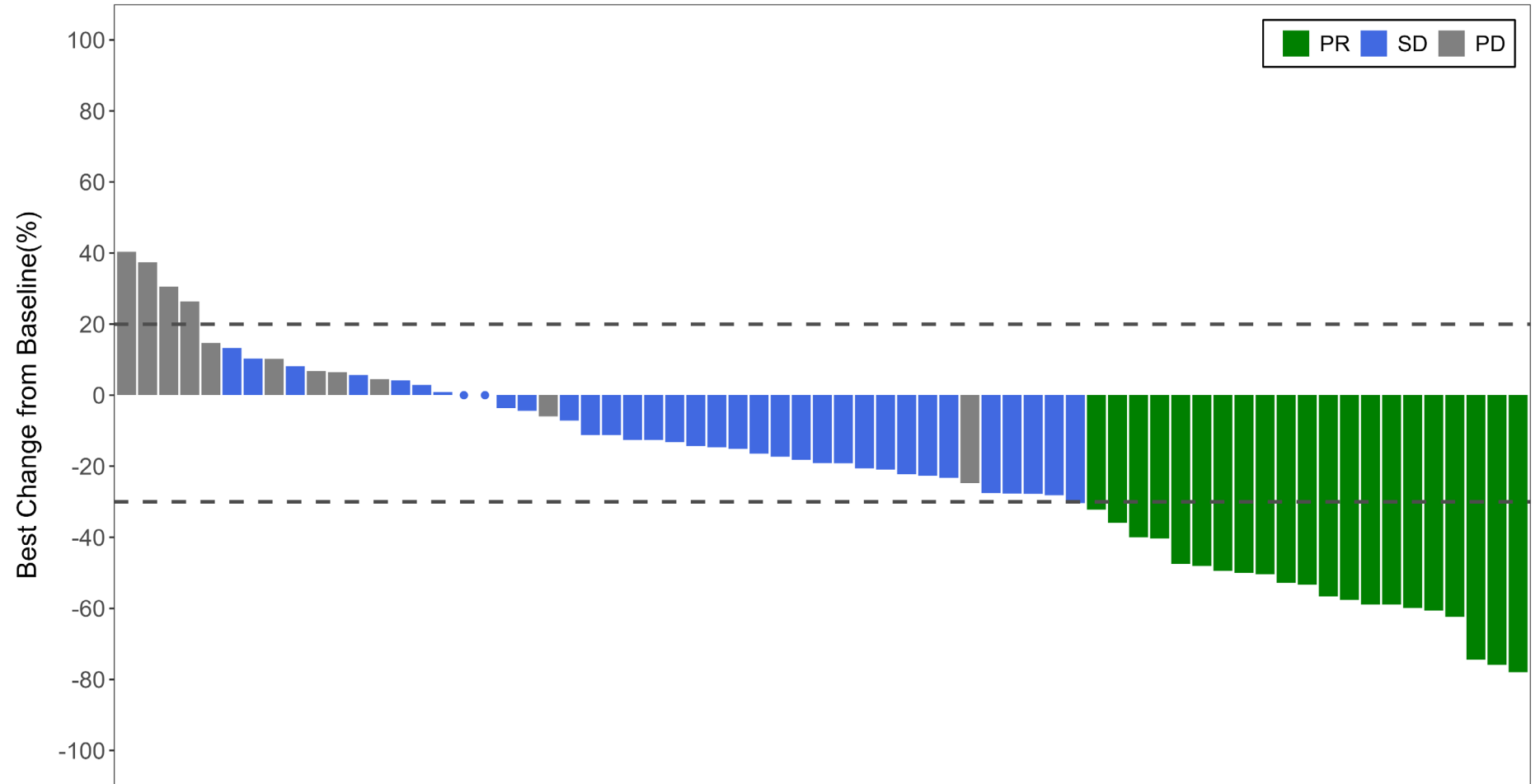


Well tolerated up to the highest doses tested (45mg/kg Q3W), without MTD

Most TRAEs/AEs were Grade 1 or 2

Early Phase 2 Data in Treatment-Naïve NSCLC Patients

Most Tumors
Decrease in
Size

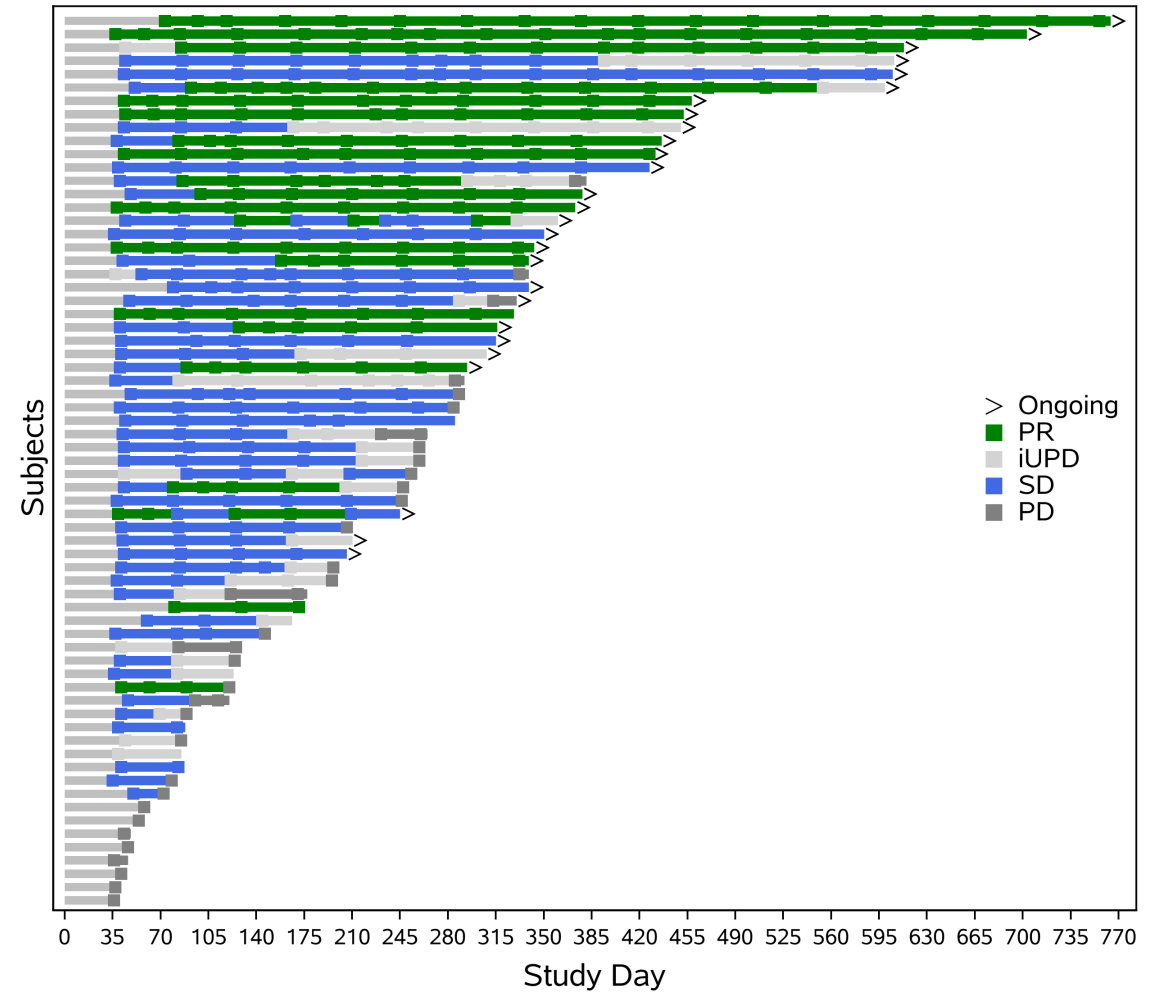


Data set time: 2023-08-10
The circles indicate the BOR of the two subject, which are SD.

Most Responses are Durable



18 of 21 patients with an objective response remain on treatment with a median follow-up of 10.8 months



Data set time: 2023-08-10

Rationale to Support Uliledlimab + Pembro + Chemotherapy in 1L mNSCLC

The addition of chemotherapy to IO monotherapy **extends the benefit of IO to lower levels of PD-L1 expression**

Uliledlimab has a **favorable toxicity profile** in combination with IO agents

Chemotherapy induces CD73 expression suggesting **additional benefit by combining uliledlimab with pembrolizumab + chemotherapy**¹

Based on this rationale, I-Mab plans to dose the first patient with **uliledlimab in combination with pembrolizumab + chemotherapy** in newly diagnosed patients with mNSCLC in 1H 2025

Uliedlimab Development Plan: Randomized Study Design for Combination with Pembrolizumab + Chemotherapy

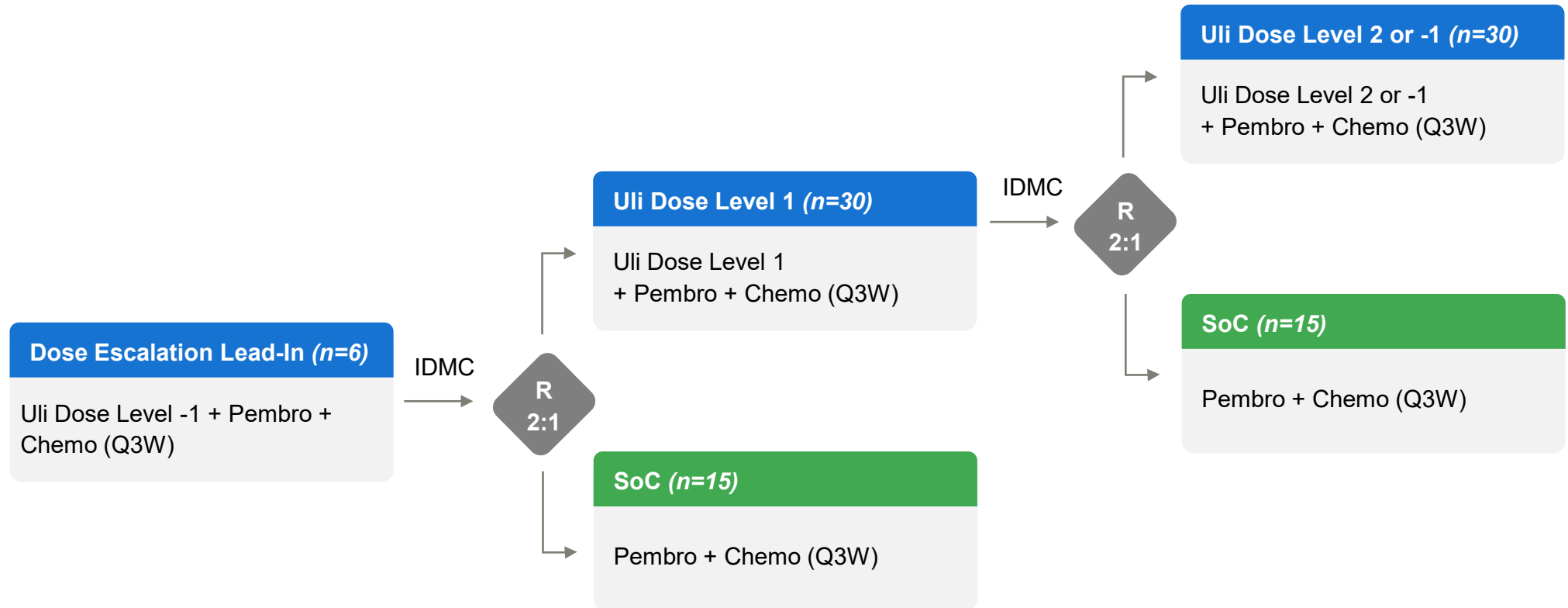
IND application cleared August 2024, on track to initiate enrollment in 1H 2025

Eligibility:

1L Advanced
mNSCLC
ECOG PS 0/1

Stratify By:

PD-L1 TPS
Histology
(n=96)

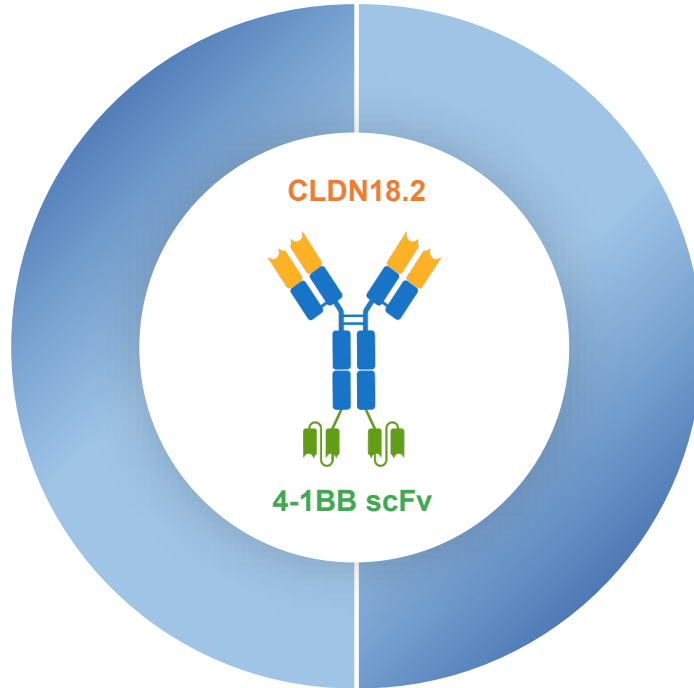


Endpoints

Primary: Safety, Efficacy (ORR) **Secondary:** PFS, DOR, OS

Givastomig (targeting Claudin 18.2 and 4-1BB)

Ongoing combination studies with nivolumab + chemotherapy across a wide range of Claudin 18.2 levels



Molecular Design

Binding activity demonstrated across **various levels of CLDN18.2 expression**

Higher-affinity binding to CLDN18.2 compared to reference antibody Zolbetuximab

Key Differentiation

Exhibits **CLDN18.2 binding** even on low expressing tumor cells

Localized T cell activation in TME to **minimize 4-1BB-mediated liver toxicity** and systemic immune response

Unique bispecific Ab integrates Claudin 18.2 as a tumor engager and 4-1BB as a conditional T cell activator

Phase 1 Monotherapy Responses in Heavily Pretreated Patients Provide Support for Further Studies

Patient Overview:

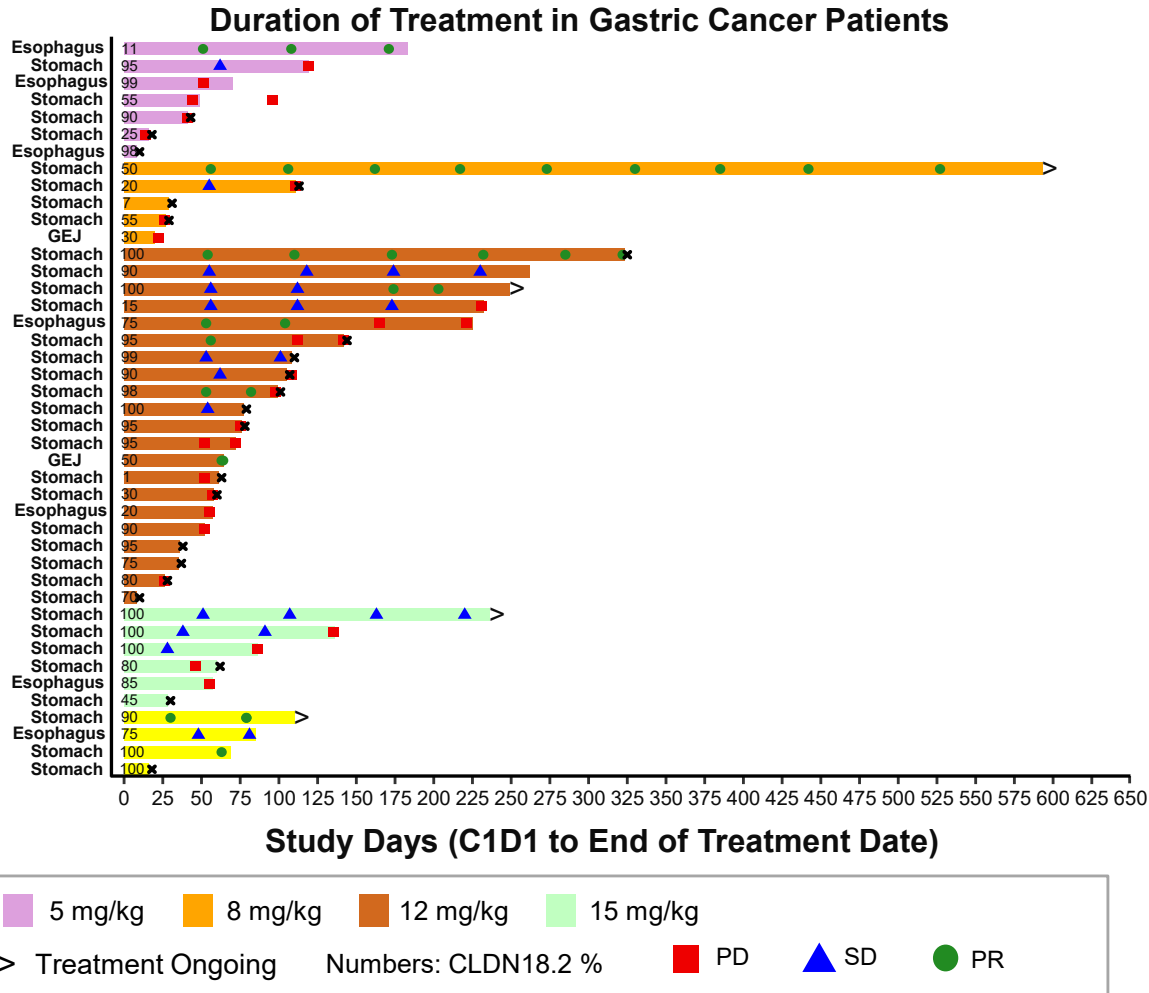
- 43 efficacy evaluable patients with CLDN18.2+ GC/GEJ/EAC
- Three median lines of prior treatment (range 1-6); dosed at 5-18 mg/kg¹
- Cohort is a subset of the Phase 1a (NCT04900818)

Responses:

- Seven partial response (PR) observed with an objective response rate (ORR) of 16.3% (7/43)
- Stable disease (SD) was reported in 14 patients, implying a disease control rate (DCR) of 48.8% (21/43)
- CLDN18.2 expression in responders ranged from 11% to 100%. Additionally, five responders had received prior treatment with PD-1 or PD-L1 inhibitors

Conclusion:

- Givastomig was well tolerated and exhibits monotherapy activity in heavily pre-treated GEC patients with a range of CLDN18.2 expression.**



Safety: Treatment Related AEs

Treatment-related adverse events (TRAEs) occurring in $\geq 5\%$ (n=43)

Preferred Term (all numbers are n(%))	Grade 1	Grade 2	Grade 3	Grade 4	Grade 5	All Grades
Nausea	6 (14.0)	4 (9.3)	1 (2.3)	-	-	11 (25.6)
Anemia	2 (4.7)	5 (11.6)	3 (7.0)	-	-	10 (23.3)
White blood cell count decreased	4 (9.3)	3 (7.0)	3 (7.0)	-	-	10 (23.3)
Vomiting	4 (9.3)	2 (4.7)	1 (2.3)	-	-	7 (16.3)
Decreased appetite	3 (7.0)	2 (4.7)	1 (2.3)	-	-	6 (14.0)
Alanine aminotransferase increased	2 (4.7)	2 (4.7)	1 (2.3)	-	-	5 (11.6)
Aspartate aminotransferase increased	3 (7.0)	-	2 (4.7)	-	-	5 (11.6)
Gamma-glutamyltransferase increased	1 (2.3)	3 (7.0)	1 (2.3)	-	-	5 (11.6)
Neutrophil count decreased	1 (2.3)	3 (7.0)	1 (2.3)	-	-	5 (11.6)
Infusion related reaction	1 (2.3)	2 (4.7)	1 (2.3)	-	-	4 (9.3)
Lymphocyte count decreased	-	-	4 (9.3)	-	-	4 (9.3)
Fatigue	2 (4.7)	1 (2.3)	-	-	-	3 (7.0)
Headache	2 (4.7)	1 (2.3)	-	-	-	3 (7.0)
Hypoalbuminemia	2 (4.7)	1 (2.3)	-	-	-	3 (7.0)
Lipase increased	1 (2.3)	1 (2.3)	1 (2.3)	-	-	3 (7.0)
Platelet count decreased	1 (2.3)	1 (2.3)	-	1 (2.3)	-	3 (7.0)
Weight decreased	2 (4.7)	1 (2.3)	-	-	-	3 (7.0)

- No DLT was reported up to 15 mg/kg Q2W and 18 mg/kg Q3W, and MTD was not reached
- Most commonly reported TRAEs (>20% of subjects): Grade 1, 2 or 3 nausea (25.6%), anemia (23.3%), white blood cell count decreased (23.3%)
- 15 subjects (34.9%) experienced at least one Grade ≥ 3 TRAE. This included one Grade 4 TRAE of platelet count decreased and no Grade 5 TRAEs
- Most gastrointestinal TRAEs were Grade 1 or 2 and do not appear to be dose-related

Givastomig Yields Better Monotherapy Responses in Patients with Low to High CLDN18.2 Expression Compared to Phase 1/2 Zolbetuximab Studies

Drug	Givastomig (bi-specific)	Zolbetuximab (CLDN 18.2 targeted mAb)	
Phase	Phase 1	Phase 1	Phase 2
CLDN18.2 – Expression of the Study Group	IHC ≥1+ in ≥1% cells	IHC ≥1+ in ≥1% cells	IHC ≥ 2+ in ≥ 50% cells
Diagnosis	Previously treated GC/GEJ/EAC	Previously treated GC/GEJ	Previously treated GC/GEJ/EAC
Efficacy Evaluable	43	15	43
ORR	16% (7/43)	Zero	9% (4/43)
DCR (CR+PR+SD)	49% (21/43)	1 SD	23% (10/43)
Source	Givastomig poster #1017P ESMO 2024	U Sahin et al. European Journal of Cancer 100 (2018) 17e26	O Tureci et al. Annals of Oncology 30: 1487–1495, 2019

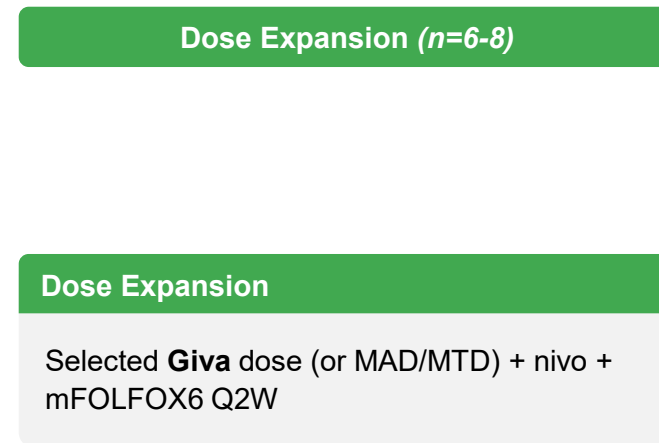
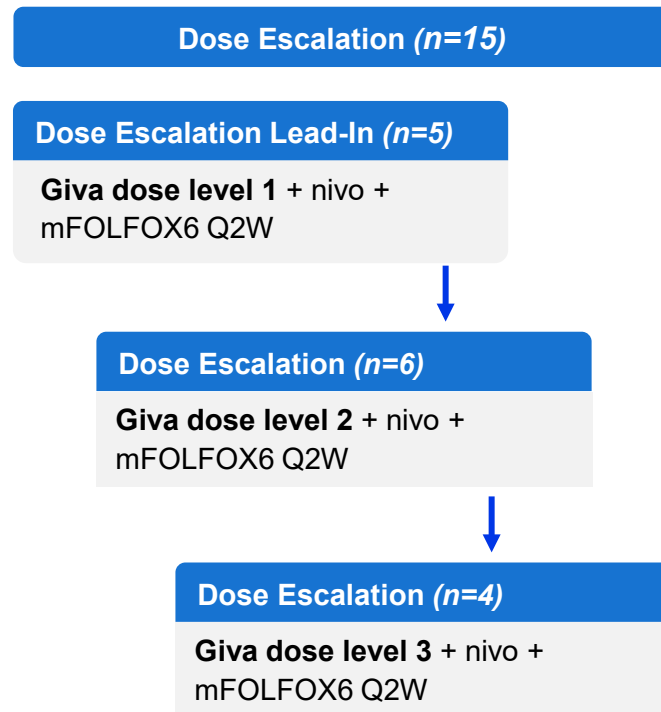
Potential Differentiations of Givastomig from Other Claudin 18.2 Targeted Competitors

	Givastomig (bi-specific)	Zolbetuximab (mAb) ¹	CMG901 (ADC) ²
Mechanism of Action	<p>CLDN18.2 dependent T cell activation in tumor</p> <p>4-1BB agonism to increase T cell expansion in tumor and reinvigorate exhausted T cells</p> <p>Bi-specific antibody designed to have conditional 4-1BB activation</p>	<p>Direct killing of CLDN18.2 tumor cells by ADCC may also release the tumor antigen</p>	<p>CLDN18.2 targeted chemotherapy and direct killing by ADCC</p> <p>Lysis of tumor cells by toxin can release the tumor antigen to mediate immune response</p>
Efficacy	~16% monotherapy ORR in previously treated CLDN18.2 + GC/GEJ/EAC	~10% monotherapy ORR in previously treated CLDN18.2 + GC/GEJ/EAC ¹	33% monotherapy ORR in previously treated CLDN18.2 + GC/GEJ
Safety	<p><5% Grade 3 neutropenia</p> <p><5% Grade 3 vomiting</p>	22% Grade 3 vomiting ¹	<p>20% Grade 3+ Neutropenia</p> <p>10% Grade 3 vomiting³</p>
Claudin 18.2 Targetable Expression	Extending to low levels of expression due to high affinity binding to CLDN18.2	Limited to targeting higher CLDN-expressing tumors	Likely limited to targeting high CLDN-expressing tumors

Givastomig Development Plan: Phase 1b Study Design for Combination with Nivolumab + Chemotherapy

Eligibility:

1L unresectable or metastatic GC/GEJ/EAC
HER2 negative
CLDN 18.2 $\geq 1+$ on $\geq 1\%$ of tumor cells



Endpoints:

Primary: Safety

Secondary: ORR, PK/PD, BoR, DoR, PFS, OS

Unique Bispecific Design Properties and Monotherapy Data in Gastric Cancers May Position Givastomig as Best-in-Class Claudin 18.2 bispecific

Unique Design to Enable Potential Wide Use Plus Favorable Initial Safety Profile

Bispecific design results in **CLDN18.2 conditional 4-1BB and T cell activation**, potentially limiting toxicity and inducing long-lasting immune memory response

Phase 1 dose escalation reached highest planned dose **without encountering DLT or liver toxicity signals**

Encouraging Responses in Previously Treated Patients, Including Those with Low CLDN18.2 Expression Levels

Objective responses seen in patients with gastric and esophageal cancer who had received multiple lines of prior treatment, including PD-(L)1, and exhibited low levels of CLDN18.2 expression

Response rate and tolerability supports combination in 1L SoC regimens

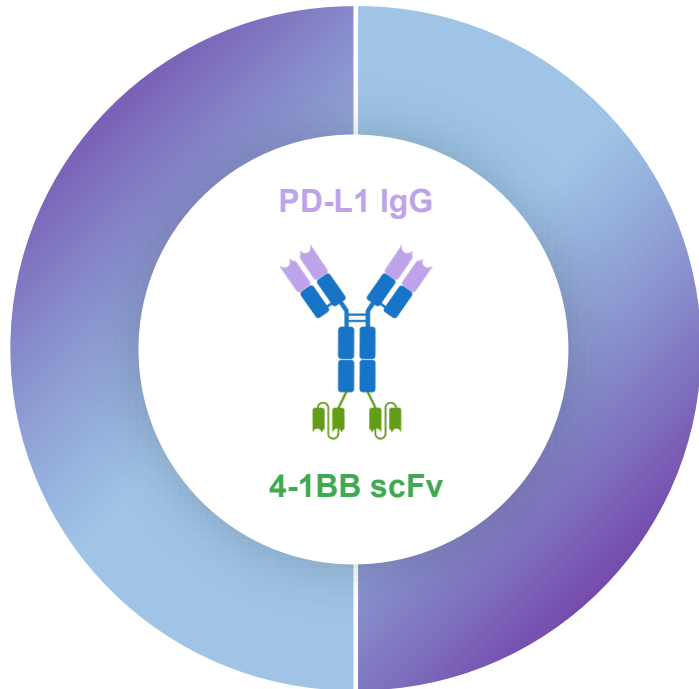
Dose Expansion Data and New Nivolumab + Chemotherapy Combo Study Ongoing

New dose expansion in combination with nivolumab + chemotherapy cohort study began in 1Q 2024 in treatment naïve patients with gastric cancers

Updated monotherapy dose expansion data in CLDN18.2+ patients with gastric cancers whose disease has progressed after previous treatment was presented at ESMO 2024

Ragistomig (ABL503/TJ-L14B, targeting PD-L1 and 4-1BB)

A novel bispecific integrates PD-L1 as a tumor engager and 4-1BB as a conditional T cell activator



Molecular Design

Molecule binds to PD-L1 to **inhibit PD-1/PD-L1 interaction**

PD-L1-dependent **4-1BB activation** at the tumor site

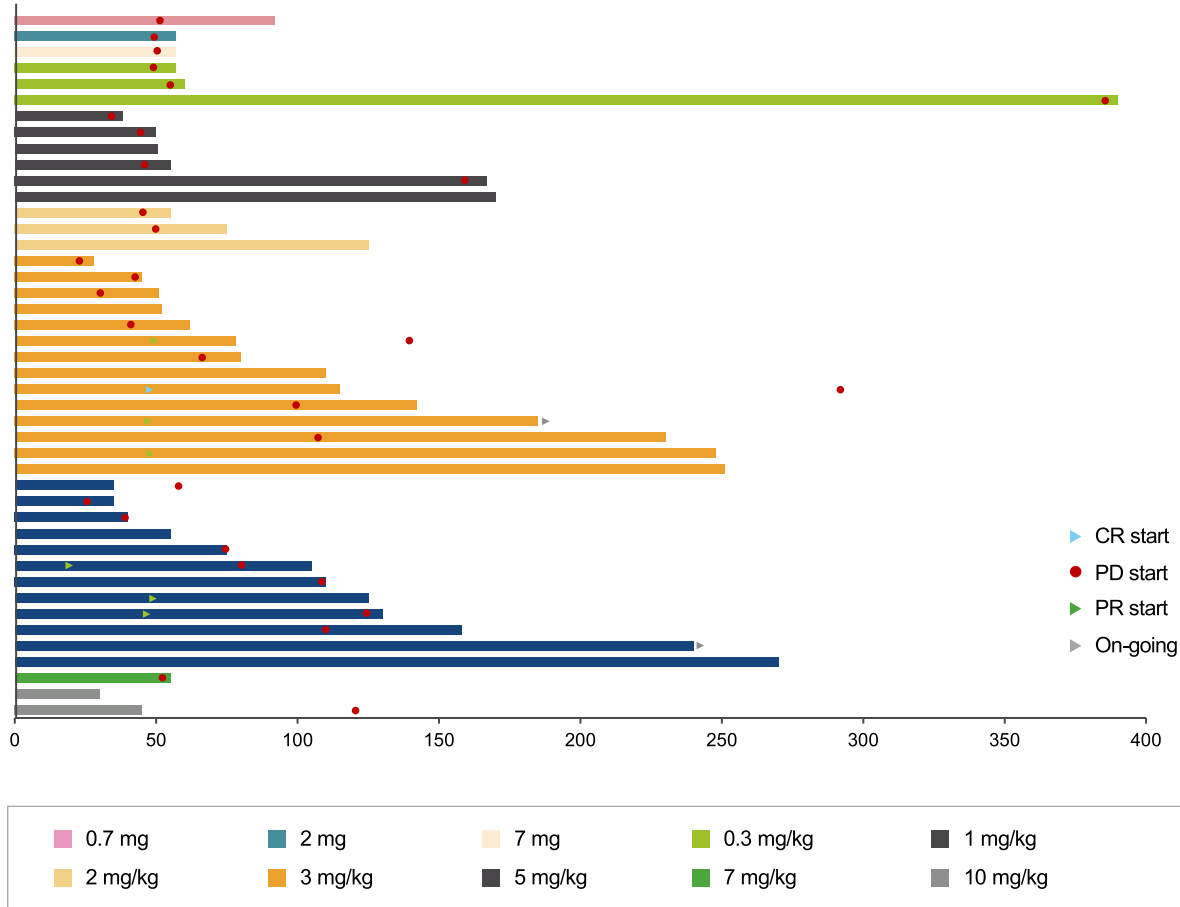
Target Drug Profile

- Targeting PD-L1+ tumor cells
- Blocking PD-L1/PD-1 immune inhibitory signaling
- Potent tumor-directed 4-1BB activation to enhance anti-tumor immunity
- Enhances anti-tumor immunity and re-invigorates exhausted T cells¹
- Localized 4-1BB activation in TME to mitigate liver toxicity and systemic immune response

Phase 1 efficacy data presented at ASCO 2024²

Phase 1 Data Support Further Development as a Monotherapy and in Combination with Other Agents

Treatment Duration (Days)



Overview:

- 44 efficacy evaluable patients (53 enrolled) with advanced or relapsed/refractory solid tumors (NCT04762641)
- 64.2% (34/53) of patients enrolled had at least three prior lines of systemic anti-cancer treatment

Efficacy Results at 3 and 5 mg/kg Q2W:

- Objective Response Rate (ORR) of 26.9% (7/26), Clinical Benefit Ratio (CBR) of 69.2% (18/26)
- One CR, six PRs, eleven SDs
- 71.4% of responders had received prior anti-PD-(L)-1 inhibitors
- The CR was observed in a heavily pretreated ovarian cancer patient dosed at 3 mg/kg (seven lines of prior therapy)

Conclusion:

- Compelling clinical data in checkpoint inhibitor relapsed/refractory and IO naïve patients**

Manageable Safety Profile

ABL503 monotherapy Demography	All patients (N = 53)	
	All grades, n(%)	Grade ≥ 3, n(%)
Any TRAE	40 (75.5)	22 (41.5)
TRAE occurring in ≥ 10% of patients		
Alanine aminotransferase increased	17 (32.1)	12 (22.6)
Aspartate aminotransferase increased	16 (30.2)	11 (20.8)
Pyrexia	8 (15.1)	1 (1.9)
Nausea	7 (13.2)	-
Rash	7 (13.2)	2 (3.8)
Fatigue	6 (11.3)	1 (1.9)
Platelet count decreased	6 (11.3)	1 (1.9)

- MTD established with 7 mg/kg every two-week dosing
- Most common TRAEs were increased ALT and increased AST
- None of the transaminase elevations were accompanied by clinically significant, treatment-related bilirubin increases
- Grade ≥ 3 ALT or AST increases occurred in 24.5% (13/53) of patients and improved with corticosteroids or ragistomig treatment interruption
- No cytokine release syndrome occurred, and one infusion-related reaction occurred at 5 mg/kg (Grade 2)

Ragistomig Results Compared to Acasunlimab Phase 1

	Ragistomig (ABL503)	Acasunlimab (GEN1046)
Phase	Phase 1 (NCT04762641)	Phase 1 (NCT03917381)
Treatment	Monotherapy 0.7 mg – 10 mg/kg, Q2W	Monotherapy 25 – 1,200 mg, Q3W
Diagnosis	Advanced or refractory solid tumors	Advanced or refractory solid tumors
Efficacy Evaluable	26 (sum of 3 mg/kg and 5 mg/kg)	61 (25 – 1,200 mg) 30 (80 – 200 mg)
ORR	26.9% (7/26)	6.6% (4/61) 13.3% (4/30, 80 – 200 mg)
DCR (CR+PR+SD)	69.2% (18/26)	65.6% (40/61)
Safety	Grade 3 AST / ALT: 24.5% (13/53)	Grade 3 AST / ALT: 10%
Source	Ragistomig poster ASCO 2024	Cancer Discovery 2022

Financial Information and Upcoming Milestones

Selected Financial Information

Cash, cash equivalents and short-term investments as of September 30, 2024, were **\$184.4M**

Expected cash runway into 2027 supporting multiple potential inflection points

Issued and outstanding ordinary shares of **187.5M representing the equivalent of 81.5M ADSs¹**

Recent and Anticipated Upcoming Milestones

Timing	Program	Milestone
Sep-2024	givastomig	Updated Phase 1 dose expansion data at ESMO 2024 Monotherapy (CLDN18.2+ patients with GC, GEJ, EAC) data
1H 2025	uliledlimab	First patient dosed in Phase 2 Randomized study in combination with pembrolizumab + chemo
2H 2025	uliledlimab	Phase 2 PFS data from uliledlimab + toripalimab Randomized study (TJ Bio China-only data)
2H 2025	givastomig	Phase 1b in combination with nivolumab + chemo Safety and ORR data in 1L GC, GEJ, EAC



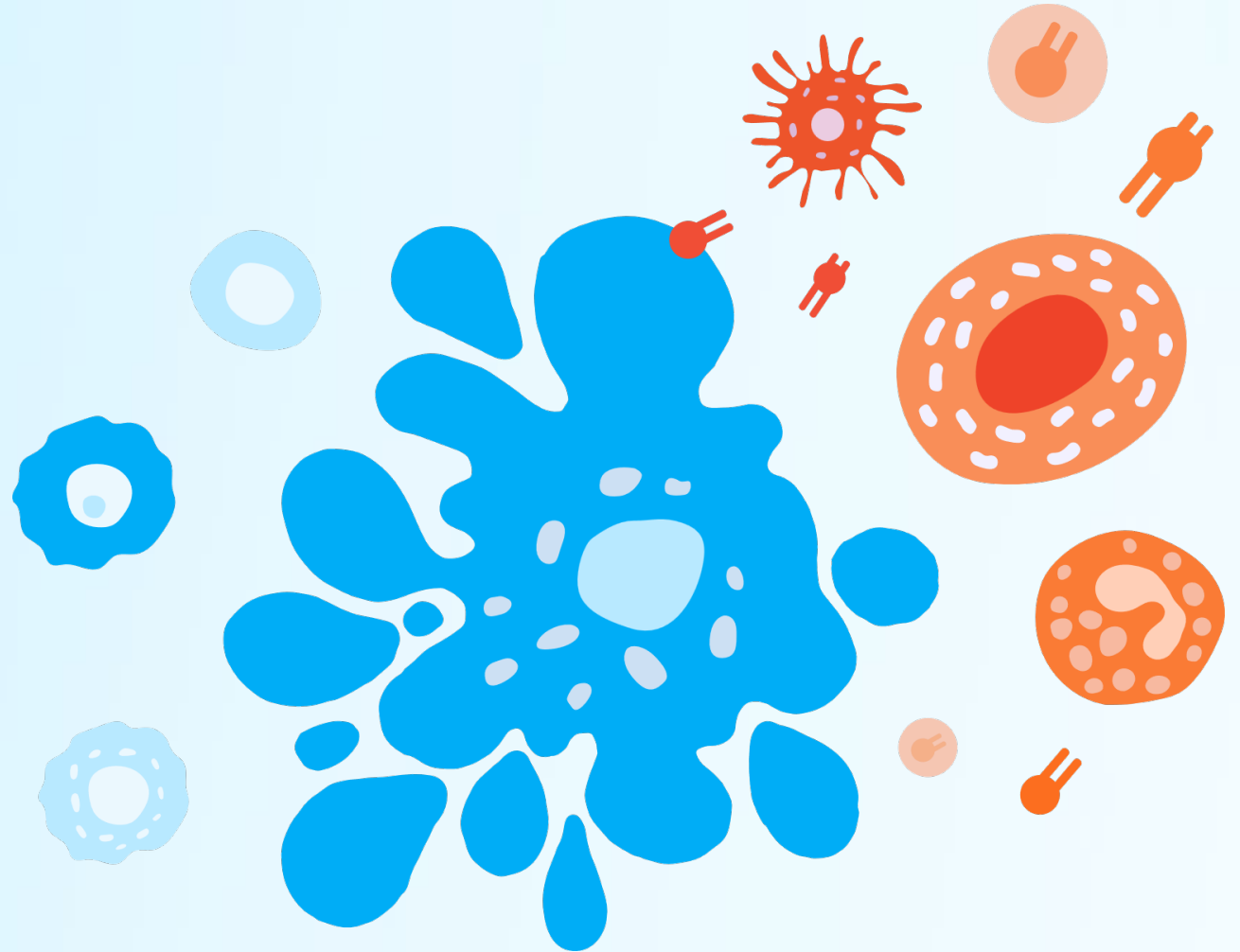
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