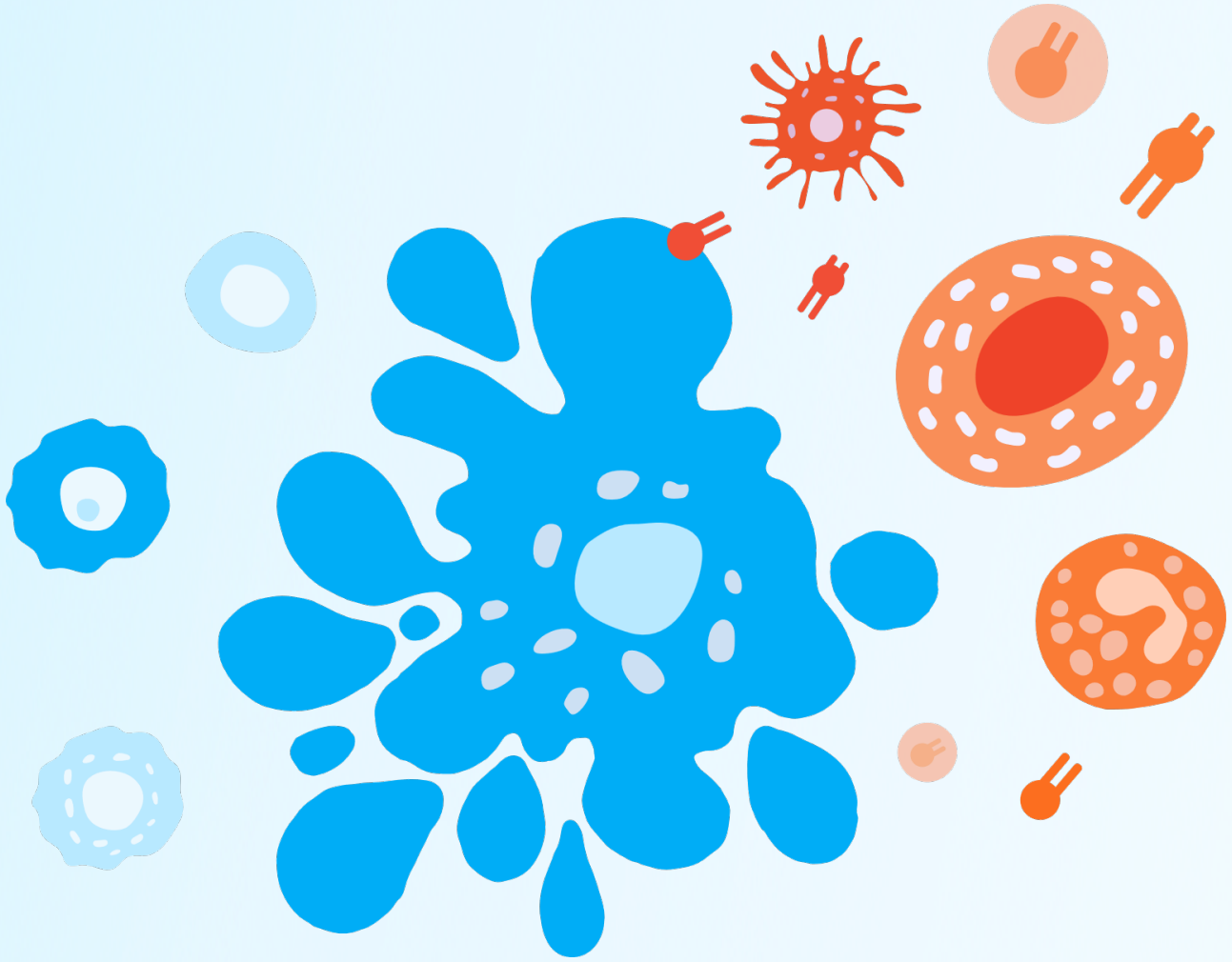
Transforming Potential into Reality

I-Mab Biopharma

April 2024



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I-Mab is Well-positioned for Meaningful Value Creation

A global biotech with an innovative portfolio and a healthy balance sheet

Advancing an Innovative Pipeline

Ulilediimab (**CD73**)

Givastomig (**CLDN 18.2 x 4-1BB**)

Ragistomig (**PD-L1 x 4-1BB**)

Emerging U.S. Entity

NASDAQ-listed

US-based Management Team

Headquartered in Rockville, MD

Divestiture of China Operations

Transaction closed April 2, 2024

Aggregate consideration of up to US$80M, contingent on certain future regulatory and sales-based milestones

Disciplined Capital Strategy

Cash balance of $321.8M[1] as of December 31, 2023

Reduced cash burn following divestiture to align resources to most promising programs

1. Cash position refers to cash, cash equivalents, and short-term investments

Notes: CLDN 18.2 = Claudin 18.2

Advancing a Differentiated and Commercially Attractive Pipeline

Numerous value-inflection milestones expected over the next two years

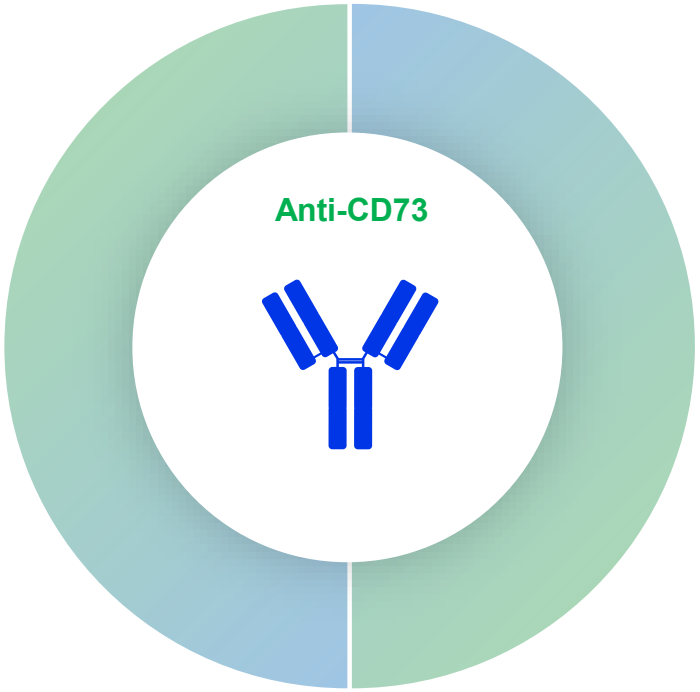
Asset	Phase 1	Phase 2	Phase 3	Market Opportunity	Status/Potential Next Steps
Uliledlimab CD73 Ab				Newly diagnosed stage 4 NSCLC: 300k+ patients[2]	**H1 2024**: New US IND for chemo + CPI combination for treatment-naïve NSCLC
Givastomig[1] CLDN 18.2 X 4-1BB Bispecific Ab				1L gastric cancer: Target population of 100k+[2]	**H1 2024**: New combo cohort initiation **H2 2024**: Phase 1 expansion data
Ragistomig/TJ-L14B[1] PD-L1 X 4-1BB Bispecific Ab				Refractory/relapsed cancers: PD-(L)1 progression impacts most patients with metastatic disease[2]	**H1 2024**: Phase 1 monotherapy data to be presented

1. Co-developed with ABL Bio
2. Global Data Epidemiology Data, Guidehouse legacy research

Notes: CPI = checkpoint inhibitors; NSCLC = non-small cell lung cancer; PD(L)-1 refers to inhibitors of PD-L1 or PD-1; Ab = antibody

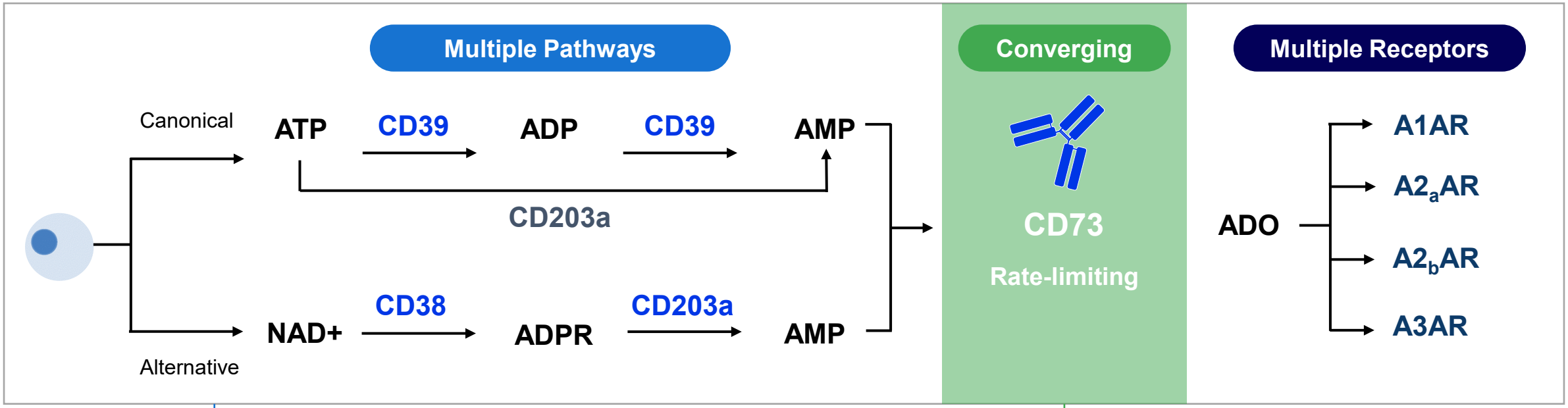
Ulilediimab (targeting CD73)

Initial development focused on newly diagnosed NSCLC with potential to expand across multiple indications in combination with immune checkpoint inhibitors



Molecular Design	Key Advantages
CD73 is the **non-redundant enzyme** in the adenosine immunosuppression pathway	Ulilediimab **completely inhibits** CD73 activity
Blocking CD73 activity leads to **complete inhibition of the adenosine pathway**	Phase 2 dose-response data with biomarker results strengthens our confidence in the MOA

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.

Source: I-MAB information on file

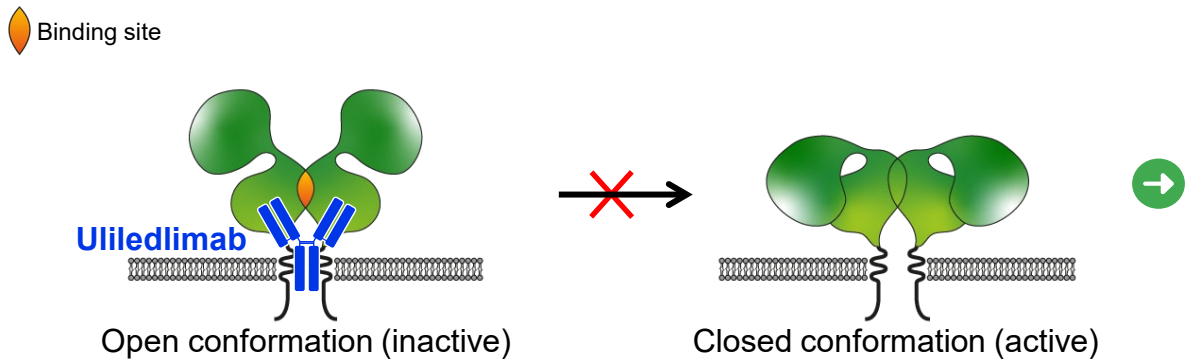
Ulileditimab: A Global CD73 Antibody with Differentiation

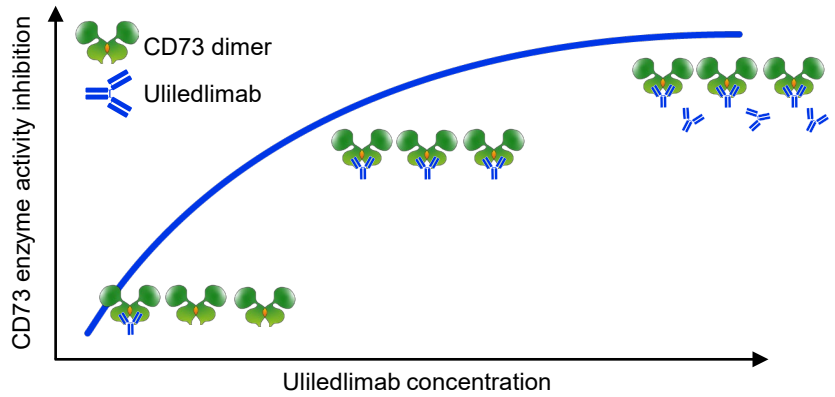
Unique intra-dimer binding through a C-terminus epitope

Complete CD73 inhibition without the "hook effect"

By binding to the C terminus, **ulileditimab** is functional through intra-dimer binding to single CD73 homodimer

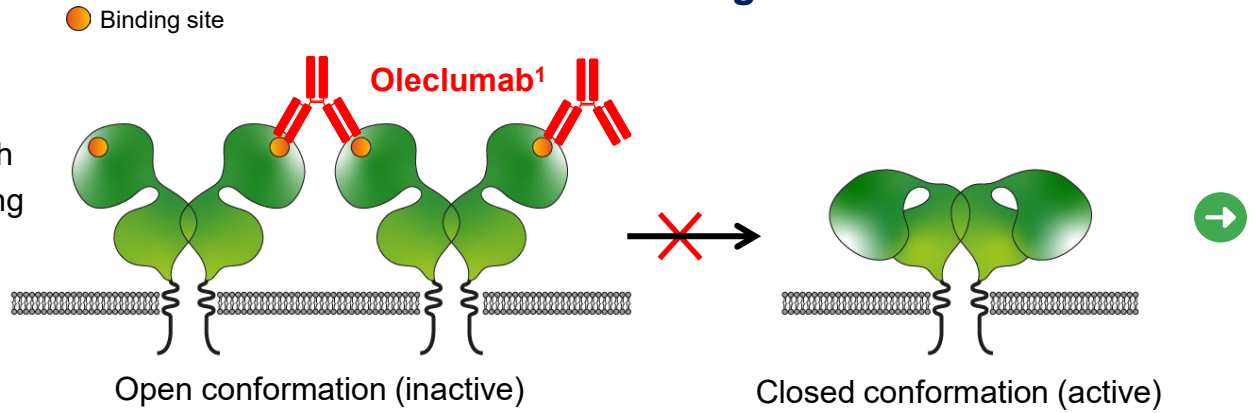
Intra-dimer binding mode

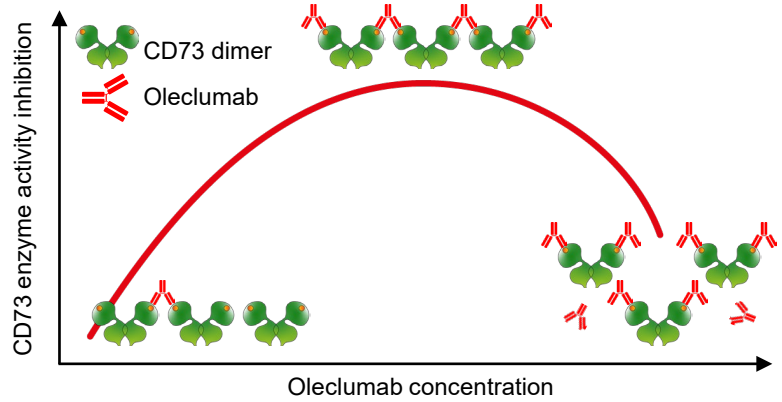




By binding to the N terminus, **oleclumab** is functional through inter-dimer binding to two CD73 homodimers

Inter-dimer binding mode



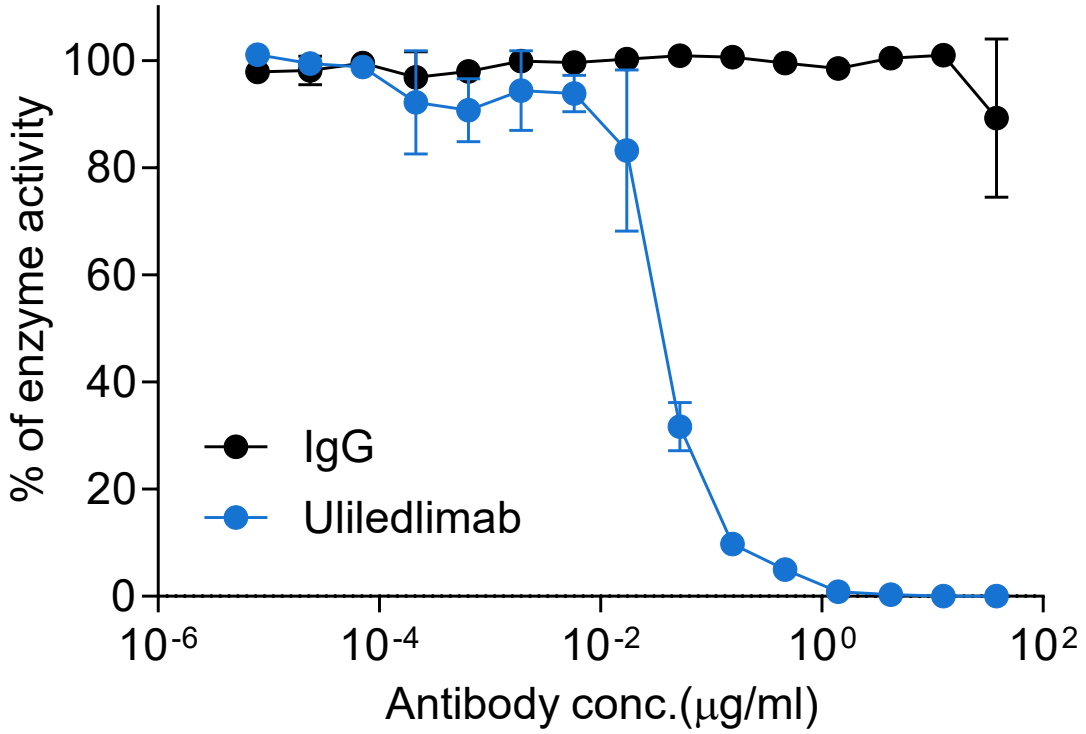


1. Oleclumab (MEDI9447) was internally produced based upon the published sequence

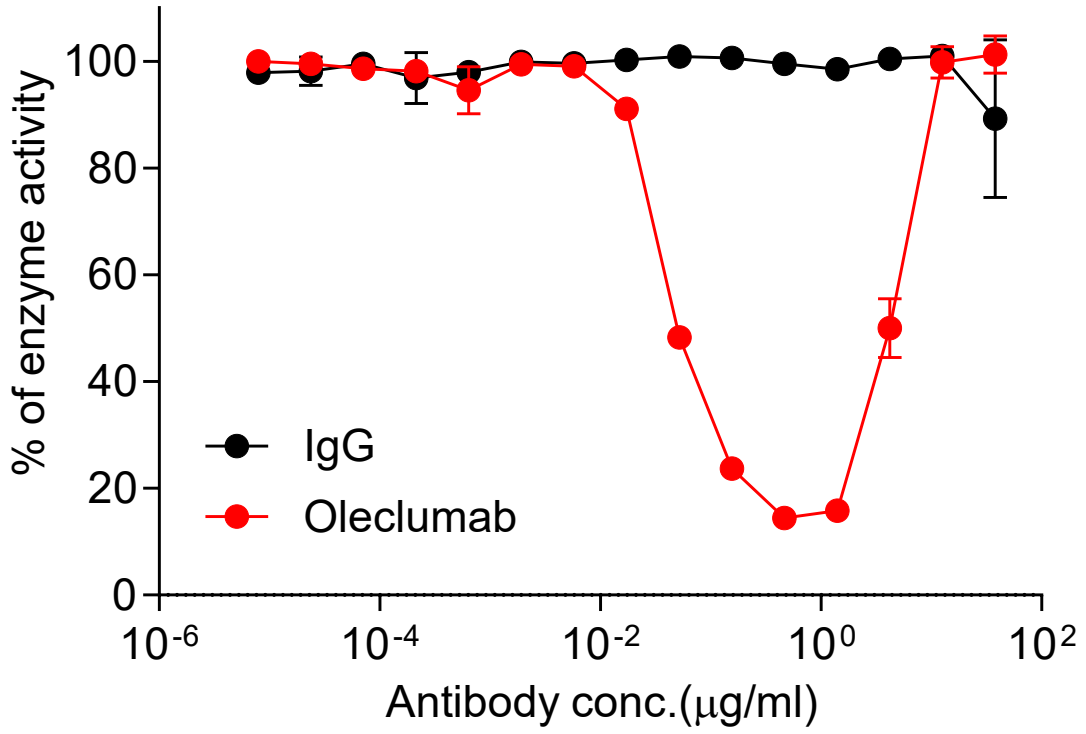
Source: I-MAB information on file

Ulileldlimab Can 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

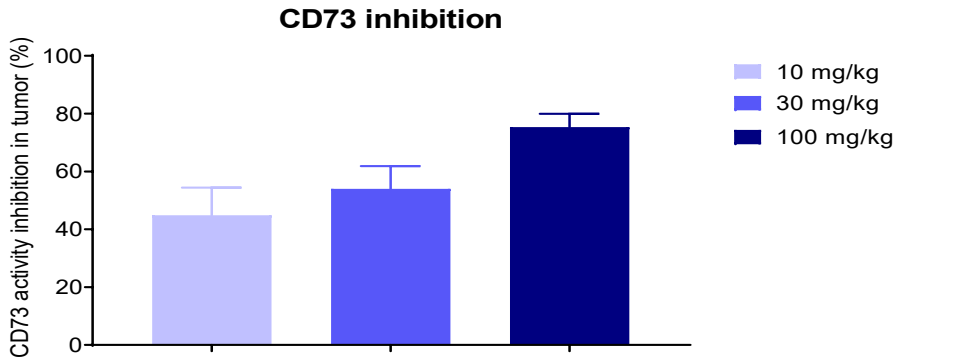


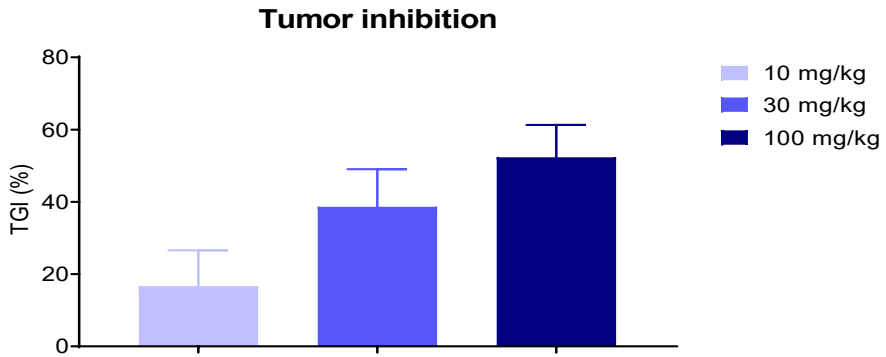
Astra Zeneca is evaluating oleclumab in a Phase 3 study in patients with Stage III NSCLC
Oleclumab (MEDI9447) was internally produced based upon the published sequence

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

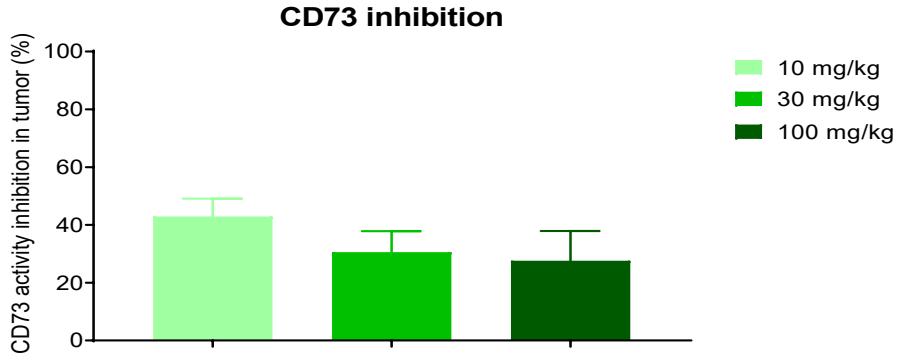
Dose-dependency not clearly observed for oleclumab

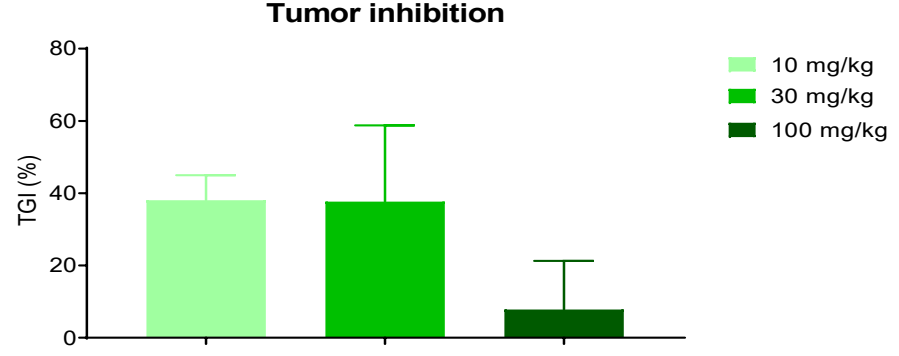
Inhibition of CD73 activity and tumor growth *in vivo* by ulilediimab is dose-dependent





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





Source: Data on file (IMAB), based on *in vivo* study on a PDX mouse model of NSCLC (LU5212, Crown Bioscience) in which CD73 inhibition in tumor was evaluated using an enzyme-histochemistry assay
Oleclumab (MEDI9447) was internally produced based upon the published sequence

Initial Anti-Tumor Data Supports Proof of Mechanism and Promising Safety

Phase 2 ORR Data from front-line NSCLC Cohort: 64 iRECIST-evaluable patients*

ORR% (n)	PD-L1 All (n=64)	PD-L1≥1% (n=41)
CD73^{High}	53% (10/19)	63% (10/16)
CD73^{Low}	18% (8/45)	20% (5/25)

Correlation of response with CD73 expression and PD-L1 levels suggest benefit driven by combination therapy

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

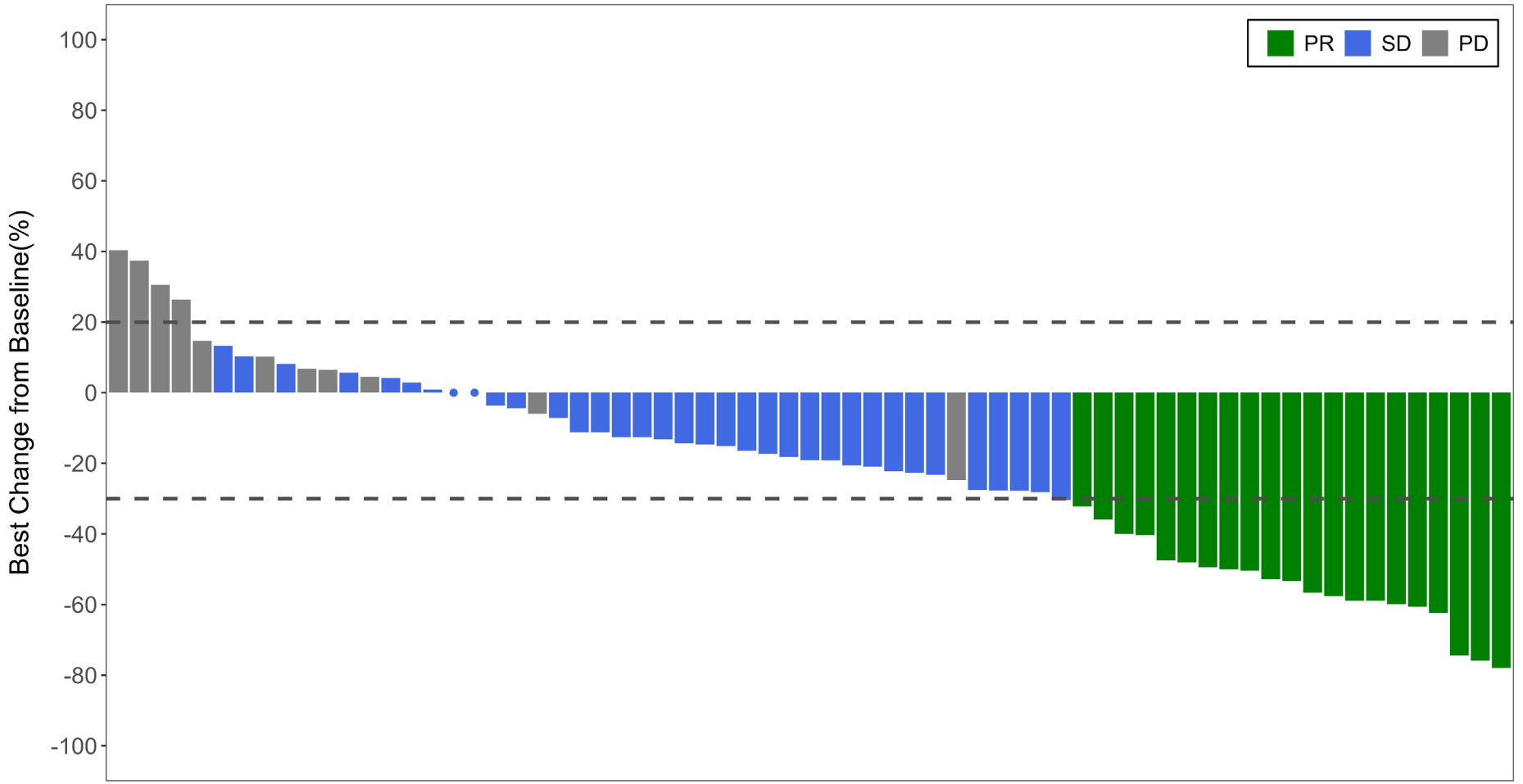
Initial safety profile of combination comparable to CPI monotherapy studies

Well tolerated up to the highest doses tested (30mg/kg Q3W), without MTD

Most TRAEs/AEs were Grade 1 or 2

Notes: ORR = objective response rate; MTD = maximally tolerated dose; Q3W = every three weeks; AE = adverse events; CPI = checkpoint inhibitors; TRAEs = treatment-related adverse events; ASCO23 = the American Society of Clinical Oncology 2023 Annual Meeting; toripalimab (used in this study) = Approved/China and the US (Shanghai Junshi Biosciences/Coherus Biosciences) *Patient disposition for slides 6-9 based on ASCO23 Poster from a cohort of 70 enrolled patients with unresectable/metastatic disease, including 67 efficacy evaluable and 64 patients who received at least one post baseline tumor assessment per iRECIST. Overall study (up to n=190) enrolled 5 cohorts (3 NSCLC sub-types, 1 ovarian, 1 all comers): data in this deck are from the treatment naïve, Stage 4 NSCLC patients.

Most Tumors Decrease in Size

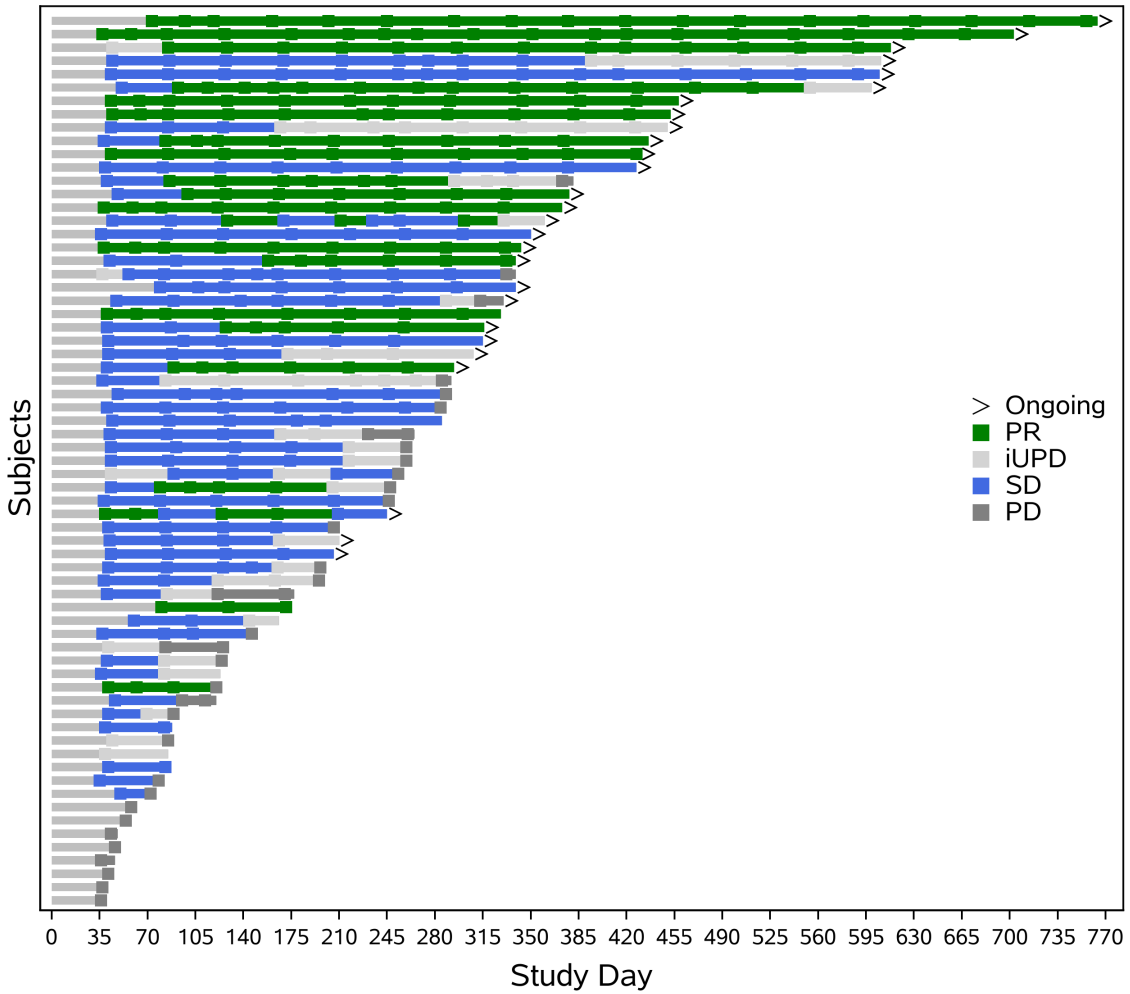


Data set time: 2023-08-10
The circles indicate the BOR of the two subject, which are SD.

Notes: Response definitions per iRECIST criteria. PR = partial response, SD = stable disease, PD = progressive disease, BOR = best overall response
Source: ASCO23 Poster

Most Responses are Durable

18 of 21 patients with an objective response remain on treatment with a median follow-up of 10.8 months[1]



Data set time: 2023-08-10

Notes: Response definitions per iRECIST criteria. PR = partial response, SD = stable disease, PD = progressive disease, iUPD = unconfirmed progressive disease, NE not evaluable. IHC = immunohistochemistry
Source: ASCO23 Poster

Emerging Data Indicate that Chemotherapy May Extend the Benefit of Ulilediimab to Patients Regardless of Baseline CD73 Expression

Expanding Therapeutic Reach

Combination of chemotherapy with a checkpoint inhibitor is a standard treatment approach across multiple advanced stage malignancies

Chemotherapy co-administration may increase the immunogenicity of cancer cells

Supporting Evidence

1. Keynote-189 and Keynote-407 studies both established that co-administration with chemotherapy extends the benefit of pembrolizumab to patients with <1% PD-L1 at baseline

2. Literature reports indicate that chemotherapy upregulates CD73 expression in cancer cells[1]

Strategic Clinical Design

I-Mab plans to submit an IND for **ulilediimab in combination with chemotherapy and checkpoint inhibitor** in newly diagnosed patients with advanced NSCLC in H1 2024

1. https://doi.org/10.1073/pnas.1718197115

Developing Ulileldlimab as an Immunotherapy Combination of Choice

Favorable Safety Profile as Monotherapy and in Combination with CPIs

- The lack of a hook effect could enable broad efficacy with optimized dosing
- Phase 2 data suggest ulileldlimab is safe and well tolerated up to the highest doses tested (45 mg/kg)

Encouraging Phase 2 NSCLC Responses Support Use in Combination Studies

- A 63% ORR observed in NSCLC patients with both high CD73 expression and PD-L1 TPS≥1% suggests that when tumors are vulnerable to PD-L1 inhibition, ulileldlimab appears to augment clinical responses
- Chemotherapy co-administration may broaden the patient population that benefits from ulileldlimab treatment

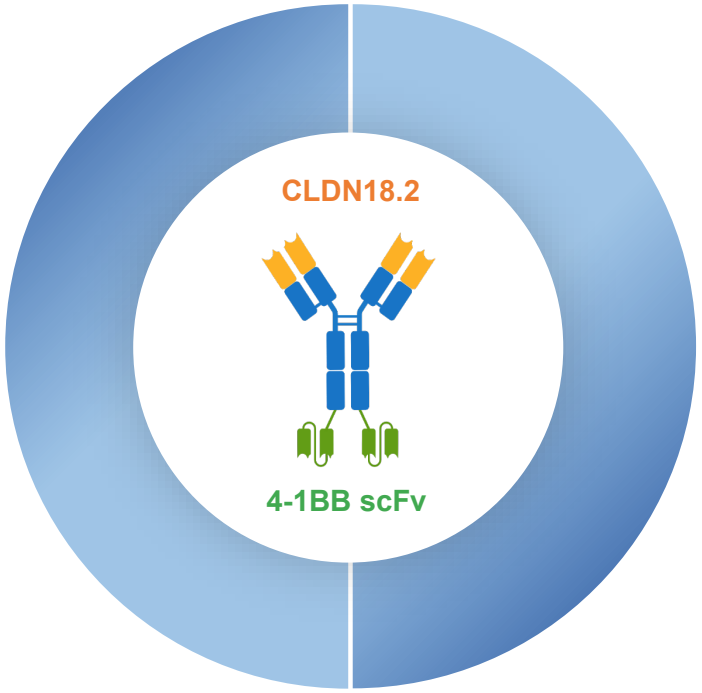
New Study Planned H1 2024

- A US IND submission for ulileldlimab in combination with chemotherapy and checkpoint inhibitors in newly diagnosed patients with stage 4 NSCLC is planned for H1 2024

Notes: TPS = tumor proportional score; CPI = checkpoint inhibitor; NSCLC = non-small cell lung cancer; ORR = overall response rate; IND = investigational new drug

Givastomig (targeting Claudin 18.2 and 4-1BB)

Potential to combine with checkpoint inhibitors and chemotherapy across a wide range of Claudin 18.2 levels

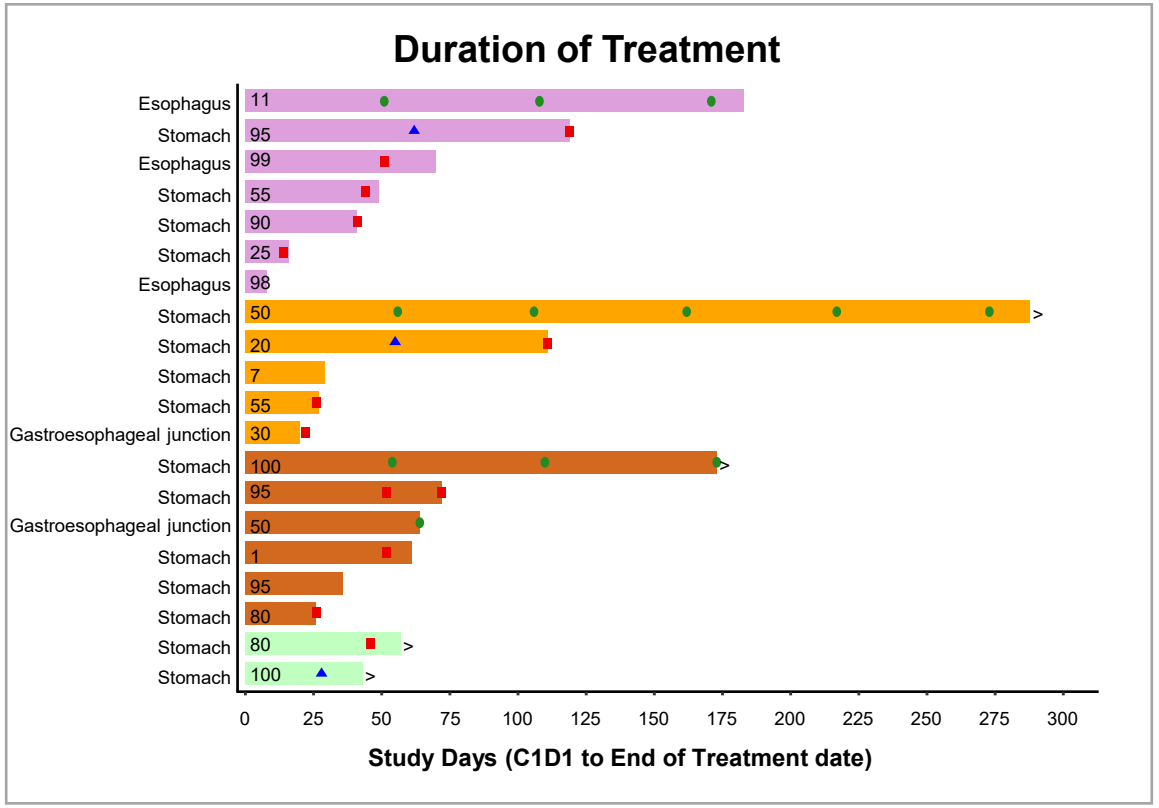


Molecular Design	Key Differentiation
Binding activity demonstrated across **various levels of CLDN18.2 expression**	Allows for **stronger CLDN18.2 binding** even in low expressing tumor cells
CLDN18.2-dependent T cell activation via 4-1BB pathway	4-1BB by conditional activation, **localized immune activation** to TME

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

Notes: scFv = single chain Fragment-variable region; TME = tumor microenvironment

Early Responses in Heavily Pretreated Patients Provides Compelling Support for Further Studies[1]



Patient Overview:

- 20 efficacy evaluable patients with CLDN18.2+ GC/GEJ/EAC
- Three median lines of prior treatment (range 1-10)
- Dosed at 5-15 mg/kg (defined as the predicted efficacious dosing range, based on preclinical studies)
- Cohort is a subset of the Phase 1a (NCT04900818)

Responses:

- Three partial response (PR) observed; two of those had received prior anti-PD-(L)1 therapy
- Stable disease (SD) observed in four patients. Of those, one had a PR on the first scan and subsequently withdrew from the study (counted as SD per RECIST1.1)
- An additional PR (not on the chart) was observed in a patient with head and neck squamous cell carcinoma receiving 12mg/kg who remains on study at 280 days

1. Source: ESMO 2023
Notes: GC = gastric cancers; GEJ = gastroesophageal junction; EAC = esophageal cancer

Safety: Treatment Related AEs[1]

Treatment-related adverse events (TRAEs) occurred in ≥5% (n=55)

Preferred Term (all numbers are n(%))	Grade 1	Grade 2	Grade 3	Grade 4	Grade 5	All Grades
Nausea	10 (18.2)	3 (5.5)	0	0	0	13 (23.6)
Vomiting	7 (12.7)	2 (3.6)	0	0	0	9 (16.4)
Fatigue	7 (12.7)	1 (1.8)	0	0	0	8 (14.5)
Anemia	1 (1.8)	4 (7.3)	1 (1.8)	0	0	6 (10.9)
Abdominal pain	2 (3.6)	1 (1.8)	0	0	0	3 (5.5)
Alanine aminotransferase increased	2 (3.6)	0	1 (1.8)	0	0	3 (5.5)
Diarrhea	3 (5.5)	0	0	0	0	3 (5.5)
Headache	1 (1.8)	2 (3.6)	0	0	0	3 (5.5)
Lymphocyte count decreased	1 (1.8)	1 (1.8)	1 (1.8)	0	0	3 (5.5)
Pruritus	2 (3.6)	0	1 (1.8)	0	0	3 (5.5)
Pyrexia	3 (5.5)	0	0	0	0	3 (5.5)
White blood cell count decreased	0	2 (3.6)	1 (1.8)	0	0	3 (5.5)

- No DLT was reported up to 15mg/kg, and MTD was not reached
- Most commonly reported TRAEs (>10% of subjects): Grade 1 or 2 nausea (23.6%), vomiting (16.4%), fatigue (14.5%), anemia (10.9%)
- 10 subjects (18.2%) experienced at least one Grade 3 TRAE. No Grade 3 TRAEs occurred in more than one subject
- Onset of gastrointestinal TRAEs: generally, after 14 days of treatment, recovery within one week; none led to drug withdrawal

1. Source: ESMO 2023

Notes: DLT = dose-limiting toxicity, MTD = maximum tolerated dose

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

Drug	Givastomig (bi-specific)	Zolbetuximab (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	20	15	43
ORR	**15%** (3/20)	0	9% (4/43)
DCR (CR+PR+SD)	**35%** (7/20)	1 SD	23% (10/43)
Source	Givastomig poster #1039P ESMO 2023	U Sahin et al. European Journal of Cancer 100 (2018) 17e26	O Tureci et al. Annals of Oncology 30: 1487–1495, 2019

Notes: Zolbetuximab (Astellas) is currently under review with the FDA; mAb = monoclonal antibody; ORR = overall response rate; DCR = disease control rate; CR = complete response; PR = partial response; SD = stable disease; GC = gastric cancers; GEJ = gastroesophageal junction; EAC = esophageal cancer

Potential Differentiations of Givastomig from Other Claudin 18.2 Targeted Competitors

	Givastomig	ADCs	CLDN18.2 mAb
MoA of Monotherapy	CLDN18.2 dependent T cell activation in tumor 4-1BB agonism to increase T cell expansion in tumor and reinvigorate exhausted T cells Bi-specific antibody designed to have conditional 4-1BB activation	CLDN18.2 targeted chemotherapy and direct killing by ADCC Lysis of tumor cells by toxin can release the tumor antigen to mediate immune response	Direct killing of CLDN18.2 tumor cells by ADCC may also release the tumor antigen
Efficacy	~20% monotherapy ORR in previously treated CLDN18.2 + GC/GEJ/EC	33% monotherapy ORR in previously treated CLDN18.2 + GC/GEJ[2]	~10% monotherapy ORR in previously treated CLDN18.2 + GC/GEJ/EC[4]
Safety	No Grade 3 neutropenia No Grade 3 vomiting	20% Grade 3+ Neutropenia 10% Grade 3 vomiting[3]	22% Grade 3 vomiting[4]
Claudin 18.2 Targetable Expression	Broad expression contributed by Giva-mediated bystander tumor-killing[1]	Higher expression v. normal gastric mucosa	Higher expression v. normal gastric mucosa

1. Givastomig-mediated T cell activation by CLDN18.2-positive tumor cells leads to the killing of nearby CLDN18.2-negative tumor cells.
2. ADC efficacy:
3. ASCO Plenary Series 2023 (Note: Examples reported are from representative molecules within ADC class as not all ADCs will have these specific numbers;
4. Annals of Oncology

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

Unique Design To Enable Wide Use Plus Favorable Initial Safety Profile

Bispecific design to bind across **various levels of CLDN18.2** and **conditional T cell activator,** 4-1BB, could induce long-lasting immune memory response and enable superior anti-tumor activity at the tumor site

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

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

CLDN 18.2 assay for patient selection is in development with a partner

Dose Expansion Data and New Chemotherapy/CPI Combo Planned for 2024

Phase 1 monotherapy data presented at ESMO 2023

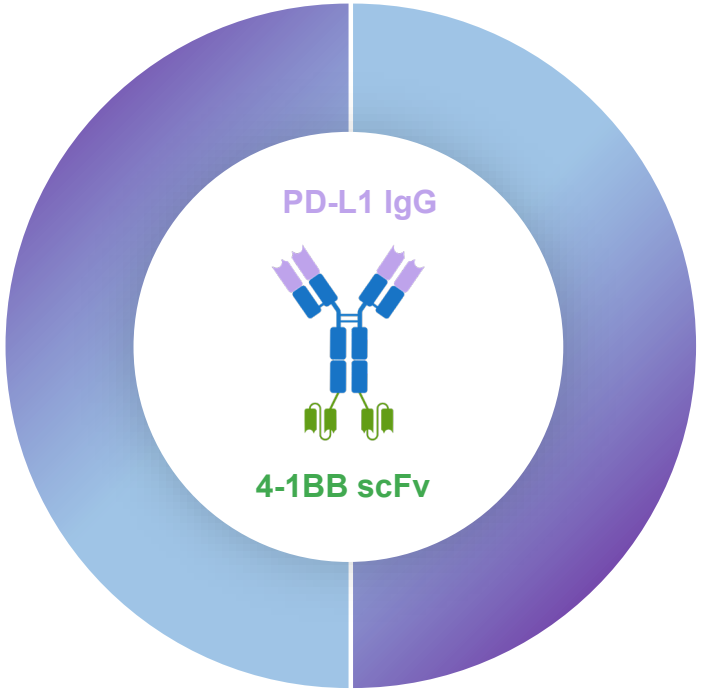
New dose expansion chemotherapy/CPI cohort study began in H1 2024 in treatment naïve patients with gastric cancers

Interim monotherapy dose expansion data planned in H2 2024 in CLDN18.2+ patients with gastric cancers whose disease has progressed after previous treatment

Notes: Gastric cancers = gastric, gastroesophageal junction and esophageal cancer

Ragistomig (TJ-L14B, targeting PD-L1 and 4-1BB)[1]

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



Molecular Design	Key Differentiation
Designed to treat **PD-(L)1 resistant cancers**	Targeting PD-L1+ tumor cells and blocking PD-L1/PD-1 interaction
PD-L1-dependent **conditional 4-1BB activation** via tumor site	4-1BB by conditional activation, **localized immune activation** to TME

1. ABL503

Notes: scFv = single chain Fragment-variable region; TME = tumor microenvironment

Unique Bispecific Design Properties and Monotherapy Data in R/R Patients

Unique Design To Enable Broader Use Plus Favorable Initial Safety Profile

- **Bispecific design** to **stimulate 4-1BB** activation **in the presence of PD-L1** expressing tumor cells to minimize off-tumor toxicity
- Dose expansion is ongoing with preliminary efficacy signals; the MTD has not yet been reached
- Additional tumor cohorts are planned as well

Preliminary Efficacy Signals Observed

- **Objective responses** seen in patients with progressive, locally advanced, or metastatic solid tumors that are relapsed or refractory following prior lines of treatment
- **One CR, One PR, Two uPR (n=14)** observed based on recent enrollment

Top-line Phase 1 Clinical Data Expected in H1 2024

- **Phase 1 monotherapy data** to be presented in H1 2024

Notes: MTD = maximum tolerated dose, CR = complete response, PR = partial response; uPR = unconfirmed partial response. R/R = relapsed/refractory. Study design per clinicaltrials.gov

I-Mab Portfolio Projected to Substantially Advance Over the Next 12 Months

Key milestones starting in H1 2024

Timing	Program	Milestone
H1 2024	ulilediimab	**US IND submission:** chemotherapy/CPI combo study (patients with newly diagnosed, stage 4 NSCLC)
H1 2024	givastomig	**New dose expansion cohort began enrollment:** chemotherapy/CPI regimens (patients with gastric, GEJ, and esophageal cancers)
H1 2024	ragistomig	**Phase 1 monotherapy data:** Presentation of dose-escalation data
H2 2024	givastomig	**Interim P1 dose expansion data presentation:** monotherapy (CLDN18.2+ patients with gastric, GEJ, and esophageal cancers)

Notes: CPI = checkpoint inhibitor, CLDN = Claudin, NSCLC = non-small cell lung cancer, GEJ = gastroesophageal junction

I-Mab is Well-positioned for Meaningful Value Creation

A global biotech with an innovative portfolio and a healthy balance sheet

Advancing an Innovative Pipeline

Ulilediimab (**CD73**)

Givastomig (**CLDN 18.2 x 4-1BB**)

Ragistomig (**PD-L1 x 4-1BB**)

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NASDAQ-listed

US-based Management Team

Headquartered in Rockville, MD

Divestiture of China Operations

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Disciplined Capital Strategy

Cash balance of $321.8M[1] as of December 31, 2023

Reduced cash burn following divestiture to align resources to most promising programs

1. Cash position refers to cash, cash equivalents, and short-term investments

Notes: CLDN 18.2 = Claudin 18.2

I-Mab Biopharma

IR Contact

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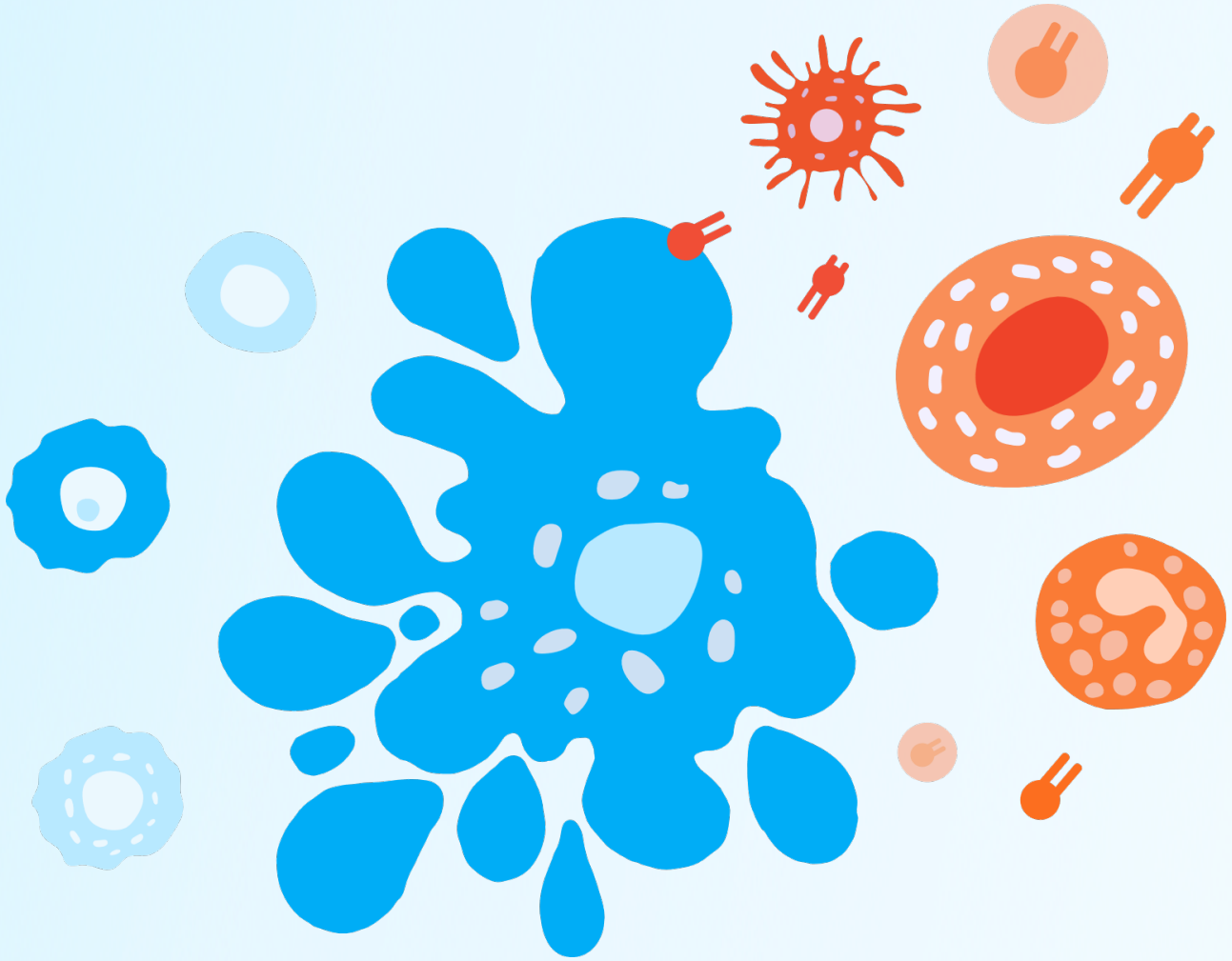

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