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SEHK: 6996.HK

Introduction to Antengene

Jefferies Global Healthcare Conference 2025

June 2025

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Antengene Pipeline Overview

Antengene (6996.HK)

Partnerships/Clinical Collaborations

Celgene | Bristol Myers Squibb Company · MERCK · Karyopharm Therapeutics · BeiGene · 君实生物 TopAlliance

Best-in-Class Global Programs

ATG-022
(Claudin 18.2 ADC)

CLDN18.2 **Moderate to High Expressing** GC **(IHC 2+ > 20%)[1]: 40% ORR (12/30)** and **90% DCR (27/30)**

CLDN18.2 **Low and Ultra-low Expressing** GC **(IHC 2+ ≤ 20%)[2]: 30% ORR (3/10)** and **50% DCR (5/10);** Incl. 2 SDs with 24% and 18% tumor shrinkages respectively

Phase II Dose Expansion (CLDN18.2+ GC & Other Solid Tumors)

Global Clinical Collaboration with MSD (Combo with Pembrolizumab)

ATG-037[4]
(CD73 Small Molecule Inhibitor)

CPI-resistant **Melanoma: 36.4% ORR (4/11)** and **100% DCR (11/11)**

CPI-resistant **NSCLC: 22.2% ORR (2/9)** and **67% DCR (6/9)**

Phase II Dose Expansion (CPI-resistant melanoma & NSCLC)

Global Clinical Collaboration with MSD (Combo with Pembrolizumab)

AnTenGager™ "2+1" T Cell Engager Platform with Steric Hindrance-masking Technology

Autoimmune Programs:

ATG-201 (CD19 x CD3)
B Cell Related Autoimmune Diseases

Undisclosed Trispecific Program

Oncology Programs:

ATG-106 (CDH6 x CD3)
Ovarian Cancer & Kidney Cancer

ATG-110 (LY6G6D x CD3)
Microsatellite Stable (MSS) Colorectal Cancer

ATG-112 (ALPPL2 x CD3)
Gynecological Tumors and Lung Cancer

ATG-102 (LILRB4 x CD3)
Acute Myeloid Leukemia & Chronic Myelomonocytic Leukemia

ATG-021 (GPRC5D x CD3)
Multiple Myeloma

ATG-107 (FLT3 x CD3)
Acute Myeloid Leukemia

Other Programs

ATG-031 (CD24 mAb)
Phase I Dose Escalation

ATG-042 (PRMT5-MTA Inhibitor)
IND-ready

ATG-008[4] (mTORC1/2 Inhibitor)
Phase II Dose Expansion

ATG-101 (PD-L1/4-1BB BsAb)
Phase I Dose Escalation

Cash and Bank Balances of USD125mm[5] to Advance Pipeline Development and Strategic Initiatives Over the Next 3 Years

[1] Data for ATG-022 in CLDN18.2 moderate to high expressing GC (IHC 2+ > 20%) is as of April 21st, 2025; [2] Data in CLDN18.2 low and ultra-low expressing GC (IHC 2+ ≤ 20%) is as of November 22nd, 2024; [3] Data for ATG-037 is as of April 27th, 2025; [4] Antengene only has rights for Asia Pacific for ATG-008; [5] USD125mm converted from RMB900mm at USD/RMB 7.1884

1

ATG-022 (CLDN18.2 ADC)

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ATG-022: CLDN18.2 ADC with Efficacy Across the Widest Patient Population and the Best Safety Profile Without Cumulative Toxicities, Allowing for Longer Treatment Duration

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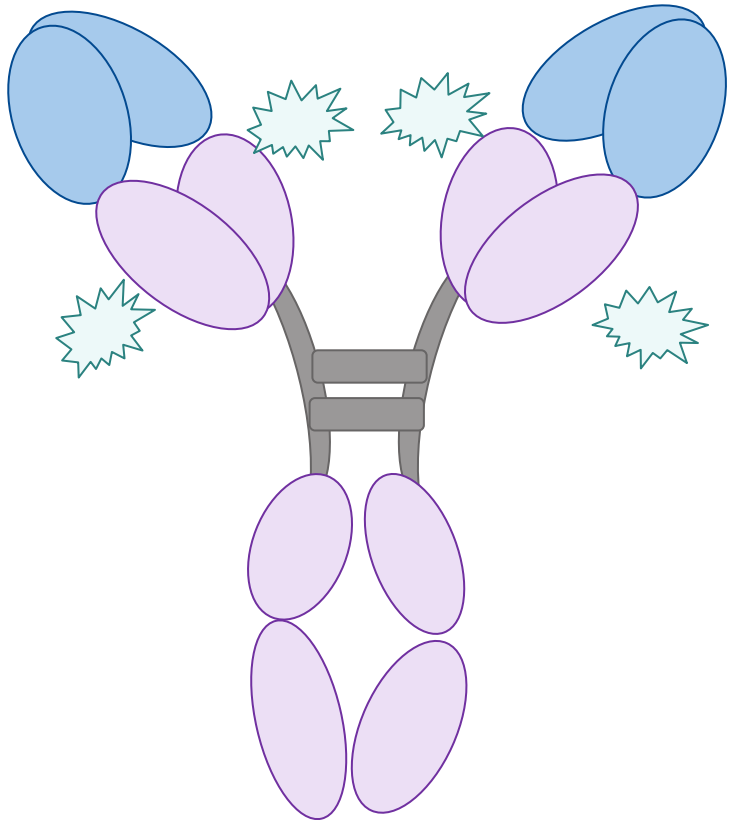
ATG-022: CLDN18.2 ADC with Differentiated Potency

High Affinity Antibody

- ✓ Enables **binding** to cancer cells with **low CLDN18.2 expression**
- ✓ Promotes **rapid internalization**, and **enhances the bystander effect**



= vc-MMAE

Cys based conjugation
Mean DAR = 4
Specific DAR4 >70%

Clinical Data Highlights

- ✓ **Efficacy across all CLDN18.2 expression levels**
- ✓ **Limited systemic toxicities**
- ✓ **Preliminary efficacy** observed in a **non-GI tumor type**

ATG-022's Differentiation by Design

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	ATG-022	Other ADCs In Development
Potential Target Population Based on Reported Data	All-comers (Including CLDN18.2 Low and Ultra-low Expression)	CLDN18.2 Moderate to High Expression
Binding Affinity of Antibody	+++	+
Speed of Internalization	+++	+
Bystander Effect	+++	+
Systemic Toxicities	+	+++
Potential Need for CDx	↓	↑↑↑
Potential to Move to Other Tumor Types Beyond GC/GEJ	↑↑↑	↓

Huge Unmet Medical Need and Market Opportunity Globally in Claudin 18.2 Positive Gastric Cancer

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Global

~1.6m

Prevalence

United States

~27k

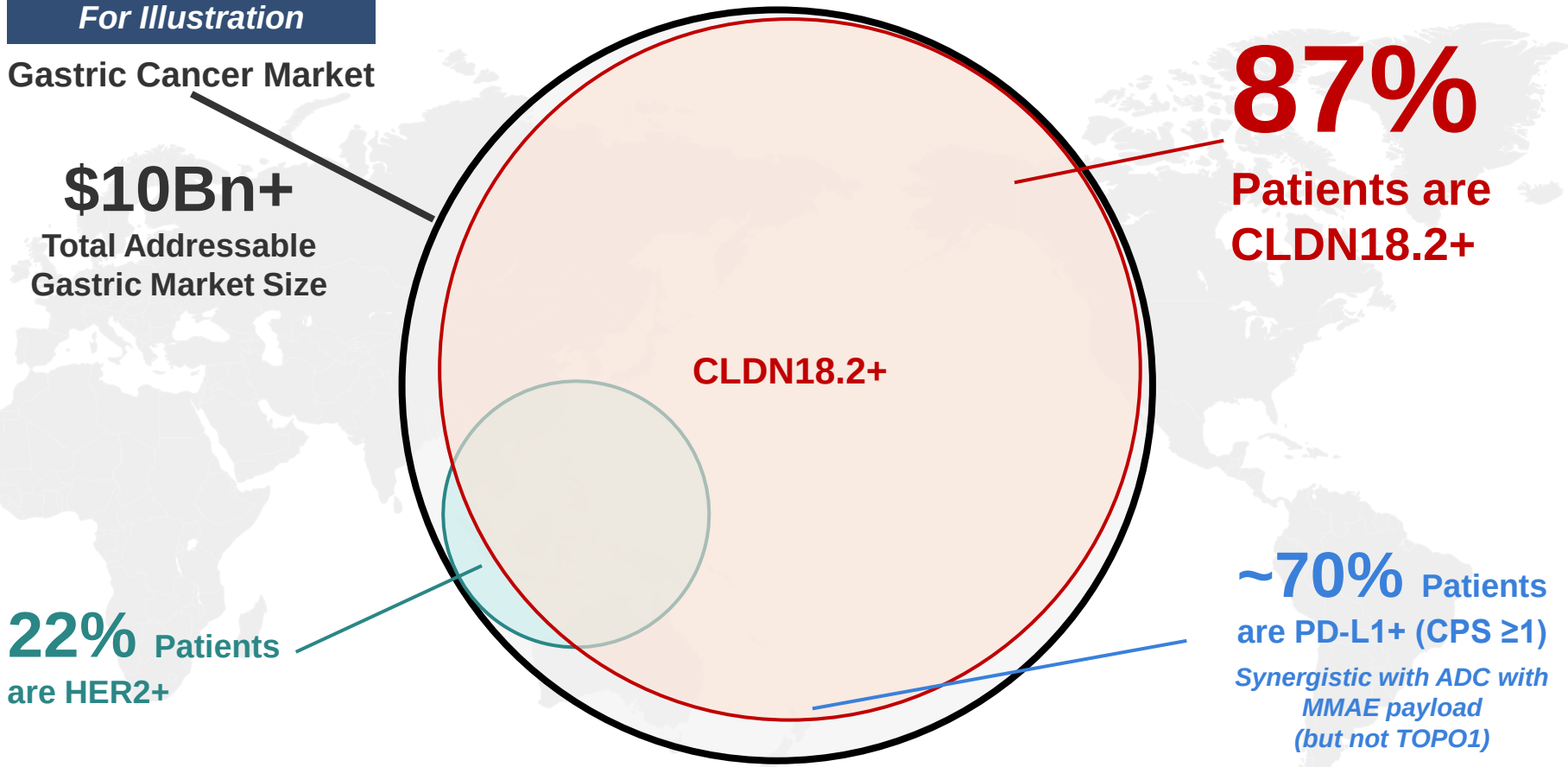
Incidence

~130k

Prevalence

The Global Gastric Cancer Market is Underpenetrated and Presents Significant Commercial Potential for Novel Therapeutics

For Illustration



*Source: GLOBOCAN; NCI SEER; Data Monitor Biomed Research; Allied Market Research; Research and Markets (Gastric Cancer Market (2024 Edition): Analysis By Indication (Gastric Cancer/Gastroesophageal Junction Cancer, Gastrointestinal Stromal Tumors), By Therapy, By Drug Class, By Region, By Country: Market Insights and Forecast (2020-2030); Cao W, Xing H, Li Y, et al. Claudin18.2 is a novel molecular biomarker for tumor-targeted immunotherapy. Biomark Res. 2022 May 31,10(1):38; Baek, J. H., Park, D. J., Kim, G. Y., Cheon, J., Kang, B. W., Cha, H. J., & Kim, J. G. (2019). Clinical Implications of Claudin18.2 Expression in Patients With Gastric Cancer. *Anticancer Research, 39*(12), 6973-6979. https://doi.org/10.21873/anticanres.13919; Türeci O, Sahin U, Schulze-Bergkamen H, Zvirbule Z, Lordick F, Koeberle D, et al. A multicentre, phase IIa study of zolbetuximab as a single agent in patients with recurrent or refractory advanced adenocarcinoma of the stomach or lower oesophagus: the MONO study. Ann Oncol. 2019;30(9):1487–1495; Van Cutsem E, Bang YJ, Feng-Yi F, et al. HER2 screening data from ToGA: targeting HER2 in gastric and gastroesophageal junction cancer. Gastric Cancer. 2015;18(3):476-484. doi:10.1007/s10120-014-0402-y; Schoemig-Markiefka B, Eschbach J, Scheel AH, et al. Optimized PD-L1 scoring of gastric cancer. Gastric Cancer. 2021;24(5):1115-1122. doi:10.1007/s10120-021-01195-4; Fuchs CS, Özgüroğlu M, Bang YJ, et al. Pembrolizumab versus paclitaxel for previously treated PD-L1-positive advanced gastric or gastroesophageal junction cancer: 2-year update of the randomized phase 3 KEYNOTE-061 trial. Gastric Cancer. 2022;25(1):197-206. doi:10.1007/s10120-021-01227-z*

ATG-022 Outperforms Competitor Molecules Efficacy in the Widest CLDN18.2+ Gastric Cancer Population, Maximizing Commercial Potential

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Over 1.4 million Claudin 18.2+ Gastric Cancer Patients Globally

Addressable Patient Population

ATG-022

Efficacy across all CLDN18.2 expression levels

- Biotech 1 — IHC Staining - 2+ ≥ 75%
- Biotech 2 — IHC Staining - 2+ ≥ 50%
- Pharma 2 — IHC Staining - 2+ ≥ 20%
- Pharma 1 — IHC Staining - 2+ ≥ 75%

High and Moderate Expression | Low and Ultra-low Expression

Claudin 18.2 Expression Level Target Patient Population – Gastric Cancer

*Source: GLOBOCAN; Cao W, Xing H, Li Y, et al. Claudin18.2 is a novel molecular biomarker for tumor-targeted immunotherapy. Biomark Res. 2022 May 31,10(1):38; Baek, J. H., Park, D. J., Kim, G. Y., Cheon, J., Kang, B. W., Cha, H. J., & Kim, J. G. (2019). Clinical Implications of Claudin18.2 Expression in Patients With Gastric Cancer. *Anticancer Research, 39*(12), 6973-6979. https://doi.org/10.21873/anticanres.13919;*

ATG-022: Advancing Global Phase II Trial in Gastric Cancer (GC) and a Broad Spectrum of Solid Tumors

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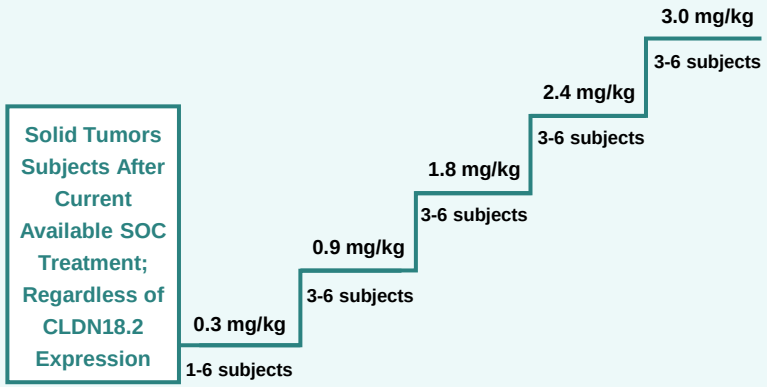
Phase II Dose Expansion Study Ongoing with Multiple Centers in Australia and the Mainland of China

Phase I: Dose Escalation

(Multiple Tumor Types without Pre-screening for Claudin 18.2 Expression Levels)



Primary Objectives: Safety, tolerability. Define MTD and RP2D
Secondary Objectives: Evaluate preliminary efficacy (RECIST 1.1), measure ADA, CLDN18.2 expression
CLDN18.2 Status: No expression requirements

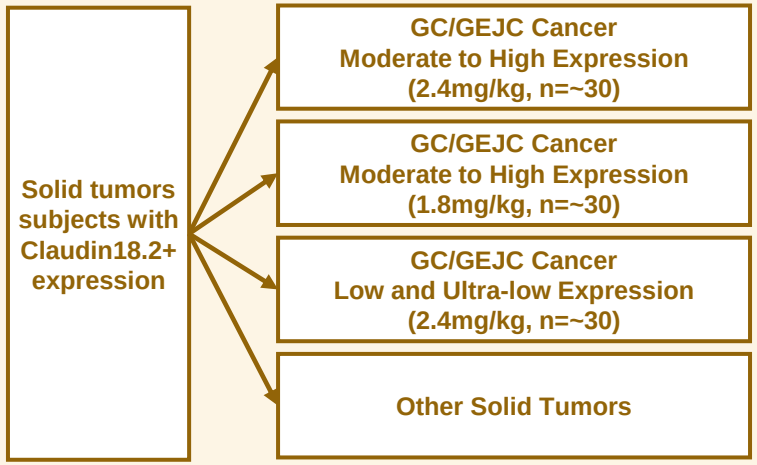
Key Observations:

- **1 CR from 2.4mg/kg dose level** (Ultra-low CLDN 18.2 expression)
- **1 PR from 1.8mg/kg dose level** (Low CLDN 18.2 expression)

Phase II: Dose Expansion

RP2D (2.4 mg/kg)

Up to 30 Subjects in Each Tumor Type / Cohort



Approximately 120 subjects, depending on the # of cohorts to be expanded
CLDN18.2+ tumors only. No prior CLDN18.2 agents

Next Stage of Development

Monotherapy – Pivotal Study (GC)

2L+ HER2-, CLDN18.2+ Gastric/GEJ Cancer for Both CLDN18.2 Moderate-to-high (IHC 2+ > 20%) and CLDN18.2 Low & Ultra-low (IHC 2+ ≤ 20%)

Combo with Anti-PD-1 – Phase Ib/II PoC Study (GC)

Frontline HER2-, CLDN18.2+, PD-L1+ (CPS ≥1) Gastric/GEJ Cancer

Monotherapy – PoC Study (Non-GC)

CLDN18.2+ Undisclosed Solid Tumor with Breakthrough Therapy Designation (BTD) Potential

Currently Enrolling Patients for the Phase II Dose Expansion Phase

ADA: anti-drug antibody; MTD = maximally tolerated dose; RP2D = recommended Phase II dose

ATG-022: Efficacy Across the Widest Patient Population in CLDN18.2+ Gastric Cancer Including From High to Ultra-low Expressors

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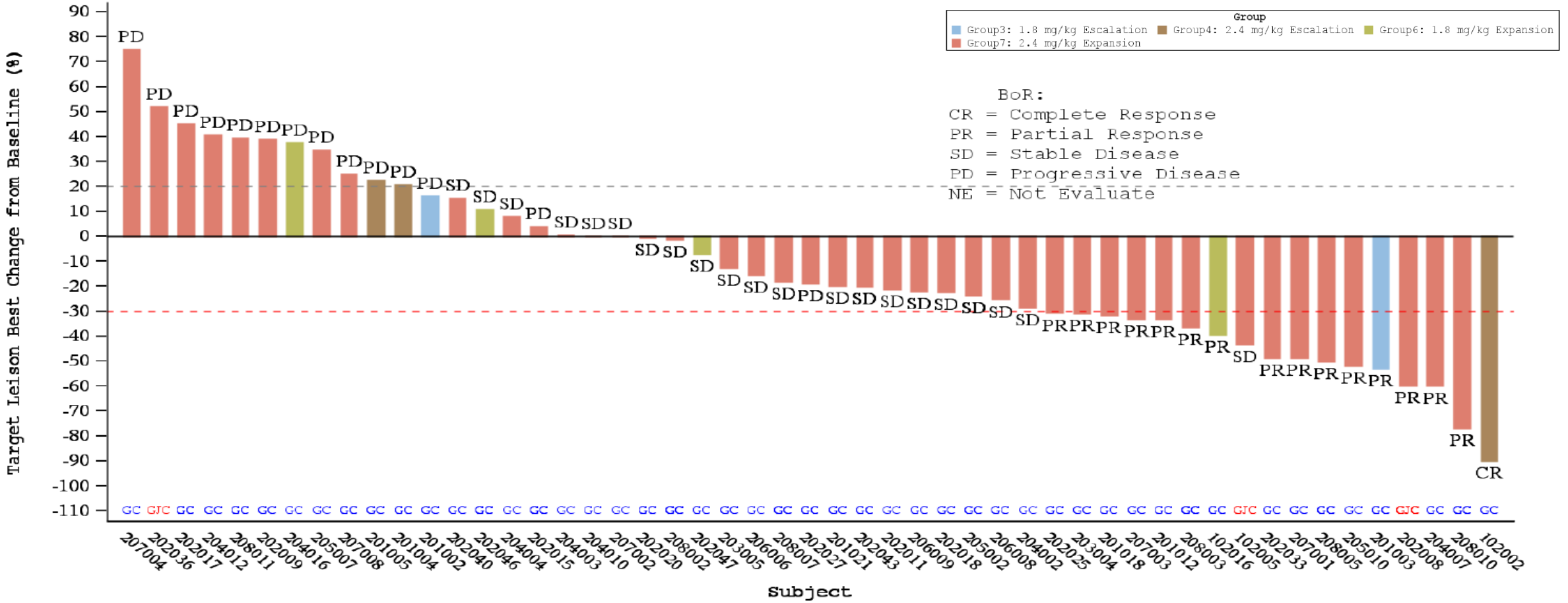
Preliminary Efficacy in CLDN18.2+ Gastric Cancer

- **IHC Staining - > 20% 2+/3+** (CLDN18.2 Moderate to High Expressors)[1]: **ORR of 40%** (12/30); **DCR of 90%** (27/30)
- **IHC Staining - ≤ 20% 2+/3+** (CLDN18.2 Low and Ultra-low Expressors)[2]: **ORR of 30%** (3/10); **DCR of 50%** (5/10)



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ATG-022: Durable Responses Demonstrated and One Patient Exceeding 21 Months

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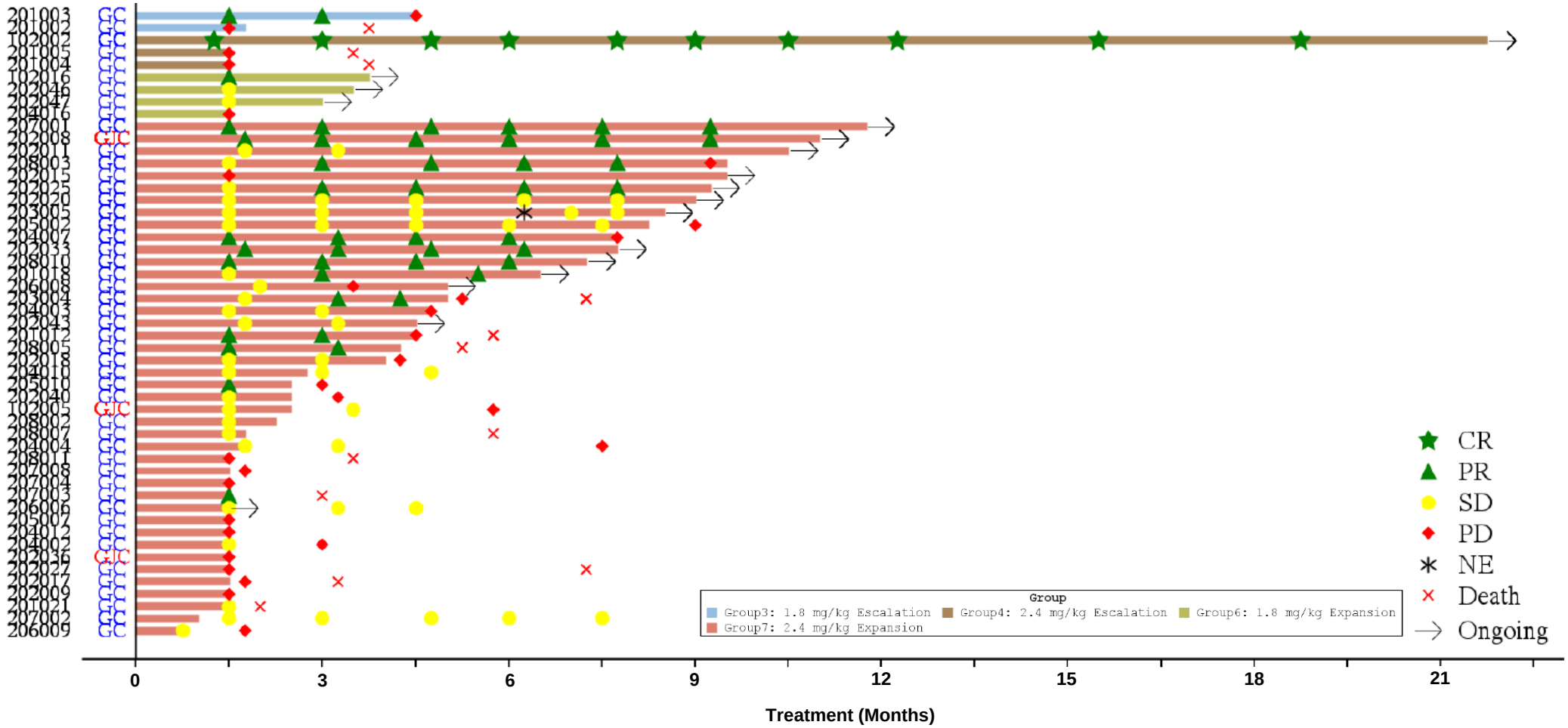
- The patient with a complete response (CR) has demonstrated **durable CR** and has been on the trial for **over 21 months**



ATG-022: Favourable Safety Profile

CLINCH (Phase I Dose Escalation & Phase II Dose Expansion) Safety Summary –TRAEs

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TEAEs							
n (%)	0.3mg/kg N=1	0.9mg/kg N=3	1.8mg/kg N=3	2.4mg/kg N=3	3.0mg/kg N=6	Expansion 2.4mg/kg N=52	Overall (2.4mg/kg) (N=55)
Subjects with at least one TRAE	0 (0)	2 (66.7)	3 (100)	3 (100)	6 (100)	50 (96.2)	53 (96.4)
Serious TRAE	0 (0)	0 (0)	0 (0)	1 (33.3)	4 (66.7)	17 (32.7)	18 (32.7)
Grade 3 or 4 TRAE	0 (0)	1 (33.3)	1 (33.3)	1 (33.3)	6 (100)	27 (51.9)	28 (50.9)
TRAE Leading to Dose Modification	0 (0)	1 (33.3)	0 (0)	1 (33.3)	5 (83.3)	24 (46.2)	25 (45.5)
TRAE Leading to Dose Reduction	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	10 (19.2)	10 (18.2)
TRAE Leading to Dose Interruption	0 (0)	1 (33.3)	0 (0)	1 (33.3)	5 (83.3)	18 (34.6)	19 (34.5)
TRAE Leading to Drug Withdrawn	0 (0)	0 (0)	1 (33.3)	0 (0)	2 (33.3)	3 (5.8)	3 (5.5)
TRAE Leading to Death	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (1.9)	1 (1.8)

Preliminary Data as of April 21, 2025

ATG-022: No Ophthalmological or Interstitial Lung Disease

CLINCH – RP2D Dose (2.4 mg/kg) TRAE By Preferred Term (PT) in ≥ 10% Patients

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TRAEs

Adverse Events	Escalation (2.4mg/kg) (N=3)		Expansion (2.4mg/kg) (N=52)		Overall (2.4mg/kg) (N=55)	
Preferred Term; n (%)	**Any Grade**	**Grade ≥ 3**	**Any Grade**	**Grade ≥ 3**	**Any Grade**	**Grade ≥ 3**
Any TRAE (n, %)	3 (100)	1 (33.3)	50 (96.2)	27 (51.9)	53 (96.4)	28 (50.9)
Neutrophil Count Decreased	2 (66.7)	1 (33.3)	28 (53.8)	7 (13.5)	30 (54.5)	8 (14.5)
Weight Decreased	0 (0)	0 (0)	28 (53.8)	2 (3.8)	28 (50.9)	2 (3.6)
WBC Count Decreased	1 (33.3)	1 (33.3)	24 (46.2)	3 (5.8)	25 (45.5)	4 (7.3)
ALT Increased	1 (33.3)	0 (0)	13 (25.0)	1 (1.9)	14 (25.5)	1 (1.8)
AST Increased	1 (33.3)	0 (0)	13 (25.0)	0 (0)	14 (25.5)	0 (0)
Blood ALP Increased	1 (33.3)	0 (0)	8 (15.4)	0 (0)	9 (16.4)	0 (0)
Blood Bilirubin Increased	1 (33.3)	0 (0)	8 (15.4)	0 (0)	9 (16.4)	0 (0)
Blood LDH Increased	1 (33.3)	0 (0)	6 (11.5)	0 (0)	7 (12.7)	0 (0)
Platelet Count Decreased	0 (0)	0 (0)	7 (13.5)	0 (0)	7 (12.7)	0 (0)
Nausea	1 (33.3)	1 (33.3)	27 (51.9)	1 (1.9)	28 (50.9)	2 (3.6)
Vomiting	1 (33.3)	0 (0)	19 (36.5)	1 (1.9)	20 (36.4)	1 (1.8)
Constipation	1 (33.3)	0 (0)	14 (26.9)	0 (0)	15 (27.3)	0 (0)
Decreased Appetite	2 (66.7)	0 (0)	24 (46.2)	2 (3.8)	26 (47.3)	2 (3.6)
Hypoalbuminemia	1 (33.3)	0 (0)	27 (51.9)	1 (1.9)	28 (50.9)	1 (1.8)
Hypocalcaemia	1 (33.3)	0 (0)	12 (23.1)	0 (0)	13 (23.6)	0 (0)
Hyponatraemia	1 (33.3)	0 (0)	8 (15.4)	1 (1.9)	9 (16.4)	1 (1.8)
Hypokalaemia	1 (33.3)	0 (0)	9 (17.3)	0 (0)	10 (18.2)	0 (0)
Anaemia	1 (33.3)	0 (0)	30 (57.7)	6 (11.5)	31 (56.4)	6 (10.9)
Malaise	0 (0)	0 (0)	12 (23.1)	0 (0)	12 (21.8)	0 (0)
Fatigue	1 (33.3)	0 (0)	8 (15.4)	1 (1.9)	9 (16.4)	1 (1.8)
Alopecia	1 (33.3)	0 (0)	9 (17.3)	0 (0)	10 (18.2)	0 (0)

- **No ophthalmological or interstitial lung disease (ILD) have been observed**

Preliminary Data as of April 21, 2025

2

ATG-037 (CD73 Inhibitor)

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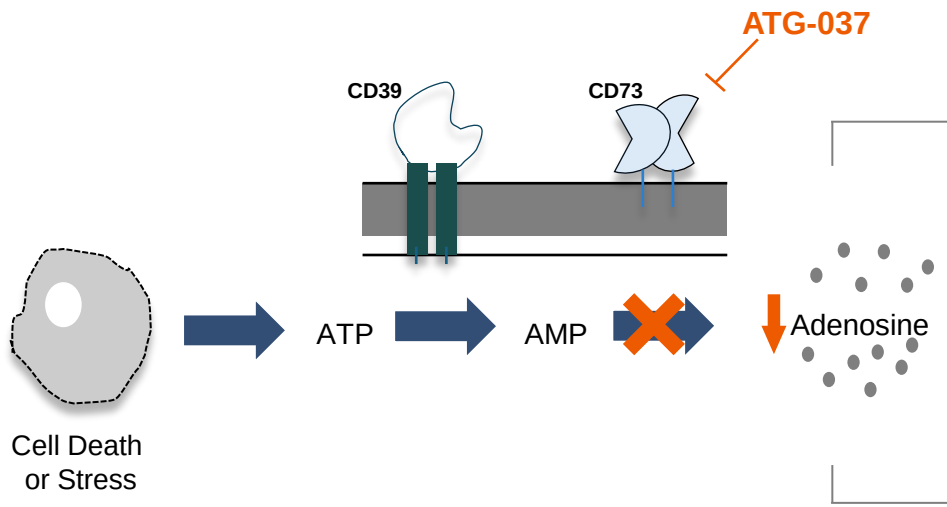
ATG-037: Potentially Best-in-Class CD73 Oral Small Molecule Inhibitor

CD73

- Cell surface receptor
- Overexpression on tumor cells interrupts adenosine processing, enabling an immunosuppressive TME
- Important in a range of solid tumor cancers, e.g., melanoma and non-small cell lung cancer

ATG-037 Reverses Adenosine Mediated Immunosuppression

- **Potent and selective, oral small molecule** inhibitor completely blocks CD73 activity
- **Activity**: Overcomes the hook effect with higher tissue penetrance v. anti-CD73 antibodies
- **Specificity**: No inhibition of related targets (including CD39)
- **Preclinical Efficacy**: Potent tumor growth inhibition as mono or combo therapy



Inhibits the Cytotoxic and Effector Function of T cell and NK cells

Induces and Enhances the function of Immunosuppressive Cell Types: Tregs, MDSCs, TAMs and Tolerogenic DCs

ATP = adenosine triphosphate, AMP = adenosine monophosphate, NK cells = Natural Killer cells, Tregs = Regulatory T-cells, MDSC = Myeloid-derived suppressor cells , TAMs = Tumor Associated Macrophages, DC = Dendritic cells, TME = tumor microenvironment

Significant Market Potential in Solid Tumors for Immuno-oncology

Market Size of Immuno-oncology (IO) is estimated to be $140+ billion in 2028, Including IO-Resistant Tumors[1]

91%

of all cancer cases are solid tumors[1]

1.8 Million

New cases of solid tumors in the US each year[1]

Expand into Other Indications

	U.S. Deaths[1]	Global Deaths[2]
Melanoma	8,000	59,000
Lung & Bronchus	125,000	1,800,000

Source:
1. GlobalData
2. National Cancer Institute Surveillance, Epidemiology and End Results (SEER) Program. 2024 Estimates. https://seer.cancer.gov (accessed May 2024)
3. World Health Organization International Agency for Research on Cancer (IARC). GLOBOCAN 2022

ATG-037 Can Address the Huge Unmet Medical Need of Melanoma Patients who Progress on Anti-PD-1 Therapy

Annual US & Ex-US Addressable Patient Opportunity in Previously Treated Advanced Melanoma[3]

~30,000

Advanced Melanoma Overall Patient Opportunity[3]

>70,000

Earlier Treatment Setting

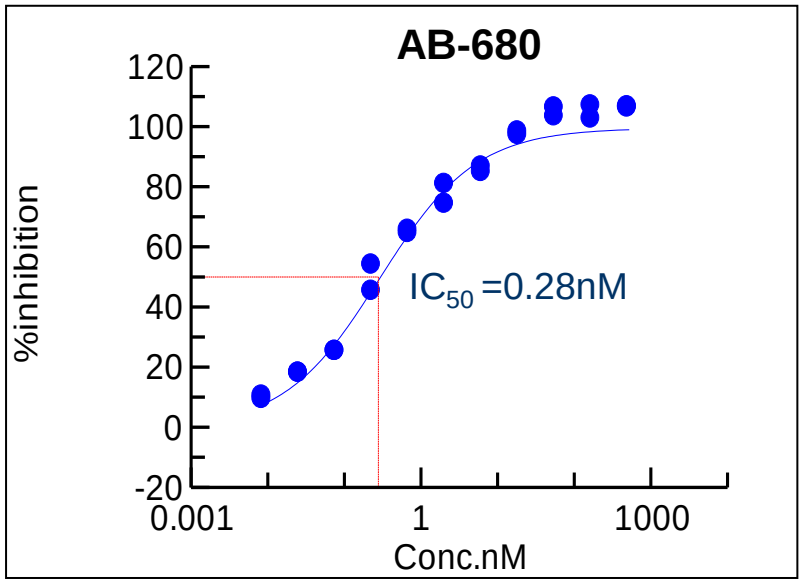
Geographic Footprint

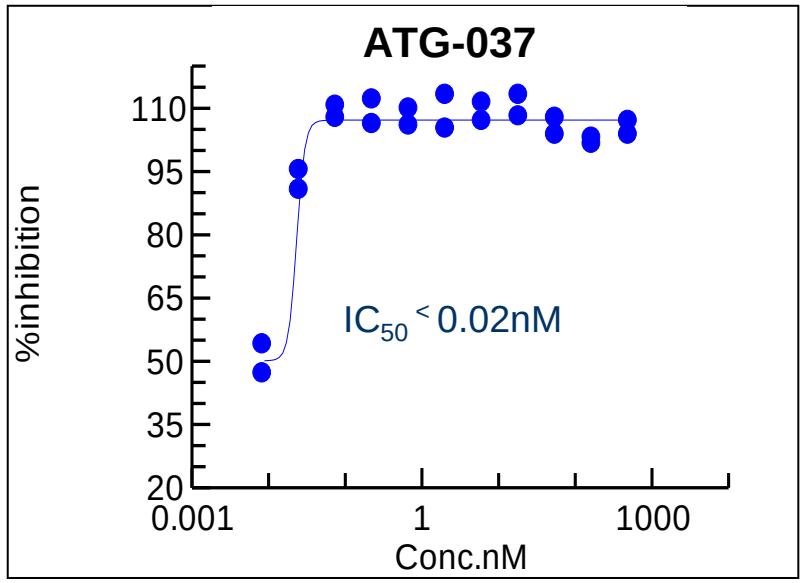
	Annual Deaths[1,2]	Frontline Addressable Patients[3]
U.S.	8K	14K
Ex-U.S. Anticipated Markets	22K	27K
Total	30K	41K

Source:

1. National Cancer Institute Surveillance, Epidemiology and End Results (SEER) Program. 2024 Estimates. https://seer.cancer.gov (accessed May 2024)
2. World Health Organization International Agency for Research on Cancer (IARC). GLOBOCAN 2022
3. Data on file as of September 30, 2024. Includes more than 20,000 patients initial target markets plus additional potential markets.

ATG-037 Shows More Potent CD73 Inhibition in Full Human Plasma, Compared with AB680 (Quemliclustat)





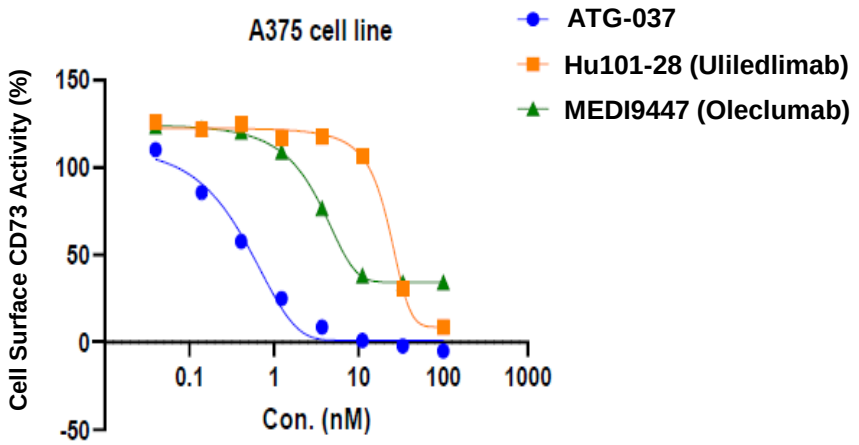
Head-to-head comparison suggests **higher recombinant CD73 inhibition potency** of ATG-037 compared with AB680

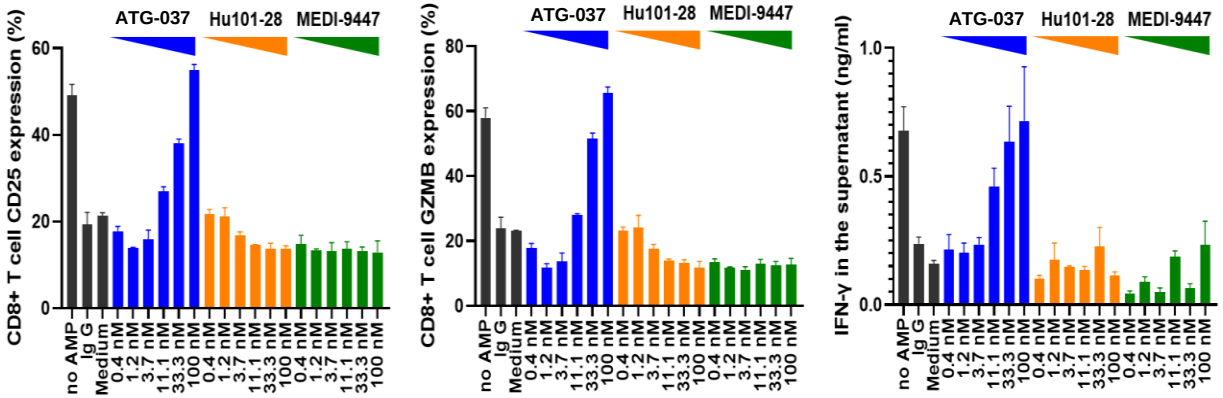
Assay	*AB680 IC_{50} (nM)	ATG-037 IC_{50} (nM)
Human Plasma CD73	19.9	0.38
Mouse Plasma CD73	790	1.0

ATG-037 shows a **50-fold higher activity in human plasma** compared with AB680

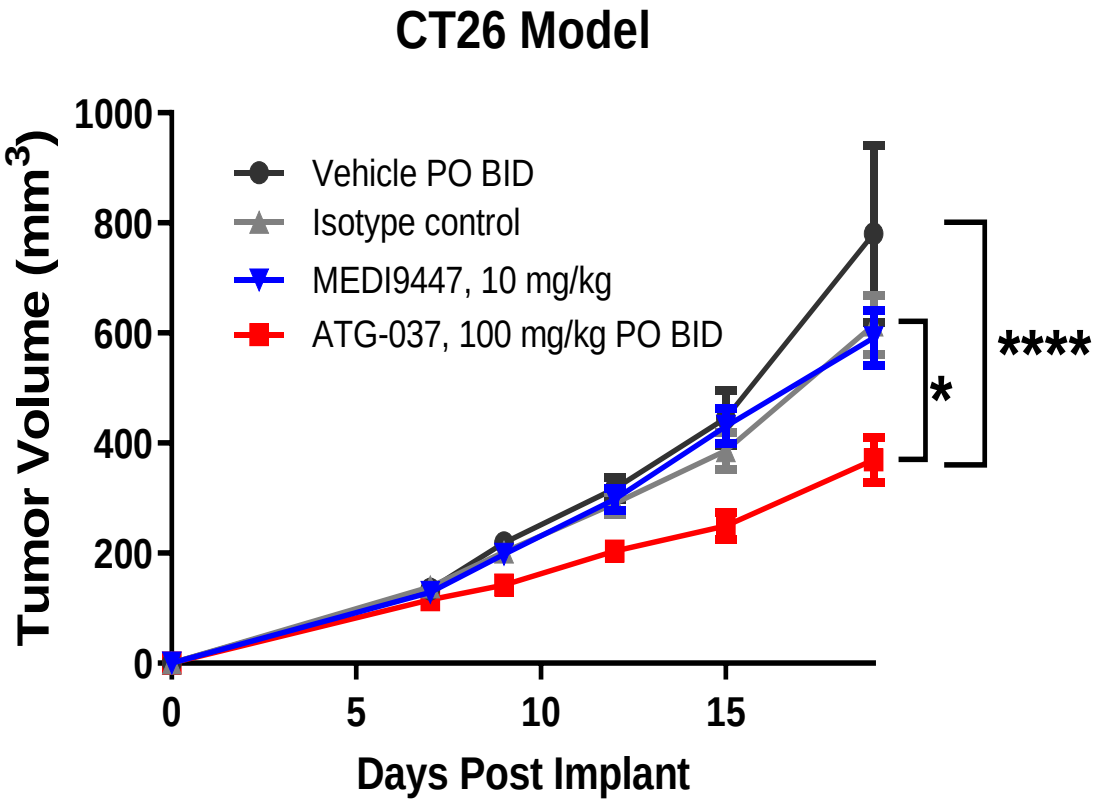
ATG-037 Showed Strong CD73 Inhibition and Potent Preclinical Mono and Combination Activity

Complete CD73 inhibition at 0.4nM with Superior Activity in Reversing T Cell Inhibition





In Vivo Efficacy with in Murine CT26 Colorectal Cancer Model

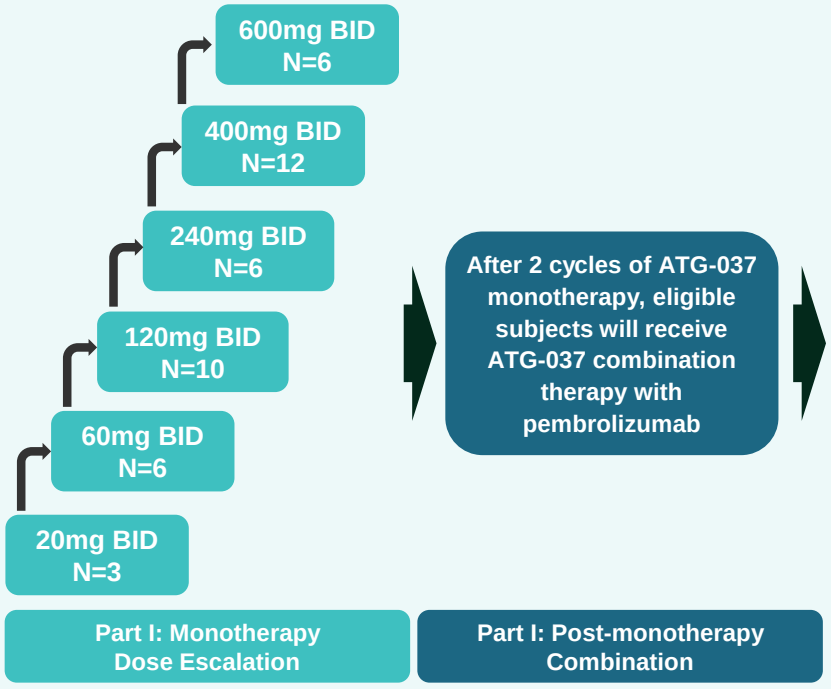


ATG-037 "STAMINA" Clinical Trial Design

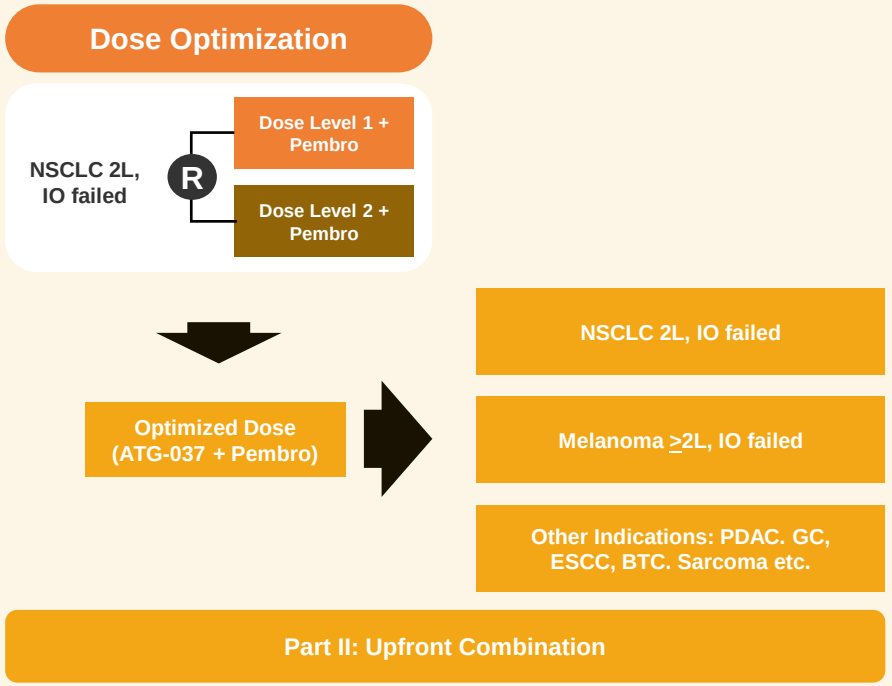
Population: Patients with locally advanced or metastatic solid tumors with acquired checkpoint inhibitor resistance (The most common tumor types enrolled include NSCLC, melanoma, SCLC, renal cell carcinoma, ovarian carcinoma); Patients received a median of 2 prior lines of treatment (ranges 0-7)

Phase I/II, Multi-center, Open Label, Dose-finding Study Ongoing in Australia and China (NCT05205109)

Phase I: Dose Escalation



Phase II: Dose Expansion



Objectives of the Study

Primary Objectives:
Safety, tolerability monotherapy and pembrolizumab combination therapy. RP2D definition

Secondary Objectives:
Evaluate preliminary efficacy, characterize pharmacology (PK/PDx profile)

ATG-037 In Combination with Pembrolizumab Demonstrated Encouraging Efficacy Signals in CPI-resistant Melanoma and NSCLC – Waterfall Plot

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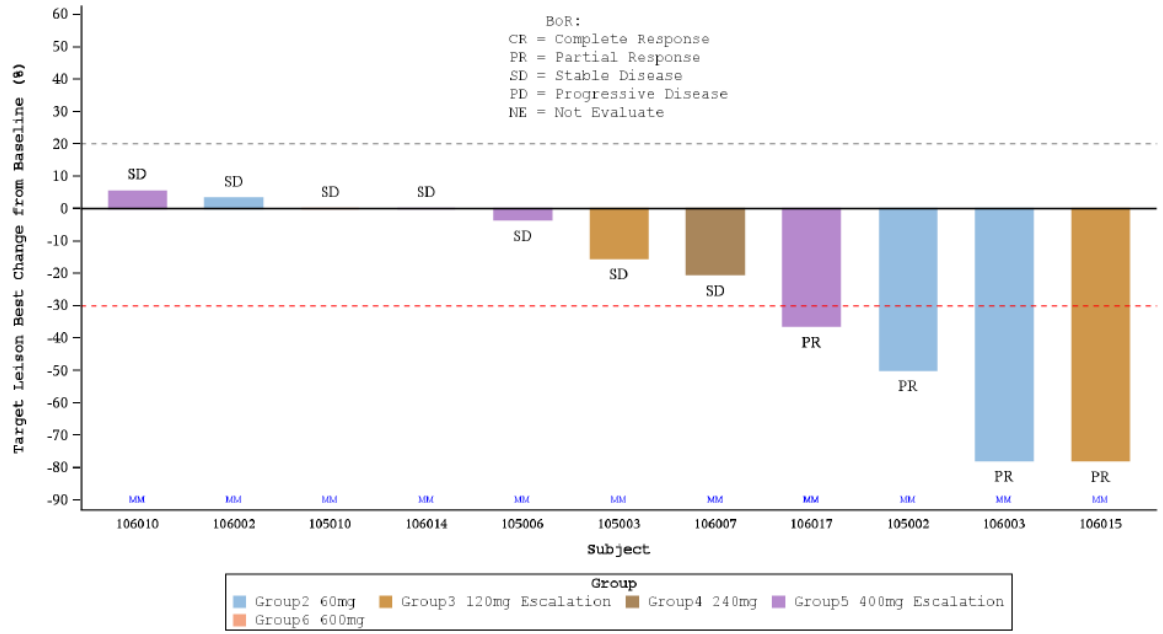
Preliminary Data (as of April 27, 2025)

- 28 patients who received combination therapy and were efficacy evaluable per protocol, 6 (21.4%) had a best response of partial response (PR), 16 (57.1%) were SD, and 6 (21.4%) were PD
- A total of **9 NSCLC patients** and **11 melanoma patients** received combination therapy and were efficacy evaluable
  - **PRs occurred in 4 of the melanoma patients (ORR 36.4%)** and **2 of the NSCLC patients (ORR 22.2%)** comparing with the screening baseline
  - The **ORR is 30.0% (6/20)** and **DCR is 85.0% (17/20)** in the efficacy evaluable NSCLC and melanoma populations comparing with the screening baseline

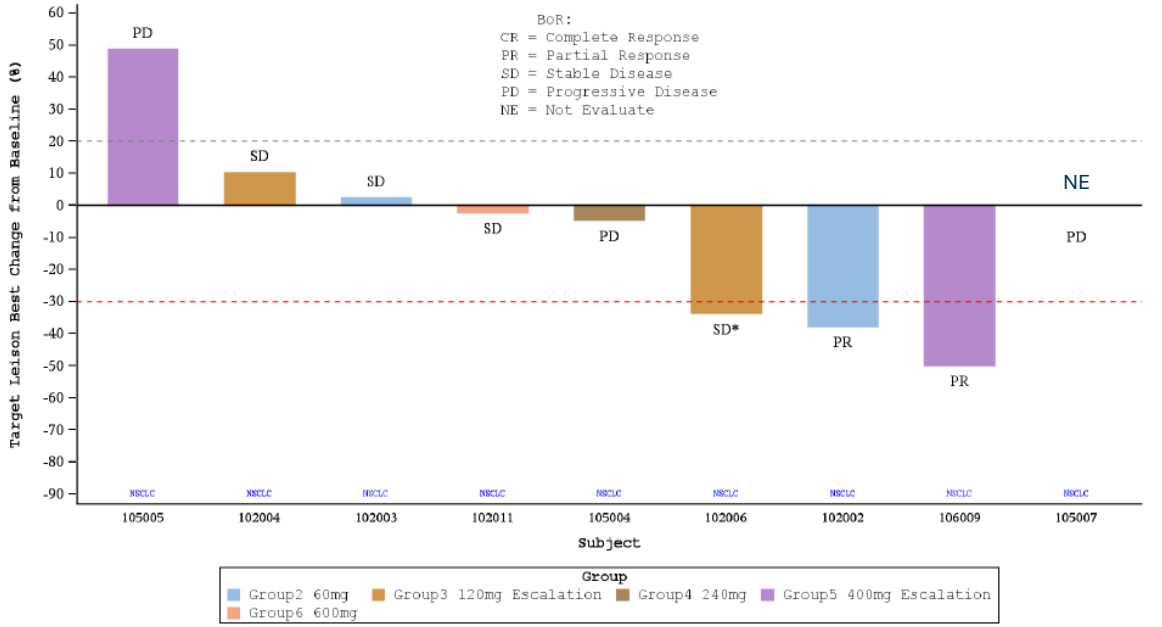
CPI Resistant Melanoma

Tumor Evaluation (Target Lesion Change from Baseline)



CPI Resistant Non-small Cell Lung Cancer

Tumor Evaluation (Target Lesion Change from Baseline)



*The target lesion of this subject reached PR with new lesion occurred. The prior best response was SD

Poster Presentation: 2025 ASCO ANNUAL MEETING

4

AnTenGager™ T Cell Engager (TCE) Platform

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AnTenGager™, a Novel Second Generation "2+1" TCE Platform with Steric Hindrance-masking Technology Enabling the Creation of TCEs with Enhanced Therapeutic Effect and Safety

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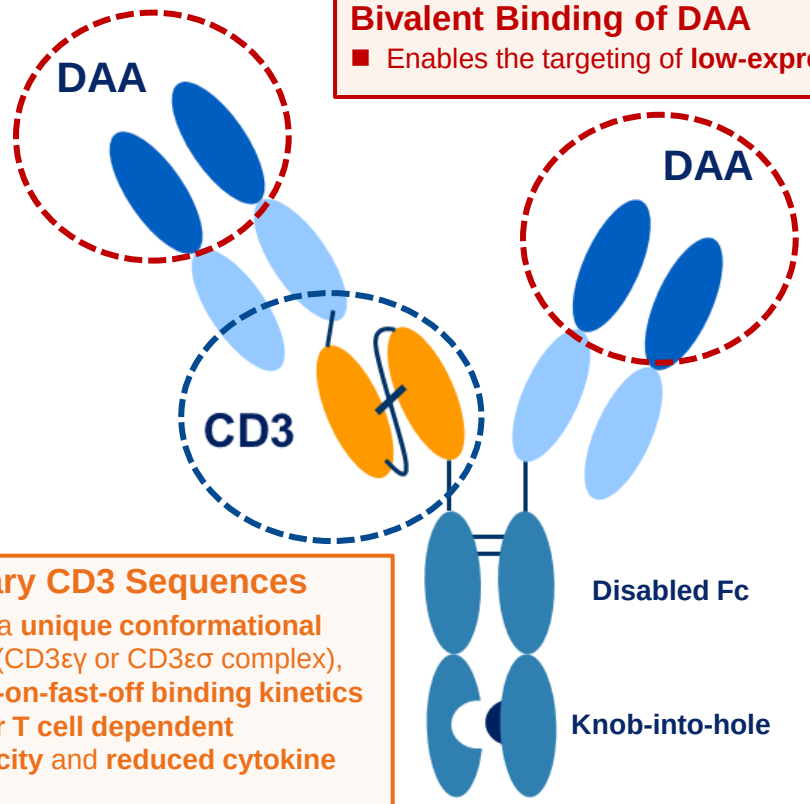
Features of AnTenGager™ TCEs



Bivalent Binding of DAA

- Enables the targeting of **low-expressing target**

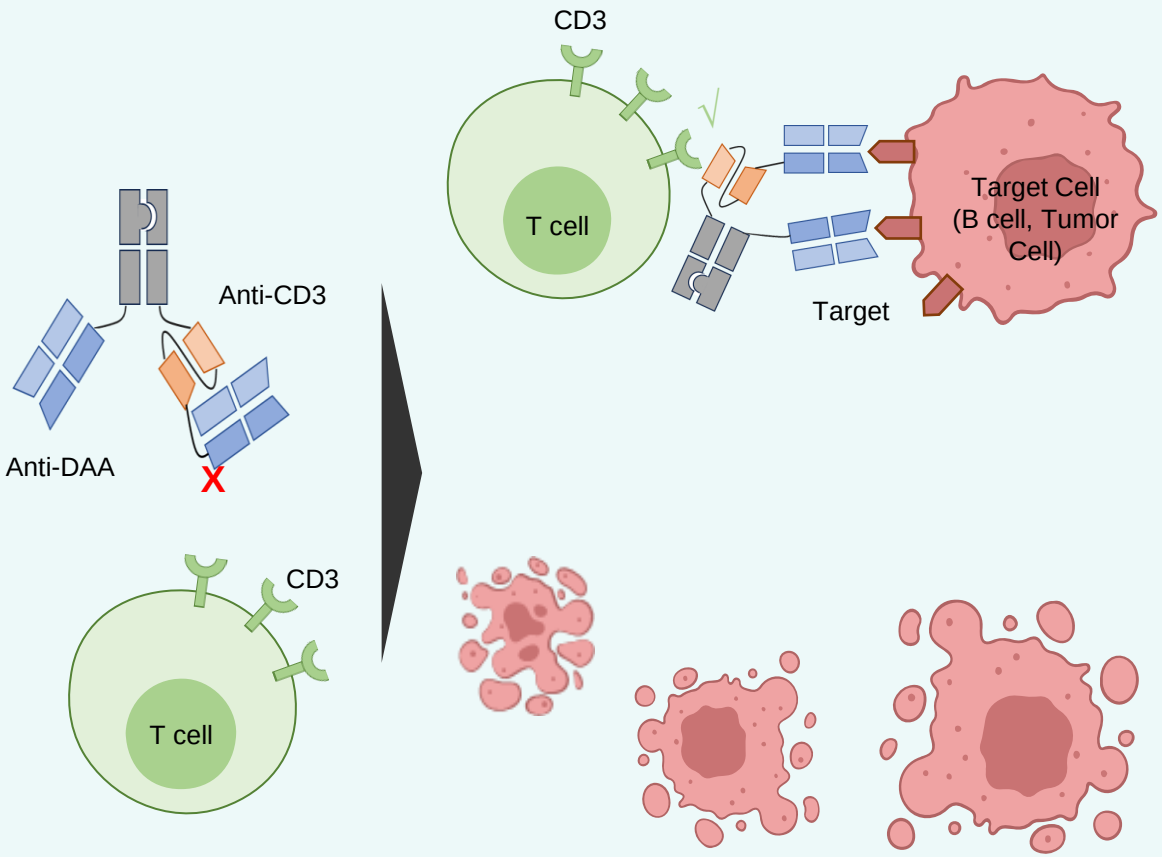
Proprietary CD3 Sequences

- Binds to a **unique conformational epitope** (CD3εγ or CD3εσ complex), with **fast-on-fast-off binding kinetics**
- **Stronger T cell dependent cytotoxicity** and **reduced cytokine release**
- **Patented**

Steric Hindrance Masking Technology

- Reduced risk of **hook effect** and **cytokine release syndrome (CRS)**

Target-Dependent CD3 Binding and Cytotoxicity



AnTenGager™ TCE 2.0 Overcoming CRS Barriers to Unlock Broader and Safer Therapeutic Applications

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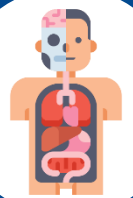


Minimizing Off-target Cytokine Release

Steric Hindrance Masking Technology

- **Minimizes off-target cytokine release** through target-dependent CD3 activation, enabling a safer therapeutic window
- Compared with protease-dependent shielding TCEs that require the tumor microenvironment, e.g. Janux platform, **AnTenGager™ is independent of the TME and can be used for broader indications beyond solid tumors**.

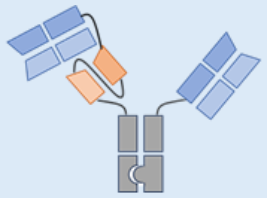
Minimizing On-target Cytokine Release

Proprietary Anti-CD3 Sequences

- **Minimizes on-target cytokine release** by binding to a **unique conformational epitope** with **fast-on-fast-off** binding kinetics while maintaining potent T cell activation

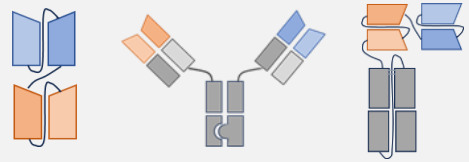
Engineered for Broader Use with Superior Safety and Efficacy

AnTenGager™ – TCE 2.0 to Transform the Treatment Landscape in Solid Tumors, Hematological Malignancies and Autoimmune Diseases



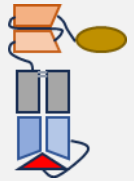
AnTenGager™ – TCE 2.0

V.S.



First Generation "1+1" TCE with Exposed CD3

or



First Generation Protease or pH Dependent Masking Technology

"2+1" Bivalent DAA Binding

Better Efficacy in Low-expressing Targets

Steric Hindrance Masking Technology

Better Safety with Lower Risk of CRS

Broad Applicability in Different Indications

Solid Tumors, Hematological Malignancies, Autoimmune Diseases

Patented Platform Technology

Proprietary Anti-CD3 Sequences

Longer Half Life

Good PK Profile with a Half Life of 100-300 Hours in Mice

AnTenGager™ Platform Pipeline Overview

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Proprietary Anti-CD3 Library

- Affinity: 10^{-6}M to 10^{-9}M
- Fast-on-fast-off binding kinetics
- Epitope: CD3εγ or CD3εσ complex

Anti-DAA Tool Box

- **Autoimmune Diseases:** CD19, CD20
- **Hematological Malignancies:** GPRC5D, LILRB4, FLT3…
- **Solid Tumor:** CLDN18.2, CDH6, GD2, LY6G6D, B7H7, B7H3, ALPPL2, undisclosed TAA…

	Assets	Target	Therapeutic Area	mAb Discovery	*In vitro* efficacy	*In vivo* efficacy	Developability	CMC/Tox	IND
Autoimmune Diseases	ATG-201	CD19 x CD3	B Cell Related Autoimmune Diseases						Expected in 2025 H2
	Undisclosed Trispecific Program	Undisclosed	Autoimmune Diseases						
Solid Tumors	ATG-106	CDH6 x CD3	Ovarian Cancer & Kidney Cancer						
	ATG-110	LY6G6D x CD3	Microsatellite Stable (MSS) Colorectal Cancer						
	ATG-112	ALPPL2 x CD3	Gynecological Tumors and Lung Cancer						
Hematological Malignancies	ATG-102	LILRB4 x CD3	Acute Myeloid Leukemia (AML) & Chronic Myelomonocytic Leukemia (CMML)						
	ATG-021	GPRC5D x CD3	Multiple Myeloma						
	ATG-107	FLT3 x CD3	Acute Myeloid Leukemia (AML)						

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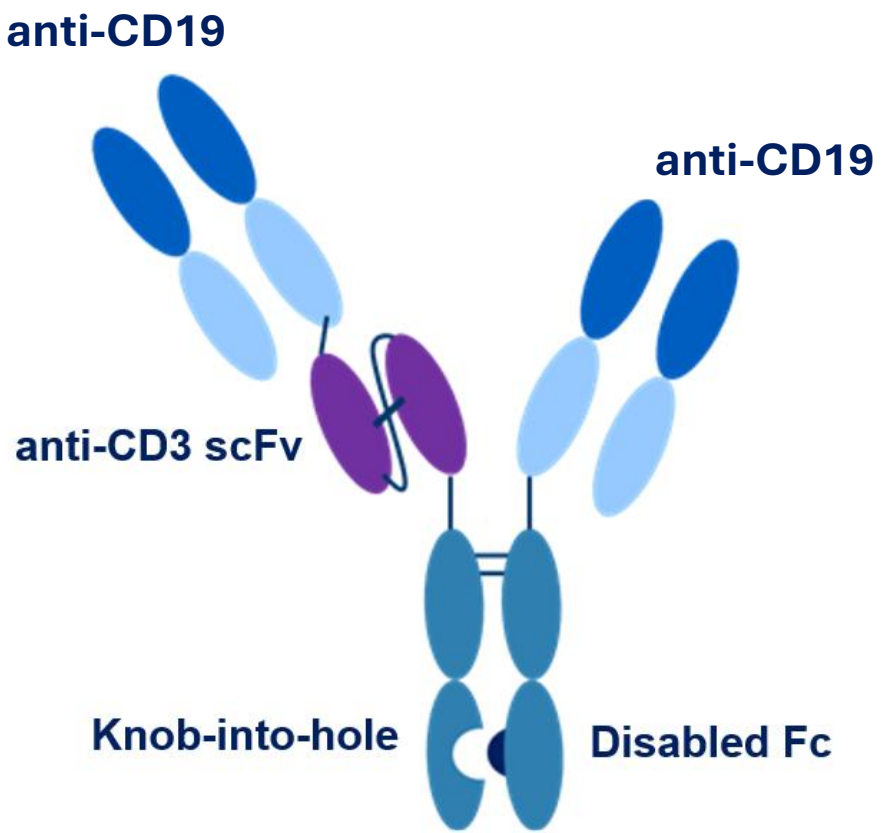
ATG-201

CD19 x CD3 T Cell Engager
for B Cell Related Autoimmune Diseases

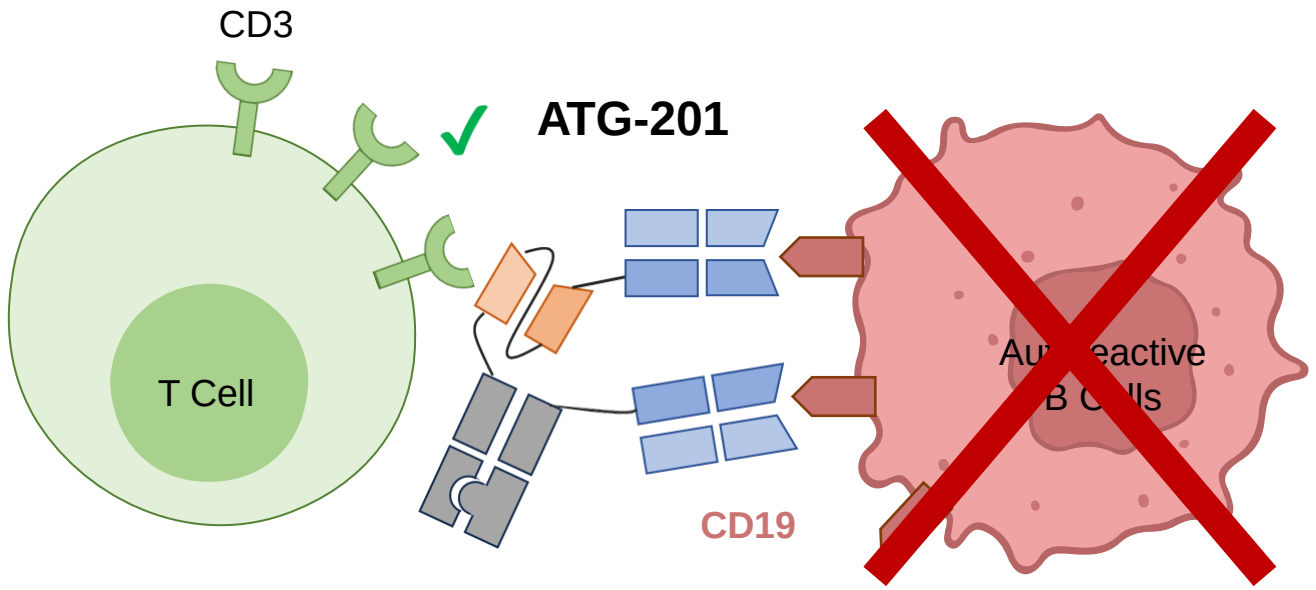
ATG-201: CD19 x CD3 TCE 2.0 With Ability to Deeply Deplete B Cells for the Treatment of Autoimmune Diseases

ANTENGENE

ATG-201 is a CD19 x CD3 TCE with Target Dependent T Cell Activation



B Cell Depletion Therapy with ATG-201 to Treat Autoimmune Diseases



B Cell Depletion Leads to the Remission of Autoimmune Diseases

IND-enabling Study and CMC Work is Ongoing with IND Targeting 2025 H2

TCE 2.0: Deeper B Cell Depletion and Better Safety for Treatment of Autoimmune Diseases

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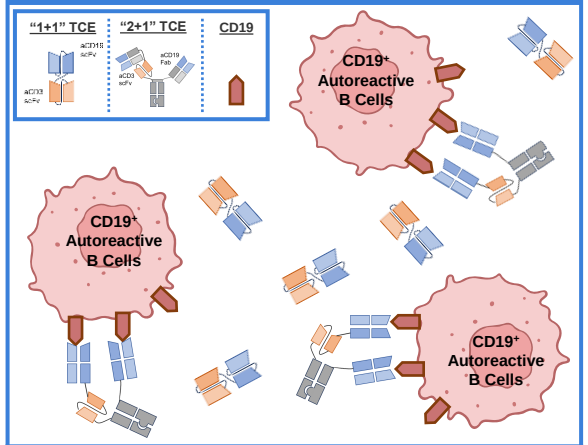
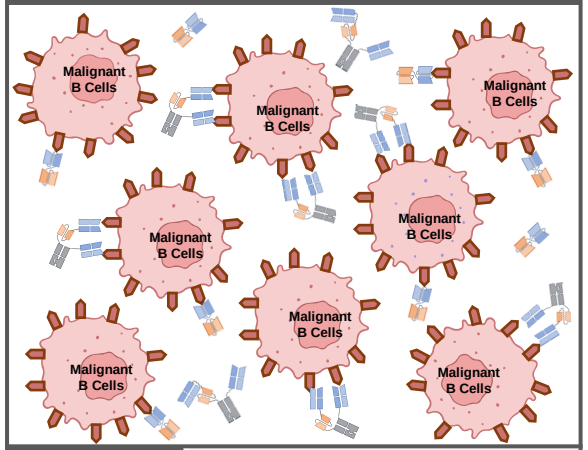
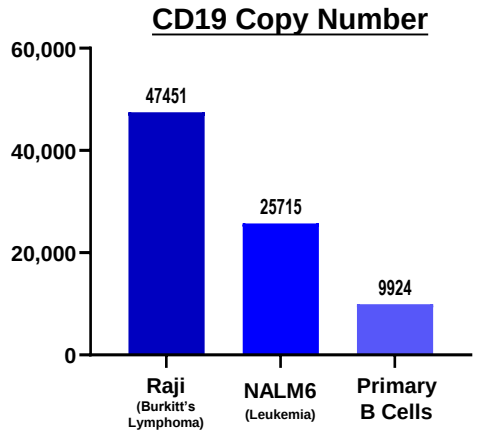
Efficacy Data from First Generation "1+1" TCEs In B Cell Malignancies May Not Translate To Comparable Efficacy In Autoimmune Diseases

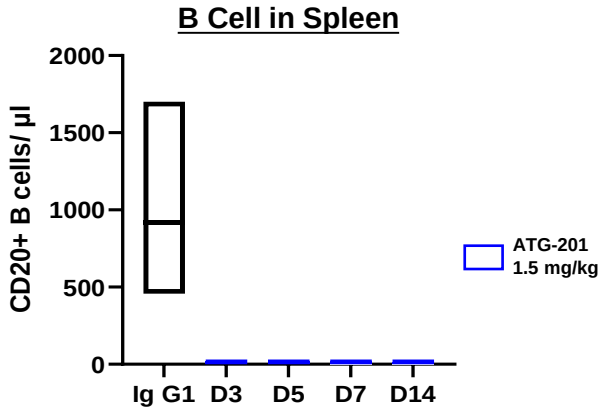
Distinct Disease Biology of Autoimmune Diseases vs. B Cell Malignancies Demands Different Drug Design Approaches

	Autoimmune Diseases	B Cell Malignancies
		
Role of TCE in Therapy	Eliminating **dysregulated autoreactive CD19+ B cells producing autoantibodies** that drive autoimmune diseases	Eliminating **malignant B cells that infiltrate bone marrow** and disrupts normal hematopoiesis
Required TCE Affinity Level	**Higher-affinity "2+1" TCEs** are needed to effectively eliminate CD19+ B cells, which exist in **much lower abundance compared to B cell malignancies**	**Lower-affinity TCEs (e.g. "1+1" TCEs) are sufficient** to effectively and rapidly deplete malignant B cells due to their high numbers

Bivalent Binding of Second-Generation "2+1" TCEs Enables Targeting of CD19-Low-Expressing B Cells in Autoimmune Diseases



CD19 copy number expressed on the surface of **autoimmune disease-related B cells** is **significantly lower** that of malignant B cells



Bivalent CD19 binding of **ATG-201** enables **deep and durable B cell depletion** for the treatment of autoimmune diseases

ATG-201 Shows Comparable or Enhanced Naïve B Cell Depletion and Reduced Cytokine Release vs. First Generation TCEs *Ex Vivo*

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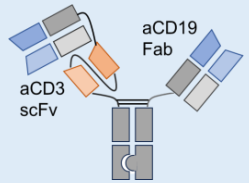
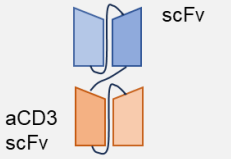
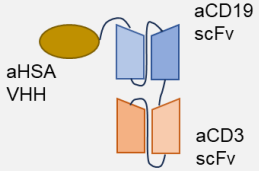
ATG-201



Benchmark 1

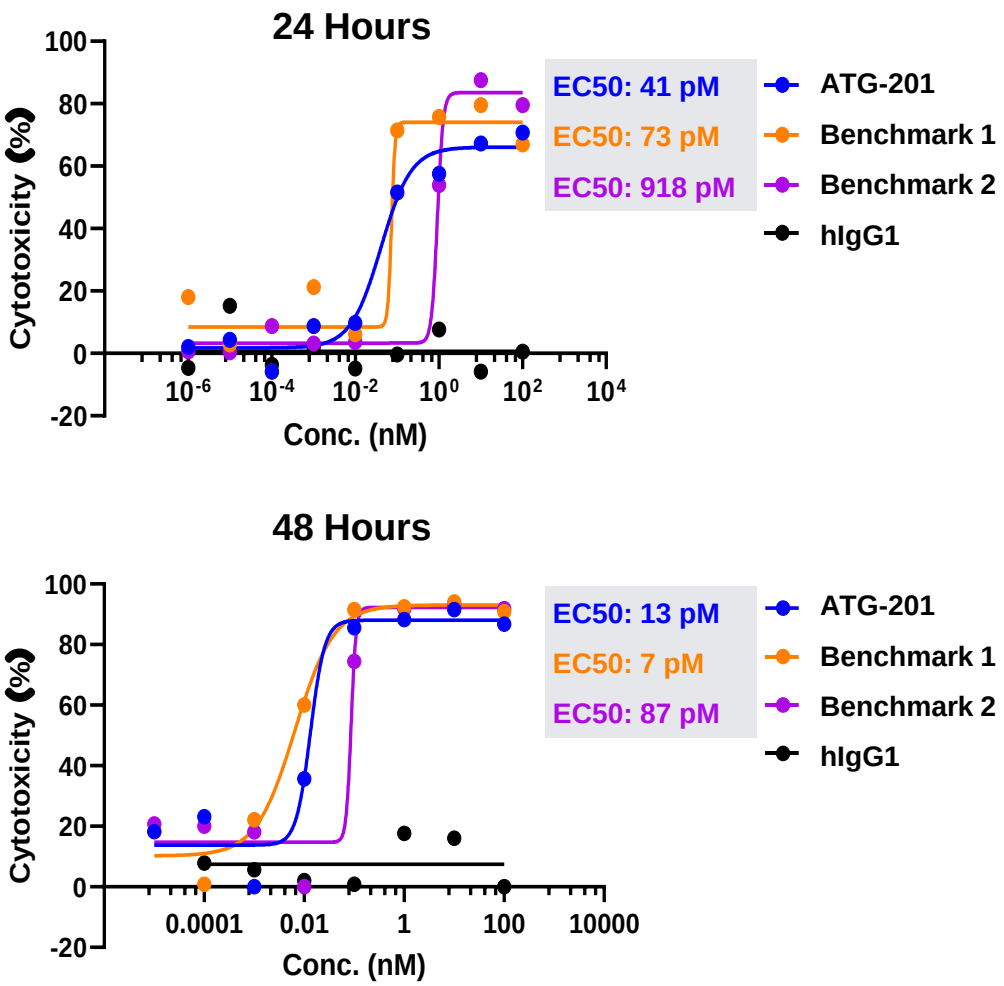


Benchmark 2

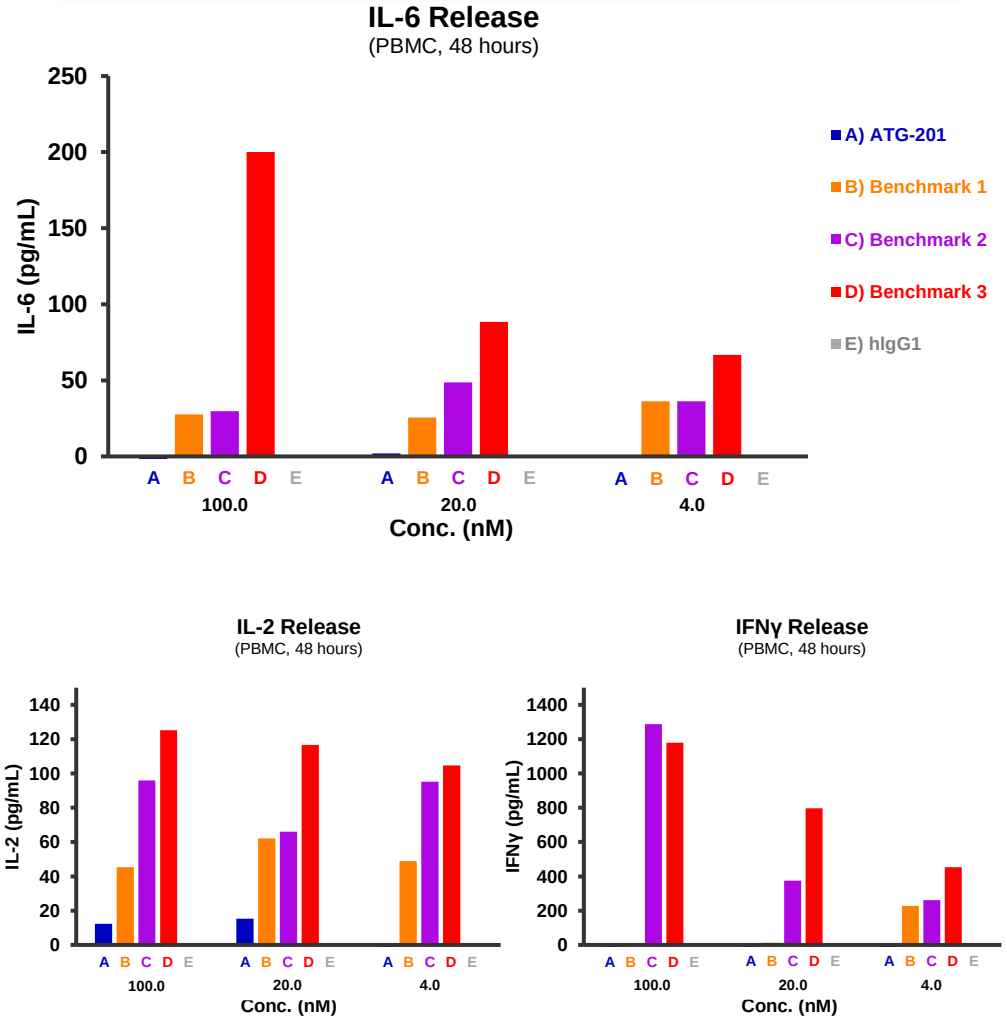


Benchmark 3

Comparable or Enhanced Naïve B Cell Depletion vs. Benchmarks

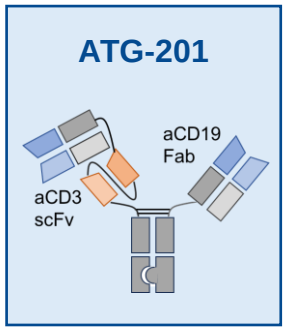


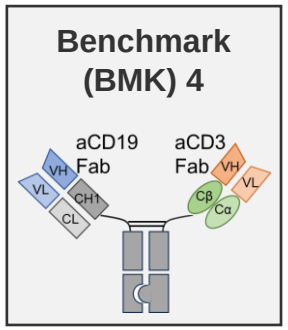
Reduced Cytokine Release vs. Benchmarks

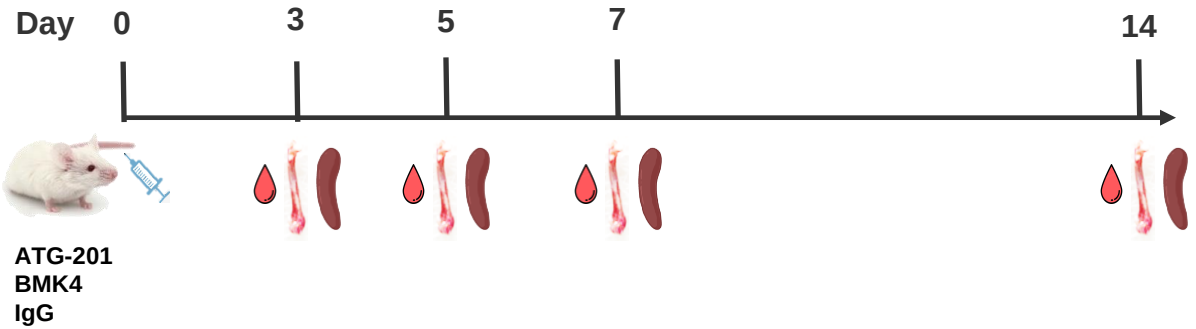


ATG-201 Demonstrated Deeper and More Durable *In Vivo* B Cell Depletion Compared to Benchmark in CD34+ Cell Humanized Mice

- **ATG-201:** A single dose **completely and deeply depleted B cells** in CD34 humanized mice, with **no detectable B cells** in blood, bone marrow or spleen **14 days post-treatment**
- **Benchmark 4: Partially depleted B cells** in bone marrow; B cells in blood and spleen were eliminated by Day 3 but began recovering by Day 5
- **Cytokine Release: ATG-201 induced significantly lower IL-6 and TNF-α release** compared to Benchmark 4



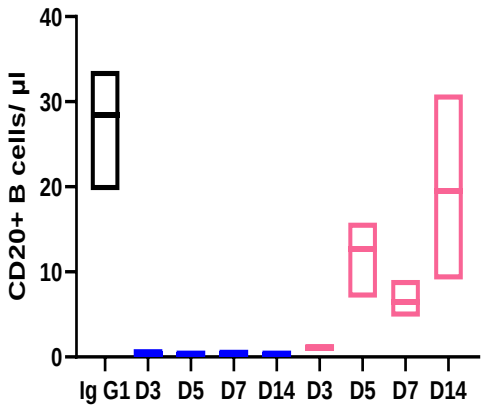


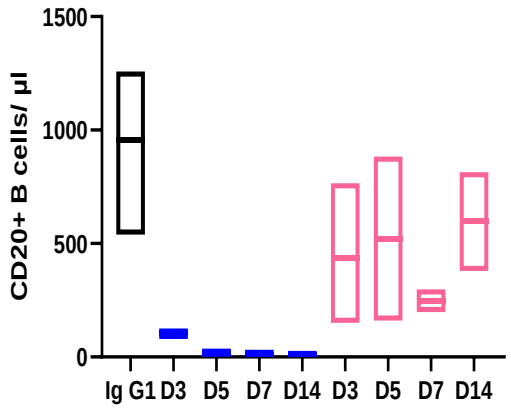


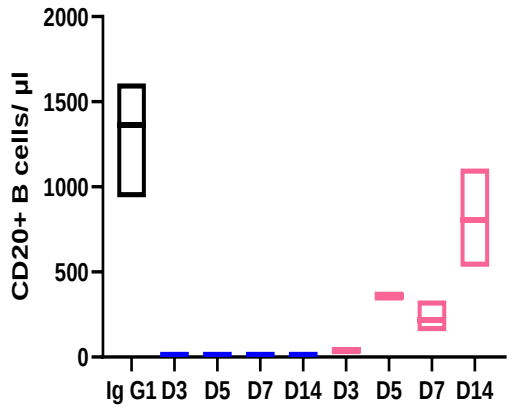
ATG-201
BMK4

Blood | **Bone Marrow** | **Spleen**

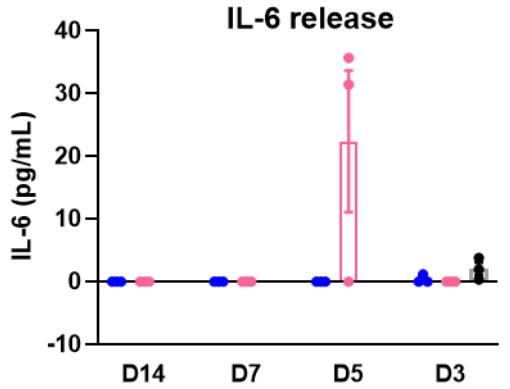
B Cells

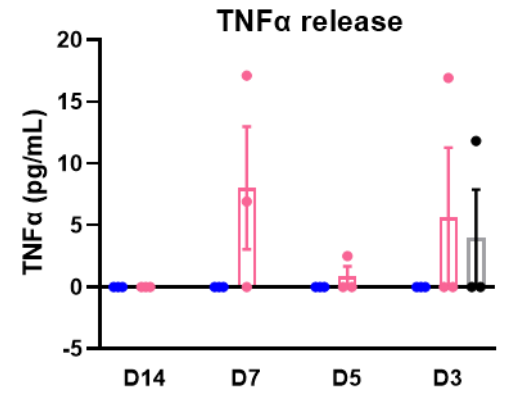






Proinflammatory Cytokine





6

Well Positioned for Long Term Growth

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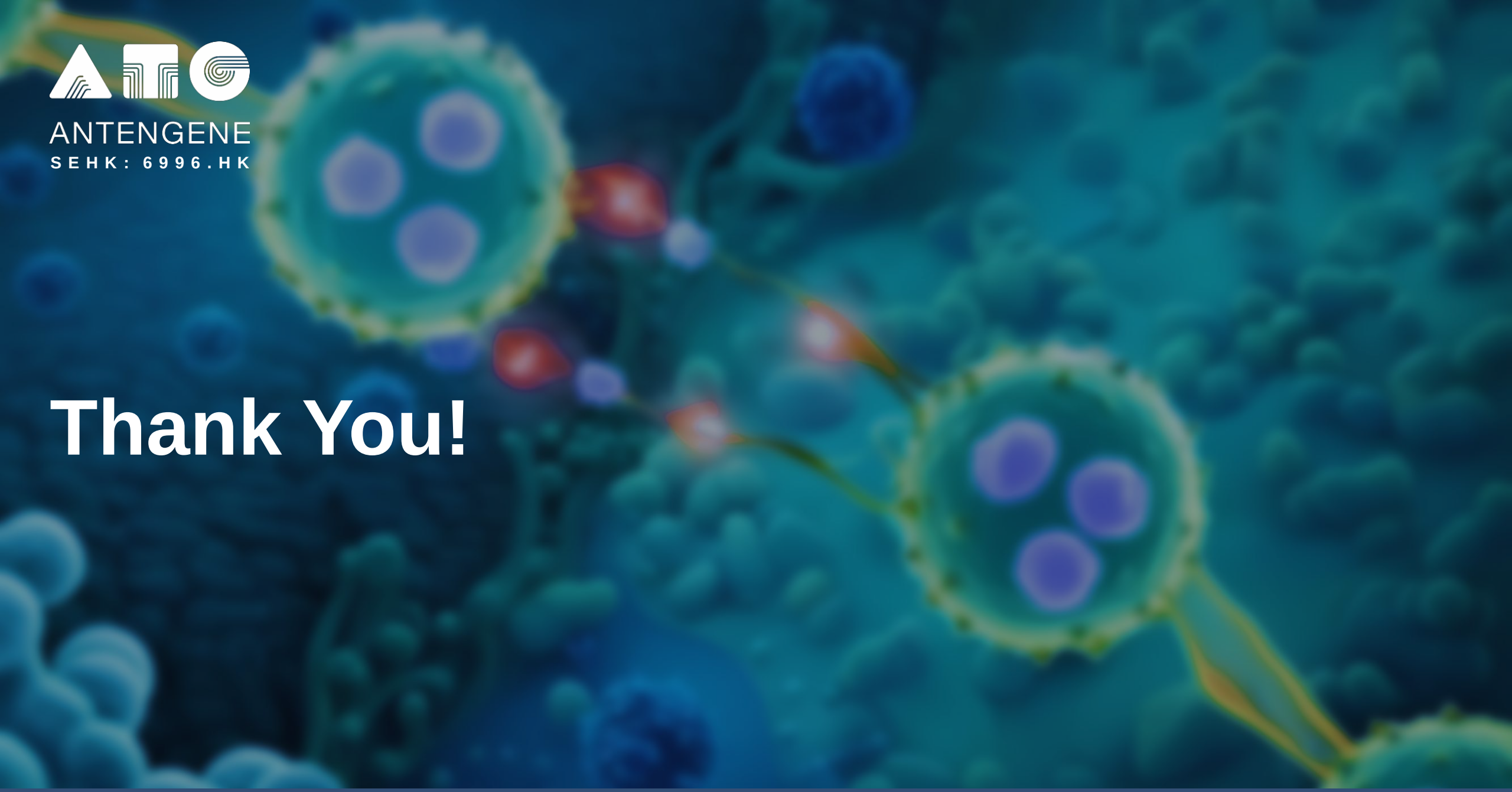
Antengene: Well Positioned for Long-term Growth with Expanding Revenue, Advancing R&D and Strong Cash Position

ANTENGENE

In-house Developed Drugs Entering Pivotal Trials and Ready for BD

Multi-market Revenue Ramp Up

3 Years Cash Runway



Thank You!

BD Contact: ariel.guo@antengene.com | IR Contact: jon.yau@antengene.com