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Antengene 2025 R&D Day

November 2025

Treating Patients Beyond Borders

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发言嘉宾 / Speakers Today

特邀嘉宾 / Guest Speaker

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德琪医药
ANTENGENE

Revolutionizing Patient Outcomes with Innovative R&D

Jay Mei, M.D., Ph.D.
Founder, Chairman, and Chief Executive Officer

Antengene Today

4 Focused R&D Areas

- Antibody Drug Conjugates (ADC)
- T Cell Engagers (TCE)
- Immuno-Oncology (IO)
- Autoimmune Diseases

10 Markets APAC Commercialization

* Approved markets in China also includes Taiwan, Hong Kong, and Macau

1 Proprietary Technology Platform

(AnTenGager™– 2nd Generation "2+1" TCE Platform with Steric Hindrance Masking Technology)

"Plug and Play" Disease Associated Antigens (DAA)

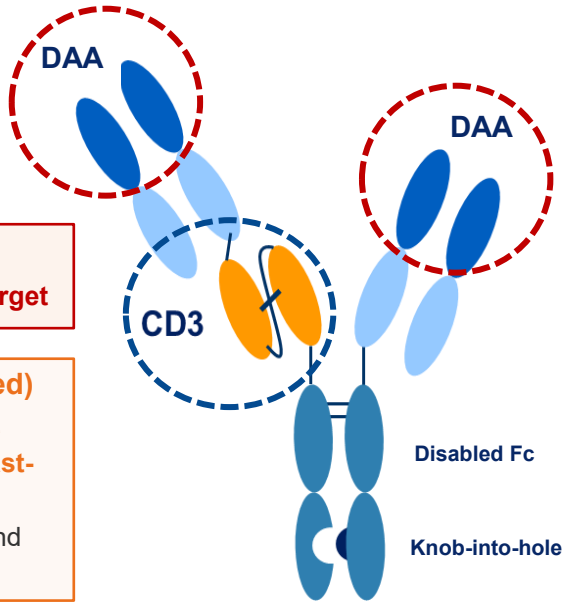
- Compatible with diverse DAAs, enabling the discovery & development of TCEs across multiple therapeutic areas

Bivalent Binding of DAA

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

Proprietary CD3 Sequences (Patented)

- 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**



Steric Hindrance Masking Technology

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

Antengene Pipeline Overview – Globally First / Best-in-Class Pipeline Across Novel Modalities

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Antibody Drug Conjugates (ADCs)

ATG-022 (CLDN18.2) *Phase II*	CLDN18.2+ Gastric Cancer (GC) and Other Solid Tumors	CLDN18.2 ADC with Efficacy Across the Widest Patient Population; BTD in GC
ATG-125 (B7-H3 x PD-L1) *Pre-clinical*	Solid Tumors	IO+ADC in One Drug
CD24 *Pre-clinical*	Solid Tumors	IO+ADC in One Drug

Immuno-Oncology (IO)

ATG-037 (CD73) *Phase Ib/II*	CPI-resistant Melanoma and Non-small Cell Lung Cancer	Oral Bioavailable; Demonstrated Efficacy in CPI-resistant Patients
ATG-101 (PD-L1 x 4-1BB) *Phase I*	Solid Tumors	No Liver Toxicity
ATG-031 (CD24) *Phase I*	Solid Tumors	First-in-class Myeloid Regulator

Autoimmune Diseases

ATG-201 (CD19 x CD3) *IND-enabling*	B Cell Driven Autoimmune Diseases	Deep B Cell Depletion with Low CRS
ATG-207 (Undisclosed Bifunctional Biologics) *Discovery*	T Cell Driven Autoimmune Diseases	First-in-Class; Induces T_{reg} and T Cell Exhaustion

T Cell Engagers (TCEs)

ATG-201 (CD19 x CD3) *IND-enabling*	B Cell Driven Autoimmune Diseases	Deep B Cell Depletion with Low CRS
ATG-106 (CDH6 x CD3) *Pre-clinical*	Ovarian Cancer and Kidney Cancer	First-in-Class CDH6 TCE
ATG-112 (ALPPL2 x CD3) *Pre-clinical*	Gynecological Tumors and Lung Cancer	First-in-Class ALPPL2 TCE
ATG-110 (LY6G6D x CD3) *Pre-clinical*	Microsatellite Stable (MSS) Colorectal Cancer	For IO-resistant Colorectal Cancer
ATG-021 (GPRC5D x CD3) *Pre-clinical*	Multiple Myeloma	
ATG-102 (LILRB4 x CD3) *Pre-clinical*	Acute Myeloid Leukemia and Chronic Myelomonocytic Leukemia	Biparatopic
ATG-107 (FLT3 x CD3) *Pre-clinical*	Acute Myeloid Leukemia	
ATG-115 (Undisclosed Bispecific TCE) *Pre-clinical*	Liver Cancer	Novel TAA Discovered by AI
Undisclosed Trispecific TCE *Discovery*	Metastatic Castration-resistant Prostate Cancer	First-in-Class
Undisclosed Trispecific TCE *Discovery*	Small Cell Lung Cancer and Neuroendocrine Tumors	First-in-Class

Key Highlights for Today

ANTENGENE

Clinical Highlights

ATG-022

Globally Best-in-Class (BIC) Claudin 18.2 ADC

- ✓ Best-in-class **efficacy (IHC - 1+ ≥ 1%)**, **mOS (14.72 months)**, and **safety profile (Gr ≥ 3 TRAE of 16.1%)** across modalities
- ✓ Potential to **transform 1L treatment** in gastric cancer via **combo with anti-PD-1 and chemotherapy**

ATG-037

Globally BIC Oral CD73 Small Molecule Inhibitor

- ✓ Proof of concept in CPI-resistant tumors, supported by encouraging activity in **CPI-resistant melanoma and non-small cell lung cancer**
- ✓ Strong potential for **broader tumor expansion** and **NextGen IO combo strategies**

ATG-101

PD-L1 x 4-1BB Bispecific Antibody with No Liver Tox

- ✓ Targeting **"cold", "hot",** and **CPI-resistant / relapsed tumors** with **combo potential**

Discovery and Pre-clinical Highlights

Antibody Drug Conjugates

ATG-125

B7-H3 x PD-L1 Bispecific ADC

IO + ADC for Solid Tumors

AnTenGager™

Proprietary Next Generation "2+1" TCE Platform

ATG-201

CD19 x CD3 TCE

B Cell Related Autoimmune Diseases

ATG-106

CDH6 x CD3 TCE

Ovarian Cancer and Kidney Cancer

ATG-112

ALPPL2 x CD3 TCE

Gynecological Tumors and Lung Cancer

Autoimmune Diseases

ATG-207

Undisclosed Bifunctional Biologic

T Cell Driven Autoimmune Diseases

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Targeting CLDN18.2 for Tumor Therapy

Investigator's Insights on ATG-022

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NCC NATIONAL CANCER CENTER 国家癌症中心

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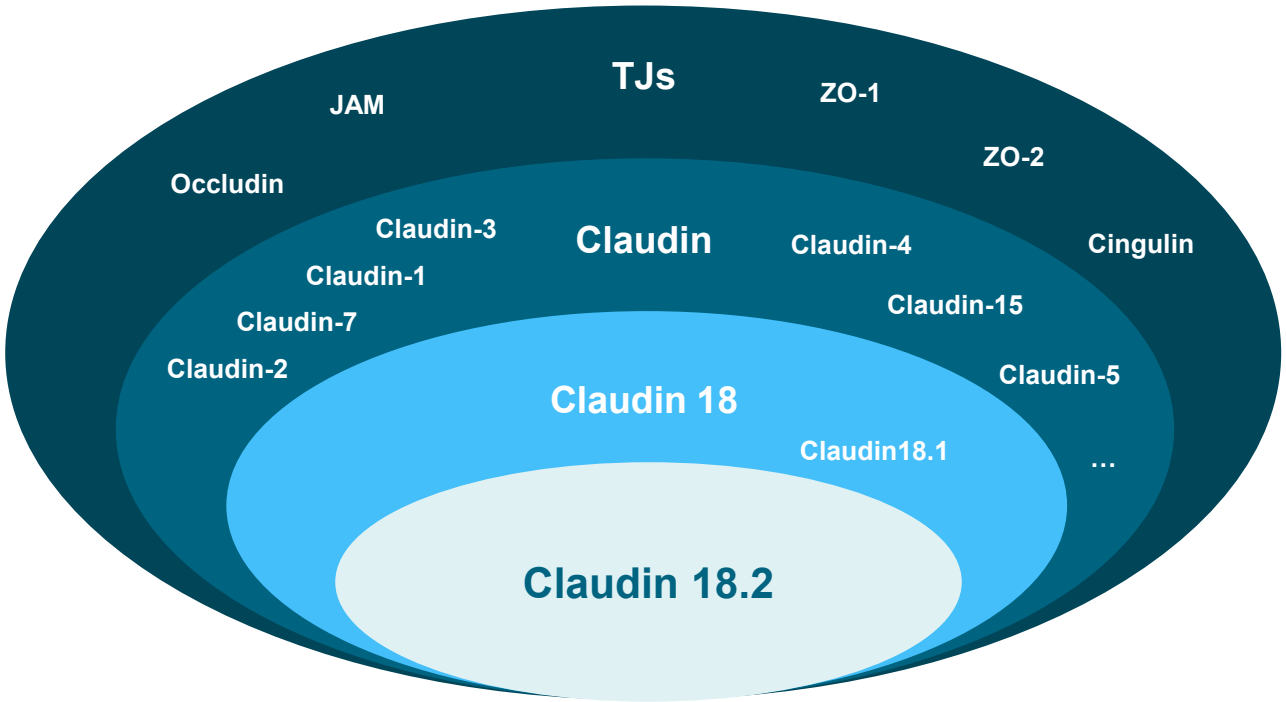
Claudin 18.2: An Over-Expressed Tumor Antigen in Multiple Tumor Types

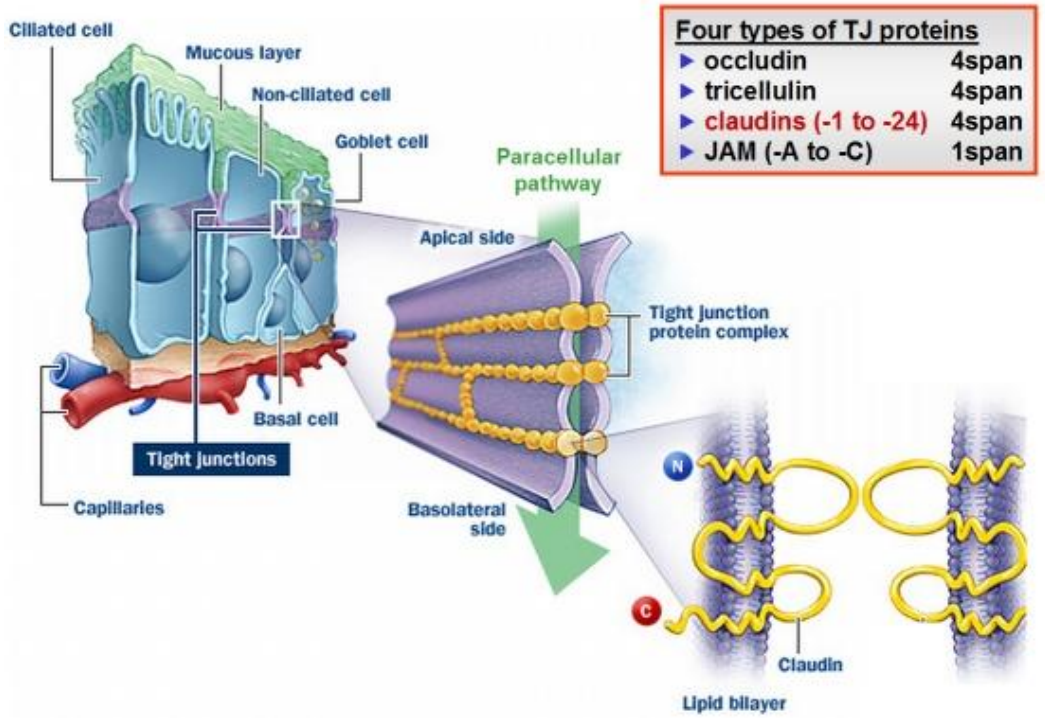
CLDN18.2 in Healthy Tissues

- ✓ Claudins form **tight junctions** that control movement between cells
- ✓ CLDN18.2 is expressed **only in differentiated gastric mucosal epithelial cells**
- ✓ **Very limited expression in healthy tissues** and is absent in undifferentiated gastric stem cells

CLDN18.2 in Cancer

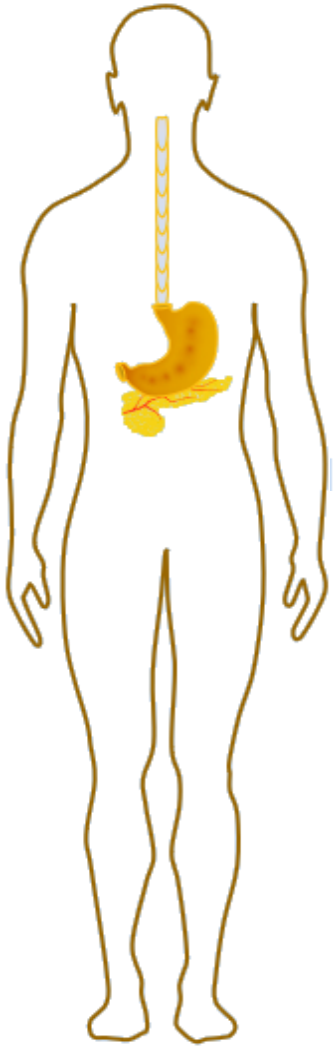
- ✓ CLDN18.2 is **overexpressed in multiple cancer types** (gastric, pancreatic, esophageal, gynecologic tumors, etc.) – **promising marker for diagnosing and treating multiple tumor types**





Source: IUBMB Life. 2009 Apr;61(4)431-7., Biomark Res . 2022 May 31;10(1):38. , https://klinphys.charite.de/for721/f_summary_e.htm

Claudin 18.2: An Over-Expressed Tumor Antigen in Multiple Tumor Types (Cont.)



Esophagus Cancer

- Positive in **18.2%** primary adenocarcinoma
- Positive in **17.9%** regional lymph node metastasis

Gastric Cancer

- Positive in **87%** primary tumors
- Positive in **80%** lymph node metastasis

Pancreatic Cancer

- Positive in **45%-90%** ductal adenocarcinoma

Other Tumors

- Claudin 18.2 mRNA expression was detected in multiple tumor types including NSCLC, ovarian cancer, and colorectal cancer

Tumor	Sample Size	Claudin 18.2 Positive
Gastric Cancer/GEJC[1] (Global)	3,576	**73.3%**
Gastric Cancer[2] (China)	300	**72%**
Gastric Cancer[3] (Japan)	262	**87%**
Gastric Signet-Ring Cell Carcinoma (SRCC)[3]	105	**95.2%**
Pancreatic Cancer[3]	174	**59.2%**
Pancreatic Neuroendocrine Tumors[3]	25	**20%**
Esophageal Cancer[4]	22	**78%**

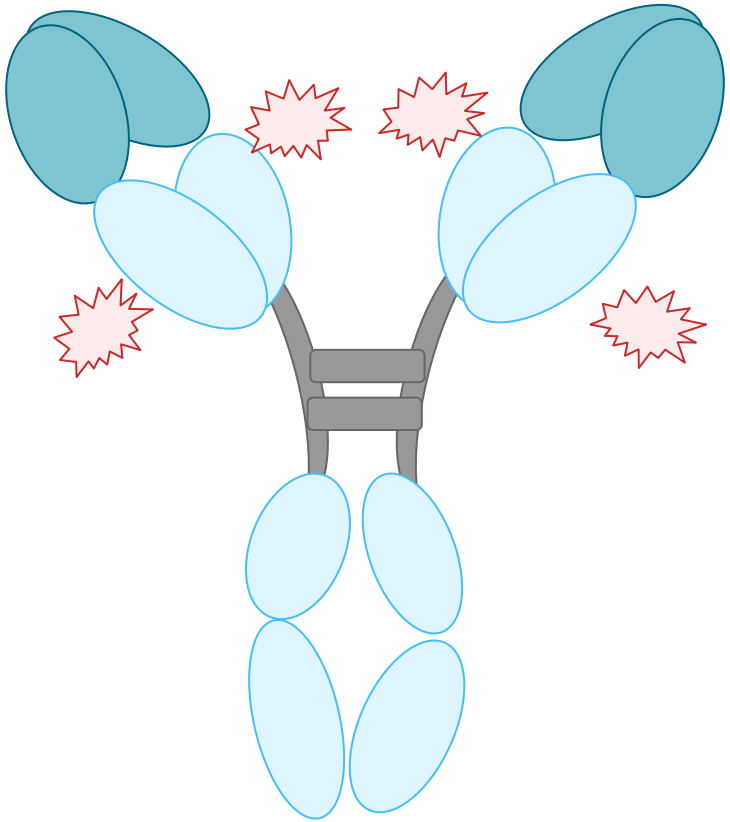
Source:
1. Shitara, K., Xu, RH., Ajani, J.A. et al. Global prevalence of claudin 18 isoform 2 in tumors of patients with locally advanced unresectable or metastatic gastric or gastroesophageal junction adenocarcinoma. Gastric Cancer 27, 1058–1068 (2024)
2. Journal for ImmunoTherapy of Cancer 2022;10:doi: 10.1136/jitc-2022-SITC2022.0105
3. Biomark Res . 2022 May 31;10(1):38
4. Sahin U, Koslowski M, Dhaene K, Usener D, Brandenburg G, Seitz G, Huber C, Türeci O. Claudin-18 splice variant 2 is a pan-cancer target suitable for therapeutic antibody development. Clin Cancer Res. 2008;14:7624–7634. doi: 10.1158/1078-0432.CCR-08-1547..

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

Molecular Design of ATG-022

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

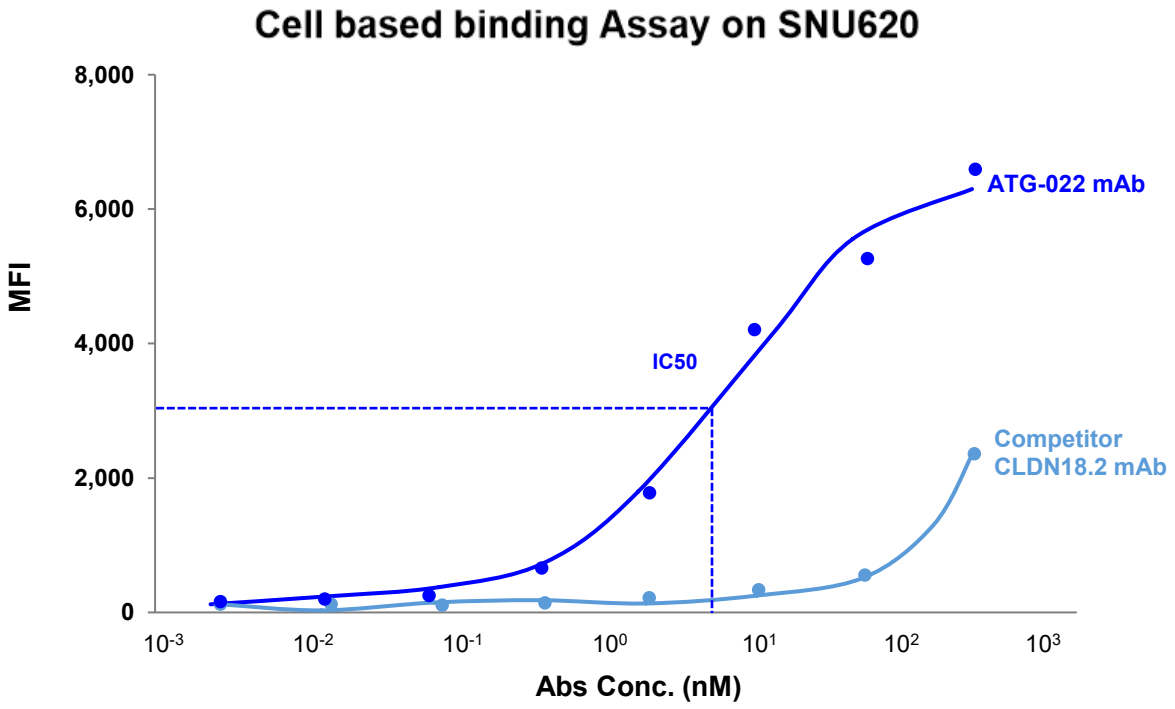
- ✓ **Efficacy across all CLDN18.2 expression levels**
- ✓ **Devoid of systemic toxicities**
- ✓ **Preliminary efficacy** observed in a **non-GI tumor type**

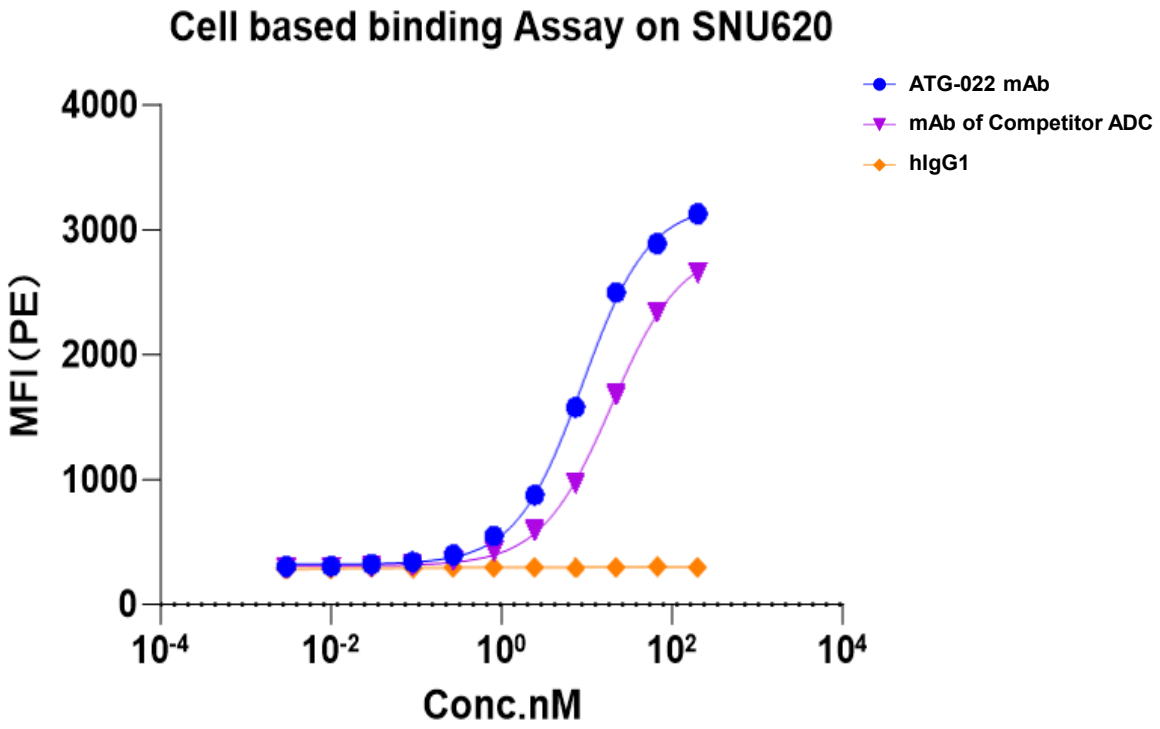
ATG-022 mAb Demonstrated Higher Binding Affinity Than a Competitor CLDN18.2 mAb and the mAb Component of a Competitor ADC in Tumor Cells Expressing Low Claudin 18.2

- The mAb of ATG-022 demonstrated **higher binding affinity (EC) to tumor cells expressing low Claudin18.2** (SNU620) than both competitive benchmarks, including a competitor CLDN18.2 mAb and the mAb component of a competitor ADC

	ATG-022 mAb	Competitor CLDN18.2 mAb
EC50	5.317nM	NE

	ATG-022 mAb	mAb of Competitor ADC	hIgG1
EC50	8.864nM	19.39nM	NE



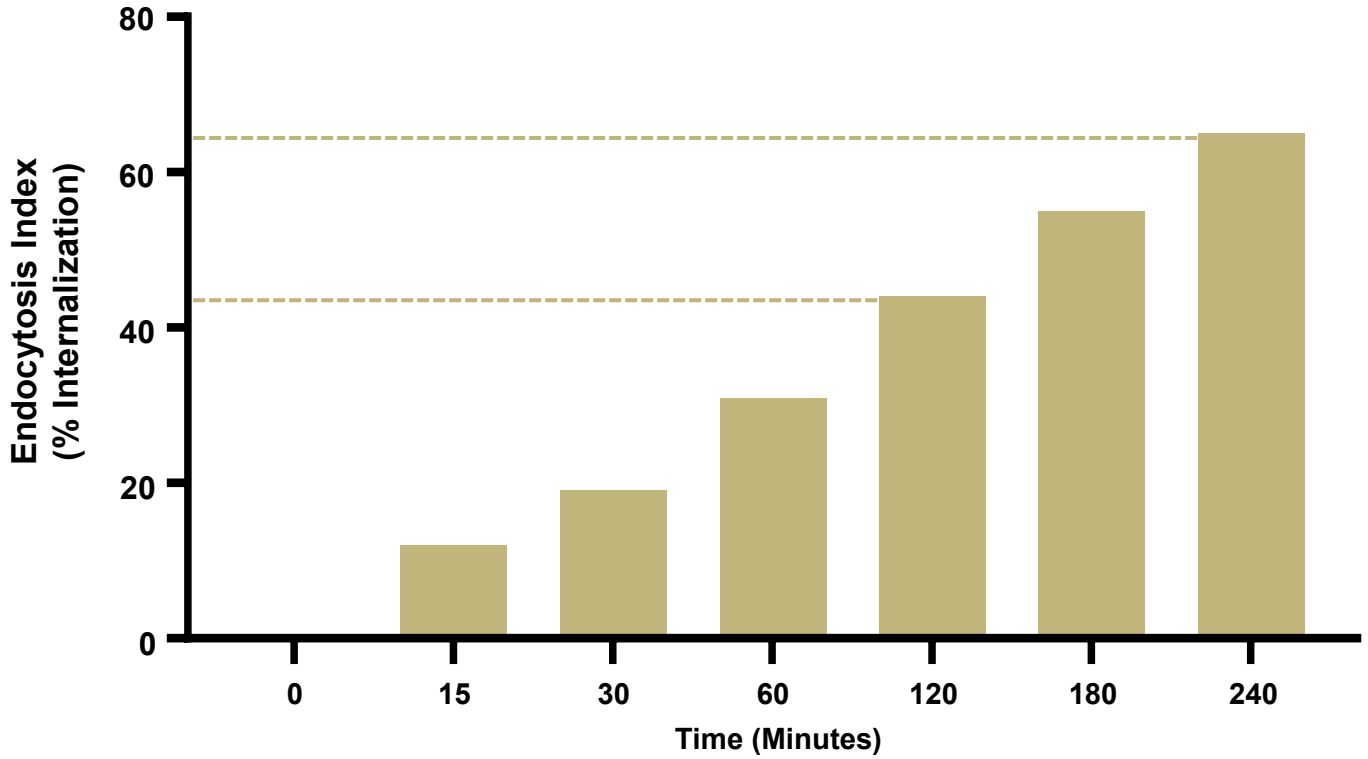


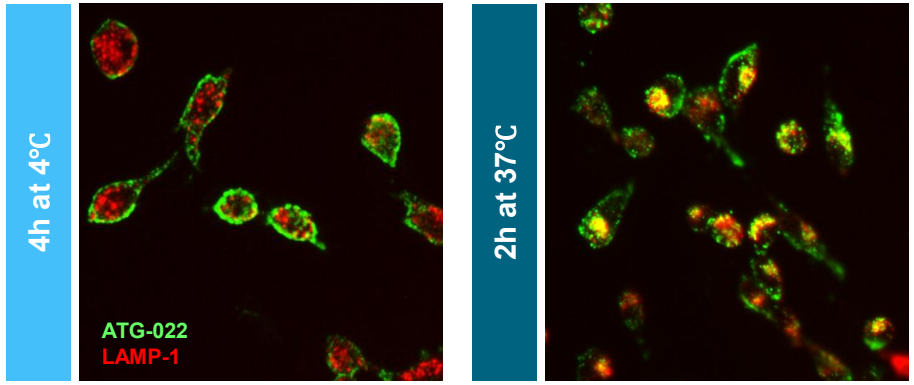
Source: AACR 2022. Cell based binding assays (EC_{50}). IMAB362 = zolbetuximab (Astellas), in Phase III studies

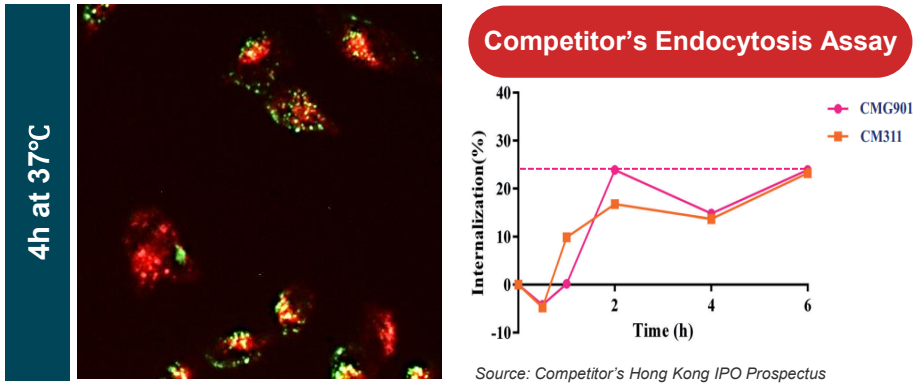
ATG-022 Demonstrated Better Internalization Capability vs. Competitor ADC

- **Higher Internalization:** ATG-022 achieves **over 60% internalization at 240 minutes**, compared to ~20% for competitor's ADC and the antibody component of the ADC at their peak
- **Faster Internalization:** ATG-022 reaches **over 40% internalization within 120 minutes**, while competitor's ADC and the antibody component of the ADC only achieve around 20-25% within the same timeframe

ATG-022 – Endocytosis Assay in CHO-L1-18.2 Cells







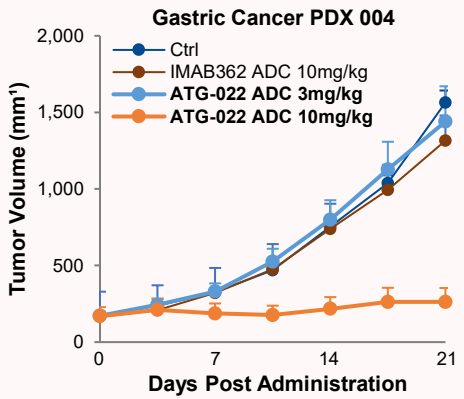
Competitor's Endocytosis Assay

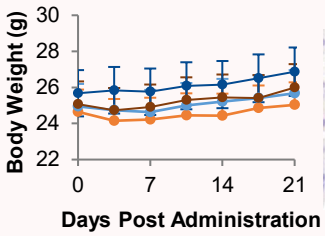
Source: Competitor's Hong Kong IPO Prospectus

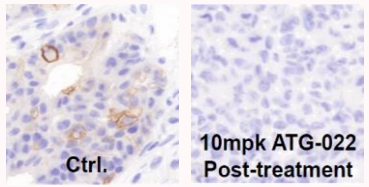
ATG-022 Demonstrated Strong *In Vivo* Efficacy in Various Claudin 18.2 Level PDX Models

Extremely Low Expression Level of Claudin 18.2

ATG-022 Inhibited Tumor Growth



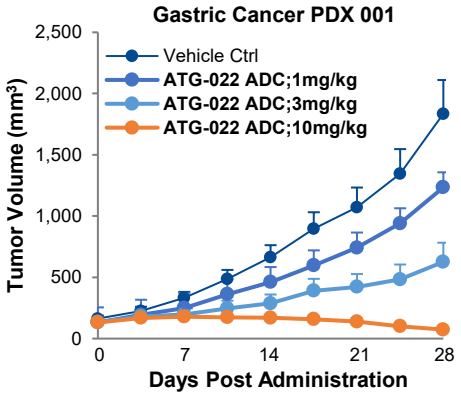


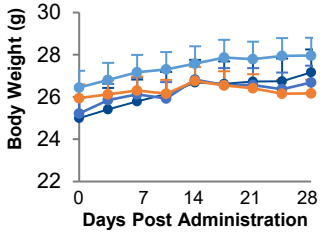


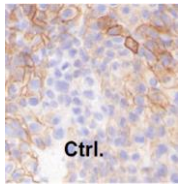
Model	CLDN18.2 Expression		TGI%		
	Positive%	IHC Intensity	BMK	3mpk	10mpk
PDX004	<5%	0~1+	NS	NS	93

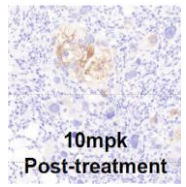
Moderate Expression Level of Claudin 18.2

ATG-022 Induced Tumor Regression (TR) or Complete Remission (CR)





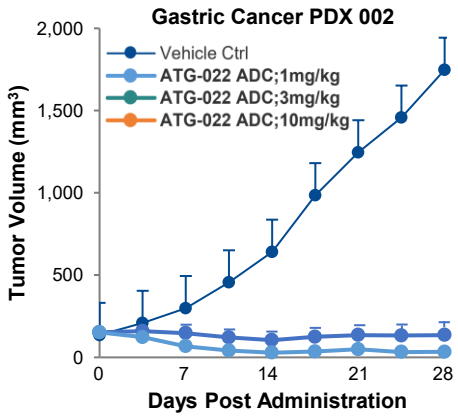


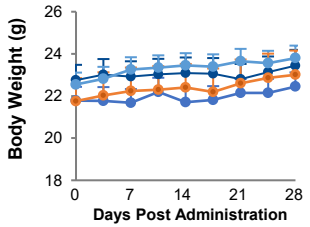


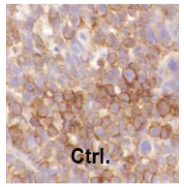
Model	CLDN18.2 Expression		TGI%		
	Positive%	IHC Intensity	1mpk	3mpk	10mpk
PDX001	60%	1+	35%	72%	TR

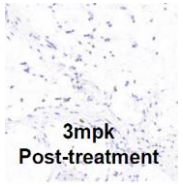
High Expression Level of Claudin 18.2

ATG-022 Induced Tumor Regression (TR) or Complete Remission (CR)

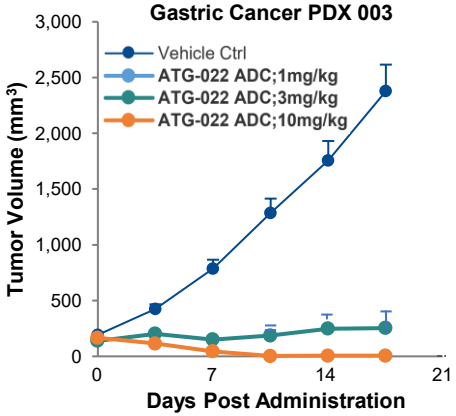


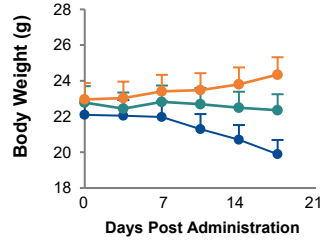


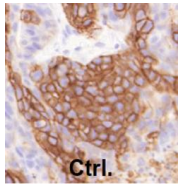


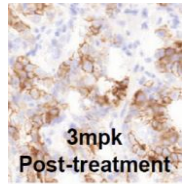


Model	CLDN18.2 Expression		TGI%		
	Positive%	IHC Intensity	1mpk	3mpk	10mpk
PDX002	90%	1+~2+	TR	TR	CR









Model	CLDN18.2 Expression		TGI%	
	Positive%	IHC Intensity	3mpk	10mpk
PDX003	70%	2+~3+	94%	CR

Source: AACR 2022.

ATG-022: Phase I/II "CLINCH" Trial Ongoing Study Design

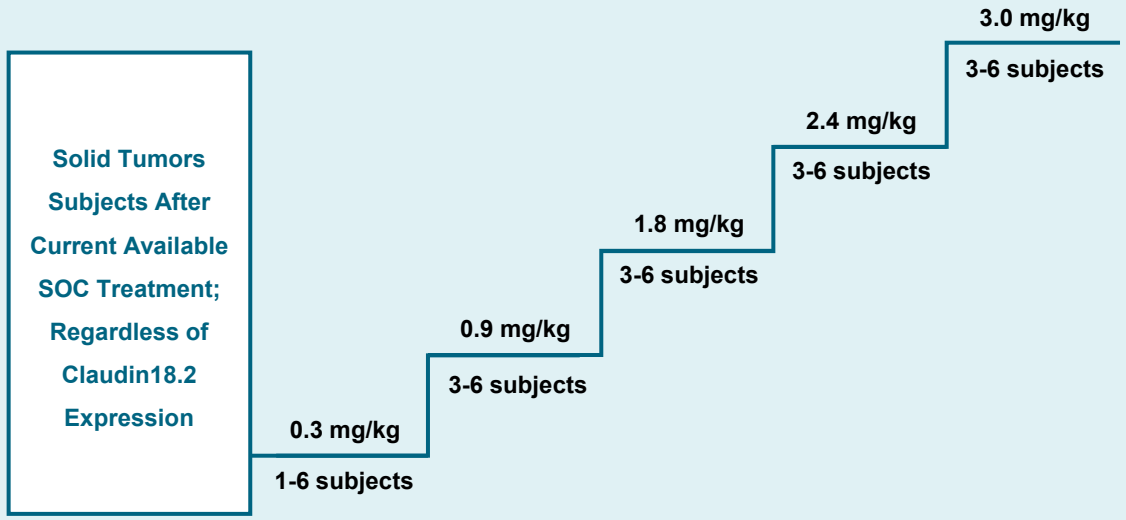
Population: Subjects with solid tumors, regardless of Claudin 18.2 expression and histology

Primary Endpoints: Safety and tolerability, MTD and/or RP2D

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

Phase I: Dose Escalation

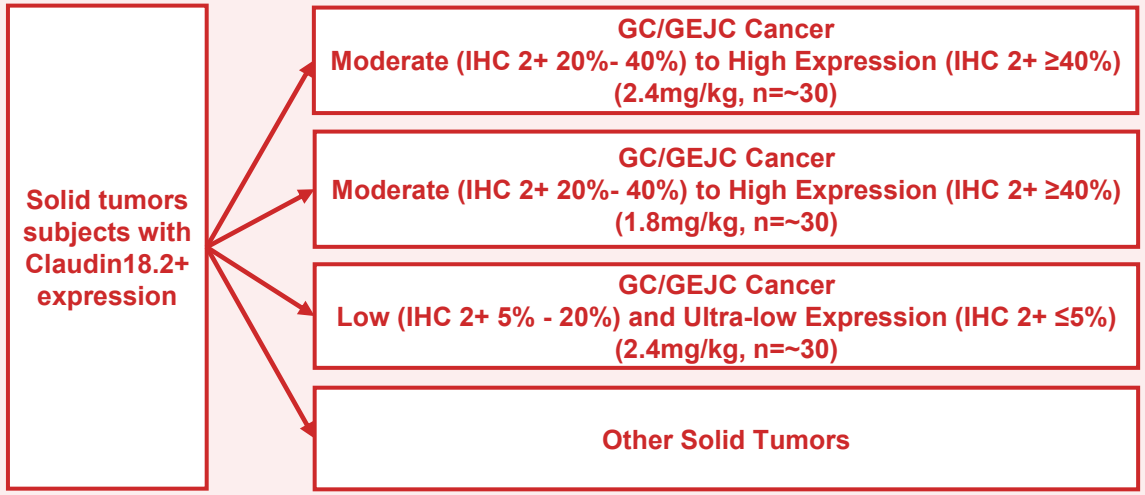
(Multiple Tumor Types without Pre-screening for Claudin 18.2 Expression Levels)



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

Phase II: Dose Expansion

20~30 Subjects in Each Tumor Type / Cohort



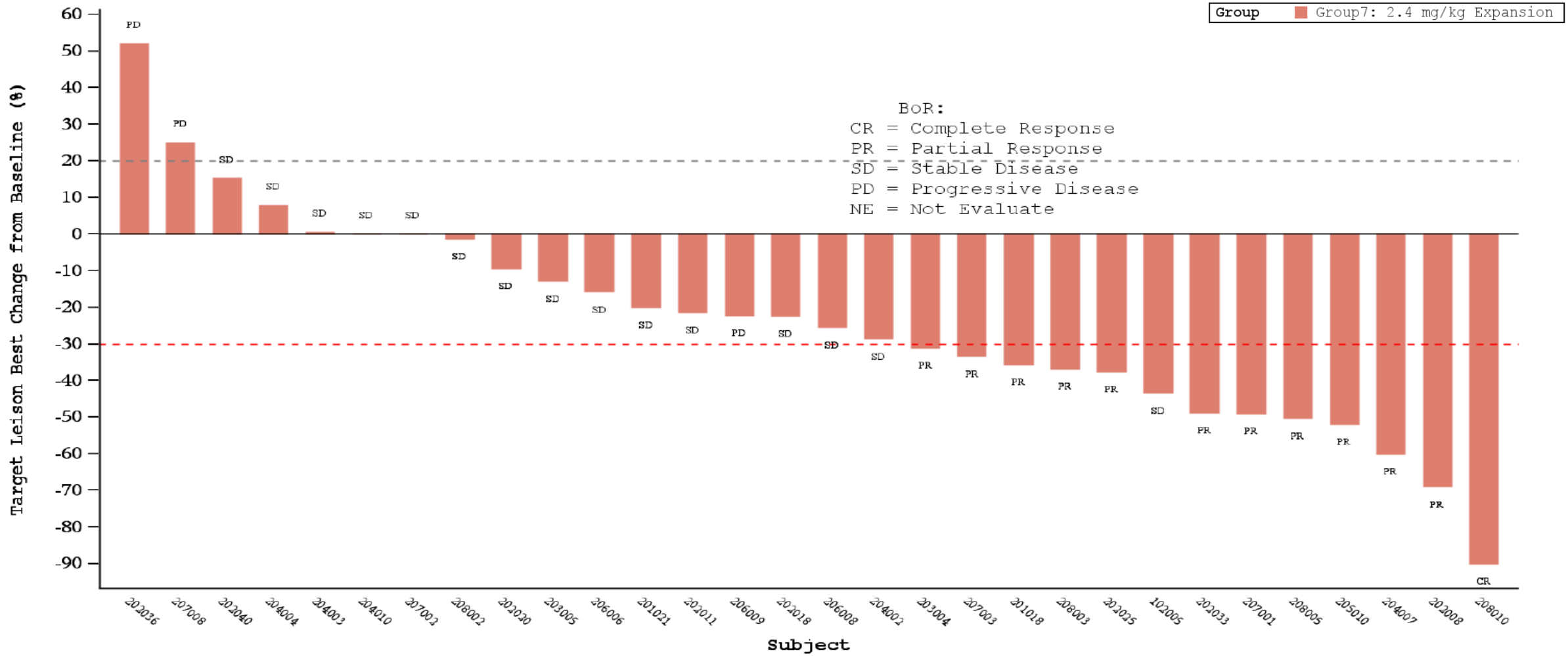
Approximately 120 subjects, depending on the number of cohorts to be expanded.
CLDN18.2+ tumors only. No prior CLDN18.2 agents

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

CLDN18.2 Moderate to High Expressors (IHC 2+ > 20%; 2.4 mg/kg) – Waterfall Plot

Preliminary Efficacy in CLDN18.2+ Gastric Cancer (As of November 10, 2025):

- IHC Staining - 2+, > 20% (CLDN18.2 Moderate to High Expressors): **Dose Expansion 2.4 mg/kg Cohort – ORR of 40%** (12/30) and **DCR of 90%** (27/30)

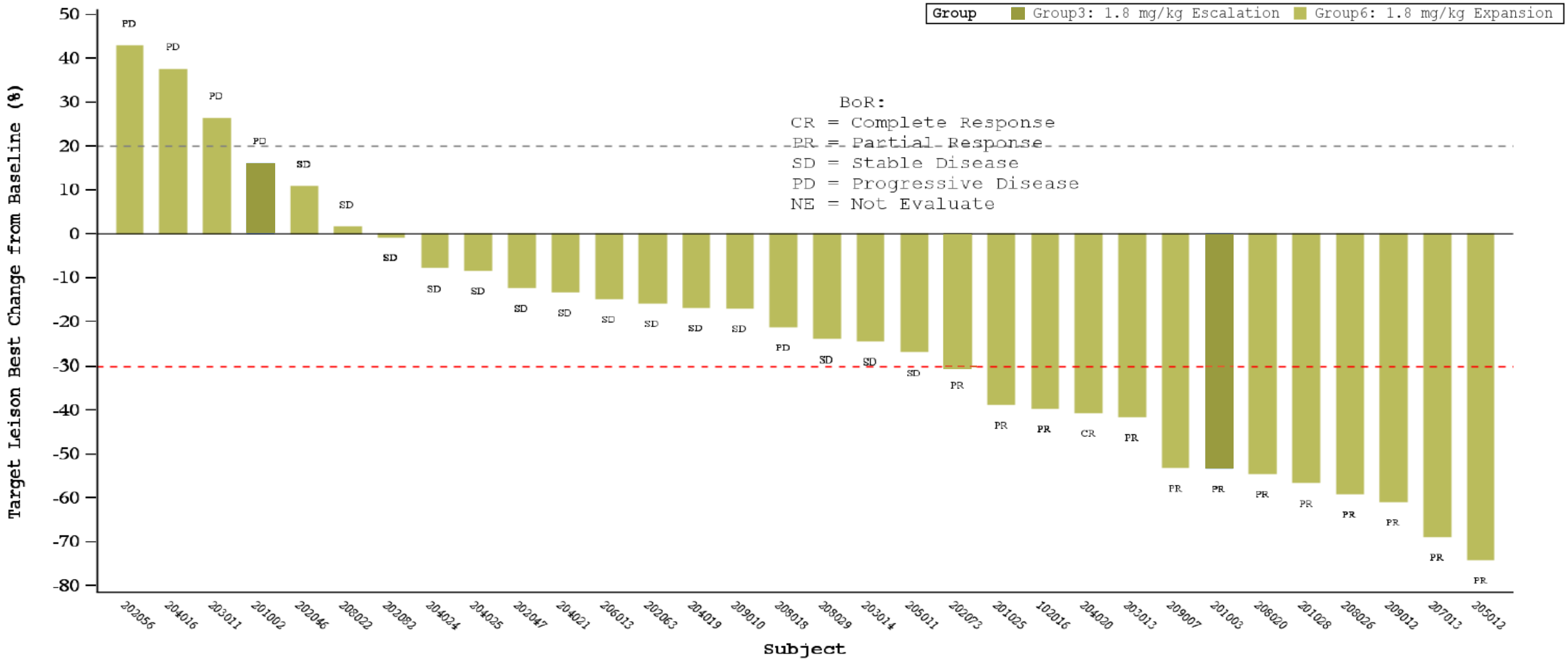


ATG-022: Efficacy Across the Widest Patient Population in CLDN18.2+ Gastric Cancer Including From High to Ultra-low Expressors

CLDN18.2 Moderate to High Expressors (IHC 2+ > 20%; 1.8 mg/kg) – Waterfall Plot

Preliminary Efficacy in CLDN18.2+ Gastric Cancer (As of November 10, 2025):

- IHC Staining - 2+, > 20% (CLDN18.2 Moderate to High Expressors): **Dose Expansion 1.8 mg/kg Cohort – ORR of 40%** (12/30) and **DCR of 86.7%** (26/30)

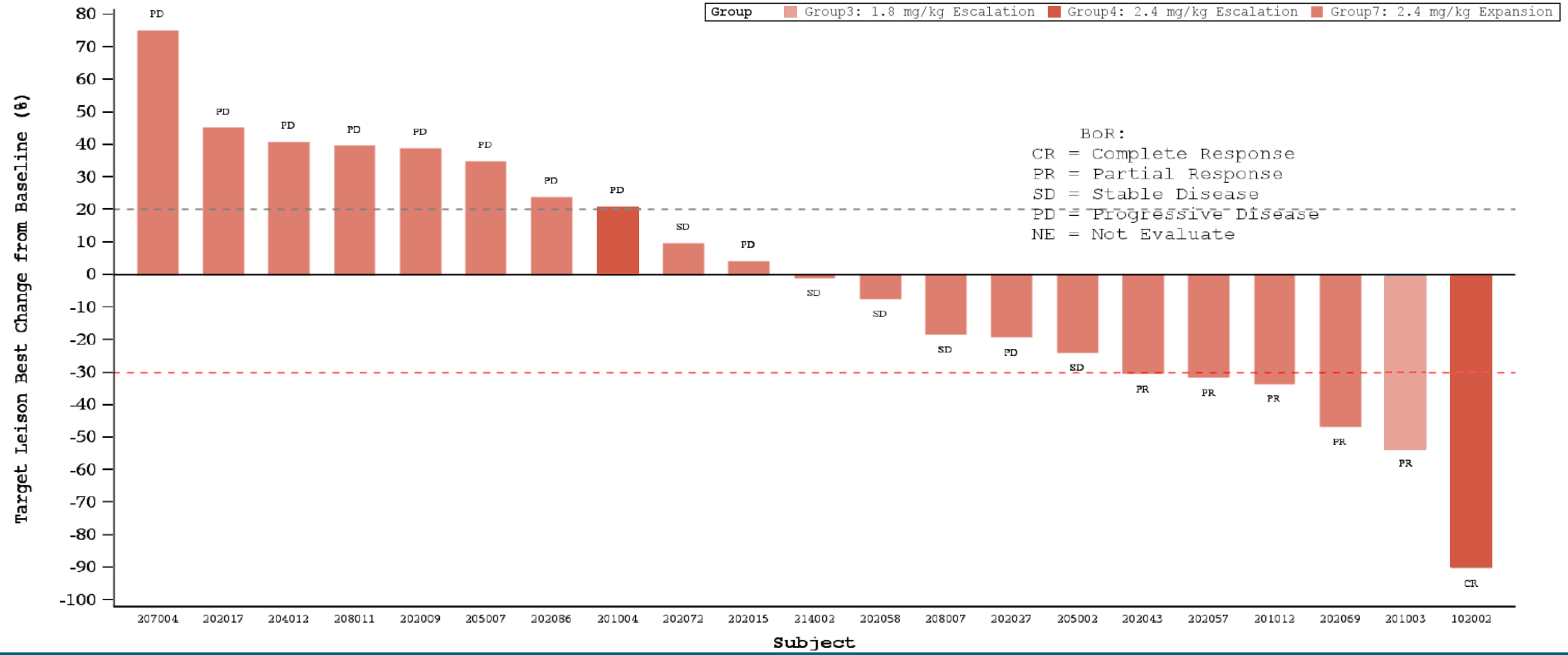


ATG-022: Efficacy Across the Widest Patient Population in CLDN18.2+ Gastric Cancer Including From High to Ultra-low Expressors

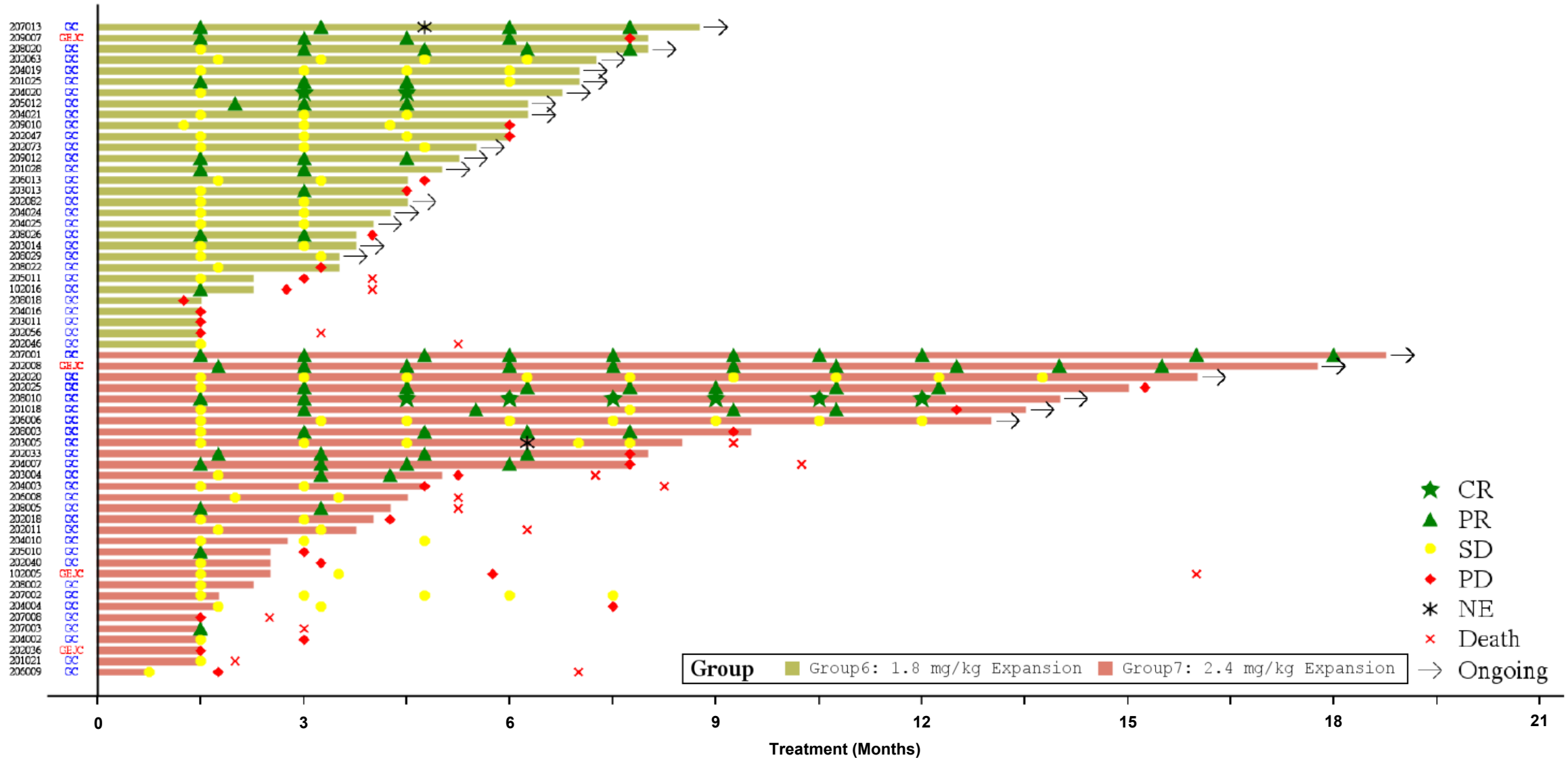
CLDN18.2 Low and Ultra-low Expressors (IHC 2+ ≤ 20%; 1.8 - 2.4mg/kg) – Waterfall Plot

Preliminary Efficacy in CLDN18.2+ Gastric Cancer (As of November 10, 2025):

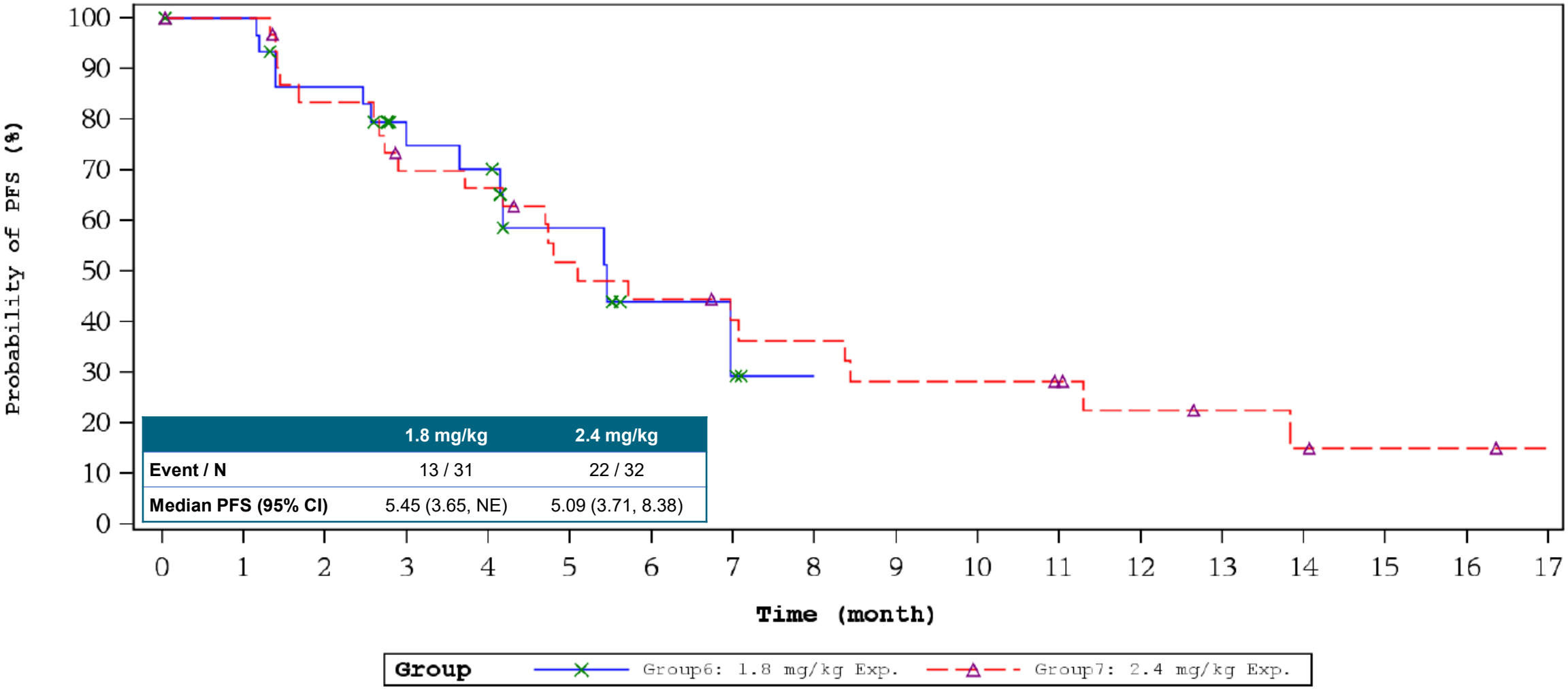
- IHC Staining - 2+, ≤ 20% (CLDN18.2 Low and Ultra-low Expressors): **Efficacious Dose Range of 1.8 – 2.4 mg/kg – ORR of 28.6%** (6/21) and **DCR of 52.4%** (11/21)



ATG-022: Durable Responses Demonstrated Across Both Dosage Levels

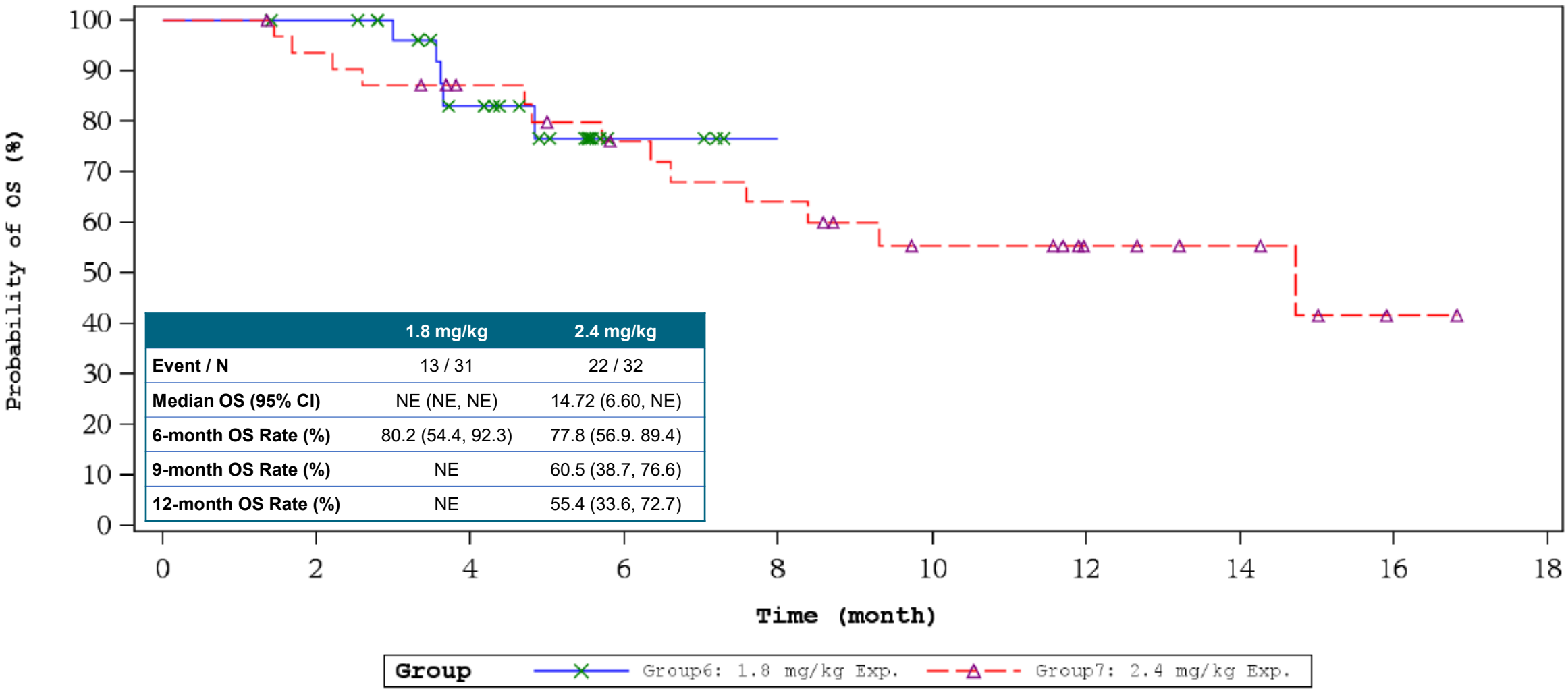


ATG-022: Encouraging Median PFS Outcomes in CLDN18.2+ (2+ > 20%) Gastric Cancer



	1.8 mg/kg	2.4 mg/kg
Event / N	13 / 31	22 / 32
Median PFS (95% CI)	5.45 (3.65, NE)	5.09 (3.71, 8.38)

ATG-022: Best-in-Class Median OS of 14.72 Months in CLDN18.2+ (2+ > 20%) Gastric Cancer



	1.8 mg/kg	2.4 mg/kg
Event / N	13 / 31	22 / 32
Median OS (95% CI)	NE (NE, NE)	14.72 (6.60, NE)
6-month OS Rate (%)	80.2 (54.4, 92.3)	77.8 (56.9. 89.4)
9-month OS Rate (%)	NE	60.5 (38.7, 76.6)
12-month OS Rate (%)	NE	55.4 (33.6, 72.7)

ATG-022: Favourable Safety Profile

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

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 1.8mg/kg N=31	Expansion 2.4mg/kg N=65
Subjects with at least one TEAE	1 (100)	3 (100)	3 (100)	3 (100)	6 (100)	30 (96.8)	65 (100)
Serious TEAE	1 (100)	0 (0)	1 (33.3)	2 (66.7)	5 (83.3)	6 (19.4)	30 (46.2)
Grade ≥ 3 TEAE	0 (0)	1 (33.3)	2 (66.7)	2 (66.7)	6 (100)	8 (25.8)	38 (58.5)
TEAE Leading to Dose Modification	0 (0)	1 (33.3)	1 (33.3)	1 (33.3)	5 (83.3)	6 (19.4)	34 (52.3)
TEAE Leading to Dose Reduction	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (3.2)	12 (18.5)
TEAE Leading to Dose Interruption	0 (0)	1 (33.3)	1 (33.3)	1 (33.3)	5 (83.3)	5 (16.1)	30 (46.2)
TEAE Leading to Drug Withdrawn	0 (0)	0 (0)	1 (33.3)	0 (0)	2 (33.3)	1 (3.2)	8 (12.3)
TEAE Leading to Death	0 (0)	0 (0)	1 (33.3)	1 (33.3)	2 (33.3)	1 (3.2)	12 (18.5)

Preliminary Data as of November 10, 2025

ATG-022: Favourable Safety Profile

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

TRAEs							
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 1.8mg/kg N=31	Expansion 2.4mg/kg N=65
Subjects with at least one TRAE	0 (0)	2 (66.7)	3 (100)	3 (100)	6 (100)	30 (96.8)	60 (92.3)
Serious TRAE	0 (0)	0 (0)	0 (0)	1 (33.3)	4 (66.7)	2 (6.5)	20 (30.8)
Grade ≥ 3 TRAE	0 (0)	1 (33.3)	1 (33.3)	1 (33.3)	6 (100)	5 (16.1)	33 (50.8)
TRAE Leading to Dose Modification	0 (0)	1 (33.3)	0 (0)	1 (33.3)	5 (83.3)	3 (9.7)	32 (49.2)
TRAE Leading to Dose Reduction	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (3.2)	12 (18.5)
TRAE Leading to Dose Interruption	0 (0)	1 (33.3)	0 (0)	1 (33.3)	5 (83.3)	2 (6.5)	28 (43.1)
TRAE Leading to Drug Withdrawn	0 (0)	0 (0)	1 (33.3)	0 (0)	2 (33.3)	0 (0)	3 (4.6)
TRAE Leading to Death	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (1.5)

Preliminary Data as of November 10, 2025

ATG-022: No Ophthalmological Toxicities or Interstitial Lung Disease

CLINCH – TRAE By Preferred Term (PT) in ≥ 10% Patients (1.8 & 2.4 mg/kg)

TRAEs

Adverse Events	Escalation (1.8mg/kg) (N=3)		Expansion (1.8mg/kg) (N=31)		Escalation (2.4mg/kg) (N=3)		Expansion (2.4mg/kg) (N=65)	
Preferred Term; n (%)	**Any Grade**	**Grade ≥ 3**	**Any Grade**	**Grade ≥ 3**	**Any Grade**	**Grade ≥ 3**	**Any Grade**	**Grade ≥ 3**
Any TRAE (n, %)	3 (100)	1 (33.3)	30 (96.8)	5 (16.1)	3 (100)	1 (33.3)	60 (92.3)	33 (50.8)
Neutrophil count decreased	0 (0)	0 (0)	10 (32.3)	2 (6.5)	2 (66.7)	1 (33.3)	33 (50.8)	9 (13.8)
Nausea	2 (66.7)	0 (0)	8 (25.8)	0 (0)	1 (33.3)	1 (33.3)	32 (49.2)	2 (3.1)
White blood cell count decreased	0 (0)	0 (0)	9 (29.0)	0 (0)	1 (33.3)	0 (0)	31 (47.7)	2 (3.1)
Decreased appetite	1 (33.3)	0 (0)	7 (22.6)	1 (3.2)	2 (66.7)	0 (0)	30 (46.2)	8 (12.3)
Anaemia	0 (0)	0 (0)	16 (51.6)	1 (3.2)	0 (0)	0 (0)	31 (47.7)	5 (7.7)
Weight decreased	1 (33.3)	0 (0)	11 (35.5)	0 (0)	0 (0)	0 (0)	29 (44.6)	4 (6.2)
Vomiting	1 (33.3)	0 (0)	4 (12.9)	0 (0)	1 (33.3)	1 (33.3)	25 (38.5)	1 (1.5)
Hypoalbuminaemia	1 (33.3)	0 (0)	12 (38.7)	0 (0)	1 (33.3)	1 (33.3)	20 (30.8)	0 (0)
Malaise	0 (0)	0 (0)	4 (12.9)	0 (0)	0 (0)	0 (0)	18 (27.7)	2 (3.1)
ALT increased	1 (33.3)	1 (33.3)	6 (19.4)	0 (0)	0 (0)	0 (0)	12 (18.5)	0 (0)
AST increased	0 (0)	0 (0)	7 (22.6)	0 (0)	0 (0)	0 (0)	12 (18.5)	1 (1.5)
Alopecia	0 (0)	0 (0)	0 (0)	0 (0)	1 (33.3)	0 (0)	10 (15.4)	0 (0)
Constipation	0 (0)	0 (0)	3 (9.7)	0 (0)	0 (0)	0 (0)	10 (15.4)	1 (1.5)
Fatigue	0 (0)	0 (0)	4 (12.9)	0 (0)	1 (33.3)	0 (0)	9 (13.8)	1 (1.5)
Hypokalaemia	0 (0)	0 (0)	0 (0)	0 (0)	1 (33.3)	0 (0)	8 (12.3)	2 (3.1)
Upper abdominal pain	1 (33.3)	0 (0)	3 (9.7)	1 (3.2)	0 (0)	0 (0)	10 (15.4)	0 (0)
Diarrhoea	0 (0)	0 (0)	2 (6.5)	0 (0)	1 (33.3)	0 (0)	7 (10.8)	0 (0)
Platelet count decreased	0 (0)	0 (0)	3 (9.7)	0 (0)	0 (0)	0 (0)	9 (13.8)	1 (1.5)
Blood bilirubin increased	0 (0)	0 (0)	2 (6.5)	0 (0)	0 (0)	0 (0)	7 (10.8)	1 (1.5)
Peripheral neuropathy	0 (0)	0 (0)	3 (9.7)	0 (0)	1 (33.3)	0 (0)	7 (10.8)	1 (1.5)

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

Preliminary Data as of November 10, 2025

ATG-022 ("CLINCH" Study): Case Study of 71 y/o Male Gastric Cancer Patient Achieving Complete Response

Summary of Patient

Patient	83 y/o, Male, Metastatic Gastric Cancer
Initial Diagnosis	July 25th, 2022
CLDN18.2 Expression	1+ (15%), 2+ (50%), 3+ (0%)
Target Lesions (Baseline)	Liver Metastasis – 18 mm Hepatic Portal Metastasis – 22.13 mm Lymph Node – 19.9 mm
Treatment with ATG-022	2.4 mg/kg Q3W, C1D1 – October 14th, 2024

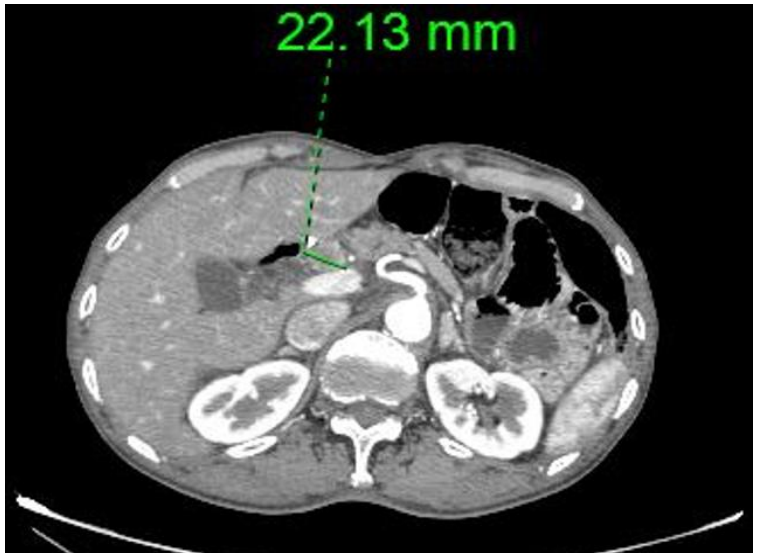
Prior Systemic Anti-Cancer Therapy

Regimen	Start Date	End Date
Oxaliplatin + Capecitabine	August 2022	November 2022
Lenvatinib + Paclitaxel + Envafolimab	April 2023	September 2023
Apatinib	January 2024	March 2024
Irinotecan	March 2024	June 2024

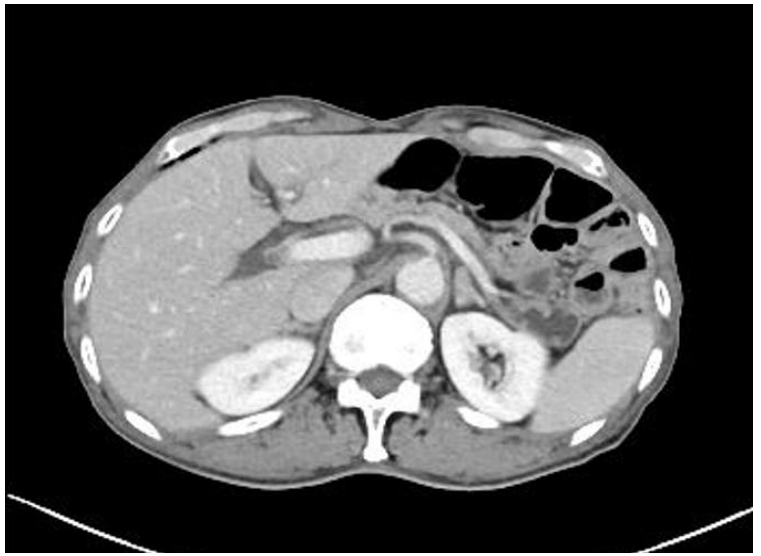
Tumor Evaluation

Week 12	PR
Week 18	CR
Week 24	CR
Week 30	CR
Week 36	CR
Week 42	CR
Week 48	CR

Hepatic Portal Metastasis – Baseline



Hepatic Portal Metastasis – Week 18 (CR)



ATG-022 Outperforms Competitor Molecules with Unprecedented Efficacy Across Gastric Cancer of All CLDN18.2 Expression Levels

842,800+ CLDN18.2+ Gastric Cancer Patients are Diagnosed Globally Each Year

ATG-022

ANTENGENE

CLDN18.2 **Moderate to High Expressors (IHC 2+ > 20%; 2.4 mg/kg): 40% ORR** (12/30), **90% DCR** (27/30), **Median PFS of 5.09 months** and **Median OS of 14.72 months**
CLDN18.2 **Moderate to High Expressors (IHC 2+ > 20%; 1.8 mg/kg): 40% ORR** (12/30) and **86.7% DCR** (26/30), and **Median PFS of 5.45 months**
CLDN18.2 **Low and Ultra-low Expressors (IHC 2+ ≤ 20%; 1.8 - 2.4 mg/kg): 28.6% ORR** (6/21) and **52.4% DCR** (11/21)

ADC 1
IHC Staining - 2+ ≥ 20%

ADC 3
IHC Staining - 2+ ≥ 40%

ADC 2 and ADC 4
IHC Staining - 2+ ≥ 50%

Zolbetuximab
astellas
IHC Staining - 2+ ≥ 75%

High and Moderate Expression | **Low and Ultra-low Expression**

Claudin 18.2 Expression Level Target Patient Population – Gastric Cancer

Preliminary Data as of November 10, 2025

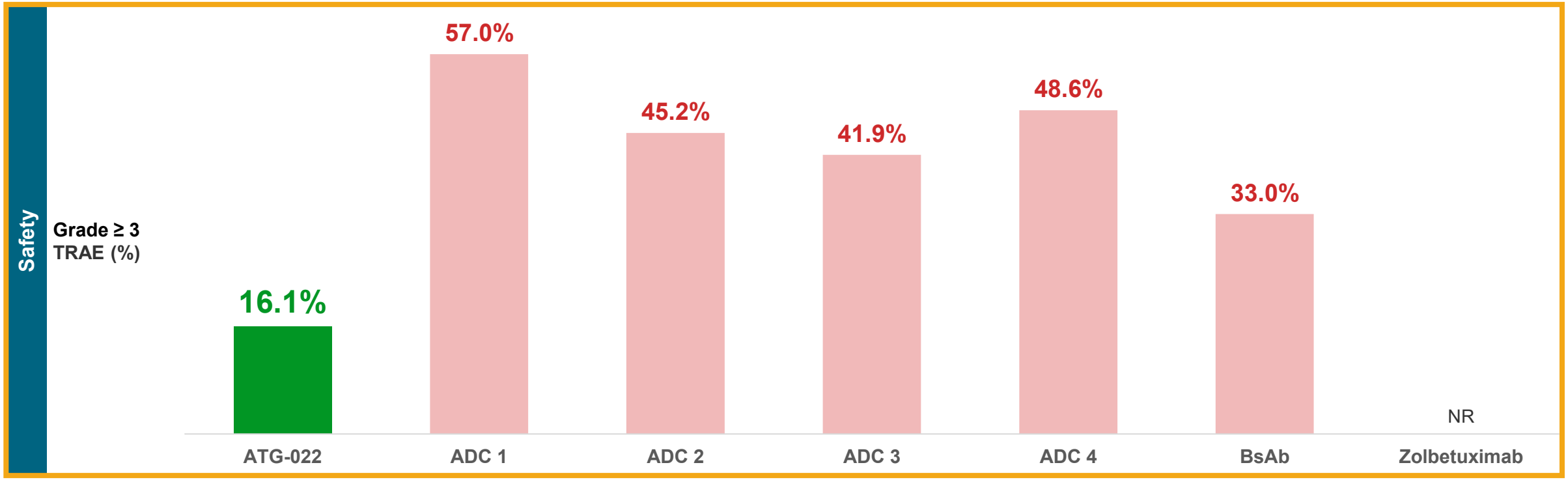
ATG-022 Demonstrated Best-In-Class Efficacy and mOS Across Modalities

	ATG-022[1] ANTENGENE	ADC 1	ADC 2	ADC 3	ADC 4	BsAb	Zolbetuximab astellas
Molecule Design	DAR 4 vc-MMAE	DAR 4 Cleavable-MMAE	DAR 4 MMAE	TOP1i DAR 4	TOP1i DAR4	CLDN18.2 x 4-1BB Bispecific Antibody	Monoclonal Antibody
Enrolment CLDN18.2 Threshold	IHC - **1+ ≥ 1%** *(Dose Expansion Cohorts)*	IHC - **2+/3+ ≥ 20%**	IHC - **2+/3+ ≥ 50%**	IHC - **2+ ≥ 40%**	IHC - **2+/3+ ≥ 50%**	IHC - 1+ ≥ 1%	IHC - **2+/3+ ≥ 75%**
ORR	**40%** (12/30; 1.8 mg/kg; 2+ > 20%) **40%** (12/30; 2.4 mg/kg; 2+ > 20%)	**25.0%** (1/4; 1.8 mg/kg)[2] 46.9% (15/32; 2.2mg/kg)[2] **22.2%** (10/45; 2.6mg/kg)[2]	**28.9%**	**38.8%** (19/49)	**36.7%** (11/30)	**17.8%** (8/45; 5 - 18 mg/kg; All Patients)[2] **17.9%** (7/39; 5 - 18 mg/kg; **2+ > 20%**)[2]	**9% (Overall); 14% (CLDN18.2 High Patients)**
DCR	**86.7%** (26/30; 1.8 mg/kg; 2+ > 20%) **90%** (27/30; 2.4 mg/kg; 2+ > 20%)	**50.0%** (2/4; 1.8 mg/kg)[2] **68.8%** (22/32; 2.2 mg/kg)[2] **62.2%** (28/45; 2.6mg/kg)[2]	**80.0%**	**87.8%** (43/49)	93.3% (28/30)	**48.9%** (22/45; 5 - 18 mg/kg; All Patients)[2] **48.7%** (19/39; 5 - 18 mg/kg; **2+ > 20%**)[2]	**23% (Overall); 31% (CLDN18.2 High Patients)**
Median PFS (95% CI)	**5.45 months** (3.65-NR; 1.8 mg/kg; 2+ > 20%)* **5.09 months** (3.71-8.38; 2.4 mg/kg; 2+ > 20%)	**4.8 months** (3.6-6.2; 2.2 mg/kg) **3.3 months** (2.2-6.1; 2.6 mg/kg)	**4.9 months**	**5.5 months** (4.1–7.0)	**5.6 months** (3.0–6.9)	**3.0 months** (1.7–3.9)	Not Reported
Median OS (95% CI)	**14.72 months** (6.60-NE; 2.4 mg/kg; 2+ > 20%)*	**11.8 months** (6.5-NE; 2.2 mg/kg) **11.5 months** (6.2-19.0; 2.6 mg/kg)	**10 months**	**~10.8 months**[3]	Not Evaluable	**7.5 months** (5.0–12.5)	Not Reported
Responses in IHC 2+ ≤ 20% Gastric Cancer Patients	**28.6% (6/21); Including 1 CR**	**0% ORR at RP2D in IHC 2+ <20% Patients**	**N/A**	**0% ORR in patients with CLDN18.2 expression below IHC 2+/3+ 40%**	**0% ORR in patients with CLDN18.2 expression below IHC 2+/3+ 50%**	**16.7%** (1/6; 5 - 18 mg/kg; **2+ ≤ 20%**)	**Minimal single agent activity**

[1] *Preliminary Data as of November 10, 2025;* [2] *Confirmed ORR/DCR;*

ATG-022 Demonstrated Best-In-Class Safety Profile with Potential to Transform 1L Gastric Cancer SoC in Combination with Anti-PD-1 and Chemotherapy

	ATG-022[1]	ADC 1	ADC 2	ADC 3	ADC 4	BsAb	Zolbetuximab
Molecule Design	DAR 4 vc-MMAE	DAR 4 Cleavable-MMAE	DAR 4 MMAE	TOP1i DAR 4	TOP1i DAR4	CLDN18.2 x 4-1BB Bispecific Antibody	Monoclonal Antibody
Patient Sample of Safety Data	Dose Expansion: 1.8 mg/kg Q3W (N = 31)	Dose Expansion (N = 107)	Dose Expansion: 1.8 mg/kg Q2W (N = 85)	Phase I: 6 mg/kg Q3W (N = 62)	Phase I: 6 mg/kg Q3W (N = 35)	Phase I (N = 45)	Phase IIa MONO Study (N = 54)



[1] Preliminary Data as of November 10, 2025; # TEAE is listed as statistics for TRAE is not reported

ATG-022 Demonstrated Best-In-Class Safety Profile with Potential to Transform 1L Gastric Cancer SoC in Combination with Anti-PD-1 and Chemotherapy

	ATG-022[1] (ANTENGENE)	ADC 1	ADC 2	ADC 3	ADC 4	BsAb	Zolbetuximab (astellas)
Molecule Design	DAR 4 vc-MMAE	DAR 4 Cleavable-MMAE	DAR 4 MMAE	TOP1i DAR 4	TOP1i DAR4	CLDN18.2 x 4-1BB Bispecific Antibody	Monoclonal Antibody
Patient Sample of Safety Data	Dose Expansion: 1.8 mg/kg Q3W (N = 31)	Dose Expansion (N = 107)	Dose Expansion: 1.8 mg/kg Q2W (N = 85)	Phase I: 6 mg/kg Q3W (N = 62)	Phase I: 6 mg/kg Q3W (N = 35)	Phase I (N = 45)	Phase IIa MONO Study (N = 54)

Safety	ATG-022[1] All Grades	ATG-022[1] Grade ≥ 3	ADC 1 All Grades	ADC 1 Grade ≥ 3	ADC 2 All Grades	ADC 2 Grade ≥ 3	ADC 3 All Grades	ADC 3 Grade ≥ 3	ADC 4 All Grades	ADC 4 Grade ≥ 3	BsAb All Grades	BsAb Grade ≥ 3	Zolbetuximab All Grades	Zolbetuximab Grade ≥ 3
Neutropenia	32.3%	13.8%	53.2%#	20.6%#	41.2%	14.1%	54.8%	22.6%	51.4%	20.0%	15.6%	4.4%	<10%#	NR
Nausea	25.8%	0%	57.0%#	3.7%#	27.1%	3.5%	43.5%	1.6%	65.7%	5.7%	20.0%	2.2%	63%#	15%#
Vomiting	12.9%	0%	56.1%#	10.3%#	28.2%	3.5%	27.4%	1.6%	51.4%	8.6%	11.1%	2.2%	57%#	22%#
ALT	19.4%	0%	29.0%#	0%	21.2%	1.2%	22.6%	6.5%	NR	NR	15.6%	2.2%	<10%#	NR
AST	22.6%	0%	42.1%#	0%	25.9%	1.2%	24.2%	0%	8.6%	0%	15.6%	4.4%	<10%#	NR
GGT	3.2%	0%	14.0%#	1%#	NR	NR	NR	NR	NR	NR	11.1%	2.2%	<10%#	NR
CRS	0%	0%	NR	NR	NR	NR	NR	NR	NR	NR	2%	0%	<10%#	NR
Blurry Vision	0%	0%	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR
Peripheral Neuropathy	9.7%	0%	19.6%#	0%	NR	NR	NR	NR	NR	NR	NR	NR	NR	NR

[1] Preliminary Data as of November 10, 2025; # TEAE is listed as statistics for TRAE is not reported

Antengene's Development Plans for ATG-022 (CLDN18.2 ADC)

Godfrey Guo, M.D.
Vice President, Clinical Development

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

ANTENGENE

Global

~1.6m

Prevalence

United States

~27k

Incidence

~130k

Prevalence

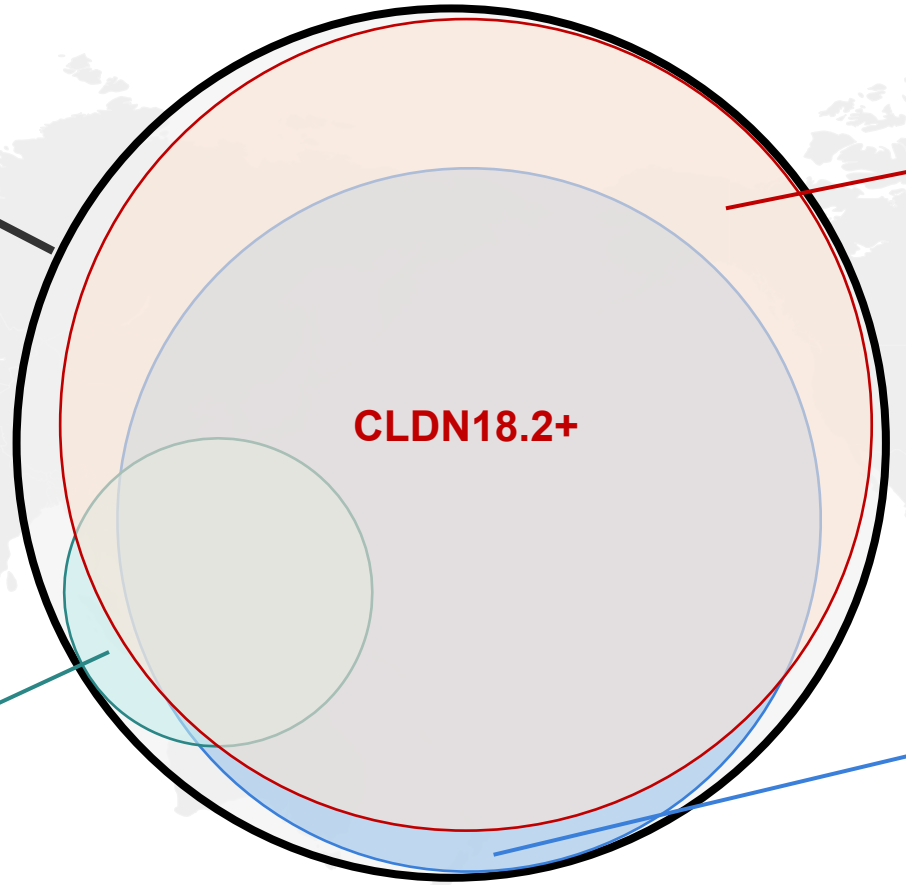
The Global Gastric Cancer Market is Underpenetrated and Presents Significant Commercial Potential for Novel Therapeutics

For Illustration

Gastric Cancer Market

$10Bn+

Total Addressable Gastric Market Size



~87%

Patients are CLDN18.2+

22% Patients are HER2+

~70% Patients are PD-L1+ (CPS ≥1)

Synergistic with ADC with MMAE payload (but not TOPO1)

*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: Improving Outcomes Where the Current Standard of Care Has Significant Room for Improvement

ANTENGENE

1L Gastric Cancer

ORR (%)

KEYNOTE-859 Study – SoC

Pembrolizumab ± Chemotherapy

CLINCH-2 Study – To Be Initiated

ATG-022 + Pembrolizumab + CAPOX

mPFS (Months)

KEYNOTE-859 Study – SoC

Pembrolizumab ± Chemotherapy

CLINCH-2 Study – To Be Initiated

ATG-022 + Pembrolizumab + CAPOX

mOS (Months)

KEYNOTE-859 Study – SoC

Pembrolizumab ± Chemotherapy

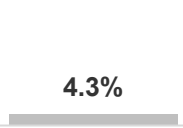
CLINCH-2 Study – To Be Initiated

ATG-022 + Pembrolizumab + CAPOX

3L+ Gastric Cancer

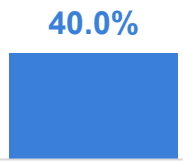
ORR (%)

JAVELIN Gastric 300 Study – SoC



Docetaxel; Paclitaxel; Irinotecan; Trifluridine + Tipiracil

CLINCH Study – Phase II



ATG-022

mPFS (Months)

JAVELIN Gastric 300 Study – SoC

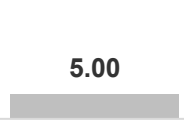
Docetaxel; Paclitaxel; Irinotecan; Trifluridine + Tipiracil

CLINCH Study – Phase II

ATG-022

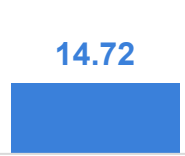
mOS (Months)

JAVELIN Gastric 300 Study – SoC



Docetaxel; Paclitaxel; Irinotecan; Trifluridine + Tipiracil

CLINCH Study – Phase II



ATG-022

Next Steps: Value Inflection Through Pivotal Phase III in 3L+ Gastric Cancer, Combo Development in 1L and Proof of Concept in Other Tumor Types

ANTENGENE

Pivotal Phase III

3L+ CLDN18.2+ Gastric Cancer

ATG-022 vs. Physician's Choice of Chemotherapy
(Irinotecan / Paclitaxel / Docetaxel)

- ✓ **HER-2 Negative** and **CLDN18.2 Moderate to High Expression (IHC 2+ > 20%)** Advanced / Metastatic Gastric Cancer with **At Least 2 Prior Lines of Therapy**
- ✓ **Primary Objectives:** OS and PFS
- ✓ **Secondary Objectives:** ORR, DCR, DOR, Safety, ADA

Phase II

1L CLDN18.2+, PD-L1+ Gastric Cancer

ATG-022 + Pembrolizumab + CAPOX

- ✓ **HER-2 Negative** and **CLDN18.2 Positive (IHC 1+ ≥ 1%)** Advanced / Metastatic Gastric Cancer with **No Prior Systemic Therapy**
- ✓ **Primary Objectives:** ORR
- ✓ **Secondary Objectives:** PFS, DOR, OS, Safety

Phase II

Other Non-Gastric Tumor Types

ATG-022 Monotherapy

- ✓ **Proof of Concept Achieved in a Certain Subtype of Gynecological Tumor:** All 7 Patients Who Have Undergone At Least One Efficacy Evaluation Demonstrated **Tumor Shrinkage**
- ✓ **20~30 Subjects in Each Tumor Type / Cohort**

ATG-022: Strong Clinical and Strategic Positioning in 1L–3L+ Gastric Cancer with Expansion Potential Across Indications – Targeting Over US$5 Billion in Peak Sales

ANTENGENE

1L CLDN18.2+ (IHC 1+ ≥ 1%), PD-L1+ (CPS ≥ 1%) Gastric Cancer

ATG-022 + Pembrolizumab + Chemotherapy (CAPOX / FOLFOX)

2L CLDN18.2+ (IHC 1+ ≥ 1%), PD-L1+ (CPS ≥ 1%) Gastric Cancer

ATG-022 + Pembrolizumab

3L+ CLDN18.2+ (IHC 2+ > 20%) Gastric Cancer

ATG-022 Monotherapy

3L+ CLDN18.2+ (IHC 2+ ≤ 20%) Gastric Cancer

ATG-022 Monotherapy

Basket Trial – Other CLDN18.2+ Tumors

Proof of Concept Achieved in a Certain Subtype of Gynecological Tumor: All 7 Patients Who Have Undergone At Least One Efficacy Evaluation Demonstrated Tumor Shrinkage

US$5+ Billion Peak Sales Potential (Not Including Potential in Other CLDN18.2+ Tumors)

Phase Ib/II study Design of ATG-022 In Combination with Pembrolizumab in Advanced/Metastatic Claudin 18.2 Positive Gastric Cancer (2L+)

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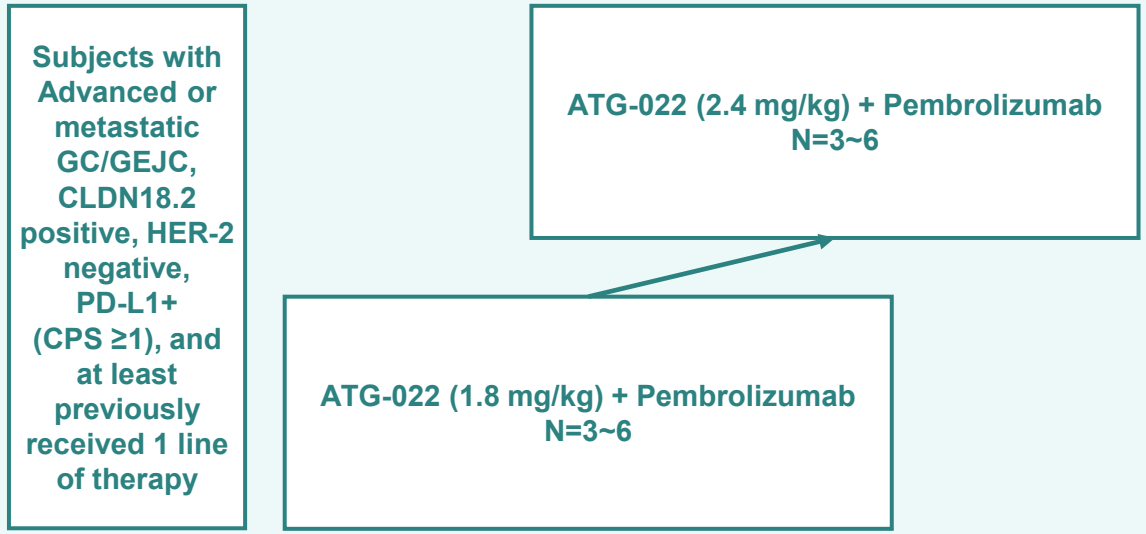
Multi-center, Open Label, Phase Ib/II Study in Advanced/Metastatic Claudin 18.2 Positive GC/GEJC

Phase Ib: Dose Confirmation



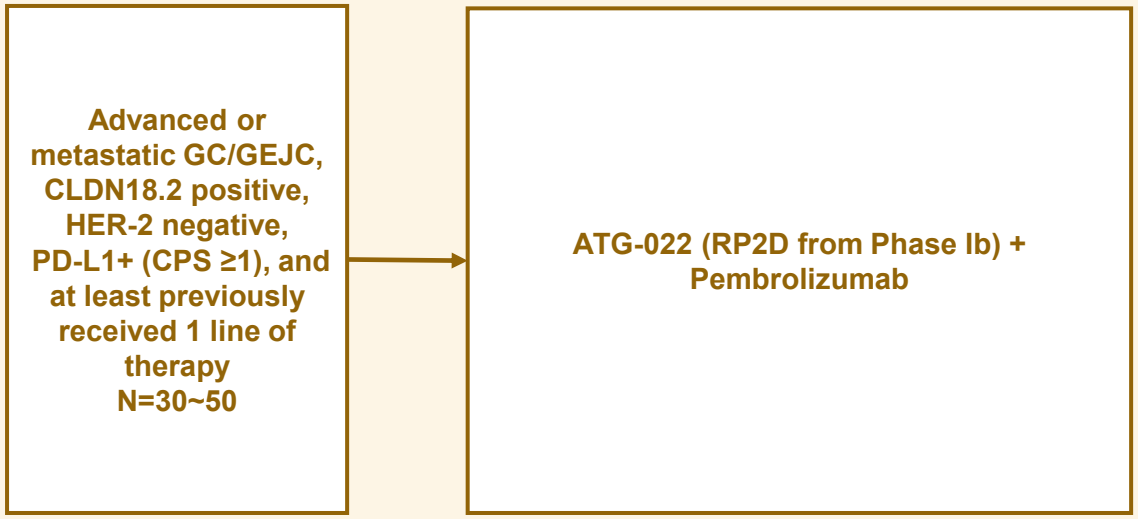
Primary Objectives:

Safety, tolerability of ATG-022 + pembrolizumab combination therapy. RP2D definition

Secondary Objectives:

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

Phase II: Efficacy Expansion



Primary Objectives:

ORR

Secondary Objectives:

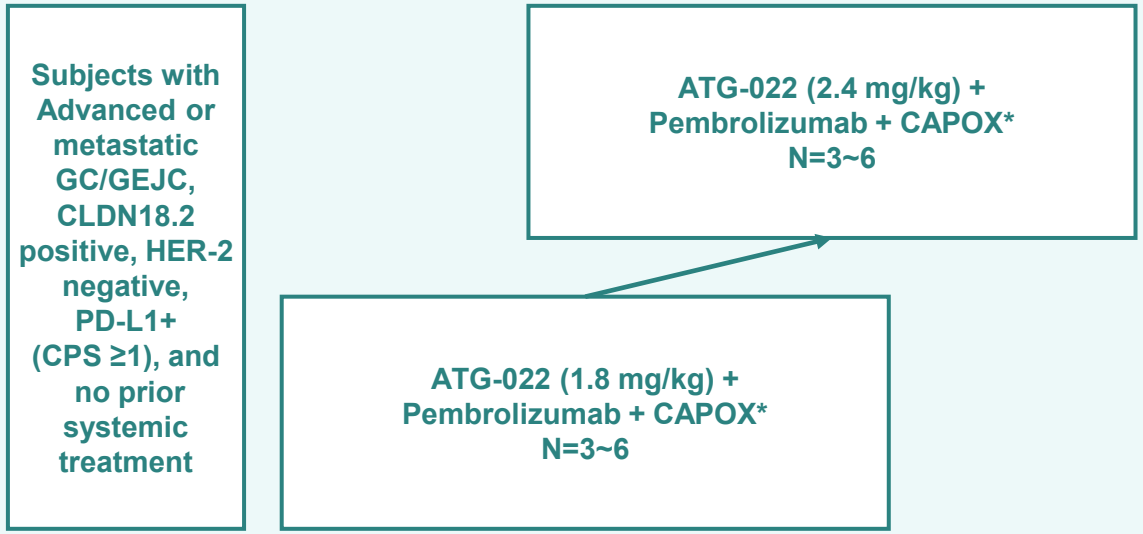
PFS, DOR, OS, Safety

Phase Ib/II study Design of ATG-022 In Combination with Pembrolizumab and CAPOX in Advanced/Metastatic Claudin 18.2 Positive Gastric Cancer (1L)

ANTENGENE

Multi-center, Open Label, Phase Ib/II Study in Advanced/Metastatic Claudin 18.2 Positive GC/GEJC

Phase Ib: Dose Confirmation



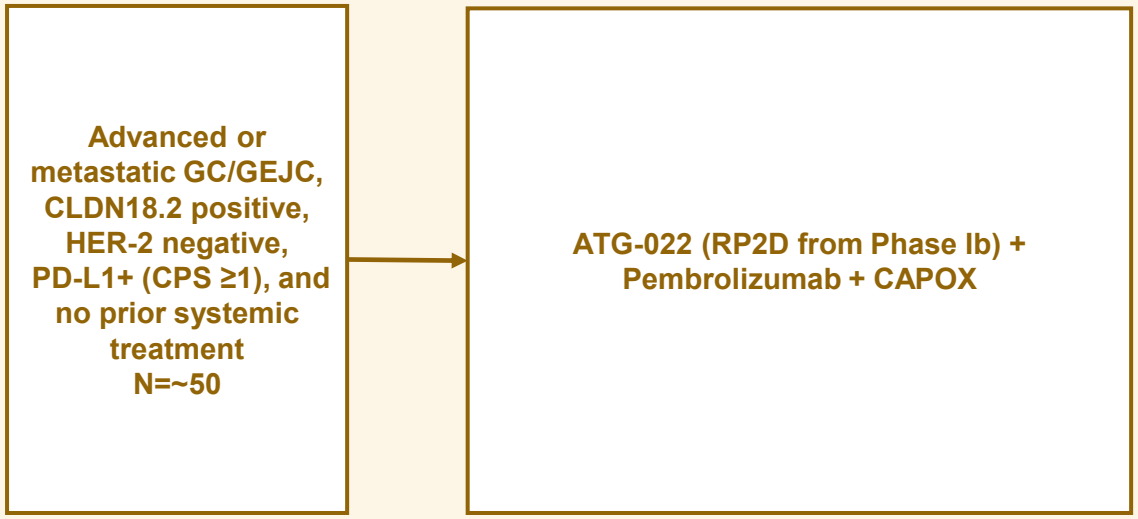
Primary Objectives:

Safety, tolerability of ATG-022 + pembrolizumab + CAPOX combination therapy. RP2D definition

Secondary Objectives:

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

Phase II: Efficacy Expansion



Primary Objectives:

ORR

Secondary Objectives:

PFS, DOR, OS, Safety

** CAPOX will be used by standard dose, or light intensity upon SRC's decision*

ATG-022 Reshapes CLDN18.2 as a Pan-Tumor Target and Delivers a Best-in-Class Safety Profile to Transform 1L Treatment in Gastric Cancer via Anti-PD-1 and Chemo Combination

ANTENGENE

Efficacy Significance

Unlocking a Pan-Tumor CLDN18.2 Opportunity

- ✓ **Meaningful activity in low expressors**, confirming expansion potential into tumors with minimal CLDN18.2 expression
- ✓ **Clear path into additional tumor types** where CLDN18.2 levels are far lower than in gastric cancer
  - PoC demonstrated in a **gynecologic tumor** subtype (tumor shrinkage observed in all evaluable patients)
- ✓ **Enables an all-comers 1L strategy** with pembrolizumab + CAPOX across **all CLDN18.2 expression levels**

Safety Significance

ATG-022 as the Only ADC Viable for 1L Combo Therapy with Chemotherapy and Anti-PD-1

- ✓ **Only 16.1% Grade ≥ 3 TRAEs**, far below competing CLDN18.2 programs (40–60%)
- ✓ **Best-in-class safety profile** supporting ATG-022 as the **only ADC** viable for 1L combination with both chemotherapy and anti–PD-1 therapy

Q&A Session

德琪医药
ANTENGENE

Clinical Program Highlights and Development Plans

ATG-037 (Oral CD73 Inhibitor) and ATG-101 (PD-L1 x 4-1BB BsAb)

Godfrey Guo, M.D.
Vice President, Clinical Development

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

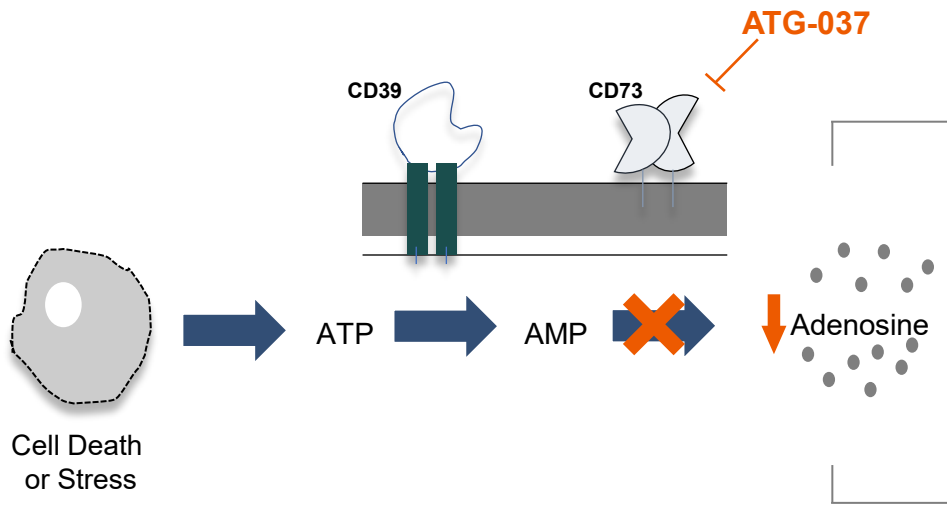
ANTENGENE

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

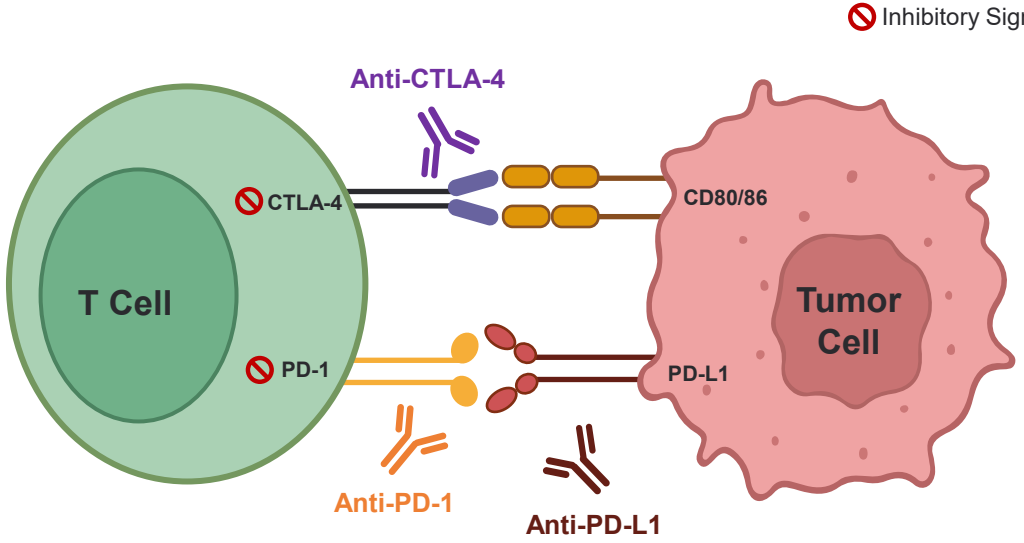
Induces and Enhances the function of Immunosuppressive Cell Types: Tregs, MDSCs, TAMs and Tolerogenic DCs

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

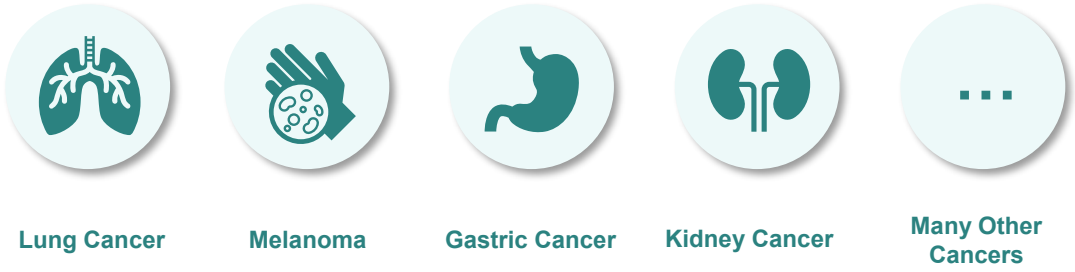
Immune Checkpoint Inhibitor Market: Rapid Growth Across Multiple Cancers

ANTENGENE

Immune Checkpoint Inhibitors (ICIs) Targets



Broad Applicability of ICIs in Oncology Treatment



Global ICIs Market Size Expected to Reach $150+ Billion by 2030

3 Million+

cancer patients globally with exposure to Immuno-oncology treatment

514,000+

cancer patients in the US with exposure to Immuno-oncology treatment

Source: GLOBALCAN, Global Data, Research and Markets, publications & primary research;

ATG-037 Can Address the Huge Unmet Medical Need of Melanoma Patients who Progress on Immune Checkpoint Inhibitors

ANTENGENE

Immune Checkpoint Inhibitors (ICIs) are Standard of Care Therapies of Advanced Melanoma (Unresectable)

Checkpoint Inhibitors **Standard of Care for Most Patients**	✓ **Anti-PD-1** (pembrolizumab / nivolumab) ✓ **Anti-CTLA-4** (ipilimumab) ✓ **Combination** of Anti-PD-1 and Anti-CTLA-4 / Anti-LAG-3
Targeted Therapies **Standard of Care Only for BRAF V600-Mutant Melanoma**	✓ **BRAF/MEK Inhibitors** (dabrafenib + trametinib / vemurafenib + cobimetinib / encorafenib + binimetinib)
Other Therapies **Limited Usage**	✓ Oncolytic Virus (Talimogene laherparepvec) ✓ High dose Interleukin-2 (rarely used today)

Significant Medical Needs of Melanoma in the US Especially in Patients Who Progress on ICIs

104,960
New Cases of Melanoma Per Year

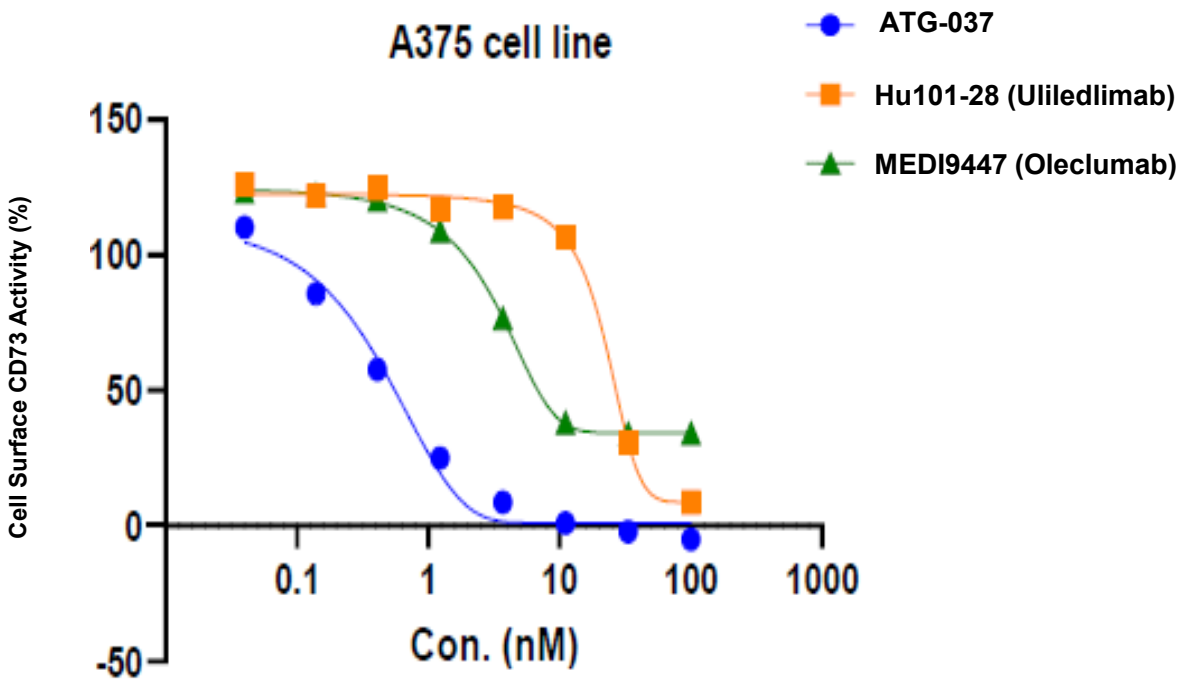
1,504,676
US Patients Living with Melanoma

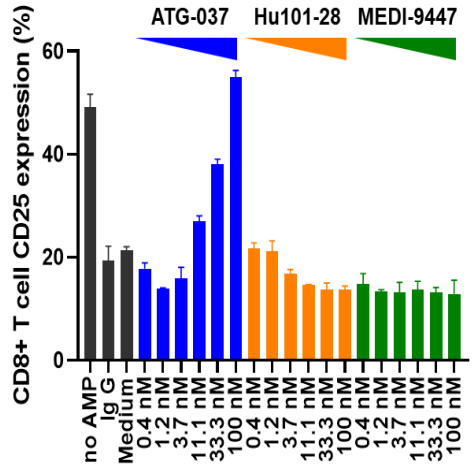
#5 Cancer in the US
(By Incidence)

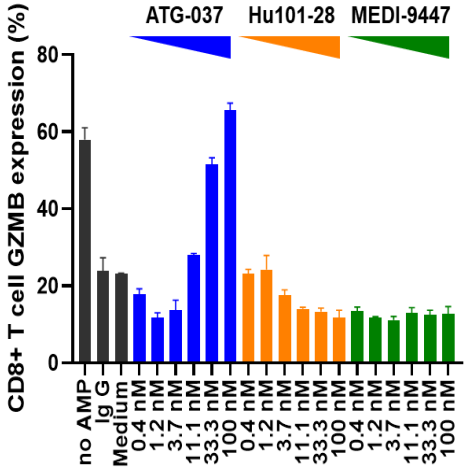
ATG-037 Showed Strong CD73 Inhibition and Activity in Reversing T Cell Inhibition

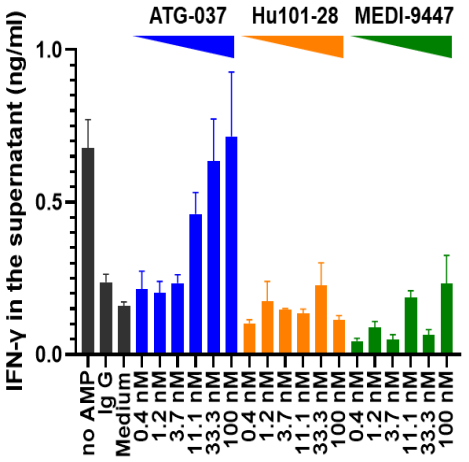
ANTENGENE

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









ATG-037 "STAMINA" Clinical Trial Design

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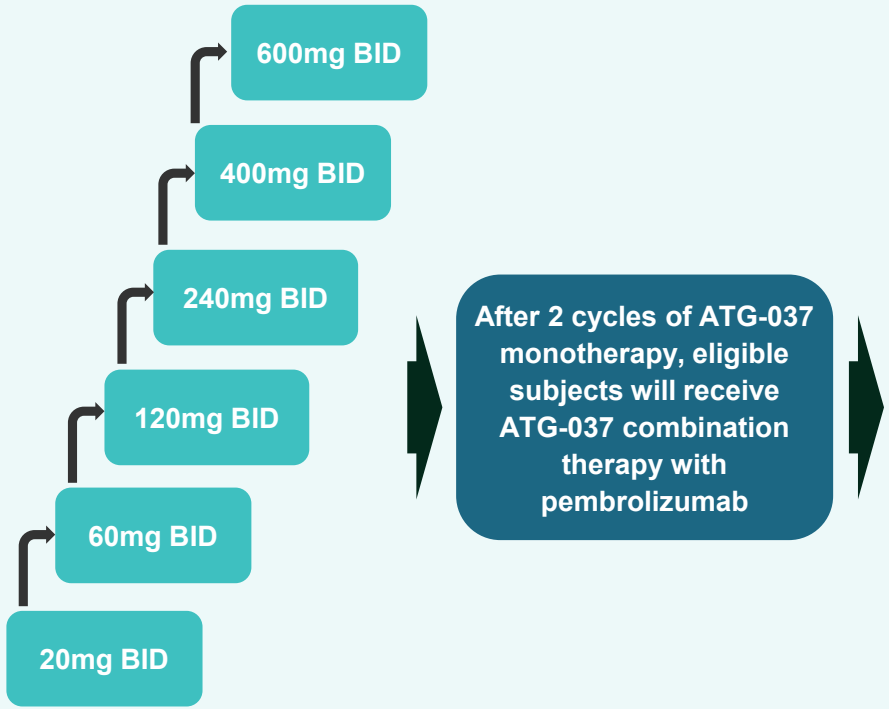
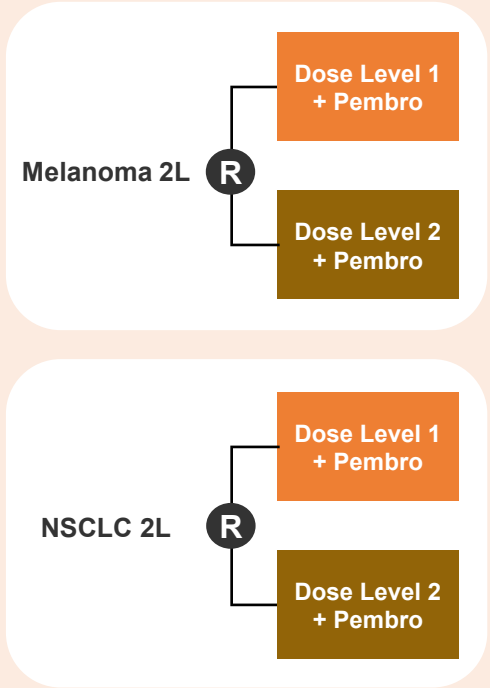
Phase I/II, Multi-center, Open Label, Dose-finding Study Ongoing in Australia and China (NCT05205109)

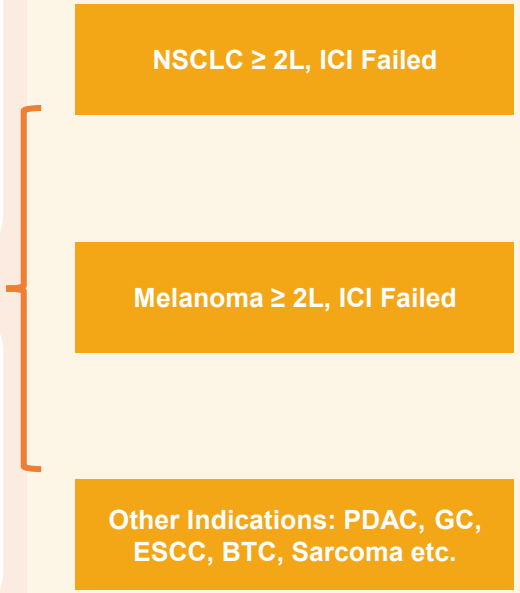
Dose Escalation



Dose Optimization



Dose Expansion



Objectives of the Study

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

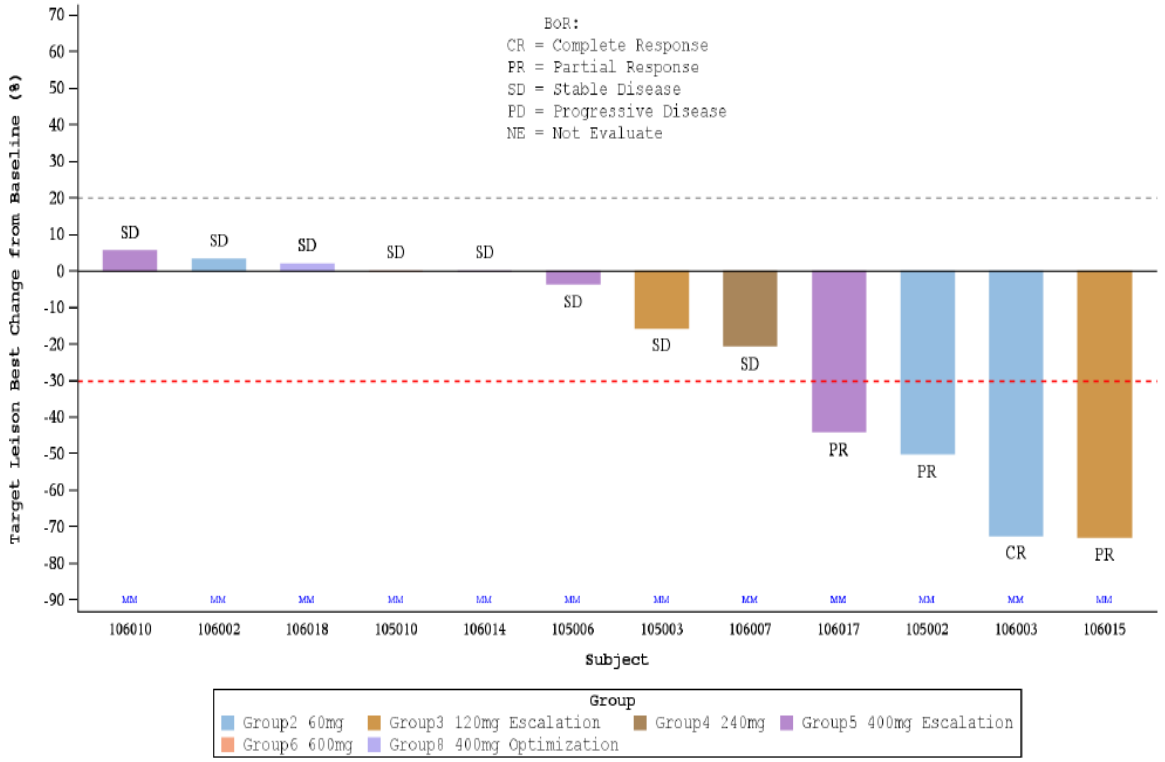
Secondary Objectives:
Evaluate preliminary efficacy, characterize pharmacology (PK/PDx profile)

ATG-037 In Combination with Pembrolizumab Demonstrated Encouraging Efficacy Signals in CPI Resistant Melanoma

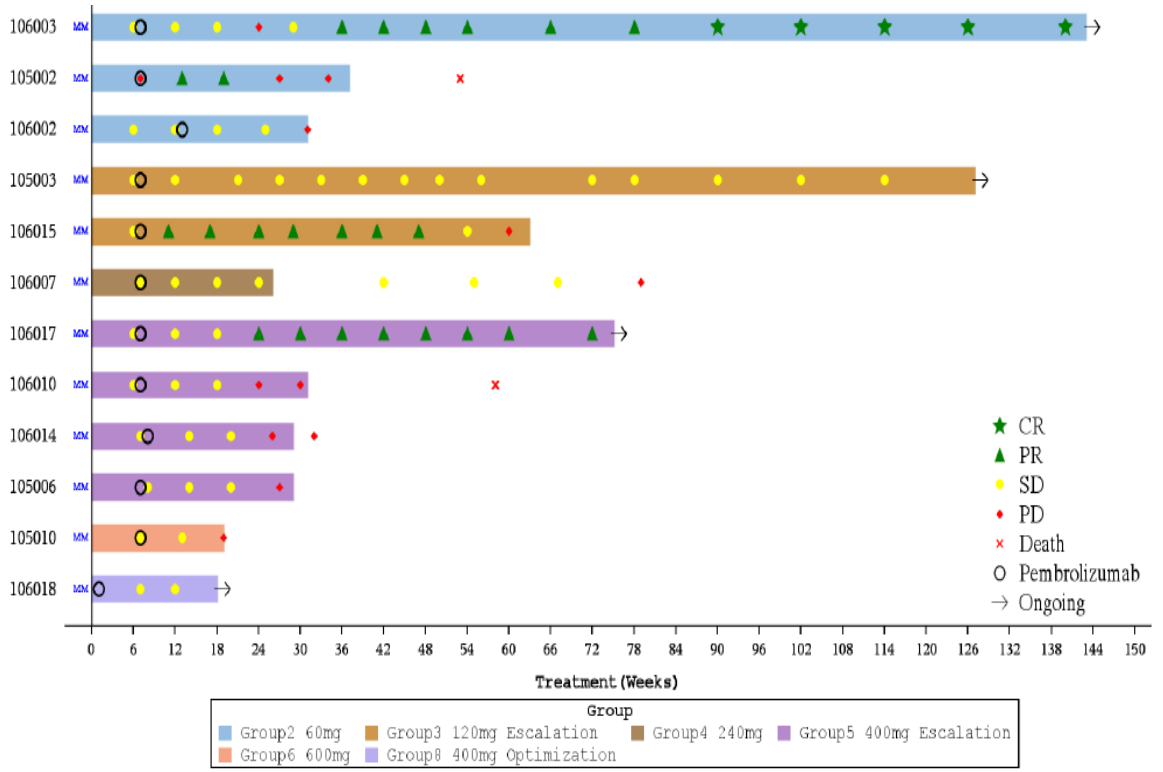
Preliminary Data for ATG-037 In Combination with Pembrolizumab (As of October 24, 2025)

- A total of **12 CPI-resistant melanoma** patients received the combination therapy and were efficacy evaluable
  - **1 confirmed CR** and **3 confirmed PRs**, with the rest achieving **SD** – **ORR 33.3%** (4/12) and **DCR 100%** (12/12)
  - **Durable benefit observed:** the **CR patient** remains on therapy with **over 34-month ongoing response** and without safety concern

CPI-resistant Melanoma – Waterfall Plot



CPI-resistant Melanoma – Swimmer Plot



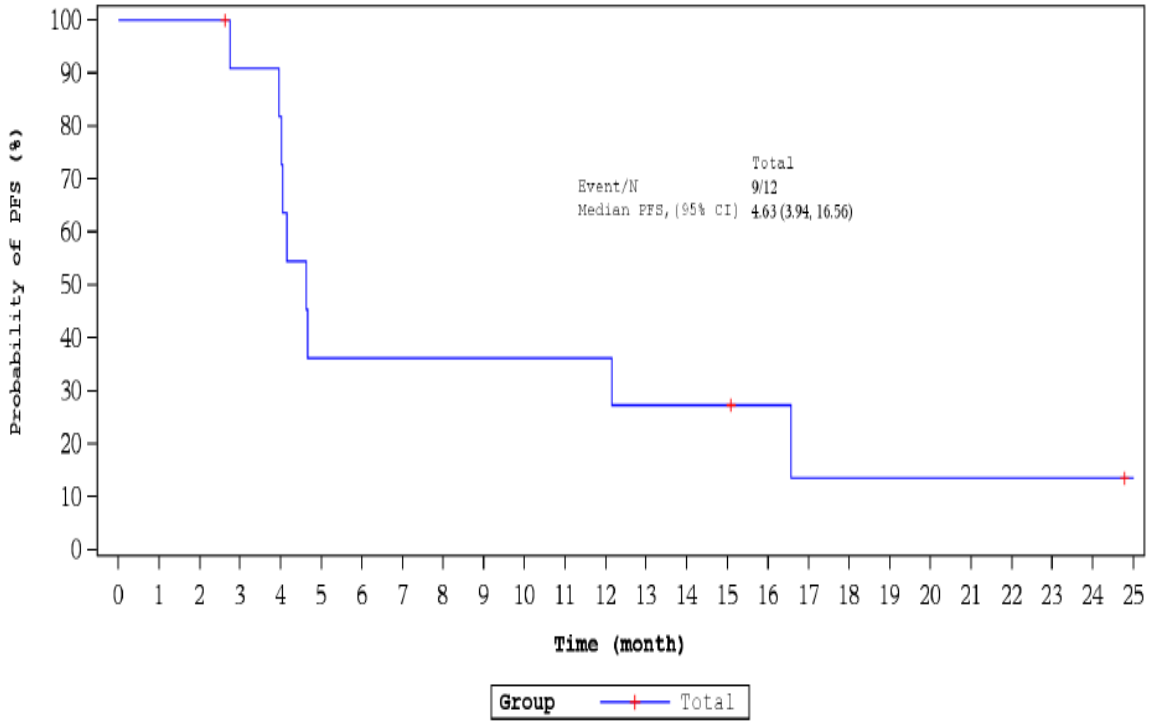
ATG-037 In Combination with Pembrolizumab Showed Promising Survival Benefits in CPI Resistant Melanoma

ANTENGENE

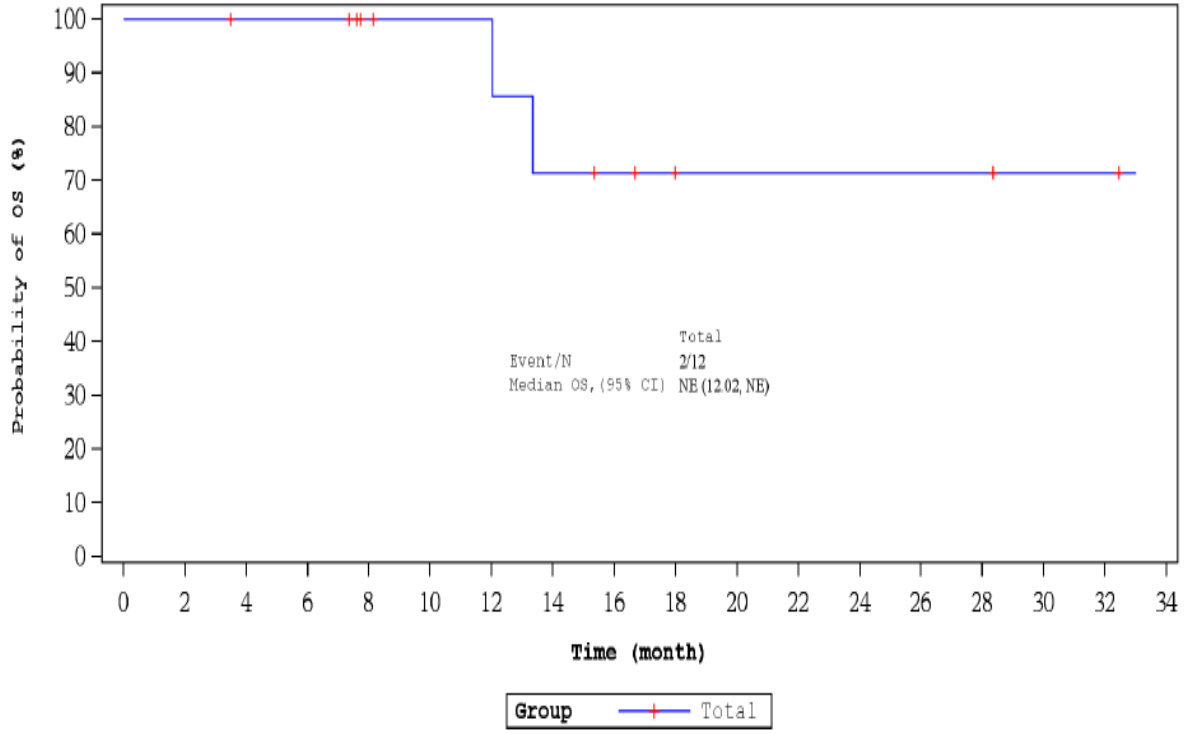
Preliminary Data for ATG-037 In Combination with Pembrolizumab (As of October 24, 2025)

- The **median PFS is 4.63 months** (95% CI: 3.95-16.56)
- The **median OS has not been reached**

CPI-resistant Melanoma – Progression Free Survival Kaplan-Meier Curve



CPI-resistant Melanoma – Overall Survival Kaplan-Meier Curve



ATG-037 + Pembrolizumab: Case Study of 73 y/o Female Melanoma Patient Achieving Confirmed Partial Response

ANTENGENE

Summary of Patient

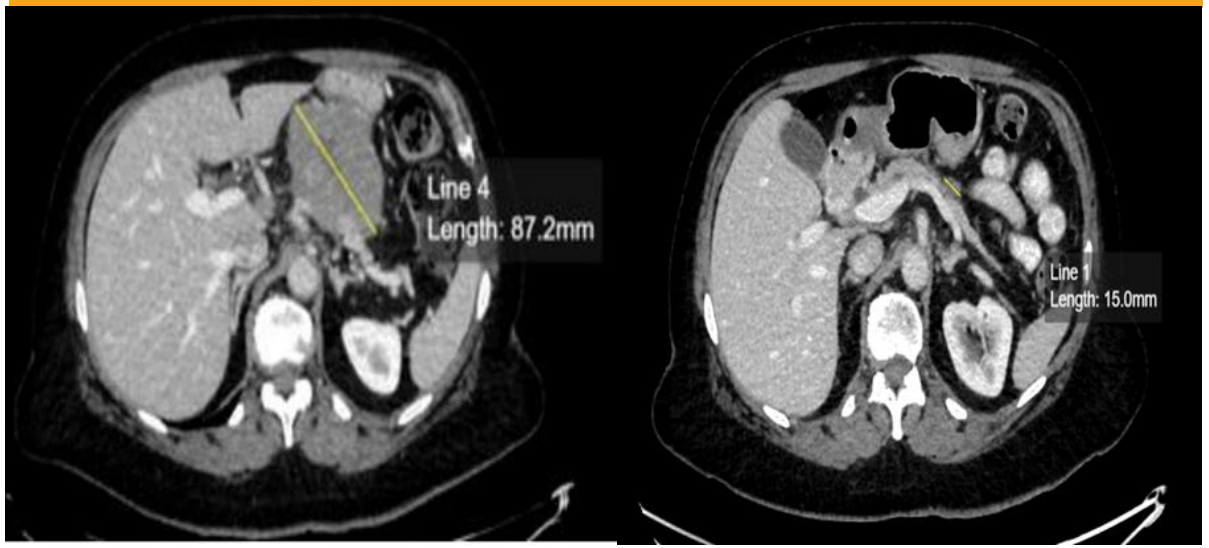
Patient	73 y/o, Female, Metastatic Melanoma (mucosal)
Initial Diagnosis	May 3rd , 2017; T4N0Mx
ECOG PS	1
PD-L1/CD73 Expression	PD-L1 (22C3): TPS NA; ICs NA; CD73: CD73+ Tumor cell NA; TAICs NA
Target Lesion	Pancreas 87mm
STAMINA Study Treatment	ATG-037 120 mg BID, C1D1 - May 2nd, 2024; Pembrolizumab 200 mg Q3W, dosed from Cycle 3 (Jun 13th, 2024)

Prior Systemic Anti-Cancer Therapy

Regimen	Start Date	End Date	Best Response	Discontinue Reason
Nivolumab + RELATLIMAB/ PLACEBO	Oct 15th, 2020	Jun 24, 2022	NA	Disease progression
NEMVALEUKIN	Nov 14th, 2021	Feb 10, 2023	Stable Disease	Withdrew

2025 ASCO Poster#3123

Tumor Evaluation – Target Lesion Change from Baseline



Baseline | Week 52

Safety Profile

TEAE	NCI CTCAE Grade	Relationship to ATG-037	Action Taken with ATG-037
Rash	Grade 1	Related	Dose Not Changed
Postural hypotension	Grade 1	Unrelated	Dose Not Changed
Bronchitis	Grade 2	Unrelated	Dose Not Changed
Right arm thrombophlebitis	Grade 2	Unrelated	Dose Not Changed
Cerebrovascular ischaemia	Grade 1	Unrelated	Dose Not Changed

ATG-037 In Combination with Pembrolizumab Demonstrated Encouraging Efficacy Signals in CPI Resistant Non-small Cell Lung Cancer – Waterfall Plot

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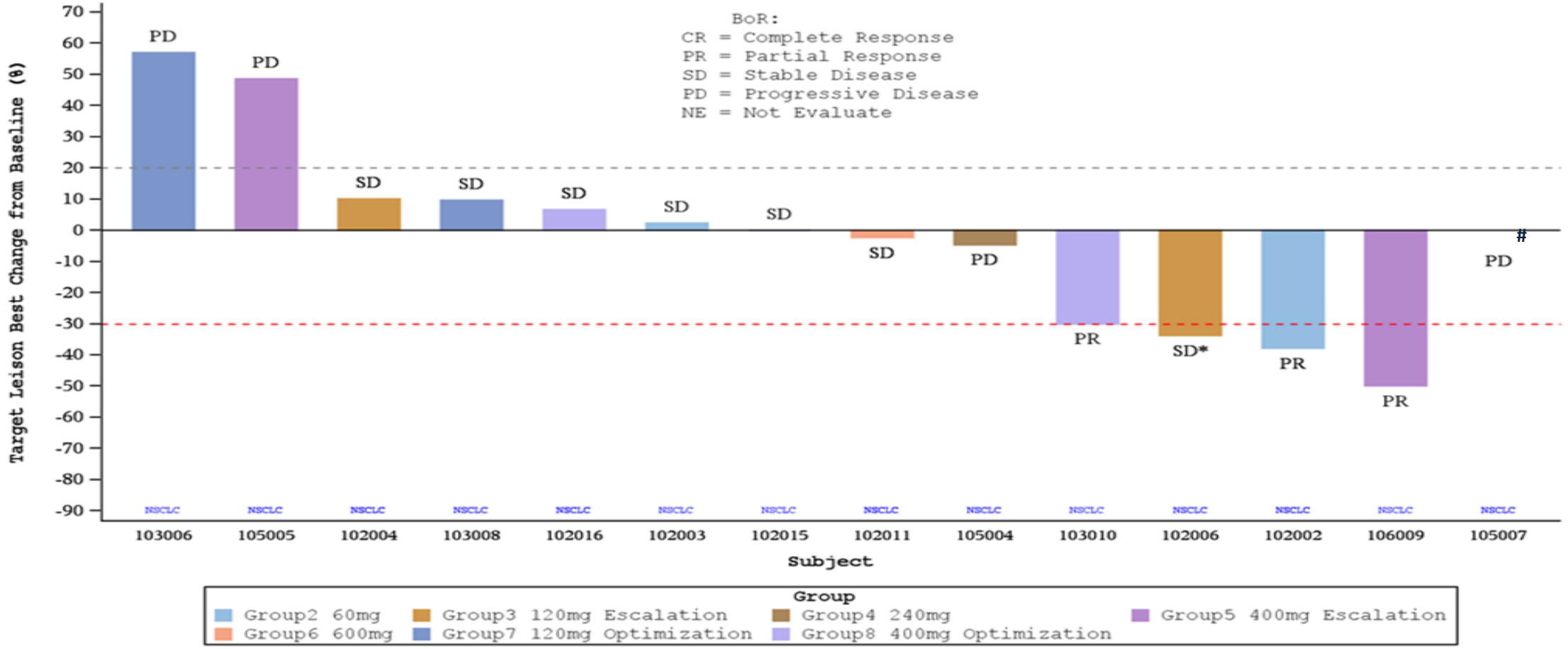
Preliminary Data for ATG-037 In Combination with Pembrolizumab (As of October 24, 2025)

- A total of **14 CPI-resistant non-small cell lung cancer** patients received the combination therapy and were efficacy evaluable
  - **3 PRs** and **7 SDs** – **ORR 21.4%** (3/14) and **DCR 71.4%** (10/14)



*The target lesion of this subject reached PR with new lesion occurred. The prior best response was SD. #The target lesion was not evaluated with new lesion occurred

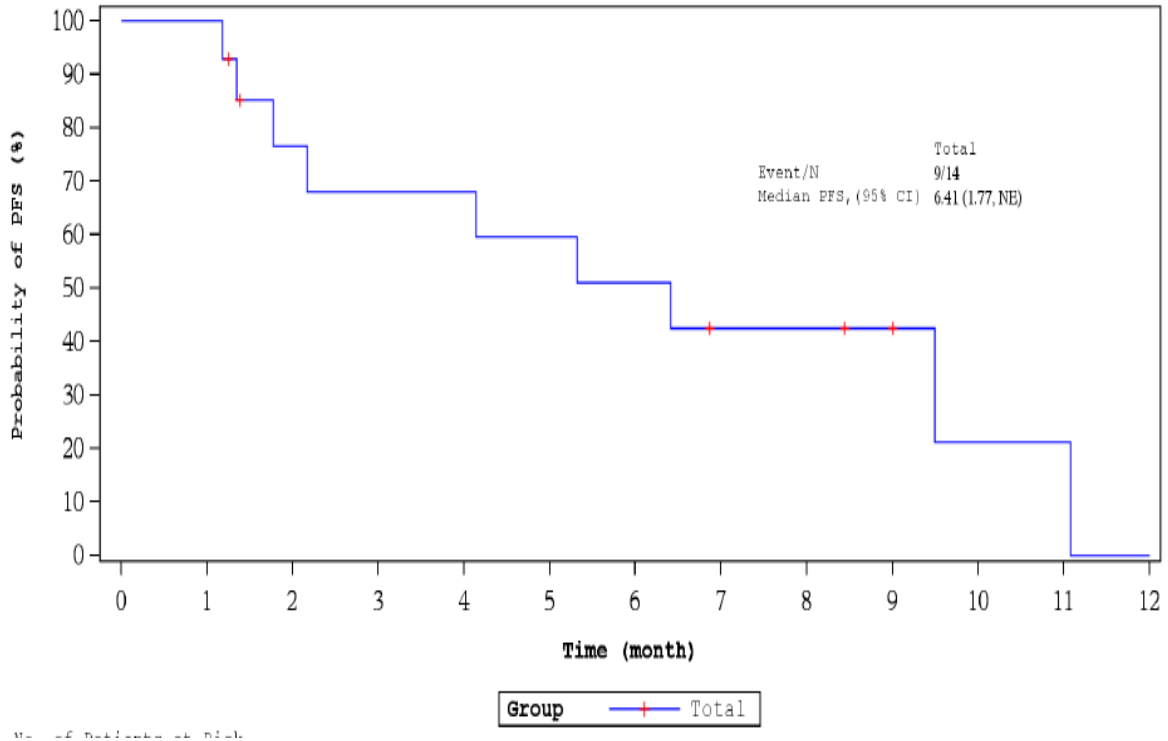
ATG-037 In Combination with Pembrolizumab Showed Promising Survival Benefits in CPI Resistant Non-small Cell Lung Cancer (NSCLC)

ANTENGENE

Preliminary Data for ATG-037 In Combination with Pembrolizumab (As of October 24, 2025)

- The **median PFS is 6.41 months** (95% CI: 1.77-NE)
- The **median OS is 15.05 months** (95% CI: 8.02-NE)

CPI-resistant NSCLC – Progression Free Survival Kaplan-Meier Curve



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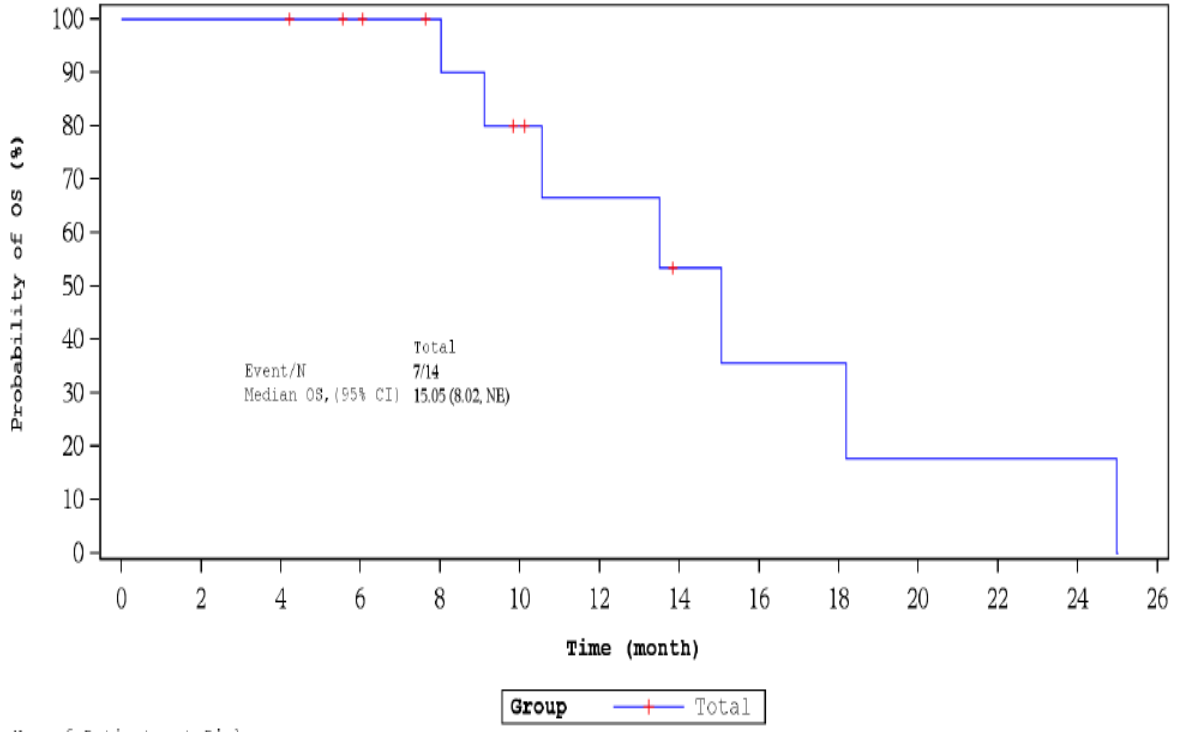

ATG-037 In Combination with Pembrolizumab: Excellent Safety Profile

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Preliminary Data for ATG-037 In Combination with Pembrolizumab (As of October 24, 2025)

- While on combination therapy, 23/38 (60.5%) patients reported TRAEs
- The **majority of TRAEs were grades 1-2**; 7.9% (3/38) were reported as grade 3. **No grade 4 or 5 TRAEs**
- Only 2 serious TRAEs (grade 3 diarrhea; grade 3 immune mediated hepatitis) were reported

TRAEs of Combination Therapy Treated Population

n (%)	N=38
Subjects with at least one TRAE	23 (60.5)
Serious TRAE	2 (5.3)
Grade ≥ 3 TRAE	3 (7.9)
TRAE Leading to Dose Modification	8 (21.1)
TRAE Leading to Dose Reduction	0 (0)
TRAE Leading to Dose Interruption	8 (21.1)
TRAE Leading to Drug Withdrawn	3 (7.9)
TRAE Leading to Death	0 (0)

ATG-037: Strong Clinical and Strategic Positioning in CPI-Resistant and 1L Melanoma with Expansion Potential in Other Tumors

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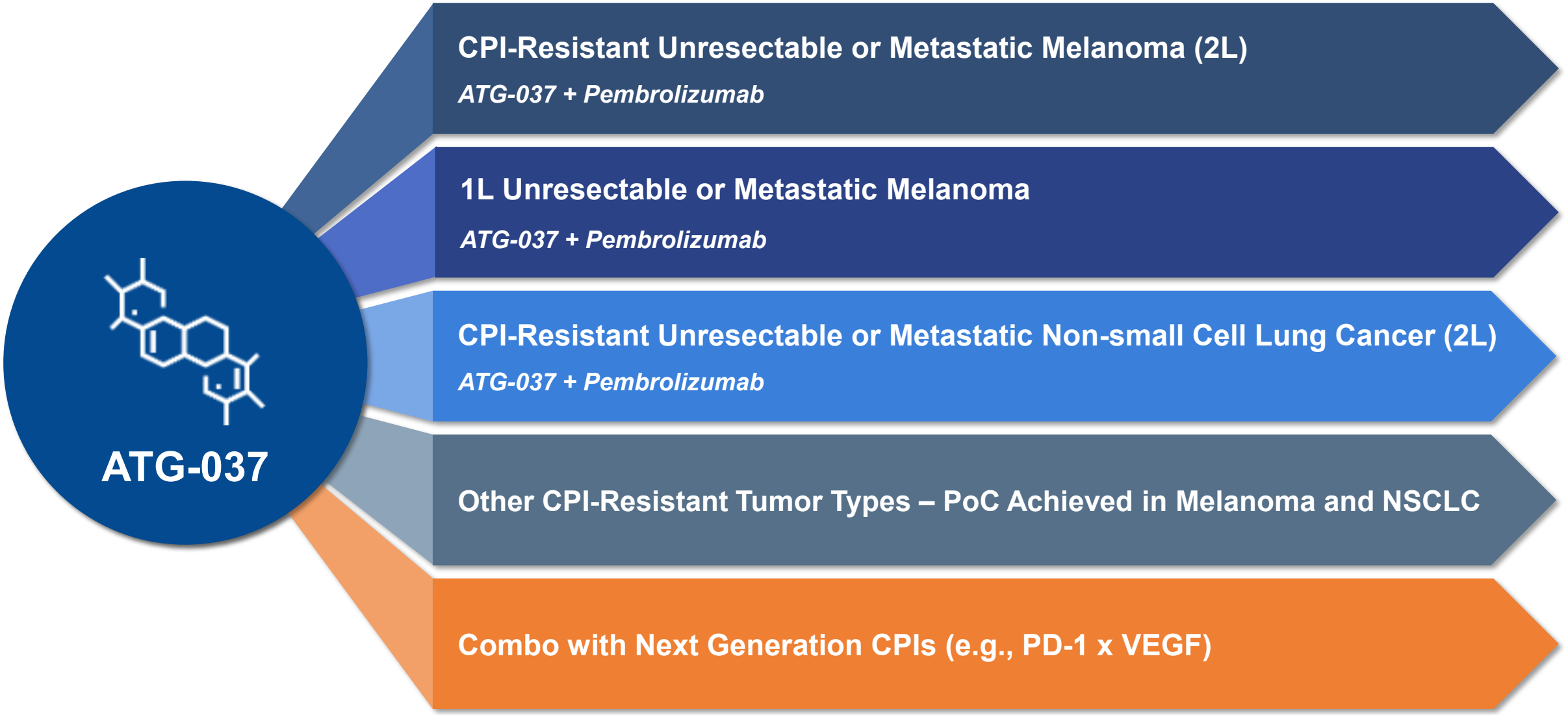

ATG-101 (Xirestomig): Potentially Best-in-class PD-L1 x 4-1BB Bispecific Antibody with PD-L1-Gating Offers Better Safety and Potential to Overcome PD-(L)1 Resistance

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How can ATG-101 Overcome PD-(L)1 Resistance?

Add a T Cell Booster

By combining with a 4-1BB agonist

Creating an "On-switch"

By using a bi-specific antibody to create a "trimer-induced-on-switch" to reduce 4-1BB driven liver tox

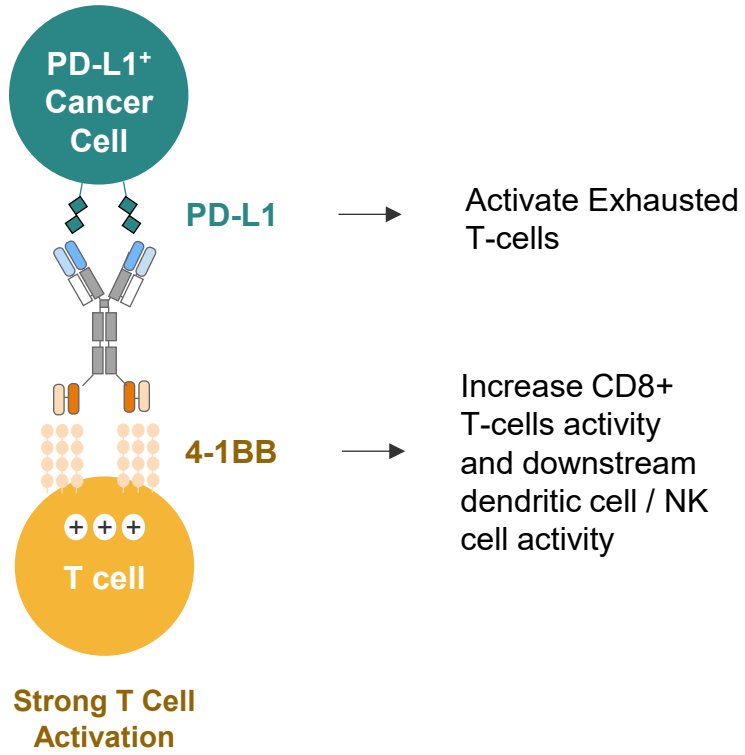
Maximize PD-L1 Binding

ATG-101's PD-L1/4-1BB arm affinity ratio of 65 ensures high PD-L1 receptor occupancy

To Render Tumors "Hot"

By increase CD8+ T-cell activity and downstream dendritic cell and NK cell activity

Complementary Mechanism of PD-L1 x 4-1BB to render "Cold" tumors "Hot"



CPI = checkpoint inhibitor; mAb = monoclonal antibody, NK cell = Natural Killer cell

ATG-101: Targeting “Cold”, “Hot”, and CPI-Resistant / Relapsed Tumors with Strong Combination Potential

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Tumor Targeting Strategy

- ✓ Focus on **“cold” tumor types** including **extrapulmonary neuroendocrine carcinoma (EP-NEC)**, with a study initiation planned soon
- ✓ Explore the potential of **immunogenic (“hot”) tumor types** such as **NSCLC and melanoma**
- ✓ Target patient population who have received **prior anti-PD-1 therapy** and subsequently **developed resistance or relapsed**

Developed In Combo with Other Anti-tumor Therapies

- ✓ Ongoing trials in Australia (Q3W), the US (Q3W), and China (Q2W / Q4W) consistently show a **favourable safety profile**
- ✓ ATG-101 demonstrates superior safety profile vs. competitor programs, with **no liver toxicity observed**, supporting its use as a **strong combination partner**

Break

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Discovery and Pre-clinical Highlights

Bing Hou, Ph.D.
Vice President, Head of Discovery Science & Translational Medicine

Discovery and Pre-clinical Highlights

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Antibody Drug Conjugates (ADCs)

ATG-022 (CLDN18.2) *Phase II*	CLDN18.2+ Gastric Cancer (GC) and Other Solid Tumors	CLDN18.2 ADC with Efficacy Across the Widest Patient Population; BTD in GC
ATG-125 (B7-H3 x PD-L1) *Pre-clinical*	Solid Tumors	IO+ADC in One Drug
CD24 *Pre-clinical*	Solid Tumors	IO+ADC in One Drug

Immuno-Oncology (IO)

ATG-037 (CD73) *Phase Ib/II*	CPI-resistant Melanoma and Non-small Cell Lung Cancer	Oral Bioavailable; Demonstrated Efficacy in CPI-resistant Patients
ATG-101 (PD-L1 x 4-1BB) *Phase I*	Solid Tumors	No Liver Toxicity
ATG-031 (CD24) *Phase I*	Solid Tumors	First-in-class Myeloid Regulator

Autoimmune Diseases

ATG-201 (CD19 x CD3) *IND-enabling*	B Cell Driven Autoimmune Diseases	Deep B Cell Depletion with Low CRS
ATG-207 (Undisclosed Bifunctional Biologics) *Discovery*	T Cell Driven Autoimmune Diseases	First-in-Class; Induces T_{reg} and T Cell Exhaustion

T Cell Engagers (TCEs)

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ATG-102 (LILRB4 x CD3) *Pre-clinical*	Acute Myeloid Leukemia and Chronic Myelomonocytic Leukemia	Biparatopic
ATG-107 (FLT3 x CD3) *Pre-clinical*	Acute Myeloid Leukemia	
ATG-115 (Undisclosed Bispecific TCE) *Pre-clinical*	Liver Cancer	Novel TAA Discovered by AI
Undisclosed Trispecific TCE *Discovery*	Metastatic Castration-resistant Prostate Cancer	First-in-Class
Undisclosed Trispecific TCE *Discovery*	Small Cell Lung Cancer and Neuroendocrine Tumors	First-in-Class

Discovery and Pre-clinical Highlights

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ADC + IO Combinations: Shaping the Future of Cancer Therapy

Growing Adoption and Proven Efficacy Highlight Their Transformative Potential and Set the Stage for the Development of Next-generation Assets

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Growing Adoption of ADC + IO Combinations

Mechanistic Synergy

ADCs deliver targeted cytotoxicity, while IO amplifies immune activation for stronger anti-tumor effects

Overcoming Resistance and Tumor Heterogeneity

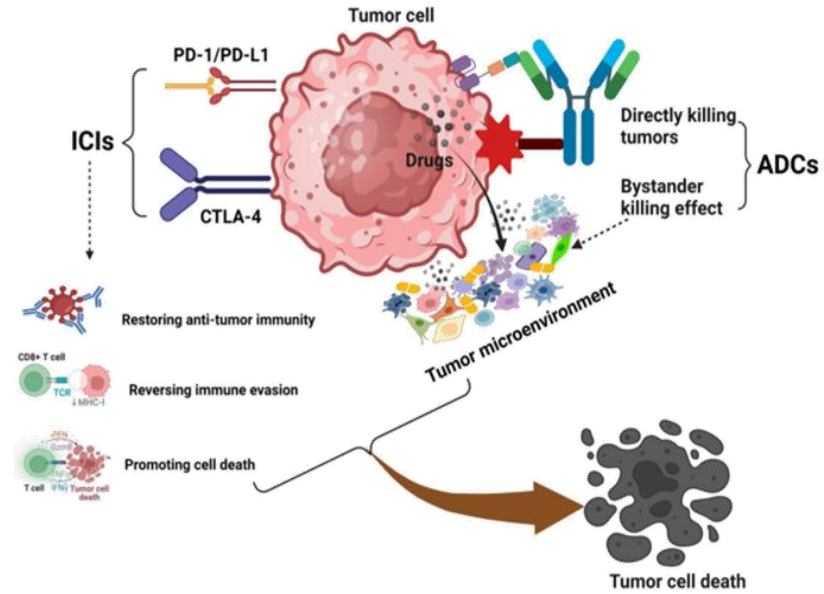
Combination converts “cold” tumors to “hot” and helps bypass resistance to either therapy alone

Durable Immune Memory and Durable Responses

IO sustains and extends ADC-driven tumor shrinkage by enabling long-term immune surveillance

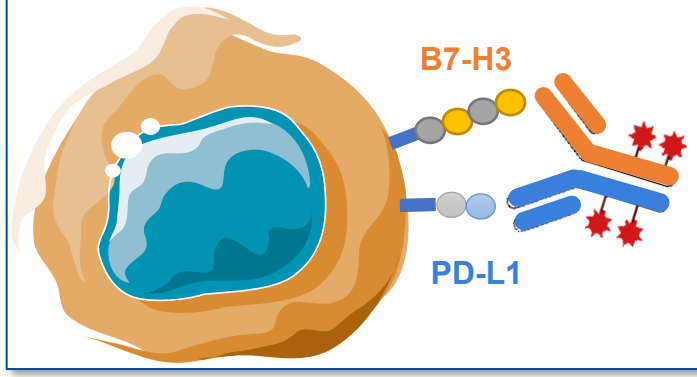
Increasing Clinical and Regulatory Momentum

Rising trial activity and early successes are driving rapid adoption and investment in ADC+IO combos



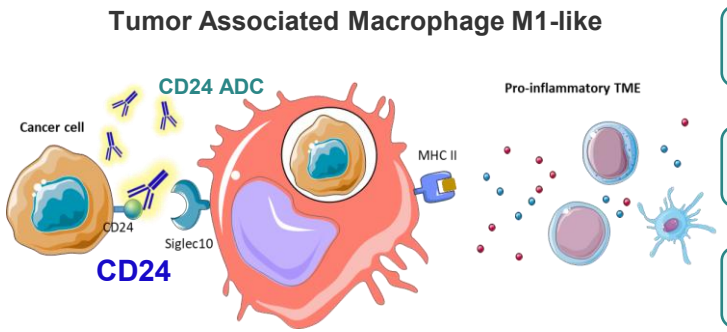
Source: Yu, P., Zhu, C., You, X. et al. The combination of immune checkpoint inhibitors and antibody-drug conjugates in the treatment of urogenital tumors: a review insights from phase 2 and 3 studies. Cell Death Dis 15, 433 (2024). https://doi.org/10.1038/s41419-024-06837-w

B7-H3 x PD-L1 Bispecific ADC – Preclinical Stage



- Dual Checkpoint Blockade
- T Cell Activation
- Direct Tumor Killing

CD24 ADC – Preclinical Stage



- Myeloid Checkpoint Blockade
- Phagocytosis Induction
- Direct Tumor Killing

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ATG-125

B7-H3 x PD-L1 Bispecific ADC

ATG-125: A Novel B7H3 x PD-L1 Bispecific ADC

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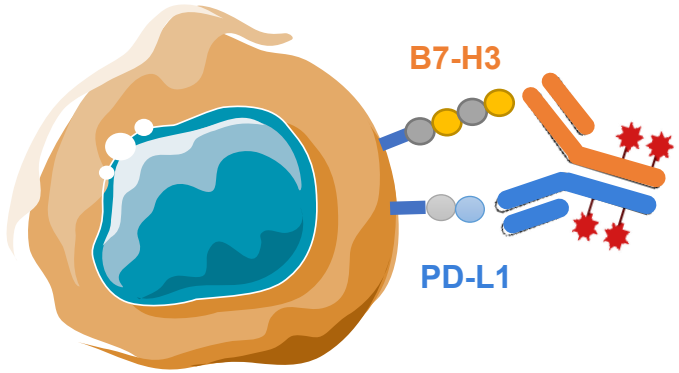
B7-H3 x PD-L1 Bispecific ADC



Dual Checkpoint Blockade

T Cell Activation

Direct Tumor Killing

- Designed to inhibit the interaction of B7-H3 and PD-L1 to their receptors, respectively, **inducing anti-tumor immunity**
- Payload induces **direct tumor killing**
- B7H3 x PD-L1 bispecific antibody demonstrated **potent *in vivo* efficacy and immunological memory**
- ATG-125 demonstrated **enhanced in vivo efficacy compared to B7H3-ADC or PDL1-ADC**
- The IND is planned for **Q1 2027**

B7-H3 is a TAA Over-expressed in Multiple Tumor Types with Immunosuppressive Function

Bispecific Antibody Component of ATG-125 Enhances T Cell Activation and Blocks PD-1 / PD-L1 Interaction

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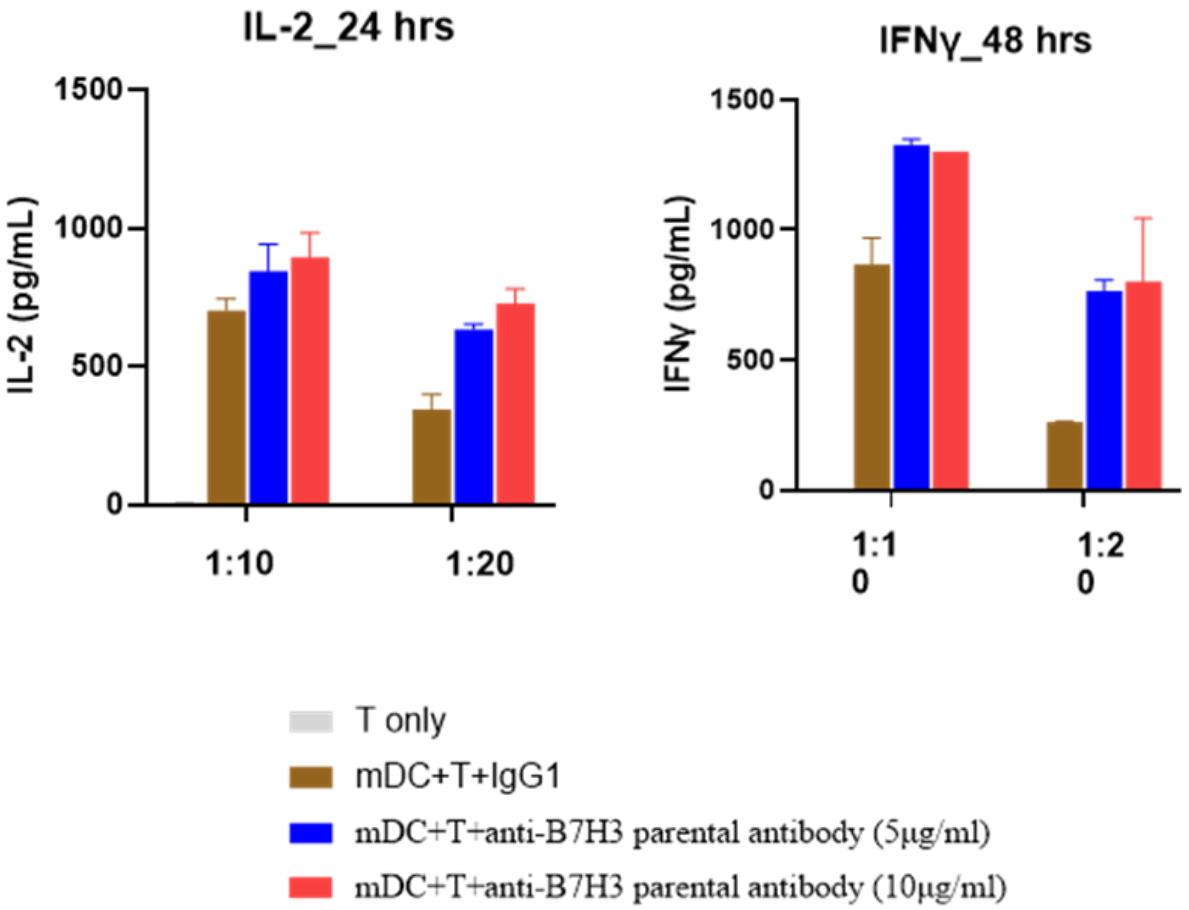
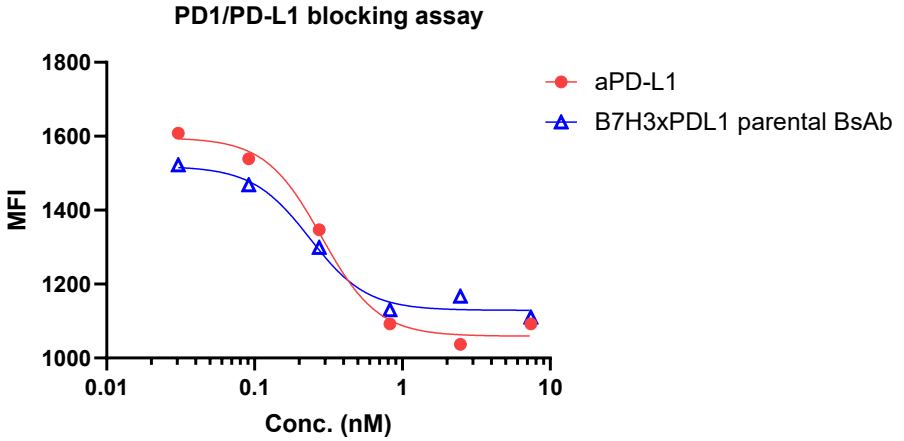
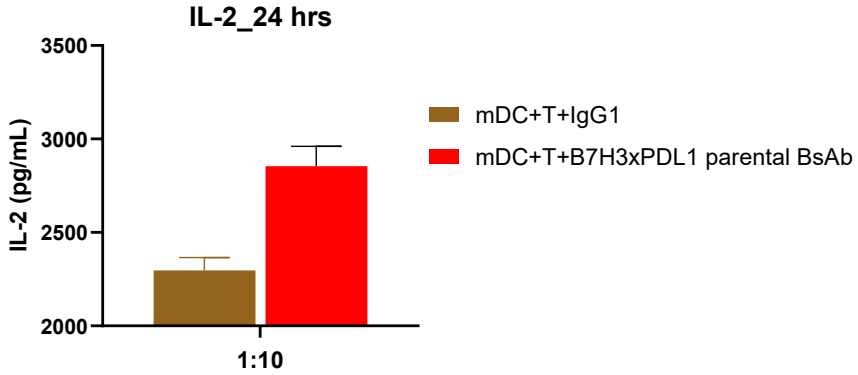
ATG-125 Parental B7-H3 Antibody Induced Potent IL-2 and IFNγ Production



Bispecific Antibody of ATG-125 Blocks PD1 / PD-L1 Interaction



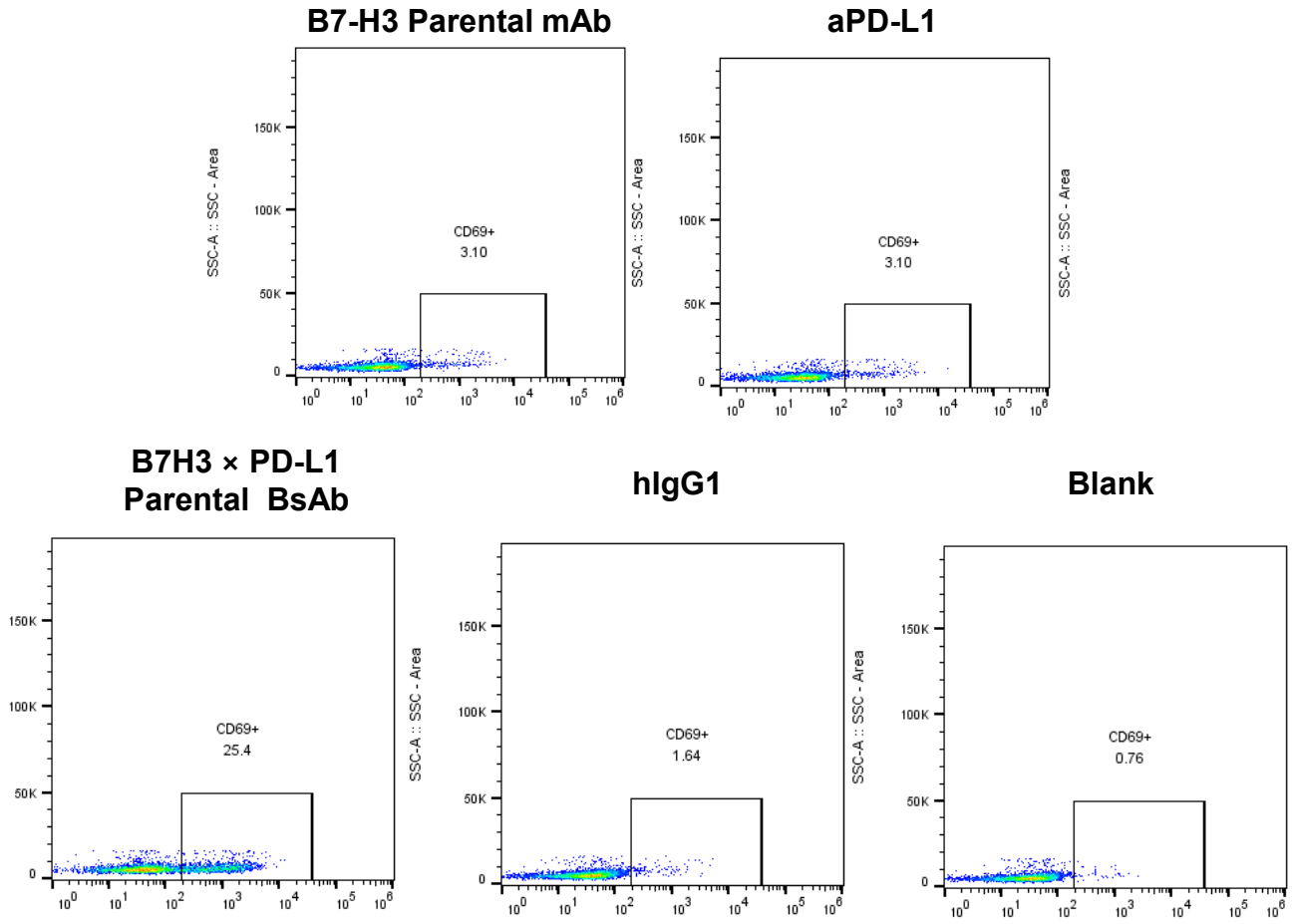
Bispecific Antibody of ATG-125 Strongly Enhanced T Cell Activation and Induced Robust IL-2 Production

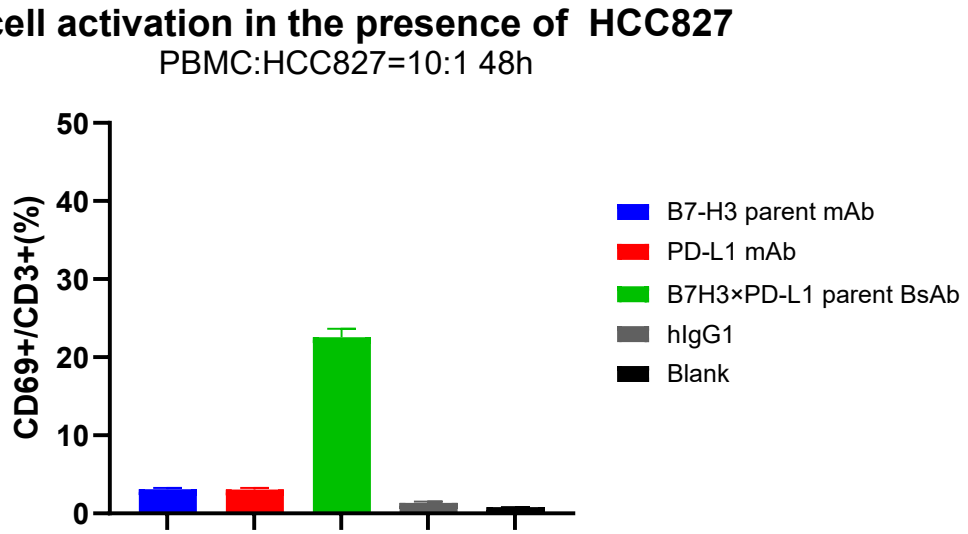


Bispecific Antibody Component of ATG-125 Activates Immune Cytotoxicity

ANTENGENE

- Bispecific antibody of ATG-125 induced **significantly enhanced T-cell activation**, as shown by a higher frequency of CD69+ CD3+ T cells in a co-culture with HCC827 cells and human PBMCs





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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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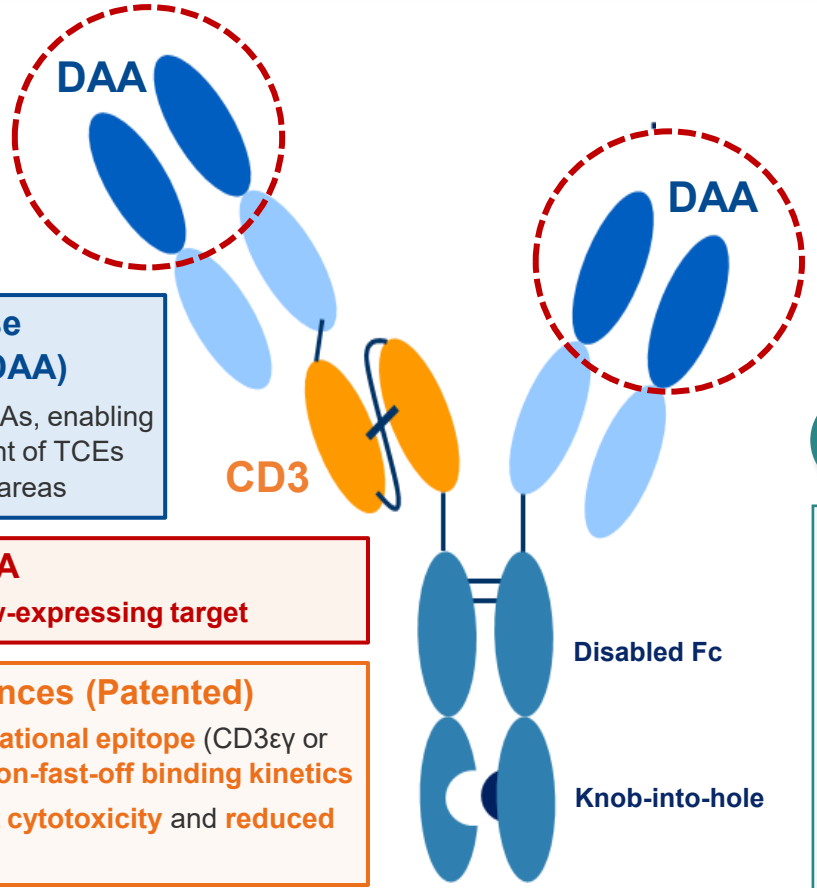
Features of AnTenGager™ TCEs



"Plug and Play" Disease Associated Antigens (DAA)

- Compatible with diverse DAAs, enabling the discovery & development of TCEs across multiple therapeutic areas

Bivalent Binding of DAA

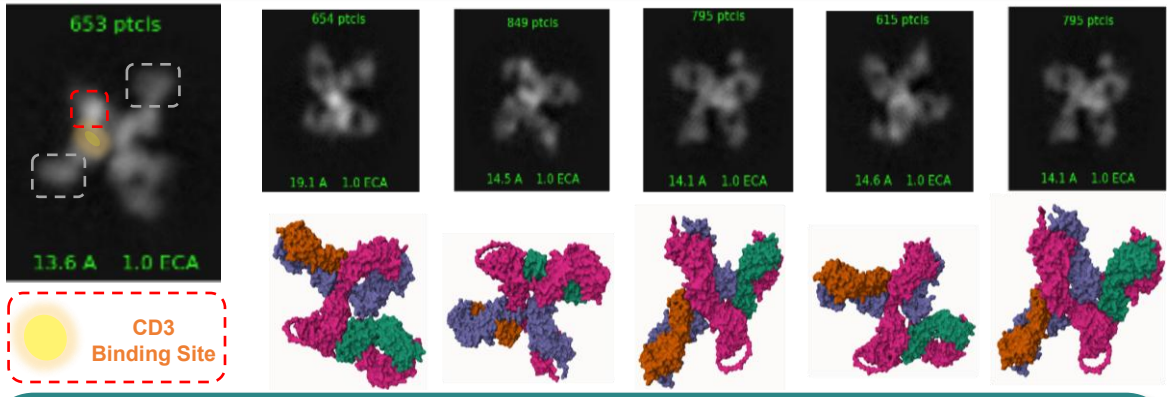
- Enables the targeting of **low-expressing target**

Proprietary CD3 Sequences (Patented)

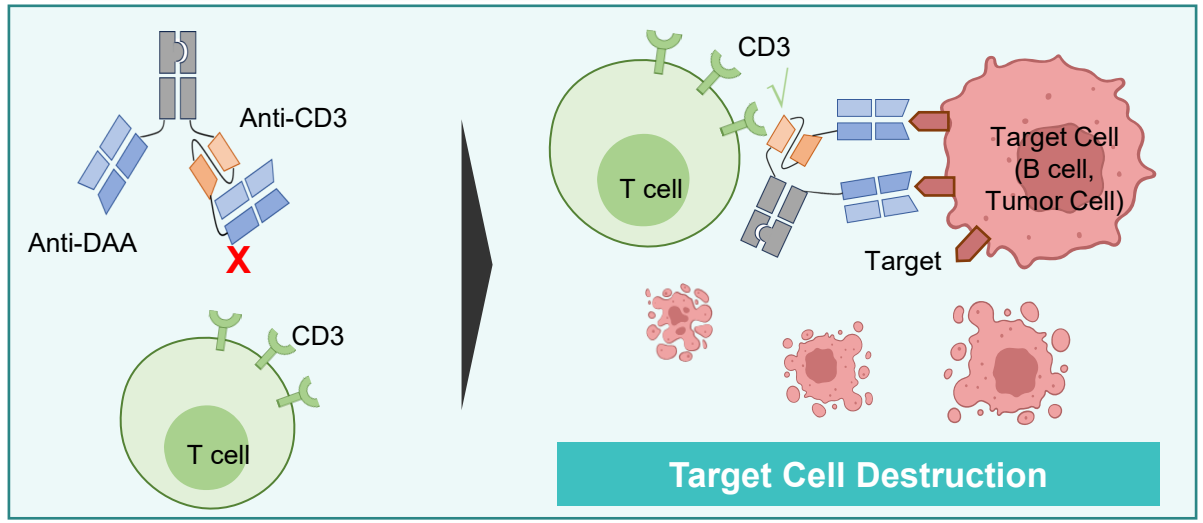
- Binds to a **unique conformational epitope** (CD3εγ or CD3εσ complex), with **fast-on-fast-off binding kinetics**
- **Stronger T cell dependent cytotoxicity** and **reduced cytokine release**

Steric Hindrance Masking Technology

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



Target-Dependent CD3 Binding and Cytotoxicity



Masking Strategies of T Cell Engagers

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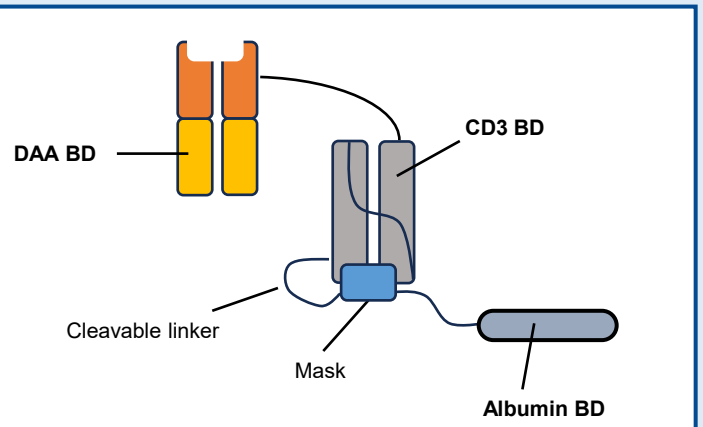
Peptide Mask

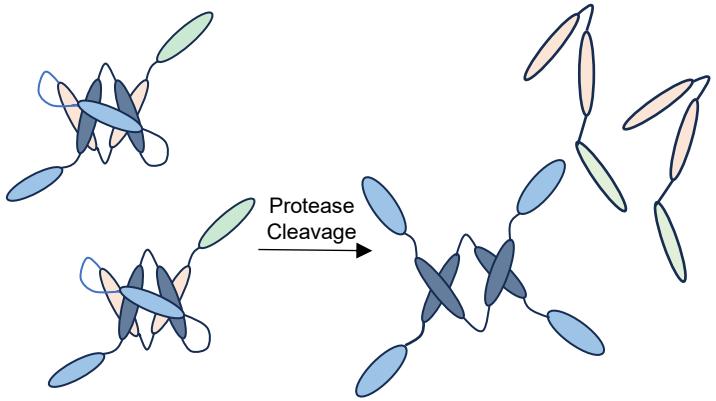


Tumor Microenvironment Dependent

Protease Dependent

Enzyme Cleavage Site Exposed

Structural Locking

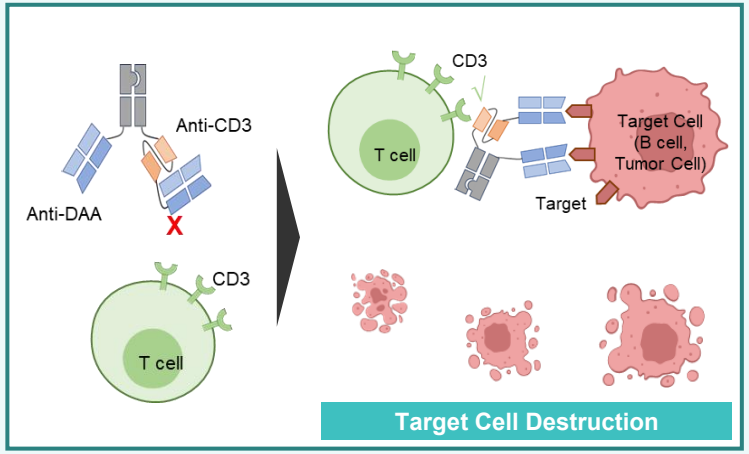


Tumor Microenvironment Dependent

Protease Dependent

Conformation Change Induced by Enzymatic Cleavage, Creating CD3 Binding Site

Steric Hinderance (Antengene AnTenGager™)



Independent to Tumor Microenvironment

DAA Binding Dependent

Target Binding Induces Conformational Changes, Exposing CD3 Binding Site

CD3 Binding Site of AnTenGager™ TCE is Concealed by DAA Fab Arm

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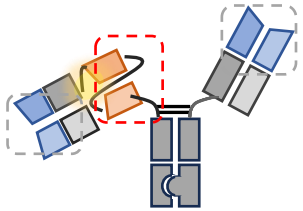
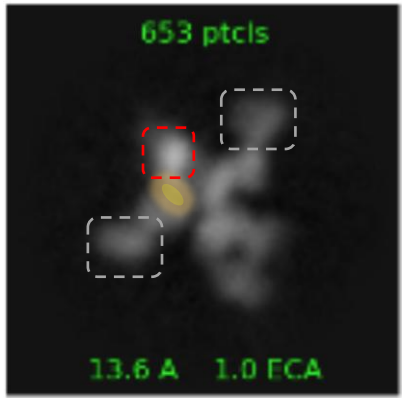
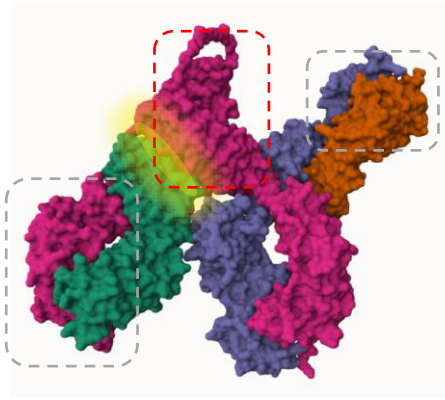
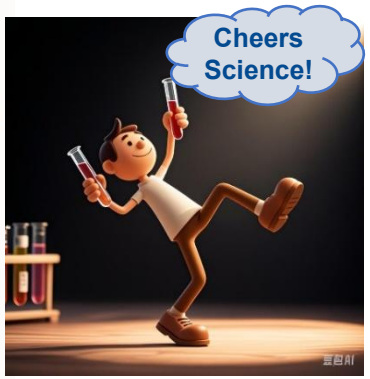
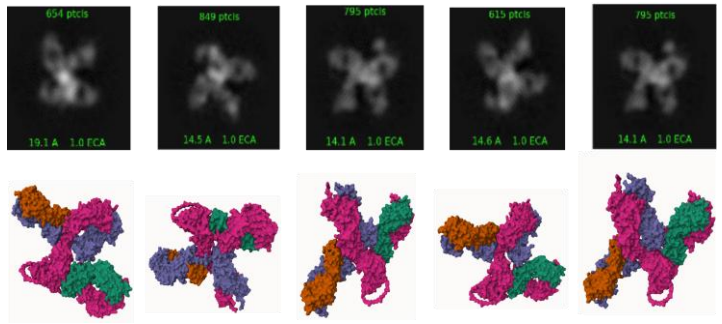
AnTenGager™ Platform

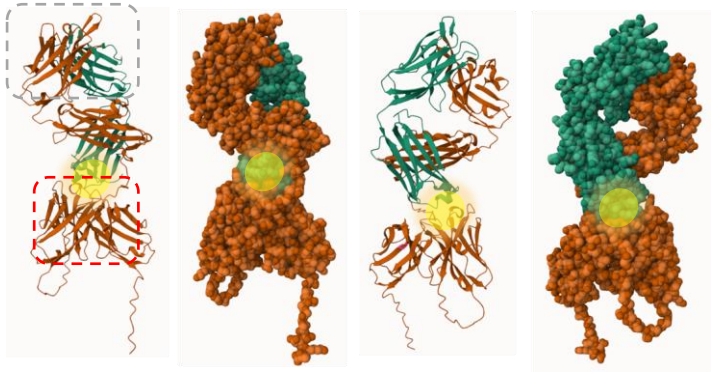








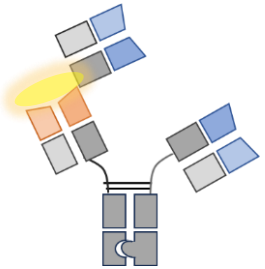


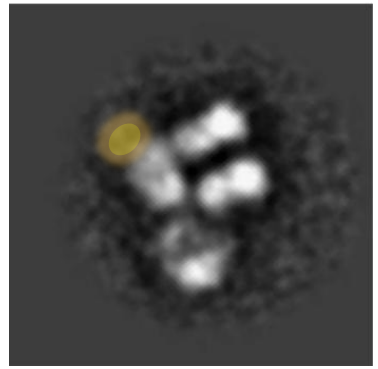


- The CD3 binding site is **tightly concealed** by the constant region of DAA-targeting Fab arms in the unbound state due to **steric hindrance**

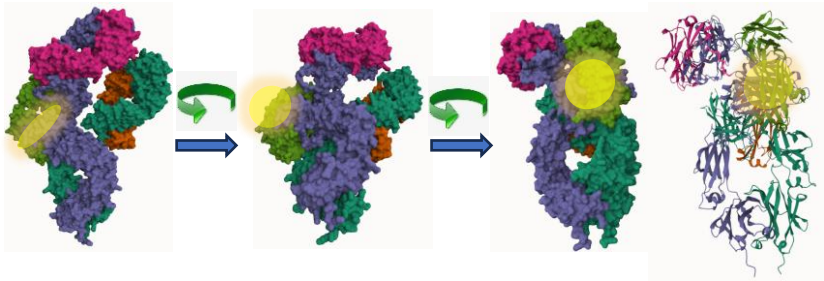
CD3 Binding Site

Fabx3 Platform





Segal, N.H. et al. Annals of Oncology, Volume 28, v134



- Fabx3 2+1 format maintains continuous exposure of CD3 binding sites due to the higher rigidity of its Fab arms

AnTenGager™ TCEs Showed Target-dependent CD3 Binding and Cytotoxicity

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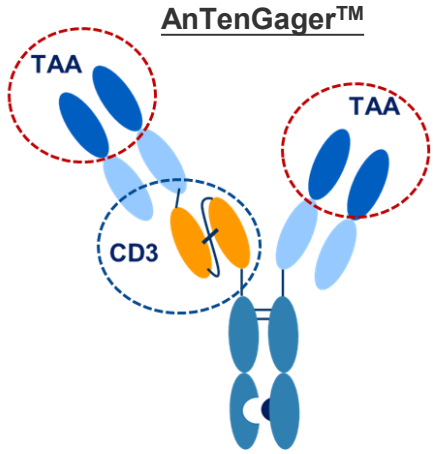
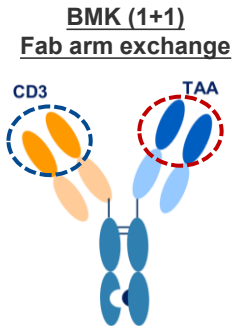
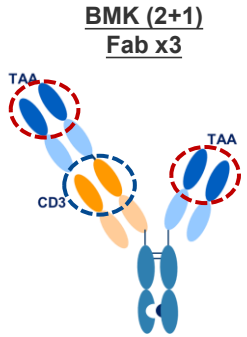
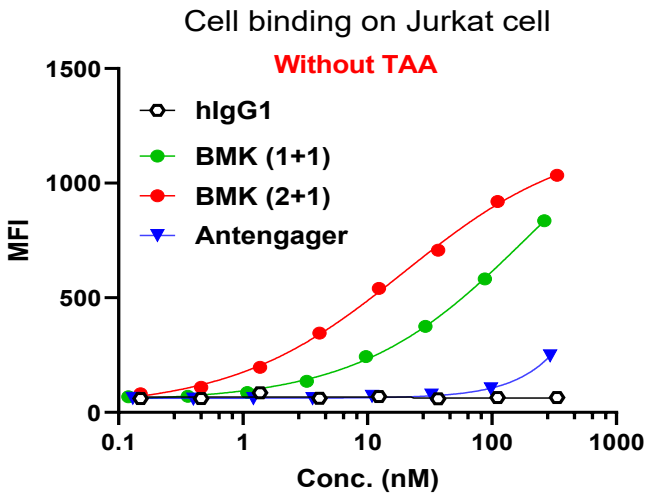
AnTenGager™ TCE Has Reduced CD3 Binding in the Absence of Target-Cross Linking and Enhanced Target-specific Cytotoxicity Compared with Benchmarks (BMKs)

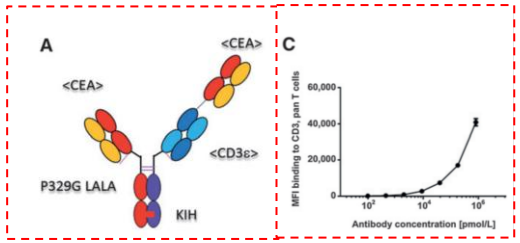






Cell Binding on Jurkat Cell (Without DAA)

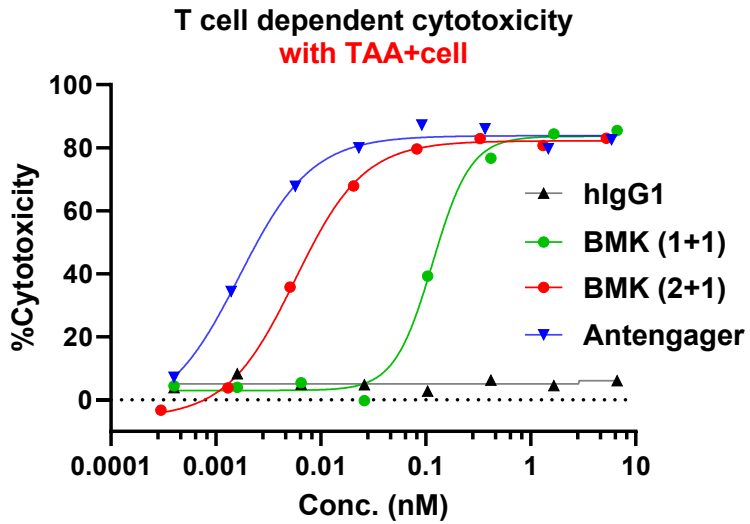




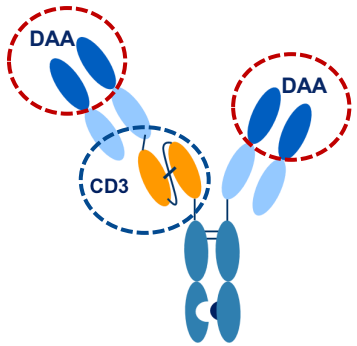
Benchmark Reported Similar Results

Clin Cancer Res (2016) 22 (13): 3286–3297

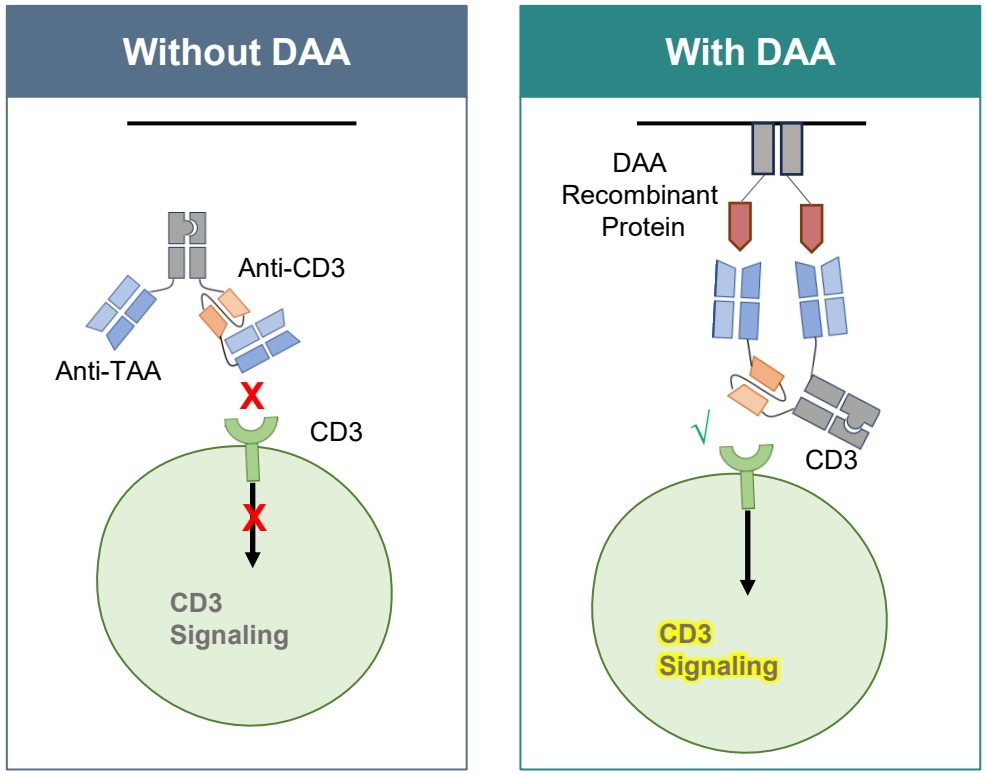
***In Vitro* Cytotoxicity (With DAA)**

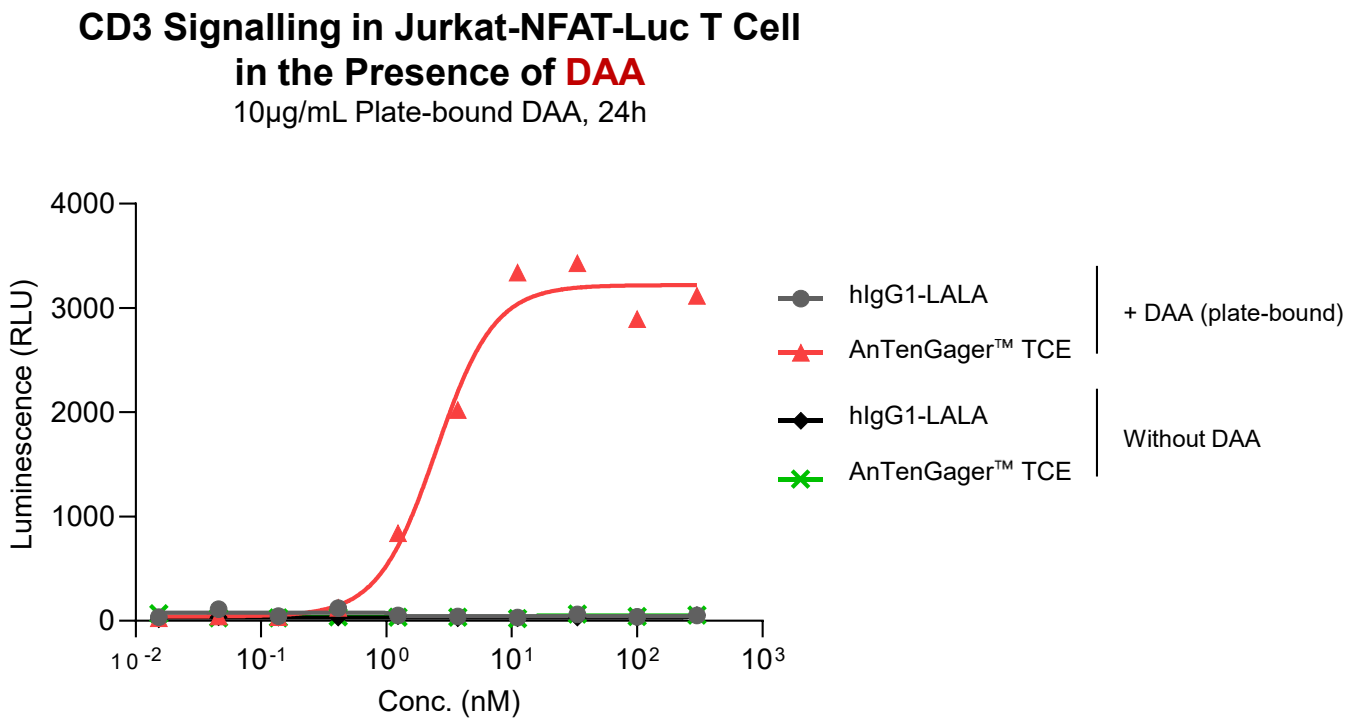


AnTenGager™ TCEs: Enhanced Safety Through DAA-dependent CD3 Activation



- **Steric Hindrance and Masking Effect:** The **CD3 binding site is concealed** by the DAA Fab arm before antigen binding, **preventing unintended activation** and enhancing safety
- **Bivalent Binding of Disease-associated Antigen (DAA):** Enables the targeting of low-expressing target with reduced risk of hook effect
- **DAA-Dependent Activation:** Upon DAA crosslinking, the **CD3 binding arm is exposed, enabling specific and potent CD3+ T cell activation** only in the presence of the target antigen





Evaluation and Selection of Antibody Affinity

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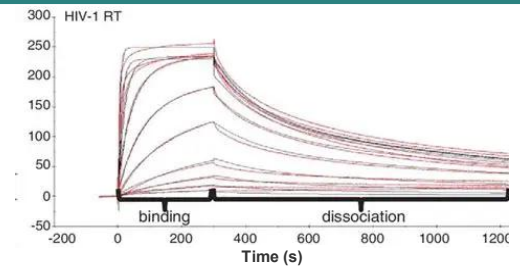
Affinity vs Avidity

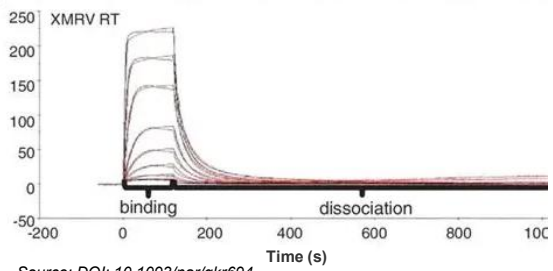
Source: https://www.novusbio.com/support/general-support/antibody-basics.html

Antibodies with **bivalent or multivalent binding** have higher **avidity**, which lowers the minimum target copy number required for achieving therapeutic effect

On Rate and Off Rate



Source: DOI: 10.1093/nar/gkr694



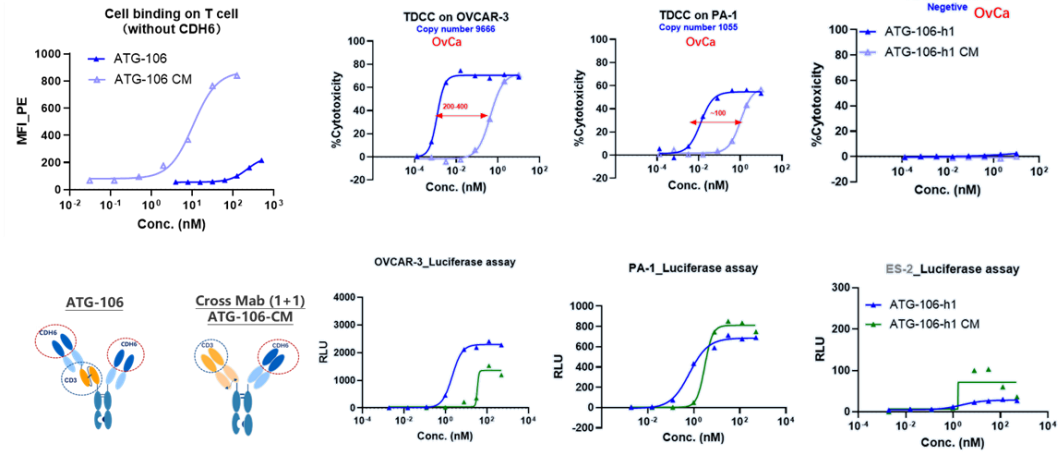
Source: DOI: 10.1093/nar/gkr694

- The **on-rate** and **off-rate** together determine how long an antibody stays on the target cell and how long it can exert its function
- A **faster off-rate** shortens the functional duration of the antibody; a **fast on-rate** can compensate for a fast off-rate
- For **TCEs, fast-on/fast-off CD3** binding results in **less cytokine release** and **reduced T-cell exhaustion**, while still maintaining **good TDCC**
- For **ADCs**, a **longer residence time** leads to **more total antibody internalization**, which is beneficial for **bystander-killing effects**

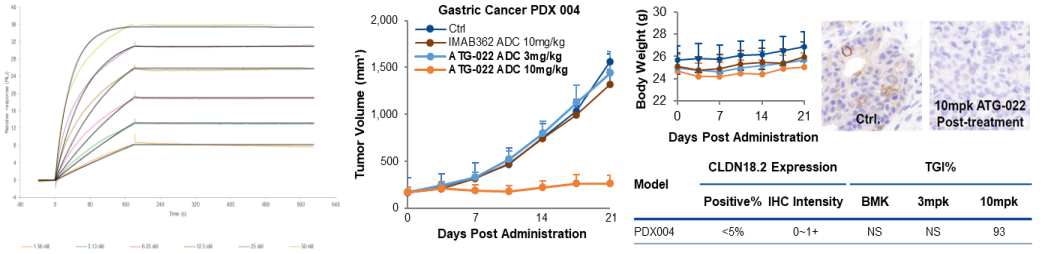
Examples

ATG-106 Has Reduced CD3 Binding in the Absence CDH6 and Enhanced Target-specific Cytotoxicity Compared with Crossmab 1+1 Format

Without CDH6 | **With CDH6** | **Without CDH6**



High Affinity CLDN18.2 ADC Demonstrated Efficacy in Low Expressor



Model	CLDN18.2 Expression		TGI%		
	Positive%	IHC Intensity	BMK	3mpk	10mpk
PDX004	<5%	0~1+	NS	NS	93

Proprietary Parental CD3 mAb for AnTenGager™ TCEs Demonstrated Differentiation Compared to SP34

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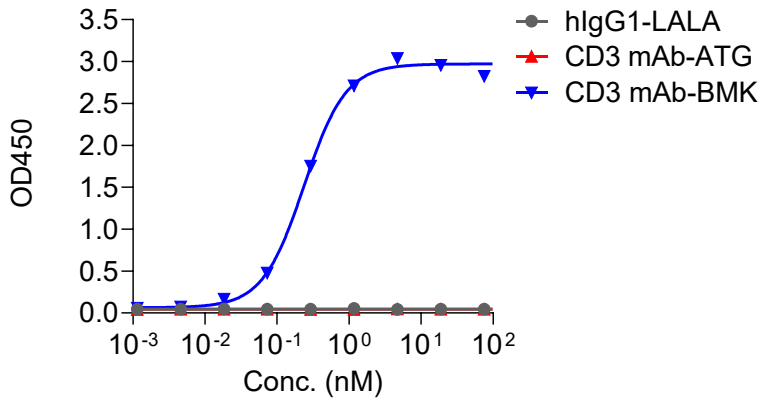
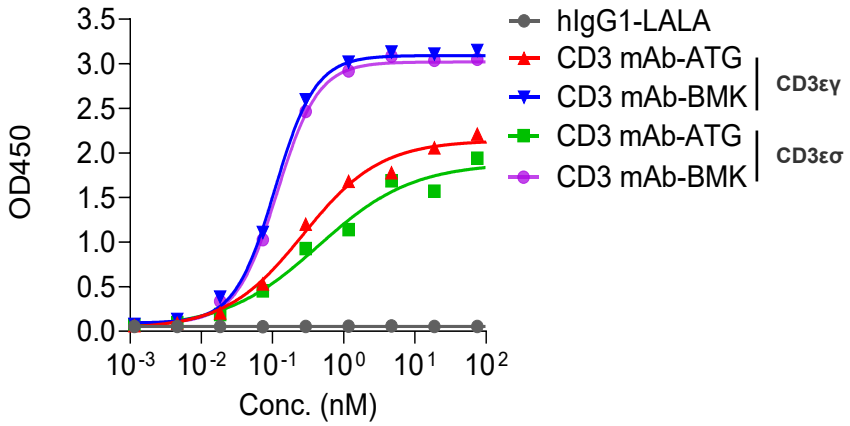
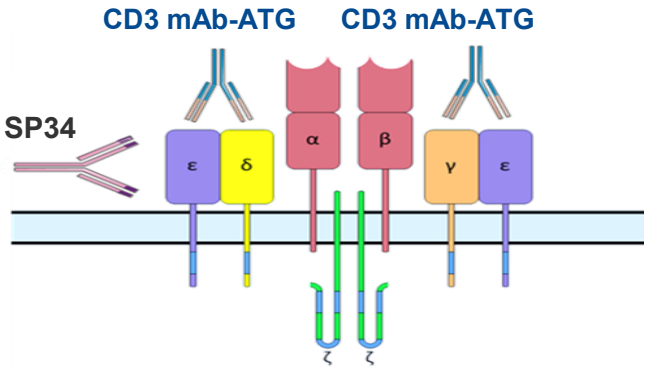
- CD3 parental mAb from AnTenGager™ TCE **did not bind to CD3ε monomer in ELISA,** while **recognizing a conformational epitope on CD3εγ or CD3εσ complex**
- Benchmark anti-CD3 (SP34) binds to a linear epitope on CD3ε monomer. SP34-derived CD3 arm was used in many competitor's TCE platforms/products
- Compared to SP34, AnTenGager parental CD3 mAb showed reduced activation of CD3 signaling , while inducing comparable or enhanced TDCC and reduced cytokine release

(A) CD3ε Binding



(B) CD3εγ and CD3εσ Binding





AnTenGager™ TCEs Demonstrated Enhanced *In Vivo* Efficacy and Low Risk of Cytokine Release Syndrome (CRS) in Human PBMC Re-established Tumor Model

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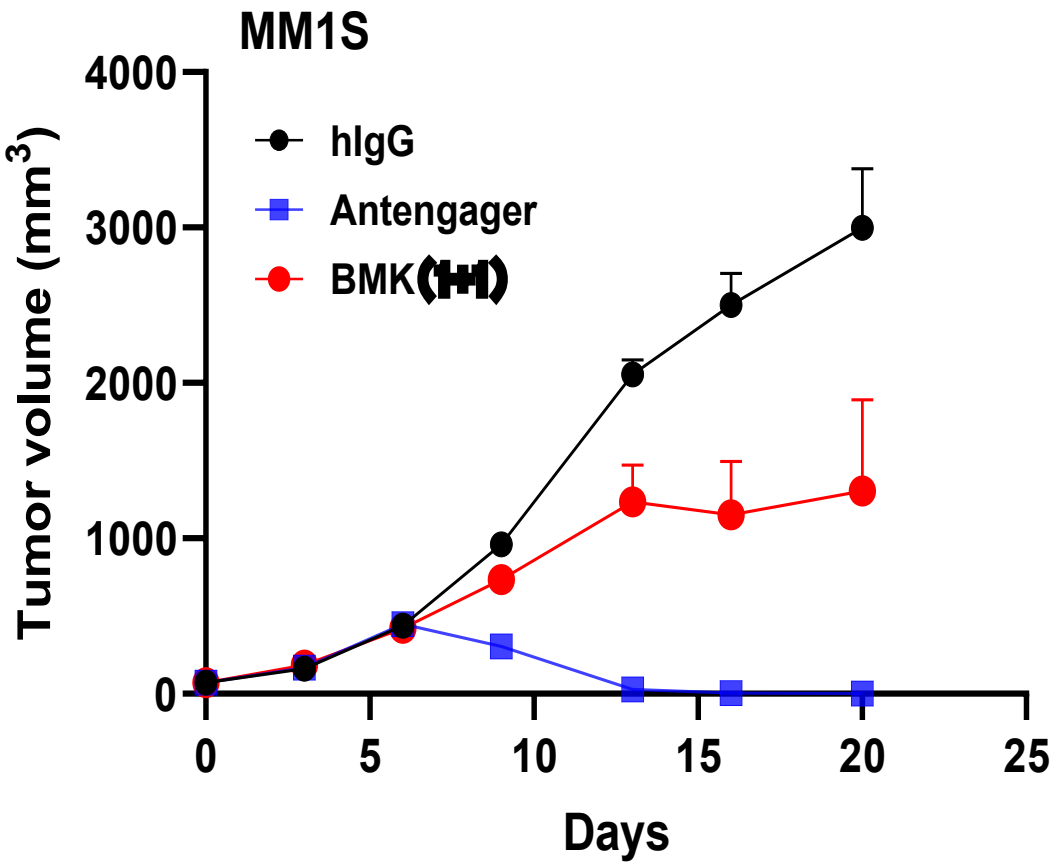
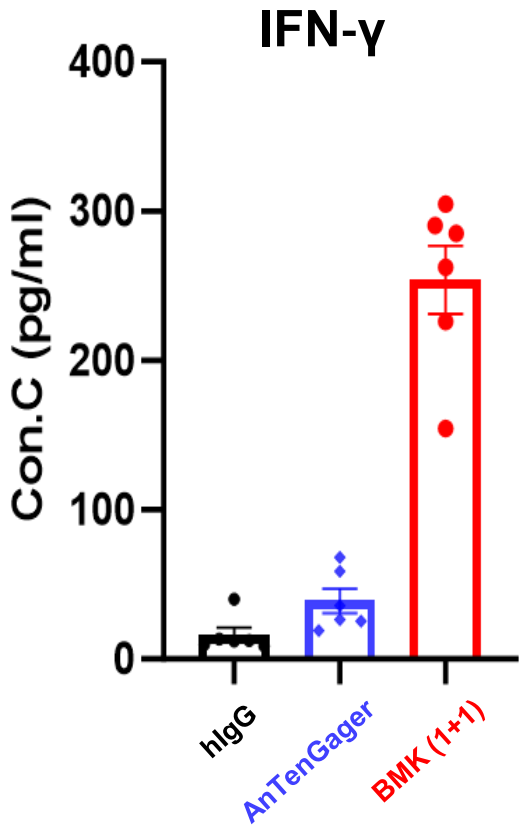
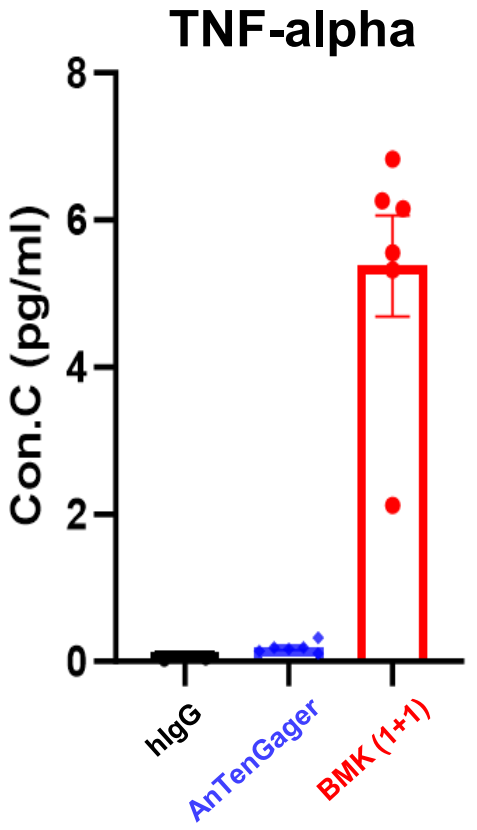
Enhanced Efficacy Compared to Benchmark



Significantly Reduced Release of Cytokines Compared to Benchmark Indicating Low Risk of CRS





AnTenGager™ TCEs: Favorable PK Profile and Long Half-Life in Mice Suggest Reduced Dosing Frequency and Improved Clinical Convenience

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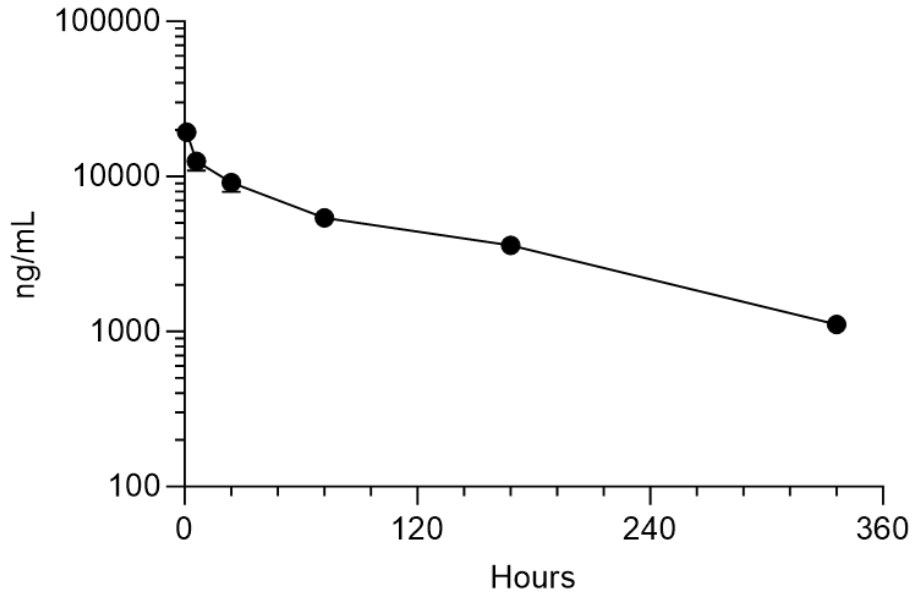
- AnTenGager™ TCEs demonstrated good PK profile in mice, with $T_{1/2}$ of 100h-300h

Pharmacokinetics of AnTenGager™ TCEs 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	Indication	mAb Discovery	*In vitro* Efficacy	*In vivo* Efficacy	Developability	CMC/Tox	IND
ATG-201	CD19 x CD3	B Cell Related Autoimmune Diseases						
ATG-106	CDH6 x CD3	Ovarian Cancer & Kidney 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-110	LY6G6D x CD3	Microsatellite Stable (MSS) Colorectal Cancer						
ATG-107	FLT3 x CD3	Acute Myeloid Leukemia						
Undisclosed Bispecific TCE	Undisclosed	Liver Cancer						
Undisclosed Trispecific TCE	Undisclosed	Metastatic Castration-resistant Prostate Cancer						
Undisclosed Trispecific TCE	Undisclosed	Small Cell Lung Cancer and Neuroendocrine Tumors						

AnTenGager™ TCE 2.0: Overcoming CRS Barriers and Preventing T Cell Exhaustion to Enable Broader, More Effective, and Safer Therapeutic Applications

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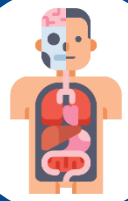


Minimizing Off-target T Cell Activation

Steric Hindrance Masking Technology

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

Minimizing On-target T Cell Activation

Proprietary Anti-CD3 Sequences

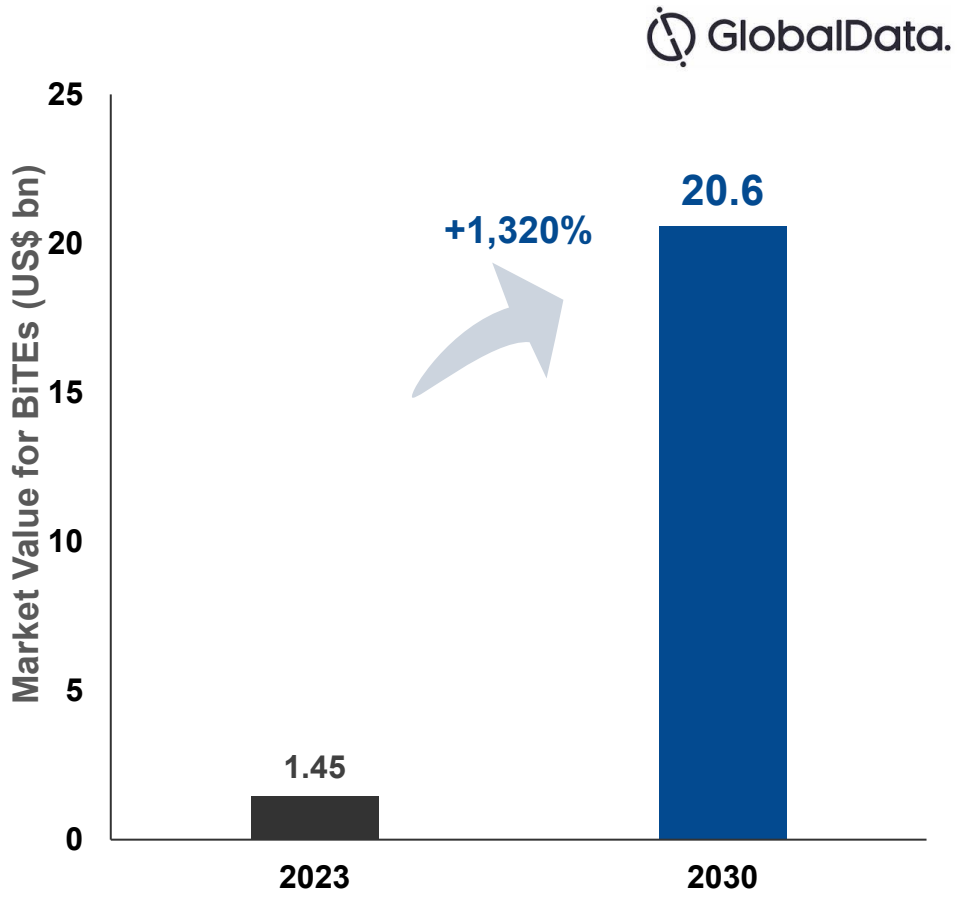
- **Minimizes on-target T cell activation and cytokine release** by binding to a **unique conformational epitope** with **fast-on-fast-off** binding kinetics while maintaining potent T cell activation

- **Enhances Efficacy**
- **Improves Safety**
- **Prevents T Cell Exhaustion**
- **Minimizes Hook Effect**

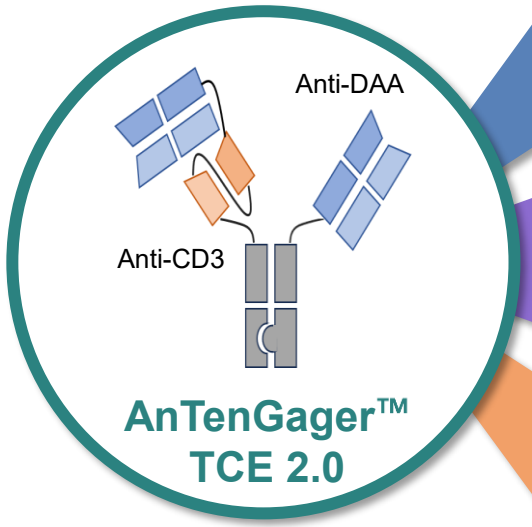
Growing TCE Market – AnTenGager™ TCE 2.0 Leads the Way Globally with Significant Commercial Potential

ANTENGENE

Explosive Growth Projected for the Bispecific T Cell Engager (BiTE) Market



AnTenGager™ TCE 2.0 with Steric Hindrance-Masking Technology Enable Broad Applications Across Multiple Indications



Solid Tumors

(e.g., CDH6, LY6G6D, ALPPL2)

Hematological Malignancies

(e.g., LILRB4, GPRC5D, FLT3)

Autoimmune Diseases

(e.g., CD19, CD20)

Source: GlobalData

ANTENGENE

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

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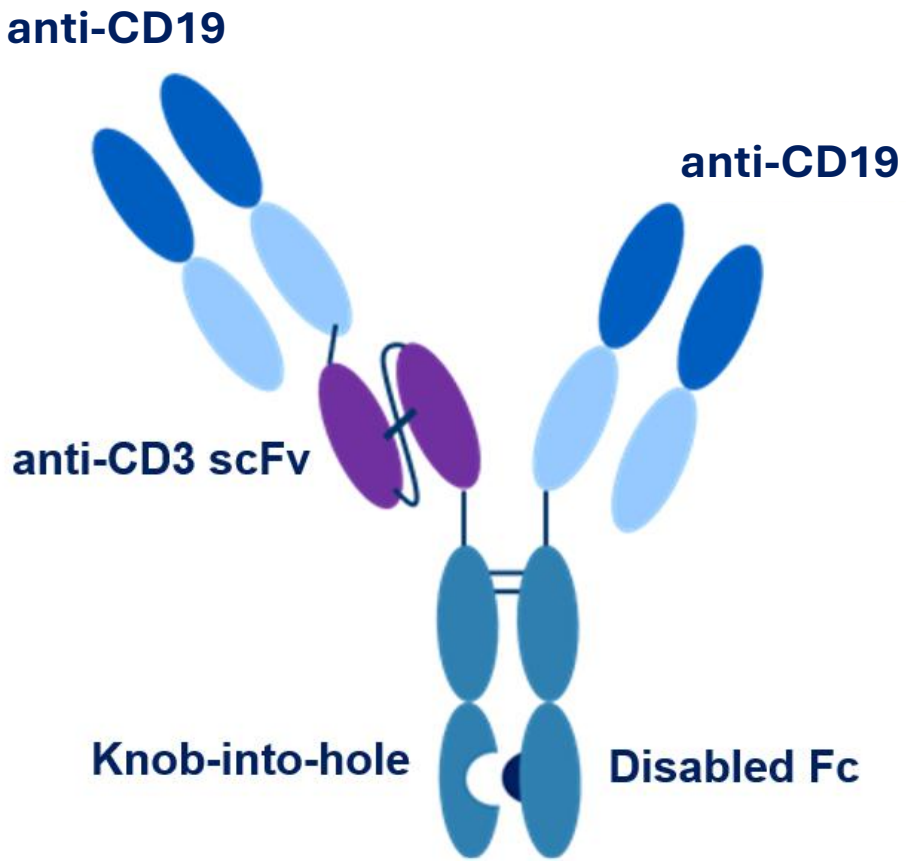
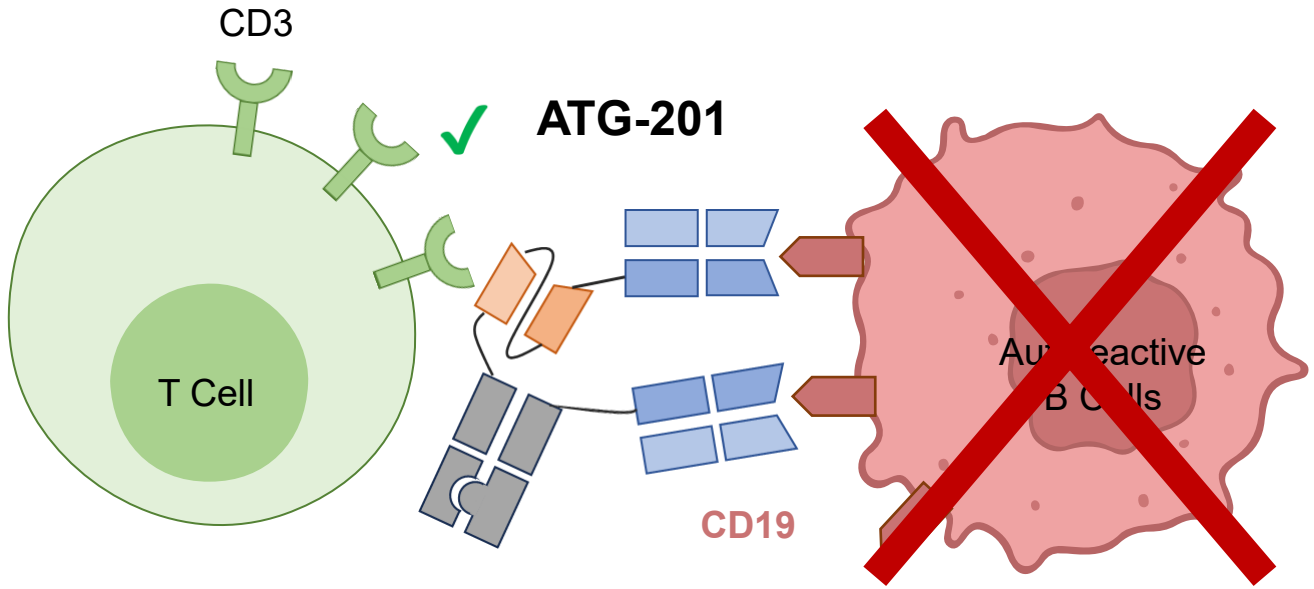
ATG-201 is a CD19 x CD3 TCE with Target Dependent T Cell Activation

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





B Cell Depletion Leads to the Remission of Autoimmune Diseases

CD19 x CD3 T Cell Engagers for Autoimmune Diseases: Competitive Advantages of Second Generation "2+1" vs. First Generation "1+1" TCEs

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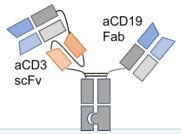
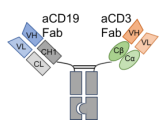
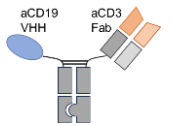
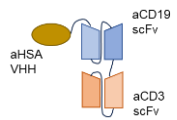
	Second Generation TCE for Autoimmune Diseases	First Generation TCEs for Oncology				
	ATG-201	**TCE 1**	**TCE 2**	**TCE 3**	**TCE 4**	
Company	ANTENGENE	Company 1	Company 2	Company 3	Company 4	
Format						
CD19 Binding	**Bivalent**	**Monovalent**	**Monovalent**	**Monovalent**	**Monovalent**	
CD19 Dependent CD3 Activation	**Yes (via Steric Hindrance; CD19-gated T cell activation reduces CRS risk)**	**No**	**No**	**No**	**No**	**… and others**
***In Vivo* Efficacy in Autoimmune Diseases**	Potent efficacy in SLE and MS model; Deep and sustained B cell depletion in mice (blood, lymph node, bone marrow, and spleen)	No autoimmune animal model data published	Not publicly disclosed	Not publicly disclosed	Sustained B cell depletion in blood in monkey	
Half-life	**mAb-like (~12.5 days in mice)**	**Short, ~2.1 hours in clinic**	~7 days	9-11 days	5-7 days in monkey	
Pros & Cons	• **Bivalent CD19 binding enables the targeting of CD19 low expressors** • **CD19-gated T cell activation reducing CRS risk** • **Potent efficacy** • **Long half-life**	• High potency, but **higher CRS risk than ATG-201** • **Short half life leads to 24/7 dosing** • **Poor drug stability: 48h in RT**	• Clinically manageable CRS using 2-step-up dosing, but **the low and monovalent CD19 binding affinity may lead to insufficient autoreactive B cell depletion**	• **Low efficacy against naïve B cells *in vitro***	• **High CRS risk and less potent efficacy against Naïve B compared to ATG-201**	

2nd Generation "2+1" Bivalent TCEs: Superior to 1st Generation Designs in Autoimmune Disease, Engineered for Deeper and Safer CD19+ B Cell Depletion

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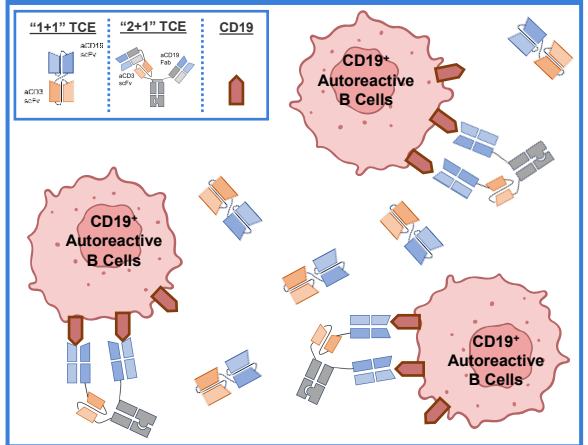
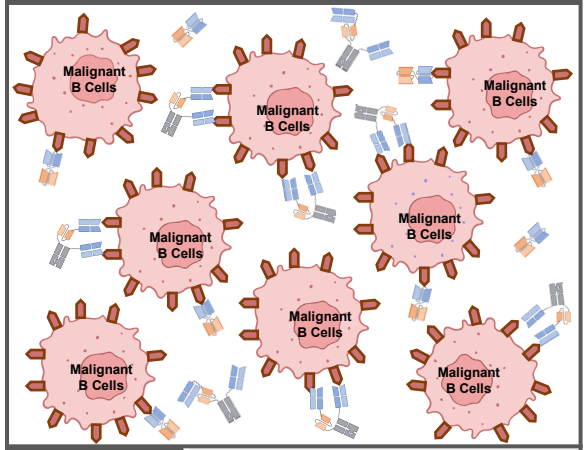
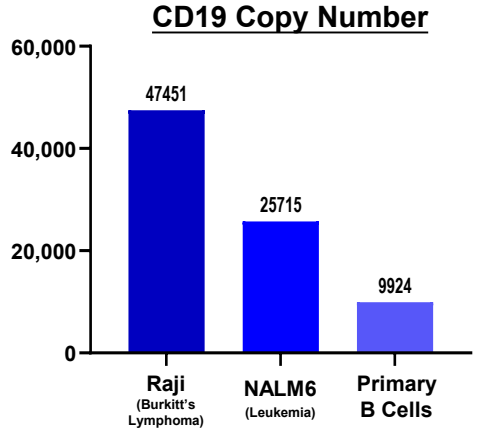
Repurposed First-Generation "1+1" TCEs Are Obsolete in Autoimmune Diseases Due to Limited Efficacy and Higher Toxicity

"2+1" TCEs: Purpose-Built for Autoimmune Disease

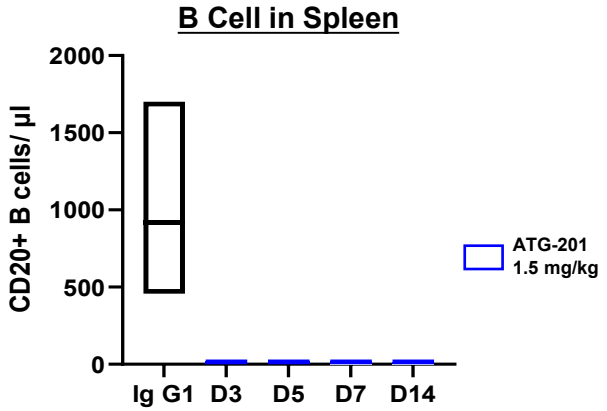
Engineered to Address Distinct Disease Biology of Autoimmune Diseases Not Targetable by Repurposed "1+1" TCEs from B Cell Malignancies

	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. Repurposed 1st Generation TCEs *Ex Vivo*

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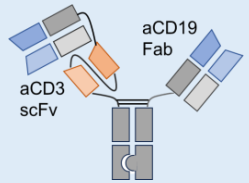
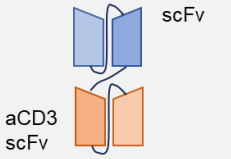
ATG-201



Benchmark 1



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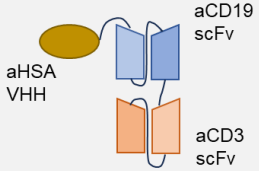
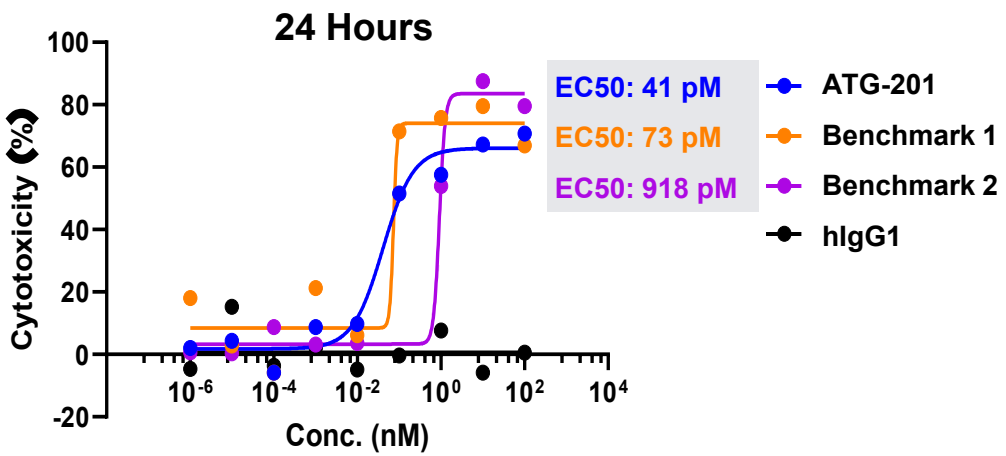
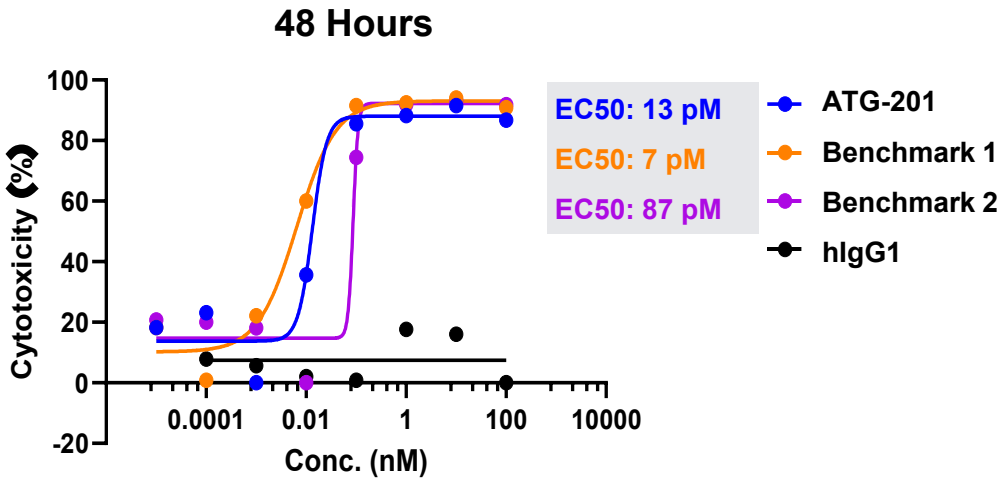


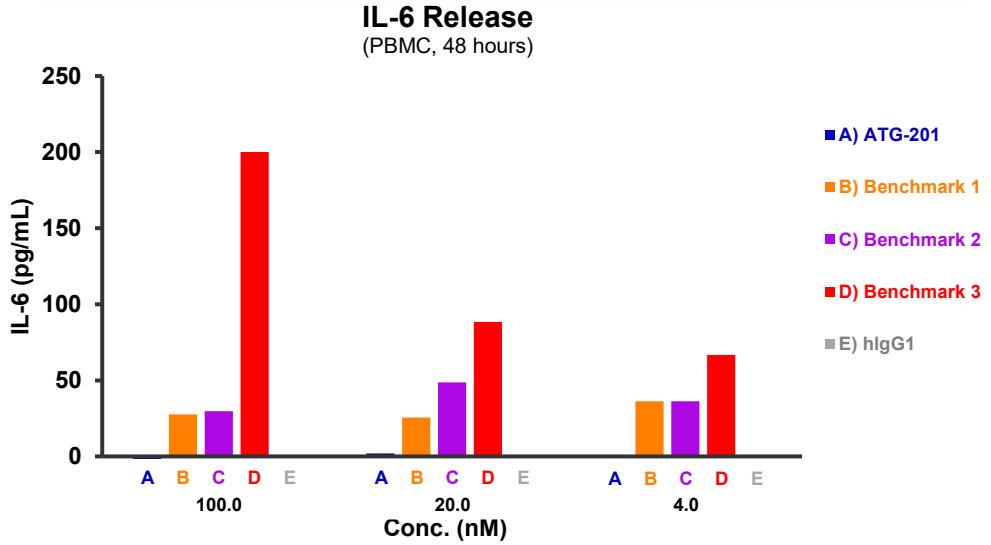
Benchmark 3

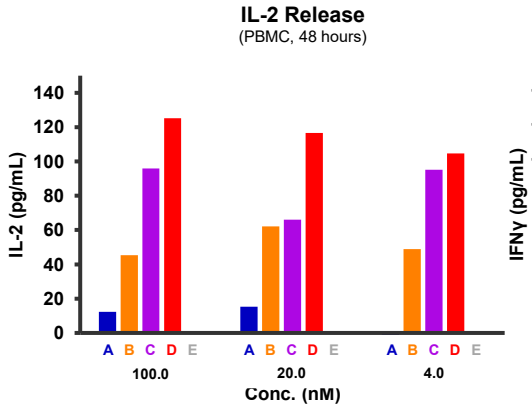
Comparable or Enhanced Naïve B Cell Depletion vs. Benchmarks

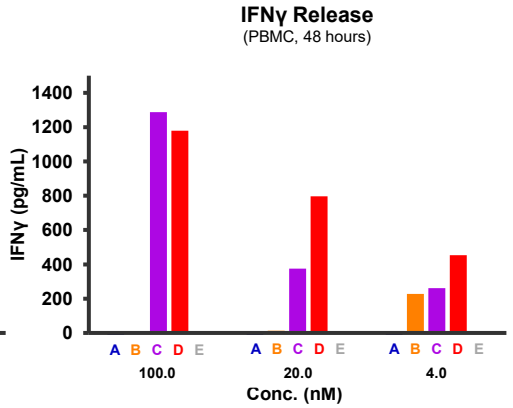




Reduced Cytokine Release vs. Benchmarks



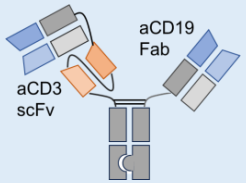




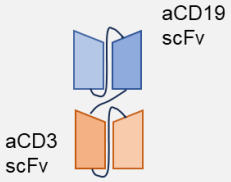
ATG-201 Induced Less T Cell Exhaustion vs First Generation TCEs

ANTENGENE

ATG-201

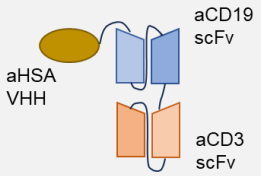


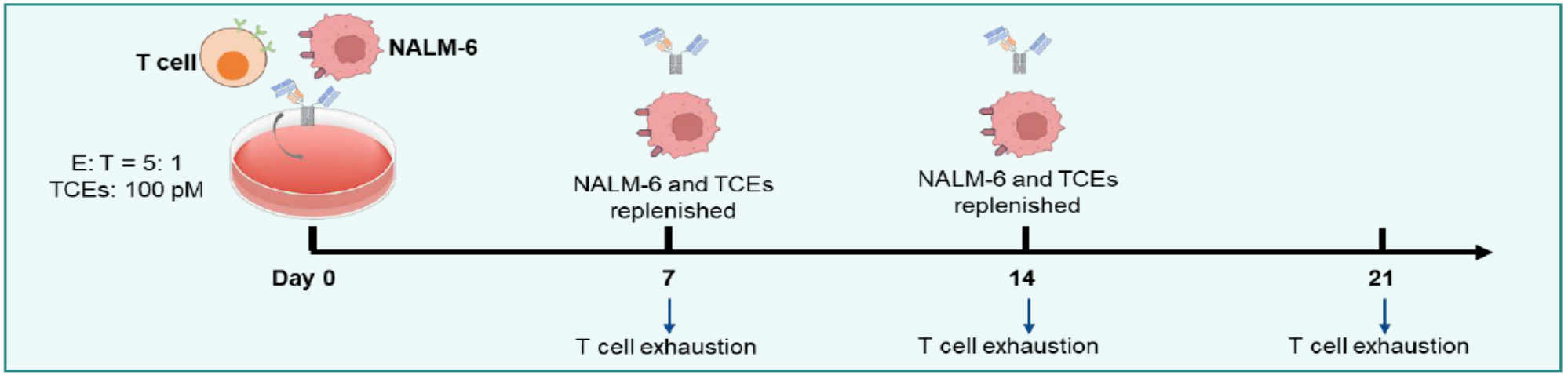
Benchmark 1

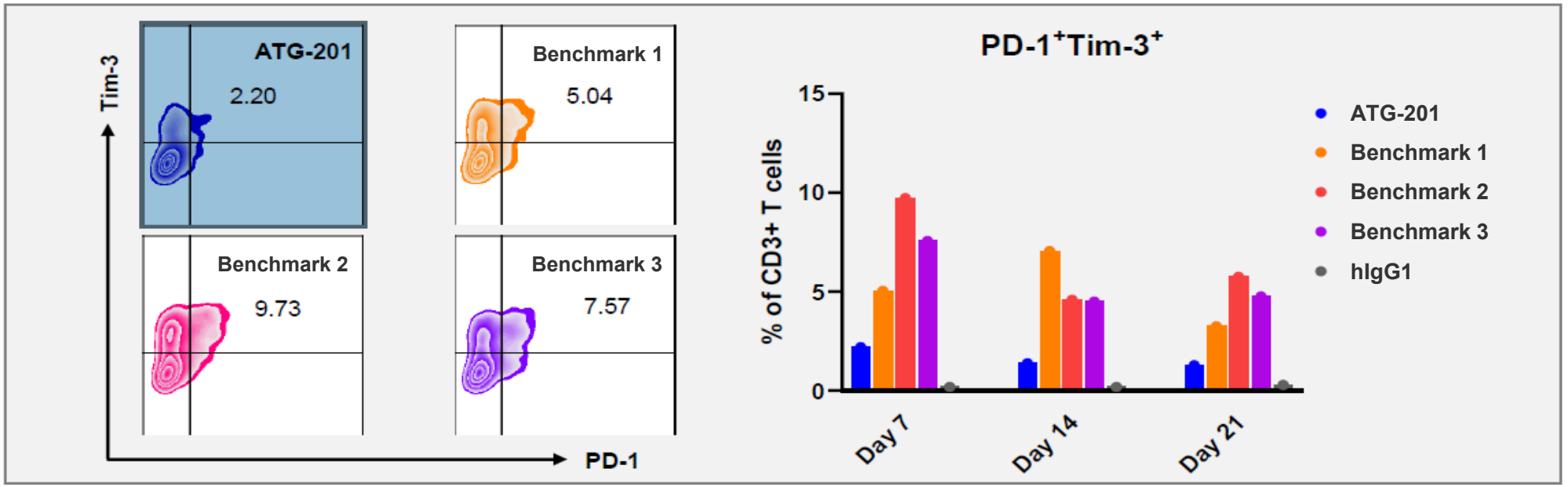


Benchmark 2

Benchmark 3



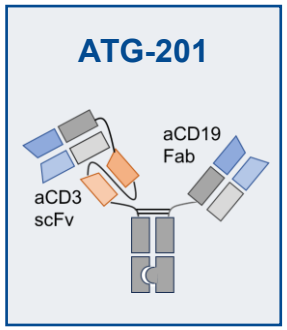


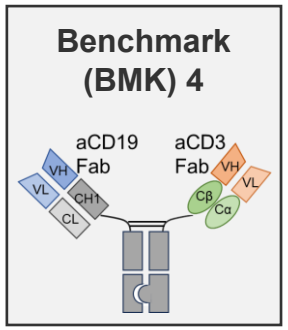


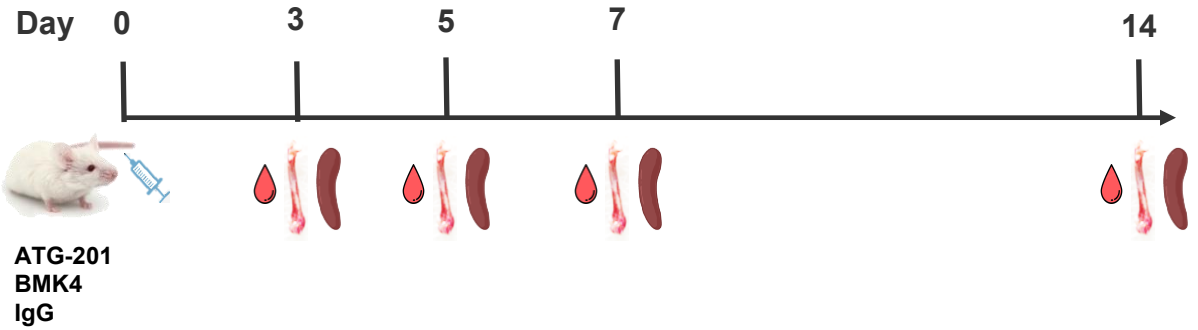
ATG-201 Demonstrated Deeper and More Durable *In Vivo* B Cell Depletion Compared to Benchmark in CD34+ Cell Humanized Mice

ANTENGENE

- **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



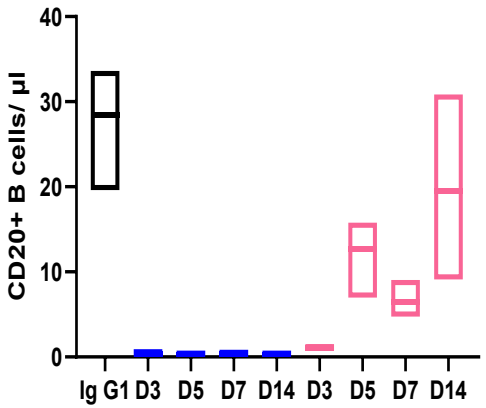


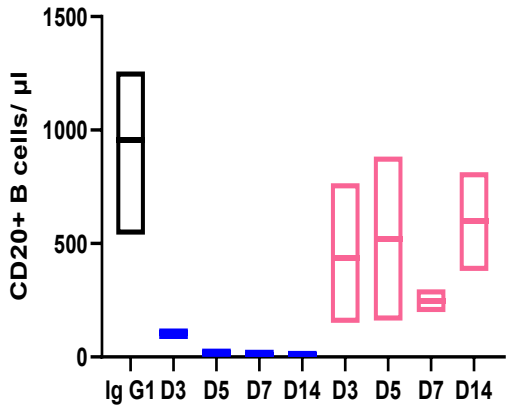


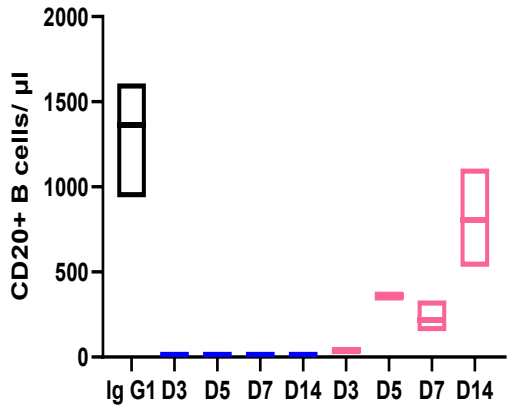
ATG-201
BMK4

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

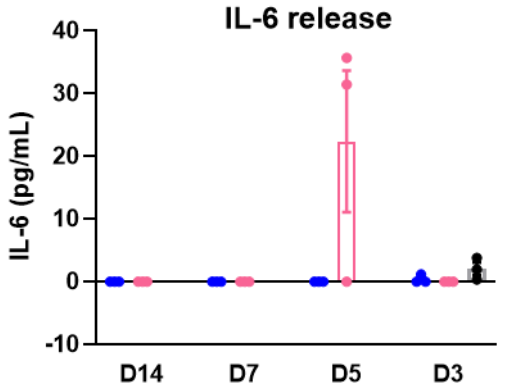
B Cells

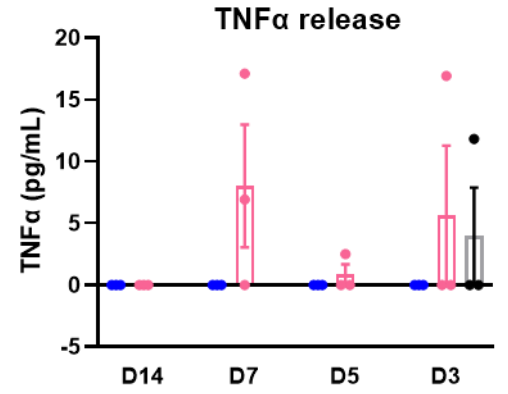






Proinflammatory Cytokine

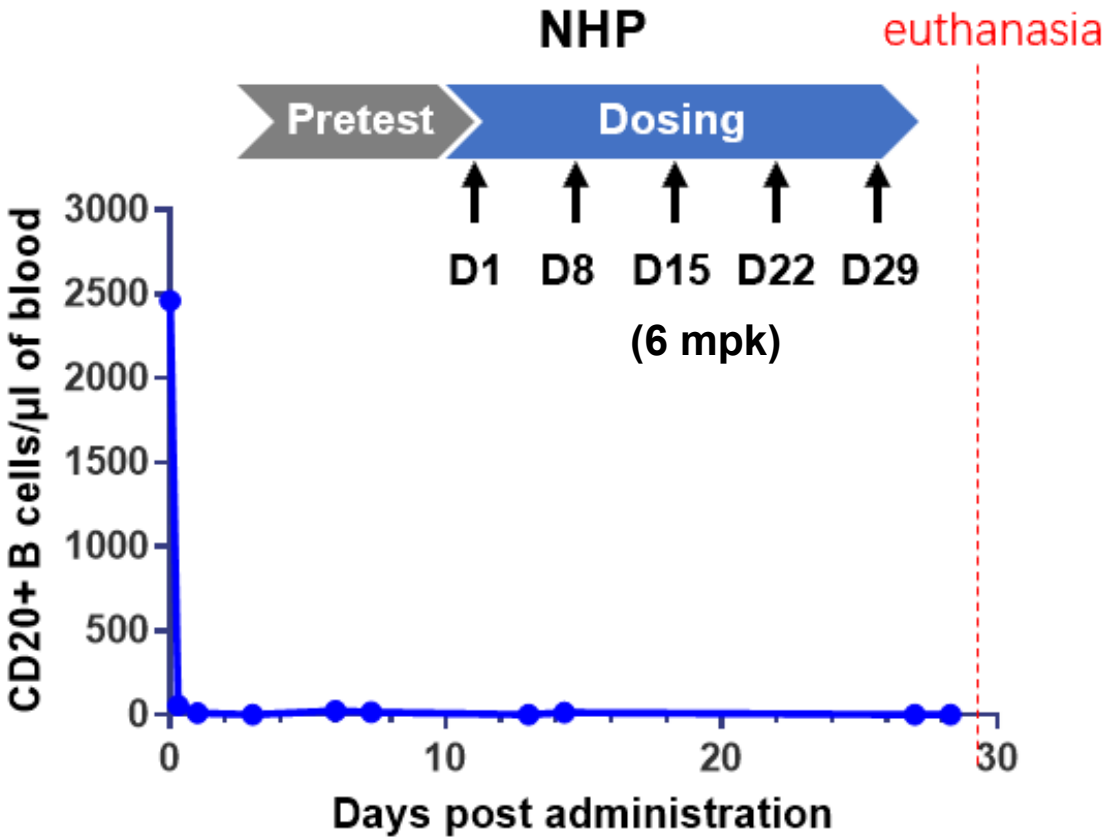


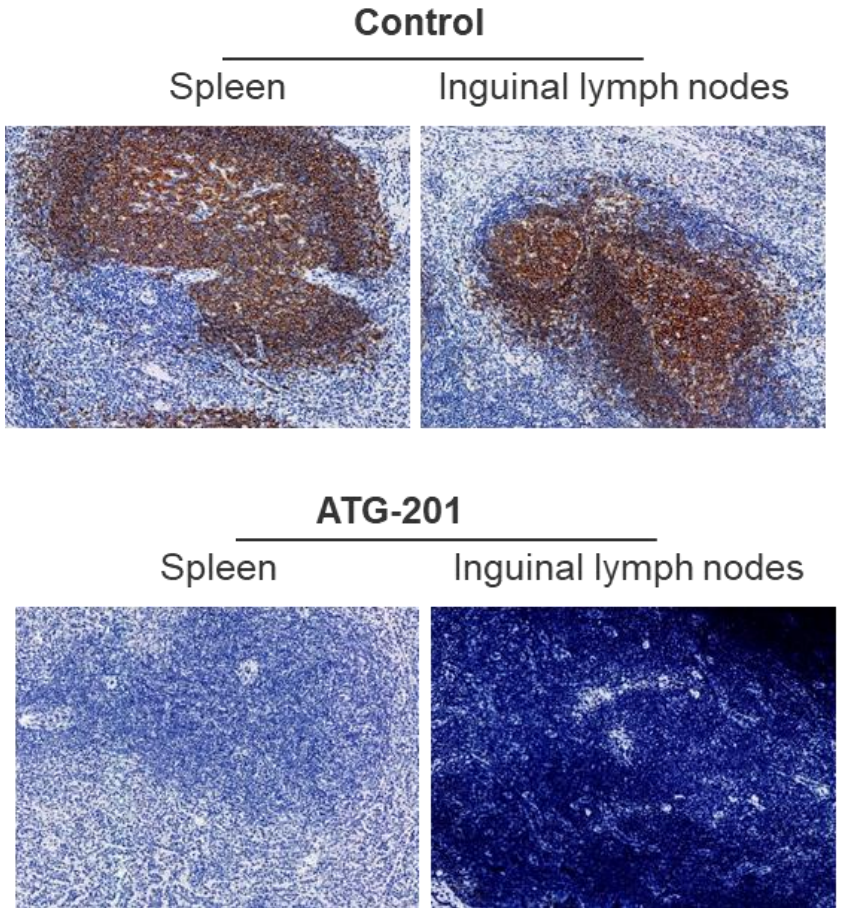


ATG-201 Surrogate Antibody in NHP Demonstrated Low Cytokine Production and Complete B Cell Depletion

ANTENGENE

- Repeated dosing of ATG-201 surrogate (1mpk, 3mpk, 6mpk) is **well tolerated in NHP**, with **low cytokine production** observed
- ATG-201 surrogate induced **complete B cell depletion in peripheral blood, spleen and lymph nodes**

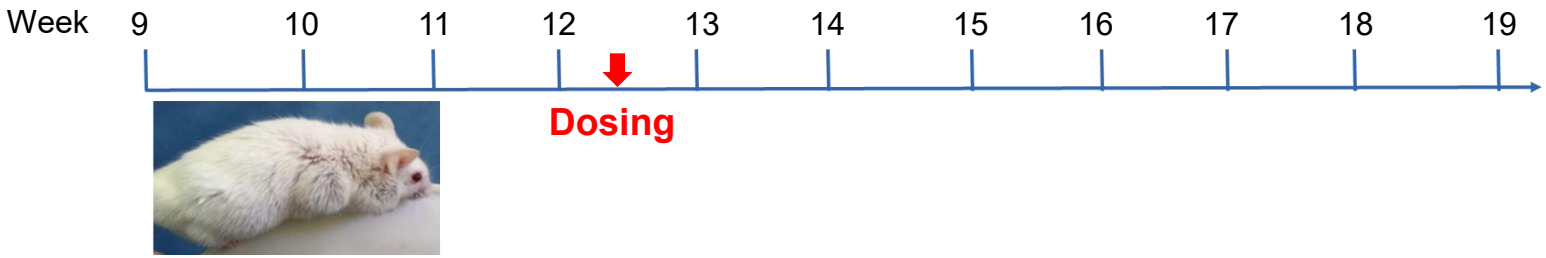


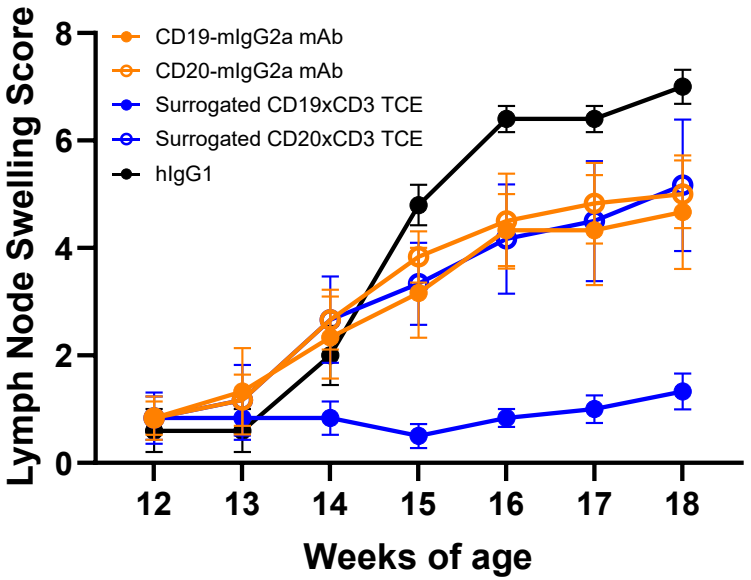


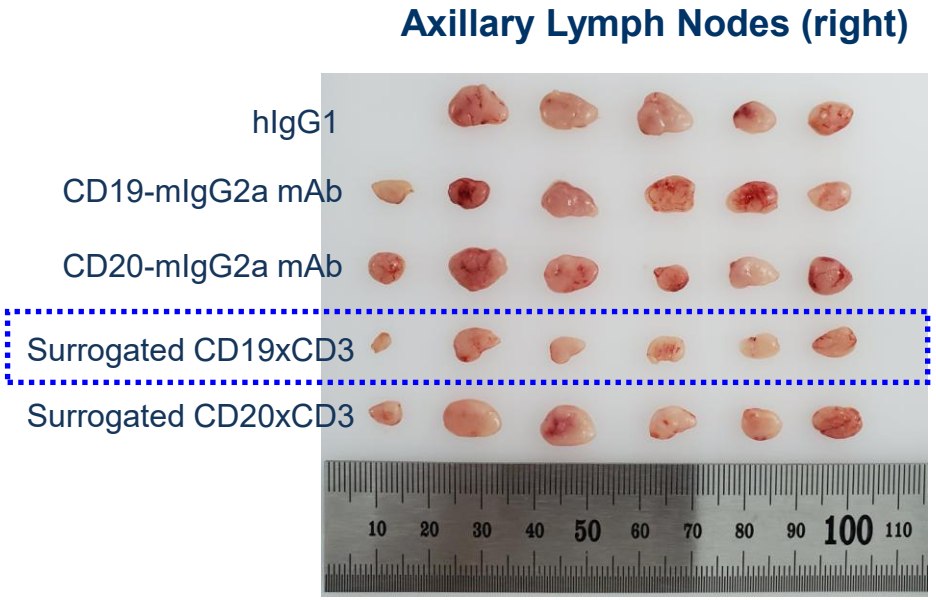
CD19 x CD3 TCE Demonstrates Potent Efficacy in Mouse MRL-lpr Spontaneous Systemic Lupus Erythematosus (SLE) *In Vivo* Model

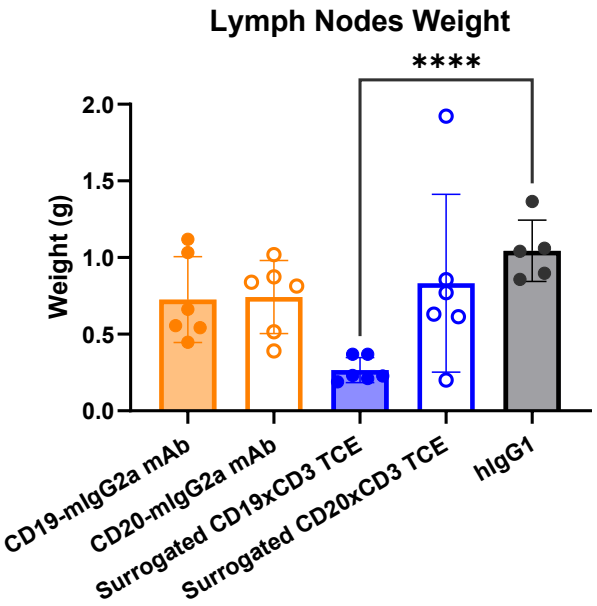
ANTENGENE

- The surrogated **CD19 x CD3 TCE demonstrated enhanced efficacy** in suppressing disease progression than CD20 x CD3 TCE, and CD19 or CD20 monoclonal antibody in spontaneous SLE mice









* Lymph nodes weight includes superficial cervicals, axillary, brachial and inguinal lymph nodes.

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ATG-106

CDH6 x CD3 T Cell Engager
for Ovarian Cancer & Kidney Cancer

ATG-106: Globally First-in-class CDH6 x CD3 TCE 2.0 for the Treatment of Ovarian and Kidney Cancers

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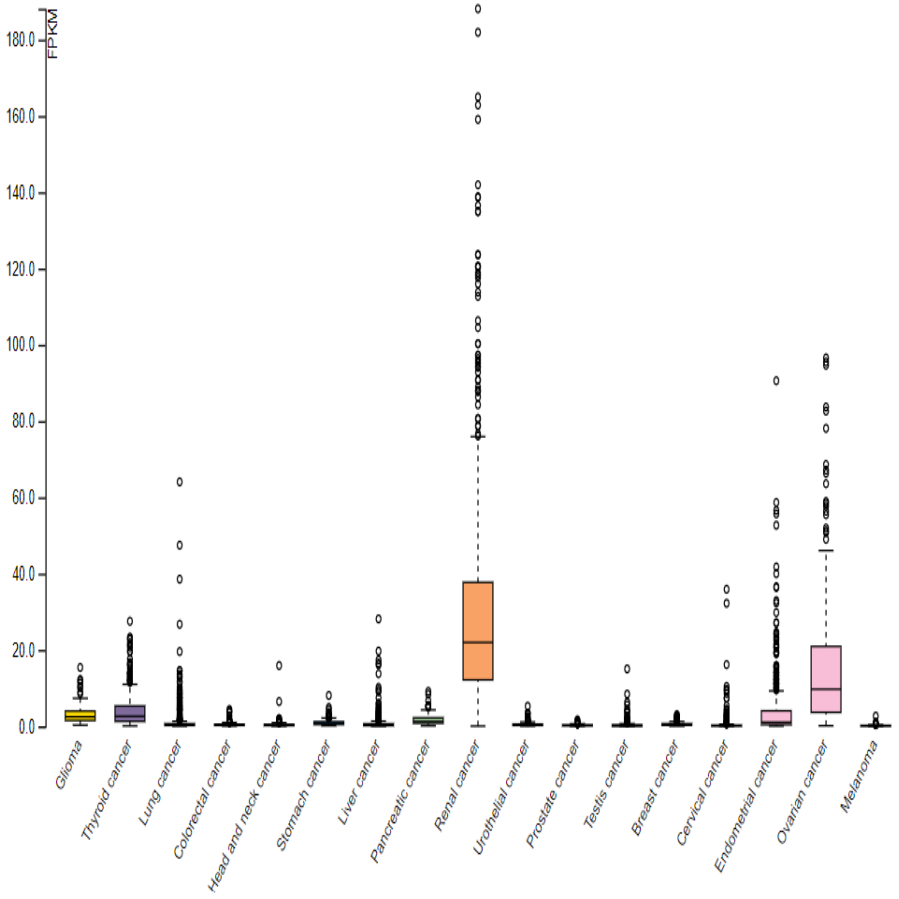
CDH6 is a TAA Highly Expressed in Solid Tumors Such as Ovarian Cancer, Renal Cancer, and Endometrial Cancer

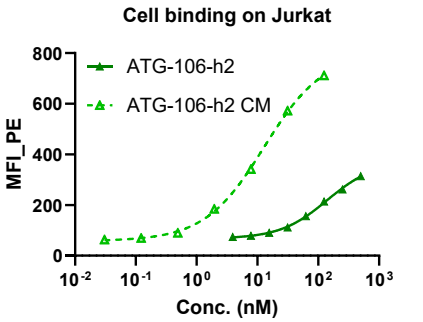
TCGA Data Set

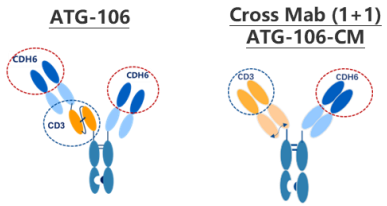


- **First-in-class Opportunity:** No CDH6 x CD3 TCE competitors in development yet
- **Compelling Preclinical Profile:** Demonstrated CDH6-dependent T cell activation, potent *in vitro* and *in vivo* anti-tumor efficacy, and good developability，well tolerated in NHP
- **IND Submission Timeline:** Planned for **Q1 2027**

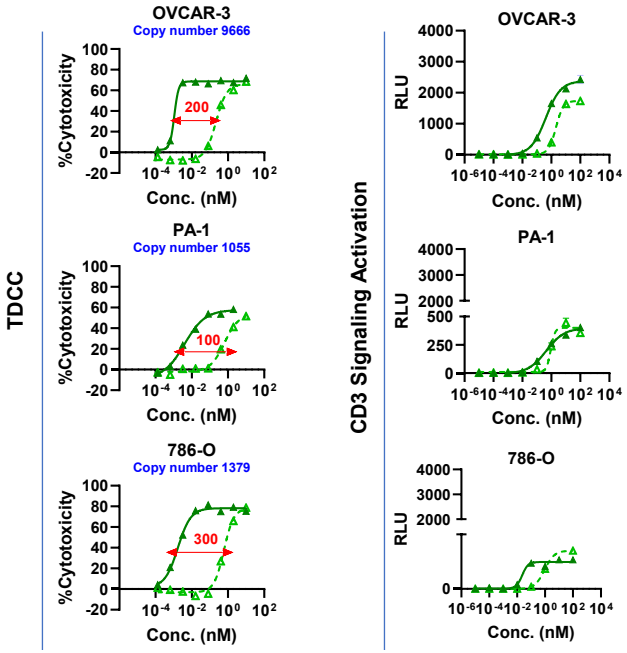
ATG-106 Has Reduced CD3 Binding in the Absence CDH6 and Enhanced Target-specific Cytotoxicity Compared with Crossmab 1+1 Format

Without CDH6

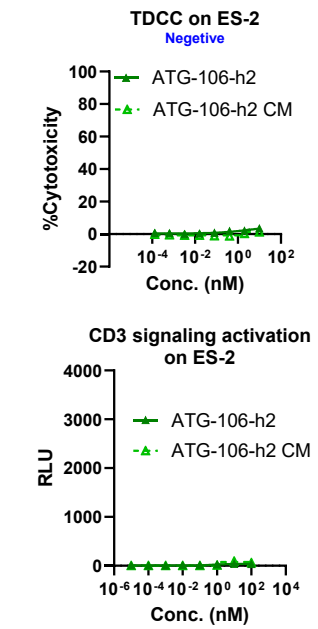




With CDH6



Without CDH6



Cadherin-6 (CDH6) TCE as a Promising Approach for Ovarian Cancer and Kidney Cancer

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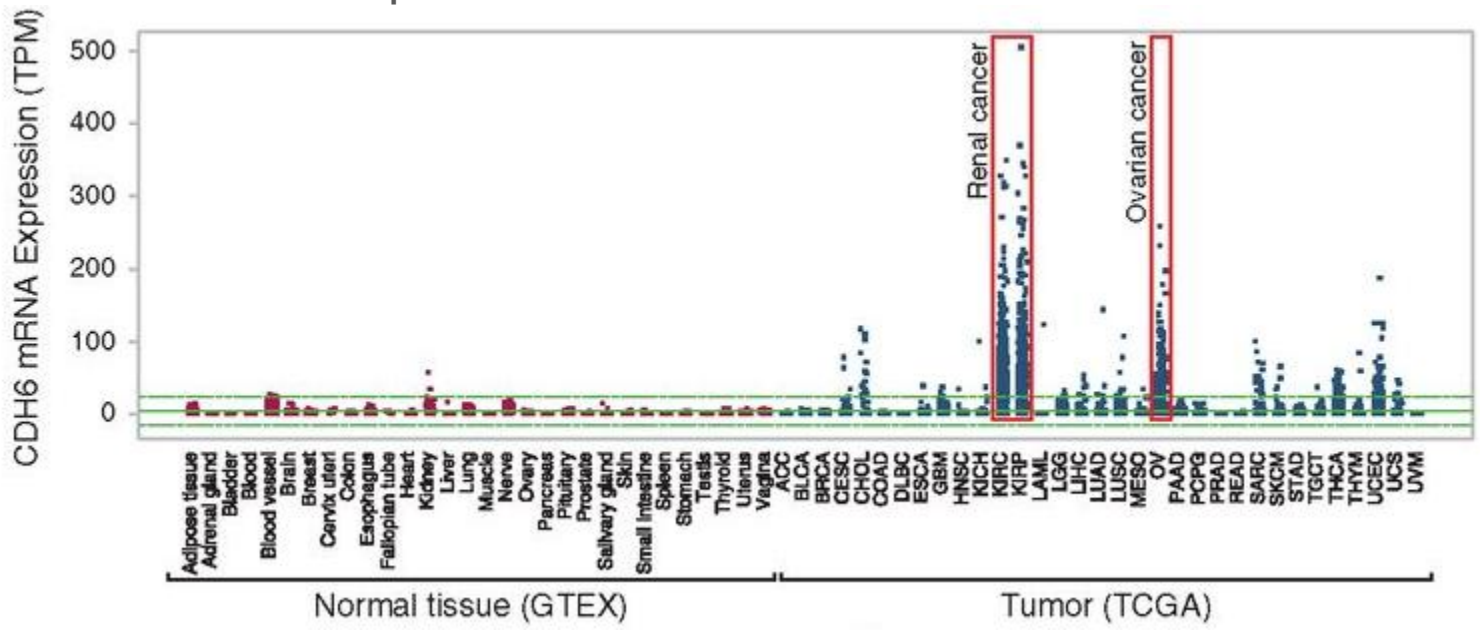
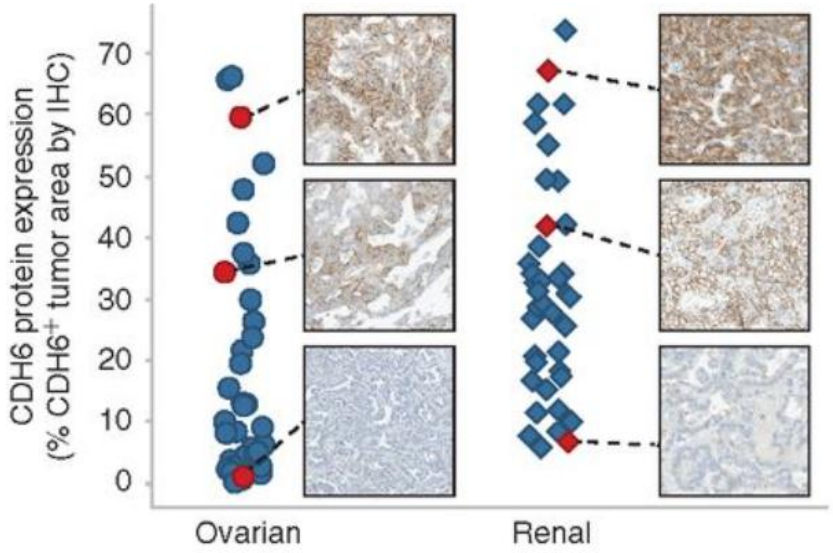
Cadherin-6 (CDH6)

- Encodes a type II classical cadherin from the cadherin superfamily. Involves in cell-cell adhesion and signal pathway, enhance epithelial–mesenchymal transition and promotes cell migration and invasion
- Positively expressed in **ovarian cancer (OV), kidney renal clear cell carcinoma (KIRC), kidney renal papillary cell carcinoma (KIRP)**, some other tumor types like thyroid cancer (**THCA**), cholangiocarcinoma (**CHOL**), hepatocellular cancer (**LIHC**), uterine corpus endometrial carcinoma (**UCEC**), lung squamous cell carcinoma (**LUSC**), lung adenocarcinoma (**LUAD**) and sarcoma (**SARC**)
- **Limited expression in normal tissue**

CDH6 mRNA Expression



CDH6 Protein Expression

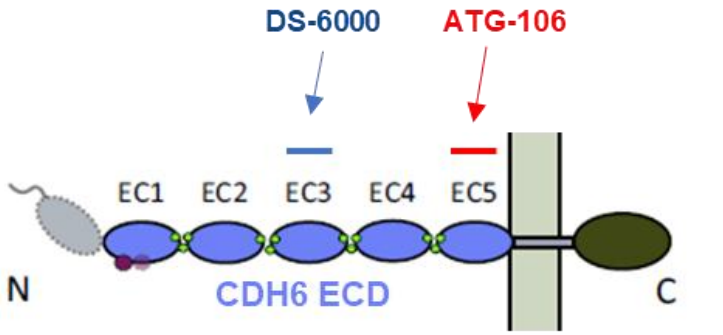


Carl U. Bialucha, et al.2017, Cancer Discovery.

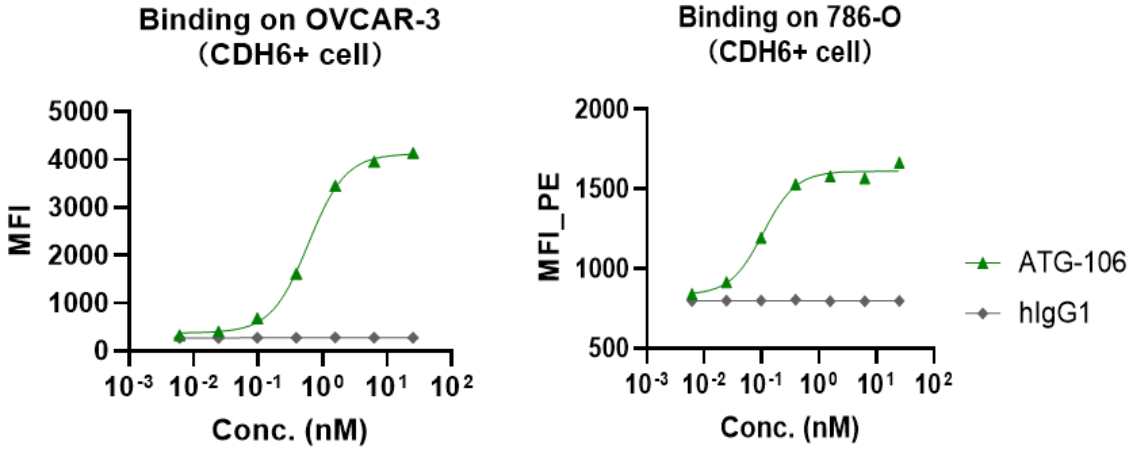
ATG-106 Bound to CDH6 with High Affinity

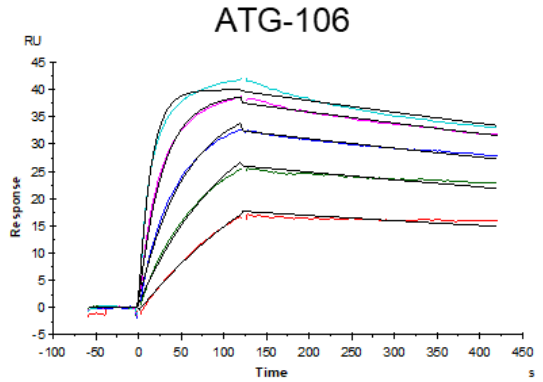
ANTENGENE

- ATG-106 showed **high binding affinity (sub-nM EC50) to CDH6 positive cells and protein, with EC5 as the binding epitope**
- **ATG-106 did not cross-react with other proteins in the CDH (Cadherin) family**



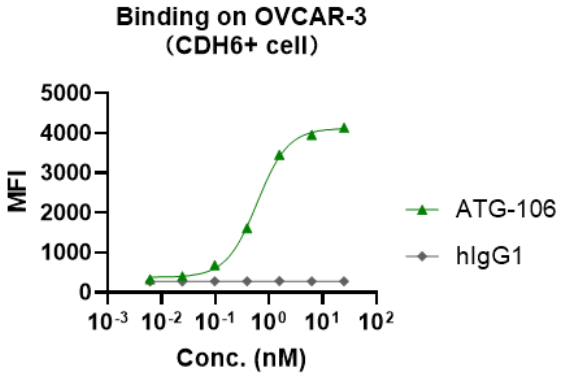
	CDH6 epitope
ATG-106	EC5
Bis-BMK	EC3

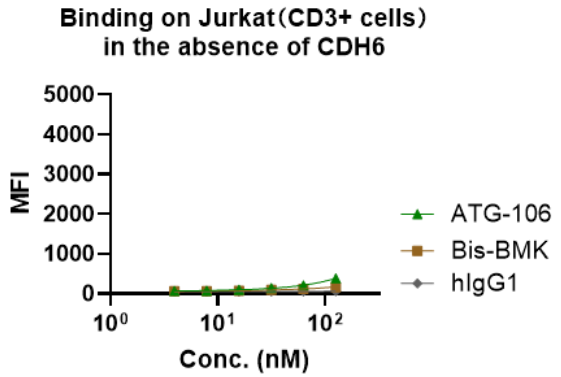


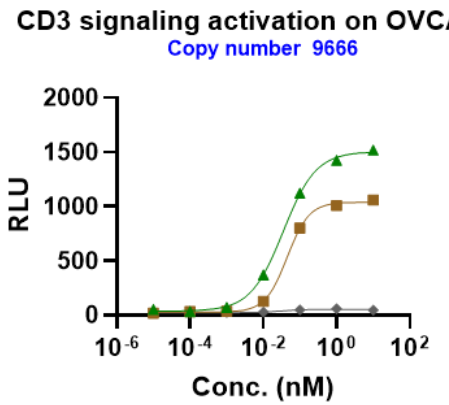


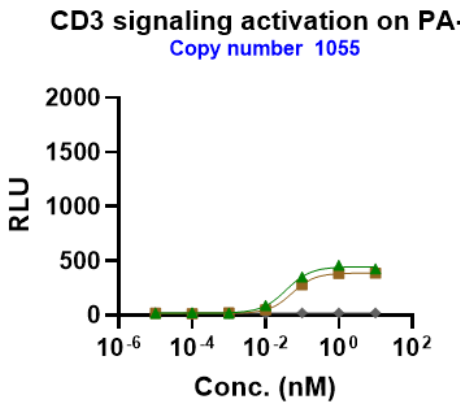
ATG-106 Binds To and Activates T Cells in a CDH6-dependent Manner

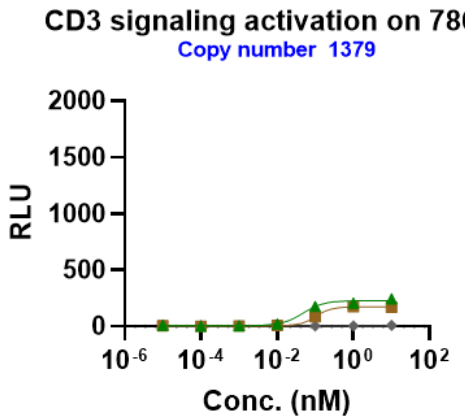
- ATG-106 (parental) and its humanized candidates (ATG-106 h1, ATG-106 h2, and ATG-106 h3) showed **high binding affinity (sub-nM EC50) to CDH6 positive cells**
- ATG-106 and ATG-106-h demonstrated **little binding to CD3+ cells before CDH6 crosslinking**, while they **potently activated CD3 signaling in the presence of CDH6 positive cells**

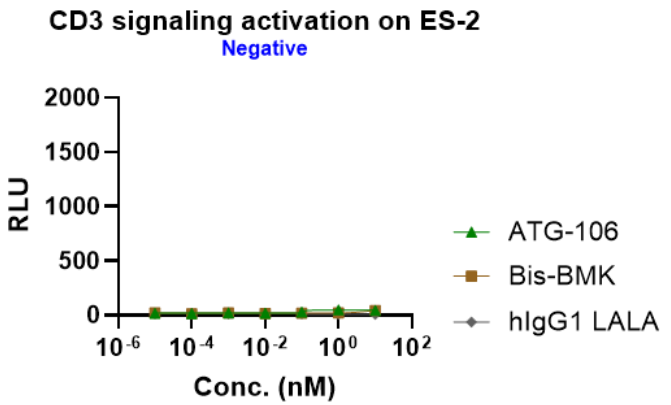








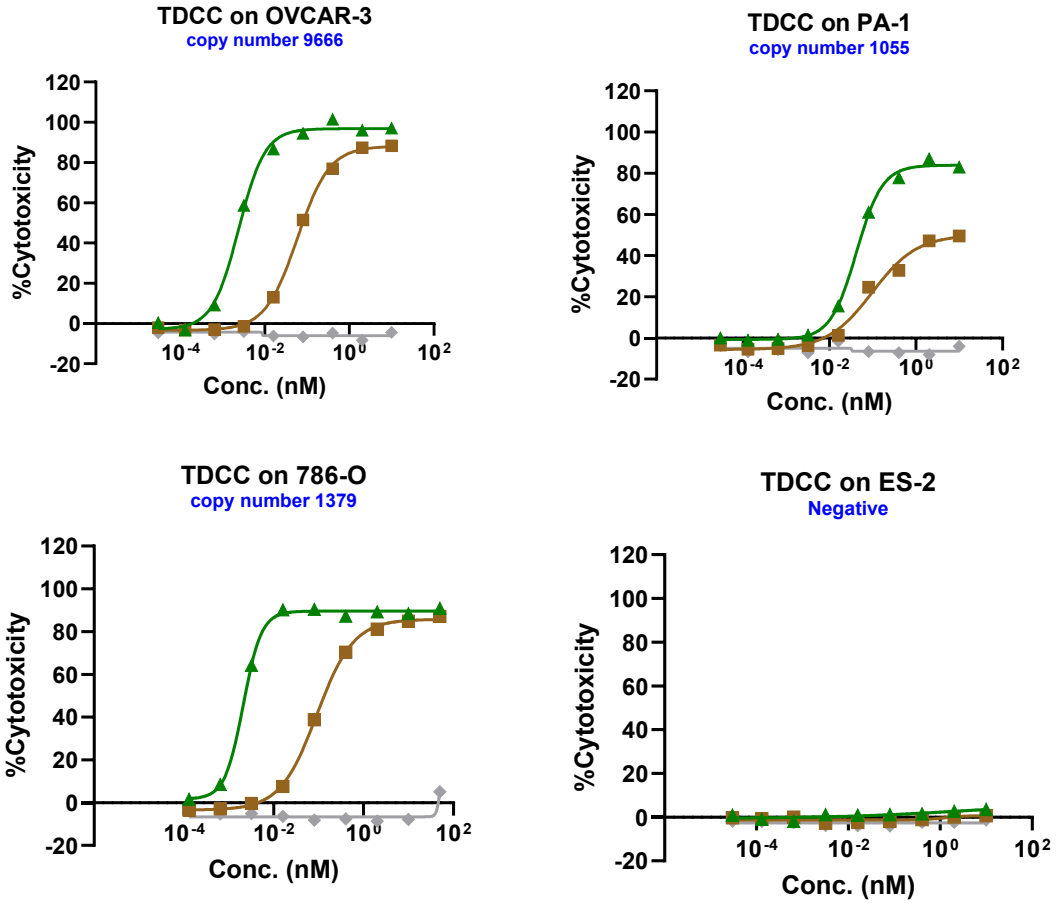


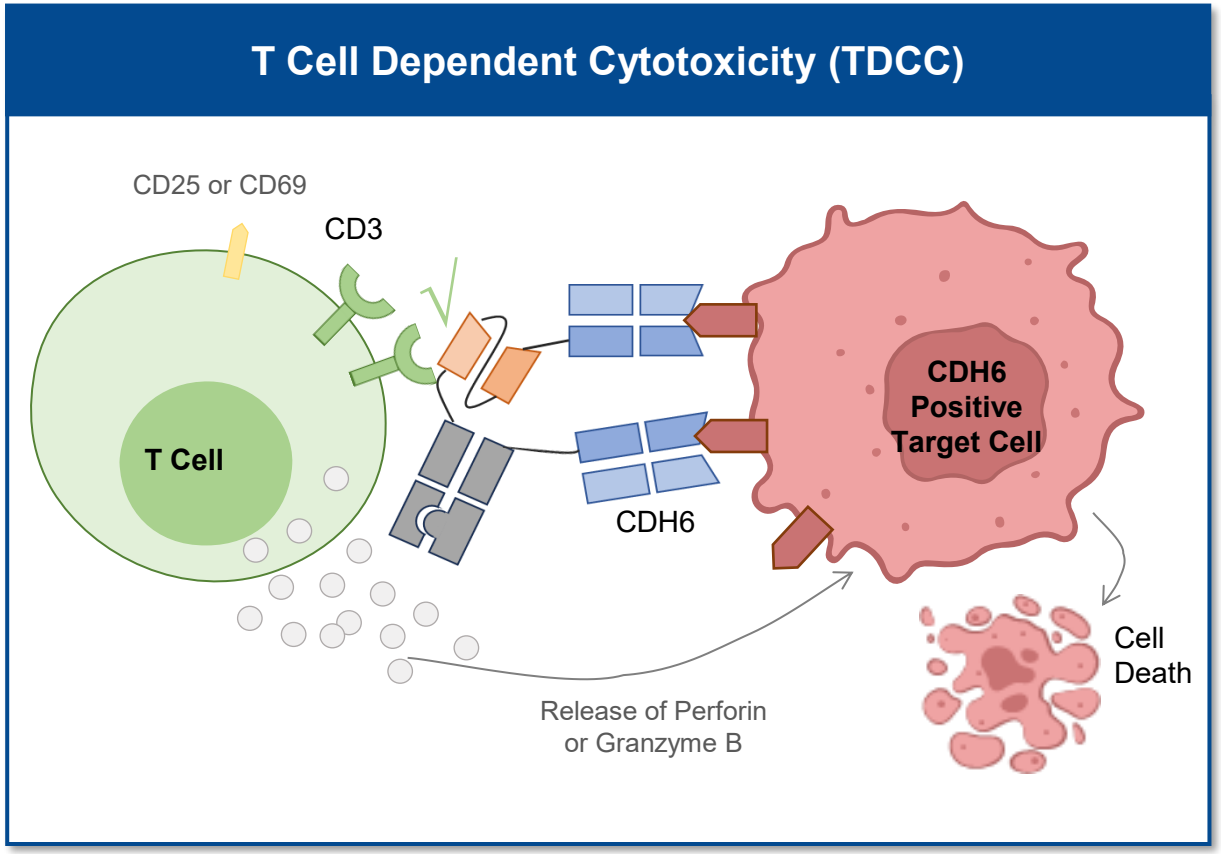


ATG-106 Exhibited Potent T Cell Dependent Cytotoxicity on CDH6+ Cells

ANTENGENE

- ATG-106 showed potent **T-cell dependent cytotoxicity (TDCC) against CDH6+ cells** (ovcar-3, PA-1 and 786-O)
- **No TDCC was observed** on CDH6 negative cells (ES-2)





ATG-106 (CDH6 x CD3 TCE) Exhibited Potent T Cell Dependent Cytotoxicity on Ovarian Cancer and Promising *In Vivo* Anti-tumor Activity

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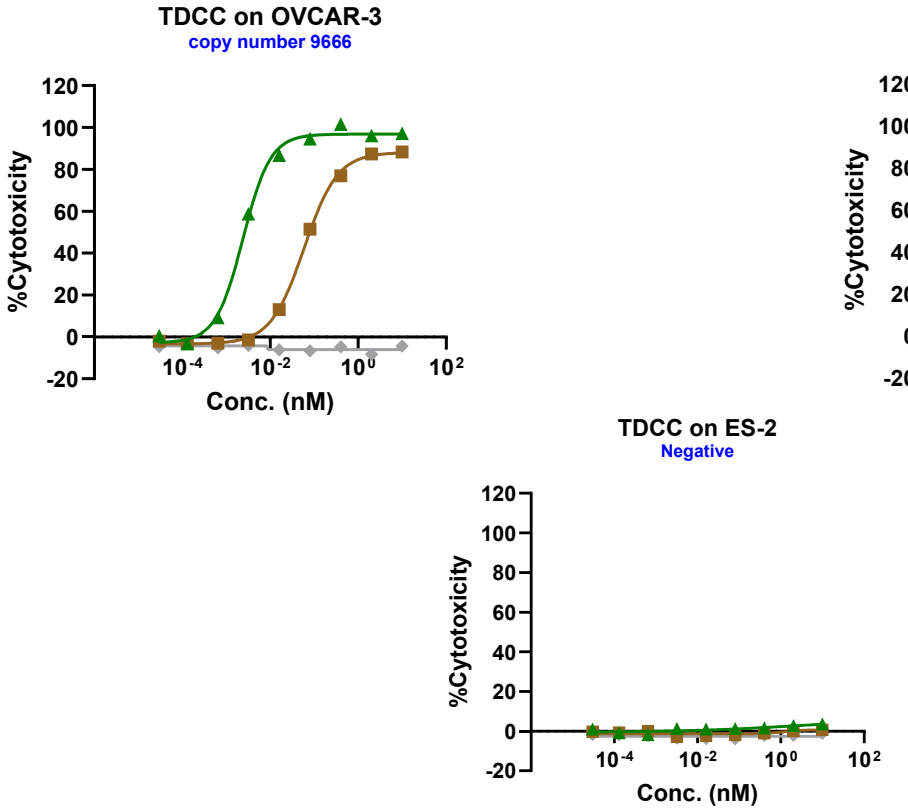
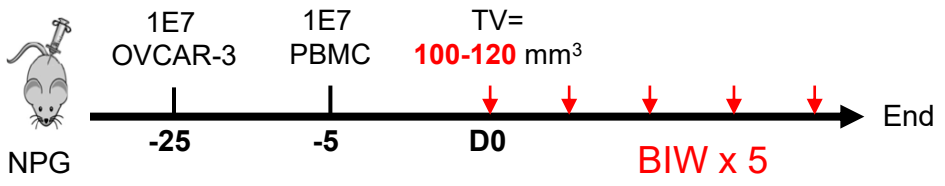
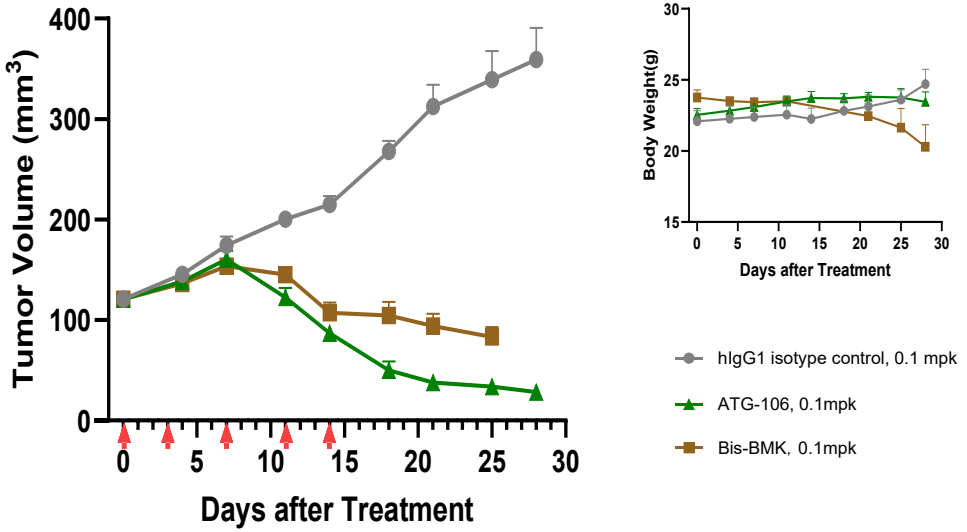
Potent T Cell Dependent Cytotoxicity on Ovarian Cancer Cell Line

- ATG-106 showed potent **T-cell dependent cytotoxicity (TDCC) against OVCAR-3 and PA-1** (CDH6+)
- **No TDCC was observed** on ES-2 (CDH6 negative)



Promising *In Vivo* Anti-tumor Activity





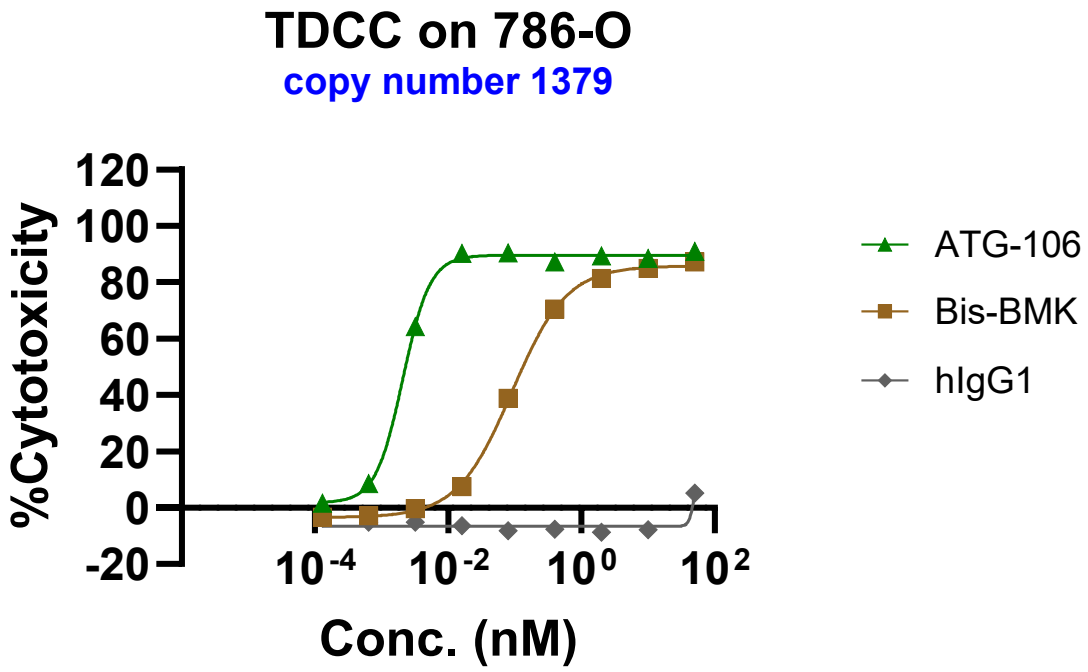
- ATG-106 demonstrated **potent *in vivo* efficacy in PBMC-humanized OVCAR-3 CDX mouse models**

ATG-106 (CDH6 x CD3 TCE) Exhibited Potent T Cell Dependent Cytotoxicity on Renal Cancer Carcinoma and Promising *In Vivo* Anti-tumor Activity

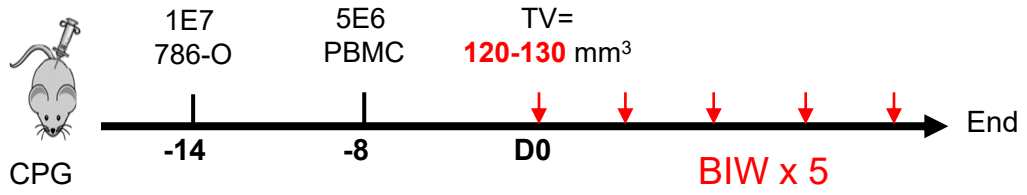
ANTENGENE

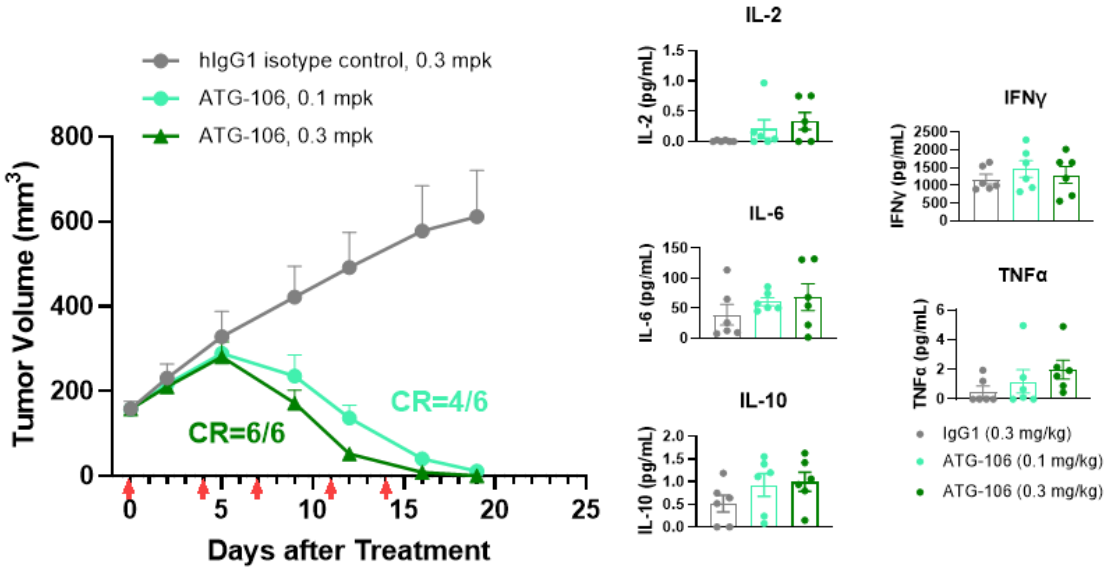
Potent T Cell Dependent Cytotoxicity on Renal Cell Carcinoma Cell Line

- ATG-106 showed potent **T-cell dependent cytotoxicity (TDCC) against 786-O cells** (CDH6 positive)



Promising *In Vivo* Anti-tumor Activity

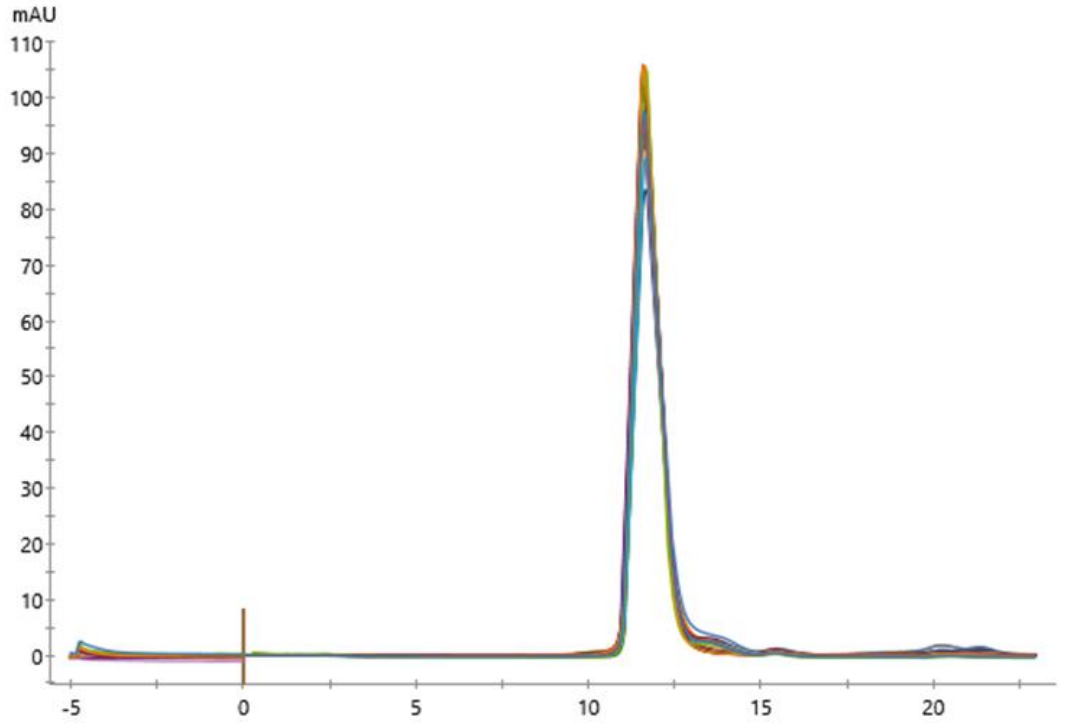




- ATG-106 demonstrated **potent *in vivo* efficacy, and low cytokines release in PBMC-humanized 786-O CDX mouse models**

ATG-106 Demonstrated Great Developability

- ATG-106 candidates demonstrated a melting temperature (Tm) of >68 ℃
- ATG-106 at 10 mg/mL showed **great stability under multiple stress conditions** (4 ℃ , 25 ℃ and 40 ℃ for 2 weeks and 4 weeks; Acceleration 3 days; Freeze-thaw 5 cycles)



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ATG-112

ALPPL2 x CD3 T Cell Engager for Gynecological Tumors and Lung Cancer

ATG-112: ALPPL2 x CD3 TCE 2.0 for the Treatment of Gynecological Cancer, Non-small Cell Lung Cancer and Pancreatic Ductal Adenocarcinoma

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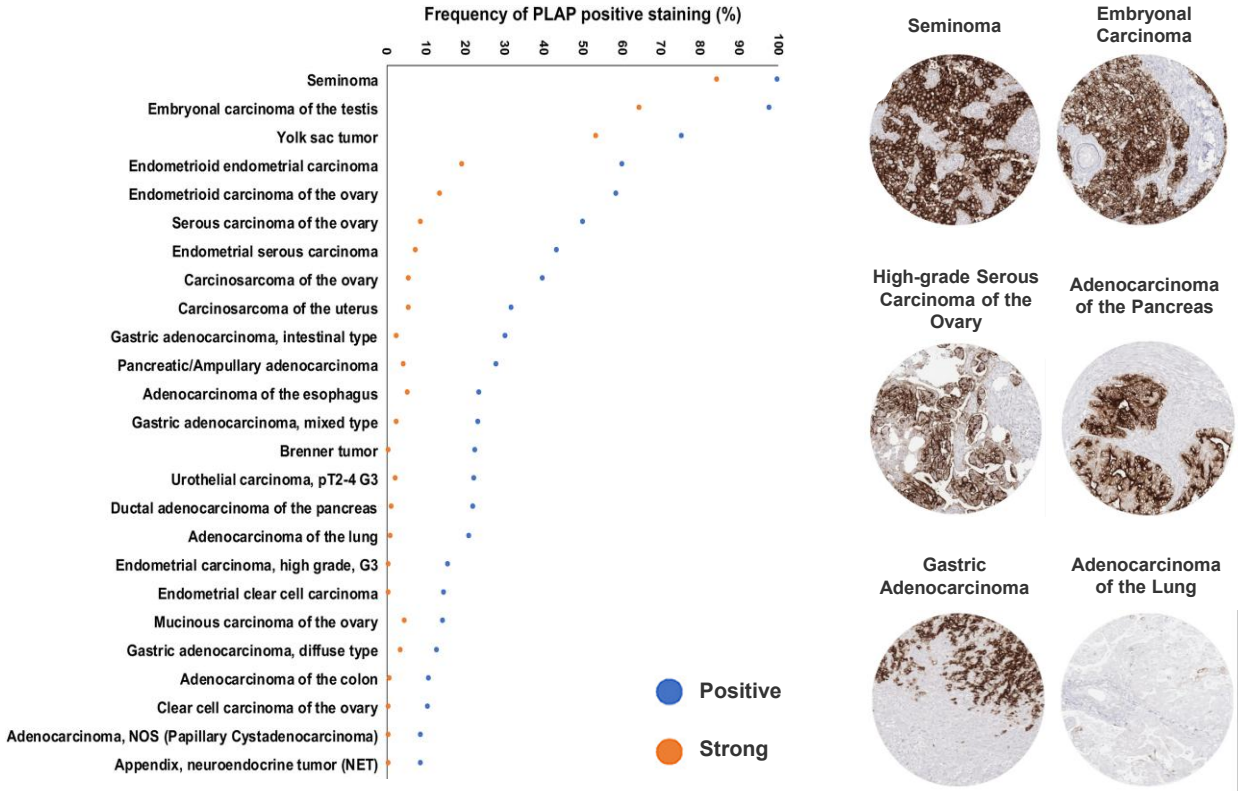
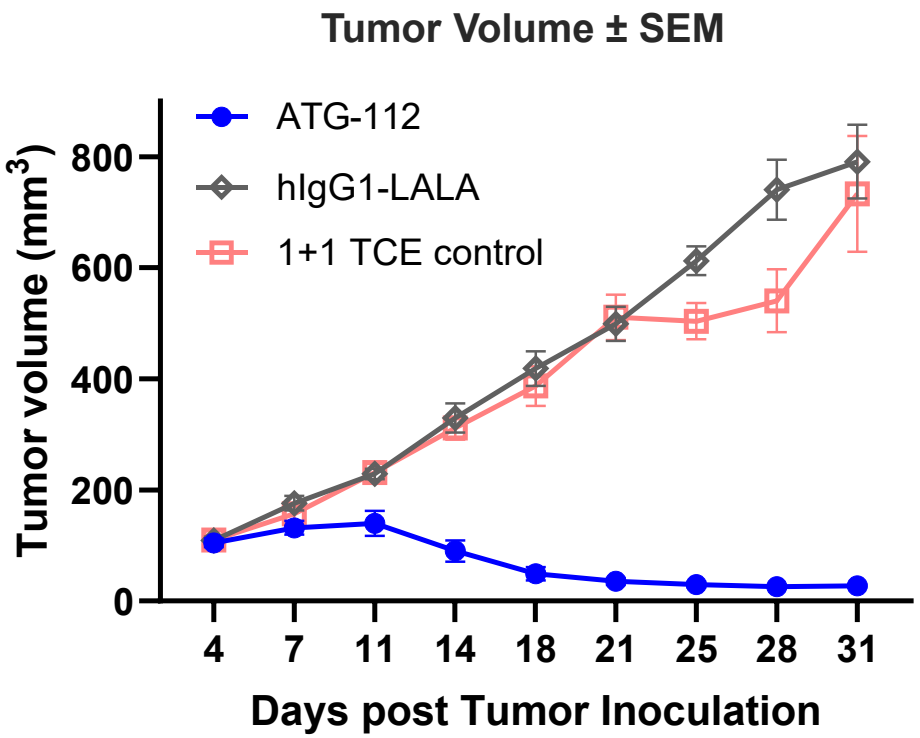
- **First-in-class Opportunity:** No ALPPL2 x CD3 TCE competitors in clinical-stage yet
- **Compelling Preclinical Profile:** Demonstrated ALPPL2-dependent T cell activation, potent *in vitro* and *in vivo* anti-tumor efficacy
- **PCC Nomination:** Planned for **Q1 2026**

ALPP/ALPG is Highly Expressed in Multiple Tumor Types with Restricted Normal Tissue Expression



ATG-112 Demonstrated Promising Pre-clinical Anti-tumor Efficacy



ALPP and ALPG: Promising Therapeutic Targets for Advanced Solid Tumors

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ALPP and ALPPL2: Closely Related GPI-Anchored Proteins with High Expression in Cancer

ALP (Alkaline Phosphatase)
- Membrane-bound glycoprotein that catalyzes the hydrolysis of phosphate monoesters at basic pH values, release inorganic phosphate
- GPI-anchored protein
- Dimeric enzymes

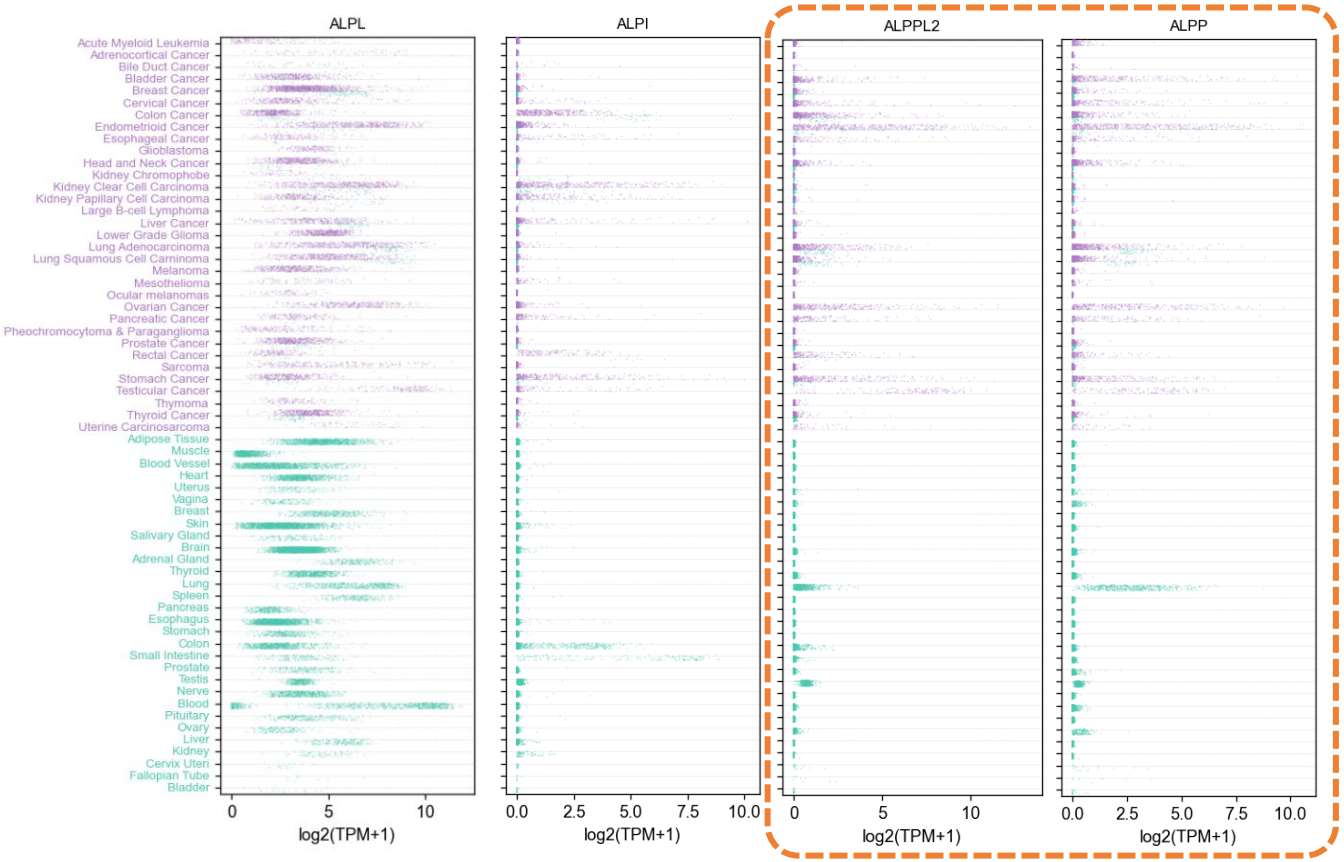
Human ALP Isoenzymes
ALPL (Tissue-nonspecific Alkaline Phosphatase)
- 57% homology with ALPPL2
- Expressed throughout the body, especially abundant in liver, bone, kidney

ALPI (Intestinal Alkaline Phosphates)
- 87% homology with ALPPL2
- High level expression in intestinal tissue

ALPP (Placental Alkaline Phosphatase), 95% Homology with ALPPL2
- High level in placenta
- High expressed in multiple cancers

ALPG (Germ Cell Alkaline Phosphates, Placental-like ALP), ALPPL2
- High level in placenta. Trace amounts in the testis and thymus
- High expressed in multiple cancers

ALPP and ALPG: Similar Gene Expression Patterns Across Tissues and Tumors



ALPP and ALPG: Promising Therapeutic Targets for Advanced Solid Tumors

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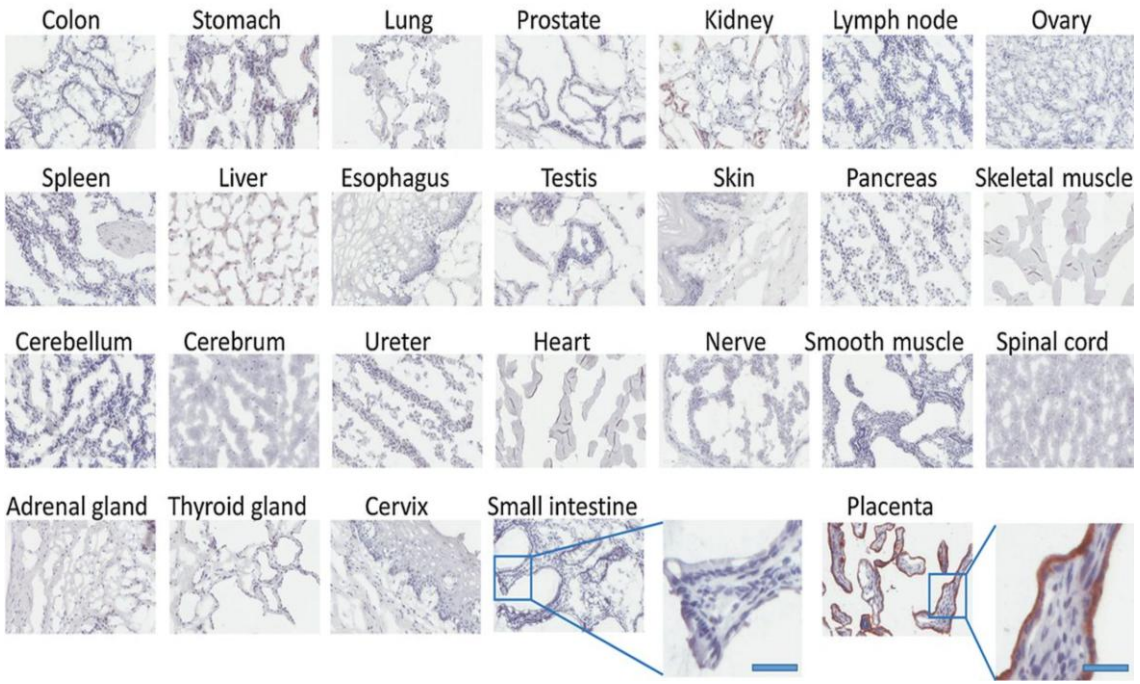
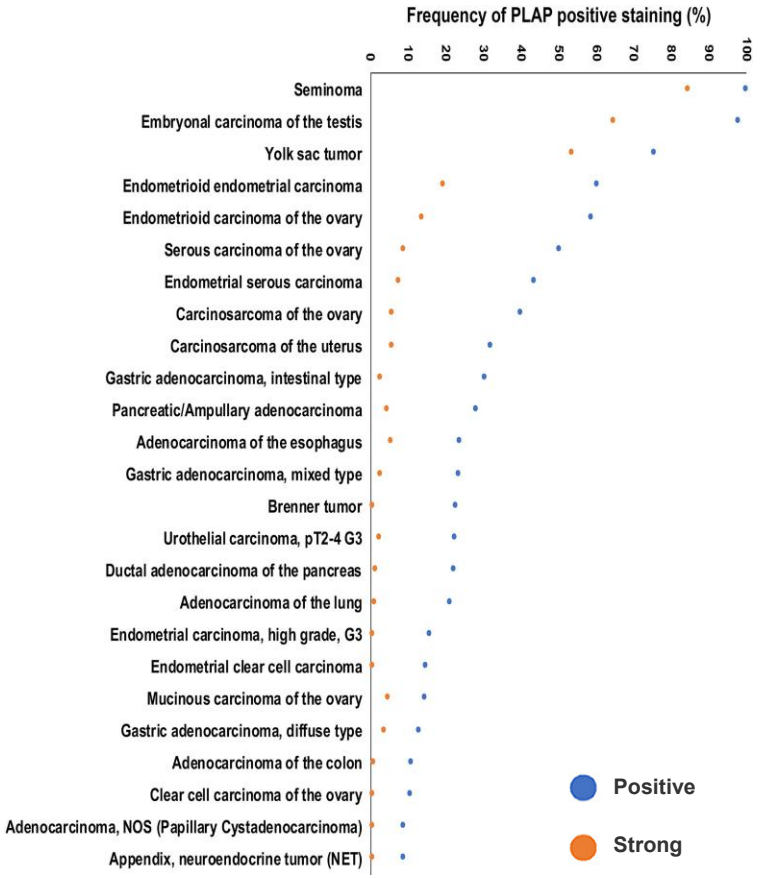
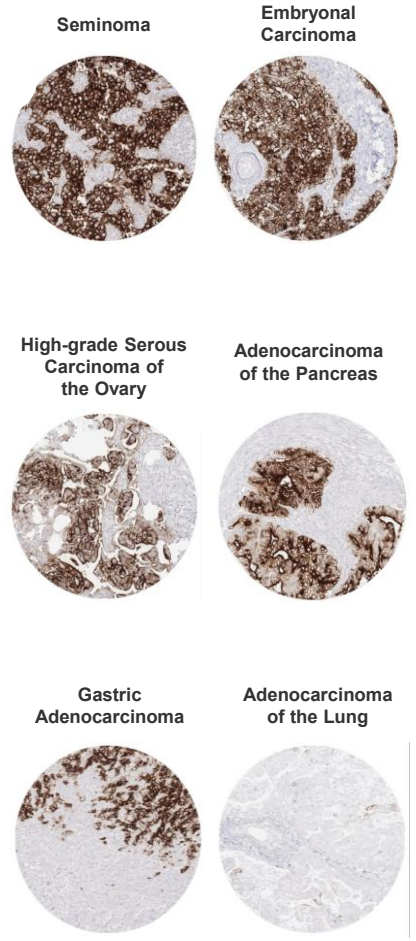
ALPP / ALPG in Normal Tissues



- The **restricted expression** of ALPP and ALPG **in normal tissues** and their **accessibility on the cancer cell surface** supports their potential as targets for cancer therapeutics

ALPP / ALPG in Cancer Tissues





The Expression of ALPP/G in Normal Tissue is Restricted to the Placenta

In-house Tissue Microarray Experiment

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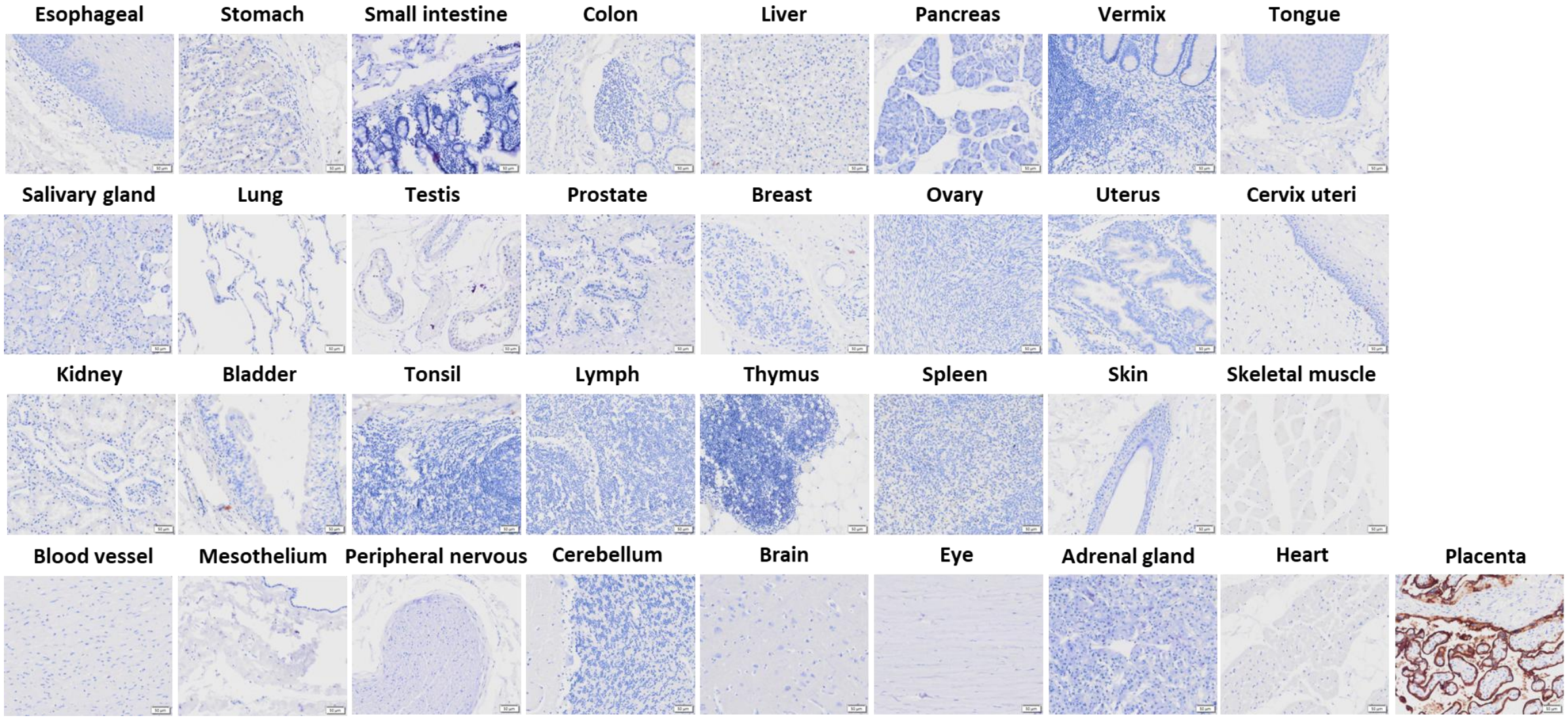

The Expression Profile of ALPP/G in Tumors: Microarray Experiment

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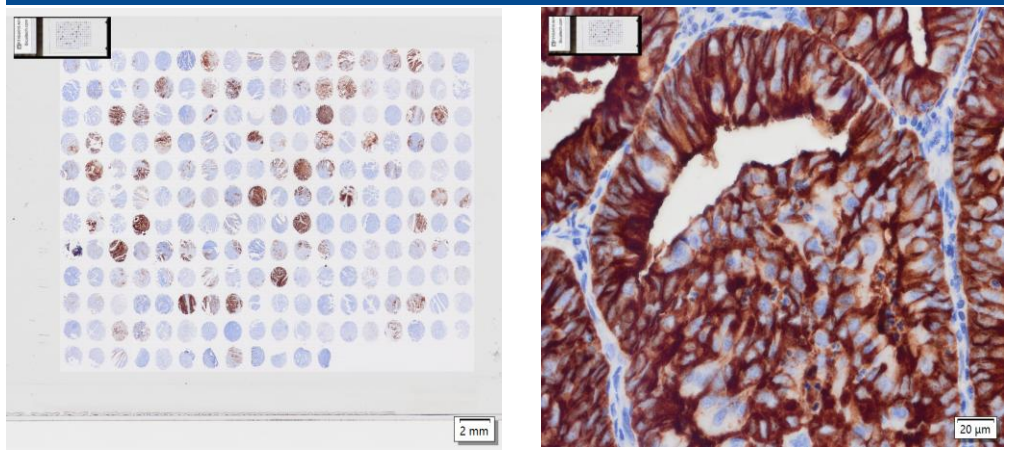
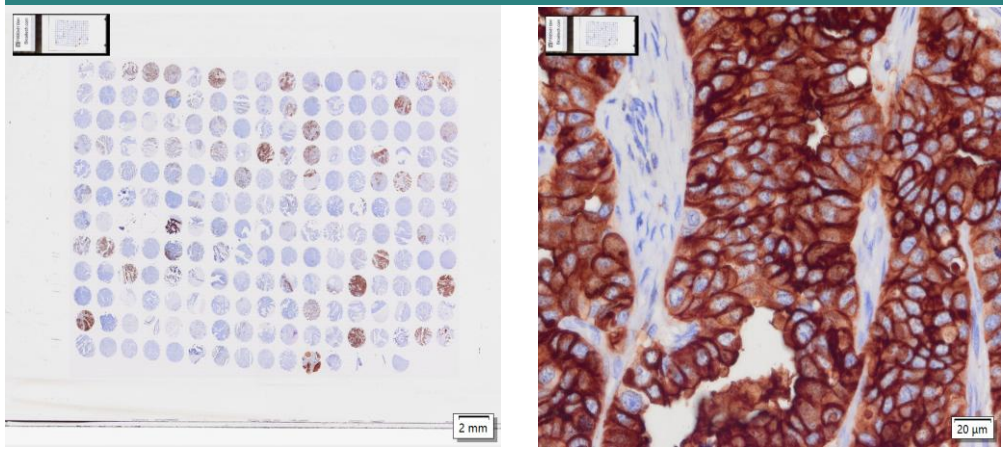
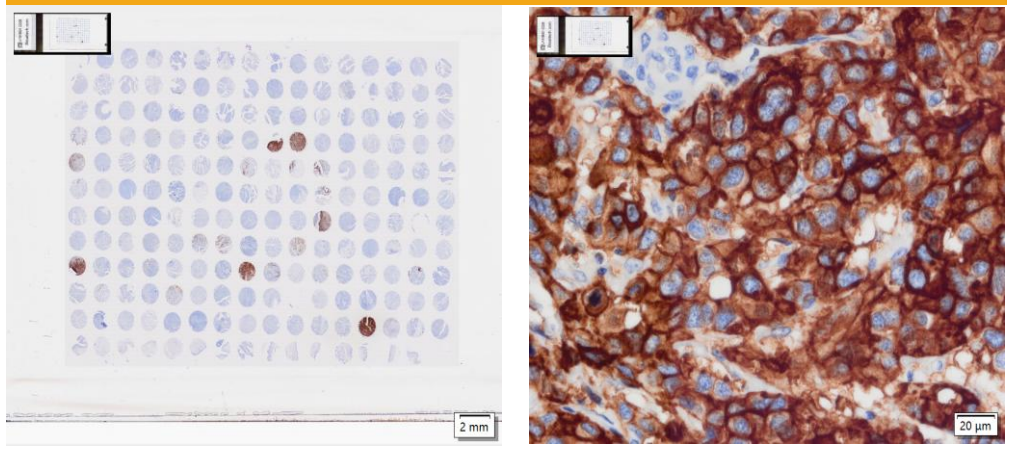
Endometrial Cancer – 58.50%



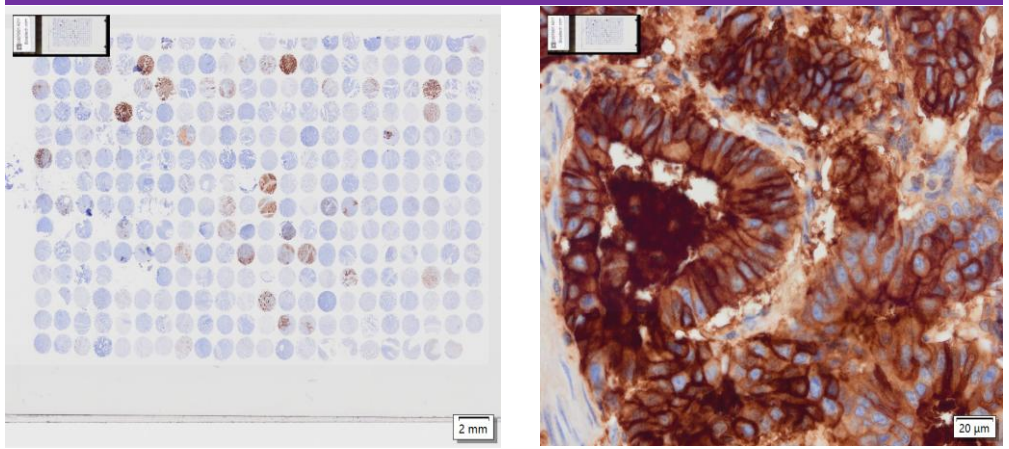
Ovarian Cancer – 51.01%



Bladder Cancer – 26.14%



Gastric Cancer – 26.53%



ATG-112 Demonstrated Promising *In Vivo* Anti-tumor Activity

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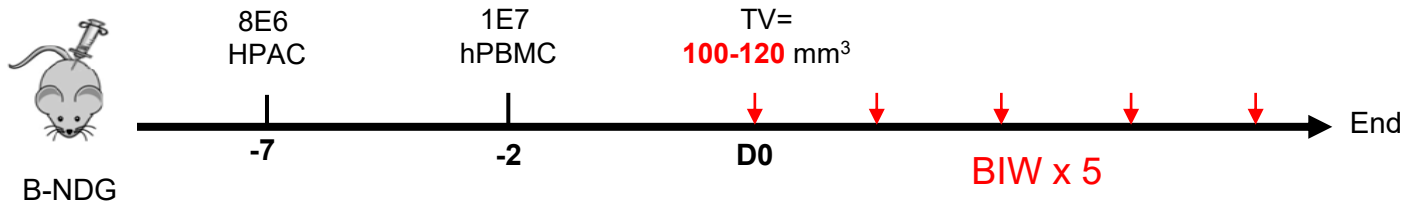
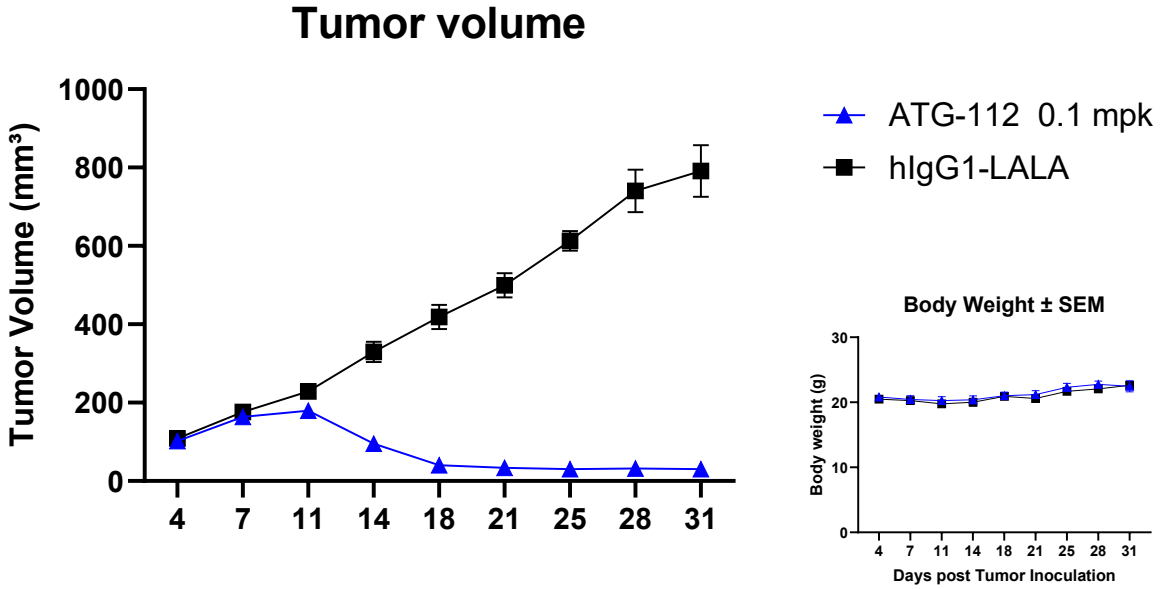
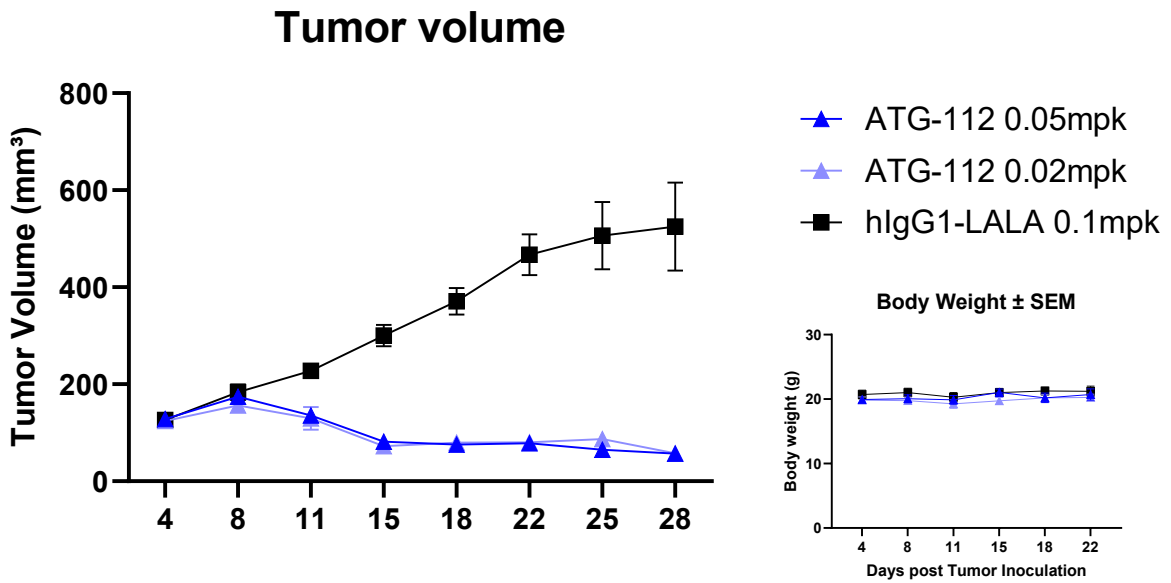
- ATG-112 achieved **potent tumor suppression** across various doses (0.1, 0.05, and 0.02 mg/kg)







ATG-112 Demonstrated Low Cytokine Release and Controllable CRS Risk

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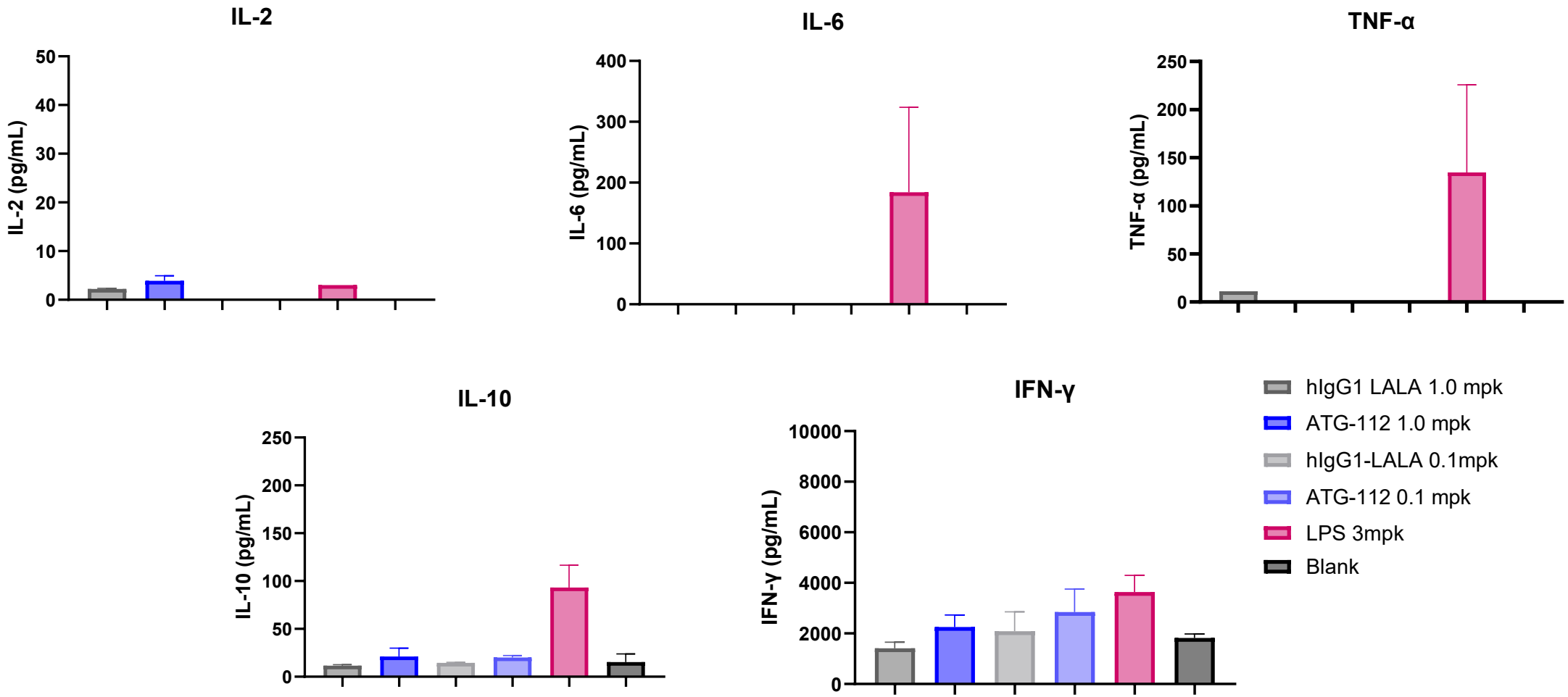
- ATG-112 candidates exhibited a **favorable in vivo safety profile**, characterized by **low cytokine release** and **controllable CRS risk**



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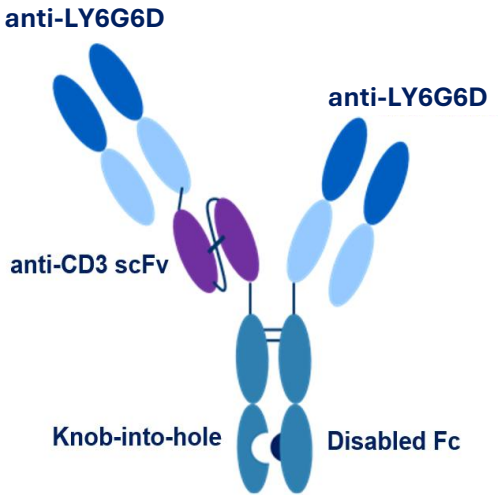
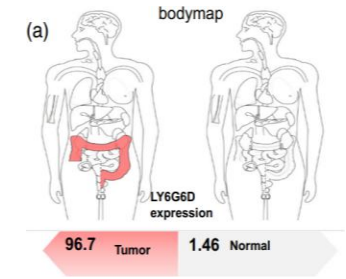
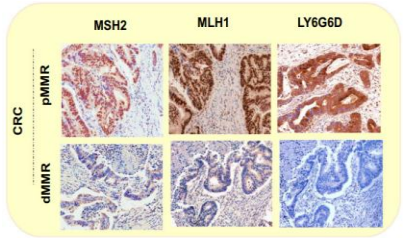
Other TCEs for Solid Tumors

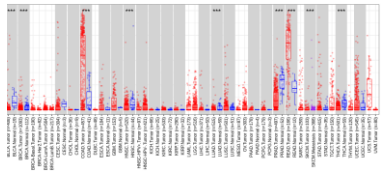
Other AnTenGager™ TCEs for Solid Tumors

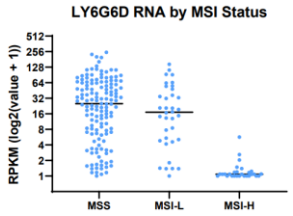
ATG-110: LY6G6D x CD3 TCE 2.0 for MSS Colorectal Cancer











- LY6G6D is a phosphatidylinositol (GPI)–anchored cell surface protein with **expression highly specific to colorectal cancer**
- LY6G6D has much higher expression level in colorectal cancer tissue compared to normal tissue, **predominantly in pMMR/MSS colorectal cancer which has primary resistance to ICI treatment**
- **ATG-110 demonstrated potent efficacy and good stability**
- **IND Submission:** Planned for H1 2027

Undisclosed AnTenGager™ TCE Programs

ATG-115

Undisclosed TAA Bispecific TCE for Liver Cancer

- ✓ Novel tumor associated antigen (TAA) **identified by AI + bioinformatics**
- ✓ **Highly expressed in liver cancer** with low normal tissue expression

2 Undisclosed Trispecific TCEs

- ✓ Targeting **metastatic castration-resistant prostate cancer (mCRPC)** and **small cell lung cancer (SCLC) / neuroendocrine tumors**, respectively
- ✓ **First-in-class Potential**
- ✓ **Enhancing efficacy with reduced toxicity**

Discovery and Pre-clinical Highlights

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Antibody Drug Conjugates (ADCs)

ATG-022 (CLDN18.2) *Phase II*	CLDN18.2+ Gastric Cancer (GC) and Other Solid Tumors	CLDN18.2 ADC with Efficacy Across the Widest Patient Population; BTD in GC
ATG-125 (B7-H3 x PD-L1) *Pre-clinical*	Solid Tumors	IO+ADC in One Drug
CD24 *Pre-clinical*	Solid Tumors	IO+ADC in One Drug

Immuno-Oncology (IO)

ATG-037 (CD73) *Phase Ib/II*	CPI-resistant Melanoma and Non-small Cell Lung Cancer	Oral Bioavailable; Demonstrated Efficacy in CPI-resistant Patients
ATG-101 (PD-L1 x 4-1BB) *Phase I*	Solid Tumors	No Liver Toxicity
ATG-031 (CD24) *Phase I*	Solid Tumors	First-in-class Myeloid Regulator

Autoimmune Diseases

ATG-201 (CD19 x CD3) *IND-enabling*	B Cell Driven Autoimmune Diseases	Deep B Cell Depletion with Low CRS
ATG-207 (Undisclosed Bifunctional Biologics) *Discovery*	T Cell Driven Autoimmune Diseases	First-in-Class; Induces T_{reg} and T Cell Exhaustion

T Cell Engagers (TCEs)

ATG-201 (CD19 x CD3) *IND-enabling*	B Cell Driven Autoimmune Diseases	Deep B Cell Depletion with Low CRS
ATG-106 (CDH6 x CD3) *Pre-clinical*	Ovarian Cancer and Kidney Cancer	First-in-Class CDH6 TCE
ATG-112 (ALPPL2 x CD3) *Pre-clinical*	Gynecological Tumors and Lung Cancer	First-in-Class ALPPL2 TCE
ATG-110 (LY6G6D x CD3) *Pre-clinical*	Microsatellite Stable (MSS) Colorectal Cancer	For IO-resistant Colorectal Cancer
ATG-021 (GPRC5D x CD3) *Pre-clinical*	Multiple Myeloma	
ATG-102 (LILRB4 x CD3) *Pre-clinical*	Acute Myeloid Leukemia and Chronic Myelomonocytic Leukemia	Biparatopic
ATG-107 (FLT3 x CD3) *Pre-clinical*	Acute Myeloid Leukemia	
ATG-115 (Undisclosed Bispecific TCE) *Pre-clinical*	Liver Cancer	Novel TAA Discovered by AI
Undisclosed Trispecific TCE *Discovery*	Metastatic Castration-resistant Prostate Cancer	First-in-Class
Undisclosed Trispecific TCE *Discovery*	Small Cell Lung Cancer and Neuroendocrine Tumors	First-in-Class

Next-Generation Therapies for B Cell- and T Cell-Driven Autoimmune Diseases

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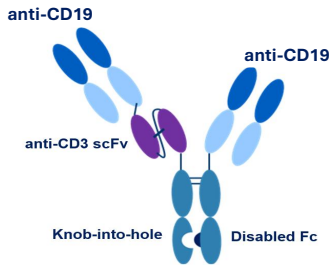
Antengene's Autoimmune Diseases Pipeline

B Cell Driven Autoimmune Diseases

ATG-201 – CD19 x CD3 TCE

Deeper and More Durable *In Vivo* B Cell Depletion with **Significantly Lower Cytokine Release** Compared to Benchmark

IND Targeting Q4 2025

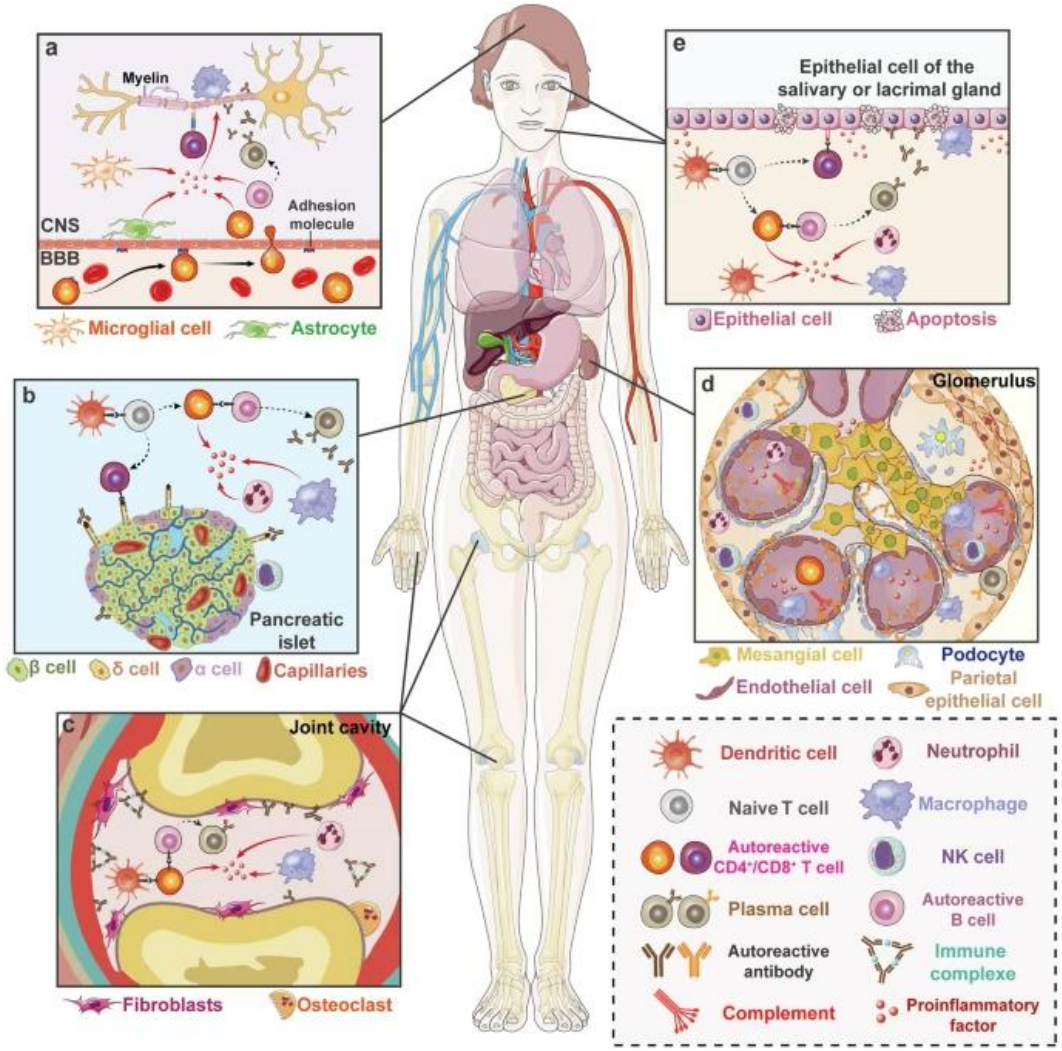


T Cell Driven Autoimmune Diseases

ATG-207 – First-in-Class Bifunctional Biologics

- Autoreactive T cells are known to cause autoimmune diseases like type 1 diabetes, rheumatoid arthritis, ankylosing spondylitis, and atopic dermatitis
- **ATG-207 is designed to induce strong T_{reg} differentiation and T cell exhaustion,** thereby alleviating T cell-related inflammation in autoimmune diseases and achieving therapeutic goals

Pre-clincal Data will be Presented in Key Conferences in 2026



Source: Signal Transduction and Targeted Therapy volume 9, Article number: 263 (2024)

Q&A Session

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Closing Remarks

Jay Mei, M.D., Ph.D.
Founder, Chairman, and Chief Executive Officer

Antengene: Well Positioned for Long-Term Growth with Expanding Revenue, Pivotal-Stage Pipeline, and Robust R&D Innovation

ANTENGENE

In-house Developed Drugs Entering Pivotal Trials

Multi-market Revenue Ramp Up

Robust R&D Engine Driving Novel Drug Innovation

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Thank You!

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