Adlai Nortye
NASDAQ: ANL



Corporate Presentation

September 2026

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Our product candidates are in preclinical or at various stages of clinical development. Our assumptions regarding the development potential of these product candidates are based in large part on preclinical data or early clinical readouts, and such data may not be predictive of future clinical outcomes. We may observe materially different results as we conduct further preclinical studies and any planned or ongoing clinical trials. Preclinical findings, including pharmacology, target engagement, biomarkers, efficacy, tolerability, safety, and translational data, may not be replicated in humans. Our development activities are subject to significant risks and uncertainties, including potential delays in the initiation, enrollment, data readout, and completion of preclinical studies and clinical trials, and the possibility of unexpected adverse events or insufficient efficacy, which may delay or prevent regulatory approval or commercialization. In addition, comparisons of product candidates between the Company and third parties based on preclinical and clinical data, are inherently limited due to differences in methodologies and models, experimental conditions, study design and patient populations, such comparisons should be interpreted with caution.

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A clinical-stage biotechnology company targeting cancer from multiple angles

3 **Three Lead Oncology Assets.**

2 **Two Innovative RAS Modalities.**

1 **One Next-Generation IO.**

1 **One Mission.** To transform deadly cancer into a chronic and eventually curable disease.

R&D and Operational Effectiveness

- Global footprint with operations in United States, Singapore, and China
- In-house R&D team has developed a library of 1,000+ RAS(ON)-targeting molecules
- Proprietary RASiCA™ (RAS Inhibitor Conjugated Antibody) platform

Financial Positioning

- ✓ $232M in cash and cash equivalents at the end of 1H26
- ✓ Expected to fund operations through 2028

Advancing first-in-class and potential best-in-class molecules:



AN9025
A **potential best-in-class** pan-RAS(ON) inhibitor oral small molecule



AN4035
A **first-in-class** pan-RAS(ON) inhibitor ADC, with CEACAM5 targeting



AN8025
A **potential first-in-class**, multi-functional T cell / APC modulator

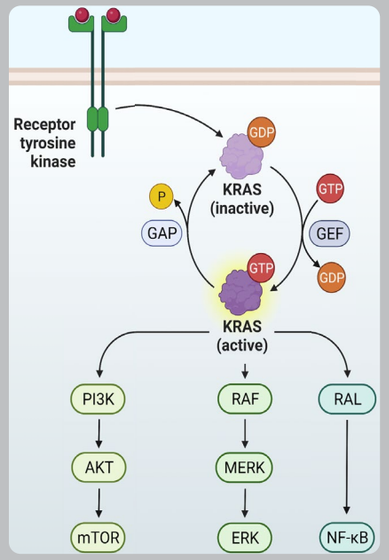
Oncology Therapeutics Clinical Pipeline

Product	MOA	Indication	Discovery	Phase 1a	Phase 1b	Phase 2	Phase 3	Upcoming Milestone	Worldwide Rights
AN9025	**pan-RAS(ON) inhibitor**	RAS-addicted tumors						Ph1 dose escalation data readout in 1H27	Ex-China[1]
AN4035	**CEACAM5 pan-RAS(ON) inhibitor ADC**	CEACAM5-enriched RAS-addicted solid tumors						Ph1 dose escalation data readout in 2H27	Global
AN8025	**αPD-L1 / Lag3v / CD86v**	Advanced tumors						Ph1 dose escalation completion by YE26	Global
Other Assets									
Palupiprant (AN0025)	**EP4 antagonist**	Neoadj RC (+CRT)[2]						Enrollment completed, topline results in 1H27	Global except Japan, Korea, Taiwan and Southeast Asian Countries
		LA EC (+CRT)[2]						Enrollment completed, clinical update in 1H27	

MOA, mechanism of action; Lag3v, Lag 3 variant; CD86v, CD86 variant; IND, Investigational New Drug; RC, rectal cancer; CRT, chemoradiation therapy; LA, locally advanced; EC, esophageal cancer
Note 1. ASK Pharm holds exclusive rights to develop, manufacture, and commercialize AN9025 in mainland China, Hong Kong and Macau; 2. Investigator-Initiated Trials.

Multiple Opportunities to Address Large Unmet Need in RAS-Addicted Cancer

RAS Mutations Drive Oncogenic Signaling



- Mutations of the RAS gene (KRAS, HRAS, or NRAS) impair normal GTP hydrolysis, favoring the GTP-bound ”on” state
- Persistent RAS activation drives downstream signaling, promoting uncontrolled proliferation and tumor growth



Pancreatic Ductal Adenocarcinoma
Total U.S. incidence: ~60k/year
~90% RAS-mutant



Non-Small Cell Lung Carcinoma
Total U.S. incidence: ~195k/year
~30% RAS-mutant



Colorectal Cancer
Total U.S. incidence: ~150k/year
~45% RAS-mutant

$9B+

The projected TAM for 1L mPDAC alone is estimated at over $9B

175k+

The total incidence of RAS-mutant tumors in the U.S. is estimated to be over 175,000, for just PDAC, NSCLC, & CRC

RAS mutations drive tumor growth across multiple solid tumor types & represent a multi-billion-dollar opportunity for our pan-RAS pipeline

AN9025	AN9025 is well-positioned to address the broad RAS-addicted tumor market, including PDAC, NSCLC, CRC, as well as additional tumor types, with either a QD or QW dosing regimen
AN4035	AN4035 is well-positioned to address the CEA-enriched RAS-addicted tumor market, especially CRC, with additional targeting opportunities from our ADC platform, RASiCA™

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Sources: RAS Signaling Pathways, BPS Bioscience; American Cancer Society, Cancer Facts & Figures 2024; NCI SEER Database; Waters AM, et al. Cold Spring Harb Perspect Med. 2018; Foundation Medicine/COSMIC KRAS-mutant NSCLC frequency; ACS CRC incidence estimates; RAS Mutations in Advanced Colorectal Cancer review (PMC, 2024). TAM estimate: Cantor Fitzgerald Research estimates. Patient figures are approximate annual U.S. incidence, illustrative of relative opportunity size, not precise market sizing.



AN9025

Oral Small-Molecule Pan-RAS(ON) Inhibitor

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AN9025: Potential Best-in-Class Pan-RAS(ON) Inhibitor

AN9025 was designed by our in-house R&D team to address a broad range of RAS mutations across multiple tumor types

Improved *in vitro* potency compared to RMC-6236

- ≈30x increase in biochemical potency
  - 4x higher affinity for CypA driven by slower dissociation rate
  - 3~8x higher affinity for tri-complex formation
- ≈100x increase in cytotoxicity

Increased *in vivo* efficacy compared to RMC-6236

- Deeper *in vivo* tumor regression across multiple CDX models
- Delayed emergence of adaptive resistance

Best-in-class potential supported by preclinical profile

- Extended *in vivo* $T_{1/2}$, and prolonged anti-tumor response following treatment cessation, supporting QW dosing
- Doubling of animal MTD by total weekly dose with intermittent dosing (QW vs QD)

Potential for differentiated dosing schedule

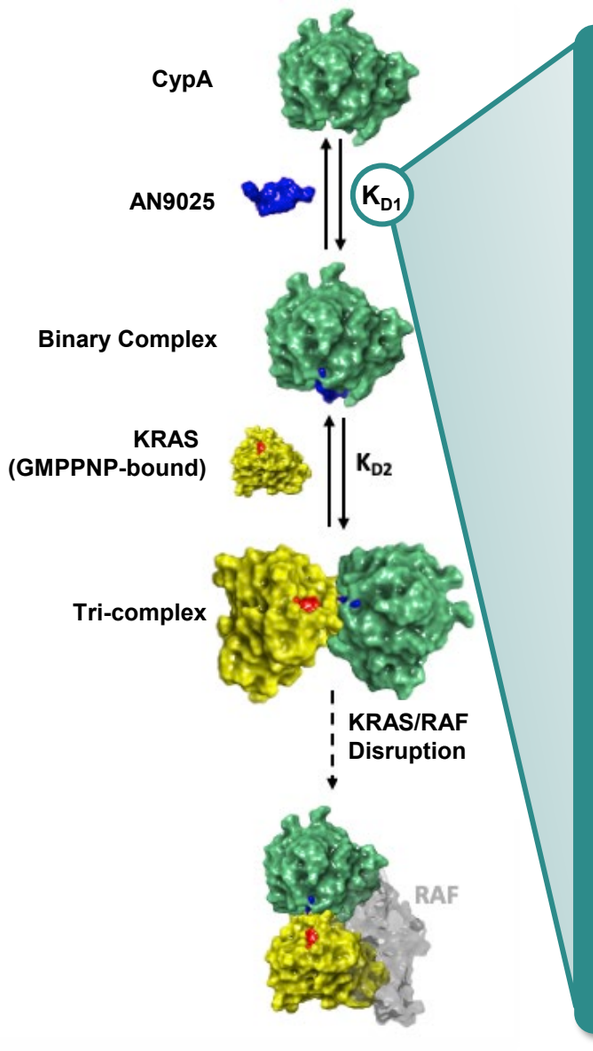
- Favorable baseline TI: comparable or slightly superior TI compared to daraxonrasib with QD dosing
- Potential for improved TI: QW regimen expanded the therapeutic index in animals

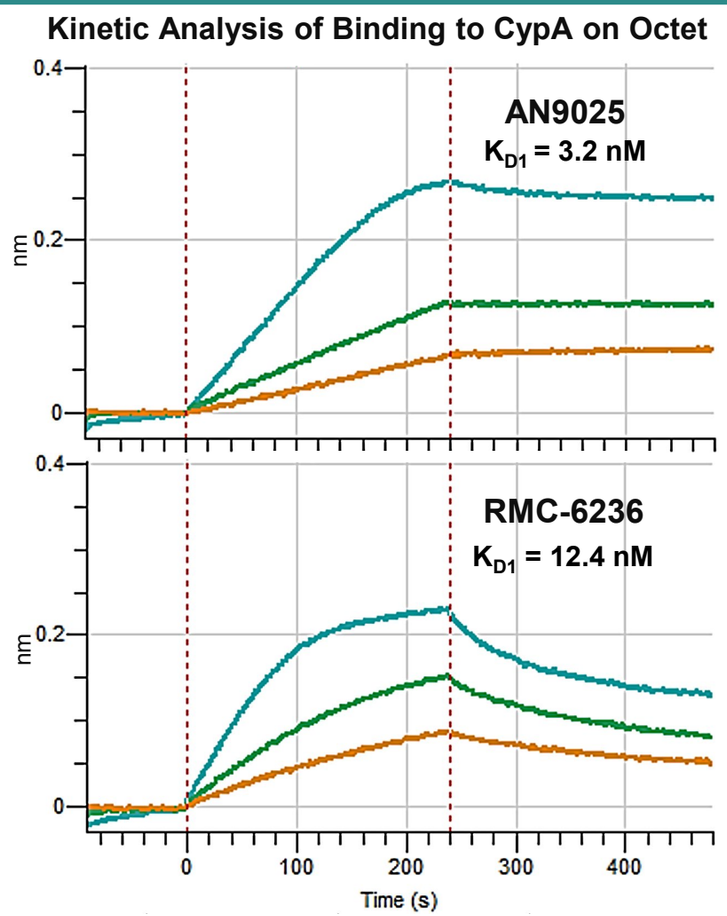
Better therapeutic index

Strong Global IP Position & Ex-China Global Rights Retained

CypA, cyclophilin A; QD, daily dosing; QW, weekly dosing; TI, therapeutic index; $T_{1/2}$, half life

Higher CypA Binding Affinity & Slower Dissociation



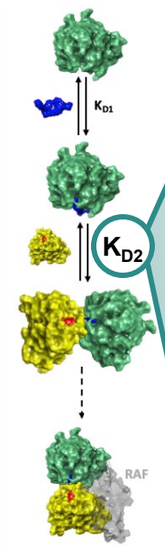


Binary Affinity	AN9025	RMC-6236
K_{D1} (nM)	**3.2**	12.4
k_a (1/[M*S])	**1.6×10^5**	2.8×10^5
k_{dis} (1/s)	**5.0×10^{-4}**	3.4×10^{-3}

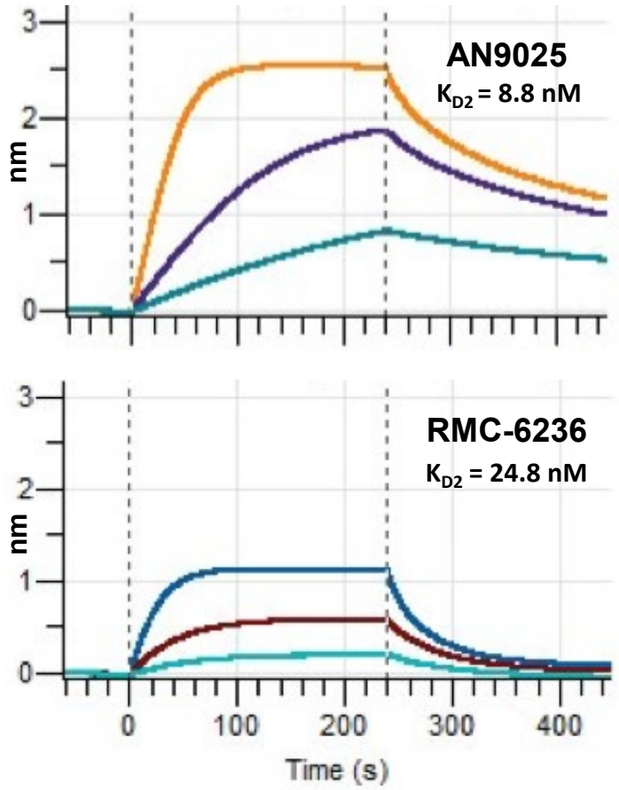
- AN9025 demonstrates a binding affinity to CypA that is **4-fold stronger** than that of RMC-6236
- AN9025 has a **7-fold slower dissociation rate** from CypA compared to RMC-6236, potentially allowing for sustained RAS inhibition
- **Taken together, these suggest that AN9025 may offer more sustained RAS inhibition**

CypA, cyclophilin A; k_a, association rate constant; K_D, equilibrium dissociation constant; k_{dis}, kinetic dissociation rate constant
Source: AN9025 Poster, Abstract #4377, 2025 AACR Annual Meeting

Stronger RAS(ON) Binding Affinity & Slower Dissociation



Kinetic Analysis of Binding to RAS(ON)
(GMPPNP-bound $KRAS^{G12D}$)



Mutant RAS(ON) Protein	AN9025			RMC-6236		
	K_D (nM)	**k_a (1/[M*S])**	**k_{dis} (1/s)**	K_D (nM)	k_a (1/[M*S])	k_{dis} (1/s)
$KRAS^{G12D}$	**8.8**	**4.9 x 10^5**	**4.3 x 10^{-3}**	24.8	5.4 x 10^5	1.3 x 10^{-2}
$KRAS^{G12V}$	**8.3**	**3.2 x 10^5**	**2.7 x 10^{-3}**	68.4	1.8 x 10^5	1.2 x 10^{-2}
$KRAS^{G12C}$	**4.5**	**4.1 x 10^5**	**1.9 x 10^{-3}**	17.8	4.5 x 10^5	8.0 x 10^{-3}
$KRAS^{G12S}$	**12.6**	**1.6 x 10^5**	**2.0 x 10^{-3}**	48.5	1.9 x 10^5	9.1 x 10^{-3}
$KRAS^{G12R}$	**15.5**	**2.4 x 10^5**	**3.8 x 10^{-3}**	80.5	2.3 x 10^5	1.8 x 10^{-2}
$KRAS^{G12A}$	**11.9**	**2.1 x 10^5**	**2.5 x 10^{-3}**	60.3	1.8 x 10^5	1.0 x 10^{-2}
$KRAS^{G13D}$	**15.5**	**2.5 x 10^5**	**3.9 x 10^{-3}**	69.7	2.5 x 10^5	1.8 x 10^{-2}
$NRAS^{Q61H}$	**11.0**	**2.5 x 10^5**	**2.8 x 10^{-3}**	45.4	2.2 x 10^5	1.0 x 10^{-2}
$KRAS^{WT}$	**10.0**	**2.8 x 10^5**	**2.8 x 10^{-3}**	52.8	2.2 x 10^5	1.2 x 10^{-2}
$NRAS^{WT}$	**6.6**	**3.7 x 10^5**	**2.4 x 10^{-3}**	33.9	2.6 x 10^5	8.8 x 10^{-3}
$HRAS^{WT}$	**4.0**	**4.9 x 10^5**	**2.0 x 10^{-3}**	20.2	3.4 x 10^5	6.8 x 10^{-3}

- AN9025 exhibits a **3- to 8-fold higher** binding affinity for tri-complex formation compared to RMC-6236, primarily attributed to slower dissociation rate leading to prolonged RAS signaling suppression
- AN9025 demonstrates **strong binding affinity** for the RAS(ON) protein, with no detectable binding to the RAS(OFF) protein

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k_a, association rate constant; K_D, equilibrium dissociation constant; k_{dis}, kinetic dissociation rate constant
Source: AN9025 Poster, Abstract #4377, 2025 AACR Annual Meeting

Significantly Higher Potency with Comparable Sensitivity Profile

AN9025 demonstrates **~100-fold more potent inhibition of cell proliferation** than RMC-6236 across RAS mutant cell lines

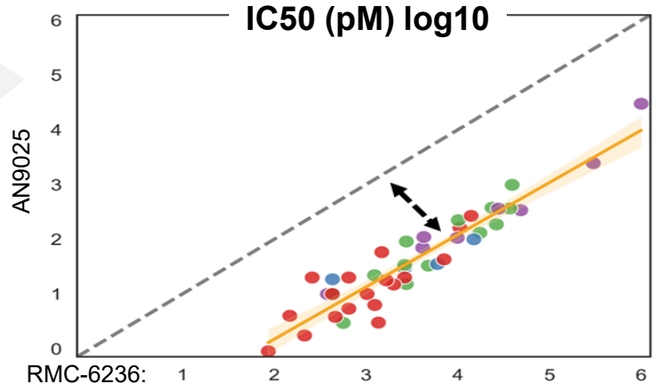
n=43 screening cell lines

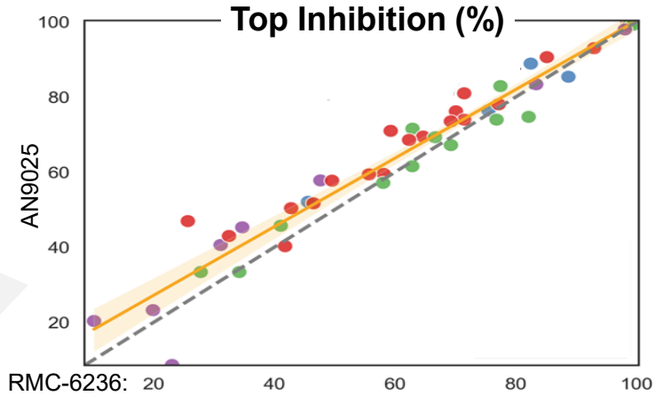
$y = 0.95 x - 1.74$
$r = 0.94$
$R^2 = 0.88$
$p = 2.70 \times 10^{-20}$

pan-RAS
- G12X
- G13X
- Q61X
- Wild Type

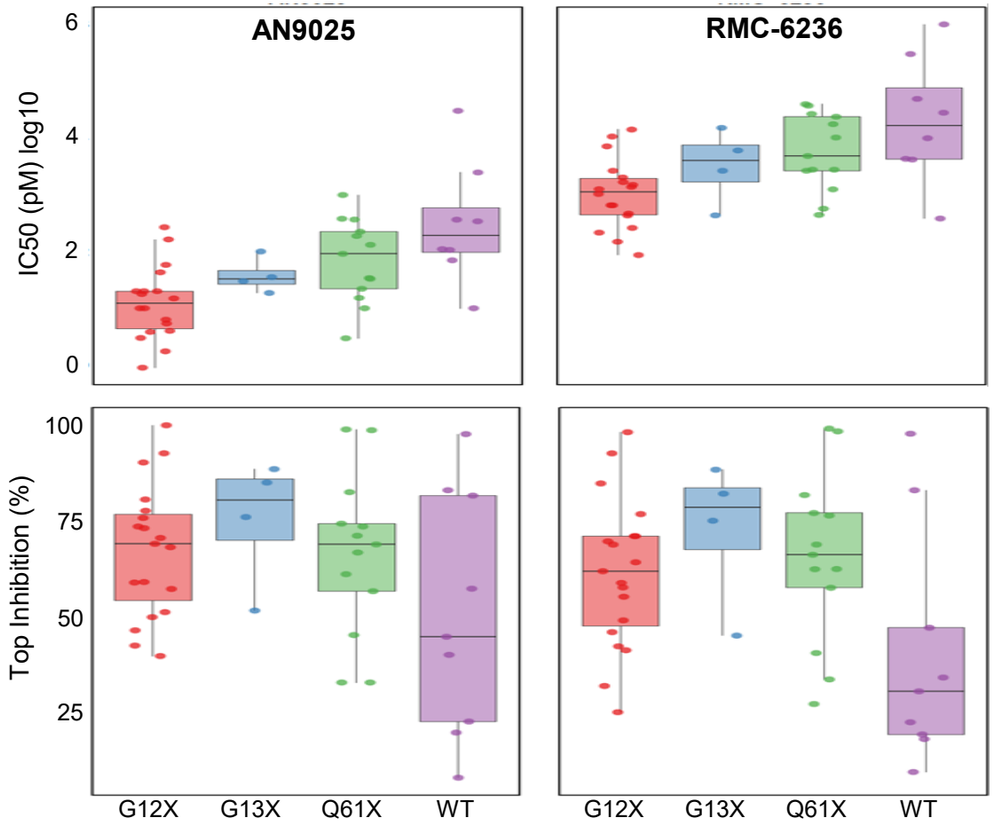
n=44 screening cell lines

$y = 0.91 x + 8.93$
$r = 0.97$
$R^2 = 0.94$
$p = 4.17 \times 10^{-27}$



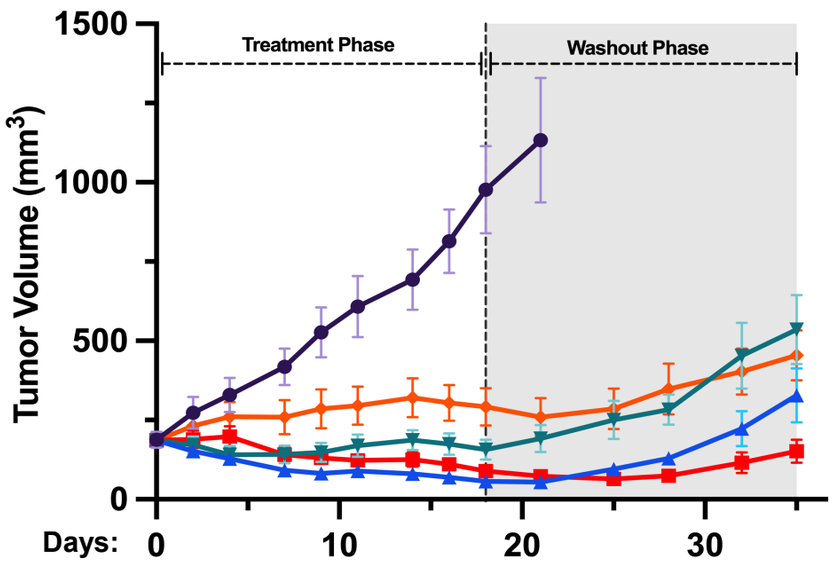


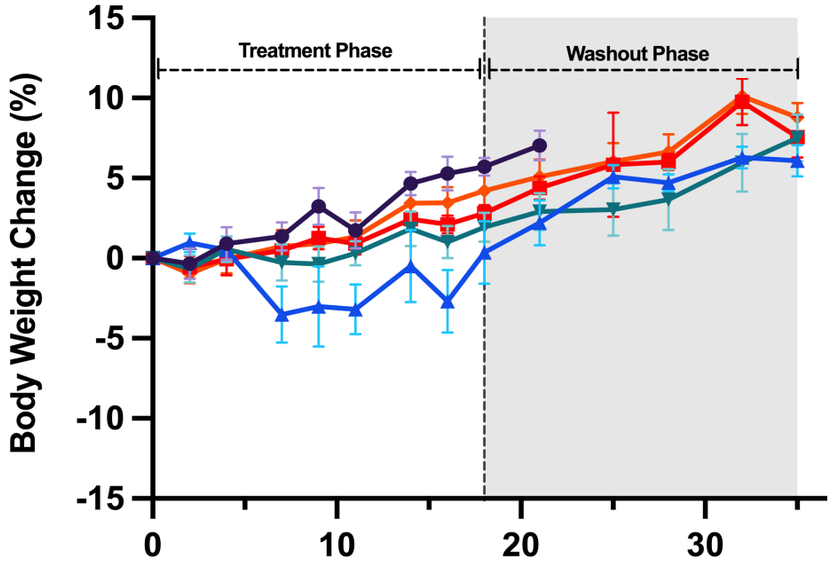
AN9025 shares a **similar RAS mutant sensitivity pattern** as that of RMC-6236 [i.e. G12X > G13X ≈ Q61X > WT]



IC50, half maximal inhibitory concentration; pM, picomolar; WT, wild-type
Source: AN9025 Poster, Abstract #4377, 2025 AACR Annual Meeting

Deep Tumor Regression & Sustained Anti-Tumor Effect





HPAC model (KRAS-G12D)

- Vehicle (p.o., QD)
- AN9025 (0.1 mg/kg, p.o., QD)
- AN9025 (0.04 mg/kg, p.o., QD)
- RMC-6236 (25 mg/kg, p.o., QD)
- RMC-6236 (10 mg/kg, p.o., QD)

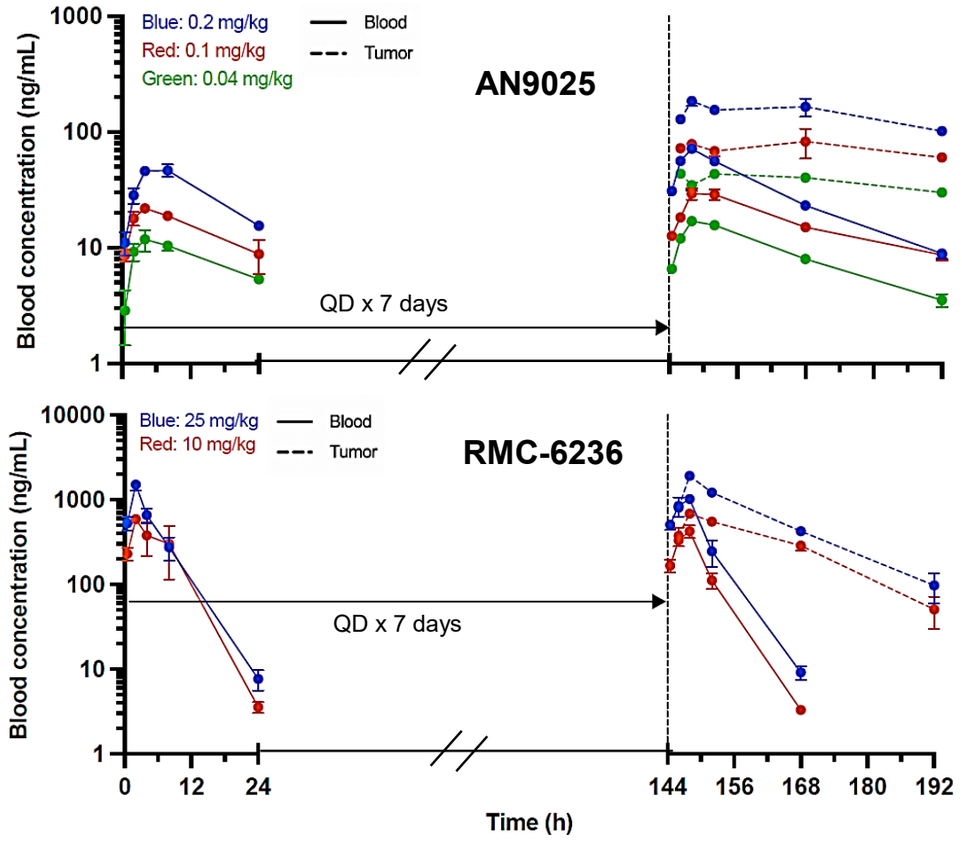
	AN9025		RMC-6236	
HPAC CTG[1] (IC_{50}, nM)	0.056		9.4	
Dose (mg/kg)	0.04	0.10	10	25
RTV[2] (%) on Day 18	150%	47%	80%	31%
RTV[2] (%) on Day 35	237%	85%	275%	164%

- AN9025 demonstrated **comparable antitumor activity** to RMC-6236
- Notably, AN9025 exhibited **more sustained efficacy** following drug withdrawal

(1) HPAC CTG = human pancreatic cancer (HPAC) CellTiter-Glo (CTG) assay, measures a drug's efficacy in killing HPAC cells via luminescent cell viability readout
(2) RTV = relative tumor volume, relative to the tumor volume at the start of treatment
Source: AN9025 Poster, Abstract #4377, 2025 AACR Annual Meeting.

More Durable RAS Inhibition In Vivo by PK-PD Analysis

PK–PD correlation was evaluated in a HPAC xenograft model after 7 days of continuous oral dosing and compared to PK after the first dose



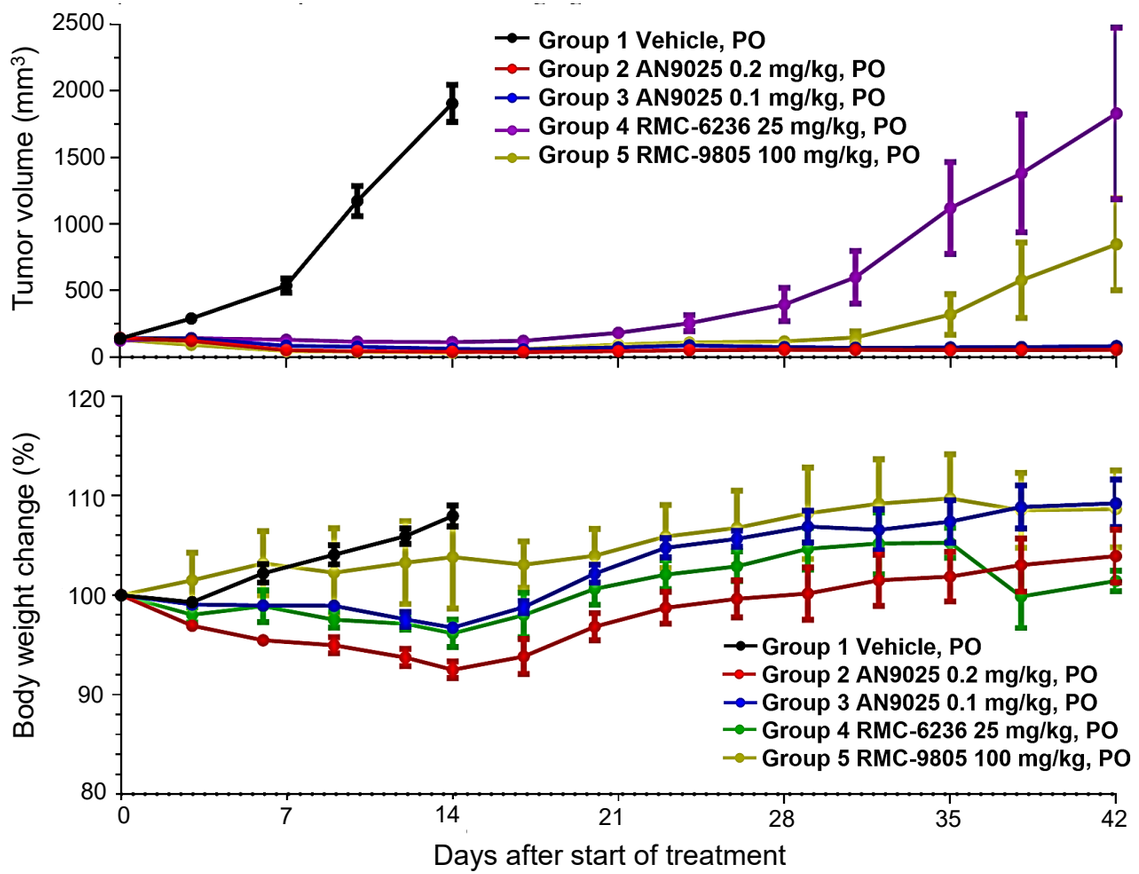
Post-Treatment PK/PD Profile (144~192 hr)		AN9025			RMC-6236	
		0.04 mg/kg, PO, QD*7	0.1 mg/kg, PO, QD*7	0.2 mg/kg, PO, QD*7	10 mg/kg, PO, QD*7	25 mg/kg, PO, QD*7
$AUC_{0\text{-}48\ hr}$	Tumor (h*ng/g)	**1,802**	**3,454**	**6,910**	14,653	29,227
	Blood	**438**	**826**	**1,476**	3,169	7,583
	Plasma (h*ng/L)	**NA**	**40**	**69**	782	2,542
C_{max}	Tumor (ng/g)	**44**	**83**	**186**	683	1,911
	Blood	**17**	**29**	**72**	425	1,018
	Plasma (ng/L)	**NA**	**2.5**	**6.4**	156	386
Tumor DUSP6 inhibition	168 hours	**87.2%**	**96.5%**	**96.2%**	63.8%	85.5%
	192 hours	**76.2%**	**94.5%**	**96.2%**	7.9%	46.3%

- AN9025 demonstrated a **more durable RAS inhibition** than RMC-6236 by tumor DUSP6 inhibition
- These results indicate that AN9025 has the flexibility to be investigated with an **intermittent dosing** strategy in a clinical setting

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AUC, area under the curve; Cmax, maximum concentration; DUSP6, Dual Specificity Phosphatase 6 (enzyme that turns off ERK1/2 MAP kinases to control cell growth, movement, and signaling); HPAC, human pancreatic ductal adenocarcinoma epithelial cell line; PD, pharmacodynamics; PK, pharmacokinetics;

More Prolonged Anti-Tumor Effect in KP-4 CDX Model



Anti-Tumor Effect	AN9025		RMC-6236	RMC-9805
Dose (mg/kg)	**0.1**	**0.2**	25	100
RTV[1] (%) on day of best tumor regression	**41.6%**	**26.6%**	81.0% (D14)	25.7% (D14)
RTV[1] (%) on Day 42	**58.2%**	**41.0%**	1331%	658%

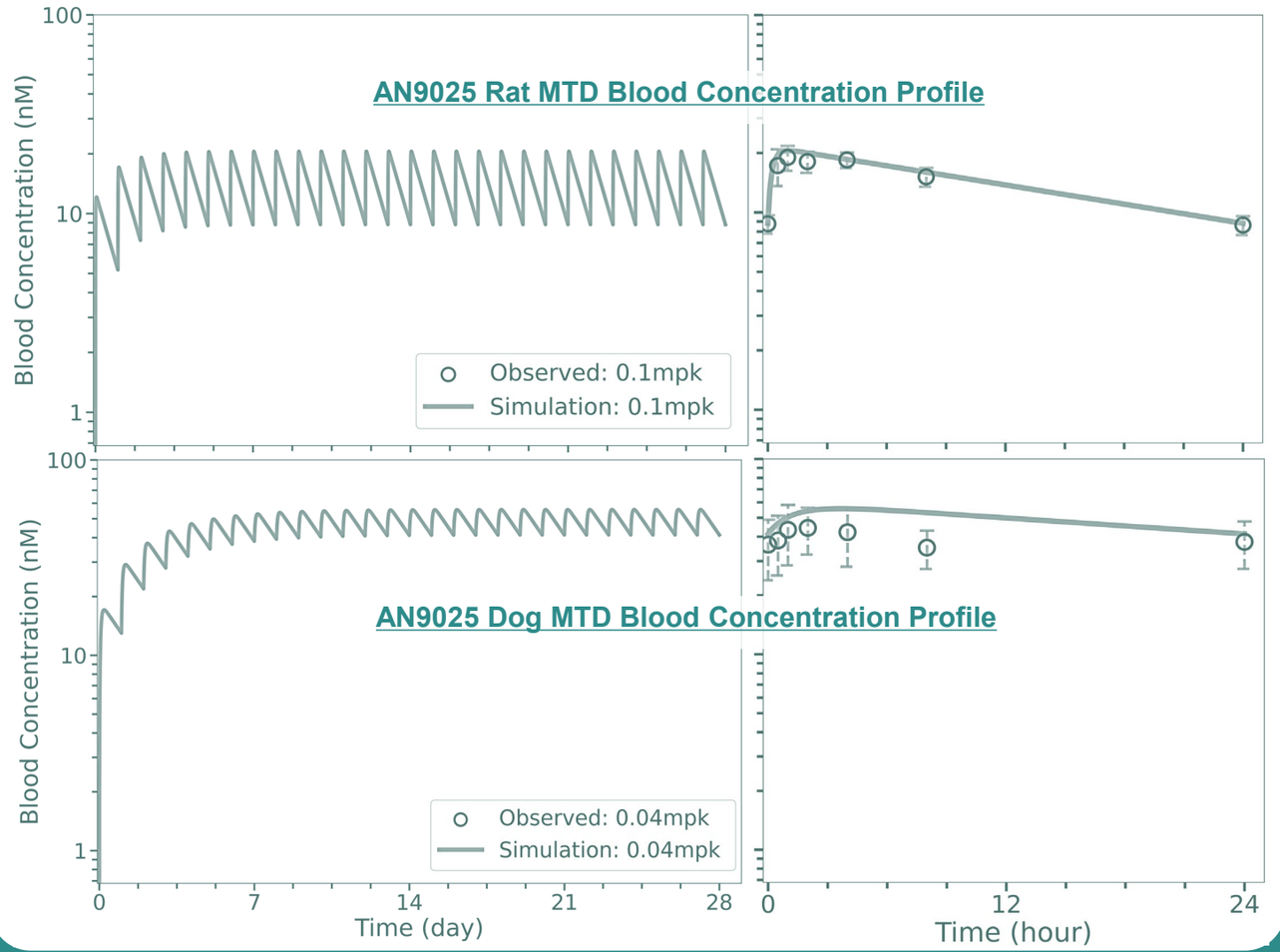
AN9025 exhibited significantly **prolonged anti-tumor efficacy** and **delayed the emergence of adaptive resistance** compared to RMC-6236 and RMC-9805 in the KP-4 ($KRAS^{G12D}$) CDX model (RTK hyperactivation and Myc amplification)

(1) RTV = relative tumor volume; relative to the tumor volume at the start of treatment

Overall Favorable PK Properties

		Mouse	Rat	Dog	Monkey
IV	**Dose** (mpk)	0.25	0.03	0.005	0.1
	$T_{1/2}$ (h)	39	21	49	18
	Vd_{ss} (L/Kg)	1.9	2.4	1.7	0.92
	CL (mL/Kg/min)	1.2	1.6	0.44	0.7
	$AUC_{0\text{-}inf}$ (ng/mL*h)	3,350	324	203	2,373
PO	**Dose** (mpk)	0.50	0.10	0.02	0.5
	C_{max} (ng/mL)	46	16	9.5	100
	$T_{1/2}$ (h)	37	20	44	12
	$AUC_{0\text{-}inf}$ (ng/mL*h)	3,300	400	450	2,100
	Bioavailability (F%)	44%	37%	57%	18%

The PK simulation model demonstrated good concordance with observed TK data at Day 28 from 4-week GLP toxicology studies in rats and dogs

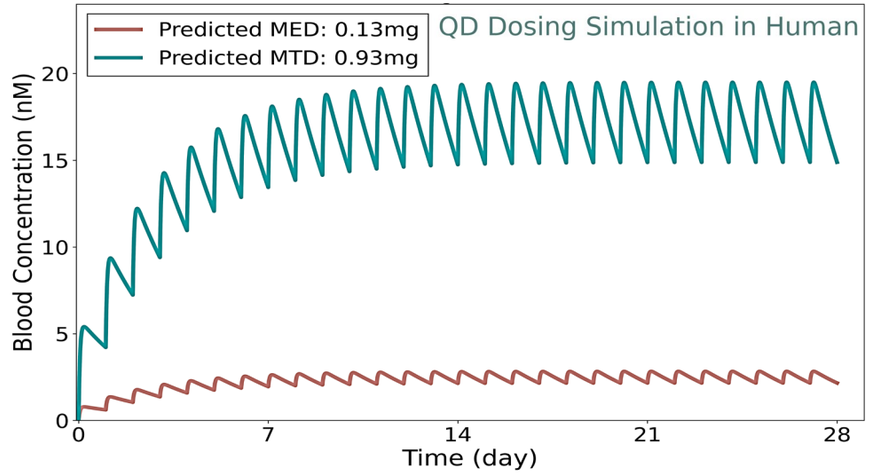


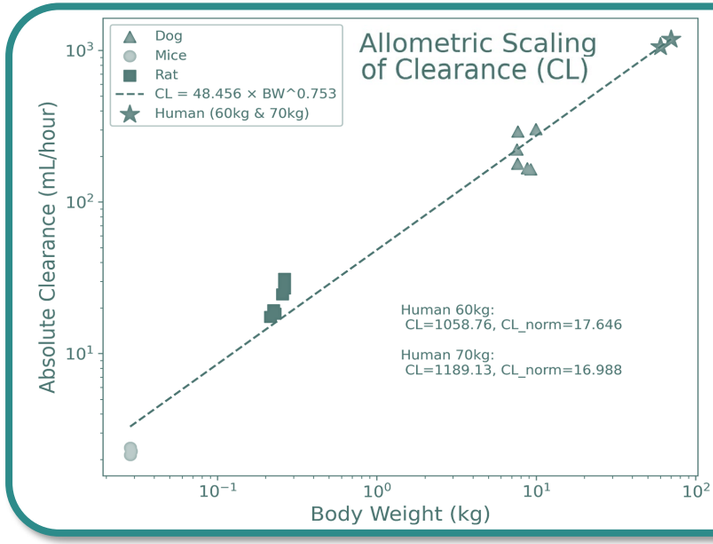
AUC, area under the curve; CL, clearance; Cmax, maximum concentration; GLP, good laboratory practice; PK, pharmacokinetics; TK, toxicokinetics; $T_{1/2}$, half life; Vd_{ss}, volume of distribution at steady state

Therapeutic Index and Human PK Simulation

		AN9025			RMC-6236		
Species		**Mouse**	**Rat**	**Human** (60kg)[4]	**Mouse**[5]	**Rat**[5]	**Human** (60kg)[6]
TSC[1] (nM)		6.9	8.2	2.45	145	350	88
MED[1]	**Dose** (mpk)	0.030	0.058	0.0022 **(0.13mg)**	8.6	48	1.8 (**105mg**)
	AUC (nM*hr)	167	198	61	3,480	8,400	2,160
	C_{max} (nM)	9.8	12	2.8	468	1,040	195
MTD[2]	**Dose** (mpk)	0.20	0.20	0.016 **(0.93mg)**	50	80	9.2 (**552mg**)
	AUC (nM*hr)	1,390	418	417	16,900	11,400	11,300
	C_{max} (nM)	72	25	19.5	3,060	1,110	1,020
TI[3]		6.6	3.4	**6.9**	5.8	1.6	**5.3**

- The simulation model demonstrated good predictability for both the human starting efficacious dose and maximum tolerated dose of RMC-6236
- With a QD regimen, AN9025 exhibited a **therapeutic index comparable to or slightly superior** to that of RMC-6236, despite its significantly greater potency





Predicted AN9025 human PK based on allometric scaling:

CL = 0.018 L/(h*kg)

V = 1.3 L/kg

Predicted human $T_{1/2}$ = 40 hr

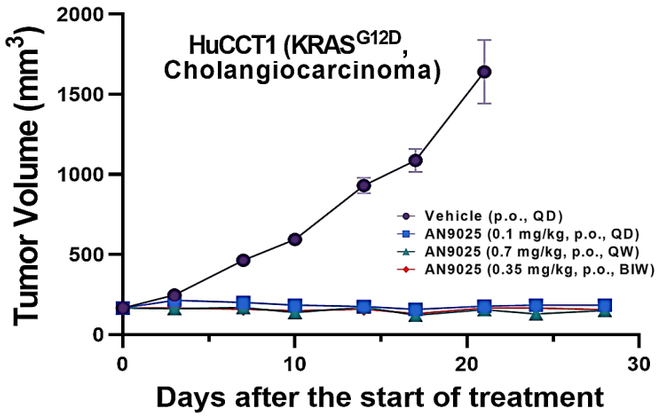
(which compares favorably to 12 hr for RMC-6236)

MED, minimum efficacious dose; MTD, maximum tolerated dose; TI, therapeutic index; TSC, tumor static concentration

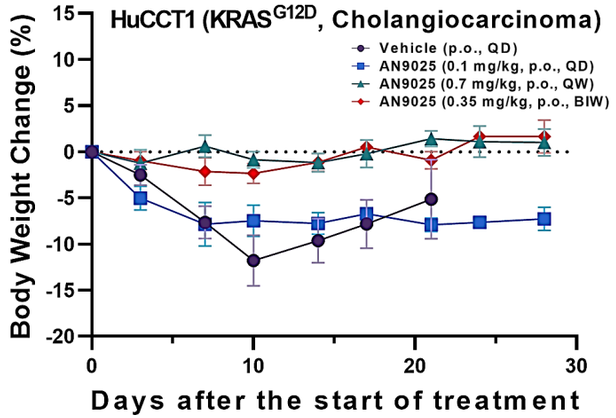
(1) TSC and MED projections are derived from PK-Efficacy modeling using the HPAC CDX model, with blood average drug concentration = tumor static concentration (TSC); (2) MTD observed in 4-week in-vivo efficacy study in mice and 2-week DRF toxicology studies in rats; (3) TI = MTD / MED; (4) AN9025 human PK was projected via allometric scaling from mice, rat and dog data shown on slide#14 of the deck; (5) The MED and MTD of RMC-6236 in mouse and rat were determined internally, with direct comparison to AN9025 performed in the same studies; (6) The human PK parameters of RMC-6236 were derived from RVMD's translational study published in Cancer Discovery (2024; Volume 14, pp. 994–1017)

Intermittent Dosing Achieves Comparable Tumor Regression

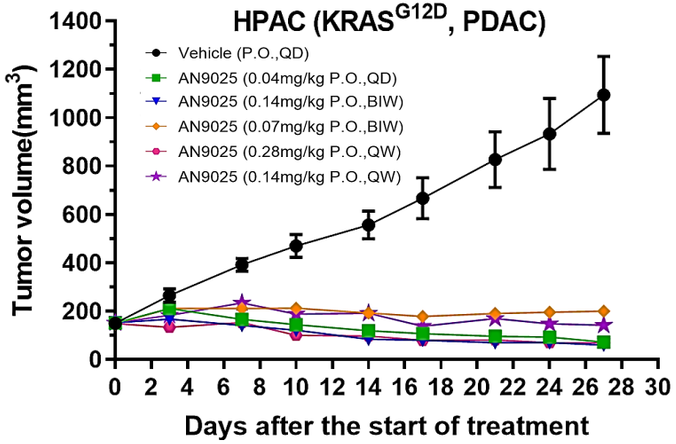
AN9025 demonstrated **comparable tumor regression** in mouse CDX models with intermittent dosing relative to QD dosing with slightly improved tolerability on intermittent regimen



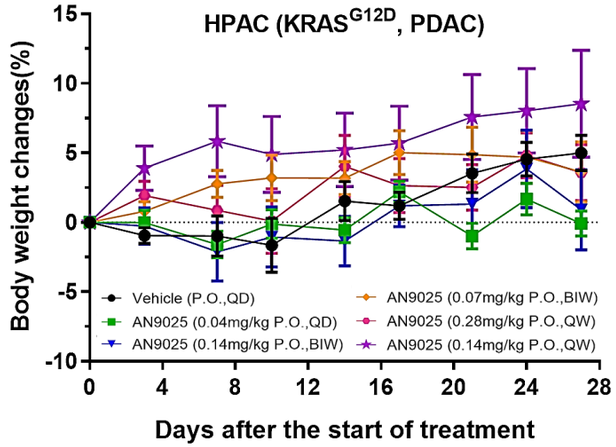
Dose (mg/kg)	RTV[1] (Day 28)	BW change[2] (%, Day 28)
Vehicle	9.9 (Day 21)	-5.2% (Day 21)
0.1, QD	1.11	-7.3%
0.35, BIW	0.94	+1.6%
0.7, QW	0.90	+1.0%



A **lower projected tumor static concentration (TSC)** was observed with QW dosing compared to QD dosing, supported by durable PD inhibition



Dose (mg/kg)	RTV[1] (%, Day 28)	DUSP6 inhibition (%, Day 28)	Projected TSC (nM)
0.04, QD	48%	79.7%	6.2
0.14, BIW	40%	87.9%	5.9
0.07, BIW	135%	73.2%	
0.28, QW	46%	89.4%	4.6
0.14, QW	96%	77.4%	



DUSP6, Dual Specificity Phosphatase 6 (enzyme that turns off ERK1/2 MAP kinases to control cell growth, movement, and signaling); HPAC, human pancreatic ductal adenocarcinoma epithelial cell line; TSC, tumor static concentration

(1) RTV = tumor volume on day 28 relative to the tumor volume at the start of treatment; (2) BW change % means the change in body weight on day 28 compared to day 1

Widening of AN9025 Therapeutic Window with Intermittent Dosing

Species	Dosing Regimen	TSC[1] (nM)	MED[1]			MTD[2]			TI[3]
			Dose (mpk)	Weekly AUC (nM*hr)	C_{max} (nM)	Dose (mpk)	AUC (nM*hr)	C_{max} (nM)	
Mouse	QD	6.2	0.028	1,050	8.8	0.2	1,390	72	**7.3**
	BIW	5.8	0.09	983	22	.	.	.	.
	QW	4.6	0.14	792	34	.	.	.	.
Rat	QD	7.4	0.053	1,300	11	**0.2**	378 Day 14 AUC(0~24h)	24	**3.8**
	BIW	7.1	0.18	1,220	23	**1.4**	1,750 Day 11 AUC(0~72h)	87	**7.8**
	QW	5.7	0.28	965	34	**2.8**	3,320 Day 8 AUC(0~72h)	290	**9.8**
Human (60 kg)[4]	QD	2.2	0.002 **(0.12 mg)**	377	2.5	0.014 **(0.84 mg)**	373 Day 14 AUC(0~24h)	17	**7.0**
	BIW	2.0	0.0064 **(0.384 mg)**	344	3.4	0.068 **(4.1 mg)**	1,730 Day 11 AUC(0~72h)	36	**11**
	QW	1.5	0.01 **(0.60 mg)**	269	3.9	0.18 **(11 mg)**	3,310 Day 8 AUC(0~72h)	69	**18**

The **broader therapeutic window** observed for AN9025 supports the selection of a **once-weekly (QW)** dosing regimen for evaluation in the Phase 1 first-in-human clinical study.

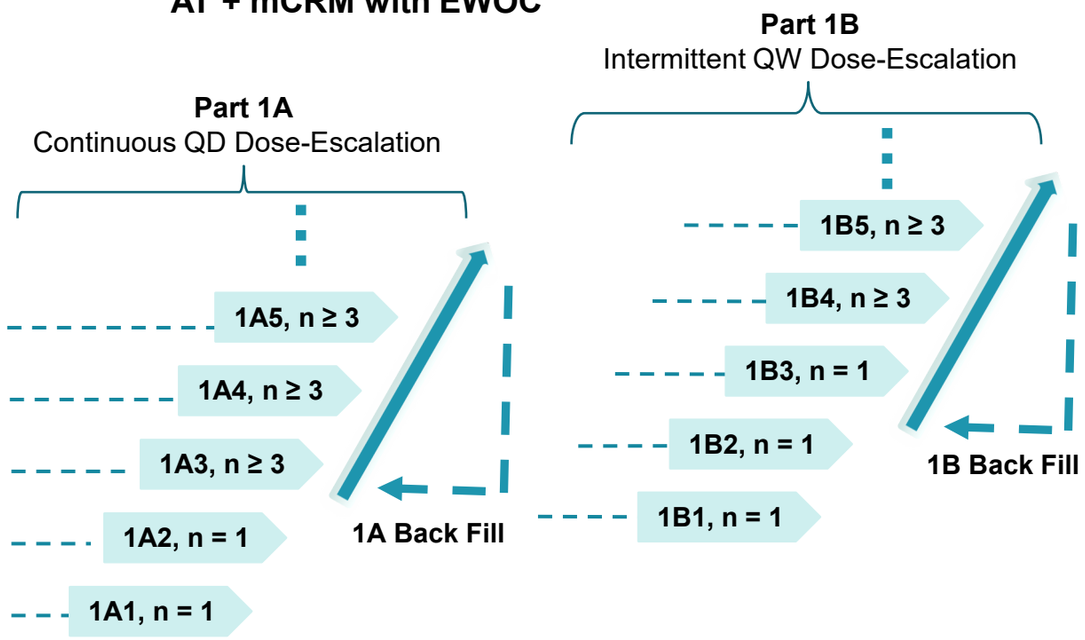
Adlai Nortye

AUC, area under the curve; Cmax, maximum concentration; MED, minimum efficacious dose; MTD, maximum tolerated dose; TI, therapeutic index; TSC, tumor static concentration

(1) TSC and MED projections are derived from PK-Efficacy modeling using the HPAC CDX model, with blood average drug concentration = tumor static concentration (TSC); (2) MTD observed in 4-week in-vivo efficacy model in mice and 2-week DRF toxicology study in rats; (3) Therapeutic index, TI = MTD / MED; (4) AN9025 human PK was projected via allometric scaling from mice, rat and dog data shown on slide#14 of the deck

AN9025 Global Phase I Multi-Regional Clinical Trial in U.S. and China

Part 1: Dose-Escalation[1]
AT + mCRM with EWOC



Key Eligibility Criteria:

- Advanced / metastatic solid tumors
- RAS-mutation (KRAS / NRAS / HRAS)
- ECOG 0-1
- pan-RAS(ON) inhibitor-naïve *(except backfill cohorts)*

Primary Endpoints
- DLTs
- Safety
- Tolerability

Secondary Endpoints
- PK
- ORR
- DCR
- DoR
- PFS

Part 2: Food Effect Assessment

Selected dose level below MTD

Part 3: Dose-Expansion and Dose-Optimization (1-2 RDE)

Cohort 3A/B:
RAS-mutated solid tumors (2L/3L)

Cohort 3C:
1L PDAC: AN9025 + Chemotherapy

Adlai Nortye

AT, Accelerated Titration; DCR, disease control rate; DLT, dose limiting toxicity; DoR, duration of response; EWOC, Escalation With Overdose Control; mCRM, modified Continual Reassessment Method; MTD, maximum tolerated dose; ORR, objective response rate; PFS progression-free survival; RDE, recommended dose for expansion

(1) Study is being conducted under a single global protocol.

Summary of AN9025 Program

AN9025's preclinical profile demonstrates best-in-class attributes among pan-RAS(ON) inhibitors:

- ✓ **CypA Binding Affinity:** Exhibited ~4-fold stronger binding to cyclophilin A (CypA) than daraxonrasib, driven by a slower dissociation rate, which may enable more sustained RAS inhibition
- ✓ **Tri-Complex Binding Affinity:** Exhibited ~3 to 8-fold higher binding affinity for tri-complex formation than daraxonrasib, also attributable to a slower dissociation
- ✓ **Potency:** Demonstrated ~100-fold greater potency in inhibiting cellular proliferation and more sustained RAS inhibition compared to daraxonrasib
- ✓ **Anti-Tumor Effect:** Induced deep tumor regression, superior and more sustained anti-tumor effects compared with daraxonrasib in mouse CDX models harboring different RAS mutations
- ✓ **Bioavailability & Half-Life:** Demonstrated favorable overall DMPK properties, including 18~57% bioavailability across multiple species and a ~2 to 5-fold longer half-life than daraxonrasib
- ✓ **Therapeutic Window:** Demonstrated a therapeutic index comparable or slightly superior to daraxonrasib with QD dosing despite substantially higher potency. QW dosing showed potential to further widen the therapeutic window

AN9025 is advancing in the clinic:

- ➢ **Both QD and QW** arms are now enrolling concurrently
- ➢ **Backfill cohort of QD schedule** has been initiated, and **ex-China, ex-US clinical sites** will be added
- ➢ The Phase I dose escalation is on track for completion and **data readout in H1 2027**



AN4035

Novel CEACAM5 Pan-RAS(ON) Inhibitor ADC

Adlai Nortye

Addressing the Limitations of Pan-RAS(ON) Inhibitors with ADC Technology

Limitations of current Pan-RAS(ON) inhibitors due to systemic exposure

- **Skin toxicities**
- **GI toxicities**
- **Combination limitations**

Our Solution

Deliver pan-RAS inhibition selectively to tumors using antibody-drug conjugate (ADC) technology, to maximize tumor targeting and minimize systemic toxicity

Advantages

- Novel mechanism beyond Topo-I and MMAE payloads to expand ADC payload diversity
- Potentially address pan-RAS(ON) therapeutic window limitations
- Enable rational combinations with both chemotherapies and targeted therapies

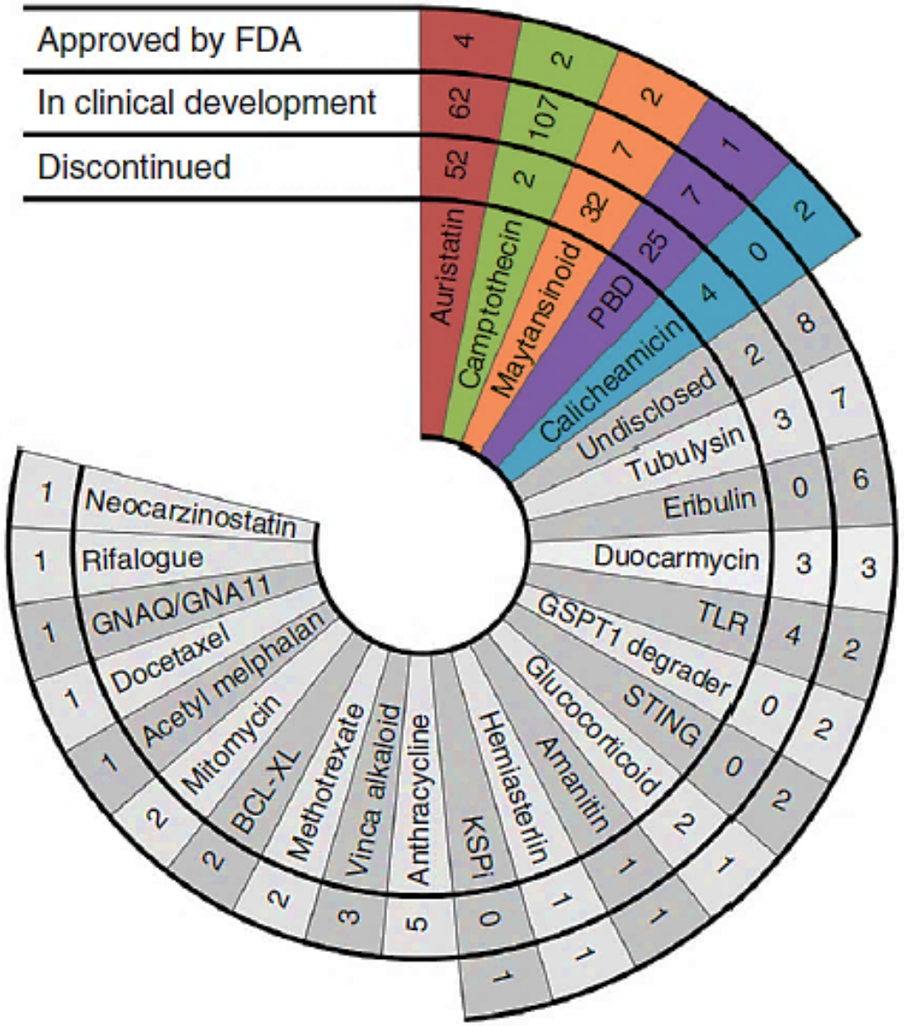
RASiCA™: Our Proprietary RAS Inhibitor Conjugated Antibody Platform

Our RASiCA™ platform was designed to overcome two major challenges:

1. **ADC field:** limited diversity of payload classes leading to cross-resistance, which then limits treatment sequencing for ADCs[1,2]
2. **RAS inhibitor field:** on-target, off-tumor toxicities associated with oral small molecule tri-complex pan-RAS(ON) inhibitors

1. We designed our RASiCA™ platform to diversify ADC payloads to overcome clinical resistance

- Payload-related cross resistance limits treatment sequencing for ADCs[1,2]
- For example, patients with HER2-high metastatic breast cancer who received a second ADC with a similar payload class showed significantly worse outcomes than those switched to an ADC with a different mechanism of action[3]
- Thus, payload-specific resistance is a key clinical challenge, creating an urgent need for novel payload classes to enable more effective treatment sequences

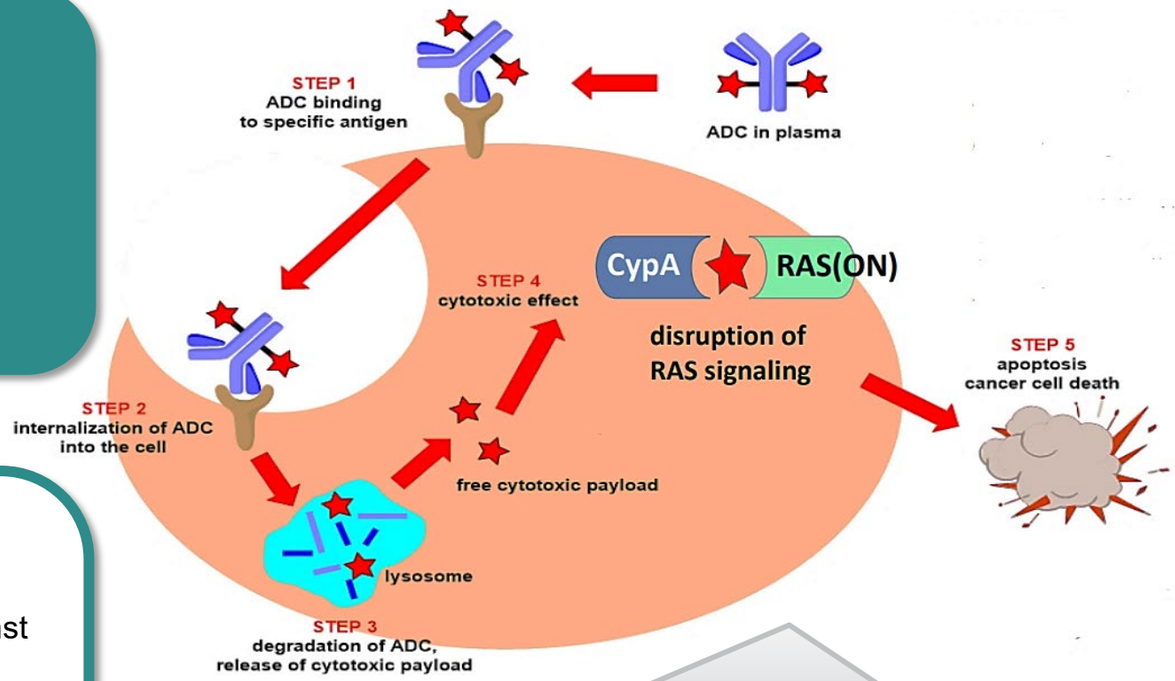


Sources: 1. Abelman et al. Clin Cancer Res 2025; 2. Peng et al. Breast, 2025; 3. Tarantino et al. Clin Cancer Res 2025 (abstr P1-08-08)X

RASiCA™: Our Proprietary RAS Inhibitor Conjugated Antibodies Platform

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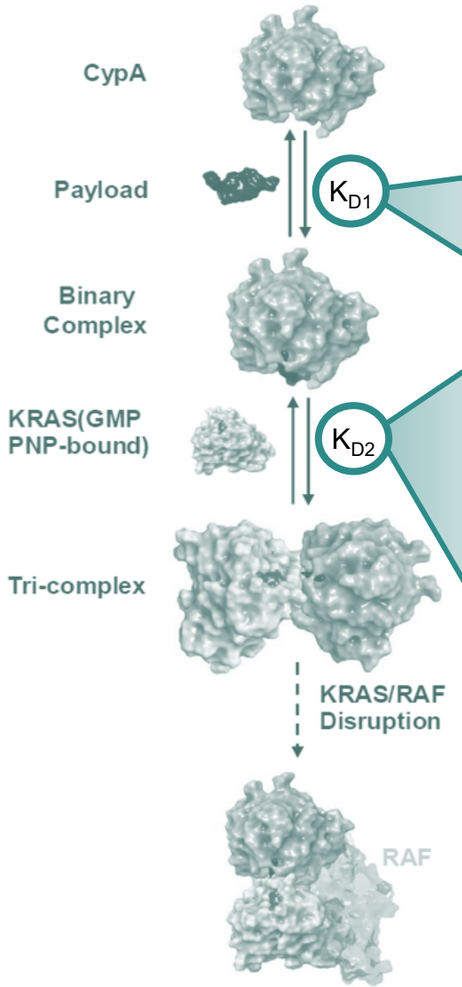


2. We designed our RASiCA™ platform to deliver a pan-RAS(ON) inhibitor as an ADC payload

- Tricomplex pan-RAS(ON) inhibitors are a promising class of therapeutics against RAS-driven cancers
- However, on-target, off-tumor toxicities may restrict dose and limit safety in combinations
- Thus, we hypothesize that targeted delivery via ADC could localize pan-RAS(ON) inhibitor activity to tumors while minimizing systemic RAS pathway inhibition
- This could potentially widen the therapeutic window and enable rational combinations

Cyclophilin A, an abundant intracellular protein (μM levels), is the target engaged by pan-RAS(ON) inhibitors, much like MMAE targeting tubulin

Payload Is a Proprietary Highly Potent Pan-RAS(ON) Inhibitor



Binding Affinity		Our Payload	RMC-6236
Binary (CypA) K_{D1} (nM)		14.9	12.5
Tricomplex (KRAS) K_{D2} (nM)	G12C	4.7	19.1
	G12D	30.5	174
	G12V	14.0	45.9
	WT	18.6	52.8

- Our proprietary ADC payload has a pan-RAS(ON) inhibitor mechanism of action
- It has demonstrated **nanomolar binding affinity** for both binary complex and tri-complex formation

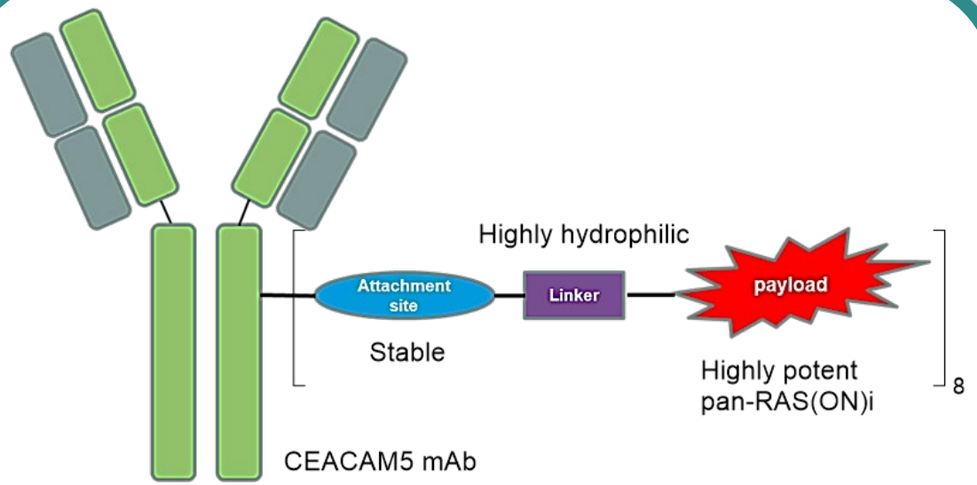
Our proprietary payload demonstrated nanomolar cytotoxic potency against various tumor cell lines *in vitro*

Indication	Cell Line	RAS mutation type	Payload 72h CTG	
			IC_{50} (nM)	I_{max} (%)
NSCLC	NCI-H2009	Kras G12A	0.02	65
	NCI-H358	Kras G12C	0.06	93
	NCI-H2030	Kras G12C	0.04	22
	NCI-H2122	Kras G12C	0.01	93
	NCI-H441	Kras G12V	<0.01	44
	SW900	Kras G12V	0.12	80
PDAC	ASPC1	Kras G12D	0.06	63
	HPAC	Kras G12D	0.07	63
	KP4	Kras G12D	0.75	56
	Capan1	Kras G12V	0.02	57
	Capan2	Kras G12V	0.09	61
CRC	SW620	Kras G12V	0.01	80
	HCT116	Kras G13D	0.41	93
	LoVo	Kras G13D	0.34	67
	T84	Kras G13D	2.59	74
	LS1034	Kras A146T	1.0	80

CRC, colorectal carcinoma; CTG, Cell-Titer Glo assay; CypA, cyclophilin A; IC_{50}, half maximal inhibitory concentration; I_{max}, maximum inhibitory effect; MOA, mechanism of action; NSCLC, non-small cell lung carcinoma; PDAC, pancreatic ductal adenocarcinoma

Source: AN4035 Late-Breaking Oral Presentation, 2025 AACR-NCI-EORTC International Conference: Molecular Targets and Cancer Therapeutics.

Overview of AN4035 Molecular Structure and Stability



CEACAM5: overexpressed in CRC, PDAC, & NSCLC where RAS is frequently mutated

Conjugator: homogenous DAR 8 with high stability; no retro-Michael reaction

Linker: highly hydrophilic; excellent circulation stability and efficient payload release

Payload: highly potent pan-RAS(ON) inhibitor

AN4035 has a stable conjugator

Compound	Human plasma incubation time	LC-MS DAR
AN4035	Day 0	8.04
	Day 7	7.85

AN4035 has a stable linker-payload

Compound	Plasma incubation time	Plasma Payload Release (%)			
		Human	Mouse	Rat	Monkey
AN4035	Day 0	<0.05	<0.05	0.02	<0.05
	Day 7	<0.05	<0.05	0.01	<0.05
	Day 14	0.05	<0.05	0.01	0.06

AN4035 shows no aggregation after incubation for 72hr at 40℃

Compound	Incubation time[1]	SEC-HPLC Aggregation (%)
AN4035	0 hr	0
	72 hr	0

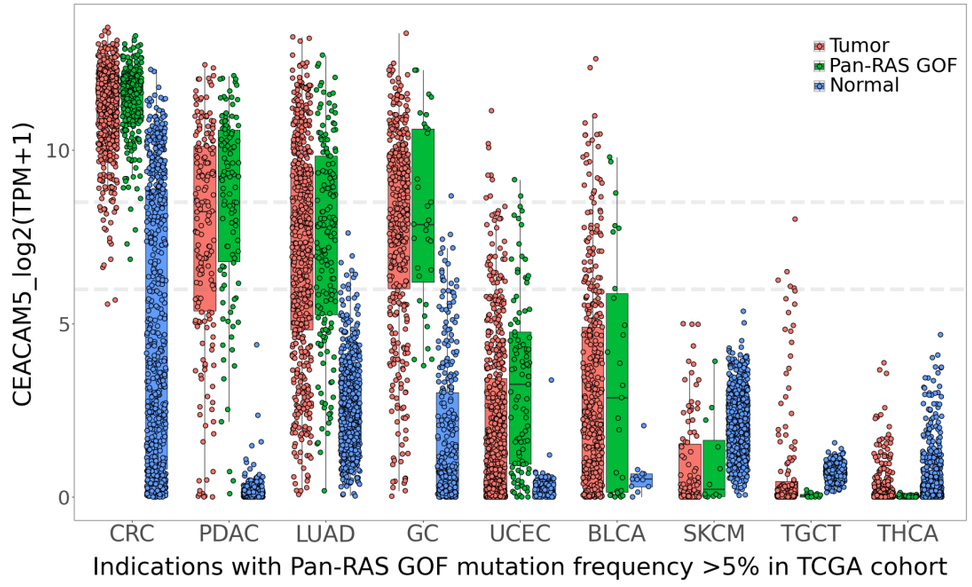
DAR, drug-to-antibody ratio; LC-MS, liquid chromatography-mass spectrometry; SEC-HPLC, Size-Exclusion Chromatography High-Performance Liquid Chromatography

(1) Incubation buffer: 20 mM Histidine, 240 mM Sucrose, pH 5.5

Source: AN4035 Late-Breaking Oral Presentation, 2025 AACR-NCI-EORTC International Conference: Molecular Targets and Cancer Therapeutics.

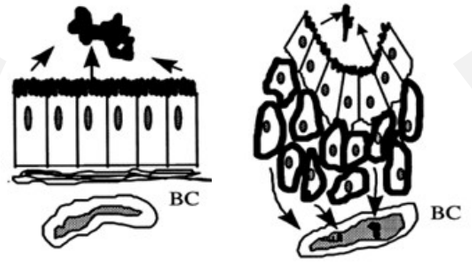
CEACAM5 as a Compelling Target for Pan-RAS(ON) Inhibitor-Based ADC

RAS-Mutant Cancer–Enriched, Skin-Sparing Expression[1]



Differential Expression in Epithelial Cells

Normal: Expression in normal cells is weak and apical



Tumor: Expression in tumor cells is high and throughout the whole surface

Clinically-Validated ADC Target Without Gastrointestinal Dose-Limiting Toxicity

- Meaningful efficacy in *RASmut*-prevalent cancers including NSCLC & CRC
- Minimal / manageable on-target GI AEs at doses up to 10mg/kg QW, despite the presence of CEACAM5 on normal gut epithelium

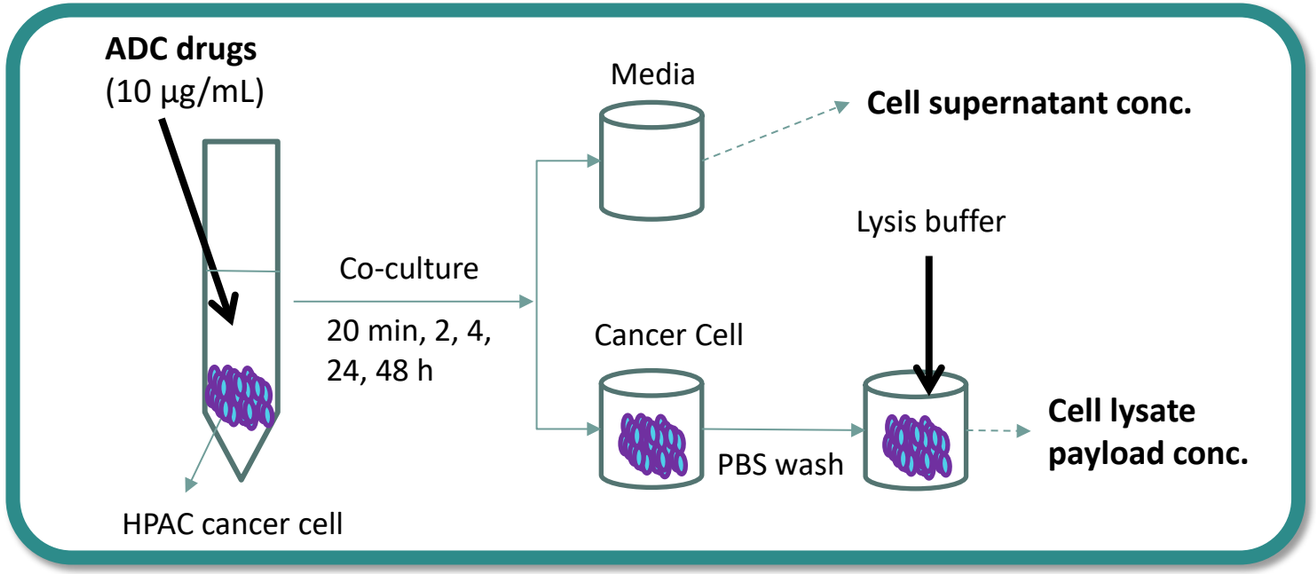
ADC	Payload / DAR	RP2D	Clinical Efficacy	Diarrhea TRAE		GI AEs as DLT
				All grade	≥Gr3	
SAR408701	DM4 DAR: 3.8	100 mg/m^2 Q2W (2.6 mg/kg @70kg)	**ORR 21.7%** 2/3L CEA-high nsq-NSCLC, Ph.III[2]	9.8%[2]	**0.5%**[2]	None[3]
M9140	Exatecan DAR: 8	2.8 mg/kg Q3W	**ORR 26.8%** 3L+ mCRC, Ph.I[4]	~25%[4]	**0%**[4]	None[5]
IMMU-130	SN38 DAR: 7.6	8 / 10 mg/kg QW	**DCR 62% / 55%** Late-line mCRC Ph.I/II[6]	46% / 52% TEAE[6] 56% TRAE	**5%** / **5%** TEAE[6] 7.9% TRAE	None[6]

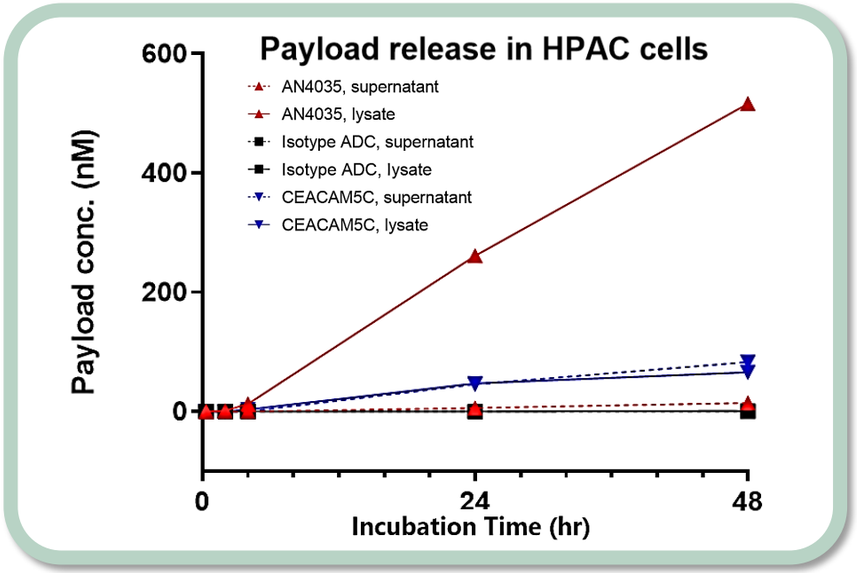
ADC, antibody-drug conjugate; CEACAM5, Carcinoembryonic antigen-related cell adhesion molecule 5, also known as CEA; CRC, colorectal cancer; DAR, drug-to-antibody ratio; DLT, dose-limiting toxicity; GOF, gain-of-function; LUAD, non-small cell lung adenocarcinoma; PDAC, pancreatic ductal adenocarcinoma; TRAE, treatment-related adverse event

Adapted from ASCO 2024 Discussant on Abs #3000, 3001, 3002

(1) Expression data are derived from TCGA, GTEx datasets, HER2 mRNA-IHC threshold: https://doi.org/10.1136/jitc-2023-SITC2023.0143; (2) WCLC 2024 OA08.05; (3) Gazzah 2022, DOI:10.1016/j.annonc.2021.12.012; (4) ASCO GU 2026 #129P; (5) Kopetz 2024, DOI:10.1038/s41591-025-03843-z; (6) Dotan 2017, DOI:10.1200/JCO.2017.73.9011

Superior Target-Mediated Intracellular Payload Retention





Incubation Time	**AN4035** (10 µg/mL)			**Isotype-payload** (10 µg/mL)		**CEACAM5C** (10 µg/mL)		
	Cell supernatant payload conc. (nM)	Cell lysate payload conc. (nM)	**Lysate/ supernatant payload ratio**	Cell supernatant payload conc. (nM)	Cell lysate payload conc. (nM)	Cell supernatant payload conc. (nM)	Cell lysate payload conc. (nM)	**Lysate / supernatant payload ratio**
20 min	BQL	BQL	-	BQL	BQL	BQL	BQL	-
2 hr	BQL	1.75	-	BQL	BQL	BQL	BQL	-
4 hr	BQL	12.9	-	BQL	BQL	BQL	3.58	-
24 hr	5.97	261	**44**	BQL	BQL	45.8	47.5	**1.0**
48 hr	14.4	516	**36**	BQL	1.41	83.1	66.0	**0.79**

ADC, antibody-drug conjugate; BQL, below quantitation limit; CEACAM5C, investigational ADC with CEACAM5-targeting and Topo1 payload; HPAC, human pancreatic ductal adenocarcinoma cell line

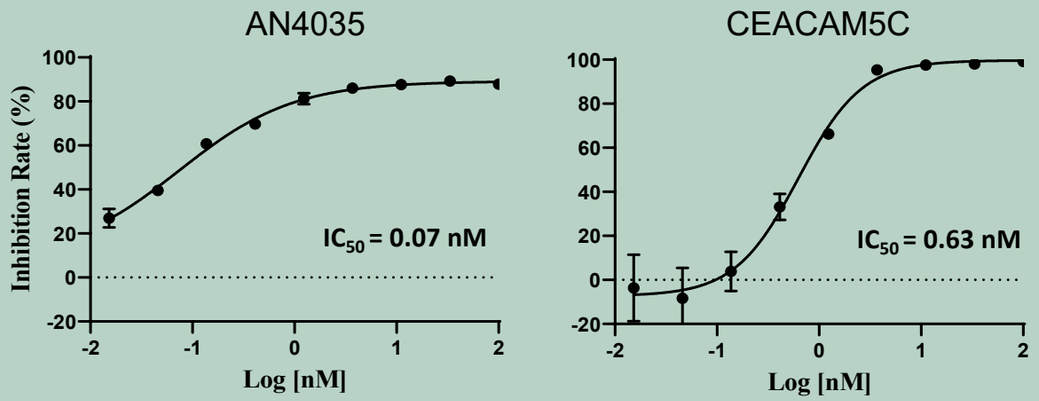
Source: AN4035 Late-Breaking Oral Presentation, 2025 AACR-NCI-EORTC International Conference: Molecular Targets and Cancer Therapeutics.

Potent Cytotoxicity and Robust Bystander Effect in RAS-Addicted Cancer Cell Models

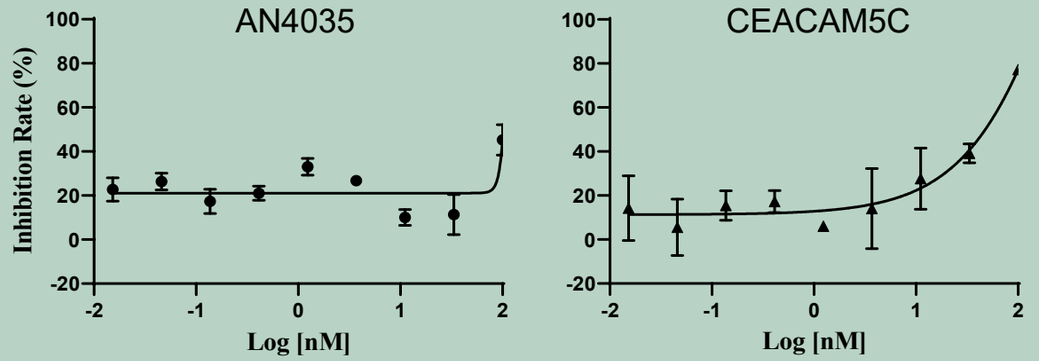
Cancer Type	Cell Line	CEACAM5 mRNA nTPM	RAS mutation type	6-day CTG		
				AN4035		Isotype ADC
				IC_{50} (nM)	I_{max} (%)	IC_{50} (nM)
CRC	CL-40	1,821	Kras G12D	0.77	98	
	SK-CO-1	784	Kras G12V	0.02	99	
	LS513	208	Kras G12D	0.15	100	
	SW403	443	Kras G12V	2.8	95	
	SW1463	198	Kras G12C	2.3	99	
	LoVo	122	Kras G13D	4.4	48	
	GP2D	42	Kras G12D	1.4	84	
PDAC	QGP-1	1360	Kras G12V	0.05	91	
	HPAC	714	Kras G12D	0.10	73	>100
	ASPC1	499	Kras G12D	0.31	88	
	Capan1	97	Kras G12V	0.23	76	
NSCLC	NCI-H2122	616	Kras G12C	0.30	94	
	NCI-H727	332	Kras G12V	0.09	93	
Gastric cancer	MKN45	1,087	Kras WT	0.13	95	>100

AN4035 showed superior bystander killing effect compared to CEACAM5 Topol-based ADC

SK-CO-1 (CEACAM5 pos) : SW480-Luc (CEACAM5 neg) = 10:1



SW480-Luc (CEACAM5 neg) only

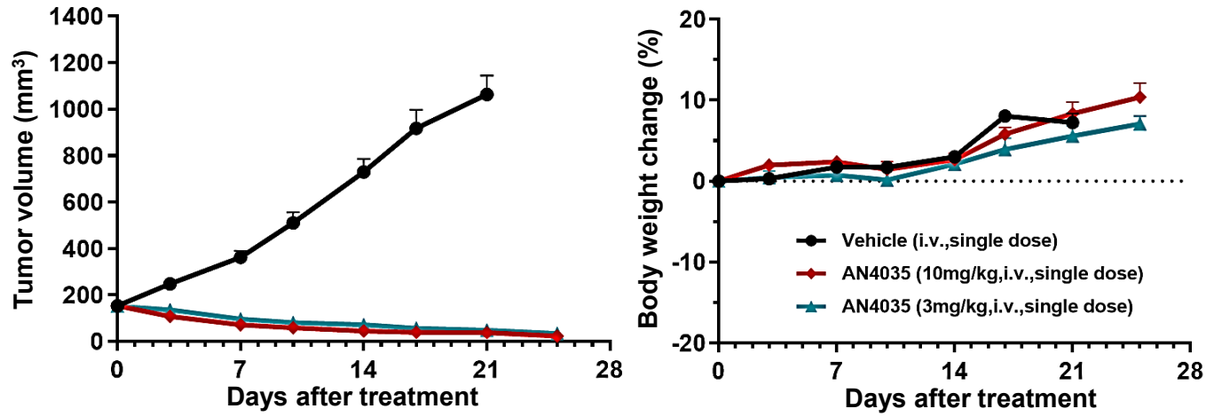


CEACAM5, Carcinoembryonic antigen-related cell adhesion molecule 5; CEACAM5C, investigational ADC with CEACAM5-targeting and Topo1 payload; CRC, colorectal carcinoma; CTG, Cell-Titer Glo assay; IC_{50}, half maximal inhibitory concentration; I_{max}, maximum inhibitory effect; NSCLC, non-small cell lung carcinoma; nTPM, normalized transcripts per million; PDAC, pancreatic ductal adenocarcinoma

Source: AN4035 Late-Breaking Oral Presentation, 2025 AACR-NCI-EORTC International Conference: Molecular Targets and Cancer Therapeutics.

Robust Anti-Tumor Activity Across CDX Models

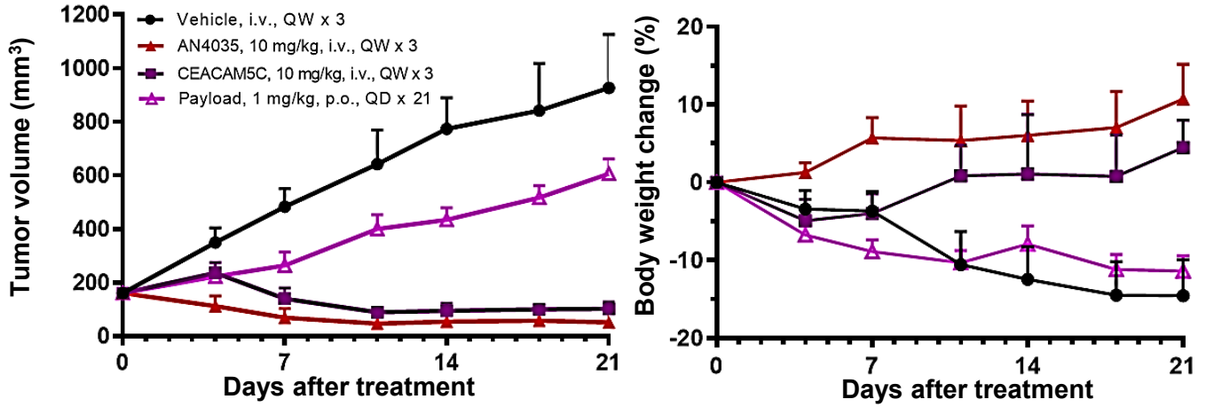
HPAC$^{KRAS\ G12D}$ model (PDAC, CEACAM5 mRNA 714)



Compound	CTG[1] (IC_{50}, nM)	Single Dose (mg/kg)	ΔRTV[2] (%, Day 25)
AN4035	0.1	3	-78%
		10	-85%

AN4035 demonstrated durable tumor regression with a single-dose of 3 mg/kg

CL40$^{KRAS\ G12D}$ model (CRC, CEACAM5 mRNA 1821)



Compound	CTG[1] (IC_{50}, nM)	Dose (mg/kg)	ΔRTV[2] (%, Day 21)
AN4035	0.38	10, QW	-89%
CEACAM5C	1.2	10, QW	-36%
Payload	0.08	1, QD	276%

AN4035 demonstrated superior or comparable efficacy to CEACAM5 Topo1i-based ADC (CEACAM5C) or naked payload at tolerable doses

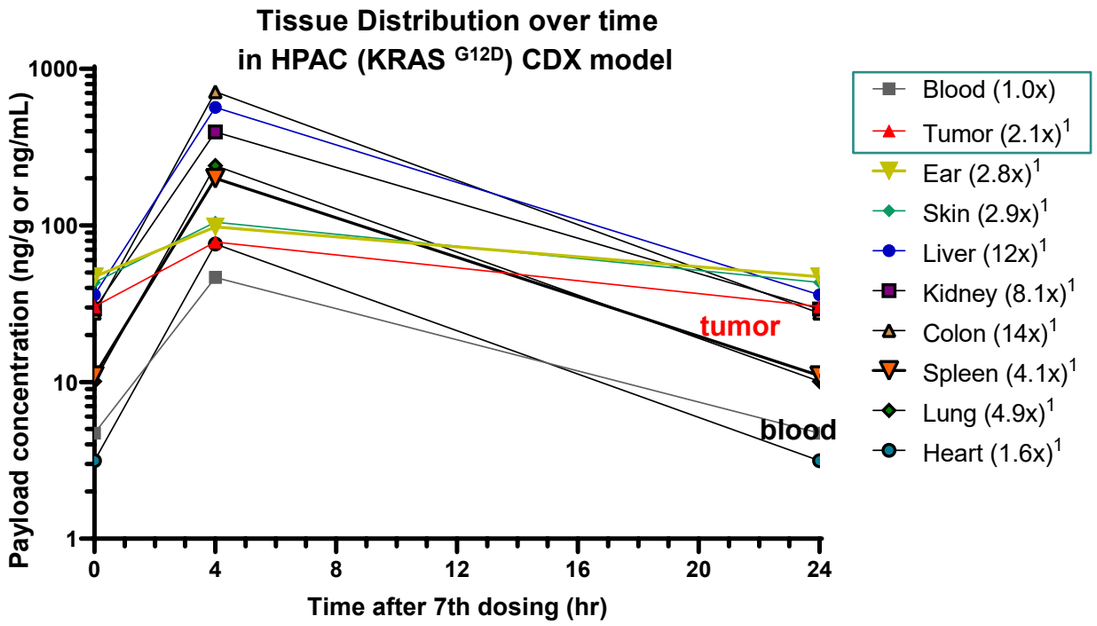
CEACAM5, Carcinoembryonic antigen-related cell adhesion molecule 5; CEACAM5C, investigational ADC with CEACAM5-targeting and Topo1 payload; CDX, cell line-derived xenograft; CTG, Cell-Titer Glo assay; IC50, half maximal inhibitory concentration; RTV, relative tumor volume

(1) CTG = cell-titer glow in vitro assay (2). ΔRTV = relative tumor volume; relative to the tumor volume at the start of treatment, ΔRTV = (Vt-V0) / V0 × 100

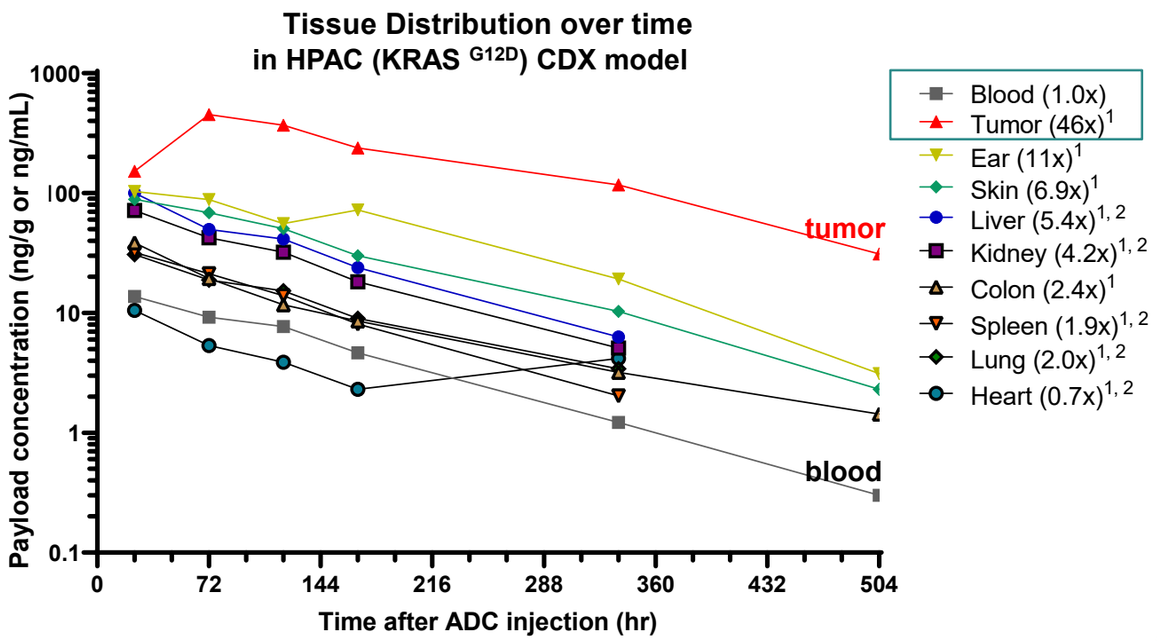
Source: AN4035 Late-Breaking Oral Presentation, 2025 AACR-NCI-EORTC International Conference: Molecular Targets and Cancer Therapeutics.

Enhanced Target-Mediated Tumor Retention, with Improved Tumor Selectivity

Free payload concentrations in tumor, normal tissues and blood after a 7-day continuous dosing of the **payload alone** at 1 mg/kg, PO



Free payload concentrations in tumor, normal tissues and blood after a single dose of **AN4035** at 10 mg/kg, IV



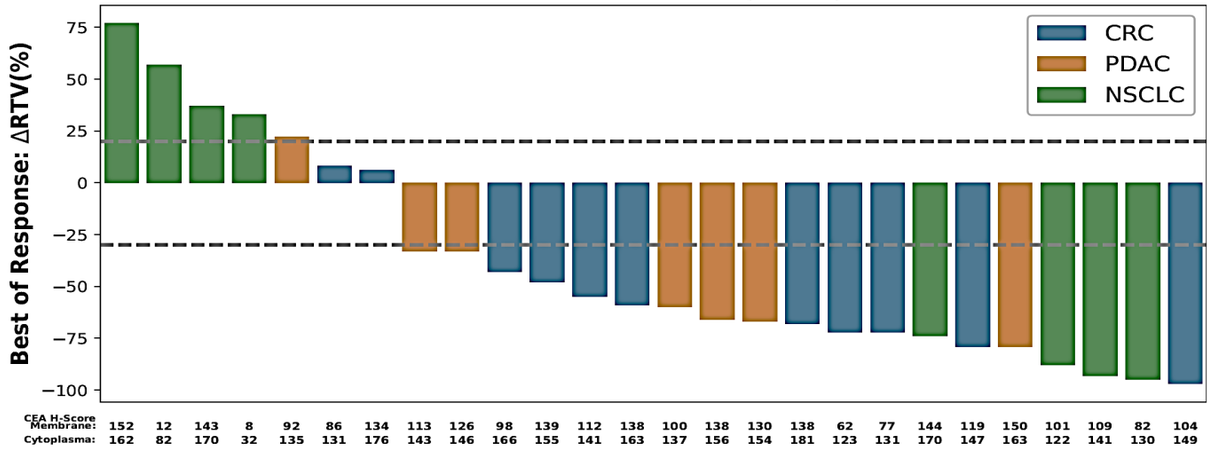
AN4035 shows enhanced target-mediated tumor retention vs. payload alone, with significantly improved tumor selectivity over normal tissue

Adlai Nortye

(1) Ratio of tumor/tissue concentration (or AUC exposure) over blood concentration; (2) Concentrations in these tissues were below the limit of quantification (BQL) at 504 hr.

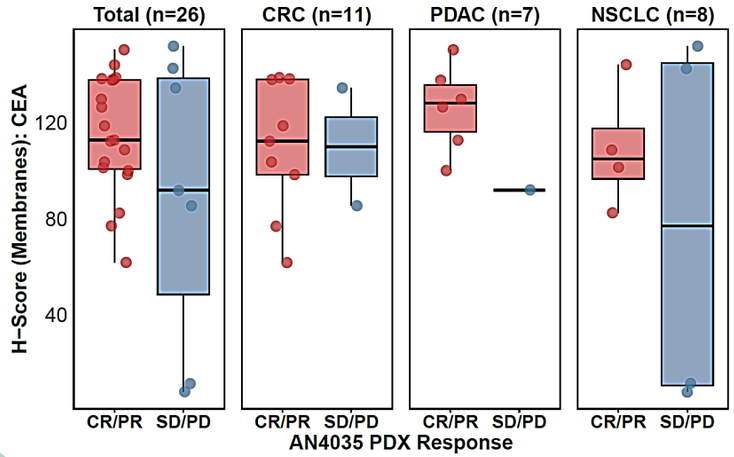
Compelling Anti-Tumor Activity in Single-Mouse PDX Trials

AN4035 10mpk QW demonstrated compelling tumor regression in mouse PDX trials[1]

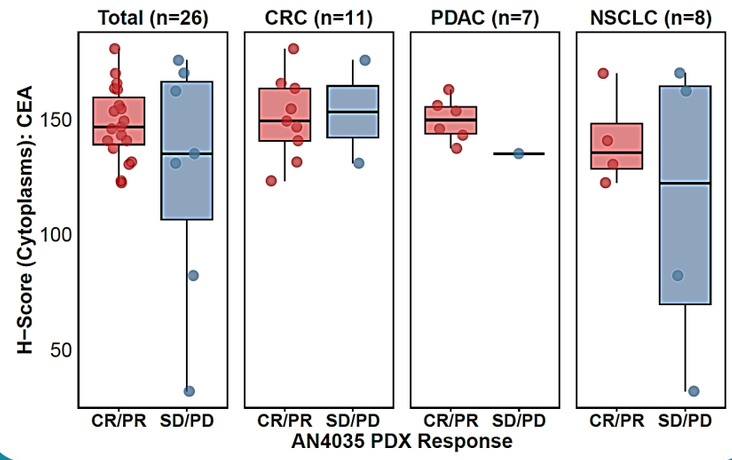


Indication	CR	PR	SD	PD	ORR	DCR
CRC	1	8	2	0	**82%**	**100%**
PDAC	0	6	0	1	**86%**	**86%**
NSCLC	3	1	0	4	**50%**	**50%**
Total	4	15	2	5	**73%**	**81%**

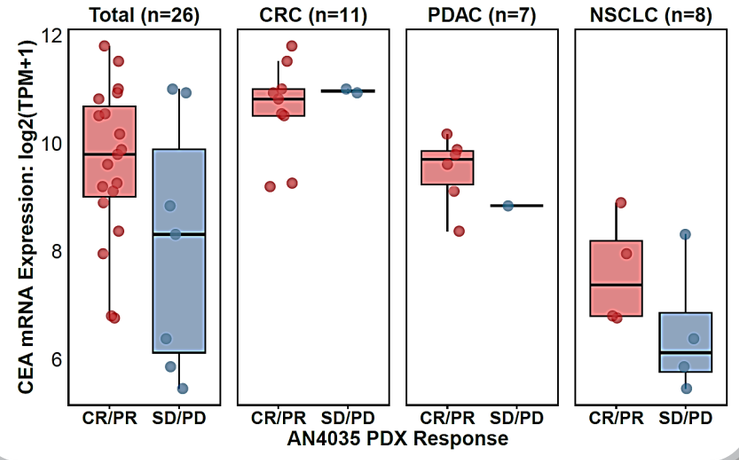
H-score[2] (membrane)



H-score[2] (cytoplasm)



mRNA



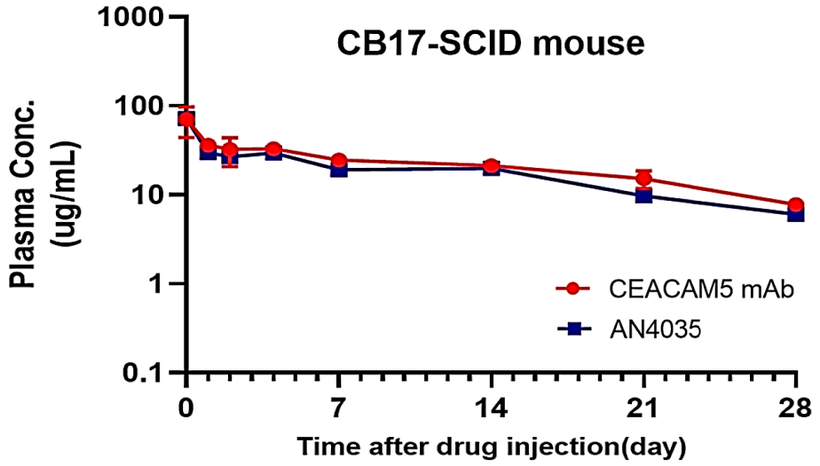
PDX, patient-derived xenograft

(1) The response was determined by comparing tumor volume change at time t to its baseline with ΔRTV = (Vt-V0) / V0 × 100; Criteria for response were adapted from RECIST clinical criteria; Complete Response (CR): at least an 85% decrease in the tumor volume compared to baseline; Partial Response (PR):At least a 30% decrease in the tumor volume compared to baseline; Progressive Disease (PD):At least a 20% increase in the tumor volume compared to baseline; Stable Disease (SD):Neither sufficient shrinkage to qualify for PR nor sufficient increase to qualify for PD; ORR, overall response rate (CR+PR); DCR, disease control rate (CR+PR+SD); (2) H-score measures target protein expression levels using immunohistochemistry

Source: AN4035 Late-Breaking Oral Presentation, 2025 AACR-NCI-EORTC International Conference: Molecular Targets and Cancer Therapeutics.

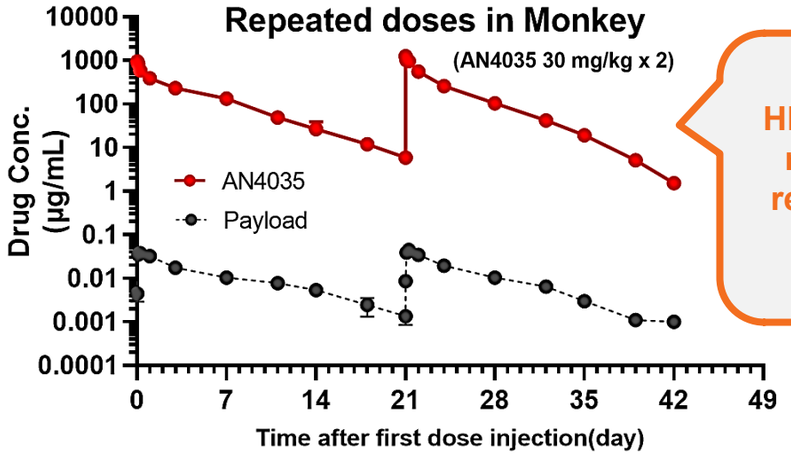
Favorable PK and Tolerability in Monkeys

AN4035 showed an antibody-like PK profile, comparable to CEACAM5 mAb in mice[1]



Compound	Dose	C_0 (μg/mL)	AUC (h*μg/mL)	$T_{1/2}$ (days)
AN4035	3 mg/kg, IV	72	13,832	8.2
CEACAM5 mAb	3 mg/kg, IV	71	17,218	9.7

AN4035 achieved high exposure with good tolerability (no skin or GI toxicities) in monkey[1,2]



HNSTD has not been reached at 30 mpk

Compound	Dose	C_0 (μg/mL)	AUC (h*μg/mL)	$T_{1/2}$ (days)
AN4035	30 mg/kg, IV	942	60,377	3.3
Payload	30 mg/kg, IV	0.038	5.40	4.3

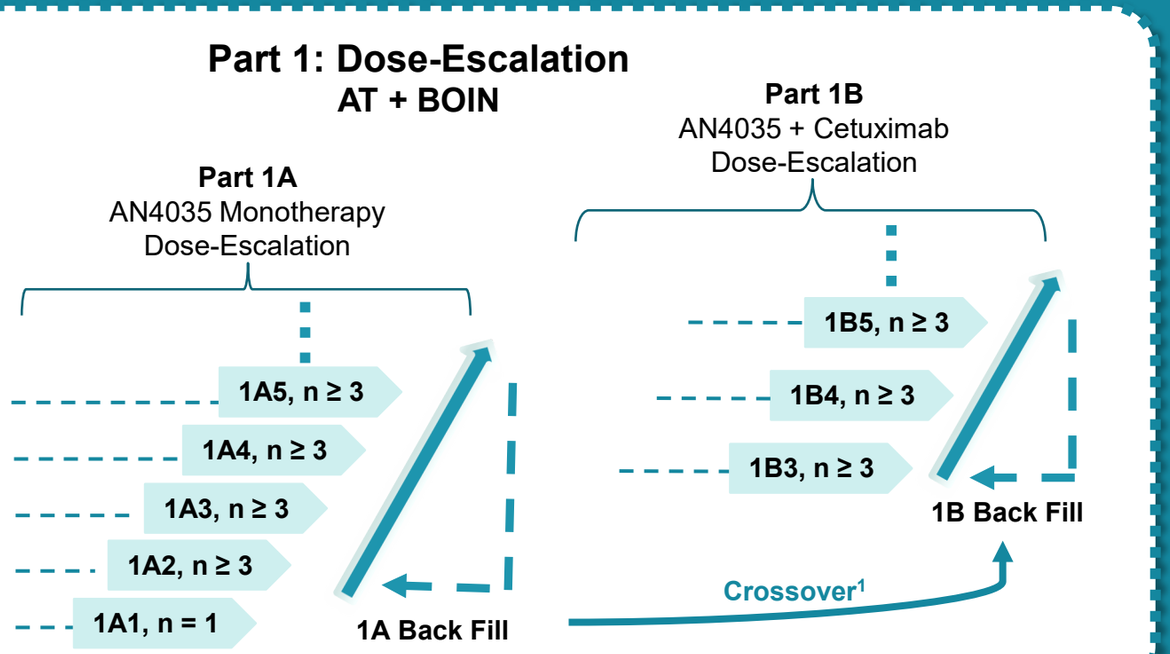
Adlai Nortye

AUC, area under the curve; CEACAM5, Carcinoembryonic antigen-related cell adhesion molecule 5; C_0, initial plasma concentration; HNSTD, highest non-severely toxic dose; mpk, milligrams per kilogram; PK, pharmacokinetics; $T_{1/2}$, half-life

1) total antibody in plasma; total antibody and payload concentration was measured in plasma and whole blood, respectively; 2) PK data from one monkey after the second AN4035 administration were excluded due to ADA development

Source: AN4035 Late-Breaking Oral Presentation, 2025 AACR-NCI-EORTC International Conference: Molecular Targets and Cancer Therapeutics.

AN4035 Global Phase I Multi-Regional Clinical Trial in Australia, U.S. and China



Primary Endpoints
- DLT
- MTD
- Safety
- Tolerability

Secondary Endpoints
- PK
- ORR
- DCR
- PFS

Key Eligibility Criteria:
- Advanced / metastatic solid tumors
- Progression after standard treatment
- RAS-mutation (KRAS / NRAS / HRAS)
- Enriched CEACAM5 expression
- ECOG 0-1
- No prior pan-RAS(ON) inhibitors / ADCs (*except backfill cohorts*)

Part 2: CRC Expansion Cohorts

Cohort 2A:
3L+ RASmut CRC: AN4035 Monotherapy

Cohort 2B:
3L+ RASmut CRC: AN4035 + Cetuximab

AT, Accelerated Titration; BOIN, Bayesian optimal interval; CRC, colorectal cancer; DCR, disease control rate; DLT, dose limiting toxicity; MTD, maximum tolerated dose; ORR, objective response rate; PFS progression-free survival; PK, pharmacokinetics; RDE, recommended dose for expansion

(1) Participants in Part 1A who experience progressive disease (PD), as determined by either clinical or radiographic evaluation during AN4035 monotherapy, may crossover into backfill cohorts of Part 1B, provided that they meet the crossover eligibility criteria.

Summary of AN4035 Program

AN4035 is a first-in-class CEACAM5-targeting ADC armed with a highly potent pan-RAS(ON) inhibitor payload:

- ✓ **Stability & Pharmacokinetics:** Favorable thermal and plasma stability with desirable pharmacokinetic properties
- ✓ **Payload Retention, Cytotoxicity, & Bystander Effect:** Strong intracellular payload retention, driving nanomolar to picomolar cytotoxicity in CEACAM5-positive/RAS-addicted cancer cell lines, along with a potent bystander-killing effect
- ✓ **Tumor Regression:** Robust anti-tumor activity with deep regression in CEACAM5-positive / RAS-addicted CDX and PDX models, as monotherapy or in combination
- ✓ **Selectivity & Retention:** Enhanced target-mediated tumor retention versus payload alone, with significantly improved tumor selectivity over normal tissue
- ✓ **Therapeutic Window:** Favorable therapeutic index in cynomolgus monkeys

AN4035 is advancing in the clinic:

- ➢ Received **regulatory clearance to begin clinical trial** in U.S. and Australia in August 2026; China IND in process
- ➢ **First patient was dosed** in monotherapy arm in August 2026
- ➢ Initial clinical **data readout is expected in H2 2027**

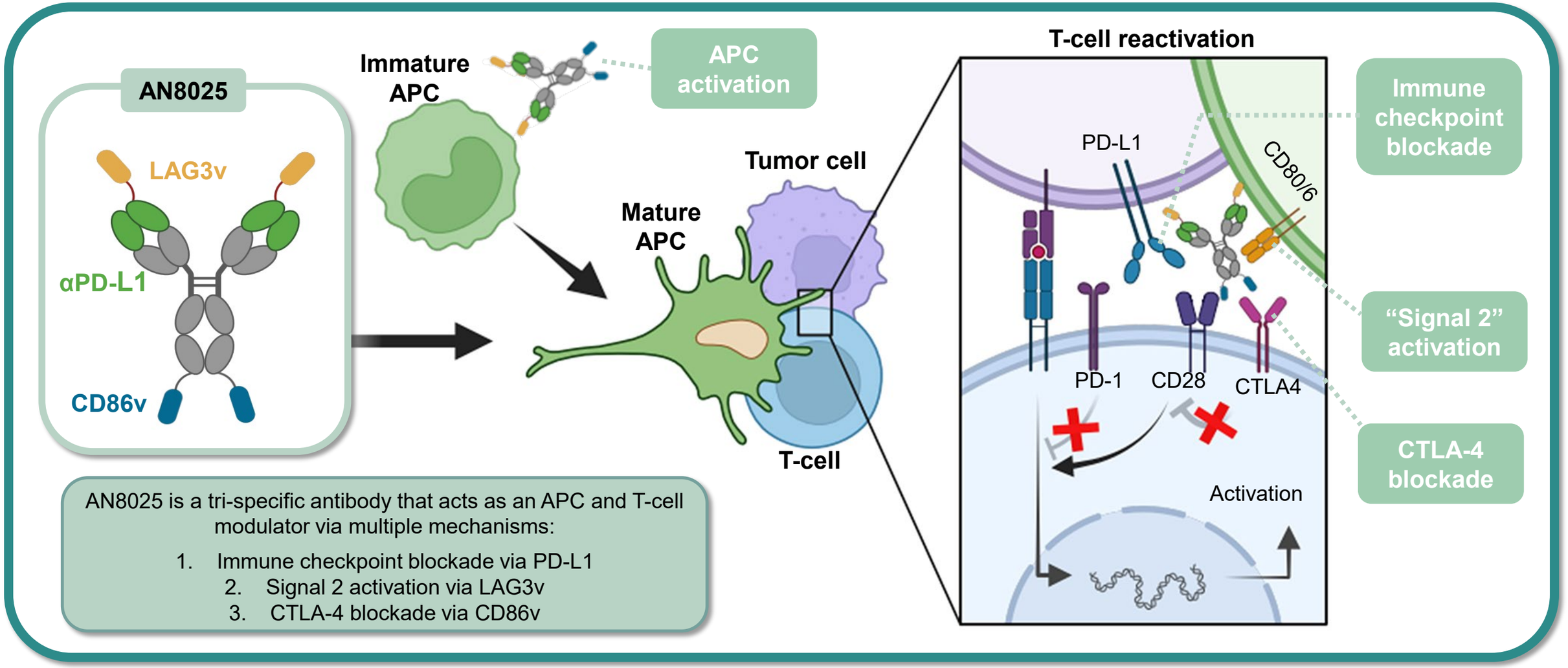


AN8025

Potential First-in-Class, Multi-Functional T Cell/APC Modulator

Adlai Nortye

AN8025 Mechanism of Action



AN8025 is a tri-specific antibody that acts as an APC and T-cell modulator via multiple mechanisms:

1. Immune checkpoint blockade via PD-L1
2. Signal 2 activation via LAG3v
3. CTLA-4 blockade via CD86v

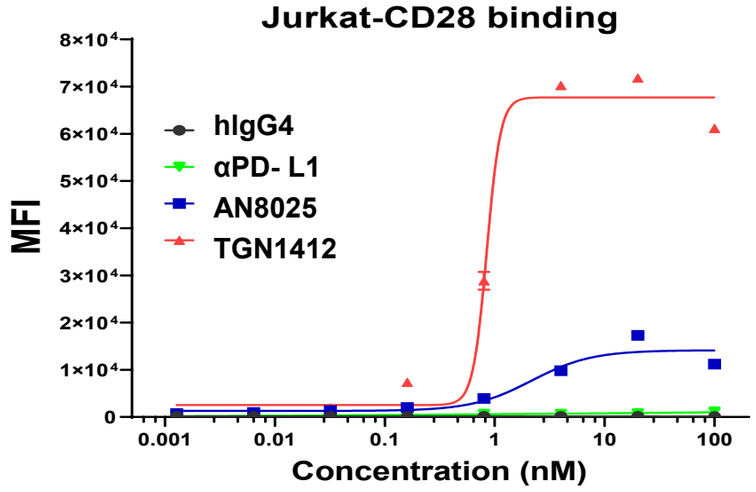
APC, antigen-presenting cell; CD86v, variant of CD86 protein; LAG3v, variant of LAG3 protein

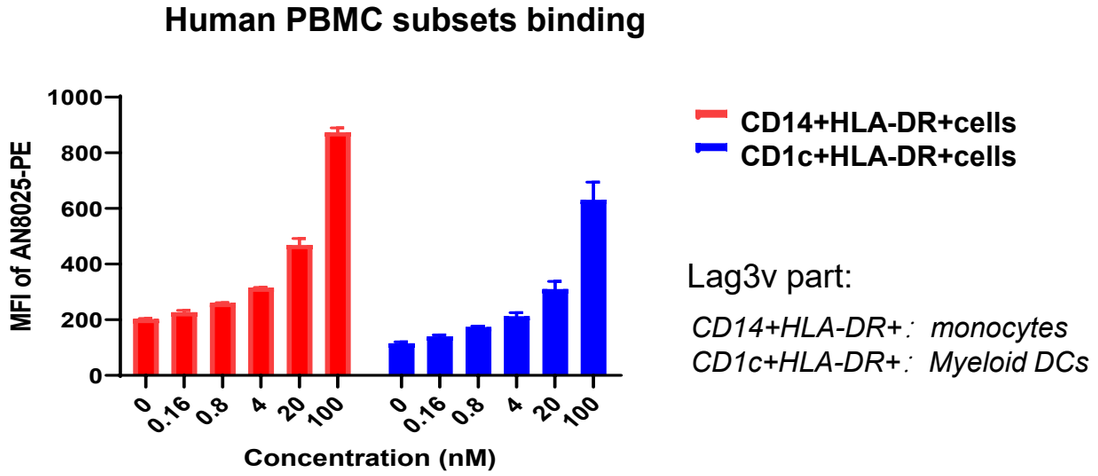
AN8025 Binding and Target Interaction

Biochemical binding of AN8025 to its molecular targets

hCTLA4		hCD28		hPD-L1	
Samples	K_D (M)	Samples	K_D (M)	Samples	K_D (M)
IgG4 isotype	NA	IgG4 isotype	NA	IgG4 isotype	NA
AN8025	5.63×10^{-9}	AN8025	1.02×10^{-8}	AN8025	1.86×10^{-9}
αPD-L1	NA	αPD-L1	NA	αPD-L1	1.42×10^{-9}
Tremelimumab	7.32×10^{-10}	TGN1412	2.88×10^{-12}		
Ipilimumab	7.37×10^{-9}				

Cellular interaction of AN8025 to hCD28+ or HLA-DR+ cells





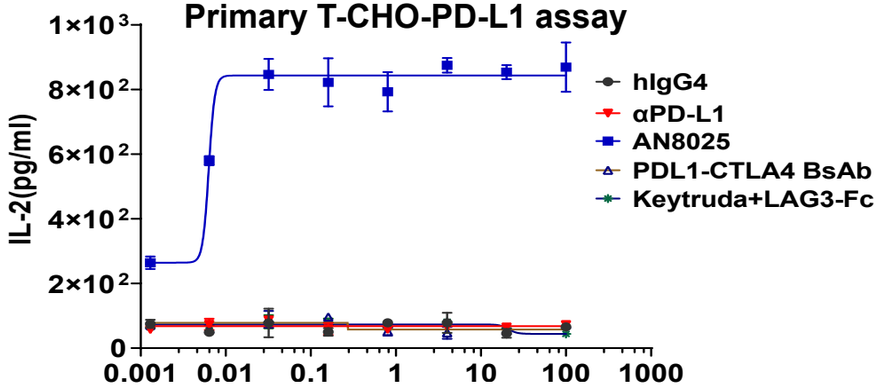
Lag3v part:

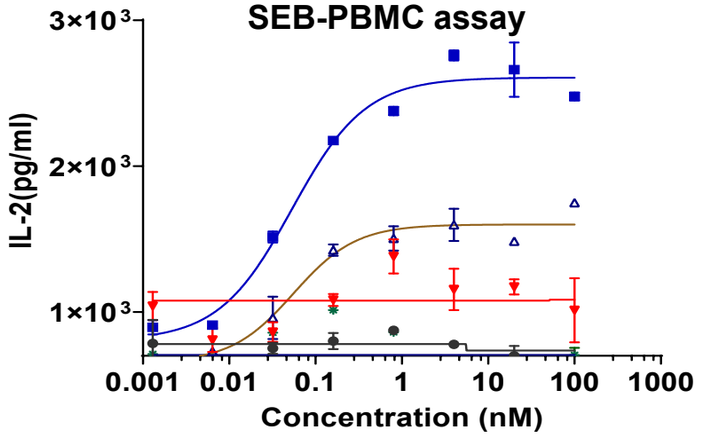
CD14+HLA-DR+: monocytes
CD1c+HLA-DR+: Myeloid DCs

Source: AN8025 Poster, Abstract #3420, 2025 AACR Annual Meeting.

AN8025 Induces Superior T-Cell Responses

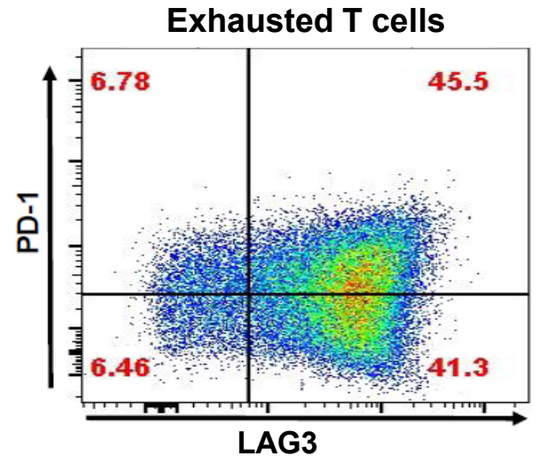
AN8025 drives stronger T-cell activation than αPD-L1 or αPD-(L)1 combinations

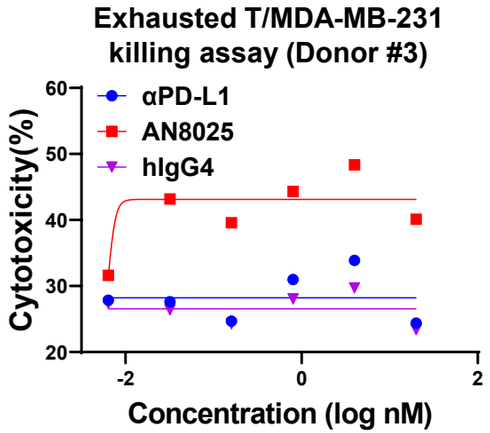


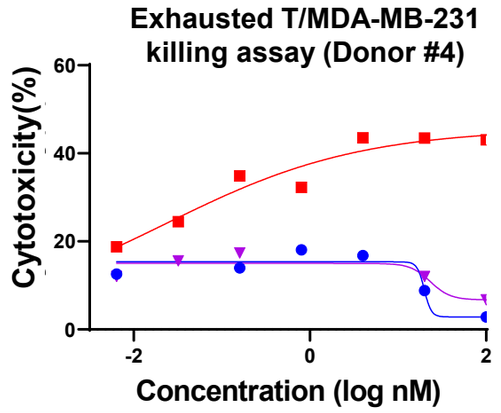


AN8025 reinvigorates exhausted T cells for efficient tumor cell killing

Exhausted T cells induced by sustained TAA+ tumor cells stimulation with TAA-targeted T cell engager over 14 days



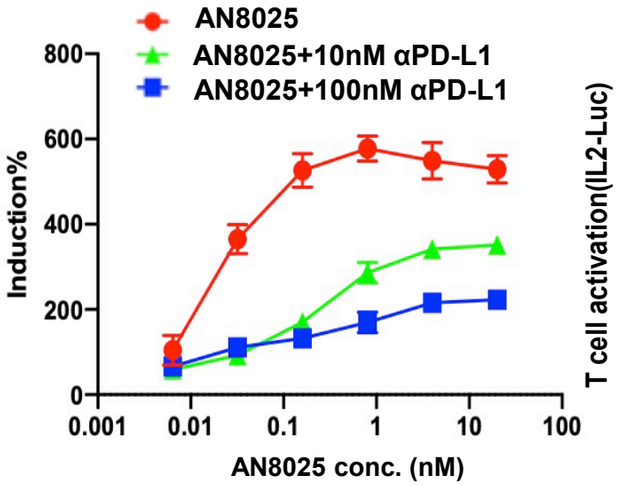


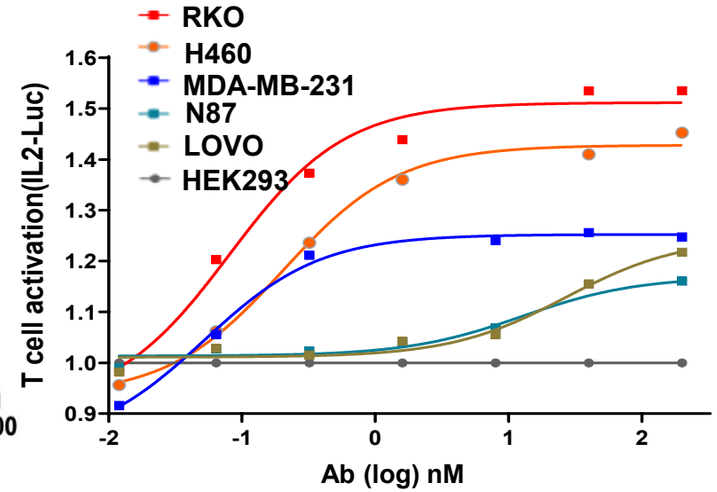


Source: AN8025 Poster, Abstract #3420, 2025 AACR Annual Meeting.

AN8025 Promotes T-Cell Activation in a PD-L1 Dependent Manner

Enhanced T-cell activation by AN8025 is PD-L1-dependent

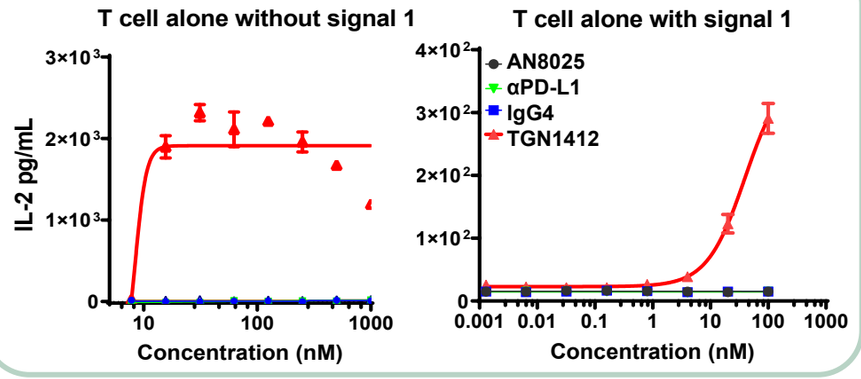




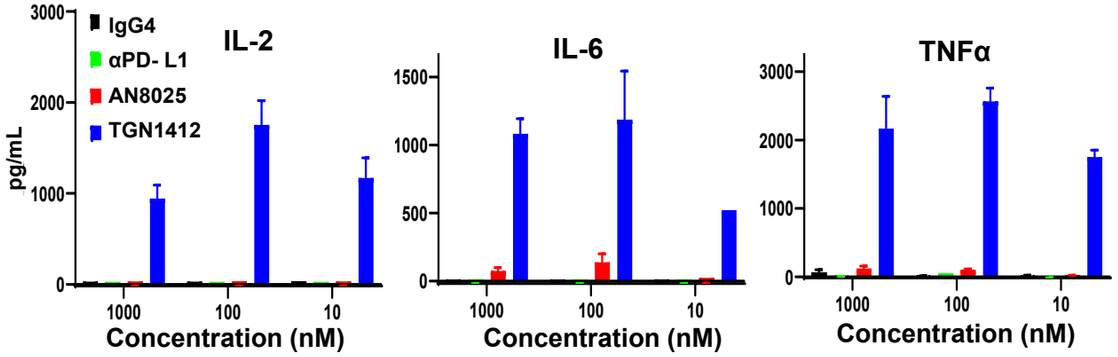
Cell line	hPD-L1 MFI	EC50 (nM)
RKO (PD-L1 high)	40,333	0.08
H460 (PD-L1 Med)	24,909	0.202
MDA-MB-231 (PD-L1 Med)	10,849	0.052
N87 (PD-L1 low)	211	12.63
LOVO (PD-L1 low)	445	26.85
HEK293 (PD-L1 neg)	40	NA

Unlike TGN1412, AN8025 does not act as a T-cell super-agonist

T-cell alone activation with or without signal 1



Cytokine release from hPBMC induced by immobilized Abs

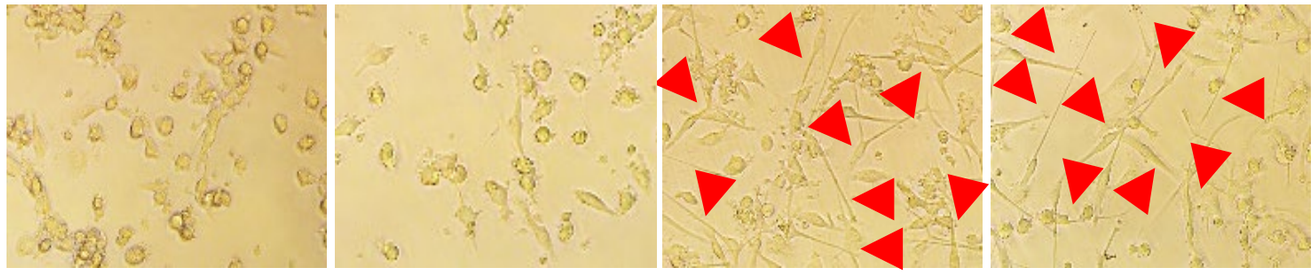


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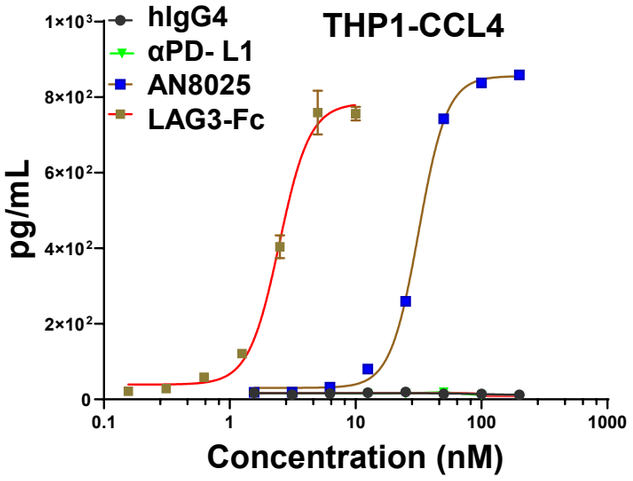
Source: AN8025 Poster, Abstract #3420, 2025 AACR Annual Meeting.

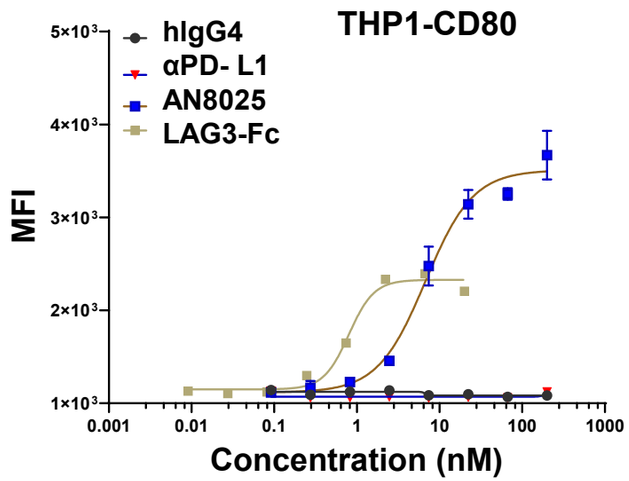
AN8025 Enhances Both the Quantity and Functionality of APCs

APC Quantity[1]

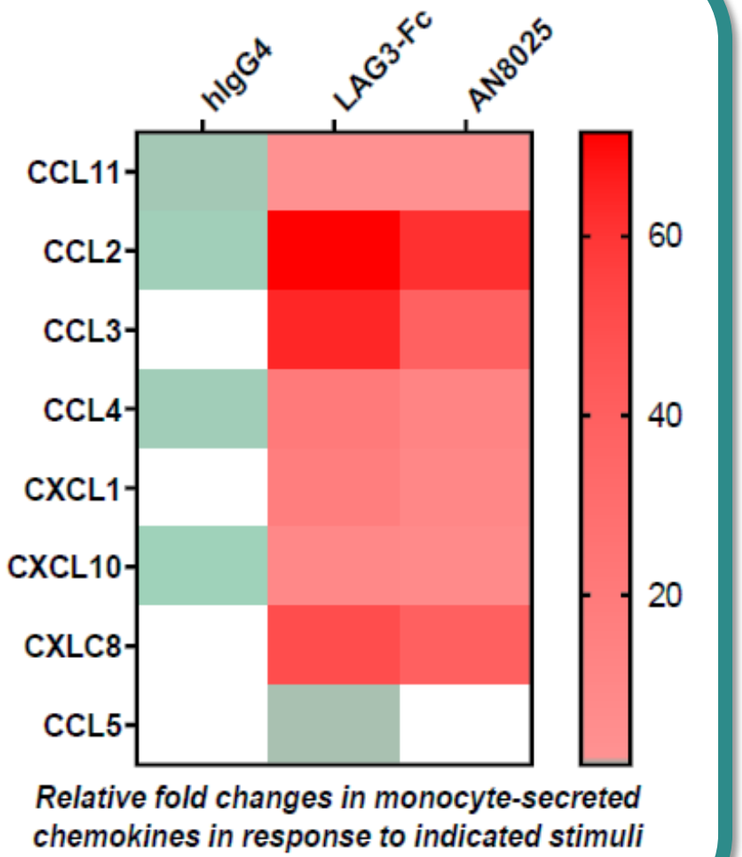


APC Quality





Human monocytes



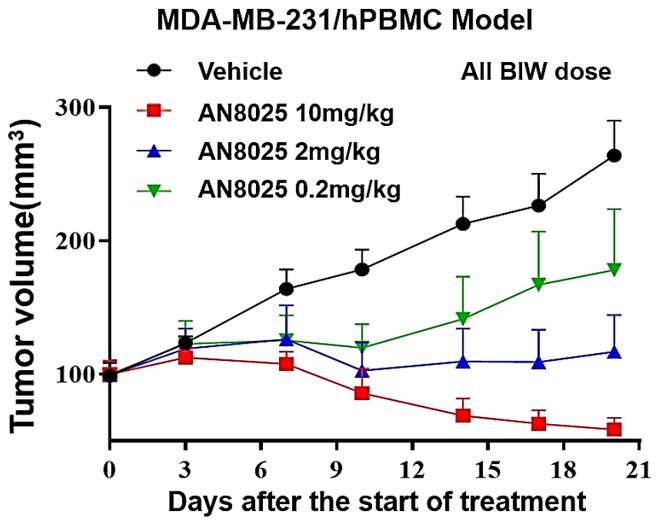
Relative fold changes in monocyte-secreted chemokines in response to indicated stimuli

APC, antigen-presenting cell

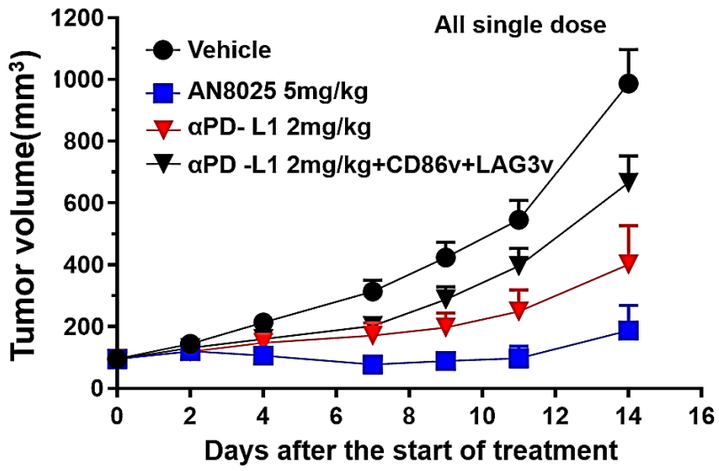
(1) Monocyte derived immature dendritic cell morphology

Source: AN8025 Poster, Abstract #3420, 2025 AACR Annual Meeting.

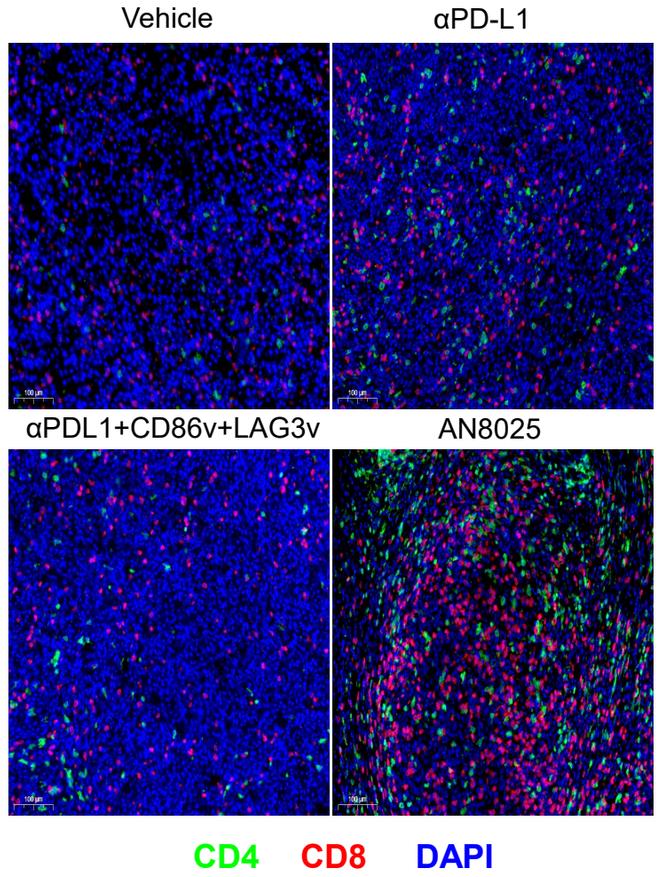
AN8025 Exhibits Potent Anti-Tumor Activity In Vivo



Group	TMV (mm^3)	TGI (%)	p-value
Vehicle	264 ± 26		
AN8025, 10 mg/kg	58 ± 9	125	<0.001
AN8025, 2 mg/kg	117± 27	89	0.008
AN8025, 0.2 mg/kg	178 ± 46	52	0.402

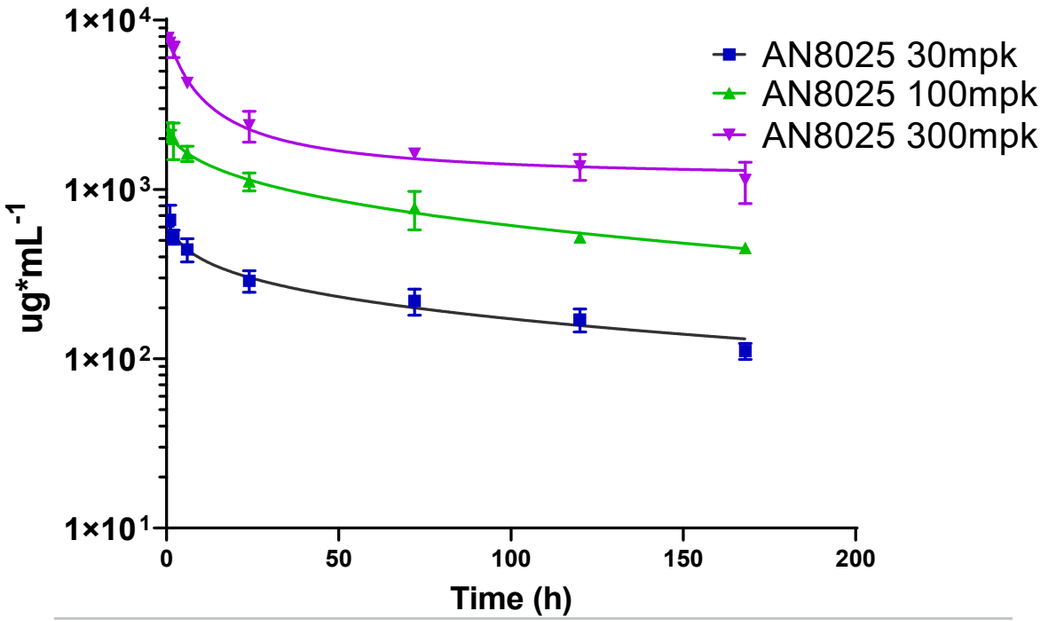


Group	TMV (mm^3)	TGI (%)	CR (%)	p-value
Vehicle	987±110			
AN8025	187±82	90	**40**	<0.001
αPD-L1	401±126	66	10	0.001
αPD-L1 + CD86v + LAG3v	666±87	36	0	0.085

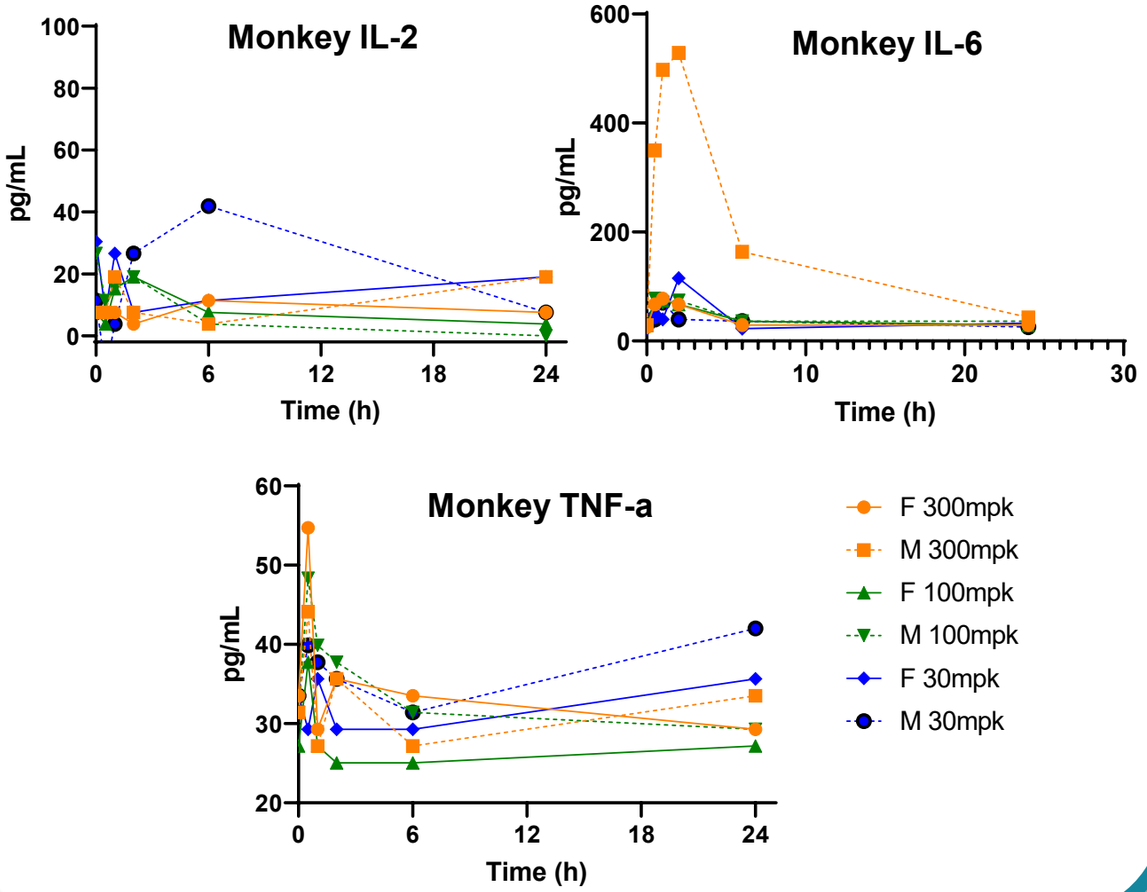


Source: AN8025 Poster, Abstract #3420, 2025 AACR Annual Meeting.

Favorable PK and Safety Profiles in Non-Human Primates



Dose	C_0 (μg/ml)	AUC (h*μg/ml)	$T_{1/2}$ (h)
30 mpk	706	37,900	107.35
100 mpk	2,200	135,000	177.15
300 mpk	7,905	323,500	248.7



Summary of AN8025 Program

AN8025 is a potential first-in-class, next-generation cancer immunotherapy with a multi-mechanistic mode of action:

- ✓ **T-Cell Activation:** Drove robust T-cell activation in a PD-L1–dependent manner.
- ✓ **Tumor Clearance:** Promoted both exhausted and non-exhausted T-cell populations to support effective tumor clearance.
- ✓ **APC Enhancement:** Enhanced both the quantity and functional quality of antigen-presenting cells (APCs).
- ✓ **Anti-Tumor Efficacy:** Demonstrated potent anti-tumor efficacy in vivo.
- ✓ **Tolerability Profile:** Showed a favorable safety and tolerability profile in non-human primates (NHPs).

AN8025 is in the clinic:

- ➢ The first patient was dosed in December 2025 in the ongoing global Phase 1 clinical trial in Australia and China
- ➢ Dose escalation is anticipated to be completed by 2026 YE