Adlai Nortye

NASDAQ: ANL

Corporate Presentation

April 2026

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Pipeline Chart:

Product	MOA	Indication	Discovery	IND Enabling	Phase 1a	Phase 1b	Phase 2	Phase 3	Upcoming Milestone	Worldwide Rights
AN9025	**pan-RAS(ON)i**	RAS-addicted tumors							Ph1 dose escalation completion in H1 2027	Ex-China[1]
AN4035	**CEACAM5 pan-RAS(ON)i ADC**	CEACAM5-enriched RAS-addicted solid tumors							IND submission in mid-2026	Global
AN8025	**αPD-L1 /Lag3v /CD86v**	Advanced tumors							Ph1 dose escalation completion in 2026 YE	Global
Other Assets										
AN4005	**Small molecule PD-L1i**	Advanced tumors							Clinical update presentation at conference expected in H2 2026	Ex-China[2]
Palupiprant (AN0025)	**EP4**	Neoadj RC (+CRT)[3]							Topline results of Ph2 study in H1 2027	Global except Japan, Korea, Taiwan and Southeast Asian Countries
		LA EC (+CRT)[3]							Clinical update in H2 2026	

Abbreviations: 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 Macao; 2. Xiamen Biotime Biotechnology Co., Ltd., holds exclusive rights to develop, manufacture, and commercialize AN4005 in mainland China, Hong Kong, Macao and Taiwan; 3. Investigator Initiated Trials

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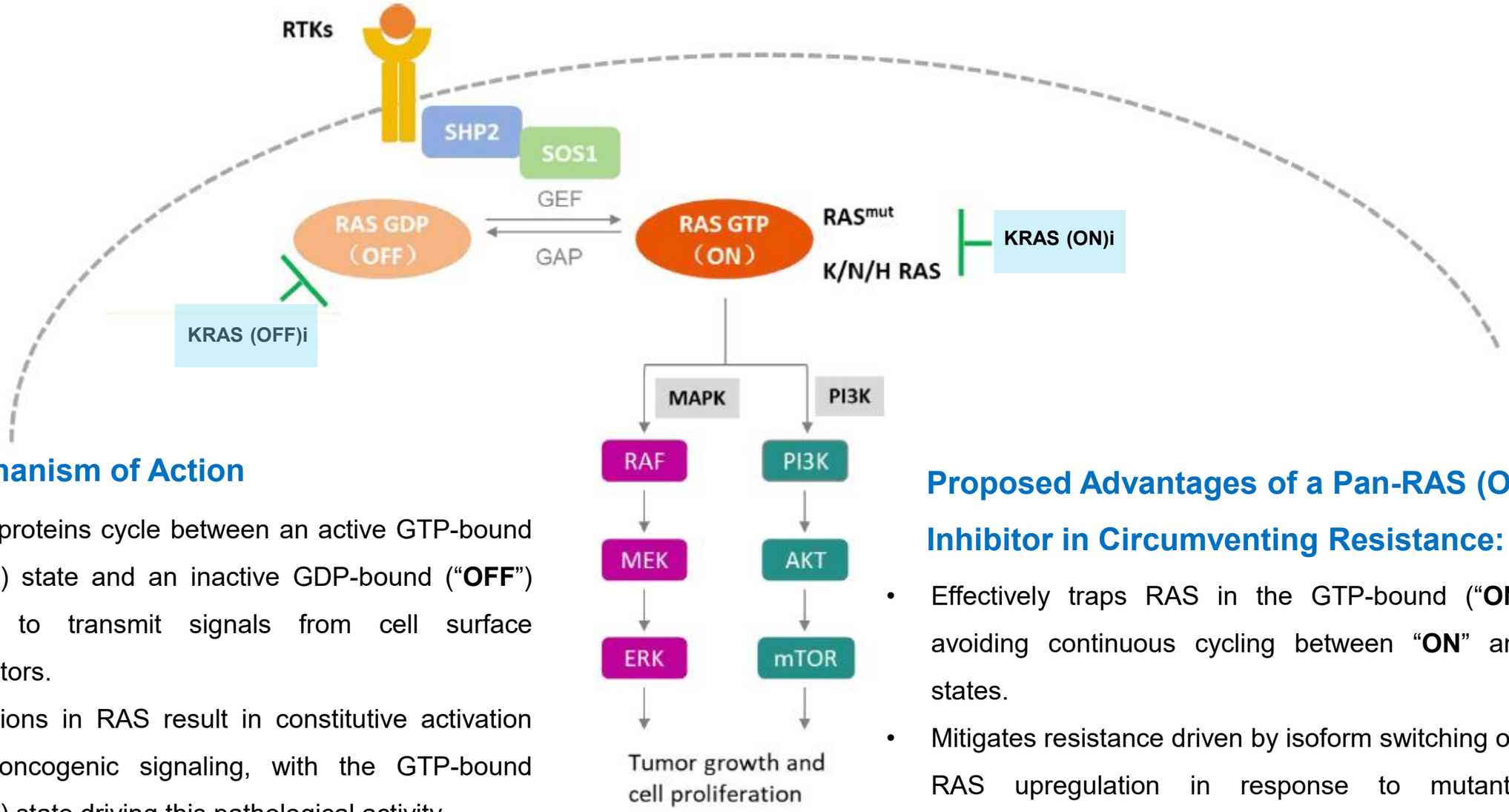
AN9025: An Oral Small-Molecule Pan-RAS(ON) Inhibitor

Why Targeting Pan-RAS(ON)



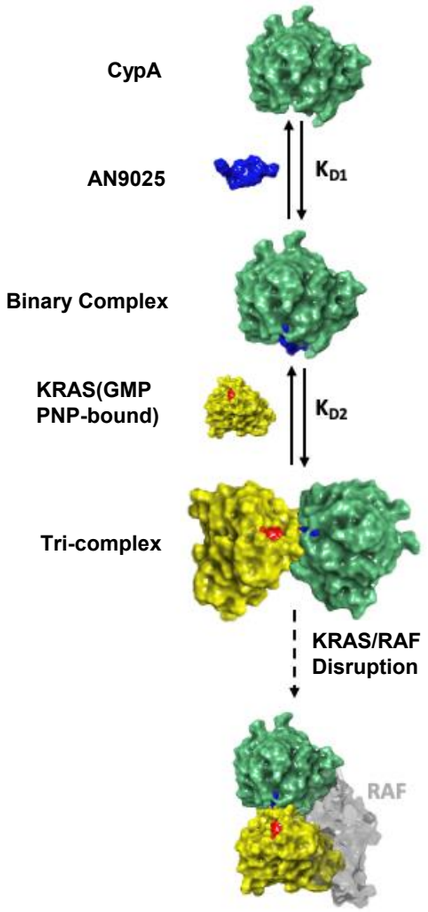
Mechanism of Action

- RAS proteins cycle between an active GTP-bound (“**ON**”) state and an inactive GDP-bound (“**OFF**”) state to transmit signals from cell surface receptors.
- Mutations in RAS result in constitutive activation and oncogenic signaling, with the GTP-bound (“**ON**”) state driving this pathological activity.

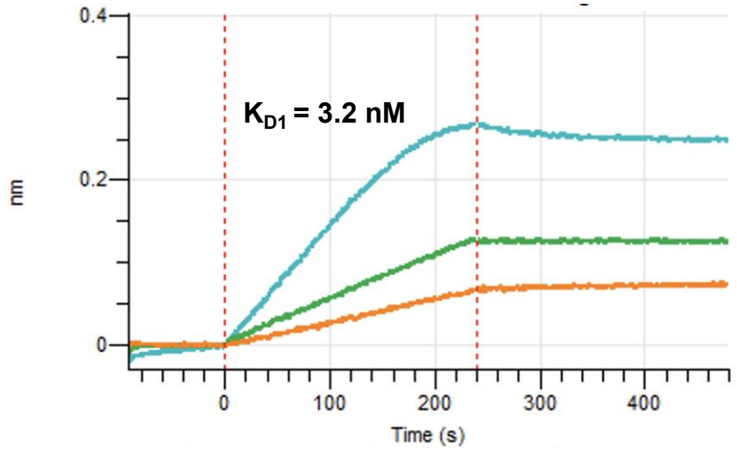
Proposed Advantages of a Pan-RAS (ON) Inhibitor in Circumventing Resistance:

- Effectively traps RAS in the GTP-bound (“**ON**”) state, avoiding continuous cycling between “**ON**” and “**OFF**” states.
- Mitigates resistance driven by isoform switching or wildtype RAS upregulation in response to mutant-selective therapies

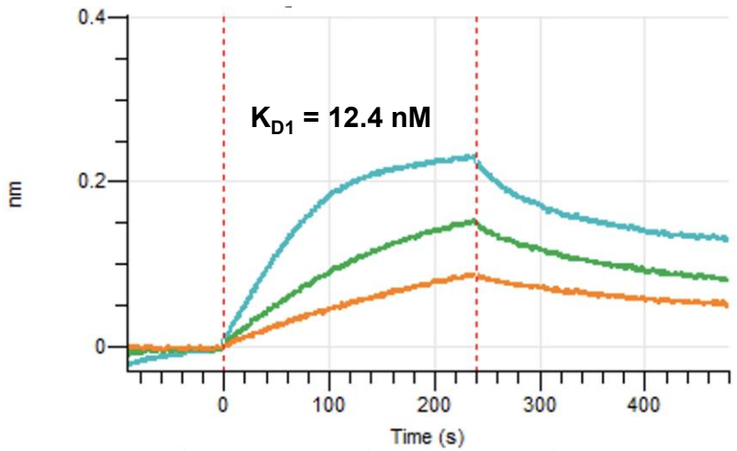
AN9025's Binding Affinity for CypA Suggests It May Offer More Sustained RAS Inhibition vs. RMC-6236



Kinetic Analysis of AN9025 Binding to CypA on Octet



Kinetic Analysis of RMC-6236 Binding to CypA on Octet



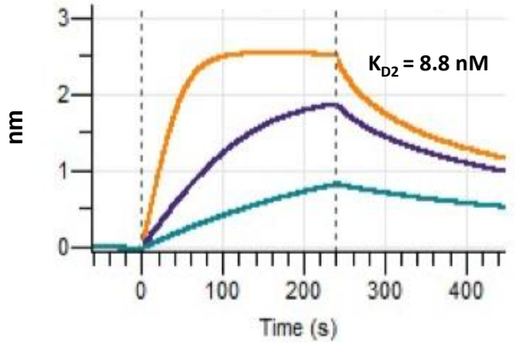
Binary Affinity	K_{D1} (nM)	ka (1/[M*S])	kdis (1/s)
AN9025	**3.2**	**1.6 E+05**	**5.0 E-04**
RMC-6236	12.4	2.8 E+05	3.4 E-03

- AN9025 demonstrates a binding affinity to cyclophilin A (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.

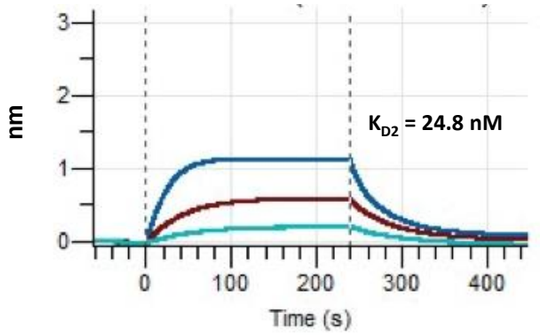
Source: AN9025 Poster, Abstract #4377, 2025 AACR Annual Meeting

AN9025 Shows Stronger Tri-Complex Binding Affinity vs. RMC-6236

AN9025 (GMPPNP-bound KRAS^{G12D})



RMC-6236 (GMPPNP-bound KRAS^{G12D})

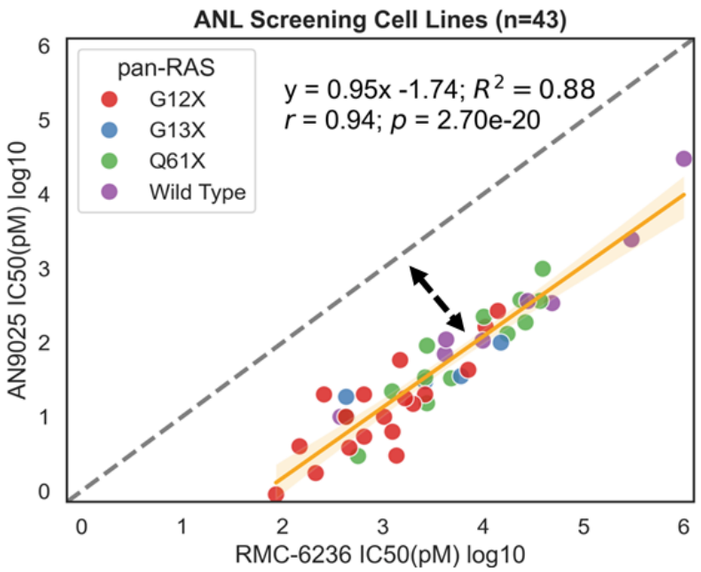


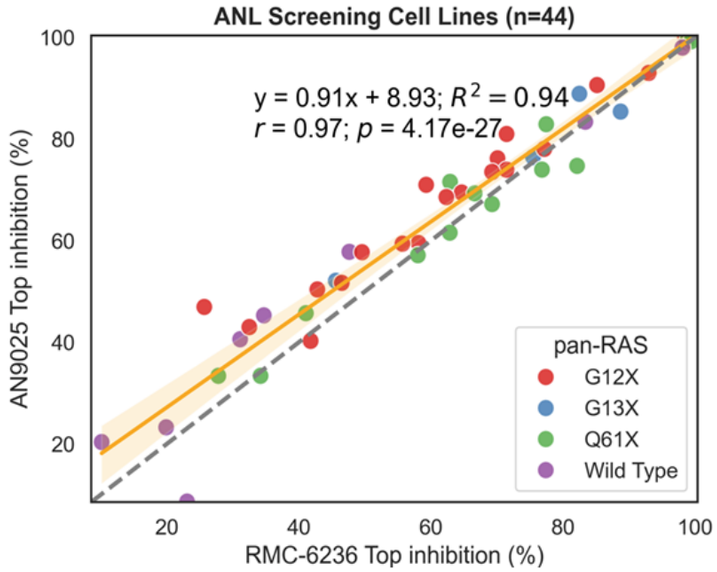
Mutant RAS(ON) Protein	AN9025			RMC-6236		
	K_D (nM)	ka (1/[M*S])	kdis (1/s)	K_D (nM)	ka (1/[M*S])	kdis (1/s)
KRAS^{G12D}	8.8	4.9 E+05	4.3 E-03	24.8	5.4 E+05	1.3 E-02
KRAS^{G12V}	8.3	3.2 E+05	2.7 E-03	68.4	1.8 E+05	1.2 E-02
KRAS^{G12C}	4.5	4.1 E+05	1.9 E-03	17.8	4.5 E+05	8.0 E-03
KRAS^{G12S}	12.6	1.6 E+05	2.0 E-03	48.5	1.9 E+05	9.1 E-03
KRAS^{G12R}	15.5	2.4 E+05	3.8 E-03	80.5	2.3 E+05	1.8 E-02
KRAS^{G12A}	11.9	2.1 E+05	2.5 E-03	60.3	1.8 E+05	1.0 E-02
KRAS^{G13D}	15.5	2.5 E+05	3.9 E-03	69.7	2.5 E+05	1.8 E-02
NRAS^{Q61H}	11.0	2.5 E+05	2.8 E-03	45.4	2.2 E+05	1.0 E-02
KRAS^{WT}	10.0	2.8 E+05	2.8 E-03	52.8	2.2 E+05	1.2 E-02
NRAS^{WT}	6.6	3.7 E+05	2.4 E-03	33.9	2.6 E+05	8.8 E-03
HRAS^{WT}	4.0	4.9 E+05	2.0 E-03	20.2	3.4 E+05	6.8 E-03

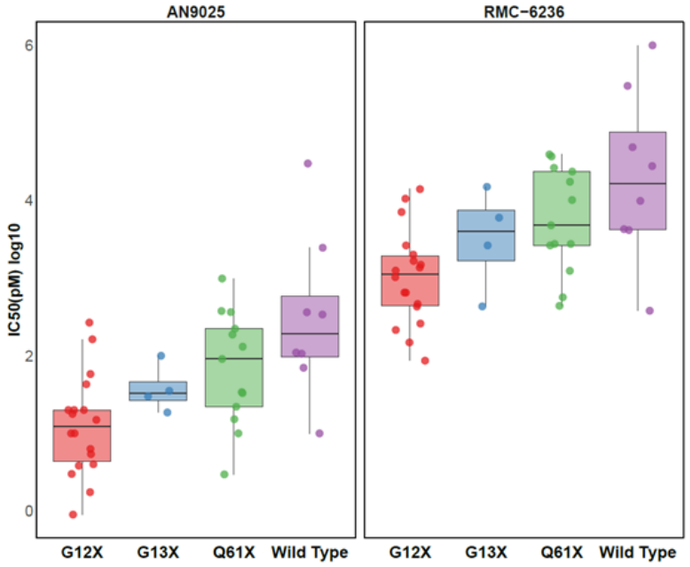
- AN9025 exhibits a **3- to 8-fold higher** binding affinity for tri-complex formation compared to RMC-6236, primarily attributed to its slower dissociation rate that leads to prolonged suppression of RAS signaling.
- AN9025 demonstrates strong binding affinity for the RAS(ON) protein, with no detectable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

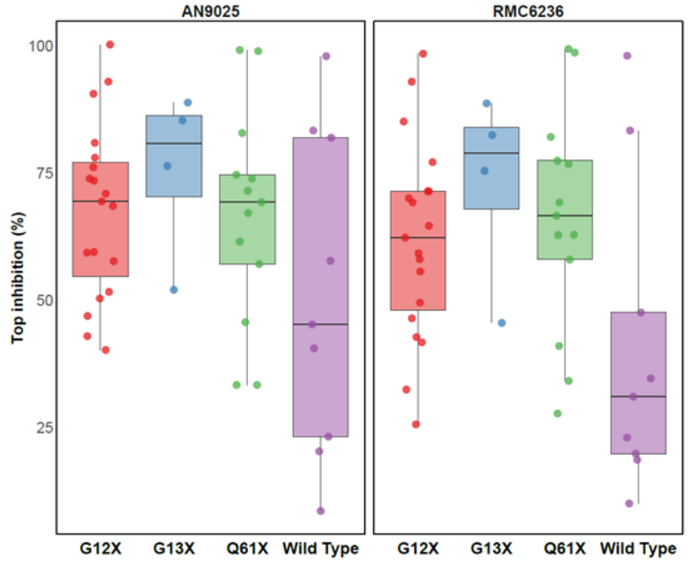
Source: AN9025 Poster, Abstract #4377, 2025 AACR Annual Meeting

AN9025 Demonstrates a Sensitivity Profile Comparable to RMC-6236 Across a Cancer Cell Line Panel but with Significantly Higher Potency









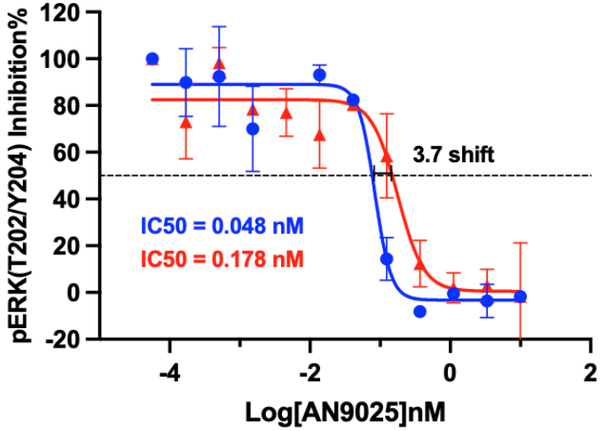
- AN9025 demonstrates approximately **100-fold** more potent inhibition of cell proliferation across RAS mutant cell lines than RMC-6236.

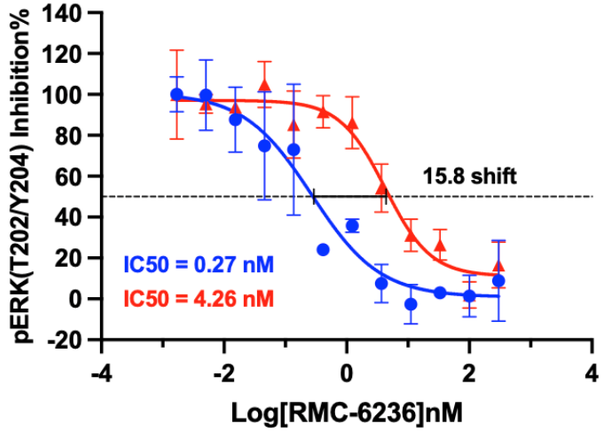
- AN9025 shares similar RAS mutant sensitivity pattern with RMC6236.
(G12X > G13X ≈ Q61X > WT)

Source: AN9025 Poster, Abstract #4377, 2025 AACR Annual Meeting

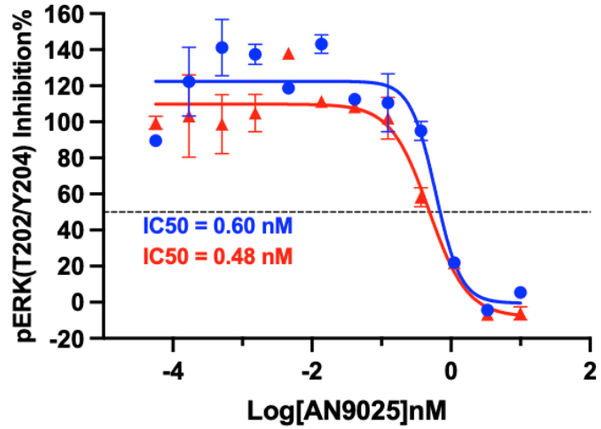
AN9025 Demonstrates More Sustained RAS Inhibition vs. RMC-6236

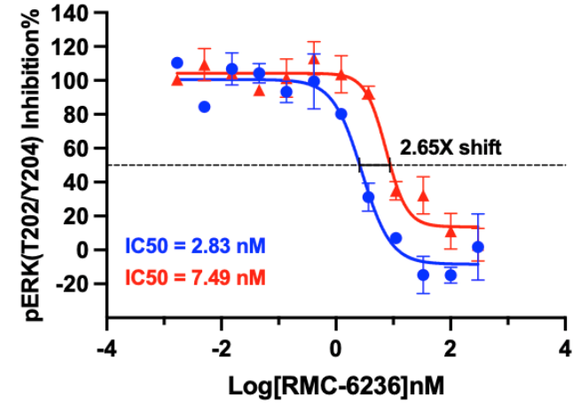
Phospho-ERK(T202/Y204) inhibition (IC_{50}, nM) in HPAC	AN9025	RMC-6236
4 h incubation	0.048	0.27
4 h incubation + 48 h washout	0.18	4.3
IC_{50} Shift	3.7 X	16 X





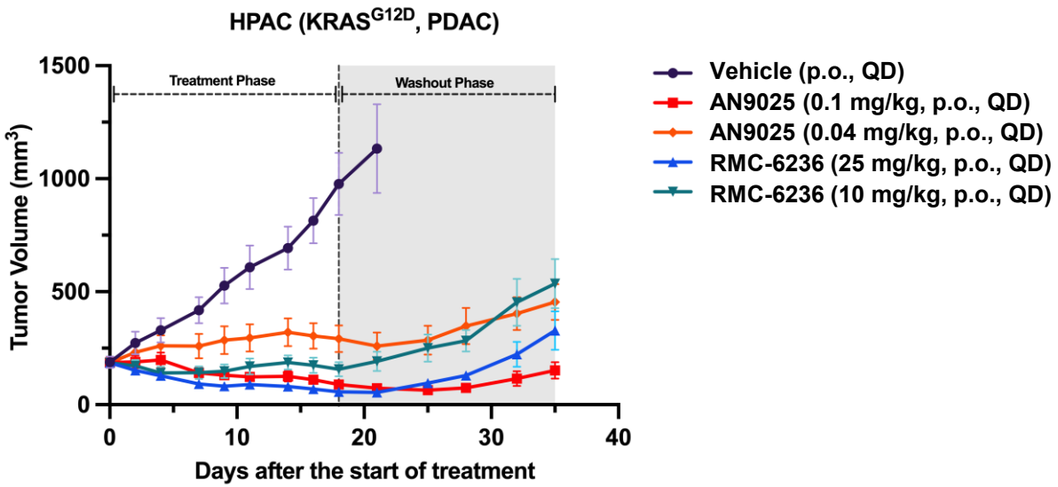
Phospho-ERK(T202/Y204) inhibition (IC_{50}, nM) in PSN1	AN9025	RMC-6236
4 h incubation	0.60	2.83
4 h incubation + 48 h washout	0.48	7.49
IC_{50} Shift	No shift	2.6 X

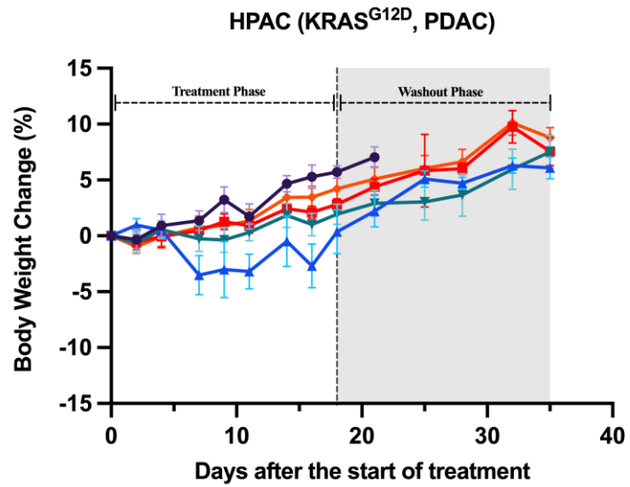




◆ AN9025 enables **prolonged inhibition** of the RAS pathway, maintaining its effects even after the drug is withdrawn.

AN9025 Achieves Deep Tumor Regression and Provides More Sustained Anti-Tumor Effects *vs.* RMC-6236 in HPAC ($KRAS^{G12D}$) CDX Model





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

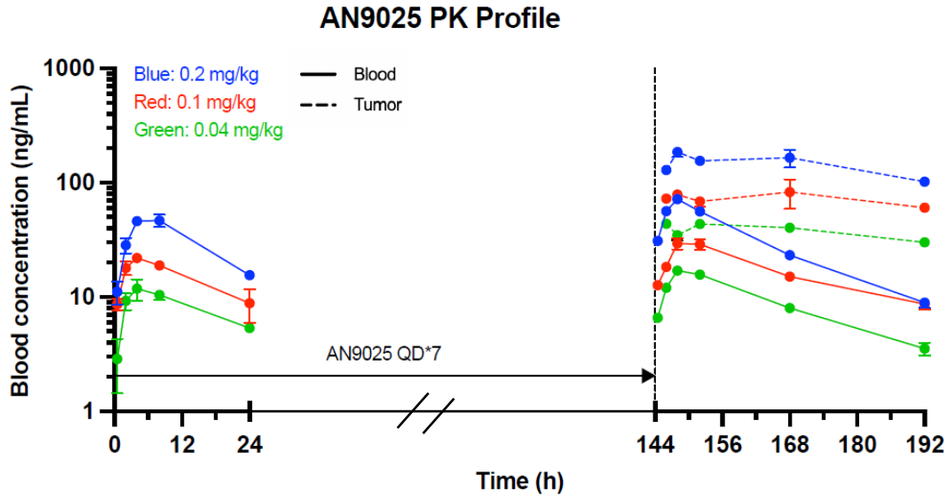
(1). RTV = relative tumor volume; relative to the tumor volume at the start of treatment

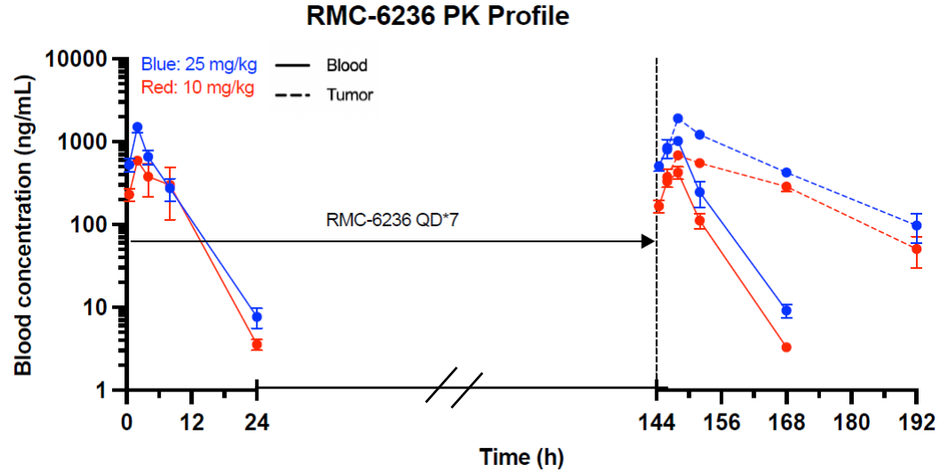
- AN9025 demonstrated comparable antitumor activity to RMC-6236; notably, AN9025 exhibited more sustained efficacy following drug withdrawal.

Source: AN9025 Poster, Abstract #4377, 2025 AACR Annual Meeting.

PK-PD Analysis Further Supports More Durable RAS Inhibition by AN9025 vs. RMC-6236 in HPAC ($KRAS^{G12D}$) CDX Model

This study evaluated the PK–PD correlation at the end of 7 days of continuous oral dosing and also assessed PK after the first dose for comparison



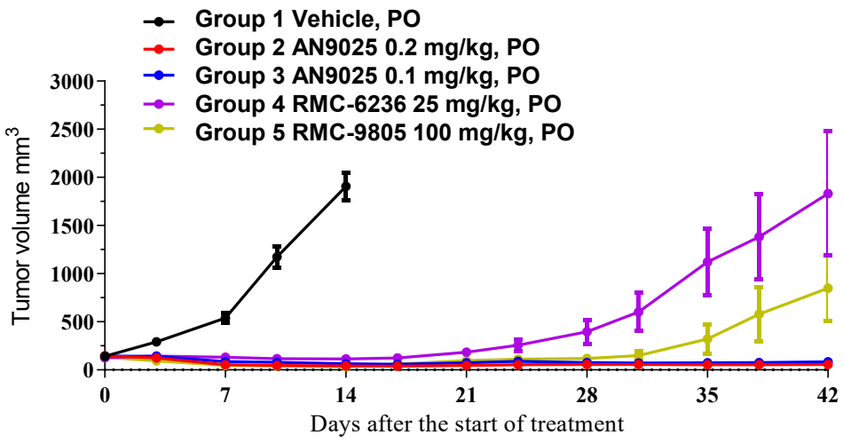


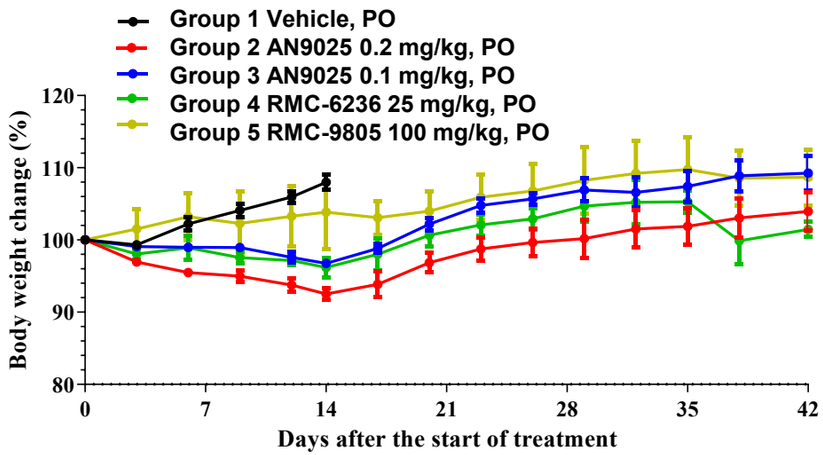
AN9025 Post-Treatment PK/PD Profile (144~192 hr)	0.2mg/kg, p.o., QD*7	0.1mg/kg, p.o., QD*7	0.04mg/kg, p.o., QD*7
$AUC_{144\sim192\ hr}$ tumor (h*ng/g) / blood / plasma (h*ng/mL)	6910 / 1476 / 69	3454 / 826 / 40	1802 / 438 / NA
C_{max} tumor (ng/g) / blood / plasma (ng/mL)	186 / 72 / 6.4	83 / 29 / 2.5	44 / 17 / NA
Tumor DUSP6 inhibition at 168 / 192 hr	96.2% / 96.2%	96.5% / 94.5%	87.2% / 76.2%

RMC-6236 Post-Treatment PK/PD Profile (144~192 hr)	25 mg/kg, p.o., QD*7	10 mg/kg, p.o., QD*7
$AUC_{144\sim192\ hr}$ tumor (h*ng/g) / blood / plasma (h*ng/mL)	29227 / 7583 / 2542	14653 / 3169 / 782
C_{max} tumor (ng/g) / blood / plasma (ng/mL)	1911 / 1018 / 386	683 / 425 / 156
Tumor DUSP6 inhibition at 168 / 192 hr	85.5% / 46.3%	63.8% / 7.9%

- The results indicate that AN9025 has the flexibility to be investigated for an **intermittent dosing** strategy in a clinical setting.

AN9025 Achieves More Prolonged Anti-Tumor Effects Compared to RMC-6236 and RMC-9805 in KP-4 ($KRAS^{G12D}$) CDX Model





Compounds	Dose (mg/kg)	RTV1 (%) on the day of best tumor regression	RTV[1] (%) on Day 42
AN9025	0.2	26.6%	41.0%
	0.1	41.6%	58.2%
RMC-6236	25	81.0% (D14)	1331%
RMC-9805	100	25.7% (D14)	658%

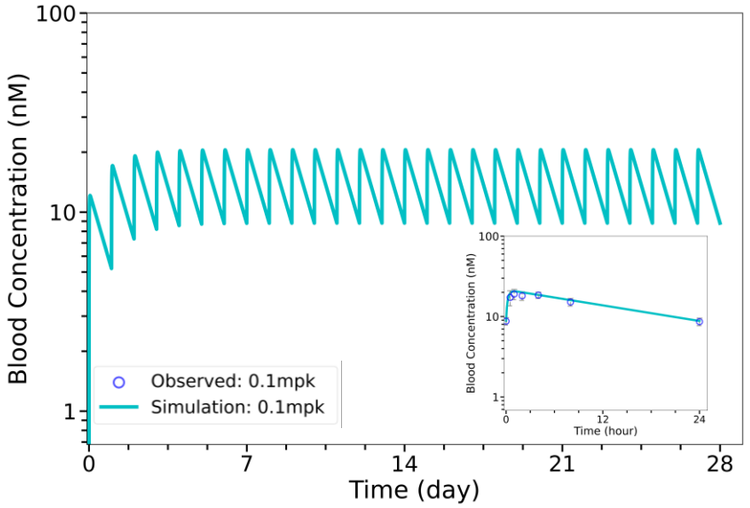
(1). RTV = relative tumor volume; relative to the tumor volume at the start of treatment

- AN9025 clearly showed more prolonged anti-tumor efficacy compared to RMC-6236 and RMC-9805 in KP-4 CDX model (RTK hyperactivation and Myc amplification).

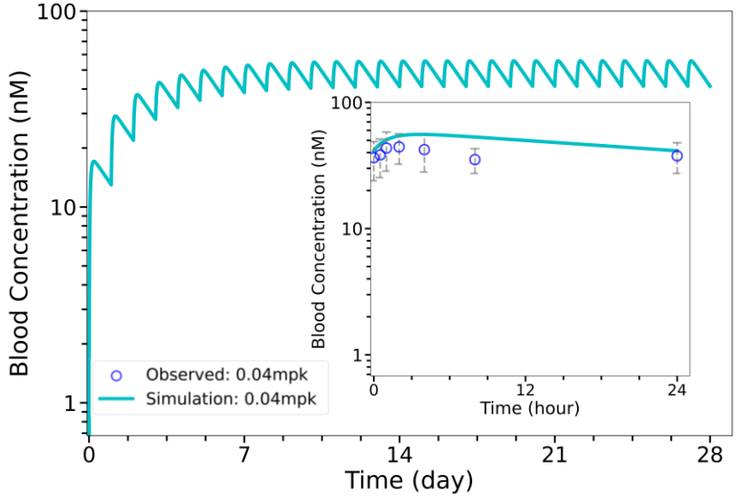
AN9025 Demonstrates Overall Favorable PK Properties

		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)	3350	324	203	2373
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)	3300	400	450	2100
	Bioavailability (F%)	44%	37%	57%	18%

AN9025 Rat MTD Blood Concentration Profile



AN9025 Dog MTD Blood Concentration Profile

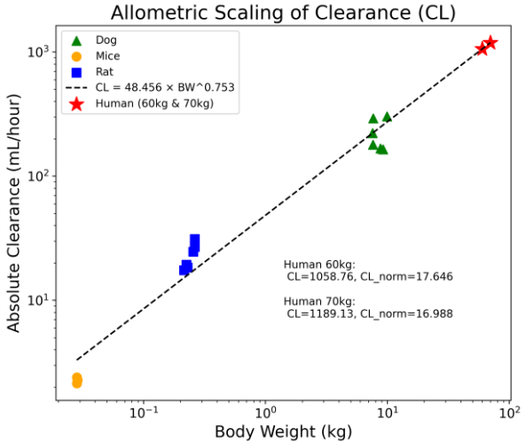


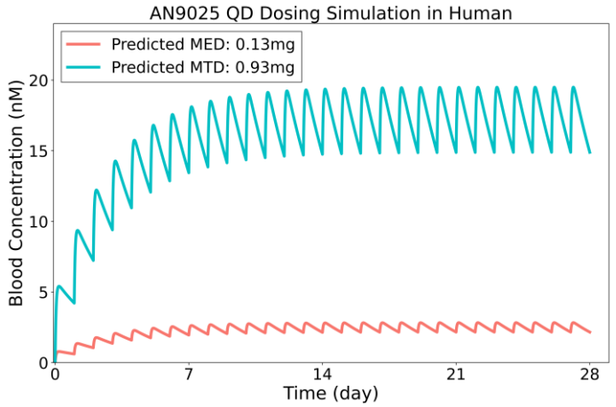
The PK simulation model demonstrated good concordance with observed TK data at Day 28 from 4-week GLP toxicology studies in rats and dogs

AN9025 Exhibited a Therapeutic Index Comparable to or Slightly Superior to that of RMC-6236

Drug	Species	TSC [1] nM	MED [1]			MTD (Observed in 4-week in-vivo efficacy study in mice and 2-week DRF toxicology studies in rats)			TI [b] (MTD/MED)
			Dose mpk	AUC nM*hour	C_{max} nM	Dose mpk	AUC nM*hour	C_{max} nM	
AN9025	Mouse	6.9	**0.030**	167	9.8	**0.20**	1390	72	**6.6**
	Rat	8.2	**0.058**	198	12	**0.20**	418	25	**3.4**
	Human [2] (60kg)	2.5	**0.0022 (0.13 mg)**	61	2.8	**0.016 (0.93 mg)**	417	19.5	**6.9**
RMC-6236	Mouse [3]	145	**8.6**	3480	468	**50**	16900	3060	**5.8**
	Rat [3]	350	**48**	8400	1040	**80**	11400	1110	**1.6**
	Human [4] (60 kg)	88	**1.8 (105 mg)[a]**	2160	195	**9.2 (552 mg)[a]**	11300	1020	**5.3**

(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) AN9025 human PK was projected via allometric scaling from mice, rat and dog data shown on slide#12 of the deck; (3) The MED and MTD of RMC-6236 in mouse and rat were determined internally, with direct comparison to AN9025 performed in the same studies; (4) The human PK parameters of RMC-6236 were derived from RMVD's translational study published in Cancer Discovery (2024; Volume 14, pp. 994–1017)

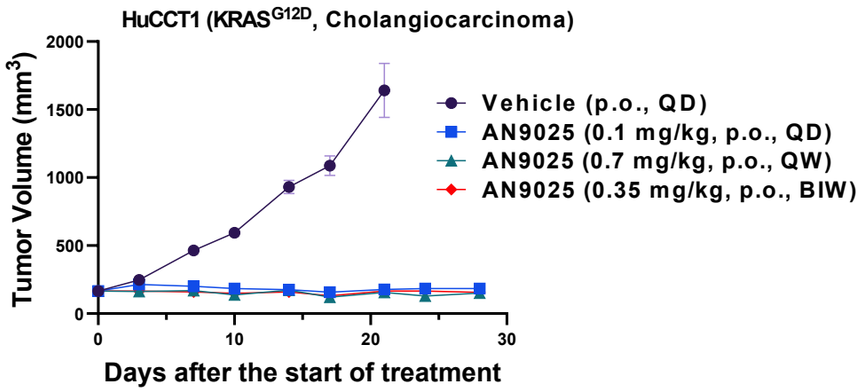


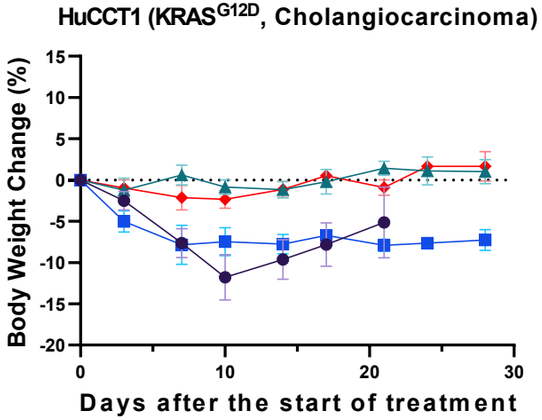


- [a]The simulation model demonstrated good predictability for both the human starting efficacious dose and maximum tolerated dose of RMC-6236
- [b]AN9025 exhibited a therapeutic index comparable to or slightly superior to, that of RMC-6236 in a QD regimen, despite its significantly greater potency
- Predicted AN9025 human PK based on allometric scaling: CL = 0.018 L/(h*kg); V = 1.3 L/kg; predicted human T1/2 = 40 h

Intermittent Dosing of AN9025 Achieves Tumor Regression Comparable or Slightly Superior to QD Dosing in mouse CDX Models

AN9025 demonstrated **comparable tumor regression** in mouse CDX models with intermittent dosing relative to QD dosing, while slightly improved tolerability was observed with the intermittent regimen

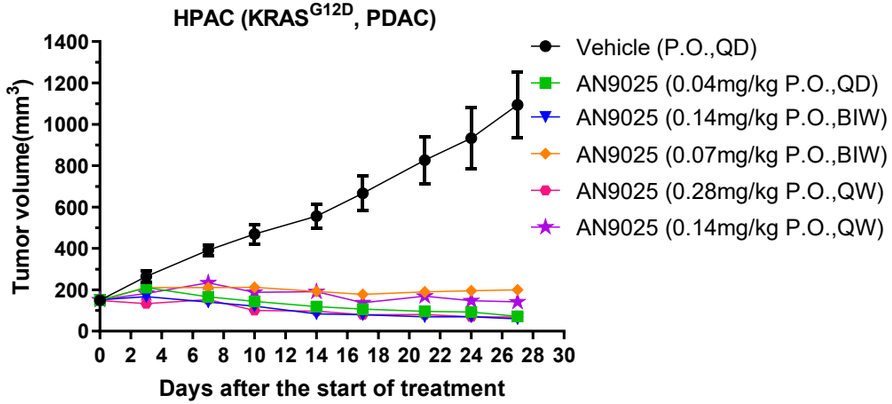


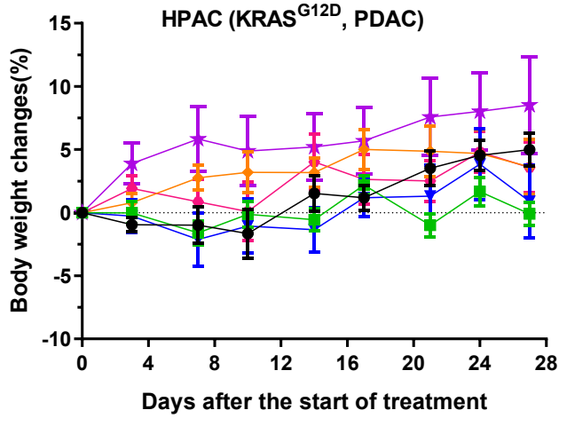


Dose (mg/kg)	RTV[1] on Day 28	BW change[2] (%) on Day 28
Vehicle	9.9 (Day 21)	-5.2% (Day 21)
0.1, QD	1.11	-7.3%
0.35, BIW	0.94	+1.6%
0.7, QW	0.90	+1.0%

(1) RTV = relative tumor volume; relative to the tumor volume at the start of treatment; (2) BW change % means the change compared to the one at Day 1

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





Dose (mg/kg)	RTV[1] (%) on Day 28	DUSP6 inhibition % at the end of treatment	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%	

Widening of AN9025 Therapeutic Window with Intermittent Dosing

Species	Dosing Regimen	TSC [1] nM	MED [1]			MTD (Observed in 4-week in-vivo efficacy model in mice and 2-week DRF toxicology study in rats)			TI (MTD/MED)
			Dose mpk	Weekly AUC nM*hour	C_{max} nM	Dose mpk	AUC nM*hour	C_{max} nM	
Mouse	QD	**6.2**	**0.028**	1050	8.8	**0.2**	1390	72	**7.3**
	BIW	**5.9**	**0.09**	983	22	.	.	.	.
	QW	**4.6**	**0.14**	792	34	.	.	.	.
Rat	QD	**7.4**	**0.053**	1300	11	**0.2**	378 Day14 AUC(0~24h)	24	**3.8**
	BIW	**7.1**	**0.18**	1220	23	**1.4**	1750 Day11 AUC(0~72h)	87	**7.8**
	QW	**5.7**	**0.28**	965	34	**2.8**	3320 Day8 AUC(0~72h)	290	**9.8**
Human (60 kg)	QD	**2.2**	**0.002 (0.12 mg)**	377	2.5	**0.014 (0.84 mg)**	373 Day14 AUC(0~24h)	17	**7.0**
	BIW	**2.0**	**0.0064 (0.384 mg)**	344	3.4	**0.068 (4.1 mg)**	1730 Day11 AUC(0~72h)	36	**11**
	QW	**1.5**	**0.01 (0.60 mg)**	269	3.9	**0.18 (11 mg)**	3310 Day8 AUC(0~72h)	69	**18**

(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) AN9025 human PK was projected via allometric scaling from mice, rat and dog data shown on slide#12 of the deck

- 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.

Summary of AN9025

- **AN9025** holds best-in-class potential within the Pan-RAS(ON) inhibitor category:

- Exhibited a binding affinity for cyclophilin A (CypA) that is 4-folder stronger than that of RMC-6236, driven by a slower dissociation rate from CypA, which may enable more sustained RAS inhibition
- Showed 3 to 8-fold higher binding affinity for tri-complex formation, also attributable to slower dissociation, potentially leading to prolonged suppression of RAS signaling
- Demonstrated 100-fold more potent inhibition of cellular proliferation and provided more sustained RAS inhibition compared to RMC-6236
- Induced deep tumor regression, superior and more sustained anti-tumor effects compared to RMC-6236 in mouse CDX models covering different RAS mutant types
- Demonstrated favorable overall DMPK properties, with 20~55% bioavailability across multiple species and a 2 to 5-fold longer half-life compared with RMC-6236
- Demonstrated a therapeutic index comparable to or slightly superior to RMC-6236 in QD regimen despite significantly improved potency

- The first patient has been dosed in early February 2026 in the ongoing global Phase 1 clinical trial in the US and China. Initiation of the intermittent dosing cohort is anticipated in mid-2026, with completion of full dose escalation expected by H1 2027.

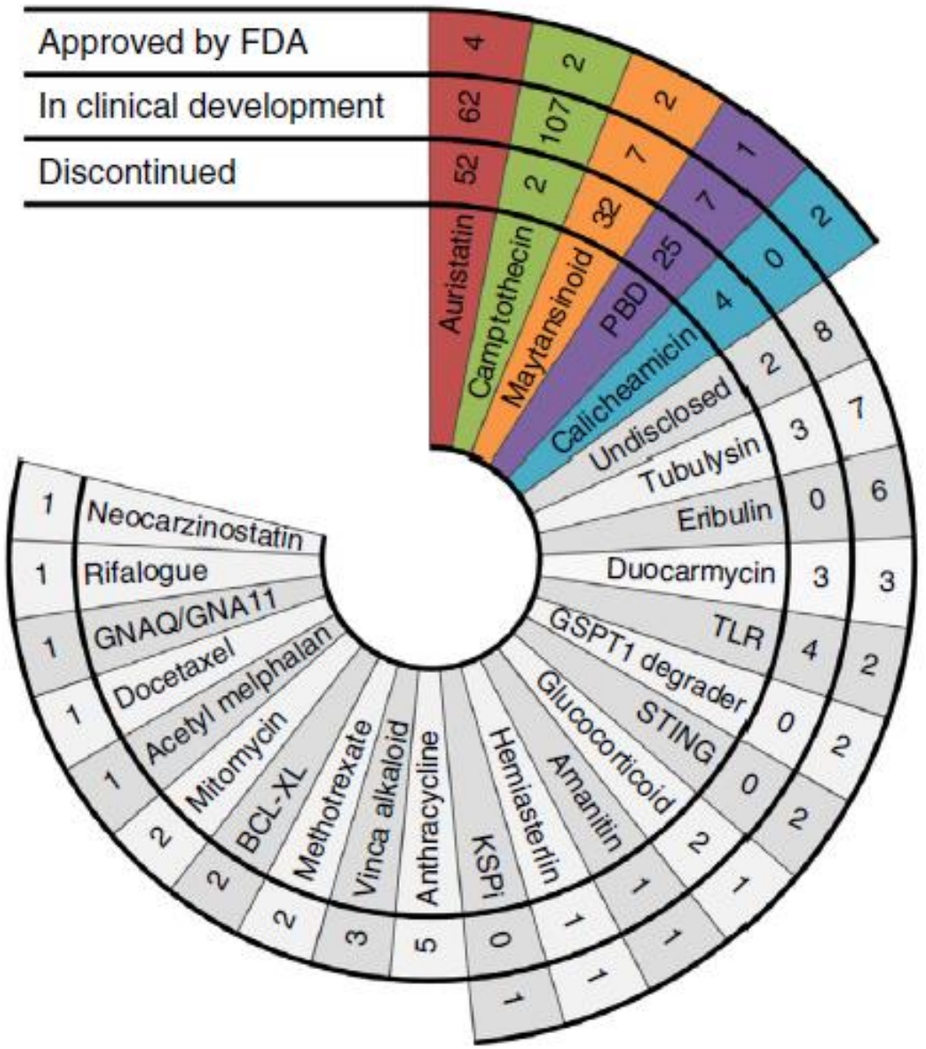
Adlai Nortye

AN4035: A Novel CEACAM5 Pan-RAS(ON)i ADC

RAS Inhibitor Conjugated Antibodies (RASiCA™) - Diversifying ADC Payloads to Overcome Clinical Resistance

- **RASiCA™ platform is designed to overcome TWO major challenges in oncology therapeutics**
  - ADC field: limited diversity of payload classes leading to cross-resistance
  - RAS inhibitor field: on-target, off-tumor toxicities associated with tricomplex pan-RAS(ON) inhibitors

- **Payload-related cross resistance limits treatment sequencing for ADCs[1,2]**
  - 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]
  - This demonstrates that payload-specific resistance is a key clinical challenge, creating an urgent need for novel payload classes to enable more effective treatment sequences



References: 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)

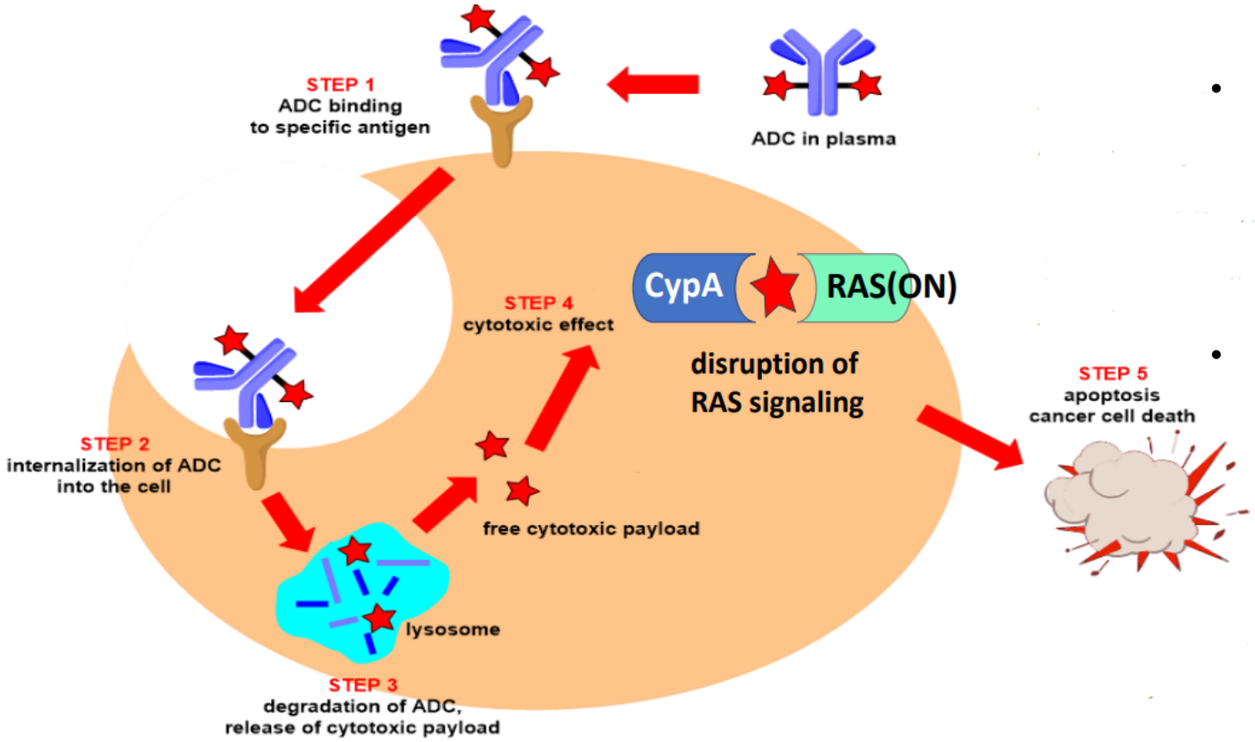
Pan-RAS(ON) Inhibitor as a Next-Generation ADC Payload

Background: tricomplex pan-RAS(ON) inhibitors (e.g. RMC-6236) are a promising class of therapeutics against RAS-driven cancers

Potential limitations: on-target, off-tumor toxicities may restrict dose and limit safety in combinations

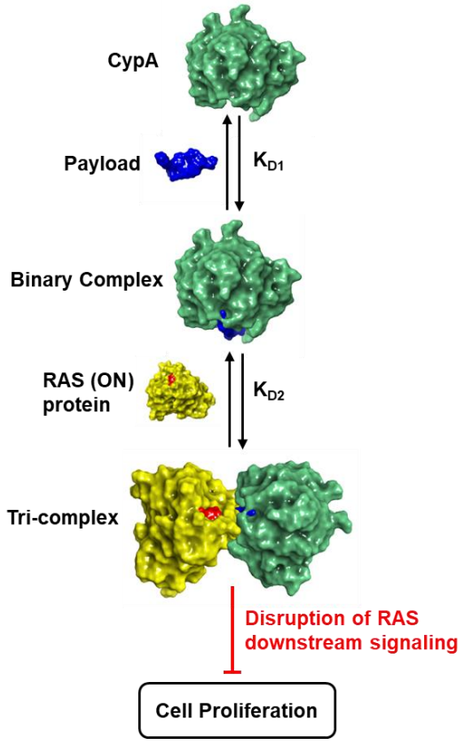
Our hypothesis: targeted delivery via an ADC could localize pan-RAS(ON) inhibitor activity to tumors while minimizing systemic RAS pathway inhibition

- ➢ Widen therapeutic window
- ➢ 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*
- *ADCs leveraging pan-RAS(ON) inhibitors could further enhance the therapeutic index*

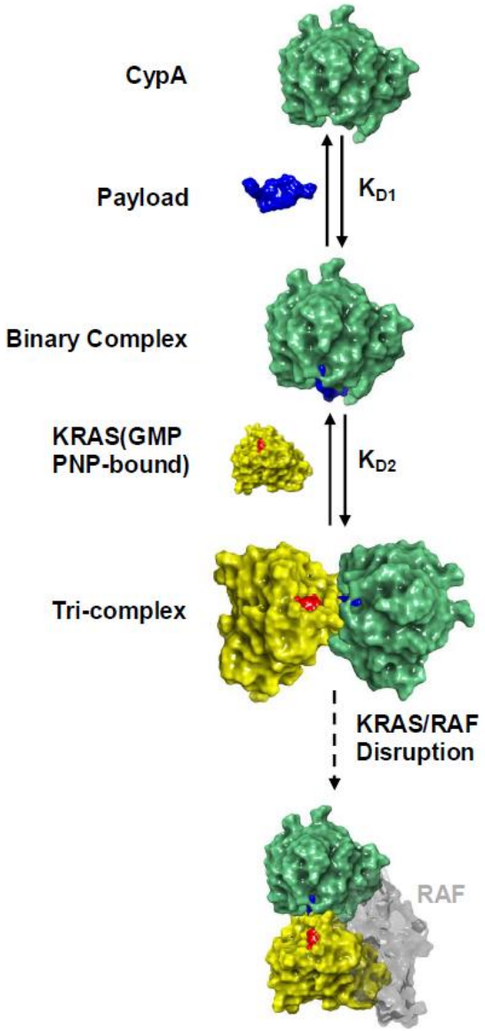
MOA of Pan-RAS(ON)i:



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

MOA of Payload:

- Pan-RAS(ON) molecular glue

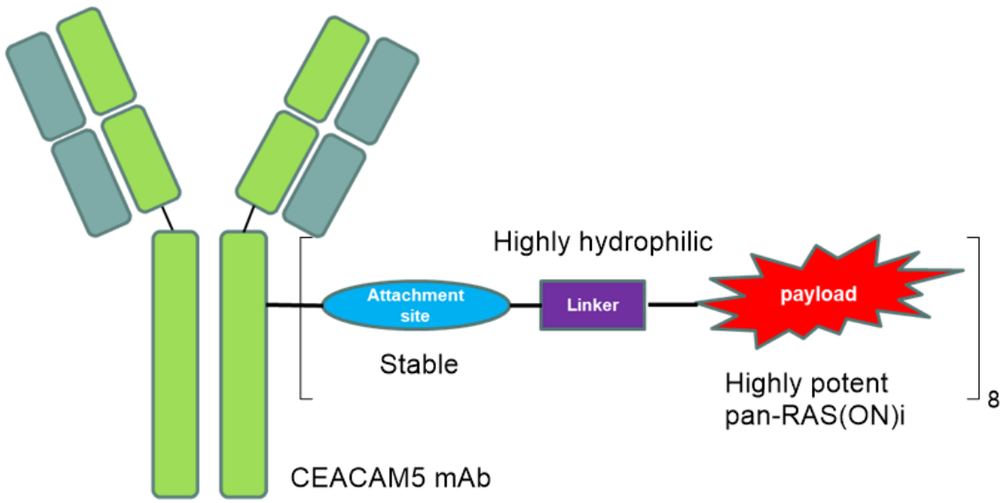


	Binary (CYPA) K_{D1} (nM)	Tri-complex (KRAS) K_{D2} (nM)			
		G12C	G12D	G12V	WT
Payload	14.9	4.7	30.5	14.0	18.6
RMC-6236	12.5	19.1	174	45.9	52.8

Cancer Cell	Indication	RAS mutation type	Payload 72h CTG	
			IC_{50} (nM)	I_{max} %
NCI-H2009	NSCLC	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
ASPC1	PDAC	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
SW620	CRC	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

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

Overview of AN4035



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

Stable Conjugator

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

Stable Linker-Payload

Compound	Plasma incubation time	Payload Released rate %			
		Human plasma	Mouse plasma	Rat plasma	Monkey plasma
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

No aggregation after incubation for 72h at 40℃

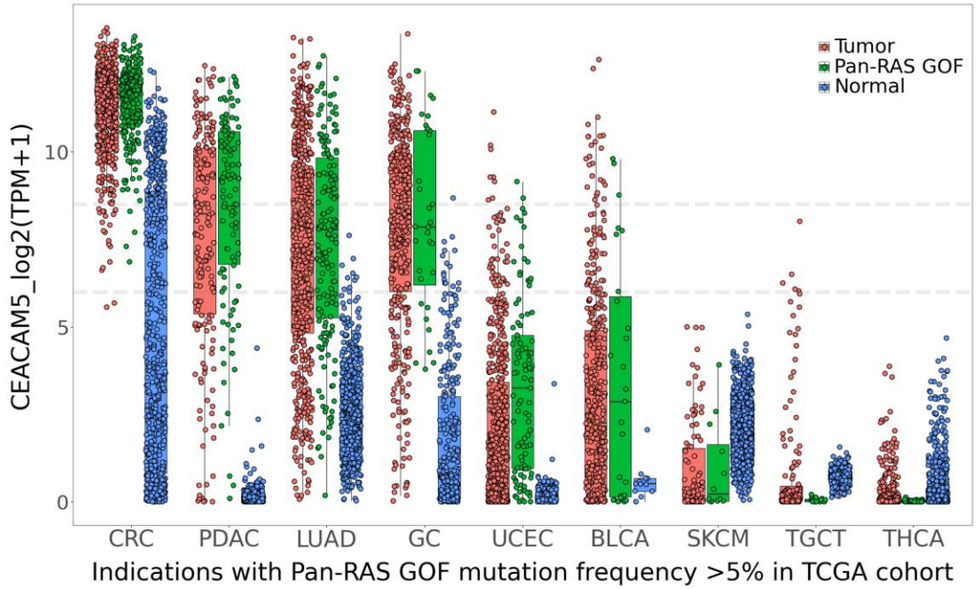
Compound	Incubation time	SEC-HPLC Aggregation %
AN4035	0 hr	0
	72 hr	0

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.

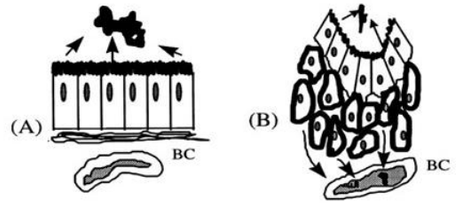
CEACAM5 as a Compelling Target for Pan-RAS(ON) Inhibitor-Based ADC

RAS-Mutant Cancer–Enriched, Skin-Sparing Expression



Expression data are derived from TCGA, GTEx datasets
HER2 mRNA-IHC threshold: https://doi.org/10.1136/jitc-2023-SITC2023.0143

Differential Expression in Normal Epithelium



NORMAL (weak, apical) **TUMOR (highly, whole surface)**

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

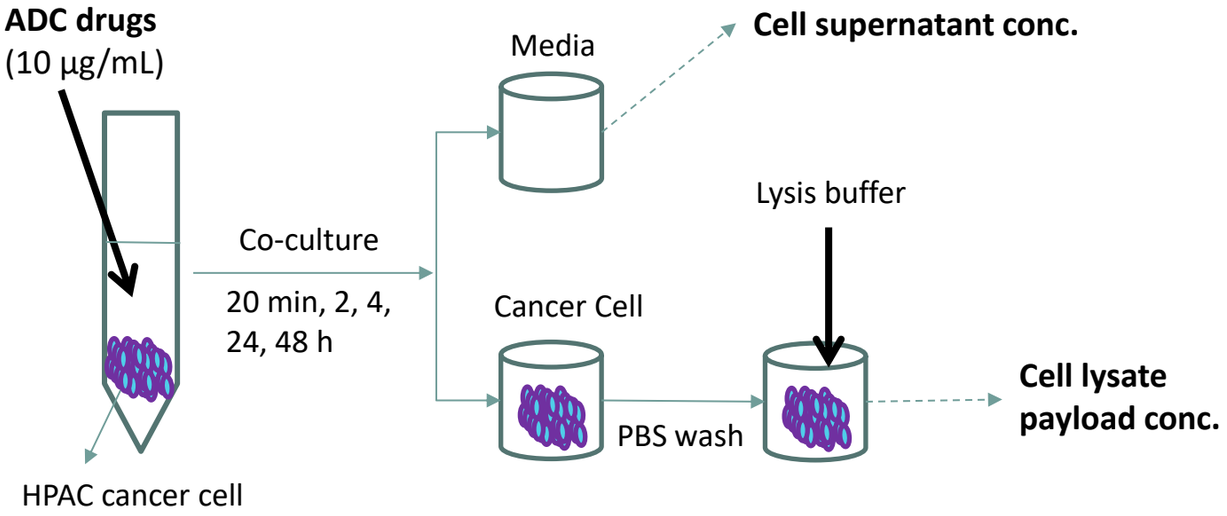
Clinically Validated ADC Target Without Gastrointestinal Dose-Limiting Toxicity

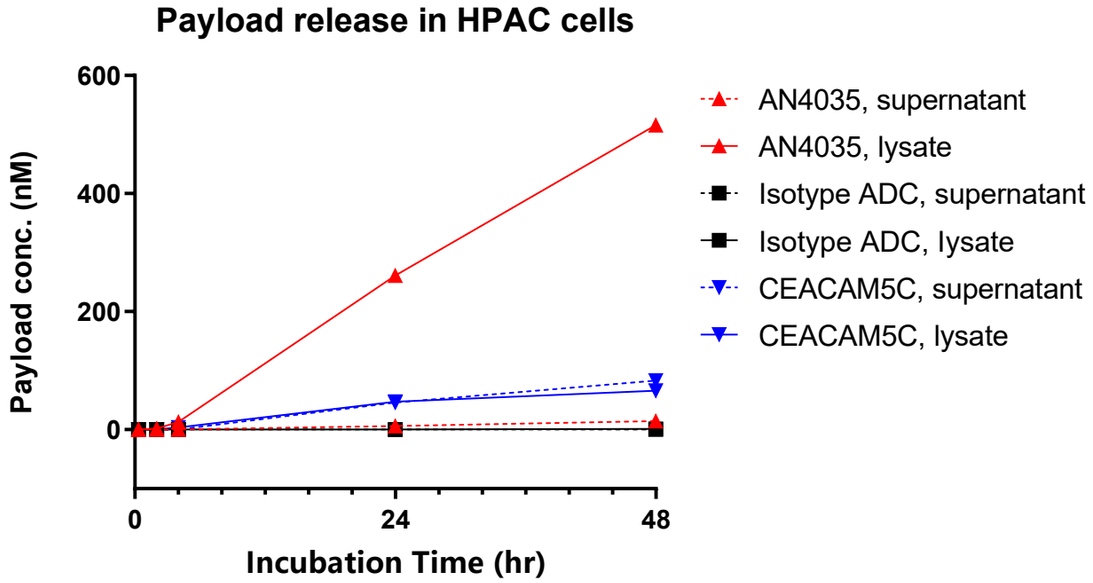
- Meaningful efficacy in *RASmut*-prevalent cancers including NSCLC & CRC
- Minimal / manageable on-target GI AEs at doses up to 10mg/kg QW, despite the presence of CEACAM5 on normal gut epithelium

ADC	Payload / DAR	RP2D	Clinical Efficacy	Diarrhea TRAE		GI AEs as DLT
				All grade	≥G3	
SAR408701	DM4 DAR: 3.8	100 mg/m^2 Q2W (2.6 mg/kg @70kg)	**ORR 21.7%** 2/3L CEA-high nsq-NSCLC, phIII [1]	9.8% [1]	**0.5%** [1]	None [2]
M9140	Exatecan DAR: 8	2.8 mg/kg Q3W	**ORR 26.8%** 3L+ mCRC, phI [3]	~25% [3]	**0%** [3]	None [4]
IMMU-130	SN38 DAR: 7.6	8 / 10 mg/kg QW	**DCR 62% / 55%** Late-line mCRC phI/II [5]	46% / 52% TEAE [5] * 56% TRAE	**5% / 5%** TEAE [5] * 7.9% TRAE	None [5]

(1). WCLC 2024 OA08.05; (2). Gazzah 2022, DOI:10.1016/j.annonc.2021.12.012; (3). ASCO GU 2026 #129P; (4). Kopetz 2024, DOI:10.1038/s41591-025-03843-z ; (5). Dotan 2017, DOI:10.1200/JCO.2017.73.9011

AN4035 Achieved 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**

BQL, below quantitation limit

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

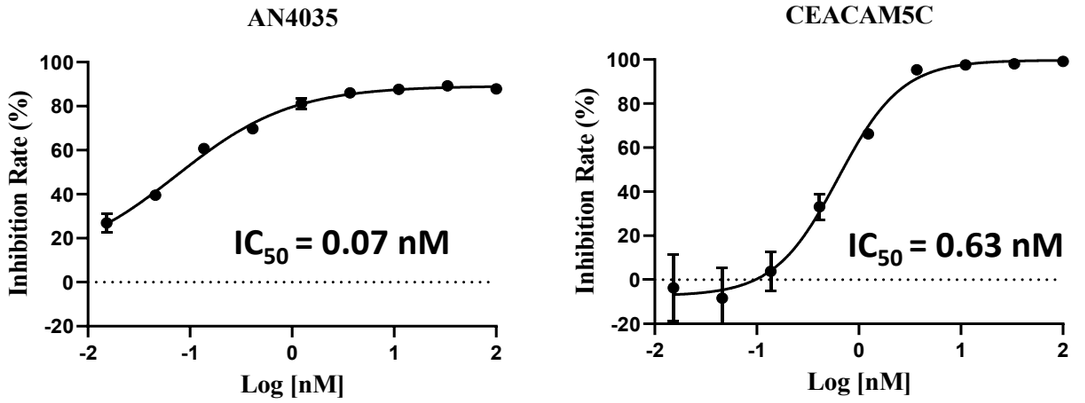
AN4035 Demonstrated Potent Cytotoxicity and a Robust Bystander Effect in CEACAM5-Positive, RAS-Addicted Cancer Cell Models

Cancer Cell Line	Cancer Type	CEACAM5 mRNA nTPM	RAS mutation type	6-day CTG		
				AN4035		Isotype ADC
				IC_{50} (nM)	Imax %	IC_{50} (nM)
CL-40	CRC	1821	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	
QGP-1	PDAC	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	
NCI-H2122	NSCLC	616	Kras G12C	0.30	94	
NCI-H727		332	Kras G12V	0.09	93	
MKN45	Gastric cancer	1087	Kras WT	0.13	95	>100

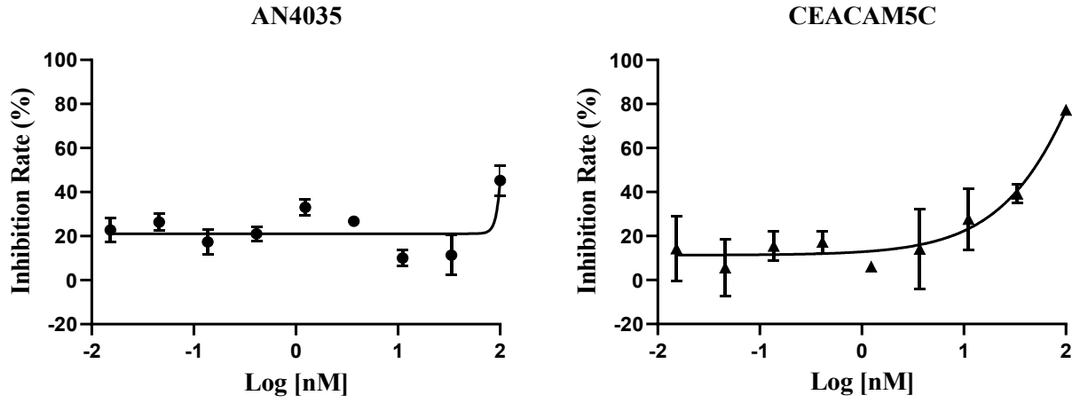
nTPM, normalized transcript per million

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

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



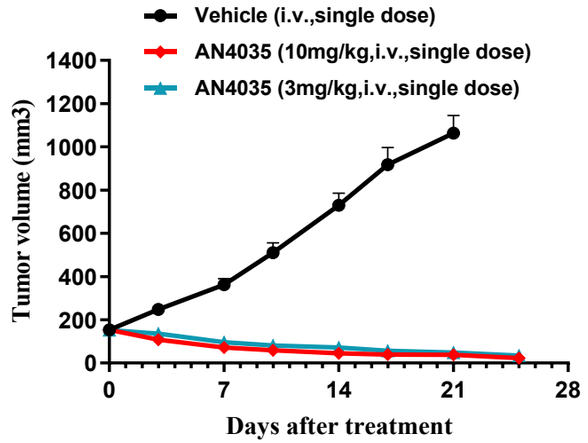
SW480-Luc (CEACAM5 neg) only

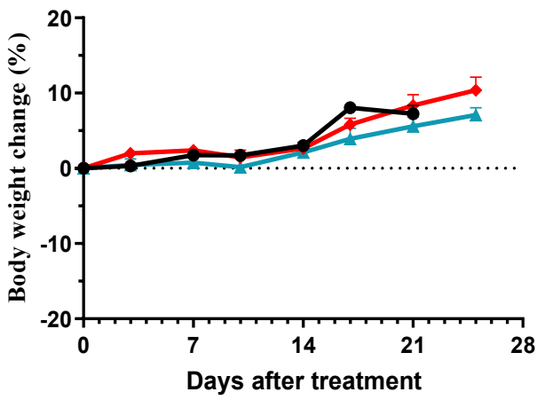


Source: AN4035 Late-Breaking Oral Presentation, 2025 AACR-NCI-EORTC International Conference: Molecular Targets and Cancer Therapeutics.

AN4035 Demonstrated Robust Anti-Tumor Activity in CDX Models

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



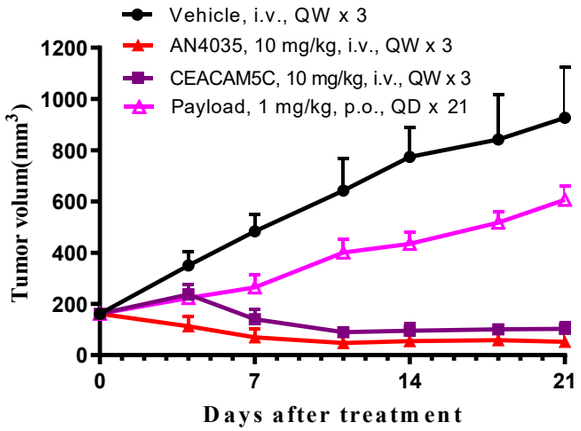


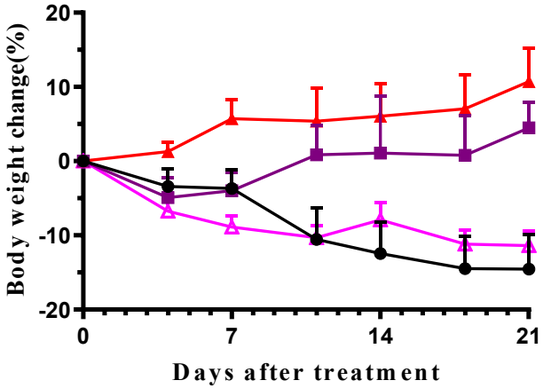
Compound	CTG[1] (IC_{50}, nM)	Dose (mg/kg)	ΔRTV[2] (%) on Day 25
AN4035	0.10	3.0, single dose	-78%
		10, single dose	-85%

(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

- Durable tumor regression with single-dose of 3 mg/kg

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





CDX, cell line-derived xenograft

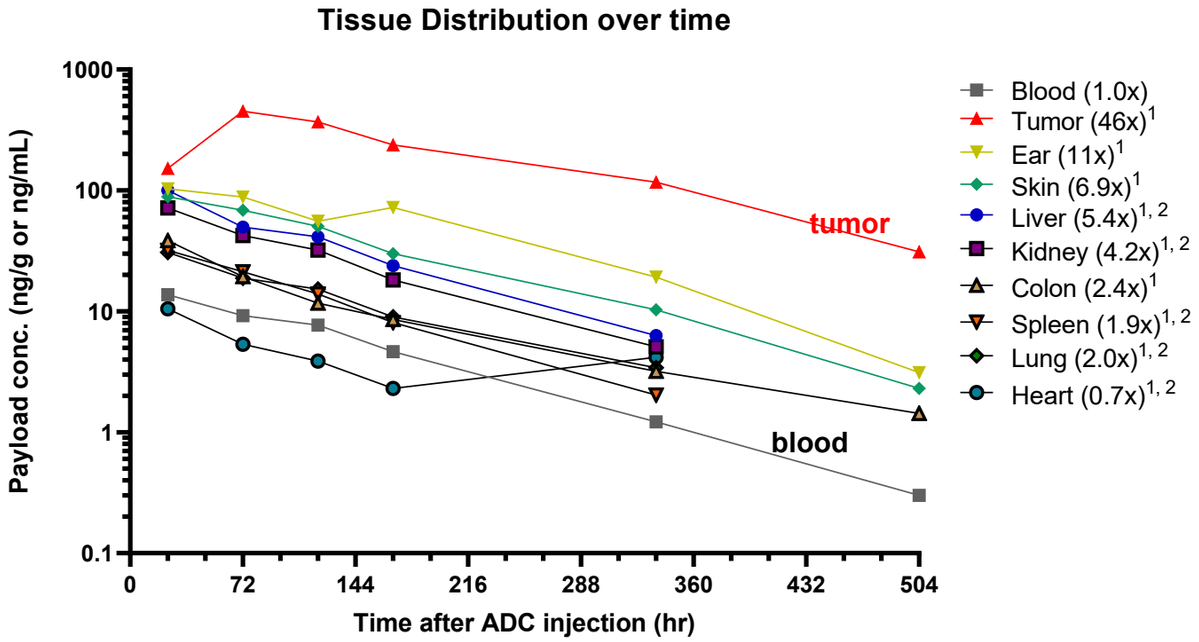
Compound	CTG[1] (IC_{50}, nM)	Dose (mg/kg)	ΔRTV[2] (%) on Day 21
AN4035	0.38	10, QW	-89%
CEACAM5C	1.2	10, QW	-36%
payload	0.08	1.0, QD	276%

- Superior or comparable efficacy to CEACAM5 Topo I-based ADC (CEACAM5C) or naked payload at tolerable doses

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

AN4035 Shows Enhanced Target-Mediated Tumor Retention vs. Payload Alone, with Significantly Improved Tumor Selectivity over Normal Tissues

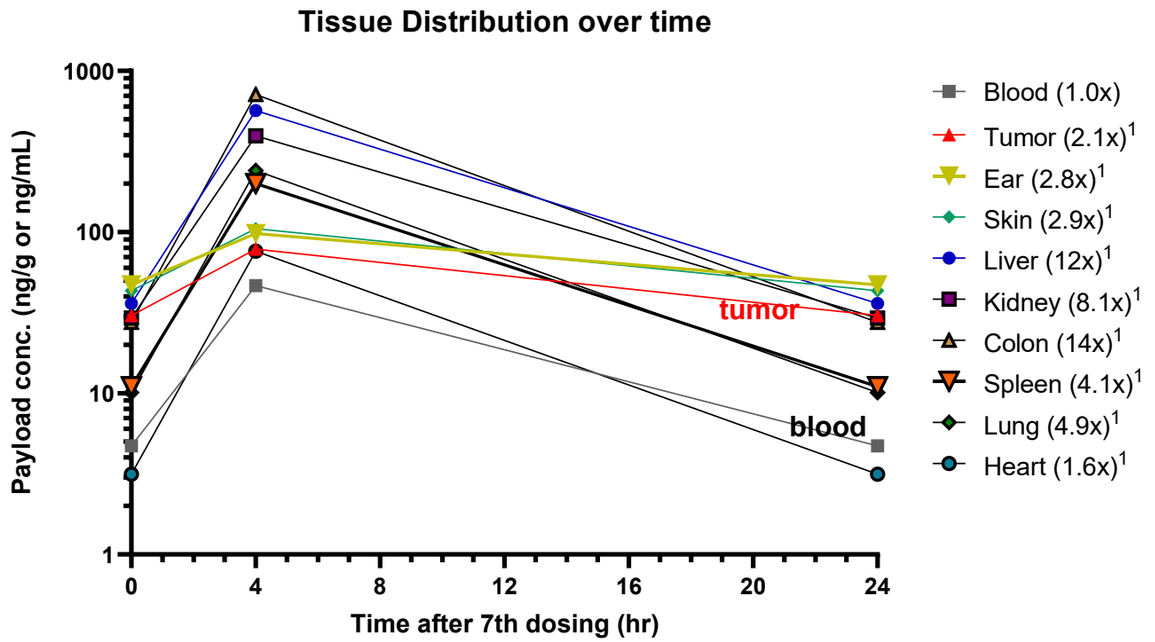
Free Payload concentrations in tumor, normal tissues and blood were measured at multiple time points after a single dose of AN4035 (10 mg/kg, IV) in the HPAC (KRAS ^{G12D}) CDX model



(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.

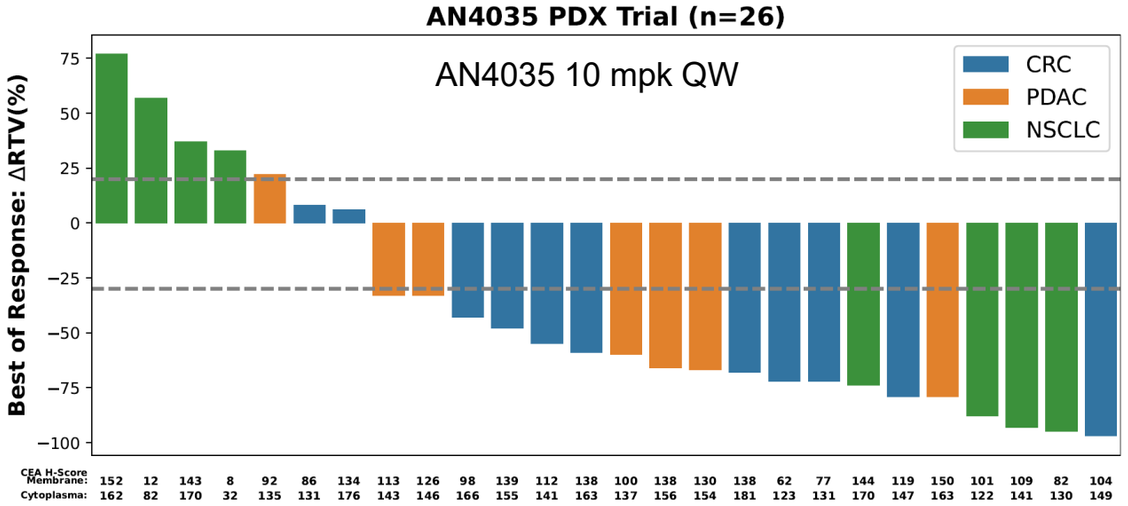
Free Payload concentrations in tumor, normal tissues and blood were measured at multiple time points after 7-day continuous dose of Payload alone (1 mg/kg, PO) in the HPAC (KRAS ^{G12D}) CDX model



(1) Ratio of tumor/tissue concentration (or AUC exposure) over blood concentration

(2) Assume steady state has achieved after 7-day continuous oral dosing, $C_{T0h} = C_{T24h}$

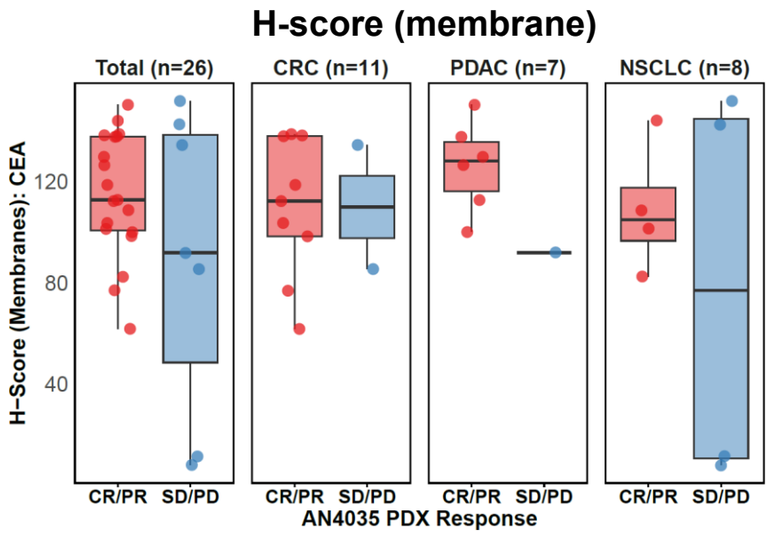
AN4035 Exhibited Compelling Activity in Single-Mouse PDX Trials

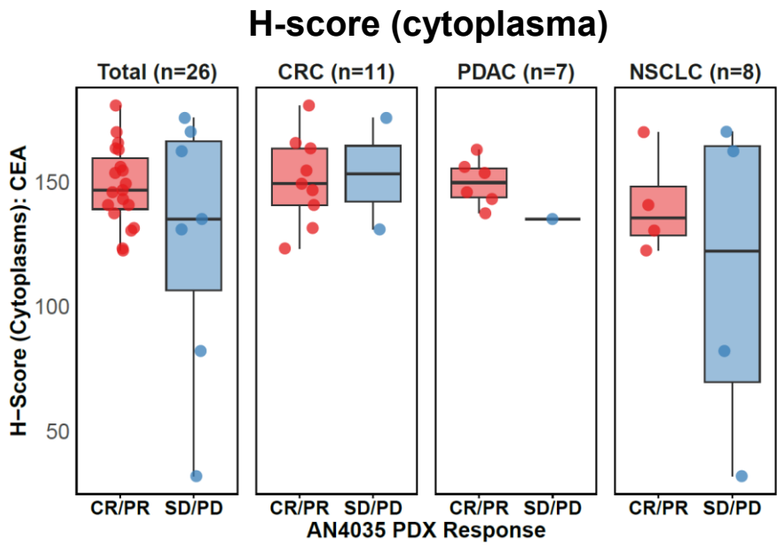


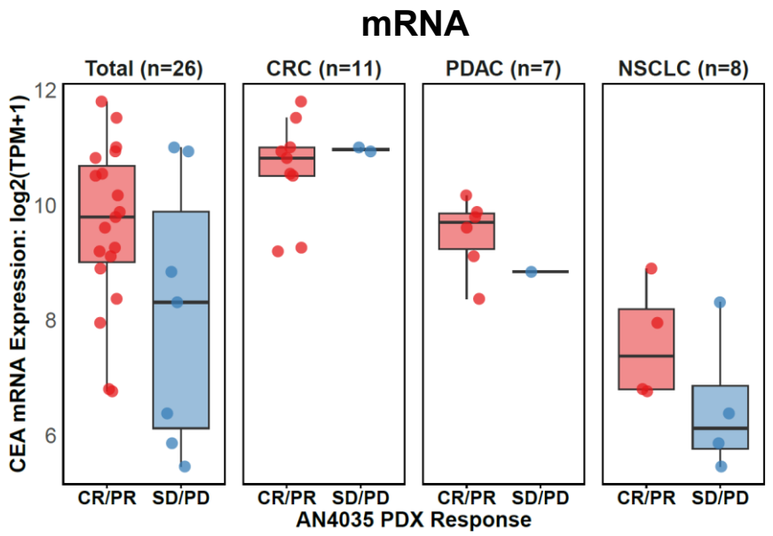
Indication	CR	PR	SD	PD	ORR	DCR
CRC	1	8	2	0	**82%**	**100%**
PDAC	0	6	0	1	**86%**	**86%**
NSCLC	3	1	0	4	**50%**	**50%**
Total	4	15	2	5	**73%**	**81%**

PDX, patient-derived xenograft

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); PDX, patient-derived xenograft





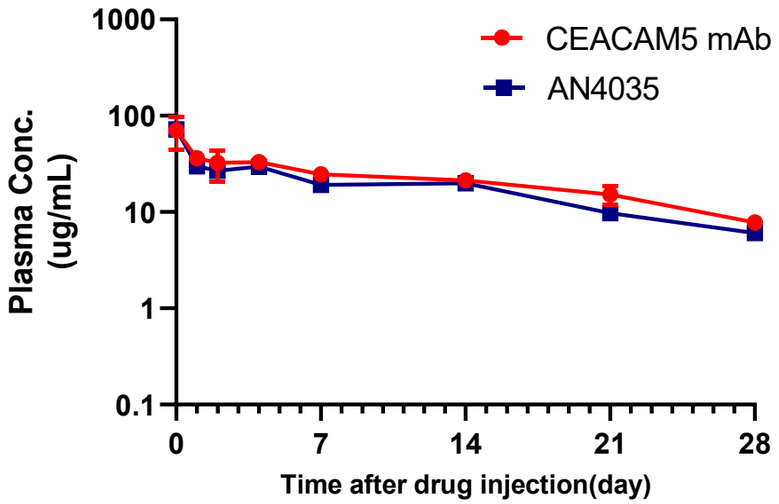


Source: AN4035 Late-Breaking Oral Presentation, 2025 AACR-NCI-EORTC International Conference: Molecular Targets and Cancer Therapeutics.

AN4035 Demonstrated Favorable PK and Tolerability in Monkeys

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

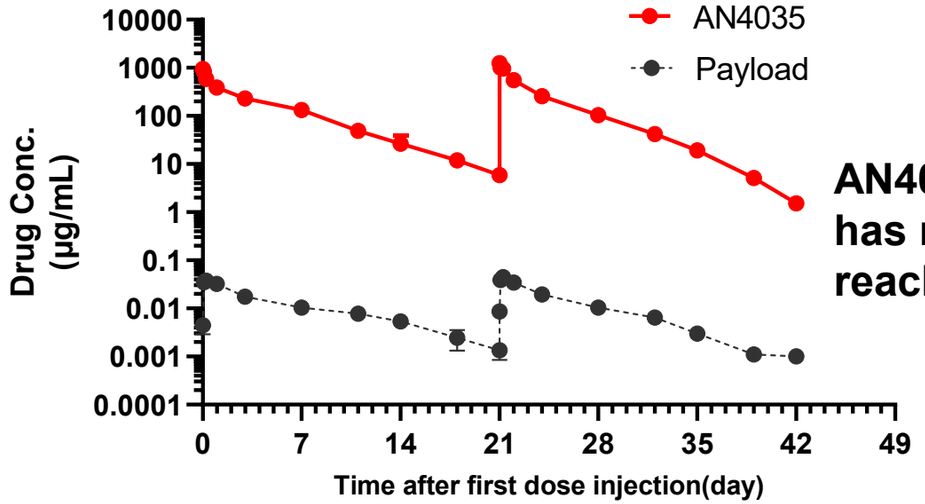
CB17-SCID mouse



Compound	Dose	C_0 (μg/mL)	AUC (h*μg/mL)	$T_{1/2}$ (day)
AN4035	3 mg/kg, IV	72	13832	8.2
CEACAM5 mAb	3 mg/kg, IV	71	17218	9.7

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

Repeated doses in Monkey (AN4035 30 mg/kg x 2)



AN4035 HNSTD has not been reached at 30 mpk

Compound	Dose	C_0 (μg/mL)	AUC (h*μg/mL)	$T_{1/2}$ (day)
AN4035	30 mg/kg, IV	942	60377	3.3
Payload		0.038	5.40	4.3

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.

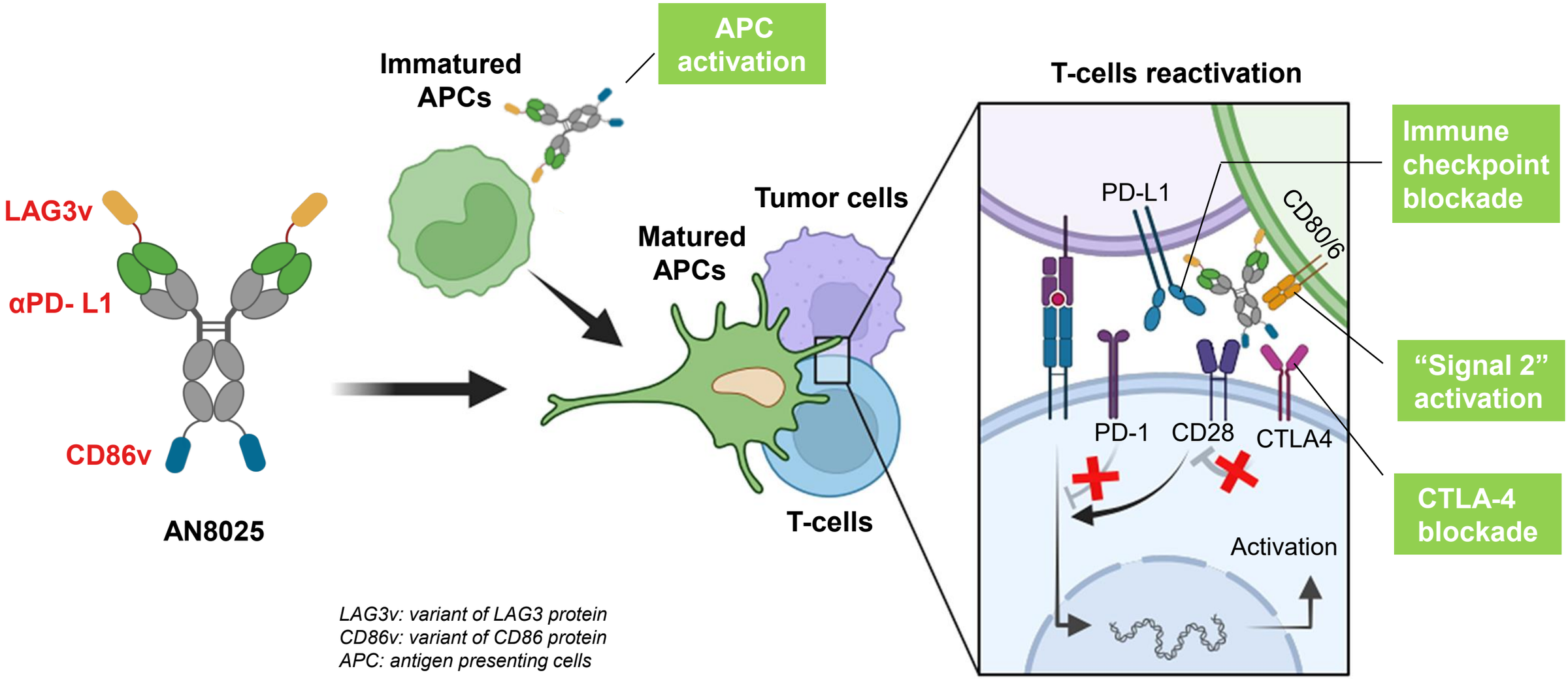
Summary of AN4035

- **AN4035** is a potential first-in-class CEACAM5-targeting ADC armed with a highly potent pan-RAS(ON) inhibitor payload:
  - Favorable thermal and plasma stability with desirable pharmacokinetic properties
  - Strong intracellular payload retention, driving nanomolar to picomolar cytotoxicity in CEACAM5-positive/RAS-addicted cancer cell lines, along with a potent bystander-killing effect
  - Robust anti-tumor activity with deep regression in CEACAM5-positive/RAS-addicted CDX and PDX models, as monotherapy or in combination
  - Enhanced target-mediated tumor retention versus payload alone, with significantly improved tumor selectivity over normal tissues
  - Favorable therapeutic index in cynomolgus monkeys
- IND submission is on track to be completed by mid-2026

Adlai Nortye

AN8025: A Potential First-in-Class, Multi-Functional T Cell/APC Modulator

Mechanism of Action: Schematic Overview of AN8025



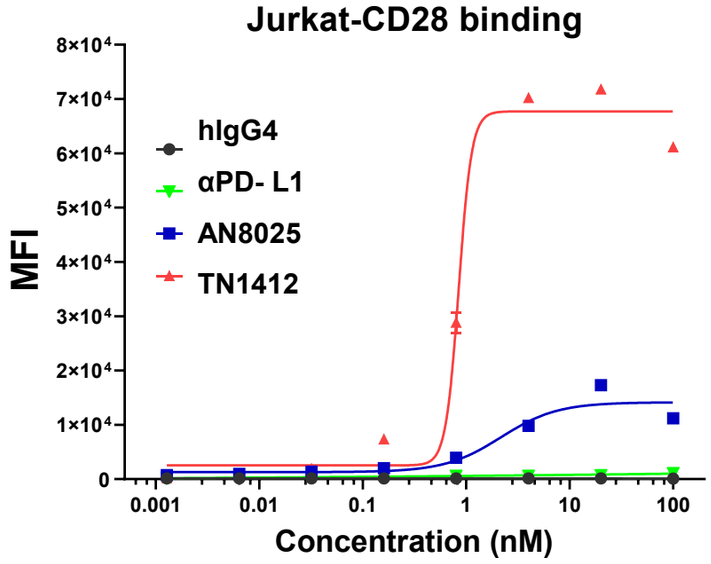
LAG3v: variant of LAG3 protein
CD86v: variant of CD86 protein
APC: antigen presenting cells

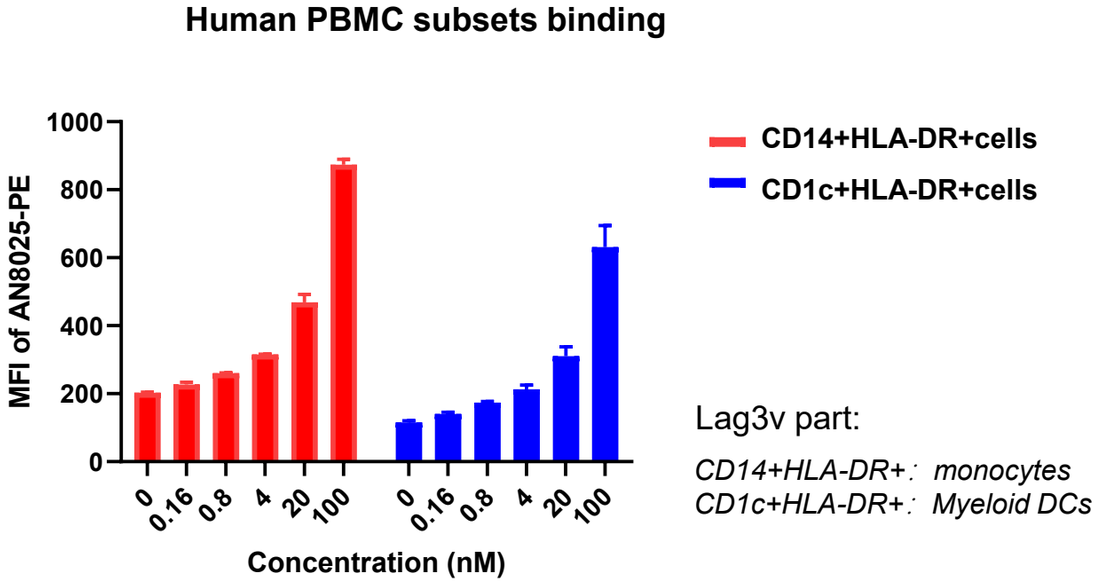
AN8025 Binding and Target Interaction

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	5.63E-09	AN8025	1.02E-08	AN8025	1.86E-09
αPD-L1	NA	αPD-L1	NA	αPD-L1	1.42E-09
Tremelimumab	7.32E-10	TGN1412	2.88E-12		
Ipilimumab	7.37E-09				

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





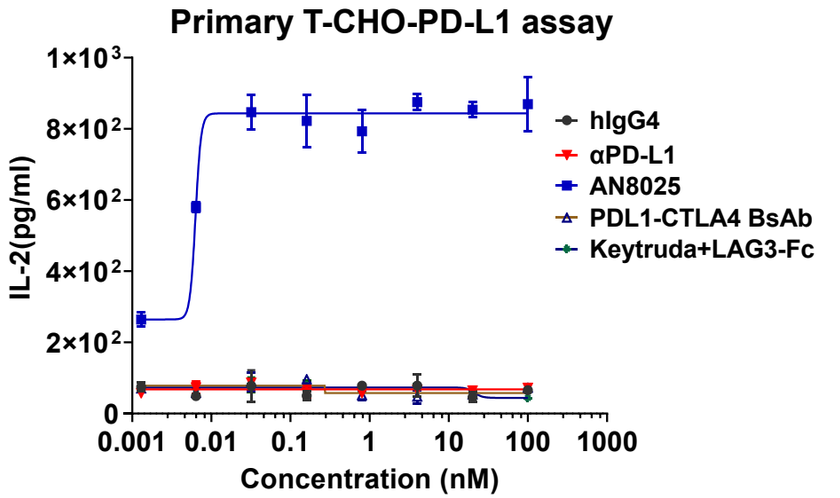
Lag3v part:

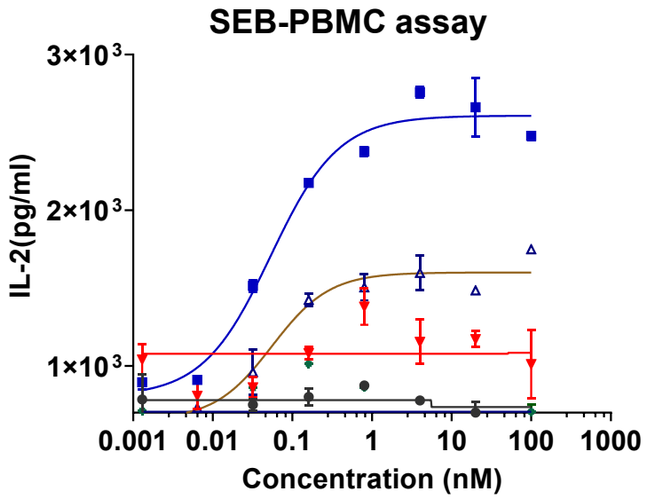
CD14+HLA-DR+: monocytes
CD1c+HLA-DR+: Myeloid DCs

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

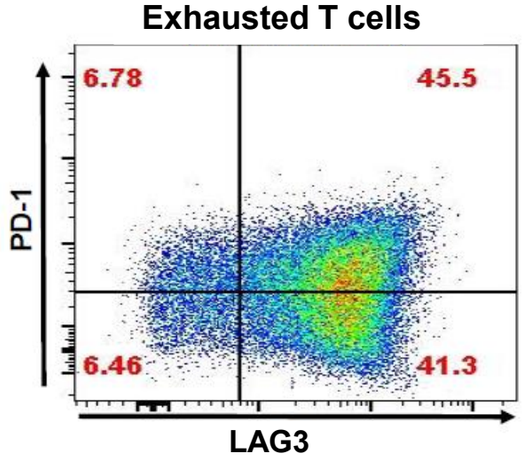
AN8025 Induces Superior T-cell Responses

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

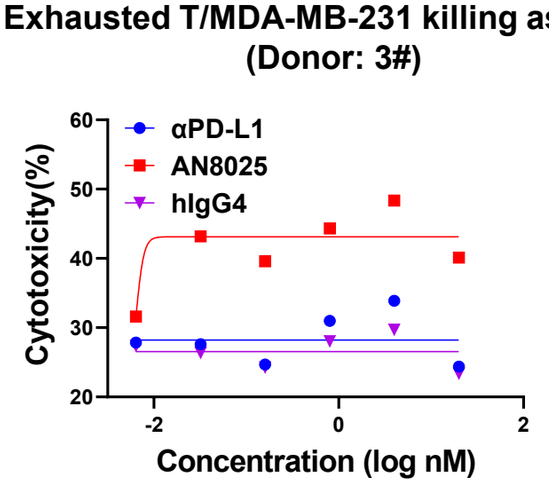


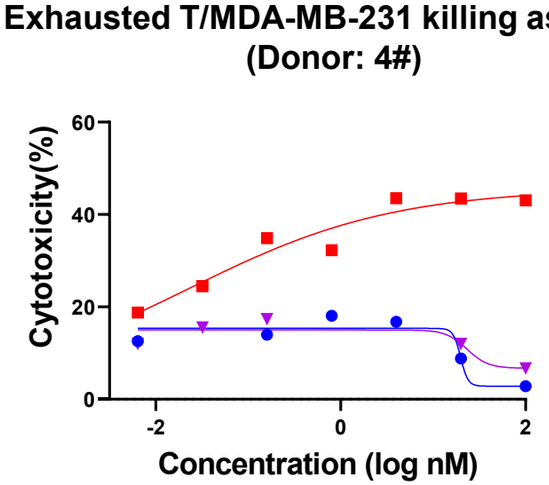


AN8025 reinvigorates exhausted T cells for efficient tumor cell killing



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

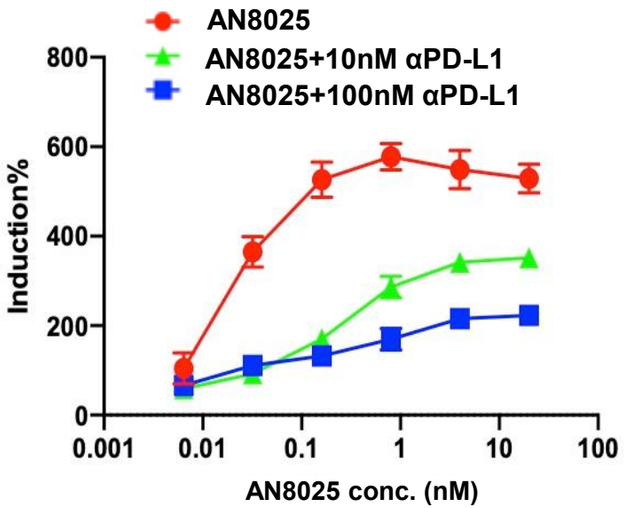


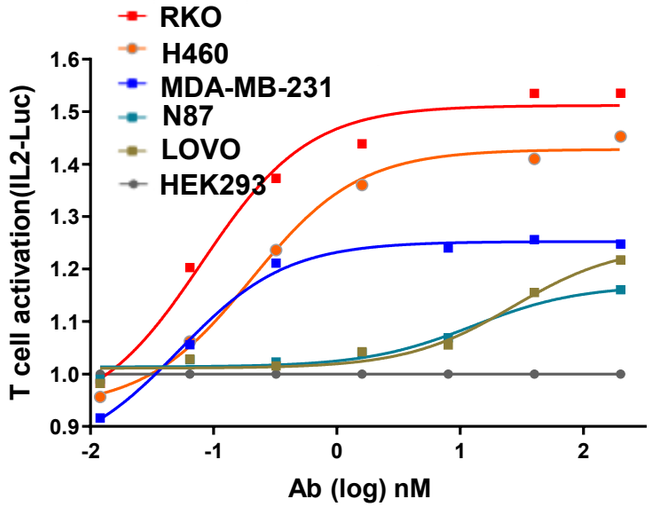


Source: AN8025 Poster, Abstract #3420, 2025 AACR Annual Meeting.

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

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

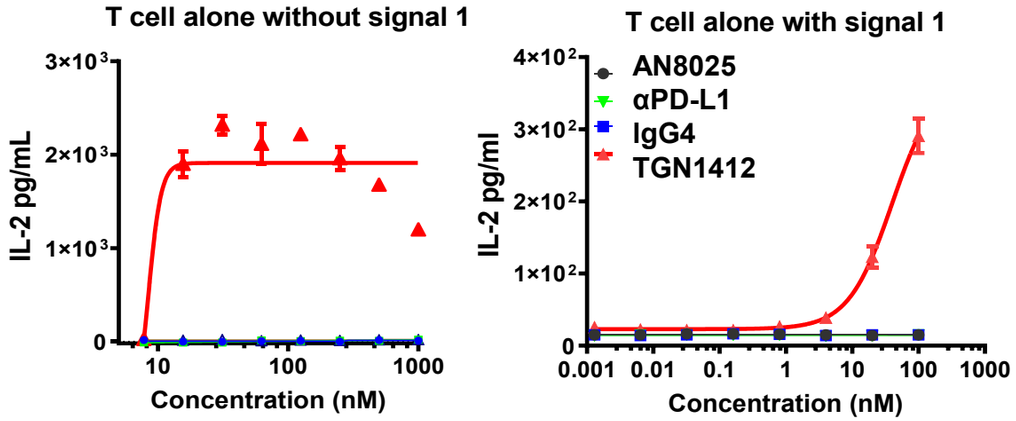




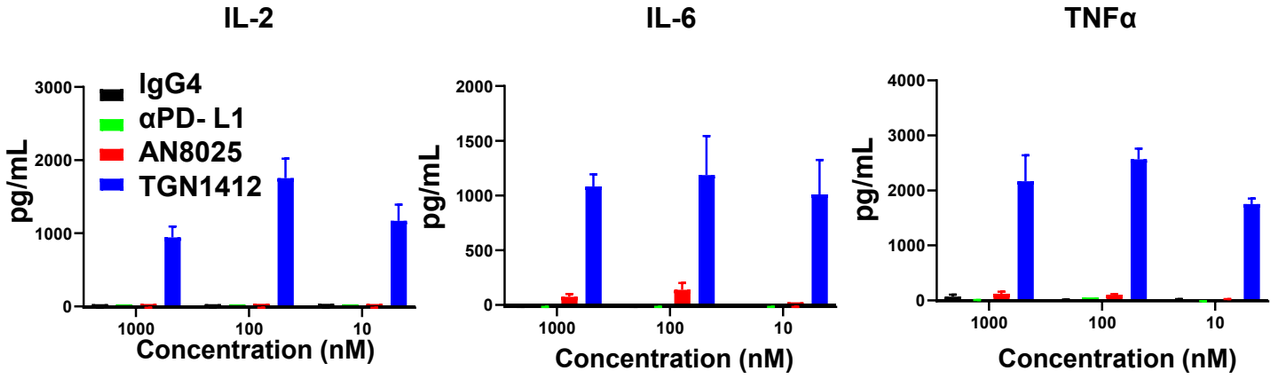
Cell line	hPD-L1 MFI	EC50 (nM)
RKO (PD-L1 high)	40333	0.08
H460 (PD-L1 Med)	24909	0.202
MDA-MB-231 (PD-L1 Med)	10849	0.052
N87 (PD-L1 low)	211	12.63
LOVO (PD-L1 low)	445	26.85
HEK293 (PD-L1 neg)	40	NA

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

T-cell alone activation with or without signal 1



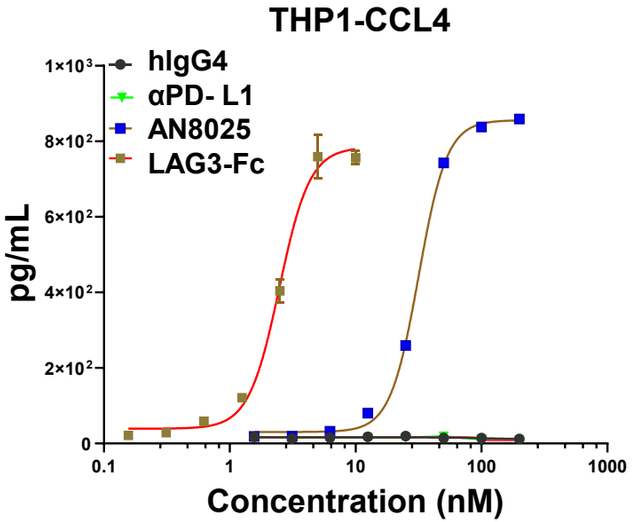
Cytokine release from hPBMC induced by immobilized Abs

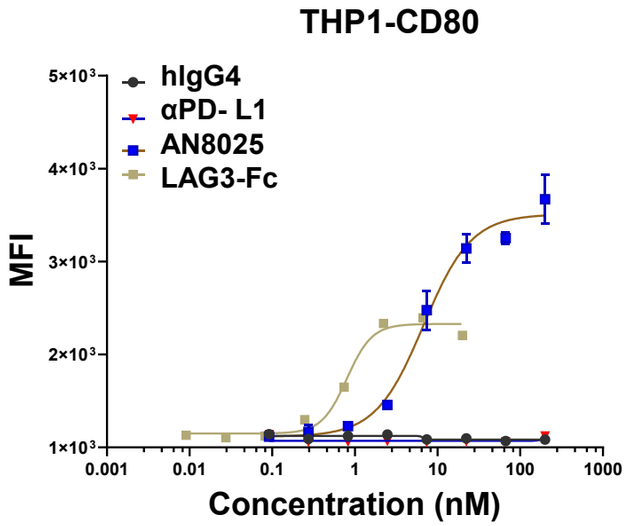


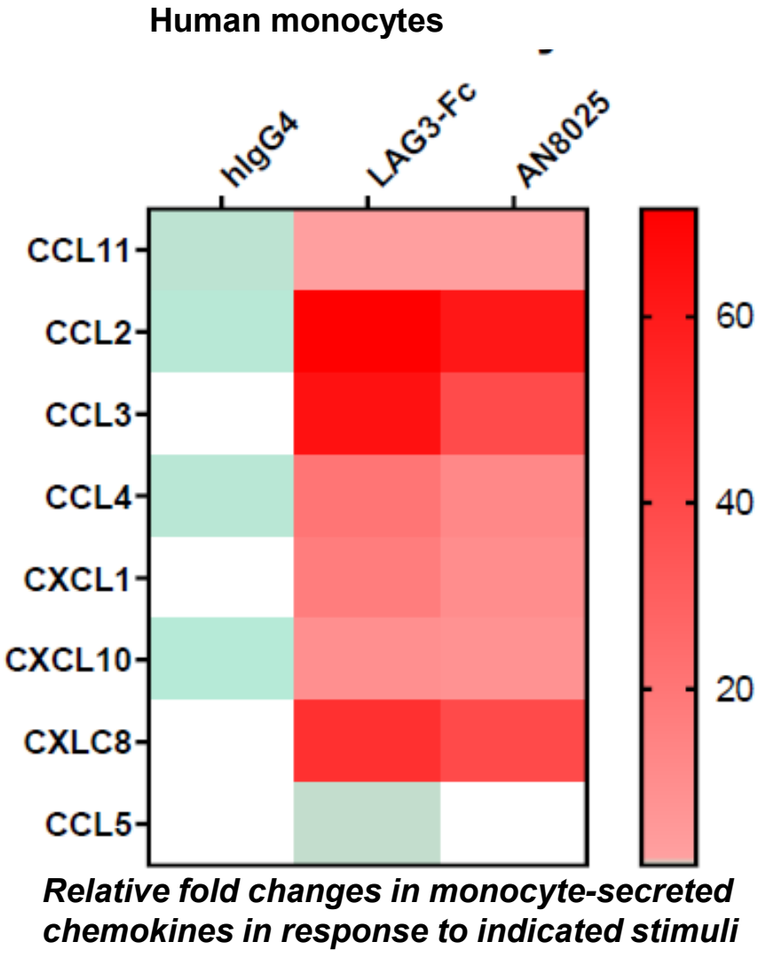
Source: AN8025 Poster, Abstract #3420, 2025 AACR Annual Meeting.

AN8025 Enhances Both the Quantity and Functionality of Antigen Presenting Cells

APC Quality:

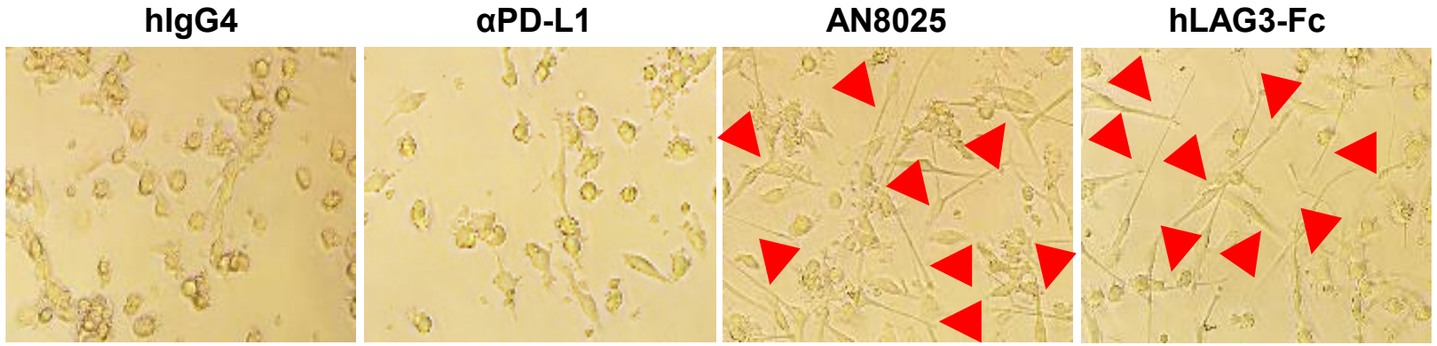






Relative fold changes in monocyte-secreted chemokines in response to indicated stimuli

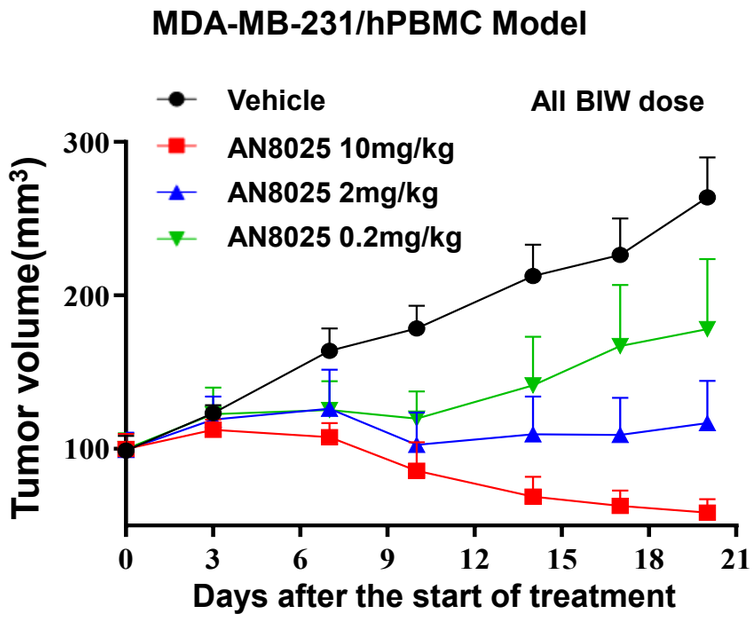
APC Quantity:

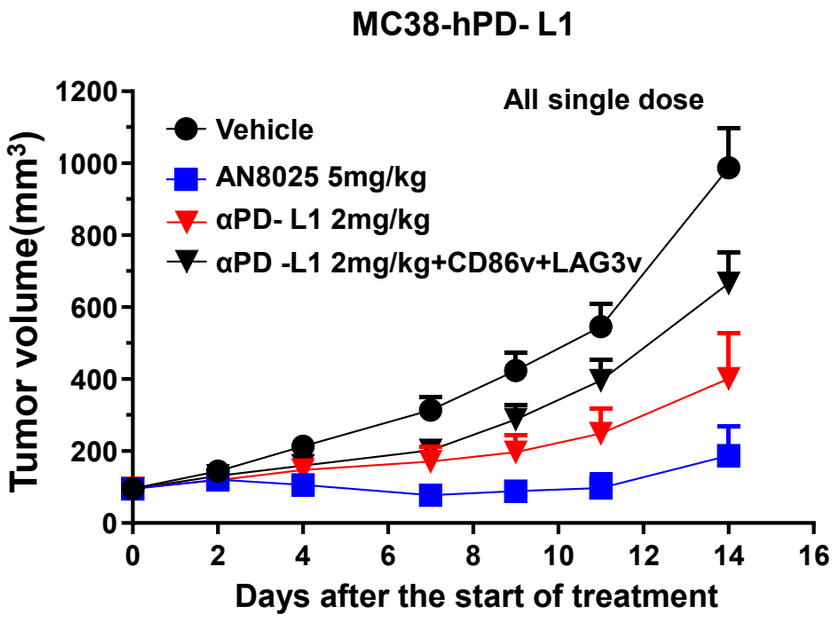


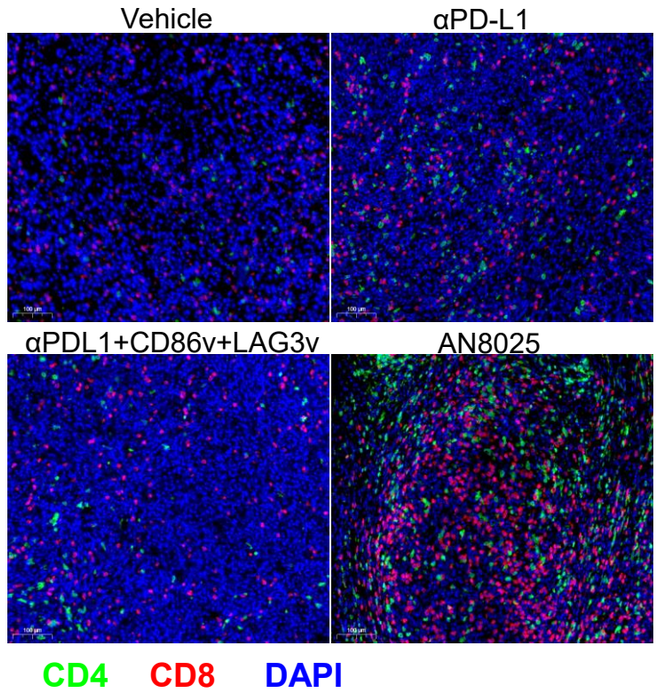
Monocyte derived immature dendritic cell morphology

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

AN8025 Exhibits Potent Anti-Tumor Activity In Vivo





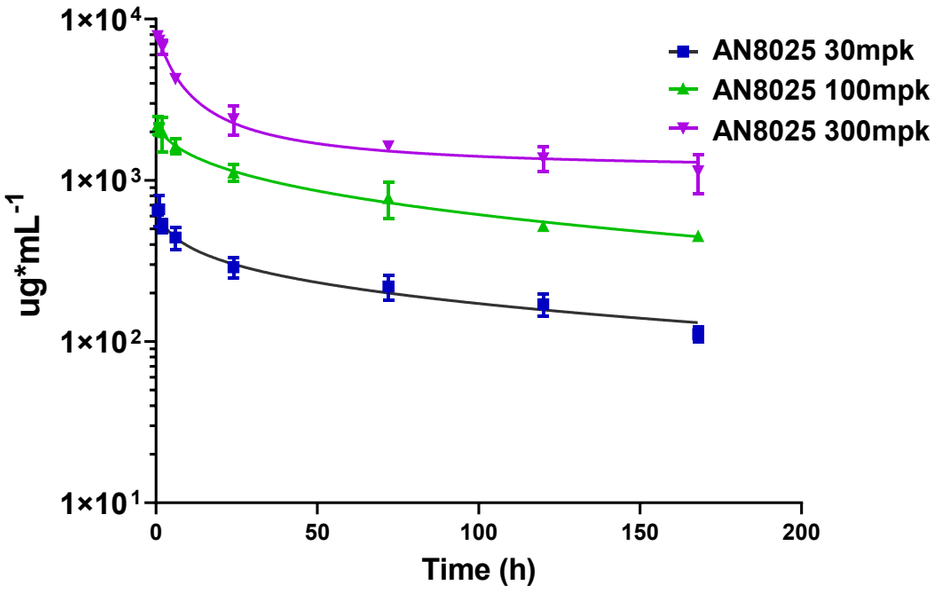


Group	TMV (mm^3)	TGI (%)	P value
Vehicle	264 ± 26		
AN8025 10 mg/kg	58 ± 9	125	<0.001
AN8025 2 mg/kg	117± 27	89	0.008
AN8025 0.2 mg/kg	178 ± 46	52	0.402

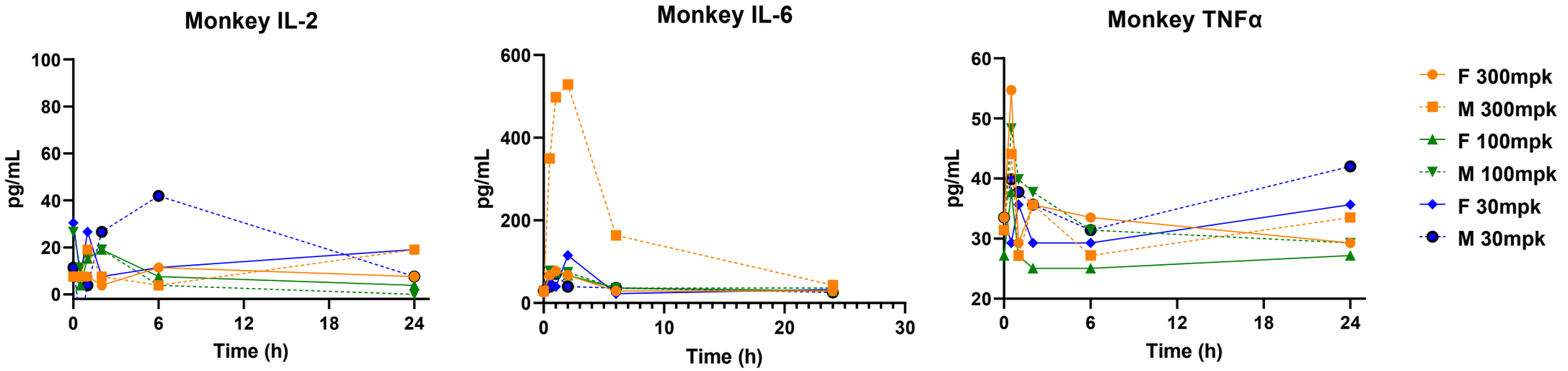
Group	TMV (mm^3)	TGI (%)	CR (%)	P value
Vehicle	987±110			
AN8025	187±82	90	40	<0.001
αPD-L1	401±126	66	10	0.001
αPD-L1 +CD86v+LAG3v	666±87	36	0	0.085

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

AN8025 Demonstrates Favorable PK and Safety Profiles in Non-Human Primates



Dose	C0 (μg/ml)	AUC (h*μg/ml)	T1/2 (h)
30 mpk	706	37900	107.35
100 mpk	2200	135000	177.15
300 mpk	7905	323500	248.7



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

Summary of AN8025

- **AN8025** is a potential first-in-class, next-generation cancer immunotherapy with a multi-mechanistic mode of action:
  - Drove robust T cell activation in a PD-L1–dependent manner.
  - Promoted both exhausted and non-exhausted T cell populations to support effective tumor clearance.
  - Enhanced both the quantity and functional quality of antigen-presenting cells (APCs).
  - Demonstrated potent anti-tumor efficacy in vivo.
  - Showed a favorable safety and tolerability profile in non-human primates (NHPs).
- The first patient has been dosed in December 2025 in the ongoing global Phase 1 clinical trial in Australia and China, with dose escalation anticipated to be completed by 2026 YE

Other Assets - Clinical Update of AN4005

AN4005: Orally Available, Small-Molecule PD-L1 Inhibitor

AN4005 as a Backbone for Our Future Oral Combination Therapies

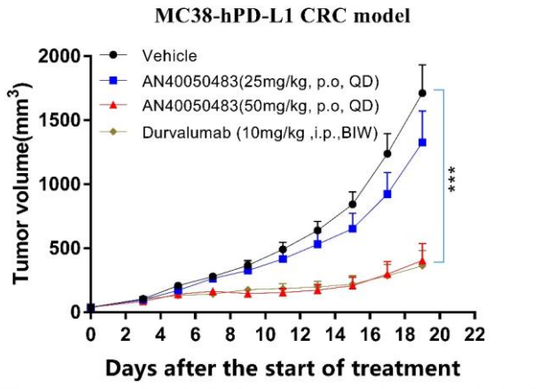
Market Opportunity

- No small-molecule PD-L1 inhibitor approved in any jurisdiction globally
- Effectively disrupt the interaction between PD-1 and PD-L1

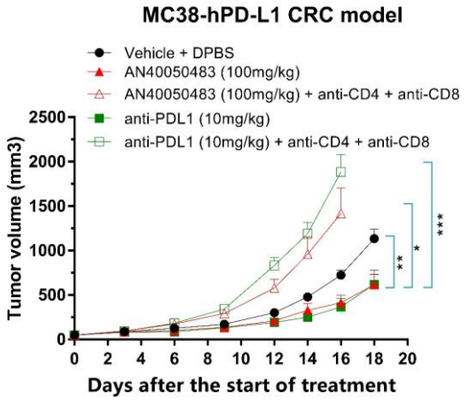
Benefits Over Antibodies

- Opportunity for oral administration, improved tumor penetration, and lack of immunogenicity

Robust Activity in Tumor Models



P value was calculated using Independent Samples t Test
*:p<0.05 **:p<0.01 ***:p<0.001



P value was calculated using Independent Samples t Test
*:p<0.05 **:p<0.01 ***:p<0.001

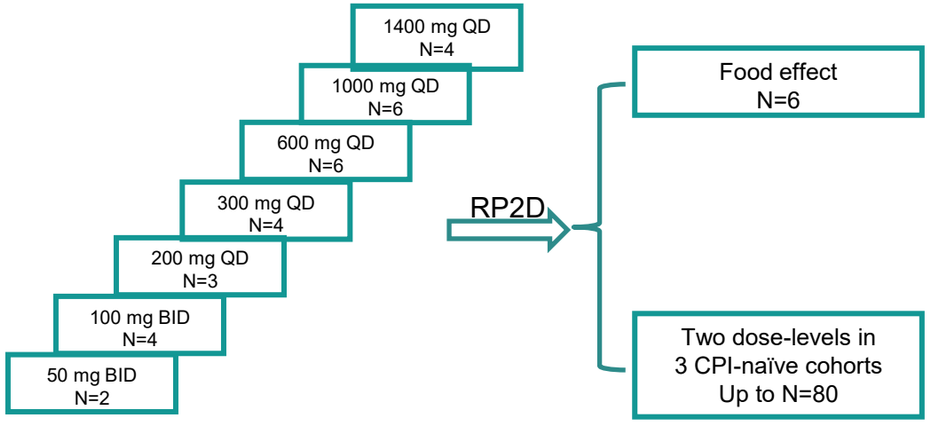
First-in-Human, Dose Escalation study of AN4005

Main inclusion criteria:

- Advanced unresectable or metastatic solid tumors, or r/r lymphomas
- No standard therapy available
- ECOG 0/1

Primary endpoints
Safety, tolerability, MTD and/or RP2D

Secondary endpoints:
PK, food effect, preliminary efficacy including ORR, PFS, DoR, OS



- ✓ 29 Patients were enrolled in 6 dose levels (50 mg BID to 1400 mg QD) in escalation part in the US and China
- ✓ No dose-limiting toxicities were observed and the MTD was not reached.
- ✓ Preliminary clinical efficacy was also observed
- ✓ The results of this study were presented at the 2024 SITC meeting
- ✓ Expansion cohorts for checkpoint inhibitor (CPI)-naïve patients are ongoing, and clinical update is expected to be presented at a conference in 2026

AN4005 Demonstrated Favorable Safety and Tolerability in Patients in Ph1 Dose Escalation Trial

Trial Registration: NCT04999384; Data cut-off data: 14 Oct 2024.

Table 1: Treatment-Related Adverse Events (TRAEs) Reported in ≥2 Patients with any Grade or in ≥1 Patient with Grade≥3

Preferred Term, n (%)	Total (N=25)	
	Any Grade	Grade≥3
Any TRAEs	20 (80%)	2 (8%)
Aspartate aminotransferase increased	4 (16%)	1 (4%)*
Nausea	4 (16%)	0
Vomiting	4 (16%)	0
Alanine aminotransferase increased	3 (12%)	1 (4%)*
Anaemia	3 (12%)	0
Decreased appetite	3 (12%)	0
Gamma-glutamyltransferase increased	3 (12%)	0
Blood alkaline phosphatase increased	2 (8%)	0
Blood bilirubin increased	2 (8%)	0
Gastrooesophageal reflux disease	2 (8%)	0
Hypoalbuminaemia	2 (8%)	0
Proteinuria	2 (8%)	0
Peripheral neuropathy	1 (4%)	1 (4%)**

*One (4%) patient in the 100 mg BID cohort experienced Grade 3 alanine aminotransferase increased and Grade 3 aspartate aminotransferase increased. These events were also considered irAEs by the Investigator.
**One (4%) patient in the 200 mg QD cohort experienced Grade 3 peripheral neuropathy which was also reported as an SAE. However, the Sponsor could not establish a causal association of the event with AN4005, due to confounders in this case.

- 25 patients with advanced solid tumors were enrolled in the U.S. and China. 44% had received prior IO treatment
- 20 (80%) patients experienced at least 1 TRAE; 2 (8%) patients experienced Grade 3 TRAEs. No Grade4 or TRAEs were observed
- 8 (32%) patients experienced 15 serious adverse events (SAEs); 1 (4%) patient experienced 1 SAE assessed by investigator as related to AN4005
- No patients experienced AEs leading to dose reduction of AN4005. 9 (36%) patients experienced at least 1 AE leading to dose interruption of AN4005. 4 (16%) patients had AE that led to discontinuation of AN4005
- No dose-limiting toxicities (DLTs) occurred at any dose level, and the MTD was not reached

Source: AN4005 Poster, Abstract #691, 2024 SITC Annual Meeting.

AN4005 Demonstrated Clinical Efficacy in Patients in Ph1 Dose Escalation Trial

Adlai Nortye

Trial Registration: NCT04999384; Data cut-off data: 14 Oct 2024.

Preliminary efficacy:

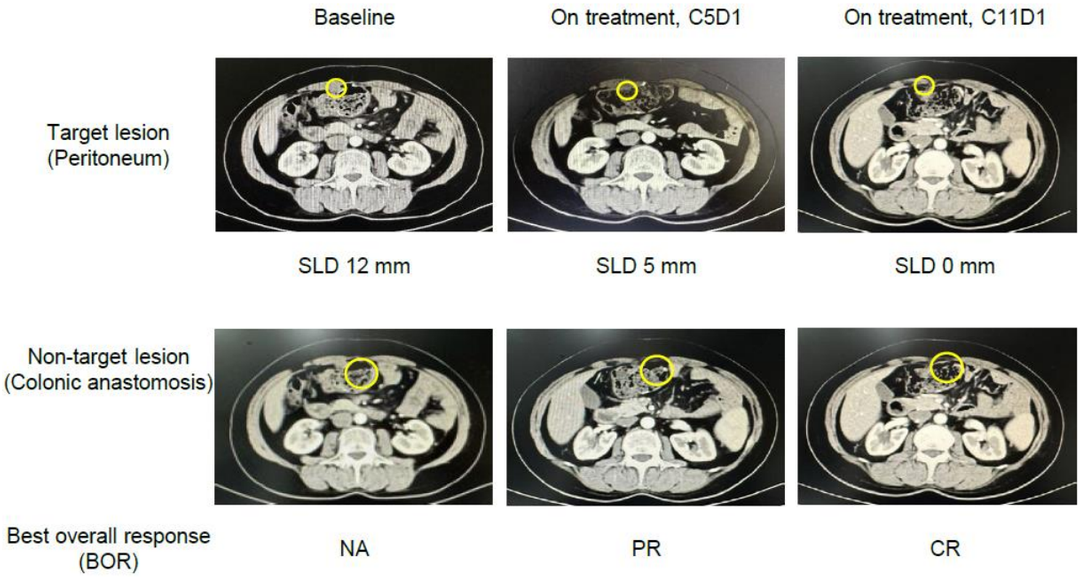
- Overall disease control rate (DCR) was 42% (10 in 24 efficacy-evaluable patients)
- One responder was observed at 300 mg QD cohort who received a confirmed complete response (CR) and maintained the CR after completing two years of planned treatment

✓ Demographics and baseline characteristics: 50-year-old Asian female, diagnosed with Stage IIIc colon cancer at initial diagnosis, metastatic disease to peritoneum (Stage IV) at baseline, with high microsatellite instability (MSI-H) / PD-L1 TPS 30%.

✓ Prior therapies: left colon extended radical resection followed by adjuvant chemotherapy; two lines of anti-PD-(L)1 mAbs at metastatic setting, achieved partial response (PR) in both lines. However, the intravenous therapy was interrupted due to the COVID-19 pandemic and the disease progressed.

✓ This case highlights the value of an oral PD-L1 inhibitor.

CT imaging of Target Lesion and Non-Target Lesion of the Responder



Abbreviations: SLD, sum of longest diameters of target lesions; NA, not applicable; PR, partial response; CR, complete response; C, cycle; D, day.

PK and RP2D Selection

• AN4005 exposure increased in a dose-dependent manner over the range of 50mg BID to 1000 mg QD.

• Based on an overall evaluation of safety, clinical benefit, and PK, the 600 mg QD and 1000 mg QD dose levels have been selected for further evaluation in the future expansion cohorts.

Source: AN4005 Poster, Abstract #691, 2024 SITC Annual Meeting.

AN4005: Development Strategy (Expansion Cohort)

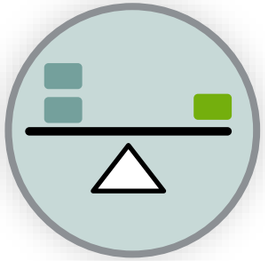
Differentiated Approach for AN4005

Expansion cohort aims to further evaluate the efficacy and safety of AN4005, benchmarking against those of aPD(L)1 mABs

- Evaluate anti-PD(L)1 naïve patients with indications fit for anti-PD(L)1 monotherapy
- Address geographical regions where insurance does not provide full coverage for approved indications

Improved Patient Convenience

- Monotherapy or in combination (e.g. cancer vaccine) in maintenance or adjuvant settings



Improved Safety Management

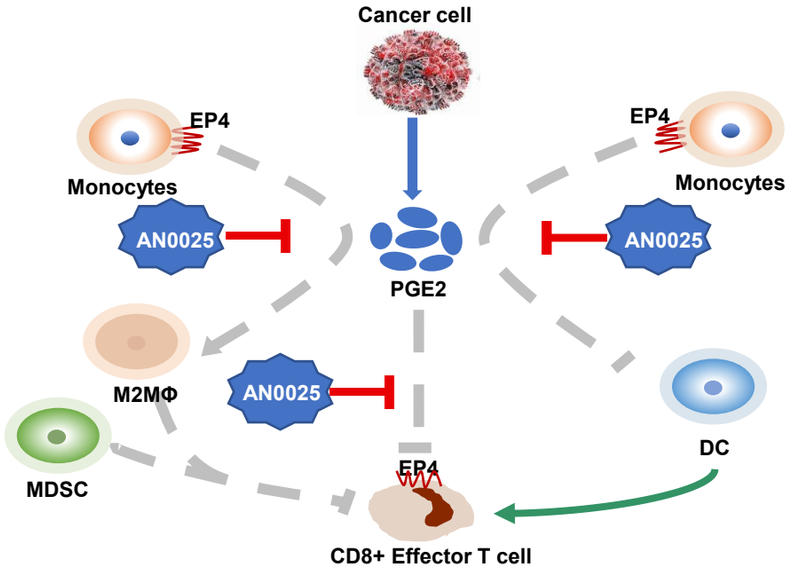
- AN4005 in combination with other small molecules, e.g. Ras inhibitor (AN9025), TKI

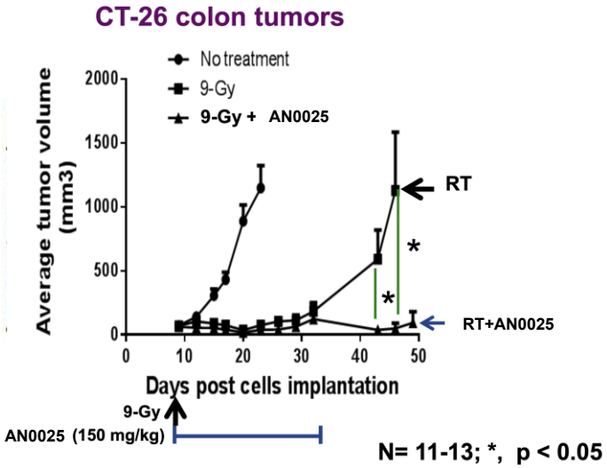
Other Assets - Clinical Update of Palupiprant (AN0025)

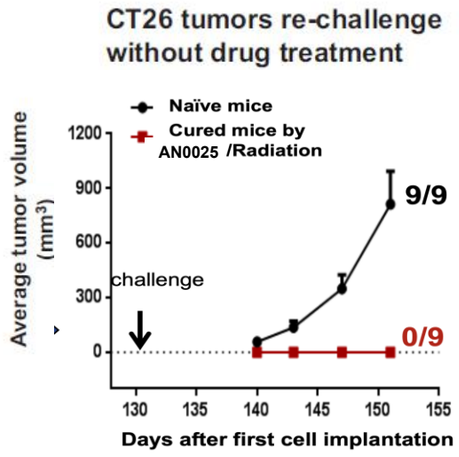
Palupiprant (AN0025): A Potential Enhancer of Radiotherapy

Adlai Nortye

Mechanism of Action







Phase 1b Clinical Trial of Preoperative AN0025 + Chemoradiotherapy in Rectal Cancer

Main inclusion criteria:

- Locally advanced rectal cancer, no metastatic disease
- Primary resection without CRT is unlikely to achieve clear margins as defined by MRI

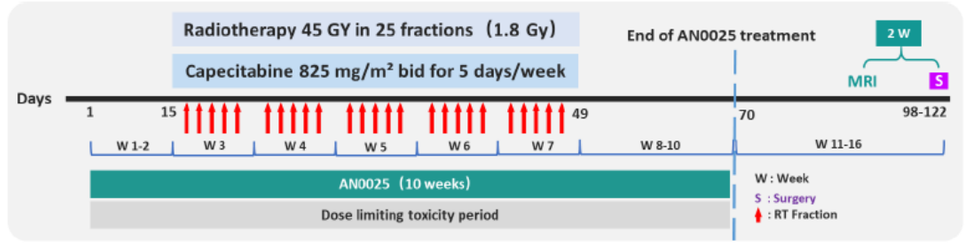
Primary endpoints

- Safety and tolerability
- MTD and/or RP2D

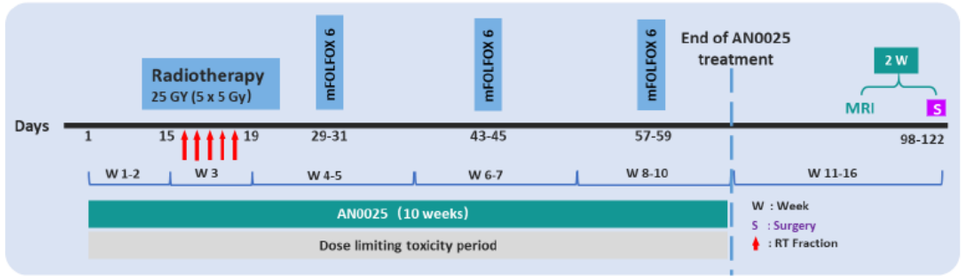
Secondary endpoints:

- pCR, CRM, pTRG, MRI-confirmed down staging in T stage, DFS, PK

Long course



Short course



- AN0025 was well tolerated in combination with CRT; No DLT occurred at either dose level, and the maximum tolerated dose (MTD) was not reached
- Preliminary efficacy results (20% clinical complete response (cCR) and 16% pathologic complete response (pCR)) are encouraging and support the further development of AN0025 in combination with CRT in this indication

Source: AN0025 Poster, Abstract #540P, 2019 ESMO Congress; AN0025 for treating advanced rectal cancer, Radiotherapy and Oncology 185 (2023) 109669

Phase 2 Clinical Trial of Preoperative Palupiprant (AN0025) + Chemoradiotherapy in Rectal Cancer

ARTEMIS (Augmenting RadioTherapy in REctal Cancer to Minimise Invasive Surgery)

A Phase 2, open-label, randomized controlled trial (140 pts)

Main inclusion criteria:

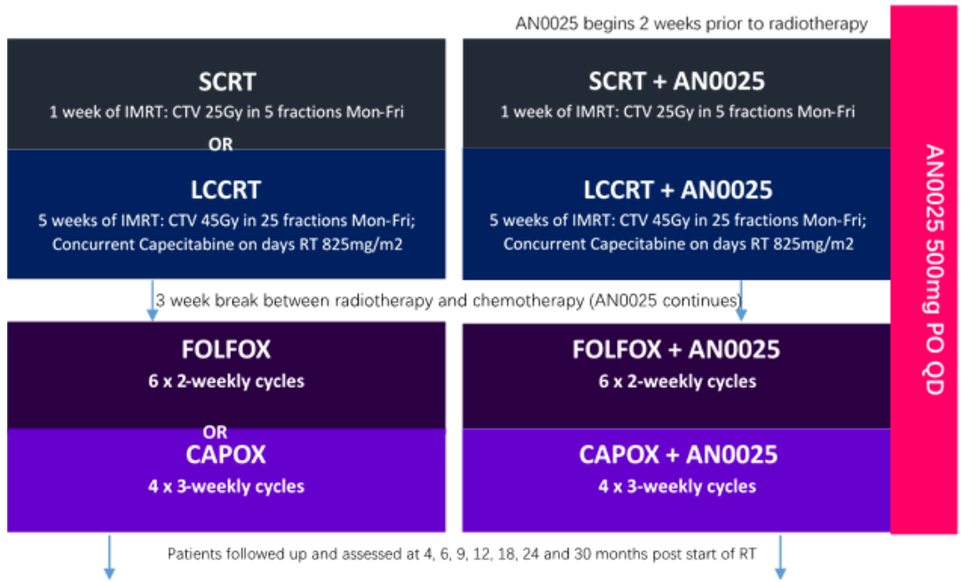
- Biopsy-proven rectal adenocarcinoma; ECOG PS 0-1
- T3b-4a or TanyN1-2 or TanyEMVI+ or with a threatened (<1mm) or involved mesorectal fascia resection margin, or low tumors with involvement of the anal intersphincteric plane or with levator involvement

Primary endpoints

- Clinical Complete Response rate at 6 months post start of RT

Secondary endpoints：

- Acute and late toxicity, HRQoL, surgical outcomes, response assessment, organ preservation, DFS, OS



LCCRT = long course chemoradiotherapy; SCRT = short course radiotherapy

Study Information

- Collaborated with Leeds University, UK
- The futility analysis of this Phase 2 trial was passed in March 2026
- Topline results are expected in H1 2027

Market Opportunities – neoadjuvant Rectal Cancer

Neoadjuvant rectal cancer in 2028

19,000 U.S. Incidence(1)

50,000 7MM Incidence(1)

Abbreviation: EMVI = Extramural vascular invasion.

(1) Data from Informa, 2023. 7MM stands for seven major markets (the U.S., the U.K., Germany, France, Italy, Spain, and Japan).