Creating Novel Therapeutics
By People With Excellent Expertise
In Drug Design

VORONOI

IR BOOK

June 16, 2025

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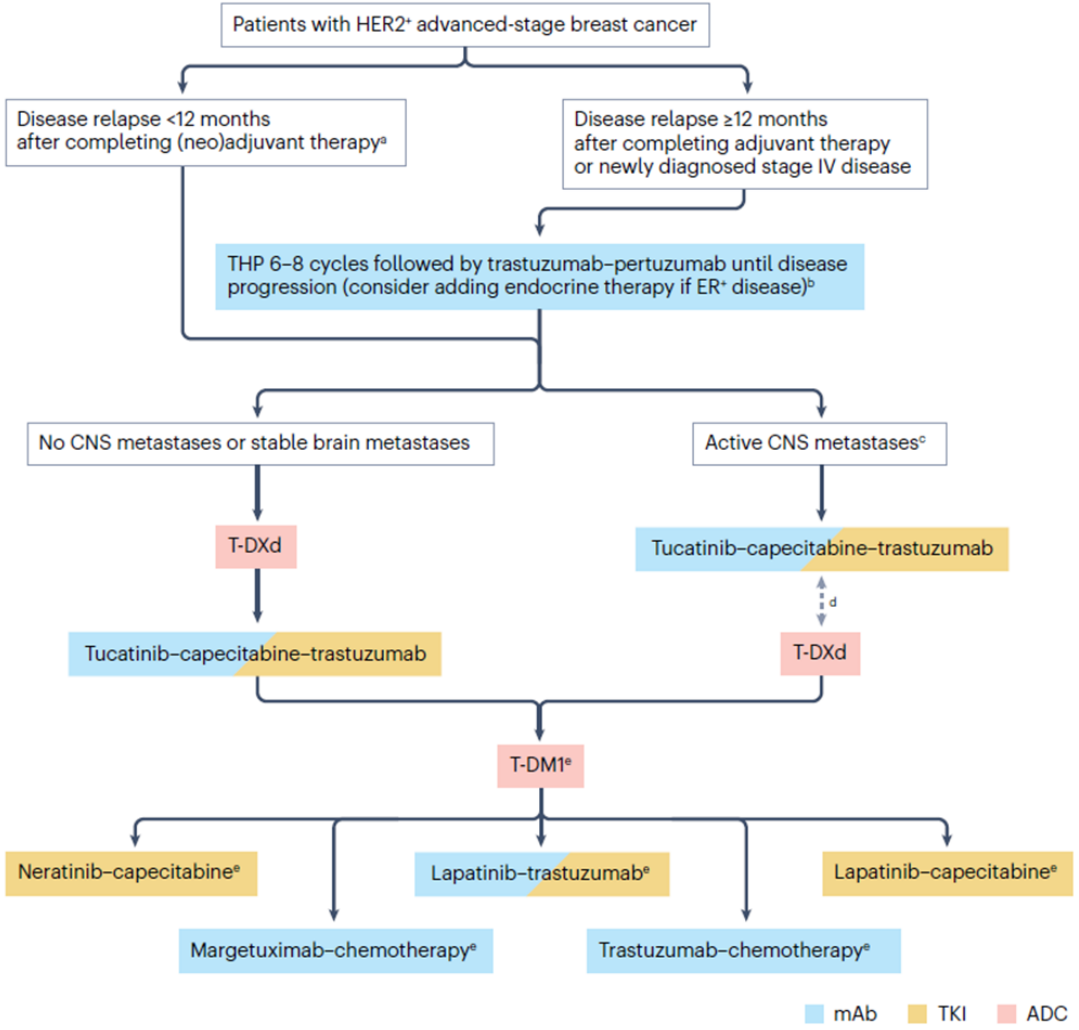
1. VRN10, A Brain Permeable and selective irreversible HER2 inhibitor for HER2 driven solid tumor
2. VRN16, a Novel PKMYT1 Selective Kinase Inhibitor
3. VRN19, USP1 inhibitor w/ excellent safety profile

VRN10, A Brain Permeable and selective irreversible HER2 inhibitor for HER2 driven solid tumor

Unmet Medical Needs in HER2-driven cancer



2024 Nature Review Clinical Oncology, Marra A et al.,

Key points to development

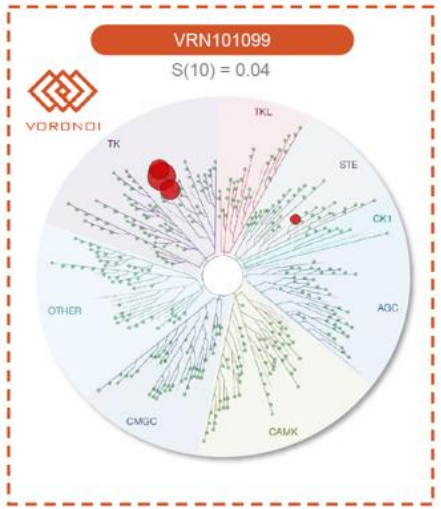
- High potency to overcome resistance mutations
- High Selectivity to HER2
- High Selectivity over EGFR
- Brain permeability for CNS metastasis

PD or discontinuation from SOC

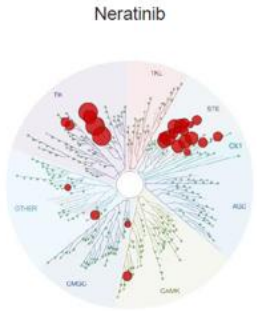
- Toxicity (ILD, Cardiactox, Skin rash, Diarrhea)
- Overcome TDxd or Abs Resistance
- Brain permeability for CNS progression

VRN10, Selective and Brain permeable HER2 Inhibitor

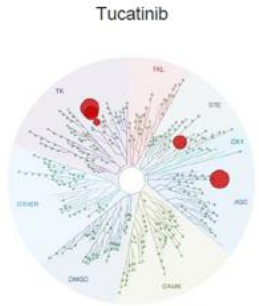
Kinase selectivity over human kinome



Irreversible



Irreversible



Reversible

Covalent, EGFR-sparing, Brain permeable

	Neratinib	Zongertinib	VRN10	ZN-1041	Tucatinib	Lapatinib
Binding mode	Covalent	Covalent	Covalent	Non-covalent	Non-covalent	Non-covalent
HER2 over EGFR	-	+++	+++	+	+++	-
Intracranial efficacy	-	-	+++	+++	+	N.D.

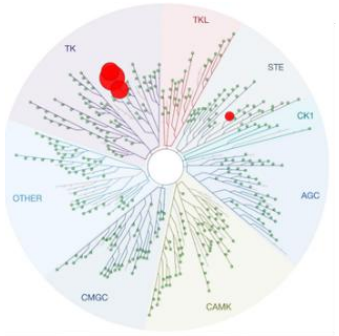
Characterization

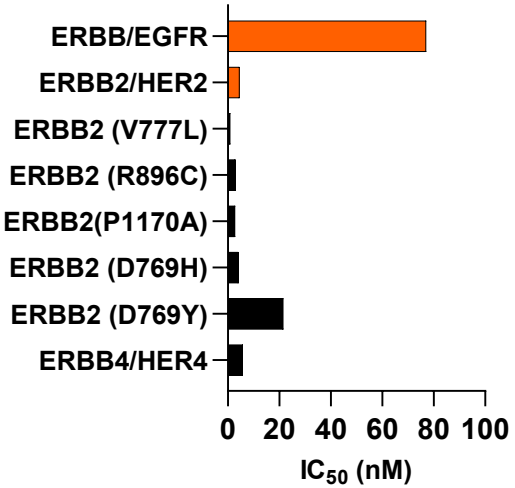
1. Mode of Action (Potency)

Selective covalent HER2 kinase inhibitor

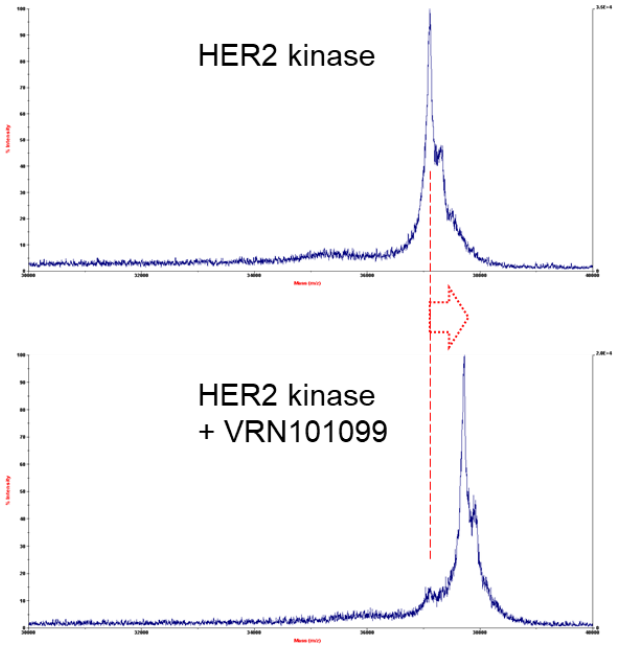
VRN10 irreversibly inhibits HER2 kinase activity and proliferation of HER2+ cancer cells

Selective to HER2 and HER2m

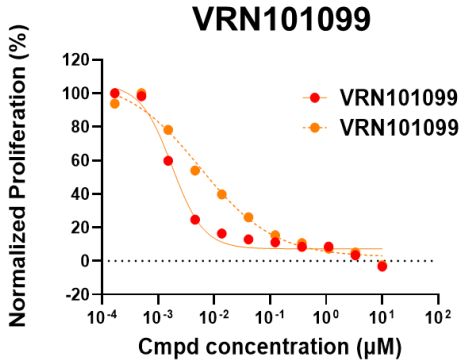


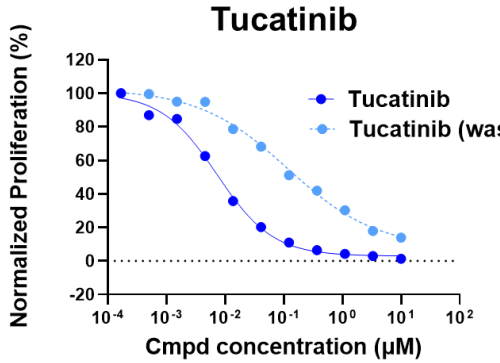


Covalent bond formation MALDI-TOF MS



Higher potency with longer resident time by covalent binding Wash-out Assay



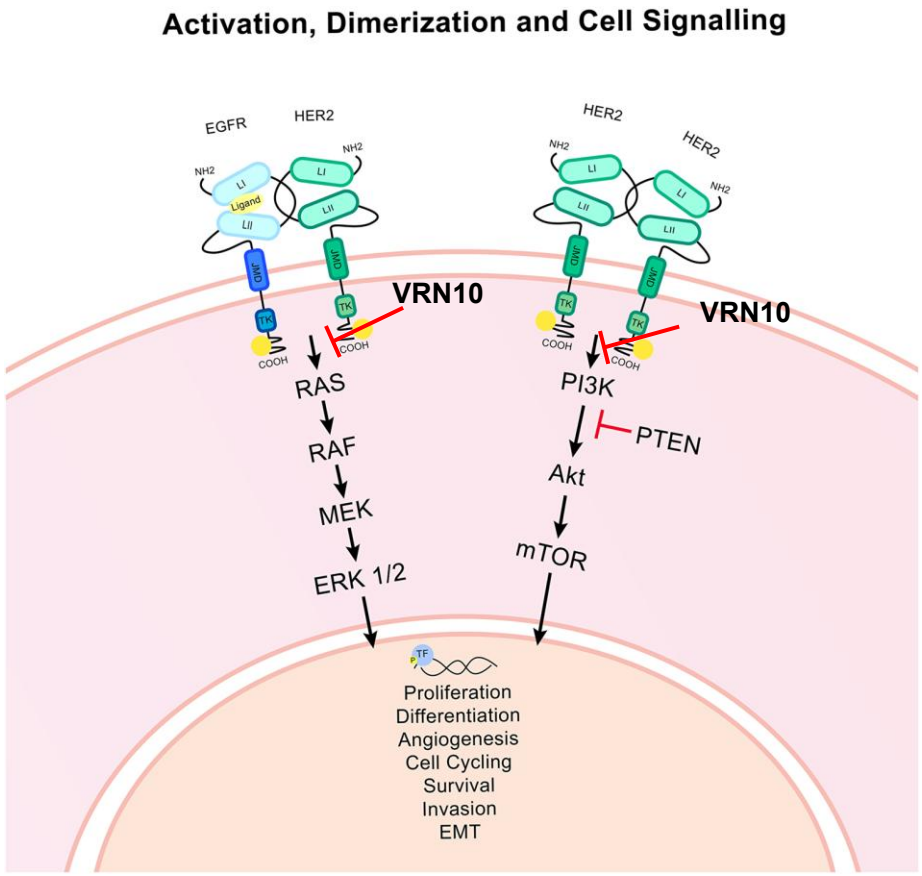


Compound	Proliferation assay BT474 GI_{50} (μM) - Washout	GI_{50} (μM) + Washout	Fold change
VRN101099	**0.002**	**0.009**	**3.6**
Tucatinib	0.009	0.193	21.9
Lapatinib	0.017	0.099	5.7
Neratinib	0.001	0.005	4.1

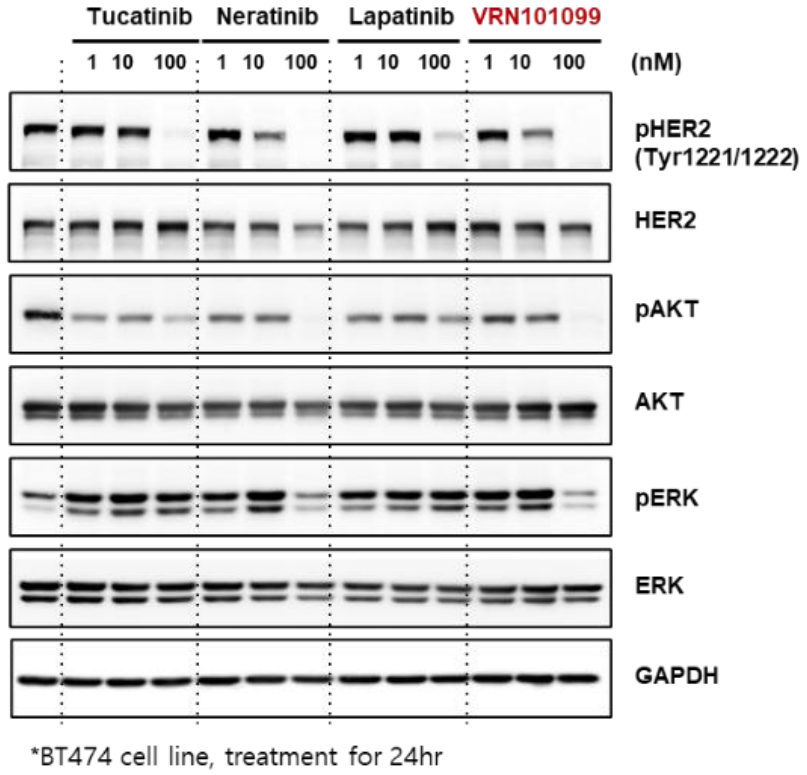
2024 EORTC-NCI-AACR, Kim S, et al.

Inhibition of HER2 signal pathways

VRN10 inhibits HER2 signal pathways in HER2-driven cancer cells



https://doi.org/10.18632/oncotarget.27789



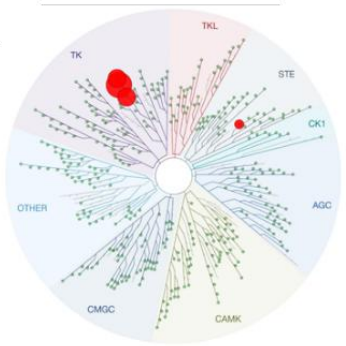
*BT474 cell line, treatment for 24hr

Characterization

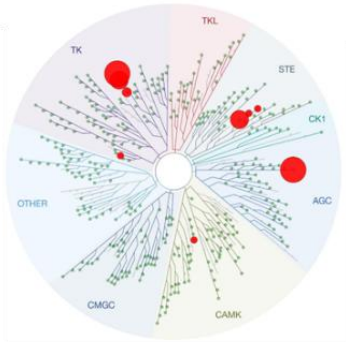
2. Selectivity (Safety)

Selectivity over other Kinases

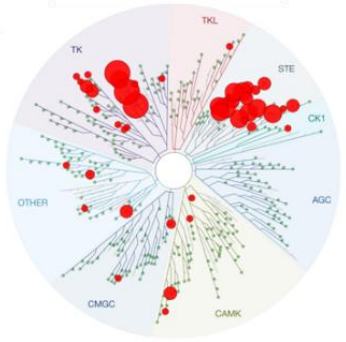
VRN101099
S(10) = 0.01



Tucatinib
S(10) = 0.015



Neratinib
S(10) = 0.069



Off target kinase profiles (<10% activity with 1000 nM)

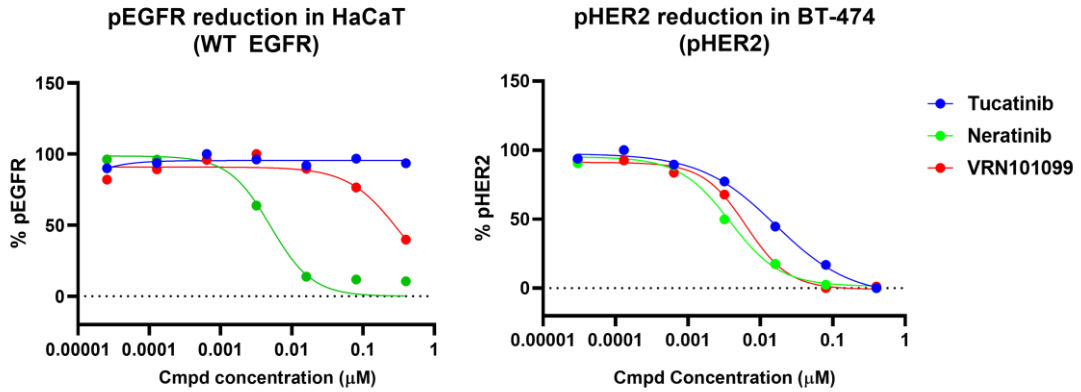
Gene	Activity
EGFR	0
ERBB2	0.35
ERBB4	0.45
MEK5	7

Gene	Activity
ADCK4	0
ERBB2	0
MRCKA	0
MEK5	0.25
EGFR	0.45
ERBB4	6

Gene	Activity	Gene	Activity
ERBB2	0	BLK	2
ERBB3	0	MAP3K3	2
ERBB4	0	LCK	2.2
LOK	0.1	MAP4K3	2.7
EGFR	0.15	MINK	3.2
MST4	0.4	TNIK	3.9
MAP4K5	0.6	HPK1	4.1
SLK	0.6	PIK4CB	5.8
MEK5	0.7	MAP4K4	6.9
MEK1	0.75	MAP4K2	7.7
MEK2	0.9	ICK	8
MST2	1	GAK	9.2
YSK4	1	MAP3K4	9.4
SNARK	1.9		

Selectivity over EGFR

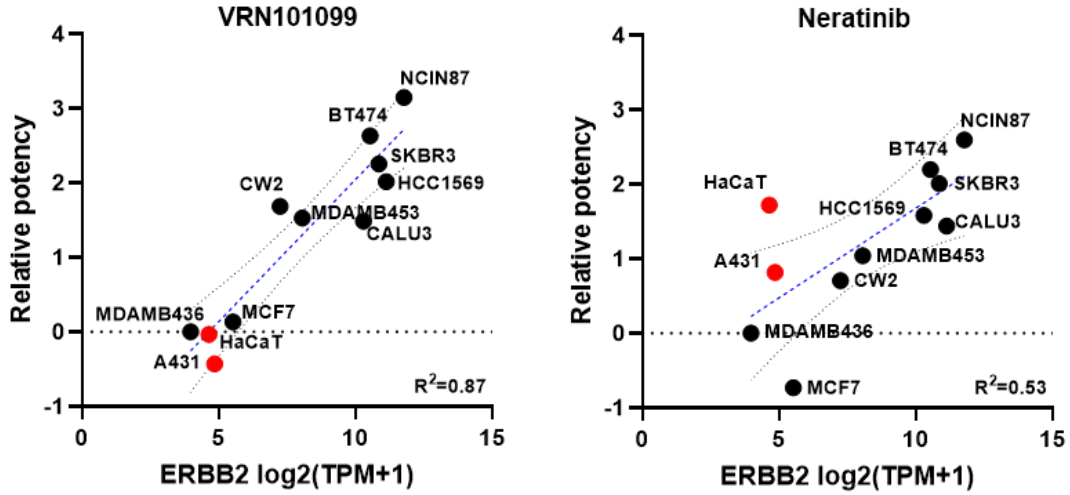
pHER2 vs pEGFR inhibition



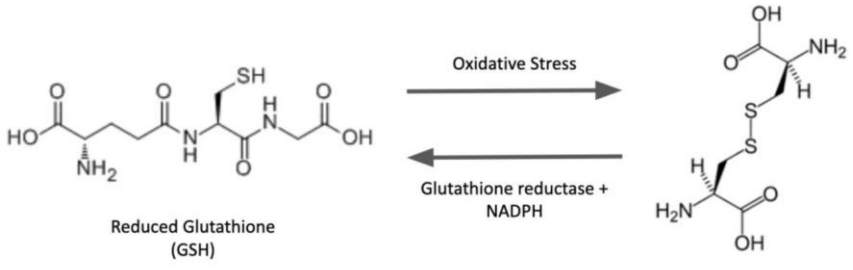
Cmpd.	Class	pIC50 (μM) HaCaT (pEGFR)	pIC50 (μM) BT-474 (pHER2)	Ratio EGFR/HER2
VRN101099	Covalent	0.3046	0.0074	41.2
Neratinib	Covalent	0.0045	0.0067	0.7
Tucatinib	Non-covalent	N.A	0.0269	N.A.

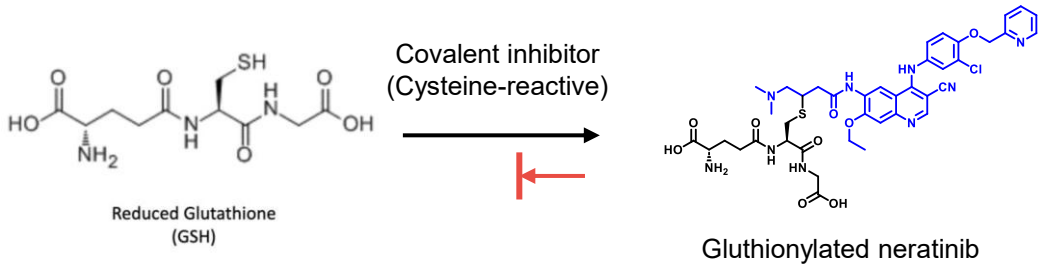
N.A, pIC_{50} couldn't be determined

Correlation between potency to HER2 expression



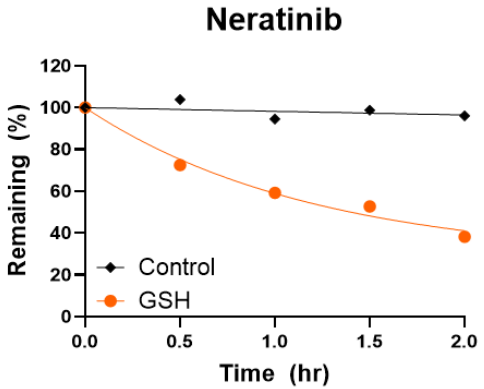
Selective covalent HER2 kinase inhibitor

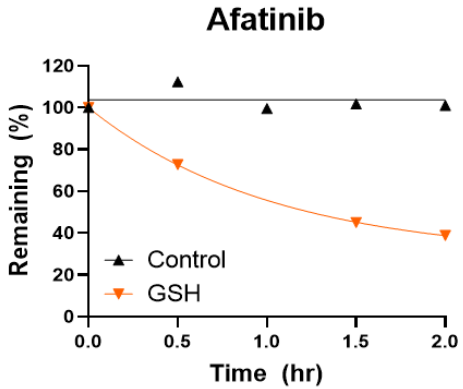




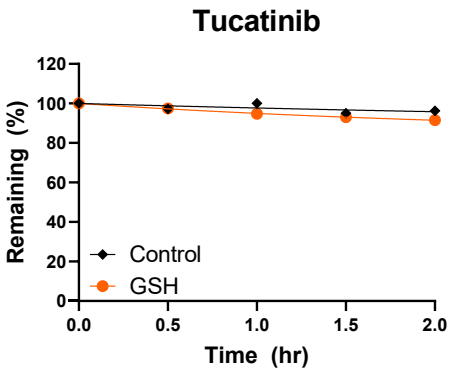
Low GSH reactivity / Less non-specific toxicity

Covalent inhibitors (non-selective)

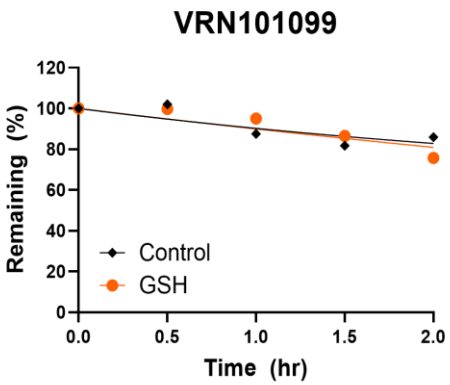




Non-covalent inhibitor



Covalent inhibitor (selective)



2024 EORTC-NCI-AACR, Kim S, et al.

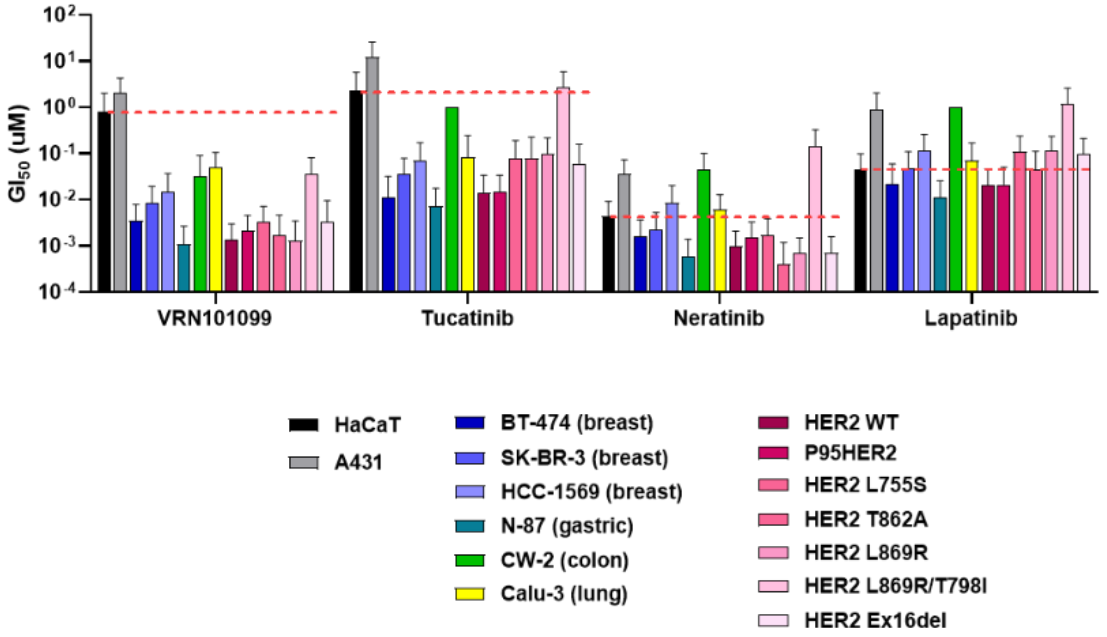
Characterization

3. Indications and Efficacy

Anti-proliferation potency against HER2-driven cancers

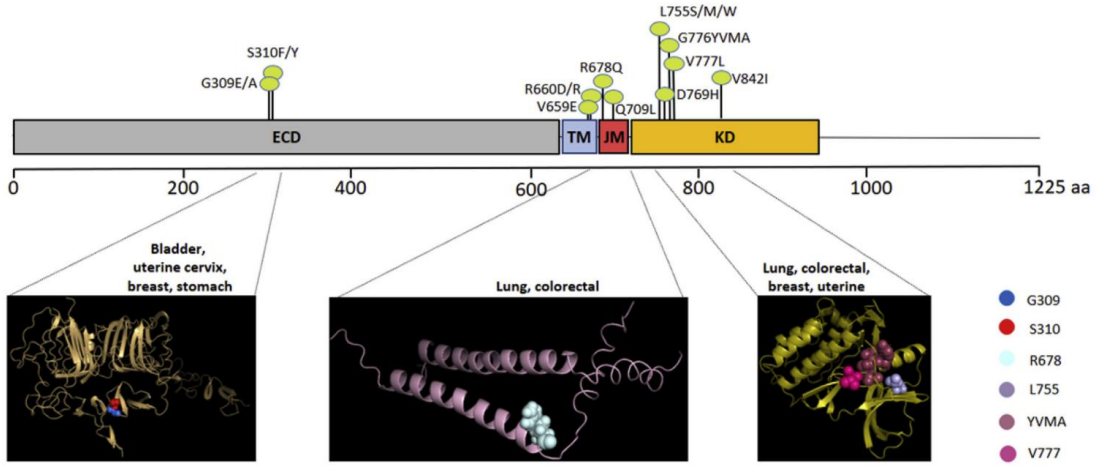
VRN10 has potency HER2+, HER2 activating TKI mutations, and Trastuzumab-resistant isoforms

High potency against cancer cells driven by
HER2 amp, HER2 mutants, and HER2 variants



	VRN101099	Tucatinib	Neratinib	Lapatinib
EGFR sparing (ratio to HaCaT GI_{50})	16~754	1~317	0~11	0~4

2024 EORTC-NCI-AACR, Kim S, et al.

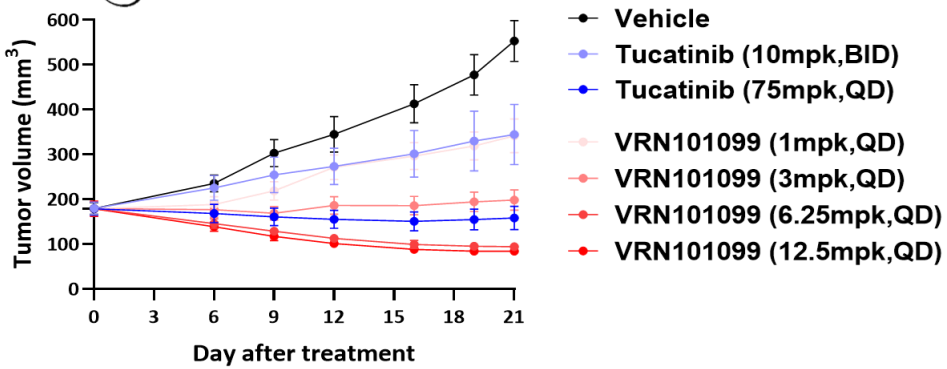


- HER2 amplification (HER2+)
- HER2 activating mutations / resistant mutations (L755S, T862A, L869R, T798I)
- Antibody-resistant isoform (p95HER2, Ex16del)

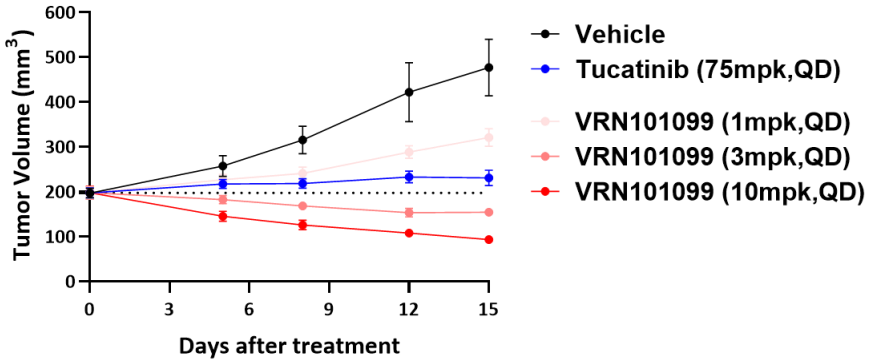
2019 Pharmacology & Therapeutics, Cocco E, et al.

Anti-tumor efficacy to HER2+ solid tumors and brain metastasis

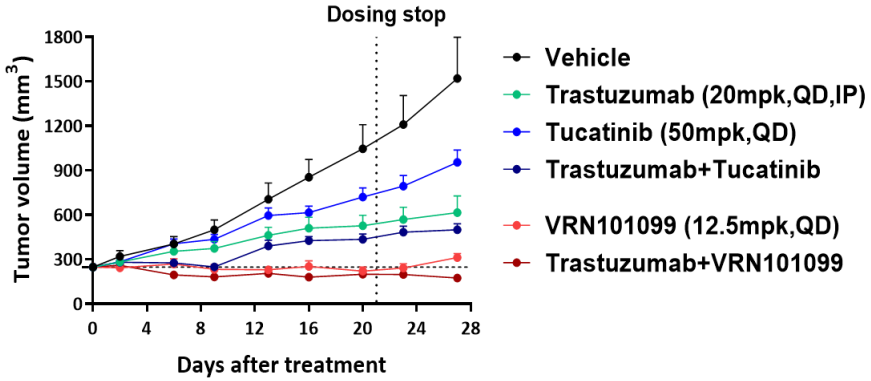
BT474 (HER2+ breast cancer)
SC xenograft model



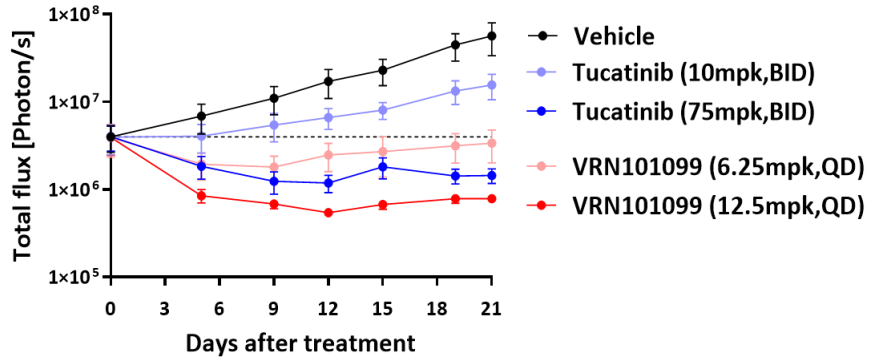
N87 (HER2+ gastric cancer)
SC xenograft model



CTG-0784 (HER2+ colon, PDX)
SC xenograft model



BT474 (HER2+ breast cancer)
Intracranial model



2024 EORTC-NCI-AACR, Kim S, et al.,

Anti-tumor efficacy to HER2+ solid tumors and brain metastasis

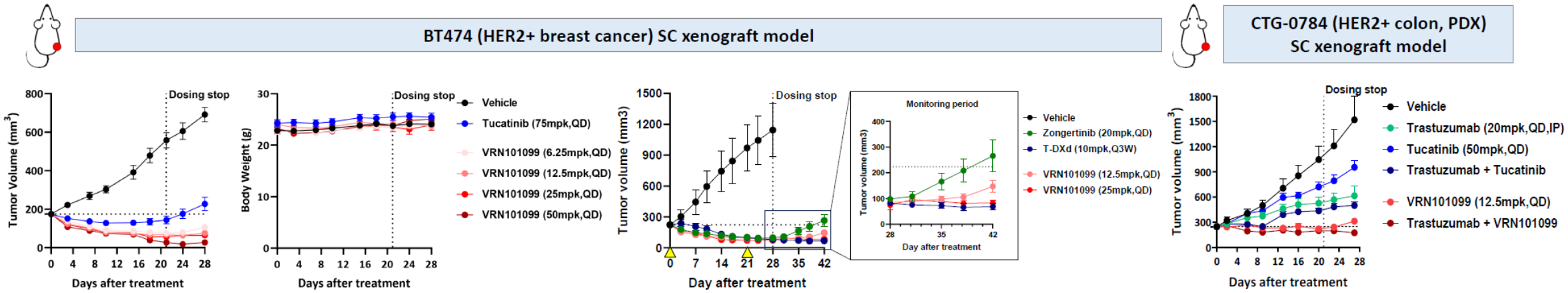


Figure 5. *in vivo* efficacy in HER2+ breast and colorectal cancer subcutaneous model

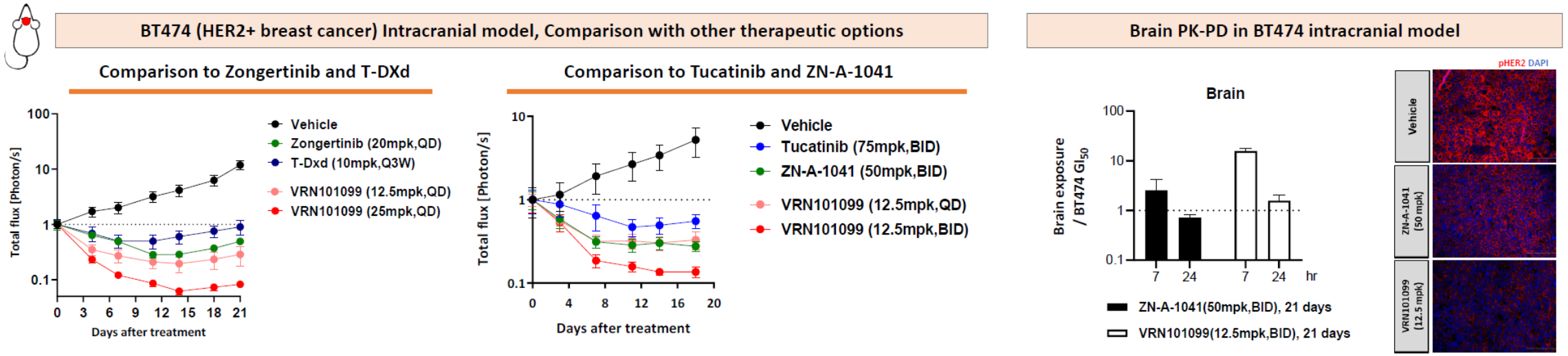


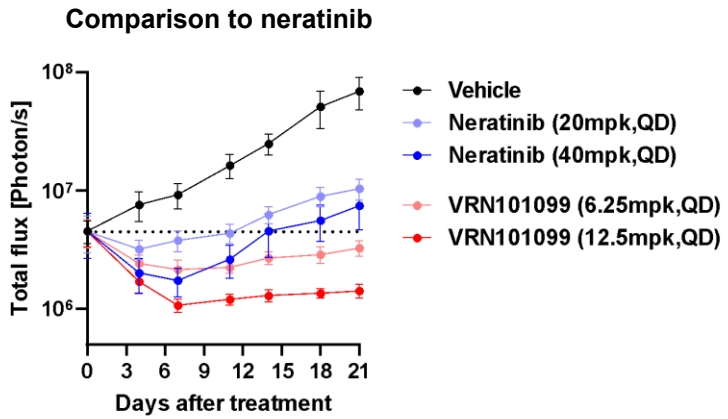
Figure 6. *In vivo* efficacy in the BT474 intracranial model. Brain PK was measured 21 days after the final dose. Immunostaining was performed 6hr after a single dose.

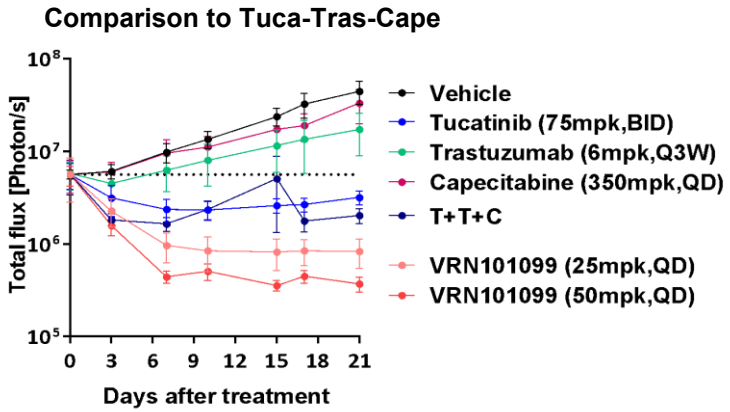
2025 AACR, Kim S, et al.

Additional comparison to other regimen in intracranial xenograft models

VRN10, superior anti-tumor efficacy compared to Tucatinib, Neratinib, Trastuzumab, T-DXd and Zongertinib

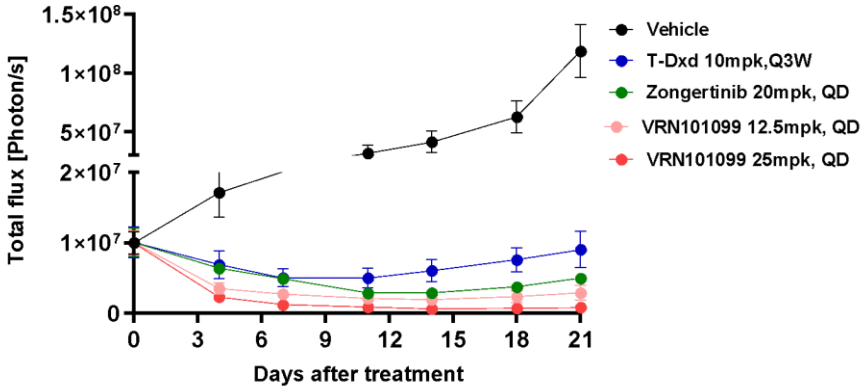
BT474 CNS model



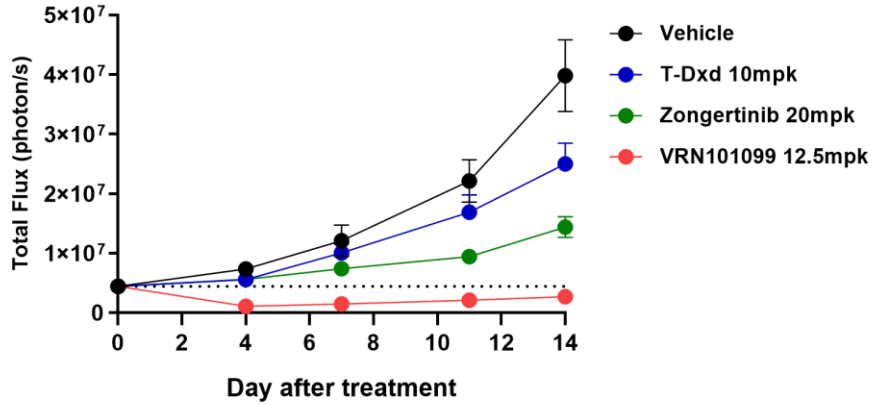


*TTC group 3/6 mice were dead

BT474 CNS model



SK-BR3 CNS model



Characterization

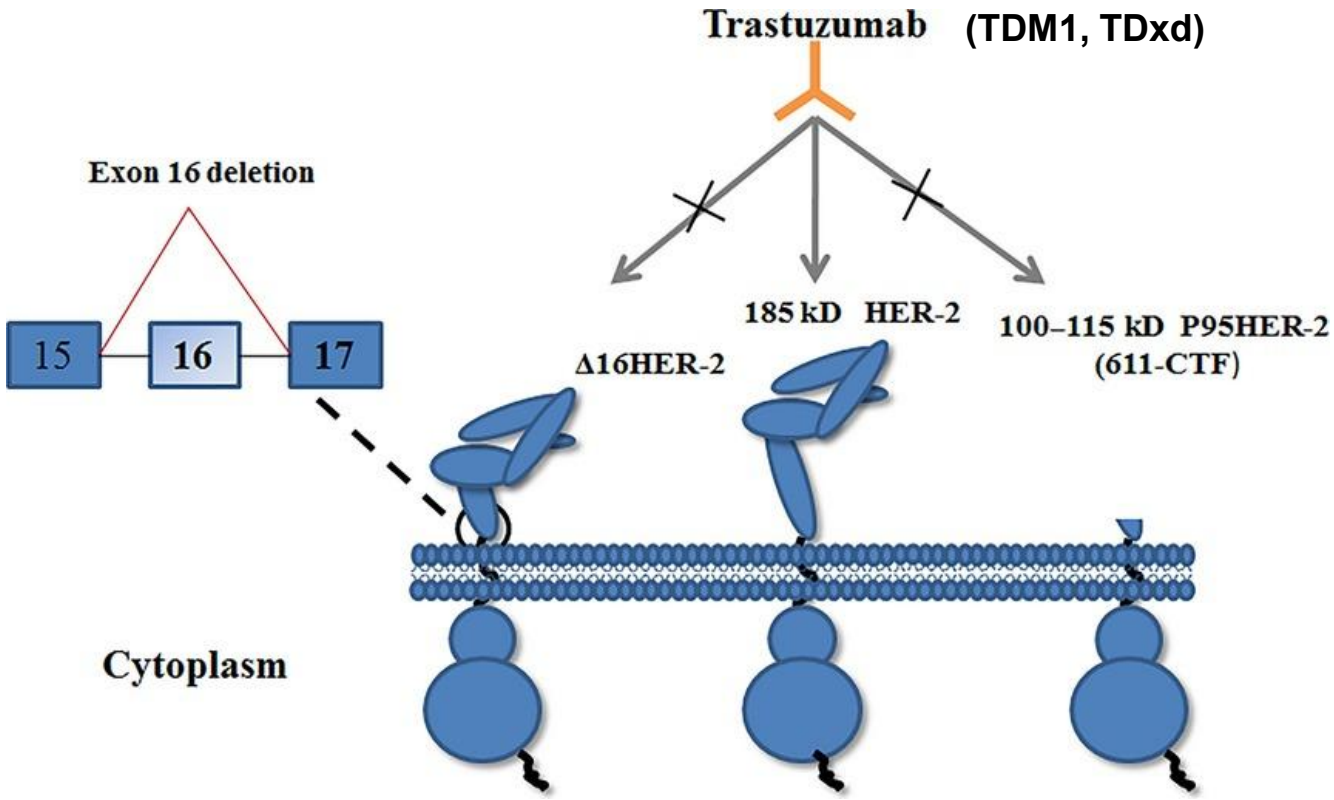
4. T-DXd resistance

Anti-tumor efficacy against acquired resistant cancer to T-DXd

Potential HER2-ADC Resistance Mechanism	T-DXd	Other HER2 ADC	Other HER2 TKI	VRN10
✓ Internalization defect	Low potency	Low	Effective	Effective
✓ Topoisomerase bypass or mutants	Low potency	case by case	Effective	Effective
✓ Extracellular domain shedding	No binding	No binding	Effective	Effective
✓ Exon16skipping	Low affinity	Low affinity	case by case	Effective
✓ BCRP overexpression	Dxd Efflux	Depending on phenotype	Depending on phenotype	Effective
✓ MRP1 overexpression	Dxd Efflux	Depending on phenotype	Depending on phenotype	Effective
✓ ARK1C overexpression	Dxd metabolism	Depending on phenotype	Depending on phenotype	Effective
✓ Decrease of HER2 expression	Low binding	Low binding	case by case	tumor by tumor

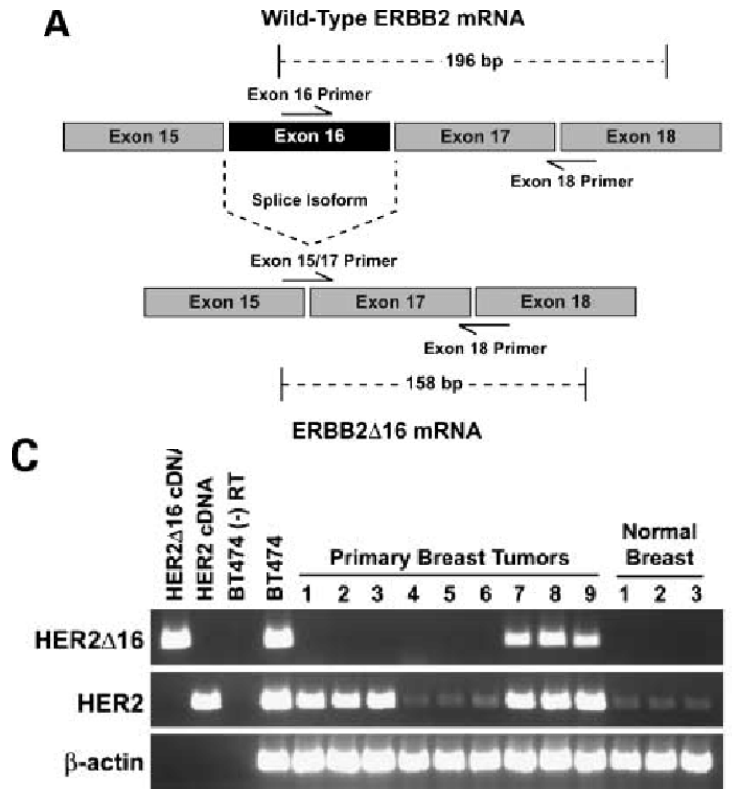
Efficacy to HER2 variants resistant to Trastuzumab (T-DM1, T-Dxd)

Extracellular shedding (p95HER2) and Exon16 skipping (Δ16HER2) can't be recognized by HER2 antibody



J. Cell. Mol. Med. Vol 19, No 12, 2015 pp. 2691-2701

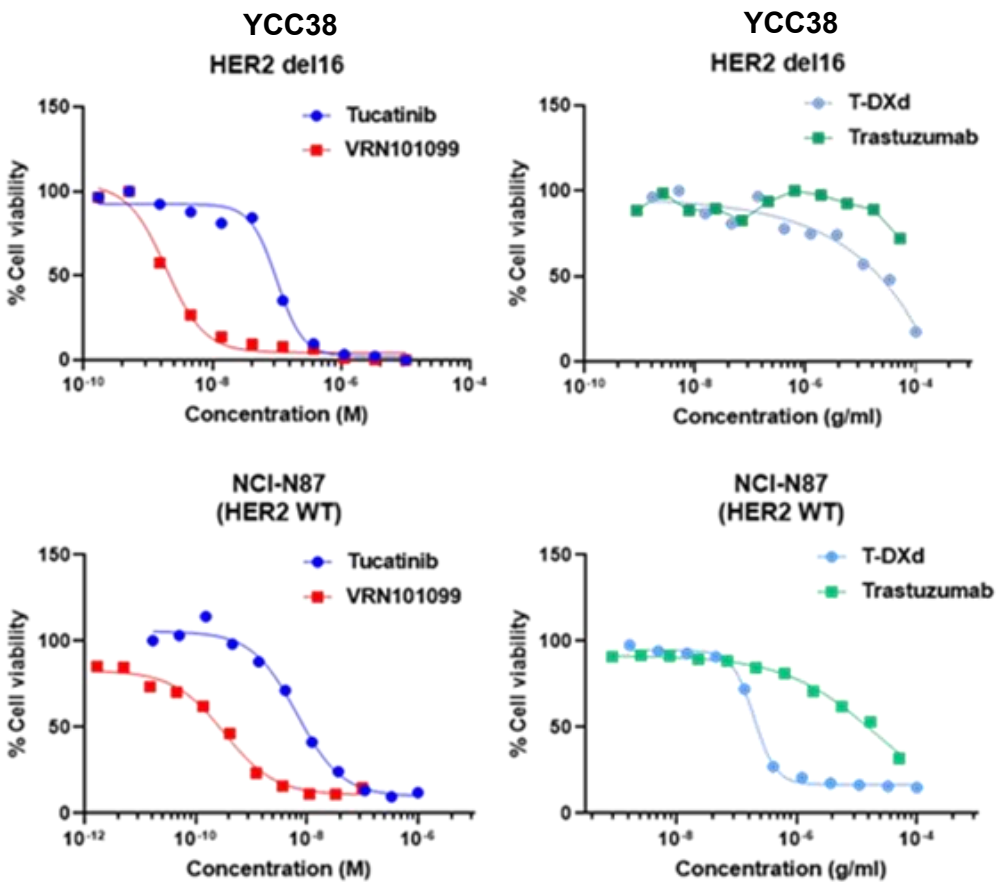
Expression of HER2 and Δ16HER2 in human tissues and breast tumors

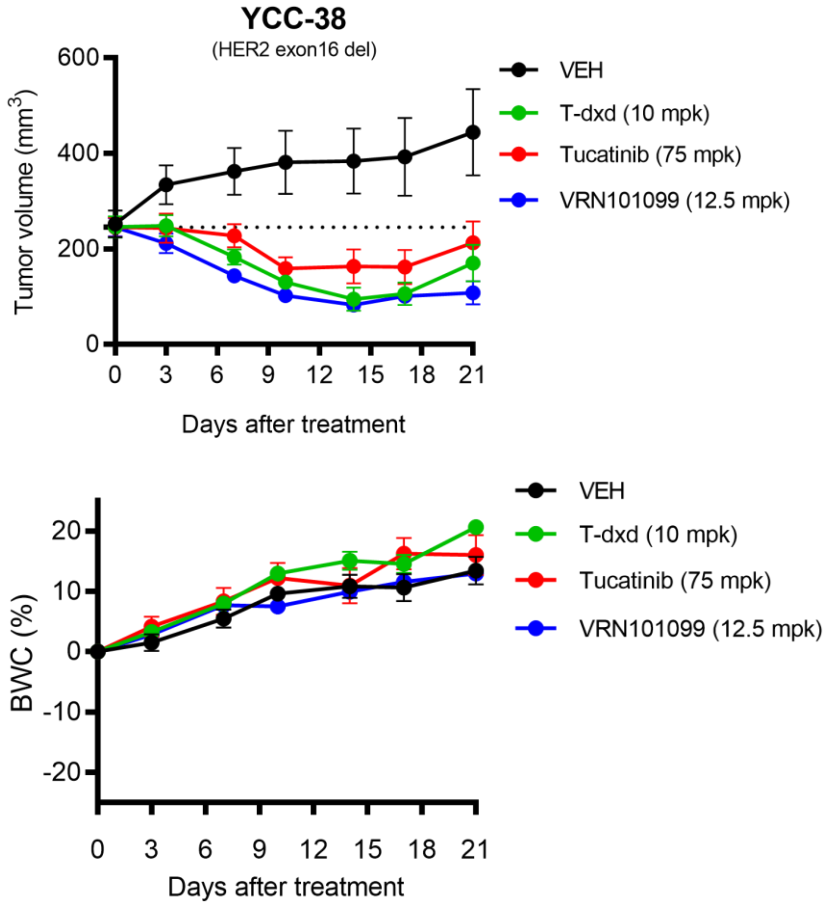


Mol Cancer Ther 2009;8(8). August 2009

YCC38, GC with HER2-Exon16skip mutation

VRN10 showed tumor regression efficacy against tumor expressing Δ16HER2 variant

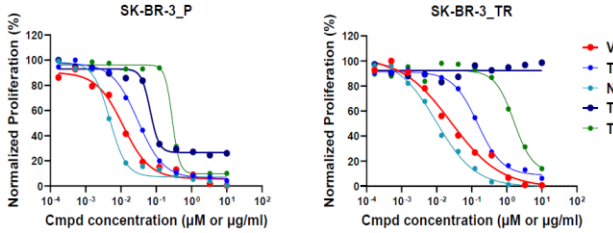




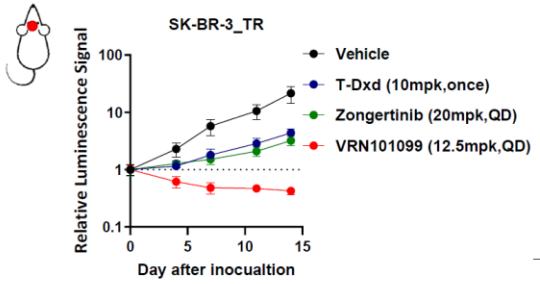
Anti-tumor efficacy against acquired resistant cancer to T-DXd

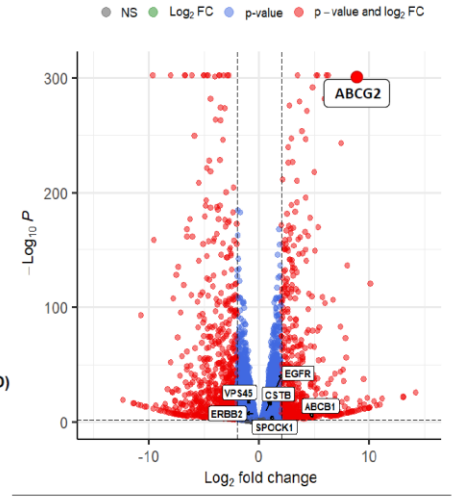
Overcoming T-DXd resistant mechanism, including ABCG2 overexpression and AKR1C overexpression

Overcome T-DXd resistance mechanisms (1)

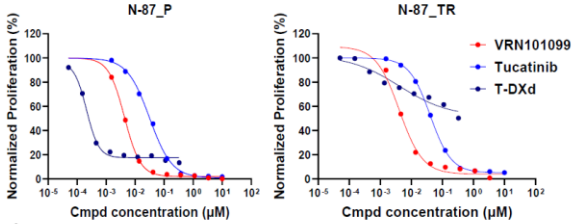


Compound	Proliferation Assay		
	GI_{50} (μM or μg/ml)		Fold change
	SK-BR-3_P	SK-BR-3_TR	
VRN101099 (μM)	0.012	0.024	2.1
Tucatinib (μM)	0.030	0.147	4.9
Neratinib (μM)	0.005	0.010	2.0
T-DXd (μg/ml)	0.068	>10.000	>147.7
T-DM1 (μg/ml)	0.285	1.602	5.6

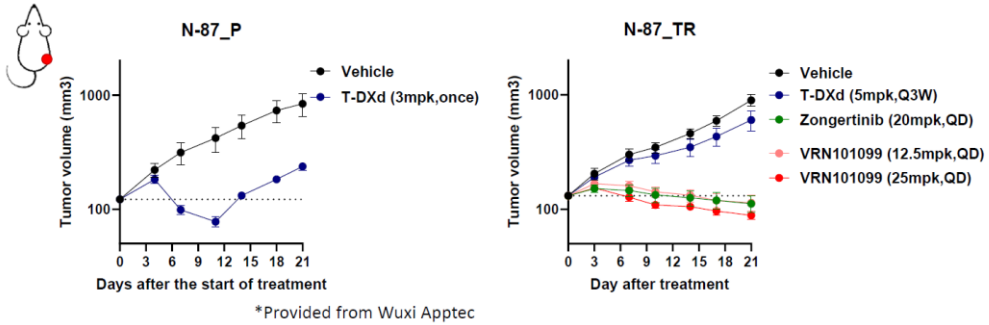




Overcome T-DXd resistance mechanisms (2)



Compound	Proliferation Assay		
	GI_{50} (μM)		Fold change
	N-87_P	N-87_TR	
VRN101099	0.004	0.005	1.1
Tucatinib	0.031	0.041	1.3
T-DXd	0.0002	>0.33	>1,650.0



*Provided from Wuxi Apptec

2025 AACR, Kim S, et al.

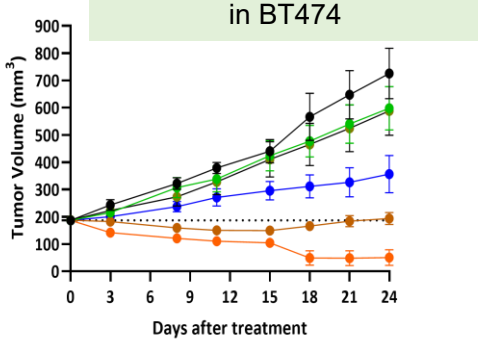
Characterization

5. Combi Potential

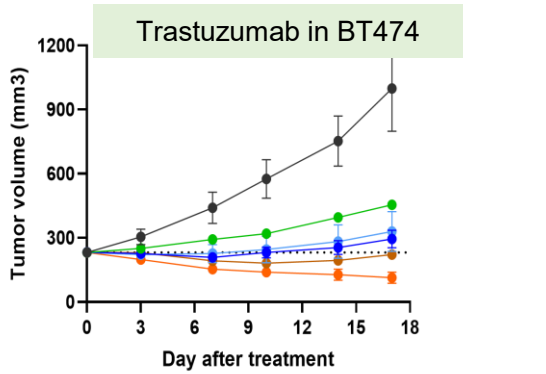
Combinatory Potential with Antibody or ADC

Combination in SC xenograft models

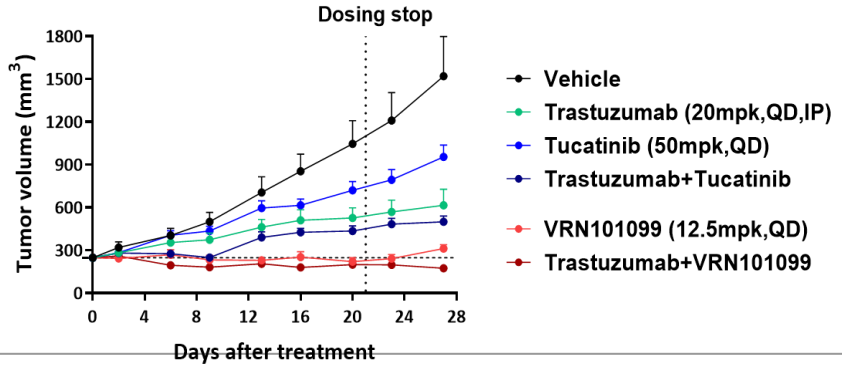
Trastuzumab + Pertuzumab in BT474



Trastuzumab in BT474

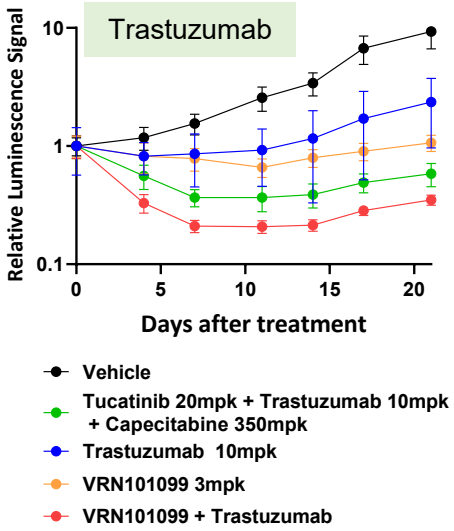


Trastuzumab in CRC PDX

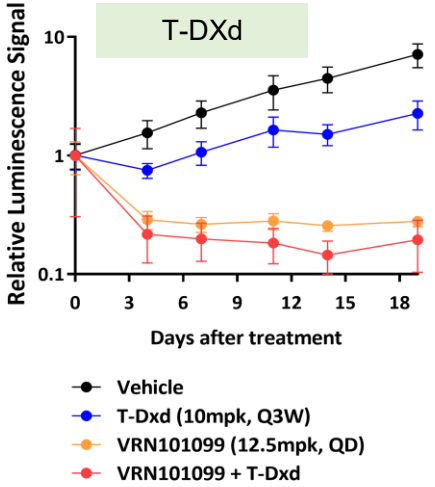


Combination in BT474 intracranial models

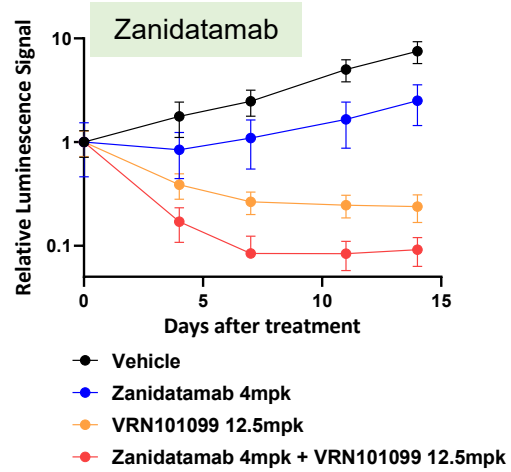
Trastuzumab



T-DXd



Zanidatamab



Clinical trials

Phase 1a, Dose Escalation

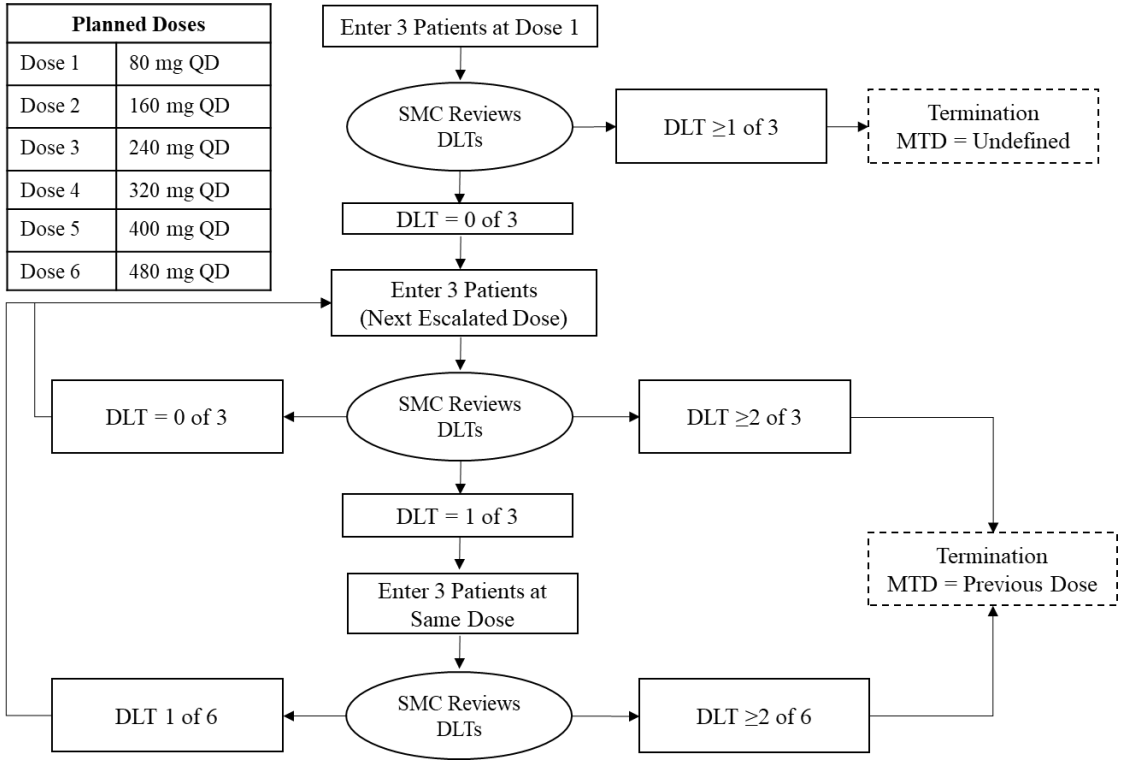
Key inclusion: Solid tumors with documented HER2+ (IHC1~3+, FISH+, or NGS) or HER2 activating mutants

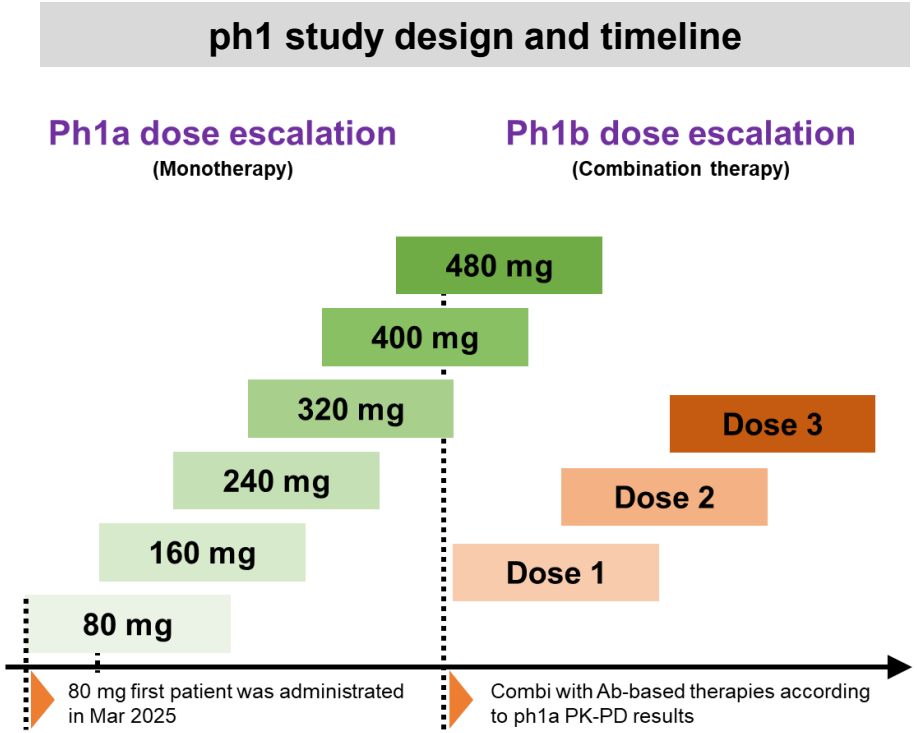
Study design: A standard "3+3"

Sites: AU 2 sites + KR 5 sites

FIH: Mar 2025

Planned Doses	
Dose 1	80 mg QD
Dose 2	160 mg QD
Dose 3	240 mg QD
Dose 4	320 mg QD
Dose 5	400 mg QD
Dose 6	480 mg QD



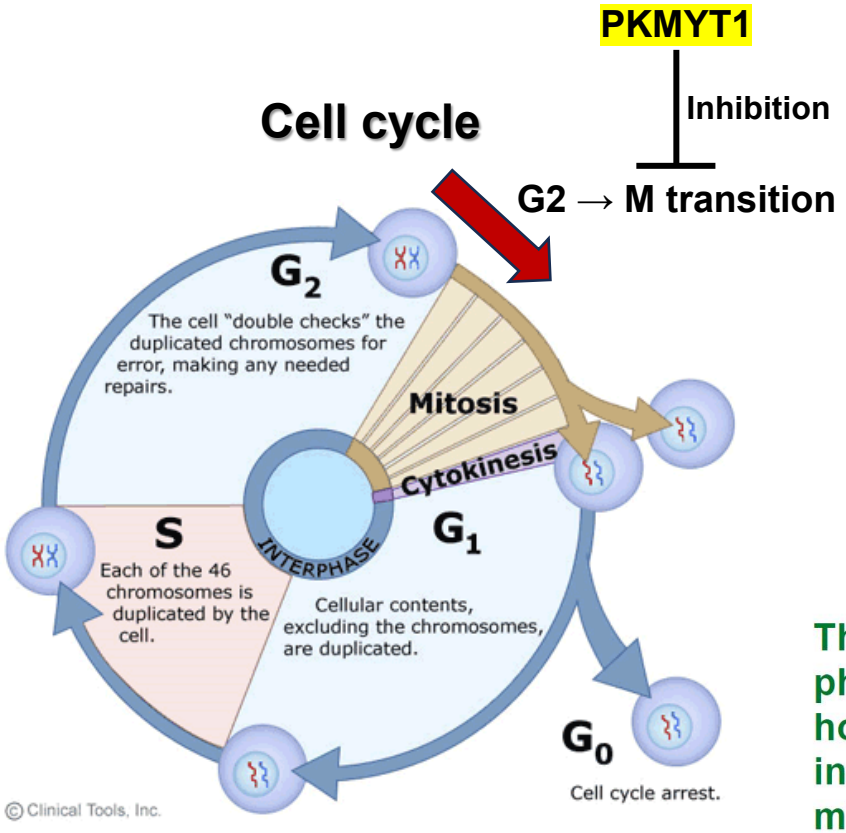


Abbreviations: DLT = dose-limiting toxicity; MTD = maximum tolerated dose; QD = once daily; SMC = Safety Monitoring Committee.

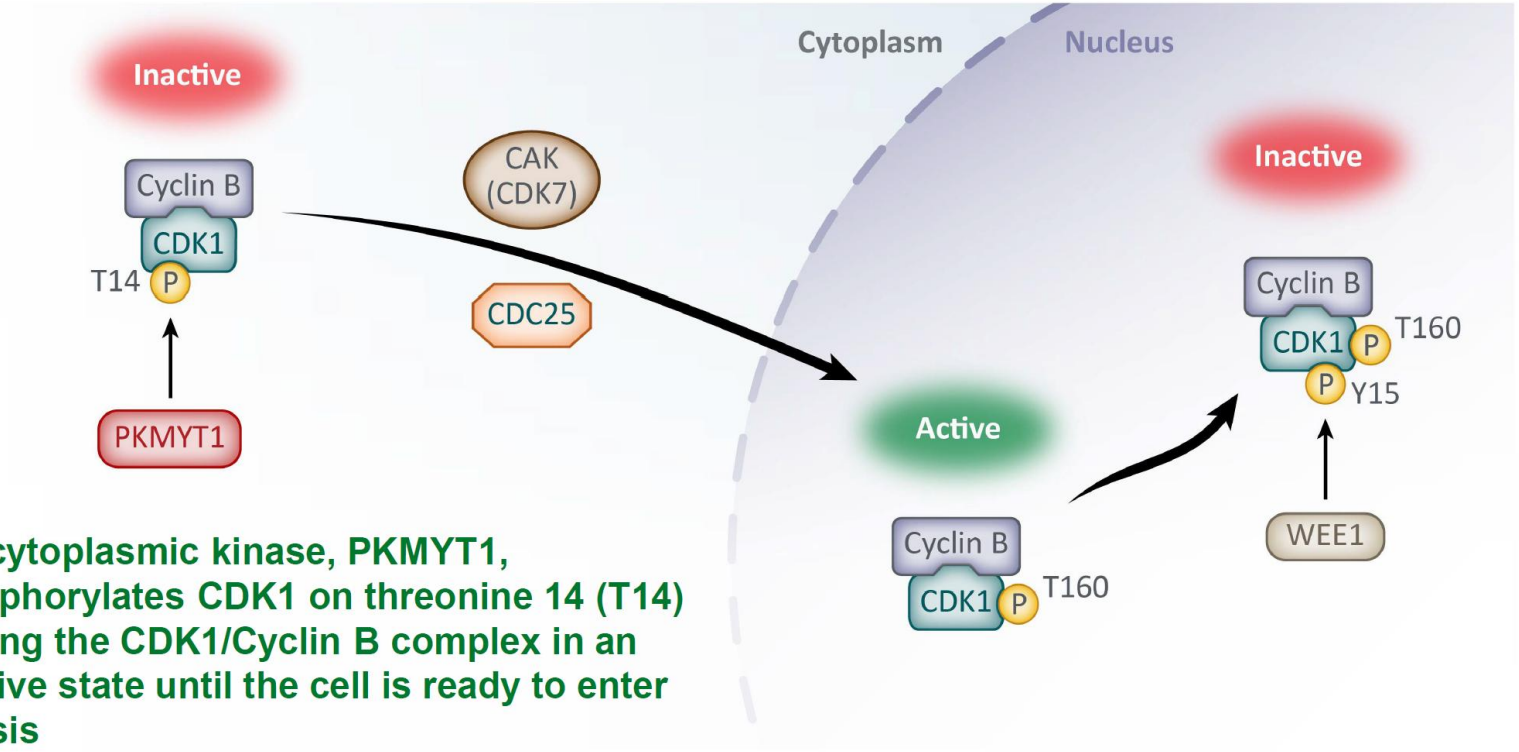
VRN16, a Novel PKMYT1 Selective Kinase Inhibitor

Function of PKMYT1 kinase: Regulation of G2/M phase transition

PKMYT1 selectively regulates CDK1 during the G2/M phase transition to prevent mitotic entry in the presence of DNA damage.



https://le.ac.uk/vgec/topics/cell-cycle/the-cell-cycle-higher-education



The cytoplasmic kinase, PKMYT1, phosphorylates CDK1 on threonine 14 (T14) holding the CDK1/Cyclin B complex in an inactive state until the cell is ready to enter mitosis

23

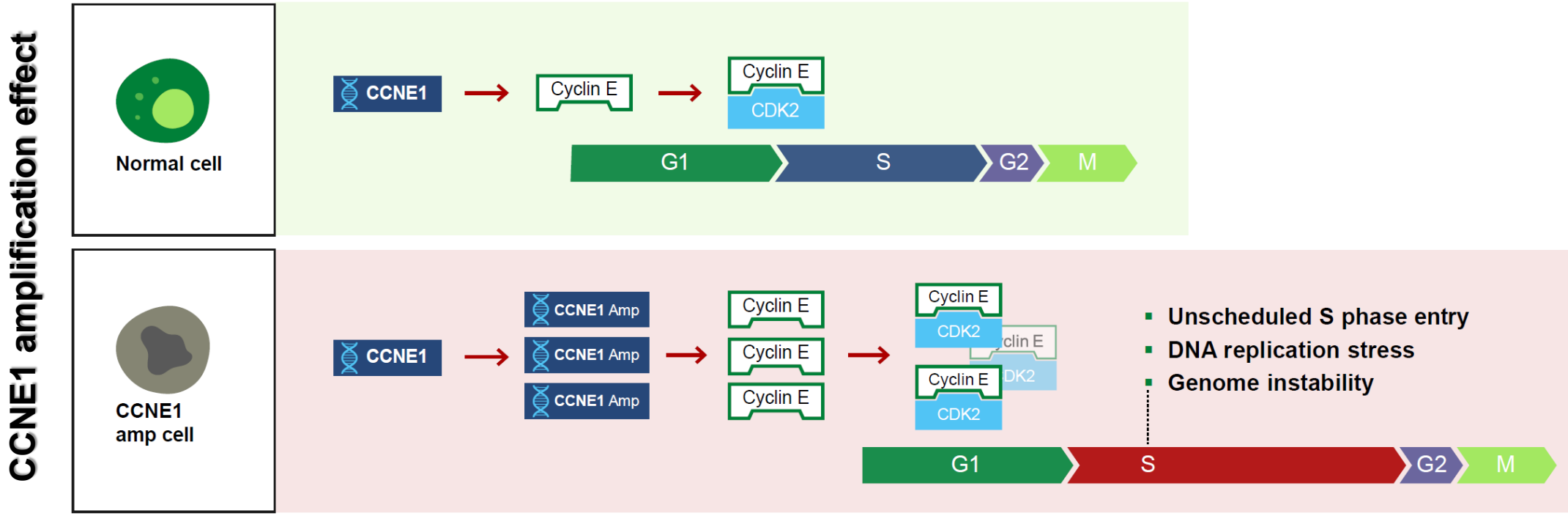
REPARE THERAPEUTICS

Repare Corporate Presentation, Q3 2023.

CCNE1 amplification: A biomarker for the PKMYT1 inhibitors

CCNE1 amplification is observed in 8% of all solid tumor patients and is particularly prevalent in ovarian cancer (31%), gastric cancer (14%), and breast cancer (8%).

CCNE1 amplification drives premature S-phase entry, resulting in genome instability due to DNA replication stress.

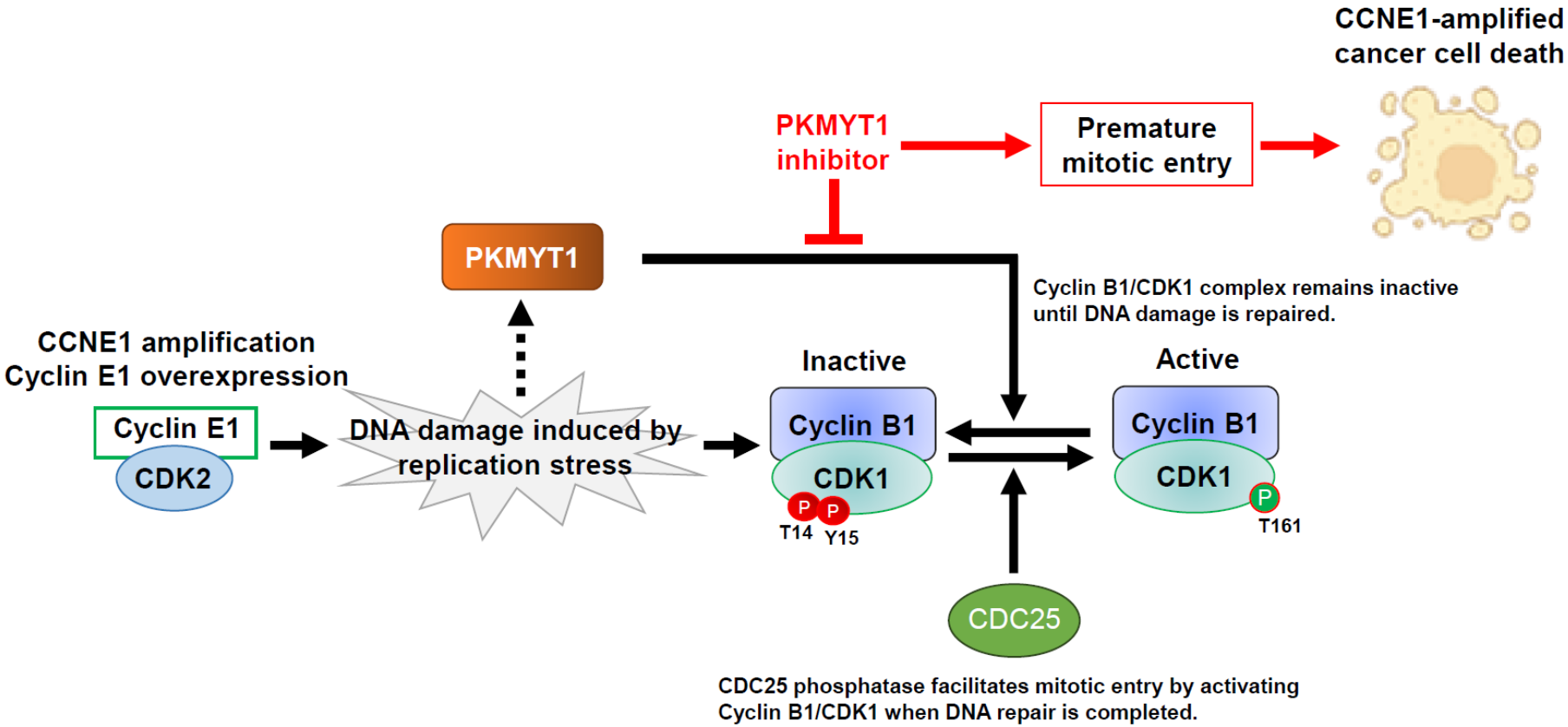


Repare Corporate Presentation, June 2023.

Synthetic lethality: CCNE1 amplification with PKMYT1 inhibition leading to cell death

PKMYT1 inhibition forces cancer cells to prematurely enter mitosis without repairing DNA damage, ultimately leading to cell death.

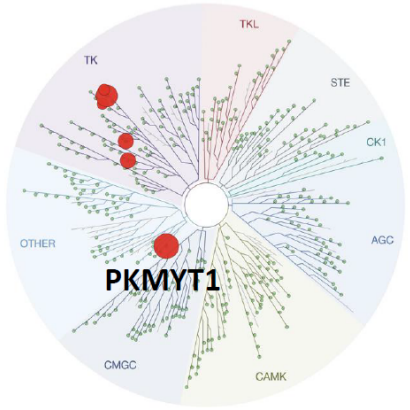
Despite the significant patient population harboring CCNE1 amplification, no targeted therapy has been approved, emphasizing critical unmet medical needs.



VRN16: A highly selective PKMYT1 kinase inhibitor

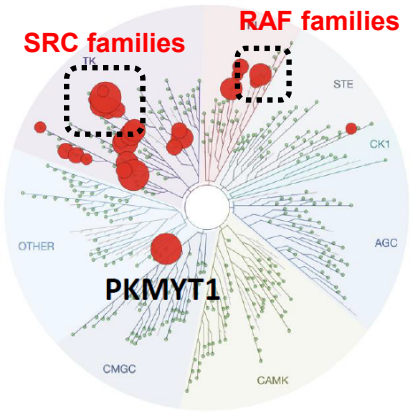
VRN16

S(10) = 0.01

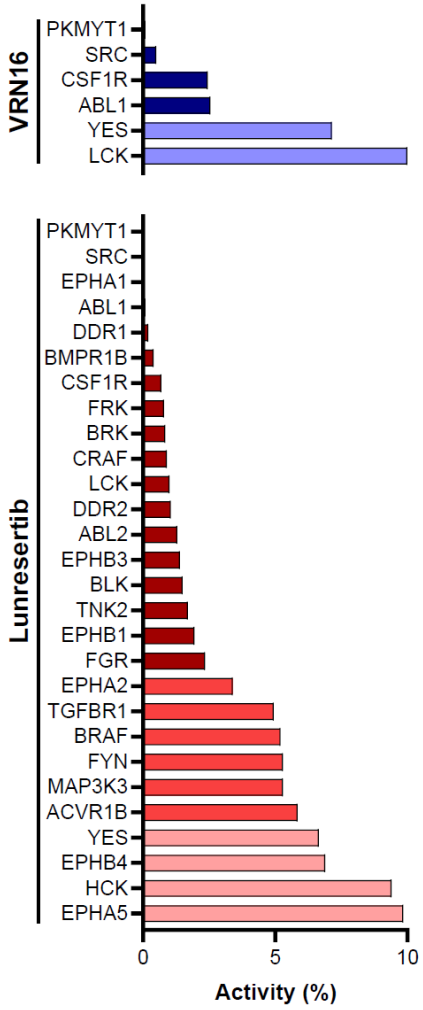


Lunresertib

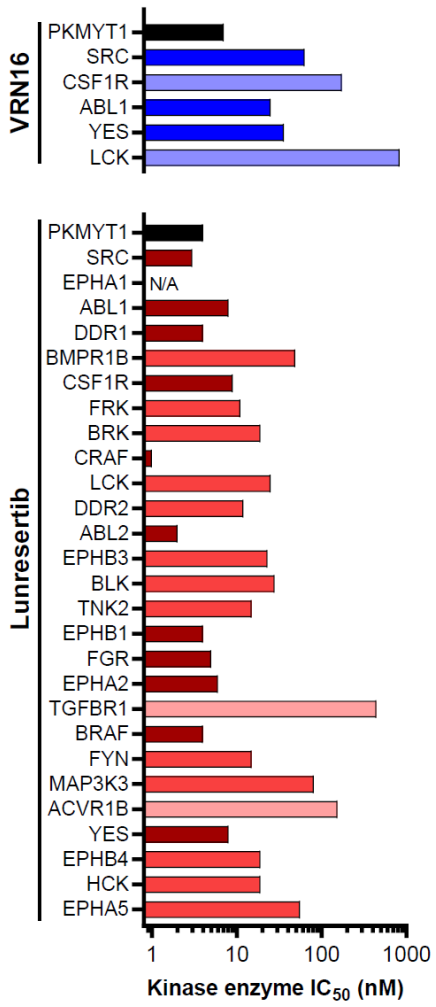
S(10) = 0.07



Kinase profile

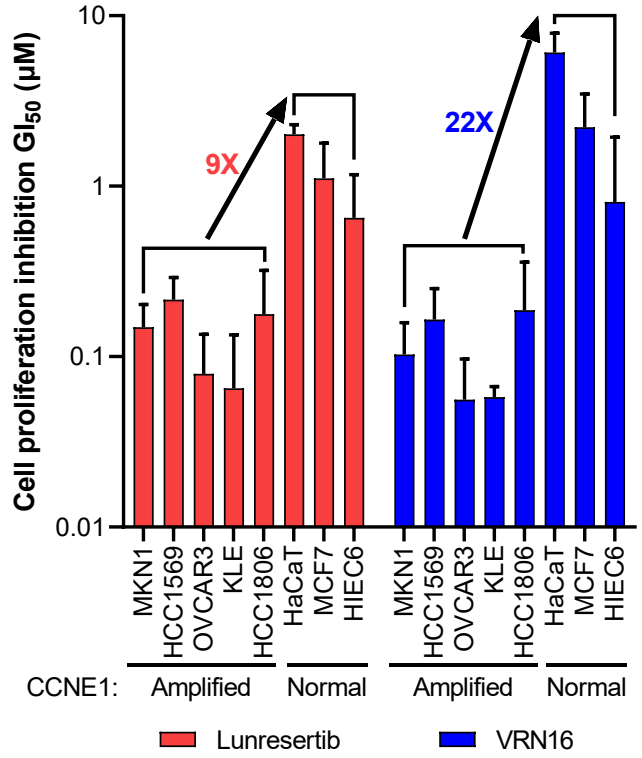


Enzyme catalytic inhibition



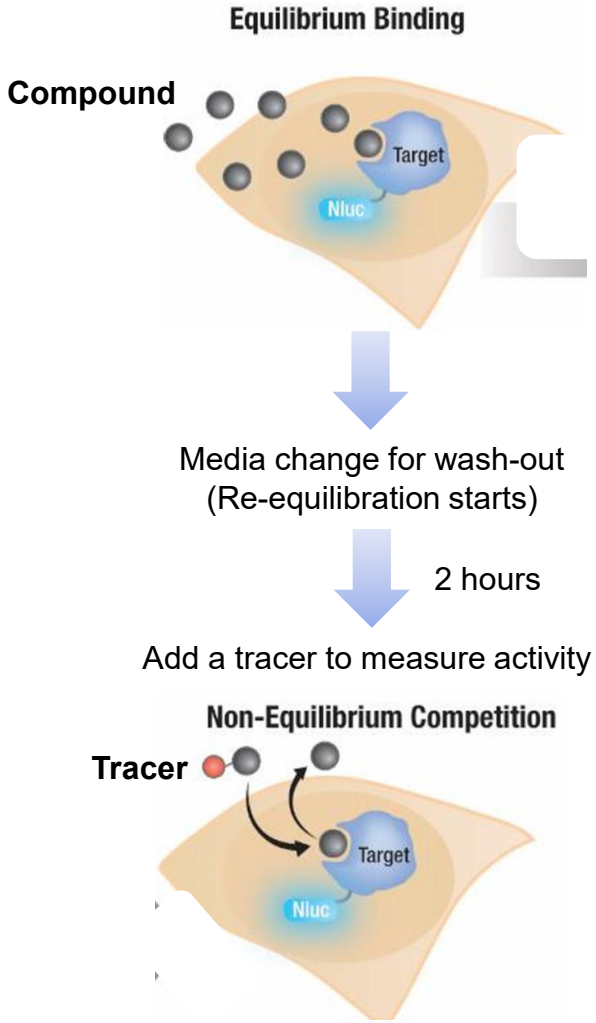
Kinase selectivity → CCNE1 target selectivity

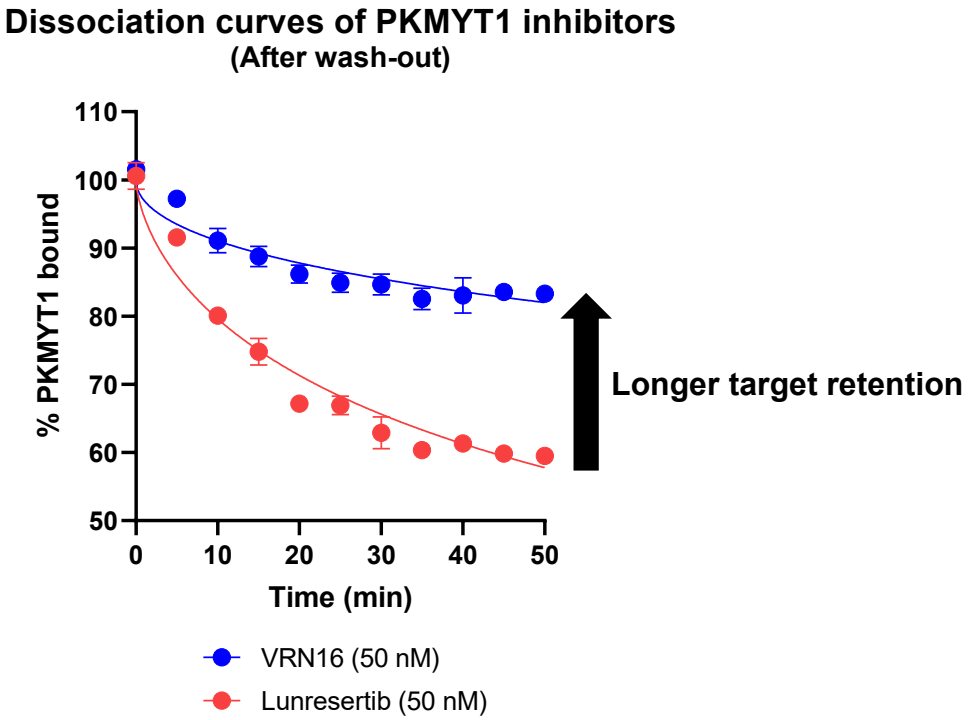
CCNE1 target selectivity



PKMYT1 NanoBRET: Longer target residence time

The PKMYT1 NanoBRET assay showed that VRN16 has a 13-fold longer target residence time than lunresertib.





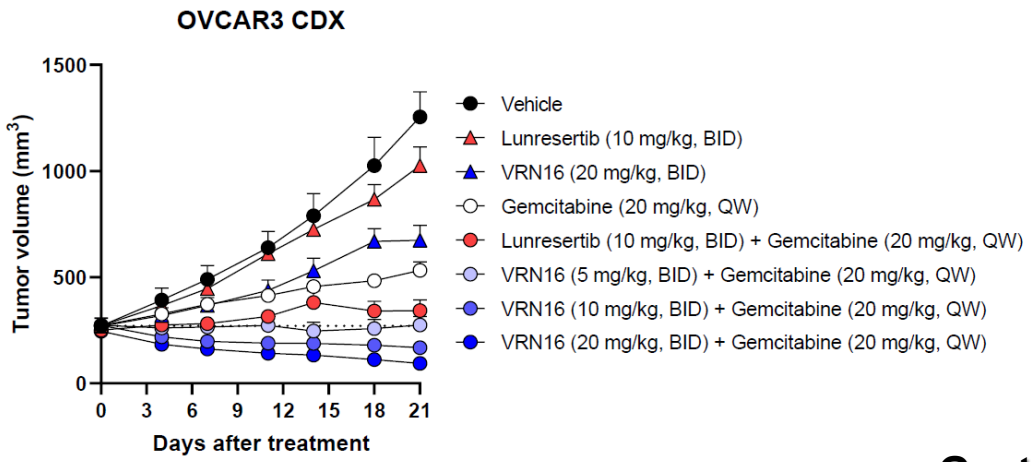
	$t_{1/2}$ (min)
VRN16	1,050
Lunresertib	81

Superior efficacy in CCNE1-amplified tumors

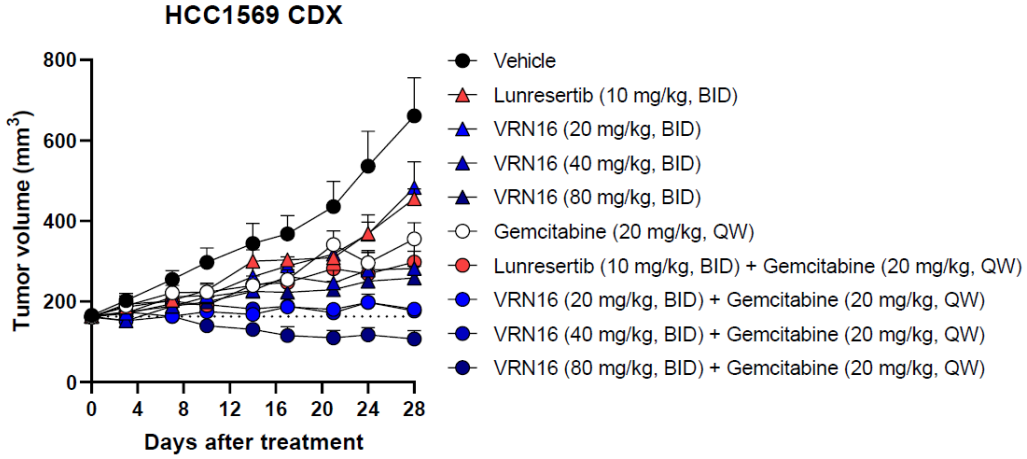
VRN16 showed superior efficacy compared to lunresertib in CCNE1-amplified ovarian, breast, and gastric cancer xenograft models.

Greater body weight loss was observed with lunresertib, demonstrating the superior tolerability of VRN16.

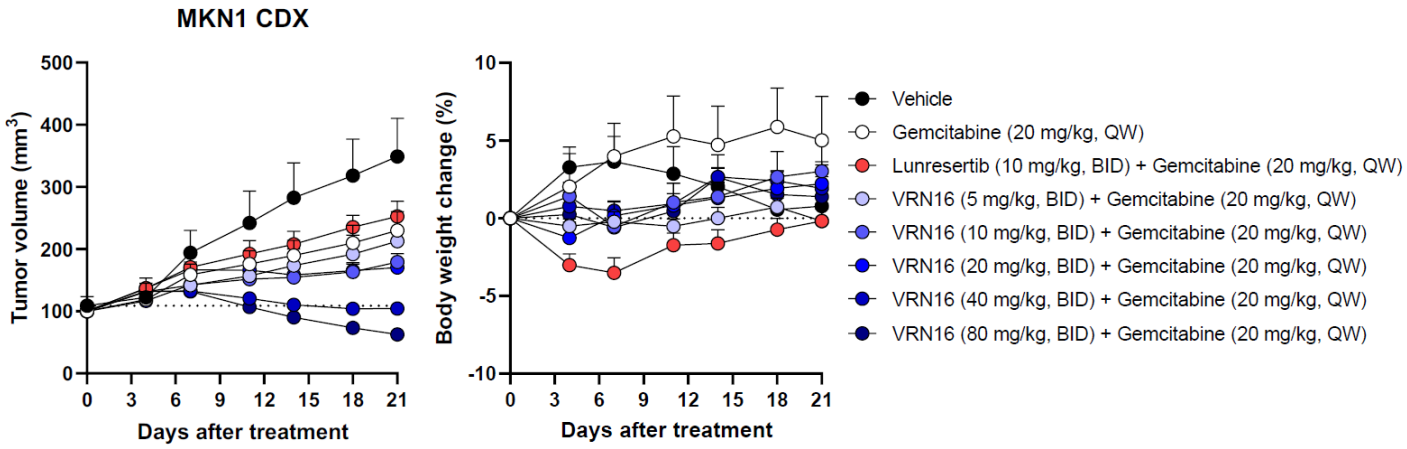
Ovarian cancer



Breast cancer



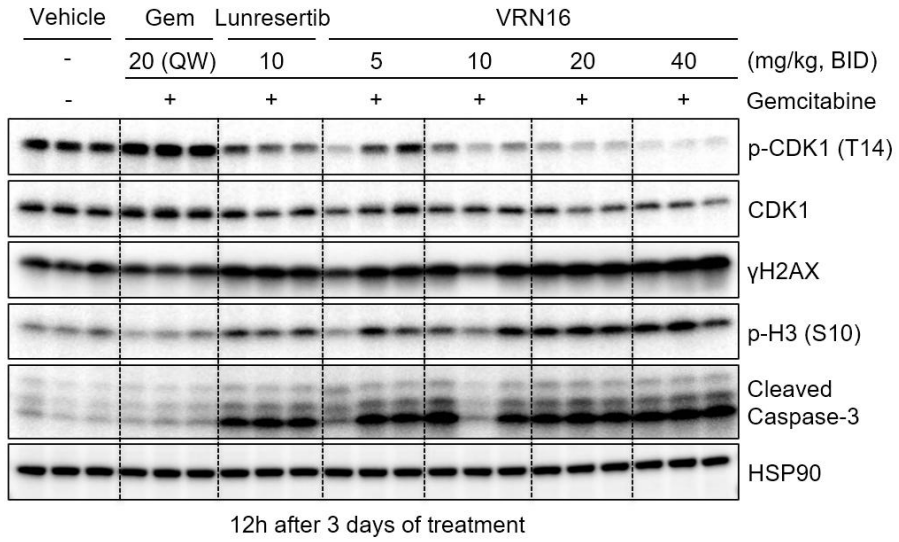
Gastric cancer



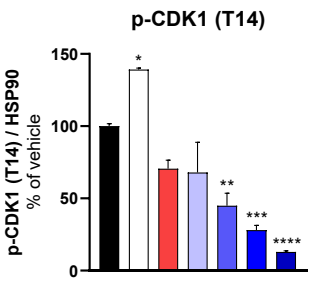
Superior target engagement and strong PK-PD correlation

VRN16 showed superior target engagement compared to lunresertib, with a well-correlated PK-PD relationship.

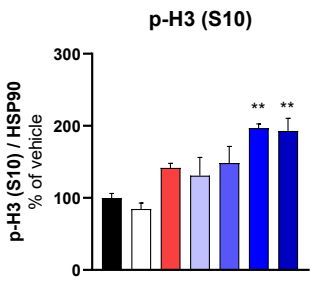
PD analysis (Target engagement)



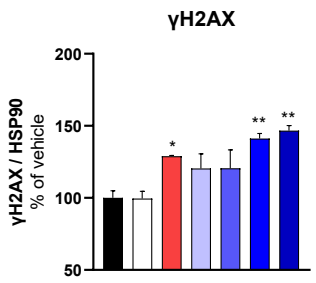
Target engagement



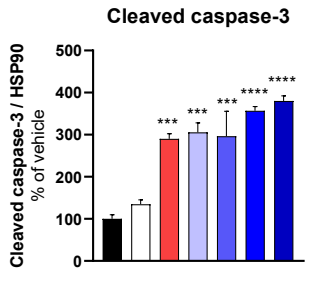
Mitotic population



DNA damage

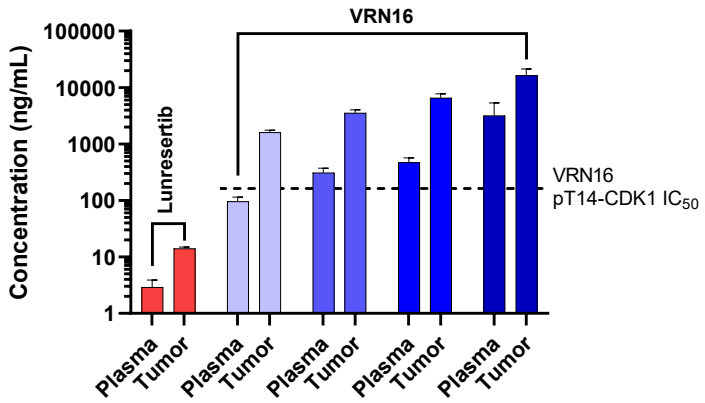


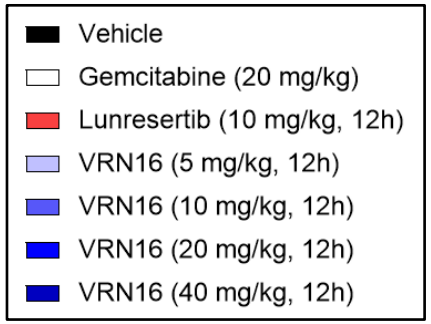
Cell apoptosis



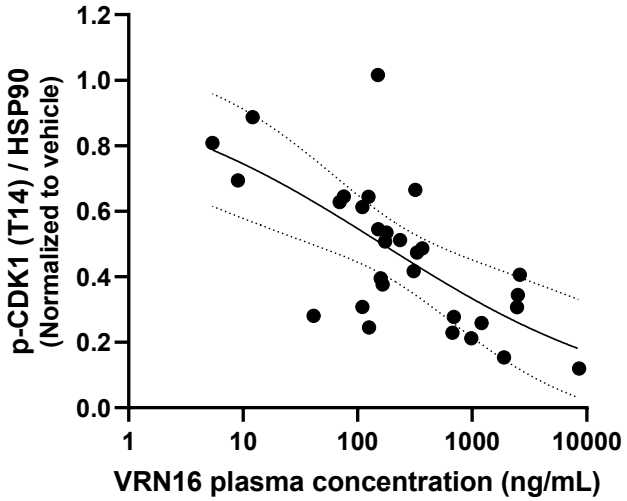
PK analysis (Ctrough)

PK (12h)





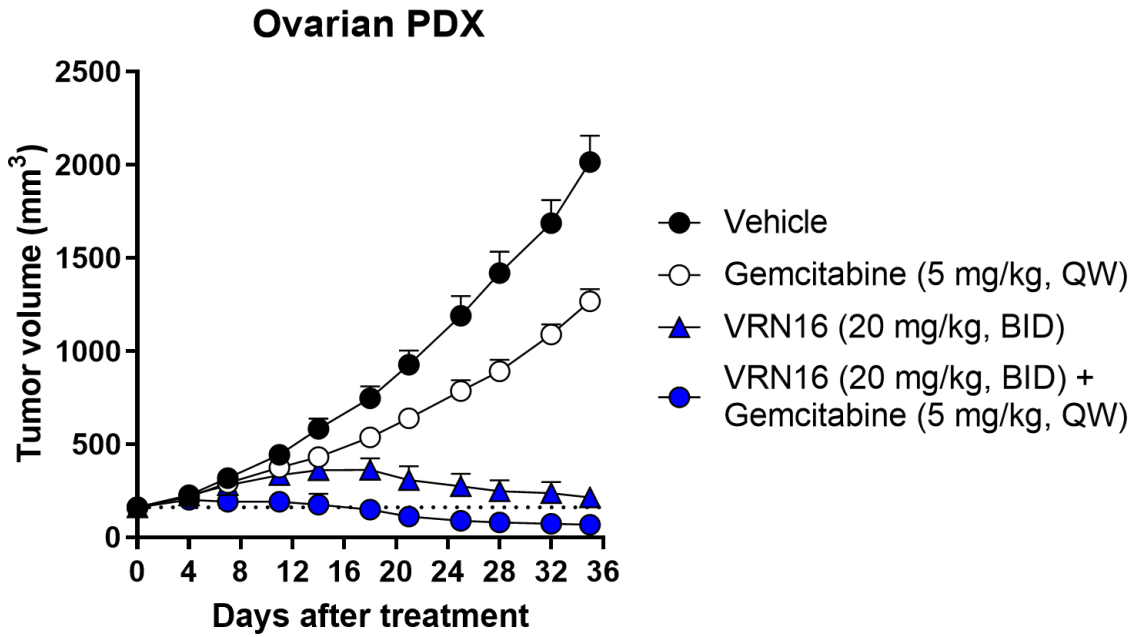
PK-PD correlation

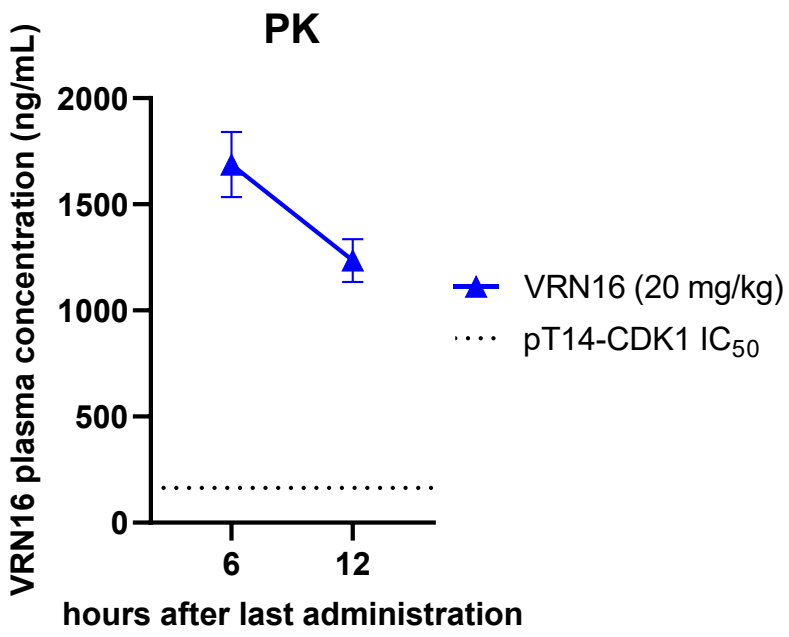


Tumor regression efficacy in a Cyclin E1-overexpressing ovarian PDX model

In a Cyclin E1-overexpressing ovarian PDX model, VRN16 monotherapy showed efficacy close to stable disease (SD), while its combination with gemcitabine resulted in efficacy approaching complete response (CR).

The plasma exposure of VRN16 remained well above the phospho-CDK1 IC_{50} for up to 12 hours.



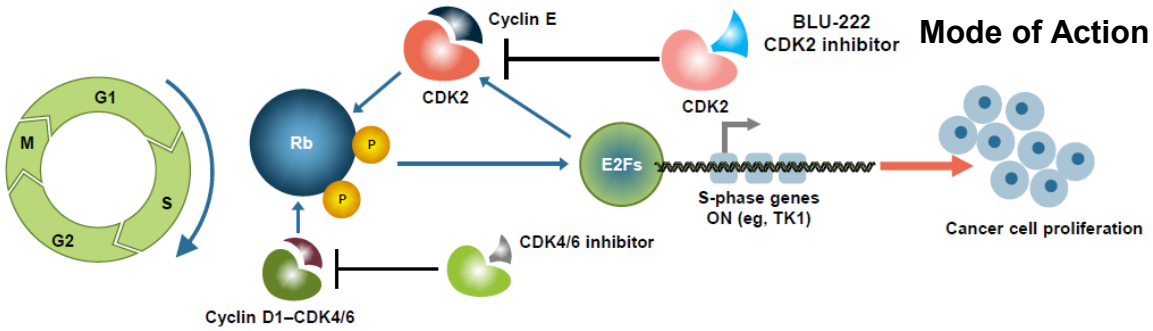


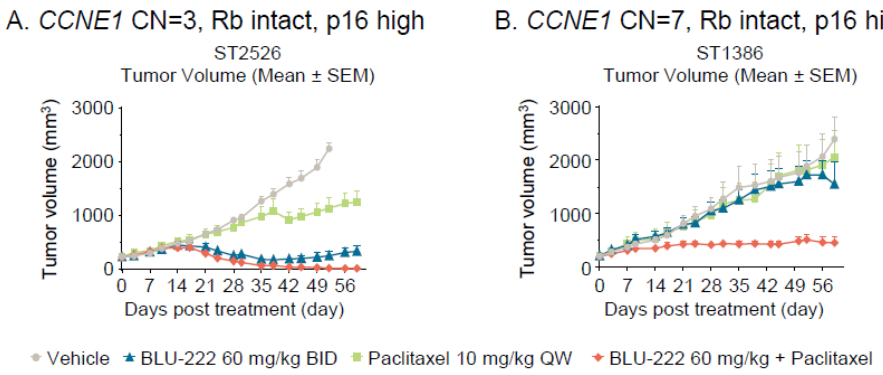
Superior efficacy compared to CDK2 inhibitor in the OVCAR3 CDX model

CDK2 inhibitors are currently undergoing clinical trials with CCNE1 amplification as a biomarker.

In CCNE1-amplified OVCAR3 CDX model, VRN16 combination with gemcitabine showed greater efficacy compared to CDK2 inhibitor combination with paclitaxel or carboplatin.

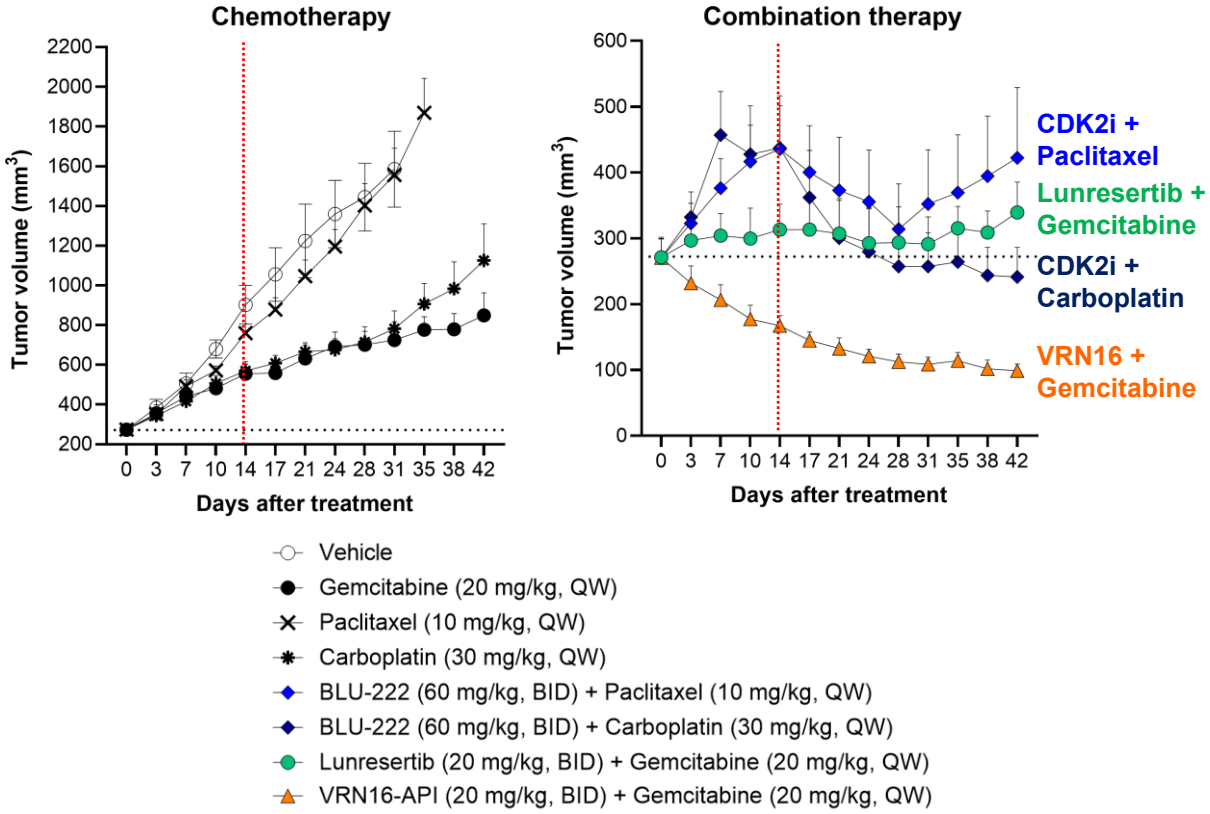
CDK2 inhibitor





Abstract #1959, 2024 AACR annual meeting

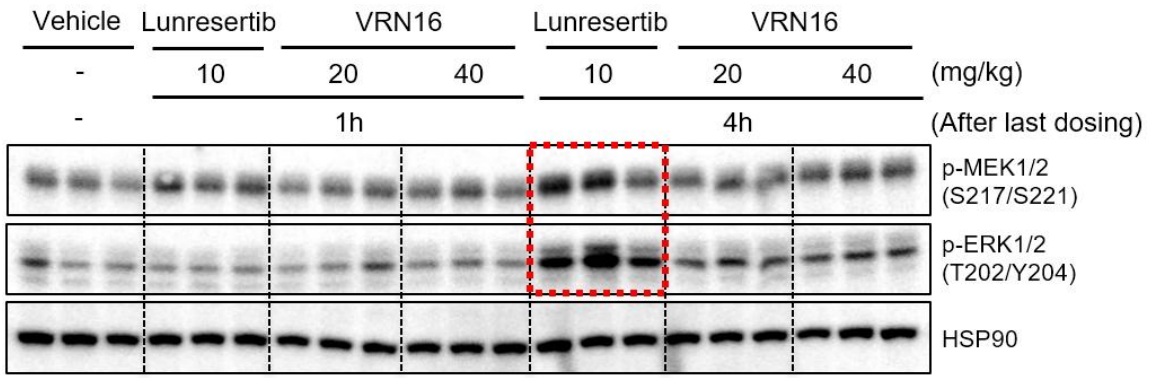
Comparative efficacy with CDK2 inhibitor

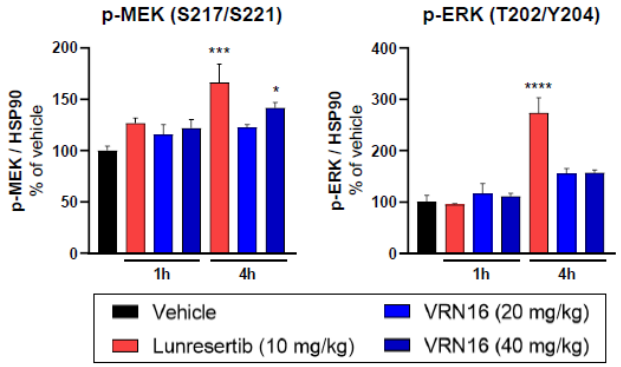


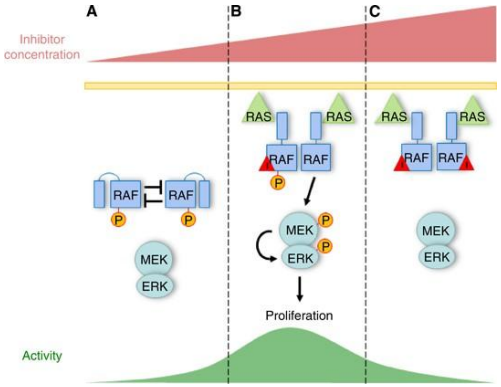
Off-targets related adverse events

Lunresertib inhibits RAF and causes paradoxical MAPK activation and related side effects, whereas VRN16 does not.

Paradoxical MAPK activation by inhibiting RAF





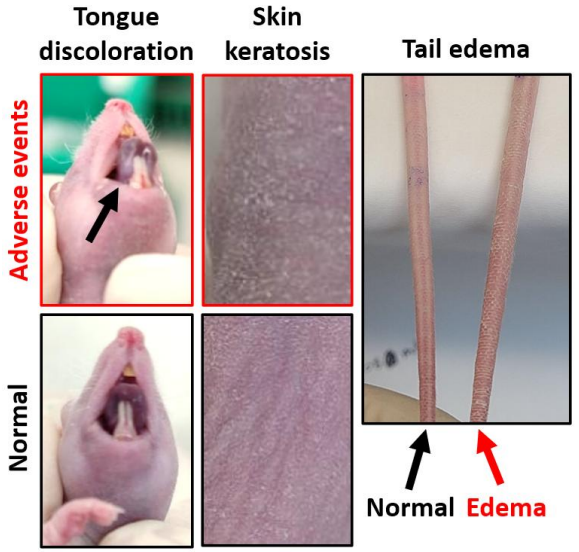


Br J Cancer 111, 640–645 (2014). https://doi.org/10.1038/bjc.2014.139

Lunresertib + Camonsertib (ATRi) safety profile

(Repare Corporate Presentation Q3 2024)

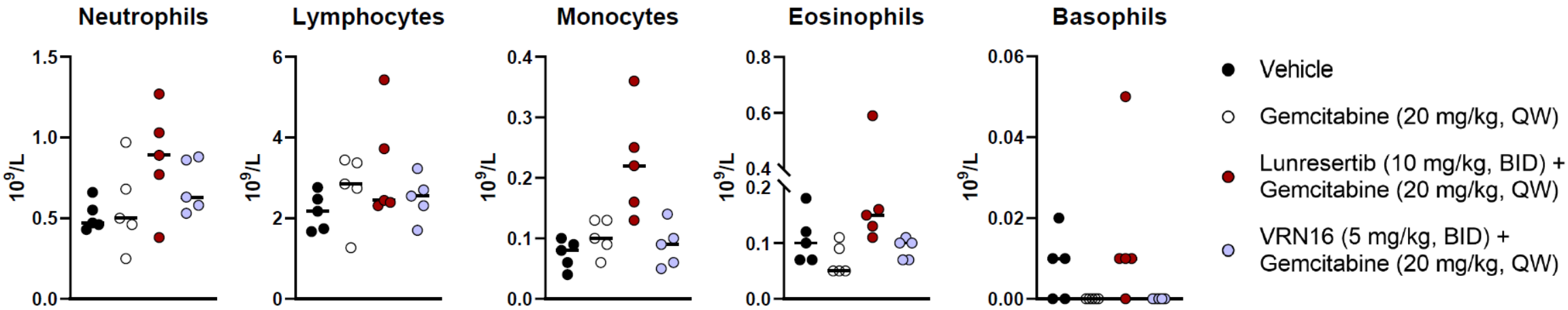
TRAEs in ≥10% of patients, n (%)	All Grades	Gr3	Gr4
Nausea/Vomiting	34 (52.3)	0	0
Rash[a]	26 (40.0)	1 (1.5)	0
Fatigue	18 (27.7)	1 (1.5)	0
Stomatitis	18 (27.7)	4 (6.2)	0
Decreased appetite	13 (20.0)	0	0
Diarrhea	10 (15.4)	0	0
Headache	7 (10.8)	0	0
Constipation	5 (7.7)	0	0



No hematologic toxicity in gemcitabine combination

In a clinical trial with gemcitabine combination, lunresertib exhibited hematologic toxicity (EORTC-NCI-AACR symposium 2024).

In ICR mice, lunresertib showed hematologic toxicity under gemcitabine combination treatment, whereas VRN16 did not.



VRN16: A superior PKMYT1 selective inhibitor that outperforms lunresertib

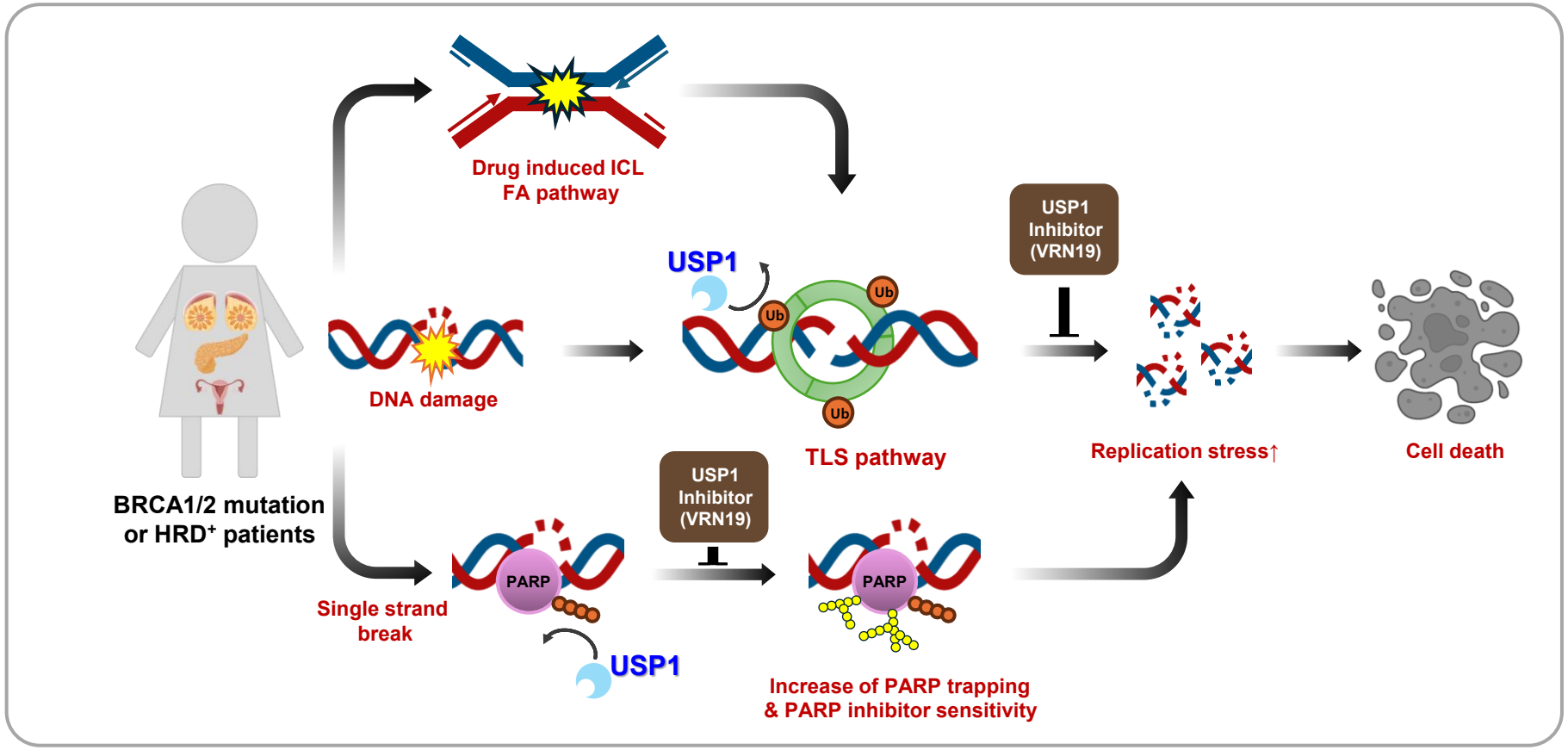
VRN16 outperforms lunresertib in every aspect, including both efficacy and toxicity.

			VORONOI VRN16	REPARE THERAPEUTICS Lunresertib (RP-6306)
Efficacy	*In vitro*	CCNE1 target selectivity	22X	9X
		PKMYT1 target residence time ($t_{1/2}$, min)	1,050 (13-fold longer)	81
	In vivo	CCNE1-amplified cancer CDX models (Ovarian, breast, and gastric cancers)	+ + +	+
		Cyclin E1-overexpressed ovarian cancer PDX model	+ + +	N/A
		Comparison to CDK2 inhibitor	+ + +	+
Target engagement	*In vivo*	PD: pT14-CDK1 inhibition	+ + +	+
		PK-PD correlation	Strong PK-PD correlation	N/A
Off-target	Kinase selectivity	S(10)	0.01	0.07 (7-fold more off-targets)
	RAF inhibition	BRAF kinase inhibition (IC_{50}, μM)	0.813	0.029 (28-fold more potent)
Toxicity	Off-targets related	Stomatitis / Keratosis / Edema	+	+ + +
	Hematologic	Abnormal numbers of WBC components	-	+ + +

VRN19, USP1 inhibitor w/ excellent safety profile

USP1, ubiquitin-specific protease 1, synthetic lethal with BRCA mutants

USP1 (ubiquitin-specific protease 1) regulates DNA repair via the FA complex and TLS pathway, making its inhibition a synthetic lethality target that sensitizes cancer cells to DNA-damaging therapies



Sci Adv. 2024 Nov 15;10(46):eadp6567.

Competitive Landscape

Generic name	Company	Line of treatment	Phase	Combination treatment	Status	Number of patients	Start ~ Completion date
KSQ-4279 (RG6614)	Roche	Second line	Phase I	Olaparib / Carboplatin	Recruiting	250	2021. 08 ~ 2027. 06
XL309 (ISM3091)	EXELIXIS	Second line	Phase I		Recruiting	377	2023. 08 ~ 2029. 08
SIM0501	Simcere	Second line	Phase I	Olaparib	Recruiting	176	2024. 03 ~ 2027. 12
HSK39775	Harsco	Second line	Phase I / Phase II		Recruiting	243	2024. 03 ~ 2028. 09

Generic name	Company	Line of treatment	Phase	Combination treatment	Status	Number of patients	Start ~ Completion date
Debio 0432 (FT-3171)	Debiopharm	Preclinical	IND enabling				
IMP13	Impact Therapeutics	Preclinical					
Protai-XX	Protai	Preclinical					
LAE120	Laekna Therapeutics	Preclinical	IND enabling				
AIG-USP1	AIGEN Sciences	Preclinical					
XZP-6924	Xuanzhu Biopharmaceutical	Preclinical	IND enabling				
VRTX531	VRise Therapeutics	Preclinical	IND enabling				
APL-2302	Asieris Pharmaceutical	Preclinical	IND enabling				

Limitation of current investigational USP1 inhibitors in clinic

Roche

First-in-human trial of the oral first-in-class Ubiquitin Specific Peptidase 1 (USP1) inhibitor RO7623066 (KSQ-4279), given as single agent and in combination with olaparib or carboplatin in patients with advanced solid tumors, enriched for deleterious homologous recombination repair (HRR) mutations

2024 ASCO Annual Meeting

Adverse Event *All TEAEs in > 20% for single agent and in > 30% for combination*	Single agent (n=42)		RO7623066 + OLA* (n=15)		RO7623066 + CARBO (n=13)	
	All Grades	Grade ≥3	All Grades	Grade ≥3	All Grades	Grade ≥3
Anemia	35.7%	4.8%	86.7%	73.3%	69.2%	30.8%
Blood creatinine increased	33.3%	0	33.3%	0	30.8%	0
Hyponatremia	28.6%	11.9%	-	-	61.5%	7.7%
GGT increased	26.2%	9.5%	40.0%	13.3%	38.5%	15.4%

*** Anemia adverse events were reversible and manageable**

Hematotoxicity + Hepatotoxicity

TANGO therapeutics

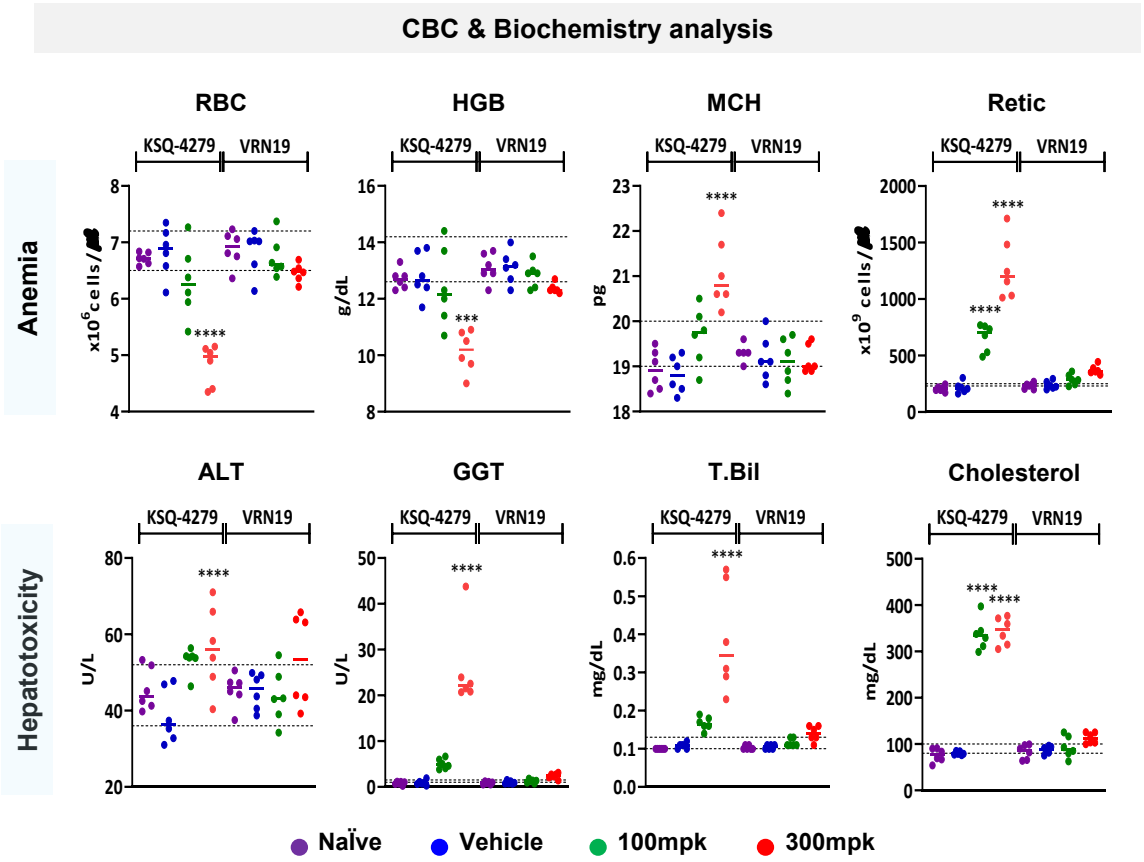
Tango drops USP1 inhibitor over liver toxicity in patients during phase 1 trial

May 23, 2024

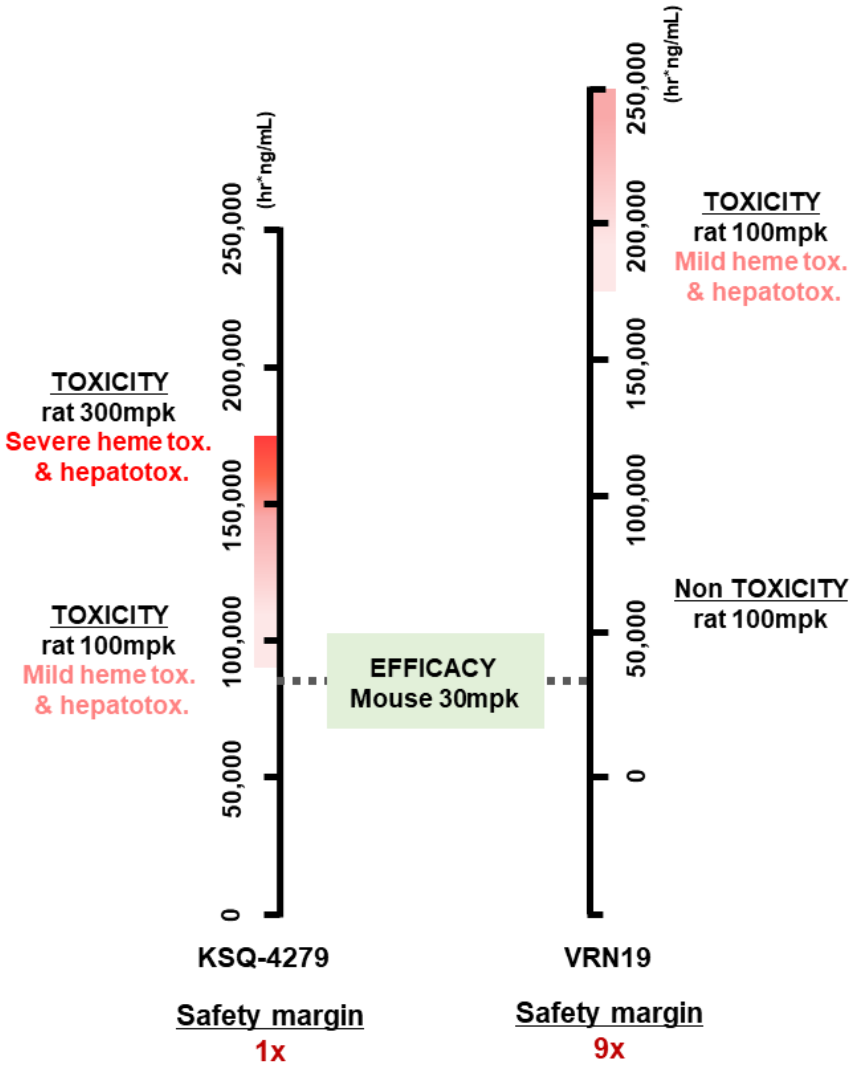
Hepatotoxicity

>> USP1 on-target toxicity or off-target toxicity?

VRN19, a novel USP1 inhibitor without Anemia and Hepatoxicity

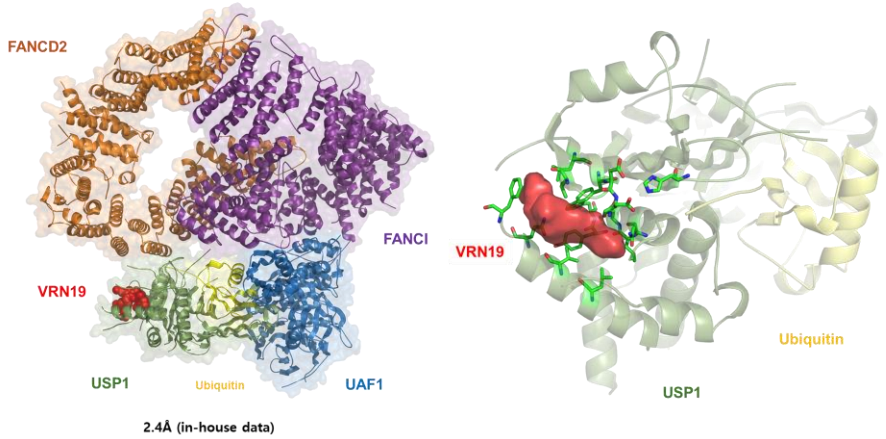


Values are in mean ± standard error of the mean; One-way ANOVA analysis test
(* : P ≤ 0.05; ** : P ≤ 0.01; *** : P ≤ 0.001; **** : P ≤ 0.0001)

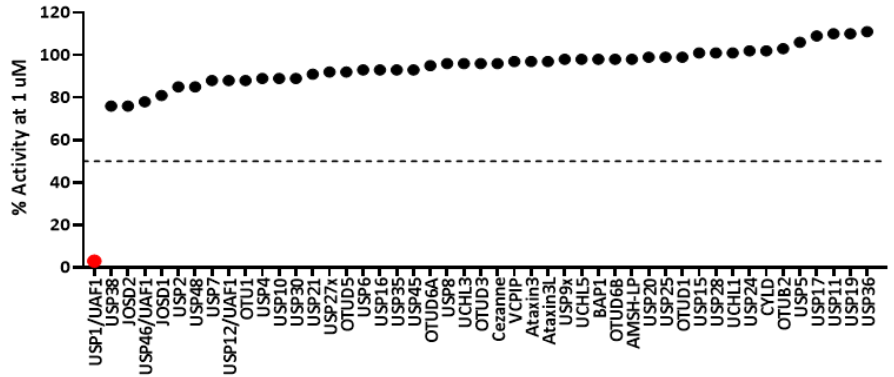


VRN19, high selectivity and potency

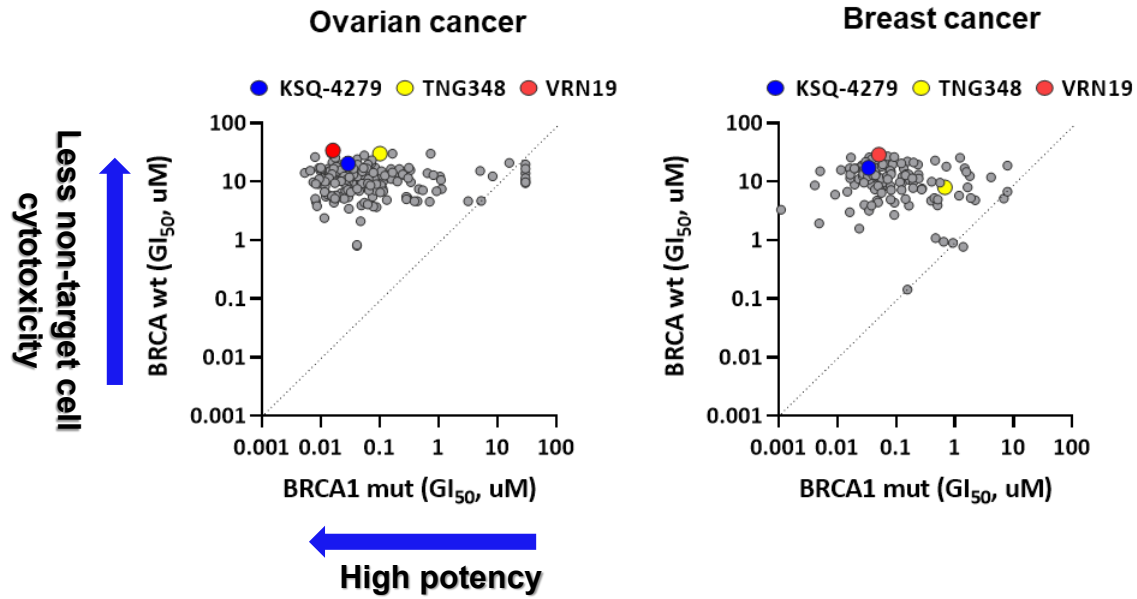
Selective binding to USP1-USA complex



DUBprofiler™ assay

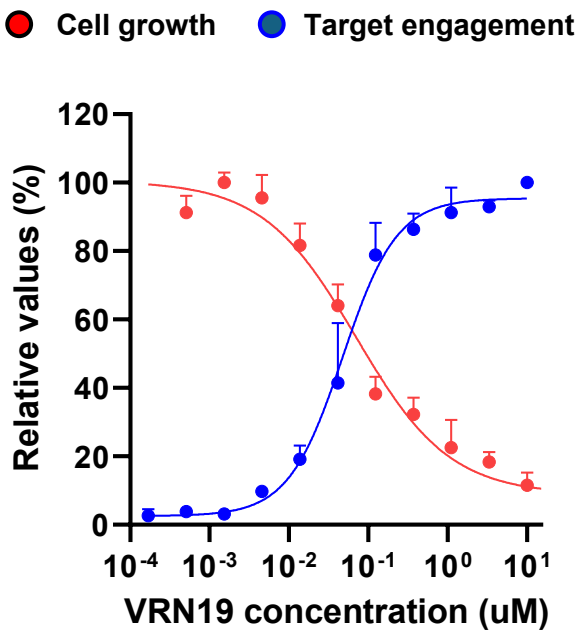


High potency against target cancer cells
Less non-target cell cytotoxicity

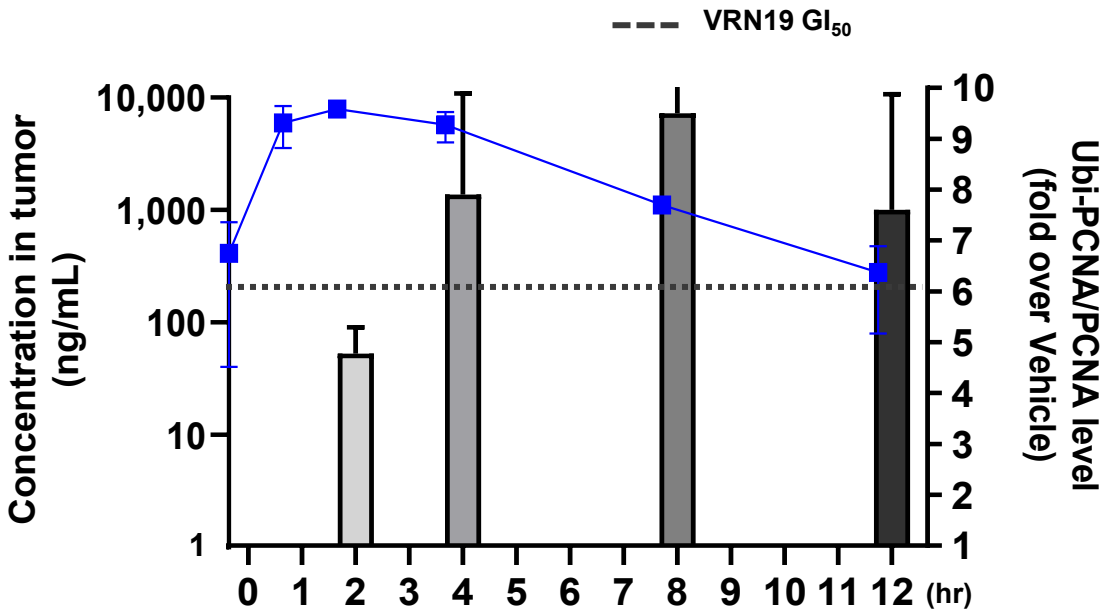


VRN19, Target engagement

In vitro cell viability & target engagement



PK/PD

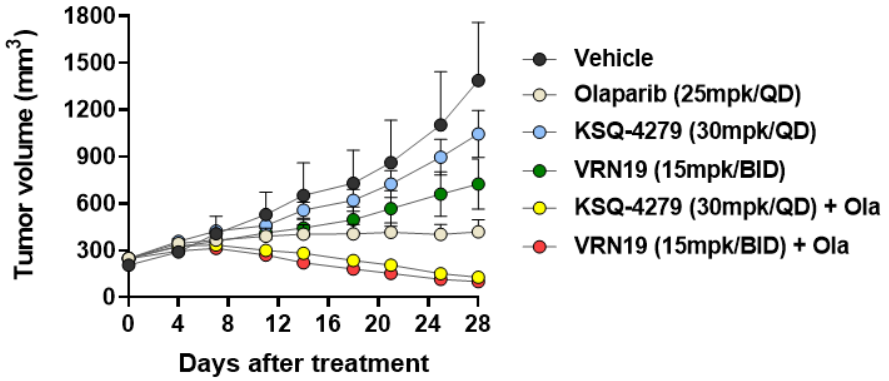


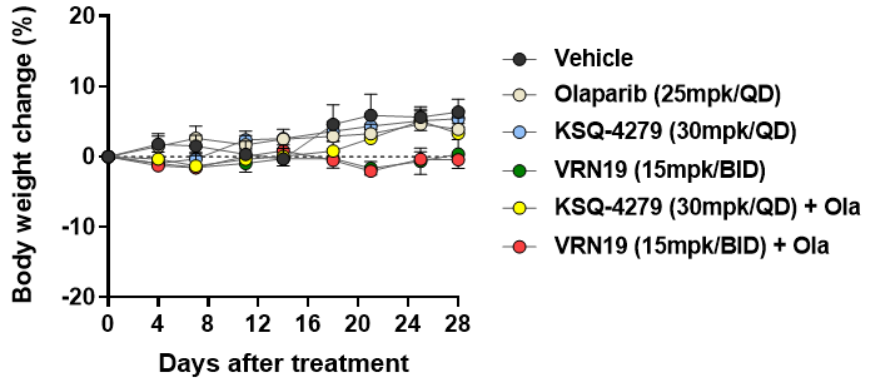
30 mg/kg (PO), BID for 7 days in the MDA-MB-436 model

VRN19, in vivo efficacy against BRCAm or HRD solid tumor

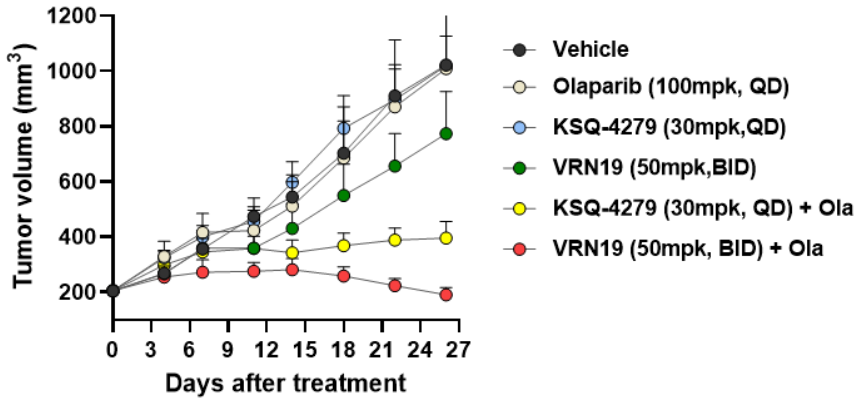
Superior synergistic efficacy with PARP Inhibitor or Carboplatin in CDX Models

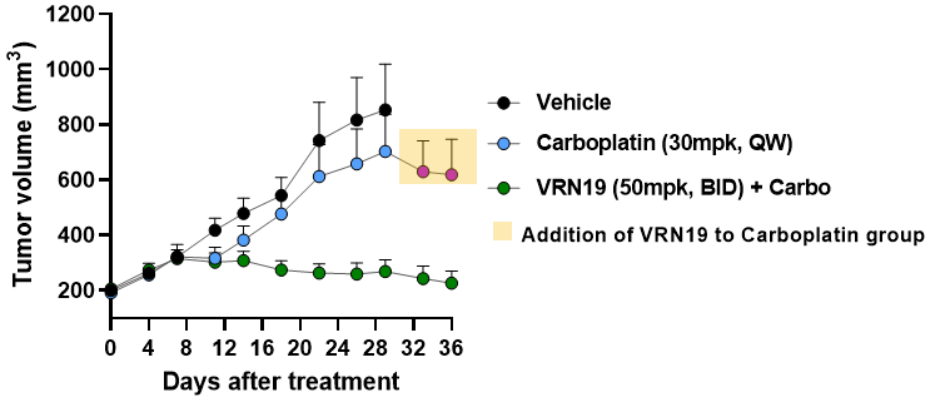
MDA-MB-436 (BRCA1 mut / HRD) SC xenograft model



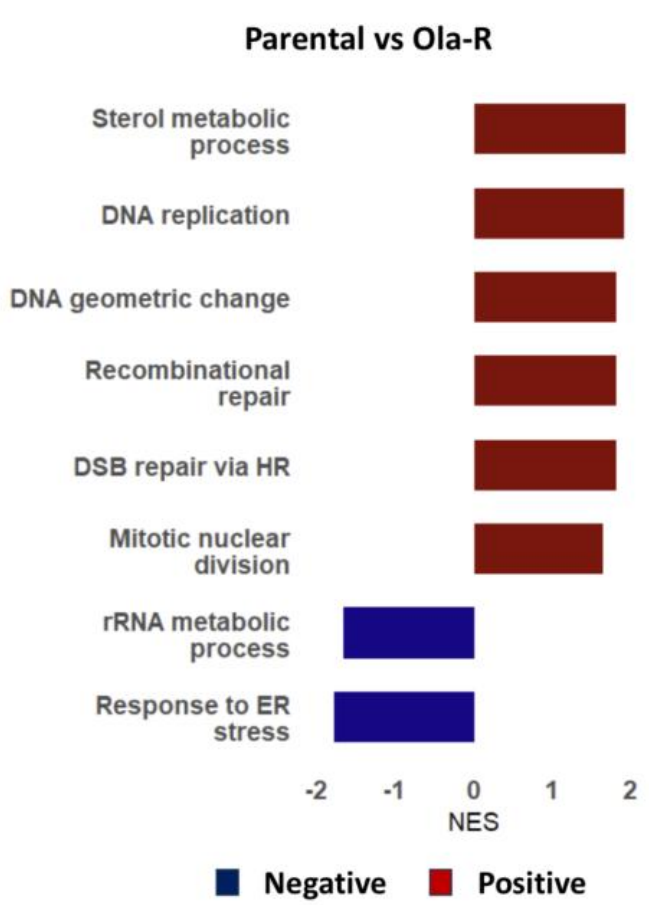


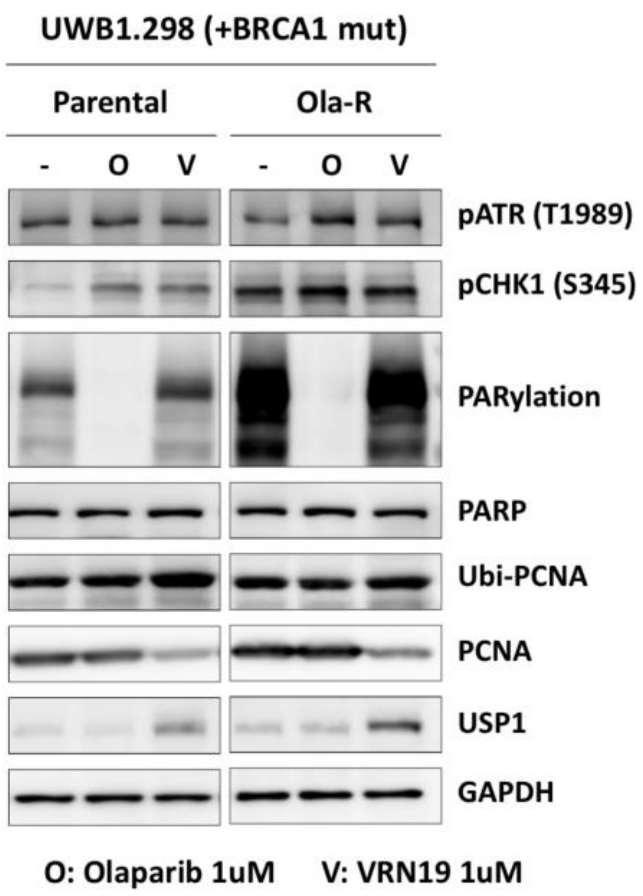
OVCAR3 (BRCA wt / HRD) SC xenograft model

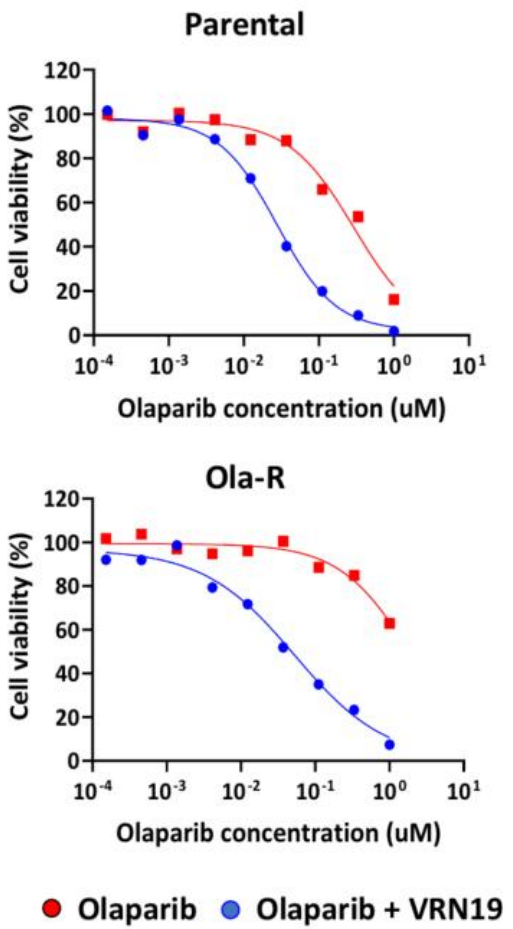


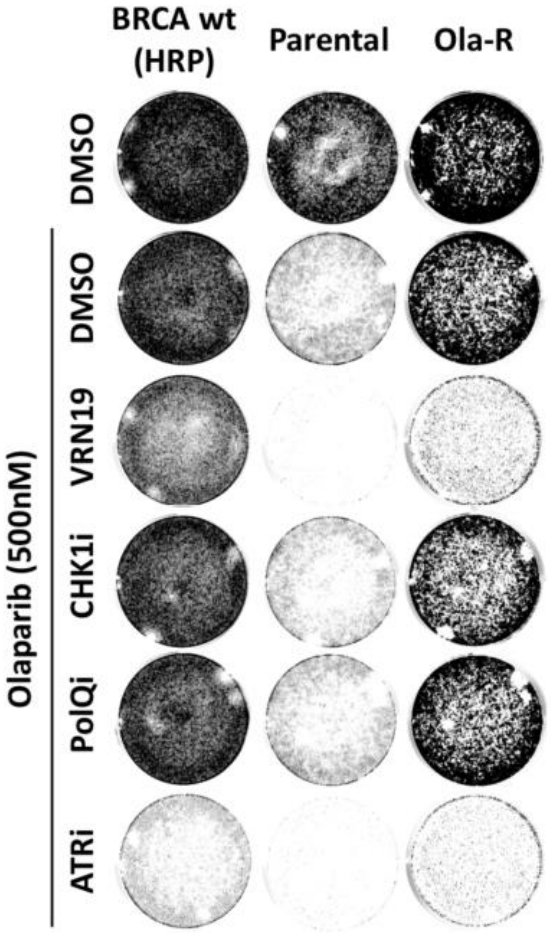


VRN19, Overcoming Acquired PARP Inhibitor Resistance





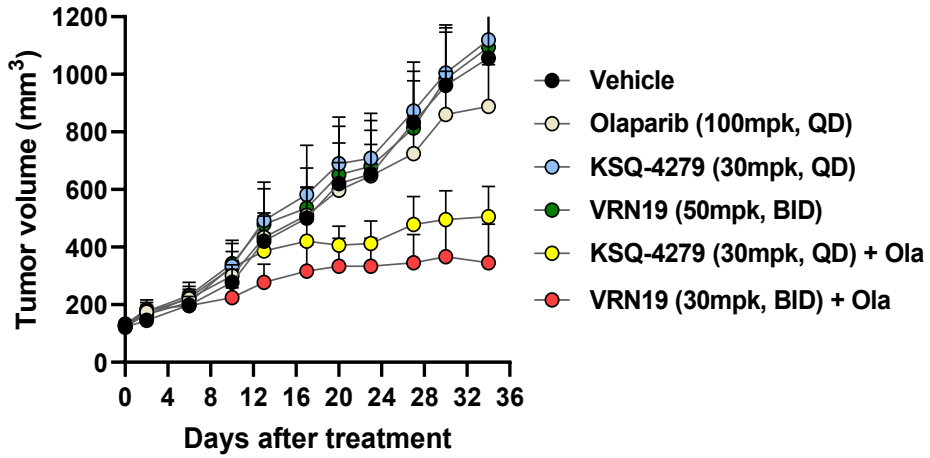


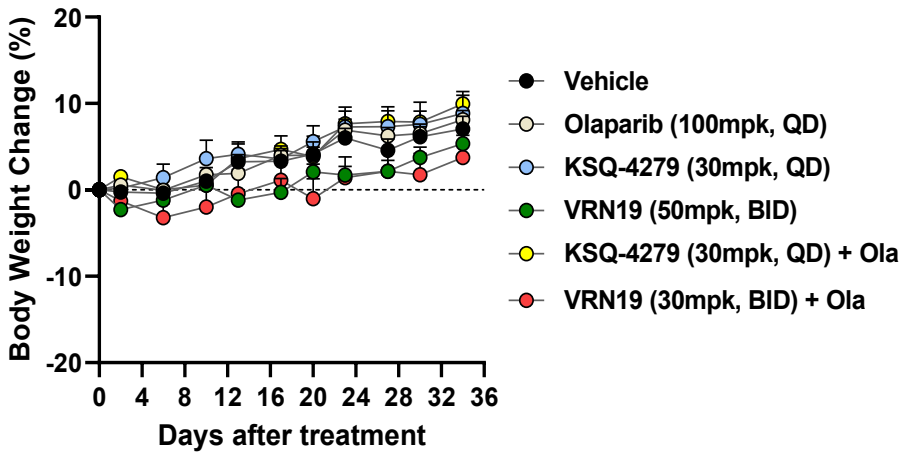


VRN19, in vivo efficacy against Olaparib resistance PDX

Combination of VRN19 and Olaparib overcomes resistance in Olaparib-resistant PDX model

Olaparib resistance TNBC PDX model





CRO: Xentech

VRN19, best-in-class USP1 inhibitor

- ✓ *VRN19 is a highly potent and selective allosteric USP1 inhibitor.*
- ✓ *VRN19 showed anti-tumor efficacy in CDX or PDX model and superior synergistic efficacy with Olaparib.*
- ✓ *VRN19 can overcome acquired PARP inhibitor resistance.*
- ✓ *VRN19 is a promising novel USP1 inhibitor with favorable safety and improved therapeutic outcomes.*
- ✓ *VRN19 is advancing under IND enabling*

Creating Novel Therapeutics
By People With Excellent Expertise
In Drug Design

Thank You