Creating Novel Therapeutics
For patients with Excellent Experts
In Drug Design

IR BOOK

July 2026

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VORONOI. Pipeline

Drug	Company	Indication	Target	Clinical Phase
				Pre-clinical / Phase 1/2 / Pivotal/Phase 3
VRN11	VORONOI	Non-small cell lung cancer (NSCLC)	1L, EGFR common mutation (treatment-naïve)	
			1L ,EGFR uncommon mutation (treatment-naïve)	
			2L EGFR-mutant with unknown resistance mechanisms (combination therapy)	
			2L, EGFR C797S-mutant	
VRN10	VORONOI	Solid Tumor	HER2-altered solid tumors	
VRN07 (Enozertinib)	ORIC	Non-small cell lung cancer (NSCLC)	1L, EGFR exon20 ins (monotherapy)	
			1L, EGFR exon20 ins (with chemotherapy)	
			1L, EGFR exon20 ins (with SC amivantamab) Janssen	
			1L EGFR atypical	
VRN04	AMVIA	Auto-immune disease	RIPK1	
VRN13	VORONOI	Pulmonary arterial hypertension (PAH)	PDGFR	
VRN16	VORONOI	Solid Tumor	PKMYT1	
VRN19	VORONOI	Solid Tumor	USP1	

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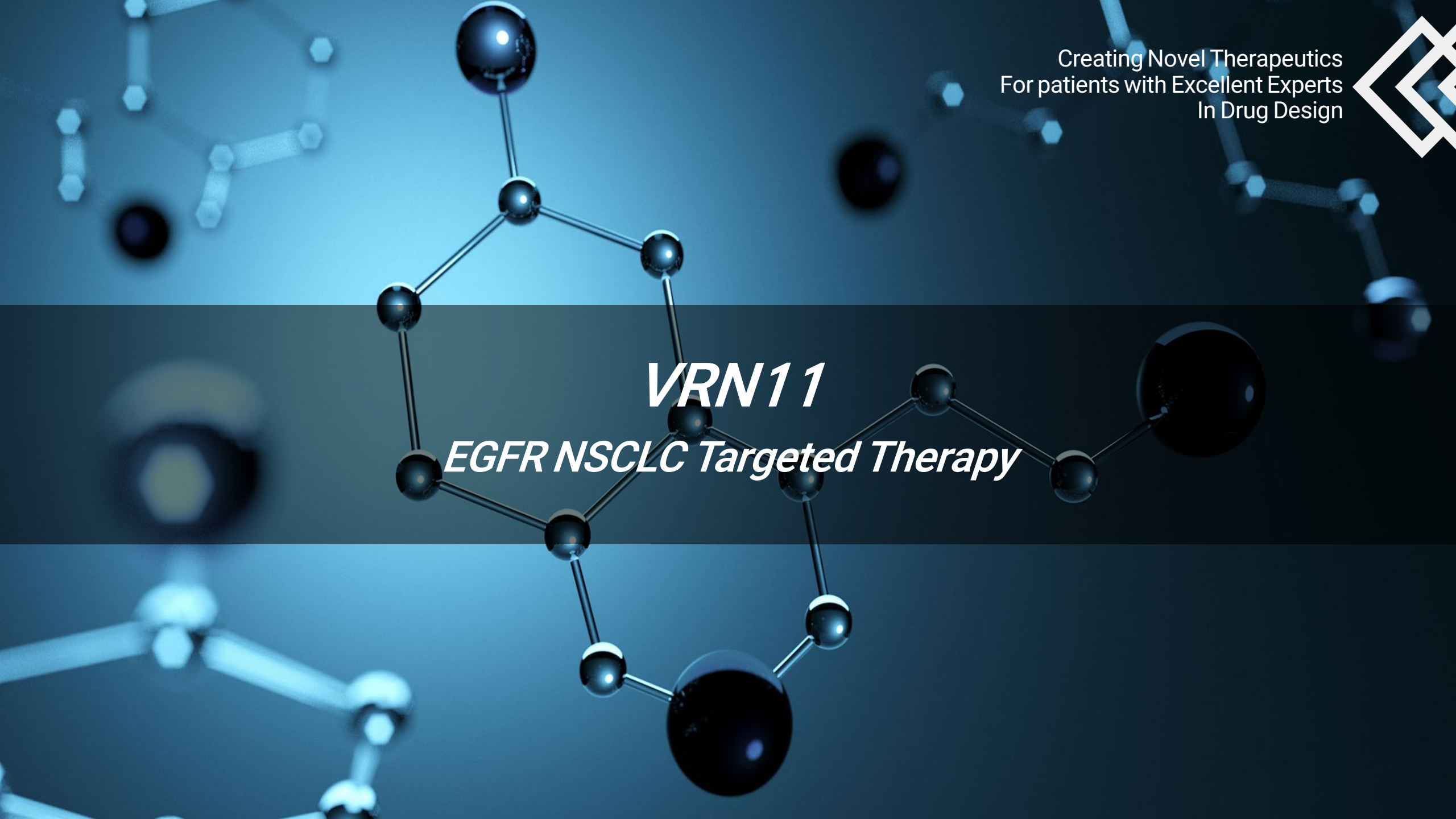
CONTENT



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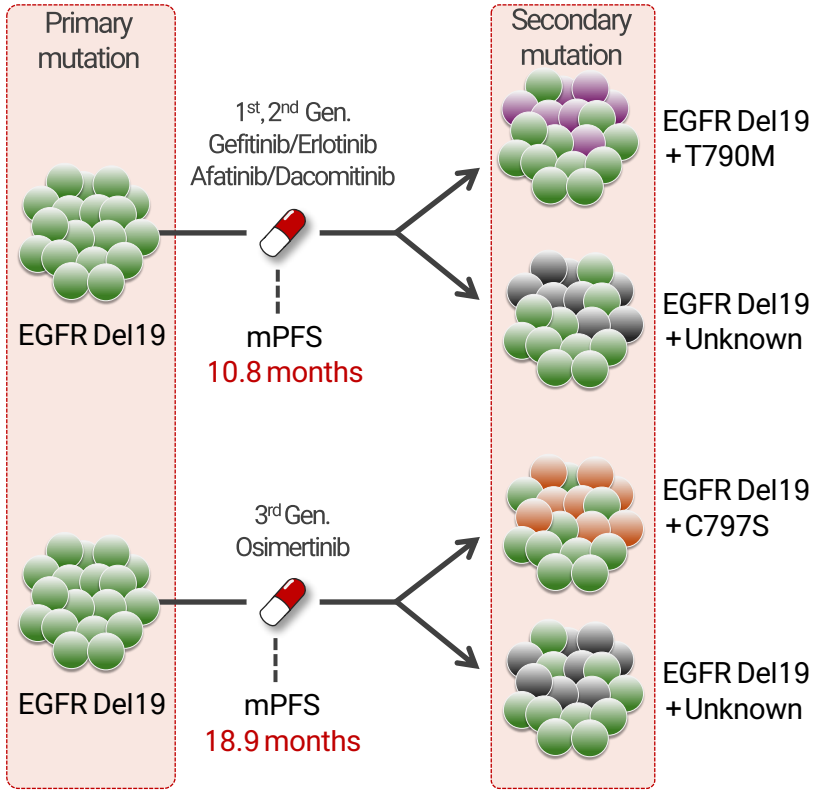
VRN11

EGFR NSCLC Targeted Therapy

EGFR Non-small cell lung cancer(NSCLC) Landscape

Phase 1a study ongoing in patients with EGFR-mutated NSCLC refractory to standard therapies, including EGFR TKIs
Based on phase1a data, phase1b/2 study ongoing in EGFR treatment-naïve patients for first-line treatment

[EGFR NSCLC] Landscape(EGFR Common mutations)[1,2]

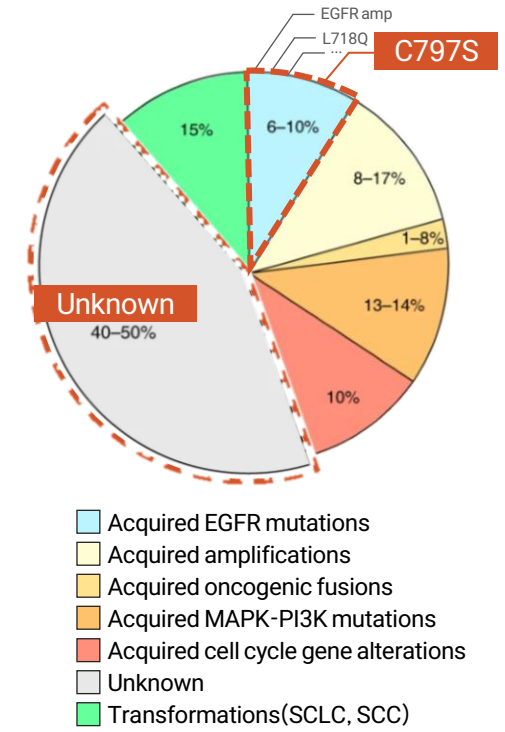


Target Market for VRN11

- The most common EGFR mutations are exon 19 deletion and the L858R point mutation, which account for approximately 80–90% of all EGFR mutations
  - Approximately 30–50% of resistance mechanisms to osimertinib remain unknown
  - EGFR C797S mutation accounts for ~10% of osimertinib resistance
- Currently, there are no approved therapies for secondary EGFR mutations(EGFR C797S)
- Based on Phase 1a data in patients with secondary EGFR mutations (~60% cases), clinical development in patients with primary EGFR mutations is planned.
- Unmet medical need for drugs with brain penetration
- Phase 1a study ongoing in heavily pretreated patients with EGFR-mutated NSCLC; Phase 1b/2 study ongoing in EGFR treatment-naïve patients

EGFR Common mutations (del19 or L858R) · EGFR T790M · EGFR Acquired mutations (unknown) · EGFR C797S

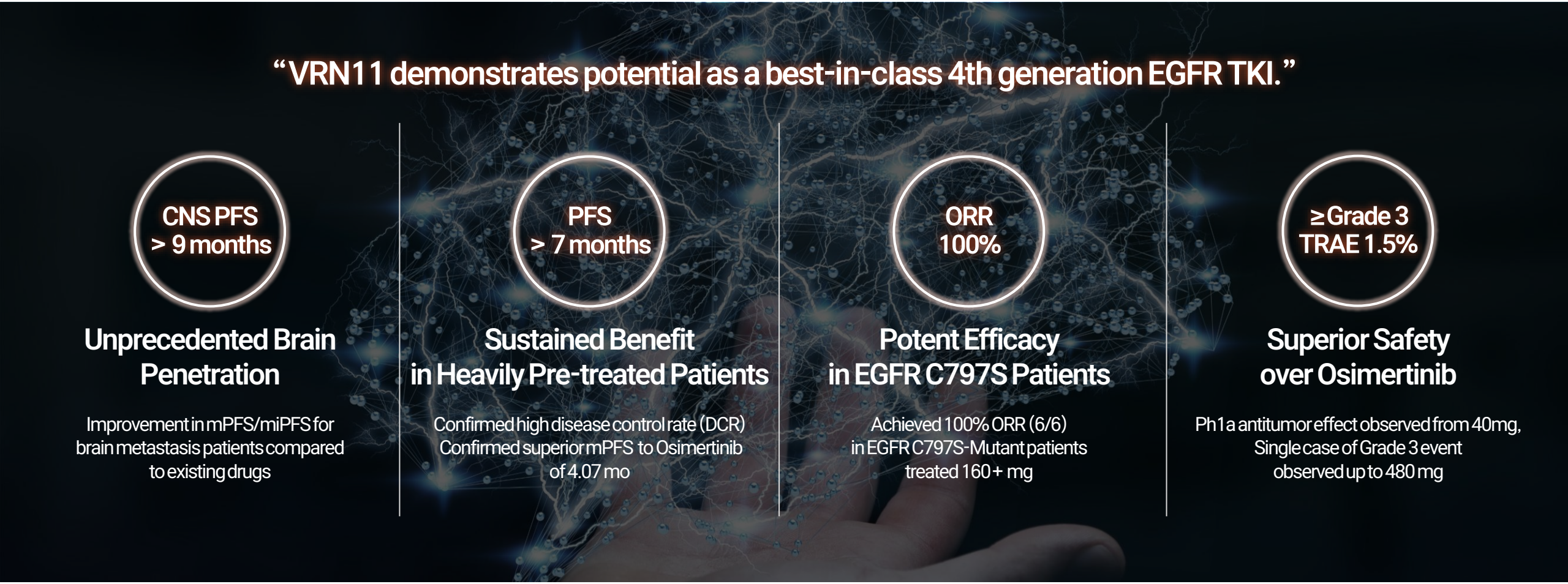
Resistance mechanisms to Osimertinib[3]



EGFR, epidermal growth factor receptor; NSCLC, Non-small cell lung cancer; TKI, Tyrosine kinase inhibitor
Source: : [1]Makoto Maemondo, et al. N Engl J Med 2010;362:2380-8, [2]J.-C. Soria, et al. N Engl J Med 2018;378:113-25 [3]Leonetti, A. et al. Br J Cancer 121, 725–737 (2019)

Clinical Update on VRN11: Game Changer in EGFR-Mutant NSCLC

Anticipated Addressing of Broad EGFR-Mutant Lung Cancer Unmet Needs, Driven by Meaningful Phase 1 Efficacy, Safety, and BBB Penetration Data



PFS, Progression-Free survival; iPFS, Intracranial Progression-Free survival ;ORR, objective response rate; TRAE, treatment-related adverse event
Data cut-off: March 12, 2026

VRN11

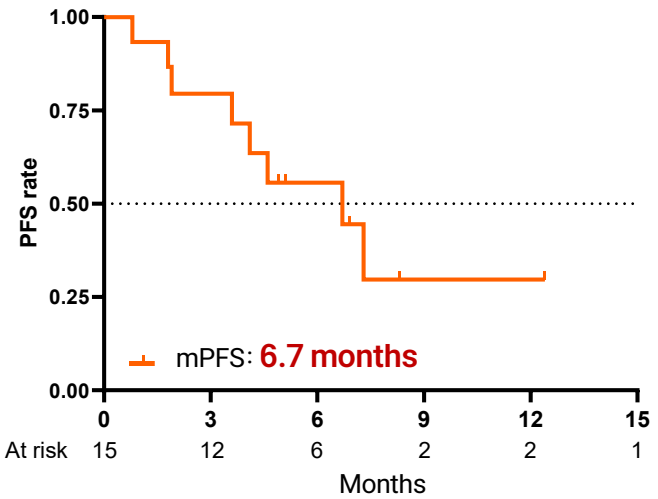
Superior brain permeability, Better intracranial PFS

VRN11 ⇔ Osimertinib Comparison: Improvement in PFS/iPFS for brain metastasis patients

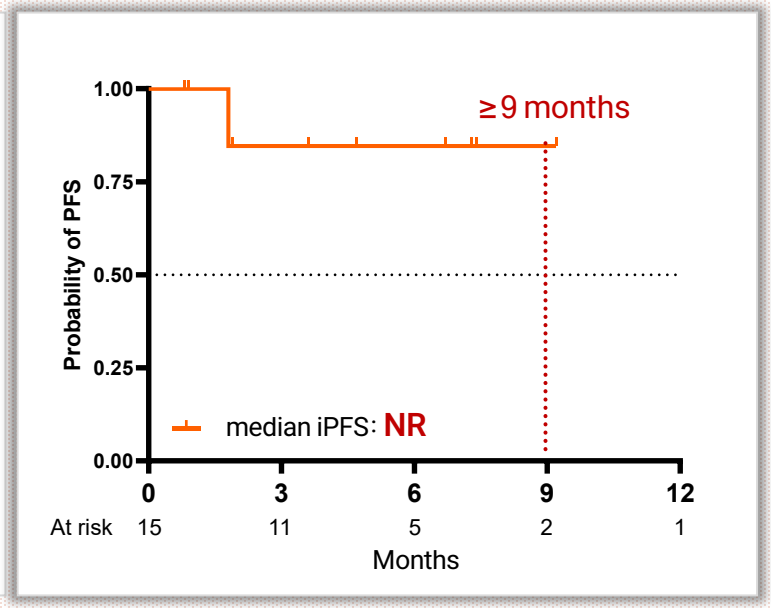
Meaningful PFS/iPFS benefit confirmed in patients with no treatment options post-3rd generation EGFR TKI
Demonstrated clinical advantage: median intracranial PFS ≥ 9 months (N.R.) vs. Osimertinib (3.5 months)

C797S -negative

Progression-Free Survival (PFS) (n=15)

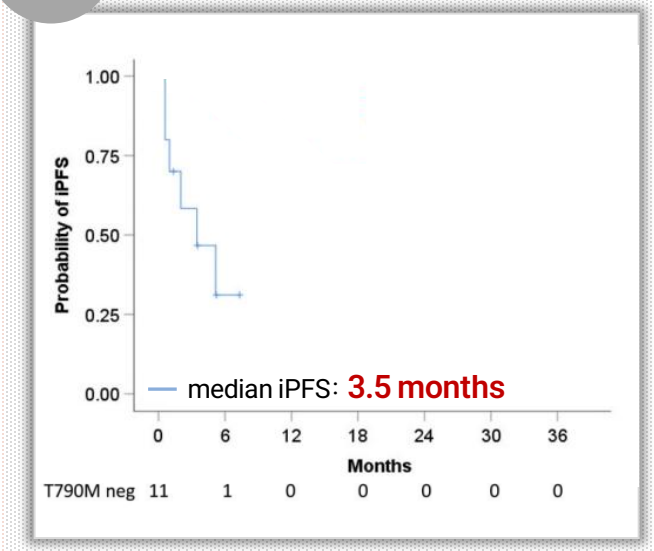


Intracranial Progression-Free Survival (iPFS) (n=15)



T790M -negative

Osimertinib: iPFS(n=11)[2]



✓14 patients previously treated with 3rd-generation EGFR TKIs

With baseline BM	VRN11 (≥160 mg, C797S negative)	Osimertinib[1,2] (T790M negative)
PFS (95% CI)	6.7 months (1.9 - NR)	1.6 months (0.9 - 2.7)
iPFS	NR	3.5 months (1.0 - NR)

PFS, Progression-Free survival; iPFS, Intracranial Progression-Free survival; BM, Brain metastases; NR, Not Reached

Source: ASCO2026, [1]Eide IJZ, et al. Osimertinib in T790M-positive and -negative patients with EGFR-mutated advanced non-small cell lung cancer: the TREM study. Lung Cancer. 2020;143:27–35.
[2]Eide IJZ, et al. Intracranial effect of osimertinib in relapsed EGFR-mutated T790M-positive and -negative non-small cell lung cancer patients: results from a phase II study. Acta Oncol. 2021;60(12):1565–1571.

Robust progression inhibition of CNS metastases

Effective CNS progression inhibition at ≥160mg, overcoming high CNS metastasis rates of current EGFR TKIs

Efficacy evaluable patients	160 mg	240 mg	320 mg	400 mg	480 mg	Total
All evaluable patients, N	12	14	6	5	2	39
CNS progression, n/N (%)	1/12 (8%)	2/14 (14%)	1/6 (17%)	0/5 (0%)	0/2 (0%)	4/39 (10%)
CNS metastases at baseline, N	7	5	2	4	0	18
CNS progression, n/N (%)	1/7 (14%)	0/5 (0%)	1/2 (50%)	0/4 (0%)	0/0 (0%)	2/18 (11%)
No CNS metastases at baseline, N	5	9	4	1	2	21
CNS progression, n/N (%)	0/5 (0%)	2/9 (22%)	0/4 (0%)	0/1 (0%)	0/2 (0%)	2/21 (10%)
Median Duration of Treatment (Weeks)	24	19	16	13	7	18

- Efficacy evaluable patients：at least one tumor assessment with ≥ 1 cycle of treatment.
- Brain and leptomeningeal metastases at baseline were confirmed by MRI.

Complete Response (CR) in Brain Non-Target Lesions
for 3 Patients with Baseline CNS Metastasis
(160-480mg, n=18)

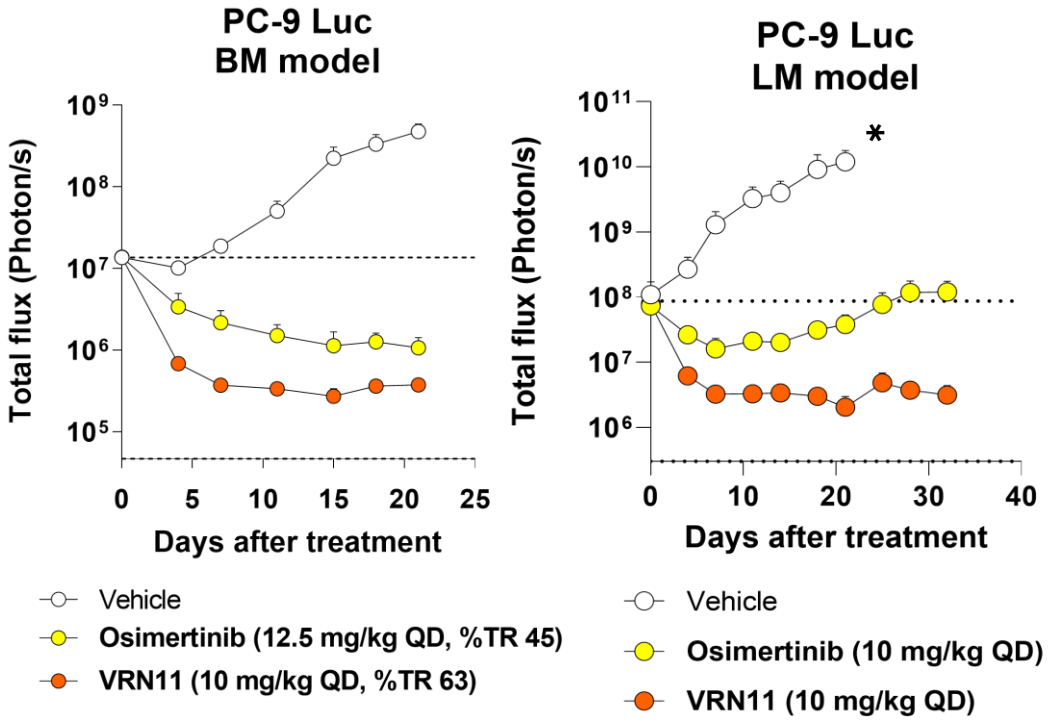
Partial Disappearance of Brain Non-Target Lesions (NTL)
at Initial Tumor Evaluation
(Osimertinib-treated patient with EGFR del19/C797S)

Source：ASCO2026

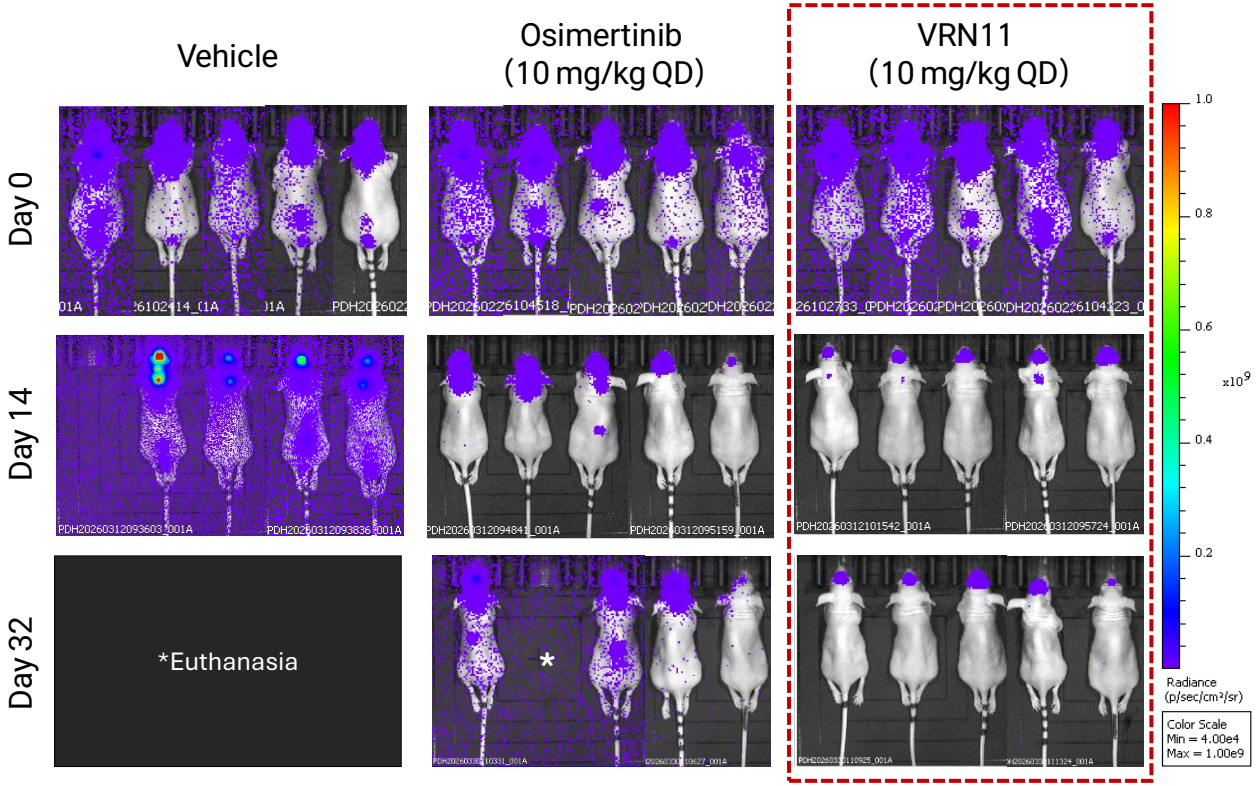
VRN11 ⇔ Osimertinib Comparison: *in vivo* BM/LM models

Superior CNS tumor inhibition vs. Osimertinib in BM and LM *in vivo* models

Intracranial (BM or LM) CDX model



Luciferase signal in LM model



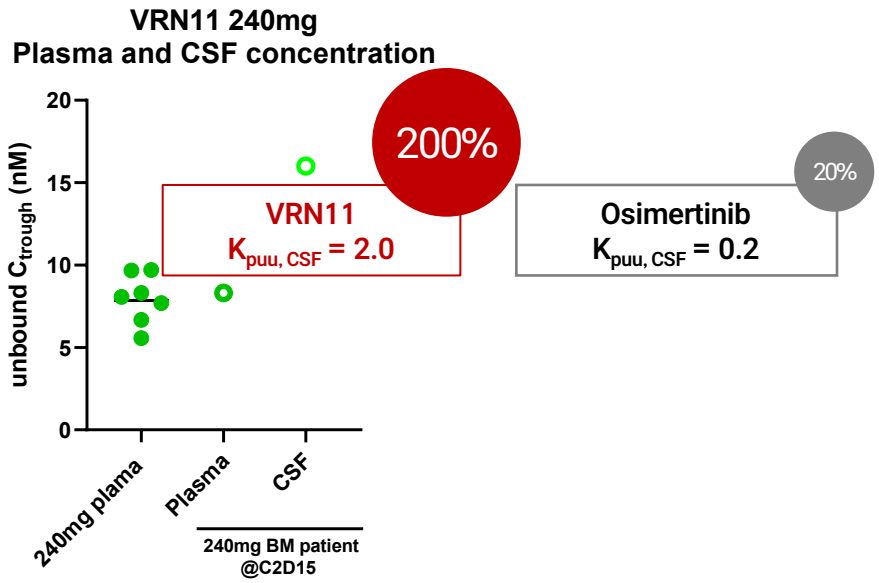
Mouse	Osimertinib[1]	VRN11
$K_{p,uu,brain}$	0.3	0.5

Source: AACR2026, [1]DOI:10.1124/dmd.118.084210
BM, Brain metastases; LM, Leptomeningeal metastases

VRN11 ⇔ Osimertinib Comparison: Brain Permeability & Target Engagement

First-in-Class Human PoC data demonstrating 200% BBB penetration vs. Osimertinib (20%)
Superior target engagement to Osimertinib: 4-fold higher in lung, 40-fold higher in brain

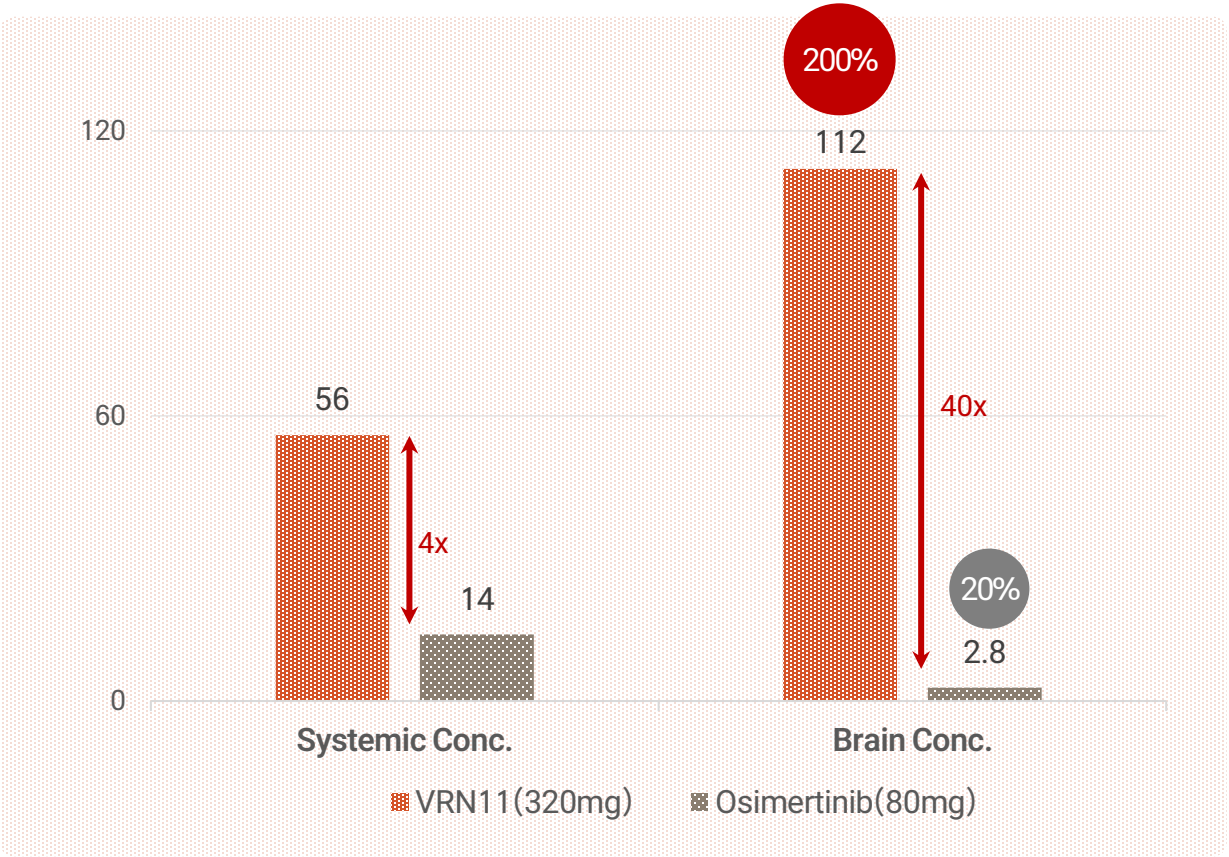
Free Drug Concentration in CSF ($K_{puu,CSF}$)



	Monkey	Human
$K_{p,uu,csf}$	2.6	2.0*

$K_{p,uu,csf}$ of Osimertinib in human has been reported, 0.22[1]

Target engagement Comparison(Systemic and Brain)

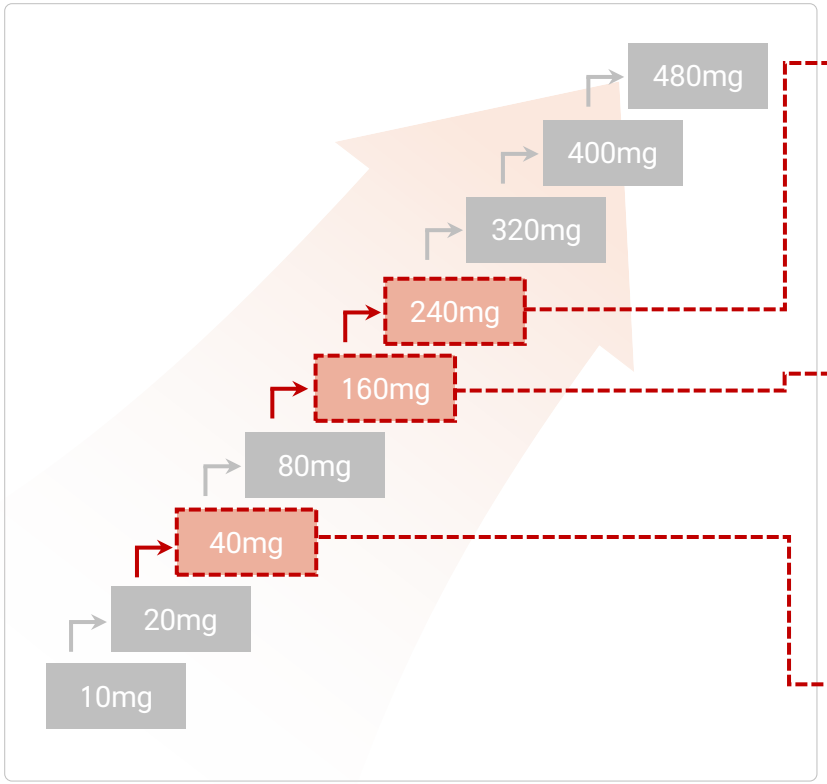


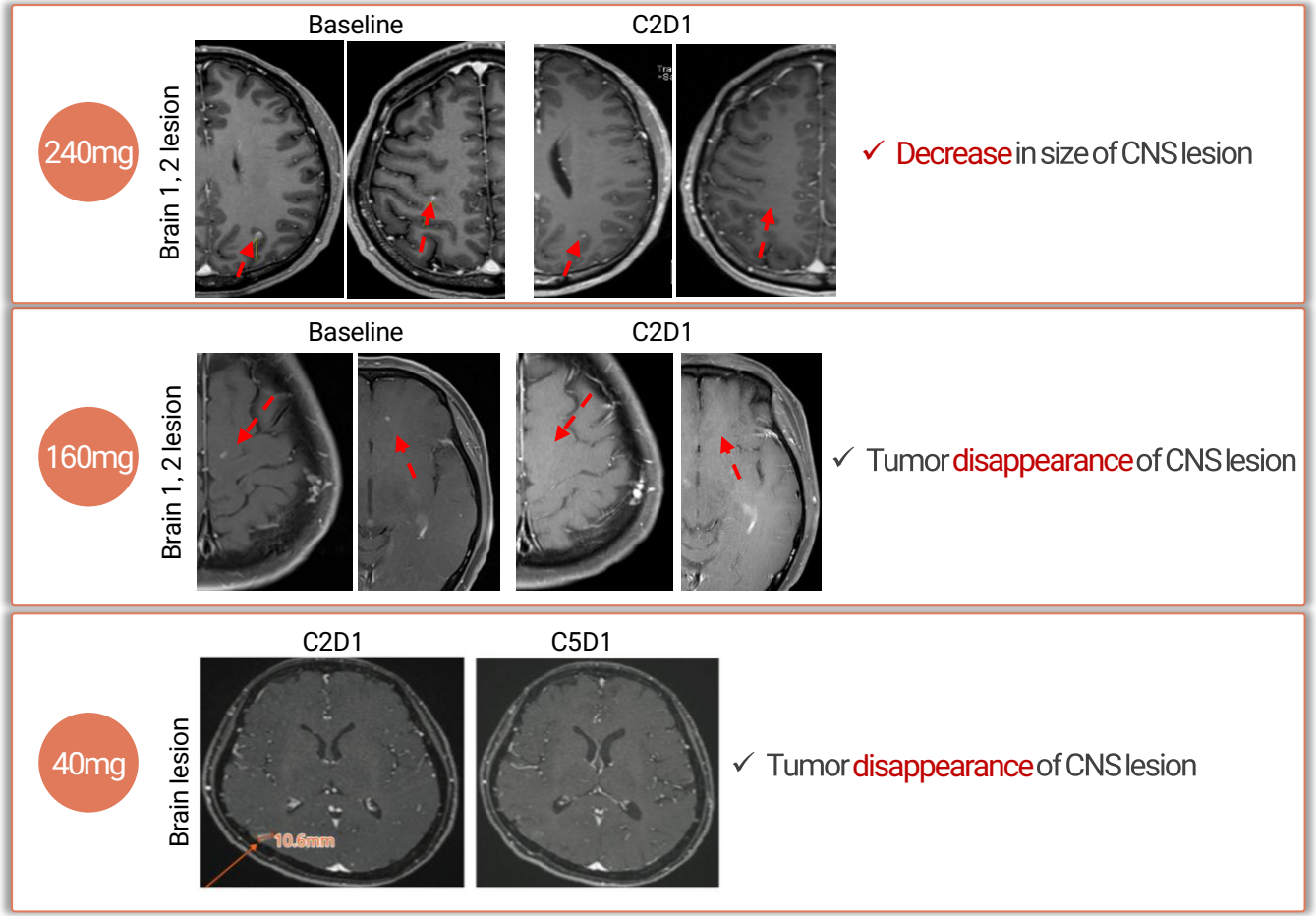
Source: [1]Park S, et al. J Clin Oncol. 2024;42(23):2747–2756
*A representative human PPB, 98.3% was used for calculation, $K_{puu, CSF}$ measured in 1 patient who received 240 mg

Case Report: Clinical efficacy in brain metastasis patients(1)

Antitumor activity confirmed from 40mg in phase 1 trial for brain metastases

Phase 1a. Dose escalation



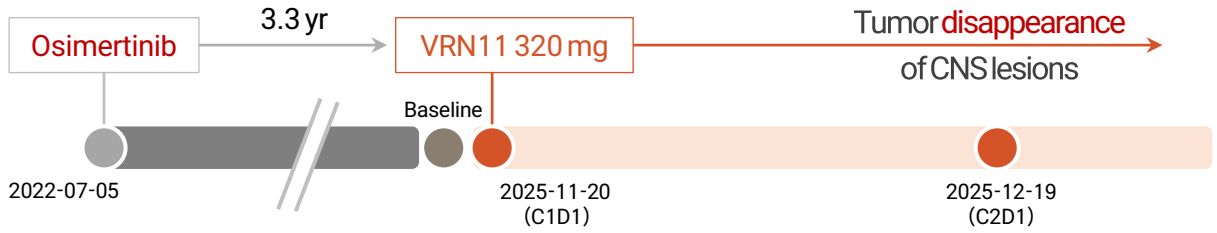


PR, Partial response; SD, Stable disease; PD, Progressive disease; TRAE, treatment-related adverse event; SAE, serious adverse event; DLT, dose limiting toxicity

Case Report: Clinical efficacy in brain metastasis patients(2)

Disappearance of brain lesions at first tumor assessment Post-VRN11 in 3rd-Gen EGFR TKI-pretreated patients
Achieved overall PR (Partial Response) with 50% reduction in lung lesions

320mg: 47-yr female with EGFR Del19/C797S NSCLC

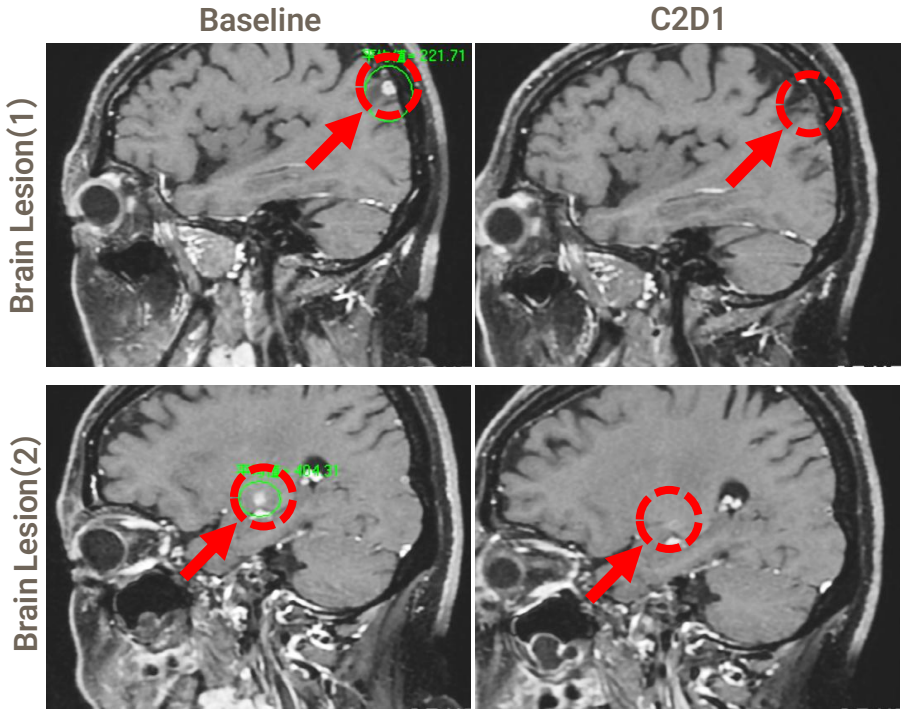


Baseline and Treatment History

- Lung, Brain, Bone metastasis
- Disease progression **after Osimertinib**

VRN11 Treatment

- Lung target lesion: 50% tumor reduction
- **Two brain lesions* disappeared** after the first cycle
- Baseline ctDNA of EGFR mutation cleared
- Overall best response: PR

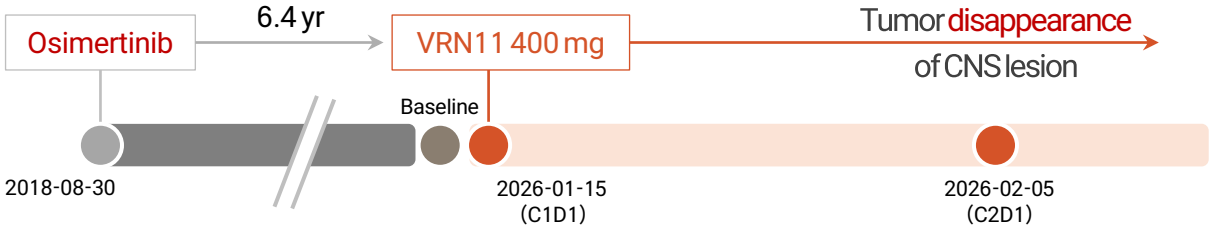


PR, Partial response; SD, Stable disease; PD, Progressive disease; TRAE, treatment-related adverse event; SAE, serious adverse event; DLT, dose limiting toxicity
*Non-target lesion

Case Report: Clinical efficacy in brain metastasis patients(3)

Disappearance of brain lesions at first tumor assessment Post-VRN11 in 3rd-Gen EGFR TKI-pretreated patients
Achieved overall PR (Partial Response) with 38% reduction in lung lesions

400mg: 71-yr male with EGFR L858R NSCLC

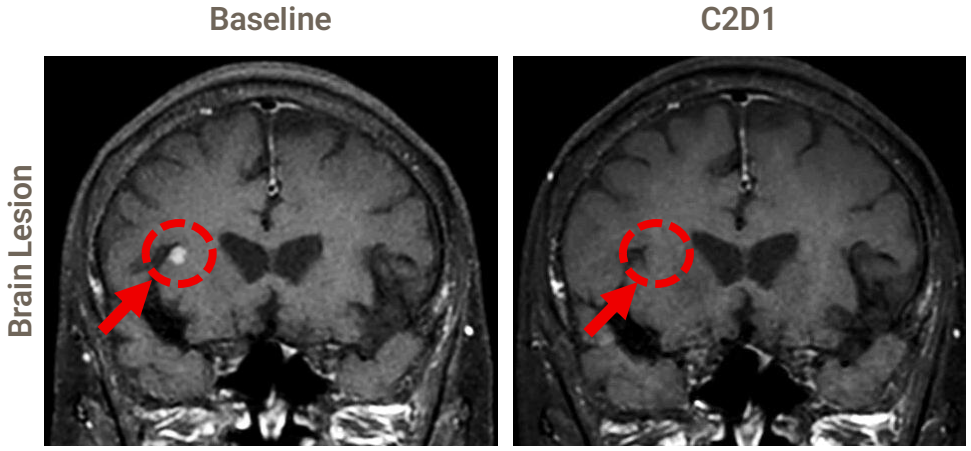


Baseline and Treatment History

- Lung and brain metastasis
- Prior EGFR TKIs: **Osimertinib, Lazertinib**

VRN11 Treatment

- Lung target lesion: 38% tumor reduction
- **Brain lesion*: disappeared** after the first cycle
- Overall best response: PR



PR, Partial response; SD, Stable disease; PD, Progressive disease; TRAE, treatment-related adverse event; SAE, serious adverse event; DLT, dose limiting toxicity
*Non-target lesion

VRN11

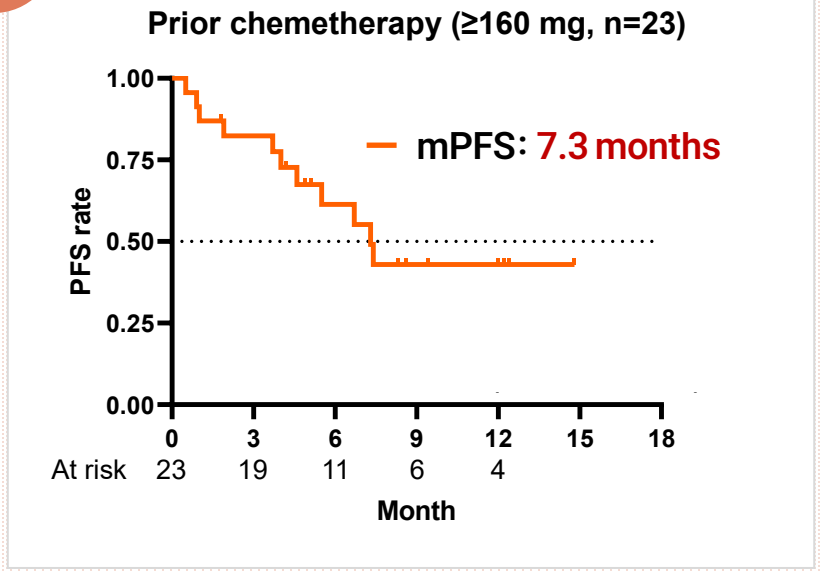
EGFR 2nd Line treatment

VRN11 ⇔ Osimertinib Comparison：Progression-free survival(PFS)；2L treatment

VRN11, mPFS of ≥ 7 months in chemotherapy-pretreated patients
Significant survival benefit over Osimertinib (4.07 months) in heavily pre-treated patients

C797S -negative

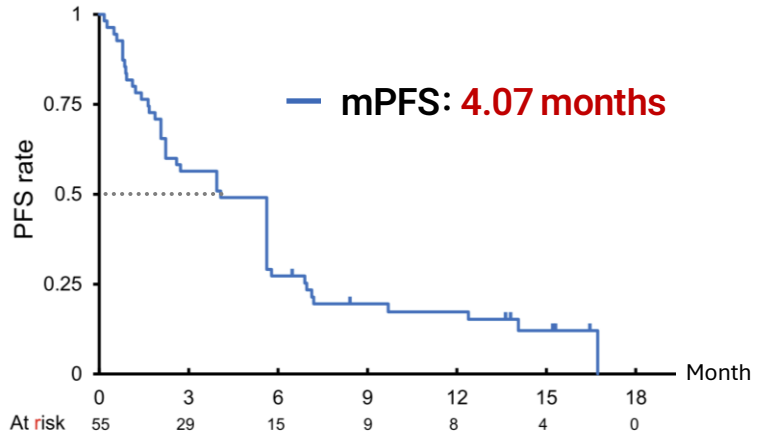
VRN11：Progression-Free Survival (PFS)



Prior EGFR TKI	
Gefitinib	22%
Erlotinib	13%
Afatinib	30%
Dacomitinib	4%
Osimertinib	48%
Lazertinib	35%

T790M -negative

Osimertinib：Progression-Free Survival (PFS)[1]



Prior EGFR TKI	
1st- or 2nd-Generation	100%
3rd generation	0%

	VRN11 (C797S negative)	Osimertinib[1] (T790M negative)
mPFS	7.3 months (95% CI, 4.0-NR months)	4.07 months (95% CI 2.1-4.3 months)

PFS, Progression-Free survival；
Source：ASCO2026, [1]Takeda M, et al. A phase II study (WJOG12819L) to assess the efficacy of osimertinib in patients with EGFR mutation-positive NSCLC in whom systemic disease (T790M-negative) progressed after treatment with first- or second-generation EGFR TKIs and platinum-based chemotherapy. Lung Cancer. 2023；177：44－50.

Unmet needs in resistant/refractory patients

Current therapies show limited efficacy and safety in SOC – resistant/refractory patients
Strong disease control observed in heavily pretreated patients supports the second-line exploration

Key updates on VRN11 (ph1a)

Efficacy

- Disease Control Rate (DCR) 96.8% (30/31)
- 17/31 patients ongoing
- Patients under treatment for ≥ 14 months at data cutoff

Safety

- up to 480mg patients (n=65), ≥ Grade 3 TRAEs 1.5%
- Permanent discontinuation 0% (due to TRAEs)

*Data cutoff March 12,2026

SOC resistant/refractory patient trial indirect comparison[1,2,3,4]

	VRN11	Dato-Dxd	Chemotherapy	Chemotherapy, Pembrolizumab	Chemotherapy, Amivantamab	Chemotherapy, Amivantamab, Lazertinib
mPFS (months)	≥9.8*	4.4	4.2	5.6	6.3	8.3
≥ Grade 3 TRAE	1.5%	25.6%	35%	43.7%	67%	87%

Treatment Option for EGFR 2L+ pts

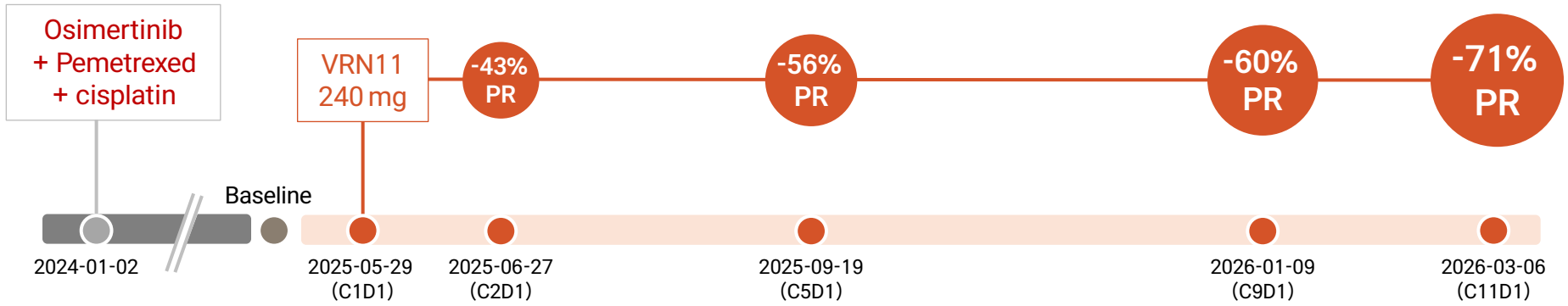
Planned VRN11 monotherapy and combination with chemotherapy in EGFR SoC-refractory patients

PFS, progression-free survival; TRAE, treatment-related adverse event; DCR, disease control rate
Source: [1]Passaro, A. et al. Annals of Oncology, Volume 35, Issue 1, 77 - 90, [2]Jänne PA, et al. N Engl J Med. 2015;372(18):1689-1699, [3]Myung-Ju Ahn et al. J Clin Oncol 43, 260-272(2025), [4]James Chih-Hsin Yang et al., JCO 42, 4029-4039(2024)
*Included patients with EGFR C797S positive mutation

Case Report: Demonstrated durable tumor suppression following standard therapy failure

Confirmed sustained tumor reduction with VRN11 in an EGFR-mutant patient with 10 prior lines of therapy

Representative Case in heavily-treated patient(240mg, EGFR L858R)



Baseline and Treatment History

- Metastatic site: Lung, Lymph node
- had received 10 prior lines of therapy, including Erlotinib and Osimertinib

VRN11 Treatment

- Target lesions showed a sustained decrease over time, reaching a 70% reduction at C11D1
  (Lymph node: **17.4 → 5.6mm**/Lung target lesion: **18.9 → 4.7mm**).
- Baseline ctDNA of EGFR L858R mutation also decreased (**VAF 5.7 → 0.06%**)

PR, Partial response

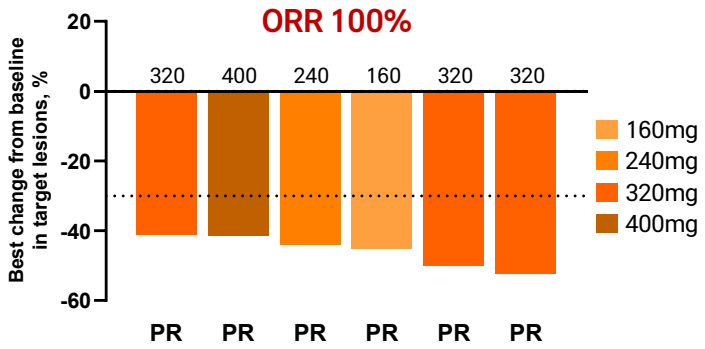
VRN11
EGFR C797S

Efficacy in Patients with EGFR C797S Mutation

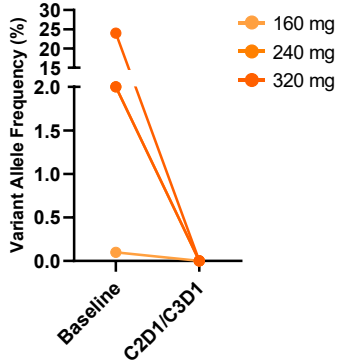
An objective response rate of 100% (6/6, partial responses) was observed in all patients with the EGFR C797S mutation treated at or above the efficacy dose (≥160 mg)

Efficacy in Patients with EGFR C797S Mutation

✓ Best Response in Target Lesion Size

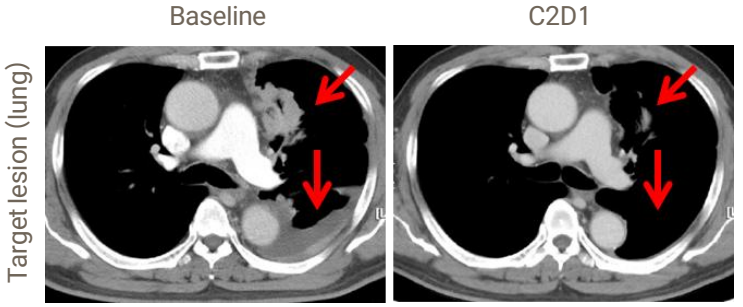


✓ ctDNA VAF dynamics

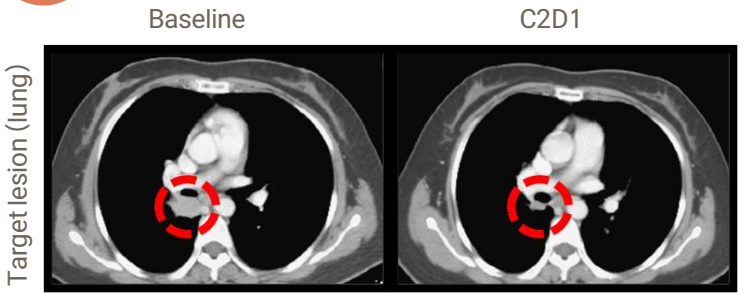


Representative Cases of Systemic Control

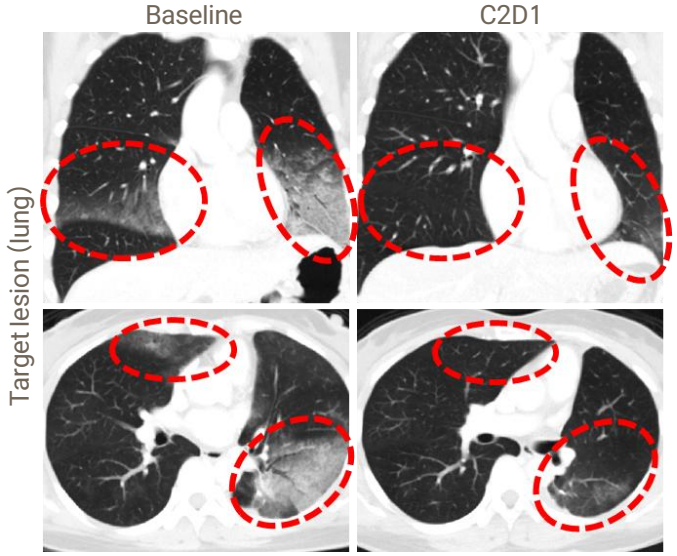
320mg EGFR Del19/C797S NSCLC(63-yr male)



320mg EGFR Del19/C797S NSCLC(47-yr female)



320mg EGFR L858R/C797S NSCLC(60-yr female)

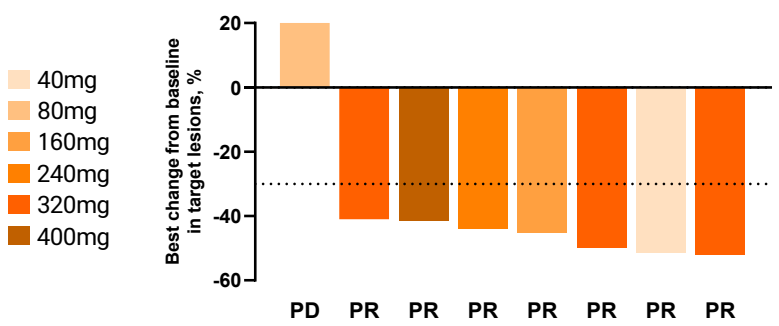


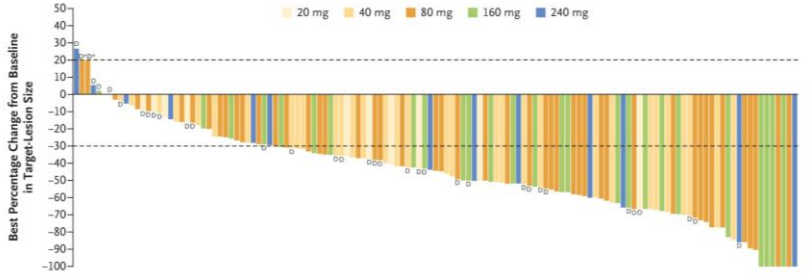
ORR, objective response rate; PR, Partial response; SD, Stable disease; PD, Progressive disease

VRN11 ⇔ Osimertinib Comparison：Comparative Clinical Profile

Indirect Comparison of 4th generation potency vs. 3rd generation standard of care

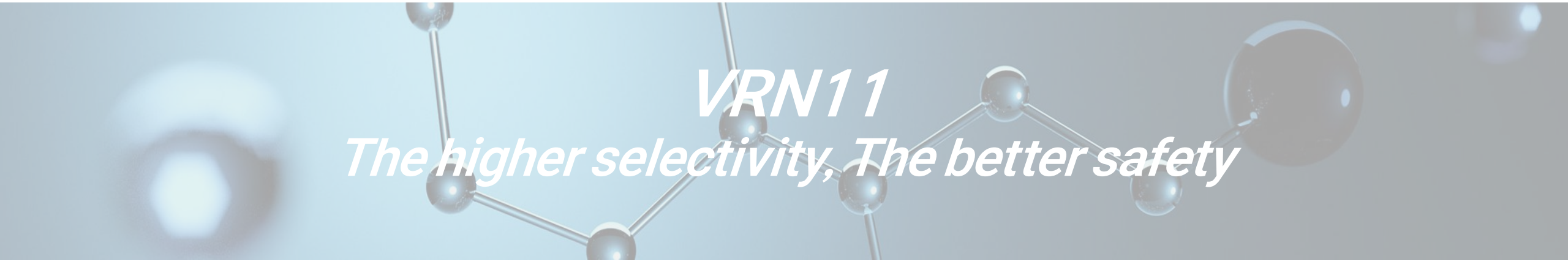
Indirect Comparison of VRN11 vs. Osimertinib





	VRN11	Osimertinib(AURA trial)
Phase	Phase 1a Dose escalation	Phase 1 Dose escalation/Dose expansion
Target resistance mutation	EGFR C797S (Acquired post-Osimertinib)	EGFR T790M (Acquired post-1G/2G TKI)
ORR	87.5%	61%
mPFS	9.8 months (included C797S+ pts, ≥160mg)	9.6 months
Grade 3+ TRAE	1.5%	13%

ORR, objective response rate; PR, Partial response; SD, Stable disease; PD, Progressive disease; mPFS, median progression-Free survival; TRAE, treatment-related adverse event
Source: Jänne PA, et al. N Engl J Med. 2015;372(18):1689-1699.



VRN11

The higher selectivity, The better safety

Favorable safety and tolerability profile in EGFR-mutant NSCLC patients

Excellent tolerability confirmed with only a single case (≥ Grade 3) of TRAE (1.5%)
No major off-target toxicities reported, particularly irreversible or unpredictable adverse events

Compound		VRN11 (80 – 480 mg QD, N = 55)				Osimertinib (80 mg QD, N=201)[1]			
Median No. of prior systemic therapies (range)		3 (1-10)				2 (1-11)			
Treatment-related adverse events		Grade 1	Grade 2	≥ Grade 3	Any grade	Grade 1	Grade 2	≥ Grade 3	Any grade
Any treatment-related AE		40%	24%	2%*	68%	*NR***	*NR*	15%	92%
EGFR-Related	Diarrhea	9%	4%	-	13%	37%	5%	<1%	43%
	Rash	18%	4%	-	22%	33%	6%	<1%	40%
	Dry skin	9%	4%	-	13%	28%	3%	-	31%
	Pruritus	11%	4%	-	16%	10%	3%	-	13%
	Stomatitis	5%	-	-	5%	10%	3%	-	13%
Non-EGFR-Related	Decreased appetite	5%	-	-	5%	8%	1%	<1%	10%
	Nausea	11%	-	-	11%	9%	1%	<1%	11%
Special interests	Interstitial lung disease (ILD)	-	-	-	-	1%		3%	4%
	QT prolongation	-	-	-	-	1%	<1%		2%

*One patient experienced Grade 3 acute kidney injury; the event was reversible, and no recurrent cases were observed in other treated patients.
**NR, not reported; Grade1/2 breakdown for overall TRAE not reported in source.

- **No DLTs** were observed, and the MTD and RP2D have not yet been determined.
- **No patients** experienced drug-related adverse events leading to **treatment discontinuation.**
- **No treatment-related clinically meaningful QT prolongation or ILD** was observed.
- Any drug-related adverse event leading to dose reduction was observed in 4 patients (6%).
- Any drug-related adverse event leading to dose interruption was observed in 8 patients (13%).

Source: ASCO2026, [1]Yang JCH, et al. Osimertinib in pretreated T790M-positive advanced non-small-cell lung cancer: AURA study phase II extension component. J Clin Oncol. 2017:35(12):1288–1296.

VRN11
Clinical Development Strategy

VRN11 Clinical development strategy

Aims to address unmet needs across EGFR-mutant NSCLC, supported by strong efficacy, safety, and brain penetration data

Second Line

Brain Metastasis

- Superior Clinical data demonstrated to Osimertinib*:
  - Kp,uu,CSF：0.2 vs 2.0
  - iPFS：3.5 vs Not Reached
- No treatment option for 2L brain metastases post Osimertinib; accelerated approval planned based on Phase 2 data

2L post-3rd gen EGFR TKI (unknown)

- Strong efficacy(DCR 96.8%) and favorable safety(≥Grade 3 TRAEs 1.5%) in heavily pretreated patients
- Initiation of a global, multi-center phase 1b/2 clinical trial

C797S

- No treatment options
- 100% ORR (6/6) Achieved at Therapeutic Dose Levels (≥160mg)
- Targeting accelerated approval based on phase 2 clinical data

First Line

Brain Metastasis

- Up to 40% of patients with CNS metastasis at diagnosis
- Enrollment plan for treatment naïve patients with brain metastases
- Expecting higher CNS Complete Response (CR) than Osimertinib of ~40%

1L settings

- VRN11 320mg achieved 4x higher target engagement than Osimertinib (approved dose of 80mg)
- Expected to demonstrate superior depth of Response and PFS to SOCs
- Initiation of a global, multi-center phase 1b/2 clinical trial

2H 2026:
Ongoing phase 1b/2 clinical trials

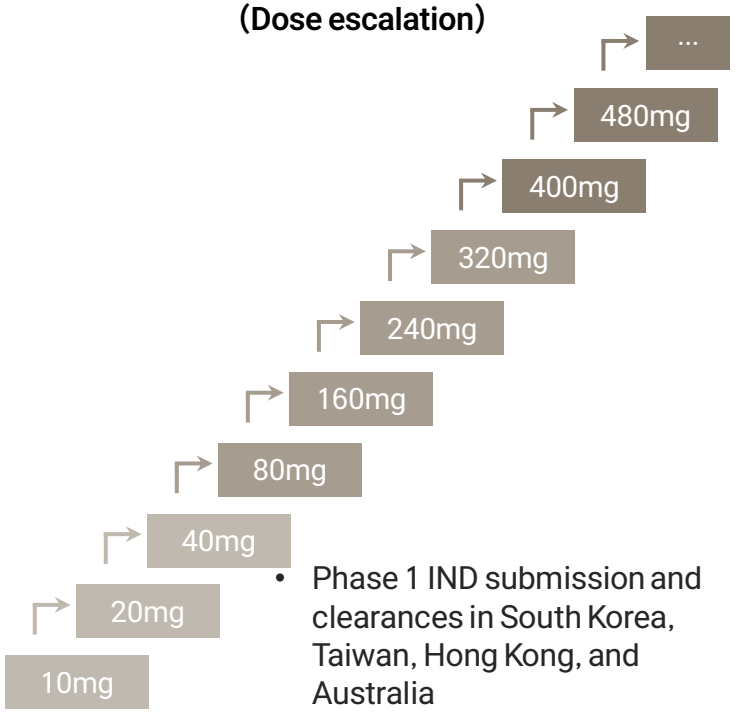
Source: *S Park, et al., 2024 J. Clinic. Oncology.

Phase 1/2 Clinical Trial Design

Ongoing global phase 1 trial in South Korea, Taiwan, and Other regions
1H 2026, Phase 1b/2 IND approval and study initiation across select countries/sites

Phase 1/2 Clinical Trial Design

Phase1a
(Dose escalation)



- Phase 1 IND submission and clearances in South Korea, Taiwan, Hong Kong, and Australia

Phase 1b/2
(Dose expansion Dose optimization)

- Phase 1b/2 IND submitted (Taiwan, Australia, Singapore, Europe, and others)
- Planned clinical site Expansion, including South Korea, Hong Kong, and Canada

Phase 1b/2. Cohort Design

Monotherapy

- Common EGFR mut. Treatment-naive
- Common EGFR mut. With C797S After 1L 3rd Gen TKI (Osi, Laz, Ami + Laz)
- Atypical/uncommon EGFR mut. TKI-naive

Combination therapy

- Any EGFR mut. VRN11 + SoC Chemotherapy

+

Planned clinical trial expansion with a brain metastasis cohort

VRN11

EGFR 1st line treatment (CNS metastases)

Targeted Therapies for NSCLC by Generation and BBB Penetration Rates (EGFR/ALK)

Targeted Therapies for NSCLC by Generation and BBB Penetration Rates (EGFR/ALK)[1,2,3,4]

EGFR TKI

1st Gen. Drug	3rd Gen. Drug	Next Gen. Drug
Gefitinib	**Osimertinib**	**VRN11**
CSF/plasma ratio	$K_{p,uu,CSF}$	$K_{p,uu,CSF}$
0.013	0.22	2.0

- Key Differentiations
  - Overcoming post-3rd Gen EGFR TKI resistance mutations
  - Significant BBB penetration vs competitors (2.0 vs. 0.2)
  - High EGFR selectivity ensuring excellent tolerability (≥Grade 3 TRAEs 1.5%)*
- Addressable market of EGFR: 30~40% of all NSCLC patients

ALK TKI

1st Gen. Drug	3rd Gen. Drug	Next Gen. Drug
Crizotinib	**Lorlatinib**	**Neladalkib(NVL-655)**
CSF/plasma ratio	$K_{p,uu,CSF}$	
0.0026	0.77	

- Key Differentiations
  - Overcoming Post-2nd/3rd Gen ALK TKI Compound Mutations
  - Superior BBB Penetration to existing options
  - Minimal Neurological Side Effects related to TRK Inhibition
- Addressable Market of ALK: 3~5% of all NSCLC patients
- June 2026: GSK acquired Nuvalent for $10.6B

Source: [1]Zhao J, et al. Clin Lung Cancer. 2013;14(2):188-193 [2]Park S, et al. J Clin Oncol. 2024;42(23):2747–2756 [3]Costa DB, et al. J Clin Oncol. 2011;29(15):e443-e445 [4]Sun S, et al. J Clin Pharmacol. 2022;62(9):1170-1176.
*Date cut-off: March 12, 2026

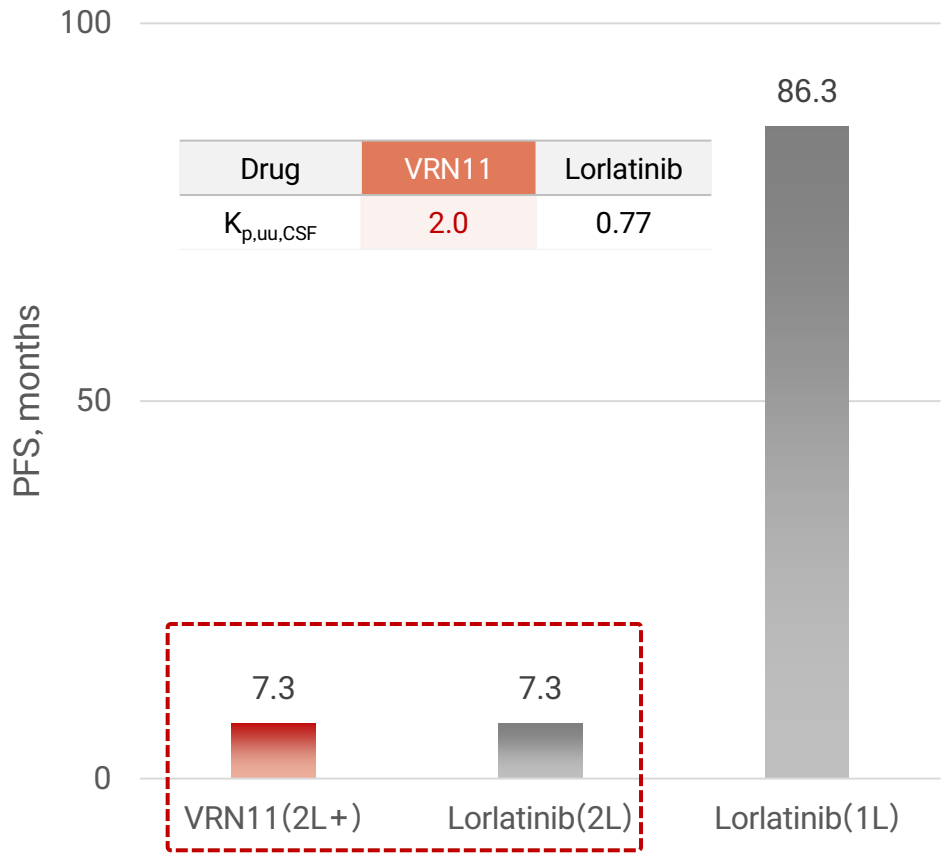
Clinical Benchmark：Analysis of Lorlatinib(ALK) historical Efficacy data

[ALK] Efficacy Comparison：Patients with CNS metastases at baseline

Drug (Clinical study)	Lorlatinib[1] (2L+, NCT01970865)	Lorlatinib[2,3] (1L, CROWN)
CNS Complete response (with any brain metastases at baseline)	-	61%* (23/38)
CNS Complete response (with measurable brain metastases)	20% (16/81)	71%* (12/17)
CNS PFS, months (with baseline brain metastases)	-	The probability of being free of intracranial progression was 83% at 7 years.
mPFS, months	7.3 (including patients with no BM)	86.3 (only BM pts.)

*The median duration of follow-up for PFS was 18.3 months

PFS Comparison：Patients with CNS metastases at baseline



Source：[1]Solomon BJ, et al. Lancet Oncol. 2018;19(12):1654-1667. [2]Shaw AT, et al. N Engl J Med. 2020;383(21):2018-2029. [3]Shaw AT, et al. Ann Oncol. Published online May 29, 2026.

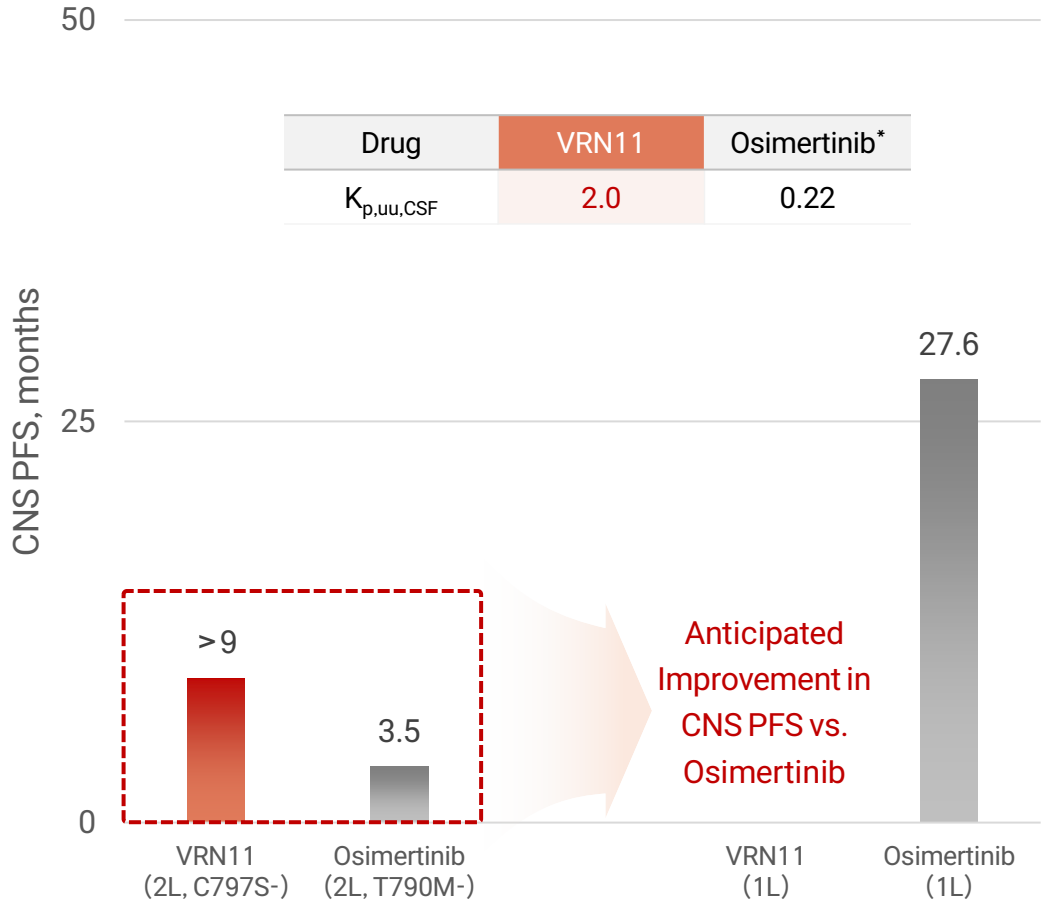
Clinical Benchmark: Analysis of Osimertinib(EGFR) historical Efficacy data

Unmet medical need for treatment options for CNS metastases in EGFR-mutant NSCLC

[EGFR] Efficacy Comparison: Patients with CNS metastases at baseline

Drug (Clinical study)	Osimertinib[1,2] (2L+, TREM, T790M-)	Osimertinib[3,4] (1L, FLAURA2)
CNS Complete response (with any brain metastases at baseline)	0% (0/11)	43% (45/104)
CNS Complete response (with measurable brain metastases)	-	16% (6/38)
CNS PFS, months (with any brain metastases at baseline)	3.5	27.6
mPFS, months (with any brain metastases at baseline)	1.6	13.8

PFS Comparison: Patients with CNS metastases at baseline



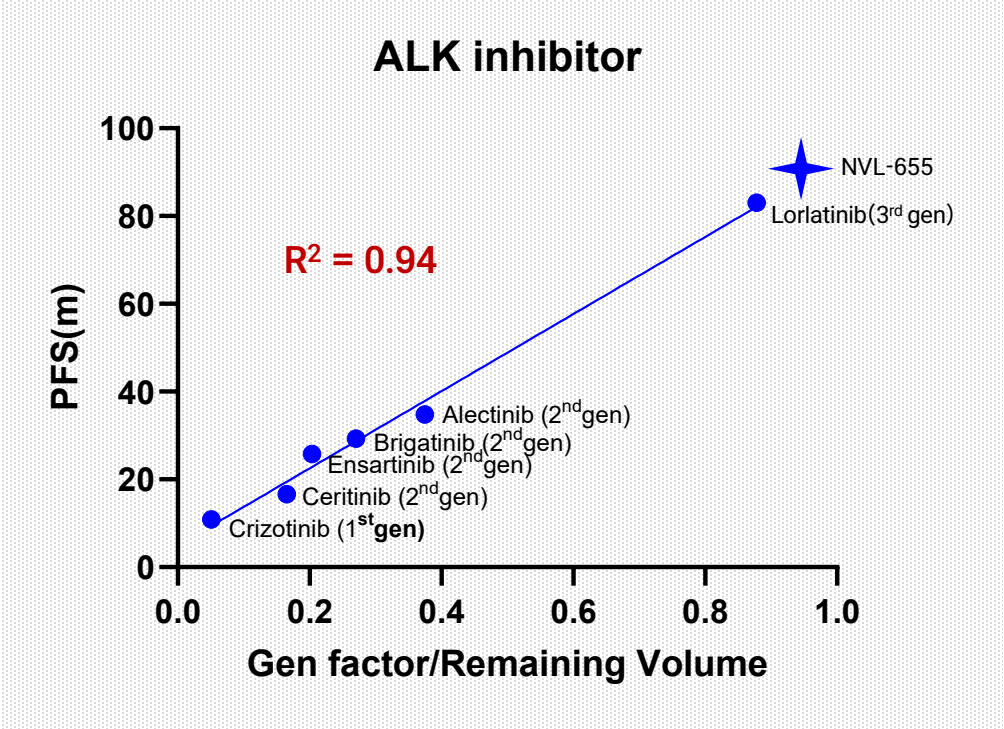
Source: Eide IJZ, et al. Lung Cancer. 2020;143:27–35.[2]Eide IJZ, et al. Acta Oncol. 2021;60(12):1565–1571. [3]Jänne PA, et al. J Clin Oncol. 2024;42(7):808-820. [4]Planchard D, et al. N Engl J Med. 2023;389(21):1935-1948.
*Park S, et al. J Clin Oncol. 2024;42(23):2747–2756

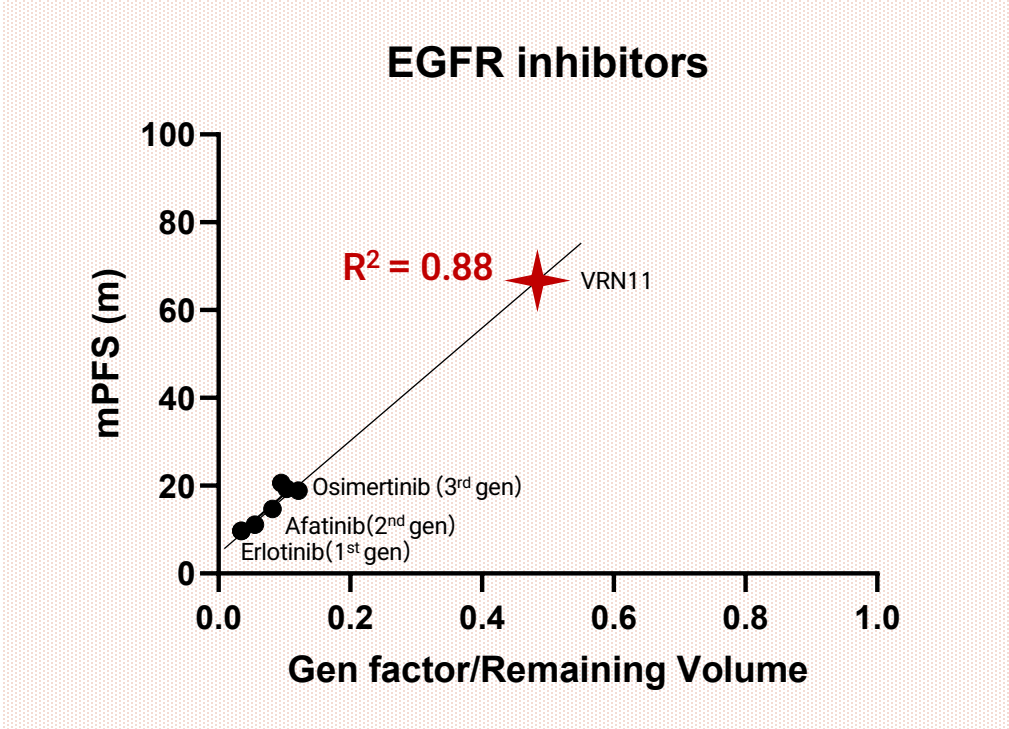
VRN11

EGFR 1st line treatment

PFS & Depth of Response Correlation (ALK, EGFR)

Correlation confirmed in ALK setting: Higher target engagement driving greater tumor reduction and extended PFS





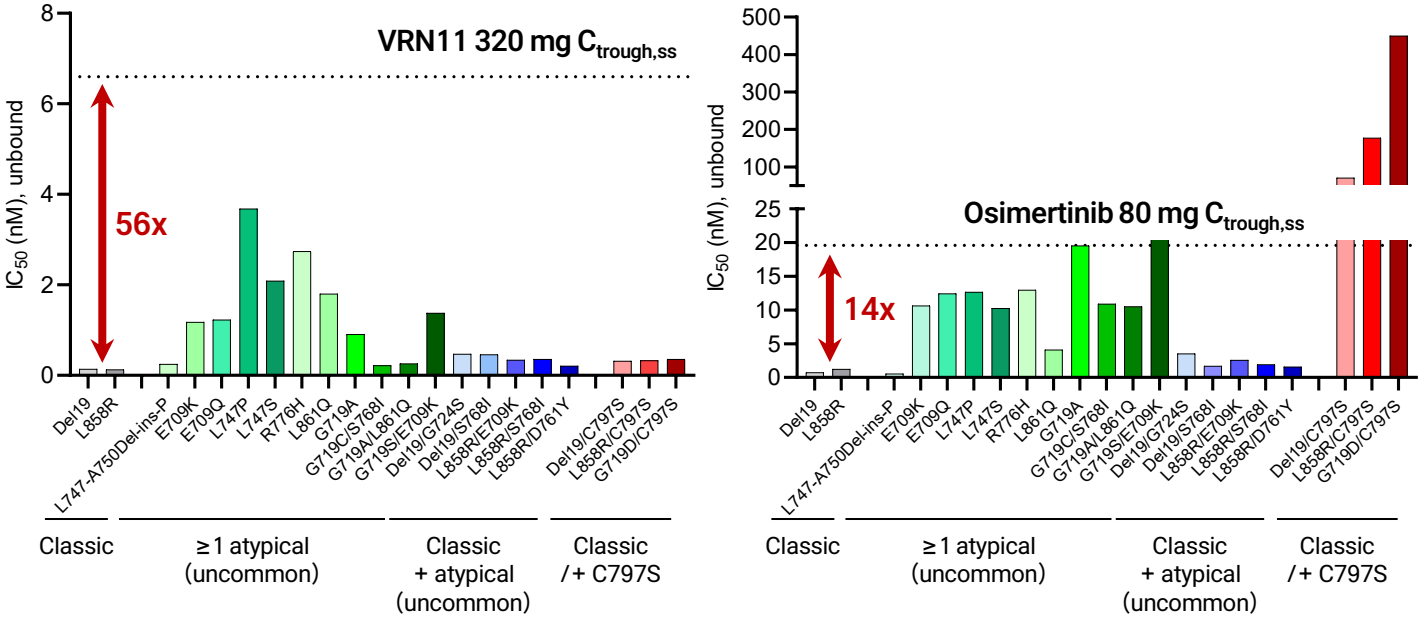
	Nuvalent	VORONOI
Market Cap(Jun 30, 2026)	USD 9.8B	USD 2.3B
Addressable Market	ALK (3~5% of NSCLC)	EGFR (30~40% of NSCLC)

1 USD = 1,527.30 KRW

Preclinical and clinical evidence of efficacy in EGFR-mutant NSCLC

Confirmed robust activity of VRN11 320mg across broad EGFR mutations
4-fold stronger target engagement vs. Osimertinib 80mg in EGFR del19/L858R, demonstrating distinct CNS superiority via exceptional BBB penetration

In vitro Target Engagement in Ba/F3-engineered Cell Lines



Target engagement ratio

Target engagement ratio (TER)

	VRN11 320mg	Osimertinib 80mg	VRN11 / Osimertinib
Classic	56	14	**4x**
Classic + C797S	22	< 1	140x
Classic + Atypical	20	9	2x
Atypical	5	2	3x

x 2.0

x 0.2

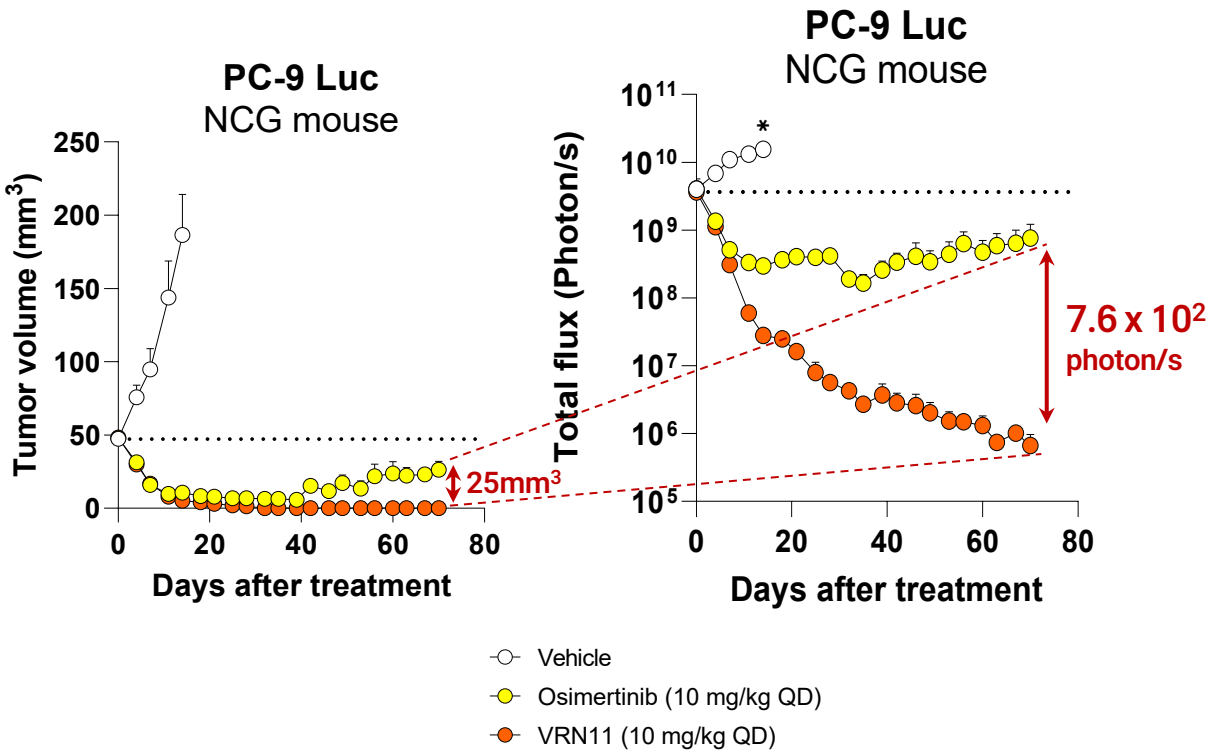
Target engagement ratio (TER); Brain

	VRN11 320mg	Osimertinib 80mg	VRN11 / Osimertinib
Classic	112	2.8	**40x**

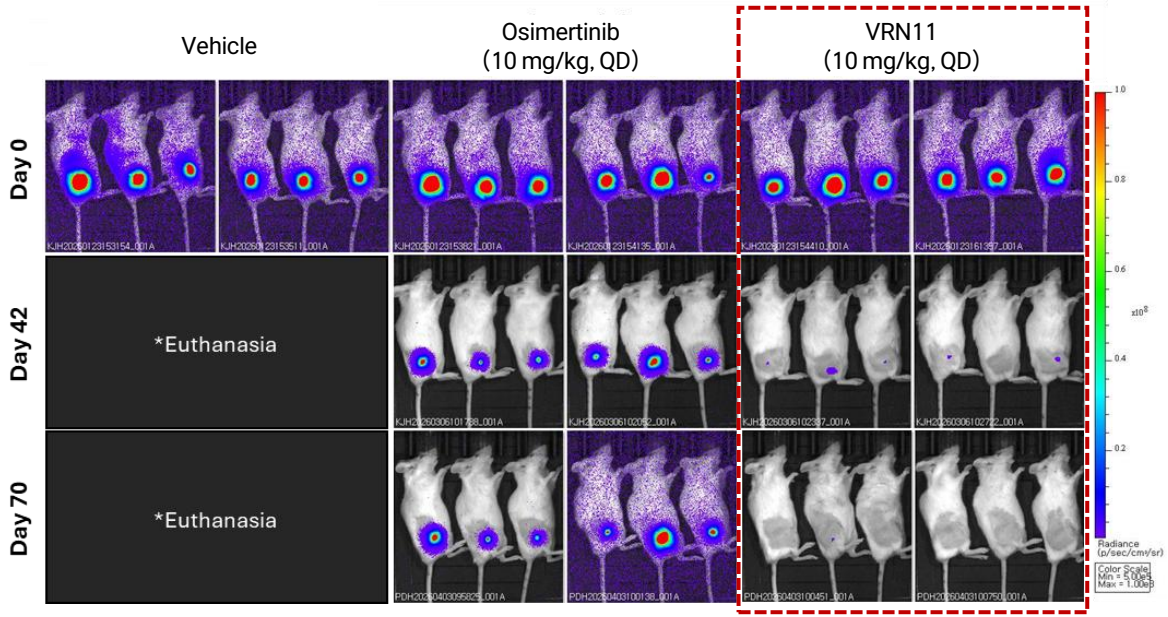
In Vivo model-proven prediction of tumor response to VRN11：Deeper Response

VRN11 exhibits longer duration of response compared to osimertinib in subcutaneous CDX model

Subcutaneous CDX model(PC9 _ Del19)



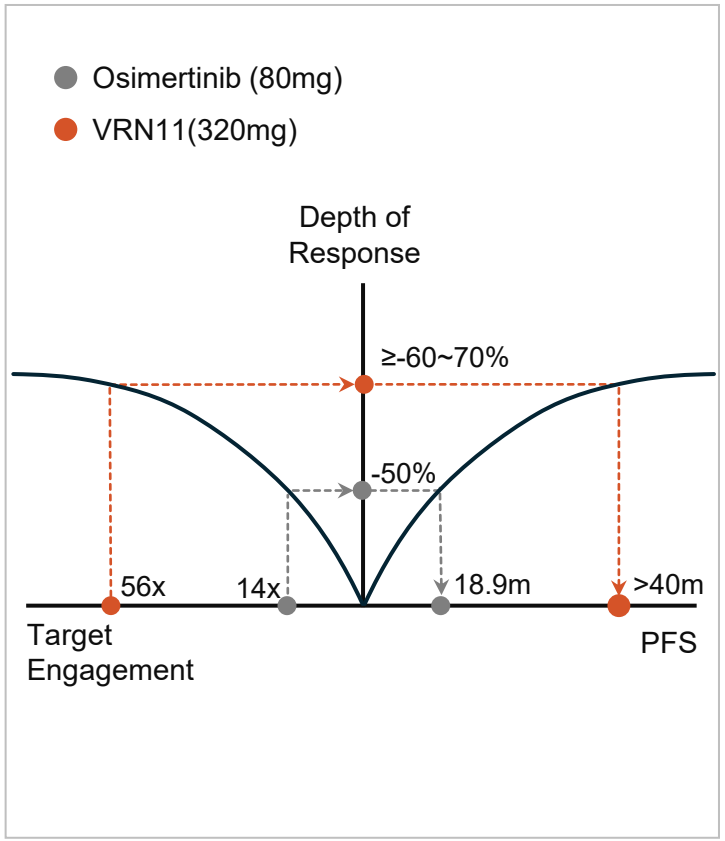
Luciferase signal in SC model

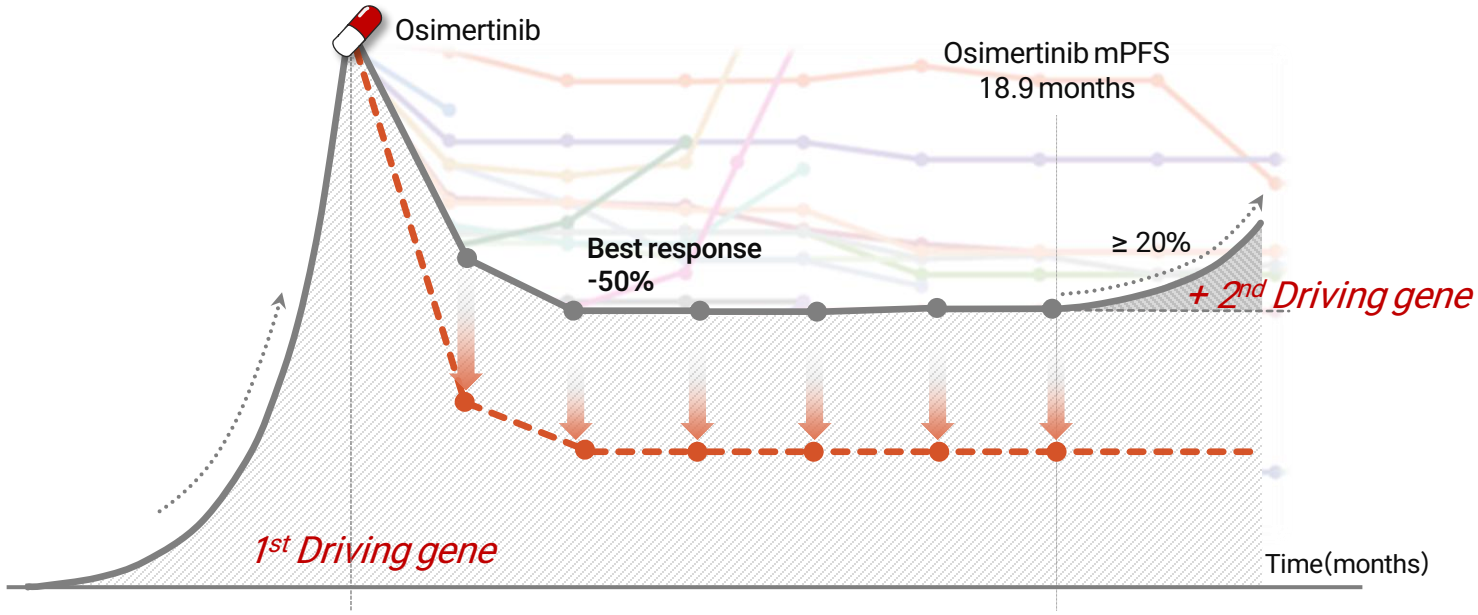


PFS & Depth of response correlation

Lower residual tumor cell count reducing risk of resistance/bypass pathways, leading to **anticipated PFS prolongation**

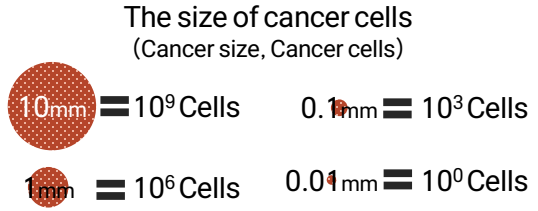
Depth of Response: remaining cells ↓ → chance of resistance ↓ [1,2]





$$P_{Event\ of heterogeneity} = f(n, v, p) + BM$$

$n = \#\ of\ cells\ in\ tumor,\ v = doubling\ time$
$p = probability\ of\ resistance$

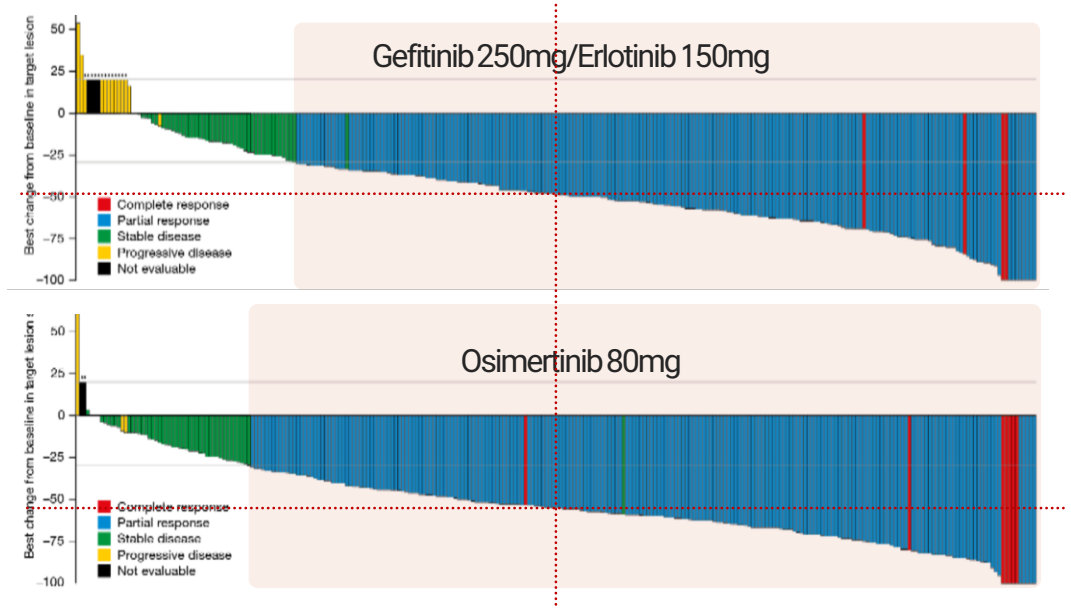


Source: [1]Park CK, et al. Cancer Res Treat. 2021;53(1):93-103, [2]David Planchard, et al. N Engl J Med 2023;389:1935-48

PFS & Depth of response correlation

Less remaining tumor cells → less chance of resistance / bypass → longer duration of response / PFS
VRN11 in 1L setting is expected with greater depth of response compared to existing EGFR TKIs

Depth of Response: short term readout / EGFR NSCLC 1L best of response comparison(1st vs 3rd gen)[2]



Gefitinib/Erlotinib 150mg
median Best response
= -47~48%

Osimertinib 80mg
median Best response
= -55%

VRN11
median Best response
= ≥-60~70%(expected)

VRN11
Data are expected to be released in 2H of 2026

PFS: long term readout[1,2]

1st Generation
Gefitinib

PFS 10.8 months
ORR 73.7%

3rd Generation
Osimertinib

PFS 18.9 months
ORR 80%

Next Generation
VRN11

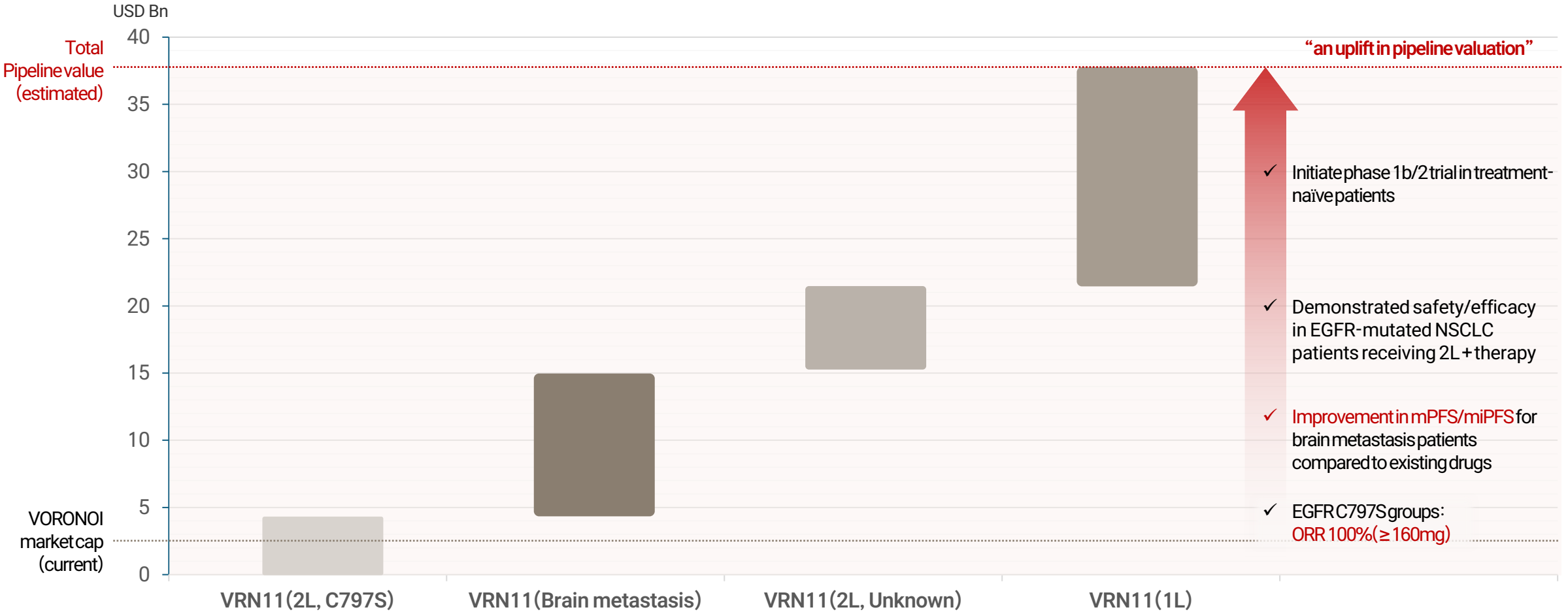
PFS improvement expected

PFS, Progression Free Survival; ORR, Objective Response Rate
Source: [1]Makoto Maemondo, et al. N Engl J Med 2010;362:2380-8, [2]J.-C. Soria, et al. N Engl J Med 2018;378:113-25


Valuation

VRN11: Pipeline valuation

VRN11's near-term potential as a second-line therapy alone is estimated to drive a corporate valuation of approximately USD 10B



Brain metastasis, BM; 1 USD = 1,527.30 KRW
Source: BIO, Biomedtracker, Amplion, Clinical Development Success Rates 2006-2015, 2016.6; Schulze U, Ringel M. *Nat Rev Drug Discov.* 2013;12(6):419-420.
The weighted average cost of capital (WACC) was conservatively set at 10%, based on the average WACC (6 – 7%) of the top 10 global pharmaceutical companies by revenue in 2024 over the past three years.
For FCF to Sales, the average was derived using the financial data of three companies specializing in kinase inhibitors.
The above information does not guarantee future performance and is subject to change depending on market conditions.

VRN11: Pipeline Valuation

(Unit: USD mn)

■ Key Pipeline

Category	rNPV	Probability of Success
VRN11(2L; C797S)	4,263	100%
VRN11(Brain metastasis)	10,660	44.1%
VRN11(2L; Unknown)	6,213	44.1%
VRN11(1L)	16,200	33.8%
Subtotal	37,336	
Pipeline Value – Total	**37,336**	

Key assumptions

	Incidence among NSCLC (EGFR TKIs)	Market share
VRN11(2L; C797S)	10%	increasing gradually*
VRN11(BM)	30%	increasing gradually*
VRN11(2L; Unknown)	50%	58%
VRN11(1L)	90%	58%

- ✓ WACC 10%, FCF to Sales 48%
- ✓ PFS for second-line therapy equals 0.5x~0.6x that of first-line therapy
- ✓ *58%(~2034), 88%(~2038), 92%(~2042), 100%(~2050)

VRN11 for EGFR mutation NSCLC

- ✓ Estimated rNPV ~USD 37,336mn if positioned as a 1st- 2nd-line EGFR treatment including in the brain metastasis patient population

Brain metastasis, BM; 1 USD = 1,527.30 KRW
Source: BIO, Biomedtracker, Amplion, Clinical Development Success Rates 2006-2015, 2016.6; Schulze U, Ringel M. *Nat Rev Drug Discov.* 2013;12(6):419-420.
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PEER Valuation

Nuvalent

	Nuvalent	VORONOI
Market Cap (Jun 30, 2026)	USD 9.8B	USD 2.6B
Addressable Market	ALK (3~5% of NSCLC)	EGFR (30~40% of NSCLC)

Relative Value Assessment

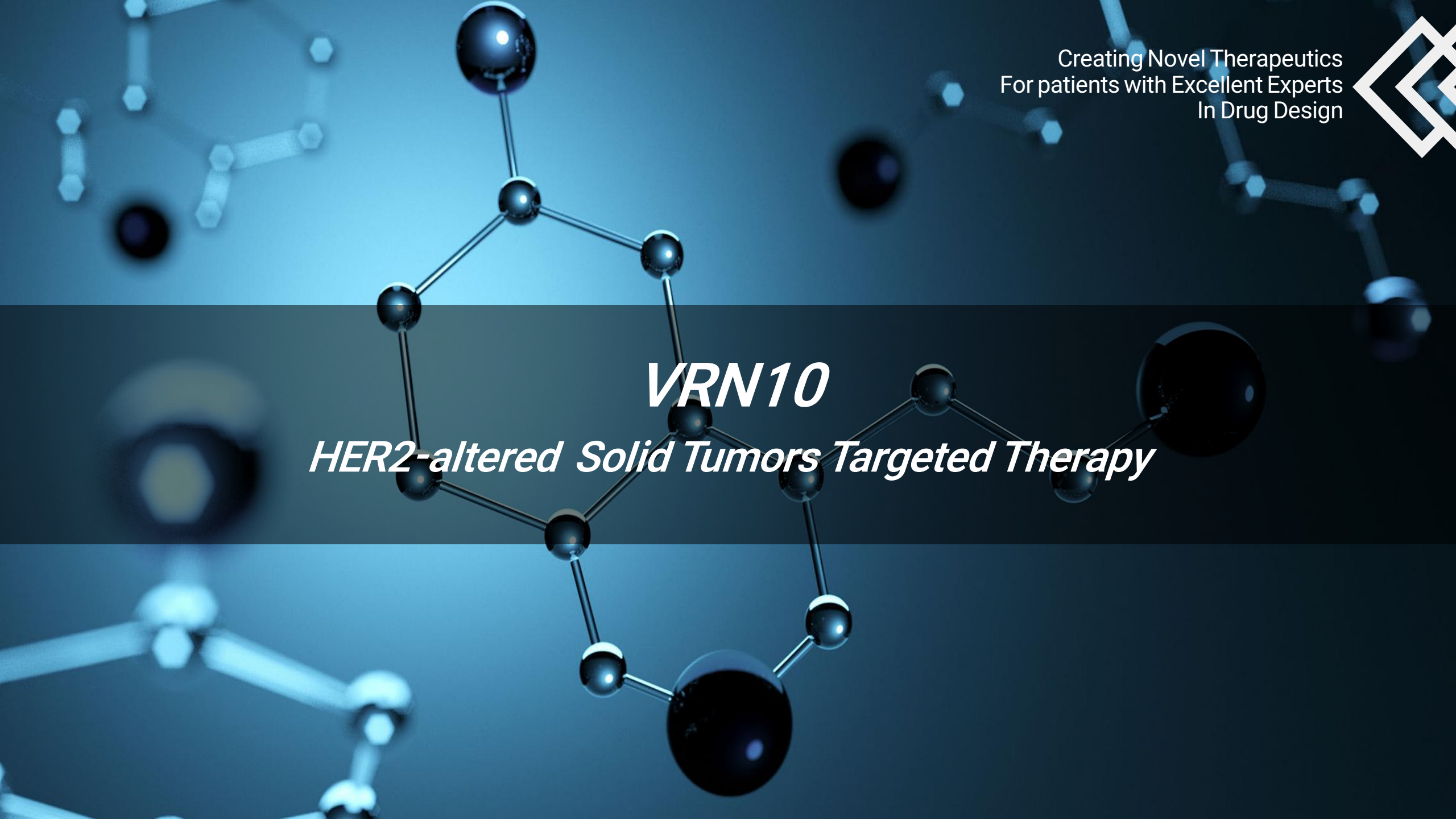
- Nuvalent's ALK-targeted therapy NVL-655: Based on phase 1/2 data, currently valued at $9.8B in the U.S. (as of Jun 30, 2026)
- June 2026: GSK acquired Nuvalent for $10.6B
- Mutation prevalence: ALK ~3–5% of NSCLC vs EGFR ~30–40% of NSCLC
- EGFR inhibitor market size (vs ALK) suggests 6-10x upside potentials

Revolution Medicines

Relative Value Assessment

- Revolution Medicines' RAS-targeted therapy Daraxonrasib: Acquisition discussion with Merck (Jan 2026)
- Estimated company value: $28–32B
- Market cap growth: $8.86B(2025 3Q) → $39.82B(2026 2Q)

USD 1 = KRW 1,527.30



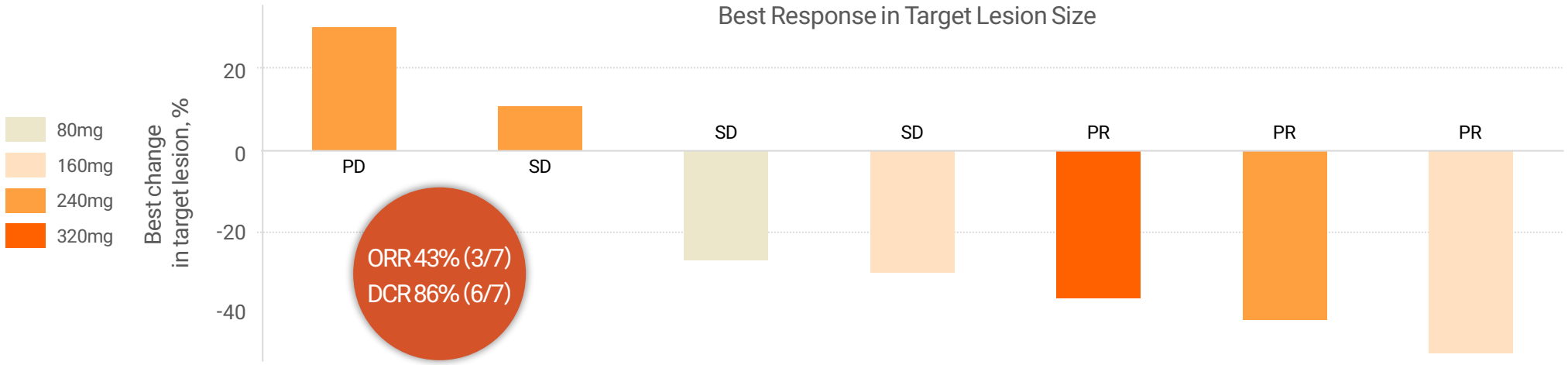
Creating Novel Therapeutics
For patients with Excellent Experts
In Drug Design

VRN10

HER2-altered Solid Tumors Targeted Therapy

Efficacy(1): HER2 mutant solid tumors

Antitumor activity confirmed in heavily pretreated HER2-mutant solid tumor patients treated with VRN10



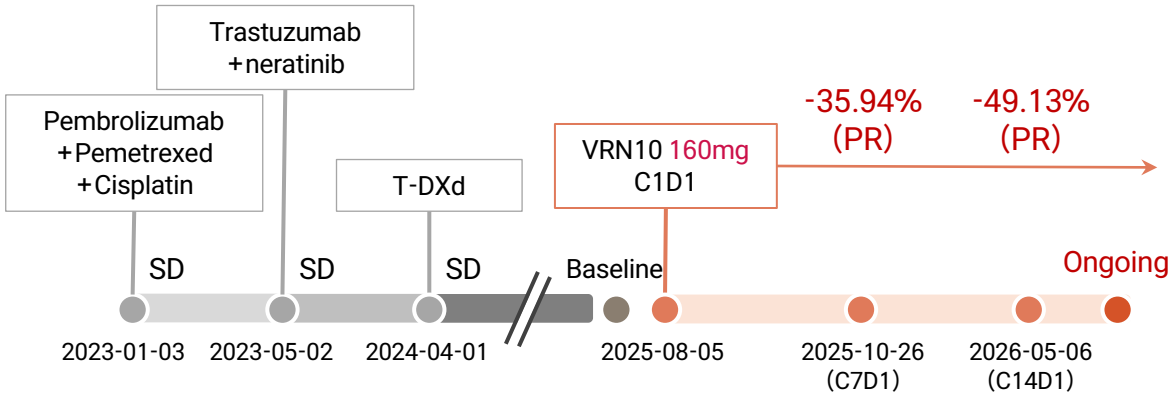
Patient #	007-002	009-004	001-001	004-001	009-006	008-001	004-003
HER2 (status)	R678Q	G727A	S310F	V777L	L755P	V659E	S310Y
Primary tumor site	Bladder	Urothelial	Pancreas	Breast	Lung	Renal	Lung
No. prior Systemic Tx	2	4	3	7	3	2	3
Prior HER2 TKI	N	N	Y	N	N	N	Y
Prior T-DXd	N	N	N	Y	Y	N	Y

S310F / S310Y: Extracellular domain, V659E: Transmembrane domain , R678Q: Juxtamembrane domain, G727A / L755P / V777L: Kinase domain
ORR, Objective Response Rate; DCR, Disease Control Rate

Case report：160 mg, HER2 S310Y lung adenocarcinoma

Durable partial response ongoing at 9+ months, with complete resolution of lung and breast lesions even after three prior regimens including chemotherapy

Patient 004-003(HER2 S310Y)：56-yr female following progression on T-DXd

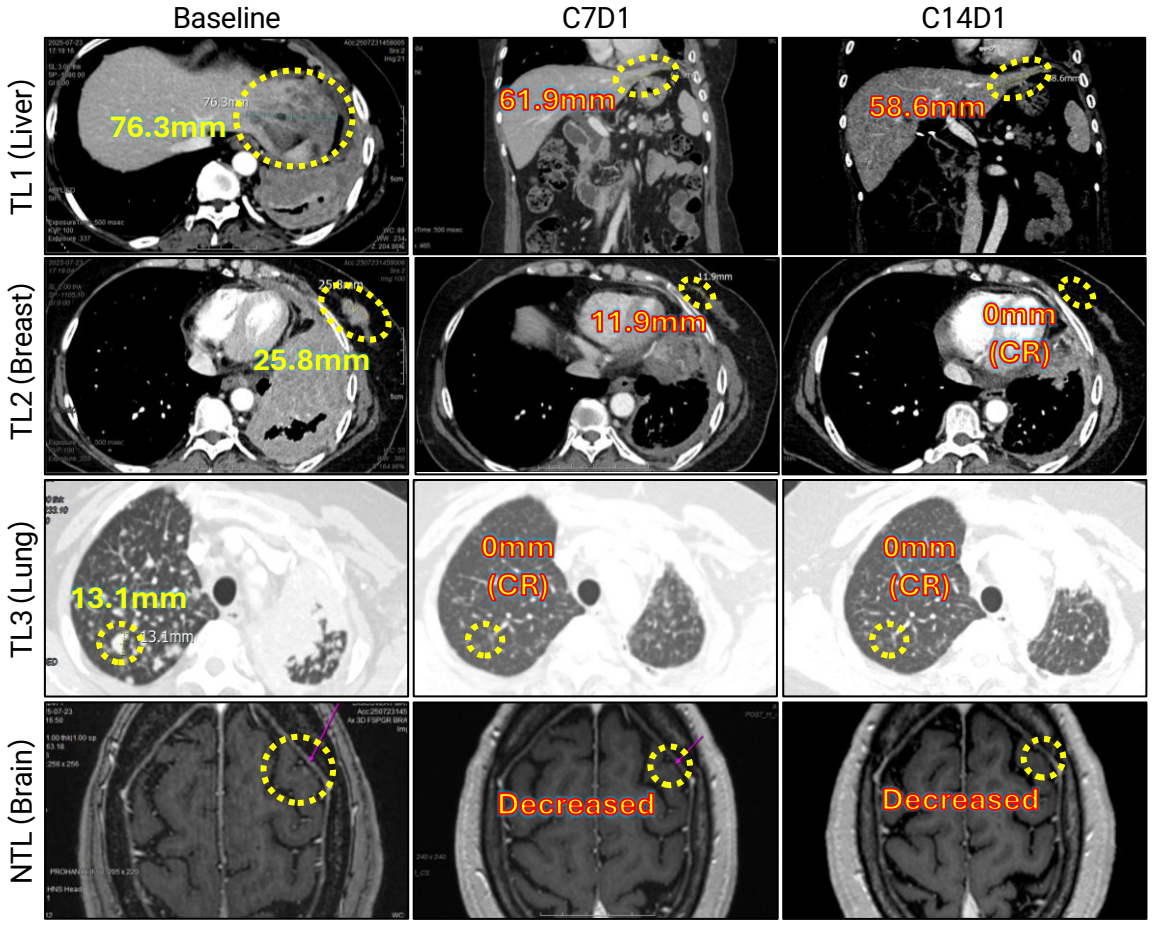


Baseline and Treatment History

- Metastatic site：Lung, lymph nodes, brain
- Disease progression after **T-DXd** treatment (~1yr)

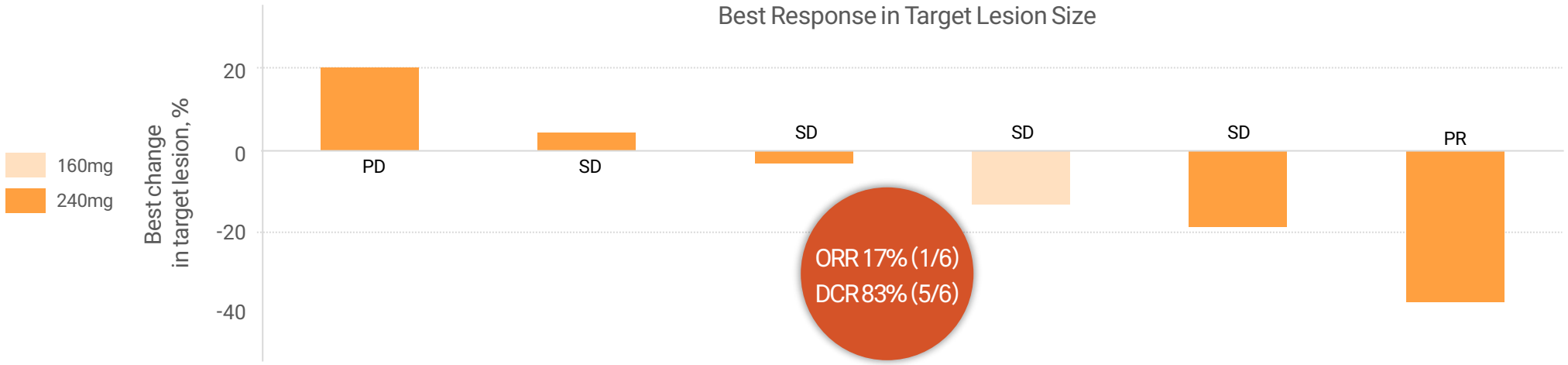
VRN10 Treatment

- Best response：**PR**
- **CR** was achieved in **2 target lesions**：lung at C3D1 and breast at C14D1
- **Brain** non-target lesion(NTL) was **decreased**
- Duration of treatment：**10 months (Ongoing)**



Efficacy(2): HER2-positive metastatic Breast Cancer

83% Disease control rate (DCR) confirmed in T-DXd-pretreated, HER2-positive breast cancer patients



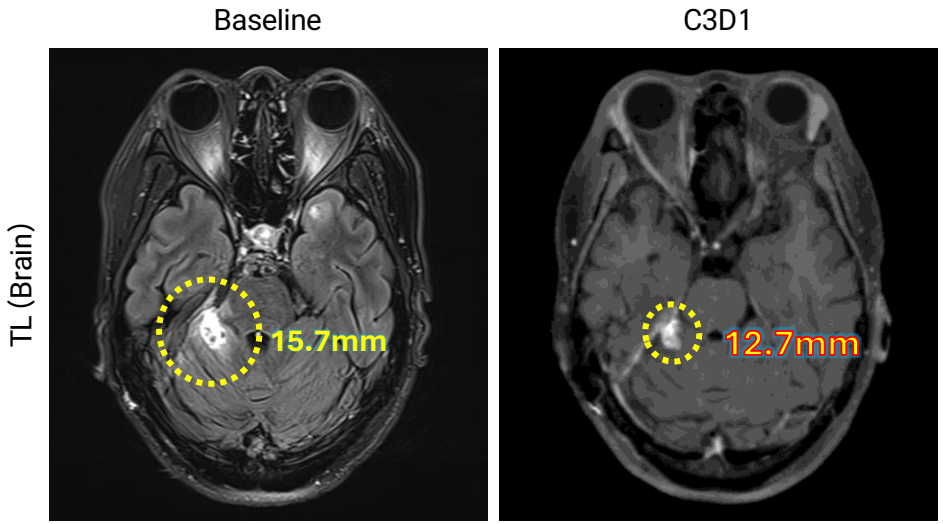
Patient #	005-001	006-003	003-001	005-002	003-003	006-001
HER2 (status)	IHC 3+	IHC 2+ ISH+	IHC 3+	IHC 3+	IHC 2+ ISH+	IHC 3+
Primary tumor site	Breast	Breast	Breast	Breast	Breast	Breast
No. prior Systemic Tx	3	6	10	8	2	3
Prior HER2 TKI	N	N	Y	Y	N	N
Prior T-DXd	Y	Y	Y	Y	Y	Y

KR005-002 Target lesion: Brain
ORR, Objective Response Rate; DCR, Disease Control Rate

Case report: 160 mg, HER2 positive Breast cancer

Stabilized disease despite progression on numerous previous treatments including radiation and systemic HER2 targeted therapy

Patient 005-002: 37-yr female patient with HER2-positive breast cancer (resected) and CNS metastases following progression on T-DXd



Baseline and Treatment History

- Metastatic site: Brain
- Early HER2 therapy + surgery (Complete resection of primary tumor)
- Radiation + systemic HER2 targeted therapy
- Tucatinib → **T-DXd** → Progression

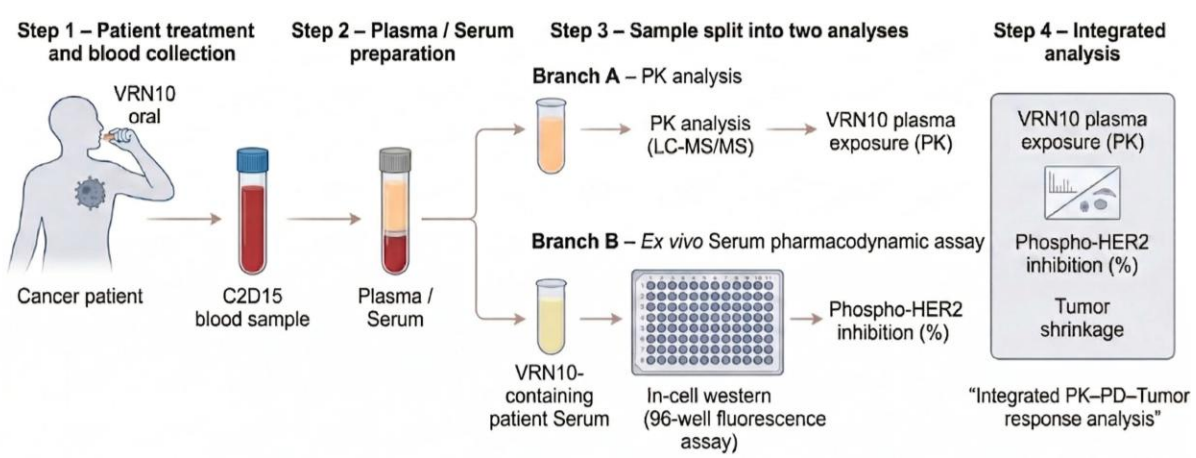
VRN10 Treatment

- Best response: **SD**
- **Reduction** in **Brain** target lesions and non-target lesion was stable
- Duration of treatment: **6 months (Ongoing)**

Tumor responses observed in HER2-positive and HER2-mutant patients

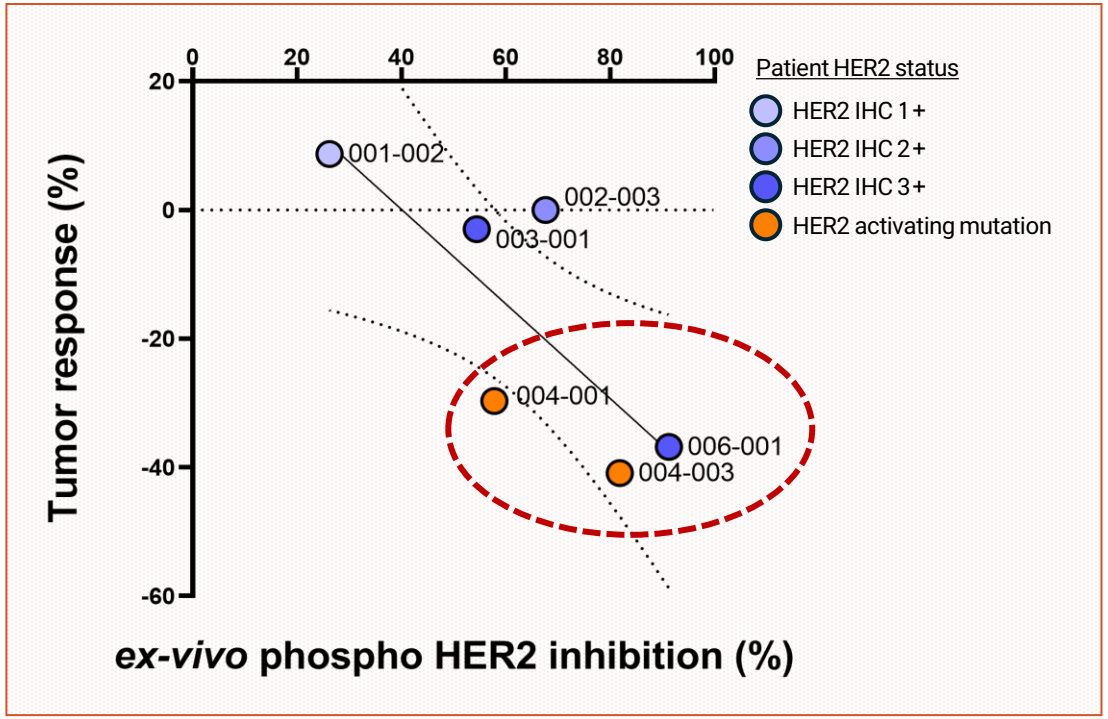
Antitumor activity confirmed in patients with HER2 overexpression and mutations in the Phase 1a study

Integrated PK-PD-Tumor Response Analysis



- ✓ Ex-vivo Serum Assay: Serum samples collected following VRN10 administration were utilized to assess pHER2 inhibitory activity in tumor cells
- ✓ Integrated Analysis: An integrated analysis of patient PK, PD, and tumor shrinkage established a causal relationship underlying therapeutic efficacy

Tumor response(ex-vivo correlation)

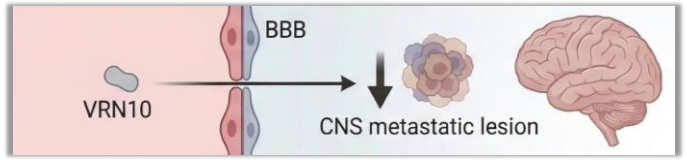


- ✓ A linear correlation was confirmed between the degree of pHER2 inhibition and tumor size reduction
- ✓ Robust pHER2 inhibition and corresponding tumor regression observed in patients with both HER2 overexpression and mutations

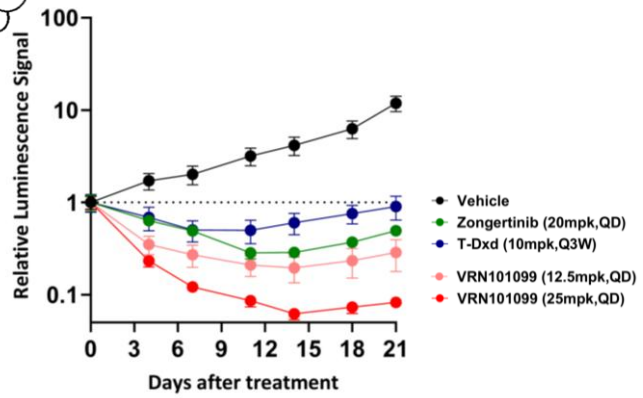
Efficacy(3)：Brain penetration

Potential to address unmet medical needs for brain metastasis patients

VRN10：Addressing unmet needs in brain mets

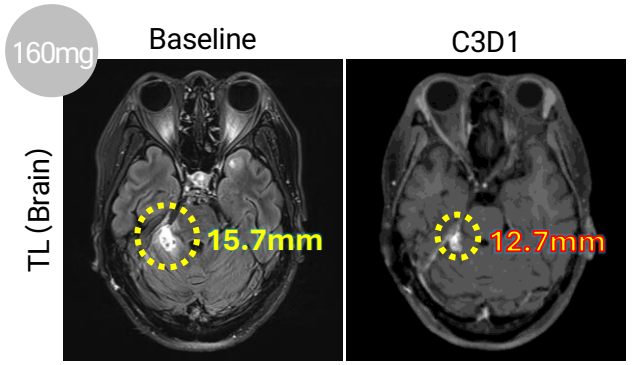


Intracranial model(BT474, HER2+ Breast Cancer)



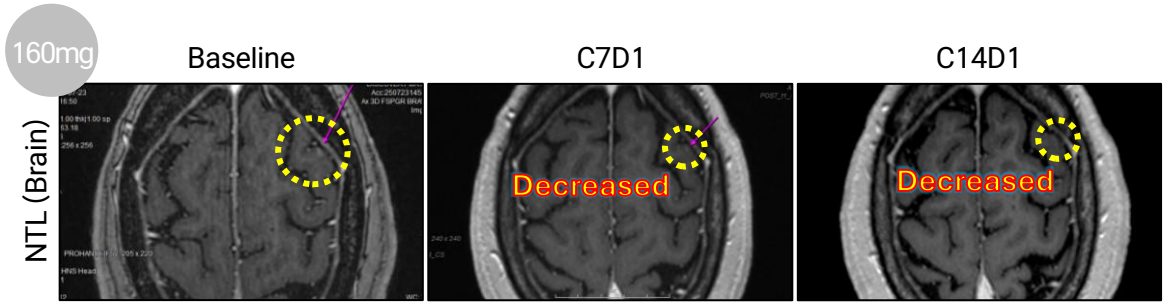
✓ **Brain Penetration**：Intracranial efficacy confirmed based on superior BBB penetration vs. competitors

Intracranial disease control confirmed in phase 1a for patients with brain metastases



CNS control in BM patients	
Patients with target lesion	1 intra SD with tumor reduction, 1 intra PD
Patients with non-target lesion	2 Non-CR/non-PD (1 with reduction)
Intracranial DCR	75% (3/4)

✓ 3/4 (75%) Treatment ongoing

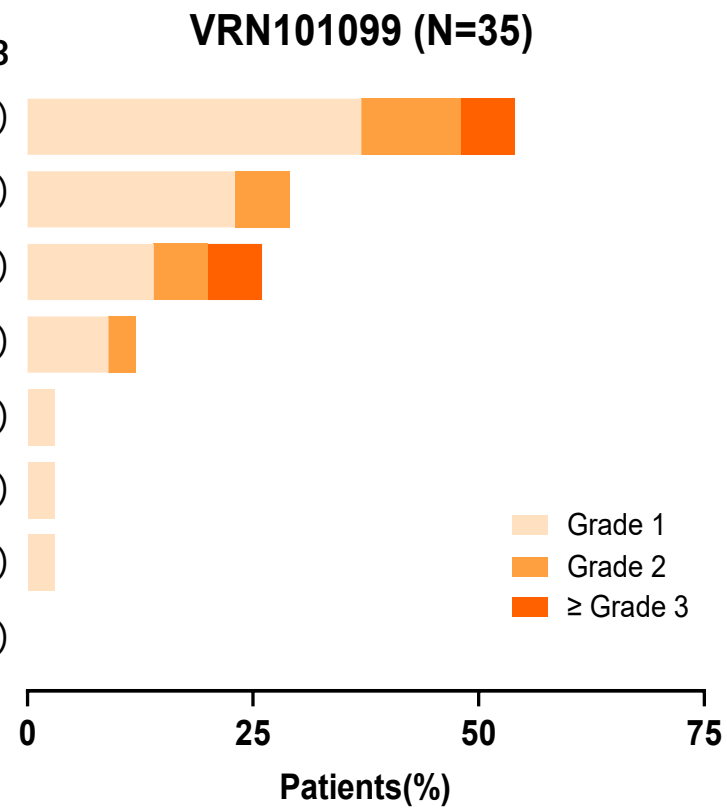


mDOT：median duration of treatment

Safety and tolerability outcomes in dose escalation

Favorable safety profile confirmed across all dose cohorts to date

TRAE, n (%)	All	G≥3
Any	19 (54)	2 (6)
Nausea	8 (23)	0 (0)
Diarrhea	9 (26)	2 (6)
Rash	4 (11)	0 (0)
Dry skin	1 (3)	0 (0)
Pruritus	1 (3)	0 (0)
AST increased	1 (3)	0 (0)
ALT increased	0 (0)	0 (0)



- ✓ Overall, the safety profile was **tolerable** and **manageable** across all dose levels.
- ✓ One drug-related Grade 1 AST increased was observed in a one patient at 160 mg; notably, no hepatotoxicity was reported at higher dose levels.
- ✓ Diarrhea was effectively managed with loperamide administered on an as-needed basis upon symptom onset
- ✓ Escalation to 480 mg is ongoing, with MTD not reached and RP2D determination ongoing.
- ✓ Ph1a results to date are supportive of further clinical development starting with a Ph1b dose expansion study

Abbreviations: adverse event; TRAE; AE, adverse event; AST, Aspartate Aminotransferase; ALT, Alanine Aminotransferase.
Combined term, includes rash, rash maculopapular, exfoliative rash.

Phase 1 clinical trial design

Ongoing global phase 1 trial in South Korea and Australia, with first patient dosed in Q1 2025
Monotherapy trial and combination therapy trial is planned in phase 1b onward

Phase 1a Highlights

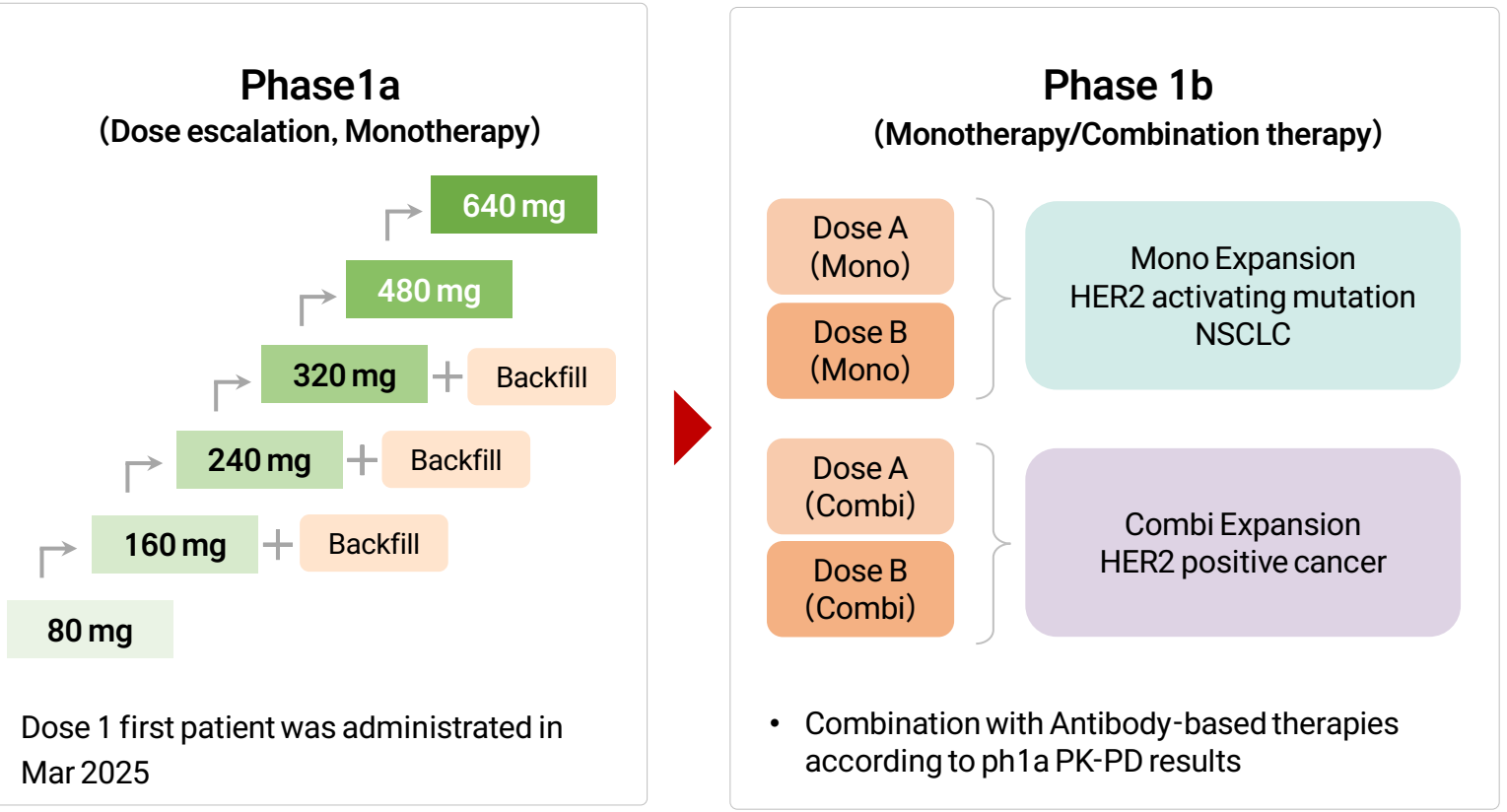
Key Eligibility Criteria

- HER2-altered solid tumors
- HER2 positive mBC or mGC with PD after prior anti-HER2 therapy
- Other HER2-altered solid tumors with not approved HER2-targeted SoC, progressed on all available therapies
- NSCLC with HER2-activating mutations after prior HER2 TKI or ADC.

Dose Escalation

- Standard "3+3" dose escalation
- Minimum of 18 and up to 72 pts, plus up to 36 additional backfill pts
- DLT assessment: first cycle of treatment (i.e. Cycle 1, 21 days of IP)

Phase 1 clinical trial design



- Dose 1 first patient was administrated in Mar 2025
- Combination with Antibody-based therapies according to ph1a PK-PD results

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In Drug Design

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Thank You