Nuvation Bio

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July 2024

Forward looking statements

Certain statements included in this presentation (this "Presentation") that are not historical facts are forward-looking statements for purposes of the safe harbor provisions under the United States Private Securities Litigation Reform Act of 1995. Forward-looking statements are sometimes accompanied by words such as "believe," "may," "will," "estimate," "continue," "anticipate," "intend," "expect," "should," "would," "plan," "predict," "potential," "seem," "seek," "future," "outlook" and similar expressions that predict or indicate future events or trends or that are not statements of historical matters. These forward-looking statements include, but are not limited to, our expectations regarding a U.S. NDA, the potential for taletrectinib to become a new therapeutic option for ROS1-positive NSCLC, taletrectinib's and safusidenib's best-in-class therapeutic potential, the expected timing for sharing results from the TRUST-I and TRUST-II studies, statements regarding Nuvation Bio's expected timing of becoming a commercial organization, potential therapeutic benefit of Nuvation Bio's product candidates and advancement of clinical studies for such product candidates, sufficiency of Nuvation Bio's current cash balance to support operations in the near term, clinical study design, or the potential of the DDC platform. These statements are based on various assumptions, whether or not identified in this Presentation, and on the current expectations of the management team of Nuvation Bio and are not predictions of actual performance. These forward-looking statements are subject to a number of risks and uncertainties that may cause actual results to differ from those anticipated by the forward-looking statements, including but not limited to the challenges associated with conducting drug discovery and initiating or conducting clinical studies due to, among other things, difficulties or delays in the regulatory process, enrolling subjects or manufacturing or acquiring necessary products; the emergence or worsening of adverse events or other undesirable side effects; risks associated with preliminary and interim data, which may not be representative of more mature data; and competitive developments. Risks and uncertainties facing Nuvation Bio are described more fully in its Form 10-Q filed with the SEC on May 14, 2024 under the heading "Risk Factors," and other documents that Nuvation Bio has filed or will file with the SEC. You are cautioned not to place undue reliance on the forward-looking statements, which speak only as of the date of this Presentation. Nuvation Bio disclaims any obligation or undertaking to update, supplement or revise any forward-looking statements contained in this Presentation.

Nuvation Bio is tackling some of the greatest unmet needs in oncology

Late clinical-stage, global oncology company with multiple candidates in development aimed at improving validated mechanisms that have encountered safety liabilities or limitations in efficacy

Taletrectinib[1], which is currently **completing two pivotal studies,** is a next-generation**,** potentially best-in-class **ROS1 inhibitor differentiated by response rate, duration of response, and tolerability**

Safusidenib is a potentially best-in-class, brain penetrant, **mIDH1 inhibitor** in **Phase 2 development** for the treatment of patients with **diffuse IDH1-mutant glioma**

NUV-868 is a **BD2-selective BET inhibitor** being evaluated in **Phase 1b** combination studies; **NUV-1511** is the Company's **first clinical-stage drug-drug conjugate (DDC)** being evaluated in a **Phase 1** study

Robust cash balance of $597 million and **newly acquired late-stage candidate** position Nuvation Bio to potentially become a **commercial stage organization in 2025**

1. Taletrectinib has been granted Orphan Drug Designation from the U.S. Food and Drug Administration (FDA) for the treatment of patients with ROS1-positive non-small cell lung cancer (NSCLC) and Breakthrough Therapy Designations by both the U.S. FDA and China's National Medical Products Administration (NMPA) for the treatment of patients with advanced or metastatic ROS1-positive NSCLC.

Nuvation Bio is developing a broad pipeline of differentiated and novel therapeutic candidates

Program	Potential Indication(s)		Current Stage of Development	Anticipated Milestones & Recent Updates
Taletrectinib[1] (ROS1)	Advanced ROS1-positive NSCLC		*Completing two Phase 2 pivotal studies (TRUST-I & TRUST-II)*	Pooled pivotal data to be presented at ESMO in Sept. 2024; TRUST-II data to be presented at WCLC in Sept. 2024; TRUST-I data published in JCO in June 2024; China NDAs under priority review by China's NMPA[5]
Safusidenib[2] (mIDH1)	Diffuse IDH1-mutant glioma		Phase 2	Phase 2 study ongoing
NUV-868 (BET)	Advanced solid tumors	Monotherapy	Phase 1	Maximum tolerated dose determined
	Advanced solid tumors[3]	NUV-868 + olaparib	Phase 1	Phase 1b dose escalation study ongoing
	mCRPC	NUV-868 + enzalutamide	Phase 1	Phase 1b dose escalation study ongoing
NUV-1511 (DDC)	Advanced solid tumors[4]		Phase 1	Phase 1 dose escalation study ongoing

BET: Bromodomain and Extra-Terminal motif; ESMO: European Society of Medical Oncology Congress; JCO: Journal of Clinical Oncology; mIDH1: mutant isocitrate dehydrogenase 1; ROS1: c-ros oncogene 1; WCLC: World Conference on Lung Cancer.
1. Taletrectinib has been granted Orphan Drug Designation from the U.S. FDA for the treatment of patients with ROS1-positive NSCLC and Breakthrough Therapy Designations by both the U.S. FDA and China's NMPA for the treatment of patients with advanced or metastatic ROS1-positive NSCLC; worldwide development and commercial rights in-licensed from Daiichi Sankyo; rights to taletrectinib have been out-licensed in China, Japan, and Korea. 2. Worldwide development and commercial rights in-licensed from Daiichi Sankyo, excluding Japan where Daiichi Sankyo retains development and commercial rights. 3. Includes patients with ovarian cancer, triple-negative breast cancer (TNBC), advanced pancreatic cancer, and metastatic castration resistant prostate cancer (mCRPC). 4. Includes patients with advanced solid tumors who previously received and progressed on or after treatment with Enhertu® and/or Trodelvy® per approved U.S. FDA labeling, human epidermal growth factor receptor 2-negative (HER2-) metastatic breast cancer, mCRPC, advanced pancreatic cancer, and platinum-resistant ovarian cancer. 5. Based on results of the TRUST-I clinical study, under priority review for the treatment of adult patients with locally advanced or metastatic ROS1-positive NSCLC who either have or have not previously been treated with ROS1 tyrosine kinase inhibitors (TKIs).

Taletrectinib | ROS1i

Advanced ROS1-positive NSCLC

Two Phase 2 pivotal studies ongoing

Taletrectinib is a next-generation, potentially best-in-class ROS1 inhibitor differentiated by response rate, duration, and tolerability

Commercial opportunity

- Breakthrough Therapy Designations (U.S. and China)[1]
- China 1L & 2L NDAs accepted and granted priority review[2]
- Sizeable population of patients with ROS1-positive NSCLC

Differentiated profile

- Potentially best-in-class efficacy and safety profile
- Highly brain penetrant
- Active against common mutations

Strong partnerships

- In-licensed from Daiichi Sankyo
- Maintain global rights except major Asian markets where rights have been out-licensed[3]

1. Taletrectinib has been granted Orphan Drug Designation from the U.S. FDA for the treatment of patients with ROS1-positive NSCLC and Breakthrough Therapy Designations by both the U.S. FDA and China's NMPA for the treatment of patients with advanced or metastatic ROS1-positive NSCLC. 2. Based on results of the TRUST-I clinical study, under priority review for the treatment of adult patients with locally advanced or metastatic ROS1-positive NSCLC who either have or have not previously been treated with ROS1 TKIs. 3. Worldwide development and commercial rights in-licensed from Daiichi Sankyo; rights to taletrectinib have been out-licensed in China, Japan, and Korea.

The ROS1-positive NSCLC market represents a sizeable commercial opportunity

Key takeaways

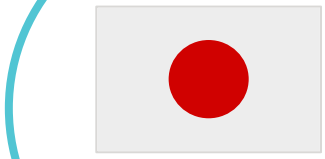
- NSCLC accounts for ~80-85%[1] of all lung cancers
- ROS1-positive lung cancer represents ~1-3%[2] of new NSCLC cases
- There are currently three therapies approved to treat patients with ROS1-positive NSCLC:

1st generation
- Crizotinib (Pfizer, approved 2016[3])
- Entrectinib (Roche, approved 2019[4])

2nd generation
- Repotrectinib (Bristol-Myers Squibb, approved 2023[5])

Estimated diagnosed patient population


~2,000-4,000[1,2]


~1,000-2,000[2,6]


~2,600-5,200[2,7]

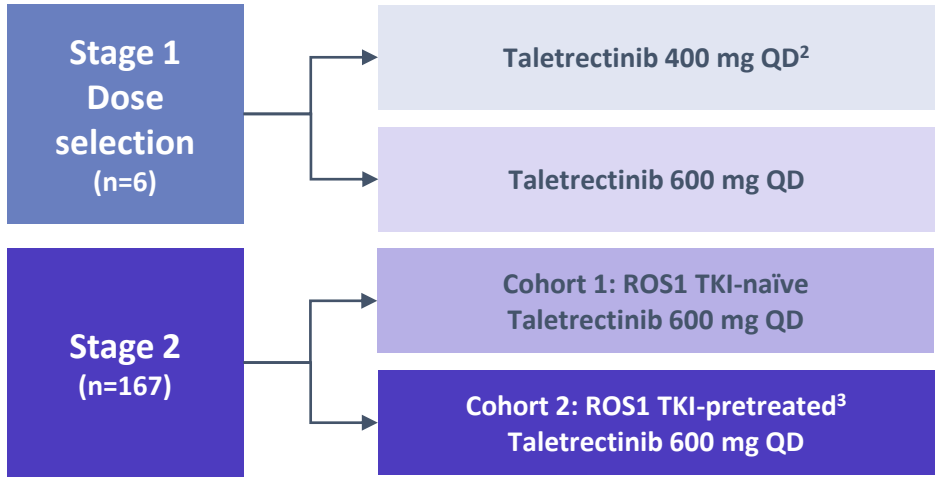

~16,500[8,9]

1. American Cancer Society (2024). 2. National Center for Biotechnology Information: Gendarme et al., Curr Oncol (2022). 3. Initially approved by U.S. FDA in 2011 for the treatment of patients with advanced or metastatic ALK-positive NSCLC; later approved in 2016 for the treatment of patients with metastatic ROS1-positive NSCLC. 4. Approved by U.S. FDA in 2019 for the treatment of patients with metastatic ROS1-positive NSCLC and the treatment of patients with neurotrophic tyrosine receptor kinase (NTRK) gene fusion without a known acquired resistance mutation. 5. Approved by U.S. FDA in 2023 for the treatment of patients with advanced or metastatic ROS1-positive NSCLC. 6. National Cancer Center Japan (2019). 7. European Cancer Information Systems (2021). 8. Gao et al., J Thorac Oncol (2020). 9. Zhang et al., Thorac Cancer (2019).

Taletrectinib is being studied in two pivotal registrational studies that have included >300 patients in total, with results supporting BTDs[1] in U.S. & China

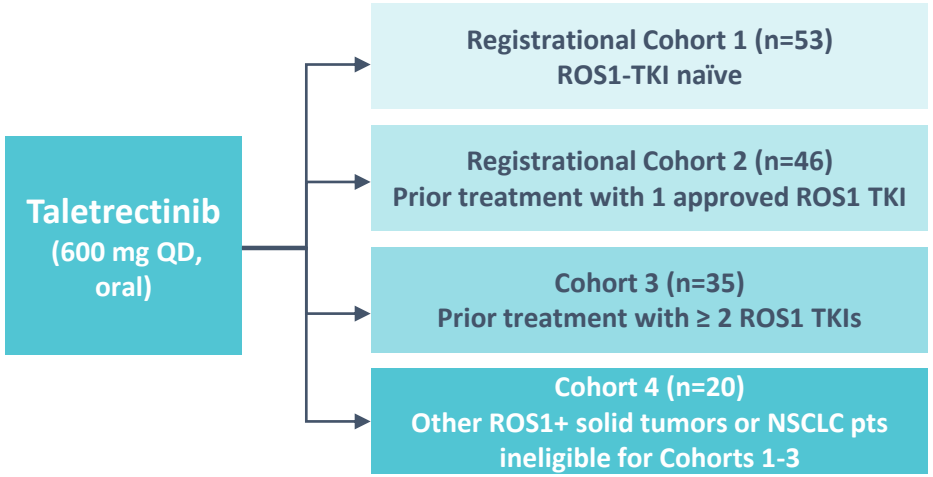
TRUST-I

Pivotal China Phase 2
n=173



TRUST-II

Pivotal Global Phase 2
n=154



- *Pooled TRUST-I & TRUST-II data to be presented in a poster presentation at ESMO in September 2024, which will support Nuvation Bio's NDA in the U.S.*
- *TRUST-II data to be presented in an oral presentation at WCLC in September 2024[4]*
- *TRUST-I data were presented in an oral presentation at ASCO and published in the Journal of Clinical Oncology in June 2024[5]*

ASCO: American Society of Clinical Oncology; ESMO: European Society of Medical Oncology Congress; NDA: New Drug Application; WCLC: World Conference on Lung Cancer. 1. Taletrectinib has been granted Orphan Drug Designation from the U.S. FDA for the treatment of patients with ROS1-positive NSCLC and Breakthrough Therapy Designations by both the U.S. FDA and China's NMPA for the treatment of patients with advanced or metastatic ROS1-positive NSCLC. 2. Three patients treated at the 400 mg dose level, two of which escalated to 600 mg. 3. Includes patients who had previously been treated with crizotinib. 4. Previous interim update was presented at ESMO 2023 (Perol et al., ESMO 2023 presentation). 5. Zhou et al., *Journal of Clinical Oncology*, 2024 and Li et al., ASCO Presentation, 2024.

TRUST-I data published in the *Journal of Clinical Oncology* and presented at ASCO includes efficacy data and long-term follow up from 173 patients

Key eligibility criteria

Inclusion Criteria:

- Locally advanced or metastatic NSCLC
- Age ≥18 years
- ECOG PS 0-1
- Evidence of ROS1 fusion in tumor tissue

Cohort A: ROS1 TKI-naïve
Taletrectinib 600 mg QD[1]

Cohort B: ROS1 TKI-pretreated[2]
Taletrectinib 600 mg QD[1]

Endpoints

Primary:

- IRC-assessed cORR per RECIST v1.1

Secondary:

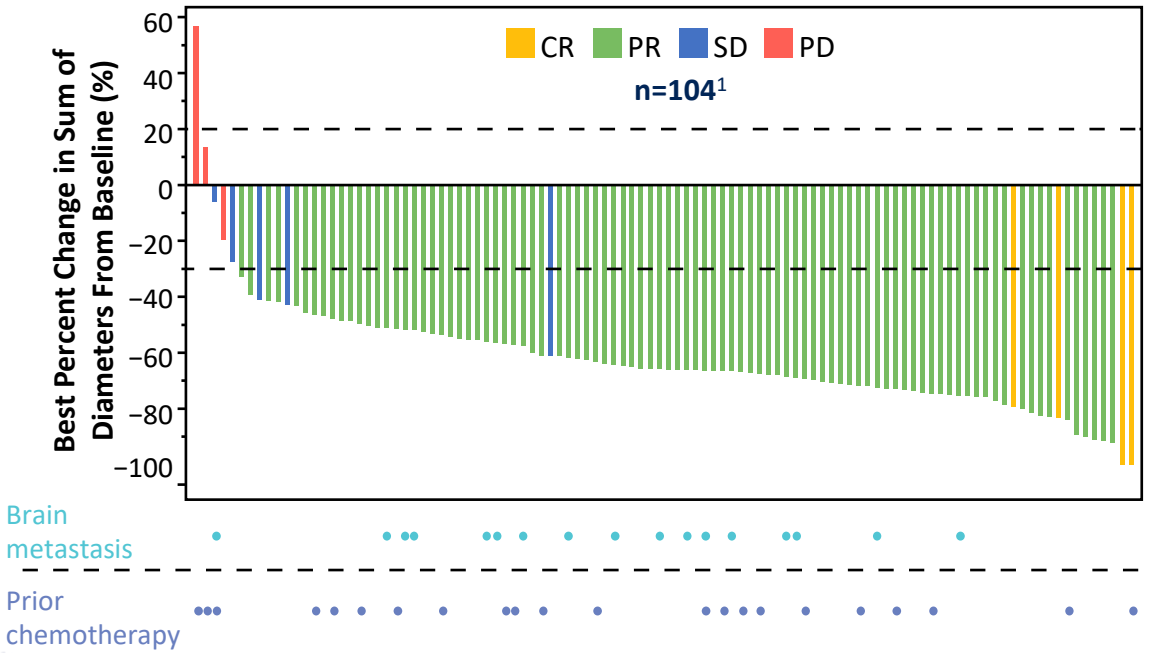
- DOR
- IC-ORR
- BOR
- DCR
- TTR
- PFS
- Safety[3]

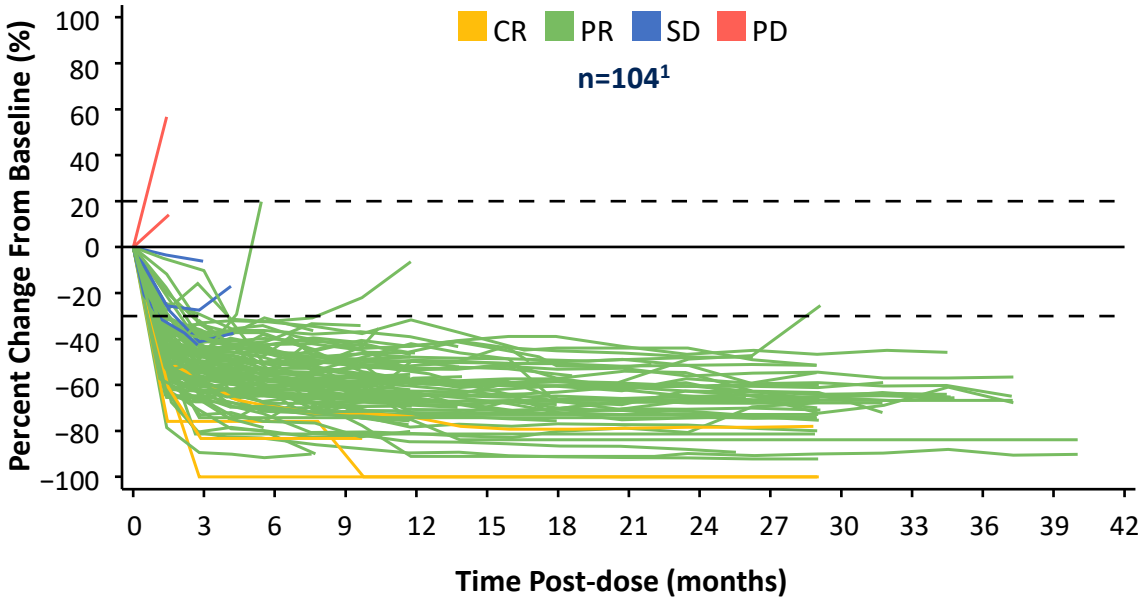
Category	TKI-naïve n=106	TKI-pretreated[2] n=67	Overall n=173
Median age, years (range)	56.0 (26–78)	51.0 (31–77)	55.0 (26–78)
Female, n (%)	59 (55.7)	41 (61.2)	100 (57.8)
ECOG PS 0/1, n (%)	20 (18.9)/86 (81.1)	19 (28.4)/48 (71.6)	39 (22.5)/134 (77.5)
Stage IV disease	97 (91.5)	65 (97.0)	162 (93.6)
Prior anticancer chemotherapy, n (%)	22 (20.8)	23 (34.3)	45 (26.0)
Never smoker	78 (73.6)	49 (73.1)	127 (73.4)
Brain metastasis, n (%)	18 (17.0)	28 (41.8)	46 (26.6)

Source: Zhou et al., *Journal of Clinical Oncology*, 2024 and Li et al., ASCO Presentation, 2024. BOR: Best overall response; cORR: confirmed Objective response rate; DCR: Disease control rate; DOR: Duration of response; ECOG PS: Eastern Cooperative Oncology Group performance status; IC: Intracranial; IRC: Independent review committee; PFS: Progression-free survival; RECIST: Response Evaluation Criteria in Solid Tumors; TKI: Tyrosine kinase inhibitor; TTR: Time to response. 1. Dose confirmation lead-in stage evaluated the safety of taletrectinib at dose levels of 400 mg QD (n=3) and 600 mg QD (n=3). Two of the three patients dosed at 400 mg escalated to 600 mg. 2. Includes patients who had previously been treated with crizotinib. 3. Safety was analyzed in patients receiving ≥1 dose of taletrectinib until 30 days after the last dose of taletrectinib or the start date of new anticancer therapy minus 1 day, whichever occurred first.

Tumors shrank in 91% of taletrectinib-treated patients with advanced ROS1-positive NSCLC who were TKI-naïve

Responses	TKI naïve (n=106)[1]
IRC-assessed cORR, % (95% CI)	90.6 (83.33, 95.38)
DCR, % (95% CI)	95.3 (89.33, 98.45)
Median TTR, months (95% CI)	1.4 (1.38, 1.41)

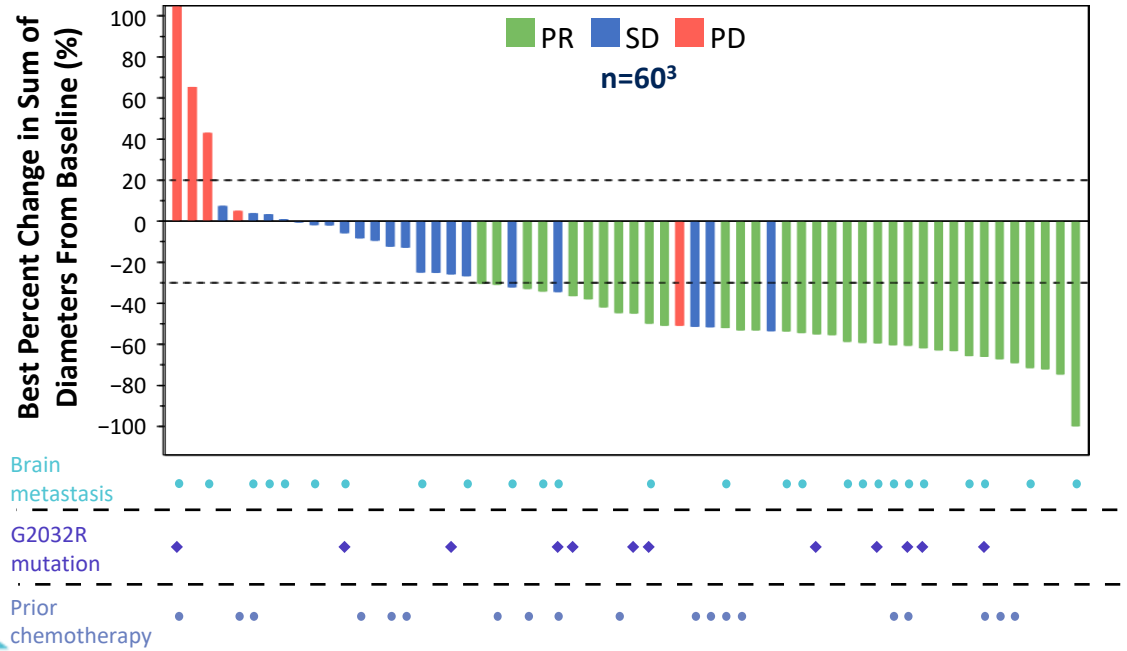


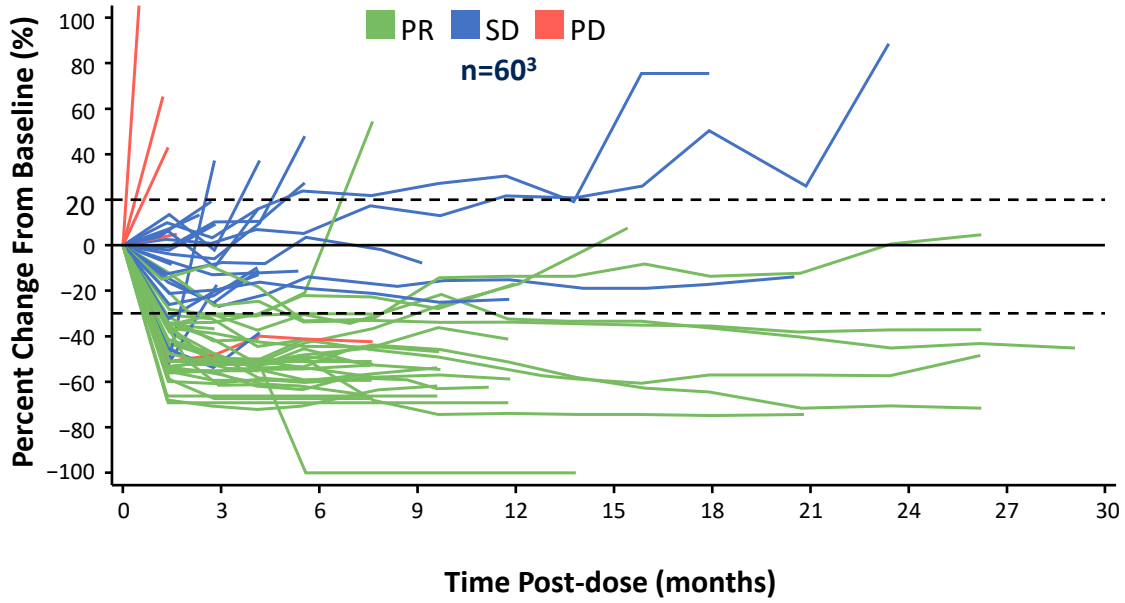


Source: Zhou et al., *Journal of Clinical Oncology*, 2024 and Li et al., ASCO Presentation, 2024. TRUST-I study data cutoff: November 29, 2023. BOR: Best overall response; cORR: confirmed Objective response rate; CR: Complete response; DCR: Disease control rate; IRC: Independent review committee; PD: Progressive disease; PR: Partial response; SD: Stable disease; TKI: Tyrosine kinase inhibitor; TTR: Time to response. 1. Two patients with confirmed BOR as not evaluable are not displayed in the waterfall and spider plots.

Tumors shrank in 52% of taletrectinib-treated patients with advanced ROS1-positive NSCLC who were previously treated with a ROS1 TKI[1]

Responses	TKI-pretreated[1] (n=66)[2]
IRC-assessed cORR, % (95% CI)	51.5 (38.88, 64.01)
DCR, % (95% CI)	83.3 (72.13, 91.38)
Median TTR, months (95% CI)	1.4 (1.38, 1.41)
cORR: G2032R mutations, % (n/N)	66.7 (8/12)





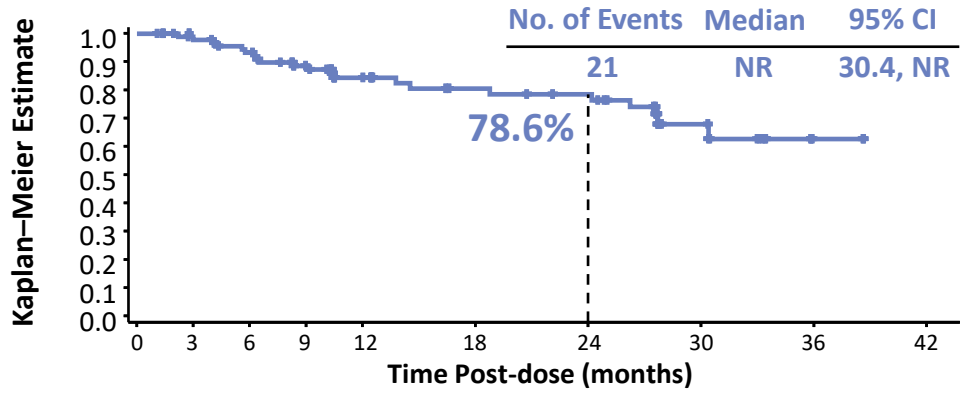
Source: Zhou et al., *Journal of Clinical Oncology*, 2024 and Li et al., ASCO Presentation, 2024. TRUST-I study data cutoff: November 29, 2023. BOR: Best overall response; cORR: confirmed Objective response rate; DCR: Disease control rate; IRC: Independent review committee; PD: Progressive disease; PR: Partial response; SD: Stable disease; TKI: Tyrosine kinase inhibitor; TTR: Time to response. 1. Includes patients who had previously been treated with crizotinib. 2. One patient was excluded from the response-evaluable population due to the presence of secondary cancer. 3. Six patients with confirmed BOR as not evaluable are not displayed in the waterfall and spider plots.

Median duration of response and progression free survival were not reached after median follow-up of 23.5 months in TKI-naïve patients

TKI-naïve (n=106)

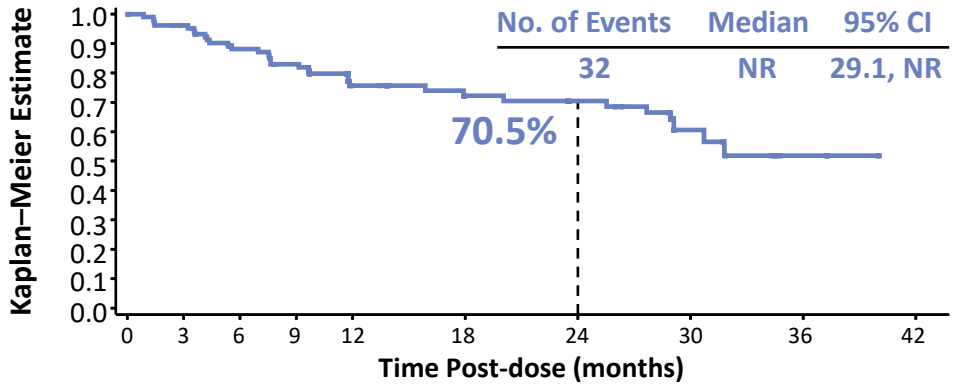
Median follow-up: 23.5 months[1]

DOR



Number at risk 96 89 82 70 50 40 37 14 2

PFS

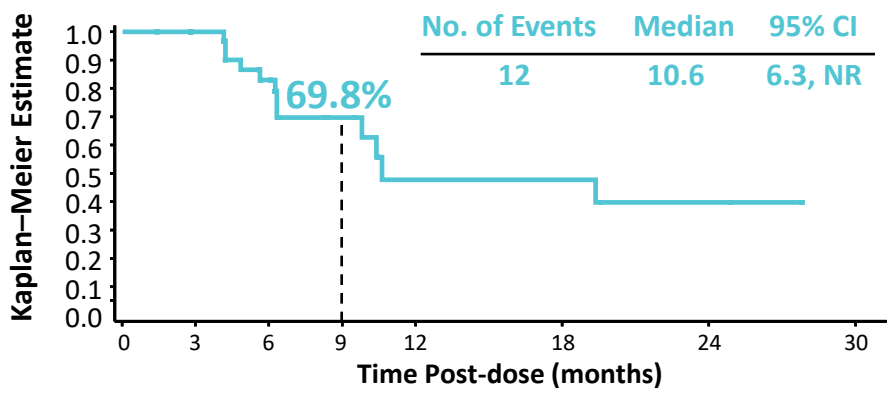


Number at risk 106 98 85 79 50 40 37 15 5

TKI-pretreated[2] (n=66)[3]

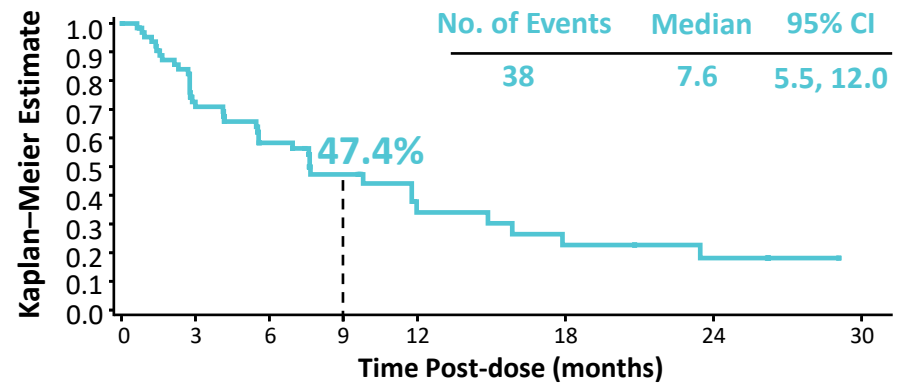
Median follow-up: 9.7 months[1]

DOR



Number at risk 34 31 22 11 6 6 4

PFS

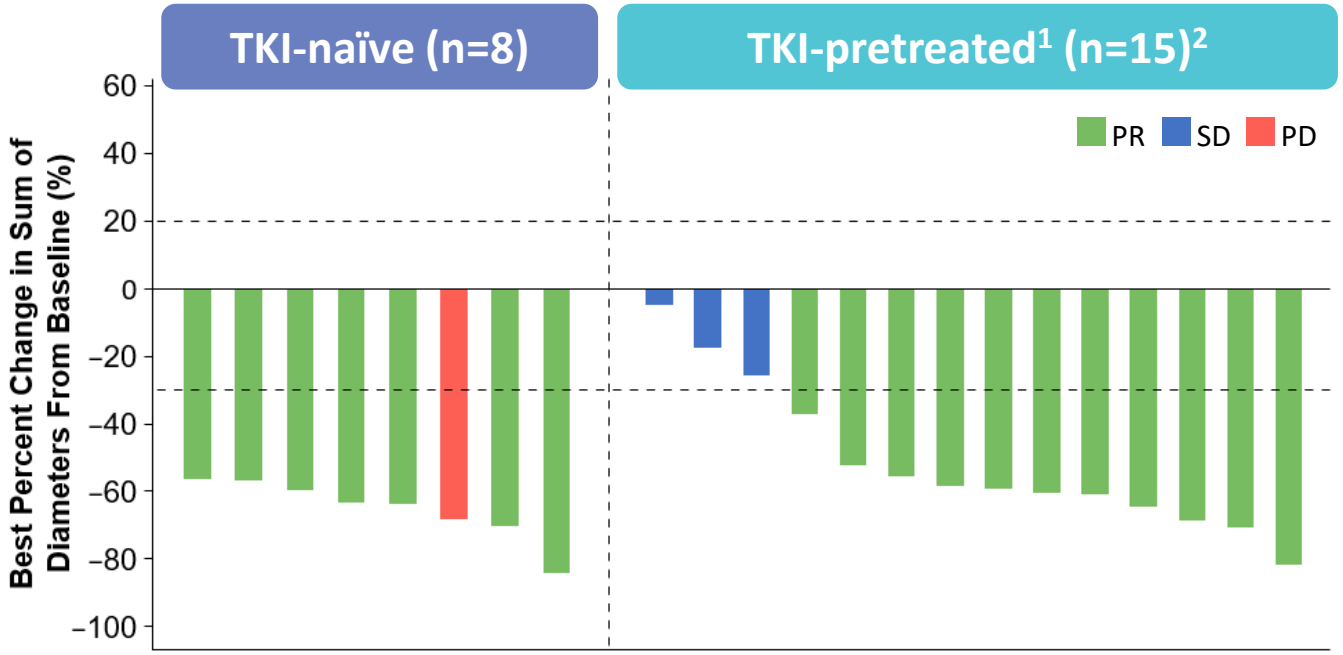


Number at risk 66 42 30 18 9 6 4

Source: Zhou et al., Journal of Clinical Oncology, 2024 and Li et al., ASCO Presentation, 2024. TRUST-I study data cutoff: November 29, 2023. DOR: Duration of response; NR: Not reached; PFS: Progression-free survival; TKI: Tyrosine kinase inhibitor. 1. Represents median follow-up for PFS; median follow-up for DOR was 22.1 months and 8.4 months in TKI-naïve and TKI-pretreated patient groups, respectively. Median DOR and PFS data are still maturing and may change as results from future data cut-off dates are disclosed. 2. Includes patients who had previously been treated with crizotinib. 3. One patient was excluded from the response-evaluable population due to the presence of secondary cancer.

Brain tumors shrank in 88% and 73% of taletrectinib-treated patients with measurable CNS tumors who were TKI-naïve and TKI-pretreated, respectively

Responses	TKI-naïve n=8	TKI-pretreated[1] n=15
IC-cORR, % (95% CI)	87.5 (47.35, 99.68)	73.3 (44.90, 92.21)
DCR, % (95% CI)	100.0 (63.06, 100.0)	93.3 (68.05, 99.83)



Source: Zhou et al., *Journal of Clinical Oncology*, 2024 and Li et al., ASCO Presentation, 2024. TRUST-I study data cutoff: November 29, 2023. CNS: Central nervous system; cORR: confirmed Objective response rate; DCR: Disease control rate; IC: Intracranial; IRC: Independent review committee; PD: Progressive disease; PR: Partial response; SD: Stable disease; TKI: Tyrosine kinase inhibitor. 1. Includes patients who had previously been treated with crizotinib. 2. One patient with confirmed best overall response as not evaluable is not displayed in the waterfall plot.

Taletrectinib's safety profile was consistent with previously reported data; only 5% of patients had a TEAE leading to treatment discontinuation

	Grade 1; n (%)	Grade 2; n (%)	Grade 3; n (%)	Grade 4; n (%)	Grade 5[1]; n (%)	Any grade; n (%)
Increased AST	92 (53.2)	26 (15.0)	14 (8.1)	0	0	132 (76.3)
Diarrhea	99 (57.2)	16 (9.2)	6 (3.5)	0	0	121 (69.9)
Increased ALT	79 (45.7)	29 (16.8)	8 (4.6)	1 (0.6)	0	117 (67.6)
Vomiting	75 (43.4)	16 (9.2)	1 (0.6)	0	0	92 (53.2)
Anemia	52 (30.1)	30 (17.3)	3 (1.7)	0	0	85 (49.1)
Nausea	64 (37.0)	8 (4.6)	1 (0.6)	0	0	73 (42.2)
Decreased neutrophil count	25 (14.5)	10 (5.8)	6 (3.5)	4 (2.3)	0	45 (26.0)
Abnormal hepatic function	21 (12.1)	8 (4.6)	14 (8.1)	0	1 (0.6)	44 (25.4)
Decreased WBC count	27 (15.6)	14 (8.1)	3 (1.7)	0	0	44 (25.4)
Increased blood bilirubin	34 (19.7)	6 (3.5)	2 (1.2)	1 (0.6)	0	43 (24.9)
Dizziness	36 (20.8)	3 (1.7)	1 (0.6)	0	0	40 (23.1)
Proteinuria	34 (19.7)	5 (2.9)	0	0	0	39 (22.5)
Increased weight	17 (9.8)	16 (9.2)	3 (1.7)	0	0	36 (20.8)
Increased blood creatinine	33 (19.1)	2 (1.2)	0	0	0	35 (20.2)
QT prolongation	26 (15.0)	4 (2.3)	5 (2.9)	0	0	35 (20.2)
Hypercholesterolemia	29 (16.8)	4 (2.3)	0	0	0	33 (19.1)
Hyperuricemia	30 (17.3)	2 (1.2)	0	0	0	32 (18.5)
Decreased weight	23 (13.3)	8 (4.6)	0	0	0	31 (17.9)
Constipation	28 (16.2)	2 (1.2)	0	0	0	30 (17.3)
Decreased appetite	26 (15.0)	3 (1.7)	0	0	0	29 (16.8)
Increased conjugated bilirubin	22 (12.7)	3 (1.7)	2 (1.2)	1 (0.6)	0	28 (16.2)
COVID-19	10 (5.8)	15 (8.7)	3 (1.7)	0	0	28 (16.2)
Pyrexia	23 (13.3)	3 (1.7)	1 (0.6)	0	0	27 (15.6)
Increased blood CPK	21 (12.1)	5 (2.9)	0	0	0	26 (15.0)
Hypertriglyceridemia	24 (13.9)	2 (1.2)	0	0	0	26 (15.0)

Commentary

- Median exposure of taletrectinib was 12.2 months (range: 0.23 to 40.04)
- **5% (9/173) of patients had a TEAE leading to treatment discontinuation**
- 19% (33/173) of patients had a TEAE leading to a dose reduction
- 40% (70/173) of patients had a TEAE leading to treatment interruption
- Rates of neurologic TEAEs were low (dizziness: 23%; dysgeusia: 10%) and mostly grade 1

Source: Zhou et al., *Journal of Clinical Oncology*, 2024 and Li et al., ASCO Presentation, 2024. Note: Worst grade per patient is reported. TRUST-I study data cutoff: November 29, 2023. ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; CPK: Creatinine phosphokinase; TEAE: Treatment-emergent adverse event; WBC: White blood cell. 1. Taletrectinib-related grade 5 TEAEs occurred in 3 patients: 2 TKI-naïve (1 hepatic failure, 1 pneumonia) and 1 TKI-pretreated (abnormal hepatic function).

Taletrectinib is well-positioned against approved ROS1 TKIs in the first line setting (TKI-naïve patients) based on both response rate and durability

	Taletrectinib[2]	Repotrectinib[3]	Entrectinib[4]	Crizotinib[5]
Study	*TRUST-I*	*TRIDENT-1*	*ALKA-372-001, STARTRK-1, STARTRK-2*	*PROFILE 1001*
n	106	71	168	53
ORR	91%	79%	68%	72%
Median DOR	Not Reached	34.1 months	20.5 months	24.7 months
Median PFS	Not Reached	35.7 months	15.7 months	19.3 months
IC-ORR[1]	88% (n=8)	89% (n=9)	80% (n=25)	N/A

Note: These data are derived from different clinical studies, with differences in study design and patient populations. No he ad-to-head studies have been conducted. Comparisons in a head-to-head study may yield different results. DOR: Duration of response; IC-ORR: Intracranial overall response rate; ORR: Objective response rate; PFS: Progression free survival. 1. Reflects IC-ORR in patients with measurable CNS tumors. 2. Zhou et al., *Journal of Clinical Oncology*, 2024 and Li et al., ASCO Presentation, 2024. 3. Drilon et al., *New England Journal of Medicine*, 2023. 4. Drilon et al., *JTO Clinical Research Reports*, 2022. 5. Shaw et al., *Annals of Oncology*, 2019.

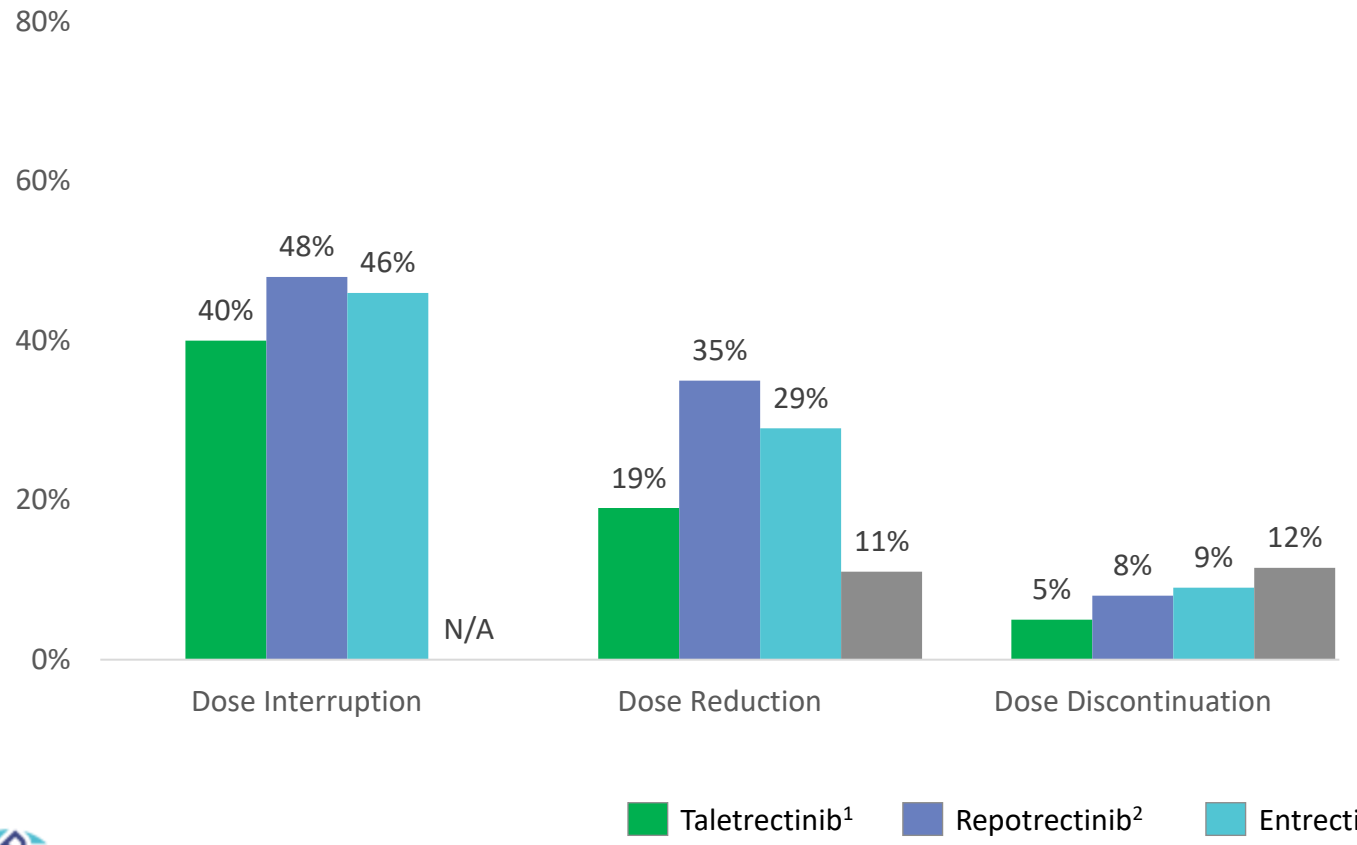
Taletrectinib's response rate in TKI-pretreated patients with G2032R mutations and brain metastases compares favorably to other ROS1 TKIs

	Taletrectinib[1]	Repotrectinib[3]	Zidesamtinib[4]
Study	*TRUST-I*	*TRIDENT-1*	*ARROS-1*
n	66[2]	56	21[5]
ORR	52%	38%	48%
Median DOR	10.6 months	14.8 months	Not reported
Median PFS	7.6 months	9.0 months	Not reported
ORR G2032R	67% (n=12)	59% (n=17)	78% (n=9)
IC-ORR	73% (n=15)[2]	38% (n=13)	100% (n=3)

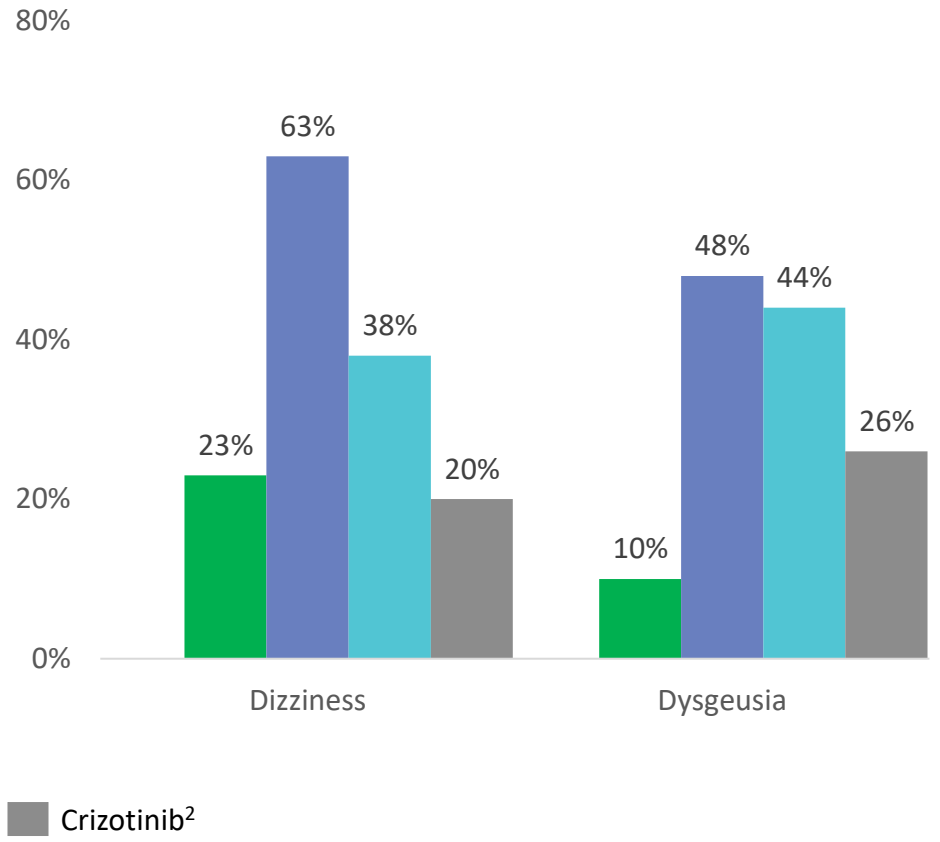
Note: These data are derived from different clinical studies, with differences in study design and patient populations. No head-to-head studies have been conducted. Comparisons in a head-to-head study may yield different results. DOR: Duration of response; IC-ORR: Intracranial overall response rate; ORR: Objective response rate; PFS: Progression free survival. 1. Zhou et al., *Journal of Clinical Oncology*, 2024 and Li et al., ASCO Presentation, 2024. Median duration of response and progression free survival data are still maturing and may change as results from future data cut-off dates are disclosed. 2. Includes patients who had previously been treated with crizotinib; one patient was excluded from the response-evaluable population due to the presence of secondary cancer. 3. Drilon et al., *New England Journal of Medicine*, 2023. 4. *European Journal of Cancer (ENA)*, Drilon et al., 2022. 5. Reflects response-evaluable patients only and includes patients treated with more than one TKI.

Taletrectinib data show an encouraging safety profile relative to approved ROS1 TKIs, including improved dose modification rates and low rates of neurological AEs

Dose modification



Neurological AEs[3]



Taletrectinib[1] Repotrectinib[2] Entrectinib[2] Crizotinib[2]

Note: These data are derived from different clinical studies, with differences in study design and patient populations. No he ad-to-head studies have been conducted. Comparisons in a head-to-head study may yield different results.
AE: Adverse Event. 1. Zhou et al., *Journal of Clinical Oncology*, 2024 and Li et al., ASCO Presentation, 2024. 2. Prescribing information for Augtyro (repotrectinib), Rozlytrek (entrectinib), and Xalkori (crizotinib – Study 1 & 2). 3. Taletrectinib data represents treatment-emergent adverse events in the TRUST-I study.

Taletrectinib positions Nuvation Bio to potentially become a commercial stage company in 2025

Upon regulatory approval, Nuvation Bio is positioned to commercialize a novel targeted therapy for patients with advanced ROS1-positive NSCLC in need of better treatment options

Currently granted:

Priority review of two NDAs for taletrectinib by China's NMPA[1]; Breakthrough Therapy Designations from U.S. FDA and China's NMPA[2]

U.S. launch to be led by:

David Hung, M.D., who successfully developed and commercialized XTANDI®, with current annual worldwide sales of ~$6 billion[3]

License agreements to provide:

Royalties from Innovent Biologics' commercial launch in China and Nippon Kayaku's commercial launch in Japan

1. Based on results of the TRUST-I clinical study, under priority review for the treatment of adult patients with locally advanced or metastatic ROS1-positive NSCLC who either have or have not previously been treated with ROS1 TKIs.
2. Taletrectinib has been granted Orphan Drug Designation from the U.S. FDA for the treatment of patients with ROS1-positive NSCLC and Breakthrough Therapy Designations by both the U.S. FDA and China's NMPA for the treatment of patients with advanced or metastatic ROS1-positive NSCLC. 3. Annual revenue recorded by XTANDI commercialization partners, Astellas Pharma and Pfizer.

Safusidenib | mIDH1i

Diffuse
IDH1-mutant glioma

Phase 2 study ongoing

Safusidenib is a potentially best-in-class, brain penetrant, mIDH1 inhibitor in Phase 2 development for the treatment of patients with diffuse IDH1-mutant glioma

Unmet need

- People diagnosed with glioma have no targeted treatment options[1]

Validated target

- Positive Ph. 3 data[2] with vorasidenib[3] in glioma
- 15% royalty on future U.S. sales of vorasidenib acquired for $905M[4]

Differentiated profile

- Encouraging early data[5]
- Potential in broad population
- Limited competition

Global rights

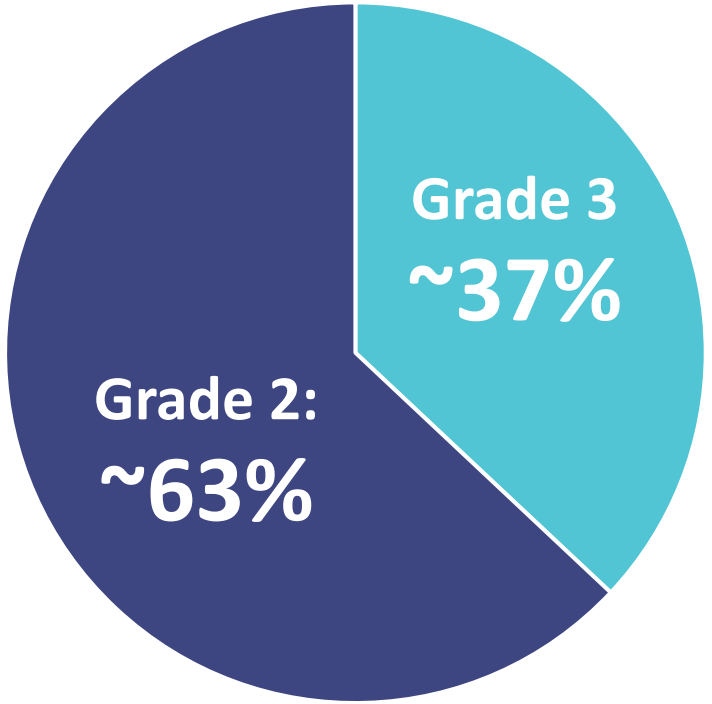
- In-licensed from Daiichi Sankyo
- Daiichi Sankyo retains rights in Japan[6]

1. Current treatment options include surgery, radiation, and chemotherapy. 2. Mellinghoff et al., *New England Journal of Medicine*, 2023. 3. Vorasidenib is being developed by Servier Pharmaceuticals. 4. In May 2024, Royalty Pharma agreed to acquire a 15% royalty on U.S. net sales of vorasidenib in low grade diffuse glioma for $905 million from Agios Pharmaceuticals; Agios will retain 3% of the 15% royalty on sales above $1 billion and the right to receive a $200 million milestone payment from Servier Pharmaceuticals upon U.S. FDA approval. 5. Natsume et al., *Neuro-Oncology*, 2022. 6. Worldwide development and commercial rights in-licensed from Daiichi Sankyo, excluding Japan where Daiichi Sankyo retains development and commercial rights.

The diffuse IDH1-mutant glioma market represents a sizeable commercial opportunity

~13.3K – 18.3K

people living with diffuse IDH1-mutant glioma in the U.S.



Diffuse IDH1-mutant glioma patients are in need of better treatment options

Source: CBTRUS Statistical Report: U.S. Cancer Statistics – NPCR and SEER, 2018-2020.

Safusidenib clinical study data set approaches 100 patients

Sponsor: Daiichi Sankyo

J101 – Phase 1

Japan

Grades 2-4 IDH1-mutant glioma

n=47

J201 – Phase 2

Japan

Grade 2 IDH1-mutant glioma

n=27

Sponsor: Nuvation Bio

G203 – Phase 2

Global

Diffuse IDH1-mutant glioma

Part 1: Dose evaluation (n=25)

Part 2: Design under discussion

Royalty Pharma's acquisition of rights to Agios Pharmaceuticals' royalty on potential U.S. net sales of vorasidenib validates safusidenib's market potential

Transaction highlights[1]

- On May 28, 2024, Royalty Pharma announced it acquired an interest in Agios Pharmaceuticals' royalty on U.S. net sales of Servier's vorasidenib
- Royalty Pharma will pay Agios **$905 million** in cash upon FDA approval of vorasidenib for a **15% royalty on annual U.S. net sales up to $1 billion**
- Royalty Pharma will receive a 12% royalty and Agios will retain a 3% royalty on potential U.S. net sales >$1 billion
- Agios retains rights to a $200 million milestone payment from Servier upon FDA approval of vorasidenib

Implications to safusidenib

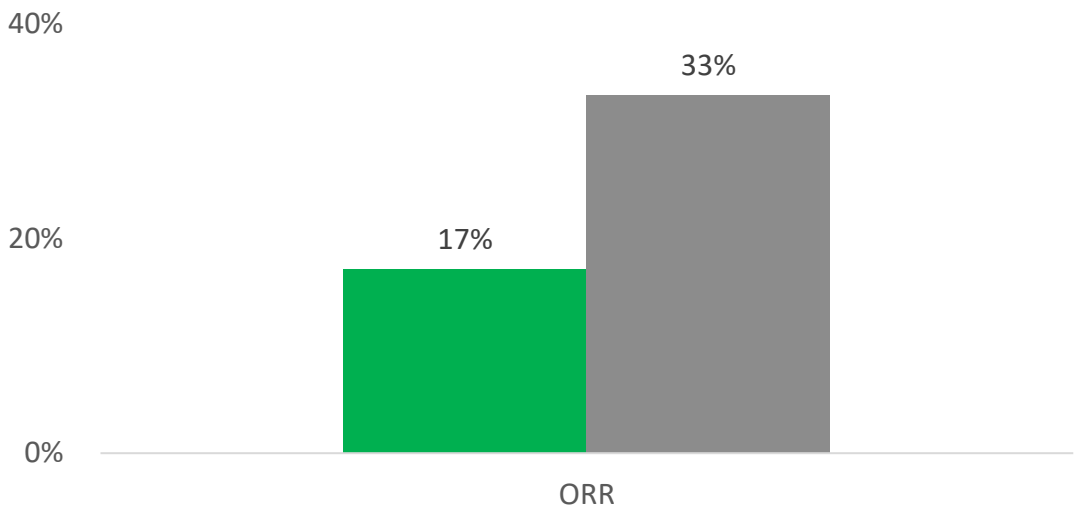
- Transaction anticipates >$1 billion in peak U.S. net sales for vorasidenib[1]
- Safusidenib showed a 17% response rate in enhancing gliomas[2]
- Vorasidenib showed a 0% response rate in enhancing gliomas[3]
- Safusidenib may potentially address an important vorasidenib unmet need

1. Royalty Pharma plc press release, Royalty Pharma investor presentation, and Agios Pharmaceuticals press release. 2. Natsume et al., *Neuro-Oncology*, 2022. 3. Mellinghoff et al., *Clinical Cancer Research*, 2021.

Safusidenib generated particularly encouraging efficacy signals in IDH1-mutant enhancing and non-enhancing glioma relative to vorasidenib

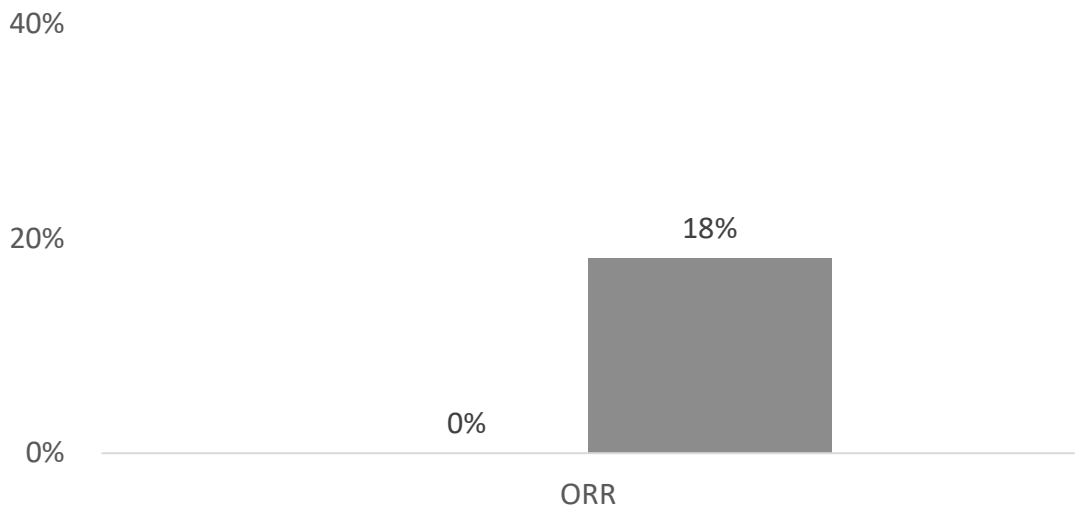
Safusidenib[1]

Enhancing glioma n=35
Non-enhancing glioma n=12



Vorasidenib[2]

Enhancing glioma n=30
Non-enhancing glioma n=22



Note: Information depicts response rates with IDH inhibitor in IDH1-mutant gliomas in Phase 1 studies; Enhancing and non-enhancing glioma was assessed by Response Assessment in Neuro-Oncology (RANO). Contrasting enhancement is generally associated with a higher degree of malignancy. These data are derived from different clinical studies, with differences in study design and patient populations. No head-to-head studies have been conducted. Comparisons in a head-to-head study may yield different results. ORR: Objective response rate. 1. Natsume et al., *Neuro-Oncology*, 2022 2. Mellinghoff et al., *Clinical Cancer Research*, 2021.

NUV-868 | BETi

Advanced solid tumors	Phase 1 monotherapy study completed; MTD determined
Ovarian, TNBC, pancreatic, mCRPC & other solid tumors	Phase 1b combination study ongoing

First generation BET inhibitors have been toxic and poorly effective against solid tumors

NUV-868 is the most selective BD2 vs BD1 BET inhibitor in development

- BET proteins regulate the expression of many oncogenes, including cMYC – an oncogene that has not been targetable directly with a drug
- Non-selective BD1/2-inhibitors have been associated with tolerability issues, many apparently due to BD1 inhibition[1]
- **NUV-868 inhibits BD2 almost 1,500 times more potently than BD1, which may improve efficacy and tolerability**

	BRD4 Affinity[2]		
	BD2 (nM)	BD1 (nM)	Selectivity
NUV-868*	2	2920	1460x
ABBV-744[3]	1.05	340	324x
PLX-2853[4]	Modest BD2 selectivity		
CPI-0610[3]	17	85	5x
ABBV-075[1]	3	11	3.7x
MK-8628/OTX-015[5]	17	26	1.5x
BI-894999[6]	41	5	0.1x
ZEN-3694[7]	Non-selective		

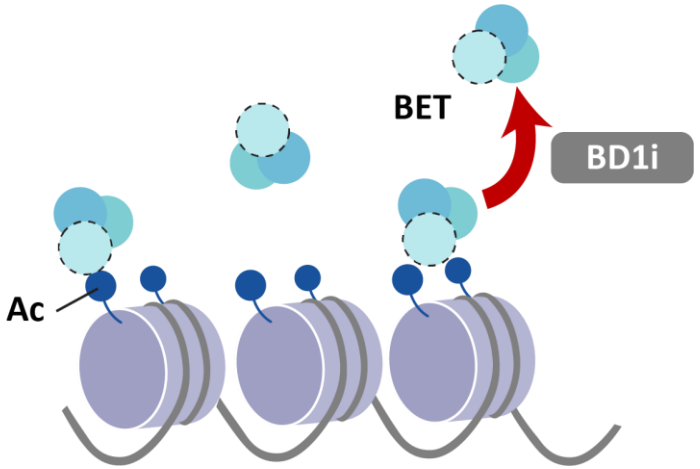
*high plasma protein binding, > 1% free fraction

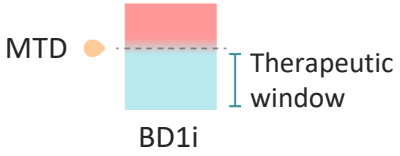
1. Faivre et al 2020. 2. Various assays used. 3. Internal Nuvation Bio data. 4. https://ash.confex.com/ash/2020/webprogram/Paper140138.html. 5. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5474678/.
6. https://www.nature.com/articles/s41388-018-0150-2. 7. 2016-EORTCposter-ZenithEpigenetics.pdf.

BD2 selectivity blocks the ability of cancer cells to induce resistance pathways and, by avoiding BD1 inhibition, increases tolerability

BD1 inhibition

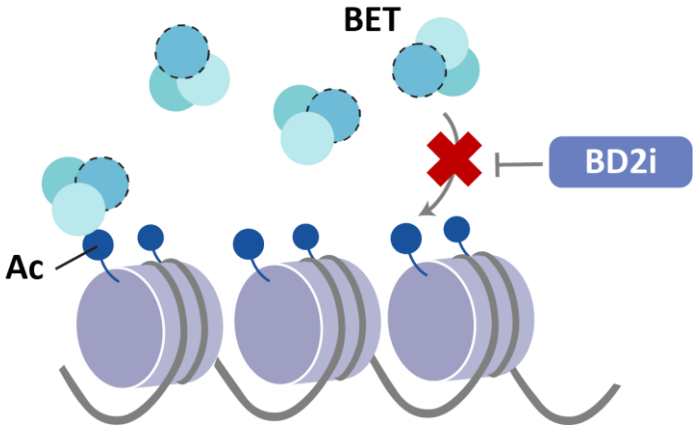
- Regulates steady state gene expression
- **Displaces** BET proteins already associated with histones
- Toxicity minimizes the therapeutic window

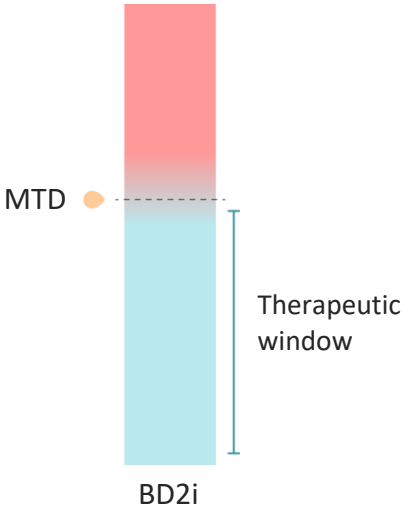




BD2 inhibition

- Regulates rapid gene induction
- **Prevents** BET proteins from becoming associated with histones
- Effective in models of cancer and inflammatory diseases
- BD2 selectivity increases the therapeutic window

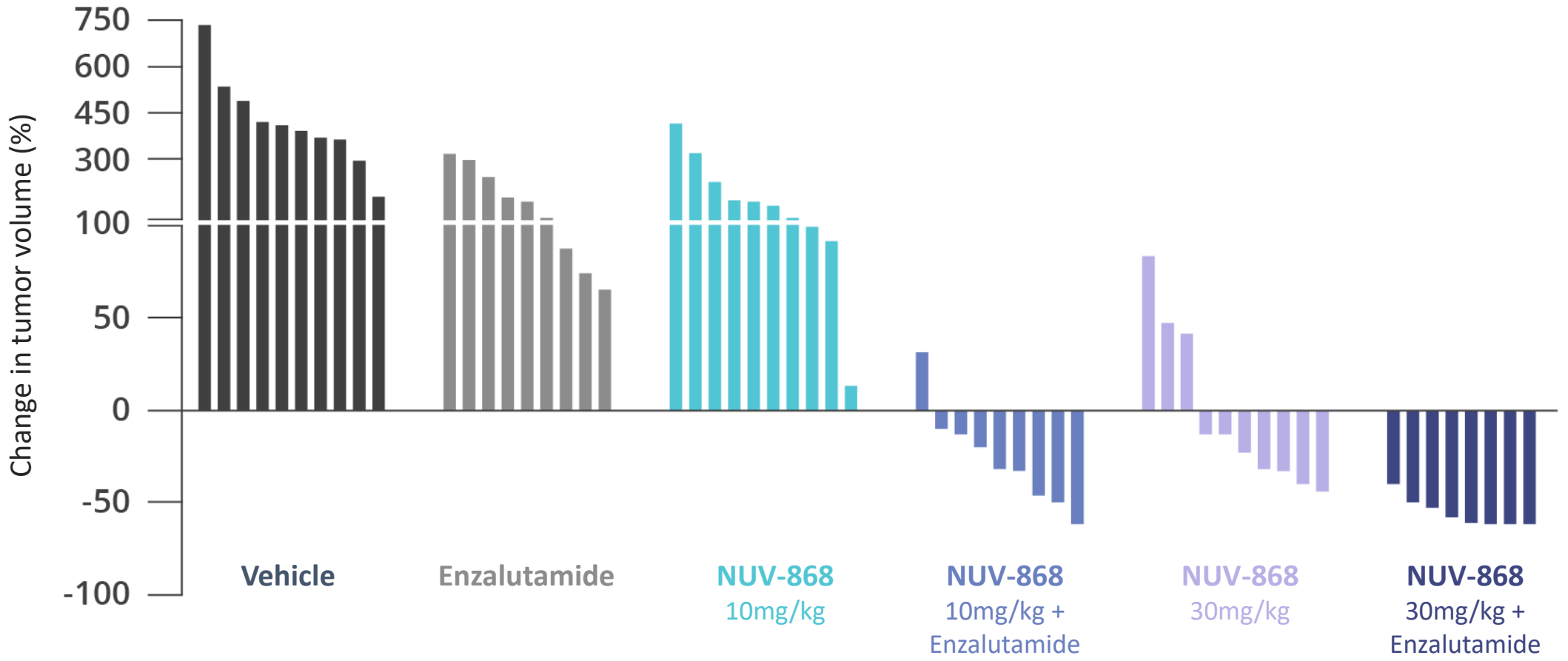




Ac: Acetylated chromatin; MTD: Maximum tolerated dose

NUV-868 treatment converts enzalutamide-resistant patient derived prostate cancer xenografts to again be enzalutamide-sensitive

Individual animal tumor volume



Note: Experiments conducted using BID dosing.

NUV-868 reduces PARP inhibitor resistance possibly by preventing the induction of alternative DNA repair pathways

Limitations of PARP inhibition

- Genes will find other ways to repair themselves even in presence of PARP inhibitors; repaired DNA leads to cancer growth

DNA damaged

↓

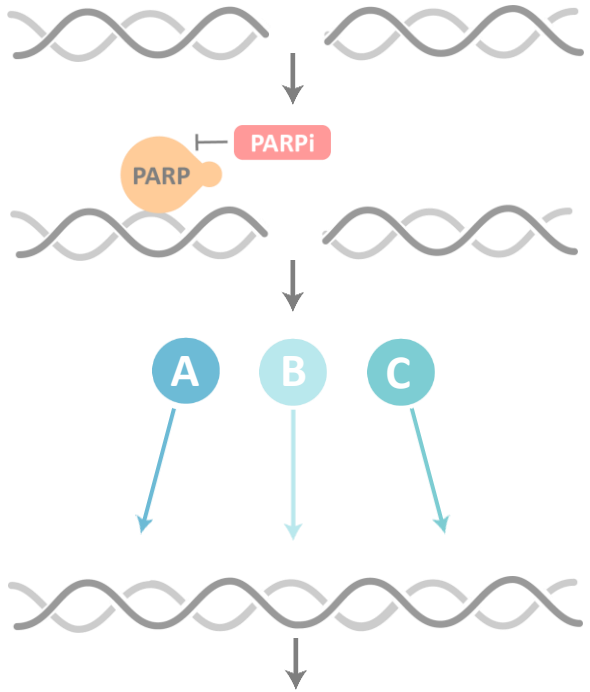
PARPi blocks repair

↓

Genes find other ways to induce DNA repair even in presence of a PARPi

↓

DNA repaired



Cancer Growth

Benefits of PARP and BET inhibition in combination

- BET inhibitors inhibit genes from inducing other repair pathways to combat PARP inhibition, resulting in cell death

DNA damaged

↓

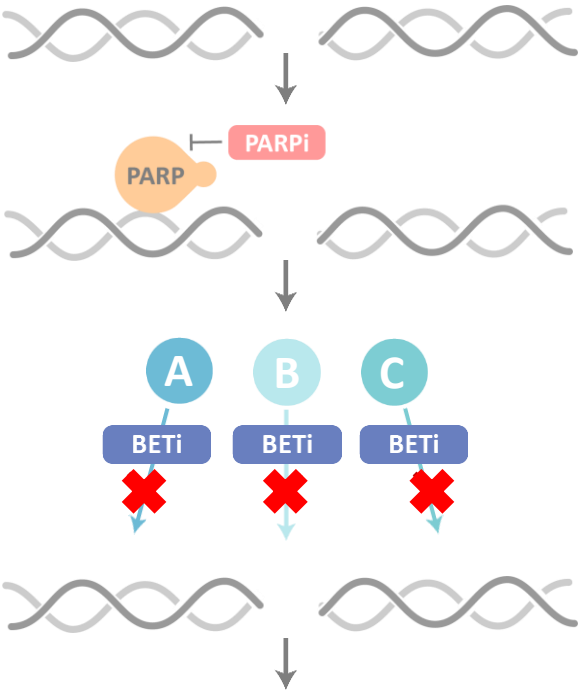
PARPi blocks repair

↓

Genes try to induce other pathways for DNA repair; **NUV-868 blocks induction of new pathways**

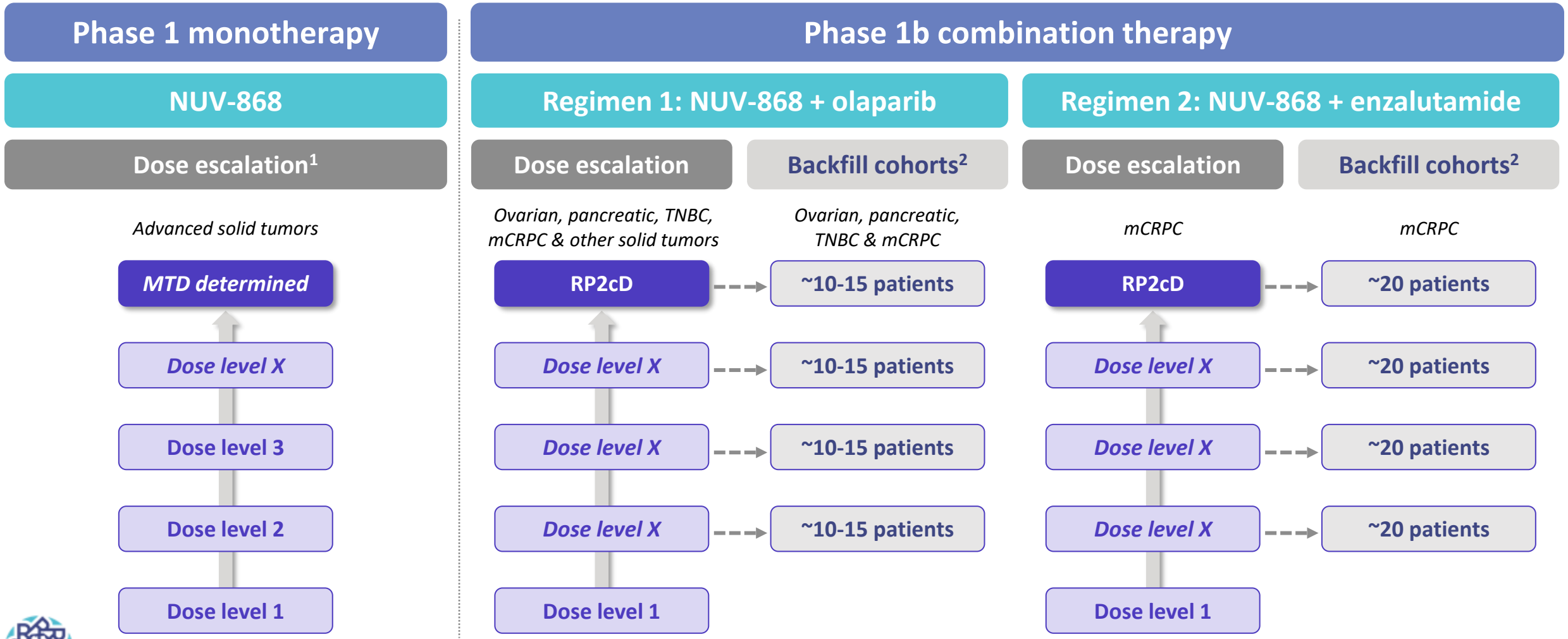
↓

DNA not repaired



Cell Death

Modified NUV-868 Phase 1 and Phase 1b development plan expedites ability to reach a larger number of patients and determine RP2D/RP2cD



MTD: Maximum Tolerated Dose; RP2D: Recommended Phase 2 dose; RP2cD: Recommended Phase 2 combination dose. 1. Treatment in Phase 1 monotherapy dose escalation study completed; Maximum Tolerated Dose determined. 2. Backfill cohorts to include ~10-15 patients per tumor type.

NUV-1511 | DDC

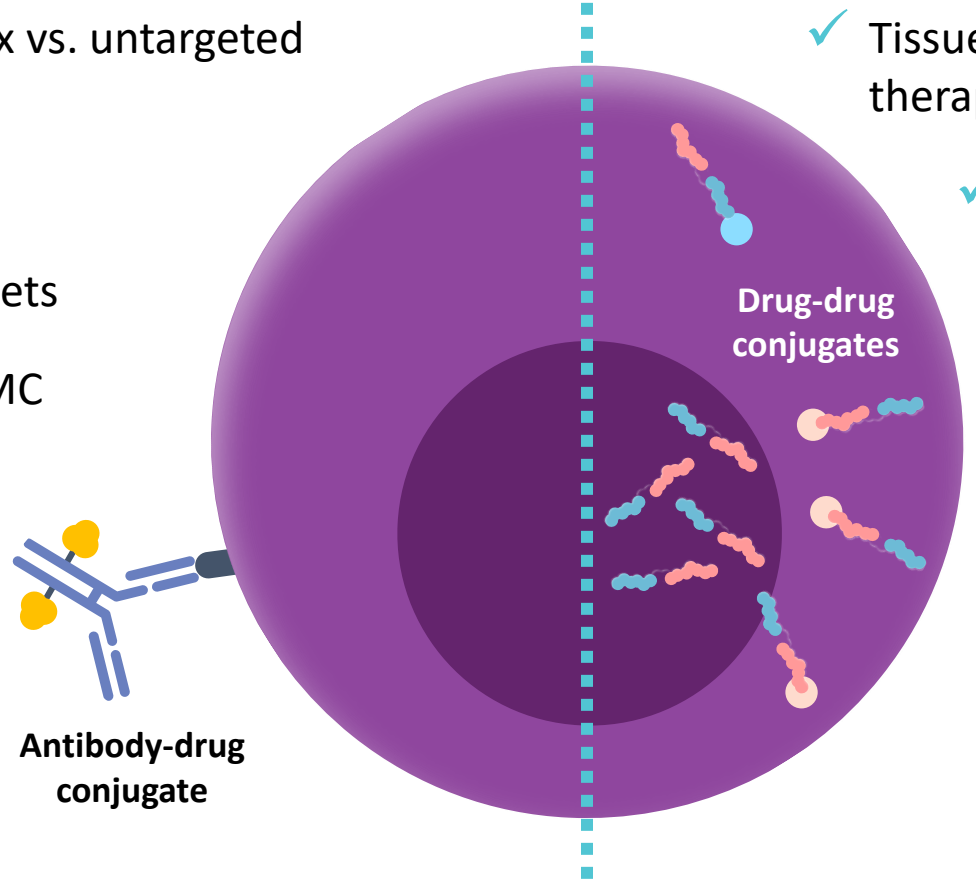
Advanced solid tumors

Phase 1 study ongoing

Nuvation Bio's drug-drug conjugate (DDC) platform is a potentially revolutionary advance beyond ADCs

Antibody-drug conjugates

- ✓ Improves therapeutic index vs. untargeted warhead
- × IV delivery
- × Limited to cell-surface targets
- × Complex and expensive CMC

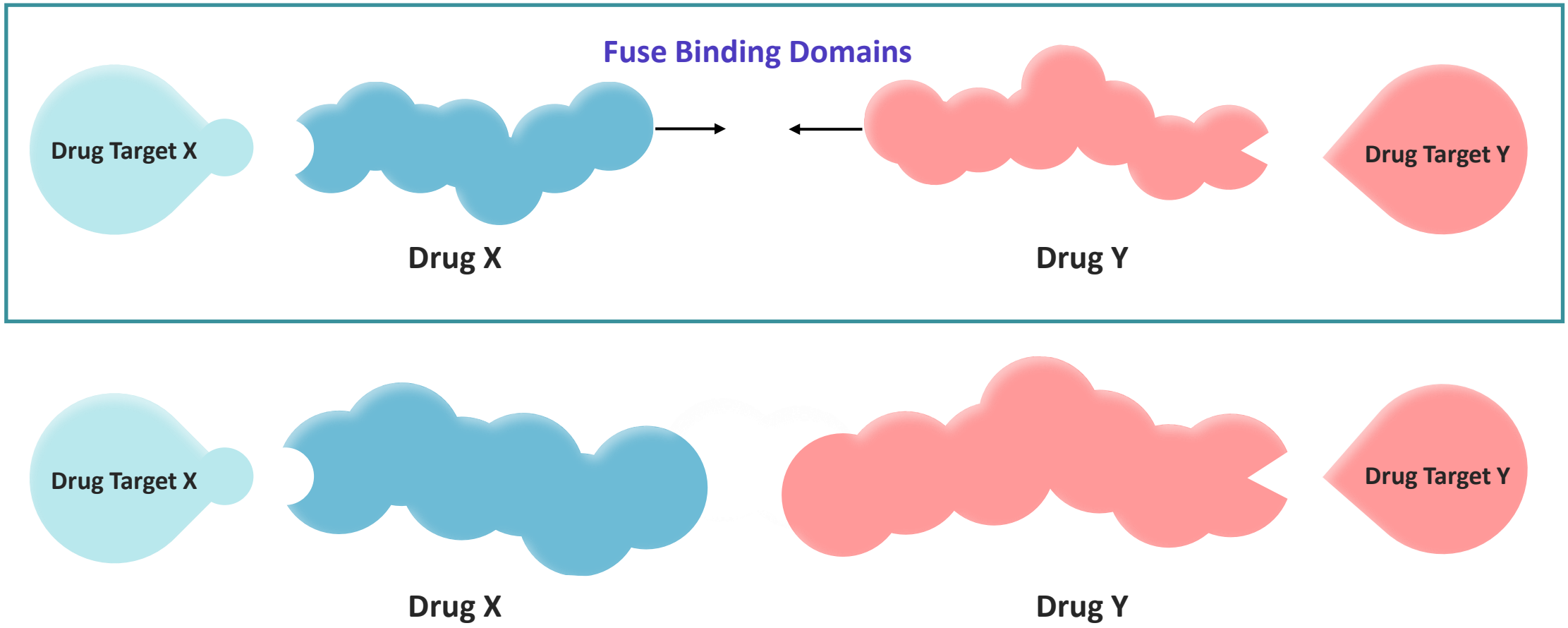


Drug-drug conjugates

- ✓ Tissue-selective targeting improves therapeutic index vs. untargeted warhead
- ✓ Oral or IV delivery
- ✓ Binds intracellular and cell membrane targets
- ✓ Highly cell permeable
- ✓ Simpler and less expensive to manufacture

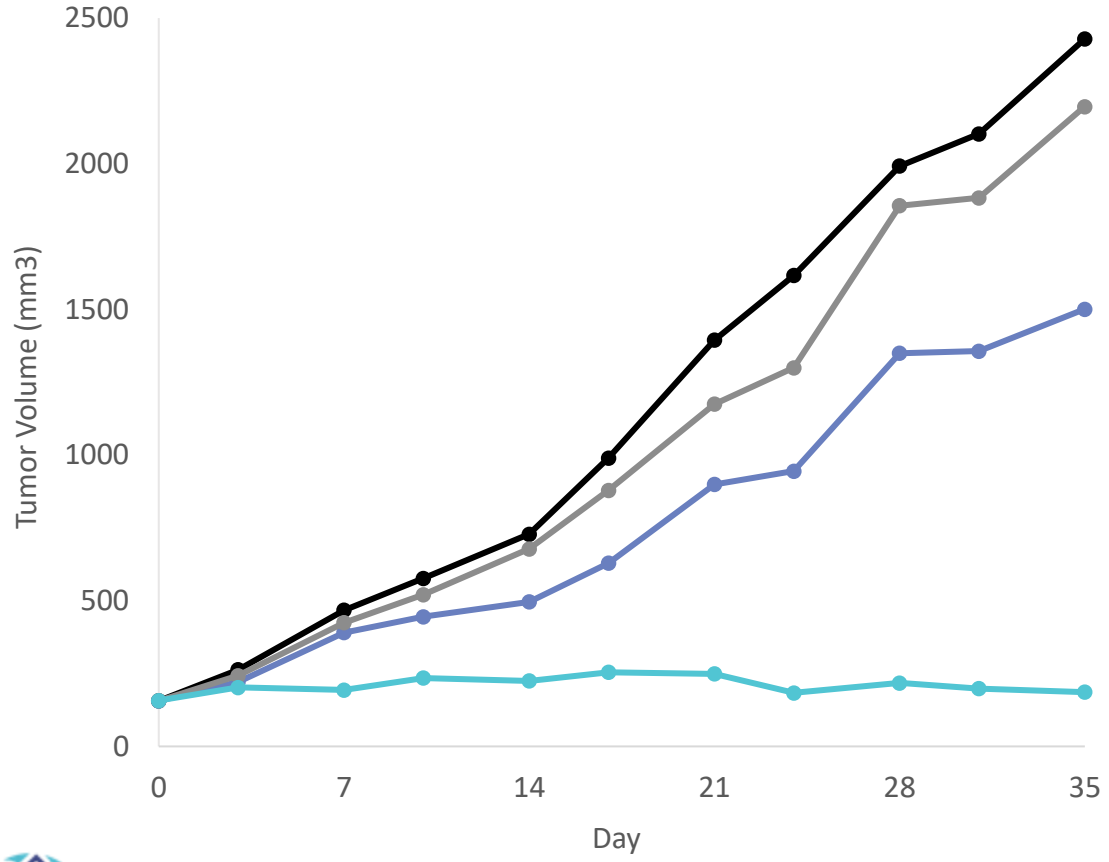
DDCs are designed to bind TWO different targets simultaneously

Two separate drugs with two separate targets

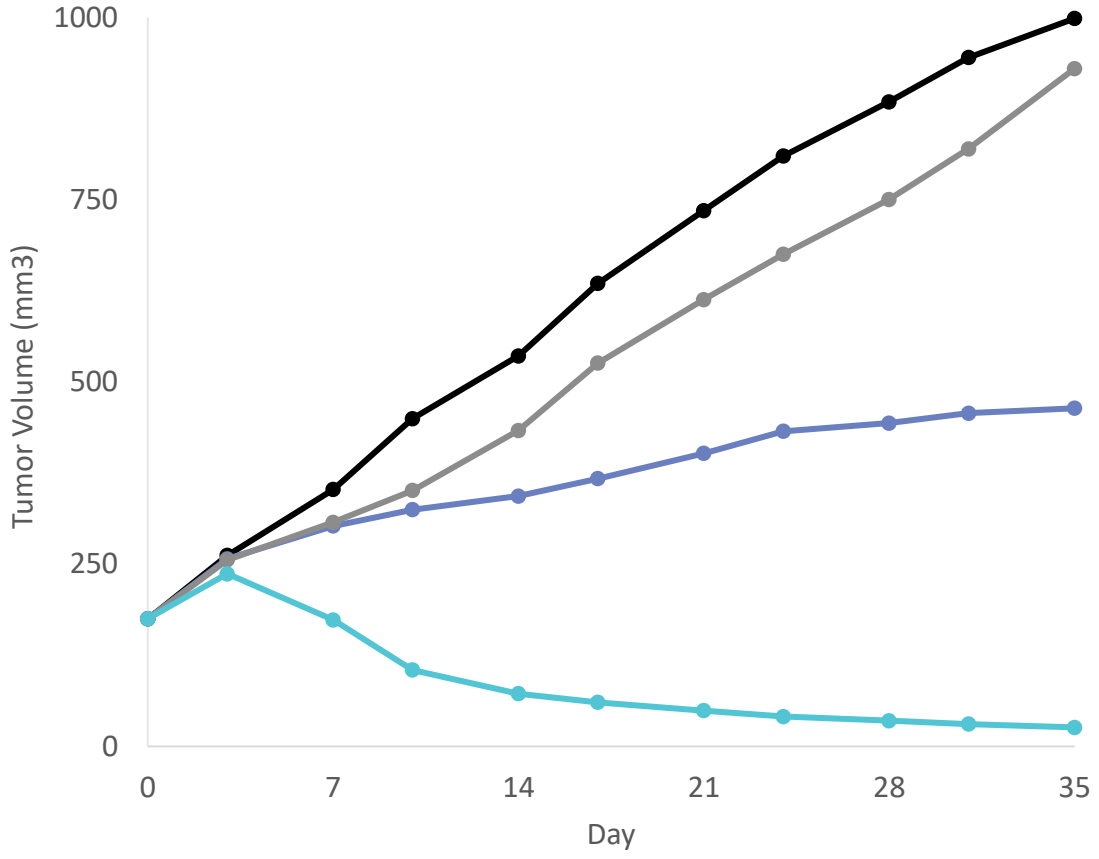


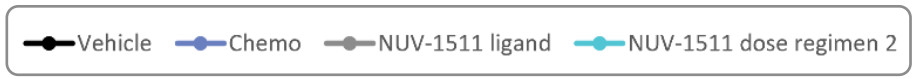
NUV-1511, a DDC derivative of a widely used chemotherapy agent, suppresses prostate and breast cancer growth in xenografts

Prostate cancer CDX (LNCAP)



ER+ breast cancer CDX (T47D)

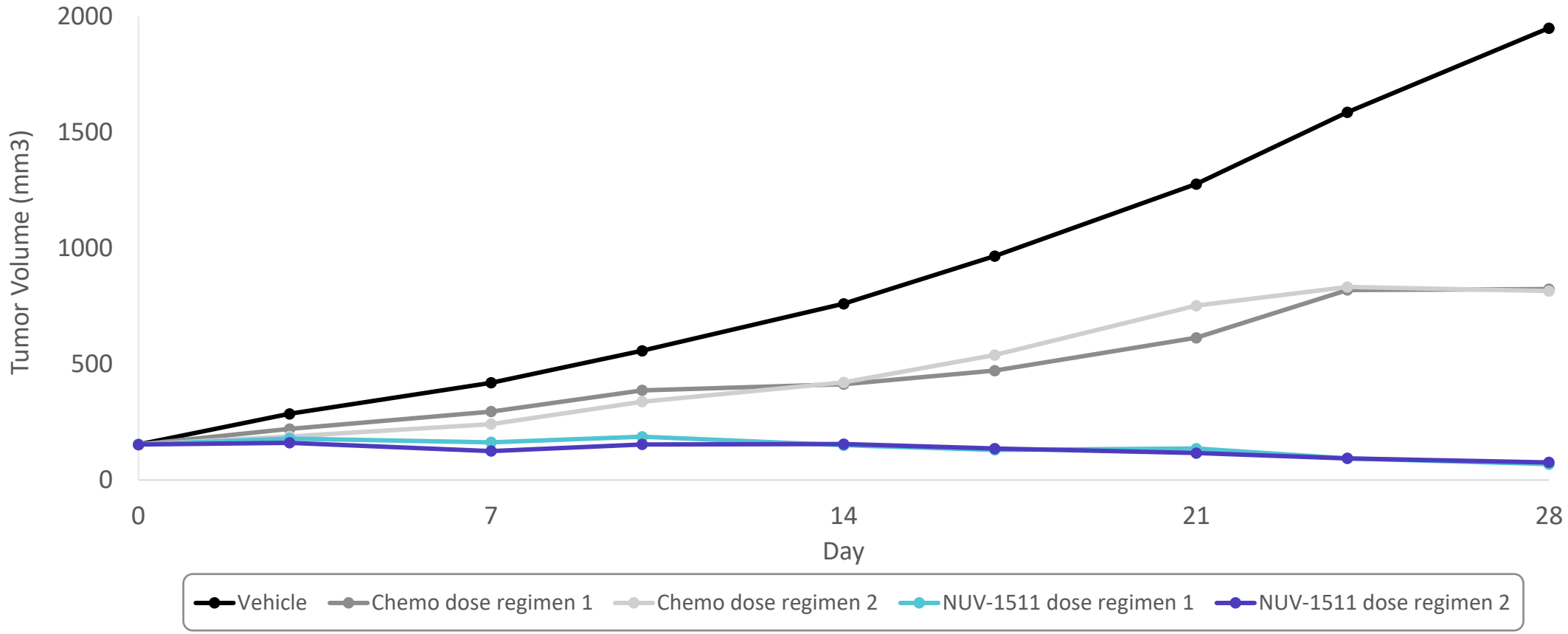




CDX: Cell-line derived xenograft; ER: Estrogen receptor.

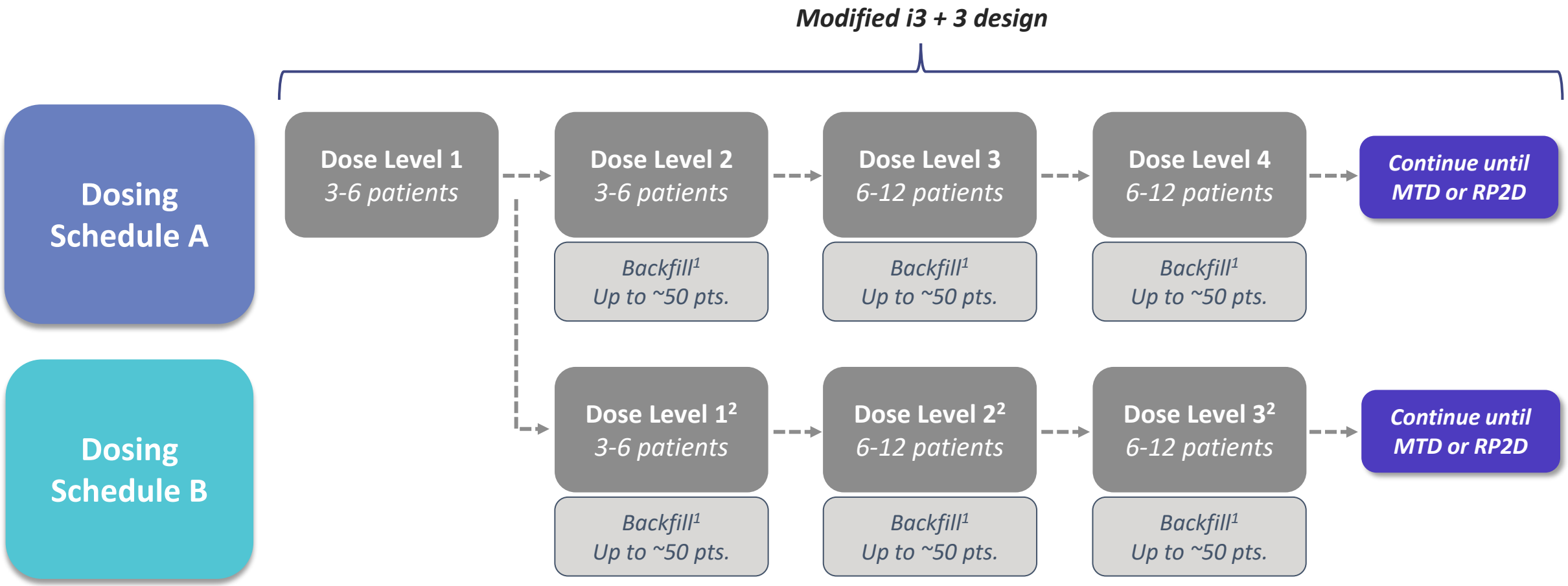
Intermittent dosing of NUV-1511 leads to sustained tumor inhibition for weeks

Prostate cancer CDX (LNCAP)



CDX: Cell-line derived xenograft.

NUV-1511 Phase 1 study protocol provides flexibility to explore two dosing schedules while efficiently determining a RP2D



MTD: Maximum Tolerated Dose; RP2D: Recommended Phase 2 dose. 1. Nuvation will determine when and if to open backfill cohort(s). 2. Nuvation in consultation with Dose Escalation Committee will decide when to open Dosing Schedule B cohorts.

Committed team tackling the greatest unmet needs in oncology

Experienced biotech leadership team

- Founded by Dr. David Hung, previously the founder and CEO of Medivation, who successfully developed and commercialized XTANDI®

Potentially best-in-class candidates leveraging and improving validated mechanisms

- Potential for better efficacy and tolerability

Strong cash position to support operations in near term

- $597.0 million as of March 31, 2024

Broad pipeline across multiple stages of development

- **Taletrectinib | ROS1 inhibitor:**
  Two Phase 2 pivotal studies ongoing
- **Safusidenib | mIDH1 inhibitor:**
  Phase 2 study ongoing
- **NUV-868 | BD2-selective BET inhibitor:**
  Phase 1b combination studies ongoing
- **NUV-1511 | Drug-drug conjugate:**
  Phase 1 dose escalation study ongoing

Potential to become a commercial stage organization in 2025

- Taletrectinib has been granted Breakthrough Therapy Designations in the U.S. and China for the treatment of patients with advanced or metastatic ROS1-positive NSCLC