UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

FORM 8-K

CURRENT REPORT
Pursuant to Section 13 or 15(d)
of the Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): January 10, 2022

Nuvation Bio Inc.
(Exact name of registrant as specified in its charter)

Delaware	**001-39351**	**85-0862255**
(State or other jurisdiction of incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

1500 Broadway, Suite 1401 **New York, NY**	**10036**
(Address of principal executive offices)	**(Zip Code)**

Registrant's telephone number, including area code: (332) 208-6102

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligations of the registrant under any of the following provisions:

☐ Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Class A Common Stock, $0.0001 par value per share	**NUVB**	**The New York Stock Exchange**
Redeemable Warrants, each whole warrant exercisable for one share of Common Stock at an exercise price of $11.50 per share	**NUVB.WS**	**The New York Stock Exchange**

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01. Regulation FD Disclosure.

Nuvation Bio Inc. (the "***Company***") intends to conduct meetings with securities analysts, investors and others in connection with the 40th Annual J.P. Morgan Healthcare Conference beginning on January 10, 2022. As part of these meetings, the Company intends to utilize the corporate slide presentation furnished with this report as Exhibit 99.1.

The information in this Item 7.01 is being furnished, not filed, pursuant to Regulation FD. Accordingly, the information in Item 7.01 of this report will not be incorporated by reference into any registration statement filed by the Company under the Securities Act of 1933, as amended, unless specifically identified therein as being incorporated therein by reference. The furnishing of the information in this report is not intended to, and does not, constitute a determination or admission by the Company that the information in this report is material or complete, or that investors should consider this information before making an investment decision with respect to any security of the Company.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description
99.1	Corporate Slide Presentation, dated January 10, 2022
104	Cover Page Interactive Data File (embedded within the Inline XBRL document)

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, as amended, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

NUVATION BIO INC.

Date: January 10, 2022

By: /s/ Jennifer Fox
Name: Jennifer Fox
Title: Chief Financial Officer

Exhibit 99.1

Nuvation Bio

DRIVEN BY SCIENCE

FOCUSED ON LIFE

January 2022

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, statements regarding Nuvation Bio's business strategies, cash resources, current and prospective product candidates, the potential therapeutic benefit of Nuvation Bio's product candidates, the expected timing of regulatory filings and clearance and clinical trial dose selection, initiation and data presentation, as well as the potential for market acceptance of any approved products and the related market opportunity. 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. Actual events and circumstances, many of which are beyond Nuvation Bio's control, are difficult or impossible to predict and may cause actual results to differ materially from those anticipated by these forward-looking statements. Factors that could cause or contribute to such differences include, but are not limited to, the inherent uncertainty associated with pharmaceutical product development and clinical trials; the risk of unexpected emergence of adverse events or other undesirable side effects; delays in clinical trials due to difficulties or delays in the regulatory process, enrolling subjects or manufacturing or supplying product for such clinical trials; disruptions to normal business operations relating to the COVID-19 pandemic; the risk that Nuvation Bio will be unable to successfully market or gain market acceptance of its product candidates; the risk that Nuvation Bio's product candidates may not be beneficial to patients or successfully commercialized; the risk that Nuvation Bio has overestimated the size of the target patient population, their willingness to try new therapies and the willingness of physicians to prescribe these therapies; and developments in the competitive landscape. The risks and uncertainties facing Nuvation Bio are discussed more fully in its Quarterly Report on Form 10-Q filed with the SEC on November 10, 2021, under the heading "Risk Factors," and other documents that Nuvation Bio has filed or will file with the SEC. There may be additional risks that Nuvation Bio does not presently know, or that Nuvation Bio currently believes are immaterial, that could also cause actual results to differ from those anticipated by the forward-looking statements. In addition, forward-looking statements reflect Nuvation Bio's expectations, plans or forecasts of future events and views only as of the date of this Presentation. Nuvation Bio anticipates that subsequent events and developments will cause its assessments to change. However, while Nuvation Bio may elect to update these forward-looking statements at some point in the future, Nuvation Bio specifically disclaims any obligation to do so.

Recent accomplishments and potential 2022 milestones

Recent Accomplishments

- First patient treated with NUV-422 in high grade glioma (HGG) trial in December 2020
- Dose escalation ongoing in Phase 1 study in High Grade Glioma (HGG), breast and prostate cancer
- IND accepted for advanced Beast Cancer (aBC) Phase 1b combination study with fulvestrant and NUV-422
- IND accepted for metastatic Castration-Resistant Prostate Cancer (mCRPC) Phase 1b combination study with enzalutamide and NUV-422
- Fast track designation granted for NUV-422 in HGG
- Wee1 clinical development candidate, NUV-569, declared
- Bioavailable Drug-Drug conjugate (DDC) candidate in lead optimization

Potential 2022 Milestones

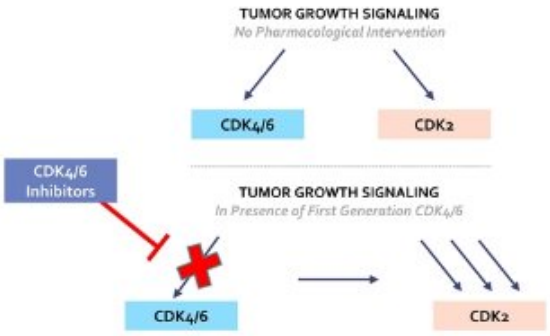
- Identify Recommended Phase 2 dose (RP2D) for NUV-422:
  - Initiate Phase 2 monotherapy dose expansion cohorts in recurrent Glioblastoma (rGBM), aBC and mCRPC
  - Initiate combination Phase 1b studies in aBC and mCRPC
- Present initial data from Phase 1 NUV-422 dose escalation study
- Acceptance of IND for BETi NUV-868 and treatment of first patient in NUV-868 Phase 1 study in advanced solid tumors
- Submit Wee1 IND
- DDC Clinical Candidate Selection
- A2A Clinical Candidate Selection

NUV-422 | CDK 2/4/6i

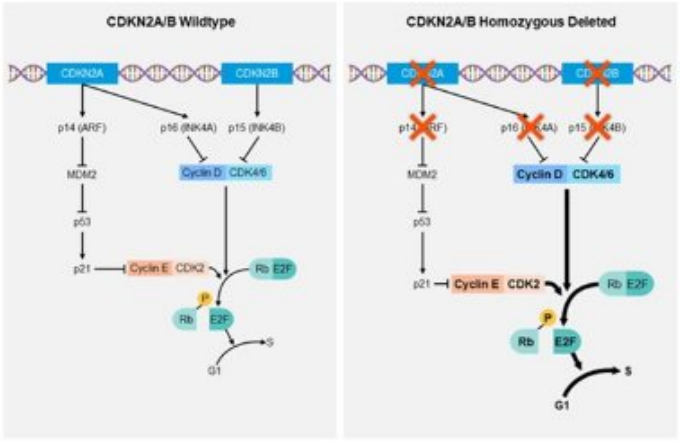
rGBM	Dec 2020 First patient dosed
HR+ aBC	Phase 2 Initiation by Year End 2022
mCRPC	Phase 2 Initiation by Year End 2022

Nuv-422 selectively targets CDK2 in addition to CDK4/6 and may prevent or reverse resistance

CDK2 Drives Resistance to CDK4/6 Inhibitors



CDKN2A Deletion or alterations commonly Drive Cancer growth Through CDK2/4/6



CDK2/4/6 inhibition that avoids CDK1 may be associated with better efficacy and tolerability

DRIVES EFFICACY

1st Generation	CDK 4	CDK 6	CDK 2	METASTATIC *Monotherapy Label*	Adjuvant Setting
KISQALI® ribociclib	2	2	10000	X	? NATALEE
IBRANCE® palbociclib	4	2	2470	X	X PALLAS X PENELOPE-B
Verzenio® abemaciclib	2	10	504	✓	✓ monarch-E

2nd Generation	CDK 4	CDK 6	CDK 2	CDK 1
PF-06873600	2	4	0.3	2
NUV-422	2	1	7	73

CAUSES TOXICITY

K_{ip} (nM)

NUV-422-02 phase 1/2 monotherapy study

PHASE 1 Dose Escalation
PRIMARY OBJECTIVE: safety, tolerability, RP2D

HGG, HR+/HER2- aBC, and mCRPC

Including Surgical Substudy in rGBM, and Dose Backfill*

Phase 1 Dose Escalation Data By Year End

RP2D

PHASE 2 in Multiple Tumor Types
PRIMARY OBJECTIVE: efficacy

Isocitrate dehydrogenase wild-type glioblastoma (**IDHwt) rGBM**
COHORT 1: Up to 40 pts with measurable disease

HR+/HER2- aBC POST CDK4/6i
COHORT 2: Up to 40 pts with measurable disease
COHORT 4: Up to 40 pts with active brain mets

mCRPC POST ANDROGEN RECEPTOR THERAPY (AR) & TAXANE
COHORT 3: Up to 40 pts with measurable disease or rising PSA

**Dose Backfill will enroll additional pts at cleared dose levels to further evaluate safety and PK*

Glioblastoma

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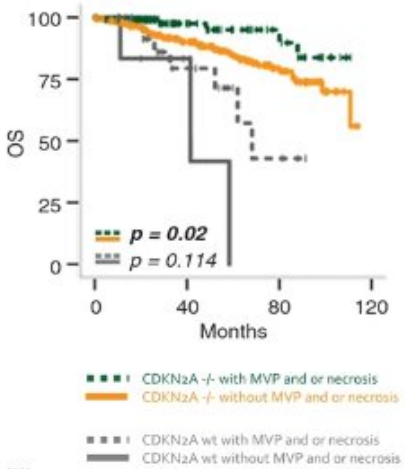
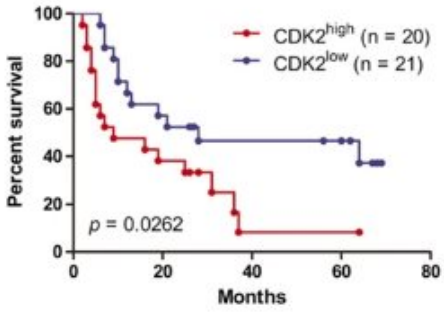
CDKN2A deletion and CDK2 overexpression associated with worse survival in primary high-grade gliomas

CDKN2A Deletion is Associated With Worse Survival[1]

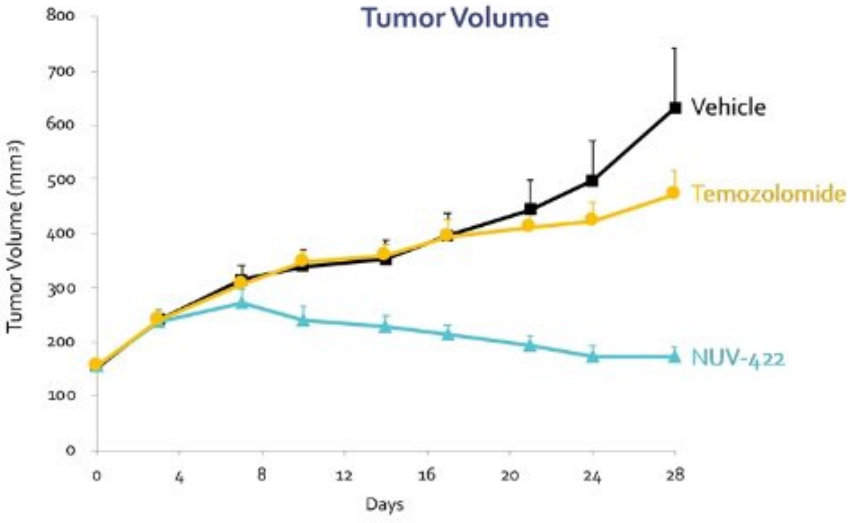


CDK2 Expression Is Associated With Lower Overall Patient Survival[2]



[1]Appay et al., 2020
[2]Wang et al., 2016

NUV-422 is superior to standard of care temozolomide in xenograft model of GBM



NUV-422 30 mg/kg PO QD

NUV-422-02 rGBM monotherapy phase 1/2

PHASE 1 Dose Escalation
PRIMARY OBJECTIVE: safety, tolerability, RP2D

HGG, including rGBM

Dose Escalation & Dose Backfill

↓

Surgical Substudy: rGBM

PRIMARY OBJECTIVE: PK of NUV-422 in resected tumor tissue
Up to 30 patients randomized (2:1)

Phase 1 Dose Escalation Data By Year End

RP2D

PHASE 2 Dose Expansion
PRIMARY OBJECTIVE: efficacy; ORR & DOR

IDHwt rGBM CDKN2A known status
Up to 40 patients

Breast Cancer

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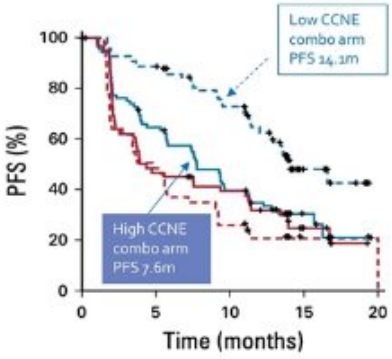
NUV-422 inhibits growth of palbociclib-resistant ER+ breast cancer cells with high CDK2/Cyclin E

Cyclin E predicts resistance to palbociclib

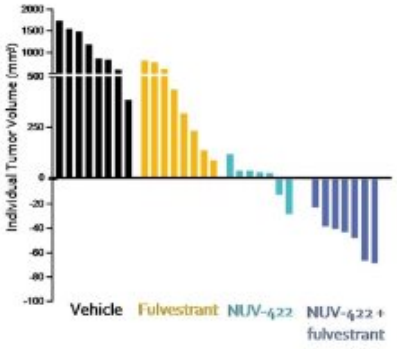


NUV-422 has similarly strong potency in palbociclib-sensitive and palbociclib-resistant cells

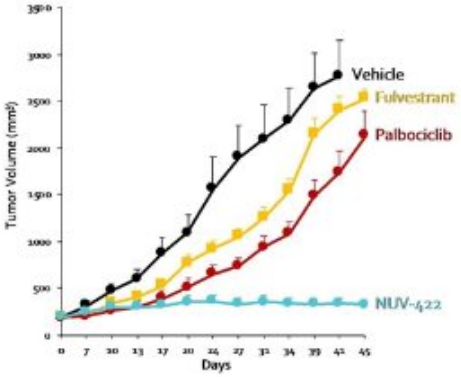
	Proliferation Inhibition IC50 (nM)	
Compound	Palbociclib-sensitive cells	Palbociclib-resistant cells
Cisplatin	11580	10070
Palbociclib	288	1401
NUV-422	229	325

NUV-422 shows activity across ER+ breast cancer xenografts

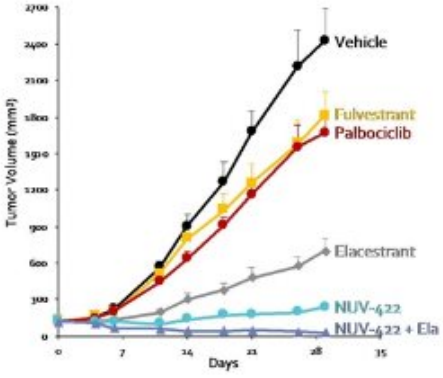
ER+ breast cancer xenograft



ER+ CDK4/6 inhibitor and fulvestrant resistant patient derived breast cancer xenograft harboring an ESR1 mutation



ER+ fulvestrant-resistant patient derived breast cancer xenograft



NUV-422 30 mg/kg PO QD

NUV-422-02 2L+ aBC monotherapy phase 1/2

PHASE 1 Dose Escalation
PRIMARY OBJECTIVE: safety, tolerability, RP2D

HR+/HER2- aBC 2L+

Dose Escalation & Dose Backfill

Phase 1 Dose Escalation Data By Year End

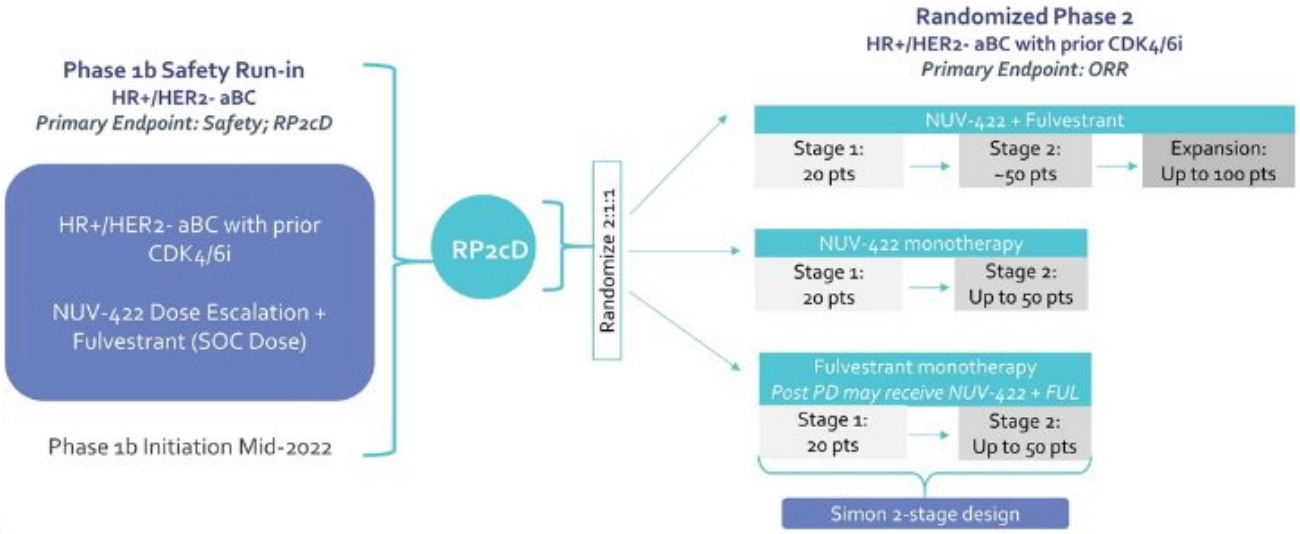
RP2D

PHASE 2 Dose Expansion
PRIMARY OBJECTIVE: efficacy; ORR & DOR

HR+/HER2- aBC POST CDK4/6i
COHORT 2: Up to 40 pts with measurable disease

HR+/HER2- aBC with ACTIVE BRAIN METS POST CDK4/6i
COHORT 4: Up to 40 pts with measurable brain lesion

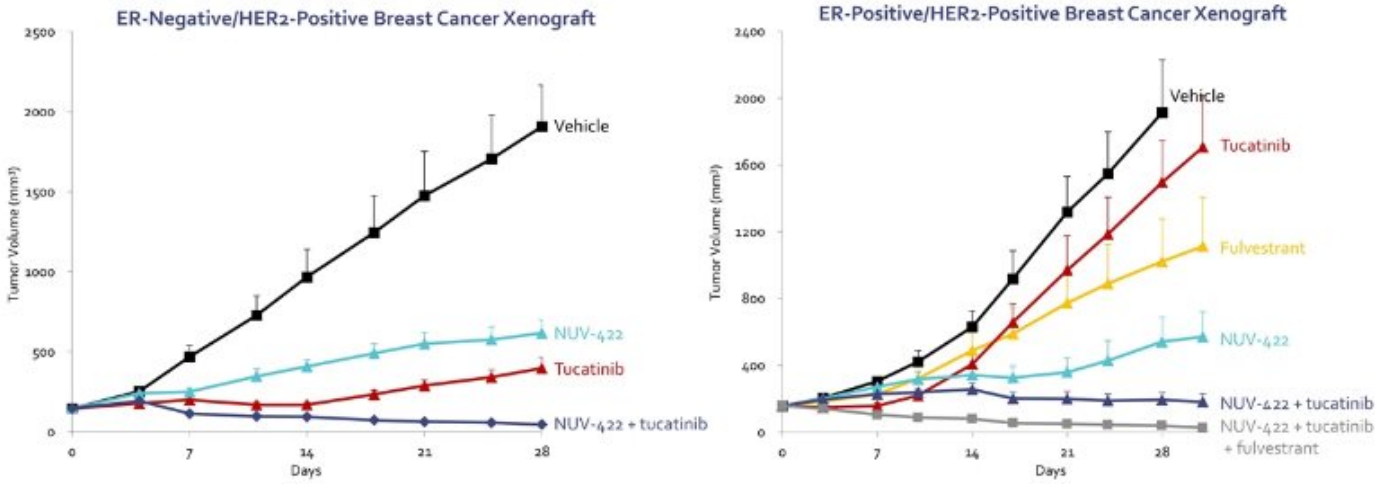
NUV-422-03 phase 1b/2 aBC study NUV-422 in combination with fulvestrant



RP2cD: Recommended Phase 2 Combination Dose
FUL: Fulvestrant

Additional xenograft data suggests broad potential for NUV-422 in endocrine-independent breast cancer

Tumor Volume



NUV-422 30 mg/kg PO QD

Prostate Cancer

Nuvation Bio

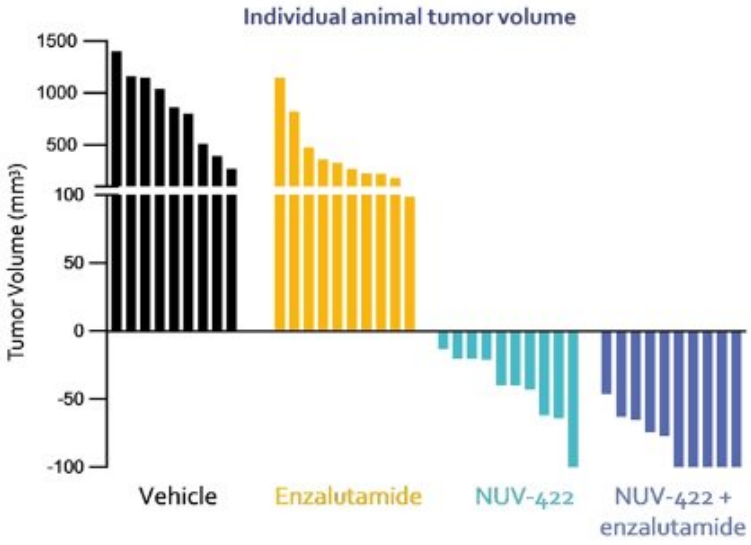
Advanced prostate cancer is associated with CDK2 overexpression

Role of CDK2/4/6 in mCRPC

- CDK2 expression increases with progression of prostate cancer and is associated with worse prognosis[2]
- Overexpression of CDK2 is associated with high probability of recurrence[2]
- CDK2 can phosphorylate and activate AR[3]
- Critical role of CDK2 as an escape mechanism for G1/S cell cycle targeting provides rationale for targeting CDK2 in addition to CDK4/6[1]

[1] Brighi et al, 2021; Schiewer et al, 2012
[2] Yin, et al 2018
[3] Jorda et al, 2018).

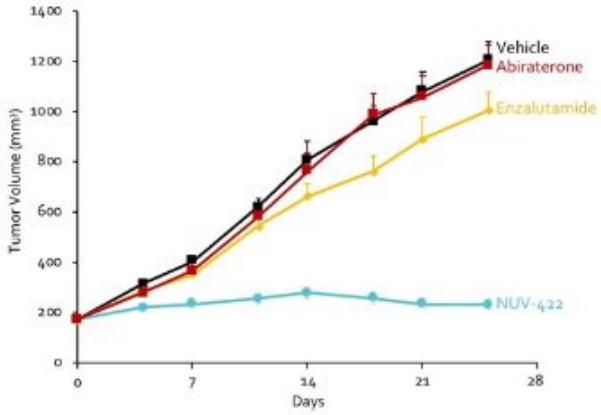
NUV-422 causes tumor regression in an enzalutamide-resistant patient derived prostate tumor xenograft



NUV-422 30 mg/kg PO QD

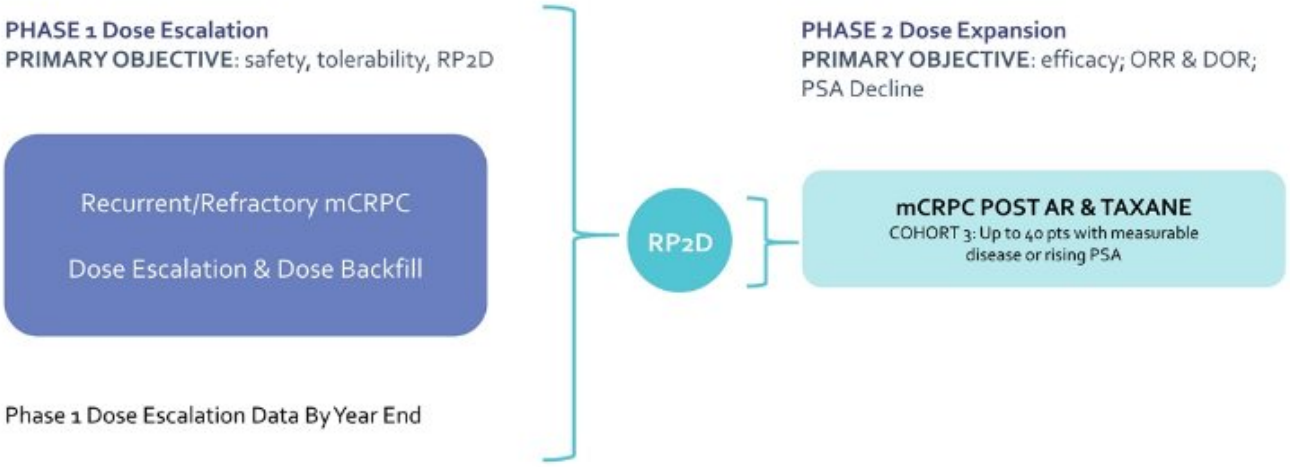
NUV-422 shows activity in a prostate cancer model resistant to standard of care

Prostate cancer ARV-7 xenograft that is resistant to standard of care anti-androgen therapies

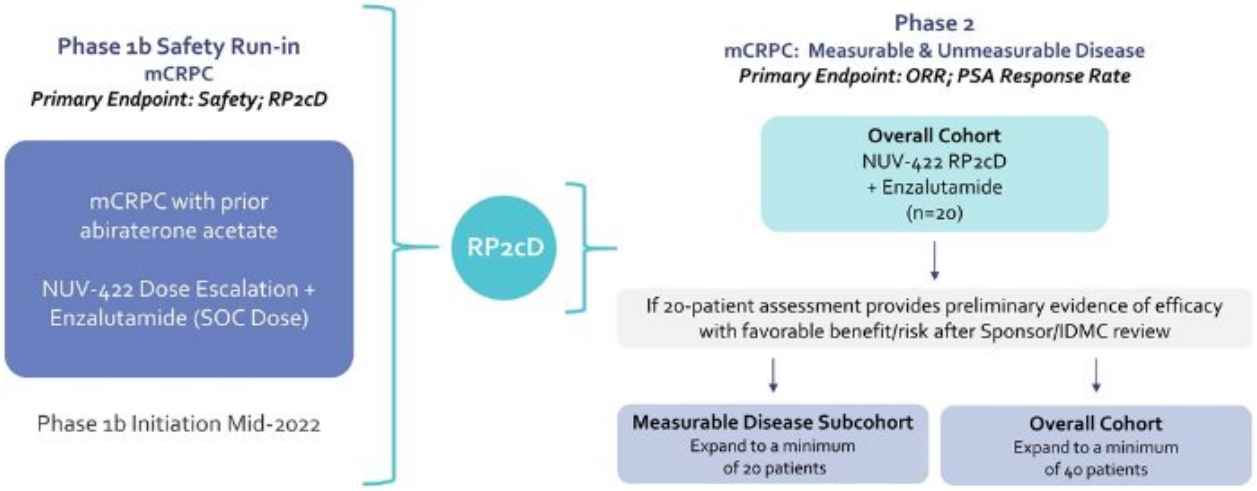


NUV-422 30 mg/kg PO QD

NUV-422-02 mCRPC monotherapy phase 1/2



NUV-422-04 phase 1b/2 study in mCRPC: NUV-422 combination with enzalutamide



RP2cD: Recommended Phase 2 Combination Dose
FUL: Fulvestrant
Overall Cohort includes Measurable and Unmeasurable mCRPC

NUV-868 | BETi

Advanced Solid Tumors

Phase 1
Initiation
Mid-2022

BET: Bromodomain and extra-terminal motif proteins

- BET are a family of proteins (e.g., BRD4) with two bromodomains (BD1 and BD2)[1]
- BET family of proteins have critical biological functions and are found to be altered in many human cancers[2]
  - BET proteins play a critical role in gene transcription[1]
- To date, BET inhibitors have largely focused on targeting both BD1 and BD2
  - Non-selective BD1/2-inhibitors in development have been associated with tolerability issues, potentially due to BD1 inhibition[3]
- Several BET inhibitors have advanced to clinical studies, but development has been limited due to PK, toxicity, or lack of efficacy[4]
  - Potential strategies to overcome development challenges include investigating BET inhibitors in combination and developing BET inhibitors with BD2 selectivity

[1]Taniguchi, 2016
[2] Bechter and Schoffski, 2020
[3]Faivre et al 2020
[4]Sun et al, 2021

BET: Bromodomain and extra-terminal motif proteins

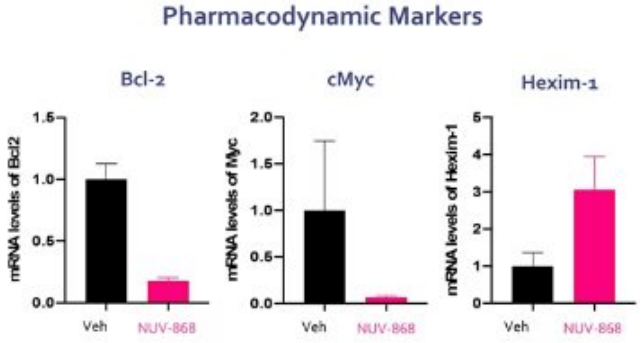
- BET are a family of proteins with two bromodomains (BD1 and BD2)
- BET proteins can induce the expression of a number of oncogenes, including MYC – an oncogene that cannot be targeted directly with a drug
- To date, BET inhibitors have largely focused on targeting both both domains (BD1 and BD2)
  - Non-selective BD1/2-inhibitors in development have been associated with tolerability issues, potentially due to BD1 inhibition[2]
- NUV-868 is a highly selective BD2 vs BD1 BET inhibitor
  - Selective BD2 vs BD1 inhibition can potentially improve tolerability but has been difficult to achieve
  - Selective BD2 inhibitors have the potential to block many oncogenes, including c-myc

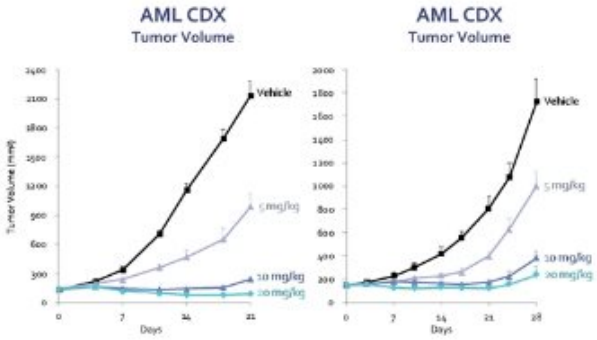
	BRD4 Affinity[2]		
	BD2	BD1	Selectivity
NUV-868	2	2920	1460x
ABBV-744[3]	1.05	340	234x
PLX-2853[4]	Modest BD2 selectivity		
CPI-0610[3]	17	85	5x
ABBV-075[4]	3	11	3.7x
MK-8628/OTX-015[5]	17	26	1.5x
BI-894999[6]	41	5	0.1x
ZEN-3694[7]	Non-selective		

LESS BD2 SELECTIVE → MORE BD2 SELECTIVE

1. Faivre et al 2020; 2. Various assays used; 3. Internal Nuvation Bio data; 4. https://ash.confex.com/ash/2020/webprogram/Paper134038.html; 5. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5474081/; 6. https://www.nature.com/articles/s41388-018-0550-7; 7. 2016 EORTC poster ZenithEpigenetics.pdf

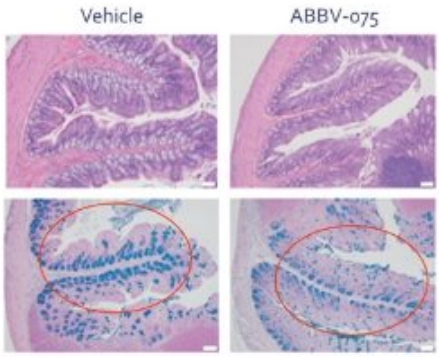
NUV-868 inhibits tumor growth by down regulating tumor promoting genes BCL-2 and MYC and up regulating tumor suppressor Hexim-1





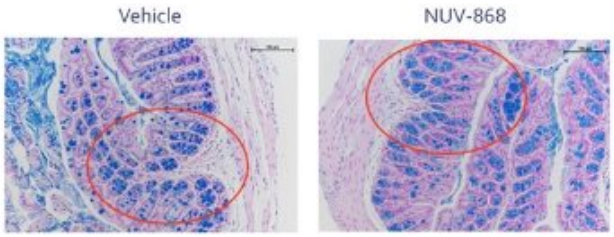
High selectivity for BD2 over BD1 significantly reduces the gut toxicity observed with other nonselective BET inhibitors

ABBV-075 (Dual BD1 / BD2)



× A non-selective inhibitor (ABBV-075) leads to marked reduction in rat small intestine goblet cells[1]

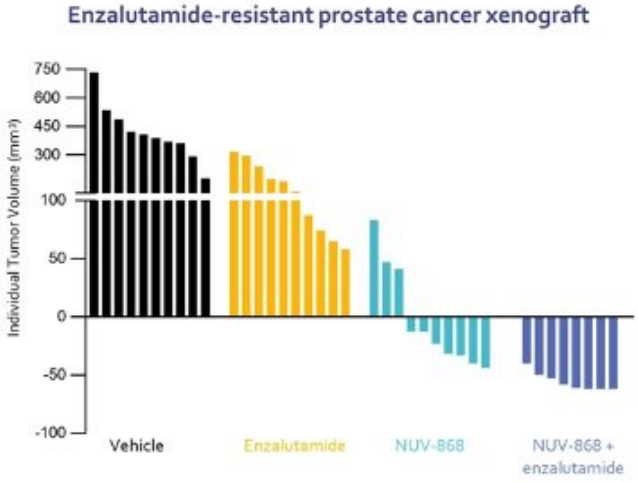
NUV-868 (BD2 Selective) May Avoid GI Toxicity



✓ Treatment of mice for 10 days with BD2 selective compound NUV-868 shows no evidence of goblet cell loss

[1]Faivre et al 2020 Nat 578

NUV-868 causes tumor reductions in an enzalutamide-resistant patient-derived prostate cancer xenograft model



NUV-868 20 mg/kg QD

BET inhibitors (BRD4) cause sensitization of HR-proficient cancers to PARP-inhibitors

SCIENCE TRANSLATIONAL MEDICINE | RESEARCH ARTICLE

CANCER

Repression of BET activity sensitizes homologous recombination–proficient cancers to PARP inhibition

Lu Yang,[1,2]* Youyou Zhang,[1]* Weiwei Shan,[1,2] Zhongyi Hu,[1] Jiao Yuan,[1] Jingjiang Pi,[1] Yueying Wang,[1] Lingling Fan,[1,3] Zhaoqing Tang,[1] Chunsheng Li,[1,4] Xiaowen Hu,[1,5] Janos L. Tanyi,[4] Yi Fan,[6] Qihong Huang,[6] Kathleen Montone,[7] Chi V. Dang,[8] Lin Zhang[1,4,8†]

Cancer Cell

Article

BRD4 Inhibition Is Synthetic Lethal with PARP Inhibitors through the Induction of Homologous Recombination Deficiency

Chaoyang Sun,[1,6,10,*] Jun Yin,[2,3] Yong Fang,[1,4] Jian Chen,[5,6] Kang Jin Jeong,[2] Xiaohua Chen,[2] Christopher P. Vellano,[2] Zhenlin Ju,[1] Wei Zhao,[2] Dong Zhang,[1] Yiling Lu,[2] Funda Meric-Bernstam,[8] Timothy A. Yap,[9] Maureen Hattersley,[1] Mark J. O'Connor,[1] Huawei Chen,[1] Stephen Fawell,[1] Shiaw-Yih Lin,[2] Guang Peng,[3] and Gordon B. Mills[2]

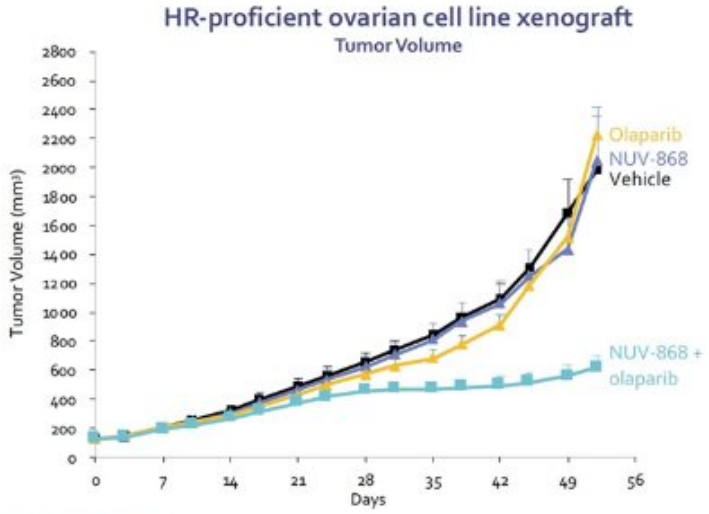
Published in final edited form as:

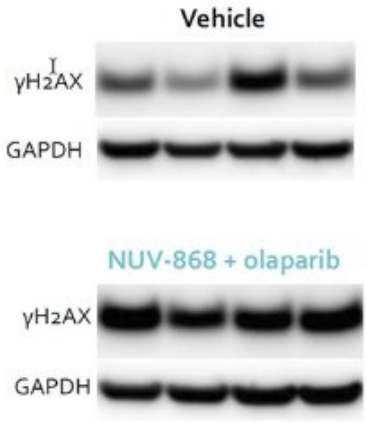
Cell Rep. 2017 December 19; 21(12): 3398–3405. doi:10.1016/j.celrep.2017.11.095.

BET bromodomain inhibition synergizes with PARP inhibitor in epithelial ovarian cancer

Sergey Karakashev[1,3], Hengrui Zhu[1,3], Yuhki Yokoyama[1,4], Bo Zhao[1], Nail Fatkhutdinov[1,2], Andrew V. Kossenkov[5], Andrew J. Wilson[1], Fiona Simpkins[6], David Speicher[2,6], Dineo Khabele[7], Benjamin G. Bitler[1], and Rugang Zhang[1,7,*]

Combination of NUV-868 + olaparib increases double stranded DNA breaks (γH2AX) in an HR-proficient ovarian tumor model





NUV-868 20 mg/kg QD

NUV-569 | WEE1i

Advanced Solid Tumors

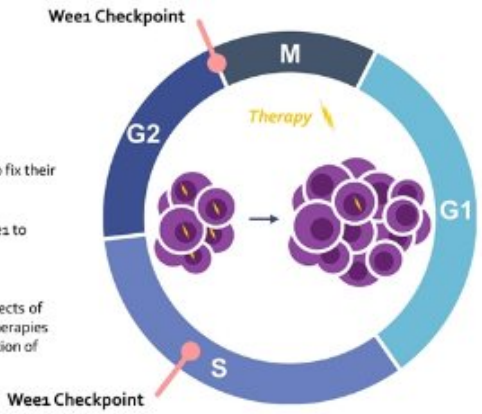
IND Submission by Year End 2022

Wee1 inhibitors increase the efficacy of DNA-damaging therapies by forcing cancers to replicate before they can repair their DNA

TUMOR GROWTH

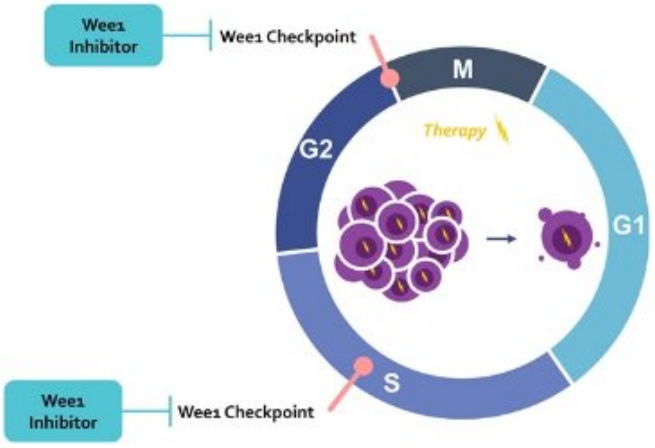
Tumors activate their Wee1 checkpoint to:

- Arrest their own replication
- Give them time to fix their damaged DNA
- Turn off their Wee1 to re-initiate growth
- Potentiate the effects of DNA damaging therapies by forcing replication of damaged DNA



REPLICATING DAMAGED DNA CAUSES CELL DEATH

Wee1 inhibitors may potentiate any therapy that causes DNA damage (chemotherapy or radiation)



NUV-569's Highly Potent and Selective profile = less toxicity

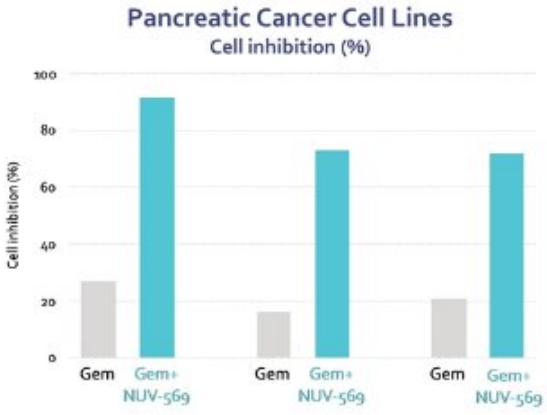
Compound	Wee1	PLK1	IEC6
NUV-569	7	687	2362
AZD1775	4	15	251

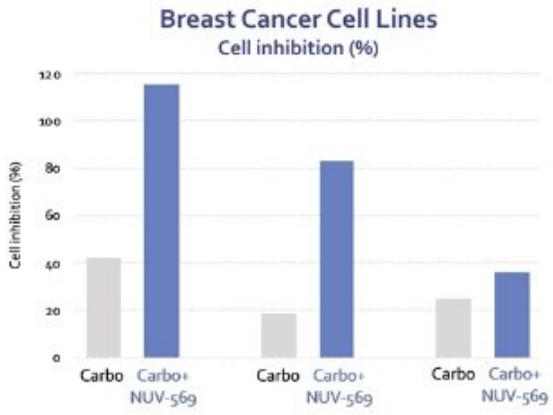
IC_{50} (nM)

PLK1 is a ubiquitous cell kinase that may be responsible for gut and bone marrow toxicity

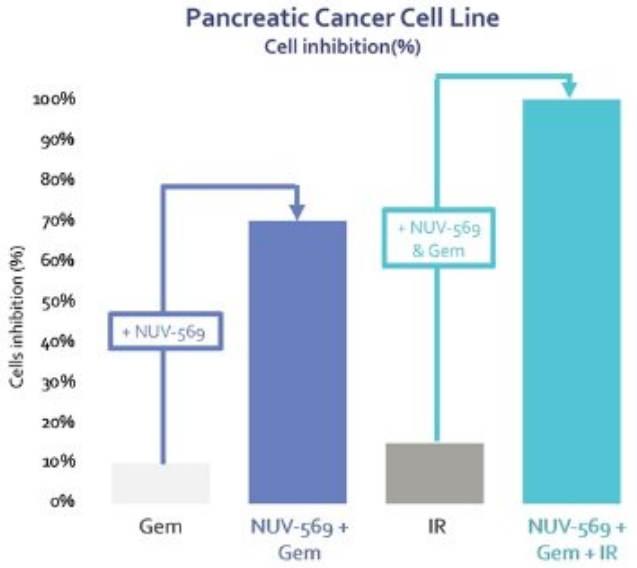
- NUV-569 is highly potent against Wee1 but avoids PLK1 unlike AZD1775
- 10X reduced potency on rat gut epithelial cells (IEC6), relative to AZD1775, suggests these new compounds have significantly improved tolerability

NUV-569 synergizes with SOC gemcitabine in pancreatic cancer cells and carboplatin in breast cancer cells to enhance cancer cell death



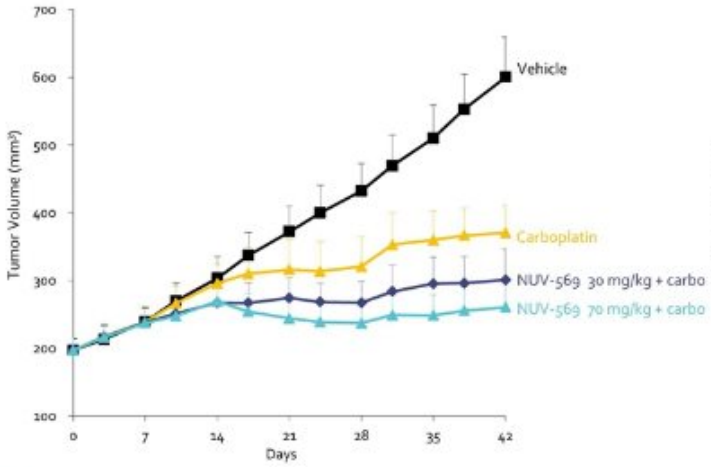


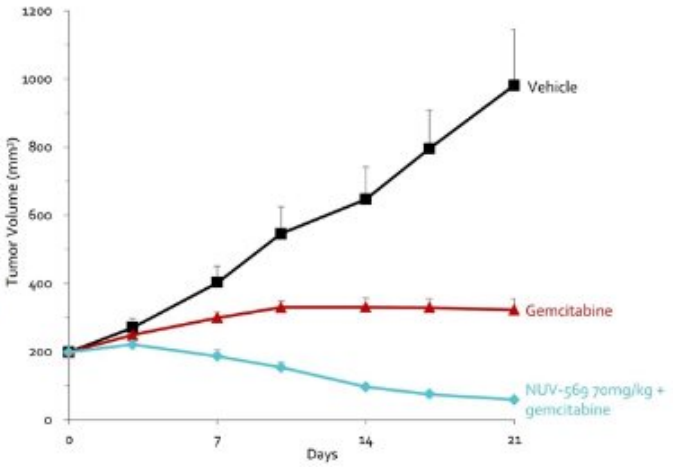
NUV-569 further enhances cancer cell death in combination with both gemcitabine and radiation



NUV-569 synergizes with SOC carboplatin and gemcitabine to inhibit tumor growth in breast cancer xenografts

Tumor Volume





Drug-Drug Conjugate (DDC) Platform

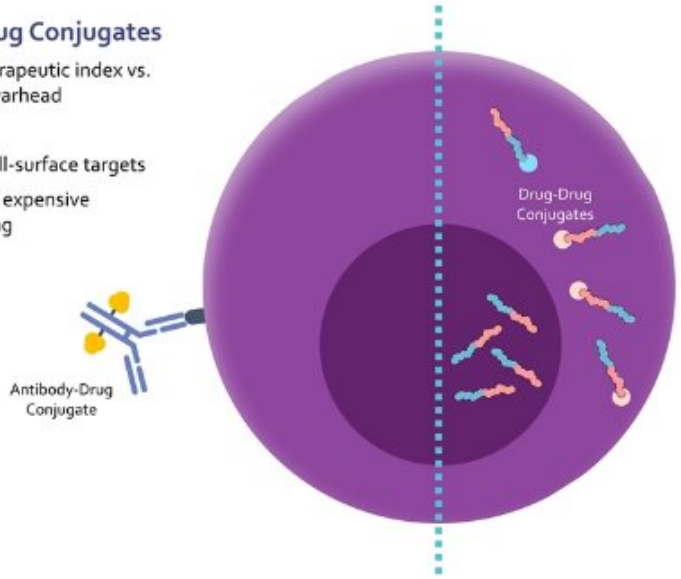
Solid Tumors

Clinical Candidate Selection By Year End 2022

The drug-drug conjugate (DDC) platform is a potentially revolutionary advance beyond ADCs

Antibody-Drug Conjugates

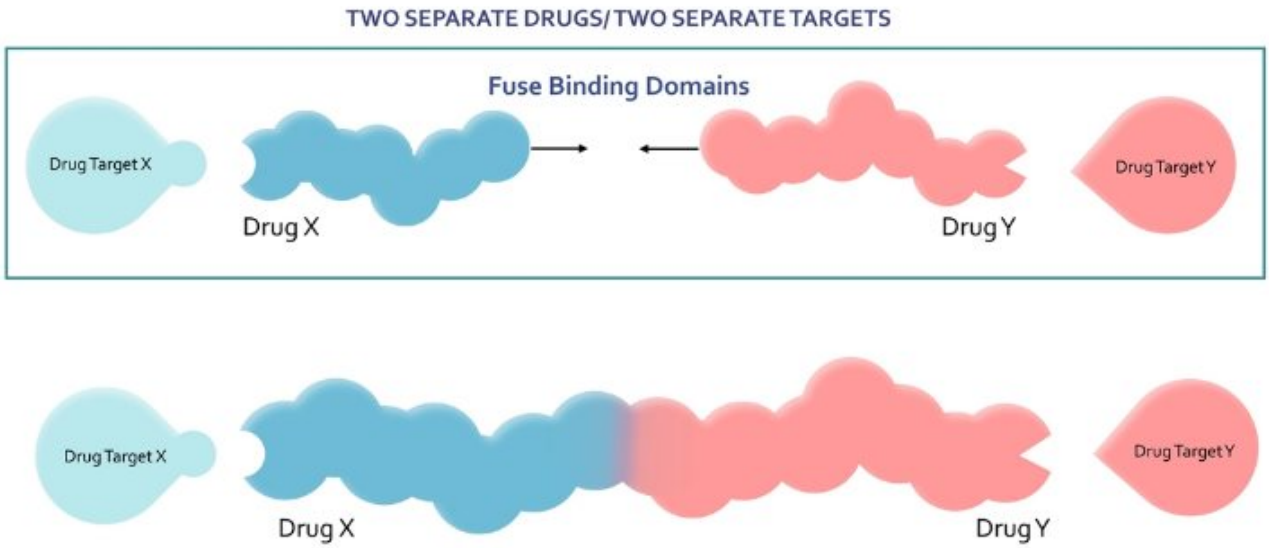
- ✓ Improves therapeutic index vs. untargeted warhead
- × IV delivery
- × Limited to cell-surface targets
- × Complex and expensive manufacturing



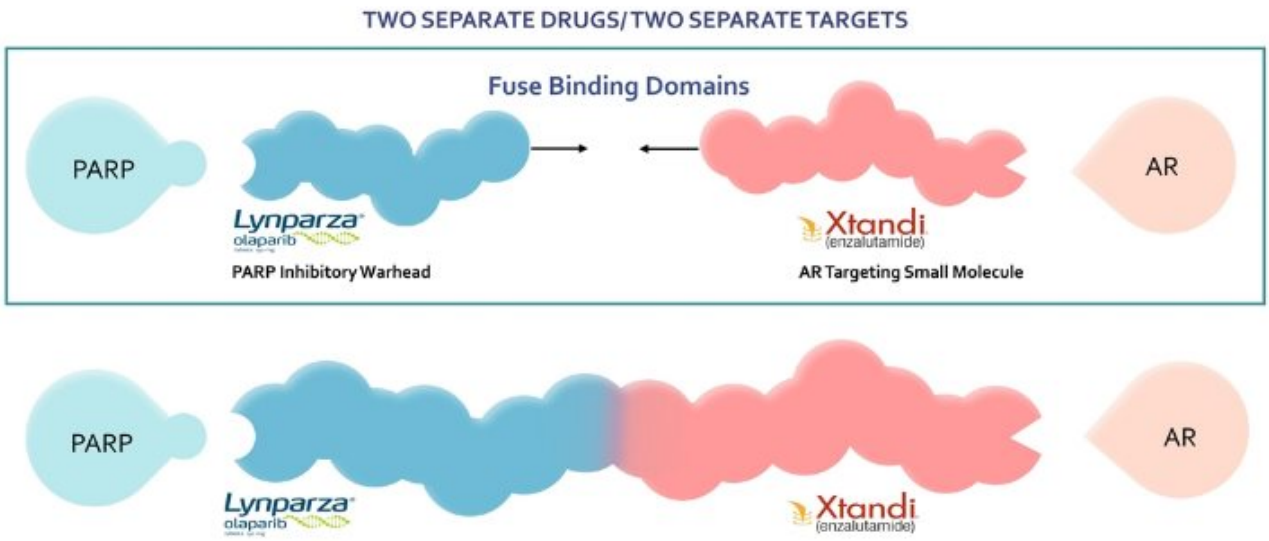
Drug-Drug Conjugates

- ✓ 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

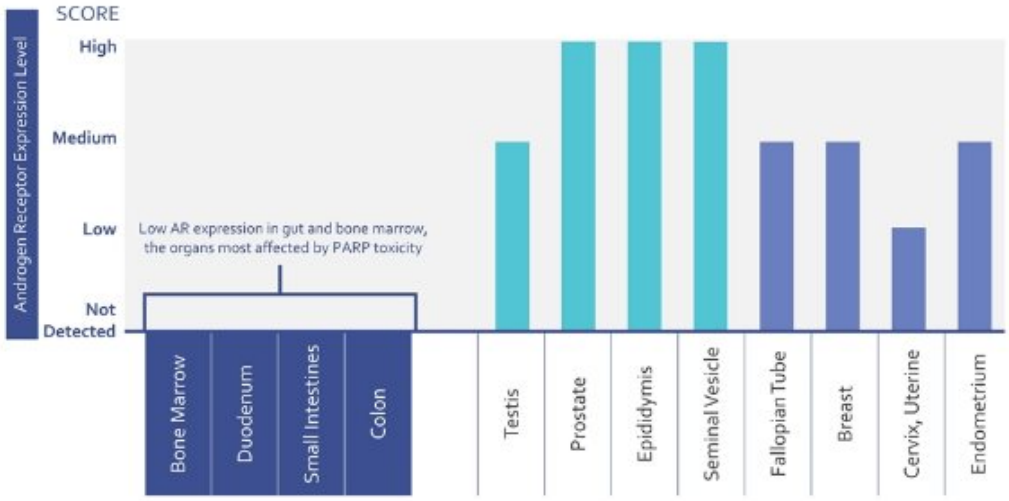
DDCs are designed to bind TWO different targets simultaneously



NUV-1156 is a novel drug-drug conjugate that targets AR and PARP



NUV-1156 targets high AR-expressing tissue like prostate cancer and avoids low AR-expressing tissue like bone marrow and GI tract

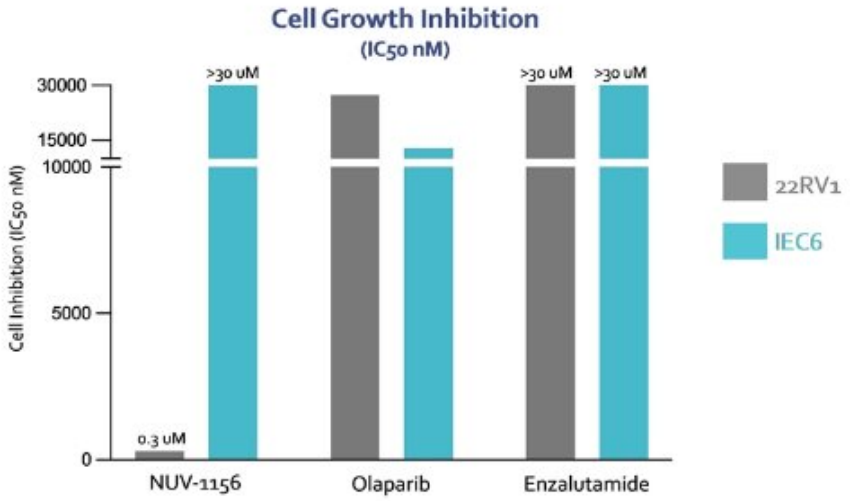


NUV-1156 DDC potently kills prostate cancer cells resistant to current standards of care

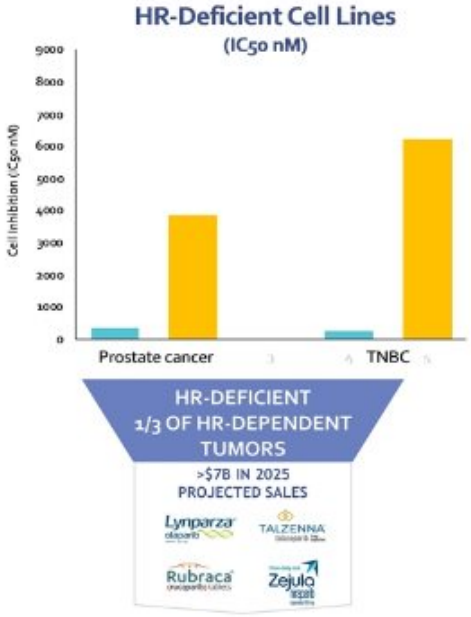
	CELL PROLIFERATION IC_{50} (nM)
Xtandi (enzalutamide)	>30,000
Lynparza olaparib	7844
Xtandi (enzalutamide) + Lynparza olaparib	6152
NUV-1156 (PARP-AR DDC)	**201**

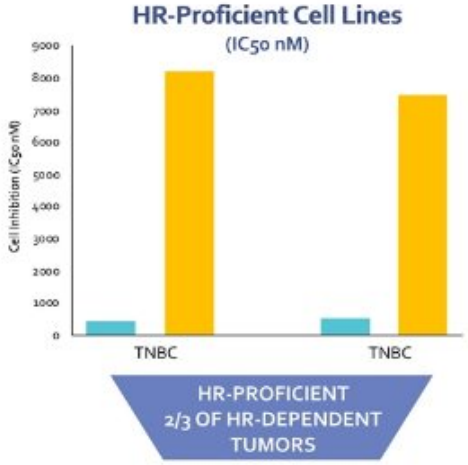
NUV-1156 is >100-fold more potent at inhibiting cell growth in prostate cancer 22RV1 cells than in IEC6 gut epithelial cells

Approved PARP inhibitors have high rates of GI toxicity

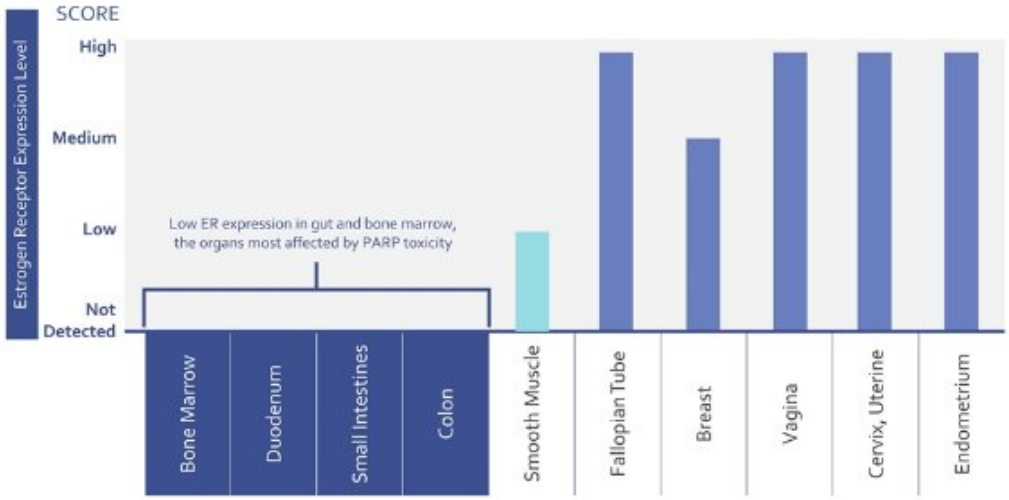


Unlike current PARP inhibitors, NUV-1156 kills HR-Deficient and HR-proficient cancer cell lines with equally high potency

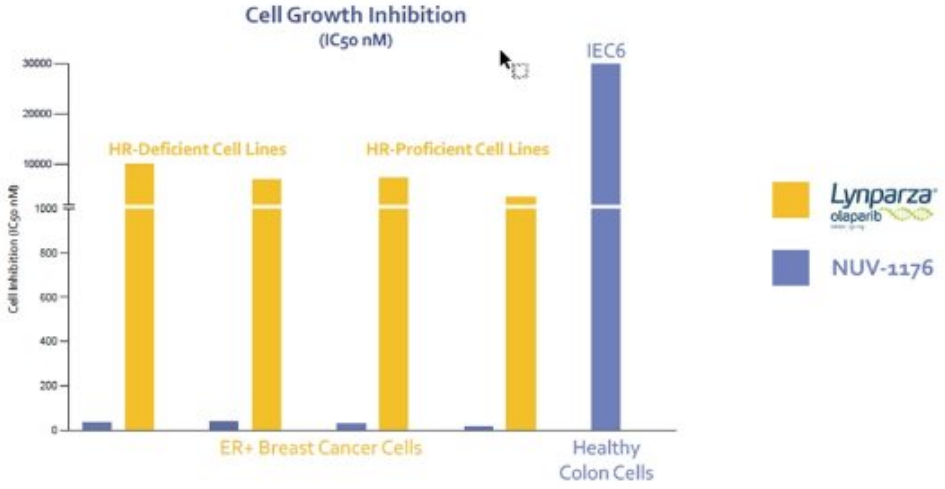




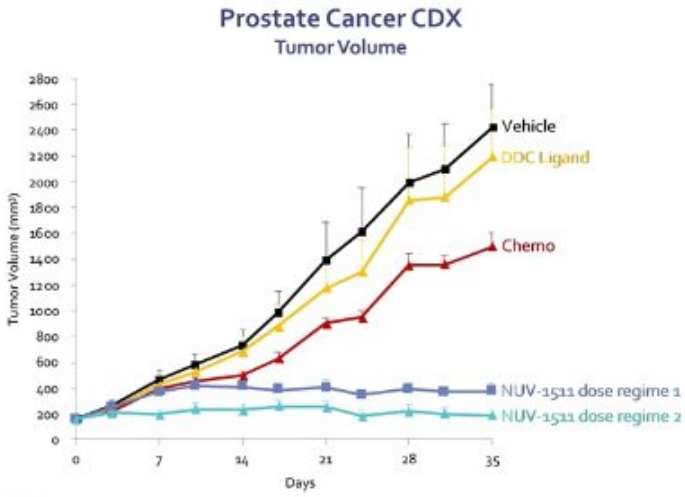
ER protein expression is limited to female sex organs; Low ER expression in sites of PARP-related toxicity like bone marrow and GI tract

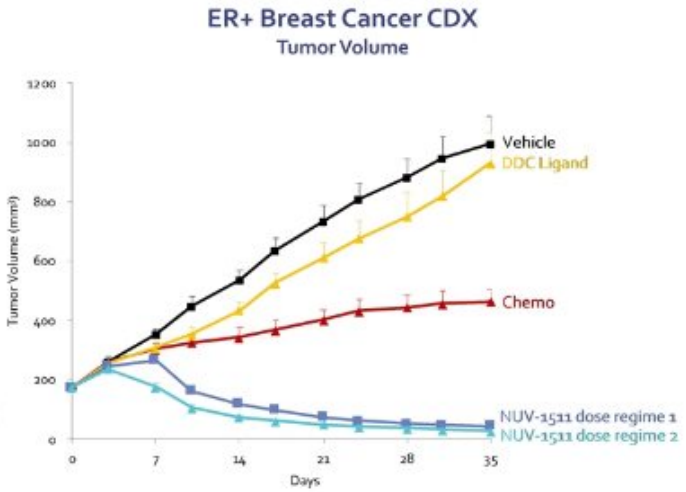


NUV-1176, an ER-targeted DDC, potently kills both HR-D and HR-P ER+ breast cancer cells without killing healthy gut epithelial cells



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





Adenosine Antagonist

Advanced Solid Tumors with IO

Clinical Candidate Selection by Year End 2022

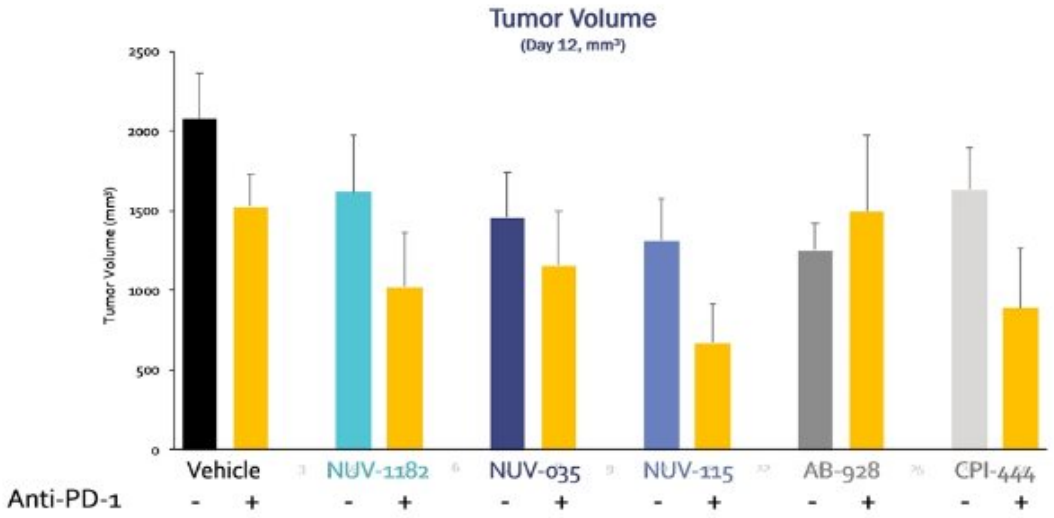
NUV-1182, an A2A adenosine receptor inhibitor, boosts immune function and may enhance the efficacy of IO-targeted therapies

- NUV-1182 targets the A2A adenosine receptor
- A2A adenosine receptor plays multiple critical roles in human physiology and pathophysiology including anti-cancer immunity
- Accumulation of adenosine in the tumor microenvironment may be a critical factor in limiting the activity of currently available immune-oncology drugs, including anti-PD1/PD-L1 drugs and anti-cancer chimeric T cells.
- Targeting A2A may overcome this blockade, leading to improved anti-cancer activity in tumors which are resistant to immuno-oncology drugs and T cell therapies.

NUV-1182 is a potent and selective A2A vs A1 adenosine receptor inhibitor

IC50 (nM)	AZD4635	CPI444	AB928	NUV-115	NUV-035	NUV-1182
A2A binding	~20	~7	~3	~1	~3	~3
A1 binding	>300	>100	>10	~2	~10	>200
A2A Selectivity (A2A/A1)	~20	~15	~10	~2	~4	>75
A2A cAMP (40 nM NECA)	>100	>50	>10	>10	>10	>50

Nuvation A2A inhibitors increase the anti-tumor activity of immune checkpoint inhibitors in an *in vivo* melanoma xenograft model



Upcoming Milestones & Summary

Nuvation Bio

Broad wholly-owned pipeline leveraging and improving upon validated drug mechanisms heading into multiple clinical trials/indications

Program	Product Candidate	Potential Indication(s)		Current Stage			Anticipated Milestones
				Preclinical	Phase 1	Phase 2	
CDK 2/4/6	NUV-422	Glioblastoma	Recurrent GB				Phase 1 Dose Escalation Data by Year End 2022; Phase 2 Initiation by Year End 2022
		Breast Cancer	2L + aBC Mono				Phase 2 Initiation by Year End 2022
			2L+ aBC Brain Mets				Phase 2 Initiation by Year End 2022
			2L/3L aBC + Fulvestrant				Phase 1b Initiation Mid-2022
		Prostate Cancer	mCRPC Mono				Phase 2 Initiation by Year End 2022
			mCRPC + Enza				Phase 1b Initiation Mid-2022
BET	NUV-868	Advanced Solid Tumors					Phase 1 Initiation Mid-2022
WEE1	NUV-569	Advanced Solid Tumors					IND Submission by Year End 2022
Adenosine Antagonist	A2A	Advanced Solid Tumors with IO					Clinical Candidate Selection by Year End 2022
Drug-Drug Conjugate (DDC) Platform	DDC	Solid tumors					Clinical Candidate Selection by Year End 2022

Upcoming catalysts across multiple programs

	1H22		2H22		2023
CDK2/4/6	Initiate Phase 1b combo study in HR+ aBC	Initiate Phase 1b combo study in mCRPC	Phase 1 Dose Escalation Data	Phase 2 Mono Initiation for rGB, HR+ aBC, & mCRPC	Present First Efficacy Data from High-grade Glioma trial
BET	Phase 1 Initiation				
WEE1	IND Submission				
A2A	Clinical Candidate Selection				
DDC	Clinical Candidate Selection				

Committed team tackling the greatest unmet needs in oncology

Experienced Biotech Leadership Team

Founded in 2018 by Dr. David Hung, previously the founder and CEO of Medivation and successful developer of major oncology drugs (XTANDI & TALZENNA)

Best-in-class Drug Candidate Profiles Leveraging and Improving Validated Drug Mechanisms

Potential for better efficacy and tolerability

Mechanisms that target multiple tumor types

Potential for accelerated approval pathways

Broad Wholly-Owned Pipeline

3 INDs cleared for NUV-422, a CDK2/4/6 inhibitor, with ongoing studies in brain, breast and prostate cancer

NUV-868, a BD2 selective BET inhibitor, entering the clinic for solid tumors

Nuv-569, a selective Wee1 inhibitor, declared a clinical candidate

A2A and DDC candidates advancing

Comprehensive IP protection

Strong Cash Position

~$765 million as of December 2021

Enables a world-class drug development team to rapidly pursue clinical development of multiple portfolio therapeutic candidates