Nuvation Bio

FEBRUARY 2021

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Nuvation Bio Overview & Pipeline

TACKLING THE GREATEST UNMET NEEDS IN ONCOLOGY

- Experienced biotech leadership team led by David Hung, which successfully developed major oncology drugs including Xtandi ($3.7B in 2019 sales) and Talzenna
- Broad wholly-owned pipeline with strong IP protection
  - First patient dosed with NUV-422 in Phase 1/2 High-grade Glioma trial in December 2020
  - Up to 5 INDs to be filed in next 6 years
  - Potential for accelerated pathways in multiple programs
  - Comprehensively protected by composition of matter filings for 28 compound families with normal expirations dates from 2038-2041
- Leveraging and improving upon validated drug mechanisms
- Best-in-class drug candidate profiles vs. competitors
- Strong cash position with approximately $830M

HIGHLY EXPERIENCED MANAGEMENT TEAM & BOARD

LEADERSHIP TEAM

DAVID
HUNG, M.D.*
Founder, President and Chief Executive Officer

SERGEY
YURASOV, M.D., Ph.D.
Chief Medical Officer

JENNIFER
FOX
Chief Financial Officer

GARY
HATTERSLEY, Ph.D.
Chief Scientific Officer

THOMAS
TEMPLEMAN, Ph.D.
SVP of Pharmaceutical Operations and Quality

LISA
DeLUCA, Ph.D.
SVP of Regulatory Affairs

STACY
MARKEL
SVP of Human Resources

BOARD OF DIRECTORS

DAN
WELCH
Former Chairman and CEO, InterMune

ROBERT
BAZEMORE
CEO, Epizyme

KIM
BLICKENSTAFF
Executive Chairman and Director, Tandem Diabetes Care

KATE
FALBERG
Former CFO, Jazz Pharmaceuticals

W. ANTHONY
VERNON
Former CEO, Kraft Foods Group

OLEG
NODELMAN
Founder and Portfolio Manager, EcoR1

MICHELLE
DOIG**
Partner and Head of Corporate Development, Omega Funds

- *Also on the Board of Directors
- ** Stepping down from the Board on June 1, 2021

DEEP PIPELINE TARGETING MULTIPLE ONCOLOGY INDICATIONS

Program	Product Candidate	Potential Indication(s)	Preclinical	Phase 1	Phase 2	Phase 3	Anticipated Milestones
CDK 2/4/6	NUV-422	High-grade Glioma					December 2020 First patient dosed in Phase 1/2 High-grade Glioma trial
		Brain metastases					1H22 Initiate Phase 1 Brain metastases trial
		ER+ mBC					2H22 Initiate Phase 1 ER+ mBC trial
		mCRPC					4Q22 Initiate Phase 1 mCRPC trial
BET	NUV-868	Acute Myeloid Leukemia/solid tumors					1H22 Initiate Phase 1 AML and/or solid tumor trial
WEE1	NUV-569	Pancreatic Cancer/other solid tumors					2H22 Initiate Phase 1 Pancreatic Cancer and/or other solid tumor trial
A2A	NUV-1182	Solid Tumors with IO					4Q22 Initiate Phase 1 IO combination trial
Drug-Drug Conjugate (DDC) Platform	DDC1 (PARP – AR)	Prostate Cancer					2H22 Nominate first DDC candidate
	DDC2 (PARP – ER)	Breast Cancer and Ovarian Cancer					

NUV-422 | CDK 2/4/6

High-grade Glioma	Dec 2020 First patient dosed
Brain Metastases	1H22 Initiate Ph1 trial
ER+ mBC	2H22 Initiate Ph1 trial
mCRPC	4Q22 Initiate Ph1 trial

CDK2 DRIVES RESISTANCE TO CDK4/6 INHIBITORS

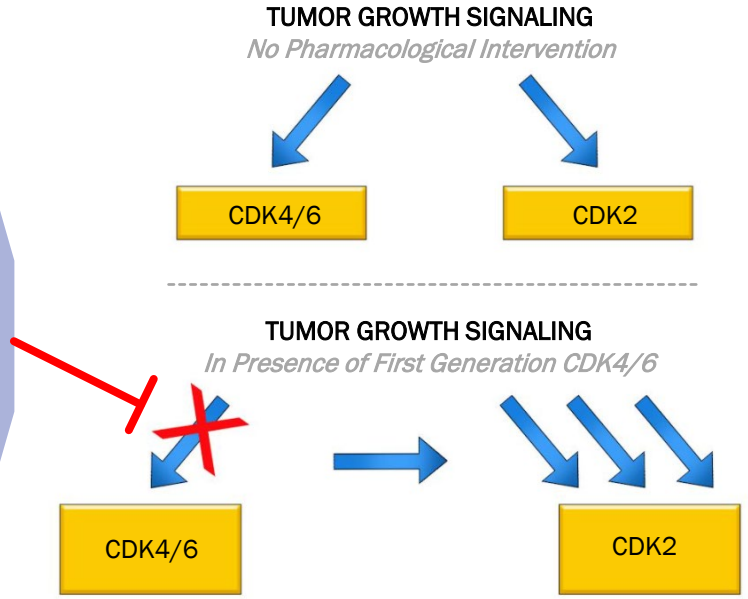
CDK4/6 Inhibitors

IBRANCE palbociclib

KISQALI ribociclib 200 mg tablets

Verzenio abemaciclib

- Three approved CDK4/6 inhibitors
- CDK4/6 class achieved revenues > $6B in 2019 class expected to grow to ~$14B in 2025



Addressing Resistance

- Tumors respond to CDK4/6 inhibition by signaling through CDK2, driving resistance
- Selectively targeting CDK2 in addition to CDK4/6 may prevent or reverse resistance

NUV-422 IS A POTENT INHIBITOR OF CDK2/4/6 THAT AVOIDS CDK1

	DRIVES EFFICACY			CAUSES TOXICITY
1st Generation	CDK 4	CDK 6	CDK 2	CDK 1
KISQALI® ribociclib 200 mg tablets	2	2	10000	*10000*
IBRANCE® palbociclib	4	2	2470	*10000*
Verzenio® abemaciclib	2	10	504	*1627*
2nd Generation	**CDK 4**	**CDK 6**	**CDK 2**	**CDK 1**
PF-06873600	2	4	0.3	*2*
NUV-422	2	1	7	*73*

IC_{50} (nM)

NUV-422 has good drug-like properties

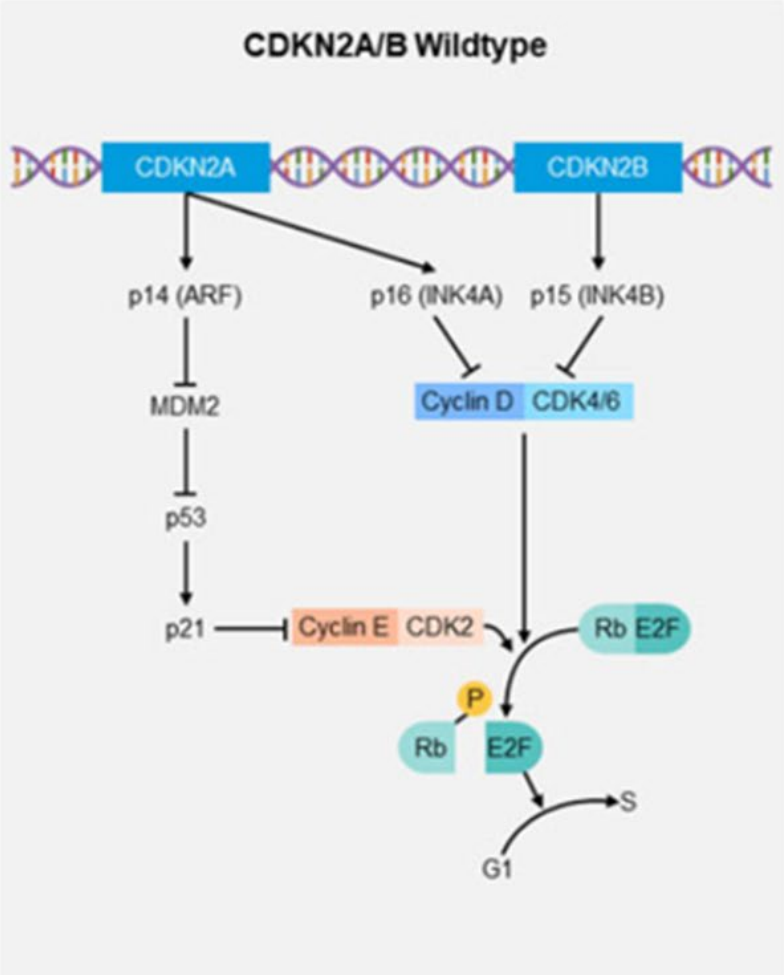
- Target selectivity
- Good oral PK
- Good CYP profile
- Scalable manufacturing process

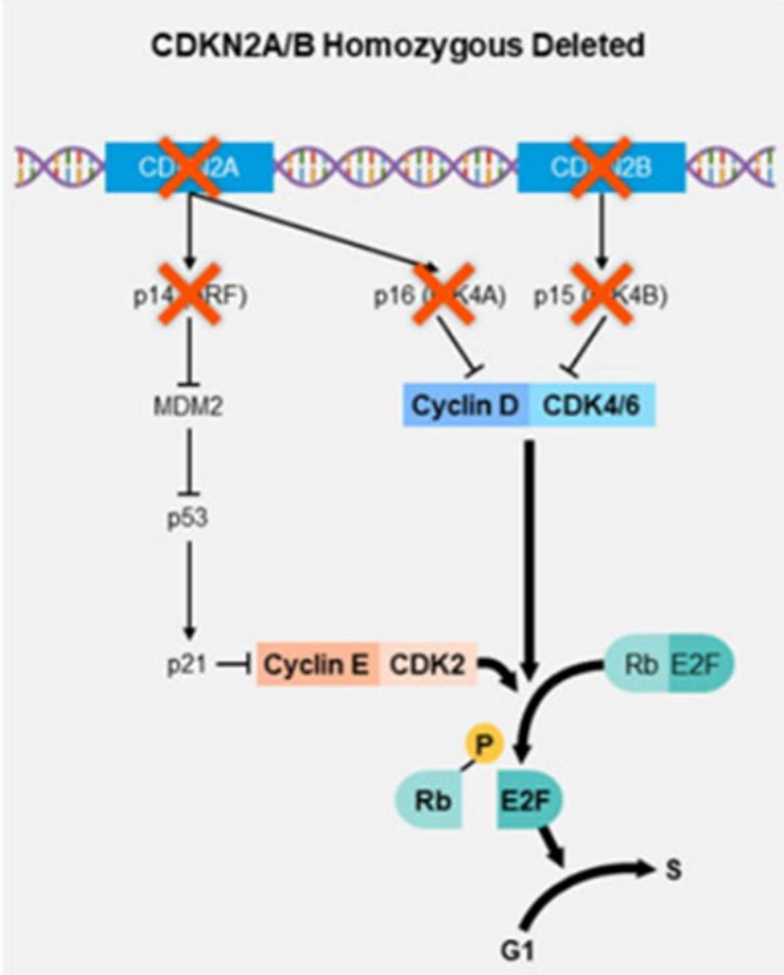
CDK2 ACTIVITY MAY EXPLAIN THE DISCORDANT RESULTS IN BREAST CANCER TRIALS BETWEEN FIRST-GENERATION CDK4/6 INHIBITORS

	IC50 (nM)					
	CDK2	CDK4	CDK6	Metastatic *Monotherapy Label*	Adjuvant Setting	
IBRANCE® palbociclib	2,470	4	2	✗	✗	PALLAS
					✗	PENELOPE-B
Verzenio® abemaciclib	504	2	10	✓	✓	monarch-E

- Abemaciclib inhibits CDK2 approximately 5x more potently than Palbociclib
- Abemaciclib's greater potency against CDK2 may explain the discordant results in the recently reported adjuvant breast cancer trials

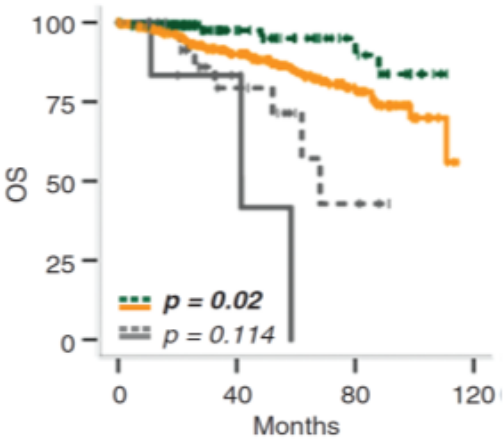
CDKN2A DELETION OR ALTERATIONS COMMONLY DRIVE CANCER GROWTH THROUGH CDK2/4/6



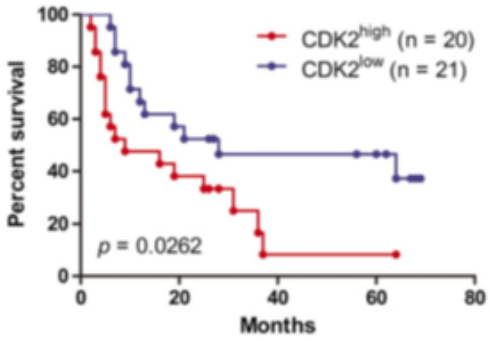


CDKN2A DELETION (DRIVING CDK 2/4/6) AND CDK2 SPECIFICALLY DRIVES GROWTH OF PRIMARY HIGH-GRADE GLIOMAS

CDKN2A Deletion is Associated With Worse Survival [1]



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



- Recent data from abemaciclib (Eli Lilly) in GBM patients supports targeting CDK2 may play an important role in controlling disease progression in HGG

(1) Appay et al., 2020
(2) Wang et al., 2016

INSIGHT PHASE 2 TRIAL SHOWED THAT WHILE ABEMACICLIB IMPROVES PFS IN GBM PATIENTS, THIS EFFECT IS NOT DUE TO CDK4 ACTIVITY

Abemaciclib (a potent inhibitor of CDK4/6 and a weak inhibitor of CDK2) demonstrated an improvement of progression-free survival (PFS) in newly diagnosed GBM patients in Phase 2[1]

More than 60% of GBM patients exhibit alteration of CDKN2 which regulates CDK2, CDK4, CDK6 [2]

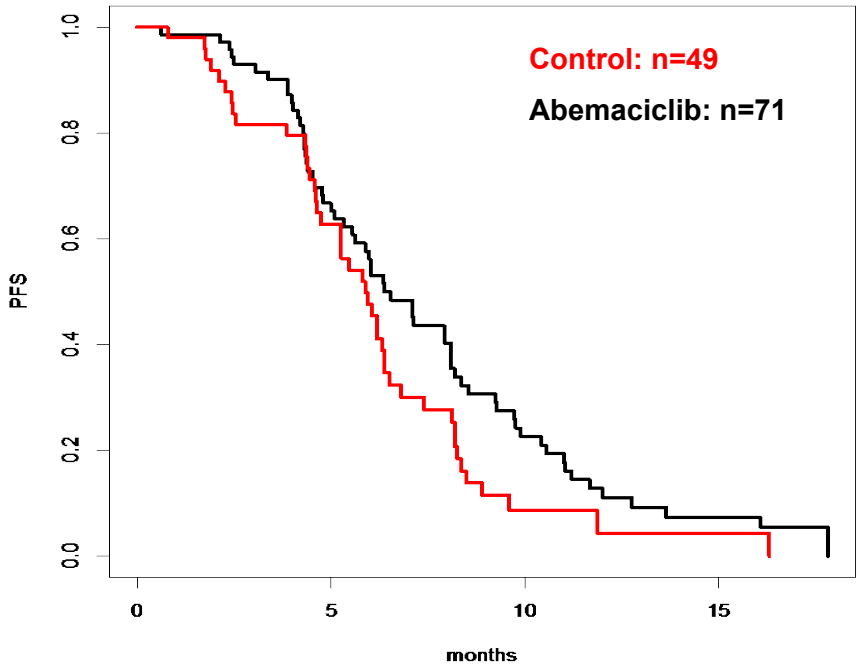
PFS was significantly longer with abemaciclib when compared to temozolomide containing control arm:

- Hazard ratio: 0.68
- p-value 0.03 (one sided hypothesis testing; log rank test)

However, there was no evidence of a positive treatment and CDK4 biomarker interaction:

- p-value: 0.84

CDK2 activity may be an important driver of tumor growth in GBM

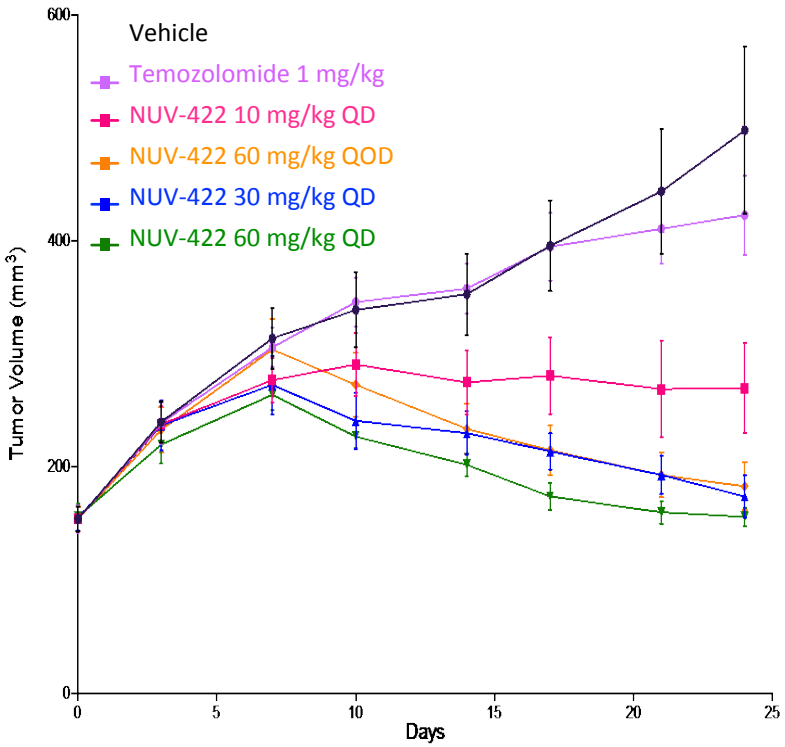


- NUV-422 is a potent inhibitor of CDK2 in addition to CDK4/6, and thus is expected to improve response in GBM patients

(1) Wen PY, et al. SNO meeting. 2020; (2) Brennan et al. Cell 2013; 155, 462–477

NUV-422 IS SUPERIOR TO STANDARD OF CARE TEMOZOLOMIDE IN *IN VIVO* XENOGRAFT MODEL OF GLIOBLASTOMA MULTIFORME

Tumor Volume



NUV-422 ACHIEVES HIGH CONCENTRATIONS IN THE BRAIN

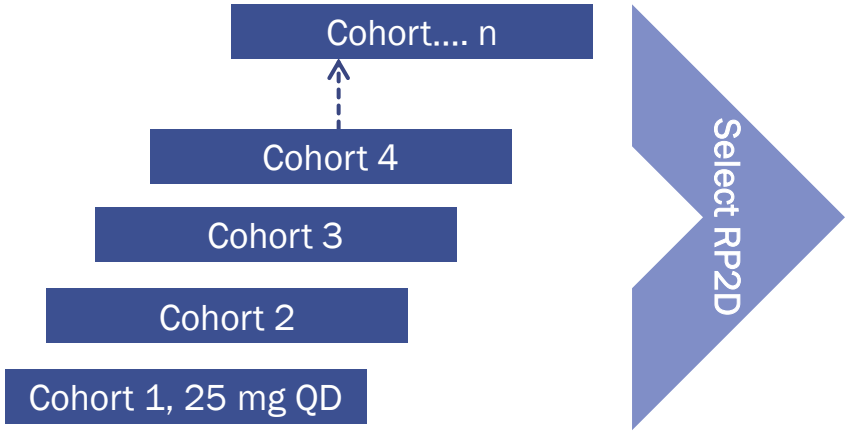
NUV-422 Concentration Six Hours Post Dose (Rat)

Dose (mg/kg)	Brain Conc (nM)	Plasma Conc (nM)	Brain / Plasma Ratio
30	4096	375	11
100	5827	506	12

12X Higher Exposure in Brain vs Plasma

NUV-422-02: SEAMLESS PHASE 1/2 TRIAL DESIGN IN HIGH GRADE GLIOMAS

Ph1 Dose-Escalation in Unselected Population



OBJECTIVES
Safety and Tolerability
Determine RP2D
PK, food effect

Ph2 Dose Expansion in CDKN2A Deleted Patients

EXPANSION COHORT 1

CDKN2A deleted relapsed/refractory high-grade glioma (up to 40 pts) with **measurable disease**

EXPANSION COHORT 2

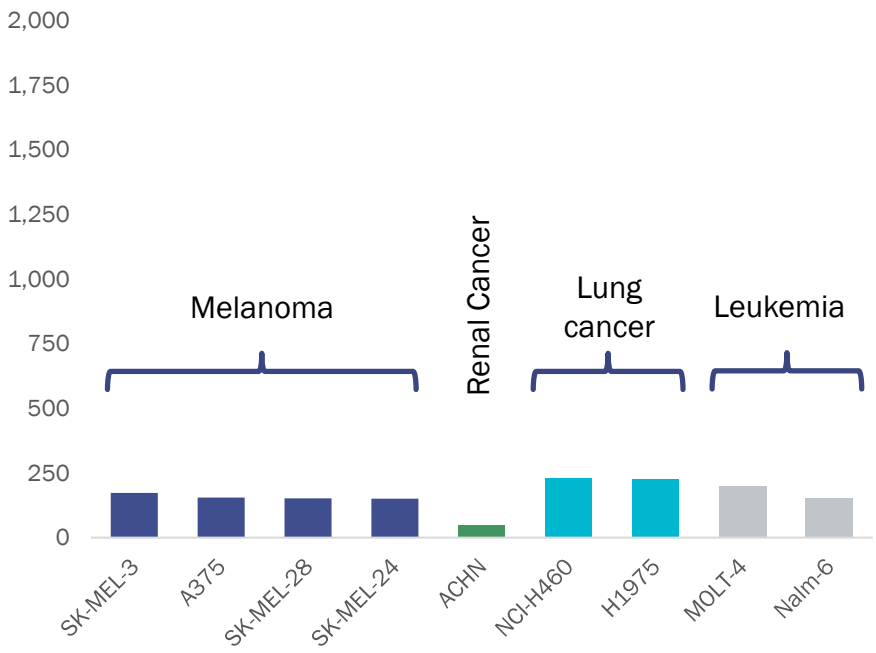
CDKN2A deleted relapsed/refractory high-grade glioma (up to 10 pts) **eligible for surgery (window of opportunity)**

OBJECTIVES
Safety and Tolerability
ORR DoR, PFS, OS
PK/PD

BEYOND PRIMARY BRAIN TUMORS, NUV-422 IS ACTIVE IN MULTIPLE CELL LINES OF CANCERS WHICH COMMONLY METASTASIZE TO BRAIN

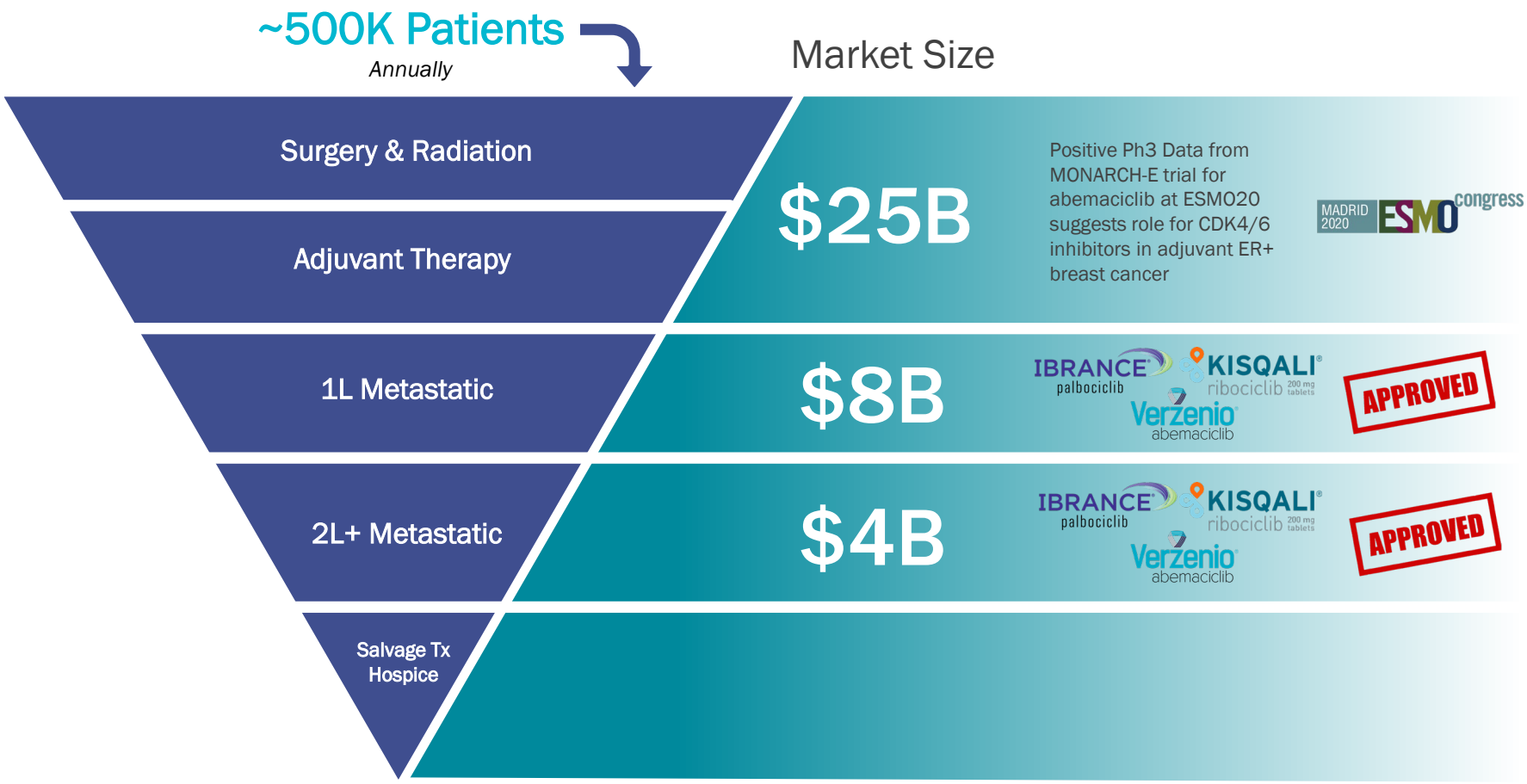
Tumor Types with Brain Mets
Breast
Colon
NSCLC
Melanoma
CDKN2A Deleted Solid Tumors (e.g., pancreas)

Cell Proliferation (IC_{50} nM)



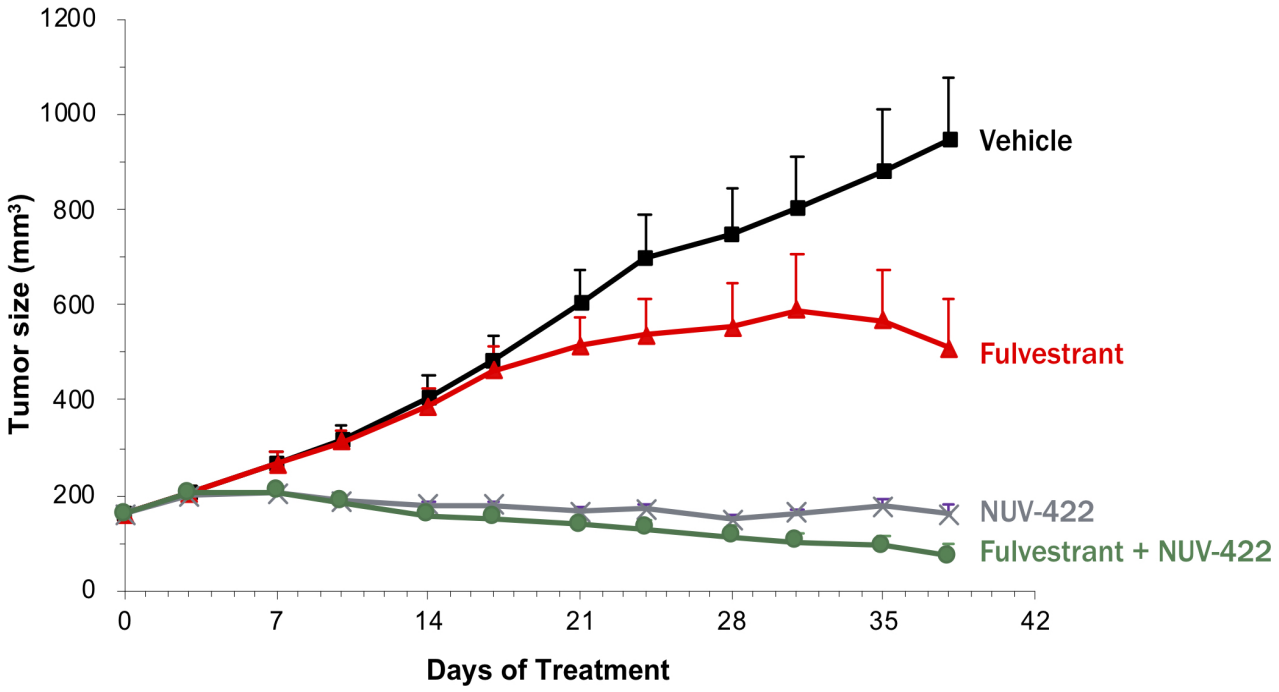
- Potent, low nanomolar IC_{50}s seen in cell lines of cancers that commonly metastasize to brain

CDK INHIBITORS DOMINATE THE ER+ BREAST TREATMENT LANDSCAPE



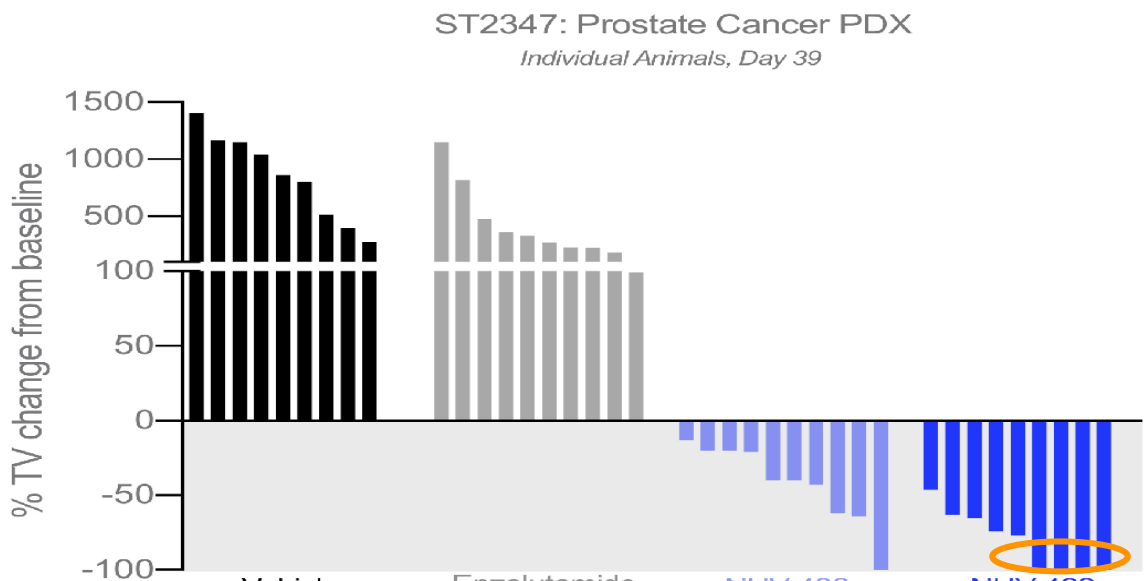
NUV-422 IS SUPERIOR TO STANDARD OF CARE FULVESTRANT IN *IN VIVO* XENOGRAFT MODEL OF ER+ METASTATIC BREAST CANCER

Tumor Volume



NUV-422 CAUSES DEEP TUMOR REDUCTIONS IN AN ENZALUTAMIDE-RESISTANT PATIENT-DERIVED PROSTATE CANCER XENOGRAFT MODEL

Tumor Volume (Day 39)



	Vehicle	Enzalutamide 30mg/kg	NUV-422 30mg/kg	NUV-422 + Enzalutamide
Complete Responses	0%	0%	10%	40%

Drug-Drug Conjugate (DDC) Platform

DDC1 [PARP-AR] mCRPC

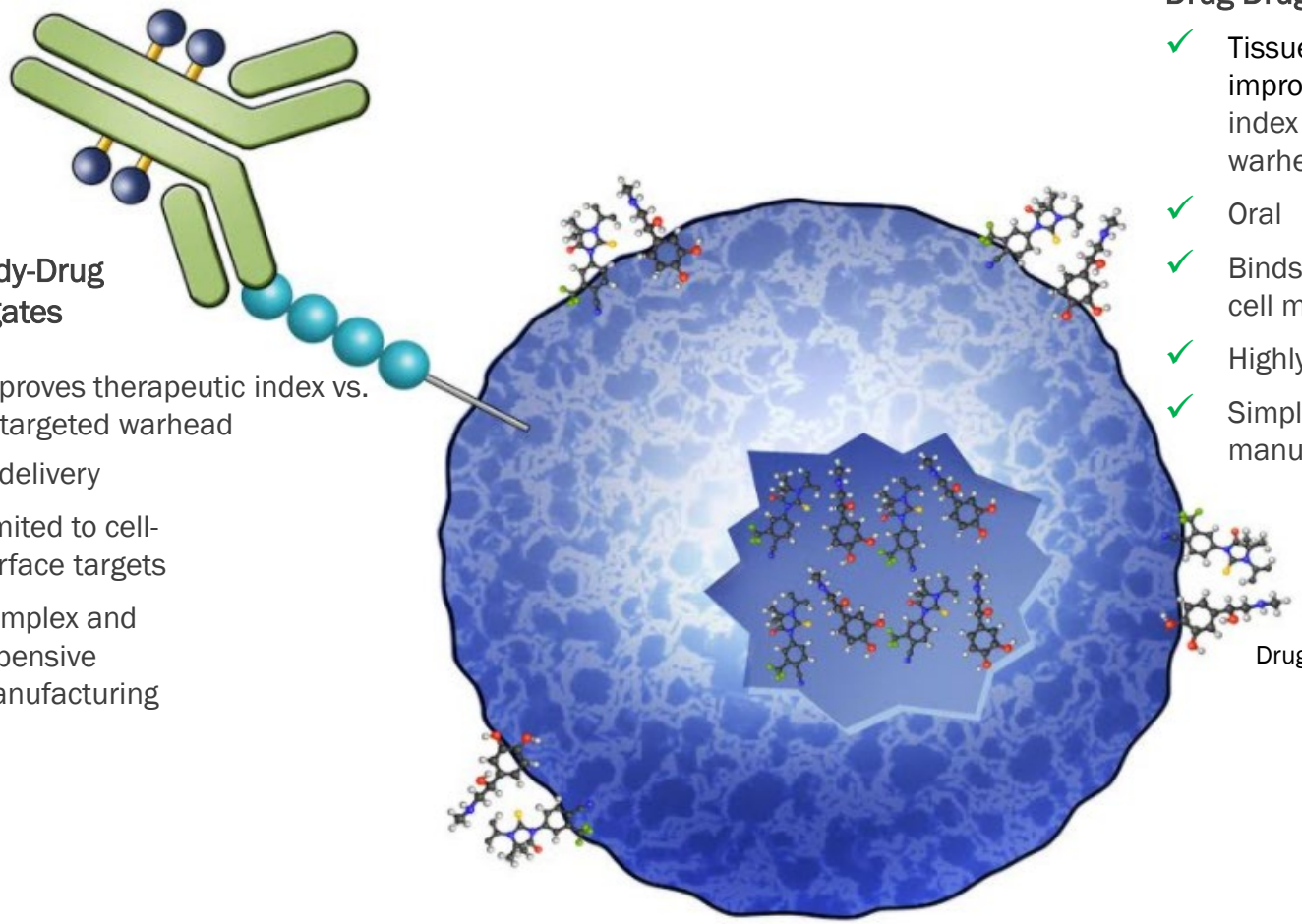
DDC2 [PARP-ER] Breast Cancer

DDC2 [PARP-ER] Ovarian Cancer

2H22 Nominate First DDC Candidate

THE DRUG-DRUG CONJUGATE (DDC) PLATFORM IS A POTENTIALLY REVOLUTIONARY ADVANCE BEYOND ADCs

Antibody Drug Conjugate



Drug-Drug Conjugate

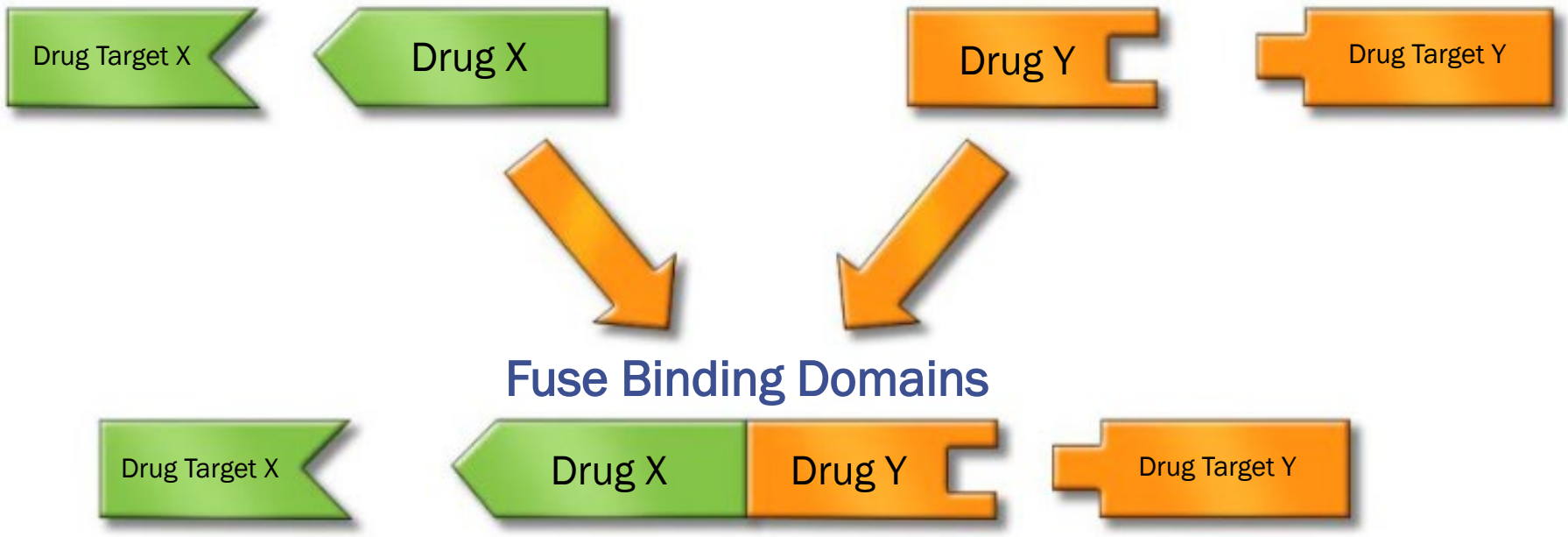
Antibody-Drug Conjugates

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

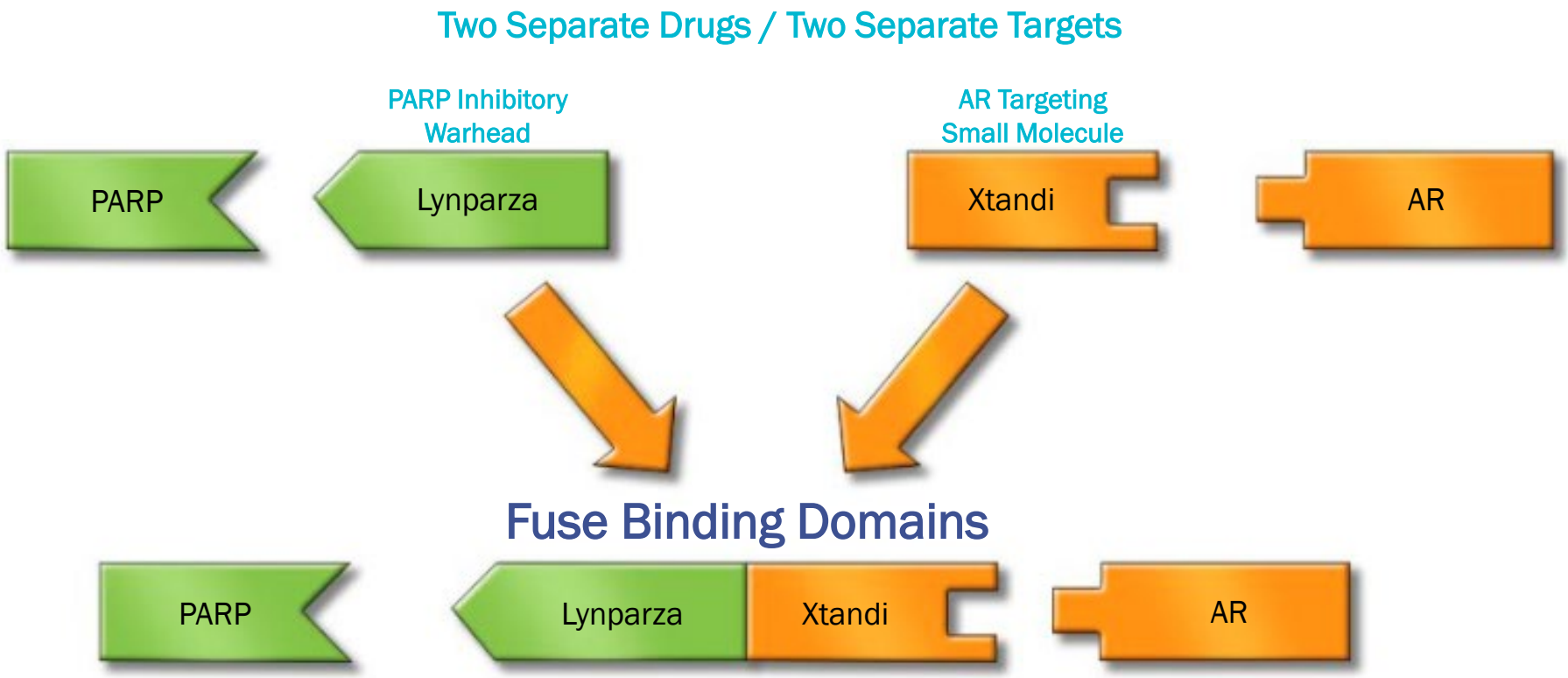
Drug-Drug Conjugates

- ✓ Tissue-selective targeting improves therapeutic index vs. untargeted warhead
- ✓ Oral
- ✓ Binds intracellular and cell membrane targets
- ✓ Highly cell permeable
- ✓ Simple and inexpensive to manufacture

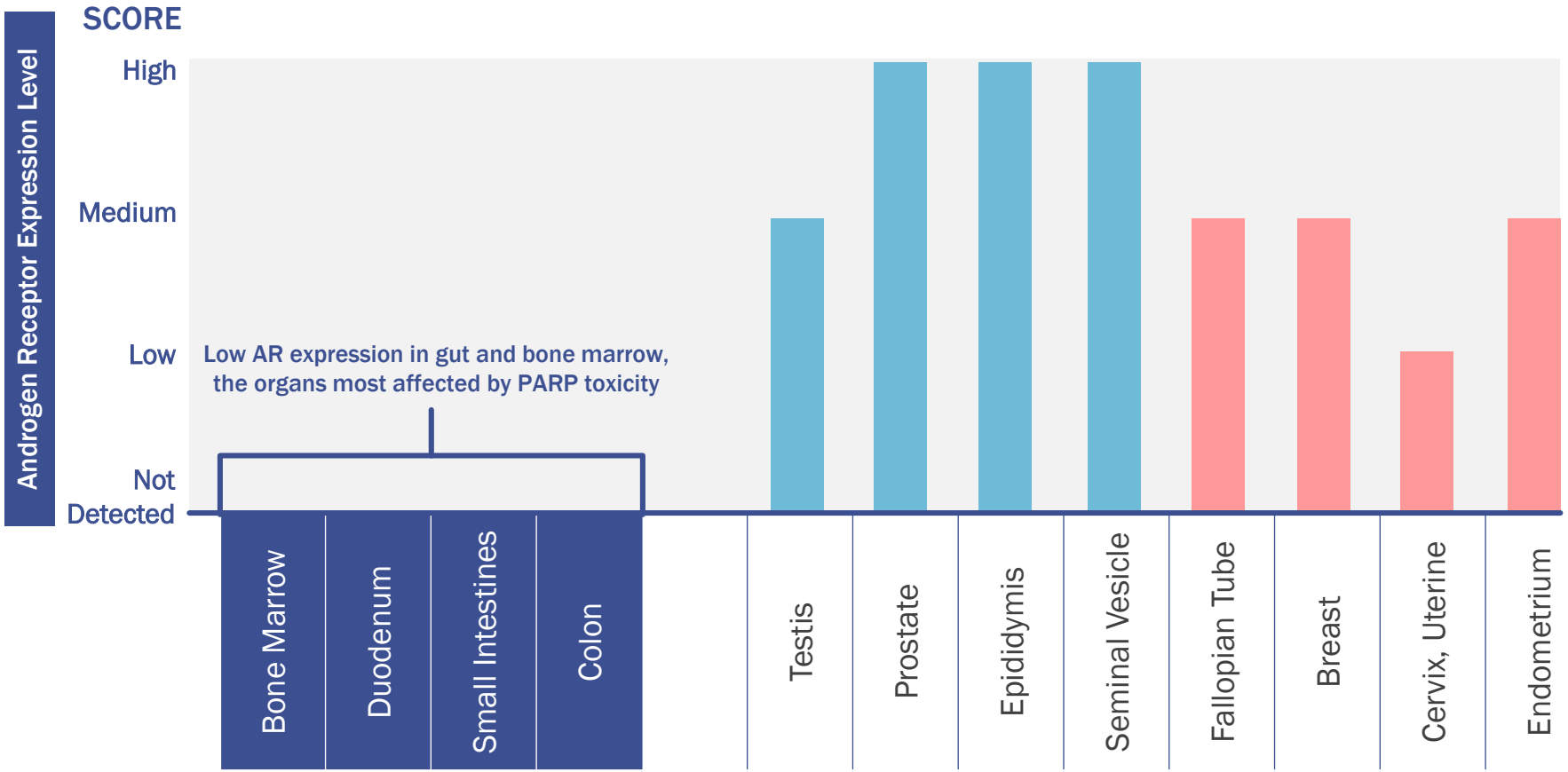
DRUG-DRUG CONJUGATES ARE DESIGNED TO BIND TWO DIFFERENT TARGETS SIMULTANEOUSLY



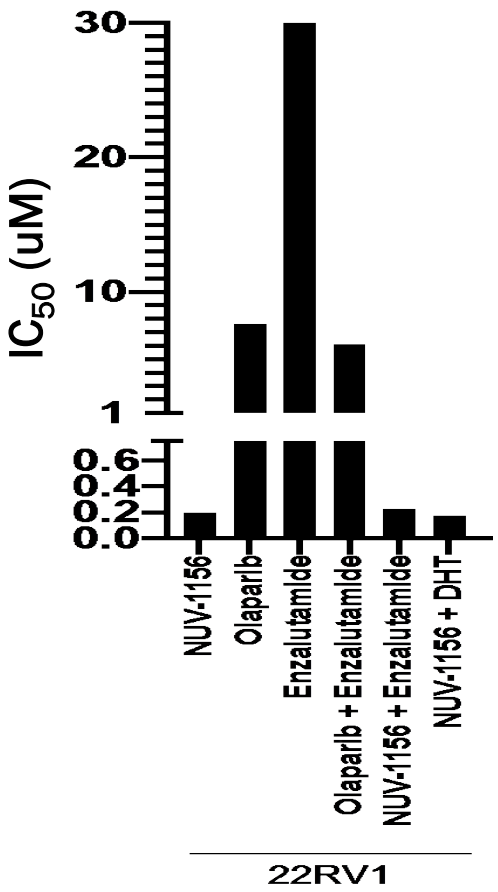
NUV-1156: A NOVEL NUVATION BIO DRUG-DRUG CONJUGATE THAT TARGETS AR AND PARP



NUV-1156 TARGETS HIGH AR-EXPRESSING TISSUE LIKE PROSTATE CANCER AND AVOIDS LOW AR-EXPRESSING TISSUE LIKE BONE MARROW AND GI TRACT

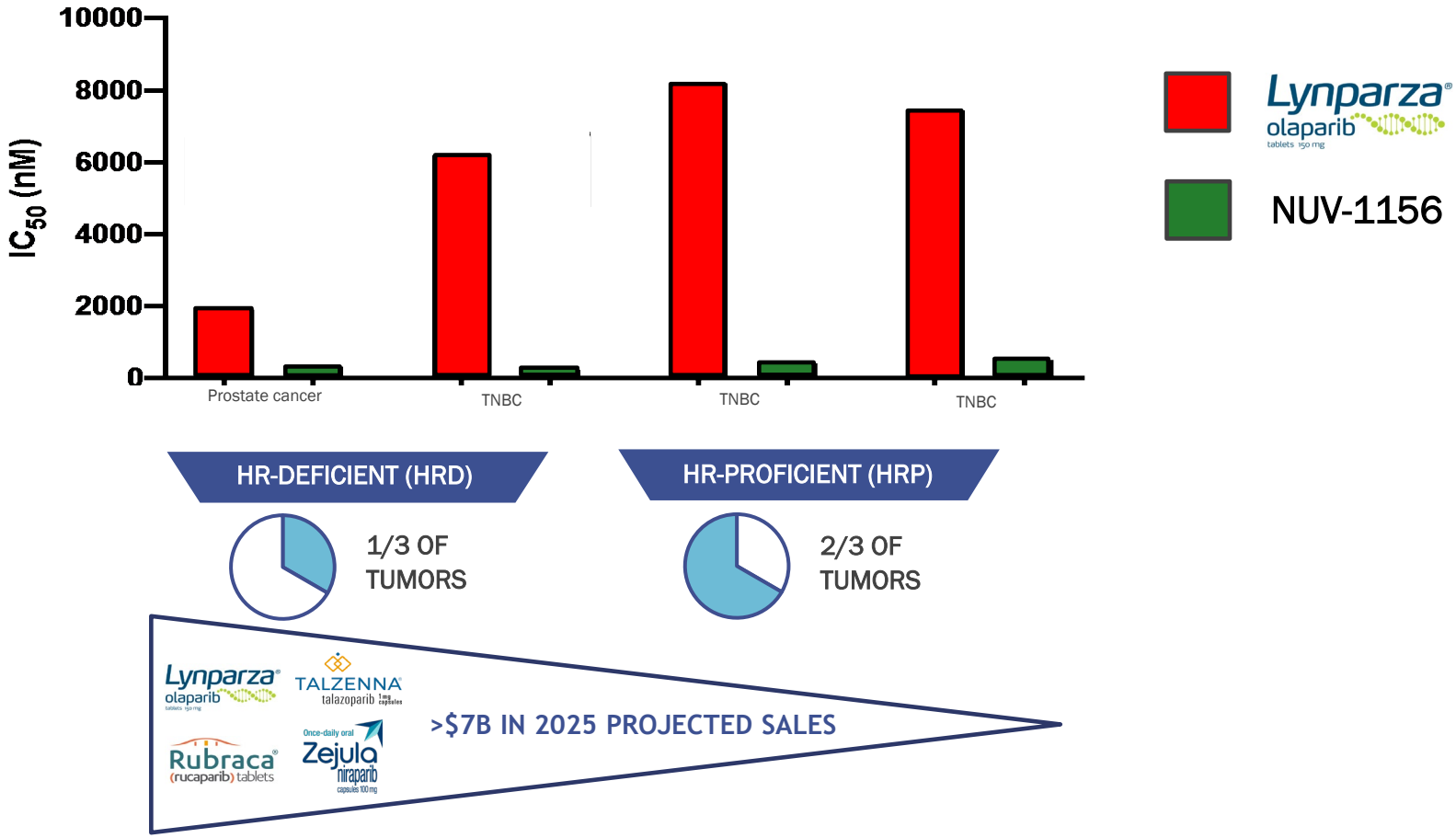


NUV-1156 DDC POTENTLY KILLS PROSTATE CANCER CELLS RESISTANT TO CURRENT STANDARDS OF CARE



	CELL PROLIFERATION IC_{50} (nM)
Xtandi (enzalutamide)	>30,000
Lynparza olaparib tablets 150 mg	7844
Xtandi (enzalutamide) + Lynparza olaparib tablets 150 mg	6152
NUV-1156 (PARP-AR DDC)	201

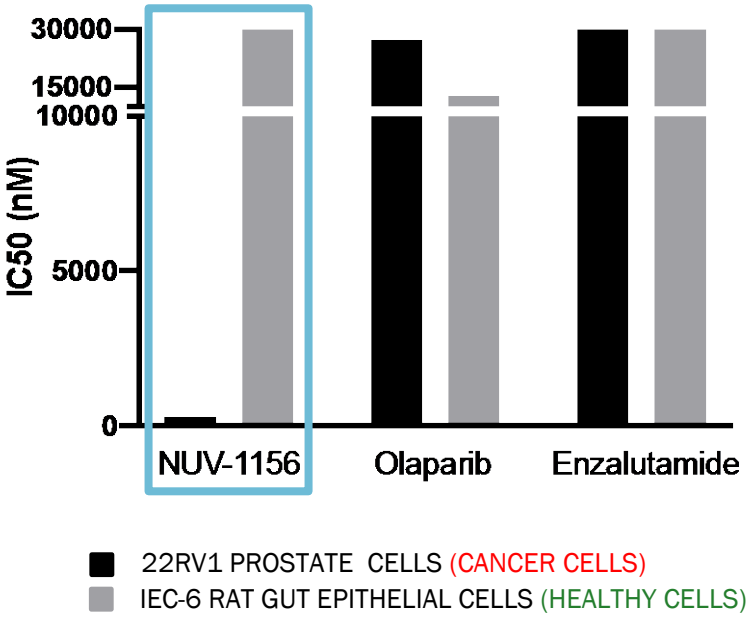
UNLIKE CURRENT PARP INHIBITORS, NUV-1156 KILLS HR-DEFICIENT AND HR-PROFICIENT CANCER CELL LINES WITH EQUALLY HIGH POTENCY



NUV-1156 KILLS ENZALUTAMIDE-RESISTANT PROSTATE CANCER (HIGH AR) CELLS BUT SPARES HEALTHY COLON (LOW AR) CELLS *IN VITRO*

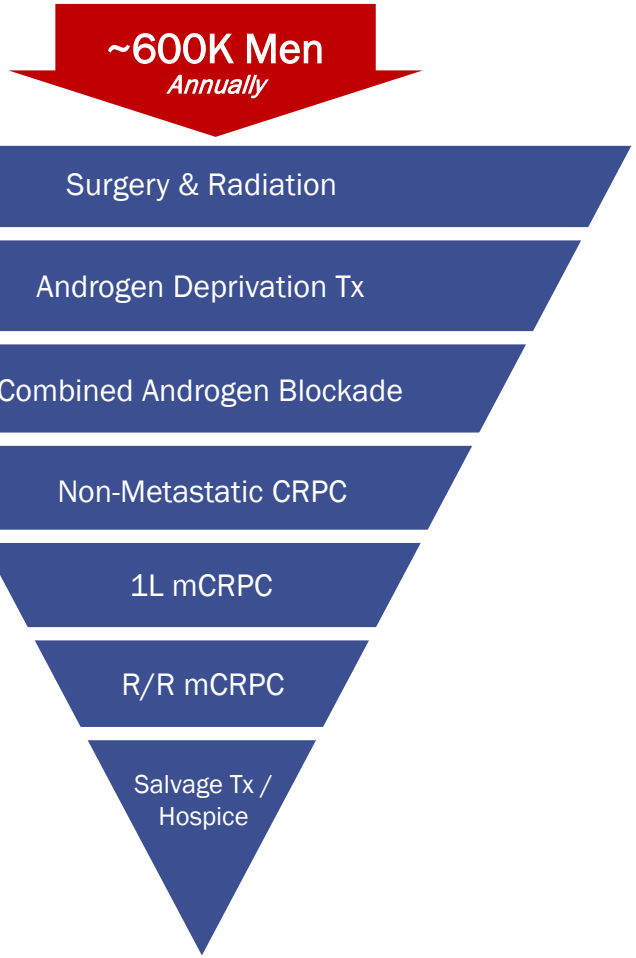
APPROVED PARP INHIBITORS HAVE HIGH RATES OF GI TOXICITY

Adverse Reactions	Lynparza tablets n=195		Placebo n=99	
	Grades 1-4 %	Grades 3-4 %	Grades 1-4 %	Grades 3-4 %
Blood and lymphatic disorders				
Anemia[b]	44	20	9	2
Gastrointestinal disorders				
Nausea	76	3	33	0
Vomiting	37	3	19	1
Diarrhea	33	2	22	0
Stomatitis[c]	20	1	16	0



Source: Lynparza Label

THE ONLY POTENTIALLY CURATIVE PROCEDURE FOR PROSTATE CANCER CURRENTLY IS SURGICAL PROSTATECTOMY/RADIATION ABLATION



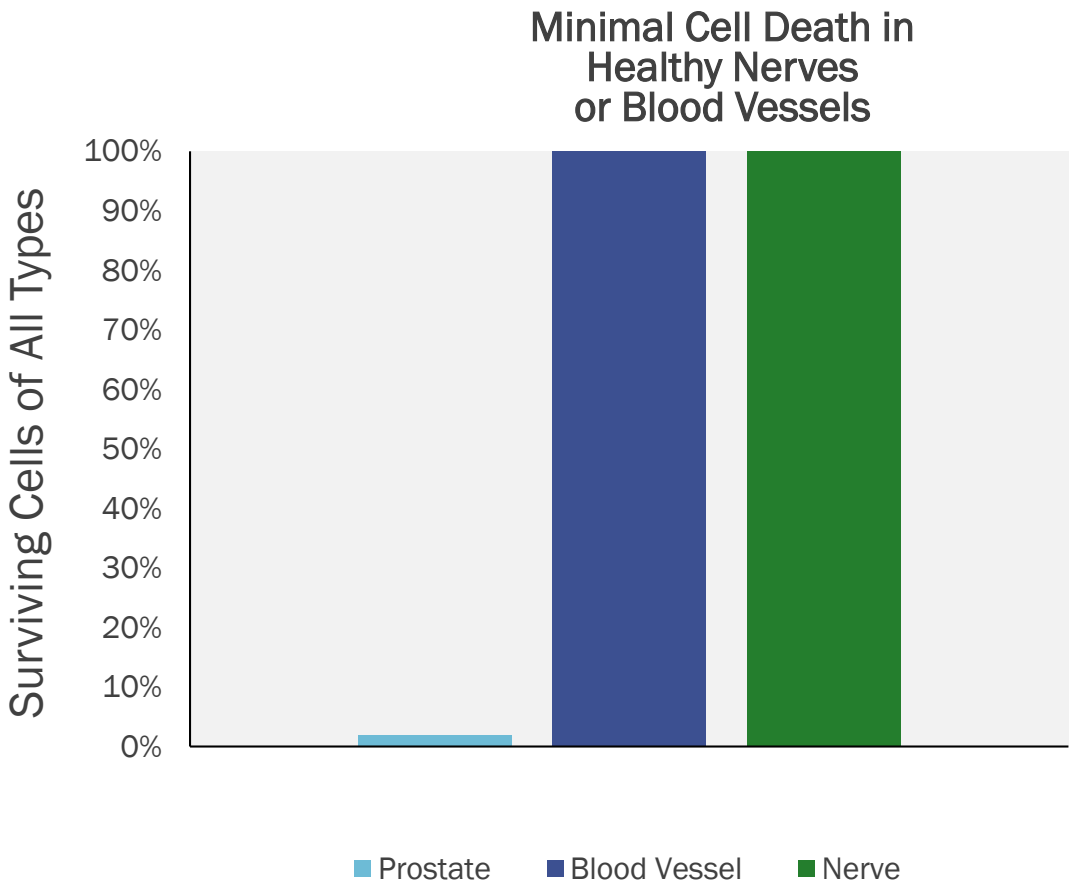
$15B Market Opportunity
No Approved Therapies

Side Effects of Standard of Care

- Erectile Dysfunction
- Urinary and Fecal Incontinence
- Invasive Surgical Risk

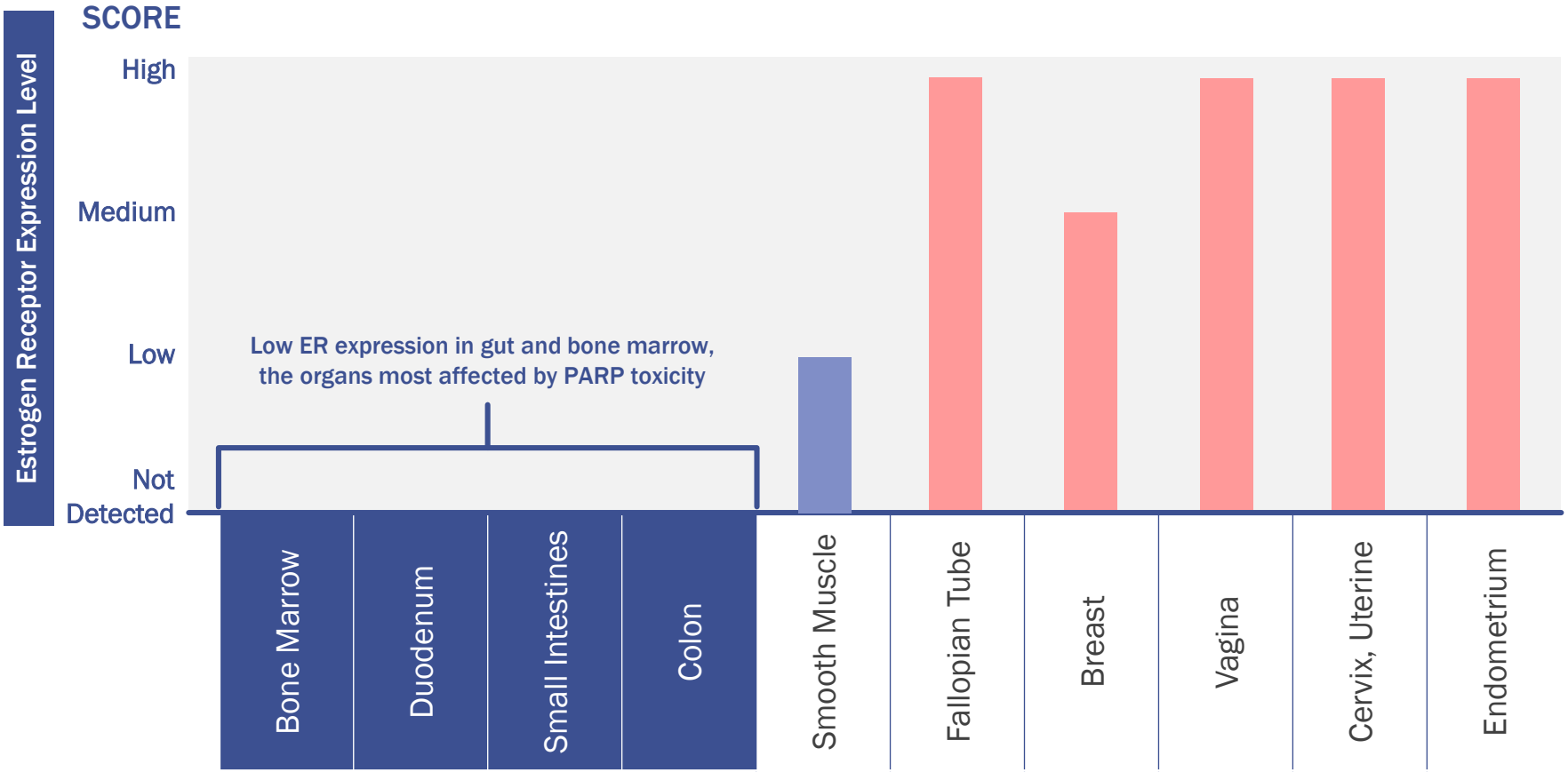
LupronDepot® (leuprolide acetate for depot suspension)
ORGOVYX™ (relugolix) 120 mg tablets
FIRMAGON® (degarelix for injection)
TAXOTERE® (docetaxel) Injection
Casodex® bicalutamide
Xtandi® (enzalutamide)
Zytiga® (abiraterone acetate) 250 mg, 500 mg tablets
JEVTANA® (cabazitaxel) injection

NUVATION BIO'S VISION: USE PROSTATE SPECIFIC DDC TO ACHIEVE A NERVE/BLOOD VESSEL-SPARING "PHARMACOLOGICAL PROSTATECTOMY"

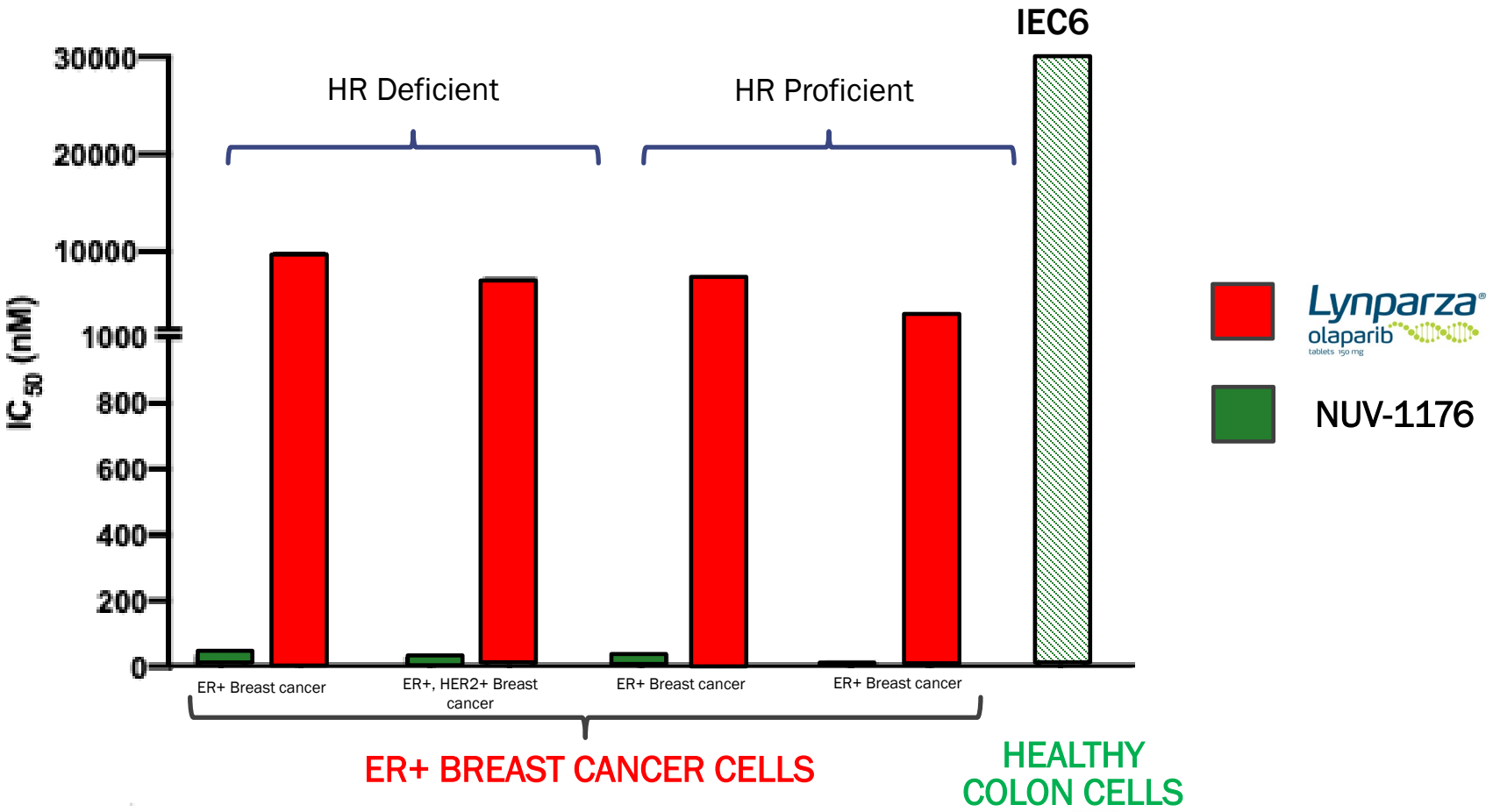


- NUV-1156 has the potential to kill prostate cancer cells with unprecedented specificity, sparing blood vessels and nerve cells
- Potentially allows men to avoid surgical prostatectomy/radiation ablation

ER PROTEIN EXPRESSION IS LIMITED TO FEMALE SEX ORGANS; LOW ER EXPRESSION IN SITES OF PARP-RELATED TOXICITY LIKE GUT AND MARROW



NUV-1176, AN ER-TARGETED DDC, POTENTLY KILLS BOTH HR-D AND HR-P ER+ BREAST CANCER CELLS WITHOUT KILLING HEALTHY COLON CELLS

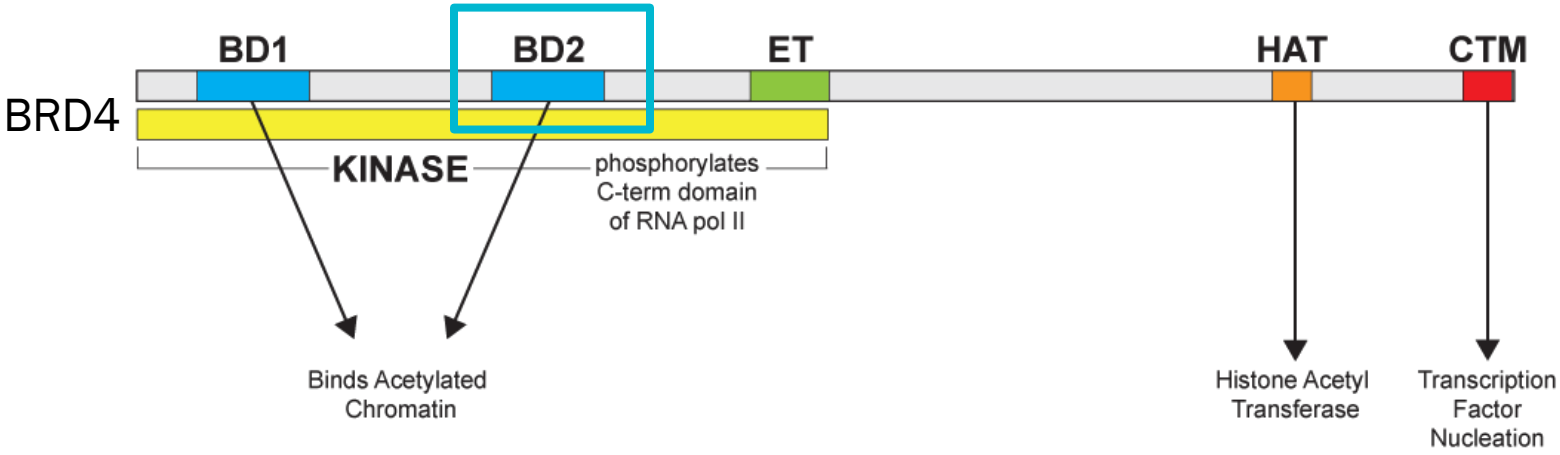


NUV-868 | BET

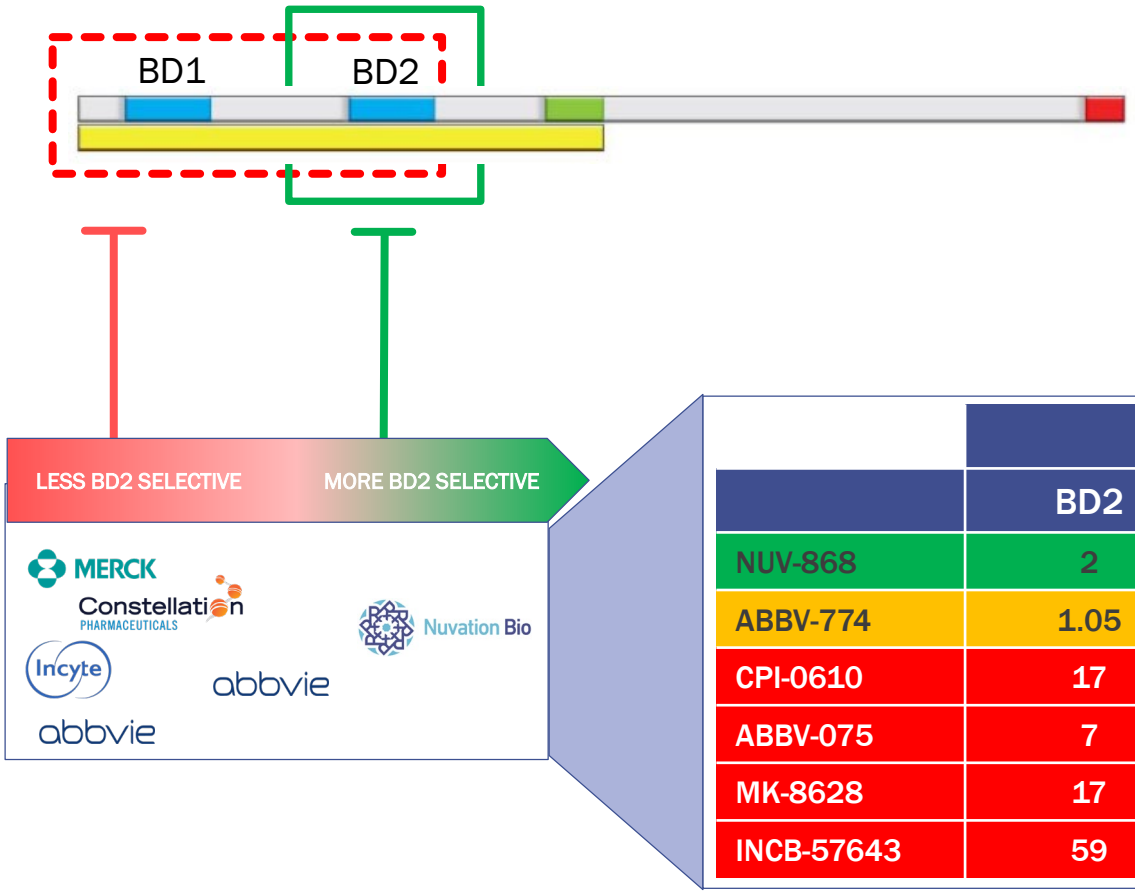
AML/solid tumors 1H22 Initiate Ph1 trial

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 (Faivre et al 2020)



NUV-868 IS A HIGHLY SELECTIVE BD2 VS BD1 INHIBITOR

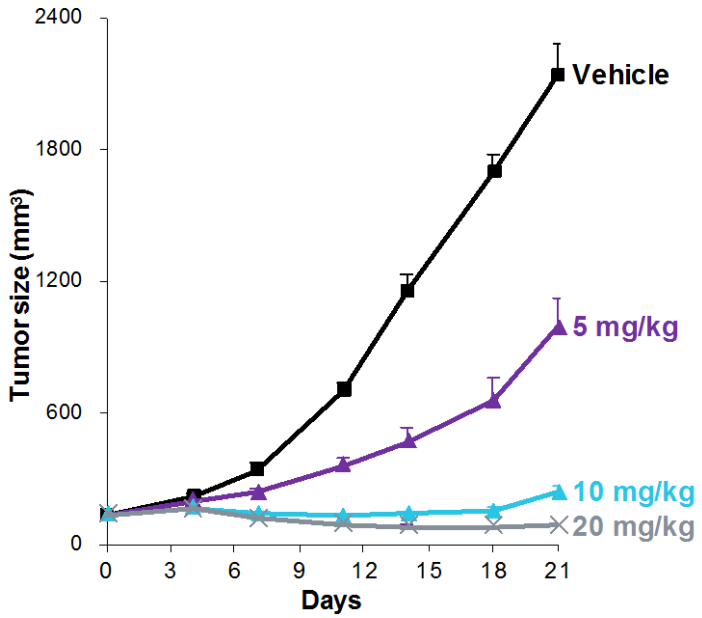


- BET inhibitors have historically targeted BD1 and BD2 non-selectively, causing GI toxicity and thrombocytopenia
- Selective BD2 vs BD1 inhibition can improve tolerability but has been difficult to achieve
- Selective BD2 inhibitors have the potential to block many oncogenes, including c-myc

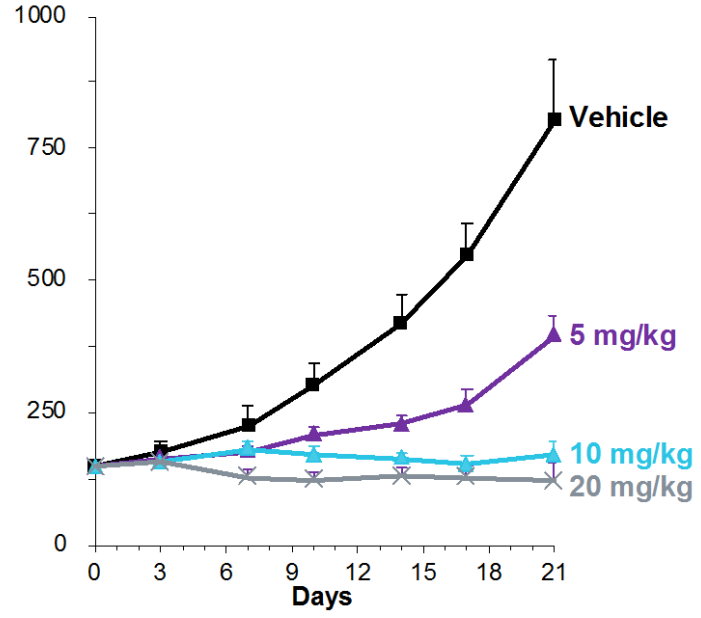
	BRD4 Affinity		
	BD2	BD1	Selectivity
NUV-868	2	2920	1460x
ABBV-774	1.05	340	234x
CPI-0610	17	85	5x
ABBV-075	7	27	3.9x
MK-8628	17	26	1.5x
INCB-57643	59	81	1.4x

NUV-868 IS HIGHLY POTENT IN KILLING ACUTE MYELOID LEUKEMIA (AML) CELLS IN *IN VIVO* XENOGRAFT MODELS

Kasumi-1



MV-4-11



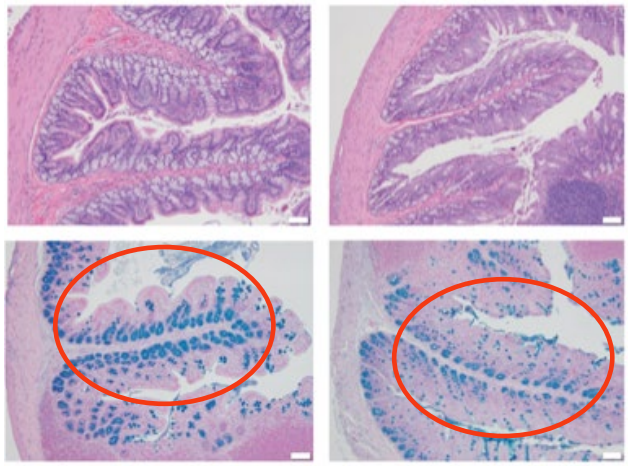
- NUV-868 demonstrates striking anti-tumor activity in two AML xenograft models

HIGH SELECTIVITY FOR BD2 OVER BD1 SIGNIFICANTLY REDUCES THE GUT TOXICITY OBSERVED WITH OTHER BET INHIBITORS

ABBV-075 (Dual BD1 / BD2)

Vehicle

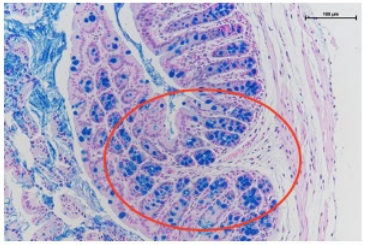
ABBV-075



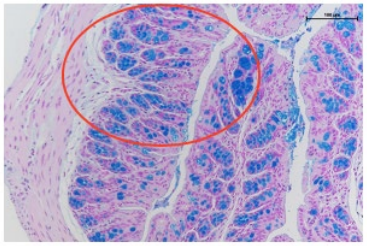
× A non-selective inhibitor (ABBV-075) leads to marked reduction in rat small intestine goblet cells(1)

NUV-868 (BD2 Selective) Avoids GI Toxicity

Vehicle



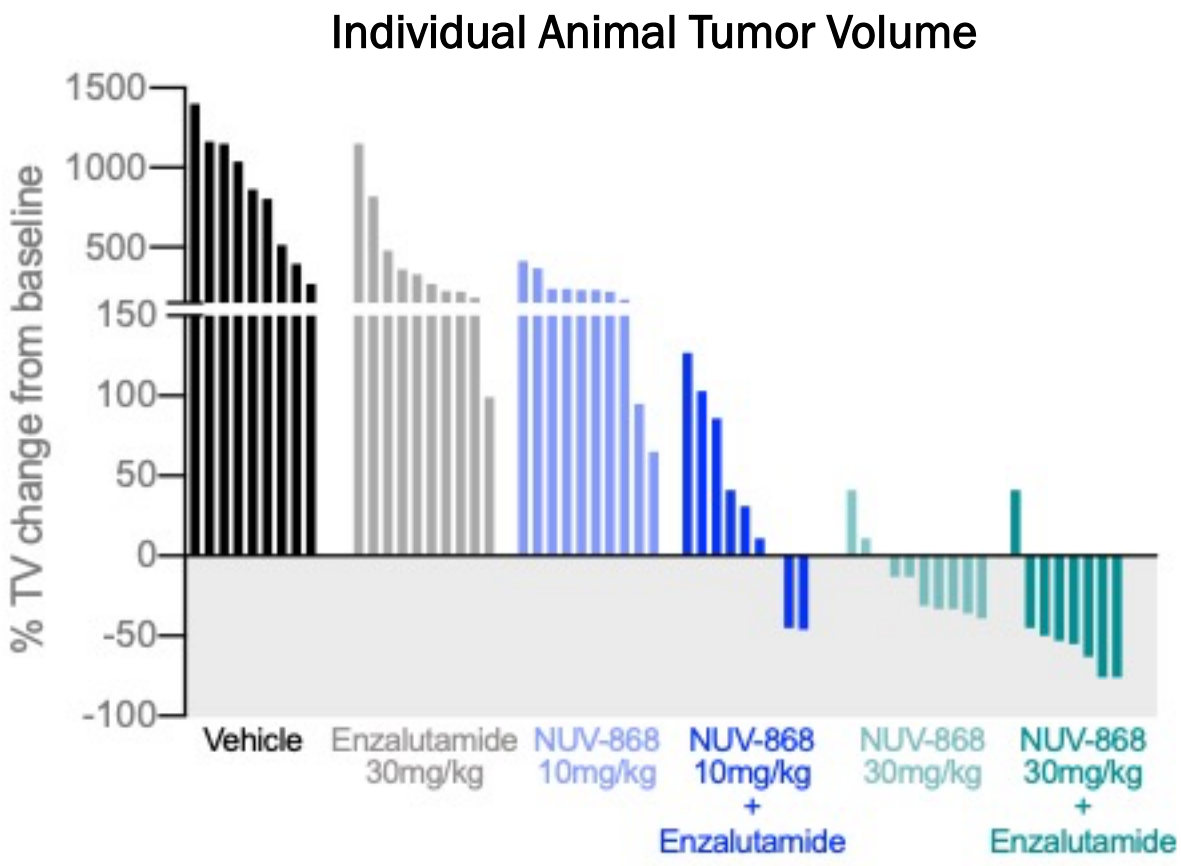
NUV-868
30 mg/kg BID



✓ 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

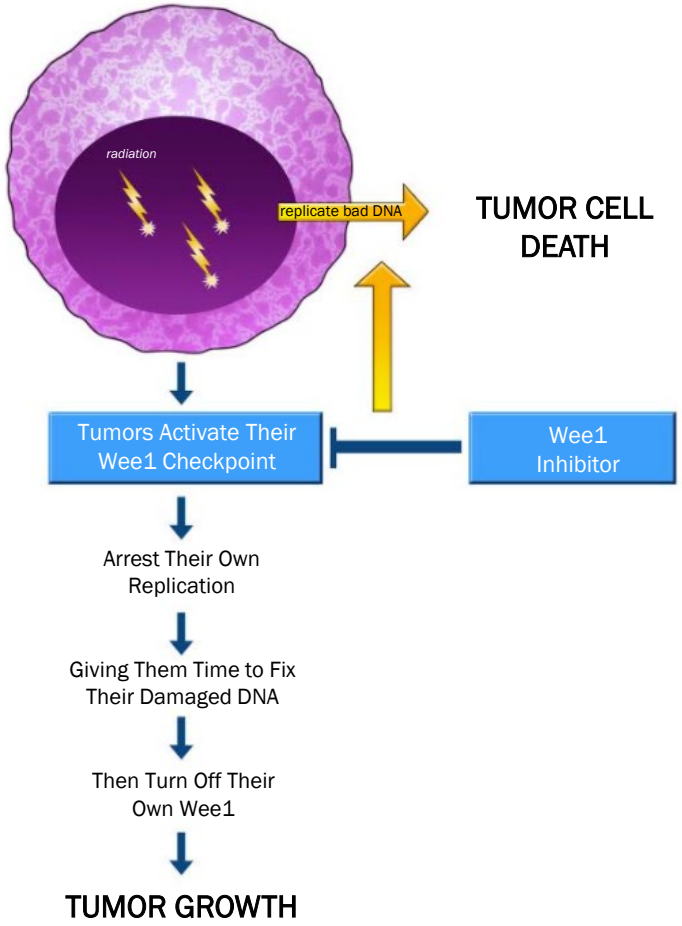
NUV-868 CAUSES DEEP TUMOR REDUCTIONS IN AN ENZALUTAMIDE-RESISTANT PATIENT-DERIVED PROSTATE CANCER XENOGRAFT MODEL



NUV-569 | WEE1

Pancreatic Cancer/other solid tumors 2H22 Initiate Ph1 trial

WEE1 INHIBITORS INCREASE THE EFFICACY OF DNA-DAMAGING THERAPIES BY FORCING CANCERS TO REPLICATE BEFORE THEY CAN REPAIR THEIR DNA



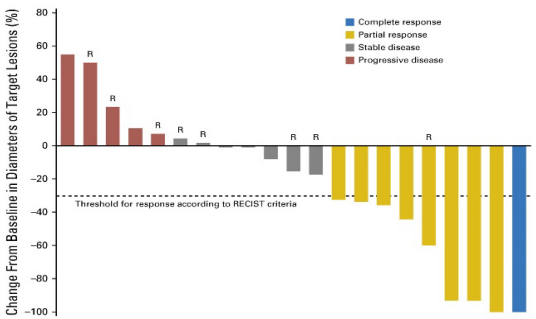
- Wee1 inhibitors force tumors to replicate damaged DNA before it can be repaired
- Replicating damaged DNA is lethal for cancer
- Wee1 inhibitors may potentiate any therapy that causes DNA damage (chemotherapy or radiation)

THE PROMISE OF EXISTING WEE1 INHIBITORS IS LIMITED BY TOLERABILITY

Efficacy

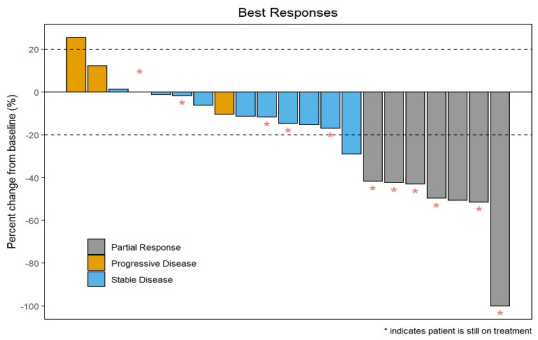
AZD1775, a third-party Wee1 inhibitor, has shown partial responses in uterine serous carcinoma, ovarian and pancreatic cancer

Ovarian Cancer(1)



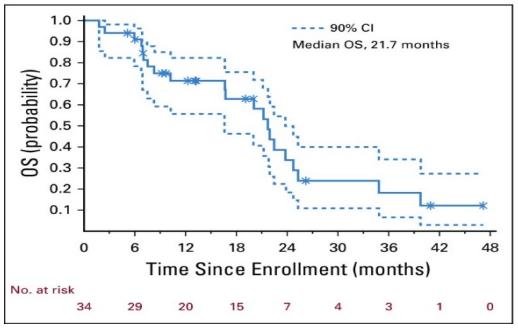
- Combo with carboplatin
- Phase 2 (n): 24 (21 evaluable)
- ORR: 43%

Uterine Serous Carcinoma(2)



- Monotherapy
- Phase 2 (n): 27 (21 evaluable)
- ORR: 30%

Pancreatic Cancer(3)



- Combo with gemcitabine
- Phase 2 (n): 34
- Median OS of 21.7 months(4)

SAFETY ISSUES WITH EXISTING Wee1 INHIBITORS

Dosing and Combination Challenges with AZD1775

- Potent inhibitor of PLK1, which contributes to bone marrow toxicity and GI toxicity
- Inhibits liver enzyme CYP3A4, which is responsible for elimination of drug and drug metabolites from the body
- Tolerability issues prevent continuous dosing

(1) https://ascopubs.org/doi/full/10.1200/JCO.2016.67.5942
(2) https://sgo.confex.com/sgo/2020/meetingapp.cgi/Paper/15031
(3) https://pubmed.ncbi.nlm.nih.gov/31398082/#&gid=article-figures&pid=fig-2-uid-1
(4) Versus 11.9 to 13.6 moths observed in a prior clinical trial

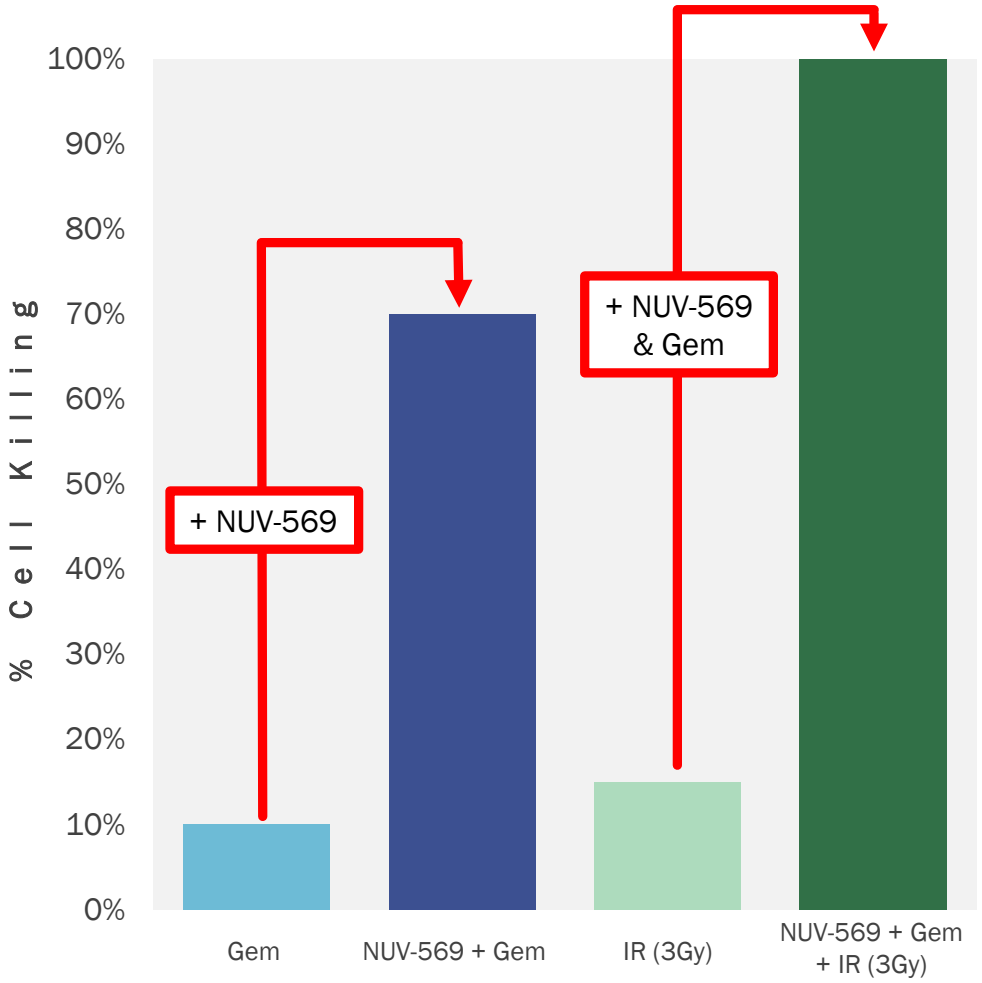
NUV-569'S HIGHLY POTENT AND SELECTIVE PROFILE = LESS TOXICITY

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 SIGNIFICANTLY INCREASES *IN VITRO* KILLING OF PANCREATIC CANCER CELLS BY CHEMO AND/OR RADIATION



- NUV-569 shows strong synergy with standard-of-care gemcitabine and radiation
- In-vivo xenograft tumor models ongoing
- In addition to pancreatic cancer, we are also evaluating NUV-569 in breast, ovarian and endometrial cancer

NUV-1182 | A2A receptor

Solid Tumors in Combo with IO

4Q22 Initiate Phase 1 trial

NUV-1182, AN ADENOSINE A2A RECEPTOR INHIBITOR, BOOSTS IMMUNE FUNCTION

- NUV-1182 is focused on targeting the A2A adenosine receptor, which 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-121	NUV-191	NUV-1182
A2A binding	20	7.1	2.4	2.4	1.4	2.9
A1 binding	366	118	16	21	9.4	234
A2A Selectivity (A2A/A1)	18	16	7	9	7	81
A2A cAMP (400 nM NECA)	1614	1007	70	607	311	441
PPB Hu/Mu (A2a IC50/Fu)	155/238	1014/87	32/42	34/14	56/175	82/24
Hu/Mu PPB (% free)	7.2/4.7	0.7/8.1	7.5/5.6	6.9/16.6	2.5/0.8	3.5/11.8

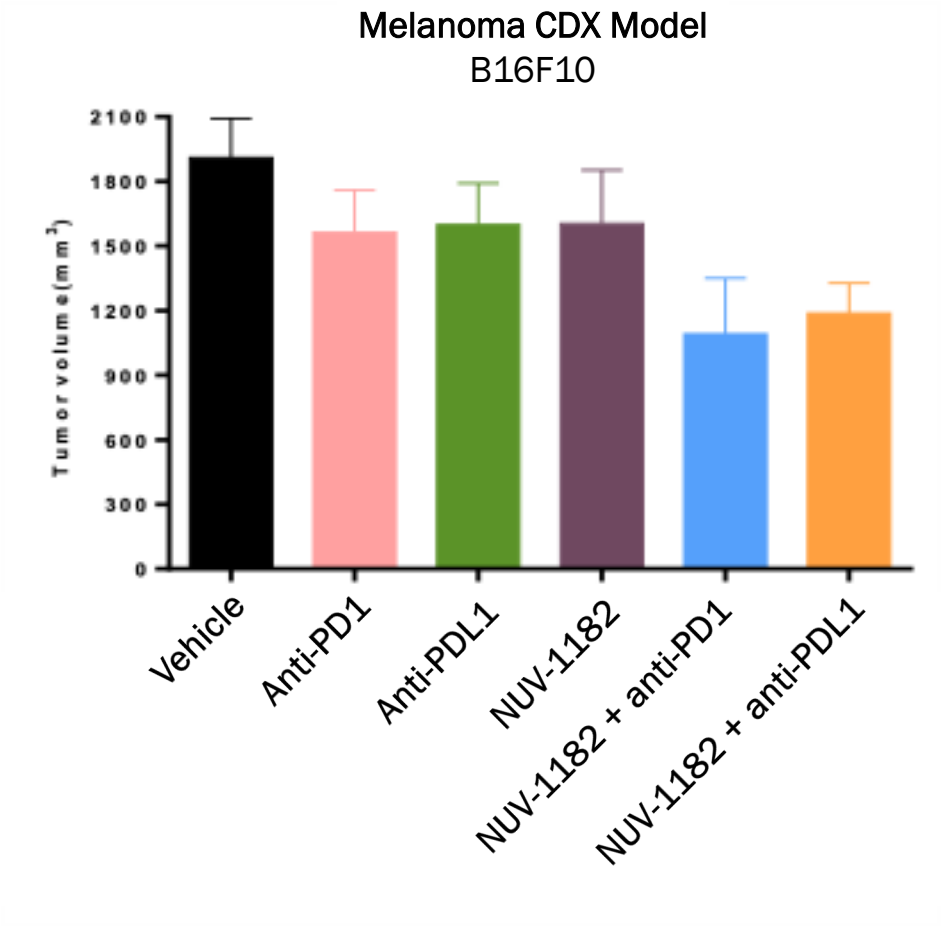
NUV-1182 HAS AN ATTRACTIVE PK PROFILE, WITH GOOD ORAL BIOAVAILABILITY AND DOES NOT EFFECTIVELY CROSS THE BLOOD-BRAIN BARRIER

Mouse Single Dose Pharmacokinetics
NUV-1182

PO Dose	Cmax (nM)	Tmax (h)	AUC_{INF} (nM*h)	t1/2 (h)
50 mg/kg	17667	0.5	34211	1.41
200 mg/kg	50029	0.5	216670	1.74

- Brain to plasma ratios are 0.02 to 0.2, demonstrating that NUV-1182 does not effectively cross the blood-brain barrier
- NUV-1182 currently being tested in several syngeneic tumor models, with tumor volume and tumor infiltrating immune cell profiling and mechanism of action studies

NUV-1182 INCREASES THE ANTI-TUMOR ACTIVITY OF IMMUNE CHECKPOINT INHIBITORS IN AN *IN VIVO* MELANOMA XENOGRAFT MODEL



Financial Overview

FINANCING HISTORY

- Nuvation Bio was founded on March 20, 2018 by Dr. David Hung
- $275M raised in June 2019 in a Series A from high profile, experienced investors including Fidelity, Baupost, Omega, EcoR1, Altitude, Citadel, Boxer, etc.
- Merger with Panacea (EcoR1 sponsored SPAC) and $500M in concurrent financing announced in October 2020. Some key investors from the PIPE include Fidelity, Avidity, EcoR1, Deerfield, Farallon, Redmile, and Baupost
- Trading on the NYSE under the stock symbol NUVB
- 218M fully diluted shares outstanding and approximately $830M of cash

Upcoming Milestones & Summary

UPCOMING CATALYSTS ACROSS MULTIPLE PROGRAMS

	4Q20	1H21	2H21	1H22	2H22	1H23
CDK2/4/6	Dosed first patient in Ph1 High-grade Glioma trial			Initiate Ph1 in Brain metastases Top-line Ph1 data from High-grade Glioma trial	Initiate Ph1 in ER+ mBC	Initiate Ph1 in mCRPC Present first efficacy data from High-grade Glioma trial
BET			Submit IND	Initiate Ph1 in Acute Myeloid Leukemia and/or solid tumors		
WEE1				Submit IND	Initiate Ph1 in Pancreatic Cancer and/or other solid tumors	
A2A				Submit IND	Initiate Ph1 IO combination trial (4Q)	
DDC1 PARP-AR DDC2 PARP-ER					Nominate first DDC candidate	

TACKLING THE GREATEST UNMET NEEDS IN ONCOLOGY

- Broad wholly-owned pipeline with strong IP protection
  - Up to 5 INDs in 6 years
- Multiple drug lead candidates addressing large markets with blockbuster drug sales potential
- Leveraging and improving upon validated drug mechanisms
- Focused on best-in-class drug candidate profiles vs. competitors
- Experienced biotech leadership team with multiple oncology drug approvals

Leading oncology company with approximately $830 million in cash resources enabling a world-class drug development team to rapidly pursue clinical development of multiple portfolio therapeutic candidates

Nuvation Bio

FEBRUARY 2021