



*Developing Novel DNA Decoy
Therapeutics For Precision
DDR Oncology Therapeutics*

Corporate Presentation

2Q 2023

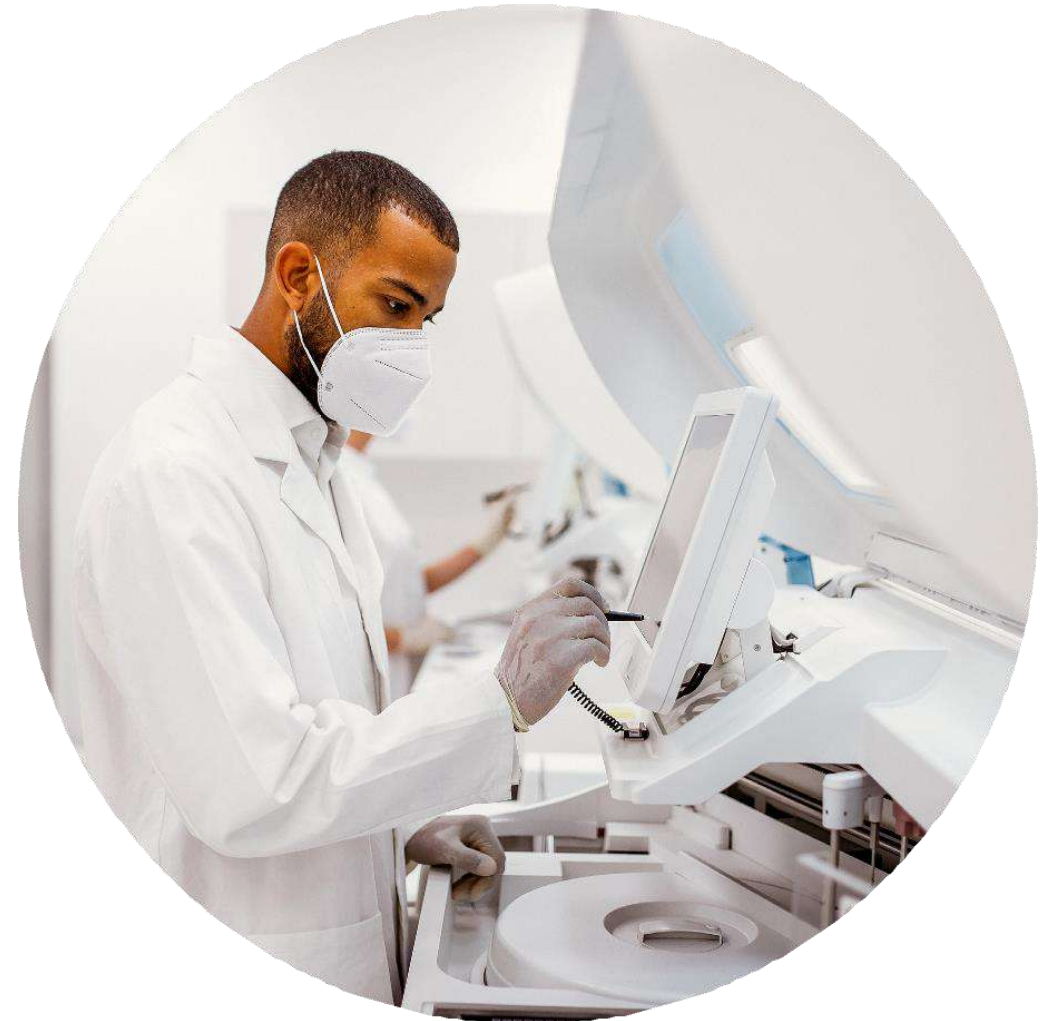
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Key Management Members



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Head of Clinical & Medical



Wael Jdey, PhD
Head of Preclinical



Huiping Jiang, PhD
Head of Regulatory & QA



Donna Cabral-Lilly, Ph.D.
VP, CMC



Joyce Carey
Head of FP&A, Exécutive Director Finance



Robin Sutherland
Head of HR



A First-In-Class DDR, DNA Decoy Therapeutics Company



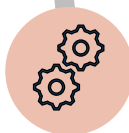
DNA Damage Repair Is Critical For Genomic Stability And Tumor Survival

DNA damage response (DDR) is a rapidly developing field in oncology, with approved PARP inhibitors and other clinically validated DDR targets including ATR and WEE1 inhibitors



First-In-Class DNA Decoy Therapeutics Mimicking DNA Damage And Trapping DNA Repair Proteins

PlatON™ platform **mimics DNA damage and traps intracellular DNA binding targets with a unique MoA** - proteins in charge of detecting and signaling. ValerioTX has a clinical-stage pipeline of first-in-class DNA decoy drugs protected by several patent families in key territories until at least 2040



DNA Decoys Are Superior To Current DDR Inhibitors & Has Applicability Beyond DDR

ValerioTX's **DNA decoys do not rely on enzymatic active sites, thereby relieving evolutionary pressure** and the rapid development of resistance mutations. The DNA decoy therapeutics toolbox is being further expanded to include additional targeting moieties such as bispecific antibodies and aptamers to target transcriptional activation and epigenetic factors



VIO-01 Is A Pan-DDR Decoy Targeting Multiple Proteins & Repair Pathways

VIO-01 is a validated monotherapy with efficacy in late-stage IND-enabling studies. Favorable safety and a high anti-tumor activity



Strong Capabilities In End-to-end Product Development

Management team with proven track record and **20+ years of experience in Pharma/Biotech, including deep oncology drug expertise**, experience working with FDA and regulatory agencies, network of KOLs and partners including Agenus, Gustave Roussy, Institut Curie & USO



Overview Of Valerio Therapeutics

AsiDNA™ - Targeted Sequestering Of DNA-PK

VIO-01 - Multi-Modal DDR Decoy

DecoyTAC - 3rd Generation

Summary

Innovative Pipeline Of DNA Decoy Therapeutics

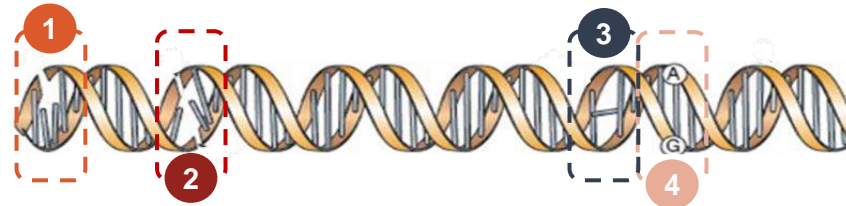
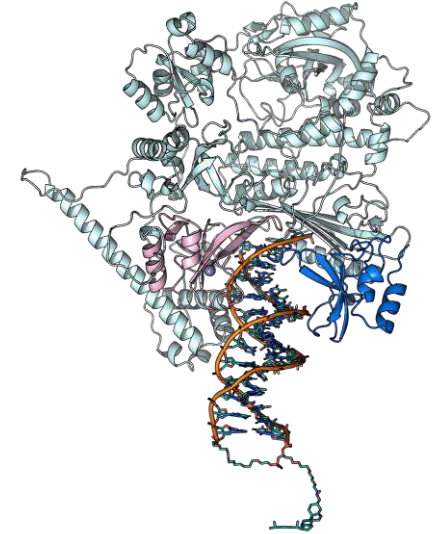


PlatON™	Program (Route Of Admin)	Target	Target Indication	Discovery	IND-Enabling	Phase 1/2	Phase 3	Partner(s)	Next Milestone
1 st Generation	AsiDNA™ 1.0 (IV)	DNA-PK	Recurrent ovarian, breast, prostate cancer (combination with PARPi)	US Clinical Trial					FPI U.S. trial Mar-23
			Maintenance for ovarian cancer with rising CA-125 (combination with PARPi)	REVOCAN				GUSTAVE ROUSSY CANCER CAMPUS GRAND PARIS	Readout in 1Q24
			Pediatric and adolescent glioma (combination with radiotherapy)	GLIOMA				institut Curie	Readout in 1Q24
2 nd Generation	VIO-01 (IV)	PARP1, MRN, KU70/80	mHRR or HRD+ solid tumors (monotherapy)						IND submission 2H23
3 rd Generation	DecoyTAC	DDR, Epigenetics, Transcription Factors	Undisclosed						IND enabling studies 2H24

Therapeutic Targeting Of The DNA Damage Response (DDR) To Treat Cancer

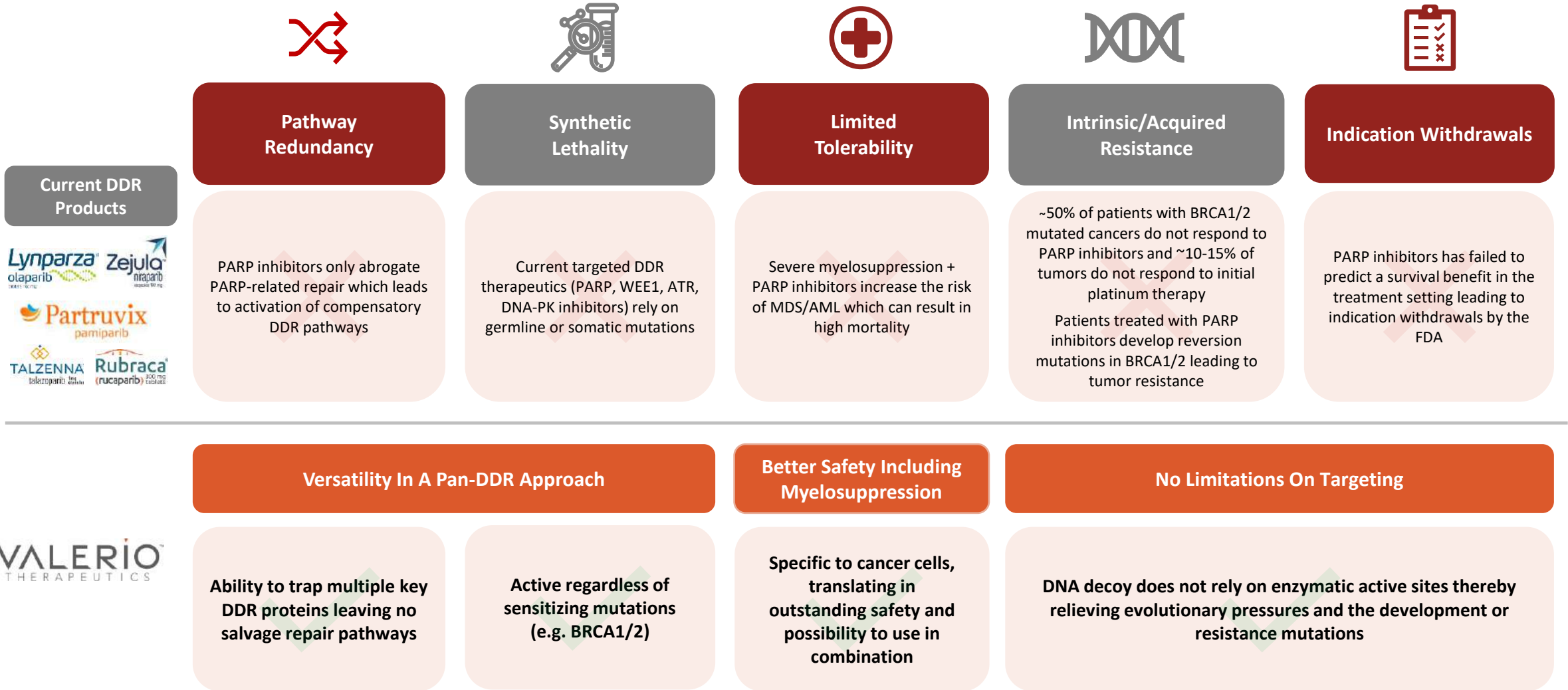


- **The DNA repair process is a molecular mechanism activated to restore genomic integrity**
- Mutations in genes found in cancer cells can lead to the loss in function of one or more DNA repair pathways, causing cells become hyper-dependent on the remaining pathways
- **Inhibiting the remaining repair pathways (synthetic lethality)** causes the further damage to the DNA, turning single-strand breaks into double-strand breaks and **triggering cell death**
- **There are five PARP inhibitors (base excision repair mechanism) already on the market generating >\$3.5B (2022):** olaparib (AZ), niraparib (GSK), rucaparib (Clovis Oncology), pamiparib (BeiGene) and talazoparib (Pfizer)

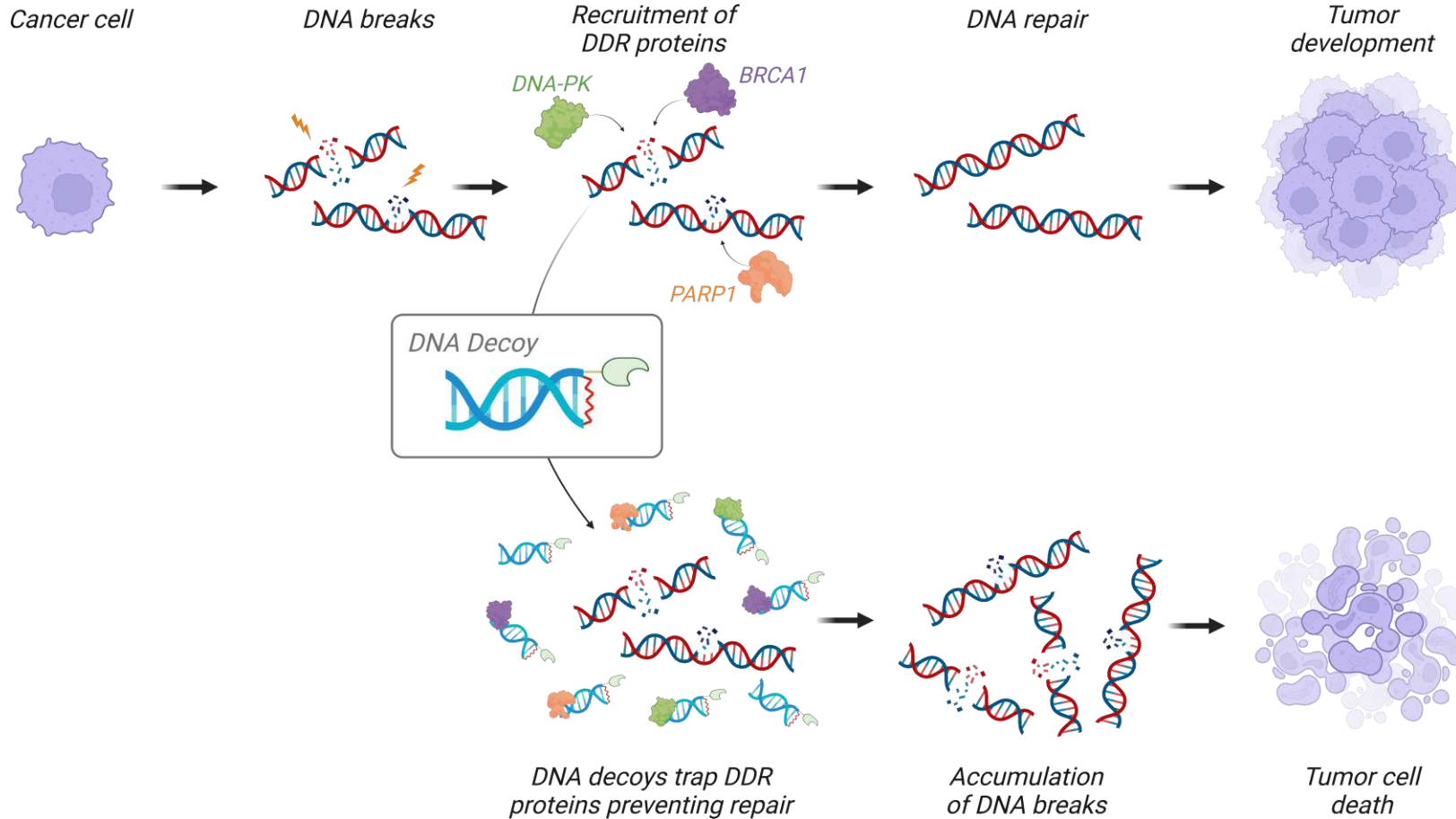


DNA Damage Type	1 Single-Strand Break (SSB)	2 Double-Strand Break (DSB)		3 Bulky Adducts	4 Base Mismatches, Insertions, Deletions
Repair Pathway	Base Excision Repair (BER)	Homologous Recombination (HR)	Non-Homologous End Joining (NHEJ)	Nucleotide Excision Repair (NER)	Mismatch Repair (MMR)
Effector Proteins	PARP1 XRCC1	BRCA1 BRCA2 PALB2 ATM CHEK1 CHEK2 RAD52	KU70/KU80 DNA-PK	ERCC4 ERCC1	MSH2 MSH3 MSH6 MLH1 MLH2 MLH3

Addressing The Severe Limitations With Current DDR Therapies



DNA Decoy: Sequestering Key DNA Repair Proteins With A Differentiated Mechanism Of Action



Trapping DNA Repair Proteins

DNA decoys mimic DNA breaks in tumor cells, saturate them with false alarm signals (**decoy**), then traps key DDR proteins

Hindering DNA Repair Functions

This prolonged signaling, trapping and overactivation **depletes** the DDR and hinders DNA repair mechanisms

Cell Death

Actual lesions are not repaired and accumulate, cancer cells die when they divide with damaged DNA

PlatON™, Proprietary DNA Decoy Platform Targeting Intracellular DNA-Binding Targets



The platON™ DNA Decoy platform uses three components:

Specificity And Functionalisation



1 DNA Decoy

- *Double-stranded DNA fragment (oligonucleotide) of variable sequence and length*
- *Ability to target DDR, epigenetic proteins and transcription factors*

2 Linker

- *Tethered with a loop to prevent dissociation*

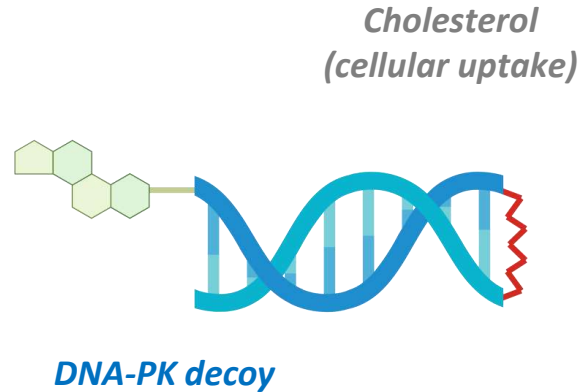
3 Vectorisation

- *Facilitating tumoral and nuclear uptake*
- *Ongoing work to incorporate small molecules, nanobodies and aptamers*

Imagining Beyond - Driven By The PlatON™ Platform

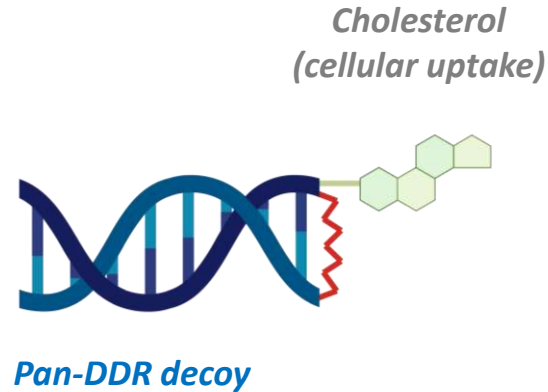


1st Generation



AsiDNA™

2nd Generation

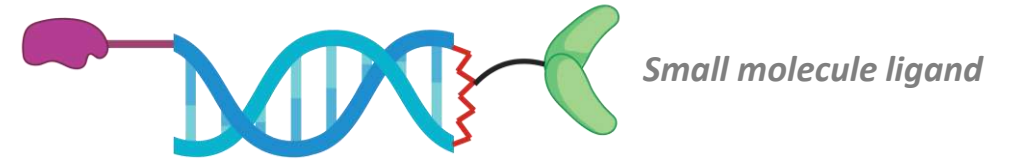


VIO-01

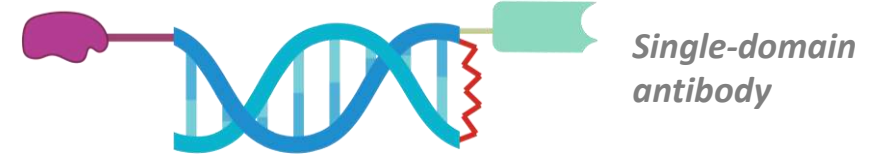
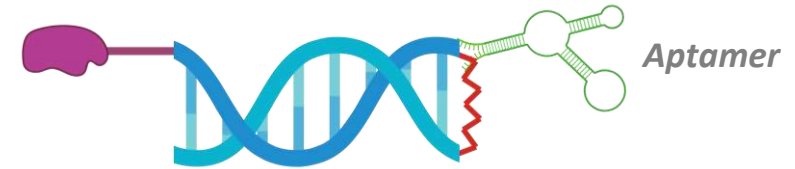
3rd Generation

Combining DNA decoy with PROTAC and tumor specific vectorization to develop cutting edge anti-cancer therapeutics

Active moiety
(protein degradation)



Transcription factor decoy



DecoyTAC

Increase tumor specificity with aptamer, bispecific antibodies, etc.

Agenda



Overview Of Valerio Therapeutics



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DecoyTAC - 3rd Generation

Summary

Overview Of 1st Generation PlatON™ Product, AsiDNA™



1 Key DNA Targeting Decoy

Targeting and sequestering:

1) **DNA Protein Kinase (DNA-PK)**, a key kinase required for DSB repair by NHEJ, which is the primary DNA repair pathway for DSBs in human cells



DNA-PK is a key target for cancer therapies as genetic deficiencies sensitize cancer cells to radiotherapy and DNA damaging agents such as chemotherapy and PARP inhibitors



Trapping DNA-PK To Prevent Repair

AsiDNA™ traps DNA-PK and prevents a response to DNA damage



Potential Combination Therapy

AsiDNA™ can be combined with other DNA damage therapies without additive toxicity



Unique MoA

AsiDNA™ does not target the active site of DNA-PK, relieving the pressure to develop resistance mutations



Strong Patent Protection

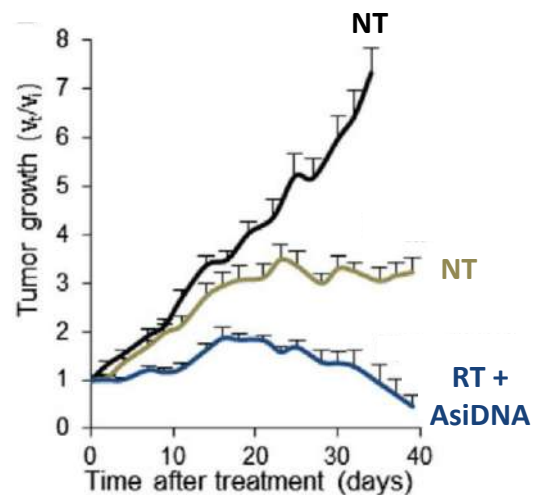
A patent portfolio built on 9 different patent families, granted in major markets, covering compositions of matter, methods of use, and combinations

Preclinical Data Demonstrating Synergistic Effects Of AsiDNA™ With Standard Cancer Therapy



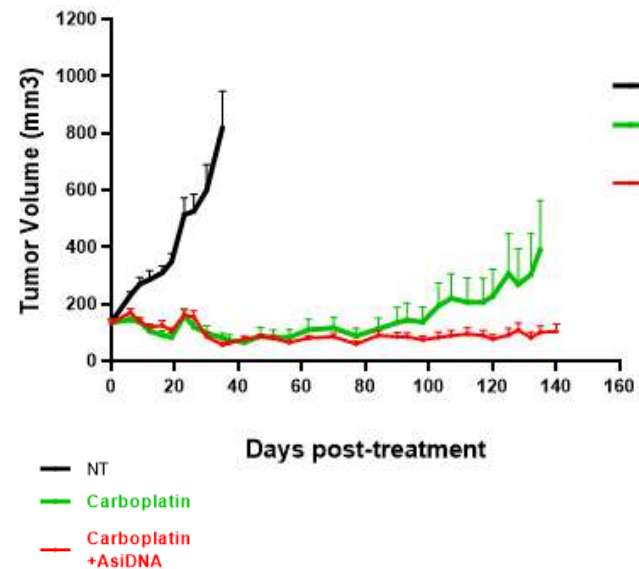
Radiotherapy

SK-28 (Human Melanoma)
xenograft in mice



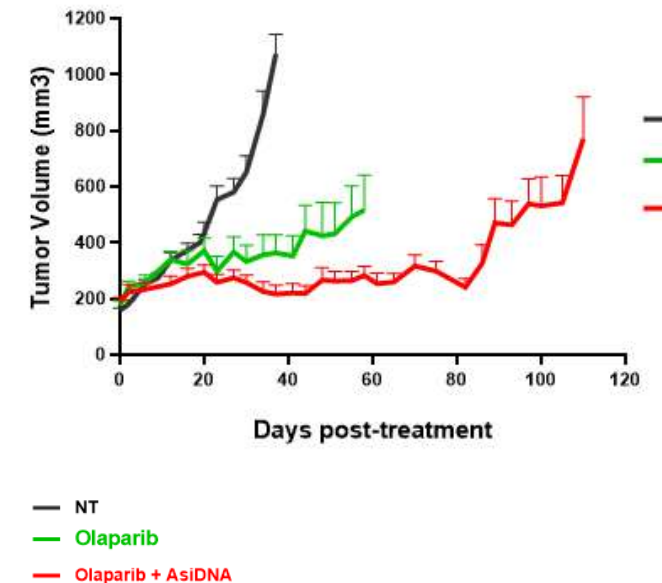
Chemotherapy

MDA-MB-436 (TNBC) xenograft in mice (HRD – BRCA1mutated)



PARP Inhibition



MDA-MB-436 (TNBC) xenograft in mice (HRD – BRCA1mutated)



AsiDNA™ Clinical Proof Of Concept In Solid Tumors



 **Approx. n=80 patients have already been treated with AsiDNA™ to date (completed and ongoing trials)**

Trial	Phase	No. Patients	Status	Key Results
DRIIM IT admin in combo with RT in Melanoma	1	n=23	Completed	<ul style="list-style-type: none"> ✓ ORR = 67% (CR-5%; PR-62%) ✓ Durable response (up to 12-month follow-up period) ✓ Favorable safety profile
DRIIV Part 1 IV admin as monotherapy in advanced solid tumors	1	n=22	Completed	<ul style="list-style-type: none"> ✓ 4 out of 17 evaluable patients achieved SD ✓ Proof of mechanism shown through hyper-activation of DNA-PK at the cell level ✓ Favorable safety profile: No drug-related SAEs < 900 mg; No DLTs < 900 mg
DRIIV Part 2 IV admin in combo w/wo Paclitaxel & Carboplatin	1	n=11	Completed	<ul style="list-style-type: none"> ✓ Heavily pre-treated patients with advanced metastatic tumors, progressing at inclusion ✓ 1 PR and 4 SD yielding a DCR of 55.6% ✓ Favorable tolerability in combination therapy
 REVOCAN (IST) IV admin in combo with PARPi (patients progressing on prior PARPi)	1/2	n=15	Ongoing	<ul style="list-style-type: none"> ✓ Interim analysis (10 patients): 1 CR, 6 SD showing a DCR of 70% ✓ No new safety signals identified
 High Grade GLIOMA (IST) IV admin in combo with RT (children, adolescents & young adults)	1/2	n=5	Ongoing	<ul style="list-style-type: none"> ▪ Not reported yet

IT, intratumoral ; IN, intravenous ; ORR, objective response rate ; DCR, disease control rate ; CR, complete response ; PR, partial response ; SD, stable disease

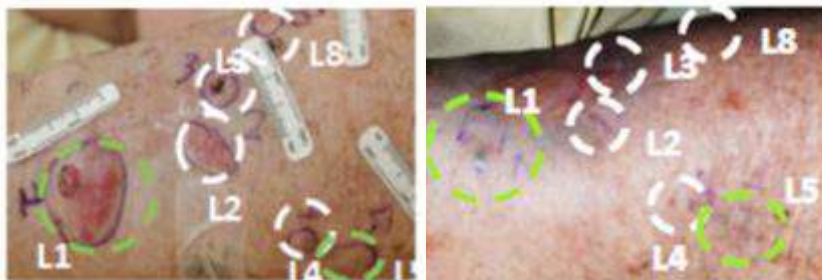
DRIIM: AsiDNA™ Clinical Proof Of Concept Demonstrated In Melanoma



DRIIM: AsiDNA™ Combination with Radiotherapy

- Intratumoral injection
- Combo with Radiotherapy in patients with skin metastases from Melanoma
- N = 23 patients (76 skin lesions)

Melanoma skin lesion responses to treatment

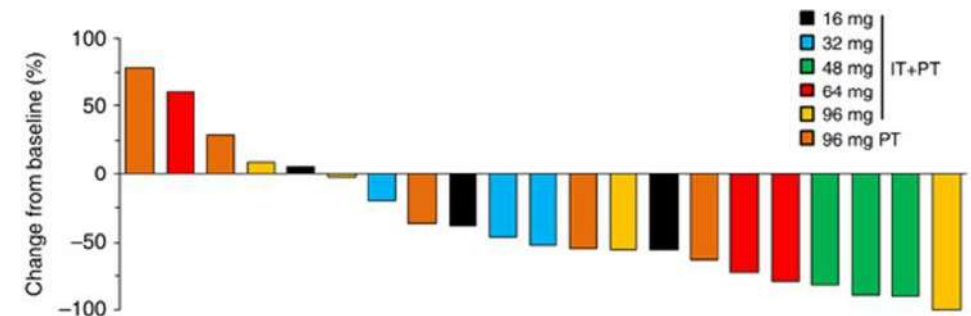


Before Treatment

90 Days After Treatment

Established Proof of Concept

- **Favorable safety profile**
- **Overall response rate (ORR) of 67%** in 21 evaluable patients
 - 1 patient (5%) experienced a CR to treatment
 - 13 patients (62%) experienced a PR to treatment
- **Durable response** (up to 12-month follow-up period)



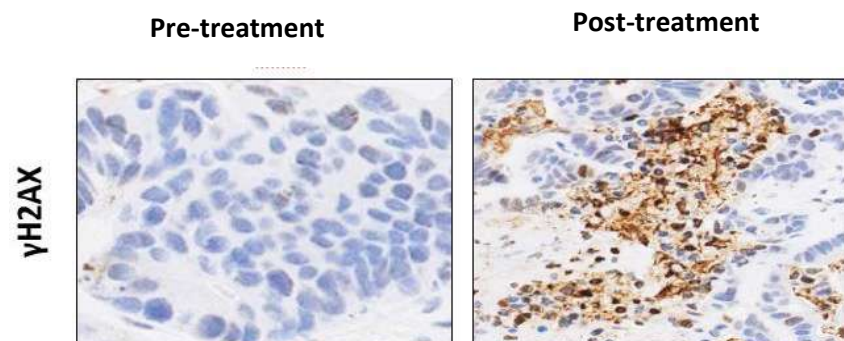
Source: Le tourneau C. et al, Br J Cancer. 2016, Vol.114, Issue 11

AsiDNA™/Chemotherapy Combination Achieves Disease Control In Solid Tumors



DRIIV-Part 1: Favorable Safety Outcome

- Open-label, 3+3 dose escalation n =22
- 5 doses (200mg - 1,300mg)
- Successful development of IV administration
- 600 mg dose selected for clinical development
- Mechanism of action demonstrated



yH2AX (DNA-PK biomarker) readout in tumor biopsies

DRIIV-Part 2: 56% Disease Control Rate

- Open label 3+3 cohorts: Patients eligible to carboplatin +/- paclitaxel
- Heavily pre-treated patients with advanced metastatic tumors, progressing at inclusion
- **No DLT and very good tolerance** of combo therapy in 8 evaluable patients
- **Efficacy signals** in 4 patients: **disease controlled for significantly longer** than with any of the prior lines

AsiDNA™ 600mg	Tumor	Treatment Line	Treatment Duration (Months)	Response
+ carboplatin	TNBC	6 th line	5.5	Stable Disease
	NSCLC (Epidermoid)	3 rd line	8.5	Stable Disease
+ carboplatin + paclitaxel	NSCLC (Adenocarcinoma)	4 th line	3.0	Partial Response - 40%
	NSCLC (Adenocarcinoma)	2 nd line	11	Stable Disease

Source: Kotecki N. Long Stabilization and Disease Control with AsiDNA™, a first-in-class DNA Repair Inhibitor in Combination with Carboplatin. *Oncology & Cancer Case Reports* 2021, Vol.07, Issue 2, 001-007 - 03/2021; Final results to be published

REVOCAN Interim Analysis Shows Positive Efficacy Signals



REVOCAN: Phase Ib/II Study

- 26-patient, open label, multicentric, phase Ib/II to assess the safety and efficacy of AsiDNA™, administered IV in combination to PARPi
- Patients with relapsed platinum sensitive ovarian cancer already treated with PARPi
- 15 patients enrolled to date
- AsiDNA™ is combined with PARPi when patient have increased CA-125 level for two weeks

Interim Analysis On 10 Patients Shows 70% DCR

- One Complete Response (CR)*
- Six Stable diseases (SD)*
- Disease Control Rate: 70%
- Overall reduction in % of CA125 across responding patients: potential sign of disease stabilization

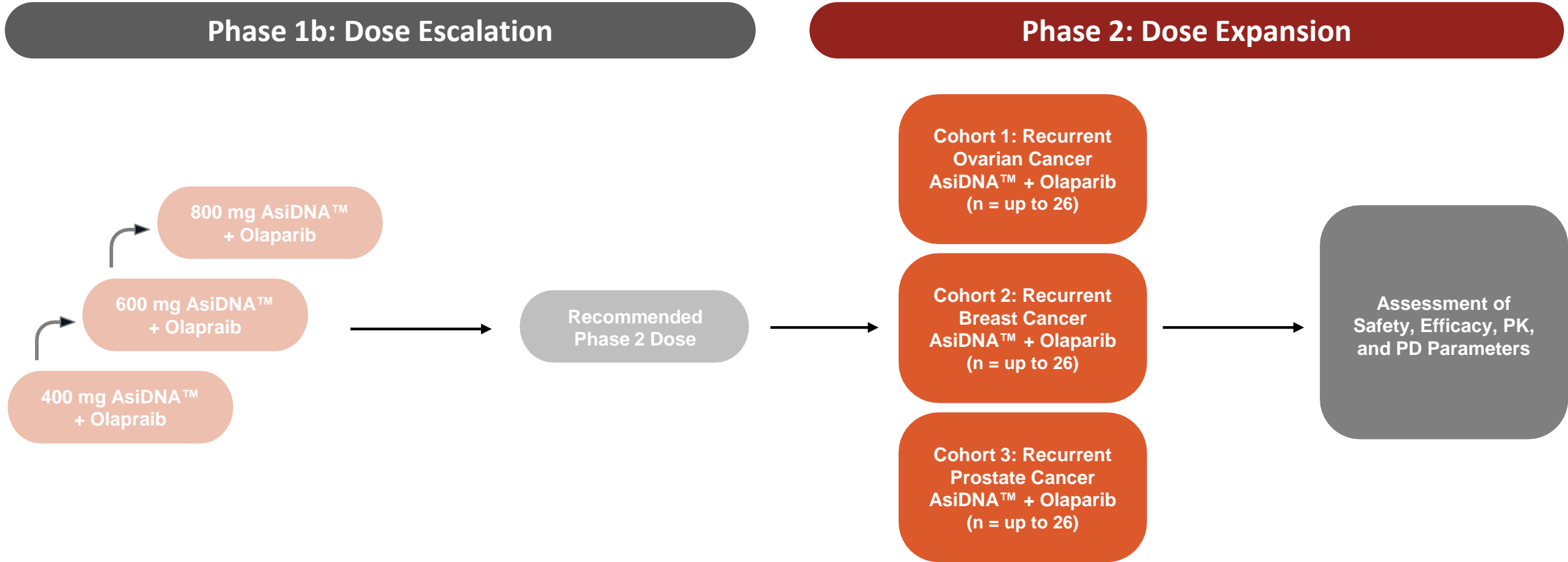


Note: * - as per RECIST 1.1

First US Clinical Trial Initiated: FPI Dosed On 21st March 2023



- *Evaluating AsiDNA™ in combination with olaparib to resensitize patients who have progressed on a prior PARP inhibitor*



Agenda



Overview Of Valerio Therapeutics

AsiDNA™ - Targeted Sequestering Of DNA-PK



VIO-01 - Multi-Modal DDR Decoy

DecoyTAC - 3rd Generation

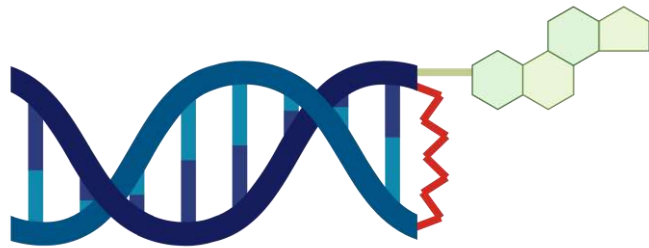
Summary



Overview Of 2nd Generation PlatON™ Product, VIO-01

- *VIO-01 Traps Several DDR Proteins Inhibiting Different Repair Pathways*

1 Key DNA Targeting Decoy



Targeting and sequestering:

- 1) **KU70/KU80**, a part of the NHEJ machinery and is responsible for recruiting other proteins to assist in the NHEJ repair
- 2) **PARP-1**, a key DNA damage sensor protein that identifies and recruits DNA repair machinery to damage sites
- 3) **MRN**, a complex playing an important role in DDR recognition and signaling, and HR or NHEJ repair
- 4) **MSH2**, an important protein involved the recognition of single-base insertions



Multi-Trapping

VIO-01 traps several DNA repair proteins (PARP1, KU70/80, MRN, MSH2)



Pan-Inhibition

VIO-01 inhibits SSB and DSB repair leading to extensive DNA damage accumulation



Independence Of Repair Mechanisms

VIO-01 Displays Antitumor Activity Independently From Repair Status



Higher Antitumor Activity Vs Competitors

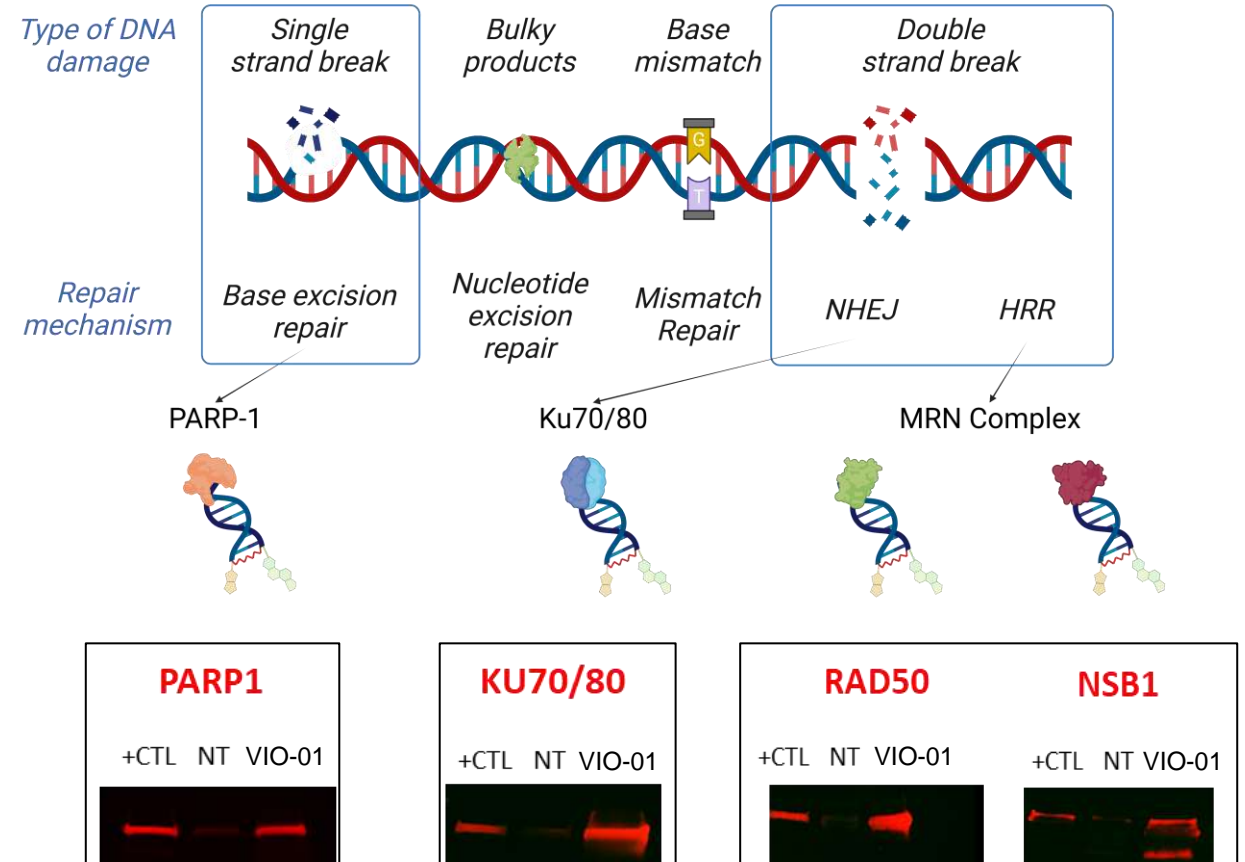
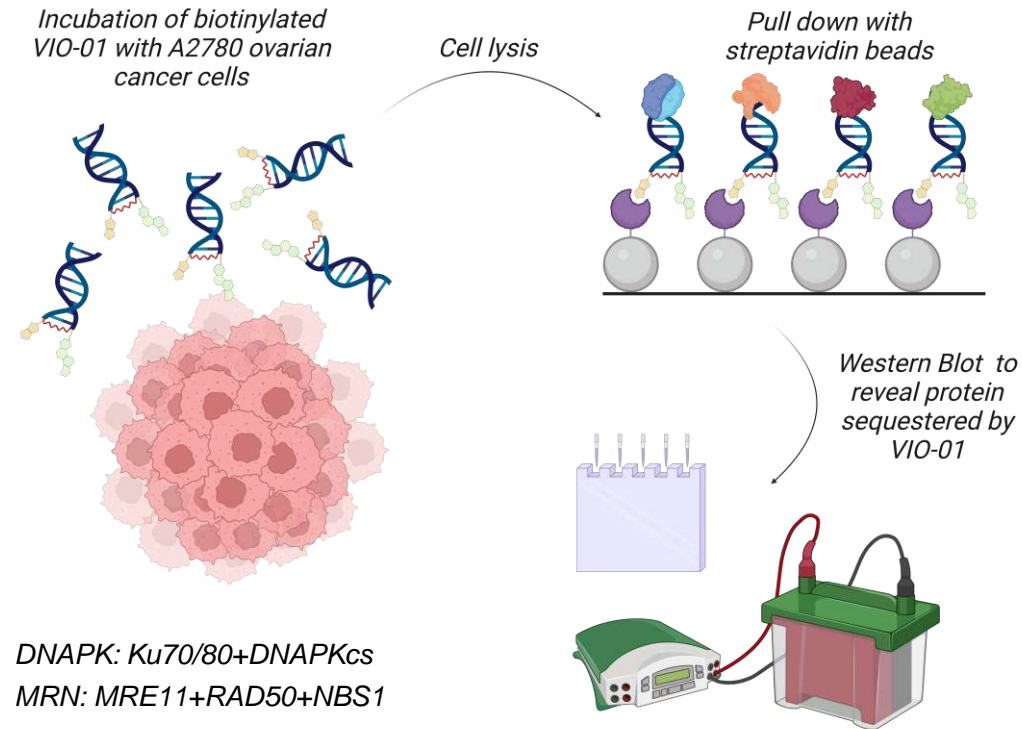
VIO-01 Displays Higher Antitumor Activity Compared To PARP And DNA-PK Inhibitors And An Enhanced Tumor Accumulation



Strong Patent Protection

A patent portfolio built on 3 different patent families, focused on compositions of matter

VIO-01 Is A Pan-DDR Decoy Trapping Multiple Repair Proteins In Tumor Cells

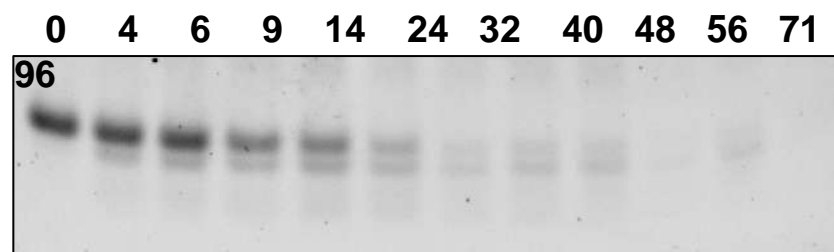


VIO-01 reaches the nucleus and acts as a decoy for several DNA repair enzymes

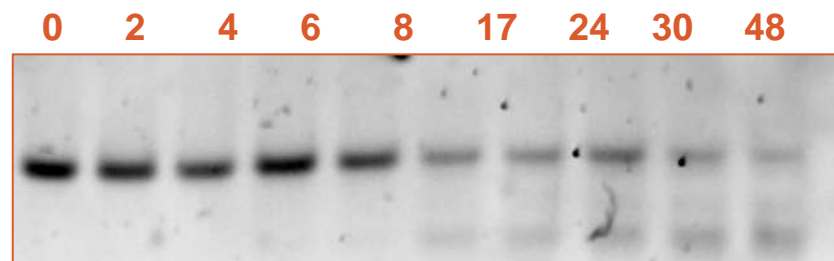


VIO-01 Resists Endonuclease Degradation In Human Serum

VIO-01 Stability In Human Serum

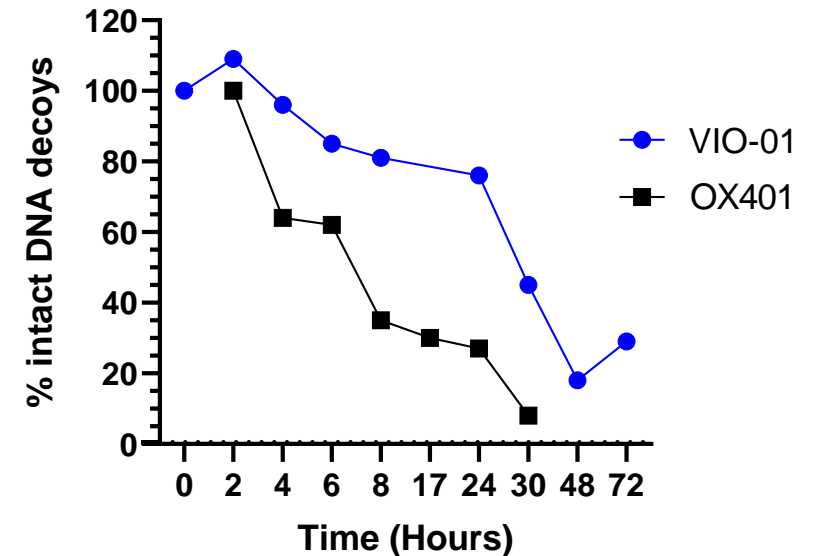


VIO-01 Stability in Human Serum



Electrophoresis of intact versus metabolized DNA decoys after incubation in human serum

VIO-01 versus OX401



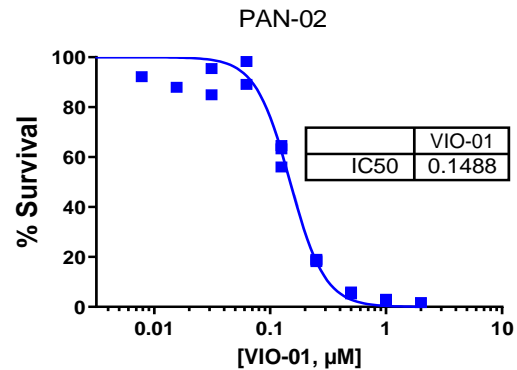
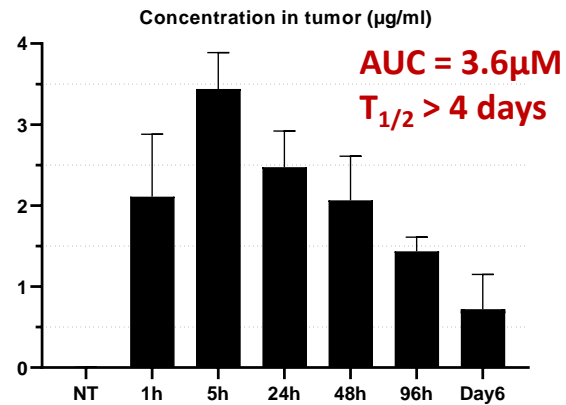
VIO-01: 31% PS and 56% FANA modifications
OX401: just 18% PS

VIO-01 has an increased resistance to nucleases and plasmatic stability

VIO-01 Accumulates In Tumors And Reaches The Nucleus



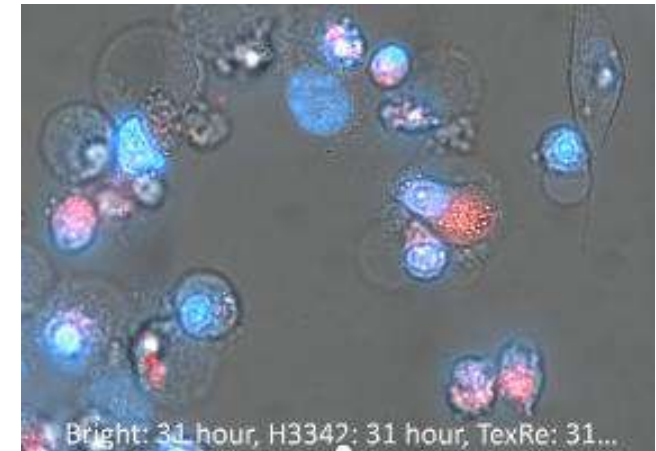
VIO-01 Tumor Accumulation



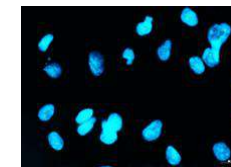
VIO-01 accumulates in tumors at 30x concentration needed to achieve anti-tumor activity for up to 1 week

PAN-02 (Pancreatic cancer) xenograft in mice

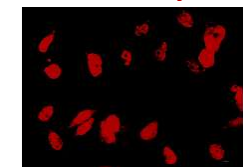
VIO-01 Nuclear Localization



Nucleus



VIO-01-Cy5



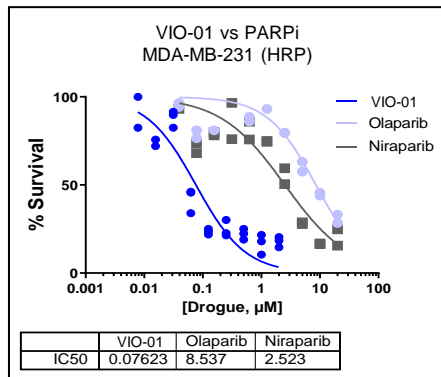
MDA-MB-231 TNBC cells

VIO-01 Displays Higher Anti-Tumor Activity Compared To PARP And DNA-PK Inhibitors

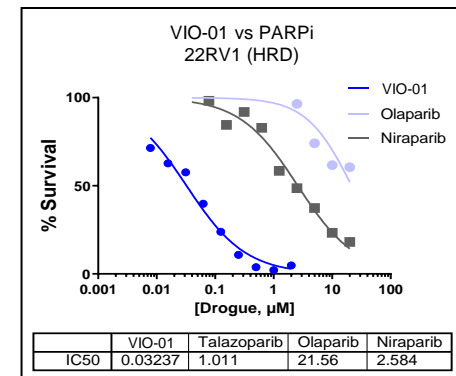
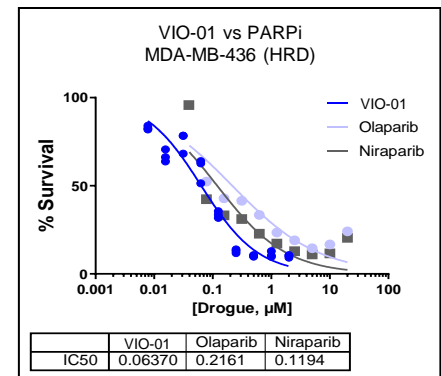
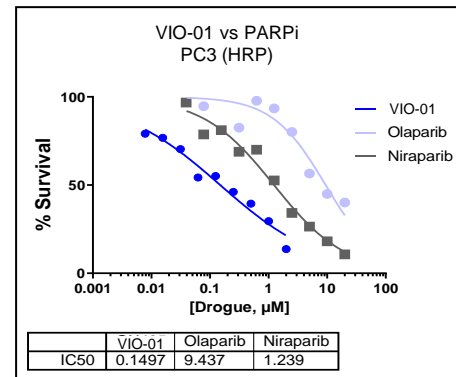


VIO-01 vs PARP Inhibitors

Breast Cancer Models

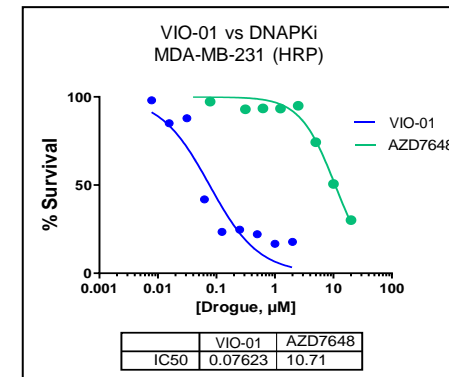


Prostate Cancer Models

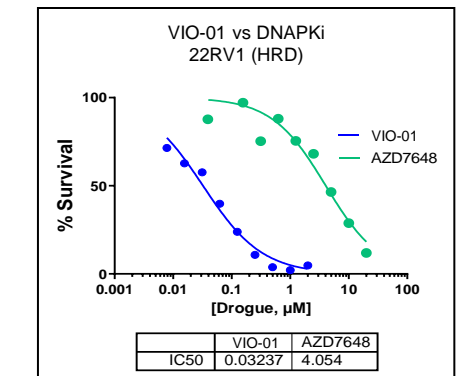
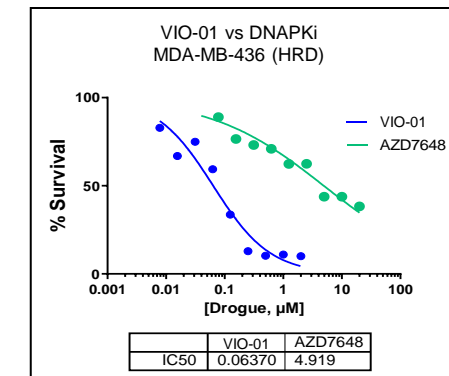
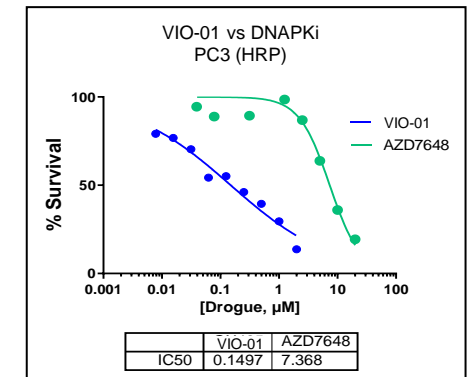


VIO-01 vs DNA-PK Inhibitor

Breast Cancer Models

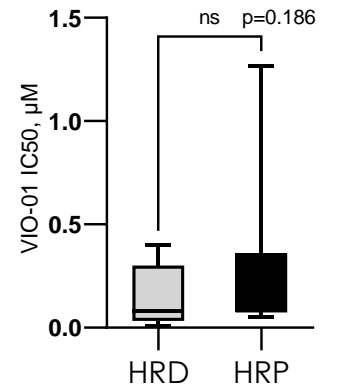
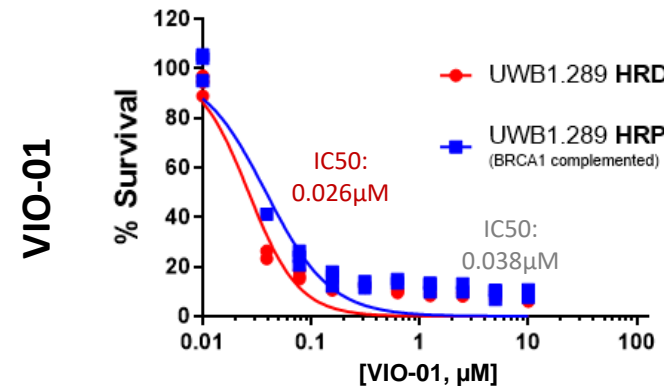
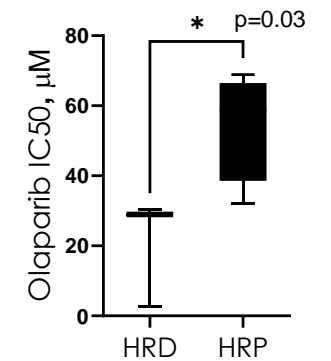
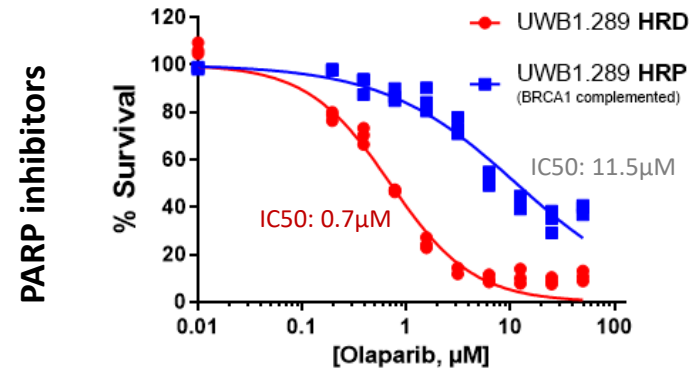
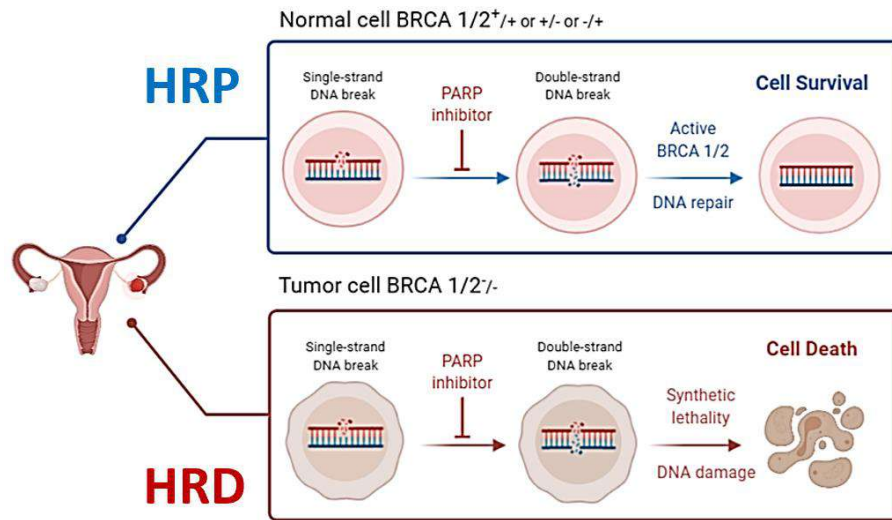


Prostate Cancer Models



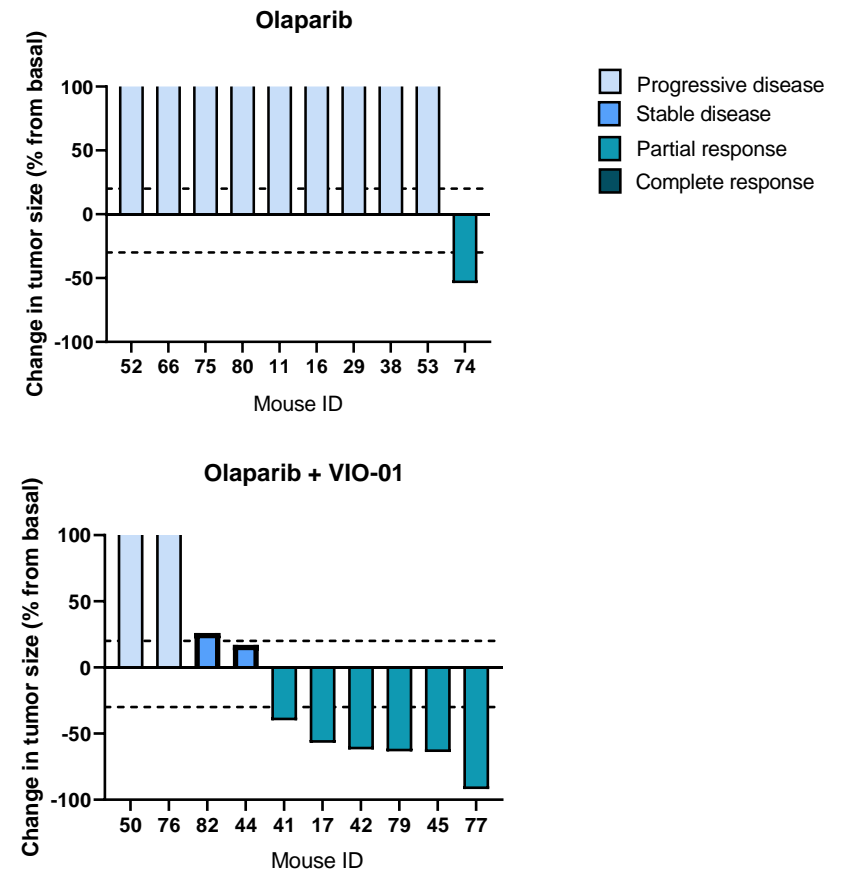
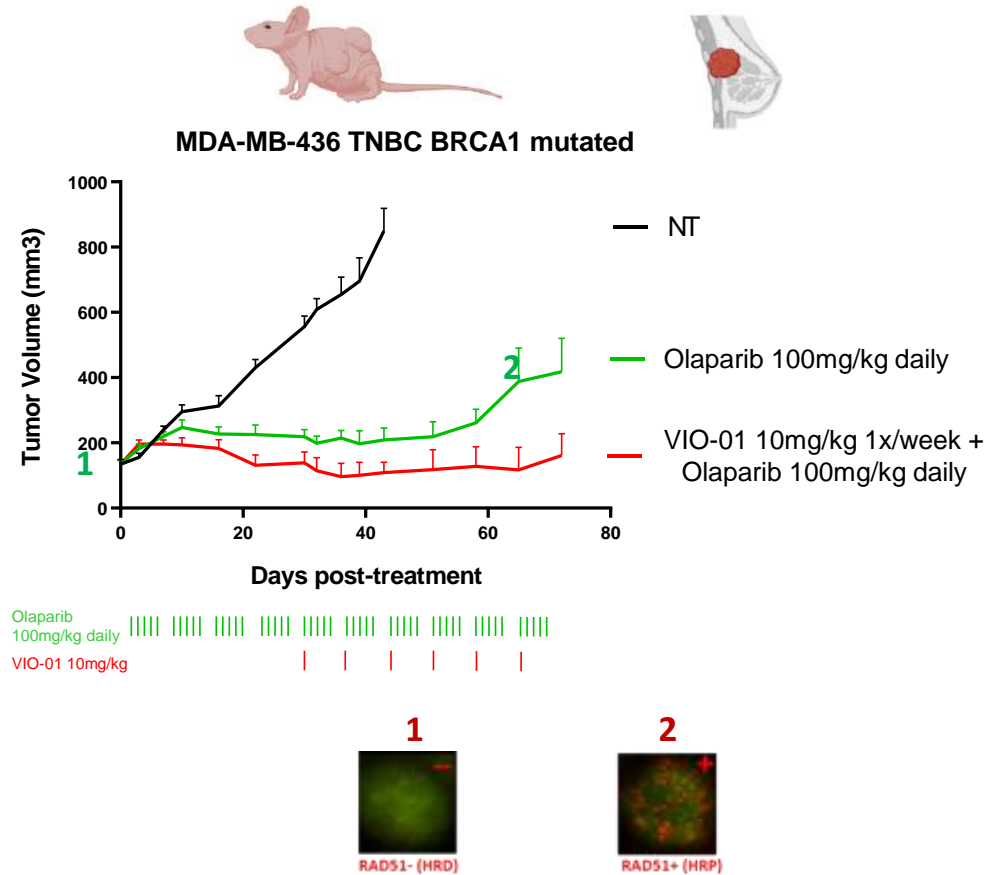
Compared to PARP and DNA-PK inhibitors, VIO-01 is highly active independently of tumor type and genetic mutation

VIO-01 Anti-Tumor Activity In Ovarian Cancer Is Independent Of Genetic Mutations



- PARP inhibitors require defects in HRR to be active (ie: BRCA1/2 mutation)
- Compared to PARP inhibitors, VIO-01 display the same activity in tumor cells with no defects in DNA repair

VIO-01 Resensitizes Tumors That Have Acquired Resistance To Olaparib



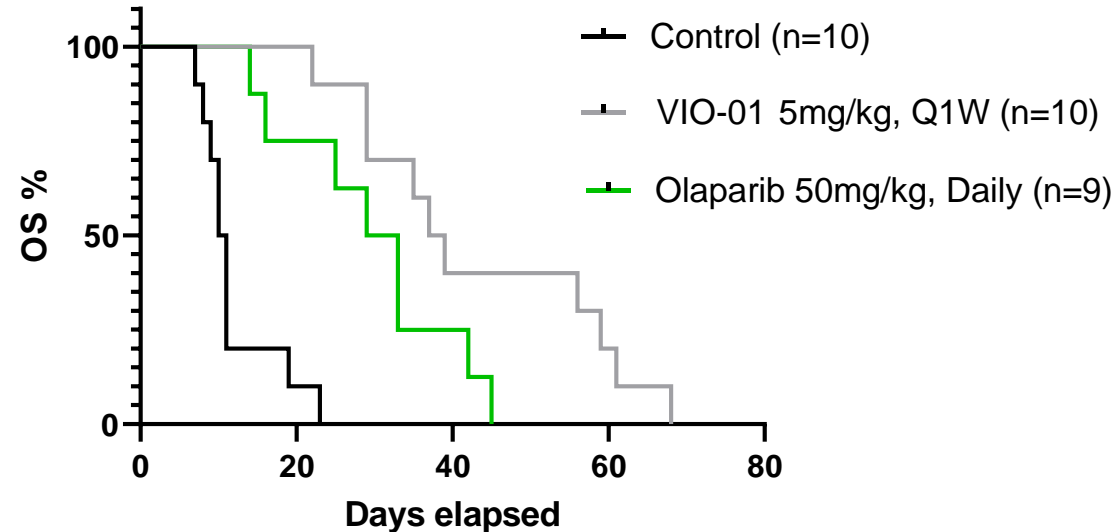
- MDA-MB-436 CDXs developed a rapid acquired resistance to Olaparib (one to two months after treatment start)
- Addition of VIO-01 during early resistance delayed tumor progression

VIO-01 Displays Potent Antitumor Activity *In Vivo* Compared To Olaparib In HRD Models



M/D-driven Breast Cancer – Orthotopic Homologous Recombination Repair Deficient (HRD)

Survival

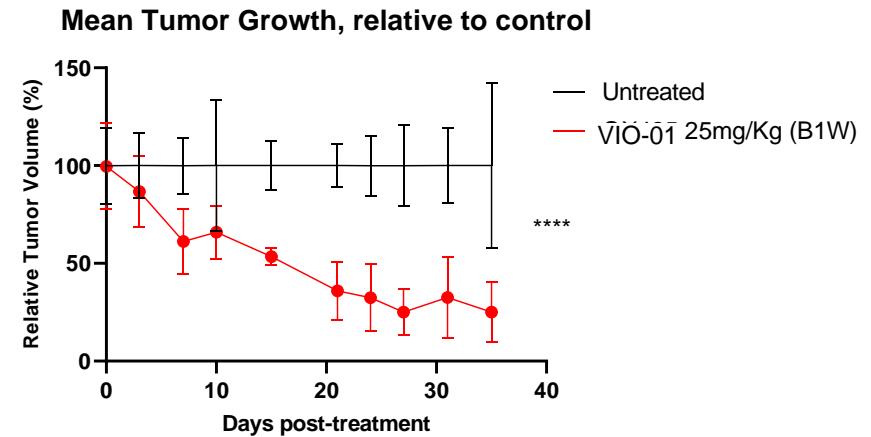
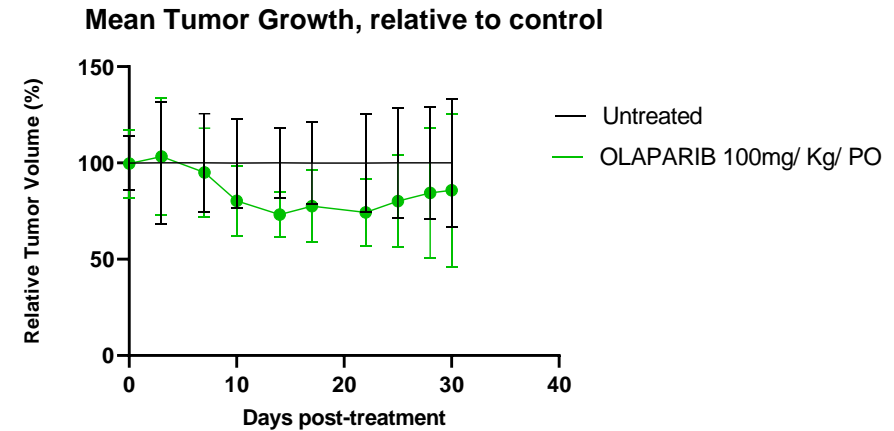
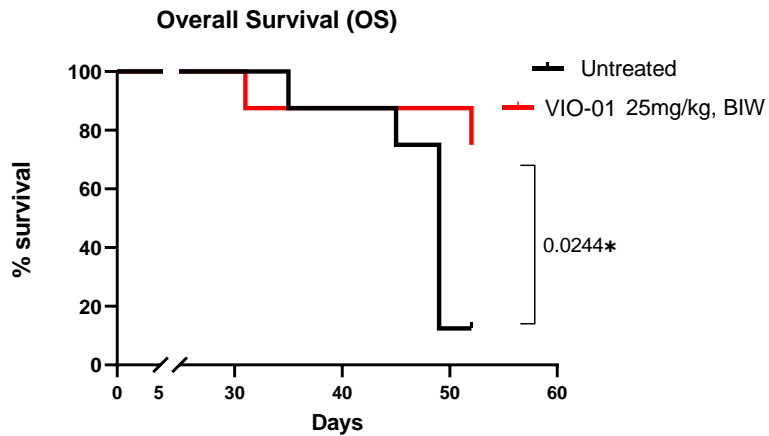
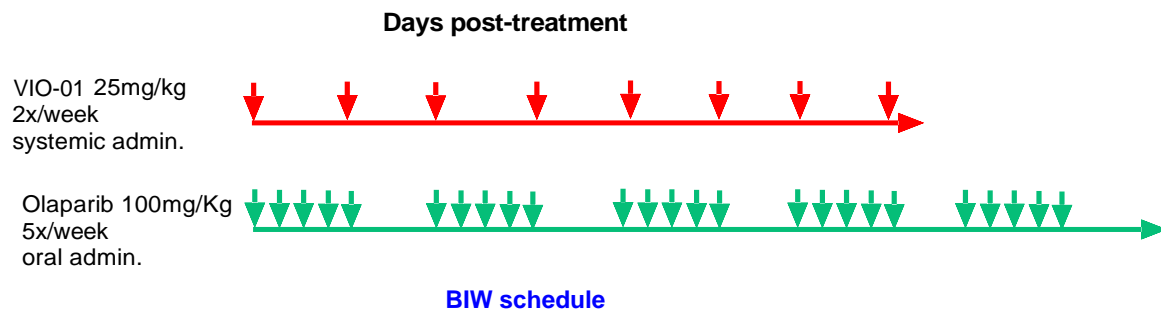


VIO-01 significantly improved OS compared to Olaparib even at a cumulative 50 times lower dose

VIO-01 Displays Potent Tumor Shrinkage In Pancreatic Models Insensitive To Olaparib



PAN-02 (Pancreatic Cancer) Xenograft In Mice Homologous Recombination Repair Proficient (HRP)



VIO-01 is more active in HRP tumors despite a x4 lower dose compared to Olaparib

Preliminary Results From The IND-Enabling GLP Toxicology Study In NHP Demonstrates VIO-01's Favorable Safety Profile



4-week treatment / 4-week recovery IND enabling study

- No evidence of myelosuppression
- Transient increases in complement factors and cytokines
- No body weight loss
- Minimal neuro-toxicity at high doses
- No liver or kidney toxicities

Wide therapeutic window: minimal active dose 1.25mg/kg (equivalent to 5mg/kg in mice)

Clinical chemistry (Day 25, n=5)

	Vehicle	VIO-01 20mg/kg
AST _{U/L}	52	43 _{ns}
ALT _{U/L}	55	50 _{ns}
ALB _{g/L}	46	44 _{ns}
UREA _{mmol/L}	7	6.25 _{ns}

ns: Not significant compared to vehicle

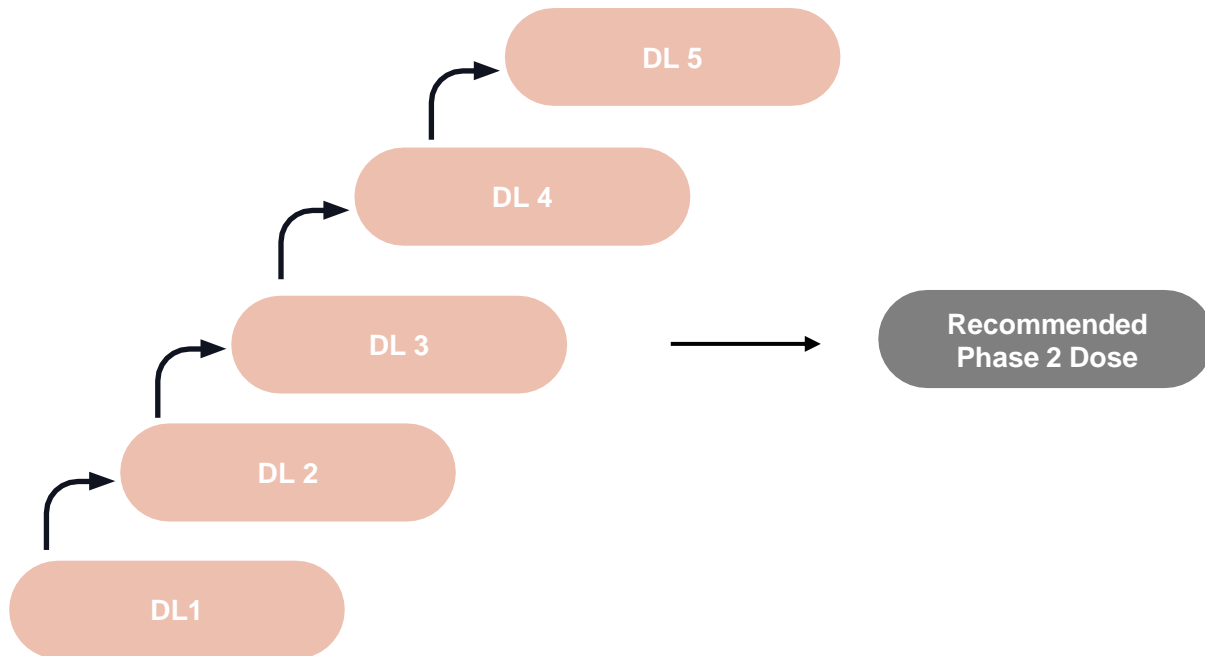
Compared to Olaparib, VIO-01 did not induce neutropenia, thrombocytopenia or anemia



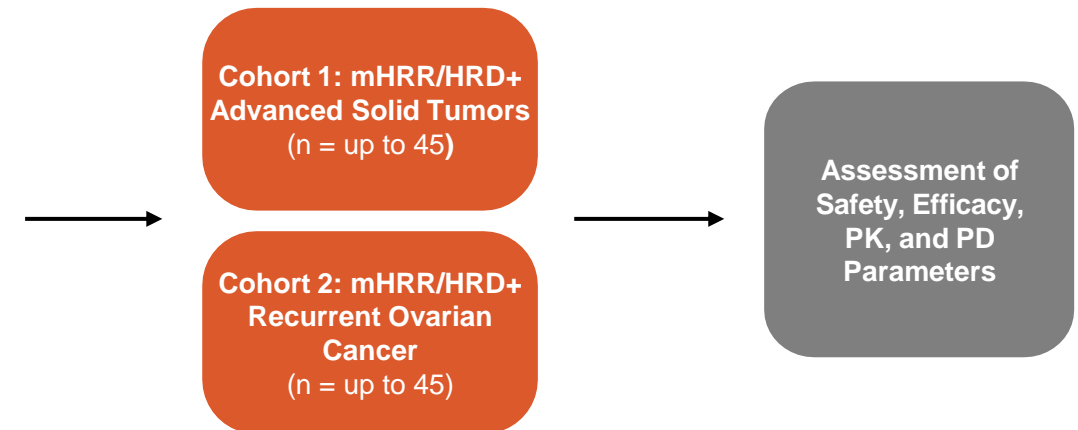
VIO-01 Proposed IND Opening Study: FPI Expected 2H23

- Pursuing tumors with HRR genomic alterations or HRD+ score
- (BRCA1, BRCA2, ATM, ATR, CHK1, CHK2, DSS1, RPA1, NBSI, FANCD2, FANCA, CDK12, PALB2, BRIP1, RAD51B, RAD51C, RAD51D, RAD54)

Phase 1b: Dose Escalation



Phase 2: Dose Expansion



Agenda



Overview Of Valerio Therapeutics

AsiDNA™ - Targeted Sequestering Of DNA-PK

VIO-01 - Multi-Modal DDR Decoy



DecoyTAC - 3rd Generation

Summary



Transcription Factors, DDR, and Epigenetics Are A Unique Class Of Oncology Targets

Transcription Factors are proteins that **bind to DNA and mediate gene expression** programs involved in fundamental cellular processes such as differentiation, cell death, and proliferation.

Mutated or dysregulated **transcription factors** are frequently **expressed in numerous cancers** leading to aberrant downstream genes expression

Mutated transcriptions factors are **key drivers of tumor formation** and oncogenesis

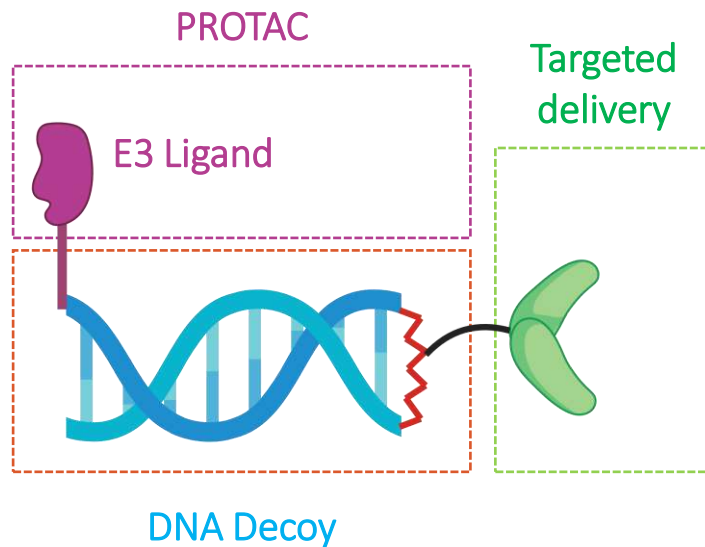
Undruggable - traditional small molecule inhibitors and antibodies are limited in the ability to drug transcription factors due to their interaction with DNA

DNA decoys can bind and trap transcriptions factors overcoming the key limitations of other therapies

DecoyTAC - Targeting Oncogenic DNA interacting proteins (such as Transcription Factors)



DecoyTAC: Degradation Of TF Through UPS



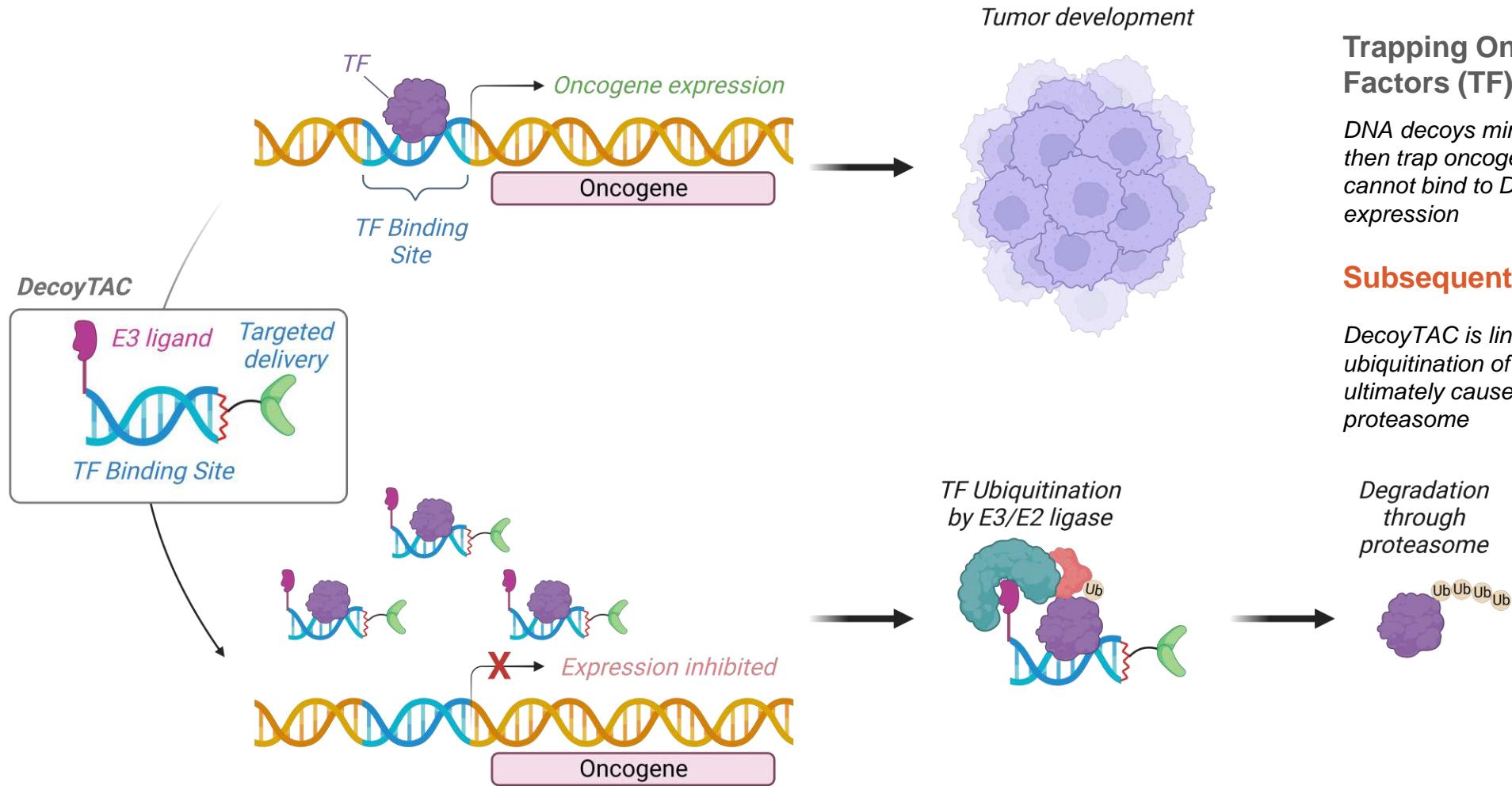
DecoyTAC can drug the “undruggable”

- Does not rely on an enzymatic binding pocket for activity
- Can enter the nucleus
- Can mitigate off-target effects through use of selective delivery technologies
 - Bispecific Small Molecule ligands – PSMA, FOLRa, EphA2
 - Nanobodies
 - Aptamers

DecoyTAC combines DNA decoy with protac and tumor specific vectorization for targeted suppression of oncogene expression



Mechanism of Action of DecoyTAC



Trapping Oncogene Transcription Factors (TF)

DNA decoys mimic TF binding site (**decoy**), then trap oncogene transcription factor. TF cannot bind to DNA and induce oncogene expression

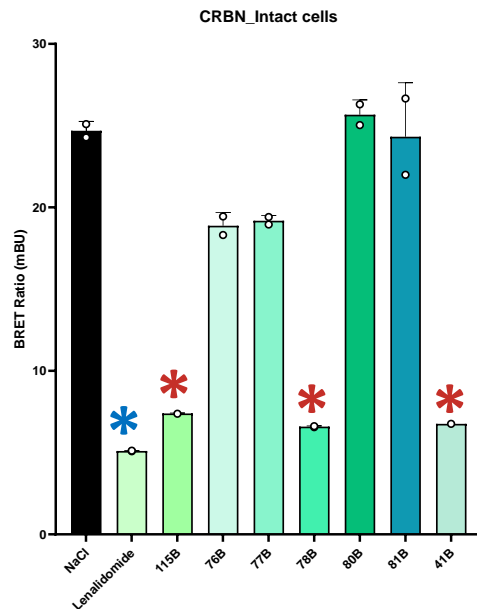
Subsequent TF Degradation

DecoyTAC is linked to E3 ligand leading to ubiquitination of the Oncogene TF, which ultimately causes TF degradation through the proteasome

DecoyTAC Proof Of Concept Validated In Discovery Stage Assays

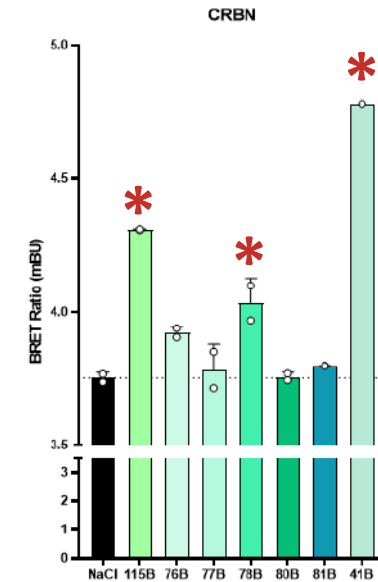


Cell Membrane Permeability Analysis



DecoyTAC screening compounds almost as permeable as the **small molecule PROTAC positive control**

Formation Of Ternary Complex Analysis



DecoyTAC traps the target TF and recruits E3 ligase

DecoyTACs are efficiently delivered to tumor cells and form ternary complexes with the target TF and E3 ligase

Agenda



Overview Of Valerio Therapeutics

AsiDNA™ - Targeted Sequestering Of DNA-PK

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DecoyTAC - 3rd Generation



Summary

Financial Resources To Execute Key Development Milestones Objectives

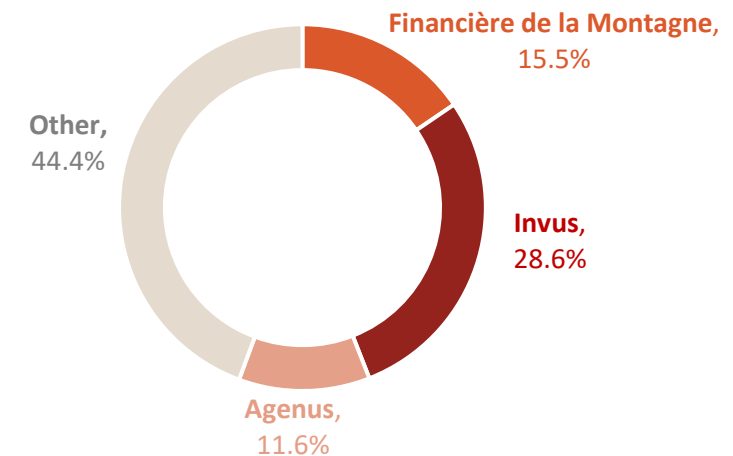


- Net cash position of €14.6 million as of December 31, 2022*
- Financial visibility until the 2nd quarter of 2024

Listing Details

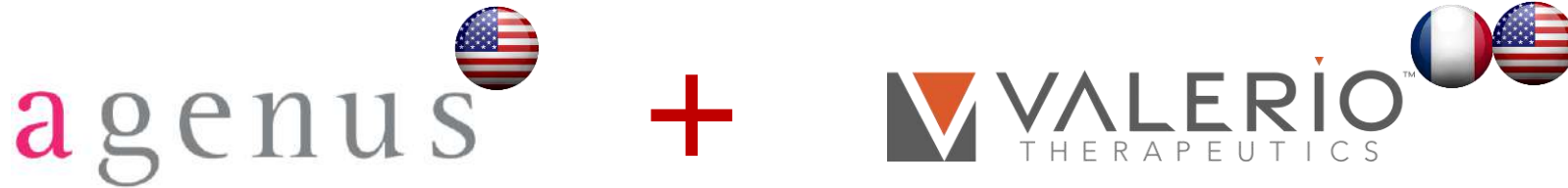
EURONEXT GROWTH | Paris - EPA: **ALVIO**
ISIN: FR00100955

Strong Support From Two Core Shareholders



* - including EUR 14.3 million commitment from Invus, Financière de la Montagne and a new investor

Valerio's Strategic Collaboration With A Leading US Immuno-Oncology Biotech Agenus



Strategic Collaboration

€5M

equity investment in Valerio Therapeutics

Potential Cross-Access To The Other Party's Technologies



Agenus Overview

- Agenus is a NASDAQ-listed Biotech with a market capitalisation of approx. \$656M (NASDAQ: AGEN)
- The Company is a clinical-stage, immuno-oncology Biotech with a R&D pipeline of 13 clinical programs (lead program Botensilimab in phase 2 in various solid tumour types) and has 7 partnerships with leading Big Pharma including BMS, Gilead, Merck & Co and GSK



Details of Collaboration

- Agenus has agreed to acquire 17,857,143 shares representing 11.57% of Valerio's capital and voting rights
- Participation in this operation is carried out with the general aim of supporting the development of oncology drug candidates, which includes the company's development portfolio and potential future scientific partnerships
- Possible ways to implement such potential future scientific partnerships could take the form of a collaboration agreement, license agreement, access by Agenus to Valerio's DDR technology platform, support by Valerio of Agenus development plans, etc.

Near Term Catalysts Driving Value Over The Next 18 Months



PlatON™	Program (Route Of Admin)	Target Indication	Current Stage	2023				2024			
				1Q	2Q	3Q	4Q	1Q	2Q	3Q	4Q
1 st Generation	AsiDNA™ 1.0 (IV)	Recurrent ovarian, breast, prostate cancer (combination with PARPi)	Phase I	Phase I / Phase II							
		Maintenance for ovarian cancer with rising CA-125 (combination with PARPi)	Phase I/II	Phase I/II							
		Pediatric and adolescent glioma (combination with radiotherapy)	Phase I/II	Phase I/II							
2 nd Generation	VIO-01 (IV)	mHRR or HRD+ solid tumors (monotherapy)	IND	Phase I Dose Escalation				Phase II			
3 rd Generation	DecoyTAC	Undisclosed	Discovery	Undisclosed							

-  - First in human trial start
-  - Publication / Abstract Presentation
-  - Full readout
-  - End of clinical trial meeting

Valerio Therapeutics Investment Highlights



- Unique MoA Sequestering DDR Proteins**
 - DNA fragments called **DNA decoys** mimic DNA damage and trap multiple proteins in charge of repairing DNA, leading to an accumulation of unrepaired DNA and tumor death
- 1st-In-Class DNA Decoy Therapeutics Pipeline**
 - **AsiDNA™ (DNA-PK) - Phase I/II** - x3 oncology trials ongoing in combination with PARP or radiotherapy
 - **VIO-01 (MRN, KU70/80, PARP-1) - IND** - Highly promising pre-clinical monotherapy data and superior data in head-to-head studies with competitive landscape
- PlatON™: A Versatile Toolbox And Library Of DNA Sequences**
 - DNA decoy capabilities beyond DDR proteins including transcription factors, and incorporating additional targeting moieties such as E2/E3 ligases, aptamers and bispecific antibodies
 - Early-stage PoC data using PROTAC-like approach to permanently degrade target proteins
- Deep Industry Experience**
 - **Management / Advisors:** Strong management and R&D team located across France and US, supported by a world-class DDR-focused SAB
 - **Investors:** Invus, Agenus, Financiere de la Montagne
 - **Cash Runway:** Financial visibility until 2Q24
- Near Term Catalysts**
 - AsiDNA™ (Phase I): Ovarian, breast, prostate cancer (with PARP) interim clinical trial readout in 2Q24
 - AsiDNA™ (Phase I/II): Ovarian (with PARP) clinical trial readout at the end of 1Q24
 - AsiDNA™ (Phase I/II): Glioma (with radiotherapy) clinical trial readout at the end of 1Q24
 - VIO-01 (Phase I): Entering Phase I in 4Q23