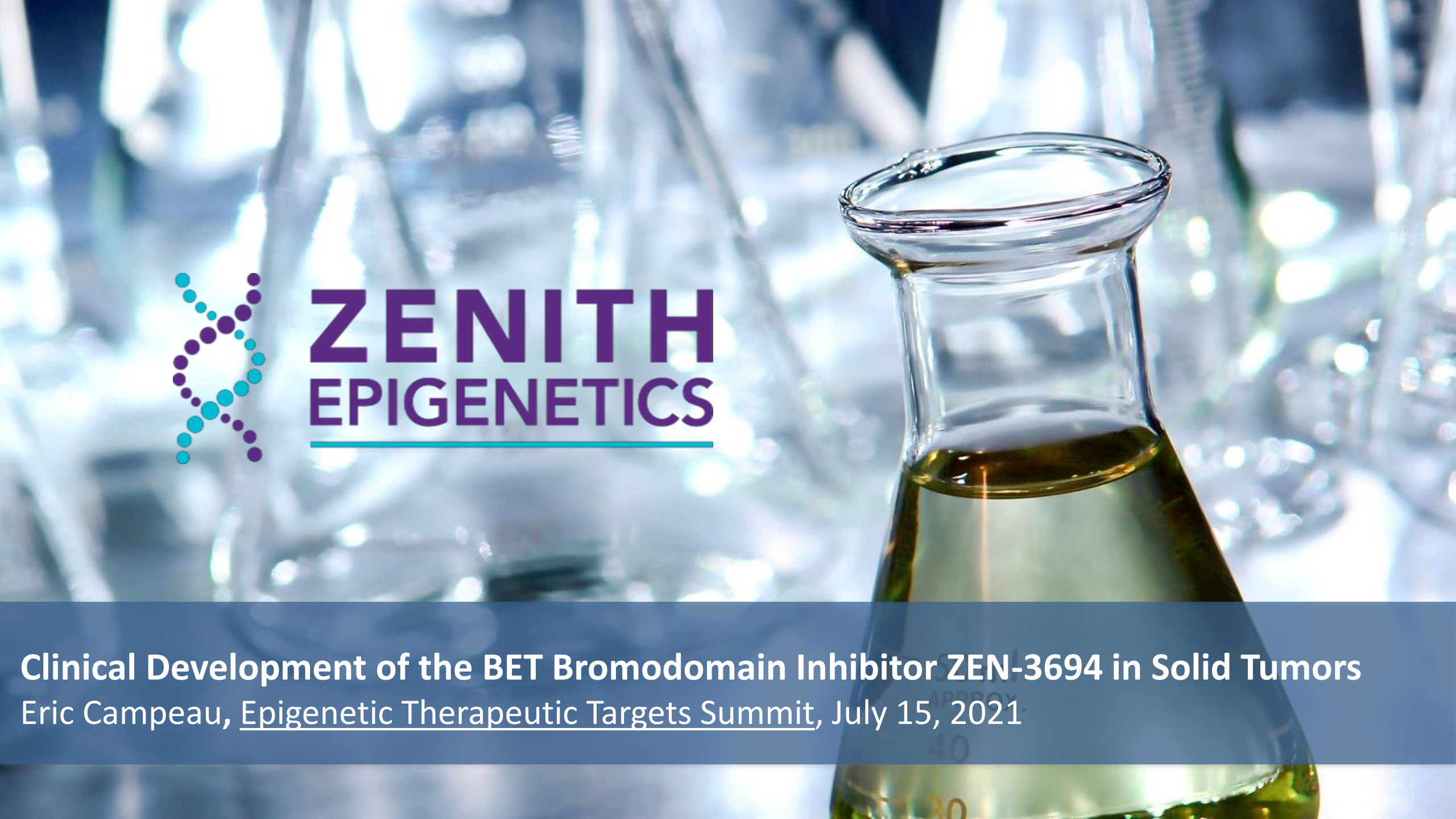
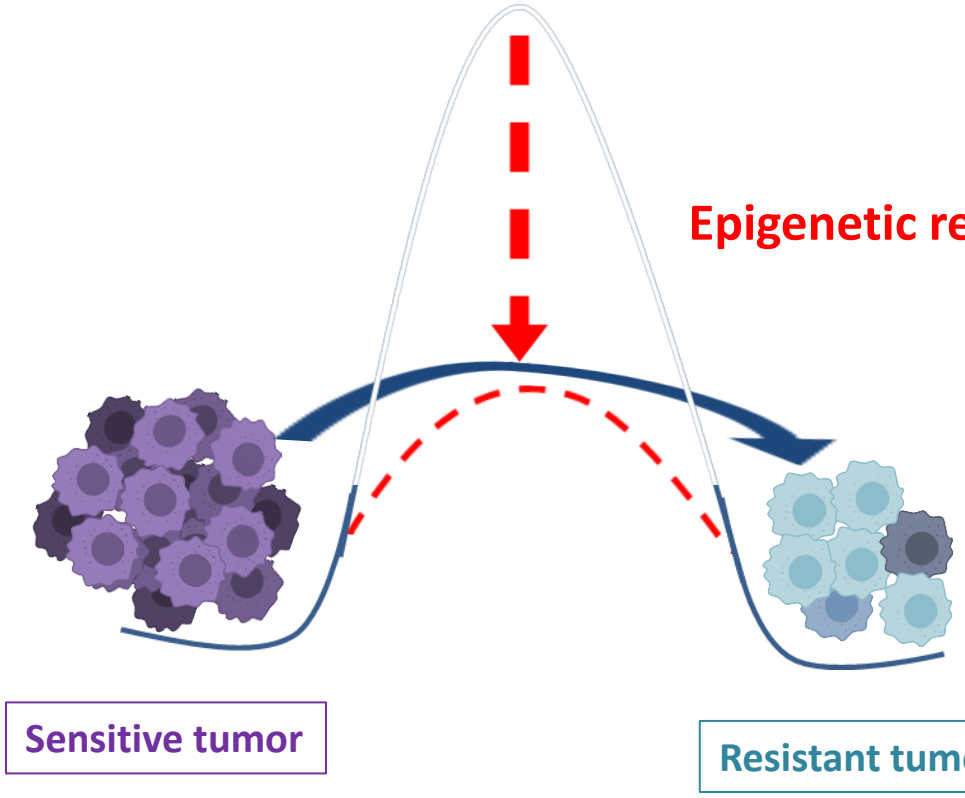


ZENITH EPIGENETICS

Clinical Development of the BET Bromodomain Inhibitor ZEN-3694 in Solid Tumors

Eric Campeau, Epigenetic Therapeutic Targets Summit, July 15, 2021

Acquired resistance to anti-cancer therapies through epigenetic mechanisms

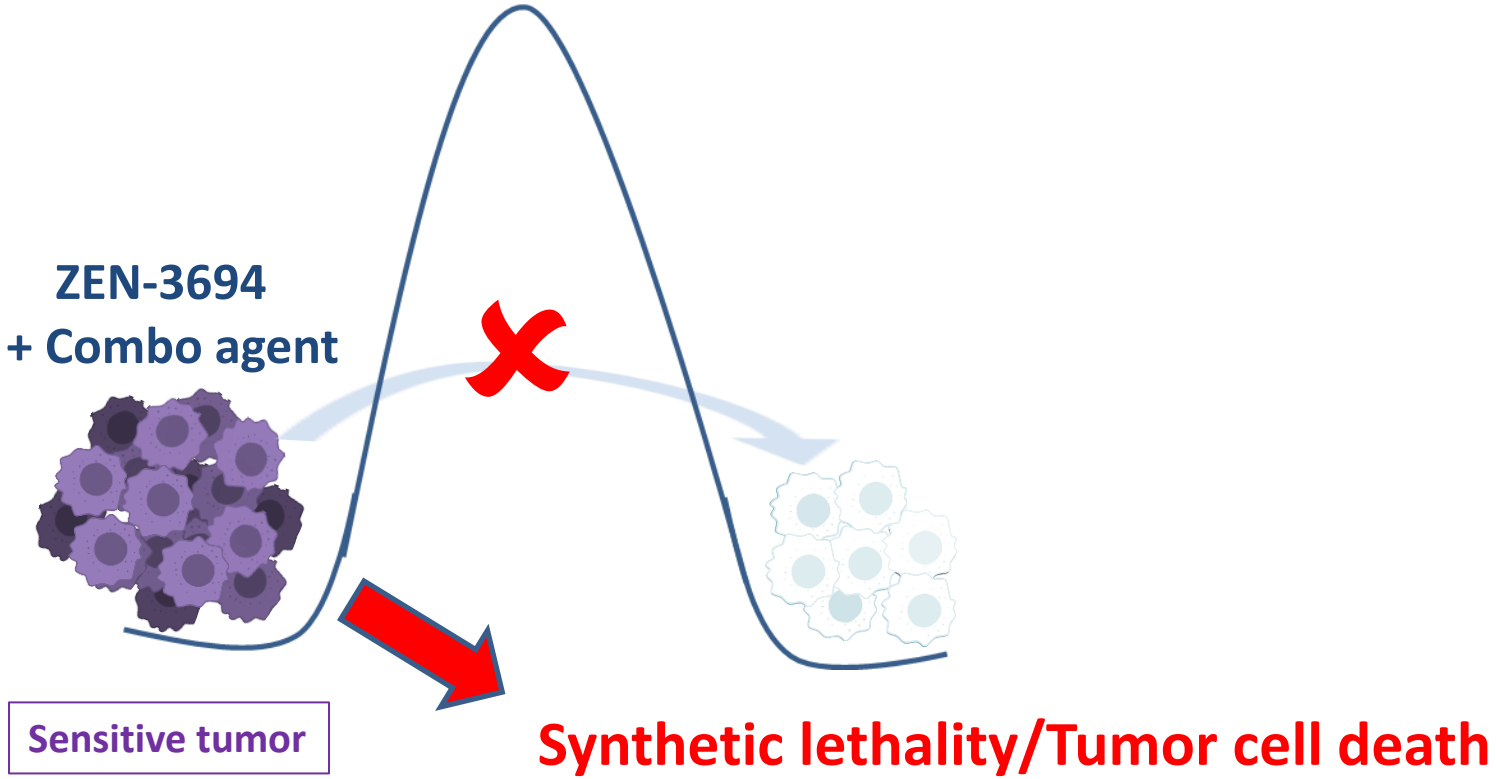


Epigenetic remodeling

- Enhancer reprogramming
- Kinome reprogramming
- Lineage plasticity

- **Tumors initially respond to treatment**
- **Acquisition of drug resistance almost invariably occurs**
- **Epigenetic mechanisms often involved**
- **Epigenetic inhibitor to prevent and/or reverse resistance**

Targeting epigenetic mechanisms of resistance to anti-cancer therapies: examples with the BET bromodomain inhibitor ZEN-3694



Two examples from recent clinical trials with ZEN-3694:

- **Reversion of ARSI resistance → AR-independent resistance in prostate cancer**
- **Induction of synthetic lethality → PARP inhibitor in BRCA1/2 wild-type triple-negative breast cancer**

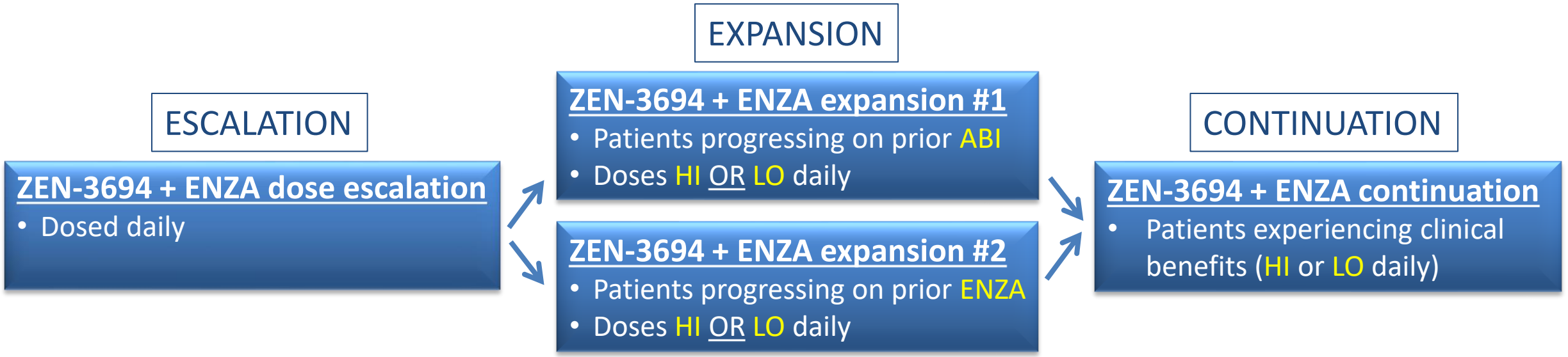
A Phase 1b/2a Study of the Pan-BET Bromodomain Inhibitor ZEN-3694 in Combination with Enzalutamide in Patients with Metastatic Castration Resistant Prostate Cancer

Aggarwal et al. Clin. Can. Res. 2020

Kim et al. Clin. Can. Res. 2021

Phase 1b/2a: ZEN-3694 in combination with enzalutamide in mCRPC

(NCT02711956, NCT04145375)



Summary of findings:

- **75 patients dosed, MTD not reached → RP2D 96mg**
- **ZEN-3694 target engagement seen in whole blood and tumor biopsies**
- **Clinical activity at well tolerated doses, prolonged daily dosing without dose interruptions/reductions**
- **Clinical activity seen at LO and HI doses**

⇒ One ongoing patient at LO dose (> 4.3 years with PSA90 response, prior progression on ABI)

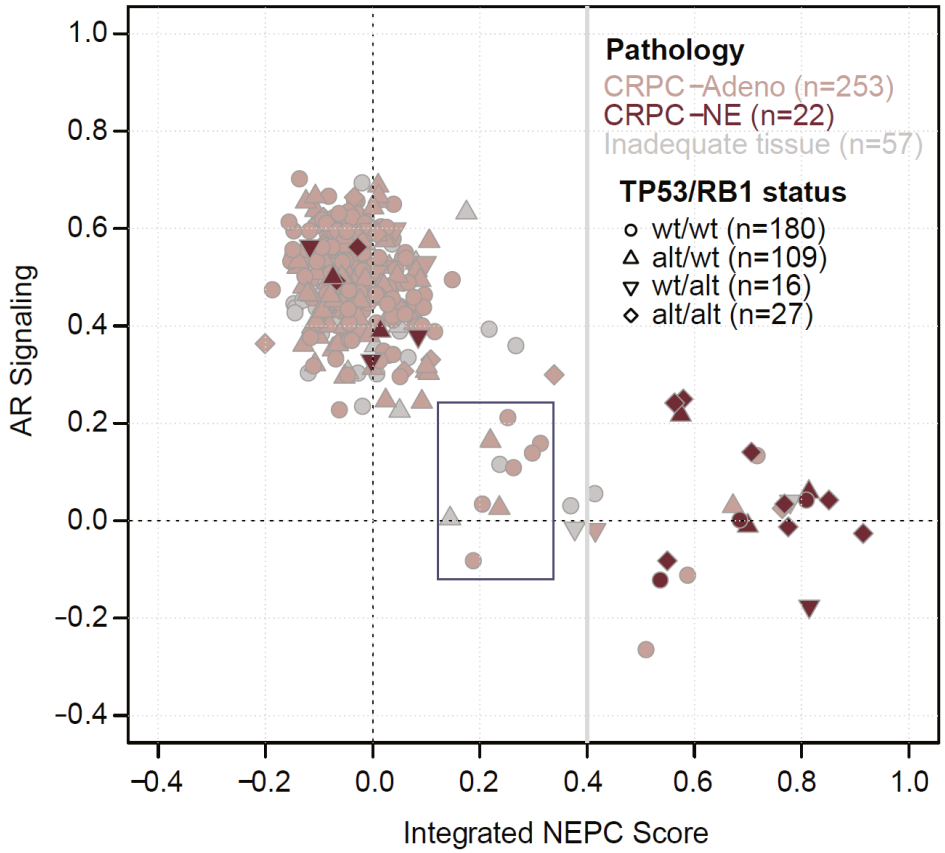
⇒ One ongoing patient at HI dose (> 2.7 years, prior progression on bicalutamide, ABI, and ENZA)

- **Median radiographic progression-free survival of 9.0 mo vs. 3 mo** (historical value for second line ARSI)
- **Evidence for activity in tumors from patients with low androgen receptor (AR) signaling**

ABI = abiraterone; ARSI = AR Signaling Inhibitor; ENZA = enzalutamide; mCRPC = metastatic castration-resistant prostate cancer, RP2D = recommended Phase 2 dose
HI Dose = 96 mg ZEN-3694, LO Dose = 48 mg ZEN-3694

Aggarwal et al. CCR 2020

Loss of AR signaling is associated with gain of neuroendocrine characteristics (NEPC): lineage plasticity



AR signaling score: 21 gene signature upregulated upon incubation of prostate cancer cell line with androgen

Integrated NEPC score: 70 gene signature upregulated in NEPC

- **Shift from adenocarcinoma (AR-dependent) towards neuroendocrine (AR-independent) → lineage plasticity**

⇒ Involvement of several epigenetic processes

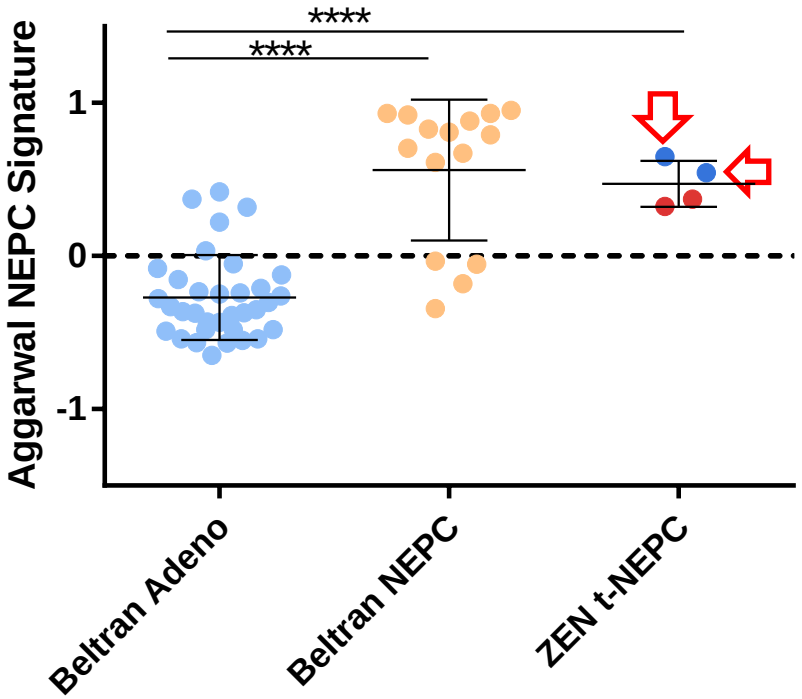
- **Occurs in ~20% of patients treated with ARSI → associated with poor prognosis**
- **Treatment-induced NEPC (t-NEPC): limited treatment options (unmet treatment need)**

Abida et al. 2019, Beltran et al. 2016

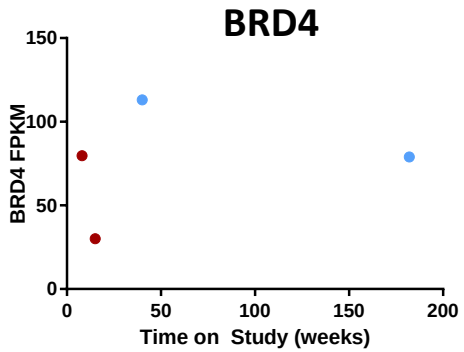
ZEN-3694 blocks a BRD4/E2F1 lineage plasticity program associated with ARSI resistance in prostate cancer

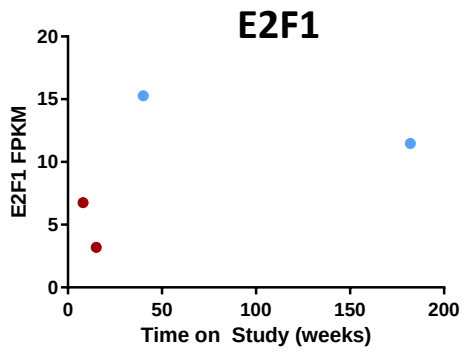
- Identification of a BRD4/E2F1 axis responsible for lineage plasticity in prostate cancer
- Two t-NEPC patients on ZEN-3694 + ENZA trial with BRD4^{HI}, E2F1^{HI}, AR^{LO},(+ AR repressed signature) had longer time on study

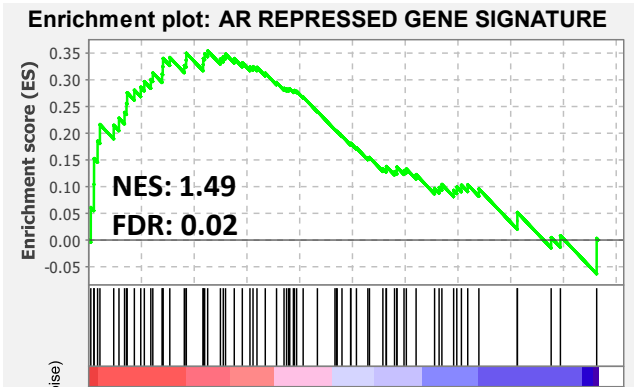
Baseline tumor biopsies from four evaluable patients had t-NEPC signature

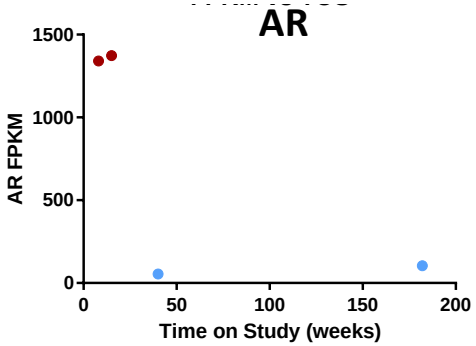


Higher expression of BRD4, E2F1, and lower AR activity was associated with longer time on study









ARSI = AR signaling inhibitor, t-NEPC = treatment-induced neuroendocrine prostate cancer

Aggarwal et al. 2018, Abida et al. 2019, Aggarwal et al. PCF2019, Kim et al. 2021

Mechanisms of resistance to ADT and ARSI in prostate cancer

Prostate Cancer (HSPC, CRPC)

ADT/ARSI THERAPY

Maintenance of AR signaling (80%)

- AR amplification (enhancer) + mutations
- AR splice variant (AR-V7)
- Upregulation of alternative steroid receptor (GR)

AR repression (20%)

- AR-independence (low AR, AR null)
- Transdifferentiation + neuroendocrine markers (t-SCNC, t-NEPC)
- Activation of alternate proliferation pathways (BRD4/E2F1-dependent)

- **Recent approval of ARSIs in earlier disease setting (HSPC) is associated with increased cases of AR-repressed CRPC**
- **Patients with loss of AR activity have a worse prognosis on ARSI and fewer treatment options**

ARSI = Androgen Receptor Signaling Inhibitor; CRPC = castration-resistant prostate cancer; GR = Glucocorticoid Receptor, HSPC = hormone sensitive prostate cancer

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- **Activation of alternate proliferation pathways (BRD4/E2F1-dependent)**

Increased ZEN-3694 + ENZA activity

- **Recent approval of ARSIs in earlier disease setting (HSPC) is associated with increased cases of AR-repressed CRPC**
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Mechanisms of resistance to ADT and ARSI

Prostate Cancer (HSPC, CRPC)

Mainte
- AR amplific
- AR splice v
- Upregulati

-NEPC)
F1-dependent)

- **Recent a**
- **Patients**

essed CRPC

How to enrich for patients with AR-independent prostate cancer (HSPC, CRPC)?

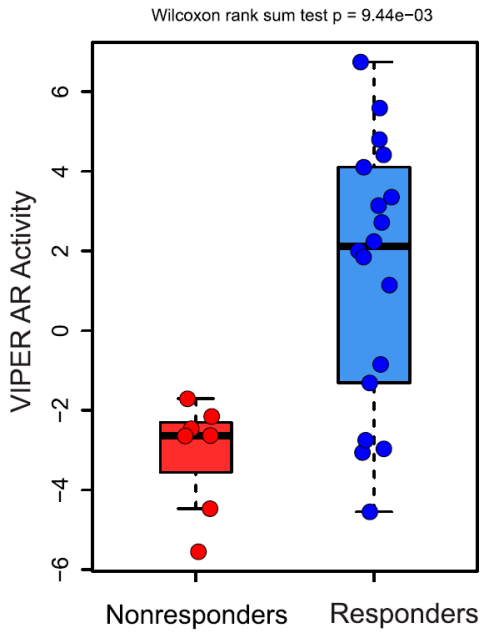
Biopsies:
- Hard to get (bone)
- Archival biopsies might not be reliable (esp. before prior ARSI)
- What is the best signature(s)/score cut-off?
- How to implement in the real world?

→**Clinical history readout to enrich for AR-independence**

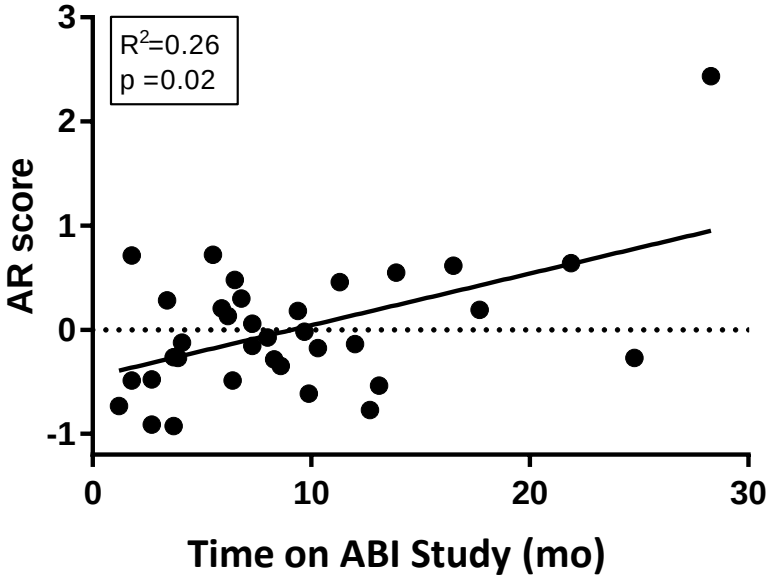
ARSI = Androgen Receptor Signaling Inhibitor; CRPC = castration-resistant prostate cancer; GR = Glucocorticoid Receptor, HSPC = hormone sensitive prostate cancer

Low AR signaling associated with shorter time (primary resistance) on ARSI in patients with mCRPC

Low AR activity in CRPC tumors associated with shorter time on ENZA



Low AR activity in CRPC tumors associated with shorter time on ABI



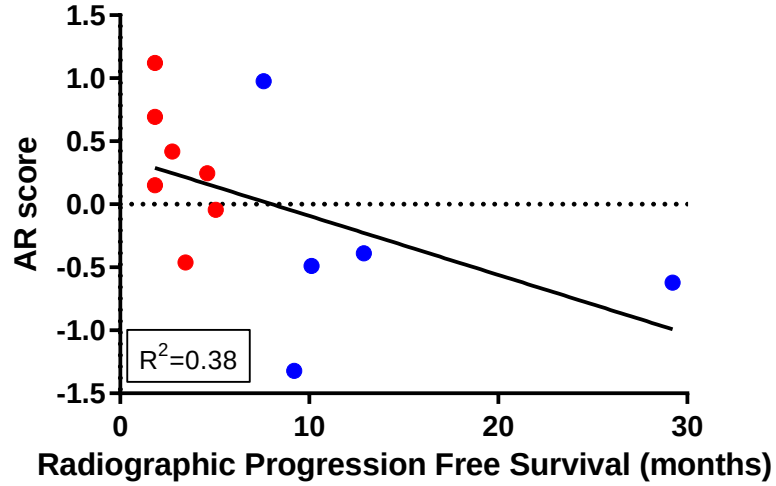
Low AR activity associated with rapid progression (primary resistance) on ARSI

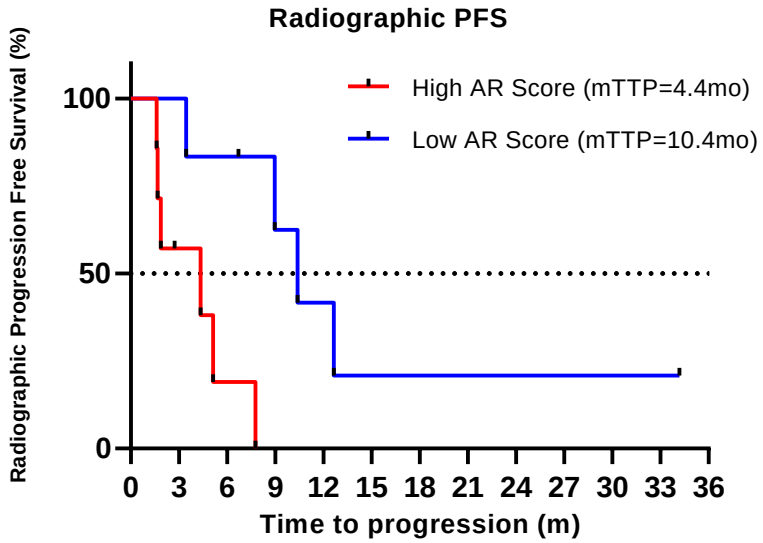
ABI data calculated from Abida et al. 2020, ENZA data from Alumkal et al. 2020

Low AR signaling and primary ABI resistance associated with longer time on ZEN-3694 + ENZA in patients with mCRPC

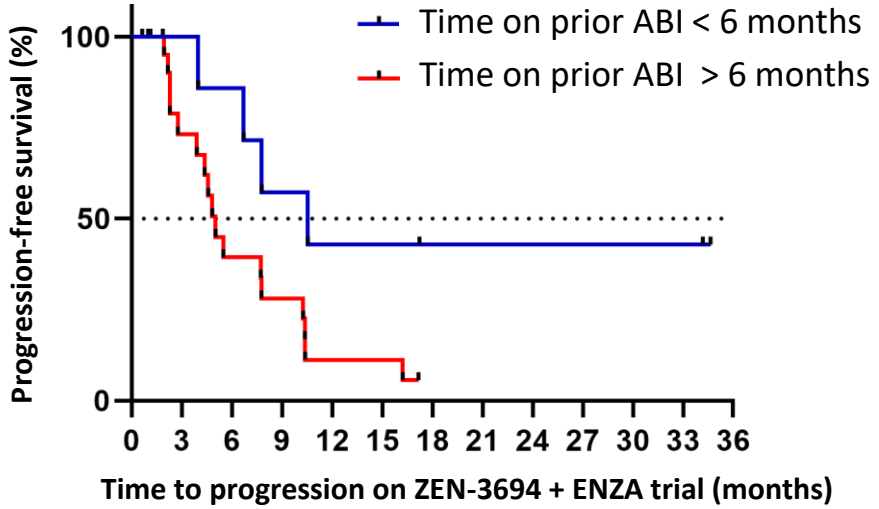
ZENITH EPIGENETICS

Low AR activity in baseline biopsies associated with longer time on ZEN-3694 + ENZA





Patients with prior primary resistance to ABI associated with longer time on ZEN-3694 + ENZA



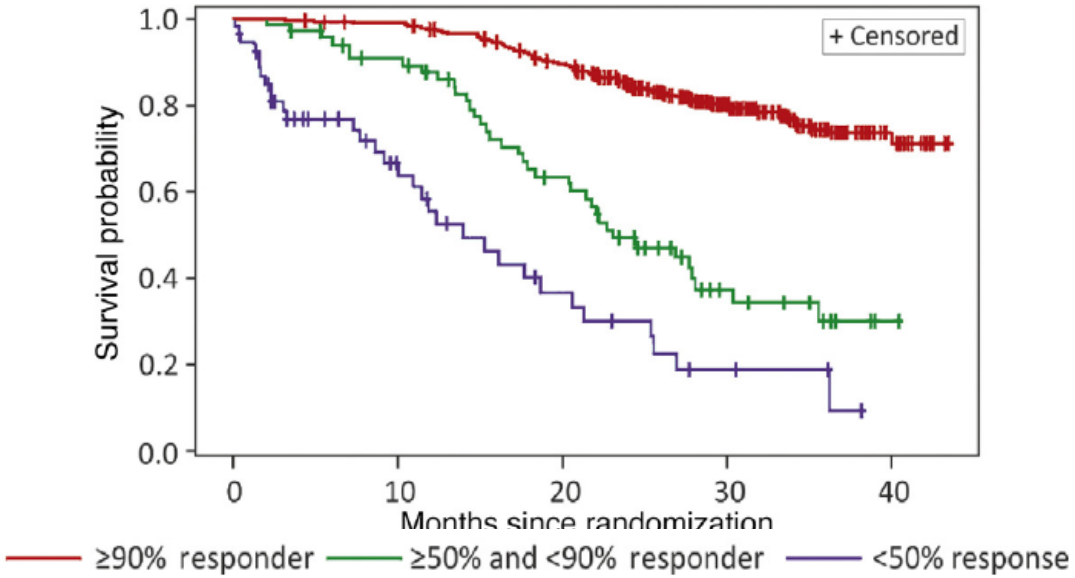
	ABI < 6 mo (n=7)	ABJ > 6 mos (n=25)
Number of events	4	17
Median PFS (months)	**11**	**5**

Low AR activity and rapid progression on prior ABI associated with longer time on ZEN-3694 + ENZA study

Poor PSA responses associated with lower survival in mHSPC and mCRPC

Latitude Phase 3 trial (mHSPC), Sequencing ABI and ENZA trial (mCRPC)

Lack of PSA50 response with ABI is associated with lower survival of patients with mHSPC



mCRPC patients with poor response to 1st ARSi have a worse response to a 2nd ARSi

	Time to confirmed PSA progression on 1st ARSI		HR (95% CI), p-value
	≥ 3 mo	< 3 mo	
% of patients with PSA30 response on 2nd ARSI	**40%** (21/53)	**19%** (3/16)	**2.92** (1.5-5.9), **p=0.003**

Failure to reach PSA ≤ 0.1 ng/ml nadir with ABI is associated with more rapid progression and lower survival

	PSA ≤ 0.1 ng/mL ≤ 6 months (n=239)	PSA > 0.1 ng/mL ≤ 6 months (n=358)
Median rPFS, months (range)	**NE (35.2, NE)**	**25.8 (21.9, 29.6)**
Median OS, months (range)	**NE (NE, NE)**	**42.0 (34.8, 48.8)**

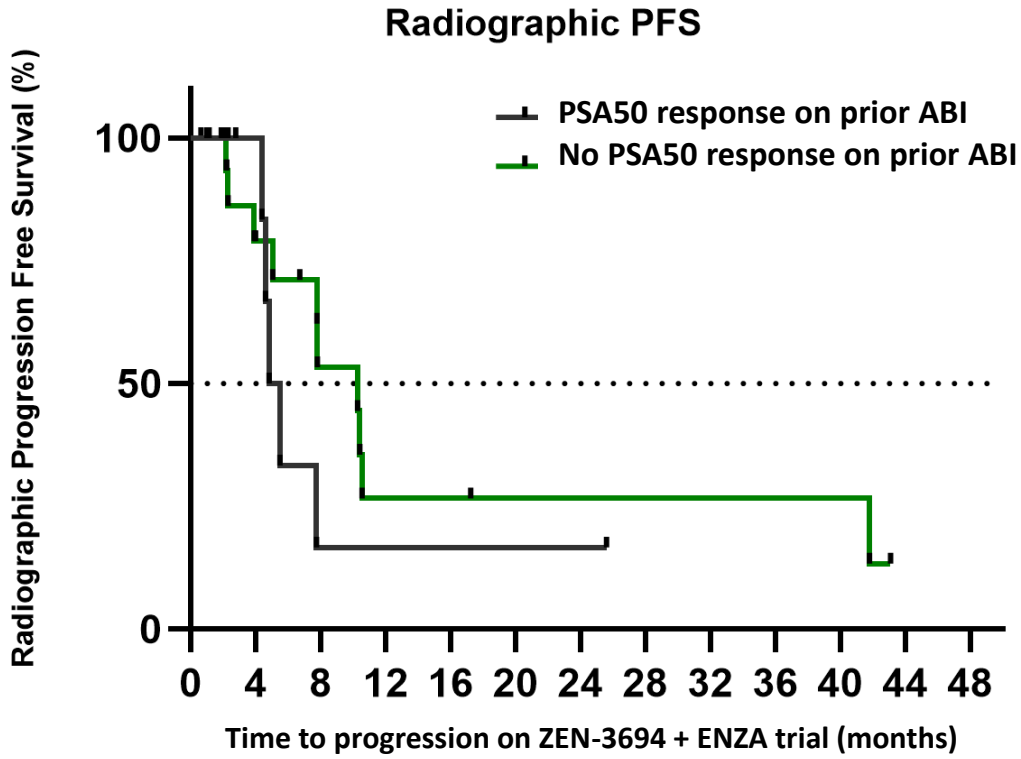
Poor PSA response to ARSI is associated with:

- Rapid progression in both mHSPC and mCRPC
- Poor response to 2nd ARSI

Matsubara et al. 2020, Khalaf et al. 2019

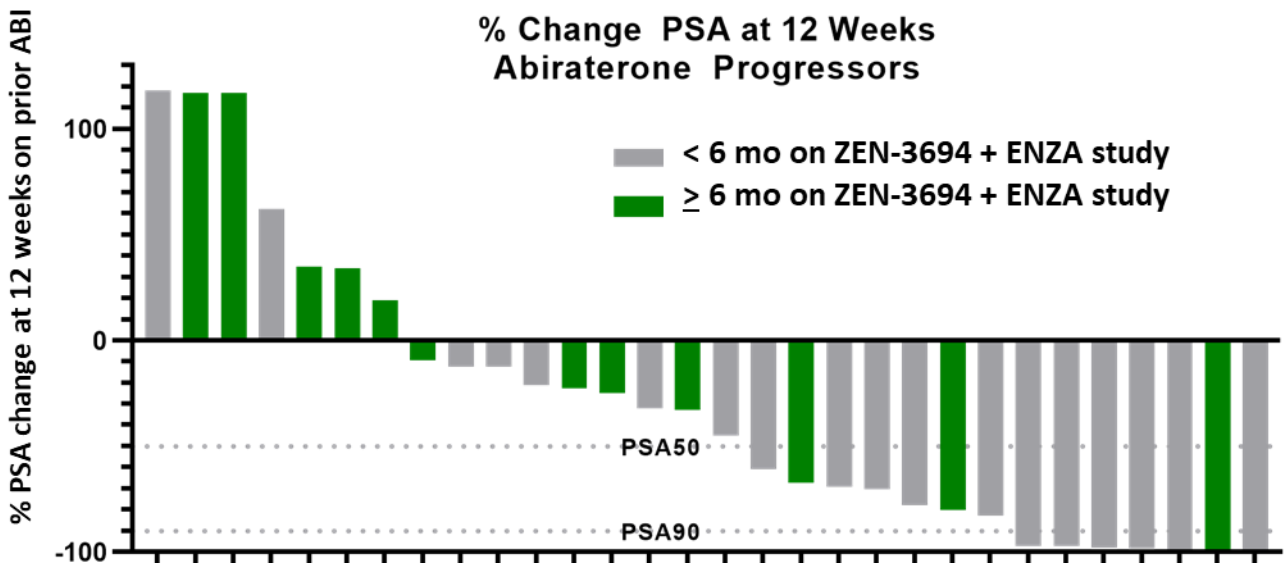
Poor PSA50 response on prior ABI associated with longer time on ZEN-3694 + ENZA study

Lack of PSA50 response with prior ABI is associated with longer time on ZEN + ENZA



	PSA50 response on prior ABI (n=14)	No PSA50 response on prior ABI (n=16)
Number of events	5	10
Median rPFS (months)	5.2	10.3

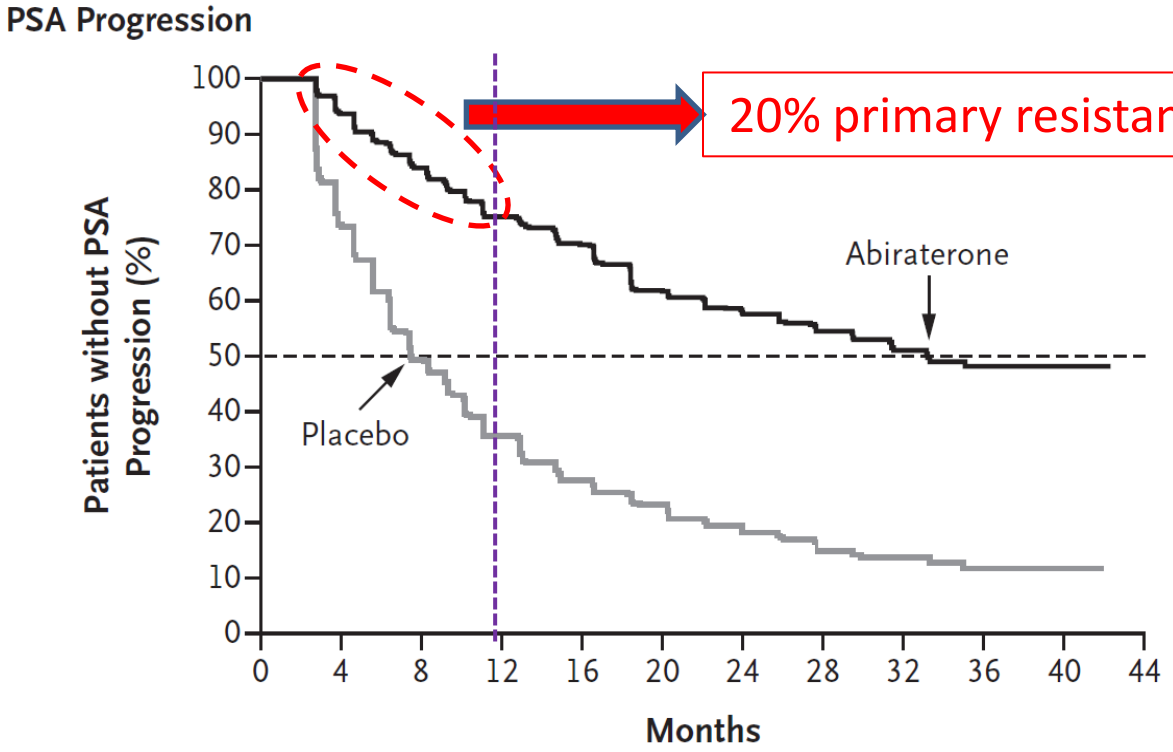
Lack of PSA50 response with prior ABI is associated with longer time on ZEN + ENZA



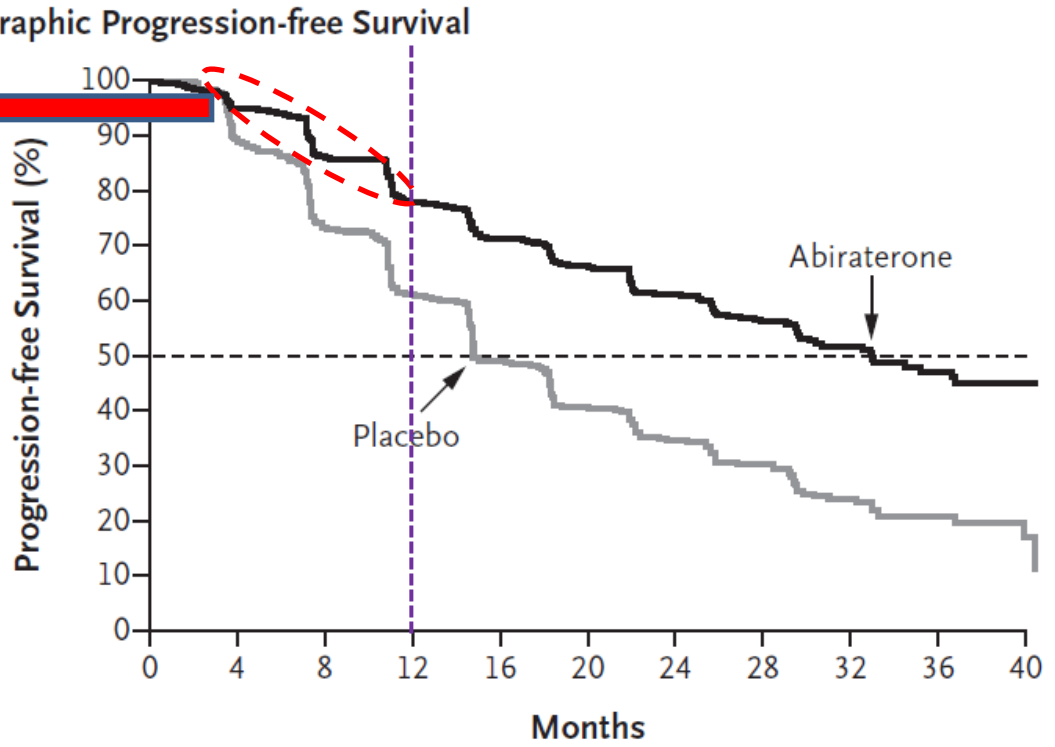
3/14 patients with PSA50 to prior ABI had rPFS> 6 mo
9/15 patients without PSA50 to prior ABI had rPFS>6mo

Prior poor PSA response on prior ABI associated with longer time on ZEN-3694 + ENZA study

~ 20% of mHSPC patients progress in less than 12 mo. on ABI (primary resistance) (LATITUDE trial)



20% primary resistance to ABI



No. at Risk												
Abiraterone	597	520	447	379	340	285	227	162	95	48	18	0
Placebo	602	393	250	172	129	102	65	33	19	8	5	0

No. at Risk												
Abiraterone	597	533	464	400	353	316	251	177	102	51	21	
Placebo	602	488	367	289	214	168	127	81	41	17	7	

- **Primary resistance to ABI in either HSPC or CRPC is predicted to enrich for AR-independence**

⇒ **Enrichment for patients with predicted poor response to 2nd ARSI with fewer therapy options**

Fizazi et al. 2017

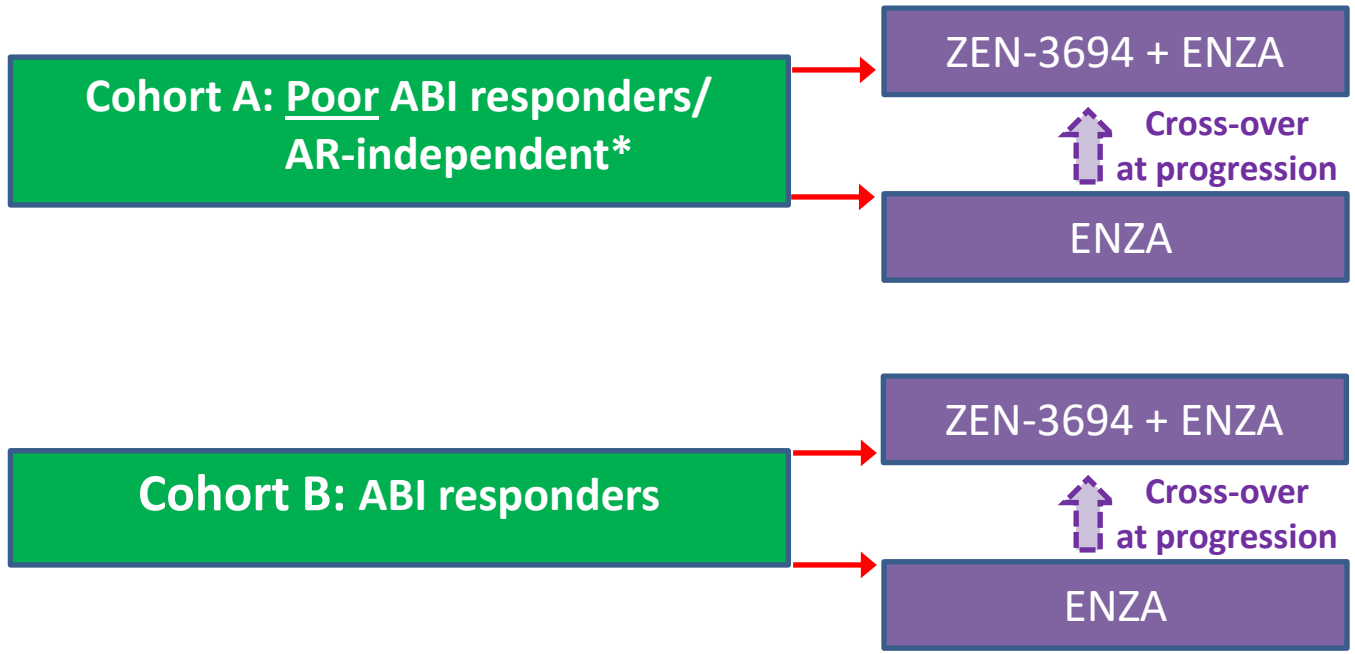
Phase 2b mCRPC study design: Pre-select patients with poor response to prior ABI (AR-independent/BET-dependent) Scheduled start in August 2021

Objectives:

- Test ZEN-3694 + ENZA in mCRPC patients that have progressed on ABI
- Evaluate efficacy in both poor ABI responders/AR-independent and ABI responders
- Open label, randomized, Blinded Independent Central Review (BICR)

Key Eligibility Criterion

- mCRPC progressed on ABI
- Not candidates for chemotherapy (Physician judgment)
- Patients with prior enzalutamide/apalutamide /darolutamide excluded



Primary Endpoint

- rPFS Cohort A

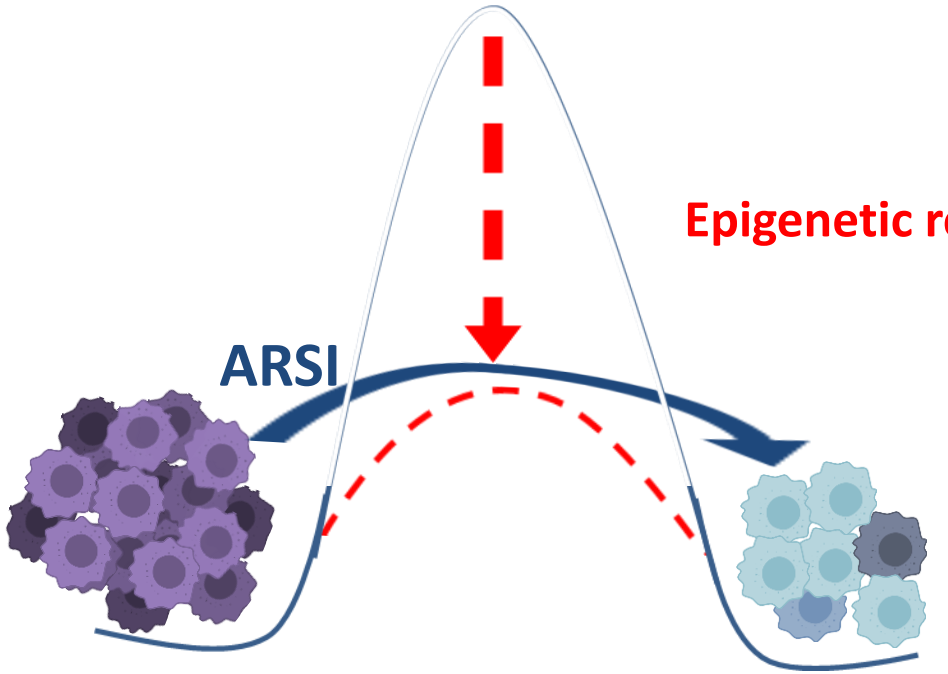
Key Secondary Endpoints

- rPFS Cohort A+B
- PFS Cohort A
- PFS Cohort A+B
- OS: Cohort A

Collaboration with Astellas and Newsoara

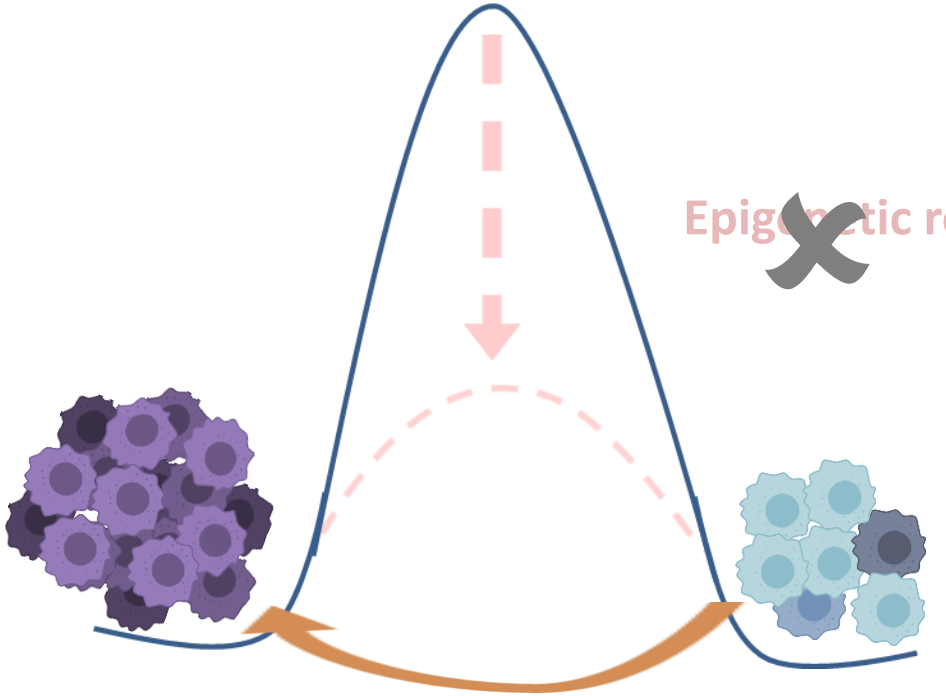
***HSPC: < 12 months duration on prior ABI, or failure to achieve a PSA nadir of 0.2 ng/ml**
CRPC: < 6 months duration on ABI, or failure to achieve PSA50 response

Epigenetic modulation by ZEN-3694 restores sensitivity to enzalutamide



- ARSI induces loss of AR signaling
- Gain of AR-independent features
- BET-dependent transcriptional reprogramming

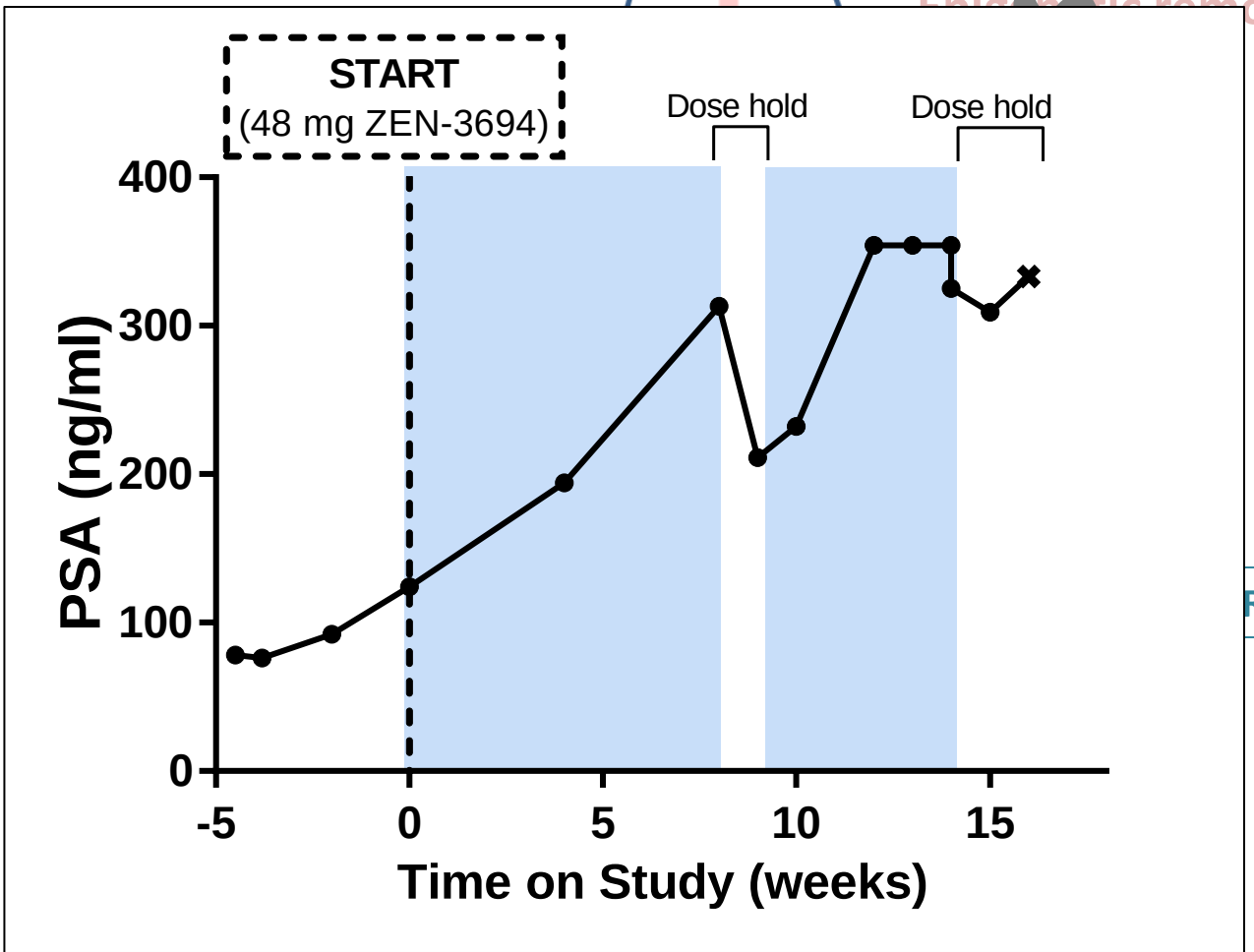
Epigenetic modulation by ZEN-3694 restores sensitivity to enzalutamide



- ZEN-3694 inhibits maintenance of AR-independence
- Restoration of ARSI sensitivity

Epigenetic modulation by ZEN-3694 restores sensitivity to enzalutamide

Single Agent ZEN-3694

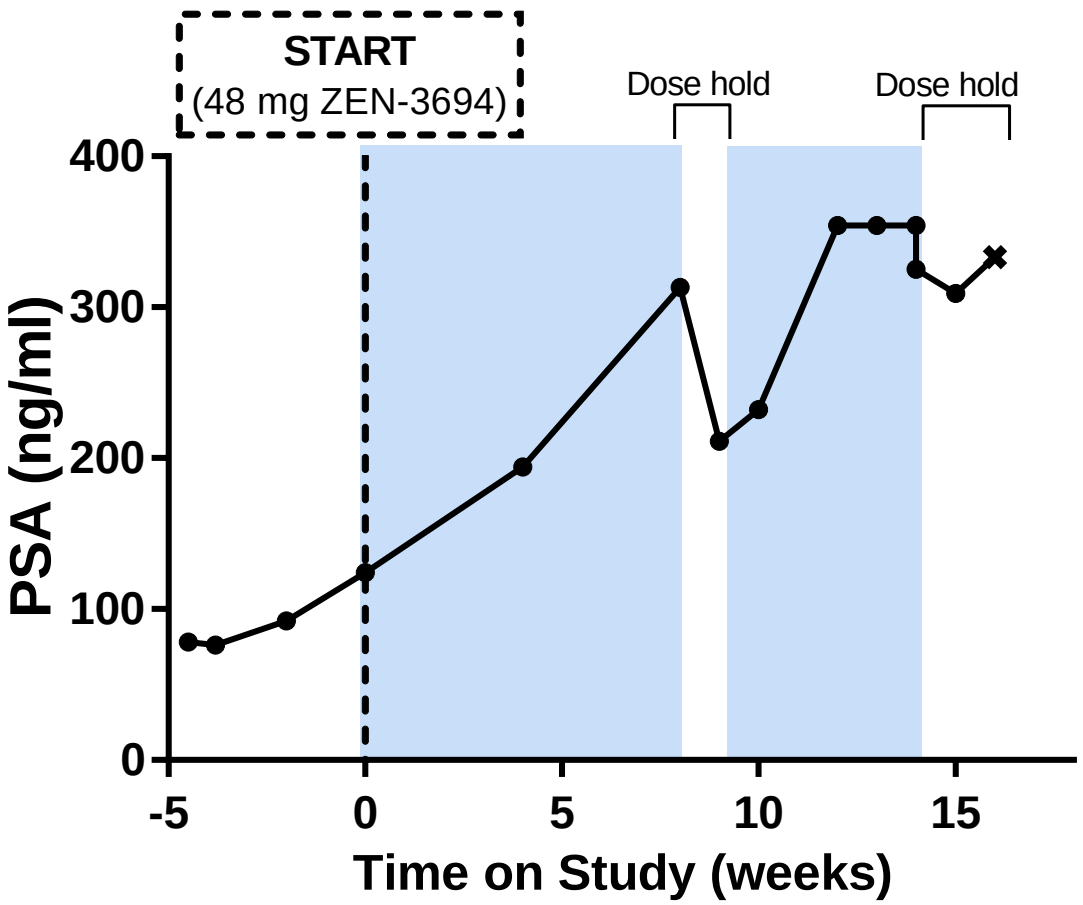


- ZEN-3694 inhibits maintenance of AR-independence
- Restoration of ARSI sensitivity

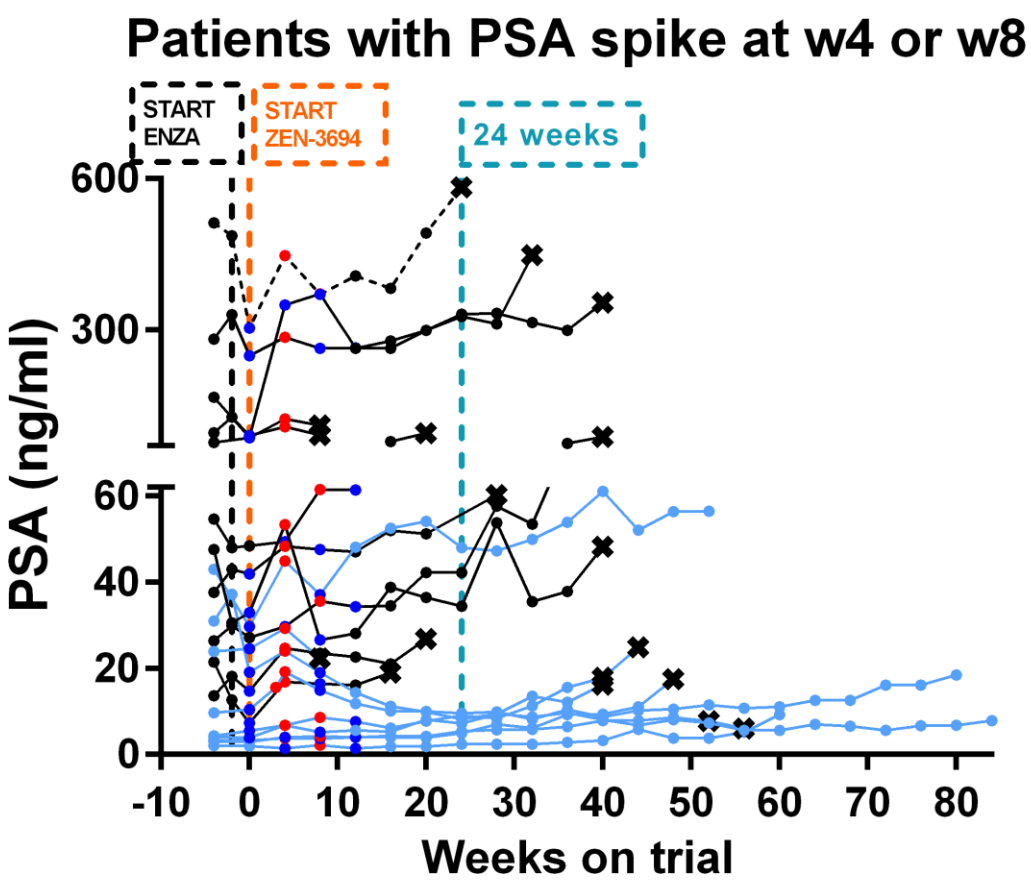
RPC

Epigenetic modulation by ZEN-3694 restores sensitivity to enzalutamide

Single Agent ZEN-3694



Combination ZEN-3694 + ENZA



A Phase 1b/2 Study of ZEN003694 and Talazoparib in Patients With Triple Negative Breast Cancer (TNBC) and Without Germline BRCA1/2 Mutations

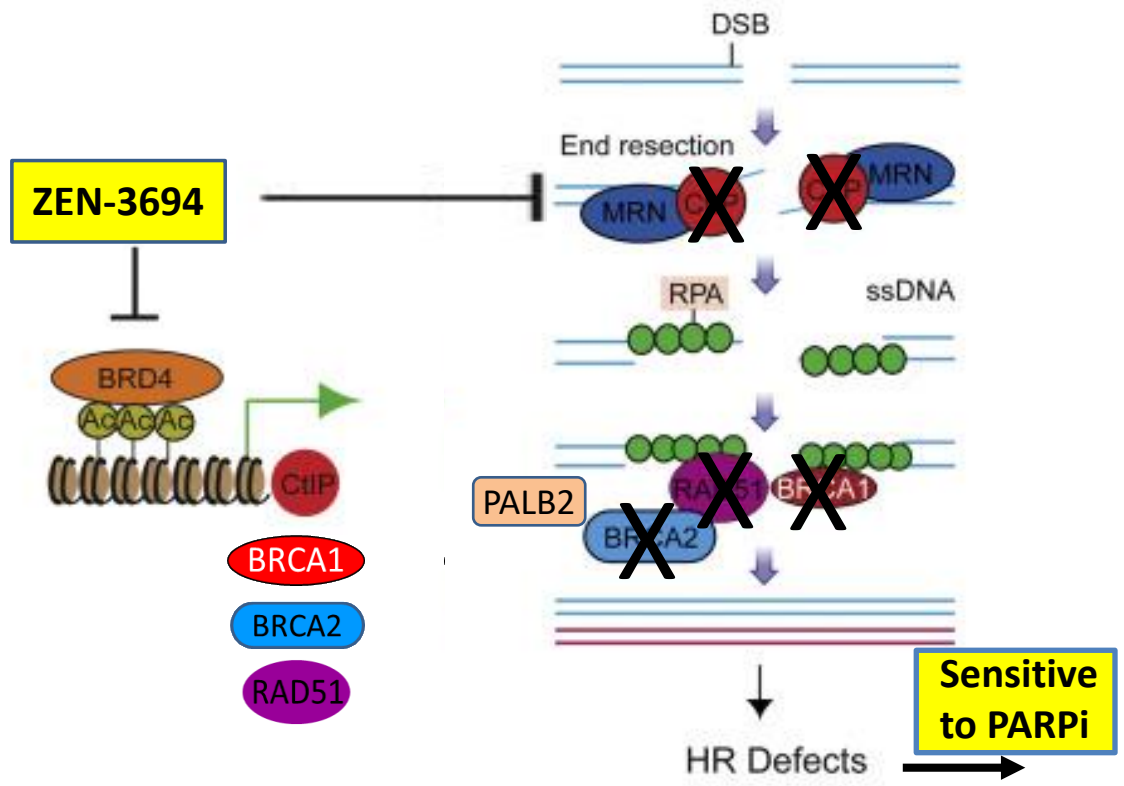
Aftimos et al. SABCS 2020 (PS11-10)

Induction of homologous recombination deficiency by ZEN-3694 and sensitization to PARP inhibitors in BRCAwt cells

- In breast cancer, only ~20% of patients are eligible to receive a PARPi (germline BRCA1/2 mutant)
- Additional clinical activity in advanced breast cancer is currently limited to somatic BRCA1/2 or germline PALB2 mutations, not in other DNA repair genes
- Acquired resistance limits the clinical activity of PARPi (recovery of DNA repair capacity)
- ZEN-3694 reduces the mRNA levels of several DNA repair genes as a potential mechanism of sensitization to PARPi

⇒ BRCAwt tumors

⇒ BRCA1/2 mutant tumors PARPi-resistant



Adapted from Sun et al. 2018

ZEN-3694 + talazoparib trial design (Phase 2, Pfizer/Zenith collaboration)

Patients with advanced TNBC and no germline BRCA1/2 mutations

ZENITH EPIGENETICS

Dose Escalation
Patients with at least one prior cytotoxic chemotherapy

⇨

Simon 2-Stage Dose Expansion
≤ 2 prior chemotherapy regimens for mTNBC

Locally advanced/metastatic TNBC

- **No germline mutations in BRCA1 and BRCA2 (gBRCA1/2m) (CLIA test)**
- No prior progression during platinum treatment
- No prior exposure to BETi or PARPi

Objectives: Show safety and activity of ZEN-3694 + talazoparib
Identify potential biomarkers of response

Design: Dose escalation followed by Simon 2-stage, n= 17 1st stage, n=20 2nd stage

Patient population: TNBC: locally advanced or metastatic

Endpoints: Part 1: Safety, pharmacokinetics/pharmacodynamics, maximum tolerated dose, Phase 2 dose (RP2D)
Part 2: Objective response rate (ORR), clinical benefit rate (CBR), duration of response (DOR), progression free survival (PFS)

NCT03901469

Common treatment-related adverse events (AEs)

Grade 3/4 AEs across all cohorts	DE Cohort 1 48 mg ZEN + 1.0 mg Tala (n = 6)		DE Cohort 2 48 mg ZEN + 0.75 mg Tala (n = 6)		DE Cohort 3 36 mg ZEN + 1.0 mg Tala (n = 3)		Simon Stage 1 48 mg ZEN + 0.75 mg Tala (n = 17)		Total n = 32	
	Any Grade	Grade 3/4	Any Grade	Grade 3/4	Any Grade	Grade 3/4	Any Grade	Grade 3/4	Any Grade	Grade 3/4
ALT increase^			1				4	2 (G3)	5 (15.6%)	2 (G3)
AST increase^	1		1				3	1 (G3)	5 (15.6%)	1 (G3)
Diarrhea	2	1 (G3)			1		1		4 (12.5%)	1 (G3)
Hyperglycemia	1						1	1 (G3)	2 (6.3%)	1 (G3)
Nausea	3		4	1(G3)			6	1 (G3)	13 (40.6%)	2 (G3)
Neutropenia	1		2	2(G3)			2		5 (15.6%)	2 (G3)
Thrombocytopenia	6	3 (G3), 2 (G4)#	5	3 (G3), 1 (G4)#	1	1 (G3)	5	5 (G3), 1 (G4)	17 (53.1%)	12 (G3), 4 (G4)#

^ALT/AST self resolved

#DLTs (thrombocytopenia) = two patients in Cohort 1, one patient in Cohort 2

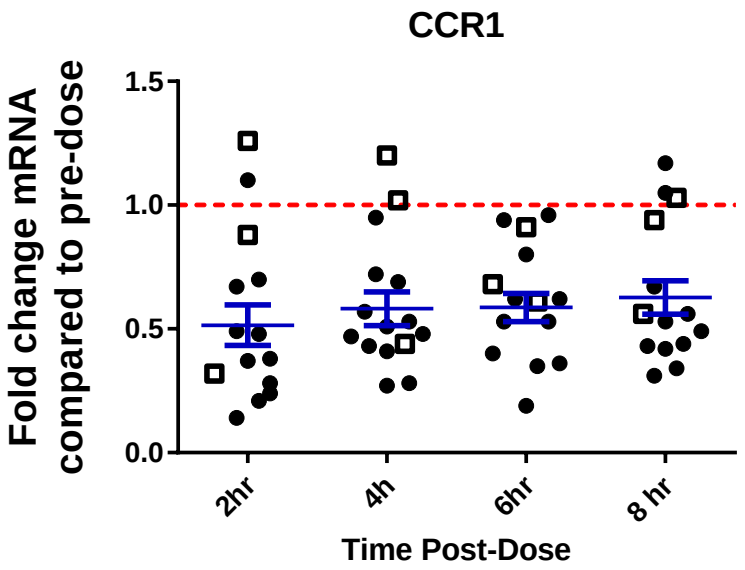
- **48 mg QD ZEN-3694 + 0.75 mg QD talazoparib selected as RP2D**
- **Thrombocytopenia reversible with dose hold and reduction in sensitive patients**

List of Grade 1/2 AEs presented at SABCS2020 and available at https://www.zenithepigenetics.com/Science-Epigenetics/publications-posters

Sustained whole blood target engagement for > 8 hours

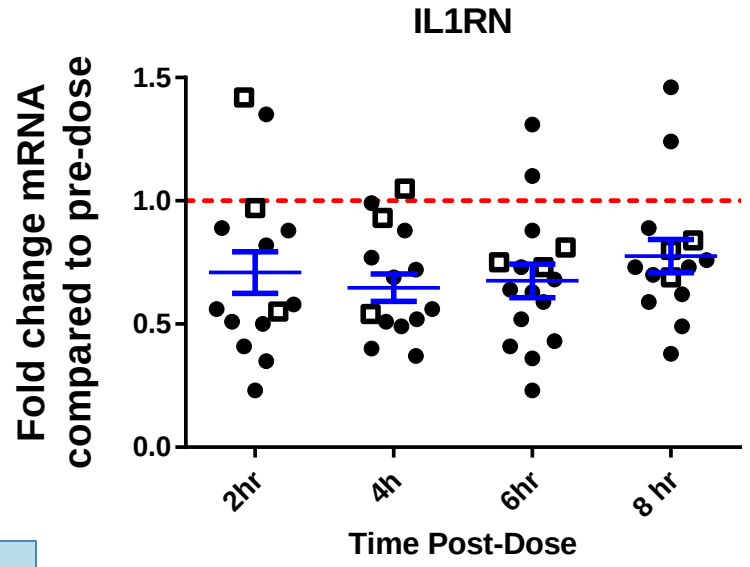
Similar exposure-dependent target engagement as prior trials in prostate cancer

TNBC trial



36 mg ZEN003694

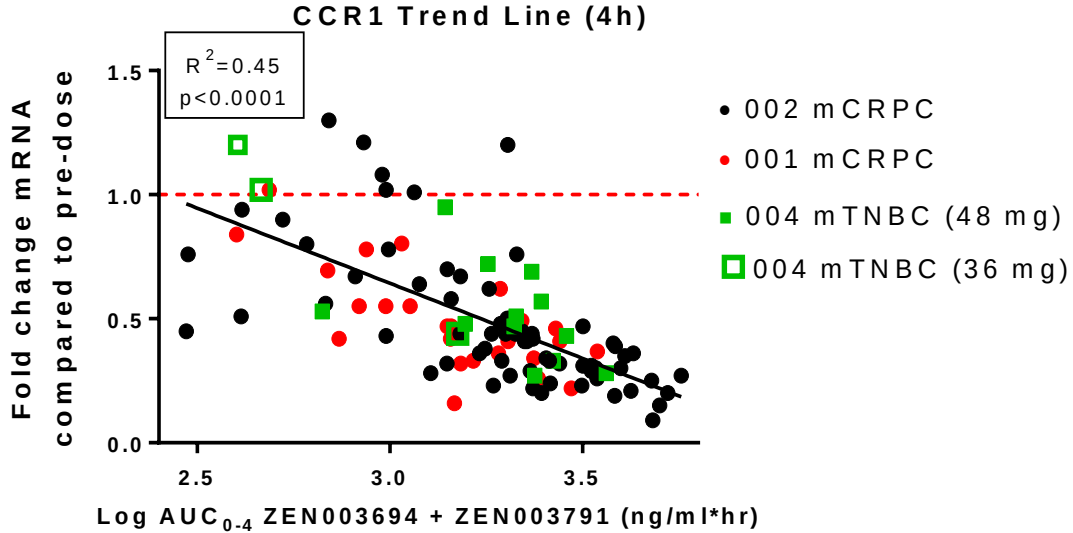
48 mg ZEN003694



36 mg ZEN003694

48 mg ZEN003694

TNBC + CRPC trials

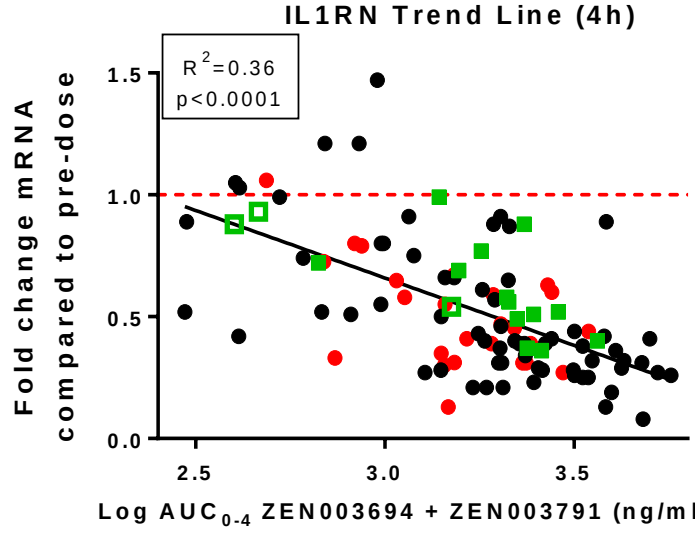


002 mCRPC

001 mCRPC

004 mTNBC (48 mg)

004 mTNBC (36 mg)



002 mCRPC

001 mCRPC

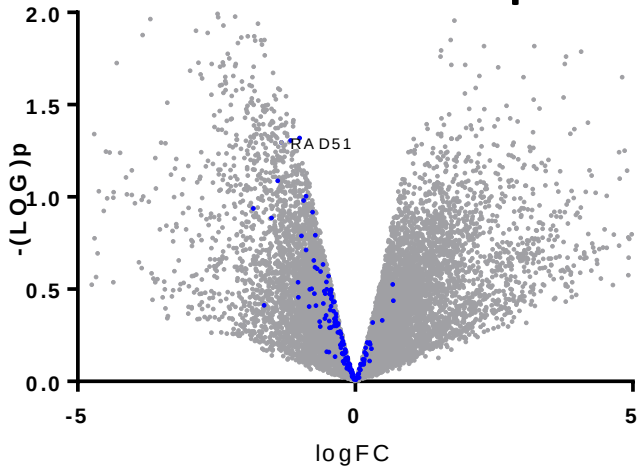
004 mTNBC (48mg)

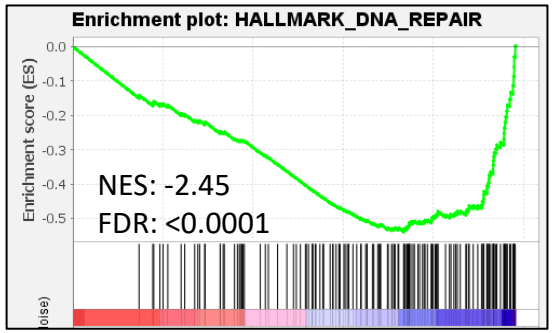
004 mTNBC (36 mg)

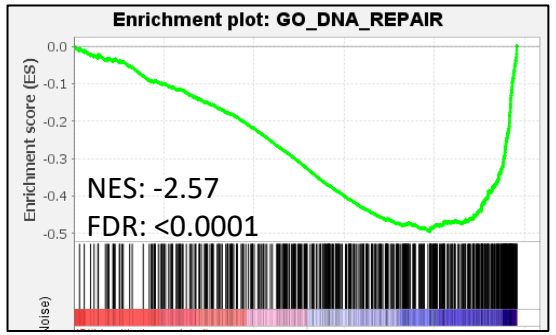
Inhibition of DNA repair and HRR gene expression in tumors from two TNBC patients On-Treatment

Significant inhibition of DNA repair (GSEA) in tumors

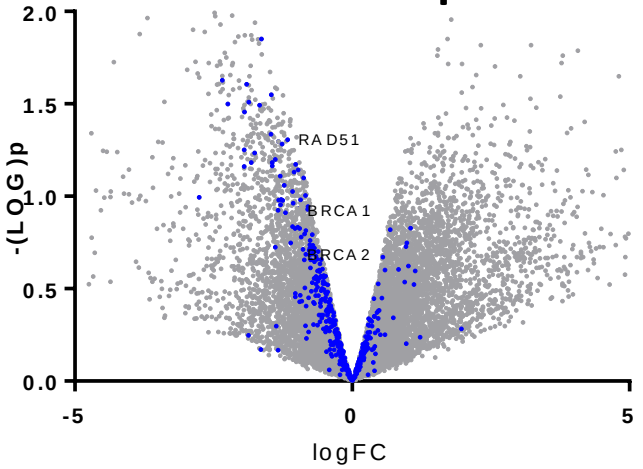
Hallmark DNA repair



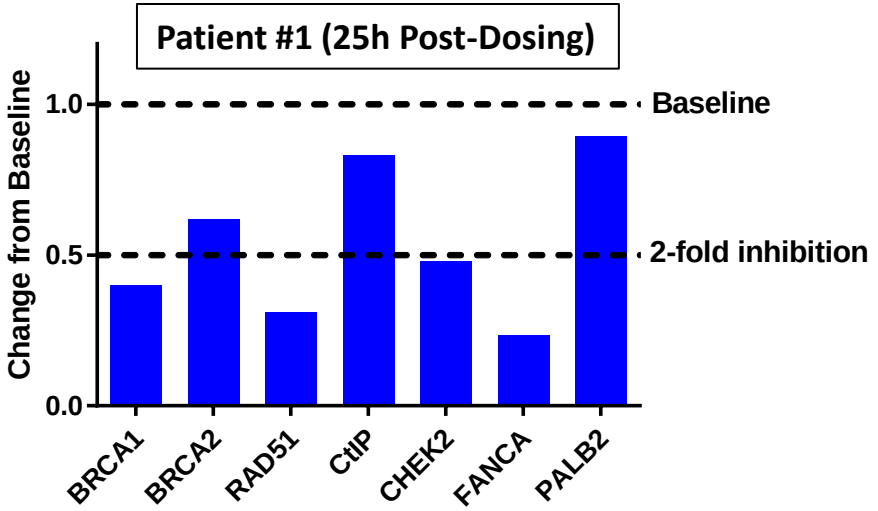


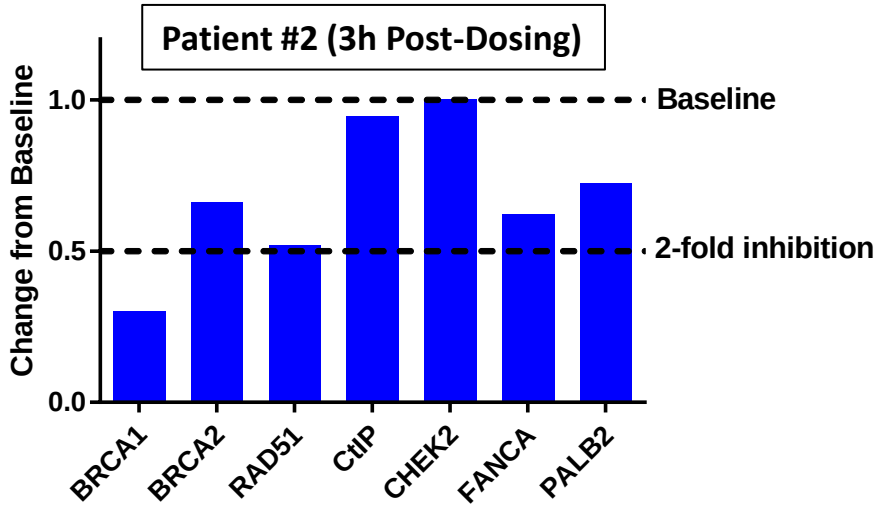


GO DNA repair



Inhibition of HRR gene expression in tumors



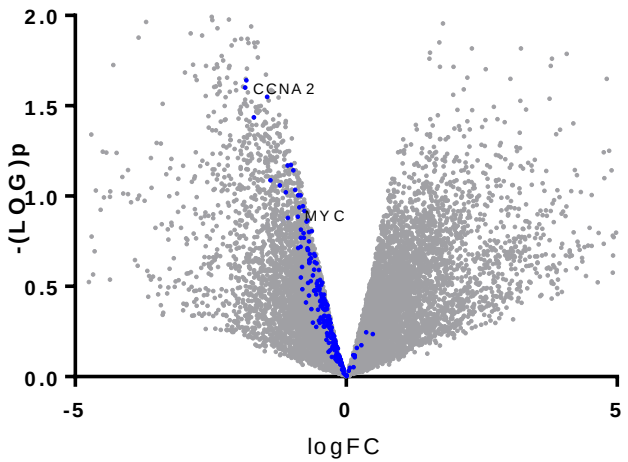


HRR= homologous recombination repair

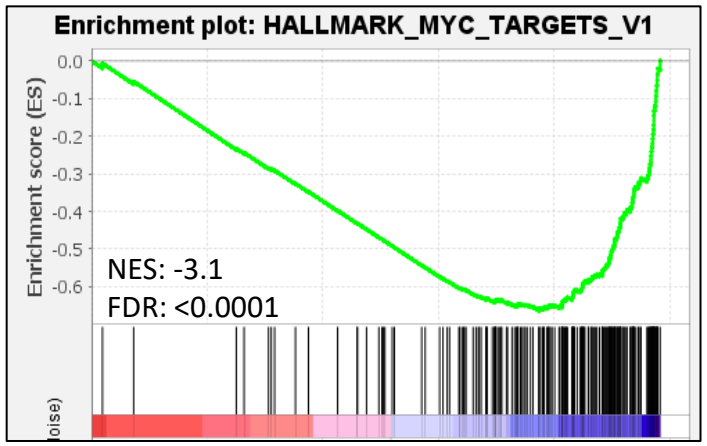
Significant inhibition of oncogenic hallmarks in tumor biopsies On-Treatment (GSEA)

ZENITH EPIGENETICS

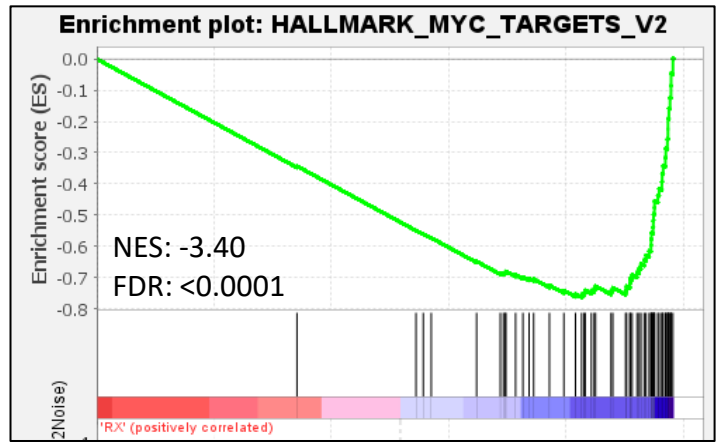
Volcano plot (Hallmark MYC V1)



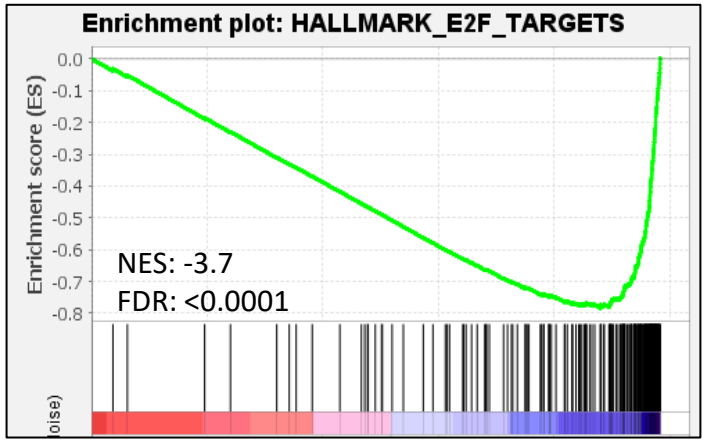
Hallmark MYC V1



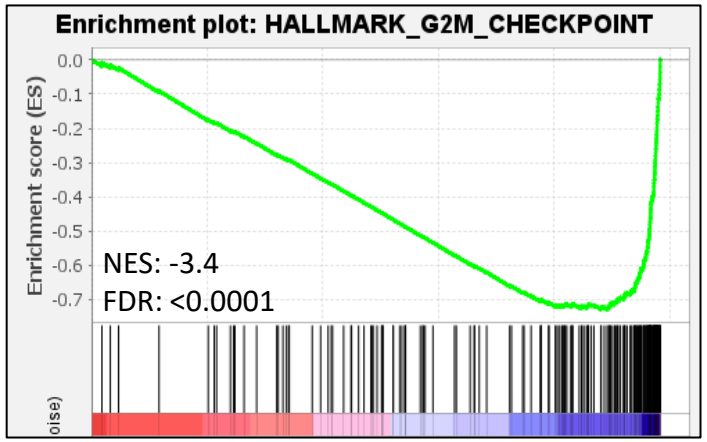
Hallmark MYC V2



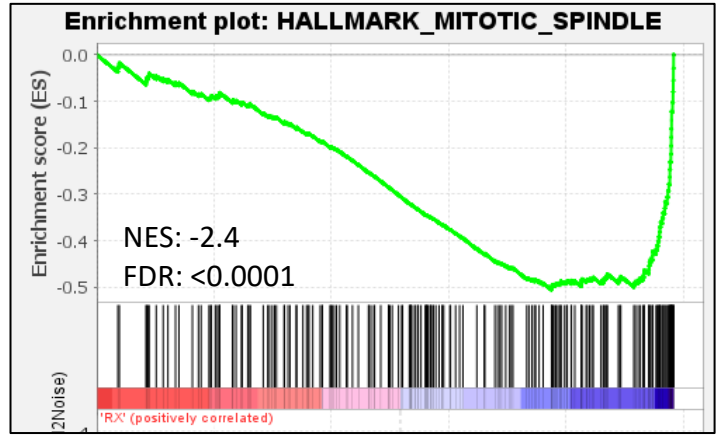
Hallmark E2F targets



Hallmark G2/M checkpoint



Hallmark mitotic spindle

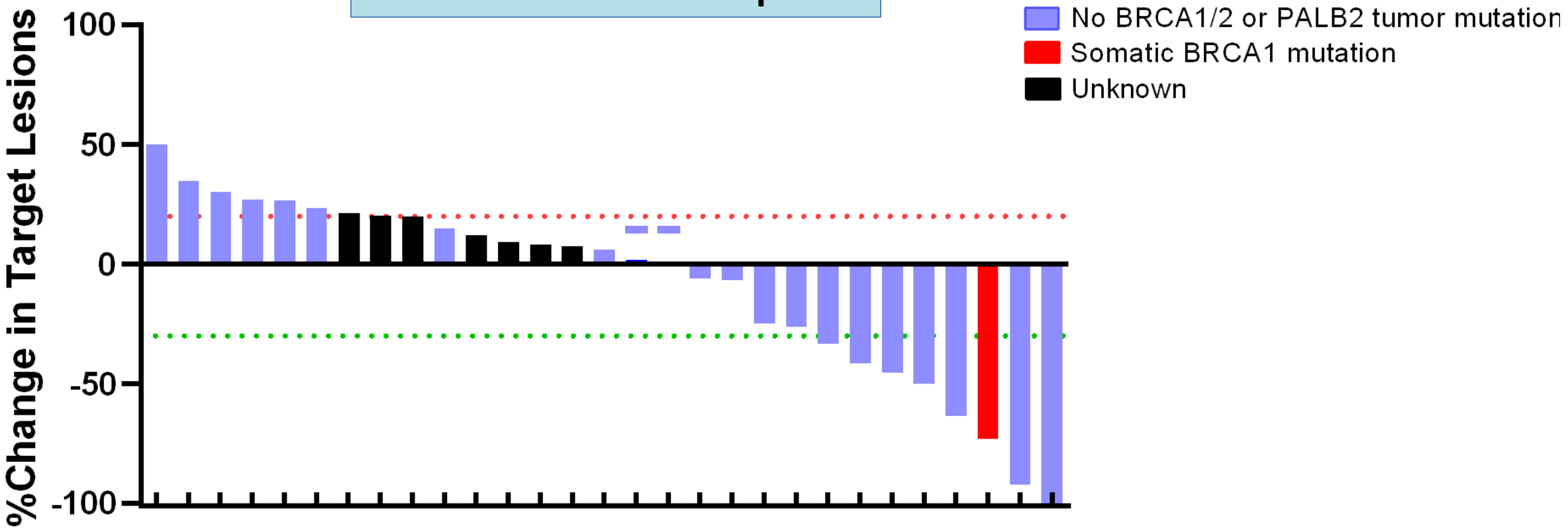


Inhibition of oncogenic hallmarks and perturbation of cell cycle regulation On-Treatment

Activity of ZEN-3694 + talazoparib in HRRwt TNBC tumors

Dose escalation + Stage 1 (December 2020)

Best overall tumor response



- **Patients screened for absence of gBRCA1/2m for enrollment on trial**
- **Sequencing of tumor biopsies from patients to rule out somatic mutations in BRCA1/2 or PALB2**

⇒ **Combination activity unlikely due to single agent talazoparib**

HRR= homologous recombination repair

Clinical activity of PARP inhibitors in advanced breast cancer

Limited activity in **BRCA1/2 wild-type** breast cancer patients

Pathway	Agent(s)	BRCA1/2 and PALB2 status	
		MUTANT	"WT"
ZEN + TALA vs. single agents	ZEN-3694 + TALA		✓
	BETi		✘
	PARPi	✓	✘
	ATRi	✘	✘
DNA damage response	ATRi + PARPi	✓	✘
	ATRi + carboplatin	(✘)	(✘)
	WEE1	(✘)	(✘)
	WEE1 + PARPi	✓ (toxic)	✘
PI3K/AKT/mTOR	AKTi + PARPi	✓	✘
	AKTi + paclitaxel	✘	✘
	panPI3Ki	✘	✘
	PIK3CAi + PARPi	(✘)	(✘)
	mTORi + PARPi	✘	✘
MAPK	EGFRi + PARPi		(✘)
Immunotherapy	αPD-1 + PARPi	✓	(✘)

✓= evidence of clinical activity
✘= limited clinical activity in unselected patient population or compared to single agent
(✓) or (✘) = initial clinical evidence (currently low number of TNBC cases)

Initial clinical results (advanced breast cancer):

- Limited activity of PARPi outside BRCA1/2m or PALB2m

⇒ ~ 5-10% tumor response rates in unselected populations
⇒ Need to identify additional biomarkers of response

- Potential to increase and extend current PARPi activity

⇒ Increase response rates and/or duration of response?
⇒ Promising strategy

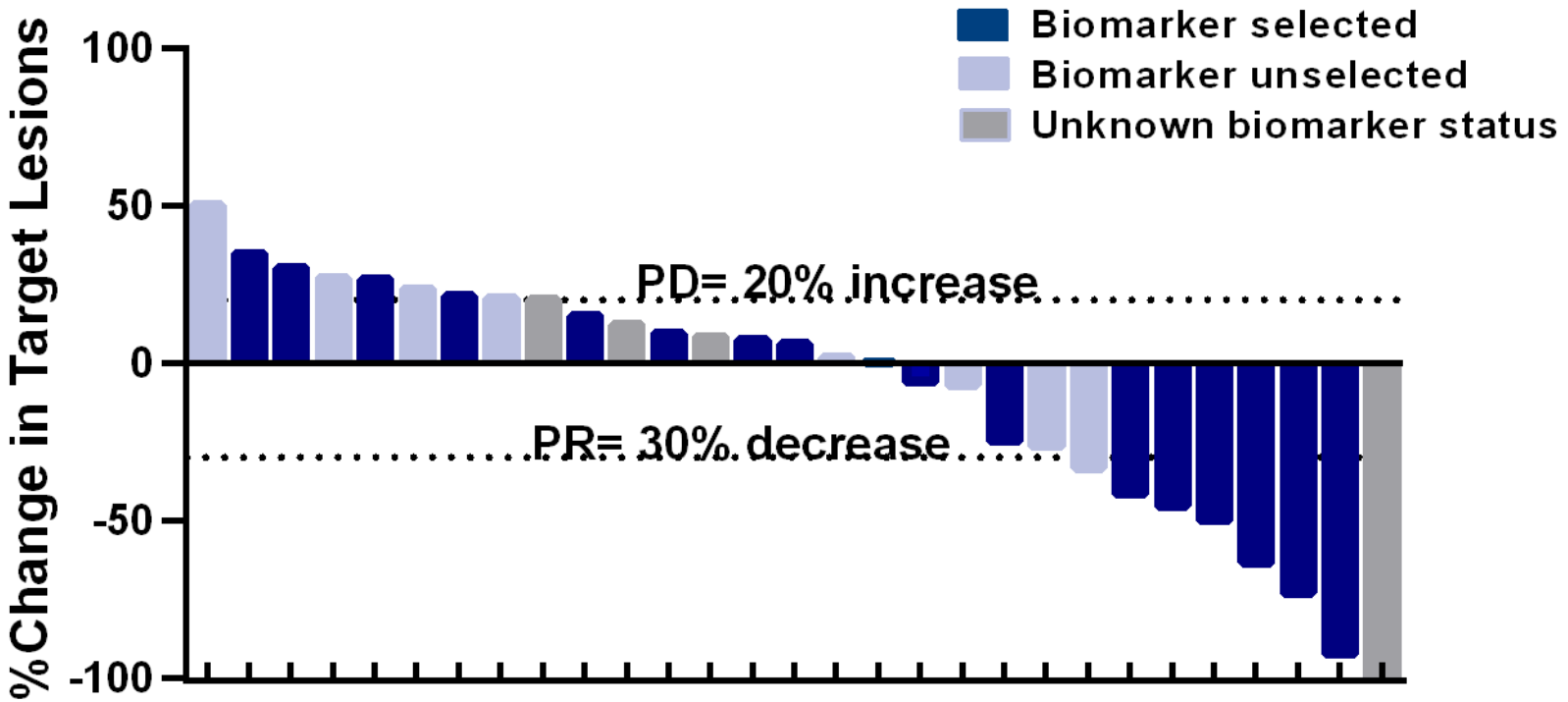
- Most agents currently tested did not sensitize to PARPi

⇒ Limited evidence of creation of "BRCAness" phenotype in the clinic

Tung 2020, Gruber 2019, Patsouris 2020, Stringer-Reasor 2021, Yap 2020, Krebs 2020, Westin 2018, Cousins 2020, Hamilton 2019, Vinayak 2019, Konstantinopoulos 2019, Naqash 2020, Garrido-Castro 2020, Domchek 2020, Domchek 2021

Biomarker identification in the ZEN-3694 + talazoparib trial

Preliminary retrospective results suggest patient enrichment strategy



	All patients (N=31)	Biomarker unselected (N=8)	Biomarker selected (N=19)	Trodelvy (FDA approved)
ORR	27%	13%	**33%**	35%
CBR (≥ 6 mo)	32%	13%	**47%**	45%

ORR = overall response rate (complete + partial tumor responses, confirmed and unconfirmed)
CBR = clinical benefit rate (ORR + stable disease for ≥ 6 months)

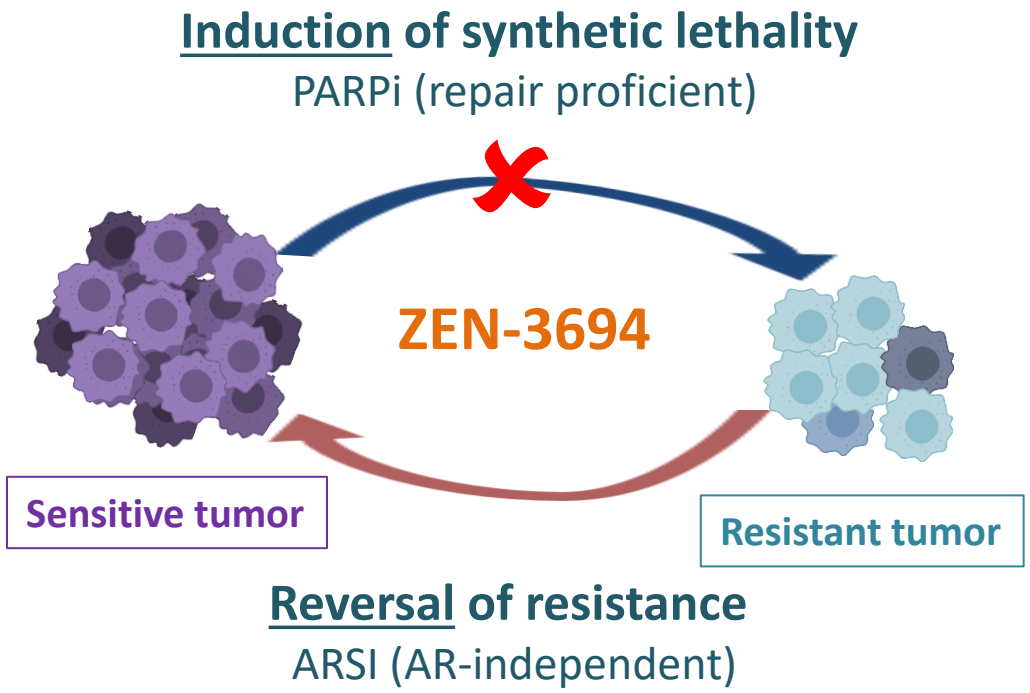
Summary and conclusions: TNBC study

- Combination of ZEN-3694 + TALA demonstrated evidence of anti-tumor activity in previously treated patients with metastatic TNBC without gBRCA1/2 mutations.
- The combination is generally well-tolerated. Thrombocytopenia is the most common adverse event and dose-limiting toxicity, but it is manageable with dose adjustments. High dose intensity was maintained.
- PK is predictable, and PD data show meaningful and durable target engagement.
- Evidence that ZEN-3694 can induce synthetic lethality in combination with PARP inhibitors
- ZEN-3694 + talazoparib Simon Stage 2 is fully enrolled
- Translational Program to prospectively test identified biomarkers involved in response to combination regimen ongoing

ZEN-3694 can sensitize BRCA1/2 wild-type TNBC tumors to PARP inhibitors

Use of ZEN-3694 to prevent and reverse drug resistance

Tackling epigenetic-based drug resistance using epigenetic inhibitors



Common themes

Requirement of the combination agent

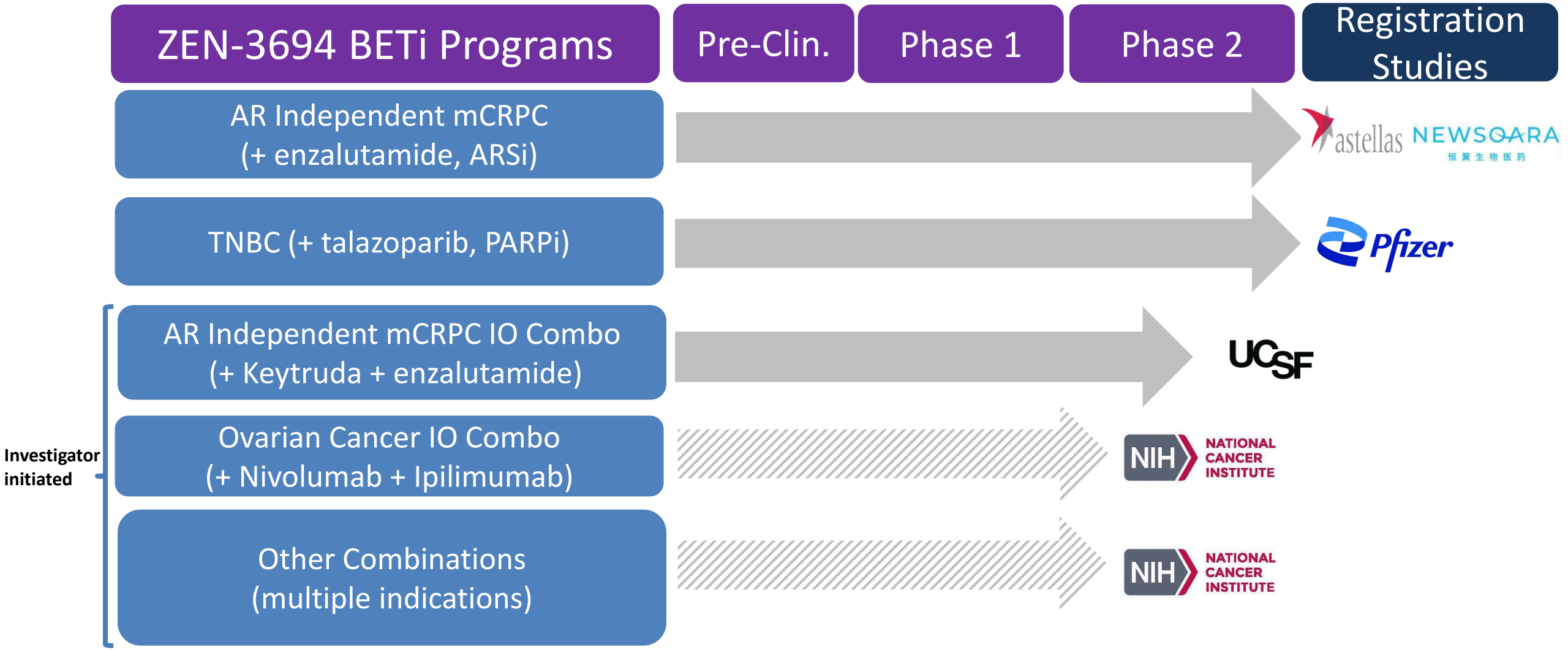
⇒ Induce DNA damage (PARPi)

⇒ Kill re-sensitized tumor cells (ARSI)

Early identification of biomarkers of response

- Additional BETi-based combinations with immunotherapies in clinical development
- Optimal length of target engagement (hours vs. days)? Epigenotype specific?
- Post-BETi? EZH2, LSD1, HDAC, CBP/P300, PRMT inhibitors?

Zenith advancing pipeline with strong collaborators

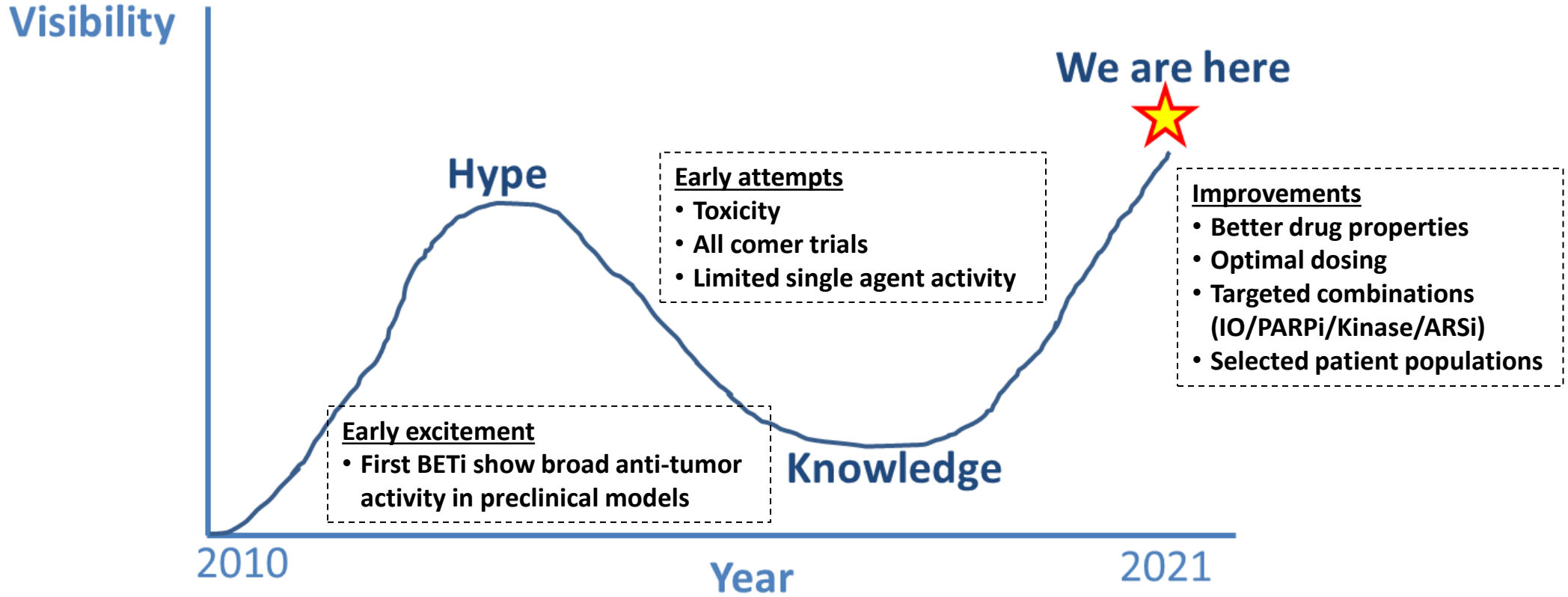


- **Collaboration with the National Cancer Institute Cancer Therapy Evaluation Program (NCI-CTEP)**
- **Leverage knowledge gained from prostate and breast cancer trials**

10 years of BET inhibitor development in oncology indications

ZENITH EPIGENETICS

2010	2012	2015	2021
First BETi published	BETi enter the clinic	20 BETi in clinical trials	5 BETi in clinical trials
• First BETi (benzodiazepines) • Broad activity in cell lines and animal models	• Potent, long half life molecules ("kinase inhibitor approach") • **Biology of epigenetic readers** • Single agent approach	• CYP liabilities, off-target toxicities • Dosing near DLT, requiring dose holds and intermittent schedules • Limited efficacy due to epigenetics biology	**Combination-based approach** • Hematological cancers, myelofibrosis, and solid tumors (Ph. 2/3) • Combinations target BET-dependent mechanisms



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Principal Investigators CRPC Trial

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- Adam Foye (UCSF)
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- Eva Corey (U. Washington)
- Moon Chung (U. Washington)
- Colin Pritchard (U. Washington)
- Eric Small (UCSF)
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