Corporate Update – September 12, 2018
Annual Meeting of Shareholders

Link to webcast archive: http://services.choruscall.ca/links/zenithagm20180912.html

Todays Agenda for Zenith Capital Corp.

1. Corporate Profile
2. Epigenetic Mechanism Review
3. Prostate Cancer Rationale Review
4. Phase 1 Details & Early Results
5. Enzalutamide Combination Trial – Phase 2a
6. Next Steps

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Share Structure Profile

Founded	Corporate spin out from Resverlogix in June 2013
Status	Private company, full reporting issuer
Cash Raised 2014-2018	US$50MM @ US$1.00 & US$2.00 per share (all pre-clinical results based)
Enterprise Value est.	US$325MM (US$2.50 per share) est.
Shares Outstanding	129.6MM 142.0MM fully diluted
Cash Burn	$2MM per quarter - Current

Epigenetics Mechanism

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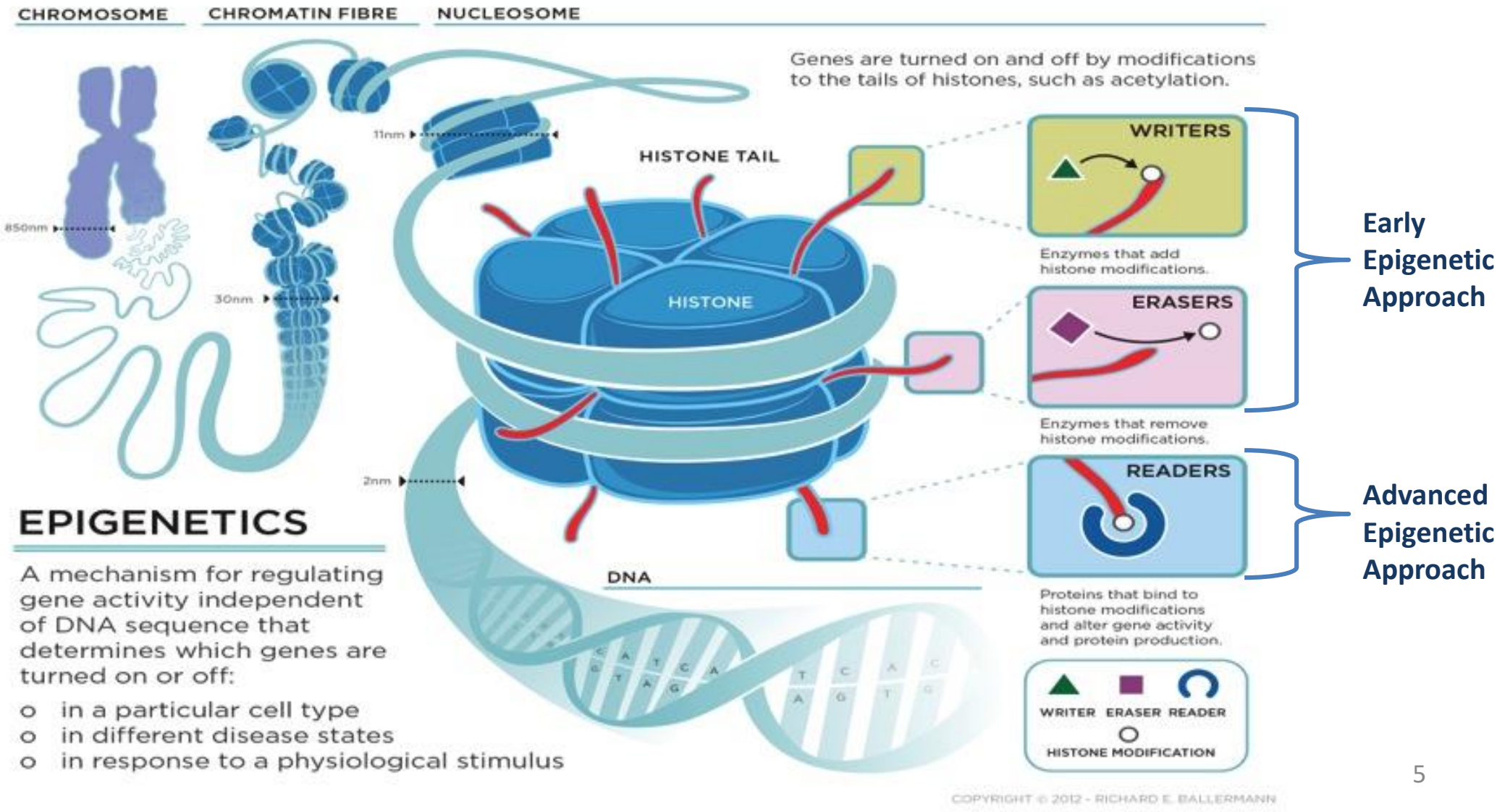
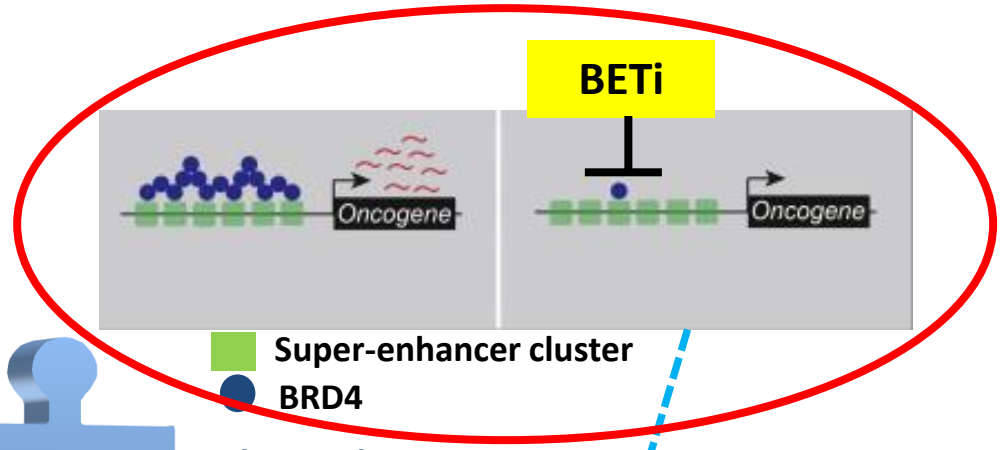
Epigenetics, the Mechanism Behind Our Approach



Zenith's BRD4 Targets Resistance Mechanisms

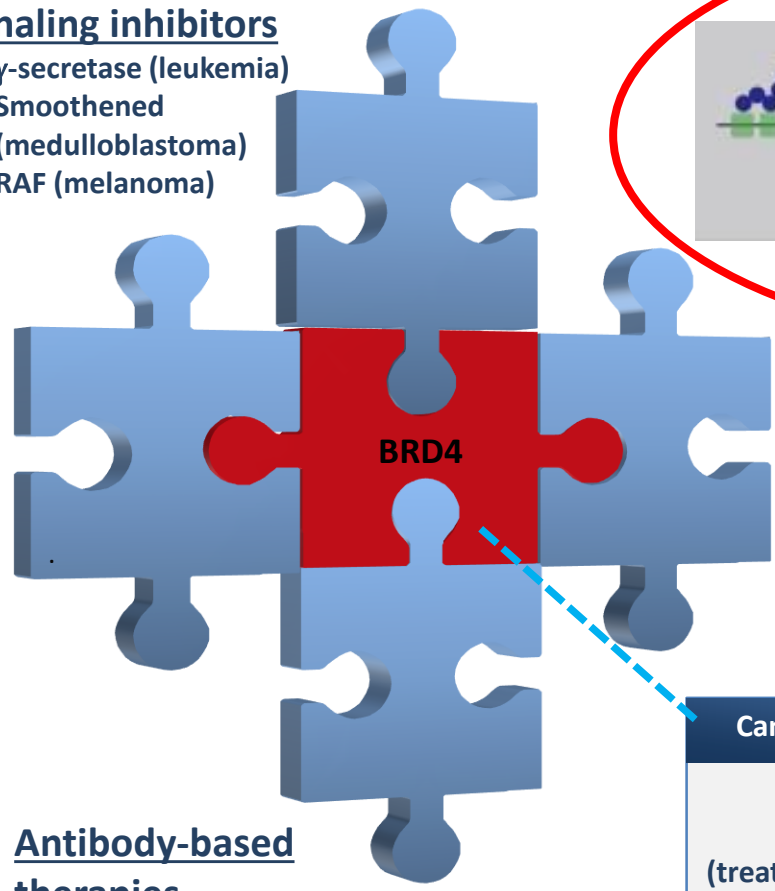


Signaling inhibitors

- γ-secretase (leukemia)
- Smoothened (medulloblastoma)
- RAF (melanoma)

Hormone-deprivation therapies

- Androgen (prostate cancer)
- Estrogen (breast cancer)



Chemotherapies

- Tamoxifen (breast cancer)
- Everolimus (breast cancer)

Antibody-based therapies

- Rituximab (lymphoma)

Cancers Escape Mechanism

Resistance mechanism involving BRD4 (treatment with BETi resensitizes cancers to these therapies)

Resistance to several standard of care treatments does not impede sensitivity to BETi

Epigenetic Combination Therapies - Addressing Resistance & Increasing Revenue of $B Franchises

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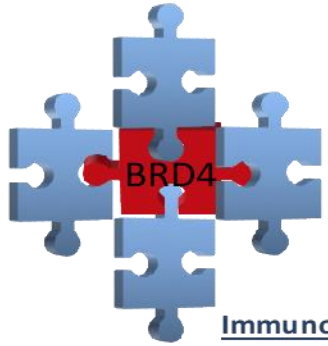
Hormone-Modulaton
Androgen (CRPC)
Estrogen (breast cancer)



DNA Repair
PARPi (Breast, Ovarian, CRPC)

Kinase-Signaling
PI3K (breast, CRPC)
RAF (melanoma)

Immuno-Oncology
Checkpoint Inhibitors (melanoma, NSCLC, etc.)

Revenue

Current multi $B Markets
- AR antagonists (lead program)
- PD-1/PD-L1Mabs
- CDK 4/6 inh.
- PARPi

Increase duration of therapy

Increase patient pool (responders)

Differentiation

Increase IP life

Combination with ZEN-3694 (BETi)

Prostate Cancer Rationale Review

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ZEN-3694 Potential in Patients Developing mCRPC Resistance to Enzalutamide

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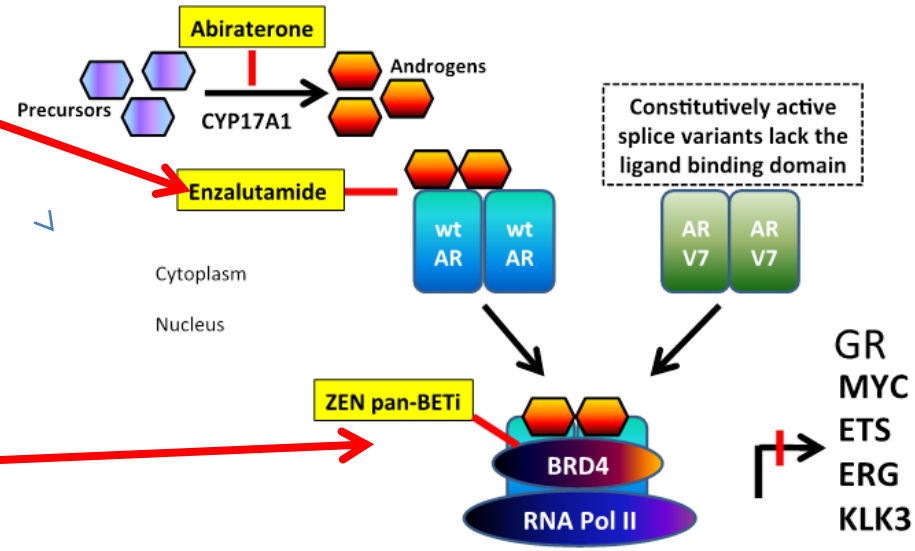
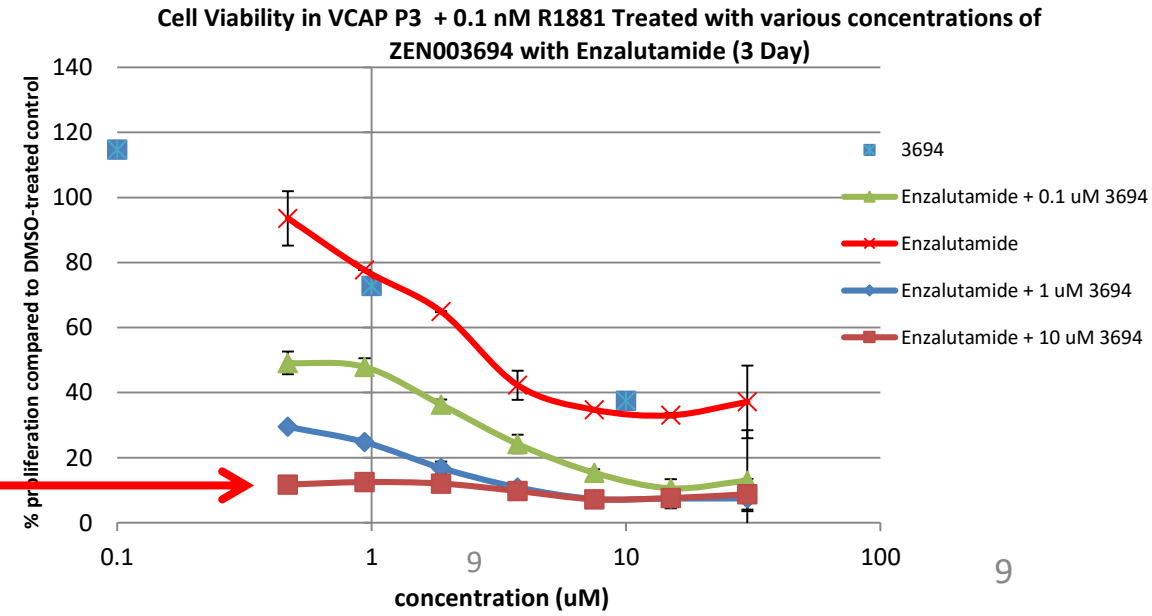
ZEN-3694 works down stream of current therapies

ZEN-3694 works where resistance to existing lead drugs, like Enzalutamide, has evolved

The combination of ZEN-3694 and enzalutamide shows strong synergy and is expected to reduce the development of resistance





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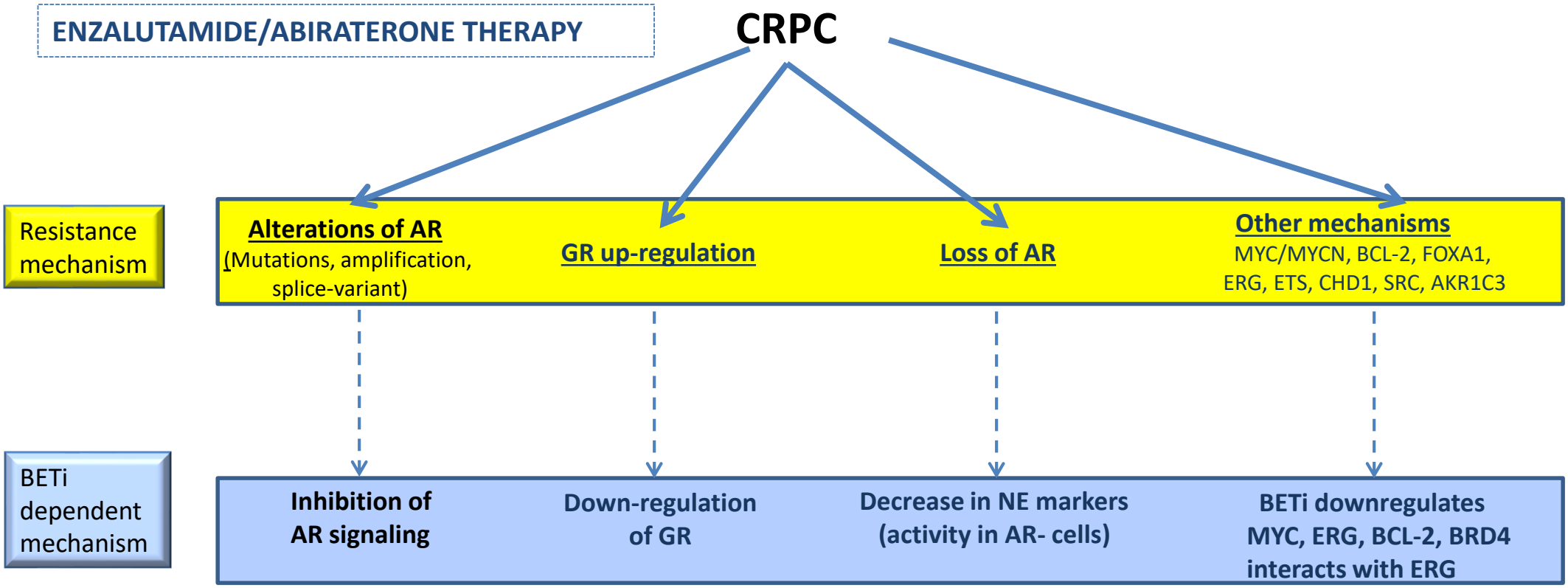
Potential Resistance Pathways in CRPC in response to Enzalutamide and/or Abiraterone

ZENITH



ZEN-3694 shows good efficacy in different CRPC models that are resistant to AR antagonists

Phase 1 Details & Results

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Zenith's Principal Investigators

Name	Institution	Comments
Eric Small, MD *Chief, Dept. of Medicine* **Rahul Aggarwal, MD** *Developmental Therapeutics Specialist, Genitourinary Oncologist*	University of California, San Francisco (UCSF)	Developed abiraterone - #2 CRPC drug, owned by J&J.
Howard Scher, MD *Chief, Genitourinary Oncology* **Wassim Abida, MD, PhD** *Medical Oncologist*	Memorial Sloane Kettering Cancer Center (MSKCC)	Developed enzalutamide - #1 CRPC drug, now owned by Pfizer. Developing ARN-509 for J&J
Joshi Alumkal, MD *Associate Professor*	Oregon Health Sciences University (OHSU)	Expert in epigenetics in prostate cancer research
Allan Pantuck, MD *Professor, Dept. of Urology*	University of California Los Angeles (UCLA)	Involved in enzalutamide and provenge development
Elizabeth Heath, MD *Professor, Dept. Hematology/Oncology*	Karmanos (Wayne State)	Genitourinary oncology specialist
Michael Schweizer, MD *Oncologist*	University of Washington	Genitourinary oncology specialist
David M. Nanus, MD *Chief, Division of Hematology and Medical Oncology*	Weill Cornell Medicine	Genitourinary oncology specialist

ZEN-3694 Development in mCRPC- Phase 1 Single Agent Study Results

2016		2017	
1H	2H	1H	2H

Single agent dose escalation; enzalutamide and/or abiraterone failures N~12

Single agent expansion at RP2D; same population as dose escalation N=12

Key Learnings

- Maximum tolerated dose (MTD) defined ✓
- Dose proportional PK ✓
- Good safety profile, prolonged dosing without dose interruption/reduction is feasible ✓
- Target modulation shown at doses below MTD ✓
- Some single agent anti-tumor activity/disease stabilization observed in multiple patients ✓

Single agent study key to understanding drug characteristics and supporting combination study

Patient X: Prolonged Disease Stabilization

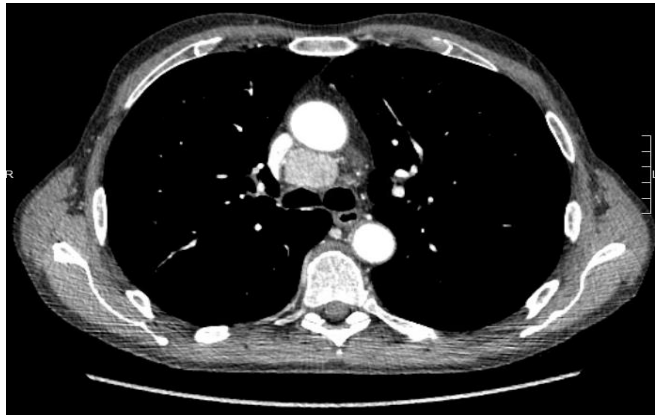
Prior Therapy for mCRPC

- Provenge
- Enzalutamide: 6/5/2014 – 5/5/2016 – acquired resistance
- Abiraterone: 5/22/2016 – 8/12/2016 – primary resistance

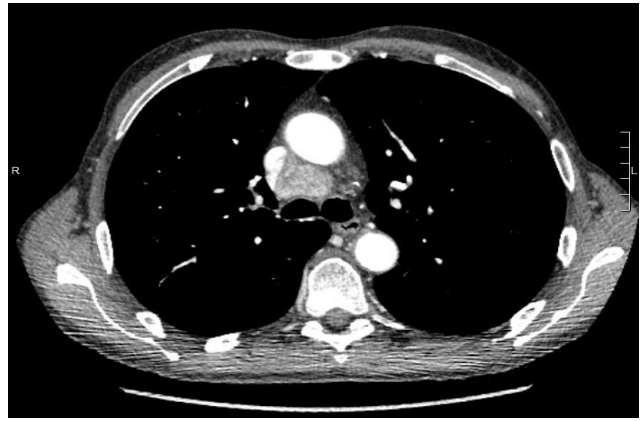
- ZEN-3694: 8/24/2016 – 7/16/2016, 45 weeks

Stable mediastinal nodes over 8 months

Study Entry

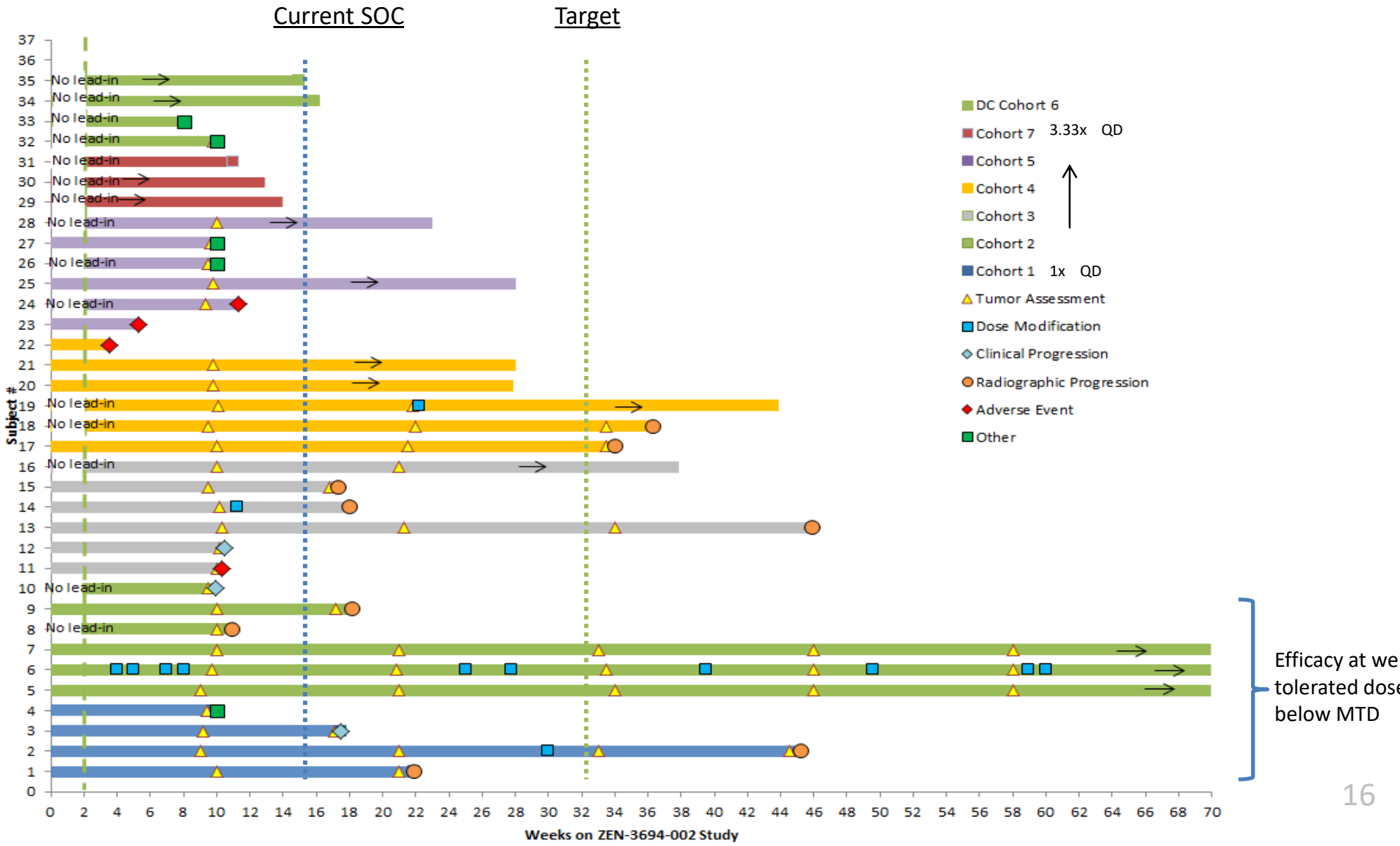


32 Weeks



Enzalutamide Combination Trial – Phase 1b/2a

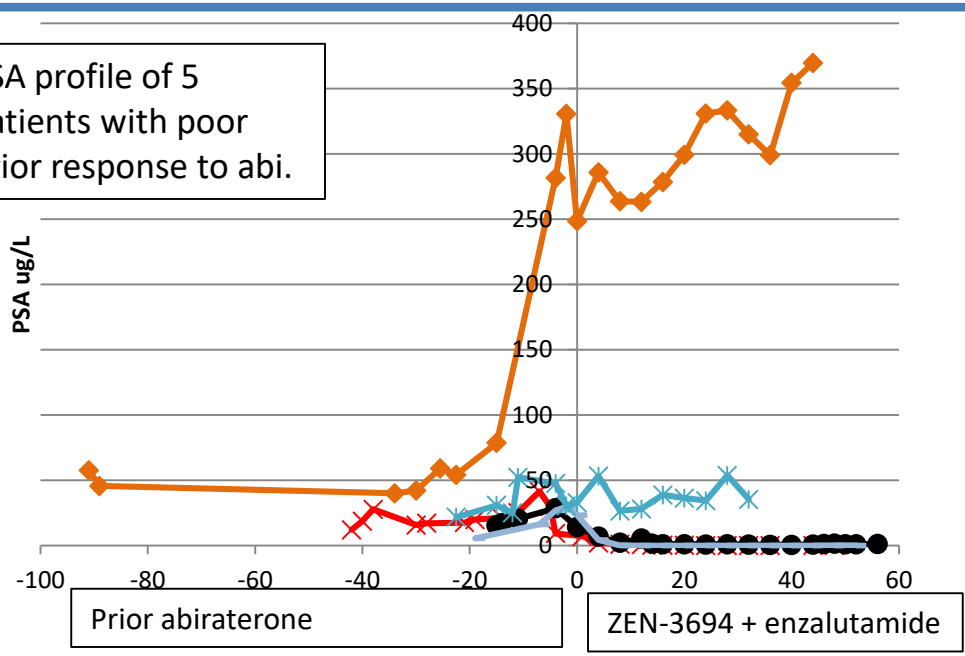
ZEN-3694-002 Treatment Duration (Updated August 22nd, 2018)



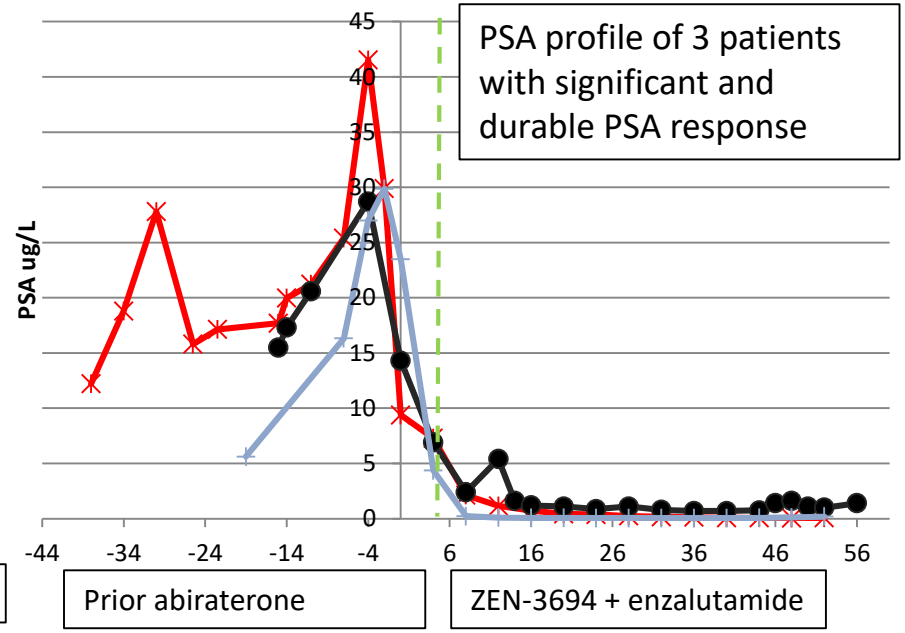
Efficacy at well tolerated doses below MTD

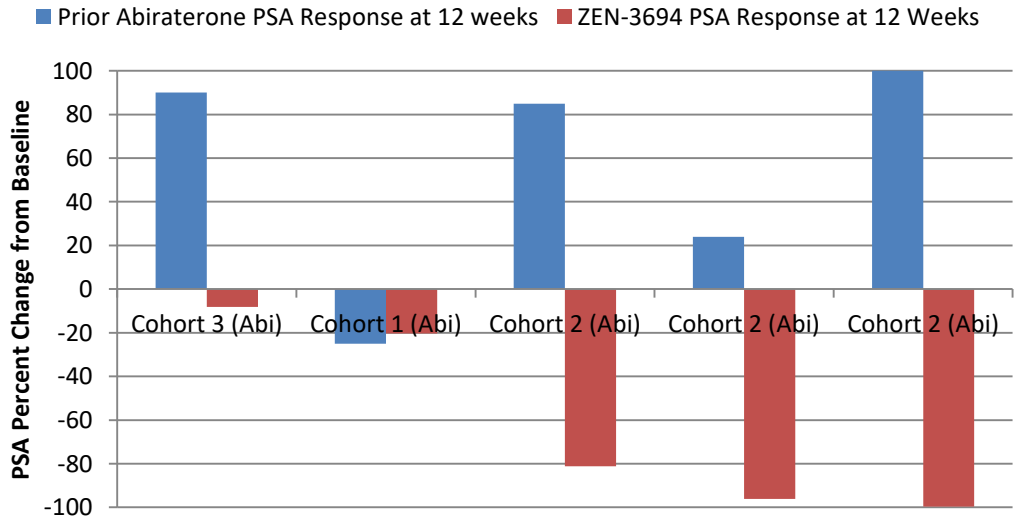
Strong PSA Response with ZEN-3694 + Enzalutamide in Patients with Poor Prior Response to Abiraterone

PSA profile of 5 patients with poor prior response to abi.



PSA profile of 3 patients with significant and durable PSA response





Artificial Intelligence Program Developed Based on Zenith's Clinical Data

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FULL PAPER

Artificial Intelligence

ADVANCED THERAPEUTICS

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Modulating BET Bromodomain Inhibitor ZEN-3694 and Enzalutamide Combination Dosing in a Metastatic Prostate Cancer Patient Using CURATE.AI, an Artificial Intelligence Platform

Allan J. Pantuck, Dong-Keun Lee, Theodore Kee, Peter Wang, Sanjay Lakhotia, Michael H. Silverman, Colleen Mathis, Alexandra Drakaki, Arie S. Belldegrun, Chih-Ming Ho,* and Dean Ho**

Combination chemotherapy is a cornerstone of cancer treatment. Optimizing its effectiveness requires dose- and time-dependent regulation of drug synergy. In this report, CURATE.AI, an artificial intelligence platform, is used to prospectively guide the dosing of a bromodomain inhibitor (ZEN-3694) and enzalutamide administered in combination to a patient with metastatic castration-resistant prostate cancer to reduce serum prostate-specific antigen (PSA) levels. CURATE.AI successfully identifies substantial ZEN-3694 and enzalutamide dose adjustments, increasing both treatment efficacy and tolerance. CURATE.AI analysis also confirms that the patient's durable response is mediated by ZEN-3694 inclusion in the regimen. Due to CURATE.AI-enhanced efficacy and safety, the patient was able to continue with the combination regimen, resulting in a durable response and no disease progression based on CURATE.AI-sustained control over PSA levels and reduced lesion size.

1. Introduction

Conventional chemotherapy simultaneously addresses multiple aberrant disease pathways to potentially improve treatment outcomes. Drug doses are typically determined using dose escalation to reach a maximum tolerated dose (MTD) or via dose expansion to identify suitable regimen administration guidelines.[1,2] These combinations are subsequently administered at fixed doses. While the administration of combination therapy using these approaches has served as a clinical standard for clinical care, the patient's response to therapy evolves during the course of treatment due to the time-dependent, dose-dependent, and patient-specific nature of drug synergy and resulting efficacy and

UCLA Artificial Intelligence Program Confirmation

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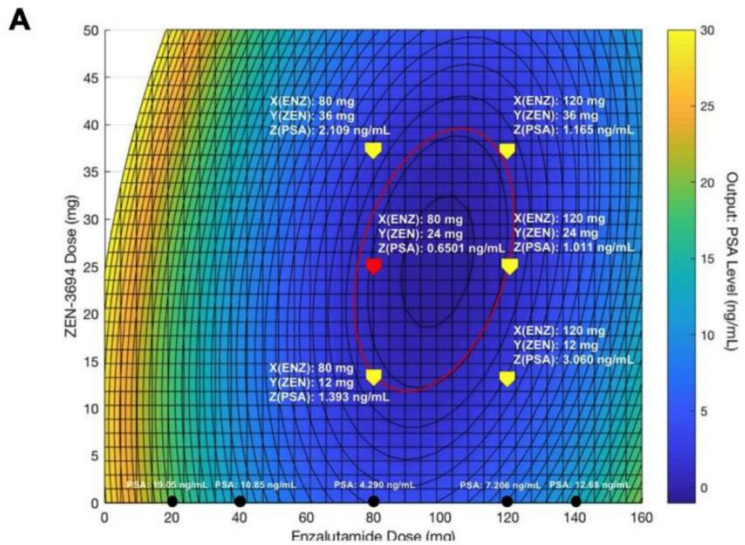

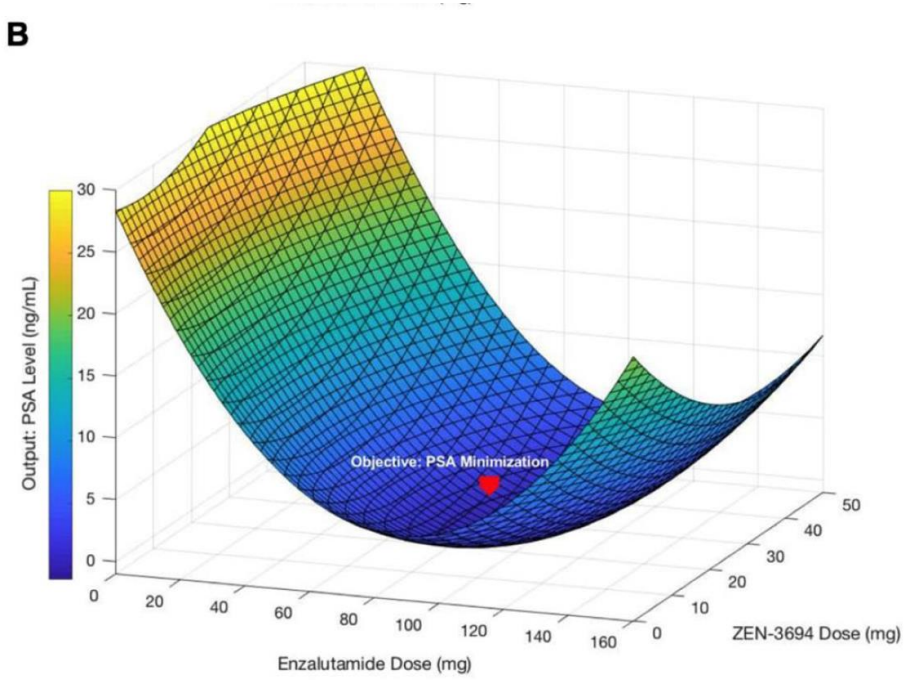

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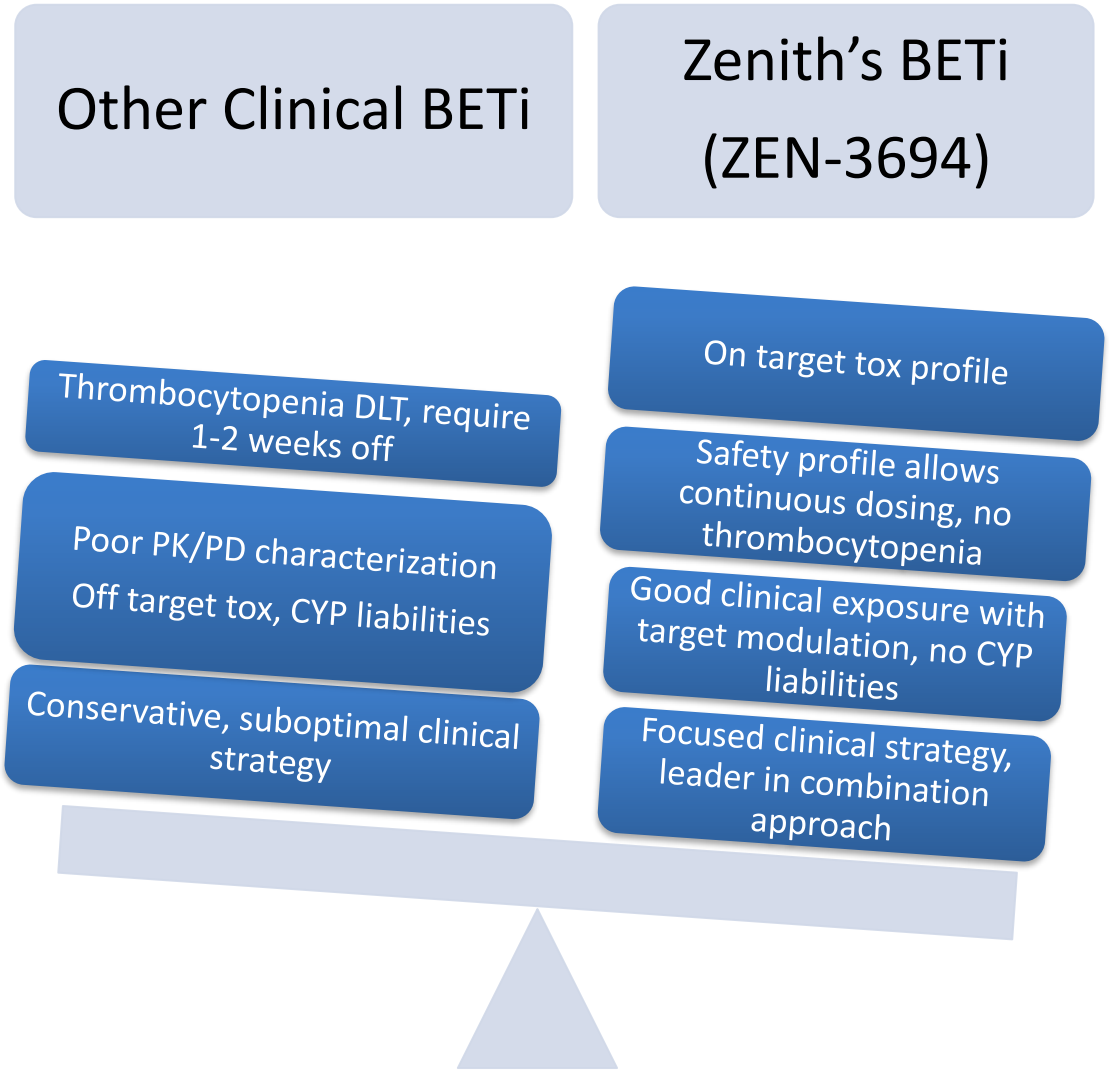
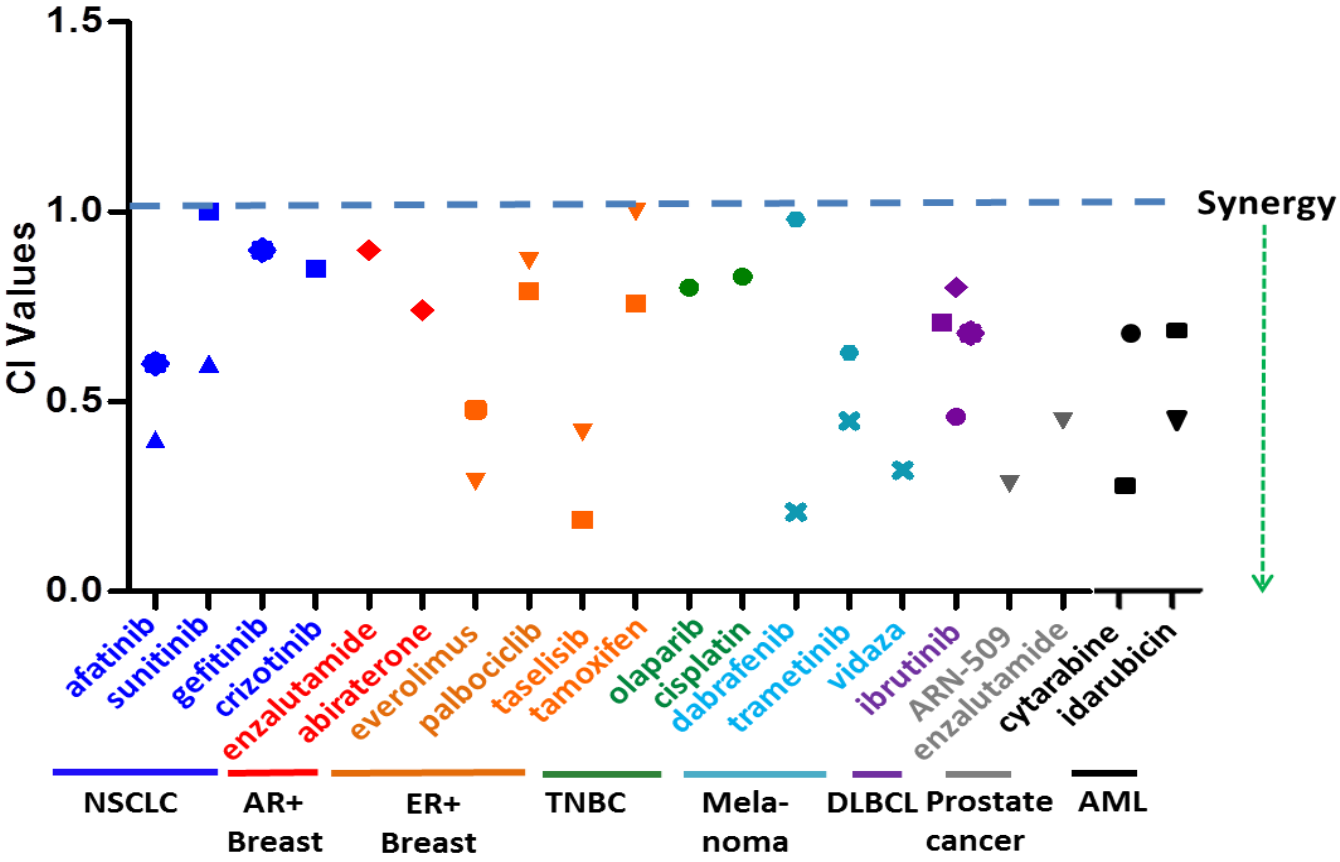
Zenith's BETi Program is Clinically Differentiated



Other companies developing BETi for CRPC
Gilead – Phase 1b/2a (Single agent and combination)
GSK – Phase 1, just initiated (combination)

ZEN-3694 Synergizes With Several Standard of Care Cancer Drugs



Indication		Cell line (mutation)
NSCLC	▲	H1975 (EGFR L858R T790M)
	⬢	H820 (EGFR T790M)
	■	H2228 (ALK)
AR+ Breast	◆	MDA-MB-453
ER+ Breast	■	MCF-7 (ER+)
	▼	ZR-75-1 (ER+)
TNBC	●	HCC1937 (BRCA1)
Melanoma	✖	C32 (BRAF V600E)
	⬢	A375 (BRAF)
DLBCL	⬢	CARNAVAL (MYC/BCL2)
	●	OCI-LY18 (MYC/BCL2)
	◆	NU-DUL-1
	■	OCI-LY3 (A20)
Prostate	▼	VCAP (AR AMP/AR-V7)
AML	■	MV4-11 (MLL-AF4/FLT3-ITD)
	●	OCI-AML2 (DNMT3A/MLL)
	▼	OCI-AML3 (DNMT3A/NPM1)

CANCER

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

Article

Cancer Cell

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

Report

Cell Reports

BET Bromodomain Inhibition Synergizes with PARP Inhibitor in Epithelial Ovarian Cancer

Q & A