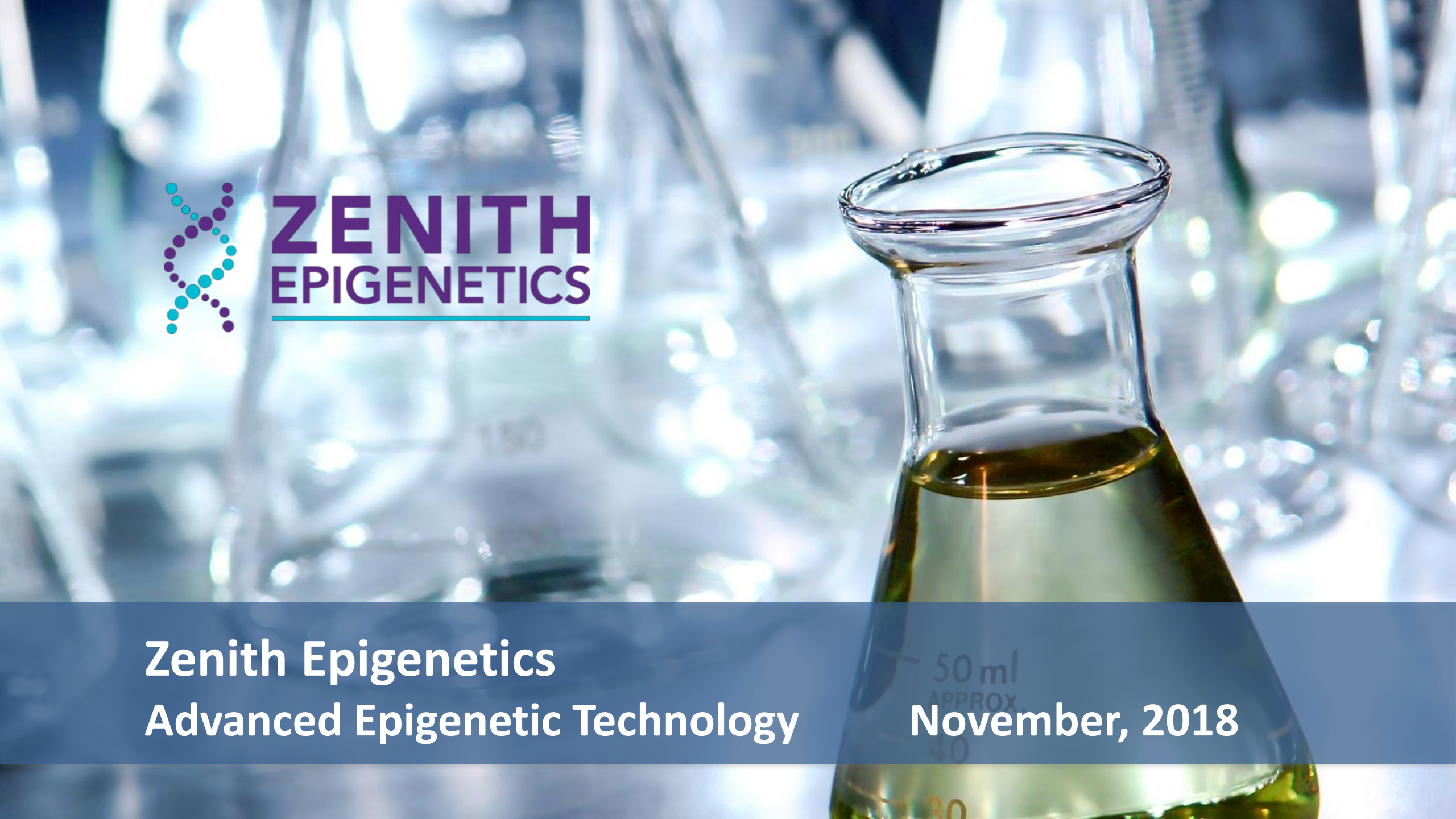


Zenith Epigenetics

Advanced Epigenetic Technology

November, 2018

Forward Looking Statement

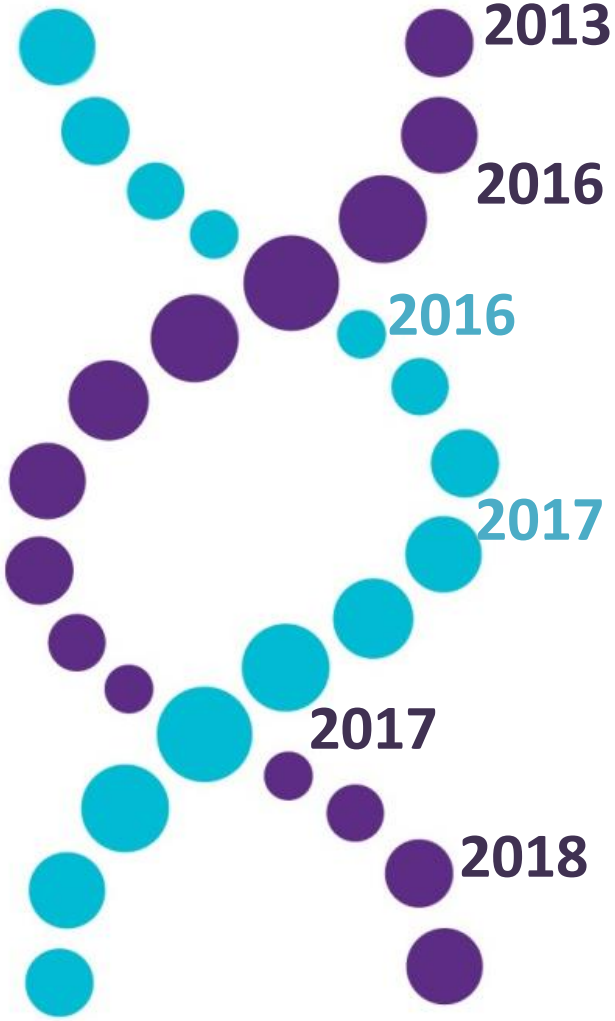
Safe Harbor Statement. This presentation contains forward-looking statements that involve risks and uncertainties, which may cause actual results to differ materially from the statements made. For this purpose, any statements that are contained herein that are not statements of historical fact may be deemed to be forward- looking statements made pursuant to the safe harbor provisions of the Private Securities Litigation Reform Act of 1995. Without limiting the foregoing, the words "believes," "anticipates," "plans," "intends," "will," "should," "expects," "projects," and similar expressions are intended to identify forward-looking statements. You are cautioned that such statements are subject to a multitude of risks and uncertainties that could cause actual results, future circumstances, or events to differ materially from those projected in the forward-looking statements. These risks include, but are not limited to, those associated with the success of research and development programs, the regulatory approval process, competition, securing and maintaining corporate alliances, market acceptance of the Company's products, the availability of government and insurance reimbursements for the Company's products, the strength of intellectual property, financing capability, the potential dilutive effects of any financing, reliance on subcontractors and key personnel. The forward-looking statements are made as of the date hereof, and the Company disclaims any intention and has no obligation or responsibility, except as required by law, to update or revise any forward-looking statements, whether as a result of new information, future events, or otherwise.

CONTACT: Donald J. McCaffrey
Chairman, President & CEO

Suite 300, 4820 Richard Road S.W. Calgary, AB, Canada T3E 6L1
Tel: (403) 254-9252, Fax:(403) 256-8495, http://www.zenithepigenetics.com

Corporate Profile and Milestones

Cash Raised 2014-2018	~US$50MM @ US$1.00 & US$2.00 per unit based on pre-clinical results
Enterprise Value est.	US$325MM (US$2.50/Share) est.
Shares Outstanding	129.6MM 142.0MM fully diluted
Cash Burn Current	US$2MM per quarter



2013 — Founded June 2013
Spin out from Resverlogix Corp.

2016 — June 2016
Dosing first mCRPC patient with ZEN-3694

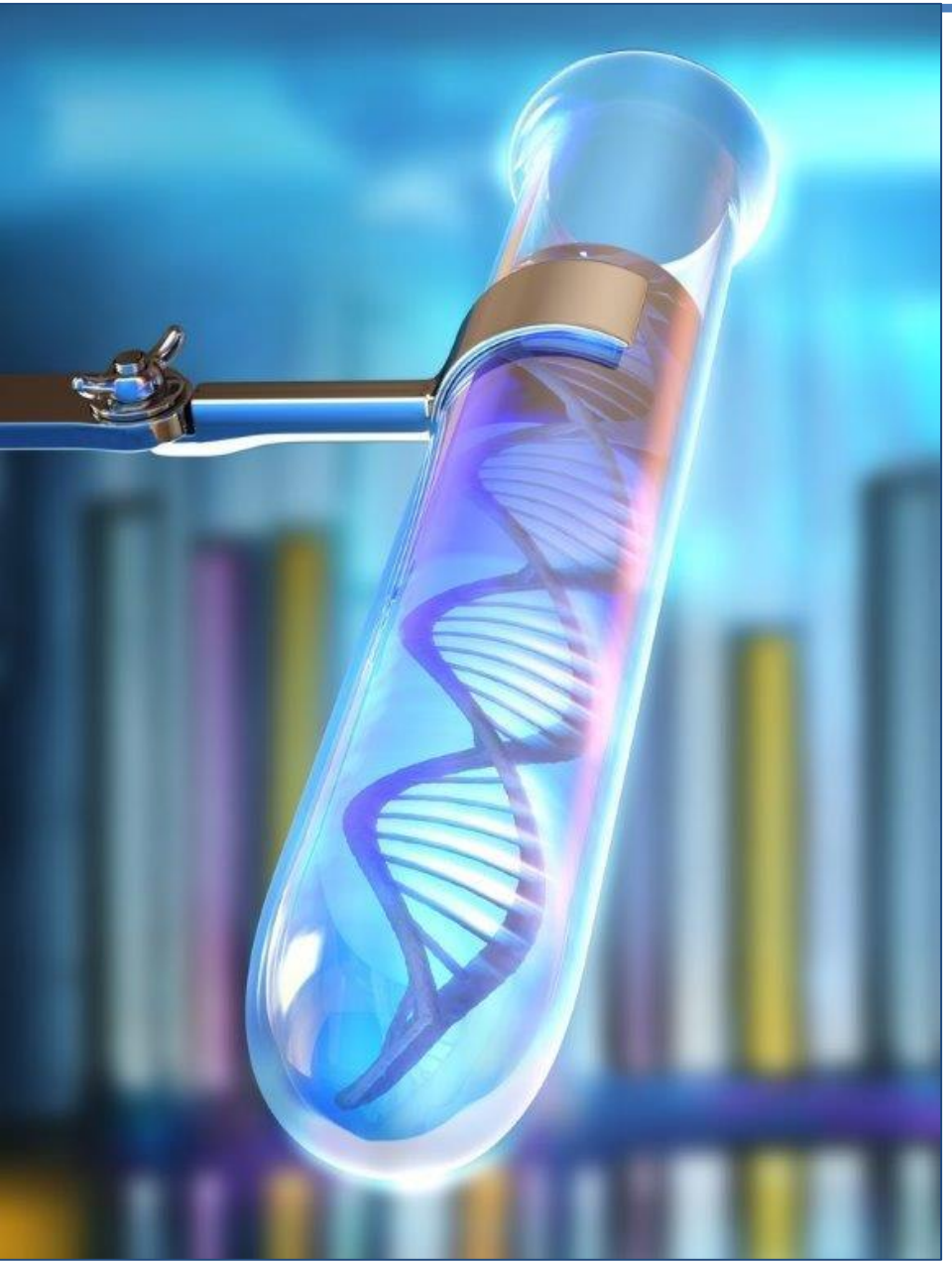
2016 — Dec 2016
Initiating combination study with Enzalutamide in MCRPC patients

2017 — June 2017
Announce issuance of US patent for ZEN-3694

2017 — Oct 2017
Successful completion of single agent study with ZEN-3694

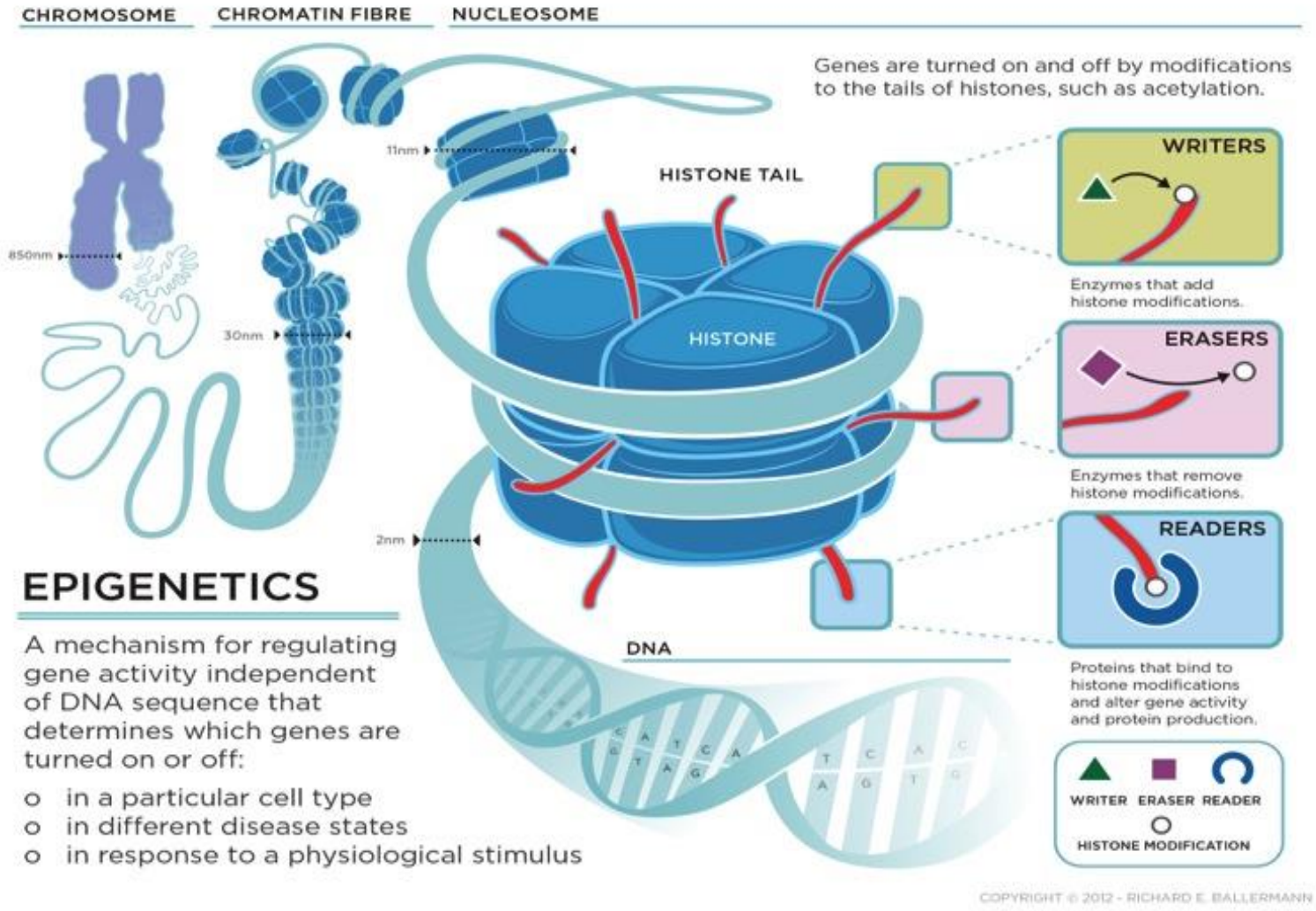
2018 — Nov 2018
Successful publications and 80 patients dosed with ZEN-3694

Todays Agenda for Zenith Capital Corp.



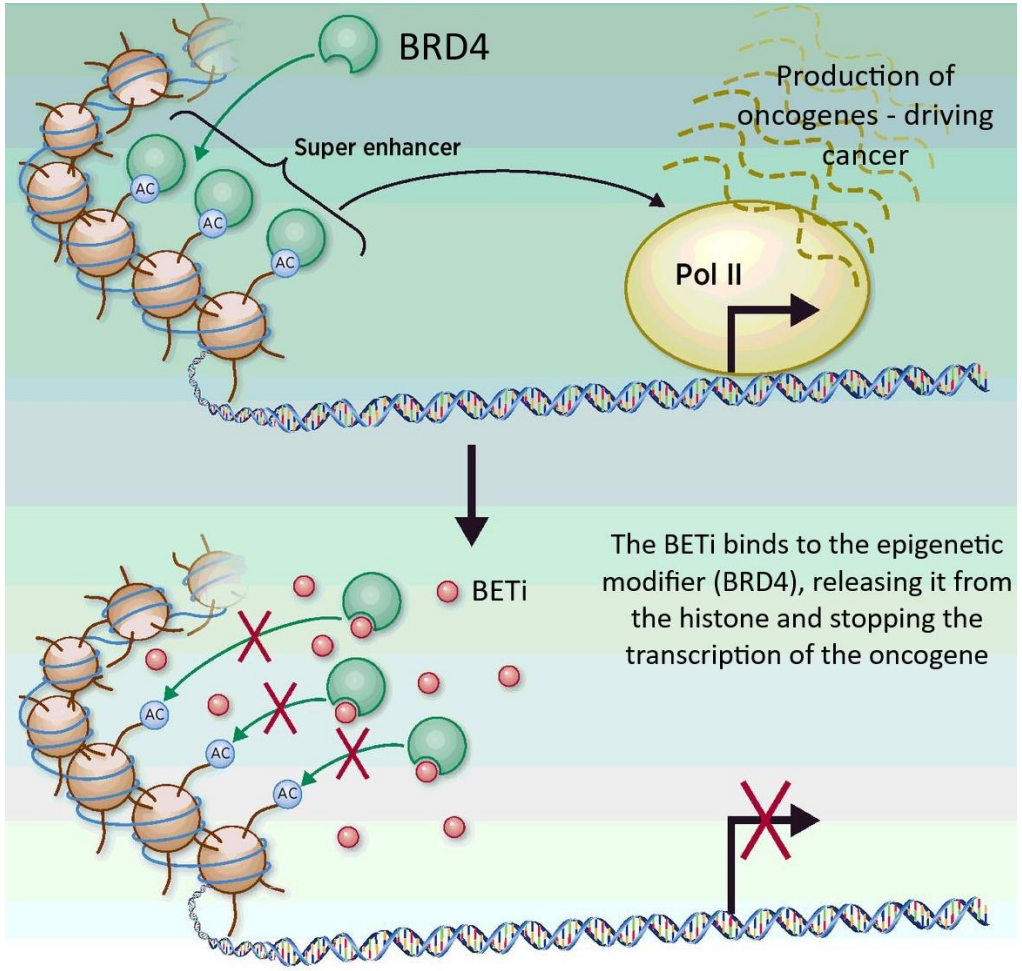
- **Epigenetics – The Mechanism Behind Our Approach**
- The Potential of BET Inhibition
- Prostate Cancer Rationale Review
- Promising Early Clinical Results & Case studies
- Next Steps for Zenith

Epigenetics - The Mechanism Behind Our Approach



- The epigenetic code refers to modifications to chromatin components that regulate its activity
- Turning genes on or off is regulated by these modifications
- BET (Bromodomain and Extraterminal Domain) proteins recognize these modifications and turn genes on/off

BET Inhibitors Target Resistance Mechanisms – Sensitizing the Tumor to Existing Therapy

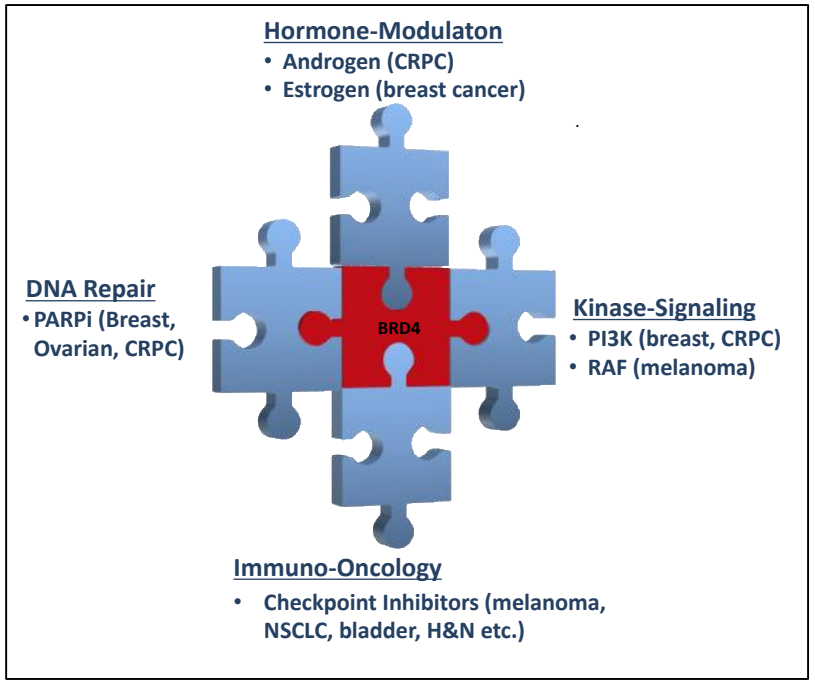


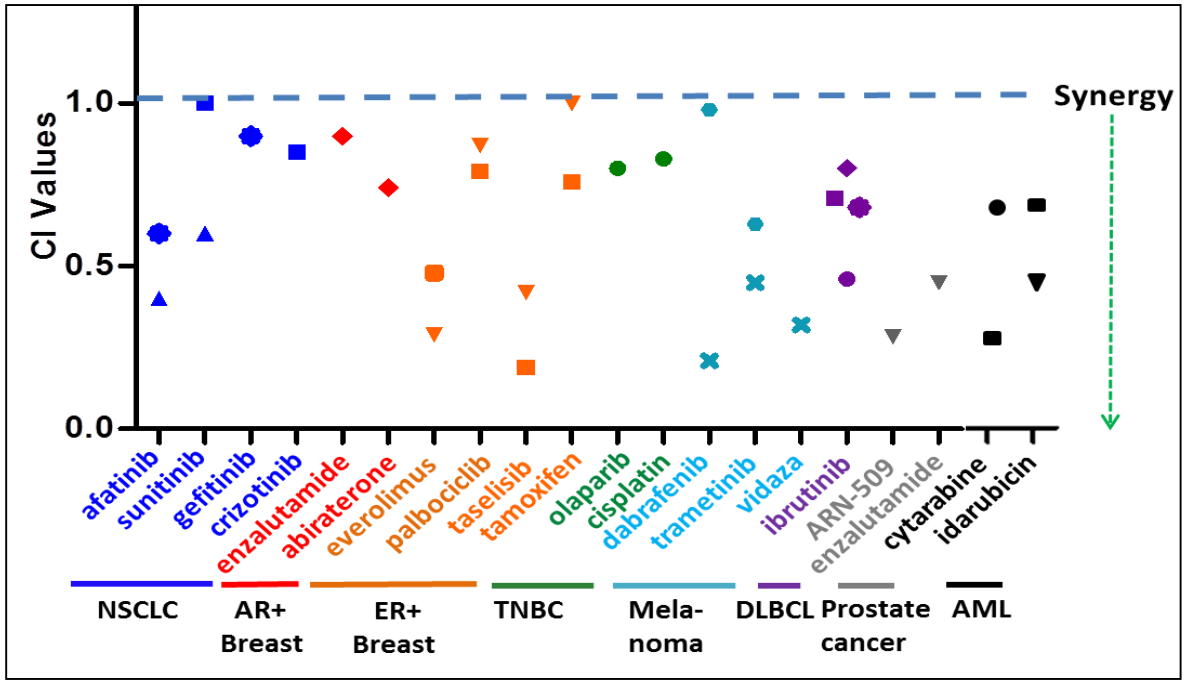
- Many of the escape resistance mechanisms to standard of care treatments involve BRD4
- BETi blocks BDR4 binding, resulting in inhibition of tumor oncogenes by disruption of super-enhancers
- Resistance to several standard of care treatments does not impede sensitivity to BETi, allowing for valuable combination therapy

Adopted from Clinical Cancer Research 2017, 23(7), 1647-55.

Combination Therapy: The Potential of BET Inhibition and ZEN-3694

BET inhibitors has the ability to work synergistically with other therapies overcoming resistance and enhancing the response to the combination, resulting in broader and extended use of existing therapies

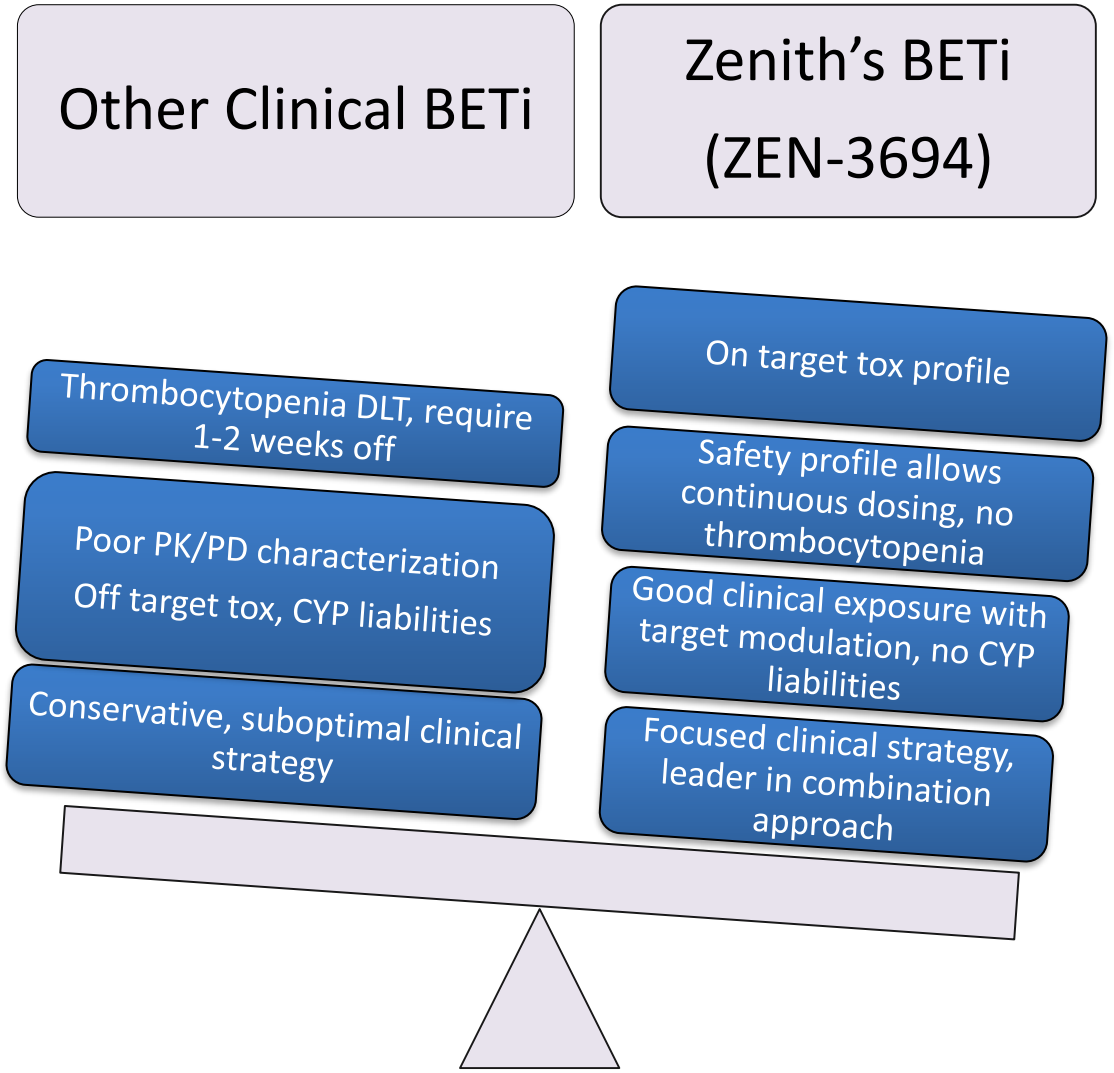




The use of BETi is applicable to a number of cancers and therapies

ZEN-3694 synergizes with several standard of care cancer drugs

Zenith's BETi program is Clinically Differentiated

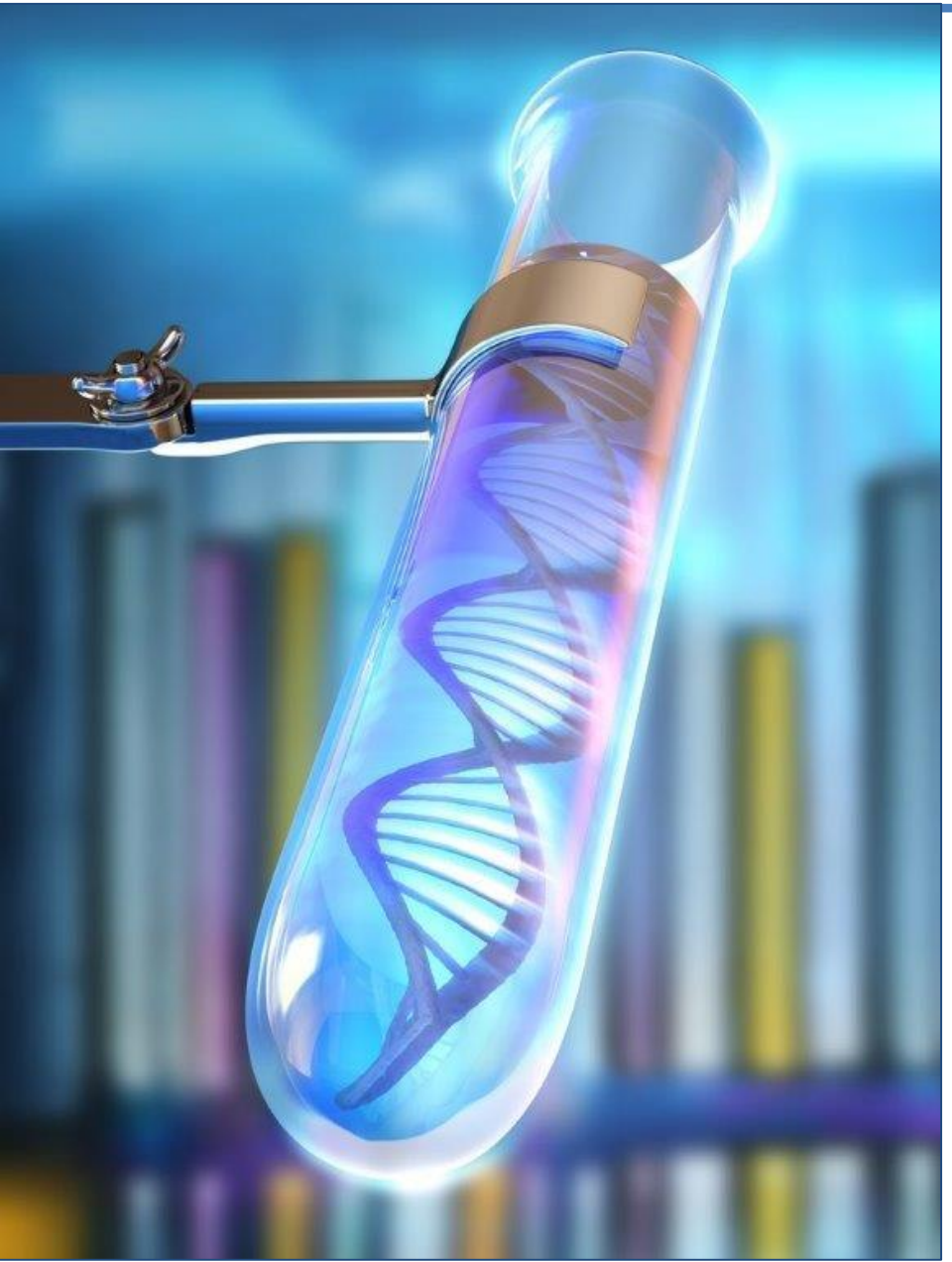


Significant initial excitement with BETi

Poor compounds (benzodiazepine derivatives) and suboptimal single agent clinical strategy in all comer trials led to lack of proof of concept

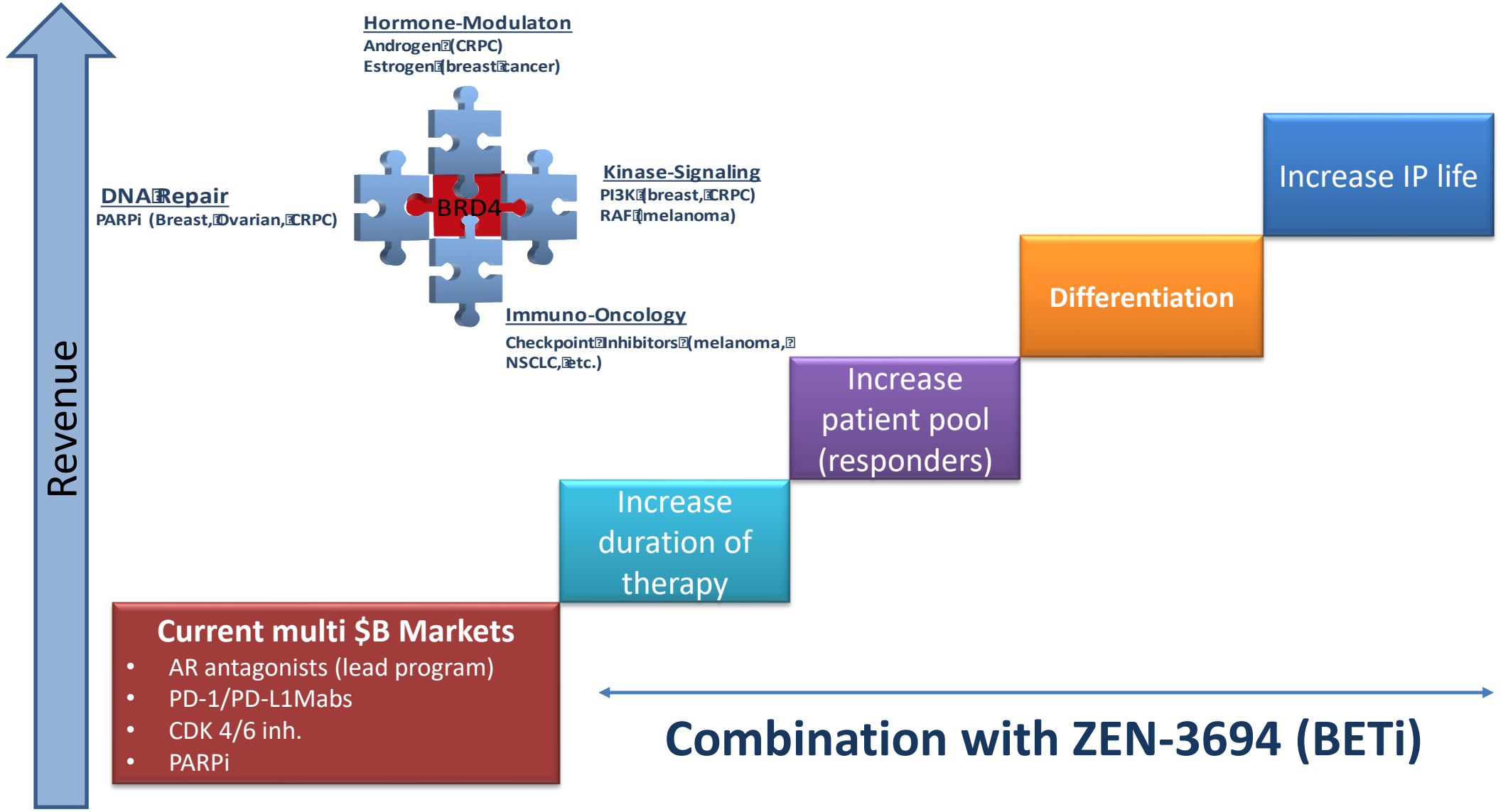
The BETi field now pushing ahead with combination trials in focused clinical trials (BMS, Roche, GSK, Abbvie, BI etc.)

Todays Agenda for Zenith Capital Corp.

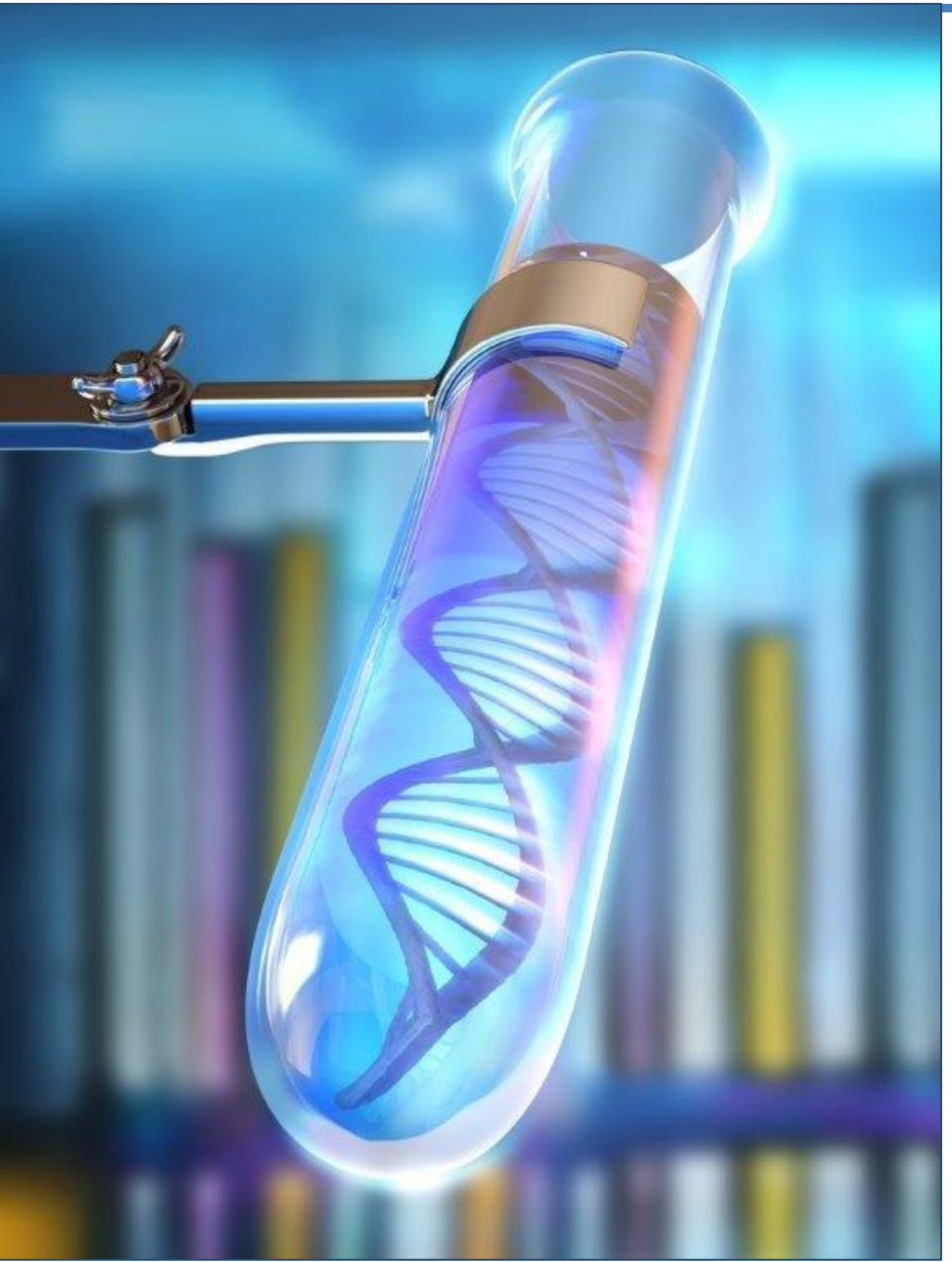


- Epigenetics – The Mechanism Behind Our Approach
- **The Potential of BET Inhibition**
- Prostate Cancer Rationale Review
- Promising Early Clinical Results & Case studies
- Next Steps for Zenith

The Potential of BET Inhibition & ZEN-3694



Todays Agenda for Zenith Capital Corp.



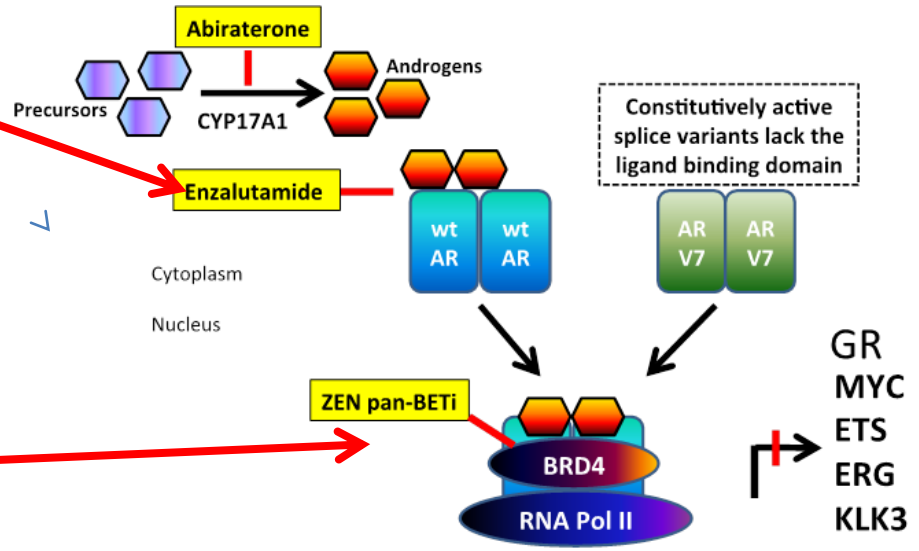
- Epigenetics – The Mechanism Behind Our Approach
- The Potential of BET Inhibition
- **Prostate Cancer Rationale Review**
- Promising Early Clinical Results & Case studies
- Next Steps for Zenith

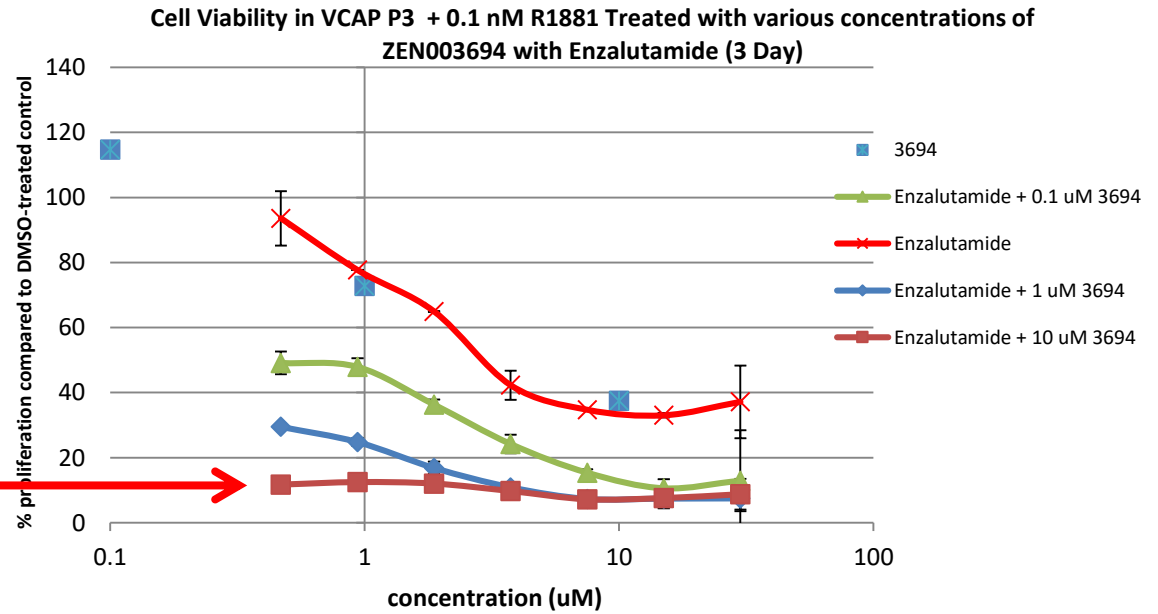
ZEN-3694 Potential in Patients Developing mCRPC Resistance to Enzalutamide

ZEN-3694 works down stream of current therapies

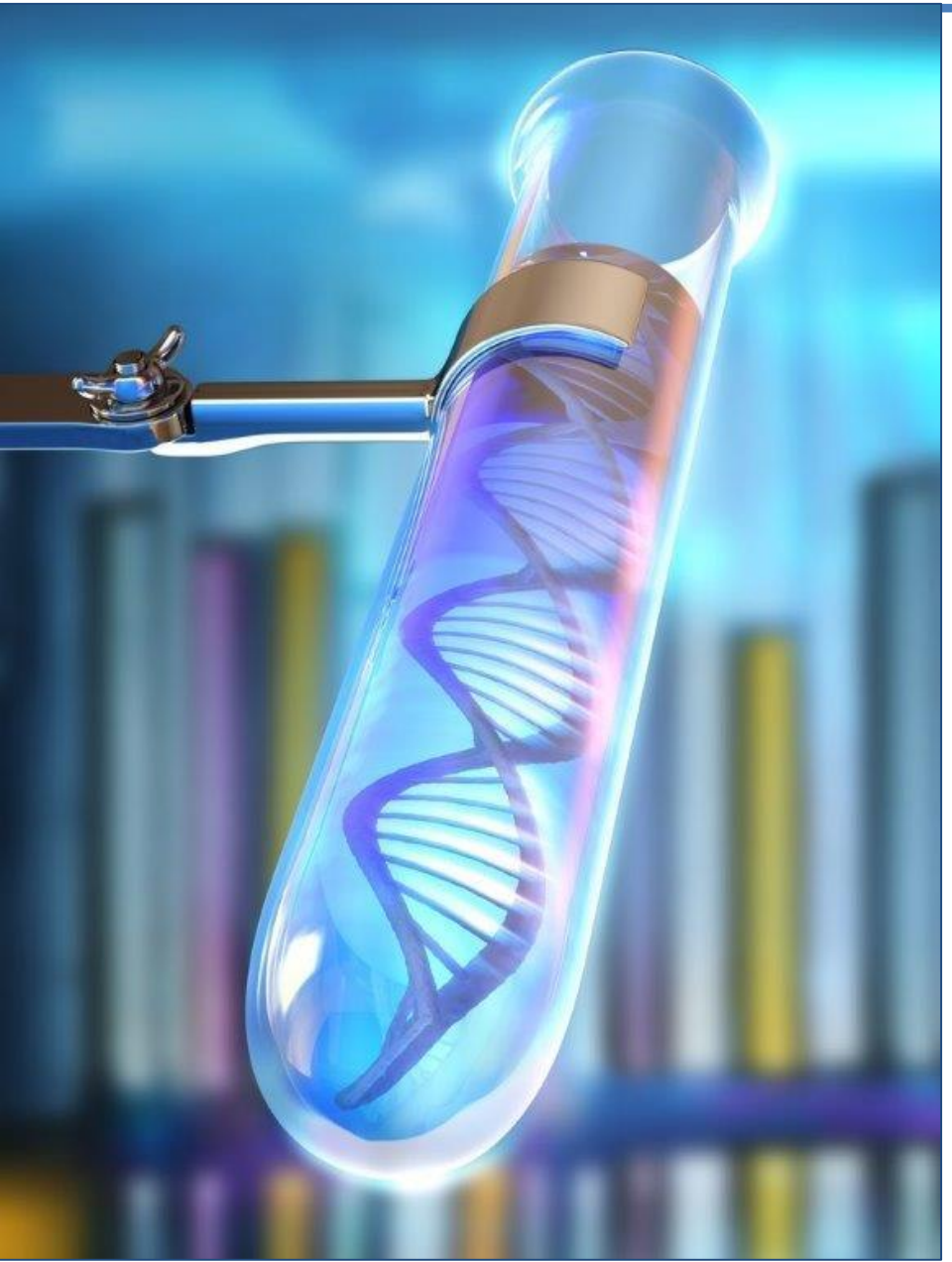
ZEN-3694 works where resistance to existing lead drugs, like Enzalutamide, has evolved

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





Todays Agenda for Zenith Capital Corp.



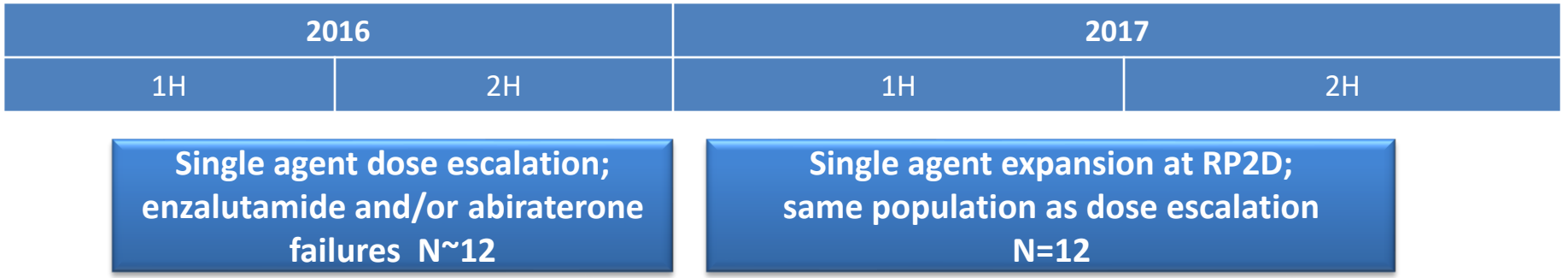
- Epigenetics – The Mechanism Behind Our Approach
- The Potential of BET Inhibition
- Prostate Cancer Rationale Review
- **Promising Early Clinical Results & Case studies**
- Next Steps for Zenith

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** **Fred Hutchinson Cancer Center**	**Experience with AR antagonists**
David M. Nanus, MD *Chief, Division of Hematology and Medical Oncology*	**Weill Cornell Medicine**	**Genitourinary oncology specialist**

ZEN-3694 Development in mCRPC: Single Agent Study Results

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



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 ✓

Patient X: Prolonged Disease Stabilization

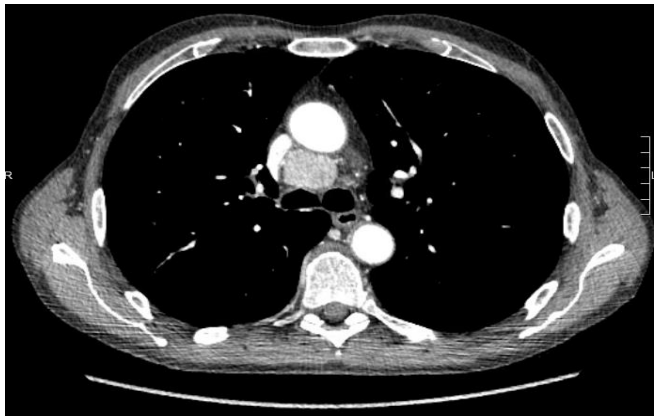
Patient Profile:

Prior Therapy for mCRPC

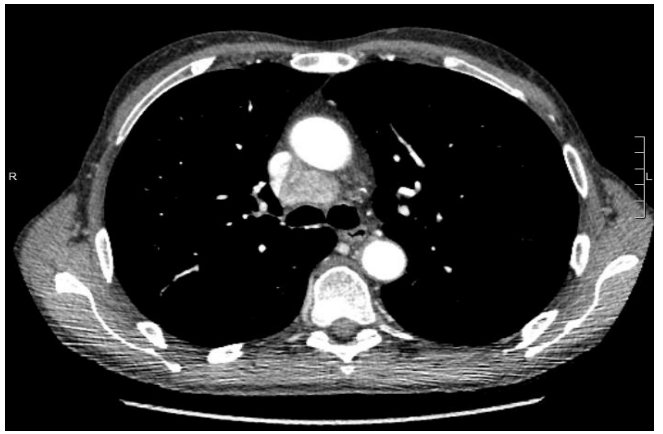
- Provenge
- Enzalutamide: 6/5/2014 – 5/5/2016 – acquired resistance
- Abiraterone: 5/22/2016 – 8/12/2016 – primary resistance

The prognosis for a patient with this profile is typically very poor and the patient would often be offered chemotherapy as the next option.

ZEN-3694: 8/24/2016 – 7/16/2016 (45 weeks) with stable mediastinal nodes over 8 months



Study Entry



32 Weeks

ZEN-3694 Development in mCRPC: Combination with Enzalutamide

2017		2018		2019
1H	2H	1H	2H	1H

Combination dose escalation
ZEN-3694 + enzalutamide; Patients progressing on abiraterone or enzalutamide N~35

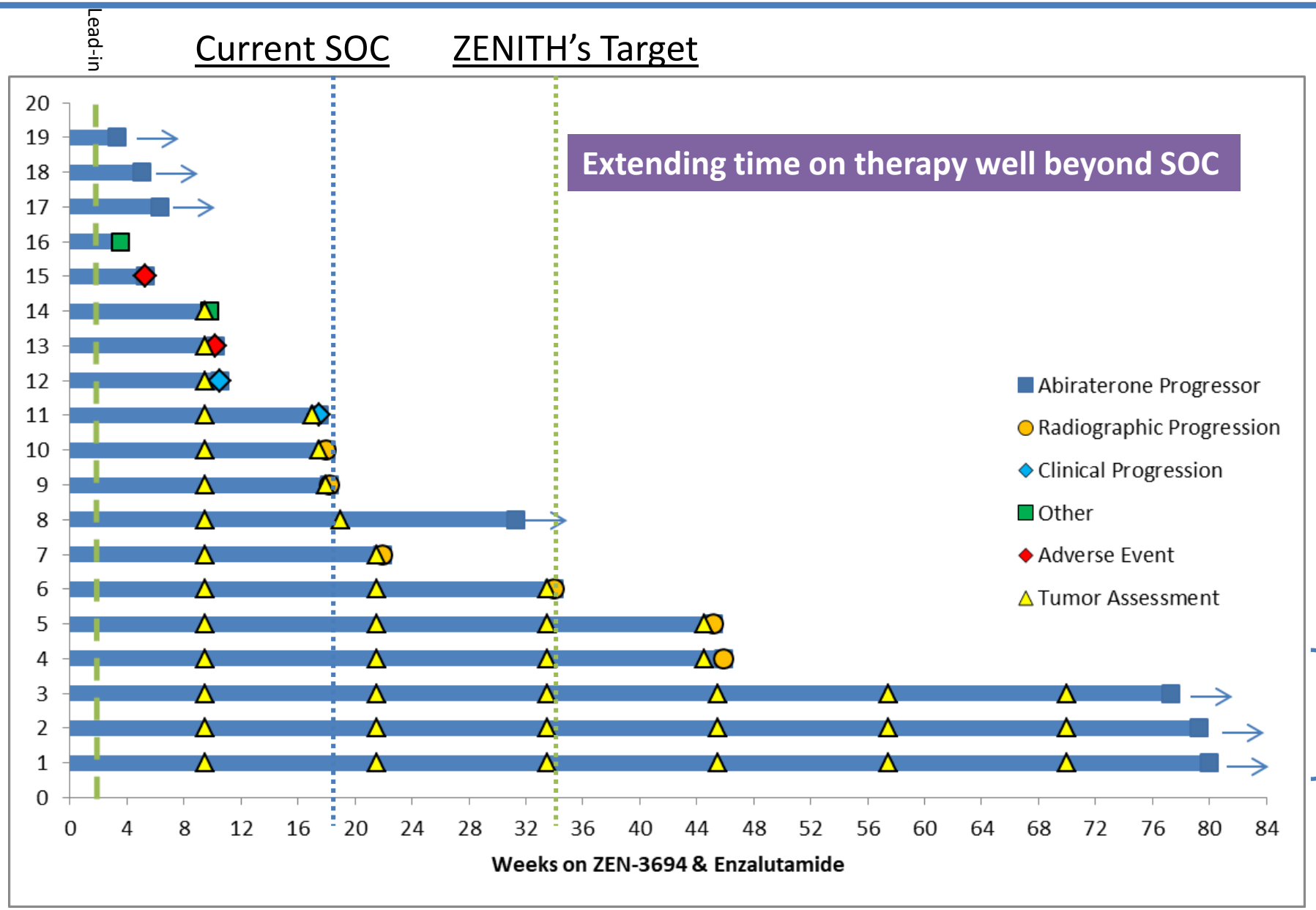
Combination expansion
ZEN-3694 + enzalutamide; Patients progressing on abiraterone N~20-25

Combination expansion
ZEN-3694 + enzalutamide; Patients progressing on enzalutamide N~10-15

- Dose escalation near completion, Expansion cohorts enrolling
- Robust target modulation at well tolerated doses, prolonged dosing without dose interruption/reduction is tolerated
- Clinical activity in patients progressing on abiraterone/enzalutamide
- Significant response in primary abiraterone progressors (rPFS and PSA)
- **>65 patients dosed to date**

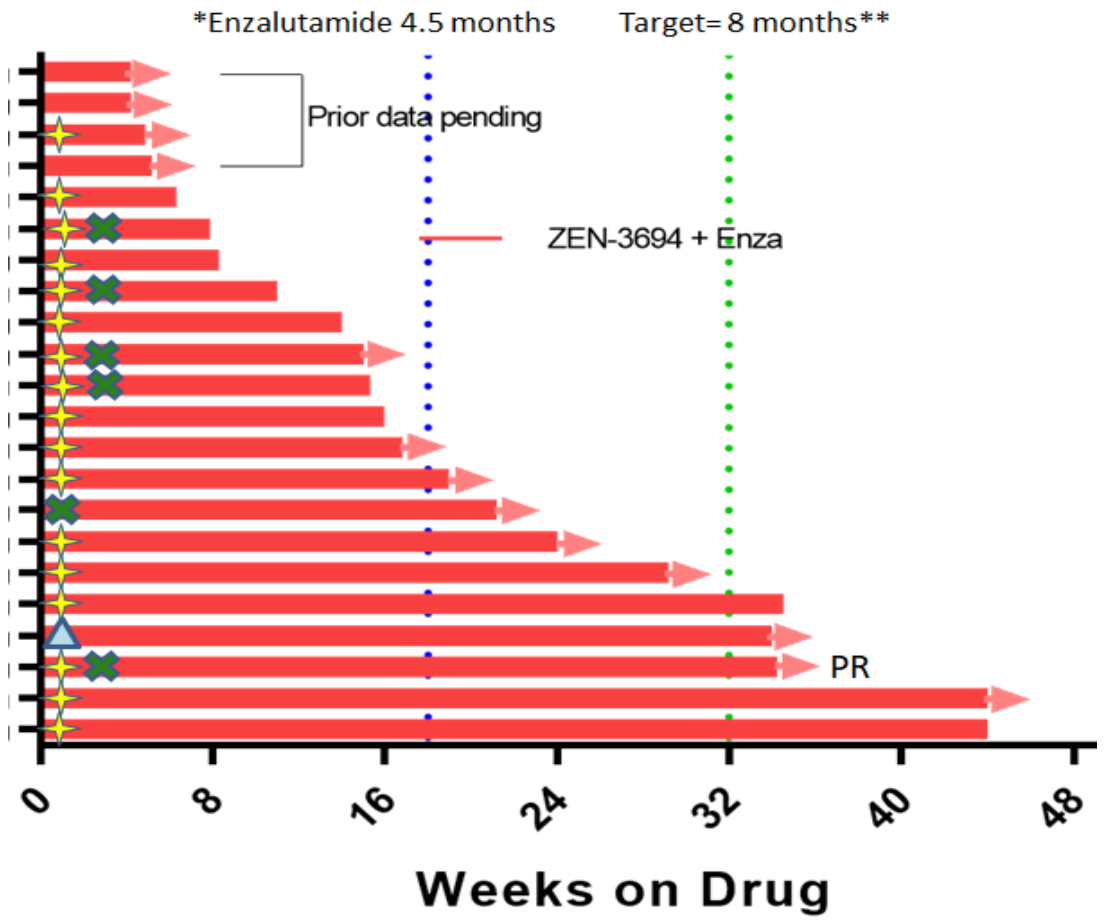
ZEN-3694 Development in mCRPC: Combination Study Update

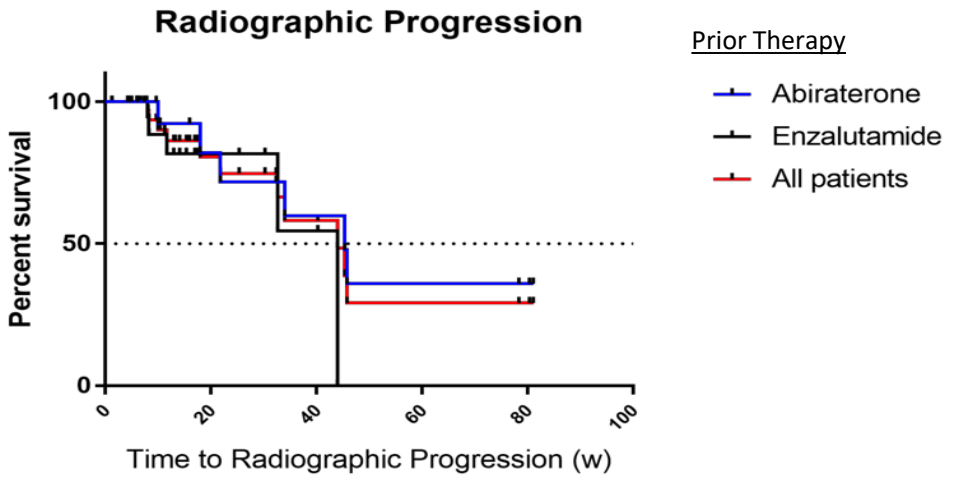
Abiraterone Progressors (Updated September 19, 2018)



Efficacy at well tolerated doses below MTD

Response of ZEN-3694 + enzalutamide in patients progressing on enzalutamide: ZEN-3694 re-sensitizing tumors





Median time to radiographic progression = 10.5 mo., similar for prior abiraterone or enzalutamide therapy

PR = partial response
✖ Progressing radiographically upon study entry
✦ Progressing by PSA upon study entry
△ Progressing clinically upon study entry

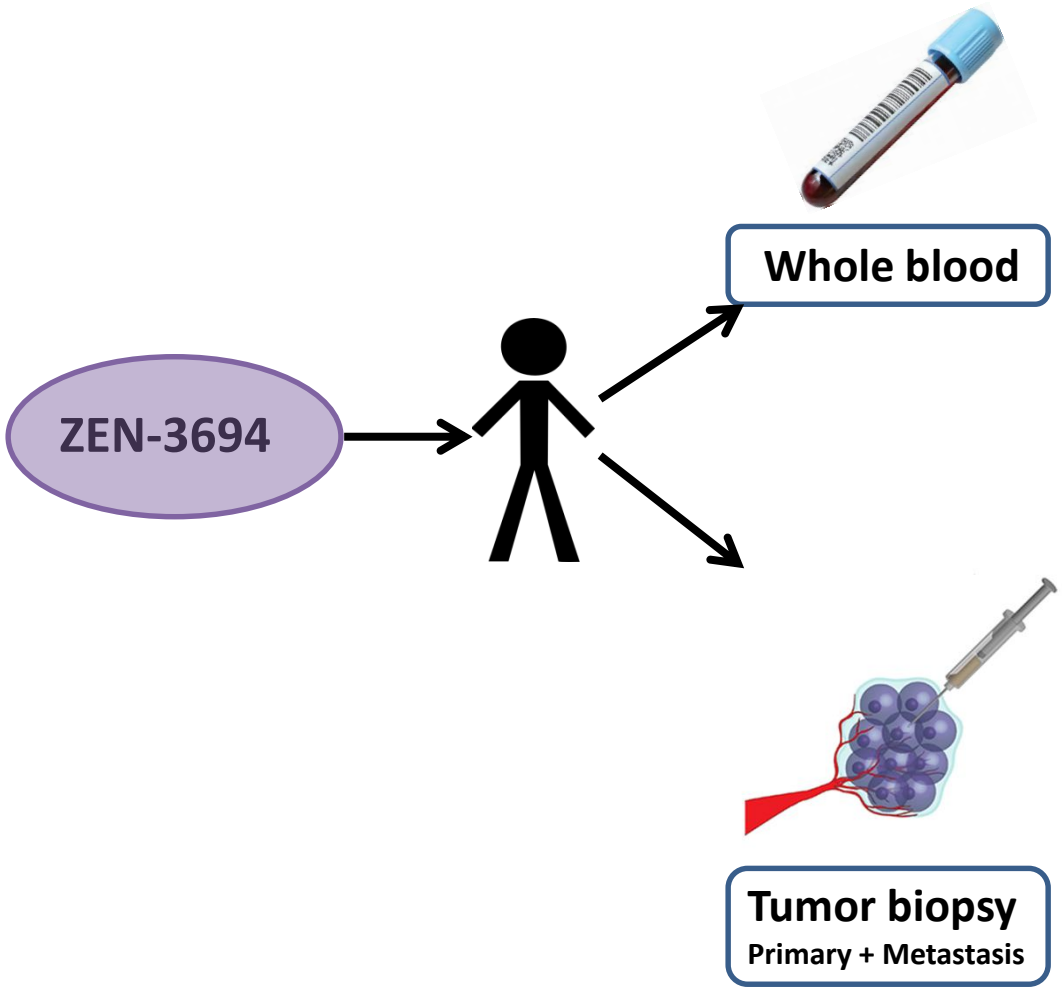
- Expected time to radiographic progression (3-6 mo.) after PSA progression: Attard et al. 2017, PREVAIL study

** Target for ZEN-3694 +enzalutamide, 32 weeks

Swim lanes only shown for patients ongoing and with confirmed radiographic/clinical progression

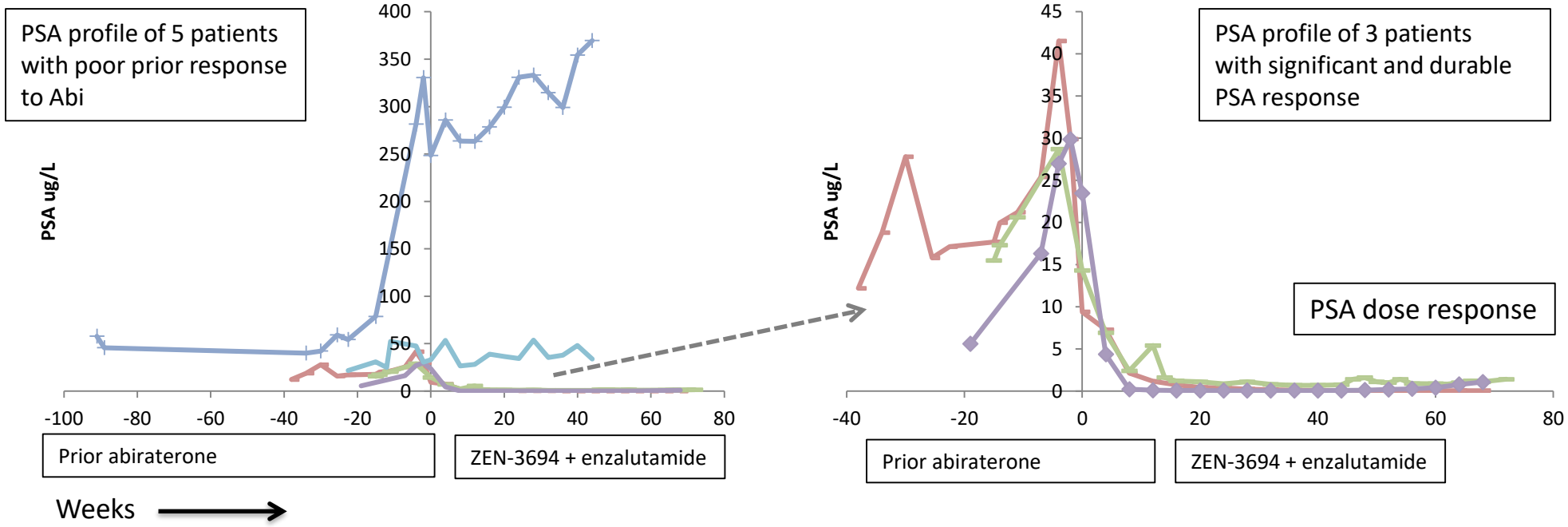
Extensive Translational Medicine Plan
Understand Responders/Non-Responders to Design Future Trials

Translational program designed for molecular profiling ARi resistant patients and effect of ZEN-3694 on resistant markers, potential correlation of response to molecular signature



Circulating tumor cells	• Enumeration, markers, and signatures
Whole blood	• PD marker assay for target modulation • Cancer immune panel • Target engagement
PBMCs Peripheral blood mononuclear cells	• Immuno Oncology markers, Tolerance Markers, T cell receptor sequencing
Plasma	• Exploratory markers and cytokine panel
Immunohistochemistry	• Oncology markers • Histology
RNA-sequencing	• Expressed mutations and profiles • Pathway analysis • Immuno-oncology markers

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



Artificial Intelligence Program Developed Based on Zenith's Clinical Data

FULL PAPER

Artificial Intelligence

ADVANCED THERAPEUTICS
www.advtherap.com

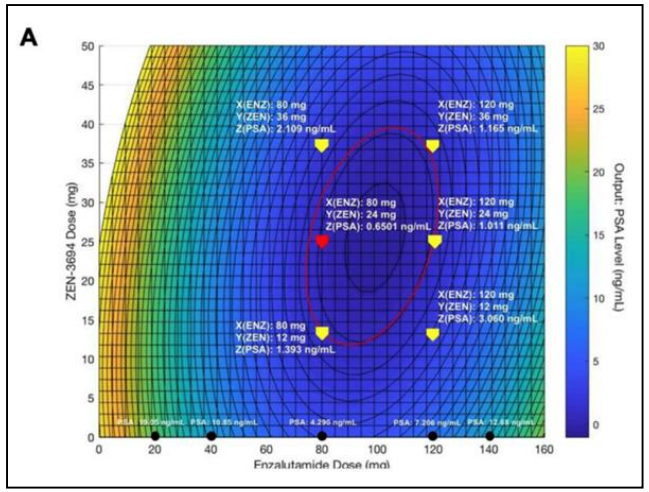
Modulating BET Bromodomain Inhibitor ZEN-3694 and Enzalutamide Combination Dosing in a Metastatic Prostate Cancer Patient Using CURATE.AI, an Artificial Intelligence Platform

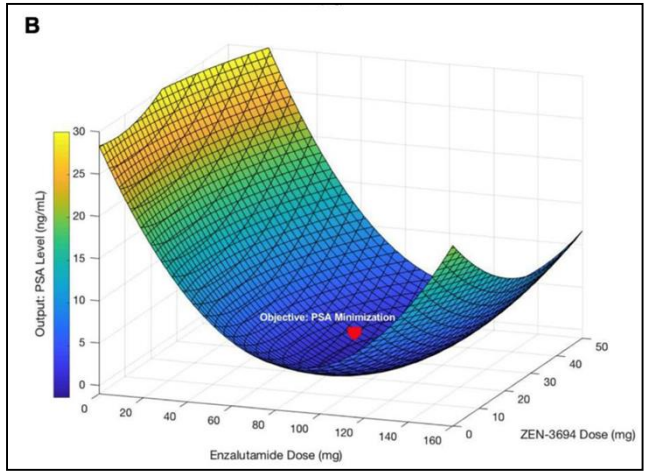
Allan J. Pantuck, Dong-Keun Lee, Theodore Kee, Peter Wang, Sanjay Lakhotia, Michael H. Silverman, Colleen Mathis, Alexandra Drakaki, Arie S. Belldegrun, Chih-Ming Ho,* and Dean Ho**

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.

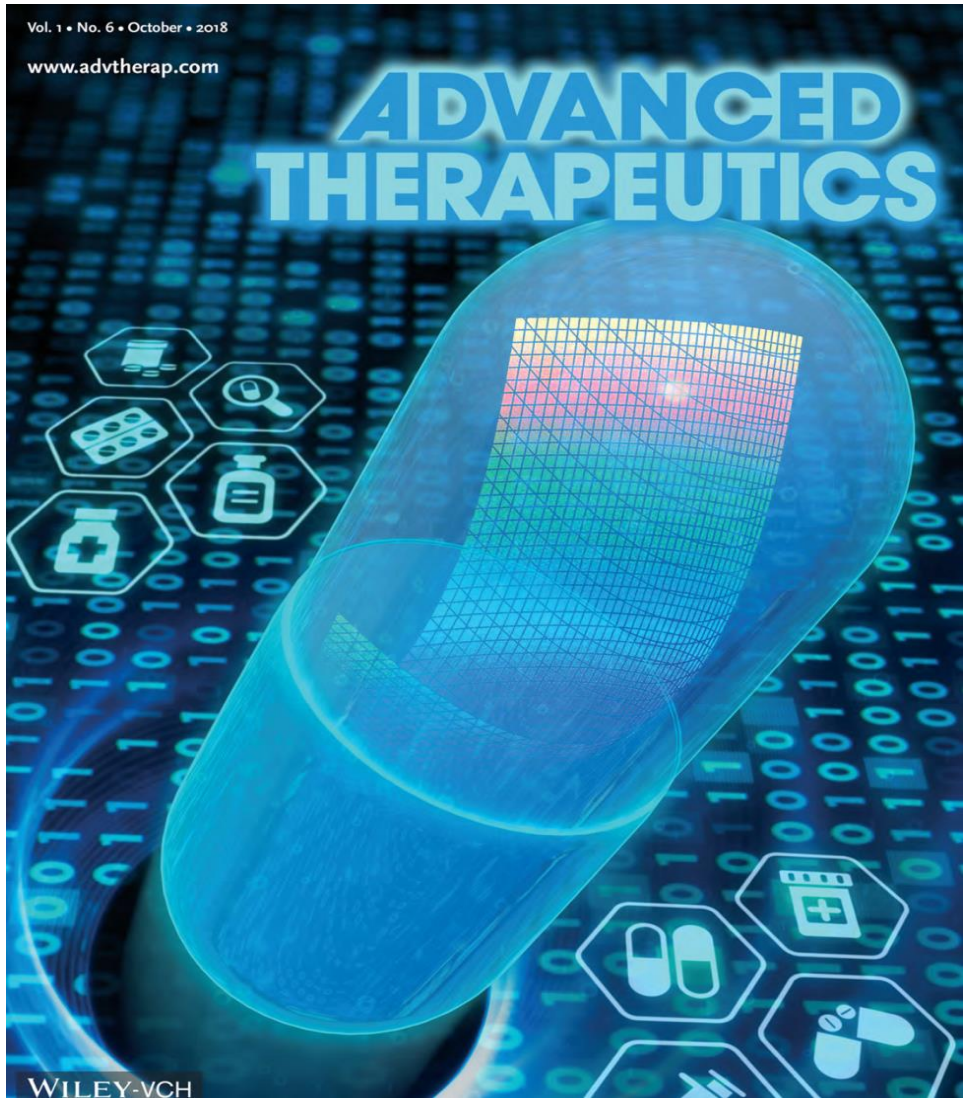
1. Introduction

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

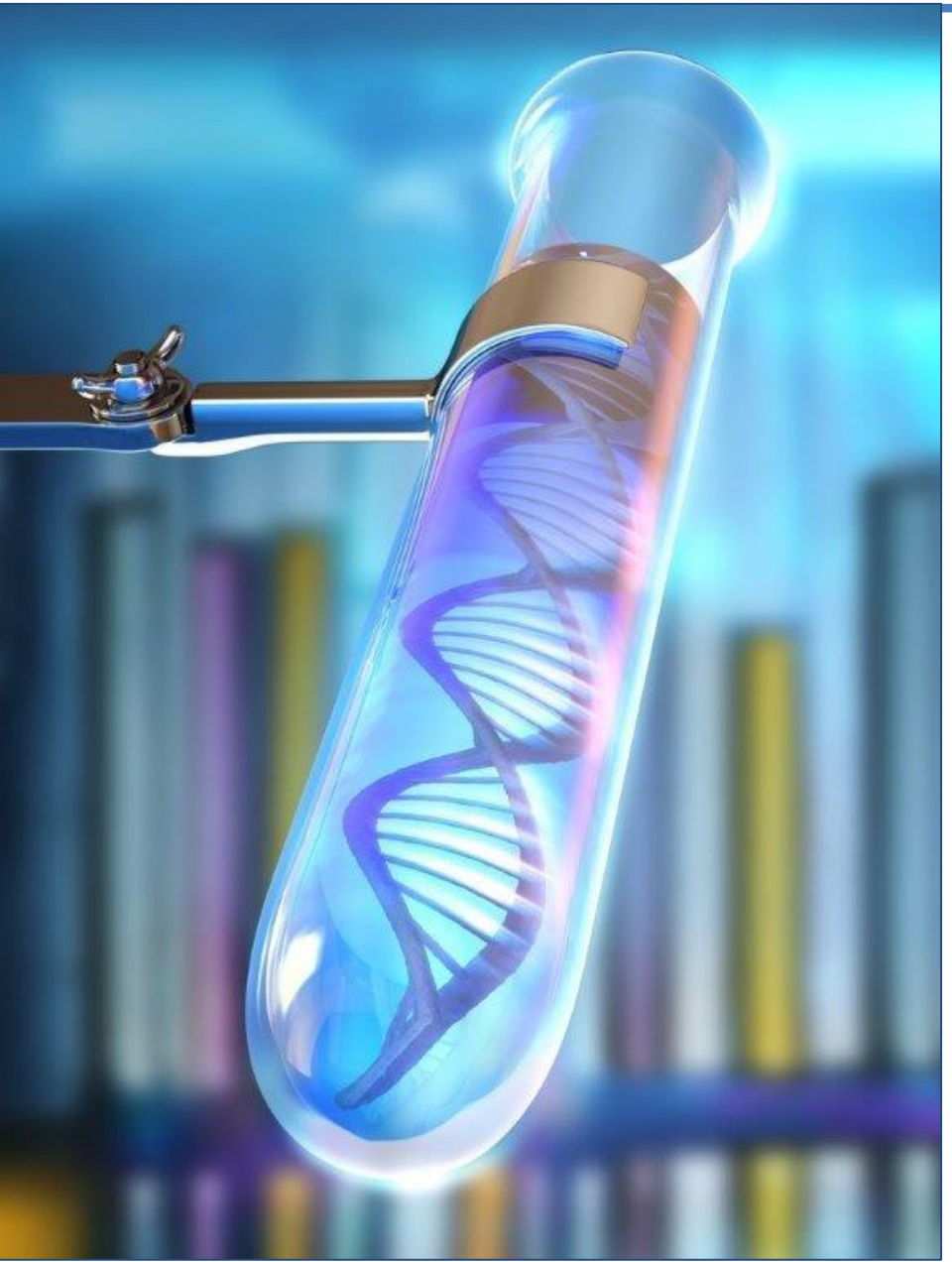




Recent Zenith Publication Covers



Todays Agenda for Zenith Capital Corp.



- Epigenetics – The Mechanism Behind Our Approach
- The Potential of BET Inhibition
- Prostate Cancer Rationale Review
- Promising Early Clinical Results & Case studies
- **Next Steps for Zenith**

Strong Rational for BETi/PARPi Combination in TNBC

Combination of BETi/PARPi is an innovative strategy to overcome intrinsic and acquired resistance to PARP inhibition, allowing for use of PARP inhibitors across a wider spectrum of cancers and use past the initial development of resistance to PARPi

Sensitize cancers with primary resistance

(HR-proficient cancers)

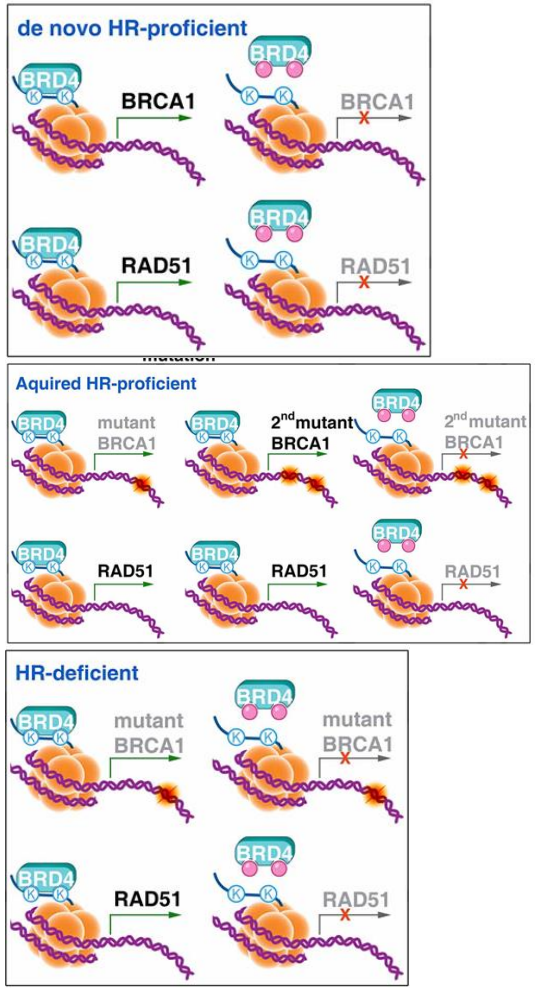
- Repressed BET activity impairs BRCA1 and RAD51 expression, subsequently converting HR-proficient tumors to HR-deficient ones

Re-sensitize tumors with acquired HR-proficiency to treatment with PARPi

- BET inhibition overcomes PARPi resistance by repressing the expression of secondary BRCA1 mutations that restore BRCA1 function, or by blocking the expression of BRCA1 and RAD51

Enhance response to PARPi in tumors with HR-deficient cancer (BRCA1/2 mutated)

- BET inhibition further blocks RAD51 expression



ZEN-3694 Combination with a PARP Inhibitor in TNBC: Efficient POC Study

2018	2019	2020

Phase 1b/2: Combination with PARPi in TNBC non gBRCA1/2m
N~50

Objective	• Show safety and activity of the combination in TNBC patients
Study design	• Phase 1b dose escalation • Phase 2 Simon two step , open label non randomized
Patient Population	• TNBC: non germline BRCA1/2m, advanced metastatic, $\leq$ 3 prior chemo therapy regimen, ER<10%, PR<10% and HER2-negative by IHC and/or FISH
Number of patients (N)	• N~ 9-12 for Dose escalation • Simon 2-stage design • H_0 TNBC = 20% ORR, Target ORR = 40%, N= 17 1st stage, N= 17 for 1st stage, progress to second stage if number of responders $\geq$ 4, N=20 for second stage, 10% alpha, 90% power
Dose	• ZEN-3694 starting dose: 72mg once daily
Duration	• 6 months for dose escalation; 12 months for expansion cohorts (assuming 10 clinical sites)
Endpoints	• Phase 1b: Safety, PK/PD, MTD, RP2D • Phase 2 TNBC: ORR, DOR, PFS • Exploratory: Explore biomarkers of activity and resistance

Prominent Clinical Sites and Investigators

Institution	Investigator	Background
MSKCC	Mark Robson	Led Olympiad study for Astra Zeneca
MD Anderson	Jennifer Litton	Led Embraca Ph. 3 study for Pfizer
University of Kansas	Priyanka Sharma	TNBC specialist
University of Pennsylvania	Susan Domcheck	Breast cancer specialist
Banner Health MD Anderson Cancer Center	Lida Mina	Breast cancer specialist
Jules Bordet (Belgium)	Philippe Afthimos	Led Merck's and BI's BETi studies
VHIO (Spain)	Mafalda Olivera	Involved in ER+ BETi trials for Gilead and GSK
CIOCC (Spain)	Valentina Boni	Breast cancer specialist
UZ Leuven (Belgium)	Kevin Punie	Breast cancer specialist

Opportunity in Immuno Oncology: Strong Rationale for Checkpoint Combinations

ZENITH EPIGENETICS

Cell Reports

Article

BET-Bromodomain Inhibitors Engage the Host Immune System and Regulate Expression of the Immune Checkpoint Ligand PD-L1

BET bromodomain inhibition enhances T cell persistence and function in adoptive immunotherapy models

Yuki Kagoya,[1] Munehide Nakatsugawa,[1] Yuki Yamashita,[1] Toshiki Ochi,[1] Tingxi Guo,[1,2] Mark Anczurowski,[1,2] Kayoko Saso,[1] Marcus O. Butler,[1,2,3] Cheryl H. Arrowsmith,[4,5] and Naoto Hirano[1,2]

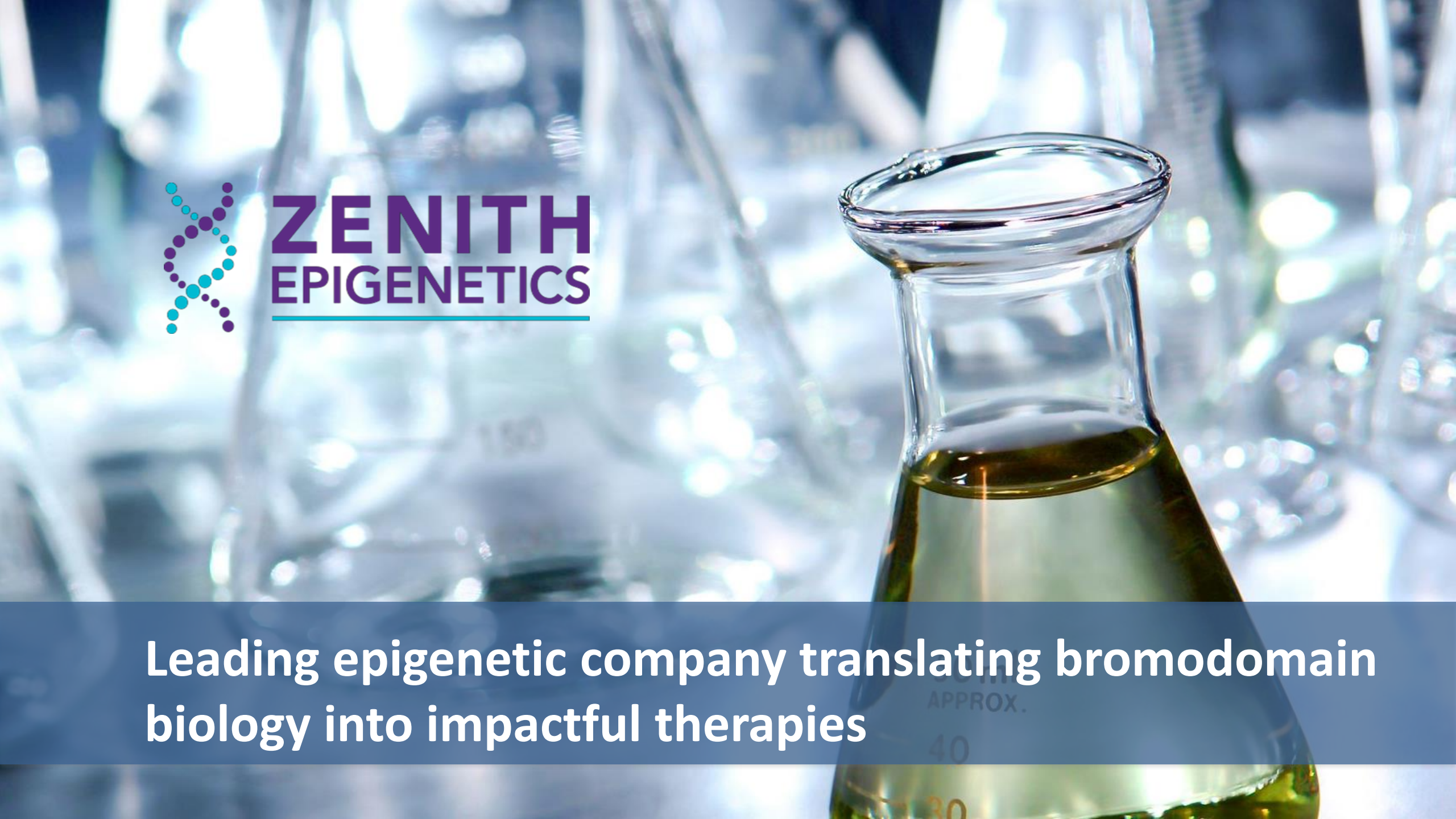
[1]Tumor Immunotherapy Program, Campbell Family Institute for Breast Cancer Research, Campbell Family Cancer Research Institute, Princess Margaret Cancer Centre, University Health Network, Toronto, Ontario, Canada. [2]Department of Immunology, University of Toronto, Toronto, Ontario, Canada. [3]Department of Medicine and [4]Structural Genomics Consortium and Department of Medical Biophysics, University of Toronto, Toronto, Ontario, Canada. [5]Princess Margaret Cancer Centre, University Health Network, Toronto, Ontario, Canada.

BET bromodomain inhibition cooperates with PD-1 blockade to facilitate antitumor response in Kras-mutant non-small cell lung cancer. Adeegbe DO, et al. Cancer Immunol Res. 2018

Summary

Zenith is focused on ZEN-3694 combinations with SOC extending and expanding the value of existing therapeutics

- **ZEN-3694 can be administered safely at doses that modulate BET targets**
- **Prostate/XTANDI** combination: Promising clinical activity of ZEN-3694 + Enzalutamide in ARi resistant mCRPC patients
- **TNBC/PARPi** combination: Ph. 1b/2 of ZEN-3694 + PARPi in TNBC (non germline-BRCA1/2m) to commence soon
- **PD-1/PD-L1** combination with ZEN-3694 has compelling pre-clinical and clinical rationale
- **ER+ Breast Cancer:** Preclinical rationale to address resistance to CDK4/6 inhibitors



Leading epigenetic company translating bromodomain biology into impactful therapies