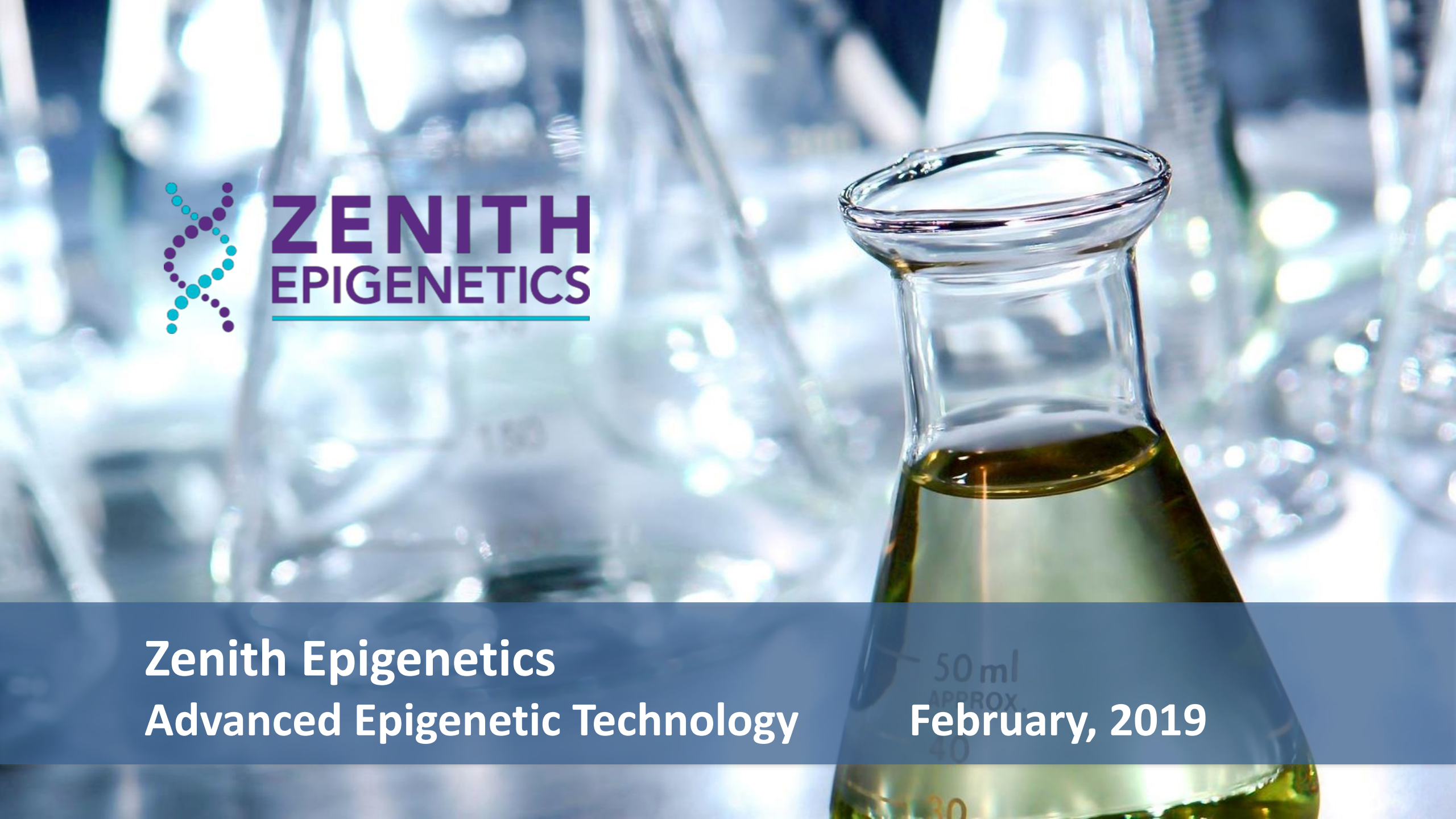


ZENITH EPIGENETICS

Zenith Epigenetics

Advanced Epigenetic Technology

February, 2019

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 Leading to Pfizer Collaboration

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. (TSX: RVX)

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

2016 — Dec 2016 - Initiated combination study with Enzalutamide in mCRPC patients

2017 — June 2017 - Announced 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

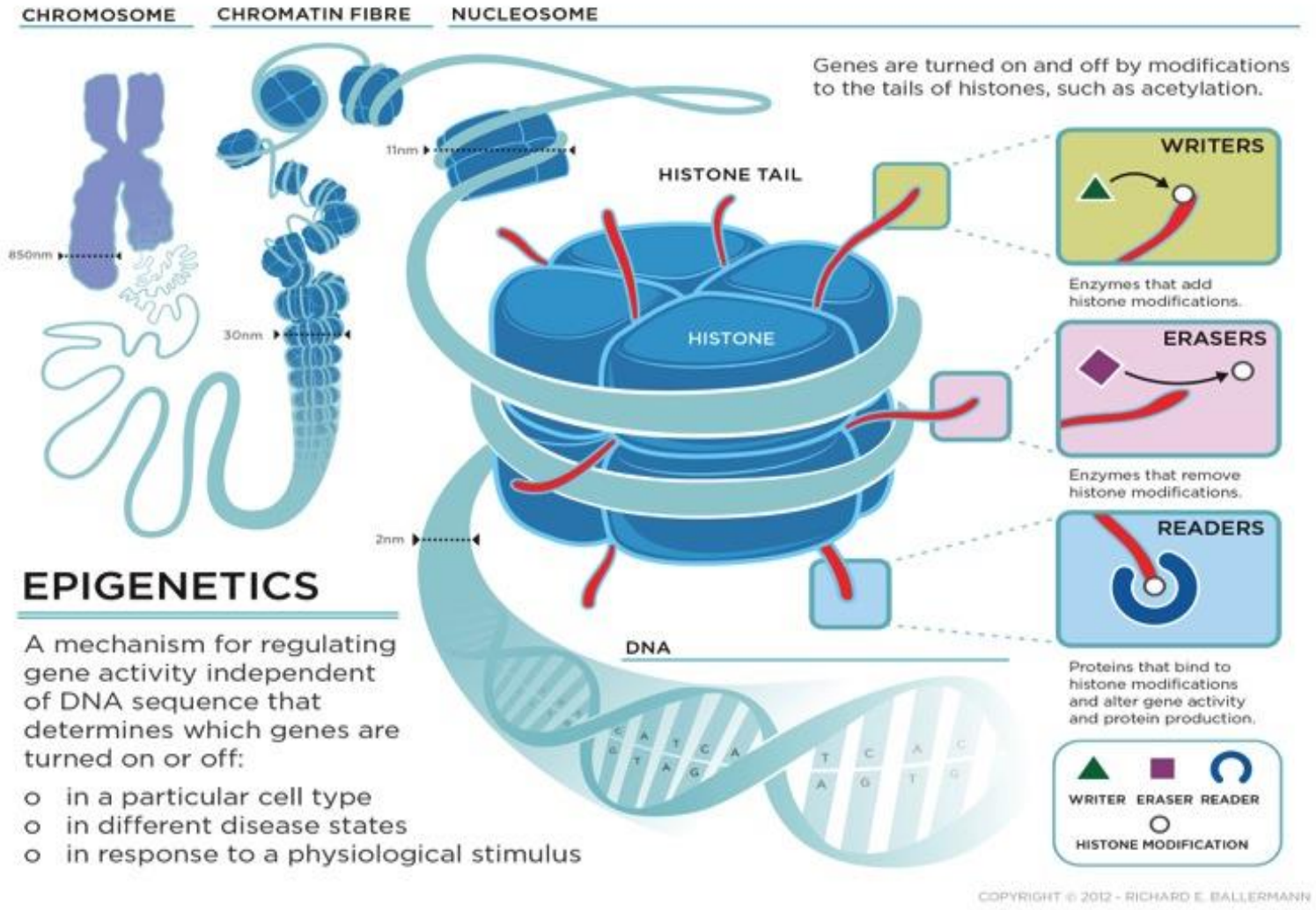
Nov. 20/'18 - Pfizer clinical trial collaboration announced

Advancing Development Pipeline

- Significant development progress with our lead product, **ZEN-3694**, a bromodomain and extra terminal domain inhibitor (**BETi**), **currently in clinical development** for combination treatment of solid tumors, including **prostate** and **breast cancer**

Indication	2019	2020
Metastatic Castration-resistant Prostate Cancer (mCRPC)	Combination expansion ZEN - 3694 + enzalutamide; Patients progressed on ***abiraterone*** (N~15) or ***enzalutamide*** (N~25)	
Pfizer Collaboration: Triple Negative Breast Cancer (TNBC)	Phase 1b/2: Combination with PARPi in TNBC (N~50)	

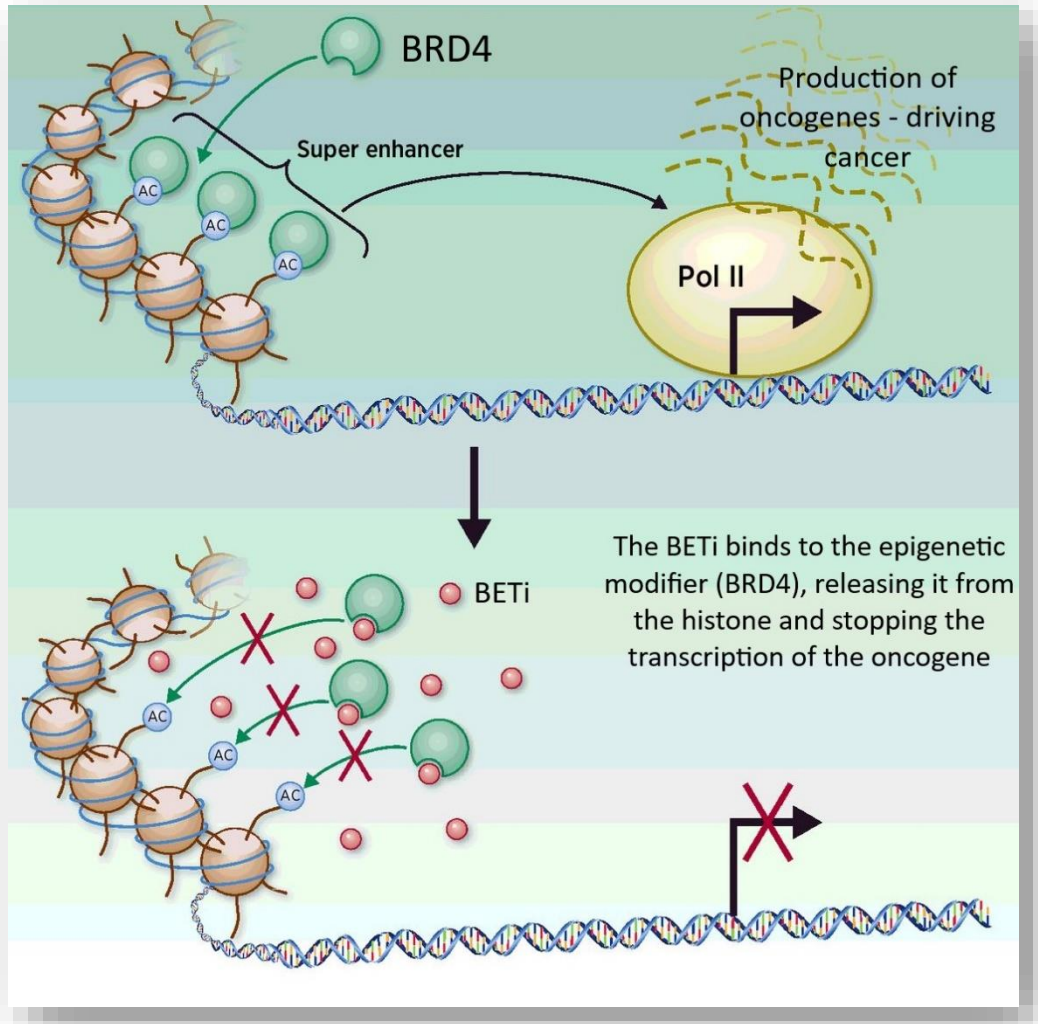
Epigenetics - The Mechanism Behind Our Approach



- 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



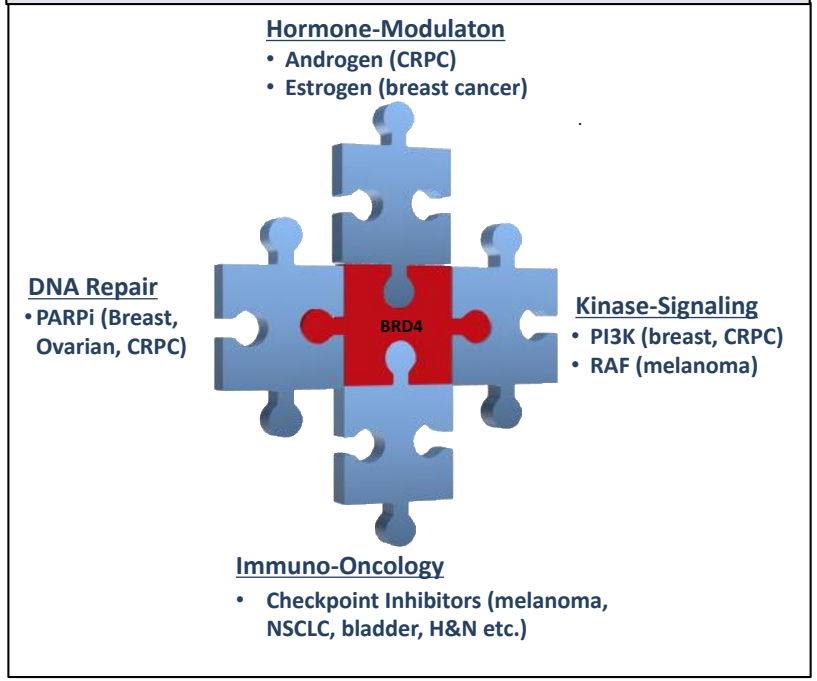
Adopted from Clinical Cancer Research 2017, 23(7), 1647-55.

- Many of the escape resistance mechanisms to standard of care treatments involve BRD4
- BETi blocks BRD4 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

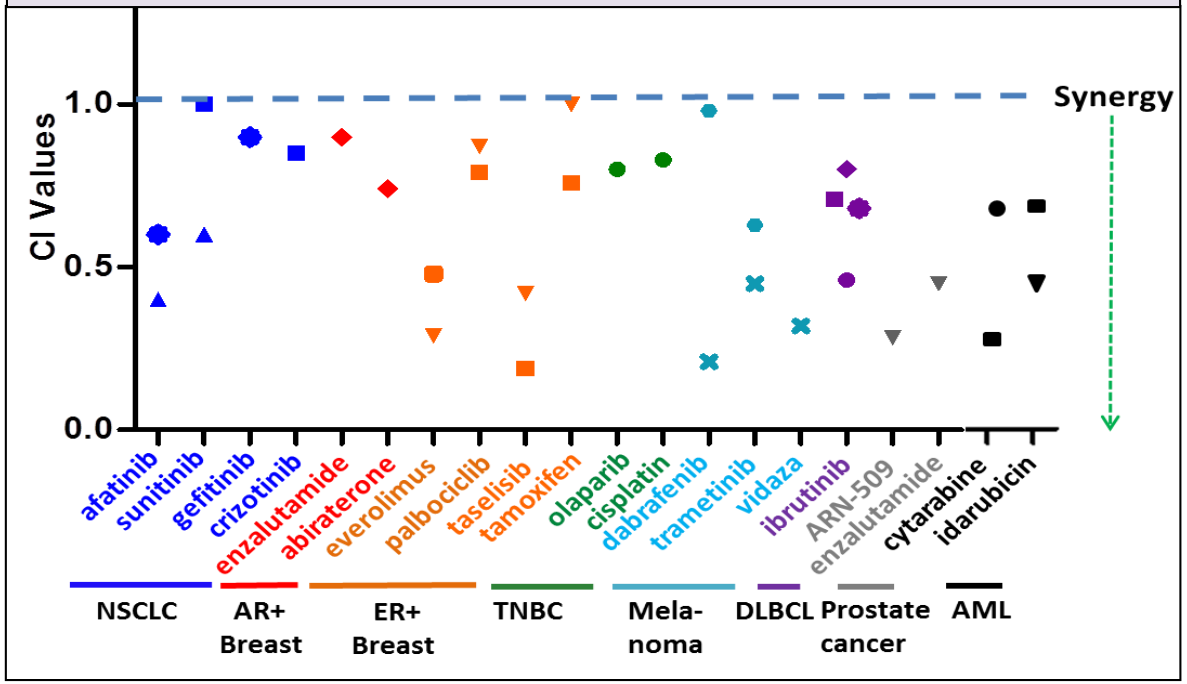
Combination Therapy: The Potential of BET Inhibition and ZEN-3694

- BET inhibitors have 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

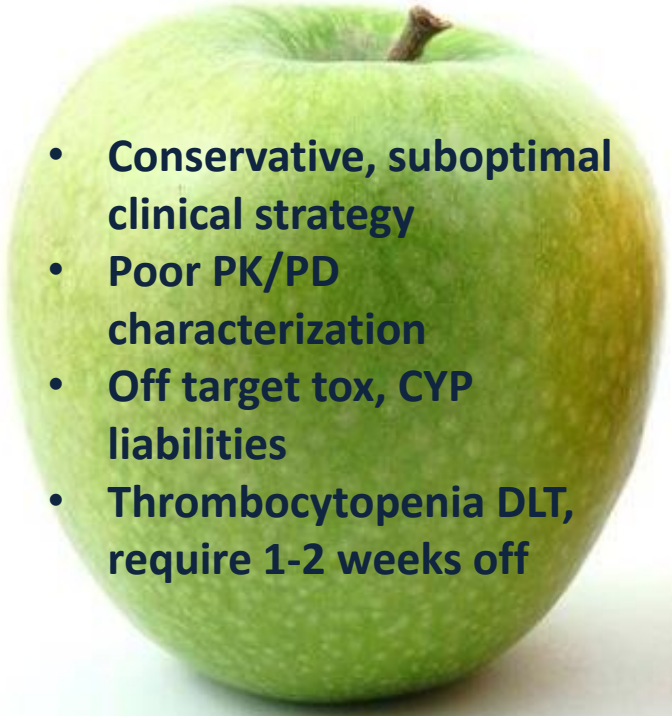


ZEN-3694 synergizes with several standard of care cancer drugs



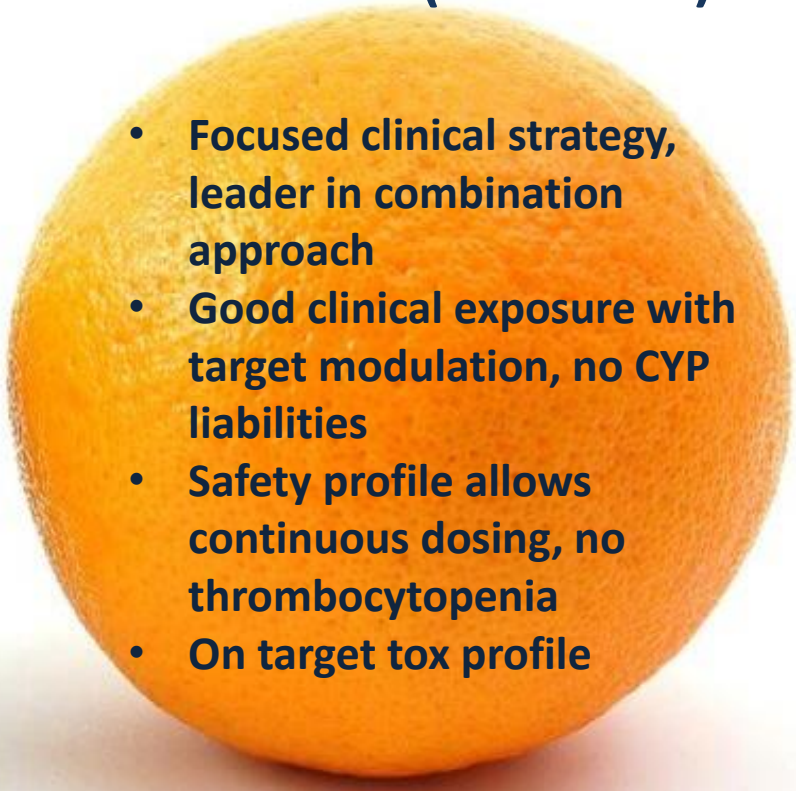
Zenith's BETi program is Clinically Differentiated

Other Clinical BETi



- Conservative, suboptimal clinical strategy
- Poor PK/PD characterization
- Off target tox, CYP liabilities
- Thrombocytopenia DLT, require 1-2 weeks off

Zenith's BETi (ZEN-3694)

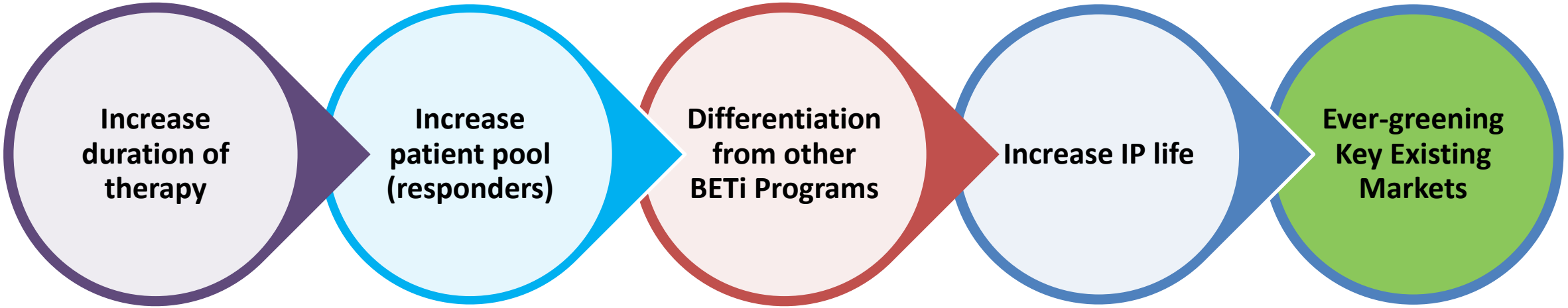


- Focused clinical strategy, leader in combination approach
- Good clinical exposure with target modulation, no CYP liabilities
- Safety profile allows continuous dosing, no thrombocytopenia
- On target tox profile

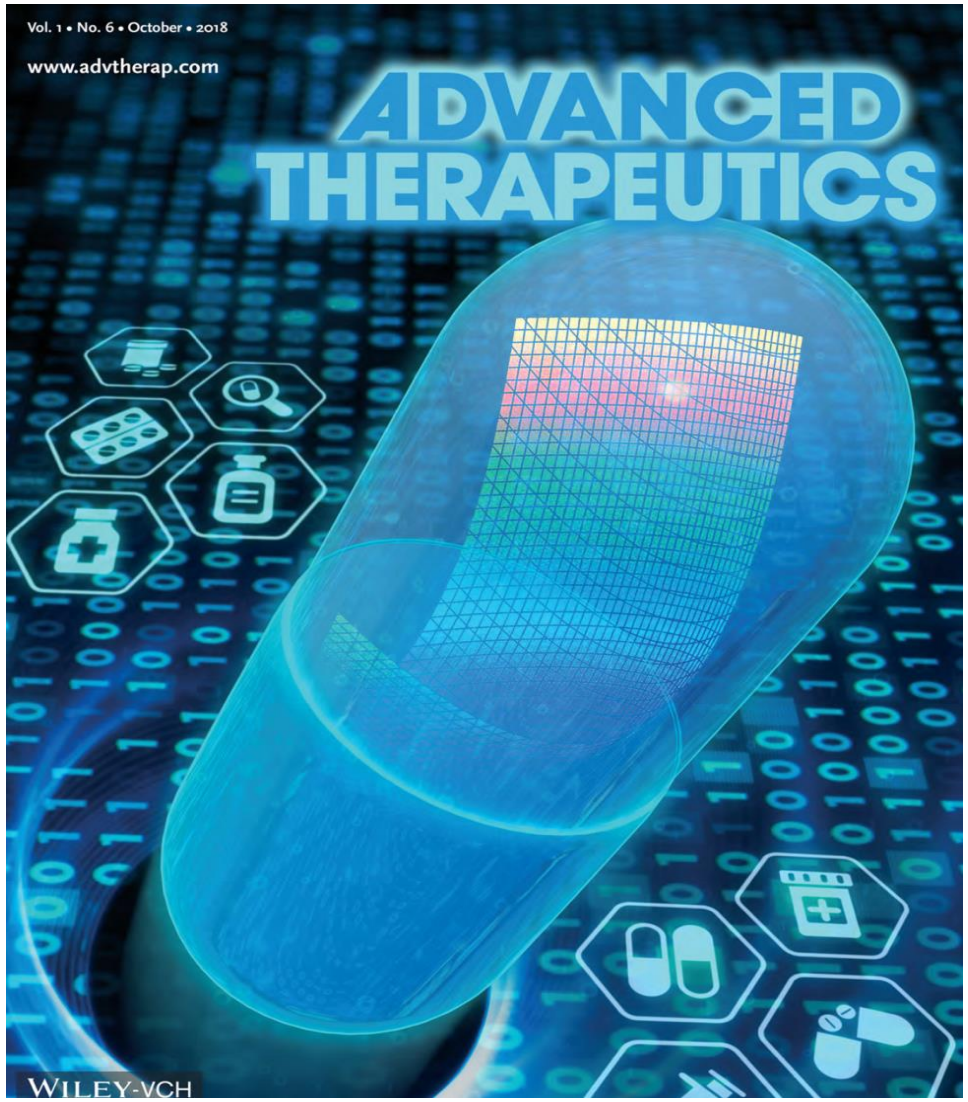
Our Synergistic Approach – Making Great Drugs Work Better & Longer

- Combination Therapy with ZEN-3694 represents a multi-$Bln addressable market

Current markets include: AR antagonists, PD-1/Pd-L1Mabs, CDK 4/6 inh., PARP inhibitors



Recent Zenith Publication Covers



Validation of Artificial Intelligence Program with Zenith's Clinical Data

FULL PAPER

Artificial Intelligence

ADVANCED THERAPEUTICS
www.advtherap.com

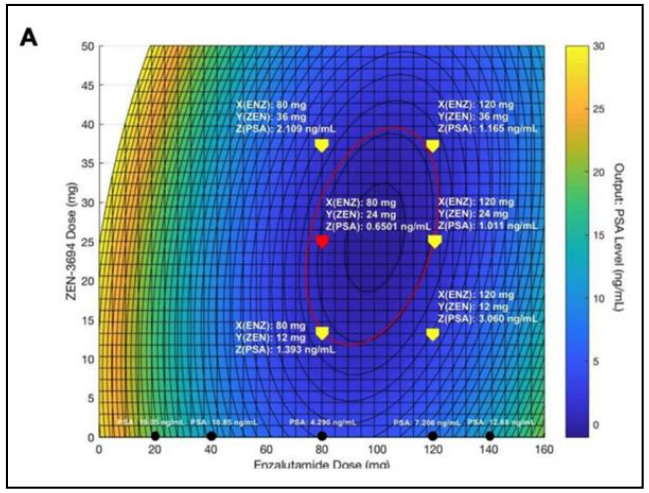
Modulating BET Bromodomain Inhibitor ZEN-3694 and Enzalutamide Combination Dosing in a Metastatic Prostate Cancer Patient Using CURATE.AI, an Artificial Intelligence Platform

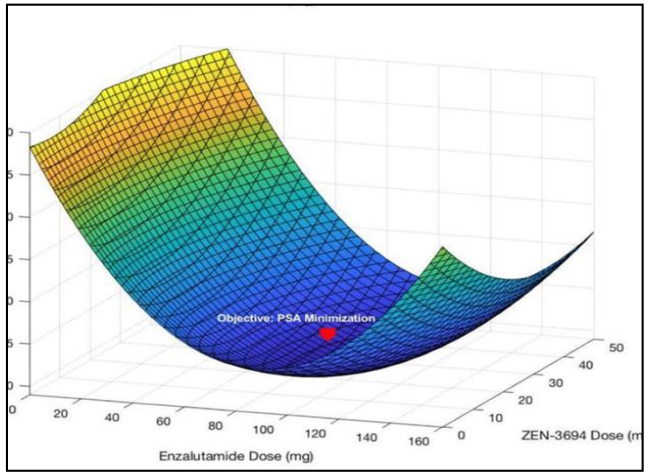
Allan J. Pantuck, Dong-Keun Lee, Theodore Kee, Peter Wang, Sanjay Lakhotia, Michael H. Silverman, Colleen Mathis, Alexandra Drakaki, Arie S. Belldegrun, Chih-Ming Ho,* and Dean Ho**

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





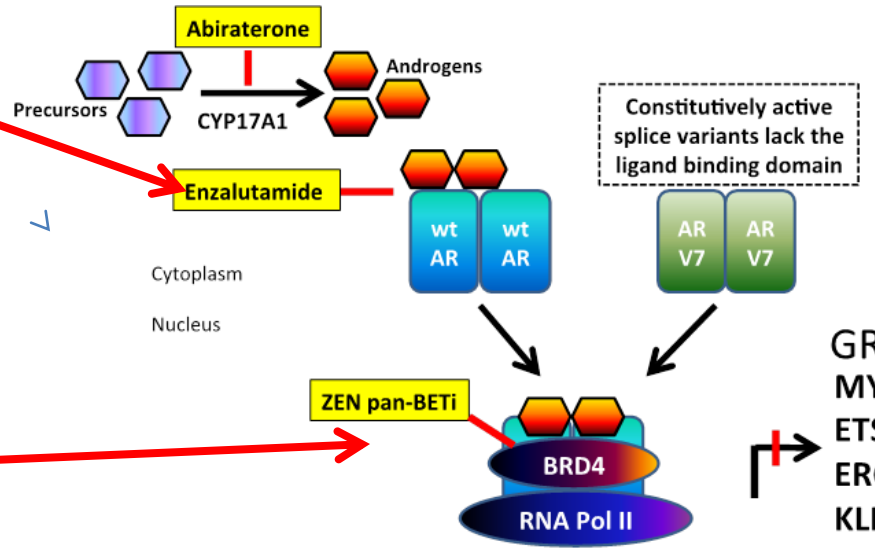
Prostate Cancer Program Review

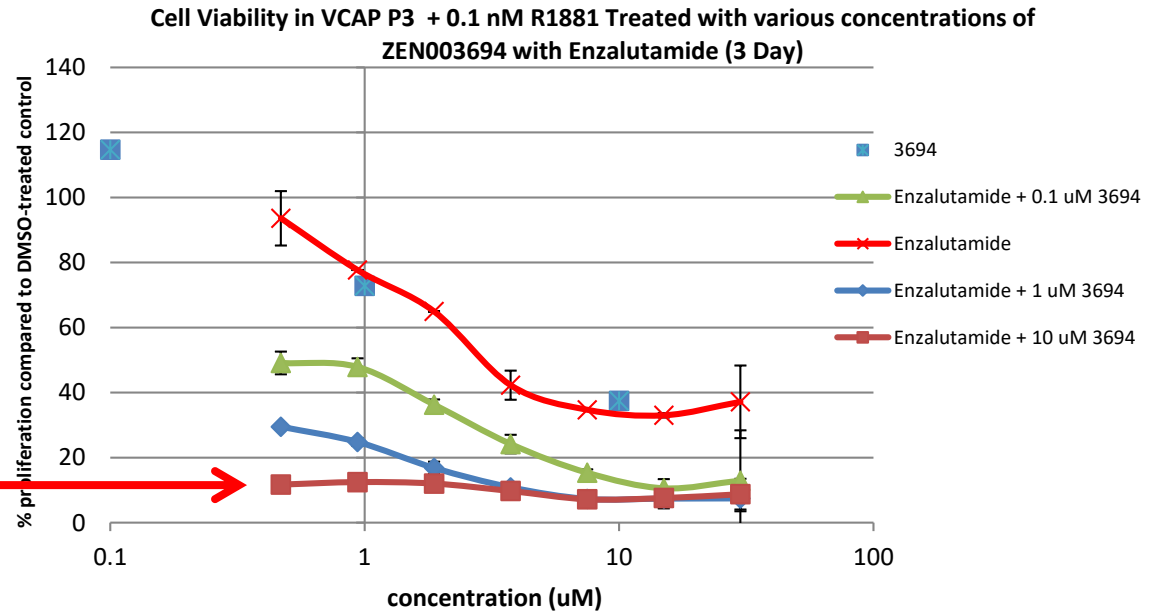
ZEN-3694 Potential in Patients Developing mCRPC Resistance to Enzalutamide

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





Prostate Cancer Program Review

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

Prostate Cancer Program Review

Phase 2 Ongoing; Phase 1b Completed

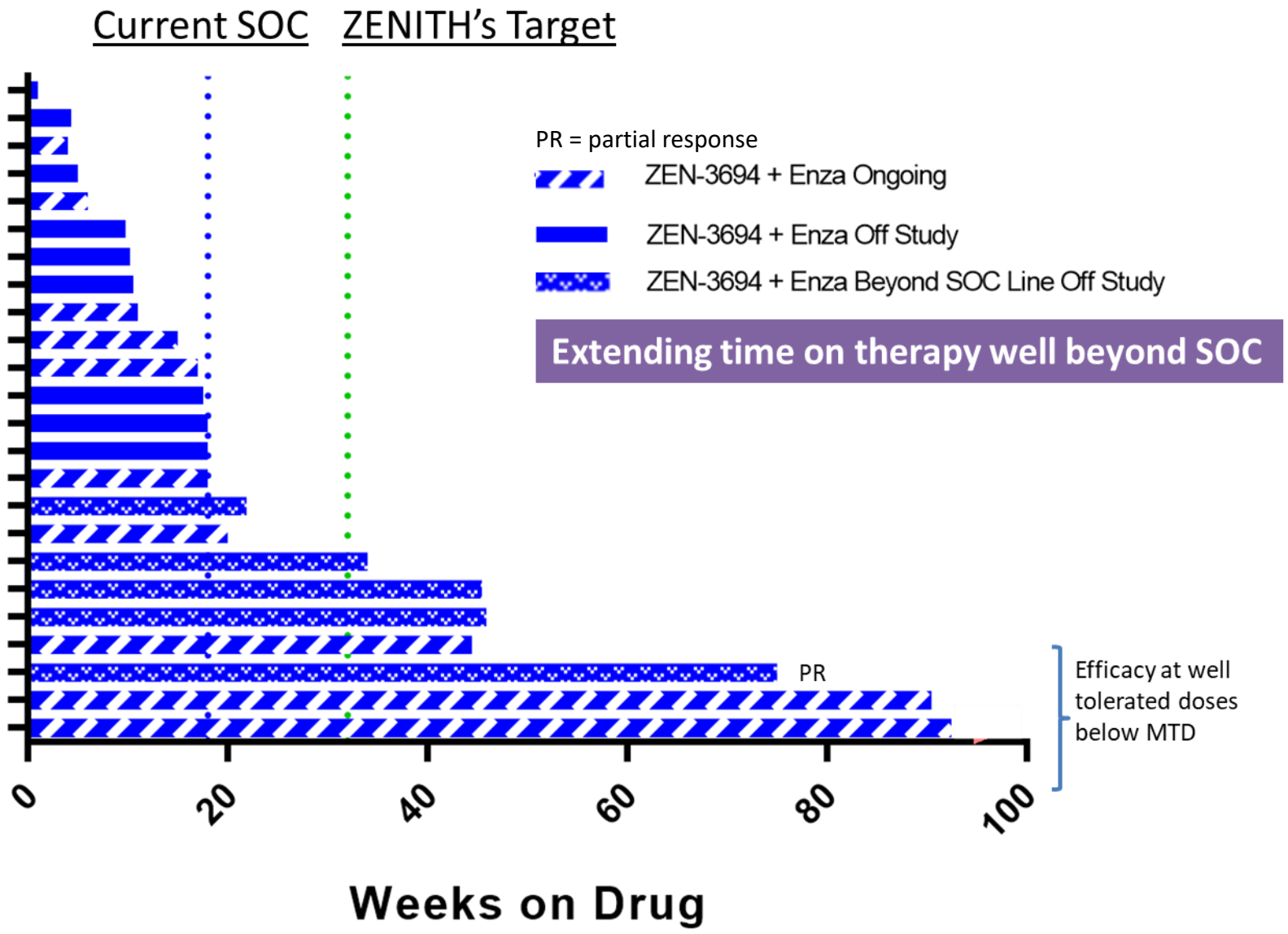
Indication	2019	2020
Metastatic Castration-resistant Prostate Cancer (mCRPC)	Combination expansion ZEN - 3694 + enzalutamide; Patients progressed on ***abiraterone*** (N~15) or ***enzalutamide*** (N~25)	

Study Summary

- Dose escalation completed, 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)
- **~90 patients dosed to date in this study**

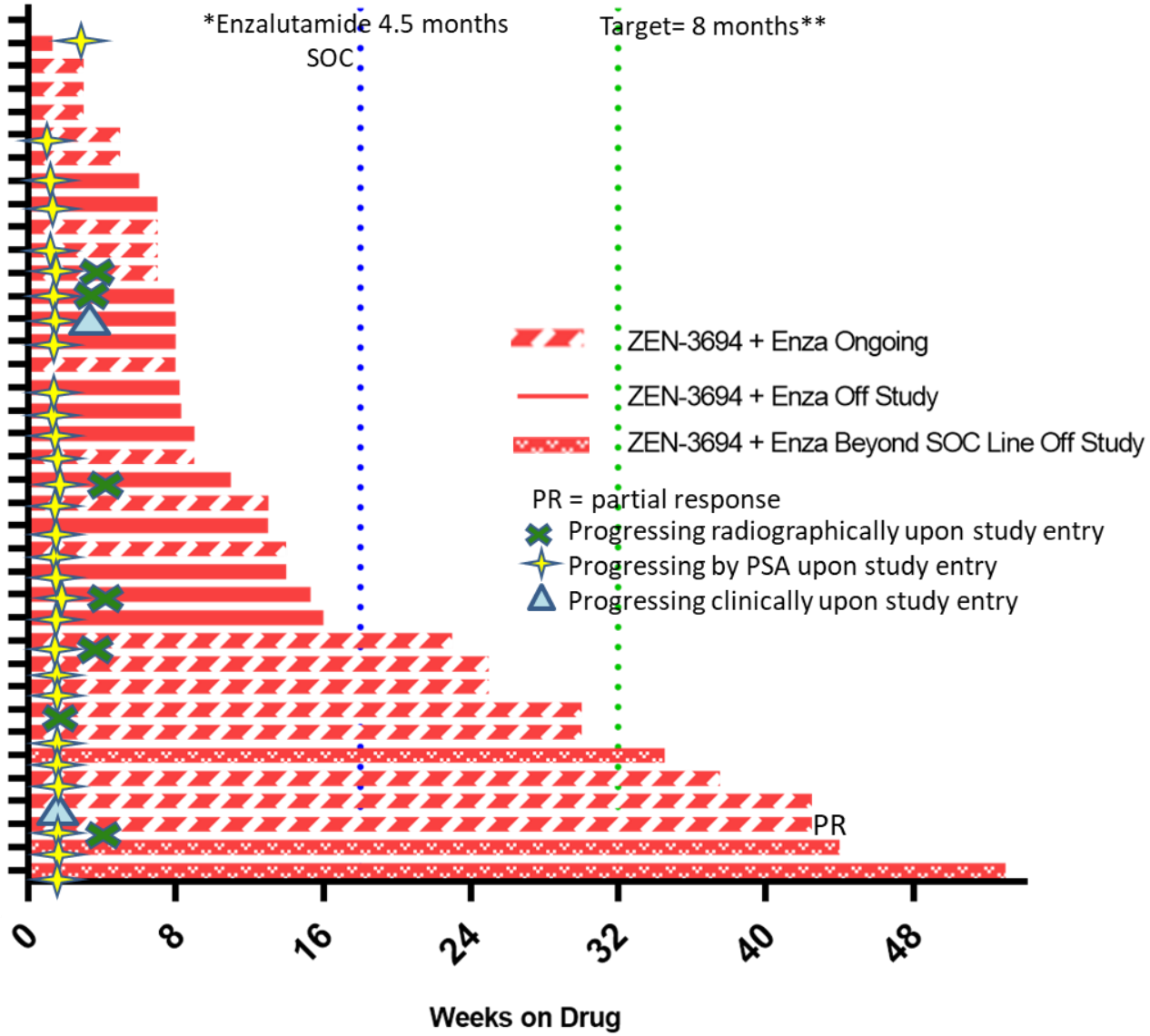
Prostate Cancer Program Review: Combination Study Update

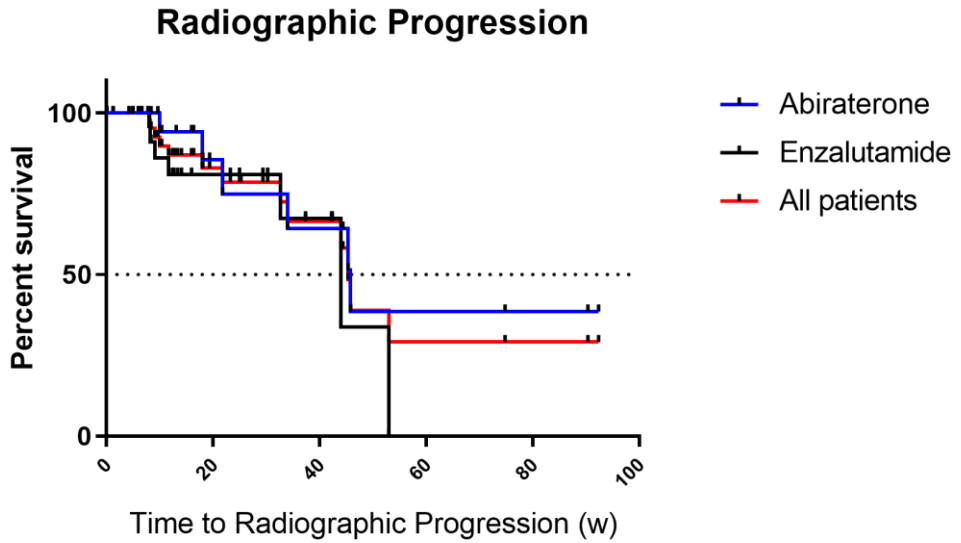
Abiraterone Progressors (Updated January 3, 2019)



Prostate Cancer Program Review: Promising Data

Duration on ZEN-3694 + Enzalutamide by Patients that Progressed on Enzalutamide





Median time to radiographic progression = 10.2 mo., similar for prior abiraterone or enzalutamide therapy

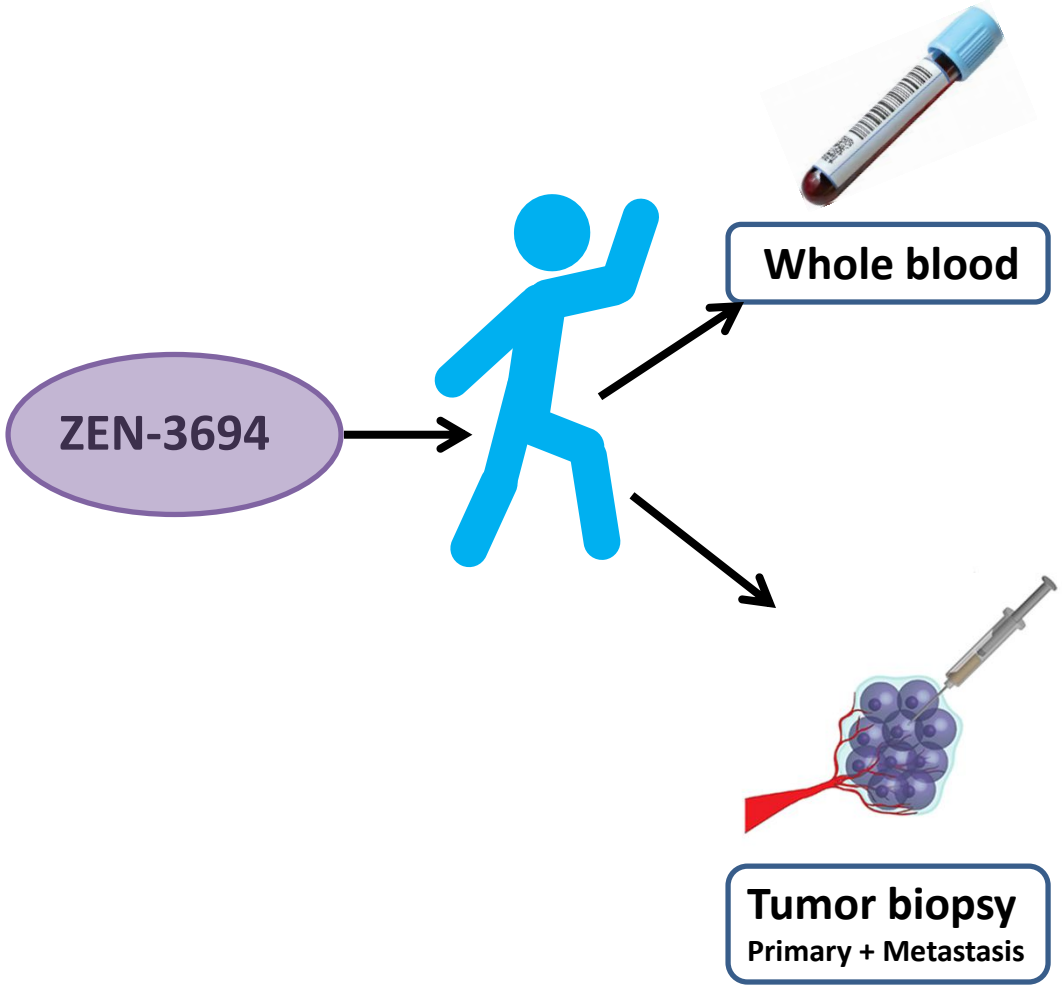
- Expected time to radiographic progression (3-6 mo.) Attard et al. 2017

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

Prostate Cancer Program: 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

Pfizer / Zenith Clinical Trial Collaboration

Zenith Epigenetics Announces Clinical Trial Collaboration with Pfizer

November 20, 2018

Collaboration to evaluate ZEN-3694 in combination with Talazoparib in TNBC patients; Phase 1b/2 trial expected to initiate 1Q 2019

CALGARY, Alberta, Nov. 20, 2018 (GLOBE NEWSWIRE) -- Zenith Epigenetics Ltd. ("Zenith" or the "Company"), a wholly-owned subsidiary of Zenith Capital Corp., announced today that it has entered into a clinical trial collaboration with Pfizer Inc. ("Pfizer"; NYSE: PFE) to evaluate the safety and efficacy of a novel anti-cancer combination of Zenith's investigational bromodomain and extra-terminal domain inhibitor ("BETi"), ZEN-3694, and Pfizer's poly ADP ribose polymerase inhibitor ("PARPi"), talazoparib, in patients with locally advanced or metastatic triple negative breast cancer ("TNBC").

"Zenith is excited to announce this partnership with Pfizer, a leader in oncology," said Don McCaffrey, Chief Executive Officer of Zenith. "This novel approach of combining a BETi and a PARPi in patients who do not have inherited BRCA gene mutations may prove to significantly increase the potential of PARP inhibition in different indications, with an initial focus on triple negative breast cancer."

Preclinical data indicate that combining talazoparib with ZEN-3694 is a rational combination to test in patients that are proficient in homologous DNA repair. BETi have been shown pre-clinically to modulate homologous DNA repair genes and can thus potentially sensitize BRCA1/2 proficient patients to talazoparib.

Under the terms of the agreement, Zenith Epigenetics and Pfizer will collaborate on a Phase 1b/2 TNBC clinical study. Pfizer will provide talazoparib, Zenith will provide ZEN-3694, and both parties will fund the study. Zenith Epigenetics retains all rights to ZEN-3694.

Pfizer / Zenith Clinical Trial Collaboration Summary

Indication	2019	2020
Pfizer Collaboration: Triple Negative Breast Cancer (TNBC)	Phase 1b/2: Combination with PARPi in TNBC (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_o 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

Pfizer / Zenith Clinical Trial Collaboration

Strong Rational for BETi/PARPi Combination Therapy

ZENITH EPIGENETICS

SCIENCE TRANSLATIONAL MEDICINE | RESEARCH ARTICLE

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

Pfizer / Zenith Clinical Trial Collaboration

Prominent Clinical Sites and Investigators

Institution	Investigator	Background
MSKCC	Mark Robson – Study Lead PI Ayca Gucalp - PI	Led OlympiAD trial
MD Anderson	Jennifer Litton	Led EMBRACA trial
Banner Health	Lida Mina	Investigator on Phase 1, 2 and 3 Talazoparib trials
University of Kansas	Priyanka Sharma	TNBC specialist
University of Penn	Payal Shah - PI (Susan Domchek)	Talazoparib investigator, breast cancer specialist
Sarah Cannon	Erika Hamilton	Breast cancer specialist
Jules Bordet, Belgium	Philippe Aftimos	Led Merck and BI BETi trials
UZ Leuven, Belgium	Kevin Punie	Breast cancer specialist
VHIO, Spain	Mafalda Oliveira	Investigator on Gilead and GSK ER+ BETi trials
StartMadrid, Spain	Valentina Boni	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]

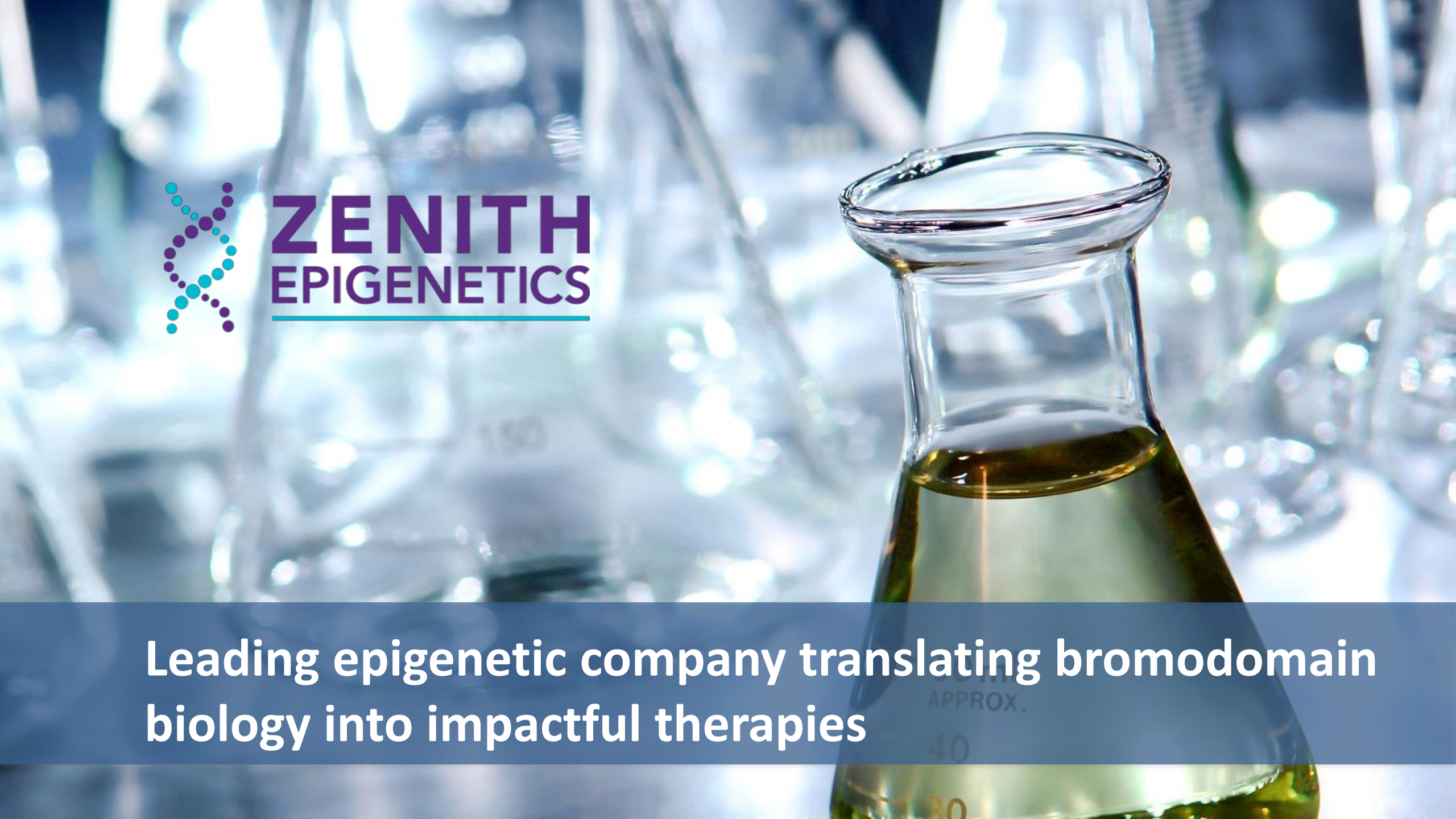
[1]Tumor Immunotherapy Program, Campbell Family Institute for Breast Cancer Research, Campbell Family Cancer Research Institute, Princess Margaret Cancer Centre, University Health Network, Toronto, Ontario, Canada. [2]Department of Immunology, University of Toronto, Toronto, Ontario, Canada. [3]Department 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
- **Pfizer and Zenith collaboration (TNBC/PARPi):** 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