TWO NOVEL TARGETED THERAPIES YIELDING A DEEP ONCOLOGY CLINICAL PIPELINE

Prescient Therapeutics Limited (ASX: PTX)
Biotech Showcase, San Francisco
January 2017

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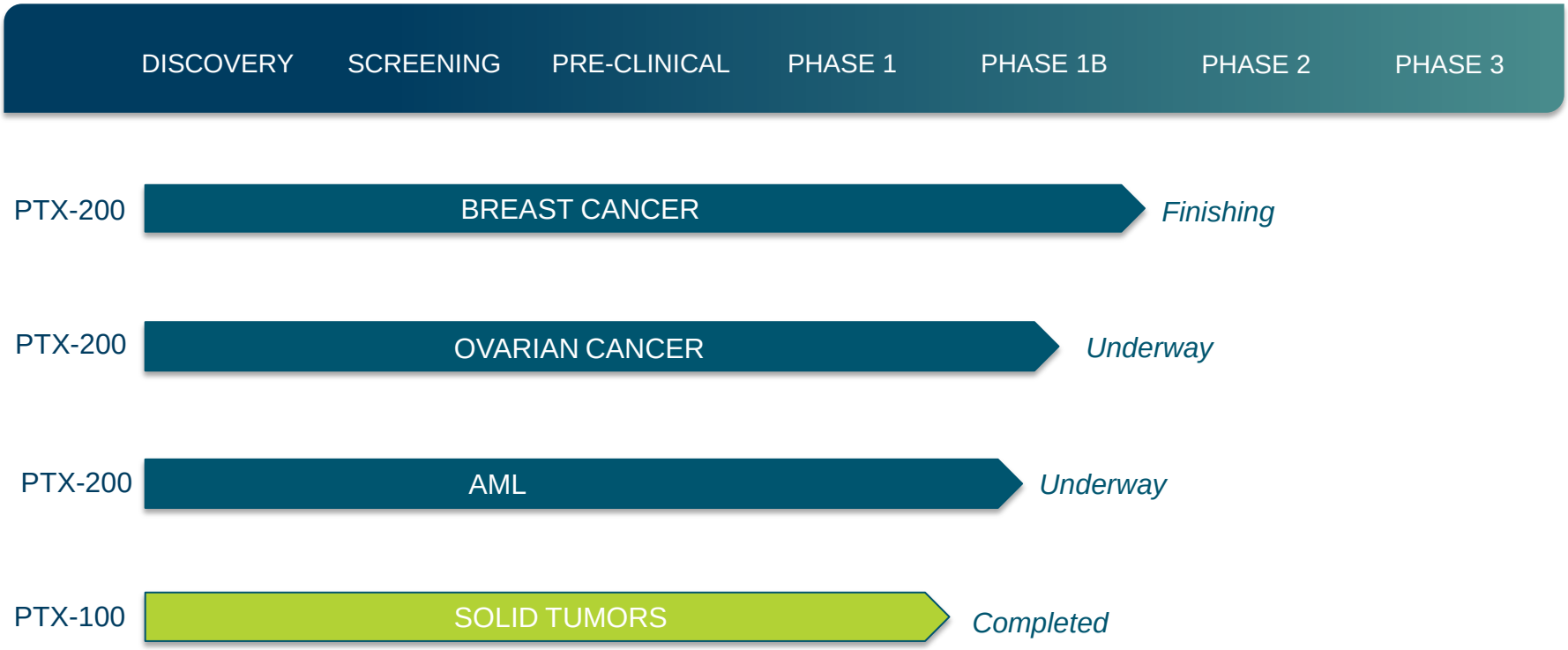
COMPANY OVERVIEW

COMPANY OVERVIEW

Two Clinical Stage Oncology Drugs	• 2 novel drug candidates (small molecules) targeting key cancer pathways • Akt (PTX-200) and Ras (PTX-100)
One of Deepest Clinical Pipelines on ASX	• 3 clinical trials in cancer (AML, ovarian & breast cancers) • All under INDs
Distinguished Scientific Provenance	• Compelling science from leading US institutions – Yale University & Moffitt Cancer Center • >65 peer reviewed publications
Significant Investment Already Made	• Over $20 M invested to date • Technologies have been awarded multiple prestigious US government grants
Proven Leadership & Management	• Experienced and proven drug development team on board to aggressively drive product development
Rich Upcoming News Flow	• Multiple milestone announcements and valuation inflection points across all clinical programs over next 12 to 18 months

DEEP, CLINICAL STAGE PRODUCT PIPELINE

- PTX-200 currently in three clinical trials
- Focus is now on further development of PTX-100 in Ras & Rho-mutant cancers



Prescient Therapeutics

CORPORATE SNAPSHOT

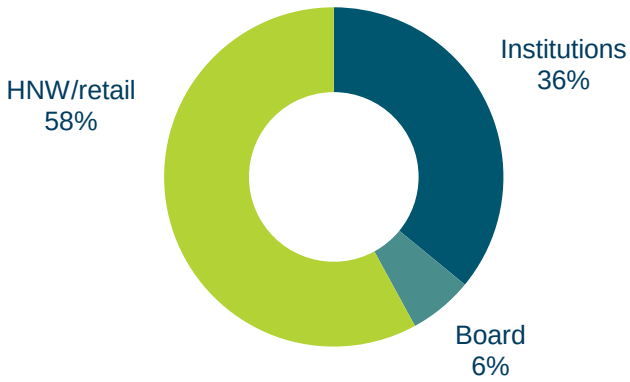
KEY METRICS

ASX Ticker	PTX
Total Issued Capital	211.3 M shares
Options	57.8 M
Share Price[1]	A$0.09 (US$0.065)

Market Capitalisation[1]	A$19.2 M (US$13.8 M)
Cash Position	A$9.3 M (US$6.7 M)
Top 20 Own	52%
6 month turnover[1]	56.7 M shares; A$6.0 M (US$4.3 M)

SHARE PRICE PERFORMANCE

SHAREHOLDER BASE



1 - AS AT 3 JANUARY 2017

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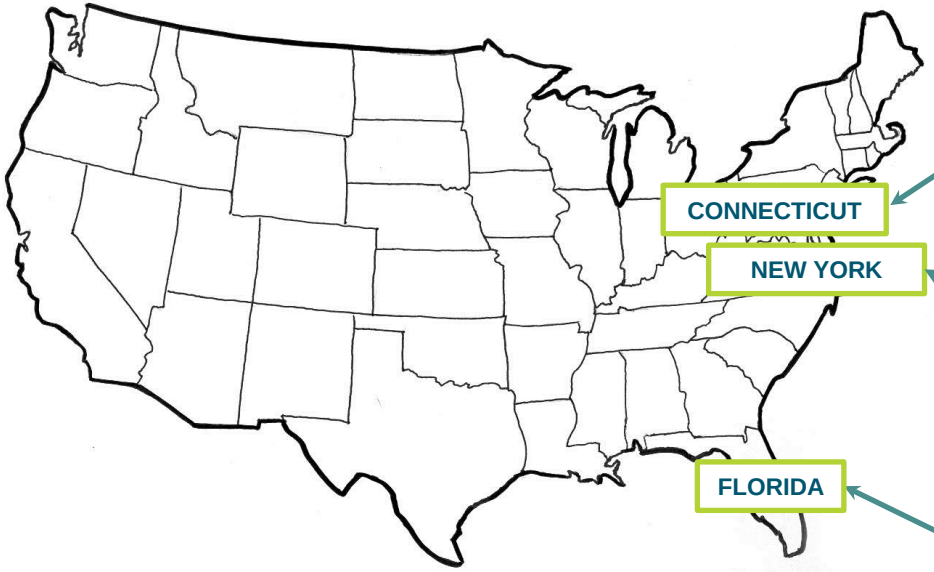
WORLD CLASS CENTERS & COLLABORATIONS



Yale CANCER CENTER

EINSTEIN
Albert Einstein College of Medicine
OF YESHIVA UNIVERSITY

MOFFITT CANCER CENTER

PREVIOUS CLINICAL TRIALS CONDUCTED AT:

THE UNIVERSITY OF TEXAS
MD Anderson Cancer Center
Making Cancer History

Memorial Sloan Kettering Cancer Center

Penn
University of Pennsylvania

INDIANA UNIVERSITY

NATIONAL CANCER INSTITUTE

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DEVELOPMENT TEAM WITH BENCH TO BEDSIDE SUCCESS

Proven success from discover and clinical development, through to FDA approvals

Said Sebti, PhD
Chief Scientific Officer

- Professor and Chair, Department of Drug Discovery - Moffitt Cancer Center
- Co-Program Leader, Chemical Biology and Molecular Medicine - Moffitt Cancer Center
- Inventor of PTX-100 & PTX-200
- **Named among top 20 Translational Researchers in the world by Nature Publishing Group**

Terry Chew, M.D.
Chief Medical Officer

- Hematologist/oncologist with 20 years experience in biotech & pharma
- Formerly with Argos and Peregrine Pharmaceuticals
- **5 New Drug Applications** including DaunoXome, Taxotere and DepoCyte
- **PTX is only 1 of only 2 ASX biotechs with a CMO that has successfully approved drugs**

Mandeep Grewal
Vice President – Clinical Operations

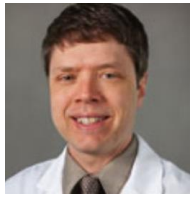
- Extensive clinical trial management experience with pharma, biotech & CROs
- Certifications: CRCP, CCRA, CCRP
- Formerly Exelixis, Quark Pharma, Fibrogen, Cytokinetics, Chiron, Abbott, Quintiles

Chaline Strickland
Clinical & Regulatory Affairs

- Doctor of Pharmacy
- Senior Director of Clinical Affairs at Ground Zero Pharmaceuticals
- Involved in dozens of New Drug Applications

CLINICAL ADVOCATE DRIVING OUR PROGRAMS

Acute Myeloid Leukemia



Jeff Lancet, M.D.

- Chair of the Department of Malignant Hematology at Moffitt Cancer Center
- Was Principal Investigator on Celator's transformative VYXEOS AML trial

MOFFITT CANCER CENTER

Breast Cancer

Joseph Sparano, M.D.

- Prof. of Medicine & Obstetrics, Gynecology, & Women's Health – Albert Einstein College of Medicine
- Assoc. Chairman for Clinical Research – Montefiore Medical Center Dept of Oncology

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Albert Einstein College of Medicine
OF YESHIVA UNIVERSITY

Heather Han, M.D.

- Assistant Prof. of Medicine at University of South Florida College of Medicine
- Medical oncologist, specializing in breast cancer
- The Center for Women's Oncology – Moffitt Cancer Center

MOFFITT CANCER CENTER

Ovarian Cancer

Robert Wenham, M.D.

- Section Head, Gynecologic Cancer Research, Moffitt Cancer Center
- Principal investigator Total Cancer Care Protocol

MOFFITT CANCER CENTER

PTX-200

NOVEL AKT INHIBITION
OVERCOMING KINASE PROMISCUITY
& LIMITATIONS OF PREVIOUS ATTEMPTS AT AKT INHIBITION

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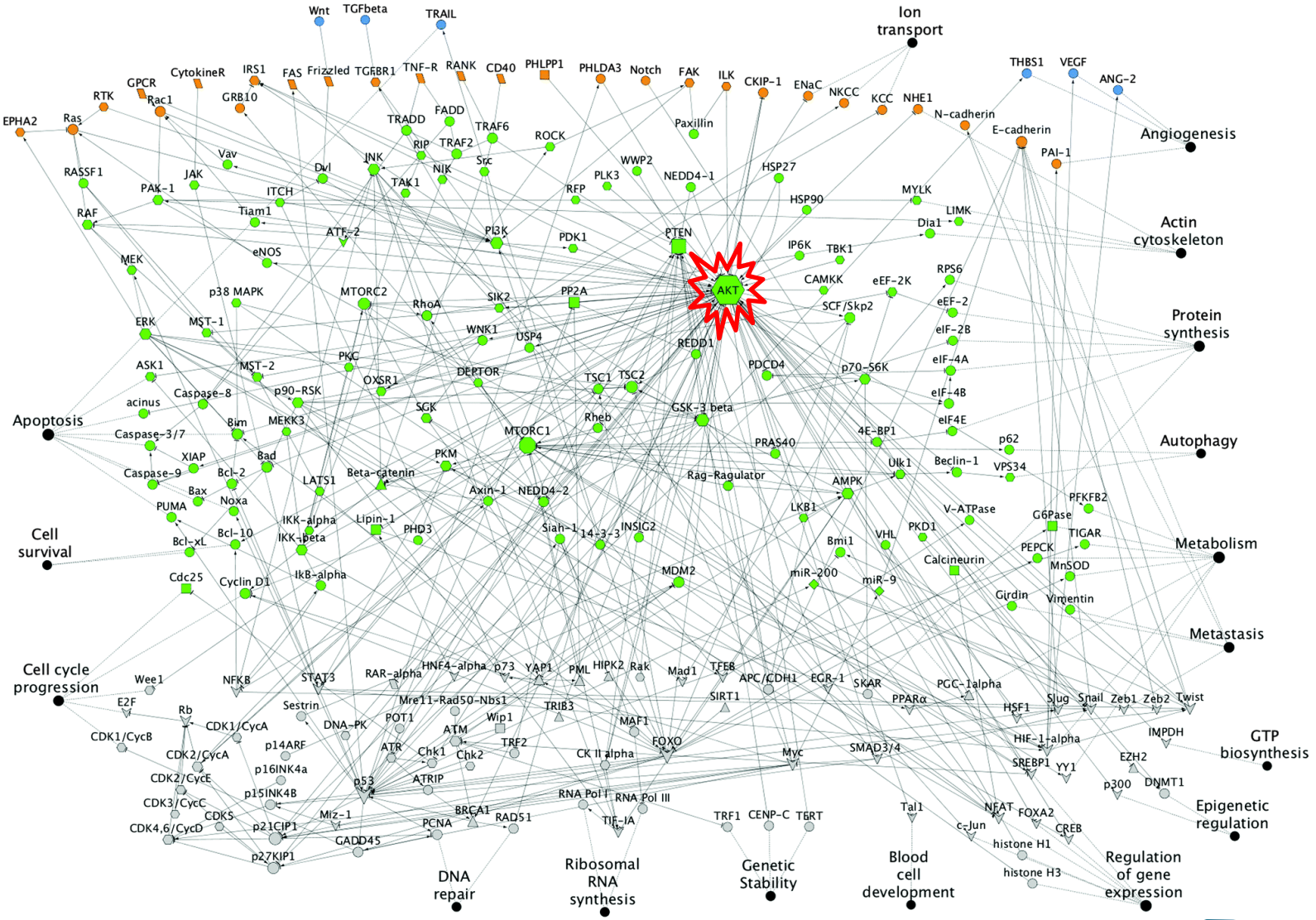
AKT IS A MASTER SWITCH FOR CELLULAR GROWTH & SURVIVAL



ERSAHIN, T. ET AL; MOL. BIOSYST., 2015,11, 1946-1954

Prescient Therapeutics

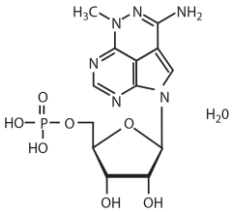
AKT REMAINS AN IMPORTANT DRUG TARGET

- Akt pathway promotes cellular survival and growth
- **Hyperactive** Akt signaling has two deleterious effects:
  - » Plays key role in the **development of many cancers** including breast, ovarian, colorectal, prostate, pancreatic and hematologic cancers
  - » **Confers resistance** to chemotherapy
- Therefore there is strong interest in Akt as a drug target
- Previous attempts at blocking Akt encountered fundamental problems leading to toxicities and/or lack of efficacy
  - » Focusing **too far upstream** (e.g. PI3K) or on **single arms** of pathways (including mTOR)
  - » Multikinase inhibitors/ATP mimics : **promiscuity leading to off target effects & toxicity**
- PTX-200 avoids these shortcomings

PTX-200 (TRICIRIBINE PHOSPHATE MONOHYDRATE) INTRODUCTION

- PTX-200 (TCN-P) is a small molecule Akt activation inhibitor that is highly selective for killing tumors with hyperactivated Akt
- **Inhibits Akt without the toxicity** of other attempts
- **Anti-proliferative** AND **pro-apoptotic**
- Selectively inhibits regulatory T cells[1]
  - » Significant anti-tumor effect that was Treg dependent
- **Novel mechanism of action**
  - » Inhibits Akt activation by binding to Akt PH domain and preventing localization to plasma membrane where Akt must be to be activated
  - » Vast advantage in MoA; **avoids off target effects**
- Overcomes chemotherapy resistance and causes cancer cells to die
- PTX-200 synergistic with chemotherapy
- Biomarkers of PTX-200 clinical activity: p-Akt; p-BAD; p-PRAS40
- **Completed Phase 1 trials demonstrated it is well tolerated and exhibited clinical activity in advanced acute leukemia patients**

PTX-200 (TCN-P)



MOFFITT CANCER CENTER

USF UNIVERSITY OF SOUTH FLORIDA

1. ABU-EID, *ET AL*. *CANCER IMMUNOL RES*; 2(11); 1080-9; 2014

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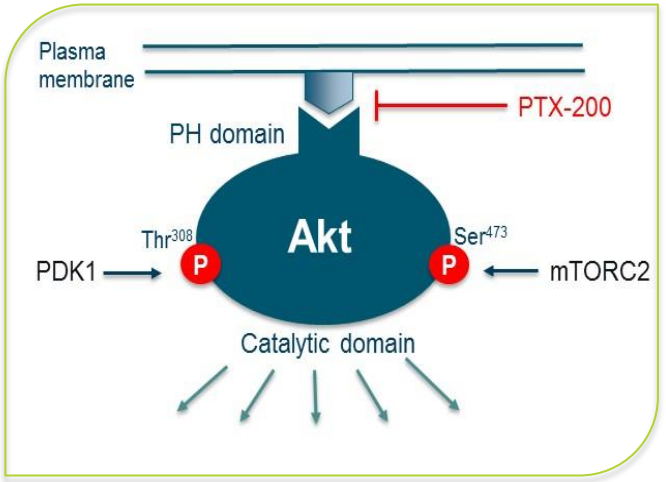
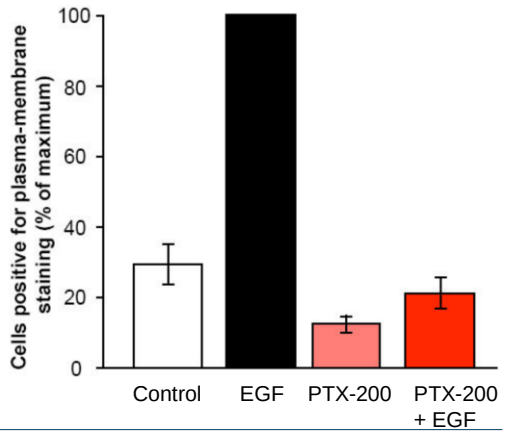
PTX-200: NOVEL AKT INHIBITION VIA PH DOMAIN BINDING

- PTX-200 is NOT an ATP mimic/direct kinase inhibitor, and therefore avoids off target effects associated with ATP mimic inhibitors.
  - » By binding to the PH domain, PTX-200 prevents binding to the plasma membrane, thus inhibiting activation of Akt by preventing phosphorylation of both Ser^{473} and Thr^{308}
- By inactivating Akt, PTX-200 suppresses the multitude of downstream targets of Akt
- PTX-200 inhibits EGF-induced recruitment of Akt to the plasma membrane
  - » Binds to Akt's PH domain required for membrane association
  - » Mimics the phosphate of the natural ligand for the PH domain, PIP3
  - » **By preventing plasma membrane association, PTX-200 inactivates Akt**





BERNDT, ET AL. CELL DEATH DIFFER. 2010; 17(11): 1795-1804
YANG, ET AL. CANCER RESEARCH 2004; 64, 4394-4399

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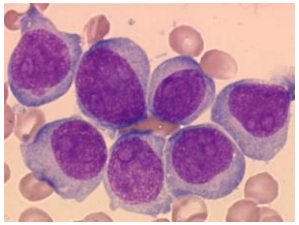
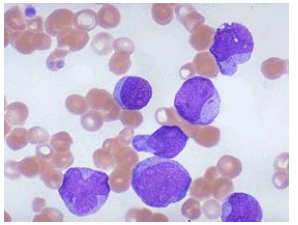
ACUTE MYELOID LEUKEMIA MARKET OVERVIEW





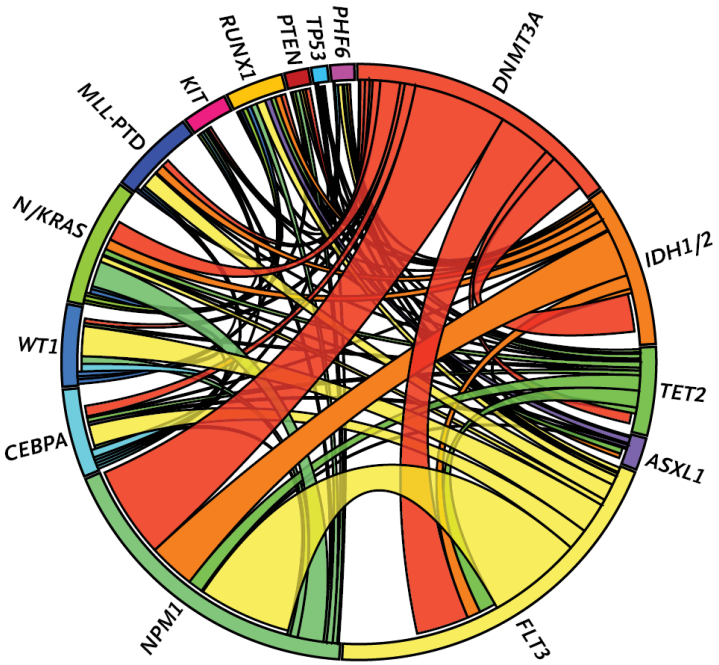
- AML is a type of cancer that affects the blood and bone marrow
  - » Patient cannot produce normal blood cells
  - » Blood cells cannot function properly nor fight disease
- Progresses very quickly & 5 year survival is a dismal 25%
- More common in adults over 60 years old, so the market is growing rapidly in developed economies
  - » 50% increase in incidence since 2013 in the US alone!
- After initial chemo, most patients relapse
- There are poor options for relapsing and refractory AML patients. **Treatment has barely changed in ~40 years!**
- These ingredients explain the massive interest in relapsing & refractory AML
  - » A growing, ageing disease in rich countries
  - » Dismal survival
  - » No treatment options!
- **PTX-200's approach mirrors other current successful development approaches in AML of using targeted therapies complementing a "backbone" of standard chemo**
- PTX-200's compelling efficacy signals has attracted interest of renowned clinicians and investors

PTX-200 IN AML – EXECUTIVE SUMMARY

- Akt is highly relevant in AML
- PTX-200 address the “phenotype, not the genotype” in AML mutations
- Like other recent successful strategies in AML, PTX-200 is a targeted therapy complementing a “backbone” of standard chemotherapy
- PTX-200 synergizes with cytarabine in AML cells
- Successful Phase 1 trial in acute leukaemias as a monotherapy
  - » 1 CR, 2 PRs in r/r AML; 1 response in refractory CMML
  - » Overall 53% SD in a highly pre-treated population with advanced disease
- PTX-200 reduced pAkt in AML patient blasts
- Phase 1b trial now underway (PTX-200 + cytarabine) under the leadership of Prof Jeff Lancet

MUTATIONAL COMPLEXITY OF AML

- Although AML is a mutationally complex disease, many different types of mutations (and combinations of mutations) result in hyperphosphorylation of Akt
  - » In fact, **72% of AML patients have high p-Akt**
- Previous Akt drugs have failed because of the heterogeneity of mutations, and/or toxicities
- PTX-200 suppresses p-Akt in human tumors due to its novel MoA



Gene	Overall Frequency (%)
FLT3 (ITD, TKD)	37 (30, 7)
NPM1	29
DNMT3A	23
NRAS	10
CEBPA	9
TET2	8
WT1	8
IDH2	8
IDH1	7
KIT	6
RUNX1	5
MLL-PTD	5
ASXL1	3
PHF6	3
KRAS	2
PTEN	2
TP53	2
HRAS	0
EZH2	0

Many mutations, and combinations of mutations, result in high p-Akt

PATEL, ET AL. NEJM 2012; 366:1079

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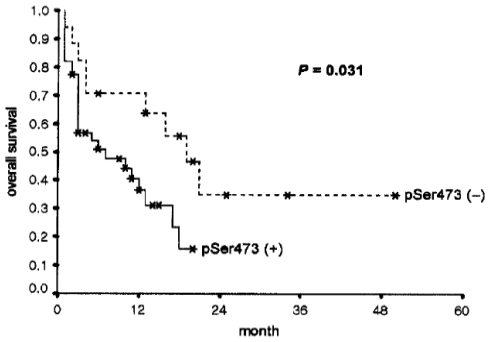
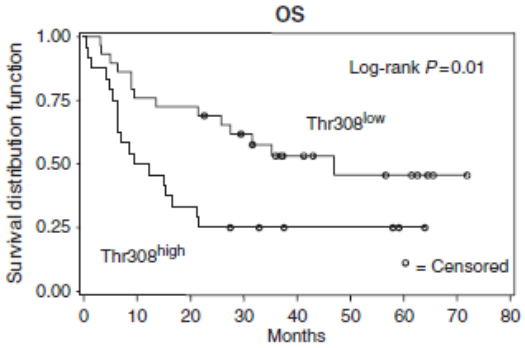
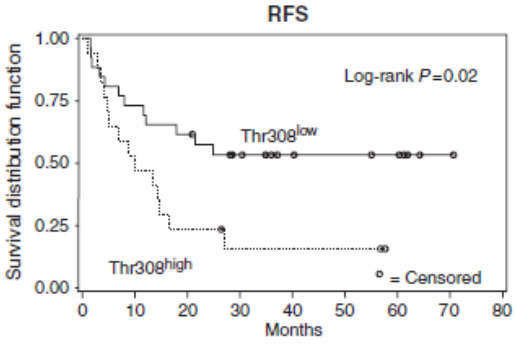
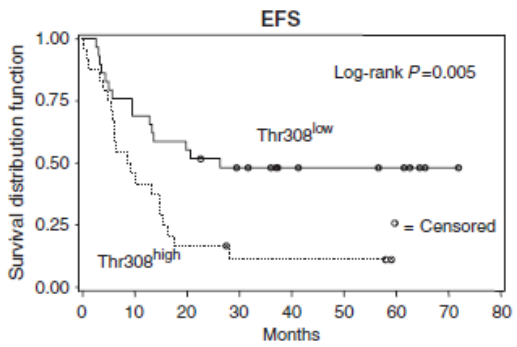
RELEVANCE OF AKT IN AML

- Frequent constitutive Akt activation (phosphorylation) in AML
  - » Constitutive phosphorylation (Ser^{473} and Thr^{308}) of Akt in AML compared to normal bone marrow cells in 44 out 66 of patients (72%)
- Implications for Akt as a modulator of chemotherapy resistance in AML
- **High Akt phosphorylation (on either phosphorylation site) = inferior survival**
- **PTX-200 inhibits Akt phosphorylation of both Ser^{473} and Thr^{308}**









1. MIN YH, ET AL. LEUKEMIA 2003; 17:995
2. GALLAY, ET AL. LEUKEMIA 2009; 23:1029

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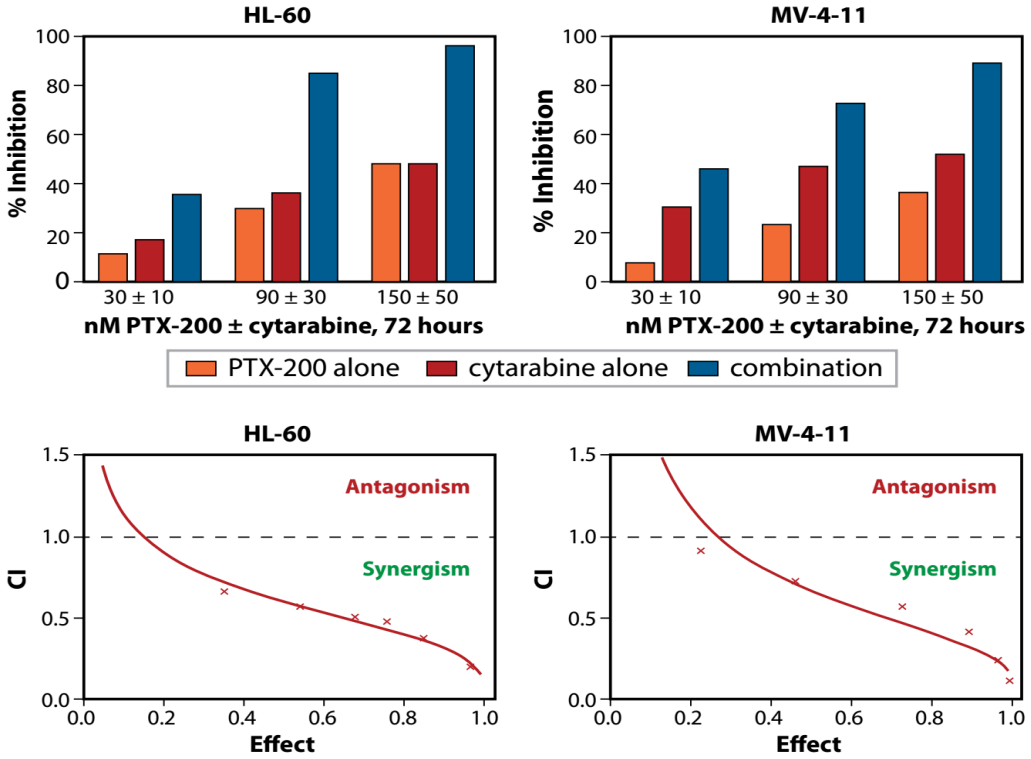
PTX-200 SYNERGIZES CYTARABINE IN AML CELLS

- PTX-200 highly synergistic with the current standard of care (cytarabine) in AML cells
  - » PTX-200 + cytarabine = much more effective effect than the simple additive effect of either compound (i.e. 1 + 1 ≥ 2)

- As cytarabine is the current standard of care in AML, this suggests that **PTX-200 may potentiate the standard of care, and have significant clinical relevance in the relapse/refractory setting**



SEBTI, S & LANCET, J

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PTX-200: COMPLETED PHASE 1 MONOTHERAPY IN HEMATOLOGIC MALIGNANCIES (MAINLY AML)

Patients	• 32
Trial Centers	• MD Anderson & Moffitt
Patient Inclusion	• Advanced hematologic malignancies (mainly AML)
Study Objectives	• To establish dosing regime and biological dose
Methods	• 1 hour IV infusion on days 1, 8, and 15 • Cycle repeated every 21 days
Results	• 8 patients received at least 2 cycles • 17 out of 32 patients had stable disease (53%) • 4 patients with complete or partial response (12.5%) » 1 refractory AML patient had complete response after 2 cycles » 2 r/r AML patients had partial response after 1 cycle » 1 refractory CMML patient had normalization of WBC and dramatic reduction in spleen size • Compelling signals of efficacy • Further investigation of PTX-200 alone or in combination in patients with high p-Akt levels is warranted

THE UNIVERSITY OF TEXAS MD Anderson Cancer Center

MOFFITT CANCER CENTER

Published Leuk Res. 2013 Nov;37(11):1461-7

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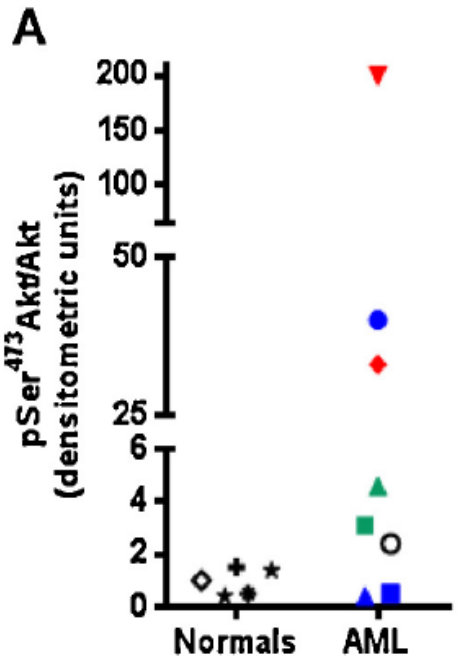
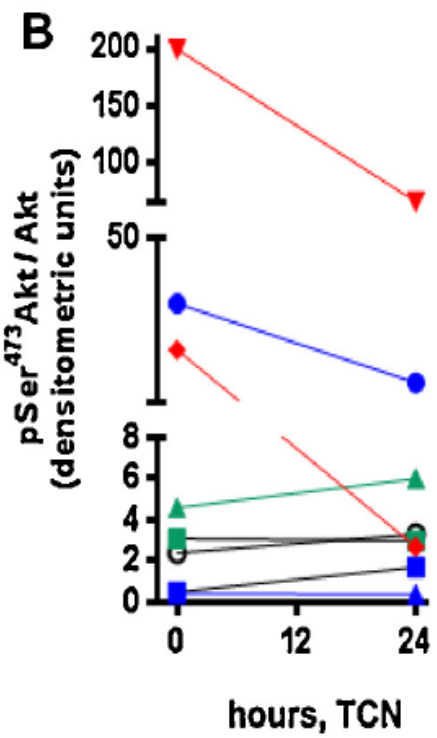
PTX-200 REDUCED P-AKT IN AML BLASTS

- Phase 1 trial demonstrated that blast cells with high p-Akt from AML patients are more sensitive to PTX-200's ability to reduce p-Akt levels

» **p-Akt/Akt in AML samples before therapy**



» **Action of PTX-200 on Akt phosphorylation in AML blasts**



LANCET, ET AL. LEUK RES 2013; 37:1461

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COMPELLING EVIDENCE FOR PTX-200 IN AML

Efficacy hypothesis	PTX-200 Evidence
• High p-Akt is correlated with inferior survival in AML	• PTX-200 **decreased pAkt in AML blasts**
• Inhibiting p-Akt improves response to chemo in the clinical setting	• PTX-200 decreased pAkt in AML blasts, suggesting this method of reducing pAkt would similarly improve clinical outcomes
• Phase I achieved safety?	• Yes
• Any evidence of clinical benefit?	• Yes. **53% SD** in very heavily pre-treated, sick patients with rapidly progressing disease, despite only using a single cycle of monotherapy • **3 patients had >50% blast reduction**.
• Is there a comparable with any other attempted Akt inhibitor in AML?	• **PTX-200 had more compelling results than another Akt candidate MK2206** in Phase1 AML. (MK2206 development has since been discontinued by Merck)
» MK2206 successfully demonstrated apoptosis of AML cell lines in vivo, but failed to meaningfully inhibit p-Akt in the clinical setting	» PTX-200 successfully inhibited p-Akt in the clinical setting
» MK2206: Only 1 response out of 19 patients (5% SD)	» 17 out of 32 achieved stable disease (53% SD)
» MK2206 failed at MTD	» Succeeded well **below MTD**
• How does it combine with current standard of care?	• PTX-200 is **highly synergistic** with cytarabine in AML cells
• Lessons from other trials currently running?	• In current Phase 1b breast & ovarian cancer trials for PTX-200, interim analysis showed encouraging efficacy

➔ PTX-200 has lots of supportive data and efficacy signals that combine to give confidence leading into the Phase 1b/2 trial.

PHASE 1B TRIAL UNDERWAY: ACUTE MYELOID LEUKEMIA

- PTX-200 plus cytarabine in refractory or relapsed AML
- Phase 1 results very encouraging
- Phase 1b/2 IND allowed by FDA
- 3+3 design, single arm
- Jeff Lancet at Moffitt Cancer Center leading the trial
- Yale Cancer Center second site participating in trial
- 15 -18 patients
- **First patient recruited**
- Bolstered PTX's Scientific Advisory Board with world class leukemia expertise from Moffitt, Yale and MD Anderson

Jeffrey E Lancet, M.D.
Principal Investigator

MOFFITT CANCER CENTER

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PHASE 1B BREAST CANCER TRIAL ALMOST COMPLETED

- PTX-200 in combination with paclitaxel, followed by doxorubicin and cyclophosphamide
- Patients with metastatic and locally advanced HER2- breast cancer (mostly TNBC)
  - » Recruiting at Albert Einstein College of Medicine Montefiore Medical Center and the H. Lee Moffitt Cancer Center
- **Encouraging early data**
  - » Evidence of safety & anti-tumor activity
- **24 patients already dosed – now in expansion phase**
  - » n = 12 patients in Phase 1b expansion stage

Joseph Sparano, M.D.
Principal Investigator

EINSTEIN
Albert Einstein College of Medicine
OF YESHIVA UNIVERSITY

Heather Han, M.D.

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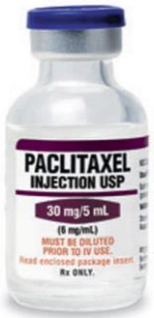

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PHASE 1B OVARIAN CANCER TRIAL COMMENCED

- Significant need for new products to treat platinum-resistant ovarian cancer
- Testing PTX-200 plus carboplatin in patients with platinum resistant ovarian cancer
- PTX-200 already proven **overcome cisplatin resistance** and **synergize with cisplatin** in pre-clinical studies
- **Phase 1b already underway**
- Currently recruiting at H. Lee Moffitt Cancer Center
- Up to 12 patients with an additional 18 in expansion cohort
- **7 patients in total already dosed**
- **First patient now dosed at second dose level**

Robert Wenham, M.D.
Principal Investigator

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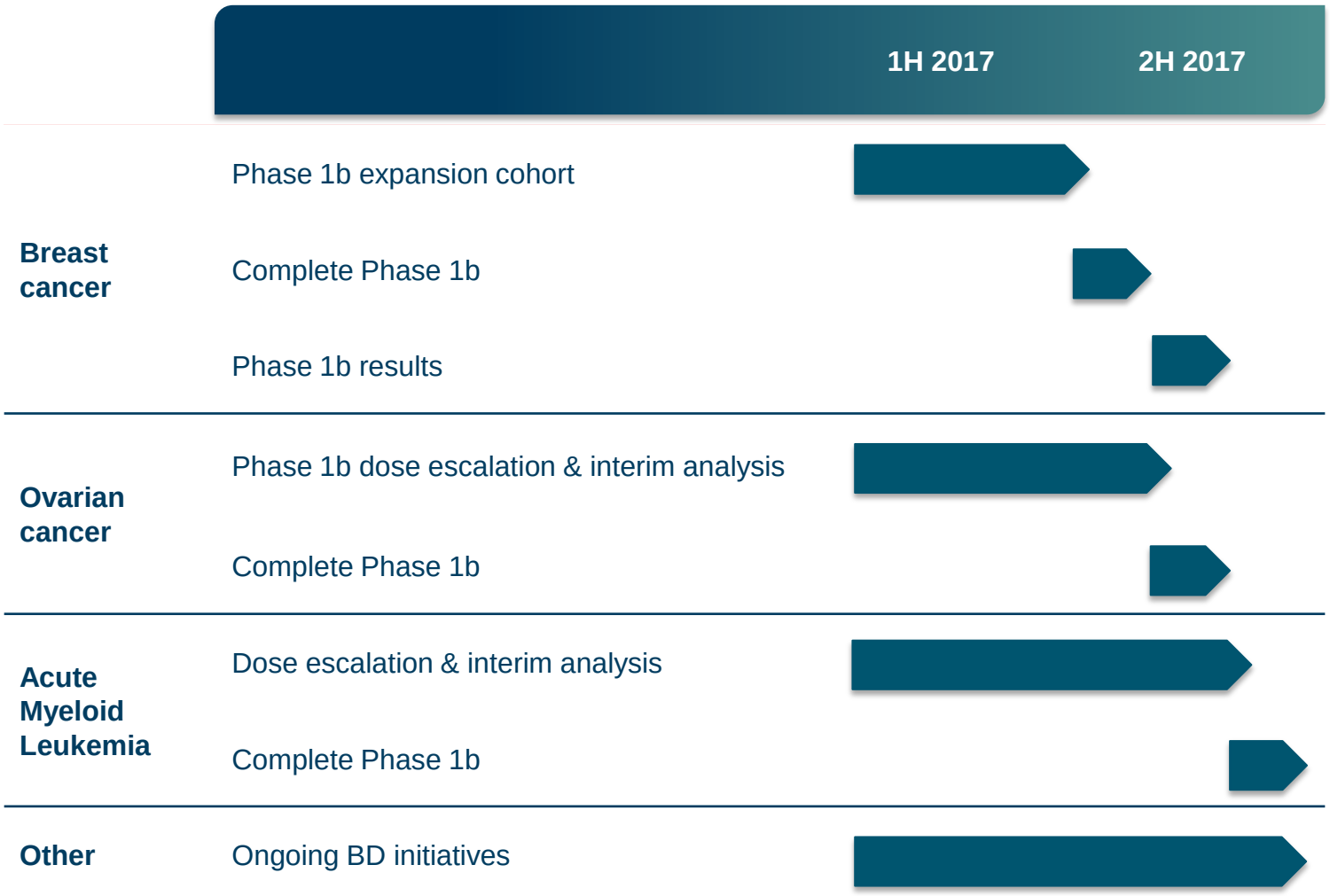
IMMEDIATE PTX-200 MILESTONES & INDICATIVE TIMING



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PTX-100

FIRST IN CLASS, FIRST IN MAN
GGT-1 INHIBITOR OF RAS PATHWAY

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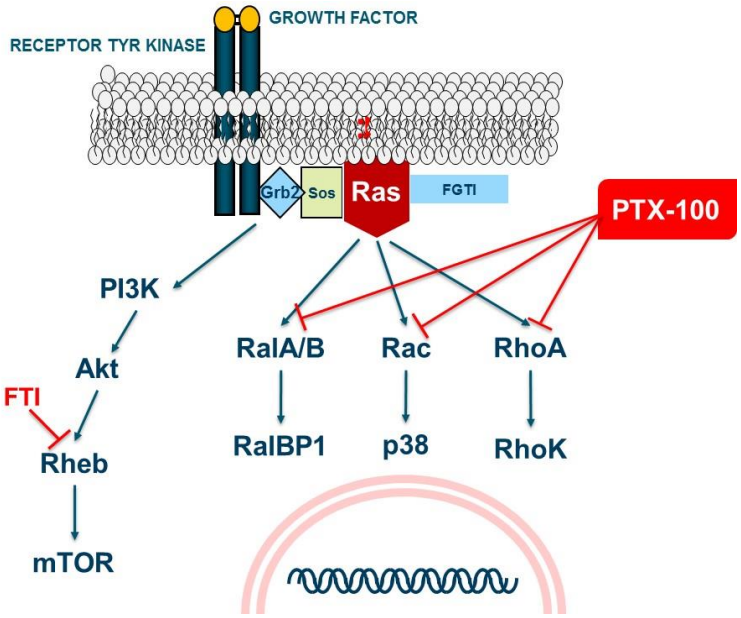
RAS PATHWAY IS AN IMPORTANT BUT AN ELUSIVE TARGET

- Ras mutated in 30% of all human cancers and 90% in certain cancers
  - » A staggering 3 million new cancers diagnosed worldwide each year with Ras mutations
- Mutant Ras tumors are often unresponsive to current treatments
- **Patients with Ras mutant cancers are still significantly underserved due to a lack of suitable targeted therapies**
- NCI identified targeting Ras as a high priority with a major initiative to discover therapies that target patients whose tumors harbor mutant Ras
  - » National Comprehensive Cancer Network guidelines: Treat with EGFR inhibitors only patients whose tumors do not harbor mutant KRas



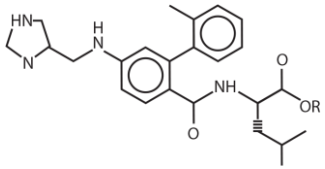
- Ras, Rho and Ral post-translational modifications with the lipids Farnesyl and/or GeranylGeranyl are critical to their cellular localization and cancer-causing activity
- Targeting Ras directly has proven elusive; **PTX disrupts the Ras pathway by inhibiting post-translational modification of Ral, Rac and Rho required for their activation**

Prescient Therapeutics

PTX-100 (GERANYLGERANYL TRANSFERASE) INTRODUCTION

- PTX-100 (GGTI-2418) small molecule inhibitor of the GGT-1 enzyme
- **Geranylgeranyl transferase (GGT-1) is key activator in the Ras pathway via Ral, Rac & Rho**
  - » Overcomes failures of Farnesyltransferase inhibitors (FTIs)
  - » FTIs cause escape via GGT-1, but not vice versa
  - » PTX-100 inhibits GGT-1 potently and selectively over FT
- Invented at Yale University and Moffitt Cancer Center
- p27 a potential companion diagnostic for PTX-100
- Completed Phase 1 trials demonstrated it is well tolerated, patients achieved stable disease
- **Single agent activity in lung, pancreatic and breast cancers, and multiple myeloma in mouse models**
- **Combination therapy is also very effective**, due to PTX-100's large therapeutic index and safety profile, and efficacy in mutant Ras tumors
- PTX-100 shown to reduce cancer stem cell population in animal models

PTX-100 (GGTI-2418)



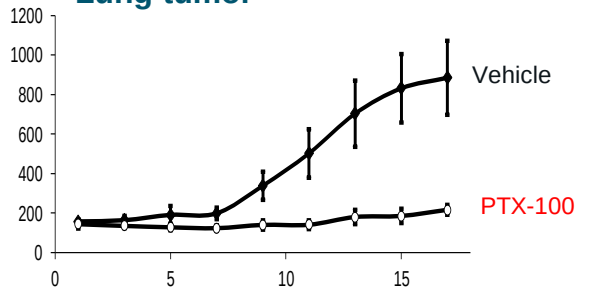
Yale University

MOFFITT CANCER CENTER

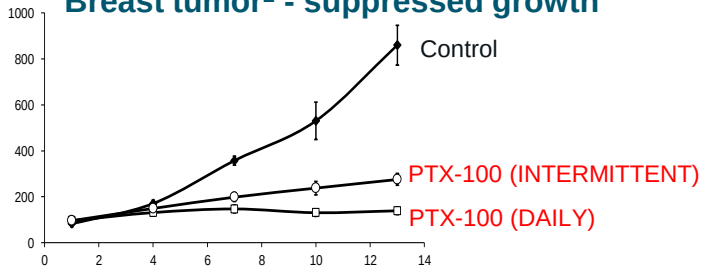
Prescient Therapeutics

PTX-100 IS A HIGHLY EFFECTIVE ANTI TUMOR AGENT IN PRE-CLINICAL MODELS AND PATIENT FRESH BIOPSIES

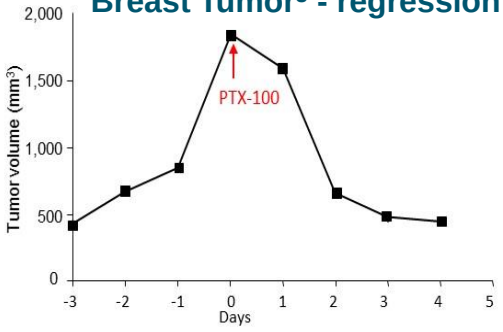
Lung tumor[1]



Breast tumor[2] - suppressed growth



Breast Tumor[3] - regression



- PTX-100 inhibits tumor growth and metastasis, induces tumor regression, and increases survival in various mouse models:
  - » **Inhibits tumor growth** in human lung, breast, multiple myeloma and pancreatic cancer mouse xenografts
  - » **Induces regression** in Her2-driven breast cancer in transgenic mice
  - » **Dose regimen response** in breast cancer model
  - » **Inhibits metastasis to the liver** in a pancreatic cancer mouse model
  - » **Increases the survival** of mice in an aggressive multiple myeloma mouse model
- PTX-100 is effective at inhibiting the viability of **Multiple Myeloma** fresh biopsies from patients refractory to Multiple Myeloma standard therapy
- PTX-100 is **highly synergistic** with Bortezomib and Carfilzomib at inhibiting the viability of Multiple Myeloma fresh biopsies from patients refractory to Multiple Myeloma standard therapy

1. A54 MOUSE XENOGRAFTS
2. MDA-231 MOUSE XENOGRAFTS
3. ERBB2 TRANSGENIC MICE

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PTX-100: COMPLETED PHASE 1 IN ADVANCED SOLID TUMORS

Patients	• 13
Trial Centers	• University of Pennsylvania & Indiana University
Patient Inclusion	• Heavily pre-treated patients with refractory, advanced solid tumors » 5 rectal; 5 colon; 1 hepatocellular; 1 carcinoid; 1 esophageal • Median 4 prior regimens.
Study Objectives	• Determine dose limiting toxicity (DLT) • Assess safety, tolerability & pharmacokinetics • Observe clinical response
Methods	• 30-min IV infusion on days 1-5 every 21 days • 8 dose levels from 120 – 2060 mg/m^2. 1 patient/dose level until tox, then 3+3
Results	• Well tolerated – nausea main adverse event • Elevation in Liver Function Test identified as dose limiting toxicity for only 1 patient out of 6, thus DLT was never reached • **Durable stable disease achieved in 4 cancer patients (31%)** » 2 rectal cancer patients had durable SD (6-7 cycles at 500-750 mg/m^2) » 1 hepatocellular carcinoma patient had durable SD (3 cycles at 330 mg/m^2) » 1 carcinoid tumor patient had durable SD (8 cycles at 2,060 mg/m^2) • **PK at dose level 5 (1050 mg/m^2) was 36,000x the IC50 value to inhibit GGT-1 in vitro**

Penn
University of Pennsylvania

INDIANA UNIVERSITY

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PTX-100: CANCER STEM CELL OPPORTUNITY

- Cancer stem cells (CSCs) are one of the reasons cancers rebound
- CSCs resistant to conventional therapies
- Targeting CSCs is crucial to treating malignant disease
- **PTX-100 reduces breast cancer stem cells** both *in vitro* and *in vivo* in patient derived breast tumors
- Offers exciting new way to treat cancer with the potential to inhibit CSC-mediated tumor relapse
- Warrants further investigation in future trials

INSTITUT
CENTRE RÉGIONAL DE LUTTE CONTRE LE CANCER
PAOLI-CALMETTES

Published Stem Cells 2012;30:1327-1337

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MANY PTX-100 DEVELOPMENT OPPORTUNITIES

- Mutated N- & K-Ras pathway is a feature of many cancers, including:
  - Pancreatic & other GI cancers
  - NSCLC
  - Multiple Myeloma
- Considering Phase 1b/2 trial in Ras mutant all-comers with PTX-100 as monotherapy
- RhoA (a direct target of PTX-100) implicated in many haematological malignancies
- Additional PTX-100 drug product will need to be manufactured
- Wide therapeutic index, MoA and safety profile lend PTX-100 to combination therapies
  - PTX-100 has Demonstrated high degree of synergy with a number of agents to date (including gemcitabine, bortezomib, carfilzomib) in mouse models as well as in fresh biopsies from refractory patients
- p27 as a companion diagnostic (personalized medicine approach)

INVESTMENT HIGHLIGHTS

2 DRUGS » 3 TRIALS » IMMINENT CATALYSTS » FUNDING IN PLACE » UNDISCOVERED VALUE

- **2 targeted therapies** with impeccable scientific pedigree
- Multiple shots on goal with **Akt and Ras pathway inhibitors** in **multiple trials**
- One of deepest clinical pipelines on the ASX
  - » Targeting important areas of unmet clinical need
- Funded through to value-accretive catalysts, with a fantastic share register
- Phase 1b/2 AML trial is being led by renowned leukemia expert, Dr Jeff Lancet
  - » Dr Lancet was also the Principal Investigator on Celator Pharmaceuticals' ground-breaking VYXEOS trial in AML
- Great scientific and clinical team with a proven record of success
- Catalysts not far away

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