



**DEVELOPING NOVEL TARGETED
THERAPIES TO BEAT CANCER**

SPOTLIGHT ON PTX-100

Prescient Therapeutics Limited (ASX: PTX)
October 2017

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COMPANY OVERVIEW



INVESTMENT HIGHLIGHTS

2 DRUGS » 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
- **Multiple catalysts** for value creation
- **Funded through to value-accretive catalysts**, with a fantastic share register
- Phase 1b/2 AML trial is being led by globally renowned leukemia expert, Professor Jeff Lancet
 - » Professor Lancet also led Celator Pharmaceuticals' ground-breaking VYXEOS trial in AML
- Great scientific and clinical team with a **proven record of success**
- Recent **encouraging efficacy breast cancer results, despite SAE resulting in clinical hold**
- **Transformative opportunity in rare blood (heme) cancers**

CORPORATE SNAPSHOT

KEY METRICS

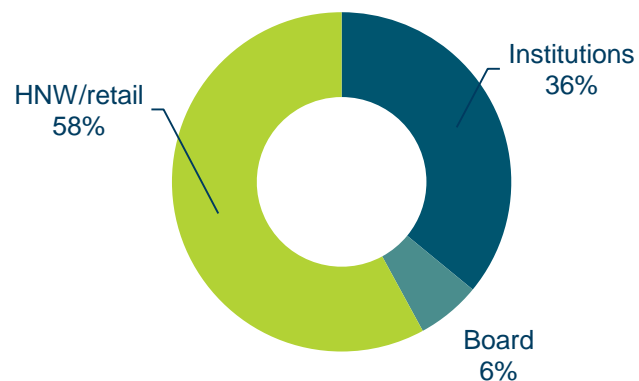
ASX Ticker	PTX
Total Issued Capital	211.3 M shares
Options	57.8 M
Share Price ¹	A\$0.062 (US\$0.049)

Market Capitalisation ¹	A\$13.0 M (US\$10.3 M)
Cash Position ²	A\$7.7 M (US\$6.1 M)
Top 20 Own	52%
6 month turnover ¹	39 M shares; A\$3.0 M (US\$2.4 M)

SHARE PRICE PERFORMANCE

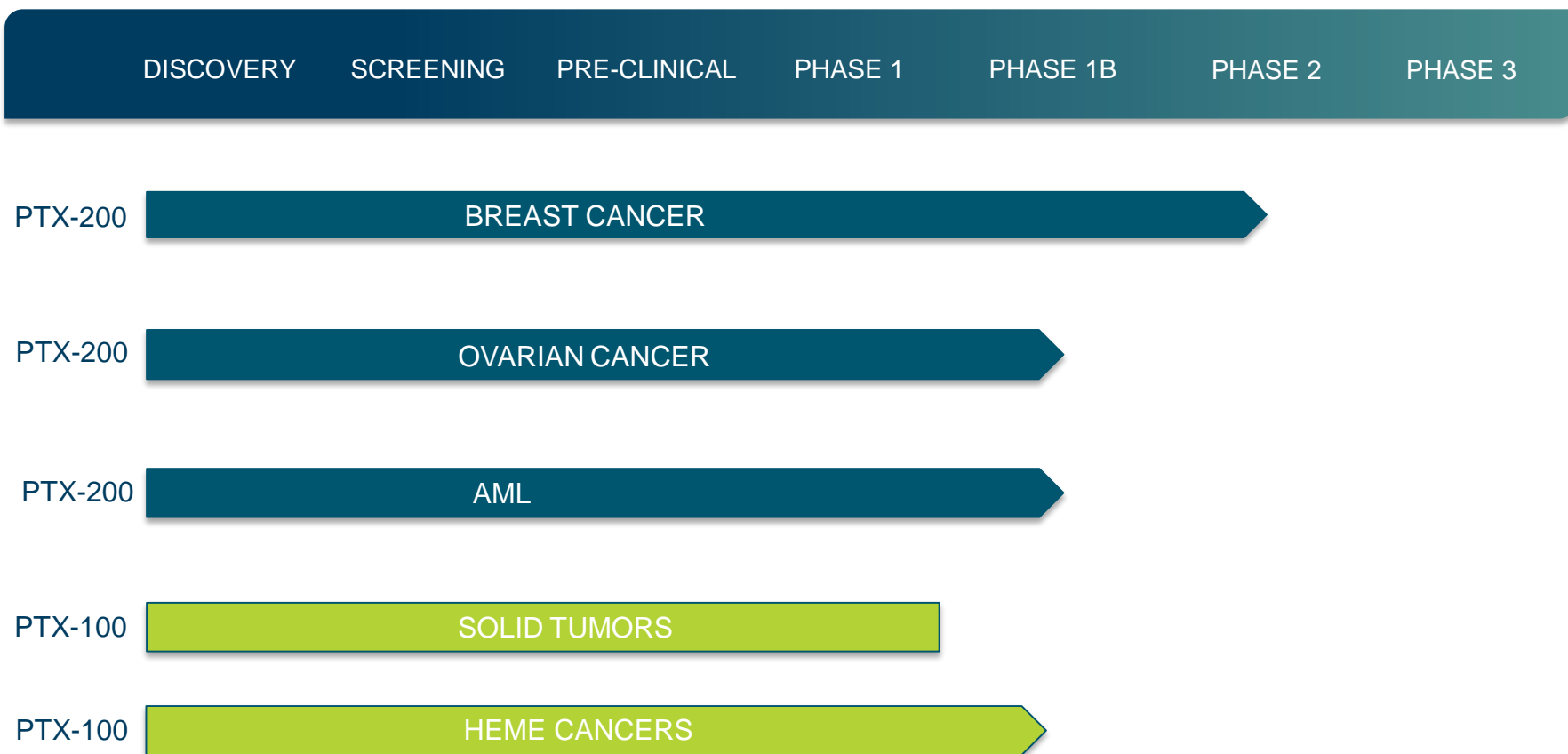


SHAREHOLDER BASE



DEEP, CLINICAL STAGE PRODUCT PIPELINE

- PTX-200 currently in three clinical trials (BRCA and OVCA recruitment currently on hold pending SAE response)
- Advancing PTX-100 in rare hematological cancers - a transformative opportunity



CONTINUING TO DELIVER MEANINGFUL PROGRESS

RECENT PROGRESS

PTX-200

- ✓ AML: Phase 1b study initiated at Moffitt and Yale; first cohort successfully completed
- ✓ Breast Cancer: Phase 1b completed and Phase 2 initiated
- ✓ Ovarian Cancer: First cohort completed
- ✓ New drug product manufactured

PTX-100

- ✓ New clinical plan developed

Corporate

- ✓ \$10.5 M capital raising; reputable institutional investors added to share register
- ✓ IP bolstered with granted patents
- ✓ Bolstered team

PLANNED UPCOMING ACTIVITIES

PTX-100

- Commence activities in new hematology indication

PTX-200

- Work with FDA to recommence recruitment
- AML: Completion of second cohort
- Ovarian cancer: Completion of second cohort

Corporate

- Continue to build awareness amongst clinicians, investors and corporates
- Pipeline development

DRUGS DON'T DEVELOP THEMSELVES!

PTX DEVELOPMENT TEAM WITH BENCH TO BEDSIDE SUCCESS

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



Said Sebti, PhD
Chief Scientific Officer

- Professor and Chair, Department of Drug Discovery - Moffitt Cancer Center
- Co-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
- **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
VP – Clinical Operations

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



Mike Preigh, PhD
VP - CMC

- **Led CMC at Array BioPharma for 10 years**
- Successfully brought >20 drug candidates to IND & clinical development
- Previously Pfizer



Claudia Gregorio-King, PhD
VP - Operations

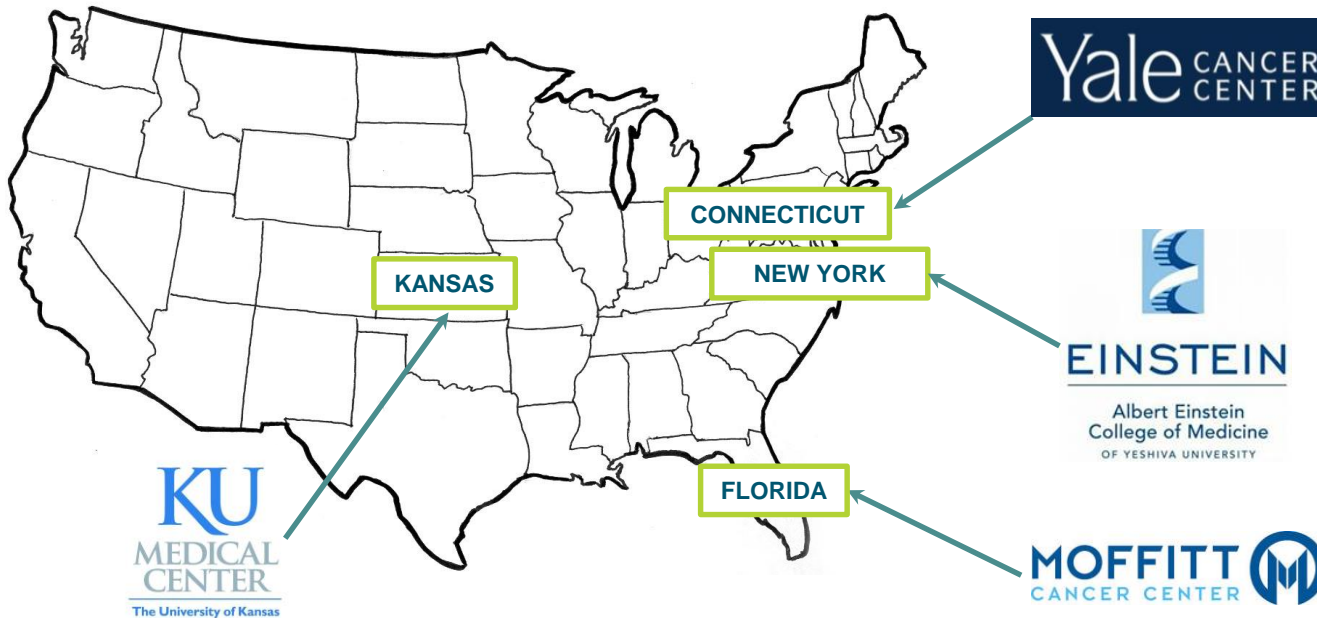
- Extensive experience in the management of pre-clinical and clinical research and intellectual property
- Regulatory affairs and clinical project management experience with small and large CROs



Chaline Strickland, Pharm.D.
Regulatory Affairs

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

WORLD CLASS CENTERS & COLLABORATIONS



PREVIOUS CLINICAL TRIALS CONDUCTED AT:



Memorial Sloan Kettering Cancer Center



INDIANA UNIVERSITY



PTX-100

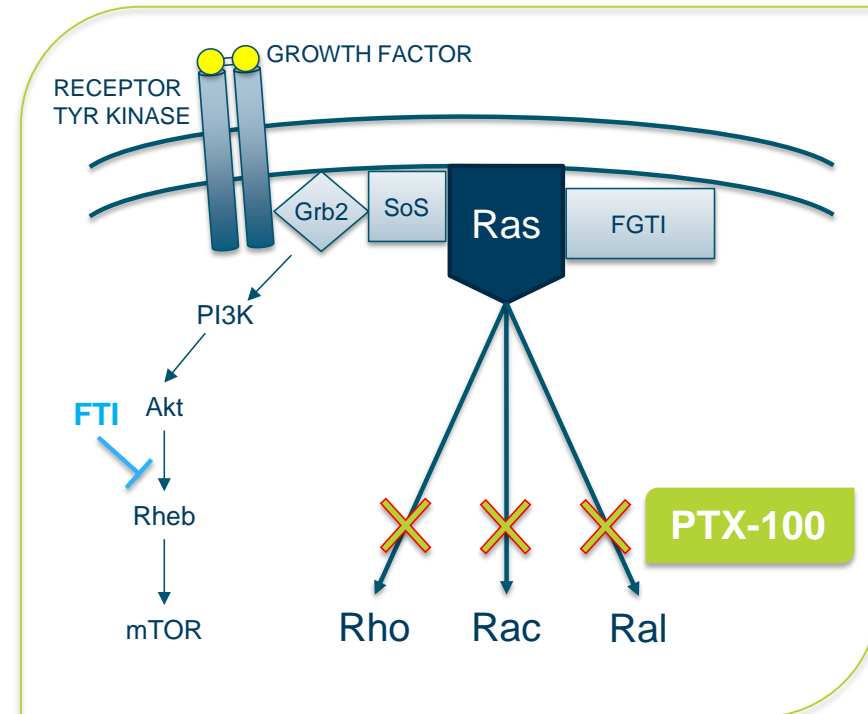
**FIRST IN CLASS
INHIBITOR OF RAS PATHWAY**

Phase 1 in solid tumors completed;
Now pursuing a transformative opportunity in rare blood cancers



RAS PATHWAY IS AN IMPORTANT BUT ELUSIVE TARGET

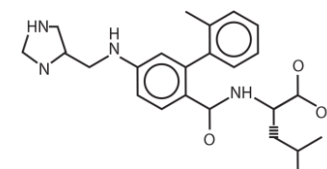
- Ras mutated in 30% of all human cancers and 90% in certain cancers
 - » A staggering 3 million new cancers diagnosed worldwide each year with Ras mutations
- Mutant Ras tumors are often unresponsive to current treatments
- **Patients with Ras mutant cancers are still significantly underserved due to a lack of suitable targeted therapies**
- NCI identified targeting Ras as a high priority with a major initiative to discover therapies for Ras mutant cancers
- Targeting Ras directly has proven elusive; **PTX-100 disrupts the Ras pathway by inhibiting the activation of Ral, Rac and Rho**
- **PTX-100 recently discovered to also inhibit a novel cancer causing pathway FBXL2**



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

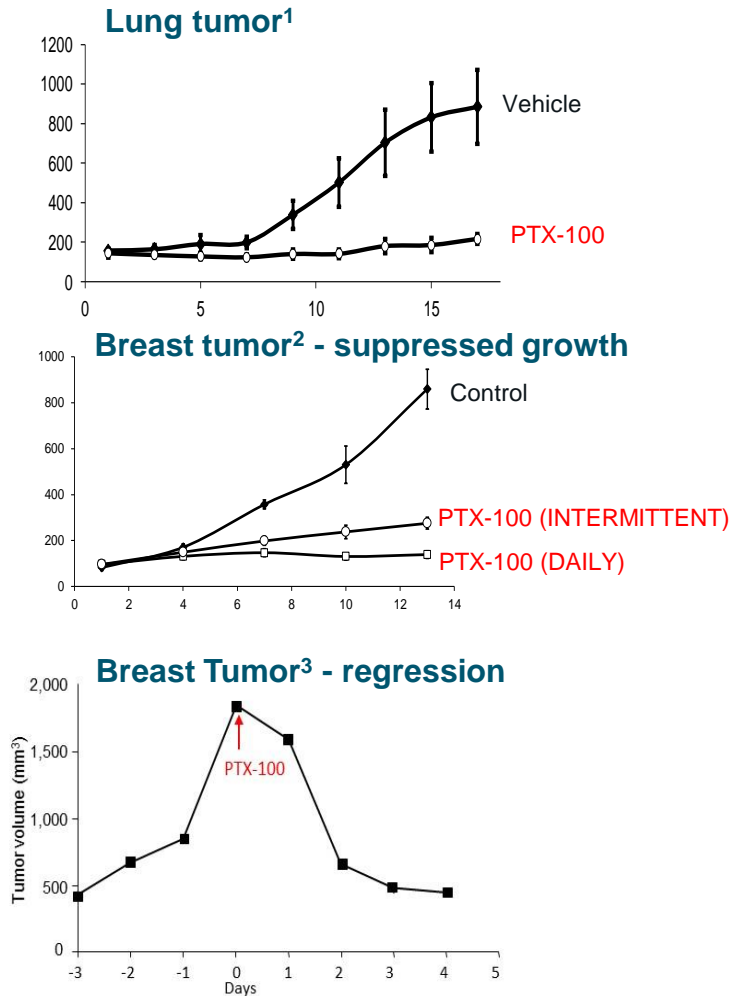
PTX-100 (GGTI-2418)



Yale University

MOFFITT
CANCER CENTER

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



- 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

PTX-100: COMPLETED PHASE 1 IN ADVANCED SOLID TUMORS

Patients	<ul style="list-style-type: none">• 13
Trial Centers	<ul style="list-style-type: none">• University of Pennsylvania & Indiana University
Patient Inclusion	<ul style="list-style-type: none">• Heavily pre-treated patients with refractory, advanced solid tumors<ul style="list-style-type: none">» 5 rectal; 5 colon; 1 hepatocellular; 1 carcinoid; 1 esophageal• Median 4 prior regimens.
Study Objectives	<ul style="list-style-type: none">• Determine dose limiting toxicity (DLT)• Assess safety, tolerability & pharmacokinetics• Observe clinical response
Methods	<ul style="list-style-type: none">• 30-min IV infusion on days 1-5 every 21 days• 8 dose levels from 120 – 2060 mg/m². 1 patient/dose level until tox, then 3+3
Results	<ul style="list-style-type: none">• 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%)<ul style="list-style-type: none">» 2 rectal cancer patients had durable SD (6-7 cycles at 500-750 mg/m²)» 1 hepatocellular carcinoma patient had durable SD (3 cycles at 330 mg/m²)» 1 carcinoid tumor patient had durable SD (8 cycles at 2,060 mg/m²)• PK at dose level 5 (1050 mg/m²) was 36,000x the IC₅₀ value to inhibit GGT-1 in vitro



INDIANA UNIVERSITY

RARE DISEASES CAN TRANSFORM SMALLER COMPANIES

- Rare diseases (<200,000 patients in US) can present big opportunities for smaller companies
- Markets may be too small for some Big Pharma, but are big enough to transform smaller companies
- Attractions of rare diseases
 - » Typically much **smaller trials** required
 - » Lower development **cost**
 - » Faster development **time**
 - » Support from regulators, including potential **expedited review**
 - » **Guaranteed market exclusivity** post approval (irrespective of patent status) 7 years in US; 10 years in EU
- Implications for a small biotech:
 - » Typically require **fewer resources**
 - » Means a company is not forced to partner earlier than it would like
 - » Ability to find a niche with **less competition**
 - » Small patient populations **may not require a large sales force**. Patient can be well informed and networked with other patients with the same disease

BIG OPPORTUNITY IN RARE HEMATOLOGICAL NICHE

- Recent discovery that RhoA mutations are a key driver in certain haematological cancers
- Some of these conditions are characterized **high unmet need**:
 - » Rare
 - » Poor prognoses with short median survival
 - » Very few existing treatment options
 - » Very few drugs in development
- **PTX-100 is uniquely positioned to address this unmet need due to its mechanism of action**
- Prescient recently lodged new patent application in this area.
- Case study : Folutyn (Spectrum Pharmaceuticals)
 - » For relapsed & refractory Peripheral T-cell lymphoma
 - » 5,600 cases/year in US
 - » Approved on overall response rate of 27%
 - » Currently priced at US\$450,540 per year



RAS PATHWAY, AND RHOA IN PARTICULAR, ARE IMPLICATED IN A NUMBER OF HEMATOLOGICAL MALIGNANCIES

- Angio-immunoblastic T-cell lymphoma (AITL) is a rare lymphoma with a substantial unmet clinical need
 - » **Around 70% of AITL patients harbor RhoA mutations**
- Other heme malignancies where **RhoA** may be implicated:
 - » Peripheral T-cell lymphoma not otherwise specified (PTCL-NOS) (Mutant RhoA in 18 % of patients)
 - » Adult T-cell leukemia/lymphoma (ATL) (Mutant RhoA in 15 % of patients)
 - » Burkitt Lymphoma (Mutant RhoA in 8 % of patients)
 - » Diffuse large B-cell lymphoma (DLBCL) (Mutant RhoA in 5 % of patients)
- Many of these diseases have poor prognoses and are under-served by current available treatments
- In addition, **N-Ras** and **K-Ras** contribute to several heme malignancies including:
 - » Acute Myeloid Leukemia (AML)
 - » Chronic Myelomonocytic Leukemia (CMML)
 - » Juvenile Myelomonocytic Leukemia (JMML)
 - » Multiple Myeloma (MM)
 - » Ras-associated Autoimmune Leukoproliferative Disorder (RALD)

AITL IS A RARE DISEASE WITH FEW TREATMENT OPTIONS AND POOR PROGNOSIS

- Angioimmunoblastic T-cell lymphoma (AITL) is rare, aggressive type of T-cell lymphoma
- Mostly effects the elderly
- Accounts for 1-2% of all non-Hodgkin lymphomas
- Estimate ~ 1,000 new cases in US per year
- 70% of AITL are driven by RhoA mutations
- Prognosis is poor; 1-3 years median survival

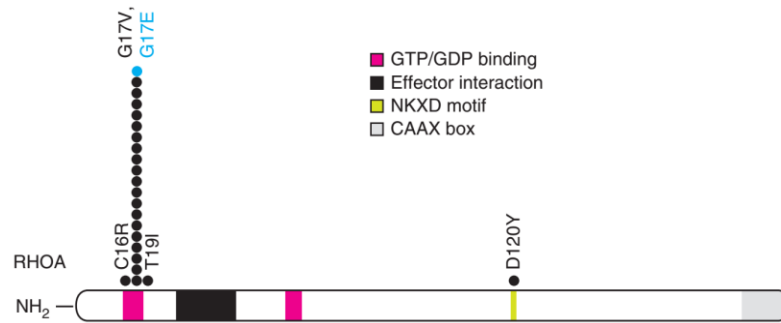
Treatment

- Very few treatment options
- Steroids are used to treat symptoms
- Multi-agent chemotherapy is currently used, but effects are short term and are associated with early relapse
- Very few drugs in development for AITL; no current RhoA inhibitors
- **Treatment of AITL represents a high unmet need**
- **PTX-100 is uniquely positioned to address this unmet need**

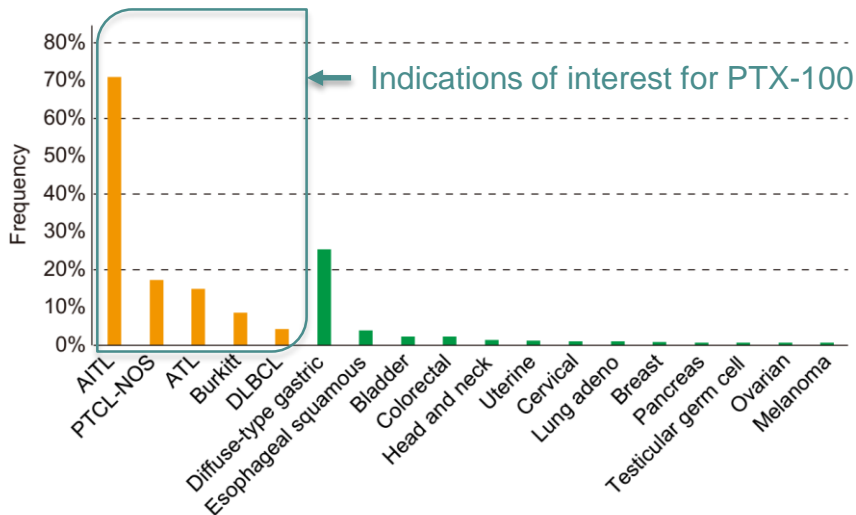
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LACASCE, AS; BLOOD; 2014; 123: 1293-1296

RHOA MUTATIONS PLAY A PROMINENT ROLE IN AITL PATHOGENESIS

Schematic of RhoA protein structure, with alterations



Frequency of RhoA mutations in human malignancies

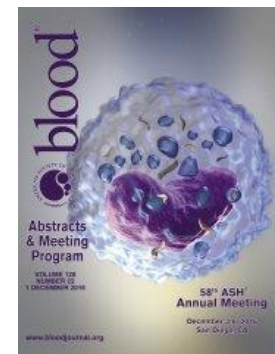


- AITL originates from follicular T helper cells and is characterized by the presence of RhoA G17V mutation together with genetic alterations
- RhoA G17V plays a prominent role in the pathogenesis of AITL via disruption of RhoA signalling.
 - » Fails to incorporate GTP in response to activated guanine exchange factors (GEF)
 - » Fails to interact with rhotekin (effector protein)
 - » Lack of RhoA activation is not the result of defective RhoA-GEF interaction

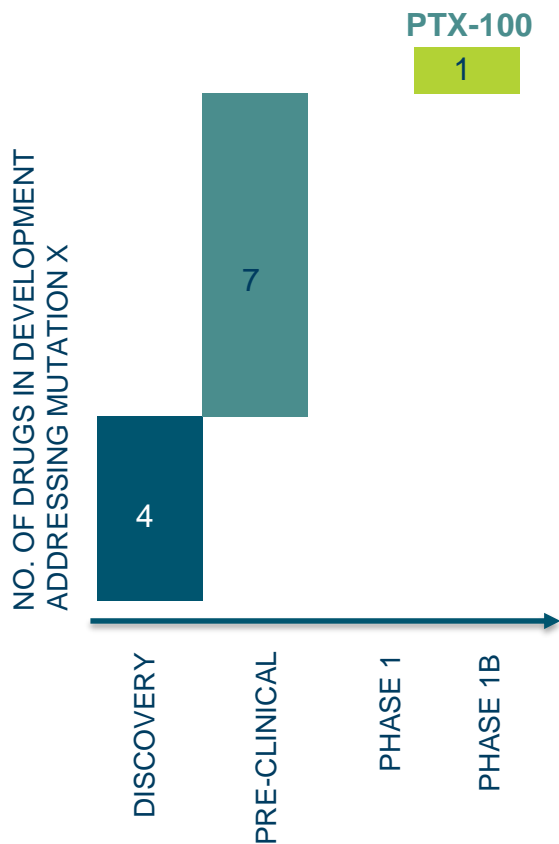
nature
genetics



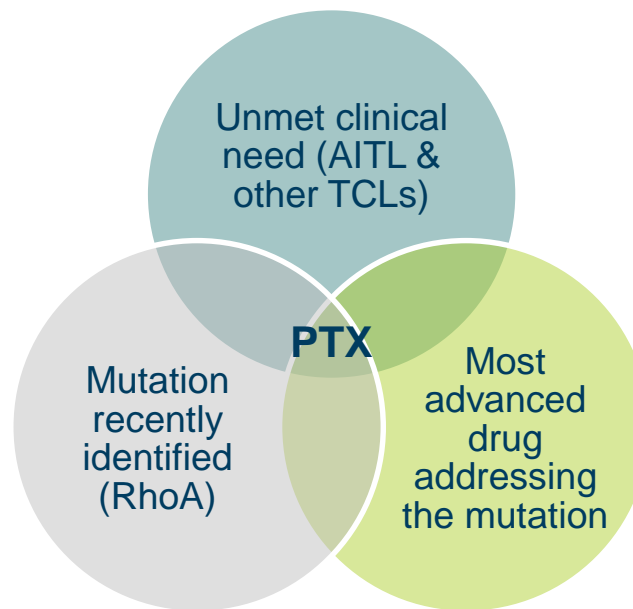
Experimental
Hematology



PTX-100 THE MOST ADVANCED DRUG TARGETING RHOA



- Only 12 RhoA inhibitors in development in oncology
 - » No others are in the clinic
 - » None are in hematology indications
 - » PTX-100 is the most advanced, with Phase 1 trial in solid tumours completed
- **PTX-100 has a head start and unique position in RhoA mutant lymphomas**

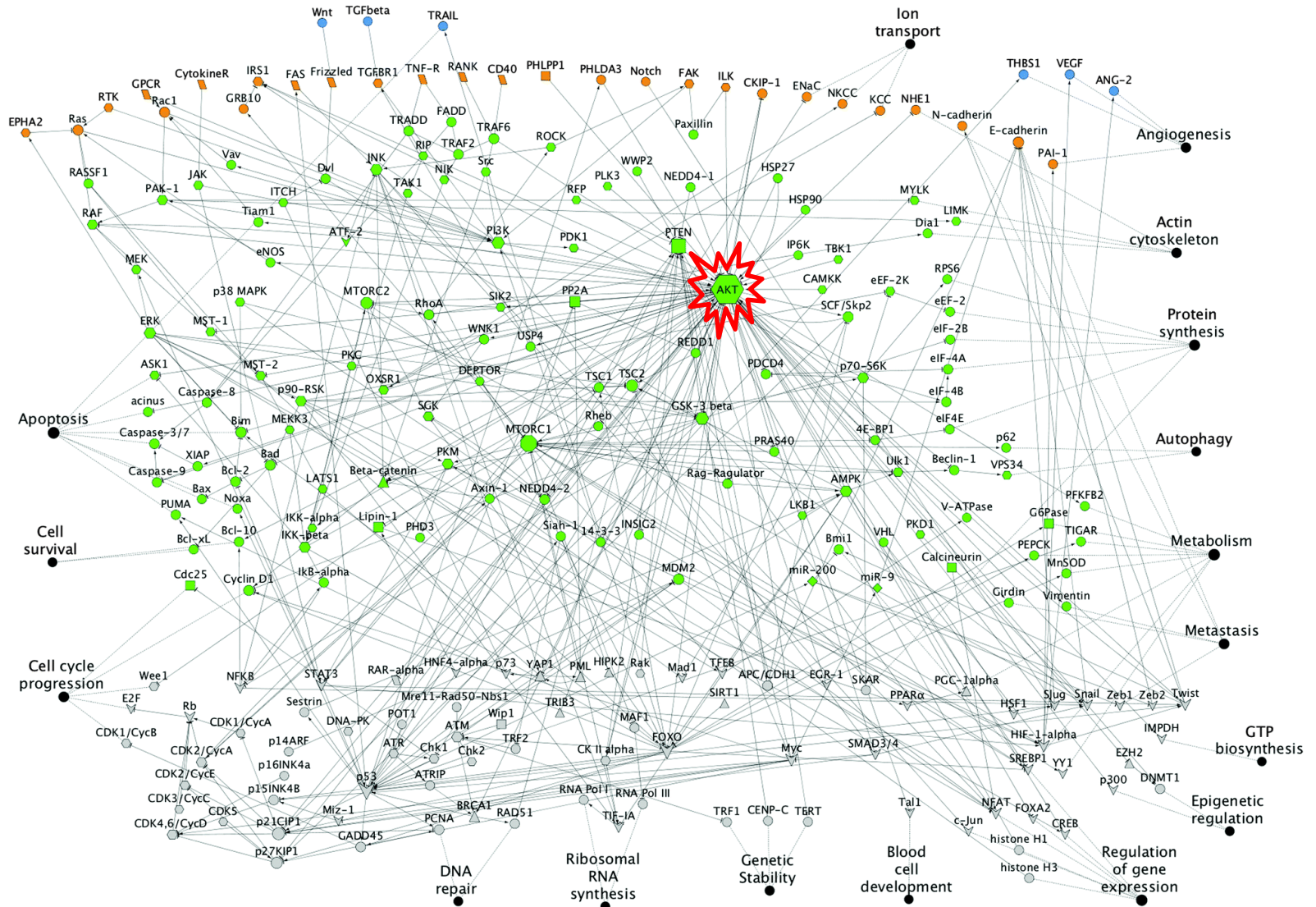


PTX-200

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



AKT IS A MASTER SWITCH FOR CELLULAR GROWTH & SURVIVAL



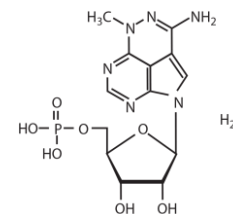
AKT REMAINS AN IMPORTANT DRUG TARGET

- 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

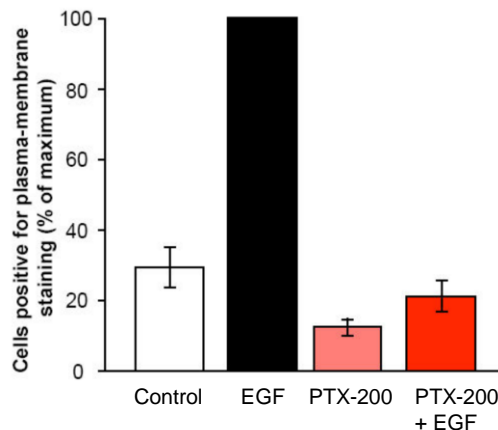
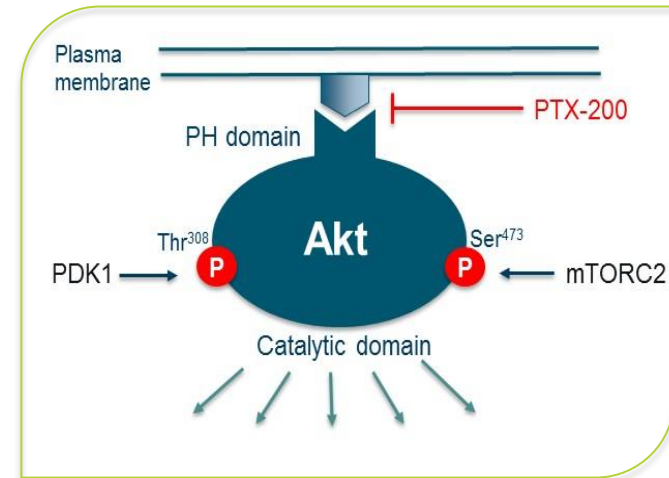
- PTX-200 (TCN-P) is a small molecule Akt activation inhibitor that is highly selective for killing tumors with hyperactivated Akt
- **Inhibits Akt without the toxicity** of other attempts
- **Anti-proliferative AND pro-apoptotic**
 - Selectively inhibits regulatory T cells¹
 - » Significant anti-tumor 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)

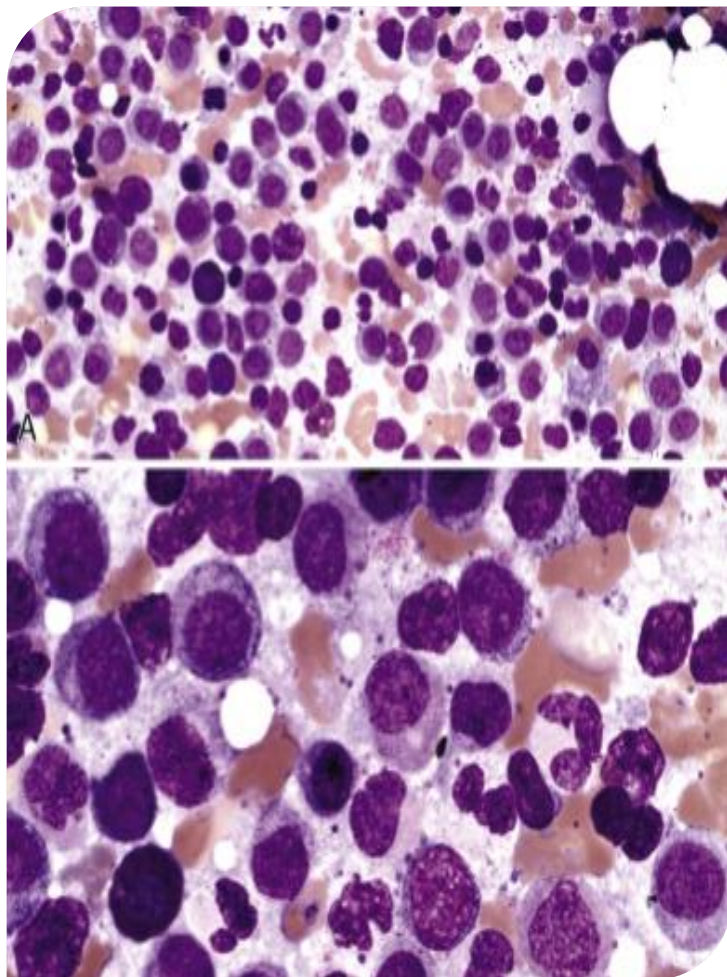


PTX-200: NOVEL AKT INHIBITION VIA PH DOMAIN BINDING

- 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 Ser⁴⁷³ and Thr³⁰⁸
- By inactivating Akt, PTX-200 suppresses the multitude of downstream targets of Akt
 - » 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**



ACUTE MYELOID LEUKEMIA OVERVIEW



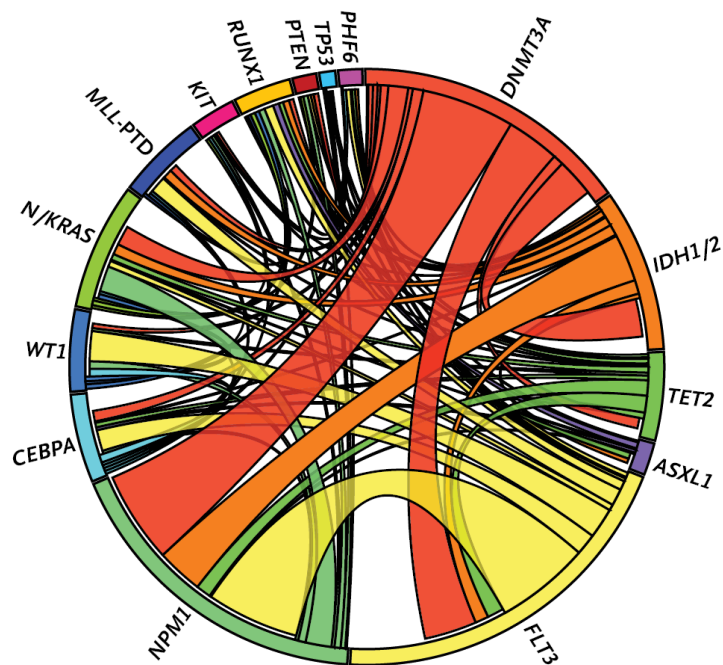
- AML is a type of cancer that affects the blood and bone marrow
 - » Patient cannot produce normal blood cells
 - » Blood cells cannot function properly nor fight disease
- Progresses very quickly; 5 year survival only 25%
- More common in adults over 60 years old, so the market is growing rapidly in 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!**
- **PTX-200's approach mirrors other current successful development approaches in AML of targeted therapies complementing a "backbone" of 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
 - » 1st cohort at 25mg/m² – safe and tolerated, no DLTs, with early signals of efficacy
 - » 2nd cohort at 35 mg/m² now open

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

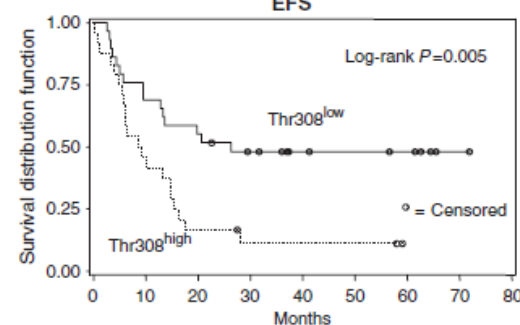
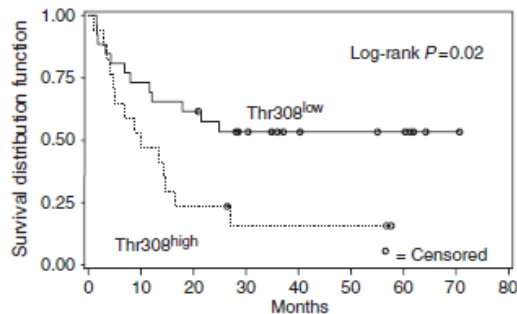
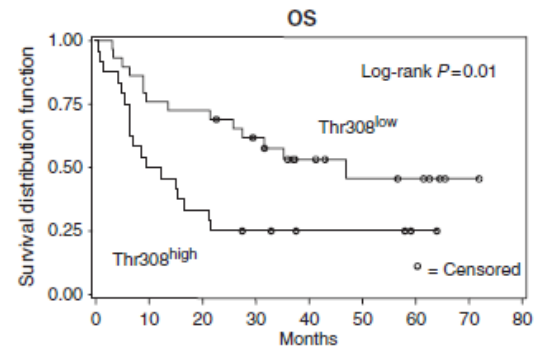
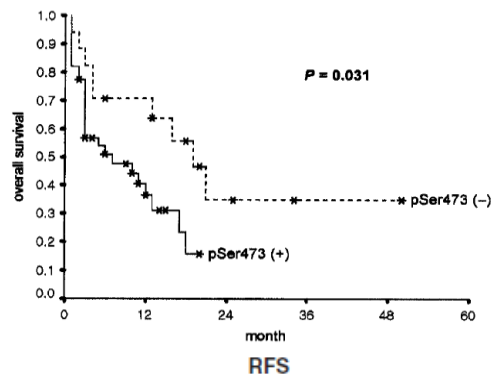


Gene	Overall Frequency (%)
FLT3 (ITD, TKD)	37 (30, 7)
NPM1	29
DNMT3A	23
NRAS	10
CEBPA	9
TET2	8
WT1	8
IDH2	8
IDH1	7
KIT	6
RUNX1	5
MLL-PTD	5
ASXL1	3
PHF6	3
KRAS	2
PTEN	2
TP53	2
HRAS	0
EZH2	0

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

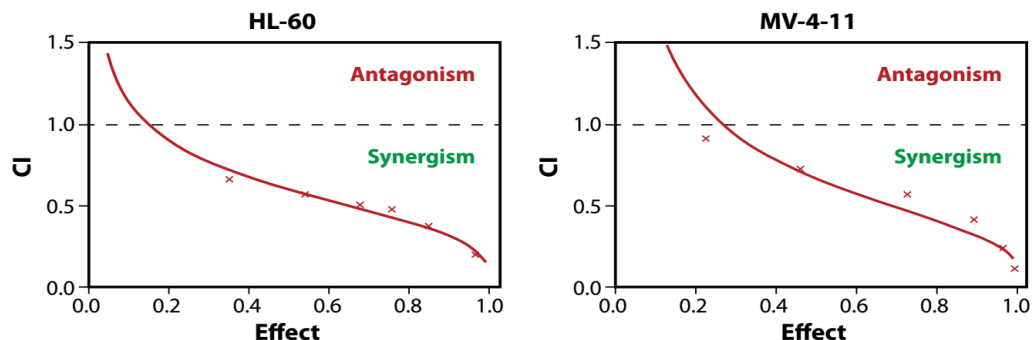
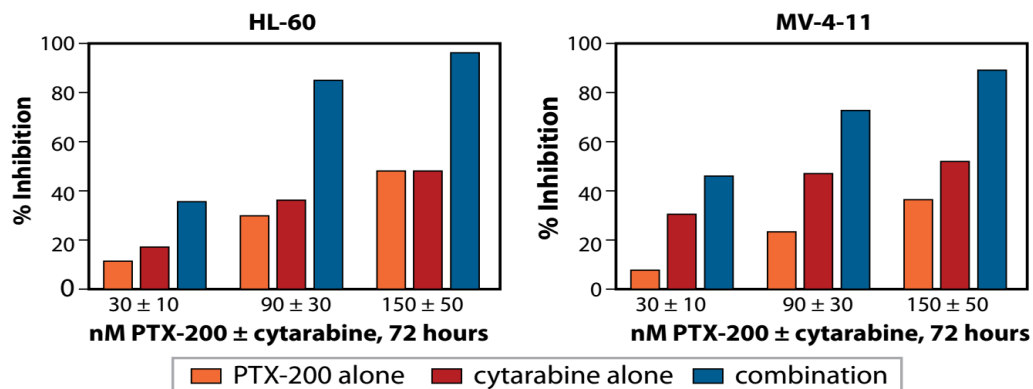
HIGH P-AKT CORRELATED WITH INFERIOR SURVIVAL IN AML

- Frequent constitutive Akt activation (phosphorylation) in AML
 - » Constitutive phosphorylation (Ser⁴⁷³ and Thr³⁰⁸) of Akt in AML compared to normal bone marrow cells in 44 out of 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 Ser⁴⁷³ and Thr³⁰⁸**



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. $1 + 1 \geq 2$)
- 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**



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

Patients	<ul style="list-style-type: none">• 32
Trial Centers	<ul style="list-style-type: none">• MD Anderson & Moffitt
Patient Inclusion	<ul style="list-style-type: none">• Advanced hematologic malignancies (mainly AML)
Study Objectives	<ul style="list-style-type: none">• To establish dosing regime and biological dose
Methods	<ul style="list-style-type: none">• 1 hour IV infusion on days 1, 8, and 15• Cycle repeated every 21 days
Results	<ul style="list-style-type: none">• 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%)<ul style="list-style-type: none">» 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

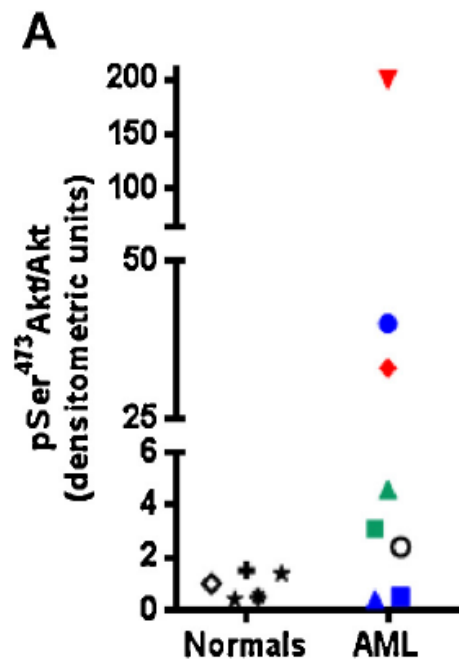


Published Leuk Res. 2013
Nov;37(11):1461-7

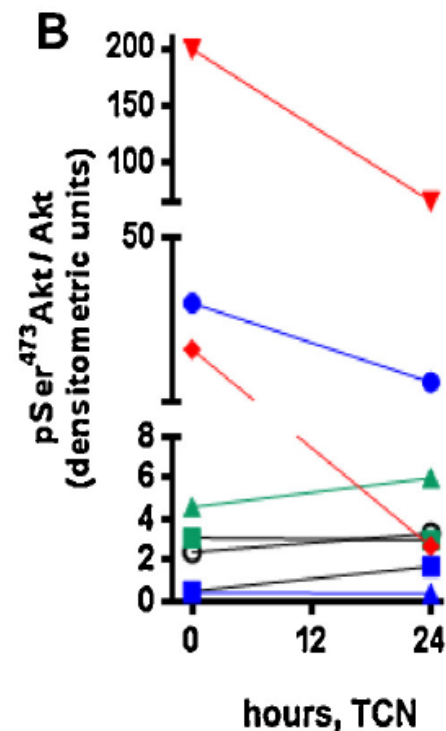
PTX-200 REDUCED P-AKT IN AML BLASTS

- 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



COMPELLING EVIDENCE FOR PTX-200 IN AML

Efficacy hypothesis	PTX-200 Evidence
<ul style="list-style-type: none"> High p-Akt is correlated with inferior survival in AML 	<ul style="list-style-type: none"> PTX-200 decreased pAkt in AML blasts
<ul style="list-style-type: none"> Inhibiting p-Akt improves response to chemo in the clinical setting 	<ul style="list-style-type: none"> PTX-200 decreased pAkt in AML blasts, suggesting this method of reducing pAkt would similarly improve clinical outcomes
<ul style="list-style-type: none"> Phase I achieved safety? 	<ul style="list-style-type: none"> Yes
<ul style="list-style-type: none"> Any evidence of clinical benefit? 	<ul style="list-style-type: none"> 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.
<ul style="list-style-type: none"> Is there a comparable with any other attempted Akt inhibitor in AML? 	<ul style="list-style-type: none"> PTX-200 had more compelling results than another Akt candidate MK2206 in Phase1 AML. (MK2206 development has since been discontinued by Merck)
<ul style="list-style-type: none"> » MK2206 successfully demonstrated apoptosis of AML cell lines in vivo, but failed to meaningfully inhibit p-Akt in the clinical setting 	<ul style="list-style-type: none"> » PTX-200 successfully inhibited p-Akt in the clinical setting
<ul style="list-style-type: none"> » MK2206: Only 1 response out of 19 patients (5% SD) 	<ul style="list-style-type: none"> » 17 out of 32 achieved stable disease (53% SD)
<ul style="list-style-type: none"> » MK2206 failed at MTD 	<ul style="list-style-type: none"> » Succeeded well below MTD
<ul style="list-style-type: none"> How does it combine with current standard of care? 	<ul style="list-style-type: none"> PTX-200 is highly synergistic with cytarabine in AML cells
<ul style="list-style-type: none"> Lessons from other trials currently running? 	<ul style="list-style-type: none"> In current Phase 1b breast & ovarian cancer trials for PTX-200, interim analysis showed encouraging efficacy
<p align="center">→ PTX-200 has lots of supportive data and efficacy signals that combine to give confidence leading into the Phase 1b/2 trial.</p>	

PHASE 1B AML TRIAL UNDERWAY

- Phase 1 results with PTX-200 (monotherapy) very encouraging
- Now PTX-200 + cytarabine in refractory or relapsed acute leukemia
 - » 15 -18 patients
 - » 3+3 design, single arm
 - » Up to 4 dose levels of PTX-200 starting at 25 mg/m² (days 1, 8, 15)
 - » Cytarabine held constant at 400 mg/m² as continuous infusion (days 2-6)
- Professor Jeff Lancet at Moffitt Cancer Center leading the trial
- Yale Cancer Center and Kansas University Medical Center also participating in trial
- **First cohort successfully completed (announced March 8)**
 - » 3 AML patients treated at 25 mg/m²
 - » Early signs of efficacy
- Now at second cohort at 35 mg/m²



Jeffrey E Lancet, M.D.
Principal Investigator



BREAST CANCER OVERVIEW



- Breast cancer market currently US\$10 B; due to double by 2023
- HER2- breast cancer has “flown under the radar” of drug developers, due to high profile successes in HER2+ drugs...but **~80% of breast cancers are still HER2-** (TNBC & ER+)
- Comparative lack of new drug development for HER2- patients, despite the need
- Evidenced by ASCO issuing a new practice guidelines in 2014 encouraging trials for new HER2- drugs
- **pAkt** overexpression is an **adverse prognostic factor** for breast cancer and correlated with **worse disease-free survival**
- **MSKCC identified utility of Akt inhibition in ER+ & TNBC (2017)**
- **Roche Akt inhibitor ipatasertib (ATP mimic) demonstrated positive Phase 2 data in metastatic TNBC (ASCO 2017)**
- **PTX’s targeted niche: preoperative (neoadjuvant) therapy for HER2- disease**

IMS HEALTH

BOYLE, ET AL; ANN ONCOL (2012) 23 (SUPPL 6): VI7-VI12.

PARTRIDGE AH, RUMBLE RB, CAREY LA, ET AL. AMERICAN SOCIETY OF CLINICAL ONCOLOGY CLINICAL PRACTICE GUIDELINE. JCO 2014

THE PROGNOSTIC VALUE OF PHOSPHORYLATED AKT IN BREAST CANCER: A SYSTEMATIC REVIEW SCIENTIFIC REPORTS | 5 :7758, 2015

CANCER RESEARCH PUBLISHED ONLINE ON JUNE 26, 2014

HYMAN, ET AL; J CLIN ONCOL 2017 JULY 10; 35(20):2251-2259

KIM, ET AL; LANCET ONCOL 2017 AUG 8 PII: S1470-2045(17)30450-3

PHASE 1B BREAST CANCER TRIAL COMPLETED

- PTX-200 in combination with paclitaxel, followed by AC (doxorubicin & cyclophosphamide)
- Patients with metastatic and locally advanced HER2- breast cancer
 - » Albert Einstein College of Medicine Montefiore Medical Center and the H. Lee Moffitt Cancer Center
 - » Single arm
 - » Exploring 3 dose levels of PTX-200 15 -35 mg/m² (3/4 weeks up to 9 doses)
 - » Paclitaxel 80mg/m²/week x 12 weeks
 - » Expansion cohort: dose-dense AC every 2 weeks
- 29 patients dosed; 12 in expansion cohort at 35 mg/m²
- **Preliminary efficacy on 8 patients encouraging:**
 - » 1 complete response
 - » 4 partial responses
 - » 2 stable disease
 - » 1 progressive disease
- 5 patients from Phase 1b qualifying for Phase 2 analysis
- Company has paused recruitment following recent adverse event
- Revising risk management and patient protocols, to ensure superior safety in a high risk patient group



Joseph Sparano, M.D.
Principal Investigator



Albert Einstein College of Medicine
OF YESHIVA UNIVERSITY



Heather Han, M.D.



OVARIAN CANCER OVERVIEW



- One of the most common cancers in women - increasing with an ageing population
- Due to reach US\$1.7 B by 2019
 - » Market size currently constrained by old generic drugs that just aren't good enough
- Standard of care has not changed in decades (often generic paclitaxel & carboplatin)
 - » Initially effective, with 70% of patients entering remission, but...
 - » ...almost all patients eventually relapse
 - » They have become chemoresistant
- **There remains a severe gap in the market for new drugs for relapsing patients and platinum resistant patients**
- **This is the gap that PTX is pursuing in ovarian cancer**

PHASE 1B OVARIAN CANCER TRIAL

- 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 underway (recruitment on hold)
- Currently recruiting at H. Lee Moffitt Cancer Center
- Up to 12 patients with an additional 18 in expansion cohort
- **Now at second dose level**



Robert Wenham, M.D.
Principal Investigator



VALUE PROPOSITION



PTX OPERATES IN AREAS WITH RICH DEAL ACTIVITY

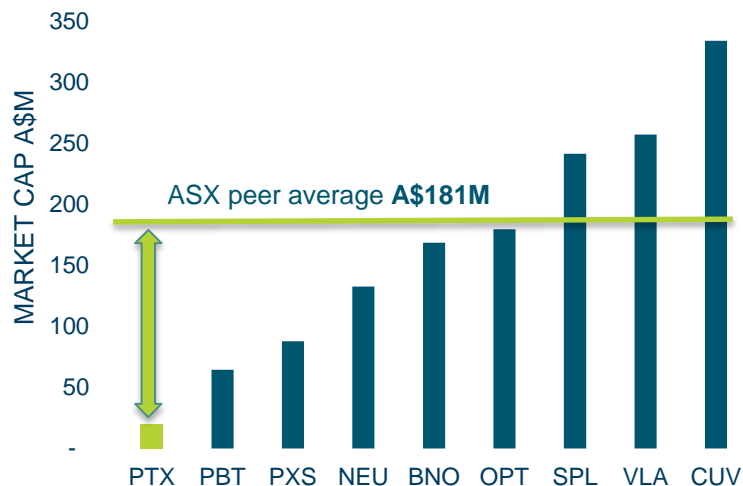
Deals¹ since Jan 2016

	# transactions	Median deal size (US\$M)
AML	44	143
Ovarian cancer	36	234
Breast cancer ²	30	255
Rare diseases (oncology) ³	972	154

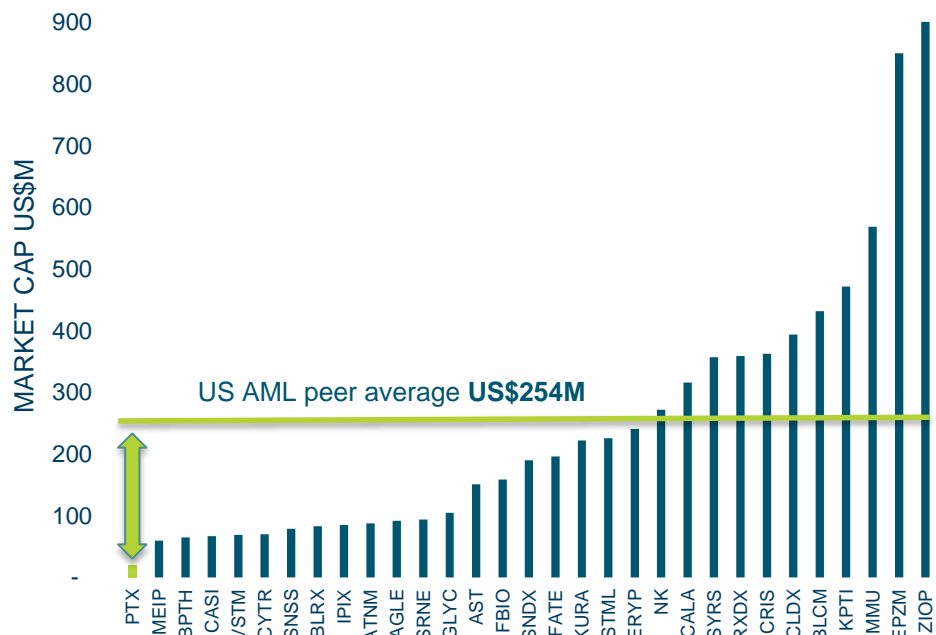
1. Deals defined as mergers, acquisitions, licenses and strategic alliances
2. Breast cancer deals only includes small molecules (like PTX-200) and does not include biologics
3. Totals all deals in oncology for indications that are rare diseases (<200,000 patients in US)

SIGNIFICANT VALUATION ARBITRAGE

Significant valuation arbitrage against comparable ASX peers...



...Arbitrage against US AML peers is even more pronounced



Comparisons are complicated by most companies in having multiple indications (as does PTX). For illustrative purposes this comparison was narrowed to US biotechs with AML drugs in development and no revenue

INVESTMENT HIGHLIGHTS

2 DRUGS » 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
- **Multiple catalysts** for value creation
- **Funded through to value-accretive catalysts**, with a fantastic share register
- Phase 1b/2 AML trial is being led by globally renowned leukemia expert, Professor Jeff Lancet
 - » Professor Lancet also led Celator Pharmaceuticals' ground-breaking VYXEOS trial in AML
- Great scientific and clinical team with a **proven record of success**
- Recent **encouraging efficacy breast cancer results, despite SAE**
- **Transformative opportunity in rare blood (heme) cancers**



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