OVERCOMING THE CHALLENGES OF KNOWN CANCER PATHWAYS WITH NOVEL TARGETED THERAPIES

Prescient Therapeutics Limited (ASX: PTX)

RODMAN & RENSHAW CONFERENCE
SEPTEMBER 2018
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INVESTMENT SUMMARY

- Novel targeted (precision) cancer therapies overcoming limitations of previous approaches to problematic pathways

- Multiple shots on goal: Akt inhibitor in 3 trials; preparing a 4th transformative study in Ras/Rho mutant cancers

- Robust body of science underpinning programs

- Seasoned scientific and clinical team with a proven record of success

- Following in the footsteps of US targeted therapy companies that have enjoyed spectacular success

- Encouraging efficacy signal in Ph1b breast cancer
DEEP, CLINICAL STAGE PIPELINE

- PTX-200 currently in three clinical trials
- Advancing PTX-100 in Rho & Ras mutant cancers - a transformative opportunity

DISCOVERY SCREENING PRE-CLINICAL PHASE 1 PHASE 1B PHASE 2 PHASE 3

PTX-200
- BREAST CANCER

PTX-200
- OVARIAN CANCER

PTX-200
- AML

PTX-100
- SOLID TUMORS

PTX-100
- RhoA & Ras MUTANT HEME CANCERS
## KEY METRICS

<table>
<thead>
<tr>
<th></th>
<th>ASX Ticker</th>
<th>PTX</th>
<th>Total Issued Capital</th>
<th>211.3 M shares</th>
<th>Options</th>
<th>57.8 M</th>
<th>Share Price&lt;sup&gt;1&lt;/sup&gt;</th>
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<td>6 month turnover</td>
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<td>22.7 M shares; A$1.6 M (US$1.3 M)</td>
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## SHARE PRICE PERFORMANCE

![Graph of share price performance](image)

## SHAREHOLDER BASE

- **HNI/retail**: 58%
- **Institutions**: 36%
- **Board**: 6%

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1 - AS AT 4 SEPTEMBER 2018
2 – AS AT 30 JUNE 2018
PTX-200
NOVEL AKT INHIBITION

Breast cancer
AML
Ovarian cancer
PTX-200
NOVEL AKT INHIBITION
OVERCOMING LIMITATIONS OF PREVIOUS ATTEMPTS AT AKT INHIBITION

- Novel mechanism of action
- Highly selective
- Anti-proliferative AND pro-apoptotic
- Selectively inhibits regulatory T cells
- Overcomes chemo-resistance
- Biomarker of PTX-200 clinical activity: p-Akt
- Completed Phase 1 trial in advanced acute leukemia patients
  » demonstrated target inhibition, tolerability and clinical activity
PTX-200 is NOT an ATP mimic/direct kinase inhibitor
» avoids off-target effects associated with ATP mimic inhibitors.

PTX-200 binds to the PH domain
➔ prevents Akt binding to the plasma membrane
➔ inactivates Akt

Control EGF PTX-200 PTX-200 + EGF

Cells positive for plasma-membrane staining (% of maximum)

Control EGF PTX-200 PTX-200 + EGF

Low pAkt tumor High pAkt tumor
AKT PLAYS A KEY ROLE IN HER2 - BREAST CANCER

80% of breast cancers are HER2-

But this is still underserved by new drugs

41% Generics

2015

$5.4B

$10.6B

2025

Market need:
Lack of pipeline agents addressing
- Resistant ER+ disease
- Neoadjuvant therapy

Ptx’s niche:
Neoadjuvant targeted therapy
for HER2- disease

Akt is adverse prognostic factor
Correlated with worse disease-free survival
Drives resistance to endocrine therapy

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; DOI: 10.1158/0008-5472.CAN-13-3382
PHASE 1B BREAST CANCER TRIAL SUCCESSFULLY COMPLETED; NOW IN PHASE 2

• PTX-200 in combination with paclitaxel, followed by AC (doxorubicin & cyclophosphamide)

• Patients with metastatic and locally advanced HER2-breast cancer

• 28 patients dosed; 12 in expansion cohort at 35 mg/m²

• 5 patients from Phase 1b qualifying for Phase 2 analysis

• Phase 2 trial currently underway in locally advanced breast cancer
WHAT DOES SUCCESS LOOK LIKE FOR THIS DISEASE?

- Studies on all sub-types of locally advanced breast cancer receiving weekly chemo reports a wide range of pCR (8-28%)

- For women with locally advanced ER+, HER2 negative breast cancer, typical expectations are:
  - pCR of 16% (11-22%)
  - ORR of 25%
  - Treatment with palbociclib + fulvestrant shows ORR of 25%; almost all were partial responses

- Whilst Prescient is not measuring PFS in this study, pCR is recognised by the FDA as an endpoint to accelerated approval

- A meaningful improvement on these response rates would be seen as very encouraging

HORTOBAGYI, G, ET AL; J CLIN ONCOL 23:5983-5992; 2005
SPARANO, J, ET AL; BREAST CANCER RES TREAT; JUNE 2014
SPARANO, J, ET AL; BREAST CANCER RES TREAT; JUNE 2013
**PHASE 1B EFFICACY RESULTS VERY ENCOURAGING**

**Metastatic & locally advanced patients**

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<thead>
<tr>
<th></th>
<th>ER+</th>
<th>Triple negative</th>
<th>Total</th>
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<tbody>
<tr>
<td>pCR/CR</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>PR</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>SD</td>
<td>1</td>
<td>2</td>
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</tr>
<tr>
<td>PD</td>
<td>0</td>
<td>2</td>
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</tr>
<tr>
<td><strong>ORR</strong></td>
<td><strong>75%</strong></td>
<td><strong>33%</strong></td>
<td><strong>50%</strong></td>
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**Locally advanced patients**

<table>
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</tr>
<tr>
<td>PD</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>ORR</strong></td>
<td><strong>100%</strong></td>
<td><strong>100%</strong></td>
<td><strong>100%</strong></td>
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</table>

**Visual representation of response by breast cancer sub-type**

**Receptor status**
- ER+
- Triple negative

**Disease status**
- Locally advanced
- Metastatic

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PCR=PATHOLOGICAL COMPLETE RESPONSE; CR=COMPLETE RESPONSE; PR=PARTIAL RESPONSE; SD=STABLE DISEASE; PD=PROGRESSIVE DISEASE; ORR=OVERALL RESPONSE RATE (=PCR/CR + PR)
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 advanced hematologic malignancies (mainly AML) 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 p-Akt in AML patient blasts

• Phase 1b trial now underway (PTX-200 + cytarabine) under the leadership of Prof Jeff Lancet

• Recently granted Orphan Drug Designation by FDA
• AML is a mutationally complex disease. It is exacerbated by the fact that mutations may be gained or lost at relapse.

• Many different types of mutations (and combinations of mutations) result in hyperphosphorylation of Akt:
  » In fact, 72% of AML patients have high p-Akt.
  » As a case in point, FLT3-ITD induced growth and survival is dependent upon Akt mediated signaling.

• PTX-200 has the potential to complement other targeted therapies, and capture what they cannot.

Many mutations, and combinations of mutations, result in high p-Akt (72% of AML patients)
HIGH P-AKT CORRELATED WITH INFERIOR SURVIVAL IN AML

» p-Akt/Akt correlated with inferior survival in AML

» PTX-200 reduces pAkt patients’ in AML blasts
• Phase 1 results with PTX-200 (monotherapy) very encouraging

• Now PTX-200 + cytarabine in refractory or relapsed acute leukemia

• Professor Jeff Lancet at Moffitt Cancer Center leading the trial

• Yale Cancer Center and Kansas University Medical Center also participating in trial
  » 13 patients with cytarabine held constant at 400 mg/m2 as continuous infusion (days 2-6)
  » 2 CRs
  » Additional arm with Cytarabine held constant at 200 mg/m2 as continuous infusion (days 2-6)

• Granted Orphan Drug Designation by FDA
PHASE 1B OVARIAN CANCER TRIAL UNDERWAY

- 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 to overcome cisplatin resistance and synergize with cisplatin in pre-clinical studies
- Phase 1b underway
- Currently recruiting at H. Lee Moffitt Cancer Center
- Up to 12 patients with an additional 18 in expansion cohort
- Now at second dose level
PTX-100
PHASE 1 IN SOLID TUMORS COMPLETED
NOW PURSUING A TRANSFORMATIVE OPPORTUNITY IN RAS & RHO MUTANT CANCERS
PTX-100
FIRST IN CLASS, FIRST IN MAN
GGT-1 INHIBITOR OF RAS PATHWAY

• Single agent activity in mouse models of various cancers
• Combination therapy is also very effective, efficacy in mutant Ras tumors
• Reduces cancer stem cell population in animal models
• p27 a potential companion diagnostic for PTX-100
• Completed Phase 1 trial in advanced solid tumors
  » well tolerated, large therapeutic index, patients achieved stable disease
• PTX-100 recently discovered to also inhibit a novel cancer causing pathway FBXL2 important in PTEN defective cancers
RAS PATHWAY IS AN IMPORTANT BUT ELUSIVE TARGET

- Ras mutated in 30% of all human cancers and 90% in certain cancers
  - 3 million new cancers diagnosed worldwide each year with Ras mutations

- Mutant Ras tumors are often unresponsive to current treatments

- Still a lack of suitable targeted therapies for Ras

- Rho now identified as a target in its own right

- Targeting Ras directly has proven elusive; **PTX-100 disrupts the Ras pathway by inhibiting the activation of Ral, Rac and Rho**
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.
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**
RHOA MUTATIONS PLAY A PROMINENT ROLE IN TCL PATHOGENESIS

- RhoA G17V mutation is a driver of pathogenesis of AITL, PTCL-NOS
- Other RhoA mutations driver a number of other malignancies
- PTX-100 a pan RhoA inhibitor regardless of mutation
PTX-100 THE ONLY DRUG TARGETING RHOA

• Only RhoA inhibitor in the clinic

• Phase 1 trial in solid tumours completed

• PTX-100 has a unique position in RhoA mutant malignancies
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• Transformative opportunity in Ras/Rho mutant cancers

• Following in the footsteps of US targeted therapy companies that have enjoyed spectacular success

• Encouraging efficacy signal in Ph1b breast cancer
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