INVESTOR NEWSLETTER

Personalised therapies targeting chemotherapy resistant cancers

LETTER FROM THE CEO

Dear Shareholders,

I am pleased to present our investor newsletter, which will be published on a regular basis with the intention of keeping you informed of the developments at Prescient Therapeutics. We have made steady and positive progress in all our clinical trials over recent months - including the advancement of our PTX-200 Phase 1b breast cancer trial meeting its pre-specified safety criteria, with preliminary efficacy results looking quite encouraging. However, on May 29, we were saddened to inform the market that the last of 29 patients in our Phase 1b trial of PTX-200 for breast cancer encountered a serious adverse event (SAE) and passed away. Regrettably, patient deaths are an unfortunate reality of trialling new drugs on very sick, often unstable, cancer patients.

This patient, who had metastatic breast cancer, and a poor prognosis, was being treated with paclitaxel and PTX-200, as well as pioglitazone to treat diabetes, and experienced liver failure.

Although a high-risk patient, we took the decision to immediately pause recruitment across all of the PTX-200 trials – in line with our company operating procedures. As required, we also immediately informed the FDA.

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From here, we are now currently reviewing trial protocols, and updating our risk mitigation plan to ensure the protection of patients and maintain that we are running a trial that has the best chances of meeting its efficacy endpoints. We are also working with the FDA to ensure any recommendations they provide are implemented, and to ensure they are fully aware of, and approve of, any protocol changes we make.

Once the investigation of this event is complete, and we have implemented the FDA's modifications and recommendations into our protocol, the FDA can lift the clinical hold and the trials can resume.

While this does create a delay, of possibly several months, we are taking the prudent action that will support a successful outcome over the longer term. Prescient will continue to update on the status of the trial hold in a timely manner, and remain committed to complete transparency with our shareholders as we move forward.

PTX-200 has previously passed its safety requirements in the clinical setting as a single agent; we have a team that is highly experienced in running clinical trials and working with the FDA and, as such, we remain confident of resuming the trials in due course. PTX-200 remains an important component of our development pipeline.

Over the past quarter, we have made solid progress including:

- Completed dosing of the first cohort of patients in the Phase 1b / 2 trial of PTX-200 in Acute Myeloid Leukemia (AML);
- Granted orphan status for PTX-200 in AML – which will assist in fast-tracking our development;
- Welcomed prestigious medical institute Kansas University as an additional site in this trial;
- Received four patents that secure the novelty of PTX-200; one of these was granted in Europe and three in the US. Securing additional patent protection in both these markets is a crucial step in our continued efforts to create shareholder value and move closer to the commercialization of our unique assets.

We are also very excited about our other novel compound, PTX-100. PTX-100 targets a very highly-sought after cancer pathway called Ras, and has a unique mechanism of action. Although early days, our focus on rare hematological cancers with PTX-100 is attracting particular interest and I look forward to informing you of our progress in this exciting area.

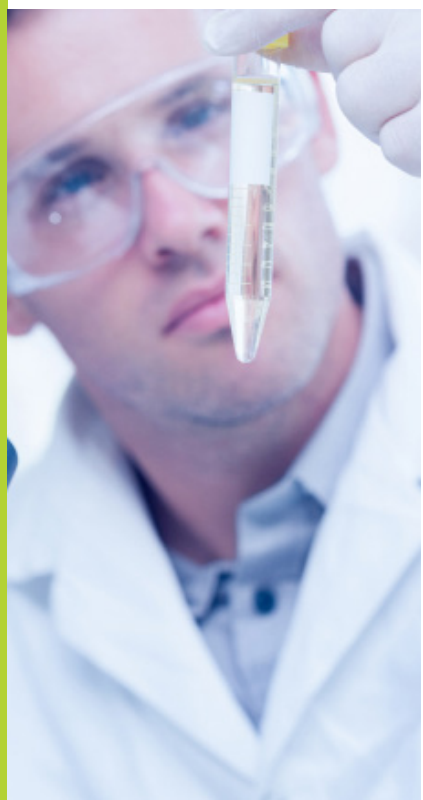
Before signing off I would like to acknowledge my fellow Director, Paul Hopper, and his immense contribution to Prescient. We recently announced that Paul would transition to a Non-Executive Director role, effective from 1 July this year. As an Executive Director, Paul has been very "hands–on" in the Company's operations since 2014.

Paul's transition to a traditional Non-Executive Director's role was a planned move and is appropriate given the bolstering of Prescient's operational team, with highly experienced professionals assuming specific roles. I thank Paul for his involvement and support to date and I am pleased to continue working with him at Board level as we guide Prescient forward.

We look forward to continuing to advance Prescient's valuable research, resuming the clinical trials, and building ongoing awareness of the Company as a leader in the development of drug therapies to treat challenging cancers.

Sincerely,

Steven Yatomi-Clarke
CEO & Managing Director



PTX-100 and Rare Hematological Cancers

The Ras pathway, which PTX-100 can target, is a key driver in certain haematological cancers. Prescient is pursuing a transformative indication using PTX-100 in rare haematological cancers, building on the previous Phase 1 trial of PTX-100 in advanced solid tumours. While the mutation of the Ras pathway occurs in 30% of all human cancers and 90% of all certain cancers, the discovery of its role in rare haematological cancers is fairly new.

Currently, there are very few treatment options for these types of cancers.. As it stands, there are only 12 drugs in development targeting this mutation, however, PTX-100 is the only one in the clinical stage of development - making PTX-100 the most clinically advanced drug in this space. PTX-100 already has strong proof of concept data behind it in solid tumours, with the validation of the role of this mutation in other forms of cancer. We are well placed to pursue further trials using this compound and advance as an authority targeting this pathway.

This is a very important and exciting opportunity that can add significant value to this company. We have a strong competitive position in this space, and the lack of treatments currently available means this has the potential to be very attractive to possible partners.

Along with initiating the trial, we will build additional value around this asset by expanding the intellectual property position with further publications in peer reviewed journals expected to be published.

How does PTX-100 work?

PTX-100 works by blocking the cancer growth enzyme Geranylgeranyl Transferase (GGT), thereby disrupting the Ras pathway. Patients with Ras mutant tumours are often unresponsive to current treatments, and patients with these types of cancers are in critical need of suitable targeted therapies. Despite this prevalence, there are currently no drugs that effectively target Ras mutant cancers.

Mutation of the Ras pathway

The disruption of the Ras pathway is a common factor in rare hematological cancers. Patients with these diseases have very few treatment options and, unfortunately, survival rates are discouraging. Recent research has revealed a specific mutation that drives many of these blood cancers; PTX-100's unique mechanism of action may help address this mutation. Furthermore, Prescient is not aware of any other drug in the world in clinical stage research that targets this problematic mutation. This gives us a very unique positioning and a big head start on the competition in addressing these rare blood cancers.

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Precision Medicine and its Role in Cancer Treatment

Precision medicine is an individualized approach to medicine that considers the differences in ones' genes, environment, and lifestyle to identify the most preferential treatment and prevention strategies specific to that person. Contrary to a generic 'one size fits all' approach, precision medicine is premised on the idea that because individuals and their genes are unique, their treatment should also be unique.

There's often a misconception that customized healthcare means creating treatments for specific people; this isn't the case. Precision medicine is about taking a new or existing treatment option, identifying the group of people (for example, based on a particular cancer mutation) it will best suit, and using the treatment for only that group instead of for everyone.

Using designated oncology treatments to target specific cancerous mutations is an example of precision medicine. Prescient employs this strategy by acknowledging that patients' tumours have different genetic mutations, and using PTX-200 and PTX-100 as targeted therapies to solely treat patients with Akt and Ras mutations.

By using precision medicine in this way, and by augmenting existing treatments, we hope that patients will have better treatment outcomes.

Why is targeting rare diseases a viable option?

Targeting rare disease might sound counterintuitive, but for biotechs it can present a very clever strategy. Pursuing therapies to treat rare diseases can present big opportunities for smaller companies, opportunities that are potentially transformative.

The FDA defines rare diseases as those affecting fewer than 200,000 patients. These markets are often overlooked by big pharma, however, they lend themselves well to smaller biotechs, due to fact that regulators, such as the FDA, have an interest in promoting development resources towards rare diseases.

Given this, the FDA will often allow smaller trials and offer other incentives to aid a lower cost and faster development timeline. Because the trials are typically smaller, they can also be quicker and cheaper to undertake. This also means that companies may be able to hold on to their programs for longer (rather than having to out-license to a partner earlier), thereby extracting greater value.

Indeed, many small biotechs in the US have been transformed when they have had drugs approved via this route, as they employ a modest sales force to access a market that can generate substantial revenues.

What's next for PTX-100 in rare hematological diseases?

Prescient is currently designing a proof of concept pre-clinical study as we work towards a transformative clinical program in this area.

PTX-100 has already completed a Phase 1 trial in advanced solid tumors at both Pennsylvania University and Indiana University, which demonstrated that it was well tolerated and achieved disease stabilization in some patients. This gives us confidence moving forward with new blood cancer trials using the same drug.

As we have done successfully for PTX-200 in AML, we will apply for orphan drug status for PTX-100; a designation provided by the FDA for treatments of rare diseases.

This status offers benefits such as: a seven-year guaranteed exclusivity in the market once regulatory approval is received,50% tax credit on US based trials, and the potential for faster review.

PTX-100 featured in prestigious scientific journal Nature

A pre-clinical study published in Nature this month highlights the efficacy of PTX-100 in inhibiting a newly identified cancer-causing pathway.

The study demonstrated new details about the tumor suppressor gene, PTEN, which is defective in 30 to 60 per cent of certain breast, brain and uterine cancers, and which was discovered recently by Professor Michele Pagano at New York University's Langone Medical Center in New York. "When defective, PTEN cannot control a protein known as FBXL2, which is thought to be responsible for cancer growth in many patients." said Professor Pagano.

Professor Pagano's study also showed that in mouse models, when administered with Prescient's drug PTX-100, plus photodynamic therapy, FBXL2 is "switched-off" allowing abnormal cells to self-destruct. Therefore, patients whose tumors harbor defective PTEN may also be more likely to respond to a combination of PTEN and photodynamic therapy.

This publication is important for Prescient on a number of levels. Firstly, it opens up a new frontier of clinical possibilities for PTX-100, adding potential value to this asset. Nature is one of the world's most cited and prestigious scientific journals.

As such, this publication provides a strong validation of this pre-clinical data. It also serves as another proof point that PTX-100 is emerging as a leader in this area of drug development, and this additional data further enhances our competitive position.

AML AND PTX-200: HIGHLIGHTS FROM OUR DISCUSSION WITH THE PRINCIPAL INVESTIGATOR AND CSO

Professor Said Sebti, Prescient Therapeutics - CSO

Professor Jeffrey Lancet, Principal Investigator - AML trial

"I believe that the ability to overcome chemotherapy resistance creates tremendous opportunity to really expand the horizons of traditional chemotherapy in AML..."

- Professor Jeffrey Lancet

Prescient has been granted orphan drug status for PTX-200 as a treatment for Acute Myeloid Leukemia (AML). In late April, we held a conference call for investors with Professor Said Sebti, our co-founder and Chief Scientific Officer and Professor Jeffrey Lancet, the Principal Investigator on our AML trial.

This was a unique opportunity to hear firsthand, where PTX-200 fits in the AML treatment landscape, and why this is a potentially very important development. This article covers the key highlights of the call (a full transcript and recording can be found on our website).

What is AML?

Acute Myeloid Leukemia (AML), is a type of blood cancer that affects the bone marrow and prevents a patient from producing normal blood cells. Innovation in this disease has been minimal over the past 40 years, and generally after initial chemo treatments most patients relapse. When considering that that survival statistics in this disease are rather grim - even by cancer standards - then it becomes evident why there is a significant amount of interest in developing new drugs for this disease.

Why is AML on the rise?

AML is more common in adults over the age of 60, so the prevalence of the disease is increasing much faster than population growth in ageing, developed economies. AML also has a shocking mortality rate; the mortality rate closely mirrors that of incidence.

We deal with a lot of poor outcomes in this disease and this is attributable to the fact that AML is a disease primarily of older individuals where treatment options are frequently less viable, more difficult, and maybe frequently associated with toxicity.

What is the premise behind PTX-200 as a treatment for AML?

Following decades of unsuccessful attempts of displacing chemotherapy as the standard of care, a new approach emerged a few years ago that uses targeted therapies against the various mutations present in AML and combines this with the backbone of chemotherapy. There are so many mutations contributing to AML and, as such, from a drug development perspective, there are many targets to develop drugs for.

This approach is yielding some very exciting developments in AML. PTX-200 works on the same approach – and is designed to work in combination with chemotherapy, specifically the main chemotherapy drug for AML, Cytarabine.

So how does PTX-200 work?

AKT is a protein that sends the signals responsible for many

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cellular processes, including the regulation of cell growth. When AKT is hyperactivated, and abnormal signals are being sent, this can result in the uncontrolled growth of cancerous tumour cells present in various cancers, such as AML, and it leaves the cells very resistant to chemotherapy. As a key oncogene enabler, AKT is an elusive target for drug developers and one that PTX-200 seeks to target.

PTX-200 works by interrupting the AKT signals and effectively switching off the AKT hyper-activation that is continually telling the mutated cancerous cells to grow, and as a result it makes the cells more receptive to chemotherapy treatment. PTX-200 is designed to work in parallel with the primary AML chemotherapy medication, Cytarabine, for maximum efficacy.
Given the rarity of AML, there are only a few other drugs currently in development, but PTX-200 works in a different way than these other inhibitors using a unique method that avoids the toxicity issues the others face.

What did the Phase 1 study tell us about PTX-200 and its potential effect on AML?

In Phase 1, we tested PTX-200 without Cytarabine, as a monotherapy, in 41 patients with refractory or relapsed acute leukemias to confirm the safety and potency of the drug in killing cancerous cells. We modelled the trial on a dose escalation format, meaning we continually increased the dose of PTX-200 to see what level was ideal; the result was a lower dosage than we had initially expected.

In the 32 evaluable patients, half experienced stable disease after one cycle of PTX-200, with three seeing a major decrease in their cancerous bone marrow cells. This trial validated that PTX-200 succeeds in invading cells and hitting, and thereby inhibiting, the correct AKT targets. It was also important for identifying the correct dosage level of PTX-200 to be used in subsequent trials and confirming the drug can be well tolerated by patients.

What is the focus for the current Phase 1b / 2 trial?

This is a Phase 1b/2 trial and for those unfamiliar with clinical trial design, a Phase 1 trial is designed to assess safety as the primary endpoint. In addition, pharmacokinetics and biologic activity of the drug is measured are also measured at this stage by some pharmacodynamics assessment to see if the drug is hitting its target and working as intended. Following this, a Phase 2 study is initiated to determine the efficacy of a drug once safety and tolerability has been established.

This trial currently being conducted is an overlap trial with a Phase 1 component and a Phase 2 component. The Phase 1 component utilises a dose escalation schema for the PTX-200 drug in combination with continuous dosing of Cytarabine. The Phase 2 component of the study, which the Phase 1 component will feed into as part of the same study, will focus on treating a more defined group of patients with AML in first relapse with the combination of continuous infusion Cytarabine plus PTX-200 administered at the maximum tolerated dose based on the Phase 1 study.
The Phase 1b design allows for several dosing cohorts of PTX-200, and we have begun dosing at the lowest dosing level of 25 mg/m2 with allowance of dose escalation of up to 55 mg/m2 - a total of four discrete dosing levels based on tolerability. Again, this is in combination with continuous infusion of Cytarabine on days one through five while PTX-200 is given on days 1, 8 and 15.

How is PTX-200 differentiated as an AML Therapy?

PTX-200 is not without its competitors in the field, and we recognise that within the next year or two there will likely be two or more agents proved for AML. Most of these agents are targeting a relatively selective group of patients with a recurring type of mutation. The benefit of a drug like PTX-200 is that it allows targeting of a very highly and commonly expressed protein in AML which gives it an opportunity to be utilised in a larger fraction of patients with this disease overall.