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Operator: Thank you for standing by and welcome to the Prescient Therapeutics AML Clinical Trial update call. All participants are in a listen only mode. There will be a presentation followed by a question and answer session. If you wish to ask a question you will need to press the star key followed by the number one on your telephone keypad.

I would now like to hand the conference over to Mr Steven Yatomi-Clarke, CEO. Please go ahead.

Steven Yatomi-Clarke: Thank you, Bethany. Welcome everyone to the AML Clinical Trial update for Prescient Therapeutics. My name is Steven Yatomi-Clarke and I'm the CEO of Prescient Therapeutics.

I feel it's important to state upfront that this is not an end of study call and therefore no data will be presented. However this call was organised in response to many requests from investors who wished to speak with our CSO and our Principal Investigator about the AML trial that we currently have underway.

I'll also like to state upfront that I acknowledge that there's a blended audience on the call today ranging from generalist investors all the way through to highly experienced biotech analysts. I've previously found that the breadth of questions from a blended audience like this tends to be very productive and can help us zoom in and out and get a more well-rounded perspective on the matter but we'll try to be cognisant of the fact that the audience is blended.

Introductions

Let's move to introductions. I'd like to introduce you to our CSO Professor Said Sebti and the Principal Investigator of our AML trial Professor Jeffrey Lancet.

Firstly Professor Sebti is our esteemed CSO. He has a decorated career studying aberrant cellular pathways that contribute to cancer and indeed has become a global expert in his field. He is Professor and Chair of the Department of Drug Discovery at the Moffitt Cancer Centre in Tampa, Florida which is the third largest cancer centre in the United States.

He is the co-inventor on both of our drugs, PTX-100 and 200 and fantastic too for us he was named by Nature Publishing Group as one of the top 20 translational researchers in the world. If that wasn't enough of an accolade, last year Professor Sebti was also

awarded the highly prestigious Outstanding Investigator Award from the NCI, the National Cancer Institute.

I'm also delighted to introduce you today to Professor Jeffrey Lancet who is the Principal Investigator of our AML trial. Dr Lancet is a genuine international authority on acute leukaemias and is recognised internationally for his clinical trial research in this field. A word of warning from personal experience, if you try to get into one of his sessions at ASH then I fear it will be standing room only, which is testament to his standing in the field. Professor Lancet's the Chair of the Department of Malignant Haematology at the Moffitt Cancer Centre where he leads an incredible team of haematologists.

I'm certain that many of you on the call today will know of Professor Lancet from his role as Principal Investigator on Celator Pharmaceuticals' transformative trial with CPX-351 otherwise known as VYXEOS. It represented a major and long overdue breakthrough in AML and only a few months after its Phase 3 trial Celator was acquired by Jazz Pharmaceuticals for $1.5 billion. It seemed to take the biotech world by surprise but I would suggest perhaps not to Jeff who identified the program as promising and backed it in its very early stages.

I don't want to embarrass Jeff any further but if I had my worldwide choice of someone to design, lead and recruit for our AML trial then Jeff would certainly be my first choice. Suffice to say we are delighted and honoured to have both Said and Jeff instrumentally involved with Prescient Therapeutics.

Agenda

I just want to give, now that the introductions are done, just an overview of the agenda of the call and I certainly realise that the audience has not dialled in to hear me talk - well not today anyway - but please bear with me as I outline today's agenda.

I'll take the new ball by leading off with providing a very brief overview of the Company in the context of our AML trial. Professor Sebti will then speak about the relevance of Akt as a target of interest in AML and provide an overview of how PTX-200 works to inactivate Akt and how it synergises with the current standard of care.

Then Professor Lancet will provide more of an overview on AML as a disease, provide the background of our Phase 1 result in this and then go on to our Phase 1b trial design and a trial update to date again with the understanding that no data will be presented here today. Professor Lancet may also provide a view on how he feels Prescient's drug PTX-200 may be differentiated as an AML therapy.

Then we'll open up the call to questions.

Introduction to Prescient Therapeutics

With the introductions and agenda behind us let's kick off the agenda. By way of an overview of Prescient Therapeutics we're an ASX listed company with Australian and US operations. We have two targeted therapies in clinical trials in oncology and one of our drugs, PTX-200, is a novel Akt inhibitor that has significant advantages over other attempted Akt inhibition which Said can discuss. PTX-200 is currently in Phase 2 trials in HER2 negative breast cancer and a 1b trial in relapse and refractory ovarian cancer.

Importantly and for the purpose of this call it's in a 1b trial in acute leukaemias and is strategically very important for us and a trial that we've got a particularly high amount of enthusiasm about.

Importantly this is not the first trial for PTX-200 in acute leukaemias having previously completed a successful Phase 1 trial as a monotherapy that clearly justified progressing this program further.

AML landscape

Just let's speak for a moment on the landscape of AML rather than the disease. As a snapshot AML is a type of blood cancer that affects the bone marrow and prevents a patient from producing normal blood cells. It is more common in adults over 60 so the prevalence is growing much faster than population growth in ageing, developed economies. There hasn't really been too much innovation in the last 40 years in this disease and after initial chemo most patients relapse. When you combine that with the fact that survival statistics in this disease are rather grim - even by cancer standards - then you can see why there is a significant amount of interest in developing new drugs for this disease.

Following decades of unsuccessful attempts of displacing chemotherapy as the standard of care a new approach emerged a few years ago using targeted therapies against the various mutations present in AML and combining this with what they call a backbone of chemotherapy. There are so many mutations contributing to AML and from a drug development perspective there are therefore many targets to develop drugs for. This approach is yielding some very exciting developments in AML.

Prescient has the same approach. PTX-200 is a targeted therapy to address the high phospho-Akt and using that in combination with standard chemo. In this regard I feel that Prescient is in the right place at the right time with this clinical approach.

That's enough from me and without further ado I'd like to pass the call now on to Professor Said Sebti, our CSO. Thank you, Said.

Said Sebti: Good afternoon everybody or good morning I should say in Australia and thank you Steve for a very generous introduction.

Akt prevalence and role in AML

What I'd like to do actually - I have five minutes - I'd like to introduce you to the cancer causing protein called Akt that PTX-200, the subject of today, targets. What I'd like to do really is to show you through the next five minutes that Akt is a major contributor to human oncogenesis with significant potential as a target for personalised precision medicine against many cancers of course including today's focus, AML.

Let me just start by saying that Akt is actually a protein found aberrantly hyperphosphorylated in many human tumours and as such it is hyper-activated persistently activated and mediates many of the hallmarks of cancer including uncontrolled tumour growth and most importantly for us today, resistance to chemotherapy. When a patient with AML has hyperphosphorylated Akt in their cells, in their tumour cells, those cells are likely to be resistant to therapy. That's an important point.

In addition, in AML itself it happens to be one of the cancers with the most prevalent phospho-Akt levels so it is about 70% to 75% of all AML patients harbour hyperphosphorylated persistently activated cancer causing Akt. That is not only cancer causing and it causes problems for resistance, it's also directly related to poor prognosis. Patients whose cells, leukaemia cells, harbour hyperphosphorylated Akt live less and they respond to chemotherapy less. That is an important point to remember as well as Dr Lancet will talk about the disease.

This cancer causing protein actually - and Steve alluded to this but I want to emphasise it further and that is that phospho-Akt, this cancer causing gene, actually mediates the action of several cancer causing genes and proteins. These are - and these are only a few of them that phospho-Akt mediates or helps cause cancer - Raf which is another cancer causing protein that uses phospho-Akt to cause cancer and that's in about 25% of

leukaemia patients, [unclear] which is also about 25% to 30% is another cancer causing gene as well as KIT as well as [unclear] kinase. All of these cancer causing genes use phospho-Akt to cause AML.

That's another point. If you take all this together, the fact that the prevalence of hyperphosphorylated Akt in AML has been shown to correlate with chemo-resistance and poor prognosis one would predict that inhibition of Akt hyperphosphorylation is a strategy that is expected to broaden the spectrum of AML patients that can be treated.

PTX-200 inactivates Akt

Prescient Therapeutics' PTX-200 does just that. It is an inhibitor which lowers the hyperphosphorylation levels of Akt in leukemic cells and therefore makes them more sensitive to chemotherapy such as the clinical trial that is ongoing right now, combining our hyperphosphorylation inhibitor PTX-200 with standard of care Cytarabine.

One could really define - and this is the point I wanted to make and said in the beginning - one can define that this targeting hyper-activated hyperphosphorylated Akt with PTX-200 as a precision medicine approach tailored to cancer patients whose tumours are addicted to hyperphosphorylated Akt for survival.

I'll end with a couple of important points and that is that we have already done several trials with PTX-200 and the good news is as a single agent PTX-200 inhibits the levels of hyperphosphorylated Akt most importantly at doses that are safe. Our ongoing clinical trials have shown so far that combining PTX-200 is safe especially when combined with chemotherapeutic agents such as Paclitaxel in our breast cancer trial, with Carboplatin in our ovarian cancer trial and in of course today's topic, combining PTX-200 with Cytarabine in AML patients.

The final point I would make is that one of the uniqueness of this drug compared to other Akt inhibitors is its mechanism of action. The Akt inhibitors other than ours are working by seeing what's called an ATP mimic and there are several kinases that - all the kinases use ATP so therefore other kinase inhibitors, other Akt inhibitors are more likely to actually lead to toxicity whereas our inhibitor works by a totally different mechanism. That maybe is the reason why it's highly selected only for those tumours that harbour hyperphosphorylated Akt. That is truly a significant differentiator from other Akt inhibitors.

I'll stop and pause right there and hand it over to Professor Jeff Lancet.

Jeffrey Lancet: Thank you Said and thank you very much Steve for the very, very kind and I think overly generous introduction. I have to say that the pleasure and privilege has really been all mine. To be able to work with a company such as yours with so much insight, knowledge, fortitude and a good plan moving forward is very refreshing and helpful and enjoyable and I'm very excited about the continuing relationship we have so thank you for that.

My name is Jeff Lancet. I'm the Chair of the Malignant Haematology Department at Moffitt Cancer Centre and also an experienced AML investigator for several years. I've been asked to provide an overview of the disease in general and to provide some background on the clinical trial and to discuss a little bit about how this treatment, PTX-200, can fit in to the overall treatment armamentarium for AML moving forward.

AML background

Yes so starting out AML is an incredibly complex and difficult disease. It's a rare disease - it's not [really] amongst the most common cancers - with an incidence that approaches 70 to 80 per 100,000 in the older age population. What I think's particularly significant about AML is its mortality rate. The mortality rate fairly closely mirrors the incidence.

We deal with a lot of poor outcomes in this disease and this is attributable to the fact that AML is (1) a disease primarily of older individuals where treatment options are frequently less viable and more difficult and maybe frequently associated with toxicity. And (2) a cancer that's clearly driven by a heterogeneity of molecular pathways that definitely impact its ability to be successfully treated with any one drug or any one targeted therapy for that matter.

Although we've I think unmasked a lot of novel information regarding the pathogenesis of AML and the genetic drivers of the disease the ability to intervene in these areas is still limited and the overall treatment arsenal is still relatively limited although novel therapies are beginning to push to the forefront.

AML has relatively speaking a low rate of remission, meaning that a minority of patients will achieve a complete remission and this bodes poorly for the future. The minority of patients are cured from AML and the likelihood of cure or long term remission steadily decreases with age. This is related to the fact that the disease relapses frequently and when the disease relapses the outcomes are particularly dismal in all age groups but in particular with the elderly. In the relapsed or refractory setting the response rate's maybe

20% or less and the survival is usually measured on the order of a few months rather than years.

The overall landscape of AML as I mentioned is evolving therapeutically. There have been some breakthroughs in chemotherapy approaches and in targeted therapy approaches but these breakthroughs are still relatively small in scale and many times affecting a small component of the overall diseased population.

There's a lot of work to be done in optimising therapy for AML and a lot of room for novel agents that could provide benefit for this disease.

PTX-200 in AML

With that brief overview I would like to just speak a little bit about the background of our experience with the PTX-200 compound and the inhibition of phospho-Akt in general. Said gave a very nice overview of the importance and relevance of phospho-Akt. Based largely on his work through the years we had clinically developed an interest in approaching this target and did so in the context of a Phase 1 trial that was conducted a few years ago and published and I'll briefly review some of those results.

In that particular trial we were focusing on the single agent therapy utilising PTX-200 also known as triciribine phosphate. It's a single agent in a Phase 1 study of patients with refractory or relapsed acute leukaemias. In this particularly trial we treated 41 patients over the course of the trial in a dose escalation format where we began at the lowest dose of 15 milligrams per metre squared and dose escalated subsequent to that.

In this particular trial we observed that there were dose limiting toxicities occurring at the highest planned dose level of 65 milligrams per metre squared that led to the maximum tolerated dose being 55 milligrams per metre squared with the drug being administered weekly times three.

The primary toxicities of the trial that we felt were possibly drug related included toxicities such as a triglyceride elevation, lipase elevation and mucositis, all of which were reversible with discontinuation of the drug. We also observed common toxicities such as febrile neutropenia, infection, nausea and other types of toxicities you often encounter in AML, the majority being lower grade and reversible and as I mentioned the maximum tolerated dose in the Phase 1 trial was 55 milligrams per metre squared.

The interesting aspects of the Phase 1 trial were that amongst the evaluable patients - 32 patients were evaluable - about half of them experienced stable disease following one cycle

of therapy and we did observe three patients that achieved major decrease in bone marrow blast percentage of 50% or higher compared with baseline.

Although we did not observe any modified responses as per the IWG working criteria we did, as I mentioned, observe patients with reduced blast percentage in the marrow including one patient that had complete blast clearance and another patient with a form of leukaemia known as chronic myelomonocytic leukaemia, a particularly difficult form of leukaemia, who experienced reduction or actually resolution of massive splenomegaly and resolution of his high white blood count during the course of treatment.

As with any Phase 1 study you're also interested in the biological effect of the drug, the pharmacokinetic, pharmacodynamics effects and what we observed in summary was that in the patients that had the highest expression of phospho-Akt at baseline had the greatest reduction in phospho-Akt following administration of the drug as measured by peripheral blood blast studies.

In addition to that the drug, the PTX-200 known then as TCN-P, was taken up by the blast cells in a dose dependent fashion so the drug was definitely getting into the cells and hitting its target once there so that's very important confirmatory information for any early phase clinical trial.

We were pleased that the drug was tolerated well in general and the appropriate targets were being inhibited in the Phase 1 study with some preliminary evidence of activity and that really paved the way for further development in this arena, in particular with combination. I'd like to point out in addition to what Said had mentioned that our primary interest following the study was to really understand how we might be able to capitalise upon the importance of phospho-Akt and the high incidence of phospho-Akt in AML by employing a strategy that maybe allowed us to combine an Akt inhibitor with more conventional therapy to really get at the root of whether we're making an impact on the chemotherapy resistance that is conferred by the presence of phospho-Akt.

We recognised that phospho-Akt is frequently expressed in AML. Said mentioned it is an important mediator of chemotherapy resistance. Even at the bone marrow micro environment level it seems to be very important and we think about AML resistance occurring within the bone marrow micro environment as a result of leukemic cells adhering to their local stroma or endothelial cells or fibronectin and other related proteins that then protect the cell from chemotherapy related death. The phospho-Akt pathway, as

mentioned by Said, is instrumental in actually facilitating this resistance that occurs within the bone marrow micro environment.

For several reasons this was a very attractive target for us to look at and the logical next step was to combine it with chemotherapy in light of the hypothesis that this was a targeted protein that was mediating resistance to chemotherapy. Certainly chemotherapy resistance is the major reason for death in leukaemia. People die from their disease much more than they die from toxicity. If we can overcome that and achieve a higher rate of response then certainly we'll have ultimately an impact on the disease and the survival of patients with this disease.

Current AML trial

That had set the stage for the current trial that we're running and just as an aside, this was a trial that actually Said and I had been very interested in and had in planning stages for quite a long time. We're very grateful that Prescient was able to allow us to do this trial and to provide the support to finally move it forward after quite a long period of time waiting for this and being frustrated by an inability to do a trial that we thought was very promising that has now finally moved forward.

This is a Phase 1b/2 trial and for those people who may not be familiar with clinical trial design, a Phase 1 trial is really designed to assess safety as the primary end point and pharmacokinetics and biologic activity of the drug is measured by some pharmacodynamics assessment to see if your drug is hitting its target and whether it's doing what you want it to do. A Phase 2 study is a trial that is inclined to really determine the efficacy of a drug once you've established its safety and tolerability.

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 and the primary goal of this phase of the study is to establish safety and maximum tolerated dose. The Phase 2 component of the study which the Phase 1 component will feed into as part of the same study will then 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.

That is the current trial design. The Phase 1b design allows for several dosing cohorts for the PTX-200 drug and we have begun dosing at the lowest dosing level of 25 milligrams per metre squared and we have allowance of dose escalation of up to 55 milligrams per

metre squared so 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 the PTX drug is giving on days one, eight and 15.

Currently the trial is in the midst of the Phase 1 dose escalation component with fairly robust accrual at one institution with another one just about to join. We are currently in the middle of the second dosing cohort and have been able to, like I mentioned, accrue several patients over a short period of time, largely because of the fact that treatment options are limited for this group of patients and a clinical trial is always the preferred method of therapy whenever available.

We're in the midst of the dose escalation component and we're looking forward to having two other sites join us in the very near future which will certainly expedite accrual to what is already a robust accrual rate.

Obviously I can't really discuss specific trial results so far because of the fact that - well for obvious confidentiality reasons and lack of publicly disclosed date but the trial is ongoing. There have not been any major safety signals that have been observed to date and the expectation is that the trial will continue to accrue in the dose escalation phase over the next several months with hopeful completion of the Phase 1 component within the calendar year.

PTX-200's differentiation as an AML therapy

That's a very brief update as to the trial status. I've also been asked to finish up by speaking about how this compound, PTX-200, may be differentiated as an AML therapy and how it may fit into the overall treatment arsenal in AML. I think it's a very important type of strategy to employ. Certainly it's not without its competitors in the field and we recognise that within the next year or two there will be most likely two or more agents proved for AML. Most of these agents are targeting a relatively selected group of patients with a recurring type of mutation.

The benefit of a drug like PTX-200 I think is that it allows targeting of a very highly and commonly expressed protein in AML that gives it an opportunity to be utilised in a larger fraction of patients with this disease overall. In addition I feel strongly that the ceiling for chemotherapy in AML has not really been reached yet by any stretch of the imagination.

I gained an appreciation for that during my experience as the Principal Investigator of the CPX-351 compound which of course is nothing more than chemotherapy drugs delivered in a unique way. I believe that the ability to overcome chemotherapy resistance creates

tremendous opportunity to really expand the horizons of traditional chemotherapy in AML that could provide the future platform for additional combinations that may include targeted agents.

I think there's a lot to be excited about with this type of a compound that has some of its own single agent activity and really applies to I think a large subset of AML. As Said mentioned the phospho-Akt signal access I think is really affected by several mutational events that signal downstream through this pathway. You can think of it perhaps as a common or a generalised pathway that is common to many different molecular events that occur in AML and could be the common denominator for achieving a response if the drug is administered in the right way and the right patients are selected.

I think there's a lot of promise in this approach. I really think that the target is very important and the ability to look at this as a chemotherapy modulator, this phospho-Akt protein, really opens the door for creating a higher ceiling for the overall efficacy of chemo in this disease.

I'm quite excited about the potential for this compound and where it could take us in the future if successful. So I'll guess I'll end with that and turn it back over to Steve.

Q&A Session

Steven Yatomi-Clarke: Many thanks Jeff and thanks to Said before you. So I guess that concludes the formal part of our call and I'll hand it back to the operator now to start the Q&A session.

Operator: Thank you. If you wish to ask a question please press star 1 on your telephone and wait for your name to be announced. If you wish to cancel your request, please press star 2. If you are on a speaker phone please pick up the handset before asking your question.

Your first question comes from Dennis Hulme with Edison Group. Please go ahead.

Dennis Hulme: (Edison Group, Analyst): Good evening. My question is for Jeffrey. The first one is about the Phase 1 trial that's being completed and the responses that you saw there. Can you talk to us a little bit about how meaningful it is to see a 50% reduction in bone marrow blasts, what response you would need to see to actually describe that as a clinical response - a complete response or partial response? And for how long the patients need to be stable to consider it to be a stable disease in response to the therapy?

Jeffrey Lancet: Yeah, are you referring to the requirements of the current trial that we're running or the previous trial?

Dennis Hulme: (Edison Group, Analyst) Well if they're different, if you could clarify what the differences might be in identifying a patient response in the two trials.

Jeffrey Lancet: Oh, well yeah so in the original Phase 1, in the single agent trial like I mentioned, we didn't observe any formal responses by the international working group criteria and we didn't expect to, to be quite honest, because of the fact that AML is such a heterogeneous disease driven by multiple molecular anomalies. But the fact that we were able to observe a number of patients that had blast reduction after treatment, and stable disease, indicated that there was some disease modifying activity as well as a patient that had normalisation of his white blood count and reduction of his spleen size.

There's really not much more to say in the original Phase 1 study other than we saw some preliminary signals of our clinical activity in a very very refractory group of patients where a single agent would not be expected to have a significant role.

In the current trial, the expectations are higher because we're combining the PTX drug with a more aggressive chemotherapeutic agent namely Cytarabine given at standard doses - so the expectation is that we will observe complete remissions as defined by bone marrow blast clearance and recovery of blood counts to normal.

The Phase 2 component of the study which is focusing on the patients who have AML in first relapse, clearly defines an end point as to what we need to achieve for this to be considered a successful therapy. So the Phase 2 component of the current trial is designed specifically to achieve a certain end point that would be a high enough complete remission rate in patients with first relapse AML to justify what would be a future randomised study, I think is the best way to put it. I don't know if that answers your question but.

Dennis Hulme: **(Edison Group, Analyst)** Yeah, well if you could just talk us through what you would expect to see in response to Cytarabine on its own in the [unclear] population and what you'd need to achieve to consider that to Phase 2 trial [unclear].

Jeffrey Lancet: So we're looking - the Phase 2 part of the trial, we are enrolling patients with first relapse of a duration of one year or less and I guess the null hypothesis or the historical expectation of response is about 20%. So in our trial we are proposing that our combination will induce a response rate of 40% which would then allow us to have convincing evidence that this is truly having a disease modifying effect that will impact on patients' survival because historically response rates to chemotherapy have translated into

survival differences. So we're looking for an improvement over the baseline 20% response rate to a target of 40% with this combination.

Dennis Hulme: (Edison Group, Analyst) So if the target is 40%, and that will be really convincing, would something a bit less than 40% still be good enough to take it forward into a randomised [2b]?

Jeffrey Lancet: It's a good question. I think that it's difficult to design a trial statistically when the numbers - a pilot type of trial with small numbers to gain enough confidence that that would be the case. So if we were to see a 30% response rate in a small trial, statistically I don't think we'd have enough confidence to say that that's going to be a high enough response rate above 20% to take us to the next step. But if we see improvement that is maybe short of 40%, I would expect that there would be a lot of interest in expanding the trial to gain statistical confidence that we are actually seeing a significantly higher response rate than 20% to justify moving forward. But when the trial numbers are small, like they are right now, in terms of the plan you have to be a little bit more ambitious to gain that confidence level and you have to have a greater difference in the response expectation between the null hypothesis and the alternative hypothesis.

Certainly if we see that number looking like it's promising but not maybe quite at 40%, I would be very interested in expanding the current trial to hopefully give us that statistical significance that would then allow us to say yes, it's better than Cytarabine alone and the likelihood of a successful randomised phase 2 trial is good.

Dennis Hulme: (Edison Group, Analyst) Thanks. I think you've explained that really well. Just to clarify a final thing - so that 40%, is that 40% complete remissions or is it 40% of some other [unclear]?

Jeffrey Lancet: Yes, 40% complete remission which includes patients with complete remission and incomplete count recovery. We're talking about the overall response rate which is comprised of the sum of the complete remissions, the CRs plus, the CRi - which is again for all practical purposes a CR without full count recovery - and we do see that [not infrequently] in this particular setting but they're definitely considered responses.

Dennis Hulme: (Edison Group, Analyst) Great. Thanks very much for taking my question.

Jeffrey Lancet: Thank you.

Operator: Thank you. Your next question comes from Joe Pantginis with Rodman & Renshaw. Please go ahead.

Joe Pantginis: **(Rodman & Renshaw, Analyst)** Hi guys, good afternoon or good evening or good morning, whatever it is with these time zones. Thanks for holding the call and thanks for taking the question.

I want to go a little more macro here with regard to 200. First with regard to - right now you're going after relapse refractory population because it has potential, a quicker path to market. But as you look down the line can you discuss its potential ability to move a little earlier in the treatment continuum, be combined with the likely approval of VYXEOS which is a safer version of 7+3 with better efficacy and a survival benefit. Beyond that, can you discuss what might be some other attractive combinations for the drug as we look to get away from the more deleterious chemos? Thanks a lot. I know that was a mouthful.

Jeffrey Lancet: No, thank you, that's a great question. I think to answer your first question a resounding yes in the sense that we would like to - we always think about how we can move effective therapies more towards the frontline where the chances of success are higher. I think in this case, we look at the relapse setting as the best opportunity to study a new drug where clearly there's an unmet need and there is kind of a built in chemotherapy resistance phenotype across the board that is maybe a little bit easier to study. But ultimately if successful, I would fully expect that we want to move the strategy into the upfront setting, in particular with respect to patients who have high risk features for a treatment failure at the outset.

So I think that we would have - and I forward towards the upfront setting - once we establish the activity of the drug - and in fact that's kind of what we're doing in some ways with the current trial because we're taking it from an empiric, a very highly refractory group of patients that were studied in the initial Phase 1 study. In the current Phase 1b study we're again allowing refractory patients. In the Phase 2 we're taking it into only first relapse so patients that have at least a history of chemo sensitivity. In this population I think we'd expect better results with just about any treatment we use and I think that it offers the patients more if you can offer them a therapy that's effective earlier on because once you relapse it's usually too late.

Combinations

In terms of combinations, I definitely think it's got major potential in the sense that if you look at the drug like VYXEOS will be the new chemotherapy - likely chemotherapy platform

foundation for future combinations, so I see no reason that you would not combine a successful PTX-200 drug with a successful primary chemotherapy drug. Obviously you'd have to do the due diligence in early phase studies to make sure you've assessed it's toxicity in combination, but to me that's a no brainer as far as combination. You're just building upon a successful chemotherapy regimen that is still not perfect but better than what we have.

I think that combining with other targeted agents is also attractive for several reasons. We have knowledge that again AML is driven by multiple pathways and some of them are overlapping, some of them are not. If you think about the [FLT3] pathway for example that is a fairly frequently mutated in AML but its effect is - one of its effects is upregulation of the [PI3 kinase] pathway, that if you can create more potent inhibition of that pathway with this drug PTX in combination with a more generalised FLT3 inhibitor, that you could gain a better overall effect. On the other end of the spectrum, if you have pathways that don't intersect necessarily with the PI3 kinase pathway, that you could potentially duly inhibit two separate pathways that are driving the cancer and that would be an attractive approach as well.

So I think it's got a lot of potential in combination both with a chemotherapy backbone and with targeted therapy backbone. Again, one of the nice things about this particular target is that it's more ubiquitous than several of the other targets that are currently being developed therapeutically.

Joe Pantginis: (Rodman & Renshaw, Analyst) Great, thanks for that Dr Lancet.

Jeffrey Lancet: Thank you.

Operator: Thank you. Once again if you wish to ask a question, please press star 1 on your telephone and wait for your name to be announced. Your next question comes from Mark Pachacz with Bioshares. Please go ahead.

Mark Pachacz: (Bioshares, Analyst) Hi Jeff. I just what to know in that Phase 1 trial, did you see a dose response as you increased the dose? Also as the dose increased, was there a response in reduction in Akt levels and did that reflect for treatment outcomes as well?

Jeffrey Lancet: Yeah, so the answer to that question unfortunately is no. We did not see any type of dose response and I think the small numbers of patients that responded were

limiting in our ability to really understand any type of dose response relationship. We did see that the patients that had the highest baseline [phospho-Akt] expression had significant downregulation of phospho-Akt following treatment. As it turned out, and I think Said is familiar with this data as well, those patients that had the highest baseline phospho-Akt expression were also treated with the higher doses of the PTX - or triciribine at the time - drug.

So it's hard to draw any relationship unfortunately between the dosing and the clinical response or biologic response. And the biological responses were somewhat limited by the number of viable samples that we had and the small numbers of patients in the trial overall so we didn't observe a clear and consistent trend in that particular aspect of the trial. But like I said, the highest Akt expressers clearly had a downregulation of phospho-Akt as a response to the drug and it happens that the doses were higher in that group anyway. I don't know if Said, if you have anything to add to that?

Said Sebti: Yeah, I would just add that the trial that we have at the moment, in combination with Cytarabine, is designed to answer that question. And we will and we are actually, with those that we accrue so far collecting the samples pre-PTX-200 and post-PTX-200 so we'll be able to answer whether there is a dose response. We have several questions. The two most important [1] is the baseline - like Jeff was talking about - does the baseline predict - the baseline of phospho-Akt predict a response to PTX-200, and [2] most importantly is the decline of the phospho-Akt levels by PTX-200 correlates with the clinical activity that we will see. So I agree with Jeff, the numbers were too small and all that but we will answer this question with the present trial as designed.

[Over speaking]

Jeffrey Lancet: Sorry, the only other thing I would add would be that the numbers are small but in the responding patients as measured by blast reduction, the two best responders in terms of blast reduction and spleen reduction occurred at the higher dosing cohorts as opposed to the lower dosing cohorts. At the 45 and the 55 cohorts are where we saw the best responses in a very - a limited number of responders so it's hard to say much about that but like Said mentioned, this question will be heavily addressed in the current trial.

Mark Pachacz: (Bioshares, Analyst) Okay, just another question for Said. Said, can you just tell me a bit more about the history of this Akt target, how crowded the space is

and just expand a little bit about the competing [dynamics] about how they have more off target effects and why yours doesn't have those off target effects.

Said Sebti: Sure, yeah. So well basically the typical kinase inhibitor, and Akt is a kinase, it's based on a substrate mimicry. So you make a drug that mimics ATP which is a substrate for Akt and about another 500 kinases in our human proteome or the ensemble of all the proteins in our body. There are about 500 of them that are kinases, and they all utilise this ATP that we're trying to mimic, that the competitors have mimicked.

Therefore it is easy to see how no matter how selective is your ATP mimic, you are going to have off target effects because the pockets on these kinases, the 500 kinases are similar. Our inhibitor does not at all bind to the ATP binding sites of Akt, instead it attaches itself - PTX-200 attaches itself to another area of Akt which is very very critical to its cancer causing activity. So where PTX-200 binds is in an area away from the ATP binding sites - is an area called the PH domain and that PH domain happens to be very critical to the ability of Akt to become phosphorylated and that phosphorylation - that hyperphosphorylation is what's driving the disease and that's what's driving the resistance to chemotherapy. So therefore what PTX-200 does really is prevent the phosphorylation of Akt by attaching itself to the PH domain rather than the ATP sites where others are.

We know that this is more selective because we have done this and published it where we take tumours that have high phospho-Akt and tumours that have low phospho-Akt and we put them in animal models, like a mouse model. In the same mouse the tumour that has high phospho-Akt melts away. The tumour that has low phospho-Akt or no phospho-Akt keeps growing. So that's evidence that - the biochemistry is the ATP mimicry that is not there and the PH domain binding that is there, but the proof of concept can be even extended phenotypically by showing that actually only hyperphosphorylated tumours are responding.

Mark Pachacz: (Bioshares, Analyst) Great. Alright, thanks very much Said.

Said Sebti: You're welcome.

Operator: Thank you. Your next question comes from Paul Hart with InSync Equity Services. Please go ahead.

Paul Hart: (InSync Equity Services, Analyst) G'day. My question is for Jeff in relation to other chemotherapy drugs that are coming through the system. If we do get other

drugs which are more effective than the current standard, would PTX-200 have to go through full trials in order to get approval to be able to be used with those drugs, those new drugs coming through?

Jeffrey Lancet: I think that with any type of new combination you would have to study PTX in combination to assess its toxicity profile and safety profile so that you can really establish the role of PTX in that setting. Is that what you're asking, is whether you need to do trials to ascertain the [unclear]?

Paul Hart: (InSync Equity Services, Analyst) Whether you'd have to go through the full three phases of trials or would you start at Phase 2 - that's my question.

Jeffrey Lancet: Yeah, I think many times when you've already established a track record of safety with one compound or other - one or more compounds in combination, you would typically do future combination trials with perhaps a small lead-in safety phase that may have one or two dose levels and then jump right into the meat of the trial and the randomisation phase.

I think that you'd be looking at relatively brief - not brief- but less intensive trials once the full safety profile has been established in combination with what we're doing right now. The combination with other agents would be faster and could go right from a very short lead in combination period right into a randomised study. A lot of trials designs are like that right now, they're are all inclusive in really trying to get it all done at one time. I think if you have the safety profile established through a single agent Phase 1 study, and in combination with chemotherapy, you've already done a lot of the work and you don't have to go through the extensive dose escalation approaches you would before.

Paul Hart: (InSync Equity Services, Analyst) Thanks Jeff, and Said are you aware of many other new chemotherapy drugs, new classes of chemotherapy drugs coming through the trial phases?

Said Sebti: Is your question am I aware of other targeted therapies that are ongoing in terms of clinical trials?

Paul Hart: (NSec Equity Shares, Analyst) I'm thinking - I mean PTX is being trialled with the Cytarabine, but my question relates to well what are the chances of a new class of drug coming through that becomes the new standard of care that displaces the current Cytarabine. I know drug trials obviously take a lot of time but it could be a risk if PTX is showed to work - yeah, I just see it as a potential risk.

So are you aware of any other - many other new classes of chemotherapy drugs coming through?

[Over speaking]

Steven Yatomi-Clarke: I can step in that quickly and I think Jeff addressed that previously. The drug that he was involved with recently that he spoke about CPX-351 is an enhanced form of chemotherapy, a reformulated one, and that's what he spoke about with respect to potential combinations down the track. But over to you Said if you have anything else to add.

Said Sebti: Yeah. No, what I was going to say - thanks Steve - is that really Cytarabine - and Jeff of course can correct me if I'm wrong - but Cytarabine has been the backbone of chemotherapy for a long time and even the new [Celator] as Jeff said, is just way of delivering the same chemo. So Cytarabine and Doxorubicin, these have been - Cytarabine based therapies have been - and for myself I don't see any other chemotherapies that would replace that. It's been like that for a while and I don't see any coming that are other than this backbone of Cytarabine.

So Jeff do you agree? Do you have other? Please.

Jeffrey Lancet: No, I agree. I think that we're not likely to see Cytarabine replaced as the common backbone drug for induction or relapsed AML. There may be variations of Cytarabine through different dosing schedules or different formulations like CPX, but I'm not aware of any drugs that are on the horizon that are really looking as something to replace Cytarabine. [LS Cytarabine] drug failed a few years ago. Other drugs such as vosaroxin are really being tested with the Cytarabine not as a replacement for Cytarabine. Clofarabine doesn't really have a solid path moving forward based on fairly negative results over the years.

So I think that there's not a threat of an intensive chemotherapy regimen that would be replacing Cytarabine outside of the CPX drug which is Cytarabine plus daunorubicin. I think in terms of chemotherapy backbones, the hypomethylating agents like azacitidine and decitabine are the chemotherapy backbones in the elderly group of patients who are considered not fit for regular traditional chemotherapy. That's really a different population than what we're talking about here but nonetheless one that we I think would want to explore in the future because chemotherapy resistance in the elderly is definitely a problem. I could envision wanting to combine PTX with one of those less intensive

hypomethylating agent backbones for a more frail less fit group of patients, in the elderly group for sure.

Paul Hart: (InSync Equity Services, Analyst) Thanks very much guys.

Jeffrey Lancet: Sure.

Operator: Thank you. We are showing no further questions at this time. I'll now hand back to Mr Yatomi-Clarke for closing remarks.

Steven Yatomi-Clarke: Thanks Bethany and many thanks to everyone for attending. It looks like that concludes the call. Thank you for Jeff and Said for taking the time to speak with us today. I hope it gave you all a better understanding of our AML program and the calibre of people involved and why we're so enthusiastic about this trial.

I understand that a copy of this trial and recording and I think also a transcript will be made available via our website and if you would like further information, you can check our ASX announcements (our ASX code is PTX) or indeed via our newly developed website. The web address is www.ptxtherapeutics.com or you can contact me directly steven@ptxtherapeutics.com. My contact details are also at the bottom of all our ASX announcements.

So many thank you again to everyone on this call, and to Jeff and Said, and I wish you all a wonderful day. Thank you.

End of Transcript