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CTMX - CytomX Therapeutics, Inc. - Special Call

EVENT DATE/TIME: JUNE 04, 2018 / 10:00PM GMT

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PRESENTATION

Operator

Good afternoon. My name is Valerie, and I'll be your conference operator. At this time, I would like to welcome everyone to the CytomX Therapeutics ASCO 2018 Presentation Review. (Operator Instructions) As a reminder, this call may be recorded.

I will now turn the conference over to your host, Mr. Christopher Keenan, Vice President, Investor Relations and Corporate Communication.

Christopher S. Keenan - *CytomX Therapeutics, Inc. - VP of IR & Corporate Communications*

Good afternoon, and thank you for joining us to discuss the company's clinical data presentations at the 2018 Annual Society of Clinical Oncology (sic) [2018 Annual Meeting of the American Society of Clinical Oncology.] I'm joined here today by CytomX President and Chief Executive Officer, Dr. Sean McCarthy; Debanjan Ray, our Chief Financial Officer; Dr. Mike Cavanagh, our Chief Scientific Officer; and Dr. Rachel Humphrey, our Chief Medical Officer.

Before we begin, I would like to remind you that we'll be making forward-looking statements during the call, including guidance on research and development activities, including preclinical and clinical data pertaining to CX-072. Because forward-looking statements relate to the future, they are subject to inherent uncertainties and risks that are difficult to predict and many of which are outside of our control. Important risks and uncertainties are set forth in our most recent public filings with the SEC at sec.gov. We undertake no obligation to update any forward-looking statements whether as a result of new information, future developments or otherwise. This data is interim, and future results in this trial or future trials may not be consistent with the data discussed today.

We will be using slides on today's call. For those of you who have joined by phone, I invite you to view the slides as well as a replay of this call on the Investors page of our website at cytomx.com.

I will now turn the call over to Sean.

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

Thank you, Chris, and good afternoon, everyone. Thanks for joining us as we review the preliminary proof-of-concept clinical results for CX-072, our lead wholly owned Probody targeting PD-L1.

These first clinical data marked a major milestone for both CytomX and our Probody technology. We're advancing what we believe is a fundamentally new approach to antibody therapeutic drug development; and in CX-072, a potential new centerpiece of combination cancer therapy.

Turning now to our slides, I'd like to start with a brief review of our Probody technology. As shown in Slide 4, Probodies are fully recombinant antibody therapeutic drugs with masked antibody binding sites. The mask blocks the ability of the antibody to bind targets until the mask is removed selectively in the tumor microenvironment by tumor-associated protein. Mask removal in the tumor allows the antibody to bind to target on cancer cells while maintained masking in the periphery substantially diminishes binding to target in normal healthy tissues. Our vision at CytomX is to use this technology to bring safer and more effective therapies to patients with cancer.

In recent years, we've generated substantial amounts of preclinical evidence in support of the Probody concept. The data we're presenting today at ASCO is our first look at how our CX-072 preclinical data is translating into the clinic. And I'm pleased to say upfront that the data look very encouraging.

Before taking a deep dive into CX-072, let's take a look at Slide 5, which shows the broad pipeline of Probody therapeutics and active development at CytomX and with our partners. By the end of this year, we expect to have 5 Probody programs in the clinic, including cancer immunotherapies against the clinically validated targets, PD-L1, CTLA-4 and PD-1; and first-in-class Probody drug conjugates against highly attractive noble targets, CD166 and CD71, which have been considered inaccessible to conventional antibody drug conjugates due to their presence on healthy tissue. The pipeline is advancing very well, and we anticipate multiple clinical updates during the second half of 2018 and throughout 2019.

Focusing now on CX-072, let's turn to Slide 6. We believe this molecule has the potential to become a differentiated centerpiece of cancer therapy in multiple indications by enabling treatment with combinations that are currently limited by toxicities. Our ultimate vision is to advance CX-072-based combinations with the potential to elicit deeper and more durable responses in cancer patients.

On this call, we'll review data from 2 posters presented this morning at ASCO. These posters are available under the Investors sections of our website. The posters, as listed on Slide 7, describe initial data from 2 of the 5 arms of PROCLAIM-CX-072, our ambitious ongoing Phase I/II trial evaluating CX-072 both as monotherapy and in certain combinations.

At ASCO, we presented a comprehensive summary of safety and efficacy data for the monotherapy dose-escalation arm Part A and the initial snapshot of Part B, in which we are evaluating the safety and efficacy of CX-072 in combination with Yervoy, ipilimumab.

Before Rachel discusses the clinical data in some detail, Mike will briefly review the CX-072 preclinical package. And I'll conclude with some closing comments before opening up the call to questions.

Before turning it over to Mike, though, I'd like to review the overarching goals seen on Slide 8 of the PROCLAIM-CX-072 trial. We entered the clinic last year looking to address 3 important initial platform and product-related questions all aimed at translating our robust preclinical data for the anti-PD-L1 Probody into clinical proof of concept. First, does the Probody therapeutic remain predominantly masked in the systemic circulation? Second, what is the safety profile, and is it consistent with the potentially differentiated PD inhibitor? And third, is the Probody therapeutic active in the tumor microenvironment? Today, I'm very pleased that with these preliminary data, we can say yes to all of these questions, representing a substantial step-forward for CytomX.

Now I'm delighted to turn the call over to Mike, and I'll be back a little later on.

W. Michael Kavanaugh - *CytomX Therapeutics, Inc. - Chief Scientific Officer and Head of Research & Non-Clinical Development*

Thanks, Sean. The foundation of the CX-072 clinical program is a series of preclinical studies summarized on Slide 10, that demonstrate in mouse models that a PD-L1-directed Probody is equally efficacious as PD-L1 antibodies but with greater safety.

First, in the upper left panel, you can see that in standard mouse tumor model widely used for studying PD-1 inhibitors, a PD-L1 Probody has equal antitumor activity as an antibody when given at the same dose. In the bottom left panel, imaging of labeled CX-072 demonstrates that CX-072 concentrates in the tumor to the same extent as the antibody. However, as shown in the upper right panel in a mouse model, while the PD-L1 antibody induces autoimmune diabetes in most of the animals treated, the PD-L1 Probody doesn't induce autoimmunity in any of the animals even when given at the same dose and despite the fact that it is equally efficacious. Finally, the bottom right panel demonstrates that PD-L1 antibody binds to PD-L1-expressing T cells in the circulation, but the PD-L1 Probody does show only as much higher doses and to a lesser extent. These data highlight what Probody therapeutics are designed to do, that is, as shown in Slide 11, by remaining masked while in the blood, Probodies avoid bonding to target outside of the tumor and, thereby, minimize induction of toxicities such as autoimmunity. However, because they are unmasked by proteases enriched in the tumor microenvironment, they're designed to have at least the same efficacy as the antibodies they were derived from.

Rachel and I will return to these points when discussing the efficacy, safety and clinical pharmacokinetics of CX-072 in the trial. And with that, I'll turn the call over to Rachel for a review of the clinical data.

Rachel Wallach Humphrey - *CytomX Therapeutics, Inc. - Chief Medical Officer*

Thanks, Mike. The data we announced today is a first for PROCLAIM, marking the first clinical results for a Probody. Although immuno-oncology had emerged as a pillar of cancer treatment, at CytomX, we believe that the full potential of combination immunotherapy is limited by immune-related toxicities and that CX-072 has the potential to address this limitation.

Let's review the design of this study. Slide 14 shows eligible patients who are at least second line with solid tumors. They were immunotherapy naïve, meaning that they had no PD-1 or PD-L1 inhibitor in the past. Furthermore, eligible patients could not have PD-1 or PD-L1 inhibitor antibodies available for their disease at the time of enrollment. Patients were dosed with CX-072 from 0.3 to 30 milligrams per kilogram every 2 weeks intravenously. As of the April 20, 2018, data cutoff, the dose escalation was completed with 22 patients enrolled. Follow-up continues in this (inaudible) study.

Slide 15 shows the summary of the baseline characteristics and tumor types of the 22 patients enrolled in the trial. There are a few important things to note here. First, these are heavily pretreated patients with tumors that are thought to be poorly responsive to PD pathway inhibitors, and they were not selected for PD-L1 expression. Also, the majority of patients, in fact, were PD-L1 low or negative.

Now let's review the safety data. Slide 16 shows that at the data cutoff, all 22 patients were evaluable for safety. The MTD in this study was not reached. Most treatment-related adverse events were Grade 1 or 2, with Grade 3/4 treatment-related adverse events occurring in 2 of 22 patients, which is 9.1%. These included neutropenia and thrombocytopenia in a patient with thymic cancer who received 3 milligrams per kilogram of CX-072. This event was the only dose-limiting toxicity observed in this monotherapy dose-escalating part of the study. The second was a Grade 3/4 -- the second Grade 3/4 adverse event was transaminase elevation in a patient with breast cancer who received 30 milligrams per kilogram CX-072. Both events reversed rapidly with steroids and drug discontinuation. The table at the bottom of the slide is a detailed safety summary.

Moving on to Slide 17. We were pleased to see antitumor activity in this heavily pretreated group. 20 patients were evaluable for efficacy, including 1 patient with nonmeasurable disease. We saw 3 partial responses. The first response is a thymic cancer patient who received 3 milligrams per kilogram of CX-072. This is the same patient who experienced Grade 3/4 neutropenia and thrombocytopenia. Her tumor progressed, therefore, she was taken off CX-072 for toxicity. We also saw 2 responses in patients who received 10 milligrams per kilogram of CX-072: one was a triple-negative breast cancer patient; and the other was a cervical cancer patient. Both of these patients remained on study after about 8 months of treatment. The breast cancer partial remission has been confirmed. The confirmation scan for the cervical cancer patient is pending. We also saw 8, who were about 42% of patients, achieved stable disease.

On Slide 18, the waterfall plot provides an overview of tumor types and responses. As noted above, 42% or 8 of the 19 patients with measurable target lesions at baseline saw a decrease in target lesion per RECIST version 1.1. And among the patients who received at least 3 milligrams per kilogram of CX-072, 60%, or 6 of 10, saw a decrease in target lesions from baseline.

On Slide 19, the spider plot demonstrates the durability of the anticancer effect and the slow steady decline of tumor burden in the breast cancer and cervical cancer patient, a pace of response that is classic for checkpoint inhibitors. The study is still ongoing, and further follow-up will be presented at future meetings.

Now I'll present to you a case study of the triple-negative breast cancer patient with the confirmed partial response. Slide 20 shows a heavily pretreated patient with metastatic disease, microsatellite stability, low tumor mutational burden and negative PD-L1 expression. Her disease included inflamed skin lesions, which, as you can see from the photographs at the bottom of the slide, resolved over the first 4 to 5 months of treatment. The scan on the left at the top of the slide is the baseline. And the circle on the scan marks the tumor mass just below her right axilla. The scan on the right, taken several months after the start of the therapy, shows a meaningful reduction in this lesion and a partial response, which was later confirmed. This patient is now approximately 8 months from the start of her therapy and continues to be treated with CX-072. This and the other responses observed in monotherapy is strong evidence that the Probody is active in the tumor microenvironment.

Let's look at another patient case study here on Slide 21. We stained a tumor biopsy taken from an esophageal cancer patient receiving CX-072 at 30 mgs/kg for the presence of CD8+ T cells. On the left, you see the baseline image before treatment; and on the right, a threefold increase in CD8+ T cell infiltration after 4 weeks of treatment. Increases in CD8+ T cell infiltrate hv been seen after treatment with other PD pathway inhibitors. These data, therefore, provide additional evidence for biologic activity within the tumor.

I'll now turn it over to Mike, who will review an important pharmacokinetic analysis.

W. Michael Kavanaugh - *CytomX Therapeutics, Inc. - Chief Scientific Officer and Head of Research & Non-Clinical Development*

Thank you, Rachel. Slide 22 reviews preliminary single-dose pharmacokinetic findings from CX-072 monotherapy.

The left-hand panel shows the concentration of CX-072 over time at doses ranging from 0.03 to 30 mgs/kg. Note that there are separate curves shown for the amount of masked CX-072 in the blood, shown in solid lines; and the amount of total CX-072 in the blood representing both masked and unmasked drug, shown as dashed lines. These 2 lines are about the same. These data demonstrate that CX-072 circulates predominantly as the intact masked prodrug species as the Probody was designed to do.

For comparison, we also show on the right-hand side of the slide the reported PK of atezolizumab, a marketed anti-PD-L1 antibody. Note that the data highlighted in the green boxes. These data demonstrate that at low doses, atezolizumab exhibited rapid clearance and evidence of target-mediated drug disposition; while at the same doses, CX-072 does not. This profile is what would be expected. The CX-02 -- CX-072 has reduced binding to PD-L1 outside of the tumor compared to atezolizumab and again is consistent with the Probody therapeutic design.

I'll now turn it back again to Rachel.

Rachel Wallach Humphrey - *CytomX Therapeutics, Inc. - Chief Medical Officer*

Thanks, Mike. As shown here on Slide 23 in summary, as monotherapy, we observed that CX-072 is well tolerated, demonstrated antitumor activity and remains masked in circulation. Now let's turn to the combination results with ipilimumab.

Turning to Slide 25. As Sean noted, our vision for the CX-072 program is for CX-072 to become the combination partner of choice. The first combination that we are evaluating in this study is CX-072 plus ipilimumab, with the goal of maintaining antitumor activity with an improved safety profile compared to prior experiences with nivolumab plus ipilimumab in combination. The published data on nivolumab and ipilimumab on this slide is meant to provide context on the issues with this combination that we are seeking to address with CX-072. In patients with advanced

melanoma, the combination of 3 milligrams per kilogram of ipilimumab and reduced dose of 1 milligram per kilogram of nivolumab is highly effective, with nearly 60% of patients responding. However, this combination is also highly toxic, with 55% of the patients developing Grade 3/4 treatment-related adverse event. This is in contrast to ipilimumab alone, where 20% of patients respond, and 27% of patients developed Grade 3/4 treatment-related adverse events. These data show that the cause of the dramatic increase in efficacy for the combination is a dramatic increase in toxicity compared to monotherapy. To address this high rate of toxicity, recent studies have decreased ipilimumab dose and extended the dose schedule. We think there are opportunities with CX-072 to realize the full potential of this combination in multiple indications by enabling more optimal dose and schedule.

Slide 26 shows that the primary objectives of this ongoing arm of study are to assess safety and tolerability and to determine the MTD and DLT of CX-072 when administered in a concomitant combination schedule with ipilimumab. Eligible patients are at least second line at solid tumors, immunotherapy naïve and have no PD-1 or PD-L1 inhibitor available for their disease at the time of enrollment.

As of the data cutoff on April 20, 2018, patients were dosed with escalating CX-072 doses up to 10 mgs/kg in combination with ipilimumab at 3mgs/kg every 3 weeks intravenously for 4 cycles, followed by CX-072 monotherapy every 14 days.

Slide 27 is an overview of the patient demographics of the 16 enrolled patients and their corresponding tumor types. Key things to note include the patients are heavily pretreated, and the cancer types are those that don't typically respond to PD pathway inhibitors.

Let's now review the data on Slide 28. Beginning with safety, 16 patients were evaluable in this ongoing study in which an MTD has not yet been reached. One patient experienced the DLT of Grade 3 dyspnea at the 0.3 mgs/kg 072 and 3 mg/kg ipilimumab dose. Most treatment-related adverse events were Grade 1/2. And Grade 3/4 treatment-related adverse events occurred in 5 of 16 patients or 31% at the time of the data cutoff. The details of these patients are listed on the slide. For those of you who have studied the poster carefully, you will have noted a footnote regarding a Grade 3 treatment-related adverse event in 1 patient that was designated as a nontreatment related shortly after the data cutoff. For completeness, that is a patient in the 1 milligram per kilogram CX-072 cohort. And of course, that reduces the overall rate to 25% or 4 out of 16. It's still early days, and we'll learn more as the study progresses. We also saw the study progresses. The rate of Grade 3/4 treatment-related adverse events observed in this combination is consistent with ipilimumab monotherapy. The number of patients in this series is small, but we are encouraged by this safety data. Dose escalation continues in this arm.

Slide 29 reviews the efficacy results among the 12 evaluable patients, including 2 patients with nonmeasurable disease at baseline. We saw a durable complete response in a patient with anal cell cancer who received 0.3 milligrams per kilogram of CX-072 along with 3 milligrams per kilogram of ipilimumab. The tumor was HPV positive but also PD-L1 negative, microsatellite stable and with intermediate tumor mutational burden. We also saw 2 partial responses: 1 in a patient with testicular cancer who received 1 milligram per kilogram of CX-072 with 3 milligrams per kilogram of ipilimumab; and a second in a patient of cancer of unknown primary that was suspected to be a small bowel cancer who received 3 milligrams per kilogram of 072 with 3 milligrams per kilogram of ipi.

Slide 30 shows the waterfall plot demonstrating the rate of tumor shrinkage with 30% or 3 of 10 patients experiencing a target lesion reduction from baseline from RECIST version 1.1.

Slide 31 is the spider plot for the combination arm. All 3 responders experienced slow and steady decline of their tumor burden. The complete response is confirmed. The confirmation scans for the 2 partial remissions are pending, and all 3 patients remain on treatment. The poster presented this morning details 2 patient case studies from this combination arm and is available to view on our website.

Slide 32 shows our key takeaways. What we have learned from the preliminary data from this ongoing combination arm is that CX-072 is well tolerated in combination with the full-label dose of 3 milligrams per kilogram of ipilimumab. This combination is demonstrating antitumor activity, including 1 complete response and 2 partial responses observed to date in a population of patients that is historically more resistant to PD pathway inhibitors that is melanoma. We believe that these encouraging data reinforced the opportunity for CX-072 to be a potential differentiated centerpiece of immunotherapy.

With that, I'll turn the call over to Sean.

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

Thank you, Rachel. So just to start to wrap up, first of all, let's revisit those 3 key platform and product-related questions that I laid out at the beginning of the call.

They are: Does the Probody therapeutic remain predominantly masked in the systemic circulation? We feel really great that we've been able to comprehensively check that box. The circulating performance of the prodrug across patients in the monotherapy arm, where we have comprehensive PK, looks very, very encouraging. Secondly, the safety profile of the program overall is, we believe, also very encouraging, both this monotherapy and in the combination with ipilimumab, although, as Rachel mentioned, it is still fairly early days with the ipi combo, but nonetheless, very encouraging. And then, thirdly, of course, is there antitumor activity? And we feel really good about that, having shown 3 responses for monotherapy, 3 responses for the combo, including a complete response, and in a very heavily pretreated, tough-to-treat patient population, which we wouldn't have expected to have been this responsive to this agent. So a very good progress towards platform and product POC, and we've been delighted to share this data today.

In terms of next steps for the program, the PROCLAIM-CX-072 study, as I've mentioned before, is an ambitious clinical trial with multiple arms. We've focused today on Parts A1 and B, the monotherapy and the ipi combo. We have an ongoing A2 arm, which is enrolling PD-L1 positive patients and also mandating biopsies. We expect to have data from that arm in the second half of this year, which, among other things, will allow us to further interrogate the biochemical performance of the Probody within tumor tissue.

We also have now opened expansion cohorts in 8 tumor types at the dose of 10 milligrams per kilogram. Those expansion cohorts have been initiated based on the very encouraging data that we've seen thus far with monotherapy. And our goal overall is to explore several tumor types to pick at least one in due course to drive rapidly towards monotherapy registration.

The Part B, we've talked about today the last remaining arm is the vemurafenib combination, which is ongoing. And we expect to present initial data sometime in 2019.

Slide 36 just summarizes our recent progress and the upcoming milestones. And as those of you who have followed the company for a while have often heard me say is CytomX is a company that lays out milestones and does what it says it will do. We have achieved a tremendous amount over the last year and very proud of the team, and that's actually within our pipeline and also with our partnerships.

And we can now point to multiple upcoming milestones, laid out on the right-hand side of this slide, in the second half of this year and throughout 2019, with additional readouts from a number of our clinical studies, not just, of course, the PROCLAIM-CX-072 PD-L1 Probody but also the CX-2009, CD166 PDC, the BMS CTLA-4 Probody is in the clinic. We have no formal guidance on when data will be presented, but they are moving now forward in clinical development. As you would've seen recently, we have the IND declared for the CX-2029, CD71 PDC. In collaboration with that, we thought we'll enter the clinic shortly. That, by the way, resulted in a $25 million milestone payment to CytomX. It's a terrific achievement. And we plan to put the CX-188 PD-1 Probody into the clinic in the second half of this year as well. So a tremendous amount going on, a lot of news to come over the next 6, 12, 18 months and continued strong execution across the platform and, of course, the overall pipeline.

So the progress that we have outlined today really represents, we believe, truly innovative science at work and is a very big step towards our goal of building a long-term commercial-stage oncology company that can make a real difference to patients.

Before we open the call to questions, I'd like to take a moment to give special thanks to our employees, who are working tirelessly towards realizing our company vision; to our clinical investigators and contract organizations; and most importantly, to the patients who have participated in our study to advance the course of cancer research.

I'll now turn the call back to Chris to manage the Q&A.

Christopher S. Keenan - *CytomX Therapeutics, Inc. - VP of IR & Corporate Communications*

Operator, you can go ahead and open up the call for questions.

QUESTIONS AND ANSWERS

Operator

(Operator Instructions) Our first question comes from Christopher Marai of Nomura.

Christopher N. Marai - *Nomura Securities Co. Ltd., Research Division - MD and Senior Analyst*

I was wondering, first, if you could elaborate a little bit on the single-agent data and some of the AEs that we saw at the low dose, I guess, in the thymic cancer patient, the breast cancer patient. I suppose neutropenia and thrombocytopenia are typically seen with PD-L1 antibodies, but could be. And wondering if you have any information on those. As well as the transaminase elevations, is that liver meds, or do you think that at 30 mg/kg, you hit a PD-L1 tox? I have a follow-up.

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

Yes, so I'll hand that one over to Rachel to comment on.

Rachel Wallach Humphrey - *CytomX Therapeutics, Inc. - Chief Medical Officer*

Great. Rachel here. So let's start with the neutropenia and thrombocytopenia patient. This is a woman with a thymic cancer. And as you know, Chris, thymic cancer patients can get neutropenia and thrombocytopenia. Good's syndrome is neutropenia. Thrombocytopenia is also observed. But you're right, neutropenia is also seen, although rarely, with PD inhibitors. It's very hard to tell what the contribution of each is, but what I can say is that neutropenia, in general, was not seen in any meaningful way in the rest of the monotherapy program. On the poster, you can see treatment-emergent adverse events that occurred regardless of attribution, and neutropenia is just not on that list. The patient with 30 mgs/kg who had transaminase elevation was a woman with breast cancer and responding liver mets. The tumor burden hasn't reduced to the formal response level, but it was clearly shrinking. So it's not completely clear what contribution potential inflammation in her liver made to the grade of her events. But hereto, it's worth noting that a transaminase elevation was not seen in any grade in the rest of the monotherapy's setting. And once again, if you go to the treatment-emergent adverse event table in the poster, regardless of attribution, transaminase elevation doesn't show up there either.

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

And Chris, maybe just one other -- for maybe more high-level comment on monotherapy safety, you'll recall that we've said for some time that given the small number of patients in these initial study arms and 22 patients in this first dose escalation, we have never expected to show any significant difference in tox necessarily relative to the benchmark out there for PD agents because the numbers are small. And as Rachel mentioned, a couple of patients, (inaudible) about 2 patients here, nonetheless, an AE rate overall, a Grade 3/4 AE rate of still below 10%, which we think is pretty good. So just some additional context there.

Christopher N. Marai - *Nomura Securities Co. Ltd., Research Division - MD and Senior Analyst*

Okay, now that's helpful. And just with respect to the biopsy data showing the greater CD8+ T cells infiltrating the tumor microenvironment, did you measure any other markers of inflammation there and any protease levels? And then just maybe remind us where you're at on measuring sort of the protease levels in tumor or unmasked Probody in the tumor?

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

So a couple of answers to that question. One is, as I mentioned earlier on, the A2 arm, which is actively enrolling, is really designed to give a lot more information on comprehensive analysis of biopsies, including looking at Probody activation, looking at inflammatory cell infiltration, looking at cytokine profiles as well. We had a very limited amount of biopsy information from Part A because we didn't mandate biopsies in that escalation arm since, of course, we wanted to get through the escalation as quickly as possible in case of opening of several other arms in the study. Specifically, so more to come over the second half of the year, so stay tuned. With regards to that particular biopsy, the team is continuing to look at it in some T cell, including looking at gene expression profiles for inflammatory markers, and we may have more to say about that particular patient also as the year progresses.

Operator

Our next question comes from Mohit Bansal of Citigroup.

Mohit Bansal - *Citigroup Inc, Research Division - VP and Analyst*

My question is on -- another one on safety. So as you are noting that the safety of ipi, CX-072 combo was similar to what you see with ipi alone, however, we are getting some queries that's suggesting that doctors can manage ipi safety issues better as they understand them better. Could you help us understand if there were any proactive safety management techniques that were employed in this trial? And what are the best ipi data that compare these data sets better? And I have a follow-up.

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

So I think if I could repeat the question you're asking is, well, first of all, we would agree with you, we think that -- again, we're encouraged by this early data. So the Grade 3/4 adverse event rate of the combo in -- let's say, in the 25% to 30% rate, that's where we are right now. Rachel discussed that footnote on the poster, which we thought might be a helpful clarification for you guys. So that's what we are seeing. That is about the generally accepted, understood Grade 3/4 AE rate for ipi as a monotherapy. So that's -- that looks pretty good. I think your question is whether, in the study, there were any particular patient management measures taken to minimize the tox, and I'm going to ask Rachel to comment on that. I'm pretty sure the answer is no.

Rachel Wallach Humphrey - *CytomX Therapeutics, Inc. - Chief Medical Officer*

Yes, the answer is no. There was nothing special about it. So that's all I can say. It was handled as any Phase I study, with all the bells and whistles of a Phase I study.

Mohit Bansal - *Citigroup Inc, Research Division - VP and Analyst*

Got it. And then if you can help us -- provide some more color on the 8 expansion cohorts you are going forward with, which cancers are those and how you are selecting those cancers? And then would you be testing any (inaudible) sensitive tumors in those expansion?

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

Yes, great question. I guess all I would say at this point is that -- well, just to -- so just to reiterate that we've been fairly aggressive there based on the very encouraging activity that we've seen in monotherapy so far in these very difficult strict patient populations that we are opening up a significant number of arms. In terms of the achievements, actually, we're not disclosing specific tumor types at this point, but I can tell you that there is some overlap with tumors that we have seen respond in the study; makes sense, of course. And it does include certain tumor types that are already known to be PD sensitive.

Mohit Bansal - *Citigroup Inc, Research Division - VP and Analyst*

Got it. And then maybe if I can squeeze one more in. So as you are choosing 10 milligram per kilogram as the dose to go forward, do you have any data to compare to the -- to current PD-1s that are out there at this dose in terms of activity? Is it higher than a liver dose or a pembro dose, anything you can help us understand this part?

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

I think the high-level answer to that question, Mohit, is that we're right in the range of where one might expect to be with an antibody-like therapeutic against this class of targets. We -- so nivo, pembro, durva, atezo, they're dosed in the 3 to 20 mg/kg range in general. And so we're at the 10 mg/kg dose. I have to underscore that, that is quite an achievement for a platform because the work that Mike and the team did over a number of years to fine-tune the CX-072 Probody preclinically so that we wouldn't have to do anything unusual in the clinic with regards to those levels has worked out extremely well. I mean, this is translated into the clinic, I think, in a way that is just terrific and we're very excited about. So let's just be a little bit more specific on how did we pick this 10 mg/kg dose. Well, a combination of all available data at the time, including detailed clinical pharmacologic modeling, computational modeling, preclinical modeling, but perhaps more importantly than anything, the observation that 2 out of 3 patients dosed in the monotherapy at 10 mg/kg responded really well. That's the cervical cancer patient and the triple-negative breast cancer patient that Rachel mentioned earlier on. So we think it's a good dose. Is it the be-all and end-all dosing, dose selection? Probably not, it might not be, but we think that it's a very relevant dose to move forward within these initial expansion cohorts.

Operator

Our next question comes from Peter Lawson with SunTrust Robinson.

Peter Richard Lawson - *SunTrust Robinson Humphrey, Inc., Research Division - Director*

Maybe for Sean or Rachel, just on the monotherapy safety, which side effect are you most worried about? I know they're kind of small numbers, but was there any sense that's coming from the PD-L1 side or the masked or anything else which we should kind of be concerned about?

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

I don't -- there is nothing in particular we're worried about there, Peter. Again, this is a pretty beaten-up patient population, and the numbers are small. And in the context of a Phase I study, yes, these could be the kinds of things that you can see. So nothing that overly concerns us.

Peter Richard Lawson - *SunTrust Robinson Humphrey, Inc., Research Division - Director*

And then just on the ORR of around 15% in these late-line patients, that looks really encouraging. At what point do you feel comfortable that you perhaps got a more efficacious PD-L1? Or do you just think it's the better safety profile that's driving that better efficacy?

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

Well, we're -- like I said, I mean, we're really pleased with the activity. And you have to remember that this drug, when dosed, is an inactive prodrug, and no one has done this before. I mean, this is groundbreaking science that we are doing. And we agree with you. We think that this response rate is in the context of a dose escalation in a heavily pretreated patient operation is encouraging. As to whether there's any evidence that side of the monotherapy or the combination is more active, it's too early to tell. The patient numbers are just too small.

Peter Richard Lawson - *SunTrust Robinson Humphrey, Inc., Research Division - Director*

Got you. And then just on the ipi combo, any side effects that you're worried about? And is it, in any way, changing the way you are thinking about enrolling patients, whether it's on the side effect side of things or the efficacy side?

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

Well, the -- I'll ask Rachel to comment in just a moment, but really no, I mean, I -- again, we feel very encouraged. I think the types of tox that we're seeing, it's not just the number of events that we're seeing, it's also the nature of the events, they kind of look and feel like ipi. And so everything -- at this stage in the game, everything kind of makes sense to us, but we'll learn more. More patient follow up the additional dose escalation, but I -- yes, again, I think we're encouraged by what we're seeing.

Rachel Wallach Humphrey - *CytomX Therapeutics, Inc. - Chief Medical Officer*

Yes, I have nothing really to add. It looks and feels like ipilimumab, a drug we know very well. There are no surprises, no new toxicities that you didn't expect from either agents alone. And if anything, 2 of the 4 events are hyponatremia, amylase, lipase, those are not worrisome at all. No, I'm not troubled.

Peter Richard Lawson - *SunTrust Robinson Humphrey, Inc., Research Division - Director*

How many patients do we get for the next update? And that's my -- the last question.

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

We're not in a position to guide on that right now, Peter.

Operator

Our next question comes from Ying Huang of Bank of America.

Ying Huang - *BofA Merrill Lynch, Research Division - Director in Equity Research*

First one is I think the marked reactions today on stock price was probably a result of investors comparing the 31% treatment-emergent AE related to drug to the extended ipi trial data, which showed about 30% also are treatment-related AE Grade 3 and above. So maybe you can provide some context around that. And then also, secondly, I think on the response, do you guys -- I mean, I know it's a very small number of patients, but do you think you have started to see some dose-responsive efficacy here already at this point?

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

Let me take the second question first. It really is too early to tell. We feel good about the 10 mg/kg dose selection for monotherapy expansion. I think we need to collect more data. Let me -- and I'll ask Rachel to comment, Ying. I'll ask Rachel on your first question.

Rachel Wallach Humphrey - *CytomX Therapeutics, Inc. - Chief Medical Officer*

Yes, thanks for that question. So I think what you're referring to is there are several examples. I think checkpoint -- CheckMate 227 is the perfect example, where, in the interest of dropping toxicity, BMS meaningfully reduced the dose of ipilimumab to 1 mg/kg every 6 to 12 weeks. We would say, based upon an analysis of a wide range of combinations with PD agents, not just with ipilimumab but across a range of combinations, that efficacy and toxicity go hand in hand; and that if you reduce the toxicity without a Probody, you are potentially, in fact, one might argue, likely to be reducing the efficacy as well. Ipilimumab has unmistakably a dose effect. And if that is in 3, 3 is better than 1, toxicity creates a real challenge. And there are examples from CheckMate 032 and other places, Pad Sharma has some data, that if you use 3 of ipi in a combination with nivolumab, you begin to see real benefits in terms of activity with more toxicity. But if you have 1 milligram per kilogram of ipi, it doesn't look very much different than nivolumab alone. And those data are all extremely early, but they give us a hint that's worth pursuing, that we believe that if we can push the dose of ipilimumab up without added toxicity, in fact, you give me an opportunity to say if we could keep the toxicity at 30%, at 30%, and achieve the kind of efficacy that we could achieve with higher doses of ipilimumab, it's a huge win. Every patient on Part B was dosed with 3 milligrams per kilogram of ipilimumab.

Operator

(Operator Instructions) Our next question comes from Biren Amin of Jefferies.

Biren N. Amin - *Jefferies LLC, Research Division - MD and Senior Equity Research Analyst*

I noticed for the A2 cohort -- I mean, you're requiring mandatory biopsy. But given the data today, do you think that you would also encourage biopsy in the ipi combo cohort or the vemurafenib combo cohort?

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

We're not planning to do that just yet. I mean, I think that -- yes, I -- the A2 cohort is specifically designed to having made good initial progress on monotherapy dose-escalation without required biopsies. We feel comfortable doing an A2 cohort and taking the inevitable hit in the enrollment rate by requiring biopsies in A2. It's always a careful balance, right? So I think we'll get to that in due course, but we're not planning to do that at this point.

Biren N. Amin - *Jefferies LLC, Research Division - MD and Senior Equity Research Analyst*

Got it. And then just for the ipi combo [Group B] cohort, I know you continue to dose escalate up to 30 mgs/kg with 072. But you've also decided on a go-forward dose on a monotherapy of 10 mgs/kg. So I guess what the rationale to continue upwards to 30 mgs/kg in the combo cohort?

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

Well, I'd like to use a maximum with -- yes, with the company. Don't make too many assumptions. And we have, I think, a well-laid-out clinical experiment to do, and we should continue to collect data. We don't know that the combo is going to behave in exactly the same way as monotherapy, and I think that there are more questions to be asked there, so that's the main reason.

Operator

I'm showing no further questions at this time. I'd like to turn the call back over to Sean McCarthy for any closing remarks.

Sean A. McCarthy - *CytomX Therapeutics, Inc. - President, CEO & Director*

Great. Thanks very much, and thank you all for joining us today. I hope we've conveyed to you all that this is a very exciting time for us at CytomX, and we look forward to updating you as our programs make additional progress and as we announce additional data from the portfolio throughout 2018 and into 2019. So have a good evening, everybody.

Operator

Thank you. Ladies and gentlemen, this does conclude today's conference. Thank you for your participation, and have a wonderful day. You may all disconnect.
