

## Q1 2020 Earnings Conference Call

### **Executives and Speakers**

Garó Armen, Ph.D. – Chairman and Chief Executive Officer

Jennifer S. Buell, Ph.D. – President and Chief Operating Officer

Christine Klaskin – Vice President, Finance

Dhan Chand – Head of Drug Discovery

Burcu Yigit, PhD – iNKT Lead Scientist, AgenTus

### **Operator**

Good morning, ladies and gentlemen. Thank you for standing by, and welcome to the Agenus First Quarter 2020 Conference Call.

Please note this event is being recorded.

I would now like to turn the conference over to Dr. Jennifer Buell, President and Chief Operating Officer of Agenus. Dr. Buell, please go ahead.

### **Jennifer Buell**

Thank you. Thank you Sienn. Today's call is being webcast and will be available on our website with our accompanying slide material for replay.

Before we start, we'd like to remind you that this call will include forward looking statements, including statements regarding our clinical development and regulatory plans and timelines, as well as timelines for data release and cash projections. These statements are subject to risks and uncertainties and we refer you to our SEC filings for more details on these risks.

As a reminder, this call is being recorded for audio broadcast

I'm Jennifer Buell, President and Chief Operating Officer of Agenus and joining me today are Dr. Garó Armen, Chairman and Chief Executive Officer; Dr. Dhan Chand, Head of Drug Discovery; Burcu Yigit, Lead Scientist for allogeneic iNKT cells and Christine Klaskin, our Vice President of Finance.

I will now turn the call over to Garó.

### **Garó Armen**

Thank you very much, Jen, and a special thank you to all of you for joining us during this time when the world has turned upside down because of the COVID-19 pandemic. I hope you and your families are safe and well.

Our team at Agenus has found a productive way to operate in this new norm. At the earliest signal of the pandemic, our team reacted swiftly and took proactive measures to protect our employees, their families, and our business. As a result, our operations in Berkeley, Lexington, and Cambridge have remained opened with a rotating work schedule for safety, and with limited interruption to our key priorities. We operate with approximately one third of the workforce onsite and the remainder working from home, and that has not disrupted any part of our operations so

far. And importantly, to the best of our knowledge, none of our employees have been infected with the virus so far, and we'd like to keep it that way.

Earlier this year, we projected receipt of approximately \$60M in cash milestone payments for 2020. As we have announced, we have already received \$15M based on the sales of Shingrix, which has our QS-21 adjuvant in it. However, given the environment, the remaining \$45M is uncertain because of the disruptions caused by COVID-19, and hence, we've taken measures to result in annualized cost savings of approximately \$50M. These savings are driven by reductions of both external and internal expenditures, and importantly, they will result in slowing down of several programs, but we do not expect these steps to affect our near- or medium-term commercial launch prospects. We also expect that our high priority clinical development and research programs will proceed as planned.

Our proactive measures are designed to deliver our key milestones this year, including plans to file our BLA filings; our acceleration of clinical development of our next-gen, multi-purpose CTLA-4 molecule, 1181, about which you will hear much more from Dr. Buell; our advancing of iNKT cell therapy to the clinic for the treatment of cancer and COVID-19 patients – that you will hear from Dr. Burcu, who will also tell you about the advantages of our iNKT cells, including, for example, substantially reduced costs associated with cell therapy when we practice iNKTs; and also, you will hear that we're advancing important programs like our TIGIT molecules into the clinic, and as you know, we have started this program a number of years ago and now we're ready to take that into the clinic.

I will now turn the presentation to Dr. Jen Buell, our President and Chief Operating Officer, to provide you with an update on our highest priority programs starting with AGEN1181, our multi-purpose next-gen CTLA-4. Jen?

### **Jennifer Buell**

Thank you very much, Garo and hello everyone. As Garo mentioned, I'll start by providing an update on AGEN1181 and there's a summary of this program on Slide 4 that we'll review today.

This molecule is familiar to some of you. As Garo said, it's our multi-functional T cell antibody which also binds to CTLA-4. Now, importantly, this molecule is engineered. The Fc region of this molecule is engineered. We did this because we knew of some key features that this engineering could provide. Those features included increased immunogenicity; increased T-cell activation, beyond which we see with first-generation CTLA-4s; superior combinations with PD-a, as you see on Slide 4; as well as important potential safety benefits such as a reduction in endocrinopathies for patients treated. And we expect to increase or broaden the patient population of responders, patients who will respond who are unlikely to respond to a first-generation CTLA-4 due to a genetic polymorphism that impacts about 40% of the population.

And what we're going to talk to you about today are data from this trial, which will show that as the molecule has been designed to benefit patients who harbor this genetic polymorphism, patients are benefitting from AGEN1181. We have also seen no endocrinopathies, none of the safety-related side effects that we see with Yervoy related to hypophysitis or otherwise; very important features.

Fc engineering is something that we know to be really quite important. We've published on this technology and our techniques on our 1181 -- AGEN1181 molecule in Cancer Cell last year.

We've also, as Dhan will speak to you today, we've also engineered our TIGIT molecule in order to bring similar enhancement. We've shown you data that supports the benefit of this engineering, and we're also now seeing data from late-stage clinical trials with an Fc-engineered HER2 antibody called margetuximab. You may be aware of some of that data.

So, on slide 5, I'd like to summarize. AGEN1181 is advancing in a phase 1 dose-escalation trial. This next generation antibody has shown profound activity in the form of objective responses which are unusual to see this early in clinical development. We're hopeful that based on the current trends, and as patients continue on therapy with longer follow up, we'll see more patients with durable responses.

Now, as you may have heard a number of hospitals have been converted to treat patients with COVID-19, and this could impact, of course, some clinical trials, as well as patient access - - patients with cancer getting access to their therapies. However, in spite of this reality, we continue to accrue to our trial without interruption. Our principal investigators have informed us that based on the data to date, AGEN1181 has life-saving attributes that support the continued enrollment of this trial, and today, we have a queue of patients waiting to be enrolled.

Now, you may remember, as I mentioned earlier, that AGEN1181 was deliberately designed to improve the efficacy and safety of first generation CTLA-4s. In addition, this antibody is expected to expand the patient population who will benefit, and now I'm going to highlight some data that supports the design of this molecule and the activity that we're seeing.

- In our dose escalation trial, AGEN1181 alone and in combination with balstilimab, demonstrated a clinical benefit rate of 70%. This includes complete, partial responses, as well as disease stabilization of patients with late stage cancers.
- Now, during our last call, we described a patient with refractory endometrial cancer. This patient had late stage disease. She had failed all prior therapies. And importantly, she had a very poor prognosis based on available biomarkers that help us determine a patient's likelihood to respond to therapy. These markers include: The patient was negative for PD-L1; the patient had microsatellite stable disease, which is unlikely to respond to immune therapies; and this patient had the genetic polymorphism in her CD16 allele that rendered her unlikely to respond to a first generation CTLA-4. In spite of her negative odds, we are thrilled to see that this patient is a confirmed complete responder on AGEN1181 monotherapy at 1mg/kg.
- Now, on Slide 6, I want to highlight another very exciting case. This patient is a patient with refractory endometrial cancer and a similarly poor prognostic profile with metastatic disease in multiple locations, as you can see here. This patient is now also a confirmed partial response in her target lesions with a complete response in her non-target lesion. This patient responded to very low dose of AGEN1181 in combination with balstilimab, our PD-1 antibody.
- If I can now direct your attention now to Slide 7. Last week, we convened with our advisory board. Now this advisory board is comprised of top immune-oncology experts, some of whom are specialists in the CTLA-4 mechanism, some of whom were the first to dose patients with the first generation CTLA-4 over 20 years ago. After an extensive review session, our advisors endorsed our accelerated development path for AGEN1181, which we are in the process of launching as we speak.
- For this phase of development, we will pursue a fast to market strategy. This means going after cancers where there is limited or no effective treatment option available to these patients. These tumors are highly prevalent, or said differently, large cancer markets, large commercial

market opportunities. These opportunities include PD-1 refractory NSCLC, PD-1 refractory melanoma, and MSS tumors, like CRC as well as endometrial, which are the two cases that I've shared with you just a few moments ago. We'll continue to keep you updated as these programs mature.

I'll now turn to our lead programs: our PD-1 program, balstilimab, which we call Bali and our CTLA-4 program, zalifrelimab, which we call Zali. The results that you see on Slide 8 show the evolution of cancer therapies for women with cervical cancer, starting with chemotherapies on the left and VEGF inhibitors, which are still widely used, despite their limited benefit.

The data that we've presented now from our programs, balstilimab and zalifrelimab, are based on a large cohort of 55 patients who had a median of 12 months of follow-up. Importantly, all responses were confirmed by an INDEPENDENT radiology review, which is a gold standard in assessing outcomes in cancer. We have summarized our findings in a recent Agenus newsletter which is available on our website.

Notably, we have observed 14 objective responses. These include 4 complete responses and 10 partial responses in these 55 patients evaluated so far. This is a response rate of 26%.

As a reference MRK was granted accelerated approval for their PD-1 antibody based on 11 responses in 77 patients with a response rate of 14%. As you can see here, our combination has the potential to become the most effective treatment available to metastatic cervical cancer patients.

And a few comments regarding cervical cancer patients and our plans:

1. Cervical cancer is a horrifying disease, particularly for young women who come from economically disadvantaged backgrounds and have limited access to healthcare. Patients are diagnosed and then treated with toxic chemotherapy which has many difficult side effects and little clinical benefit. Their cancer ultimately progresses with little to no effective treatment options available to them, as you can see here.
2. We're committed to changing this reality. Data from cervical cancer clinical trials with Agenus' "bali/zali," our PD-1 and CTLA-4 antibodies, has shown a near doubling of responses as compared to currently available treatments, and importantly, most importantly for us, is that these responses are durable. These patients are not converting or progressing once they're responding, which is incredibly important to patients. And, also importantly, we're seeing patients with long-term disease stabilization who later convert to response.
3. During our recent year end call we mentioned that we had received Fast Track designation from the FDA for the investigation of balstilimab alone and in combination with zalifrelimab in relapsed, refractory or metastatic cervical cancer.

We plan to submit our BLA filings this year and will continue to keep you informed on our progress.

Now, I'm going to wrap up with two very exciting programs. During a recent meeting with clinical experts, they noted that we have the most productive research engine in I-O. I agree. Today, I'm joined by two of our lead scientists responsible for our next innovations expected to enter the

clinic very soon. Dr. Dhan Chand and Dr. Burcu Yigit are leading our TIGIT and our iNKT programs, respectively. They're joining us here to talk about these programs today.

As you all know, TIGIT is shaping up as a powerful combination partner with PD-1 antibodies; especially in tumors expressing TIGIT. We've designed two different approaches to optimally target TIGIT.

- 1) Our Fc engineered anti-TIGIT antibody which has outperformed all tested competitor antibodies and showed superior T cell activation when combined with PD-1 or LAG-3 antagonist AND;
- 2) Our TIGIT bispecific molecule, AGEN1777, which has demonstrated potent tumor killing as monotherapy in a difficult to treat cancers where PD-1 antibodies alone are ineffective.

In addition to these two TIGIT antibodies, we are rapidly advancing our allogeneic cell therapy to the clinic to treat patients with cancer and patients with COVID-19. We expect both INDs to be cleared shortly, and as a matter of fact, one may clear as early as this week. I note that, like us, the FDA has been working through weekends to process applications which is very heartening during this health crisis.

Burcu will tell you more about these cells. She's trained in molecular biology and immunology and she joined us from Harvard/Beth Israel Deaconess Medical Center. Burcu?

### **Burcu Yigit**

Thank you, Garo and Jen.

So, invariant NK Tcells, short for iNKT cells, have several advantages over other allogeneic approaches in development including, but not limited to their significant expansion capacity, their use in an allogeneic setting without requiring genetic manipulation and their ability to suppress GvHD. I am excited to report that our team filed 2 INDs to advance our iNKT cells to combat cancer and separately COVID-19.

iNKT cells are a rare population of lymphocytes and importantly, in late stage cancer, the frequency and the function of iNKT cells is highly correlated with overall survival. Unmodified iNKTs are a natural component of innate immune system and their reduced numbers or function is associated with poor immune response to cancers and poor prognosis of patients with late stage disease. You can see on Slide 9 some of the notable advantages of iNKT cells.

So, first of all, iNKT cells can penetrate into the tissue, which gives them a critical advantage to target solid tumors that are not served by approved cell therapies.

Secondly, iNKT cells can kill cancer without requiring genetic manipulation. So, employing iNKTs in an unmodified form, patients can receive the treatment quickly and at a significantly lower cost.

And third, iNKTs are expected to target SARS-CoV-2 and cells infected with the virus. iNKTs should be agnostic to mutations of the current strain and other coronaviruses. While severe complications of SARS-COV-2 infection are characterized by a life-threatening respiratory disorder and other organ failure, our allogeneic iNKT therapy, agenT-797, is expected to help clear the virus while controlling harmful inflammation caused by the virus. Preclinical data demonstrates that iNKT activation induces a rapid antiviral response and enhances immunity to respiratory and other infections. Also, iNKTs have been shown to prevent lung injury in preclinical

models by reducing the number of inflammatory cells dampening exacerbated inflammation and thus preventing or controlling tissue damage. This may be especially important in COVID-19.

Agent-797 could also promote long term immunity against COVID-19, which would be an important outcome to protect both recovered patients from re-infection and to protect healthy individuals from the disease.

So, finally, an important benefit of our allogeneic approach is its scalability. We have demonstrated that iNKT cells from a single donor are manufacturable and scalable – just based on our early process development work, we have developed a scalable process designed to manufacture about 1,000 doses from a single donor.

While our near-term clinical trials will help define the key features of agent-797 in cancer and infectious disease, we are ready to quickly design optimal combinations with our cell therapy and checkpoint antibodies. My colleagues will be presenting data at AACR on our optimal combinations with key insights into the criticality of these approaches and the significant differentiation that Agenus has in delivering these combinations.

Thank you very much and now, I will turn it over to my colleague, Dhan Chand to discuss our TIGIT program.

### **Dhan Chand**

Thank you, Burcu and hello everyone.

Today, I'm excited to discuss two of our TIGIT antibodies, why they're important and how we believe they will change the treatment paradigm.

As Jen mentioned, TIGIT is shaping up as a powerful combination partner with PD-1 antibodies; especially in tumors expressing TIGIT. Our portfolio of TIGIT targeting antibodies includes an Fc enhanced TIGIT monospecific (AGEN1327) and our TIGIT bispecific (AGEN1777). We believe both molecules have unique advantages over other TIGIT antibodies that are in clinical development.

Since TIGIT may be new to many of you, I will first describe what TIGIT is, as seen on Slide 10.

TIGIT is a receptor primarily expressed on T cells and NK cells. TIGIT attenuates innate and adaptive immune responses by inhibiting the actions of T cells and NK cells. In addition, it increases the immune suppressive activity of regulatory T cells. TIGIT is overexpressed in multiple tumors and is known to be a key player in driving resistance to anti-PD-1 – and as a result, tumors grow.

Blocking TIGIT with antibodies like our monospecific TIGIT antibody, AGEN1327, or our TIGIT bispecific, AGEN1777, unleashes important immune cells, such as T cells and NK cells to kill many types of cancer.

Agenus was the first to discover and report, in Cancer Cell, and at AACR in 2019 that TIGIT antibodies require Fc gamma receptor co-engagement to promote optimal T cell activity against tumors.

Our TIGIT, AGEN1327 is engineered with this Fc enhancement and has outperformed all tested competitor antibodies and showed superior T cell activation when combined with PD-1 or LAG-3 antagonists or OX40 or CD137 agonists - we presented this data at AACR 2019.

Our TIGIT is an ideal combination partner for addressing known resistance mechanisms to current checkpoint therapy and with the potential to provide deeper responses.

In addition to superior function demonstrated against tested competitors; our molecule was designed to:

1. Maximize anti-tumor activity like the robust activity observed with our Fc engineered AGEN1181, that has already shown remarkable activity in early clinical trials. Our preclinical data with our TIGIT also showed superior tumor killing compared to competitor molecules, as seen on Slide 11
2. Be an optimal combination partner for anti-PD-1 antibodies for more tumor – potent tumor killing; particularly for TIGIT expressing tumors, including, non-small cell lung cancer (NSCLC), and,
3. Expand the population of cancer patients who will benefit from TIGIT by targeting all genetic polymorphic variants of this particular Fc receptor.
  - a. We described this vulnerable population earlier and showed benefit in patients with the polymorphism experienced benefit from our Fc-engineered AGEN1181, including a complete response, in patients with this polymorphism.

We are actively advancing IND enabling activities to bring AGEN1327 to the clinic early next year.

Importantly, TIGIT has also been implicated as an important target for overcoming resistance to anti-PD-1 therapy. Our Bispecific TIGIT Antibody (AGEN1777) is designed to be used as monotherapy for tumors which are unresponsive to PD-1 antibodies. AGEN1777 is a first-in-class TIGIT bispecific that leverages our internal multi-specific platform to co-target another inhibitory receptor (not yet disclosed), but also expressed on T cells and NK cells. We discovered, that co-targeting TIGIT with this undisclosed target using our bispecific approach provides superior immune activation as compared to the combination of monospecific antibodies to the same target. This TIGIT bispecific approach, when used alone, has potent tumor killing activity in a colon cancer model where PD-1 monotherapy is ineffective. Therefore, AGEN1777 can be important therapy in PD-1 relapsed/refractory tumors.

While PD-(L)1 antibodies have been a spectacular commercial success, only a small proportion of patients have had sustainable long term benefit. Therefore, there is substantial need for therapies in patients who relapse or do not respond to PD-1 monotherapy. We expect to file an IND on AGEN1777, our TIGIT bispecific, by the end of 2020.

There is growing conviction that targeting TIGIT will provide a breakthrough in I-O, and we are uniquely positioned with two distinct molecules on track to be launched into clinical development as early as the first half of 2021.

Now I will turn the call over to Christine Klaskin to provide a financial update.

**Christine Klaskin**

Thank you Dhan.

We ended the first quarter of 2020 with a cash balance of \$92M as compared to \$62M at December 31, 2019.

For the first quarter ending March 31, 2020, we reported a cash burn from our operations of \$32M. Net loss for the quarter was \$45M or \$0.31 per share which included non-cash expenses of \$16M. We generated net income for the same period in 2019 of \$17M or \$0.14 per share. In the first quarter of 2019 we recognized revenue of \$80M which included revenue related to the upfront license fee from our transaction with Gilead in addition to non-cash royalties earned. For the same period in 2020 we recorded revenue of \$15M primarily related to non-cash royalties earned.

Now I will turn the call over to Garo to close.

### **Garo Armen**

Thank you, Christine, and thank you very much to the team, who explained our current state of affairs and our prospects very well.

Today we are a company of 260 persons, but it's not the number that matters; we have capabilities to innovate, to develop, to manufacture our discoveries, and we're currently building our commercial capabilities to become a fully integrated company. We have stressed all along, speed and innovation are key in our business, particularly a business that has very, very exciting prospects going forward, and Jen Buell talked about the fact that our external advisors have made comments about the fact that we may be the most innovative, productive company in the field of I-O.

Now, we can all, of course, do these things because we have end-to-end capabilities from novel target discovery to full GMP manufacturing for antibodies. This has been critical to our productivity. We have brought 14 new discoveries to the clinic and launched six clinical trials, and we are gearing up to file our first BLA in the third quarter of this year. We've developed our clinical operations team and delivered the full target accrual of our clinical trials in the last two years, and these may sound like -- trivial to some, but we have delivered 11 GMP-manufactured batches for our own trials and partnered programs at our manufacturing site at Agenus West, and the team there have done an absolutely terrific job. And as we've said before, we're continuing operations with no interruptions. We have a queue of additional batches to manufacture this year.

And as Burcu said, we have launched a cell therapy company, AgenTus, which designed a very unique allogeneic cell therapy approach. With the kind of advantages that we're talking about, for example, for COVID-19, it offers, potentially, the capability of being an antiviral therapy for anti-inflammation therapy all in one, and do this with a single cell source, which means it's an off-the-shelf cell therapy that drives cost down and, most importantly, it drives the speed of patient availability up.

Now, with all this, we've also talked about in the past that to date, we have generated over \$540M in cash from partnerships, collaborations, transactions with multiple pharmaceutical companies like Gilead, Merck, GSK and Incyte, as well as UroGen, and all in the last four-years-plus. And, also, importantly, we expect additional partnership transactions this year. We are in term-sheet discussions with companies already.

Now, I think given the fact that my colleagues have done a terrific job of keeping you abreast of everything that is going on, I will stop here and entertain any questions that you may have.

Thank you, I will now take questions.



## Questions & Answers

### **Operator: (Operator Instructions)**

Your first question comes from Matt Phipps from William Blair.

**Unknown Analyst:** This is Hunter on for Matt. Just a couple of quick questions. I was wondering if you could provide some color on the unmentioned part of the TIGIT bispecific. I know you didn't mention specifically what the other part was, but maybe sort of a mechanism that you're targeting there.

And then just a follow-up: I think previously you'd mentioned that, with the lowest dose of 1181, you had a pending response, and I was just wondering if that was still pending, or a confirmed nonresponse? Thank you.

**Garro Armen:** Okay. So, let me just make a general comment and then I'll turn it over to Dhan and then to Jen to address the 1181 question. But as some of you may know, we have a very sophisticated competitive intelligence capability at Agenus, and this has been a critical component of our decision-making, our strategy, that drives from research strategy onwards. So I had mentioned that we've been working on TIGIT for quite some time, but all along, we have evaluated what is happening with competitors out there in the TIGIT field, and what is it that we can add incrementally or leapfrog the other programs so that we come up with an advantage with a product that offers advantages to our company, of course, but also, importantly, advantages to the patients? And our TIGIT programs are specifically designed to address what is needed, what are the advantages we can provide? So Dhan will answer your question more specifically, Matt.

**Dhan Chand:** Thank you for the question. The second target in our bispecific, which we have not yet disclosed, is a first-in-class target that is expressed on T cells and NK cells. What we have discovered is that the best way to target this first-in-class unidentified -- which we have not yet disclosed -- target is to do so in the form of a bispecific that includes TIGIT. Overall, what we have discovered is that when we co-target TIGIT and this yet-to-be-identified receptor, we see superior activity as compared to that of monospecific approaches to each of these targets. I would like to emphasize that the second target, which we have not yet disclosed, is a first-in-class target.

**Jennifer Buell:** Thanks, Dhan, and thanks, Hunter. Regarding 1181, we presented data on two different patients; both of the responses have been confirmed by independent radiology review.

**Garro Armen:** And also, I might add, they have been continuing. I think it --

**Jennifer Buell:** Yes.

**Garro Armen:** One of the hallmarks of immunotherapy for everybody is the benefit of having responses last for a long time or convert to a curative outcome, and I think everything that we're seeing, from our first-generation products, bali and zali, to our second-generation products so far, 1181, are indicative of lasting responses that may convert into a curative outcome.

**Operator:** Your next question comes from Mayank Mamtani.

**Mayank Mamtani:** I appreciate the efforts you are putting in on COVID-19. Just quickly on zali/bali, on the BLA filings, could you just remind us? It seems like the clinical section is full. Are you just working on the nonclinical side? What are some of the other things you have to do, if there's an FDA meeting before you're able to submit the BLA? Could you just remind us on the

process?

**Jennifer Buell:** Sure. Thanks very much, Mayank, for the question. Nice to hear you. Regarding the process, so we're pursuing rolling submissions, which include meeting with the FDA to discuss different components of the filings. And what I'll share with you is that for elements like our manufacturing component, the quality -- the agency has remarked on the quality of our manufacturing and the completeness of our prior interactions with them for INDs and otherwise. We have already engaged and discussed and are very confident in the completeness of a number of the modules, including manufacturing.

On the clinical side, we're continuing to collect data. We've completed accrual, and as you know, patients with refractory cervical cancer, if they are to progress, they do so in a relatively short amount of time. So, we'll have a good sense of our complete cohort very actively, and we're in the process of analyzing the data and readiness for the submission.

So the interactions -- we've had a series of interactions with the agency because we accelerated this program from a first-in-man study rapidly to a Phase 2 expansion in refractory cervical cancer, at which point we met with the agency and we discussed our strategy and our trial design, and now it's a matter of continuing to engage them to provide updates on each of our molecules, our pharmacology and nonclinical, our manufacturing and clinical. And those -- some of which have been completed, and of course, a couple of which are continuing to proceed until our filing date. And as Garo mentioned, we'll be filing in the second half of this year.

**Mayank Mamtani:** Great. And then on 1181, about the recent advisory board meeting you had, could you just talk maybe qualitatively to the stable disease that you're seeing? Are these in lung and colon cancers that is informing sort of your next choice to go into these bigger indications? Any color there would be great.

**Jennifer Buell:** So Mayank, I'll be thoughtful about my response here because we are anticipating some upcoming data presentations at major medical conferences. So what I will share with you are some of the -- this is a solid-tumor study, and we have represented cancers that we've discussed with the advisory board, and you see on the slides I presented today, including lung and melanoma, enrolled in the trial. And we've seen activity beyond those indications as well, including other gynecologic cancers like ovarian, for example.

And an important case that will be described in much more detail soon is a gynecologic case with very long and durable disease stabilization, nearly a year, which is incredibly unusual for this highly refractory case. So, this is disease stabilization that has not yet met the RECIST criteria for a response but looks really quite active.

We're seeing some similar trends in other tumor types, disease stabilization in the majority of patients treated. And so, you could -- I'll leave it at that so that we don't restrict ourselves to what we can present at some upcoming conferences.

**Mayank Mamtani:** And I understand and appreciate the qualitative color. And maybe just one final one on the COVID-19 efforts you have: The -- it seems like you're nearing IND acceptance, so any color on how you're thinking to develop and what institutions that you may decide to partner with? Obviously, there are certain hotspots that could benefit from having a trial like this. So, any color there?

**Garo Armen:** Well, I think, Mayank, we need to first get signals from the clinic, and the objective

of our first trial now is to see -- to look at a whole bunch of blood markers for these patients and see how they respond to therapy in a dose-escalation study. And so, bear with us; I think it's premature for us to really determine what the next steps are going to be, beyond this exploratory clinical trial.

**Mayank Mamtani:** So, to clarify, these are going to be non-severe COVID-19 initially, patients?

**Jennifer Buell:** So Mayank, first, on the hotspots, yes, we'll be -- the cells will be in the New York population. We have collaborators in important New York hospitals who have been at the front line of this. The cells we will be exploring, because the cells can mitigate the virus and also dampen harmful inflammation -- which in some cases patients experience after-viral clearance, so there are two very important features -- we will first explore these cells in a more severe setting. We will evaluate the persistence of the cells, of course, the activity of the cells in viral clearance as well as in dampening harmful cytotoxic release syndrome, CRS.

**Mayank Mamtani:** Really great. And just one last thing on financials: I believe there were two different tranches of milestones you were anticipating, one Shingrix-related and one progression of, I think, some pipeline molecules with Gilead. So, in your revised guidance, any color on what you are seeing that might be addressed for the remainder of the year, Garo, maybe?

**Garo Armen:** Okay. So, as I mentioned, we have received the first tranche of the GSK Shingrix royalty milestone, so that's behind us. That's \$15M. There was a second tranche potentially due in the second half of this year, but given the guidance provided by GSK, which suggested a significant downturn in Shingrix revenues going forward, it's unlikely that we will meet the revenue milestone which would have driven the second payment for us. So that's one.

Now, additionally, there are two sets of milestone payments that we were contemplating from two separate companies. And what we've done is have a more conservative posture and say, we have no control over the timing of the clinical trials associated with either one of these situations, so we're planning for no milestones. Now, that doesn't mean we're not going to get them; it just means that we want to be conservative and plan for no additional milestones for the balance of the year so that we can prudently manage our finances. And as I had mentioned, we have initiated \$50M worth of annualized savings, which are already in place, I might add, and that will, of course, give us a little bit more comfort.

And then, in terms of new potential collaborations, we are in term-sheet discussions with two separate parties, and I expect that that will result in upfront cash payments that will help us manage our cash for the balance of the year and into next year.

Any other questions? Okay. Well, that concludes our call, and I may want to turn this over to Sienn, who will do a stellar job of concluding remarks. Sienn, would you end the conference?

**Operator:** Thank you. The conference has now concluded. Thank you for attending today's presentation. You may now disconnect.