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Apexigen

Apexigen Investor Day

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The following is a transcript from the Apexigen, Inc. Investor Day conference call held on May 16, 2022.

CORPORATE PARTICIPANTS

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PRESENTATION

Tara Sobierajski

Good morning and welcome to the Apexigen Investor Day. At this time all attendees are in a listen-only mode. A question-and-answer session will follow the formal presentations. If you would like to submit a question, you may do so using the Q&A text box at the bottom of the webcast player or by emailing your questions to questions@lifesciadvisors.com.

As a reminder, this call is being recorded and a replay will be made available on the Apexigen website following the conclusion of the event.

I would now like to turn the call over to your host, Dr. Xiaodong Yang, President and Chief Executive Officer of Apexigen. Please go ahead, Xiaodong.

Xiaodong Yang

Thanks, Tara. Good morning, everyone. Welcome to Apexigen's Investor Day presentation. This event is sponsored by Apexigen and LifeSci Advisors. Next slide, please.

Joining me today is our Chief Medical Officer Frank Hsu, and also our Chief Operating Officer Francis Sarena. Next slide, please.

Before we get started, I just want to draw your attention to our Disclaimer Statements. Next slide.

Today at this Investor Day presentation we'll go through the following agenda. We will start with an overview of the company, then we'll focus on our lead asset sotigalimab, its mechanism of action, competitive landscape analysis, more importantly focusing on updating our clinical development programs and data. Then we'll talk about our preclinical pipeline, APX601 and our platform technology APXiMAB. Towards the end, we will give you an overview of our recently announced business combination agreement with Brookline Capital Acquisition Corp., the PIPE and the equity line transactions. In the end, we'll be happy to answer any questions you may have. Next slide, please.

Apexigen is a clinical stage oncology company focusing primarily on immuno-oncology. We are a leader in discovering and developing innovative therapeutic antibodies in oncology. We have built a very strong pipeline with our lead molecule sotigalimab, or APX005M, in late stage clinical development with multiple data readout milestones within the next 12 to 18 months. We do believe sotigalimab is potentially the first-in-class and best-in-class CD40 agonist.

In addition to our lead molecules, we also built a pipeline with two pre-IND and preclinical assets. The next one APX601 is a TNF receptor 2 antagonist. We plan to file an IND this year. This is followed by APX801, which is an NK cell engager program.

Beyond our pipeline, we also have developed a strong partnership with five licensing partners who are developing their therapeutic antibody created from our APXiMAB platform. In 2019, the first APXiMAB-derived therapeutic antibody Beovu was approved, which has great value for validation of the platform and is generating royalty revenues for Apexigen.

As you can see, the company pipeline, lead molecules and our partnerships are all built on our validated APXiMAB antibody discovery platform. Next slide, please.

Slide 6 highlights and summarizes our pipeline. The Company's wholly-owned pipeline is shown in blue and our partner programs are shown in grey. As you can see on the top, the Company's wholly-owned program, sotigalimab is the most important asset and also the most important program for the company. We have multiple Phase 2 trials ongoing with a focus on melanoma, esophageal/GEJ cancer, sarcoma, rectal and ovarian. Also, we have two preclinical, pre-IND programs in the pipeline.

In addition to Beovu, which was approved in 2019, our other licensing partners are continuing to advance their programs. We look forward to more BLA approvals and more validation of our APXiMAB platform.

Now I will introduce Francis to give you a highlight of our pipeline in detail, and also the milestones. Next slide, please.

Francis Sarena

Thank you, Xiaodong.

As you can see, we have multiple milestones in our pipeline through the remainder of this year and into 2023. In our sotiga program in the melanoma setting, we are building on the positive readout we reported at SITC last year for our Phase 2 trial in PD-1 refractory patients. We plan to meet with the FDA in a Type C meeting mid this year to inform the design of a potential registration-enabling trial in this setting.

I'll now move to our study in esophageal and GEJ cancers in the neoadjuvant setting.

We have completed enrolment in our Phase 2 trial and plan to disclose PCR data from this trial from all enrolled patients in the second half of this year. As those data mature, we plan to disclose six-month duration of response data in 2023.

Our Phase 2 sarcoma trial continues to enroll and we plan to disclose updated response rate data, ideally for all enrolled patients, in the second half of this year.

Moving to our preclinical pipeline, we are excited to be moving our next pipeline asset towards the clinic. We are on track for a midyear IND filing for our APX601 program targeting TNFR2.

I'll now turn the call back over to Xiaodong to introduce our sotiga program. Xiaodong?

Xiaodong Yang

Thanks Francis. Next slide, please.

Next slide please.

Why are we interested in CD40? As you know, CD40 plays a very important role in controlling the regulation of immune response; therefore, targeting CD40 has multiple benefits. Number one, as you can see on the right-hand cartoon, CD is expressed on antigen-presenting cells such as dendritic cells, B cells and macrophages. Activation of antigen-presenting cells can activate both innate and adaptive immune systems, which can lead to more effective antitumor response.

Secondly, T cells cannot really see the tumor antigen unless the antigen is presented by activated antigen-presenting cells, and CD40 plays a very important role in activation of antigen-presenting cells.

Last but not least, activation of the CD40 receptor on a macrophage can convert an immunosuppressive macrophage to an M1 macrophage, which is an immunostimulatory macrophage, which can convert a cold tumor to hot, which allows other immunotherapeutic agents to act more effectively in the tumor microenvironment. Next slide.

We believe sotigalimab is first-in-class and best-in-class CD40 agonist because we have an innovative design of the CD40 agonist antibody sotigalimab, we have demonstrated clinical activity in multiple indications, and we have a broad clinical development program for sotiga. Next slide, please.

Because CD40 is such an important target, we have done things to make sure that our CD40 agonist sotigalimab has the potential to be the best-in-class. We have done two very important things to our molecules. Number one, we select sotigalimab which binds to CD40 ligand binding domain. This is very important because the binding to CD40 ligand binding domain allows our antibody to activate CD40 and mimic the natural ligand, which also increases the CD40 agonist potency. Secondly, we introduced a single mutation on the Fc region which increased the binding to FcgIIbR which is a very important receptor to crosslink sotigalimab to increase the potency. The same mutation abolished binding to other Fc receptor, FcgIIaR. This will prevent depletion of CD40-expressing antigen presenting cells or IgG1 antibodies.

Next slide, please.

What are the key differentiations?

If you look at that left-hand side, the key differentiation of sotigalimab is twofold. Number one, sotigalimab is the only known CD40 agonist antibody binding to CD40 ligand binding domain, as I mentioned earlier, which is very important for the antibody to mimic the natural ligand stimulation of CD40 receptor. Secondly, it's also important to increase potency.

The second aspect of differentiation is the mutation engineered into the Fc region. We have a single mutation introduced into the Fc region to increase the potency and avoid potential toxicity of depleting antigen-presenting cells. The combination of the unique ligand binding domain epitope coupled with the Fc engineering makes sotigalimab differentiated from the other CD40 agonist antibodies in clinical development.

In terms of our clinical development stage, as you can see on the right-hand side, we have eight ongoing Phase 2 trials targeting seven different indications. We believe sotigalimab is the most advanced CD40 agonist program in clinical development.

Now, I will introduce our CMO Frank to give you more updates on our clinical development of sotigalimab. Frank? Next slide.

Frank Hsu

Thank you Xiaodong. Can you hear me now?

I guess I'm not seeing myself on the screen so I just wanted to make sure you can hear me okay, I'm assuming.

Thank you Xiaodong.

Our clinical development program has been based, really, on trying to maximize potential sotigalimab mechanism of action. As discussed, sotigalimab is a very potent agonist of CD40 and an important function of CD40 is to activate antigen-presenting cells, particularly by dendritic cells. What I'm referring to is sotigalimab, through CD40 signaling, has a key role in activating immature dendritic cells to become fully mature professional aDCs. An immature DC is what circulates in the blood with antigens such as tumor antigens and it then has to receive the right activation signals to become a fully functional aDC. That's really when it becomes a much larger cell, it expresses critical immune host stimulatory molecules and produces a whole array of cytokines that allow it to potentially stimulate T cells and other immune cells very effectively.

These activated dendritic cells are believed to be the most potent stimulators of both primary as well as secondary immune responses. That means that you can stimulate initial responses and then re-stimulate responses as needed.

This is an important immunologic event which is very different than checkpoint inhibitors such as PD-1. Whereas PD-1 may release the brakes on existing T cells, that's a later effect; you have to have the T cells first. So, sotigalimab can help generate new antitumor immune responses, which can then increase the number of and potentially the repertoire of responding T cells.

So, based on this activity, we would expect sotigalimab could potentially have potent antitumor responses by itself as single-agent activity, and we've seen this in a patient population that was I/O naïve melanoma patients in an ongoing study. We have seen two durable complete responses, each one lasting more than a year.

Now, if you were to expand antitumor T cells and the repertoire, then you might have more T cells that could be acting synergistically with the novel anti-PD-1 or other checkpoint inhibitors, and we've seen this. In treatment in a study with sotigalimab and epo, we've been able to enhance what we believe is the clinical activity against anti-PD-1 refractory melanoma—and I'll describe those findings in a moment.

If one treats patients first with a tumoricidal agent that kills the tumor cells, releases novel antigens for immunogenic tumor cell death and then feeds that essentially to dendritic cells, one might expect that when you combine that with sotigalimab you might actually stimulate new and potentially effective antitumor responses, and we've seen this. In the combination with chemotherapy and radiation or through the introduction of new antigens by vaccines, we believe we observed greater immunity and activity.

Now, fortunately sotigalimab has been reasonably well tolerated and we have not seen any observed—not observed—any additive or new toxicities when we combine it with other agents. So, potentially, this combinability allows this agent to become a backbone of multiple different agents in the future, and that's what we're hoping and exploring.

Can I have the next slide, please?

These are indications that I will be focusing on today, which we will have further readouts this year. We have completed a study in PD-1 blockade refractory melanoma patients which we plan to discuss with the FDA later this year to discuss possible registration pathways, and I'll describe the findings in just a moment.

We have an ongoing study of sotigalimab in combination with neoadjuvant chemoradiation in the treatment of resectable esophageal/GEJ cancers. We plan to update that data as it becomes more mature later this year, and I'll give you the interim data in just a moment.

We also have an investigator sponsored trial in soft tissue sarcomas in combination with standard of care doxorubicin. I'll show you the interim data in a moment, but the study is ongoing and we hope to update with more mature data later this year.

Can I have the next slide, please?

This is our study in PD-1 blockade refractory melanoma. As you may know, in metastatic melanoma anti-PD-1 therapy, either alone or in combination with anti-CTLA-4 or anti-LAG-3, has really become the primary therapy for these patients. However, if you fail it or become resistant to the anti-PD-1 therapy, there are really no current, truly effective treatment options. This refractory group represents an unmet medical need.

Now, we're encouraged to test the combination of sotigalimab with an anti-PD-1 in this patient population because preclinical data has demonstrated that there is an additive tumor or synergistic antitumor effect when you combine the two.

Also, very important, in preclinical studies, we have shown that this combination can actually overcome resistance and be effective in tumors that were refractory and resistant to treatment with an anti-PD-1.

Now, as mentioned earlier, sotigalimab has demonstrated single-agent activity in melanoma which also has achieved two durable complete responses, and this also encourages us that we might be able to see such an effect against this particular cancer indication.

Now, in this study we have examined the combination of sotigalimab and nivo in patients who were refractory to PD-1. What that means is these patients had progression while on a PD-1 therapy. They had their progression of disease confirmed at least four weeks later to rule out pseudoprogression, which is an inflammatory response which can mimic progression.

So, in these truly refractory patients, one would not expect them to respond again to an anti-PD-1. Nevertheless, we have shown, on the spider plot and on the tables on the right, we have observed clinical activity with the combination therapy, with an overall response rate of 15.2% and a stable disease response rate of 30%.

Now, of importance is that these patients have had very long durations of response, and this is shown graphically in the spider plot. The median duration of response for the PR patients has not been reached. Four out of five patients at the time of the study close had ongoing responses lasting upwards of 25 months. That's at the study close. In fact, we know that several of these patients after this study have continued to be in unmaintained responses as per discussions with their investigator.

By the way, the fifth patient with a PR was in remission for 18 months before developing an isolated brain lesion. No systemic disease. This person had radiation to the brain lesion and has done well without requiring any systemic therapy.

Now, patients with stable disease have also had clinical benefit by remaining in stable disease for more than 14 months at the end of the study. It's also important to note that some of these patients completed therapy after a year or more of combination therapy. They felt well so their PIs took them off study and these patients in some cases remained untreated for more than 16 months by the end of the trial, which is really a clinical benefit because they did not have to be on continuous therapy. They had a drug holiday and they remain in remission, so very durable responses.

We believe our agent has clearly demonstrated clinical activity and has been of clinical benefit to our patients. We're planning to discuss our findings with the FDA later this year in order to help inform what a potential registration trial might look like.

Can I have the next slide, please?

This is one of our patients, treated with sotiga-nivo. A 54-year-old patient with mucosal melanoma who was initially treated with surgical resections and radiation, but then developed systemic disease. The patient was then treated with ipi/nivo and received three cycles of therapy. But unfortunately, as many patients do, they developed toxicity to the ipi and that was removed and the patient was then treated with nivolumab alone. The patient had stable disease for about 10 months on therapy but then developed this rapid progression of multiple sites including the abdomen, the liver and including a thoracic lesion which had to be irradiated at the beginning of therapy.

Now, these CT scans of the abdomen showing you the baseline on the left, the circles represent tumor lesions that have been highlighted for you. You can see the person had multiple liver lesions. In fact, if you were to do cuts throughout the liver you would see many more. They also had this very large lesion in the abdomen, the mesenteric lesion. Now, just two months after treatment with the combination these lesions had nearly resolved, dramatically reduced, and the patient went on to receive 15 cycles of therapy for 11 months and had nearly resolved all the target lesions, with only very small non-target lesions remaining. The patient was taken off sotiga because they were doing well and feeling well and has remained in the unmaintained response since that time with that additional therapy.

We believe that these patients have had clinical benefit, not only in a response but also the duration of response after completing active therapy.

Next slide, please.

Our next study that I'd like to describe is one which we have combined sotigalimab with standard of care neoadjuvant chemotherapy in the treatment of patients with GEJ and esophageal cancers. In these patients with resectable disease the standard of care is neoadjuvant chemotherapy plus radiation before surgical resection. In the left panel I'm showing you historical data. These are patients treated with standard therapy and as shown in the green curve, if one obtains a pathologic complete response, that means that they have no tumor remaining in the surgical specimen, that patient tends to have a longer survival than those patients who have persistent disease remaining. Therefore, obtaining a pathologic CR, or a pCR, may be a surrogate for survival.

Now, the goal of this study was to examine the combination of sotigalimab with this neoadjuvant chemoradiation in patients with resectable esophageal and GEJ cancers with the goal of achieving a higher PR rate. Enrolment of the study was completed at the end of last year, and some patients have not yet reached the end points and follow-up, so it is ongoing.

At the time of this data cut a few months ago, 22 patients had completed all therapy and were evaluable post surgery. Overall, as shown in the bottom table, our pathologic complete response rate was 41% and the PR rate was 50%, or an ORR of 91%.

Now, it's important to realize that response rates vary per the histology of the cancer. Adenocarcinomas do not do as well as squamous cell carcinoma with this type of treatment, and in the United States and Western countries, adenocarcinoma is the most common, much more common than squamous cell, with the prevalence about 80% of adeno versus squamous. In Asian countries such as China, the incidences actually float and squamous cell carcinoma is much, much more predominant. In fact, it's considered a bit of an epidemic in certain parts of Asia.

Now, the standard of care radiotherapy, chemoradiotherapy reported response is shown in the top table for adenocarcinoma in the range of 19% to 23%, and for squamous cell carcinoma 42% to 49%. Thus far, we've achieved pathologic CR rates of 35% for adenocarcinoma and 60% for squamous cell carcinoma. We're very encouraged by this data because it's tracking well compared to historical data. We are finishing off the study now. We hope to expect—we expect to update this data with all patients in the second half of this year.

Could I have the next slide, please?

We also have an interesting study in soft tissue sarcomas. This is an investigator sponsored trial that was started at Columbia University and it is now at a few other centers. Soft tissue sarcomas are a heterogenous group of malignancies made up of many different histologic types. Over the past several decades there have been a few advances in treatment of these malignancies, but the new treatments have not really offered significant improvements in survival. The mainstay treatment for these disorders remains chemotherapy, and in fact single agent doxorubicin, which typically has a response rate of anywhere from 14% to 18%, with PFS reported in the range of 4% to 7%. Unfortunately, you can't use doxorubicin continuously because it does have a cumulative cardiac toxicity.

In this study, sotigalimab was combined with the standard, a dose of doxorubicin, and given for up to eight cycles. This was then followed by sotigalimab alone. At the time of this data cut, 20 patients have enrolled and were evaluable, and thus far the response rate has been 20% with 60% stable disease.

Now, the important thing that we're following are the durations of responses. You can see that they've been quite long in some cases. PRs have lasted upwards of 11 months and stable disease has lasted up to two years. We expect that this trial will complete enrollment in the next few months and we hope to be able to discuss additional data in the later part of this year. Next slide.

To summarize our clinical program, we believe sotigalimab is possibly a best-in-class CD40 agonist antibody and, if approved, has the potential of being first-in-class. We believe that there may be broad applicability of this agent across multiple different cancers. We've shown that it has single agent activity and appears to have added or synergistic effects with I-O agents as well as standard of care chemotherapy treatments. It has had a reasonable safety profile and we have not seen or observed additive or synergistic toxicity. Therefore, we believe it has the potential to be clinically beneficial in multiple different cancer indications and could become a backbone treatment with many other therapeutic regimens.

In addition, we continue to explore new indications including the combination of sotigalimab with other I-O agents, which we believe may have additional potential.

We expect to have a readout of Phase 2 data updated for the esophageal and sarcoma studies later this year. And we expect to have a meeting with the FDA later this year to determine a potential registration path forward for the PD-1 blockade refractory melanoma.

With that, I'll turn it back over to Xiaodong.

Xiaodong Yang

Thanks, Frank. Now, let me introduce our next IND candidate, APX601. Next slide, please.

In addition to sotigalimab we are also very excited about our APX601 program because it addresses this very important question in immuno-oncology or immunotherapy, which is how can we reverse immunosuppression in the tumor microenvironment and unleash immune response against cancer?

It turns out TNF receptor 2 is selectively expressed in the tumor-infiltrating T regulatory cells and MDSC. The mechanism of APX601, which is a humanized IgG1 antibody against TNF receptor 2 are twofold. Number one is APX601 combines to Tregs and MDSCs in the tumor microenvironment to deplete the cells through antibody-mediated effector functions. It can also block the receptor, block TNF signaling to reverse the immunosuppressive phenotype.

Secondly, many epithelial cancer cells also express TNFR2 and use TNFR2 as an oncogene to drive up proliferation and growth, therefore blocking TNF receptor 2 on tumor cells can block tumor cell growth; can also mediate tumor cell killing through antibody-mediated effector functions.

We have very nice preclinical proof-of-concept data shown in the next slide. Next slide, please.

On Slide 22, we show two pieces of in vivo data that validate APX601 as a prominent antitumor immuno-oncology agent. On the left-hand side this shows the single agent activity of APX601 in a very aggressive mouse colorectal cancer model CT26. As you can see, we show beautiful dose-dependent antitumor activity for APX601 as a single agent.

On the right-hand side, we show a synergistic antitumor activity when combining APX601 in anti-PD-1 antibody treatment. The reason we did this experiment with anti-PD-1 is because there is increasing evidence showing that one of the key mechanisms contributing to PD-1 and PD-L-1 resistance is the increase of TNFR2 expressing T regulatory cells in the tumor microenvironment. Therefore, it makes sense to target these immunosuppressor cells with our APX601 in Phase 1b or Phase 2 trials down the road.

Next slide.

As I mentioned earlier, our pipeline and our partnerships are built based on our APXiMAB platform. What is the APXiMAB platform? This is shown on the next slide, please.

The APXiMAB platform is a rabbit based antibody technology. We are the only company that has fusion cell line hybridomas. Also, we have our own proprietary MLG humanization technology, which allows us to humanize a rabbit's antibody without losing affinity.

The key attributes for the technology are that we harness the power of a very interesting gene conversion mechanism which occurs in rabbits and chickens to increase the antibody affinity and broaden the antibody diversity. These two attributes—high affinity and the diverse repertoire, really allow us to select, engineer and build the best-in-class therapeutic antibody. We are the leader in developing therapeutic antibodies from rabbits. We have six molecules in our clinical development program and one approved product on the market.

I think now I'm going to hand it over to Francis to introduce to you the next agenda.

Francis Sarena

Thank you, Xiaodong.

I'll now review our plan to become a public company and upcoming milestones before opening the call up to questions. Next slide, please.

In March we announced that we had entered into a business combination agreement with Brookline Capital Acquisition Corp., BCAC, which is a Nasdaq listed special purpose acquisition company, or SPAC, with $51 million in trust.

BCAC was formed by Brookline Capital Markets, a boutique healthcare investment bank, for the purpose of combining with a life sciences company, like Apexigen. BCAC's team and sponsor bring a deep understanding and knowledge of the healthcare sector which they leveraged to search for companies with which to combine. Next slide, please.

Our transaction to become a public company has three key parts. The first part is the SPAC merger with BCAC, which I just mentioned. The second part is that we have secured a $15 million committed PIPE transaction. And lastly, we have put in place a $50 million equity line with Lincoln Park. The SPAC merger values Apexigen at a pre-money, fully diluted, net equity basis of $205 million. We expect the combination with BCAC to close in July of this year.

Simultaneous with the combination, we will close the fully committed $15 million PIPE transaction. The PIPE includes the same 50% warrant coverage that was provided to the SPAC IPO investors and is priced at $10 per unit, comprised of one share and half a warrant. As noted, we have also secured a $50 million equity line with Lincoln Park.

We estimate that the gross proceeds from the SPAC merger and PIPE transaction will be $66 million comprised of the $15 million fully committed PIPE transaction and $51 million from BCAC's trust account, assuming no redemptions.

It's important to note that the potential $66 million in gross proceeds do not include any potential proceeds from the $50 million equity line with Lincoln Park, which will be available to us after the closing.

We will use the proceeds of these transactions to continue to advance our sotiga program through our ongoing Phase 2 trials and advance our APX601 program through an IND filing. Additional information regarding these planned transactions can be found in BCAC's Form S-4 Registration Statement and other filings that they have made with the SEC.

Next slide, please.

As we discussed, we have multiple milestones in our pipeline through the remainder of this year and into 2023. Recapping quickly, in our sotiga program, we look forward to meeting with the FDA in a Type C meeting mid this year to inform the design of a potential registration-enabling trial in melanoma.

In our esophageal and GEJ cancer trial, we plan to disclose PCR data from all enrolled patients in the second half of this year, and six-month duration of response data in 2023.

In our sarcoma trial, we plan to disclose updated response rate data in the second half of this year. We also are on track for a mid-year IND filing for our next pipeline candidate, APX601.

Next slide, please.

To recap, Apexigen is a leader in discovering and developing innovative therapeutic antibodies against cancer. As Xiaodong noted, our company was built on our validated APXiMAB antibody discovery platform, and from that platform we have built a proprietary pipeline of product candidates, the lead of which, sotiga, is advancing in multiple Phase 2 trials and towards a possible registration-enabling trial in melanoma.

We also have five validating partner relationships with licensees of product candidates that we discovered through our APXiMAB platform, one of which – Beovu – is approved and marketed in over 70 countries.

As we look forward, we have multiple near-term milestones through the remainder of this year and into 2023. This is an exciting time for Apexigen and we look forward to updating you on our progress.

We will now open up the call for questions, and as a reminder, you may submit questions at the bottom of the webcast player. Operator?

I guess I am the operator, actually.

Tara Sobierajski

Yes. Thank you, Francis. You can begin reading the questions now.

Francis Sarena

Yes. The first question, when you look at the—this one is directed to Frank. When you look at the melanoma program going forward, how will you look to define resistance/refractory to anti-PD-1?

Frank Hsu

That's a good question. What we are planning to do is the same sort of criteria that we designed for the Phase 2 study, that I just mentioned. Patients who have true refractory disease or people who have progressed while on an anti-PD-1 therapy and have had that confirmed at least four weeks later, to confirm that it's not pseudoprogression.

So, ideally patients must have received a certain minimum amount of treatment and as defined by the SITC group it should be at least three months and we believe that actually it's better longer than that because the incidence of pseudoprogression tends to happen within that first timepoint, and much, much fewer incidence much later, so that helps eliminate that as a confounding factor.

Francis Sarena

Thank you, Frank.

The next question is actually for Xiaodong. It says, through the pandemic mRNA researchers have gained more insight of this technology. Would you please share your topline thoughts comparing the Apexigen platform versus mRNA platforms for anticancer applications?

Xiaodong Yang

I think the mRNA approach, my understanding is that it is primarily targeting the upstream of any interest of your targets, and the therapeutic antibody program tends to try to block and neutralize the protein produced from the mRNA.

I think in terms of antibody platform, I think we have the potential to be very best-in-class therapeutic antibody, because as I mentioned earlier, we utilize the power of this gene conversion mechanism. It can really create high affinity, extremely high affinity. We can actually even further increase affinity and specificity to really make antibody binds to target that was associated.

Secondly, as I mentioned earlier, we are using this gene conversion mechanism to create a really large repertoire like small molecule libraries, almost in theory has unlimited repertoire, so you can—if you want to select best-in-class therapeutic antibody with very well defined design goals, I think the rabbit antibody platform is the way to go.

Francis Sarena

Great. Thank you, Xiaodong.

I'll take the next question. It's actually two questions that are semi-related so I will read both of them. When does the Street get visibility on the level of redemptions from the SPAC? Then the other related question is, do we have an estimate of the redemption rate of the trust?

I'll start with the second question first. We do not have an estimate of the redemption rate .We are certainly cognizant of past SPAC merger transactions and the level of redemptions in those transactions and are aware that they have had relatively high redemption rates. We are anticipating that could be a result in our transaction as well.

Going to the other question, when does the Street get visibility on the level of redemptions, those redemptions, if I recall correctly, need to be made within I think it's two business days of the closing. So, those will come in right at the end and we'll report those out in, I believe, the Super 8-K that gets filed shortly after the closing. That's when we'll have that information available.

The next question is for Frank. How is sotigalimab differentiated from other CD40 agonists in development?

Frank Hsu

Our antibody was, as you know, developed to have some certain specificities. It was rationally designed. Sotiga binds to the CD40 ligand binding site and that mimics—we believe it's the best mimicker of natural ligand delivering the natural activation signal through CD40. We're the only antibody that really binds to the binding site in trials.

We've also designed it to have Fc mutations that allow it to bind more avidly to certain receptors such as FcgIIbR. That allows it to crosslink the receptor which is an important part of the signaling cascade. But it also was mutated to prevent it from binding to other receptors such as FcgIIaR, which can actually induce ADCC. That's an important function to eliminate because once you bind to a dendritic cell and stimulate, you don't want to kill it.

Now, other companies have been binding outside the binding site, and we believe that in our tests that we've actually—and for many of them, not all of them of course, but many of them, we have a much more

high affinity binder. Also, as mentioned, already, it binds to the ligand bind sight. In addition, there are a couple of companies that have done Fc mutations to try to increase the crosslink without ADCC. We're not quite sure about some of those; some of those have reported toxicities and aren't being used intratumorally. Other ones have reported increased activity by using fucosylation and we're not quite sure how that affects their ADCC function.

I think there's some questions about some of the other ones, how effective they are compared to us, but we do believe that our rational design makes it a first-in-class type of molecule.

Francis Sarena

Great, thanks. The next question I think Xiaodong, you might be in the best position to answer this one. What are your thoughts on the potential to combine sotigalimab with APX601?

Xiaodong Yang

Very interesting question. I think one of the approaches we developed our pipeline with new molecules and new targets, is to find more validated by the complementary pathway to CD40. As you can see, we have our APX601 which is targeting primarily the immunosuppressive Treg and MDSCs, which is really complementary to activation of antigen-presenting cells. We believe it makes a lot of sense, especially, for instance, if we are talking about for patients who have become refractory/resistant to PD-1. I think we already have data to show in combination with nivolumab we can reverse the resistance to PD-1 in melanoma patients, but in the future we can actually, certainly combine APX601 with our sotigalimab in similar patient population, maybe in different indications which shows PD-1 refractory and shows the patients have increased TNFR2 expression in the tumor microenvironment. I think that's one of our rationales, is obviously it's a possibility we'll combine sotigalimab with our APX601 when we pass the Phase 1 dose escalation safety study.

Francis Sarena

Great, thank you. The next one is somewhat related; I'll direct it to Frank. Do you think sotigalimab has enough activity as a monotherapy or is it likely to be used in an I-O combination strategy?

Frank Hsu

We have certainly been very encouraged by the single agent activity. That really demonstrates this is an active drug and can potentially be active by itself. But at the same time, we do believe that it will be probably pursued and best when it's combined with other agents such as either a tumoricidal agent, chemotherapy radiation, or other I-O agents. We believe its greatest potential is in combination and that's where we're intending to pursue.

Francis Sarena

Great. Thanks, Frank. As a reminder, you may submit questions by clicking at the bottom of the webcast player and entering them there.

I'll address the next question, Frank, to you as well. How does the CD40 target compare to other immune stimulatory targets such as CD137?

Frank Hsu

That's an interesting question. There are many targets—many agents out there that act in a similar fashion. 4-1BB or CD137, as well as OX40 , IL2 and other agents tend to activate T cells. There is some effect on dendritic cells, but one of the primary things is to act on prime T cells that are sort of preactivated, and then help to expand them, increase their proliferation and activation.

Now, some of the studies have shown that you really are inducing sort of a generalized stimulation to T cells when you do that, and that's very different than what we're doing with CD40. That generalized stimulation may prove against you because you could have side effects, inflammatory responses and toxicity. In fact, of these CD137 antibodies, one has been discontinued. The other has only been listed as one study open, so I think it's too early to know, but I do believe that they have experienced some toxicities with those agents. We'll see in the future if some of them pan out, but at this point it's still kind of early.

But in contrast, CD40 agonist antibodies, act earlier. They act on the antigen presentation itself. As mentioned, we're really trying to stimulate or activate dendritic cells that present antigens, in which case you would be able to expand T cells and their repertoire.

The use of agents after that could be very important, but the first step is to really stimulate those T calls, new T cells hopefully, first. And since you're not expanding them necessarily using the same methodologies, you would have a different set of toxicities than you would with an agent like an IL2.

Francis Sarena

Great. Thank you. We have another question, Frank, for you. When you consider dosing questions overall for immune stimulation, where is the balance for overstimulation and AEs versus not enough and potential anergy?

Frank Hsu

Well, I think I'll start with the second part. When studies were first done with the vaccines, and one of the clear thoughts is that you can't stimulate against a tumor antigen because you're already anergic, you've had it, it's circulating all the time and when we worked on any type of vaccines that was a common question and idea that you couldn't overcome that.

It really probably has to do with the level of stimulation. What we found in those earlier studies with vaccines is that dendritic cells are the most potent way of vaccinating. If you can feed an antigen to the dendritic cell directly and you can activate them, become mature, they can overcome that. You can actually generate very potent immune responses.

Now, keeping those immune responses after that going requires restimulation and sometimes other effects such as checkpoint inhibitors in order to keep those ongoing. But the anergy, overcoming that, it's very complicated, but the least of it may be because they're not being stimulated in the most appropriate and most effective fashion, which can be done by fully activated antigen-presenting cells such as dendritic cells.

Francis Sarena

Great. Thanks, Frank. Our last question in the queue: you have multiple—Frank, this is probably for you as well. You have multiple ongoing Phase 2 trials. What are you looking to glean from these ongoing trials that could further de-risk the clinical development program and provide proof of mechanism?

Frank Hsu

That's a good question. We have obviously many studies ongoing. Some of them are sponsored, some of them are investigator sponsored. In order to de-risk the program there are a lot of investigator-sponsored studies which were done in order to look at indications which are not perhaps on the primary path, but every one that we've done are looking for signals that might be new methods or new ways of looking at the mechanism of action.

For instance, for sponsored trials, we've done single agent activity; we've shown that, and which is a validating effort for sotiga alone. We've done combinations in sponsoreds, as mentioned, with PD-1 as well as with chemotherapy and tumoricidal agents. But with ICs we have also looked at not only chemo approaches but we've also looked at in combination with vaccines. We have some interesting data that has been produced by some of our collaborators that indicate that the vaccine approach we can further stimulate those immune responses and perhaps make them even more potent.

We've also looked at intratumoral approaches. There was a recent presentation by one of our collaborators at AACR which shows that you can actually give it intratumorally and along with, in this case pembro, which can also further stimulate this kind of an approach for another way of vaccinating a person against their own tumor.

We're also exploring with other combinations with I-O agents in investigator-sponsored trials, and all of these approaches may help de-risk the program by looking at a whole variety of indications and combinations that we may not be able to do so by ourselves as a sponsored trial, but hopefully will provide insights into other opportunities and de-risk our program.

Francis Sarena

Great. Showing no further questions in the queue I'll turn the call back to Xiaodong to provide some closing remarks. Xiaodong?

Xiaodong Yang

Thanks Francis, thanks Frank for answering the questions.

I just want to thank you everyone for joining the presentation. With that, we will now adjourn our Apexigen Investor Day presentation. Thank you so much.

Additional Information and Where to Find It

In connection with the proposed business combination, Brookline Capital Acquisition Corp. ("BCAC") filed a registration statement on Form S-4 (the "Registration Statement") containing a preliminary proxy statement and preliminary prospectus of BCAC, and after the Registration Statement is declared effective, BCAC will mail a definitive proxy statement/prospectus relating to the proposed business combination to its stockholders. BCAC's and Apexigen, Inc.'s ("Apexigen") stockholders and other interested persons are advised to read the Registration Statement, including any amendments thereto and other documents filed with the Securities and Exchange Commission ("SEC") in connection with BCAC's solicitation of proxies for its special meeting of stockholders to be held to approve, among other things, the proposed business combination, because those materials contain important information about Apexigen, BCAC and the proposed business combination. When available, the definitive proxy statement/prospectus and other relevant materials will be mailed to BCAC stockholders as of a record date to be established for voting on the proposed business combination.

Stockholders may obtain a copy of the preliminary or definitive proxy statement/prospectus, once available, as well as other documents filed with the SEC by BCAC, without charge, at the SEC's website located at www.sec.gov or by directing a request to Patrick Sturgeon, Chief Financial Officer, Brookline Capital Acquisition Corp., 280 Park Avenue, Suite 43W, New York, New York 10017, or by telephone at (646) 603-6716, or by contacting Morrow Sodali LLC, BCAC's proxy solicitor, toll-free at (800) 662-5200.

Participants in the Solicitation

Apexigen, BCAC and their respective directors and executive officers and other persons may be deemed to be participants in the solicitations of proxies from BCAC stockholders in respect of the proposed business combination. Information regarding BCAC's directors and executive officers is available in its final prospectus filed with the SEC under Rule 424(b)(4) on January 29, 2021. Additional information regarding the participants in the proxy solicitation and a description of their direct and indirect interests is contained in the proxy statement/prospectus related to the proposed business combination, which was filed on a Form S-4 (File No. 333-264222) on April 11, 2022, and which can be obtained free of charge from the sources indicated above.

Forward-Looking Statements

This transcript includes forward-looking statements within the meaning of the "safe harbor" provisions of the United States Private Securities Litigation Reform Act of 1995. Forward looking statements may be identified by the use of words such as "estimate," "plan," "project," "forecast," "intend," "will," "expect," "anticipate," " believe," "seek," "target" or other similar expressions. All statements other than statements of historical fact contained in this transcript, including any statements with respect to the proposed business combination and other proposed transactions described herein, and future business plans of the Apexigen and BCAC management teams, including expectations regarding the potential benefits, activity, effectiveness and safety of Apexigen's product candidates; Apexigen's expectations with regard to the results of its clinical studies, preclinical studies and research and development programs; and Apexigen's preclinical, clinical and regulatory development plans for its product candidates, are forward-looking statements. These forward-looking statements speak only as of the date of this transcript and are subject to a number of risks, uncertainties, and assumptions, including, but not limited to: Apexigen's early stages of clinical drug development; Apexigen's ability to timely complete clinical trials for its product candidates; Apexigen's ability to demonstrate sufficient safety and efficacy of its product candidates in its clinical trials; changes in domestic and foreign business, market, financial, political and legal conditions; the inability of the parties to successfully or timely consummate the proposed business combination, including the risk that any required regulatory approvals are not obtained, are delayed or are subject to unanticipated conditions that could adversely affect the combined company or the expected benefits of the proposed business combination or that the approval of the stockholders of BCAC is not obtained; failure to realize the anticipated benefits of the proposed business combination; the amount of redemption requests made by BCAC's public stockholders; and the ability of BCAC or the combined company to issue equity or equity linked securities in connection with the proposed business combination or in the future. Additional factors that could cause actual results to differ are discussed under the heading "Risk Factors" and in other sections of BCAC's filings with the SEC and in BCAC's current and periodic reports filed or furnished from time to time with the SEC. This transcript concerns product candidates that are under clinical investigation, and which have not yet been approved for marketing by the U.S. Food and Drug Administration ("FDA"). Each product candidate is currently limited by federal law to investigational use, and no representation is made as to its safety or effectiveness for the purposes for which it is being investigated. In light of these risks, uncertainties and assumptions, these forward-looking events and circumstances are inherently uncertain and may not occur, and actual results could differ materially and adversely from those anticipated or implied in the forward-looking statements. Accordingly, you should not rely upon any forward-looking statements as predictions of future events. Neither BCAC, Apexigen nor any of their respective affiliates have any obligation to update or revise any forward-looking statements or this transcript, to conform any statements contained herein to actual results, or to make changes in their expectations. Although all information and opinions expressed in this transcript were obtained from sources believed to be reliable and in good faith, no representation or warranty, express or implied, is made as to its accuracy or completeness. This transcript contains preliminary information only, is subject to change at any time and is not, and should not be assumed to be, complete or to constitute all the information necessary to adequately make an informed decision regarding your engagement with BCAC and Apexigen.

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