Corbus Pharmaceuticals Holdings, Inc.
Update Conference Call
June 1, 2021

Operator
Hello, and welcome to the Corbus Pharmaceuticals conference call. As a reminder, all participants are in listen only mode. And if anyone should require operator assistance during the conference, please press "*0" on your telephone keypad. A question-and-answer session will follow the formal presentation. This conference is being recorded at the company's request and will be available at the company's website approximately two hours following the end of the call. I would now like to call to your host, Ted Jenkins, Senior Director, Investor Relations and Corporate Communications. Please go ahead, sir.

Theodore Jenkins
Thank you, operator. Good morning, everyone. At this time, I'd like to remind our listeners that remarks made during this call may state management's intentions, hopes, beliefs, expectations, or projections of the future. These are forward-looking statements involve risks and uncertainties. Forward-looking statements on this call are made pursuant to the Safe Harbor provisions of the federal securities laws. These forward-looking statements are based on Corbus' current expectations and actual results could differ materially. As a result, you should not place undue reliance on any forward-looking statements.

Some of the factors that could cause actual results to differ materially from those contemplated by such forward looking statements are discussed in the periodic reports, corporate files with Securities and Exchange Commission. These documents are available in the investor section, the company's website and on the Securities Exchange Commission's website and we encourage you to review these documents carefully.

Joining me on the call today are Dr. Yuval Cohen, our Chief Executive Officer, Dr. Barbara White, our Chief Medical Officer and Head of Research, Sean Moran, our Chief Financial Officer, and Craig Millian, our Chief Commercial Officer. With that is my pleasure to turn the call over to Yuval.

Yuval Cohen
Thank you, Ted. It's my pleasure to welcome everyone to today's call. And thank you for joining us on such short notice. As you know, we have been emphasizing our interest in expanding our pipeline through a series of bolt-on acquisitions. The strategy is straightforward. Leverage our expertise in immunology and our experience in drug development to build a diverse pipeline that mitigates risk by using multiple modalities to focus on inflammation, fibrosis, metabolism, and immuno-oncology.

Today, we announce our first transaction, expanding our pipeline by adding two novel, anti-integrin monoclonal antibodies that we intend to develop for immuno-oncology and fibrosis. One from University of California San Francisco, and the other from Panorama Research Incorporated. As Barbara will detail shortly, the cytokine TGF beta and its role

in promoting inflammation, fibrosis, and cancer has been the focus of extensive academic and industry research, giving rise to testing different approaches to its inhibition.
One recent approach is to target specific cell surface integrins that activate the TGF beta, and we joined others who are pursuing this approach. A number of external clinical studies are underway exploring this mechanism of action in both solid tumors and fibrotic diseases. And there has been significant recent business development activity in integrin targeting assets. The two antibodies we've acquired have properties we find attractive, and we believe position them favorably in the field. CRB-601 is highly specific for the integrin alpha V beta 8 license from UCSF and invented in the lab of Dr. Stephen Nishimura.

As Barbara will review CRB-601 has generated preclinical data related to binding and functional activity, showing high potency. We believe CRB-601 could offer advantages over competitor approaches and expect to initiate phase one clinical studies next year.

CRB-602 has dual specificity for alpha V beta 6 and alpha V beta 8 integrins and is licensed from TRI. We think targeting both of these integrins may be particularly helpful in targeting fibrotic diseases. Currently, anti-alpha V beta 6 therapeutics are being developed for IPS and PSC. We also plan to start clinical studies with CRB-602 next year.

These two new programs fit neatly into our pipeline alongside our existing programs, which focus on the endocannabinoid system. They align with and extend the disease areas we are pursuing and will benefit from our expertise in drug development. Lastly, we reported $125 million in cash as of March 31st, which we expect to fund us through the first quarter of 2024. So, we are well capitalized to advance our expanded pipeline to several meaningful clinical milestones.

I would now like to turn the call over to our chief medical officer and head of research, Dr. Barbara White, to provide us with details of our program expansion plan and the news we announced today. Thank you, Barbara.

Barbara White
Thank you, Yuval. We are delighted to have licensed two monoclonal antibodies, or MABs, that bind to integrins and block integrin mediated activation of transforming growth factor beta, or TGF beta. TGF beta needs to be activated from its latent form to exert its biologic activities. Active TGF beta is a multifunctional cytokine involved in many cellular processes, including cell growth and differentiation, immune responses, wound healing, and tissue repair. Our new MABs will be developed as treatments for solid tumors and fibrotic diseases.

CRB-601 was designed in Dr. Stephen Nishimura's lab at UCSF and binds to the integrin alpha V beta 8 with high affinity with IC 50 for inhibiting activation of TGF beta in the picomolar range. Integrins are membrane bound, alpha beta heterodimers that mediate interactions between cells and between cells and extracellular matrix. Integrins are involved in many body processes, including cell growth, differentiation, survival, malignancy, fibrosis, immune responses, wound healing, and angiogenesis.
The alpha V integrins including alpha V beta 6 and alpha V beta 8, activate TGF beta. Some human epithelial malignancies, or carcinomas, express alpha V beta 8 themselves, which activates latent TGF beta in their microenvironment. Active TGF beta suppresses immune responses that control growth and metastases of established tumors. It converts non-regulatory CD4 positive T-cells into regulatory T-cells, which might be considered checkpoint cells because they suppress activity of other activated immune cells and turn off anti-tumor immune responses.

Activation of TGF beta leads to both reduction in CDA T-cell numbers and killing of tumor cells. TGF beta has other effects beyond the immune system that promote tumor growth and metastases. Importantly, overexpression of alpha V beta 8 by tumor cells and expression of TGF beta in tumors have both been linked to poor clinical outcomes. Thus, we and others believe that inhibiting TGF beta activation may provide efficacy and treatment of cancers.

CRB-601 is a higher affinity humanized version of an anti-alpha V beta 8 monoclonal antibody called C-64, also developed by Dr. Nishimura's lab, which has been found to be active in animal cancer models. In the MC 38 colon carcinoma tumor model, C-64 reduced tumor volume at day six by nearly 50% as a single agent, and nearly eliminated tumor growth when combined with anti PD-1 monoclonal antibody. Complete response rates in the MC 38 model were 10% for C-64, 40% for anti PD-1, and 60% when the two agents were combined. We look forward to sharing with you data specific to CRB-601 after they are made public by Dr. Nishimura's lab.

We believe potential advantages of CRB-601 are the following: it inhibits TGF beta from ever becoming activated, it is very potent, and it has been specifically designed to inhibit activation of TGF beta, not only in a form that is released from its association with the TGF beta latency associated peptide, but also in a newly described form of active TGF beta that remains associated with the TGF beta latency associated peptide.

This latter form of active TGF beta was recently described by Dr. Nishimura and colleagues and is thought to be the predominant form of TGF beta activated by alpha V beta 8 on tumors. We will develop CRB 601 for the treatment of solid tumors in combination with standard treatments, including checkpoint inhibitors. We expect to be in the clinic in 2022.

We've also announced today that we have acquired CRB-602 from Dr. Jim Larrick, and his team at Panorama Research Inc. CRB-602 is the MAB that binds both alpha V beta 6 and alpha V beta 8 to inhibit their activation of TGF beta. The alpha V beta 6 integrin is expressed by epithelial cells during wound healing and tumors of epithelial cell origin. Overexpression of alpha V beta 6 has been linked to fibrosis in idiopathic pulmonary fibrosis. We expect that CRB-602 may provide potential therapeutic benefit in both fibrotic diseases and cancer. We also expect to complete IND enabling studies and manufacturing in 2022. With that, I'll turn the call back over to Yuval.

Yuval Cohen
Thank you, Barbara. Under the combined terms of the two exclusive licensing agreements, Corbus will pay $2 million upfront and will make potential development and sales milestone payments totaling up to $206 million and pay low single digit royalties on sales. To close, I want to reiterate how excited we are about our expanding pipeline, and also mention that we have posted a new investor presentation on our updated website that details the data and upcoming expected milestones we refer to on today's call.

Given our skillset of understanding global complex clinical programs and our expertise in immunology along with a strong cash position, we believe we have a significant opportunity to advance novel therapeutics that will have a meaningful impact on patient care. I would like to thank you all for your time and attention. I will now turn the call back over to the operator to open the call for questions from the audience.

Operator
Thank you. We'll now be conducting a question-and-answer session. If you'd like to be placed in the question queue, please press "*1" on your telephone keypad. A confirmation tone will indicate your line is in the question queue, you may press "*2" if you'd like to remove your question from the queue.

For participants using speaker equipment, it may be necessary to pick up the handset before pressing "*1." One moment please while we poll for questions. Our first question today is coming from Brian Abrahams from RBC Capital Markets. Your line is now live.

Brian Abrahams
Hey guys, good morning. Congrats on the transaction, and thanks very much for taking my questions. I guess maybe starting off for 601, Barbara, I was wondering if you could expand a little bit on how the high potency and the differential interactions with LAP and the TGF beta pathway might create ultimately a differentiated mechanism and clinical profile versus other alpha V beta 8's development.

Barbara White
Sure. Thank you, Brian, for the question. And the work is a very recent work from Dr. Nishimura's lab and was published in 2020, in which they use some very sophisticated structural analysis and propose that in the context of tumors where alpha V beta 8 is expressed on the tumor and TGF beta may be expressed, for example, on a T regulatory cell, that the activation, which means the ability to exert this biologic effect, of TGF beta occurs without TGF beta actually being physically released from the cage that it's in, which keeps it inactive.
But it remains in close proximity, and that it nonetheless can activate TGF beta on a range of T cells and cause T regulatory cells to exert their activity and induce immunosuppression in tumors. And in this actual physically constrained environment, it would be somewhat difficult to target the TGF beta with, for example, an anti TGF beta monoclonal antibody. And the antibody that they've developed is very specifically designed to have high potency to inhibit this type of activation of TGF beta. So, it's simply that, very high potency inhibits the activation of TGF beta that remains associated with its cage, which is thought, by their lab at least, to be the predominant form involved in activation of TGF beta in a tumor microenvironment.

Brian Abrahams
Got it. That's really helpful. And then, maybe for 602, can you talk about some of the preclinical work that you might do and some of the preclinical models you'll look at that might best discern the potential differentiation of the dual beta 6, beta 8 interaction and where that might be best developed?

Barbara White
Sure. We think that the combination of targeting beta 6 and beta 8 may be particularly important in fibrotic diseases or in cancers of epithelial cell origin where both are expressed. We think that what we're looking for in this antibody will, again, be high potency. We think that's obviously reasonably important. And we will be looking for its ability to have activity and models of both fibrotic disease and tumor. You know, we'll certainly be looking in the tumor models for its ability to augment effects of standard therapies such as checkpoint inhibitors. And we'll be looking to see how it compares to similar antibodies that are being developed in the space.

Brian Abrahams
One more quick one, if I could squeeze it in. Just sort of, I guess, bigger picture. Can you talk about what this means for additional business development plans? You did mention you have a balance sheet that will take you out several more years. So, I guess, how are you thinking about additional BD, potentially for later stage assets and your appetite

there? And then, just your overall, I guess, implications for your overall views and strategy with respect to lenabasum and the upcoming read out there? Thanks.

Yuval Cohen
Thanks, Brian. The strategy is as follows. We are looking for assets that are in immunology that we can leverage our expertise. These two assets, we think fit neatly with what we've already have, which is the endocannabinoid system. Together, they're really focused on these four areas, inflammation, fibrosis, immuno-oncology, and then through the CD one work on metabolism. Having said that, we're very, very keen on additional bolt-on acquisitions. And we're particularly interested in one that would be in the clinic or ready for the clinic.

But again, this is sort of the filter that we're looking at these opportunities through. It's unlikely, for example, Brian, to use a sort of an extreme example, I can't see Corbus bringing in anything around sort of gene therapy. It just doesn't fit who we are and what we do. But expect to look for additional acquisitions.

And one of the guiding principles there, as well, to think about is, if you think about our pipeline as it stands now, we have programs that are, we think, have a high probability of success because they're following a validated target. And we have programs where we will be the first to use that mechanism of action. We'd like to continue having that mix, so expect to see more of that.

Brian Abrahams
Great, thanks so much.

Operator
Thank you. Next question today is coming from Maury Raycroft from Jefferies. Your line is now live.

Maury Raycroft
Hi, good morning, everyone, and congrats on the update today. Question I was going to ask is just whether for both antibodies, do you have rights to go beyond 601 and 602 to next gen molecules, and do you know for sure that 601 and 602 are going to be the candidates that you eventually move into the clinic?

Barbara White
Do you know, Maury, I wonder if you could repeat that question again. It dropped off. At least, I didn't hear it at the end.

Maury Raycroft
Sure. For 601 and 602, I was just wondering if you have rights that go beyond those two candidates to the next gen molecules from either the lab or from the company? And are these the candidates that you're going to move into the clinic?

Barbara White
Thanks. The 601 MAB is actually probably third generation to speak of out of Dr. Nishimura's lab. And we do intend to take it into the clinic. And we do, as part of the licensing deal, have access to patents that cover a number of other monoclonal antibodies that could be related. But it is the one that we plan to take into the clinic. We really are very happy with its properties.

But let me say, absent a few minor tweaks that may happen, but it is basically the antibody we're taken into the clinic. For 602, the arrangement that we have with Panoramic Research Inc., is that they have a variety of anti-integrin antibodies, and that we will work with them to develop a number of them. This is the first of them. We're most interested in starting with this alpha V beta 6 beta 8 combo. And again, we are working with them to optimize this particular antibody. But in essence, it's also the one that we will be taking into the clinic.

Maury Raycroft
Got it. That's helpful. And also, just wondering if you can talk more about how you sourced both of these assets in the deals? And was it a competitive process?

Yuval Cohen
So, Maury, we have been actively looking as we've been saying, I think, pretty much since the end of last year, we're fortunate to have a very, very good BD team. And both of these were assets that we approached. And I'd say that, you know, it's certainly competitive in terms of the field. We've seen a lot of business development around integrins in general and including alpha V beta 8 integrins. I think that our timing was just especially good, and we picked them at a time where the counterparties were very open to this transaction.

Maury Raycroft
Got it. And maybe last quick question just on 601 and the TGF beta tumor signature that you could potentially look for. I guess, first for competitors, are there any updates expected from competitor programs that can help inform Corbus? And do you have a strategy for what an initial clinical study could look like to potentially select patients based on biomarkers?

Barbara White
Craig, do you want to take the first?

Craig Millian
Sure. Maury, can you just repeat the question? I temporarily dropped off the call.

Maury Raycroft
Sure, just wondering if there are competitor updates on the horizon that could inform your strategy that you can talk about and then yeah, so that was the first part of the question.

Craig Millian
Sure. Right. Yeah, there's a number of competitive integrins in very early development. I think, you know, some have reported some preclinical data. Specifically, Morphic had some early data that they presented at AACR, which was interesting. So that's a program that's a small molecule program that is slightly ahead of us, but I think some of the learnings there could be leveraged.

Most interesting is Pfizer has a phase one integrin program for their alpha VB8 in solid tumor. That's an antibody, and they haven't, as far as I know, given specifics in terms of when they'll have data, but that's one we're paying close attention to as well. And beyond that, I'd say those are probably the two that are slightly ahead of us, but that we're anticipating, you know, additional data readout on the alpha V beta 8 front.

Barbara White
Maury, this is Barbara. Then to follow up, we definitely plan to take advantage of learnings from those in front of us. We think that's a really good thing about what we're doing is that, in these particular instances, we will have that advantage, that opportunity to learn from the work that's done by others in front of us. But as we think come along with a really great MAB close behind.

We think, we believe, we plan to develop biomarkers, tools, to identify those patients who are most likely to benefit from the therapy. We think that will be necessary. We have a number of approaches that we are considering. And again, we will certainly learn from the work that others are doing. We won't be working in isolation. So, we plan to do that.

In terms of the design of the phase one, we anticipate it will be a fairly straightforward phase one design oncology started with some tumors in which the alpha V beta 8 is expressed, look for early reads, expand. So, sort of the basket philosophy for the early phase one studies. But at the same time, we will be evaluating certain potential biomarkers for further development into diagnostics.

Maury Raycroft
Got it. That's all very helpful. Thanks. Thanks for taking my questions.

Operator
Thank you, as a reminder that's "*1" to be placed in the question queue. Our next question is coming from Leland Gershell from Oppenheimer. Your line is now live.

Leland Gershell
Good morning. Thanks for taking my question. And thank you for the BD update. Just a couple of broad questions maybe for Barbara. Just with respect to the potential for 601, if you have a sense of any particular solid tumor types that may be responsive to this strategy of targeting alpha V beta 8. And furthermore, if you see a role for 601 in being able to address certain tumors that may be refractory to checkpoint inhibitors. And I have a follow up. Thank you.

Barbara White
Thank you. To start, we don't expect that this therapy will be used as sort of as a monotherapy. We certainly expect as is in the case in most of oncology treatments, that will be used in combination, and we think it will be especially helpful and useful in combination with checkpoint inhibitors. So that will be an early part of its development.

In terms of which particular tumors, there is some work that has been done to look at alpha V beta expression by tumor. So, we think it should be tumors that have been reported to express alpha V beta 8 and tumors in which we think there's some evidence that activation of TGF beta has played a role. Tumors in which perhaps don't have a rich, immune inflammatory infiltrate associated with them, those that may have it, those that are more excluded, or those that are actually a desert, sort of, for immune responses.

So that there are a couple of things we'll need to look at when we select the tumors, probably not only tumor type, but what is the immune response that's going on in that particular tumor. But there are a number that popped to mind upfront, things such as colon cancer or myeloma. But again, there are a number of others. And we will look more broadly, initially have several tumor types that we look at initially in phase one, before we focus down in.

Leland Gershell
Thanks. And then in the press release you'd mentioned activity and syngeneic tumor models. Just wondering if in the interim between now and, perhaps, the phase one we'll be seeing an easy to grasp based models and data they're from.

Barbara White
Yes, we would you should expect to see that.

Leland Gershell
All right. Terrific. Thanks very much.

Operator
Thank you. We've reached the end of our question-and-answer session. And ladies and gentlemen, that does conclude today's teleconference and webcast. You may disconnect your lines at this time and have a wonderful day. We thank you for your participation today.