Corbus Pharmaceuticals Holdings, Inc.
Update Conference Call and Webcast
September 20, 2018

Operator: Greetings, and welcome to Corbus Update Conference Call and Webcast. At this time, all participants are in a listen-only mode. A question and answer session will follow the formal presentation. If anyone should require operator assistance during the conference, please press Star 0 on your telephone keypad.

As a reminder, this conference is being recorded. I would now like to turn the conference over to your host, Ted Jenkins, Senior Director of Investor Relations and Corporate Communications. Thank you, you may begin.

Ted Jenkins: Thank you, Sherry, and thank you all for joining us. As a reminder, a copy of the press release we issued this morning, announcing that Corbus has licensed the rights to develop, manufacture, and market drug candidates from over 600 novel compounds, targeting the endocannabinoid system from Jenrin Discovery, is available on the Investor section of the Corbus website. We have also posted a new investor presentation on our website. We'll be referring to select slides from this presentation on this call.

Before we begin, I'd like to remind listeners that remarks made during today's call will include forward-looking statements and may state management's intentions, hopes, beliefs, expectations, or projections of the future. These are forward-looking statements that involve risks and uncertainties, and 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 statement. 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 Corbus files with the Securities and Exchange Commission. These documents are available in the Investor section of the Company's website and the Securities Exchange Commission's website. We encourage you to review these documents carefully.

On the call today are Dr. Yuval Cohen, Chief Executive Officer, and Dr. Barbara White, Chief Medical Officer, and Dr. George Kunos, Scientific Director of the National Institute on Alcohol Abuse and Alcoholism, a component of NIH. With that, I'm pleased to turn the call over to Yuval.

Dr. Yuval Cohen: Thank you, Ted. Good morning everyone, and thank you for joining us on such a short notice. This is an exciting day for Corbus. As you know, we only have these types of calls on occasions when we have something meaningful to share, and today certainly falls into that category.

As Ted mentioned, this morning we announced that we have licensed the exclusive worldwide rights to manufacture, develop, and market drug candidates from a portfolio of more than 600

unique compounds, targeting the endocannabinoid system from Jenrin Discovery, LLC. One of the most exciting drug candidates in this portfolio is CRB-4001, a second generation peripherally restricted CB1 inverse agonist, targeting fibrotic liver, lung, heart, and kidney diseases. And, you'll hear much more about that later in this presentation.

As highlighted on Slides 5 and 6 in our new deck, our focus on the endocannabinoid system is backed by an ever-expanding body of research and robust underlying science, based on the ECS as a master regulator of inflammation and fibrosis in the body. This research demonstrates that targeting of the endocannabinoid G-protein coupled receptors, or GPCRs, CB1 and CB2, in the immune system, as well as in some key organs, reduces inflammation and inhibits or halts fibrosis with applicability across a range of potential indications and diseases. This is a key differentiator for Corbus as you look at the broader competitive landscape. And, we'll talk about that more in a second.

As many of you know, our lead endocannabinoid system targeting drug candidate, lenabasum, targets rare inflammatory and fibrotic diseases. And, by adding the Jenrin assets to our portfolio, we can now target new indications with a clearly differentiated, yet complementary mechanism of action. As you can see on Slide 7, this transaction meaningfully expands our market opportunities and positions us to become a leader in inflammatory and fibrotic diseases by targeting the endocannabinoid system with what we believe is one of the industry's most innovative pipelines.

Before I outline this exciting transaction and what it means for Corbus, I'd like to briefly review how far we've come over the past four years, since our inception. I think this will offer good perspectives on why today's announcement is so meaningful for us. In 2014, when we were founded, Corbus did not have a drug in the clinic, but we recognized the significant opportunity of ECS targeting drugs to treat inflammation of fibrosis, and we had a strategic plan and vision to go after it.

We began to pursue that opportunity with lenabasum, a truly pioneering drug candidate with a unique mechanism of action. As we have since demonstrated with lenabasum, rationally designed, small molecules can be successfully applied to target the ECS, and there are considerable advantages to doing so. Over the past four years, we've been working hard and we've moved lenabasum into the clinic in four important and rare indications.

Today marks another step forward in our journey to advance Corbus. With a Jenrin transaction, we now have what we believe is a leading pipeline of ECS targeting drug candidates in the industry with multiple late, as well as early stage programs. As you can see on Slide number 8, CRB-4001, like lenabasum, targets the endocannabinoid system, but does so with a differentiated mechanism of action that opens the door to additional indications that previously, we could not target.

While lenabasum is a CB2 agonist, targeting immune cells in rare inflammatory diseases, CRB-4001 targets the second endocannabinoid GPCR, CB1. And, rather than activating it, it inhibits its activity. In fact, it reverses its activity. CRB-4001 is differentiated from first generated CB1 reverse agonist, due to its reduced penetration of the blood brain barrier and CB1 receptor occupancy. We believe that this will allow it to avoid the side effect profile that previous generations of such, at that time, promising drugs encountered.

The inhibition of CB1, by reverse agonism, has been well documented to reduce inflammation and fibrosis in specific target organs, such as the kidney and the liver. Also, human polymorphisms of CB1 confirm this. I want to underscore here that CRB-4001 requires only limited near-term resources, so Corbus' cash position will not be materially impacted through the end of 2019.

Slide 9 outlines our expanded pipeline, with three late stage clinical studies for lenabasum, a Phase 2 lupus study and CBR-4001, which is expected to enter Phase 1 next year, followed by an NIH supported, first-in-patient, Phase 2 study. We are continuing, also, to target 2021 for the launch of lenabasum and a reminder, lenabasum is currently in Phase 3 studies, as well as other late stage studies.

Beyond lenabasum and CRB-4001, Corbus now has a portfolio of more than 600 unique compounds that we believe will support the advancement of one to two new drug candidates into clinical testing each year, starting in 2020. As you can see on Slide number 10, targeting multiple inflammatory indications with a single drug is a common and very successful model in the pharmaceutical industry. This is also a reminder of how each one of these drugs, commercial drugs, that comprise four of the top five drugs annually in sales, is at the end of its product lifecycle and that we, as an industry, have yet to replace them with equally successful drugs.

With that, let me turn back to the specifics of today's announcement. Jenrin is a privately held pharmaceutical company, developing a pipeline of proprietary, first-in-class small molecules, designed to selectively target the endocannabinoid receptors in peripheral tissues. Jenrin developed activities have primarily focused on potential therapeutics for chronic diseases, including diabetes, non-alcoholic fatty liver disease, NASH, and obesity.

CRB-4001 was developed in collaboration with, and in financial sponsorship of, the NIH. As you can see on Slide number 11, and later on Slide 37, CRB-4001 shows promise in multiple pre-clinical models. Potential indications include NAFLD, NASH, PBC, IPS, and others. On that topic, I'd like to turn over briefly to my colleague, Dr. Barbara White, to provide additional color on the potential of CRB-4001. Barbara.

Dr. Barbara White: Thank you, Yuval. We believe that CRB-4001 has the potential to help people who have or who are at risk of developing organ dysfunction or failure because of inflammation and fibrosis, including people with the diseases mentioned by Yuval. We're particularly excited by the potential of CRB-4001 to treat people with NASH. NASH is a chronic metabolic liver disease that affects between 2 to 5 percent of Americans. Fat builds up in the liver where it can cause inflammation and fibrosis. This leads--can lead to liver cirrhosis and cancer.

Blocking CB1 activation has the potential to limit this fat buildup, reduce inflammation, and improve fibrosis in the liver of people with NASH. There are a number of other possible indications. Among them, we believe CRB-4001 provides an opportunity to address radiation-induced pulmonary fibrosis. Radiation therapy is a part of the treatment regimen for many cancer patients today. Radiotherapy is well tolerated in most people, but it can damage the tissues that are radiated.

When people with lung cancer receive radiation, one side effect can be inflammation and fibrosis in the lungs. This can restrict the amount of radiotherapy that a patient can receive and even cause severe damage to the lungs. We believe that blocking CB1 with CRB-4001 has the potential to limit inflammation and fibrosis, caused by radiation treatment of lung cancer. Yuval, back to you.

Dr. Yuval Cohen: Thank you, Barbara. Turning to Slides 12 and 13, earlier this year, we engaged Health Advances to provide detailed, demographic and market opportunity research on our targeted indications for lenabasum. For the first time today, we are providing you with an insight into this data and look forward to giving you more and more clarity on that as we progress.

Lenabasum is currently in Phase 3 study for the treatment of the rare autoimmune disease, systemic sclerosis, otherwise known as scleroderma, which affects approximately 200,000 patients in the U.S., Europe, and Japan. It has an estimated annual market opportunity of up to $2.2 billion. A Phase 3 study for lenabasum, for the treatment of our second rare autoimmune disease, dermatomyositis, is planned to begin towards the end of this year.

Dermatomyositis affects approximately 80,000 patients in the U.S., Europe, and Japan, and represents an estimated annual market opportunity of up to $2 billion. Lenabasum is currently in a large Phase 2 study for pulmonary exacerbations in cystic fibrosis patients that is funded, in part, by up to a $25 million award from the Cystic Fibrosis Foundation, something we're incredibly proud and delighted with.

There are approximately 70,000 cystic fibrosis patients in the U.S. and in Europe. Treatment and prevention of cystic fibrosis using lenabasum represents an estimated annual market opportunity of up to about $1 billion. Tying this all together, for these three lenabasum indications currently in our pipeline, there are approximately 350,000 patients in the U.S., Europe, and Japan. There is

a total market opportunity of up to $5 billion potentially, and this excludes lupus, which is an earlier program of ours.

With unencumbered commercial rights in the major markets, as well as in China, we are well positioned with an expanded strategic optionality. It's worth noting, for example, that we have an agreement with the Japanese Regulatory Authorities that does not require an additional Japan specific clinical study beyond our current international Phase 3 study with lenabasum, in which we have a number of Japanese sites. This provides us with a number of obvious commercial advantages.

In short, we believe Corbus today is in an even stronger position than before, and we are excited about the value creation potential inherent in our pipeline, especially given the unencumbered commercial rights in these key geographies.

Let me now turn briefly to Slide 14, which outlines the competitive landscape. On the one hand, you have phytocannabinoid companies that focus on CB1 agonists for CNS, primarily for brain diseases or conditions, such as pain, nausea, epilepsy, without having an impact on the immune system. These used plant-derived cannabinoids also known as phytocannabinoids, and include companies with recently approved commercial products, such as GW Pharma and Insys.

Our approach, of course, is very, very different, relying on rationally designed, man-made small molecules that are specifically designed to target and modulate inflammation and fibrosis. Our candidates have a unique mechanism of action that can target either the CB1 or CB2 receptors with reduced or minimal penetration of the blood brain barrier, and without immunosuppression. They also have a different safety and tolerability profile to phytocannabinoids that target CB1 in the brain.

On the other hand, a number of both big and small pharma companies compete in our target indications with immunosuppression drug candidates that typically target a specific pro-inflammatory mediator. This year has seen quite a few fails in the clinic with this approach, including Roche with their upcoming Phase 3 systemic sclerosis data presentation at ACR.

This is a great segue to turn briefly to Slide 17. Earlier this year, we engaged DiscoverX to use their BioMAP platform to profile lenabasum against other commercially available, often successful, anti-inflammatory drugs. I'm going to ask Barbara to comment on this data. Barbara.

Dr. Barbara White: These data are from the DiscoverX BioMAP platform, which is a pre-clinical system that models fibrotic diseases under inflammatory and pro-fibrotic conditions. It provides biomarker readouts that can be compared to the profiles of the selected clinical drugs. In this case, Roche's anti IL-6 receptor monoclonal antibody, which failed to meet its primary efficacy outcome in Phase 3 testing in systemic sclerosis and Boehringer Ingelheim's Ofev (nintedanib)

and Genentech Esbriet (pirfenidone), which were the treatments for idiopathic pulmonary fibrosis.

In this system, lenabasum caused little inhibition of T and B cell and monocyte activation, which is consistent with the lack of immunosuppression seen in clinical studies with lenabasum to date. In contrast, lenabasum had significant effects on biomarkers of myofibroblast activation, fibrosis related matrix activities, and tissue remodeling, wound healing activities, which would be relevant to the treatment of fibrotic diseases, such as systemic sclerosis and NASH.

Dr. Yuval Cohen: Thanks, Barbara. You can find additional information on lenabasum in our investor presentations on Slide 16 to 35. I'm sure many of you are very familiar with this data, but of course, always take the time and review it at your leisure.

But, I want to go back to our announcement for today and let's turn to Slide number 37. As an overview, Jenrin is a Pennsylvania based, private company, focused on endocannabinoid system targeting drugs that are designed to avoid penetrating the brain. One of their most exciting candidates is CRB-4001, a peripherally restricted CB1 reverse agonist with promising pre-clinical data.

I'm going to ask Barbara to walk us through why we're so excited about CRB-4001.

Dr. Barbara White: We are excited because this compound, CRB-4001, provides us with the opportunity to develop new treatments for a number of serious inflammatory and fibrotic diseases by blocking CB1 in the periphery. We believe that CRB-4001 should not have the psychological side effects that limited treatment with first generation CB1 agonists, such as Rimonabant, because it is not expected to penetrate the blood brain barrier well.

This slide shows very low levels of CRB-4001 in the brain when compared to the plasma, and minimal binding of CB1 in the brain, as assessed by PET scanning in mice. At the same time, it is a more potent activator of cyclic AMP than Rimonabant.

Slide 39 shows disease pathways in NASH, from obesity to fat deposition in the liver, to liver inflammation, and then fibrosis, and lastly, liver failure with cirrhosis. Pre-clinical data show that CRB-4001 inhibits fat deposition in the liver in mice on a high fat diet and reduces biomarkers of liver inflammation and damage in these mice on a high fat diet. These are key initiating events in NASH.

Slide 40 shows blockage of CB1 normalizes expression of genes involved in fibrogenesis in a mouse model of radiation induced pulmonary fibrosis, blocks expression of collagen, alpha smooth muscle actin, and both transforming growth factor beta and connective tissue growth factor. Absence of CB1 signaling also attenuates fibrosis in another organ, the heart.

In summary, we are excited by the applicability of peripherally restricted CB1 blocker, CRB-4001, to the treatment of serious fibrotic diseases in which the unmet need remains great. With that, I turn it back to you, Yuval.

Dr. Yuval Cohen: Thank you, Barbara. As we start to wrap up, I'd like to point out that while we're proud of the pipeline we're building, our most valuable asset is our team, and we are incredibly excited today to announce that Dr. George Kunos is joining our Scientific Advisory Board.

Dr. Kunos is the Scientific Director of the National Institute on Alcohol Abuse and Alcoholism, a division of the NIH, the National Institutes of Health. He will provide invaluable scientific support for the development of compounds that target the endocannabinoid system as therapeutics for a broad range of diseases. Importantly, Dr. Kunos led the work at the National Institutes of Health to advance CRB-4001 to clinical testing, and he plans to coordinate a first-in-patient Phase 1b/2a proof-of-concept clinical study at the National Institutes of Health, following the completion of a Phase 1 study by Corbus next year.

Dr. Kunos will be an important asset to Corbus and we're delighted to welcome him to the team. And, now, I'd like to invite Dr. Kunos, who's on the line with us, to say a few words, share a few thoughts with us. And, Dr. Kunos, if I could just guide some of the topics, I'm wondering, that our audience would be interested in hearing, and I'd say the following:

Could you share with us why are you excited about this program? What have you seen to date with this compound, with this approach? How would you envision the Phase 2 study that you will be conducting? And, last but not least, maybe a few words on the potential overall of this mechanism of action, of targeting the endocannabinoid system in the periphery for these types of inflammatory/metabolic diseases.

Dr. George Kunos: Thank you, Yuval, and I'm really pleased to be here. And, just by way of introduction, let me say that my laboratory has been focusing on the biology and pharmacology of the endocannabinoid system for the last 20/25 years. Some 17 years ago, we published an important paper in Nature in 2001 that demonstrated, for the first time, that endocannabinoids promote appetite by interacting with the hypothalamic leptin regulated appetitive systems.

This observation, and then similar observation later by others, was one of the rationales for the development by the Sanofi company of the first globally acting CB1 antagonist, Rimonabant as an anti-obesity agent, based on the assumption that by blocking appetite, body weight could be reduced. Now, clinical, 30 clinical studies with Rimonabant were very encouraging, because it turned out that Rimonabant not only reduced body weight, but simultaneously inhibited practically all of the important metabolic complications of visceral obesity, which are usually referred to as the metabolic syndrome.

For example, it reduced liver fat accumulations, it improved plasma lipid profile, and it improved insulin resistance in the subject. Now, these findings and all of these effects reducing CB1 blockage led to the concept that an increased activity of the endocannabinoid system may be a primary epigenetic factor in the metabolic syndrome.

So, following these studies, our laboratory, a few years later, made an unexpected observation that cannabinoid receptors, surprisingly, were present in the peripheral tissue in the liver where previously they were not suspected to be present. Not only they were present, but functional, and their stimulation in insulin resistance and increased lipogenesis.

So, about this time, the clinical studies started to reveal that Rimonabant, unfortunately, had a side effect, neuropsychiatric side effect, which ultimately led to the failure of this compound and its withdrawal from the market. And, beyond that, it even threatened this whole class of compounds from being further developed for clinical benefit.

However, our observations that cannabinoid receptors, functional receptors, are present in the liver and others at the same time as reported similar findings in adipose tissues, skeletal muscle, the endocrine pancreas, led us to speculate that maybe some or most of the metabolic benefit of CB1 blocking is due to blocking receptors in peripheral tissues, and if it could develop second generation compound with limited brain penetrance, we could minimize or eliminate the neuropsychiatric liability and retain some or most of the metabolic benefit.

And, this was clearly outlined as a proposal or speculation in an early paper, following which I was approached by the Jenrin company saying that they, based on a similar consideration, have already developed a series of CB1 antagonists with reduced or minimal brain penetrance and would I be interested in collaborating with them, as at the time, the main proponent of this hypothesis, which I was very glad to do. This was about in 2011.

And, in addition, because our early--yeah, so let's go on the early studies. We used diet-induced obesity model in mouse, which fairly well reproduces the human metabolic syndrome. These animals become not only obese, develop fatty liver, have lipid edemas, and they develop insulin resistance. And, all of these effects are very effectively practically normalized by the Jenrin compound, which I refer to as the JB5037, now called [CRB-]4001.

And, at the same time, we could document that there was no significant occupancy of CB1 receptors in the brain, as demonstrated by CB1 positive remission tomography studies, and there were no behavioral effects that are usually accompany the administration of Rimonabant that are analogous to the psychiatric side effects in humans.

Now, based on these encouraging results, I submitted a grant application to the National Center for the Advancement of Translational Sciences, or NCATS, a component of NIH, and was fortunate to win a competitive award from them in the amount of about $2 million, and the award was for the development of promising compounds of clinical potential to do all the IND enabling studies. And, the way this is done at the NIH, the NCATS team collaborates with the economic investigators, in this case, my lab, as well as the Jenrin group, and that there's several years, this study led ultimately to an IND application to the FDA, which was successful.

And, this actually is considered an NCATS a significant success. They support many such projects, only few of which reach an IND, and as a result, I just learned that the NCATS director will give an award to the team, including their people, my lab, and the Jenrin people. So, this is one level, how NIH is committed to this work.

The second level is that our scientific ambition, and I have to say I have no commercial interest, I'm not in the patent and we had no agreement with Jenrin for any of the commercial benefits. My interest is purely to see this drug through from the bench to the bedside, and in that spirit, I suggested that, with a team of prominent colleagues in the intramural program, mostly in the National Institute of Diabetes and Digestive and Kidney Diseases, NIDDK, and also at NINH. We will set up a team to design and conduct an intramural study of Phase 1a first in human proof of principle study. It's a mechanistic study looking for biomarkers, and it will include such parameters as measuring liver fat content by monitoring it by MRI, and monitoring 24 hour glucose handling plasma leptin endocannabinoid levels, and also a quite unique approach to do human metabolic studies in metabolic chamber that was set up at NIDDK, which allows 24 hour monitoring of oxygen consumption, CO2 production, and that would allow calculating energy expenditure in subjects that would take either vehicle or the compound.

Furthermore, we're also planning to include CB1 PET studies in humans, similar to the ones we conducted in our animal model, to verify that a critically affected growth does not lead to significant occupancy of receptors in the brain. So, this is a second level of NIH commitment, and we--all of us scientists who committed to involve in this project have our own budget and we use our own resources to cover most of the cost of the program, and of course, we expect that Corbus will help us with the compound.

And, there is also a third potential level of commitment. The NIH has large critical research networks. One such network is for NASH. It's coordinated through NIDDK. It involves 10 or 12 prominent pathology centers nationwide, and with NIH support, their commitment is to test emerging new compounds, which have a potential of treating NASH. Two years ago, they invited me to the meeting of the principles to present our pre-clinical data, after which I was contacted by them that they would be very excited, once the compound, at the time which we called JB5037, becomes available for a multi-center Phase 2 study, they would be very interested in running such a study at significant cost savings to the sponsor. So, this will cost money, but due

to the way this metric is set up, NIH offers certain resources that reduce the cost. So, these are the three levels of NIH commitments for this compound. I will stop here and I will be happy to answer questions.

Dr. Yuval Cohen: Thank you so much. Thank you, Dr. Kunos, and again, I can't express enough how delighted and honored you are that you'll be part of our Scientific Advisory Board. Now, let me just start to wrap up, briefly cover the terms of the transactions as summarized on Slide 37, how this is a classic licensing agreement. It was structured with an upfront milestone in royalty payments. Importantly, this transaction will have a minimal impact on company cash flow through the end of 2019. And, as you can also see, much of this further development work will actually be in collaboration with the National Institute of Health. The IND for Phase 1 is already open. You can read more about this in the 8K filings that we filed this morning.

And, so before we open the call to questions, I'd just like to reiterate how excited and focused we are by the opportunity to expand our portfolio with Jenrin's complementary assets. We believe that there is tremendous potential here, and we are confident that the expansion of our pipeline will create long term value and potential tangible benefits for populations suffering from these chronic inflammatory diseases.

With that, Operator, we will open the lines for Q&A. Operator, first question, please.

Operator: Thank you. At this time, we'll be conducting a question and answer session. If you would like to ask a question, please press Star 1 on your telephone keypad. A confirmation tone will indicate your line is in the question queue. You may press Star 2 if you would like to remove your question from the queue. And, for participants using speaker equipment, it may be necessary to pick up the handset before pressing the star keys.

Our first question is from Elemer Piros with Cantor Fitzgerald. Please proceed with your question.

Elemer Piros: Yes, my first--excuse me. My first question will be to Dr. Kunos, if I may.

Dr. Yuval Cohen: Sure.

Elemer Piros: Dr. Kunos, the presentation references a compound AM6545, which is apparently a CB1 antagonist. The JD5037 drug is an inverse agonist on the CB1 receptor. Now, if you look at the data that was generated with the antagonist in a long fibrosis model, what would your expectation be, the difference between the responsiveness of what we call now, CRB-4001? So, what is the essential difference in this model, between an antagonist and an inverse agonist?

Dr. George Kunos: Yes. So, that's an interesting question. As you may have noted, we published also a paper in the Journal of Clinical Investigation on the AM6545 compound, which is a so-called

neutral antagonist, which was also very effective in improving the metabolic end points in the same mouse model that we used later for the Jenrin compound. And, some of the endpoints were very effectively reversed, or even normalized. At other endpoints, the drug was partially effective, and that led to the question, is it possible that if we use an inverse agonist, which produces not only occupies the receptor and blocks the effective endocannabinoid, but produces an effect opposite to the endocannabinoid, that would improve the metabolic efficiency of such compound.

So, we--that was defined and Jenrin approached me and we tested their compound, the JD compound, and it turned out to be an inverse agonist. So, with that hypothesis, we started to do the study and the end result suggests that, at doses which were comparable to produce near maximal or maximal peripheral CB1 blocking. At some of the endpoints, the inverse agonist, JB compound, was significantly more efficacious in reversing the metabolic syndrome endpoints than the AM compound, which is a neutral antagonist.

Now, I'm not sure that one can predict that this difference will exist for many other types of endpoints, such as fibrosis, lung fibrosis, or any other endpoint that might be affected with CB1. That may need to be tested, but there's a theoretical consideration that inverse agonism could be an added factor of efficacy for these compounds.

Elemer Piros: Thank you very much. And, Yuval, if you would, please, explain to us what some of the gating guidance before the Phase 1/2a trial could be initiated.

Dr. Yuval Cohen: Sure. Barbara?

Dr. Barbara White: The gating items are, first of all, we have to do the transition. There has to be a transition from Jenrin through to Corbus, and we are already starting that. That's underway, and when that's done, then, in fact, we would and could be ready to do a Phase 1 at that point. So, we do anticipate being able to do that in 2019. We will internally, and we are again, in the middle of discussions about how to absolutely optimize the development plan. This compound has lots and lots of promise, and we want to make sure that the initial steps will facilitate its ultimate speedy approval.

So, we will fill you in later on the details of just exactly what that plan looks like. But, certainly, the plan is to start the Phase 1 in 2019, followed very quickly by the study that Dr. Kunos described, to give us all the biomarkers that we need to understand, treatment effect and issues to design additional studies.

Dr. Yuval Cohen: And, if I can chime in, Elemer, again, one of the nice things about this transaction is the human element of it. Jenrin really are bringing highly, highly specialized skills to the table.

Their team has been just remarkably collaborative, and we very, very much look forward to working with them together.

Elemer Piros: Thank you very much.

Dr. George Kunos: If I may also add something.

Dr. Yuval Cohen: --Please, George--

Dr. George Kunos: Throughout the Jenrin process, we plan to stay in close contact with Corbus and get information about the outcome of the Phase 1 studies and in parallel, we will go ahead and, based on our extensive pre-clinical information, we will start designing the first-in-human clinical study, and even move it through the IRB. So, once the compound becomes available for testing, we would like to have it start this way.

Dr. Barbara White: I just want to echo that. There's almost nothing more exciting, for those of us in industry, to get to work with such fabulous experts with so many things at their disposal, as we have, in our opportunity to work with Dr. Kunos and the NIH. It's just a truly exciting opportunity for us.

Elemer Piros: Thank you again.

Dr. Yuval Cohen: Thank you, Elemer.

Operator: Our next question is from Laura Chico with Raymond James. Please proceed with your question.

Timur Ivannikov: Hi, good morning, this is actually Timur Ivannikov on for Laura Chico and thanks for taking our questions. So, first question, maybe step back a little bit. Could you expand a bit on the range of assets you considered adding and what stood out about Jenrin's program specifically that made it a good fit?

Dr. Yuval Cohen: Sure. So, if you think about it, take a step back, what is lenabasum? Lenabasum is, on the one hand, our most advanced program. We are ready for launch in 2021. It's in Phase--it is or will be in Phase 3. It's already in Phase 3 in scleroderma. It's going into Phase 3 in dermatomyositis. It's in a very, very large second Phase 2 study and it's in an early Phase 2 in lupus.

But, on the other hand, what lenabasum is, in a sense, is us going and planting a stake in the territory of using the endocannabinoid system to modulate a disease processes. Once we've done that, once we've built that expertise, once we have--that vision has now been backed by so

much data, it was a very logical conclusion to go and say, “Okay, what else out there exists that we can bring in-house that will strengthen our pipeline along the same biology?”

But, on the other hand, as I mentioned, not necessarily the son of lenabasum, but rather the brothers and sisters of lenabasum. We looked at a number of opportunities and the Jenrin one made the most sense to us. First of all, their compounds are unique. Secondly, their IP portfolio is strong, it’s fresh, it’s expandable. They are a small private company, and it makes it much easier for us to deal with a company of that size, because we ourselves are not a very big company. And, last but not least, it wasn’t just a purely a collection of early stage programs, but also a program that’s very promising, has a lot of data, again, strong intellectual property around it, but that’s ready for Phase 1 and where preparations for Phase 2, with the NIH are already underway. We thought that just ticked all the right boxes for us.

Timur Ivannikov: Okay, thanks. And, could you elaborate a little bit more on what outcomes will be assessed in the Phase 1 study?

Dr. Barbara White: So, this is Barbara--I just wanted to say, the very first study is a straightforward safety PK study. So, now, I’ll pass it to Dr. Kunos, because the subsequent study will be the one that focuses on biomarkers. George.

Dr. George Kunos: Yes. So, as I pointed out, we were trying to select biomarkers that are likely to show an effect within the 28-day treatment period, because the current toxicology data only allow up to 28 days of treatment. And, this was the result with extensive discussion between myself and my clinically more experienced colleagues, and we came to this conclusion that the endpoints worth monitoring would be liver fat content, which could be serially measured by MRI, 24-hour glucose handling and plasma insulin glucose, and plasma leptin levels, which all displayed very rapid, normal in patient in the animal studies.

And, even critically, I was told by my diabetologist colleague, that an effective compound could be expected to have a significant effect, not hemoglobin A1B, but some of these other glycemic parameters. And, then we will also do the CB1 PET study in the subject that were chronically treated with different doses but particularly the highest effective dose of the compound, to test or verify whether or not CNS CB1 receptors are occupied. And, there will also be close neuropsychiatric monitoring of the subjects, again, to potentially exclude or document the absence of significant neuropsychiatric side effects.

And, then the metabolic chamber study, that’s one of the effects which we were very interested in, because animal studies suggest that even a single dose of atopy CB1 inverse agonist in an overweight obese animal causes a measurable significant increase in energy expenditure and fat oxidation, which could be monitored in this human metabolic chamber. So, this will be--this will add, what I would call a human laboratory element to the early study.

And, these studies, these mechanistic studies, could then identify these biomarkers, which would allow us to get more sophisticated and use these preliminary data calculations for subsequent bonafied Phase 2 study for other indications as well.

Timur Ivannikov: Thank you.

Dr. Yuval Cohen: Cool. Next.

Operator: Our next question is from Ted Tenthoff with Piper Jaffray. Please proceed with your question.

Ted Tenthoff: Hi, congratulations, really interesting transaction, and I'm pleased to see the clear collaborative nature. I mean, it's really refreshing to hear that. So, I--following up on kind of similar questions they've asked, because this is such a rich portfolio or rich potential pipeline of leads, do you anticipate kind of going down different paths with respect to tissue selectivity for a lung fibrotic versus a liver fibrotic model? In other words, is this going to be one drug that treats fibrosis broadly, or do you see sort of really kind of screening that library for maybe more tissue specific or disease indication specific leads? Thanks a ton.

Dr. Barbara White: Hi, this is Barbara. That's just a fascinating question. Yes, I think that the approach that you have outlined makes sense, and at the same time, I'd like to say that we have every confidence that the lead compounds here, be 4001, should provide effective in multiple organs. It actually, preferentially, binds the specific isoform of CB1 called CB1-B, which is really hyper expressed in the liver. So, it's especially good for the liver, which why--which is what makes targeting the liver so good, this additional information.

Because the library, or the series of compounds that we acquired is so rich, we think that in fact, as we move forward, we will learn more about which ones are best in which tissues, and that will help guide us. But, you're exactly on target in terms of the value of using the endocannabinoid system to select compounds that provide promise in treating fibrosis in multiple organs. There is such an unmet need, whether it's lung or liver or kidney or heart. We believe that this is the way to go, target the endocannabinoid system, pick the right compounds, because the underlying biology is so robust.

And, I would also say that our strategic direction of targeting using an endocannabinoid system to develop novel compounds applies beyond the periphery. That's where we are right now. We're in the periphery and we're going after inflammation and fibrosis in the periphery. But, please remember that there are multiple, for example, neuro-inflammatory diseases, where just the right rationally designed compound, targeting the endocannabinoid system, would provide real promise for treating some of the neuro-degenerative diseases.

Dr. Yuval Cohen: And, Ted, if I can chime in and to all of our audiences. I want to share with you again, just take a step back and look at this really interesting--the strategic optionality that this gives us. Our working hypothesis, and it's more than that because we're now seeing it in action, is that endocannabinoid system for the use--for the treatment of these diseases is going to be an incredibly significant next wave in developing drugs.

In fact, speaking of CB1 targeting for NASH, the competitor we would have here is Johnson and Johnson. Johnson and Johnson, last year, acquired the rights to a CB1 agonist. It's a monoclonal antibody from Bird Rock Bio, targeting specifically the liver and targeting specifically NASH. My expectation is that more and more big pharma are going to turn their attention to this completely untapped biological system that is vast and whose potential is potentially really extraordinary.

The advantage we have as Corbus and because of our vision, because of our nimbleness, and now also because of our patent portfolio, we are considerably ahead of the pack. And, so having these unencumbered rights, commercial rights globally, having the really unique expertise of developing these drugs, I think places us in a position that could be quite dramatic.

Ted Tenthoff: Yeah, it clearly does--

Dr. George Kunos: --If I may add--If I may add two points.

Dr. Yuval Cohen: Please, George, go ahead.

Dr. George Kunos: Yeah, so there's--I'd like to remind people to two facts. The metabolic efficacy of Rimonabant has been very well documented in these large Phase 3 multi-center studies. But, I like to remind people that in all of these studies, Rimonabant was used at the single daily dose of 20 milligrams, which only produces a 30 to 40 percent receptor occupancy. And, the reason they never exceeded this dose, to get full receptor occupancy and increased efficacy, because as the dose was increased, the rate of neuropsychiatric side effects increased in parallel.

So, that really limited the efficacy of Rimonabant, which is a brain penetrant compound. We will not have this limitation with peripheral restricted compound that very likely would be able to be used at clearly effective, that is doses that cause full receptor occupancy. So, the efficacy that we expect will be better than what has been documented with Rimonabant, which was already encouraging. So, that's one.

The other thing about tissue specific targeting, I'd like to point out that in our hands, all CB1 antagonists, including several peripheral CB1 antagonists, which we had the opportunity to test, they all highly accumulate in the liver, which on the one hand, looks fortunate if the liver is your target. On the other hand, it should always raise a flag about hepatotoxicity. So, we monitor that

very carefully for any sign of hepatotoxicity by measuring plasma aminotransferase ALT levels, and not only they were not increased, but they actually, they were elevated due to the obesity, but they were normalized by the CB1 antagonist treatment.

Now, this is not always so. It was brought home to us when we tested a compound by a chemist colleague who also developed a non-brain penetrant antagonist, which was also effective in reducing liver fat, but it causes a marked increase in plasma ALT and AST, suggesting that its chemical structure, for some reason, was hepatotoxic. So, we could safely say that in all the pre-clinical studies, we had no evidence of the hepatotoxicity of this particular compound, despite the fact that it's highly concentrated in the liver.

Ted Tenthoff: Very interesting, a lot of good detail there. Excellent, thanks so much for the update, and congrats. Very, very cool transaction.

Dr. Yuval Cohen: Thank you so much. And, with that, thank you to all of the folks who joined us, again, especially on such a short notice. We are very, very gratified to see just so many people on this call. And, with that, Operator, we will sign off, and thank you for all your hard work.

Dr. George Kunos: Thank you.

Operator: Thank you. This concludes today's conference. You may disconnect your lines at this time and thank you for your participation.

[END]