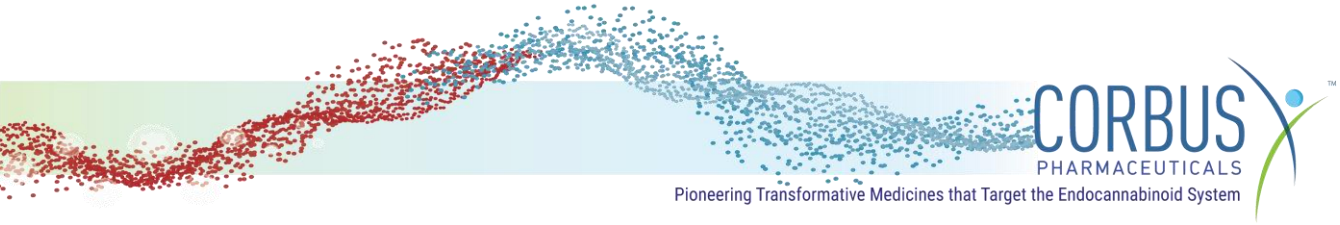

Corbus Pharmaceuticals Holdings, Inc. (CRBP)
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Leland Gershell, MD, Ph.D., Analyst, Oppenheimer & Co. Inc.: Welcome back everyone and good afternoon. Welcome to the afternoon session on the second day here at Oppenheimer's 31st Annual Healthcare Conference. We're delighted to have with us now Corbus Pharmaceuticals. Corbus is a R&D focused drug company up in the Boston area that is focused on developing drugs for the endocannabinoid system, but is also branching out into other areas as time moves forward. On behalf of the Company, I've got the CEO, Dr. Yuval Cohen, with us and we'll be having a so-called fireside chat over the next half hour. Welcome, Yuval. Thank you for joining us.

Dr. Yuval Cohen, Ph.D., Chief Executive Officer, Corbus Pharmaceuticals: Thank you, Leland. Thanks for having me.

Dr. Leland Gershell: Great. So, I think historically you have been focused on endocannabinoids and that's kind of been your roots. Maybe just to orient people who may not be as familiar, just so we can set the record straight, so as to not confuse you with some of those consumer companies who are oriented toward cannabis-derived products, the distinctions that are there.

Dr. Yuval Cohen: Yes, the nomenclature is confusing. There are three types of cannabinoids out there. They are the phytocannabinoids, they come from the plant and there are hundreds of them. Then there are the molecules that our body makes, those are known as endocannabinoids. And the third bucket is the one that you can find Corbus under, which are pharmaceutical, synthetic rational design compounds that were designed to engage with the endocannabinoid system. So that's us.

Dr. Leland Gershell: Terrific. So last year we had the unfortunate disappointment of the study with lenabasum in systemic sclerosis ("SSc"). Maybe talk a little bit about kind of what happened there just to review with us and maybe just the challenges in developing drugs for systemic sclerosis as there has been a lot of disappointments, frankly, in that field and maybe how we should think about things going forward now that ACTEMRA has gotten an approval for SSc-associated interstitial lung disease, which is a recent twist. There's a lot there, but I'll – just go back and please tell us a little bit about the trial.

Dr. Yuval Cohen: That is a lot. Okay. So systemic sclerosis - and you're right, it has been very frustrating to develop a drug successfully, partly because the endpoints are awfully complex. If we look at systemic sclerosis, we, for example, were one of I think seven clinical studies that all adopted this new and exciting endpoint called the ACR CRISS. It's an endpoint that was validated academically and that really looked very sensitive and very promising. The one problem turned out to be that – that endpoint was validated in a much older study that databases that did not have patients on immunosuppressive drugs as a background. And in that setting it works.

Even in our study, we had a small subgroup of patients that, for whatever reason, were not on immunosuppressive drugs and there that endpoint just worked beautifully. A really distinguished, I think, placebo and lenabasum.

The problem is in patients who are on background immunosuppressive drugs and especially, as we discovered, patients who are recently introduced to background immunosuppressive drugs - that endpoint just gets overwhelmed. We were the first to sort of step on that landmine. I think three days after us Galapagos had their data, exactly the same issues. I think since then, we've heard that a number of other studies have simply stopped because it's very challenging to use this endpoint with real-world population.

On the positive side, we have had two drugs approved for systemic sclerosis. Now to be very precise, they're approved for a complication of systemic sclerosis known as interstitial lung disease. Both of these drugs have been around for a while and have been approved for other things. One is OFEV® from Boehringer Ingelheim and the other one is ACTEMRA® (tocilizumab) from Roche.

What's interesting about that is a couple of things. The first one is the endpoints that they were approved for were just lung function, forced vital capacity (FVC), it's one of the secondary endpoints that we measured as well. OFEV is a much more a straightforward approval - big study focused on FVC showed a modest but clear win and went on to approval.

ACTEMRA is much more different, is much different and a little bit confusing, if that's the right term. So ACTEMRA failed in Phase 2 and in Phase 3 on their primary endpoint, which was modified Rodan skin score (mRSS), one of the components of CRISS. Roche itself announced, I think, two or three years ago I think, announced that they were abandoning the program. So, it came as a bit of a surprise to see them approved. What was perhaps equally surprising is they were approved based on a post-hoc analysis of a sub-population using a secondary endpoint and on patients who are not on standard of care. So that's a lot of sort of little strings to figure out.

It's interesting that in our post-hoc analysis, and I cannot emphasize this enough, it is a post-hoc analysis, we also saw a change in FVC in a sub-population and it was actually comparable to the change that was seen with ACTEMRA. One big difference is our patients are on standard of care and so they're more reflective. I think the big question that everyone is going to start grappling with is, is the ACTEMRA approval from the FDA a once off, because it's a peculiarity of ACTEMRA or is the agency perhaps indicating a willingness to look at post-hoc analyses of sub-population. I think there is one way to find out and time will tell.

Dr. Leland Gershell: I think on that point, we do have data coming up from another Phase 3, very important for the Company, in dermatomyositis (DM). Lenabasum is an agonist of the so-called CB2 receptor and so, this plays a role in the inflammation in a number of disorders in which inflammation is kind of the general theme. So, DM, like SSc, has these significant commentary components. Maybe just tell us for those who are less familiar with dermatomyositis because it's been less pursued by the industry, what that condition is like, how maybe similar to SSc mechanistically or symptomatically and how it's currently managed?

Dr. Yuval Cohen: Both diseases are rare, serious, autoimmune diseases with significant morbidity and mortality. Scleroderma tends to be more lethal than DM, but DM - some data out there suggest 30% mortality at a five-year mark, that's really very significant. They are both multi-organ and multi-system diseases. Both of them involve skin, internal organs, including the lung sometimes. Both are treated with immunosuppressive drugs that tend to not be approved for these diseases, they're just part of what rheumatologists will throw at these types of diseases. There are some differences. dermatomyositis, as the name implies, also can be a myopathic disease. It can have muscle involvement. It's not a dystrophy, the muscles are not dysfunctional, they're simply inflamed and therefore they start to waste, and there are some other subtle differences, for example, photosensitivity, pruritus, et cetera.

In terms of the landscape, there are some differences there as well. We were the first company to have Phase 3 data using the ACR CRISS, this composite score in scleroderma. In DM, the composite score is known as TIS, and we will not be the first. We'll be the third ones, in fact, to have data with it. There are two companies ahead of us. One of them is OCTAGAM® with IVIG. IVIG has been used for DM for decades - this is more of an insurance reimbursement play. And they hit on their primary quite nicely, I should point out, in a shorter study, much shorter than the year-long study we were envisioning. So that helps us a lot. It helps us understand what placebo should look like. It helps us understand what success could look like. It also really motivated us to shorten our study, especially from competitive reasons. We've known about those studies for a while, what has changed is they actually hit on their primary and are probably going to get approved. In that case, there's no reason for us to have a product out there that takes a year to demonstrate efficacy.

Dr. Leland Gershell: Right. So, you went from, I think it was a year study to 28 weeks, so effectively cutting in half.

Dr. Yuval Cohen: Correct.

Dr. Leland Gershell: And we had obviously positive signal from prior data in the DM population. How should we think about going from what you saw in the Phase 2 and that design? And would you have now as your current Phase 3 with the TIS endpoint and any concerns that may relate to the experience that we had in SSc, as the

investor may be approaching your stock for the first time perhaps now and wondering, hey, this could be a great opportunity ahead of the data. What should I be thinking about in terms of their so-called risk factors?

Dr. Yuval Cohen: Yeah, I think we need to be very sober about this. If we look at the two-study population, the baseline characteristics have been published for both. In both cases, the majority of patients are on background immunosuppressive drugs and the differences lie in the following, and I think it will really boil down to whether those differences make a difference. In the systemic sclerosis study, as I pointed out, where CRISS sort of collapsed were in patients who were newly introduced to immunosuppressive drugs. If I remember correctly, more than half the patients in our scleroderma study were newbies like that. Far, far fewer of those patients are in our DM study. Our DM study by luck has many more patients who were on established, long-term immunosuppressive drugs.

The second thing is we mentioned that it wasn't just immunosuppressive drugs that were the issue with the scleroderma study. There was one in particular, which is mycophenolate. Mycophenolate in the first two years turns out to work really well. We have far fewer mycophenolate patients in DM - mycophenolate is rarely used in DM. I think it will boil down to honestly is those two factors plus the fact that the diseases do have some differences between them, is that going to make the difference.

As you said, the Phase 2 study in all of these looks promising, biomarker data looks very promising across the board. It's really important again, to understand that the problem is scleroderma was not a placebo response. It was a background medication response in a population of patients that just again overwhelmed the endpoint. We'll find out. We're certainly optimistic. If we do hit on the primary, we will certainly go down to FDA and have a discussion around a path forward to approval.

Dr. Leland Gershell: And yes, so on that point, I mean, just on your last earnings call this past Monday, you intimated that we will see how the DM data may inform the approach to the FDA on maybe reopening the door on SSc. So, I mean, would you – is it fair to say that a hit in DM would increase the likelihood that SSc would be entertained, or would they be more independent than that? Just kind of from a sentiment point of view?

Dr. Yuval Cohen: I think, it's always so difficult to predict. Certainly I think a hit in DM on the primary is very helpful to say the least, not just for DM. DM itself will be a very lucrative indication for us as commercial company. But certainly, the ability to go down to the Agency and say, look, we have a clear wing on one indication and a mixed result on the other is easier to have that conversation. I think the other subtle piece may lie in what else gets better in DM, other than the primary.

DM patients can have interstitial lung disease. It's not as common as systemic sclerosis, but we've got quite a few of them that have it. If we see changes in that sub-population, if you see positive changes in their FVC, then I think it's within the realm of logical that we can then leave a story, a narrative around that to say – Look. We have two populations. We both have ILD and sub-population. We seem to be making a change there. Can we have a discussion around the path forward?

Dr. Leland Gershell: So, we'll wait and see. We'll stay tuned and those DM data are, I think, as you say, Q2, right.

Dr. Yuval Cohen: Correct. The last patient was dosed, you should expect last patient last visit announcement shortly. And we reckon sometime, probably early June, we should have a date.

Dr. Leland Gershell: Terrific, good. And again, inflammation is so common to so many disorders. So, this lupus study with the NIH, that's kind of been going on sort of in the background, as Company-sponsored work has been progressing. Just so maybe just tell us about your expectations. Sounds like we might have more visibility on timing for update on results and enrollment and where we are there?

Dr. Yuval Cohen: This is a study that is not only funded by, but also run by NIH. And so, we're very, very grateful for that, but the NIH has its own way of doing things. They are – I believe we're down to just three patients left to enroll out of 100. So, we expect that to be done shortly. It is a 16-week study. We reckon sometime in the second half of this year, they should have top line data. It is a first in-patient study dose finding, four cohorts -- placebo, low, medium, and high dose.

It's an interesting study because they're really looking for all the classic lupus outcomes: SLEDAI, BILAG, a lot of self-assessment, musculoskeletal pain. It's sort of a large dashboard of outcomes. We'll see what signals we get out of it. If we get enough positive signals and I think it's – there's room to then consider moving forward. Obviously, SLE will be our biggest indication to-date. To put it in perspective, there are probably 50,000 DM patients in the U.S., there are between half a million to a million and a half SLE patients in the U.S., so it's really orders of magnitude more.

Dr. Leland Gershell: Right. And would there be – as we think about overseas potential development, would there need to be any differences in terms of the design you would need or could you do a global pivotal the same way that has been done for your other?

Dr. Yuval Cohen: Yeah, I think there's – lupus studies have been around for quite a while. I'm trying to think of an exception and I can't. They're all multinational. They

all measured clinically both SLEDAI and BILAG, now there's a new composite I think SLEDAI plus BILAG but you're really looking at the same toolbox.

Dr. Leland Gershell: Terrific. Okay, good. As we move on to pipeline compounds. So we have the CB1 inverse agonist program that is based on a lot of observations have been made for that receptor and for compounds in the past that had issues. Tell us kind of what the opportunity would be if you were able to have this successfully safe - it seems like we're pretty good there on, the fact that if you antagonize that receptor, it's going to work for metabolic disorders, things like NASH and so forth. But in terms of if we were to have a safe compound and one that didn't get into the CNS much, what that would mean to really open up that therapeutic angle as a way to treat a variety of metabolic disorders?

Dr. Yuval Cohen: We're really excited about this one. So, this states all the way back to our Jenrin acquisition of that library, and it's fun to sort of juxtapose that program with lenabasum. Lenabasum's safety by this stage of the game, it's incredibly well understood. We really understand what it does safety wise and it tends to be well tolerated.

The big question with lenabasum is, does it actually work? Can it clinically be validated? The CB1 program is exactly the opposite. It's got a not only clinically validated target, it's a commercially validated as well. CB1 antagonists, or inverse antagonism, was a series of drugs back in the early 2000's culminating with Rimonabant from Sanofi, that was actually launched. Pretty much every big pharma had one of those in Phase 3. It's known that they work and I think Sanofi were clocking Rimonabant at peak sales of $3 billion in 2006. These are very large markets.

The problem with those drugs was, as you mentioned, they penetrated the brain, they antagonize CB1 in the brain, and that led to depression and suicidality, which was really unacceptable. The second generation, and this is part of what we're doing, or we are part of it, is really exactly the same mechanism of action but you're forcing the compound to be peripheral. You're really making it very difficult for the compound to engage with CB1 in the brain. That has turned out to be actually very difficult but we think we finally cracked it. We have a number of candidates internally that seem to be at least as potent as Rimonabant in preclinical animal models and yet seem to be orders of magnitude, less of brain penetration compared to Rimonabant. I think that the big question there is going to be looking at primate brains, because they mimic our brains much more accurately. But I think that once we've done that, that's a pretty convincing case to move forward.

The other thing I'll mention here and to the audience is, I think a super interesting development in Rimonabant was approved for weight loss and as you know, weight loss has been sort of a desert indication for pharma - very few wanted to deal with it.

This year though, we saw data for both Novo Nordisk and Eli Lilly separately under a GLP-1 agonist showing really impressive weight loss in overweight diabetics. But what was even more interesting was a paper that came out in diabetes in February from Novo Nordisk and a French academic group looking at what happens when you combine their GLP-1 agonist with a CB1 inverse agonist. They showed in a rodent model that independently they both work, we've known that, but if you put them together, they work even better.

One interesting twist is that the compound they're using as a CB1 inverse agonist, JV 5037, is actually CRB-4001. And so, it's not our favorite compound because of PK issues we're dealing now with the children of 4001, but that's really intriguing. And it totally has changed our thinking around pivoting from NASH, which has also turned out to be very difficult, to actually pivoting towards metabolic syndrome, obesity, Type 2 diabetes, et cetera, that nexus, which I think is really exciting.

Dr. Leland Gershell: Yeah. I don't think anybody would say that you're looking at a smaller market if you're looking at diabetes.

Dr. Yuval Cohen: No. And you're right, because again, the ability to do late-stage studies there for a company like ours would be very difficult. Having said that, I think that an asset with such a validated target, if we can just demonstrate the safety of it with some biomarker data, I think it makes for a very solid basis for a partnership.

Dr. Leland Gershell: So, it sounds you've done as Corbus, some, a lot of the work in terms of understanding kind of the PK, blood brain barrier, because back in the day of Sanofi and so forth, they weren't as concerned, because I don't think we knew, we didn't really have the wherewithal to really know that the CNS liability. So, it sounds like we should take comfort in the fact that these are not mutually exclusive. We should be able to have compounds that are restricted and also that are active in the way we would like to be.

Dr. Yuval Cohen: It's certainly what we're seeing and it's interesting, because the original thinking was you need to have the brain inhibition in order to reduce appetite. That turns out to be completely incorrect. It's all about peripheral CB1 in the liver and the pancreas and the gastrointestinal system. That's really where all the action is.

Dr. Leland Gershell: Good, well we look forward to that. In the first human data from I know, you kind of took a little bit of a slowdown because 4001 did have some liabilities. And if anything, I think that as somebody wearing an investor hat would say, look, we'd rather you not go forward with something which looks like it might be problematic, than take a little bit more time to take something forward that looks better. So that's okay from my perspective. When might we see the initial clinical data from that?

Dr. Yuval Cohen: Our aim is for both our CB1 metabolism program and our CB2 oncology program to be in the clinic next year.

Dr. Leland Gershell: Terrific. Good. And I think, we have a few minutes left and on the last call you were extending the outlook of the Company to go broader. Both I think in terms of therapeutic areas and also mechanisms of how the drugs would act. I don't know if you can share more thoughts here. I know it's kind of an open-ended question and there may be some things going on behind the scenes that down the road we find out about - maybe some conversations happening with external entities. But I'd love to hear about what the thoughts are down the road as Corbus maybe looks to broaden the scope.

Dr. Yuval Cohen: I don't think we've been very subtle about it. So, we have a deep interest and deep commitment to expanding our pipeline. That's really important for us. The philosophy that we have is, we want to stay in immunology. If you think about, our programs are either inflammation fibrosis, inflammation metabolism nexus, or oncology - sll of those fall under the same umbrella there, so, we want to stay there. I think what we're looking for are mechanisms of action that are well understood. Again compare CB1 inverse agonism to CB2 agonism/CB1 inverse agonism - there really is no argument over the mechanism. It's clearly worse. So, we like those types of situations . So what we're searching for are situations where we can find one of these assets or platforms, but where there's a strong case where it could be best-in-class. We're okay not being the first ones out there, but we want to be the best ones out there. And it's a really interesting.

We certainly we have the capital to do so, we have $127 million in the bank. We have the human capital to do so. We shrunk a lot, obviously. We did a big reduction in force, but we still have all the functionalities of a micro pharma company. But I think what's interesting is, I think our ability to look for assets that others perhaps neglect. We have, I think, a very, very, very good ability to do due diligence. We have some great contacts out there. And yes, we certainly have been on this journey now for a number of months and I think that it's safe to assume that it's been progressing well.

Dr. Leland Gershell: Maybe one last question just as you've had to deal with kind of the throws of the data disappointments and cash preservation and downsizing, but also having other opportunities that are not far away if they work like DM. On the commercial side, how has that organization or, I mean, I know you still have Craig Millian, the head of commercial – did you have to suffer any kind of step back in that as you went through the rest of 2020, and now have to rebuild or were things in such a state of development that you didn't overshoot, and we're kind of moving in the right progression for what would have been the FDA timelines and so forth.

Dr. Yuval Cohen: Yeah, it's exactly the latter, you nailed it. Our entire commercial department were and remain three individuals. They are extraordinary, and they've turned out to be also extraordinarily versatile and very, very helpful in other areas within the Company. But yeah, you're talking about a tiny team because yeah, we look at mistakes other companies have made and sort of a classic one is you build your commercial force too aggressively, too early when you still don't have certainty of what the data is. So, we were very good about that.

Dr. Leland Gershell: Terrific. Good. Well, I think we're up against the time here. Yuval, thanks so much for joining us. It was a great discussion. Thanks for dialing in and we look forward to seeing everyone at another session at the healthcare conference.

Dr. Yuval Cohen: Outstanding. Thank you.

Dr. Leland Gershell: Stay safe. Thank you.