Corbus Pharmaceuticals Holdings, Inc.
Cystic Fibrosis Phase 2b Study Update Conference Call and Webcast
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Operator: Greetings, and welcome to the Corbus Pharmaceuticals 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, Ms. Jenene Thomas. Thank you. You may begin.

Ms. Jenene Thomas: Good morning, everyone, and thank you for joining the Corbus Pharmaceuticals conference call and webcast to discuss the Company's announcements regarding the Phase 2b program of lenabasum for the treatment of cystic fibrosis, as well as the Company's $25 million award from the Cystic Fibrosis Foundation.

At this time, I would like to remind our listeners that remarks made during this call may state management's intentions, hopes, beliefs, expectations, or predictions of the future. These are forward-looking statements that involve risk and uncertainties. Forward-looking statements on this call are made pursuant to the Safe Harbor Provision of the Federal Securities Laws.

These forward-looking statements are based on Corbus's 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 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 Investors section of the Company's website and on the Securities and Exchange Commission website. We encourage you to review these documents carefully.

Joining us on the call today are Dr. Yuval Cohen, Corbus's Chief Executive Officer, Dr. Barbara White, the Company's Chief Medical Officer, and Sean Moran, the Company's Chief Financial Officer. It is now my pleasure to turn the call over to Yuval Cohen.

Dr. Yuval Cohen: Thank you, Jenene and good morning, everyone. Thank you for joining us on this conference call. We are delighted to be here today. The announcements over the past 24 hours mark three very important milestones for Corbus. Firstly, we have reached an understanding with FDA on the use of event rate of pulmonary exacerbation as the primary efficacy outcome in our clinical development program. This agreement provides a clear opportunity to demonstrate the clinical value of lenabasum in an area of great unmet need in cystic fibrosis, and a clear path to the registration of lenabasum for the treatment of cystic fibrosis.

Secondly, we unveiled the design of our Phase 2b study. It is the product of significant inputs from our Principle Investigators, Dr. James Chmiel and Dr. Stuart Elborn, other experts in pulmonary exacerbations in the CF community, the CFF TDN, and the ECFS CTN. We are immensely grateful to all the experts who have set aside their time and effort to guide us all the way from the preliminary design to the actual meeting with FDA and beyond.

This study is large and a first-of-its-kind, designed to provide clear cut answers to the therapeutic potential of lenabasum in reducing pulmonary exacerbations in cystic fibrosis. It will enroll more than 400 participants at about 100 centers in a multi-national study. The help of the CF community in extending--is extending to us will be an essential part of successfully completing this study. The team at Corbus is excited about undertaking such an important study, and look forward to results from this study in early 2020.

Thirdly, as you saw in this morning's press release, we are the recipient of a $25 million development award from the Cystic Fibrosis Foundation that enables us to undertake this large and comprehensive study. This follows a $5 million development award we received back in April of 2015 that permitted us to carry out the previous first in cystic fibrosis patient study. We can hardly express our deep gratitude to the Cystic Fibrosis Foundation, and to the thousands of people across the country who dedicated their time and their resources to raise funds for the discovery and development of novel therapies for cystic fibrosis.

Your commitment humbles us, your dedication empowers us, and your quest for a better future inspires us. Our commitment goes well beyond developing a potential new drug for all people living with cystic fibrosis and includes our genuine dedication to being part of this inspiring cystic fibrosis community. All of us at Corbus participate in year-round Cystic Fibrosis Foundation activities because we genuinely believe that being engaged is an essential part to making a difference.

With that, let me turn to my colleague, Dr. Barbara White, our Chief Medical Officer.

Dr. Barbara White: Good morning, everyone. Like Yuval, I'm absolutely delighted to be with you to share our good news about the agreement with the FDA on the design of the Phase 2b trial in CF, the design of that trial itself, and our funding award from the Cystic Fibrosis Foundation. Today, I'm going to cover pulmonary exacerbations in CF and their importance as the major driver of morbidity and mortality in this disease. Then, I'll cover the Phase 2b trial design.

So, what is a pulmonary exacerbation in CF? First, there is no standard definition. You might have thought there was or should be, but there isn't. Generally, a pulmonary exacerbation describes an acute event for which the physician initiates new antibiotics and that acute event is associated

with new or worsening respiratory signs or symptoms, such as a cough, new sputum, worsening sputum, shortness of breath, and generally, significant worsening in pulmonary function.

For example, a patient may have a fairly abrupt decline of 10 or 20% or even more in their FEV1. The patients are generally quite ill during a pulmonary exacerbation and many require hospitalization for treatment.

So, who gets pulmonary exacerbation? First, some patients are more likely to get them than others, and we can identify them. This is directly relevant to our trial design, because we will identify them in our upcoming Phase 2b trial by the number of pulmonary exacerbations they've had in the previous year. So, the number of pulmonary exacerbations treated with IV antibiotics in the previous year is the greatest risk factor for having a subsequent pulmonary exacerbation, requiring treatment with IV antibiotic.

It's shown in this graph and you can see the grey triangle that would correspond to a six-month time period, which is the time period of our study. As the number of previous pulmonary exacerbations increases from none to one, to two, to three, and beyond, the likelihood of having another pulmonary exacerbation during that six months increases. So, we have taken advantage of this to include in the pace--in the study, patients who've had two or three pulmonary exacerbations, treated with IV antibiotics in the last year, or if they've had one treated with IV antibiotics, if they've had a second treated with oral antibiotics, they're also eligible.

Next, how many exacerbations do CF patients get? First, any one that they get is too many, because they're associated with bad outcomes, and I'll cover that later. We can estimate the number of pulmonary exacerbations that the average patient has by data on the number of exacerbations treated with IV antibiotics and then add to that a ratio of 1.5 exacerbations treated with oral or inhaled antibiotics for every one treated with IV antibiotics.

So, from U.S. data, one can estimate a rate of about 1.8 pulmonary exacerbations treated with antibiotics per year per patient, and there's data from the UK that's somewhat similar that suggests a slightly higher rate of 3.6 across five centers in the UK. So, generally, at least several a year that they will have treated with any new antibiotic. So, it's a significant problem, particularly because pulmonary exacerbations cause bad outcomes. They reflect how the patient feels, functions, and survives. They are major drivers of morbidity and mortality in the disease, and that's why they're so important to the patient and to the regulators.

The more pulmonary exacerbations the patient has, the worse it is for that patient. They're associated with reduced survival, with failure to recover lung function during the month or six months after an exacerbation, and over longer periods of time, with greater decline in lung

function over time. They're clearly associated with reduced quality of life, and they have high financial costs. For example, some of the more severe exacerbations that might require a prolonged hospitalization for IV antibiotics could cost $125-130,000 in the U.S. So, these are very major clinical event with bad outcomes for the patient.

Here, we show, and emphasize that having a pulmonary exacerbation causes a stepwise drop in lung function over time. It's been estimated that half of the decline in lung function over the lifespan of a patient is associated with pulmonary exacerbation. For any given exacerbation that the patients are followed after treatment, only 25 to 39%, approximately, fully recover lung function by six months. Only 65% recover to at least 90% of their best FEV1 in the previous six months. And, on average, they lose about 4% FEV1 for exacerbation. Importantly, the greater the drop in FEV1 during the exacerbation, the less likely they are to recover immediately after the exacerbation, and the more likely they are to have greater decline in their FEV1 long term.

So, what causes these clinical events called pulmonary exacerbation? They are acute inflammatory events of unknown cause. There's been a lot of work looking at infections as the precipitating cause of pulmonary exacerbations. Data looking at the lung microbiome shows that it's largely stable during periods of pulmonary exacerbations and antibiotic treatment, and that short-term changes in the airway microbiota, are difficult to use to account for these pulmonary exacerbations. It can be that infections deeper in the lung, or past infections in the lung, may drive them, but it's difficult to detect this systematically.

So, as we said, they're acute exacerbations of inflammation that causes this abrupt worsening and signs and symptoms of disease. It's important to note that patients with CF have chronic inflammation in their lungs. It's a neutrophilic inflammation that is present even without infection.

When the patients are infected, this inflammation gets worse, it's excessive, it is ineffective at clearing bacteria, and it persists long beyond the time that it should. Because of the mediators that are released during this inflammation, you can get destruction of the lungs, scarring of the lungs, and loss of pulmonary function. This is how we get the bad long-term consequences of pulmonary exacerbations. And, as I said, an acute increase in inflammation is associated with pulmonary exacerbations.

It's been determined that an increase in inflammation precedes the start of an exacerbation. It can be seen during an exacerbation, generally in patchy areas throughout the lung and this radiograph shows a PET scan, and the dark spot underneath the crosshairs shows an increase in uptake of a tracer that should show inflammation, that is present during the exacerbation and the crosshairs below, gone when the exacerbation resolves. The greater the increase of

inflammation at the start of a pulmonary exacerbation, as measured by a variety of biomarkers of inflammation, the greater the risk of failure. And, if inflammation persists at the end of treatment, the more likely it is that treatment will fail or has failed. So, again, inflammation is clearly associated and drives the pulmonary exacerbation.

When a patient has an exacerbation, the primary treatment goals are to recover lung function and to make the patient feel better, because they're pretty miserable. The CFF has issued guidelines on the treatment of pulmonary exacerbations based on an analysis of the evidence. And, the evidence shows that continuing current treatment, current medications, current airway clearance therapies are likely to provide at least moderate benefit to the patients during an exacerbation. Unfortunately, there's insufficient evidence to provide clear guidance for the duration of antibiotic therapy, the number of antibiotics, the use of steroids, or even whether to treat in a hospital or as an outpatient.

It has been stated recently, avoiding pulmonary exacerbation is a key therapeutic goal in this disease. Today, there are very limited approaches and they're limited to mutation specific approaches. As I'm sure you're aware with both Kalydeco and Orkambi, secondary efficacy outcomes show either a reduction in time to or an event rate in pulmonary exacerbation. A recent publication, however, showed no improvement in short-term or long-term recovery of lung function with these CFTR modulating therapies.

So, we at Corbus, we support an input from experts in the field. The CFF and the ECFF all recognize the need for new treatments that target pulmonary exacerbations. There is a need for drugs that target inflammation without immunosuppression. Remember, these patients are infected and if you cause immunosuppression, you in fact could make these pulmonary exacerbations more or worse, not better. There's a need for drugs that will reduce the number of these exacerbations, prevent long-term loss in lung function, and very importantly, are applicable to all patients without regard to their mutations, their bacteria, or their treatment.

So, I will remind you that lenabasum is an agonist of resolution of innate immune responses. It is an upstream trigger that it helps clear inflammation from the tissue, clear bacteria from the tissue, and return tissue to homeostasis. In our completed Phase 2 study, again I remind you that we saw evidence of clinical benefit of lenabasum in pulmonary exacerbation. The first graph plots time without a pulmonary exacerbation, and you see that lenabasum provides longer time without a pulmonary exacerbation than did placebo in this study.

The second set of graphs shows the event rate. That is how many--what was the mean number of exacerbations requiring new antibiotics. And, again, there was reduction with lenabasum treatment. And, finally, we did see and could measure a reduction in inflammatory cells and

inflammatory mediators in the sputum in this completed Phase 2 study, consistent with a mechanism of action.

So, the planned Phase 2b study, as Yuval covered, we anticipate enrollment of about 415 patients. The patients--the inclusion criteria are ages 12 and above. This is really important. Because the FDA allowed us to include these adolescents, they are at risk for pulmonary exacerbations and bad outcomes, just as are the adults, and the ability to include them, I think, reflects confidence in the safety profile to date and the pharmacokinetic data on the drug.

As I mentioned, we're going to enrich for patients at risk of exacerbations. This allows us to have a very reasonable number of patients in the trial and help keep the size quite manageable. We will enroll without regard to mutation, infecting organism, antibiotic or other background treatment, including with CFTR modulators, Orkambi and Kalydeco.

The primary efficacy outcome is event rate of pulmonary exacerbations, comparing 20 milligrams twice a day with placebo. The definition, as I said before, that will be used for the primary efficacy outcome is physician decision to treat with oral, intravenous or inhaled antibiotics in the presence of at least 4 of 12 Fuch's of criteria. Fuch's criteria is an established set of criteria that has been used by others and we are happy to be using it in this trial.

As secondary efficacy endpoints, we will us other measures of pulmonary exacerbation, such as other definitions and time to. We'll look at patient quality of life with the CFQR respiratory domain score, and also FEV1 percent predicted. The expected milestone for this Phase 2b study are, that we have already activated the first site, and we anticipate dosing the first patient in the very near future. We expect to complete patient enrollment next year, complete dosing next year, and to be able to report topline results in 2020.

Again, I just want to say how very grateful we are to the CFF for all their help and the support and allowing to make this study happen, and we are so excited about having this study start. Yuval.

Dr. Yuval Cohen: Thank you. So, at this time, Operator, are there any questions for us?

Operator: Thank you. If you'd 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'd like to remove your question from the queue. For participants using speaker equipment, it may be necessary to pick up your handset before pressing the Star keys.

Our first question comes from the line of Liisa Bayko with JMP Securities. Please proceed with your question.

Ms. Liisa Bayko: Hi, guys. Congratulations on getting the trial to this point, and we're looking forward to conducting it and seeing the outcomes. So, congratulations.

Dr. Yuval Cohen: Thanks, Liisa.

Dr. Barbara White: Thank you.

Ms. Liisa Bayko: When you talked about all the sort of background level of exacerbations, I've got to believe one thing that influences that is age. Is there--is that the rate in the relevant population here of over age 12, or does it include kids, who I imagine might have a lower rate, just because they're not as advanced in their disease? And then, also, are you--does that also consider kind of the new world now with Orkambi and what not, and sort of the rates, assuming some patients are on those drugs?

Dr. Barbara White: Liisa, first, all patients, without regard to age, are at risk for pulmonary exacerbations. There is a difference in how patients preferentially treat the patients, depending upon age and other clinical characteristics so that younger patients tend to get treated with oral antibiotics, and sometimes they're not counted when you see patient numbers and rates based on IV antibiotics. But, still, I would point out that younger patients do have pulmonary exacerbations.

We're thrilled about the adolescents because their risk, at least of those with exacerbations treated with IV antibiotics, it's the same that it is, as it is for young adults. So, the risk is about the same from about age 12 to age 30, and that seems to be the highest risk group and the group that we expect to have the most patients from in this trial.

Ms. Liisa Bayko: Okay. So the numbers you gave us are probably before Orkambi was introduced, and just curious about how much impact that may have had, or maybe it's--

Dr. Barbara White: So, some of the numbers are pretty new. So, I think the numbers are pretty accurate.

Ms. Liisa Bayko: Okay, great. Um, and then just as a--just matter of background--understanding the background, so you have two doses you're exploring. Let's say you have, you know, it's a three to two randomization of the three patients that will be on drug. How does it break down

between the two different doses? I noticed only the higher dose will count towards a primary endpoint, correct?

Dr. Barbara White: Right, and that's absolutely correct, Liisa. So, the primary outcome is a comparison of 20 milligrams twice a day to placebo twice a day. And, we have 80% power to detect that given the sample size, which is about 166 patients in each of the 20 milligrams twice a day and placebo arms based on some conservative assumptions. The 5 milligrams twice a day will have about 88 patients in it. I think that's right if I can divide, right, 84 patients in it, and is intended to be able to give us an idea about dose response for both safety and efficacy. Analysis of the 5 milligram BID data will be undertaken as secondary efficacy outcomes.

Ms. Liisa Bayko: Okay, and then just one more question about the study design. Will you be stratifying patients for background therapy, you know, with CFTR correctors or anything else that you think could have an influence? And, then I'll go back into the queue. Thank you.

Dr. Barbara White: So, Liisa, we will be stratifying by geography because we think that best reflects overall differences in treatment, background medications and treatment in pulmonary exacerbations. So, that is a key factor, as well as FEV1.

Ms. Liisa Bayko: Thanks.

Operator: Thank you. Our next question--

Dr. Barbara White: And, if I could say--Liisa, if I may. I wanted to add to that a little bit. We actually looked in the Phase 2 study about who was exacerbating and who was not in terms of their baseline characteristics. And, really, just as--has been--I showed from the data, it was the patients that had the history of the most pulmonary exacerbations in the year coming into our trials who were likely to exacerbate.

Those patients actually were already heavily pre-treated with all the drugs that have been thought to have a positive influence in any way on pulmonary exacerbation. So, those patients that exacerbate are already recognized by the physicians, are already heavily treated to prevent these very bad clinical events. And, we didn't see any association with CFTR modulators in the Phase 2 study, of limited data acknowledge.

Operator: Thank you. Our next question comes from the line of Elemer Piros with Cantor Fitzgerald. Please proceed with your question.

Dr. Elemer Piros: Good morning and congratulations. Um, and--

Dr. Yuval Cohen: Thank you, Elemer.

Dr. Elemer Piros: You're welcome. Barbara, if you could just simplify a little bit on the impact on the exacerbations by the CFTR modulators. What did--has it been quantified in the past where they do have an impact?

Dr. Barbara White: Certainly. There's data that's available in their labels and in publications that certainly quantify their impact and it is a reduction that various from study to study but is thought to be a substantial reduction. And, as we know with, uh--we think it was key, actually the approval of Orkambi as a very important secondary outcome. At the same time, patients still exacerbate a lot. The data from Kalydeco does not show an improvement long term in FEV1 in folks who have exacerbations on Orkambi and the outcomes are still bad. They're still having inflammation, and a lot of it. And, that's what underlies the exacerbations.

Dr. Elemer Piros: Okay. And, you know, you elaborated a little bit about how younger patients tend to be treated with oral antibiotics, at least, those are tried – but, beyond the age factor, those--the type of exacerbation that is treated with an oral versus an inhaled versus an IV antibiotic, tell us anything about the severity of the event, or perhaps the patient's history is also involved in determining which ones to use? And, maybe a follow up to this question is, what is your expectation based on the practices of these centers? What would be the rough, distribution of how these incoming patients will be treated for the event itself?

Dr. Barbara White: Elemer, let me just start and see if I get all of them - when I'm done, if I happen to ask it again--. First of all, how did physicians decide whether to treat with oral, inhaled, or IV antibiotics? There are multiple factors that go into this, and not necessarily just severity, however you're going to quantify that. But, it includes age, it includes FEV1, it includes number in the previous year, it includes history of how well they have responded, it includes a back and forth negotiation between the patient and the physician about what the patient has to do and whether or not they want to come into the hospital.

It includes age, as I said. Older patients tend to get, perhaps, treated with IV antibiotics. All of those come to bear with the selection of the routes administration of antibiotics. There also have been follow up of does it make any difference what route someone has used? And, in terms of at least six-month outcome, it doesn't seem to make any difference. However, I would say that there's been additional data that looks in the U.S. and shown that centers that are more likely to treat with IV antibiotics, generally are the CF centers that have patients with the higher baseline FEV1.

So, that's the range of data that are available. I expect that all of those factors will come to bear when it comes to the proportion of subjects that are treated with what route of administration. But, again, I don't know it's the route that's so important in terms of the bad outcome as it is that they've had a pulmonary exacerbation, acute flare inflammation and acute opportunity to destroy some lung tissue.

Dr. Elemer Piros: Yes, and my last question--thank you for that, Barbara. My last question is what sort of reduction did you power the study to be able to achieve?

Dr. Barbara White: So, I'm not sure I'm going to disclose exactly that. But, I can say that they were conservative estimates, and there was much discussion with the experts and the CFF TDN coordinating center about exactly what we should use. And, we have made every effort to power this trial so that we can detect a statistically significant difference with a reasonable number of patients. We think it's very appropriately powered with conservative estimates.

Dr. Elemer Piros: Thank you so much, Barbara. Thanks, Yuval.

Dr. Yuval Cohen: Thank you, Elemer.

Operator: Thank you. Our next question comes from the line of Kumar Raja with Brookline Capital Markets. Please proceed with your question.

Dr. Kumar Raja: Congratulations and thanks for taking my questions. So what kind of--

Dr. Yuval Cohen: Thank you, Kumar.

Dr. Kumar Raja: Yeah. So, what kind of figure data do you guys have in adolescents and what proportion of patients are adolescents? What sort of exacerbation characteristics do you see in those patients?

Dr. Barbara White: So, our--we have toxicology data that were done in animals that were equivalent to human adolescents. That's important and critical to have that, Kumar, and we have that available. And, we also have what's called population PK modeling that helps us estimate what the exposure lenabasum is likely to be. And, that is driven largely by dose, not by age. To help ensure safety of the adolescents, we do restrict that patients need to weigh at least 40 kilograms.

So, that's an additional safety factor that we cooked into this. So, again, we were comfortable that we have adequate animal safety data and adequate PK population modeling data, and

certainly, adequate understanding of the need in adolescents. The adolescents who have pulmonary exacerbations really are the ones that are not going to do well over the next few years compared to the other adolescents.

So, the need is really great. We don't expect their exacerbations to be particularly different in signs or symptoms or drop in FEV1 or treatment, initiation from adult--those in adults.

Dr. Kumar Raja: Okay. And, in terms of differences in the number of severity of exacerbations, is there more benefit in terms of the number versus the severity and how this can be captured in the Phase 2b trial?

Dr. Barbara White: Kumar, what's--I just want to make sure I understand. Was the question whether including adolescents will, in some way, alter the severity of exacerbations we're likely to see? I'm not sure I followed the question.

Dr. Kumar Raja: No, this is not just in the context of adolescents, just in the general context. Like, you mentioned that there are some differences in terms of exacerbations. Some are more severe versus others. So, how this can be captured in the Phase 2b trial in terms of - I know, maybe some of the CF exacerbations are getting more mild as they're less--the number of reduction in the number of exacerbations.

Dr. Barbara White: So, in addition to following, and as we said the primary outcome, will be event rate, one of the secondaries will be time to. We will be capturing symptoms, we will be capturing FEV1 during the exacerbations. So, we'll be capturing the numbers of days of antibiotics. We will be capturing need to re-treat with antibiotics. So, we will have a lot of data that will allow us to compare what happens between patients who are randomly assigned to receive placebo or lenabasum. So, we think any sort of baseline differences really should be taken care of during the randomization process with the stratification that we've talked about.

Dr. Kumar Raja: Yeah, in that context, like, what are the differences in standards of care in various countries? Maybe you can touch a little bit about that.

Dr. Barbara White: Well, I think--again, I'm not sure there's a lot of hard data comparing country to country. What might be inferred, for example, from what data are out there are the physicians' willingness to initiate new antibiotics. Some may do it sooner than others. There may be some geographic differences with that. It's a little harder to pull out. Although, in the data from the 5 centers in UK, with a rate of about 3.6 and an extrapolated rate in U.S., maybe there's a difference there. But, that's pretty soft data. I think it's really going to come down to what drives the physician to treat. And, that's why, in fact, we are doing for the primary outcome, requiring Fuch's

criteria, so we at least know we're comparing apples to apples in the placebo and the lenabasum treated arm.

Dr. Kumar Raja: And, is there a potential for interim look based on the even trade you guys are looking at?

Dr. Barbara White: We are not planning interim efficacy analysis. We have an un-blinded data safety committee that will--is populated by representatives of the Cystic Fibrosis Foundation and the ECFS. They are un-blinded to allow them to best assess the safety. They certainly will have access to data related to the operations of the trial, as well as related to efficacy outcomes. But, there is no plan for an interim efficacy assessment.

Dr. Kumar Raja: Okay, great. Thanks for taking my questions.

Dr. Yuval Cohen: Thank you, Kumar.

Operator: Thank you. Our next question comes from the line of Caroline Palomeque with Noble Capital Markets. Please proceed with your question.

Mr. Tarun Aswani: Hi, this is Tarun Aswani calling in for Caroline. Thank you for taking my question. Our question is on the SLE trial - how's the Phase 2 enrollment going for SLE? And, is there a timeline update for a data readout?

Dr. Barbara White: So, the SLE trial, as you know, is being operationally run by the NIH, not by Corbus. So, what I can say is that the SLE trial has been activated and that enrollment is ongoing.

Mr. Tarun Aswani: Okay. Thank you very much.

Operator: Thank you. Our next question comes from the line of Laura Chico with Raymond James. Please proceed with your question.

Dr. Laura Chico: Good morning, guys. Thanks and congratulations on the update. I just wanted to run through real quickly, in terms of the, I guess, filing requirements, should we be assuming that if you were successful with the Phase 2b design here, and this study, that this could serve as the basis for a filing in CF?

Dr. Barbara White: Laura, good morning. Our discussion with the FDA was a Type C meeting, which is not an end of Phase 2 meeting. So, our discussions with the FDA were all about adequacy of this trial to demonstrate safety and efficacy if we were to use it to support registration. And,

they provided the clarity that we're delighted to have about primary efficacy endpoint. So, I think that I wouldn't want to predict. We need to see data. We need to look at the clarity and robustness of that data. And, we have designed the endpoints knowing that they are acceptable to the FDA. So, I hope that's adequate.

Dr. Laura Chico: Yeah, that's helpful, Barbara. And, then, I guess one other question, there was an interesting study that was published in 2015 that demonstrated that a collection of inflammatory markers actually predicted treatment response to CF pulmonary exacerbations. And, I'm just curious, among the other measures of pulmonary exacerbations, are you going to be employing any sort of biomarker analysis or something that demonstrates kind of target engagement?

Dr. Barbara White: Yes, we think that's a very important thing to do. We were delighted to see, in that first, short, small study, any movement in sputum biomarkers. And, we agree, we think that provides information that can be very helpful and very supportive. So, we'll look at sputum in the upcoming study and we'll look at the same outcome - cells and mediators.

If I could, I wanted to pause and let you know we've been joined by Dr. Stuart Elborn, who is one of the Principle Investigators in this study. Stuart is such an expert in cystic fibrosis. He's been so helpful and he has a passion for pulmonary exacerbations in CF and the need for treatment. So, if you don't mind, I just wanted to take a second and see if Stuart had any comments. And, if any of you have any particular questions that you'd like to ask Stuart, I'm sure he'd be glad to take them.

Dr. Stuart Elborn: Thanks, Barbara. I'll just very briefly mention a couple of things. Firstly, that CFTR modulation will not fix infection and inflammation in the airways. And, I think this will be a new front, even with the development of drugs which, may be disease modifying, but will not fix the structural damage and will not overcome inflammation.

The second issue, which answers really that last question, is, I think developing better biomarkers is going to be really key. I think we're some way from specific biomarkers for specific treatments that would predict outcome. But I think there's useful growing literature on, modulating inflammation across the piece and I think the study that UC did shows that with this product, we can make an impact on inflammation.

And, just finally, exacerbations are catastrophes for people with CF. And, in data cross, a number of respiratory conditions, I think that that we're seeing that exacerbations that drive lung destruction and that's why they're absolute predictive of--per outcomes and survival. And, CF being, I feel like where there's most skin on that in terms of the inflammatory processes, I think

by reducing exacerbations, we can have an important impact, which will be added to CFTR once you get a therapy, over the next number of years for people with CF. And, that's why I think the endpoint of exacerbations in this coming study is so important.

Dr. Barbara White: Does anyone actually--could we ask, if anyone has any questions for Stuart, really, he is so knowledgeable. And if you've got any remaining one, I'd urge you to take advantage of him being on the phone.

Operator: If you'd like to ask a question, please press Star 1 on your telephone keypad. We'll pause a moment to allow for any other questions. Dr. Cohen, there are no further questions at this time. Would you like to make any final comments?

Dr. Yuval Cohen: Yes, please. I would like to thank all of you, and especially Professor Elborn for joining us this morning. Again, I thank all the external experts, the Corbus staff, who have made this upcoming clinical study possible, and the Cystic Fibrosis Foundation for its invaluable support. Without all of you, this would not be possible. Thank you all so very much and we look forward to keeping you informed on our progress.

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