Operator: Greetings and welcome to the Matinas BioPharma Holdings MAT2203 Clinical Development Update Call. 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, zero 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, Investor Relations for Matinas BioPharma. Thank you. You may begin.

Ms. Jenene Thomas: Good morning, everyone, and thank you for joining us for the Matinas BioPharma MAT2203 Clinical Program Update Call.

At this time, I would like to remind our listeners that remarks made during this call may statement management's intentions, hopes, beliefs, expectations or projections of the future. These are forward-looking statements that involve risks and uncertainties. Forward-looking statements on this call are made pursuant to the Safe Harbor provisions of the Federal Securities Laws. These forward-looking statements are based on Matinas BioPharma'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 to differ materially from those contemplated by such forward-looking statements are discussed in the periodic reports Matinas BioPharma 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's website. We encourage you to review these documents carefully.

Following the Company's prepared remarks, the call will be opened up for a question and answer session. Joining me on the call today are Roelof Rongen, the Company's CEO, Jerome Jabbour, President, Dr. Raphael Mannino, Chief Scientific Officer, and Douglas Kling, Senior Vice President, Clinical Development and Project Management. I will now turn the call over to Roelof Rongen.

Mr. Roelof Rongen: Good morning, and thank you for joining us today. The purpose of today's call is to discuss the topline data from our Phase 2 clinical study of MAT2203 for the treatment of vulvovaginal candidiasis (VVC).

Speaking simply, while we are disappointed in not meeting our expectations with respect to both clinical and mycological responses for MAT2203 in this patient population, we were very encouraged by further demonstrating that oral delivery of encochleated amphotericin B is safe and well tolerated without delivering kidney toxicities typically seen with administration of intravenous amphotericin B. Further, we were pleased to see what we believe could indicate
clinical response, which may provide support for the systemic delivery of MAT2203 to the site of infection.

Before we go into more detailed review of the data from the study, I wanted to remind everyone of the study design and purpose of this Phase 2 study. This completed proof-of-concept Phase 2 study of MAT2203 was a multi-center, randomized trial with the primary objective to evaluate the safety of two orally administered doses, 200 and 400 mg, of MAT2203 compared to the 150 mg of Fluconazole in the 137 women enrolled in this study. Secondary efficacy objectives were to assess the clinical cure rate and the mycological eradication rate of oral MAT2203 compared with Fluconazole in the 79 women with confirmed vulvovaginal candidiasis at the baseline in the modified intend to treat population. And third tier objectives were to assess the pharmacokinetics of MAT2203 after five days of oral administration.

In the context of our overall development program, this Phase 2 study was not designed or powered to support an indication for the treatment of VVC and therefore supplant Fluconazole as the standard of care. Instead, our goal was, in addition to further establishing the safety and tolerability of MAT2203, to demonstrate efficacy of MAT2203 in the non-life threatening fungal infection consistent with the development of other antifungal therapies as we prepare MAT2203 to enter a pivotal study in the prevention of invasive fungal infections.

In this particular study, we were not successful in demonstrating meaningful clinical or mycological responses of MAT2203 compared to Fluconazole. However, upon review of the improvements over baseline of the composite clinical score of patient signs and symptoms at Day 5 and Day 12 in both the 200 and 400 mg arms, we were pleased to see that what we believe could indicate clinical response, which may provide the support for the systemic delivery of MAT2203 to the site of infection.

I would now like to ask Douglas Kling, our Senior Vice President of Clinical Development, to provide an overview of the efficacy and safety information from the study.

**Mr. Douglas Kling:** Thanks, Roelof.

In looking first at the safety and tolerability in this study, we were able to further demonstrate that MAT2203 is safe and well tolerated. There were no serious Adverse Events reported in the trial, and the majority of treatment emergent adverse events were mild in severity and unrelated to study drug.

Drug-related Treatment Emergent Adverse Events of orally-delivered encochleated amphotericin B should be evaluated in the context of the side effects of IV-administered amphotericin B, which is well known for its severe and potentially lethal side effects. In this trial, drug-related Treatment Emergent Adverse Events occurred in 20% of the 200 mg patients
and 18% of 400 mg patients compared to 2% of patients on Fluconazole. The most frequently occurring drug-related Treatment Emergent Adverse Event in the MAT2203 groups were gastrointestinal and mild in nature.

Secondary efficacy endpoints included evaluation of clinical and mycological response. Clinical response was evaluated using a composite scoring system that compared signs and symptoms of VVC from baseline to the test of cure on Day 12. Signs and symptoms included itching, burning, irritation, erythema, edema or excoriation.

MAT2203 demonstrated a clinical cure in 52% of patients at 200 mg per day and 55% of patients at 400 mg per day compared to 75% of patients on Fluconazole. In the mycology outcome, 36% of patients in the 200 mg arm and 32% in the 400 mg arm experienced eradication compared to 84% of patients in the Fluconazole arm.

In the composite clinical score of signs and symptoms at Day 12, MAT2203 demonstrated an 81% improvement in clinical symptoms at 200 mg per day and an 80% improvement at 400 mg per day compared to 94% of improvement in clinical symptoms for the patients on Fluconazole.

In terms of pharmacokinetics, we are in receipt of draft data which shows levels consistent with what we have seen in the NIH and our healthy volunteer studies. Once finalized, we expect these data will be able to tell us more about the systemic availability of MAT2203 in this patient population.

That is the extent of the data generated to date from this study, so now I can turn the call back over to Roelof Rongen.

**Mr. Roelof Rongen:** Thank you, Doug.

Importantly, we continue to believe in the potential of our unique platform technology as it relates to the development of MAT2203 for the prevention and treatment of invasive fungal infections. In looking at the data generated from this study, contrasted with the data from our ongoing Phase 2 study at the NIH, it appears that both higher doses and longer duration of therapy, which yielded to significant clinical response in the immunocompromised patients in the NIH study, could be important factors in demonstrating efficacy in mucosal candidiasis. Accordingly, we believe that utilizing a higher dose for a longer duration in this study may have resulted in improvement in the overall clinical and mycological responses.

Despite the results from this single study of MAT2203 at low doses and for short duration, we believe that the extensive existing body of preclinical and human data established to date with the cochleate technology warrants continued development of MAT2203, and we look forward to advancing MAT2203 to build a solid overall efficacy data package. We believe that, with this
Phase 2 study, we continue to generate important data about MAT2203 and the unique mechanism of action of the cochleate technology, which will be invaluable as we design additional studies in support of this development program.

We will be receiving additional data from the study over the next few months, and we intend to continue to evaluate these data from the study as it becomes available, including data on pharmacokinetics, and we will provide guidance on details and timing of additional development plans for MAT2203 during the third quarter of 2017.

That concludes our prepared remarks for this morning, and I would like to turn the call back to the operator for question and answers.

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, one on your telephone keypad. A confirmation tone will indicate your line is in the question queue. You may press star, two 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 key.

Our first question comes from the line of Jason McCarthy with Maxim Group. Please proceed with your question.

Mr. Jason McCarthy: Hi, guys. Um, a couple of questions - could you talk about the difference between this study in VVC and the NIH study, in terms of the mycology response because they're different in the way that the endpoints are being determined. One is qualitative, one is more quantitative. Can you discuss those nuances?

Mr. Douglas Kling: Yeah, I think the biggest difference is the patient population. At the NIH, we're studying immunocompromised patients, so you'd expect that you would not get those patients to culture negative. These patients--the two patients we have enrolled have Job's, which is an inherited immune deficiency, whereas the patients in the VVC trial, these are otherwise healthy patients.

Dr. Raphael Mannino: And, Jason, I think there's another understanding that the major difference in terms of how you report mycological response in both of those studies. In the NIH study, as we reported two weeks ago, you can see that the patients were swabbed and colonies were counted so that you could see a reduction in colony count, which we saw was very effective in both of these patients, even though they had compromised immune systems.

In this study, it was an eradication baseline, which means a patient could have gone from 500 colonies or 100 colonies down to 5 or 10 colonies, showing a mycological effect. But, according to the standards of this study, if you didn't go down to zero, you were called a failure. Uh, and I
think, again, in the context of what we believe to be the mechanism of action of cochleates, that the ability to reach across the mucosal membrane to the surface, in terms of bringing drug there, is why we're indicating that we believe a higher dose and a longer duration may affect positively a greater mycological response. But, again, I think zero is a standard that's very different from significant reduction in mycology.

Mr. Jason McCarthy: Right. And just to follow up on that, in terms of more time, you know, you only went out to Day 12 and I would think that for systemic delivery, it might take some more time to get across the mucosal membrane. How far out in treatment will you go to get PK data? And will you have any additional efficacy measures later on?

Mr. Douglas Kling: So, in this trial, we only dosed for 5 days. In the NIH trial, we're dosing for, you know, up to 54 days in the acute phase of the trial. So, we're dosing for much longer in the NIH trial--but, we'll collect PK from every study we do. We collected PK in this trial. Going forward, we'll collect PK out to, you know, 54, 80 days, as we go through the development program.

Mr. Jason McCarthy: Okay, great. I'm gonna jump back in the queue. I might have a follow-up question after that.

Mr. Roelof Rongen: Thank you, Jason.

Operator: Thank you. Our next question comes from the line of Ram Selvaraju Rodman and Renshaw. Please proceed with your question.

Mr. Ram Selvaraju: Thanks very much for taking my questions. Can you hear me?

Mr. Roelof Rongen: Yes, Ram, we can. Thank you.

Mr. Ram Selvaraju: Okay. So, two very quick things - firstly, in terms of how long you expect to potentially have to dose in order to see efficacy in VVC, could you comment on what's likely to be not only necessary but also, clinically feasible in this context, given the fact that Fluconazole is effective? Can you legitimately get to a point to where this would be a viable candidate in VVC, if you needed to dose for, you know, two weeks or longer, or is it really too early to speculate at this point?

And then, secondly, with respect to the clinical activity that you expect to see, could you comment on what additional indications beyond the ones in which you are currently studying 2203, would 2203 be potentially applicable where you believe you might be in a better position to see a rapid response and a response that is competitive with or commensurate with existing,
anti-fungal agents, simply based on, uh, the possibility of greater bioavailability than potentially what we're seeing here? Thank you.

**Mr. Roelof Rongen:** Yeah, excellent questions. First of all, this VVC study was a proof-of-concept study. It is not our intention to develop the product in vulvovaginal candidiasis. You know, these were patients that had a non-lethal fungal infection in an early stage of antifungal development. You typically, for ethical reasons, do not go with a drug that is, you know, not sufficiently tried and tested yet, to patients that are at acute risk of dying. So, that's why these studies are in a development plan. And you can see that, across the board, that's what had to be done for other antifungal therapies.

There's both an efficacy but also, very importantly, a safety component to that. Here we're looking at amphotericin B - this is known by many doctors who go to medical school. They learn a nickname called “ampho-terrible,” because of its severe side effects. So, our goal was to establish the proof-of-principle for this product and you know, we as a company, are not going to put endless effort into VVC, because it's simply not our development goal. We want to take this product to those patients where other anti-fungal products do not work for reasons of resistance, which we see in increasing rates with azole therapies, with echinocandin therapies, and to patients that are already kind of use these agents for drug/drug interaction reasons.

So, we have here the most broad-spectrum, you know, now demonstrating consistently safe and convenient, orally-administered anti-fungal therapy. And our goal is to take those--that product to the patients of the highest need.

So, when you look at our extensive body of evidence, there's a number of elements there. One, you know, the technology that has been explored and continues to be explored, in terms of how does delivery work, most experience has been with invasive fungal infections. And I think what you saw most recently was a very strong poster that was presented by NIH researchers at the New Orleans ICAAC/ASM Microbe Meeting. And in the cryptococcal meningitis study that was conducted by Dr. Williamson from the NIH, both a strong survival effect was seen as well as a beautiful set of pictures demonstrating that elements of the cochleate particles were in the brain of the infective mouse, right next to the crypto cells. A picture is worth a million words in this case.

I'd also like to point out that, over the years, we've done extensive work with institutions like the PHRI, which is associated with Rutgers University. Dr. David Perlin has done extensive work in invasive candidiasis - again, an invasive fungal infection, potentially lethal, and invasive aspergillosis, very much a lethal fungal infection - and we've consistently shown that we can have high efficacy rates beating all comparators that were used in these studies.
And so, that builds our confidence that the product can deliver in invasive fungal infections. And we are with VVC, you know, slightly outside of that box, you know, purely for the purpose of, you know, a first, or a second, in this case, steppingstone study. And I think we have the great fortune to contrast this to the NIH study where you can actually see a very significant clinical response. These patients asked the investigator if they could stay longer on the drug after they were done with the 54 day regimen. And the NIH, you know, went back to their IRB and wrote a protocol amendment to make that happen because the patients wanted to be on it. It made a difference in their life, and that's what we're hanging our hat on.

You know, we have done so much work. We have patients who did not want to stop that trial and for us, this is a Phase 2 study - we are learning about the product. The one thing I've seen in my career, biotech companies often jump the gun, go straight to Phase 3, you know, for a variety of reasons. But, there is no surrogate for first learning about your product, understand how to best use it and then go through your Phase 3 trials. And has been a learning experience. We didn't see what we expected to see, but we will learn and already have learned very much from this Phase 2 study.

Mr. Ram Selvaraju: Okay. And just a quick follow-up - I mean, you mentioned obviously, and I think this has been the case in other conference calls and presentations, that the intent has never been to formally pursue the approval or commercial launch of 2203 in vulvovaginal candidiasis. But, maybe you could make it more clear - how much further exploration you intend to conduct with 2203 in VVC in order to learn about the product candidate, before moving into other areas?

Mr. Roelof Rongen: That's a fair question. We are evaluating that at this point in time. We have a variety of options available. That's one option, but we also have options available in the invasive fungal infection area, and, you know, we feel very supported there with our preclinical data.

So, for us, this is now a time to go the next layer of data and to put a plan together, discuss with our Key Opinion Leaders and advisors how it all weighs together and how they would like to go into the next step. You know, we are not working in a vacuum. We are working to address the need of patients and of the physicians that deal with these patients often, you know, in a lethal situation.

So, we are strongly guided by how they see it fit and, you know, I think we're encouraged by, what we have heard over the last year or two, with respect to how they would like to take the product forward. And I'm sure that they're still as interested moving forward into the next level of studies, and those could include potentially so more work in VVC, but also invasive fungal infections are very high on the list.
Mr. Ram Selvaraju: Thank you very much.

Mr. Roelof Rongen: Thank you, Ram.

Operator: Thank you. Our next question is a follow-up from the line of Jason McCarthy with Maxim Group. Please proceed with your question.

Mr. Jason McCarthy: Hi, guys. I wonder--I wanted you to comment on this: I know VVC is really not the indication. You know, if patients had an additional option with amphotericin, I think docs would use it. Same thing if you have drug-resistant, oral infections in the mucosa. But, I think the bottom line is that, if you can get systemic delivery of amphotericin, you really would use this to prophylax patients, maybe for bone marrow transplant. Could you talk about the utility of 2203 in that setting because I think that--you know, if you had an amphotericin that you could use to prevent infections, it would be widely adopted.

Mr. Roelof Rongen: Yes, thank you, Jason. That's an excellent question. And, you know, the prevention of invasive fungal infection - we've stated that now, since last year, that is the primary goal of development of our MAT2203 product. What we're thinking of here is the downside of patients getting an invasive fungal infection. If we step back and look back 100 years, you know, in those days, fungal infections, invasive fungal infections were rare. You know, the fingernail and other types of external infections were the most common forms, but these invasive fungal infections were rare, and you would see them almost essentially in immunocompromised patients.

And it is the advance of medical technology that has really made that a growth problem, if you will. It is the use of immunosuppressive therapies in oncology, in autoimmune disease, it's--in transplant, that has driven the rise of these invasive fungal infections. Once you use these terrible drugs that are needed to treat these conditions, now you see the immune system is down regulated. Sometimes, there's a side effect, sometimes by purpose like in transplant. That gives the fungi the opportunity to start growing. And unfortunately, for patients who get into that situation, the lethality rate is tremendous - you know, typically more than 50%. If it's something like cryptococcal meningitis, it could be 70 or 80%.

So, with that prospect, the logic to use an antifungal therapy to go into a prevention mode is very strong and I think there's a number of practical reasons why that has, only partially, taken hold so far. Because when you think of the patients and the base regimen of drugs that they have to take for their underlying condition, whether it is leukemia or whether it is a transplant situation, drugs are metabolized through the liver, excreted through the kidney. And if there is a drug/drug interaction that makes one of the other drugs is not, you know, coming to the level where it's supposed to be, you can get very severe side effects from, you know, suddenly P
concentrations of these very toxic oncology drugs, for instance. So that has been the practical limitation.

Now, amphotericin does not have a liver metabolism through the typical cytochrome pathways. So, there is a limit to degree of drug interaction in that respect. It was very toxic, and that's why it could never be used in a broad sense. It was IV, so it was very inconvenient to use it for months in a row. And so we addressed those two attributes of amphotericin, those negative attributes, and turned it into a positive—you know, the tolerability and safety profile is holding up very well. We have now 91 patients in this trial if you subtract the Fluconazole group exposed to our product for multiple days. For many months now, patients have been exposed to amphotericin B MAT2203 in the NIH trials. We see data consistent there with what we've seen on the poster a few weeks ago in New Orleans.

So, we now have a drug that can be used for prevention. The physicians we work with have indicated that that's where they need us, that's where we continue to see growth, and that's where we want to take the product. And the profile seems to be right on. This study was a steppingstone study to basically get to the point where we can start that Phase 3 Program.

Mr. Jason McCarthy: Right. And there was a comment earlier about, you know, treatment for only five days. The NIH study, you've gone out for a significant amount of time. Can you just talk about how long you've gone out with amphotericin and, you know, how that relates to the period of vulnerability, you know, for prophylaxis, when a patient might be neutropenic because I think that's really important for investors to understand.

Mr. Roelof Rongen: Yeah, thank you. That is actually, a very, very good question. In the NIH study, we have gone so far out to six months. And why is that a significant duration? Well, one, if you step back and look at IV amphotericin, just to give people listening an idea of how bad amphotericin can affect kidney function with IV amphotericin, typically, you see a 20% discontinuation rate because of kidney function in a week, 40% after two weeks. And so it's very difficult to maintain patients on amphotericin therapy and the issue usually is these invasive fungal infections last much longer.

Now, if we go to prevention, a typical AML regimen can use--can be a month where you see very low counts of white blood cell. That is the immunocompromised period for the patient. Neutropenia is the term that's been used in the medical literature.

But if you go to a condition like ALL or a stem cell transplant, the duration is often more like three months. And so, seeing six months with no issues on the safety side in our NIH study is now a double margin. And you know, we will continue to support the patients in the NIH study, you know, in their medical clinical needs and so that will give us a much better perspective on how large that margin can be, how long that margin can be. But, the longer we can maintain
patients, the more comfort there will be that, in a larger population in the Phase 3 setting or in a practical clinical setting later on, the lower the probability would be that there is a safety issue. So I think the technology, you know, has a fundamental edge here that is starting to really bear out.

**Mr. Jason McCarthy:** Okay. Thanks for taking my questions, guys.

**Mr. Roelof Rongen:** Thank you, Jason.

**Operator:** Thank you. This concludes our Q&A session and thus concludes our conference today. Thank you for your participation, and have a wonderful day. You may now disconnect your lines.