MATINAS BIOPHARMA

Clinical Development Program Update

October-06-2016

Operator: Greetings and welcome to the Matinas BioPharma 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. Thank you, Ms. Thomas. You may begin.

Ms. Jenene Thomas: Thank you, Tim.

Good afternoon, everyone. I'd like to remind you that today's webcast will be accompanied by a slide presentation that can be found under the Investors section of the company's website, www.matinasbiopharma.com, under Events and Presentations.

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 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 Investor section of the company's website and on the SEC'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 us on the call today are Roelof Rongen, Matinas BioPharma's Chief Executive Officer, Jerry Jabbour, the Company's President, Dr. Raphael Mannino, Chief Technology Officer, Doug Kling, Senior Vice President of Clinical Development, and special guest, Dr. Peter Pappas, William E. Dismukes Professor of Medicine in the Division of Infectious Diseases at the University of Alabama at Birmingham, President of the Mycoses Study Group Education and Research Consortium and a member of Matinas' Scientific Advisory Board.

It is now my pleasure to turn the call over to Jerry Jabbour.

Mr. Jerry Jabbour: Thank you, Jenene.

Good afternoon, everyone, and thank you for joining us today. We are excited to provide you with an update on Matinas BioPharma and specifically on our lead clinical product candidate, MAT2203, our lipid crystal nano-particle formulation of the broad spectrum antifungal drug amphotericin B. We believe this product and the underlying technology used to formulate this drug has the potential to really become a promising solution for physicians and patients.

As you likely recall, in 2015, we acquired the exclusive license to our cochleate delivery technology platform. Since then, our focus with respect to MAT2203 has been on establishing and optimizing GMP manufacturing, applying for and receiving key regulatory designations, and taking the time to understand our product and its unique and differentiated profile. One of our key objectives was to design an overall clinical development strategy, which we believe provides us with a clear and rapid path to approval and commercialization for use in patient populations where MAT2203 has the potential to generate the greatest value.

Having dosed our first patient in our NIH-sponsored Phase 2a open-label trial of MAT2203 last week, we believe it is a good time to lay out our clinical development strategy for our flagship product candidate. As we sit here today, we have never been in a better position as a company. We want to use this opportunity today to help guide you through the areas of significant unmet medical need that we are looking to fulfill, the disruptive nature of our delivery technology, our vision and strategic commercial positioning for MAT2203, and the subsequent development path we have chosen to optimize the value and time to approval for this potentially revolutionary drug.

Ultimately and importantly, we believe MAT2203 will be able to fill an unfortunately ever-growing gap and solve significant limitations in the prevention and treatment of fungal infections.

Over the course of the last year, we have developed very positive relationships with some of the most noted infectious disease specialists in the world in order to inform our development program strategy for MAT2203. Joining us today on the call is Dr. Peter Pappas, a member of our Scientific Advisory Board and one of the leading mycologists in the world. Dr. Pappas will begin today's call by providing his perspective on the significant treatment shortcomings in the antifungal area and the important role that MAT2203 could play effectively treating and preventing invasive fungal infections (IFI).

Dr. Pappas, thank you for joining us today. You now have the floor.

Dr. Peter Pappas: Thank you, Jerry.

Well, first of all, I'd like to thank you all for inviting me and, provide the perspective of a clinician and a clinical investigator, and to really talk about what I see as a very, very difficult and serious problem moving forward into the next decade, and that specifically is the treatment and prevention of invasive fungal infections.

We have, over the last few decades, seen the immergence of new therapies that have expanded our activity against a number of pathogens, molds and yeasts most especially, and we have made good strides. But, ever-present in these sorts of situations are the development of resistance, complications to taking the medications themselves. And they're occurring in or these occur in the most vulnerable of patients, patients who have stem cell solid organ transplants, patients on high dose steroids, those with hematologic malignancies, really the most vulnerable of the patients, among those that we see. These fungal infections occur with rates of up to 30 percent and account for a significant amount of morbidity and mortality.

Now, my perspective as a clinician and clinical investigator is as, President of the MSG, or the Mycoses Study Group, and we have performed studies that have enrolled literally hundreds of patients over the past several decades. So, my perspective is unique in this area, and so that's the perspective that I really come from.

As I've indicated earlier, our advances have been coupled with some setbacks and that is that as we move forward and we treat patients effectively with immunosuppressive therapy to help them manage their diseases, whether it be rheumatoid arthritis or lupus or cancer, what we sacrifice in most of those situations is we render the patient immunologically incompetent. That is, we make them more liable to sustain one of these devastating infections.

You couple that with the development of drug resistance and drug intolerance, and it really sets up a situation where there's a crying need for better tolerated more effective agents, and there's probably no area in clinical medicine where this need is any greater.

Should just provide as an example that, among the patients that we deal with, who are at risk for these types of infections, their mortality rates, once they develop an invasive fungal infection, ranges anywhere from about 30, up to 75 percent for those with fulminant sepsis. So, we're really in need of drugs that allow us to dose people in a more smart sort of fashion, to use better therapy and really to the extent that it's possible, identify people who are at risk and begin initiating treatment for prophylaxis, or I should say prophylactic type of therapy, as opposed to waiting until the disease is well established.

In order to do that, you need an agent that's pharmacokinetically favorable, that has excellent activity, that has fungicidal activity against most yeast and fungi, that is, when I say fungicidal, I mean the ability to kill as opposed to stun the organism. And of course, in a perfect world, this

would be well tolerated, non-toxic even after prolonged use, and would have minimal drug-drug interactions. And that is, really kind of the Holy Grail.

And really also from the standpoint of convenience and health economy, this ideal therapy could also be administered orally, not precluding IV therapy but certainly, oral administration would be a very, very important cost effective type of feature.

We became interested in working with Matinas after we had an opportunity to review the abundance of data that they have from preclinical studies and with its cochleate formulation of amphotericin B. I came away believing that, if Matinas can duplicate the same strong results we saw in their animal models of candidiasis, aspergillosis, and cryptococcal disease, and replicate that in human clinical trials, particularly with the oral dosing, this would be a real game changer for the clinician using this compound, MAT2203. It would really change our ability to manage the risk of highly lethal fungal infections in these very, very compromised patients.

So, I would just conclude saying this is really something unique to this group. It's a very unique and eloquent cochleate technology that's taken us to this point. We certainly want to see this compound developed, and we are perfectly willing to partner and happy to partner with Matinas in helping them to develop it and get it to the next label or to the next level.

And so, those are really the content of my thoughts at this point, and I'd like to turn the call back over to Raphael Mannino of Matinas, who will walk you briefly through the attributes of this very fascinating delivery technology that he invented.

Mr. Raphael Mannino: Thank you, Dr. Pappas and good afternoon, everyone.

I would just like to give you a brief overview today of the cochleate lipid crystal nano-particle delivery technology that Dr. Pappas has just mentioned and try to bring out why this is such a revolutionary technology with its unique attributes and why its specific use with the fungicidal broad spectrum drug, amphotericin B, has the potential to completely change the pharmacotherapeutic paradigm for invasive fungal infections that Dr. Pappas just mentioned a few minutes ago.

So let's get started. From a structural perspective, cochleates are prepared from natural components, specifically soybean, lipid and calcium. These nanoparticles form spontaneously, and the resulting nano-crystal is a solid, multi-layered structure with little or no internal space. This natural and spontaneous formulation process is critical because it allows for highly consistent and reproducible large scale manufacturing. This is something that has been very difficult for nanomedicines until now.

So, when looking at the structure of our nanocochleate formulations, we can see that the drugs are formulated into the internal lipid bilayers. The diagram that you can see on slide seven

provides a good visual of these layers. It is the presence of calcium that gives these particles their stability. This is one of the reasons why we're able to get oral bioavailability.

The calcium ion concentration in biological fluids, including gastrointestinal secretions, plasma and interstitial fluid, is high enough to stabilize cochleate formulations within the body. Therefore, when administered orally, cochleates remain intact within the gastrointestinal tract. They do not open and they do not release free drug into the GI tract. This is another one of the unique attributes that sets cochleates apart from other oral drug delivery technologies.

Now, once absorbed, cochleates appear to enter the circulatory system by crossing the intestinal epithelium of the GI tract via the intestinal lymphatic system. Once within the lymphatic circulation, intact drug cochleate formulations are taken up by activated cells and transported directly to the sites of infection or inflammation.

However, as illustrated in slide eight, once inside the cell, the necessarily low intracellular calcium concentration results in the destabilization of the cochleate nanocrystal and the subsequent intracellular release of the encochleated drug, again protecting the body from the drug because it is inside the cell and protecting the drug from the body.

Now as you can see in slide nine, cochleates are essentially changing the pharmacokinetics and biodistribution of drugs. We have been able to consistently and repeatedly demonstrate high drug concentrations at the site of infection and very low free drug in the plasma. This process is different from the classic penicillin model, but analogous to the uptake in tissue targeting of macrolides such as azithromycin. These low levels of free drug in the blood and high drug concentrations at the site of infection results in drug cochleate oral delivery, demonstrating high efficiency and low to no toxicity.

Another example of the effectiveness of cochleates in the battle against fungal infections is demonstrated nicely in the picture on slide 10. There we can see how cochleates are also taken up directly by the fungal cells, which are traditionally resistant to the uptake of free drug. Fungal hyphae aggressively take up cochleates, adding to the effectiveness of this delivery vehicle.

As far as we know, this is the only antifungal drug formulation which has exhibited this phenomenon. It is pictures and evidence like this, that originally got us and the National Institutes of Health so excited about the promise of cochleates, a promise which we are working hard to fulfill using an aggressive and opportunistic development strategy and clinical plan.

And now I would like to turn the call over to our CEO, Roelof Rongen, to talk about our vision, our strategy and the developmental plan for this potentially breakthrough product.

Mr. Roelof Rongen: Thank you, Raphael.

Now that we understand the unmet medical need and the promise of our delivery technology, what I'd like to do next is to outline our clinical development strategy, our product positioning, and the overall market opportunity for MAT2203.

When we looked at the product profile for MAT2203, we saw the following: First, amphotericin B is a proven molecule. It's both fungicidal and a broad spectrum antifungal agent. Secondly, amphotericin B is not metabolized through the typical liver pathways, and therefore does not have the drug-drug interactions we see with triazole antifungal products. This allows broad use together with complicated oncology regimens. And third, our unique cochleate technology provides oral bioavailability while dramatically reducing toxicity.

So, based on this product profile and the wealth of positive and consistently duplicated preclinical data, one of our first goals was to gain designation for the applicable regulatory exclusivities that potentially apply to our product. In the filing for and receiving Qualified Infectious Disease Product (QIDP) and Fast Track designations for the treatment of invasive candidiasis and aspergillus, and along with the prevention of invasive fungal infections for patients on immunosuppressive therapy, we have set ourselves up to be eligible for five years of exclusivity upon approval of our products in these designations.

This designation is also extremely important and we believe highly valuable because it, in essence, makes up for the lack of what is called new chemical entity, or NCE exclusivity, given that the amphotericin B molecule has been approved for a long time already.

Next, we are seeking designations for the treatment of orphan diseases under the Orphan Drug Act, as well. So, once granted, the Orphan Drug designation can add up to seven years of additional exclusivity on top of that five years of QIDP exclusivity, providing a total of up to 12 years of market exclusivity, all of which is not dependent on the existence or strength of patents.

With that said, we've taken deliberate steps to also build a very robust IPS state. With 19 issued patents and more than 20 additional patents pending, our assets are well protected on all fronts.

In terms of product positioning, as we spoke more and more with the leading Key Opinion Leaders in the antifungal space and we saw the problems that they are facing on a daily basis, we began to look very closely at prevention as the potential lead indication - no surprise, given the discussions that we had so far. So, understanding the strengths of our products combined with the like of broadly usable prophylactic treatments, usually because of drug-drug interactions, we determined that an indication for prevention represents the most attractive opportunity from an unmet medical need perspective.

So, we think here of the treatment of hematological malignancies, which involve the use of a range of immunosuppressive therapies, and these therapies place patients at a significant increased risk for developing severe fungal infections, and you heard from Dr. Pappas how lethal they can be - 30 to 75 percent potentially.

The prevention of invasive fungal infections in patients with compromised immune systems is therefore a very significant opportunity for a drug like MAT2203, which can impair the efficacy of a broad spectrum agent with targeted, and safe, and oral delivery afforded by the cochleate technology.

Currently, there are very few antifungal agents approved for preventative use, and those that are approved are limited by shortcomings. So, with that, we believe that MAT2203 has best in class potential as a prophylactic or preventative antifungal upon approval in that indication.

Working backwards from that approval goal, we designed a comprehensive yet efficient clinical development program moving forward. This clinical plan begins with our important collaboration with the National Institute of Health (NIH), which has become an ardent supporter of, and also an important collaborator in the development of, the cochleate delivery technology over the past few years. And in fact, we'll have our first opportunity to see patient data on efficacy and safety of MAT2203 in the coming months with the start of the NIH trial last week, the first dosing. These data from that trial will certainly represent an important milestone for our company and for our delivery technology.

Now, in an effort to strengthen our data package and to increase the number of patients exposed to MAT2203, we move expeditiously toward the pivotal registration trials, and to support that move, we've designed an additional Phase 2 clinical study in patients with vulvovaginal candidiasis (VVC) using Fluconazole, an approved medication for that condition, as an active control. And we'll also conduct a small multiple dose safety, tolerability and PK study in patients undergoing treatment for hematologic malignancies.

And Doug will cover these new studies in some detail, but we believe these incremental patients will put us in a very favorable position to add depth to our data packets, and put us in a prime position to move into registration trials quickly, while at the same time adding minimal additional time to our development program, and only very modest and reasonable increase to our projected expenditures over the next 12 months.

A prevention indication will move MAT2203 to a much earlier point in the treatment cycle, which means more patients can benefit from the prevention. Those patients need protection for a much longer timeframe than the duration of a typical treatment regimen and there are fewer competing alternatives are available in the preventative arena.

Today's global amphotericin B market is about 700 million annually, and we specifically designed this development strategy that we're laying out here today to best capture and to significantly expand on this market. This is a very exciting time for our company, and we anxiously await the results, of course, of our NIH trial in the first half of 2017, which we believe will further validate the cochleate technology platform, as well.

I'd like to ask now Douglas Kling, our Senior Vice President of Clinical Development, to walk you through the details of our various programs and the rationale for why we believe that these trials in this order enable us to maximize the opportunity in front of us in an efficient and timely manner.

Mr. Doug Kling: Thanks, Roelof.

As Roelof previously discussed, our commercial objective is to develop and receive approval from MAT2203 with an indication for the prevention of invasive fungal infections in immunosuppressed patients receiving chemotherapy for hematologic malignancy. Our development plan, therefore, reflects this strategy.

This indication will combine the broad spectrum fungicidal activity of MAT2203 with the convenience of oral administration, an improved safety tolerability profile compared to the currently available formulations of amphotericin B, and fewer drug-drug interactions versus oral azole antifungal therapies.

As the first oral formulation of amphotericin B in development for systemic use, it was imperative that we initially demonstrate safety, tolerability and collect pharmacokinetic data after a single oral dose in healthy volunteers. We did this in our already completed single ascending dose study.

Our next developmental aim was to demonstrate antifungal efficacy and collect safety, tolerability and PK data after multiple doses of orally administered MAT2203 in immunocompromised patients with active fungal infections. This is the purpose of our ongoing Phase 2a study at NIH.

Now, in order to increase the size of our safety, tolerability and efficacy database and therefore place MAT2203 in the best position to enter Phase 3 development for the prevention of IFI in patients with hematologic malignancy as quickly as possible, we are going to initiate two additional Phase 2 trials. The first trial will also be in patients with mucocutaneous candidiasis. However, it will focus exclusively on efficacy, safety, tolerability in PK in patients with vulvovaginal candidiasis.

VVC was a natural choice for a second Phase 2 trial for a number of reasons including adequate availability of patients for timely enrollment of a large trial of up to 75 patients and ability to

demonstrate systemic efficacy after oral administration. In order to round out our Phase 2 development plans, Matinas is also planning to conduct a multiple dose safety, tolerability and PK study in patients undergoing treatment for a hematologic malignancy. This will provide safety and tolerability data in the most likely target population for prophylactic use.

In the ongoing NIH study, patients with chronic mucocutaneous candidiasis, that is oropharyngeal, esophageal and vulvovaginal candidiasis, are being enrolled to receive treatment with oral MAT2203. Assessments of clinical and microbiologic efficacy will be made along with measures of safety and tolerability. In this study, we will also collect samples for pharmacokinetic analysis. The key aim of this trail is to provide evidence of antifungal activity with orally administered MAT2203 against one of the most common invasive fungal pathogens, candida.

Though not considered an invasive fungal infection, efficacy and safety in patients with certain forms of mucocutaneous candidiasis has historically supported the Phase 3 development of antifungal drugs for the treatment and prevention of invasive fungal infections. This trial will enroll up to 16 patients.

The typical response rate in these patients with oral antifungal therapies, azoles, is 5 percent. Therefore, the statistics for this trial have been designed in such a way that if 20 percent of the patients experience a clinical response, we will have achieved our primary efficacy endpoint. 20 percent represents three patients. We need to have three patients respond in order to meet the primary endpoint in this trial.

The additional 75 patient VVC trial is being initiated in the fourth quarter of this year. In order to increase the rigor and provide an efficacy and safety benchmark, a Fluconazole active comparator arm will be included in this trial. Fluconazole is the current guideline recommended therapy for first line treatment of VVC.

Fifty patients in the trial will be randomized to MAT2203, while 25 patients will be randomized to Fluconazole. We expect that data from this trial combined with the data from the NIH trial will adequately demonstrate antifungal efficacy of orally administered MAT2203 with one of the leading organisms causing invasive fungal infections - again, candida. In addition, data from this trial will be used to support the PK safety and tolerability of MAT2203.

We plan to initiate this study in the fourth quarter of 2016, and the last patient, last visit for this study is planned for Q2 2017 with data coming available midyear 2017.

Our second additional Phase 2 study will include patients being treated with the chemotherapy regimen that typically induced neutropenia, causing suppression of the immune system and subsequently puts patients at risk for developing an IFI. Approximately 12 to 16 patients will be

enrolled into this multiple dose protocol, and the primary endpoint will be safety tolerability. Efficacy assessments will be performed, and PK samples will also be collected.

The safety tolerability data from this trial combined with the antifungal efficacy data from the two mucocutaneous candidiasis trials will demonstrate that our orally administered MAT2203 has preserved the systemic antifungal efficacy of I.V. amphotericin B in patients suffering from moderate to severe immunosuppression. Furthermore, multiple dose tolerability PK trial in patients with hematologic malignancy will verify that MAT2203 is well tolerated in the Phase 3 target population, a population at high risk for developing an IFI. We believe this will be exactly the data set we need as we prepare to meet with FDA.

After completing the previously described Phase 2 trials, an end of Phase 2 meeting will be scheduled with FDA to review our Phase 3 study plans, probably in the fourth quarter of 2017. This meeting will be centered on our target indication for the prevention of IFI in patients with hematologic malignancy.

Now, in terms of Phase 3, at this time we believe that a 4 to 500 patient trial to evaluate the efficacy and safety of MAT2003 versus our control group for the prevention of IFI in patients with hematologic malignancy being treated with a chemotherapy regimen that typically induces neutropenia would support an NDA approval for this indication. We will be able to provide much more clarity on the Phase 3 study design after we have data from our Phase 2 studies and we have met with the FDA.

However, speaking in general terms, we believe that this Phase 3 trial could begin in the first half of 2018 in support of a planned NDA filing in late 2020 or early 2021.

In addition to the lead indication for the prevention of IFI in patients with hematologic malignancy, Matinas will conduct one or more additional Phase 3 studies to evaluate the efficacy and safety of MAT2203 for the treatment of invasive fungal infections such as candidiasis, aspergillosis or cryptococcal meningitis. This will be filed as NDA supplements and have no impact on our initial NDA filing for prevention.

So, to summarize, our Phase 2a study at the NIH in patients with mucocutaneous candidiasis is currently enrolling patients. In the fourth quarter of 2016, we are initiating a Phase 2 safety and efficacy study in 75 patients with VVC. In addition, we are currently designing a Phase 2 tolerability and PK study in patients receiving chemotherapy for hematologic malignancy. These three trials will establish the safety and efficacy of oral MAT2203 in treating an active fungal infection and the safety and tolerability of MAT2003 in patients with hematologic malignancy. This will set us up for an end of Phase 2 meeting with the FDA to plan our Phase 3 program for the prevention of IFI in patients with hematologic malignancy.

Now I would like to turn the call back over to Dr. Pappas, so that he can provide some insight and perspective from a clinician and mycology expert's point of view on the development plan we have just laid out. Dr. Pappas?

Dr. Peter Pappas: Sure. Thanks, Doug.

I think that it's very important to our mission, that is the mission of the mycosis study group, that we assist biotech companies in the development of novel antifungal treatments and especially to provide our clinical insights and perspectives as these compounds are thoughtfully developed, especially with attention to, not only the niche and the feasibility of doing studies, but also, the regulatory interactions and sort of the hurdles that need to be overcome in developing new compounds.

Moreover it's really important for those of us who are clinicians to begin to see efficacy in that patient--or in those patient populations that are less ill, but nonetheless have fungal infections. And that's why it's always been logical and most companies have started out looking at mucosal candida infections - that is oral and vaginal candidiasis - as a proof of principle that a compound not only is absorbed and documented through PK/PD types of data, but also works and the efficacy is visual - one can see thrush and vaginitis being cleared up. And so, that's usually the first step, and most clinicians would really demand to see that before taking it to a much sicker population who are at risk of dying from these same organisms.

So, I and my colleagues have been very satisfied with this Phase 2 program that, Matinas has outlined and with the target of prophylaxis in at risk hematologic malignancy patients. I think that in doing this and by following this plan, I think they will have generated sufficient pharmacokinetic data and also hopefully efficacy data that will make for a very meaningful applications and interaction with both FDA and EMA for this very, very unique compound. We're very excited about this.

So that's really all I have to say, and I'll just turn the call back to Roelof now for his summary comments.

Mr. Roelof Rongen: Well, thank you, Dr. Pappas. We really appreciate your comments and enthusiastic support of our technology and products here.

Before we open the call to questions, I would like to briefly summarize the key takeaways from today's call. So, our aim is clear and our strategy is one that has been informed by some of the leading clinicians and investigators, as you heard.

First, the NIH open-label study gives an early insight into the effectiveness of our technology platform in immunocompromised patients with active fungal infections. Secondly, the new vulvovaginal candidiasis study improves the overall strength of our clinical data package and

will give us the confidence going into our meeting with the FDA to agree on our Phase 3 program in the latter half of 2017. And third, this will allow us to progress rapidly into Phase 3 with IFI prevention as a lead indication, clinically supplemented by additional pivotal studies for the treatment of such lethal infections like candidiasis, aspergillosis and cryptococcal meningitis.

There is no question that we are pursuing extremely valuable indication with our MAT2203 product. Valuable most importantly in its ability to dramatically change the treatment and prevention landscape for the patients who are suffering from and are threatened by invasive fungal infections. Also valuable for our stockholders, given the size of the opportunity and the lower risk approach to drug development that we are employing here at Matinas.

Ultimately, we do not have to be concerned about the effectiveness and approvability of this proven molecule amphotericin B. It works. What we're setting out to do and to prove, is that our formulation has the potential to dramatically alter the pharmacotherapeutic paradigm for the prevention and treatment of fungal infections.

What's also worth noting is that we spent almost the entire day today, all of our time to talk about MAT2203, but we are using the cochleate technology to develop a portfolio of differentiated therapies, highly differentiated therapies, I'd like to say, including potentially the first oral immunoglycocide MAT2501, which we're developing for the treatment of chronic and bacterial infections like non-tuberculous mycobacterium (NTM) and an increasing problem that you hear a lot about gram negative bacterial infections often developing significant rates of resistance.

And unlike many biotech companies, we are not a binary product play, and there is significant value to be created here in the coming months and years. We want to be a product driven company, and I believe you can see we are on the way there very well. And the cochleate technology with its broad applicability will give us no shortage of paths that we may choose to explore in the future.

So, with that, I will thank you for your time today, and I think we can now move into the question and answer portion of the call.

Operator: At this time, we will 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, and you may press star 2 if you would 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. One moment please while we poll for questions.

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

Mr. Jason McCarthy: Hi, guys. Congratulations. Wow. I mean, you guys have really made just a tremendous amount of progress in the--just in the last few months.

I want to walk back to the Phase 2a study in mucocutaneous candidiasis. And know it's a 16 patient study. You do need a 50 percent response rate, but only in 20 percent of patients. And it is open-label, so I'm wondering would you announce data from the first three, four or five patients that you treat? What do you think the timing of that data could be, because if you get your response rates there, you could almost be done. You would wait for the rest of the data, but I think it would be very telling early on and that could be a significant catalyst in our view for the stock.

Mr. Jerry Jabbour: Hey, Jason. This is Jerry Jabbour. Thanks for your question and obviously, we appreciate the coverage.

I think the study design is one of the great things about this NIH trial and its uniqueness, and specifically our ability to not have to necessarily get to all 16 patients. And when you talk about the statistics in treating these type of fungal infections, unfortunately, the bar is pretty low with existing therapies for effectiveness.

We're planning to come in well above that bar, but it is that unique design that I think gives us an advantage and a chance to see just how effective cochleate technology is. I do want Doug to comment specifically on the number of patients and the timeline because I think that's another thing that's to our benefit, besides the fact that it's an open-label trial that doesn't necessarily mean that we will announce data, but we'll have the benefit of talking to the NIH on a patient by patient basis and seeing the data in kind of real time.

I want to be cautious, I want to caution the group that that doesn't necessarily mean that we will announce data on a patient by patient basis. That's not really how this is done. But, it's one of the unique attributes that, real time, we'll get to see how our drug is working. But Doug, why don't you talk a little bit about expected timelines?

Mr. Doug Kling: Sure, yeah. So, we need three patients, as you said, to meet the primary endpoint. Now, these patients are typically hard to treat, and they do not respond well to oral antifungal therapies or azoles. With that said, with MAT2203, we have been able to maintain the powerful fungicidal activity of amphotericin B. So, we do not expect that it will take all 16 patients before three respond.

Now, will it take 12 patients or nine patients or six patients to enroll to get 20 percent of them to have the clinical response? I can't answer that specifically today, but I can say that we expect that, in the first half of next year, we will be able to report the clinical results from this study.

Mr. Jason McCarthy: Okay, great. And if I can, I kind of want to walk back over to hematological malignancies. I've done a lot of research of my own in fungal infections and kind of biased towards aspergillosis, but I know there's a tremendous difference between yeast and mold and rates of IFI can vary significantly from center to center. So, I guess my question is for a pivotal study, would you be targeting maybe candidiasis or invasive candida infection or aspergillus, or are you going to kind of look at all types of different infections? And, how are you going to account for the different rates of infection that you find at these different centers? And I think that's even further compounded by different types of hematological malignancy, where patients are maybe more susceptible to one type and not the other.

Mr. Roelof Rongen: Yeah, thank you, Jason. That's an excellent question because these trials, there are many design considerations. And when we look at this environment, what we've seen is that many of the major cancer centers, think here of Sloan Kettering, MD Andersen, etc., they have a very extensive database and experience and they have an expected pattern, if you will, of what type of malignancies or what type of fungal infections they will see at what rate in their center with the certain types of cancer that they treat.

So, yes, these vary from center to center, but if you put that together in the multicenter consideration, it becomes a fairly predictable matter of identifying sites and patients for a trial of this design and in that context, it makes sense to look at not just candidiasis, but also aspergillosis and maybe minor rate infections like physarum and cryptococcal meningitis.

Mr. Jason McCarthy: Now, for the Phase 2 study, I know it's a small proof of concept study in hematological malignancies. The 14-day treatment window for prevention of IFI, tell me one of the rates, or can you give me an estimate of what the rates of IFI are in just a two-week period? I'm assuming that--I wonder, if you start counting the days, you establish that the patient is neutropenic or there's some other factor that would leave them susceptible to infection?

Mr. Roelof Rongen: Yeah, what we have seen from various papers and talking with opinion leaders like Dr. Pappas and his colleagues, some centers have rates that are up to 30 percent in that patient population and these neutropenic periods can be significant. What we see typically is six to eight weeks in ALL, in AML that may be somewhat longer. If you look at other immunosuppressed patients, like stem cell transplants, after the consequence of prolonged leukemia or lymphoma problems, there it might be up to 14 weeks.

So, these are extensive treatment periods, and really, therefore, set this field apart from treating an active fungal infection where you very shortly try to rescue the patient from an

almost certain death if you don't intervene. So you have very little time to accomplish almost the impossible.

We're coming from the other end where we try to make sure that these patients do not end up in that impossible situation and that means we have to treat almost all these patients with an oral safe technology that is broad spectrum and can cover all these species that I just mentioned. Really, you can really deliver the value in a product like MAT2203.

Mr. Jason McCarthy: Great. And just one more quick one, Jerry, kind of back to you - two more Phase 2 studies. They're relatively small. Can you give us a sense of changes in operating expenses that you're looking at for the next year? And maybe give us a ballpark estimate of what a pivotal study of 4 to 500 patients could cost, because I think if you get proof of concept data, that event could come up pretty quickly.

Mr. Jerry Jabbour: Yeah, Jason, that's a great question. I mean, and one of the things that we've enjoyed some success recently, and I think that's on the back of the strength of our stockholder base. We raised--we did a successful raise of $8 million just in the last 30, 45 days or so, and that really was designed to get us through data, not only on the NIH trial, but we had already been thinking about how to best position ourselves to get to Phase 3 pivotal trials with MAT2203 as quickly as we can.

As with any biotech, we are constantly faced with decisions on allocation of resources and so, even though it looks like we're adding a lot more activity, you know, these are efficient activities, and we're an efficient company, and we're able to kind of fit these within the use of proceeds from that study to put us in position where we can get data not only from the NIH study, but we're also in position to get data hopefully from the VVC study as well. And we won't incrementally increase our burn because we've decided to put off some non-essential activities until later in 2017.

But, the reality also is that there are a lot of opportunities for us to generate additional non-dilutive capital in the coming months. Another part of our strategy is obviously strategic options. And the number of patient advocacy groups that are interested in exploring more about this technology grows every day.

So, from a cash perspective, we feel that we've never been stronger from a balance sheet perspective than following this financing. And with the number of milestones we have in the short term, we expect a lot of value to be created.

So, to add two Phase 2 trials without really increasing your burn and not extending the time that we had originally been planning to kind of sit down with the FDA was pretty unique, and I think that's because of the amount of time and planning we put in with groups like the Mycosis Study Group to design an efficient strategy.

Mr. Jason McCarthy: Okay, great. Guys, thanks for taking the questions, and really, congratulations on all the progress. It's been a great year for you guys.

Mr. Jerry Jabbour: Thanks, Jason.

Operator: Our next question comes from the line of Robert LeBoyer of Aegis Capital Corp. Please proceed with your question.

Mr. Robert LeBoyer: Thanks for taking my question and congratulations on all this progress. There's a lot of information that you covered and thanks again for all this detail.

My question really looks at the change and the shift in strategy because you had trials that were looking at immunocompromised patients who developed life threatening infections, and now we're going--now this new prophylaxis indication is looking at preventing morbidity and mortality in patients who are being treated for another primary condition with the intention of avoiding these complications and avoiding the cost of treatment. So part of the difference that I see is the length of the course of therapy and some questions about the dosing and endpoints in the potential Phase 3 or what they--what you might think of them in the, as mentioned before, the wide variability in the types of infections that result. And then, looking downstream, in terms of the pricing of a course of therapy for prophylaxis versus the Orphan Drug indications since you have a much larger patient population, what looks like a longer treatment window. And then, just comment on the dosing, any cumulative toxicities and anything else in that regard.

Mr. Jerry Jabbour: Robert, thanks for your question. Just like there was a lot that we covered on the call, there was a lot that you covered in that question. So we'll take it bit by bit.

In terms of change of strategy, we don't really look at it that way because what we knew we always had was the ability to deliver the broadest spectrum drug orally and safely. And, naturally, we're gonna think of treatment first, because some of the most invasive fungal infections today, and when you're talking about aspergillus, when you're talking about cryptococcal meningitis, just don't have solutions.

But, the more we really understood the wealth of our preclinical data and our ability to consistently replicate that study to study to study. And then over the last year, really spending time with the opinion leaders and walking them through the data and getting them about--excited about our opportunity to really be potentially the ideal drug for prophylaxis is why we targeted that indication. There's just not a lot of other medicines that have the attributes that we have to go in that direction.

So, prophylaxis was always going to be something that this drug was designed for, and what we're really seeing now, with the NIH data, it's the first building block in order to get there. We'll get to see early data on our efficacy in patients that are suffering from fungal infections.

And you talked a little bit about the size of the patient populations, and we--one thing we've always done, taking the fact that we're using a drug or a molecule that's decades old, is we always wanted to position it to have exclusivity and the QIDP and the Orphan were always central to that. We're not sacrificing our ability to take advantage of those exclusivities by moving the use of amphotericin earlier up in the chain. Prevention may sound like you're talking about enormous patient populations, but you're really still talking about Orphan patient populations.

When you break down leukemia patients into their segment groups or other patients that are on immunosuppressive therapy, those opportunities are still there, and that is really what's gonna allow us to protect the drug, but it also is gonna give us an opportunity for value pricing. Obviously, drug pricing in today's world is a controversial topic and we're way too early to start talking about how we price the drug. But, you better believe that we think every day about value. And when you're able to deliver these drugs orally, you're attacking it not only from a health economic perspective, you're taking patients out of the hospital and potentially allowing them the ability to treat in the comfort of their homes. That has an enormous impact on the health economics of drugs.

And then when you think about our ability to allow patients to not have to worry about being susceptible to these infections, if they can go on this drug as part of their oncology regimen, for example, without the concern about drug-drug interactions affecting their ability to effectively fight, these--their cancer that they're fighting, it's really a change in paradigm.

So, I'm sure Roelof wants to weigh in here on that, but from a pricing perspective, it's too early. But, it's all about value. And prophylaxis for us isn't a change as much as it is capitalizing on the best attributes of our drug.

Mr. Roelof Rongen: Yes, thank you, Jerry.

Robert, you pointed out the issue of the long term treatment, and you're right. In the trial that we're doing at the NIH, we are treating between 28 and 54 days, depending on whether there's dose titration. That's relatively long for antifungal treatments.

But it does set the paradigm and experience with long term treatment. And the reason why we believe we can do this and why the NIH investigators got comfortable with that, was our extensive safety data, 28 days, rats and dogs, in rats up to 90 milligrams per kilogram, in dogs up to 45 milligrams per kilogram. To put that into context, the original amphotericin molecule,

at 6 milligrams per kilogram, rabbits and dogs died at a single dose - not 28 days, no, a single dose. That's how lethal the compound is by itself.

That is the unique feature of the technology. We got very comfortable with that in the preclinical animal studies, and the investigators got comfortable with that in treating patients. So, that's how we got to that timeframe.

Why is that important for the prevention indication? And I think I mentioned that, typically, these neutropenic periods are many weeks. When a patient has leukemia and gets chemotherapy, there are multiple courses that get repeated over several weeks or months, and during that entire period, the immune system is suppressed. So, we're talking not just about two weeks. We typically see, we start to talk about six to eight weeks, and depending on complications with the patient, whether the patient responds, that may be extended.

So, we need to support that, and we think that, with the NIH trial, we set a foundation for those clinical paradigms and get the data on the table in immunocompromised patients, which is what we're going to face there, as well.

Mr. Jerry Jabbour: Robert, not to make you think that we forgot your question about how much does Phase 3 cost. Obviously, we've done some ballparking, and we have figures and a budget. It looks like that could come in in the 20 to $25 million range - obviously, an expense that we'll start thinking about much more heavily at the end of 2017, but after all the value has been created from the data from this first three study.

So, that's not a heavy lift for a Phase 3 trial considering the fact that it's probably over a two-year trial in order to get us there. And the number of patients--actually, prophylactic trials are much more efficient to run, easier to enroll, and so we look forward to that aspect, as well. But, that's a ballpark number.

Mr. Robert LeBoyer: Okay, thank you and thank you for that safety data. That was--that's very important, especially for amphotericin. Thanks again, and congratulations on the progress.

Mr. Jerry Jabbour: Thanks, Robert.

Operator: Our next question comes from the line of John Helander of Altun Research. Please proceed with your question.

Mr. John Helander: All right. Thanks for the conference call and taking questions. I think your guys' timeline was really intelligent. You're gonna build up a de-risk asset and status. I just have two questions. For your hematological malignancy, do you have exact chemotherapy agents that you're going to be targeting, such as Cyclophosphamide or something like this?

Mr. Roelof Rongen: We have experience with Cyclophosphamide immunocompromised subjects in many of our animal data. And so, we're very comfortable with that level of immunocompromisation.

Mr. John Helander: Okay. Do you have a specific list of agents that you're going to be looking at?

Mr. Roelof Rongen: So we are--we're still narrowing down which hematological malignancies we will ultimately be targeting. As you can imagine there are multiple forms of those, we can think of the various lymphomas and leukemias, there's various rates. And as we go through assessing which segment of that market involves which centers and how we design an efficient Phase 3 trial, we will narrow that down to a particular condition and particular chemotherapeutics.

I think the animal data that I just mentioned will help us to kind of frame the issues and the trial that we're going to do as a PK tolerability trial will further inform us how we go forward into the future.

So we take a very data driven approach. We have thesis on where we might go. And I think, before we start thinking loudly with everybody else, we first want to see the data, narrow it down to reasonable alternatives, and then we're ready to communicate that with individuals like you with very deep interest and understanding of this field.

Dr. Peter Pappas: Uh, can I make a comment here, Roelof?

Mr. Roelof Rongen: Sure.

Dr. Peter Pappas: Yeah, so this is to the question that was just asked. I think a lot of this is, as I see it, as someone just looking on the outside in, sort of like you guys-- I would think that the design of the trial would in part be--well, to a great extent will be dependent on, you know, who-- at the time that this Phase 3 is designed, who is receiving any fungal prophylaxis in this population, with what agents, and how uniform do you want it to be. This is going to be a clinical design type of issues, but, one of the compounds that comes up that would be an issue for the azoles, for instance, is Vincristine.

So, the hematologists do not like to give Voriconazole, which is a standard kind of prophylactic agent, to patients receiving Vincristine because of severe drug-drug interactions and enhancement of its neurotoxicity. So, those might be patients that you would have to take out of the mix unless you want--it just depends on how you want your control group to look. I think those are trial design types of issues.

But, I guess one of the things that excites us about this compound is that the drug-drug interactions in theory should be minimal or absent, compared--certainly compared to the azoles. And so there should be--one would think that you would have far less concern about which immunosuppressive or which myeloablative therapy they get that--as an issue as just sort of does this really--does it cause neutropenia for a period of time that's long enough to make this a useful population to study. Does that make sense?

Mr. John Helander: Okay, great. Yeah.

Mr. Roelof Rongen: Did we answer your question?

Mr. John Helander: Yeah, that's good. So, you guys are probably gonna--there's no outline yet, but in the future data driven probably selection.

Mr. Roelof Rongen: Correct.

Mr. John Helander: Okay. And then my last question is, have there been any material changes to the technology since its existence with Bioscience Liberty Incorporated?

Mr. Roelof Rongen: Yes. That's a very good question. In fact, what I like to point at is a very important patent that was issued last June of this year and what Dr. Raphael Mannino and his team have been working on for a while is to transition from a very expensive, uneconomic, synthetic lipid, to a lipid that is derived from soy. And that is not only cheaper, but most importantly, even better in formulating cochleate technology.

That's a key innovation that was recently started. The patent runs until 2033. There are other innovations that we are working on that have not seen the light of day. So we're filing a number of patents that are in the que, and you will probably see those coming, as well in the coming year.

So, yes, there is a second generation technology, in short. It is very promising. It's more efficient. It's well scalable. We've developed a very nice manufacturing process that is highly controllable, and I think we're very happy to look at the next technology level. It sets us apart from other nanotechnology levels because scalability is a key issue. A lot of very interesting nanotechnologies out there very often, the hurdle has been to go into the clinic with a sizeable enough batch to actually treat a number of patients. So, we crossed that hurdle, and I think we are very close to semi-commercial scale at this point in time.

Mr. Jerry Jabbour: And, John, but I know where your question comes from, and the reality is that Dr. Mannino started working with soy, in the late 2000s. So, the patent was just issued, but this is a kind of an inventive step that took place while he was at BDSI. And so there was a significant change in the cause during the time it was at BDSI but the timing of that is really

important because those people that way to say that this whole change in the call structure happened before it came to Matinas, and what are the real reasons why it took so long for the technology to come to fruition where it's being used in the NIH today.

The reality is, at the time that a decision on investment at BDI was necessary, the cost was $60 a gram. And so when the work on cochleates was slowed down at BDSI, it was in the early 2000s. And that is the time at which BDSI brought in Mark Sirgo and the BEMA technology and made its bet. It didn't make its bet because of a lack of confidence in the cochleates. It made its bet because that was the technology that the CEO was most comfortable with. It happens in biotech all the time.

And so, it was that improvement in the technology, going to the soy based cheap phosphatidylserine, a natural based grass material, that really attracted a lot of investment in the late 2000s. And the fact remains that BDSI was granted a significant grant from the government just around the time that Dr. Mannino left, and BDSI couldn't figure out how to make cochleates. And so, that's why that technology sat there, and Rutgers fought to pull it back, and they got it back in 2012.

An amazing amount of improvements have been made since 2012. You're talking about the optimization of formulations, the ability to identify how much lipid to use has a dramatic effect on the ability of cochleates to have that efficacy and to see the data from the animal studies that were conducted since the technology has left BDSI. And when you look at the investment and the interest from the NIH, it happened subsequent to BDSI.

And so whether or not we're talking about amphotericin B, whether or not we're talking about amikacin, improvements have been made, and they continue every day because this is not a cookie cutter technology. Different drugs load into the cochleates different. Whether it's hydrophobic or hydrophilic makes a difference. But, I appreciate the question because it gives us an opportunity, to really explain that transition. But, I want Dr. Mannino to comment a little bit, too.

Dr. Raphael Mannino: Right. I think one of the important things building on what Jerry said is, that after around 2010 and 2011 when we were working with the soy material is when we began to realize that, because of the composition of the soy material, we were actually able to encochleate different types of molecules like the amikacin much more readily and much more effectively. And a lot of that data is in this issued patent, which is why, besides the cost, which is why this patent was issued is because it was not obvious that using the soy material not only would be less expensive, but would enable us to make a broader spectrum and more effective cochleate formulation.

Mr. John Helander: Great. Thanks, for answering my question. That was good.

Mr. Jerry Jabbour: Great. Thanks, John, very much. Appreciate it.

Operator: At this time, we would like to take the time to conclude the call. This does conclude today's conference. Thank you for your participation. You may disconnect your lines at this time, and have a wonderful rest of your day.