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Peter Lawson: Okay. Good afternoon. My name's Peter Lawson. I'm one of the biotech analyst at Barclays. Welcome to our lunchtime deep dive with Biomea Therapeutics. And the goal of these calls is to kind of help preview and kind of level set. And this is third year we've been doing these fireside chats with Barclays. Biomea is the 14th in this series. We've got another one tomorrow. And hopefully, you're finding them useful. And just as a way of orientation, and this is a 30-minute call, if you have questions, email me directly, probably the best way is. I'm on Bloomberg now, so you can ping me with questions and I relay those anonymously. And it gives me great pleasure to introduce from Biomea, Thomas Butler, CEO, and founder. And time permitting, we'd love to cover the diabetes side of things, the Menin inhibitor in AML. But I know that we'd probably fill out hour or two just on that and then we won't get to the FLT3 or KRAS stuff you're doing, which is also interesting.

But maybe just the first question would be kind of, there are other menins that have been essentially more specific for like menin MLL interaction. Menin can bind multiple partners. How do you think with the degrading, how does that potentially impact? And does it benefit? Does it impact in a negative or positive way the interruption of those other binding partners? Thank you.

Thomas Butler: Yeah, absolutely. And thank you so much for having me on this afternoon's call. And good morning and good afternoon, everyone. Great question. I think just to take a step back when we started Biomea Fusion, we inherited a reversible inhibitor program. So, we spent a long time understanding the biology of menin, understanding the structural components of the target, and then understanding what benefit can a reversible inhibitor do.

It was through that exercise that we learned that covalency, we think is the best way to disrupt menin. And you're trying to, in a sense, disrupt scaffold protein that actually has no function on its own. It only has function as part of a complex. And I think that that sometimes gets lost. This target menin has a very short half-life in the cytoplasm on the order of 45 minutes. And then in the site of action, the nucleus, it has a half-life of approximately six to eight hours.

And so when we learn that menin serves as a core scaffold, not just to MLL, but it serves as a core scaffold to multiple protein partners over 1,000, we learned that trying to create a wedge between just one binding partner MLL would only serve a very small subset of patients and even a fraction of that subset because MLL in itself has over 100 different flavors. There's AF6, AF9, ENL, ELL are just a few examples. They all have a different affinity to menin.

And so, what we decided to do is scrap our reversible library, scrap the reversible inhibitors that we had started the research project with, and then start to design molecules that were independent of the binding partner. That's when we discovered the cysteine, and that's when we started to use and leverage warheads to form a covalent bond to menin.

And as we did so, we learned that not only are we partially disrupting menin, we're actually just partially disrupting menin independent of the binding partner, whether it's NPM1, whether it's MIC whether it's MLL, you name it. And so, menin MLL interaction assays are an assays that are really set to understand the interface interaction of your molecule to one particular binding partner. It doesn't really measure your ability to disrupt or degrade menin. And 219 does a really good job of partial degradation or partial disruption of menin. I hope that helps.

Peter Lawson: Yes. No, definitely. And how much degradation do you think you have of menin? Is that variable or consistent? Just ideas around that.

Thomas Butler: Yeah, it's certainly concentration dependent and it depends on, on the tumour type and the sensitivity to menin. And so, we have 50%-plus degradation in some cell lines. And in other cell lines, it can be anywhere between 25% and 40%. But again, it's variable and you have the ability to tune it accordingly and you have to understand what's the degradation that's necessary to provide which outcome.

Peter Lawson: Do you think you get a variability of that degradation in patients? I guess the diabetes data, did that reflect any variability in that degradation? Is that worry or concern or?

Thomas Butler: Yeah. Not from the eyelid perspective, no. It was fairly consistent in terms of how much control that we saw with BMF-219. I think at the end of the day, what we see from a variability perspective is everyone has a different background proliferation rate. And so, as you go to partially disrupt menin, you can variably get a different proliferation rate depending on the individual and depending on their capacity to proliferate, if that makes sense.

Peter Lawson: Got you. Okay. I'd love to pivot over to diabetes. Really interesting data at the weekend and the ability to kind of knock down A1C and just that - I guess the questions that have come up following that are just like, what is the optimal place do you think, for this drug? I know some KOLs have been bullish about it, some concerned about where it gets placed if it could potentially be a first line therapy. Because you've got availability of low cost medicines and therapies that tackle weight loss, cardiac function, et cetera. So, where do you think it kind of fits into that big picture?

Thomas Butler: Yeah, absolutely. And maybe I'll just start from the top just to set the record straight. And I think what I've learned in the past and certainly at Pharmacyclics and here at Biomea is the kind of the physician that you're asking, you kind of have to know where they are on the adoption curve versus where you are in development.

So, if you're in a phase one, phase two, obviously you need to be talking to the early adopters, not those who wait until they have five years worth of approval data and the drug's been in the market for half a decade and because they just have a different way of thinking about treating patients. So, that just set the stage there. And the reason why we embarked on this effort with BMF-219 as a covalent menin inhibitor is we learned that nobody wants to have diabetes. Nobody wants to stay on a prescription drug for the rest of their life. Nobody wants to pay for prescription medication for the rest of their lives. That should be obvious, right?

And in diabetes, oral agents are prescribed first, then injectables. That's why metformin is the cornerstone of diabetes treatment. It's frontline therapy. And so, if you think about that, metformin actually has minimal weight loss, and the weight loss is driven through gastrointestinal side effects. I think it's also important to know that the new era of these weight loss drugs, they encourage weight loss through side effects, right?

They're not helping your body in a natural way or any way. And so, why are we here? Why are we doing this? If you look at healthcare costs for diabetes, it's at all time highs despite having all of these new therapies available. So, what's going on? How come that's the case? It's because no one's addressing the root cause of the disease.

Patients cycle through medication, they start first with metformin, then they get prescribed an SGLT-2, right? Because it's oral. Then they get prescribed a GLP-1 and then other older agents followed by insulin. And you just go through this cycling episode. So, we're just kicking the can down the road, putting the problem of diabetes on the next medication. And we do this every two to three years.

And so, when you think about it that way, we want BMF-219 to be able to help patients with diabetes wake up one day and say, hey, I don't have diabetes anymore. We think this is

absolutely possible and we have ways to go in our understanding of BMF-219. That's no question. It's very early days with clinical trial. But the data today is very exciting.

And so, thinking about that, the mechanism of action for 219 is very complimentary to all the other agents out there. And so, we think BMF-219 can be used across the entire spectrum of patients with diabetes. From a clinical path perspective, where would we develop this drug? And that's a question that we get from investors. And if you think about what I just said, treatment-naive and frontline makes a lot of sense. What if 30% to 50% of patients who receive a short course of 219 never need another therapy for a year or for years?

The remaining patients will start metformin, but delaying the time to starting SGLT2, et cetera is the key. We think delaying the time a patient progresses is significant benefit, delaying the time to complications to insulin use.

This approach would also open up the door to breed diabetes, right? Again, trying to delay the progression of type two diabetes and the start of potential problems and complications down the road. We think patients want to take matters into their own hands and patients with pre-diabetes and type two diabetes post-BMF-219 still need to adjust their lifestyle and improve their nutrition and diet. I hope that helps.

Peter Lawson: Yeah, no, definitely. I guess the way I kind of think about it is like there's insulin deficiency and then insensitivity kind of. Do you think kind of regrowing or restimulating beta cells can help with both sides of that equation? Just say that - I want to kinda get that into perspective.

Thomas Butler: Yeah, we do. And I think what we have to learn here with 219 is depending on where you are starting with your current pool of beta cells, how much of 219 do you need to see, and for how long? I think that will dictate the scheme of what is appropriate for what particular patient.

So, if we're talking about frontline diabetes or maybe even pre-diabetes, perhaps the four-week or six-week course at 100 or 200 milligrams is appropriate.

For those who maybe only have 10% of a pool or 5% of a pool, maybe 200 or 100-milligram combo is appropriate. But maybe they need more like ten to 12 weeks and maybe they need a couple of cycles of it. So, that's what we have to sort out as we move forward in our clinical development.

Peter Lawson: Got you. What does the kind of clinical path look like for development here in, I guess that early line setting? Is it kind of a head-to-head versus metformin or is it a combination? Kind of just ideas around that.

Thomas Butler: Yeah, I think head-to-head is a really smart idea. We're still putting our plans together, but no one's gone head-to-head versus metformin, right? And I think it's because you have this agent that's set to actually delay or prevent the progression. And I think that would be a great point to start. BMF-219 is an incredibly potent molecule. And so, you have the ability to go into frontline as an oral agent. And then at the end of the day, we want to delay or prevent the progression of diabetes and prevent or delay the complications of diabetes and that's where the value comes in.

Peter Lawson: Yeah. Because I guess one of the comments we heard of just like introducing endogenous insulin is, does that cure everything? I mean, essentially delivering it in more beta cells, but kind of - just help us kind of understand that dialogue that the beta cell has with the rest of the body.

Thomas Butler: Yeah. And there may be other things that are going on with this patient, right? And so, we will, as we go down into later phase threes, you can look at okay, maybe the patient starts 219 and a GLP-1 at the same time. And based on where they are in the progression of their

disease, and maybe they want to stay on a GLP-1 to maintain their weight loss or stay on an SGLT2 because they need some of those additional cardio-productive benefits. But the rationale for what we're thinking about 219 is if we handle your blood sugar and we know that high blood sugar over many years causes all these complications, well, if we handle the issue upfront, perhaps those issues never happen. And that's the way we're thinking about it.

Peter Lawson: Got you. Thank you. And in the post, the ADA, it seemed very obvious there was a lot of pharma, a lot of investor, a lot of pharma interest as well. How do they think about positioning this in? And what could be considered a crowded landscape and a landscape with drugs that hit multiple components of the disease? What elements they think that this can help with their drugs?

Thomas Butler: Yeah. With the ADA data, a lot of KOLs, obviously big pharma is extremely excited about what we're doing because we're the agent that actually addresses the root cause. And there are drugs out there that address multi-component like SGLT2, like the GLP and the GLP GYPS However, they're doing so because they need to because they're treating patients who have already been kicked the can down the road, as I mentioned before. We've already let them fall down the hill. Well, what we're trying to say is, let's not let them fall down the hill.

Peter Lawson: Got you. Okay. So, do you - it's squarely focused at that very early phases of diabetes? And because I know we've heard maybe it's this kind of ten-year patient that's been with diabetes for ten years where there's fewer medicines.

Thomas Butler: We will certainly have multiple phase 3s. I think the first ones will be the earlier lines, but you're going to have to run all the phase threes that others do over time once you're commercial because you want to be able to show that you can go in combination with a GLP-1, you can go in combination with an SGLT2. And then for patients who are on insulin, our goal obviously is to get them off of insulin even though they've had diabetes for ten to 15 years. We

think we can get them off insulin at least a portion of our patients. But those are things that we have to prove out in larger studies and we'll certainly run those studies.

Peter Lawson: Got you. Okay. And then the other component that came up was the hypoglycemia seen in one or two patients. It seems that it was asymptomatic. If you could kind of talk through how transient it was, how severe, how deep did that hypoglycemia go, that definitely will be helpful.

Thomas Butler: Yeah, absolutely. So, those two patients with hypoglycemia, and for folks on the line who's not familiar, if you look at the CGM data, there's two patients who has some red in it. If you look at the ADA guidelines, as long as you stay under 4% and under 1%, you've had them controlled. I think both patients fit within those ADA guidelines, just to start with. The hypoglycemia we saw was asymptomatic, so they're mild or moderate and they were transient. One patient had it at week four, but it looks like it disappeared at week 12. And then same for the other patient. It looks to be transient as well.

Peter Lawson: Got you.

Thomas Butler: And also to keep in mind, we're on top of other agents, right? So, perhaps it's appropriate that these patients don't maintain their high dose metformin or if they're on a metformin SGLT2, for example. Those are things that we're trying to think about weaving into the protocol is if patients already are in normal glucose range, and if you look at the CGM for the one that had the hypo, one of the patients was at I think 90% in normal range. So, certainly for that patient, maybe they can start reducing or discontinuing their standard of care. I think that is actually what we're trying to accomplish with 219, right? Is to get folks off of therapy. And maybe these are the first signs of it.

Peter Lawson: But the hypoglycemia, was that present - I know you didn't have the baseline CGMs because of what seemed to - that they were collected over the Christmas period and there was some kind of glitches with sync in the data sets. I guess a couple of questions there. Is there any way of possibility kind of backing that out? And then for the patients that had hypoglycemia, was that also present in their initial phase CGMs?

Thomas Butler: Right, exactly. I think we have to just look back and say what does the medical history look like? If we have baseline CGM, what does that look like? But at the initial data, at least what we're seeing with the data sets that we have from week one and beyond, it looks to be transient. Not consistent. So, we'll obviously continue to monitor that.

Peter Lawson: Got you. Okay. No, that's great. The next set of data that we see, when is that and do we see the 200-milligram dose and do you have the kind of the baseline CGMs? That's kind of inbound question we keep on getting.

Thomas Butler: Yeah, exactly. I think we get that question from big pharma as well. They're excited to see the 200 milligram, and I think so far so good. We're putting all that data together now and the good news is that 219 is delivering for us. So, that mishap with the baseline was really relegated to cohort three and only for baseline. All of those issues were corrected for the other cohorts moving forward. So, that's great. We haven't seen those issues again, and it was really just something simple as moving to a receiver instead of relying on a patient's phone to get the CGM data.

And then the holiday thing, obviously, those are the holiday kinks that happened, but those have been corrected. So, we think for 200-milligram dose, an additional follow-up, that'll be sometime in the second half. We were planning to present that at ASAD, but the abstract wasn't accepted. So, we're sorting out as a company, do we just do an ad hoc call like we did with the March

presentation, or do we wait until the next conference, which could be IDF in December? We're weighing those.

Peter Lawson: Got you. Okay. But the idea there is like to have a cohesive set of data that you got baseline, et cetera, and kind of stands the scrutiny of the market and care wells, et cetera.

Thomas Butler: Yeah. And then at the end of the day, when we showed the data to probably six of the ten top KOLs in the industry, when they saw the fact that 219 while off treatment was just holding the A1C down, they were stunned. It's unprecedented, right? And so, obviously, we want to share all the supporting information behind it, the C peptide, the fasting glucose, the CGM, all those things that kind of tell the story, but the end result is unprecedented. And I think - and no question. Everyone appreciates that.

And so, for us, we're going to continue to share more data on BMF-219 and we're really excited for the expansion phase that's coming as well as getting into type ones. Now that we've shown in our poster, you get c peptide production, you get tremendous increase in HOMA beta. You can believe that we're moving into type one as soon as possible.

Peter Lawson: Got you. And I guess the pushback around type one is autoimmune disease, right? So, does that just enhance the disease by just providing more beta cells, or is there - do you think there's a trick around that immune issue?

Thomas Butler: Yeah. And it's true that obviously, you have this autoimmune component to deal with. But what we've seen in papers is that what keeps the autoimmune going is the rapid destruction and turnover of the beta cells. So, if you're to stop the turnover and preserve the pool and/or be able to proliferate the beta cells, you should be able to have a dramatic impact on c peptide production.

This is something that's never been tried before, but you can see that insulin production and insulin reestablishment kind of wards off the autoimmune component. So, we have to look at that. And then I think what's also interesting is with the advent of Teplizumab, perhaps enrolling patients who were coming off of Teplizumab would also be a great patient to see 219.

Peter Lawson: And an inbound question is, I always like them because they remind me bits I've missed. EASD, it wasn't accepted. Why?

Thomas Butler: Yeah. It's hard to say. They don't give you a reason. But when we looked back, it's the encore abstract that we submitted for ADA. So, there was no new information that was provided in the abstract. It was basically just a replicate of what we saw in the ADA abstract. And so, it just got rejected for whatever reason. They don't have a format for late breaker, otherwise, we would've just submitted the late breaker with the new data. And it's unfortunate. When we spoke to our PIs, because our PIs are on the abstract, they weren't surprised because it's just a repeat of ADA data.

Peter Lawson: Okay, got you. So, it is kind of don't want on calls at EASD versus what we saw at ADA. Thank you.

Thomas Butler: I know. Yeah.

Peter Lawson: Yeah. I guess we also potentially get like five-month follow-up data as well. And I guess most, the emphasis seems to be around the 200 milligrams, but anything else we should be kind of reading through from the five-month follow-up data.

Thomas Butler: Yeah. We'll get into a good cadence and obviously, we understand the buy side. We're going to continue sharing clinical data from COVALENT-111. We're not going to wait a

long time to do it. And so, stay tuned. We want to continue sharing COVALENT-111 data because of how well it's going.

Peter Lawson: Got you. I've got another inbound question just around, is there any correlation between the magnitude of A1C change and the amount of drug exposure, C peptide increase, or HOMA B value?

Thomas Butler: Certainly when you compare people with the same baselines, yes, we see that there is a correlation to exposure. And what I can do is point to cohort two, where on average there was lower exposure, but there were patients who had not as much as cohort three, but reasonable exposure. Those were patients who kind of did really well and caught up, so to speak. And you can see that in our waterfall plots when we look at the prognostic factors, those patients had actually really meaningful exposure.

And I think what we're seeing is the patients who are responding the quickest or show the strongest response are those who are earlier in their progression of their disease. But I think that makes good sense because they typically have a larger beta cell pool to start with. And all it means is that four weeks is probably enough time for them. Maybe six weeks is the best, but they just - they don't need that much time on drug. But for those who maybe are out to eight years or ten years, or even 14 years that we had on the study, maybe they just need eight weeks or ten weeks or 12 weeks. And that's what we're going to sort out in the expansion phase.

Peter Lawson: Got you. I know it's come up the idea of hitting menin. Some physicians are kind of worried about that whether it's like a drug coming from oncology or hitting tumor suppressors and potentially lean to neoplasms, et cetera. How do you alleviate those concerns?

Thomas Butler: Yeah, absolutely. And that was something that we looked at ourselves and looking at the biology and making sure that this made really good sense. Keep in mind that 219 is using a

natural pathway, a pathway that pregnant women use to build their pool of beta cells. And they use this during pregnancy, right? For partial disruption of menin. And that's the key word, partial disruption.

Obese people use the same exact mechanism. That's why the majority of pregnant women and the majority of obese people don't develop diabetes. They use prolactin to partially disrupt menin and allow their beta cell pool to expand. And we believe that those that do get gestational diabetes, or those obese people that eventually do develop type two diabetes just didn't properly disrupt menin, they didn't expand the pool well enough.

When you think about MEN1 tumors, what you have to understand is that MEN1 tumors come from a genetic defect, which results total loss of the gene. 219 is not a gene therapy. It's a small molecule that targets the protein from your working genes, from your non defective genes. And so, 219 disrupts impartial menin disruption, just like what happens in pregnancy and in obesity.

Peter Lawson: Got you. Okay. There's a series of inbounds. I guess one of them kind of extending from the menin inhibition and the worries about this, but is there any worry about uncontrolled cell expansion? Does the data kind of point to a feedback loop in any way?

Thomas Butler: No. So, those were - these were some of the key experiments that we did going into the study is we did cadaver experiments where we looked at BMF-219 concentration with beta islets. Here what we did was we first did mechanism of action studies, so we look at what are the genes being disrupted with BMF-219, and we did a whole panel of the reversible menin inhibitors as well, even the clinical ones and non-clinical ones.

And then we focused on what are those key genes that support beta cell proliferation? PBK, CCNA2, there's a whole suite. And that's where we saw 219 just remarkably superior moving the genes in the right direction. What we then learned is that when you create these microtissue

experiments, BMF-219 only grows beta cells. Alpha, delta, any other cell within the pancreas does not proliferate. And we only see proliferation under hyperglycemia. So, if you were to soak these islets with 219 in normal glucose concentrations, there's no proliferation. So, it's regulated by the sugar levels.

And then if you wash out 219 because the worrisome is covalent. Is that going to continue? It doesn't continue. BMF-219 has a short half-life. And so, as the protein turns over, that means that free menin is available within the day. And that helps us from a diabetes perspective. That also helps us from an oncology perspective. And so, the third final piece that we learned is that if you wash out BMF-219, even under hyperglycemic conditions with these beta cells, there's no proliferation. Proliferation does not continue unless you have continuous exposure of 219.

Peter Lawson: Got you. Another inbound question I guess off the back of this really of just like the FDA feedback you've had and at what point you discuss it on the preclinical data that they were kind of satisfied for a longer exposure, like this 12-week exposure experiment as well.

Thomas Butler: Yeah. The FDA has been phenomenal. And I think whenever you have a novel mechanism of action, you have to do a lot of work and really craft your IND. And so, they really were collaborative helping us get through that. And then they've been super collaborative in helping us make sure that we're doing all the studies we need to so that we don't delay our program. If anything, they're pushing us.

And so, when we did the 12-week, 13-week talks, they gave us a lot of guidance and it's been really helpful for us. So, we're putting all the pieces together, but the 13-week data showed exactly what we expected to show that there is no safety concern for MEN1 tumors. There is no proliferation outside of beta cells. And in the animals, because they're all in normal glucose conditions, there's no proliferation. And so, that was very supportive and very exciting and

expected internally to see. And now we're just going through the motions of putting the package together and submitting it, and we'll submit it this summer.

Peter Lawson: Got you. We're kind of at the bottom of the hour, but I'd looked to sneak in a couple of extra questions just around kind of, it's a large opportunity. Large studies have to be run. Kind of just the thoughts about funding this yourself versus through partnerships.

Thomas Butler: That's exactly right is, at the end of the day, we're sensitive to shareholder delusion and we want to make sure that we're efficient with our development, and that's why we're going to make sure that we push forward through the most efficient and we think the most impactful patient segment for BMF-219 first. And then once we're close to commerciality, then you can feather in all the additional phase threes and post-marketing requirements, right? So, that's how we're thinking about it.

And the fast path to market could come from a segment maybe the treatment-naive frontline type two going against metformin, or it could come from type one. So, we're going to enroll a small cohort of type one and see what kind of C peptide production we get to see, that could certainly accelerate 219s development path to approval. And so, we'll balance those two things and see what makes the most sense. But I think given the interest we've seen by big pharma, given the interest we've seen outside of the United States for regional deals, there's a lot of levers that we can pull. And so, you can believe that myself and Ramsey's and the rest of the team we are going to pull the right levers.

Peter Lawson: Got you. This question keeps on coming up around. Oral GLP-1s seem to be the new phase in first one, in first line. Is that something you'd want to combine with? Is it combinable with that? Those are kind of discussions you'd want to be having internally, externally?

Thomas Butler: I'm sorry, I missed the first part.

Peter Lawson: Sorry, long question, but essentially, would you combine with an oral GLP-1? Just because that seems to be like the standard almost, or the new standard for first-line therapies.

Thomas Butler: Yeah. Oral GLP-1s are coming and certainly, they don't have the same efficacy as the injectables. We all know that. And they come with a little bit extra side effects which probably is expected. But I think you hit the nail on the head because orals are just positioned first before injectables. I could imagine a fixed-dose tablet regimen of 219 and an oral GLP in one tablet. And there you could really have a dramatic effect because there you can handle some of the weight, you can handle some of the efficiency of each beta cell with the GLP and then you pull in the proliferation and preservation with 219. I think that's a powerful one, two-punch.

Peter Lawson: Got you. I know I haven't probably addressed all the questions that we're getting for diabetes but we've got a couple for AML, so I just want to kind of balance things out a little bit. The data we see for AML, is that kind of a ASH event or anything else? And how many patients should we be thinking about when we see that data set?

Thomas Butler: Yeah. For AML, and I think that created some disappointment and some confusion in the press release. And I just want to set it straight here is that the reason why we just said second half is that we didn't have - we don't want to move it again. We're very close to announcing data. It's going to happen very soon. And we just didn't want to make sure that if it didn't squeak into July or August, then we would have to say, okay, maybe Q4. We just said, let's just say second half. But AML is going really well.

When we changed the protocol to focus on the key subtypes, that's when we really turned the corner. Keep in mind that we really spent the first year, year and a half enrolling all comers as mandated by the FDA. And also we had to go through several dose escalations, right? We

started at 100 milligram. We should have started probably at 300, 325 because at 500 we're seeing really strong activity. So, we have to see how does that strong activity translate into response? And then as we go from 500 to 325 BID, what are the benefits sitting there? What does the durability look like? And so, we're putting all those pieces together. And like we said before, we want to share escalation data so about ten patients in escalation. Then we want to see ten patients at the - of the key subtypes, whether it's NPM1 or MLLr.

Peter Lawson: That's ten patients of each of those MLLr, NPM1.

Thomas Butler: Most likely total. NPM1 or MLLr ten patients.

Peter Lawson: Perfect. Okay. Thank you. And do you think with - at that point, we'd see the recommended phase two dose as well and data from that recommended phase two dose?

Thomas Butler: Yes.

Peter Lawson: And it sounds like it's looming, which is always encouraging and it's going to - and I assume it's going to be a company press release and a call as opposed to being presented at a conference, right?

Thomas Butler: That's exactly right.

Peter Lawson: And just another question that we keep on getting, are you enrolling patients that are post-SNDX, post KURA or all the other kind of menin out there post other menin?

Thomas Butler: Yeah, absolutely. So, we did our work, so we opened up the protocol to accept prior menin inhibition. We haven't got one yet. All of the patients that we've been dosing are pre-menin experience, so they haven't seen a menin inhibitor yet. But we hope within the next

several months that we'll hope to get at least one or two that have seen prior menin experience or have seen a prior menin inhibitor and see how 219 performs in those patients.

Because if you think about it, if they didn't receive a response or they didn't tolerate, 219 is a great option here because we're not as sensitive to the other - to the MLL protein Fusion partner. And that's why we scrapped the original program, as I mentioned earlier on the call, is we're really dependent on menin and we're focusing on menin. And we're independent of the Fusion partner. And so, we won't see those sensitivities that the others see.

Peter Lawson: The NPM1 and rearrange patients, would they be at the target dose levels?

Thomas Butler: Yes. When we share the data?

Peter Lawson: Yeah, exactly. So, we get ten patients at the target dose level for NPM1 and MLLR rearranged.

Thomas Butler: And that's why we're waiting, right? We want to make sure that we're not putting forth data at sub-R exposure. We always mentioned even back when we IPOd that 2,000 AUC is our target. And for the first time when we finally got to 500 milligrams, we're hitting that target. Keep in mind that 500 milligrams is for the patients that are not on a SIP inhibitor. Those that are on a SIP inhibitor, the dose is obviously much lower; 175.

Peter Lawson: Okay. Wait, you have the - your drugs metabolized through the CYP3A4 pathway?

Thomas Butler: It is, yes. It's a CYP3A4 substrate.

Peter Lawson: Okay. So, you have this kind of similar to SNDX CYP3A84 high and low kind of thing.

Thomas Butler: Yes. That's right.

Peter Lawson: Okay. And I apologize, we ran through extra time when I get into penalty shootouts, so thank you so much for the time.

Thomas Butler: No problem. It's a great call.

Peter Lawson: We appreciate all the time.

Thomas Butler: Yeah, absolutely. Thank you so much.

Peter Lawson: With that, I'd love to close the call. And tonight, we actually got a dinner with Relay and we've got rep room for a fireside chat tomorrow. So, thank you so much.

Thomas Butler: Thank you. Appreciate it.

Peter Lawson: Take care.