Company Name: Climb Bio, Inc. (CYLM)
Event: Stifel 2024 Healthcare Conference
Date: November 18, 2024

<<Analyst, Stifel Financial Corp.>>

Good afternoon, everyone. It's my pleasure to introduce Climb Bio. Aoife, Bill and Brett here with us this afternoon. So maybe before we get into some Q&A, Aoife, do you want to give a quick overview of the company and then we'll do a fireside?

<<Aoife Brennan, President and Chief Executive Officer>>

Yeah, my pleasure. Hi, everyone, my name is Aoife Brennan. I'm the CEO. Thanks, Alex, and the team for the invite here. We're a relatively new company. Eliem has been around for a couple of years, but we're a rebrand. The company Eliem, was originally a neuroscience company. The company has pivoted since July of this year to focus on INI and with the acquisition of Tenet Medicines. Now the company has rebranded since October 1st as Climb Bio.

We are formed around a very interesting clinical phase asset called budoprutug, which will be the focus of a lot of the Q&A here. But just wanted to give you a little bit of that background because given the ticker change recently, there's been some kind of confusion around the Eliem-Climb story. But going forward, the focus of the company will be on the development of budoprutug, which is a CD19 B cell depleting asset that we're developing in a number of B cell mediated diseases.

It's an asset that we acquired as part of an acquisition of Tenet Medicines. Bill was one of the founders of Tenet and we'll give you a little bit of background around why we're so excited about the compelling data that we have around that asset. It's part of a very exciting class of medicines focused on B cell mediated diseases. The three indications that we're pursuing are membranous nephropathy, which is a rare kidney disease, an ITP, which is a B cell mediated hematological condition, and then systemic lupus erythematosus, which is a less rare systemic rheumatological condition. All three diseases associated with high unmet need. And we're moving forward in clinical trials in all three.

<<Analyst, Stifel Financial Corp.>>

Great. Yeah. And maybe to kick things off, this is obviously an incredibly competitive emerging space, really right now in autoimmune. But why really is B cell depletion kind of such a powerful strategy to begin with? And then we'll get into kind of the specific nuances.

<<Aoife Brennan, President and Chief Executive Officer>>

Yeah. So B cells are really at the heart of a lot of diseases where there's an antibody mediated component to the disease pathophysiology. So historically, a lot of these diseases have been

really managed in a downstream way by suppressing the immune response using predominantly steroids. So these patients are often managed by chronic immunosuppression with chronic high dose steroids, which are associated with a tremendous burden for patients because there are many long term consequences associated with chronic high dose steroid administration.

There's been a lot of data in the past, I would say maybe year or two, demonstrating that B cell depletion can really get to the underlying pathophysiology of these diseases. It can be used chronically to maintain remission in these diseases. And there's also increasing evidence that it may be possible to induce "immune reset". And I think that's the kind of new piece of data that's really driving tremendous excitement in this space.

And the concept there is that you can do short-term treatment to result in long-term benefit for these patients so that if you deplete these pathogenic B cells and if you deplete deeply enough, you can potentially give these patients a long-term disease remission. So you can deplete all of the disease causing B cells in patients and then kind of reset their immune system essentially so that when the B cells come back, their immune system is reconstituted and they no longer have that disease. So they get to kind of return to a normal B cell or reconstitute their immune system without that kind of bad actor B cell subset.

So, together the kind of chronic B cell depletion being a good and relatively safe strategy for long term maintenance of remission, as well as this emerging evidence that you can kind of do this immune reset has really created a tremendous amount of interest and excitement in the space. And ACR was this weekend where we saw some additional data and that's kind of going to continue to drive excitement, I think around the space.

<<Analyst, Stifel Financial Corp.>>

Yeah, for sure. And I think coming out of ACR, obviously one of the big topics of discussion, after thinking about B cell depletion is just the target specifically. So CD19, CD20, BCMA, CD38, what are kind of the pros and cons of each of those types of targets? And why do you feel like CD19 is the path forward?

<<Aoife Brennan, President and Chief Executive Officer>>

Yes. So for those of you who are not don't live and breathe B cell biology, it's a very complicated area. And I think there's two components to the Climb Bio story. The first component is what's the right target? And then the second component is, what's the right modality? And I think the question that Alex is asking about is what's the right target? And there's multiple cell surface kind of proteins that are expressed at different parts of the B cell lineage. And there's also kind of overlap in terms of some of those cell surface markers are also expressed on other cells.

So we fundamentally believe that CD19 is a very, very exciting marker and is the better marker when it comes to B cell depletion, because of its expression profile. It's expressed from the very early stages of B cell development right up to the plasma blast and early plasma cell development. You do not want to deplete plasma cells. That's where BCMA is. Because if you

deplete plasma cells, you're getting rid of humoral immunity. We all received vaccination early when we were, most of us did when we were born. Kind of a hot topic these days. So maybe I won't get political. Most of us receive vaccination. If you deplete your plasma cells, you're going to lose all your long-term humoral immunity, right? So you're going to basically wipe out all of your protective immunity. So you don't want to do that.

So the CD19 actually has a really interesting profile, in terms of when you deplete B cells that express CD19, you're getting this really nice profile where you're getting like almost all of the B cells, but you're still sparing those long lived plasma cells that are associated with humoral immunity.

<<Analyst, Stifel Financial Corp.>>

Yeah.

<<Aoife Brennan, President and Chief Executive Officer>>

CD20 is expressed in a narrower subset of cells. Obviously rituximab has been around for a long time. It has been associated with efficacy in some autoimmune diseases, but it's expressed in a narrower subset of diseases. So it's not surprising that while it's efficacious, many patients fail to respond or have a relapse pretty quickly after they have that initial response, because it's not really getting into the plasma blast compartment. And I think that's kind of a limitation of CD20.

And then, we've spoken about BCMAs expressed on those plasma cells. So it's kind of going to get into that long lived plasma cell compartment and interfere with humoral immunity. And then CD38 is an interesting target, but is also expressed in plasma cells and is also expressed in neutrophils and some other cell types. So, each target has pros and cons in terms of its expression profile. And it may be potentially worth thinking about kind of benefit risk.

If you have multiple myeloma, for instance, BCMA may be a great target for you. But in an autoimmune patient, maybe the benefit risk there is not so favorable. So that's how we think about that.

<<William Bonificio, Interim Chief Business Officer>>

I would add – excuse me, I would add one way to measure this risk would be certainly infection risk. And we have a lot of experience with anti-CD19 therapies. There's an approved anti-CD19 called the inebilizumab. And of course there's a lot of experience in the anti-CD20s rituximab. They generally don't have a dramatic increase in infection. It does not increase over time. We have long-term follow up now on other therapies. It does not necessarily increase over time.

So the safety is actually very clean with these drugs and not a major concern. Whereas now it's just a question of how hard can push on that efficacy. But that clean safety profile is really an advantage not only compared to the CD38s or the BCMAs, but also these other modalities that we're going to talk about. I'm sure momentarily around the T cell engagers and the CAR T's

right. In terms of a safety window, CD19 monoclonal antibodies certainly among the best you can do.

<<Analyst, Stifel Financial Corp.>>

Yeah. And I think to that point, obviously what we're trying to see in conferences like ACR et cetera is, just how do you think about the progression of these modalities, CAR T, TCE, antibody from oncology to autoimmune. Is there an association between breadth and depth of response in terms of like broader tissue distribution, et cetera with some of these other modalities and efficacy, like will the antibody be able to live up to the type of immune reset that you're alluding to as the potential in autoimmune?

<<William Bonificio, Interim Chief Business Officer>>

Yeah. I think this is the promise of CAR T is that it's a one and done immune reset, where you don't have to dose this chronically. And so far they've had some really nice signs of the ability to do that from early academic studies, but have really been unable to recapitulate that. Now in a broader setting, in the industry setting where they're having relapses or having recurrence of disease after initial efficacy, which is okay as B cells come back. So then you go into TCEs, which have the promise of maybe being a little safer, can dose chronically, but those also have an associated cytokine release syndrome ICANs as well. And nobody has really been able to show a safe CD19 TCE to date.

Even a competitor of ours called [indiscernible] (09:49:00) is seeing CRS even at their lowest doses without the associated B cell depletion necessary in these diseases. And so that leaves you with an antibody which maybe, you would think, maybe conventionalism would suggest is not able to get the kind of deployment depletion needed or the durability of depletion as it compares to the CAR T's and the TCs. But in fact the antibodies do get full B cell depletion. As you measure circulating CD20 cells, they get full B cell depletion for a long time for say six months after just a single or two doses of the antibody. And you can dose that repeatedly as needed.

In terms of efficacy, we're seeing with our drug and with a competitor, inebilizumab, tremendous efficacy in a variety of settings right now. So inebilizumab, of course, is approved in NMOSD. It has positive Phase 3 data now in IgG4-RD and myasthenia gravis. And all again with just the naked map. And in our hands we have positive data in membranous nephropathy, that rare renal nephropathy, and a single patient in the disease called minimal change disease. Now, you talk about immune reset. We're actually seeing it with the antibody in our hands here with the anti-CD19 antibody.

In our MN population we have a single patient. We had a small data set, but we have a single patient. We've been able to follow out now two and a half years since initial dosing, that single patient entered into a complete remission of disease. They came off of all their concomitant medications. They're essentially, in the words of the treating physician, they're essentially cured of this disease. And the same with the minimal change disease subject now who we also have two years follow up now since initial dosing. That patient is also completely off of all steroids. Anything else needed, that patient is essentially cured. And so we see that, we do see this

immune reset with the anti-CD19 antibodies and we expect we'll see it, as we enter into new patient populations and as we get more data from inebilizumab as well, that the CD19 antibodies are, can do the job, are as good as the CAR T's and the T cell engages without any of the safety and tolerability issues.

<<Analyst, Stifel Financial Corp.>>

So for budoprutug…

<<Aoife Brennan, President and Chief Executive Officer>>

Budog.

<Analyst, Stifel Financial Corp.>>

Budog, so like how is your antibody differentiated from UPLIZNA? And I guess also, I think you could probably sneeze and another TCE will fall off the shelf somewhere.

<<William Bonificio, Interim Chief Business Officer>>

Yeah, yeah, yeah.

<Analyst, Stifel Financial Corp.>>

But why are there not more CD19 antibodies out there right now.

<<William Bonificio, Interim Chief Business Officer>>

Yeah, I mean there's a – I'll answer the second question first. There's a moat in developing any of these and that's that B cell depletion inherently is challenging to do with a healthy volunteer because it is depleting a cell type. And so you often have to dose escalate in an oncology setting. And that's what most of these TCEs are doing as well. And so it's difficult to enter. Right. And for a while the antibodies, the CD19 antibodies were kind of not as much attention as the CAR T's should being approved.

So this drug itself, our budoprutug was developed by Merck Serono originally in oncology. And it has some really nice benefits, or rather on paper advantages compared to inebilizumab. For one, on paper it has better B cell killing properties. Right. So the EC50 in terms of the ability to deplete B cells is about five to 10 times better for budoprutug compared to inebilizumab in a cross lab comparison. In addition, we believe based on our modeling, have a slightly longer half life than inebilizumab as well. But again, both of these drugs are going to do a really good job of depleting antibodies.

The major kind of differentiating factor is that we intend to go subcutaneous with our drug. We have the ability to formulate up to 175 mgs/mil, which should afford us a 2 ml therapeutic dose in our patients. Inebilizumab, on the other hand, was unable to – they've tried dosing SC in an

early study they were unable to match the exposure profile from the intravenous dosing. And so they've kind of shelved that. We don't really know exactly what they're doing, but for now it's just intravenously dosed.

So yeah, we do believe that has some inherent advantages and we've seen it play out. Right. We know in our MN study we had doses that were say 200 milligrams, which is lower than the label dose of inebilizumab lead to full B cell depletion in those patients. Even at 100 milligram dose, we had full B cell depletion in our patients, again a lower dose than what inebilizumab uses. And in that setting we really put up data in those patients that was really the best data that we generated in MN to date. So we had 60% of our patients reach a complete remission at one U.S. time.

<Analyst, Stifel Financial Corp.>>

And I think one of the interesting parts, but also challenges with this space is just the number of potential indications you could target. And I think that your framework for thinking about indications is super interesting. Can you walk us through kind of the three sort of types of indications that you're looking at and why you've selected that particular lead indication in each one of those?

<<Aoife Brennan, President and Chief Executive Officer>>

Yeah. So we felt it was important to kind of have a rational approach, because the space is so broad. So we came up with this three kind of bucket or three pillar framework. The first one is IgG4-mediated autoimmune diseases. That's a subgroup of autoimmune diseases where the pathogenic antibody is an IgG4 isotype. And we believe that the biological risk there is very low, because those IgG4 secreting B cells all express CD19. And we know that from multiple lines of evidence that a B cell deplete will work particularly well in those diseases and those indications. pMN is a good example of that. We've seen some very compelling data.

We know that UPLIZNA works very well in IgG4-RD and there was some data that confirms that released over the weekend. So we think the biology risk is the probability of success is particularly high when the pathogenic antibodies and IgG4 isotopes and there are a number of different diseases where the disease is driven by an IgG4 antibody. So that's kind of the first pillar, if you will.

The second are where the antibody is an IgG4 1 to 3, but they're single organ diseases like ITP, CIDP, MG. These are kind of rare single pathogenic antibody, often a single target organ that's been implicated, pretty clear regulatory paths often being de-risked by other assets in development. We like those indications for a small company perspective. Often they're clear kind of path to registration, approval.

And then the final bucket are these complex multisystem rheumatological conditions like systemic sclerosis, SLE, rheumatoid arthritis. Often these are more complex diseases, but more common, much bigger commercial opportunities, often kind of commercially validated because they're easier to model. There's good data in terms of pricing, prevalence, the commercial

opportunity is more clearly understood and a bigger opportunity from a commercial perspective. And there SLE is a good example of that. And we're currently undertaking a study in SLE. We just announced that our IND is cleared.

So that's kind of how we think about it from a framework perspective, and we have one study underway in each of those three indications with multiple others coming along behind that can be kind of unlocked once we understand the dosing and the biology and the kind of lead from each of dose. And I think it's a really kind of good way for us to think and rationalize the very broad opportunity set across the space.

<<Analyst, Stifel Financial Corp.>>

Yeah. And Bill, I think you alluded to some of the data in membranous nephropathy. Can you talk a little about that study design and the data a little bit more in more detail?

<<William Bonificio, Interim Chief Business Officer>>

Yeah, yeah, for sure. So we embarked on this study because we thought it was a terrific indication to study because it has some really nice biomarkers. And the proteinuria that is seen in these patients is the reduction that proteinuria is the endpoint for approval. So yeah, so the study design was such that we dose patients four times. So we dosed at weeks zero and two and then six months later at weeks 24 and 26. And we measured B cell depletion. We measured the autoantibody PLA2R, so about 70% of patients that have this autoantibody PLA2R, which targets a glomerular antigen.

And then we also measure proteinuria. And again, our goal in this therapy was to get to complete remission of proteinuria. And these complete remission of proteinuria in these patients. So less than, say, 0.3 grams per gram of proteinuria. So we measure – we studied five patients. We had five patients with data out to a year. And in those patients, all five achieved rapid and durable B cell depletion. So two of the patients had some B cells coming back between weeks 12 and 24. But then on the second course of therapy, at weeks 24 and 26, they were fully depleted. So we had B cell depletion in all patients. In three of the patients who were PLA2R positive, all three had a serological remission where their PLA2R went negative.

And then again, in those five patients we had three out of five are 60% had complete remissions. Five out of five had partial remissions. The two patients that didn't were doing very well. At the end of the study, one patient just barely missed that CR mark and I mentioned that one patient we have even more follow up on, I would say two and a half years now is still in complete remission. So that 60% number is again the best data that have been generated in this disease. And we can compare it kind of directly to other B cell targeting agents in this space. So CD20s with rituximab and Gazyva both showed around a 14% complete remission rate at one year. CD38 felzartamab was studied in this setting, had zero CRs at one year.

You can look at the BAFF/APRIL, so metastasis. I've just had data from Vertex at ASN where they had two out of three patients had a partial remission. And you know the total, the percent of – proteinuria reduction looks to be higher with budoprutug.

And then finally the FcRns were studied here and Argenx was studying this and they recently discontinued the study. So ostensibly we believe it must have failed in this indication. So right now we're feeling pretty good about the place of an anti-CD19 antibody in this setting and we will continue to study here and looking towards late-stage studies next year.

<<Analyst, Stifel Financial Corp.>>

So what are the next steps then for moving forward?

<<William Bonificio, Interim Chief Business Officer>>

Yeah. Brett, do you want to give the formal guidance…

<<Brett Kaplan, Chief Operating Officer>>

From the pMN side? What we've guided to is moving into late stage clinical trials in 2025.

<<Analyst, Stifel Financial Corp.>>

Okay. And then I think you looked at minimal change disease too, right? So that was one patient. Is that an indication that you are going to look to move forward in or is that still TBD?

<<William Bonificio, Interim Chief Business Officer>>

Yeah. We're pretty excited about this indication. In general, it's another rare renal glomerular nephropathy. Same treating physicians as the MN, also very likely autoantibody driven given what we know about the disease and even more recently a kind of identified auto-antibody here. Similarly, we expect to see rapid proteinuria reductions and that proteinuria would be the endpoint for approval. And like I mentioned earlier, we did study one patient who had a dramatic improvement and is now in complete remission even off of drug for two years since the initial dosing.

We've not necessarily disclosed our exact plans for minimal change disease. It is thought to be rarer than MN. And so I think we're not saying much now about our plans there. But of course, with this drug we can do a lot of things with it. We do intend to expand the pipeline at some point and that will certainly be in consideration.

<<Analyst, Stifel Financial Corp.>>

And I guess other indications here. So you talked about SLE, probably the most competitive of all the CD19 indications. Is that fair to say at this point?

<<Aoife Brennan, President and Chief Executive Officer>>

I think so for sure. There's a lot of other modalities I would say with targeting CD19 in SLE that is likely to change over the next two years. So I think it'll be interesting to see how that gets winnowed or expands. I think a lot will change in the next 18 months as the trials progress. So we're absolutely watching the space and. But I think as of today, it's fair to say very, very crowded space.

<<Analyst, Stifel Financial Corp.>>

So then he talked about your proof-of-concept trial that, you're going to be running here and kind of what you want to see. Just what the thesis here differentiation?

<<Aoife Brennan, President and Chief Executive Officer>>

Yeah. So I think the key question for us that we plan to answer in this next study is, can we achieve something with a naked antibody that's similar to what we're seeing with the CAR T in terms of being able to achieve this immune reset? Because I think that's a key question that everybody wants to know. Because if we can achieve something where we're able to achieve very deep B cell depletion such that when the B cells come back, they're coming back in that naïve phenotype and even over the weekend, we're seeing more and more of the biomarker data around what shifts in the immune system are associated with this kind of – I'm using air quotes, right, because the immune reset, we still don't really understand it as a clinical concept.

<<Analyst, Stifel Financial Corp.>>

It's sound like a real thing, but it's not…

<<Aoife Brennan, President and Chief Executive Officer>>

It's not really a real thing. So we still don't. There's no real clinical definition of autoimmune reset means, but there does appear to be a collection of biomarkers in terms of changes in the immune system that are associated with this long-term remission, right in these patients with SLE, which I think is just clinically, it's really important. These are young women most of the time with a high burden of disease. If you can give them even a couple of years, free from heavy duty immunosuppressants, I think that's very, very clinically relevant to these patients.

So we're starting to understand more and more about what that biomarker looks like? What that signature looks like? And I think the question is, can we achieve that with the naked antibody? And if the answer to that is yes, I think that sends us down one path. If the answer to that is no, there still may be an approach, it's maybe a chronic dosing approach. Right. Where it looks more like, how some other monoclonal antibodies are used in the management long term of an SLE. Obviously it's less kind of compelling. Right. It's a different development path. It's more kind of chronic B cell suppression, which I think could still have a very important place to play. But it's a different Phase 2 design compared to something that's pursuing kind of an immune reset path.

So I think that's what we really need to answer with this study that we've just initiated. And I think it'll be very exciting to see the data when it starts to come in.

<<Analyst, Stifel Financial Corp.>>

So with all the competition here, how has – how have conversations gone with investigators with sites? Is it going to be a hard trial to enroll? Or is it feedback been good so far?

<<Aoife Brennan, President and Chief Executive Officer>>

Anyone who tells you SLE is an easy indication doesn't know what they're talking about. So that's the first thing. Right. But I will say that we do have an advantage in that being a naked antibody. It's something that's very familiar to sites right like you, you know infliximab, we're just like that. So it's doing the T cell and the CAR T therapies are limited to a certain type of infrastructure that you need to have in place to do those studies, similarly with T cell engagers. So we actually have kind of a broader set of sites that we can pursue. So, we're looking at a broader subset of sites to engage.

But, finding these patients, getting them enrolled in studies. We are pursuing patients who have severe disease that has not responded to, you know, currently available therapeutics. So it is challenging to find those patients. But I do think we have some tailwind and some things in our favor, given that we don't necessarily have to go to those few kind of academic centers that are like kind of being bombarded by every single cell therapy company and NK cell therapy company and CCE company, because that's what you're seeing. Right. And we're avoiding those centers because they're just completely backlogged right now with trials. And we're trying to kind of work around those centers, which I think is something that's slightly in our favor compared to some of the other competitors that are in the space.

<<Analyst, Stifel Financial Corp.>>

Then moving on to sort of single organ. Can you talk about the rationale for ITP as the lead here?

<<Aoife Brennan, President and Chief Executive Officer>>

Well, number one, I think it's an indication where there is an unmet need. And sometimes that comes with a raised eyebrow because there have been some product…

<<Analyst, Stifel Financial Corp.>>

I totally agree. It's why would you use the same drug multiple times?

<<Aoife Brennan, President and Chief Executive Officer>>

Exactly. And I think some of the other products that have launched in the last year or two haven't necessarily gotten off to a great start from a commercial launch perspective. But when you understand the mechanism of action and the data, that's not surprising. But when you look at, there's a lot understood about ITP from pathophysiology perspective because there's a lot of tissue available, because these patients, when they don't respond, get their spleen taken out. So

you can look at what's happening in terms of the tissue level. And what you see when you look at those splenectomy samples is in patients post-rituximab who have not responded. It's not that rituximab hasn't worked.

Their CD20 cells are gone, but their spleens are full of CD19 positive cells that are still producing the pathogenic antibody. So I think there's really good biological rationale to support that CD19 depleting antibody could work in kind of a post-rituximab setting and could potentially even displace rituximab as first-line if we can put up a good number. So if you can offer a patient presenting with ITP today potential to have a much higher chance of having response and a much greater chance of maintaining clinical remission in three years time. I think that's going to be, over time, the preferred first line agent. Right. And that's what you're seeing with, NMOSD, for instance, where rituximab had been used previously, where an UPLIZNA has recently been launched, and you're seeing it displacing, right, the inferior kind of previously used B cell depleter.

We don't know yet whether obviously we're going to work or whether we're going to put up the right kind of numbers to support that. But I do think there is space for that kind of a complete transformation of the therapeutic paradigm in ITP. And that's what we're aiming to demonstrate with our study that we're getting going. So we'll know.

<<William Bonificio, Interim Chief Business Officer>>

We do have some confidence in this approach in patients with ITP who receive rituximab, often when they fail, they'll have splenectomy and they can actually physicians have studied those spleens and seen that the population of antibody secreting cells is the CD19 positive. Right. And so presumably if you can deplete those cell types, which we think we can, it should eliminate those antibody secreting cells, eliminate the antibodies, involute the germinal centers, et cetera, and treat that, treat those patients. So we have some confidence going in that a CD19 targeting agent will be a best-in-class therapy here as well or best-in-disease therapy. We'll have to find out.

<<Analyst, Stifel Financial Corp.>>

And that trial has now started your CD19 – and what's the timing?

<<Aoife Brennan, President and Chief Executive Officer>>

So the guidance is we'll dose our first patient in the first half of next year.

<<Analyst, Stifel Financial Corp.>>

Okay, great. So maybe you'd wrap things up. Current cash runway and embedded assumptions around that.

<<Brett Kaplan, Chief Operating Officer>>

Yeah. So what we've guided to is, we have around $218 million. This lasts us through 2027. We've spoken about the three programs, ITP, SLE and MN that are all funded through in those timeframes with the – with guidance towards having first patients in the first half for SLE and ITP and advancing the MN program that we referenced earlier. What we haven't spoken about is the subQ formulation we're advancing that should have non clinical data next year and we'll be moving into clinical studies soon thereafter. So lots going on, eventful year, we're well funded, doesn't mean we don't want more cash. But we just got to find the right time.

<<Analyst, Stifel Financial Corp.>>

So all these studies will be IV and then presumably next studies would ideally be subQ.

<<Brett Kaplan, Chief Operating Officer>>

Got to work through the strategic and the commercial rationale there. Yeah.

<<Analyst, Stifel Financial Corp.>>

Great. Awesome. Well, thank you all for joining us.

<<Aoife Brennan, President and Chief Executive Officer>>

Thank you so much.

<<William Bonificio, Interim Chief Business Officer>>

Thank you.