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EDITED TRANSCRIPT

REGN.OQ - Regeneron Pharmaceuticals Inc at RBC Capital Markets Global Healthcare Conference

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OVERVIEW:

Company Summary

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CORPORATE PARTICIPANTS

George Yancopoulos Regeneron Pharmaceuticals Inc - Board co-Chair, President, Chief Scientific Officer

Mark Hudson Regeneron Pharmaceuticals Inc - Senior Director of Investor Relations

CONFERENCE CALL PARTICIPANTS

Brian Abrahams RBC Capital Markets - Senior Biotech Analyst

PRESENTATION

Brian Abrahams - RBC Capital Markets - Senior Biotech Analyst

Okay, hi, everyone. I'm Brian Abrahams, one of the Senior Biotech Analysts at RBC Capital Markets. Our next featured company, Regeneron, has always been known for its R&D engine, and there's a lot more coming from their pipeline, really over the next 6 to 12 months and beyond. And the architect of that has been their Board co-Chair, President, and CSO, George Yancopoulos. So we're really pleased to have George with us today, and alongside him on the stage, Mark Hudson, Senior Director of IR for Regeneron. So thanks again for joining us and looking forward to the discussion.

George Yancopoulos - Regeneron Pharmaceuticals Inc - Board co-Chair, President, Chief Scientific Officer

Appreciate you having us.

Mark Hudson - Regeneron Pharmaceuticals Inc - Senior Director of Investor Relations

And before we get started, obviously have to read a forward-looking statement, otherwise my legal folks will get mad at me. So I'd like to remind you that remarks made today may include forward-looking statements about Regeneron and each forward-looking statement is subject to risk and uncertainties that could cause actual results and events to differ materially from those projected such statement. The description of material risk and uncertainties can be found in Regeneron's SEC filings. Regeneron does not undertake any obligation to update any forward-looking statement, whether as a result of new information, future events or otherwise. And it's a pleasure to be here, Brian, and we're glad, we have George with us, so we'll get right to Q&A.

QUESTIONS AND ANSWERS

Brian Abrahams - RBC Capital Markets - Senior Biotech Analyst

Thanks. So why don't we start with the next upcoming catalyst, Phase 3 data for itepekimab in in COPD. Can you tell us what you've learned from the prior data in asthma and COPD about the IL33 mechanism, and how should we be thinking about some of the analyses you've done so far? You mentioned an interim analysis, that's been done halfway through this ongoing study. In your view, what does that tell you about -- how does that inform you about the potential outcome of the study? What are you looking for out of it?

George Yancopoulos - Regeneron Pharmaceuticals Inc - Board co-Chair, President, Chief Scientific Officer

Well, maybe the first point to make is that I think a lot of people, just assume that all the antibodies, all antibodies against a particular target, they're all created equally. I think the important point to make is even in today's world. that's not true. You look at many of our most important medicines. Eylea, Dupilumab, everybody else tried to make an IL4 receptor antibody, including the biggest companies in the world like Amgen. They went to clinical trials, they failed in every single Phase 3 trial. We succeeded in 8 out of 8 Phase 3 trials, which is probably a record. And what -- why, and it

comes down to technology and it comes to understanding biology and it comes down to having the technologies that can maybe predict which setting and why.

So what I think that we've shown over the years that whether it's with soluble receptors like EYLEA or whether it's with antibodies like Dupilumab or whether it's bispecifics, we continually to have the best-in-class type molecules that have and deliver the best data. Why? Because we spend a lot of time in the labs, creating the technology to give us the best antibodies, but then we also have these incredible technologies that allow us to choose not between most people they make one antibody, they're happy and they go with it.

Okay, we don't make dozens of antibodies, we don't make hundreds of antibodies. We literally screen millions of antibodies. Because we've created, we were the first people to develop a lot of these technologies, so we've created these incredible high throughput technologies. So it starts with the molecule. All molecules, all IL33 blockers, all IL4 blockers, all VEGF blockers, they're not all created equally. We think we have the best molecule here. We tested it against every existing agent that's out there, that's number one.

Number two, as I said, why is it that we've hit on 8 out of 8 Phase 3 trials for Dupixent? Well, we have a cheat code. We built the world's largest DNA sequence-based, big data set on the planet where we can see how genetic variation can impact disease variation and nobody else has the ability and these tools because they didn't invest in the time and the effort and the dollars to build such a resource. So as we've shown for example for Dupixent, we have the genetics that shows that if you have up activation of the IL4/13 pathway that promotes certain diseases, and you're protected if you have the genetics that show that you have low tone in that genetic pathway. Well, we did the same thing with IL33.

So one is you start, you create the best possible antibody, screening millions of antibodies using the world's best technologies, and then you use the genetics database that we have that other people don't have to tell us which diseases, can this really make an impact in. And we've shown a lot of this, publicly, the genetics are really pretty strong that in certain diseases asthma, chronic rhinitis with nasal polyps and COPD that the genetics suggests that too much of this pathway is associated with more of this disease, less of this genetic pathway, less of this disease. And so, these are things that we validate over time that these genetic pathway scores are really good predictors of where you should go with your best-in-class molecules.

And so, we've done some of the early studies as you mentioned our asthma data, if you look across the field, other people's molecules that they're going forward in COPD actually failed to show any sort of benefit in asthma. We had pretty much Dupi-like data in asthma. And now, in COPD, we've focused on a different subset of patients where the data suggests us that it could make a difference for these patients and a different subset of patients and the patients that are currently benefiting from Dupixent in COPD.

Brian Abrahams - RBC Capital Markets - Senior Biotech Analyst

And tell us a little bit more about that subset. I know it's a little bit different, maybe there's a little overlap, but it's generally different from what is Dupi is now approved for. What would you view as compelling data just given the lack of options for low eosinophils patients? What are you hoping to see?

George Yancopoulos - Regeneron Pharmaceuticals Inc - Board co-Chair, President, Chief Scientific Officer

I think, as you said, I mean, clearly Dupi has changed the field for patients with eosinophilic COPD, with over 30% reductions in exacerbations that's really, it's unprecedented. No other molecule has delivered anything close to that. We think that in this unmet need population with the low eosinophils, we believe that if we if we hit 20% decrease in exacerbations, that can make a really big difference for these patients that really right now, have very few new kinds of treatments available to them for years.

Brian Abrahams - RBC Capital Markets - Senior Biotech Analyst

We talked about antibody design, this discovery, leveraging the genetic databases as well, to what extent does the additional information from the interim analysis further support your confidence in in success here?

George Yancopoulos - Regeneron Pharmaceuticals Inc - Board co-Chair, President, Chief Scientific Officer

I think that we haven't disclosed the numbers and so forth, but we basically announced that we had included into the program an interim efficacy analysis which if we didn't need it, then we would not go forward. And certainly, it gives us more confidence since we met that hurdle, that there's increased chances that the trial would work. If we didn't meet it, we'd have much less chance that the trial is going to work.

Brian Abrahams - RBC Capital Markets - Senior Biotech Analyst

Well, looking forward to those data.

George Yancopoulos - Regeneron Pharmaceuticals Inc - Board co-Chair, President, Chief Scientific Officer

So are we. Yes.

Brian Abrahams - RBC Capital Markets - Senior Biotech Analyst

On the oncology front, a lot of efforts there as well. You obviously have antibody expertise as well as Libtayo as a strong backbone to build around. You've had some early successes like Linvoseltamab and at least so far and with Fianlimab. But you've also faced some challenges as well, such as with the PSMA bispecific. Can you talk about which of the oncology assets you're most excited about these days and what approaches are going to be of highest focus over the next few years in the cancer space?

George Yancopoulos - Regeneron Pharmaceuticals Inc - Board co-Chair, President, Chief Scientific Officer

Yeah. Well, I'm excited about the breadth across the portfolio, I think that that's what makes it exciting. Once again, it starts with the best molecules. I mean, you mentioned Libtayo, our PD-1 blocker, as you guys probably know. I mean, there's so many PD-1s out there, there's only 2 that have met the high bar for efficacy in first line lung cancer. Libtayo and Keytruda and arguably, our data now, our long-term data looks like it's best-in-class. It's still hard to make the best antibodies. I don't care where you're making them, the data just says, right now, there's only two PD-1s that are approved broadly across the first line lung cancer setting.

Well, and we've now, for example, shown settings now. For example, an adjuvant cutaneous squamous cell carcinoma where ours is the only molecule that works, where for example Keytruda failed. So it's important to start with the best molecules. Same thing you mentioned our BCMA bispecific for myeloma. We have double the complete response rates of the two competitors out there, and that's what it's all about, getting complete responses and remissions.

And so, once again, you start with the best molecules but then you also have to figure out creative ways in which to employ them and where to go, and there, again, we're excited across our portfolio. We're excited that we have what we think are two of the best-in-class checkpoint inhibitors we're combining our PD-1 Libtayo with our LAG-3 Fianlimab, and we're going to be getting Phase 3 data in first line metastatic melanoma in the second half of this year. I think that that could really make a big difference for patients.

I mean we've been -- we, the field, has been looking for advances by combining checkpoint inhibitors since the first checkpoint inhibitors were approved more than 15 years ago and nobody has yet succeeded. We think this may be the best shot for getting both increased efficacy without dramatically promoting safety issues, so we're excited about that. But I mentioned we also think we have the best-in-class bispecifics they've been validated in terms of producing very impressive data in the last lines of both myeloma or lymphoma, for example. But what we're very excited about is moving it into the earlier line settings.

We've recently released and we're going to be talking at ASCO a lot more about how these agents are behaving in earlier lines as monotherapies or as in very restricted combinations. And we think that they have the real opportunity to completely change the field if you guys know the treatment

paradigm for myeloma and lymphoma, these are very tough, complicated regimens where you're putting together many toxic molecules to try to get efficacy and the data suggests that our bispecific either alone or with dual combinations can actually produce better data, and hopefully, more safely in the in the long run. So moving our bispecifics to earlier stages even moving to pre-malignant disease, we think that these agents are so well tolerated, and so safe in comparison to traditional chemotherapy that we can even go into premalignant settings. And for example, cure precursor conditions that ultimately lead for example to myeloma.

These are really exciting opportunities where you can start imagining treating cancer more by prevention. Then by waiting to get a serious advanced cancer and then having to treat the very serious advanced cancer. I mean, I can see a future world just like everybody gets tested for their cholesterol and gets put on lipid lowering therapies, hopefully PCSK9 blockers, but I can see the same thing for example, a very substantial proportion of the population over the age of 60 has what we call monoclonal gammopathy of unknown significance which has a very significant a few percent a year rate of transforming to myeloma. Which even in the setting with bispecifics and CARTs is still relatively incurable.

We can imagine setting up taking all those patients and essentially short-term treatment clearing the precursor conditions, so they have no risk of developing disease. So these -- I think having these best-in-class molecules across so many different settings, I think opens up a lot of opportunities for doing things and going into spaces where people haven't even imagined going before.

Brian Abrahams - RBC Capital Markets - Senior Biotech Analyst

That's really fascinating. I know that's something they're looking at in Alzheimer's diseases as well, and it seems to be a concept we're seeing what we could see more and more of, especially if you have a safe enough agent. Just on the Fianlimab melanoma data that that that you mentioned, what do you think is the biggest differentiator that's kind of led to some of the data that you've advantages you've seen so far? Is it the better LAG-3 or the better PD-1 backbone? And as a second to market agent, what do you think you need to show to gain the most commercial traction?

George Yancopoulos - Regeneron Pharmaceuticals Inc - Board co-Chair, President, Chief Scientific Officer

Yeah. What as I was saying before, especially in cancer, you don't want to be going in with two inferior agents and combining them and hoping that they combine and do a little bit better than the agent individual. You want to go with the best-in-class agent, so we think it really matters that you go in with the best PD-1 and with the best LAG-3, and hope in the early data that we've now seen prior to our Phase 3 suggest that putting two of the best-in-class checkpoint inhibitors for these checkpoint pathways, the PD-1 and LAG-3 really can produce better data. We're seeing -- we were seeing in the early studies, 50% to 60% response rates as opposed to 40% response rates with the combo of the competitors with much longer control of disease, doubling in terms of time frame.

If we get anywhere near that data, I think that this is obviously game changing for these first line metastatic melanoma patients, and we can only hope for their sake that the data really at all comes close to resembling that sort of data, but it would be transformational obviously. And the other good news about it so far in the studies is that we have very good safety profiles. Once again, I think it has to do a lot with the design of the molecules and how you screen for them and so forth you know. Just go with the one antibody you have, you have a variety of screens that lets you go through thousands or millions to get the best-in-class agents. And so, right now, the data suggests that there's a real opportunity that this can really take treatment of this terrible cancer to a whole another level both in terms of the efficacy but also with a very desirable safety profile.

Brian Abrahams - RBC Capital Markets - Senior Biotech Analyst

Okay, great. Well, shifting gears a bit, we've been getting asked more and more about the factor 11s. It seems like people have started to take notice. You've had some initial proof of concept data there, but you've taken a different approach, having two different -- you're taking the antibody route and two different antibodies. What were some of the learnings from maybe the prior efforts in the factor 11 space that you were able to draw from in designing this pathway that you're going after? And what are your plans going forward? Are you planning to look at higher risk patients, go broad on a lower risk in patients who would otherwise be eligible for factor 10s? How should we foresee this Phase 3 program kind of taking shape because I know it -- it's going to be kicking off pretty soon.

George Yancopoulos - Regeneron Pharmaceuticals Inc - Board co-Chair, President, Chief Scientific Officer

Those are great questions. I think the way we look at it is that one can stop blood clot formation cold, okay. But when you do that with traditional approaches up until now, the problem is you unfortunately cause a lot of bleeding, and this is why anticoagulation therapy is not as widely used as it could be used if it was safe, so you know it's all about the bleeding. That's what it's all about.

It's not about making a better anticoagulant that can stop clotting better. It's about doing it more safely, so you don't have to worry about a brain bleed or other significant bleeds. I know I struggled with this with my dad, he had atrial fibrillation, and he went on anticoagulants, and he was bleeding all over the place, he goes I'm not taking this. It's going to the bleeding is going to kill me. So patients don't like to see bruises and blood all over themselves, and God forbid they have a fall, and they have a really bad bleed and they realize and recognize that this may be a greater risk to me than the clot is which I can't see. And that's the other thing is you're seeing a lot of this bleeding so that's the thing that we're focused on and that's why we've developed two antibodies and that's what's different from the small molecules and the factor 10s and all that.

Once again, going back to our large genetics databases, it suggests that the factor 11 pathway may be able to approach, if not meet, the sort of anti-coagulation control that you can get with the factor 10s or other approaches, but much more safely. So it all depends, I think more than on the efficacy we've demonstrated as you said in our early studies, efficacy that as I said, is approaching if not comparable to that of existing agents, but if the data continues to support the genetics evidence that suggests that this could be a much safer approach you can imagine once again something akin to putting this. I'm not saying literally, but like putting in the drinking water if you could stop clotting safely with minimal risk of bleeding, many more people should take it.

Right now, these are limited to the higher risk situations because of the bleeding risk. You have to balance. I want to prevent the clot, but I don't want to kill someone or cause a lot of problems from the bleeding and that limits the use of these. We think the safety profile is going to determine how broadly these can be used and across what populations. I'm sure we've all seen, you go to the hospital, you're an increased risk for clot. Okay, what do they do? They put these cuffs. You ever visit any family members, the cuffs aren't on, they're not working whatever. People aren't being anticoagulated. You could literally argue that a very high percentage of the population as you get older needs substantial anticoagulation, but we know even baby aspirin, has its risks. So the safer the profile, the more widely it can be used across more and more indications. And of course, you know we are going into the higher risk settings, but we also have some ambitions, depending on the safety profile of really going much broader into areas that people haven't really ventured before.

Brian Abrahams - RBC Capital Markets - Senior Biotech Analyst

So start with higher risk. And then as the as you get a better sense of whether this is indeed as safe as hoped, go broad.

George Yancopoulos - Regeneron Pharmaceuticals Inc - Board co-Chair, President, Chief Scientific Officer

Yeah, I didn't say start with higher risks. I think that, you know, we have two antibodies where we're exploring multiple opportunities simultaneously.

Brian Abrahams - RBC Capital Markets - Senior Biotech Analyst

Got it. Regeneron has lots of genetically validated targets coming out of the platform that you've talked a lot about, things like NPR1, GPR 75, that we're starting to see advance into clinical development. Can you tell us a little bit more about the Regeneron Genetics Center, some of the key differentiating elements and really how it's prompted and shaped both your internal pipeline and some of your external collaborations including this week's acquisition of 23andMe?

George Yancopoulos - Regeneron Pharmaceuticals Inc - Board co-Chair, President, Chief Scientific Officer

Sure, no, great question. So, we believe nothing else exists like it on the planet. We've invested more than anybody else in terms of building a sequence-based big data set. The problem that we've recognized, and I hope everybody recognizes, is you can't count on the published literature. Some number of its 70%, 80%, 90% of it is either wrong or misleading, and there aren't that many other resources out there that you can use. I don't even read the literature anymore. I want to know whether is the IL4/13 pathway relevant to eosinophilic esophagitis? I could read a hundred papers, half of which say one thing, half of which say the other thing, or I just go to my database and my database tells me the truth, okay?

People talk about AI like it's going to be deliver us some sort of incredible, great opportunities. The problem is in our field, if you train AI on the existing data that exists, you're going to get random garbage out, okay, garbage in, garbage out. We've taken the time and put in both the time, the incredible scientists and technology and the investments to build what we think is the most valuable big data set in our industry. And that is something that literally will empower machine learning and other approaches and that's what we use all the time. Like I said, you know, we want to understand which pathway and which disease and so forth we can get definitive answers from our genetics data set.

And we've made about a little bit more than 10% of it public. We're the ones, for example, who sequenced and made public the UK biobank data set that is outside of Regeneron and that's the most widely used DNA sequence-based data set on the planet. We created that, we made it public, but it's only a little bit more than 10% of the data that we have, and more data is power here. And so, I think this is a huge differentiator for us.

As you mentioned just this week, we announced that we were involved in a process, which we hope will be finalized by the courts and closed in the third quarter where we're acquiring 23andMe. We think that this is an interesting piece to our puzzle. We've been -- we believe somewhat stealthily, the world leaders in DNA sequence and genetic-based research, and I do want to remind you we're probably the first company that bet its entire future on the power of DNA. We've been doing high throughput genetics longer, I believe better and certainly faster than anybody else, has been doing for over 30 years. But we've been doing it, like I said, for our internal purposes.

We think that there is a lot of room to help benefit individual patient's personal health taking advantage of this information. This was the vision, and this is how and why 23andMe was created. We think that they were created with a great vision and a great brand. They didn't necessarily have the best technology, but we think that we can help achieve the initial mission and goals of 23andMe to help individuals with their personal health, but we think we can go broader than that. We think that this could be a platform that we can use to more broadly impact societal, health and well-being by taking advantage of our DNA and genetics approaches. We're going to really be taking I think those capabilities that mission, that dream to a whole another level. I mean the way we think of it is 23 and more.

Brian Abrahams - RBC Capital Markets - Senior Biotech Analyst

It's great. Maybe just a quick question on Dupi and kind of the longer-term plans, understanding the field is is obviously competitive and Dupi is pretty tough to improve upon. But broadly speaking, how much of a priority do you have on developing next generation versions or derivations of Dupi internally or externally, thinking with that may have certain advantages? And just how do you think about much longer term kind of leveraging the fantastic data set and antibody that you have to go for the next 20, 30 years with it.

George Yancopoulos - Regeneron Pharmaceuticals Inc - Board co-Chair, President, Chief Scientific Officer

Well, we think EYLEA is an interesting example. People can continue to debate how the science is going to turn out from a commercial point of view. But as many of you may or may not remember, when we first launched EYLEA almost 15 years ago, it was clearly like we always do, we delivered the best-in-class molecules that really took over the space because it was the most potent biologic blocking VEGF ever designed.
And if you guys have followed the industry, there are dozens and dozens of companies that try to match or take down EYLEA over the subsequent 15 years. We can argue about it, but I think that the only people who delivered an agent that delivered clinical data in the label that actually is better than the original EYLEA is Regeneron with EYLEA HD. And we all know there's been certain hurdles and limitations that are maybe limiting it a little bit commercially, but clearly, the clinical data is the best-in-class data.

So for 15 years, dozens tried, they all failed to meet or beat EYLEA except for Regeneron. So we're using the same types of thinking and approaches and so forth for all the programs that we're working on whether it's Dupilumab or whether it's some of the cancer programs and so forth. We're always trying to take things to the next level and history shows that we have a pretty good track record of doing that so I would hope we'd be able to do it with Dupi. And it's not just making the Dupi better, it's thinking about maybe more creative ways to use it or use it in special proprietary combinations and so forth and so on. But you know, we built the field, and we fully expect to continue to lead the field for years to come.

Brian Abrahams - RBC Capital Markets - Senior Biotech Analyst

On that note, lots more we could cover, but unfortunately, we have to wrap up. George, thank you so much. Thank you, guys. I really appreciate it.

George Yancopoulos - Regeneron Pharmaceuticals Inc - Board co-Chair, President, Chief Scientific Officer

Thanks.
