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PRESENTATION

Seamus Christopher Fernandez - Guggenheim Securities, LLC, Research Division - Senior Analyst of Global Pharmaceuticals

Okay. Great. Well, thanks, everybody, for joining us. Good afternoon. This is going to be a 25-minute discussion with Merck's EVP and President of MRL, or Merck Research Laboratories, Dr. Dean Li. I'm Seamus Fernandez, one of the biopharma team senior therapeutics analysts here at Guggenheim, and I'm really pleased to have Dr. Li join us as part of our fourth annual oncology conference. Unfortunately, still virtual, but I'm confident that when we do this again next year, we're going to be doing this in person. Boy, I hope that -- I'm touching wood as we speak. So hopefully, we'll be in the right place.

QUESTIONS AND ANSWERS

Seamus Christopher Fernandez - Guggenheim Securities, LLC, Research Division - Senior Analyst of Global Pharmaceuticals

So while Dr. Li leads the company's worldwide human vaccines and therapeutics research development organization, in the spirit of today's discussion and focus, we are going to focus on oncology, in particular.

So maybe just before we get to the categories and the products, Dr. Li, on the third quarter call, I'm sure you're getting this question a lot. Both you; and CEO, Rob Davis, commented on a couple of things. First that Merck is going to continue to aggressively pursue outside innovation. No surprise there. Rob further commented that he believes Merck can remain the #1 oncology company, even after the expiration of KEYTRUDA. But during that same period, we also will see LOEs for Lenvima and Lynparza. And cumulatively, that's probably, at the time, upwards of \$30 billion of revenue after 2025.

So maybe just as the key question here is, is it fair to assume that Merck can only sustain that kind of leadership through larger business development? And if not, what are investors missing? I think that's really sort of the crust of the argument here. Dr. Li, you're muted.

Dean Y. Li - Merck & Co., Inc. - Executive VP & President of Merck Research Laboratories

Can you hear me now? I apologize for that. I think business development is going to be incredibly important, not just for oncology, but in our aspirations to also expand our footprint in non-oncology. So I'll lay that out there. I think the business development is going to happen throughout the spectrum, from discovery to early-stage development to late-stage development. So I'll just put that out there.

The third thing that I would sort of emphasize is from a business development standpoint, it's -- sometimes, we speak about this, but I just want to reemphasize the importance of how we think about business development. We talk about, for example, for KEYTRUDA, Lenvima, Lynparza, I think it's like 150 registrational trials for KEYTRUDA, 45 registrational trials of Lenvima, 35 registrational trials for Lynparza. But the critical thing is we have far more clinical trials than those registrational trials. And many of those non-registrational that aren't sort of Merck's trial are in collaboration with other companies who are really interested in moving their mechanism, their molecules, their hypothesis through the clinic. And what they recognize is the troika of KEYTRUDA, Lenvima and Lynparza, those -- each one of them by themselves, each in combination with KEYTRUDA or even in combination with other things provide a really strong basis that actually sort of repositions the field.

And so I could see a situation whereas those clinical collaborations and partnerships sort of turn their card over, that, that would be a trigger for doing a meaningful business development rearrangement, should we say. So I think there's many different ways to sort of skin the cat. I think you can do large ones, but I also think you can do many middle-sized ones. And clearly, we're going to have to do many early ones as well. So I think it's going to be a combination of all of them.

In relationship to the time frame, I would just emphasize that KEYTRUDA works really well with many therapies. And we have been focused internally on how do we take the pembro arm and ask how do we use different I-O strategies with other checkpoint inhibitors and T cells, other checkpoint inhibitors with myeloid cells, bispecific T-cell engagers, cytokines and the such, and we'll see what those data look like in '24, '25, in that range. And if you see meaningful readouts in those combinations and co-formulations in '24, '25, in those range or even '23, '24, '25, what you're going to do is you're going to reset where pembro is. And the minute you reset pembro with the I-O combination, you're going to reset pembro I-O with what we would call the non-I-O agents, such as how does it look like with surgery or adjuvants? How does it look like with chemo? How does it look like with other non-I-O agents? And so I think those give us an enormous range of possibilities, and we'll have to see how this plays out. But increasingly, the field and our data and all of them have shown signals in relationship to that.

I do want to emphasize the importance of Lenvima and Lynparza is giving us a real foundation, and I will just sit there and go, as we learn more about Lenvima and KEYTRUDA and Lenvima, there is a way of thinking, okay, angiogenesis is important. Blocking angiogenesis is important. What would be the next stage? And one of the next stage that you could think about is what induces angiogenesis. It's hypoxia-inducible, so you start asking yourself, as we advance KEYTRUDA and Lenvima and as we advance ROR1 and cetuximab, will the 2 ever collide together? In relationship for Lynparza, this is telling us about DNA repair and recombination. It's telling us about Part I and II. It's teaching us where we should go next. And there are other collaborations and other internal programs that, as those clinical trials read out, will allow us to know where to focus. And clearly, there's many other sort of agents and physicians that I can talk in relationship to oncology.

So the dip -- let's see what the dip looks like as these molecules make through -- make it through. And so I think there's a lot of different ways that I can sort of unpack what could play out in '26, '28 and 2030. I don't want to tell you that I know for sure, but the degree of optionality I have at this company in relationship to the clinical data that's going to ultimately define where you should emphasize is something that I think I have certain advantages, and I plan to exploit that.

Seamus Christopher Fernandez - *Guggenheim Securities, LLC, Research Division - Senior Analyst of Global Pharmaceuticals*

Got it. Great. And then in terms of some other sort of BD-related decisions we saw following the deal, the LIV-1 deal with Seagen and the acquisition of VelosBio, Ken Frazier kind of expressed the view that while ADCs were of growing BD interest, that M&A in that space was sort of off the table because it sounded a little bit like valuations were out of that mix, not necessarily specific to the question around ADCs, but more so around the possibility of the pullback in biotech that we've seen. Does it make it a little bit easier for you to envision transactions actually closing, whereas maybe there was more of a patient view from potential collaboration partners or even acquisition candidates?

Dean Y. Li - *Merck & Co., Inc. - Executive VP & President of Merck Research Laboratories*

I love that you ask me this question because, as you know, there's another part of my hat that has been involved with spinning out biotech companies from a university setting. One of them is public. And so we have these conversations in that setting as well.

I think what you said is really important. So we have to have a clinical signal reason to do these studies, and we have to have a strategic reason when we think about valuation about whether this is -- what we're trying to accomplish with this. And if we think a trajectory of a size is going towards not -- is meaningful, from our standpoint, we have to be flexible about how we think about valuation because we're trying to lay a foundation for '25, '30, '35, not so much in relationship to loss of exclusivity, but where the field is.

And I'm going to just make one analogy, too, and it happens to be a non-oncology, which is the fact that I actually have studied the pulmonary arterial hypertension field. I've actually treated patients with it. I actually have papers in the field related to that. And the fact that we have internal programs that allowed us to understand, for example, Acceleron and sotatercept, what it could do to reshape the field in relationship to all the

things that we know. I think some people were sitting there and went, "Wow, you guys paid, what, for something in pulmonary arterial hypertension." So the way that we thought about it wasn't about the valuation for this moment, but what it could do to impact the field in the future.

So if we see that, we'll make those choices. But I do think it's also -- you need to do it with a partner who wants to do the deal. And previously, what happens with the pullback is they kind of sat there and said, well, if I need another buck, I can easily raise money. And we'll see whether that changes in their mind. We're interested in partnering with people to move and advance innovation, not just to grab something and then get rid of it, but to really move that. And so you need a willing partner, not just to do the transaction. It can move the field with you. So it's not just a one-and-done sort of thing that we're thinking about in terms of the scientists, in terms of the clinicians.

So we'll see whether it resets how people think, but I -- if this is a reset for 2 years, then I would sit there and go, "Maybe you're right." If this is reset for only 3 to 6 months, I think the appetite on the other side may continue to be one of, 'Hey, there's a lot of money there. Why do I actually need to do it? I can just raise money. I don't need to have a product in the next 5 years. I can just raise money.' And I think that will be the balance of how we think about it. But when we think about it, it has intrinsically moved the trajectory of the science that allows us to know that we're changing the clinical trajectory of the field.

Seamus Christopher Fernandez - *Guggenheim Securities, LLC, Research Division - Senior Analyst of Global Pharmaceuticals*

Got it. Super helpful. Let's jump into the individual products. Just wanted to maybe start with some upcoming data. We'll see the PROpel data. You guys talked about the PROpel clinical trial of Lynparza in prostate cancer pretty enthusiastically on the fourth quarter results call. On their third quarter results call, though, it seemed like Astra was maybe tempering expectations a little bit to perhaps think about areas like HRR, having a bigger signal. Just wanted to get a sense of just how you see the opportunity with the PROpel data kind of unfolding over time.

Dean Y. Li - *Merck & Co., Inc. - Executive VP & President of Merck Research Laboratories*

Right. So let me separate it in terms of 2 things: prostate, and then let me also separate it in terms of thinking about DNA repair and recombination and PARP inhibition broadly. So last year was clearly the year of women's cancer for Merck, right? We made substantial impact in endometrial, cervical, in breast cancer, being both at early stage and late stage. So that was a really important sort of advance for us.

There was also enormous advancement in early-stage cancer, which is really important to us because that's going to be a major place where KEYTRUDA can do extremely well but also in terms of thinking past 2028. As those early stage comes up, there's going to be a demand for other innovation that we are confident that we can provide, that will be important for patients and also are protectable past 2028. So that's how I think about it.

And we're now moving to prostate. So I would just emphasize PROpel with Lynparza but also KEYNOTE-921 with KEYTRUDA, but importantly, the KEYNOTE-010 with Lynparza and KEYTRUDA in prostate. So it's a whole series of readouts that potentially will be coming out in the next year, 1.5 years that could potentially do the same thing that had happened in 2021. In 2022, 2023, we could have an impact in prostate cancer that we never had before. So that's why PROpel is important from that standpoint.

I would also just emphasize again how I spoke about the adjuvant. The minute you start talking about Lynparza and KEYTRUDA and seeing positive readout, especially in maintenance dosing and relationship, the same argument that I made for adjuvant you now have with the patient on taking an oral pill, you're asking him to come in an infusion center every 3 weeks. "Really? Is that what you really want me to do?" And I think they're going to demand the same innovation that those people in adjuvant are. So they're important for our market in terms of women and prostate and how we think about '25, '28 and beyond.

Relationship to PARP inhibition and DNA repair and recombination, in many ways, parallels the story with WELIREG, at least in my mind. And the concept is that in when -- PARP inhibition came, it came in relationship with a concept of synthetic lethality to germline mutations and relationship to BRCA1. Then it moved to sporadic mutations in BRCA1. Then it moved to HRRm in relationship to PARP inhibition.

What's interesting to me for PROpel is whether or not there is any signal that Lynparza, in combination with another drug, such as a hormonal receptor antagonist, will broaden the impact past the genetic sort of biomarker. And the way that I give that sort of analogy is in lung cancer, for us to move quickly, we went to PD-L1 high with PD-1. But the minute you did PD-1 plus chemo in lung, all of a sudden, you melted away the biomarker.

So we'll have to see that PROpel data, but I just wanted to lay out why the PROpel is interesting to me in relationship to how we think about prostate cancer but also how I think about PARP inhibition. And then what's the next step? Is the next step thinking about PARP inhibition has really, in some sense, DNA repair and recombination and molecularly targeted agents as really chemo and a really more targeted pill form, that, I think, could be very interesting. But we'll have to see the data as it evolves.

Seamus Christopher Fernandez - *Guggenheim Securities, LLC, Research Division - Senior Analyst of Global Pharmaceuticals*

Got it. So let's shift to the I-O combination pipeline and sort of the '23- plus time frame for data sets. Maybe first off, rather than go to TIGIT, I did want to cover ILT4 because in June of last year, Eric Rubin highlighted the possibility that we might see data from some of the larger signal-finding studies. Just hoping that -- could you confirm that that's actually still the case? And then the separate question is just -- maybe you could just help characterize your enthusiasm for the target and its relevance given that it targets MDSCs.

Dean Y. Li - *Merck & Co., Inc. - Executive VP & President of Merck Research Laboratories*

Right. So fundamentally, as you've outlined, we have spent a lot of internal efforts in relationship to pembrolizumab and what we would say other I-O strategy. We have a whole slew of checkpoint inhibitors in relationship to T cells, CTLA-4, TIGIT, LAG-3. We had many others that everyone else had. We actually have an agonist in relationship to CD27. So that's one big group. We have a series of cytokines that are coming in, and we're watching other people's data and our own sort of engineering. So that's coming in.

One hypothesis that's been laid out there has been about the myeloid cell line in relationship with that. That started with many people being interested in TGF-beta. There was a bispecific that was interesting in relationship to that. We played in that field with TGF-beta. And then there's other sort of assays -- other sort of assets and pathways, such as CD47 and ILT4 and the such. So our interest is to see whether this myeloid hypothesis is true. And if it's true, I think it's going to set up a lot of interest in multiple ways to sort of interrogate that.

In relationship to ILT4 and ILT3, I think Eric Rubin is continuing to do to a cell signal finding. It's a robust signal finding readout, but it's not a robust blind. There are clear associations of what tumors have myeloid infiltrations and where it's best to position those products, and so that's where he's focused on.

That hypothesis is not just -- it's a robust program, but it's not willy-nilly sort of thing. You have to ask yourself, where does the myeloid hypothesis? And that's true for CD47. That's true for TGF-beta. That's true for ILT4. We'll have to advance. Any moves in the field by any of us will trigger enormous amount of effort. And so we think that we looked at TGF-beta. We looked at CD47. We looked at this. We think, at least, for us, the best place to place that bet is in ILT4 and 3, and that was based on molecular data and cellular data that we have in our own studies about patients who didn't respond to pembro as much as we would like them. So that's why we like that program simply because it was kind of gave hints from our own human biology, but we're also being very aware of what other people are doing in the field.

Seamus Christopher Fernandez - *Guggenheim Securities, LLC, Research Division - Senior Analyst of Global Pharmaceuticals*

Got it. And I guess that kind of brings us to TIGIT, to some degree, and LAG-3 in terms of maintaining that awareness of what other competitors are seeing. We know that we're likely to get the TIGIT data later this year from Roche's program. Just hoping what you're hoping to learn from that program and maybe what you feel the field was educated on, not just from your own internal data, but from the Roche data as well. If I were to just sort of say it simply, I felt like PD-L1 was really the target location in lung cancer for that and no better company than Merck to be positioned to advance that, given KEYTRUDA's presence there, but interested to just hear your thoughts in that regard.

Dean Y. Li - Merck & Co., Inc. - Executive VP & President of Merck Research Laboratories

Yes. So let me just step back. Anyone who can get -- especially in lung, for example, anyone who can get a new standard of an I-O agent, we believe that pembro with monotherapy and pembro with chemo is pretty much the standard. But if you could make pembro better, either in monotherapy, then immediately people are going to say, "in combination with non-I-Os, with chemo, with targeted therapies, with surgery with radiation," you're going to have the same thing happen all again. So it's kind of like showing that TIGIT does something, in addition to PD-1, will be important. And if you can prove that cleanly and it's meaningful, that's not going to be a new storm. You know what's going to happen in every other combination of a non-I-O agents in relationship to pembro, but -- and that's going to reset the field.

In relationship to TIGIT, I would just emphasize there has been a lot of checkpoint inhibitors and T cells that have been put in multiple nature review, cancer and all of this, many of them have not panned out. And they haven't panned out oftentimes because people haven't done the clinical experiment in a precise way. And so I do want to give credit to both our group and competitor group where they really look to ask themselves does TIGIT do something in addition to my compound.

And I think that's a really important point because when people don't do that, it makes it murkier. And also, what is really important is I think the FDA is increasingly going to hold that standard, not just to us, but to other people who have molecules in other countries trying to come in here. And so that way of developing it will be very important.

When you look at the SKYSCRAPER, there was clearly, at least in my mind, evidence that TIGIT did something on top of a PD-L1. And so that gives you a sense of TIGIT doing something on top of PD-L1. If you look at that data, you would also squint into it and say, "The monotherapy for PD-L1 was not the greatest." And so you're asking yourself, did you see the addition of TIGIT with that PD-L1 because it was truly adding something? Or was it that the PD-L1 just had so much room for improvement?

And that's what will be very important is that when we stare at the data from the PD-L1, PD-L1/TIGIT, I think we need to stare up whether TIGIT does something to PD-L1, but also where does that TIGIT PD-L1 stack up against PD-1 and PD-1/chemo because it's one thing to do the hypothesis that TIGIT does something more, but it's also, from a patient standpoint, if I can prove to you that PD-L1 plus TIGIT does something more than PD-L1, but the PD-L1 plus TIGIT isn't better than PD-1 or better than PD-1 chemo, as a patient, you're going to ask yourself, "Okay. What did you really prove to me?" And that, I think, is what -- how we're going to look at that data carefully, and that's how we look at our own data carefully.

Seamus Christopher Fernandez - Guggenheim Securities, LLC, Research Division - Senior Analyst of Global Pharmaceuticals

Got it. So unfortunately, we only have 25 minutes. So I have one last question that I want to ask. Of the assets that you have in clinical development that we haven't talked about, whether it be LAG-3, the ROR1 ADC, between those 2, I think ROR1 is probably the one asset that isn't talked about much and maybe isn't fully appreciated. I was hoping you might be able to give us a sense. I think in our previous conversations, you said the game-changing event for this acquisition and this compound won't be in lymphomas per se. If it's going to be a game changer, it's going to be in solid tumors. When might we have that kind of data for the ROR1 opportunity?

Dean Y. Li - Merck & Co., Inc. - Executive VP & President of Merck Research Laboratories

Yes. So let me just -- let me put that in the context that I want. We need to prove that the ROR1 ADC, where the data is, is in hematologic malignancy. And so we have to advance that because we're very interested in moving in hematologic malignancies, and we have a BTK inhibitor. We've done more dose exploration and confirmation that was required. And again, a fulsome development program is going to be deployed. There's competition in that field. But the ROR1 ADC is critical to our strategy of really laying out a foundation in relationship to hematologic malignancy. I would also say that some of our IO-IO agents may also be important there as well. And so we have a fulsome program. There's a fast-to-market strategy, and there are full registration trials that are being actively deployed. And I think -- I'll let Peter and other people correct me. I think we posted 2 such trials for diffused B-cell -- large B-cell lymphoma just recently in the last month or 2, and additional studies will be forthcoming. So that ROR1 ADC is, in many ways, a lead compound for us to make a bigger foray in relation to hematologic malignancy.

In relationship to solid tumors, one is -- looking at the ROR1 ADC in that context, one may be very important to use the ROR1 as a target, but it may be that the design of that ADC may not be ideal for solid tumor as it is for that. So we're interested in LIV-1, ROR1, many of the other sort of targets as a general rule in ADCs, but for the ROR1 ADC, we're very focused in using it as a lead compound to really make a bigger impact in hematopoietic malignancy than we've had previously.

Seamus Christopher Fernandez - *Guggenheim Securities, LLC, Research Division - Senior Analyst of Global Pharmaceuticals*

Got it. So it sounds like to get into solid tumors, if ROR1 is a target, you probably need another linker -- or not a linker, but a different payload...

Dean Y. Li - *Merck & Co., Inc. - Executive VP & President of Merck Research Laboratories*

We've got to compare -- just to let you know, when we bought Velos, there was a number of agents without, say, combined with ROR1. The lead compound -- there is more than just the lead compound.

Seamus Christopher Fernandez - *Guggenheim Securities, LLC, Research Division - Senior Analyst of Global Pharmaceuticals*

Got it. Super helpful. Great. Well, Dr. Li, thank you so much for spending the time with us. Peter, I apologize for not introducing you at the beginning of the call, but thank you, as always, for joining us. I very much look forward to doing this in person in 2023. Thanks so much. Really appreciate it, and good luck with all of the development that's going on at Merck this year. I know patients are all counting on you.

Dean Y. Li - *Merck & Co., Inc. - Executive VP & President of Merck Research Laboratories*

Thank you very much. Take care.

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