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EDITED TRANSCRIPT

MRK.N - Merck & Co Inc at Jefferies Global Healthcare Conference

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OVERVIEW:

Company Summary

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PRESENTATION

Akash Tewari - *Jefferies LLC - Analyst*

Day 4 of our Jefferies Healthcare Conference. This is not one of those conferences where you can leave on the second day, which I love doing sometimes. But it's a long haul, and I really do appreciate everyone sticking around. I can tell you personally it's been a joy to see all the clients and all the companies, and it does seem to be such an exciting time in healthcare.

My name is Akash Tewari. I am head of our biotech and pharma research efforts. And we have Merck and Eliav and a fireside that I'm really looking forward to. Why don't I hand it off to you for some intro remarks and we'll get started.

Eliav Barr - *Merck & Co Inc - Senior Vice President, Head of Global Clinical Development and Chief Medical Officer, Merck Research Laboratories*

Well, great. Well, thanks very much for the invite. I really appreciate it. And I'm really happy to be here at a time when we're able to see a really exciting period of R&D for the company.

We've had, over the past few years, the beginning of the transformation into the meaningful post-KEYTRUDA era, with nine new medicines and vaccines being approved over the past few years. We've had some really exciting Phase 3 readouts, including sac-TMT and intismeran autogene, as well as our HIV portfolio. And we're going to have a very data-rich next few months, both in Phase 3 and in important Phase 2 proof-of-concept studies with drugs like alimatravir, tulisokibart, among others.

I think that the strategy that we laid out of diversification and following the sciences really paid off. Not everything is going to work because that's science and science is hard. But I think overall, we're very optimistic that we'll be able to provide a very meaningful set of data that will help patients improve and extend their lives, help communities prevent disease, and return good value to shareholders.

QUESTIONS AND ANSWERS

Akash Tewari - *Jefferies LLC - Analyst*

Love it. So I want to start off with cancer vaccines. And I know there's a lot of focus on the renal data that there may or may not be an interim. As Peter reminds me, Merck does not comment on interims.

But I actually want to focus on something more important. I remember at ESMO, your teams talked about in general, like, hey, there's one of the dynamics here is TMB, high and low, but I feel like you've also learned a lot and there is more nuance. It's beyond just TMB. There's IO sensitivity. It could be the types of mutations. It could be kind of neoantigen load itself. So there's definitely, I think, a more dynamic framework of where a cancer vaccine works and where it doesn't.

So your publication came out with Moderna. We were waiting for some more information. I know that's going to take some time. So maybe help us with what your evolving view is beyond just TMB and responsiveness to a cancer vaccine.

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Eliav Barr - *Merck & Co Inc - Senior Vice President, Head of Global Clinical Development and Chief Medical Officer, Merck Research Laboratories*

Yeah. So this is all new biology and it's a very, very exciting time. We're thrilled with the results of the INTerpath-001 study in melanoma. We're learning a lot from it. It's a very data-rich program, and we're going to be able to make a lot of important discoveries about what constitutes how efficacy might have been better or less, how we could improve algorithms, what are the relevant patient populations that particularly responded, and so on.

I think the Phase 2 program that we have going forward also provides us with an important assessment of how wide is the aperture for INT. Do we have to include -- is KEYTRUDA as background necessary? Is it IO-responsive tumors that's important? Can you give the INT by itself? Can you give the INT with other immune stimulants like BCG? And so all of that work will provide a lot of the answers that you're asking about.

But I think it won't be just TMB, that's for sure. It's going to be HLA. It's going to be the tumor microenvironment. We're looking at -- we have a couple of studies in the metastatic setting just to see how that looks. But again, it's new biology. We'll just have to see how it goes.

Akash Tewari - *Jefferies LLC - Analyst*

Understood. And maybe just as a follow-up to that, your team did have interesting data in pancreatic, as did BioNTech. But I can also tell that that's not necessarily where you're steering investors. You're like -- it's an interesting proof of concept. So talk about why, renal, you feel like that was in that initial set of bets we're making, whereas for pancreatic, that really wasn't the highest probability bet for your team.

Eliav Barr - *Merck & Co Inc - Senior Vice President, Head of Global Clinical Development and Chief Medical Officer, Merck Research Laboratories*

Sure. Well, I think that pancreatic cancer is very cold from the immune response point of view. There was never much response with pembro or any of the other immunotherapies. The patients are fairly advanced when they get diagnosed. There's very few patients that can undergo surgery, and even when they do, it's a pretty gargantuan surgery.

Whereas renal cell cancer is incredibly well studied. We've had multiple trials in the renal cancer setting, renal cell cancer, and in early disease. So the combination of pembro in early disease, immunoresponsive, was the first -- was kind of the beachhead of where we were going to go.

If we see strong results, we'll then expand even further and ask the questions about cold tumors, but that's not the first place to go even you see some Phase 1 proof of concept. Right now, I think physicians who are interested in treating pancreatic cancer are focusing on the RAS inhibitors.

Akash Tewari - *Jefferies LLC - Analyst*

Understood. Now, on the interim, which I know you're not going to comment on, I'm going to actually shift in a different way. I feel like there's too much focus on the interim. When I saw the landmark analysis you showed in melanoma after a year, where it's not that large of a study, you can see enough variability in terms of how IO can perform.

But the real thing that stands out, at least to me, when I look at your melanoma data is it works in PD-1-high, it works in PD-1-low. We didn't see that with LAG-3. And it's the duration of response on top of, obviously, the side effect profile. So let's skip the interim question. But why is your team interested in looking at the long-term follow-up in renal? And why would that maybe be the right focus for Merck?

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Eliav Barr - *Merck & Co Inc - Senior Vice President, Head of Global Clinical Development and Chief Medical Officer, Merck Research Laboratories*

Yeah. So I mean, you have to -- so thank you and I agree with you about the nature of the -- of course, interim analyses tell you different things. And I know investors really want to know as soon as possible where things are going, but science has its own rhythm and timing.

If you look at the analysis of the melanoma data, you saw this piecewise analysis where, the first few months, there really wasn't much of a response. Then afterwards, you have this huge response that continued and improved over time. That is completely consistent with the immunotherapy approach and why we use nine doses. You need to prime and then repeatedly boost the immune response and train it.

And so the upside of that is that obviously you get long-term responses that make all of us here excited. The downside is that it's the enemy of interim analyses because there's a time basis that does not -- that simply, there's no amount of money that you could throw at it that'll change, that it takes time in terms of follow-up time. I'm not talking about time to recruit. So I think it's going to be important for us to not overreact to any interim analyses, yes, no, whatever, and to look at the overall picture.

The renal data are very important because there's a TMB-low patient population. So it tells us whether or not we need to focus just on the TMB-high tumors which, by itself, is a huge opportunity. But it tells us like, okay, you really need to stay within the sweet spot of KEYTRUDA and focus in that area. But again, I think doing this study at this stage was important because we needed to understand whether it was KEYTRUDA responsiveness or TMB or both.

Akash Tewari - *Jefferies LLC - Analyst*

Understood. Now, one of the really provocative data points in that Phase 2 publication, but unfortunately, I think it was only in 10% of your patients, was when you looked at longitudinal ctDNA positivity and negativity. That was, in and of itself, actually the biggest predictor of whether a patient was responsive or non-responsive to a cancer vaccine.

Unfortunately, because it was such a small subset, we can't apply that to the full group and I'm suspecting you might have more data. But let's say that if you did measure that in every one of those patients in your Phase 2 and that signal held up, what conclusions could you make about whether TMB is driving or non-driving the responsiveness to a cancer vaccine?

Eliav Barr - *Merck & Co Inc - Senior Vice President, Head of Global Clinical Development and Chief Medical Officer, Merck Research Laboratories*

Well, I mean, I think ctDNA's role in monitoring patients' responses is a relatively new concept. I mean, ctDNA has been around for a long time, but as we build more and more ultra-sensitive tools, we get more information about that.

The issue with ctDNA, to be frank, is that its shedding is very much tumor dependent. Different tumors have different shedding kinetics. When you have larger -- there's an intrinsic bias about the size of the cancer and its location, just because of how much shedding happens. So I'm not so sure that that's going to be a great tool for assessing responsiveness or something like that.

What I do think is going to be important, and I don't have any data right now because it's required a lot of number crunching, is to try to understand are we beginning to see correlates of response and will that be different for different cancers. And so I mean, this is a longer-term project, obviously. But I think the first step, especially as far as investors are concerned, is do you have positive results. And then after that, can you enrich for even more or is there some algorithm change that you can do to make the efficacy even better?

And here, one of the things within intismeran that's really important is the enormous barrier that having all that data and having it now places in front of other developers. For us, it creates an enormous opportunity to be the leaders and to maintain that leadership.

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Akash Tewari - *Jefferies LLC - Analyst*

Understood. You made a passing comment at ESMO, but it was a very provocative one. You said, we are learning some things about renal. One of the things you did say was we're actually doing a good job generating neoantigen, not responses, but we are generating neoantigens. We just don't know, as you mentioned with correlates, whether that actually correlates to efficacy. But can you expand on that a bit? What are you seeing with renal and the ability to at least hit the target correctly?

Eliav Barr - *Merck & Co Inc - Senior Vice President, Head of Global Clinical Development and Chief Medical Officer, Merck Research Laboratories*

Yeah. The thing with -- the big question with INT just in general is whether what we predict by algorithm in terms of mutation and how mutation translates to actual protein, which then is presented to the immune system through MHC, and that's HLA dependent. So the question is how do you know which one's the best one?

And right now, there's a lot of trial and error. The thesis had been that those mutations that are highly predictive to be expressed, that create epitopes that are completely novel, and that are well matched with the relevant HLA for the patient, that's the ones that are going to do the trick. But that's a hypothesis.

What was good about the renal data was that we were able to create some of these neoantigens because it's not TMB-no, it's TMB-low, and so we had that, but also to now test other kinds of mutations that might be relevant but weren't necessary for the melanoma trial. So if it works, and I really don't have any data -- awaiting the data results when they come, we'll have a better -- there'll be a richer repertoire of mutations that we'll be able to look at.

And again, it gives you an opportunity to understand what's the aperture here and what's the work that needs to be done. By the way, this is a great place where AI is helping us a lot because the amount of data that comes out of these studies is gargantuan, over 1,000 genomes and various immune responses and our choices and so on and so forth.

Akash Tewari - *Jefferies LLC - Analyst*

Makes sense. Last part on cancer vaccine, and I asked you this in ESMO, you could argue the first cancer vaccine was GARDASIL in many ways. And there is this framework of, okay, there's a post-resection setting, but there's a limited patient group that would be there and a different cost structure. You are noting that you're exploring monotherapy and then combinations outside of KEYTRUDA.

What did you learn from some of the monotherapy data we've seen with the cancer vaccine about its ability to generate neoantigen responses durably? And then what did you learn with that landmark analysis where, it was really the -- it does seem like the cancer vaccine is doing something, but it takes some time for that to really load up, I would say?

Eliav Barr - *Merck & Co Inc - Senior Vice President, Head of Global Clinical Development and Chief Medical Officer, Merck Research Laboratories*

Yeah. So I'll say that if we can create an INT that doesn't require pembrolizumab as background, it's going to be very helpful. Because pembro is a great drug, god knows and MSD knows for sure. MSD has been the leader here. But I have to say that when you start to look at people with very early-stage tumors, you don't want to expose them to autoimmune phenomena and so on. So my preference would be to have, for the very early stage, just the INT because the safety profile is relatively well tolerated, just like injection site stuff.

So I think one of the biggest proofs I think will be in bladder and non-muscle-invasive bladder cancer, because there, we have a pretty big experience that we're going to develop with INT only, as well as with INT plus BCG. So that gives us some idea about whether or not you

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need that second immunostimulation. We also have a high-risk Stage 1 case, a Phase 3, but that's going to take forever to read out because it's still 10 years or so.

I would say, by the way, the last thing is the prime-boost stuff, the repeated priming, no surprise. If you look at all -- my analogs are Shingrix and GARDASIL and you need to have continued priming before you actually get a magnitude of effect that's durable.

Akash Tewari - *Jefferies LLC - Analyst*

Right. Actually, that will bridge to, I guess, a regulatory question, which is, and we'll talk about this with PD-1/VEGFs and like when the FDA considers accelerated approval or not, there's a lot of different factors. It doesn't seem like a bright-line test. It's a holistic decision, and a big part of that is safety. So when I look at, let's say, combining EGFR TKI and chemotherapy, like, yeah, it'll work, but there's a toxicity burden. You could argue the same thing with a HIF-2 alpha, pembro in renal.

How does the discussion about accelerated approval change when you have a drug that has very limited additional side effects to the background medication like KEYTRUDA? Is there more receptiveness with the agency, especially for, let's say, elderly patients who can't tolerate some of these regimens to get it on the market?

Eliav Barr - *Merck & Co Inc - Senior Vice President, Head of Global Clinical Development and Chief Medical Officer, Merck Research Laboratories*

Well, I mean, I think first of all, I don't speak for FDA or EMA or any of the other agencies. I agree with you that benefit-risk is really important, both the benefit and the risk, and so that's critical.

When you have a background of pembrolizumab, you have a clear understanding of what to expect. The regulatory databases both in the US and Europe and other countries is pretty rich. Adding INT to that really just increases injection site reactions. So I think that the calculus has to be magnitude of benefit, confidence intervals. And is this a clear signal? And can you take analogs from other areas and apply them?

So for example, oftentimes, accelerated approval can be facilitated by presence of data from other tumors, both from the safety database and the correlation between, let's say, an early signal versus a late signal. And so those are the kind of things you'd think about when thinking about AA.

Unmet medical needs are also really important. And so it's a multifactorial thing. And you're right, it's a totality of data sort of thing. FDA has their systems. We never count on it. We just provide the data and see what they have to say.

Akash Tewari - *Jefferies LLC - Analyst*

Understood. Maybe bridging to the PD-1/VEGFs, and I was personally a little surprised that you guys made the announcement at ESMO about moving forward with -- you talked about we want to replace KEYTRUDA. There was, let's say, an acknowledgement that this was a worthy backbone to combine with sac-TMT. And I think that has been -- you've incrementally shifted your tone on this over a period of time.

I find it really interesting that it was really now where you put your foot forward. How much of that was -- because you said LaNova was a Phase 3-ready asset? So what did you see externally that made you feel comfortable that it's worth adding a PD-1/VEGF to an ADC to really replace front-line standard of care in NSCLC?

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Eliav Barr - *Merck & Co Inc - Senior Vice President, Head of Global Clinical Development and Chief Medical Officer, Merck Research Laboratories*

Yeah. So we're still in the -- we're doing it in a disciplined way and not in the KEYTRUDA sort of try-everything approach. But we saw HARMONi-6, which did show an OS benefit. We saw that there was positive results in biliary tract cancer. And Akeso's data in China seemed pretty reasonable. We still need to make sure that this is for real. And I'm not talking about the early PFS or even early OS. I just want to make sure that cumulative toxicity isn't an issue and it seems like it's not.

I would also say that nothing helps more than having a lot of data on your own drug. And we've had a pretty big program, again, in China where things can be done pretty quickly in the early stages that led us to be much more confident about what we see with our drug, what's the dose that we would choose. And so then, when we saw the results of HARMONi-6 and other studies like that, we see our own data, which looks really quite nice, and being able to achieve a dose that we think is going to be exciting in terms of ease of use.

And then the sac-TMT data from China from Kelun made us realize that A plus B is going to be -- the two together are going to be great. We need to understand whether there's cumulative some sort of toxicity that needs testing, but that can be done in a parallel Phase 2 program.

I would also point out that we've had very productive discussions with the FDA. And the way that I would just say that it's clear that there's been a lot of interest in moving this forward on their part. So I think we'll see. Proof is in the pudding.

Akash Tewari - *Jefferies LLC - Analyst*

That is true. That is the one thing about this. There's always a result. But we did get the HARMONi CRL recently. And I think one of the things that stood out to me, we've seen the FDA-approved trials where maybe 70% of patients are in China. One of their sticking points seems to be not just patients, but number of events. But you also have -- you're looking at a lot of China data, you relied on that.

So what did you learn from that CRL? And how do you apply that to some of your discussions with the agency about your front-line strategy combining both of these assets?

Eliav Barr - *Merck & Co Inc - Senior Vice President, Head of Global Clinical Development and Chief Medical Officer, Merck Research Laboratories*

Right. So first of all, the US is pretty clear that they want data that also includes US patients, period. What they've been also clear about is that they don't love, and they said it in the CRL, they don't like studies that started off in China, enrolled most of the patients in China, and then you have kind of like a little add-on from international. So I think part of it -- and so why is that? What makes that different than, let's say, a study that has 75% and 25% ex-China but it's enrolled contemporaneously.

The reason for that is the maturity of the US data are very immature, so they can't tell whether the overall data and the US data are consistent. And you'll notice that a lot in clinical trials where the US underperforms, and the reason for that is that it takes sometimes longer to enroll in the US. So what we've moved to is to front load the US in our oncology trials so that you have mature data. It's not the percentage; it's the maturity that's important. And that really has made a difference for us, and I think we've had really good interactions with FDA regarding those elements.

Akash Tewari - *Jefferies LLC - Analyst*

Understood. I thought one of the -- you didn't get as many questions on it at ESMO, but I could tell your team was quite excited, the G12C data. I find that interesting on multiple fronts. A, it is an AI-optimized drug, but B, that was a -- I feel like it's a part of a broader take.

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Merck's approach in first-line with targeted agents are I want a very clean selective drug and want to avoid chemotherapy. So it's not just about efficacy, but it's really about tolerability. We've seen pan-KRAS agents, which are -- they have beautiful efficacy, but they do have a tolerability burden.

So here's really what I'm trying to get at. Is your view of the world I'd rather go in a front-line setting, targeted selected agent, ideally chemo-free, versus let's say we take a pan-KRAS agent front line because it has a broader signal? How are you thinking about the KRAS space evolving over time for both lung and other indications?

Eliav Barr - *Merck & Co Inc - Senior Vice President, Head of Global Clinical Development and Chief Medical Officer, Merck Research Laboratories*

I really think that the credit should go where -- we should give credit where credit's due, and that's to AZ and Tagrisso. I mean, I have to say that a drug that you can take once a day that has a -- it's not a walk in the park, but it's a decent safety profile and you can take it for years and patients really do well, that to me is now there's a good drug. And so I don't see any reason why you need a pan-KRAS or pan-RAS in lung because it's not difficult to get the mutation status.

So I'd much rather have a tolerable drug, an oral drug that can be administered with real inhibition and hitting hard the mutant KRAS. Calderasib or MK-1084 is exactly that. It's really well tolerated. In combination with pembro, we have really, really durable efficacy. I mean, really long-term efficacy. Why bother with chemo? You see the olomorasib data for Lilly and they actually had lower responses.

So what we want from driver mutation cancers is to be able to give a drug that's actually going to allow patients to live a relatively normal life. And it's for that reason why we have a G12D that we in-licensed and we have our G12C. And I think that the trick will be to have a really potent narrow-focus drug that will be well tolerated and can be given oral with pembro, which is pretty easy to do with QLEX now. And so it completely transforms care for those patients. And that's what really matters.

Akash Tewari - *Jefferies LLC - Analyst*

Just to sneak it in, okay, so that's a framework for lung. Let's apply that to pancreatic because that's where it's maybe harder to move away from chemo. Do you feel like that same approach is the right one in first-line pancreatic cancer?

Eliav Barr - *Merck & Co Inc - Senior Vice President, Head of Global Clinical Development and Chief Medical Officer, Merck Research Laboratories*

No. I mean, right now, pancreatic cancer is in a very different space. I mean, you're starting from it's a death sentence and you have drugs now that really make a difference. But if you look -- there was Ben Sasse, the former senator from Nebraska, former President of the University of Florida. He's been writing a blog about his experiences with pancreatic cancer. And he said that he felt like his face and body were melting there for a while when he was on the pan-RAS inhibitor. But he's alive. He is alive. And he has continued to do well.

So that's the metric there. And ultimately, though, we can do better across the board. That's why we do research. We try to improve things and hopefully get to a point where pancreatic cancer is like what melanoma is. Melanoma used to be a death sentence. Now, it's not at all. The same is true now. Hopefully, we'll get there with pancreatic cancer and with lung cancer.

Akash Tewari - *Jefferies LLC - Analyst*

Understood. This is a wonderful discussion. Thanks so much for everyone for joining us. Eliav, thank you so much.

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Eliav Barr - *Merck & Co Inc - Senior Vice President, Head of Global Clinical Development and Chief Medical Officer, Merck Research Laboratories*

Thank you.

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