LSEG STREETEVENTS

EDITED TRANSCRIPT

MRNA.OQ - Moderna, Inc. Investor Event

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OVERVIEW:

Company Summary

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PRESENTATION

David Berman - Moderna, Inc. - Chief Development Officer

Good evening and thank you for joining us today. This is a very exciting day for cancer patients. It's an exciting day for cancer immunologists. And it's an exciting day for Moderna.

Here's our forward-looking statement.

Today, I'm going to start by giving just some brief contextual statements on intismeran. I'll then invite up Dr. Georgina Long, who was the lead investigator for INTerpath-001 and gave a fantastic presentation today to give a reprise of that presentation. I'll conclude with a few concluding statements.

And then, we're very fortunate to have two world experts in melanoma: Professor Ascierto, who was the senior author of INTerpath-001; and Professor Ribas, who is a melanoma expert and a world leader in cancer immunotherapy, as well.

Immune surveillance controls emerging cancer cells. And it does this by detecting neoantigens that form in cancer cells. These neoantigens drain or are carried to regional lymph nodes.

They are processed in antigen-presenting cells and then, brought to the surface of the antigen-presenting cell and presented to T cells. T cells that recognize these neoantigens are activated. They circulate throughout the body, looking for the cancer and they eliminate the cancer.

However, as we know, cancer can escape immune surveillance, when either the cancer lacks sufficient neoantigens; the neoantigens aren't released or aren't transported to the lymph node; the antigen-presenting cells don't process them or present them on the surface.

The T cell is insufficiently activated or the T cells become exhausted. Checkpoints act on T cells, anti-PD-1, PD-L1, reverse T cell exhaustion, and anti-CTLA-4 lowers the threshold for T cell activation. However, they act only on T cells and are limited by whatever available neoantigens are present in that lymph node.

Intismeran, as shown today by the Phase 3 data, has an entirely different mechanism. It optimizes neoantigen presentation.

And how does it do this? Well, first, we take the tumor out of the patient and use an algorithm to select the most promising neoantigens. We package those neoantigens into an mRNA lipid nanoparticle. And that's used to inject into the muscle of the patient.

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Now, we know from non-human primate studies and from rodent studies that when an mRNA-LNP is injected into muscle -- it drains into the regional lymph node -- is taken up by antigen-presenting cells and dendritic cells.

The RNA is produced into protein. And that protein, in the case of our neoantigen, intismeran, can be presented on the surface. So there are three important points to take away about why we think intismeran is so potent:

First, high neoantigen expression in the cells where it's required - in the antigen-presenting cells and in the dendritic cells.

Number 2, this is in an optimal environment. It's far away from the immunosuppressive microenvironment of the tumor.

And number 3, because CD4 and CD8 T cells are activated, dendritic cells are activated, and cytokines are released, there's a perfect milieu to create polyfunctional CD4 and CD8 T cells. And we have translational data to support some of these.

Let me take you to ASCO 2026 just a few months ago, where we presented the Phase 2 data. I'd, first, like to draw your attention to the initial Phase 2 data that was presented a few years ago:

Recall the hazard ratio at the primary analysis, which had the same follow-up as INTerpath-001 today -- the hazard ratio was [0.561] at the primary analysis. With longer follow-up, as we showed a few months ago, the hazard ratio improved to [0.510].

Now, why did the hazard ratio improve? This can be seen, looking at the arts of the Kaplan-Meier curve. For the first six months, on the left-hand side, the Kaplan-Meiers are overlapping.

And as you heard today from Dr. Long, this is, in part, due to the weeks required to produce intismeran and then, weeks or a month for the immune system to be activated. At month 6, the curves separate. And we know they'll eventually remain separated.

However, in the primary analysis, that first six months takes up about a quarter of the entire Kaplan-Meier curve - that hazard ratio [1] for the first six months takes up a quarter of that curve.

In the longer five-year follow-up, that six-month hazard ratio 1 is still there but, now, we have follow-up up to 60 months. And, therefore, that initial six months takes up about a fraction of the entire hazard ratio.

And this is why we pres-pecified to conduct a piece-wise analysis at six months. And you'll hear that from Dr. Long during her presentation.

But it's very important, I think, to recall that the primary hazard ratio was essentially identical to what you'll hear today, 0.561, in this analysis. And we do believe the Phase 3 data, for this reason, will continue to improve.

Intismeran, as I mentioned, activates T cells. And we should expect to see novel T cell clones in the peripheral blood.

This is a figure. The y-axis is the sum of novel T cells that we detect in the blood. And the x-axis is four different time points: baseline; post the pembrolizumab lead-in; and just prior to Intismeran at cycle 5 so in the middle of the intismeran regimen; and then, at a follow-up.

And this is the Phase 2 intismeran plus pembro -- versus pembro. For the first portion, notice the two arms are overlapping because both groups of patients are receiving pembrolizumab -- exactly what you would expect to see.

However, once intismeran is administered, the combination curve continues to go up, more novel T cell clones are generated compared to the control arm.

This leads to the question we asked: Would higher levels of novel T cell clones be associated with clinical benefit? And, in theory, this should be most evident in the combination arm. And that's exactly what we see.

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So the same y-axis is the number of unique clones. X-axis is the same. On the left-hand side is the combination arm. And on the right-hand side is the pembrolizumab arm.

Now, we've separated the patients into two groups: those who had "benefits", i.e., never had a recurrence; and those who had, let's say, no benefit, those who had a recurrence. We did that for both arms.

And you'll notice, for the combination arm, patients who had no recurrence, which is the top, their novel T cell clones continue to increase during treatment; whereas, the ones who had recurrence plateaued. For the pembrolizumab arm, there's an initial increase and then, there is a plateau.

So this is evidence that there's an association, mechanistically, between the number of novel T cell clones and clinical benefit.

Now, these are novel T cell clones. And we don't know if they are related to the intismeran-drug product. And it is a little more complex to match T cells to neoantigens. But we were able to do it in seven patients, which requires a lot more blood than is typically taken during the routine blood assessment.

So we had -- so we consented seven patients. And we matched their T cells to the 34 neoantigens they received in their cassette.

And what we found is, on average, 1 out of every 3 neoantigens in the 34 cassette is immunogenic. That is, it induces a T cell that is novel in the blood that reacts specifically to that neoantigen peptide. And it ranges from about [1] to [18]. And, interestingly, it includes both CD4 and CD8 T cells.

We then sought to further characterize the phenotype of these T cells. And we find -- this is an example. It's a CD8 T cell that recognizes neoantigen 14 from patient 3 and a CD4 T cell that recognizes neoantigen 27.

And when we phenotype it using flow cytometry, what we see is that both T cell clones are polyfunctional -- they make different types of cytokines -- and they also have markers of a cytotoxic phenotype; meaning that they're ready to go out and kill the cancer cell.

Because we were able to clone out those T cells that recognize those neoantigens, we had the TCR sequences, and we could go back to whole blood, and we could follow the frequency or the prevalence of those T cells during treatment.

So it's the same timeframe on the bottom, screening, lead-in with pembrolizumab in the middle of the intismeran regimen and then, at the end.

And what you'll see in these three examples -- that all T cell clones only appear after intismeran, not during pembrolizumab; and most of them remain elevated for the entirety of the treatment.

As a side note for those who are immunologists, one of the interesting features, which is known, is that there are usually multiple T cell clones that can recognize the same neoantigen peptide.

So, at this point, I'd like to invite up Dr. Long to give the presentation of INTerpath-001 and then, I'll return to give a few closing comments.

And please come up this way so that you don't fall. Okay.

Georgina Long - Melanoma Institute Australia - Medical Director

Thank you. (multiple speakers) -- okay. Thank you. It's a pleasure to present these data, again, to a smaller, more cozy audience. So -- and it is a true honor to be able to present results on something so transformational for cancer.

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So this is a Phase 3 randomized clinical trial of intismeran combined with pembrolizumab. We refer to Intismeran as a type of INT therapy versus pembrolizumab alone for the adjuvant treatment of stage 2b up to stage 4 melanoma.

Now, I don't have my glasses but I can see this slide -- I'm not that blind but -- so -- and I do present this on behalf of my co-investigators -- there are 157 global sites -- and all of their teams.

These are my disclosures.

So we know that there is a clinical benefit for the adjuvant treatment of melanoma patients with anti-PD-1 monotherapy alone. That's clear. But shown here are the two curves for stage 3 resected melanoma and stage 2 resected melanoma -- stage 2b.

The red curve is placebo. The blue curve is pembrolizumab alone. And yet, despite their significant clinical benefit and absolutely changing the field, we still have 44% and 33% of patients recurring or dying at five years.

So in the melanoma field, we see that there is a lot more work to do in terms of getting to zero deaths and to curing cancer.

Now, it's not that we haven't been working at it. These are three landmark Phase 3 clinical trials combining anti-PD-1 with three different checkpoints, CTLA-4, LAG-3 and anti-TIGIT -- all negative. And so we have not had any improvement in the adjuvant setting over and above anti-PD-1.

Sorry. Go back again.

And what I presented today was really asking the oncology world: How did we get here to reflect on how we got here in 2026. And there really is over 100 years of work in immunology, cancer biology -- and the technological innovation that's happened, particularly in the last 60 years.

And so if you're looking at where we are today with intismeran, there are three really important scientific advances:

First of all, the discovery that neoantigens exist back in the 1980s. These are unique mutations that are only found in an individual's tumor. They are not found in their normal cells. And they are not found in anyone else's cancer.

And that begs the question: If they're so unique, could they be seen as foreign to that patient's immune system?

Then, the second biggest scientific advance is really built on next-generation sequencing. And that's the ability to rapidly sequence the tumor and also use the computational algorithms to identify and rank which neoantigens we think or we predict will give us the most robust anti-tumor response.

And then, the third is a platform that really is built for speed. And that's the fact that mRNA -- you can include many multiple antigens in a single strand of mRNA.

And there's a demonstrated robust CD4 and CD8 tumor response or responses to these proteins. That's wrapped in a lipid nanoparticle, injected into the patient. So, altogether, that is a very rapid individualized manufacturing process, giving us intismeran.

Intismeran, as you heard before, encodes up to 34 neoantigens. And this is based on the patient's individual, what we call, tumor mutanome and their HLA type.

The workflow for Intismeran is standardized from collecting the tumor tissue and blood right around to administration of -- to the patient takes six to eight weeks.

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In terms of the mechanism of action -- this is now a busy slide without the animations -- but -- so it's injected intramuscularly. The mRNA is then taken up by nearby cells -- all sorts of cells. Antigen-presenting cells take this up, as well.

They process the protein; present the protein as neoantigens, in this case; and elicit a T cell response: clonal expansion, migration to the tumor, and then, tumor cell kill. And it is hypothesized that, with the release of more epitopes, as the tumor is killed, that this enhances that cycle of immune activation.

(inaudible - microphone inaccessible) -- you've just seen the data for the Phase 2b trial, just to reiterate that the clinical benefit is durable out to five years.

Left is relapse-free survival. So this was the proof of concept. Right is distant metastasis-free survival, hazard ratio of [0.410] at the last analysis -- and 0.510 and 0.410.

But, most importantly, is that there is translational data, which you've just seen, which linked the T cell clonal increase to the lack of relapse and that that was mapped precisely to the neoantigens in the intismeran. So I will not go through that in any more detail than -- as we've already heard.

So this is the design. This is a standard adjuvant-trial design in terms of eligibility -- resected cutaneous melanoma, AJCC 8th edition; stage 2b right up to stage 4 -- these are high-risk patients of recurrence.

Each patient had to have formal and fixed paraffin-embedded tissue that was of sufficient quality and quantity for intismeran manufacture, which required next-generation sequencing and then, no prior systemic therapy.

Patients were randomized 2:1 to the active arm of Intismeran 1-milligram intramuscular every three weeks for nine doses plus standard dosing of pembrolizumab every six weeks versus placebo and standard pembrolizumab.

The pembrolizumab was started, first, at randomization within 13 weeks of resection. The intismeran was started as soon as it was available to the site, the earliest was cycle 1, day 22; and the latest was cycle 4, day 1.

The primary endpoint was the relapse-free survival, as per the investigator. The secondary endpoints are shown here but the key one for today is a distant metastasis-free survival.

The statistical considerations are very important because, first of all, we had hierarchical testing, which is pretty standard for these large Phase 3 trials. If the relapse-free survival is positive, you then move on with power to the distant metastasis-free survival then to the overall survival.

The treatment differences, just to see if they are significant, was tested by the log-rank test. That doesn't tell you the size. It just tells you that it's statistically significantly different.

The size or the magnitude of the treatment difference is measured using the hazard ratio. For the primary endpoint, it was the Cox model, which is a standard approach in the intent-to-treat population.

However, because of that delay in the separation that we saw in the Phase 2, there were pre-planned sensitivity analysis to look at the robustness of these results. And one is called the piece-wise hazard ratios.

And I really do think that this trial will be the first to set this as one of the first standards for immuno-oncology because this is not a new phenomenon that you may have a little bit of a later separation.

And then, the other thing is the modified intent-to-treat. And that is all people who actually got the placebo or the intismeran.

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The interim analysis was conducted when we reached at least 240 recurrence events and at least 18 months since the last patient was randomized. And this is 24.6 months of median follow-up at the data cut.

So 1,749 patients were randomized -- sorry -- were screened; 1,137, randomized. The biggest reason, the most common reason for screen failure was due to inadequate tissue supplied by the site.

And I can talk about that in a minute -- because I was one of the sites -- how did we learn from that experience so that we became very efficient at getting the right amount of tissue.

757 patients were randomized to the combination, 380 to pembrolizumab alone. In both arms, one patient didn't receive treatment, the rest all did.

Then, when you move to the addition of the placebo intismeran that was equal in both arms, 96% of patients received placebo or their intismeran in each arm.

70% completed treatment, similar in both arms. But the reason was different for discontinuation. In the combination arm, it was adverse events. But in the pembrolizumab arm, it was due to recurrence that they didn't continue treatment.

So 91% of all patients in the trial had the maximum of 34 neoantigens identified for the intismeran design. Over 99% of those randomized to the combination -we were able to -- when I say we, Moderna -- to successfully manufacture the intismeran.

The median time -- and this is really important to describe those curves -- to first dose of intismeran was 64 days.

Baseline characteristics is what we would expect for this population and similar in both arms. The most common stage was stage 3, which is what we would expect and the least common, stage 4, which is also what we would expect.

So this is the Kaplan-Meier analysis for the recurrence-free survival. In the green is the combination. The combination of pembrolizumab with Intismeran significantly improved the recurrence-free survival over pembrolizumab alone.

And the hazard ratio overall was [0.580], with a p-value of less than [0.001]. So this represents an overall 42% reduction in the risk of recurrence or death with the combination of a single agent. I've also got the two-year landmark relapse-free survival, with [82.2%] and 70%.

Now, looking at the piece-wise hazard ratios, as expected, in the first six months, the hazard ratio was [0.99]. And then, after six months, the hazard ratio was at [0.42]. So a 58% reduction in risk of recurrence or death after six months.

And the reason this was done -- as I'll reiterate, I said it before -- is the mechanism of action. I know David said it, as well.

The mechanism of action of intismeran is at the early stage of antigen presentation. So that requires time for the T cells to get activated. But, secondly, the median time to injection of intismeran was nine weeks.

This is the modified intent-to-treat. So this is all patients who actually received placebo or the vaccine or Intismeran. And the hazard ratio was 0.510 so a 49% reduction in the risk of recurrence or death.

Now, looking at the distant metastasis-free survival -- one of the key secondary endpoints -- this was the second in the hierarchy. It was significant, as well, with the combination hazard ratio of [0.62] and a p-value of [0.002]. So a 38% reduction in the risk of distant metastases or death with the combination.

Similar sensitivity analysis, hazard ratio in the first six months, [1.01]; hazard ratio after six months, [0.50] so a 50% reduction in the risk of distant metastases or death. And the confidence intervals after six months do not cross 1.

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The sensitivity and the modified intent-to-treat population, hazard ratio of [0.55]. That's a 45% reduction in the risk of distant metastases or death in the two-year landmarks for distant metastases -- are shown there.

In the pre-specified sub-groups, nearly all of them favored the combination over pembrolizumab alone. There are some groups that are very small numbers.

When you have very small numbers and you have very small numbers of events, the confidence interval is very wide but it also means the estimate so the diamond can switch like this with one patient.

So you really have to be careful at looking any information with wide confidence intervals. We don't. We don't at this early juncture at this first interim analysis.

Also, what was really very nice to see was in the risk stratification by stage. So it's risk-based. We saw both groups benefit from the combination.

In terms of adverse events, the left are what we call all-cause adverse events. So, as per trial protocols, there are a lot of things that we call adverse events that have nothing to do with the actual treatment. And then, on the right are the treatment-related adverse events. And so I'm going to focus in on those.

There was a slight higher rate of all-grade treatment-related adverse events in the combination, which is what we'd expect. Intismeran does cause local adverse events.

But, importantly, when we look at grade 3 to 5 or serious adverse events, they were broadly similar in both arms.

In terms of adverse events that led to discontinuation, this was higher in the combination arm compared to pembrolizumab. And the deaths -- the two deaths -- due to treatment in the combination arm all occurred before intismeran was commenced so they were due to pembrolizumab.

The eight deaths, due to all-cause adverse events, not related to treatment -- only the two I just explained -- five of them occurred before the intismeran started and three after; and were not related to either treatment.

These plots are really nice because they quickly visualize where you're seeing the adverse events. So this is all-cause adverse events in more than 10%.

And what we've boxed there are those that are greater-10% incidents in the combination because that's what we want to know. Well, what is this combination doing?

Well, what was really interesting is they're very low -grade. They are all grade 1 or 2. They're very transient -- injection-site pain, chills, pyrexia or fever, injection-site reaction. And these were of no concern to the investigators looking after these patients.

When we look at the pembrolizumab or checkpoint-inhibitor adverse events, you can see they're broadly similar in both groups. So what we can say is that it does not appear that intismeran is potentiating pembrolizumab-adverse events.

So the conclusions, focusing in on the efficacy first, the primary endpoint and the key secondary endpoints demonstrated that the combination did reduce the risk of recurrence or death over pembrolizumab alone by 42%, overall, and 38% for distant metastases in death.

If we look at the sensitivity analysis, the combination reduced the risk of recurrence or death by 58% after month 6. The benefit was observed in almost every pre-specified sub-group.

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The overall survival is immature. And so a follow-up -- this trial is ongoing. We are continuing to collect data on our patients who continue to remain blinded, as are we.

Safety was consistent with what we've seen before in the stage 2. No potentiation of pembrolizumab, toxicities, or adverse events.

Intismeran was reliably delivered at a global scale. The trial is evidence of that. And there are ongoing trials in many cancers in the neoadjuvant, adjuvant, and metastatic setting.

So this is not as bold as it could be but INTerpath-001 really does set the new benchmark for melanoma. But, critically, it is a huge advance. It is a landmark for the advance in personalized cancer medicine.

Thank you. I just want to also make note of all of the people, all the work that goes into this trial -- lots of sites, a data-safety monitoring board, the support from the sponsors; and, as well, Jeff Weber -- we always mention him -- he was a huge supporter, had a leading role in the Phase 2, and he actually contributed patients and enrolled them right up to his death in '24.

And this will be published in the New England Journal of Medicine. Thank you.

David Berman - Moderna, Inc. - Chief Development Officer

Thank you very much, Dr. Long.

One group I also want to thank and note is Merck. I've been at two large pharma companies that have had intense competition with Merck. And I can say it's been absolutely fantastic to be collaborating with Merck for once, rather than competing with them. They're absolutely excellent partners.

So just a couple of words in conclusion:

Beyond melanoma, there still remains an unmet need across many different adjuvant settings, as you can see here. And we have intismeran clinical trials in all of these tumor types.

Now, one of the common questions that is frequently asked is: Will intismeran work outside of melanoma? And we have the trials ongoing. But here are the reasons why I walk away believing that it does work:

First, there's the science. All of these tumors, except for pancreatic cancer, are PD-1 sensitive. PD-1 -- anti-PD-1s activate T cells. And T cells kill cancer.

Intismeran also activates T cells but it does it on the front end. And, in fact, we know, based on the T cell clones, that Intismeran activates different T cells against different neoantigens than pembrolizumab dose.

Second is the data. So you heard from Dr. Long today, the [ITT], the hazard ratio of 0.580. If you remember, the famous KEYNOTE-054, which was KEYTRUDA versus placebo, their hazard ratio was [0.570]. So the fact that KEYTRUDA then went on to have success in many other tumors, I think, bodes very well for Intismeran.

You heard about the modified intent-to-treat, which means this is the ideal clinical-trial setting, limited to only the patients who received placebo or who received Intismeran. So it excludes 46 patients who never made it to receive an injection. When you look at that comparison, which is the ideal clinical trial, the hazard ratio is 0.510.

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And, finally, you heard about the piecewise. So this is patients who are six months and longer. This treatment effect, this hazard ratio of 0.42 tells you what is possible for intismeran. It's not the top line, it's not the ITT, but it tells you, scientifically, what treatment effect is possible with this drug.

I really do believe Intismeran will work, but of course, we need to run the trials. There's one more reason why I have belief intismeran will work outside of melanoma. And that's the tumor-mutational burden analysis.

And, by the way, in case you're going to ask, we don't have that data yet for the Phase 3 trial but, hopefully, soon.

In the Phase 2 trial, we analyzed RFS by high TMB and non-high TMB or low TMB. And, interestingly, the RFS was identical in the intismeran plus -- or the hazard ratio was identical in both the high TMB and the low TMB.

If you draw an imaginary box on the right for low-TMB melanoma, you see it does cross most of the tumors that we're studying.

One of the tumors, of course, I'd like to draw your attention to is pancreatic cancer -- pancreatic ductal adenocarcinoma -- because we do have a poster tomorrow. And I just want to share data in the abstract because I don't like to leak data before it's presented.

So this is a Phase 1 study of 20 patients who had resection of their pancreatic tumor. And the goal was, really, to see feasibility. Can we actually create an intismeran-drug product from a pancreatic-tumor sample, which is low TMB? And then, we also were curious: How does intismeran interact with chemotherapy.

And here's the key data from the abstract -- once again, full data tomorrow -- 20 patients. They had low TMB. 65% had KRAS mutation. The median number of neoantigens per intismeran cassette was actually [34]. So we actually did pretty well.

Of the 19 of the 20 who were immunogenicity evaluable -- and all 19 had neoantigen-specific T cell responses seen. It's very promising that we can induce T cell responses.

In the sub-set who were evaluable, we could see polyfunctional T cell responses. And, interestingly, the T cell response increased over time, as we had seen. But there does seem to be an interaction with chemotherapy. And the intismeran-induced T cells go up even further after the FOLFIRINOX is complete.

So it is feasible and possible to do this. And full poster, to be presented tomorrow.

Now, I'd like to invite up our experts to come and join here. And we certainly open this up for questions and answers.

Oh, actually, before I do -- sorry. Sorry. Before I do -- sorry, just let me close on this. And then, actually, I want to invite up our two experts to give some thoughts and comments.

So you saw the melanoma today. The three lung-cancer trials, one of them should be finished randomizing soon. The non-pathologic complete response, post-neoadjuvant, is still enrolling, as is the stage 1.RCC in -- was the first to complete enrolling. And the data read-out, the interim analysis, will be event-driven.

Bladder cancer is fully enrolled. And we expect to have an event-driven read-out, as well. And then, non-muscle invasive-bladder cancer, it's still enrolling, as is first-line melanoma and first-line lung cancer. And then, we have solid tumors.

Now, I'm going to invite up our two experts, Professor Ascierto, who I have had the pleasure of collaborating with across four different companies; and then, Professor Ribas, who is one of the world's experts in immunotherapy. So maybe you can (multiple speakers) --

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Antoni Ribas - University of California, Los Angeles - Professor - Medicine, Surgery, and Molecular and Medical Pharmacology

We have got the pleasure of –competing with each other for four different companies. And we're still friends.

David Berman - Moderna, Inc. - Chief Development Officer

I didn't (multiple speakers) -- yeah. Yeah.

Actually, just as an interesting side note, the first investigator I ever visited for -- when I joined BMS was Jeff Weber on an ipilimumab trial. And Dr. Ribas here was the lead of the tremelimumab trial.

And the science was always better for tremelimumab. It's a big program. They did a great job.

So maybe I could ask Professor Ascierto just to give some clinical context. And then, maybe you could just give some of your immunotherapy expert context, as well.

And then, we'll open up for questions.

Paolo Ascierto - Istituto Nazionale Tumori IRCCS Fondazione Pascale - Director - Department of Melanoma, Cancer Immunotherapy and Developmental Therapeutics

Yeah. So how many time I have? So just to set -- 5, 10 minutes. That's good. Okay. Take some notes.

So, first of all, good evening, everybody. My name is Paolo Ascierto. I'm a medical oncologist. I work in Naples, Italy.

And the daily practice -- before to speak about daily practice, the first question that probably discussed on -- should discuss -- was, it will be intismeran, the new standard of care -- intismeran plus pembrolizumab because this is an important question; and if it will be for the stage 2b and 4 because this is -- the patient's population enrolled.

And Georgina showed you in the forest plot, this is effective for all the groups at the moment.

So if we look, the data from anti-PD-1 BRAF, MEK in stage 3, stage 2b, 2c, we have only anti-PD-1, the data in terms of relapse-free survival at five years looks similar with double trauma and anti-PD-1. So we cannot say that there is one better than another one.

So if we have, in terms of relapse-free survival, a treatment better than anti-PD-1, I guess that this will be the new standard of care in the high-risk patients' population.

Now, the discussion about adjuvant and neoadjuvant, that was the topic that was discussed by the discussant.

So in clinical practice, in real life, I see in my institution, per month, 60 patients -- high-risk melanoma patients, Stage 2b, 4, resected, that could be treated with adjuvant.

So, among these 60 patients, I see, probably, 10 patients, stage 3, with clinical palpable lymph node. That is the population that can be treated with neoadjuvant or perioperative approach.

So, in the daily practice, that population is a population where we can treat with the neoadjuvant. I strongly support neoadjuvant, as Georgina knows, but it's a limited number of patients.

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LSEG DATA & ANALYTICS

The big group are the patients with sentiment node-positive that we cannot treat with neoadjuvant. We need to treat with adjuvant. So it's probably, surely, the higher number of patients that we will treat with adjuvant.

And, now, with this new data, surely, more patients will be treated with this regimen because we have seen the data.

What is really important with this data was pointed out by Dave: the consistency of the data -- Phase 2, Phase 3. I have seen consistency of data with ipilimumab, you remember.

If you look at the survival curve of CTLA-4, it's always the same in the different clinical trial. If you look at the survival curve of the sequence, BRAF, MEK and CTLA-4, PD-1 is the same in (inaudible) -- so consistency is the key to better understand.

And I believe you will see in the future interim analysis. By interim analysis, the data will improve, like we have seen for the KEYNOTE-942 (inaudible).

Second, what is really exciting with this new treatment is that, after three trial that failed -- and Georgina show it: the combination of PD-1, CTLA-4; the combination PD-1, LAG-3; the combination of PDL, anti-TIGIT.

Why? Because in the field of adjuvant, what is important is the minimal residual disease. We don't have the tumor -- from an immunological point of view is totally different from the condition where you have the tumor, metastatic disease and neoadjuvant.

So with the minimum residual disease, we don't need to add checkpoint, checkpoint, checkpoint. We need to improve the priming. And messenger RNA vaccine is able to improve the priming. This is the key in the mechanism of action for the results that we have seen.

I believe that I said about everything that I wanted to tell.

Antoni Ribas - University of California, Los Angeles - Professor - Medicine, Surgery, and Molecular and Medical Pharmacology

So it's perfect to build up on what Paolo was just finished on, which is the reason why are these curves separated? Why do we have a hazard ratio of 0.58.

And the difference is because you stimulate more immune cells against what the immune system uses to differentiate between cancer cells from normal cells. That's the key to all kinds of therapies.

Can you get a therapy that attacks the cancer cells and specifically spares the normal cells?

By doing the process that Georgina has explained -- and David -- about sequencing the tumor, knowing the sequences that are different from the cancer cells from the normal cells; making an mRNA vaccine, like, for every patient, you then are able to stimulate -- to turn on more T cells that recognize the cancer.

And then, with pembrolizumab, they get unleashed. If we get pembrolizumab alone, which is the control group, you're only relying on the immune cells that the cancer has turned on by itself.

Immuno-cancers are not -- they don't want to be turning on T cells. They want to hide from them. So this is a way to use those neoantigen sequences and turn on much more in T cells.

I'm excited to have Georgina present the data today. I'm excited to see that big room and overflow rooms of all these people coming to see -- to look at the melanoma presentation.

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This is a landmark in cancer-drug development, which is -- we've been -- as Georgina said, for over 100 years, people have tried to inject somebody to patients with cancer to increase the immune response against it.

Now, we have a rational design platform that make feasible, validated by the billions of people who received the COVID-19 vaccines, and personalized to this level of precision; and then, positive clinical data.

QUESTIONS AND ANSWERS

David Berman - Moderna, Inc. - Chief Development Officer

Great. So, Georgina, can we invite you up now? Sorry. And we're happy to take questions online or in person.

Unidentified Company Representative

(Event Instructions)

Salveen Richter - The Goldman Sachs Group, Inc. - Analyst

Salveen Richter, Goldman Sachs.

Could you put the data in the context of the competitor regimens, including the neoadjuvant; and the pros and cons that were laid out by the discussant?

And maybe walk us through how [INT] really is positioned, based on that totality, to become standard of care.

Georgina Long - Melanoma Institute Australia - Medical Director

Yeah. Well, it is not unusual that you have multiple tools in the toolkit that are developed together. That's not -- we see this all the time in cancer.

So the next steps are: How do we integrate the two tools that are doing really well?

The one thing about neoadjuvant is we get a very early read-out at six weeks as to those who are not doing well with neoadjuvant treatment and checkpoint inhibitors. I refer to that group as the third space. My colleagues have heard me say that.

But that's the group of people who are dying of cancer. In melanoma, it's those who are resistant to all the checkpoints and checkpoint combinations. So neoadjuvant gives us an early read-out. They are still people in the third space.

So one natural question is -- well, Intismeran in that group -- we know from the Phase 2 study, for example, that the group that benefited the most based on biomarkers at baseline -- so so lower tumor mutation burden, no PD-L1 expression. They actually had the greatest benefit by the addition of intismeran.

So the next few years are about us sorting that out in this field, how we're going to combine two excellent treatments.

We are not worried about that. (multiple speakers) -- we were not worried about integrating and doing this work. They work very differently.

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The Intismeran is completely different from the checkpoints. They're very complementary. So it's about working out what we give (inaudible) -- to which patients.

However, as Paolo beautifully explained -- is that not a lot of patients in that stage 2b to stage 4, resectable, the population in INTerpath-001, fit the [NADINA] population exactly.

Paolo Ascierto - Istituto Nazionale Tumori IRCCS Fondazione Pascale - Director - Department of Melanoma, Cancer Immunotherapy and Developmental Therapeutics

If I can add something?

Just coming back to the discussant, the right study for a formal comparison is not NADINA, it's the [SWOG] because, in the SWOG, we have the pembrolizumab, we have the perioperative, okay?

Probably, if I was discussant and to discuss this aspect, I would have been used the SWOG trial more than NADINA.

Terence Flynn - Morgan Stanley & Co Ltd - Analyst

Terence Flynn, Morgan Stanley. A couple of questions.

One is just for the physicians. Maybe, the likelihood that these data translate to an [OS] benefit. I think we're still waiting to see that for KEYTRUDA
but just confidence level there?

And then, maybe for Dr. Long, one question we've gotten is, just, investigator versus central review. So I know in a lot studies you do, typically, central review but, here, it was investigator review -- and how to think about that in the context of functional unblinding. That's another question we've got.

Paolo Ascierto - Istituto Nazionale Tumori IRCCS Fondazione Pascale - Director - Department of Melanoma, Cancer Immunotherapy and Developmental Therapeutics

Okay. So about the overall survival, we see. As Georgina showed, there is a hierarchical concept in the study, study design. RFS, these are metastasis-free survival. And we see overall survival.

In general, we consider the distant metastasis-free survival a very interesting endpoint because it's close to the overall survival. We have seen that it's positive.

And if we consider the Phase 2 improved year by year -- and, probably, I believe and I feel, the overall survival will be positive, as well.

It's my personal feeling, of course.

David Berman - Moderna, Inc. - Chief Development Officer

Can I add just one other point on that? And then, we can go to Dr. Long.

One of the things also to keep in mind is, for all of the anti-PD-1 adjuvant trials, there was a rescue anti-PD-1 at time of catastases. There's no rescue intismeran at time of progression, just as a small point.

Do you want to talk about the -- was there a functional blinding? You -- actually, you and [Paolo], probably, two largest enrollers.

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Georgina Long - Melanoma Institute Australia - Medical Director

We just had a discussion about this because we are finding that independent blinded review can be very problematic, particularly with immunotherapies. And immunotherapies is a big -- it's a big umbrella of many treatments but checkpoint is one type.

Now, we have individualized neoantigen therapy; of which, Intismeran is one.

In that the radiologists that do the blinded central review have to be very well trained and have to understand these drugs.

And so, recently, there have been trials which would have been positive, had the radiologist been trained appropriately to interpret, oh, everything's shrinking but the lymph nodes are growing; oh, that's progression. No. These are immune-stimulated lymph nodes that don't actually have cancer.

So I even proposed yesterday or the day before: Can we just get rid of blinded independent review? So that's just that point.

So, in this, it does not bother me at all. I don't know if it's a concern that you're expressing. I think the relapse-free survival -- it was done in 157 global sites. So you're going to -- everything is going to come out in the wash anyway.

We were blinded. So we didn't know what patients were receiving. And I think that speaks most highly.

And the other thing is, this is an adjuvant trial. So it's not like you need the radiologist to watch and see how things are shrinking and growing. They just need to pick up a recurrence. And that, technically, is much easier than monitoring multiple metastases in the setting of immuno-oncology.

So I don't know if that was your question. But it doesn't worry us, put it that way, in terms of the strength of these results, one iota.

David Berman - Moderna, Inc. - Chief Development Officer

I want to add to what Georgina is saying, the local radiologists have more (inaudible - microphone inaccessible) -- ability to query if it's a questionable finding.

And, sometimes, they have more parts of the body, more higher resolution than the central radiologists.

Also, in some of our centers, our radiologists are the local radiologists and the central radiologists at night. And I would query how they report differently.

Unidentified Participant

This is [Dina], from UBS, on -- for behalf of Mike Yee. Just two quick questions.

You reminded us kindly that, in the Phase 2, we saw a really great benefit in the PD-1-negative population BRAF and also in the low-tumor-mutation burden.

How do you think about that benefit translating into the Phase 3 -- I'm sure we'll see that sub-group data -- if you do have it or have any color on that. But how do you see the right population to treat, first and foremost, based on those biomarkers?

And then, on the back of the conversation for neoadjuvant, is there maybe a scenario in the real-world practice where, if there are patients that do get neoadjuvant checkpoint could they go on to adjuvant therapy and do get INT and KEYTRUDA?

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Georgina Long - Melanoma Institute Australia - Medical Director

Yeah.

Unidentified Participant

And so I just wanted to get your thoughts about that.

Paolo Ascierto - Istituto Nazionale Tumori IRCCS Fondazione Pascale - Director - Department of Melanoma, Cancer Immunotherapy and Developmental Therapeutics

Sorry. The first question is about biomarker. So, at the moment, we don't have biomarker. No PD-1. No TMB.

In the field of adjuvant, the only biomarker that was explored -- but in the target therapy -- was interferon-gamma signature that Georgina reported years ago but it's not for the clinical practice. It's still something in the science.

So, at the moment, we cannot predict patients who can get adjuvant and benefit from another patient.

The second was -- the second question?

David Berman - Moderna, Inc. - Chief Development Officer

Can intismeran eventually be administered after neoadjuvant therapy?

Paolo Ascierto - Istituto Nazionale Tumori IRCCS Fondazione Pascale - Director - Department of Melanoma, Cancer Immunotherapy and Developmental Therapeutics

So, again, the neoadjuvant patient is just 20% of the patients, okay? Having said that, now, with this data, of course, there will be a clinical study in order to explore all the several things, even in the neoadjuvant and, of course, in the adjuvant, which will be the patients that should receive adjuvant.

Pathological complete response? No. But all the other? Probably, yes.

And, now, if the standard of care is, this combination, this will be the best option for patient.

Georgina Long - Melanoma Institute Australia - Medical Director

I was just going to add to your very first question. And I think what you were asking is, since we were seeing a greater benefit, is that going to -- no. We see a benefit in all groups.

The point I was making with that is, it would be easy for some people to say, oh, it's higher-tumor-mutation burden, easy to identify neoantigens. Also, that's who's going to benefit.

My point there was not necessarily -- they do benefit. We know that. The data shows that. But, hey, we've got patients with lower-tumor-mutation burden in the pancreatic and we've got patients with no PD-L1 that are benefiting. So that was the point. It's not to pick out a population.

At this point, as Paolo said, there is no population. It's -- every sub-group we've looked at seems to be benefiting. So that's that answer.

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Paolo Ascierto - Istituto Nazionale Tumori IRCCS Fondazione Pascale - Director - Department of Melanoma, Cancer Immunotherapy and Developmental Therapeutics

And, again, we're increasing the priming. So the priming is something different from the effector phase.

Unidentified Company Representative

Okay. So before we go to the next live question, I'm going to take a question from online:

To the physicians, what's the best way to rationalize why the hazard ratio was much more impressive for RFS versus [DMFS] at this interim?

Georgina Long - Melanoma Institute Australia - Medical Director

That's usually standard. So, if you look at most trials, you will always see that trend -- Phase 3 landmark trials. You see relapse-free survival is often a greater hazard ratio at very first interim analysis than distant metastasis-free survival.

So what we saw in the Phase 2, it was actually a little unusual. So it is standard that that's what we see. We are not surprised by this data. It's exactly as we expected.

Well, we were actually super impressed by this data is what I should say. (multiple speakers) -- would you support that?

Paolo Ascierto - Istituto Nazionale Tumori IRCCS Fondazione Pascale - Director - Department of Melanoma, Cancer Immunotherapy and Developmental Therapeutics

No. Yes. I agree with you. We need time because we need the event at the moment. So I'm sure that, in the next follow-up, we will see an improvement.

David Berman - Moderna, Inc. - Chief Development Officer

Yeah. And just to add, this trial, of course, included stage 2 patients who have a slower time to distant metastasis.

And the additional point is, if you look at the modified intent-to-treat, which is patients who got placebo or INT injection -- so people actually received something -- there, the hazard ratio was 0.55.

So I think those are the two things to keep in mind. Next question?

Luisa Hector - Joh Berenberg Gossler & Co KG - Analyst

Luisa Hector, from Berenberg. And wonderful to see this data so thank you. So I have a question on the tissue sampling -- the 17% of the samples that were too small.

I think, Dr. Long, you implied that you were learning through the trial how to do that better. So how much could that be reduced? And anything you can add on that?

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Georgina Long - Melanoma Institute Australia - Medical Director

Yeah. I can't tell you how much that will be reduced but it will be reduced. My own experience -- and I'd love to hear the other investigators -- was the first few patients we screened, some got on but some, we didn't supply enough tissue. So we had to go back to the blocks and give more tissue.

So what I did is I got the team together in our pathologists and we had that a protocol that worked for our site because of the way our site works in our pathology.

And, then we didn't have screen failures after that. We were able to pick patients so that we had enough tissue upfront because it's not fun for a patient to consent; and then, we send the tissue, it's not enough; and then, have to wait longer. And so we really wanted to get that right upfront.

This is not new to oncology. You probably remember 15 years ago when eGFR inhibitors in lung cancer -- tissue was the issue. It's not an issue anymore because we've got infrastructure and resolved those issues.

Luisa Hector - Joh Berenberg Gossler & Co KG - Analyst

And then, it may be too early to comment -- I don't know how much data you have -- but in the patients who are recurring, is there anything about the T cell clones or any pattern you can see from that?

Georgina Long - Melanoma Institute Australia - Medical Director

I don't know if I explained it (inaudible - microphone inaccessible) --?

Paolo Ascierto - Istituto Nazionale Tumori IRCCS Fondazione Pascale - Director - Department of Melanoma, Cancer Immunotherapy and Developmental Therapeutics

But they're blinded, still. They're blinded, the patients. So I will not know.

Georgina Long - Melanoma Institute Australia - Medical Director

But we did look at recurrence in the Phase 2. And I thought we presented that. I can't remember.

But, anyway, we did, didn't we? Yeah, yeah. So there is some data in the Phase 2.

Eliana Merle - Barclays Services Corp - Analyst

Ellie Merle, from Barclays. Just on a similar vein, you mentioned that there were some neoantigens that are more promising at inducing T cell responses, any learning, so far, from the data on which ones might be?

And then, you've also shown that the patients with more novel T cell clones have greater clinical benefit. Any thoughts or early hypotheses on which patients might be more likely to have more novel T cell clones versus others?

And the reason for these questions is how you interpret this data in terms of what it could mean for success in other tumor types?

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David Berman - Moderna, Inc. - Chief Development Officer

I'm going to answer that question. And then, I'm going to invite Toni to maybe give a scientific answer.

That's an area -- both of those are areas of active investigation on what makes a good neoantigen. Now that we have clinical efficacy outcome, we can go back. We actually have neoantigen prediction for both the control arm and for the experimental arm.

And which and how you can understand -- and your question was about which patients do get novel T cells so one is about neoantigens and is patient-level question? Is that the -- yeah. That's another great question.

Toni, you've been doing this for a couple of decades. What are your thoughts?

Antoni Ribas - University of California, Los Angeles - Professor - Medicine, Surgery, and Molecular and Medical Pharmacology

Yeah. I have trouble trying to answer the question directly because we do not have the way to measure, count how many T cells to neoantigen HLA presented peptides.

It's -- these are the most polygenic areas of our genome between the T cell receptor. It's like an antibody that recombines -- and then, the HLA Class 1, which there's one more common, HLA 2.1.

But there's 24,000 HLA Class 1 alleles in the human population so far. There's -- the [TCRs] are up to 10 to the 15, the potential sequences.

A long explanation to say that the measurements that we have are associations with what the real thing is happening and that I don't think, in any realistic time point, we'll be able to answer your question directly, which is the key question on any neoantigen therapy.

(inaudible) -- said this, the efforts that have been made are very substantial at Moderna and Merck, to be able to study from the Phase 2 trial -- and see that the vaccine, the personalized therapy, and neoantigen therapy can turn on T cell receptor prototypes that were not there.

And you would assume that some of them will be to the mRNA because that's -- the protein made from the mRNA will be the most immunogenic part.

So could we then decide which -- what's the best algorithm to get those T cells that work the best? I think that's a question that, in clinical trials, I don't think -- large clinical trials may not be realistic about.

Unidentified Participant 2

This is Greg Torres, representing Tyler Van Buren from TD Cowen. Congrats on this outcome.

So given the apparent INT-specific T cell responses you've shown, how seriously should we think about the potential for INT monotherapy? And could removing pembro ultimately expand the addressable population more than adding INT onto pembro, particularly in stage 2 patients?

David Berman - Moderna, Inc. - Chief Development Officer

I'm going to take that one. And then, of course, I invite Toni and the others.

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I personally believe -- there's been a belief out there that, in immunotherapy -- for a novel immunotherapy, you have to add on to an anti-PD-1 to work. And a lot of that has come from the fact that a lot of novel immunotherapies have not had any significant monotherapy activity and, therefore, that's that belief.

I don't really think that's case. I personally believe that intismeran should have monotherapy activity. And, as you know, we are studying it in stage 1 lung cancer.

And part of the reason is that first slide I showed, which is, evolutionary speaking, the immune system evolved to detect and eliminate viruses. But it can also detect and elate cancer.

It doesn't require an anti-PD-1 to do that. We don't walk around with anti-PD-1 antibodies. And so, if we can stimulate those T cells, they should be able to do what T cells normally do.

So this is hypothetical speaking. We do have some intismeran monotherapy data showing that we can induce T cells.

But, Toni, what are your thoughts? And we're in the speculation phase now because we don't have the data but what are your thoughts? Intismeran monotherapy.

Antoni Ribas - University of California, Los Angeles - Professor - Medicine, Surgery, and Molecular and Medical Pharmacology

So if you have more -- the field has realized that if you have more T cells for cancer, those patients do better. And that is, again, by very crude measurements.

But we know from studies in colon cancer from 20 years ago that the colon cancers that have more T cells, that was like being a stage lower in the HHCC staging.

So more T cells is better independent, if you block any checkpoint.

Georgina Long - Melanoma Institute Australia - Medical Director

Prognostically.

Antoni Ribas - University of California, Los Angeles - Professor - Medicine, Surgery, and Molecular and Medical Pharmacology

Prognostically. But that tells us that, if you stimulate them with INT, as you're saying, you're probably going to do better.

In the earlier stage, you're right that you start worrying about the pembro side effects and hypothyroidism that can be long term and adrenal insufficiency or (inaudible) -- the rare patient that has really, really bad side effects. You don't want to risk that.

So it's great that you're testing on stage 1 lung cancer. I think it's a great study.

Georgina Long - Melanoma Institute Australia - Medical Director

Yeah. I was going to say the same thing. I think, hypothetically, given the mode of action where it's acting the algorithm, what we've seen so far, it should have some activity in earlier stages and in a single agent.

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Yeah. (inaudible) -- the issue is in something like melanoma, stage 1, which is very common -- stage 1 melanoma -- is only about 5% will recur. So we need to team things like that up with tests that tell us who's going to recur and then, give them these sorts of treatments to stimulate their T cells.

But, theoretically, it should work.

Unidentified Company Representative

So a few other questions coming from those who are listening online. And apologies if these are basic questions.

But to the physicians, are you concerned about the KM curves crossing at 33 months for DMFS? (technical difficulty) -- that's one question.

Of course, Georgina, please do speak to it. And then, I have a few others.

Georgina Long - Melanoma Institute Australia - Medical Director

(multiple speakers) -- it means -- it's nothing. It's -- you'll see, there were several Kaplan-Meier curves today in other trials where you see that. It is a phenomenon of the follow-up and the fact that when you get out to 33 months, when you go to a median follow-up of 24 months, you don't have any patients at risk.

So the curves are not to be looked at. A rule of thumb for those who are not in oncology, if you've got less than 10% of patients at the number at risk, you cannot judge the shape of the curve. It will change at next analysis, guaranteed.

So that does not worry us. We see this all the time at these sorts of follow-up early on. It's nothing to be concerned about.

Antoni Ribas - University of California, Los Angeles - Professor - Medicine, Surgery, and Molecular and Medical Pharmacology

But maybe -- let me know. Could we put the slide back?

David Berman - Moderna, Inc. - Chief Development Officer

Slide 37.

Antoni Ribas - University of California, Los Angeles - Professor - Medicine, Surgery, and Molecular and Medical Pharmacology

Because I think this is one experiment that took many years.

David Berman - Moderna, Inc. - Chief Development Officer

Oh. Slide 36.

Antoni Ribas - University of California, Los Angeles - Professor - Medicine, Surgery, and Molecular and Medical Pharmacology

I think it's important to get the figure right. Otherwise, you have it in your hand-outs.

So, the curves don't separate the first six months, as Georgina well explained. Then, they start separating.

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And if you look at the bottom of the Kaplan-Meier -- has two rows of numbers.

Georgina Long - Melanoma Institute Australia - Medical Director

That's the number of (multiple speakers) was talking about. (multiple speakers) -- keep going, Toni. Keep going.

Antoni Ribas - University of California, Los Angeles - Professor - Medicine, Surgery, and Molecular and Medical Pharmacology

Yeah. So, here, the patients' at risk? And then, the tick marks are the sensor patients. So can we go to the primary analysis, go to the one that says -- backwards more, backwards more.

Georgina Long - Melanoma Institute Australia - Medical Director

The RFS.

Antoni Ribas - University of California, Los Angeles - Professor - Medicine, Surgery, and Molecular and Medical Pharmacology

Here. (multiple speakers) --

Georgina Long - Melanoma Institute Australia - Medical Director

They're the same curves. They're the same curves as (multiple speakers) --

---

Antoni Ribas - University of California, Los Angeles - Professor - Medicine, Surgery, and Molecular and Medical Pharmacology

So they don't separate for six months. They start separating. There's 600 patients at risk in one group, 200 on the other.

And then, you get to 24 months, you see all these tick marks. And then, at the end, there's only 35 -- there's 10 patients -- followed them one or the other.

That's when it's not reliable. But we're here trying -- we're trying to interpret this and interpret the future or anticipate what will happen in the future.

Somebody asked about overall survival. All these curves are separating until there's not enough patients at risk. You would assume they'll continue to separate it. That's just logical.

David Berman - Moderna, Inc. - Chief Development Officer

A lot of enthusiasm on this. And, actually, we have this exact example from the primary analysis on slide 10 of the Phase 2. Remember the curves come together in the primary analysis because there's too few patients. In the longer follow-up, that disappears -- exactly what they said.

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Georgina Long - Melanoma Institute Australia - Medical Director

All of those tick marks are patients who are alive and well, who have not had an event, but they're just alive and well and they've got to 24 months. So they've all got to come through the curve, so to speak.

So it is completely unstable from, as I said, 10% of the starting number. So [75, 7] -- basically, once you get up below 75 patients, ignore it. It's going to change. It's going to do this. And it doesn't tell you anything about the science or the patients. It's not reliable.

Great.

Can we ram that [home] any other way?

Unidentified Participant 2

Yes. Sorry. Thank you. Thank you for education.

One last question on RFS as a primary end point versus OS in the adjuvant setting: Is OS required for approval here?

Paolo Ascierto - Istituto Nazionale Tumori IRCCS Fondazione Pascale - Director - Department of Melanoma, Cancer Immunotherapy and Developmental Therapeutics

This is a question that -- it's usual when we talk about adjuvant. So it's clear now, after a long discussion, that relapse-free survival, it's an important endpoint that we should consider, when we discuss with patients.

Now, [Christian Blank] is almost Italian. And, during the meeting, we have a funny fight about neoadjuvant and adjuvant.

And the question is, of course, about relapse-free survival and overall survival.

The point is, if we assume -- a regulatory agency assume the relapse-free survival, it's a good end point; and for me and colleagues, it's a good end point, stop.

Overall survival is important. We know from a scientific point of view. But when we discuss with the patients, the patients will not be relapse-free.

David Berman - Moderna, Inc. - Chief Development Officer

Great. Thank you very much. I'd like to thank three experts. It's been a busy day. And thank you for taking the time. Thank you, all, for attending today.

Georgina Long - Melanoma Institute Australia - Medical Director

Thank you.

David Berman - Moderna, Inc. - Chief Development Officer

Toni, thank you so much. I really appreciate you coming here.

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LSEG DATA & ANALYTICS

Antoni Ribas - University of California, Los Angeles - Professor - Medicine, Surgery, and Molecular and Medical Pharmacology

Absolutely.

David Berman - Moderna, Inc. - Chief Development Officer

Thank you so much.

Unidentified Company Representative

Thank you.

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