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ADAP - Adaptimmune Therapeutics plc - Special Call

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PRESENTATION

Operator

Good day, ladies and gentlemen, and welcome to the safety update from Adaptimmune conference call. (Operator Instructions) I would now like to turn the call over to Juli Miller, Investor Relations. Please, go ahead.

Juli P. Miller - *Adaptimmune Therapeutics plc - Director of IR*

Good morning, and welcome to Adaptimmune's conference call to discuss our clinical update here at ASCO as well as our ongoing MAGE-A4 study. We issued a press release detailing our updated MRCLS data on Saturday and 2 additional press releases earlier this morning. I would ask you to refer to the full text of our forward-looking statements there.

As a brief reminder, we anticipate making projections during this call, and actual results could differ materially due to a number of factors, including those outlined in our latest filings with the SEC.

James Noble, our Chief Executive Officer; and Rafael Amado, our Chief Medical Officer are with me for the prepared portion of this call and will be available for Q&A. Also joining us for Q&A is Dr. Helen Tayton-Martin, our Chief Business Officer.

With that, I'll turn the call over to James Noble. James?

James Julian Noble - *Adaptimmune Therapeutics plc - CEO and Director*

Thank you, Juli, and good morning, everyone, and thank you for joining us. ASCO has been a very exciting time for immuno-oncology and Adaptimmune, in particular. Today, we are presenting a poster with detailed safety data from patients treated in the 100 million safety cell dose cohorts in the 2 ongoing MAGE-A10 pilot studies, namely the triple tumor study in bladder, melanoma and head and neck cancers and other study in non-small cell lung cancer. In brief, as reported before, there was no evidence of off-target toxicity, and we are now dosing patients at the next dose level of 1 billion transfused cells in both studies. We remain on track to report response data later this year.

Moving on to MAGE-A4, and independent of our data at ASCO, we are announcing today that the safety review committee met at ASCO and recommended dose escalation from 100 million to the next dose 1 billion cells to the basket study. As in the MAGE-A10 studies, there was no evidence of off-target toxicity at 100 million cells, and overall safety was favorable to support dose escalation.

In addition, we are announcing today after confirming expression levels of MAGE-A4 from synovial sarcoma and myxoid/round cell liposarcoma or MRCLS tumor samples, we have amended the study to add these 2 indications.

Thirdly, during oral presentation on Saturday, Dr. Sandra D'Angelo from Memorial Sloan-Kettering shared the results of the ongoing pilot study of NY-ESO SPEAR T-cells in MRCLS, and Rafael will now speak to this in more detail. Rafael?

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

Thank you, James, and thanks everyone for joining us this morning. As Jim said, the MRCLS data continues to be promising and demonstrates that these tumors are sensitive to SPEAR T-cells. In this study, patients had a median of 4 prior lines of systemic therapy, and therefore, have limited therapeutic options. Of note, the preconditioning regimen that was used in this trial was a lower-intensity regimen of Cytoxan and fludarabine than that that was used in Cohort 1 of the synovial sarcoma study. We now know that this is a suboptimal conditioning compared to Cohort 1.

As Dr. D'Angelo presented on Saturday, out of 7 patients assessed to date, the best overall response includes 3 confirmed partial responses, 1 partial response to be confirmed and 3 patients with stable disease. And 8 patients have recently been treated and is awaiting assessment. Of these patients, 3 have since progressed, 2 of whom had best overall response of stable disease and 1 who was a responder.

There was an overall trend in tumor burden decrease among the majority of patients, which range from approximately 17% to 50% across target lesions. Although that are preliminary with only 7 patients evaluated to date, there appears to be a correlation between SPEAR T-cell persistence and response. We also see clear infiltration of SPEAR T-cells as well as endogenous T-cells into tumors post treatment and this is the first demonstration to our knowledge of this phenomenon. This is further validation that T-cells bearing affinity-matured TCRs contracted into previously noninflamed or occult tumors and exert antitumor responses.

Safety also remains encouraging with favorable (inaudible) of tolerability. There was one event of cytokine release syndrome that was Stage III and the patient was treated with tocilizumab and CRS event resolved 6 days post infusion.

There were 4 SAE reported by 3 patients that included the aforementioned Grade 3 CRS, 2 Grade 2 CRS events and a Grade 2 event of pleural effusion. All of these events resolved. Overall, most adverse events were consistent with those typically experienced by cancer undergoing -- cancer patients undergoing cytotoxic chemotherapy or other cancer immunotherapy. The results in MRCLS bode well for the broad therapeutic potential of our SPEAR T-cells across multiple solid tumors.

Now moving on to MAGE-A10. We have a poster today summarizing the safety data from the first 8 patients treated in the 100 million cell safety dose cohort from our 2 ongoing pilot studies. Among these patients, we have established that there is no off-target toxicity at 100 million cells, but that this dose is insufficient to lead to high-peak SPEAR T-cell expansion. These data are consistent with data in the NY-ESO synovial sarcoma program where we saw limited expansion and poor persistence at less than 1 billion SPEAR T-cells. We're now treating patients in MAGE-A10 pilot studies, with 1 billion transduced cells, which, as we said, was the lower limit of the therapeutic dose of (inaudible) sarcoma. We remain on track to complete this second dose cohort of 1 billion cells in the MAGE-A10 pilot studies as planned, and we'll move on to a third cohort if there is good tolerability.

As Jim said, we also announced today that we have dose escalated to 1 billion cells in our ongoing MAGE-A4 basket study. There was no evidence of off-target toxicity in patients who received the 100 million SPEAR T-cells in this first dose cohort. And the overall safety profile was favorable enough to support dose escalation.

We also announced that we have added 2 additional indications to this study, namely MRCLS or myxoid/round cell liposarcoma and synovial sarcoma. We routinely test all our tumor samples from our ongoing trials for our target antigens and it was confirmed that MAGE-A4 is expressed

in synovial sarcoma and MRCLS. We are very encouraged about our MAGE-A4 program as this target is broadly expressed and has shown optimal potency and specificity in our preclinical validation test.

I also wanted to take the opportunity to discuss some changes in overall study design we made to our ongoing Phase I/II studies, including MAGE-A10 and MAGE-A4. First, now that we have long-term data from our synovial sarcoma study, in particularly comparing varying preconditioning regimens of fludarabine doses, we have determined that this data supports the use of higher doses of fludarabine with respect to SPEAR T-cell engraftment and expansion. And based on these observations, the MAGE-A4 and MAGE-A10 studies have been amended to include an additional data of fludarabine given the preconditioning in the third cohort, and we have also expanded the maximum dose to 10 billion SPEAR T-cells for this cohort and for the expansion phase of this trial.

Overall, we're very excited by our data. We continue to see validation of our preclinical safety testing platform as there have been no evidence of off-target toxicity with either the MAGE-A10 or MAGE-A4 SPEAR T-cells at doses that are 100 million cells.

Data have also confirmed that doses higher than 100 million cells are required for antitumor activity.

Therefore, moving on to the second- and third-dose cohorts of 1 billion cells and beyond is very exciting and has the potential to deliver response and data throughout the remainder of 2018.

And now we'll turn the call back to James. James?

James Julian Noble - *Adaptimmune Therapeutics plc - CEO and Director*

Thank you, Rafael. These are very exciting times for Adaptimmune. We now have clearance to [indiscernible] what we hope are therapeutic doses (inaudible)assets and we are on track for response data in the second half of 2018. In addition, I'm excited to have added synovial sarcoma and MRCLS to the MAGE-A4 basket study because we have already demonstrated these malignancies are responsive to SPEAR T-cell therapy with NY-ESO. We continue to see promising response data from our NY-ESO SPEAR T-cells, and we now know that MAGE-A4 expression is high in both synovial sarcoma and MRCLS. So with that and -- with that encouraging note, I would like to open the call up for questions. Operator?

QUESTIONS AND ANSWERS

Operator

(Operator Instructions) And our first question is from Reni Benjamin with Raymond James.

Reni John Benjamin - *Raymond James & Associates, Inc., Research Division - Senior Biotechnology Analyst*

Congratulations on the progress. Maybe, we can start off by just talking a little bit more about the response rates that you're seeing in MRCLS. I understand these are small numbers, but can you just expand a little bit more on that drop in response rate? It seems like from the prepared remarks, it all seems to be focused on the preconditioning regimen, but just any additional color that you have there? And can you also talk about the duration of response from those patients that progressed?

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

Yes, Reni, this is Rafael. So as you mentioned, this is a series of very few cases. So we've been very cautious about talking about response rates in aggregate until we have more patients and more durability. But we have been looking for trends in those patients that exhibited brisk responses that appear to be durable like the patient that is a responder and continues -- tumors continue to respond out to 24 weeks and those that had a brief response and progress. Of the 7 patients evaluated, the 3 progressers received around 1 billion cells; so 1, 1.02, and 1.05 billion cells. And the

rest of the patients have received between 4 and 5 billion cells. And I think that has to do more with the evolution of manufacturing at the time that these patients were being treated. So we really think that cell dose is important. Now that is one variable that dichotomizes these 2 groups pretty clearly. We have looked at expression and that doesn't seem to be a factor. There are patients with 3 plus 100% of the cells that have progressed, some patients with much less than that that have had a good response. So I think what we need to do is to continue to follow these patients, and ideally these patients should be conditioned with a higher more dosing terms of fludarabine regimen and aim for higher doses to try to get the optimal outcome for them.

Reni John Benjamin - *Raymond James & Associates, Inc., Research Division - Senior Biotechnology Analyst*

Got it. And then just related to that. Can you just expand a little bit more on the kind of additional data of fludarabine? I understand it's based on the synovial sarcoma data, but maybe you can just refresh our memory as to why that -- you feel that might be the optimal strategy versus 2 days or a different strategy and may be the rationale for going up to 10 billion cells versus something in between?

James Julian Noble - *Adaptimmune Therapeutics plc - CEO and Director*

Yes. So these are actually questions that pertain to what we have learned from our NY-ESO soft tissue sarcoma study, the synovial sarcoma study. And there, we looked at expression as a variable, and we looked at conditioning as a variable. We didn't look at cell dose as a variable, but we obtained data from cell dose just based on what numbers patients received. And so one thing that has become clear with time as the data matures is that the comparison of Cohort 4 with Cohort 1. So Cohort 1 received a high dose intensity fludarabine, cyclophosphamide, and Cohort 4 receives like half the dose of, as I said, cyclophosphamide and one less day of fludarabine. That comparison shows an inferior response rate for Cohort 4. And we're going to show this data later on in a scientific meeting. But more importantly, the patients' durability of response was shorter. And we've done a lot of corollary studies to try to look at cytokine expression and so on really to determine whether a day of fludarabine affects the (inaudible) population of T-cells and it really does. So we're not going to increase cyclophosphamide to the levels of Cohort 1 because that accounts for most of the [myelosuppression] and probably not -- does not very much for engraftment. But what we'll do is, is add one more day of fludarabine, so it will be 600 milligrams per meter squared every day for 3 days and 30 milligrams per meter squared fludarabine every day for 4 days. And that is a regimen that's very similar to the CAR regimen, that more dosing in CAR regimen that's worth its uses. So that's one of the lessons, I think, that's come out very clear from a sarcoma study. In terms of cell dose, we know that there is a ceiling -- sorry, that there is a floor around 1 billion. And even though we're seeing responses surrounding 1 billion, those responses tend to be short. And interestingly enough, we -- in Cohort 1 where we dose a bunch of patients with cell doses of about 1 billion-or-so and these were patients that had responded and then lost their response and would still have material and they still express the antigen, and we -- I think we saw was only one response around 4 or 5 patients that we treated. And more recently, we treated 5 patients, and we are dosing up 4 or 5 billion cells, we're redosing these patients, and in the first 5, we've got response in 3, and 2 of them have a partial response and one of them has a complete response. So it's again telling us that cell dose really matters in this space, and because now we have our own manufacturing facility where we can routinely make 5 billion cells, we're going to aim to give those kinds of doses and that was the rationale to try to test out to 10 billion cells in these pilot trials.

Operator

Our next question is from Peter Lawson with SunTrust Robinson.

Max Lewin - *SunTrust Robinson Humphrey, Inc., Research Division - Associate*

This is Max Lewin, in for Peter Lawson. I was just wondering if you could give us a little bit more color on what might have triggered the CRS in the patients that got it since it seems that it wasn't due to an antitumor effect.

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

So if I understand the question is whether CRS had occurred in patients that did not have antitumor effects?

Max Lewin - *SunTrust Robinson Humphrey, Inc., Research Division - Associate*

Yes. That was indicated by the press release. I was wondering if you could give us some detail on what was the trigger for that CRS.

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

All these patients have measurable disease, and we think the CRS has to do with cell expansion in the presence of antigen. So the CRS -- Grade 3 CRS was a patient that had quite bulky disease, and it was also complicated by arrhythmias because the tumor was located in the mediastinum. And the other patients also had some degree of tumor shrinkage. So we don't believe that there has been CRS in the absence of response in this study.

Max Lewin - *SunTrust Robinson Humphrey, Inc., Research Division - Associate*

Okay. And I was also wondering if you could give us any more details on the patient who received 90 million cells. What was the situation there?

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

In the MRCLS study? You mean, they're getting at 100 million cells. That's just the manufacturing. I'll have that. There is actually a range around 100 million cells, as it happens one of the patients got 90 million cells rather than exactly 100 million cells. It's just a manufacturing offset. It wasn't intentional. We did put it in the detail.

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

Yes. The range -- the lower bound in that cohort is 0.8. So the patient qualified with 90 million cells.

Operator

Our next question is from Robyn Karnauskas with Citi.

Robyn Karnauskas - *Citigroup Inc, Research Division - Director and Senior Analyst*

This is a couple. Do we -- and first of all, do we understand exactly why you have to go up in cell dose. Is there anything going on in the T-cells, are they getting tired or exhausted or are they just being absorbed by the tumor, why it should go up so high? And then, is there anything you could do to modify that, so you could use low dose? And then the third question is on manufacturing. What is the significance that you had to get really high into the billions rapidly?

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

The manufacturing.

James Julian Noble - *Adaptimmune Therapeutics plc - CEO and Director*

Yes, so the manufacturing. The question is, we have -- that was all manufacturing, actually it's within our own facility. The only ones we manufacture are the MAGE-A4. And as Rafael said, we, in our own facility are consistently getting above 5 billion cells. There was clearly a problem with the manufacture of that particular dose. Sometimes you get lower cell numbers coming out of the other end. It was a problem with that particular

manufacturing dose. I have to say that we do not see any of those problems recently. We -- as I said, we routinely get more than 5 billion transfused cells. We don't anticipate that particular issue happening again. And we've now got permission in our own pilot plant to make MAGE-A10 and MAGE-A4 and AFP's. And we have days where we're in a position that we should routinely be able to make very substantial numbers of cells. I think that problem is behind us and in future zone.

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

I think with respect to the question of the composition of the cell in the manufactured product, there's a mixture of cell types that mostly affect our memory cells. So as you would imagine, because they're activated by CD28, CD3 with the beads. And then, as time goes on, those cell compartments shift more towards the stem cell phenotype. So the cells that persist are stem cell memory phenotype cells that we cannot harvest from the periphery, we can expose them to antigen, and they do react and actually kill. So the cells are basically capable of reacting to antigens. With measure of markers of the soft skin like TIM-3 and LAG-3 and PD-1 and actually -- they're absent in the cells. So these cells are perfectly functional. So the numbers gain may have to do more with the cells having to traffic to pass 2 other passes, having to penetrate the matrix of the tumor and essentially not having ready access to antigen like they do in the CAR studies where there are hematologic malignancies and fewer cells can fight antigens quite quickly. So this is a hypothesis, but it's a finding that seems to be (inaudible) now.

James Julian Noble - *Adaptimmune Therapeutics plc - CEO and Director*

Just going back to the number of cells and the preconditioning. We are, obviously, trying to get the maximum durability and the maximum response rate from patients. So where we see -- obviously there is no statistic to deal on these, there are too few patients. So where we see an observation, the 5 billion looks better than 1 billion than logically everybody get a 10 billion and see if that's better than 1.05 billion. So there's nothing to read into it beyond that. We are, as a company, trying to maximize the durability, which is why we changed the preconditioning and maximized the response rate and the depth of response by carrying on, going up with the cell dose, simply on observations, which is not statistical, there is obviously -- there is no stat finding, but the observation does look as though where you give more cells, you get a better response. So it's not really beyond that, it's just that attempt to maximize the depth of durability response.

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

And then I just want to emphasize the 1 billion is probably sufficient to see its signal. So we are still pretty excited about the fact that we're dosing in Cohort 2 at 1 billion cells. It's just that perhaps the durability is affected by giving less than 1 billion cells. And sort of our thinking that Cohort 2 may start to deliver efficacy data that idea has not changed.

Robyn Karnauskas - *Citigroup Inc, Research Division - Director and Senior Analyst*

And just a follow-up. So if you learn from these initial experiments that you really do have to dose up to 5 billion or 10 billion. Is there any mechanism, by which you can accelerate dose escalation in other studies at some point where you'll able to be start higher for other indications or you still have to go through this whole process?

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

That's an excellent question. And that's another thing that we've learned from our MAGE-A10 and MAGE-A4 studies is that we don't want to expose patients to insufficient cell doses in the future. And so we are exploring for our next targets alternative study designs and maybe that we use intrapatient dose escalation and may be that we'll start at a higher dose, maybe 500 million cells. We probably will -- we will dose some of the patients that are -- that if they don't respond to the initial cells administered in the early cohorts, but those studies are very unlikely due to have the same structure of treating a number of patients at a dose that we think is too low.

Operator

Our next question is from Jonathan Chang with Leerink Partners.

Wei Ji Chang - *Leerink Partners LLC, Research Division - Director of Biotechnology & Senior Research Analyst*

First question. Can you expand on your decision to add the 2 cohorts to the MAGE-A4 basket study and can you provide any more specific color in terms of what you're learning about MAGE-A4 expression overall?

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

Well, the first point is that we know that these tumors are sensitive to SPEAR T-cells. And so if they express MAGE-A4, the bar for efficacy in our view would be lower because we already know that they are sensitive to this technology. So that prompted us to look. We have a lot of samples fortunately from synovial sarcoma and MRCLS patients, and there was literature suggesting that this marker was expressed. So we've done a lot of work on samples, but also on samples that we purchased on microarray -- tissue microarray and we've arrived at rates of about 70% in synovial sarcoma and 50% to 60% in MRCLS, and that's with a high expression threshold. So it seems to be relatively similar to NY-ESO. And so because this is a Phase I and it's an exploratory study and we have multiple tumor types, it just makes sense to include these patients as well and see whether they are sensitive to MAGE-A4 because it is, obviously, as I said before, a sensitive tumor to this technology. So the rationale, it's 1 now out of 9 tumor types that we can enroll in this trial.

Wei Ji Chang - *Leerink Partners LLC, Research Division - Director of Biotechnology & Senior Research Analyst*

Okay, great. And just one other question for the NY-ESO update at ASCO. Can you elaborate on the handful of patients that were not treated, were there any issues of manufacturing for those patients?

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

So there were 3 patients that were not treated. There were more patients that were not enrolled, and there were (inaudible) and they met criteria, but they were in another therapy, so they weren't ready or they didn't have measurable disease. So they didn't come into the study. They may still come in to the study later on. We are still -- the study is still open. The 3 patients, one of them withdrew, the one other was very ill and progressive prior to treatment. And actually, there was a third patient that also withdrew. So we don't have much detail on those patients that withdrew. We have 2 patients that are awaiting treatment at the moment, and they do have measurable disease and the products are being made. Recall this trial was supposed to have a total of 10 patients and may be up to 15 to replace patients that dropped out. So we don't have a lot of slots open. And these are studies that will transition to GlaxoSmithKline. So the fate of the study will eventually be the decision of GSK. But there are some thoughts that perhaps this study should continue and changes to the condition should be made, but those decisions will be made by GSK.

Operator

Our next question is from Marc Frahm with Cowen.

Marc Alan Frahm - *Cowen and Company, LLC, Research Division - VP*

Just kind of following up on your responses to Robyn's questions. When you're going -- if we clear the 1 billion cell dose and you're going to go to 10 billion, your ability to kind of expand the 1 billion while you clear the safety at 10 billion, so you can, maybe, more fully answer this question of dose and maybe consider also the -- to your point of tumor burden and antigen access may be impacting how many cells you need, in different tumor types, you may actually see different doses needed?

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

The cohorts are relatively small. It's only 3 patients. And so that's one factor. The other factor is that in the Cohort 1 for both studies, for the 3 studies rather, there's an observation period of 4 weeks. So it makes it very cumbersome and slow because we have to coordinate with centers when the patients are ready, do they happen to be ready when there is a slot and so on. So now that we've crossed Cohort 1, the observation period is 2 weeks. So it's a lot easier to plan. And it is only 3 patients, and we are currently dosing at 1 billion cells, so we are very close as well to move to Cohort 3. So we may not have time to do what you mentioned in Cohort 2. And Cohort 3 is trading to up 10 billion, but we'll exercise judgment on how we dose. So the first patient in Cohort 3 is probably not going to receive 10 billion cells. And so I thought it was 5 billion. And we'll start enrolling -- and if there are no DLT's that Cohort will close with 3 patients. And then, we'll be in the expansion cohort where we will really see the higher-dose patients and see how those perform. If we are lucky to get a diversity of patients, we may be able to make conclusions as to whether the cell dose matters by histology. I'm hopeful that, that may happen, but the numbers, I suspect, will be small for us to be able to make strong conclusions on histology and cell doses.

Marc Alan Frahm - *Cowen and Company, LLC, Research Division - VP*

I got it. But there is no plan to say because 1 billion might be cleared, just start to think they can -- 1 billion -- patients at 1 billion while you clear higher doses?

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

I'm not sure I understand the question.

Marc Alan Frahm - *Cowen and Company, LLC, Research Division - VP*

So we essentially run expansion cohorts at moderate doses while you clear higher doses for safety?

James Julian Noble - *Adaptimmune Therapeutics plc - CEO and Director*

Yes. The study design is -- actually it's probably faster than you're thinking. We are dosing patients at 1 billion, but since we dose 3 at 1 billion cells, we'll go to the first cohort which is, as Rafael said, 5 to 10 billion. So we will get there very, very quickly. We should do.

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

And the expansion cohort cannot start until the cell cohort has cleared.

Marc Alan Frahm - *Cowen and Company, LLC, Research Division - VP*

Okay. And then, maybe back to the A4, actually -- I assume you would have disclosed if there was severe CRS seen in anybody, but have you seen kind of mild CRS or evidence of kind of that the cells are at least expanding relatively well?

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

So we haven't disclosed the profile. Obviously, that there wasn't anything that safety review committee deemed an adverse event serious enough to meet the definition of DLT and precludes dose escalation. We have seen expansion of those cells. And I guess, I can say with a few samples that

we have that the expansion looks or compares favorable to the expansion that we saw with MAGE-A10. I think that's about all that we can say at the moment.

Operator

Our next question is from Ying Huang with Bank of America.

Jenny M. Leeds - *BofA Merrill Lynch, Research Division - Research Analyst*

This is Jenny on for Ying. I'm just wondering with 3 patients and third dose in cohort will be enough to see a difference in responses from 5 billion to 10 billion in this cohort?

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

So recall there is an expansion cohort at the top dose. So if the third cohort is cleared, then we can treat up to 30 patients. Actually, because we added 2 more tumor types, we are now 40 patients. So we have opportunity to see a lot more patients, although we won't be measuring DLT for the purpose of dose escalation because there won't be additional dose cohorts, obviously, we'll be collecting safety and efficacy. And so we'll learn from this dose expansion. If we see efficacy, we'll probably amend the study to really hone into the tumor type and understand what the nature of the efficacy is, the nature of the signal. So I'm pretty confident that we will get good data at the various dose levels. And not every patient is going to get 10 billion. I mean, this also depends on how we're manufacturing goes. The rates of -- the average rates are closer to 5 billion than to 10 billion. But what we want to do is for patients that actually, we manage to manufacture 10 billion, not to administer only 5 billion, administer as many cells as we're able to make with the (inaudible).

James Julian Noble - *Adaptimmune Therapeutics plc - CEO and Director*

Yes, just to be clear that while we get into dose expansion, the cohort is limited to 3 per dose. But as soon as we up dose escalation rather, but as soon as we get to the top dose we can then -- we can dose up to 30 originally in that 40 patients. So there will be sufficient patients. And clearly, we see responses in the given tumor, we'll expand that cohort in any case to get more information. So if the design is actually relatively modest in numbers to start with, but we'll get much bigger as we get it through.

Jenny M. Leeds - *BofA Merrill Lynch, Research Division - Research Analyst*

Got it. It's very clear. And then, if you do have to dose at 10 billion and potentially add another round of expansion in your cell manufacturing, how much longer would that make your manufacturing time from where it is currently?

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

Won't extend the manufacturing time. That was just -- this is biology and the starting material is the biggest variable with manufacturing because obviously each starting material comes from a different patient. We routinely are getting 5 billion cells. And we've actually got occasionally a lot more than 10 billion cells. So it's not actually necessary the length of the manufacture because if we extend the length of the manufacture, the potency of the cells could be affected and the other attributes of the cells can be affected. So we -- there are lots of things you can do to increase the number of cells, you can increase the amount of vector, you can increase the number of cells you put into the starting feeding cells. And then -- so it's a -- it's not to do with the length of manufacture, it is to do with the method of manufacture and the starting material is more -- so we're very -- the manufacturing at Navy Yard has gone extremely well. I think, we've made at least a dozen, maybe 15 doses now, and we routinely get the sort of 5 billion. We sometimes get 10 billion. But we know how to go up and it wouldn't extend the manufacturing time because that will change the nature of the cells.

Operator

And we have a follow-up question from Reni Benjamin with Raymond James.

Reni John Benjamin - *Raymond James & Associates, Inc., Research Division - Senior Biotechnology Analyst*

I guess just Rafael based on some of your comments regarding the redosed patients. Can you talk a little bit about how many patients across the studies have been redosed? Kind of what leads to that redosing decision? Is it primarily response based or is it based on the number of cells you might have remaining? And I guess, as we think about redosing, have you started thinking about redosing protocols and how that might look?

Rafael Amado - *Adaptimmune Therapeutics plc - Chief Medical Officer*

Yes. The redose has started in Cohort 1, and that's already built into the protocol. And it has treated a bunch of patients at a low dose. The first study that was done at the NCI by Dr. Rosenberg they redosed some patients. And they saw 1 or 2 responses, if I recall, so it was not really very meaningful compared to the initial responses. Then when we opened Cohorts 2, 3 and 4 because the possibility of lower response is compared because these were lower expressors with less chemotherapy, we wanted to give the opportunity of redosing with higher chemotherapy. And so the characteristics of those that are redosed are as follows. So they have to have had at least stable disease. Most of them have responded before. Then they need to have a biopsy, and the tumor needs to still express the antigen. And then, it's done upon request from the physician. So we don't select the patients. And so lately, we've had a lot of requests from Cohort 3 where it was cyclophosphamide alone, but mostly from Cohort 4, and those patients can be conditioned either with Cohort 4 condition or Cohort 1 condition and that depends on whether or not they responded in the past to Cohort 4. And so as I said, we've treated the 5 patients and one of the patients had a complete response. That patient had responded for 4 weeks before and then one of the nodules started to grow -- one of the lung nodules, and the physician wanted to retreat. And then got a follow-up CT scan where the nodule appeared smaller and then eventually disappeared. Again -- and you're highlighting these responses are gradual. They've not seen prominent responses that happen at the first scan. And then, the other 2 patients that we've treated have had partial responses. So again we think that the infusion may be important, some of these patients still had circulating SPEAR T-cells, but somehow they needed activation or may be the myelosuppression allowed them to expand and grow. And so reinfusion may be a way to reignite that response in some of these patients. And we want to explore this further. We're not redosing right now in the MRCLS study. That's something that could be considered.

Operator

And I'm showing no further questions. I would now like to turn the call back to James Noble for any further remarks.

James Julian Noble - *Adaptimmune Therapeutics plc - CEO and Director*

Yes, thanks very much. I think it's a fantastic and exciting time for the company to have recorded a second complete response in a solid tumor is quite an achievement, we think. We're very excited to have that. We're very excited about the fact that we now have a second type of solid tumor, the MRCLS data coming through with a number of confirmed responses already. I think, we're also delighted in the MAGE-A4 study, in particular, to be going up the dose and Safety Review Committee, obviously, having to make that decision on Saturday, and that we're able to go up the dose and also add these 2 SPEAR T-cells sensitive tumors MRCLS and synovial sarcoma into the mix. So I'm very confident that we're going to be on track for what we've said, which is that we will get response data in the second half of 2018. I very much look forward to reporting those data as we get through the rest of the year. Thank you very much.

Operator

Ladies and gentlemen, thank you for participating in today's conference. You may now disconnect. Everyone, have a great day.