Adaptimmune Therapeutics plc(Q1 2020)

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Corporate Speakers:

- Juli Miller; Adaptimmune Therapeutics plc; Senior Director of IR
- Adrian Rawcliffe; Adaptimmune Therapeutics plc; CEO, Principal Accounting Officer & Director
- Elliot Norry; Adaptimmune Therapeutics plc; Senior VP & Chief Medical Officer
- Helen Tayton-Martin; Adaptimmune Therapeutics plc; Co-Founder & Chief Business Officer

Participants:

- Marc Frahm; Cowen and Company, LLC; Research Division, Director
- Tony Butler; Roth Capital Partners, LLC; Research Division, MD, Senior Equity Analyst & Head of Biotechnology Research
- Mohit Bansal; Citigroup Inc; Research Division, VP and Analyst
- Nicholas Abbott; Wells Fargo Securities, LLC; Research Division, Director & Associate Analyst
- David Ruch; SVB Leerink LLC; Research Division, Associate
- Mara Goldstein; Mizuho Securities USA LLC; Research Division, MD of Equity Research Department

PRESENTATION

Operator^ Ladies and gentlemen, thank you for standing by. Welcome to the Adaptimmune Q1 2020 Earnings Conference Call. (Operator Instructions) Please be advised that today's conference is being recorded. (Operator Instructions)

I would now like to hand the conference over to your speaker today, Ms. Juli Miller. Thank you. Please go ahead, ma'am.

Juli Miller^ Good morning. Welcome to Adaptimmune's conference call to discuss our first quarter 2020 financial results. We issued a press release earlier this morning. I would ask you to please review the full text of our forward-looking statements there.

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.

Adrian Rawcliffe, our CEO, is with me for the prepared portion of this call and other members of our management team will be available for Q&A. With that, I'll turn the call over to Adrian Rawcliffe. Ad?

Adrian Rawcliffe^ Thank you, Juli. Thank you, everyone, for joining us. Before I get started, I'd like to officially welcome Gavin Wood to his first Adaptimmune's earnings

call. Gavin joined us as Chief Financial Officer in early April. Looking back at the first quarter, the year began strongly for us.

In January, we announced responses in 4 new tumor indications. These are early responses, but they are a validation of our SPEAR T-cell platform for people with cancer. We are funded into the second half of 2021, having added close to $140 million to the balance sheet from our deal with Astellas and the public offering in January.

The January clinical data announcement was followed by increases in screening during the first quarter across all our clinical trials before the inevitable slowdown associated with COVID-19. The impact of COVID-19 is still evolving, including its impact on our R&D programs and the full extent is likely to vary across size.

As a company, we've implemented new ways of working together to reduce the risk of disease spread. We want to ensure the safety of our colleagues who are working on-site manufacturing SPEAR T-cells for patients enrolled in our clinical trials, all conducting time-critical research activities.

I want to thank all our employees, both those who have been working from home and those who have been working in our facilities to make sure that we can hit the ground running as the situation improves. Manufacturing of our SPEAR T-cells has continued with our facilities operating at close to full capacity throughout March.

We are still manufacturing for patients in April and May, albeit at a reduced level. And we're optimistic that patients screened and patients for whom we have manufactured cells during the pandemic, will be able to participate in our trials as clinical sites resume non-COVID-19 related care.

Although many scientific and medical meetings have been delayed or switched to virtual formats, we will continue to communicate our data at these meetings. In a press release issued on April 29, we laid out expectations of when you would see updates between now and August at ASGCT, ASCO and the International Liver Congress.

Earlier this week, we presented data from our allogeneic program at ASGCT. We've made great strides in the engineering required to make T-cells from stem cells, including the location and timing of edits to get our T-cell receptor into progenitor cells for the generation of T-cell banks.

We have also been able to produce functional, engineered T-cell from stem cells that can kill cancer targets in-vitro as effectively as engineered control cells from our autologous products.

These are significant steps in progressing our allogeneic platform towards the clinic. At ASCO, Dr. David Hong of MD Anderson Cancer Center will give an oral presentation with data from the Phase I trial for ADP-A2M4, our first-generation SPEAR T-cells targeting MAGE-A4.

This presentation will provide a full summary of the trial including more mature data from patients with synovial sarcoma as well as data from a broad range of other indications. The acceptance of this abstract as an oral presentation by ASCO is validation of the strength of our ADP-A2M4 program.

The full ASCO content will be available online, the morning of May 29, and we will issue a press release with a clinical update that same day. We will also be available in a virtual ASCO booth throughout the day of the 29th for Q&A.

We've learned a lot from the Phase I ADP-A2M4 trial. We've learned that MAGE-A4 is a viable target expressed in a broad range of tumors at levels high enough to garner responses.

We've learned that the TCR against MAGE-A4 successfully targets MAGE-A4 expressing cancer cells and can result in T-cell trafficking and responses in multiple tumor types. We've used this information to build out our program of assets targeting MAGE-A4.

These include our next-generation ADP-A2M4CD8 cells, which we are testing against a broad range of cancers in our SURPASS trial as well as the radiation sub-study of our Phase I trial.

And we are on track to present data from both these trials at medical conferences in the second half of 2020. This program also includes a Phase II combination trial with ADP-A2M4 and a PD-1 inhibitor for patients with head and neck cancer. This trial will be called SPEARHEAD-2. All clinical sites will be activated as soon as possible.

Our SPEARHEAD-1 trial in sarcoma was recently acknowledged by the EMEA's Committee for Orphan Medicinal Products with its adoption of a positive opinion for orphan drug designation for ADP-A2M4 for the treatment of soft tissue sarcomas. In Q4 2019, we announced that the FDA had granted orphan drug designation and then RMAT designation to this program.

ADP-A2M4 has clearly demonstrated the potential to offer substantial improvements over current standard of care for the treatment of advanced soft tissue sarcomas. With these 3 designations, we believe we're in a strong position to achieve our goal of launching our first product in the U.S. in 2022. Our commercial preparation is ramping up as we add key staff and develop our capabilities towards commercial scale.

Finally, we are progressing our ADP-A2AFP program in liver cancer, and we are delighted that Dr. Bruno Sangro from Navarra University Clinic in Spain will present data from the first dose cohort at the International Liver Conference in August, which was delayed from April. We continue to enroll this trial, having progressed to the expansion cohort at doses of up to 10 billion spare T-cells.

As I said earlier, 2020 started strong for us with the validation of our SPEAR T-cell platform in multiple solid tumors and our most significant deal in the past 5 years. Now I'll open the call up to questions. Operator?

QUESTIONS AND ANSWERS

Operator^ (Operator Instructions) Our first question comes from Marc Frahm with Cowen & Company.

Marc Frahm^ Maybe just to start off, you mentioned kind of the counterbalancing, the added interest in the program based on the updates in January, but then, of course, COVID had slowed things down. Can you maybe quantify that as to how much did you see screening increased maybe in February versus what you've been seeing in the back half of 2019?

And then when we get the update on the press release on the 29th, will that be restricted to MAGE-A4 or should we be thinking that that's a broader clinical update of whatever you have across kind of all the programs like you've done some other times?

Adrian Rawcliffe^ Okay. So I'll take the second question first, and then I will ask Elliot to comment on the first question and progress on the screening on the clinical trials in the first quarter of this year.

So I think the -- in terms of what we will give as a clinical update, I think it's just worth putting the whole thing in context of what's going to be presented at ASCO. David Hong's presentation will be on the all of the data we have to date on the patients enrolled in the ADP-A2M4 Phase I trial. You incorrectly stated this is the first generation SPEAR T-cells targeting MAGE-A4, and that trial has been ongoing for some time.

It's just worth noting that Dr. Hong and the team at MD Anderson have been key partners for us across the range of approaches that we're targeting MAGE-A4, including the radiation sub-study, the SURPASS second-generation trial, and you will recall that we had a strategic alliance with MD Anderson as well. So they are instrumental in treating patients across the range of these studies.

The first generation Phase I trial for ADP-A2M4 was recruiting across 9 different tumor types expressing MAGE-A4, including synovial sarcoma where we clearly recruited the most patients. Last year -- no, actually, 2 years ago at ESMO 2018, we showed safety from this.

You might remember the first 2 dose cohorts were full of ovarian patients, 6 ovarian patients dosed at 100 million and 3 billion cells. In May 2019, we showed some initial responses in a few sarcoma patients, and we showed signs of antitumor activity in other tumor types.

We updated ESMO and CTOS last year on the sarcoma patients, and roughly half of those patients were responding over that time course. Durability at that point was relatively immature. The data cutoff for CTOS was 23rd of October. And we basically use that data cut off to submit the ASCO abstract.

And then subsequently, we announced that we had seen a response in a head and neck patient alongside other responses in our other trials, including those targeting MAGE-A4, the radiation sub-study and the SURPASS study, which is the second-generation study.

We have continued to recruit patients through Q4 and into Q1 2020. Overall, we have dosed 38 patients in that study. The 6 ovarian patients in the first 2 dose-escalation cohorts, followed by 16 synovial sarcoma patients and 16 nonsarcoma patients.

Those nonsarcoma patients are split over a wide range of tumors including ovarian, head and neck, bladder, lung. And so you can be -- it's clear that there will be relatively small ends in each tumor type.

Dr. Hong's presentation basically will provide a full update on both the sarcoma patients, including durability of those responses and also what we've seen outside of sarcoma. And we're quite excited for that presentation. Following on from that presentation, we will give an update on the various trials targeting MAGE-A4.

So just to confirm, this includes an update on progress on SPEARHEAD-1, which is the Phase II trial targeting with -- in synovial sarcoma and myxoid round of liposarcoma. An update, i.e., more details on the design of the SPEARHEAD-2 trial. This is our Phase II trial in head and neck cancer in combination with pembrolizumab.

As I've mentioned above, we have seen a response in head and neck cancer in the first-generation program as a monotherapy. So we know that the SPEAR T-cells are active in this setting, and we're excited to initiate this trial this year.

An update also will be provided on SURPASS, our second-generation ADP-A2M4CD8 where we announced in January that the very first patient of this trial at the lowest dose cohort had responded.

Now I want to say -- remind everyone that we do plan not to stick to our practice of supplying data updates at major medical and scientific meetings. And so -- but I think we'll give an overview of progress on our entire MAGE-A4 targeting program on the 29th.

And just to point out, we have previously said that the AFP program will be updated at the International Liver Conference in -- now, which has moved from April to August, and Dr. Sangre will give that update on the third dose cohort.

So with that, I'll just ask Elliot to comment on the screening, the increase that we saw in the early part of this year. Obviously, I don't want to -- we're not going to give specific

details on how many patients, et cetera. But generally speaking, across our trials, Elliot, what color could you add there?

Elliot Norry^ Thank you, Ad. I would just say that there was a very palpable increase in interest in our studies from our investigators in conversations that we had with them. And that really translated into an increase in the actual number of screen patients.

As Adrian said, I'm not prepared to or don't think we should provide specific screening numbers as it relates to individual trials. But I don't think it's surprising that following the announcement of the responses that we had seen across tumor types and across our platforms that there was an increase in interest.

Operator^ Our next question comes from Tony Butler with Roth Capital.

Tony Butler^ Yes. Adrian, I just wanted to ask specifically about SPEARHEAD-1 and enrollment in SPEARHEAD-1. So there's really 2 parts to the question. One is, has enrollment continued at a pace that, let's say, doesn't necessarily protract the outcome of the finality of the trial appreciably, let's say?

And then number two, with respect to your comments, Elliot, back in January as it relates to an increased interest in clinical sites and investigators, was that skewed towards synovial sarcoma, just given the fact that you're enrolling a substantial number of patients within that particular disease setting as it relates to SPEARHEAD-1.

Adrian Rawcliffe^ Thanks, Tony. So in regards to the pace of recruitment on SPEARHEAD-1, I would not dispute your statement that what we're seeing continues to give us confidence that we'll be in a position to meet our objective of launching ADP-A2M4 patients with sarcoma in 2022.

There obviously has been some impact in the sort of mid-March onwards as it relates to screening for the SPEARHEAD-1 trial. But it is encouraging that we continue to screen a (inaudible) manufacturer for patients. And we will give an update on that in the April -- in the May 29 clinical update.

With regards to the balance of screening and interest, I would just say this. I think it's worth -- yes, the -- and lot of the patients that we have seen this year are for the SPEARHEAD-1 trial, not surprising.

That is a 60-patient trial. I mean it's much larger than our other trials. It's opening close to 20 centers at this point in time across Europe and North America, and it's on the back of compelling data, I believe, regarding the efficacy of ADP-A2M4 in synovial sarcoma.

However, I think I'll just give to Elliot to confirm that the -- we also saw on the back of the efficacy data, the response data that we put out in January. We also saw an uptick across our other trials and indications as well. Elliot, anything to add to that?

Elliot Norry^ Yes. I would just confirm that, Adrian, that we saw robust interest in enrollment really across the programs in the early part of the year. And as you said, I think that the interest in synovial sarcoma really mostly came from the data that was presented at ESMO and CTOS last year and just the general knowledge in the sarcoma community about the promise of this therapy.

The -- and we expected to have robust enrollment in that study at the beginning of the year as the centers were really opening. Where we really saw a change was in the other studies where it was on the back of the data we presented in January, across other tumor types that generated sort of a different type of interest in those programs. So I really -- in summary, the answer to the question is that it really was not restricted to synovial sarcoma, but seen across the programs.

Operator^ Our next question comes from Mohit Bansal with Citigroup.

Mohit Bansal^ Hope everyone is staying safe at team Adaptimmune. So just wanted to understand -- and so thanks for providing all the color on the ASCO data. Is it possible to put the ILC data, the liver meeting data as well in context and set expectations there? What should we expect? How many patients are there already? Anything you could provide to help us understand how to feed those data.

Adrian Rawcliffe^ Elliot, would you like to comment on what will be presented at ILC?

Elliot Norry^ So at the International Liver Congress, we will present the data for Cohort 3. Not going to announce any of that data in advance of that beyond what has already been communicated, which is the 100% reduction in target lesions seen in the first patient treated at the -- in cohort 3 with approximately 5 billion cell dose.

We also do have a -- host our presentation at the International Liver Congress discussing Cohorts 1 and 2 in summary, which we had -- that hadn't been updated in total. So there are really 2 presentations: one on Cohort 1 and 2 and then the presentation by Dr. Sangro on cohort 3.

Operator^ And our next question comes from Jim Birchenough with Wells Fargo.

Nicholas Abbott^ It's Nick on for Jim this morning. A couple of questions. The first one is, I know that you're going to be reviewing the design of SPEARHEAD-2 at virtual ASCO, but is there a reason that PD-1 dosing would just not follow the -- its label for head and neck cancer?

Is there -- I mean I guess I'm trying to get at is the some way you think you can optimize the function of the SPEAR T-cells with perhaps a different dosing paradigm or modified-testing paradigm.

Adrian Rawcliffe^ So I think I'd just say on that, that we -- if we were going to give more details on the design of that now, we're going to give more details on the design of

that on the 29th, and I'll defer that question until then, if that's okay? Anything else I can help you with?

Nicholas Abbott^ How can I get to ask it again?

Adrian Rawcliffe^ You absolutely [will].

Nicholas Abbott^ And then I noticed that you have (inaudible) in the first HLA-independent target. Can you give us an example of what an HLA-independent target is? And are these too small to be targeted by a CAR? How do you decide on a CAR versus your HLA-independent TCR strategy?

Adrian Rawcliffe^ That's a very good question. I will hand -- I will ask Helen Tayton-Martin to answer that from our side.

Helen Tayton-Martin^ Yes. Thanks, Ad, and thanks, Jim. So I think the simplest way to answer that is the HLA-independent targets that we -- the TCR targets that we've been focused on are effectively CAR targets.

So these are cell surface proteins. We obviously looked at the candidates that are well-validated as well as others that are less well-validated but emerging. And I think really, an HLA-independent TCR is a TCR that can recognize specifically a cell surface protein. And really, it then signals through the TCR, it behaves like a TCR on a T-cell, but.

What we've been able to do is find these very, very rare TCRs and then put them through our internal proprietary processes for specificity testing and potency and efficacy. And that's really the basis of the targets that we're going after in that platform.

So it's very exciting. It leverages all of the TCR capability and functional behavior, but it is obviously not HLA-restricted. So we haven't disclosed the first target that we've agreed with Astellas, obviously, but it will be a hit target. I hope that answers the question.

Nicholas Abbott^ Certainly, partially. So do you -- are you selecting something that -- do you do a head-to-head with a CAR? Or is this just a way to target HLA-independent proteins without having to develop or use a CAR technology?

I guess the point is we always hear that CAR-T cells combine targets with lower antigen density than a non-SPEAR-matured TCR, but CAR-Ts are presented to be more potent, if you like, than TCR-based therapies.

Helen Tayton-Martin^ So actually, I think that the TCR-based targets normally (inaudible) is usually very low frequency, so very low levels and therefore, having a specific TCR is critical. I think you can apply similar sort of dynamics.

So cell service protein typically many more proteins, and the CAR is seeing much more surface antigen. But the idea of a finely tuned specific TCR that can see a fragment of

that cell surface protein is really the design behind a functional TCR that can recognize the cell surface protein.

So we think that there are advantages in our preclinical testing. You wouldn't be surprised to know that we do do head-to-head comparisons, and I think that's what's given us a huge amount of enthusiasm and excitement around this program. So we've been able to generate them. We tested in comparison to published CARs on well-known targets, and we've been able to fine-tune them in terms of their potency and specificity.

Nicholas Abbott^ Great. That's very helpful. And just the last one for me on ASP. Obviously, you've started this program with a healthy degree of trepidation. Now that you're at very high doses of cells.

Is there a concern that there is enough variability in healthy tissue, potentially that there are rare events that -- safety events that you're not really going to be able to discount until you treated tens of hundreds of patients. How confident are you now that you got past as concerned about on target of tissue toxicity?

Adrian Rawcliffe^ I'll ask Elliott to take a stab at that.

Elliot Norry^ Yes. Thanks for the question. I mean I think that I would say that we have increasing confidence with each patient that we treat, and see that there's an absence of liver toxicity associated with the TCR.

So I think your point's well taken, could there be a rare patient that has a level of expression that's high enough to generate such a response and will we know that without treating many, many more patients? I think that in the world of clinical safety until you've treated that number, it's hard to make assumptions about that.

But to date, we haven't seen liver toxicity that would be of that type of concern to us. So we're gaining increasing confidence in the product and actually just the number of patients that we've treated to date and seeing safety at the highest cell dose does give us increasing confidence.

Nicholas Abbott^ I'm trying to picture what your virtual ASCO booth will look like. So you'll have to send me a picture.

Adrian Rawcliffe^ Thanks, Nick.

Operator^ (Operator Instructions) Our next question comes from Jonathan Chang with SVB Leerink.

David Ruch^ This is David Ruch on for Jonathan. Congratulations on the progress. Just to go back to that last question a little bit. You mentioned the AFP program had progressed to the $10 billion cell expansion cohort.

And you've talked about the safety profile already. But I was wondering how you think about dose escalation from here? And when do you think we might see updates from these later cohorts?

Adrian Rawcliffe^ So I'll just put out on the -- in terms of the timing for data updates, and then I'll ask Elliot to comment on the dose-ranging that we're seeing and what we -- what our thoughts are about that.

So we previously guided that we would provide an update on cohorts on the dose escalation. At the international liver conference that was subsequently moved from April to August. And as Elliot talked about earlier, that will be the case.

We, also, at the same time that we provided that guidance we provided guidance that we would update on the expansion cohorts, patients that we recruited in the second half of 2020, and we think that remains a reasonable goal for us at this point in time. And so that's when you should anticipate.

I will point out, I just want to, in the abundance of caution. We -- small numbers of patients are very meaningful for us. This trial is now recruiting at the expansion level. The advantage of that is lack of stagger between patients.

However, this is still an early Phase I trial, and we're still evaluating safety at this level, et cetera. And so I would not anticipate hundreds of patients in the update in the second half of this year. But I think we hope to give a meaningful update on the -- on patients enrolled into the expansion cohort there. Elliot, thoughts on dose escalation?

Elliot Norry^ I would just say that the study is designed at this point too, as Adrian mentioned, to treat patients with doses up to 10 billion cells in the expansion phase without a stagger.

And we don't -- there's no plan to dose escalate beyond that. We're really looking to accumulate a number of patients experience with that cell dose and to make further decisions from there.

David Ruch^ Got it. That's helpful. And you mentioned the durability data from the sarcoma patients from the ADP-A2M4 study would be presented at ASCO. How are you thinking about competitive benchmarks in the sarcoma space? And could you help set expectations here?

Adrian Rawcliffe^ Elliot, would you like to talk to that?

Elliot Norry^ Sure. So from the standpoint of competitive benchmarks in the second-line setting after systemic chemotherapy for the treatment of sarcomas, the second line therapies that are currently approved and/or used regularly are typically associated with response rates in the 5% to 15% range and are associated with a degree of toxicity depending on the treatment.

And the treatments are generally ongoing treatments where patients have to return after time for therapy. So when we're seeing 50% response rates and the durability will be discussed.

And as of CTOS, we're seeing durability out to 9 months. That represents, we believe, an advantage to those patients as compared to what's currently available.

And the advantage of a single therapy that allows patients to go through a treatment with -- that does have a degree of (inaudible) but once resolved can enjoy a period of time without further ongoing week-to-week or month-to-month treatments also is part of the advantage to the patients. So I'll leave it at that, but I think that's really how we see it.

Operator^ Our next question comes from Sarah (sic) [Mara] Goldenstein with Mizuho.

Mara Goldstein^ This is Mara, actually. Just a question on the allogeneic TCR-T program. And can you just outline for us how we should anticipate that, that would progress?

And what are the sort of milestones that we should look for into? In addition to which, on the approach that you're taking, are there ways in which as you look to develop therapeutically that you can somehow also in antigen presentation on HLA? Are there approaches that allow you to do that? And would you consider that?

Adrian Rawcliffe^ So thanks, Mara. I will ask Helen to talk to the allogeneic piece.

Helen Tayton-Martin^ Thanks for the question. So as you will have seen, our approach is focused on developing T-cells from iPSC stem cells, and that's been a focus of ours for some time, and that's where we've made the most progress with the recent presentation this week at ASGCT where we actually were able to determine the right location to put in a TCR into, those edited then differentiated stem cells to T-cells.

So there's a lot of research that's gone sort of been performed to sort of get to that stage. And it's important to generate a cell where we are basically removing Class I and the endogenous TCR and putting in a suicide gene that enables those cells to be stopped if we need to.

So the last step for us was actually getting a TCR into a functional location from editing and differentiating into a functional cell. So a huge -- that's a huge step forward to produce a T-cell in that setting.

And then we're -- the next steps are really around confirming any other final edits we may need to and then beginning the process development work in to get to cell banks that can go into the clinic. And we started all of those processes.

And so the next -- to answer the question in terms of milestones, that will be, obviously, IND filing and heading to the clinic. And we haven't -- there are a number of new moving steps on that, which we haven't bottomed out yet.

So we're not talking about specifics of the time lines, but that is on our horizon. And obviously, we have our own programs, which leverage that for our MAGE-A4. And then we have new programs through the Astellas collaboration, which will be coming along behind.

And the second question, Ad -- sorry, perhaps, if that's repeated, I'm not sure whether you want me to answer that or whether that's another member of the team?

Adrian Rawcliffe^ Yes. Mara, could you clarify your second question about driving HLA presentation?

Mara Goldstein^ Yes. So I'm just wondering that within the context of the discussion around CAR versus TCR. I'm just wondering around sort of approaches where you could increase the amount of expression or neoantigen expression on cells so that you could use that in combination with the existing TCR T program. That's all.

Adrian Rawcliffe^ Yes. So all -- what I'd say is we have looked at approaches whereby you might increase presentation of HLA and antigen on the cells. That's not the current preferred approach to driving for increased efficacy.

We've referred to a number of other approaches as sort of next-generation approaches, including the approaches that we're currently in the clinic with CD8 in terms of converting our T-cells. And the overall rationale for that, I just want to be clear, is a well-designed T-cell receptor should, in its normal state, with the right ability, be able to track down cells with very low expression

As Helen referred to earlier, I think it's a bit of a myth that TCRs are less potent than CAR-T-driven cells. I think the antigen presentation of a typical surface antigen on a cell, particularly in the context of something that's floating around in the bloodstream and therefore, immediately available for a CAR-T in the hematological setting is orders of magnitude higher than is typically seen by HLA presentation.

And yet, in the natural system, the T-cell receptor is naturally able to find normal antigen presentation for viral antigens, et cetera. And so -- and indeed, our TCRs, we've managed -- we believe that they can track down very low -- relatively low levels of HLA presentation of sales.

And so the question, therefore, from our perspective, is not necessarily how do you drive HLA and antigen presentation with the associated risks of that, that would come with, but rather how do you improve the functionality of the T-cell through a number of our next second-generation approaches in order to enable it to either be more potent, do traffic to the tumor or to overcome the tumor microenvironment.

Operator^ And our next question is a follow-up from Jim Birchenough with Wells Fargo.

Nicholas Abbott^ Just one more on the iPSC T-cell. And that is, are you just able to make CD8 T-cells at the moment? Or are you able to make CD4 T-cells as well? And given what you've done with second-generation A2M4, are you putting CD8 into those CD4 T-cells?

Adrian Rawcliffe^ This is with respect to our allogeneic program?

Nicholas Abbott^ Yes. Correct.

Adrian Rawcliffe^ Yes. Helen, would you like to comment on that?

Helen Tayton-Martin^ Sure. So the answer is yes, we can make CD4, CD8 double-positive, and we are also getting to single-positive CD8 sales, that's all through differentiation of the stem cell to a T-cell. So it's not through adding in CD8 in that sense.

But that's a specific element that we have used in our autologous second-gen program. So in the allo space, we have focused on differentiating the iPSCs to a functional CD4, CD8-positive and CD8 T-cell.

We have still some work to do, but that's basically the phenotype we've been driving for, and that's the functionality we've been starting to show, and most recently, in this week's poster.

Operator^ I am not showing any further questions at this time. I would now like to turn the call back over to Adrian Rawcliffe for any closing remarks.

Adrian Rawcliffe^ Thank you. We've made real progress in the first part of 2020, pushing forward with our product in sarcoma and generating encouraging responses across a broad range of solid tumors.

And I'm looking forward to sharing more data on May 29 at the beginning of ASCO. I'm more confident than ever that the work we do is making and will continue to make a difference for people with cancer. And with that, we'll close the call. Thanks a lot.

Operator^ Ladies and gentlemen, this concludes today's conference call. Thank you for participating. You may now disconnect.