

On June 8, 2021, representatives of Jasper Therapeutics, Inc. (“Jasper Therapeutics”) hosted a conference call and webcast and made available a presentation regarding updated 90-day efficacy, safety and pharmacokinetic data from its ongoing multicenter Phase 1 clinical trial of JSP191, its anti-CD117 monoclonal antibody. Below is a transcript of the conference call and webcast.

C O R P O R A T E P A R T I C I P A N T S

Bill Lis, Executive Chairman and Chief Executive Officer

Kevin Heller, M.D., Executive Vice President, Research & Development

Wendy Pang, M.D., Ph.D., Vice President, Research & Translational Medicine

Lori Muffly, M.D., M.S., Assistant Professor of Medicine, Blood & Bone Marrow Transplantation, Stanford Medicine

Gail Roboz, M.D., Professor of Medicine (Hematology & Medical Oncology), Director of the Clinical and Translational Leukemia Program, Weill Cornell Medicine

C O N F E R E N C E C A L L P A R T I C I P A N T S

Emma Nealon, Cantor Fitzgerald

Matthew Luchini, BMO Capital Markets

Jay Olson, Oppenheimer & Co. Inc.

Matt Phipps, William Blair

Justin Zelin, BTIG

P R E S E N T A T I O N

Operator

Greetings and welcome to the Jasper Therapeutics JSP191 ASCO Data Call.

As a reminder, this conference is being recorded.

It is now my pleasure to turn the call over to your Jasper team. Please go ahead.

Bill Lis

Good afternoon. My name is Bill Lis. I'm the Executive Chairman and CEO of Jasper Therapeutics. We're pleased to welcome you today to Jasper's Data Review of JSP191, Phase 1 Data and AML and MDS Patients.

Today we present important preliminary data for JSP191. It's our anti-CD117 antibody conditioning agent at the annual sessions of the American Society of Clinical Oncology. We have a terrific line-up of presenters to review the data today and its implications for the field in hematopoietic stem cell transplants.

On Slide 2, I'd just like to make note of our Safe Harbor statement and Forward-looking statements.

As a quick background, I'd like to make a few high-level comments about Jasper before I turn it over to the presenters.

We completed our initial financing of the Company in 2019 and since then we have successfully advanced two programs that we believe have the potential to transform the field. Our goal is quite simple, it's to become the preeminent biotech company focused on hematopoietic stem cell transplantation or what we call cellular therapy. You'll see today that we have a company that's really focused on stem cell because of its central role in these curative cellular therapies. We've defined the biology to target the stem cell to innovate in two of what we believe are the highest areas of unmet medical need.

Presented on this slide is the central role of stem cells and our two programs for hematopoietic stem cell transplant. On the left portion of the slide is our antibody known as a conditioning agent where we target the stem cells in the patient, or the host to turn off their proliferative signal or depleted them. This is to prepare our patient for transplantation.

Then on the right side is the representation of our second program that's in the research stage. Here we're targeting the curative potential of the stem cell and the donor or the graft side by turning on or providing a proliferative advantage of these cells to help cure patients with a number of life-threatening diseases.

Now I'll present our list of speakers and agenda for today.

First, Kevin Heller, M.D., the Executive Vice President, Research and Development at Jasper Therapeutics will present an overview of JSP191.

Wendy Pang, M.D., Ph.D., Vice President of Research and Translational Medicine at Jasper. Wendy will present the mechanism of action and preclinical data for JSP191.

We're pleased to have Dr. Lori Muffly, Assistant Professor of Medicine, Blood and Bone Marrow Transplantation at Stanford University, who will present the data that was presented today at ASCO.

Also pleased to have Gail Roboz, M.D., Professor of Medicine, Hematology and Medical Oncology and Director of Clinical and Translational Leukemia Program, Weill Cornell Medicine. She'll present clinical perspective and some of the impact of today's data.

With that, I'll turn it over to the presenters.

Dr. Kevin Heller

Thank you for joining us today. We're very happy to have the opportunity to talk to you about JSP191 and all the work that Jasper Therapeutics is currently engaged in.

As you could see on Slide 6, Jasper Therapeutics, we are really focused on hematopoietic stem cell transplantation or bone marrow transplantation, which we all agree has the potential to cure multiple diseases, yet it remains underutilized to a large part because of the risk/benefit profile that's associated with the limitations of the conditioning regimen, the requirement to prepare a patient's bone marrow and clear out their diseased stem cells to replace with a new set of stem cells. This involves a variety of chemotherapeutic agents and high intensity radiation that's associated with a variety of major toxicities which comes with a significant mortality risk. And these patients are required to be hospitalized in order to receive adequate supportive care.

Also, once their bone marrow is prepared and adequately conditioned, there are also some limitations with the actual transplanted cells from the donor that based on the quality or the type of the cells and how they are prepared, there is certainly a potential of clinical relapse, failed or poor engraftment and based on the other cells within the transplant itself, there are risks of graft versus host disease that may require long term immunosuppression. Because of this risk/benefit profile, although hematopoietic stem cell transplantation has great potential for patients, only a minority of patients actually will receive a transplant.

At Jasper, what we're doing, is we're focusing on one, trying to improve the safety and tolerability of conditioning regimens without losing efficacy and also we're going to talk a little bit about the work we're doing with regard to how we're manipulating graft cells such that we can really maximize this very important therapy.

On Slide 7, I'd like to focus a bit on what are the current treatment related toxicities that are associated with a variety of different conditioning regimens. As you can see in this table, myeloablative conditioning and reduced intensity conditioning regimens are currently the most commonly used conditioning regimens for patients. The myeloablative conditioning regimens, which typically involve fludarabine, busulfan, cyclophosphamide and/or high intensity of TBI, require patients to be inpatient and to have a lot of supportive care. But you could see that there are very high rates of veno-occlusive disease, mucositis and there is a real mortality risk for these patients who receive myeloablative and reduced intensity conditioning.

Now Flu/TBI alone, which is in the column on the far right, because it's a much gentler conditioning regimen, it is not typically associated with veno-occlusive disease. Likewise, the rates of oral mucositis and some of the other adverse events are significantly lower. Unfortunately, the safer tolerable conditioning regimen is also associated with a much higher risk of relapse as you can see in the bottom row. Compared to the myeloablative, or very intense conditioning regimen, it's 42% by one year as opposed to 10%.

What we are doing here at Jasper, and we've described in the ASCO presentation, is that we're combining our antibody, JSP191, with Flu/TBI in hopes to enhance that conditioning regimen while sparing the patient a lot of the adverse events.

On Slide 8 you could see that 191, our antibody, is an anti-CD117 antibody that specifically targets stem cell factor receptor, also known as CD117. By doing this, it blocks stem cell factor from binding to the cells, the patient's stem cells, and by doing so, we're taking away that signal, that survival signal that allows a patient stem cells to persist. What that ultimately leads to is a depletion of the patient's stem cells, as you could see in the middle part of this diagram, that we have an empty bone marrow niche. So, we've been able to effectively prepare a patient to receive a healthy donor stem cell engraftment.

Now, I would like to also point out on Slide 9, that the engraftment itself is not always perfect. For example, a replete or modified grafts, the hematopoietic stem cells that we are getting from donors will often come with other cells that are required in order for the donor cells to actually stick and stay in the patient's marrow. But also they can lead to the potential of graft versus host disease and other issues.

Jasper is working on an engineered hematopoietic stem cell platform where we're providing a transient advantage to the stem cells such that, for example, these stem cells could be given as a pure stem cell graft without the other cells. This could allow for potentially a higher level of engraftment, whether it's an allo or in combination with an autologous gene-therapy transplant. This would also mean the possibility of significantly decreased risk of graft versus host disease.

If we look at all of this work that we're doing, as you can see on Slide 10, here at Jasper we have a lot of trials that are opening up to evaluate JSP191 as a conditioning regimen, AML/MDS which is what we're going to focus on today. We're also very excited about some of the data as a single agent conditioning regimen in SCID patients and Jasper will be opening up a trial to evaluate JSP191 for conditioning for patients with severe autoimmune diseases. Given the enthusiasm from academic centers at Stanford and groups at the NIH will also be looking at 191 as part of conditioning regimen for other diseases.

We're just now really beginning to take off the engineered hematopoietic stem cell platform where we're going to start really evaluating this interesting program in a variety of monogenetic diseases.

Now, I think I'd like to hand off the microphone to my colleague, Wendy Pang, who is going to talk a little bit more thoroughly about the mechanism of action of JSP191.

Dr. Wendy Pang

Thank you, Kevin. It's my pleasure to share our preclinical data for JSP191 and discuss the mechanism of action.

On Slide 12, we are showing how JSP191 antibody works. Hematopoietic stem cells rely on stem cell factor binding to CD117 to provide a critical survival signal to that hematopoietic stem cell. JSP191 prevents stem cell factor from binding to CD117 thereby inhibiting the stem cell survival signal and this leads to the depletion of the hematopoietic stem cells from the bone marrow.

Additionally, inhibition of this survival signal by JSP191 makes the stem cells more susceptible to agents such as radiation and the hypomethylating agent 5-azacytidine. This synergy leads to enhanced depletion of the stem cells from the bone marrow. Additionally, JSP191 is aglycosylated and designed to remove all effector cell function and there's no toxic payload conjugated to the antibody that might lead to undesired toxicity in other cells that express CD117, such as mast cells, germ cells and melanocytes. Therefore, we don't really see mast cell related anaphylaxis in our studies and there have been no treatment-related serious adverse events in any of the patients we've dosed so far.

On Slide 13, we're showing preclinical proof of concept data as well as early data in human patients that JSP191, as a single agent, can deplete the hematopoietic stem cells. On the left, you can see that we published on JSP191 depleting stem cells in non-human primates. On the right, you can see our data in severe combined immunodeficiency patients, that single agent JSP191 can similarly deplete the human hematopoietic stem cells.

On Slide 14, this is data showing that in a xenograft mouse model of myelodysplastic syndrome, that the antibody JSP191 cannot only deplete normal human hematopoietic stem cells but also the myelodysplastic syndrome disease initiating stem cells. So, myelodysplastic syndrome or MDS stem cells, they make abnormal blood progeny and that's what leads to the manifestations of the disease. But these MDS stem cells are still sensitive to inhibition of stem cell factor binding to CD117. When we treat the MDS xenografted mice with anti-CD117 antibody, we see on the left that the MDS stem cells are depleted and it also allows for engraftment of normal human hematopoietic stem cells and these normal human hematopoietic stem cells restore normal blood production.

On Slide 15, what we're showing here, is in a mouse model of allogeneic hematopoietic stem cell transplantation that anti-CD117 antibody can synergize with low dose radiation of 200 cGy or 2Gy to enhance depletion of endogenous stem cells. This allows engraftment of purified allogeneic donor hematopoietic stem cells to a level that is normally only seen when using significantly higher, more toxic doses of radiation like 700 cGy or 7 Gy.

On Slide 16, we're showing some early data in our clinical trial in severe combined immune deficiency. Based on the preclinical data that I just showed as well as safety data from a Phase 1 study in 77 healthy volunteers, a trial was launched evaluating JSP191 as the sole conditioning agent for allogeneic transplant for patients with severe combined immune deficiency or SCID. This is an immune disorder for which transplant is the only proven cure, and without transplant, most children die before two years of age from this disease.

We have evaluated JSP191 in two patient populations. So far, we've dosed 12 SCID patients who had previously been transplanted but the previous transplant was not successful so they remained immunocompromised. Additionally, we've dosed two SCID infants who were newly diagnosed by newborn screening. In all of these patients, JSP191 has been well tolerated and appears to be safe. There have been no treatment related serious adverse events and no myelosuppression. Indeed, JSP191 can be given as an outpatient.

In these patients, we've also now seen improvement in immune function in many of our treated patients. On the right, we demonstrate production of naive CD4 T cells in these patients who were treated with JSP191 and then given a transplant. This is an outcome that would not have been expected if these patients were just transplanted without JSP191 or any other conditioning. We're very excited by these results.

Now I have the pleasure of introducing Dr. Lori Muffly, from Stanford University, who will share the results from our clinical trial in MDS and AML.

Dr. Lori Muffly, M.D.

Thank you. It's my pleasure to present our ASCO abstract entitled Early Results of Phase 1 Study of JSP191, an Anti-CD117 Monoclonal Antibody, with Non-Myeloablative Conditioning in Older Adults with MRD-positive, MDS/AML Undergoing Allogeneic Hematopoietic Cell Transplantation.

As an overview, as you can see on Slide 18, these data are a part of an ongoing study of JSP191 in MRD-positive patients who are ineligible for full myeloablative conditioning. In this abstract we are reporting the efficacy, safety and PK data in the first six patients from the dose finding portion of the study. The dose escalation phase is now enrolling at the recommended Phase 2 dose with data anticipated first half of 2022. For this part of the study, JSP191 was dosed at 0.6 mg/kg in combination with low dose total body irradiation and fludarabine.

We assessed neutrophil engraftment, CD15 myeloid chimerism and measurable residual disease or MRD status of the participants.

The key inclusion criteria, as shown in Slide 19, included patients between the ages of 65 and 74 years with MDS or AML undergoing hematopoietic cell transplantation. Patients were ineligible for full myeloablative conditioning. The primary endpoint was safety and tolerability of JSP191 as a conditioning regimen up to one year following donor cell transplant. Secondary endpoints included engraftment and donor chimerism, clearance of MRD, non-relapse mortality, event-free survival and overall survival. The demographics of the six patients enrolled are shown in the same slide to the right. Patients were between the ages of 65 to 74 years. Three had MDS and three had AML. Prior treatments are shown. Four patients received a mast generated donor transplant and two received a match related donor transplant.

JSP191 was infused between 10 and 14 days prior to transplant. This was followed by standard dose fludarabine given over three days and 2Gy of total body irradiation.

Pharmacokinetic samples were obtained in all patients in order to determine JSP clearance, which is shown on Slide 20. As you can see in the figures to the left, clearance was found to be consistent across all subjects.

JSP191 when added to low dose total body irradiation and fludarabine appears to be safe and tolerable. In the first six patients, we did not note any infusion reactions. There were no JSP191 related toxicities. The protocol allowed for outpatient conditioning therapy; one subject had grade 1 acute skin graft versus host disease, which resolved. There was no evidence of grade 2 to 4 acute graft versus host disease and one subject was diagnosed with chronic graft versus host disease to date.

As you can see on Slide 21, all patients became neutropenic followed by successful neutrophil engraftment which occurred between 19 and 26 days following transplant. Chimerism subsets are shown to the right. We focused on myeloid CD 15 chimerism; full myeloid chimerism occurred in five of six patients by 90 days post transplant.

In Slide 22, we show the efficacy outcomes. All patients coming to this protocol had detectable MRD prior to receiving JSP191. The first column shows the actual MRD that was detected in each subject prior to antibody infusion. As you can see, by transplant Day 90, five of six patients cleared all MRD. One patient had transient MRD recurrence at Day 180 which cleared on subsequent evaluation. Responses are encouraging and ongoing.

The takeaway from the trial thus far are that JSP191 at a dose of 0.6 mg/kg, when added to low dose radiation and fludarabine, was well tolerated in all six patients and led to successful transplants, as evidenced by full chimerism greater than 95% donor in five of six patients, elimination of MRD in five of six patients with a very substantial reduction in MRD in the sixth patient. The combination also appeared very safe and tolerable as evidenced by no infusion reaction, no treatment related toxicities. The protocol allowed for outpatient conditioning and outpatient transplantation. The current dose escalation phase is currently enrolling. We will be evaluating a radiation dose of 3Gy in the dose escalation.

Thank you very much.

Dr. Kevin Heller

Thank you, Lori, for that overview. Now I'd like to introduce Dr. Gail Roboz, who's Professor of Medicine and Director of the Clinical and Translational Leukemia Program at Weill Cornell Medical Center in New York, to talk about the clinical perspectives and what this might mean as a practicing physician.

Dr. Gail Roboz

Hi. Thank you for the introduction. It's my pleasure to be included in this exciting meeting and to give just a couple of minutes of background of what are we really talking about in AML, current landscape and why this can be such an important addition to our therapeutic armamentarium. I'm going to start with Slide 25 just talking for a minute about background.

AML is the most common acute leukemia in adults. It happens all through ages, from zero to 100 but it's actually most common in older adults with a median age of onset ranging from about 67 to 70 years old. It's divided into biological subgroups based on molecular and cytogenetic features usually in sort of good, bad and medium ELN or European Leukemia Net favorable, intermediate and adverse. Unfortunately, most people are in the intermediate and adverse categories with, as you can see here, very poor overall survival and very few of those patients reaching even two years let alone five after the initial diagnosis. The only known curative therapy for this disease is allogeneic stem cell transplantation.

On Slide 26, I wanted to show that starting from 2000, this slide goes to 2013 but this trend has continued that for older patients who used to be absolutely excluded from consideration at all of allogeneic stem cell transplant, we've been trying very hard to get as many older patients as possible to be eligible for allo transplant because that was really the only therapy that offered any possibility of long-term survival. Most of the chemotherapy and hypomethylating agent regimens offered rather poor remission rates with poor overall survival, so you can see the trend here in red was to try to get these patients to an allo transplant even when they were older than 70.

But if you go to Slide 27, the problem is that there's very high non-relapse mortality in older patients than 60, and as you can see, if they have scores, baseline scores basically reflect comorbidities and other aspects of the patient's clinical scenario which makes them at increasing risk for transplants and most older patients have risks related to comorbidities or other aspects of their clinical condition. And you can see really astronomical rates of non-relapse mortality which is why many older patients simply cannot be considered for allogeneic transplant.

But if you go to Slide 28, you can see that even our efforts to try to use reduced intensity conditioning, and you can see here, RIC or reduced intensity conditioning, that was our way of saying, "Okay, they're not eligible for myeloablative conditioning. Let's at least try to get them into reduced intensity conditioning." But what this slide showed, and I want to draw your attention to the bottom curve, the worst curve, which were patients who were next-generation sequencing positive who went into a reduced intensity transplant.

So the point of what I'm showing here is that most patients with AML have "leftovers" or "MRD measurable residual disease. In this study, this was measured by next-generation sequencing. If you take those patients into a traditional reduced intensity treatment, it is unlikely to be curative. So the myeloablative is too strong. The reduced intensity is not strong enough. And these patients who represent the majority of AML patients are left with non-curative options.

So what else can we do? Well, if you look at Slide 29, QUAZAR results were presented recent. This was recently published in the New England Journal and we were happy to show that for patients over 55 who got intensive chemo but who weren't eligible for whatever reason to go on to a myeloablative allo transplant, there's something we can do. We can give oral azacitidine, which is better than doing nothing and did prolong overall survival versus placebo. But this study is for patients who couldn't go on to an allo transplant. So, again, there's this illusive goal that you want to get to an allo transplant for cure but there are patients who simply can't get there due to potential toxicities at the procedure.

If you go to 30, so this is where the current landscape for older patients has absolutely seismically shifted recently to a new standard of care that older patients actually are increasingly getting the combination of venetoclax combined with azacitidine versus alone, which has resulted in significant improvements both in overall survival and in remission rates. In this study, patients who were older than 75 were included. Those patients do sometimes go for allo transplant but not usually. The younger patients between 18 and 75 had significant comorbidities in order to get on to this particular study, so those patients were deemed "not candidates for allo transplant."

But if you go here to Slide 31, you can see even for these patients who are allegedly not candidates for allo transplant, we still try to do an allo transplant for them, and sometimes patients are deemed ineligible at the outset of their therapy but improve during the course of their treatment and they actually become eligible for transplant. These are very early data presented by Dan Pallier (phon) at the last ASH meeting suggesting that rather than going on for ongoing cycles of azacitidine and venetoclax for older patients for some of them who are eligible to continue on to transplant, again, very preliminary data but if you can get them to transplant it did look like there was a favorable overall survival.

So, the landscape here is that AML remains a very deadly and bad disease. It is extremely difficult to even consider long-term survival with allo transplants and without an allo transplant and therefore we need better, safer and more attainable allo transplants

Now I will turn it back over to the team to moderate questions and answers.

Operator

Thank you. Our first question comes from Emma Nealon with Cantor Fitzgerald. Please proceed with your question.

Emma Nealon

Hi. Thanks for taking the question. I guess, looking at the engraftment of these initial five patients, where are you hoping to see some improvement on these markers at the higher 3Gy dose when you're escalating?

Dr. Kevin Heller

Hi. Thank you for the question. So, I just would like to clarify, so you're asking really about what the value of going with 3Gy is. Because if we go back to Slide 22, I think we do demonstrate some good clearance of detectable residual disease. But I'm sorry, I'll have to ask for a clarification of your question.

Emma Nealon

Yes, just what you're hoping to see when escalating or where you think you could push this further, since it does look so good already.

Dr. Kevin Heller

Well, why don't I have Lori Muffly respond to this because she's been following the patients and certainly played a role in providing some feedback on why we were able to go up a little bit. I think, perhaps, on the chimerism, Slide 21, might help provide as a good background.

Dr. Lori Muffly

Sure. Thanks, Kevin. Thanks, Emma, for the question, it's a really good one. As you can see on the Slide 21—well, really, putting together Slides 21 and 22, what we saw was that MRD clearance and actual disease control has been really excellent in the first six patients treated. But one of our concerns on Slide 21 is when you look at panel C, which is the T-cell chimerism, and that suggests that the lymphodepletion that we're achieving with the fludarabine three-day dosing and the 2Gy of total body irradiation is perhaps not quite as robust in terms of lymphodepletion as we would like, and part of that is probably because several of the patients in this cohort, including the patient in the purple triangle, had no prior treatment for MDS, so came into transplant with a fully intact immune system. So, it's always a bit harder to lymphodeplete those patients. Secondly, several of the patients due to the COVID pandemic received cryopreserved hematopoietic stem cells, which has really not been a standard and we know we would use cells that way.

So, anyway, long story short, the goal of increasing from 2 to 3 TBI is really to try to enhance the lymphodepletion. We don't perceive based on many studies and our clinical experience that we're going to have more toxicity with 3Gy over 2Gy but we are hoping that we better the T-cell chimerism.

Emma Nealon

Perfect. That's helpful, thank you.

Dr. Lori Muffly

Sure.

Operator

Our next question comes from Matthew Luchini with BMO Capital Markets. Please proceed with your question.

Matthew Luchini

Hi, good afternoon. Thanks for taking the questions. A couple for me. Maybe first, going back to the data that was presented on Slide 16, don't want to overreach here at all, but I'm just curious if you can provide any additional color on—it looks like maybe there's one patient who didn't quite see a benefit, the green with the circles. And then maybe in contrast, it looks like perhaps a patient with the blue circles had a particularly strong response. I'm just wondering if there's anything you can say to provide additional color about what drove the response or perhaps lack thereof. And relatedly, if we go forward, I think it was Slide 20, I just wanted a little bit of additional color on subject 3 in the chronic graft versus host it was that was observed. And then I have one question for the physician as well, please.

Dr. Kevin Heller

Hi there. Thank you for asking the question. I'll take the first one regarding the SCID responses on Slide 16. It is important to point out that this was a dose escalation trial, so there are a variety of doses. However, we generally did see some excellent activity in stem cell depletion at all dose levels. The patient who is the blue circle that seem to have the most impressive naive CD4 T-cell counts, I think we might want to reemphasize here is that these are the naive CD4 T-cells, and what that means is these are cells that tend to really appear a little bit later after engraftment, but are specifically because they're naive are derived from the donor stem cell. So we're not seeing any residual dysfunctional T-cells that were in the SCID patient originally.

Now why some patients may have done a little better than the others, this is a mixed group of different underlying genetic mutations that led to SCID in these patients. So, for example, a couple of the patients who did not do nearly as well were individuals with X-SCID or IL-2 receptor gamma defect. So, as we move forward in the development of the SCID program, there might be additional questions as to whether or not a combination might be suitable, or other ways to better define patient populations. But I think the real take-home message on Slide 16 I would like to emphasize is number one is the safety and tolerability among patients from three to six months old, all the way into children, toddlers, up to young adults, and the single agent activity. And that's really what's giving us that momentum and enthusiasm to go into the AML and MDS program.

With regard to your question for Slide 20, with regard to the safety profile, I think it would be best if I refer back to Lori Muffly, who's the physician who's been at the patient's bedside, to answer that question.

Dr. Lori Muffly

Sure. I think the question asked was about chronic graft versus host disease in patient 003. What I know—this is not a patient that I'm treating. This patient is treated at another center, but the patient developed mild chronic graft versus host disease of the eyes. So relatively superficial chronic graft versus host disease and the patient has been doing well.

Matthew Luchini

Okay, that's helpful. Perhaps, for you as well, Dr. Muffly. I'd just be curious, obviously, this is early data still, but I'd just be curious your perspective on how you view this approach relative to some of the others that are in development in the space, whether it's the CD117 and CD47 or the Magenta ADC, for example. Just how you're thinking about this approach and limited data as there is sort of a little bit of compare and contrast would be really helpful.

Dr. Lori Muffly

Yes. Well, thanks for the question. I think it's a bit—I mean, I think we can conceptually compare our approaches, but I would say that this agent and this program is the furthest along of those that you mentioned. So, it's nice to see the actual data versus kind of the preclinical or theoretical. I think one of the things that is nice about this sort of naked antibody approach defined system, cell factor binding site is that it's targeting kind of the appropriate place but because it's an antibody we know the pharmacokinetics and the really important critical piece here is clearance of antibody prior to donor cell infusion.

One of the things that I think remains to be seen with the ADC approach and I think will be very interesting is how they sort of work in the clearance. So I don't know kind of exactly how it's going to work but it's great to see the sort of consistent pharmacokinetics and clearance with this antibody and the fact that we can dose about 10 days before transplant.

As a broader kind of stepping back, I think that all of these sort of innovations to transplant are really just incredible positive aspects to the field. We've been long overdue for innovation, particularly for myeloid malignancies, which represent the most common indications for transplant.

I'm certainly very excited. I think most of us in the field are very excited about all these opportunities with really this JSP product leading the way, I think, in terms of bringing it to the clinic.

And Kevin, I don't know if there's anything you wanted to add to that?

Dr. Kevin Heller

Yes, no, Lori, that was a great perspective. Yes, that's a great perspective and I think I would just emphasize that, again, that JSP191 because it blocks, as you see on Slide 12, that Wendy had already commented on, because it blocks stem cell factor's interaction, we're taking away that survival mechanism, we're taking away a biological pathway that allows stem cells to persist and that's how we're killing them. So it really eliminates a lot of risk of off-target toxicity because there are many cells in our body that are actually CD117 positive, but we're focusing on the stem cell because it is uniquely and exclusively sensitive to the presence or loss of activity through stem cell factor.

So I think that actually gives us a significant advantage over an ADC. And a point of fact, we have had other folks ask would we want to make an ADC version of JSP191, and on the surface I just don't see the advantage of that because we really do get to that underlying biological process that's required for stem cell persistence and we're taking it away.

Matthew Luchini

Great. Thank you for taking all the questions. Appreciate the color.

Operator

Our next question comes from Jay Olson with Oppenheimer. Please proceed with your question.

Jay Olson

Hey everybody. Congrats on these results, and thank you for the update and taking the question. Curious about subject #11 who had reduced MRD at Day 90. It seems like that subject also had higher absolute neutrophil count before the stem cell transplant. I was wondering if that was correlated. And then I had a follow-up question, if I could.

Dr. Kevin Heller

Thank you. Lori, would you like to respond to that, and I think Wendy might also be able to provide some insight on our antibody's activity, especially in this patient.

Dr. Lori Muffly

Sure. I think that was a very astute observation, actually, because I had to go back to the slides to pick that up. I think whether or not the sort of starting neutrophil count, I don't think we have enough data.

Wendy, I'd be curious to hear what you say but sort of to associate. I think what that just tells me is that the patient had good count recovery from their therapies and as you can see they were a little more heavily pre-treated than the other folks treated on the study.

I think we're very interested to see what the Day 180 is going to show in this patient because as you can see the SRSF2 VAF declined substantially but has not gone to zero.

So, I don't know about the correlation between neutrophil count. I do know the patient had a bit more treatment previously but I think the upcoming analysis at the Day 180 is going to be very interesting.

Wendy, what do you think about the neutrophil association?

Dr. Wendy Pang

Yes. Patient 11 was an AML patient and actually received several rounds of chemotherapy in order to achieve remission. So, this patient has that particular kind of history. I also agree with Lori that we don't necessarily I think have enough data, we only have the single patient example here to really draw any major conclusions but I would hypothesize that depending on the burden of disease entering transplant and the nature of that disease we may see different outcomes in terms of how fast they could potentially clear their measurable residual disease.

Jay Olson

Great. Thank you for that. If I could ask a follow-up to either Dr. Muffly or Dr. Roboz. If they could comment on their experience with the toxicity of busulfan or diosolsan (phon) and how its safer conditioning agent, like JSP191, might expand the use of transplants. And then related to that, if you could talk about the stem cell transplant success rate in MDS and AML patients who are eligible for full dose myeloablation. Do you think JSP191 based conditioning could provide incremental benefits to those patients?

Dr. Lori Muffly

I'm happy to take those questions, first Gail and then maybe you can also add in. Does that sound good?

Dr. Gail Roboz

Sure.

Dr. Lori Muffly

I think there were a few questions there. One about the toxicity of myeloablative conditioning and one about the incremental benefit, and then I think the third was just (inaudible) of patients with AML and MDS who undergo transplants. Is that correct? Did I get all three of them?

Jay Olson

Yes, and the potential to expand use of transplants (multiple speakers) patient population.

Dr. Lori Muffly

Yes. I think for the first one in terms of the toxicity that we see with the alkylating agent, busulfan, I think melphalan kind of falls under that, patients typically have to undergo transplant in the hospital, so that means from the start of conditioning therapy through donor cell and graft engraftment. The hospitalization is typically between 21 and 30-plus days. The most common toxicities regimen related are mucositis, diarrhea. These toxicities also somewhat increase the risk of graft versus host disease because they create an inflammatory environment in the bowel in particular. So, these types of conditioning therapies can be very difficult for older patients, for frailer patients and as Gail mentioned in her talk the transplant related mortality historically of using more intensive prep, particularly older patients, can approach 30% plus. So, not insubstantial toxicities associated with more intensive conditioning.

In terms of the outcomes, so I think the comparable populations here are older adults with AML/MDS who are transplanted with non-myeloablative conditioning regimens. It's a bit difficult to compare across studies mostly because these patients, these sick patients all have MRD coming into transplant and up until recently that wasn't really kind of well defined in our studies. But typically, we think of survival and relapse-free survival being in the 30% range for all comers, older adults undergoing non-myeloablative conditioning.

So, lots of room for improvement. For patients with MRD-positive disease, the outcomes go down from there. So we're looking at probably 20% of patients being kind of longer-term disease free in the traditional non-myeloablative setting.

in terms of the ability for JSP191 to sort of complement or add on to myeloablation, I think the thought here is that the JSP191 is providing some degree of myeloablation to patients who wouldn't otherwise receive it. I think the question of whether JSP191 could be added to more intensive conditioning regimens for younger patients to sort of further improve outcomes is an open question. But I think here the whole point of this trial is to provide myeloablation through an antibody without using sort of toxic conditioning agents.

I don't know, Gail, if you want to add on.

Dr. Gail Roboz

Yes, no, I think just a simple statement of it's very tough to imagine anybody who would have trouble with the concept of less brutal conditioning. So one of the main bugaboos that we have to deal with is just miserable toxicities of current conditioning, they are variable, they hit patients differently, but I think the blanket statement of 'I can zap your marrow and myeloablate it with less toxicity.' A less toxic regimen is yes, please. I would say that while the landscape changes in AML have certainly been absolutely positive and directionally positive, we are not one and done with either chemotherapy or transplant in either MDS or AML, and the availability of targeted therapies, the availability of the currently approved and pending agents to optimize the very poor outcomes that we have been dealing with are undeniable and thrilling but they're not curative regimens.

I think that in the same way as when I'm asked on all of these calls that, oh, but isn't chemo dead? Isn't transplant dead? Absolutely not. Those are still what cure patients with AML and again it's very difficult to not get greedy in thinking about it that if something is making myeloablation less toxic, well why would that be restricted to only frail or older patients? In my mind, it would be an easy stretch to start investigating that to see whether the broader group of patients who might be able to go through a miserable toxic conditioning might also have a less dire experience because there is upfront mortality with myeloablative transplant even in 20 and 25 year olds. So I would argue that if something looks good, it's very easy to imagine it being more broadly applied.

Jay Olson

Excellent. Super helpful. Thank you so much for taking the questions and congrats again on these results.

Operator

Our next question comes from Matt Phipps with William Blair. Please proceed with your question.

Matt Phipps

Thanks for taking my questions. Congrats on some nice data here from these initial patients. For Dr. Muffly and Dr. Roboz, just wondering if you can comment on, I guess, the logistics of an antibody a week before—well, more than a week before kind of the rest of the regimen if that has any issues on kind of patient flows and managing patients.

Dr. Lori Muffly

Yes. That's a really great question. This is Lori Muffly. One of the nice things about the experience thus far has been that we have not seen any toxicities with the antibody. So, of course, this is a Phase 1 and we're following the patients very closely through the week after the antibody—obtaining the pharmacokinetic sample. But, really, it has not been disruptive in any way to the patients, so I could see how once we move up beyond Phase 1 or this becomes ideally in the future a standard of care, where really you give the antibody you don't need to kind of check in with the patient everyday, the patients are doing great and then you start the low dose conditioning outpatient.

So it really has not been much of a big deal I think really because of the toxicity profile being so minimal to none.

Dr. Gail Roboz

I would just add that I don't care if it's a big deal. I think that, and I've been saying this for 25 years, I will administer the therapy intraocularly everyday including Sunday if it works. So, AML's bad. People are dying of this. I don't really care what day it is, whether the IV is blue, purple, upside down or needs three IV pulls, if you have something that is getting the job done in a non-toxic manner, there will be 100% uptake, period, full stop. Similarly, I think it's seductive to look at AML therapy

I would strongly argue that regardless of the administration's schedule or convenience, this is 100% efficacy and tox driven. Everything else is gravy.

Matt Phipps

Good. Thanks. Really good endorsement. Thanks Dr. Roboz. A question for the Company. Dr. Roboz may have answered this. For the Company, when you think about development in a broader AML/MDS Patient population—is this 191 plus Flu and TBI what you want to stick with, or will you look at other things when into those younger patients? I don't know if they need more if they haven't had the chemo like that one patient and such.

Kevin Heller

That's a great question, and that's looking beyond the data that we here. First of all, safety—I was impressed by what Dr. Roboz just said, but it really is nice to have a safer option. I'm a pediatric oncologist by training, so thinking about long term side effects, the fertility of your patient as time passes. Many of these tinotoxic regimens that include the busulfan, oflan, for example, have some serious long-term side effects. If we can significantly improve on the safety profile, which I believe we've begun to demonstrate that, but without taking away that efficacy, for example, the higher rates of relapse that's seen with Flu-TBI alone or other reduced intensity conditioning regimens. If we could continue to see effective conditioning but we're doing it with JSP191 and Flu-TBI, it really would make sense that we would continue expanding.

So, from a regulatory standpoint, how will we get this approved? There are a couple of populations with just AML/MDS that we're going to be looking at, and we believe that 191 plus Flu-TBI versus a reduced intensity conditioning regimen could be one comparator where we could have superiority. Certainly, we'll have the safety advantage but again because of that synergy with radiation and our ability to kind of go up a bit on radiation relative to other programs and still get that really great myeloablation to allow for successful engraftment, we should be able to beat any other Flu-TBI regimen alone or another reduced intensity conditioning regimen. That would be a superiority situation.

Eventually, later down the road, another thing we could look at is—I think Dr. Roboz was going in that direction. What about patients who are in their 40s or 50s and have the misfortune of developing AML? They might be eligible for myeloablative, but if we can demonstrate that 191 plus Flu-TBI is just as effective as a conditioning regimen, why would that 45 year old person want to endure a couple of weeks of inpatient supportive care, mucositis, and all of the other toxicities when they can possibly get the same quality of conditioning with an outpatient procedure with 191 as the backbone.

If we could look at Slide 10 just for a second, our pipeline, it has a very wide range of potential indications for 191, and it's because any disease that could benefit from a transplant—if 191 continues to demonstrate the safety, tolerability, and efficacy as a conditioning agent as it has thus far, we could easily see 191 becoming a backbone to effectively change the risk benefit profile of transplantation such that it will continue to go into SCID. We already have a commitment from our Company. We intend to open—by the end of this year we hope to have open a trial in autoimmune diseases.

But also, we're looking at other monogenetic diseases like Fanconi anemia, sickle cell disease, chronic granulomatous disease, as well as gene therapies because one of the biggest obstacles has been, what's a safe tolerable way to get transplant, which is an indication for the most severe autoimmune diseases, for example. Now, what is a safe way that we can get patients to that point. It's getting over the toxicity and the risk benefit profile.

So yes, we generally—it's a very wide range in direction that we can go in. We have a lot of enthusiasm here. I hope it's shared.

Matt Phipps

Thanks for that. If I could ask one last thing. Longer term, I believe when you all have the ability to start combining with the engineered stem cells, you can kind of give the drug log JSP191, it's still in the system, if I recall correctly, some of the plans. At that point, would you change the timing or dose of JSP191 if that were the case?

Kevin Heller

Well, look Matt, some of those experiments have to done and one of the things I do like and I know Dr. Muffly showed this with the PK profile, of the general consistency, the antigen antibody that's given IV and this has been fairly consistent from patient to patient is that it does allow us to provide an infusion and gauge when we expect levels to hit the adequate point, but it also means that between the dose of 191 and the ultimate transplant, there is plenty of time to insert in the appropriate conditioning agents for other conditions.

Now, with regard to how we combine with the engineered stem cells, we weren't really going to talk much about the engineered stem cells in this conversation but, yes, all of that will play in together, and I think what's exciting about what we can do by bringing these two programs together, we're really out flanking the disease by improving the conditioning regimen, but then also with the engineered stem cell platform that Dr. Pang has really spearheaded and developed. We're able to also improve the potential of the stem cells being able to stick, stay, and outpace the disease stem cells. So, we've got some really exciting times that we are looking forward to over the next couple years with this Company.

Matt Phipps

Great. Thanks again.

Operator

Our next question is from Justin Zelin with BTIG. Please proceed with your question.

Justin Zelin

Hey, team. Congrats on this data. I had a question for the physicians. Could you just remind us roughly what percentage of older AML/MDS patients are deemed ineligible for standard myeloablative pre conditioning regimens? So, those who would go on to seek non-myeloablative or reduced intensity approach?

Kevin Heller

I'm happy to defer to our physicians who are still actively treating patients. Lori, Gail, if you'd like to...

Gail Roboz

I can certainly answer that. So, I think if you look at some interesting registry data that were just presented across the last ASH and possibly last ASCO as well, the number of patients actually across the United States who are ultimately referred for transplant with AML is unbelievably small. It's a couple of percentage points of the total number of AML patients. That number goes up in academic centers, but most AML patients in the United States are treated in the community, and the vast majority of them are never even referred for stem cell transplant because there's still a wide belief that patients over 60 can't even go for stem cell transplant.

Among younger patients, it's a little bit more difficult to quantify because there are certain patients who are not sent for transplant, let's say, the TP53s or others who are tried for sometimes investigational therapies, first, since transplant doesn't have great outcomes there. But there definitely is an attrition rate of patients during casualties during induction or other factors, lacking social support, or economic support that results in even the majority of younger patients who are supposed to go onto an allo transplant don't ultimately get one, and those numbers are all ever bigger in Europe where very, very few patients older than 65 ultimately get transplanted at all. And these are primarily due to, for the older patients, primarily due to either actual or perceived toxicity concerns.

Lori, I don't know if you want to add anything to that.

Lori Muffly

No. I think you captured it. I mean, I would say in practice, the needle is moving in terms of trying to get more eligible patients to transplant. Really, the use of intensive regimen for older adults, if anything, it should be going the opposite direction. We should be doing less and less of that. There's really kind of very clear data on the toxicity, and so I think very, very few patients—particularly we see patients well over their 70s, and so very, very few of these patients are going to be offered a myeloablative conditioning regimen.

Justin Zelin

Got it. That's helpful. Thank you.

Operator

There are no further questions at this time. This concludes today's teleconference. You may disconnect your lines at this time and we thank you for your participation.

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In connection with the proposed transaction between Amplitude Healthcare Acquisition Corporation (“Amplitude”) and Jasper Therapeutics, Amplitude has filed relevant materials with the SEC, including a registration statement on Form S-4, which includes a proxy statement/prospectus. Promptly after the registration statement is declared effective by the SEC, Amplitude will mail the definitive proxy statement/prospectus and a proxy card to each stockholder as of a record date for the meeting of Amplitude stockholders to be established for voting on the proposed business combination. **Investors and security holders of Amplitude are urged to read these materials (including any amendments or supplements thereto) and any other relevant documents in connection with the transaction that Amplitude will file with the SEC when they become available because they will contain important information about Amplitude, Jasper Therapeutics and the transaction.** The preliminary proxy statement/prospectus, the definitive proxy statement/prospectus and other relevant materials in connection with the transaction (when they become available), and any other documents filed by Amplitude with the SEC, may be obtained free of charge at the SEC’s website (www.sec.gov). The documents filed by Amplitude with the SEC also may be obtained free of charge upon written request to 1177 Avenue of the Americas, Fl 40, New York, New York 10036.

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