Senti Bio (Nasdaq: SNTI)
SENTI-202 Initial Clinical Results
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Corporate Speakers:

- Dr. Timothy Lu, Senti Bio Co-Founder & Chief Executive Officer
- Dr. Stephen Strickland, Sarah Cannon Research Institute, Director - Leukemia Research
- Dr. Kanya Rajangam, Senti Bio President, Head of R&D & Chief Medical Officer
- Thomas Chung, Senti Bio Vice President, Strategic Finance & Corporate Development

Participants:

- Daina Graybosch, Leerink Partners Analyst
- Geulah Livshits, Chardan Capital Analyst

PRESENTATION

Operator: Good morning and welcome to the SENTI-202 Initial Clinical Data webcast. Your host for today's call is Dr. Tim Lu, Chief Executive Officer and Co-Founder of Senti Bio. All lines have been placed on mute to prevent any background noise. After today's presentation, there will be an opportunity to ask questions. (Operator Instructions) I will now turn the call over to Dr. Lu. Please go ahead.

Timothy Lu: Thank you very much for joining Senti Bio's presentation today. My name is Timothy Lu and I'm the CEO and Co-Founder at Senti Bio. Senti is a pioneer in engineering gene circuits with the goal of enhancing the efficacy, specificity and controllability of cell and gene therapies. And today, we're excited to share our first data readout from our clinical trial on our lead program SENTI-202.

On Slide 2, before we begin, I would like to remind everyone that except for statements of historical facts, the statements made by management and responses to questions on this conference call are forward-looking statements under the Safe Harbor Provisions of the Private Securities Litigation Reform Act of 1995. These statements involve risks and uncertainties that can cause actual results to differ materially from those in such forward-looking statements.

Please see the forward-looking statement disclaimer in the company's press release accompanying this presentation, as well as risk factors included in our most recently filed quarterly report on Form 10-Q filed with the SEC. Undue reliance should not be placed on forward-looking statements which speak only as of the date they are made, as the facts and circumstances underlying these forward-looking statements may change. Except as required by law, Senti undertakes no obligation to update these forward-looking statements.

So on Slide 3, I'm really excited to share with you an overview of Senti and then to introduce and then lead into SENTI-202 initial clinical data. On Slide 4, Senti's mission is to enable enhanced precision, control and activity of cell and gene therapies using our Gene Circuits platform. This platform was developed at leading labs in synthetic biology from universities such as MIT, Harvard, BU and Stanford. And what Senti has done over the last several years is to super charge

and industrialize this platform by integrating synthetic biology with new technologies such as automation, high-throughput engineering, machine learning, to enable design of gene circuits for any cell and gene therapy modalities.

The four key strategies encapsulated by our Gene Circuit Technology, the first one is Logic Gating. The idea of Logic Gating is to enable our therapeutics to recognize and respond to multiple targets rather than just a single one and by doing so, enhance the ability to overcome heterogeneous disease as well as to protect healthy cells from being affected. And this Logic Gating technology is embodied in our SENTI-202 program.

The second component of our Gene Circuit platform is called Multi-Arming. The goal here is to overcome heterogeneity in multiple disease mechanisms by engineering a cell and gene therapy to essentially target multiple mechanisms at the same time.

A Regulator Dial allows us to achieve control over therapeutics in order for us to optimize the therapeutic window. And this control can be done with exogenous FDA-approved small molecule drugs or through endogenous signals.

And finally, Senti has built a library of different Smart Sensors that can basically dictate where and when a particular cell and gene therapy should be active in the body.

On Slide 5, leveraging our Gene Circuit platform, we have built an exciting pipeline of internal programs as well as partner programs. Today's meeting will focus on SENTI-202, which is a product designed to attack CD33 as well as FLT3 in diseases such as AML and MDS, as well as other blood cancers. In addition to that, SENTI-301A is a liver cancer program addressing GPC3. Our SENTI-202 program, as described today, validates the Logic Gating platform and opens up potential opportunities for additional cancer indications in other solid tumors downstream.

We've also developed collaborations with leading pharmaceutical companies, including Roche/Spark around AAV gene therapy engineering using our Smart Sensor Promoter platform as well as with Bayer and BlueRock around iPSC cell engineering.

On Slide 6, I'm happy to introduce SENTI-202 which is a potentially first-in-class, selective, off-the-shelf cell therapy designed to overcome key outstanding problems in AML. Specifically SENTI-202 leverages healthy NK cells from selected adult donors and is genetically engineered with three key mechanisms. The first one is Logic Gating to overcome AML heterogeneity by targeting the clinically validated targets of CD33 and FLT3. By doing so, this product has the potential to trigger killing of both AML blast and AML leukemic stem cells, which may have the potential to drive deeper responses.

The second component is meant to address the issue that AML targets in general are not uniquely expressed just on AML cells. They're oftentimes also expressed on healthy cells and this limits the therapeutic window of conventional modalities. To overcome this, we use our Logic Gate technology to build an inhibitory CAR, which recognizes healthy hematopoietic stem cells through a target called Endomucin or EMCN, which is selectively expressed on those healthy

hematopoietic stem cells, but not on the cancer cells. And this essentially provides a do-not-eat-me signal for the product.

As Kanya will mention later, this product is also engineered with our calibrated release IL-15 technology. We're happy to report that in 2024, we dosed our first patient in the second quarter of this year and we are reporting today initial clinical data readouts. In our first dose cohort, two out of three of our relapse or refractory AML patients achieved MRD negative complete responses at that first dose level. In addition to that, the completion of our private placement financing will continue to support the SENTI-202 clinical development path and allow us to obtain additional efficacy and durability data for this product.

So with that, on Slide 7, I'd love to now have us jump into the SENTI-202 clinical information. It's my great pleasure to introduce Dr. Stephen Strickland, Director of Leukemia Research at the Sarah Cannon Research Institute. He's one of the trial investigators who has enrolled patients on this trial. Dr. Strickland?

Stephen Strickland: Thank you, Tim. Good afternoon everyone. Today, I will review the unmet need in acute myeloid leukemia or AML, along with key considerations to developing clinically meaningful and effective AML therapies. On Slide 8, AML is an aggressive leukemia with poor prognosis and is a heterogeneous group of rapidly progressive blood cancers whose hallmark is clonal proliferation of leukemic blasts arising out of the myeloid lineage. This translates to impaired normal blood cell production, leading to low white cell counts and platelets, making the patient susceptible to infection and bleeding.

At the time of initial diagnosis, the treatment has historically included intensive chemotherapy followed by hematopoietic stem cell transplant for younger or fit patients and more recently, Venetoclax and hypomethylating agent or HMA based therapy for our older or unfit patients. Most patients unfortunately will eventually relapse even with treatment of intensive chemotherapy and at the time of relapse, the prognosis is quite dire with a median survival of 5.3 months.

In terms of available therapies, once a patient has relapsed or refractory response rates of only 20% to 30% have been reported with the use of targeted agents in subsets of patients with FLT3 or IDH mutations, and FLT3 and IDH inhibitors, but also, even with recycled chemotherapy for those without these mutations. Therefore, additional therapies are urgently needed for this disease and it meets a huge unmet need.

On Slide 9, there are multiple challenges to developing effective new AML therapies. One, the durability of response is often limited because of leukemic cell clonality and heterogeneity that requires the use of multi-targeted therapies or combination therapies. Second, the presence of a small AML cell population called leukemia stem cells or LSCs, which also have stem cell features such as being undifferentiated. They're drug resistant and they have the capacity to self-renew. They're held to be responsible for the relapse initiation even with bulk AML blast clearance.

Another major challenge is treatment tolerability limitations. This is because AML targets are often present on the healthy HSCs or hematopoietic stem cells, or cells on the myeloid lineage as well. So this results in therapies which not only kill AML cells but also cause considerable bone marrow toxicity and because of overlapping toxicity profiles of the approved AML therapies including this bone marrow toxicity that limits combination as well as the ability to sequentially use these therapies in managing patients.

On Slide 10, therefore, the hallmarks of effective AML therapies include ones which can address these issues and which can achieve deep responses, translate to increased durability and prolonged survival, and two, can do so well minimizing off-tumor, on-target toxicity and support normal blood count recovery. Both of these also correlate with better prognosis. Detection of measurable residual disease or MRD positivity in patients, even ones in CR by conventional methods, correlates with shorter remission and survival.

Measurement of MRD is not standardized across care centers but is used quite frequently. And common methods used include multi-parametric flow cytometry, next generation sequencing, or PCR in those patients with specific mutations. Detection of any MRD positivity by these techniques including the most sensitive methods, has been shown to correlate with a poor prognosis.

Secondly, achievement of CR with full blood count recovery also correlates with better prognosis compared to a CR where AML blasts are reduced, but the count is not fully recovered. By definition, CR includes bone marrow blasts of less than 5% along with recovery of both neutrophils and platelets.

So in summary, effective AML therapies are ones that can achieve deep and MRD negative CRs, translating hopefully to durable omissions and longer survival, as well as ones which can selectively kill AML blasts, translating hopefully to robust blood count recovery and a better prognosis for patients.

I'll now pass the mic to Kanya to review the clinical data. Thank you.

Kanya Rajangam: Thank you, Dr. Strickland. My name is Kanya Rajangam. I'm the Head of R&D and Chief Medical Officer at Senti Biosciences, and it's my great pleasure to present initial clinical data from our ongoing SENTI-202 trial.

On the next slide, Slide 12, as Tim mentioned, SENTI-202 is our selective, off-the-shelf CAR-NK cell therapy that we are developing for AML and other blood cancers. The cellular backbone of SENTI-202 is healthy NK cells from selected adult donors. We screen donors and we bring back donors with specific characteristics such as activation and expansion potential for GMP manufacturing. We've added the Logic Gated Gene Circuit and Multi-Arming into the SENTI-202 product.

The three major elements here have been selected to increase the natural anti-AML activity of NK cells. First is our activated Chimeric Antigen Receptor or CAR that consists of a bicistronic binder that recognizes either CD33 and/or FLT3, and upon such recognition triggers a kill

mechanism via the co- stimulatory and the intracellular signaling domain. CD33 is a validated AML target. SENTI-202 also kills cells that express FLT3, which is nearly universally expressed on leukemia stem cells.

The second chimeric protein is our inhibitory CAR. This is unique to SENTI-202. We selected a protein, endomucin that is found on the surface of healthy hematopoietic stem cells shown here in green, but not on the surface of leukemia stem cells or leukemic mass. Then, we attached an intracellular inhibitory domain to this endomucin. So upon the binder recognizing endomucin on the healthy hematopoietic stem cells, the inhibitory CAR intracellularly overwrites the kill mechanism of the activating CAR on SENTI-202 even if the healthy cell, as shown in this cartoon, expresses FLT3 or CD33.

This allowed us to engineer in the selectivity that Dr. Strickland highlighted as being one of the key limitations of current AML therapies. Over months and years of pre-clinical work, we perfected this interplay, ensuring that SENTI-202 selectively kills leukemia cells and LSCs and at the same time selectively protects the healthy hematopoietic stem cells without the lower effects in either direction.

Finally, we have Interleukin-15, a key cytokine necessary for NK cell activation, persistence and expansion, engineered in as a calibrated release protein such that not only the IL- 15 activating SENTI-202 in an autocrine fashion, but it is also released by cell-surface proteases to enable it to influence post-immune cells and activate their anti-cancer response in a (inaudible) fashion.

Moving on to the next slide, Slide 13. Slide 13 is an overview of our Phase I trial design. The first thing I want to highlight is we start with a high starting dose. We were able to leverage the well-known excellent safety profile of NK cells from other clinical trials to start with a high dose supporting early efficacy signal detection. Our patient population on this trial consists of adult patients who have CD33 or FLT3 expressing heme malignancies with a focus on AML. AML patients must have received one to three prior treatments including the targeted agent if they have a FLT3 or an IDH1/2 mutation.

The study follows a standard three-plus-three study design with dose escalation followed by disease-specific expansion. We start at a high dose of 1 billion CAR-NK cells per dose, with a second dose level of 1.5 billion cells per dose. The protocol is written to allow us to transition from Phase I to pivotal study quickly. We also have the ability to look at additional dose-intensive schedules if we so choose to. In terms of trial endpoints, we evaluate safety, efficacy including assessment of measurable residual disease or MRD using methods available at each site and correlatives including PK.

Our dosing regimen, which is illustrated in the schema at the bottom of the slide, we start first with lymphodepletion, which is disease specific and includes fludarabine and Ara-C or cytarabine. Then we follow with three doses of SENTI-202 on Day Zero, Seven and 14 for a total of 3 billion cells per cycle at the first dose level. A bone marrow assessment is done on Day 28 to assess the disease status. As long as patients have some blast reduction and the therapies are tolerated, they are eligible to get additional cycles consisting of lymphodepletion followed by three doses of SENTI-202.

On the next slide, Slide 14 summarizes our clinical trial results as of the date of this data cut. The first thing I want to highlight is we observed early efficacy signals at the first dose level. In terms of enrollment, we finished enrolling our first dose of 1 billion CAR-NK cells per dose and that dose level has been cleared. Dose level two of 1.5 billion CAR-NK cells per dose is actively enrolling.

In terms of safety, SENTI-202 is well tolerated with a profile consistent with patients with underlying AML receiving LD chemotherapy. In terms of efficacy, we are really excited to report we noticed two deep CRs out of the three relapse/refractory AML patients enrolled at the first dose level. Finally, SENTI-202 PK showed the transgene was consistently detected in the periphery in all three of the 1 billion CAR-NK cells per dose patients with a profile consistent with that of allo NK therapies.

On the next slide, Slide 15 summarizes the time of study of all three patients. The first patient achieved a PR at the end of first cycle, which deepened into a CR with no AML mutational clones detected by next generation sequencing or NGS. The second patient achieved a CR for the first cycle of therapy, including no detectable leukemia clones by the most sensitive methods the site uses, which is MRD flow or multi-parametric flow. The third patient was refractory to therapy. Both CR patients are continuing in remission.

Slide 16 summarizes the data for Patient One. To orient you to the slide, the graph on the upper left plots the bone marrow blast percentage the patient has a different time points on study for the latest common methods for assessing them. The graph on the upper right plus the patient's peripheral blood counts which are an important component of AML response. And in the text box in the bottom is the case summary.

To start with the patient's summary, Patient One is a 26-year-old female patient with adverse risk relapsed AML with relapse of the intensive chemotherapy and prior HCT. SENTI-202 was well tolerated in both cycles with no DLTs or adverse events of interest or AEIs. The patient experienced Grade-4 hematologic toxicity as shown in the graph on the upper right, consistent with lymphodepletion. That resolved with AML response. The patient also had some Grade-3 infections. The patient continues in CR with about two months follow-up.

Going back to the graph, this patient entered trial with 70% blast in the bone marrow as evaluated by IHC. That reduced by half at the end of one cycle, qualifying for a (technical difficulty) and by the end of cycle two, the red -- the AML blasts had completely cleared and what was observed by morphologic analysis was a normal cellular matter with tri-lineage hematopoiesis. His bone marrow was also found to be negative for AML mutations by next generation sequencing or NGS.

The graph on the right plus the patient's neutrophil count or ANC, platelets or PLT, as well as the peripheral blasts. Upon receiving lymphodepletion, all the blood counts pretty much zero out followed by recovery of the counts, meeting the threshold for a CR and indeed normalization of counts by the end of cycle two.

On the next slide, Slide 17 similarly summarizes Patient Two. Again to start with the case history, patient is a 72-year-old male with FLT3 mutated intermediate risk relapsed AML that relapsed after intensive chemotherapy and prior FLT3 inhibitor. SENTI-202 was well tolerated with some Grade-4 heme toxicity consistent with lymphodepletion, which resolved with AML response, and Grade-2 fever reported as CRS that resolved with supportive care. The patient is currently receiving a second cycle of consolidation therapy.

As shown in the graph on the upper left, this patient entered the study with about 17% blast in their bone marrow, which normalized at the end of cycle one. What you're seeing there at the end of cycle one is healthy blasts. The end of cycle one bone marrow was also sent for assessment for residual AML clones by multi-parametric flow cytometry with a sensitivity of 0.02%, which turned out to be negative. Similarly, on the upper right, this patient two had complete recovery of ANC and platelet count, exceeding the threshold for CR and normalization of his blood counts.

Slide 18 summarizes PK for all three trial patients and includes both cycle one and cycle two data for Patient One. The overall PK profile shows detectable transgene in the peripheral blasts after each dose in both cycles and it is consistent with that observed with allo NK therapies with cells typically detected in the first few weeks and being naturally cleared from the peripheral blood circulation with host immune cell recovery.

Slide 19, in summary, the mechanism of action of SENTI-202 including the engineered targeting of both blasts and LSCs with validated AML targets and the engineered selectivity providing healthy marrow cell protection, along with these early clinical results, are promising indicators of what we hope to be a differentiated clinical profile in this patient population with a huge unmet need.

The team has been executing well this year with our first trial opening, dosed, and now initial clinical efficacy noted in the first dose level. SENTI-202 is well tolerated and the early deep responses in two of the three AML patients with robust count recovery are promising. The PK is consistent with allo CAR-NK therapies.

We look forward to continued dose escalation and reporting additional efficacy and durability data next year. Thank you. Back to you, Tim.

Timothy Lu: Thank you so much Dr. Strickland and Dr. Rajangam for the overview on SENTI-202.

On Slide 20, as a summary, we are very excited to have shared data with you on SENTI-202 from our first initial dose cohort with evidence of deep efficacy and safety. We continue to believe that SENTI-202 is a potential first-in-class logic gated CAR-NK program for AML and as mentioned, this product is designed to incorporate our Logic Gate platform technology to address AML and other heme malignancies with high unmet need.

I wanted to point out here that this Logic Gate platform that we have developed actually works not just in NK cells but also in T cells. And we firmly believe that there are great opportunities for this technology in AML, but also in other cancer types including solid tumors, anywhere

where there is a need for going beyond just a single target to distinguish cancer cells and healthy cells. In addition to that, we have shown that other elements of our Gene Circuit technology platform do work across modality, both cell and gene therapies, and we are quite excited to continue to make progress on our pipeline development.

With that, I want to thank you so much for the time. And operator, we will now take questions.

QUESTIONS AND ANSWERS

Operator: Thank you. Ladies and gentlemen, we will now begin the question-and-answer session. (Operator Instructions) Our first question comes from the line of Daina Graybosch from Leerink Partners. Please go ahead.

Daina Graybosch: Hi, congratulations on the data. I wonder if you could talk about your dose plans going forward. I noticed in the PK, it looks very similar to other NK cell therapies where your [CMAC] sort of declines as you move away from lymphodepletion. I wonder if you could talk about compressed dosing and also second cycle dosing, and in what situations you would do a second cycle. Thank you.

Timothy Lu: Good morning, Daina. Thanks so much for the questions. So I'll pass that over to Kanya to talk about our thoughts on dose options. Kanya?

Kanya Rajangam: Thank you, Tim. Thank you, Daina. So I heard a couple different questions, so let me answer them in order. One is the product -- we are currently evaluating 1.5 billion per dose given three doses in a cycle. The protocol allows us to also evaluate more frequent dosing, up to four or five doses per cycle. That's up to us if you want to open that cohort.

And in terms of your question about how are we thinking of second cycle, our current protocol does allow patients to get a second cycle where we repeat the lymphodepletion and give them the cells as long as they meet both efficacy criteria, which says that they should have at least some blast reduction, and meet safety criteria which is absence or resolution of [AEIs] mainly.

Daina Graybosch: So on the first question, you have flexibility to do that? What kind of data would trigger you to change how frequently you give a dose?

Kanya Rajangam: Thank you for the question, Daina. So, based on the responses we are seeing at our first dose level, obviously that's really promising to have two out of three at three doses. So it'll be a totality of safety, efficacy, as well as PK and other correlatives to see if we even need to give more additional doses. That's something certainly we are actively evaluating internally in terms of, as the study progresses, should we be looking at more frequent and more often dosing. The protocol gives us the flexibility. So look at the totality of all that data at the right time and make that decision.

Daina Graybosch: Got it. Thank you.

Kanya Rajangam: Thanks, Daina.

Operator: Thank you. Our next question comes from the line of Geulah Livshits. Please go ahead.

Geulah Livshits: Hi, good morning, congrats on data and thanks for taking the questions. I was wondering if there's anything about the patient who did not respond to therapy that could point to a mechanism? For example, there's CD33 levels, FLT3, et cetera, or something about the prior treatments, and then I have a follow-up. Thanks.

Timothy Lu: Hi, Geulah, good morning. I will pass it to Kanya to answer your first question. Then we'll take the second question afterwards. Thanks.

Kanya Rajangam: Thanks, Tim. Hi, Geulah. So the third patient -- well, first of all, no drug is going to give us 100% response rate, right? But at the same time, just to answer your question, the third patient came onto our trial with TP53-mutated AML, which as you know is the particularly hard type of AML to treat. And they had -- they were refractory to very brief remissions from previous therapy before they came on trial. So what we are doing, of course, is we continue to enroll patients, is monitor very closely to see if there is something there in terms of, do we need to modify eligibility criteria.

In terms of the CD33 levels for that particular patient, our protocol requires all patients who come on trial to be CD33 positive as assessed locally and they certainly were, they met the criteria. Our plan is once we used to also retrospectively analyze in a central lab, the CD33 levels in a little bit more consistent manner to do those sort of analysis to see if that correlated at all with the responses.

Geulah Livshits: Got it. Great, thanks. And just in terms of the Logic Gate component you talked about, the inhibitory CAR that SENTI-202 contains, what -- I guess, what kind of signals are you seeing that, that part is working? You talked about obviously hematologic recovery that you're seeing. What would it look like if that inhibitory CAR hasn't or wasn't working? In other words, can you talk about the evidence that you're seeing for the proof of concept that it's doing what you expect it to be doing?

Timothy Lu: Yes, Kanya, go ahead and then I'll add on.

Kanya Rajangam: Thanks, Geulah. So in terms -- so obviously, the clearest things of the iCAR working is that robust healthy hematologic recovery, which is what it's designed to do. It's designed to selectively inhibit healthy hematopoietic stem cells from being killed by the SENTI-202 product. So the fact that we saw in both of our CR patients that really robust count recovery is certainly very promising. It's early days still. We are going to continue to monitor this sort of a count recovery for subsequent patients too.

In terms of correlated plans, we do have plans to do [site-off] analysis, so we can take a look at the cell pool in the bone marrow, to see how many of them express endomucin as an example and things of that nature. We plan on doing some of those in a more retrospective grouped analysis fashion.

Timothy Lu: Yes, what I'll add to that is, there have been previous efforts to target targets like FLT3 with T-cell engagers or CAR T-cells or other modalities, and some of those programs have reported or seen challenges with the fact that FLT3 is expressed on healthy bone marrow cells and you can see the suppression of bone marrow in those situations. So, I think another piece of indirect evidence that we'll continue to look for is that we can safely and efficaciously target CD33 and FLT3 with our [Circuit] without seeing those types of toxicities.

And so, that's surely another element as we continue to advance the trial that we'll keep a close eye on.

Geulah Livshits: Fantastic. Thanks. Congrats again.

Operator: Thank you.

Thomas Chung: Looks like we have a few questions that come through the Q&A portal. I'll hand them, I think off to -- the first one off to Tim. The data shown was from September 19th, 2024. Can you provide an update on the patients who were in complete remission?

Timothy Lu: Yes. Thank you. So, with the initial two months of follow-up since that data cut, that we showed in the slides, both patients are continuing to maintain MRD negative, mACR status in remission. So they're at four-plus months and three-plus months, respectively. We are now dosing patients at the 1.5 billion dose level, and we do continue to look forward to providing additional data updates in 2025 on these patients as well as new ones.

Thomas Chung: And the second one -- second question is, are there any insights you can provide on SENTI-202 durability based on the data you have collected to date?

Timothy Lu: Okay. Why don't I turn that over to Kanya?

Kanya Rajangam: Thank you, Tim, and thank you for that question. So what I will say is that in general, it's been shown in AML that achieving MRD negative status correlates with increased durability and it's really heartening to see that both of our patients did achieve that MRD negativity based on the most sensitive methods available at the local sites. So, that we believe is promising in terms of projecting for increased durability.

Dr. Strickland, would you like to add a comment on MRD negativity as well as the durability of response?

Stephen Strickland: Sure. Thank you, Kanya. I think that I can speak from the experience of the patient I treated in the sense that the patient did achieve after one cycle, a very deep remission and was MRD negative and that has been maintained and the patient is successfully moving on to hematopoietic stem cell transplant as well. So, I think we've seen the response to be maintained. We've seen the ability to get subsequent cycles. We've seen -- and maintain tolerability, maintain good or rapid count recovery. So it seems to be sort of the perfect triad of

things that we're seeing. So, but the patients have done well and continue to do well over a period of time.

Thomas Chung: Great. Now, we have the last question through the portal is, how do you think SENTI-202 compares to other drugs for relapse/refractory AML, including menin inhibitors?

Timothy Lu: Yes, so I'll start on this. And so, I think we are very excited about what SENTI-202 could do in relapsed/refractory AML. And as we continue to accumulate and share data about applicability of this product across the broad patient population, in AML, I think we'll continue to share that. One comment just to highlight is, SENTI-202 as is currently designed is not limited to subsets of patients with specific mutations. We are looking broadly about the applicability of this. Kanya mentioned some of the enrollment criteria we looked at as part of our trial. But, most patients in AML are positive for the markers such as CD33, as well as potentially FLT3 that the product targets.

I'll turn it to Kanya and see if she has some more detailed comments about comparisons or ways we think about SENTI-202 fitting into the treatment paradigm.

Kanya Rajangam: Thank you, Tim. I don't have too much to add, maybe just one comment that it is premature to make these comparisons. We are obviously really excited to see the strong initial signal. And as we continue to progress our trial, we are quite confident in our plans to progress the program rapidly towards the late-phase development and beyond, assuming the data continues to hold.

Thomas Chung: Great. We actually have one more submitted question that just came in. Is SENTI-202 cryopreserved prior to dosing, or is it generated fresh for each dose? Did patients number one, two, and three receive the same drug lot of SENTI-202?

Timothy Lu: Thanks for the question. Kanya, do you want to talk about how we are manufacturing at a high level and then dosing these patients?

Kanya Rajangam: Yes, so SENTI-202 is cryopreserved. So, this is an off-the-shelf product. It's available when the patients come on and then they get lymphodepleted.

In terms of the second part of the question, are all of them are from the same lot? Not necessarily. We are developing this as an off-the-shelf product where our belief is that as long as our lots pass through the battery of tests we need to do to release it, that we are developing it as interchangeable as well. So, that's really how we've designed our trial and I hope that answers the question.

Operator: That concludes our Q&A session. I'd now like to turn the call over back to Dr. Lu for final closing comments.

Timothy Lu: Well, we really appreciate everyone taking the time to hop onto the webinar today and learn more about SENTI-202. We are very excited about this product being one of the first logic gated CAR cell therapy that has data and now in humans. And we're really excited about

not just the data we presented today, but also the data we'll continue to share with the community going forward. It's been a pleasure just to talk today and thanks for joining the call. Now, please let us know if you have any additional questions and we look forward to sharing more updates as the trial progresses. Have a great morning.

Operator: Thank you. That concludes our conference call for today. You may now disconnect.