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Elicio Therapeutics and Angion Biomedica Corp.
Merger Call

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PRESENTATION

Operator

Greetings and welcome to the Elicio Therapeutics and Angion Biomedica Corp. merger announcement call.

At this time, all participants are in a listen-only mode. A question-and-answer session will follow the formal presentation. If you would like to submit a question, you may do so at any time throughout the webinar by using the Q&A function below the webcast player. As a reminder, this event is being recorded and a replay will be available following the conclusion of the event.

It’s now my pleasure to introduce your host, David Miller, Angion’s Senior Director of Corporate Affairs. Please go ahead, sir.

David Miller

Thank you, Operator. Please advance the deck to Slide number 2.

Good morning and thank you for joining us on this conference call to discuss the recently announced merger between Elicio Therapeutics and Angion. On the call with me today are Robert Connelly, the Chief Executive Officer of Elicio, and Dr. Jay Venkatesan, Angion's President and Chief Executive Officer.

Prior to yesterday's opening bell, our two companies issued a joint press release describing the merger. The release is accessible in the Investor section of our website at www.angion.com.

In addition, Angion filed a report on Form 8-K with the United States Securities and Exchange Commission containing important information about the merger, including a summary of the key merger terms and the merger agreement. This filing is available via the website maintained by the SEC at www.sec.gov and is also available on Angion's website at ir.angion.com/sec-filings.

This conference call and accompanying presentation include forward-looking statements subject to the safe harbor provisions under the Private Securities Litigation Reform Act of 1995. Actual outcomes and results may differ materially from those contemplated by these forward-looking statements. Please see the cautionary language in our press release of January 17, 2023, and in Slide 2 of our presentation accompanying this conference call, for further information. The forward-looking statements included in this communication are made only as of the date hereof, and Angion assumes no obligation and does not intend to update these forward-looking statements, except as required by law.

Angion will be filing a registration statement with the Securities and Exchange Commission. We encourage you to read it and the other relevant materials filed by us with the SEC, because these documents have or will have important information about the proposed transaction.

At this time, I will turn the call over to Dr. Jay Venkatesan. Jay, the floor is yours.

Jay Venkatesan

Thank you, David.

Good morning everyone and thank you for joining the call.

On July 25, 2022, Angion announced the beginning of a process to explore strategic alternatives for our company. The conclusion of this process will result in the merger of Angion with Elicio Therapeutics. Bob will present Elicio's science and programs in a moment, but I just want to say how delighted I am with our announcement yesterday of this transaction.

During Angion's process, we reviewed numerous strategic alternatives for creating shareholder value. More than 230 parties were contacted to solicit their interest and we received more than 40 written responses. We heard presentations from more than 30 companies and did additional due diligence on a number of these. After reviewing the many alternatives, this transaction with Elicio was the clear choice.

We see tremendous potential in the Amphiphile, or AMP platform, and ELI-002, right at a time when the oncology community is rediscovering the value of cancer vaccines. We believe this merger will provide Angion shareholders the opportunity to meaningfully participate in a company treating cancer patients in an innovative way.

Operator, please advance the deck to Slide 3.

On Slide 3, we summarize a few key terms of the merger agreement. The expected equity split at closing will have 34.5% of the combined company owned by Angion shareholders at the time of the close of the transaction, and 65.5% of the combined company owned by Elicio shareholders, all measured on a fully-diluted basis. Each share of Elicio common stock will convert into a number of shares of Angion common stock equal to an exchange ratio determined as provided in the merger agreement. This split is based upon a relative valuation of the companies of Elicio at $95 million and Angion at approximately $50 million.

As set forth in the merger agreement, the split is based on Angion delivering between $26.5 million and $31.5 million of cash at the close, with adjustments if Angion's cash is outside that range and a minimum net cash requirement of $25 million.

So that Elicio can continue its clinical program for ELI-002 without delay while the merger process works towards a projected close in the second quarter of this year, Angion has agreed to provide a bridge loan of up to $10 million which will be forgiven at closing. Angion advanced the first $5 million upon the signing of the merger agreement yesterday and will advance the next $5 million at a later milestone.

The deal has customary closing and termination provisions. The full merger agreement was, as David mentioned, filed with the SEC yesterday morning.

The next step in the process will be to file a Form S-4 registration statement with the SEC covering our two companies and to obtain approval via a vote from both companies' shareholders for this transaction. As I mentioned, we expect the deal to close in the second quarter of 2023.

At this time, I'd like to introduce Bob Connelly, Elicio's CEO. I've really enjoyed working with Bob and his team to learn about Elicio and ELI-002. I'm looking forward to continuing to work with Bob after the merger as a member of his Board of Directors alongside two other current Angion board members. I'll turn the call over to Bob to describe Elicio's science and its lead program, ELI-002. Thank you.

Bob?

Robert Connelly

Thanks, Jay. I'd also like to echo Jay's words on how much I've enjoyed working with Jay and the Angion team as we've put this merger together. We all look forward to adding Jay's experience to our Board of Directors once the merger is complete.

If we could move to Slide 4.

The Elicio premise is to develop immunotherapies that engage the lymph nodes to generate a more powerful immune response to treat cancer and prevent its return. Lymph nodes are the sites in our bodies where the site immune response is orchestrated. When engaged, the lymph nodes can deliver powerful adaptive immune responses. But unfortunately, it is very difficult to engage the lymph nodes effectively, and most immunotherapies don't, so this limits their effectiveness and can prevent the development of promising therapies.

Our founder, MIT Scientist Darrell Irvine has spent two decades developing methods to enhance the performance of vaccines, with a primary focus on engaging the lymph nodes. Elicio's Amphiphile technology, developed over the last decade in Darrell's MIT lab and since 2017 with Elicio, precisely delivers a wide range of different immunotherapies to the lymph nodes.

We jump to Slide 5.

The most advanced AMP program is ELI-002. This is a lymph-node targeted therapeutic vaccine for mutant KRAS-driven tumors. ELI-002 is designed to stimulate an immune response against the seven KRAS mutations that drive 25% of all solid tumors. ELI-002 has begun the fifth cohort of a Phase 1 dose escalation trial in adjuvant stage colorectal and pancreatic cancer patients. The AMPLIFY-201 trial has accelerated ELI-002 clinical development with a 2-peptide formulation, which targets the two most common KRAS mutations, G12D and G12R. The AMPLIFY 7-peptide Phase 1/2 study will transition ELI-002 development to a two-step peptide formulation in first half of 2023.

The AMPLIFY-201 interim clinical data from mutant KRAS pancreatic and colorectal cancer patients, including the effects of dose on proof-of-concept safety, antitumor biomarkers, relapse-free survival and immune mechanism of action endpoints is anticipated to be presented at a medical conference in first half of 2023.

We also plan to study ELI-002 in combination with checkpoint inhibitor and we have a supply agreement in place with Regeneron for combination trials with their anti-PD-1 LIBTAYO.

ELI-002 has the potential to be a universal, all-stage immunotherapeutic for treating and preventing mutant KRAS-driven cancers, which is 25% of all solid tumors.

We can go to the next slide.

ELI-002 will target the seven most common KRAS mutations, again, that drive 25% of solid tumors, and this creates a multi-billion dollar opportunity with the potential to impact the lives of 93% of pancreatic cancer and 52% of colorectal cancer patients. But as you can see on this slide, the market potential and the need for a therapeutic vaccine for KRAS driven tumors is much broader than the scope of our current trial in pancreatic and colorectal cancer patients. As the development proceeds, we can explore ELI-002 in other cancer types in future trials.

You can move to the next slide.

I want to introduce the AMPLIFY-201 clinical trial and our lead program ELI-002, starting with Slide 8.

Historically, there have been several challenges in this field that have limited the effectiveness of cancer vaccines. On the left side of the slide, one major advance is the smart trafficking of ELI-002 to lymph nodes after sub-Q administration, which generates immune responses of increased magnitude, function, and durability. Other vaccines do not engage the lymph nodes, and by contrast, we simply administer in the same way vaccines are routinely given, as an injection in the upper and lower limbs, and the drug then moves itself, achieving broad distribution to draining lymph nodes. In primates, we have seen AMP therapeutic vaccines going to many nodes after injection, which has the potential to increase the magnitude, the function, and the durability of the immune response generated by this vaccine.

We use immunogens and in this case peptides that are already known to generate immune response in humans. So 25% of solid tumors are dependent on these KRAS mutations, so elimination of the KRAS driver will potentially kill the tumor.

On the right side of the slide, the clinical innovation of advancing the studies in the adjuvant setting is highlighted.

By adjuvant what we mean is the stage where treatment is given after the patient has completed surgery and the initial standard of care treatment, such as chemotherapy. Despite standard therapy, these patients remain at high risk for relapse. Pancreatic cancer patients, as an example, with a persisting biomarker after surgery and treatment generally have about 5.5 months before their disease begins progressing. This allows us to treat with ELI-002 at the perfect time when the number of T cells generated by ELI-002 are maximized compared to the number of residual tumor cells present following surgery and other standard treatments. Treatment at this point has the potential to eliminate any microscopic residual disease in order to give the patient a much longer period of disease control. Other oncology vaccines have typically been used in later line therapy for advanced metastatic disease, and the downside to this approach is that by this time the tumor has commonly developed resistance mechanisms that make it much more challenging for T cells to kill it.

If we could move to the next slide, Slide 9?

We call these trials the AMPLIFY studies because the intent is to amplify the T cell’s ability to engage in the immune attack against the tumor. The study has been designed to enroll a variety of patient histologies with primary focus on pancreatic and colorectal cancer, but patients are eligible if they have any of tumors where RAS mutations are common, including lung, ovarian, bile duct, and gallbladder tumors.

The key criteria are important to allow proof-of-concept assessment with biomarkers at baseline and then watch to see if they are reduced or eliminated with ELI-002 treatment. We also assess blood and tumor itself, when possible, to confirm the mechanism of action validation with the generation of T cell responses from ELI-002 treatment.

Patients are treated with ELI-002 for a period of six months where they receive a priming series weekly for the first month, and then every two weeks for the second month. After a three-month rest period, we boost the responses with weekly doses of ELI-002 for another month. This is quite analogous to many standard vaccination programs with an initial prime and a later boost period. Then we follow the patients for 18 months to characterize the durability of the response.

We can move to Slide 10.

The adjuvant stage is unique because the patients have had surgery so there is a limitation on the use of radiographic endpoints like RECIST. In the development of immune therapy, standard radiographic endpoints are problematic because there can be an atypical pattern of response, that is different than what happens with cytotoxic treatment. In immune therapy, an initial increase in size of the tumor lesion may reflect the destruction of the tumor cells by immune cells, and this is called a pseudoprogression. Pseudoprogression can lead to an inaccurate assessment of the immune therapy effects and incorrect treatment for these patients.

So, we are excited to incorporate in this trial serum tumor biomarkers, like CT19-9 for pancreatic cancer and CEA for colorectal cancer, as well as circulating tumor DNA, or ctDNA, which can all have more rapid identification of antitumor effect than radiographic responses. We can look at their kinetics as well as their completeness for initial characterization of antitumor responses. We can assess how they dovetail with traditional time to event endpoints, like overall survival and relapse-free survival, which have been the endpoints that have been used for product registration. Key opinion leaders are excited about the use of biomarkers such as ctDNA, and the novelty of trial designs like our AMPLIFY studies, as well as the potential that these markers can actually predict outcomes.

The primary endpoint of the planned Phase 2 AMPLIFY 7-peptide study will be relapse-free survival, or RFS, an endpoint that has supported regulatory approval in post-surgical patients.

On the right are listed more detail on the trial's readouts. To measure the proof-of-concept for mechanism of action, we are looking at mutant KRAS specific T cells through an assessment of effector cytokine production using flow cytometry and fluorospot, and assessment of antigen-specificity using tetramer and dextramer assays.

We then conduct antitumor proof-of-concept studies with ctDNA and the serum tumor biomarkers I mentioned to look at change in kinetics from the baseline, such as a change in the doubling time from first evidence of antitumor activity and whether with deeper responses we can see clearance of the ctDNA or serum tumor biomarker completely.

So far in this trial 80% of—I'm sorry. Eighty percent of pancreatic patients relapse within nine months and 80% of colorectal patients relapsed within 13 months. There is great unmet need for novel therapies in this window of opportunity where patients are being monitored for progression by CT scans and known therapies have already been completed.

In a subset of patients, we will be able to show biopsy data and this is important because gastrointestinal tumors have typically been called cold tumors, meaning T cells aren't able to infiltrate into the tumor, which is necessary for the immune system to contribute to control of the cancer. When we see many T cells infiltrating the tumor per high powered field, we can conclude that the tumor is not exhibiting the common features of a cold tumor but is now receptive to T cell infiltration and the immune system can engage in fighting the cancer. This has major implications for Elicio's therapies.

Based on the natural course of history of disease, we will be able to look at the comparison of the radiographic relapse-free survival times, comparing that to the historical data with similar patients post-surgery and initial treatment. Radiographic relapse-free survival may be used as an endpoint to support product approval.

If we can move on to Slide 11?

The design of our AMPLIFY-201 trial includes escalating the dose of ELI-002, and specifically the AMP-CpG adjuvant component of ELI-002, which is a danger signal that signals immune cells in the lymph node that danger has been detected and potentially a strong immune response could be generated.

Initially, we are giving antigen in a 2-peptide formulation to target G12D and G12R KRAS mutations, two of the most common in gastrointestinal disease. Patients have been treated through an ascending series of five dose groups that cover a 100-fold range of doses from 0.1 mg up to 10 mg. The interim data from this study is anticipated to be presented at an upcoming conference later in the first half of 2023. That data will include effects of dose on proof-of-concept safety, antitumor biomarkers, relapse-free survival and immune mechanism of action endpoints. These results will inform the recommended Phase 2 dose, which we expect in the first half of 2023, and will be used as we transition from the 2-peptide formulation to the 7-peptide formulation that allows us to fully cover the mutations that occur in one quarter of all solid tumors. The first 7P formulation Phase 1/2 trial is anticipated to start in the first half of this year.

If we could move to Slide 12?

The clinical program, as you could see on this slide, is an ongoing open label study with new data becoming available from cohorts every quarter. The cohorts are sequential so we have longer follow-up data on the patients treated at the lowest doses, but as the study proceeds we will continue to generate data as patients reach six months post treatment. This is a meaningful timepoint which will allow us to compare ELI-002 treated patients to the historical data of patient progression post treatment.

We intend to move aggressively into later phase development with cohorts that systematically evaluate colon, lung, and pancreatic cancer in Phase 1b development with the 7-peptide formulation. Then we plan to seamlessly transition to the randomized Phase 2 study. The randomization will provide definitive data with the comparison group. There is potential for an orphan designation. These data could lead expedited development pathways with regulatory agencies, such the FDA's Breakthrough designation, or Europe's Priority Medicines scheme. But we'll discuss what data is needed to support a product registration when we present the data from these initial early clinical experience to those agencies that I've mentioned.

If we could move on to the next slide?

On Slide 14, ELI's proprietary AMP technology precisely traffics immunotherapies to the lymph nodes, which we call the schoolhouse of the immune response, enhancing the magnitude, the potency, the functionality and the durability of the immune response.

The lymph nodes are a primary site in our bodies where the vast majority of immune cells are located. It is the site where the natural immune mechanisms of our body are localized. The lymph nodes are where the immune system is accustomed to constantly collecting information about our health and disease in order to orchestrate mechanisms of immunity which protect us from pathogens and tumors. By efficiently targeting these sites within our bodies, we are taking advantage of the power and unique biology of the lymph nodes to improve responses across a broad range of disease.

We can move to Slide 15.

The AMP platform is a very innovative solution and came about through the recognition that most vaccines and some immunotherapies don't generate the desired and strong enough immune response. After injection in the tissues, vaccines and some immunotherapies enter the blood and circulate to immunologically irrelevant or tolerogenic sites, preventing them from effectively engaging immune cells entirely or promoting accumulation in other settings where the immune responses are switched off.

On the right side of the slide, the control of biodistribution is based on the molecular size of the agent. Molecules that are small are preferentially distributed by the blood vessels to irrelevant sites. In contrast, molecules that are larger in size, like protein albumin, are efficiently collected by the lymph vessels and go on to accumulate in the lymph nodes.

Going on to the next slide.

The AMP strategy effectively reprograms the biodistribution of vaccines or immunotherapy agents that are unable to reach lymph nodes on their own, so that they are efficiently targeted for uptake in lymph nodes and delivered directly to the immune cells which coordinate the resulting immune response against disease. This is a bioconjugation methodology where we can use simple chemistry to modify a variety of different therapeutic payloads.

These are all agents which are designed to influence the immune response. Be they a vaccine component or immunotherapy component, they represent different classes of molecules, but the unifying feature is that they are designed to drive immunity, but because of their size they are inherently limited in their ability to get to lymph nodes.

We correct that by creating lymph node targeted versions of these agents by chemically installing the structure you see on the left and then center of this slide. These are two very important functional domains here.

I'll note that albumin is an endogenous lipid transporting protein. Albumin is abundant in circulation and naturally traffics to the lymph nodes. The first domain on the far left of this slide is an albumin-binding lipid molecule. This lipid has been designed to allow binding with endogenous albumin in the tissue and this lipid allows the AMP-modified agent to take on the biodistribution properties of endogenous albumin for lymph node targeting and retention.

To create a better drug, the second domain in the middle is a polymeric linker that we use to improve solubility and pharmaceutical properties of the agents and control the payload release in the lymph node.

We have reduced to practice the incorporation of these antigens, adjuvants, small molecules, and proteins, and have designed the chemistry to be simple to apply to a diverse variety of potential targets.

We can go to the next slide.

On Slide 17, this is one preclinical mouse study where we've demonstrated the enormous functional consequence of lymph node education. In this mouse study, we evaluated the use of a lymph node targeted therapy to train T cells to recognize and destroy tumors that are driven by the human papilloma virus oncogene E7. In this case, the E7 protein antigen is large enough that it gets into the lymph node on its own. The CpG, an adjuvant that tells the lymph nodes to begin the education process, was modified with Amphiphile and compared to conventional CpG adjuvant which does not target lymph nodes.

The AMP modification, which you can see with the red plots, led to a substantial increase, a massive increase in the T cell response, going from about 20% of T cells to approximately 80% of T cells. The increase in the number of T cells also led to a profound outcome shown in the two panels to the right.

The upper right shows the kinetics of tumor size, and while the conventional vaccination had a transient dip in the size of the tumor, 8 of those animals had regrowth of the tumor; whereas with lymph node education, the larger number of T cells induced by AMP-CpG were able to eradicate the tumor in 80% of the animals and these animals all resisted subsequent rechallenge, indicating the presence of robust protective memory responses. It was exciting for us to see the potential of long, durable tumor control.

If we can go on to Slide 18.

We've seen a few major themes in all of our preclinical studies. First, the improvement in lymph node delivery over conventional therapies has been observed in both primate and in mice models. Second, we see enhanced delivery of therapeutic cargos, not just to the lymph nodes, but within the nodes to dendritic cells. These specialized immune cells act as teachers to educate disease-fighting T cells inside this lymph node immune schoolhouse. Third, when the lymph nodes are utilized to orchestrate an immune response, it promotes the development of T cell and innate cell responses which exceed what can be achieved with conventional vaccines in mice and primate studies. The AMP therapeutics have led to durable cures of large aggressive tumors in multiple mouse studies.

We can move on to Slide 19.

Our ELI-002 will continue to be the dominant focus of Elicio, but there is additional value in our early stage pipeline.

We have received grant funding from the Gastrointestinal Disease Research Foundation to extend the lymph node targeting application to target two other mutations for gastrointestinal tumors. ELI-007 targets the mutated BRAF oncogene, which is another driver like KRAS, that is present in subset of patients. ELI-008 targets mutated tumor protein 53, or TP53, and that is applicable to a very large number of tumors.

We have proof of principle for the broad applicability of the platform to target cancers, but we have only just begun to tap the potential for additional platform applications like infectious disease vaccines and cancer cell therapies.

We have published on the infectious disease vaccines including SARS-CoV-2, or COVID-19. We have studied undisclosed infectious disease targets that could be applicable with advantage over conventional therapies, including the recent publication that demonstrates that this platform is amenable to dosing into the nose to generate immunity along the mucosal tracks. This would be especially applicable for infections that spread through the respiratory droplet route.

We also anticipate combining our AMP platform with cancer cell therapies. In preclinical models, the combination with cell therapies that had little to no activity by themselves promoted the eradication of established solid tumors in the majority of the animals tested. We look forward to developing these additional applications through partnerships.

We go on to the next slide.

We have already established two collaborations that we've announced.

Our collaboration with Regeneron is a clinical supply agreement to study ELI-002 in combo with Regeneron's checkpoint inhibitor LIBTAYO. Regeneron will supply LIBTAYO to us at no cost, and we are considering multiple studies and cohorts across multiple tumor types and treatment settings with this combination. The agreement allows us to retain full worldwide rights to ELI-002; Regeneron has no rights, and the agreement does not restrict freedom to operate with ELI-002.

Our collaboration with the Gastrointestinal Disease Research Foundation provided grant funding for ELI-007 and ELI-008. Elicio retains full worldwide rights to these programs.

We are continuing to explore partnership in additional applications of the AMP platform for therapeutic cancer vaccines, infectious disease vaccines and cell therapies.

In conclusion, let me just say a few things about our executive team which I believe is the strongest asset within Elicio.

On Slide 22.

This is a very experienced team. The experience is highly relevant to our mission and the all key pieces are in place. We have a great combination of large pharma, biotech, and entrepreneurial experiences and successes. Several of the people on this slide have been the first employee in a company that went on to have a very successful exit.

I think I am pretty confident in saying that we know what it takes to build a company from the ground up with a novel platform technology and lead it to success.

I will hand this back over. Thank you.

Jay Venkatesan

Thank you, Bob, for the update on Elicio's science and programs.

Operator, we would now like to open the call for questions.

Tara Sobierajski

At this time we will be conducting a question-and-answer session with our speakers.

As a reminder, you may do so by using the Q&A text box at the bottom of the webcast player or by emailing your questions to questions@lifesciadvisors.com. Please hold for a brief moment while we poll for questions.

Our first question comes from Justin Kim from Oppenheimer. Please go ahead, Justin.

Justin Kim

Hi. Good morning and thanks for taking our questions.

Maybe just to start, based on what you've seen in preclinical studies and know so far about 002, do you anticipate sort of a dose response, a dose-related immune response across the higher dose levels being investigated? Just trying to understand what you're looking at in terms of the RP2D.

Robert Connelly

Yes, in preclinical models this has been a dose responsive drug, and so while I can't speak specifically to what we've seen so far in the earlier doses, I can speak to data which we presented on the preclinical models which definitely showed a dose response.

Justin Kim

Okay. Can you talk a little bit about ctDNA in the adjuvant setting of some of these initial indications? Just sort of curious in pancreatic and colorectal cancer how widely used it is, what sort of investigations are currently being implemented with the biomarker.

Robert Connelly

Sure. The FDA approvals of cancer treatments based on biomarkers rather than on tumor histology have already been granted. There is recent FDA guidance notes that ctDNA could be used to help in signal finding in early phase clinical trials. So, the use of biomarkers as surrogate endpoints in an approval in time to benefit the ELI-002 program is still being assessed. But overall survival or an alternate primary endpoint will be a conversation we will have to have with regulators when we're discussing our pivotal trials.

Justin Kim

Understood. Maybe just a final one from me. In terms of the AMPLIFY study, can you just talk a little bit about the surgical outcome inclusion criteria? How homogenous this patient population may be from the perspective of relapse rates, and any evidence of sort of any variation based on KRAS subtype?

Robert Connelly

No. No evidence of variance based on KRAS subtype. Typically, these patients have gone through either chemotherapy and then surgery or surgery and then chemotherapy and exhibit one of the biomarkers that we've mentioned, either 19-9 or CEA and/or ctDNA. So, we see not a major variance within these patients that we enrolled in the study.

Justin Kim

Okay, great. Thanks for taking the questions.

Jay Venkatesan

Thank you, Justin.

Tara Sobierajski

Our next question comes from Ram Selvaraju from H.C. Wainwright. Please go ahead, Ram.

Ram, you might be on mute?

Ram Selvaraju

Can you hear me?

Tara Sobierajski

Yes.

Ram Selvaraju

Sorry about that.

Firstly, I just wanted to ask how you are thinking about the overall KRAS mutant space and how your approach is likely to fit into this overall continuum given the extensive activity that has already occurred with respect to the development of small molecule therapeutics that specifically target KRAS mutated cancers. In particular, if you could provide some context around your indication selection and prioritization approach relative to these things.

Robert Connelly

Sure. I think just really four categories that you can break the KRAS space into. And you're correct, there sure has been a lot of activity in this space.

You mentioned the small molecules that directly inhibit mutant KRAS, we have the Amgen product, we have Mirati, and there's a number of other companies working in that space. The issue there is that there's a short duration of response and we are already seeing mutant escape in those patients.

The second segment are other small molecules that inhibit the pathway of KRAS and they have—we expect them to be pretty much in the same class with a smaller degree or a smaller period of effectiveness and also subject to mutant escape. So, moving from a G12C to another mutant as the disease progresses.

Now you have the emergence, as Jay said at the beginning, of vaccines that target mutant KRAS, and some interesting data that are emerging from those as well. What we've seen with those technologies, both in the preclinical data that they've published as well as what we know about the trials, that number one, they typically don't target all seven of the mutations, which we believe is critical. They also have not demonstrated the kind of massive T cell increases that we've spoken about and that we've seen in preclinical studies.

So, really the fourth category is lymph node target and believe we're the only player in that category. The vaccines in the third category I mentioned typically have very little interaction with the lymph nodes, but in our case we're targeting all seven of the key mutations. We're going to the lymph node, which we believe will really—so those two points right there and the resulting magnitude of the T cell response, the functionality of the T cells and the durability and the learning that has taken place in the lymph nodes really differentiates us from that third group of vaccines, and very much so from the small molecule approaches.

I think that's how we really kind of try to summarize it in a simple way.

Ram Selvaraju

If we look at your clinical development paradigm, particularly as this pertains to the utilization of the biomarkers, how would you characterize this as being meaningfully differentiated from prior cancer vaccine approaches overall?

Robert Connelly

Well, I think that one of the major differences is that most of the prior vaccine experiences have been in later stage trials where you have typically cold tumors, typically metastatic, a metastatic setting, and so the bar there is extremely high, particularly for vaccines that don't generate the kind of T cell response that we do.

We're going at an earlier stage initially, but that is not the only stage that we see this technology applying in, and we believe there is both a path for monotherapies as well as combination therapies, and perhaps combination therapies at all different levels with our approach.

Ram Selvaraju

Then lastly, you had mentioned specifically talking about the point of intervention, this being important to ensure that cells that are trained in the lymph nodes do not encounter a recalcitrant tumor microenvironment. I was just wondering whether you could elaborate on this, specifically within the context of the kinds of combinatorial regimens that are most likely to be applicable in the event that these cells do encounter a resistant tumor microenvironment that may be difficult for them to overcome in the monotherapy context.

Robert Connelly

Yes. I think to prioritize those, we—and this is where our Regeneron relationship comes in. We would look probably initially at where these checkpoint inhibitors have already been approved and then combine with those in those cancers and at those stages. That gives us actually a fairly rapid—we believe it could provide a fairly rapid path to registration because you're working with a product that's approved. The initial announced target for our relationships with Regeneron is in advanced stage lung disease, so that's a good example of that.

Ram Selvaraju

Thank you.

Tara Sobierajski

Thanks for the questions, Ram.

This concludes the verbal portion of our Q&A session. I'll now turn it back over to David to read the remainder of the questions.

David Miller

We have a question: What kind of runway does the cash from the transaction provide?

Jay Venkatesan

I will take that, and Bob, if you want to comment, feel free.

Based upon the minimum agreement threshold of $25 million and assuming no added financing, we expect cash until the end of the year 2023 at a minimum. Then, obviously that would be different depending on what cash Angion ultimately delivers at the time of the close.

David Miller

The next question is, can you speak to the status of Angion's programs going forward?

Jay Venkatesan

As mentioned previously on conference calls, following the Phase 2 results for 3070, Angion paused that program and also paused 3777. Angion has a number of clinical and preclinical programs which we do not intend to further develop internally at the combined Elicio/Angion. We do, however, think that there may be an opportunity to out-license one or more programs and are at least looking into those options to see whether there are interested parties who could in-license these programs from us. At this point in time though we don't have any plans to further develop these programs.

David Miller

The next question is, you indicate a number of data presentations in 2023. What kind of data should we expect to see?

Jay Venkatesan

I'll let Bob take that question.

Robert Connelly

I think, first off, it's hard to predict what some of them will be, but the initial presentation that we will make, hopefully in the first half of this year, should be a very data-rich presentation on the AMPLIFY-201 study. This is the Phase 1 with the 2-peptide version of our lead program ELI-002. I think as I went through the slide I mentioned everything from the biomarkers that we presented, the dose response effect on those, dose response effect on safety, looking at impact to relapse-free survival. So, a quite data-rich package on five cohorts of study, somewhere over 20 patients in this. Then we'll continue to follow the patients which gives us additional opportunities to present. Then from there, really before the end of this quarter probably, we'll start the first trial with the 7-peptide version of this, of ELI-002 and then pretty quickly move on to the first Phase 1b. The first Phase 1b will probably have initial, probably interim data either towards the late in this year or early in next year.

And as we've mentioned, we have a lot of other programs in the pipeline which also give us opportunities to talk about what we're doing with the technology.

I think from this company, news flow is an absolute asset that we have, both with the lead program which can be applied in so many different KRAS indications, both as a monotherapy and as a combination therapy, in itself is a major generator of news flow. But we also have the other programs and the other uses of the platform and things that we're doing in collaboration and will do in collaboration to move other programs forward.

David Miller

Great. For our last question, can you talk about where the sites are for the studies for the ELI-002 program?

Robert Connelly

I'm not sure that I can mention them all individually, but I can tell you that they are basically household names and some of the great, best cancer institutes, cancer research institutes in the United States. All the sites are in the United States. We go from the east coast to the middle of the country, all the way out to California, and we have 10 sites. We're working with what we feel are the very best research sites in the United States.

Tara Sobierajski

Great. Thank you everyone for joining us today. This concludes today's call. You may now disconnect.

Additional Information and Where to Find It

The transaction referenced in the above communication has not yet been consummated. This communication is for informational purposes only and is not a substitute for any materials that Angion Biomedica Corp. ("Angion") will file with the U.S. Securities and Exchange Commission (the "SEC"). In connection with the proposed merger between Angion and Elicio Therapeutics, Inc. ("Elicio"), pursuant to the Agreement and Plan of Merger and Reorganization, by and between Angion and Elicio, dated January 17, 2023 (the "Merger Agreement"), Angion has filed and/or intends to file relevant materials with the SEC, including a Current Report on Form 8-K (the "Form 8-K") and a registration statement on Form S-4 that will contain a proxy statement and prospectus to register the shares issued (the "Form S-4"). ANGION URGES INVESTORS AND STOCKHOLDERS TO READ THESE MATERIALS CAREFULLY AND IN THEIR ENTIRETY WHEN THEY BECOME AVAILABLE BECAUSE THEY WILL CONTAIN IMPORTANT INFORMATION ABOUT ANGION, ELICIO AND THE PROPOSED TRANSACTION AND RELATED MATTERS. Investors and stockholders will be able to obtain free copies of the Form 8-K, the Form S-4 and other documents filed by Angion with the SEC (when they become available) through the website maintained by the SEC at www.sec.gov. In addition, investors and stockholders will be able to obtain free copies of the Form 8-K, the Form S-4 and other documents filed by Angion with the SEC by contacting Investor Relations by email at investors@angion.com. Investors and stockholders are urged to read the Form 8-K and the Form S-4, including the proxy statement / prospectus contained therein, and the other relevant materials when they become available before making any voting or investment decision with respect to the proposed transaction.

Cautionary Statement Regarding Forward-Looking Statements

This communication contains forward-looking statements within the meaning of Section 21E of the Securities and Exchange Act of 1934, as amended, and the Private Securities Litigation Reform Act of 1995, known as the PSLRA. This includes statements regarding: the anticipated completion and effects of the proposed merger; anticipated communications regarding each of Angion's and Elicio's entry into the Merger Agreement; and other statements regarding management's intentions, plans, beliefs, expectations or forecasts for the future, and, therefore, you are cautioned not to place undue reliance on them. No forward-looking statement can be guaranteed, and actual results may differ materially from those projected. Angion undertakes no obligation to publicly update any forward-looking statement, whether as a result of new information, future events or otherwise, except to the extent required by law. Angion uses words such as "anticipates," "believes," "plans," "expects," "projects," "future," "intends," "may," "will," "should," "could," "estimates," "predicts," "potential," "continue," "guidance," and similar expressions to identify these forward-looking statements that are intended to be covered by the safe-harbor provisions of the PSLRA. Such forward-looking statements are based on Angion's expectations and involve risks and uncertainties; consequently, actual results may differ materially from those expressed or implied in the statements due to a number of factors, including, but not limited to, risks relating to: the completion of the merger, including the need for stockholder approval and the satisfaction (or waiver) of closing conditions; the ability of Angion to remain listed on the Nasdaq Global Market; and the occurrence of any event, change or other circumstance or condition that could give rise to the termination of the Merger Agreement.

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Participants in the Solicitation

Angion and Elicio, and each of their respective directors and executive officers and certain of their other members of management and employees, may be deemed to be participants in the solicitation of proxies in connection with the proposed transaction. Information about Angion's directors and executive officers is included in Angion's Annual Report on Form 10-K for the year ended December 31, 2021, filed with the SEC on March 30, 2022, and the proxy statement for Angion's 2022 annual meeting of stockholders, filed with the SEC on April 27, 2022. Additional information regarding these persons and their interests in the transaction will be included in the proxy statement / prospectus included in the Form S-4 relating to the transaction when it is filed with the SEC. These documents can be obtained free of charge from the sources indicated above.

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