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This filing relates to the proposed transaction pursuant to the terms of that certain Agreement and Plan of Merger and Reorganization, dated as of January 30, 2024, among AVROBIO, Inc., a Delaware corporation ("AVROBIO"), Alpine Merger Subsidiary, Inc., a Delaware corporation ("Merger Sub") and a wholly-owned subsidiary of AVROBIO, and Tectonic Therapeutic, Inc., a Delaware corporation ("Tectonic") (the "Merger Agreement"), pursuant to which, and subject to the satisfaction or waiver of the conditions set forth in the Merger Agreement, Merger Sub will be merged with and into Tectonic (the "Merger"), with Tectonic continuing after the Merger as the surviving corporation and a wholly-owned subsidiary of AVROBIO. The following is a transcript of the joint conference call and webcast hosted by AVROBIO and Tectonic on January 30, 2024 to discuss the announcement of the proposed Merger transaction involving AVROBIO and Tectonic. The slides that are referred to herein are furnished as Exhibit 99.1 of the Current Report on Form 8-K filed by AVROBIO with the Securities and Exchange Commission on January 30, 2024.

AVROBIO and Tectonic Therapeutic
AVROBIO and Tectonic Therapeutic Merger Agreement Conference Call
January 30, 2024

Presenters

Erik Ostrowski, CEO, AVROBIO
Alise Reicin, CEO, Tectonic Therapeutic

Operator:

Good morning and welcome to today's joint conference call regarding the Tectonic and AVROBIO merger. At this time, all participants are in listen-only mode. Please be advised that the call is being recorded.

Now, I'd like to turn the call over to Erik Ostrowski, Interim Chief Executive Officer and Chief Financial Officer of AVROBIO. Please go ahead.

Erik Ostrowski:

Thank you, operator, and good morning, everyone. Joining me on today's call is Alise Reicin, Tectonic's President & CEO.

Before I begin, I want to remind everyone that this discussion and the accompanying presentation will contain forward-looking statements based upon the current expectations of AVROBIO and Tectonic, which include, but are not limited to, statements regarding the expected timing, completion, effects and intended outcomes for the proposed transactions, and our future expectations, plans and prospects for the combined company. Such statements represent management's judgment and intention as of today and involve assumptions, risks and uncertainties.

Further, as indicated on these slides, AVROBIO intends to file a registration statement and accompanying proxy statement and prospectus with the Securities and Exchange Commission relating to the proposed transactions. Please be advised to read, when available, these and other relevant documents filed with the SEC. Please refer to the accompanying slides for more details on these forward-looking statements.

Turning to the transactions, earlier today, we issued a press release that outlines a merger agreement between AVROBIO and Tectonic Therapeutic, a privately-held clinical stage biotechnology company focused on developing GPCR-targeted therapeutic proteins, and the related private placement financing. This release is available at avrobio.com under the Investors and Media tab.

As we communicated in July of last year, AVROBIO halted further development of its programs and conducted a comprehensive review of strategic alternatives for its business, with a focus on maximizing shareholder value. Following this thorough review, our Board of Directors believes the proposed merger announced this morning is in the best interests of our shareholders.

Tectonic is advancing a pipeline of novel therapies made possible by unlocking the potential of biologics in the GPCR space. The company is at an exciting stage in its development, having generated exciting initial clinical data with its Fc-relaxin fusion protein, TX45, a potential best-in-class agent, focused on addressing areas of high unmet need in cardio-pulmonary indications. Tectonic's programs are enabled by its proprietary GEODe™ platform which aims to overcome major limitations of antibody-discovery addressing GPCRs, and has the potential to develop novel compounds across a broad range of indications.

I will now provide an overview of the transactions, including the private placement financing.

In support of the combined company's future operations, Tectonic has entered into subscription agreements for a $130.7 million private placement with a syndicate of new and existing leading institutional life sciences investors. These proceeds, together with existing cash expected from both companies at closing, are expected to provide the combined company with approximately $165 million of cash and cash equivalents at closing. These cash resources are currently expected to fund operations into mid-2027 and intended to be used to advance Tectonic's pipeline through multiple clinical data catalysts.

After giving effect to the private placement financing, pre-merger AVROBIO shareholders are expected own approximately 22% of the combined company, pre-merger Tectonic stockholders are expected to own approximately 40% of the combined company and purchasers in the private placement are expected to own approximately 38% of the combined company, subject to certain assumptions. The percentage of the combined company that AVROBIO shareholders will own as of the close of the transaction is subject to adjustment based on the amount of AVROBIO net cash at the closing date.

The merger and related financing are expected to close in the second quarter of 2024, subject to satisfaction of closing conditions for each transaction. Upon closing of the Merger, the combined company is expected to operate under the name Tectonic Therapeutic, Inc. and to trade on Nasdaq under the ticker symbol TECX.

Phillip Donenberg from the AVROBIO board of directors will join the Tectonic board at the time the transaction closes. We believe this transaction will result in a combined entity with a strong cash position backed by an impressive investor syndicate, and are confident in the Tectonic leadership team's ability to deliver on their vision.

I'd now like to turn the call over to Alise, who will present an overview of Tectonic's business and plans.

Alise?

Alise Reicin:

Thank you, Erik.

The Tectonic team and I are also delighted with this transaction, and I want to start by thanking the AvroBio team for their really outstanding collaboration throughout this process.

The total funds generated from this process will be critical to advancing Tectonics vision of creating novel biologic therapies for patients and creating value for our shareholders. And we are excited to share that vision with you today.

At Tectonic we are leading the way in the development of GPCR-targeted biologics the development of which has been a real challenge in the field. To be successful we need the right technology, the right assets and development plans, and the right people. And I believe we have all of these.

Tectonic has a validated platform that we call GEODe that enables us to reproducibly discover GPCR targeted biologics.

GPCRs have been a rich area of drug discovery and development but the overwhelming majority of approved drugs against this class of targets are small molecules.

We're not aiming to replace small molecules where these have worked well.

Instead, our focus is on those targets where we think a biologic is the optimal modality to unlock the potential of that target.

We have a rich emerging pipeline with our first 3 compelling targets against high unmet need indications including two where there is no approved therapy.

Our lead asset, an Fc-relaxin fusion protein, is in Phase I and the early data is quite promising, suggesting it has the potential to be a best-in-class therapy for a mechanism that has blockbuster potential in cardiopulmonary diseases. We will have more complete PK/PD data later this year to reinforce the profile of the drug. This will be followed by Phase 1B Hemodynamic data in 2025 and Randomized Phase 2 data in 2026.

Tectonics leadership team and founders have an extensive history of drug discovery and development success resulting in the first approval of 20 drugs and many lifecycle management indications

We have a great group of investors and post-closing we'll have cash that is expected to provide runway into mid-2027 with multiple value creating inflection points along the way.

I want to spend a few minutes telling you about my leadership team with a focus on the scientific leaders**.**

I'm a physician scientist and a 25+ year pharma veteran. My first 19 years were at Merck Research labs where I worked across more than 10 therapeutic areas and both early and late drug development. I led the Keytruda program from first in man through its initial approval. Prior to coming to Tectonic, I was President and Head of Global Clinical Development at Celgene.

Peter McNamara our CSO joined us after 15 years at Novartis where he worked across therapeutic areas and modalities. He's had 16 INDs and 2 approved drugs.

Tony Muslin our CDO was a professor of cardiology at Wash U and then spent 10 years at Novartis and Sanofi in leadership roles in cardiovascular and metabolism and

Marcie Ruddy our CMO spent 10 years at Merck working across therapeutic areas in early development and most recently was VP of Clinical Immunology at Regeneron where she led the Dupixent team through multiple Phase II, III and life cycle management approvals.

Tim Springer and Andrew Kruse, our founders who both remain active in the company, are Professors at Harvard Med School.

Tim is not only a highly respected scientist and recent winner of the Lasker award, but also a very successful serial entrepreneur.

Andrew is a world-renowned expert in the biochemistry of GPCRs.

Now let me give you a sense of the breadth of opportunity available to us.

GPCRs are cell surface proteins that play a major role in transmitting signals from outside the cell to inside the cell.

They have been an incredibly rich area for drug discovery and development, with over 30% of all currently marketed drugs modulating GPCRs; but these approved therapies only address 12% of known GPCRs, so many potentially compelling targets remain untapped.

Because of the challenges of making GPCR targeted biologics, of the approved drugs there are only 3 GPCR targeted antibodies, so there is a significant opportunity for biologics to play a much bigger role in this space.

The right hand of the slide outlines those places where we think a biologic might be the better modality over a small molecule.

First, there are GPCRs that have been very difficult to target with small molecules, where we believe a biologic is more likely to be successful.

Also, there are families of GPCRs where the target site is very similar across members of the family, and small molecules often cannot provide the needed specificity resulting in off target toxicity. Biologics are likely to provide that specificity.

We also have a use case, where small molecules have been on the market for decades but there's a black box warning for a high unmet need indication because of very specific tissue toxicity in that indication. We have the ability to make a biologic that can avoid that specific tissue potentially enabling the development of a novel drug in an indication without approved therapies.

And lastly, you can develop bi or tri-specifics, to achieve multi-target impact, which you can't do with small molecules.

Now turning to the first 3 programs in our pipeline.

Our lead program as I mentioned is an Fc-relaxin Fusion protein optimized using the protein engineering part of our GEODe platform. We are initially developing it for a subset of Group 2 pulmonary hypertension which is pulmonary hypertension in patients with left sided heart failure. I'll describe this program in detail in the coming slides, but we believe we have an ideal match between the mechanism of our drug and the pathophysiology of the disease.

Next, we have a GPCR antagonist, which was discovered using all 3 pillars of our GEODE platform. It acts as an anti-angiogenesis agent targeting hereditary hemorrhagic telangiectasia, otherwise known as HHT, which is a disease arising from abnormal vascular formation. It is the second most common hereditary bleeding disorder and there are no approved therapies for patients affected.

There are mouse models of HHT which have been shown to translate into efficacy in the clinic and we have early data demonstrating efficacy in one of these mouse models of the disease.

We anticipate choosing a development candidate in the coming months.

In our third program, we are taking a bispecific approach to fibrosis, targeting two targets expressed on the same cell but with complementary pathways.

One of the targets already has proof of concept in the clinic, and we believe this has the potential to be a best-in-class approach to treating fibrotic diseases.

In every one of our programs, we know how to efficiently get to proof of concept in a capital efficient way.

In terms of timelines, we expect this raise will give us a greater than 3-year runway which enables us to achieve multiple value creating milestones across our program. This should include getting us through POC for relaxin and getting our second program in HHT into the clinic in Q4 2025 or Q1 2026, with the potential to start Phase 2 in 2026.

Other assets can then be used for partnership opportunities, and discovery deals using our platform can be considered. We believe there is no systematic platform risk because the risk of the assets are uncorrelated.

Now let's turn to the challenges in the discovery of biologics targeting GPCRs, and how our GEODe platform addresses them.

First, GPCRs are embedded in the cell membrane and if you extract them from the lipid membrane to produce material for screening, the receptors often cannot retain their native conformation.

Second, GPCRs are often expressed at very low levels, which creates an additional obstacle to purifying enough material for a screen.

Third, they are often conserved across species, so, trying to induce antibody formation against them through immunization in animals is often unsuccessful, because it requires breaking tolerance.

Last, because GPCRs spend most of their time in an inactive state, developing agonists is especially challenging as you need to stabilize the receptor in an active form for agonist discovery.

The GEODe platform we assembled over the last three years, consists of three main components to address these challenges:

1. First, it includes a wide range of biochemistry techniques and GPCR structural insights that help us boost receptor expression and stabilize them outside cell membranes.
2. Second, we have Proprietary yeast-display libraries and sorting protocols to productively generate hits without the need for animal immunization.
3. And finally, the platform has protein engineering techniques to produce chaperones that can stabilize GPCRs in active form and also to optimize lead molecules as they arise.

So switching to our lead program, TX45, which is an RXFP1-agonist, based on a relaxin-fusion protein.

So what do we know about the human hormone relaxin? Well, it's is often referred to as the hormone of pregnancy because it is upregulated during pregnancy. And what it does in pregnancy helps inform what we hope it will do in patients.

First, it's a pulmonary and systemic vasodilator, which enables the pregnant person to increase cardiac output to accommodate the increased demand from the developing fetus. It also demonstrates antifibrotic effects as it remodels the pelvic ligaments to enable a vaginal birth.

What else we know about relaxin came from the Novartis serelaxin program – the first major attempt at developing a recombinant form of the hormone as a cardiovascular therapy.

But the major issue with serelaxin was its short half-life which required that it be given by continuous IV infusion. This limited development to Acute Heart Failure where a 2-day infusion was given on top of standard of care. A meta-analysis of 6 studies, with more than 11,000 patients demonstrated both its safety and activity with a 23% reduction in 5-day worsening heart failure. However, one of two pivotal studies, which included 6-month cardiovascular mortality, as a co-primary endpoint, failed. Expecting a 2-day infusion to impact a 5-day endpoint, let alone expecting an impact on an endpoint six months later was a high bar to set in the study.

We've used the engineering capabilities of our platform to address the half-life issue and enable development in the optimal disease settings. Specifically, we increased the half-life by making an Fc-relaxin fusion protein. On the right you can see rat PK for one of our early compounds in black and for a competitor relzxin Fc fusion, in orange. For both, you can see the half-life is prolonged from minutes to days, but you can also see you're losing 90% of the exposure in the first few hours, which is not typical for Fc fusions. It turns out, it is typical for Fc fusions for antibodies with a high isolectric point, and both these molecules have a high pI, as does native relaxin. The high pI means these are highly charged proteins which have been shown to bind to heparin sulfates in the glycocalyx, which traps them so they get pulled out of the blood, reducing the free drug that then can get into the tissues.

We hypothesized that we could increase overall exposure and improve free drug that could penetrate the tissue if we engineered our compound to lower its isolelectric point. Our clinical compound TX45 can be seen in green, and as you can see, with a reduced pI we have a ten-fold increase in exposure.

How does this translate into potency in animals? Renal blood flow in rats is an excellent pharmacodynamic marker for relaxin because it captures its rapid systemic vasodilation effects. That makes it an ideal setting to benchmark our compound against a high pI comparator. Start on the left of this slide. As I mentioned, when you give equal amounts of these drugs, you get a ten-fold increase in exposure with TX45. However, engineering to reduce the pI, also decreased potency in vitro by a factor of ten. If this was just about optimizing blood PK, one would expect that equal amounts of the drugs, would show equal in vivo potency, because you would need ten times more TX45 to activate RXFP1.

However, despite the decreased in vitro potency, in vivo, as you can see, our drug is ten times more potent than the comparator compound. 0.3 mpk of the high pI comparator compound has similar effects on renal blood flow as 0.03 mpk of TX45. In contrast, 0.3 MPK of TX45 shows much greater effect than the comparator. And we attribute this to less drug trapping in the glycocalyx, resulting in more free drug becoming available to activate the receptor in tissues.

We have data suggesting we have a potential best in class relaxin fusion protein, and we've chosen Group 2 Pulmonary hypertension in patients with Preserved ejection fraction heart failure (HFpEF) as our first indication for several reasons:

First, Group 2 PH with HFpEF is a substantial unmet need population with no approved therapies: it is a large population in the US and the five-year mortality in this disease is high.

Second, the mechanism of action of relaxin matches the pathophysiology of the disease.

We also believe there is a clear, reasonably quick path to approval because an outcome study is not needed to get approval or reimbursement in Group 2 PH, which should enable an earlier entry to market compared with CHF. Also, there may be a pricing and reimbursement advantage if the first indication is in a subset of CHF, with highest unmet need.

Beyond Group 2 PH there is the potential to expand to other types of pulmonary hypertension, to larger heart failure indications, and also renal disease.

When people talk about pulmonary hypertension, they're usually referring to Group 1 PH, also known as PAH, because that is where most drug discovery and development efforts have focused to date. Group two, PH is pulmonary hypertension associated with left heart failure. And over the last few years, two subtypes have been identified which I will describe on the next slide. Preserved ejection fraction heart failure (HFpEF), affects several million patients in the US, and more than 600,000 of those have elevations in the pulmonary artery pressure and therefore have Group 2 pulmonary hypertension. More than 500,000 of these have Ipc-PH, isolated post, capillary pulmonary hypertension.

With these patients, elevated pressures in the left side of the heart, backflow into the pulmonary circulation, leading to elevated pulmonary pressures. Their pulmonary vasculature is normal. And if you can treat their heart failure adequately, which has been difficult with current therapy, their pulmonary artery pressures should come down.

In contrast, combined pre and post capillary pulmonary hypertension (Cpc-PH) represent more than 100,000 patients in the US. These patients have both increased pulmonary pressure due to backflow, but in addition, their pulmonary vasculature starts to look like Group 1 PH. They get muscularization of the arteries and a narrowing of the lumen. And as blood is pushed through a smaller lumen you get an increase in pulmonary vascular resistance, which is how you differentiate them from patients with Ipc-PH.

In our Phase 2 study, we are going to enrich for Cpc-PH patients because if we ran an all-comer study, it would mainly enroll Ipc-PH. With a more balanced population, we will be able to determine whether TX45 works in either or both subsets, which provides 2 shots on goal.

So, how does TX45 work and why do we believe it is ideal in this setting?

First as I mentioned, it's both a pulmonary and systemic arterial vasodilator. And it does that by inhibiting ET-1 (on the left) and by activating the production of nitric oxide (in the middle). It is also an anti-fibrotic and remodeler of the pulmonary arteries and the heart. And it does this by activating matrix metalloproteinases 2 and 9 and inhibiting the TGF-Beta pathway via inhibition of phospho-SMAD 2 and 3.

So putting it together you can see the pathophysiology of Group 2 PH on the left in this table and how relaxin's MOA addresses this on the right.

We have already spoken about pulmonary artery narrowing in CpC-PH and the fact that relaxin both dilates and remodels the pulmonary arteries. Long standing Group 2 PH can lead to dysfunction of the right side of the heart as well and preclinically relaxin has been shown to improve this. In preserved ejection fraction heart failure, you get a thick and stiff left ventricle that doesn't relax to allow adequate filling of the heart in diastole.

Relaxin could increase cardiac output via 3 different mechanisms.

- One, by dilating the peripheral circulation making it easier to pump blood out of the heart (that's called decreases afterload)
- Two, by relaxing the heart muscle, diastolic filling should improve
- And lastly with long term treatment remodeling of the left ventricle should further enhance function.

Lastly, many of these patients have compromised renal function and relaxin, via dilation of the renal vascular system has been shown in the clinic to improve kidney function.

So in sum, relaxin's actions really match the pathophysiology of the disease. So just a brief comparison of Group 2 PH versus Group 1. Group 2 PH has many more patients in the US than PAH, and like PAH, is likely to be a multi-billion-dollar opportunity. In the middle you can see that Survival is similar to, or worse than PAH, so the unmet need is high. And lastly, unlike PAH where there are many approved drugs, there are no approved therapies for Group 2 PH aside from drugs used to treat heart failure.

A logical question is whether the drugs approved for PAH also work in Group 2 PH. To date, studies evaluating these have failed. We think one reason is that some of these drugs mainly by dilating the pulmonary vasculature. If you're doing nothing to enhance cardiac output, you're going to push more blood into the left side of the heart, but the heart can't push that blood out and heart failure can worsen.

The PDE5 inhibitors also failed in an all-comer Group 2 PH population but in 3 separate studies, in patients with Cpc-PH, they demonstrated improvement in hemodynamics, exercise tolerance, and a reduction in heart failure hospitalizations. We think this increases the probability that TX45 will work in Cpc-PH.

And the reason for that is reflected on this slide. PDE5 inhibitors work by activating the nitric oxide pathway, by inhibiting the breakdown of nitric oxide.

I think of relaxin as being a "PDE5 inhibitor plus" because it not only activates the nitric oxide pathway, it does also all the other things we already talked about. So based on this, we believe TX45 has the potential to be effective both in CpCPH and in IPCPH. The other data that we believe increases our probability of success in Group 2 pulmonary hypertension comes from the serelaxin program.

A right heart cath study was done in patients with acute heart failure, where pulmonary and cardiac hemodynamics were measured pre and post starting a serelaxin infusion.

In orange are the data from patients treated with a 20-hour infusion of relaxin and in blue are data from placebo.

Focus on the first 8 hours, as diuretics were allowed after that. You can see a drop in pulmonary artery pressure on the left, pulmonary vascular resistance in the middle, and a drop in systemic vascular resistance on the right. Not shown here, was a drop in pulmonary capillary wedge pressure, which is a measure of heart failure, and also an increase in cardiac index. In a similar study in chronic heart failure an increase in cardiac output and a decrease in wedge pressure was demonstrated.

We're going to do a very similar study to this in Phase 1B in patients with Group 2 PH and HFpEF. We are in the middle of our phase one study, and in addition to getting pharmacokinetics, we're getting renal blood flow data several time points after dosing. We found that the exposures that give you near maximal effects on renal blood flow are the same exposures where you get the chronic effects in the pulmonary hypertension and congestive heart failure pre-clinical models. So at the end of the Phase 1 study, we should have a very good idea of the target dose and dosing interval.

In just a few months we plan to start our Phase 1B right heart cath study, in patients with Group 2 PH looking at hemodynamic effects. In the 2nd half of the year, we plan to start a placebo controlled six-month study in Group 2 PH patients with HFpEF. We've completed dosing of the first 3 cohorts of our Phase 1 study as outlined on the right-hand part of this slide and we've started dosing the next two cohorts.

To date, TX45 has been well tolerated with minimal adverse effects and no drug related SAEs. Based on 42-day data from the 0.3 mg/kg cohort and 14-day data from the 1 mg/kg and 150 mg cohort, the drug appears to be drug is well behaved with low variability and dose proportional PK. We've also seen a 38% increase in RBF at the lowest dose which is in line with what has been reported with serelaxin and which meets our Go Criteria

Shown here on the left is the preliminary PK from the 0.3 mg /kg IV cohort. Six patients were dosed with drug, but you can only 4 patients because the variability is so low that two of the patient data lines are not visible. You'll also note the curves start to become linear after day 14.

One the right is Day 2 and Day 8 renal blood flow data from the same cohort demonstrating that the increase in renal blood flow is maintained through day 8 of dosing. Longer term renal blood flow measurements are pending.

Based on available data, a PK model was constructed which suggests that with monthly dosings of 150 mg SC the trough levels exceed the levels predicted to provide max effects on renal blood flow. Given that we can go up to 300mg monthly with a single shot we anticipate monthly dosing.

There are 2 major biopharma companies who are also attempting to develop longer half life forms of relaxin. This is validation of the substantial market opportunity for drugs that effectively tackle this mechanism and speaks to the potential strategic value of our program if we can confirm that we have the best-in-class molecule.

In summary, I want to close by reiterating how strongly I believe that the combination of our assets, our expertise, and our capital will enable us to develop transformative biologic therapies for patients and create substantial value for our shareholders over the coming years.

- Our lead asset is ideally matched mechanistically to address the treatment of Group 2 PH in HFpEF, a disease without approved therapies
- Our platform has the potential to consistently generate compelling pipeline candidates across therapeutic areas
- Our team is unmatched

Thank you very much.

Operator:

This concludes the AVROBIO and Tectonic call today. A recording of today's call will be available for playback, and more details can be found on the AVROBIO's website, avrobio.com later today. Thank you for joining. You may now disconnect.

Participants in the Solicitation

AVROBIO, Tectonic, and their respective directors and certain of their executive officers may be considered participants in the solicitation of proxies from AVROBIO's shareholders with respect to the proposed merger transaction under the rules of the U.S. Securities and Exchange Commission (the "SEC"). Information about the directors and executive officers of AVROBIO is set forth in its Annual Report on Form 10-K for the year ended December 31, 2022, which was filed with the SEC on March 23, 2023, subsequent Quarterly Reports on Form 10-Q and other documents that may be filed from time to time with the SEC. Additional information regarding the persons who may be deemed participants in the proxy solicitations and a description of their direct and indirect interests, by security holdings or otherwise, will also be included in a registration statement filed on Form S-4 that will contain a proxy statement (and prospectus and other relevant materials) to be filed with the SEC when they become available. You may obtain free copies of this document as described above.

No Offer or Solicitation

This communication does not constitute an offer to sell or the solicitation of an offer to buy any securities nor a solicitation of any vote or approval with respect to the proposed transaction or otherwise. No offering of securities shall be made except by means of a prospectus meeting the requirements of Section 10 of the U S. Securities Act of 1933, as amended, and otherwise in accordance with applicable law.

Additional Information and Where to Find It

This communication relates to the proposed merger transaction involving AVROBIO and Tectonic (the "Merger") and may be deemed to be solicitation material in respect of the proposed Merger. In connection with the proposed Merger, AVROBIO will file relevant materials with the SEC, including a registration statement on Form S-4 (the "Form S-4") that will contain a proxy statement (the "Proxy Statement") and prospectus. This communication is not a substitute for the Form S-4, the Proxy Statement or for any other document that AVROBIO may file with the SEC and/or send to AVROBIO's shareholders in connection with the proposed Merger. BEFORE MAKING ANY VOTING DECISION, INVESTORS AND SECURITY HOLDERS OF AVROBIO ARE URGED TO READ THE FORM S-4, THE PROXY STATEMENT AND OTHER DOCUMENTS FILED WITH THE SEC CAREFULLY AND IN THEIR ENTIRETY WHEN THEY BECOME AVAILABLE BECAUSE THEY WILL CONTAIN IMPORTANT INFORMATION ABOUT AVROBIO, THE PROPOSED MERGER AND RELATED MATTERS.

Investors and security holders will be able to obtain free copies of the Form S-4, the Proxy Statement and other documents filed by AVROBIO with the SEC through the website maintained by the SEC at http://www.sec.gov. Copies of the documents filed by AVROBIO with the SEC will also be available free of charge on AVROBIO's website at www.avrobio.com, or by contacting AVROBIO's Investor Relations at https://investors.avrobio.com/.

Forward-Looking Statements

This communication contains "forward-looking statements" within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995, including but not limited to, express or implied statements regarding the structure, timing and completion of the Merger; the combined company's listing on Nasdaq after the closing of the proposed Merger (the "Closing"); expectations regarding the ownership structure of the combined company; the anticipated timing of the Closing; the expected executive officers and directors of the combined company; expectations regarding the structure, timing and completion of the private placement financing, including investment amounts from investors, timing of closing, expected proceeds and impact on ownership structure; each company's and the combined company's expected cash position at the Closing and cash runway of the combined company following the Merger and private financing; the future operations of the combined company, including commercialization activities, timing of launch, buildout of commercial infrastructure; the nature, strategy and focus of the combined company; the development and commercial potential and potential benefits of any product

candidates of the combined company; the location of the combined company's corporate headquarters; anticipated clinical drug development activities and related timelines; and other statements that are not historical fact. All statements other than statements of historical fact contained in this communication are forward-looking statements. These forward-looking statements are made as of the date they were first issued, and were based on the then-current expectations, estimates, forecasts, and projections, as well as the beliefs and assumptions of management. There can be no assurance that future developments affecting AVROBIO, Tectonic, the Merger or the private placement financing will be those that have been anticipated.

Forward-looking statements are subject to a number of risks and uncertainties, many of which involve factors or circumstances that are beyond AVROBIO's control. AVROBIO's actual results could differ materially from those stated or implied in forward-looking statements due to a number of factors, including but not limited to (i) the risk that the conditions to the Closing are not satisfied, including the failure to timely obtain stockholder approval for the transaction, if at all; (ii) uncertainties as to the timing of the consummation of the proposed Merger and the ability of each of AVROBIO and Tectonic to consummate the proposed Merger; (iii) risks related to AVROBIO's ability to manage its operating expenses and its expenses associated with the proposed Merger pending the Closing; (iv) risks related to the failure or delay in obtaining any required consents necessary to consummate the proposed Merger; (v) the risk that as a result of adjustments to the exchange ratio, AVROBIO stockholders and Tectonic stockholders could own more or less of the combined company than is currently anticipated; (vi) risks related to the market price of AVROBIO's common stock relative to the value suggested by the exchange ratio; (vii) unexpected costs, charges or expenses resulting from the transaction; (viii) potential adverse reactions or changes to business relationships resulting from the announcement or completion of the proposed Merger; (ix) the uncertainties associated with Tectonic's product candidates, as well as risks associated with the clinical development and regulatory approval of product candidates, including potential delays in the completion of clinical trials; (x) risks related to the inability of the combined company to obtain sufficient additional capital to continue to advance these or other product candidates; (xi) uncertainties in obtaining successful clinical results for product candidates and unexpected costs that may result therefrom; (xii) risks related to the failure to realize any value from product candidates currently being developed and anticipated to be developed in light of inherent risks and difficulties involved in successfully bringing product candidates to market; (xiii) risks associated with the possible failure to realize certain anticipated benefits of the proposed Merger, including with respect to future financial and operating results; and (xiv) the risk that the private placement financing is not consummated upon the Closing. Actual results and the timing of events could differ materially from those anticipated in such forward-looking statements as a result of these risks and uncertainties. These and other risks and uncertainties are more fully described in periodic filings with the SEC, including the factors described in the section titled "Risk Factors" in AVROBIO's Annual Report on Form 10-K for the year ended December 31, 2022, which was filed with the SEC on March 23, 2023, subsequent Quarterly Reports on Form 10-Q filed with the SEC, and in other filings that AVROBIO makes and will make with the SEC in connection with the proposed Merger, including the Proxy Statement described below under "Additional Information and Where to Find It." You should not place undue reliance on these forward-looking statements, which are made only as of the date hereof or as of the dates indicated in the forward-looking statements. AVROBIO expressly disclaims any obligation or undertaking to release publicly any updates or revisions to any forward-looking statements contained herein to reflect any change in its expectations with regard thereto or any change in events, conditions or circumstances on which any such statements are based. This communication does not purport to summarize all of the conditions, risks and other attributes of an investment in AVROBIO or Tectonic.