Vertex Pharmaceuticals Incorporated

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Event Participants

Analysts 1
Salveen Richter

Executives 2
Reshma Kewalramani, Stuart Arbuckle

Salveen Richter Analyst

Great. Good afternoon, everyone. Thanks for joining us. I'm Salveen Richter, I cover the biotechnology sector at Goldman Sachs, and we're really pleased to have Vertex team with us today. So we have Reshma Kewalramani, who's the CEO and the President, as well as Stuart Arbuckle, who's the Chief Operating Officer of the company.

Thank you both for joining us.

Salveen Richter Analyst

To start here, maybe we could give a snapshot of the business, where it stands today, what we should anticipate in terms of strategy, commercial execution and the pipeline as we look towards the second half of the year and beyond?

Reshma Kewalramani Executive

Sounds great. Well, first of all, it's very nice to see all of you in person as we are still all getting back to in-person meetings. Let me say a few words about where we are at the highest levels, and I'll ask Stuart to comment on where we are in terms of the CF franchise. The first thing to say is that we have had a really great start to this year. And as we have been talking about for the last 18 months, we're at an inflection point for the company.

The CF franchise which Stuart will talk about, has been going exceptionally well as we reach more and more patients with our medicines. Next in line for CF is the vanzacaftor triple combination, let's call it, the next-generation CFTR modulator regimen, which we expect towards the end of this year, beginning of next. And also in CF, we're expecting to see results

from our mRNA program with our partners at Moderna in the next year, shall we say. That program, VX-522, is in its Phase I/II study in the last 10% of patients with cystic fibrosis. Those are those patients who make no CFTR protein.

On the R&D pipeline side, we now have 8 disease areas in the clinic, 5 of which are beyond Phase II, and we'll certainly have time to talk about all of those. But maybe the important point to make is we are now on the brink of launching 5 disease areas in the next 5 years. So that is sickle cell disease and beta thalassemia with exa-cel as well as vanzacaftor in CF and VX-548 in acute pain. And for the fifth one, it's a bit of dealer's choice. You can think about 548 in neuropathic pain, the type 1 diabetes program, AATD, the inaxaplin program in kidney disease as some examples for what that -- what could round out those 5 and 5.

And on the finance side, our balance sheet, as you know, is very strong and we feel really good about how we've invested our capital in terms of investing in innovation, something you can expect to continue with modest share buybacks that we have also implemented over the last 18, 24 months. Stuart, I'll turn it over to you for a little bit more color on CF.

Stuart Arbuckle Executive

Yes. Obviously, CF as a franchise, has delivered incredible growth for the company over the last decade. As we entered 2023, we updated our estimates for the epidemiology of cystic fibrosis to 88,000 people living with cystic fibrosis in North America, Europe and Australasia. And of those 88,000, about 20,000 or so, as we entered the year, were not being treated with one of our CFTR modulators. They asked the question why?

Really, it's 1 of 4 reasons. The first one is that we are regulatorily approved. We also have reimbursement, but we're just early in the launch curve. So I think of countries like Australia and New Zealand. Secondly, there are a diminishingly small number of countries where we don't yet have reimbursement, which we're continuing to work on.

And thirdly, we're continuing to expand our labels to younger and younger patient groups in the U.S. We now have approval for TRIKAFTA down to 2- to 5-year-olds; ORKAMBI to 2-year-olds and KALYDECO now amazingly down to children between 1 month and 4 months of age, and we are continuing to get those label expansions around the globe. So those are 3 reasons why we see continued growth in cystic fibrosis with our existing medicines. The last group of patients in that 20,000 is about 6,000 or so patients around the world who've discontinued one of our CFTR modulators. We think they're going to be great candidates for vanzacaftor, our next-generation triple combination which, as Reshma said, is deep into its Phase III program and we expect to see the results end of this year, beginning of next.

The last area of growth for our cystic fibrosis franchise is getting to those 7%, about 5,000 or so patients who won't respond to our CFTR modulators because they produce no CFTR protein at all. They are going to be good candidates for our mRNA therapy, which has just begun its SAD program, which is the VX-522 program in partnership with mRNA. So we've seen incredible growth in CF over the last decade. We expect to see continued growth for our CF franchise for many years to come.

Salveen Richter Analyst

Maybe diving into vanzacaftor here. So we're going to see data, I believe, by year-end. What do you need to see to know that this is -- or why do you believe that this is superior to what you're seeing with TRIKAFTA and where do you think this will be then positioned within the marketplace?

Reshma Kewalramani Executive

Yes. Let's tag team that one. Let me tell you why we believe that vanzacaftor has the chance to be even better than TRIKAFTA. And I know that's a tall order, and that's a big statement. There are really 2 lines of evidence that make us think that, that is possible.

The first is our HBE assays. These are the human bronchial epithelial cell assays that have been predictive not only qualitatively, but quantitatively so for KALYDECO, ORKAMBI, SYMDEKO, TRIKAFTA and the other medicines that we advanced through late stages of development but chose not to commercialize because we selected TRIKAFTA. Those HBE assays tell us that the vanzacaftor triple is even better than TRIKAFTA. So that's one line of evidence. And the second line of evidence comes from our Phase II program.

We haven't studied every combination that you need to, to make direct comparisons. But if you look at the totality of our Phase II data, in het/min patients or the FMF patients, as they're called, or the FF patients and you look across those, some studies which had a TRIKAFTA arm, the vanza triple is better than TRIKAFTA consistently so in terms of sweat chloride, and that's important because that's the direct readout of CFTR function. In certain studies, you can even see hints of it in terms of ppFEV1, but that's harder because the sample size in Phase II is small and the variability of ppFEV1 is high. But those are really the 2 lines of evidence that give us confidence that vanzacaftor has the potential to be even better than TRIKAFTA. Stuart, a little bit on commercial potential?

Stuart Arbuckle Executive

Yes. From a commercial potential, there's really kind of 2 separate populations. There's those patients who were already established on a CFTR modulator, the vast majority of which now are on TRIKAFTA. If we have a product that has the clinical profile that Reshma has just described, there is a sort of upgrade even on TRIKAFTA and it's also once a day which is going to be more convenient for the patients. We think there's going to be a lot of interest from patients and physicians to switch from TRIKAFTA to vanzacaftor.

And then as importantly is that discontinued population. These are patients who've already demonstrated they would like to be on a CFTR modulator, but for a variety of reasons, have not been able to stay on a CFTR modulator. As I said, there's about 6,000 of those patients today. And we think they're going to be very good candidates for vanzacaftor because it will be a new potential treatment option for them. So we think it's going to have great potential in both of those segments of the market.

Salveen Richter Analyst

When we think about the future of your portfolio in the context of competition, are the assays at this point just providing this -- I guess, this level of understanding as well as allowing you to get -- given your portfolio these sweat chloride levels that it's going to be hard for any

competitor to kind of step in and show superiority over. I mean, how should we think about that?

Reshma Kewalramani Executive

Yes. The assays, these HBE assays are certainly one of the absolute keys to our success, but it's not the only thing. So when you think about cystic fibrosis today, it is a world of triple, and the standard of care is TRIKAFTA with a fairly significant improvement in ppFEV1 in the clinical trial. That's the regulatory enabling end point, a really good-looking benefit-risk profile. And if you are going to compete with TRIKAFTA, you have to go head-to-head versus TRIKAFTA, which, as we've already discussed, is a tall order.

Now if you work backwards from there, if you say, okay, understood that in the clinical trial scenario, you have to go head-to-head versus TRIKAFTA, you have to be able to compete there, how do you do that? Well, first, it's a world of triples. To actually get 2 correctors and a potentiator that work together and have the right drug-like properties, that's a tough, tough thing to do. Then there is the assays where you can study these in a high-throughput fashion, so you can pick the right combination to bring into the clinic. That's a hard thing to do.

And the last thing, I would say, is for patients who are on CFTR modulators, as Stuart expressed, we're now down to age 2-plus in the U.S. with TRIKAFTA. These are patients who are now taking medicines in the instance of KALYDECO from the age of 1 month; in the case of TRIKAFTA since the age of 2. It's very, very difficult to believe that you could simply come in with a new medicine and have all of these patients move over. The way you do the drug development in this disease state is you start at 12-plus.

So all the 1-month-olds and the 2-year-olds and the 5-year-olds, they're going to be on the medicines like TRIKAFTA or vanza and moving all of those patients to another medicine, that's a tall order. And the HBE assays are absolutely critical, but there's all of this additional context to be had as well.

Salveen Richter Analyst

Okay. And when you look at the mRNA partner program you have with Moderna, what would proof-of-concept look like there when you finally see that data set in those ineligible patients?

Reshma Kewalramani Executive

Yes. So for the last 10%, who simply don't make protein, what we're doing is a Phase I/II study, so it's already in CF patients. The readout is not going to be sweat chloride because this is a nebulized therapy. So it's direct delivery to the lung. We expect to see changes in ppFEV1 very similar to what we've done with our CFTR modulators.

I expect that, that's going to happen when we get to the MAD portion of the study. We're only in the single ascending dose portion now. And I expect that the readout will be changes in ppFEV1 very, very similar to what you've seen across the CFTR modulator portfolio.

Salveen Richter Analyst

Great. So maybe switching to an upcoming potential launch by year-end. So exa-cel which is your CRISPR-gene editing or edited product for beta thal and sickle cell. You've recently gotten your PDUFA dates. Do you expect a panel here as you look to both beta thal and sickle cell?

Reshma Kewalramani Executive

Yes. Whether -- we are thrilled to have the filings accepted. And now what this means is in Europe, in the U.K. and in the U.S., we are under active review for both sickle cell disease and beta thalassemia. That is a huge milestone in science and medicine.

It's the first CRISPR-Cas9 based therapeutic to get to this point. Whether or not we have an AdCom in the U.S. is the discretion of the FDA, and they don't have to tell us until they're ready to do so. If you ask me whether I thought it was more likely or less likely, I would say, it is more likely that we have an AdCom simply because of the novel nature of this therapeutic, it is the first CRISPR-Cas9 base treatment. But we don't get to find out until they're ready to say.

Salveen Richter Analyst

And you're going to have authorized treatment centers ready to go at launch and then you'll probably add some over time. Help us understand how many will be ready to go globally and what proportion of these patients will be addressed through these centers?

Reshma Kewalramani Executive

Stuart?

Stuart Arbuckle Executive

Yes. So let's start with the patients first. So there's about 150,000 patients with sickle cell and thalassemia between the U.S. and the EU. For exa-cel and it's kind of current iteration.

So with the current busulfan based conditioning regimen, we think about 32,000 or so of those patients are likely to be thought of as good candidates given the severity of their disease. They're going to be patients like we've seen in our clinical trials. They're having multiple transfusions up to 1 every 3 or 4 weeks, and in sickle cell disease, they're having multiple of these vaso-occlusive crisis, these very severe pain crisis, which lead to them being hospitalized. So about 32,000, about 25,000 sickle cells, 7,000 thalassemia. So when you think about the concentration of those patients in the United States, about 90% of those patients are concentrated in 25 states.

And we've said that we are planning to have 50 authorized treatment centers up and running approximately by the time that we launch, obviously largely concentrated in and around those states where the majority of patients are. In Europe, over 75% of patients are concentrated in the U.K., Germany, France and in particular, for thalassemia in Italy. And we said that we're targeting about 25 treatment centers in total across those 4 countries. We've been having dialogue with those treatment centers over the last few months, a lot of enthusiasm to get involved in exa-cel. I'm anticipating we'll have close to those numbers, if not exactly those numbers by the time we get our approvals.

Salveen Richter Analyst

And there's always been a debate about whether there truly is demand here for therapy or gene therapy or gene editing asset for this population. What gives you the confidence that these patients are willing to get on therapy?

Reshma Kewalramani Executive

I'll let Stuart go into the details, but the one thing that Stuart has taught me is how quickly a study enrolls and the demand for a study is good, as Stuart says, biomarker for demand. And I'll tell you that both the sickle cell and the beta thalassemia studies were oversubscribed. Stuart?

Stuart Arbuckle Executive

Yes. No, I mean, I do think that is a great biomarker of the unmet need and how excited people are about your technology is how quickly your clinical trials ramp up and went incredibly quickly, which is fantastic. In addition to that, as you can imagine, we've done numerous rounds of research with physicians and patients as lately as EHA, where we presented new data, updated data on our exa-cel program. Enthusiasm across the physician community is high. And indeed, awareness and enthusiasm in the patient community is high.

And up to 25% of sickle cell in TDT patients now say that they think a genetic therapy might be the best approach for them. So we think there's going to be significant demand for exa-cel, and we're excited to get ready to launch it, approvals willing.

Salveen Richter Analyst

So bluebird has a gene therapy on the market right now. Maybe you could just speak to the learnings that you're seeing there, be it pricing, reimbursement, their version of these access centers and demand and how that applies to you globally, but also how you think that demand might be divided between the 2 assets?

Stuart Arbuckle Executive

Yes. So it's kind of a tale of 2 cities in some ways. So obviously, there's their experience in Europe where they decided after a few months to kind of pull out of the European market and withdraw their licenses. We've obviously sought to learn from their experience. I think we've learned a lot about what we need to do to be able to demonstrate the level of unmet need and the value of these types of technologies is one thing.

And that supports pricing. The other one is around payment models. And I think one of the experiences we've had in cystic fibrosis and also in our discussions about payment models for exa-cel is one size does not fit all and that different governments are going to want different types of payment models based on their individual situations and what their individual needs are. And I do think bluebird have been very public. That was one of their learnings that they had a kind of one-size-fits-all approach in taking their medicine to Europe from a payment model point of view, and that didn't seem to get much traction.

So that's one thing. In the U.S., I would say, our learnings from their experience with their

current therapy in TDT is very limited because they've really only been on the market for a brief period of time. So I'm not sure we can really glean many learnings from that. Certainly, from our own work in the U.S., we've had great conversations with payers, both commercial and importantly, government, in particular, Medicaid. The payer mix for exa-cel is going to be sort of 60-40 government, commercial.

And Medicaid is the primary payer. We've had great conversations with many, many states, including all 25 of the states where the vast majority of patients are. Those discussions have been very, very productive. They're very well aware of the unmet need in sickle cell disease, the fact that this is an underrepresented and underserved population and they're excited about the profile of exa-cel. And similarly, with commercial payers in the U.S., our conversations have been very productive with them, and we are trying to work with them to get as close as we possibly can to kind of getting day 1 access.

As soon as we're approved, we want to be able to try and get this medicine to patients in need.

Salveen Richter Analyst

Great. You're also looking at potentially a second launch via your acute pain program that's in Phase III and data is coming shortly. Help us understand what that optimal profile is that you're looking for, for this to be a success? And why you have confidence based on the Phase II data sets?

Reshma Kewalramani Executive

Yes. So I think it's going to be a race for what's next faster exa-cel in terms of launch because there's the vanza data that are going to come out end of this year, beginning of next, and it's the VX-548 acute pain data around the same time frame. But focusing on VX-548, so this is a NAV1.8 inhibitor. And both NAV1.7 and NAV1.8 have been called the Holy Grail of targets for the pain indication because these are genetically validated targets for which we can see people who have mutations can have a high tolerance for pain in the case of 1.7 and a constant sensation of pain in the case of a gain of function mutation in NAV1.8. We have studied this particular target for longer than we've studied cystic fibrosis at the same site right here in San Diego.

The predecessor molecule, VX-150 has already shown effectiveness in both acute pain and neuropathic pain. And it also showed effectiveness in musculoskeletal, let's call it, osteoarthritis body pains of that kind. We see acute pain and neuropathic pain as fully Vertexian in terms of developing and commercializing. And in terms of acute pain for VX-548, not only did the predecessor molecule demonstrate success, but VX-548 itself demonstrated success in abdominoplasty and in bunionectomy in Phase II. In the Phase II trial, we also had an opioid arm for context.

It wasn't there for statistical comparison, but you can see the numbers there. And the VX-548 molecule performed very well in terms of efficacy and also in terms of benefit/risk profile. For Phase III, we do expect the results tail end of this year, beginning of next. It's the same exact pain conditions, bunionectomy and abdominoplasty. It's the same dose, the high dose from Phase II.

It's the same treatment duration, 48 hours, and it's the same exact endpoint. So because of those similarities, we have high confidence in the Phase III program. Maybe the last thing to share is at the end of Phase II meeting with the regulators, we discussed what the program needs to look like in order to get the label that we seek. And the label we seek is not a post-bunionectomy or a post-abdominoplasty or even a postsurgical label, it's a broad label for acute pain. And so we have a third study in our Phase III program that's a single-arm, safety and effectiveness study that allows patients of all kinds of pain conditions, surgical and nonsurgical so that we can get this broad label.

Salveen Richter Analyst

And Stuart, you've talked about this being a $4 billion opportunity. What gets you there? And how are these hospitals and physicians incentivized to work with you to get access to this drug? Maybe just help us understand the logistics around that.

Stuart Arbuckle Executive

Sure. So it's a $4 billion market today. Acute pain despite 90% of the prescriptions being generic because there's been no innovation really in the pain market for literally decades, 90% of prescriptions are generic, and despite that, it's a $4 billion market today. So we don't need to get a very big share of that very big market at a branded price to have a multibillion-dollar asset on our hands, and that's what we believe we have. But why we think it's going to be successful, I'll give you a couple of reasons.

The first one is what is the level of unmet need. Because of the very, very well-known issues with opioids in acute pain, including their contribution to the opioid epidemic, just about every state, every hospital institution in the country has policies in place that restrict who can prescribe opioids, for whom, for how long and in what settings. What has that done? That has created a gap between prescription strength NSAIDs and people who are able to get access to opioids. It's created an incredible unmet need in the market for better pain treatment.

Then there's the profile. You go to somebody and say, we've got something that even is opioid like in terms of its efficacy, but without all of the baggage and including the addictive potential, is that something you're interested, overwhelmingly that is something that people are interested in. So then the challenge becomes what about pricing? You're going to price this as a branded medicine, and they can prescribe generic opioids in the hospital and that's going to be -- those are really, really cheap, and they're all going to do that to maximize. I think that is a true criticism.

Until you see some of the policy changes, which are being made, which I think are being made to provide a tailwind to the use of non-opioid pain medicines. I'll point you to something that was signed into law by the Biden administration in December of last year. It was called the NOPAIN Act. The NOPAIN Act understands that there is essentially a financial disincentive to prescribe a better branded medicine because of the DRGs that govern hospital reimbursement. And so the NOPAIN Act instructs CMS to provide an add-on payment in the outpatient and in the ambulatory surgical center setting specifically over and above the DRG to compensate people for non-opioid pain medicines.

I think that is the first of many such policy tailwinds that we will see instead of policy

headwinds, which opioids have faced for good reasons. I think we're going to see the kind of the winds change and provide policy tailwinds to try and encourage and/or remove the financial barriers to people using better, branded non-opioid pain medicines. So we're really excited about VX-548 in acute pain and hopefully subsequently in neuropathic pain as well.

Salveen Richter Analyst

And remind us where you stand right now with the neuropathic pain program, but also your outlook towards chronic pain?

Reshma Kewalramani Executive

Yes. So as Stuart outlined, we really see pain as 3 distinct categories: acute pain, neuropathic pain and then the musculoskeletal pain, osteoarthritis and such. The latter 2 often people call chronic pain. We divided in these a thirds because we see acute and neuropathic pain as fully Vertexian where we would commercialize ourselves. Just like with 548 in acute pain, 548 in neuropathic pain has the predecessor molecule, VX-150, which already demonstrated success in neuropathic pain.

For VX-548, we're currently in Phase II. The trial is well underway. I expect results towards the tail end of this year or beginning of next for neuropathic pain as well. And pending positive, fingers-crossed, results, the next steps from there would be to proceed on to Phase III. And again, there, we are looking for a broad peripheral neuropathic pain label.

Salveen Richter Analyst

Great. So you have a pretty deep pipeline beyond that, and we're going to get some data at the upcoming diabetes meeting from your naked cell, stem cell program, regenerative stem cell program in type 1 diabetes. Help us understand how we should think about where this program sits right now in your whole vertical from Semma and then the steps towards getting to address the whole population? And is the science ready at this point? Like it seems like it's an optimization question, but are we here?

Reshma Kewalramani Executive

Yes. When we talk about this inflection point for Vertex and '23 being such an important year, think about it, we are now at the point where in our CF program, we're down to 2-year-olds with TRIKAFTA. The next, next gen is going to deliver results any moment. We're already in CF patients with the mRNA program, the first-ever CRISPR-Cas9 program for sickle cell and beta thal has been accepted by the FDA. And by the way, we have our cell-based regenerative medicine for type 1 diabetes and those results are going to come out in the next week or so at ADA.

So it really is a really important and very special year for the company. On type 1 diabetes, in particular, we have 3 distinct programs under the type 1 diabetes umbrella. The first, as Salveen, you mentioned, is let's call it, the naked cell program. It's the cells alone and you need to use off-the-shelf immunosuppressive to protect the cells from the immune system. Those are the data that are going to be presented at ADA.

We have Part A, and we've already shared those data from the first 2 patients dosed at half

dose. So the first patient is insulin independent. That is a huge, huge accomplishment, and it's never been done in an allogeneic sense before. The second patient also derived benefit. And based on those 2 patients worth of data, we declared proof-of-concept.

We have completed Part A, that's those 2 patients. We've also completed Part B, which is full dose with a stagger. And now we're on to Part C, full dose without a stagger. At ADA, you'll see the Part A patients, so longer duration of follow-up. And you'll also see all the patients in Part B, so more patients.

The second program, the same exact cells where we've already shown proof-of-concept enveloped in a device, no immunosuppressions necessary. The CTA has cleared in Canada. The IND has cleared here and in the coming weeks and months, you should expect to see us dosing those patients. And the third program is those same exact cells that we make certain edits to so that those edits protect the cells from the immune system, they make them, let's call them, hypoimmune or immuno-evasive cells. That third program is still in the labs, in the research stage.

So that's what it looks like in terms of the program and where you can expect at ADA. And just to close on it in terms of, is the science ready? Absolutely, yes. We are actively dosing patients, and you'll see the results for yourself in a couple of weeks. Whether or not the best format for patients is cells alone, cells plus device, edit itself, we're going to have all of it to look at.

And depending on the patient population, they can make the right choice for the benefit risk profile that's best for them.

Salveen Richter Analyst

And with the third program, the edited program, how are you contrasting that with your ViaCyte acquisition and the programs you got through that?

Reshma Kewalramani Executive

Yes. So the -- once we saw the first 2 patient's data and we're able to declare proof-of-concept, once we had that leadership position, we wanted to make sure we covered the waterfront as it were. ViaCyte has a very similar goal that they were trying to achieve as we do, which is to cure type 1 diabetes. Their approach is slightly different. What we gained from the ViaCyte acquisition was people, tools, capabilities, manufacturing.

As an example, they had already -- ViaCyte had already worked with CRISPR on editing cells albeit with a different starting material. And through this acquisition, we get access to tools, technologies, IP, cell banks, but also the program that's already in the clinic using the ViaCyte cells and CRISPR edited cells, so we can explore those as well. You put it all together, and we feel really great about the position we now have, including access to already edited cells that are already in patients.

Salveen Richter Analyst

Great. And remind us, you have a few other programs in the pipeline that are in the clinic. When might we see data from those programs?

Reshma Kewalramani Executive

Right. So if we move to some of the other programs that are in the mid-stage, let's say, let's maybe go through those. VX-548 type 2 -- in peripheral neuropathic pain that should be end of this year, beginning of next. Inaxaplin previously known as VX-147 in a particular kind of genetically mediated kidney disease. It's a Phase II/III adaptive design program that also has a pathway to accelerated approval in the U.S.

that we've already agreed to with the agency. The part -- the Phase II part of the II/III program should also be ready sometime towards the end of this year. We've talked about the diabetes program, maybe a quick word on AATD. We have 2 programs running there. One is the first-generation program.

It's called VX-864. That is being run in a longer-term study, so we can assess both the liver and the functional AAT levels. And a next-generation program called VX-634, which we're running through Phase I. It's a more potent molecule. At some point next year, we'll have access to all of that data, so that we can make our appropriate decisions for next steps.

I should say for AATD, this disease is important to understand in that it has a liver manifestation and a lung manifestation of disease. And there's no other program out there that's targeting both simultaneously. That's why we think this is so important in terms of the Vertex approach.

Salveen Richter Analyst

Great. And maybe just a last question here. You have over $11 billion cash on your balance sheet. How are you thinking about capital allocation in terms of whether you would consider doing M&A at this point even though you have a deep pipeline or even dividends?

Reshma Kewalramani Executive

Yes. I am going to sound like a broken record on this. The primary use of our capital is going to be in innovation, both internal and external. And you've seen us do the kind of deals that just fit perfectly in our strategy. It's not a matter of size.

It's not a matter of whether it's cheap or not. It only matters whether it fits our strategy. You've seen us do modest share buybacks. You should expect that to continue. And as I look at my crystal ball, I don't see dividends in the near future.

Salveen Richter Analyst

Great. Well, perfect. Thank you so much, Reshma and Stuart. Appreciate it.

Stuart Arbuckle Executive

Welcome. Thank you.

Reshma Kewalramani Executive

Thank you so much.