SPARC/Sec/SE/2022-23/065 October 28, 2022

National Stock Exchange of India Ltd.,
Exchange Plaza, 5th Floor,
Plot No. C/1, G Block,
Bandra Kurla Complex,
Bandra (East), Mumbai – 400 051.

BSE Limited,
Market Operations Dept.
P. J. Towers,
Dalal Street,
Mumbai - 400 001.

Ref: Scrip Code: NSE: SPARC; BSE: 532872

Dear Sir/Madam,

Sub: Transcript of the Investor Presentation for Update on Clinical Programs and R&D Pipeline

Further to our letters dated September 29, 2022 and October 13, 2022 bearing reference nos. SPARC/Sec/SE/2022-23/056 and SPARC/Sec/SE/2022-23/060 respectively, please note that the Investor Presentation was made as per the scheduled time. Pursuant to Regulation 30 of the SEBI (Listing Obligations and Disclosure Requirements) Regulations, 2015, we hereby share the copy of the Transcript of the said Investor Presentation. The same will also be made available on the website of the Company.

This is for your information and dissemination.

Yours faithfully,

For **Sun Pharma Advanced Research Company Ltd.**

DINESH POOSRAJ LAHOTI

Digitally signed by DINESH POOSRAJ LAHOTI
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Dinesh Lahoti
Company Secretary and Compliance Officer
ICSI Membership No. A22471

Encl: As above

"Sun Pharma Advanced Research Company Ltd. (SPARC) Update on Clinical Programs and R&D Pipeline Conference Call"

October 13, 2022

MANAGEMENT: **MR. ANIL RAGHAVAN – CHIEF EXECUTIVE OFFICER**
DR. SIU-LONG YAO – HEAD, CLINICAL DEVELOPMENT
DR. NITIN DAMLE – CHIEF INNOVATION OFFICER
DR. VIKRAM RAMANATHAN – HEAD, TRANSLATIONAL DEVELOPMENT
MR. CHETAN RAJPARA – CHIEF FINANCIAL OFFICER
MR. JAYDEEP ISSRANI – SENIOR GENERAL MANAGER, BUSINESS DEVELOPMENT

Moderator: Ladies and gentlemen, good day, and welcome to SPARC's Update on Clinical Programs and R&D Pipeline. As a reminder, all participant lines will be in the listen-only mode, and there will be an opportunity for you to ask questions after the presentation concludes. Should you need assistance during the conference call, please signal an operator by pressing '*' then '0' on your touchtone phone. Please note that this conference is being recorded. I now hand the conference over to Mr. Jaydeep Issrani. Thank you, and over to you, sir.

Jaydeep Issrani: Good evening, ladies and gentlemen. I am Jaydeep Issrani. On behalf of SPARC, I welcome you all to SPARC's Annual Update on Clinical Programs and R&D Pipeline.

We have our CEO, Mr. Anil Raghavan and members of the SPARC's senior team on the call today.

I hope that you have received the presentation that was sent out some time ago. The slides are also available on our website, www.sparc.life. The presentation will be similar to what we have been following in the previous versions, i.e. will walk you through the presentation, and then open the call for questions.

Before I hand it over, I want to mention and remind you all that our discussion today includes forward-looking statements that are subject to risks associated with our business that may cause actual results to differ from those projected in the presentation.

I will now hand over the call to our CEO, Mr. Anil Raghavan. Over to you, Anil.

Anil Raghavan: Thank you, Jaydeep, for the introduction and the opening comments. Good morning and good afternoon, everybody, if you're joining from the US or Europe. A very warm welcome to the 12th edition of our Pipeline to engage and make this such a special event on our calendar. So, welcome back to this important call and thank you for your time.

As Jaydeep mentioned, we have the management team on the call today. In the interest of time though we don't plan to do detailed introductions.

Slide #3 has the agenda for the day. We will start with brief comments on our strategy and some important program updates, including a brief snapshot of the portfolio

performance and the listing of upcoming catalysts. Subsequently, we will dedicate most of our presentation today to provide additional color on six opportunities with four NCE clinical programs plus one important biologics platform with IND visibility and a new first-in-class NCE asset in dermatology.

Dr. Siu-Long Yao, who heads our clinical development, will cover the clinical projects, Dr. Nitin Damle, our Chief Innovation Officer, will provide an update on the biologics platform and the MUC-1 ADC, and Dr. Vikram Ramanathan, who heads our Translational Development, will speak to us about an interesting collaboration with Johns Hopkins University on a potentially first-in-class intervention in alopecia areata which is the autoimmune form of hair loss. We will conclude the presentation with brief comments from our CFO, Chetan Rajpara, on our financial performance and cash situation.

So, with that, let's start. I'm going to start with Slide #4, which is a nice set of numbers summarizing the state of our business. This is meant more of a scorecard reflecting the current outcomes of our journey, navigating different shades of risk starting with our 505(b)(2) days to building an organization which can now take real bets on new targets and complex modalities. Please allow me to take a few minutes to go over these numbers.

On the commercial or close to market end of our portfolio, we have five assets. Elepsia, which is a high dose levetiracetam pill is commercialized through a US CNS specialty company called Tripoint Therapeutics. And Xelpros, which is our BAK-free formulation of a first-line glaucoma drug, latanoprost, is with Sun Pharma in the US.

Two more NDAs are under review and another one is in planning. First one, phenobarbital benzyl alcohol - and propylene glycol-free formulation is under review with FDA with this PDUFA date coming up next month.

And the second one, brimonidine once-a-day eye drop goes by the project name PDP-716 was submitted recently. PDP-716 and ophthalmic steroid SDN-037, are both licensed to Visiox Pharma as we disclosed earlier.

These programs which are either in the market or have had successful clinical outcomes in the late stage trials, go a long way in validating the SPARC model. In

addition to the cash, these experiences did offer substantial learning in terms of building competencies to conceive and convert full development program.

The second pillar on the slide shows six ongoing clinical trials leveraging three NCEs. These assets offer really significant commercial opportunities, particularly vodobatinib in Parkinson's Disease and more broadly across other neurodegenerative conditions. Vibozilimod in dermatology is in a spectrum of autoimmune disorders.

The primary development space for the CNS leg of vodobatinib centers on a bunch of diseases driven by the neuronal toxicity of aggregated alpha synuclein. Parkinson's disease is the largest slice of that pie in terms of prevalence. Lewy Body Dementia or LBD, and Multi System Atrophy or MSA, are the other two components of the spectrum. LBD is quite a substantial disease in terms of the total disease burden, and while MSA is not a large patient pool, the unmet need is quite high. It doesn't have an appropriate standard of care currently.

Vodobatinib can offer a potentially disease modifying intervention across alpha synucleinopathies once we establish a clinical proof-of-concept, which is very consequential for patients across the globe with these debilitating conditions.

On vibozilimod, the next generation of S1PR1 agonist are becoming class alternative to JAK inhibitors in dermatology after the recent FDA safety warning for the JAK as a class. That was quite an important development, and may be one of the primary drivers of Pfizer buying Arena Pharmaceuticals. We are among the few players developing S1Ps in dermatology with Arena and BMS being the other two.

On the third column is on our preclinical effort. We intend to bring two more projects to clinic in the next 18 months. Alopecia Areata program later this year if everything goes on plan and MUC-1 ADC next year. But more importantly, we have seen our operating capabilities and external collaboration mature to a point where we feel comfortable taking early stage risk.

I want to leave you with the following thoughts on this slide. SPARC has validated its operating model with a significant number of assets across commercial, late, and mid-state clinical development, and early stage programs. So, in that sense, SPARC offers one of the more validated translational bridges to navigate the opportunity to innovate from India for patients across the world. And more importantly, we are

approaching some meaningful catalysts in the next 12-to-18-months for our stock and that's what we intend to cover in the rest of the deck.

I am going to use the next couple of slides to give some additional color on a few points I just made.

Slide #5 please. On capital efficiency, as a matter of strategy, we went after lower incremental innovation initially. A positive outcome from that approach was the non-dilutive capital it has generated to fund more complex science and high-value opportunities we built in the last few years. As you can see in this graph here, SPARC has spent upwards of $500 million up to the end of FY'22 running our operations in the last 15-years and building what we have today.

Fresh equity rights issues in 2012 and 2016 contributed around $79 million in total, while preferential issues brought in the rest. In this period, we invested around $277 million from our internal revenue accruals.

Two things are worth noting besides the substantial non-dilutive cash flows used to create the model and the portfolio build-out. These cash flows are primarily coming from our 505(b)(2) products plus some platform licensing transactions and some service revenues in addition to one upfront payment on vibozilimod. The economics of all the NCE or NBE programs are fully retained within SPARC, with the exception of vibozilimod. So, the non-dilutive funding mentioned in this slide happened without diluting the more promising programs.

Secondly, our journey has been marked by substantial promoter commitment. The lion's share of fresh equity infusion mentioned here in this slide for SPARC has come from our promoter group, and that's a huge show of confidence on the SPARC story and a demonstration of commitment to the vision.

Now, Slide #6. We've been talking about our portfolio pivot from incremental to more riskier NCE opportunities in the past few years, with the rationale and nature of that transformation many times. Early on that pivot, we focused on validated targets like BCR-ABL in chronic myelogenous leukemia and S1PR1 agonism in autoimmunity. But we've now moved to the next phase in that evolution with increased confidence in taking early-stage risk position in novel biology. These numbers on the chart here are worth taking note of. Almost 60% of our portfolio now has first-in-class potential. The

composition of the portfolio from a modality mix standpoint continues to evolve. Almost 1/5 of our programs now are outside of the traditional, straightforward new chemistry, and that component is growing faster. They either represent complex modalities like antibody drug conjugates, or other conjugated entities of biologics. This transformation wouldn't have been possible without our robust external innovation initiative. Most of our ideas involve substantial partnering components either as traditional development collaborations or for sourcing important development competencies, which are difficult to find or build in India. In this process, our own identity has shifted quite a bit towards being a translational engine which looks to access exciting science and differentiating competencies from wherever we can find them from groups across the world.

And secondly, the confidence in our portfolio decision-making has evolved quite a bit. Some of that is driven by the disciplined portfolio review process that we have followed to take Go/No-Go decisions, and some of it is driven by the changing emphasis on certain parts of the process itself like the identification of key experiments which can give us early signals of proof-of-mechanism, or early signs of potential safety and efficacy, or a conscious decision to invest substantially in largish reproducible global trials in the Phase-II stage itself.

While we are still very much a work in progress, it's important to say that we've taken deliberate steps to mitigate the risk as much as possible, while embracing early stage risk as a matter of business reality that we're dealing with. A large part of that process is our relationship with external advisors, and the space that we provide to them to input into our decision-making process.

So, with that, let's go to Slide #7 for a few examples. I want to spend some time on three programs here, vodobatinib in PD, ADC using antibodies against MUC-1 alpha/beta junction, and SCD-153 for a novel immunological target to treat alopecia areata.

Vodobatinib is probably the first serious effort to track the oxidative stress response in neurodegenerative diseases. The toxic cascade of events initiated by Abl-mediated oxidative stress response is affected through a complex web of interrelated events involving on one side, the aggregation of intercellular alpha synuclein and on the other side compromising of multiple protein clearance pathway.

Our decision to move to a clinical proof-of-concept study was driven by critical pieces of evidence for the impact of disrupting this cascade with a highly potent, but super-selective c-Abl inhibitor gathered through several important experiments done at some of the best labs in this area globally, like Dr. Ted Dawson at Johns Hopkins University, or the Ann Romney Neurology Institute at Brigham and Women's Hospital, or Atuka, Inc. in Canada, in addition to of course our own internal experiments. So, the totality of evidence coming from these experiments gave us the confidence to move to clinic. Our initial focus in the clinical study was to establish an appropriate clinical dose, meeting the brain exposure targets indicated by the preclinical program and confirm the safety of the proposed doses. Once we achieved that, we moved to a clinical proof-of-concept study, that's large and global enough to provide a comprehensive and reproducible proof-of-concept for the mechanism. So, that's what I was talking about de-risking early-stage risk taking through a deliberate translational framework.

Moving on to the second project here on the slide, here, again, you can see several parts of the same strategy at play. MUC-1 has been a high interest cancer target for ADC and other tumor-targeting programs because of its high tumor-specific expression. Our strategy was built on four antibodies from Biomodifying against a set of unique α/β junctional epitopes looks to avoid the trap of peripheral floating MUC-1 which led to failure of earlier attempts. So, that gives us the differentiated opportunity to deliver high potency cytotoxic payloads and other targeted agents using the ADC construct.

On a separate note, we are very excited about ADCs as a modality, which has clearly become mainstream on the back of clinical successes of products like Trodelvy and Enhertu recently. These successes, and several others have demonstrated the stability and scalability of clinical system, and safety windows for the commonly deployed payload. In fact, substantial activity using clinical ADC framework, as well as this novel construct involving targeted agents or immunological intervention and MUC-1 program and other programs in preclinical mix, give us an opportunity for unique targeting in oncology.

And finally, on this slide on the right column SCD-153, which we are disclosing for the first time here. SCD-153 is being evaluated through an option-to-license agreement

with a highly accomplished group at Johns Hopkins and leveraging a compound with significant anti-inflammatory property.

As you will see later on in Vikram slides, we have developed substantial data and are in the process of establishing a topical formulation with appropriate safety margin. We are hopefully headed towards an IND in the early part of 2023.

In both these preclinical programs, you can see key elements of a model playing out in terms of an attempt to leverage novel biological insights, willingness to learn from external growth, and robust early translational work to set up clinical programs appropriately. We believe that's the right approach and offers the responsible path towards growing the portfolio with potentially attractive and clinically relevant assets.

So, let me now pivot to certain near-term consideration before I hand over the call for progress and that's cash runway on Slide #8. We have an expected spend of around $60 million in a year. Numbers for the outer years as you can imagine are estimates, but we are more or less in the ballpark here. In terms of cash flow, we expect warrant conversions of up to $93 million by end of December 2022. In addition, we will have certain milestones and royalties from existing and upcoming commercial products and technology licenses. Without taking into account royalties and milestones, we have a cash runway that will take us to the end of next financial year FY'24.

Slide #9 please. Now, let me spend few minutes on the slide to give you some ideas around Sezaby which is our benzyl alcohol-free phenobarbital formulations for treatment of neonatal seizures. Starting with a little more context before covering project status on the slide. The currently marketed products did not go through the current approval process requiring a clear demonstration of safety and efficacy as they're in medical practice before the current process was established. FDA formulated a plan to sunset such unapproved products if a company obtains an approval through the due process. The current policy therefore aims to remove all unapproved products from the market when such approvals are given on the basis of fresh evidence presented. The policy also offers certain market exclusivity benefits to incentivize companies to generate new data.

Phenobarbital injectable is an unapproved product and is primarily used for treating newborns experiencing seizure episodes. In addition, current unapproved phenobarbital formulations contain benzyl alcohol or propylene glycol, which many regulatory agencies across the globe removed because of the potential toxicity.

We filed a new drug application for phenobarbital in February 2022. The FDA has granted a priority review and pediatric rare disease designation, which if confirmed can offer seven years of market exclusivity. If approved, the SPARC product can become potentially the first approved phenobarbital, which can enjoy the associated market exclusivity, pending certain agency determinations subsequent to the approval. SPARC may be eligible for certain other incentives associated with it, depending on the outcome of the ongoing review. We are in advanced stages of evaluating our options for commercialization of this product in the US. The PDUFA data as I mentioned is in November 2022. We hope to become an exclusive provider of the first approved formulation of phenobarbital in the US. We're looking forward to working with an appropriate commercial partner to grow the franchise responsibly. We can come back to this in more detail if you have questions during our Q&A.

Now, let's spend a few minutes on Slide #10 to go over the upcoming catalysts for the company. The first column discusses the commercial or late stage clinical program. We had an excellent start for Elepsia with Tripoint, though, these are still in very early days. The early performance gives a significant confidence to scale up for commercial success for Elepsia.

Xelpros is our second product in this category, which is with Sun Pharma in the US. We are working very closely with the Sun team to continue to build the franchise.

In addition, we expect to see commercialization of Sezaby a bit later in this financial year and brimonidine or PDP-716 in the next financial year. We're still discussing the NDA submission for SDN-037 with Visiox.

So, on the commercial side, we have a few levers which we can use to generate additional cash from royalties and regulatory milestone payments in the short-to-medium term.

On the clinical side, vodobatinib CML program which is in a pivotal study is actively recruiting now, though slower than we would like, given the rare indication that we're pursuing. We are seeing excellent efficacy for protocol eligible patients in the study, Siu will go over this in a bit. We are looking to file NDA in 2024.

PROSEEK is our Phase-2 PD trial for vodobatinib and we have crossed the 70% recruitment mark a few weeks back, and we are pushing forward on all cylinders to complete the accrual by the end of this financial year. And that sets up an important data event in the coming financial year. Vodobatinib as you know is also in a single center study in Lewy Body Dementia at Georgetown University, which is expected to read out around the same time.

Vibozilimod is in two Phase-2 trials in atopic dermatitis and psoriasis. We're ramping up the site infrastructure significantly with additional sites in Europe and Latin America. Finally, SCO-120 is in Phase-1 dose escalation study in patients. We hope to get early efficacy signals as early as next year for the oral SERD program. We are carefully navigating the space though given the complex nature of the SERD field right now, and we will set up a Go/No-Go event for this program basis the quality of the efficacy signals we receive going into next year.

And on the preclinical side, I have spoken in detail about SBO-154, that's a MUC-I ADC and SCD-153 in alopecia areata, so I won’t go into more details on that, but both projects are on track to go into INDs by financial year '24.

Finally, we will continue to look for additional assets for partnering. We will look for exciting early science which synergizes with our current basket, but we will be conscious as it has the potential to float out clinical spend going into next year and even beyond. So, our interest would be primarily driven by strategic portfolio considerations and not opportunism.

Now, on to Slide #11, my last slide in the deck. This chart is the pipeline summary. I guess we've gone through most of this in a fair bit of detail. It offers indication, current development status plus next set of data milestones. And we can come back to this again for the Q&A.

So, with that, thanks again for your time today. I will now hand over the call to Dr. Siu-Long Yao who heads clinical development for additional detail on four clinical programs.

Dr. Siu-Long Yao: Thank you, Anil. Again, my name is Siu Yao and I oversee clinical development. The next set of slides go over SCO-088 for the treatment of chronic myelogenous leukemia. So, with SCO-088, we believe that we have a safe, effective option for the treatment of heavily pretreated, last-line patients. This Slide #14 summarizes the clinical development program for this drug. The program started with a healthy volunteer single ascending dose and food effect study in 40 subjects and then proceeded to a multiple ascending dose study in patients. Currently, we're in the midst of a pivotal study involving chronic phase, accelerated phase and blast phase CML. As noted in the first bullet in the lower right-hand part of the slide, this is last line therapy. So, patients are required to have disease that is refractory and/or intolerant to greater than or equal to three prior tyrosine kinase inhibitors, one of which must have been ponatinib. For this study, we have sites in the US, Belgium and there's a host of other sites listed on the slide and you can refer to it.

The next Slide #15 gives you some of the data we've seen from the multiple ascending dose study in patients. This slide shows the major cytogenetic response rates that we've observed, and breaks it down into the status of the entering patients at baseline on the top bar, and then what happened to them following treatment on the bottom bar. As you can see, 71% of patients came into the study with uncontrolled disease. Only 29% had a major cytogenetic response at baseline. Following treatment with vodobatinib however, there was a marked increase in the proportion of patients with a major cytogenetic response as you can see in the lower bar to over two-thirds of the patients had disease that was responding to treatment.

Next Slide #16 shows results shows major molecular response rates following treatment. Again, the top bar represents the status of patients at baseline just prior to enrollment and the bottom bar represents results observed following treatment with vodobatinib. You can see that there has been a major molecular response, which represents a three-log reduction in the number of cancer cells, has increased from 2.8% prior to treatment to 43% following treatment.

Finally, slide #17 here depicts a swimmer's plot that gives you a feel of what we're observing overall with vodobatinib. On the x-axis is duration of treatment, which is a measure of progression-free survival, and on the y-axis in each row are the results for individual patients. Patients are treated until they no longer respond and the point at which response stops is depicted by a dark circle. The duration depicted on the x-axis is substantial, or it can be substantial, with time ranging out to 65 months or almost five and a half years. A swimmer's plot is somewhat of an unequal assessment as it doesn't show the maximum benefit. Because people at the top of the graph have only experienced treatment for a short period of time. So, you don't know if they're going to continue to respond like some of those at the bottom of the graph. This was a dose escalation study so the doses at the bottom of the graph are generally lower than those at the top. Nonetheless, you can see that there have been some dramatic responses, with some patients at the lower doses benefitting for well over five years now.

Slide #18 goes over our current plans for the program. We'll be providing additional updates at the Goldman Conference in October in France, and at the Annual Meeting of the American Society of Hematology (ASH) in New Orleans in December. We're happy to say that our abstract for the ASH Meeting has been selected for an oral presentation for the third year in a row now, and results from the pivotal study are anticipated in fiscal year '24.

This slide transitions to the use of vodobatinib for neurodegenerative diseases. The mechanism of action here is relatively unique, I want to point that out and that we are trying to modify the disease course rather than just treat symptoms, like the existing therapies do.

Slide #20 is here to remind you about the underlying science supporting the role of Abl and neurodegenerative diseases, including Parkinson's disease. As noted in the bullets on the left, Abelson kinase is expressed in all parts of the brain and has a pivotal role in promoting neurodegeneration, specifically under conditions of toxic stress, Abl causes cells to die in order, for example, to rid the body of defective or malfunctioning cells.

Slide #21 goes over results from the preformed fibril model of Parkinson's disease in mice. So, let me walk you through this slide. In this model, toxic aggregates of synuclein, a putative basis of Parkinson's disease are injected into the brain to cause

Parkinson's disease in mice. The top two graphs, summarize results from mice turning around at the top of a pole on the left graph, and then descending the pole on the right graph. In each graph, time is on the y-axis, and different treatments are on the x-axis. The first two groups in each case or in each graph consists of control treatments, where disease has not been induced in the mice.

The third column in each case is the result you get when you induce disease in the mice. You can see that the amount of time required to turn on top of the pole and then descend the pole increases in those third columns, because the mics are unsteady and they had disease induced in them.

When you treat with a low or high dose of vodobatinib represented by the fourth and fifth columns respectively, the time for the mice to complete the task decreases, and it's essentially the same as the unaffected mice represented by the first two columns in each graph. Similar results occur when you look at the strength of grip of the mice as depicted in the two graphs in orange shading at the bottom of the slide.

On the y-axis in these graphs, you have grip strength, and going across the x-axis, you have the same treatment as before. Again, the first two columns represent unaffected mice, whereas the third column represents affected mice not given any treatment. Columns four and five in each graph represent the results from treatment with vodobatinib either a low or high dose. You can see that higher doses of vodobatinib are able to protect the mice from the insult of the preformed fibrils that cause Parkinson's disease in the mice.

So, next Slide #22 is from another model of Parkinson's disease, but in rats. Here, a gene encoding a mutant misfolded toxic version of alpha synuclein, originally derived from a person with Parkinson's disease is expressed in the right half of the brain. The left side of the brain is left alone as a control to tell you what things would look like in an unaffected brain, that's not subject to treatment with a toxic disease inducing alpha synuclein. So, following treatments the whole brain is removed and evaluated for cell death by using a radioactive probe. Since in each case, the left side of the brain is normal, you want to compare the right side which has been treated to cause disease with the left normal side each time when you look at the graph. The first group of left and right is a control, just to make sure that the virus used to deliver the alpha synuclein isn't itself causing some type of unanticipated effect. You can see that the left and right columns there in the first group match so the delivery system is not

causing any real effect. The second group shows you what happens if you induce disease without any treatment. The left side is normal, and the right side, which has been treated with the toxic synuclein shows much fewer functional cells as represented by the lower bar or column.

Groups three, four and five represent increasing doses of vodobatinib that, again, you are comparing the right disease brain in each case to the normal left brain for each group. You can see that by the time you get to the high dose of vodobatinib shown in the rightmost group, the left and right brains are matching, suggesting that vodobatinib is essentially completely blocking the effects of the disease-inducing mutant alpha synuclein.

Now, we're on Slide #23. And this summarizes the study design of the ongoing PROSEEK clinical study, evaluating the effect of vodobatinib in Parkinson's disease. There are three arms here, consisting of placebo and a low and high dose of vodobatinib. The total sample size is 504 split between the three arms, and part one is the big study, whereas part two is an extension where all subjects received vodobatinib. As summarized in the bullet on the right there, there are currently 77 active sites and over 70% of the study has been enrolled.

Slide 24 summarizes progress with a Lewy Body Dementia study that we're collaborating on with an investigator at Georgetown University in the United States. Lewy Body Dementia is a disease that is thought to have a cause similar to Parkinson's disease. So, you can see as noted in the figure and bullets, this is 45 patient study primarily designed to evaluate safety, tolerability and biomarker results in this population and we're now about halfway through the study.

This slide just summarizes the main milestones for this program. Again, there's the Phase-2 PROSEEK study and the Lewy Body Dementia study, both of which are maturing in fiscal year 2024. I'm now going to transition to vibozilimod or a selective S1PR1 agonist, which we believe will be a safer alternative to JAK inhibitors for the treatment of dermatologic disorders.

Slide #27 and I'm going to walk you through some of the bullets here. This provides an overview of vibozilimod along with some context regarding the therapeutic area and existing molecules. So, overall, the goal of our program is to develop an oral standard of care agent for the treatment of dermatologic disorders. And as you may

know, the existing oral agents such as methotrexate or apremilast have limited efficacy and/or significant toxicity concerns, and they're generally unable to reach the efficacy seen with the injectable biologics. Our goal is to have a best-in-class sphingosine agonist with efficacy and safety that can approach that of the injectable biologics.

So, going through the first bullet on the left, as you may know, fingolimod was a pioneer in class and served as a proof-of-principle that targeting this pathway can be effective in autoimmune diseases. Fingolimod however has significant cardiovascular limitations and sometimes even overnight inpatient monitoring can be required in some patient populations. Older drugs in the class are approved for non-dermatology indications, but they don't always provide selectivity against the S1PR3 receptor for example, which can lead to hypertension, macular edema, shortness of breath, and even cancer. This and other safety concerns have largely limited the ability to explore the use of this class in dermatologic disorders.

The last bullet on the left there just reiterates that everyone is looking for an oral agent that treat psoriasis with activity close to that of the biologic. For a while, it was hoped that the JAK inhibitors, for example, would be able to fulfill that goal, but the recent addition of black box warnings has resulted in roadblocks to those agents. The sphingosine agonists, actually have a pretty extensive history of safety now, and hence people are moving to them into this space with some renewed vigor when possible. I've touched on several of the bullets on the right there. They give more details specifically about vibozilimod.

Additional things to know about vibozilimod include that we have clear preclinical and early clinical validation and corresponding study that we're in the midst of clinical trials in atopic dermatitis and psoriasis, and we've seen preclinical synergy with other mechanisms, such as the IL-23 pathway in inflammatory bowel disease.

Slide #28 summarizes the design of the ongoing Phase-2 study in psoriasis. In this study, 240 patients are randomized amongst three doses of vibozilimod and placebo and treated for 16-weeks to obtain results on PASI75, the primary endpoint for the study. This is basically depicted as a second column on the slide.

There are a series of subsequent re-randomizations to provide insight into things like the durability of response on therapy, and the durability of response following

cessation of therapy, and these are represented by the subsequent columns in the slide. The study has just started and approximately 20% of the subjects have been randomized.

Slide #29 represents the Phase-2 of the atopic dermatitis study. This study also consists of 240 patients randomized to three different doses of vibozilimod or placebo. The primary endpoint also occurs at week-16, although, of course, it's different, it's EASI-75 instead of PASI-75 reflecting differences in their disease. This study is ongoing and involves the US, EU and Latin America.

This Slide #30 transitions to our brain penetrant program targeting mutant estrogen receptor in the treatment of breast cancer.

Slide #31 gives you some background and rationale for estrogen receptor antagonists program. The first bullet points out that fulvestrant, is the main Selective Estrogen Receptor Degrader available for patients failing first line therapy, but it's limited by the mode of administration, that is intramuscular and inactivity against mutations at clinically practical doses. Elacestrant, a newer estrogen receptor antagonist with activity against immune forms of this receptor, met its primary endpoint in a Phase-3 study for providing proof-of-concept for this approach. It's important to note however, that other similar drugs in the class have not always correspondingly succeeded. The success seem to be molecule dependent.

The last bullet just points out that most of these hormonal therapies are now given in combination with other treatments, but that other treatments have not usurped the role of these estrogen receptor degraders and antagonists.

Slide #32 goes over our current clinical program for SCO-120, which completed both single ascending and multiple ascending dose escalations in volunteers, and we're at/or near exposures that we expect to be efficacious at this point. So, far, the drug has been generally safe and well tolerated. In the middle of the slide there, we want to confirm the tolerability of these doses in patients and see if we can escalate a bit more in Part 1 of a patient study that's already in progress for patients who have failed at least one prior endocrine therapy and no more than three prior chemotherapies. Our main focus will be to confirm the safety and pharmacokinetics we saw in volunteers, in patients. Subsequently, we will focus on obtaining preliminary efficacy information and several cohorts listed at the bottom right of the

slide. These include patients with ESR1 mutations and those resistant to aromatase inhibitors, resistant to aromatase inhibitors and fulvestrant as well as those with brain metastases Finally, slide #33 is just a short summary of our plans for this program. We anticipate a readout from the ongoing study I described in the previous slide in fiscal year 2024, followed by initiation of Phase-2 study and a potential NDA submission in fiscal year 2027.

And with that, I'd like to turn the presentation over to my colleague, Dr. Nitin Damle, who will walk you through some exceptional data with one of our upcoming biologics projects. Nitin?

Dr. Nitin Damle: Thank you very much, Siu, and good evening, everyone. My name is Nitin Damle, and I would like to provide an update on our first biologic therapeutic program SBO-154.

During our presentation to this audience a year ago, we had described SPARC's initiative to invest in biologic therapeutics and introduce our strategy to explore antibody drug conjugates as anti-cancer therapeutics. SBO-154 is the first product of such exploration and represents an antibody drug conjugate, in which a humanized IgG1 antibody with a high affinity for MUC-I alpha/beta heterodimer, is linked to a potent cytotoxic drug that preferentially kills dividing cells when delivered to tumors via antibody drug conjugates. The cytotoxic drug used as a payload in SBO-154 is clinically and commercially validated for clinical use in different types of cancers.

The tumor target that we have been interested in focusing on is human Mucin-1, also known as MUC-1, as shown on slide 36. MUC-1 is a glycoprotein overexpressed on the surface of a wide variety of carcinomas and had been the focus of immunotherapies over the last two decades. Most of those efforts were clinically unsuccessful in large part due to the presence of a high level of circulating MUC-1 carrying the epitope recognized by anti-MUC-1 antibodies used in those studies. Mature MUC-1 antigen displayed on a cell surface as shown on this slide is a heterodimer of extracellular MUC-1 alpha and a transmembrane subunit MUC-1 beta. In this MUC-1 alpha subunit is the one that gets shed and can be found in large quantities in the sera of cancer patients. Such shed MUC-1 in circulation can intercept anti-MUC-1 antibodies used denying them the opportunity to bind to tumor cells resulting in their therapeutic failure.

The MUC-1 alpha/ beta epitope targeting antibody used by us in creating SBO-154 was originally in-licensed as a mouse antibody and was subsequently humanized at SPARC and further covalently linked to a cytotoxic drug to create SBO-154 and used in our preclinical evaluation. One of the key reasons for the use of anti-MUC-1 antibody for intracellular drug delivery is that any antibody bound to MUC-1 is rapidly internalized, and thus is ideally suited for intracellular delivery of potent cytotoxic agents.

The next Slide #37 shows that red fluorescence humanized anti-MUC-1 antibody, bound to MUC-1 on the cell surface, is rapidly internalized over time and inside the tumor cells. And thus, is not recognized by green fluorescence secondary antibody outside the cells into mice red fluorescence anti-MUC-1 antibody, continues to appear as red fluorescence, whereas the antibody that is still on the cell surface and has not been internalized, is able to be recognized by green fluorescence secondary antibody to create a yellow-orange fluorescence as you see in this slide. In contrast, similar red fluorescent Rituximab, targeted at human CD-20 and used as a non-binding control antibody does not show any fluorescence, indicating its lack of tumor binding and internalization.

In the next two slides, I would like to share with you anti-tumor efficacy of SBO-154 against high MUC-1 expressing human pancreatic carcinoma xenografts, established in immunodeficient mice. In this evaluation, pancreatic carcinoma xenografts were first established prior to the initiation of systemic therapy with MUC-1 targeted ADC SBO-154. Similar ADC created using Rituximab was used as a non-binding control in all evaluations.

Slide #38 shows that SBO-154 causes dose dependent inhibition of growth for the xenografts and at the highest dose causes shrinkage of the established tumors.

We further evaluated anti-tumor efficacy of SBO-154, in a model in which xenografts of the same pancreatic carcinoma were allowed to grow to large tumor masses, accounting for up to 5% of the body weight.

As shown in slide #39, SBO-154 was able to cause regression of such pre-existing large tumor masses of MUC-1 expressing tumors. In contrast, non-binder ADC of Rituximab, allowed for further uninhibited growth of such large tumors. Thus SBO-154 exhibits the ability to cause strong growth inhibition in a MUC-1 overexpressing

carcinoma model. We have undertaken detailed anti-tumor efficacy assessment of SBO-154 in various other carcinomas that express varying levels of MUC-1. These assessments would allow for greater appreciation for the potential of SBO-154 as an anti-cancer biologic therapeutic.

In light of the above preclinical proof-of-concept and as shown in the next Slide #40, we have advanced SBO-154 program to the preclinical development stage with the intent to file IND in the US in in the financial year '24.

With this update, I would like to stop and hand over further discussion to my colleague, Dr. Vikram Ramanathan. Vikram?

Dr. V. Ramanathan: Thank you, Nitin, and good morning, good afternoon or good evening to you all based on where you're located.

My name is Vikram Ramanathan, and I'll give you an update on SCD-153. SCD-153 is an NCE that we're working on with potential for use in autoimmune disease called Alopecia Areata. SCD-153 is a topical agent for this disease which has a significant unmet medical need.

Slide #43 gives you some background on the disease. Alopecia Areata is an autoimmune disease that causes loss of scalp hair and clumps, and it's a psychologically very debilitating disease. This occurs because the hair follicles, which are normally protected from the effects of patrolling immune cells lose their so-called immune privilege. So, on the upper left is a diagram of a healthy human hair follicle. At the base is the bulb of the follicle. Immune cells are present, but the bulb of the follicle is normally immune from their effects. To the right of it, is a depiction of a diseased hair follicle in alopecia areata. Some changes are immediately apparent. The hair has fallen off, and there's a big swarm of immune cells present at the base of the follicle.

The text on the right summarizes the changes in the disease. The hair follicle rapidly progresses from growing or anagen phase to the transition or catagen phase and then eventually to the resting or telogen phase.

Secondly, there's a collapse of the normal immune privilege in the bulb. The culprit CD4+ and CD8+ T cells infiltrate the area at the base of the follicle. In particular, it's a

subset of the CD8+ called NKG2D positive CD8+ cells that cause the damage. However, the hair follicle structures and stem cells are preserved, suggesting that hair growth is possible in principle if the hair follicle reverts to its original state.

Finally, the picture in the bottom left shows how the disease manifests in real life. And this remains an unmet medical need at this time. Our SCD-153 is a topical agent that seeks to reverse these immune changes and allow normal hair growth.

So, if you move to Slide #44 now, you're looking at in vivo studies in a telogenic hair growth model. The hair on the back of the mouse is clipped at eight and a half weeks of age. The right side is treated with topical SCD-153 and the left side is left untreated. The top row shows that vehicle treatment does not result in hair growth. The images in the second-row show that the treated area on the right side of the mouse shows hair growth, but not the untreated left side. In the bottom row, we see the effects of the treatment of the JAK inhibitor tofacitinib.

In summary in this model, SCD-153 stimulates robust hair growth after two doses given on alternate days. It promotes re-entry of the hair follicle into the antigen growth phase, possibly by activation of stem cells at the base of the hair follicle.

So, moving on to Slide #45 now, there is evidence that the topical SCD-153 also stimulates hair growth in an immune model of alopecia areata. C3H/HeJ mice are a strain that spontaneously develop alopecia areata in about 20% of the cases. The figure on the left shows pictures before and after topical treatment.

The box in green shows good hair growth on the back of the treated area. The head region was not treated and as expected did not show hair growth. On the right are images of the alopecic skins of these mice when seen under the microscope. In the untreated skin, there is a strong staining for the culprit CD8+ immune T-cells, and this staining is greatly reduced in the treated skin. We also have separate evidence for reduction of inflammatory cytokines in this treated skin.

In summary, we are working on a potential topical treatment for alopecia areata. We have separate data which we have not shown here that the compound suppresses inflammatory cytokines in vitro in cell culture, and that here we have shown you a data that show that it promotes hair growth in two animal models of disease.

Finally, slide #46 indicates that the IND enabling tox studies are underway, and we will be filing an IND soon on the completion of these studies in the early part of 2023. We look forward to sharing an update on this effort in the coming time.

This concludes my part of the presentation and I would like to hand over the baton to our CFO, Mr. Chetan Rajpara for his Financial Update. Chetan?

Chetan Rajpara: Thank you Dr. Vikram. Good evening, everyone. This is Chetan Rajpura, CFO at SPARC.

I plan to go over SPARC financials and cash position at a high level.

Slide #48. During FY'22, total income was at Rs.144 crores, equal to US$19.3 million, while total expenses were at Rs.347 crores, equal to $46.6 million, resulting into a net loss of Rs.203 crores, equal to US$27.3 million. FY'22 income was lower as compared to FY'21, as previous year income included an upfront non-recurring receipt of US$20 million from SCD-044 licensing deal.

Let me update you on our financial results for first quarter of FY'23. For Q1 FY'23, total income was at Rs.29 crores, equal to US$3.7 million, while total expenses were at Rs.111 crores, equal to US$14.4 million, resulting into a net loss of Rs.82 crores, equal to US$10.7 million.

Slide #49, as you may be aware company raised Rs.1,112 crores, equal to US$148 million in July '21 by way of a preferential issue of convertible warrants. The company has already received Rs.409 crores, that is US$55 million, being 25% payable on application as well as conversion of warrants. The balance sum of Rs.703 crores, equivalent to US$93 million, is expected to be received by end of December '22 upon the conversion of all warrants by the investors. The company has a line of credit in place for Rs.250 crores, equivalent to US$31 million from the parent company, in addition to the bank facilities for Rs.245 crores, equivalent to US$31 million. Bank facilities for Rs.183 crores, equal to US$23 million were utilized as on September 30, 2022, which is planned to be repaid in full before March '23.

The company has obtained shareholders' approval at the last AGM for raising sum of up to Rs.1,800 crores, that is US$225 million by way of issuance of securities.

The company is in process of licensing the late stage clinical asset, which will generate the additional liquidity. For FY'23, approximately, 1/3 of our expenses are budgeted for the clinical costs. We are aggressively managing our costs and working to control our non-clinical expenses.

That's all from me today on the financial update. A big thanks to all for joining the call. I will now hand over the call to Jaydeep for facilitating the Q&A.

Jaydeep Issrani: Thank you, Chetan, and we will now open the call for Q&A.

Moderator: We will now begin the question-and-answer session. We have the first question from the line of Kunal Randeria from Nuvama. Please go ahead.

Kunal Randeria: Sir, my questions are around vibozilimod. Firstly, you are pitching this as an alternative to JAK inhibitor, right, because of safety, signals in JAK inhibitor. But recently, TYK2 inhibitor Sotyktu was approved by the FDA. It is probably the first product without the safety warning that has limited the use of JAK inhibitor. I am just wondering how to see your product versus Sotyktu?

Anil Raghavan: Thanks, Kunal, for that question. I think you may be referring to the recent approval of BMS's TYK2 product which works through the JAK pathway, but the long-term safety data for the product is yet to be awaited. So, if you look at the JAK as a class, the concerns came through when Pfizer came out with five-year data for their first product. So, TYK2 in that sense, is in very early days to conclude if the safety profile is established. So, I think we need to wait for long-term safety data to have a final verdict on the safety of that class.

Kunal Randeria: The reason I ask was, I don't think there is any boxed warning that typically comes with other JAK inhibitors, so I thought maybe physicians might be more amenable to using this.

Anil Raghavan: For sure, I mean, at the moment, they are not treating it as a straightforward JAK inhibitor because it works through TYK2, even though if you look at the downstream has a similar pathway. In that sense, there are apprehensions about how that is going to play out in the long-term safety trial.

Kunal Randeria: My second question is again on S1PR1 receptor. Maybe fingolimod could be going generic in a couple of years, and it's a multi-functional S1P receptor agonist. So, I'm just wondering, what advantage would your product have over fingolimod?

Anil Raghavan: If you look at fingolimod, it is primarily approved and used in multiple sclerosis. And we are seeing the second generation of S1PR agonists also coming into the multiple sclerosis. But our development of the S1P1 program is in dermatology, and we are not going after the S1P1 pathway in multiple sclerosis. But having said that, if you look at our data on both the agonistic potency and the extent of internalization as we have disclosed in previous presentation, vibozilimod is probably a best-in-class agent, and we are currently running clinical trials, both in psoriasis and atopic dermatitis. And our data from those trials will dictate the competitive efficacy of our product against some of the other S1P1 programs which are actively explored in dermatology.

Dr. Siu-Long Yao: I just want to add to Anil's comment there, that we also believe that we have a very clean compound compared to some of the others. So, because of the great potency, we're hoping to have a better therapeutic index, which would give us an advantage over the existing compounds.

Kunal Randeria: There have been a lot of S1P1 products in the market for several years now. So, just wondering why the companies are not pursuing clinical trial in the dermatology space?

Anil Raghavan: If you actually look at the dermatology field, which has now begun to form. There are three programs in clinical development including ours. Arena Pharmaceuticals, which is now Pfizer, is actively pursuing this, and BMS recently entered not with ozanimod, but with a different agent, which is in dermatology, which requires a very clean safety profile as Siu mentioned. So, this field is becoming active, especially after the JAK experience, both in atopic dermatitis and psoriasis. We may probably see this even expanding beyond that in the dermatology space.

Moderator: We have our next question from the line of Narottam Garg from Chanakya. Please go ahead.

Narottam Garg: My question is on vodobatinib. You mentioned that you would complete the recruitment for the program by the end of this financial year. And given that, the first study is a 40-week long study, if I'm not wrong, so, would that be fair to say that the

first read out for the Phase-2 will probably be by the end of the next calendar year, which is CY'23, is that how one should think about it?

Anil Raghavan: So, the expectation that we are setting through definition and also our previous presentation is that we will have top line data readout in the second half of financial year '24. We haven't made a more precise expectation setting in terms of specific order for data readout, but our expectation is that we will complete the accrual by this year and then nine months will give you the top line data which is towards the end of 2023 or early part of 2024 calendar year.

Moderator: We have our next question from the line of Jayesh Gandhi from Harshad Gandhi Securities. Please go ahead.

Jayesh Gandhi: Sir, can you provide me with peak sale expectation for PDP-716 and SDN-037?

Jaydeep Issrani: Since the assets are commercialized by our partners, we don't provide a forecast or peak sales expectation for our products.

Jayesh Gandhi: If you can help me with out-licensing fee that we have got from Visiox Pharma for these two programs?

Jaydeep Issrani: We've got 10% equity in Visiox, but that's subject to the approval from SEBI. And we are eligible for additional regulatory milestones on the filing and approval of the program.

Anil Raghavan: In addition to the milestone and the 10% equity, we are also eligible for low double-digit royalties in this program.

Jayesh Gandhi: We are in process of out-licensing phenobarbital also. Any ballpark number if you can provide?

Anil Raghavan: Not at this point, I mean, we are in active negotiations on phenobarbital licensing. We hope to conclude a transaction soon as the PDUFA date for the program is coming up in November of this year. Our hope and expectation is that we will be able to conclude a transaction before the PDUFA outcome, so that, we can go into a launch as soon as possible. So, at the moment, we're not in a position to give you details on the transaction that is shaping up.

Moderator: We have our next question from the line of Manish Jain from GormalOne LLP. Please go ahead.

Manish Jain: On CML, how are we comparing to asciminib in particular? And later, I'll go to PD on vodobatinib.

Anil Raghavan: Hi, Manish. Thank you for that question. In CML, if you look at the setting that we are pursuing, and the data that you have seen in this presentation, is different from what Novartis has pursued for their registrational trials and subsequent trial. What we have here is last line setting, these are patients coming off ponatinib, failing three lines of tyrosine kinase therapy, and one of which needs to be ponatinib. Asciminib trial was in two lines of failure against bosutinib. So, in that sense, both the severity of resistance and the challenges in the composition of the resistance is significantly more, and there are no last line trials in this setting. So, we cannot directly compare the two programs.

Manish Jain: So, in terms of additional patients to be recruited on this program, on Slide #14, the numbers highlighted those are enrolled. How many more to get enrolled?

Anil Raghavan: I think as per the original FDA discussions, the target was in the range of roughly 50 patients for registration submission, but that's something which we hope to discuss with the Agency once we go back to them towards the end of this year given the rare disease nature of this indication.

Manish Jain: On PD essentially, what kind of risks do you foresee more than looking at the quantum of peak sales, what are the things that can actually determine the sales potential of the product, what are the risk factors or performance factors, if you can give a little bit of insight on that?

Anil Raghavan: We are in a Phase-2 proof of mechanism study for the Parkinson's disease, where we are exploring the viability of novel hypothesis. As you've seen in this presentation and also in earlier presentations, we are pursuing a fairly large and robust data set and being fairly transparent in terms of why we think it's a reasonable shot to take. At the same time, the translational risk of failure in this area is quite substantial. So, the translatability of these preclinical models into clinical outcomes, which we will see once we have a top line readout from the program. I think I would rate that as one of the more significant risk items. And if we are successful, and if we get statistically

relevant difference between treatment arms and placebo, the extent of that difference in terms of both how much of slowing down of the deterioration of the MDS UPDRS score plus, how long you can keep patients from having to need symptomatic therapy which is levodopa, they're going to be important factors in this program.

Manish Jain: Given that vodobatinib is quite significant, so, what are our manufacturing plans here to ensure that there is no slippage or delay led to manufacturing concerns?

Anil Raghavan: At the moment, we are not looking at diversifying the manufacturing base with a Phase-2. This is an active consideration for us going into Phase-3. And once we have success, we will definitely expand the manufacturing base with this given both the scale or potential scale of the product plus the potential value impact on our portfolio. We will not spare any effort in terms of creating multiple manufacturing options for this, but that's more of Phase-3 question as against the Phase-2 question.

Moderator: We have our next question from the line of Girish Bakhru from OrbiMed Advisors LLC. Please go ahead.

Girish Bakhru: Just continuing on vodobatinib, actually on the PD side, just wanted your assessment of the risk benefit ratio, which I think FDA has been focusing on when they are looking at new treatments coming in this class. So, from what all approvals have been coming in PD, we have not seen any major breakthrough so far, and so many different pathways are being tried, like biologics or alpha synuclein and all these things. So, where do you think FDA will focus on Phase-2 readout if it comes to that?

Anil Raghavan: I think as you mentioned, we have seen a real dearth of options here, especially after the failure of some of the alpha synuclein targeted antibody programs. From our standpoint, looking forward to FY'24 for readout of the PROSEEK program, what is going to be important is the extent of activity that we see and the toxicity profile that we see. I mean, when we look at our program so far, we have around 350 - 355 patients and we are not seeing as significant or alarming signals on safety so far unlike some of the antibody programs both in AD and PD, which was, I guess the basis of your question, people have seen significant level of neuro-inflammation. We haven't seen any alarming signs on toxicity. But when I say this, I'm talking about just blinded visibility of safety data. So, a real sense of where we stand in terms of risk benefit, can only be commented upon once we have an opportunity to see the data from this

fairly large as in 505 patients study next year, that will be an important proof-of-concept moment for this hypothesis also is not just for us, as to your earlier point in terms of the dearth of options. If you look at everything that's going on in sporadic PD as in non-genetic PD, Abl inhibitors is probably the most advanced and probably the more viable hypothesis that is being explored at the moment.

Girish Bakhru: Just to clarify, you're not pursuing PD psychosis?

Anil Raghavan: No, in PROSEEK trial we are recruiting very early stage Parkinson's patients. They haven't experienced symptomatic dopamine altering therapies like levodopa. So, in that sense, what we are trying to catch is early stage neuro degeneration to the extent that's possible, and try and slow down the progression of disease, so that the trajectory of our patients, as in patients under treatment, will be different from the trajectory of patients under placebo. And by slowing down the disease substantially, your hope is that you can modify the disease over a course of longer period and keep patients off the need to have symptomatic therapy, which creates subsequent complications like dyskinesia, etc.

Girish Bakhru: I think we discussed this last time. You said that there's been of course a lot of interest around this asset and several parties are probably looking at it. So, if you could just give some sense on is there a potential of monetizing this soon after positive Phase-2 readout?

Anil Raghavan: At the moment, we're committed to kind of seeing the Phase-2 clinical trial through and we are resourced to do that. And it opens up a lot of strategic options to SPARC both in terms of encashing partially, or kind of staying on with the asset. Those are all options that we will evaluate as we go into next year. But unfortunately, I don't have a firm guidance on that right now.

Girish Bakhru: Last one on SCD-153. I couldn't get. Is the asset also a tinib here for alopecia?

Anil Raghavan: The lead indication for that is alopecia areata.

Girish Bakhru: I mean, there is already like two, three tinibs like Olumiant I think got approved recently.

Anil Raghavan: This is not a kinase inhibitor. This is not a tinib. This is a novel mechanism of action and we haven't disclosed mechanism of action for IP reasons in this presentation.

Girish Bakhru: But any comment on, how even I don't know if Olumiant is expected to do well or what, but I notice a very high unmet need, but with your mechanism action do you think could be far better than this.

Anil Raghavan: That is probably premature for me to take up a position on this and we are in late preclinical setting at this point and we will closely watch this space and as we accumulate more data and go into clinic, I'm sure we will be in a position to kind of articulate the relative performance factors for both these classes.

Moderator: We have our next question from the line of Ketan Gandhi from Gandhi Securities. Please go ahead.

Ketan Gandhi: Sir, can you please throw some light on how many programs do we have fast track approval, particularly LBD, PD and CML?

Anil Raghavan: LBD and PD are not in registrational trials, they are in Phase-2 trials, and in CML, we don't have fast track, but we have an orphan drug designation and path to an accelerated outcome, in the sense, surrogate endpoint, which needs to be confirmed with confirmatory trials later on. So, it's not a priority review or fast track as you indicated.

Ketan Gandhi: Where is Xelpros and Elepsia manufactured at present?

Anil Raghavan: Xelpros and Elepsia, the manufacturing base is Halol.

Ketan Gandhi: Phenobarbital and brimonidine, where we plan to manufacture?

Anil Raghavan: We have two sites for phenobarbital. We haven't disclosed the manufacturing strategy for the product. Once we have a finalization of the commercial partner, we will be in a position to communicate that.

Ketan Gandhi: What would be the timeline for phenobarbital to reach the peak sales potential?

Anil Raghavan: So, again, we're expecting an outcome from our current review process by next month. And as you see, this is a conversion of a DESI product, I mean, in the sense

that there are unapproved products in the market and the DESI exclusivity works like once you have an approval and using due process, FDA has a commitment to take out the unapproved products in the market. And that decision to enforce the DESI exclusivity is driven by certain determinations the Agency needs to make during the course of the first year or so, in a sense, the robustness of the manufacturing plan and the unmet need So, there is a certain set of determinations that the agency need to make in terms of the timing of enforcing that exclusivity. We don't want to set a stipulative expectation in terms of specific time for seeing the unapproved product going out of the market. But the estimation of the peak sales is going to be linked to that event.

Moderator: We have our next question from the line of Harith Ahamed from Spark Capital. Please go ahead.

Harith Ahamed: My first question is on Xelpros, which is partnered with Sun Pharma. It's been like three years since launch. So, how has been the ramp up so far and have we kind of reached the peak sales or peak Rx for this product?

Jaydeep Issrani: Thank you for the question, Harith. As I mentioned earlier, this is a program partnered with Sun Pharma, and they would be providing an update on the sales at appropriate time or whenever they disclose about the sales potential. So, we wouldn't be commenting on where or how much they are doing. Sun may provide guidance, as they have been doing in the past about the program.

Harith Ahamed: On PDP 716 and SDN-037, just curious as to Sun Pharma is not the partner for these assets given ophthalmology is one of their focus in specialty segments and we've also partnered with them on Xelpros. If you could comment a bit on the capabilities at Visiox and your expectations in terms of how they plan to take these assets forward?

Anil Raghavan: For each of these out-licensing commercialization decisions, you go through a fairly robust process in terms of identifying a partner, who gives us the best shot of maximizing the deal from the product. And in this case, also, we have gone through a process and we had multiple players involved in terms of interested parties, and Visiox was probably the most aggressive both in terms of the investments that they're committing and the quality of the team that they're bringing to the table, and their track record in terms of having succeeded in other areas, not in ophthalmology, I mean, this is their first ophthalmology effort. So, there were several factors, which

gave us confidence in terms of the potential trajectory of the product with this proposal. And they also, as Jaydeep mentioned earlier, gave us an opportunity to have contingent share of value in addition to a traditional structure. So, it's a traditional structure plus access to a slice of value that this product may create. So, in that sense, both on commercial terms and other dimensions of the proposal, both in terms of capability, investments, committed, etc., that is a very healthy proposal, and that's the reason why we went to Visiox.

Harith Ahamed: On phenobarbital, can you give some color on the other therapies in the neonatal seizures indication against which phenobarbital will be competing and given that we are very close to the PDUFA date, any comments on the interest level in the assets from your licensing discussions with potential partners?

Jaydeep Issrani: Currently, there are no approved agents for treatment of neonatal seizures. Phenobarbital as you would know is the standard-of-care currently in the US and other markets. And the second alternative is injectable levetiracetam. So, those are the two agents, but both of them are unapproved therapies being used by the physicians. So, that's the answer to your first question. And on the interest, of course, we have been in discussion with potential partners, and we're trying to close this at the earliest possible so that the partner would have enough time and a longer period for commercializing the asset during the exclusivity period that we may get after the approval.

Harith Ahamed: On SCD-044, you mentioned that you're expecting completion of Phase-2 in FY'24. So, any timelines that we can expect for Phase-3 trials and maybe filing any target?

Anil Raghavan: So, the practice that we are following on these partnered programs is that, we're basically leaving the disclosure on the execution of those programs to the licensee since both parties are dealing with the same set of investors. We haven't in this presentation indicated a timeline. But, as you know, we are in two trials in atopic dermatitis and psoriasis and Sun would be in a position to set expectation in terms of the registration trajectory of the product.

Moderator: We have our question from the line of Manish Jain from GormalOne, LLP. Please go ahead.

Manish Jain: So, essentially, I just wanted to know on phenobarbital. My assumption is it will primarily be a hospital setting product. So, typically, how many sales reps would be required for such a kind of product?

Jaydeep Issrani: The anticipation that this is going to be a hospital product is correct. It will primarily be used in the ICU setting or neonatal centers, which are not many, few in US. And the number of reps or the sales team will be dependent on the partner that we engage with and their strategy to commercialize the asset. So, I will not be able to give you an exact number, but it should be in the similar ballpark range as any other asset targeting neonatal centers.

Manish Jain: Because, at some point of time, I had a broader question from this which I have been raising with you is for such kind of products, at what stage, does it make sense for SPARC to actually go out and try and do the marketing on its own rather than being dependent on an external party?

Anil Raghavan: Thank you, Manish. I think that's an interesting question. Your question is do you actually try and optimize it for a smaller product which requires a smaller scale of effort or do you go for a bigger product? Because the extent of bandwidth that we need to spend at management levels and also the distraction that it may cause for the rest of the portfolio, is going to be considerable. So, that's an interesting question and an active question in terms of what would be an appropriate time to look at other options for commercialization. But, at this point, we have concluded that phenobarbital may not be that opportunity.

Manish Jain: Given that we are now getting into very interesting biologics like SBO-154 and others, what's going to be our manufacturing strategy there?

Anil Raghavan: At this point, SPARC has no plans to invest and build manufacturing capabilities for these novel modalities or for that matter traditional modalities also. So, our manufacturing strategy is to broad-base the manufacturing or supplier base and de-risk as much as possible. So, in this case also, we will continue to build up multiple options for manufacturing and as we kind of start preparing for the IND next year for this, we are already evaluating multiple external vendors for the MUC-1 ADC program.

Moderator: As there are no further questions, I would now like to hand the conference over to Mr. Jaydeep Issrani for closing comments. Over to you, sir.

Jaydeep Issrani: Thank you, Yash, and thank you, everyone for joining today for the call. We tried to answer the questions you had, but in case there are any additional questions or follow on questions, you may reach out to us and we will respond to you through e-mail. Thank you once again for joining the call today.

Moderator: On behalf of Sun Pharma Advanced Research Company Limited, that concludes this conference. Thank you for joining us and you may now disconnect your lines.