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

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MANAGEMENT: **MR. ANIL RAGHAVAN – CHIEF EXECUTIVE OFFICER**
DR. SIU-LONG YAO – HEAD CLINICAL DEVELOPMENT
DR. VIKRAM RAMANATHAN – HEAD PRECLINICAL DEVELOPMENT
DR. NITIN DAMLE – CHIEF INNOVATION OFFICER
MR. CHETAN RAJPARA – CHIEF FINANCIAL OFFICER
MR. JAYDEEP ISSRANI – HEAD BUSINESS DEVELOPMENT, CORPORATE COMMUNICATION & INVESTOR RELATIONS

Moderator: Ladies and gentlemen, good day and welcome to SPARC'S Update on Clinical Programs and R&D Pipeline Conference Call.

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 this 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 from SPARC. Thank you and over to you, Mr. Issrani.

Jaydeep Issrani: Thank you, Neerav. Good evening, ladies and gentlemen. My name is Jaydeep Issrani, and I Head the Business Development and Investor Relations at SPARC.

On behalf of SPARC, I welcome you to the SPARC's Yearly Update on Strategy and Program Updates.

I'm joined by our CEO, Mr. Anil Raghavan, and members of SPARC's senior management team for the call today.

As we have done in the past, we will be using a similar format for our discussion today, that is, the presenters will walk you through the slides after which the call will be open for questions and discussion.

The presentation for today's discussion was shared earlier. If you have not received, you can download the presentation from our website, which is www.sparc.life.

Before we start today's discussion, I would like to remind you that our discussion today includes forward-looking statements that are subject to risks and uncertainties associated with our business. Hence, actual results may be different from those projected in today's presentation.

I will now hand it over to Mr. Anil Raghavan for his presentation. Over to you.

Anil Raghavan: Thank you very much Jaydeep for kicking this off.

Hello everyone. It's my pleasure to welcome you all to SPARC's 13th annual portfolio update. As always, we are immensely grateful for your continued engagement and support. This means a lot to us. Thank you, for taking the time.

I am sure, most of you have been regulars here and you know the routine. We have the SPARC's management team on this call today. Most of us are on these calls for some time. But we have a new addition. That's Dr Venkat Palle who joined us as the head of Drug Discovery and Pre-clinical Development late last year. Dr Palle comes with an impressive trail of achievements and we are lucky to have him leading the charge in discovery and preclinical development.

In terms of our agenda today, we will begin by covering certain important elements of our strategy. I will also give a bit of the 'lay of the land' of our late stage portfolio and provide some guidance on what to expect in the short to medium term. After which we will spend much of our time today on three things… Dr Siu-Long Yao will talk about our clinical programs, that's Vodobatinib in Parkinson's disease and CML, followed by Vibozilimod in Atopic Dermatitis & Psoriasis. Dr. Vikram Ramanathan and Dr. Nitin Damle will provide updates on the progress of two important programs which we introduced last year, SCD-153 and the SBO-154. We will conclude the presentation with brief comments on our financial situation and cash flows from Chetan.

So, with that, let's get going with slide 5 please

Here we have a recap of the shifts that we have taken in the last few years from being a primarily delivery system focused company to an approach which embraced the risk profile of a modern biotech fully. In the sense,

towards a portfolio of assets holding significant promise of improving the standards of care and making an impact on the lives of patients we hope to serve while carrying multiple layers of risks. But our model differs from the contemporary biotech business model on many fronts. We have spoken about these differentiating factors at length in the past, Our captive operations, Low cost of failure, Breadth of our portfolio, Compelling founder vision/financial commitment and so on. We have also spoken about our efforts to narrow our therapeutic focus and sharpen our execution. So, I don't plan to go over these foundational elements of our pivot again today.

But I would like to highlight two other points before we go. In the last few years, we have made a significant push towards strategic partnerships with tier 1 academic research systems globally to access high quality science and ideas early. We can clearly see the impact of that sustained effort in terms of collaborative programs moving to Clinic now. That is an important validation for a key tenet of our strategy. We also have additional early stage, undisclosed pre-clinical programs in collaboration with external marquee institutions. A productive bridge to external science has always been part of our vision and differentiation strategy. We are hopeful that the bonds and competencies we have built in establishing these structures are going to be extremely useful in further building out our portfolio. We really look forward to that.

I also want to touch upon an element which is not specifically called out on this slide. That's our changing modality mix and the maturation of our biologics group. We have completely moved out of NDDS programs which was our mainstay in years past. Traditional small molecule chemistry is a significant share of our effort and will continue to be so. We think small molecules will remain an important part of the therapeutic tool kit because of the larger swathe of intercellular targets that can be addressed with appropriately designed small molecules. We have made exploratory investments in building an in-house biologics capability 'ground up 'under Dr

Nitin Damle's leadership. That effort is now turning real with its first asset, an antibody drug conjugate, approaching its clinical entry in 2024. But more importantly we have built a competitive competency which can engineer antibody therapeutic constructs, be it traditional naked antibodies, drug conjugates or bispecific or multi specific antibodies and scale them to clinic. We are also leveraging our understanding from the ADC effort to create small molecule drug conjugates which is an important emerging therapeutic class. Additionally, we have several exploratory efforts in newer modalities and are taking some really meaningful bets around pushing our modality mix even further. We look forward to bringing additional programs from the biologics basket for sure and hopefully even from newer modalities in the short to medium term.

So, in recap, I want to re-emphasize the point I made during our call last year. For investors with an appropriate risk appetite and an ability to grapple with translational risk, SPARC offers a compelling story. And that story is getting more real now than at any point in the past as we have several important catalytic events coming up in 2024. So, let's go to the next slide to check out what is in store.

Slide 6 please

This slide has a straight forward listing of a series of data and phase transition events set up for the next calendar year. Some of them will have the ability to move the SPARC valuation substantially based on data either way. So, let's go over the list.

First up is the PROSEEK interim analysis. We are doing an administrative interim analysis which will give us the primary endpoint and secondary endpoints trends for 442 patients plus additional biomarker trends. The important business objectives of this administrative process are two-fold, first to initiate potential licensing discussions before the public release of the

full data set and secondly, to initiate planning and set up activities for the regulatory interactions and potential phase 3 trials of Vodobatinib in neurodegenerative disorders. We plan to maintain the study blind for everyone outside of a small group covered under a very strict confidentiality commitment.

We expect two more important program milestones in Q2, of 2024. SCD-153, will have its first data read out which will help us develop an initial understanding of the potential safety profile of the product. Also, more importantly, an ability to move to MAD studies in patients which can give us a lot more information regarding target engagement and efficacy markers plus some early indication of potential therapeutic dose.

We will also close out the patient enrolment of our Atopic Dermatitis program of Vibozilimod in the second quarter.

In the third quarter – PROSEEK will read out fully. As you can see, that is probably the most important data catalyst for the company currently and certainly most keenly watched. That will lead to setting up the End of Phase 2 meeting with the FDA to agree on the registrational path for Vodobatinib in PD. We will look to initiate the phase 3 program in Q4 and start exploring the fuller opportunity set which I will touch upon in a moment. We will also look to conclude our partnership strategy for the asset. PROSEEK will also give us clarity in terms of our resourcing requirements and options. So that's a big one.

We have three significant milestones setup for the last quarter of the calendar. Our first biologics IND. Our antibody drug conjugate program is currently tracking to that plan. We will then have phase 2 data from the Atopic Dermatitis study. From a SPARC perspective that would set the stage for additional milestone payments from our licensee + we can initiate the

FDA consultation and pivotal trial planning for AD. Finally, we will also have the MAD studies of SCD-153 getting started in Q4 24.

We have only listed the data or milestone events from our larger programs here. We will also have additional milestones like the refiling of Brimonidine and potential progress in enforcing our exclusivity for Sezaby. I will come back to those programs towards the end.

Before we go to the next slide, I want to summarize all these very briefly. 2024 will see several big moments for SPARC including opening the lid on a potentially game changing program for the company and the field. I can't overstate the significance. So, I want to spend the next few slides to fully unpack the true meaning of these milestones for SPARC and its shareholders.

Let's go to slide number 7

First the big news. PROSEEK achieved its recruitment target late last month. We set out to recruit 504 evaluable patients. We ended up with 513. Siu will give you more color in terms of both the state of the study and expectations, but I want to recognize the team and the effort that went into completing the accrual of this study which is one of the largest Phase 2b studies currently active in early stage Parkinson's disease globally. We started this trial in full swing when health systems around the world began to shut down because of the pandemic. Covid created enormous challenges in prosecuting this program, but we are genuinely happy to be here and want to thank patients, their families, our investigators and site staff around the world including some really passionate physicians in India who supported this program wholeheartedly and our execution partners and a large team at SPARC who drove this program with enormous zeal. As I mentioned in the previous slide, we expect the interim analysis completion by end of Q1, 2024

and a full readout and Phase 3 initiation soon thereafter. And that indeed is something to look forward to.

So why are we so excited.

PROSEEK will give us a definitive proof of concept for one of the most important hypotheses in the neurodegenerative diseases field. that's c-Abl inhibition as an approach for neuroprotection. The role of excessive reactive oxygen species and the redox imbalance it creates in triggering a cascade of events that results in neuronal toxicity and death has been identified as a potentially intervenable pathway across the neurodegenerative spectrum for some time now. Dr Ted Dawson of Johns Hopkins and several other groups around the world provided compelling mechanistic and pharmacological evidence supporting the c-Abl story. But this hypothesis has never been fully tested as the field lacked an appropriately specific agent with the relevant pharmacological properties, particularly good blood barrier penetration at safe doses. That's the promise of Vodobatinib. A sub nano molar inhibitor of c-Abl, ultra-specific to Abl 1 and Abl 2 that crosses the brain sufficiently to ensure adequate coverage with a peripheral safety profile which makes clinical exploration in the neurodegenerative context feasible. And importantly that promise was delivered by Vodobatinib in preclinical and in early clinical studies. The totality of evidence we have from everything we have done so far in this program was directionally consistent and supported translation.

In that sense, PROSEEK promises the first real validation for the c-Abl pathway in a sufficiently sized global trial which has the right patient setting i.e. pre-L DOPA patients with a DAT confirmed PD diagnosis, and right duration to see the effect beyond the placebo impact, 40 weeks in part 1 followed by an LTE period of another 40 weeks. I want to talk about the broader implications of a positive outcome in terms of opportunities it presents. But before that, from an operational standpoint, our immediate

priorities coming out of PROSEEK is the finalization of a late stage development plan including our partnership strategy, getting regulatory concurrence with FDA and other agencies around the world and initiation of its first registrational program. These will be the most important execution priorities for SPARC next year if we see the data we hope to see with PROSEEK. Now let's go to slide 8 for a bit of a big picture view.

Next slide please.

The way to look at the opportunity PROSEEK validates, is to look at various layers of the hypothesis and see the context in which it is tested. At its core, PROSEEK gives direct validation to the relevance of Abl inhibition and oxidative stress response modulation in Parkinson's disease. So, if inhibition of c-Abl means bending of the degenerative curve in a significant manner, that not only slows the progression during the initial, pre 'symptomatic therapy' phase which PROSEEK targets, but can also extent the period of effective management under dopamine therapies. And at the starting phase of the spectrum, biological understanding of prodromal PD or precursor conditions like constipation and REM sleep disorder can offer additional opportunities to intervene early and intervene proactively. So, the inner core of opportunity for Vodobatinib & other selective, brain penetrant Abl inhibitors is to become a background therapy across the PD treatment continuum.

The next layer comprises a set of diseases characterized by α Syn aggregation like the Multi System Atrophy and Lewy Body Dementia. In these indications several pathological hallmarks of PD are at play, but also some important differences. Like the area of the brain that gets affected. We believe that a compound like Vodobatinib which can moderate oxidative stress response and get distributed uniformly across the brain is an agent worthy of testing in these immediate adjacencies. That basket from an

epidemiological and inadequate SOC standpoint, offer some really large opportunities.

Then there is an outer ring of possibilities driven by other culprits like activated Tau, A Beta, SOD, etc. in diseases like Alzheimer's, & ALS. Our early studies in IPSC derived neurons indicated some of those possibilities and we plan to continue studying Vodobatinib and its promising backup series in these conditions.

So, summarizing these two slides, we have reached a significant and important milestone with the completion of the enrolment target for PROSEEK. We expect substantial data-flow which not only validates our hypothesis, but has the potential to teach us a lot more in terms of the disease biology, heterogeneity of the disease & response, correlation of several important candidate biomarkers, and so on. That sets up multiple patient settings which can potentially make Vodobatinib the backbone of the standard of care across the neurodegenerative continuum. And that is a huge deal. And we are understandably super excited about it. Now let's move on and examine the key "what if" question, which I am sure all of you have in your mind. Let's go over it.

Can we have slide 9 please

Much of the current biotech landscape consists of companies created to test one interesting hypothesis. Investors and teams move on, either cash out on success or move onto something else on failure, the next shot. That's the typical story. We are architected somewhat differently with a substantial internal discovery, translational & clinical development capability with a cost of failure advantage. We are designed to spread the risk across an actively managed portfolio, learn from our successes and failures, strive to improve the quality of our competencies and deliberately build OPTIONALITY as we come to critical decision points like the one that we are approaching now.

And we believe we do have meaningful optionality. Let me take a minute to explain.

SPARC's optionality has two coordinates. At a program level and at a competency level. At program level, we have multiple options and combinations to pivot to if we need to. Immunology offers two high value options in Vibozilimod and SCD-153. We expect to see additional value unlocking data from Vibozilimod in the coming year. We will also see the data build up for SCD-153. We have a broader set of options to go to in Oncology, where the highest value bet is the Muc-1 platform which can generate multiple products using the ADC and bispecific constructs. Vodobatinib CML offers a relatively lower value, but significantly surer option to leverage the asset if we unfortunately fall short in neuro.

But I think it is also equally important to consider the optionality provided by our competencies. SPARC represents one of the more sophisticated translational engines attempting to innovate for the world from India. Across the value chain, from the ability to develop assets in multiple modalities, to self-sufficiency in various pieces of translational development in multiple therapeutic areas and the ability to design and execute clinical programs globally, we have had substantial learning from our successes and failures. That's one of our biggest assets differentiating our program. I will go over a few specific points on our program hedges in the next couple of slides. But let me say this in summary. Investors evaluating SPARC should look at our portfolio as one substantial opportunity for standard of care altering innovation in a large unmet area – that is Vodobatinib – backed up by a larger number of decent hedges across immunology and oncology, with many of them reaching important developmental milestones in the near future

Now let's go over slide 10 to take a look at the immunology programs

I have already spoken about these two programs in my earlier comments. Just a few high-level observations. On Vibozilimod, proof of mechanism for SIPR1 agonists exist from competing clinical programs in dermatology and a wider swathe of indications outside of dermatology. As previously disclosed, we have reached target pharmacodynamic levels at safe doses, that is well correlated with efficacy in this class. We have completed pooled analysis of the safety data from 125 patients across Atopic Dermatitis and Psoriasis and managed to remove most of the extensive cardiac monitoring initially required in the trial. We will go over this in more detail during our clinical segment. We are now aggressively scaling the program with significant expansion to Europe and Canada. We are also adding new sites in the US. From a competitive positioning standpoint, in Atopic Dermatitis and Alopecia Areata, the oral standard of care currently consists of JAK inhibitors which carry a black box warning. Both Vibozilimod and SCD-153 has the potential to be much safer options in AD and AA. On a separate note, both these programs offer excellent examples of our strategic partnering program. Vibozilimod, as you may know, was developed in partnership with a French biotech, Bioprojet and SCD-153 is an ongoing collaboration with a two very accomplished groups from the US and Czech Republic. We started our collaboration with these groups as a joint development effort with an option to license their IP. I am happy to announce that SPARC exercised the option to license the Intellectual Property supporting this program. You may have seen our press release today announcing the definitive licensing agreement. That allows us to move forward with SCD-153 as a fully owned SPARC program.

Next slide, that's slide no 11 explores additional pages we can turn to in oncology.

CML component of Vodobatinib was always meant as a backup for the Parkinson's program. We have had significant regulatory interactions recently about the nature of the data package the agency would like to see

for registration. In line with FDA's broader strategic shift towards encouraging companies to explore marketing approval in earlier settings, FDA suggested a comparative trial in 2L failure patients as against the last line, that's the post ponatinib setting. We will go over additional detail on the emerging design of the expected study in the clinical session. We are also re-evaluating the costs and timelines before deciding to go it alone or partner at this stage. But Vodobatinib in CML represents a surer hedge, surer because of clinical validation, and alignment with FDA on a path to registration. We will have clarity and readiness to move forward when we reach that decision point post PROSEEK.

We have discussed the ADC program earlier also and Nitin will do a deeper dive later. But let me say this, we see enormous opportunities in Antibody or small molecule targeted preferential delivery to cancer cells. These platforms can be leveraged for the delivery of a wide set of payloads ranging from small molecule traditional cytotoxins, highly potent targeted therapies with limited safety margins, RNA therapeutics, etc. Muc-1 gives us a differentiated platform which can open up multiple product opportunities. Our oncology effort also carries significant interest in certain other target classes, like new synthetic lethality pairs or exploring ways to intervene in the RNA process.

Before I look to summarize, want to talk briefly about how our competency has evolved and how it offers another level of risk mitigation. Please go to slide 12.

As I said, we don't look at SPARC just as a portfolio of programs, though they are the key drivers of current value. A significant part of our proposition is our process which is refined over a long period of time, imbibing the learning from our successes and failures. We believe that SPARC development process in terms of targets we seek to address, validations that we insist, developability considerations around the nomination of an asset,

clinical strategy and execution, adoption of biomarkers, and our overall development tool kit, all have come a long way since we spun out of Sun. Equally importantly our portfolio management process evolved into a rigorous, objective methodology which puts a premium on establishing and enforcing smart data stage-gates which allow us to kill unviable programs early, cheap and completely. So, SPARC is not just a bet on a high value portfolio, but on a system and competency which has been evolving and invested into over a long period of time. I hope you share our excitement and confidence beyond the here and now, which is certainly exciting, but also for the long run.

Let's go to Slide 13. We will have additional comments on cash flow and burn from Chetan towards the end. But want to make a few observations on additional cash events outside of Vodobatinib and other earlier stage programs we spoke about here.

Our partners have successfully launched Elepsia, Xelpros and Sezaby in the US. Elepsia is commercialized by our partner Tripoint and we have achieved early run rates indicative of a robust growth trajectory. Unfortunately, we have had supply disruptions from the Halol import alert. We have managed to find an alternative vendor and are in the process of finalizing our plans for the relaunch. Sezaby, that's the Benzyl Alcohol free formulation of Phenobarbital, got approved in the US last November. Sun, our commercialization partner has launched Sezaby in the US during Q1, 2023. As you may know this product has a 7-year exclusivity and enforcement of that exclusivity is a key value driver for the growth of this franchise. We are working diligently to make the case with the agency and other stakeholders in order to remove unsafe options from the market. Let me not go into specifics on the numerous initiatives we are currently undertaking to safeguard the interest of patients and caregivers, we are hopeful that we can make progress on this important objective in the near term. And on PDP-716, as we have communicated earlier, we have received a complete

response letter from the FDA indicating certain deficiencies with the external API manufacturing plant. There were no additional clinical data requirements indicated at this time. We have already identified an alternative API source and are in the process of planning a resubmission in 2024. Finally, as we mentioned earlier, we expect the top line and initiation of the phase 3 program in Atopic Dermatitis for Vibozilimod, that triggers a cash milestone for SPARC. Plus, we believe that there may be opportunities to commute future revenues from this program to generate additional cash to support the development programs if that is required post PROSEEK readout.

In the next two slides, 14 and 15, we have summarized the portfolio and key milestone expectations. We don't plan to go over them. Except for one program, which is our oral SERD, Bexirestrant. We took the program into dose escalations in patients. But based on data from competing programs, it was becoming clear that effective clinical differentiation for this class as whole is becoming a challenge. With small effect sizes, the program needs very large clinical studies and may not be the best of use of capital for SPARC. So, our portfolio review, as we disclosed earlier, down-prioritized the program late last year. We can go into additional detail if required during Q&A.

With that, let me transition the call to Dr Siu-Long Yao who heads up our Clinical Development group for the next leg of the presentation. He will cover the clinical stage programs in a bit more detail. I look forward to seeing you later for Q&A. Thanks again for your time. Over to you Siu.

Siu-Long Yao: Thank you, Anil. My name is Siu-Yao Long and I'd like to add my warm welcome, especially to the many who have accompanied us over the years. I oversee clinical development and I'll walk you through our major clinical programs. So, slide 17, the first program I'll discuss is Vodobatinib, our c-Abl inhibitor of Parkinson's disease.

Slide 18 please. This slide gives you some basic background on Parkinson's Disease. There are a lot of people affected, approximately 7 million across the world and the prevalence is growing. There are anticipated to be 14 million affected by 2040. In fact, the growth of the disease is outpacing total population growth.

The therapy we have is one where we believe we can modify the disease, and actually change its trajectory or course, rather than just treating its symptoms.

The pictorial at the bottom of the slide is there to give you a general overview of Parkinson's Disease. I'd like you to focus on the middle row first. There, you have the disease starting from a prodromal stage on the left and progressing to diagnosis, maintenance treatment, complex treatment and finally, palliative treatment.

On the other rows, both above and below the middle row, there are major clinical events depicted that need to be managed.

As Anil mentioned, we are initially studying Vodobatinib in the prodromal/diagnosis stage but, if it works, there is the potential for all stages of disease.

Slide 19, please. This slide is a reminder of the design of the Phase 2 proof of concept study named PROSEEK. The study consists of 3 arms of placebo, a high dose of Vodobatinib and a low dose of Vodobatinib. Part 1 is the key part of the study where subjects are treated for 40 weeks and the effects of treatment on the primary endpoint of Part 3 of the UPDRS scale is assessed. The study is fully enrolled and we anticipate having some initial interim data in March, 2024.

Part 2 of the study is a long-term extension where subjects who initially received placebo, along with subjects initially treated with 384 mg, receive

additional treatment with 384 mg for an additional 9 months. Subjects randomized to 192 mg will continue that dose during the extension.

Approximately 87% of eligible Part 1 patients have rolled over to Part 2 and completion of the study overall is anticipated in May, 2025.

Slide 20 gives you an idea of the distribution of patients across the world. On the y axis is the number of patients from each region, and going across the bottom on the x axis is the region. Over 40% of the patients have come from the US.

Some additional detail about what we've seen so far is summarized in the other bullets on the right. There were grade ¾ events in about 6% of patients and GI events and rash were the most common AEs. We do have a DSMB, which is unblinded to treatment allocation, and reviews all safety data in an ongoing fashion and they have not recommended any changes in the conduct of the study following 6 interim reviews.

Slide 21 summarizes some of our biomarker plans in the study. The biomarker subgroup in the study consists of over 150 of the 504 patients in the study, which translates into over 50 patients per arm that will have biomarker data. What I just told you, though, is a simplification. In fact, some of the biomarkers will be obtained in all subjects but, in general, we are targeting to get 50 subjects in each arm with all biomarkers.

Biomarkers that will be obtained upon study entry and exit include dopamine transporter scanning and skin alpha synuclein content. Target engagement will be assessed by c-Abl and CRKL statuses, and neuronal death will be assessed through neurofilament light. Pharmacodynamics will be assessed through various downstream targets. We have smartphone-based measures of patient functional status.

Slide 22 provides a summary of some of the concomitant studies in the program. There is an ongoing carcinogenicity study and we plan to perform

a relative bioavailability study along with some drug-drug interaction studies to support Phase 3 plans. A human ADME study is also planned.

Finally, in slide 23 there is a summary of some of other related alpha synuclein based disorders that could benefit. There are the other classical synucleinopathies such as dementia with Lewy bodies and multiple system atrophy along with the prodromal synucleinopathies such as primary autonomic failure and REM sleep behavior disorder.

On the right is a Western blot that summarizes the activity of Vodobatinib in a model of Alzheimer's Disease. Molecular weight is depicted on the y axis and treatments used are displayed as columns. In this case, treatments were done in duplicate so there are two rows for treatment with control DMSO and 2 columns for treatment with Vodobatinib. As you may know, Tau is the protein of interest in Alzheimer's Disease so I'll ask you to focus on the phospho-Tau and Total Tau rows in the middle of the blot. As you can see, treatment with Vodobatinib significantly diminishes both, suggesting that treatment with Vodobatinib may be effective for Alzheimer's Disease.

Slide 24 transitions to our program with Vodobatinib in chronic myelogenous leukemia.

On slide 25 is a summary of the commercial landscape for CML. The graph on the left consists of rate per 100,000 on the y axis and year going across the x axis. The top curve is the incidence of the disease and you can see that it is steadily increasing over time. At the same time, the rate of death from CML is decreasing so that, overall, the prevalence of the disease, or patient population, is increasing.

In fact, the current market is valued at 3.5 billion US dollars. The graph on the right gives you an idea of the major drugs used in the treatment of CML and their respective market shares.

Slide 26 summarizes more recent data we've seen with Vodobatinib in CML. The figure you see is a summary of all the patients that we have efficacy data on. The rows represent the disease status of the patient population either at baseline in the top row or following treatment with Vodobatinib in the second row. Percent of the population is represented on the x axis. As you can see, most patients were not in response at baseline. Following treatment, over 40% of the patients achieved responses, which is really remarkable in this patient population, many of whom were resistant to ponatinib. Responses were very durable as you can see in the last bullet where median time on drug was over 32 months.

Slide 27 provides some preclinical data that supports our plans to do an earlier line study with a larger market potential. On the y axis you have tumor volume in mouse xenografts and on the X-axis is time in days. The top curve represents results obtained with vehicle control and the line below that is tumor size following treatment with nilotinib, a representative 2nd generation TKI for CML. You can see that there is some control of tumor size, but treatment with Vodobatinib, either at a lower or higher dose in the bottom 2 curves, is much better. This, along with other, data suggest that we can beat existing 2nd generation treatments in earlier lines of therapy and capture a larger market share.

In slide 28, we've had discussions with FDA and they are actually quite supportive of getting an initial approval in an earlier line of therapy. This is consistent with their project Frontrunner initiative. Our plans are summarized in the diagram below. We are planning a randomized study against one of the 2nd generation treatments for CML with a primary endpoint of major molecular response.

Slide 29, please. The remaining slides summarize Vibozilimod, our sphingosine agonist, for the treatment of psoriasis and atopic dermatitis.

Slide 30, please. As I mentioned, the 2 initial indications we are targeting for Vibozilimod are psoriasis and atopic dermatitis. The prevalence of psoriasis

in the US is approximately 8 million and the market is dominated by biologics. Though there are biosimilars, penetration has been limited to date.

For atopic dermatitis, the US prevalence is even higher, approaching 18 million. Though JAK inhibitors are available, they are associated with several black box warnings such as thromboembolism, cancer, major adverse cardiac events and death.

Slide 31 summarizes the design of the Phase 2 dose ranging study for psoriasis. There are 4 arms consisting of placebo and various doses of Vibozilimod. The primary endpoint is at 16 weeks which is Part 1 of the study. In Part 2, we will evaluate the effectiveness of switching to higher doses if efficacy is suboptimal. Part 3 is a safety extension. Currently, 15 sites are active in the US and 3 sites are active in Europe.

Slide 32 summarizes the atopic dermatitis study. Again, there are the same 4 arms but this time we've eliminated some of the re-randomizations. The primary endpoint is also at Week 16 and there are 18 US sites and 15 European sites that are active.

Slide 33 summarizes major events for the atopic dermatitis study consisting of a futility analysis in the 2nd quarter of 2024, an interim analysis for the primary endpoint in 4th quarter of 2024 and study completion in 2nd quarter of 2025.

That's my last slide. At this point I would like to hand you over to my colleague, Vikram, who will walk you through some very nice preclinical data with one of our up and coming drugs.

Vikram Ramanathan: Thank you, Siu, and good evening everyone. My name is Vikram Ramanathan and I oversee Preclinical Development at SPARC, and in the next few minutes I will be giving you an update on our compound SCD-153 which is being developed for Alopecia Areata. We are happy to share that the IND was filed

in India recently, and we have now received approval from DCGI to conduct Phase 1 clinical studies.

Slide 35 Alopecia Areata is a disease where the body's own immune system attacks the hair follicles and thereby causes hair loss. There is a large patient population that suffers from the disease and current treatments are inadequate. JAK inhibitors have recently been approved but they carry an FDA black box warning for cancer, stroke, and death. The other alternative is steroids but these are also not preferred because they require intradermal injections and have serious side effects. The pictures show how the disease manifests. It disproportionately afflicts the younger population <30 years of age and causes them significant psychological stress. There is clearly an unmet medical need.

Slide 36 gives some background on the compound SCD-153 which is being developed as a topical therapy for Alopecia areata. On the left is shown a healthy hair follicle. The hair follicle has so called immune-privilege which means that it is shielded from the immune cells in the vicinity. The AA diseased follicle is represented on the right. In this case, the immune privilege is lost and there are now disease-causing T-cells present at the base of the follicle, in what is called a swarm of bees phenomenon. These T cells secrete inflammatory cytokines and damage the follicle and the so hair falls out. SCD-153 inhibits this inflammatory process and can counter the disease. Importantly SCD-153 has been designed as a topical agent, and so systemic exposure and systemic side effects would be minimized.

Slide 37 shows preclinical data in an immune cell driven mouse model of AA which spontaneously develops hair-loss. The disease here is caused by attack by the CD8+ cytotoxic T cells which is also the culprit in the human disease. The graph on the left shows the Hair growth index on the Y axis and time of treatment of SCD-153 on the X-axis. Applying SCD-153 on the skin three-times-a-week results in the animals recovering hair growth compared to the vehicle treated animals. We also studied other dose strengths and

regimens of SCD-153, and have seen meaningful hair growth in those cases also.

The hair growth can be visualized in the photographs. The hair growth index is derived from software-based analysis of photographs. The animals start out with similar level of disease at Week 0. The animals in the top row on the right received vehicle for 16 Weeks and showed continued disease and no hair growth. Animals in the bottom row on the right received SCD-153 applied to the skin for 16 Weeks and show good hair growth. We have also examined the skin samples under a microscope and found that the disease-causing T-cells are reduced after SCD-153 treatment. So, these data demonstrate good single agent therapy. Importantly, there is room now to also look combination therapies with SCD-153 for hair growth, and we have initiated combination studies with low dose JAK inhibitors.

Slide 38 shows quantitative PCR based measurement of gene expression of inflammatory markers in skin of the AA diseased mouse skin. These are the interferon gene signature associated with AA in humans. The Y-axis shows the relative fold increase in the mRNA gene transcript levels above the baseline. In each set, the first bar is the level in healthy mice, they are low as they lack disease. The second black bar in each case is the fold-increase in the diseased mice and the difference between the first and second bar is the disease-associated change. In each set, the last two bars are from the diseased mice that have received SCD-153 treatment on the skin, and a reduction in the transcript levels of these genes is evident. These data provide a mechanistic basis for how SCD-153 causes hair growth in this disease model. The markers shown here attract the disease-causing T cells to the site, and these then initiate the disease. We should particularly note the increase in the chemokine CXCL9, 10 and 11 in this slide; and we will come back to the same CXCL9, 10 and 11 after a couple of slides.

Slide 39 summarizes the current status. Following Toxicology and Safety Pharmacology testing the IND was recently filed with the DCGI. We now

have approval for the First-in-human Phase 1 study. The Phase 1 study will be initiated in India in Q4 of 2023. The Single ascending dose study will be a randomized double-blind vehicle-controlled study and as shown in the figure we will have 5 ascending dose cohorts. As usual, there will be inhouse assessments up to 48 h post dose and subsequent follow up. The primary objective is safety and tolerability and plasma PK of SCD-153 will also be studied. This will be followed by multiple ascending dose study.

Slide 40 is the last slide on SCD-153. We are now evaluating the potential for SCD-153 in another autoimmune skin disease: vitiligo. Vitiligo is a condition where skin pigment is lost, and white patches develop. The chemokines CXCL9, 10 and 11 which we discussed just a couple of minutes ago, also attract the vitiligo-causing immune T cells to the site in the epidermis. These then secrete inflammatory cytokines that target and destroy the melanocytes which are the pigment cells. Inhibition of this cycle by SCD-153 suggests that the drug has potential in this disease as well. Vitiligo also causes a large psychological burden for the patient and available options are few. So, we are in the process of testing this possibility in preclinical models.

To recap, we have shown that topically applied SCD-153 promotes hair growth in a spontaneous immune mouse model of Alopecia Areata, and SCD-153 inhibits inflammatory cytokine gene expression levels in the underlying skin of these mice. An IND has been filed with the DCGI, and the Phase 1 study will soon commence in India with results due by the middle of CY2024.

I will now hand over to my colleague Dr Nitin Damle who will share an update on our work in the Anti-body drug conjugates area. Nitin…

Dr. Nitin Damle: A very good afternoon to you all. I am Nitin Damle and I oversee the Biologics function at SPARC. I will be providing update on SPARC's first antibody-drug conjugate, SBO-154. Antibody-drug conjugate (ADC) field has exploded in the last 6 years during which 11 novel ADCs were approved by the US FDA as meaningful treatment options for cancer patients. As a result, the ADC

strategy has blossomed into a commercially viable therapeutic strategy for strategic investments and the ADC market is projected to cross $25 billion in sales by 2028, with 24% increase in the compound annual growth rate during the next 5 years as shown on the slide 42.

During the last investors' call, we had introduced SBO-154 as the first biologic therapeutic from SPARC and described preclinically its antitumor therapeutic potential. SBO-154 is a humanized antibody drug conjugate of microtubule disrupting cytotoxic payload capable of potently inhibiting growth of actively dividing tumor cells. SBO-154 is designed to bind with high affinity to a broadly expressed tumor-associated antigen, MUC1. The cytotoxic payload and its linker, both have been clinically validated and are represented in 5 already marketed ADCs.

MUC1, the tumor associated antigen target, is synthesized as a single protein that undergoes autocatalytic proteolysis into MUC1 alpha and MUC1 beta prior to its display on the cell surface as a heterodimer of MUC1 alpha and MUC1 beta subunits. While MUC1 alpha is entirely extracellular in its display, it is tightly but noncovalently bound to the transmembrane MUC1 beta subunit that holds the entire MUC1 protein on the cell surface. The region in MUC1 where MUC1 alpha binds to MUC1 beta is recognized as the SEA domain as shown in the MUC1 cartoon on slide 43. MUC1 alpha subunit includes a variable number of tandem repeats (VNTR) of a 20 amino acid long peptide sequence against which a large number of monoclonal antibodies had been made and evaluated as naked antibodies, radio-immunotherapeutics or ADCs during the past 25 years or so.

None of these therapeutics managed to provide meaningful clinical benefit in cancer patients, in a large part believed to be due to the presence of cell-free or shed MUC1 alpha antigen both in circulation and also in the intratumoral compartment where it could effectively compete with MUC1 antigen on the tumor cell surface, intercept the anti-MUC1 antibody therapeutics, and thus denying them the opportunity to bind to tumor-

associated MUC1 to cause therapeutic activity. Our ADC, SBO-154, is a high affinity binder to the cell membrane proximal SEA domain of MUC1, and shown as circled in the MUC1 cartoon on slide 43. Unlike MUC1 alpha domain containing VNTRs, the SEA domain of MUC1 is not actively shed or shed in very small quantities so as not be able to compete with cellular MUC1 SEA and intercept the MUC1 SEA-targeted therapeutic entities such as SBO-154. We have further confirmed that sera from cancer patients show minimal presence of MUC1 SEA while continuing to show the sizable presence of MUC1 VNTR antigen. Hence the shed MUC1 SEA is far less likely to interfere with the binding of SBO-154 to MUC1 expressing cancer cells.

The next slide, slide 44, shows the relative strength of antitumor activity of SBO-154 evaluated against large xenografts of human carcinomas expressing different levels of MUC1 SEA on their cell surface. Whenever the expression of MUC1 SEA is high, SBO-154 is able to cause regression of pre-existing tumor xenografts. On the other hand, low MUC1 SEA expressing xenografts, although showed appreciable growth inhibition or disease stabilization, these xenografts failed to regress upon treatment with SBO-154. In contrast, an isotype-matched nonbinding control ADC of CD20-specific rituximab failed to show any growth inhibition in these evaluations. Thus, the antitumor benefit conferred by SBO-154 is preferential towards high MUC1 SEA antigen expressing tumors.

Our own immunohistochemical assessment of the expression of MUC1 SEA in patient-derived tumor biopsies and human tumor tissue microarrays suggests high level expression of MUC1 SEA in a wide variety of carcinomas, and thus, given opportunity, SBO-154 may be able to provide meaningful therapeutic activity in cancers with high expression of MUC1 SEA.

The SBO-154 program is currently in the preclinical development phase with the focus on manufacturing of the targeting antibody and the payload linker, and further the assembly of the ADC, SBO-154. We hope file the IND with the US FDA in the second half of calendar year 2024. While we

advance this program through its preclinical development, as shown on Slide 45, we have engaged the US FDA via the INTERACT meeting to seek guidance for various IND-enabling preclinical development activities as a prelude to the formal Pre-IND meeting in 2024 ahead of the IND submission. We are looking forward to the FDA response sometime before the end of this month.

I shall now hand over the discussion to SPARC's CFO, Chetan Rajpara. Chetan…….

Chetan Rajpara: Thank you, Dr. Damle.

Good evening, everyone. This is Chetan Rajpara, CFO at SPARC.

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

Slide No. 47 please…

During FY23, Total Income was at Rs. 250 Cr (USD 31.1 MN), while Total Expenses were at Rs. 472 Cr (USD 58.8 MN), resulting in to a Net Loss of Rs. 223 Cr (USD 27.7 MN). FY23 income was higher compared to FY22 on account of upfront and milestone payments received for out-licensing SEZABY and higher royalties on certain products.

Let me update you on our financial results for the first quarter of FY24.

For Q1-FY24, Total Income was at Rs. 34 Cr (USD 4.2 MN), while Total Expenses were at Rs. 129 Cr (USD 15.8 MN), resulting in to a Net Loss of Rs. 95 Cr (USD 11.6 MN).

Slide no. 48 please…

As you may be aware, the Company has raised Rs. 1,112 Cr (~USD 148 MN) in July 2021, by way of a preferential issue of convertible warrants.

The Company has received Rs. 703 Cr (~USD 93 MN) in Jan-2023 against the conversion of all warrants by investors. With this, the entire proceed of the Preferential Issue (i.e. Rs. 1,112 Cr) stands received.

Cash and cash equivalents as at September 30, 2023 was Rs. 363 Cr (USD 44MN).

The Company has sanctioned bank facilities for Rs. 175 Cr (~USD 21 MN) in place, in addition to a line of credit for Rs. 250 Cr (~USD 30 MN) from the parent company. Utilization of limits as at September 30, 2023 is NIL.

The Company has obtained shareholders' approval at the last AGM for raising a sum up to Rs. 1,800 Cr (~USD 220 MN) by way of fresh issuance of securities.

For FY24, approx. 30% of the expenses are budgeted for the clinical costs. We are aggressively managing our costs and working to control 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, we will now open the call for Q&A.

Moderator: We'll now begin the question-and-answer session. The first question is from the line of Ketan Gandhi from Gandhi Securities. Please go ahead.

Ketan Gandhi: So, on Slide 9, can you please help us understand Vodabatinib in CML recalibrating to a changing regulatory and market landscape?

Anil Raghavan: I think what this refers to is a couple of significant changes in the way FDA is reviewing clinical programs in oncology. I mean, if you go back to the prevailing paradigm in oncology for many years, the first registration window for a program is usually the last line setting. You have to basically test a compound in the last line setting. FDA is now for many classes actively encouraging sponsors to go to earlier lines through comparative trials as against an open label study in the last line setting. And this initiative is called the project "Frontrunner." So that is one important regulatory change which has consequences or implications for our program. The second significant paradigm shift is the way in which the agency looks at the acceptable doses. In the earlier paradigm, FDA always allowed, going up to the maximum tolerated dose and then coming down a step from maximum tolerated dose and using that as a dose for clinical exploration of the program. But now they're looking for at least some randomized data to identify the minimally efficacious dose. And this is coming from recent experience across several classes where approved products then manifesting significant level of toxicity

in clinical practice. So, both these as implications for us in terms of the path that we are following based on earlier set of recommendations on the agency in a prior consultation and what the agency would like us to do at this point and that is the reset that we are alluding to.

Ketan Gandhi: So, to understand it better, it will cost us more and it will cost us more time also, is my understanding, right?

Anil Raghavan: Let me take a step back. See what happens is two-fold. One, the current expectation for a registrational program in Vodabatinib is completing a last line study. And that last line study may require fewer patients, but they are far more difficult to recruit patients. The earlier line study would require more patients, but may be easier to recruit cohort. As I said in my comments, we are evaluating the cost and timeline implications and that will become a decision point post PROSEEK. Once we have results from PROSEEK whether we will pursue CML, is a strategic decision that we need to take and we will also decide whether we pursue this alone as ourselves or in partnership. I also want to highlight one other aspect of this change, that is, this will be a comparative study as in there will be an active comparator in this program as against an open label study where there is no comparator. So, in that sense, there is an opportunity for the cost to go up for sure. The timeline implications will need to be assessed based on the design.

Moderator: Next question is from the line of Ishita Jain from Ashika Stock Broking. Please go ahead.

Ishita Jain: So, my first question is on the PROSEEK trial. In terms of the primary endpoint, we're measuring change in baseline on the MDS UPDRS scale. Can you quantify that? How many points of change would take us successfully to the finish line?

Anil Raghavan: I think Dr. Siu-Long Yao can give a little bit more detail on the MDS UPDRS part-3. But, if the question is about the design of the trial and what would be a meaningful change from a clinical perspective, the study was designed

against the natural history data that we have from multiple natural history studies, particularly the MDS and Michael J. Fox Foundation study. So, in that setting, if you take part-2 plus part-3 together, it's a 6.5-point deterioration over a nine-month period, which is the baseline deterioration that we assume, and the 35% improvement of over that is deemed clinically significant. And that is proportionately split between part-2 and part-3 even though a larger component of that is part-2. So, in that sense from points perspective, four plus points in part-3 and around two in part-2. So, we still look for a 30% or 35% improvement on that trajectory, which is from our consultations with KOL deemed clinically significant if that's the question that you are asking.

Ishita Jain: Yes, absolutely. Thank you. My second question is slightly a broad-based question on PROSEEK. Inhibikase Therapeutics for their drug have unblinded a part of their trial two weeks ago and there seems to be signs of efficacy. I think only for the highest dose 200 mg and they also got orphan drug designation for MSA which you know is also an alpha synucleopathy. So, I do understand that you perhaps cannot comment on a competitor, but strictly from a proof-of-concept point of view, can you draw some parallels with our c-Abl inhibitor, Vodobatinib?

Anil Raghavan: I mean, they clearly claim a positive trend line for their study especially in the higher dose and that augurs well for our program. But we will take that with not just a pinch of salt, with a lot of apprehension because the sample sizes there are very, very low, I mean, this is 11 patients data and if you look at the duration of treatment, it is a 12-week duration and it is a known fact in the Parkinson's phase that the placebo response tapers only after six months, right. So, we expected to see some level of moderation of the trajectory in that earlier phase. So, we think that the overall treatment window and the patient size is inadequate to make the claims. And also, the lower dose did not have an effect, but that is to be expected to a certain level because the blood brain penetration requires substantial peripheral concentration. So, in

that sense, that's reasonable. But if you look at the window for treatment and also the number of patients involved, I think it's too early to comment.

Ishita Jain: So perhaps a little far-fetched. Post our Phase-2 read out, what are we thinking in terms of Phase-3, how big would Phase-3 be? We have about 300 crores cash on books. Will there also be an agency meeting before we initiate protocol for Phase-3, and finally, will Phase-3 include L-Dopa or MAO inhibitors patients as well, which is slightly more advanced stages of PD or are we sticking to early PD?

Anil Raghavan: Our registration program for the initial indication will not have the baseline L-Dopa. We are allowing MAO-B in the protocol currently. We will have an end of Phase-2 discussion sometime in November 2024 timeframe based on our current projections. And in the end of Phase-2 meeting we will be going in with multiple possibilities, some of them are really positive and some of them in the worst case, we may require two more Phase-3s to get this into a registration depending on the data that we see and depending on the view the agency takes. But our final sign-off on our registrational study design can happen only after we have a consultation with the agencies, with the full data readout in August 2024. But we will not be going into additional settings because if we introduce L-Dopa as a background therapy into this, that induces a significant level of variability of response. That is certainly an avenue for us to explore and pursue at an appropriate time because if we are bending the trajectory for the disease, we will definitely have an impact on later stages of disease, but that's a different program and our most important priority would be to replicate what we've done in PROSEEK and get this product to registration as soon as possible.

Ishita Jain: If you can just give an update on Phase-2 for Lewy body dementia?

Anil Raghavan: Lewy body dementia program is a single center trial out of Georgetown. That's a very small trial, some of the issues that I spoke earlier include that the treatment period is small and also very few numbers of patients. And we are looking at that trial for biomarker guidance and we will have data around the

same time as PROSEEK, Georgetown had shut down their clinical trial activity for due to COVID. So, there were significant delays in that program. But from a proof-of-concept standpoint even for lewy body dementia, we think PROSEEK is a more relevant proof-of-concept because of both the mechanisms involved in both diseases and also a number of patients involved in PROSEEK trial. So, we will consider PROSEEK readout as a trigger point or a stage gate for deciding on LBD.

Moderator: Next question is from the line of Chandpal Singh, individual investor. Please go ahead.

Chandpal Singh: I have just first one question regarding Vodobatinib for Parkinson's. Are we eligible for the disease modifying therapy drug?

Anil Raghavan: Disease modifying therapy designation in the label is one thing. And actual disease modification from a clinical experience standpoint is different. Let me comment on both. So, disease modification or disease modifying therapy designation on the label may require a different design, in the sense that you may probably need to have a delayed start design which will further delay the program. So, that is something which we may not explore on day one and we may try to add that later and we will also have a consultation with the agency in terms of what we are seeing. If you look at the nature of data that we will have both in terms of the actual trajectory of deterioration and also the time to introduction of symptomatic therapy will give practical data and guidance on whether the drug is modifying with disease trajectory? So, we hope to have a conversation with the agency in terms of disease modifying nature of the program and make the case for including disease modification designation in our label with this design. But if FDA has different views on that, then we will go ahead with the registration as a therapy in this disease before we pursue the disease modification.

Moderator: Next question is from the line of Jigar Valia from OHM Group. Please go ahead.

Jigar Valia: My question pertains to Vodobatinib. We have these catalyst events coming up in Q1, Q3. Just a clarification that this certainly would not affect any optionalities on LBD or even further on CML?

Anil Raghavan: I didn't fully follow the question. Can you repeat the question, please?

Jigar Valia: So, with regards to the catalyst events coming up for Vodobatinib for PD, that shouldn't affect the optionalities with regards to the LBD indications or even with regards to the CML which is again with regards to same modality?

Anil Raghavan: Well, it will, I mean, going back to my earlier response to Ishita, MSA and LBD essentially will depend on a successful completion of PROSEEK, in the sense that we are looking at PROSEEK as a definitive proof-of-concept for diseases driven by alpha synuclein. So, if there is a negative readout on PROSEEK, then it is a negative proof-of-concept for not only Parkinson's disease, but also for the mechanism more broadly, and that means across these diseases, like MSA and lewy body dementia. It is not going to affect CML because in CML we already have clinical proof-of-concept as Siu explained in his slide deck. And we see CML as a hedge even though a lower value hedge against the failure of the neurodegeneration hypothesis.

Jigar Valia: Also, similarly with regards to SCD-044, would psoriasis or atopic dermatitis be either/or, scenarios as a hedge for dermatitis?

Anil Raghavan: No, we will be driven by the data from their Phase-2 programs and as Sun Pharma is our commercialization partner Sun has the rights to take those decisions. And I'm pretty sure they will take appropriate decisions based on the data that they're seeing in these programs and the competitive landscape of these areas. So, I don't think we can give a generic response about whether it is going to be either/or at this point without actually seeing the outcome from the Phase-2 programs which will be coming soon.

Jigar Valia: Lastly, in terms of the incremental one run rate for the coming year, $60 million could be more?

Anil Raghavan: So, you see, next year calendar year '24 or Financial Year ‘25 will be a true discontinuity from a trending standpoint for the burn rate because we have been having a consistent burn rate for the last few years we've been pursuing, in a certain number of clinical programs and there is a certain level of predictability to that spend. But depending on what we do with Vodobatinib and depending on how some of these early program’s scale and also depending on the partnering strategy in terms of how much development is retained within SPARC. There is a potential for bigger swing in development spend next year. So, unfortunately, I'm not in a position to give you a trend guidance on operating costs for next year, because we are going to have significant dependence and variability on some of the data readouts.

Jigar Valia: Any timelines for the site transfer for Elepsia, Xelpros?

Anil Raghavan: In the case of Elepsia, we're looking for a site transfer in the next financial year. Xelpros hasn't gone out of supply at this point. So, we are still waiting for an actual plan from Sun in terms of transitioning that to a different plant. So, we don't have definitive guidance on Xelpros. But in Elepsia, we are looking at as in Financial Year ‘25 as a target for tech transfer.

Moderator: Next question is from the line of Manish Jain from GormalOne. Please go ahead.

Manish Jain: Really delighted to see the kind of biologics capabilities that have been built up, and ADC already really gunning for IND. So, I would say hats off to the team and I had three questions. The first one was, can't we pursue Vodobatinib both for PD and CML if we license it to two separate people altogether?

Anil Raghavan: This is an interesting question in terms of pursuing PD and CML together. There are two or three complicating factors here. One is obviously the commercial landscape and the price implications. If you take late-stage cancer and the price ranges usually runs to several hundred thousands of dollars per year per patient. But Parkinson's and the neurodegenerative diseases

spectrum being significantly large disease burden with significantly larger number of patients under management, per patient, per year pricing tends to be significantly lower. So, if you have a product even at a high dose, as in the Parkinson's dose is going to be higher than the CML dose with much lower price hence the substitution risk for someone who is commercializing CML is quite high. Two, there are safety profile implications, in the sense that patients who are going to come to CML with this drug are significantly sicker than patients who you see in early-stage Parkinson's. So, a lot of commercial partners will have trouble reconciling the fact that they have to deal with the complex safety profile, which carries some of the encumbrances of late-stage disease in cancer in trying to market a drug in early-stage Parkinson's disease and in competing with programs which are not really encumbered with that kind of issues in leukemia. So, there are some practical issues in terms of doing it both in terms of the cannibalization risk and also safety profile.

Manish Jain: My second question was pertaining to SEZABY that is Phenobarbital. So, where are we going to manufacture it?

Anil Raghavan: So, we are in the process of inducting one more manufacturing plant. Currently, it's supplied out of one of the Sun's facilities. And the second facility our partner is now inducting is a third-party outside of the Sun network. So, as we work towards enforcing market exclusivity, we will have two sources one from the Sun network and one from outside.

Manish Jain: Related to PDP-716, in terms of alternate API partner in how much time do we think before we can respond to the CRL?

Anil Raghavan: So, we have already identified an alternative manufacturing partner and we are in the process of initiating the exhibit batches with alternative partners' API. It would require significant stability data which is from a timeline standpoint, and we expect it to happen towards the end of the second half of next financial year.

Moderator: Next question is from the line of Ishita Jain from Ashika Stock Broking. Please go ahead.

Ishita Jain: So, moving away from PROSEEK, on PDP-716, the one with Visiox, I think that you already answered the timeline, so Visiox also in-licensed Omlonti, which is for the same indication as PDP-716. And since SPARC is a shareholder of Visiox, can you give some color on how has the ramp up or the scale up been from Omlonti?

Anil Raghavan: No, we cannot comment on that program in this call, I mean, we would like to stay with the SPARC program, not qualified or equipped to comment on Visiox or other programs, sorry, Ishita.

Ishita Jain: On Vibolizimod which is with Sun… I have a feeling you won't be able to answer this question either, but can you give us some color to better understand the terms of contract, so we can understand what is the payout especially in calendar year 2024, which is we're expecting a clinical proof-of-concept?

Anil Raghavan: As you anticipated, we are not in a position to say. We haven't disclosed those terms. What I can say at this point is, the read out is a milestone event for us, and we also have substantial royalties expected from this program once we cross data thresholds in Phase-3 into a registration.

Moderator: Next follow up question is from the line of Manish Jain from GormalOne. Please go ahead.

Manish Jain: On the ADCs, we have highlighted one or two indications. At what stage post IND can we look at running three, four or five, six multiple indications?

Anil Raghavan: I think the early-stage dose escalations in a program like antibody drug conjugates would have patients from multiple indications. So, we are clearly looking ahead here, because these are matters which are under discussion and finalization at this point. But we will look for certain threshold of expression of MUC-1. We will have a cut off range and we think that we can

get patients from across three, four tumor types, you have possibly breast cancer, lung cancer and a few other. And there is a possibility to do this more as a basket trial, as against going up in one program or one entity.

Moderator: Next follow up question is from the line of Ishita Jain from Ashika Stock Broking. Please go ahead.

Ishita Jain: So just one last question on PROSEEK. I think I missed asking this. We have had six DSMB meetings. Were they all 100% green light to go ahead on both dosages?

Anil Raghavan: Yes, we specifically asked if they would like us to change anything in terms of the doses used or the design or inclusion, exclusion criteria. They didn't want to change anything about the design or the dosages used.

Ishita Jain: I also noticed that I think last month we set up a wholly owned subsidiary called SPARC Life in the US. Can you comment on the goal to create this, I mean apart from monitoring clinical trials in the US, can this subsidiary someday become somewhat of a front end?

Anil Raghavan: I don't want to get ahead of myself here. I mean, we have an operation in the US, including Dr. Siu and others who spearhead our clinical activity. And I think from a regulatory and governance standpoint, it's cleaner to have them in a subsidiary. And we also have a branch structure. At the moment, it is a part of a cleanup process, but that would become an operating entity for us in the US and if we scale into a late-stage development program for Vodobatinib that would become meaningful arm for us.

Moderator: Next question is from the line of Tushar Bohra from MK Ventures. Please go ahead.

Tushar Bohra: Sir, this is regarding the PROSEEK outcomes. We mentioned that we saw potential collaborations or outlicensing opportunities or something on those lines. If you can highlight a bit more details who are the players we are

approaching, what kind of interest we are seeing, anything around that will be useful?

Anil Raghavan: I won't be able to disclose who are the players that we are talking to, because we are governed by fairly strict confidentiality commitments on these conversations. But, what I can say is that right through this Phase-2 program, there have been consistent interest from larger pharmaceutical companies and we will initiate a process as we go towards data flow from this program to see what kind of partnership structures are possible, and we will also explore what is probably a smart way to split the value between us and a potential partner depending on where we actually land with the data. But I'm not able to give you a specific answer in terms of names that you're looking for.

Tushar Bohra: I was looking for number of players and the kind of players we are discussing with, what kind of companies, whether you -?

Anil Raghavan: So, we are looking for at large commercial players, I mean, bigger pharma companies with the commercial footprint in the CNS space or the larger companies with strategic intent in CNS space and we have a fairly significant field which is interested.

Tushar Bohra: Second, if we were to proceed to a full-fledged Phase-3 clinical trial assuming that we need one more trial for this after PROSEEK, is there an estimate as to how much resources we would need, and what could be the timelines possibly for this?

Anil Raghavan: So, it clearly a function of what is the extent of pursuit that we can have, right, and what is the regulatory expectation. If we're going to have one more clinical program, then we are talking about something in the range of $50, $60 million to do that one more clinical trial. And if we need to have two more, then we are talking in the range of something in excess of $100 million. But these are all very exploratory numbers. It's a larger question and probably a driver for collaboration is pursuing this program more fully, I mean we have

gone to some length to describe some of the possibilities both in late phase disease and also through prodromal disease, all the possibilities in the next ring of opportunity which is MSA and things like lewy body dementia. So, if you treat PROSEEK as a proof-of-concept for synucleopathy, then how aggressively you pursue this both the inner core of opportunity and also the peripheral set of opportunities is going to determine what is the kind of partnership or resourcing that is required and that will be a major consideration going into.

Tushar Bohra: You mentioned that in the most conservative case, we could maybe need two more trials, but what about the most optimistic scenario given the safety profile of Vodobatinib which is clearly at least as per the data upto now, clearly very, very strong as well as the efficacy part, what is the most optimistic scenario that you guys are hoping for or are building in as one of the probabilistic scenarios?

Anil Raghavan: I think we will be getting to a speculative realm at that point. I don't want to get into that issue with this group, because at this point, we did not have any such conversation with the agency about the future of this program. So, anything that I say is going to be based on my read of what is possible and what is not possible and that could be misleading. I don't want to be misled by what I can say at this point.

Tushar Bohra: Maybe I'll rephrase the question. What are the possible scenarios after PROSEEK depending on the data, what are the pathways available from a regulatory standpoint and whichever one may happen?

Anil Raghavan: See, PROSEEK is an unusually large Phase-2 program. You have 504 evaluable patients. You have clinical endpoint which is what FDA has advocated for this disease indication for a long time and we will also have some significant biomarker data depending on the nature of the samples that like CSF or biopsies or blood and we will have fairly large numbers in some cases and smaller numbers in some cases. So, this is a substantial data set. How FDA would actually look at this and how FDA would look at the disease is to be

seen, because we don't have too many precedents in Parkinson's disease for neuroprotective therapies coming to a registrational path. FDA's CNS division has been fairly progressive in dealing with indications like Alzheimer's and ALS. If you want to look for the most optimistic scenario, my guidance would be to take a look at what happened in ALS and in AD recently. There are three approvals in AD and ALS. In the most optimistic scenario that can become a guidance benchmark for us with the agency. But I'm not saying that because there is no precedent and the contours of this disease is somewhat different to like ALS and AD. So, I think any speculation on what is possible is of limited value because of these differences in my mind.

Tushar Bohra: If you can highlight some of the newer drugs that you are either looking to in license or are at early stages of development. You said in the beginning of the call that you're looking at multiple modalities extending into newer modalities. If you can just highlight a little bit more around that?

Anil Raghavan: If I look at our trajectory, we have a historical focus in smaller molecules, and we have added biologics as a competency in the last two-three years. And there are you know different possibilities with that broader area, antibodies drug conjugates which has traditional cytotoxin, which is essentially much of the ADC field at this point. But we think that ADC will shift substantially with other kinds of payloads like other targeted therapies and even other modalities. I don't want to speculate there, but even things like RNA therapeutics are all possibilities coming from the ADC kind of constructs. Plus, you will see bispecific or multispecific structures, both bispecific or multispecific standalone antibodies plus bispecific or multispecific ADC constructs, try to improve target expression by adding in another specificity. So, there are different possibilities there. And clearly one other possibility given our strengths in small molecule is small molecule directed on cytotoxin ADC. And then you can use a small molecule of synthetic ligand to target specific cancer cells, and that's something which we are definitely doing, and we have a program in consultation with UCSF, I think one of the slides have

reference to that. We already have a program in fairly late-stage preclinical program, and we will have news flow from that program going forward.

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

Jaydeep Issrani: Thank you, Neerav, and thank you everyone for being on the call today. In case you have any additional questions, feel free to reach out to us. We'll be available to answer your questions over e-mail or through discussions. Thank you again for being on call today.

Anil Raghavan: Thank you very much.

Moderator: On behalf of SPARC, that concludes this conference. Thank you for joining us. You may now disconnect your lines.