SPARC/Sec/SE/2025-26/50 January 13, 2026

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

Scrip Symbol: SPARC

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

Scrip Code: 532872

Dear Sir/Madam,

Sub: Transcript of the Investor's call for SPARC R&D Day - Updates on Prioritized Programs

This is with reference to our Investor Call which was scheduled on January 08, 2026. Pursuant to Regulation 30 of the SEBI (Listing Obligations and Disclosure Requirements) Regulations, 2015, we hereby attached the Transcript of the said Investor Call for SPARC R&D Day – Updates on Prioritized Programs and the same is also available on the website of the Company on the weblink - https://sparc.life/presentations/

This is for your information and dissemination.

Yours faithfully,

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

Kajal Kiran Damania

Digitally signed by Kajal Kiran Damania
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Kajal Damania
Company Secretary and Compliance Officer

"Sun Pharma Advanced Research Company Limited (SPARC)

R&D Day Conference Call"

January 08, 2026

MANAGEMENT: **MR. ANIL RAGHAVAN – CHIEF EXECUTIVE OFFICER**
DR. MUDGAL KOTHEKAR – VICE PRESIDENT, CLINICAL DEVELOPMENT, IMMUNOLOGY
DR. SANDEEP INAMDAR – VICE PRESIDENT, CLINICAL DEVELOPMENT, ONCOLOGY
MR. JAYDEEP ISSRANI – HEAD, BUSINESS DEVELOPMENT, CORPORATE COMMUNICATION AND INVESTOR RELATIONS

Moderator: Ladies and gentlemen, good day and welcome to the R&D Day Conference Call hosted by Sun Pharma Advanced Research Company Limited, SPARC. 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 star then zero on your touch-tone phone.

I now hand the conference over to Mr. Jaydeep Issrani from SPARC. Thank you and over to you Mr. Jaydeep.

Jaydeep Issrani: Thank you, Ikra. Hello everyone, I am Jaydeep Issrani and I lead Business Development and Investor Relations here at SPARC. Thank you for taking the time to join us for the R&D Day presentation. We are excited to walk you through our strategy and the progress that we made across our pipeline programs. I am joined today by our CEO, Mr. Anil Raghavan and several members of our leadership team. If you have been on our previous calls, the format today will feel familiar.

We will walk you through the presentation first and then we will open the call for questions and comments. The deck was shared earlier today, hopefully all of you have had a chance to look at it. Before we get started, just a quick reminder, some of the things we will talk about today are forward-looking and as always come with their own risks and uncertainties. The actual results could differ from what we discussed on the call today.

With that, let me hand it over to Anil to start his presentation.

Anil Raghavan: Thank you, Jaydeep. Good evening, everybody. A very warm welcome to all of you to SPARC's R&D Day. Thank you for your time today. It means a lot to us. We will begin with a brief review of our portfolio strategy and operational priorities. In the initial part of our presentation today, we will revisit some of the expectations set in our last call. I will also touch upon several early stage and preclinical programs, which we don't intend to cover in a lot of detail.

In the second half, our therapeutic leads, Mudgal and Sandeep, will join us to share updates on two prioritized clinical programs that are now leading our portfolio. So with that, let's start with slide 4, please. If you remember, we discussed this chart last time we spoke.

Over the last 24 months or so, we had two significant data readouts which did not go the way we hoped. That's PROSEEK Phase 2 study in PD and Vibozilimod's Phase 2 trials. This slide summarizes the most important elements of our reset since then, built on three key pillars.

The most important one was to narrow our therapeutic focus, buckle down on oncology and immunology, and execute well on two promising programs that were nearing entry into clinic at that time. The first product was our MUC1 ADC in solid tumors and the second one was a topical intervention in Alopecia Areata. Since then, we have aggressively reshaped our pipeline around similar themes.

The second pillar here is centered on reducing the burden of clinical risk within SPARC and explore alternative structures like NewCos to develop certain parts of our work. We have made progress with our collaboration with Tiller Therapeutics, a company set up by a very accomplished UCSF team to focus on developing small molecule drug conjugates or SMDCs. And the last piece here focused on converting some of the short-term cash options we have along with a really sharp focus on optimizing our cost structure, both our fixed cost base and the operational cost supporting the execution. That's obviously super important given where we are and important to continue resourcing the aggressive build of the portfolio. We are entering the New Year with significant movement on the most important parts of this agenda, as you will see in the rest of the presentation.

Even though we have our fair share of challenges, we're genuinely excited about the potential of our clinical and preclinical portfolio as we go into the New Year. Especially on the reshaping of the early pipeline around a few big themes which we believe have the potential to reset those spaces in the future. But before we go there, I would like to give a little bit more color on some elements of this plan to help you understand the short-term better.

So next slide please. Mudgal and Sandeep will cover these programs in a lot more detail, but the key message that I want to leave you with is that we achieved all the operational milestones on both these programs ahead of schedule. For those of you who are new to SPARC, SCD-153 is a topical formulation of an itaconate analogue developed through a collaborative development partnership with Johns Hopkins.

Itaconate, as you may know, is an endogenous immunosuppressive metabolite, which is getting a whole lot of traction now across multiple autoimmune conditions. We completed the healthy volunteer component of the Phase 1 trials and started on the Phase 1B program in alopecia areata patients in India recently. And this is an early signal seeking study and we expect to have safety and preliminary efficacy data by the fourth quarter of this calendar year and transition the program to a, hopefully to a larger Phase 2 trial thereafter.

SBO-154 is our MUC1 target ADC delivering a toxin called MMAE. We filed INDs in the US, Australia, and India and got approvals from all three geographies and the Phase 1 dose escalation study in solid tumors is currently underway and recently completed the first two dose levels and now entering cohort three with doses which are beginning to approach the pharmacologically relevant dose.

We've prioritized these two programs for resource allocation through the last year and a half and are really happy to be ending the year with visibility on safety and early efficacy signals soon, maybe later this calendar year for one program and early next year for the next one. This not only begins unlocking the value of these programs, but also sets up some important adjacent programs across the pipeline.

Let's move to Slide 6, please. I'm sure most of you are familiar with the background of this program, which has two important elements in terms of value. First is our claim for a pediatric rare diseases voucher or PRV, which is a tradeable device that can help accelerate the NDA review of a non-priority review program to a six-month review cycle. And second is the market exclusivity, which we are eligible for because of the orphan drug status.

We got some good news on the PRV matter from the district court of District of Columbia, which ruled that the agency's decision to deny the voucher earlier

was contrary to law. This judgment comes with a two-month appeal window that is now, you know, getting to that expiry of that appeal window by end of Jan. Subject to a possible appeal, SPARC expects to receive a voucher, which can lead to a meaningful encashment.

The PRV market has been off late on an uptake, especially after the scheme got sunset an year back. The demand remained robust while the supply situation is beginning to shrink, resulting in transaction values going north of $100 million. We remain hopeful of a positive outcome, which would be quite significant for SPARC.

On the other end, we continue to make the case for enforcing the exclusivity. Our Citizen Petition is still under review, and the agency indicated that they need more time for resolving the CP, given the complexity of the matter. We will continue to press for the removal of the DESI phenobarbital formulations, especially given the presence of these toxic alcohol-based excipients, which are potentially impacting vulnerable populations. This is indicated for neonatal seizures. We remain hopeful.

Let's go to Slide 7, please. This is on PDP-716, which is our once-a-day formulation of Brimonidine. This product was licensed to an ophthalmic specialty pharma company called Visiox Pharma for commercialization. And Visiox was recently bought out by Ocuvex Therapeutics, and Ocuvex now holds the rights to this product. Our original NDA received a complete response letter, driven by the OAI status of the third-party API vendor.

Ocuvex recently completed the resubmission, as the API vendor's regulatory status changed. While the review process is still underway, we may not be completely out of the woods here, as the finished product manufacturing site has compliance issues which require further remediation. We are working with our partners, Ocuvex Therapeutics, and are in the process of qualifying third-party sites for the finished product manufacturing. We hope these steps will see us through to the market and the regulatory gate.

Now, let me give you an update on the NewCo we are setting up for progressing SCO-155. What is SCO-155? Slide 8 here, please. SCO-155 is a small-molecule drug conjugate, as I mentioned earlier, which leverages a synthetic ligand of PSMA, which is a key cell surface antigen significantly

over-expressed in prostate cancer. We have reviewed preclinical data of this asset in our last call, and that data set and subsequent experiments that we've done indicate MMAE gets significant tumor accumulation through PSMA-mediated internalization, and causes dose-dependent tumor kill.

Earlier this year, SPARC and UCSF signed a binding Letter of Intent with Tiller Therapeutics, a company formed by our scientific collaborators at UCSF. As part of this process, Tiller obtains exclusive global rights, and SPARC, in return, is eligible for significant equity of Tiller. Our teams made meaningful progress on the development plans last year, nearly completing the IND-enabling tox studies and making substantial strides towards manufacturing clinical-grade material.

Tiller has also completed the pre-IND consultation with the FDA Oncology Division, getting some clarity on regulatory expectations. They've raised pre-seed capital in last year, 2025, and now in the process of raising priced external seed capital for funding the early-stage clinical program. We're also very encouraged by how this whole NewCo structure has panned out for advancing a late preclinical program into clinic.

It can also be an interesting preposition for some late-stage programs, maybe even for vodobatinib in CML. So let's go to the next slide to talk a little bit more about vodobatinib.

Here, you know, certainly our diversification into Parkinson's disease for this compound took a toll. Not just the spend, which was quite significant, but also the opportunity cost in terms of lost time and pipeline position.

We tried to get back to CML as soon as PROSEEK's full results came in. But as we discussed earlier, we've negotiated a protocol for a registration program in second-line CML with FDA, but the pathway remains challenging. The CML landscape, as you can see in this chart, has been changing quite a bit with increased generalization of TKIs and the progression of second-generation and now third-generation set of products into earlier lines of treatment.

The standard of care and pipeline has evolved with asciminib and other allosteric inhibitors of BCR-ABL coming in. Asciminib is on the way to becoming a significant option for CML patients across the spectrum. While the development pipeline has expanded with new investigational products, all

having exciting early clinical data, like less than 100 patients, new products, including ours, need validation through larger pivotal trials to establish their true position in the pipeline and the therapeutic continuum.

At the same time, the licensing interest from large pharmaceutical companies has been limited, largely on account of perceived market-size limitations, even with Asciminib's commercial success and the rising industry interest in the next-generation TKI companies. So that is proving to be a moderating force on the outlook going forward.

This complex dynamic puts us, as in SPARC, in a bind as progress now requires resourcing a costlier late-stage study with an active comparator, like several others in the field are planning to do. We've been carefully gauging the implications of this emerging situation, especially with the best use of capital considerations in mind, given that we have several other pipeline opportunities.

We may prefer finding a way to work with potential partners and investors using alternative structures to continue pursuing marketing approval for this product, which we believe for sure can help CML patients significantly. We hope to take a final decision on the future of this program fairly soon. So let me leave you with the following thoughts from this segment.

SPARC has made significant effort to reposition its program around a pair of exciting assets, a differentiated antibody drug conjugate and novel immunology pathway in the derma autoimmune segment. As you will see later today, these projects have important catalysts coming up in the short term.

The whole SCO-155 experiment is also maturing and gives us confidence that such partnerships can be an alternative path for some of our assets. And a positive outcome on the PRV matter can be transformational and can give us room to explore our pipeline more aggressively, especially after the cost structure optimization that we achieved through the last year or so.

I'll touch upon some of the specifics of our cost reduction and funding situation in a bit. Before I do that, let's take a look at the preclinical side of our portfolio.

Over the last couple of years, we've been trying to move away from a broader, somewhat disparate set of projects, all chosen for individual attractiveness as a scientific hypothesis. Synergy was not a consideration that drove those

choices. But two things changed in the recent past. We exited neurodegeneration almost completely. That leaves us with oncology primarily and certain pockets of immunology. Secondly, we deliberately tried to bring in more thematic cohesion in our approach to portfolio building.

We are now focused on three tightly defined themes. We're interested in targeted delivery to tumors, either mediated by antibodies, internalized through cancer-specific antigens, or using synthetic small molecule ligands similarly. We're interested in different modalities of payload delivery using this approach. And a substantial chunk of our effort now, the preclinical effort now, is focused on that.

The second thread here is this broad area of synthetic lethality. Exploiting vulnerabilities like BRCA, loss of function mutations, or induced DNA damage, like in the case of radiation and chemotherapy. It's emerging as an important area of cancer research, and especially after the success of agents like PARP inhibitors. We are interested in multiple targets in this area.

And the last piece here, we see a clear opportunity in certain dermatology autoimmune disorders, where the current standards of care have significant liabilities, either long-term safety concerns, or in some cases, meddling efficacy. We are focused on exploring novel pathways and potentially rational combinations as topical alternatives to the current oral and I would say somewhat challenging set of options that these patients have.

We plan to maintain this focus in the medium term so that we can benefit from the mechanistic and operational synergies within a tightly defined field. I want to add a little bit more color to these three broad objectives to help you understand our specific interest in each of these areas.

Next slide, please. Our overarching strategy is to develop modular platforms with plug-and-play options. The targeted oncology space offers best examples of this approach. We have at least six platform concepts, which can all expand into multiproduct baskets.

The MUC1 SEA targeting classical ADCs with payloads like MMAE or exatecan is probably the most advanced, the lowest hanging fruit, followed by a range of options covering multiple payloads and structures like immune-stimulating agents such as STING agonists, T-cell engagers, bispecific ADCs,

SMDCs like SCO-155, which we just spoke about, and antibody-coated nanoparticles, which carry siRNA that can knock down target proteins. We have programs at various levels of maturity in each of these six areas.

The next thread, as I mentioned earlier, is synthetic lethality, where we are going after, one, a key driver of PARP resistance, and two, an important player in the repair of DNA damage, which is a significant liability for most cancers. And we spoke about the last block, which is our focus on dermatology autoimmune disorders.

It's primarily driven by CD8 positive T-cells, where the current standard of care is oral or topical JAK inhibitors. And we are looking to explore new biological insights to create safer topical alternatives for better efficacy, either for better efficacy through mechanistically independent combinations or just improving the safety profile.

Let me try to give you a few examples on each of these elements, starting with slide 13, with targeted oncology. Smart oncology therapeutics has had significant successes of late. Products like Enhertu and Trodelvy helped rediscover the ADC field, which is turning out to be one of the richest pipeline segments in oncology.

Novartis' PLUVICTO added RLTs, as in radioligand therapies, into the mix, leveraging synthetic small molecule ligands, like what we are doing in SCO-155. This momentum is built on the back of superior clinical data and driven by the bigger therapeutic window, enabling that superior clinical data. We believe that this wave is just the beginning, with significant activity expected from novel antigens, bispecific targeting, and novel payload classes and their combinations in a bifunctional manner.

Payload diversity is going to be the key, and we are actively testing several novel propositions. On the multispecifics, T-cell engagers are having a big impact on Haem-Onc. Their progress in solid tumors is somewhat constrained, largely because of cytokine release issues. Next generation in this class will look to include features that will enable tumor-specific T-cell activation and reduce the risk of CRS more.

We believe this will bring a segment of solid tumors into play, especially the ones showing activity against classical immuno-oncology products. We are

seeing evidence of this already in the development pipelines, where the ADCs and bispecific antibodies have now become the mainstay of many big pharma portfolios.

We are not very different in that regard. Let me walk you through a few examples.

As I mentioned earlier, ADCs targeting the MUC1-SEA domain containing the alpha-beta combinatorial epitopes are the most proximal platform opportunity for SPARC.

Once the targeting hypothesis is validated in the ongoing trial, we have the flexibility to plug in several payload options using the same MUC1 series antibodies. On the chart here, we have two such examples. On the left is our MUC1 MMAE ADC with, as you can see, significant tumor activity in colo-357 pancreatic cancer model.

And on the right, another ADC delivering a TOPO1 payload in the same model demonstrates dose-dependent tumor regression. It is important to point out the potential of this approach even in moderately expressing populations. With an H-Score of 130, this is the line with moderate expression of MUC1.

Later in the presentation, Sandeep will discuss the actual expression level in patients' tissues, and that's higher. We are looking forward to early signs from current studies on the MMAE ADC, as I indicated earlier, and we will proceed with the TOPO1 variant only after the MUC1 hypothesis is validated. We believe the diversity of expression profiles will allow us to position multiple payload classes with distinctiveness.

In the next slide, we have an example from another payload, an immune-stimulating agent. Slide 15. This is a whole new class of ADCs coming into the clinical pipeline now, where an intercellular immune-stimulating protein is activated by the payload to develop an immune response.

In parallel, the Fc component of these ADCs binds with Fc receptors of players in the innate immune system, such as macrophages, activating them. This multipronged approach provides an opportunity to convert cold tumors, hot and enabling combinations with classic IO agents like checkpoint blockers.

With several active players experimenting with this approach, including ADC class leaders like Daiichi Sankyo, providing early validation for this approach.

Next slide, has more specifics on our plan. We are using a proprietary STING agonist series as the immune-stimulating payload in our program. As many of you know, STING has been an active area of interest as a cancer target for some time, but the field couldn't make much progress because of the safety issues.

STING agonism, as you can imagine, can unleash broad immune response, which can be problematic. That's where ADCs come in handy through the specificity of antigen-mediated internalization and its ability to bridge macrophages into tumor microenvironment. We have excellent preclinical proof of concept for this approach, as you can see in the chart here.

In this experiment, we have administered three infusions of three different doses, 0.3, 1 and 3 mg/kg IV with the COLO-357 tumor bearing mice. These are T-cell deficient nude mice with active innate immune system, and we see dose-dependent regression, including several complete responses, even in the middle dose. We also tested mouse variants of these ADCs in fully immune-competent animals, again eliciting exciting tumor response without any significant safety issue.

We are at the moment finishing the preclinical efficacy battery, and we are exploring ways to bring them into clinic. Before I move on from this theme, I want to briefly talk about T-cell engagers and our plans in that area in Slide 17. Let me give a bit more background.

T-cell engagers as a class is probably more mature and validated than immune-stimulating ADCs, especially in hematological malignancies like lymphomas and myelomas. The chart on the right side gives a simplistic illustration of the way T-cell engagers are engineered and structures of three generations of T-cell engagers. Essentially, a TCE is a bispecific, combining a tumor-specific antibody with a CD3 binder or something similar.

First-generation products using this concept, like Amgen's BiTE platform, had two ScFvs joined together by a flexible linker. While they proved efficacious, they had circulating half-life issues, which led to the second-generation TCE, which is a fuller bispecific antibody structure with a functional Fc. That did

improve the PK properties, but aggravated the off-target cytokine release because of the Fc receptor binding to T-cells and bridging outside of the tumor.

The field tried to address this issue through mutated or partially incompetent Fcs, which partially took care of these issues. But we feel there is significant opportunity in solid tumors, provided the field comes up with constructs with sufficient safety margin to dose meaningfully higher. That's what SPARC is attempting to do.

In our first shot at this concept, we substituted the Fc component with human serum albumin, which lent similar half-life extension without the risk of Fc receptor binding and T-cell complexation. We have a promising proof of concept for this construct, with an illustrative asset built with MUC1 antibody as the targeting mAb and a CD3 binder and a full-length human serum albumin. We call this MUC1-SEA Albufusion TCE.

Let's go to the next slide to take a look at the data. See the waterfall plot on the right side. In this experiment, using our workhorse COLO-357 pancreatic line, we have tested the first construct using admixed PBMCs, which has a mix of immune cells and a nonspecific control TCE binding to, in this case, HepB.

The data cannot be clearer. Almost all animals responded, except one. 60% of animals had complete response. Encouraged by this data set, we are working on a second-generation construct, which will further improve the specificity and mitigate the CRS risk. We are making it trispecific now by adding a CD8 binder for preferential activation of the cytotoxic CD8 T-cells and reducing the CD4 helpers. CD4s are primarily the drivers of CRS, and we are confident we can proceed to IND-enabling studies next financial year with a differentiated asset using this design.

Our intent in this part of the presentation was to give you a glimpse into our focus on smart oncotherapeutics. I didn't talk about everything we have in the basket. A couple of them maybe deserve mention before we move on. We consider bispecific ADCs as a natural expansion of the ADC space. We are working on a couple of novel combinations using Exatecan as the payload.

Further down the line, antibody mediated delivery of nanoparticles containing siRNA as the payload is another area of active pursuit for us. So, SPARC, in the last couple of years, demonstrated the engineering capability to modularly

put together these kinds of differentiated structures. This effort is going to be a major driver for our portfolio expansion going into the future.

Now, let's move to our second pillar, which is synthetic lethality in Slide Number 20. Because of consistent DNA damage and genomic instability, cancer cells are, dependent on DNA damage repair pathways. This is especially true in tumors which are homologous recombination deficient, or HR deficient. HR is a crucial DNA process which is extremely critical to repair, and its deficiency creates an exploitable vulnerability which was successfully exploited by agents like PARP1 inhibitors in the BRCA context.

We are working on two opportunities in this space. One is a sensitization agent which shuts down a key player in DNA repair. Such an agent can be an excellent sensitizer to radiation and also to certain chemotherapeutic agents.

The second idea originated from the field of PARP inhibitor resistance. Our target of interest has emerged as a key compensatory mechanism in PARP1 resistance, and we are looking to leverage this as a PARPi alternative or as a PARPi sensitizer. In the next couple of slides, I want to very briefly comment on the early proof of concept data coming out of these programs.

21. As I said earlier, in the first program in this theme, we got what we consider as a master regulator of cellular damage repair, which helps cancer cells maintain genomic stability. This has been a target of interest for some time with multiple large pharmaceutical companies in the race.

The first generation programs failed to take off because of PK limitations. Adequate brain penetration was also an issue with GBM as a key opportunity for this approach. SPARC has developed now a series of brain penetrant high-potency inhibitors with good PK profile and has developed data along with multiple chemotherapeutic agents as shown in this chart.

We also have data with radiation. In all these experiments, SPARC compounds outperformed the leading second generation compound, which is going through early stage clinical trials now. We are in the process of finishing the preclinical efficacy trials and looking forward to transitioning the lead compound to clinic before the turn of the next financial year.

Now, on the second concept, let's go to Slide 22. This program targets a chromatin remodeller, which is a key player recruited into DNA damage repair process and significantly over-expressed in multiple tumors as cancer cells fight for stability. We have developed extensive genetic and pharmacological validation for this target internally within SPARC as this is a potentially first-in-class opportunity.

This protein has also emerged as an important compensatory mechanism as cancer cells become resistant to PARP inhibitors. As you can see in the chart below, our tool compounds exhibit excellent anti-tumor activity standalone and in combination with another PARP inhibitor, Olaparib, in this in-vivo model in a dose-dependent manner.

This early data is really relevant as it validates our chemistry and opens potential path to a nomination by mid-next year. We are looking to accelerate the IND process as soon as we have an optimized lead compound given the first-in-class window on this area.

That takes me to the last set of this three-leg stool, derma autoimmune disorders. Let's go to Slide 24 to briefly discuss our priorities in this area. Diseases like Alopecia Areata and Vitiligo has been managed with a fairly dormant standard of care for a long time, primarily corticosteroids and calcineurin inhibitors, which left significant room for growth, both in terms of efficacy and safety, in spite of the recent progress with JAKs.

As I mentioned earlier, this is an area where SPARC sees an opportunity in the short term. We made our first move with SCD-153 already. As we move forward, we are building other ideas which can be alternative standalone options as well as synergistic combination partners for SCD-153.

This can take two paths, either establishing SCD-153 as a safe chronic maintenance option after a combination induction or developing lower-dose combination regimen, which can push up the efficacy without some of the concerning safety risks of the current standard of care. At this point, we are yet to disclose these alternative mechanisms we are pursuing, but hope to do that in the near future.

With that, let me conclude with a summary of our portfolio and short-term catalyst. I also want to make a few comments on our cash burn before I

transition. Let's go to Slide Number 25 for a view of the development pipeline. I've already spoken about most of these, so let me not recite this again.

We have not included a few things which are pre-POC, but I've alluded to some of them also. In terms of things to watch out for in terms of value creation, two clinical programs are really important. We expect to add another vitiligo trial for 153 and bring the radio/ chemosensitizer to the clinic.

We also hope to see Tiller making progress with SCO-155 to an IND, hopefully in the short term. We will also be busy on the biologics side of the portfolio with additional assets expected to graduate to clinic in the short to medium term.

We will stay in these three broad thematic lanes I spoke to you about for further building on the story. Slide 26 has a snapshot of what to expect in the calendar year. Just a summary of expectations during the course of my comments today. Our overriding priority will be executing as well as we can to ensure that these milestones are delivered.

Let's go to Slide 27. We've been trying to optimize our cost structure very aggressively after PROSEEK without compromising our ability to deliver our critical priorities or diluting potentially differentiating competencies built over the years.

We certainly made progress on this front as you can see from this chart. The primary driver of our fixed cost is obviously manpower cost. During FY '24, we had 400 plus people across three different geographies. Next year, our headcount is expected to be around 250-ish. And the location distribution is also important.

We will reduce US footprint significantly, which is primarily built to run late-stage clinical trials. We're also in the process of reducing the number of lab centers from four to two. To enable this transition, we've tried to align our projected workload with the scale and competencies required and have taken a decision to outsource certain competencies which require maintenance of costly infrastructure and capabilities inside.

These changes as you can see has resulted in an annual fixed cost savings of about $10 million, which is super significant relative to a 50 million-ish annual

spend. Let's go to the slide -- our next slide. We also have reined in our operational spend considerably. From 31 million last year, we are projected to go down to 29 million this year, even though we plan to scale our clinical program significantly.

Our overall ops cost is expected to only increase marginally. This is the outcome of several interventions that we focused on in the last couple of years now. But let me highlight just one thing. We strategically started increasing the India component of our clinical development effort.

The local clinical research environment has improved quite a bit in the recent past and we believe India provides an excellent opportunity to conduct early signal-seeking studies at a very low cost of failure. And that can be an important differentiating driver of value for companies like SPARC.

But before I transition, I want to ensure you see this on balance. While there is significant promise, ours is an early-stage portfolio with key assets yet to have even early clinical proof of concept. So investors who want to review this portfolio need to approach this with probability-adjusted view to valuation.

Plus we've been funding our early development using promoter-backed debt, which is cumulatively 45 million-plus now as of Q2 FY '26. We need now a significant additional resources to deliver the outcomes discussed in this presentation and we are in the process of finalizing a funding plan to extend the cash window to FY '28 and hope to complete that process in the first half of this calendar year. Thanks again for listening in. I'll see you at the other end of the call for Q&A. I'm now transitioning the call to my colleague, Dr. Mudgal for the next leg of this presentation, which is an overview of SCD-153.

Mudgal Kothekar: Thank you, Anil. I'll present an update on the program SCD-153, which is a first-in-class agent currently being evaluated for alopecia areata. Next slide, please. SCD-153 is a novel topical agent that has the potential to be an alternative to JAK inhibitors in the treatment of several autoimmune dermatological disorders.

SPARC licensed the IP rights for this compound from JHU and IOCB. SCD-153 employs a novel approach of using an itaconate derivative to address the immune pathogenesis. The topical delivery of this compound provides a dual advantage of targeted delivery at the site of action and minimizing the risk of

systemic adverse events that are the major limitations of the currently approved therapies.

We have completed several preclinical studies in which SCD-153 demonstrated hair growth in the animal model of alopecia areata. The preclinical PK studies have also been completed and the toxicology and safety package is also completed. Our Phase 1 first-in-human study showed that SCD-153 was well-tolerated up to the highest dose that was evaluated in the study. The Phase 1B clinical trial in alopecia areata has been initiated and an interim readout from this study is expected in the Q4 of the calendar year 2026.

Next slide, please. This slide shows the overall design of the Phase 1B clinical trial that is currently ongoing in alopecia areata. This study is enrolling male and female alopecia areata patients with a SALT score between 25 and 90, which corresponds to the loss of hair from 25% to 90% of the scalp. This study will enroll a total of 70 patients, 15 at each dose level.

So, these four are the four increasing dose levels. So, 15 patients will be enrolled at each dose level and 10 additional patients at the top dose level. In each cohort at each dose level, patients will be randomly assigned in a 4:1 ratio to active or vehicle in sequential cohorts of four dose strengths of SCD-153.

Initially, in each cohort, patients will receive a single application of the assigned treatment on day 1, followed by safety and tolerability assessments up to day 8. Once the single dose is tolerated, patients will continue to receive once daily treatment of the drug. Patients initially randomized to SCD-153 will continue to receive the same drug for a total of 24 weeks and patients who are initially randomized to vehicle will be switched to the active drug for the next 12 weeks.

Patients will be enrolled in subsequent higher dose levels after the evaluation of safety data up to day 22 from the previous dose code by an independent DSMB. SCD-153 was applied as a topical solution in the first cohort and we have made a change and optimized the formulation to foam formulation, which is being evaluated cohort 2 onwards.

The study will assess the safety as well as efficacy of increasing doses of the drug and we will also evaluate Pharmacokinetics, both the systemic PK as well

as the local concentrations at the site of action and we also will be evaluating biomarkers in the study by means of transcriptomics and proteomics.

Next slide, please. This is an update on the current status of this study. So, while the cohort 1 was ongoing, we amended the protocol to change the formulation from solution to foam and also to revise the cutoff for the extent of hair loss to be eligible to participate in the study.

Additionally, in the initial approval, the CDSCO had put up a condition to submit the cohort 1 safety data to the CDSCO and initiate enrollment in cohort 2 only after a review and approval from the DCGI to initiate the cohort 1. This would have significantly impacted the overall study timeline.

Therefore, along with the protocol amendment, we requested a waiver for this condition and proposed to allow opening up of this subsequent cohort based on the review by an independent DSMB that was already a part of the study.

The protocol amendment as well as the request for the waiver was approved by the DCGI, saving us a lot of time in the study. The study has completed enrollment of all required 15 patients in the cohort 1. Nine of these 15 patients have already completed 12 weeks of treatment with no safety concerns.

The DSMB has reviewed the safety data from the cohort 1 and recommended opening up of the second cohort as no safety issues were found in the first cohort. Patients are currently being enrolled in the second cohort.

Moving on to the next slide. In terms of the key milestones, the enrollment in all cohorts of the study is targeted to be completed in the quarter 3 of the year 2026. Top line results are expected in Q4, and we plan to initiate a global Phase 2 clinical study in the quarter 2 of the calendar year 2027.

Next slide, please. We are also exploring this compound in an additional indication and another dermatological condition that shares the immune pathogenesis with alopecia areata. The image on the left illustrates the underlying mechanism involved in alopecia areata, and the image in the middle shows the mechanism involved in vitiligo.

As shown in these two cartoons, CD8+ T cells are the implicated cell type in both these conditions. As shown in the image on the left, CD8 T cells infiltrate around the base of the hair follicle in the dermis in alopecia areata. And as

shown in the middle figure, the CD8+ T cells localize near the melanocytes in the epidermis in vitiligo.

In both the diseases, the CD8+ T cells release the cytokines such as interferon gamma and their cytolytic granules. In alopecia areata, they damage the hair follicles, causing loss of hair. In vitiligo, they damage the melanocytes that produce melanin, resulting in depigmentation of the skin.

As shown in the image on the right side, both hair follicle stem cells and melanocyte stem cells are preserved in the bulge of the hair follicle in these conditions, creating a potential for clinical benefit in both these diseases.

Next slide, please. We have conducted in-vitro studies to explore the effect of SCD-153 on the chemokines and cytokines that are involved in the pathogenesis of vitiligo. Studies were conducted in human melanocytes, human keratinocytes, and peripheral blood mononuclear cells, that is, PBMCs, which are the cell type basically involved in the pathogenesis of vitiligo.

In the top panel, human melanocytes were stimulated using interferon gamma, and the expression of CXCL9, CXCL10, and CXCL11 genes were assessed. As shown in the figure, the blue bar shows normal cells, that is, those that were not stimulated with interferon gamma. You will see that this bar is very low, and there is no increase in the expression of these genes.

The red bar shows the melanocytes that were stimulated and incubated with vehicle. So, these melanocytes showed significant overexpression of these chemokines. The bars after the red bar show the melanocytes that were stimulated with interferon gamma in the presence of increasing concentrations of SCD-153. So, it is evident from this figure that the drug inhibited the overexpression of these genes for CXCL9, 10, and 11 in a dose-dependent manner.

Now, as shown in the bottom panel, similar results were seen in the human keratinocytes that were stimulated with interferon gamma. The figure on the lower right side shows inhibition of interferon gamma production from anti-CD3, CD28-stimulated human PBMCs, that are the key circulating immune cells.

Next slide, please. After promising results in the in-vitro studies, we have initiated evaluation of SCD-153 in the animal model of vitiligo. This model of vitiligo was developed based on finding that a noticeable proportion of melanoma patients developed vitiligo after receiving checkpoint inhibitor therapy.

This is believed to be a result of immune response mounted by endogenous autoreactive CD8 T-cells against the melanocyte antigens, along with a decrease in the regulatory T-cells, resulting in an unrestricted immune attack on the melanocytes. The melanoma cells and the melanocytes carry the similar antigen, that is gp100.

A similar milieu is created in this animal model that we are using for evaluation in vitiligo. In this model, melanoma cells are inoculated intradermally in the skin of the mice. After this, anti-CD4 antibody is injected to deplete the regulatory T-cells to allow for an unrestricted immune attack by the CD8 T-cells on the melanocyte antigens.

The tumor is then resected to ensure survival of the animal. After around a month of injection of the melanoma cells, the endogenous autoreactive CD8 T-cells infiltrate the epidermis and start mounting an attack on the melanocytes causing their destruction. The visible effect in the form of depigmentation of the tail skin of the mice is evident around Day 60.

The drug is expected to inhibit the depigmentation caused by the CD8 cells by its anti-inflammatory effect, as it is expected to inhibit the migration of CD8 T-cells to the site of immune response. This evaluation of SCD-153 in the animal model of vitiligo is ongoing. We intend to plan and initiate a clinical study in vitiligo once we have the results from this animal model. So, this concludes the update on the SCD-153 program.

Now, I would like to hand over to Dr. Sandeep Inamdar for the next update.

Sandeep Inamdar: Sure. Thanks, Mudgal. So, we will now move on to our oncology clinical asset, that's SBO-154, which is an anti-MUC1 antibody drug conjugate with the payload of MMAE.

Moving to the next slide. So, SBO -- so MUC1 is a glycoprotein that is widely expressed on cell surface of epithelial cells, including the epithelial tumors.

Expression of MUC1 in epithelial tumors differs from that in normal epithelium, making it a suitable antigen for targeting via an ADC.

While in normal tissues, the protein is expressed on only on the apical surface of the glandular epithelium, expression of MUC1 on tumor cells is more widespread across the entire cell surface, what we call as circumferential expression pattern.

The MUC1 protein consists of an extracellular glycosylated alpha chain, along with a cell membrane bound beta chain that you can see on the image on the right. Previous efforts at targeting MUC1 focused on the VNTR region of the alpha chain.

However, it is well known that the VNTR region undergoes proteolytic cleavage, resulting in large quantities of circulating VNTR subunits in the peripheral circulation. This may serve as a peripheral sink for ADCs targeting the alpha subunit, thereby limiting access of the therapeutic to the tumor site. SPARC's approach is unique in that our binding epitope of SBO-154 targets the alpha beta junctions, which is less likely to be impacted by proteolytic cleavage, thereby reducing the potential sink effect, allowing better tumor targeting. We have evaluated plasma samples of cancer patients with different solid tumors and have been able to demonstrate significantly lower levels of the alpha beta subunit in the peripheral circulation compared to the VNTR region, which further strengthens our hypothesis.

Go to the next slide. So in order to understand the expression pattern of our specific MUC1 epitope on various tumors, we evaluated patient tumor samples for MUC1-SEA domain with a proprietary immunohistochemistry assay. This assay is semi-quantitative in the form of H-Score for each sample. These H-Score can range from 0 to 300. Scores above 150 are typically considered as high to very high expression of the antigen.

As you can see, the H-Score in more than half the patients with late-stage ER-positive breast cancer and adenocarcinoma of the lung, which are two highly prevalent tumors, exceed the threshold of 150, particularly as it pertains to the circumferential expression pattern, which is relevant for ADC targeting.

In addition, ovarian and pancreatic cancers also have exceedingly high expression, although that expression tends to skew more towards apical

expression. Based on these data, we have designed our Phase 1 study to evaluate these tumors for evidence of efficacy.

Next slide. So this slide shows a representative dataset for the in vivo efficacy of SBO-154 in CDX mouse models. This particular experiment is done using the COLO-357 cell line, which is a pancreatic cancer model. The image on the left is with the same cell expressing MUC1-SEA at a moderate level of 130 H-Score. SBO154, as you can see, is significantly superior in terms of tumor reduction compared to vehicle control, as well as MMAE conjugated to anti-HBV, which is an untargeted control. However, this activity is significantly improved in the model on the right, where the H-Score is higher than 200, where all eight animals saw a deep response in the form of tumor regression.

Similar results were also obtained with other models of breast and ovarian cancer. This suggests that the activity of SBO-154 is closely linked to the level of MUC1 expression on the tumor cell. The H-Score of 200 is closer to the level of expression in human tumor samples that we observed. That's the data that I shared on the previous slide.

Moving to the next slide for the Phase 1 study. Coming on to the Phase 1 study design, as Anil had alluded to earlier, we initiated our Phase 1 study in July-August of 2025 in the US, in Australia, and in India. This is a study design of the adaptive dose escalation and expansion trial. The Phase 1a dose escalation is currently ongoing, where we enroll all solid tumors, irrespective of their MUC1 expression.

We are collecting the tumor samples of these patients to retrospectively test for MUC1 expression and correlate with clinical activity. Once we are near completion of the dose escalation portion, we plan to enroll a backfill cohort of approximately 25 patients with high MUC1 expression.

The goal of the backfill cohort will be to get additional safety and PK at specific dose levels, but also to get early evidence of clinical activity, since the patients will all be pre-selected for high levels of antigen expression. Once we complete the part 1a, we expect to initiate 1b in three specific tumor types. These are expansion cohorts in estrogen receptor positive breast cancer, adenocarcinoma of the lung, and ovarian cancer. And the choice of these tumors is based on the high level of MUC1 expression known to be present in these patients.

Again, patients will be pre-selected for high MUC1 expression in this portion of the study. The study design will be an adaptive Simon's 2 stage, where we will initially enroll about 13 to 15 patients in the first stage. Once we observe a sufficient level of threshold clinical activity, we will enroll an additional 15 or so patients in the second stage for a total of approximately 30 patients in each cohort. This will provide definitive evidence of clinical activity that will enable us to proceed to the next phase of development.

Next slide. A quick update on the study status. The study is actively enrolling at 11 sites across multiple geographies. The first two dose cohorts have completed enrollment with no unexpected safety findings, allowing us to recently initiate the third dose escalation cohort. We expect to complete dosing to the highest dose cohort of 2.4 milligrams per kilogram by the end of the third quarter of this year.

This brings me to my final slide, which is highlighting the upcoming milestones, summarizing the upcoming milestones for this trial. We expect, as I mentioned, to expect to have the maximum tolerated dose, or the MTD, identified by the end of the third quarter of 2026, allowing us to proceed with cohort expansion by the end of the calendar year, and a potential early clinical proof of concept by the second half of 2027.

I'll now hand it back to Anil for his concluding remarks before we take questions.

Anil Raghavan: Thank you, Sandeep. I'm not planning to go through this slide. This is just a summary of the key drivers of our value that we have discussed through the course of this presentation. Thank you again for joining in, and we're really looking forward to the Q&A.

Moderator: Thank you very much. We will now begin the question-and-answer session. As there are no questions from the participants, I'll now hand the conference over to Mr. Jaydeep for closing comments. Thank you, and over to you.

Jaydeep Issrani: Thank you, Ikra. Thank you, everyone, for joining the call today. If you may have any questions, you can reach out to us on the details provided on our website, and we'll answer your questions. Thank you again, and have a good day. Bye-bye.

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