SPARC/Sec/SE/2025-26/47 January 08, 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: Investor Presentation

Further to our letter SPARC/Sec/SE/2025-26/45 dated December 31, 2025 and pursuant to Regulation 30 of the SEBI (Listing Obligations and Disclosure Requirements) Regulations, 2015, we enclosed herewith the investor presentation to be held today i.e. January 08, 2026 at 4:00 PM (IST), which we shall be uploading on our website after sending this letter to you.

This conference call will be reachable through an audio dial-in.

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Management presentation: The presentation pertaining to this discussion can be accessed through the link given below on the date of audio conference.

https://links.ccwebcast.com/?EventId=SUN08012026

This is for your information and dissemination.

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

Kajal Kiran Damania

Kajal Damania
Company Secretary and Compliance Officer
Encl: As above

R&D DAY

Updates on prioritized programs

8 January 2026

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Discussion flow

01

SPARC's growth charter & priorities

Anil Raghavan

02

SCD-153 update

Mudgal Kothekar

03

SBO-154 update

Sandeep Inamdar

04

Q & A

Revisiting the post-PROSEEK focus – Portfolio execution progressing as planned

Portfolio optimization

10+
assets under development

- Oncology and Immunology as key TA focus
- Prioritize resourcing for SBO-154 & SCD-153 to achieve meaningful value inflection points

Business model flexibility

7
strategic & commercial partnerships

- Early stage licensing
- Pivoting to smart deals (NewCo. formation, Equity transactions)

Short-term catalysts & costs

4
near-term milestones

- Multiple levers to drive near-term cash events
- Cost reduction through reorganization and execution efficiency

Momentum on significant strategic priorities creating a path to meaningful value inflection

TA= Therapy Area

SCD-153 and SBO-154 lead the SPARC portfolio and are set for important catalysts in the medium term

Both programs achieved their planned milestones ahead of time

SCD-153

- Prodrug of an analogue of an endogenous immunosuppressive metabolite, Itaconate
- Phase 1a study in healthy volunteers completed
  - Safe & well tolerated at all doses tested
  - Measurable concentration detected in dermis and epidermis
- Foam formulation finalized
- Phase 1b study initiated in alopecia areata patients
  - Dose cohort 1 completed
  - Recruitment for dose cohort 2 initiated
- Safety validation and early efficacy signals expected by Q4 2026

SBO-154

- Antibody Drug Conjugate targeting tumour associated MUC1 SEA domain, delivering Monomethyl Auristatin E (MMAE)
- IND filed and accepted by US FDA, Australia TGA and India DCGI
- Phase 1a dose escalation study initiated in advanced solid tumor patients with sites active in USA, Australia and India
  - Dose cohorts 1 and 2 completed
  - Recruitment for dose cohort 3 initiated
- Phase 1a (~50 patients) expected to be completed in 2026

Prioritized resource allocation to accelerate human proof-of-concept and unlock value of clinical assets

FDA= Food and Drug Administration | TGA=Therapeutic Goods Administration | DCGI=Drugs Controller General of India

Sezaby PRV summary judgement granted in SPARC's favour

Pediatric Rare Diseases Voucher (PRV)

- U.S. District Court for the District of Columbia granted summary judgement in favour of SPARC
- Agency has 60 days to appeal against the motion
- Subject to a possible appeal from the agency, SPARC expects to receive a tradable PRV
- Market demand remains robust for PRVs, as it can accelerate NDA review timeline

Enforcement of market exclusivity

- SPARC's citizen petition is still under review by US FDA
- Continuing efforts including agency engagement to remove DESI formulations

DESI=Drug Efficacy Study Implementation

PDP-716 CRL resubmission completed

- Ocuvex Therapeutics acquired SPARC's commercialization partner, Visiox Pharmaceuticals in August, 2024
- API manufacturing site of third party partner received Voluntary Action Indicated (VAI) status from US FDA
- Ocuvex resubmitted PDP-716 NDA with US FDA in November, 2025
- Building back-up sites to ensure continuous supply
  - SPARC qualified additional API vendor
  - SPARC & Ocuvex evaluating additional sites for finished product manufacturing

API=Active Pharmaceutical Ingredient | NDA=New Drug Application

NewCo creation accelerates clinical development

Tiller Therapeutics on track for IND filing and clinical proof-of-concept study

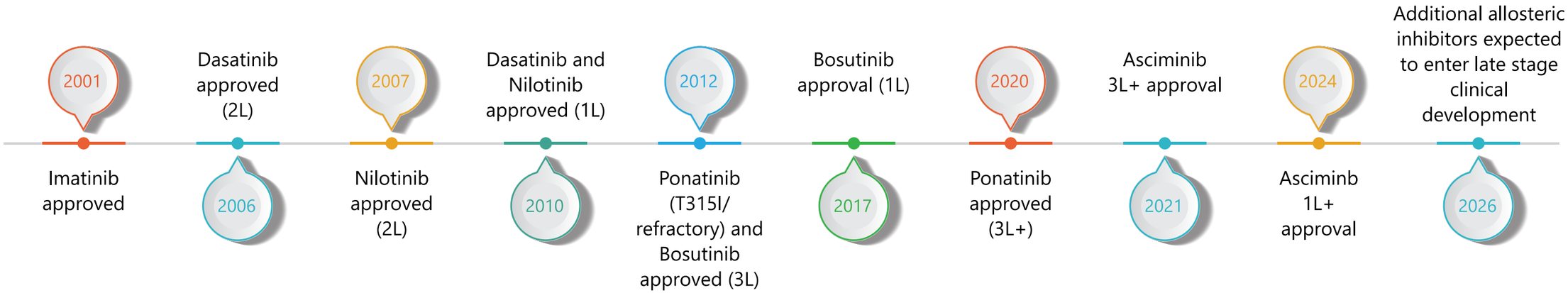
- SCO-155/TILR-097 is a Small Molecule Drug Conjugate (SMDC) developed under research collaboration with University of California, San Francisco (UCSF)

- Strategic Partnership*
  - SPARC and UCSF signed a binding LOI with Tiller Therapeutics Inc. (Tiller) granting exclusive worldwide license for development and commercialization of SCO-155/TILR-097 to Tiller
  - SPARC eligible to receive upto 55% equity stake in Tiller's fully diluted capital stock in exchange of the license rights

- Tiller update
  - SCO-155/TILR-097 GLP toxicology studies completed supporting impressive safety and efficacy relative to alternative, expensive formats such as ADCs and TCEs
  - Pre-IND filed with US FDA by Tiller in Oct'25; Feedback received in Nov'25 providing clear pathway through proposed early clinical studies
  - Tiller raised pre-Seed external capital in 2025; currently raising priced external Seed round to fund the initial clinical study of SCO-155/TILR-097 in mCRPC patients
  - CEO Eileen McCullough, manufacturing, and regulatory teams hired

*SPARC's Press Release dated December 17, 2024 | IND=Investigational New Drug | LOI=Letter of Intent | ADCs=Antibody Drug Conjugates | TCEs=T-Cell Engagers

Vodobatinib poses resource allocation challenges in a shifting CML landscape



- CML market is in a phase of dynamic evolution, with new entrants driving innovation through allosteric inhibitors
- While early-stage data from multiple players is promising, the lack of major licensing deals underscores a significant long-term growth opportunity
- Vodobatinib, like others, must establish robust clinical data, however, resource allocation is critical given SPARC's broader portfolio and competing priorities
- As the market matures, we anticipate strategic collaborations and value-accretive partnerships to unlock value
- SPARC continues to engage potential partners and also exploring options to create alternate structure

Navigating an evolving market with measured expectations

Streamlined, thematically cohesive portfolio

Focused on high growth therapeutic areas and emerging modalities

Targeted delivery of multi-modal cancer therapeutics

- Antibody Drug Conjugates, including bi-specific targeting and bi-functional payloads
- Immune-Stimulating Antibody Conjugates (ISACs)
- Small molecule drug conjugates
- siRNA nanoparticle platform for extra hepatic delivery

DDR pathway inhibitors for synthetic lethality

- Mono-therapy and PARP inhibitor sensitization in HR deficient tumors
- Sensitization of standard of care agents causing DNA damage

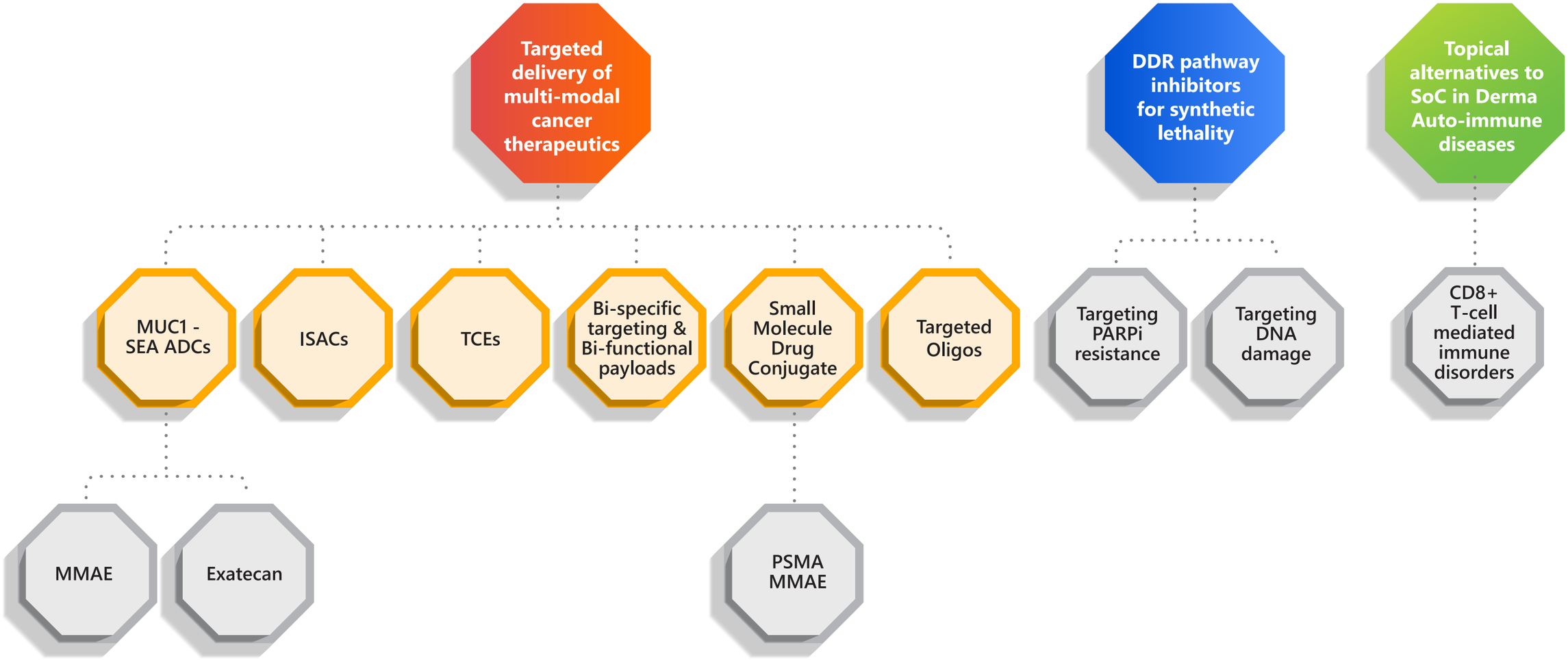
Topical alternatives to SoC in Derma Auto-immune diseases

- Non-JAK pathways to address immune mediated disorders
- Rational combinations including fixed dose combinations

SoC=Standard of Care

Modular constructs with platform potential

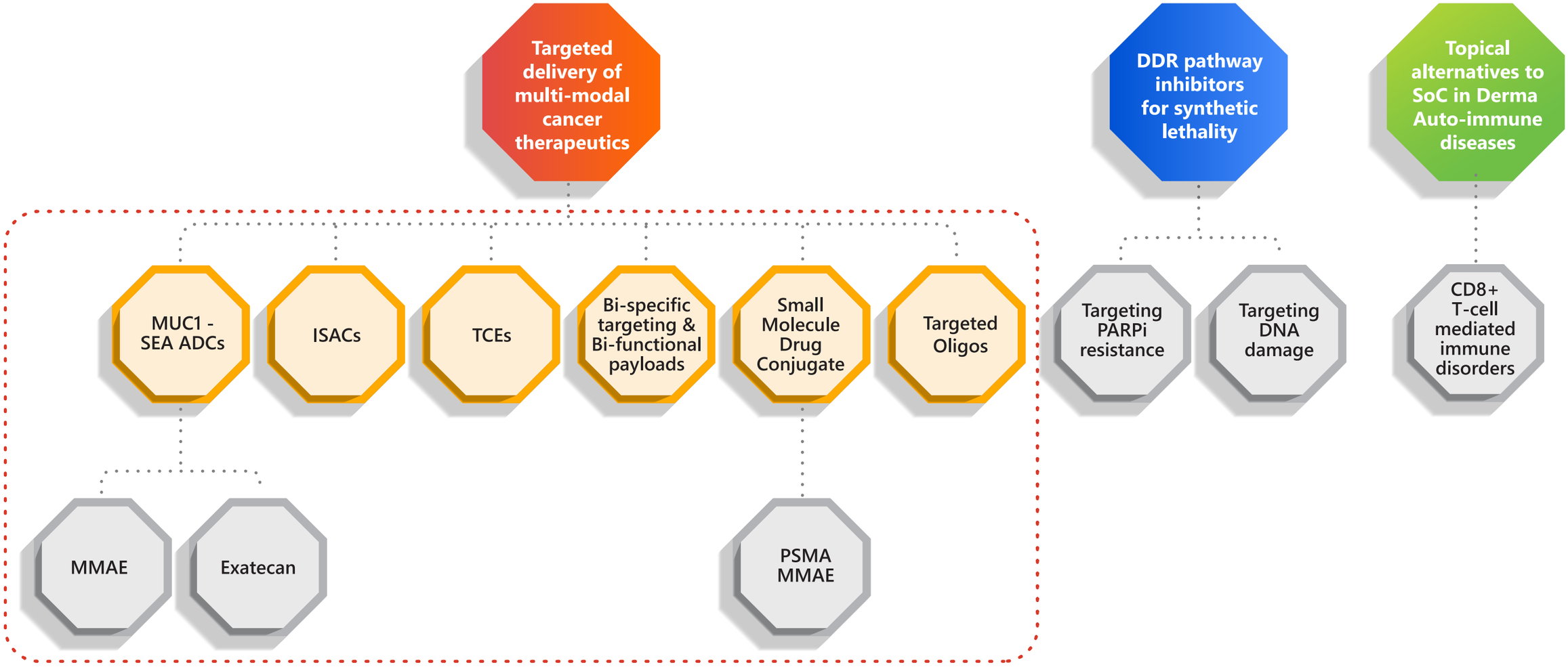
SPARC portfolio carries several scalable prototypes



PSMA=Prostate Specific Membrane Antigen | DDR=DNA Damage Response | PARPi=Poly (ADP-ribose) polymerase inhibitor

Modular constructs with platform potential

SPARC portfolio carries several scalable prototypes

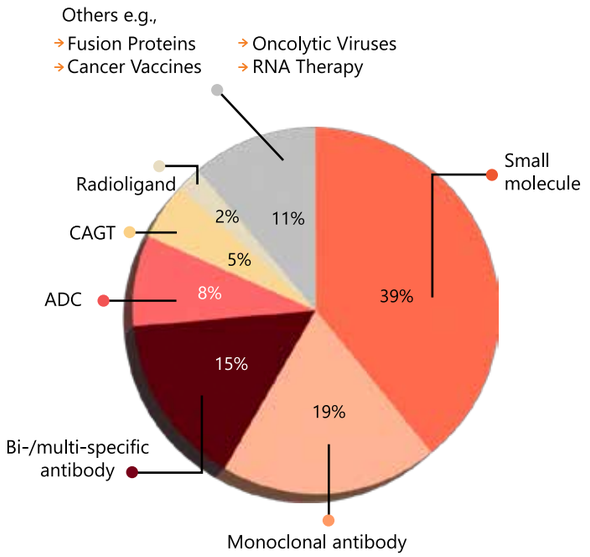


Targeted delivery of multi-modal payloads expected to drive the next big wave in Oncology

- ADCs are on the way to becoming one of the most impactful therapeutic modalities in Oncology on the back of improved technology and targeting
- The number of oncology deals related to key novel modalities has grown in the past 5 years, with ADC deal volume surging more than threefold
- Multi-specific antibody trials have grown significantly, being represented in 17% of hematological-oncology trials and 11% of solid tumor trials in 2024
- While PD-1/PD-L1 checkpoint inhibitors still accounted 15% of all oncology trial starts, they have been on a downward trajectory in recent years
- ADCs offer a vast and comparatively de-risked pipeline, as measured by probability of technical and regulatory success (PTRS)-especially compared with the overall oncology category

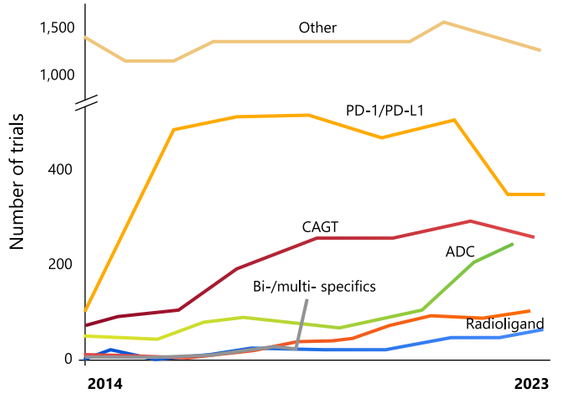
Oncology Pipeline

All assets in Phase 1-3 by modality (n=1924)



Oncology clinical trial activity

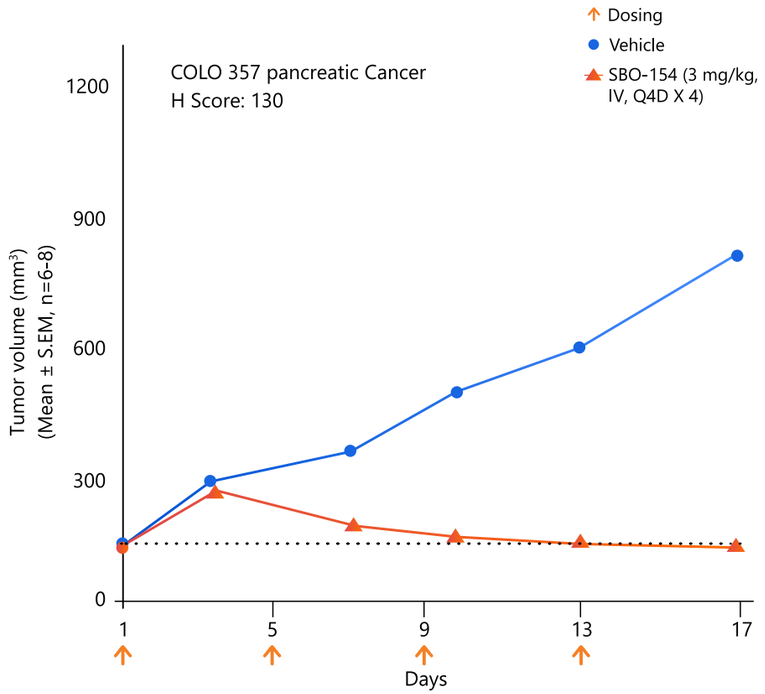
Clinical trial starts, Phase 1-3, primary tested drug type



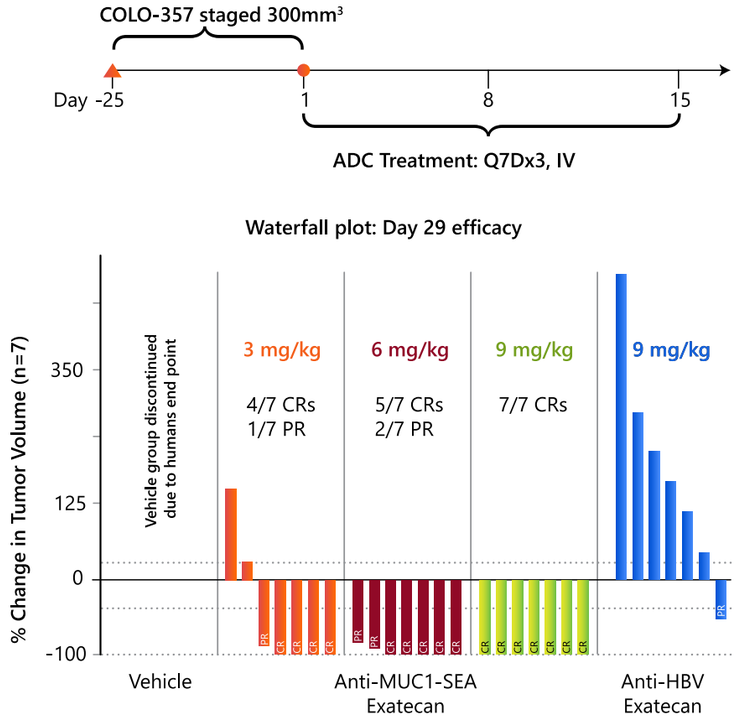
Adapted from IQVIA, Breaking New Ground: Advancing Cancer Care with Novel Therapeutic Modalities; Deep dive: Antibody drug conjugates, bi-/multi-specifics and radioligand therapies
PD-1=Programmed Death-1 | PD-L1=Programmed Death-Ligand 1 | CAGT= Cell and Gene Therapy | RNA=Ribonucleic Acid

SPARC's humanized anti-MUC1-SEA mAbs provide an opportunity to create multiple MUC1-SEA-targeted ADCs with different payloads

Anti-MUC1 ADC of MMAE (SBO-154)



Anti-MUC1 ADC of TOPO1 inhibitor



Key upcoming catalysts:

SBO-154 clinical proof-of-concept

- Dose escalation completion by Q4 2026
- Dose expansion completion by Q2 2028

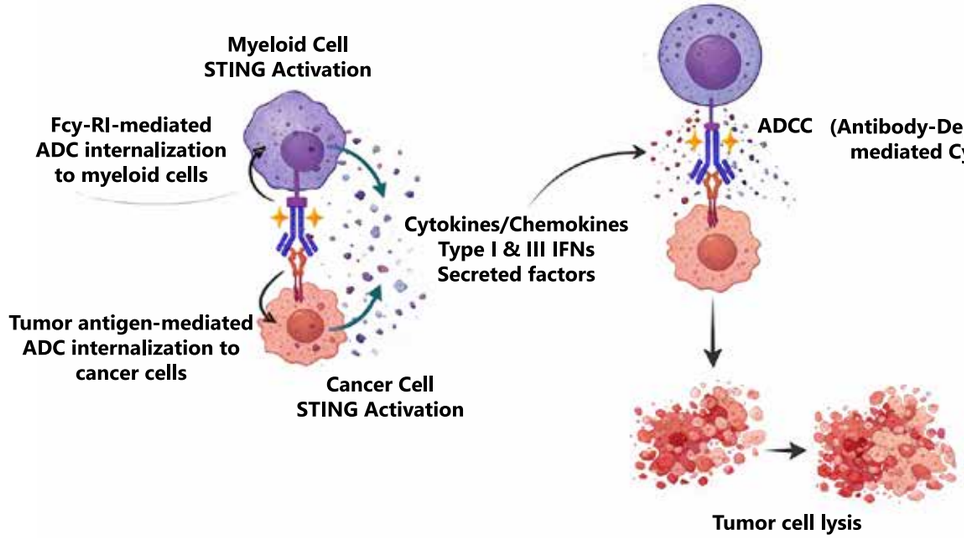
SPARC's humanized anti-MUC1-SEA antibody demonstrates robust tumor growth inhibition with multiple payloads, validating platform flexibility and provides the opportunity to build a broad ADC portfolio

Immune-Stimulating Antibody Conjugate (ISAC)

Harnessing the immune system against cancer

- ISACs combine targeted drug delivery with activation of cells of the innate immune system for effective tumor control and long-term remission
  - ISAC bridges tumor cells with FcR1+ cells of the innate immune system and brings about activation of the latter to exert cytotoxic activity against tumor cells
- ISACs initially stimulate cells of the innate immune system and later cells of the adaptive immune system to ensure effective tumor growth control
- Work synergistically with immune checkpoint inhibitors, expanding treatment options for resistant cancers
- Key players in the emerging class – Daiichi Sankyo, Mersana (Day One Therapeutics)

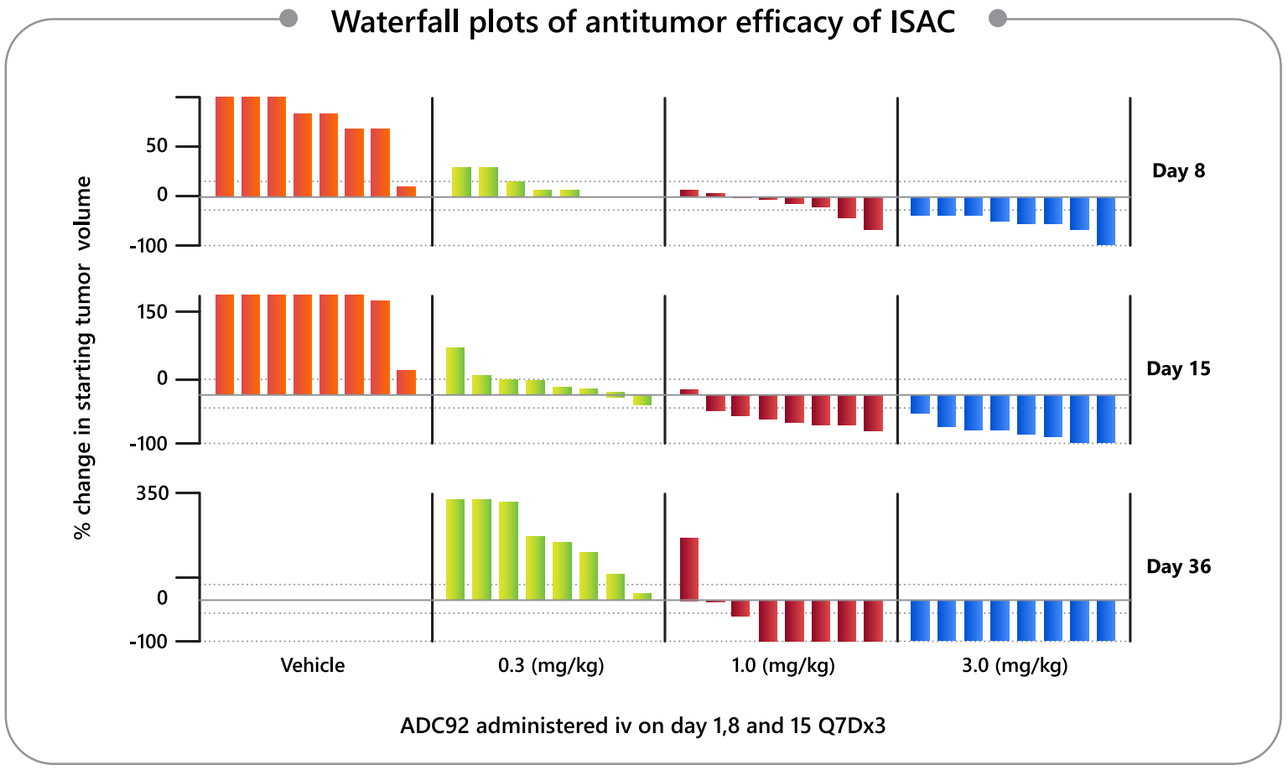
Hypothesized mechanism of the synergy between STING pathway activation & ADCC function*



*Adapted from https://xueqiu.com/8965749698/265525906

MUC1-SEA Immune-Stimulating Antibody Conjugates deliver SPARC's proprietary STING agonists

Leverages a novel, internally developed linker system for better internalization & safety margins



Current status:

- Pre-clinical proof-of-concept achieved in immuno-incompetent T-cell-deficient nude mice with intact innate immune system

Next steps:

- Progress anti-MUC1 ISAC to clinic after validation of SBO-154
- Explore additional targeting options using the same payload linker combination

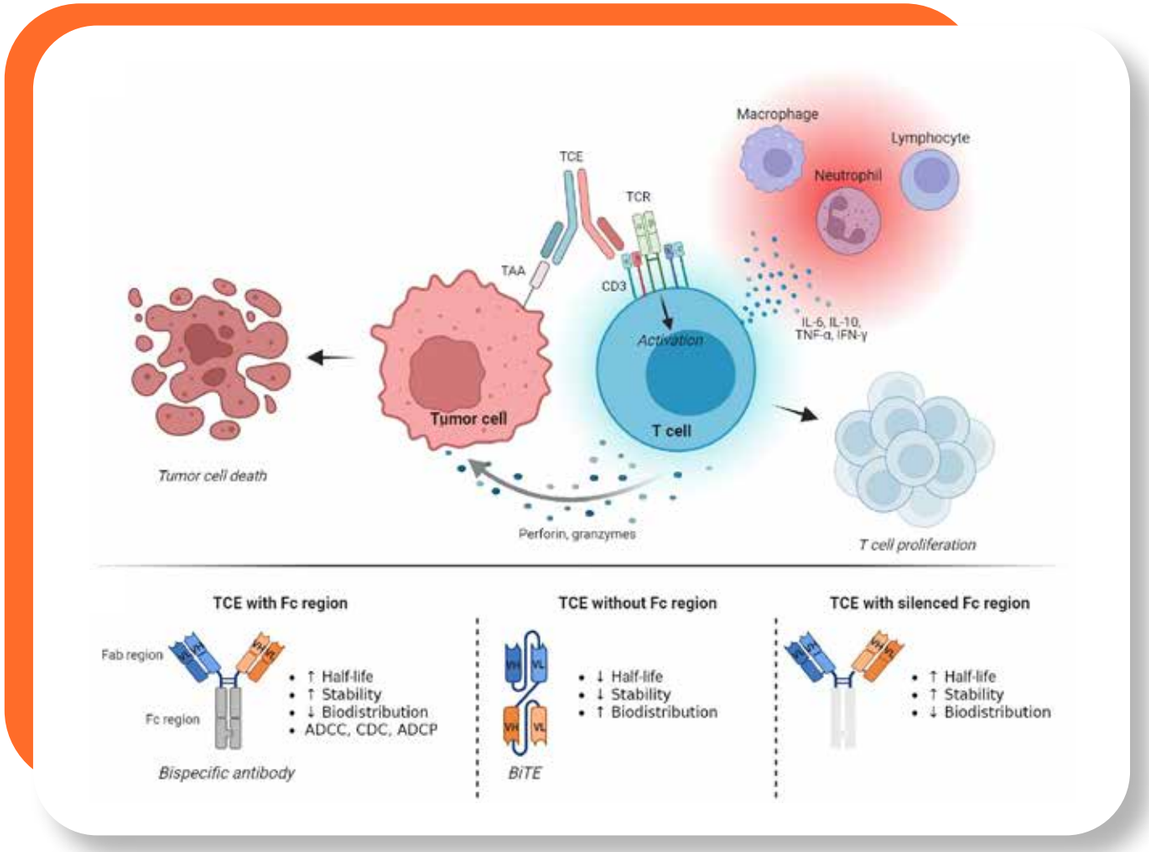
Dose-dependent human tumor regression with human MUC1-SEA-targeted ISAC validates opportunity to combine immune activation and targeted delivery

STING=Stimulator of Interferon Genes

T-cell engagers (TCE) orchestrate tumor targeted T-cell activation

Remodeling TCEs to limit the risk of broad cytokine release syndrome

- T-cell engagers represent a revolutionary approach in cancer immunotherapy, leveraging the immune system's inherent cytotoxic potential to selectively target and eliminate cancer cells
- First generation TCEs hampered by the dual challenges of:
  - Limited circulating half-life : New Fc-engineered scaffolds are enabling less frequent dosing and better patient compliance
  - Cytokine release syndrome (CRS) and associated immune-related toxicities
- SPARC TCEs replace Fc with Human Serum Albumin without loss of half-life and augment the design further with preferential activation of CD8+ cytotoxic T cells

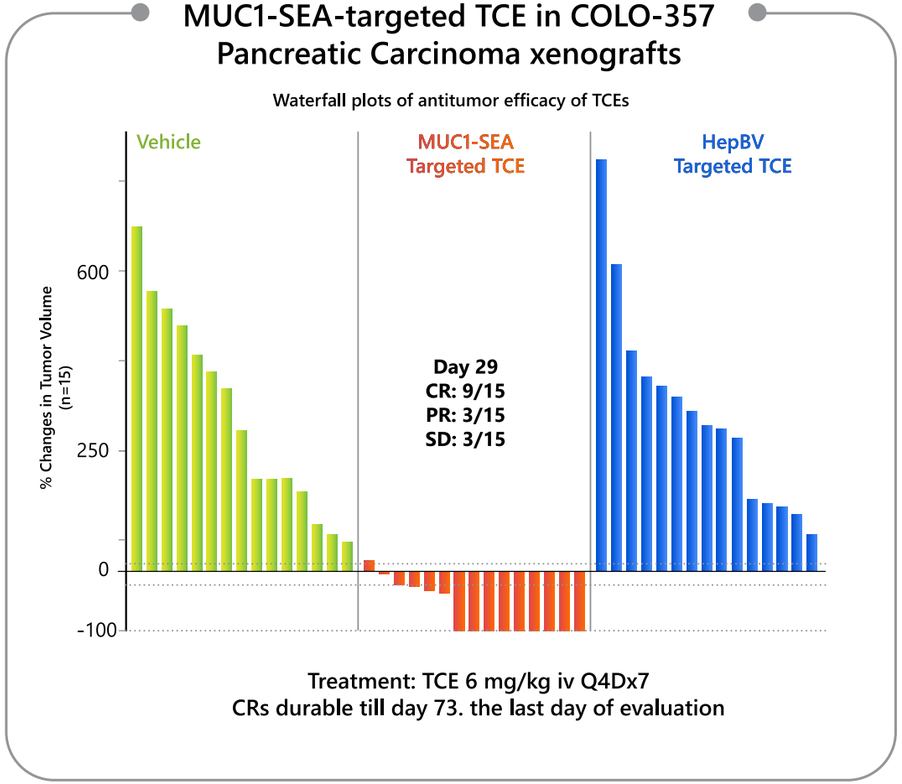


Adapted from Front. Immunol., 06 October 2025; Sec. Cancer Immunity and Immunotherapy | CD8=Cluster of Differentiation 8

Anti-MUC1 TCE Albufusion achieved proof-of-concept

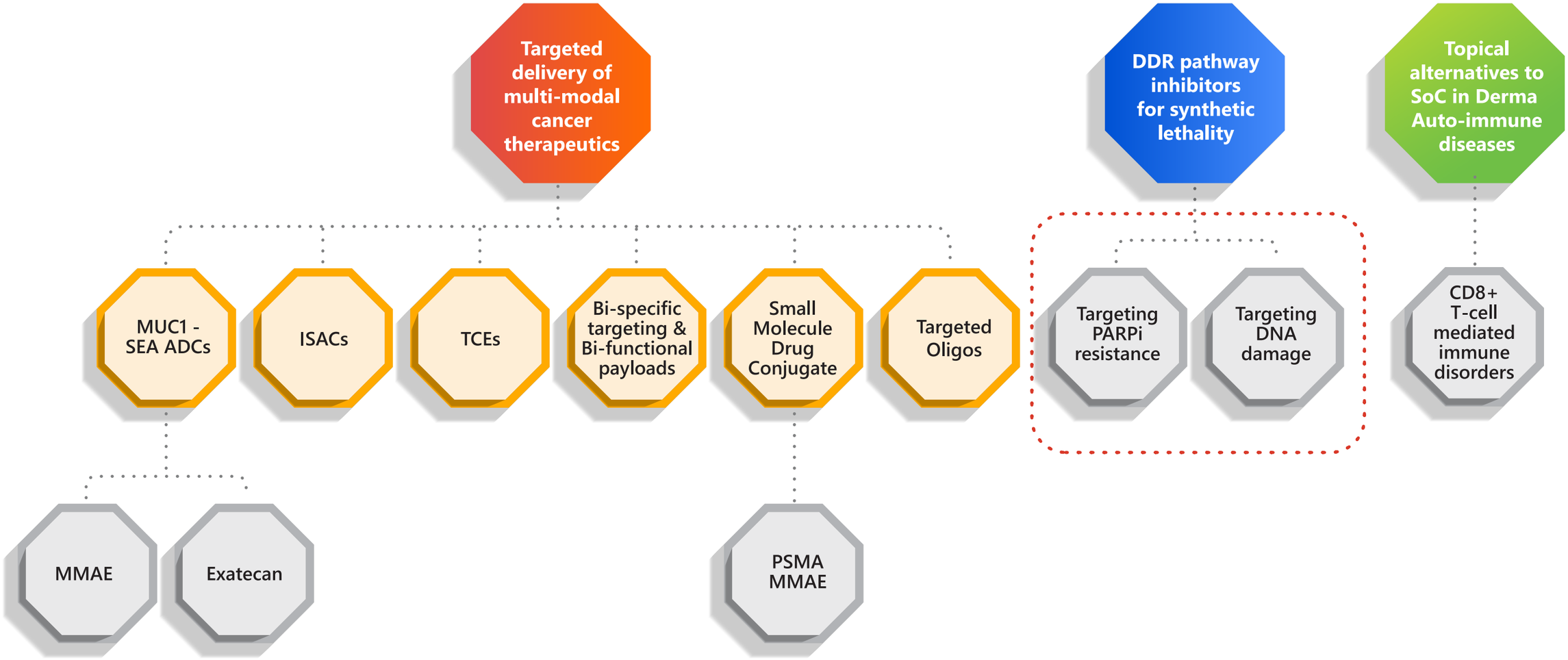
SPARC's TCE shows potent cytotoxicity and durable tumor regression, promising a highly differentiated TCE technology

- SPARC's MUC1-SEA x CD3 bispecific TCE binds to both MUC1+ human carcinoma cells and CD3+ human T cells
- SPARC's bispecific TCE causes dose-dependent cytotoxicity by T cells against MUC1+ tumor cells but not against MUC1 negative tumor cells
- Induction of cytotoxicity of bispecific TCE is directly proportional to the surface MUC1 density on tumor cells.
  - **Current status:**
    - Pre-clinical proof-of-concept for Albufusion achieved with MUC1-SEA
  - **Next steps:**
    - Proof-of-concept for conditional activation of T-cells
- Beyond TCEs, SPARC is actively investigating bispecific ADCs as part of its broader strategy and to complement TCE strategy



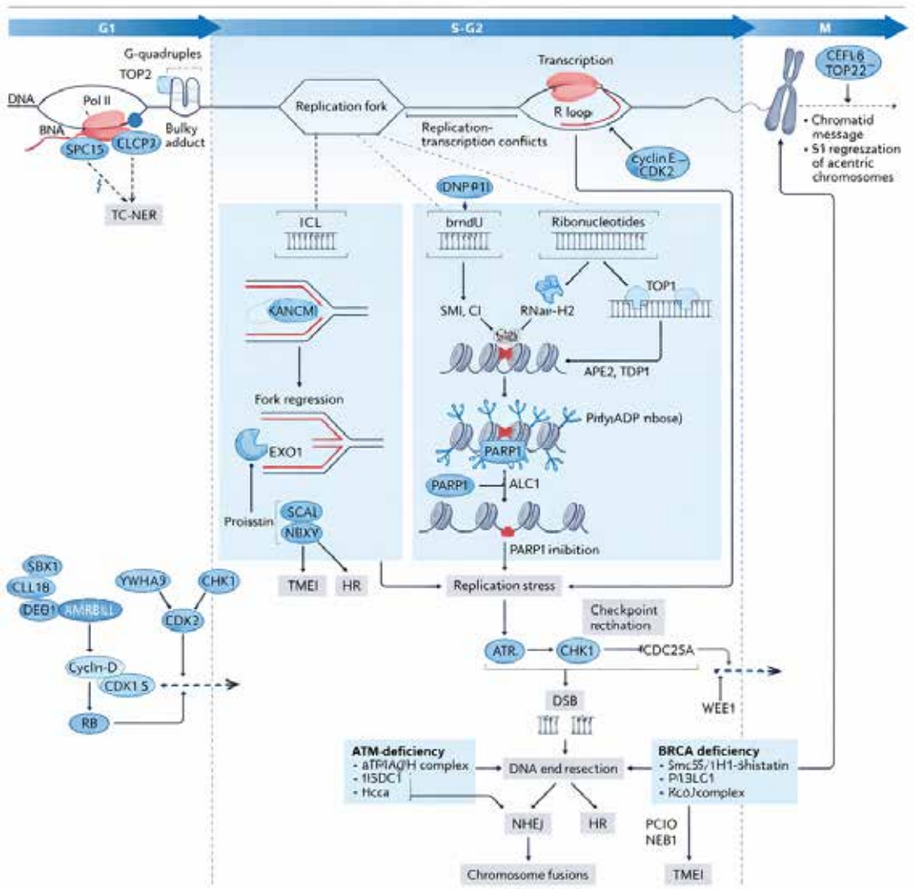
Modular constructs with platform potential

SPARC portfolio carries several scalable prototypes



Synthetic lethality and DNA Damage Response (DDR) pathway blockade expanding beyond PARP inhibition

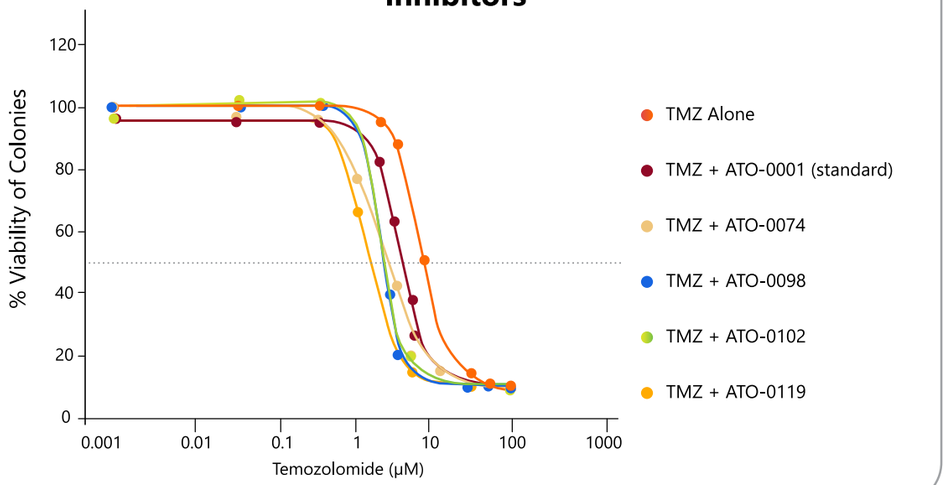
- Homologous Recombination (HR) deficiency is observed with many solid tumors and is mechanistically connected with DDR
- HR is one of the major repair pathways activated by DDR to fix double-strand DNA breaks
- Inhibition of components of DDR may render HR-deficient tumor cells susceptible to synthetic lethality
- HR-deficient tumors become resistant to treatments over time, often by restoring HR capability
  - High unmet medical need for HR-deficient tumors
- Exploiting synthetic lethality through targeted inhibition of DDR pathway in HR-deficient tumors would provide new avenues for improved treatments
- Two pronged approach adopted by SPARC to bridge this gap
  - Inhibition of pathway that targets DNA repair, which has potential as both chemo/radio-sensitizer [Target 1]
  - Inhibition of a key protein overexpressed in PARPi resistance [Target 2]



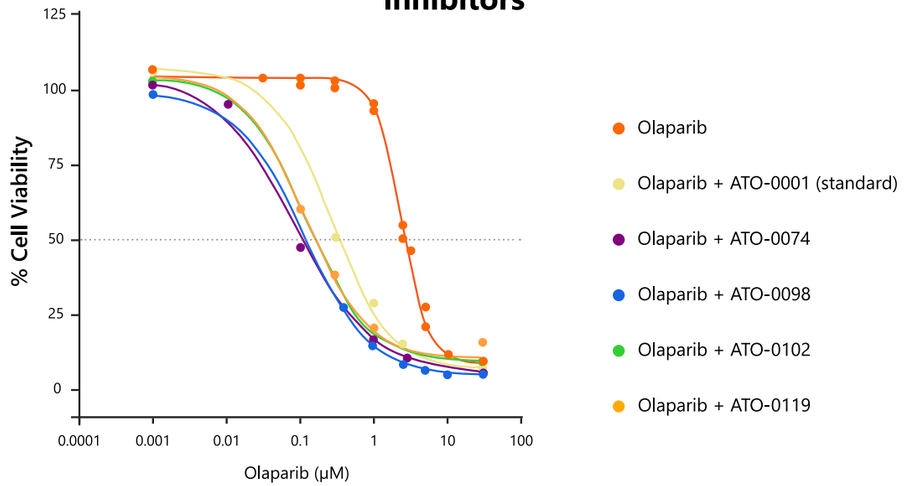
Adapted from Nature Reviews Molecular Cell Biology, Vol 24, 2023, 477–494

Target 1: SPARC lead assets outperform key competitor asset in GBM and TNBC

U251MG RedFluc Cells: TMZ + 10 nM ATO inhibitors



SUMI49PT Cells: Olaparib + 10nM ATO inhibitors

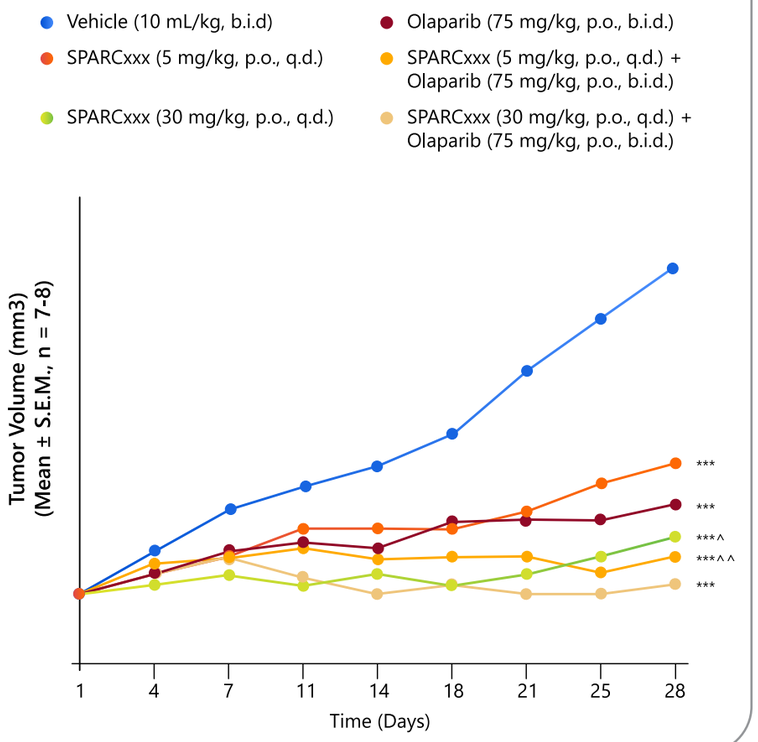


- SPARC's DDR-targeting approach is agnostic to mutational background, applicable across tumor types
- SPARC lead compounds show superior chemo-sensitization vs competitor assets in GBM (U251MG) and similar benefit was observed in Head & Neck, TNBC, NSCLC, PDAC, and breast cancer

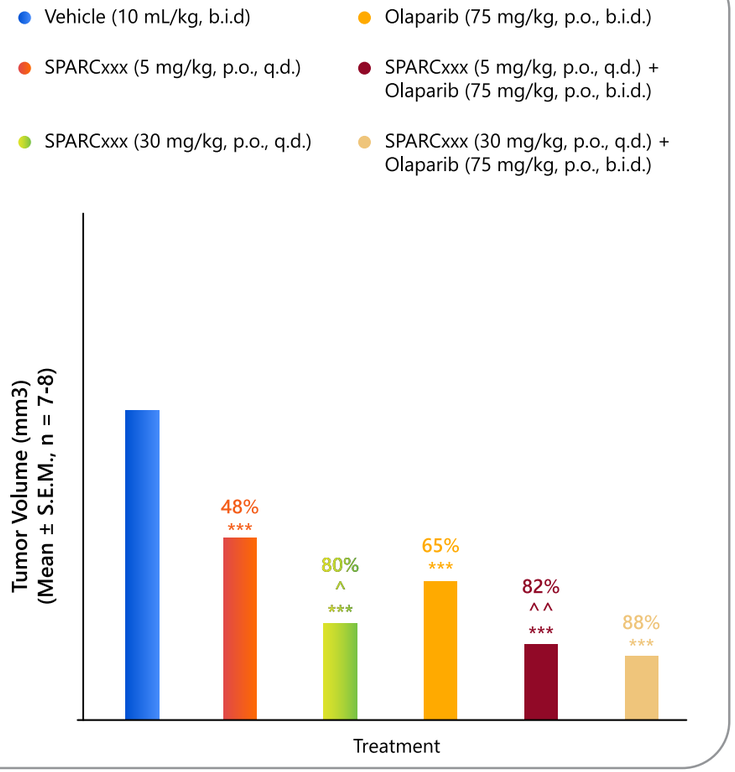
NSCLC=Non-Small Cell Lung Cancer | PDAC=Pancreatic Ductal Adenocarcinoma | GBM=Glioblastoma Multiforme | TNBC=Triple Negative Breast Cancer

Target 2: SPARC's asset demonstrates strong dose-dependent tumor growth inhibition in BRCA1-mutant TNBC models

Tumor growth kinetics



% Tumor growth inhibition on day 28



- Exhibits selectivity over HR-proficient TNBC and non-oncogenic cell lines
- Demonstrated significant tumor growth inhibition in BRCA1 mutant mouse models and strong synergy observed with PARPi
- No effect on body weight was observed in both solo and combination therapy groups

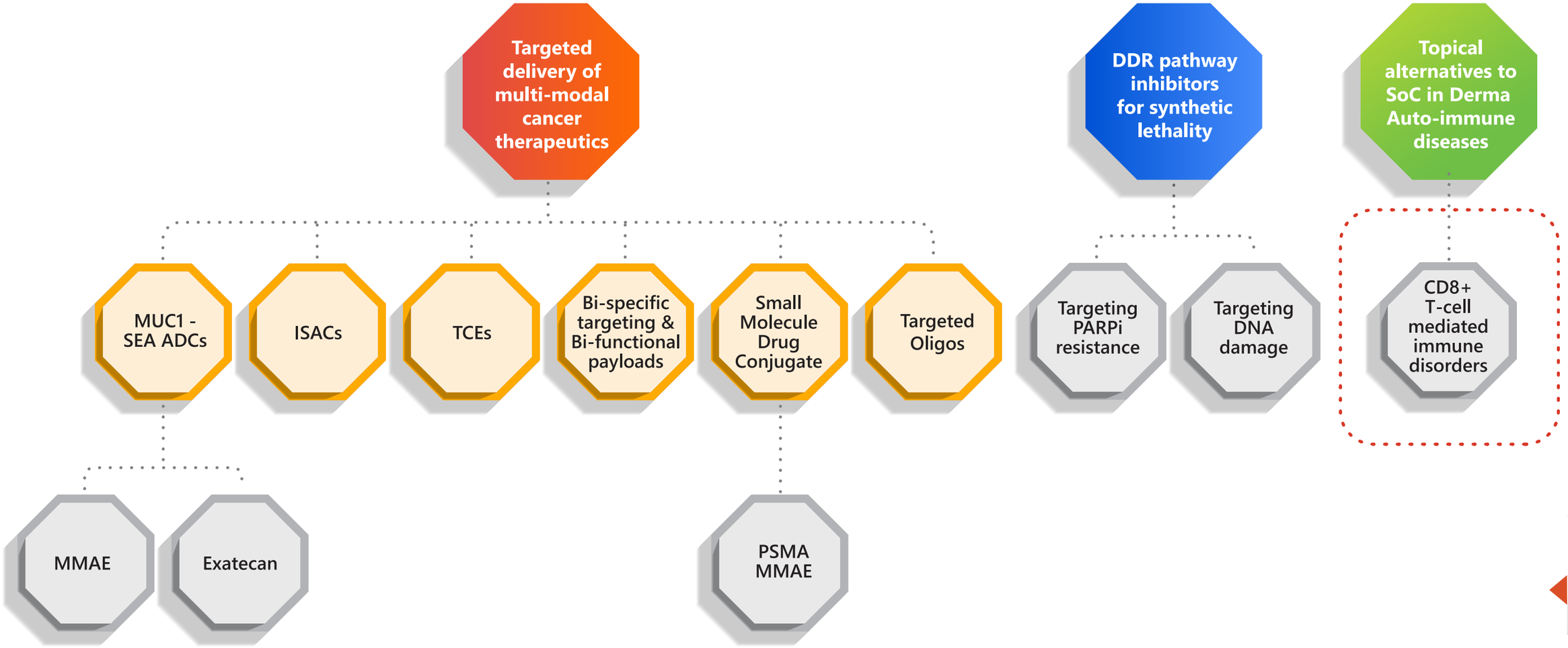
One way ANOVA followed by Tukey's test on day 28 *** p < 0. 001 vs Vehicle; ^ p< 0.05 vs AL0822 (5 mg/kg, QD);^^ p< 0.01 vs AL0822 (5mg/kg, QD)

Delivers strong monotherapy activity and synergy with PARPi in BRCA1-mutant TNBC models, positioning it as a differentiated DDR-targeting asset with potential for expansion into multiple solid tumors

BRCA=Breast Cancer gene

Modular constructs with platform potential

SPARC portfolio carries several scalable prototypes



Safer alternatives and synergetic combinations will drive growth in Immunology

- Dermato-immunological conditions such as alopecia areata, vitiligo, atopic dermatitis etc. present a persistent challenge in balancing efficacy, safety, and long-term disease control
- Agents that are often used first-line, including corticosteroids and calcineurin inhibitor have modest efficacy and are limited by adverse effects, tolerability issues, and patient concerns
- Systemic therapies, including biologics and oral JAK inhibitors, have transformed care for severe disease, delivering rapid and sustained control. However, the need for monitoring and risks of infection, malignancy and thromboembolic complications limit their long-term use while the diseases flare upon discontinuation of therapy
- This creates a significant unmet need for novel strategies that combine efficacy with safety and patient acceptability for long-term usage
- Combination regimens leveraging complementary mechanisms provide a promising solution aimed at faster onset of efficacy and a sustained response while minimizing systemic adverse effects.
- Novel topical therapies could also be used adjunctively to enhance the efficacy or sequentially to sustain the remission obtained with systemic agents with a potential to reduce the overall exposure to systemic agents thereby alleviating the safety concerns in addition to enhanced outcomes

SCD-153 has potential as a standalone agent, as a combination partner with JAKi, and as maintenance therapy following hair growth induction with JAKi

Diversified portfolio with multiple high-value bets and maturing platforms

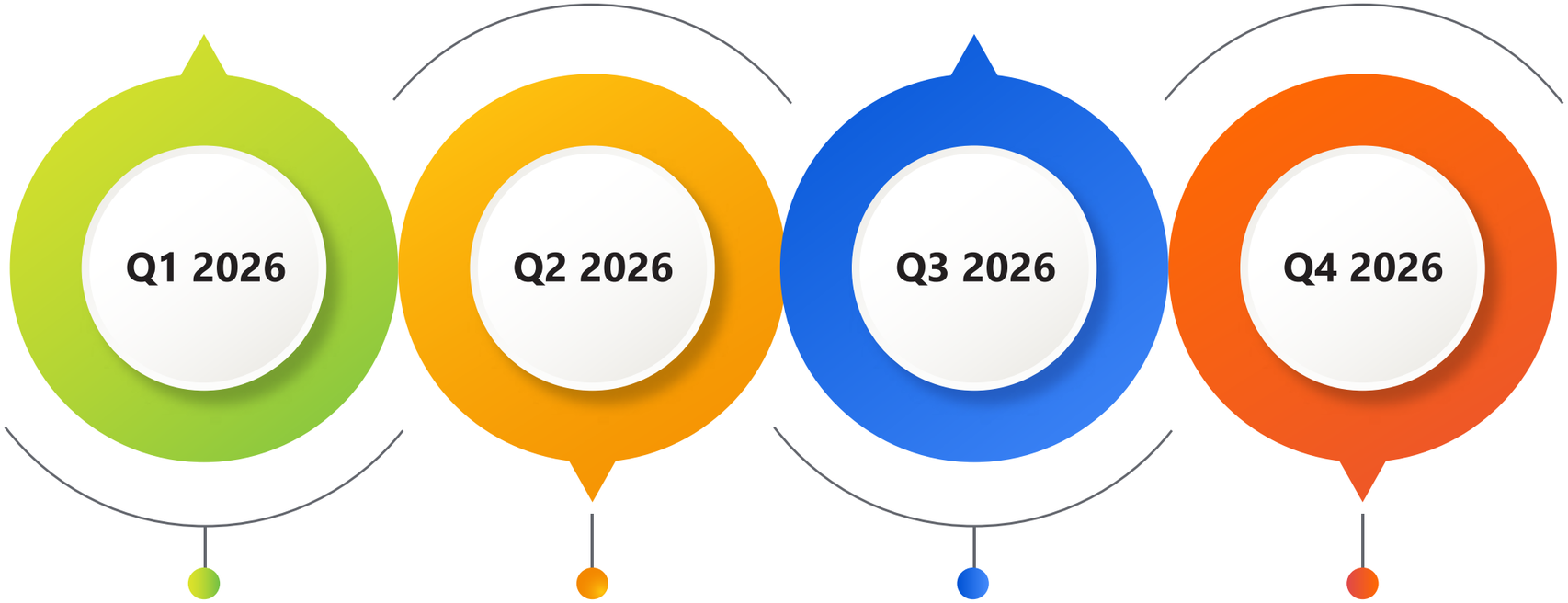
Robust pipeline spans biologics and small molecules with focus on differentiated mechanisms, targeting high unmet needs in oncology and immunology

Asset	MoA	Indication	Discovery	Preclinical	Phase 1	Phase 2	Phase 3
Biologics							
SPARC122	Anti-MUC1 ADC of MMAE	Solid tumors					
SPARC125	Anti-MUC1 ADC of Exatecan	Solid tumors					
SPARC127	Bispecific ADC	Solid tumors					
SPARC128	Bifunctional ADC	Solid tumors					
SPARC126	STING Agonist ADC	Solid tumors					
SPARC129	T-cell Engager	Solid tumors					
Small Molecules							
Vodobatinib	BCR-ABL inhibitor	Refractory CML					
SPARC121	Itaconate derivative	Alopecia Areata					
SPARC121	Itaconate derivative	Vitiligo					
SCO-155/SPARC130	SMDC	Prostate Cancer					
SPARC124	DDR Mechanism	Solid tumors					
SPARC131	CD8+ T-cells	Immunological disorders					

Oncology Immunology

MoA = Mechanism of Action | ADC - Antibody-Drug Conjugate | CML = Chronic Myeloid Leukemia | SMDC = Small Molecule Drug Conjugate | DDR = DNA Damage Response

Near-term milestones and priorities



- PRV appeal /outcome
- Tiller license agreement

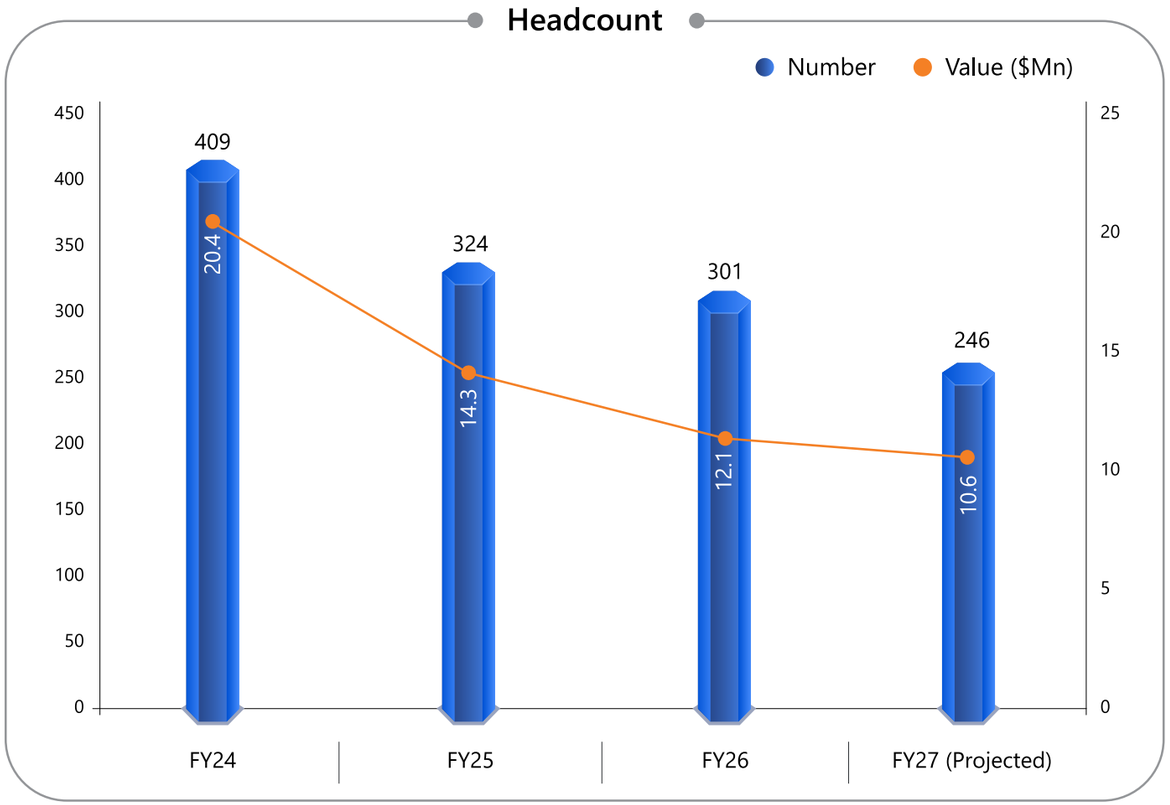
- Initiation of IND enabling studies for Target 1
- Initiation of IND enabling studies for Bispecific ADC

- PDP-716 NDA outcome
- SCD-153 Vitiligo Phase 1b/2a study initiation

- SBO-154 Phase 1a readout
- SCD-153 Phase 1b topline readout

SPARC optimized cost structure while expanding the portfolio

Achieved cost savings through lab consolidation and headcount rationalization

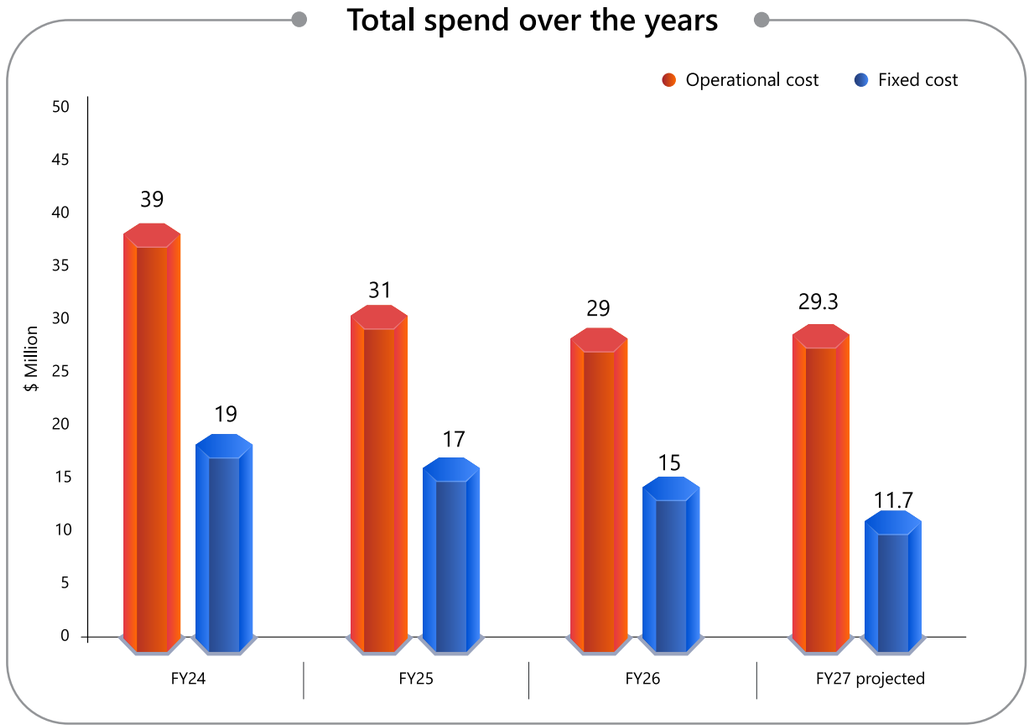


- Headcount reduction: Overall workforce to reduce by 40%, with a significant decrease in the US headcount of more than 80%
- Facility consolidation: Laboratory operations consolidated to two locations from four locations
- Business model shift: Transition from a fully captive model to a hybrid model

Focused cost optimization has led to ~ $10Mn in annual savings, strengthening operational efficiency and improving long-term financial sustainability. We will continue to optimize our cost base through judicious investments to strengthen the Core while increasing outsourcing and partnering wherever feasible

Consistent cost focus and optimized resource allocation reduces the burn

Sustained cost discipline and optimized cost allocation to extend cash runway and support growth priorities



- Streamlined clinical operations and optimized execution efficiency
- Leveraging Indian clinical trial infrastructure as a strategic advantage for cost-effective development
- While fixed costs are being streamlined, our overall spend is expected to increase in the coming year as our clinical trial expenditure increases
- SPARC currently has ~$46 Mn* debt outstanding against a fully approved debt limit of ~$125 Mn

SPARC is finalizing a resourcing plan for FY27 & FY28 involving additional promoter supported debt and internal cash accruals

*As on September 30, 2025

Clinical Programs update

sparc

SCD-153

First-in-class topical drug
for Alopecia Areata

SCD-153 overview

Novel mechanism offering class-alternative topical therapy to JAK inhibitors

Collaboration with JHU and IOCB

- Jointly developed with JHU and IOCB
- Licensed IP rights from JHU and IOCB

Novel mechanism of action

- Employs an innovative approach to address complex immune pathogenesis in diverse clinical manifestations, with potential to be a new standard of care

Topical delivery advantage

- Developed topical foam formulation for targeted delivery within the skin having potential to eliminate side effects and address the limitations of currently approved treatments

Preclinical summary

- Demonstrated hair growth in animal model of Alopecia Areata
- DMPK studies completed
- Toxicology and safety package completed

Clinical development

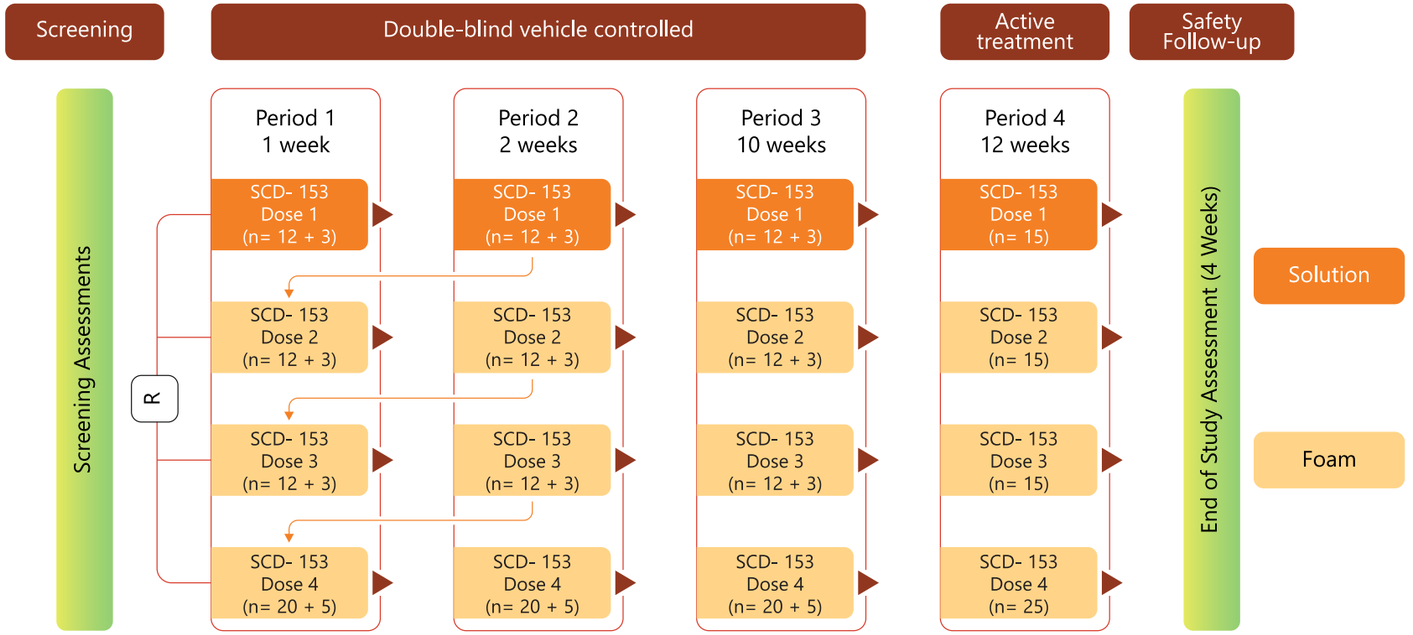
- Phase 1 single ascending dose study in healthy volunteers completed with no safety concerns
- Phase 1b study in Alopecia Areata initiated

Future steps

- Interim readout from the Phase 1b Alopecia Areata study in Q4 2026

JHU=Johns Hopkins University | IOCB=Institute of Organic Chemistry and Biochemistry | JAK= Janus Kinase | DMPK=Drug Metabolism and Pharmacokinetics

Phase 1b Mutiple Ascending Dose (MAD) Study Design



SCD-153/vehicle will be used as topical solution in cohort 1 and as foam in cohort 2 onwards

Study Population

- Adult males between 18-55 years and females between 18-45 years of age
- SALT score ≥25 to ≤90

Key Assessments

- Local skin reactions and Systemic AEs
- Efficacy assessments by Severity of Alopecia Tool (SALT) score
- Skin concentrations and systemic PK
- Biomarkers: Transcriptomics and Proteomics

R=Randomization

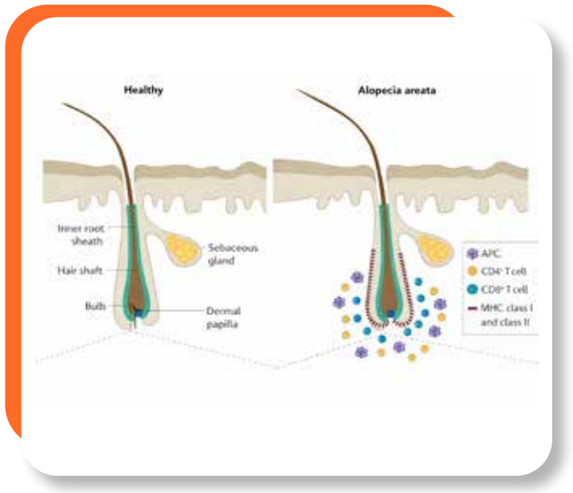
Updates and current status of Phase 1b study

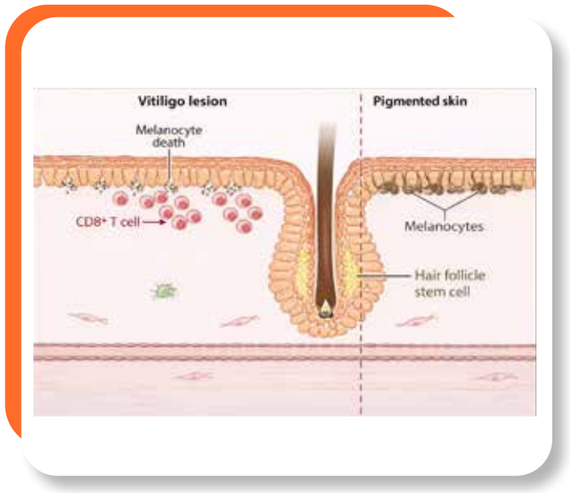
- DCGI approval received for protocol amendment of MAD study in AA patients
  - Change in formulation from solution to foam
  - Revision in baseline SALT score for eligibility
  - Waiver for review of cohort 1 safety data by CDSCO before proceeding to subsequent cohorts
- Enrolment completed in MAD Cohort 1 (N = 15); 9 patients completed 12 weeks of treatment
- DSMB review for cohort 1 completed: Recommendation to proceed to cohort 2
- Enrolment initiated in Cohort 2

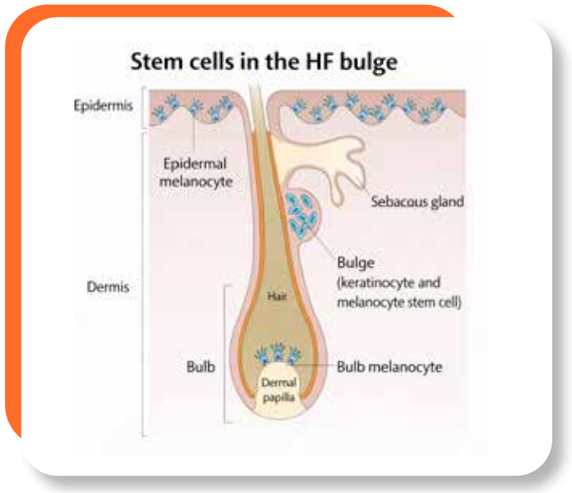
MAD=Multiple Ascending Dose | SALT=Severity of Alopecia Tool | CDSCO=Central Drugs Standard Control Organisation | DSMB=Data and Safety Monitoring Board

Next steps for SCD-153

Vitiligo is an autoimmune skin disease with similar pathogenesis as Alopecia Areata





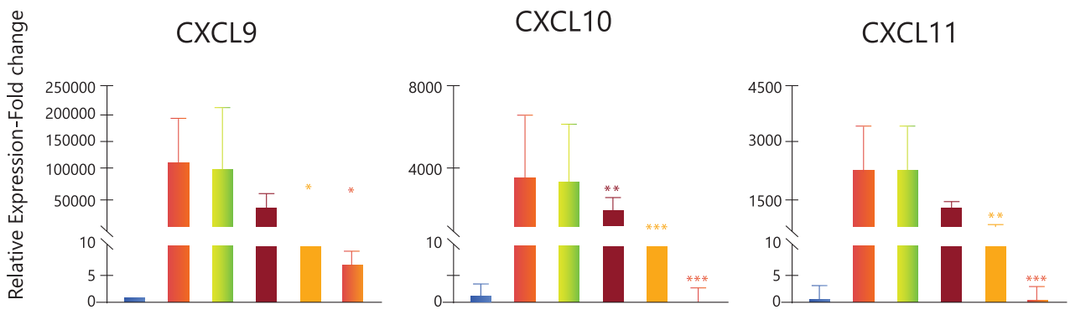


- **CD8+ cytotoxic T-cells are the major disease-causing cell-type in both diseases**
  - In AA, CD8+ T-cells infiltrate around base of the hair follicle in the dermis, while in vitiligo, they localize near melanocytes in the epidermis
  - In both diseases, the CD8+ T-cells release IFN-γ and cytolytic granules
    - In AA, it damages the HF cells causing hair loss and alteration in the hair growth cycle
    - In vitiligo, it damages the melanocytes which produces melanin and so leads to skin depigmentation
- **Both hair follicle stem cells and melanocyte stem cells (for skin pigment) are preserved in Hair Follicle bulge, suggesting potential for clinical benefit in both the diseases.**

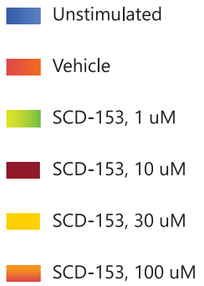
Images adapted from Picardo M et al., Nat Rev Dis Primers. 2015 ;1:15011; Pratt CH et al., Nat Rev Dis Primers. 2017;3:17011; Frisoli ML et al., Ann. Rev. Imm. 2020;38:621-648. | IFN-γ=Interferon gamma

SCD-153 inhibits chemokine expression in stimulated human melanocytes & keratinocytes

Melanocytes

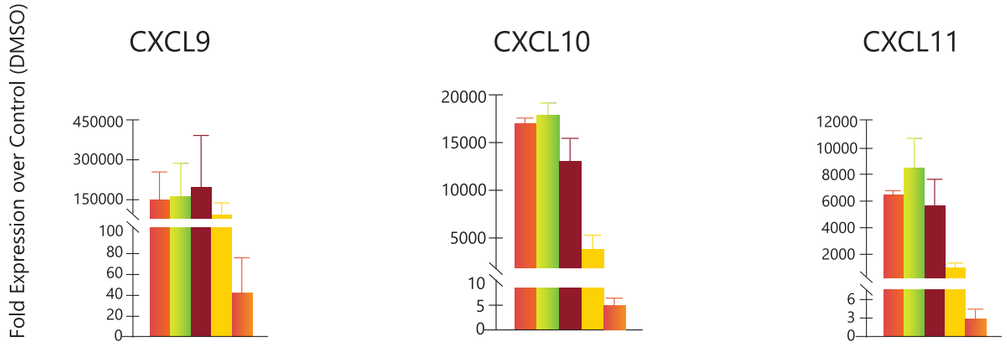


One-way ANOVA followed by Dunnet's test/Fisher's LSD test; *P<0.05, **P<0.01, ***P<0.001

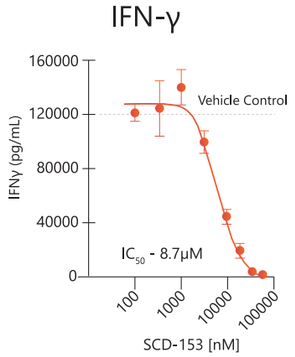


- In vitro SCD-153 inhibits the following in a dose-dependent manner:
  - IFN-γ induced expression of chemokines CXCL9, CXCL10 & CXCL11 in primary human melanocytes & keratinocytes
  - IFN-γ secretion from stimulated human PBMCs

Keratinocytes

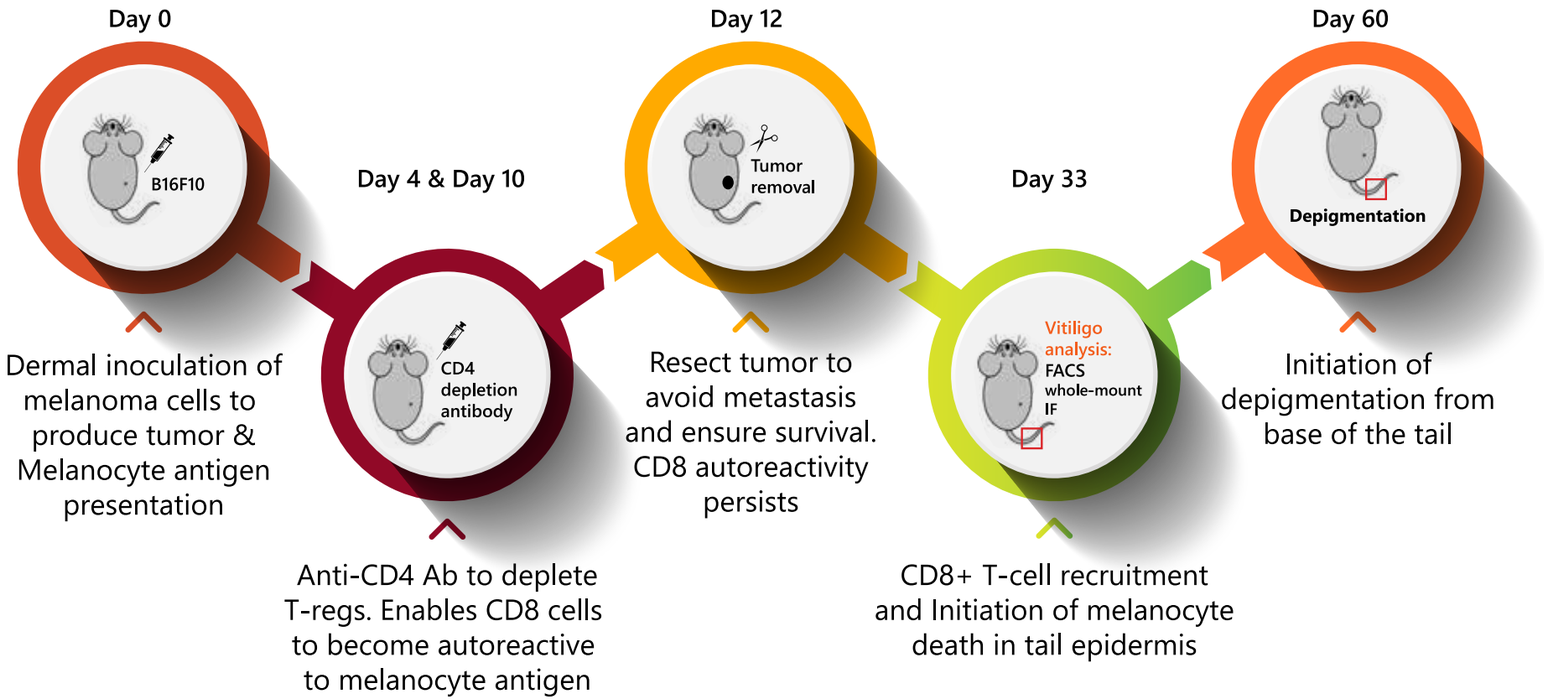


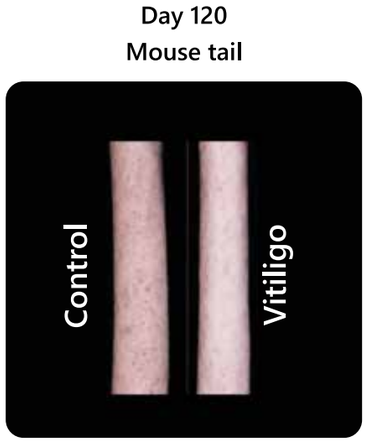
PBMCs



CXCL=C-X-C motif chemokine ligand | PBMC=Peripheral Blood Mononuclear Cells

Efficacy of SCD-153 is being evaluated in Melanoma-Treg-induced Vitiligo mouse model





Clinical study in Vitiligo is being planned

Adapted from Chen D et al., Cell Regeneration. 2022 Oct 2;11(1):31.

sparc

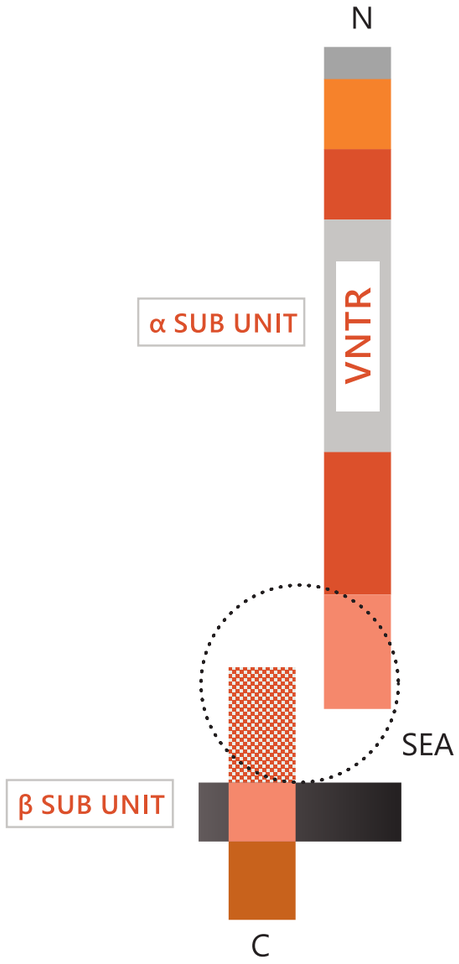
SBO-154

Anti-MUC1 ADC of MMAE

SBO-154: targets the membrane proximal SEA domain of MUC1

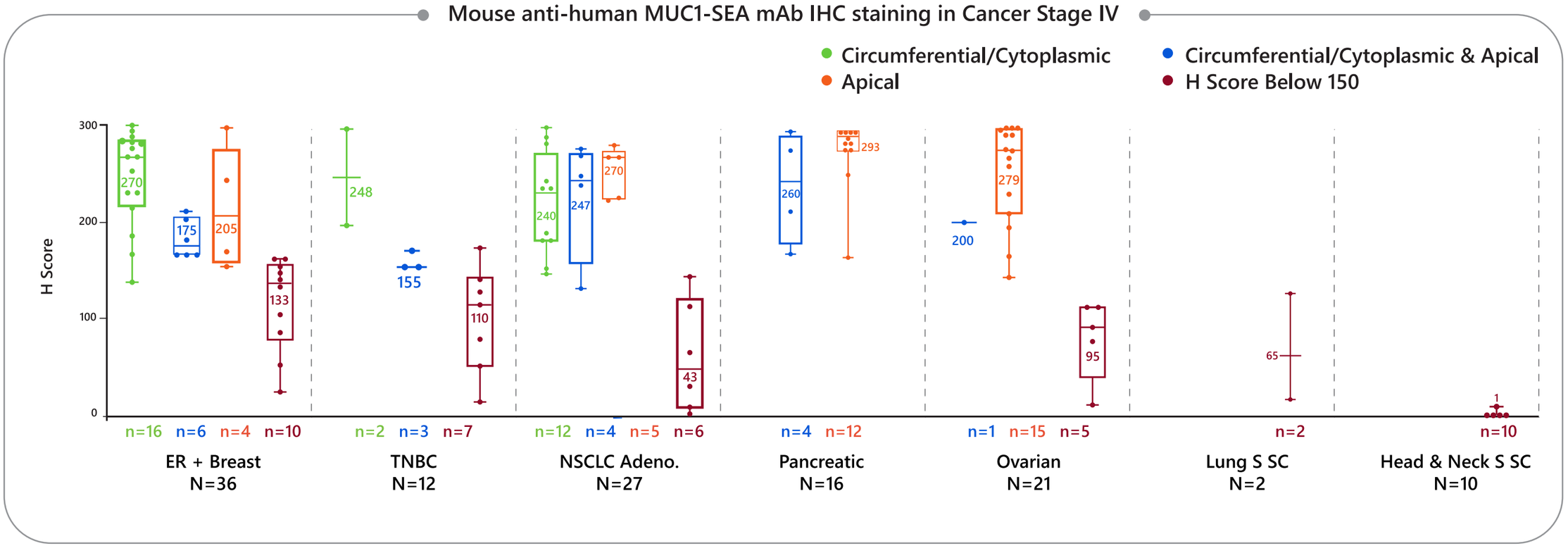
Novel approach: SBO-154 does not bind to MUC1 VNTR domains, unlikely to be subjected to a sink effect in plasma

- MUC1 is a glycoprotein antigen displayed on the cell surface as a heterodimer of extracellular α chain non-covalently but tightly associated with a transmembrane β chain
- MUC1α subunit contains a variable number of 20-amino acid long tandem repeats (VNTR)
- The carboxyl terminal portion of MUC1α and the extracellular portion of MUC1β constitute the membrane-proximal MUC1-SEA domain
- Unlike membrane-distal MUC1-VNTR, membrane-proximal MUC1-SEA domain has not been explored for therapeutic targeting
- SBO-154 represents a first-in-class MUC1-SEA targeted therapeutic agent to be developed for use in cancer therapy



SEA=Sea urchin sperm protein Enterokinase Agrin | VNTR=Variable Number Tandem Repeats

High MUC1-SEA expression level in human tumors of highly prevalent cancers



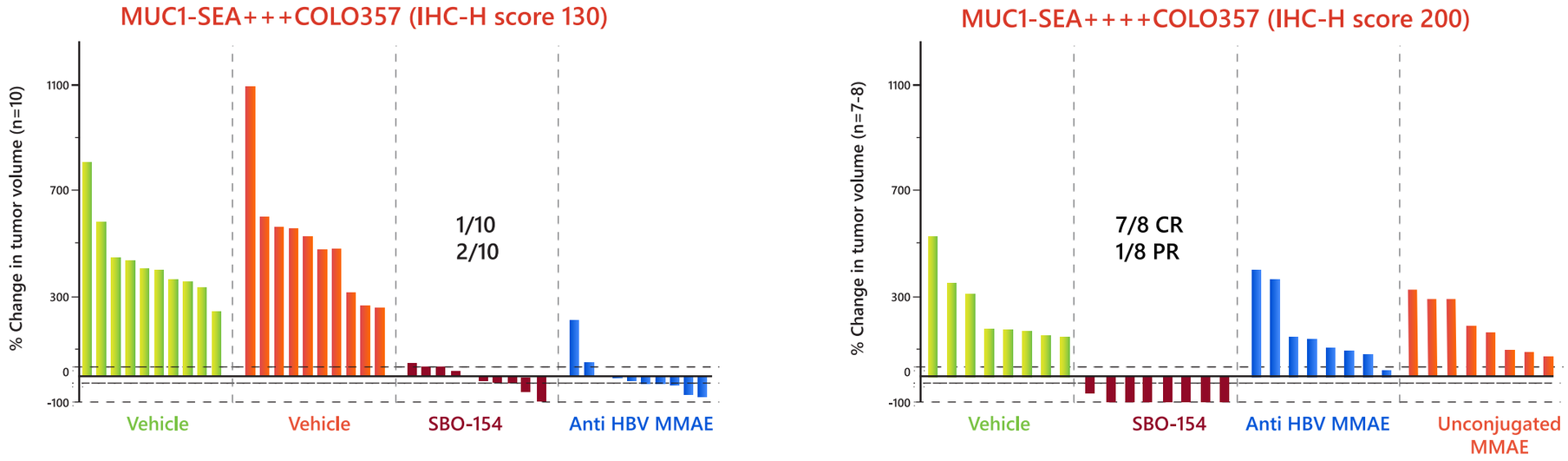
- High MUC1-SEA expression (IHC-H Score >150) in ER+ breast cancer, non-small cell lung adenocarcinomas, ovarian and pancreatic carcinomas
- Expression increases as the stage of the tumor advances
- Preclinical studies further corroborate suitability of MUC1-SEA as an ADC target and establish relevance of antigen expression level

ER+=Estrogen Receptor positive | IHC=Immunohistochemistry

In-Vivo efficacy of SBO-154 in COLO357 xenograft model

MUC1-SEA expression level is a major determinant of antitumor efficacy of SBO-154

Waterfall plots of antitumor efficacy of SBO-154 on Day 29



- Similar results were obtained with MUC1-SEA+ and MUC1-SEA+++ clones of SUM-159 TNBC, MCF7 ER+ breast carcinoma, and SKOV3 ovarian carcinoma xenografts
- Complete tumor regressions observed in over-expressing COLO357 model indicating that SBO-154 activity is linked to the density of cell surface expression of MUC1-SEA

Phase 1 study: Adaptive Dose Escalation and Expansion Design

Key inclusion & exclusion criteria

Inclusion

- ≥ 18 years,
- Evaluable disease via RECIST 1.1
- ECOG 0 – 1
- Relapsed/ not eligible to receive SoC advanced or metastatic solid tumors

Exclusion

- Pre-existing sensory or motor neuropathy (≥ Grade 2)
- Uncontrolled CNS metastases

Dose escalation (Part 1)

Q3W dosing

Backfill Cohorts

Dose 5

Dose 4

MUC1-SEA

H Score ≥150$

Establish MTD,

Recommended dose for expansion*

Dose expansion (Part 2)

Cohort 1: Non small cell lung cancer

Cohort 2: ER+ breast cancer

Cohort 3: Ovarian cancer

MUC1-SEA

H Score ≥150

Endpoints

Primary endpoints

- Safety
- Tolerability

Secondary endpoints

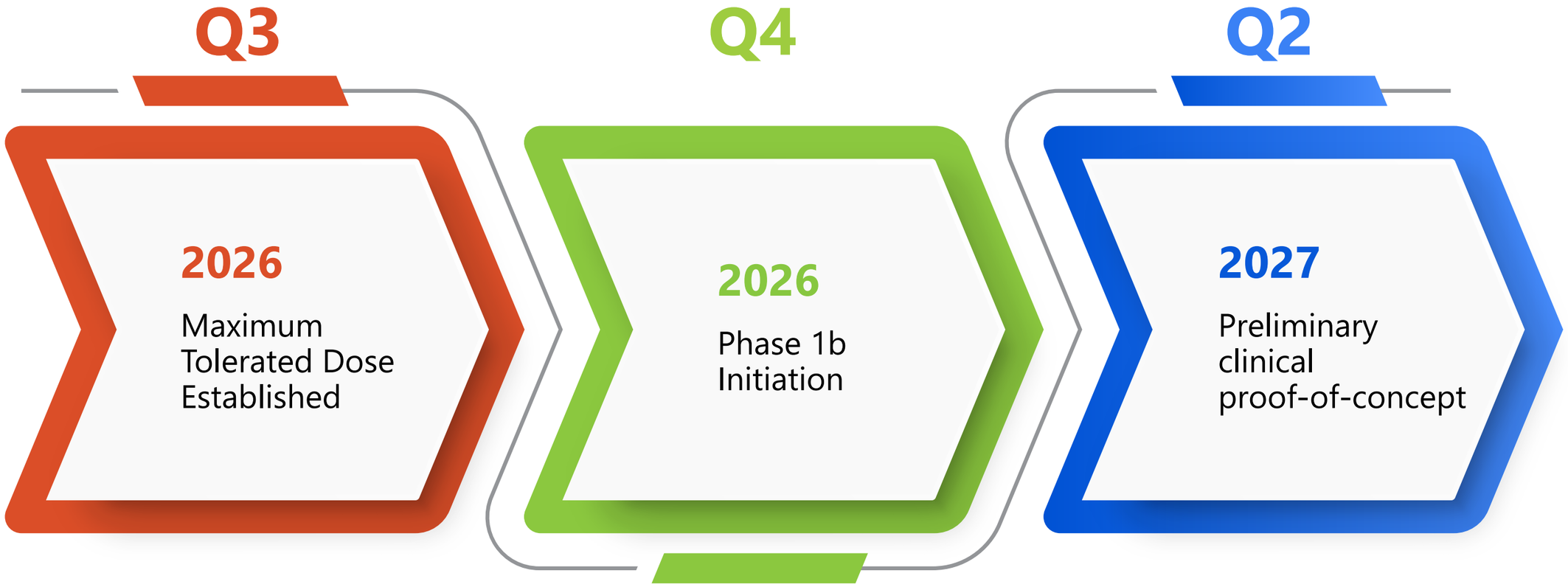
- PK
- Immunogenicity
- Anti-tumor activity
- Long term safety & tolerability

Additional intermediate dosing cohorts may be added in based on evolving safety and efficacy data | $Up to 25 subjects with NSCLC adenocarcinoma, ovarian, ER+ breast and pancreatic adenocarcinomas may be enrolled as backfill cohorts at ≤ 2 doses ≤ MTD having MUC1 SEA IHC score of ≥ 150 | *Alternative dosing schedules may be added based on the emerging PK and safety data generated through the course of the study
RECIST: Response Evaluation Criteria in Solid Tumors; ECOG: Eastern Cooperative Oncology Group; SoC: Standard of Care, CNS: Central Nervous System, | Q3W: Every 3 weeks, MTD: Maximum Tolerated Dose, HER2+: Human epidermal growth factor receptor 2; PK: Pharmacokinetics

Phase 1 study updates

- IND filed and approved by multiple regulatory agencies
- Study initiated in USA, India and Australia
- Eleven sites activated across the 3 countries
- 2 dose escalation cohorts completed
- No unexpected safety signals or Dose Limiting Toxicities observed in the initial cohorts
- Cohort 3 dosing initiated

Major upcoming milestones



Building value through portfolio, platform innovation and cost optimization

Strategic portfolio management

Optimized multi-modal portfolio aligned to global growth drivers in a narrower therapeutic field

Platform technologies creating pipeline in a product

Modular plug and play technology serving as a backbone for diverse applications

Execution focus and cost efficiencies

Execution focus while aggressively optimaizing foot print and cost

Thank You