

Corporate Overview

July 2022

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Pipeline overview & key short-term catalysts



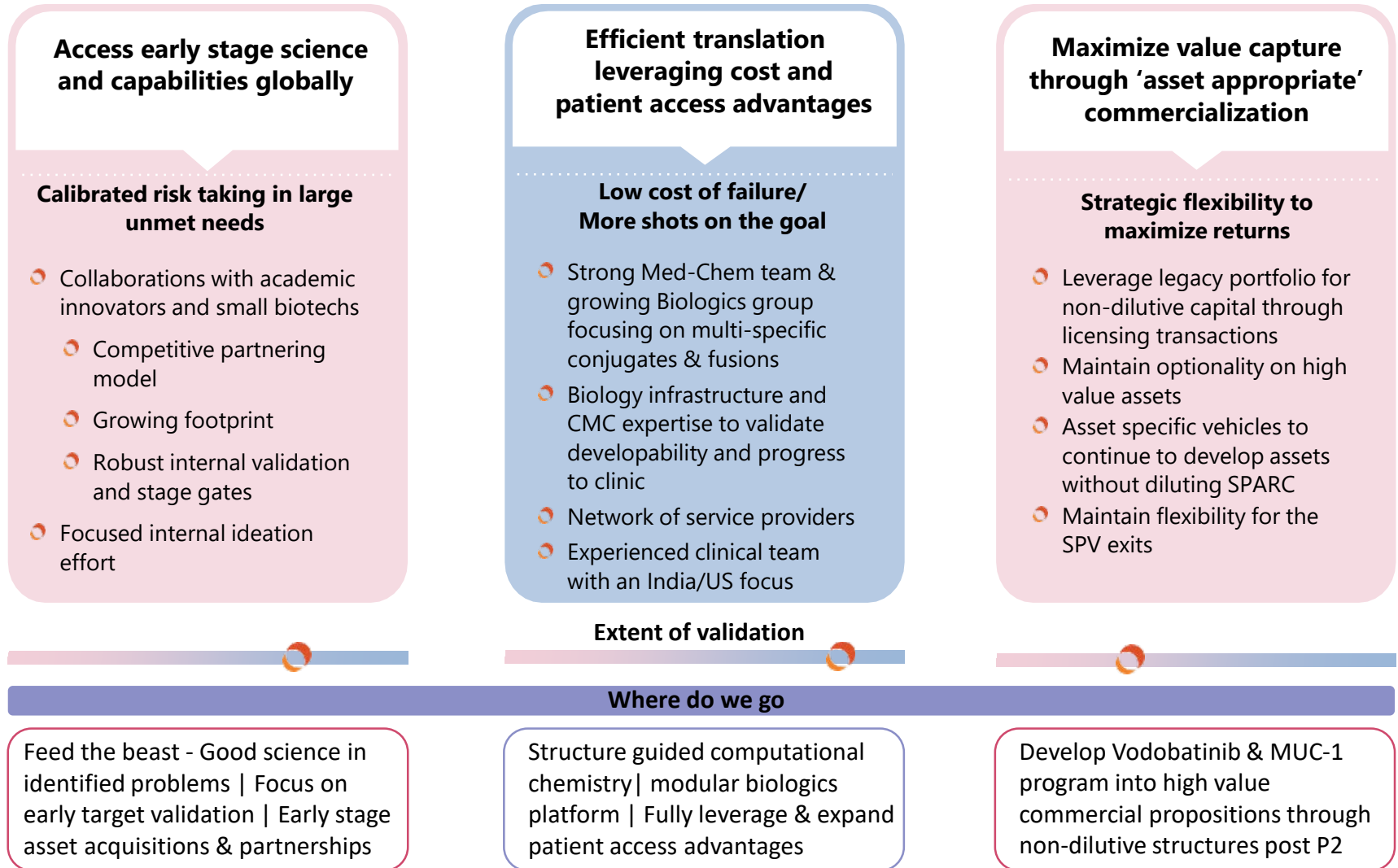
Asset / Program	MoA	Indication	Discovery	Preclinical	Phase 1	Phase 2	Phase 3/ Registration Study	Upcoming Catalyst	Partner
Vodobatinib (SCC-138)	c-ABL Inhibitor	Parkinson's Disease						PoC data from PROSEK study in 2023	
		Lewy Body Dementia ¹						PoC data in 2023	
		Alzheimer's Disease							
Vodobatinib (SCO-088)	BCR-ABL Inhibitor	Refractory CML						Pivotal data in 2024	
SCO-120	SERD	Metastatic Breast Cancer						Phase 1 data in 2023	
Vibozilimod (SCD-044)	Selective S1PR1 agonist	Psoriasis						Phase 2 data in 2023	
		Atopic Dermatitis						Phase 2 data in 2023	
		Alopecia Areata							
ADC	MUC-1	Multiple Tumors						IND Filing Targeted 2023	

Delivery Systems programs | Commercial Assets – Elepsia XR, Xelpros BAK Free, Lipodox and other platform licensing trxn + 3 NDAs planned for 2022 (2 in Ophthalmology and 1 in Neurology). **Preclinical Assets** | 10+ preclinical assets under development to ensure a robust pipeline for future growth

Neurology **Oncology** **Immunology**

1. Investigator Initiated Study | MoA = Mechanism of Action | PoC = Proof of Concept | CML = Chronic Myeloid Leukemia | SERD = Selective Estrogen Receptor Degradar | S1PR1 = Sphingosine-1-Phosphate Receptor 1
IND = Investigational New Drug | TAA-1 = Tumor Associated Antigen-1 | IBD = Inflammatory Bowel Disease

Long term intent – Realize the potential of SPARC operating model to build a high impact institution



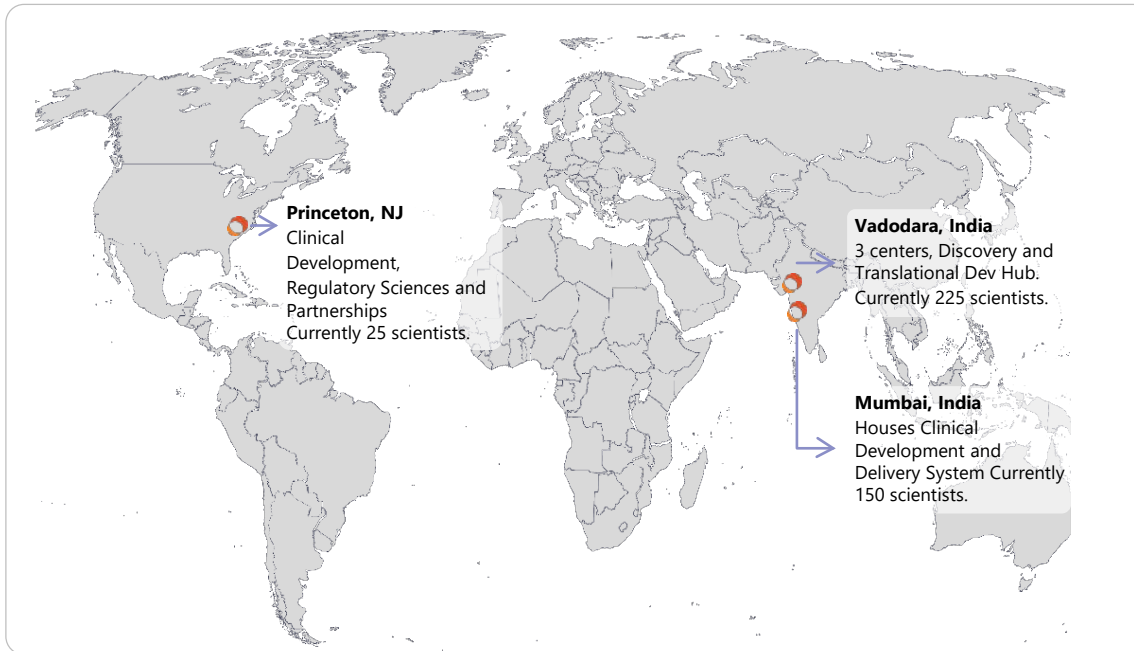
Opportunity to further extend the operating model advantages



SPARC strategic development partnerships



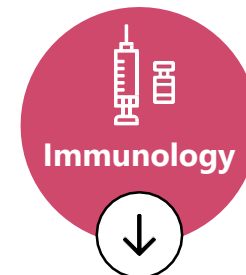
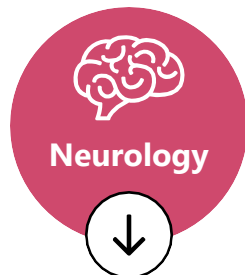
SPARC operational footprint



SPARC commercial partnerships (Sun Pharma, CMS, VisioX, Tripoint)

- Expand the pre-clinical portfolio through targeted partnerships and asset acquisitions in key focus areas
- Internalization of clinical development operations
- Increase the patient capture from India
- Create an Eastern European clinical execution hub to further expand the patient access advantage
- Start building out late stage development capability
- Fourth lab location with access to early stage biology & tax advantages for asset holding
- Build the capital base to maintain optionality going into 2023
- Start developing a commercial core for the future

Portfolio strategy focused on moving the 'standards of care' in large unmet medical needs

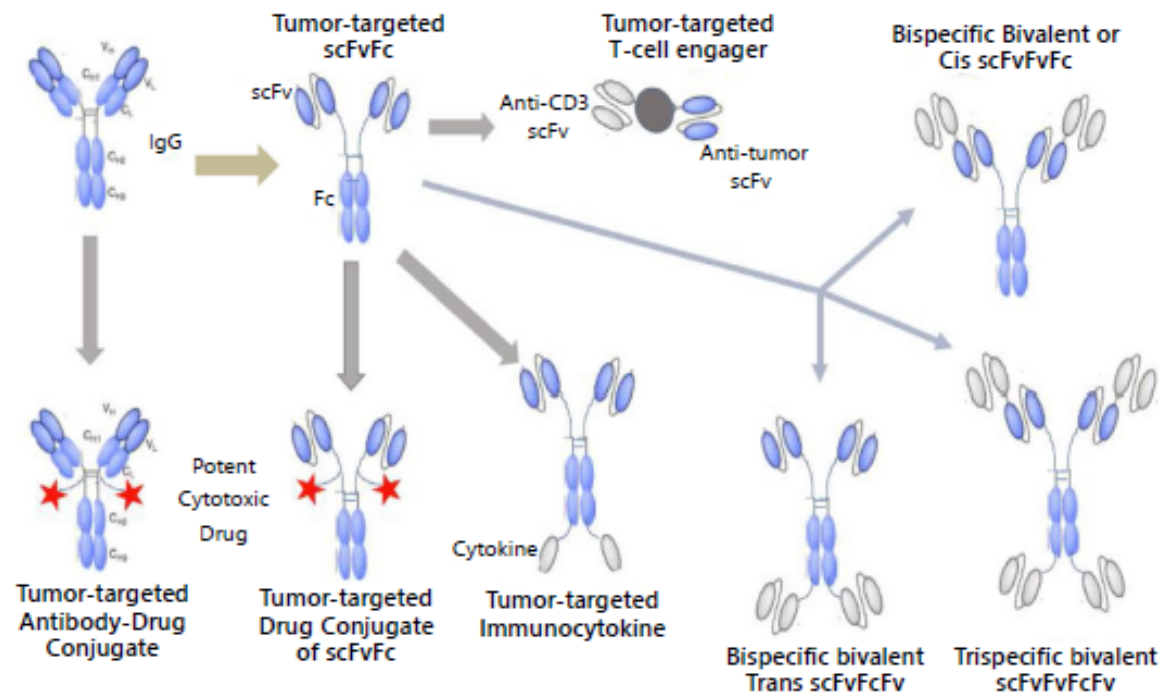


Focus area	Neurodegenerative diseases	Treatment resistance	Autoimmune disorders
Rationale	<ul style="list-style-type: none"> Stagnant standards of care in past 10- 15 years New breakthroughs in understanding disease biology offering viable targets and biomarkers Advanced imaging markers 	<ul style="list-style-type: none"> Large basic science effort leading to easier availability of testable ideas Abbreviated regulatory pathways and potential for premium pricing 	<ul style="list-style-type: none"> Limited efficacious oral options Local delivery to facilitate therapeutic window Synergistic MoA combinations
Areas of current interest	<ul style="list-style-type: none"> Explore the potential of c-Abl inhibition in neuro-degenerative diseases Invest in other critical drivers of neuro-degeneration (which can be potentially synergistic to Abl) Autophagy modulation 	<ul style="list-style-type: none"> New molecular pathways of resistance to emerging block-buster categories Tumor associated antigens for ADCs and T-Cell engagers Target combinations for Bi-Specifics 	<ul style="list-style-type: none"> Endogenous immune regulators Regulators of the inflammasome pathway Technologies for topical delivery Synergistic mechanisms

Ensure molecular clarity, tractability and viable clinical pathways before committing significant investments

Portfolio strategy focused on building platforms for 'molecular engineering' precision medicine

- Focus on modular "plug & Play" platforms which can deliver multiple products to the pipeline
- Immunofusions combining Tumor Associated Antigens with immuno-enhancers or checkpoint blockers in cancer therapy
- Immuno inhibitory/anti-inflammatory immuno-fusions for auto-immune diseases
- Antibody drug conjugates for targeted delivery of toxins or targeted therapies with difficult safety margins
- Small molecule ligands of tumour specific antigens to deliver combination payloads
- Reduces the time to clinic significantly by cutting the discovery cycle



SPARC's investments in developing viable components and engineering capability can lead to multiple clinical candidates in the short to medium term

Summary – 5 key drivers of value



4 Clinical Stage Programs Targeting Areas of High Unmet Need

- Targeting large addressable patient populations with USD 20Bn+ combined peak sales potential in 6 indications within Oncology, Neurology, and Immunology



Discovery & Development Across Validated & Novel Biology in Order to Balance the Risk

- Multi-modal portfolio covering small and large molecules and conjugated entities
- 10+ preclinical programs including an ADC program expected to enter the clinic in 2023



Proven High Quality R&D Organization with Capital-Efficient Global Operations

- 350+ scientists across 4 research centers including USD 400Mn invested to date
- 2 USFDA approvals for internally developed assets
- 3 NDAs targeted for submission in 2022



Highly Flexible Model to Maximize Shareholder Value

- Partnerships to maximize large commercial potential and provide non-dilutive capital
- Maximize multi-TA opportunity and preserve optionality for spin-offs



Experienced Management Team and Globally Recognized Scientific Advisory Board

 Bristol Myers Squibb™

 MERCK



 QUINTILES

 Schering-Plough

 Wyeth

 NOVARTIS

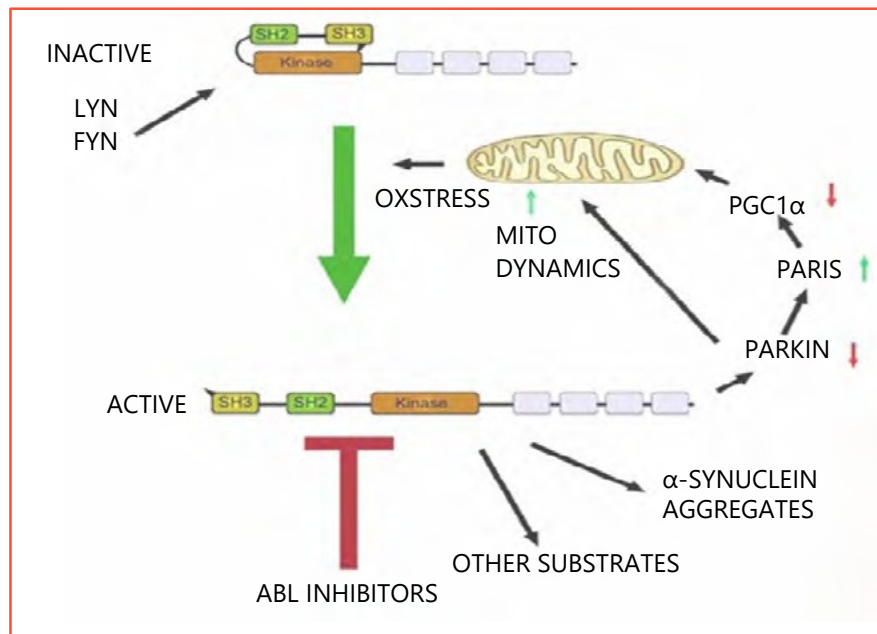
Vodobatinib for Neurodegenerative Diseases (SCC-138)

A potential first-in-class disease modifying therapy

Vodobatinib for neurodegenerative diseases

Optimal agent to test the c-Abl hypothesis

Mechanism of Action of c-Abl inhibition



Reduces neuronal toxicity caused by the aggregated neurotoxic proteins

- Vodobatinib is a potential first-in-class c-Abl inhibitor for Parkinson's disease
- Augments autophagic flux and prevents inactivation of Parkin-mediated mitochondrial quality control
- Reduces α-synuclein inclusions
- Sub-nanomolar potency against human c-Abl
- Very limited off-target activity, leading to improved safety profile
- Robust brain penetration (Brain/ Plasma levels around 0.9)

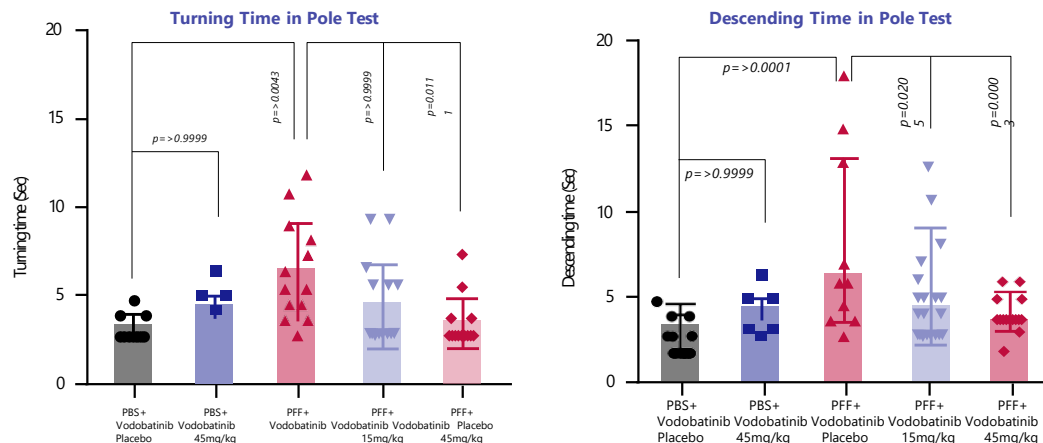
Selective Abl inhibition

Family	Kinases	IC ₅₀ (nm)
Abl	Abl (Abl-1)	0.9
	Arg (Abl-2)	0.8
SFK	Src	90.0
	Fyn	18.0
	Hck	54.0
	Lck	17.0
	Lyn	18.0
	Yes	28.0
	PTK5	3.0

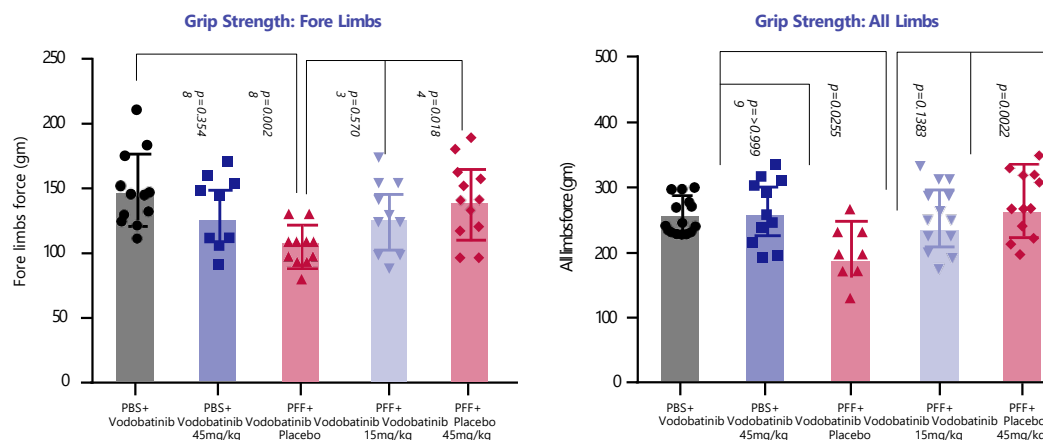
Behavioral assessments in the PFF- induced mouse model

- In the MPTP¹ mouse model, Vodobatinib prevents neuronal degeneration in substantia nigra
- In the PFF² induced mouse model, vodobatinib shows target engagement, reduction in Serine 129 phosphorylation of α -Synuclein, preservation of dopaminergic neurons and clinical improvement in motor and cognitive functions
- In the AAV³ driven rat A53T α -synuclein model, vodobatinib shows neuroprotection

Vodobatinib at 45 mg/kg improves PFF-induced movement disorder-related deficits in Turning Time and Descending Time in the Pole test

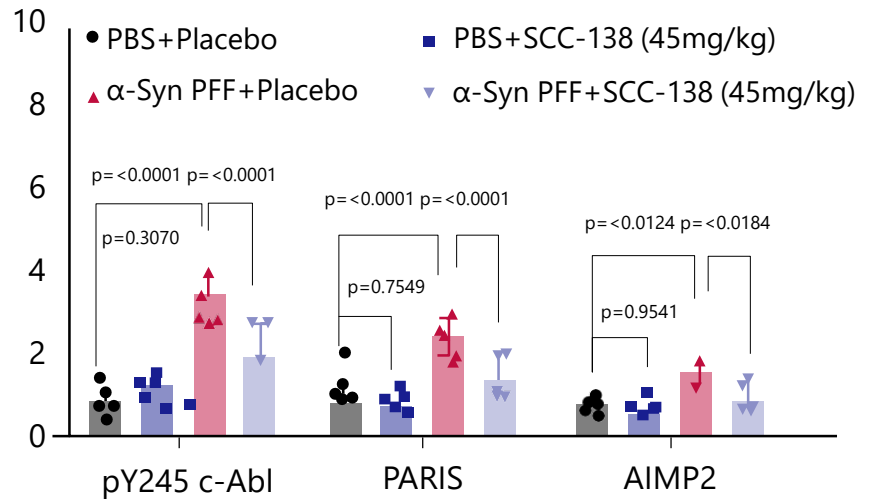
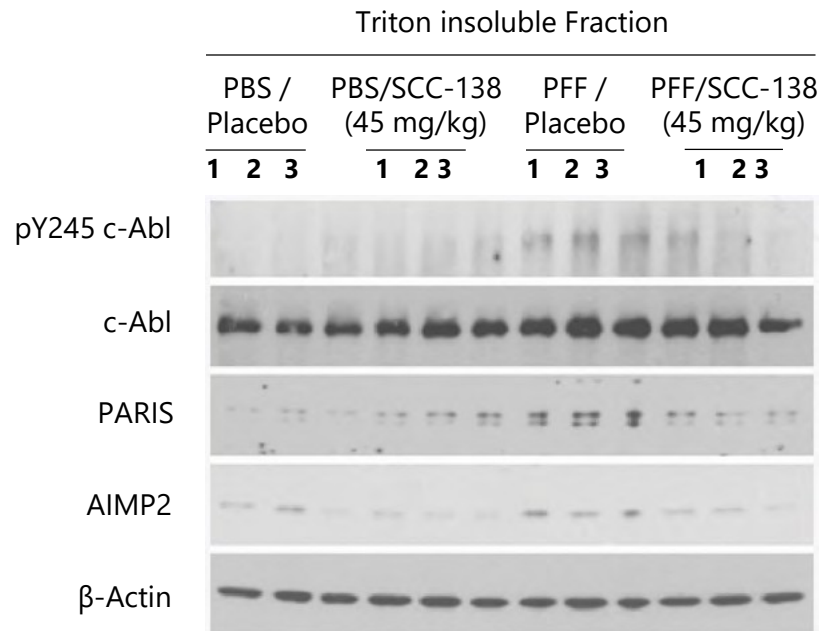


Vodobatinib treatment improves PFF-induced deficits in Grip Strength



1. Data generated in-house | 2. Study conducted at the Ted Dawson Lab, Johns Hopkins University | 3. Study conducted by Atuka Inc.
Unpublished data; not to be replicated or shared | PBS = Phosphate-buffered saline | PFF = Preformed fibril

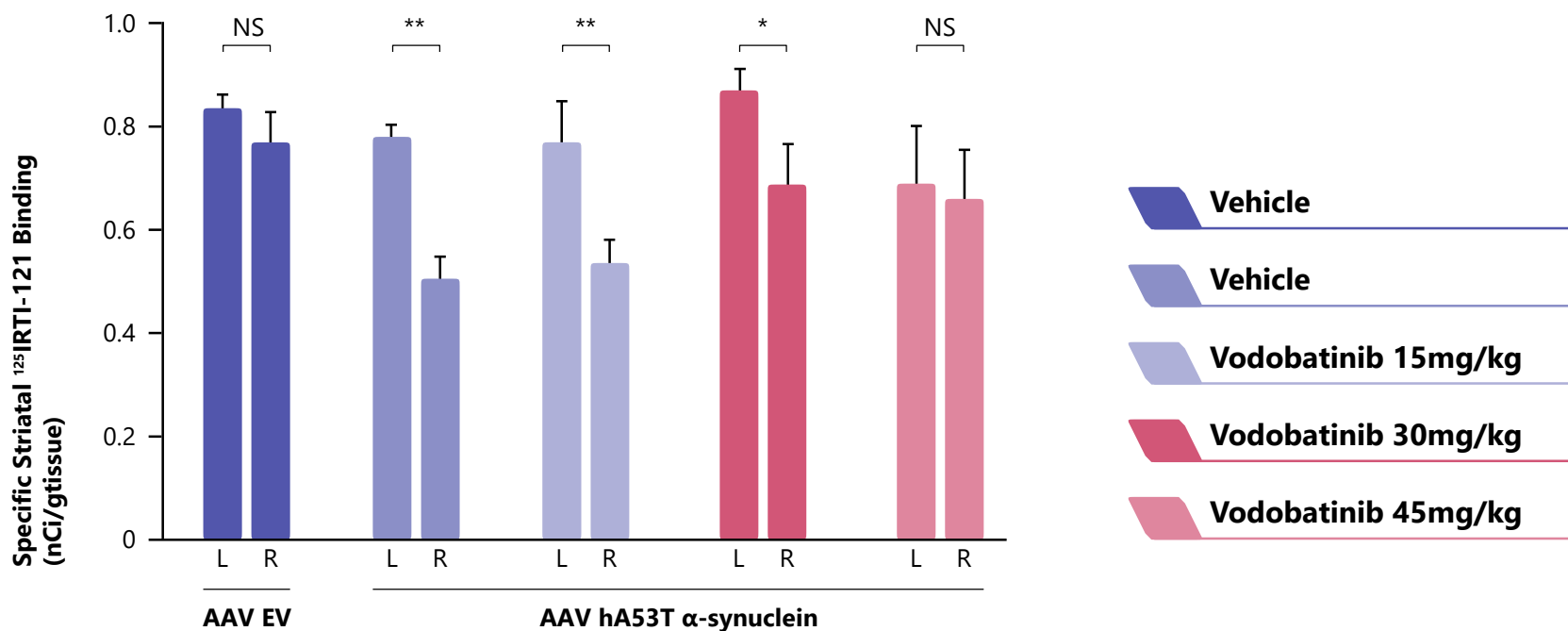
Vodobatinib reverses PFF-induced increases in key biomarkers



Vodobatinib treatment prevents PFF-induced increase in midbrain in:

- Phosphorylated Tyr245 cAbl
- Expression of PARIS and AIMP2

Vodobatinib protects dopaminergic neurons in the AAV mutant α -Synuclein (hA53T) rat model – dopamine transporter expression



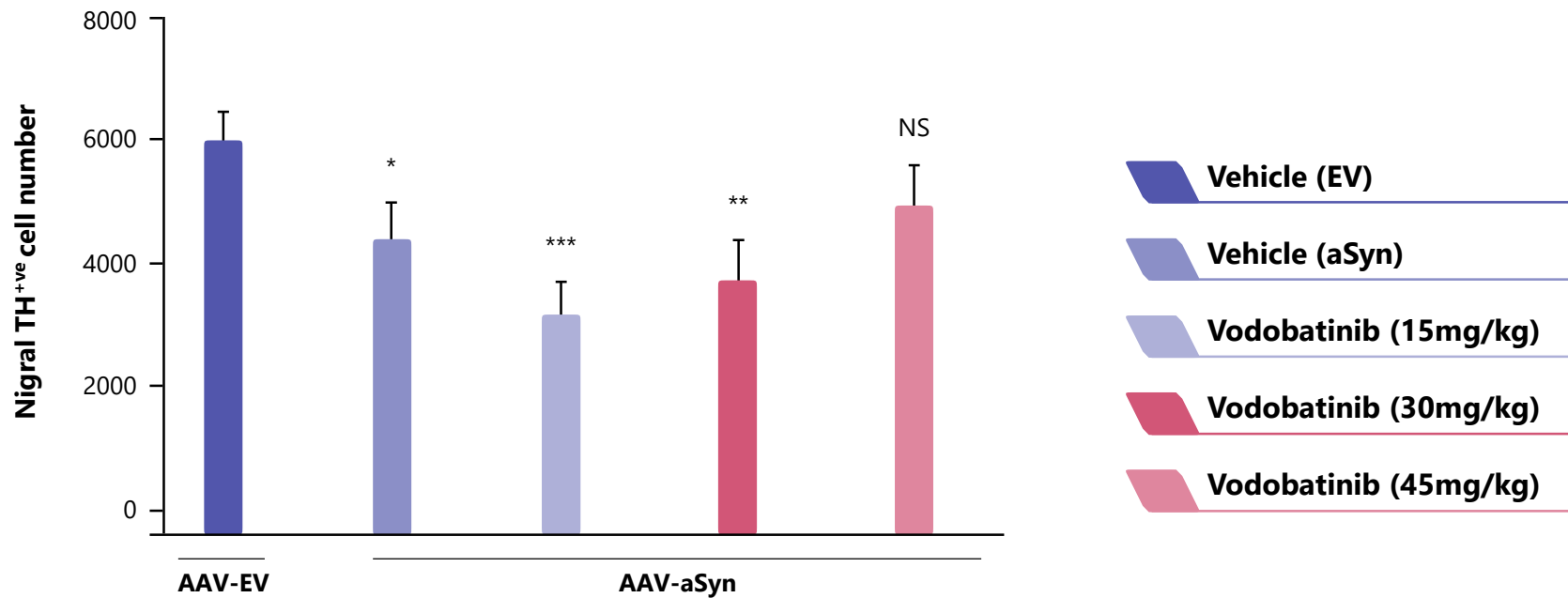
NS: $p > 0.05$; * $p < 0.05$; ** $p < 0.001$ versus the un-operated (contralateral) hemisphere. Two-way ANOVA with Fisher's LSD post-hoc test

- Vodobatinib treatment protects against dopaminergic neuronal loss measured by radiolabeled ¹²⁵I labeled RTI-121 binding in the striatum
 - Comparison of un-operated left hemisphere (L) and operated right hemisphere (R, injected with & expressing the AAV) shows that 45 mg/kg doses provides protection of dopaminergic neurons

Study conducted by Atuka Inc. | Unpublished data; not to be replicated or shared.

Vodobatinib protects dopaminergic neurons in the AAV mutant a-Synuclein (hA53T) rat model – tyrosine hydroxylase expression

Nigral TH+ve Cell count (operated side)



NS $p > 0.05$; * $p < 0.05$; ** $p < 0.01$; *** $p < 0.0001$. One-way ANOVA with Fisher's LSD post-hoc test versus Empty Vector, vehicle-treated

- Vodobatinib treatment protects against loss of TH+ve cells in the **substantia nigra** in the AAV-injected right hemisphere at the top 45 mg/kg dose (Inset is NeuN staining)

Vodobatinib met the brain exposure targets in early clinical studies

Summary of completed toxicology, safety pharmacology and clinical studies

Preclinical toxicology update

- Acute tox in mouse and rat by oral route, and in rat by ip route
- Repeat dose oral tox in rat (upto 6 months) and beagle dog (upto 9 months)
- Genotoxicity (In vitro Ames' Test and In vivo mouse micronucleus study)
- Repro toxicity
- Safety Pharmacology, including CVS safety

Clinical summary

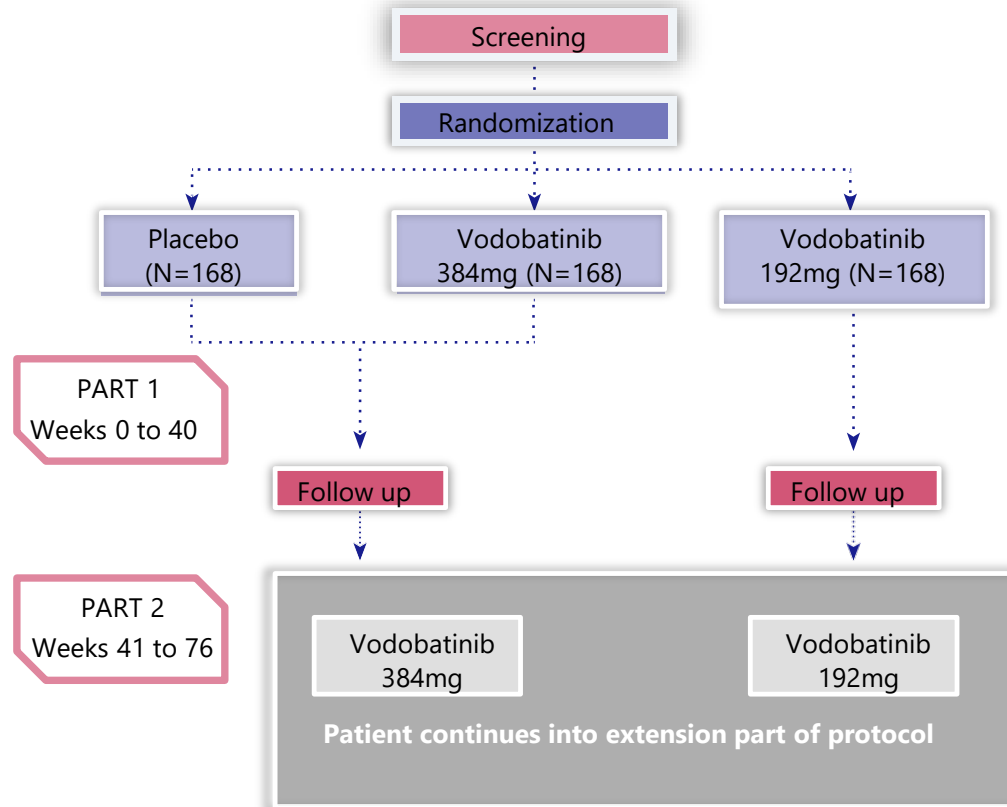
- Phase 1 completed in healthy subjects, PD subjects up to 384mg
 - Overall well tolerated
 - PK suggests adequate brain penetration over 24 hours

Trial	Population	Status	Safety findings
Phase 1 MAD	PD any stage	cohorts of 8 subjects each on 14 days of Vodobatinib or placebo capsules (6:2 randomization) 6, 12, 24, 48, 96, 192, 384mg	Well tolerated
Phase 1	Healthy men	48, 192mg, 384mg x7 days with 24 hours of CSF sampling on day 7. Study complete	Mild AEs
Phase 1 Crossover study	18 Healthy subjects per cohort	192mg powder vs 192 mg capsule 384mg powder vs 192mg capsule 384mg powder fed vs fasting	No significant concerns

Vodobatinib for Parkinson's Disease



Recruitment on track to achieve Phase 2/PROSEK enrollment target in 2022



PROSEK

- 84 sites across US, Europe and India functional; recruitment ongoing to complete enrollment in 2022
- Over 50% patients randomized (N=218)
- Phase 2 readout expected in 2023

Primary outcome

- Change in MDS-UPDRS Part 2 + Part 3 from baseline to end of treatment

Secondary outcomes:

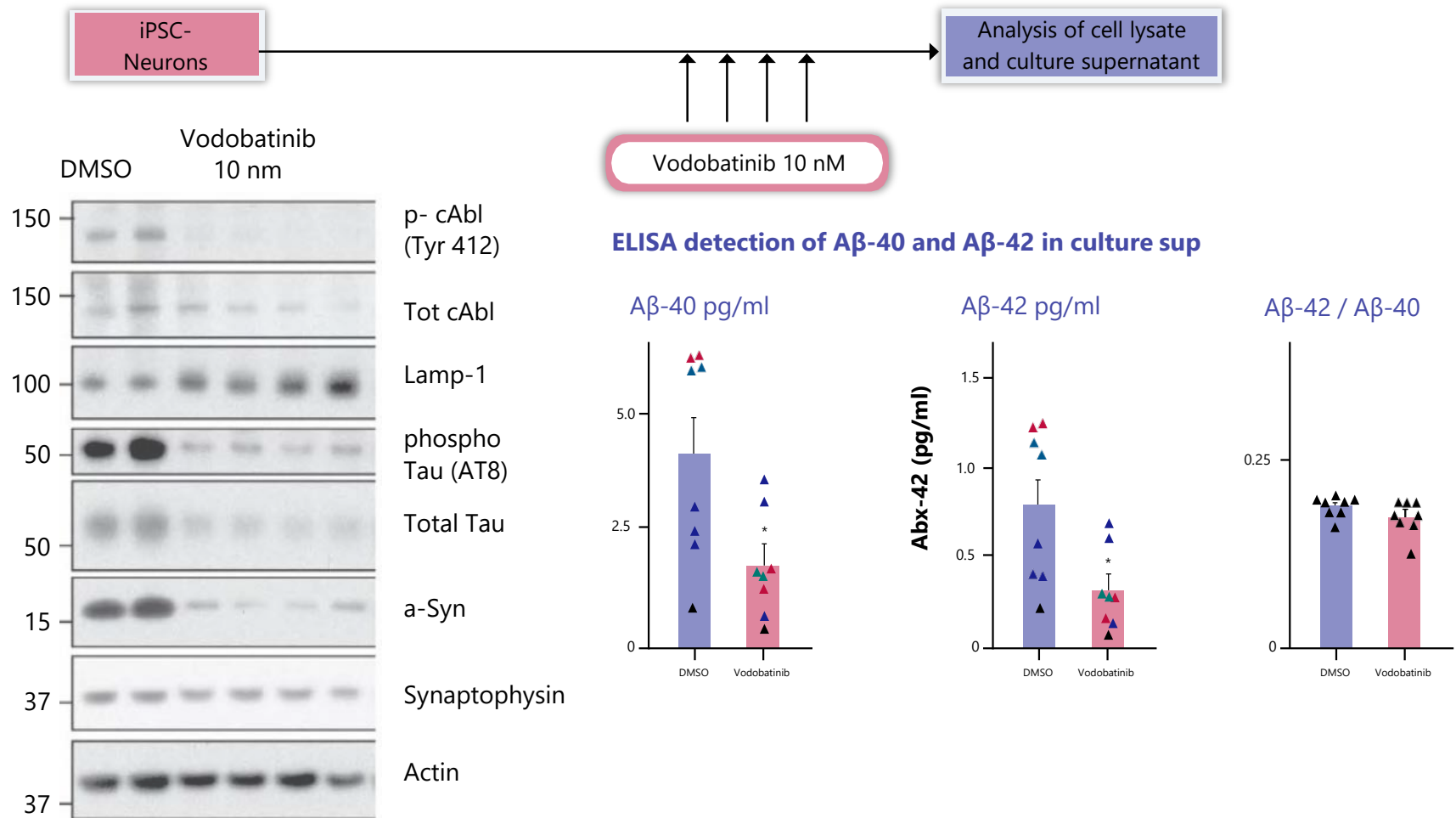
- Time to start of symptomatic medication
- CGIS - clinician global impression of severity
- PK/PD correlations

Exploratory outcomes:

- DaT SPECT at beginning (in all subjects for eligibility) and end
- Skin biopsy for synuclein deposition at Baseline and Week 36
- Smartphone-based measure of motor performance
- Exploratory CSF markers

Opportunities beyond Parkinson's Disease

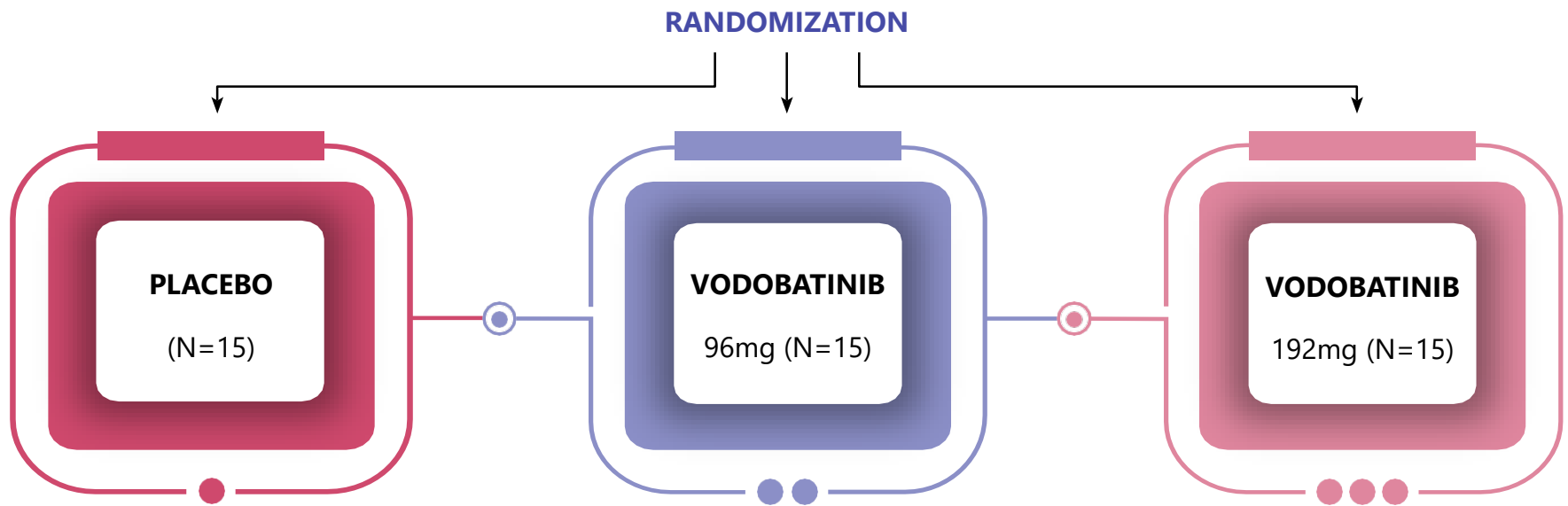
Vodobatinib reduces the intracellular load of potentially toxic proteins in iPSC-induced neurons



Opportunities beyond Parkinson's Disease

Dementia with Lewy Bodies offers an immediate next opportunity

- DLB is a neurodegenerative condition with progressive cognitive impairment, hallucinations and parkinsonism
 - Estimated to affect about 1.4 million people in the USA*
 - 2nd most common cause of dementia in the elderly
- Strong overlap with Parkinson's Disease
- Synucleinopathies with Lewy Bodies seen on autopsy. Pathophysiology similar to PD suggesting potential efficacy in DLB
- Investigator-initiated trial in collaboration with Georgetown University, Washington on-going in subjects with DLB



*<https://ghr.nlm.nih.gov/condition/dementia-with-lewy-bodies>

Vibozilimod (SCD-044) - A Selective S1PR1 Agonist

A safer alternative to JAK inhibitors

Vibozilimod (SCD-044)



An opportunity to improve oral standard of care in dermatology

Vibozilimod is a Best-in-Class S1PR1 modulator with excellent safety

S1PR1 Modulator Landscape

- Fingolimod is the First-in-Class S1PR agonist approved, but being a non-selective modulator, is associated with serious cardiac side-effects
- Multiple S1PR1 modulators are approved (siponimod and ozanimod) for non-dermatology indications; vibozilimod has opportunity to lead the field in dermatology
- Recent safety concerns related to JAK inhibitors (including topical/locally delivered agents) increase the significance of S1PR1 agonists as a 'class alternative' in several autoimmune disorders, particularly in dermatology

Vibozilimod (SCD-044)

- Developed in collaboration with a French biotech company, Bioprojet – SPARC in-licensed Bioprojet's share of IP in 2019
- Highly-selective for S1PR1 over S1PR2 and S1PR3, which can be associated with serious side effects
- Established preclinical and early clinical validation
- Currently targeting atopic dermatitis, psoriasis and other autoimmune disorders
- Potential synergy with other mechanisms in IBD – like IL-23 blockade

S1PR1 agonists	EC ₅₀		
	S1PR1	S1PR3	S1PR5
Vibozilimod ¹	0.2	>10,000	9
Fingolimod ¹	0.4	7.7	2.2
Ozanimod ¹	1.9	>10,000	3.5
Ponesimod ¹	~1	NA	10.7
Etrasimod ¹	1.5	~1000	0.7

Vibozilimod licensed to Sun Pharma with around ~50% economics retention

Vibozilimod (SCD-044)



Pharmacodynamics and safety established in Phase 1 study

Multi-part Phase 1 study completed in healthy volunteers

Single Ascending Dose

- Six dose levels in males and one dose level in females
- ~55% lymphocyte count decrease following 1 mg dose

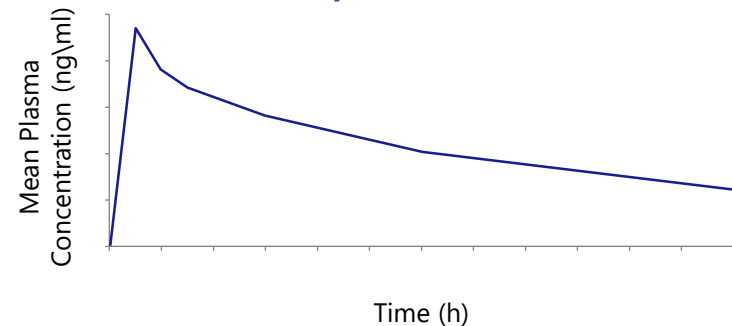
Multiple Ascending Dose

- Four dose levels including two dose up-titration schemes in males and one dose up-titration scheme in females
- ~60% lymphocyte count reduction observed at 1 mg dose with asymptomatic bradycardia
- Reduction in lymphocyte count confirms potential efficacy of vibozilimod

Lymphocyte count reduction¹



PK profile¹

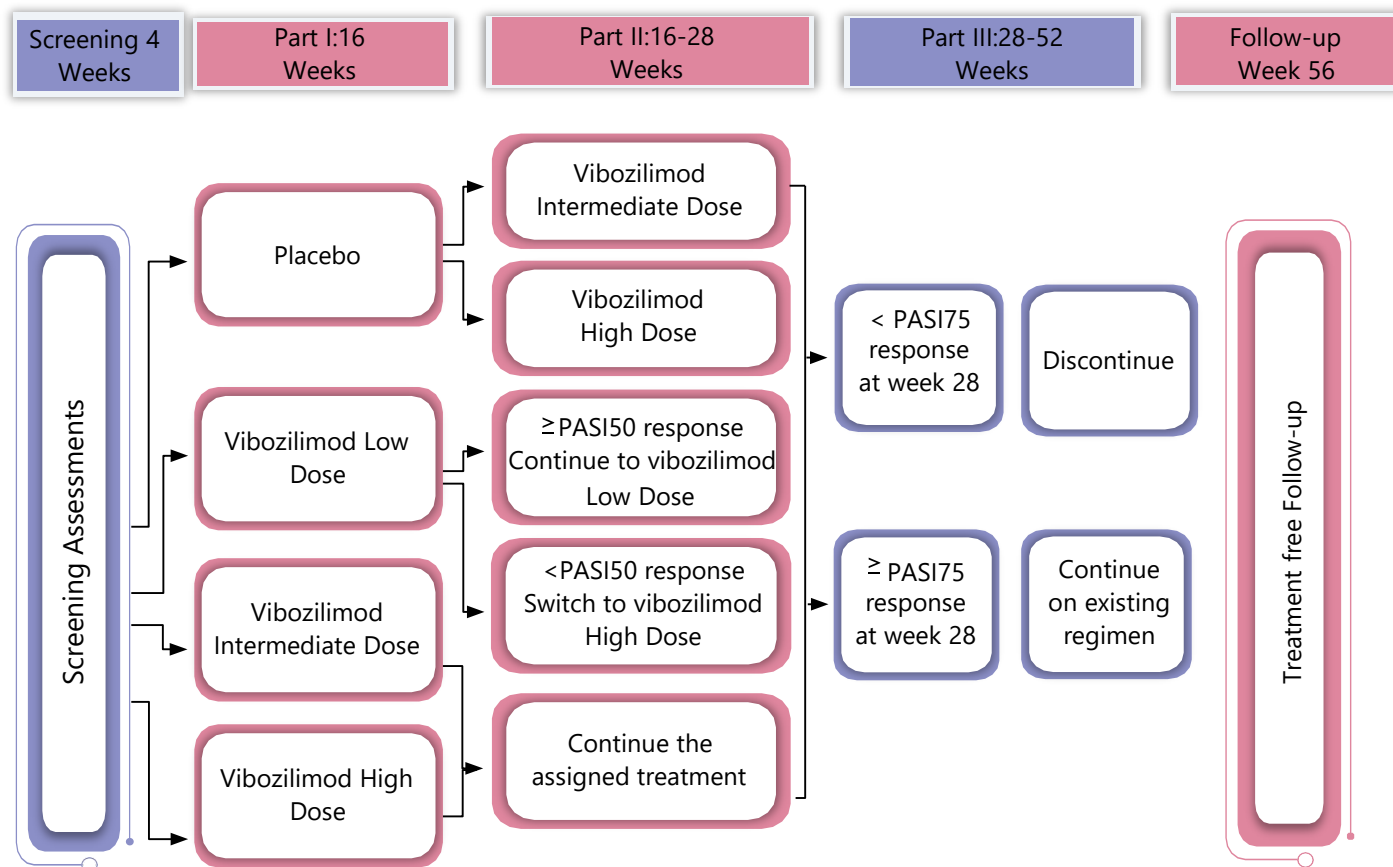


1. Phase 1 part 1 SAD study, 1 mg dose. | Vibozilimod (SCD-044) licensed to Sun Pharmaceutical Industries Limited | PK = Pharmacokinetic

Vibozilimod (SCD-044) for psoriasis



Clinical proof-of-concept by 2023

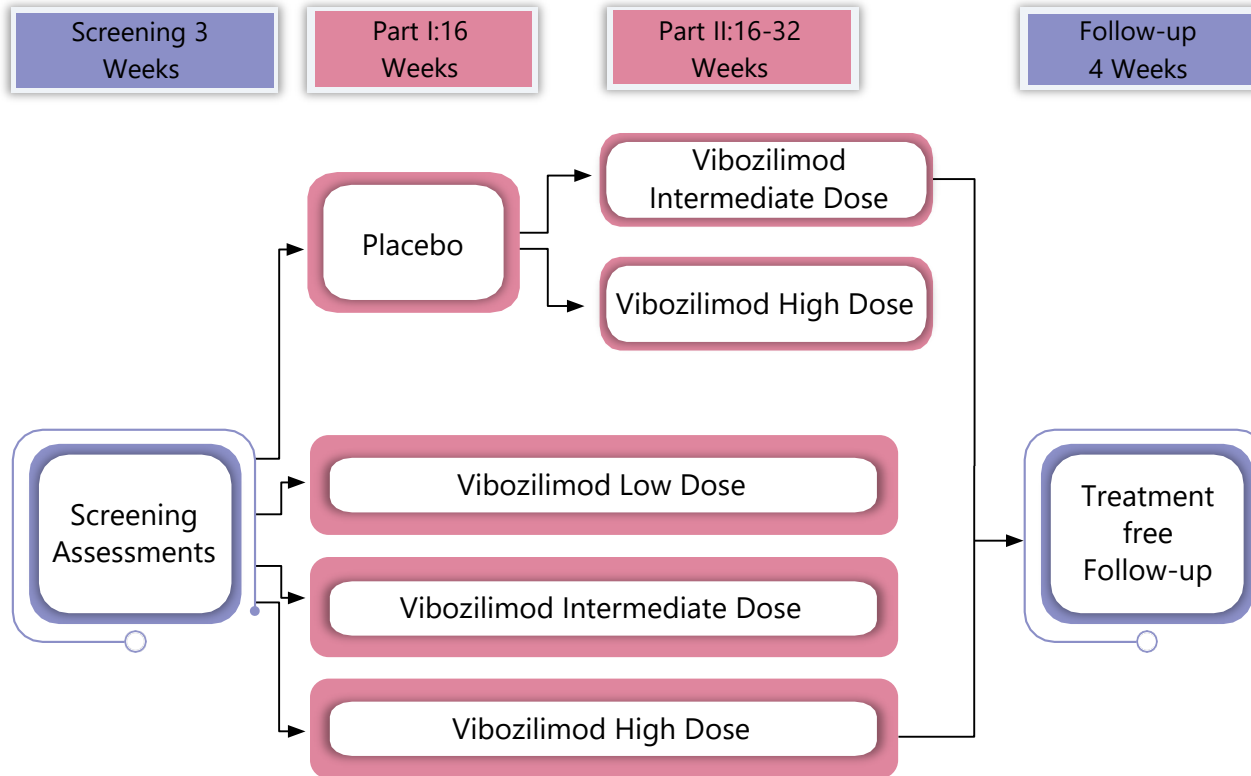


- Primary endpoint – Proportion of patients with PASI75 response at week 16
- 240 patients across three dose levels and placebo. Currently in early stage ramp-up
- Study now enrolling in the US. Expected to accelerate expansion to Latin America and Europe to accelerate in the coming months

Vibozilimod (SCD-044) for atopic dermatitis



Clinical proof-of-concept by 2023



- Primary endpoint – Proportion of patients with EASI-75 response at week 16
- 240 patients across three dose levels and placebo. Currently in early stage ramp-up
- Study now enrolling in the US. Expected to accelerate expansion to Latin America and Europe to accelerate in the coming months

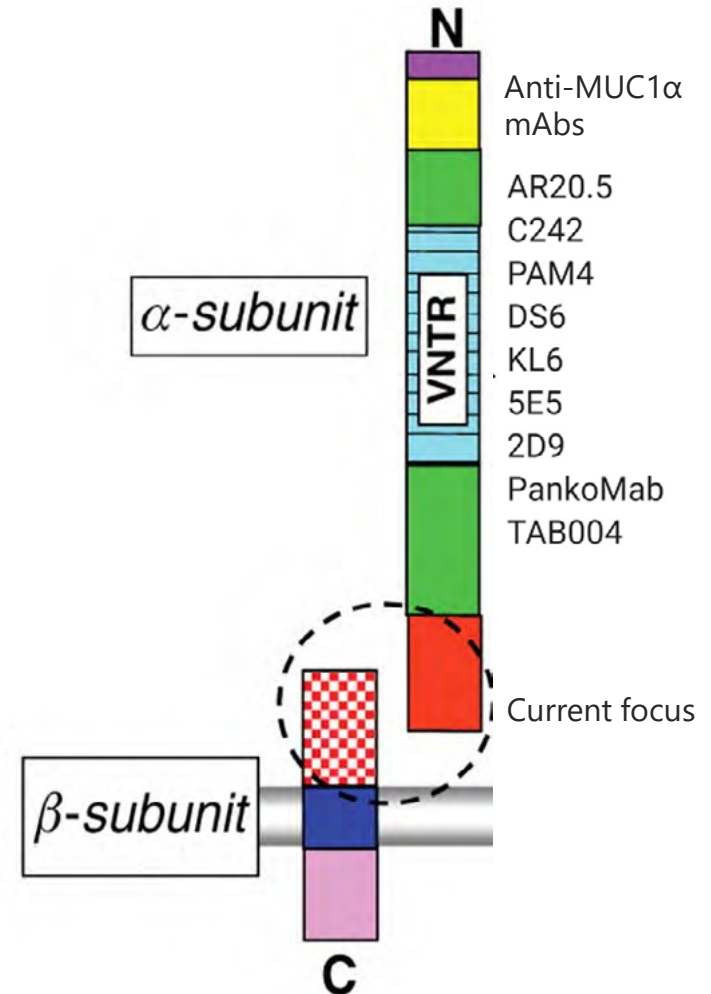
Anti MUC-1 Asset

Targeting an antigen expressed in a wide spectrum of tumors

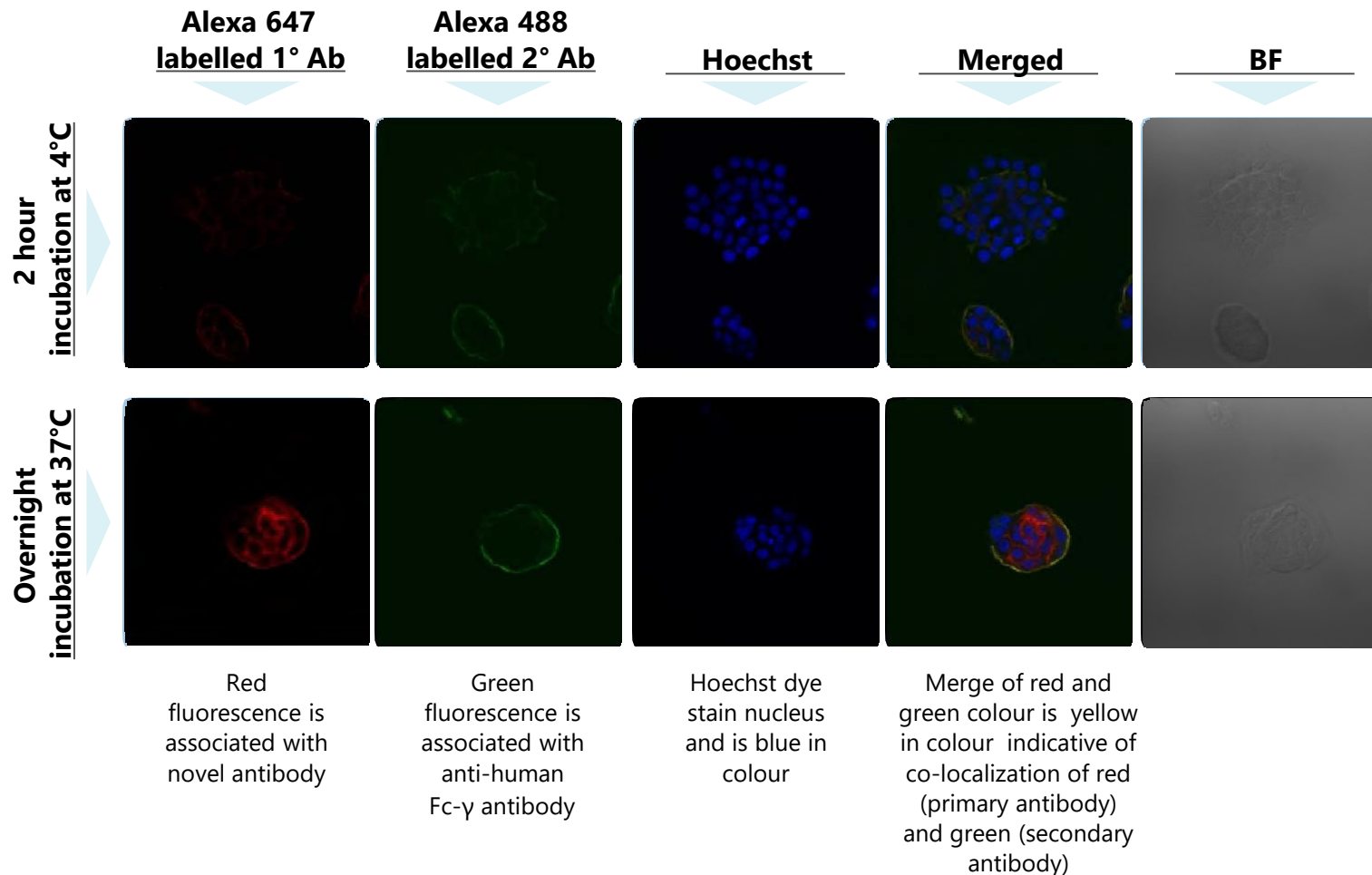
α/β complex offers a novel approach to target MUC-1



- Tumor agnostic opportunity in-licensed from Biomodifying LLC
- MUC-1 expressed extensively in majority of tumors
- Preclinical PoC of anti-tumour efficacy of Anti-MUC1 α/β targeted ADC established
- Most anti-MUC-1 mAbs under development target VNTR in the MUC-1 α
 - Circulating MUC-1 α in plasma and in peritumoral space block meaningful tumor targeting by MUC1 α -targeted therapies
 - Primary reason for the lack of efficacy
- No directly competing agents targeting α/β junction
- Potential to be a anchor for other constructs like bi-specific/multi-specific antibodies, naked mAb, etc.



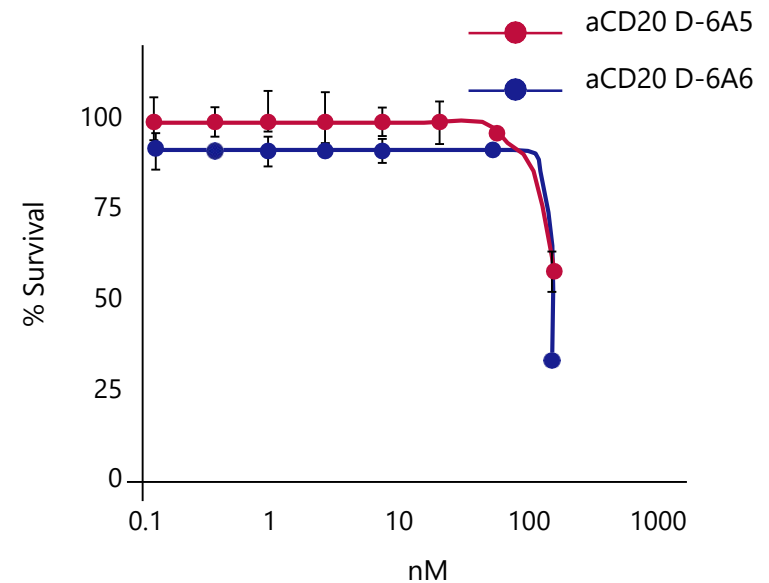
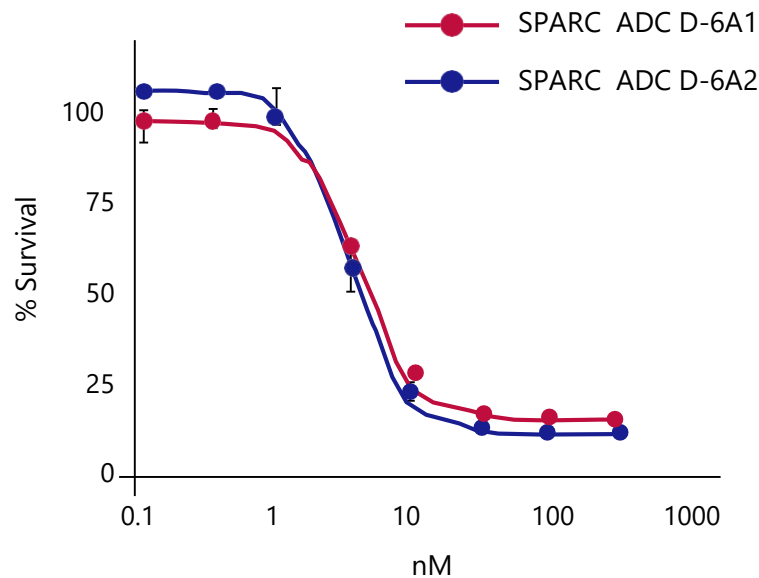
Localization of SPARC MUC-1 mAb on cell surface as well as inside the cell



SPARC MUC-1 ADC binds and exerts cytotoxicity against target-expressing cells



Cytopathic assay in a pancreatic cancer cell line



- ADC against MUC-1 antigen
- Evidence of potent cytotoxicity of SPARC ADC against MUC-1 over-expressing pancreatic carcinoma cell line
- 100-fold greater potency over a nonbinding ADC of the same payload targeted to CD20

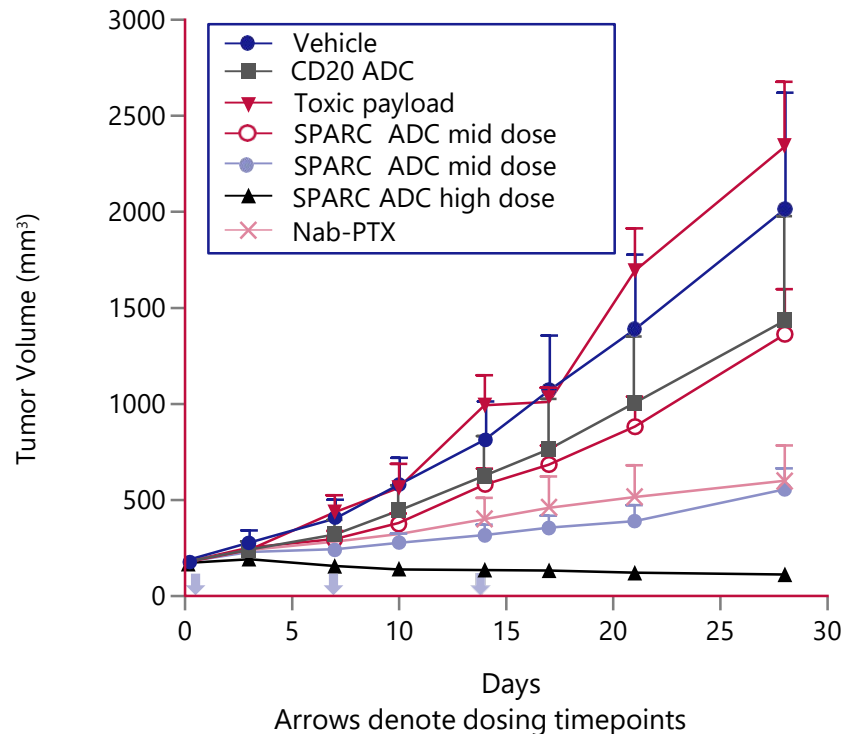
ADC = Antibody Drug Conjugate | MUC-1 = Mucin-1 | CD20 = Cluster of differentiation 20

Antitumor efficacy of SPARC MUC-1 ADC

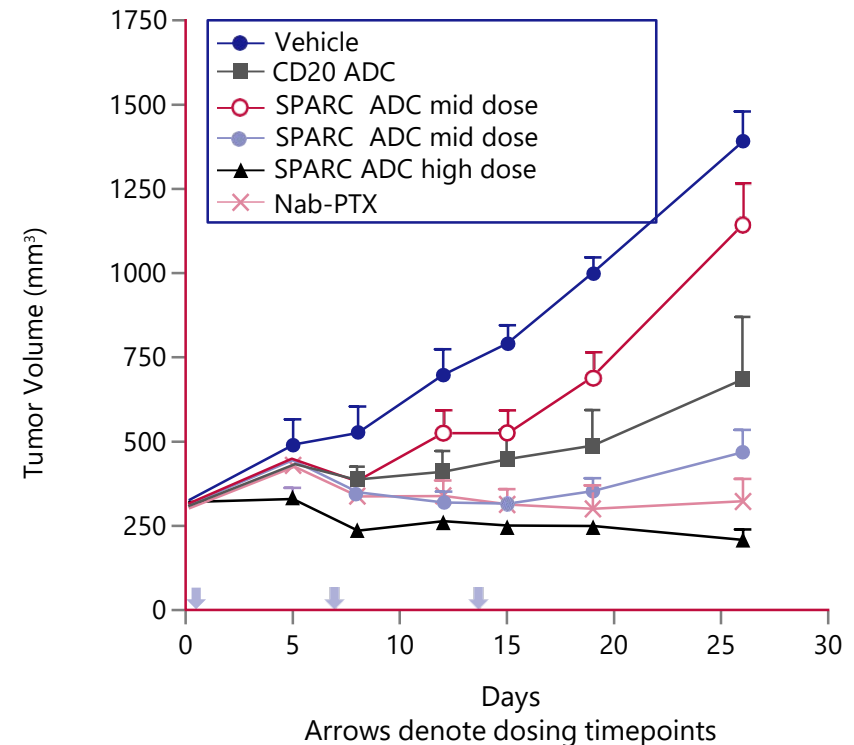


Efficacy established in multiple xenograft models

Pancreatic carcinoma xenograft



OvarianA carcinoma xenograft



- Dose-dependent growth inhibition of xenografts of pancreatic and ovarian carcinomas using SPARC ADC
- Control nonbinding anti-CD20 ADC as well as unconjugated cytotoxic agent were ineffective

SPARC MUC-1 ADC: next steps



- Advance anti MUC-1 ADC through preclinical development with IND submission in 2023
- Explore additional tumor-targeting specificities for creation of drug conjugates
- In light of the broad expression of MUC-1 in cancer, create and preclinically evaluate a series of additional immune- fusions anchored on MUC-1 targeting
 - MUC-1 targeted T-cell engager (TCE)
 - Bispecific MUC-1 targeted immune-fusion with anti-angiogenesis activity of TCEs
 - Bifunctional MUC-1 targeted immunocytokine(s) to enhance antitumor activity
 - MUC-1 targeted nanoparticles for preferential tumor-focused delivery of other targeted agents

Potential for multiple biologic product INDs in the next five years

Vodobatinib in CML (SCO-088)

A safer, last-line option for heavily pre-treated patients

Vodobatinib for CML (SCO-088)



Promising Last Line Therapy

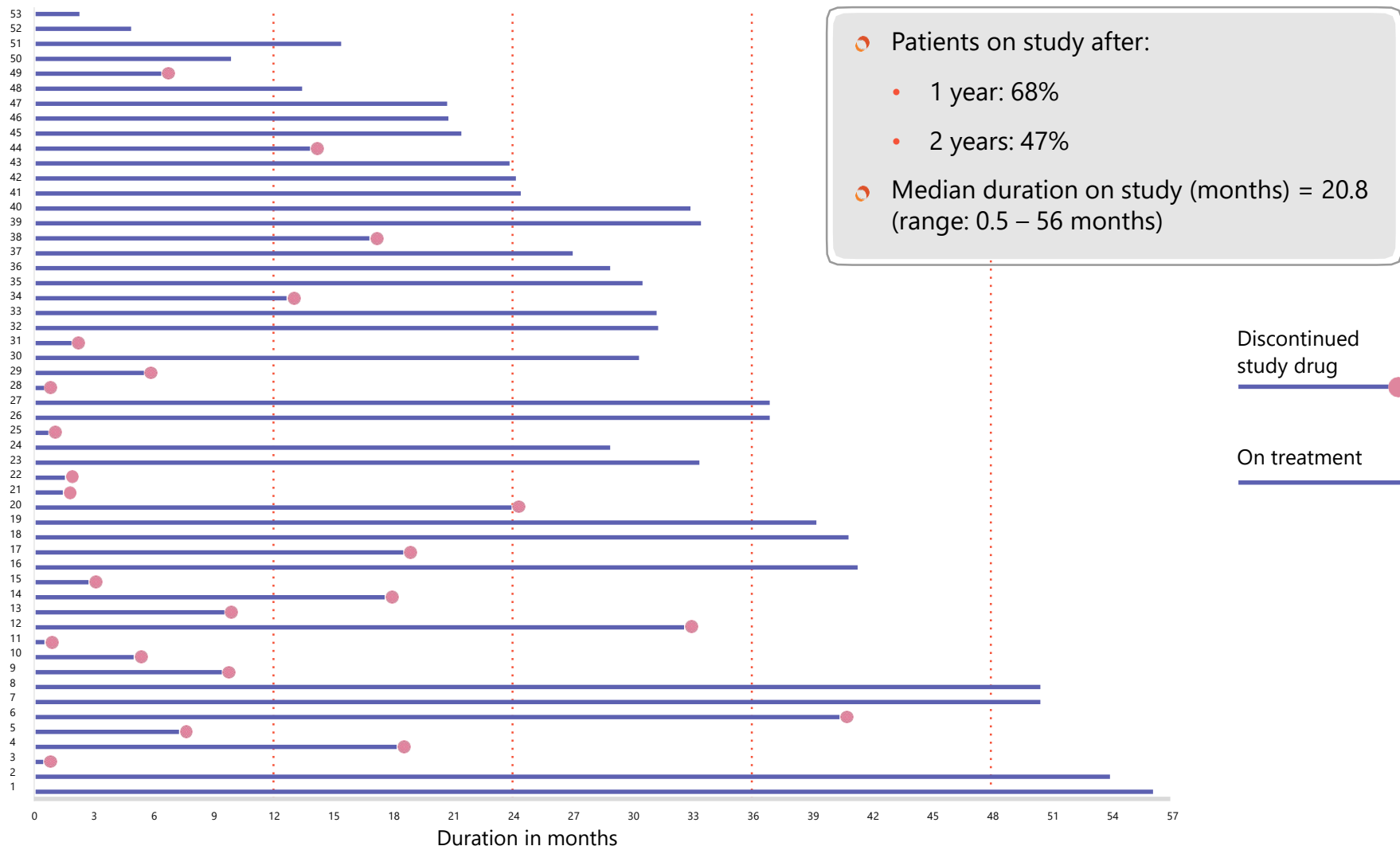


<ul style="list-style-type: none"> ○ CML is caused by a translocation of the abl gene that results in formation of Philadelphia Chromosome 	<ul style="list-style-type: none"> ○ Branded 2nd and 3rd generation TKIs retain high commercial value due to refractory nature of CML, despite genericization of 1st generation TKI 	<ul style="list-style-type: none"> ○ Targeting patients who are refractory and/or intolerant to other TKIs 	<ul style="list-style-type: none"> ○ Phase 1 completed in CML subjects
<ul style="list-style-type: none"> ○ Prior to the discovery of BCR-ABL inhibitors, CML was a fatal disease with an 8-year survival rate of ~6% 	<ul style="list-style-type: none"> ○ Large market opportunity – US drug sales of the CML TKIs over \$3Bn² 	<ul style="list-style-type: none"> ○ Well tolerated with significant coverage of the mutational field 	<ul style="list-style-type: none"> ○ Favorable safety and tolerability
<ul style="list-style-type: none"> ○ Tyrosine kinase inhibitors have changed the prognosis of CML, but patients eventually can become resistant to drugs 	<ul style="list-style-type: none"> ○ Unmet need for a potent and safe drug in patients with ≥ 3 lines of failure including failure of Ponatinib, given <ul style="list-style-type: none"> • Almost half of patients will have recurrence within 5 years of initial therapy • One-third of 2nd line patients and est. 40% of 3rd line patients are refractory or relapse within a year of initiation of that line of therapy 	<ul style="list-style-type: none"> ○ Has shown promising activity in clinical trials 	<ul style="list-style-type: none"> ○ Registration study underway. Planned US NDA filing in 2024
<ul style="list-style-type: none"> ○ Annual incidence of CML is likely to increase at a rate of 1–2 cases per 100,000 adults, est. 8,000 people in US in 2020¹ 		<ul style="list-style-type: none"> ○ Orphan Drug Designation and Accelerated Approval pathway agreed with USFDA 	

TKI = Tyrosine Kinase Inhibitor | 1. SEER database Cancer Stat Fact | 2. IQVIA 2021

Vodobatinib for CML (SCO-088)

Durable long-term responses seen across cohorts



Data cutoff 29th November 2021 | Unpublished data, not to be replicated | Number on Y-axis represents individual patients

Vodobatinib for CML (SCO-088)



Clinical Development Plan

Pivotal (Part C) study ongoing

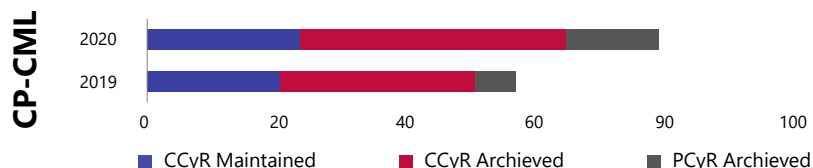
Clinical Development Plan

Part A	Single Ascending Dose study (SAD) in volunteers	✓
Part B	Multiple Ascending Dose study (MAD) in patients	✓
Part C	Pivotal efficacy study in refractory and/or intolerant patients to 3 prior TKIs	

- Orphan Drug Designation approved by USFDA and EMA
 - Market exclusivity in addition to IP coverage
 - User fee waiver
- EOP1 discussion completed; agreement with USFDA reached on accelerated approval pathway based on Part C (pivotal study)

Efficacy

Cytogenetic Response (% patients with MaCyR)

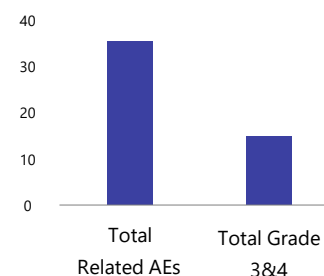


- Major Cytogenetic response in 67% of the enrolled subjects
- Major Cytogenetic response in 54% of the enrolled subjects that meet pivotal study criteria

Safety and Tolerability

- Generally well tolerated with slight excess of GI and hematological AEs

All Treatment Emergent AEs (Cases)



Planned US NDA filing in 2024

EOP1 = End of Phase 1 | MaCyR = Major Cytogenetic Response | CP = Chronic Phase | CCyR = Complete Cytogenetic Response | PCyR= Partial Cytogenetic Response | AE = Adverse Event | GI = Gastro Intestinal
| SAD = Single Ascending Dose | MAD= Multiple Ascending Dose.

SCO-120 for HR+/HER2- MBC

Potent oral SERD with preferential brain penetration

Oral SERD for Breast Cancer (SCO-120)



- Breast cancer is the second most common cancer diagnosed in women in the United States¹
- Annual incidence of ~2 million patients across the world¹
- ~70% of the breast cancer is HR+/HER2-¹

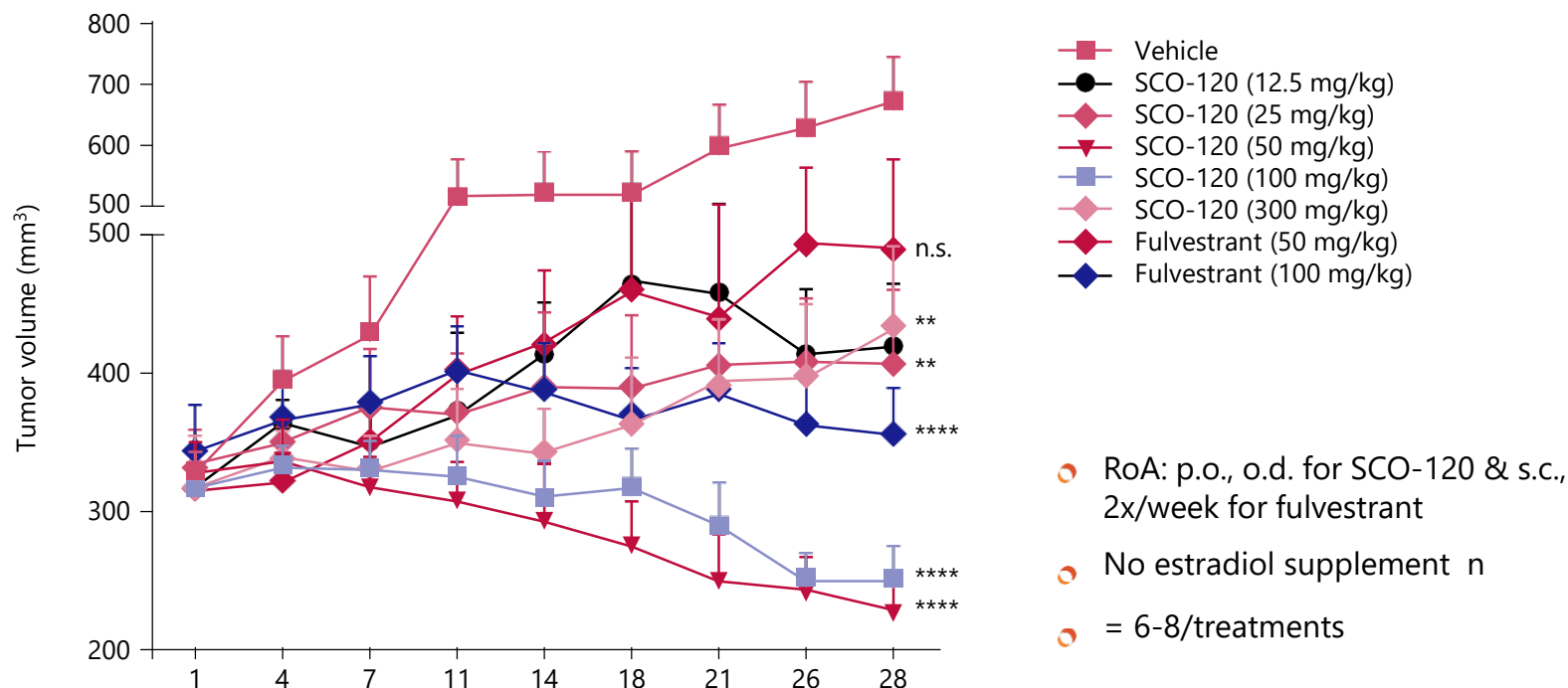
- Hormonal therapy is SoC for ~70% of HR+/HER2- metastatic breast cancer patients¹. ER α mutations develop in 20–50% of patients with metastatic disease
- Treated mostly with SERMs, 20–50% patients experience mutations or become resistant
- SERD can break down receptors and prevent cells from dividing. IM Fulvestrant is the only approved SERD but it is poorly active against mutations at therapeutic dose

- SCO-120 is a novel orally-active SERD for the treatment of HR+/HER2- breast cancer
- Active in vitro (nM to sub nM potency) and in vivo in xenograft models against WT ER α and its mutants Y537S and D538G
- In vitro and in vivo studies have shown potential for combination with CDK4/6 inhibitors (palbociclib) in both the WT ER α and the mutation setting
- Favorable Tox profile; No adverse effects seen in battery of in vivo safety pharmacology studies of central nervous system, cardiovascular system, and respiratory system

- US IND filed in Jan 2020
- SAD and MAD in healthy volunteers ongoing
- 50 – 1200 mg cohorts completed. Generally safe and well tolerated, no significant AEs

1. CancerMPact® Treatment Architecture U.S., Breast Cancer | HR = Hormone Receptor | HER2 = Human Epidermal Growth Factor Receptor 2 | ER α = Estrogen Receptor α | SOC = Standard of Care | IM = Intramuscular | SERD = Selective Estrogen Receptor Degradable | AE = Adverse Event | SERM = Selective Estrogen Receptor Modulator | MAD = Multiple Ascending Dose |

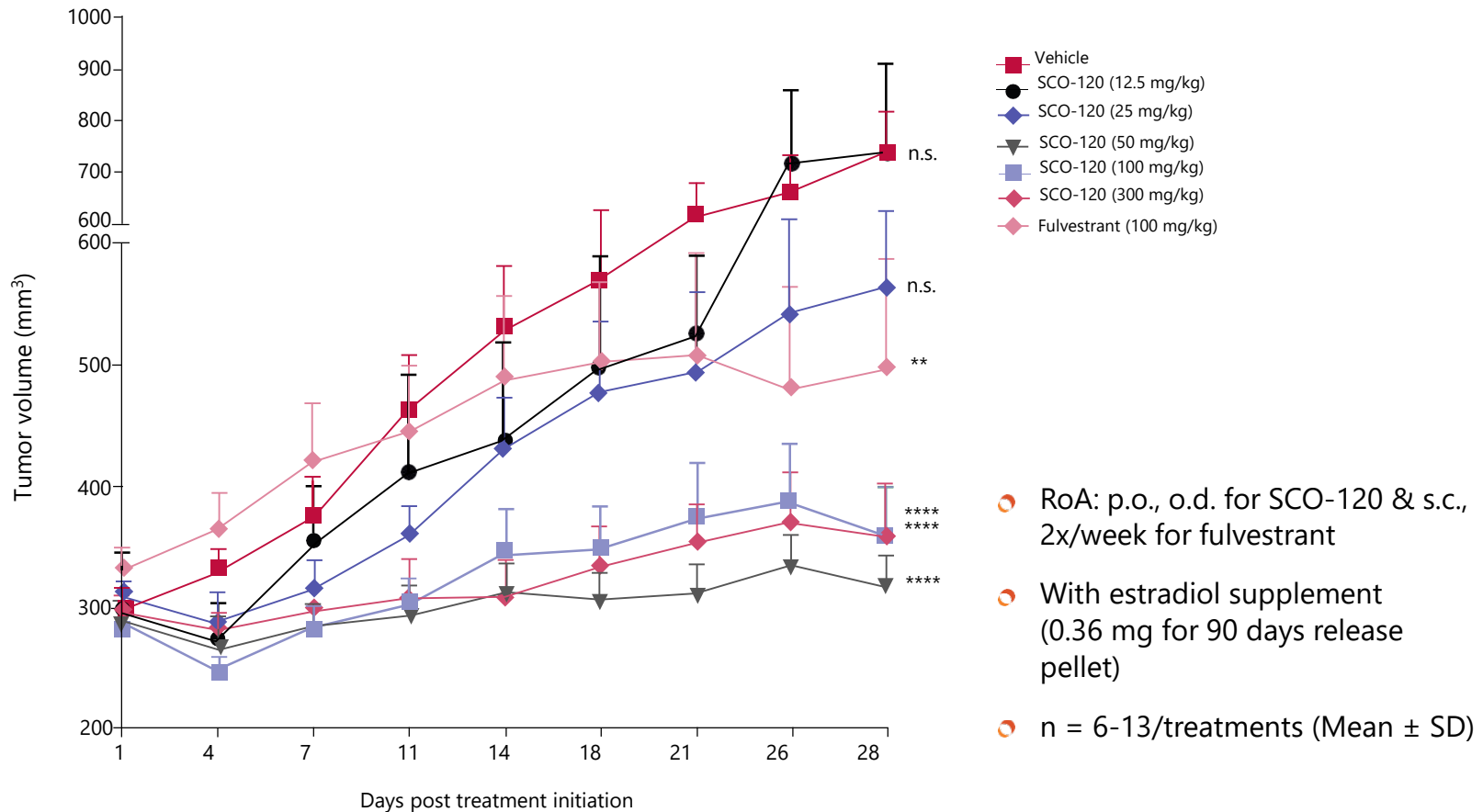
Growth inhibition of subcutaneous MCF-7 xenografts expressing ER α Y537S mutant



- SCO-120 shows inhibition of growth in tumors expressing ER α Y537S mutant at ≥ 100 mg/kg dose

Data presented as mean \pm SD | Statistical analysis was carried out using two-way ANOVA followed by Bonferroni's post hoc test for multiple comparisons | **p < 0.01 | ****p < 0.0001 as compared to vehicle treated group | n.s.-non significant.

Growth inhibition of subcutaneous MCF-7 xenografts expressing ER α D538G mutant

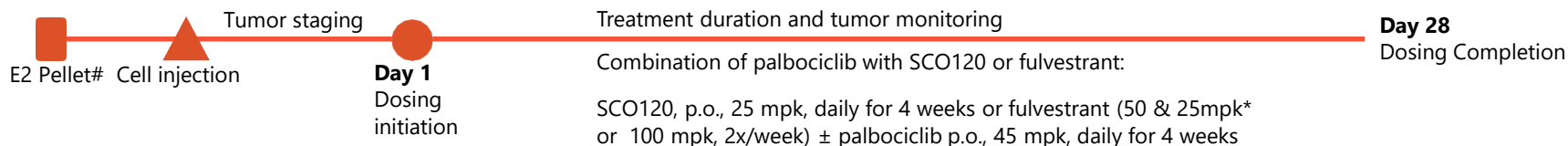


SCO-120 shows inhibition of growth in tumors expressing ER α D538G mutant at a dose of ≥ 100 mg/kg

Data presented as mean \pm SD | Statistical analysis was carried out using two-way ANOVA followed by Tukey post hoc test for multiple comparisons | **p < 0.01 | ****p < 0.0001 as compared to vehicle treated group n.s.-non significant.

In vivo efficacy of SCO-120 in combination with palbociclib

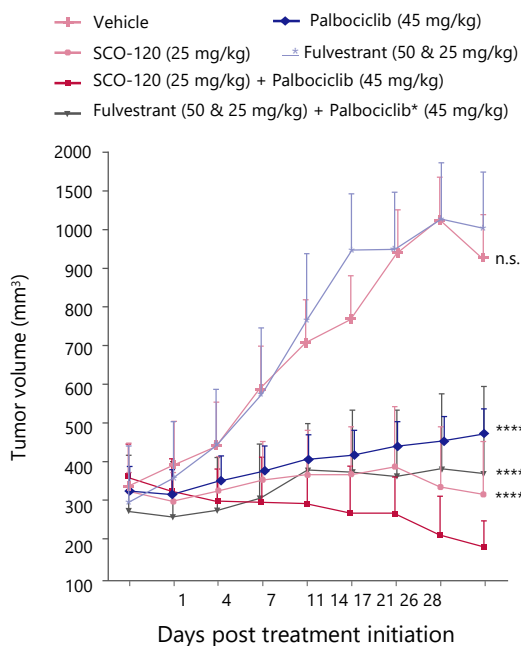
Promising activity against resistant mutants alone and in combination with palbociclib



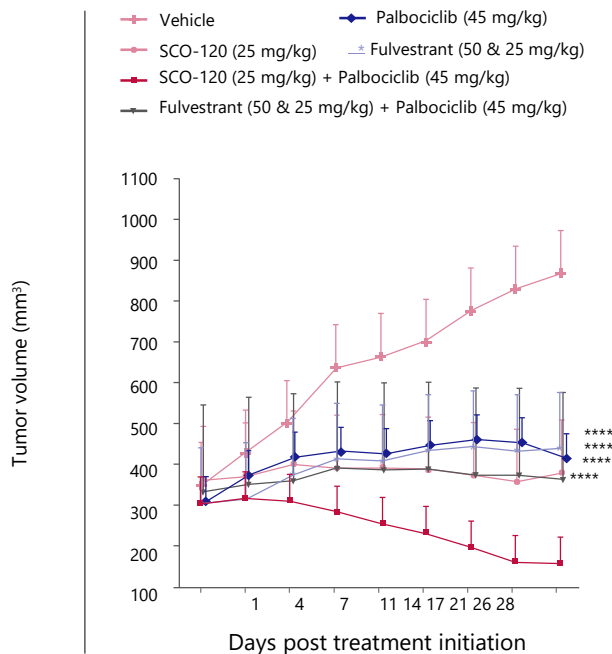
Combination of palbociclib with SCO120 or fulvestrant:

SCO120, p.o., 25 mpk, daily for 4 weeks or fulvestrant (50 & 25mpk* or 100 mpk, 2x/week) ± palbociclib p.o., 45 mpk, daily for 4 weeks

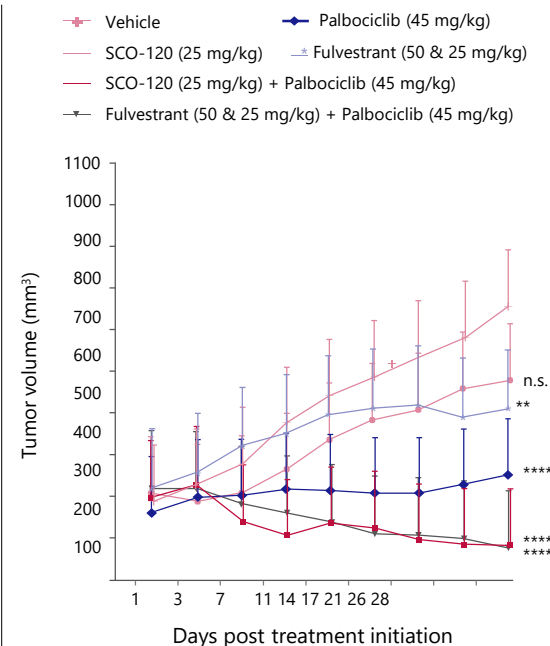
MCF7-WT



MCF7-Y537S



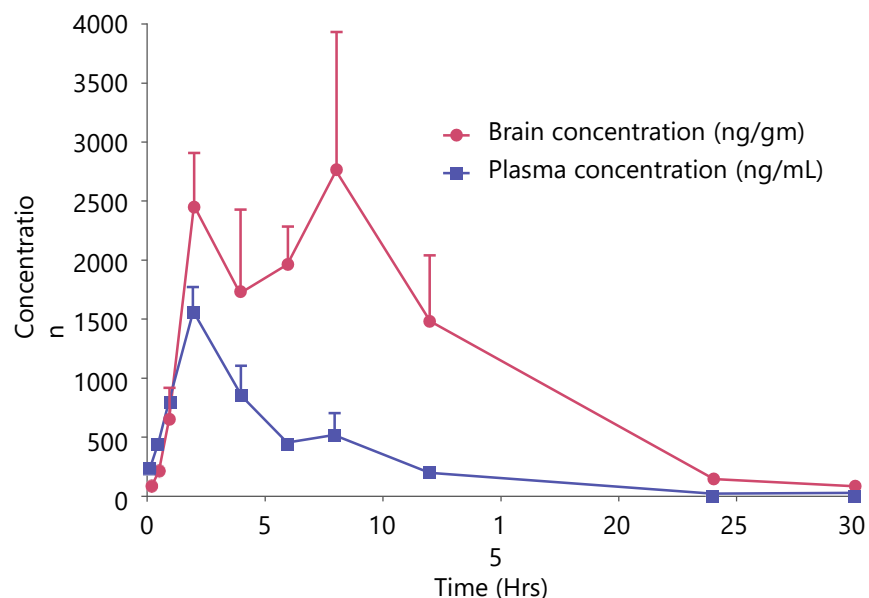
MCF7-D538G



*Fulvestrant group received 50 mg/kg as loading dose thrice-weekly for first week, followed by 25 mg/kg twice weekly for remaining 3 weeks

*p < 0.01 | ****p < 0.0001 as compared to vehicle treated group | n.s.-non significant

Plasma vs brain PK profile in mice



Brain PK Parameters	
Tmax (h)	8.0
Cmax (ng/g)	2761.8
AUC0-t (h*ng/gm)	33336.1
AUC0-inf (h*ng/gm)	33918.8
T1/2 (h)	4.35
DN_AUC0-inf	339.2

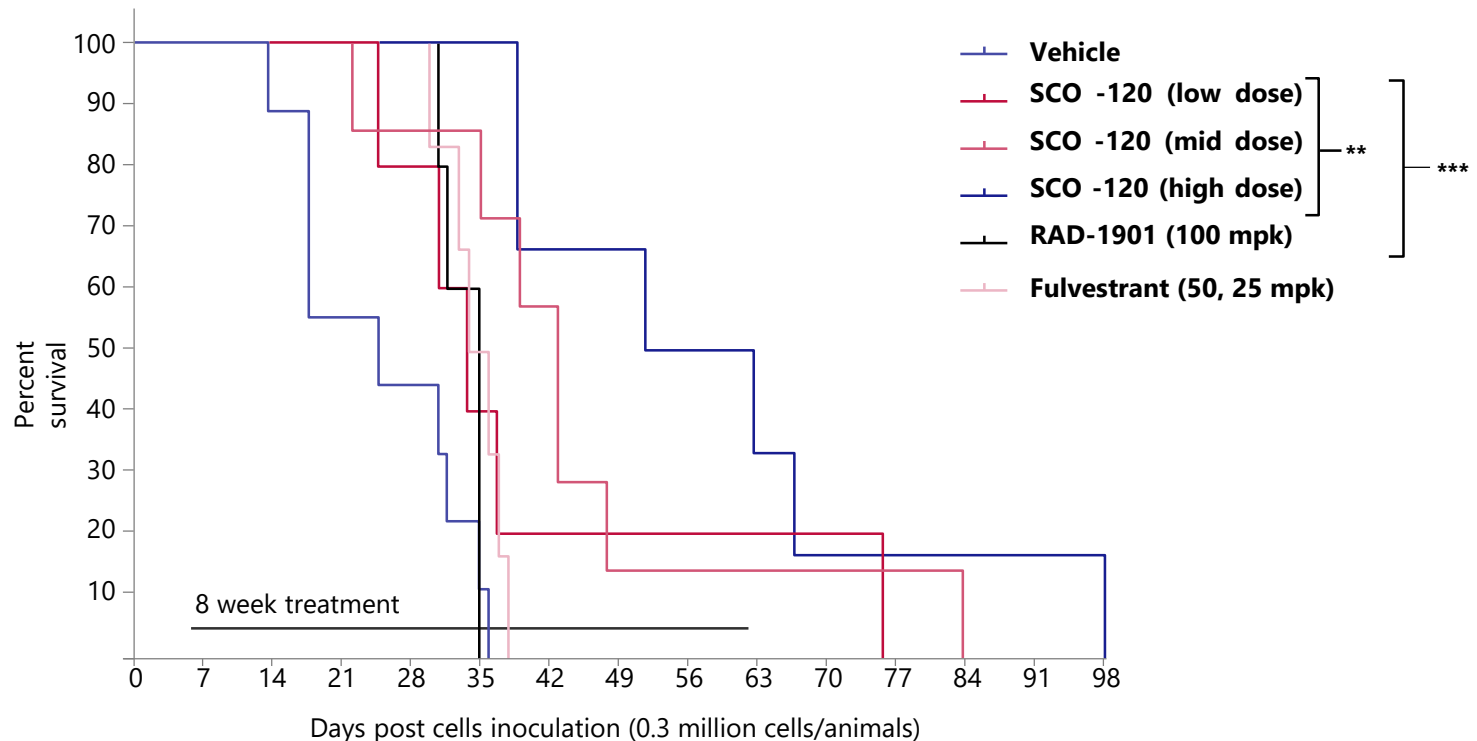
Time Points (h)	0.25	0.5	1	2	4	6	8	12
Ratio Brain/ Plasma	0.41	0.49	0.82	1.58	2.03	4.49	5.49	7.935

- SCO-120 effectively crosses blood-brain barrier with a long half-life in the brain

SCO-120 advantage in brain metastases



Prolonged survival in preclinical brain-metastasis model expressing wild type ER α

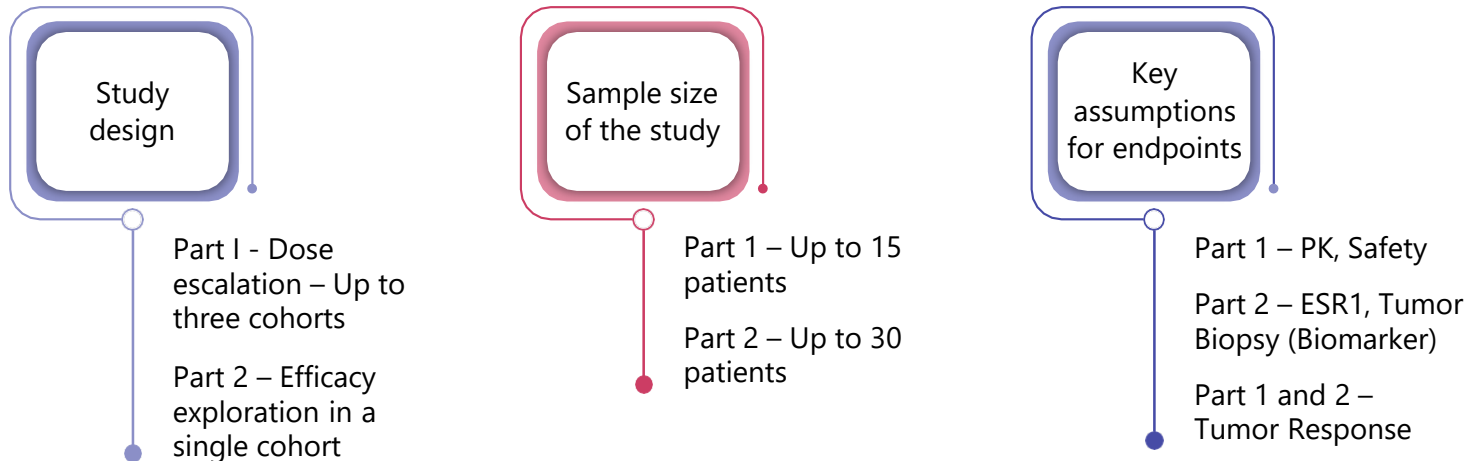


- Effectively crosses blood-brain barrier with higher accumulation in brain and tumor compared to plasma
- SCO-120 treated mice showed significant increased survival compared to RAD-1901 and fulvestrant
- Potential to be an active treatment for HR+/HER2- breast cancer patients with brain metastases

Unpublished data; not to be replicated | ER α = Estrogen Receptor α

SCO-120 enters patient trials in 2022

Clinical development plan and upcoming milestones



PK = Pharmacokinetic | ESR1 = Estrogen receptor 1




SPARC Delivery Systems

Legacy portfolio leveraging 505-B(2) pathway
provides opportunity to deliver non-dilutive
funding in the short term

Novel drug delivery systems based 505(b)(2) opportunities



Cost effective programs offering non-dilutive cash-flows

 Technology Licensing	Liposomal Drug Delivery Technology	Oral Delivery Technologies	Liposomal Drug Delivery Technology – Sun Pharma (USA) Oral Delivery Technologies – Sun Pharma (India)
 Approved Products	Xelpros BAK free	Elepsia XR	Xelpros – Sun Pharma (USA, India), CMS (China) Elepsia – CMS (China)
 Nearing submission	Phenobarbital	Ophthal assets (PDP-716 & SDN-037)	USA – Ophthal assets to Visiox Pharma China – CMS India – Sun Pharma

Licensed to Tripoint Therapeutics for commercialization in the US

- Commercialization initiated in 2021
- Tripoint completed field launch meet and training of sales team
 - 40 reps promoting ELEPSIA™ XR
 - ELEPSIA™ XR active on TX Medicaid
 - ELEPSIA™ XR contracted with ESI



MANAGING SEIZURES CAN BE COMPLEX. REDUCING PILL BURDEN IS SIMPLE.¹

Once-daily ELEPSIA™ XR: 1000 mg and 1500 mg tablets

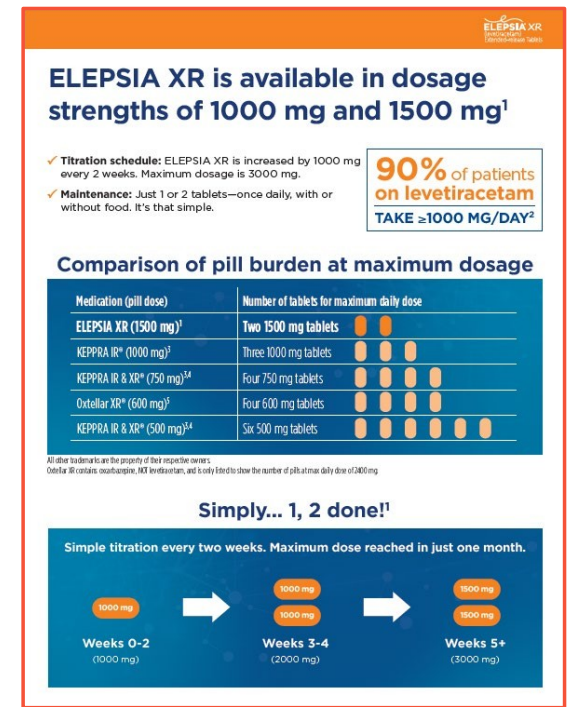
ELEPSIA XR
(levetiracetam)
Extended-release Tablets

INDICATIONS AND USAGE
ELEPSIA XR is indicated as adjunctive therapy for the treatment of partial-onset seizures in patients 12 years of age and older.

IMPORTANT SAFETY INFORMATION
RECOMMENDED DOSING
ELEPSIA XR is administered orally once daily. Initiate treatment with a dose of 1000 mg once daily. The once daily dosage may be adjusted in increments of 1000 mg every 2 weeks, to a maximum recommended daily dose of 3000 mg/day. ELEPSIA XR should be taken whole; do not split or cut tablets.

CONTRAINDICATIONS
ELEPSIA XR (levetiracetam Extended-Release Tablets) is contraindicated in patients with a hypersensitivity to levetiracetam. Reactions have included anaphylaxis and angioedema.

Please see additional Important Safety Information inside.
For more information, please see the full Prescribing Information and Medication Guide for ELEPSIA XR.



ELEPSIA XR is available in dosage strengths of 1000 mg and 1500 mg¹

✓ **Titration schedule:** ELEPSIA XR is increased by 1000 mg every 2 weeks. Maximum dosage is 3000 mg.
✓ **Maintenance:** Just 1 or 2 tablets—once daily, with or without food. It's that simple.

90% of patients on levetiracetam TAKE ≥1000 MG/DAY²

Comparison of pill burden at maximum dosage

Medication (pill dose)	Number of tablets for maximum daily dose
ELEPSIA XR (1500 mg) ¹	Two 1500 mg tablets
KEPPRA IR® (1000 mg) ³	Three 1000 mg tablets
KEPPRA IR & XR® (750 mg) ^{3A}	Four 750 mg tablets
Orvettar XR® (600 mg) ³	Four 600 mg tablets
KEPPRA IR & XR® (500 mg) ^{3A}	Six 500 mg tablets

All other trademarks are the property of their respective owners.
Orvettar IR contains oxcarbazepine, RCT levetiracetam, and is only listed to show the number of pills at true daily dose of 2400mg.

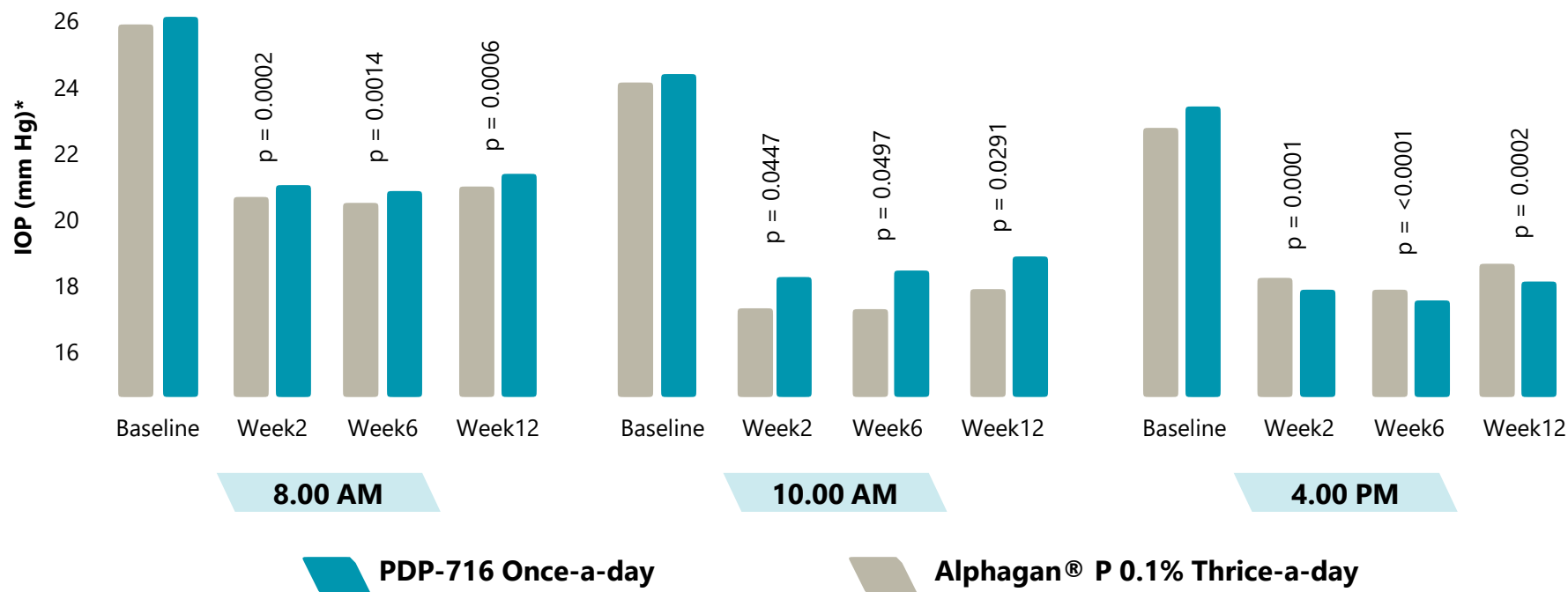
Simply... 1, 2 done!¹

Simple titration every two weeks. Maximum dose reached in just one month.

Weeks	Dose	Tablets
Weeks 0-2	1000 mg (1000 mg)	1 tablet
Weeks 3-4	2000 mg (2000 mg)	2 tablets
Weeks 5+	3000 mg (3000 mg)	2 tablets

Phase 3 study successfully met pre-specified endpoints

- Equivalent reduction in intraocular pressure was demonstrated across all required time-points
- Treatment-emergent adverse events were similar; 38.8% in the PDP-716 group vs. 33.2% with Alphagan® P 0.1% group
- NDA filing planned for 2022



Phase 3 trial met primary and secondary objectives

- Statistically significant proportion of patients treated with SDN-037 achieved an ACC grade of 0 versus vehicle with p-values <0.0001
- Generally well tolerated with adverse events consistent with the known safety profile of difluprednate
- NDA filing planned for 2022

Primary efficacy analysis

ACC Grade	SDN-037 N= 123 (%)	Vehicle N= 83 (%)
Responders		
0 (Did not receive rescue therapy)	84 (68.3)	27 (32.5)
Non-responders (Received rescue therapy)		
1	38 (30.9)	42 (50.6)
2	1 (0.8)	13 (15.7)
3	0 (0.0)	1 (1.2)
p-value	<0.0001	

Phenobarbital

Preservative-free injection for neonatal seizure

- Current standard of care for treatment of neonatal seizure
- Phenobarbital is an “unapproved drug” in USA; Approved before 1938 which did not require proof for safety and / or efficacy
- Existing marketed product is not approved by US FDA and contains benzyl alcohol as a preservative.
- Benzyl alcohol has been associated with “Gasping Syndrome” in neonates and low-birth weight infants
- NDA filed

The text "Orphan Drug Designation" is centered between two horizontal orange lines. The text is in a large, black, serif font, with "Orphan Drug" on the top line and "Designation" on the bottom line.

Orphan Drug
Designation

SPARC Human Capital

Experienced management team backed by
accomplished founder and advisory board

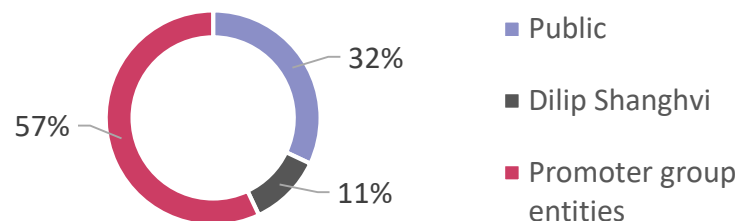
Established and supported by marquee industry leader



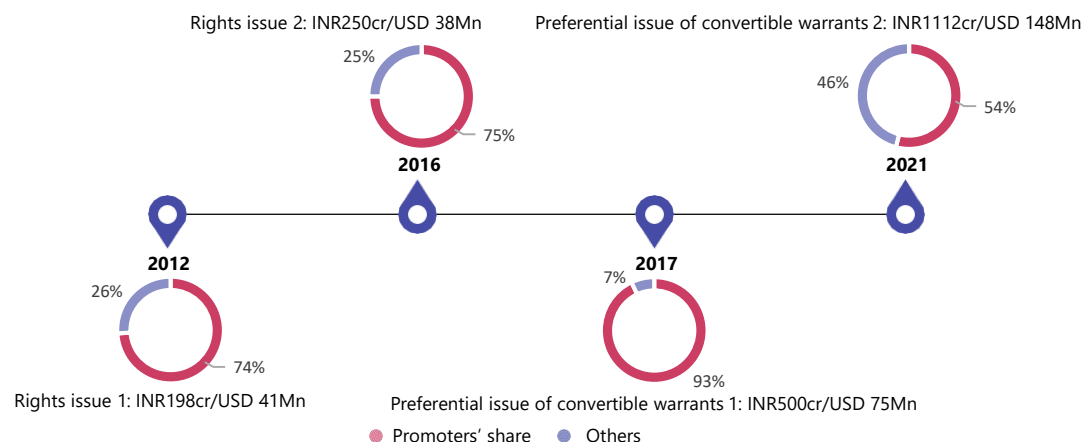
Dilip Shanghvi
Chairman

- Founded Sun Pharma in 1983.
(Current market cap of USD 24Bn+*)
- Has 35+ years of industry experience
- Awards and recognitions:
Padma Shri (Fourth highest civilian award by Govt. of India) in 2016, Forbes Entrepreneur of the year – 2014, Economic Times Business Leader of the Year (2014), CNN IBN's Indian of the Year (Business) (2011) and Ernst and Young's World Entrepreneur of the Year (2011).

Shareholding (as on 30th Sep. 2021)



Providing continuous support and investments



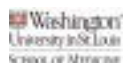
- Completed preferential issue for INR 1112 Cr. (USD 148Mn) in July 2021
- Well-capitalized for prosecuting the current clinical portfolio

*As of 13th December, 2021 | Percentage and figures rounded off to nearest number

Scientific advisory board consisting of globally recognized experts



Phil Needleman, PhD
Washington University
St. Louis



Alan Ashworth, PhD, FRS
UCSF
ICR London



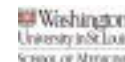
Robert Spiegel MD¹
Weill Cornell Medical
College



Richard Ulevitch, PhD
Scripps Research



John DiPersio, MD, PhD
Washington University
St. Louis



Mark Simon, MBA²
Torreya Partners, Citigroup,
Robertson Stephens, Kidder Peabody



Jorge Cortes, MD
Medical College of
Georgia MD Anderson



Adrian Ivinson, PhD
DRI UK, Nature, Harvard
Medical School



Mathew LaVoie, PhD
University of Florida



1. Member of the Board of Directors | 2. Board Advisor

Highly experienced management team with global experience



7 27

Anil Raghavan

Chief Executive Officer

Responsible for strategic prioritization and portfolio decisions

Past experience:



7 36

Nitin Damle

Chief Innovation Officer

Leads the development of Biologics

Past experience:



7 25

Siu-Long Yao

Head, Clinical Development & Operations

Oversees design & execution of clinical research globally

Past experience:



4 31

Chetan Rajpara

Chief Financial Officer

Responsible for finance, accounts, taxation and legal & secretarial functions

Past experience:



22 32

Nitin Dharmadhikari

Head, Operational Excellence & COEs

Responsible for New Initiatives, management of COEs and QA

Past experience:



14 31

Trinadha Rao Chitturi

Head, Drug Discovery

Oversees Medicinal Chemistry, In-Vitro Biology, Bio-informatics & Process Development

Past experience:



● Years with SPARC ● Years of experience

Highly experienced management team with global experience



3 25

Vikram Ramanathan

Head, Translational Development

Responsible for Preclinical Pharmacology, Drug Metabolism & PK and Bioanalysis, and Regulatory Toxicology

Past experience:



13 22

Shravanti Bhowmik

Head, Program Management

Oversees all aspects of the development / implementation of projects and programs

Past experience:



14 22

Yashoraj Zala

Head, Drug Delivery Systems

Responsible for drug formulation and analytical development

Past experience:



1 20

Rajesh Ranganathan

Head, Partnerships and Portfolio Strategy

Oversees external partnerships and portfolio management

Past experience:



1 21

Shanta Gupta

Chief Human Resource Officer

Responsible for the organization's human capital management

Past experience:



● Years with SPARC ● Years of experience

Company highlights



Successful Track Record of Development and Commercialization along with a Robust Pipeline



2

USFDA approved drugs (Xelpros™, Elepsia™)



6

Indications targeted through 4 NCEs under clinical development



10+

Preclinical programs in R&D pipeline covering 3 therapeutic areas

Targeting High Value Opportunities



USD 20Bn+

Combined peak sales potential for NCEs currently under clinical development



6

6 Licensing partners¹

Through an Innovation-focused R&D Platform with an Efficient Cost Structure



350+

Scientists across 4 research centers. Growing presence in the US (Princeton, NJ)



250+

Years of experience of management



8

Ongoing collaborations with universities / companies

1. Licensing partners include Bioprojet, CMS, Sun Pharmaceutical Industries Ltd. (Sun Pharma), Tripoint Therapeutics, Biomodifying, and Visiox.

Thank You

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