Update on Clinical Programs and R&D Pipeline

13th October 2022

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Built a robust R&D organization

Innovating from India for the world, SPARC offers operating model validation

Successful track record of development and commercialization

2
USFDA approved drugs (Xelpros™, Elepsia™)

2
NDAs submitted to USFDA in FY23

1
NDA targeted for submission in H2 FY23

Clinical pipeline targeting high-value opportunities

3
NCEs targeting 6 clinical indications in development

USD 20Bn+
Combined peak sales potential for NCEs currently under clinical development

Through an innovation-focused R&D platform

10+
Preclinical programs in R&D pipeline covering 3 therapy areas

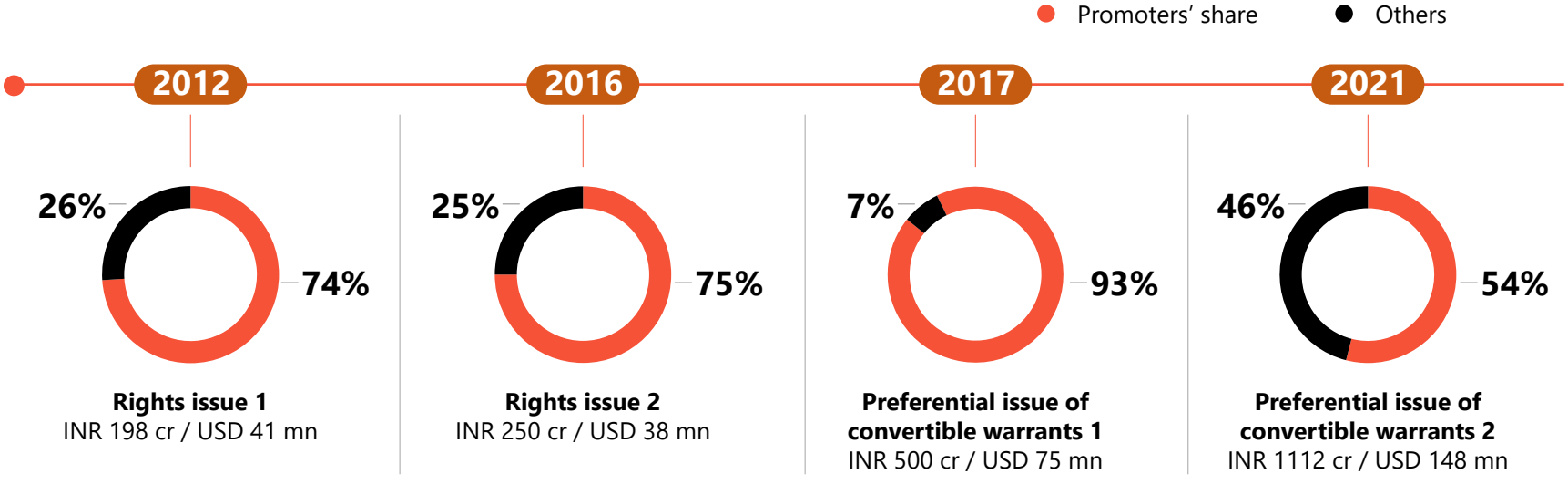
2
IND filings targeted by FY24

11
Partnerships with academic centers and commercial entities[1]

[1] Partners include JHU, UCSF, Wash U at St Louis, University of Michigan, Scripps Research, Bioprojet, CMS, Sun Pharma, Tripoint Therapeutics, Biomodifying, and Visiox | USFDA = US Food & Drug Administration | NDA = New Drug Application | NCE = New Chemical Entity | IND = Investigational New Drug

Attractive portfolio built using a cost-efficient structure

Over 50% funded through non-dilutive revenue generated



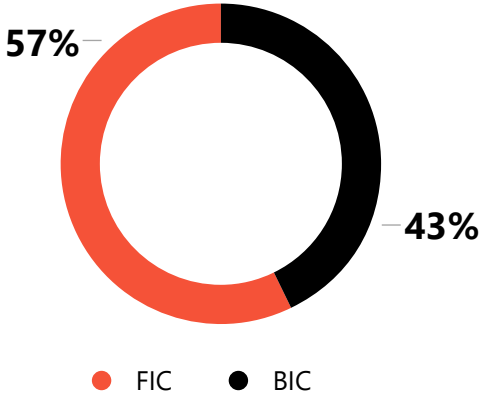
USD 523 mn
Total investment till FY22

USD 277 mn
Revenues re-invested till FY22

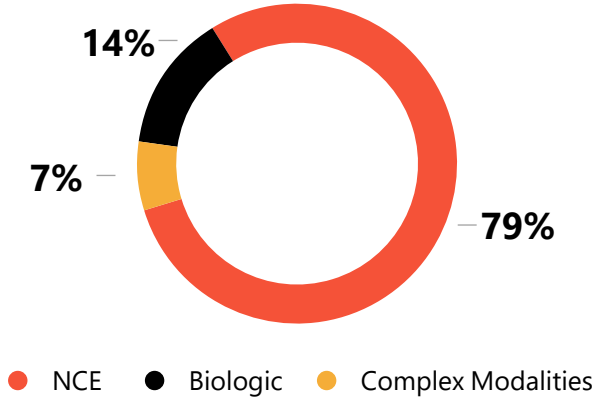
Our strategy to address unmet clinical need has transformed

With a focus on novel biology as opposed to fast follower approach

Asset Distribution by Class



Asset Distribution by type



- Increasing proportion of programs focusing on novel biology (potential first-in-class)
- Investments in new modalities/complex platforms is now translating to tangible programs
- Continued development of best-in-class assets for validated targets
- Collaboration with external innovators as a key tenet of strategy to access early science

FIC – First in Class | BIC – Best in Class

First-in-class innovation in three therapy areas

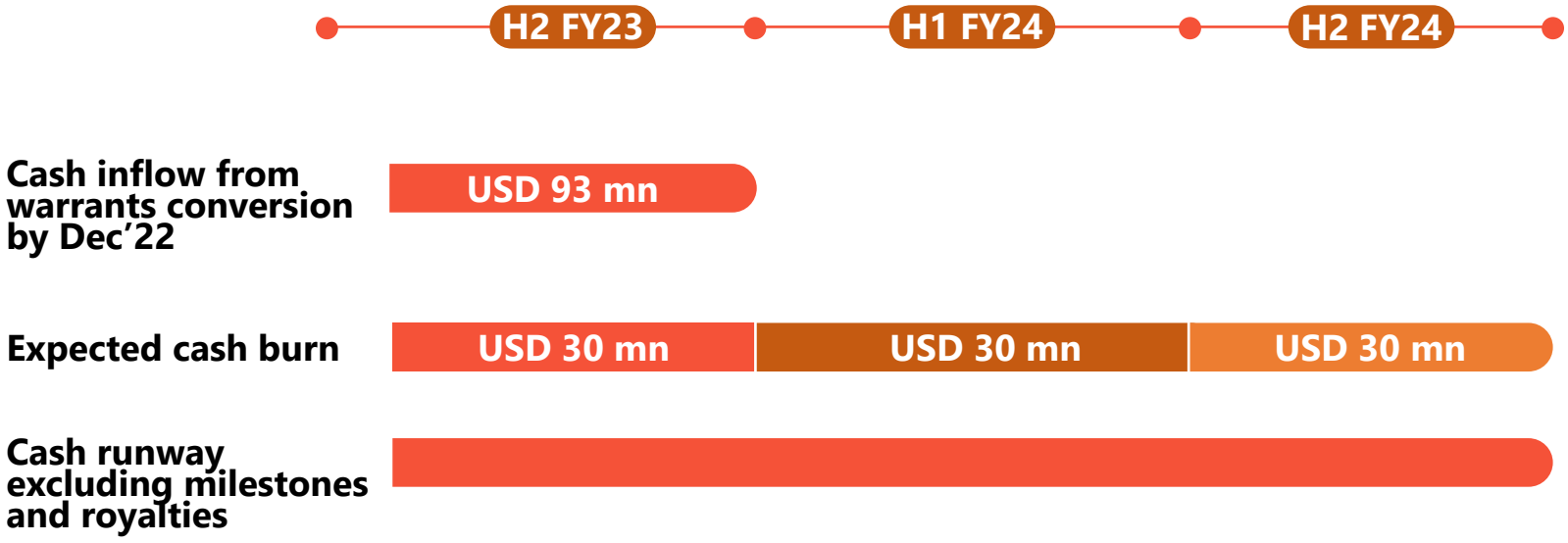
With 70% of the preclinical pipeline targeting novel hypotheses

	Neurology	Oncology	Immunology
Areas of interest	Restoring cellular function in the CNS to modify disease-course by: • Modulating oxidative stress response • Improving autophagic flux • Preventing misfolded protein aggregation	Modality-agnostic, tumor-targeted strategies to address indications that have limited treatment options	Pursuing novel targets in immune cells and inflamed tissues to modulate inflammatory conditions
Lead FIC Program	**SCC-138** Internally developed NCE - vodobatinib Selective c-ABL inhibitor with good brain penetration and superior safety for Parkinson's Disease (PD) Currently in a Phase 2 clinical study	**SBO-154** In-licensed anti-MUC-1 antibodies from Biomodifying LLC Developed antibody-drug conjugate asset for solid tumors Currently in IND-enabling preclinical development	**SCD-153** Collaboration with Johns Hopkins University NCE targeting a novel pathway to address alopecia areata Currently in IND-enabling preclinical development

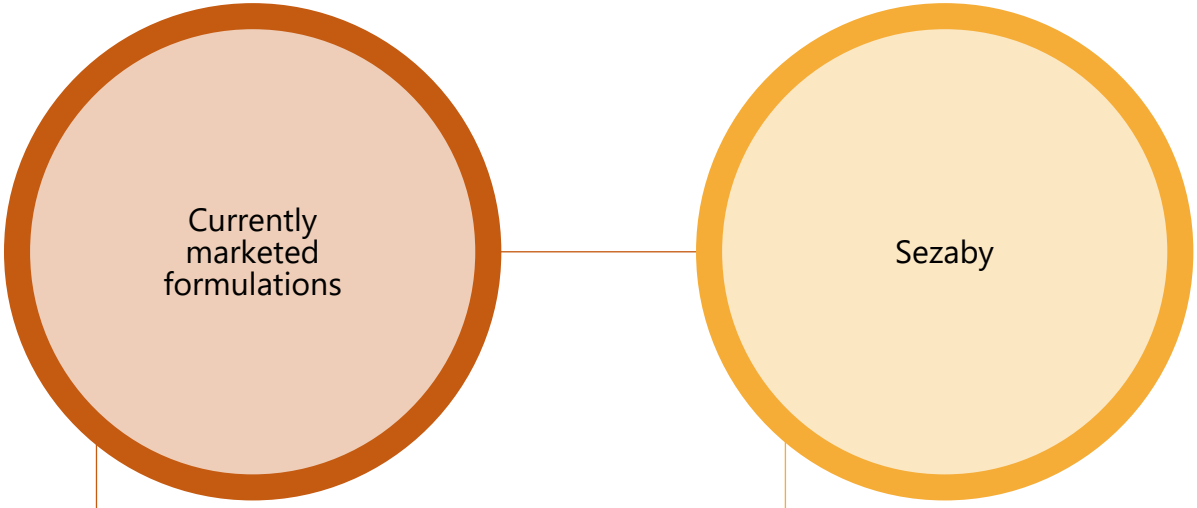
CNS = Central Nervous System | NCE = New Chemical Entity | IND = Investigational New Drug

Expected cash inflow from warrants conversion

Cash runway till FY24, with potential extension from milestones and royalties



Sezaby*: benzyl alcohol-free phenobarbital injection for neonatal seizures



- Classified under DESI products by USFDA; approved only for safety between 1938 and 1962
- Contain benzyl alcohol as a preservative which has been associated with "Gasping Syndrome" in neonates and low-birth weight infants

- Granted Orphan Drug Designation for treatment of neonatal seizures
  - Upon approval will be eligible of Orphan Drug Exclusivity
- NDA submitted in 2022
  - USFDA granted priority review of NDA
  - PDUFA goal date of Nov'22
- SPARC under advance discussion with potential partners for licensing commercialization rights of Sezaby

Sezaby as a trade name is conditionally accepted by the USFDA

DESI = Drug Efficacy Study Implementation | USFDA = US Food & Drug Administration | NDA = New Drug Application | PDUFA = Prescription Drug User Fee Act

Several high-yield assets graduating to next set of data events in the short term

Sharp execution focus to deliver key updates in the next 18-24 months

Licensing and commercialization of near-term assets

- Driving uptake of Xelpros and ELEPSIA XR
- Collaborate with Visiox Pharma for commercialization of PDP-716 and SDN-037
- Licensing of Sezaby to potential partner

Continued progress of clinical NCEs

- Vodobatinib CML read out in FY24
- Vodobatinib PD PROSEEK Phase 2 readout in FY24
- SCO-120 clinical PoC in FY24

Early PoC for new platforms

- IND filing for SCD-153 in FY23 and for SBO-154 in FY24
- In-licensing of preclinical assets

NCE = New Chemical Entity | PoC = Proof-of-Concept | CML = Chronic Myeloid Leukemia | IND = Investigational New Drug

Pipeline overview & key upcoming milestones

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 PROSEEK study in FY24	
		Lewy Body Dementia[1]						PoC data in FY24	
		Alzheimer's Disease							
Vodobatinib (SCO-088)	BCR-ABL Inhibitor	Refractory CML						Pivotal data in FY24	
SC0-120	Selective ERα Receptor Degrader	Metastatic Breast Cancer						Phase 1 data in FY24	
SB0-154	Anti-MUC-1 ADC	Multiple Tumors						IND filing targeted in FY24	
Vibozilimod (SCD-044)	Selective S1PR1 agonist	Psoriasis							SUN PHARMA
		Atopic Dermatitis							SUN PHARMA
SCD-153	Undisclosed	Alopecia Areata						IND filing targeted in FY23	

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 | ADC - Antibody-Drug Conjugate PoC = Proof of Concept | CML = Chronic Myeloid Leukemia | S1PR1 = Sphingosine-1-Phosphate Receptor 1 | ERα = estrogen receptor α | IND = Investigational New Drug

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Clinical Programs

Siu-Long Yao

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Vodobatinib in CML (SCO-088)

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

Vodobatinib for CML (SCO-088)

Potent and selective inhibitor of BCR-ABL1

Clinical study design

Healthy volunteer study

Single Ascending Dose (SAD) and Food Effect studies in healthy volunteers (N = 40)

Multiple Ascending Dose (MAD) study in patients (N = 53)

Patient studies

Enrollment ongoing

Pivotal efficacy study in refractory patients

- CP-CML N = 59
- AP-CML N = 43
- BP-CML N = 43

- Single arm study, Ph+ CML patients refractory and/or intolerant to ≥3 TKIs including ponatinib
- Participating countries: USA, Belgium, France, Italy, Spain, Romania, Hungary, Singapore, UK, Korea

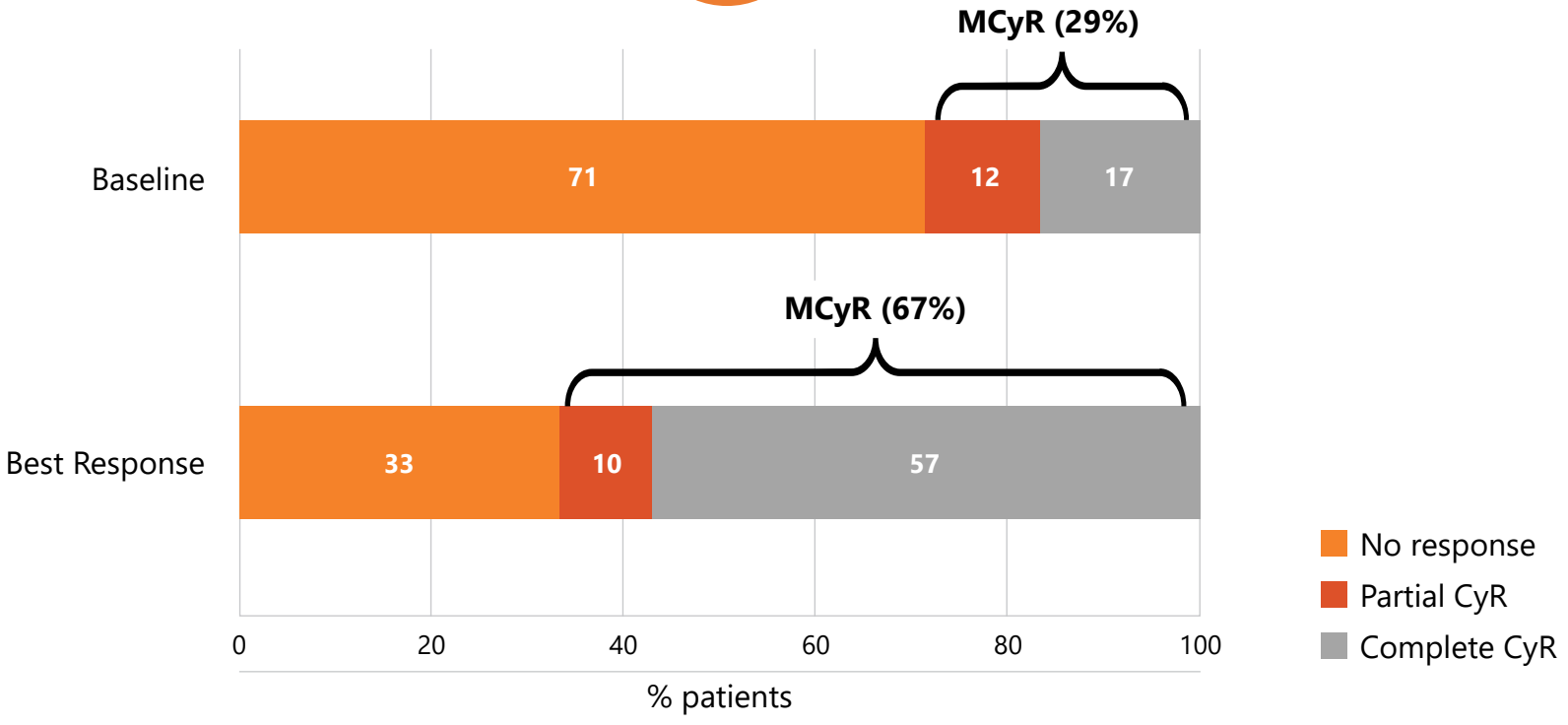
CP = chronic phase | AP = accelerated phase | BP = blast phase | Ph = Philadelphia chromosome | TKI = Tyrosine Kinase Inhibitor | CML = Chronic Myeloid Leukemia
NCT02629692

Vodobatinib (SCO-088) MAD study outcome

Major Cytogenetic Response (MCyR) rate more than doubles in CP-CML patients treated with vodobatinib

Cytogenetic Response

N=42



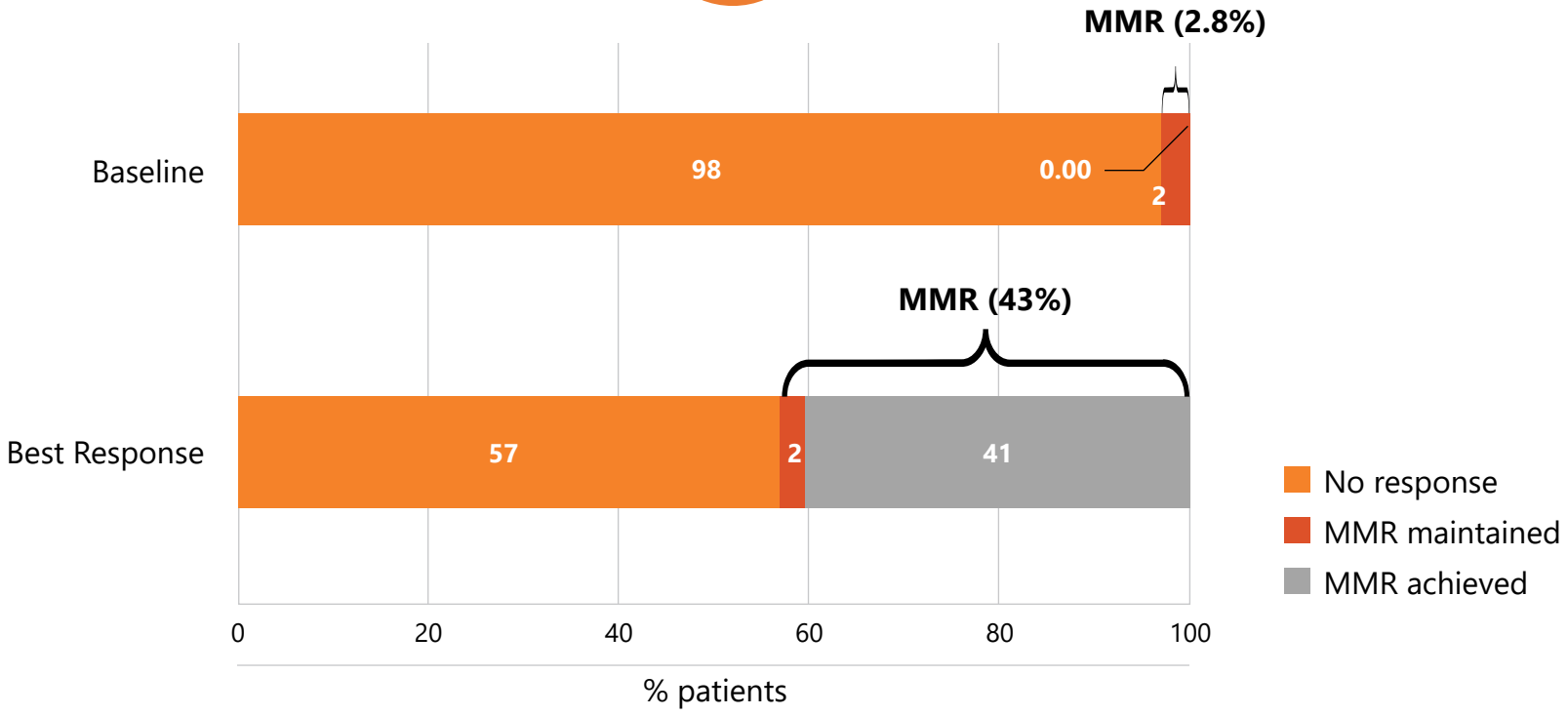
CyR = Cytogenetic response | MCyR = Major cytogenetic response | MCyR = PCyR + CCyR | Data presented at 2021 ASH Annual Meeting | Numbers rounded-off to nearest integer | CP-CML = Chronic Phase Chronic Myeloid Leukemia

Vodobatinib (SCO-088) MAD study outcome

Twenty-fold improvement in Major Molecular Response (MMR) rate in CP-CML patients treated with vodobatinib

Molecular Response

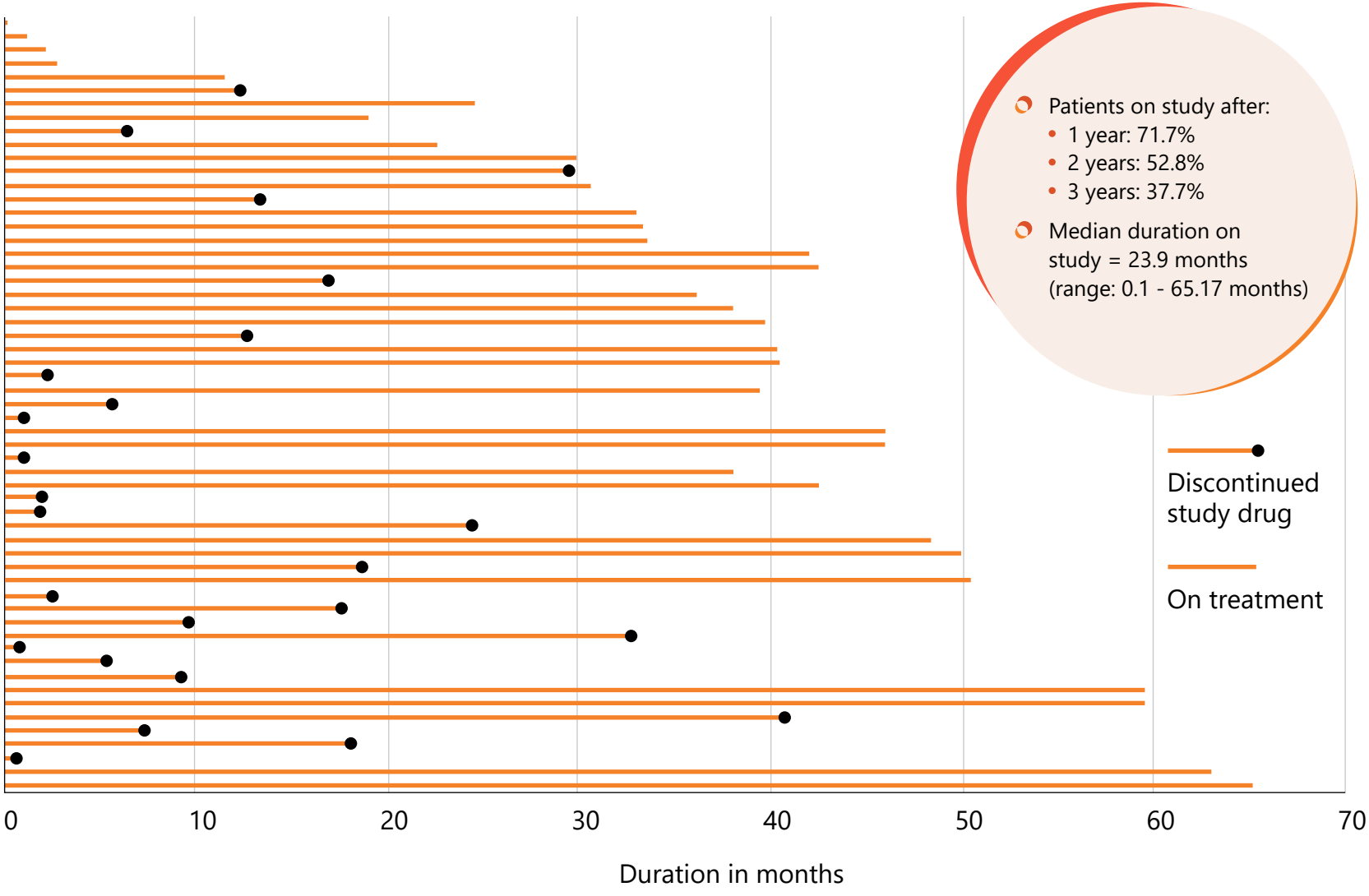
N=42



Major Molecular Response | Data presented at 2021 ASH Annual Meeting | Numbers rounded-off to nearest integer | CP-CML = Chronic Phase Chronic Myeloid Leukemia

Vodobatinib (SCO-088) MAD study outcome

Excellent efficacy and safety



Data cutoff: 1st September 2022

Next steps

Additional data to be presented at upcoming clinical conferences: the 2022 ESH John Goldman Conference and the 2022 ASH Annual Meeting

Pivotal study readout in FY24

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Vodobatinib for Neurodegenerative diseases (SCC-138)

A potential first-in-class disease modifying therapy targeting c-Abl

c-Abl: a critical component of neurodegeneration

Substantiated by multiple research groups

- Ubiquitous expression in nucleated cells
  - Expressed in all parts of the CNS (brain and spinal column, and peripheral neuronal tissue)
- Pivotal role in promoting neurodegeneration
  - Under oxidative stress, c-Abl is activated and phosphorylates a number of key substrates that bring about programmed death of oxidatively-stressed neurons

c-Abl and Parkinson's Disease: Mechanisms and Therapeutic Potential

Saurav Brahmachari[a,b,f,1], Senthilkumar S. Karuppagounder[a,b,f,1], Preston Ge[a,b,f,1], Saebom Lee[a,b], Valina L. Dawson[a,b,c,d,f,*], Ted M. Dawson[a,b,d,e,f,*] and Han Seok Ko[a,d,g,*]

Activation of tyrosine kinase c-Abl contributes to α-synuclein–induced neurodegeneration

Saurav Brahmachari, … , Ted M. Dawson, Han Seok Ko

c-Abl Inhibitors Enable Insights into the Pathophysiology and Neuroprotection in Parkinson's Disease

Dan Lindholm[1,2], Dan D. Pham[1,2], Annunziata Cascone[1], Ove Eriksson[1], Krister Wennerberg[3] and Mart Saarma[4]*

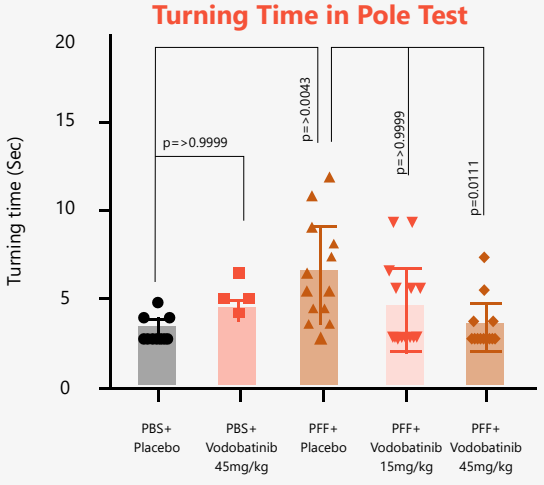
c-Abl phosphorylates α-synuclein and regulates its degradation: implication for α-synuclein clearance and contribution to the pathogenesis of Parkinson's disease

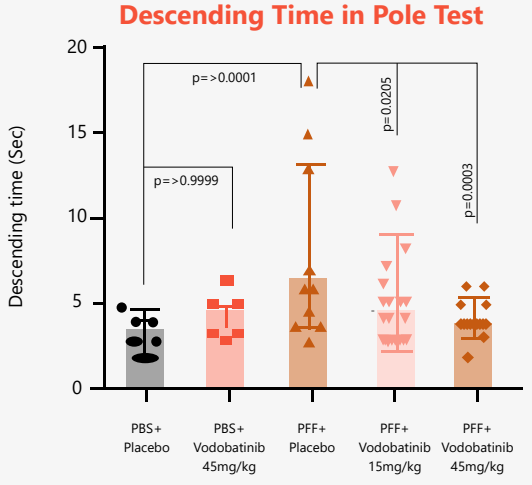
Anne-Laure Mahul-Mellier[1], Bruno Fauvet[1], Amanda Gysbers[3], Igor Dikiy[4], Abid Oueslati[1], Sandrine Georgeon[2], Allan J. Lamontanara[2], Alejandro Bisquertt[5], David Eliezer[4], Eliezer Masliah[5], Glenda Halliday[3], Oliver Hantschel[2] and Hilal A. Lashuel[1,*]

CNS = Central Nervous System

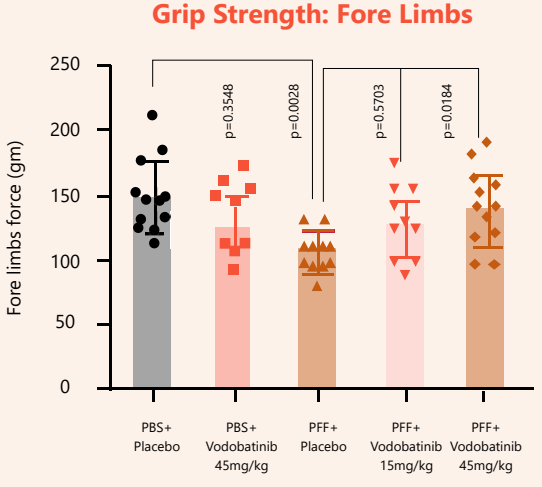
Vodobatinib improved motor and cognitive function in the PFF-induced mouse model[1]

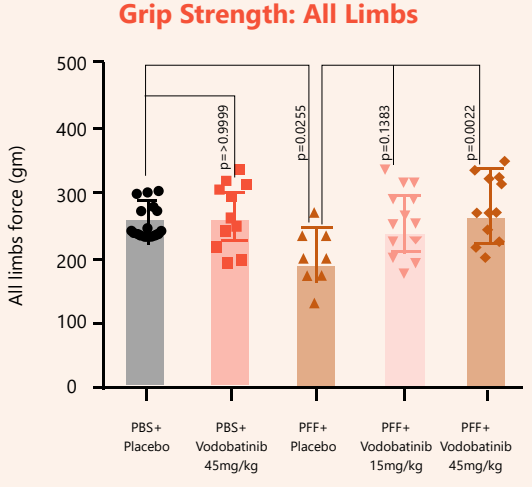
Vodobatinib at 45 mg/kg improves PFF-induced movement disorder-related deficits in Turning Time and Descending Time in the Pole test





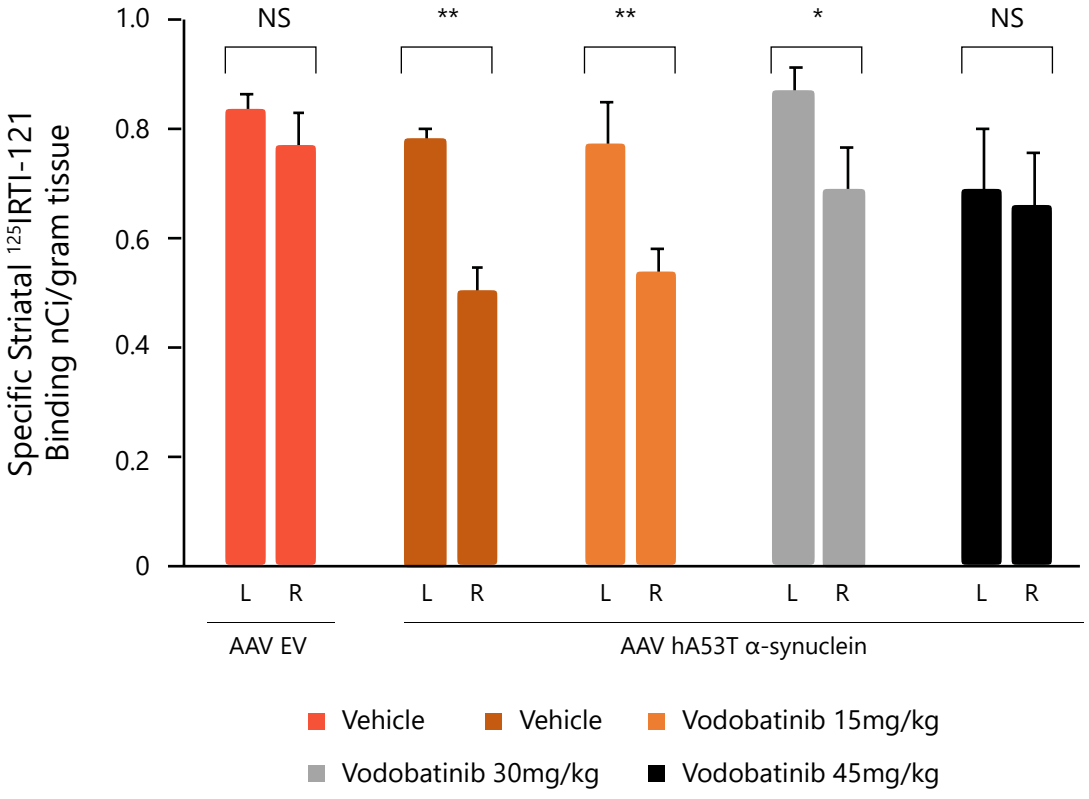
Vodobatinib treatment improves PFF-induced deficits in Grip Strength





1. Study conducted at the Ted Dawson Lab, Johns Hopkins University | PBS = Phosphate-buffered saline | PFF = Preformed fibril

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

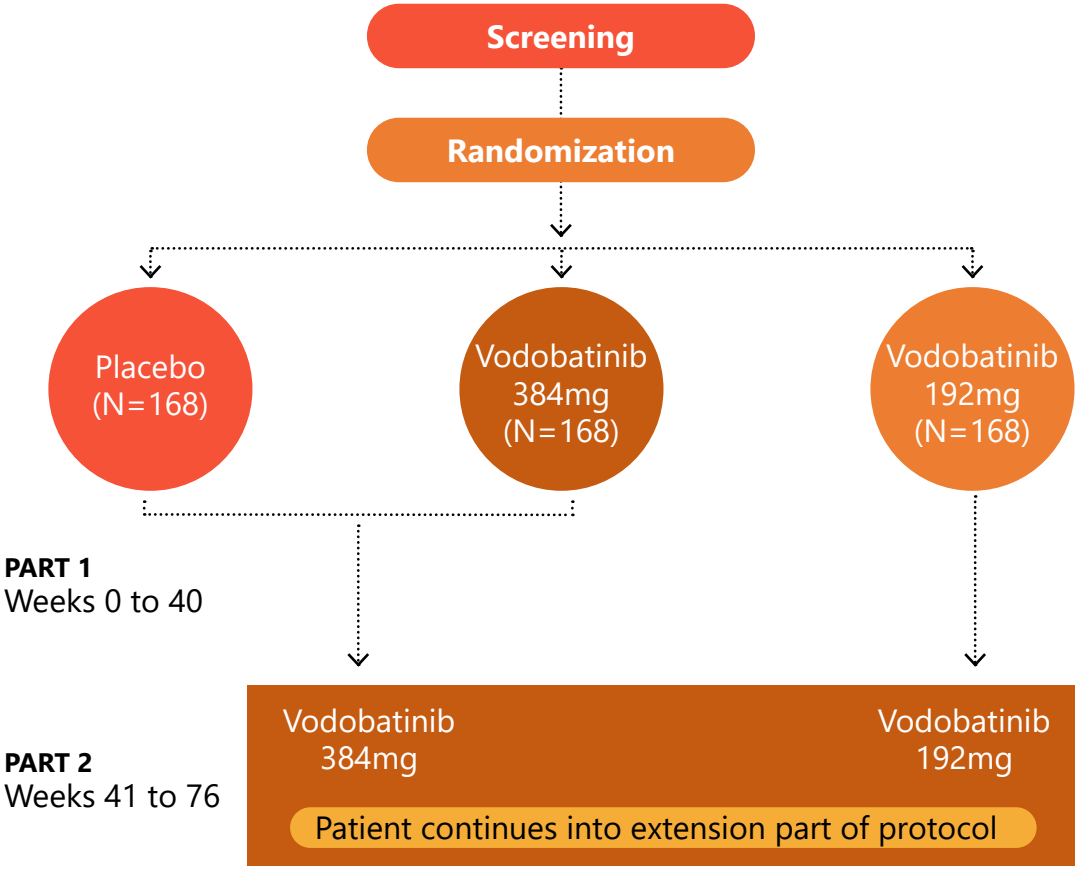


- Vodobatinib treatment protects against dopaminergic neuronal loss measured by radiolabeled ^{125}I labeled RTI-121 binding in the striatum
  - Comparison of un-operated left hemisphere (L) and operated right hemisphere (R, injected with & expressing the AAV) shows that 45 mg/kg dose provides protection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

AAV = Adeno Associated Virus | EV = Empty Vector

Vodobatinib for PD (SCC-138)

Recruitment on track to achieve enrollment target in PROSEEK



PROSEEK

- 77 sites across US, Europe and India functional; recruitment ongoing to complete enrollment in FY23
- Over 70% patients randomized (N=349)*

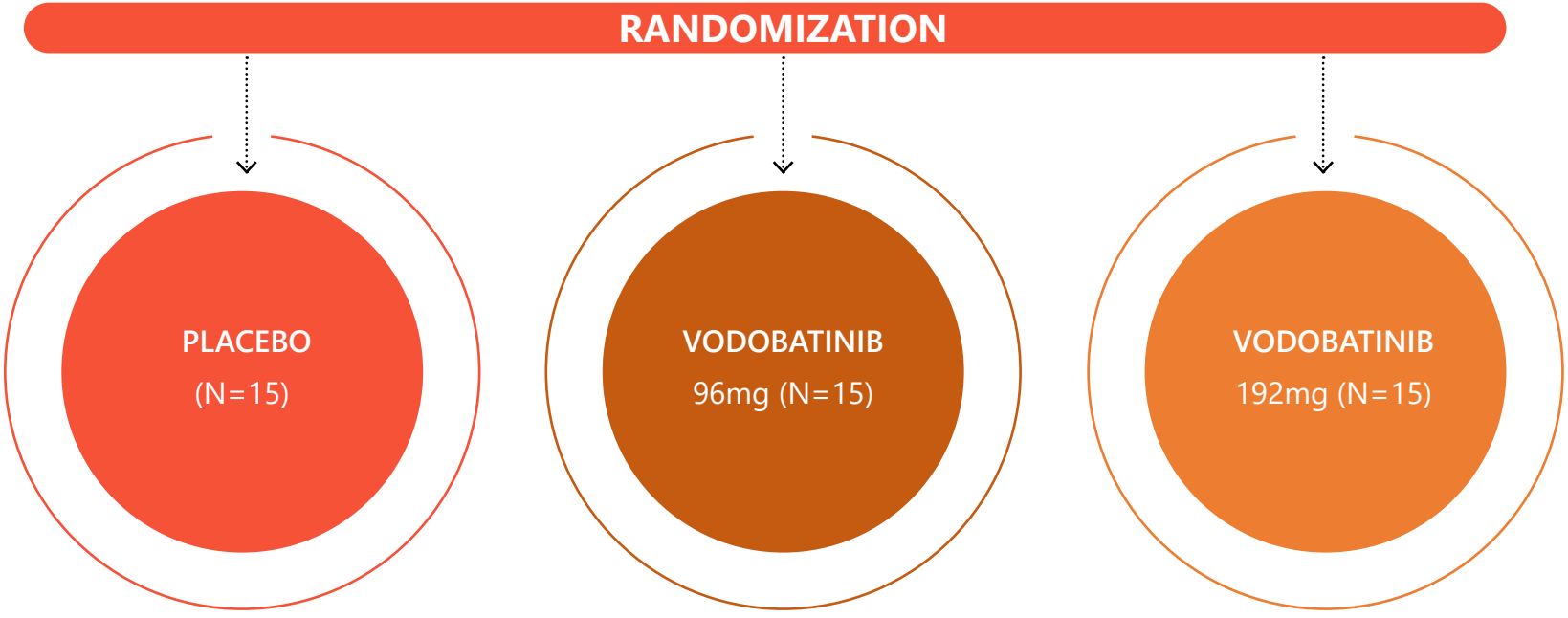
Extension study

- Patients enrolled to establish long-term safety and tolerability

*As of 14th Sept 2022 | PROSEEK = A Phase 2 Study In Early Parkinson's Disease Patients Evaluating The Safety And Efficacy Of Abl Tyrosine Kinase Inhibition Using K0706 | K0706 = Vodobatinib PD (SCC-138) | NCT03655236

Opportunities beyond PD: Lewy Body Dementia

Recruitment ongoing in an investigator-initiated clinical trial at Georgetown University



- Recruitment ongoing in a 12-week Phase 2 study in collaboration with Georgetown University
- 50% patients randomized
- Safety and tolerability being evaluated as a primary outcome
- Concentration of LBD-related plasma and CSF biomarkers form the set of secondary outcome measures

Next steps

Complete recruitment in PROSEEK

PROSEEK study readout in FY24

Complete recruitment by FY24 in the Georgetown University study

Study readout in FY24

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Vibozilimod (SCD-044) – A selective S1PR1 agonist

A safer alternative to JAK inhibitors

Vibozilimod (SCD-044) for Psoriasis and Atopic Dermatitis

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 not suitable for some indications because of safety concerns
- 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 increase the significance of S1PR1 agonists as a 'class alternative' in several autoimmune disorders, particularly in dermatology

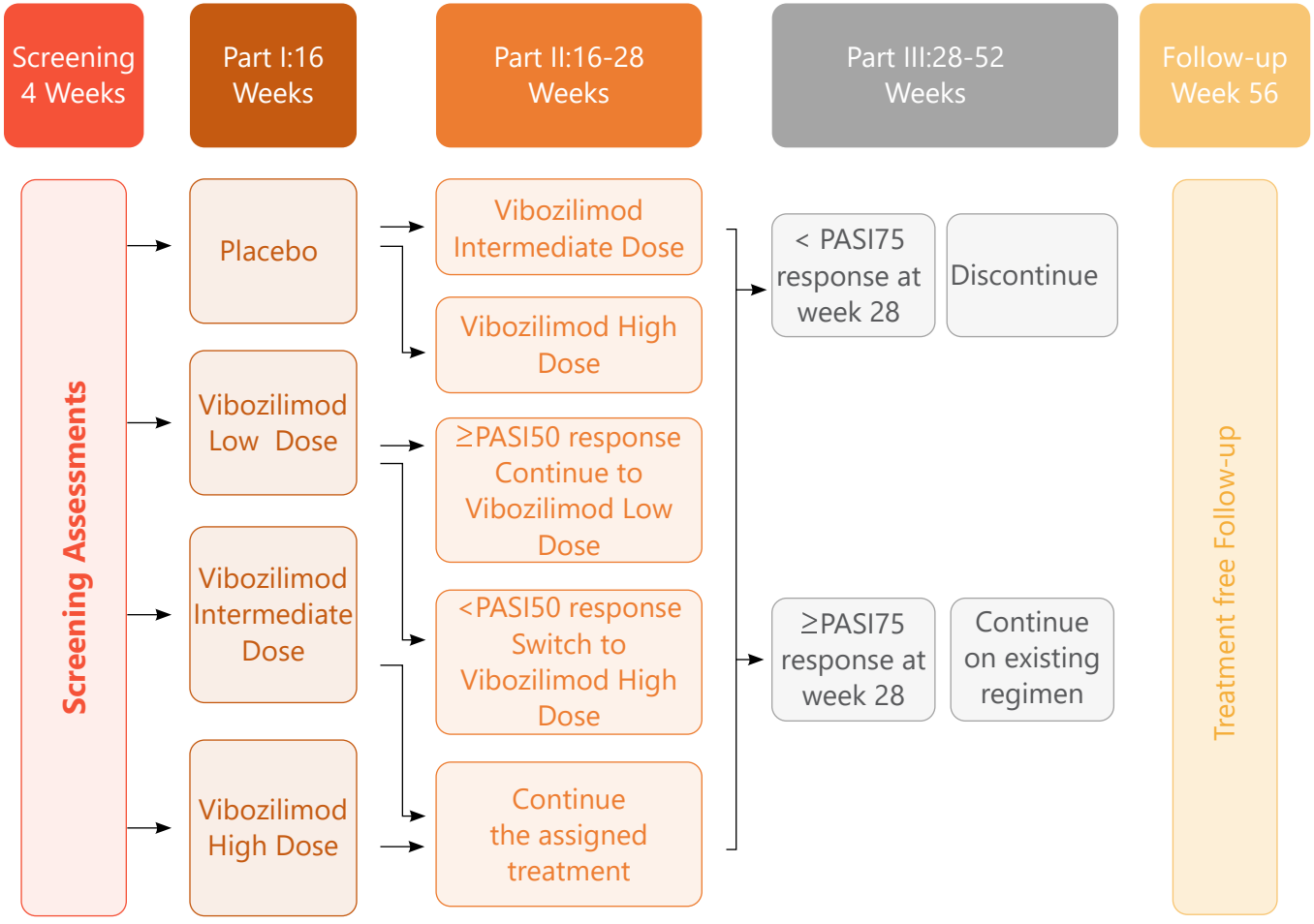
Vibozilimod (SCD-044)

- 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 = Sphingosine-1-Phosphate Receptor-1 | JAK = Janus Kinase | IP = Intellecual Property | IL-23 = Interleukin-23 | IBD = Inflammatory Bowel Disease

Vibozilimod (SCD-044) for Psoriasis

Phase 2 study design

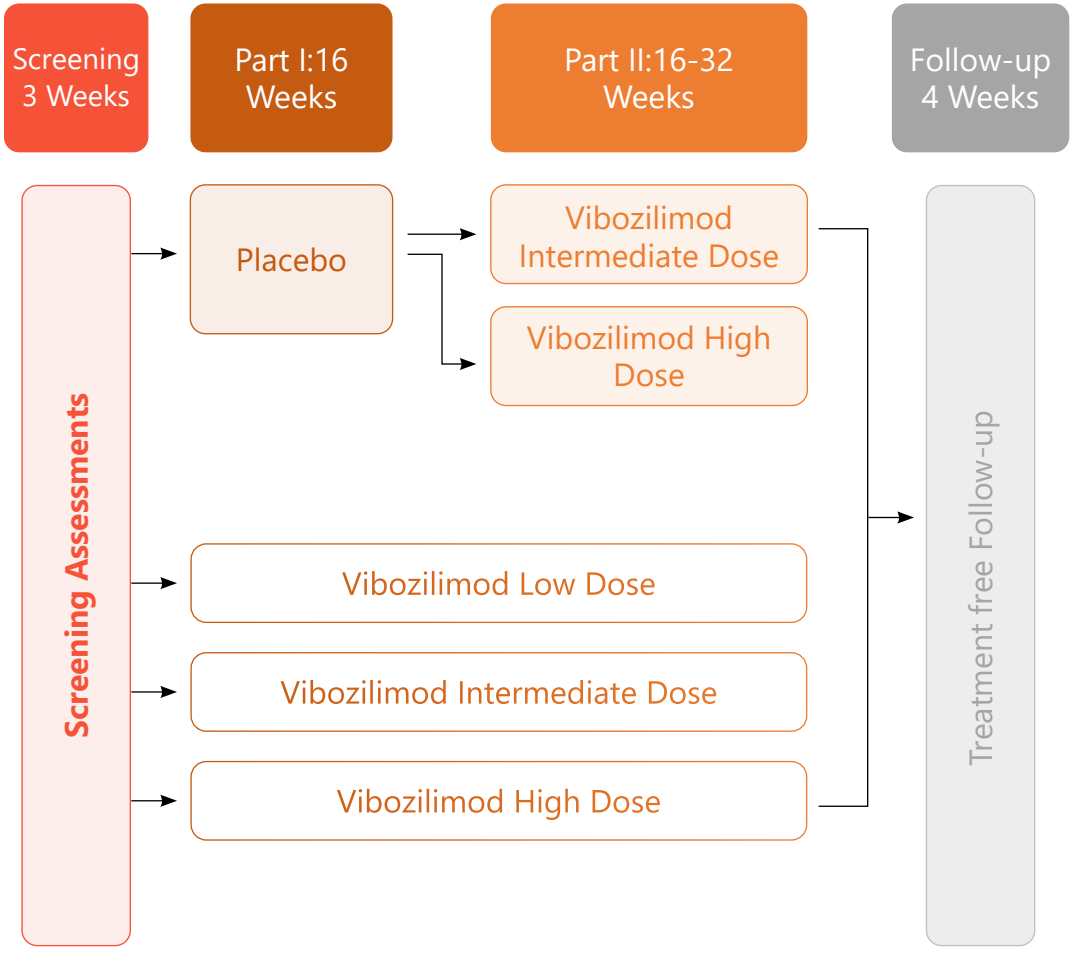


- Primary endpoint – Proportion of patients with PASI75 response at week 16
- 240 patients across three dose levels and placebo. Currently in early stage ramp-up
- Study now open in the US. Expected to expand to Latin America and Europe to accelerate in the coming months

PASI = Psoriasis Area and Severity Index | NCT04566666 | (SCD-044-19-14, Version 1, September 23, 2020)

Vibozilimod (SCD-044) for Atopic Dermatitis

Phase 2 study design



- 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 open in the US. Expected to expand to Latin America and Europe to accelerate in the coming months

EASI = Eczema Area and Severity Index | NCT04684485 | (SCD-044-19-14, Version 1, November 9, 2020)

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SCO-120 for HR^{+}/ HER2^{-} MBC

An oral SERD with brain penetration

Current treatment paradigm

Dominated by endocrine therapy except in patients with visceral disease

Endocrine backbone	AI / Tamoxifen	AI	Fulvestrant / Exemestane
Combination partner		CDK 4/6i	CDK 4/6i or mTORi or PI3Ki

- Fulvestrant is currently the only SERD available for patients failing 1L setting
  - It is limited by intramuscular (IM) administration and its inability to address mutations
  - Elacestrant phase 3 study met its co-primary endpoints of improved PFS in patients with wild type and mutant disease in 2nd line patients.
- CDK4/6i has emerged as the gold standard in 1L but requires an endocrine backbone
  - SERDs in development have the potential to become that backbone

SERD – Selective Estrogen Receptor Degrader | AI – Aromatase Inhibitor | ESR1 – Estrogen Receptor 1
CDK – Cyclin-Dependent Kinase | PI3K – Phosphoinositide-3-Kinase | PFS – Progression Free Survival | mTORi - Mammalian Target of Rapamycin inhibitor

SCO-120: Oral SERD for HR^{+}/HER2^{-} MBC

Clinical study design

	Single Ascending Dose (Part A)	Food Effect (Part B)	Multiple Ascending Dose (Part C)
Phase 1 Healthy Volunteer Study Design	Double blind, placebo controlled, single oral dose	Open label, two period, cross over, single dose, fast/fed study	Double blind, placebo controlled, once daily, 14 day repeat dose

	Multiple Ascending Dose study
Phase 1 Patient study	HR^{+}/HER2^{-} metastatic breast cancer patients that have failed at least 1 prior endocrine therapy and no more than 3 prior chemotherapy treatments

Dose escalation
(MTD/RP2D, safety)
(N~44)

→ **MTD/RP2D reached** →

Dose expansion
(Safety & Preliminary efficacy) (N~105)

- Part a: ESR1 mutations
- Part b: Resistant to AI ± CDK4/6i
- Part c: Resistant to AI & Ful+ CDK4/6i
- Part d: Secondary brain metastases to breast cancer

MBC = Metastatic Breast Cancer | MTD = Maximum Tolerated Dose | RP2D = Recommended Phase 2 Dose | ESR1 = Estrogen Receptor 1 | AI = Aromatase Inhibitor | CDK = Cyclin Dependent Kinase | Ful = Fulvestrant

Next steps

MAD in patients study completion and read out in FY24

Phase 2 initiation in FY24

Target NDA submission in FY27

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Biologics

Nitin Damle

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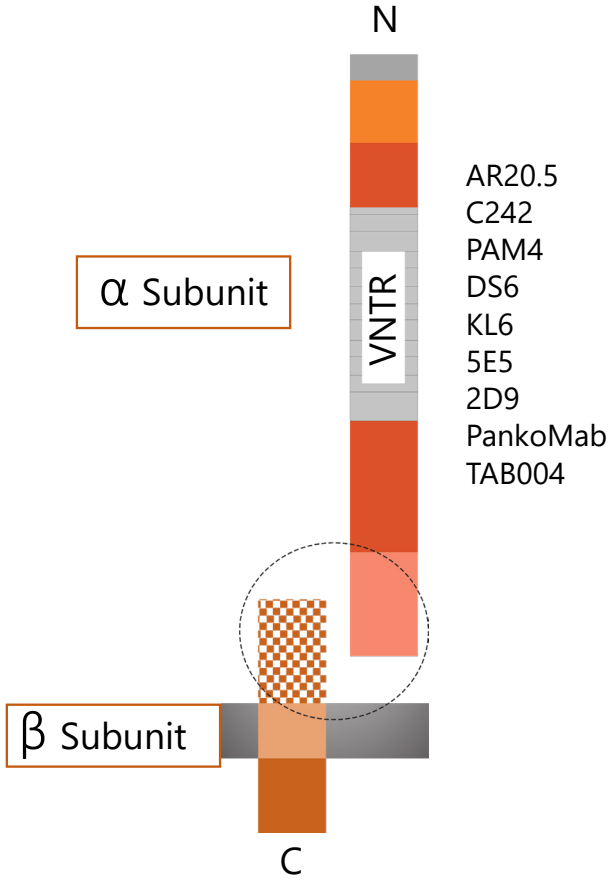
SBO-154
(Anti-MUC-1 ADC)

Targeting an antigen expressed in a wide spectrum of tumors

SBO-154: Anti-MUC-1 ADC

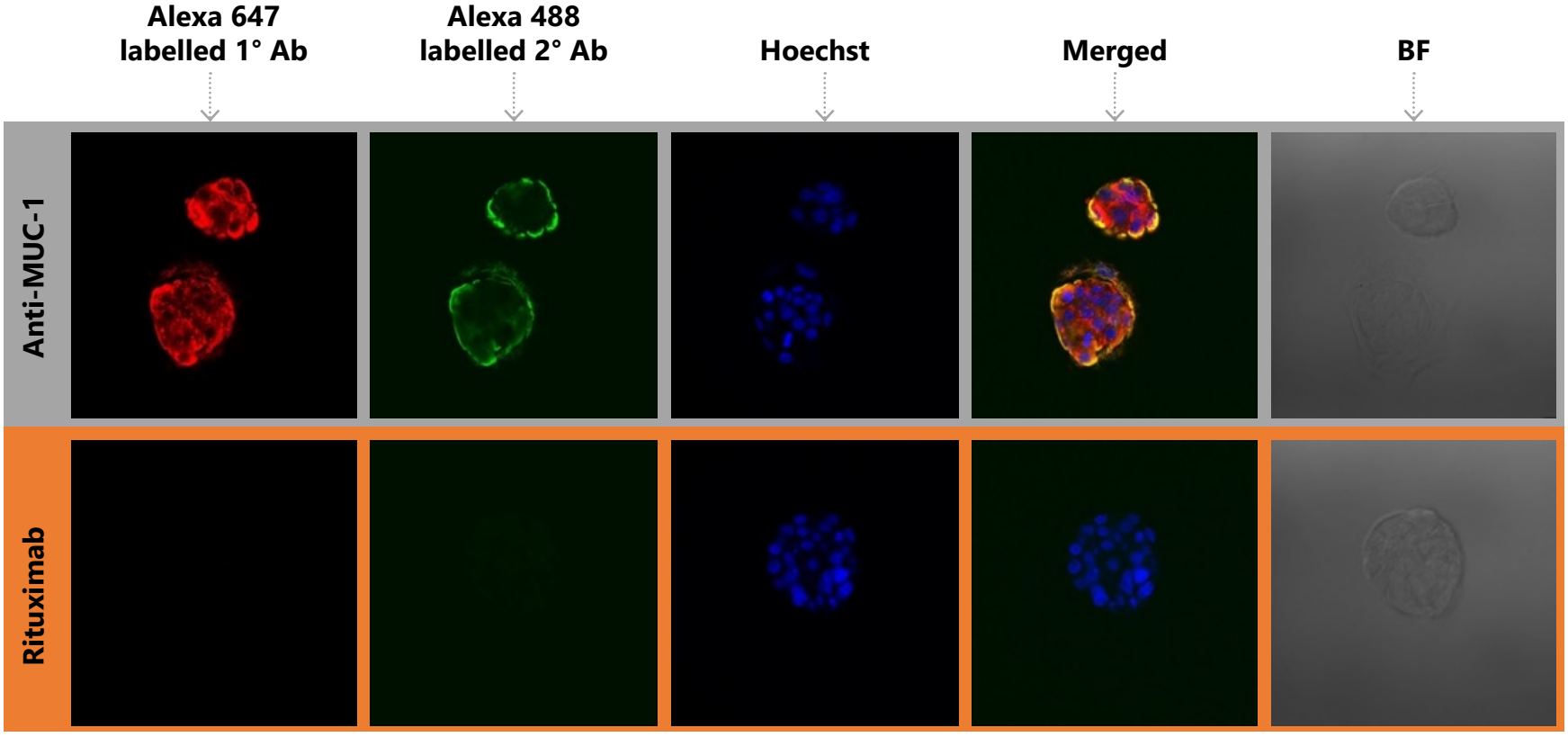
Novel approach to target α/β complex, with an opportunity to target multiple tumor types

- Tumor agnostic opportunity in-licensed from Biomodifying LLC*
- MUC-1 expressed extensively in majority of tumors
- Preclinical PoC of anti-tumour efficacy of anti-MUC-1 targeted ADC established
- Most anti-MUC-1 mAbs under development target VNTR in the MUC-1α
  - Circulating MUC-1α in plasma and in peritumoral space block meaningful tumor targeting by MUC1α-targeted therapies
  - Primary reason for the lack of efficacy
- No directly competing agents targeting α /β junction
- Potential to be an anchor for other constructs like bi-specific/multi-specific antibodies, naked mAb, etc.



*Anti-MUC-1 antibodies licensed from Biomodifying LLC | MUC-1 - Mucin-1 | ADC - Antibody Drug Conjugate | PoC – Proof-of-concept

Anti-MUC-1 mAbs* internalize in pancreatic carcinoma cells

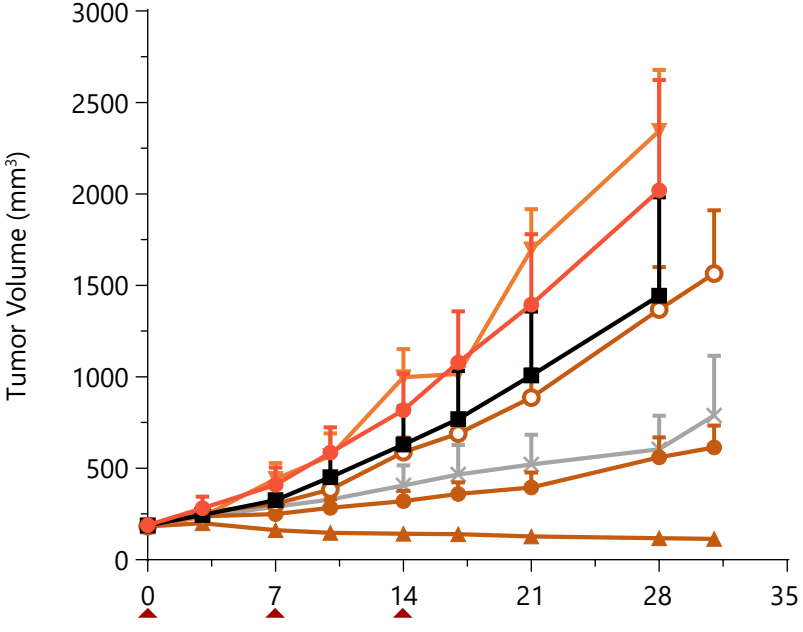


Red fluorescence is associated with anti-MUC-1 antibody, green fluorescence is associated with anti-human Fc-γ antibody & Hoechst dye stains nucleus and is blue in colour

*Anti-MUC-1 antibodies licensed from Biomodifying LLC

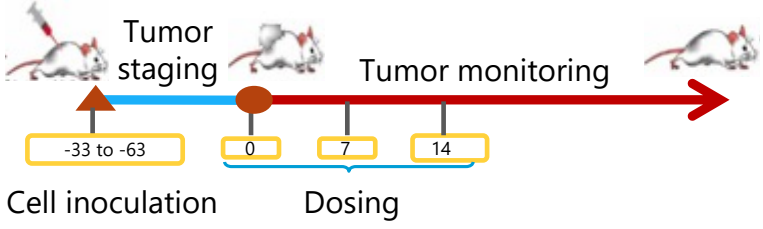
SBO-154 strongly inhibits growth of MUC-1 expressing tumors

Established in subcutaneous pancreatic carcinoma xenografts



Regimen: Q7D x 3, i.p.

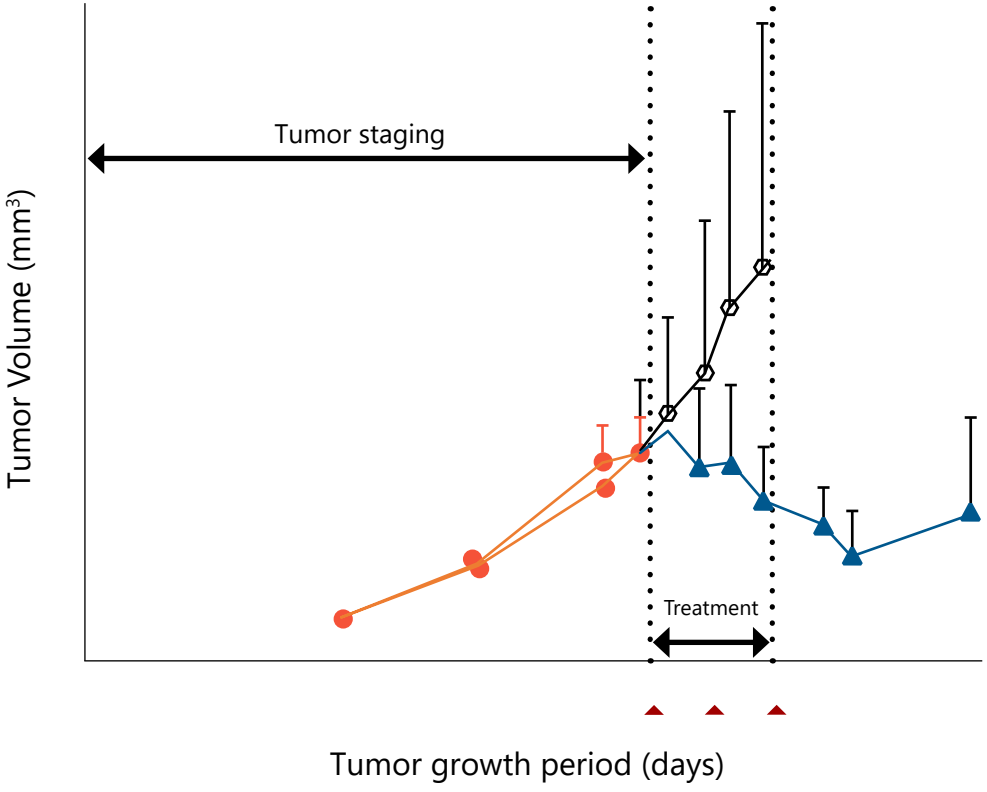
- Free Payload
- Vehicle, 10ml/kg
- Rituximab-DC
- SBO-154, low dose
- Nab-PTX
- SBO-154, medium dose
- SBO-154, high dose



- Dose-dependent efficacy observed with SBO-154, with sustained regression observed at high-dose
- Rituximab-drug conjugate and paclitaxel do not show similar activity

DC = Drug Conjugate | Nab-PTX = Paclitaxel

SBO-154 causes regression of large tumor mass xenografts of a pancreatic carcinoma cell line



Treatment: ip Q7Dx3

- Vehicle
- Rituximab-DC
- SBO- 154

- Large tumor study is used to determine cytoreductive potential
- SBO-154 but not isotype matched control (rituximab-DC) causes significant regression of large established pancreatic tumor xenografts

DC = Drug Conjugate

Next steps

IND enabling tox studies completion

Pre-IND meeting

IND filing in FY24

sparc

Preclinical NCE Program

Vikram Ramanathan

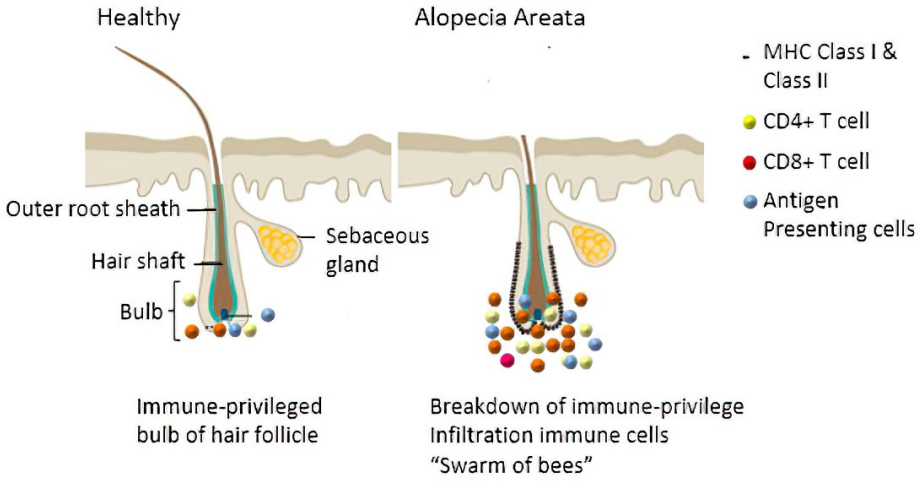
sparc

SCD-153 for Alopecia Areata

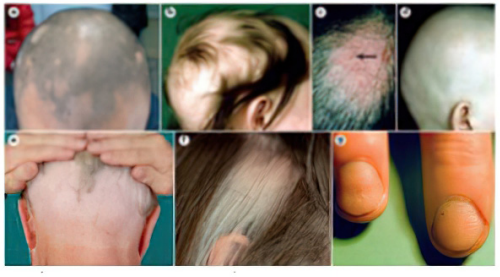
A potential first-in-class opportunity in an autoimmune disease with significant unmet need

Alopecia Areata – Autoimmune disease causing hair loss

Hair follicles lose immune privilege and they move into Telogen (resting) phase



Clinical manifestations of Alopecia Areata



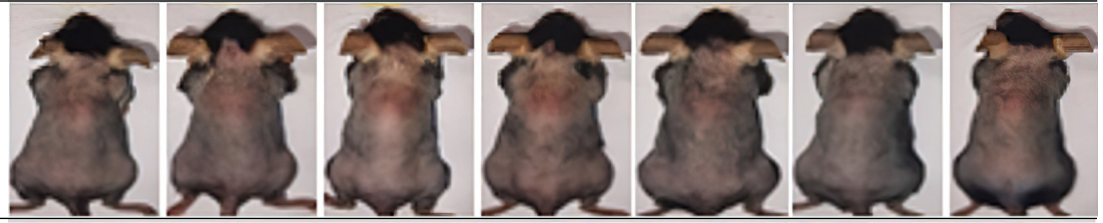
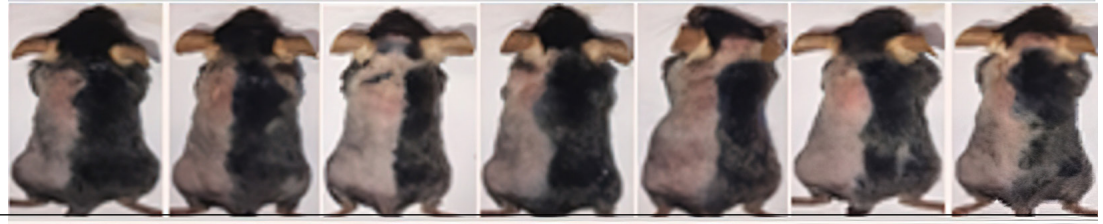
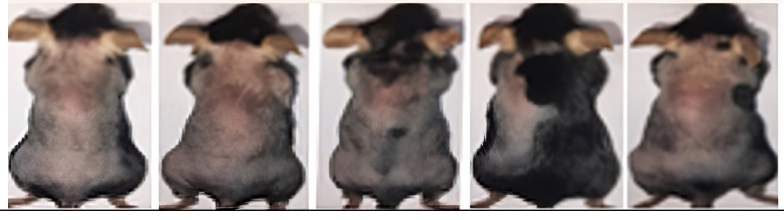
Alopecia Areata is characterized by

- Rapid progression of hair follicle from anagen (growing) phase to catagen (transition) phase to telogen (resting) phase
- Collapse in immune privilege in hair follicle bulb
  - $CD4^+$ and $CD8^+$ T cells infiltrate and damage the hair bulb
  - NKG2D positive CD8+ T cells are the major effectors of hair follicle damage
  - Alters normal hair growth cycle and causes hair to fall out
- However, the hair follicle structure and stem cells are preserved, suggesting potential for hair growth

Adapted from Nature Reviews. Disease Primers. 3:17011, 2017

SCD-153 stimulates hair growth in animal models

C57BL/6 telogen - anagen alopecia model

Vehicle, QOD, 2 applications	
SCD-153, QOD, 2 applications	
Tofacitinib, QOD, 2 applications	

Female mice, 8.5 weeks, Dorsal hair clipped. Treated on right side, left side is untreated. QOD: every other day

- SCD-153 stimulates robust hair growth after 2 doses given on alternate days
- Promotes re-entry into anagen possibly via activation of stem cells at the base of the hair follicle

SCD-153 stimulates hair growth in animal models

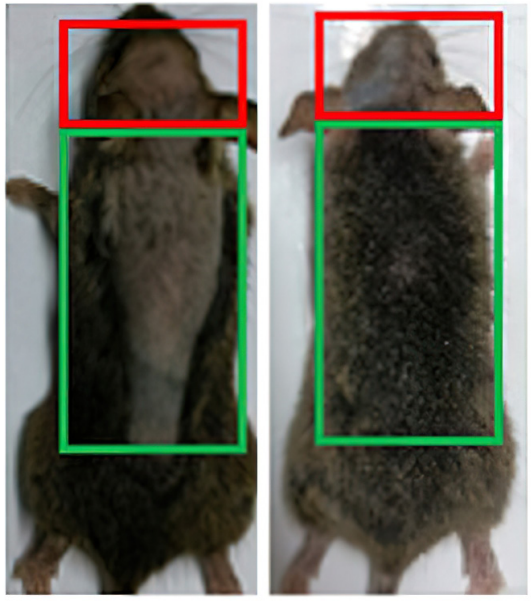
C3H/HeJ alopecia areata immune disease model

Red: Untreated

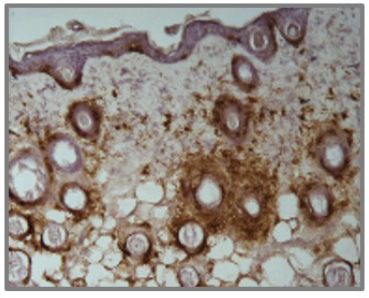
Green: Treated

Before

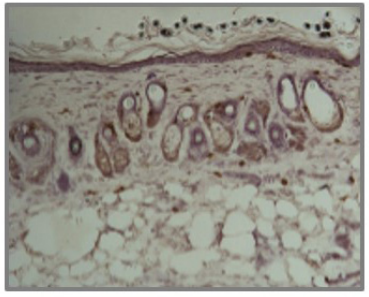
After



Untreated



Treated



- Robust hair growth in a disease model
- Decrease in $CD8^+$ immunostaining proximal to hair follicles. Reduction in "swarm of bees"
- Decrease in gamma-interferon and CTL gene signature

Next steps

IND enabling tox studies completion

IND filing in FY23

sparc

Financial Update

Chetan Rajpara

Financial summary

Year	FY18	FY19	FY20	FY21	FY22	Q1FY23
USDINR	64.46	69.95	70.91	74.23	74.49	77.16
						INR Cr
Total Income	83	196	87	258	144	29
Total Expenses	329	342	399	410	347	111
Exceptional Item	49	-	-	-	0	0
Profit / (Loss) after Tax	(197)	(145)	(312)	(151)	(203)	(82)
						USD Mn
Total Income	12.9	28.1	12.2	34.8	19.3	3.7
Total Expenses	51.1	48.9	56.3	55.2	46.6	14.4
Exceptional Item	7.6	-	-	-	-	-
Profit / (Loss) after Tax	(30.6)	(20.8)	(44.1)	(20.4)	(27.3)	(10.7)

Cash and liquidity

- Issued convertible warrants for Rs. 1,112 Cr (~USD 148 Mn) in July 2021 by way of preferential issue
- Received Rs. 409 Cr (~USD 55 Mn) being 25% payable on application & upon conversion of warrants
- Balance Rs. 703 Cr (~USD 93 Mn) to be received by Dec 2022 upon conversion of warrants by investors
- Line of credit from parent company Rs. 250 Cr (~USD 31 Mn) and bank facility for Rs. 245 Cr (~USD 31 Mn) in place, of which Rs. 183 Cr (~USD 23 Mn) is utilized as on Sept 30, 2022
- Obtained shareholders' fresh approval in Sep 2022 for raising additional sum up to Rs. 1,800 Cr (~USD 225 Mn) by way of issuance of fresh equity or debt

THANK YOU