SCHOLAR ROCK

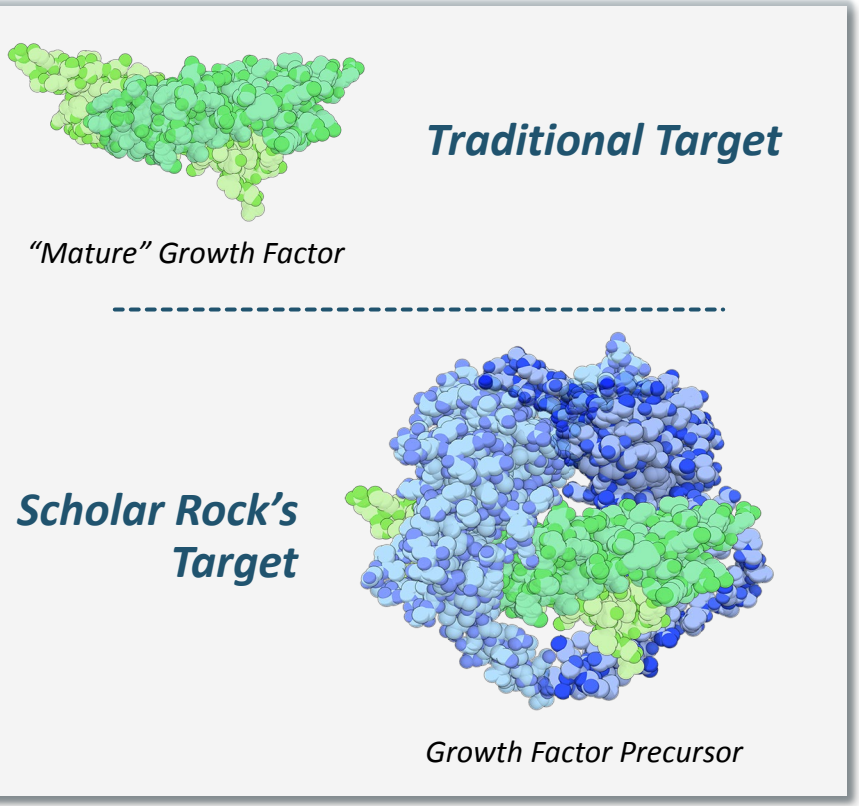
From New Insights to New Medicines

August 2019

Disclaimers

Various statements in this presentation concerning Scholar Rock's future expectations, plans and prospects, including without limitation, Scholar Rock's expectations regarding its strategy, its product candidate selection and development timing, including timing for the initiation of and reporting results from its clinical trials for its product candidates, its disease indication selection and timing for such selection, the ability of SRK-015 to affect the treatment of patients suffering from Spinal Muscular Atrophy (SMA) either as a monotherapy or in conjunction with the current standard of care, the ability of SRK-181 to affect the treatment of cancer patients in a manner consistent with preclinical data, and the projected use of cash constitute forward-looking statements for the purposes of the safe harbor provisions under The Private Securities Litigation Reform Act of 1995. The use of words such as "may," "might," "will," "should," "expect," "plan," "anticipate," "believe," "estimate," "target," "project," "intend," "future," "potential," or "continue," and other similar expressions are intended to identify such forward looking statements. Actual results may differ materially from those indicated by these forward-looking statements as a result of various important factors, including, without limitation, Scholar Rock's ability to provide the financial support and resources necessary to identify and develop multiple product candidates on the expected timeline, competition from others developing products for similar uses, Scholar Rock's ability to obtain, maintain and protect its intellectual property, Scholar Rock's dependence on third parties for development and manufacture of product candidates including to supply any clinical trials, and Scholar Rock's ability to manage expenses and to obtain additional funding when needed to support its business activities and establish and maintain strategic business alliances and new business initiatives as well as those risks more fully discussed in the section entitled "Risk Factors" in the Quarterly Report on Form 10-Q for the quarter ended June 30, 2019, which is on file with the Securities and Exchange Commission, as well as discussions of potential risks, uncertainties, and other important factors in Scholar Rock's subsequent filings with the Securities and Exchange Commission. Any forward-looking statements represent Scholar Rock's views only as of today and should not be relied upon as representing its views as of any subsequent date. Scholar Rock explicitly disclaims any obligation to update any forward-looking statements unless required by law.

A Novel Approach to Targeting Growth Factor Signaling



Traditional approaches to targeting growth factors have been limited by:

- Structural similarities
- Overlapping sets of related receptors
- Diverse and overlapping physiological roles

Scholar Rock's novel approach:

- Targeting signaling proteins at a cellular level
- Nature's way of regulating growth factor activity
- Targeting activation of growth factor precursors has the potential to offer:
  - High selectivity
  - Potency of inhibition
  - Localization of effect
- Approach has applicability over a wide variety of growth factors

Robust Pipeline Portfolio

	Target / Program	Indication	Discovery / Early Preclinical	Preclinical	Phase 1	Phase 2	Rights / Partner	Next Anticipated Milestone
INTERNAL PROPRIETARY PROGRAMS								
Pro/Latent Myostatin	SRK-015	Spinal Muscular Atrophy (3 distinct Type 2 and Type 3 patient populations)						Preliminary PK/PD Data by End of 2019
Pro/Latent Myostatin	SRK-015	Myostatin-Related Disorders						Identify Next Indication in 2020
Latent TGFβ	SRK-181 (Context-Independent Latent TGFβ1)	Immuno-oncology (Primary resistance to CBTs*)						Initiate Phase 1 Trial Mid-2020
Latent TGFβ	SRK-181 (Context-Independent Latent TGFβ1)	Oncology						
Latent TGFβ	Context-Dependent Latent TGFβ1 / Immune Cell	Oncology/Immuno-oncology						
RGMc	BMP6 Signaling Pathway (anti-RGMc)	Iron-Restricted Anemias						Nominate Product Candidate in 1H20
PARTNERED PROGRAMS								
Latent TGFβ	Context-Independent Latent TGFβ1	Fibrosis					GILEAD	
Latent TGFβ	Context-Dependent Latent TGFβ1 / LTBP1 & LTBP3	Fibrosis					GILEAD	
Latent TGFβ	Undisclosed Program	Fibrosis					GILEAD	
Latent TGFβ	Context-Dependent Latent TGFβ1 / GARP	Oncology/Immuno-oncology					Janssen Biotech, Inc	

**CBTs = checkpoint blockade therapies*

Upcoming Key R&D Milestones

SRK-015 in SMA

- ✓ Initiate Phase 2 SMA proof-of-concept trial by the end of 1Q19
- ✓ Commence patient dosing in Phase 2 SMA proof-of-concept trial in 2Q19
- ✓ Present final Phase 1 results at Cure SMA Annual Conference being held June 28-July 1, 2019
- ○ Announce Phase 2 trial read-outs:
  - Preliminary PK/PD analysis by end of 2019
  - Interim efficacy and safety analysis at 6 months in 1H20
  - Top-line results of 12-month treatment period 4Q20-1Q21

SRK-015

- ○ Identify next indication in 2020
  - Neuromuscular disorders
  - Other myostatin-related disorders

TGFβ1 Inhibitor

- ○ Advance cancer immunotherapy product candidate, SRK-181, into a Phase 1 trial mid-2020
- ○ Announce initial data from Phase 1 trial of SRK-181 in patients with solid tumors by end of 2021
- ○ Continue to advance active discovery programs for context-dependent inhibition of TGFβ1
- ○ Conduct fibrosis discovery and preclinical studies in partnership with Gilead

RGMc

- ○ Nominate product candidate in 1H20

SRK-015: Inhibitor of Myostatin Activation

Potential First Muscle-Directed Therapy for SMA

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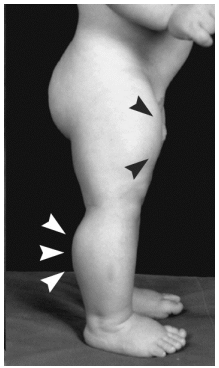
SRK-015: Highly Specific Inhibitor of Latent Myostatin



- Myostatin is a genetically-validated, negative regulator of muscle mass expressed in skeletal muscle tissue
- Vertebrates lacking the myostatin gene are healthy and display increased muscle mass and strength

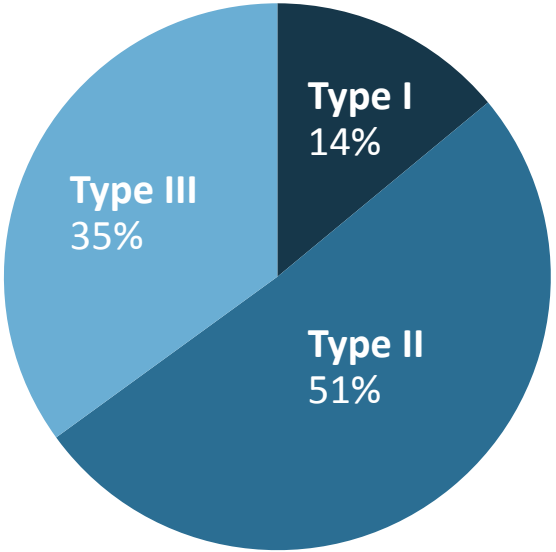
Differentiated approach with SRK-015:

- ***Fully human monoclonal antibody (mAb)***
- ***Highly selective inhibitor of the activation of myostatin precursor***
- ***Half-life of 23-33 days***
- ***Orphan Drug Designation for SMA granted by FDA and EC***
- ***US Patent 9,758,576 covers mAbs that inhibit the activation of the myostatin precursor (expiry in 2034)***

SRK-015 Opportunity in Spinal Muscular Atrophy

Overall Prevalence of 30,000-35,000 in U.S. and Europe

Relative Prevalence Among Patients Living With SMA



Type I:

- Infant-onset; often fatal

Type II and non-ambulatory type III:

- Later-onset but still early childhood
- Severe deficits in motor function

Ambulatory type III:

- Limited mobility and substantial morbidity

Type IV:

- Population not well-characterized

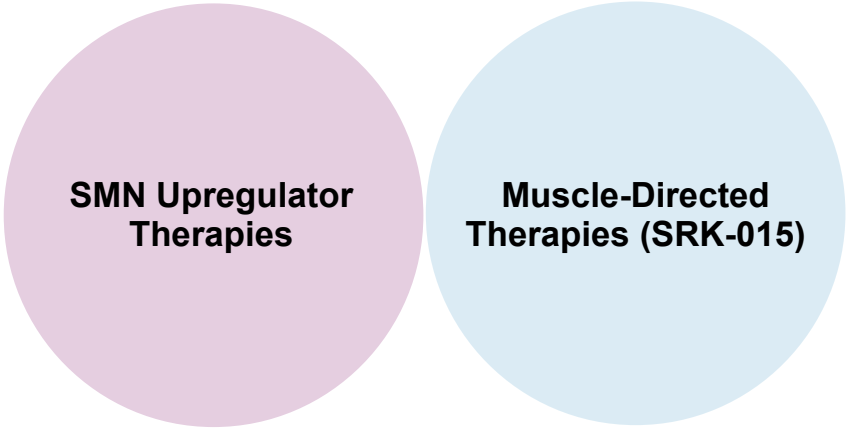
Focus of Phase 2 Trial

Potential to use SRK-015 in conjunction with SMN upregulators

Potential to use SRK-015 as monotherapy or in conjunction with SMN upregulators

Lally et al, Orphanet Journal of Rare Diseases, 2017

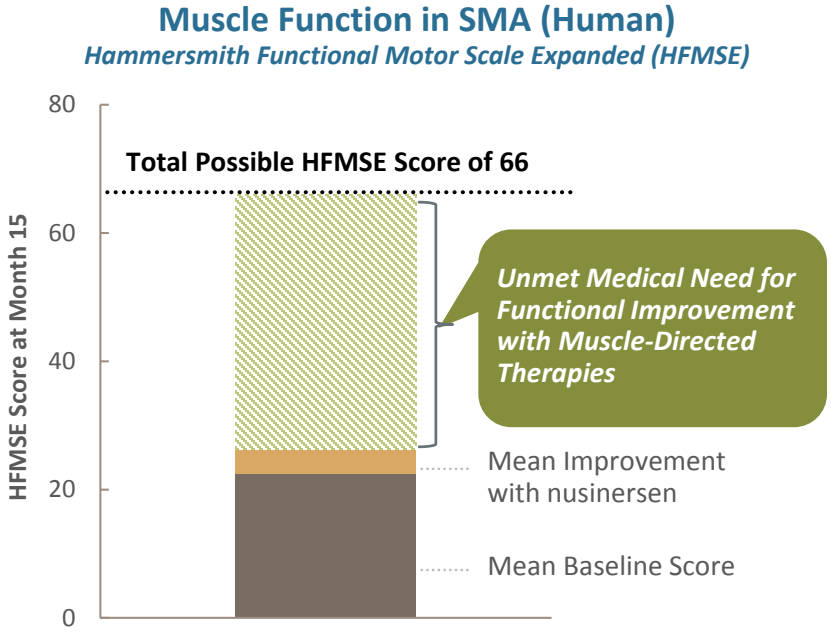
Significant Unmet Need Remains Despite Current Therapies



Address SMN deficiency to prevent further motor neuron deterioration

Act directly on muscle with aim to improve functional performance

SRK-015 has the potential to drive functional performance across a range of severity observed in SMA either as a monotherapy or in conjunction with any SMN upregulator/corrector therapy



Mean improvement in HFMSE score experienced by patients with later-onset SMA in the Phase 3 CHERISH clinical trial of nusinersen

SMN = survival motor neuron

SRK-015 Preclinical and Phase 1 Data Support Evaluation in SMA

Preclinical and translational data support myostatin as a drug target in SMA

- Confirmed presence of target in disease setting
- Achieved multi-fold increase in serum latent myostatin levels indicating target engagement
- Treatment of SMNΔ7 mouse model led to improved muscle mass and strength

Phase 1 data in healthy volunteers showed robust target engagement and no apparent safety signals

- No dose-limiting toxicities identified up to highest evaluated dose of 30 mg/kg
- Serum half-life of 23-33 days supports planned evaluation of once every 4-week (Q4W) dosing in Phase 2
- Single dose of SRK-015 led to marked increases in serum concentrations of latent myostatin; no meaningful change observed with placebo
- **Target saturation**: peak latent myostatin levels plateaued starting with a single dose at 3 mg/kg
- **Durability of saturation**: plateau was sustained up to Day 140 after multiple doses at 20 mg/kg

Phase 1 results provide first proof-of-mechanism in humans of Scholar Rock's therapeutic approach of targeting the latent form of growth factors

SRK-015 Target Profile in SMA

GOALS	EVIDENCE TO DATE
Effectively increase motor function to drive clinically meaningful outcomes	✓ Translational/preclinical data support myostatin as a drug target in SMA ✓ Preclinical data demonstrate potential for substantial increases in muscle strength ✓ Phase 1 PD data demonstrate SRK-015 can successfully engage the target in a durable fashion
Safety profile to enable chronic dosing, including in pediatric populations	✓ Well-tolerated with no apparent safety signals based on Phase 1 data ✓ Binds myostatin precursors with high selectivity in vitro
Low drug administration burden to offer broad accessibility	✓ Minimally invasive route of administration (IV) ✓ PK and PD data support an infrequent dosing regimen (e.g. once every 4 weeks)

Emerging evidence supports investigating the safety and efficacy of SRK-015 in SMA

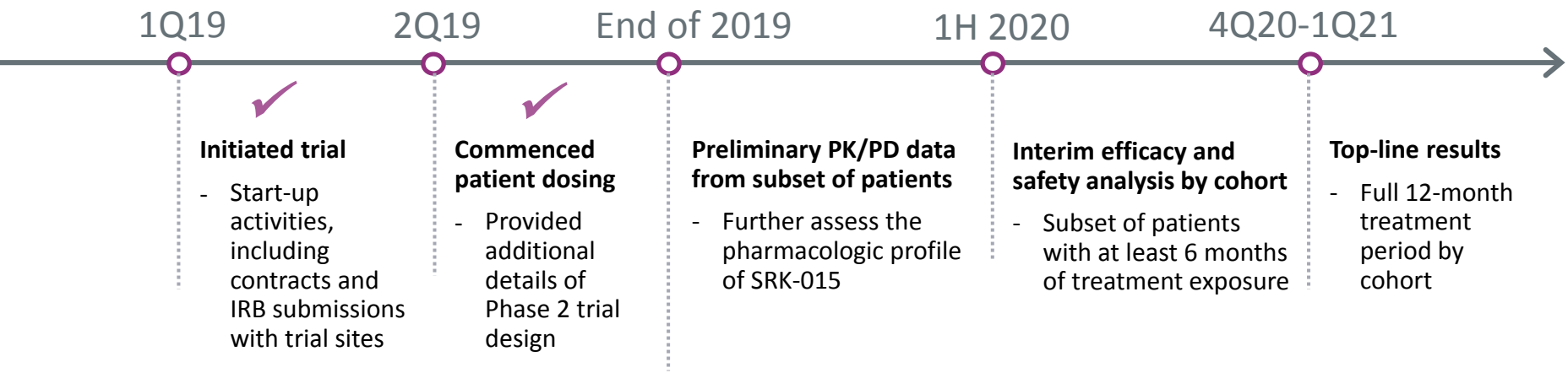
SRK-015 Phase 2 Trial Design

TOPAZ

	Cohort 1	Cohort 2	Cohort 3
Design	• N= 20; ages 5-21 • Open-label, single-arm • 20 mg/kg SRK-015 IV Q4W • 12-month treatment period	• N= 15; ages 5-21 • Open-label, single-arm • 20 mg/kg SRK-015 IV Q4W • 12-month treatment period	• N= 20; ages ≥2 • Double-blind, randomized (1:1) to 2 mg/kg or 20 mg/kg SRK-015 IV Q4W • 12-month treatment period
Subjects	• Ambulatory Type 3 SMA	• Type 2 or non-ambulatory Type 3 SMA • Receiving treatment with approved SMN upregulator	• Type 2 SMA • Initiated treatment with approved SMN upregulator before age 5
Primary Objectives	• Safety • Mean change from baseline in RHS	• Safety • Mean change from baseline in HFMSE	• Safety • Mean change from baseline in HFMSE

HFMSE – Hammersmith Functional Motor Scale Expanded; RHS – Revised Hammersmith Scale

SRK-015: Path to Top-Line Results in SMA



SRK-015 has the potential to be the first muscle-directed therapy for patients with SMA

TGFβ1: Significant Opportunities in Oncology/Immuno-oncology and Fibrosis

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TGFβ1 Plays Central Role in Multiple Diseases with Unmet Need

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Oncology

- Immuno-Oncology
- Tumor-Directed Therapy
- Myeloproliferative Disorders

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GILEAD

Fibrosis

Upfront cash and equity investment:
$80 million*

One-time preclinical milestone:
$25 million

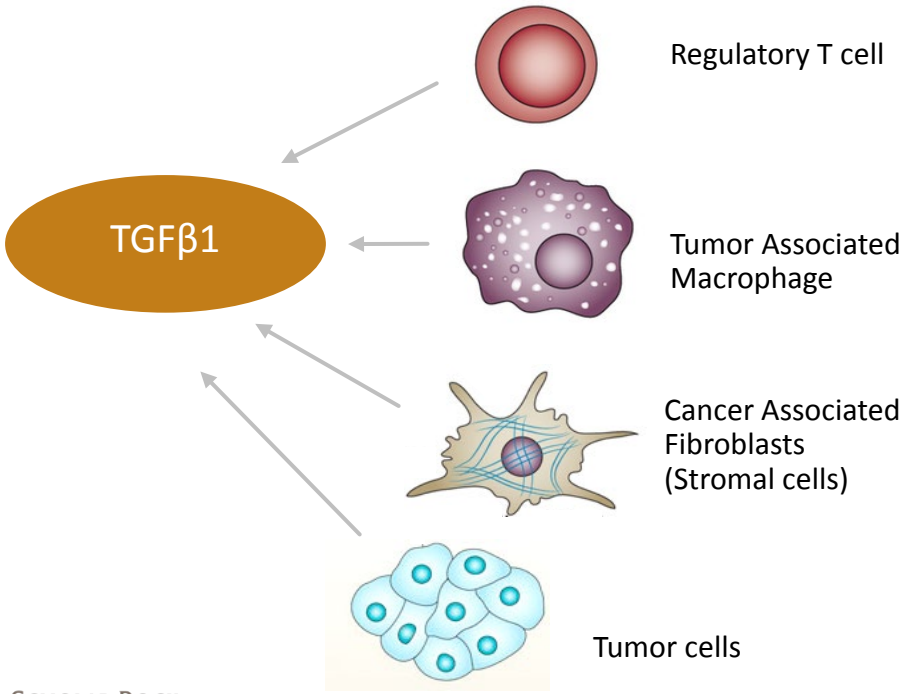
Additional milestones across 3 programs:
Up to $1,425 million

High single- to low double-digit tiered royalties on net sales

Includes $30 million purchase of Scholar Rock common stock at price per share of $30.60

Inhibition of TGFβ1: Multipronged Approach for Immuno-Oncology

TGFβ1 is a key driver of immune system evasion by cancer cells



Nature (on-line), Feb. 14, 2018

doi:10.1038/nature25501

TGFβ attenuates tumour response to PD-L1 blockade by contributing to exclusion of T cells

Sanjeev Mariathasan[1]*, Shannon J. Turley[1]*, Dorothee Nickles[1]*, Alessandra Castiglioni[1], Kobe Yuen[1], Yulei Wang[1], Edward E. Kadel III[1], Hartmut Koeppen[1], Jillian L. Astarita[1], Rafael Cubas[1], Suchit Jhunjhunwala[1], Romain Banchereau[1], Yagai Yang[1], Yinghui Guan[1], Cecile Chalouni[1], James Ziai[1], Yasin Şenbabaoğlu[1], Stephen Santoro[1], Daniel Sheinson[1], Jeffrey Hung[1], Jennifer M. Giltnane[1], Andrew A. Pierce[1], Kathryn Mesh[1], Steve Lianoglou[1], Johannes Riegler[1], Richard A. D. Carano[1], Pontus Eriksson[2], Mattias Höglund[2], Loan Somarriba[3], Daniel L. Halligan[3], Michiel S. van der Heijden[4], Yohann Loriot[5], Jonathan E. Rosenberg[6], Lawrence Fong[7], Ira Mellman[1], Daniel S. Chen[1], Marjorie Green[1], Christina Derleth[1], Gregg D. Fine[1], Priti S. Hegde[1], Richard Bourgon[1] & Thomas Powles[8]

- Pathway analysis points to TGFβ1 as major determinant of resistance to anti-PDL1 (atezolizumab)
- TGFβ1 creates 'immune excluded' tumor microenvironment
- Anti-TGFβ antibody enhances anti-PDL1 treatment response in syngeneic EMT6 tumor model

Renewed Industry Interest in Potential Role of TGFβ Inhibition in Immuno-Oncology

Feb. 5, 2019

"GSK and Merck KGaA, Darmstadt, Germany announce global alliance to jointly develop and commercialise M7824, a novel immunotherapy with potential in multiple difficult-to-treat cancers"

June 10, 2019

"Merck to Acquire Tilos Therapeutics: Merck Gains Portfolio of Investigational Antibodies Modulating TGFβ"

Differentiated approach with SRK-181:

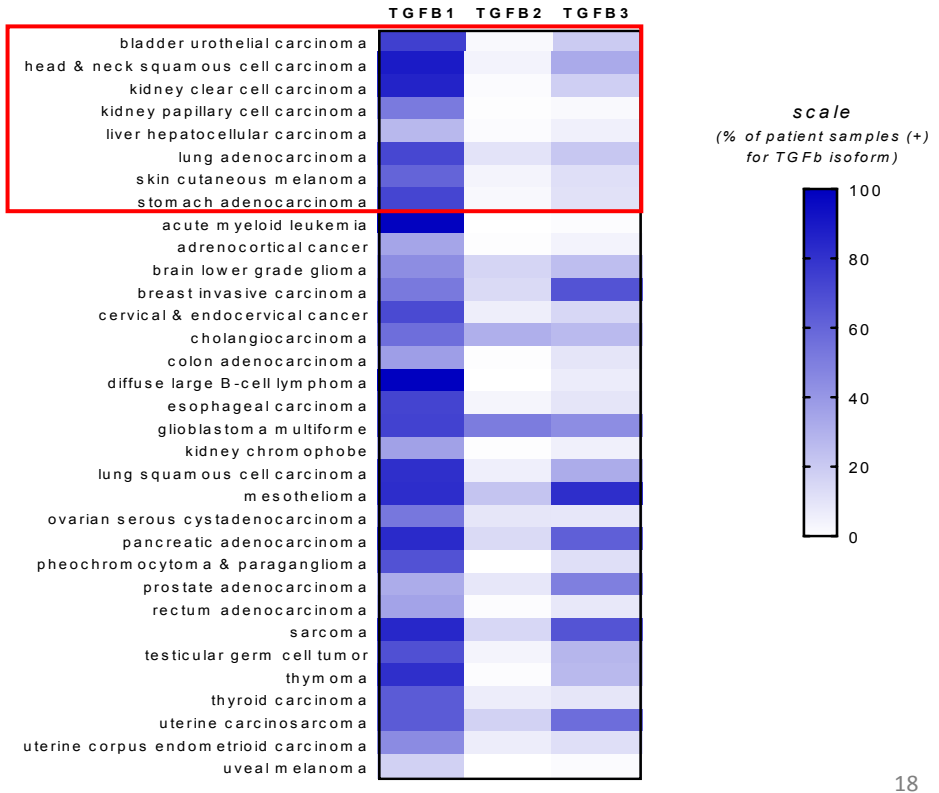
- ***Fully human monoclonal antibody (mAb)***
- ***Highly selective inhibitor of the activation of TGFβ1 precursor (latent form)***
- ***Minimal or no binding to latent TGFβ2 and TGFβ3 isoforms***
- ***In preclinical models:***
  - ***TGFβ1-specific inhibition by SRK-181-mIgG1* rendered both TGFβ1- and TGFβ1/TGFβ3 co-expressing tumor models sensitive to anti-PD1***
  - ***Combination of SRK-181-mIgG1 and anti-PD1 led to tumor regression and survival benefit***
  - ***Improved toxicity profile; avoided cardio tox associated with less selective approaches such as pan-TGFβ antibody and ALK5 inhibitor***

*SRK-181-mIgG1 is the murine version of SRK-181

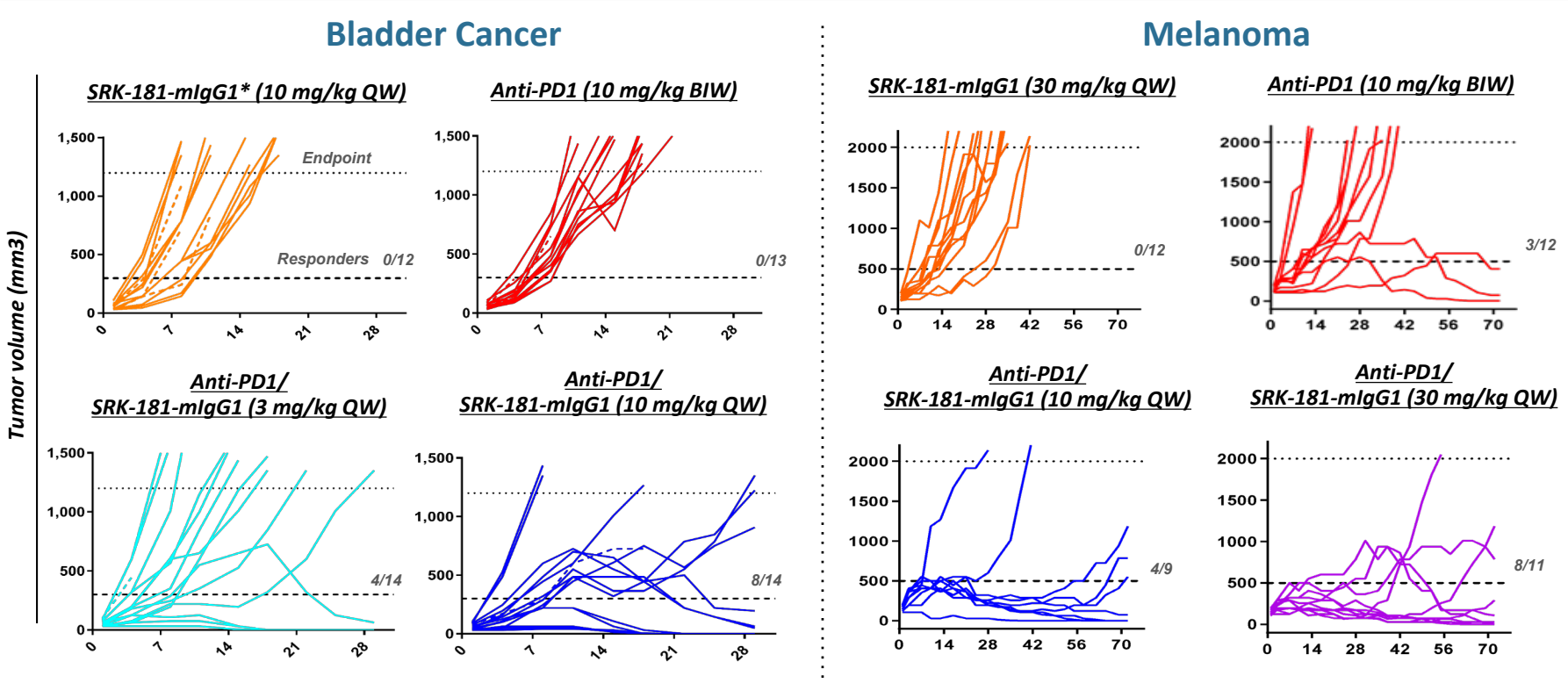
TGFβ1 is the Predominant Isoform in Most Human Tumors

The Cancer Genome Atlas RNAseq analysis: >10,000 samples spanning 33 tumor types

- TGFβ1 prevalent in human cancers for which checkpoint therapies are approved
- Expression data for most tumor types suggest that TGFβ signaling mainly driven by TGFβ1

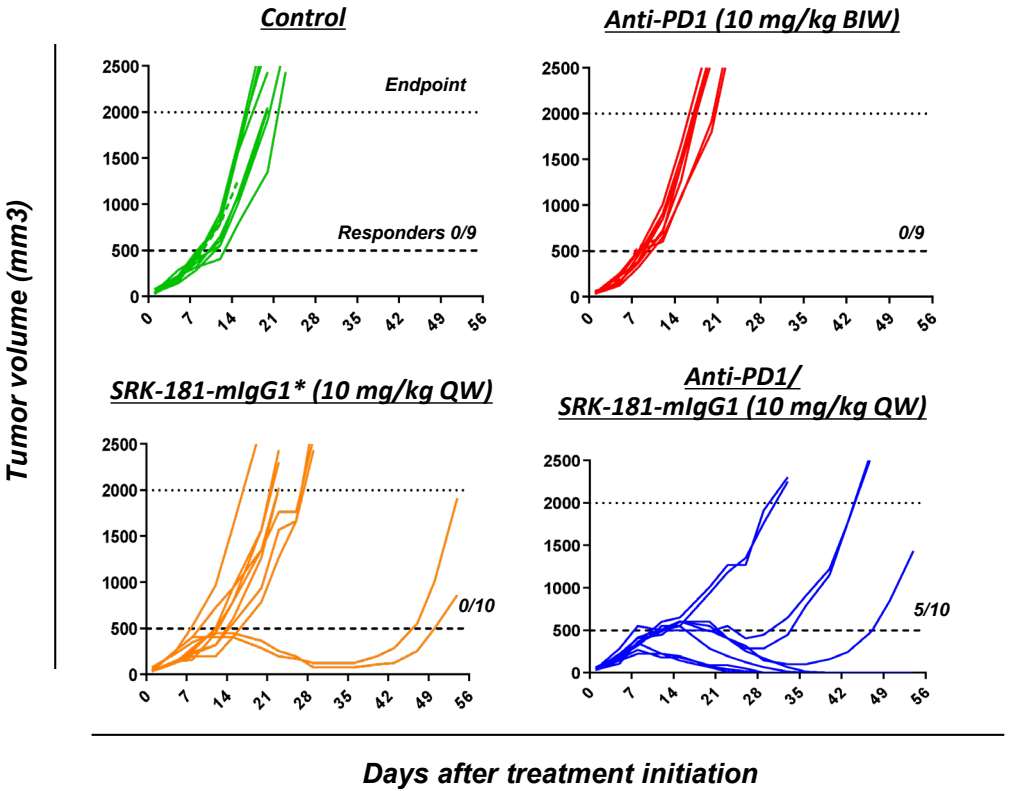


TGFβ1 Blockade with SRK-181-mIgG1 Rendered Preclinical Tumor Models Susceptible to Anti-PD1 Therapy



Preclinical data shown above as presented at American Association of Cancer Research (AACR) Annual Meeting (April 2019)
*SRK-181-mIgG1 is the murine version of SRK-181; Responder defined as tumor size <25% endpoint volume at study end

Inhibiting TGFβ1 Alone Was Sufficient to Sensitize Preclinical TGFβ1/3-Expressing Breast Cancer Model

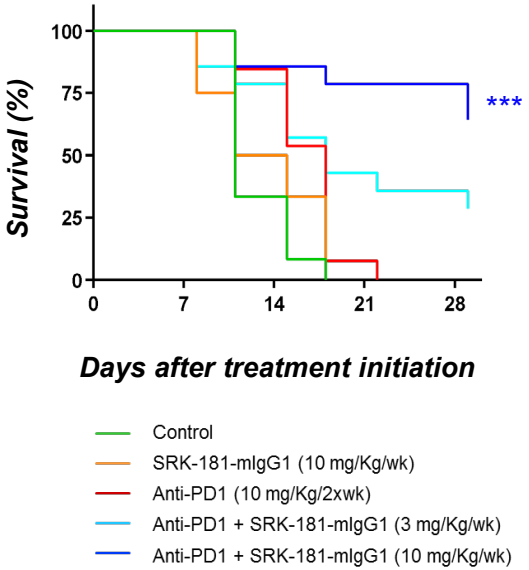


- EMT6 model expresses both TGFβ1 and TGFβ3
- Model is poorly responsive to PD1 blockade as a monotherapy
- Combination of SRK-181-mIgG1 and anti-PD1 resulted in tumor regression or tumor control

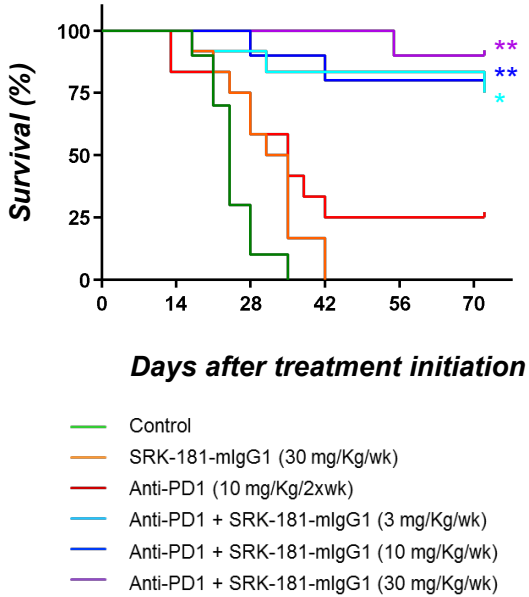
Preclinical data shown above as presented at American Association of Cancer Research (AACR) Annual Meeting (April 2019)
*SRK-181-mIgG1 is the murine version of SRK-181

SRK-181-mIgG1† Combined with Anti-PD1 Therapy Led to Significant Survival Benefit in Preclinical Tumor Models

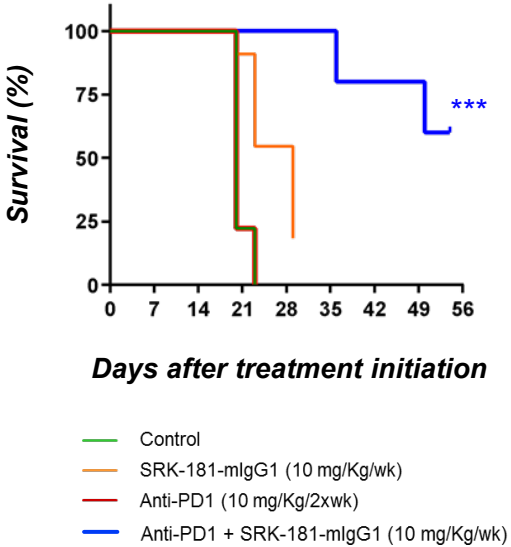
MBT-2 Bladder Cancer Model



Cloudman S91 Melanoma Model



EMT6 Breast Cancer Model

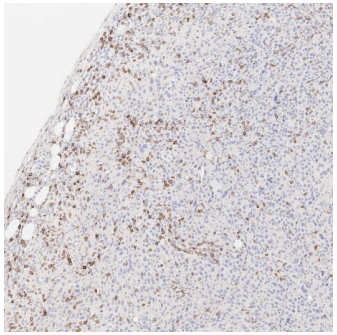


* P<0.05 Log-rank (Mantel-Cox test) vs. anti-PD1 **P<0.01 *** P<0.001

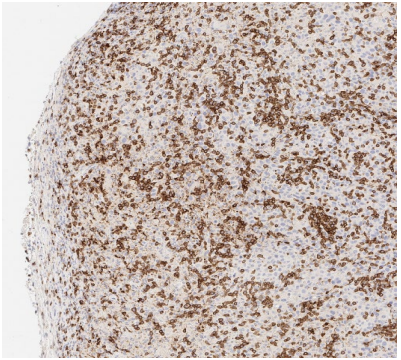
Preclinical data shown above as presented at American Association of Cancer Research (AACR) Annual Meeting (April 2019)
†SRK-181-mIgG1 is the murine version of SRK-181

SRK-181-mIgG1 Combination Therapy Enabled Infiltration and Expansion of CD8+ T cells in Preclinical Bladder Cancer Model

Anti-PD1

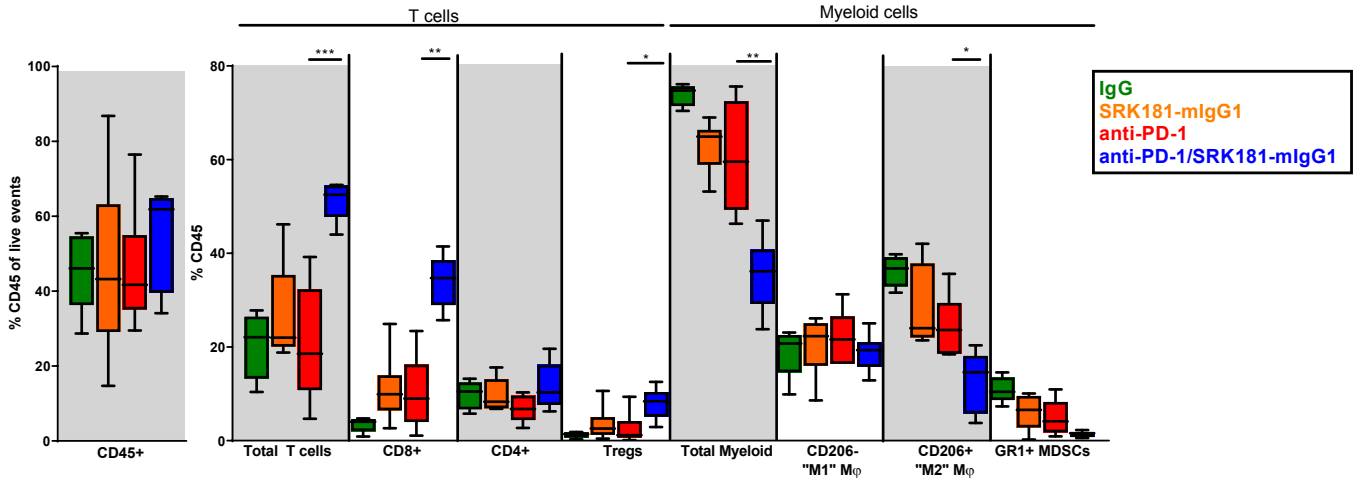


Anti-PD1/SRK-181-mIgG1 (10 mg/kg) led to increase in CD8+ cells



Combination treatment with anti-PD1/SRK-181-mIgG1 led to:

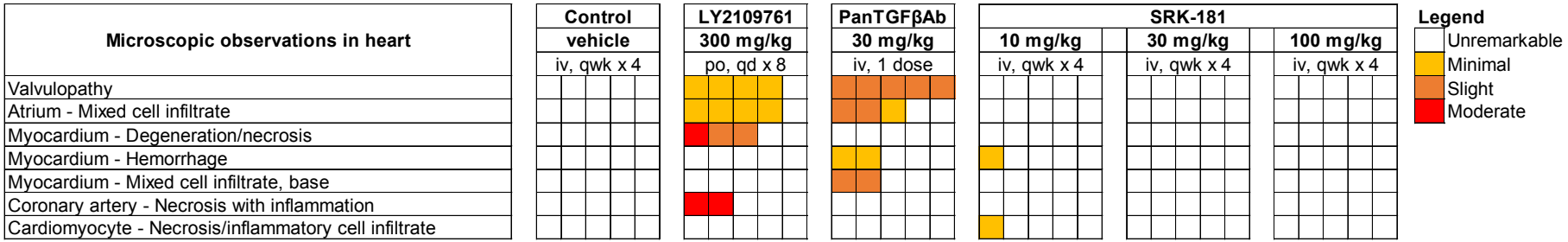
- Significant increase in effector T cells ($p<0.05$)
  - Expansion of CD8+ population to an average of 34% of the tumor's immune cells from a control average of 3.5%
- Significant decrease in intratumoral immunosuppressive myeloid cells ($p<0.05$)
  - Reduction in TAM/MDSC population to 14% of the tumor's immune cells from a control average of 47%



Preclinical data shown above as presented at American Association of Cancer Research (AACR) Annual Meeting (April 2019)
*SRK-181-mIgG1 is the murine version of SRK-181; Anti-PD1 dosed at 10 mg/kg twice weekly and SRK-181-mIgG1 dosed at 10 mg/kg weekly

TGFβ1 Isoform Specificity of SRK-181 Improved Preclinical Toxicity Profile

Repeat dose pilot toxicology study in adult female Sprague Dawley rats



- Animals dosed with pan-TGFβ inhibitors, LY2109761 (inhibitor of ALK5, common TGFβ receptor kinase) or pan-TGFβ antibody, exhibited expected cardiac findings based on published data
- Exposure as assessed by SRK-181 serum concentration reached 2,300 µg/ml following 4 weekly doses of 100 mg/kg
- No SRK-181 related adverse effects were noted up to 100 mg/kg per week
- No cardiotoxicities (valvulopathy) were noted with SRK-181
- No observed adverse effect level (NOAEL) for SRK-181 was the highest dose evaluated (100 mg/kg QW)

Preclinical data shown above as presented at American Association of Cancer Research (AACR) Annual Meeting (April 2019)

SRK-181: Advancing Development for Treatment of Cancers Resistant to Checkpoint Blockade Therapies (CBTs)

SRK-181 is a fully human antibody designed to bind to, and prevent the activation of, latent TGFβ1 with high affinity and high selectivity

TGFβ signaling	Implicated as a culprit in primary resistance to CBTs
Translational data analyses	TGFβ1 expression is prominent in many human tumor types for which CBTs is approved or showed clinical activity
Clinical correlation and preclinical model data	TGFβ1 excludes effector cell entry into the tumor and limits immune system access to tumor cells
Preclinical studies in syngeneic mouse tumor models resistant to CBT	Combination of SRK-181-mIgG1* with anti-PD1 led to tumor regression/control and significant survival benefit, including in models that express both TGFβ1 and TGFβ3
28-day pilot toxicology study in adult rats	SRK-181 showed no observed drug-related toxicity up to a weekly dose of 100 mg/kg for 4 weeks

*SRK-181-mIgG1 is the murine version of SRK-181

RGMc Program: Targeting the BMP6 Signaling Pathway

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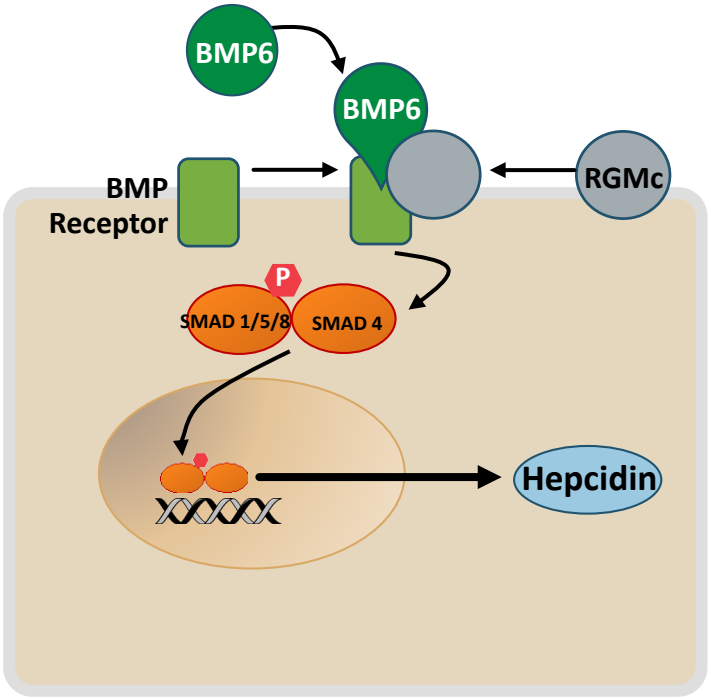
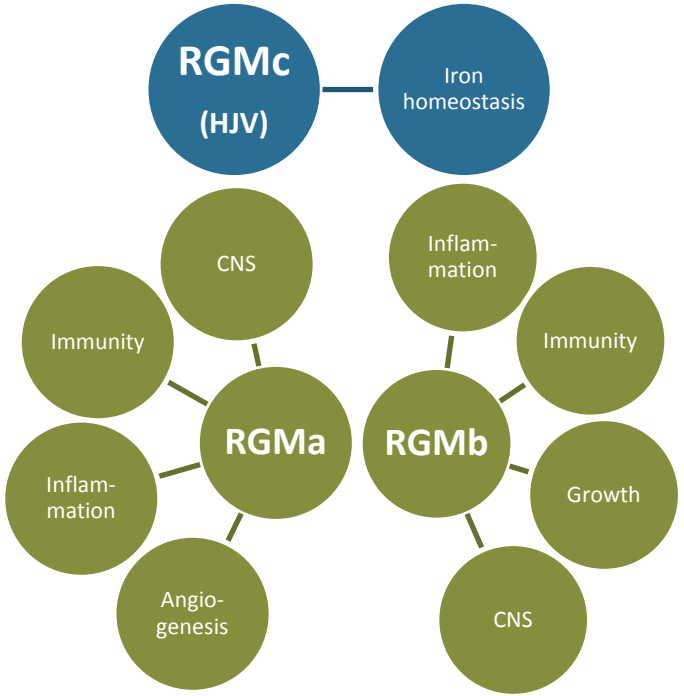
Anti-RGMc Therapy: Rational Solution that Directly Addresses the Underlying Pathobiology of Iron-Restricted Anemias

Genetically validated pathway of iron regulation in humans



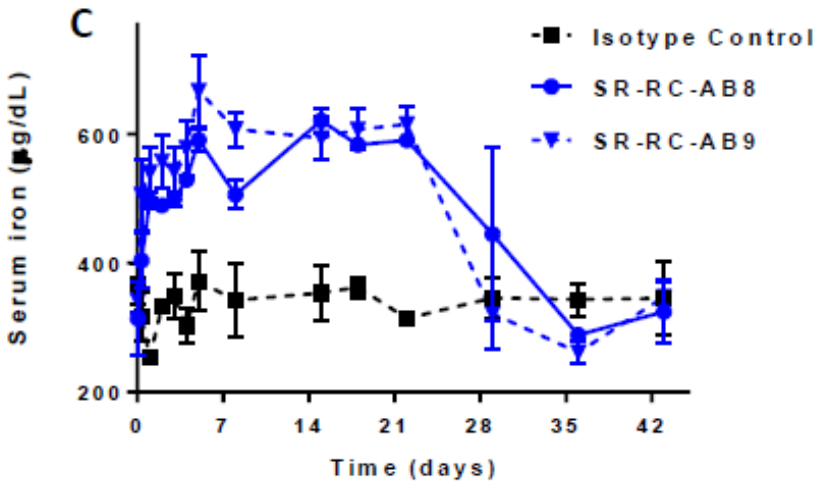
RGMa and RGMb play many different physiological roles



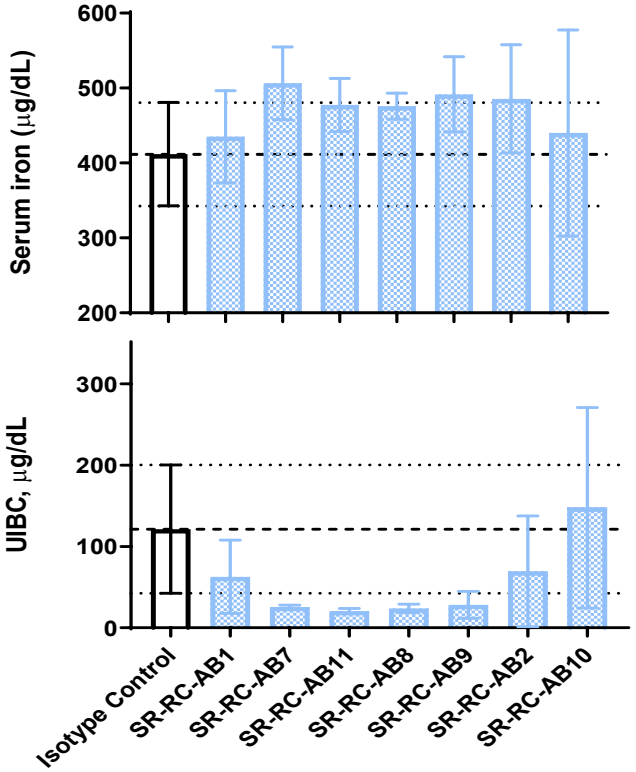
Adapted from Crielaard et al, Nature Reviews , 2017

Scholar Rock's RGMc-Selective Antibodies Show Potent Increase in Serum Iron In Vivo

Antibodies showed up to 3 weeks prolonged increase in serum iron in SD rats
(single 20 mpk dose)



Antibodies resulted in increases in serum iron and decreases in UIBC



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SRK-015

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  - Neuromuscular disorders
  - Other myostatin-related disorders

TGFβ1 Inhibitor

- ○ Advance cancer immunotherapy product candidate, SRK-181, into a Phase 1 trial mid-2020
- ○ Announce initial data from Phase 1 trial of SRK-181 in patients with solid tumors by end of 2021
- ○ Continue to advance active discovery programs for context-dependent inhibition of TGFβ1
- ○ Conduct fibrosis discovery and preclinical studies in partnership with Gilead

RGMc

- ○ Nominate product candidate in 1H20

Building Value in All Dimensions

Building on Strong Financial Foundation

Advancing Clinical Development

Executing Strategic Collaboration

Growing Innovative Pipeline

Appendix

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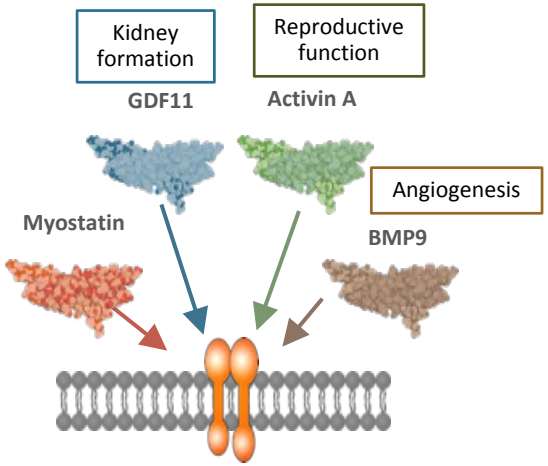
SRK-015: Aligning Therapeutic Approach with Myostatin Biology

Scholar Rock's Guiding Principles for Neuromuscular Indication Selection	Key Characteristics of Spinal Muscular Atrophy (SMA)
Younger population	Genetic disorder with onset in childhood
At least partially intact innervation and no structural muscle abnormalities	Partial neural connectivity and atrophied muscles that largely retain structural integrity
Need for increase in fast-twitch muscle fibers	Substantial deficit in fast-twitch fibers
Clinical trial endpoint driven by fast-twitch fiber function	Fast-twitch fiber function has a prominent role in SMA outcome measures

Traditional Approaches Can Raise Significant Safety Concerns

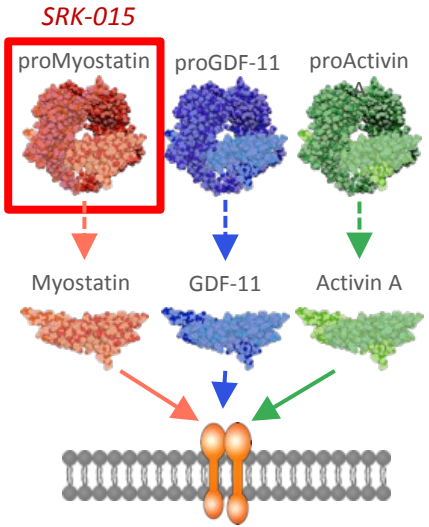
Traditional Approaches Lack Selectivity

- Most inhibitors of active myostatin also inhibit GDF11 and may inhibit other growth factors as well
- Antibodies to ActRIIb and ligand trap approaches inhibit signaling of multiple ligands

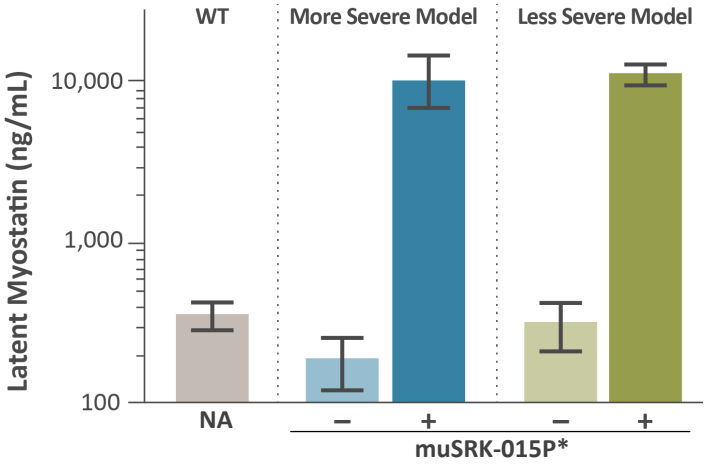


Scholar Rock Approach

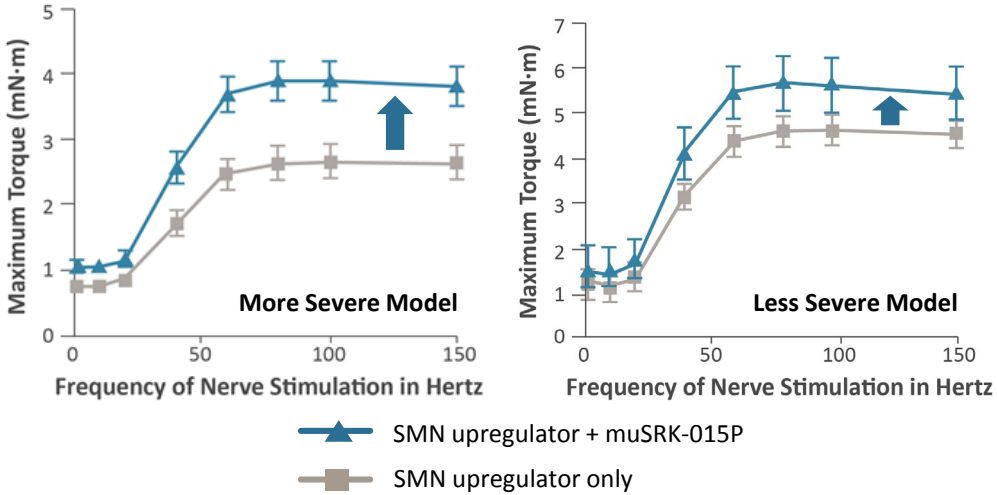
Exquisite Selectivity By Targeting Precursor Form of Myostatin



Review of Preclinical Data in SMNΔ7 Mouse Models



- **Achieved multi-fold increase in serum latent myostatin levels indicating target engagement**
- **Confirms presence of target in disease setting**
- **Lower latent myostatin levels in the SMA group may be attributable to reduced overall muscle mass**



Treatment improved muscle mass and strength

- Maximal torque of the plantar flexor muscle group increased:
  - More severe model: 44%-51%
  - Less severe model: 20%-30%

*muSRK-015P is the parental clone of SRK-015 on a mouse IgG1 framework

Preclinical data published Nov 2018: Long, K., O'Shea, K., Khairallah, R., et al. Specific Inhibition of Myostatin Activation is Beneficial in Mouse Models of SMA Therapy. *Human Molecular Genetics,* ddy382

SRK-015 Phase 1 Trial Design

KEY OBJECTIVES OF PHASE 1

Evaluate the safety and tolerability, pharmacokinetics, and pharmacodynamics of SRK-015 IV

	SINGLE-ASCENDING DOSE (SAD)	MULTIPLE-ASCENDING DOSE (MAD)
Design	Double-blind, placebo-controlled 3:1 randomization	Double-blind, placebo-controlled 3:1 randomization
Subjects	40 Adult healthy volunteers (Ages 18-55)	26 Adult healthy volunteers (Ages 18-55)
Dosing	Single doses at: 1, 3, 10, 20, or 30 mg/kg	Q2W dosing for 3 doses at: 10, 20, or 30 mg/kg

SRK-015 Well Tolerated in Phase 1 Healthy Volunteer Trial

- **Well-tolerated with no apparent safety signals**
- **No dose-limiting toxicities identified up to highest evaluated dose of 30 mg/kg**
  - No discontinuations due to treatment-related adverse events (AEs)
  - No treatment-related SAE
  - No hypersensitivity reactions
- **Anti-drug antibody tests were negative in all SRK-015 treated subjects**
- **SAD cohort: AEs* were observed in 30% (9/30) SRK-015- vs. 50% (5/10) placebo-treated subjects**
  - Most frequently reported AE: headache
- **MAD cohort: AEs observed in 35% (7/20) SRK-015- vs. 67% (4/6) placebo-treated subjects**
  - Most frequently reported AE: postural dizziness
- **Single reported SAE of gallstone-induced pancreatitis**
  - Assessed by trial investigator as unrelated to SRK-015 treatment

*Term "adverse event" noted in this presentation refers to a treatment-emergent adverse event, which is defined as an AE with onset after administration of study drug through the final follow-up visit, or in the event that onset time precedes study drug administration, the AE increases in severity during the post-dosing follow-up period

Phase 1 Pharmacokinetic (PK) Data Support Infrequent Dosing

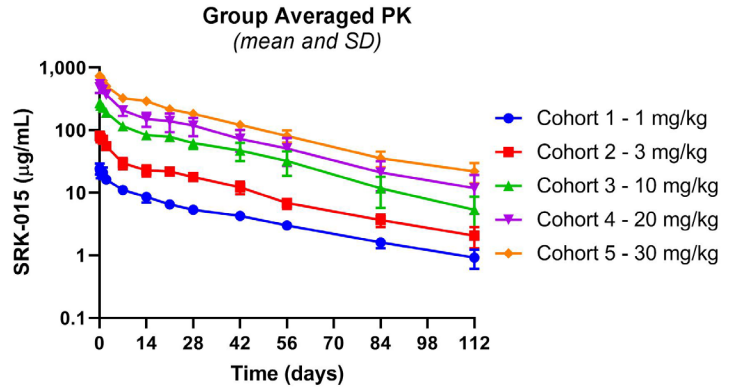
SRK-015 Displayed Well-Behaved, Linear PK Profile

- Minimal variability observed, consistent with that commonly observed with monoclonal antibodies
- Dose-proportional serum drug exposure

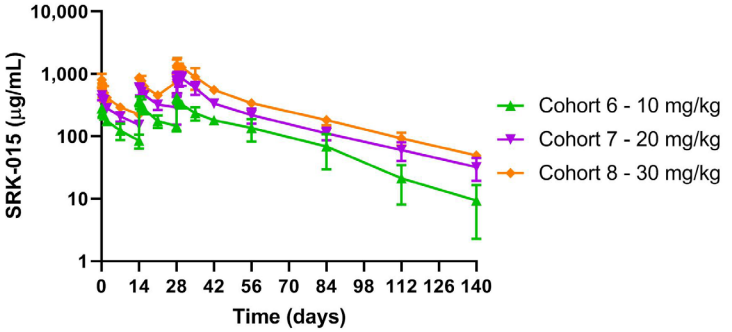
Half-Life Supports Infrequent Dosing

- Serum half-life of 23-33 days across SRK-015 dose groups
- Supports planned evaluation of once every 4-week (Q4W) dosing in Phase 2

Single-Ascending Dose



Multiple-Ascending Dose



Phase 1 Pharmacodynamic (PD) Data Demonstrate Robust and Sustained Target Engagement

Robust Target Engagement Observed

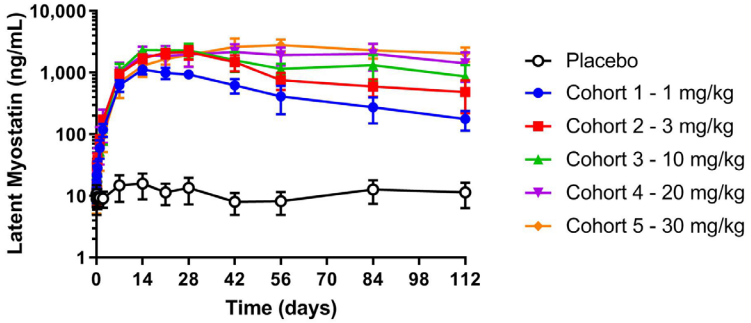
- Single dose of SRK-015 led to marked increases in serum concentrations of latent myostatin
- No meaningful change observed with placebo

Evidence Supports Durable Target Saturation

- Peak latent myostatin levels plateaued starting with a single dose at 3 mg/kg suggesting target saturation
  - Single dose at 1 mg/kg only attained approx. half of peak level
- Plateau was sustained demonstrating durability of effect:
  - Up to Day 84 after single dose at 20 mg/kg
  - Up to at least Day 140 after multiple doses at 20 and 30 mg/kg

First proof-of-mechanism in humans of Scholar Rock's therapeutic approach targeting the latent form of growth factors

Single-Ascending Dose



Multiple-Ascending Dose