SpringWorks THERAPEUTICS

Corporate Presentation

December 2019

NASDAQ: SWTX

Forward-Looking Statements

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SpringWorks Therapeutics is a Clinical-Stage Targeted Oncology Company

- **Two late-stage rare oncology programs** commenced potentially registrational trials in 2019, each supported by strong clinical data
- **Three programs addressing large opportunities in genetically defined cancers** in collaboration with industry leaders
- Leveraging **strong development capabilities** and **shared-value partnerships** to enhance portfolio value and become the partner of choice for industry leaders
- Led by an **experienced management team** with deep expertise in drug development and commercialization
- Well-capitalized to execute **important value-driving milestones** across both standalone and partnered programs

Our ambition is to ignite the power of promising science to unleash new possibilities for patients

Demonstrated Leadership Advancing Transformative Therapies

Leadership Team

Saqib Islam, J.D.
Chief Executive Officer

moderna messenger therapeutics
ALEXION

Jens Renstrup, M.D., MBA
Chief Medical Officer

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Chief Business Officer

OrbiMed Healthcare Fund Management
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Frank Perier, MBA
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Mary Smith, Ph.D.
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Bamboo therapeutics
United Therapeutics CORPORATION

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Chief Executive Officer

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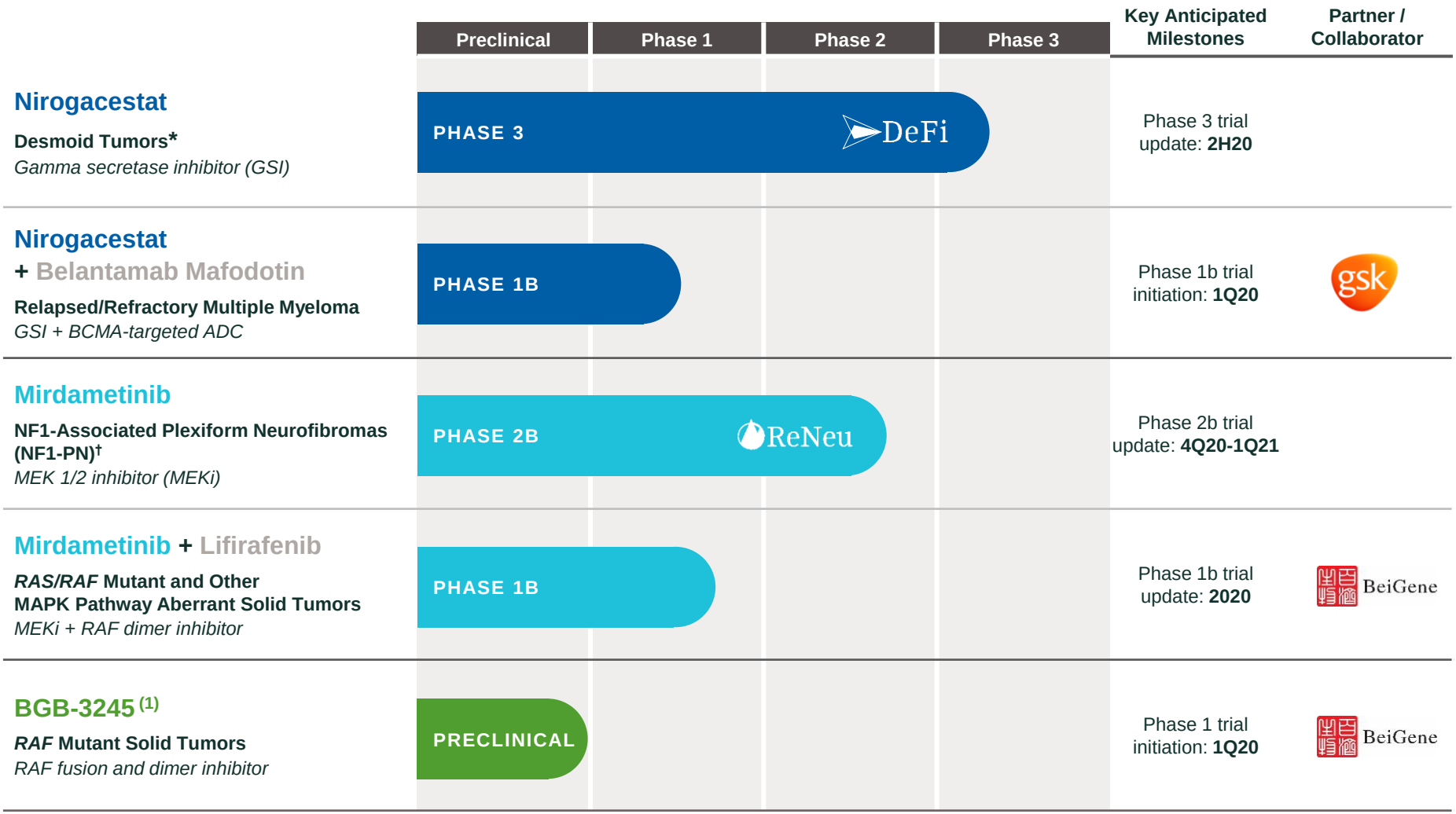
Executive Vice President

Jeffrey Schwartz
Bain Capital Life Sciences,
Managing Director

Stephen Squinto, Ph.D.
OrbiMed,
Executive Partner

Advancing Diversified Pipeline of Targeted Oncology Programs as Standalone and Combination Therapies



Collaborator Asset

Note: Nirogacestat = PF-03084014 and Mirdametinib = PD-0325901 (both in-licensed from Pfizer).
* Received Orphan Drug, Fast Track and Breakthrough Therapy Designations.
† Received Orphan Drug and Fast Track Designations.
(1) Being developed by MapKure, LLC, jointly owned by SpringWorks and BeiGene.

Two Potentially Registrational Trials Ongoing in Rare Oncology Indications

Nirogacestat

Gamma Secretase Inhibitor

Desmoid Tumors

Mirdametinib

MEK Inhibitor

NF1-Associated Plexiform Neurofibromas

	Nirogacestat	Mirdametinib
Disease Manifestation	▪ Highly morbid disease that can cause severe pain, disfigurement, and incapacitating loss of physical function	▪ Aggressive tumor growth that can lead to severe pain and functional impairment, most often manifesting in children
Current Treatments	▪ Currently no FDA approved therapies ▪ Off-label treatment options provide inconsistent clinical benefit	▪ Currently no FDA approved therapies ▪ Opportunity for differentiated profile vs. other MEK inhibitors
Existing Data	▪ Generally well tolerated in over 200 subjects ▪ Clinical activity observed in desmoid tumor patients (most of whom were heavily pre-treated) in Ph1 and Ph2 trials	▪ Clinical activity and tolerability observed in Ph2 trial in adolescents and adults with NF1-PN
Regulatory Designations	▪ Received FDA Fast Track and Breakthrough Therapy Designations and Orphan Drug Designation from both FDA and European Commission	▪ Received Fast Track Designation from FDA and Orphan Drug Designation from both FDA and European Commission
Ongoing Trial	▪ Progression-free survival is primary endpoint in ongoing Ph3 trial (DeFi) and is supported by precedent data	▪ Potentially registrational Ph2b trial (ReNeu) is enrolling NF1-PN patients of all ages (pediatrics, adolescents and adults)

Potential to become the first approved therapy in an orphan oncology indication with substantial morbidity

Potentially best-in-class program for large orphan oncology indication with no approved treatments

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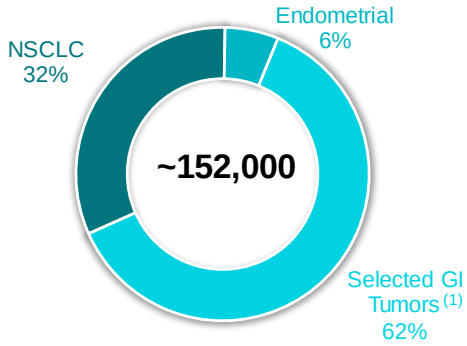
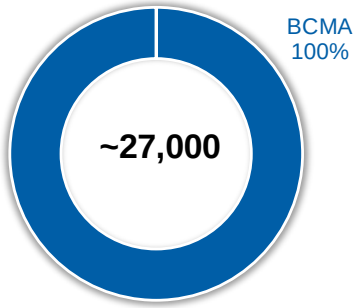
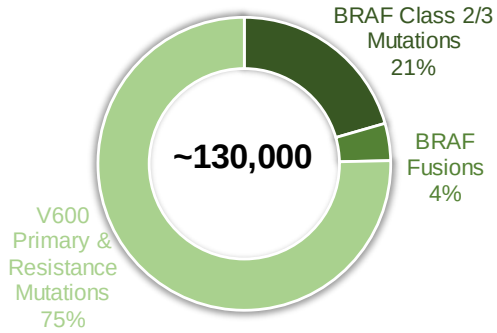
Three Programs Addressing Large, Genetically Defined Cancers in Collaboration with Industry Leaders

	Mirdametinib + Lifirafenib			Nirogacestat + Belantamab Mafodotin	BGB-3245		
	Non-Small Cell Lung	Endometrial	Selected GI Tumors (1)	Refractory/Relapsed Multiple Myeloma	Solid Tumors with BRAF Mutations/Fusions (Tissue Agnostic)		
Partner / Collaborator	BeiGene			gsk	BeiGene (2)		
Biomarker	KRAS & NRAS			BCMA	BRAF Class 2/3 Mutations	BRAF Fusions	V600 Primary & Resistance Mutations
Total Annual U.S. Incident Population	~152,000			~27,000	~130,000		







Expecting to have three programs in large cancer indications in the clinic by 1Q20, each supported by strong preclinical activity in the selected biomarker settings

(1) Includes colorectal, biliary tract and pancreatic tumors.
(2) Program being developed by MapKure, LLC, jointly owned by SpringWorks and BeiGene.

Nirogacestat

Desmoid Tumors

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Nirogacestat: A New Paradigm for Patients With Desmoid Tumors

- Nirogacestat is an oral, selective gamma secretase inhibitor with > 9 years of clinical experience (over 200 subjects exposed)
- Clinical activity observed in Phase 1 and Phase 2 trials in desmoid tumors independent of prior lines of therapy and underlying mutation
- Generally well tolerated compound suitable for long term dosing
- Received FDA Fast Track and Breakthrough Therapy Designations and Orphan Drug Designation from both FDA and European Commission
- Phase 3 DeFi trial currently enrolling desmoid tumor patients and FDA has agreed to progression-free survival (PFS) serving as primary endpoint

Update to be provided on Phase 3 DeFi trial in 2H20

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Desmoid Tumors are Highly Morbid Soft Tissue Tumors that are Poorly Responsive to Surgical Interventions and Off-Label Therapies

Painful, disfiguring, and disabling condition

- French Desmoid Advocacy Group Survey (n=102):
  - **Presence of pain in 63%** of patients
  - **Permanent pain in 38%** of patients with pain
- Memorial Sloan Kettering/Quintiles PRO tool development patient interviews (n=31):
  - **Disfigurement in 81%** of patients
  - **Restricted range of motion in 68%** of patients

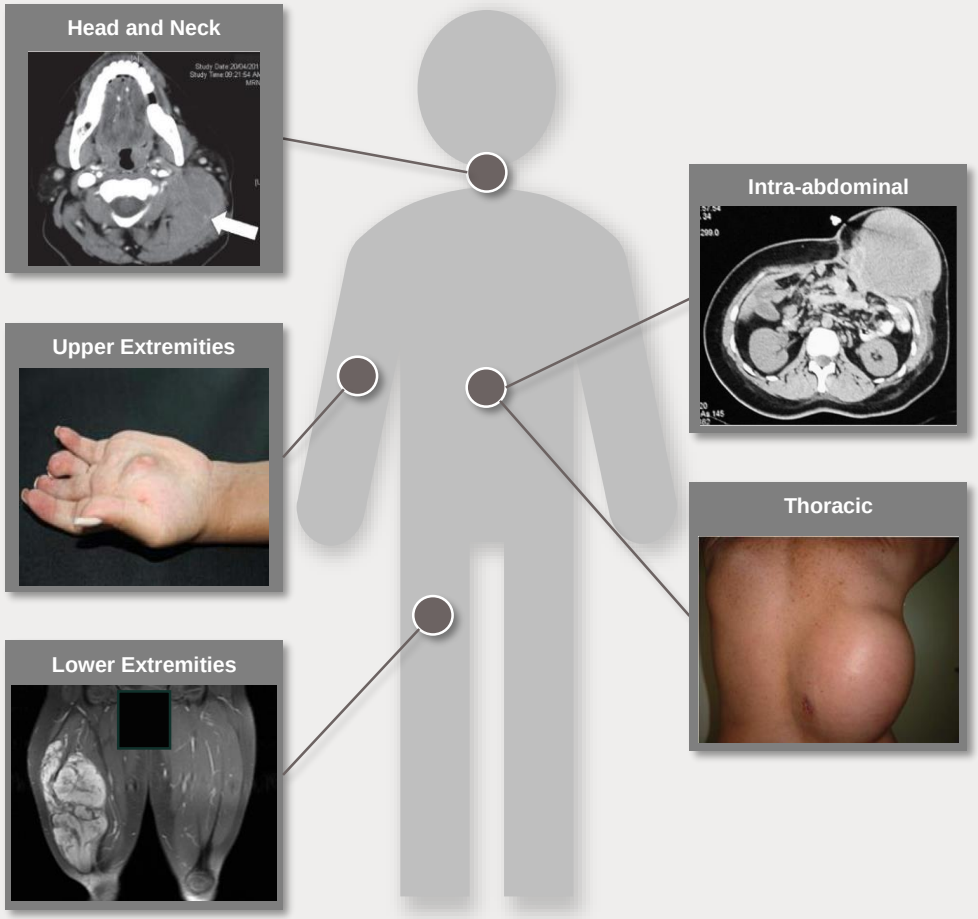
No currently approved therapies

- **Recurrence post-surgical resection of up to 70%**
- Off-label systemic therapies (TKIs, chemotherapeutics) associated with a **challenging AE profile and inconsistent efficacy**
- Physicians often **adopt a watchful waiting approach** given post-surgical recurrence rates and inconsistent benefit from available off-label systemic therapies

~1,000-1,500 newly incident patients per year in US

- Young patient population, with tumors more commonly diagnosed in the **third and fourth decades of life**
- **Estimated 5,500-7,000 patients actively receiving treatment in the US in any given year**

CLINICAL PRESENTATION OF DESMOID TUMORS



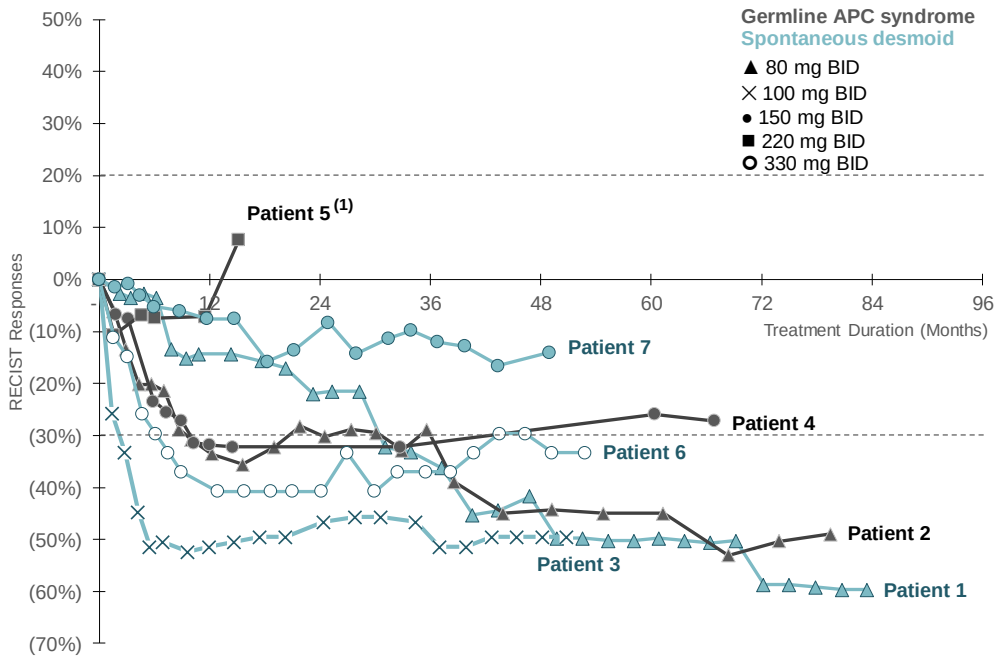
Note: PRO = Patient-reported outcomes

Source: Rigaux et al., *Bulletin du Cancer*, 2015; Penel et al., *European Journal of Cancer*, 2017; Tsagozis et al., *Annals of Medicine & Surgery*, 2017; SpringWorks market research.

Initial Clinical Activity of Nirogacestat Observed in Desmoid Tumors

PHASE 1 | PHASE 2 | PHASE 3

Most Responses Continued Past 4 Years (RECIST v1.0)



- **Median PFS (mPFS): Not reached by publication date due to lack of tumor progression**
  - Disease Control Rate (DCR): 100%
  - Objective Response Rate (ORR): 71.4% (5/7 evaluable desmoid patients)
- **Median Duration of Treatment was 49.5 months at publication**
  - Of the 7 evaluable desmoid patients on study, none discontinued due to AEs (2)

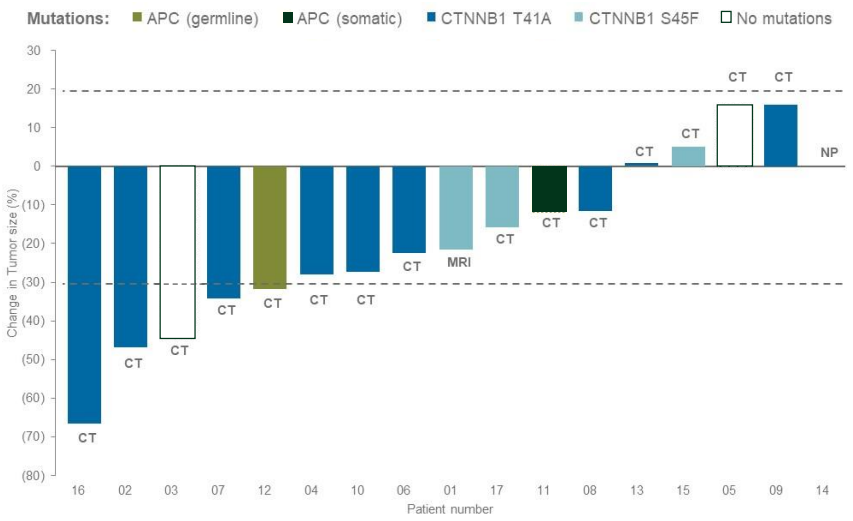
All evaluable desmoid tumor patients in the study responded to nirogacestat treatment (1)

Note: Disease control rate is percentage of patients experiencing objective response or stable disease on therapy as measured by RECIST v1.0,
Source: Villalobos, *Annals of Surgical Oncology*, 2018; Messersmith, *Clinical Cancer Research*, 2015.
(1) Per investigator "the only patient with clinical progression received PF-03084014 (220 mg BID) for 15.2 months and exhibited significant clinical improvement on therapy."
(2) Across the entire 64 patient Ph1 there were four discontinuations due to treatment-related AEs with a majority occurring during cycle 1.

Encouraging Clinical Activity and Tolerability Observed in NCI-Conducted Phase 2 Trial in a Refractory and Heavily Pre-Treated Patient Population

PHASE 1 | **PHASE 2** | PHASE 3

Clinical Responses by RECIST v1.1



Durability and Tolerability with Long-Term Dosing

- **mPFS: Not reached by publication date due to lack of progression**
  - At time of enrollment, all patients had progressing tumors
  - Patients failed a median of 4 prior lines (1-9) of systemic therapy (1)
  - ORR of 29.4% (5/17) with no Progressive Disease

- **59% of patients remained on treatment >2 years and 71% of patients stayed on drug for >1 year**
  - Median Duration of Treatment was >25 months at publication, with 6 patients continuing to receive nirogacestat as of August 2019 (treatment durations range from 50 to 60 months in these patients)
  - Well tolerated; only 1 discontinuation due to AE (grade 2 urticaria not responsive to dose reduction) (2)

Shown to arrest tumor growth in a heavily pre-treated patient population (i.e., TKIs, chemo, surgery)

Note: Per RECIST 16/17 patients were evaluable. One treatment cycle = 150 mg BID continuously for 21 days. Patient #1 had a missing baseline measurement (but had MRI). Patient #14 was not evaluable per protocol, withdrew from study after cycle 1 due to travel requirements.
Source: Kummar et al., *Journal of Clinical Oncology*, 2017.
(1) 71% had received chemotherapy, 65% NSAIDs, and 59% TKIs; 4/5 partial responses had previously failed imatinib or sorafenib.
(2) No grade 4 events, all grade 3 events related to hypophosphatemia, a known class effect easily reversible with oral supplements.

Double-Blind, Placebo-Controlled Phase 3 Trial (DeFi Trial) Has Commenced

PHASE 1 | PHASE 2 | **PHASE 3**

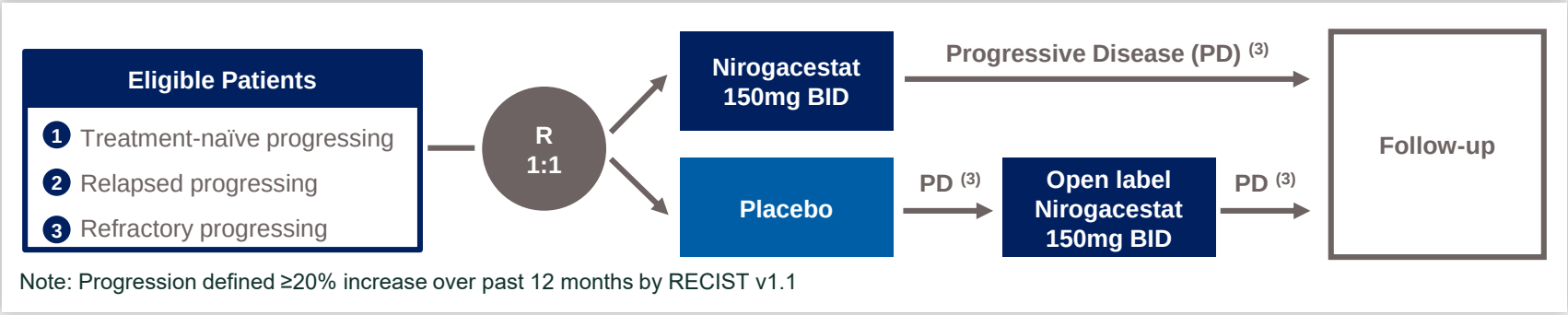
Trial Summary

- ~115 patients at ~60 sites in the US and EU
- Open label extension for patients progressing on placebo
- 90% powered to show ~12 month PFS difference between nirogacestat and placebo (1)

Summary of Endpoints

DeFi

- Primary Endpoint: Progression-free survival
  - ~50% of placebo patients expected to progress by 8 months (2)
  - Study designed to enable a potential interim analysis
- Secondary: Safety and tolerability, ORR, duration of response, volumetric tumor change (MRI), patient-reported outcomes (PRO)



Note: Progression defined ≥20% increase over past 12 months by RECIST v1.1

Key Event	Timing
Phase 3 Initiation	May 2019
Trial Update	2H20
Topline Data Readout	2Q21-3Q21

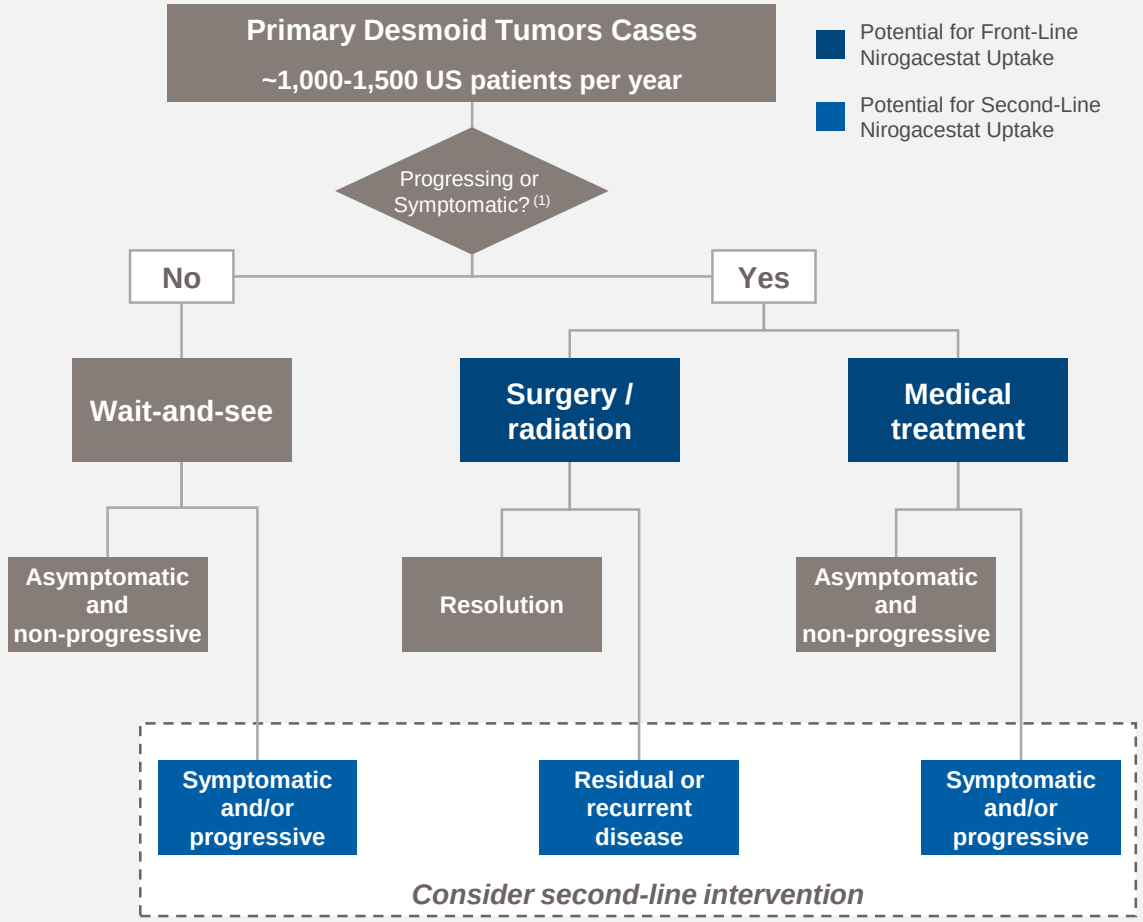
(1) A total of 51 events will provide 90% power and a 1-sided type 1 error rate of 0.025 (1-side hypothesis) to detect a difference between nirogacestat and placebo, assuming the median PFS in the nirogacestat group is 20 months and 8 months in the placebo group.
(2) Assumption based on placebo arm from sorafenib Ph3 trial presented at ASCO 2018.
(3) As defined by RECIST v1.1.

Nirogacestat Has the Potential to Become the Standard of Care Treatment for Desmoid Tumors

DESMOID TUMOR MARKET RESEARCH

- **Conducted quantitative and qualitative market research survey of 200+ physicians,** each of whom has treated at least 5 desmoid tumor patients over the preceding 5 years
- Physician feedback corroborates published findings of **substantial rates of recurrence following surgery**
- Survey suggested **~50% of patients receiving a given systemic therapy**, such as chemotherapy or a TKI, will not have a satisfactory treatment outcome and **will require subsequent treatment**
- **Up to 90% or more of desmoid tumor patients will receive at least one active intervention**, per physician feedback

DESMOID TUMOR TREATMENT ALGORITHM



We estimate 5,500 to 7,000 desmoid tumor patients are actively receiving treatment in the US per year

Note: Treatment algorithm based on published guidelines and physician interviews.
Source: SpringWorks market research.
(1) After initial wait-and-see period to determine optimal regimen given level of symptom burden and/or tumor progression.

Nirogacestat + Belantamab Mafodotin

Combination Therapy in Relapsed/Refractory Multiple Myeloma

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Advancing First-in-Class Combination of Nirogacestat with Belantamab Mafodotin in Collaboration with GlaxoSmithKline

Phase 1b Trial Initiating in 1Q20 and Supported by Strong Preclinical Data

SpringWorks THERAPEUTICS / gsk

Nirogacestat + Belantamab Mafodotin

Relapsed/Refractory Multiple Myeloma (RRMM)

GSI + BCMA-targeted ADC

~27,000 relapsed/refractory multiple myeloma patients in the US without treatment options

Belantamab mafodotin is a first-in-class BCMA ADC with demonstrated clinical activity

Strong mechanistic rationale, preclinical data, and clinical data support the combination approach

Phase 1b trial (sub-study of GSK DREAMM-5 platform trial) to begin in 1Q20 with GSK leading clinical operations and assuming all development costs

Opportunity to advance potentially best-in-class combination therapy in area of high unmet need in collaboration with industry leader

Combination Approach: Using a Gamma Secretase Inhibitor to Potentiate BCMA-Directed Therapies for the Treatment of Multiple Myeloma

BCMA has emerged as a promising target in multiple myeloma

- Universally expressed on the surface of multiple myeloma cells and clinically validated across modalities

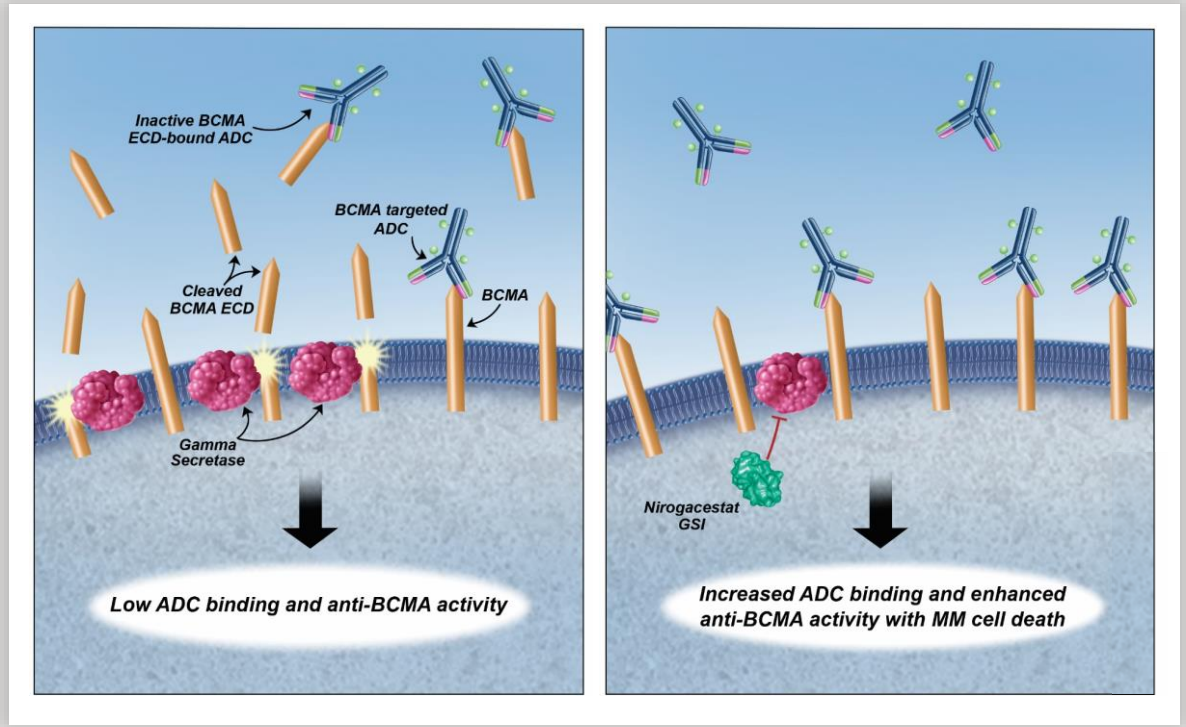
Gamma secretase directly cleaves membrane-bound BCMA

- **GSI can reduce shedding of BCMA** to improve activity of BCMA-directed therapies
- **GSI can limit soluble BCMA levels**, which act as a 'sink' for BCMA-directed therapies
- **GSI can upregulate surface BCMA expression,** including in patients that have failed prior BCMA-directed therapies

Preclinical and clinical data support combination approach

- Data reproduced with multiple BCMA agents and GSIs, including nirogacestat
- Initial clinical combo data further validate the contribution of GSI to BCMA efficacy

MECHANISM OF ACTION OF NIROGACESTAT + BELANTAMAB MAFODOTIN

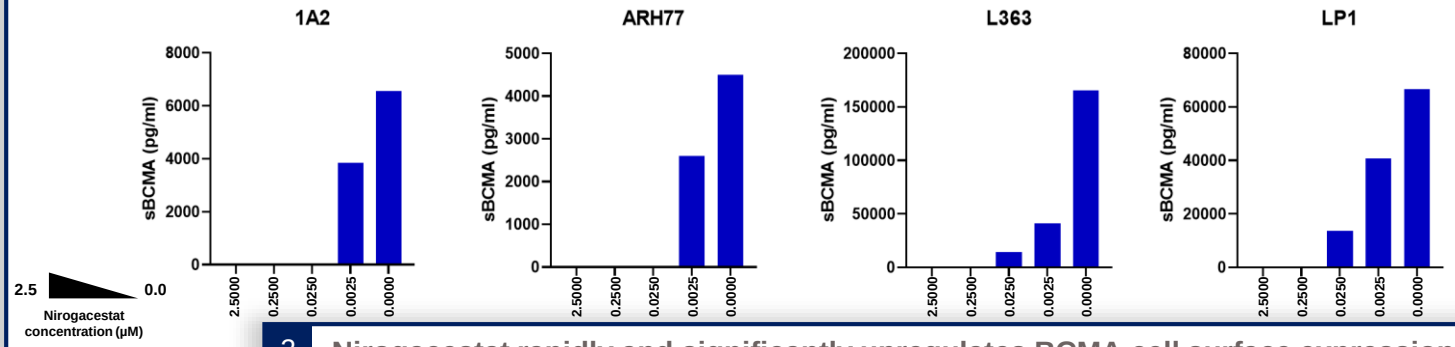


Nirogacestat has the potential to become a best-in-class potentiator of BCMA-directed therapies

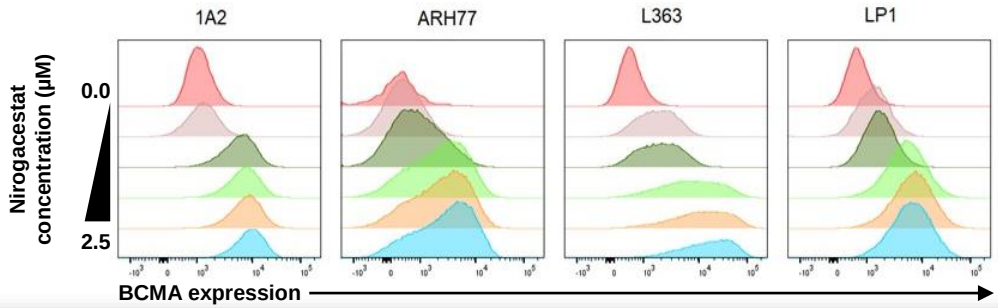
Source: Cowan et al., Abstract #204 "Efficacy and Safety of Fully Human Bcma CAR T Cells in Combination with a Gamma Secretase Inhibitor to Increase Bcma Surface Expression in Patients with Relapsed or Refractory Multiple Myeloma", ASH 2019; Eastman et al., Abstract #4401 "Synergistic Activity of Belantamab Mafodotin (anti-BCMA immuno-conjugate) with Nirogacestat (PF-03084014, gamma-secretase inhibitor) in Bcma-Expressing Cancer Cell Lines", ASH 2019; Green et al., Abstract #1856 "Response to Bcma CAR-T Cells Correlates with Pretreatment Target Antigen Density and Is Improved By Small Molecule Inhibition of Gamma Secretase", ASH 2019; Laurent et al., *Nat. Comm.*, 2015; Pont et al., *Blood*, 2019.

Nirogacestat Prevents BCMA Shedding, Upregulates BCMA Expression, and Shows Synergistic Cell Killing with BCMA ADC in Human MM Cell Lines

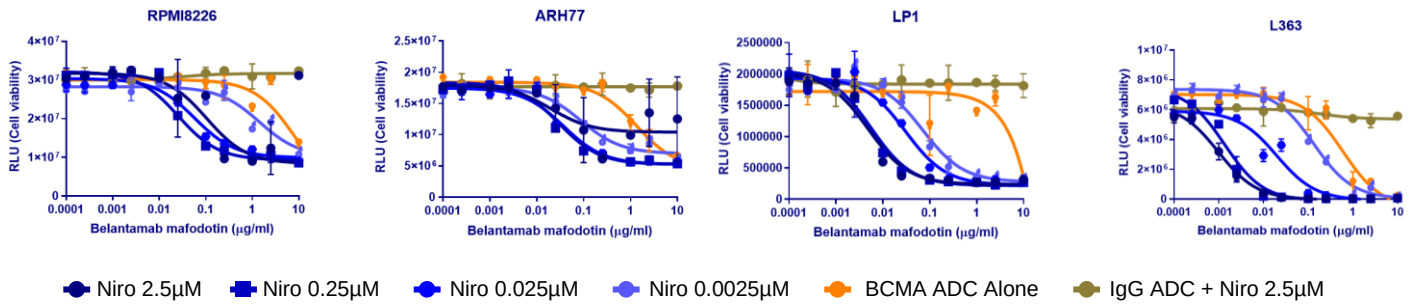
1 Nirogacestat prevents cleavage of membrane-bound BCMA and shedding of soluble BCMA ECD



2 Nirogacestat rapidly and significantly upregulates BCMA cell surface expression levels



3 Nirogacestat enhances multiple myeloma cell killing activity of BCMA ADC by up to ~3,000-fold



Note: ECD = extracellular domain; ADC = antibody-drug conjugate; MM = multiple myeloma.
Source: Eastman et al., Abstract #4401 "Synergistic Activity of Belantamab Mafodotin (anti-BCMA immuno-conjugate) with Nirogacestat (PF-03084014, gamma-secretase inhibitor) in Bcma-Expressing Cancer Cell Lines", ASH 2019.

Mirdametinib

NF1-Associated Plexiform Neurofibromas

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Mirdametinib: Potential Best-in-Class Therapy for Patients with NF1-PN

- Mirdametinib is an oral, small molecule MEK1/2 inhibitor with clinical validation and over 200 subjects exposed to date
- Encouraging results from Phase 2 investigator-initiated trial in adolescents and adults with NF1-associated plexiform neurofibromas (NF1-PN)
- Compound potency and optimized dose/schedule may allow for a potentially differentiated profile versus other MEK inhibitors
- FDA granted Orphan Drug Designation (NF1) and Fast Track Designation (NF1-PN), and European Commission granted Orphan Drug Designation (NF1)
- Phase 2b ReNeu trial currently enrolling both adult and pediatric patients, and regulatory precedent allows for single-arm trial to be potentially registrational

Phase 2b ReNeu trial update expected 4Q20-1Q21

Plexiform Neurofibromas Are Painful, Disfiguring Tumors That Grow Along Peripheral Nerve Sheaths

NF1-PN are a painful and devastating condition with significant morbidities

- Mutations in NF1 gene cause loss of neurofibromin, a key repressor of the MAPK pathway, leading to **uncontrolled tumor growth across the body**
- NF1-PN are **tumors that grow along the nerves** and can **lead to extreme pain and disfigurement**
- NF1 can have significant co-morbidities, including neurocognitive deficits and developmental delays

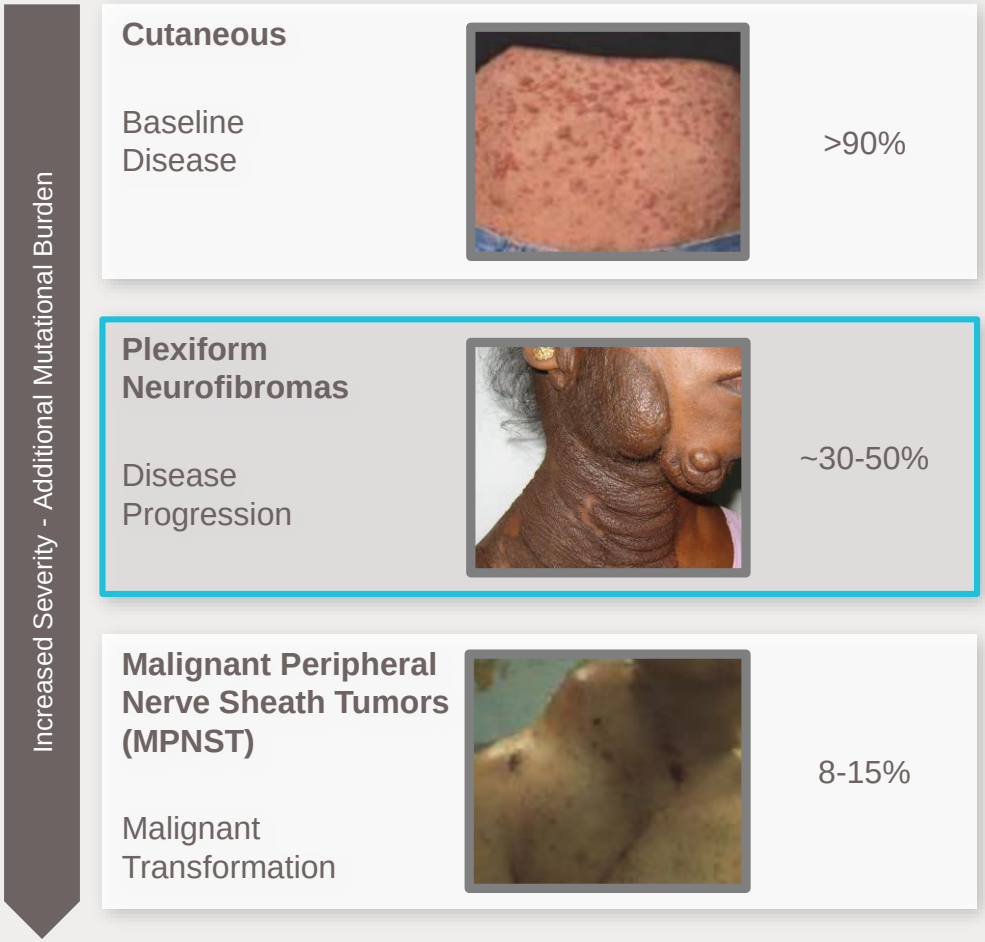
No currently approved therapies

- Infiltrative growth pattern along nerves make successful surgical resection challenging and **surgery can lead to permanent nerve damage and disfigurement**
- Off-label systemic therapies deemed inadequate

~100,000 NF1 patients in the United States

- **~30-50% lifetime risk of developing plexiform neurofibromas** in NF1 population
- **NF1-PN can malignantly transform into MPNST**, a diagnosis that has a **12-month survival rate of under 50%**

CLINICAL PRESENTATION OF NEUROFIBROMAS



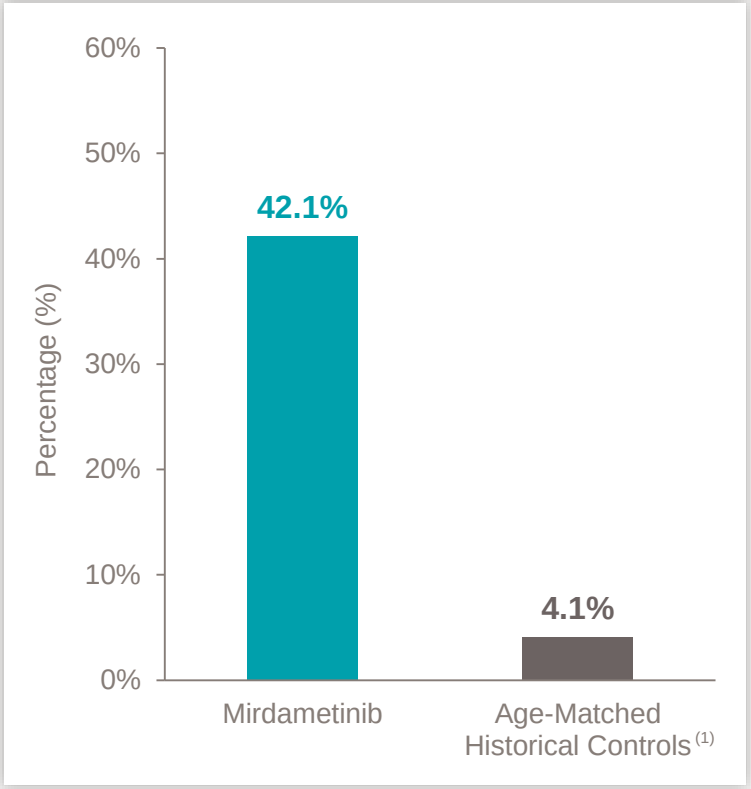
MEK inhibitors are rapidly emerging as a validated class for the treatment of NF1-PN

Source: Kim et al., *Sarcoma*, 2017.

Mirdametinib: Encouraging Phase 2 Results with Potentially Differentiated Safety Profile vs. Other MEK Inhibitors

PHASE 2 | PHASE 2B

Objective Response Rate



Trial Design and Clinical Activity

NEUROFIBROMATOSIS CONSORTIUM

- 19 patients with inoperable and symptomatic or growing PNs, ages 16-39 years (median age: 24)
- 2 mg/m^2 (up 4 mg BID) administered via intermittent (3 week on/1 week off) dosing schedule
- **8/19 (42%) responders, prospectively defined as ≥ 20% tumor reduction by course 12 of treatment**

Tolerability

- 5 dose reductions, all due to Gr2 events: rash (2), nausea (1), fatigue (1) and pain (1)
- No Gr4 events; 7 treatment-emergent Gr3 events reported in 5 patients; only 2 events (pain in the same patient) were considered treatment-related by the investigator
- **Dose and schedule minimized historical class toxicities observed with other MEK inhibitors**

We believe that mirdametinib has the opportunity to demonstrate a more tolerable safety profile than other MEK inhibitors

Source: Weiss, Children's Tumor Foundation 2017 Annual Meeting Presentation.

(1) In Nguyen et al. 2012, 95 NF1-PN patients had the volumes of single PN lesions monitored over time. Of these patients, 69 were greater than 16 years of age at the time of the initial assessment (range: 16.1 to 62.6 years), representing a total of 146 NF1-PN lesions. The duration of follow-up between scans ranged from 1.05 to 4.10 years (average: 2.40 years). Of the 146 lesions monitored, 6 were documented to have had a volumetric decrease of ≥20% (4.1%).

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Single-Arm Phase 2b Trial (ReNeu Trial) Has Commenced

PHASE 2 | **PHASE 2B**

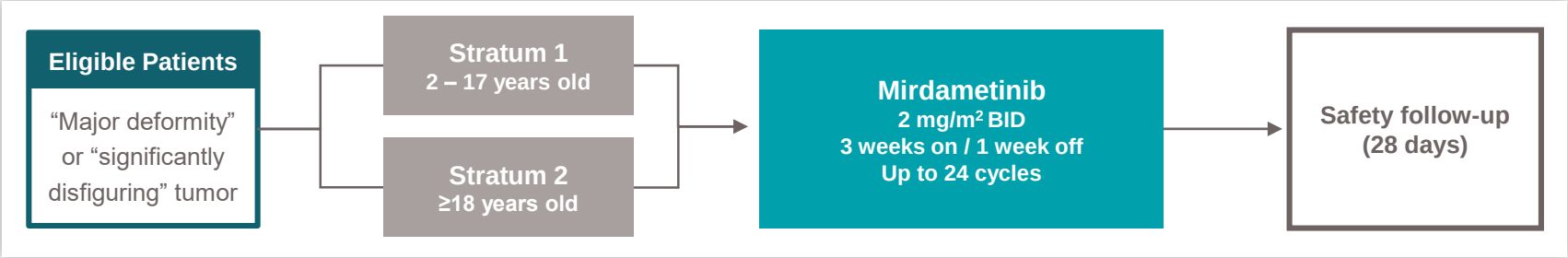
ReNeu

Trial Summary

- Expected to enroll ~100 patients in 2 strata (pediatrics, adults) across ~50 sites in North America
- 2 mg/m^2 BID dosing with intermittent 3 weeks-on, 1 week-off schedule (4-week course) for up to 24 cycles
  - Maximum dose of 4 mg BID

Summary of Endpoints

- Primary Endpoint: Objective response rate (ORR)
- Secondary Endpoints: Safety and tolerability, duration of response, overall quality of life (QoL) and change and effect of pain on QoL as measured by Pediatric Quality of Life Inventory



Key Event	Timing
Pre-IND Meeting	August 2018
Phase 2b Initiation	October 2019
Trial Update	4Q20-1Q21

Treatment duration and trial populations designed to demonstrate full potential of mirdametinib in NF1-PN

Mirdametinib + Lifirafenib

Combination Therapy in *RAS/RAF* Mutant and Other MAPK Pathway Aberrant Solid Tumors

SpringWorks THERAPEUTICS

Advancing Potentially Best-in-Class MEK/RAF-Dimer Inhibitor Combination in Collaboration with BeiGene

Phase 1b Trial Ongoing with Trial Update Expected in 2020

SpringWorks THERAPEUTICS / BeiGene

Mirdametinib + Lifirafenib

***RAS/RAF* Mutant Solid Tumors**

MEKi + RAF dimer inhibitor

- **Significant area of unmet need in cancer patients with *RAS/RAF* mutations and other MAPK pathway aberrations (approximately 25% of solid tumors)**
- **Lifirafenib possesses potentially best-in-class profile among RAF dimer inhibitors and preclinical data supports combination with mirdametinib**
- **Ph1b initiated in Australia in 2Q19 and US IND opened in 3Q19**
- **Trial update expected in 2020 from dose-escalation portion of Ph1b study**
- **Focused investment until significant clinical validation achieved**

Combination Approach: Vertical MAPK Pathway Inhibition to Address RAS-Mutant Solid Tumors

No currently approved targeted therapies for RAS mutant cancers

- RAS mutations account for **approximately 25% of all solid tumors, >200k newly incident US patients annually**
- Patients with RAS mutations typically have **poor prognoses or outcomes**

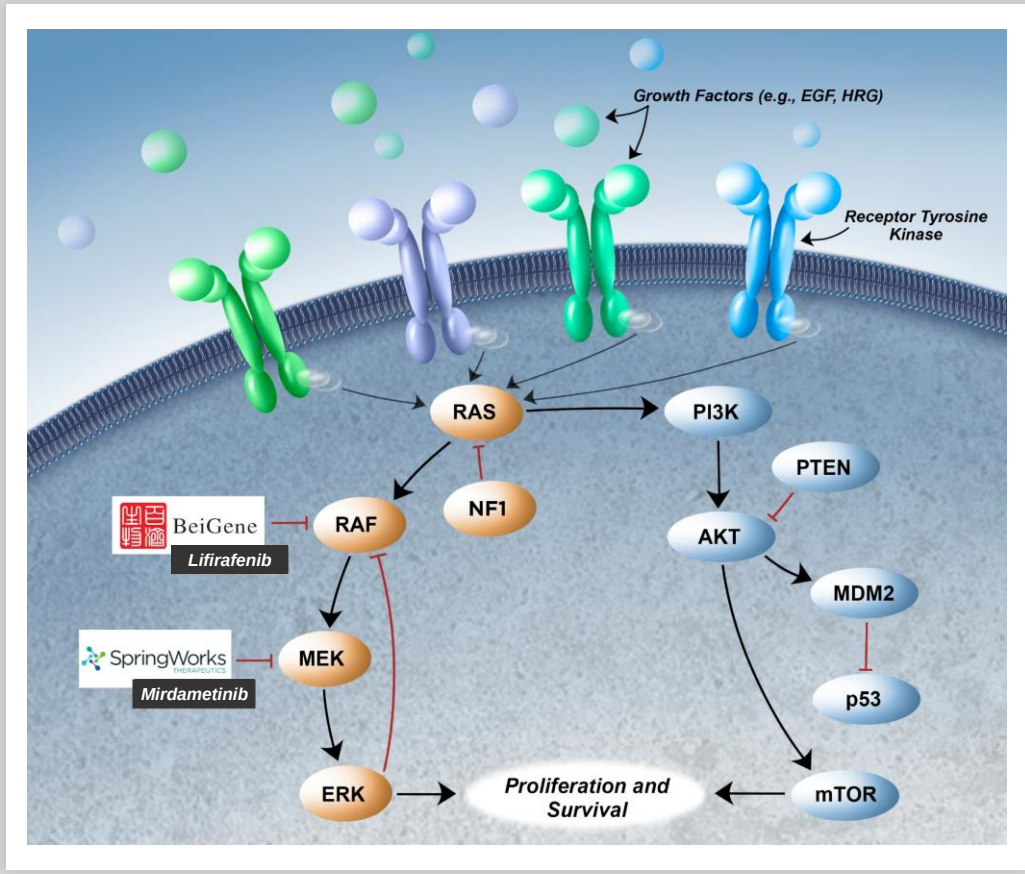
Lifirafenib is a RAF dimer inhibitor

- **Targets hetero- and homo-dimeric forms of RAF** and all RAF isoforms, unlike prior RAFi, which only inhibited monomeric signaling in BRAF V600E mutants
- **MEK/RAF vertical inhibition can abrogate the MAPK negative feedback loops** that result from targeting a single node

Combo allows for opportunity to meaningfully enhance monotherapy activity

- Lifirafenib Ph1 conducted in Australia showed **monotherapy activity in RAS and RAF mutant tumors**, which preclinical data suggest may be enhanced with addition of mirdametinib

MECHANISM OF ACTION OF MIRDAMETINIB + LIFIRAFENIB



Source: Zhao et al., *Nature Reviews Clinical Oncology*, 2014.

Mirdametinib + Lifirafenib: Encouraging Monotherapy Activity and Strong Preclinical Combo Data

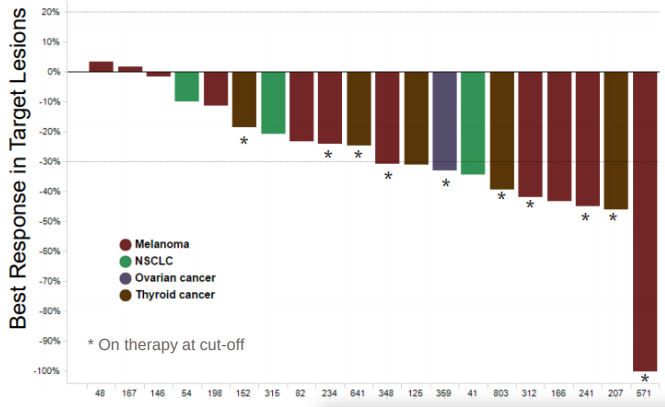
1 Potent Pharmacological Activity Demonstrated in Vitro

RAF isoforms	IC_{50} (nmol/L; mean ± SD)
$BRAF^{V600E}$	23 ± 5 nM
$BRAF^{WT}$	32 ± 8 nM
CRAF	7.0 ± 2.3 nM
ARAF	5.6 nM

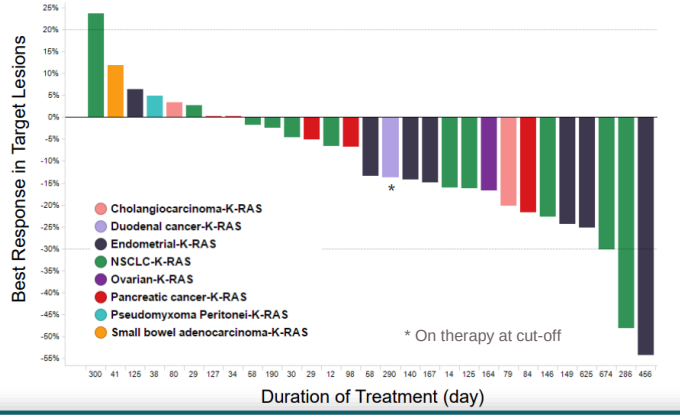
- In addition to potent RAF inhibition, lifirafenib targets both homo / heterodimers and all RAF isoforms, which is thought to be critical to efficacy in non-V600 BRAF and RAS mutants

2 Monotherapy Activity Shown in RAS and BRAF Mutant Solid Tumors

BRAF Mutant Cancers: Ph1a/b Best Objective Response

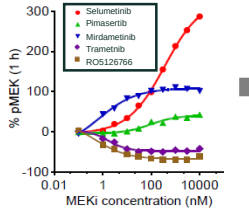


KRAS-Mut Cancers (ex-CRC): Ph1a/b Best Objective Response

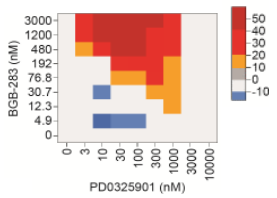


3 Compelling Preclinical Synergy Demonstrated with Mirdametinib

MEKi Monotherapy



Mirdametinib + Lifirafenib



- **Left:** pMEK at 1 hr at various MEKi monotherapy concentrations in Calu-6 cells (KRASQ61K)
- **Right:** Addition of lifirafenib improved mirdametinib activity in Calu-6 cells

Source: Desai presentation, "A Phase IB study of RAF dimer inhibitor BGB-283 in patients with B-RAF or K-RAS/N-RAS mutated solid tumors," AACR 2017; Tang et al., *Mol Cancer Ther*, 2015; Yuan et al., Abstract #669: "BGB-283 effectively enhances MEK inhibitor induced tumor suppression in RAS mutant cancers", AACR 2015.

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Patients Currently Being Enrolled in the Dose-Escalation Portion of the Phase 1b Study

PHASE 1B

Study Summary

- Adaptive Ph1b study in patients with advanced/refractory cancers harboring *KRAS*, *NRAS* and *BRAF* mutations and other MAPK aberrations
- Trial commenced in Australia in May 2019 and US IND opened in July 2019; additional clinical sites to be opened
- BeiGene leading clinical operations

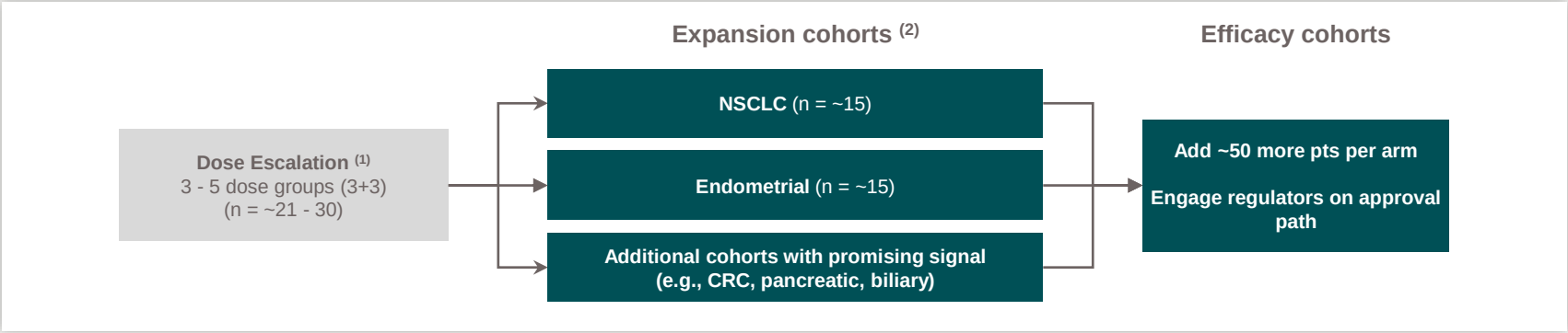
Summary of Endpoints

Part A

- Primary Endpoint: Safety and tolerability, maximum tolerated dose/recommended Phase 2 dose
- Secondary Endpoints: PK profile of combination, efficacy measures (ORR, duration, DCR, PFS, OS)

Part B

- Primary Endpoint: Objective response rate



Key Event	Timing
Phase 1b Initiation	May 2019
Trial Update	2020
Efficacy Arm Decisions	4Q20-1Q21

(1) Recruitment of no more than 50% with CRC or pancreatic cancer. Baseline tissue from either fresh tumor or archived tissue is mandatory for mutational and biomarker analysis.
(2) Paired tumor biopsies within 8 weeks of first dose and week 3 of cycle 1 are mandatory.

BGB-3245

RAF Mutant Solid Tumors

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BGB-3245: Potentially Differentiated Program for Currently Unaddressed RAF Driver Mutations

Phase 1 Dose Escalation and Expansion Trial Expected to Initiate in 1Q20

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BGB-3245

(via MapKure)

***RAF* Mutant Solid Tumors**

RAF fusion and dimer inhibitor

- **BGB-3245 is a novel RAF inhibitor being advanced in collaboration with BeiGene through MapKure, a newly formed, jointly owned entity**
- **BGB-3245 could address RAF alterations that currently lack targeted therapeutic options (newly described mutations and fusions)**
- **Preclinical activity demonstrated in tumor models with clinically-relevant biomarkers**
- **Preclinical package completed and Phase 1 trial expected to initiate in Australia in 1Q20, followed by submission of US IND thereafter**
- **Industry-leading Scientific Advisory Board guiding program and highlighted by Dr. Neal Rosen of Memorial Sloan Kettering Cancer Center**

The SpringWorks Opportunity

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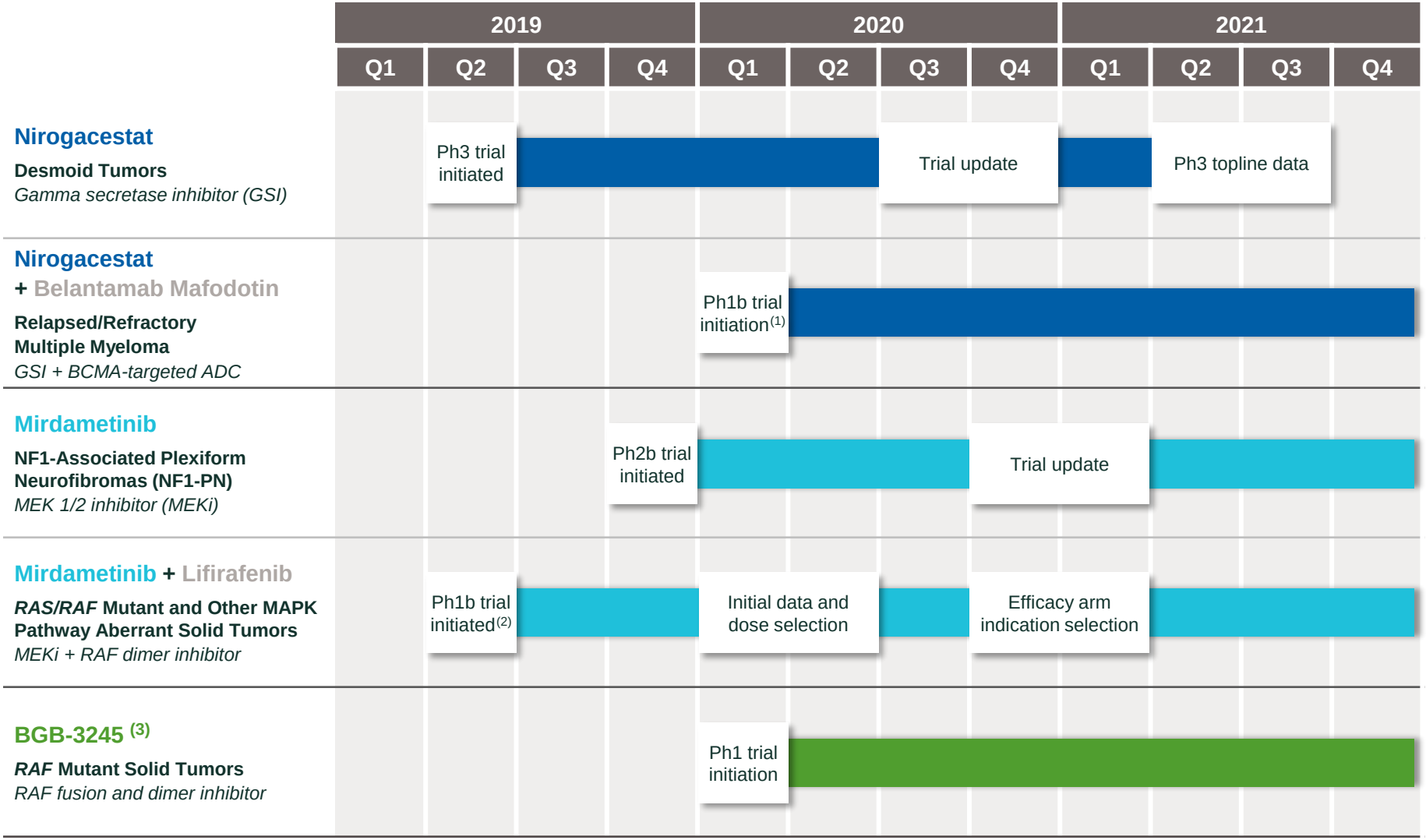
Pipeline is Rich in Near-Term Anticipated Catalysts



Collaborator Asset

(1) Phase 1b clinical trial evaluating belantamab mafodotin in combination with nirogacestat in patients with relapsed or refractory multiple myeloma will be examined as a sub-study in GlaxoSmithKline's DREAMM-5 platform trial.
(2) Clinical trial being conducted by BeiGene.
(3) Program being developed by MapKure, LLC, jointly owned by SpringWorks and BeiGene.

Financial Highlights

Over $400M raised in gross proceeds since company formation in 2017

$228M

Private Financing Proceeds

$186M

September 2019 IPO Proceeds

Current cash position expected to fund operations through 2022, enabling completion of 5 ongoing clinical trials

$344M

Cash & Cash Equivalents(1)

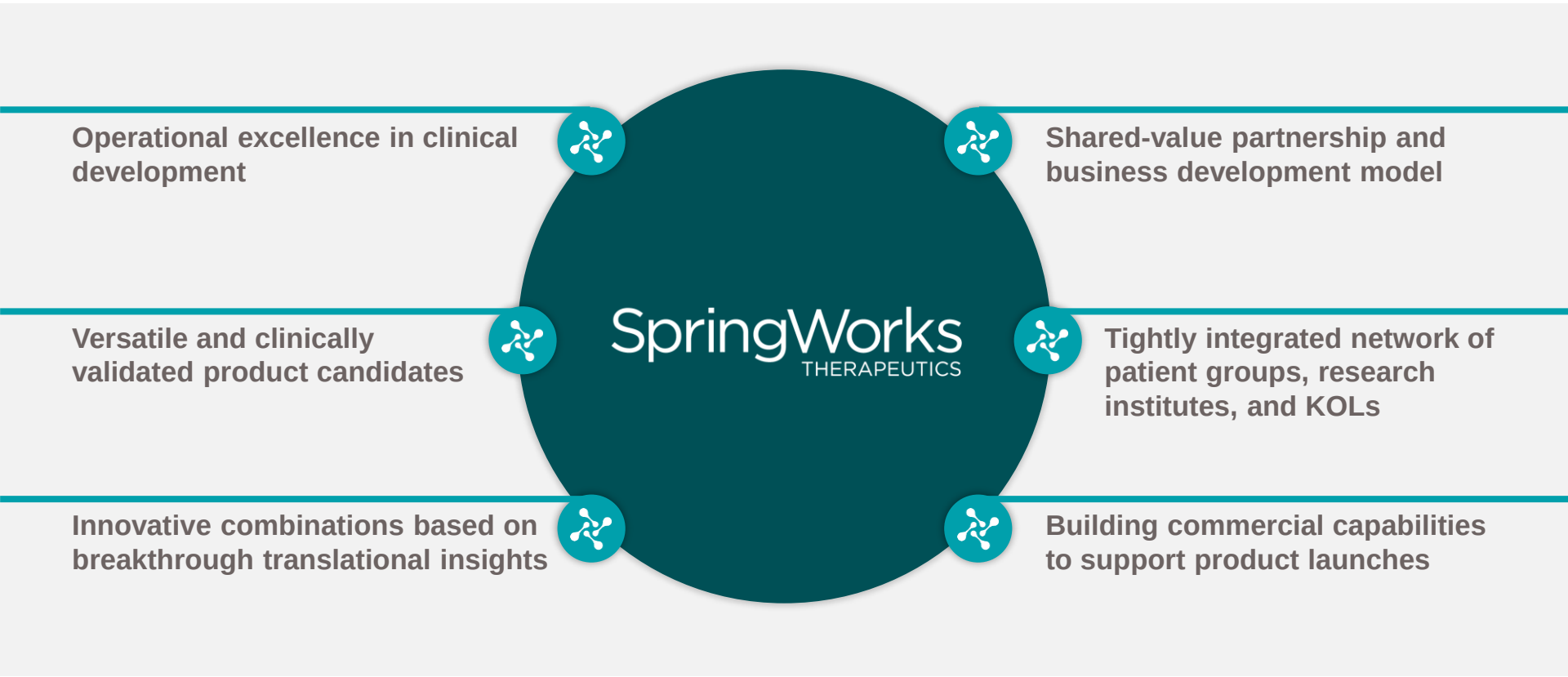
No Debt

43.0M

Common Shares Outstanding(2)

(1) As of September 30, 2019.
(2) As of November 12, 2019.

SpringWorks is Advancing a Diversified Portfolio of Targeted Oncology Therapies on the Path Towards Becoming a Commercial-Stage Company



5 programs in the clinic by **1Q20**

2 potentially registrational trials in progress

3 partnerships to develop therapies in large cancer indications


Thank You