



## Corporate Presentation

September 2020

NASDAQ: SWTX

# Forward-Looking Statements

This presentation may contain “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995 relating to our business, operations, and financial conditions, including but not limited to current beliefs, expectations and assumptions regarding the future of our business, future plans and strategies, our development plans, our preclinical and clinical results and other future conditions. Words such as, but not limited to, “look forward to,” “believe,” “expect,” “anticipate,” “estimate,” “intend,” “plan,” “would,” “should” and “could,” and similar expressions or words, identify forward-looking statements. New risks and uncertainties may emerge from time to time, and it is not possible to predict all risks and uncertainties. Any forward-looking statements in this presentation are based on management’s current expectations and beliefs and are subject to a number of risks, uncertainties and important factors that may cause actual events or results to differ materially from those expressed or implied by any forward-looking statements contained in this presentation, including, without limitation, risks relating to: (i) the success and timing of our ongoing DeFi and ReNeu clinical trials, (ii) the success and timing of our product development activities and initiating clinical trials, (iii) the success and timing of our collaboration partners’ ongoing and planned clinical trials, (iv) our ability to obtain and maintain regulatory approval of any of our product candidates, (v) our plans to research, discover and develop additional product candidates, (vi) our ability to enter into collaborations for the development of new product candidates, (vii) our ability to establish manufacturing capabilities, and our and our collaboration partners’ abilities to manufacture our product candidates and scale production, (viii) our ability to meet any specific milestones set forth herein, and (ix) uncertainties and assumptions regarding the impact of the COVID-19 pandemic on SpringWorks’ business, operations, clinical trials, supply chain, strategy, goals and anticipated timelines.

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# SpringWorks Therapeutics is a Clinical-Stage Targeted Oncology Company



- **Two late-stage rare oncology programs** in potentially registrational trials, each supported by strong clinical data
- **Five 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 a partner of choice
- 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

ALEXION<sup>®</sup>



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

ALEXION<sup>®</sup>



**Badreddin Edris, Ph.D.**  
Chief Business Officer



**Frank Perier, MBA**  
Chief Financial Officer



Forest Laboratories, Inc.



**Mary Smith, Ph.D.**  
Senior Vice President, Clinical  
Research and Development



## Board of Directors

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*Chairman of the Board*

**Alan Fuhrman**  
Chief Financial Officer,  
Amplix Pharmaceuticals

**Julie Hambleton, M.D.**  
Former Chief Medical Officer,  
IDEAYA Biosciences

**Saqib Islam, J.D.**  
Chief Executive Officer,  
SpringWorks Therapeutics

**Freda Lewis-Hall, M.D., DFAPA**  
Former Chief Medical Officer,  
Pfizer

**Jeffrey Schwartz**  
Managing Director,  
Bain Capital Life Sciences

**Stephen Squinto, Ph.D.**  
Executive Partner,  
OrbiMed



# Pipeline Provides Multiple Opportunities for Value Creation Across Three Distinct Oncology Segments



1

## Late-Stage Rare Oncology

*Two registrational trials ongoing, each supported by strong Phase 2 data and with best-in-class potential*



### Nirogacestat

Desmoid Tumors

Phase 3 topline data: 2Q21-3Q21



### Mirdametinib

NF1-PN

Phase 2b trial update: 4Q20-1Q21

2

## BCMA Combinations in Multiple Myeloma

*Advancing nirogacestat as a cornerstone of BCMA combination therapy across modalities*



### Nirogacestat + BLENREP

BCMA ADC

Phase 1b trial initiated: 1H20



### Nirogacestat + ALLO-715

BCMA Allogeneic CAR T

Phase 1 trial IND filing: 2H20



### Nirogacestat + Teclistamab

BCMA-CD3 Bispecific

Phase 1 trial initiation: Early 2021

3

## Biomarker-Defined Metastatic Solid Tumors

*Precision oncology approach to highly prevalent cancers with near-term clinical POC readouts*



### Mirdametinib + Lifirafenib

RAS/RAF Mutant Solid Tumors

Phase 1b/2 initial clinical data: 2021










### BGB-3245

RAF Mutant Solid Tumors

Phase 1 initial clinical data: 2021



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

	Preclinical	Phase 1	Phase 2	Phase 3	Collaborator	Key Milestones
Nirogacestat (Gamma Secretase Inhibitor)						
Desmoid Tumors*	Monotherapy 					Phase 3 topline data: <b>2Q21-3Q21</b>
Relapsed/Refractory Multiple Myeloma	+ BLENREP (belantamab mafodotin) (BCMA ADC)					Phase 1b trial initiated: <b>1H20</b>
	+ ALLO-715 (BCMA CAR T)					Phase 1 trial IND filing: <b>2H20</b>
	+ Teclistamab (BCMA Bispecific)					Phase 1 trial initiation: <b>Early 2021</b>
Mirdametinib (MEK 1/2 Inhibitor)						
NF1-Associated Plexiform Neurofibromas†	Monotherapy (pediatric and adult study) 					Phase 2b trial update: <b>4Q20-1Q21</b>
RAS/RAF Mutant and Other MAPK Pathway Aberrant Solid Tumors	+ Lifirafenib (RAF dimer inhibitor)				 BeiGene	Phase 1b/2 initial clinical data: <b>2021</b>
BGB-3245 (RAF Fusion and Dimer Inhibitor)						
RAF Mutant Solid Tumors	Monotherapy				 BeiGene <sup>(1)</sup>	Phase 1 initial clinical data: <b>2021</b>

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.

# Nirogacestat



Dana  
Desmoid patient

# Nirogacestat: A New Paradigm for Patients With Desmoid Tumors

- Desmoid tumors are highly morbid soft tissue tumors with an estimated 5,500 to 7,000 patients actively receiving treatment in the US per year
- Nirogacestat is an oral, selective gamma secretase inhibitor with over 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 Fast Track and Breakthrough Therapy Designations from FDA and Orphan Drug Designation from both FDA and European Commission
- Newly granted US patent provides protection to 2039<sup>(1)</sup>

**Phase 3 DeFi trial fully enrolled and topline data anticipated in 2Q-3Q21**



# 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

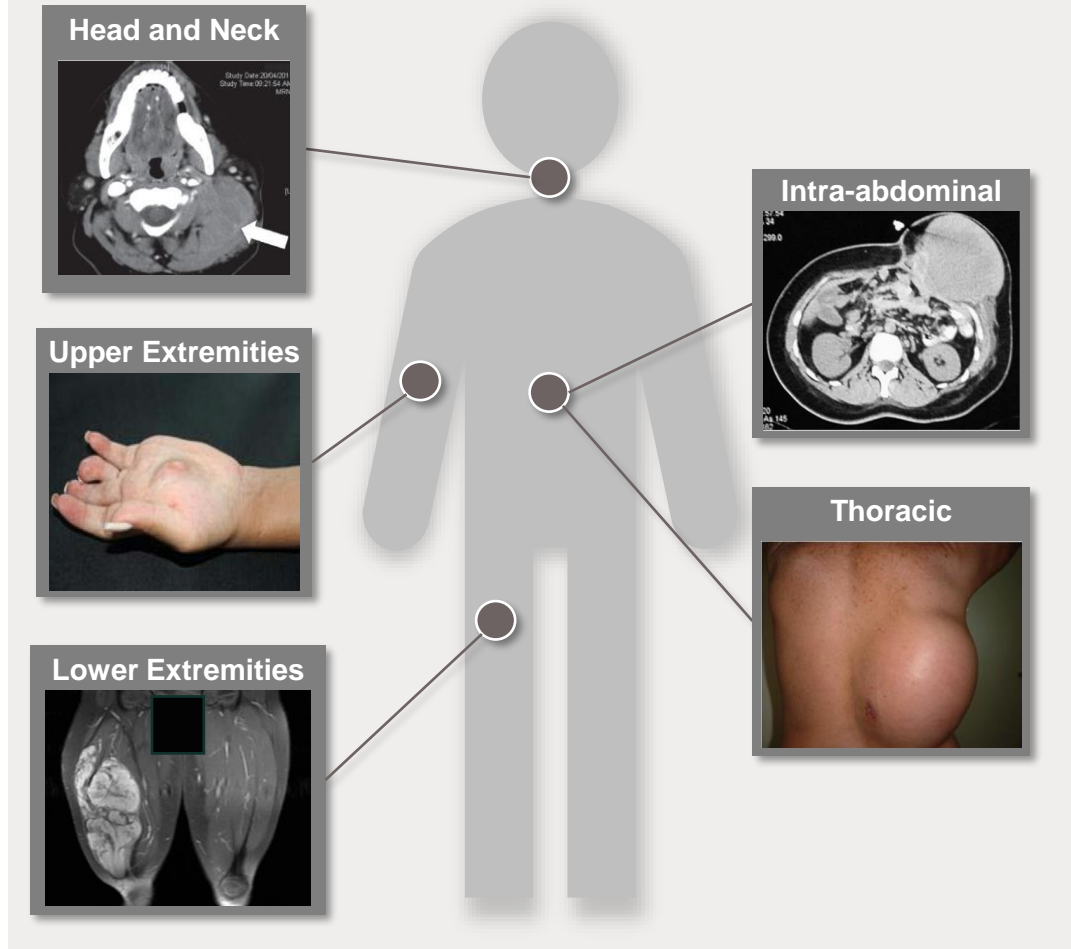
## No currently approved therapies

- 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

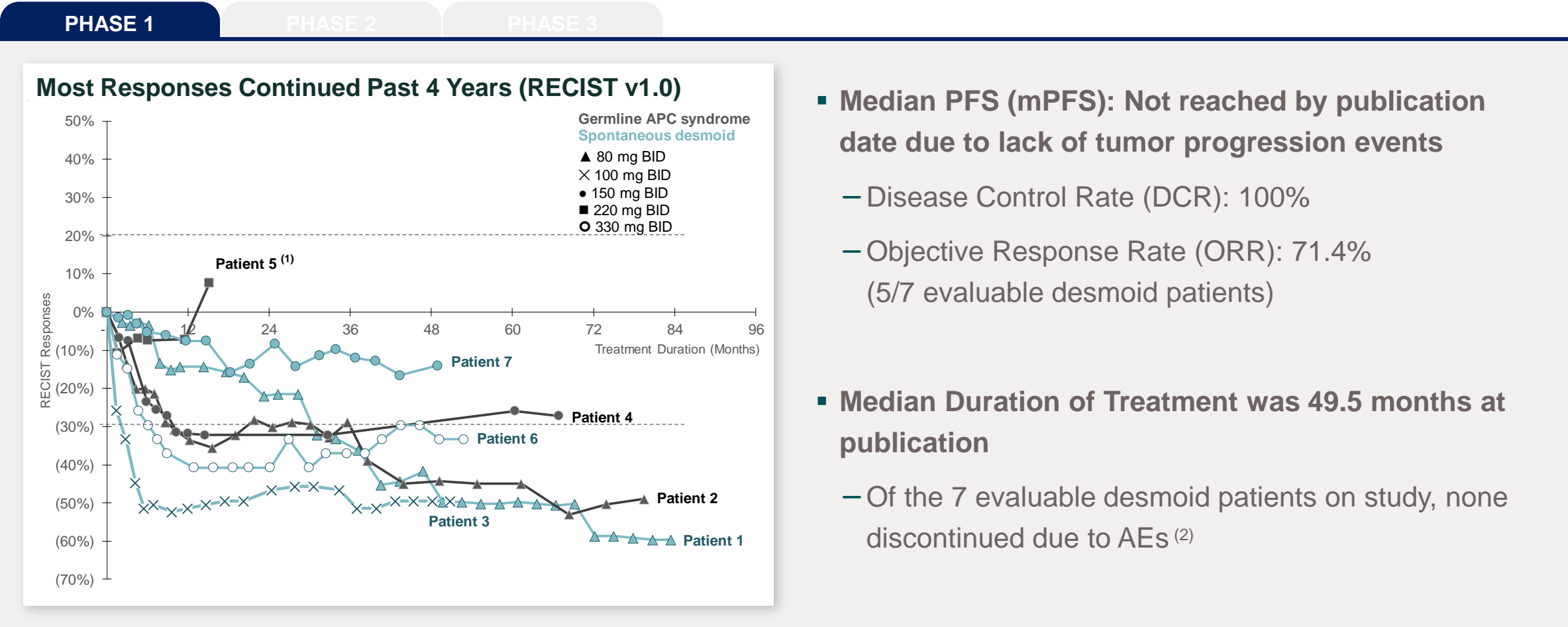
## ~1,000-1,500 newly incident patients per year in US

- 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



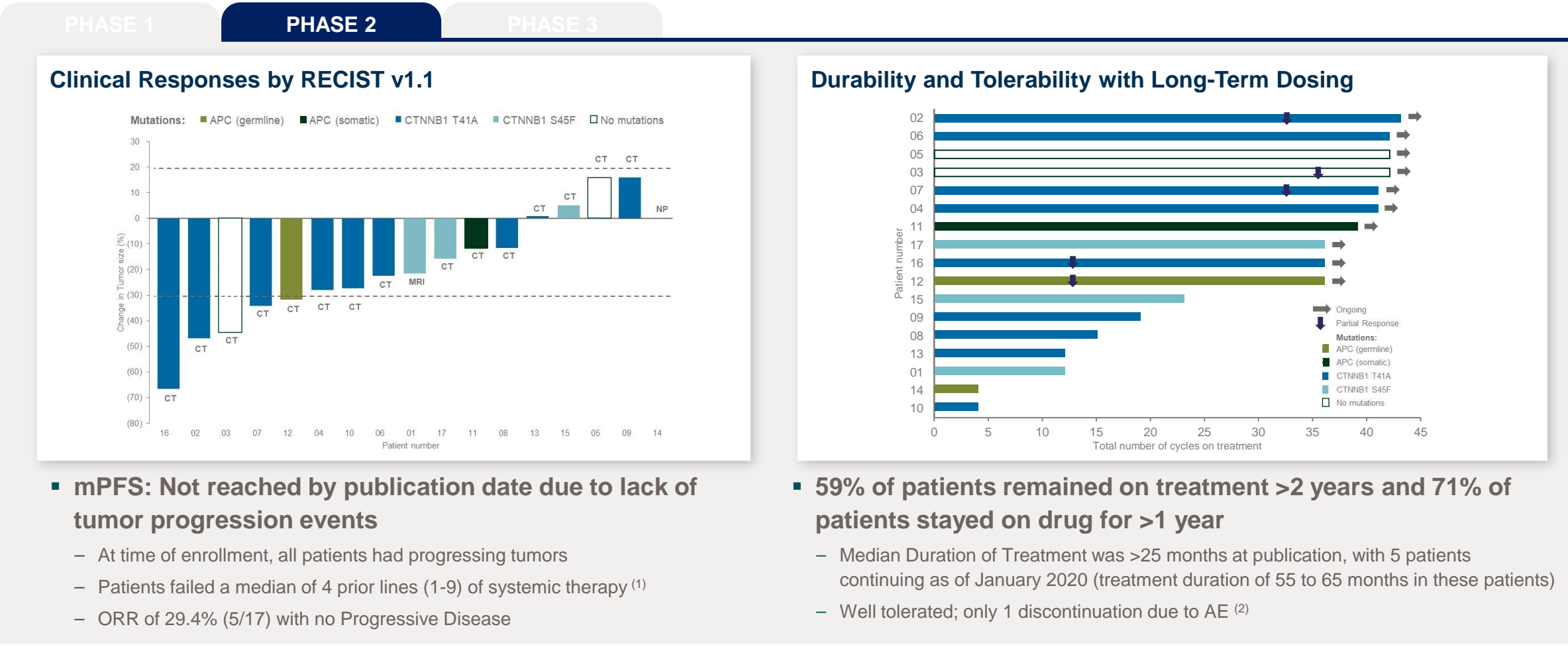
# Initial Clinical Activity of Nirogacestat Observed in Desmoid Tumors



All evaluable desmoid tumor patients in the study responded to nirogacestat treatment <sup>(1)</sup>

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



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: Kummer 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) Discontinuation due to grade 2 urticaria not responsive to dose reduction. 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 DeFi Trial Is Fully Enrolled

PHASE 1

PHASE 2

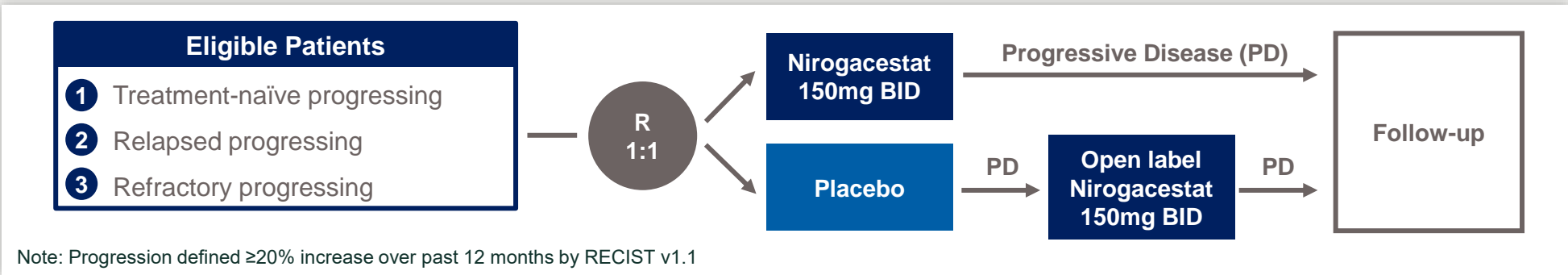
PHASE 3

## Trial Summary

- ~115 patients at ~50 sites in North America and Europe
- Open label extension for patients progressing on placebo
- 90% powered to show ~12 month PFS difference between nirogacestat and placebo <sup>(1)</sup>

## Summary of Endpoints

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



Key Event	Timing
Trial Initiated	May 2019
Trial Fully Enrolled	July 2020
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.

***Nirogacestat has the potential to be a cornerstone of BCMA combination therapy***

## Nirogacestat in Multiple Myeloma: A Potentially Best-in-Class Potentiator of BCMA Therapies

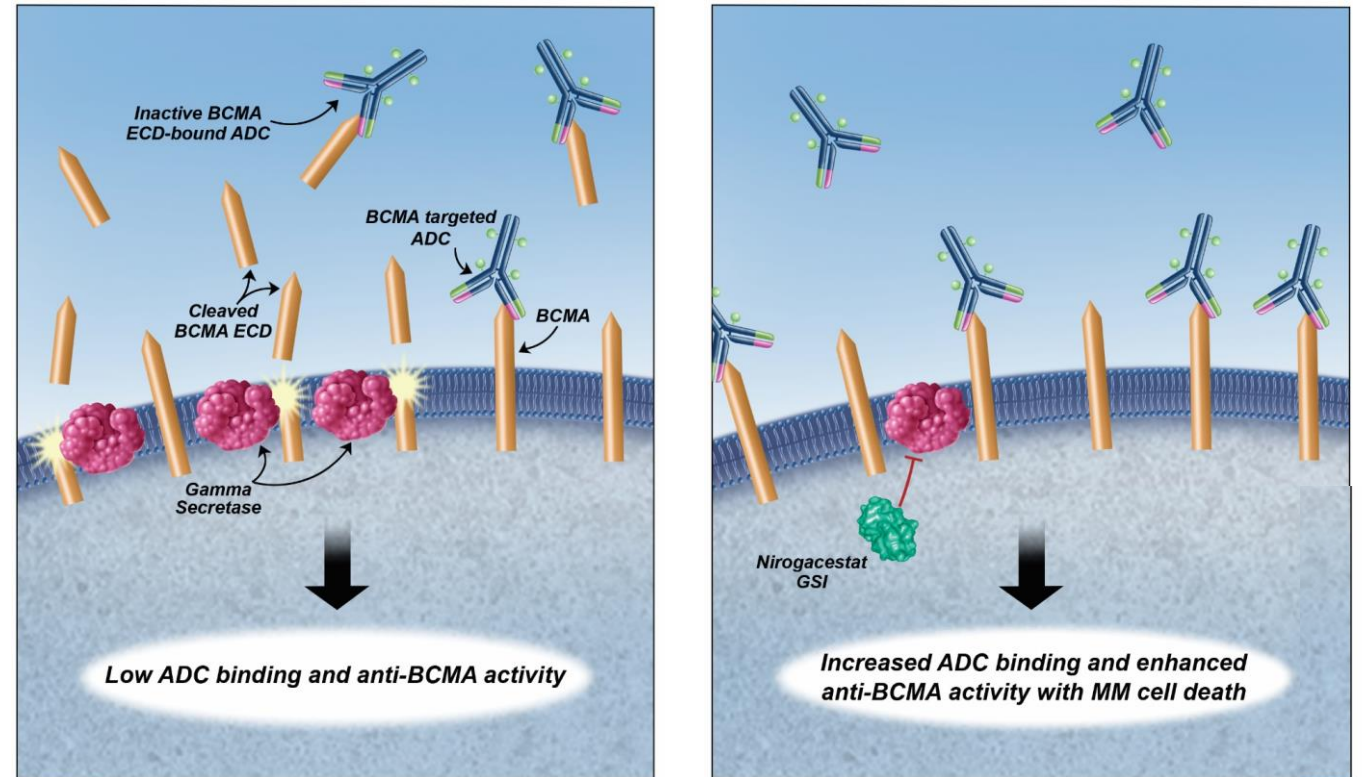
- Significant unmet need in multiple myeloma, with ~27,000 new patients in the relapsed/refractory setting in the US each year
- Gamma secretase directly cleaves membrane-bound BCMA, a clinically validated multiple myeloma target across modalities (ADC, CAR T, bispecific)
- Strong preclinical results and emerging clinical data support combining gamma secretase inhibitors with BCMA therapies
- Pursuing broad collaboration strategy with industry-leading BCMA developers to advance potentially best-in-class combinations using nirogacestat
- Newly granted US patent provides protection to 2039<sup>(1)</sup>



# Gamma Secretase Inhibition is Emerging as a Clinically Validated Mechanism to Potentiate BCMA Therapies

- **BCMA has emerged as a promising target in multiple myeloma 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**

## MECHANISM OF ACTION OF NIROGACESTAT + BCMA THERAPY (ADC SHOWN)

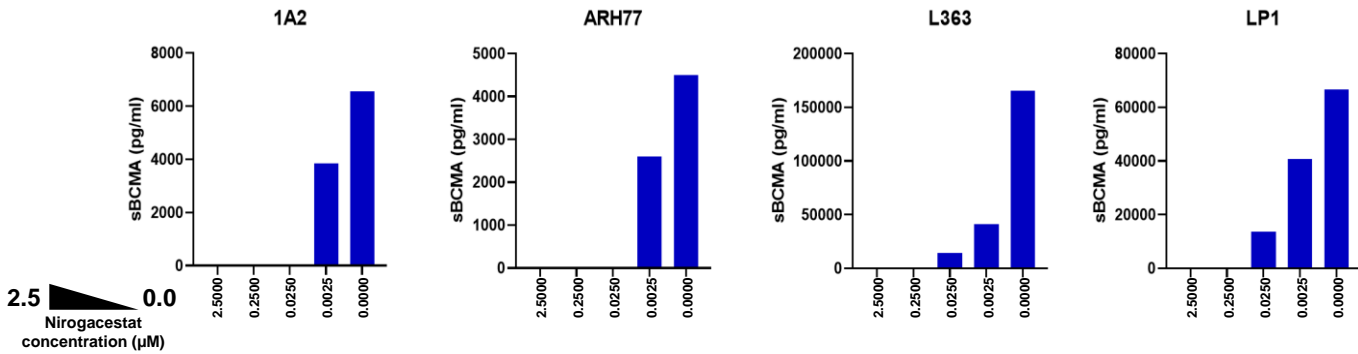


Source: Cowan et al., Abstract #204 "Efficacy and Safety of Fully Human Bcma CAR T Cells in Combination with a Gamma Secretase Inhibitor to Increase Bcma Surface Expression in Patients with Relapsed or Refractory Multiple Myeloma", ASH 2019; Eastman et al., Abstract #4401 "Synergistic Activity of Belantamab 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 Inhibited BCMA Shedding, Upregulated BCMA Expression, and Enhanced Activity of BCMA ADC Up to ~3,000-Fold

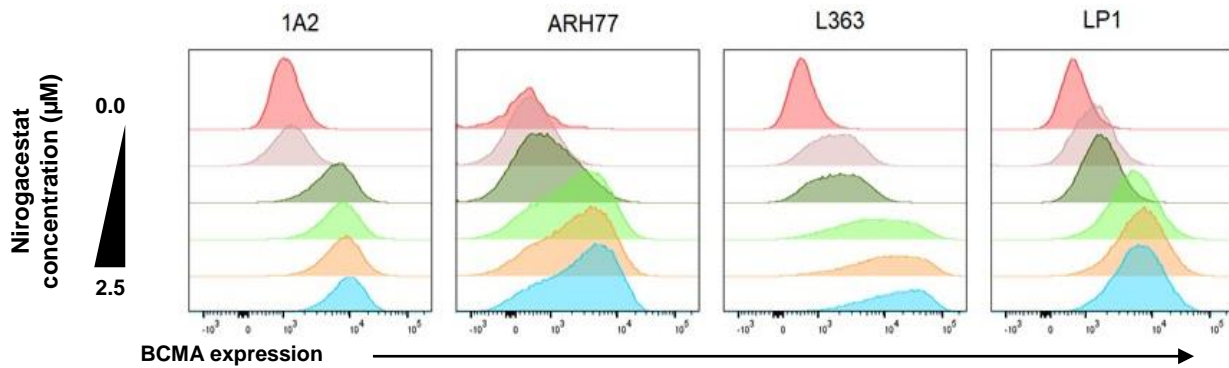
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Nirogacestat inhibited cleavage of membrane-bound BCMA and shedding of soluble BCMA ECD



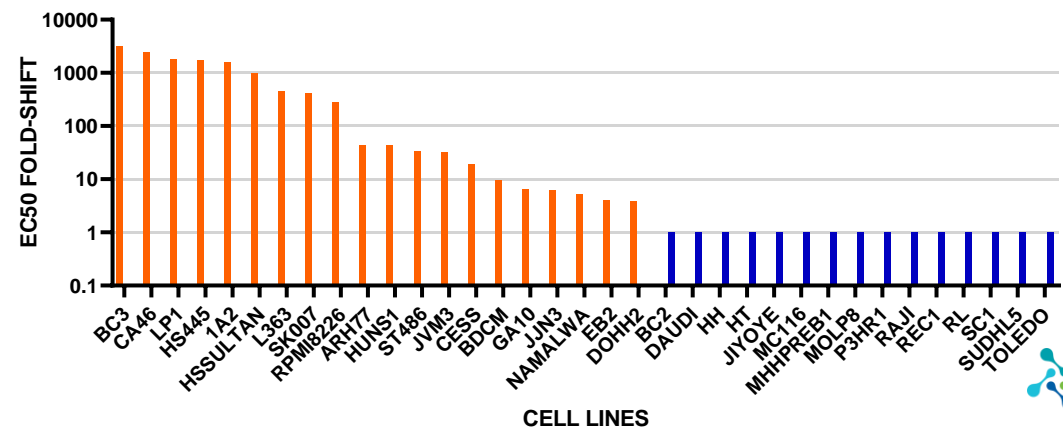
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Nirogacestat rapidly and significantly upregulated BCMA cell surface expression levels



3

Nirogacestat enhanced 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.

# Three BCMA Collaborations Signed To Date with GSK, Allogene and Janssen



## Nirogacestat + BLENREP

*BCMA Antibody-Drug  
Conjugate (ADC)*

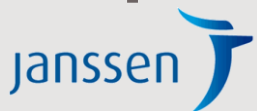
- Clinical collaboration signed in June 2019 with first-in-class BCMA ADC
- Preclinical synergy demonstrated in data presented at ASH 2019
- Combination is part of GSK's DREAMM-5 platform trial
- Nirogacestat sub-study was initiated 1H20



## Nirogacestat + ALLO-715

*BCMA Allogeneic CAR T  
Cell Therapy*

- Clinical collaboration signed in January 2020 with first allogeneic BCMA CAR T cell therapy to enter the clinic
- Working with leaders in 'off-the-shelf' CAR T cell therapy field to further explore nirogacestat's potential benefit in multiple myeloma
- IND for combination trial sponsored by Allogene expected to be filed in 2H20



## Nirogacestat + Teclistamab

*BCMA-CD3 Bispecific  
Antibody*

- Clinical collaboration signed in September 2020 with clinical-stage bispecific antibody targeting BCMA and CD3
- Combination clinical trial sponsored by Janssen is anticipated to commence in early 2021

**Nirogacestat has the potential to become a cornerstone of BCMA combinations for the treatment of multiple myeloma**

# Mirdametinib



Kendall  
NF1 patient

# Mirdametinib: A Potentially Best-in-Class Therapy for Patients with NF1-PN

- ~100,000 patients in the US with NF1 – 30-50% lifetime risk of developing disfiguring peripheral nerve sheath tumors (plexiform neurofibromas)
- 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)
- Granted Orphan Drug Designation by FDA and European Commission in NF1 and FDA Fast Track Designation in NF1-PN
- Compound potency and optimized dose/schedule may allow for a potentially differentiated profile versus other MEK inhibitors

**Phase 2b ReNeu trial currently enrolling and update expected 4Q20-1Q21**



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


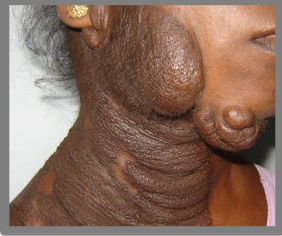

## NF1-PN are a painful and devastating condition with significant morbidities

- 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
- Infiltrative growth pattern along nerves make successful surgical resection challenging and **surgery can lead to permanent nerve damage and disfigurement**

## ~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

Increased Severity - Additional Mutational Burden	Lifetime Risk		
	Cutaneous		>90%
	Plexiform Neurofibromas		~30-50%
	Malignant Peripheral Nerve Sheath Tumors (MPNST)		8-15%
Baseline Disease			
Disease Progression			
Malignant Transformation			

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

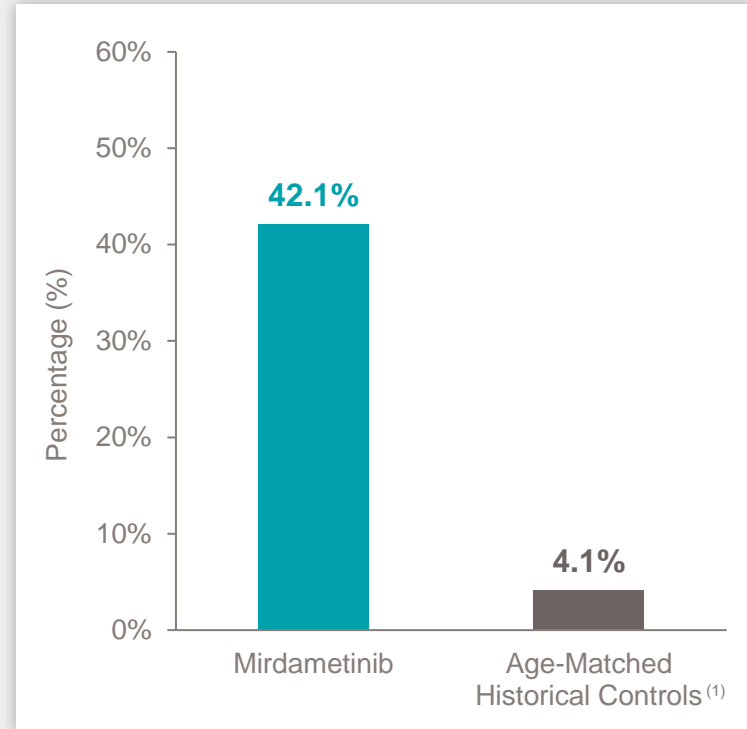
# Mirdametinib: Encouraging Phase 2 Results with Potentially Differentiated Safety Profile vs. Other MEK Inhibitors

PHASE 2

PHASE 2B



## Objective Response Rate



## Trial Design and Clinical Activity

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

## 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**

**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  $\geq 20\%$  (4.1%).

# Potentially Registrational Pediatric and Adult Phase 2b ReNeu Trial Has Commenced

PHASE 2

PHASE 2B

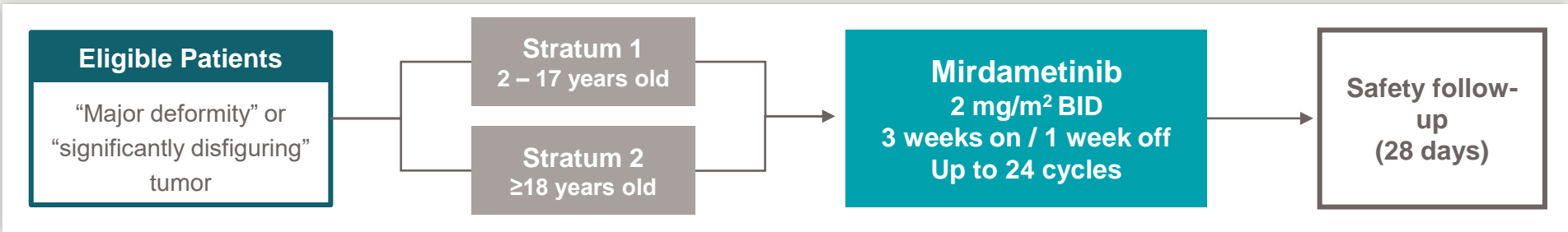


### Trial Summary

- Enrolling ~100 patients in 2 strata (pediatrics, adults) across ~50 sites in North America
- 2 mg/m<sup>2</sup> BID dosing with intermittent course (4-week cycles of 3 weeks-on, 1 week-off) 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, quality of life (QoL) assessments



Key Event	Timing
Trial Initiated	October 2019
Trial Update	4Q20-1Q21

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

# Mirdametinib in *RAS/RAF* Mutant Solid Tumors: Advancing Potentially Best-in-Class MEK/RAF Dimer Inhibitor Combination in Collaboration with BeiGene



BeiGene

## Mirdametinib + Lifirafenib

*MEKi + RAF dimer inhibitor*

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

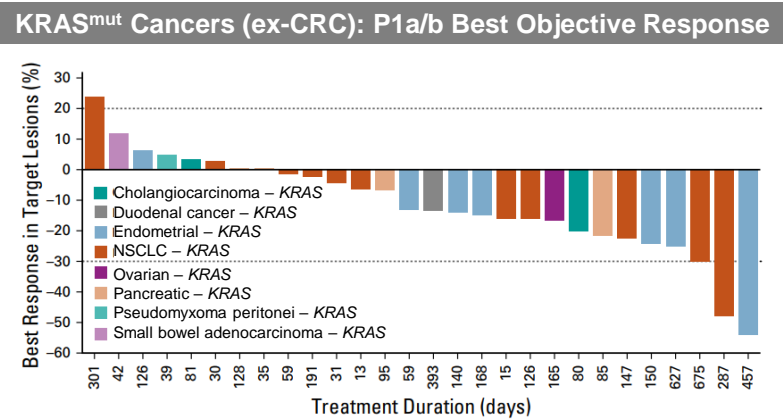
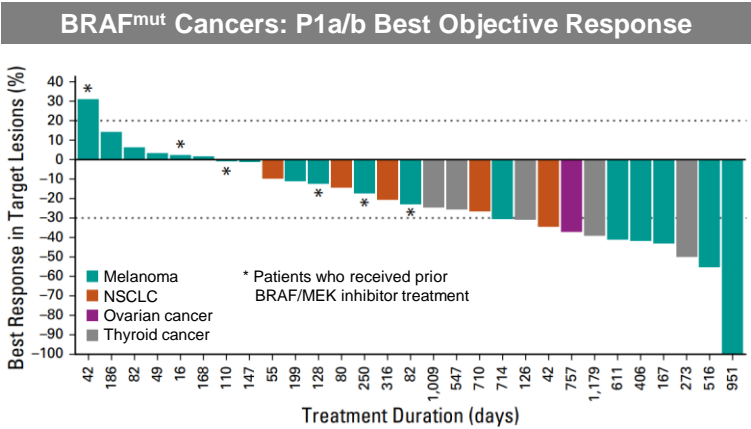
- Significant area of unmet need in cancer patients with *RAS/RAF* mutations and other MAPK pathway aberrations (approximately 25% of solid tumors)
- Lifirafenib possesses potentially best-in-class profile among RAF dimer inhibitors
- Combination synergy demonstrated across preclinical models harboring a variety of *KRAS* mutations
- Phase 1b/2 trial initiated in Australia in 2Q19 and US IND opened in 3Q19
- Focused investment until significant clinical validation achieved

**On track to complete dose-escalation portion of trial by end of 2020 with initial clinical data expected in 2021**

# Mirdametininib + Lifirafenib: Encouraging Monotherapy Clinical Activity and Strong Preclinical Combination Data

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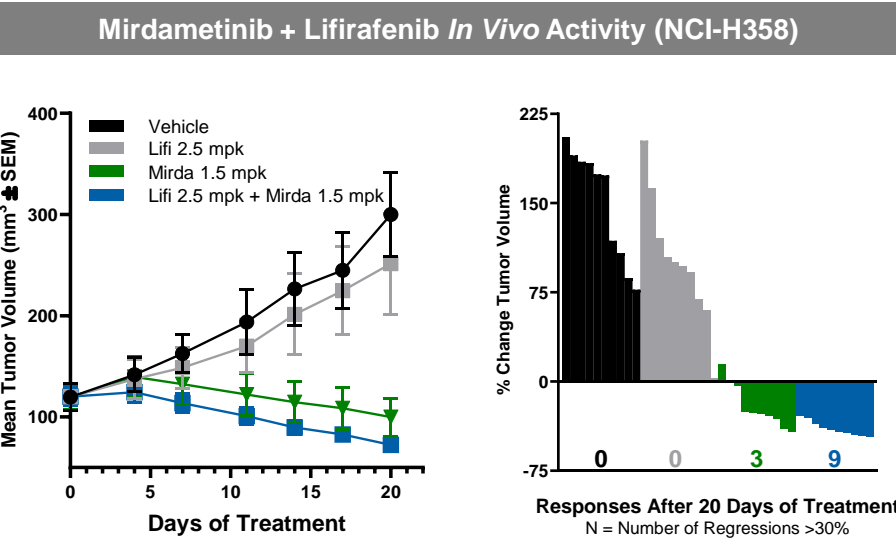
Lifirafenib monotherapy clinical activity in *BRAF* and *KRAS* mutant cancers



2

Preclinical synergy demonstrated with mirdametininib and lifirafenib *in vitro* across *RAS* mutations and *in vivo* at clinically relevant doses

NSCLC Cell Line	RAS Mutation	Max EC <sub>50</sub> shift with mirdametininib combo
Calu-6	K-RAS Q61K	59 fold ↓
SW1573	K-RAS G12C	97 fold ↓
NCI-H23	K-RAS G12C	22 fold ↓
NCI-H2122	K-RAS G12C	21 fold ↓
NCI-H358	K-RAS G12C	18 fold ↓
Calu-1	K-RAS G12C	No shift
Sk-Lu-1	K-RAS G12D	32 fold ↓
A549	K-RAS G12S	11 fold ↓
NCI-H1299	N-RAS Q61K	16 fold ↓





# BGB-3245

# BGB-3245: Potentially Differentiated Program for Currently Unaddressed *BRAF* Driver Mutations and Fusions



SpringWorks  
THERAPEUTICS

+



BeiGene

**BGB-3245**  
*Mutant BRAF monomer, dimer, and  
fusion inhibitor*  
**BRAF Mutant Solid Tumors**

- BGB-3245 is a novel BRAF inhibitor being advanced in collaboration with BeiGene through MapKure, a jointly owned entity
- BGB-3245 could address *BRAF* alterations that currently lack targeted therapeutic options (non-V600 *BRAF* mutations and fusions)
- BGB-3245 has shown preclinical activity against resistance mutations to first generation *BRAF* V600 inhibitors
- Phase 1 trial initiated in 1Q20 and dose escalation ongoing in Australia and the US
- Industry-leading Scientific Advisory Board chaired by Dr. Neal Rosen of Memorial Sloan Kettering Cancer Center

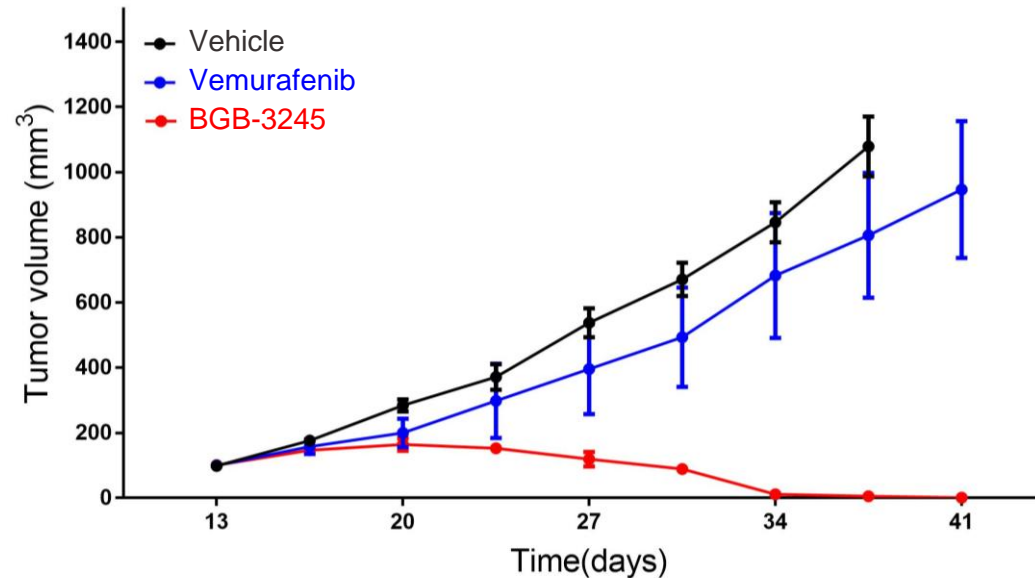
**Phase 1 dose escalation and expansion trial in progress with initial clinical data expected in 2021**

# BGB-3245 Has Demonstrated Encouraging Preclinical Activity

1

BGB-3245 is active in patient-derived xenografts driven by *BRAF* fusions and non-V600 mutations, where approved *BRAF* inhibitors do not work

*BRAF* Fusion PDX: *In Vivo* Tumor Growth Inhibition

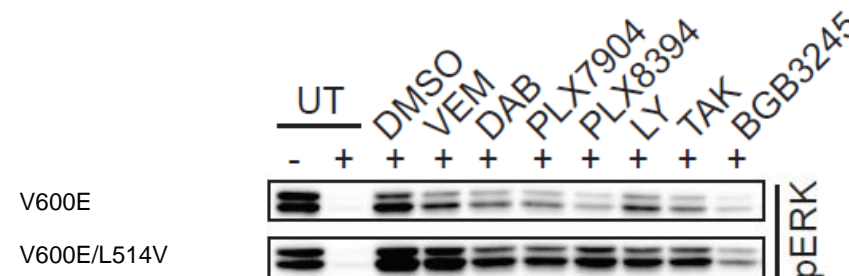


- Driver mutations and fusions potentially uniquely targetable by BGB-3245 could account for up to ~5% of all solid tumors
- BGB-3245 also active preclinically against mutant *BRAF* monomers (e.g., V600)

2

BGB-3245 is active against resistance mutations that arise in *BRAF* V600 patients treated with approved *BRAF* inhibitors

pERK Activity in *BRAF* V600E/L514V Cell Line

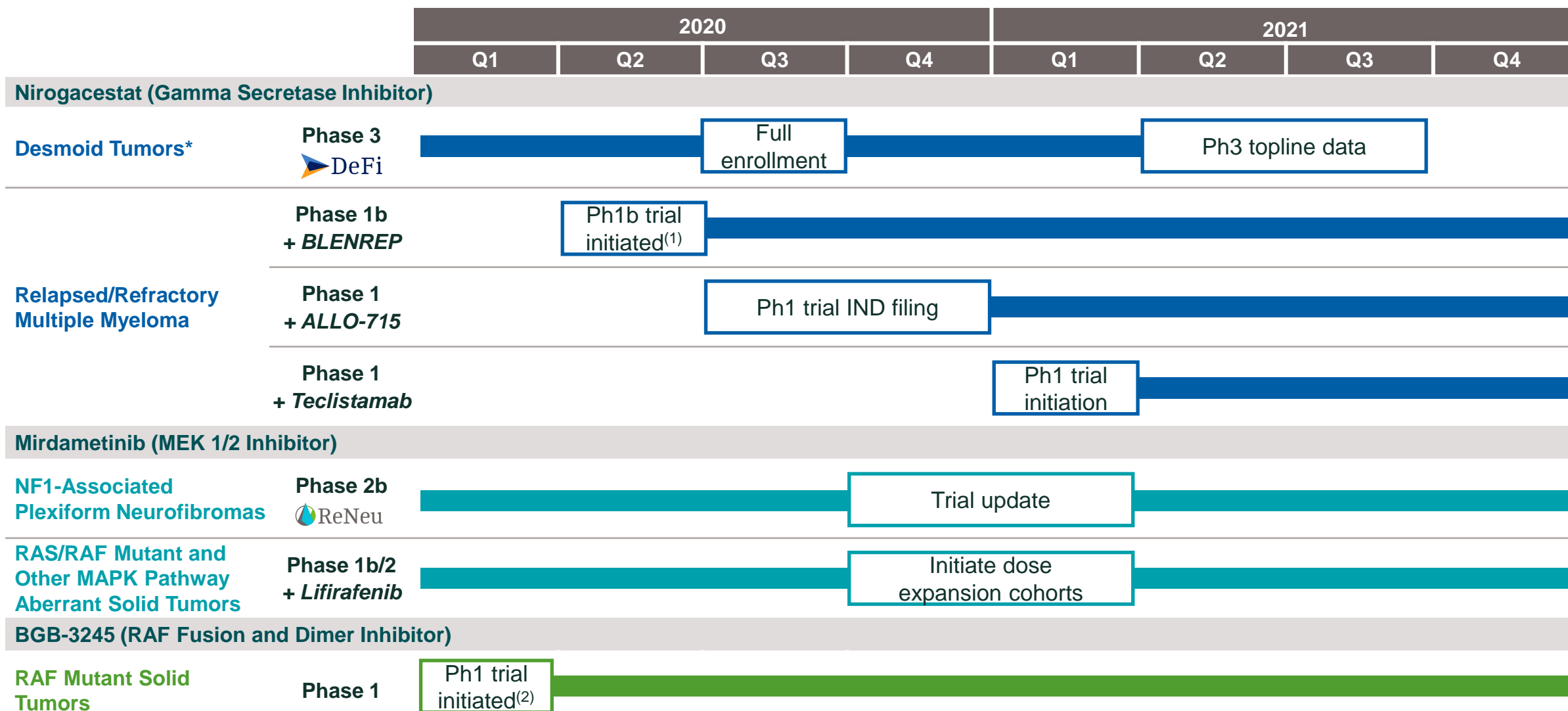


- BRAF* V600E/L514V is a dabrafenib resistance mutation
- BGB-3245 showed strongest *in vitro* activity versus other first- and second-generation *BRAF* inhibitors tested

# The SpringWorks Opportunity



# Pipeline is Rich in Anticipated Near-Term Catalysts



(1) Phase 1b clinical trial evaluating BLENREP (belantamab mafodotin) in combination with nirogacestat in patients with relapsed or refractory multiple myeloma is being examined as a sub-study in GlaxoSmithKline's DREAMM-5 platform trial.

(2) Program being developed by MapKure, LLC, jointly owned by SpringWorks and BeiGene.



# Well Capitalized to Execute on Important Value-Driving Milestones

**\$291.2M**

**Cash, Cash Equivalents  
& Marketable Securities**  
(as of June 30, 2020)

**No Debt**

**NASDAQ: SWTX**

**43.0M**

**Common Shares Outstanding<sup>(1)</sup>**

**Current cash position expected to fund operations through 2022,  
supporting completion of seven ongoing and planned clinical trials**

# Strategic Priorities and Building Blocks for Substantial Value Recognition in 2020

 Execute **two ongoing potentially registrational trials** in rare oncology indications

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 Develop nirogacestat as a **cornerstone of BCMA combinations**

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 Continue disciplined investments in **high-value early pipeline programs**

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 Drive **portfolio expansion** through additional in-licenses and clinical collaborations

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 Expand capabilities and **scale the organization** with talented employees

**7** programs in active clinical development

**2** potentially registrational trials in progress

**5** collaborations in large cancer indications





Thank You