DEFEATING CANCER:

The Challenge.
Our Mission.

July 2019

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CORPORATE PRESENTATION

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Addressing Unmet Needs for Patients

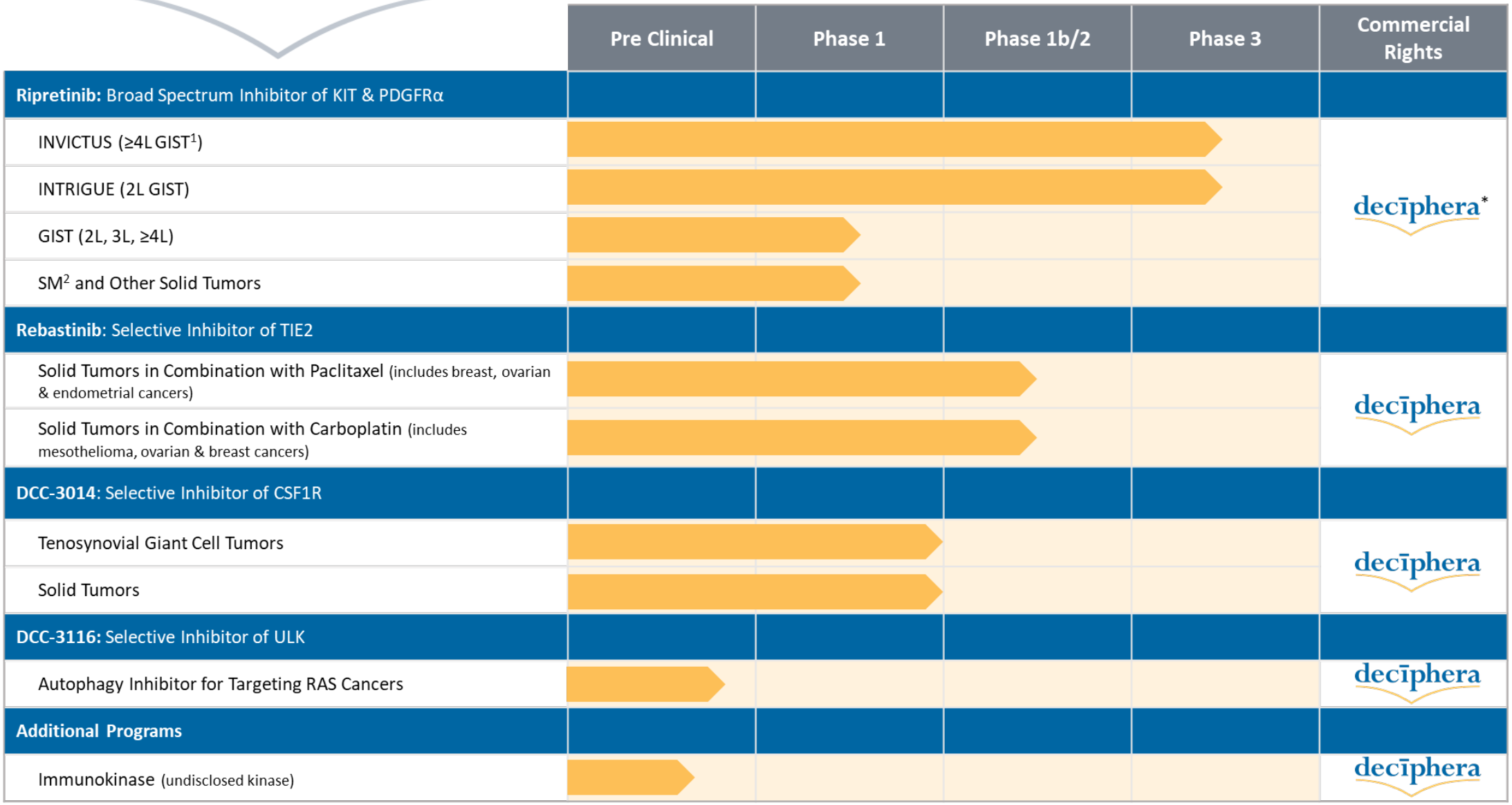
THREE CLINICAL-STAGE PROGRAMS

TWO RANDOMIZED PHASE 3 TRIALS ONGOING

COMMERCIAL PREPARATIONS UNDERWAY TO SUPPORT FIRST POTENTIAL APPROVAL

Proprietary Kinase Switch Control Inhibitor Platform

Strong Clinical Stage Oncology Pipeline of Novel Kinase Inhibitors



*Notes: (1) GIST = gastrointestinal stromal tumors; (2) SM = systemic mastocytosis; *Development and commercialization partnership with Zai Labs in Greater China*

Ripretinib: Designed to Address Relevant Mutations in GIST

Highly potent small molecule KIT and PDGFRα inhibitor

- Designed to inhibit the full spectrum of known KIT and PDGFRα mutations

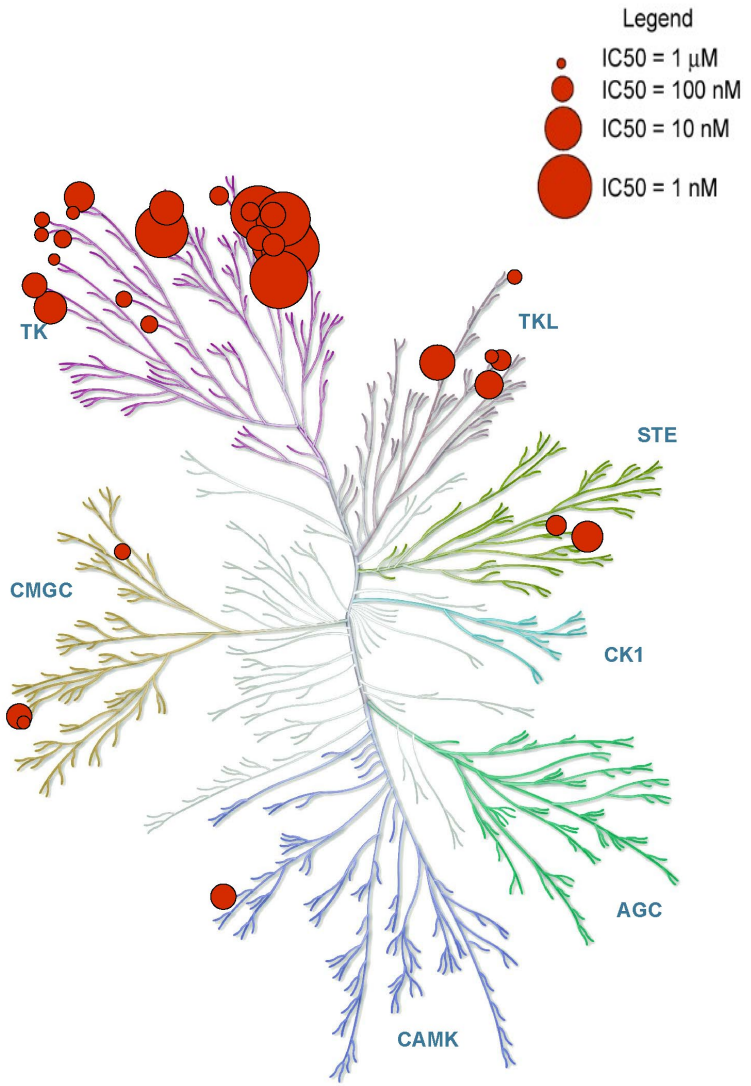
Two Randomized Phase 3 Clinical Trials Ongoing

- INVICTUS: Placebo-controlled, pivotal trial in ≥4th line GIST
  - PFS primary endpoint
  - Top-line data read-out expected mid-2019
- INTRIGUE: Compared to sunitinib, pivotal trial in 2nd line GIST
  - PFS primary endpoint
  - Initiated December 2018
- Phase 1 expansion study ongoing

Clinical proof-of-concept demonstrated in 178 GIST patients in Phase 1

Favorable tolerability profile

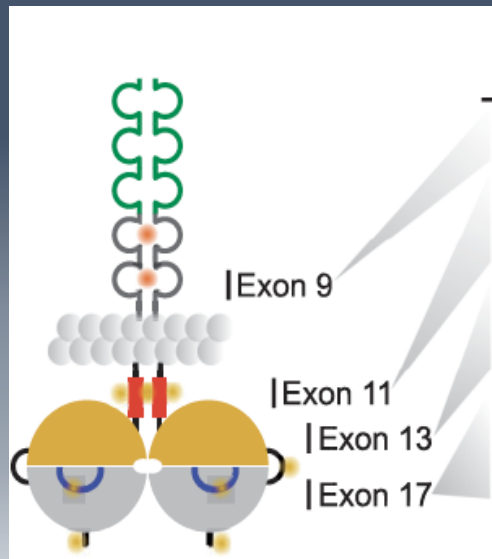
IP: Composition and method of use (2032)



Source and notes: Smith et al. Cancer Cell 35, 738-751, May 13, 2019; Composite of enzyme and cellular kinase phosphorylation data was used. The size of the red circle corresponds to the IC_{50} value obtained. No circles are plotted for kinases with IC_{50} > 1 µM; Illustration reproduced courtesy of Cell Signaling Technology, Inc. (www.cellsignal.com).

Mutations in KIT Drive ~80% of GIST

- Majority of patients with KIT primary mutations respond to 1st line imatinib
  - Resistance develops most commonly due to secondary mutations in KIT
- Approved 2nd and 3rd line agents (sunitinib and regorafenib) confer modest clinical benefit compared to imatinib
  - Multiple drug-resistant mutations often arise in individual tumors
- Unmet medical need for agents that can address relevant primary/secondary KIT mutations across all lines of therapy



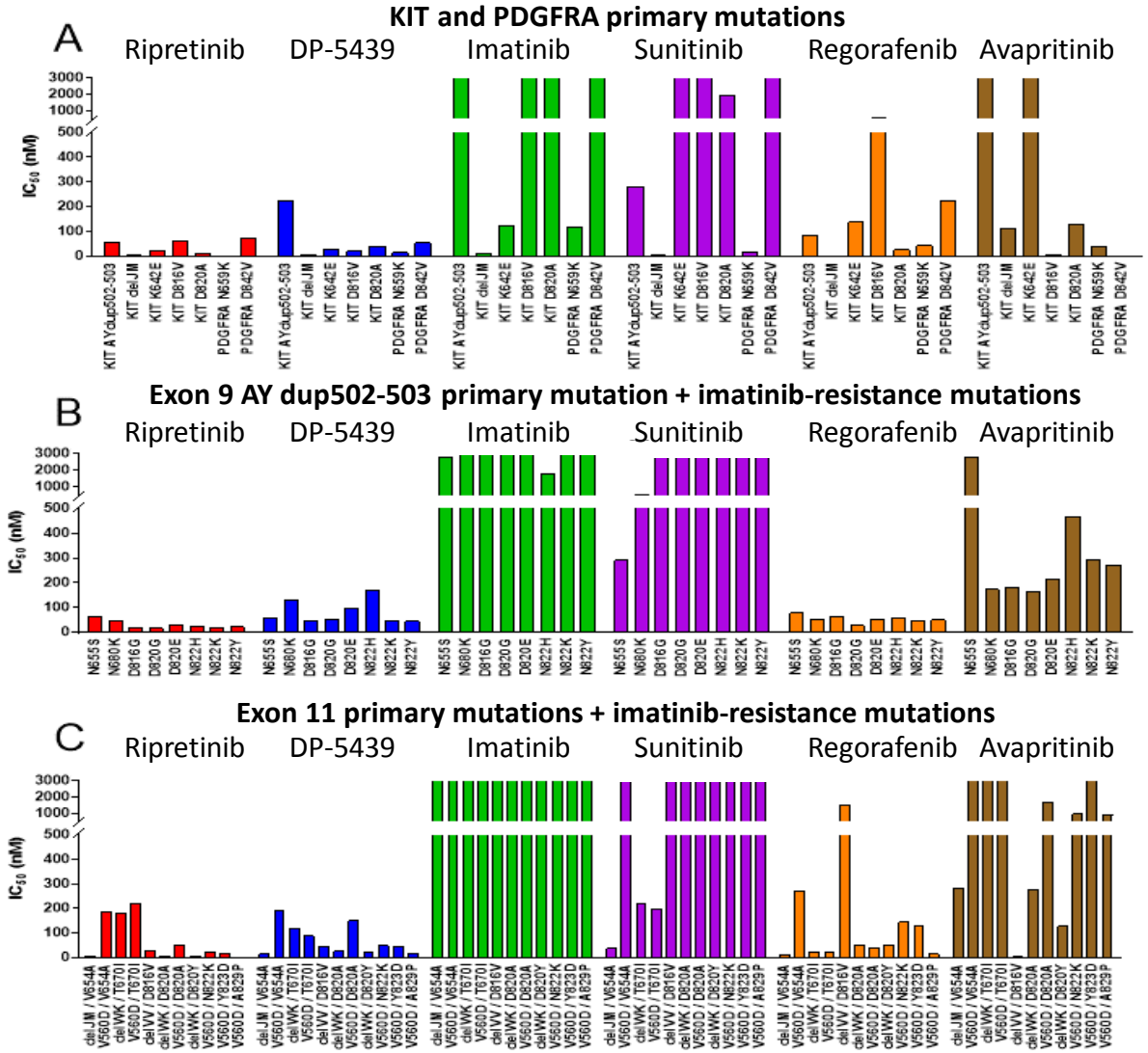
Domain	Gene	1° Mutation Frequency	2° Mutation Frequency
D5	*KIT*	10%	
JM	*KIT*	67	
	PDGFRA	1	
TK1	*KIT*	1	56
	PDGFRA	1	
A-Loop	*KIT*	1	41
	PDGFRA D842V	5	
	PDGFRA	1	3

Source: Hemming M et al. Translational insights into gastrointestinal stromal tumors and current clinical advances. Annals of Oncology; 0:1-9, 2018.

Ripretinib: Broad Mutational Coverage in KIT and PDGFRα (In Vitro Data)

- Broadly inhibits relevant GIST mutations
  - KIT mutations: Exons 9, 11, 13, 14, 17, 18
  - PDGFRA mutation: Exon 18
- Type I inhibitors exhibit weak activity in relevant GIST mutations
  - Primary KIT mutations: Exon 9, exon 11 V560D and exon 13 K642E
  - Secondary KIT mutations: Exon 13 and exon 14

Ripretinib Inhibits Phosphorylation of KIT and PDGFRα in Cellular Assays



Notes: (A) GIST primary mutations; or imatinib-resistant KIT mutations with (B) exon 9 or (C) exon 11 primary mutations.

Source: AACR 2018

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Approved Therapies for GIST: Clinical Goal is Disease Control

	First Line	Second Line	Third Line
	imatinib (n=147) (Blanke et al. 2008)	sunitinib (n=243) (Demetri et al. 2012)	regorafenib (n=133) (Demetri et al. 2013)
Progression Free Survival[1] (weeks)	104.0	26.6	20.8
Objective Response Rate (%)	68.1%	7.0%	4.5%
Stable Disease (%)	15.6%	53.0%	48.1%
Disease Control Rate ("DCR") (%)	**83.7%[2]**	**60.0%[2]**	**52.6%[3]**

No approved therapy for 4th line patients

Notes: (1) Includes progression free survival and time to progression converted to weeks; (2) Time point not disclosed; (3) Time point at 12 weeks.

Ripretinib: Phase 1 Trial Summary

Part 1: Dose Escalation

- Key Objectives: MTD, recommended Phase 2 dose, safety, tolerability, pharmacokinetics and anti-tumor activity
- Design: 3+3 design with enrichment of targeted patients
- Dose Levels: 20, 30, 50, 100, 150, and 200 mg BID; and 100, 150 and 250 mg QD
- MTD: not determined

Advanced Malignancies (n=68) → **Recommended Dose 150 mg QD(1)**

Part 2: Dose Expansion

- **10 cohorts up to 270 pts; 4 cohorts fully enrolled**

Enrollment Complete

- 2nd – 3rd Line GIST n=55
- 4th Line GIST n=40
- >4th Line GIST n=35
- Other Solid Tumors n=10

Enrollment Continuing

- Systemic Mastocytosis
- Melanomas
- Malignant Gliomas
- Soft Tissue Sarcomas
- NSCLC, Germ Cell & Penile
- Renal Impairment(2)

Notes: (1) 150 mg BID dosing also available for dose escalation for GIST patients and for SM patients; (2) Includes patients with GIST and other solid tumors with renal impairment.

Ripretinib: Phase 1 Demography and Baseline Characteristics

ESMO 2018				
GIST Patients at >100 mg/d	**2nd Line (n=38)**	**3rd Line (n=29)**	**≥ 4th Line (n=111)(4)**	**Total (n=178)**
Age Median (min, max)	60 (32, 80)	64 (48, 82)	60 (27, 87)	61 (27, 87)
Primary Mutation(1) n (%)				
KIT Exon 9	4 (11%)	8 (28%)	22 (20%)	34 (19%)
KIT Exon 11	31 (82%)	20 (69%)	71 (64%)	122 (69%)
Other KIT(2)	0 (0%)	1 (3%)	12 (11%)(3)	13 (7%)(3)
PDGFRα	3 (8%)	0 (%)	6 (5%)	9 (5%)
Pts at RP2D(5) (150 mg QD)	32 (84%)	27 (93%)	83 (75%)	142 (80%)

Notes: (1) Primary mutation per local assessment; (2) KIT exon 13 (4), KIT exon 17 (5), not done (3); (3) Includes one SDH deficient patient; (4) Mean # of prior therapies is 4.63 (range 4-7); (5) RP2D = Recommended Phase 2 Dose.

Ripretinib: Preliminary Phase 1 Results Provided Encouraging Efficacy Measures Across All Lines of Treatment >100 mg/d (n=178)

Line of Therapy	Objective Response Rate[1][2]	Disease Control Rate @ 3 Months[1]	Median Progression Free Survival (mPFS)[1]	Censored Patients for mPFS[1]	Median Treatment Duration[5]
2nd Line (n=38)	18%[3]	79%	42 weeks	58%	48 weeks
3rd Line (n=29)	24%	83%	40 weeks	52%	NR
≥4th Line (n=111)	9%[4]	66%	24 weeks	35%	28 weeks
2nd & 3rd Line (n=67)	21%[3]	81%	40 weeks	55%	52 weeks

Notes: (1) Based on cut off date of August 31, 2018; RECIST data per investigator assessment; *(2)* ORR by Best Response includes nine unconfirmed responses in 2nd line (n=1), 3rd line (n=3) and ≥4th line (n=5)*; (3)* Does not reflect one PR reported after cut off date; *(4)* Excludes five patients due to missing data at the time of data cut off (n=2), lack of first tumor assessment (n=1), withdrawal of consent prior to first assessment (n=1) and unrelated death at C1D4 prior to first assessment (n=1)*; (5) Includes 14 patients who elected for intra-patient dose escalation.*

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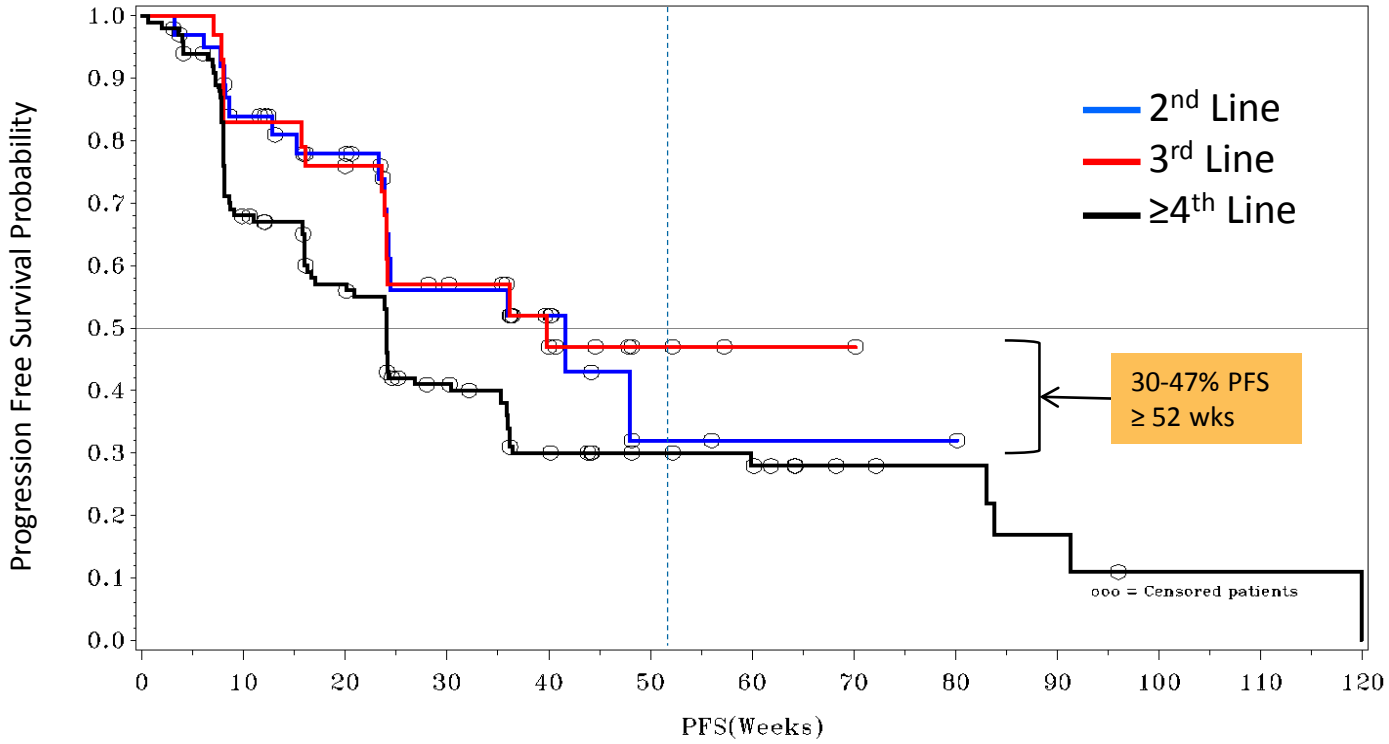
Ripretinib Phase 1 Data Demonstrated Prolonged Progression Free Survival in a Meaningful Subset of Patients Across All Lines of Treatment

Tumor Control per RECIST(1)
KIT & PDGFRα @ ≥ 100 mg/d (n=178)



2nd Line	**38**	**30**	**21**	**13**	**8**	**2**	**1**	**1**	**1**	**0**	**0**	**0**	**0**
3rd Line	**29**	**24**	**21**	**14**	**8**	**4**	**1**	**1**	**0**	**0**	**0**	**0**	**0**
≥4th Line	**111**	**71**	**53**	**32**	**20**	**14**	**12**	**6**	**5**	**3**	**1**	**1**	**0**

Notes: (1) Based on cutoff date of August 31, 2018; RECIST data per investigator assessment; (2) RECIST data per central review in registration trial; (3) mPFS of 4.8 months converted to weeks.

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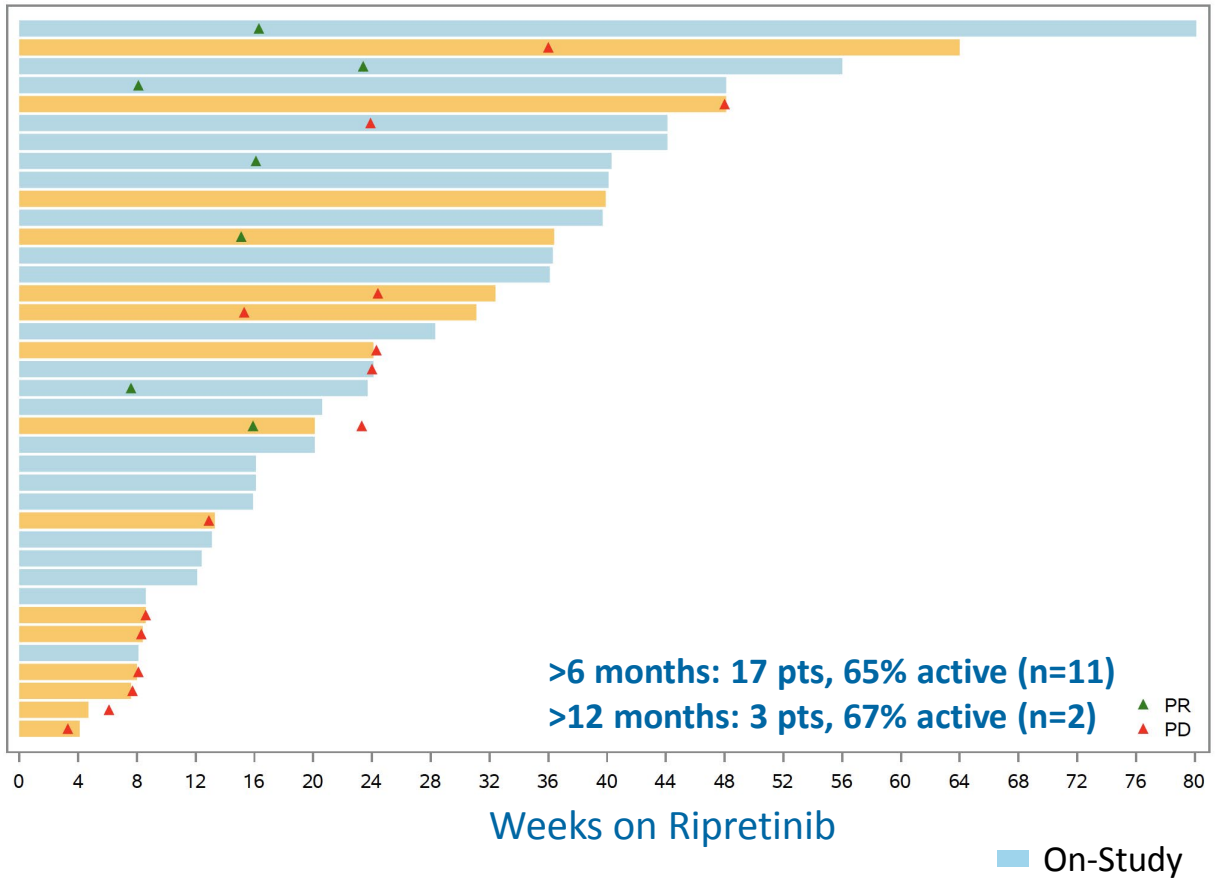
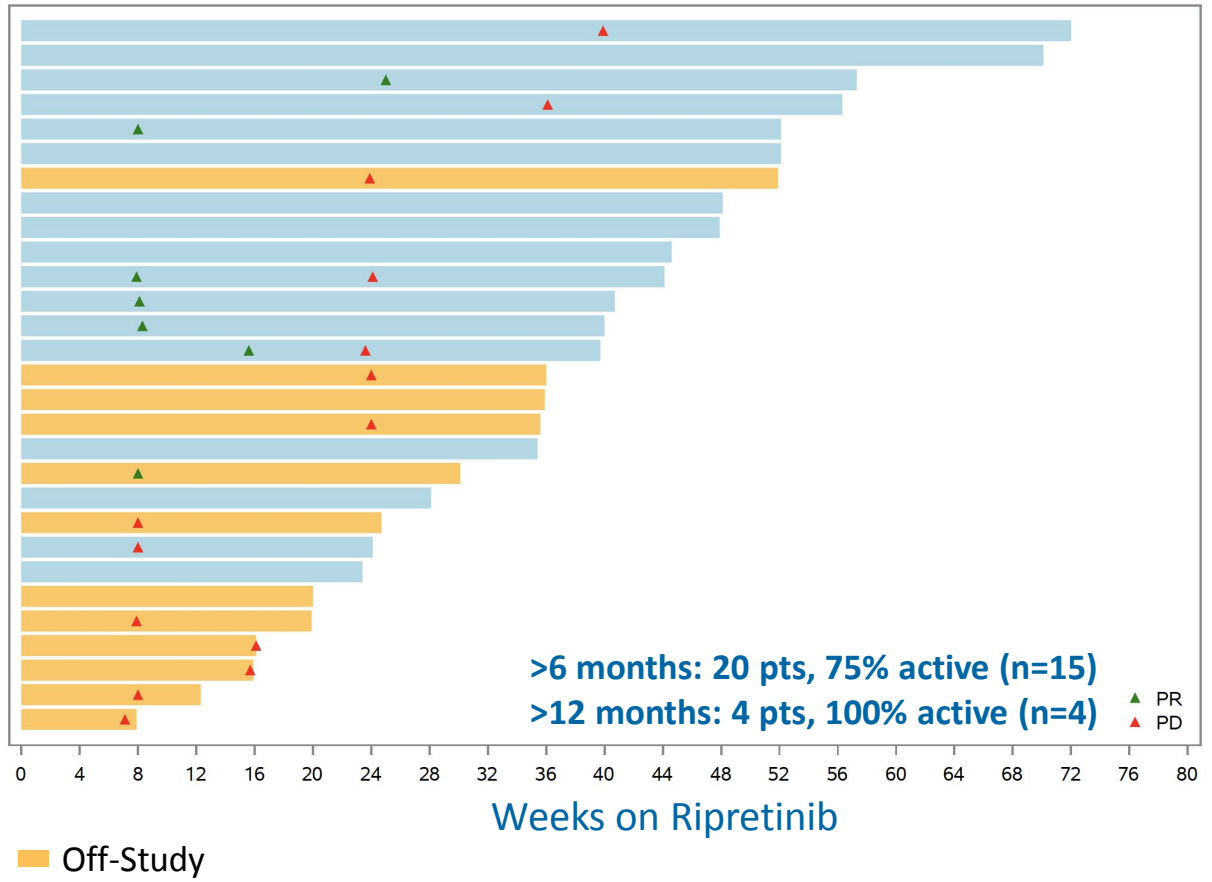
Ripretinib: Generally Well-tolerated in Phase 1 Data; Allowed for Prolonged Treatment Duration in 2nd & 3rd Line GIST Patients at ≥100 mg/d (n=67)

2nd Line KIT and PDGFRα Patients (n=38)(1)(2)



3rd Line KIT and PDGFRα Patients (n=29)(1)(2)



On-Study Off-Study

Notes: (1) Based on cutoff date of August 31, 2018; RECIST data per investigator assessment; (2) Includes unconfirmed responses in 2nd line (n=1) and 3rd line (n=3). Does not reflect one PR in 2nd line reported after cutoff date. Includes 14 patients who elected for intra-patient dose escalation.

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Ripretinib: Favorable Tolerability Profile @ ≥100mg Daily

Treatment-emergent Adverse Events (TEAE) in >10% GIST Patients (n=178) @ ≥100 mg Daily

- 14% (24 of 178) patients experienced dose reductions due to TEAEs
- 11% (19 of 178) patients experienced treatment discontinuations due to TEAEs
- Clinically asymptomatic lipase elevations most frequent G3 TEAE

GIST PATIENTS @ ≥100 MG DAILY			
ADVERSE EVENT	GRADE 1-2 (N=178)	GRADE 3-4 (N=178)	GRADE 1-4 TOTAL (N=178)
Alopecia	89 (50%)	0 (0%)	89 (50%)
Myalgia	79 (44%)	0 (0%)	79 (44%)
Fatigue	74 (42%)	2 (1%)	76 (43%)
Constipation	60 (34%)	0 (0%)	60 (34%)
Hand-Foot Skin Reaction[1]	56 (32%)	1 (1%)	57 (32%)
Nausea	53 (30%)	0 (0%)	53 (30%)
Decreased appetite	47 (26%)	2 (1%)	49 (28%)
Weight decreased	43 (24%)	0 (0%)	43 (24%)
Abdominal pain	33 (19%)	8 (5%)	41 (23%)
Diarrhea	38 (21%)	3 (2%)	41 (23%)
Lipase increased	21 (12%)	20 (11%)	41 (23%)
Vomiting	32 (18%)	1 (1%)	33 (19%)
Arthralgia	32 (18%)	0 (0%)	32 (18%)
Hypertension	22 (12%)	10 (6%)	32 (18%)
Dry skin	31 (17%)	0 (0%)	31 (17%)
Rash	31 (17%)	0 (0%)	31 (17%)
Muscle spasms	30 (17%)	0 (0%)	30 (17%)
Anemia	14 (8%)	13 (7%)	27 (15%)
Dyspnea	25 (14%)	2 (1%)	27 (15%)
Cough	26 (15%)	0 (0%)	26 (15%)
Headache	25 (14%)	0 (0%)	25 (14%)
Dizziness	23 (13%)	0 (0%)	23 (13%)
Back pain	20 (11%)	2 (1%)	22 (12%)
Blood bilirubin increased	15 (8%)	6 (3%)	21 (12%)
Pain in extremity	21 (12%)	0 (0%)	21 (12%)
Dysgeusia	18 (10%)	0 (0%)	18 (10%)
Hypomagnesaemia	18 (10%)	0 (0%)	18 (10%)
Pruritus	18 (10%)	0 (0%)	18 (10%)

Notes: (1) Palmar-plantar erythrodysaesthesia syndrome reported in 19 patients; (2) Data presented at ESMO on October 19, 2018 based on cutoff as of August 31, 2018.

Ripretinib: INVICTUS Pivotal Phase 3 Top-line Data Expected Mid-2019

Global Pivotal Phase 3 GIST Programs

invictus

3 prior lines of therapy[1]

2:1 Randomization
Double Blind
n=129

- Ripretinib 150 mg QD
- Placebo N/A

Primary Endpoint for Approval = PFS

Following progression: (a) placebo patients can crossover to ripretinib and (b) ripretinib patients can continue on treatment or escalate to 150 mg BID

intrigue

Prior imatinib therapy[2]

1:1 Randomization
Open Label
n=358

- Ripretinib 150 mg QD n=179
- Sunitinib 50 mg QD n=179

Primary Endpoint for Approval = PFS

No cross-over option

Notes: (1) Phase 3 pivotal study in ≥4th line patients who previously received at least imatinib, sunitinib, and regorafenib; (2) Phase 3 pivotal study in 2nd line patients who previously received imatinib.

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Ripretinib: Estimated GIST Market Opportunity: US, EU & Japan

	US	EU & Japan	Total (exc. ROW)
GIST KIT 4th Line[1&2]	~2,100	~4,100	~6,200
GIST KIT 2nd Line[1&2]	~2,600	~5,000	~7,600
GIST PDGFRα[1&2]	~400	~760	~1,160

Estimated Annual Incidence of New Patients by Indication

Sources: Internal Deciphera estimates based on applying epidemiology data reported in the following publications to population estimates for US, EU (28) and Japan:

[1] Zhao *et al.* J Gastrointest Oncol 2012;3(3):189-208

[2] Metaxas Y, *et al*. ESMO Open 2016

Expanding Clinical Stage Portfolio

Rebastinib

- Two ongoing Phase 1b/2 trials
- Data from Part 1 of the Phase 1b/2 paclitaxel study expected 2H 2019

DCC-3014

- Phase 1 trial ongoing
- TGCT(1) expansion cohort planned in 2H 2019
- Phase 1 escalation data update expected 2H 2019

DCC-3116

- Potential first-in-class ULK kinase inhibitor for autophagy inhibition
- Targeting mutant RAS cancers
- IND filing expected mid-2020

Note: (1) TGCT = tenosynovial giant cell tumors

Rebastinib: A Highly Potent and Selective TIE2 Inhibitor

Potent, small molecule inhibitor of TIE2

Preclinical anti-tumor activity

- Single agent and I/O or chemo combination

TIE2 microenvironment mechanisms

- Tumor vascularization, dissemination, metastasis, immunotolerance

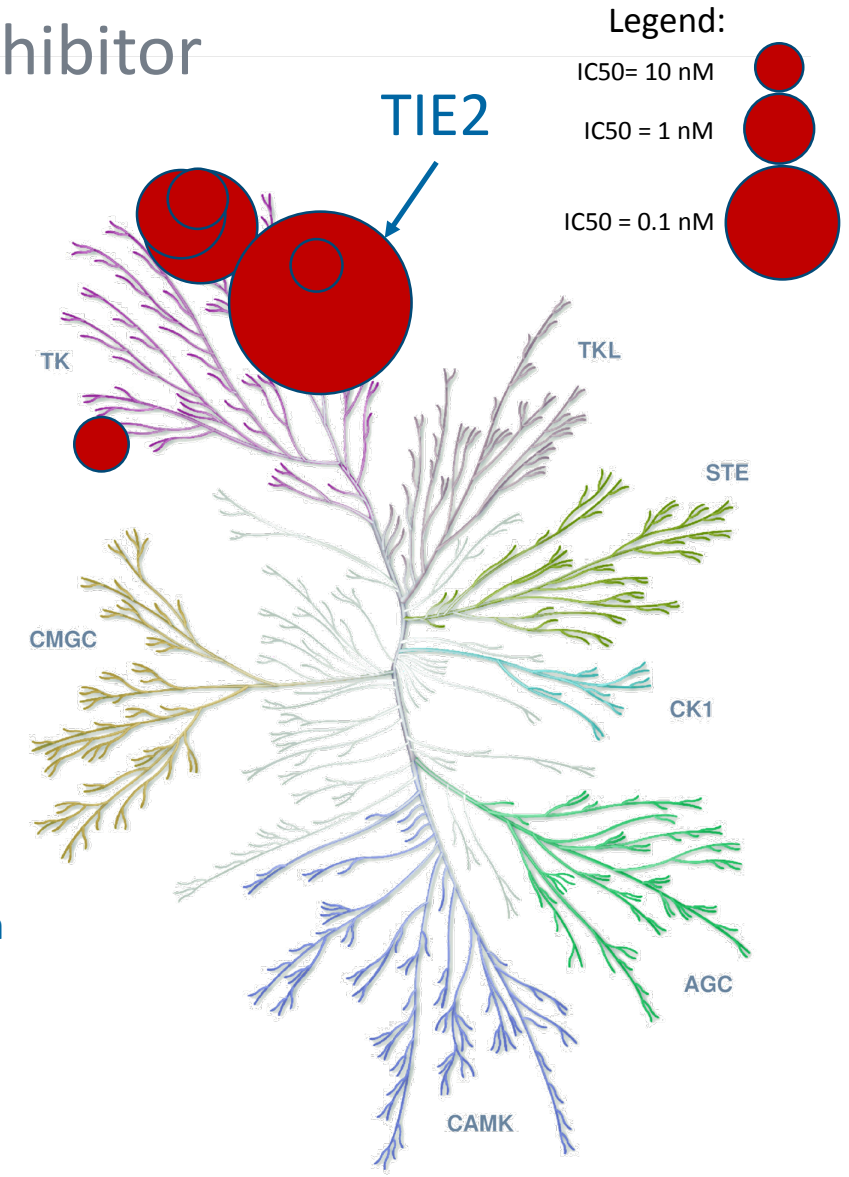
Phase 1 study completed

- Identified 150 mg BID dose as maximum tolerated dose

Development Status

- Two ongoing company-sponsored chemo combo trials with paclitaxel and carboplatin
- Data from Part 1 of the Phase 1b/2 study with paclitaxel expected 2H 2019

IP: Composition (2027) and method of use (2034)



Notes: All kinases within 100-fold of TIE2 IC_{50} (0.058 nM) are shown. Includes enzyme data at low ATP and 4 mM ATP, and cellular data; Illustration reproduced courtesy of Cell Signaling Technology, Inc. (www.cellsignal.com).

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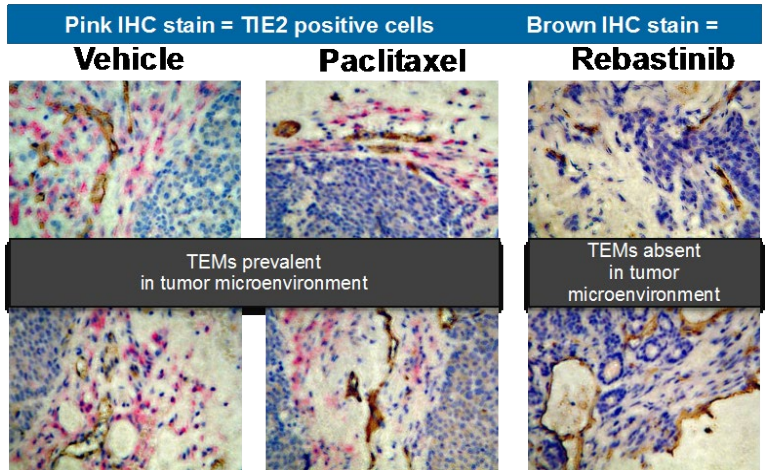
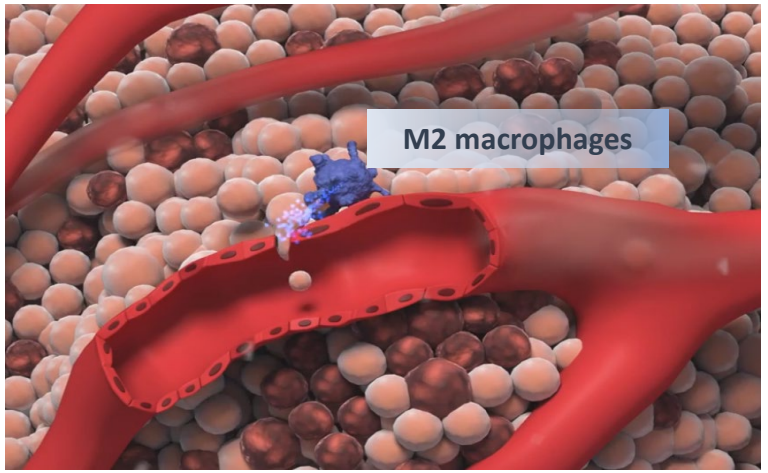
Rebastinib: Potential Benefits in Combination with Chemotherapy

Rebastinib targets TIE2 expressing macrophages (TEMs) and endothelial cells in tumor environment

Combination of rebastinib with chemotherapy may increase tumor killing through multiple mechanisms



- Chemotherapy leads to recruitment of pro-tumoral M2 macrophages from the bone marrow
- Rebastinib is designed to inhibit chemotherapy-induced recruitment of M2 macrophages to tumors



- M2 macrophages serve as pumps for tumor cell intravasation and metastasis
- Rebastinib is designed to block rebound angiogenesis and inhibit M2 macrophages

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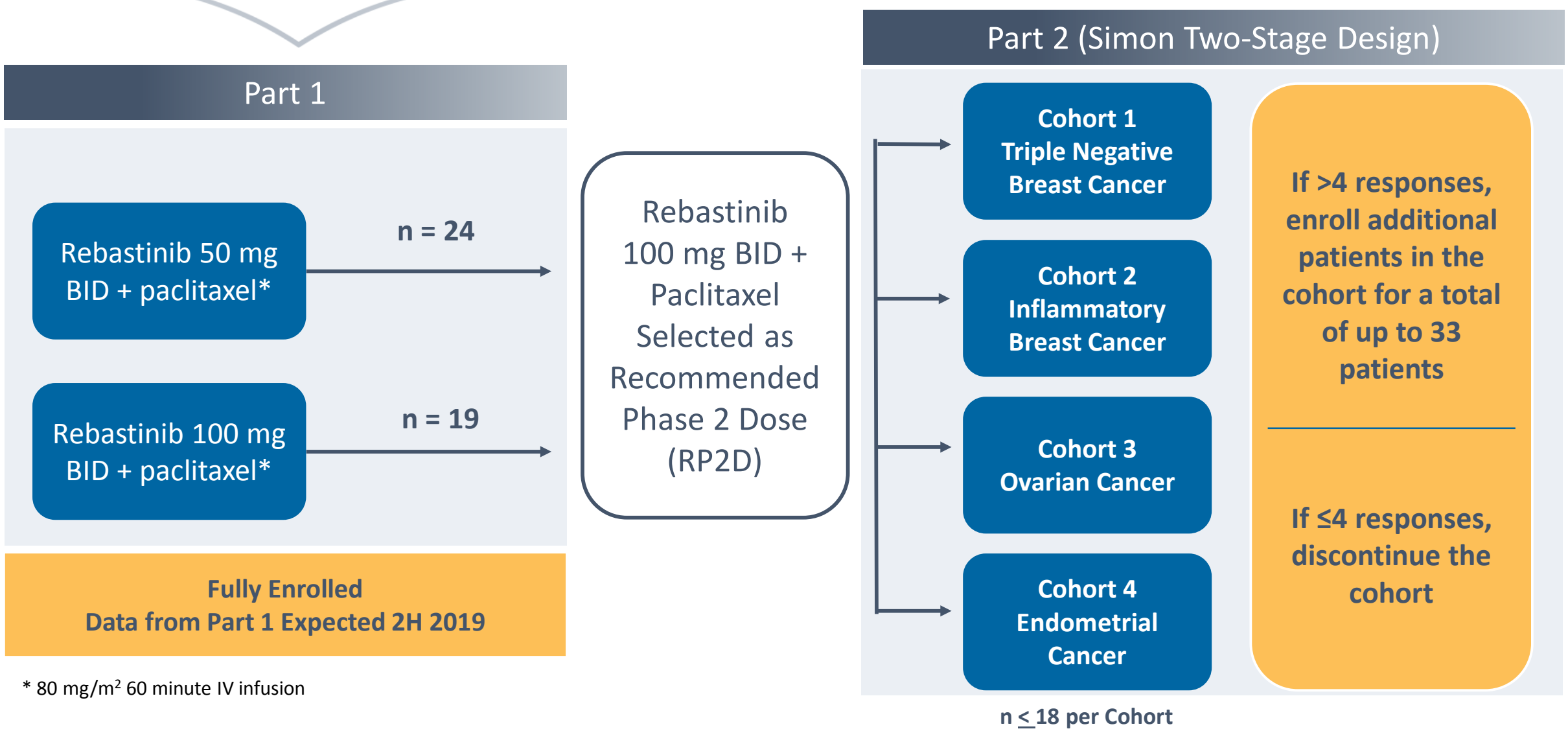
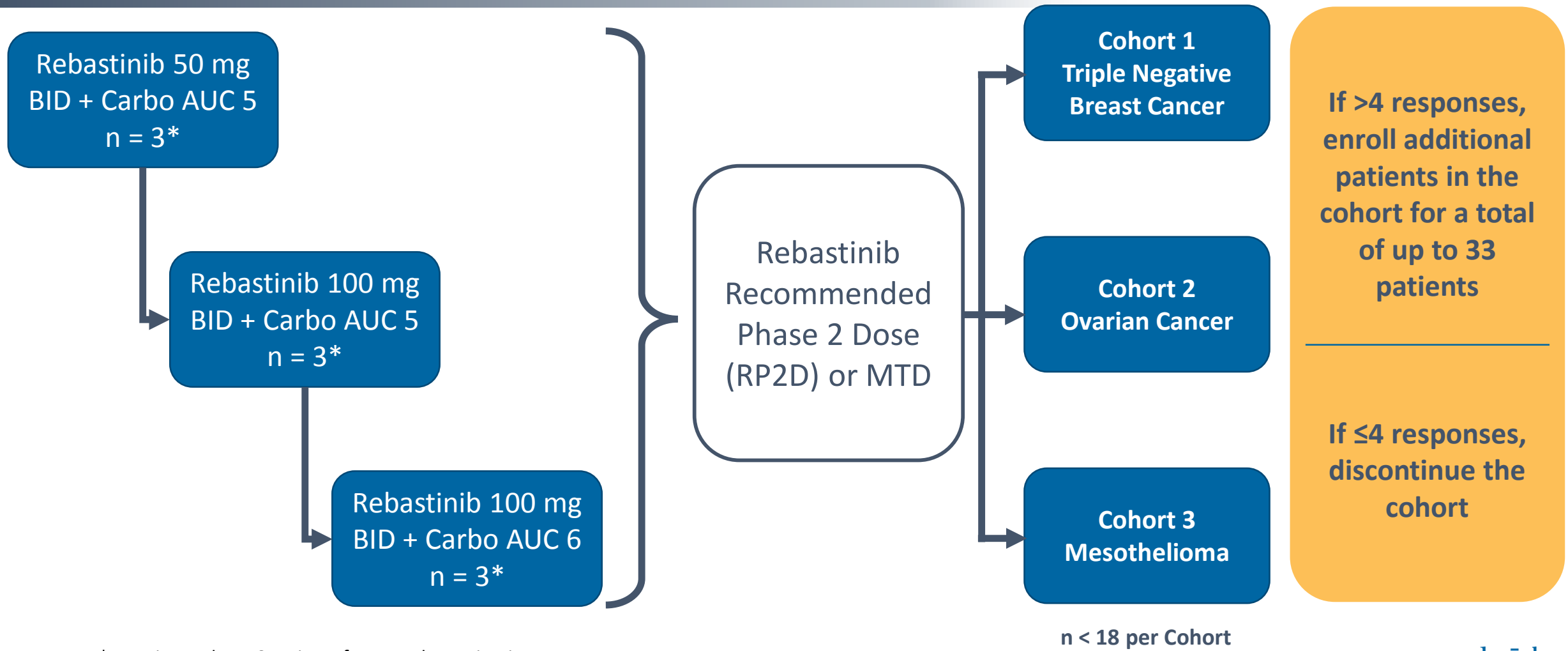
Rebastinib: Phase 1b/2 Study Combination with Paclitaxel



* 80 mg/m^2 60 minute IV infusion

Rebastinib Phase 1b/2 Study Combination with Carboplatin

Simon Two-Stage Design Applied at MTD or RP2D



* Requires at least 6 patients for MTD determination

DCC-3014: A Highly Selective and Potent CSF1R Inhibitor

Phase 1 escalation trial ongoing, with data update in 2H 2019

Mechanistic Proof of Concept (mPoC)[(1)] Achieved

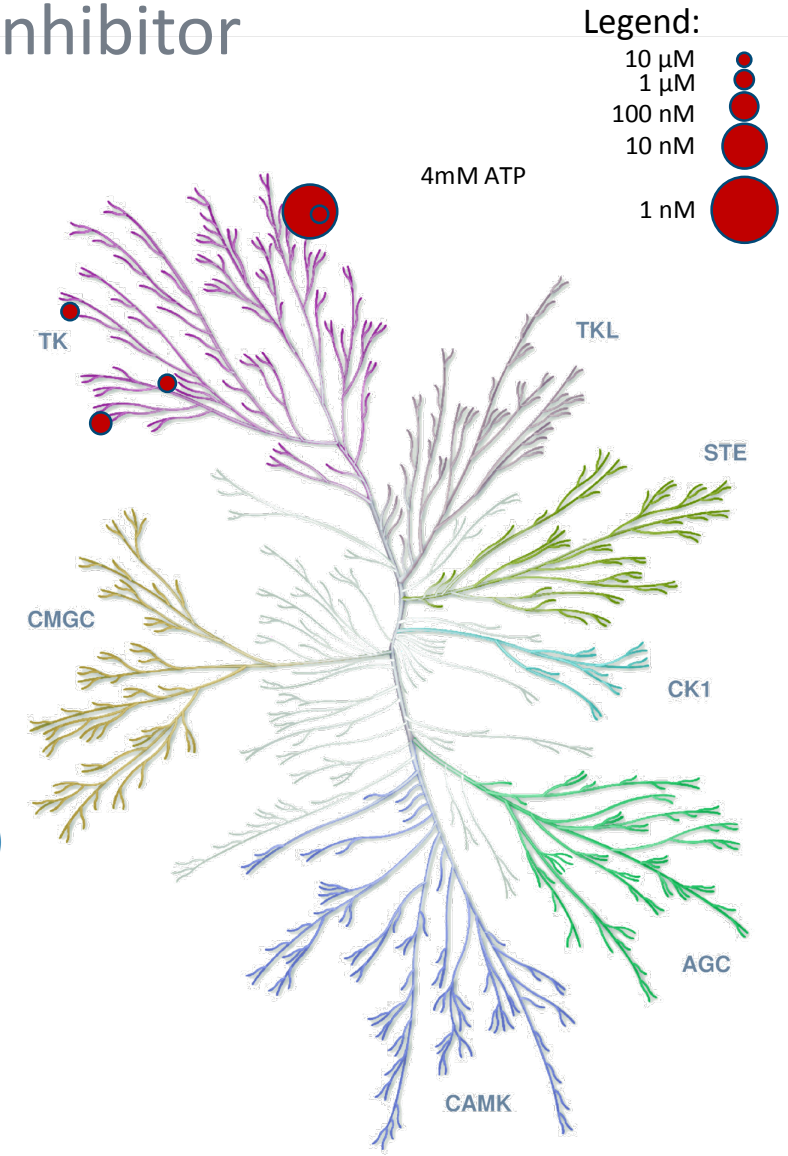
- Material reductions in circulating CSF1R+ macrophages

Generally well-tolerated at doses of up to 30mg in patients receiving five-day loading, followed by twice-weekly maintenance regimen

- No DLTs in loading and maintenance regimens
- No DCC-3014 related G3/4 TEAEs in ≥ 10% patients
- PK analysis demonstrated dose-proportional exposure for loading and maintenance regimen

Study expanded to include patients with tenosynovial giant cell tumors (TGCT)

IP: Composition and method of use (2034)



Notes: (1) Results from preliminary Phase 1 data reported in January 2019. Composite of enzyme and cellular kinase phosphorylation data was used. The size of the red circle corresponds to the IC_{50} value obtained. No circles are plotted for kinases with IC_{50} > 3 µM. Illustration reproduced courtesy of Cell Signaling Technology, Inc. (www.cellsignal.com).

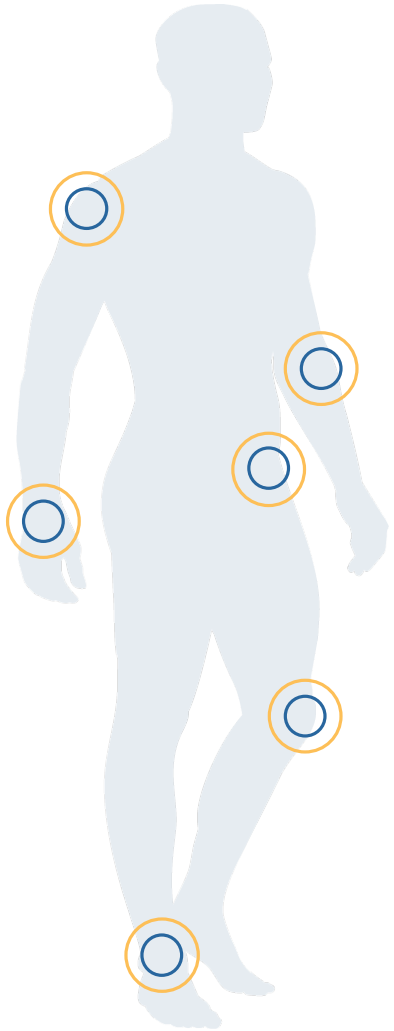
Single Agent Opportunity in Tenosynovial Giant Cell Tumor (TGCT)

Overview

- Rare, benign tumors involving the synovium, bursae and/or tendon sheath that damage surrounding tissues inducing pain, swelling, limitation of movement of the joint and cause severe disability
- Genetic translocation causes overproduction of CSF-1, the ligand for the CSF1R receptor, triggering migration of inflammatory cells to tumor sites

Two Types of TGCT

1. **Localized TGCT**
   - Affects knee, wrist and ankle
2. **Diffuse TGCT (also known as PVNS)**
   - Mostly commonly affects the knee, as well as hip, ankle, elbow and shoulder



Unmet Medical Need

- Surgical resection is standard treatment but with a high rate of recurrence in diffuse TGCT
- CSF1R inhibition has demonstrated clinical benefit in diffuse TGCT patients, but no currently approved systemic therapies
- Pexidartinib
  - 39% ORR VS. 0% for placebo in Phase 3 (n=120)
  - Hepatotoxicity concerns (off-target) may require a REMS and registry if approved
- Unmet medical need for effective CSF1R inhibitor with favorable safety/tolerability profile for diffuse TGCT patients

Sources: Mastboomet al. ActaOrthop. 2017; 88(6):688-694; Staals et al. Eur j Cancer. 2016; 63: 34-40.

DCC-3116: A Potential First-in-Class ULK Inhibitor Designed to Inhibit Autophagy

Highly Potent (IC50 at 1 mM ATP)

- ULK1 4.7 nM
- ULK2 35 nM

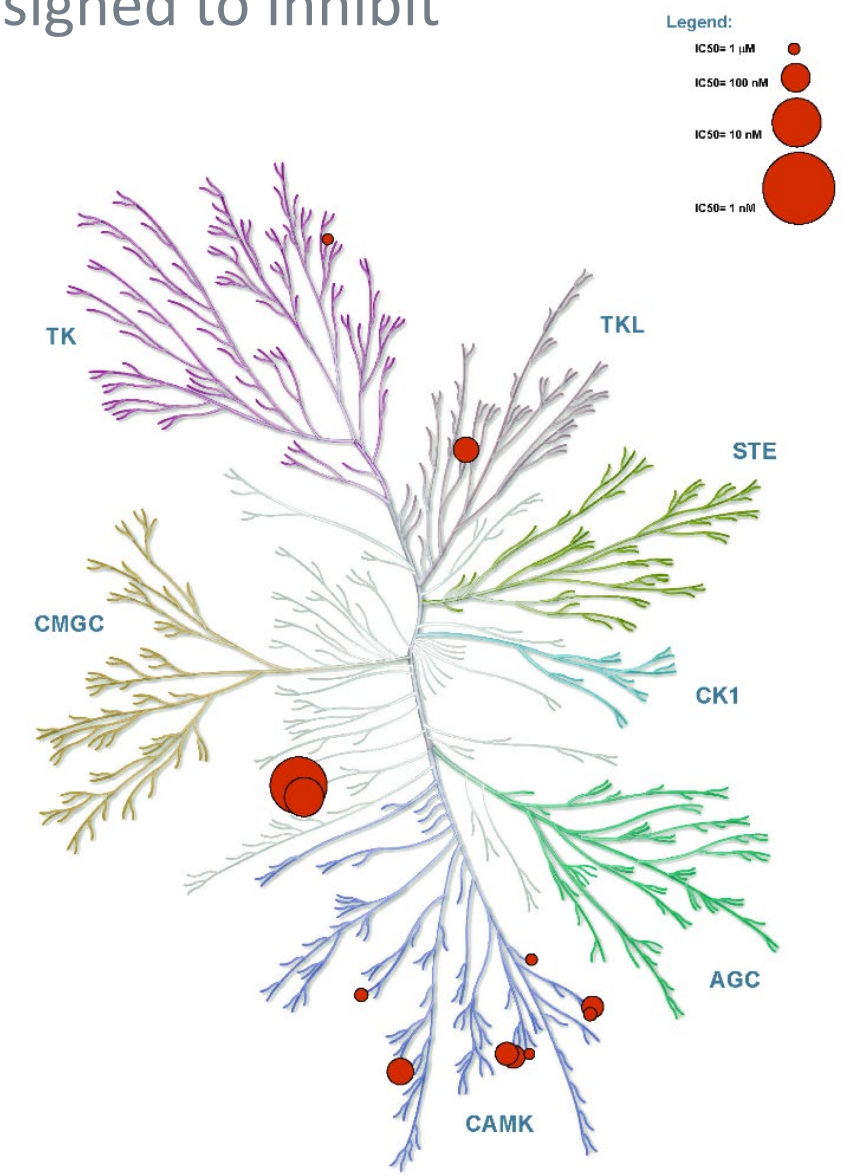
Highly Selective

- No off-target kinases within 30-fold of ULK1
- Only 5 kinases within 100-fold of ULK1

Designed to Avoid CNS Exposure

- Low Ratio $Brain_{ff}/Plasma_{ff}$ (4.3%) to avoid CNS autophagy

IND Filing Expected in Mid-2020



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Rationale for DCC-3116 in RAS Cancers

RAS CANCERS DEPEND ON MEK/ERK SIGNALING & AUTOPHAGY FOR SURVIVAL

ULK KINASE IS AN INITIATING FACTOR FOR ACTIVATION OF AUTOPHAGY

DCC-3116 IS A POTENTIAL FIRST-IN-CLASS ULK KINASE INHIBITOR

STRONG PRELIMINARY PRECLINICAL VALIDATION

RAS Cancers Represent Significant Unmet Medical Need

Most Common Activating Cancer Mutations

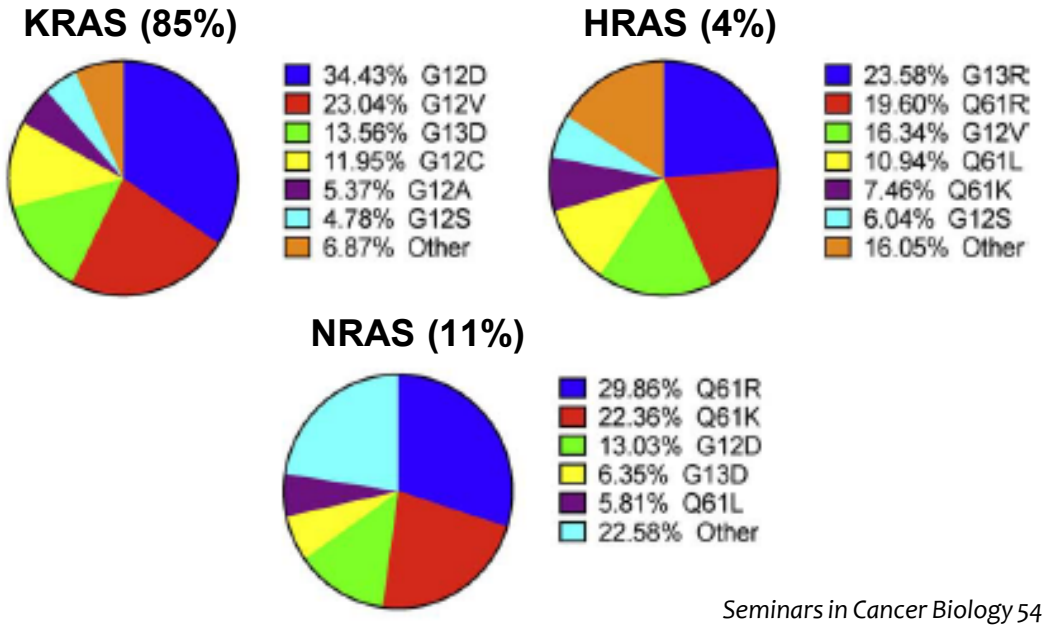
- Pancreatic: ~98%
- Colon: ~ 45%
- Lung: ~ 30%

RAS Activates Other Pathways

- MAPK (RAF-MEK-ERK)
- PI3K/AKT/mTOR
- MAPK inhibitors have not been successful thus far as single agents

Combination Strategy with Autophagy Inhibition

- MAPK (RAF-MEK-ERK) inhibitors
- KRAS G12C small molecule covalent inhibitors
- RAF inhibitors
- Targeted therapies & chemotherapies



Seminars in Cancer Biology 54 (2019) 138–148

Direct Inhibition of RAS: Quest for the Holy Grail?

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RAS Cancers Exhibit Addiction to Autophagy

THREE 2019 PUBLICATIONS INDEPENDENTLY VALIDATE COMBINED INHIBITION OF MAPK & AUTOPHAGY PATHWAYS AS NEW TARGETED APPROACH FOR POTENTIAL IN RAS CANCERS

nature medicine

Letters
https://doi.org/10.1038/s41591-019-0367-9

Protective autophagy elicited by RAF→MEK→ERK inhibition suggests a treatment strategy for RAS-driven cancers

Conan G. Kinsey[1,2], Soledad A. Camolotto[1], Amelie M. Boespflug[1,3,4], Katrin P. Gullien[1], Mona Foth[1], Amanda Truong[1], Sophia S. Schuman[1], Jill E. Shea[5], Michael T. Seipp[5], Jeffrey T. Yap[1,6], Lance D. Burrell[1], David H. Lum[1], Jonathan R. Whisenant[1,2], G. Weldon Gilcrease III[1,2], Courtney C. Cavalieri[1,7], Kaitrin M. Rehbein[1], Stephanie L. Cutler[1], Kajsa E. Affolter[1,8], Alana L. Welm[1,9], Bryan E. Welm[1,5], Courtney L. Scaife[1,5], Eric L. Snyder[1,8] and Martin McMahon[1,10]*

Articles
https://doi.org/10.1038/s41591-019-0368-8

Combination of ERK and autophagy inhibition as a treatment approach for pancreatic cancer

Kirsten L. Bryant[1], Clint A. Stalnecker[1], Daniel Zeitouni[1], Jennifer E. Klomp[1], Sen Peng[2], Andrey P. Tikunov[3], Venugopal Gunda[4], Mariaelena Pierobon[5], Andrew M. Waters[1], Samuel D. George[1], Garima Tomar[1], Björn Papke[1], G. Aaron Hobbs[1], Liang Yan[6], Tikvah K. Hayes[7], J. Nathaniel Diehl[7], Gennifer D. Goode[4], Nina V. Chaika[4], Yingxue Wang[8], Guo-Fang Zhang[8], Agnieszka K. Witkiewicz[9], Erik S. Knudsen[10], Emanuel F. Petricoin III[5], Pankaj K. Singh[4], Jeffrey M. Macdonald[3], Nhan L. Tran[11], Costas A. Lyssiotis[12], Haoqiang Ying[6], Alec C. Kimmelman[13], Adrienne D. Cox[1,14,15] and Channing J. Der[1,7,15]*

PNAS

MAP kinase and autophagy pathways cooperate to maintain RAS mutant cancer cell survival

Chih-Shia Lee[a], Liam C. Lee[a,1], Tina L. Yuan[b,2], Sirisha Chakka[c,3], Christof Fellmann[d,4], Scott W. Lowe[d,e,f], Natasha J. Caplen[c], Frank McCormick[b,g,5], and Ji Luo[a,5]

[a] Laboratory of Cancer Biology and Genetics, Center for Cancer Research, National Cancer Institute, Bethesda, MD 20892; [b] Helen Diller Family Comprehensive Cancer Center, University of California, San Francisco, CA 94158; [c] Genetics Branch, Center for Cancer Research, National Cancer Institute, Bethesda, MD 20892; [d] Cold Spring Harbor Laboratory, Cold Spring Harbor, NY 11724; [e] Howard Hughes Medical Institute, Memorial Sloan Kettering Cancer Center, New York, NY 10065; f Department of Cancer Biology & Genetics, Memorial Sloan Kettering Cancer Center, New York, NY 10065; and Cancer Research Technology Program, Frederick National Laboratory for Cancer Research, Leidos Biomedical Research, Frederick, MD 21702

Edited by Ronald A. DePinho, University of Texas MD Anderson Cancer Center, Houston, TX, and approved December 17, 2018 (received October 18, 2018)

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Strategies for Blocking Autophagy in Cancer

ULK Inhibition

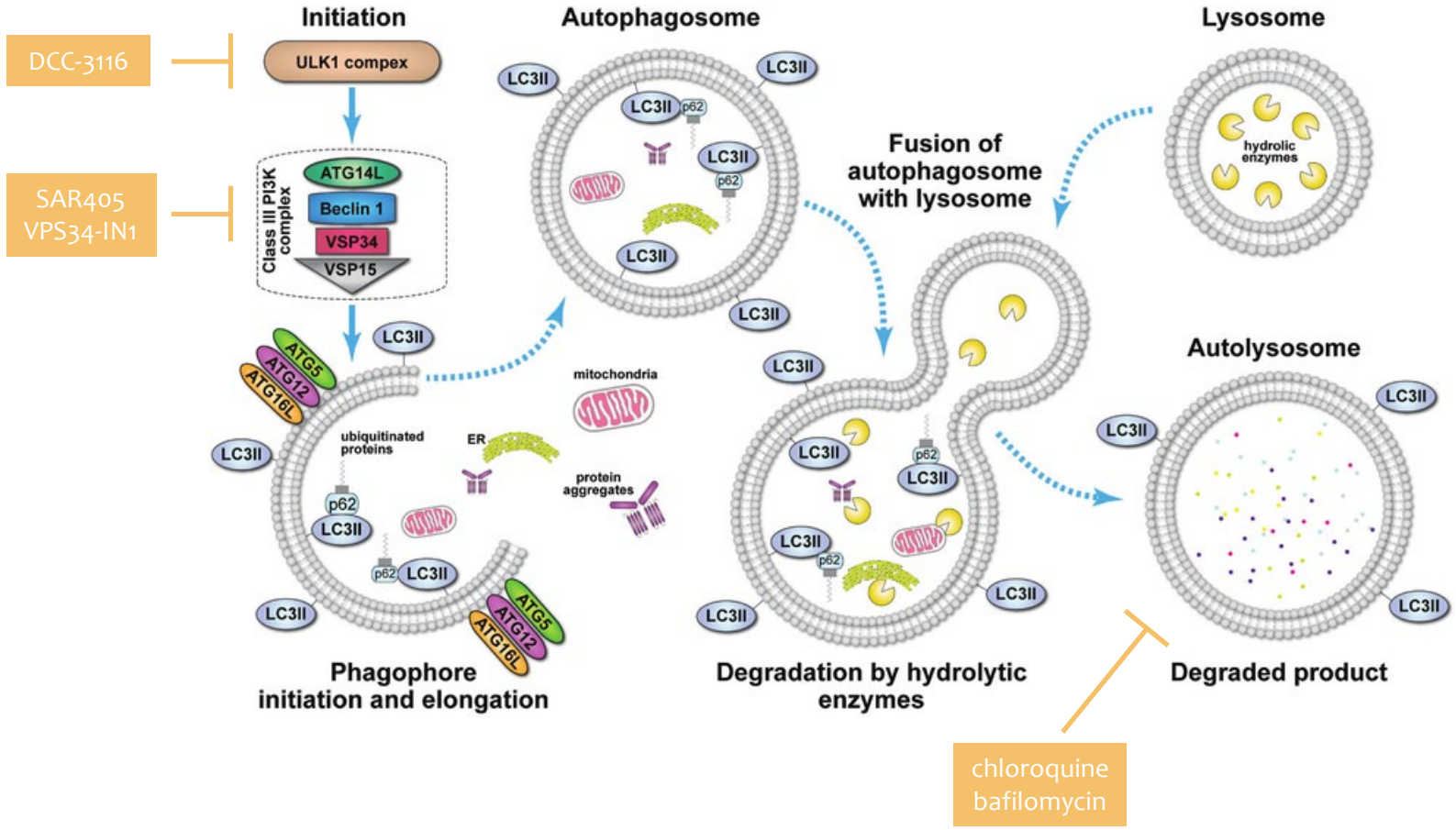
- ULK is initiating factor of autophagy
- Druggable serine/threonine kinase
- Receives and processes key input from nutrient and stress sensors

VPS34 Complex Inhibition

- Druggable lipid kinase target
- ULK can bypass VPS34
- Interference with normal lysosomal house-keeping functions

Lysosomal Inhibition

- Indirect and non-selective inhibition by preventing degradation of the contents
- Elevated pH inactivates hydrolases
- Interference with normal lysosomal house-keeping functions



Adapted from: Ndoye A and Weeraratna AT. Autophagy- An emerging target for melanoma therapy [version 1]. F1000Research 2016, 5:1888. (doi: 10.12688/f1000research.8347.1)

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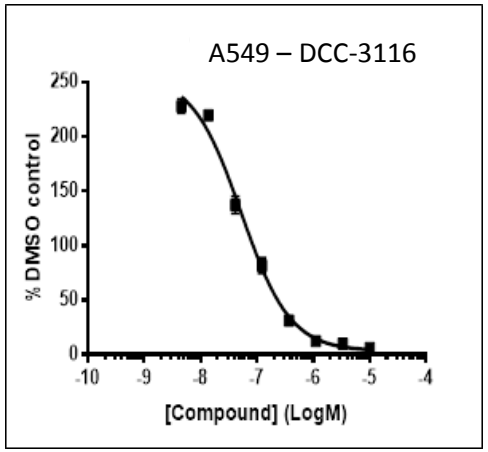
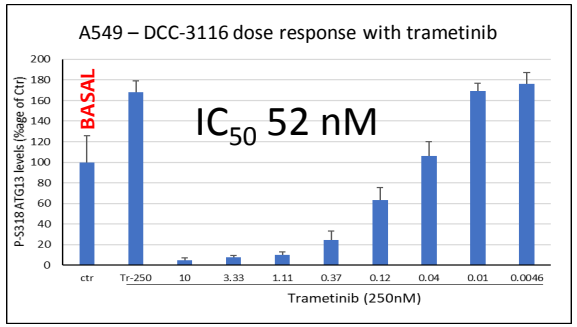
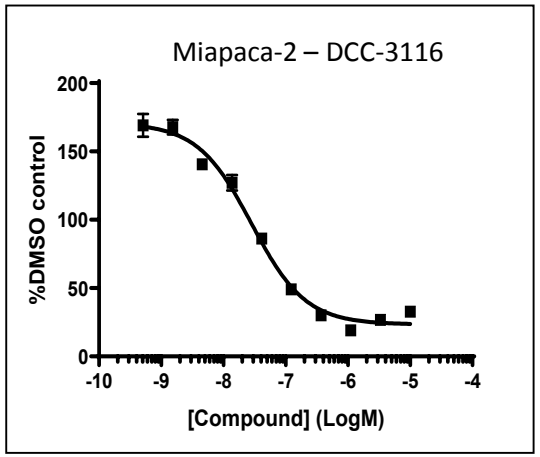
DCC-3116 Potently Inhibits ULK in Multiple RAS Cancer Cell Lines

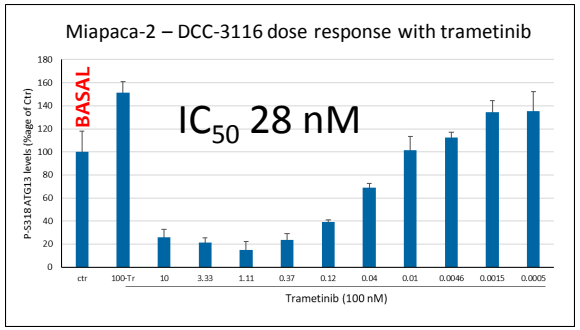
KRAS Lung Cancer



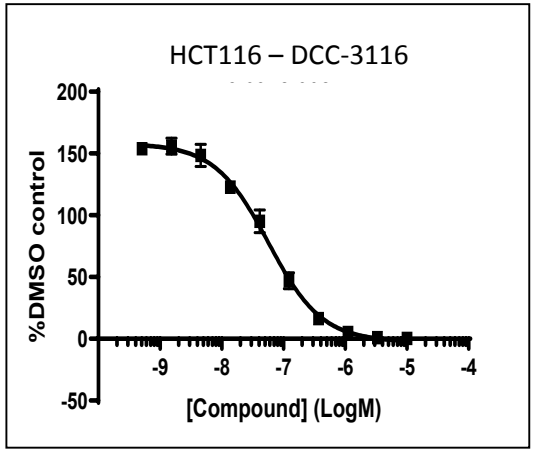


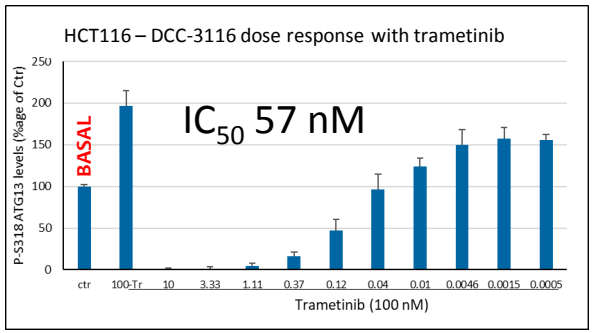
KRAS G12C Pancreatic Cancer



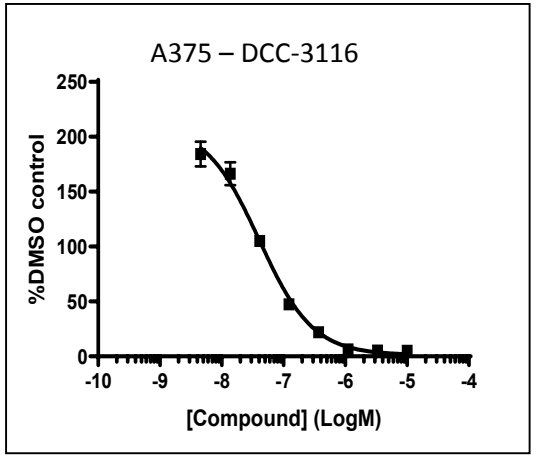


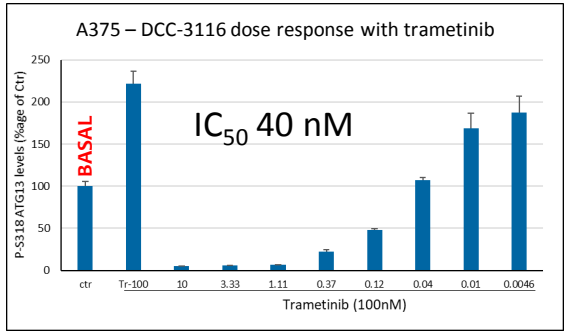
KRAS Colorectal Cancer





BRAF Melanoma Cancer





Basal and MAPK inhibitor-mediated compensatory increased autophagy are inhibited

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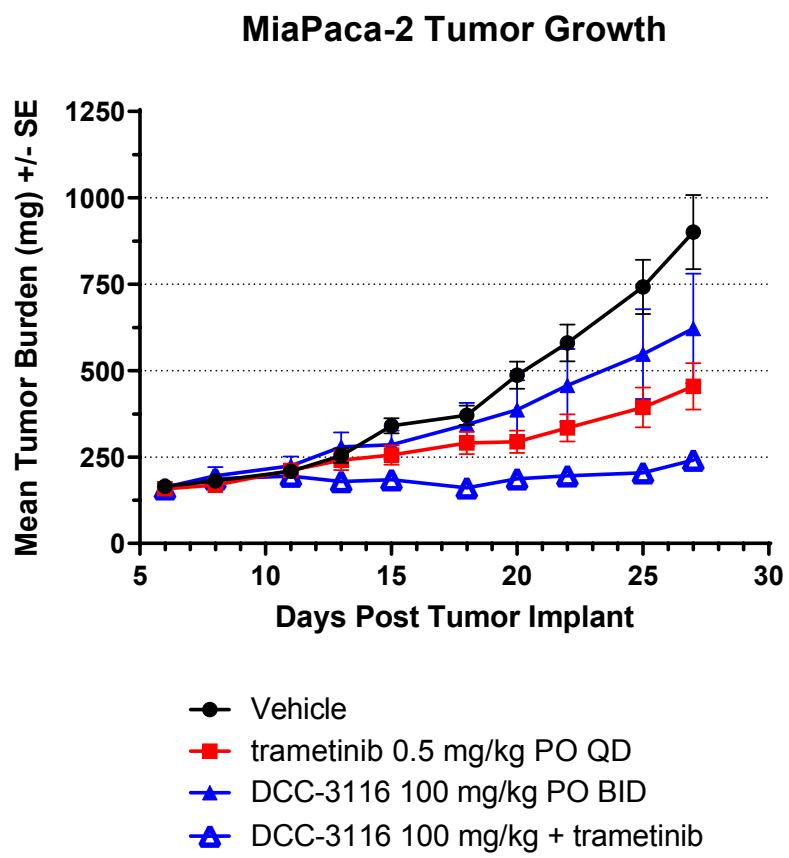
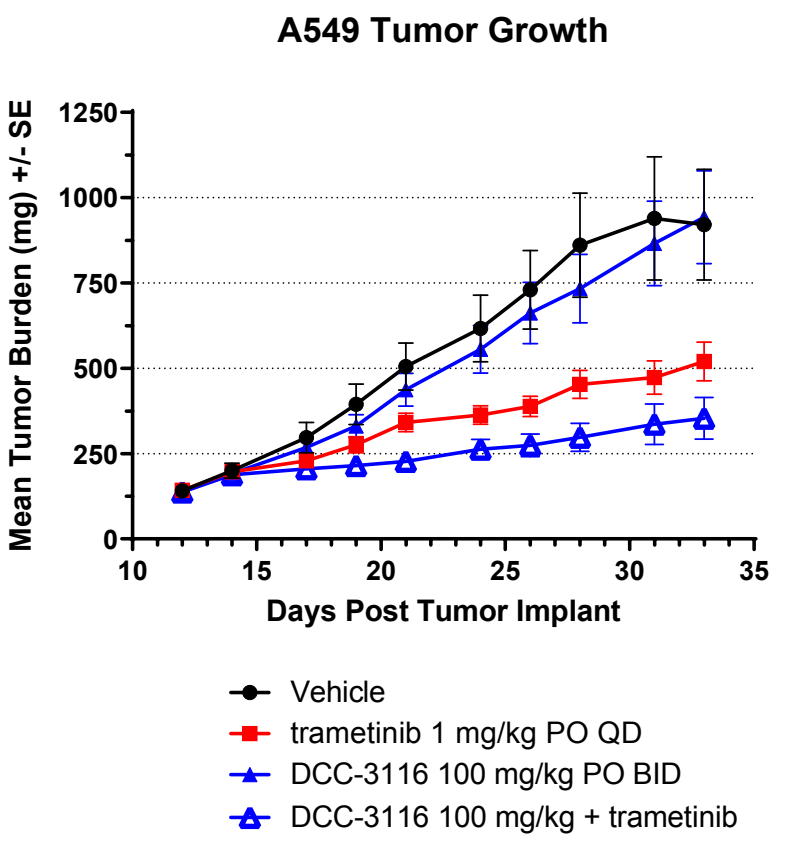
DCC-3116 + MEK Inhibitors Exhibited Reduced Tumor Growth in KRAS *In Vivo* Cancer Models

KRAS MUTANT PANCREATIC



KRAS MUTANT LUNG



Rationale for DCC-3116 in RAS Cancers

RAS CANCERS DEPEND ON MEK/ERK SIGNALING & AUTOPHAGY FOR SURVIVAL	• RAS cancers have high basal levels of autophagy • RAS cancers increase autophagy for survival as resistance mechanism to drug treatments
ULK KINASE IS AN INITIATING FACTOR FOR ACTIVATION OF AUTOPHAGY	• First-in-class target opportunity for new therapeutic in RAS cancer • Differentiated approach to autophagy inhibition
DCC-3116 IS A POTENTIAL FIRST-IN-CLASS ULK KINASE INHIBITOR	• Highly selective and potent inhibitor of ULK kinase • Designed for combination approach
STRONG PRELIMINARY PRECLINICAL VALIDATION	• DCC-3116 inhibits autophagy in RAS cancer cell lines • DCC-3116 potently and durably inhibits autophagy in vivo • Combination of DCC-3116 plus MAPK pathway inhibitors synergize to block RAS cancers *in vivo*

Significant 2019 Milestones Across the Pipeline

Ripretinib

- INVICTUS (≥4th Line GIST: Pivotal Phase 3 Results (Expected Mid-2019))
- Phase 1 Expansion Data (2H 2019)

Rebastinib

- ✓ Phase 1b/2 Carboplatin Combination Initiated (1H 2019)
- ✓ Part 1 of the Phase 1b/2 Paclitaxel Combination Completed Enrollment (1H 2019)
- Part 1 of the Phase 1b/2 Paclitaxel Combination Data (2H 2019)

DCC-3014

- ✓ Phase 1 Dose Escalation Presentation (1H 2019)
- Phase 1 Escalation Data Update (2H 2019)

Discovery Platform

- ✓ Select Clinical Candidate Targeting ULK, Potential First-in-Class Autophagy Inhibitor to Treat mRAS Cancers (1H 2019)
- ✓ Initiate IND-enabling Studies (1H 2019)

NASDAQ:DCPH

Shares Outstanding (as of 3/31/19)

38.2 MM *(basic)*
45.0 MM *(fully-diluted)*

Cash, Cash Equivalents & Marketable Securities (as of 3/31/19)

$262 MM

Cash expected to fund operating expenses and cap ex into 2H 2020

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Thank you to patients, caregivers and healthcare professionals who have participated in our clinical trials

July 2019