DEFEATING CANCER:

The Challenge.
Our Mission.

August 2019

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Disclaimer

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Addressing Unmet Needs for Patients

Positive Results From INVICTUS Phase 3 Clinical Study

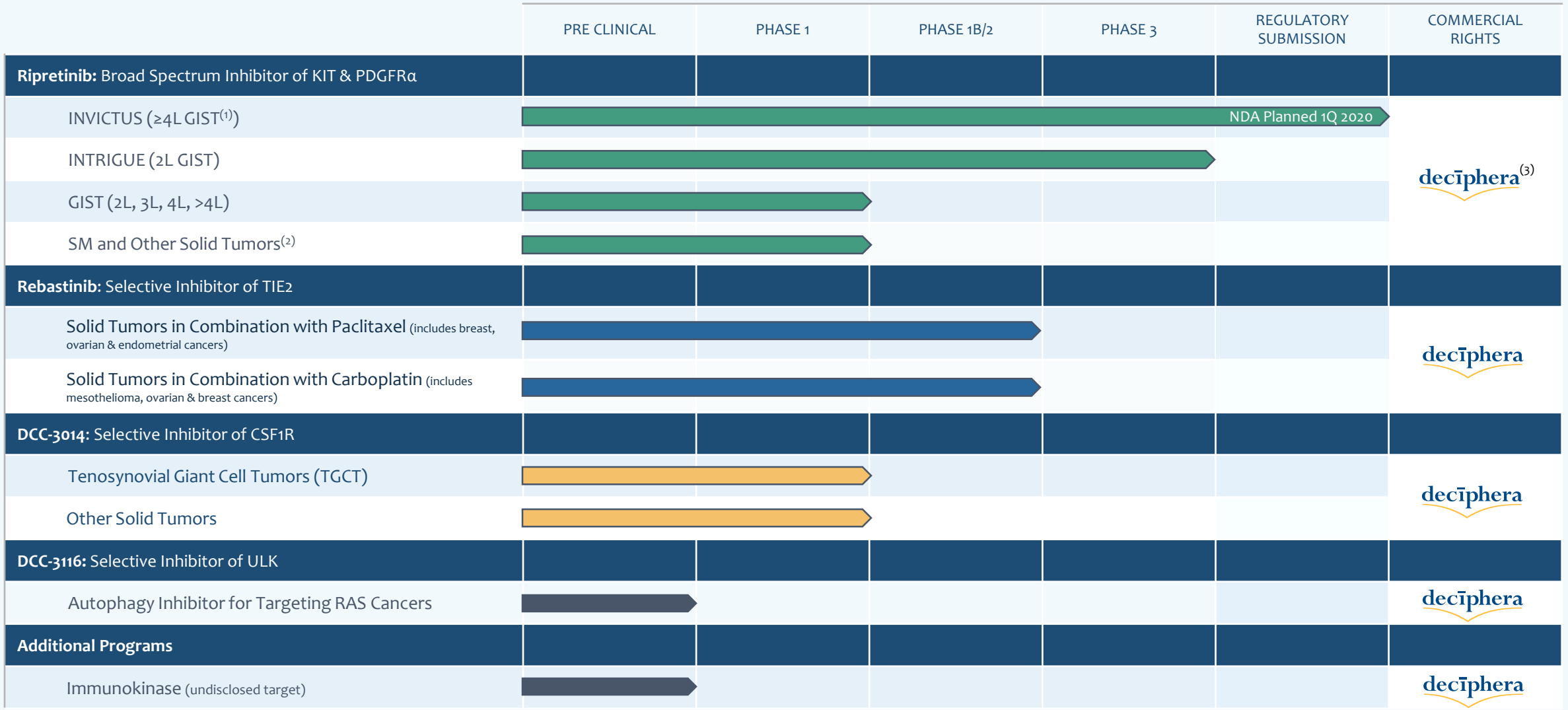
NDA Filing Expected 1Q 2020; Commercial Preparations Underway

INTRIGUE Phase 3 Clinical Study in Second-Line GIST Ongoing

Two Additional Clinical Stage Programs

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; (3) Development and commercialization partnership with Zai Labs in Greater China.

Ripretinib: Designed to Address Relevant Mutations in GIST

Summary

Highly Potent Small Molecule KIT and PDGFRα Inhibitor

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

Positive Results in INVICTUS Phase 3 Clinical Study

- Randomized, placebo-controlled, pivotal study in ≥4th line GIST

NDA Submission for the Treatment of Patients with Advanced GIST who Have Received Prior Treatment with Imatinib, Sunitinib and Regorafenib Expected 1Q 2020

Ongoing INTRIGUE Phase 3 Clinical Study

- Pivotal study in 2nd line GIST vs. sunitinib

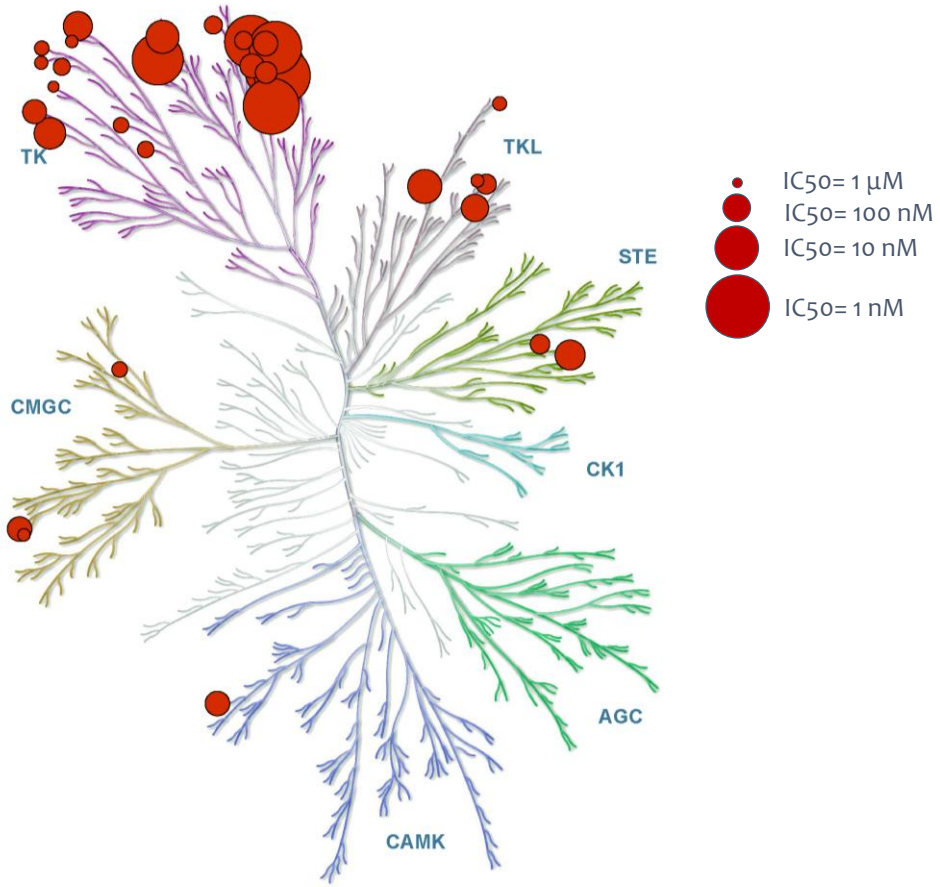
Clinical Proof-of-Concept Demonstrated in 178 GIST Patients in Phase 1

Favorable Tolerability Profile

IP: Composition and Method of Use (2032)

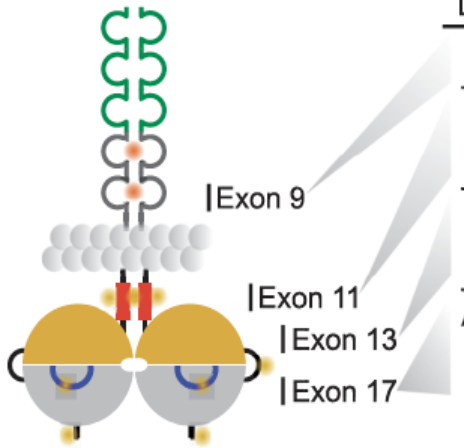
Source and notes: Smith et al. Cancer Cell 35, 738-751, May 13, 2019; Composite of enzyme and cellular kinase phosphorylation data was used. The size of the red circle corresponds to the IC_{50} value obtained. No circles are plotted for kinases with IC_{50} > 1 µM; Illustration reproduced courtesy of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

Kinome Profile



Ripretinib Designed to Address Broad Range of Mutations in GIST

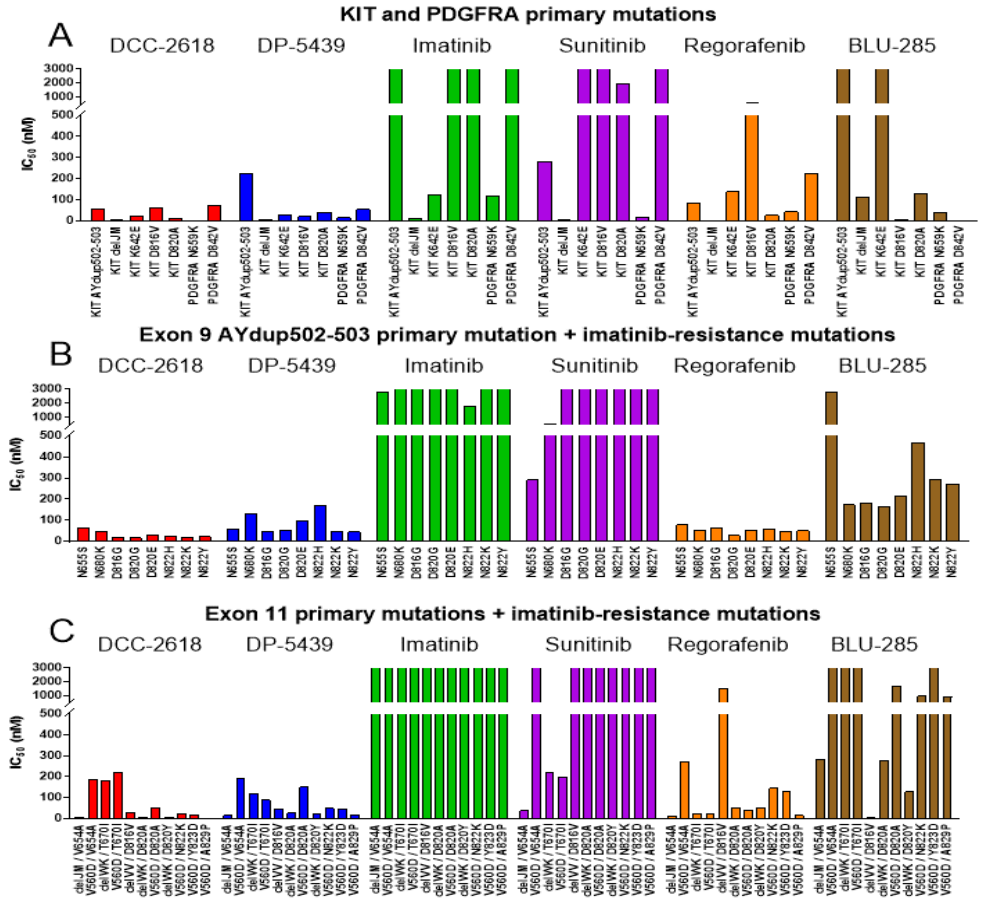
KIT Mutations Drive ~ 80% of GIST



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

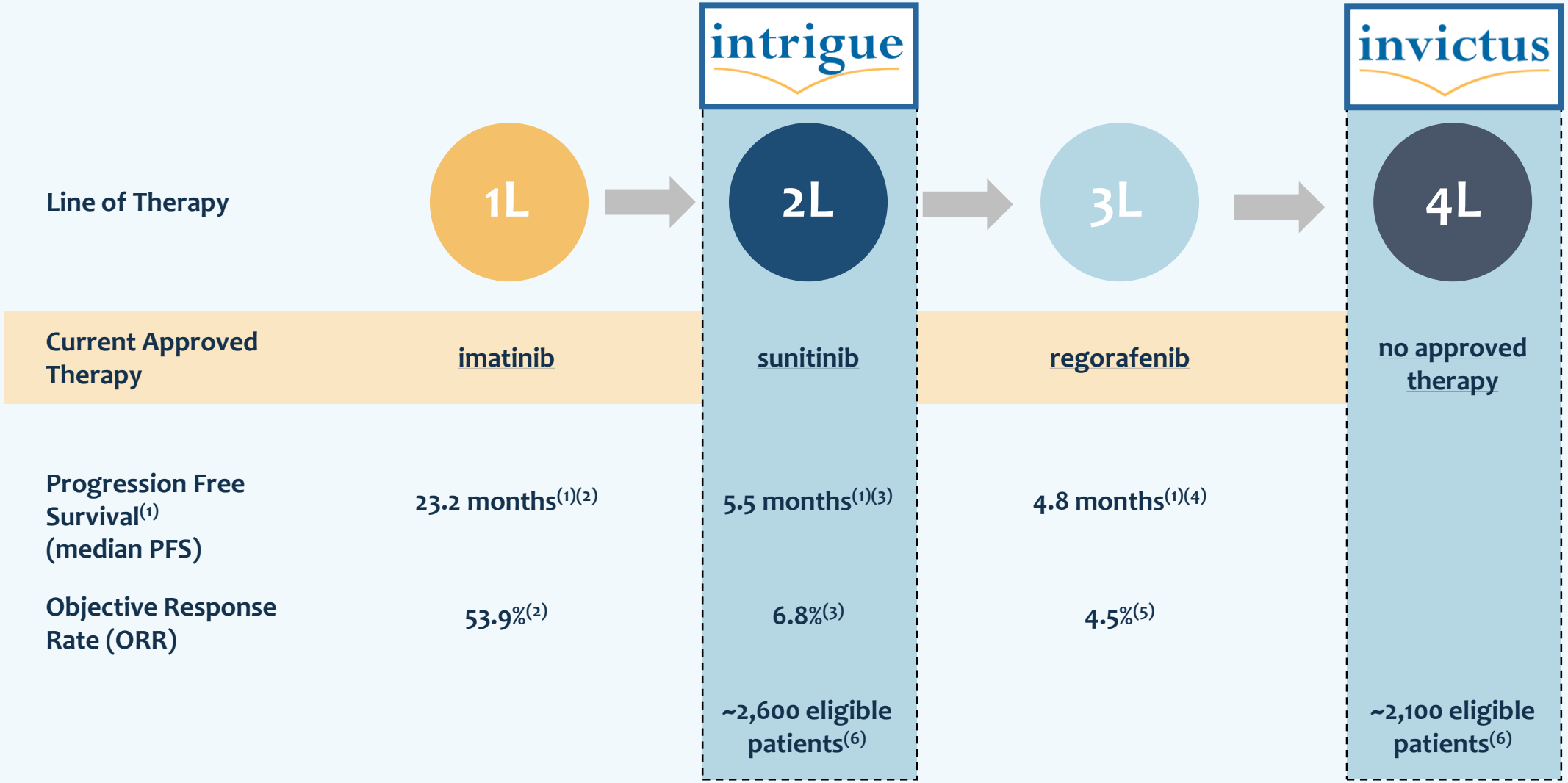
Source: Hemming M et al. Translational insights into gastrointestinal stromal tumors and current clinical advances. Annals of Oncology; 0:1-9, 2018.

Ripretinib: Broad Mutational Coverage in KIT and PDGFRα



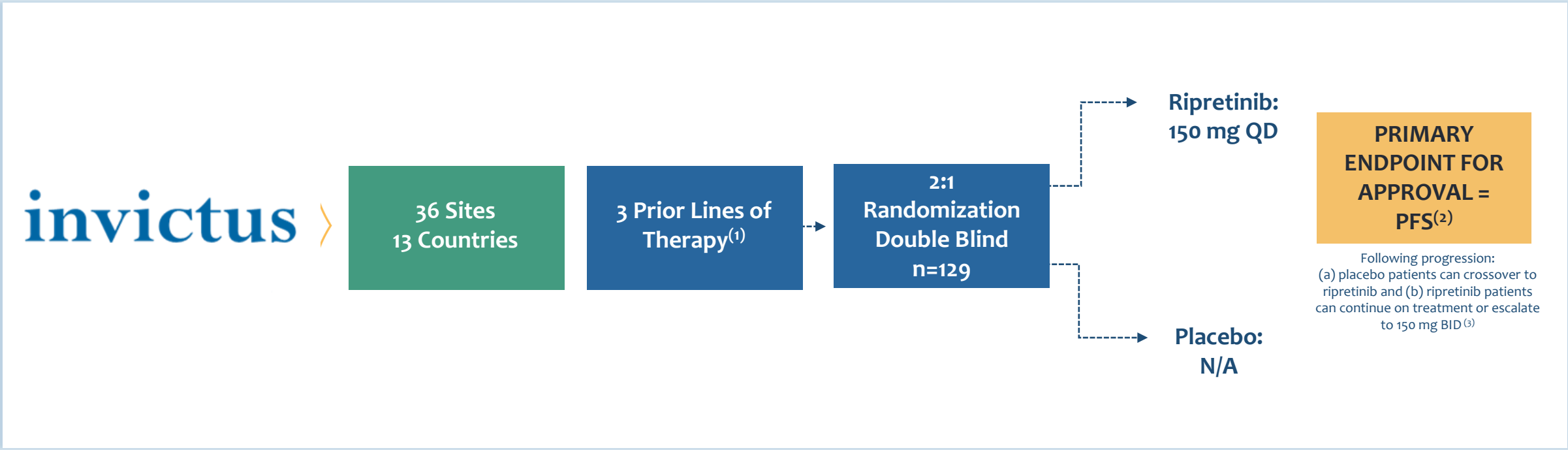
Source: AACR 2018
Notes: (A) GIST primary mutations; or imatinib-resistant KIT mutations with (B) exon 9 or (C) exon 11 primary mutations.

Ripretinib Opportunity in GIST



Notes: (1) Includes progression free survival and time to progression converted to weeks; (2) Gleevec [package insert]. Stein, Switzerland: Novartis; 2008; (3) Sutent [package insert]. New York, NY: Pfizer; 2011; (4) Stivarga [package insert]. Germany: Bayer Healthcare; 2013; (5) Demetri, George D et al. "Efficacy and safety of regorafenib for advanced gastrointestinal stromal tumours after failure of imatinib and sunitinib (GRID): an international, multicentre, randomised, placebo-controlled, phase 3 trial." Lancet. 2013; 381(9863):295–302; (6) Internal Deciphera estimates for annual incidence of treatment eligible new patients with KIT mutation per line based on applying epidemiology data reported in the following publications to population estimates for US: Zhao et al. J Gastrointest Oncol 2012;3(3):189-208; Metaxas Y, et al. ESMO Open 2016.

INVICTUS: Global Pivotal Phase 3 Study in ≥4th Line GIST



Notes: (1) Phase 3 pivotal study in patients with ≥4th line GIST who previously received at least imatinib, sunitinib, and regorafenib; (2) PFS = progression free survival; (3) BID = twice daily.

INVICTUS Achieved Primary Endpoint of Progression Free Survival

	Ripretinib (n = 85)	Placebo (n = 44)[1]	p-value
mPFS[2]	6.3 months (27.6 weeks)	1.0 month (4.1 weeks)	< 0.0001
ORR[3]	9.4%	0%	0.0504
mOS[4]	15.1 months	6.6 months	Nominal p-value = 0.0004[5]

Significantly reduced the risk of disease progression or death by 85% (Hazard Ratio of 0.15, p-value < 0.0001) compared to placebo

NDA filing for the treatment of patients with advanced GIST who have received prior treatment with imatinib, sunitinib and regorafenib expected 1Q 2020

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Notes: (1) One patient was randomized to placebo but did not receive study drug; (2) mPFS = median progression free survival; (3) ORR = objective response rate; (4) mOS = median overall survival; (5) According to the pre-specified hierarchical testing procedure of the endpoints, the hypothesis testing of mOS cannot be formally conducted unless the test of ORR is statistically significant. Because statistical significance was not achieved for ORR, the hypothesis testing of OS was not formally performed.

Ripretinib Was Generally Well Tolerated

TEAEs in >15% of Patients

Treatment Emergent Adverse Event (TEAE)	Placebo (N=43)[1]	Ripretinib 150mg Daily (N=85)
Any event	42 (98%)	84 (99%)
Alopecia	2 (5%)	44 (52%)
Fatigue	10 (23%)	36 (42%)
Nausea	5 (12%)	33 (39%)
Abdominal pain	13 (30%)	31 (36%)
Constipation	8 (19%)	29 (34%)
Myalgia	5 (12%)	27 (32%)
Diarrhea	6 (14%)	24 (28%)
Decreased appetite	9 (21%)	23 (27%)
Palmar-plantar erythrodysaesthesia syndrome	0	18 (21%)
Vomiting	3 (7%)	18 (21%)
Headache	2 (5%)	16 (19%)
Weight decreased	5 (12%)	16 (19%)
Arthralgia	2 (5%)	15 (18%)
Blood bilirubin increased	0	14 (16%)
Oedema peripheral	3 (7%)	14 (16%)
Muscle spasms	2 (5%)	13 (15%)

Notes: (1) One patient was randomized to placebo but did not receive study drug and, therefore, was not included in the safety population.

- Grade 3 or 4 TEAEs >5% of patients in the ripretinib arm were anemia (9%; n=8), abdominal pain (7%; n=6) and hypertension (7%; n=6)
- Grade 3 or 4 TEAEs >5% of patients in the placebo arm were anemia (14%; n=6)

Summary of Top-line Results for INVICTUS Phase 3 Study

Ripretinib achieved Primary Endpoint:
median PFS of 6.3 Months vs. Placebo of 1.0 Month;
Hazard Ratio of 0.15, p<0.0001

Ripretinib was generally well tolerated

Additional results expected to be presented at an upcoming medical meeting

NDA submission for the treatment of patients with advanced GIST who have received prior treatment with imatinib, sunitinib and regorafenib expected 1Q 2020

Phase 1: Positive Updated Results Across All Lines of Treatment ≥100 mg/d (n=178)

Data cut-off of March 1, 2019 is ~6 months from data cut-off at ESMO 2018

Line of Therapy[1]	**Objective Response Rate by Best Response** Includes Unconfirmed (Confirmed Only)[2]	Disease Control Rate at 3 Months[2]	Median Progression Free Survival (mPFS)[2]	Censored Patients for mPFS[2]	Mean Treatment Duration[3][4]
2nd Line (n=37)	30% (22%)	81%	42 weeks	38%	43 weeks
3rd Line (n=31)	23% (13%)	80%	40 weeks	32%	48 weeks
4th Line (n=60)	15% (8%)	73%	30 weeks	30%	49 weeks
≥4th Line (n=110)[4]	11% (7%)	66%	24 weeks	22%	41 weeks

(1) Overall number of patients (n=178) remains the same as prior data presented at ESMO 2018; based on additional data cleaning, one patient from each of 2nd line and 4th/≥4th line were reclassified as 3rd line patients; (2) RECIST data per investigator assessment; (3) Median treatment durations were: 2nd line = 44 weeks, 3rd line = 48 weeks, 4th line = 46 weeks and ≥4th line = 29 weeks (4) Includes 60 patients who elected for intra-patient dose escalation from 150 mg QD to 150 mg BID; (4) Number of patients in ≥4th line includes 60 patients from 4th line.

Key Updates from Phase 1 GIST Patients ≥100 mg/d

Supports ripretinib's potential across the broad range of KIT and PDGFRα mutations known to occur in patients with GIST post-imatinib

Updated ≥4th line data support potential commercial opportunity

- Encouraging clinical activity in 4th line GIST
- Extended treatment duration in ≥4th line GIST

Updated 2nd line data support ongoing INTRIGUE Phase 3 study

- mPFS sustained with 6 months of additional data maturity
- ORR and DCR increased since last data cut

Ripretinib was generally well tolerated

- Updated adverse events were consistent with previously presented Phase 1 data
- Most common treatment-emergent grade 3/4 adverse events ≥5% patients were lipase increase, anemia, hypertension and abdominal pain

INTRIGUE: Ongoing Global Pivotal Phase 3 Study in 2nd Line GIST

Notes: (1) Number of open sites current as of August 12, 2019; (2) Phase 3 pivotal study in 2nd line patients who previously received imatinib; (3) PFS = progression free survival.

Expanding Clinical Stage Portfolio

DCC-3014

- Phase 1 study ongoing
- Currently enrolling TGCT(1) patients in the Phase 1 study
- Phase 1 escalation data update expected 2H 2019

Rebastinib

- Two Phase 1b/2 studies ongoing
- Data from Part 1 of the Phase 1b/2 paclitaxel study 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.

DCC-3014: A Highly Selective and Potent CSF1R Inhibitor

Summary

Phase 1 escalation study 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

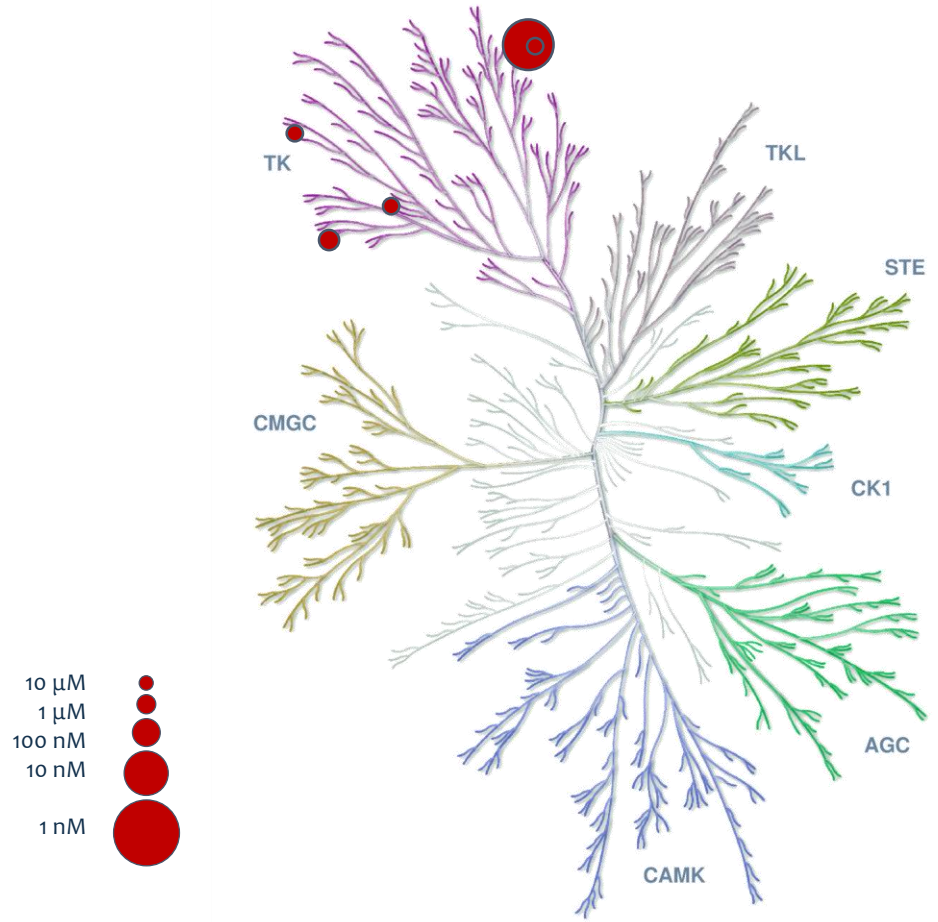
- No DLTs in loading and maintenance regimens
- No DCC-3014 related G3/4 TEAEs in ≥ 10% patients
- PK analysis demonstrated dose-proportional exposure for loading and maintenance regimen

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).

Kinome Profile



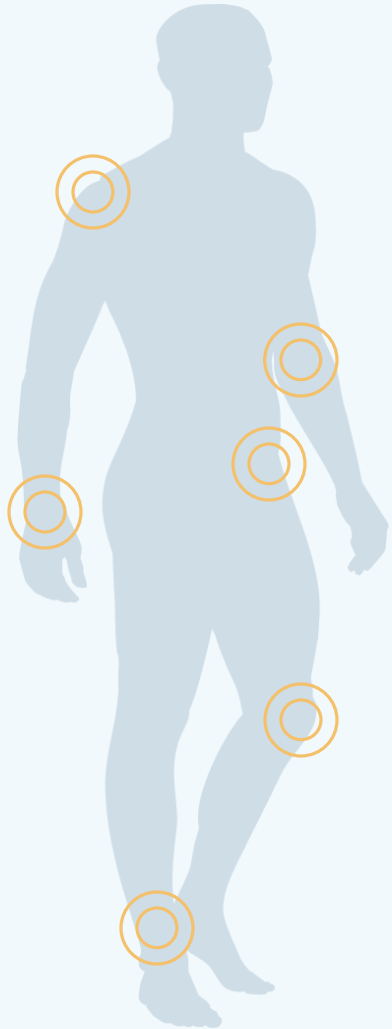
Single Agent Opportunity in Tenosynovial Giant Cell Tumor (TGCT)

Symptoms

- 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 fingers, toes, knee, wrist and ankle
   - Annual incidence of new cases in the U.S. = ~13, 000[1]
2. **Diffuse TGCT (also known as PVNS)**
   - Mostly commonly affects the knee, as well as hip, ankle, elbow and shoulder
   - Annual incidence of new cases in the U.S. = ~1,300[1]



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
- Pexadartinib FDA Approval August 2019
  - 39% ORR VS. 0% for placebo at week 25 in Phase 3 (n=120)
  - REMS and intensive monitoring required due to hepatotoxicity concerns (off-target)
- Unmet medical need for effective CSF1R inhibitor with favorable safety/tolerability profile for diffuse TGCT patients

Source: (1) Mastboom et al. Higher incidence rates than previously known in tenosynovial giant cell tumors. In: Acta Orthopaedica 2017; 88 (6): 688-694.

Rebastinib: A Highly Potent and Selective TIE2 Inhibitor

Summary

Potent, small molecule inhibitor of TIE2

Preclinical anti-tumor activity

- Single agent and I/O or chemo combination

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

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

- Tumor vascularization, dissemination, metastasis, immunotolerance

Phase 1 study completed

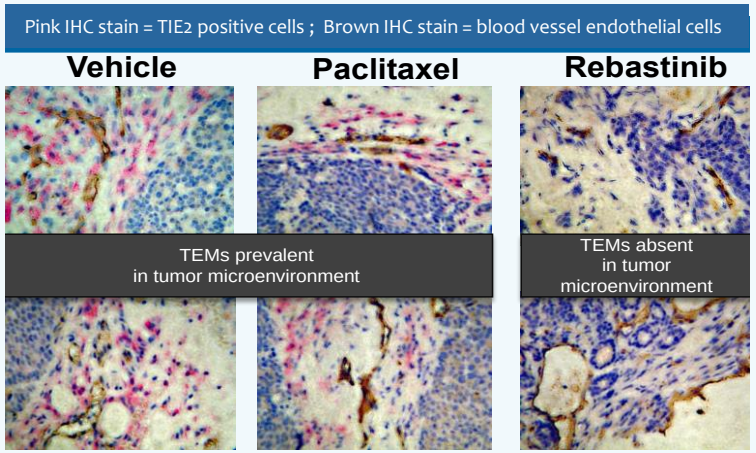
- Identified 150 mg BID dose as maximum tolerated dose

Development status

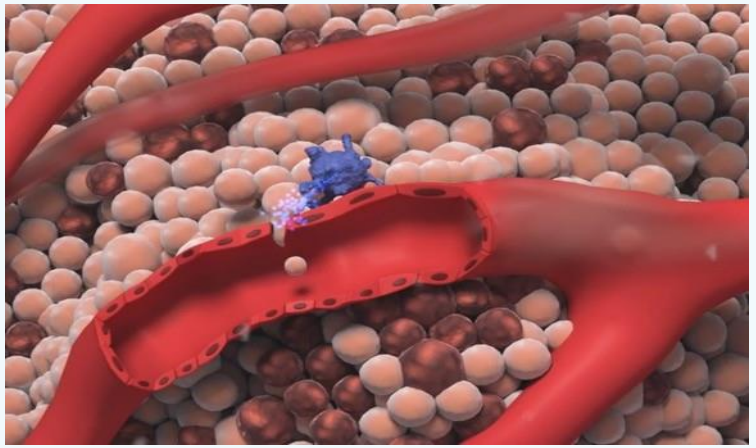
- Two ongoing company-sponsored chemo combo studies with paclitaxel and carboplatin
- Data from Part 1 of the Phase 1b/2 study with paclitaxel expected 2H 2019

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

Potential Benefits in Combo with Chemotherapy

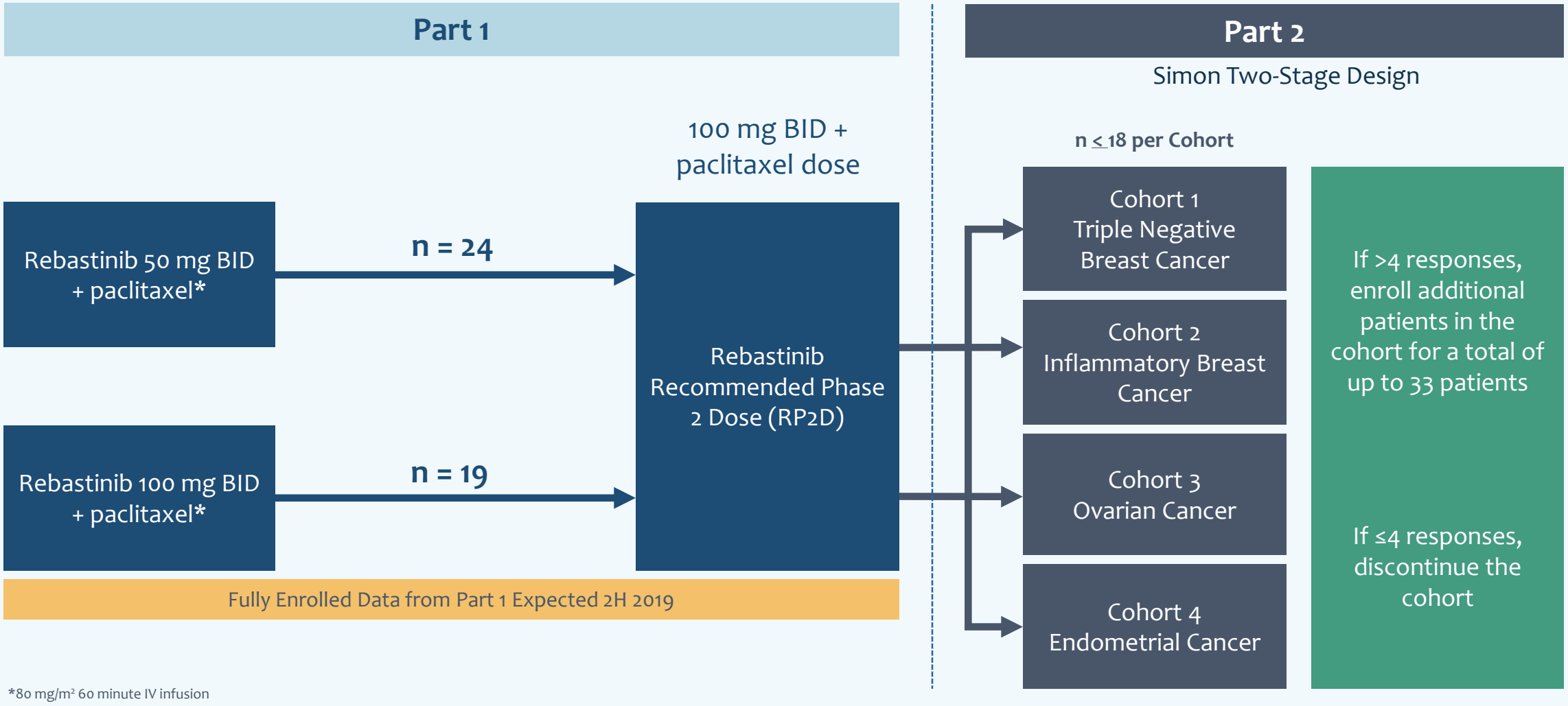


- Chemotherapy leads to recruitment of pro-tumoral M2 macrophages from the bone marrow
- Rebastinib is designed to inhibit chemotherapy-induced recruitment of M2 macrophages to tumors



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

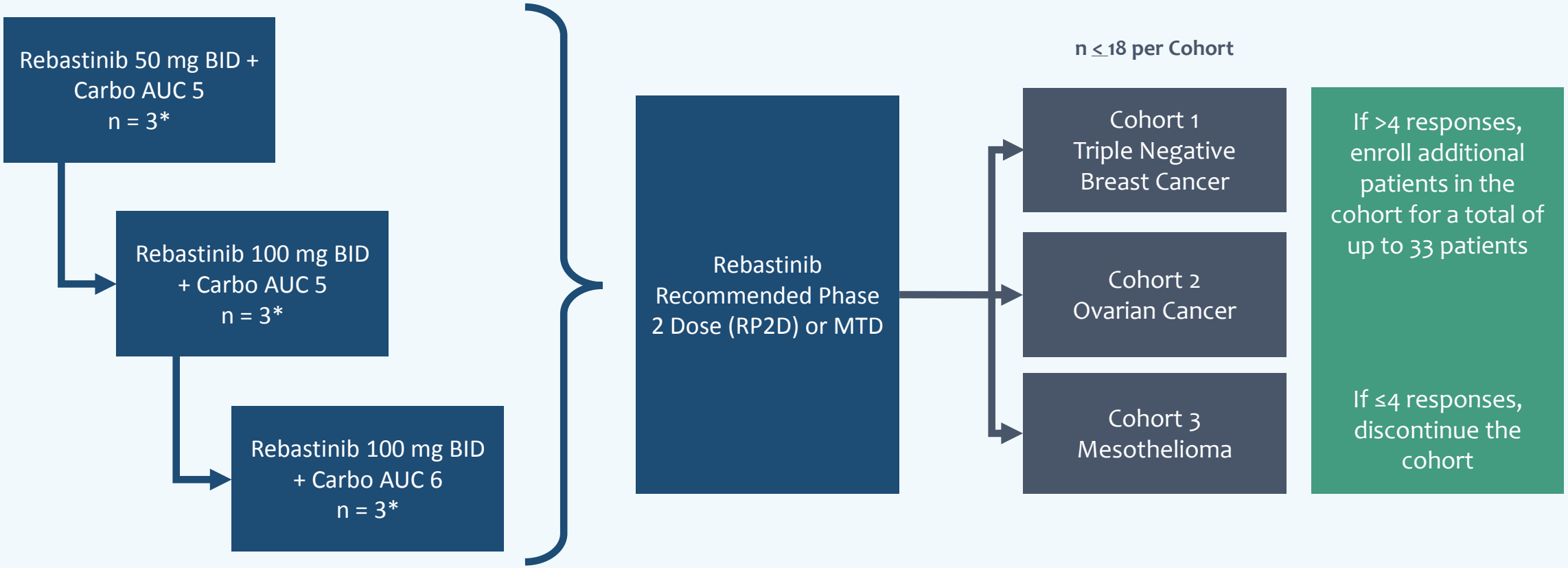
Rebastinib: Phase 1b/2 Study Combination with Paclitaxel



*80 mg/m² 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-3116 is a Potent & Selective ULK Inhibitor Designed to Inhibit Autophagy

Summary

Highly Potent (IC_{50} at 1 mM ATP)

- ULK1 4.7 nM
- ULK2 35 nM

Highly Selective

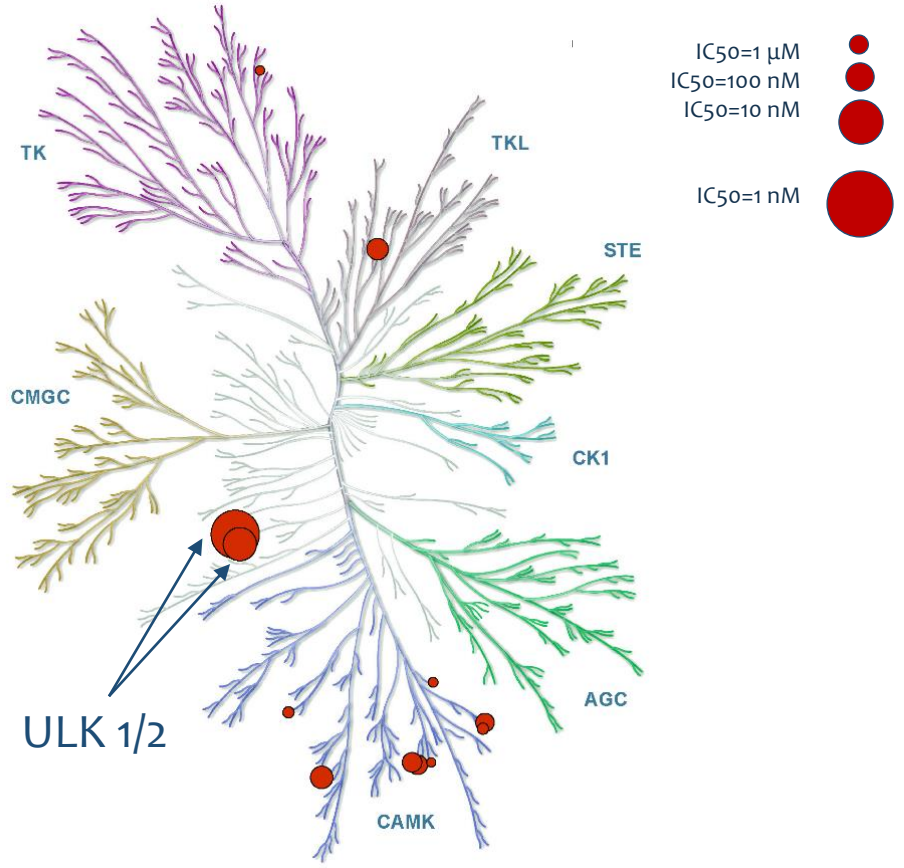
- No off-target kinases within 30-fold of ULK1
- Only 5 kinases within 100-fold of ULK1

Designed to Avoid CNS Exposure

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

IND Filing Expected in Mid-2020

DCC-3116: A Selective ULK1/2 Inhibitor



Source and Notes: 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).

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

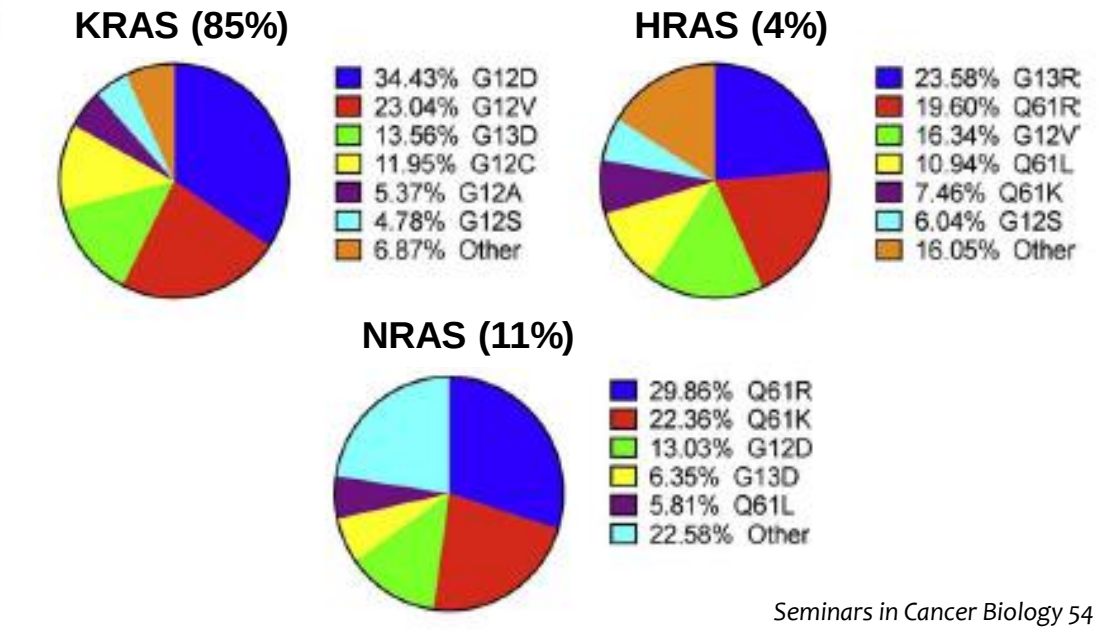
- Pancreatic: ~98%
- Colon: ~ 45%
- Lung: ~ 30%

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]

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

Strategies for Blocking Autophagy in Cancer

ULK Inhibition

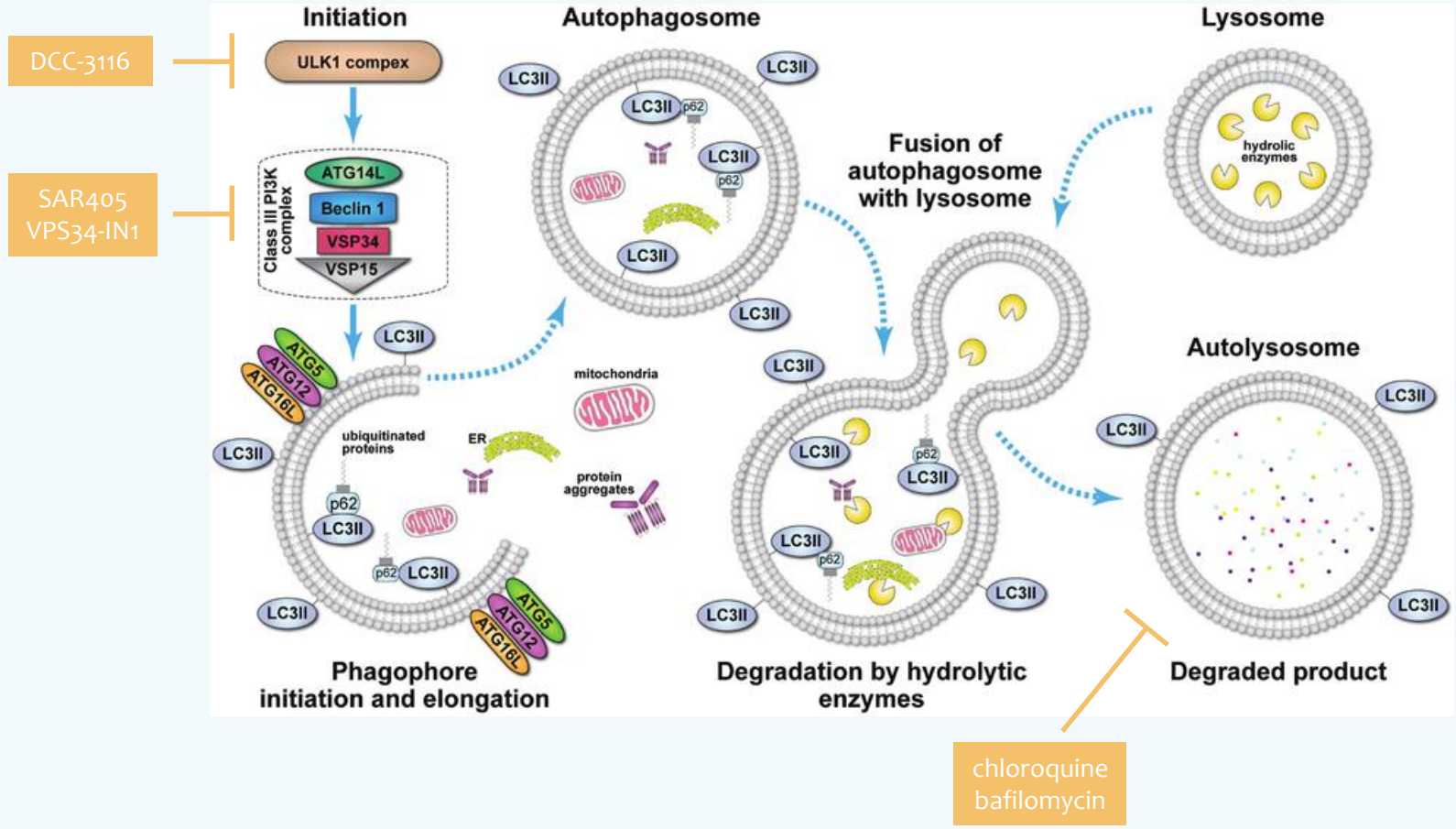
- ULK is initiating factor of autophagy
- Druggable serine/threonine kinase
- Receives and processes key input from nutrient and stress sensors

VPS34 Complex Inhibition

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

Lysosomal Inhibition

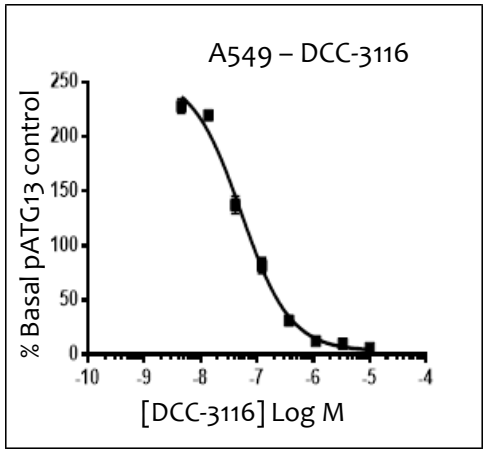
- Indirect and non-selective inhibition by preventing degradation of the contents
- Elevated pH inactivates hydrolases
- Interference with normal lysosomal house-keeping functions

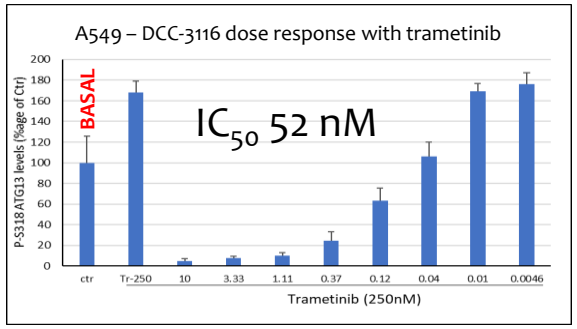


Adapted from: Ndoye A and Weeraratna AT. Autophagy- An emerging target for melanoma therapy [version 1]. F1000Research 2016, 5:1888. (doi: 10.12688/f1000research.8347.1).

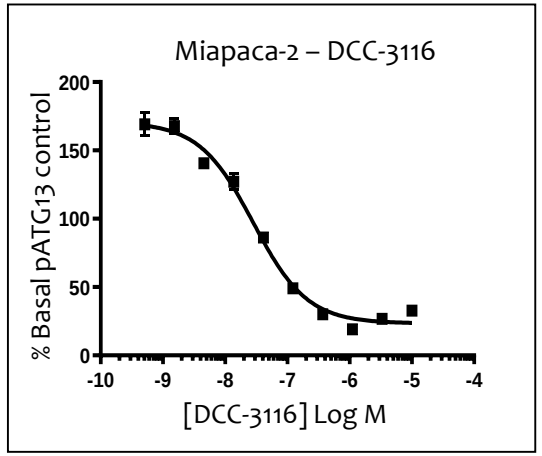
DCC-3116 Potently Inhibits ULK in Multiple RAS Cancer Cell Lines

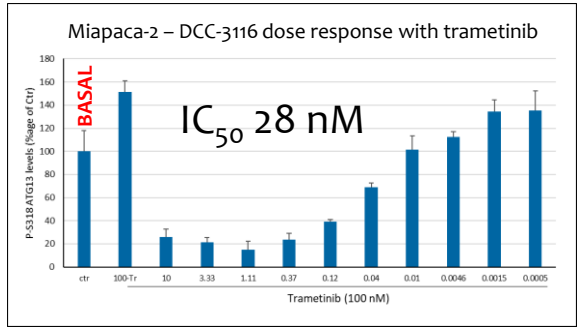
KRAS Lung Cancer



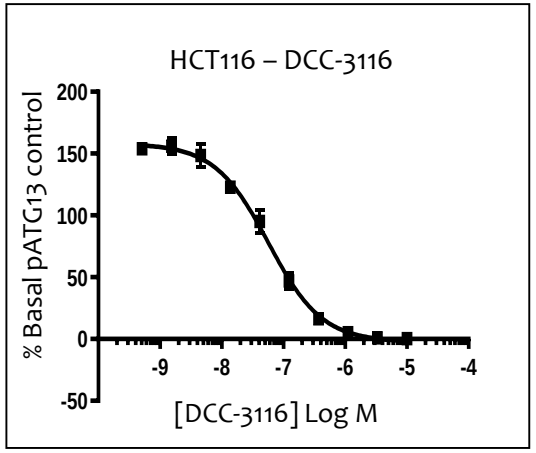


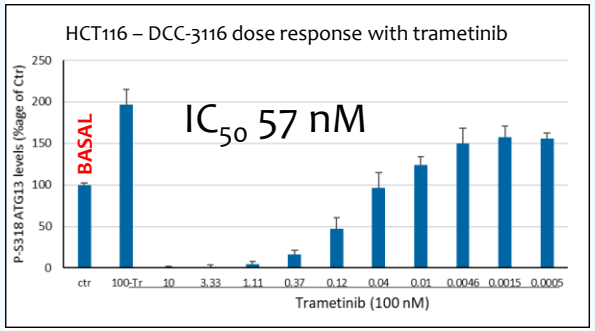
KRAS G12C Pancreatic Cancer



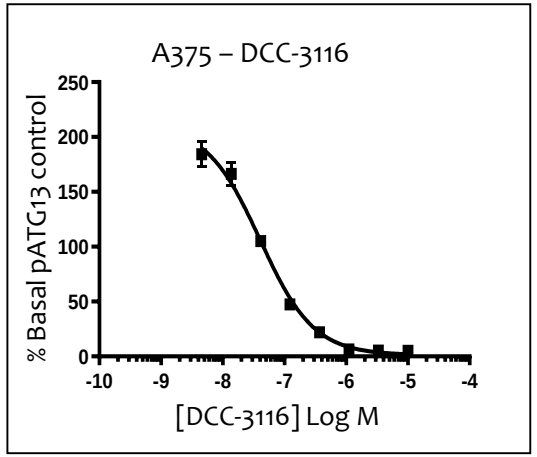


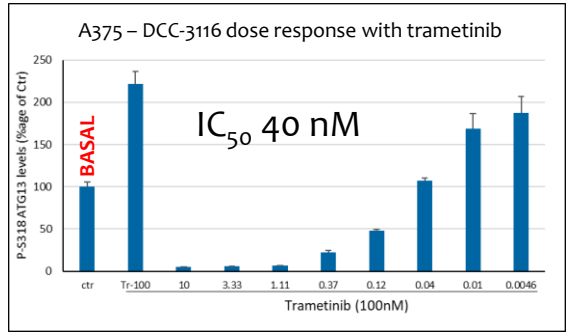
KRAS Colorectal Cancer





BRAF Melanoma Cancer

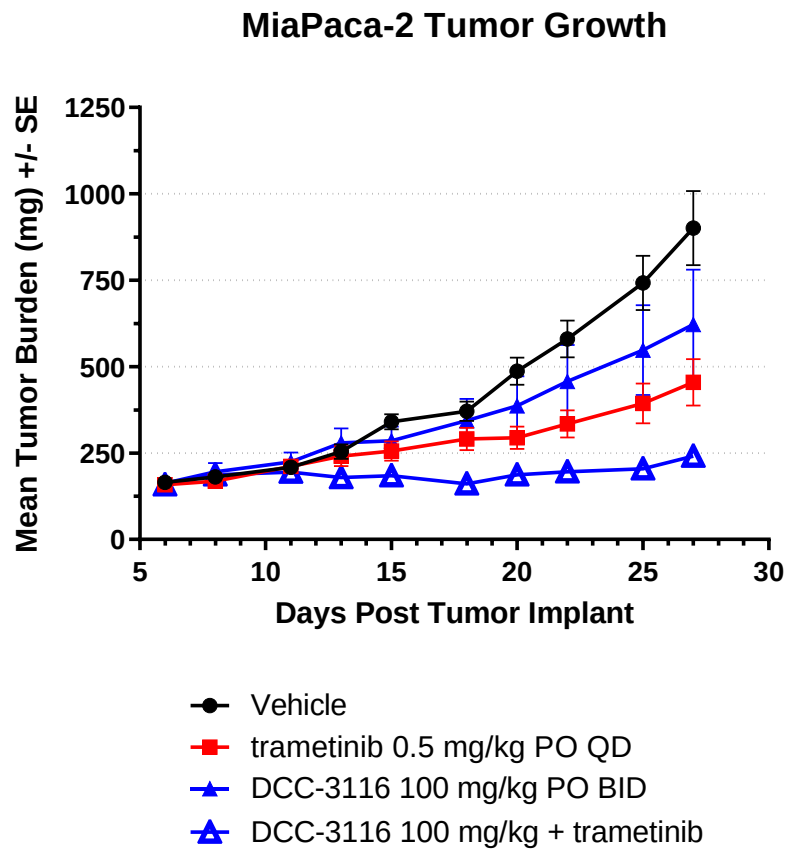




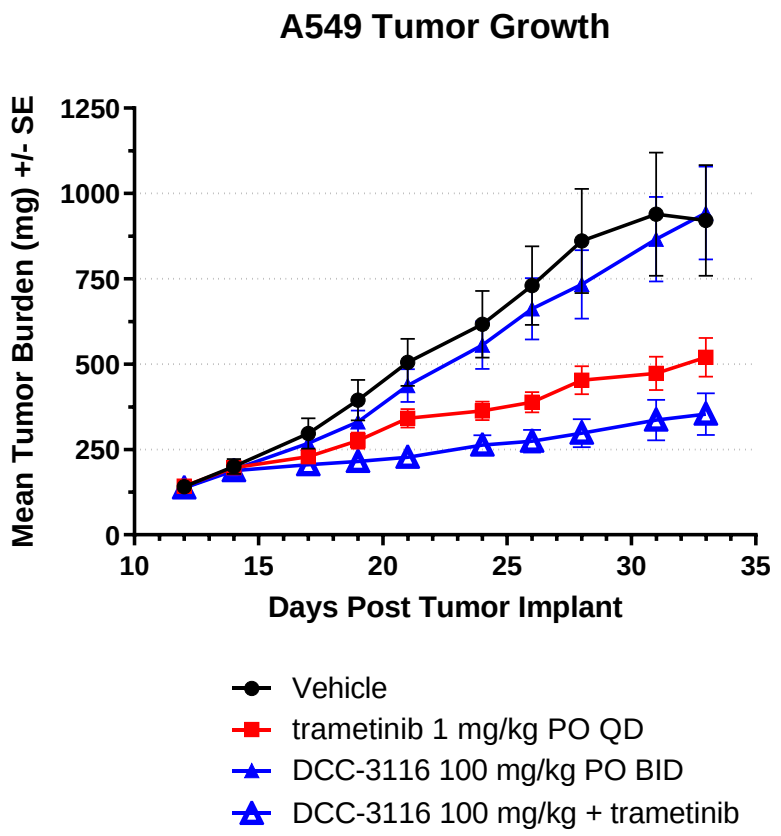
Basal and MAPK inhibitor-mediated compensatory increased autophagy are inhibited

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

- ✓ Top-line INVICTUS Data (≥4th Line GIST: Pivotal Phase 3 Results)
- ✓ Phase 1 Data Update
- INVICTUS Data Presented at Medical Meeting (2H 2019)
- Phase 1 Data Presented at Medical Meeting (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 Presented at Medical Meeting (2H 2019)

DCC-3014

- ✓ Phase 1 Dose Escalation Presentation (1H 2019)
- Phase 1 Escalation Data Update Presented at Medical Meeting (2H 2019)

Discovery Platform

- ✓ New Clinical Candidate - DCC-3116: Selective ULK Inhibitor. Potential First-in-Class Autophagy Inhibitor to Treat mRAS Cancers (1H 2019)
- ✓ Initiate IND-enabling Studies (1H 2019)

NASDAQ: DCPH

Shares Outstanding
(as of 6/30/19)

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

$225 MM

Cash, Cash Equivalents &
Marketable Securities
(as of 6/30/19)

CASH EXPECTED TO FUND OPERATING EXPENSES AND CAP EX INTO 4Q 2020

decīphera