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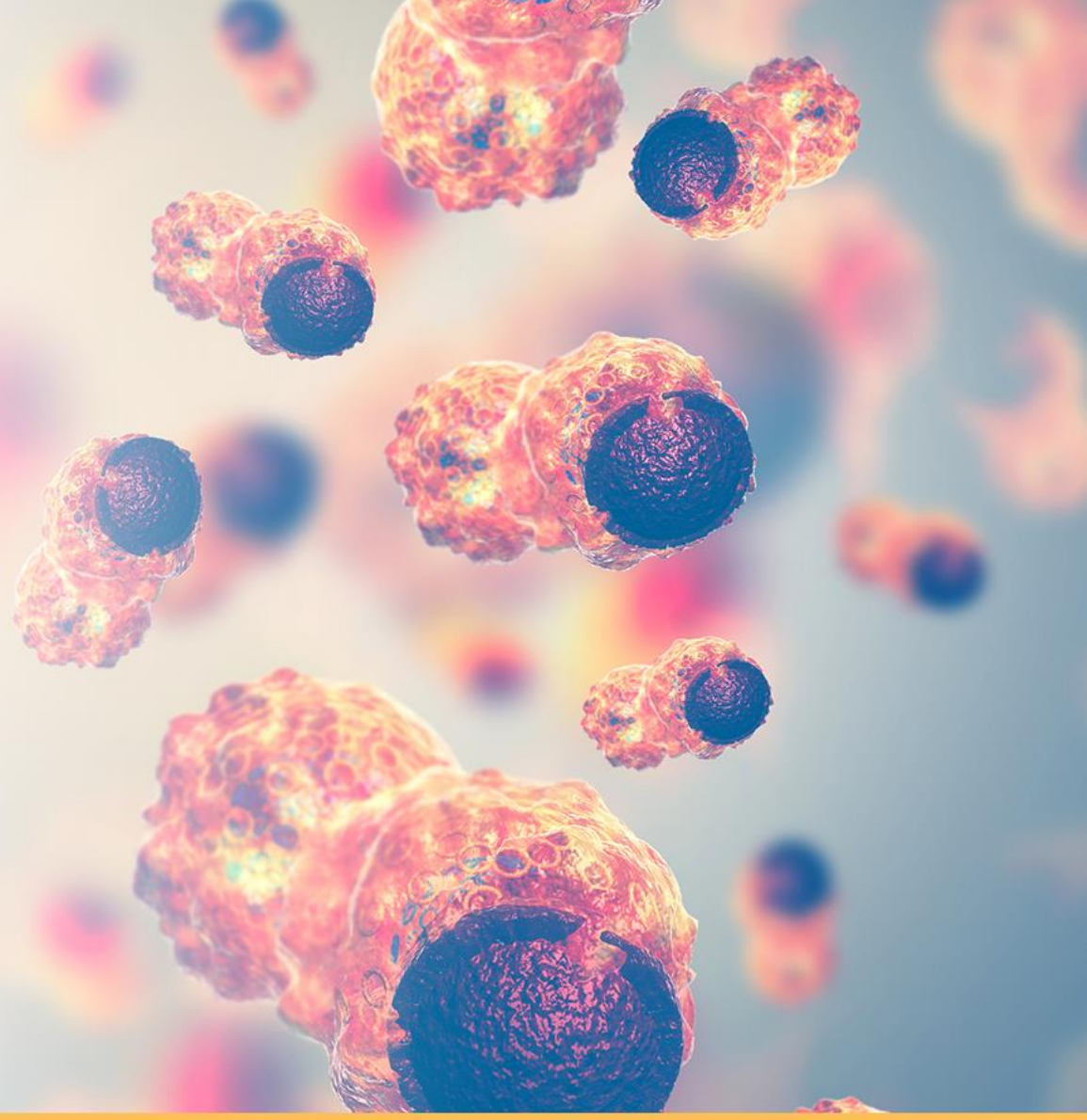
Addressing Key Mechanisms of Tumor Drug Resistance

April 2019



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Corporate Summary

Well established drug class with significant growth potential

Differentiated approach to kinase inhibition

Wholly-owned pipeline with three clinical stage assets

Kinase Inhibitors: 40+ FDA-approved Drugs but Significant Opportunities Remain

- Drug resistance mutations limit rate and duration of response
- Low potency and selectivity cause poor tolerability
- Approved drugs target less than 10% of the 500+ known human kinases

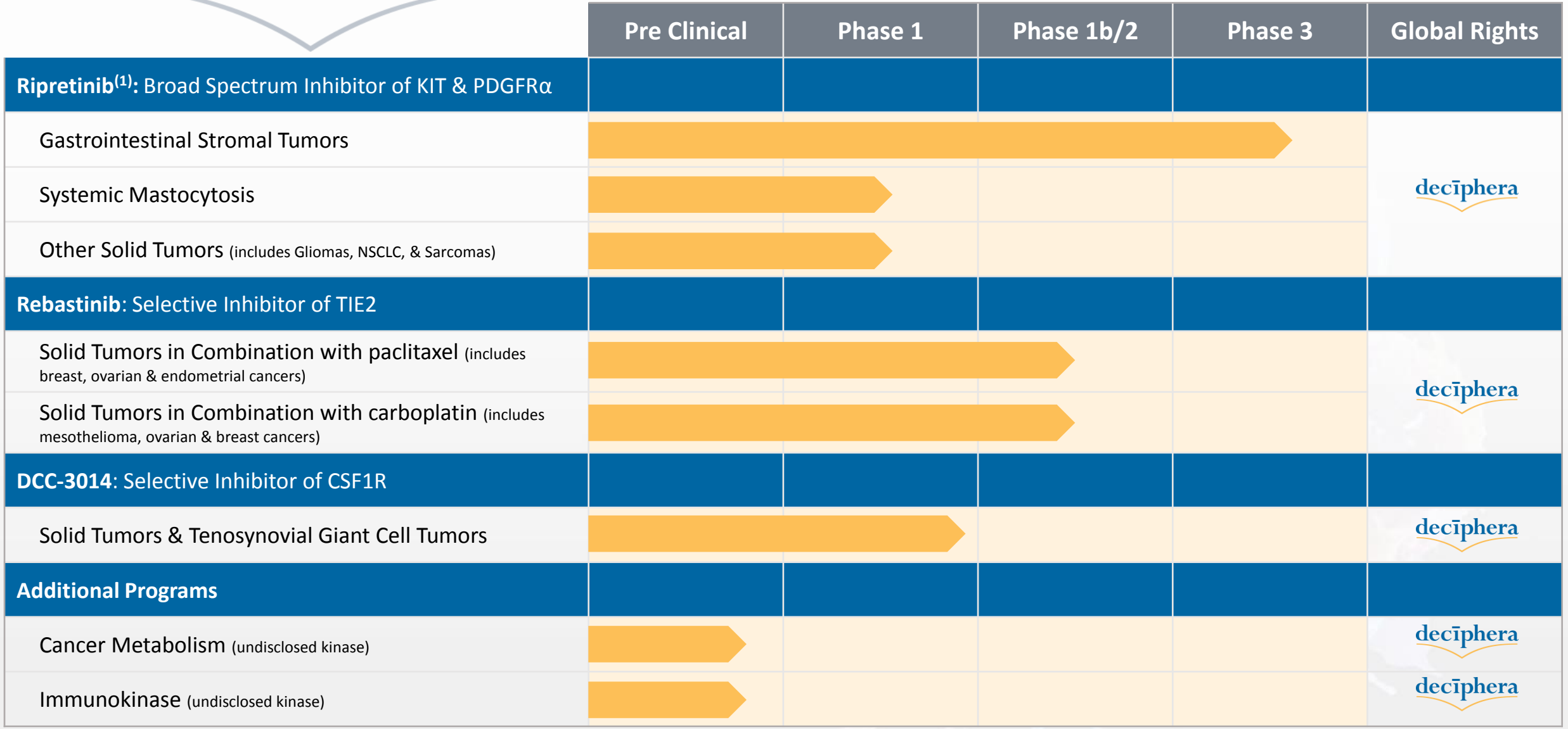
Proprietary Kinase Switch Control Inhibitor Platform

- Broad activity against disease-initiating and drug-resistant mutant kinases
- Kinase-selective and spectrum-selective profiles
- Drug discovery engine that fuels long-term growth

Strong Pipeline of Tumor-Targeted and Immunokinase Programs

- Ripretinib: Broad spectrum KIT and PDGFRα inhibitor in two Phase 3 studies
- Rebastinib: Highly potent and selective TIE2 inhibitor
- DCC-3014: Highly selective and potent CSF1R inhibitor

Strong Clinical Stage Oncology Pipeline of Novel Kinase Inhibitors



Notes: (1) DCC-2618.

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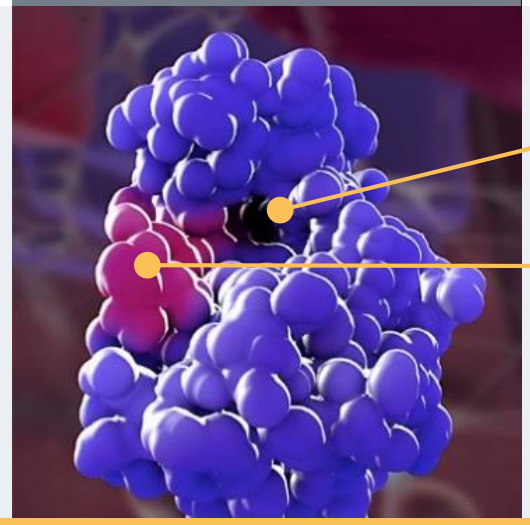
Our Proprietary Kinase Switch Control Platform

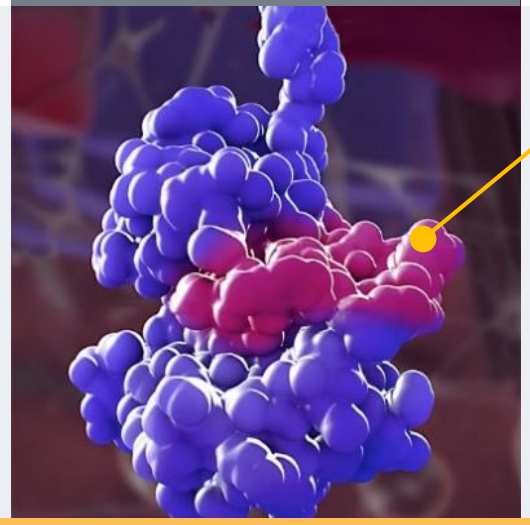
Kinase Switched "Off"



Switch pocket

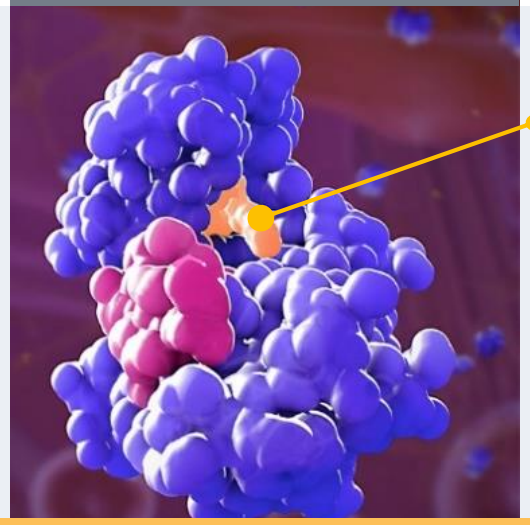
Activation switch "Off"

Kinase Switched "On"



Activation switch "On"

Deciphera Switch Control



Switch control inhibitor embeds deeply into switch pocket

Inhibits switch activation

Advantages of Switch Control Inhibitors

Tumor-Targeted Programs	**Broader Activity** **Enhanced Durability**	Inhibit wild-type and many or all mutant forms of targeted kinases Resilient to gain-of-function mutations and drug resistance
Immunokinase Programs (Macrophage Checkpoints)	**Engineered Profiles** **Superior Binding**	Highly selective or target multiple kinases at desired potency More potent and more durable; resilient to ATP concentration

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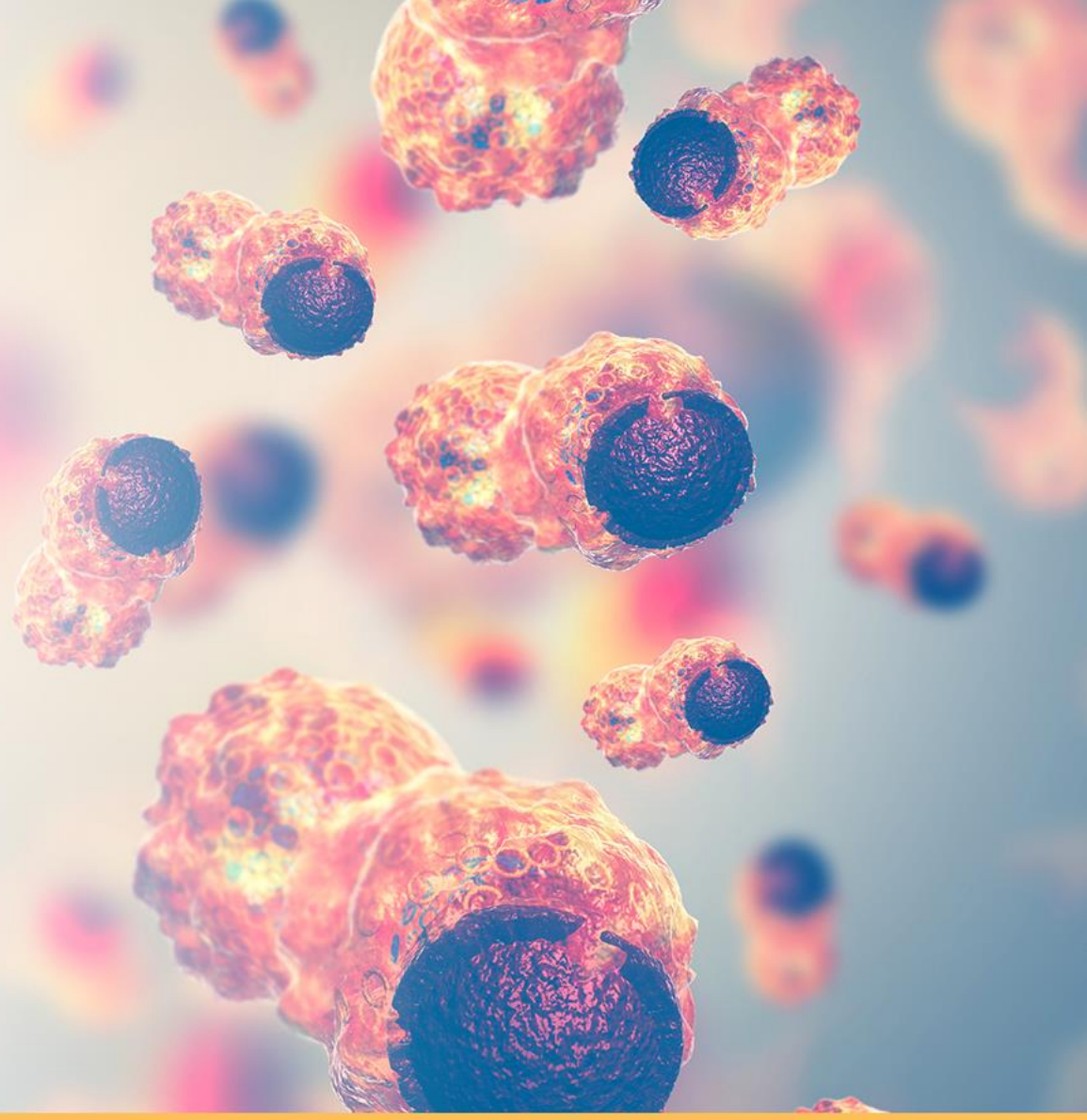
Ripretinib (DCC-2618): Broad Spectrum KIT & PDGFRα Inhibitor



Ripretinib (DCC-2618): A Potent, Broad Spectrum KIT and PDGFRα Inhibitor

Ripretinib Summary

Highly potent small molecule KIT and PDGFRα inhibitor

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

Fast-to-Market strategy with significant opportunity for label expansion

- Phase 3 pivotal trial in ≥4th Line GIST, read out expected mid-2019
- Phase 3 pivotal trial in 2nd Line GIST initiated December 2018
- Phase 1 expansion study ongoing

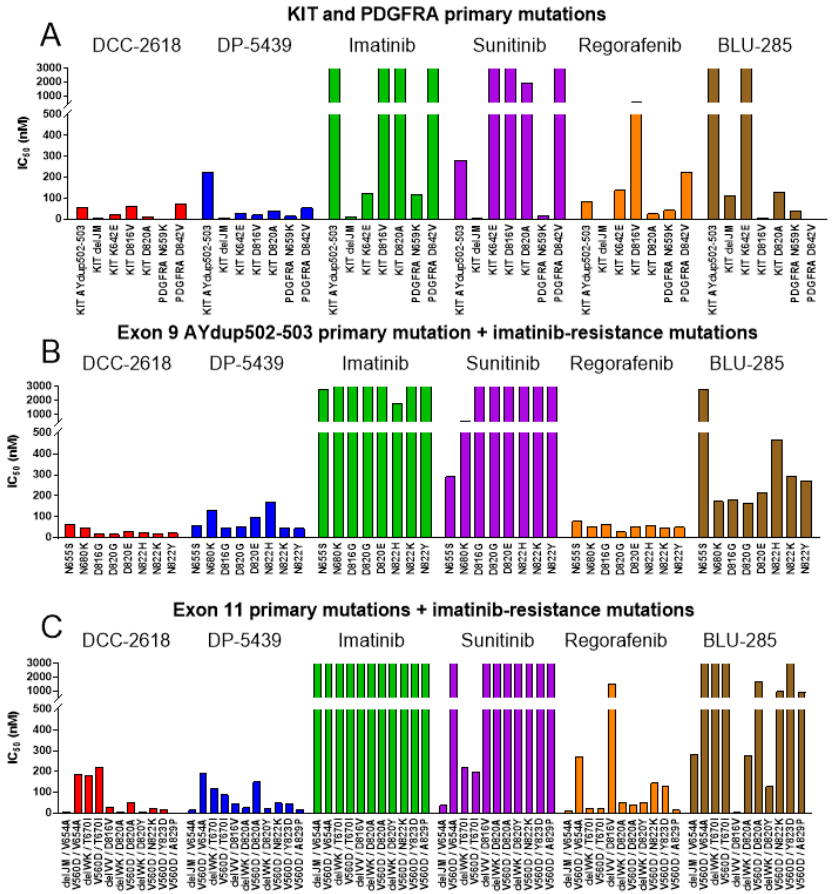
Clinical proof-of-concept in GIST ≥100 mg daily

- **2nd & 3rd Line: mPFS 40 weeks, 21% ORR Best Response, 81% DCR @3 months**
- ≥4th Line: mPFS 24 weeks, 9% ORR Best Response, 66% DCR @ 3 months

Favorable tolerability profile

- Doses up to 400 mg total per day with no MTD

IP: Composition and method of use (2032)



Preclinical Profile

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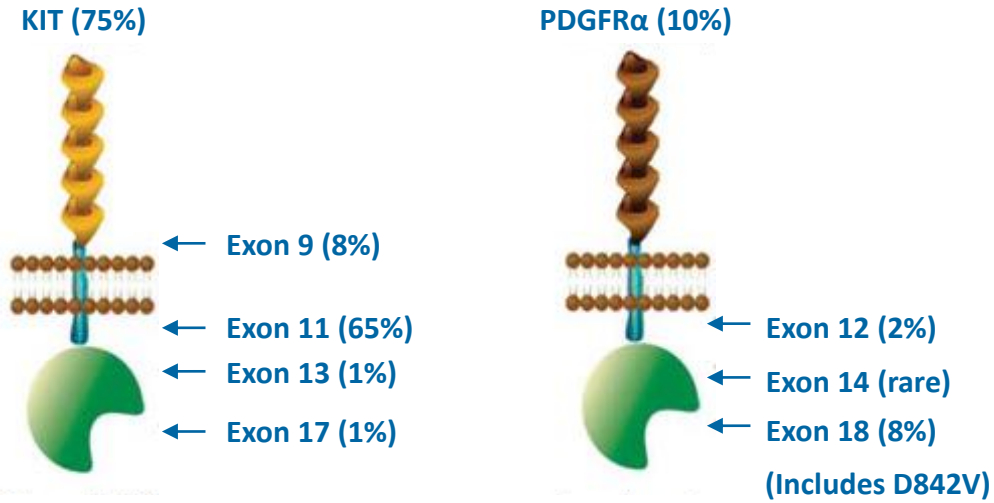
GIST: A Polyclonal Disease Driven by KIT and PDGFRα

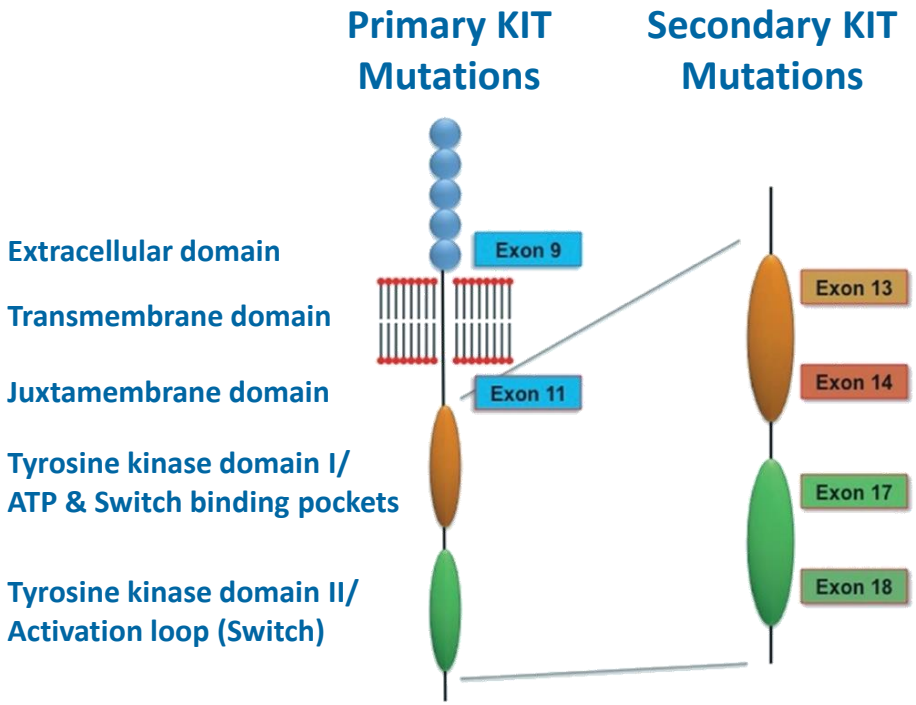
Primary KIT and PDGFRα Mutations in GIST Patients

KIT and PDGFRα Mutations in GIST
"Wild-type" tumors: 15%



Corless; *M. Pathology* 2014

Multiple Secondary KIT Mutations Produce Drug Resistance

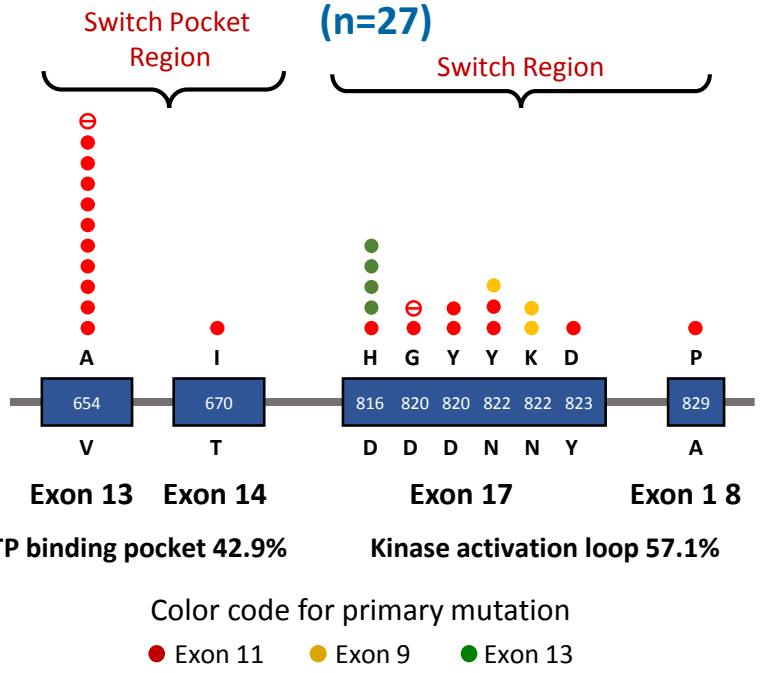


George; *Ther. Adv. in Medical Oncology* 2014

Multiple Drug-Resistant Secondary Mutations in GIST

Drug-Resistant Secondary KIT Mutations in GIST Patients Span Exon Regions 13-18

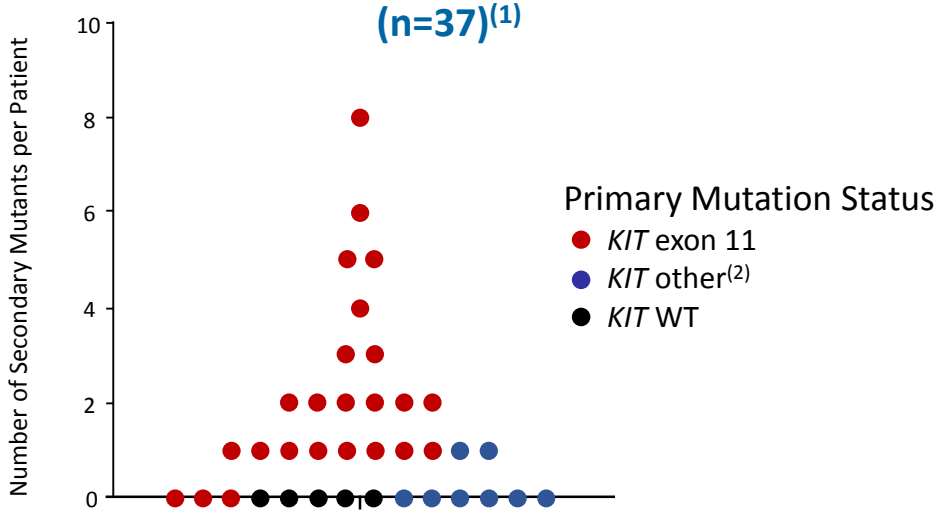
Distribution of secondary KIT mutations (n=27)



Fletcher; *J. Pathol* 2008

High Degree of Tumor Mutation Heterogeneity in GIST Patients

Number of secondary mutants detected per patient (n=37)[1]



- ≥2 secondary mutations were observed in 35% (13/37) of patients.
- 54% (13/24) of patients with KIT exon 11, with 1 patient harboring 8.

Heinrich, *Abstract 10517 ASCO 2015*

Notes: (1) Number of unique secondary mutants per patient observed across all samples analyzed; (2) Includes exon 9 (n=6), exon 13 (n=1), and exon 17 (n=1).

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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] (months)	24.0	6.1	4.8
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; (2) Time point not disclosed; (3) Time point at 12 weeks.

Ripretinib (DCC-2618): Global Pivotal Phase 3 GIST Programs

Fully Enrolled; Data Expected Mid-2019

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

Initiated December 2018

intrigue

Prior imatinib therapy[2]

1:1 Randomization
Open Label
n=358

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

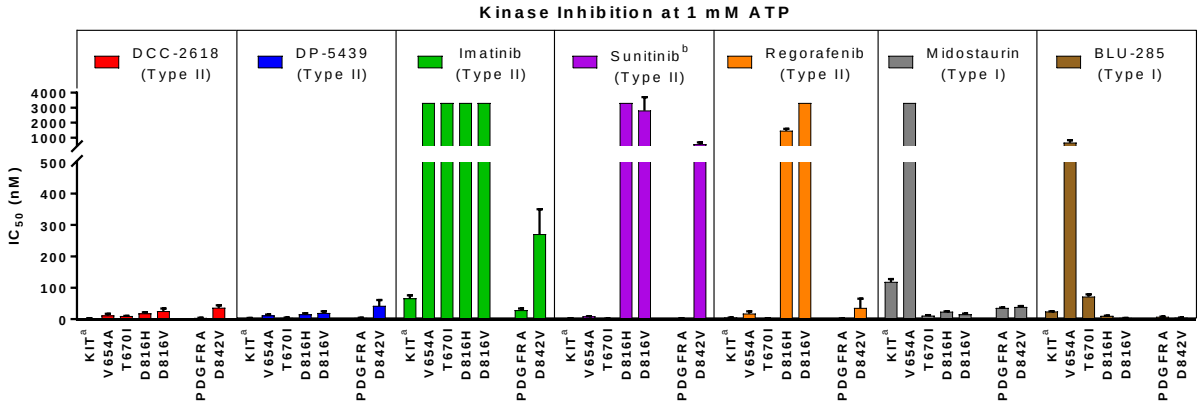
Primary Endpoint for Approval = PFS

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.

Ripretinib (DCC-2618): Broad Mutational Coverage in KIT and PDGFRα

Ripretinib Broadly Inhibits KIT and PDGFRα Mutations In Enzyme Assays

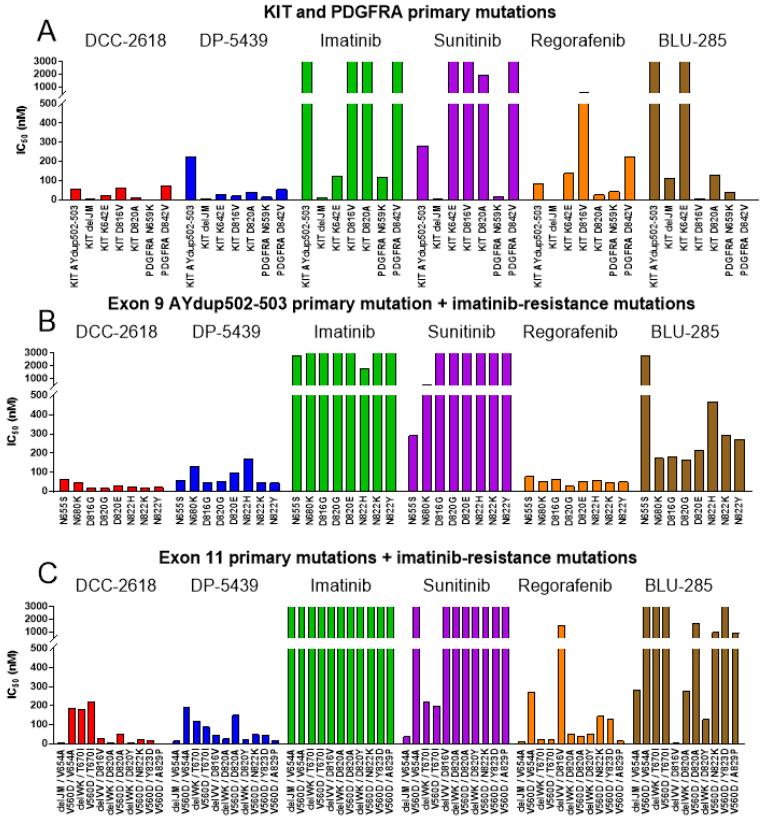


[a]Phosphorylated on the juxtamembrane domain. [b]Sunitinib is Type I-like but binds to KIT in the inactive Type II conformation.

- Ripretinib broadly inhibits KIT mutations in exons 9, 11, 13, 14, 17, 18, and a PDGFRA exon 18 mutant.
- Other Type II inhibitors do not block KIT exon 17 mutations, including D816V KIT.
- Type I inhibitors exhibit weak activity for primary KIT mutations in exon 9, exon 11 V560D, and exon 13 K642E and for secondary KIT mutations in exons 13 and 14.

Mutation	Exon
KIT V654A	13
KIT T670I	14
KIT D816H	17
KIT D816V	17
PDGFRA D842V	18

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.

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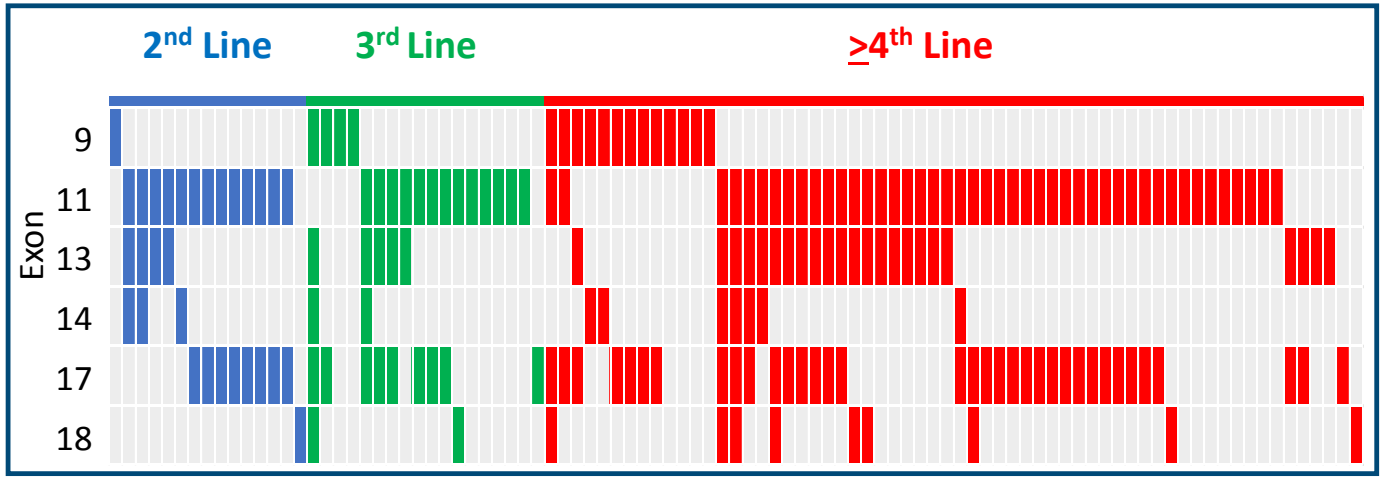
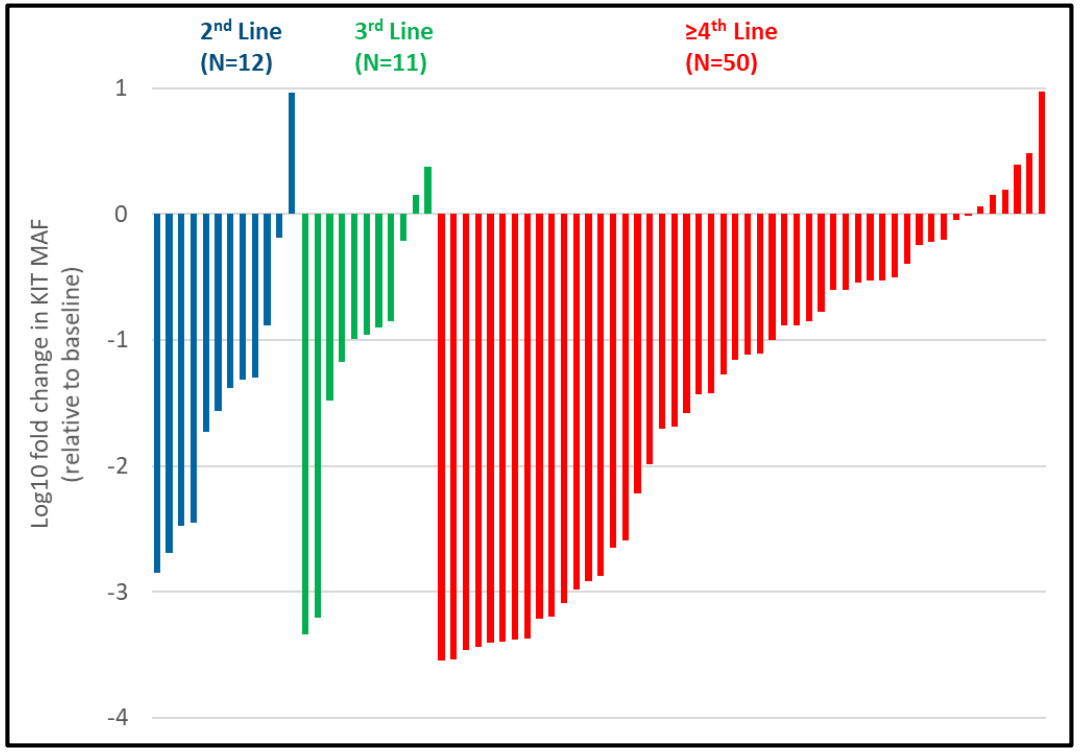
Ripretinib (DCC-2618): Clinical Validation of Broad KIT Mutant Profile

KIT Mutations in ctDNA (n=95)
in 131 GIST patients by Line of Therapy



Each column represents an individual GIST patient and each filled entry on rows indicates detection of one or more mutations in Exon 9, 11, 13, 14, 17 and 18.

Cumulative Reductions in Circulating MAF of KIT Exons 9, 11, 13, 14, 17 and 18 by Lines of Therapy (n=73)[2]
(Note log scale: -1 = 10-fold reduction, -2 = 100-fold reduction)



- Secondary KIT mutations in exons 13, 14, 17 and 18 in patients with 2nd to ≥ 4th line GIST

- 78% achieved more than 50% KIT MAF reduction
- 48% were KIT negative on treatment

Notes: (1) Based on liquid biopsy data from preliminary Phase 1 data presented at ASCO on June 2, 2018; (2) Based on data from 73 patients with detectable KIT mutations at baseline.

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Ripretinib (DCC-2618): 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	Enrollment Continuing	New Cohorts Initiated
2nd – 3rd Line GIST n=55	Systemic Mastocytosis	NSCLC, Germ Cell & Penile
4th Line GIST n=40	Malignant Gliomas	Melanomas
>4th Line GIST n=35		Soft Tissue Sarcomas
Other Solid Tumors n=10		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 (DCC-2618): Phase 1 Demographics

	ESMO 2018
GIST Patients ≥100 mg /d	**n=178**
Age (years), median (range)	61 (27-87)
GIST Subtype	n (%)
KIT-driven	168 (94%)
PDGFRα-driven	9 (5%)
SDH deficient	1 (1%)
Line of Therapy	n (%)
2nd Line	38 (21%)
3rd Line	29 (16%)
≥4th Line(1)	111 (62%)
DCC-2618 Dose	n (%)
150 mg QD	142 (80%)
Other (100 mg/d – 400 mg/d)	36 (20%)

Notes: (1) Mean number of prior regimens for ≥4th line patients was 3.6.

Ripretinib (DCC-2618): Preliminary Efficacy by Line of Treatment @ ≥100 mg Daily

Line of Therapy	Total Patients	Active[1]	Disease Control Rate @ 3 Months[1]	ORR[1][2]	mPFS[1]
2nd Line	38	61%	79%	18%[3]	42 weeks
3rd Line	29	59%	83%	24%	40 weeks
≥4th Line	111	44%	66%	9%[4]	24 weeks
2nd & 3rd Line	67	60%	81%	21%[3]	40 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).

Ripretinib (DCC-2618): Preliminary ORR by Best Response @ ≥100mg Daily

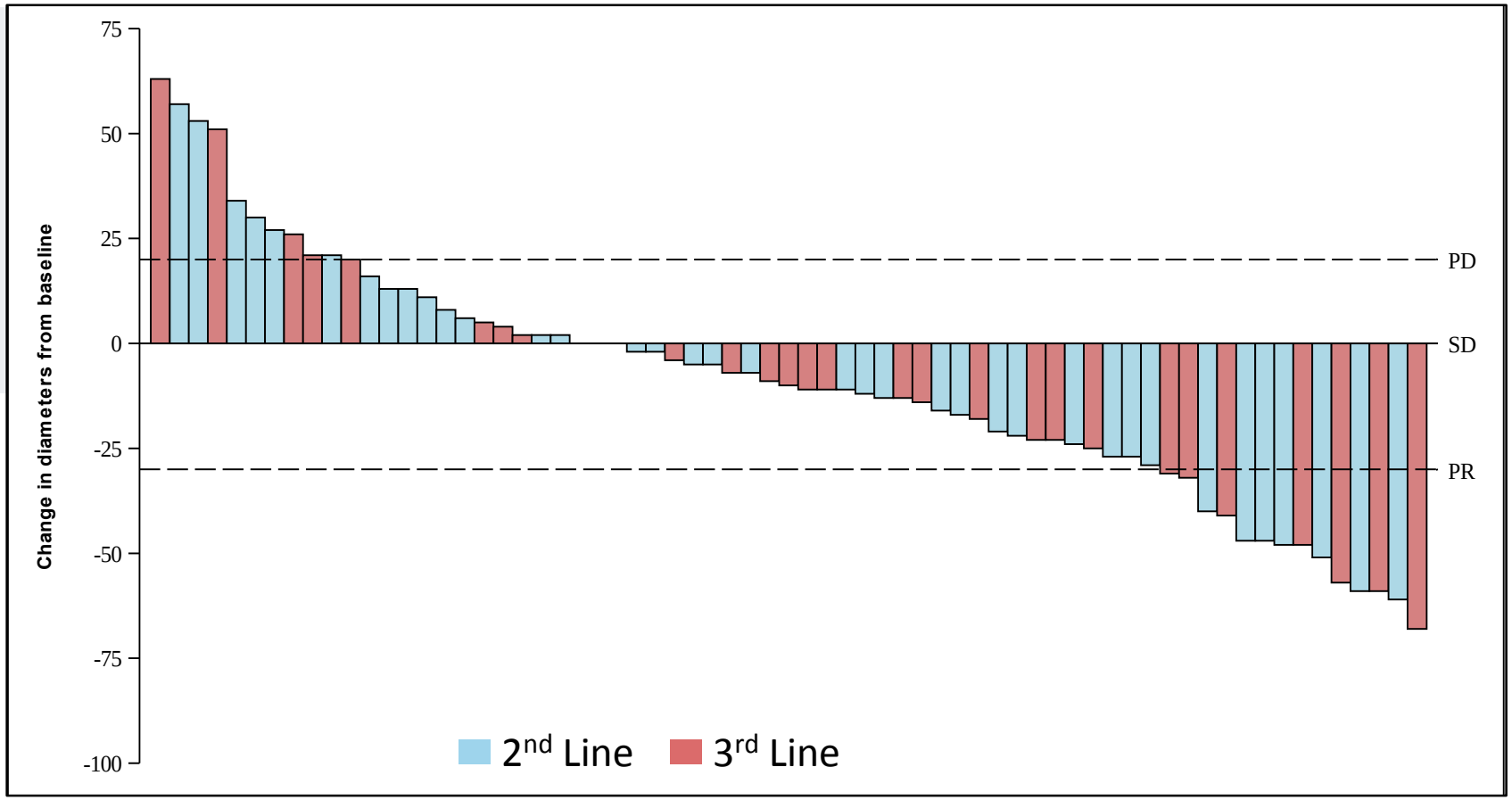
21% ORR (ripretinib 2nd & 3rd Line)

7% ORR (sunitinib)(3)

5% ORR (regorafenib)(3)

Preliminary ORR data with ripretinib @ ≥ 100mg daily exceeds registration trial data for approved 2nd & 3rd line therapies

Best Response per RECIST(1)(2)
KIT & PDGFRα ≥ 100 mg/d (n=67)



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; (3) RECIST data per central review in registration trial.

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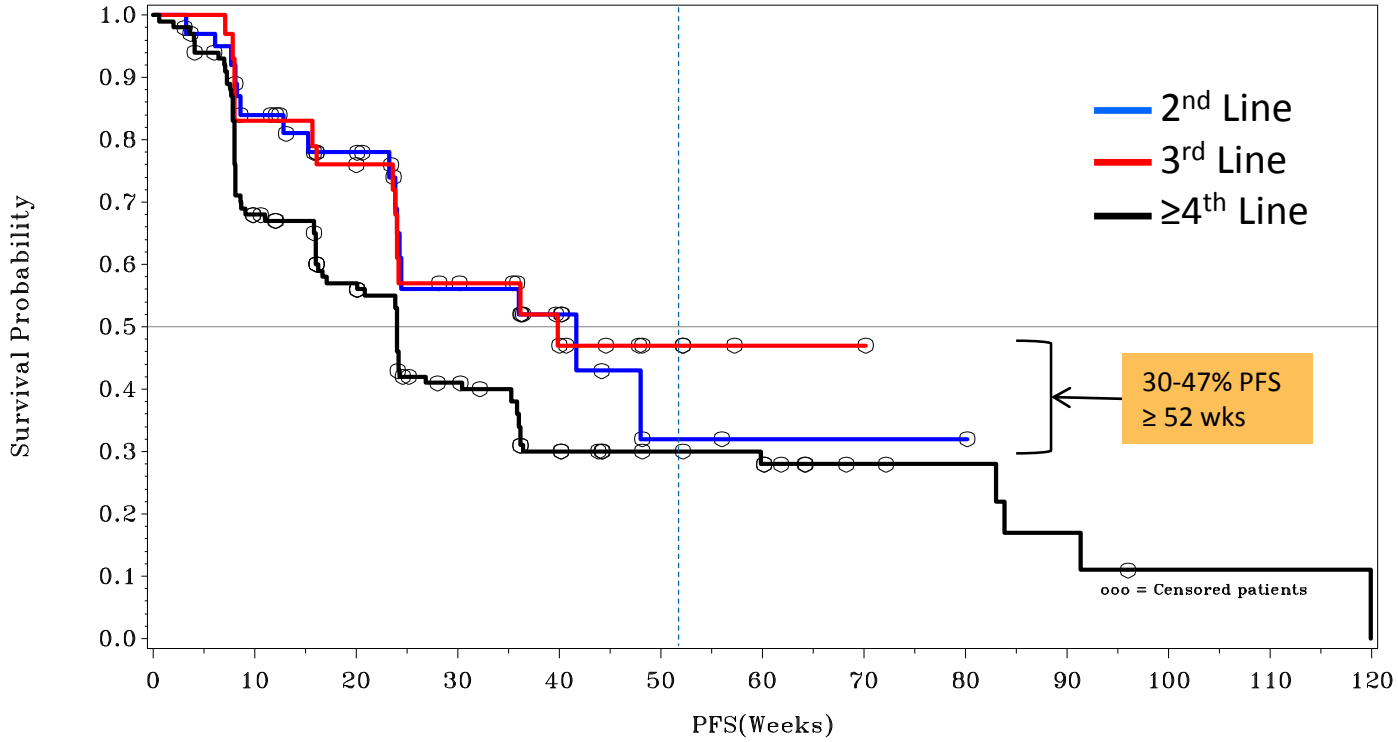
Ripretinib (DCC-2618): Preliminary mPFS @ ≥100 mg Daily

Line of Therapy	mPFS (ripretinib)	Number Censored
2	42 weeks	22 (58%)
3	40 weeks	15 (52%)
≥4	24 weeks	40 (35%)
2 & 3	40 weeks	37 (55%)

24 weeks mPFS (sunitinib)[2]

21 weeks mPFS (regorafenib)[2][3]

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

Preliminary mPFS data with ripretinib @ ≥100mg daily exceeds registration trial data for approved 2nd & 3rd line therapies

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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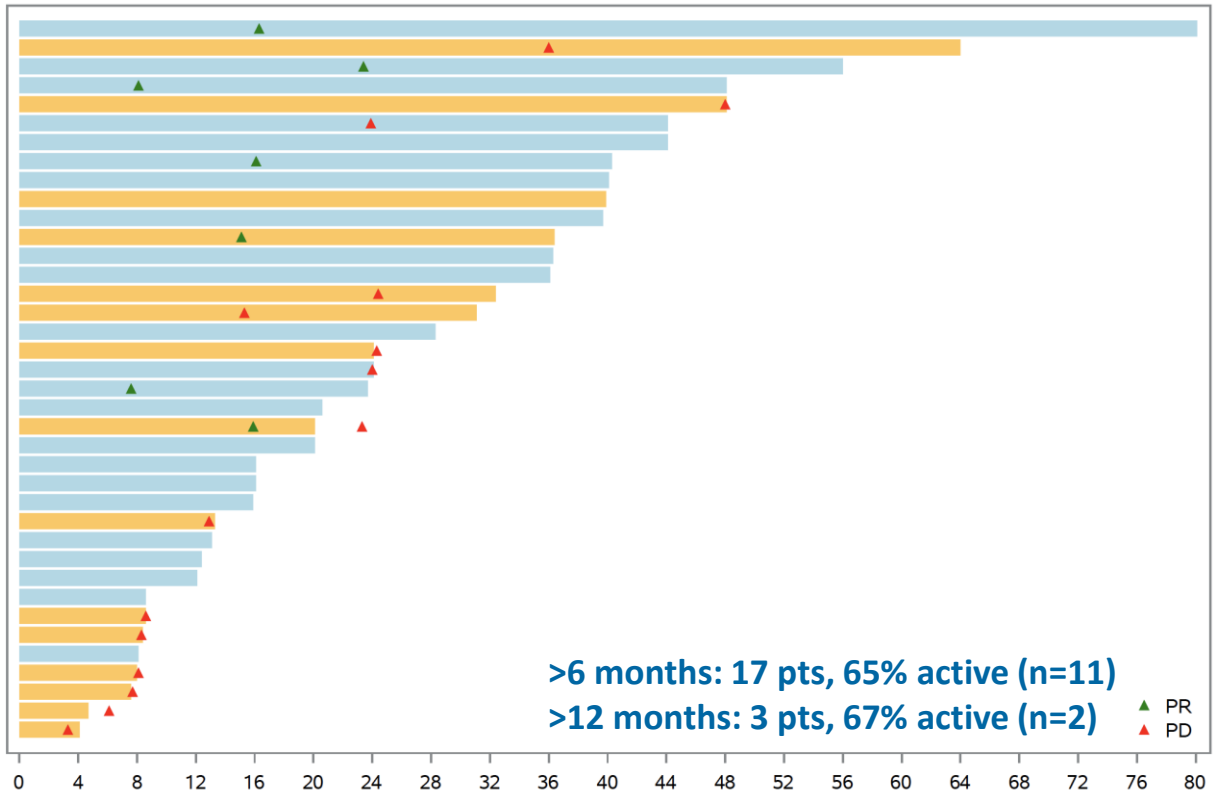
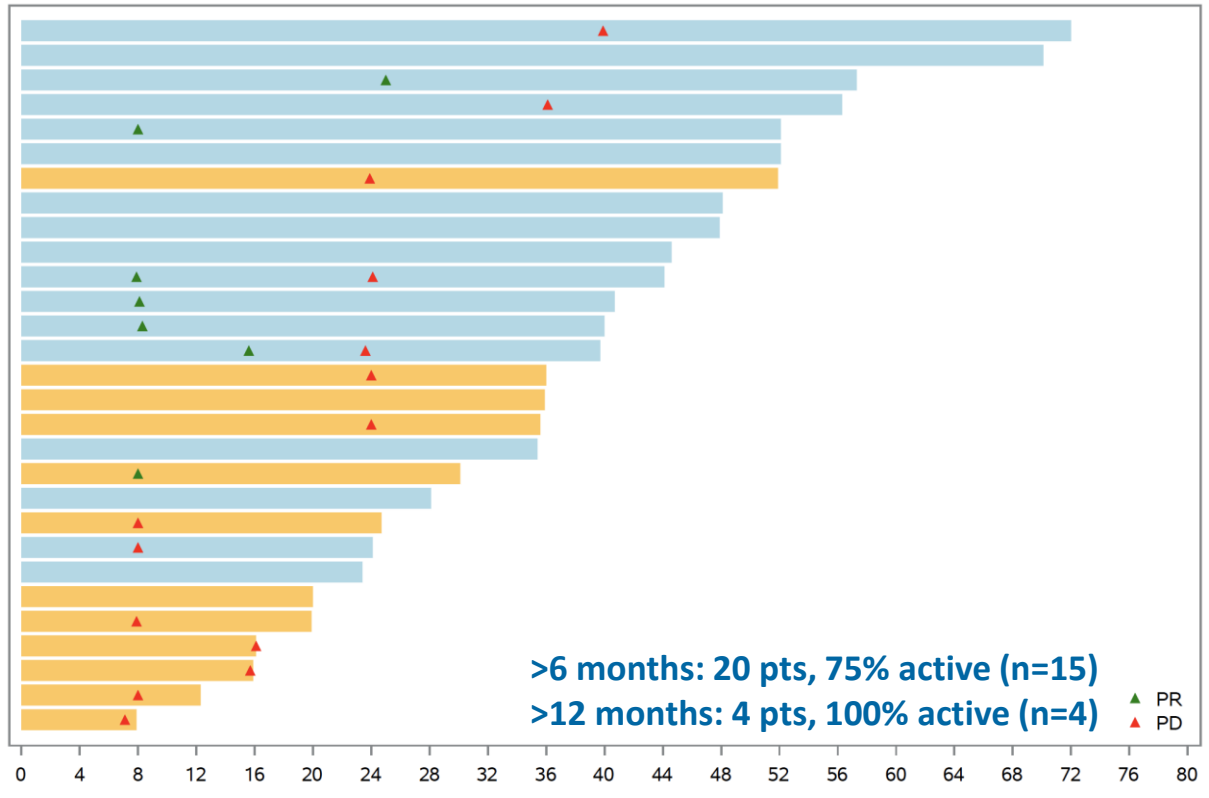
Ripretinib (DCC-2618): Preliminary Durability Data @ ≥100 mg Daily

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 (DCC-2618): 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 (DCC-2618): Opportunity in Advanced Systemic Mastocytosis (SM)

Overview

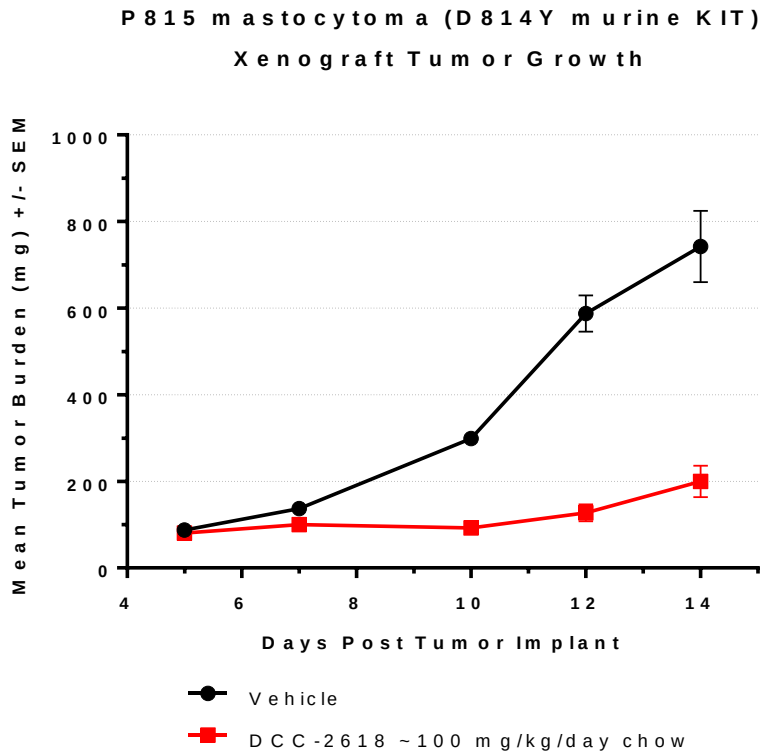
- Potent inhibitor of KIT D816V mutation that drives SM
  - 25 nM IC_{50} is nearly 1/100th of 2,000 nM RP2D mean, steady state plasma C_{min}
- Activity demonstrated in multiple SM models
  - P815 mastocytoma murine model and patient-derived SM cells (*in vitro*)
- Clinical dose response relationship with serum tryptase levels in non-SM patients
- Favorable tolerability profile important across spectrum of SM
- Currently enrolling SM patients in Phase 1 expansion cohort

	D816V Exon 17	P815 Mastocytoma (mouse D814Y KIT) Exon 17	HMC1.2 Mast Cells (V560G/D816V) Exons 11/17
	IC_{50} (nM)[2]	Cell Proliferation IC_{50} (nM)	Cell Proliferation IC_{50} (nM)
Ripretinib	25	23	47
DP-5439	19	41	47
Imatinib	>3,300	>3,000	>3,000
Sunitinib	2,800	174	1,410
Regorafenib	>3,300	>3,000	>3,000
Midostaurin	15	151	270
BLU-285	3.4	22	147

Notes: (1) green < 50 nM; yellow >50 and <100 nM; blue > 100 nM and < 500 nM; red > 500 nM ; (2) All enzyme assays run at 1 mM ATP.

Source: Abstract #3925 AACR 2018.

P815 Mastocytoma Model of SM



100 mg/kg/day in chow equivalent dose to 50 mg/kg BID by oral gavage

Source: Unpublished company data.

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Ripretinib (DCC-2618): Estimated 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
Advanced Systemic Mastocytosis[3]	~1,400	~2,600	~4,000

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

[3] Cohen *et al*. British Journal of Haematology, 2014, 166, 521–528

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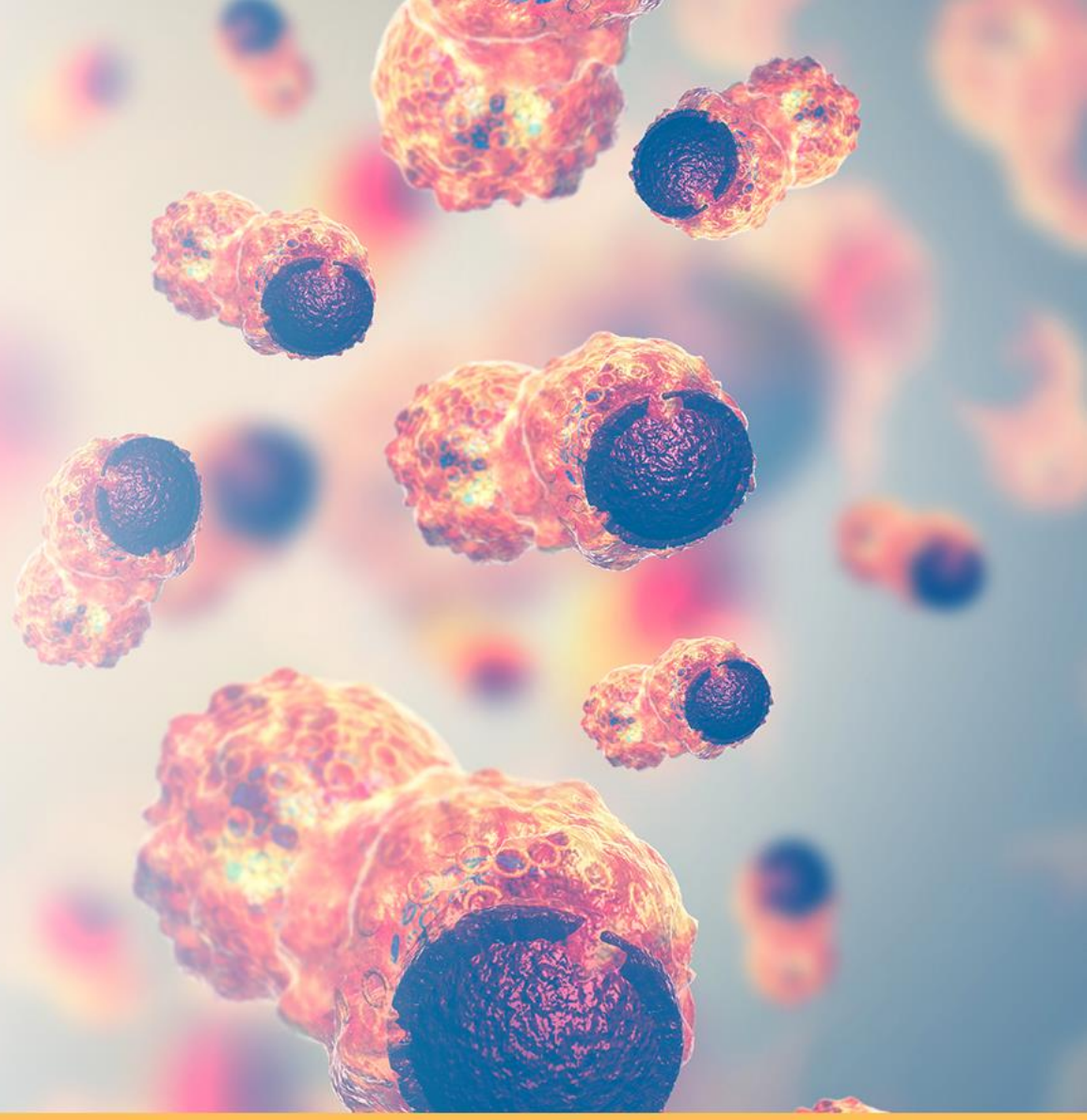
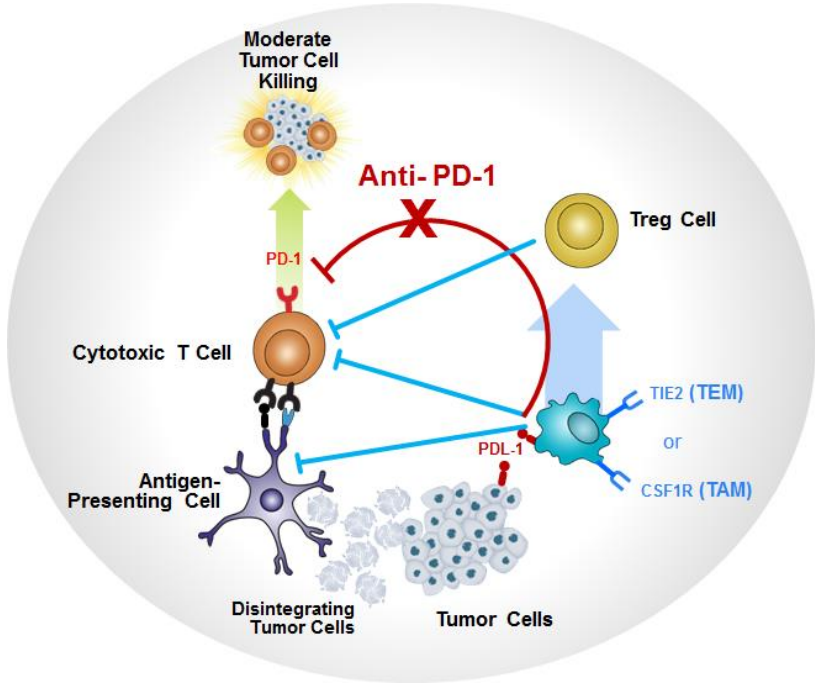
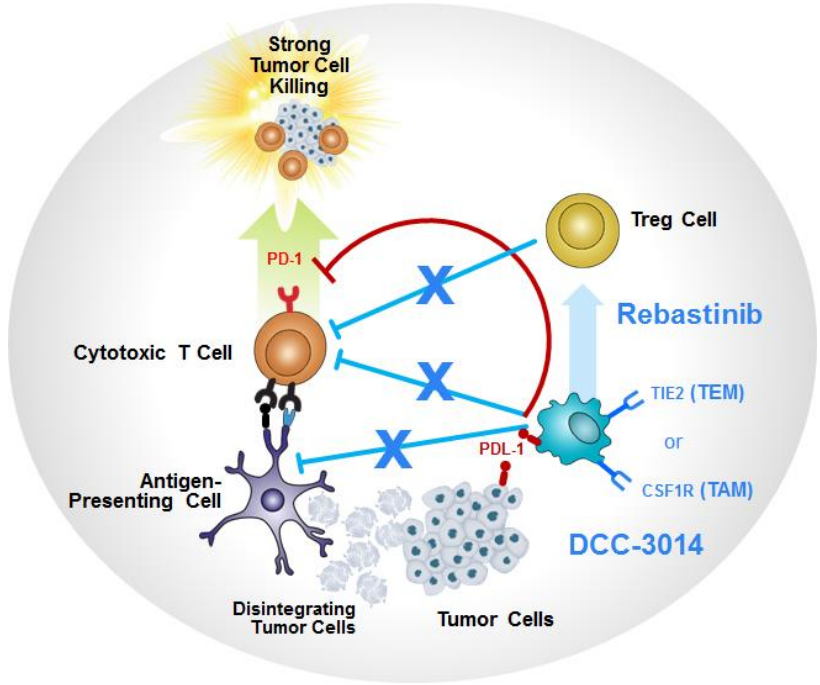
Immunokinase Programs Targeting Macrophage Checkpoints: Rebastinib and DCC-3014



Immunokinase Programs: Rebastinib and DCC-3014

Combining T-Cell and Macrophage Checkpoint Inhibitors Targeting T-Cells and Macrophages to Produce Strong Tumor Cell Killing

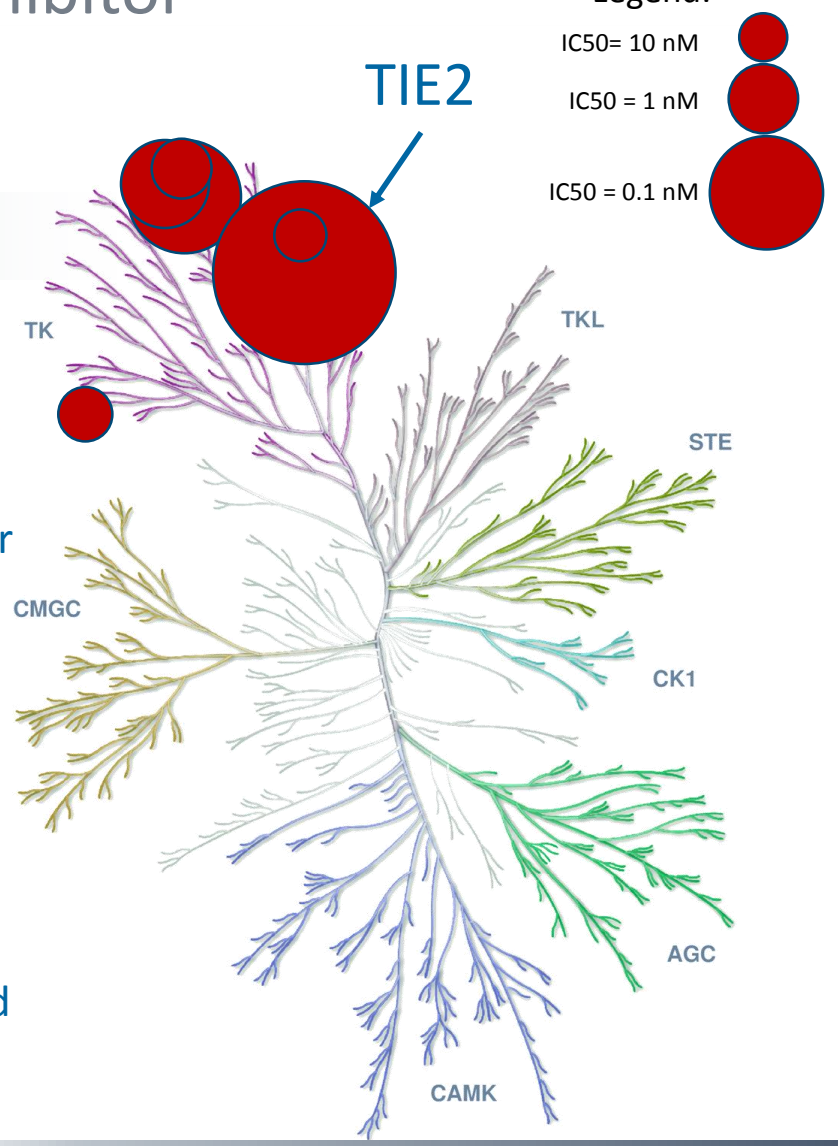




Rebastinib: A Highly Potent and Selective TIE2 Inhibitor

Rebastinib Summary

- Potent, small molecule inhibitor of TIE2
- Phase 1 study completed
  - Patients with relapsed/refractory chronic (BCR-ABL+) or AML (FLT3-ITD)
  - MTD determined based on targeting BCR-ABL. 100x more potent inhibitor of TIE2. Lower dosing to be used for future development.
- Preclinical anti-tumor activity
  - Single agent and I/O or chemo combination
- TIE2 microenvironment mechanisms
  - Tumor vascularization, dissemination, metastasis, immunotolerance
- Development Status
  - Two ongoing Company-sponsored chemo combo trials with paclitaxel and carboplatin
- IP: Composition (2027) and method of use (2034)



Kinome Profile

Notes: All kinases within 100-fold of TIE2 IC50 (0.058 nM) are shown.
Includes enzyme data at low ATP and 4 mM ATP, and cellular data.

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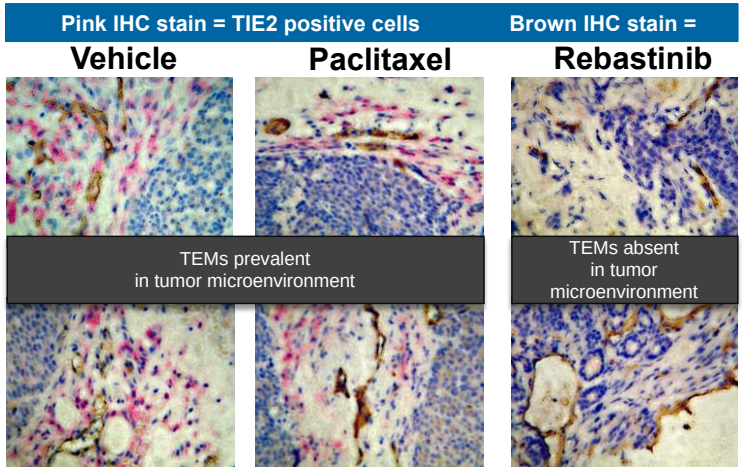
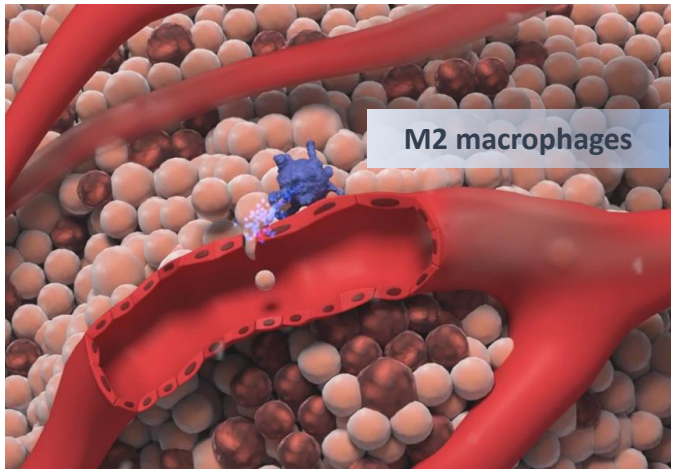
Rebastinib: Potential Benefits in Combination with Chemotherapy

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

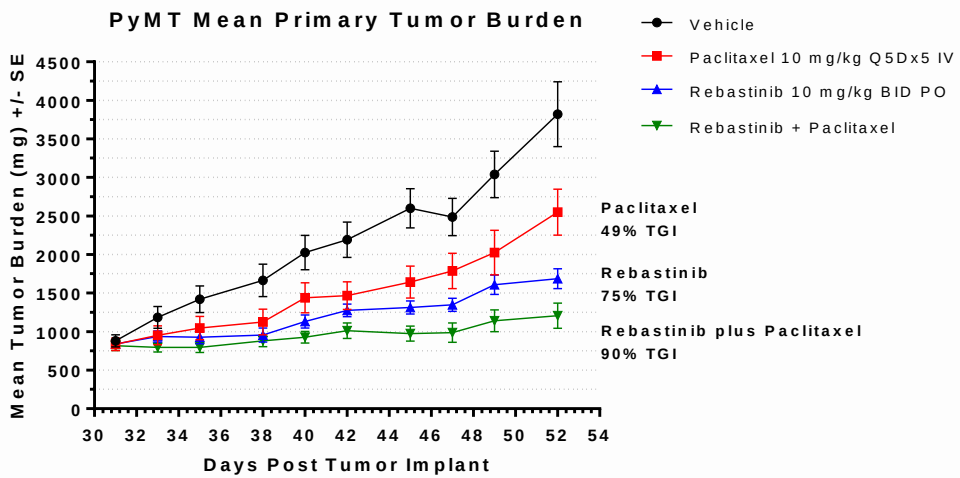


- M2 macrophages serve as pumps for tumor cell intravasation and metastasis
- Rebastinib is designed to block rebound angiogenesis and inhibit M2 macrophages

Rebastinib: Rationale for Use in Combination Therapies

Rebastinib Inhibits Growth of Breast Tumors in PyMT Mouse Model Alone and in Combination With Paclitaxel

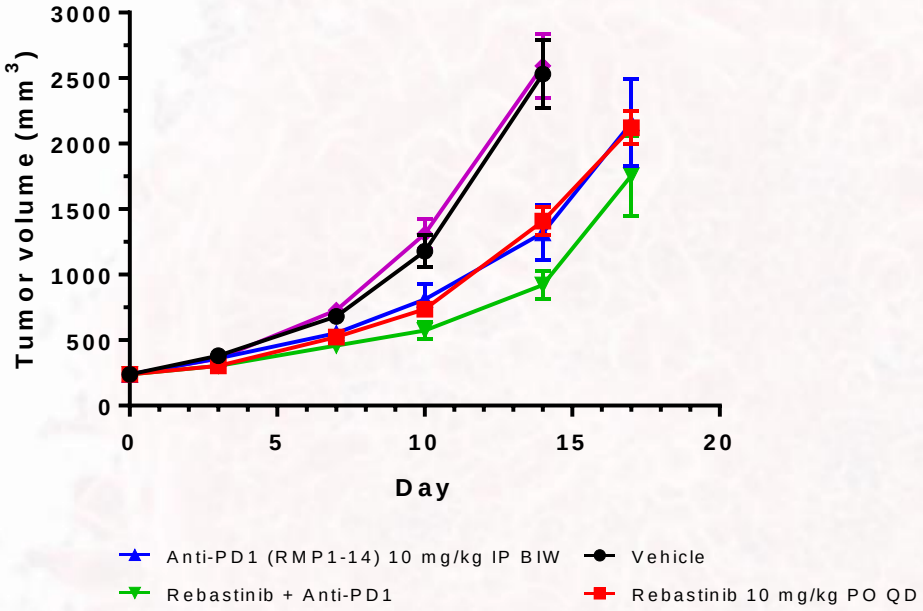
PyMT Mean Primary Tumor Burden



Notes: *TGI normalized to starting tumor size: Paclitaxel (49%TGI*) Rebastinib (75%TGI*); Rebastinib plus Paclitaxel (90%TGI*).

Rebastinib Inhibits Growth of Breast Tumors in PyMT Mouse Model Alone and in Combination With PD-1

PyMT



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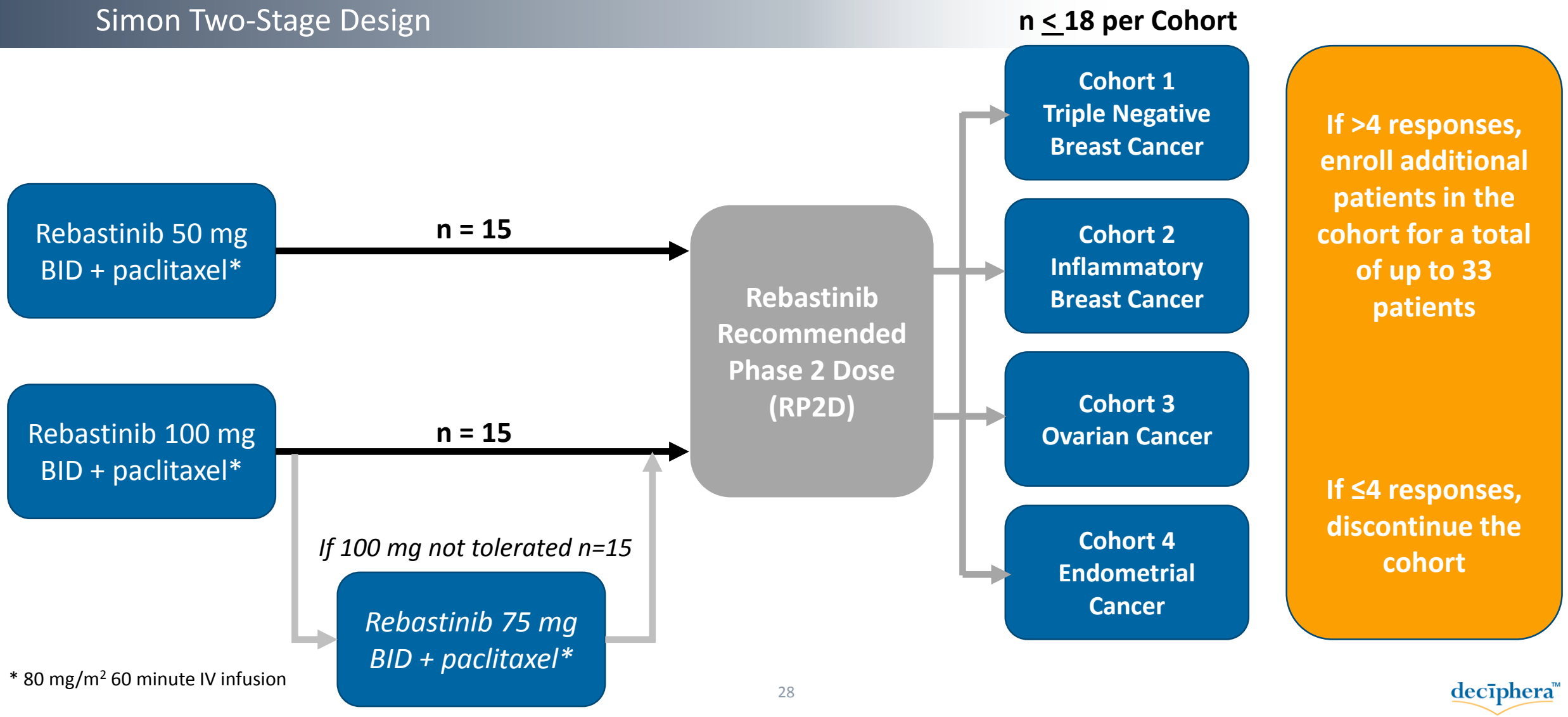
Rebastinib: Phase 1b/2 Study Combination with Paclitaxel

Simon Two-Stage Design



* 80 mg/m^2 60 minute IV infusion

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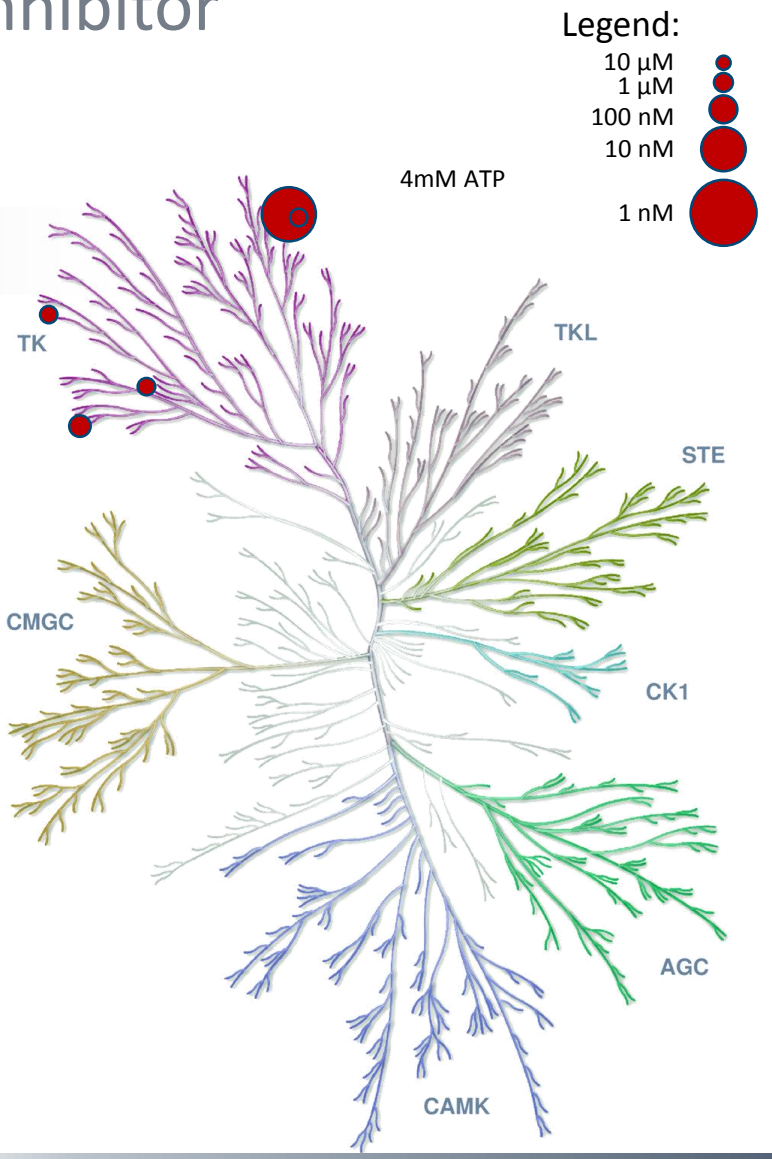
DCC-3014: A Highly Selective and Potent CSF1R Inhibitor

DCC-3014 Summary

- Highly selective, potent, small molecule CSF1R inhibitor
  - 100x selectivity over closest kinases
- Preclinical anti-tumor activity & CSF1R MOA
  - Single agent and in combination with anti-PD1
  - Inhibited immunosuppression via reduction of TAMs, increases in cytotoxic T cells and decreases in T_{reg} cells
- Ongoing Phase 1 escalation trial demonstrated mechanistic Proof of Concept (mPoC)(1)
  - Material reductions in circulating CSF1R+ macrophages mPoC
  - PK analysis demonstrated dose-proportional exposure that we believe supports twice-weekly maintenance dosing after five-day loading dose regimen
  - Generally well tolerated at doses of up to 30 mg in patients receiving twice weekly maintenance/five-day loading regimen
    - No DLTs; no DCC-3014 related G3/4 TEAEs in ≥ 10% patients
  - Study expanded to include patients with Tenosynovial Giant Cell Tumors (TGCT)
- IP: Composition and method of use (2034)



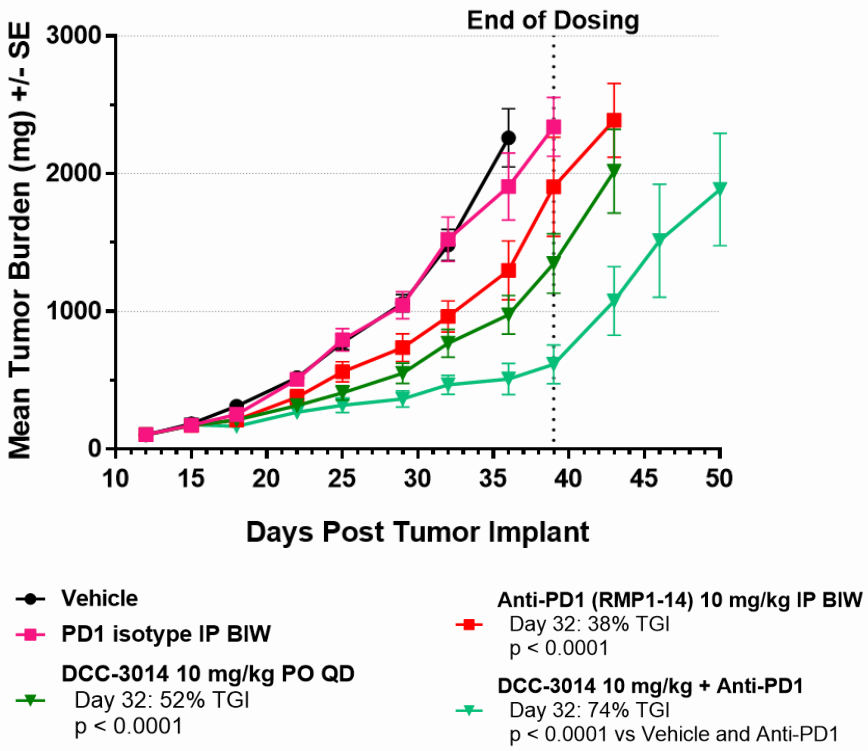
Kinome Profile

Notes: Results from preliminary Phase 1 data reported in January 2019.

DCC-3014: Rationale for Combination with PD-1 Inhibitors

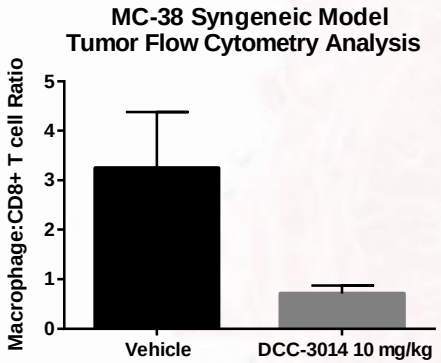
DCC-3014 Inhibits Growth of Colorectal Tumors in MC-38 Mouse Model Alone and in Combination with anti-PD-1

MC-38 Primary Tumor Growth

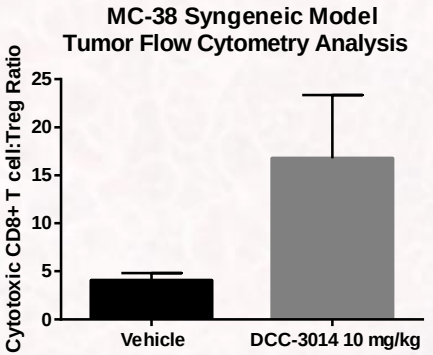


DCC-3014 Reverses Immunosuppression in MC-38 Mouse Colorectal Tumor Model

Decrease in ratio of TAMs to CD8 cytotoxic T Cells



Increase in ratio of CD8 cytotoxic T Cells to T_{reg} cells



Recent & Upcoming Milestones

	2018	2019
Ripretinib (DCC-2618)	✓ INVICTUS (≥4th Line GIST): Pivotal Phase 3 Initiated & Fully Enrolled ✓ INTRIGUE (2nd Line GIST): Pivotal Phase 3 Initiated ✓ AACR & EORTC: Phase 1 Safety/PK Data & Preclinical Data ✓ ASCO & ESMO: Phase 1 GIST Updates	INVICTUS (≥4th Line GIST: Pivotal Phase 3 Results (Expected Mid-2019) Phase 1 Expansion Data
Rebastinib	✓ Phase 1b/2 Paclitaxel Combination Initiated	✓ Phase 1b/2 Carboplatin Combination Initiated Phase 1b/2 Initial Combination Data
DCC-3014		✓ Phase 1 Dose Escalation Update Phase 1 Initial Data
Discovery Platform	✓ Initiated 2 New Research Programs	Select Clinical Candidate & Initiate IND Studies

NASDAQ:DCPH

Shares Outstanding (as of 12/31/18)

37.7 MM *(basic)*
43.5 MM *(fully-diluted)*

Cash & Cash Equivalents (as of 12/31/18)

$294MM

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

Corporate Summary

Well established drug class with significant growth potential

Differentiated approach to kinase inhibition

Wholly-owned pipeline with three clinical stage assets

Kinase Inhibitors: 40+ FDA-approved Drugs but Significant Opportunities Remain

- Drug resistance mutations limit rate and duration of response
- Low potency and selectivity cause poor tolerability
- Approved drugs target less than 10% of the 500+ known human kinases

Proprietary Kinase Switch Control Inhibitor Platform

- Broad activity against disease-initiating and drug-resistant mutant kinases
- Kinase-selective and spectrum-selective profiles
- Drug discovery engine that fuels long-term growth

Strong Pipeline of Tumor-Targeted and Immunokinase Programs

- Ripretinib: Broad spectrum KIT and PDGFRα inhibitor in two Phase 3 trials
- Rebastinib: Highly potent and selective TIE2 inhibitor
- DCC-3014: Highly selective and potent CSF1R inhibitor

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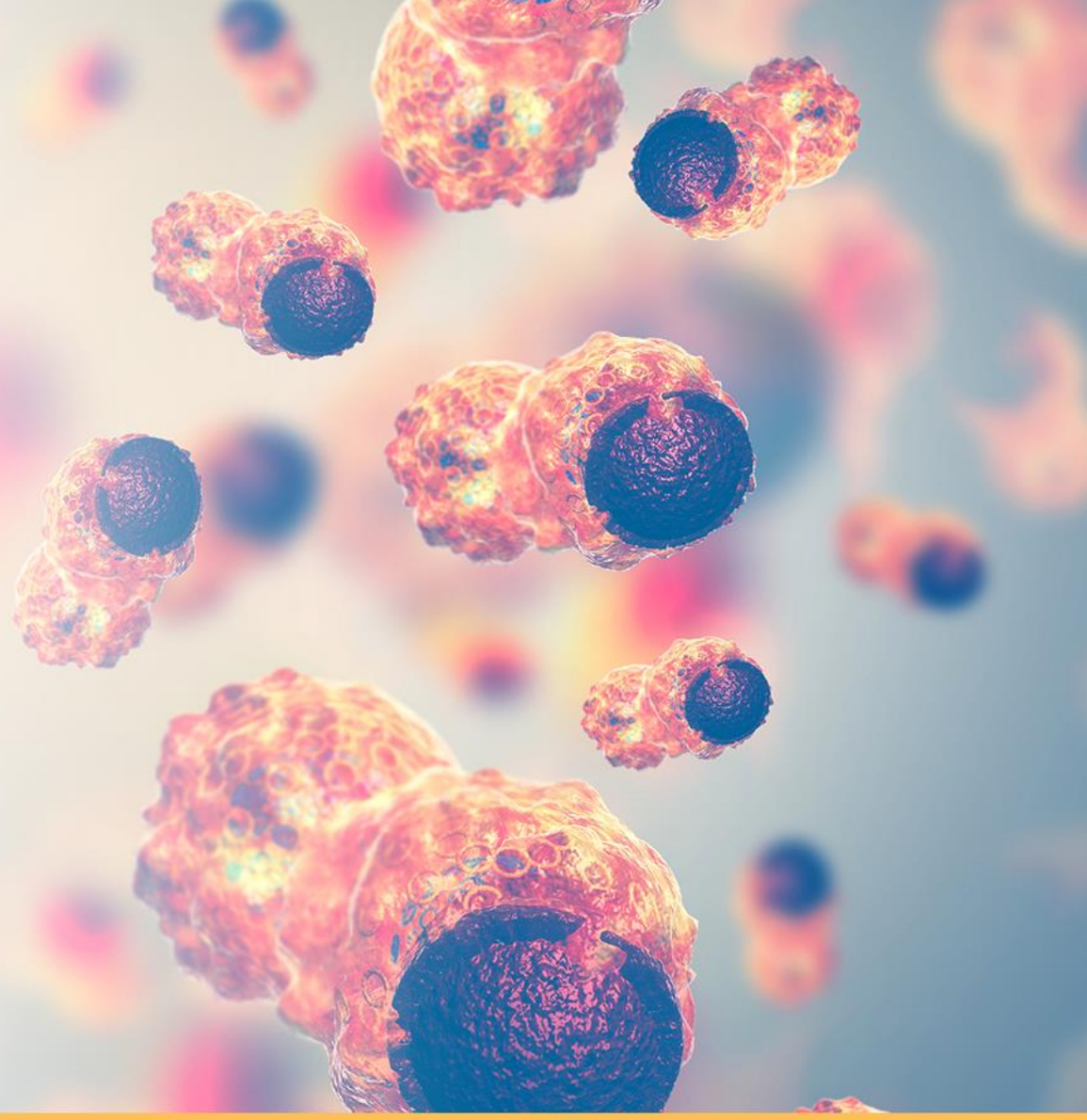
Addressing Key Mechanisms of Tumor Drug Resistance

April 2019



Thank You