One Mission, Inspired by Patients: Defeat Cancer.™

May 2023

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Forward-Looking Statements

This presentation may contain forward-looking statements that are based on our current expectations, estimates and projections about our industry, our operations and financial performance, as well as management's beliefs and assumptions. Words such as "anticipates," "expects," "intends," "plans," "believes," "seeks," "estimates," "may," "will," and variations of these words or similar expressions are intended to identify forward-looking statements. Such forward-looking statements are subject to various risks and uncertainties, including important factors that could cause actual outcomes or results to differ materially from those indicated in these statements. These statements include, without limitation, the potential for our pre-clinical and/or clinical stage pipeline assets to be first-in-class and/or best-in-class treatments, plans to continue our geographic expansion of QINLOCK in Key European markets, our planned Phase 3 INSIGHT clinical study of QINLOCK versus sunitinib in second-line GIST patients with mutations in KIT exon 11 and 17/18, our expectations regarding the aggregate potential revenue opportunity for QINLOCK, our ability to expand the market opportunity for QINLOCK in second-line GIST in our INSIGHT Phase 3 study; the vimseltinib topline readout for the pivotal Phase 3 MOTION study and phase 1/2 study of vimseltinib, each in TGCT patients; plans to initiate one or more combination cohorts in the Phase 1/2 study of DCC-3116, plans to initiate new combination studies with ripretinib in patients with GIST and encorafenib and cetuximab in patients with colorectal cancer, plans to present additional preclinical data for DCC-3116, the potential for our autophagy program to be a multi-billion dollar opportunity; submitting an IND for DCC-3084, submitting an IND for DCC-3009 in the second half of 2024; clinical studies including status, progress and results, timing for clinical data readouts, planning efforts for future clinical studies, NDA/MAA (and equivalent) filings and potential additional approvals, research and discovery efforts including the potential of our drug candidates, IND filings, regulatory designations and programs, timing and likelihood of success, plans and objectives of management for future operations, estimated patient populations, the market opportunity for our drug and drug candidates and business guidance, including discovery, clinical and regulatory milestones, cash guidance, expectation regarding our financial performance and business strategy, and the potential impact of the Inflation Reduction Act (the IRA), speak only at the time this presentation was prepared. Such statements are based upon the information available to us now and are subject to change. We will not necessarily inform you of such changes. These statements are not guarantees of future performance and are subject to certain risks, uncertainties and assumptions that are difficult to predict. Therefore, actual results could differ materially and adversely from those expressed in any forward-looking statements as a result of various factors, including, among others, the success of our commercialization efforts with respect to QINLOCK, including our launch in key European markets, our limited experience as a commercial company, our ability to obtain, or any delays in obtaining, required regulatory approvals for our drug and drug candidates, our reliance on sole source third-party suppliers, our exposure to criminal sanctions, civil penalties, contractual damages, reputational harm, and diminished profits and future earnings from our relationships with customers and third-party payors that are subject to applicable anti-kickback, fraud and abuse, and other healthcare laws and regulations, and health information privacy and security laws, our failure to obtain or maintain adequate coverage and reimbursement for new or current products, recently enacted and future legislation may increase the difficulty and cost for us to obtain marketing approval of and commercialize our drug candidates, the potential for QINLOCK or any current drug candidates, such as vimseltinib and DCC-3116, or future drug candidates, if successfully developed and approved, to cause undesirable side effects that limit the commercial profile or result in other significant negative consequences for approved products or delay or prevent further development or regulatory approval with respect to drug candidates or new indications, or cause regulatory authorities to require labeling statements, such as boxed warnings, our competition may discover, develop, or commercialize products before or more successfully than we do, our estimates for market opportunities for our approved drug and drug candidates, market acceptance by physicians, patients, third-party payors, and others in the medical community of QINLOCK, and of any future approved drugs, such as vimseltinib or DCC-3116, if approved, our failure to obtain additional marketing approvals in other foreign jurisdictions, the potential for ongoing enforcement of post-marketing requirements for QINLOCK and any drug candidate for which we obtain marketing approval to subject us to substantial penalties, including withdrawal of QINLOCK or any future approved product from the market, if we fail to comply with all regulatory requirements, our assumptions in connection with the market opportunity for the INSIGHT trial patient population including the aggregate potential revenue opportunity for QINLOCK, our ability to complete, and the costs and timing of completing, the development and commercialization of our drug and drug candidates, our success in enrolling patients in clinical trials, including in our clinical trials of vimseltinib or DCC-3116, the potential for serious adverse events or unacceptable side effects to be identified during the development of our drug or drug candidates, our ability to obtain or, if granted, retain orphan drug exclusivity for our drug or drug candidates; our incurrence of significant operating losses since our inception and our expectation that we will incur continued losses for the foreseeable future and may never achieve or maintain profitability, our ability to raise capital when needed, our reliance on third parties to conduct our clinical trials and preclinical studies, our reliance on third parties for the manufacture of our drug candidates for preclinical testing and clinical trials, and for the manufacture of QINLOCK for commercialization and clinical trials, and our ability to enforce our intellectual property rights throughout the world. New risk factors and uncertainties may emerge from time to time, and it is not possible to predict all risk factors and uncertainties. There can be no assurance that the opportunity will meet your investment objectives, that you will receive a return of all or part of such investment. Investment results may vary significantly over any given time period. The appropriateness of a particular investment or strategy will depend on an investor's individual circumstances and objectives. Investors should independently evaluate specific investments. For further information regarding these risks, uncertainties and other factors, you should read the "Risk Factors" section of Deciphera's Annual Report on Form 10-Q for the quarter ended March 31, 2023 filed with the Securities and Exchange Commission (the "SEC"), and Deciphera's other SEC filings.

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ONE MISSION, INSPIRED BY PATIENTS: DEFEAT CANCER™

Executing on our mission to discover, develop, and commercialize important new medicines to **improve the lives of people with cancer.**

Over $1 Billion	**Peak Worldwide Sales** Potential for QINLOCK® (ripretinib) and Vimseltinib
Two Phase 3 Programs	**MOTION Top-line Data** and INSIGHT Initiation Planned for 2023
Potential First-in-Class Autophagy Program	**Multi-billion Dollar Opportunity** Targeting Autophagy
Proven Discovery Engine	**High-Value Research Pipeline** of Switch-Control Kinase Inhibitors

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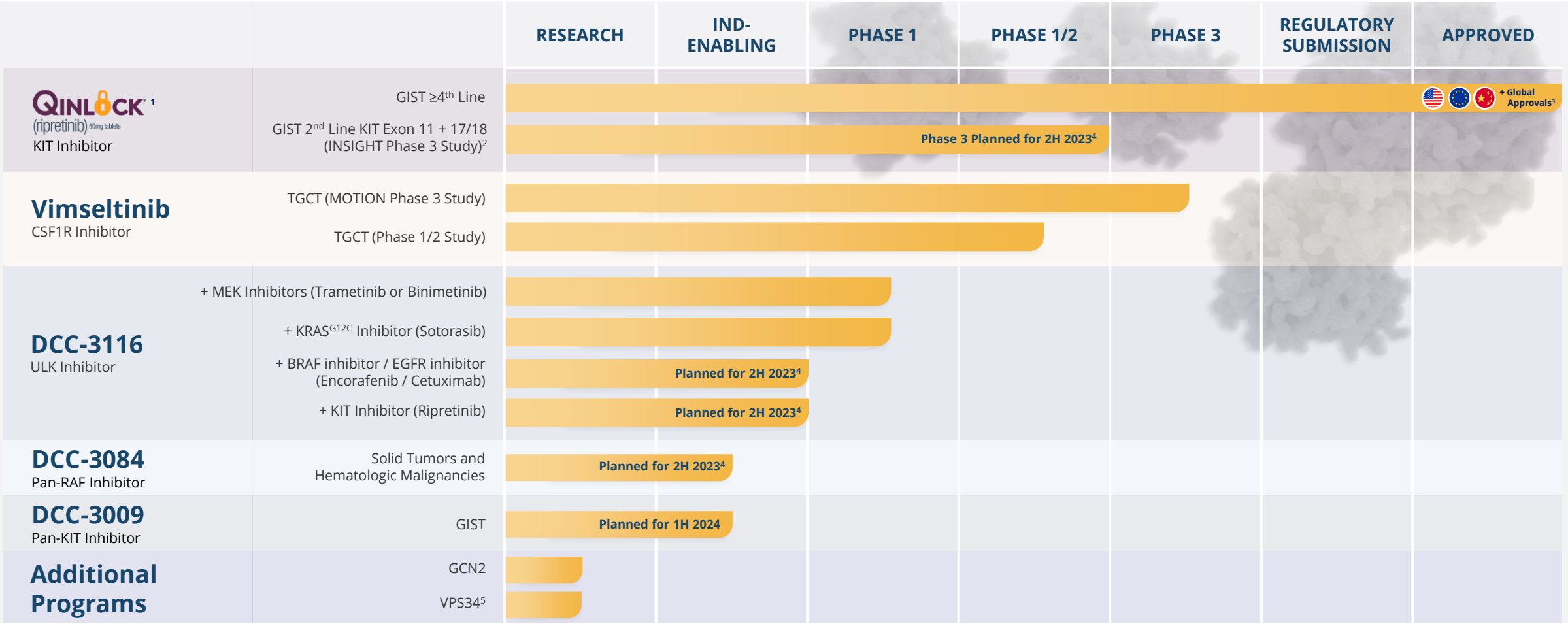
ROBUST PIPELINE OF SWITCH-CONTROL KINASE INHIBITORS



Notes: BRAF=proto-oncogene b-RAF; CSF1R=colony-stimulating factor 1 receptor; EGFR=epidermal growth factor receptor; GCN2=general control nonderepressible 2; GIST=gastrointestinal stromal tumor; KIT=KIT proto-oncogene receptor tyrosine kinase; MAPK=mitogen-activated protein kinase; RAF=rapidly accelerated fibrosarcoma; RAS=rat sarcoma gene; TGCT=tenosynovial giant cell tumor; ULK=unc-51-like autophagy-activating kinase; (1) Exclusive development and commercialization license with Zai Lab in Greater China for QINLOCK; (2) The patient population for the planned INSIGHT study consists of second-line GIST patients with mutations in KIT exon 11 and 17 and/or 18 and the absence of mutations in KIT exon 9, 13, and/or 14 (also referred to as KIT exon 11 + 17/18 patients); (3) QINLOCK is approved for 4th line GIST in the United States, Australia, Canada, China, the European Union, Hong Kong, Israel, Macau, New Zealand, Switzerland, Taiwan, and the United Kingdom; (4) 2023 Corporate Goal; (5) Agreement with Sprint Bioscience for exclusive in-license worldwide rights to a research-stage program targeting VPS34.

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STRATEGIC PRIORITIES FOR 2023

QINLOCK® (ripretinib)

- Initiate INSIGHT Phase 3 study in 2L KIT exon 11+17/18 GIST patients
- Drive continued 4L adoption in US and expansion in key European markets

Vimseltinib

- Announce top-line results from the MOTION Phase 3 study
- Present longer-term follow up from Phase 1/2 study

DCC-3116

- Initiate one or more MEK/G12C expansion cohorts in the Phase 1/2 study
- Initiate new combination study with encorafenib/cetuximab and with ripretinib

DCC-3084

- Submit IND to FDA

Proprietary Drug Discovery Platform

- Nominate development candidate for pan-KIT inhibitor (DCC-3009)

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Notes: 2L=second-line; 4L=fourth-line; FDA=U.S. Food and Drug Administration; G12C=single point mutation with a glycine-to-cysteine substitution at codon 12; GIST=gastrointestinal stromal tumor; IND=Investigational New Drug Application; KIT=KIT proto-oncogene receptor tyrosine kinase; MEK=mitogen-activated extracellular signal-regulated kinase.

QINLOCK® (ripretinib)

FIRST APPROVED TKI DESIGNED SPECIFICALLY FOR GIST

HIGHLY SUCCESSFUL U.S. LAUNCH	Clear standard-of-care in the U.S. for 4L setting across all mutational profiles
CONTINUED GEOGRAPHIC EXPANSION IN KEY EUROPEAN MARKETS	Strong momentum driven by launch in Germany and the post-approval paid-access program in France
NEW PIVOTAL PHASE 3 INSIGHT STUDY PLANNED	Study supported by compelling activity seen in ctDNA analysis in 2L GIST patients with mutations in KIT exon 11+17/18

Notes: Full prescribing information is available at www.QINLOCK.com; 2L=second-line; 4L=fourth-line; ctDNA=circulating tumor deoxyribonucleic acid; GIST=gastrointestinal stromal tumor; KIT=KIT proto-oncogene receptor tyrosine kinase; TKI=Tyrosine kinase inhibitor;

SIGNIFICANT UNMET MEDICAL NEED POST-IMATINIB REMAINS

Estimated U.S. Incidence of GIST: 4,000-6,000[1]

1L therapy
Imatinib[2]

18.9
months mPFS*

51.4%
ORR

49.0
months mOS*

*Results for imatinib 400 mg. This study compared imatinib 400 mg to imatinib 800 mg.[3]

2L therapy
Sunitinib[4,5]

5.6
months mPFS
HR=0.33

6.8%
ORR

17.0
months mOS
HR=0.87

~2,000
U.S. incident patients eligible for treatment[6]

3L therapy
Regorafenib[7]

4.8
months mPFS
HR=0.27

4.5%
ORR

17.4
months mOS
HR=0.91

~1,400–1,600
U.S. incident patients eligible for treatment[6]

4L therapy[8]
QINLOCK® (ripretinib) 50 mg tablets

6.3
months mPFS
HR=0.16[9]

11.8%
ORR[9]

18.2
months mOS
HR=0.41[9]

~1,000–1,300
U.S. incident patients eligible for treatment[6]

Avapritinib is approved in the U.S. for GIST patients with PDGFRA exon 18 mutations only, which are harbored by ~6% of patients with newly diagnosed GIST.[10,11]

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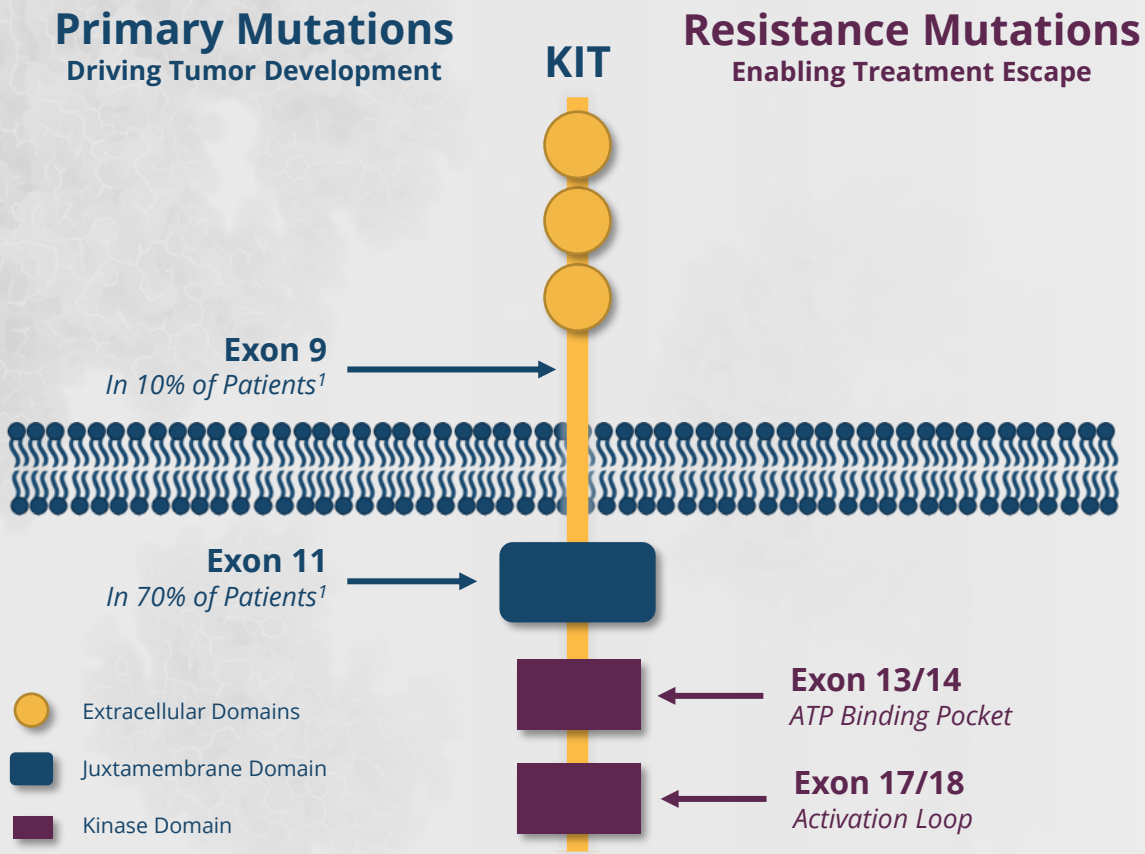
Notes: 1L=first-line; 2L=second-line; 3L=third-line; 4L=fourth-line; GIST=gastrointestinal stromal tumor; HR=hazard ratio; mOS=median overall survival; mPFS=median progression-free survival; ORR=objective response rate; PDGFRA=platelet-derived growth factor receptor α; (1) American Cancer Society, Key Statistics for Gastrointestinal Stromal Tumors, Accessed December 18, 2020; (2) Gleevec [package insert]. East Hanover, NJ: Novartis Pharmaceuticals Corp; 2020; (3) Gastrointestinal Stromal Tumor Meta-Analysis Group (MetaGIST). J Clin Oncol. 2010; 28:1247-1253; (4) Sutent [package insert]. New York, NY: Pfizer; 2020, mPFS and mOS converted from weeks to months; (5) Garrett CR, et al. Poster presented at: Connective Tissue Oncology Society: November 13-15, 2008; London, UK. Abstract 35049; (6) Internal Deciphera estimates of annual new treatment-eligible patients are based on analyses of U.S. claims data; eligible patients for 3rd and 4th lines exclude the estimated proportion of patients across lines that die, discontinue oncology treatment, or enter clinical trial and, therefore, are not eligible for treatment. Estimates are inherently uncertain; (7) Stivarga [package insert]. Germany: Bayer Healthcare; 2020; (8) QINLOCK [package insert]. Waltham, MA: Deciphera Pharmaceuticals; 2022; (9) Updated data on the INVICTUS Study presented at the ESMO Congress 2021; (10) Ayvakit [package insert]. Cambridge, MA: Blueprint Medicines Corp; 2021; (11) Lopes LF, Bacchi CE. J Cell Mol Med. 2010;14:42-50.

PROGRESSION DRIVEN BY SECONDARY RESISTANCE MUTATIONS IN KIT

KIT-DRIVEN MUTATIONS



- Early disease is driven by primary mutations in KIT exons 11 or 9
- Imatinib-resistant disease is driven by secondary mutations in KIT exons 17/18 and/or exons 13/14

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Notes: ATP=Adenosine Triphosphate; KIT=KIT proto-oncogene receptor tyrosine kinase; (1) Oppelt et al. J Gastrointest Oncol 2017;8(3):466-473.

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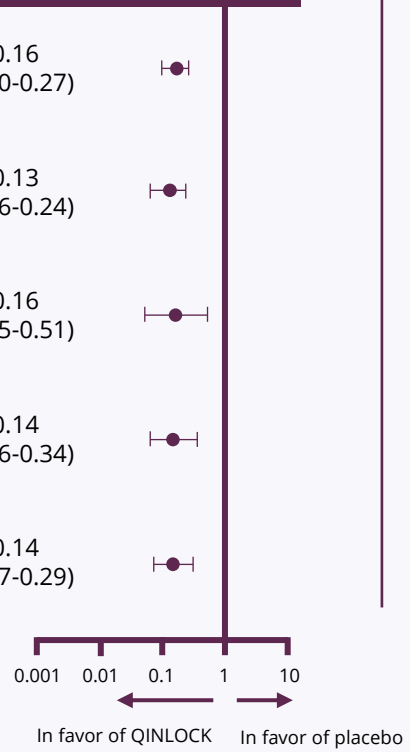
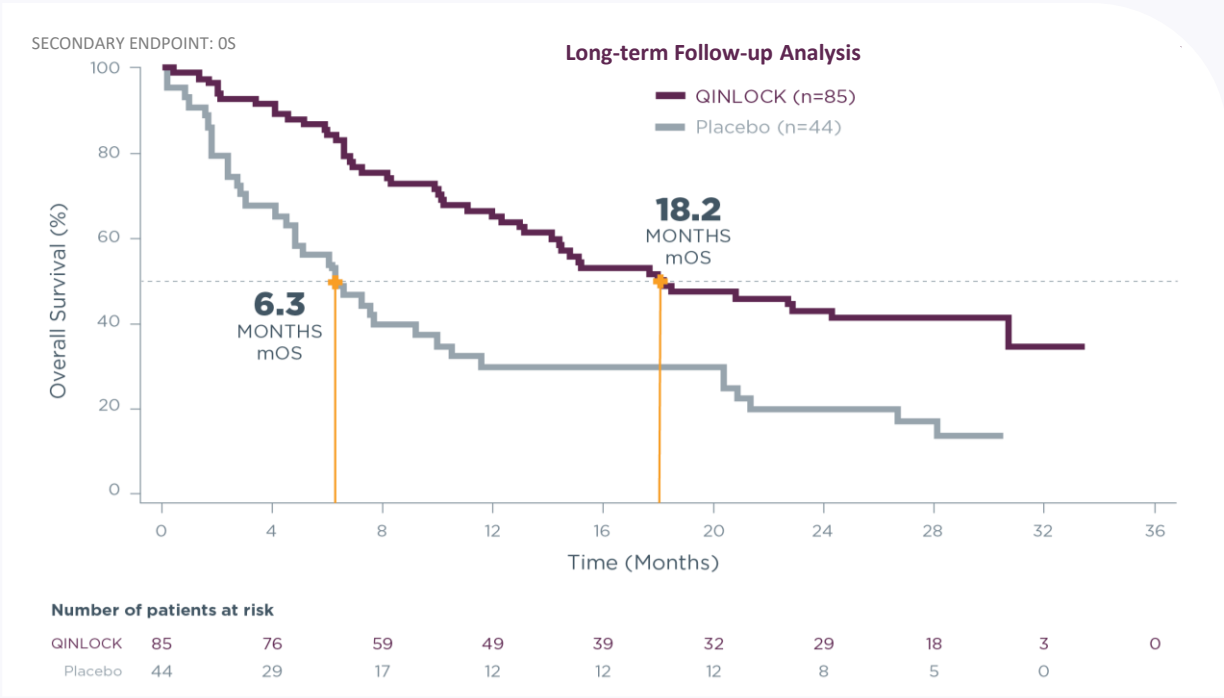
INVICTUS (4L+): QINLOCK SHOWED BENEFIT IN ALL MUTATION SUBGROUPS

Progression-Free Survival (INVICTUS 4L+)[3]

Mutation Subgroup	QINLOCK 150 mg QD (N)	Placebo (N)	Hazard Ratio (95% CI)
All Patients	85	44	0.16 (0.10-0.27)
Any KIT exon 11	52	34	0.13 (0.06-0.24)
Any KIT exon 9	16	7	0.16 (0.05-0.51)
Any KIT exon 13	27	16	0.14 (0.06-0.34)
Any KIT exon 17	44	27	0.14 (0.07-0.29)



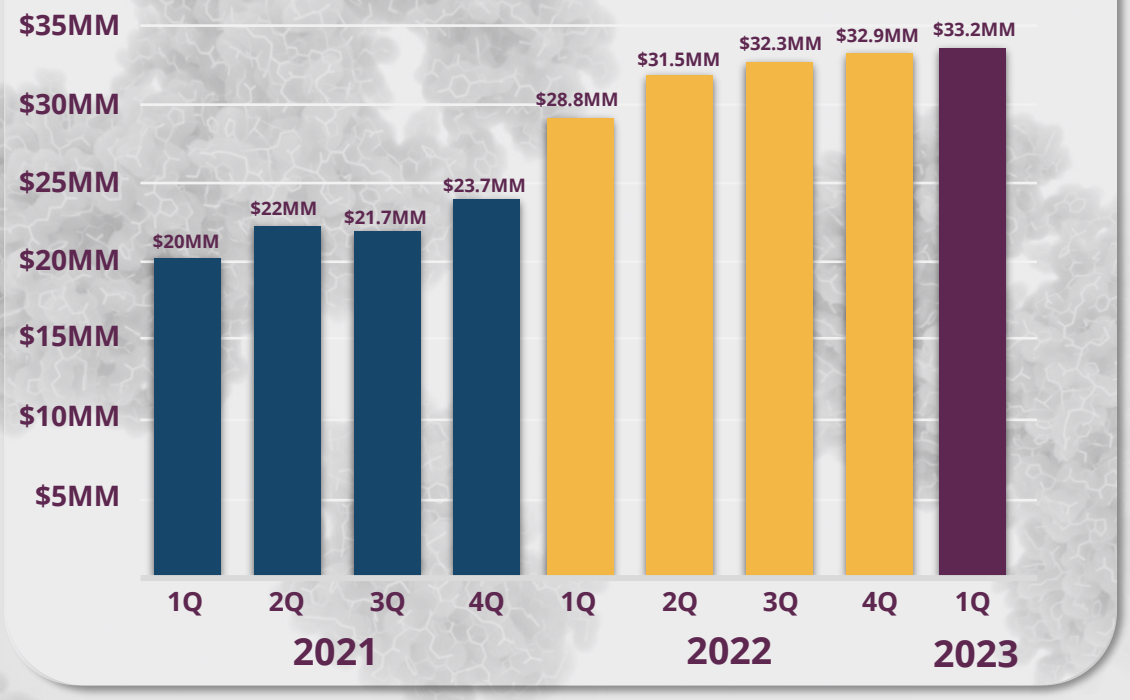
Kaplan-Meier Plots of Overall Survival (INVICTUS 4L+)[1,2]



Notes: 4L=fourth-line; CI=confidence interval; HR=hazard ratio; mOS=median overall survival; OS=overall survival; PFS=progression-free survival; QD=daily; (1) Updated data on the INVICTUS Study presented at the ESMO Congress 2021, the long-term follow-up analysis was conducted approximately 19 months from the data cutoff date in the primary analysis and was not powered to show statistical significance; (2) QINLOCK OS in the primary analysis was 15.1 months vs 6.6 months (HR = 0.36 [95% CI, 0.21 – 0.62], nominal p value 0.0004); (3) Bauer et al. Clin Cancer Res. 2021;27(23):6333–6342.

SUCCESSFUL LAUNCH OF QINLOCK AROUND THE WORLD

QINLOCK® Global Product Revenue



1Q 2023 Summary

- Total revenue of **$33.4MM** including:
  - QINLOCK product revenue: **$33.2MM**
    - U.S. net product sales of **$24.6MM**
    - International net product sales of **$8.6MM**
  - Collaboration revenue: **$0.2MM**

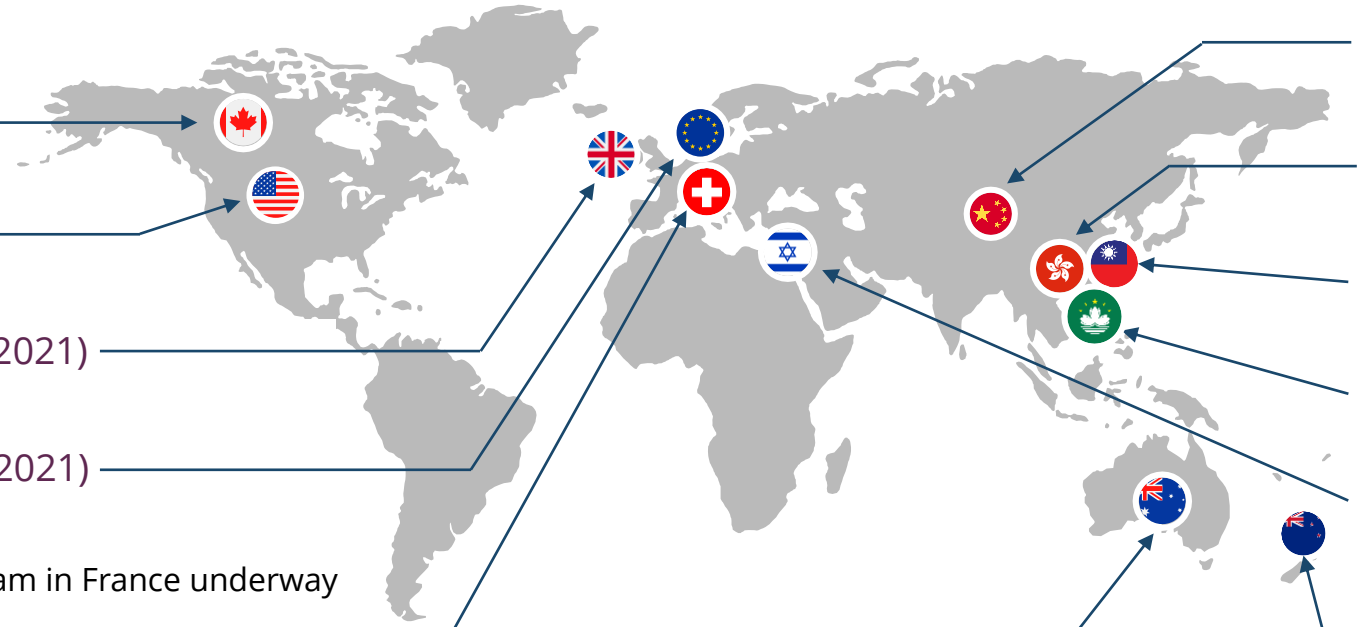
Potential Key 2023 Growth Drivers

- U.S. demand volume driven by expected gradual growth of average duration of therapy
- Continued geographic expansion in key European markets following pricing and reimbursement negotiations

Notes: Full prescribing information is available at www.QINLOCK.com.

GLOBAL APPROVALS AND EXPANSION

Significant progress expanding QINLOCK access to 4th line GIST patients globally



Canada (June 2020)

United States (May 2020)

United Kingdom (December 2021)

European Union (November 2021)

- Launch in Germany underway
- Post-approval paid access program in France underway

Switzerland (October 2021)

China (March 2021)[1]

Hong Kong (March 2021)[1]

Taiwan (September 2021)[1]

Macau (January 2023)[1]

Israel (January 2023)

Australia (July 2020)

New Zealand (December 2022)

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Notes: GIST=gastrointestinal stromal tumor; (1) Exclusive development and commercialization license with Zai Lab in Greater China for QINLOCK.

QINLOCK® | 4TH LINE GASTROINTESTINAL STROMAL TUMOR (GIST)

SUSTAINED MOMENTUM IN EUROPE DELIVERING A TOTAL OF **$8.6MM** IN 1Q 2023 INTERNATIONAL NET PRODUCT REVENUE

Strong Outcome from Germany Price Negotiations; Received "Major Additional Benefit" Rating

Planned Launch of QINLOCK in Italy in the Coming Months

Received Unanimous ASMR III Rating in France

Advancing Access Discussions with Other Health Authorities Across Europe

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Notes: ASMR=amélioration du service médical rendu; GIST=gastrointestinal stromal tumor.

INTRIGUE STUDY TESTED SUPERIORITY IN 2L GIST POPULATION[1]

INCLUSION CRITERIA

Patients ≥18 years old with a confirmed diagnosis of GIST who progressed on or had documented intolerance to imatinib

Patients were enrolled from 122 sites across North America, South America, Europe, Australia, and Asia

Stratified by

- Mutational status:
  - *KIT* exon 11
  - *KIT* exon 9
  - *KIT/PDGFRA* Wild Type
  - Other *KIT/PDGFRA*
- Intolerance to imatinib

INTRIGUE PHASE 3 CLINICAL STUDY

1:1 Randomization
Open label

QINLOCK 150 mg QD
(continuous)

No crossover option

Sunitinib 50 mg QD
(4 weeks on, 2 weeks off)

Primary endpoint

- PFS by IRR (using mRECIST v 1.1) in the KIT exon 11 ITT and AP ITT populations

Planned exploratory analysis

- Subgroup analysis for efficacy based on ctDNA mutation status

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Notes: 2L=second-line; AP=all-patient; ctDNA=circulating tumor deoxyribonucleic acid; GIST=gastrointestinal stromal tumor; IRR=individual research result; ITT=intention-to-treat; KIT=KIT proto-oncogene receptor tyrosine kinase; mRECIST=modified Response Evaluation Criteria in Solid Tumors; PDGFRA=platelet-derived growth factor receptor alpha; PFS=progression-free survival; QD=once daily; (1) Bauer et al. *J Clin Onc.* 2022. 40:3918-3928.

INTRIGUE STUDY TUMOR TISSUE BIOPSY ANALYSIS BY PRIMARY MUTATION[1]

	QINLOCK n (events)	Sunitinib n (events)	mPFS QINLOCK (months)	mPFS Sunitinib (months)	Hazard Ratio (95% CI)
Overall	**226 (146)**	**227 (130)**	**8.0**	**8.3**	**1.05 (0.82, 1.33)**
MUTATION TYPE					
KIT exon 11	163 (100)	164 (98)	8.3	7.0	0.88 (0.67, 1.17)
KIT exon 9	31 (27)	29 (14)	5.5	13.8	2.85 (1.48, 5.48)
KIT / PDGFRA Wild Type	15 (9)	18 (10)	7.0	4.1	0.90 (0.36, 2.23)
Other KIT / PDGFRA	17 (10)	16 (8)	6.8	8.4	0.90 (0.35, 2.28)

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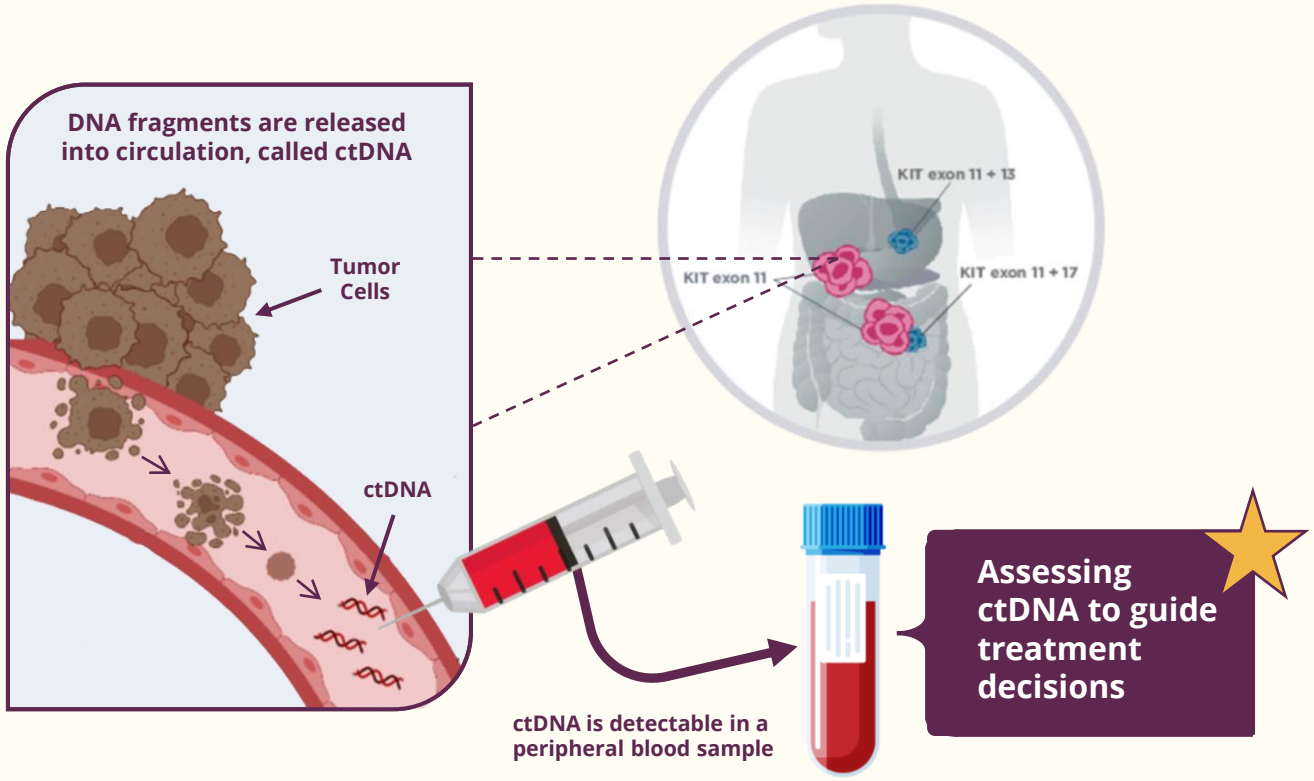
Notes: 2L=second-line; CI=confidence interval; KIT=KIT proto-oncogene receptor tyrosine kinase; mPFS=median progression-free survival; PDGFRA=platelet-derived growth factor receptor alpha; (1) Bauer et al. *J Clin Onc*. 2022. 40:3918-3928.

PRACTICE CHANGING POTENTIAL WITH ctDNA IN GIST



- ctDNA is rapidly being adopted in oncology to identify patients harboring sensitive mutations and to treat patients with precision medicines
- Tissue biopsies may not capture the full clonal heterogeneity of GIST
- ctDNA was collected at baseline in the INTRIGUE study as an exploratory analysis

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Notes: ctDNA=circulating tumor deoxyribonucleic acid; DNA=deoxyribonucleic acid; GIST=gastrointestinal stromal tumor; KIT=KIT proto-oncogene receptor tyrosine kinase.

DETECTION OF BASELINE KIT/PDGFRA MUTATIONS

Total Subjects
N=453

- Advanced GIST
- 2L after Imatinib

ctDNA Analyzed[1] — **n=362**

ctDNA Detected — **n=280**

Any KIT Mutation Detected → n=213

KIT Mutations Detected	
KIT Mutation Detected	**213 / 362 (59%)**
Any Exon 11	157 / 362 (43%)
Any Exon 9	36 / 362 (10%)
Any Exon 17/18 (Activation Loop)	89 / 362 (25%)
Any Exon 13/14 (ATP Binding Pocket)	81 / 362 (22%)

KIT Exon 11 Primary Mutation + Secondary Mutations	
Exon 11+17/18 **Only** (Activation Loop)	52 / 362 (14%)
Exon 11+13/14 **Only** (ATP Binding Pocket)	41 / 362 (11%)
Exon 11+13/14 **And** 17/18	22 / 362 (6%)

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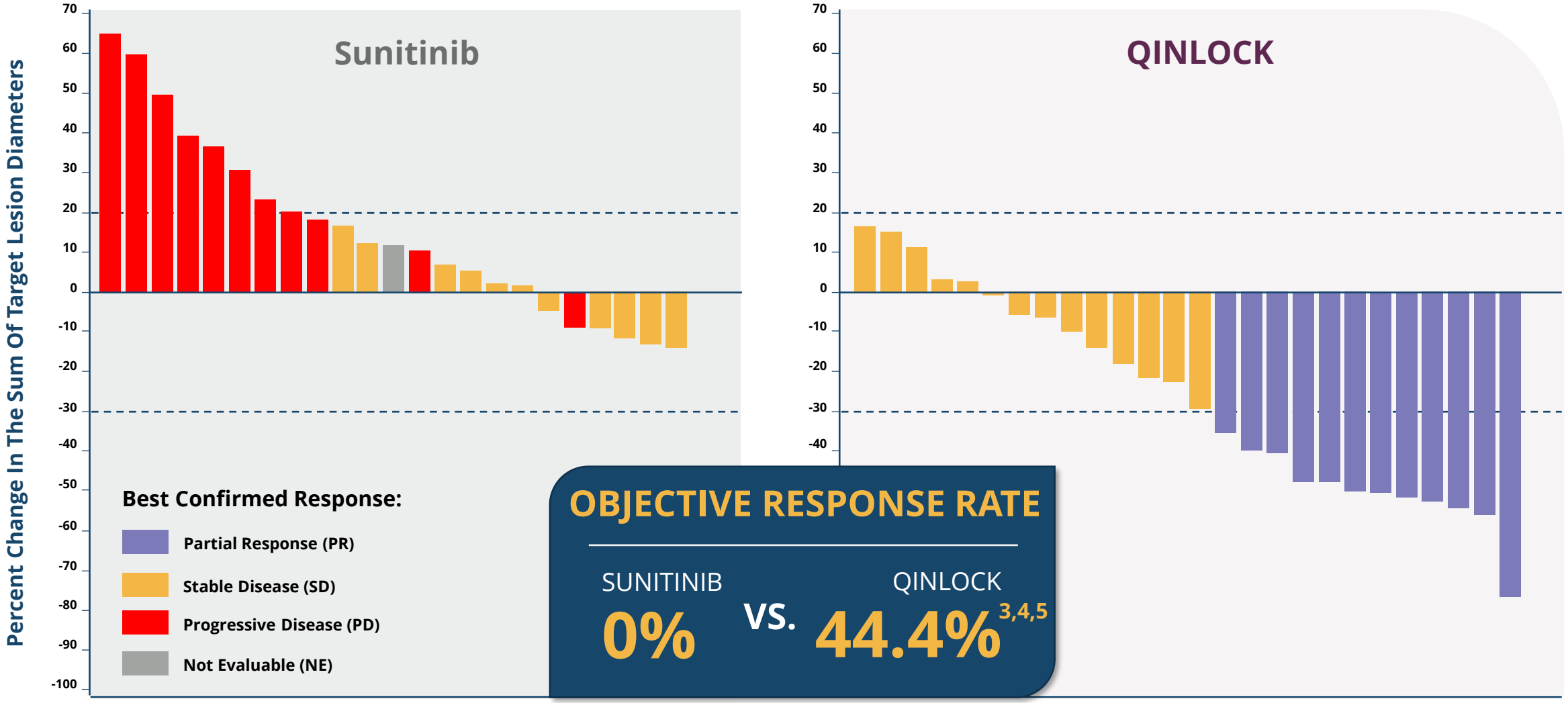
Notes: Data presented at the ASCO Plenary Series Session 2023; Full prescribing information is available at www.QINLOCK.com; Deciphera Data on File; 2L=second-line; ATP=Adenosine Triphosphate; ctDNA=circulating tumor deoxyribonucleic acid; GIST=gastrointestinal stromal tumor; KIT=KIT proto-oncogene receptor tyrosine kinase; (1) of the 453 patients in the overall intent-to-treat population, baseline ctDNA was analyzed in 362 patients for whom evaluable samples were available.

IMPRESSIVE ORR FOR QINLOCK IN KIT EXON 11+17/18 PATIENTS[1,2]



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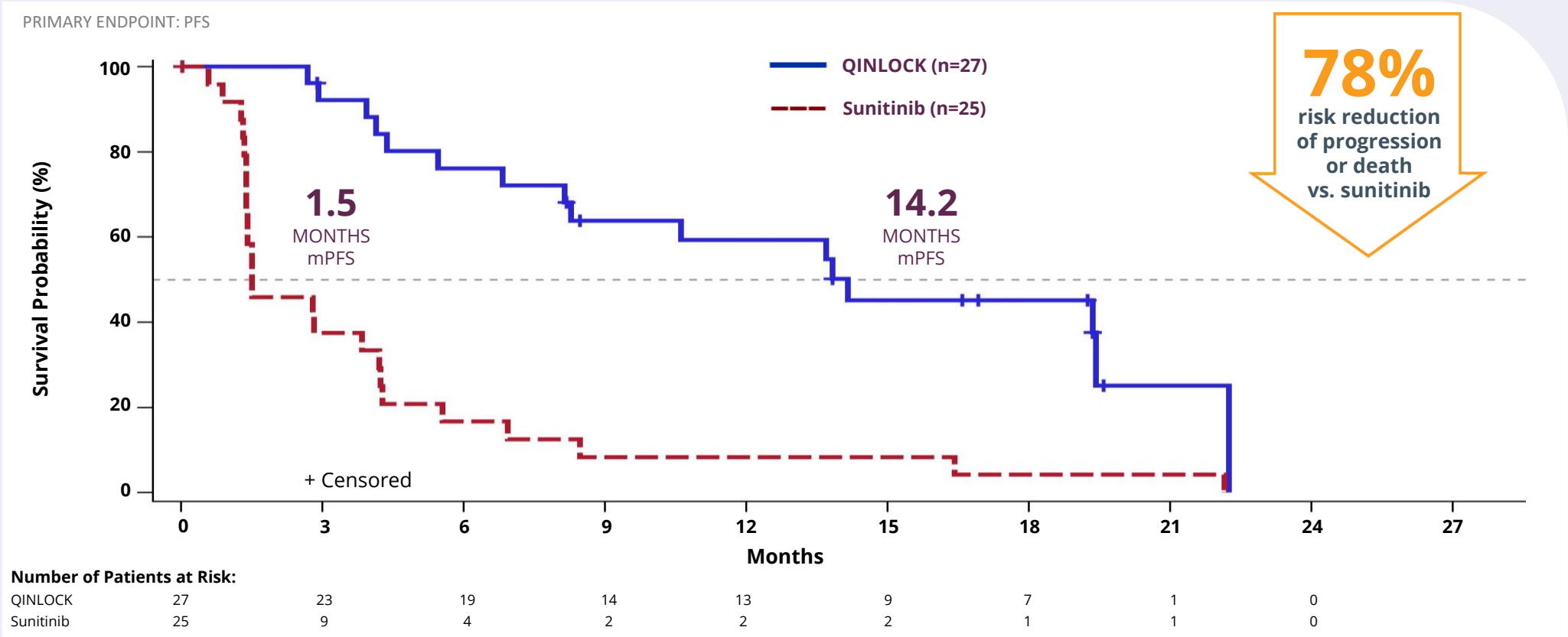
Notes: Data presented at the ASCO Plenary Series Session 2023; Full prescribing information is available at www.QINLOCK.com ; Deciphera Data on File; 2L=second-line; (1) data cut off date of September 1, 2021; (2) for 2 patients in the sunitinib arm and 1 patient in the ripretinib arm, no postbaseline disease assessment was available; (3) ORR was confirmed with follow-up imaging; (4) determined using modified Response Evaluation Criteria in Solid Tumors (RECIST) 1.1 criteria; (5) response difference=44.4%, 95% CI (23.0, 62.7), nominal p value 0.0001.

PFS STRONGLY FAVORS QINLOCK IN KIT EXON 11+17/18 PATIENTS[1]

Progression-Free Survival

KIT exon 11+17/18



(median PFS of 14.2 months vs. 1.5 months; HR=0.22, 95% CI [0.11-0.44], nominal p value <0.0001)

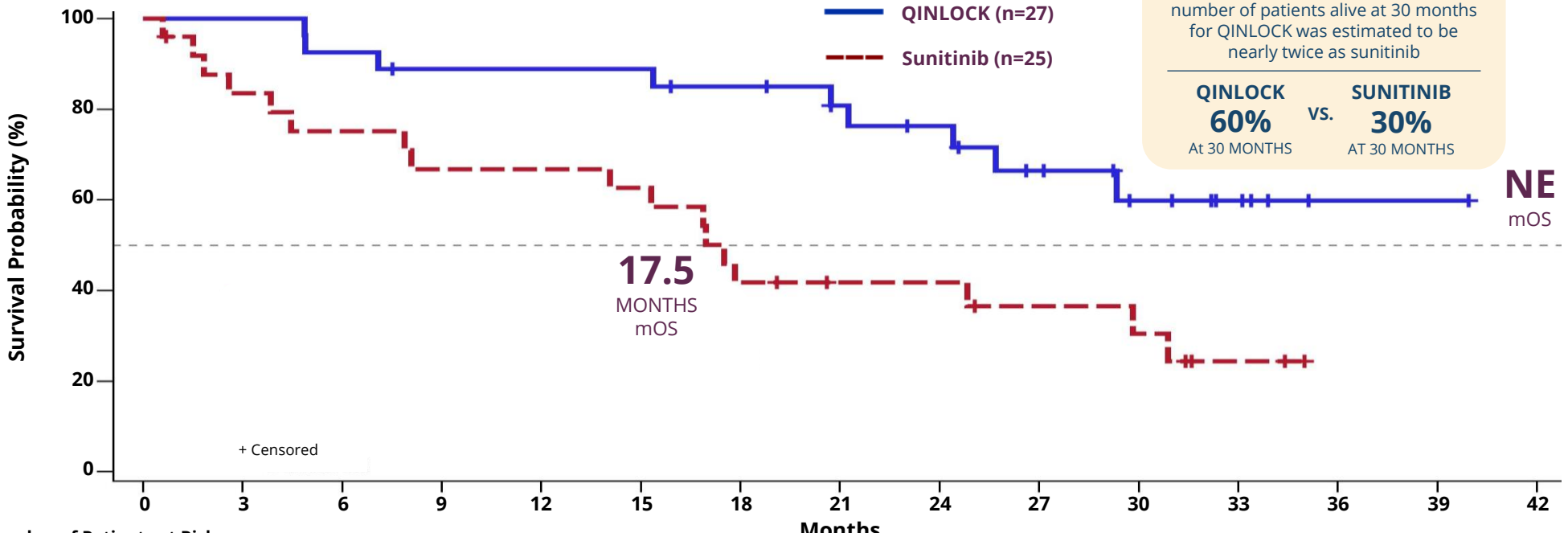
Notes: Data presented at the ASCO Plenary Series Session 2023; Full prescribing information is available at www.QINLOCK.com ; Deciphera Data on File; 2L=second-line; HR=hazard ratio; KIT=KIT proto-oncogene receptor tyrosine kinase; mPFS=median progression-free survival; PFS=progression-free survival; (1) data cut off date of September 1, 2021.

SUBSTANTIAL OS FOR QINLOCK IN KIT EXON 11+17/18 PATIENTS[1]

Overall Survival Analysis

KIT exon 11+17/18

SECONDARY ENDPOINT: OS



(median OS of NE months vs. 17.5 months; HR=0.34, 95% CI [0.15-0.76], nominal p value 0.0061)

Notes: Data presented at the ASCO Plenary Series Session 2023; Full prescribing information is available at www.QINLOCK.com; Deciphera Data on File; 2L=second-line; HR=hazard ratio; KIT=KIT proto-oncogene receptor tyrosine kinase; NE=not estimable; OS=overall survival; (1) the data cut off date for the second interim analysis for overall survival was September 1, 2022.

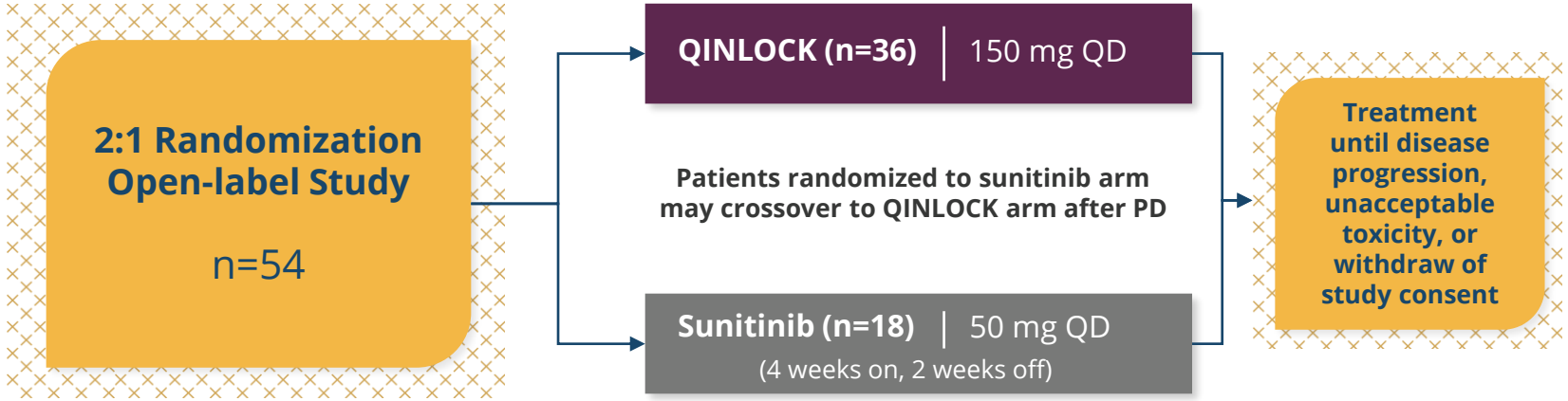
INSIGHT PIVOTAL PHASE 3 STUDY EXPECTED TO INITIATE IN 2H 2023

INCLUSION CRITERIA

Patients with GIST previously treated with imatinib

- 1 prior line of imatinib
- KIT exon 11 + (17 and/or 18) via ctDNA at prescreening
  - KIT exon 9, 13, and/or 14 are excluded
  - Other co-mutations are allowed
- Measurable disease per mRECIST
- ECOG performance status ≤ 2

PLANNED PHASE 3, RANDOMIZED, MULTICENTER, OPEN-LABEL STUDY



Primary Endpoint
- PFS by IRR using mRECIST

Key Secondary Endpoints
- ORR by IRR using mRECIST
- OS

Notes: 2L=second-line; ctDNA=circulating tumor deoxyribonucleic acid; ECOG=Eastern Cooperative Oncology Group; GIST=gastrointestinal stromal tumor; IRR= individual research result; KIT=KIT proto-oncogene receptor tyrosine kinase; mRECIST=modified Response Evaluation Criteria in Solid Tumors; ORR=objective response rate; OS=overall survival; PD=progressive disease ; PFS=progression-free survival; QD=daily.

KEY SUCCESS FACTORS FOR INSIGHT PIVOTAL PHASE 3 STUDY

Strong Scientific Rationale and Compelling Efficacy Results for QINLOCK from ctDNA analysis

- Validates preclinical evidence and KOL's expectations of differential activity of each drug
- Dramatic and consistent clinical benefit of QINLOCK shown across all efficacy endpoints
- Conviction about results reinforced by PFS hazard ratio, confidence intervals, and nominal p-value

Consistency between INTRIGUE and INSIGHT Trial Designs

- Assumptions based on contemporary data from INTRIGUE
- INSIGHT patient population is the same size as the INTRIGUE ctDNA subgroup
- Dosing regimens, outcome measures and other material features are identical in INSIGHT and INTRIGUE

Confidence in Study Execution

- Significant investigator interest in using precision medicine to improve outcomes in 2L GIST
- Patient focused design with 2:1 randomization to QINLOCK and crossover to QINLOCK
- Design based on feedback from KOLs and FDA
- Deciphera is the most experienced company at running global GIST trials (over 750+ GIST patients)

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Notes: 2L=second-line; ctDNA=circulating tumor deoxyribonucleic acid; FDA=U.S. Food and Drug Administration; GIST=gastrointestinal stromal tumor; HR=hazard ratio; KIT=KIT proto-oncogene receptor tyrosine kinase; KOL=key opinion leader; NE=not estimable; ORR=objective response rate; mPFS=median progression-free survival; mOS=median overall survival; PFS=progression-free survival.

OPPORTUNITY TO ADVANCE THE STANDARD OF CARE ACROSS MULTIPLE LINES OF THERAPY

4L

Established 4L Standard-of-Care Across All Mutations

QINLOCK (ripretinib) 50 mg tablets

6.3 mo. mPFS (HR=0.16)[1]
11.8% ORR
mOS 18.2 mo. (HR=0.41)

~90% of prescribers cite QINLOCK as the 4L standard-of-care across all mutations[2]

- ✓ KIT exon 11
- ✓ KIT exon 9
- ✓ PDGFRA
- ✓ Wild Type
- ✓ Unknown mutational status

+

2L[3]

Potential to Optimize 2L Therapy Based on Secondary Mutations

QINLOCK (ripretinib) 50 mg tablets

For exon 11+17/18 patients

ctDNA Analysis

14.2 mo. mPFS (HR=0.22)[4]
44.4% ORR
mOS not estimable (HR=0.34)

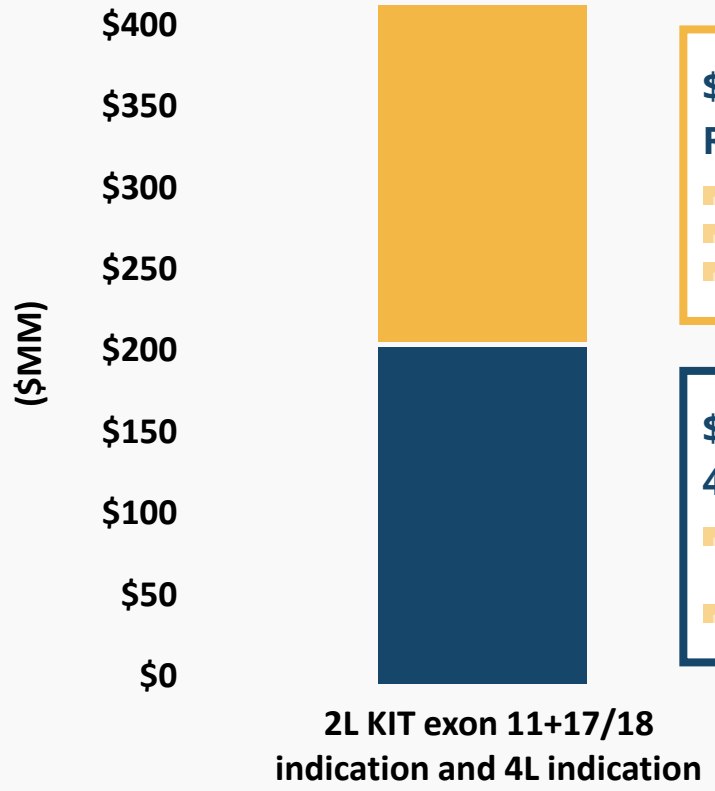
Sunitinib

deciphera

Notes: 2L=second-line; 4L=fourth-line; HR=hazard ratio; mOS=median overall survival; mPFS=median progression-free survival; (1) Updated data on the INVICTUS Study presented at the ESMO Congress 2021; Full prescribing information is available at www.QINLOCK.com; Overall survival data includes all time periods, including dose escalation. Placebo arm includes patients taking placebo who, following progression, were crossed-over to QINLOCK treatment; The long-term follow-up analysis was conducted approximately 19 months from the data cutoff date in the primary analysis and was not powered to show statistical significance; (2) Deciphera ATU survey, 1Q 2022; approximate % of GIST Treaters agreeing to strongly agreeing that QINLOCK is the standard of care for 4L+ advGIST patients; % of GIST treaters stating their likelihood to prescribe QINLOCK across GIST mutational types.; (3) Use of QINLOCK in the 2nd line setting for patients with GIST with exon 11 + 17/18 mutations is subject to positive INSIGHT phase 3 study and approval by regulatory authorities; (4) Deciphera Data on File. Data cut off date of September 1, 2021.

A 2L KIT EXON 11+17/18 INDICATION ESTIMATED TO DOUBLE QINLOCK U.S. PEAK REVENUE POTENTIAL[1]

QINLOCK Estimated U.S. Peak Revenue ($MM)[1]



$175-$200MM *Incremental* Peak Revenue for 2L Indication

- New QINLOCK-treated patients in 2L
- More months of therapy per patient
- Net price growth

$175-$200MM Peak Revenue for 4L Indication

- Modest volume growth as average DOT matures
- Net price growth

$350-$400MM Total U.S. Peak Revenue[1]

Notes: Full prescribing information is available at www.QINLOCK.com; 2L=Second-Line; 4L=Fourth-Line; DOT=Duration of Therapy; (1) These estimates are based on management's current expectations and assumptions, including, without limitation, estimates of net price growth over time, adoption of ctDNA testing for GIST patients, estimates of on-label adoption, the potential for unpromoted off-label usage and reasonable expectations of the potential impact of the Inflation Reduction Act (the full impact of which is still under review) and an assumption of a second-line approval in GIST patients with mutations in KIT exon 11 + 17/18; estimates are inherently uncertain and are subject to a wide variety of assumptions, risks and uncertainties that can cause actual results to differ materially.

QINLOCK® | EXPLORATORY ANALYSIS OF INTRIGUE STUDY IN 2L GIST

COMPELLING EFFICACY RESULTS IN KIT EXON 11+17/18 PATIENTS

OBJECTIVE RESPONSE RATE[1]

QINLOCK		SUNITINIB
44.4%[2]	vs.	0%

MEDIAN PROGRESSION-FREE SURVIVAL[1,3]

QINLOCK		SUNITINIB
14.2 MONTHS	vs.	1.5 MONTHS

MEDIAN OVERALL SURVIVAL[4]

QINLOCK		SUNITINIB
Not Estimable	vs.	17.5 MONTHS

INSIGHT PIVOTAL PHASE 3 STUDY
EXPECTED TO INITIATE IN 2H 2023

QINLOCK PEAK U.S. REVENUE POTENTIAL ESTIMATED TO DOUBLE WITH 2L KIT EXON 11+17/18 INDICATION[5]

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Notes: Data presented at the ASCO Plenary Series Session 2023; Full prescribing information is available at www.QINLOCK.com ; Deciphera Data on File; 2L=second-line; KIT=KIT proto-oncogene receptor tyrosine kinase; ORR=objective response rate; OS=overall survival; PFS=progression-free survival; (1) data cut off date of September 1, 2021; (2) ORR was confirmed with follow-up imaging; (3) determined using modified Response Evaluation Criteria in Solid Tumors (RECIST) 1.1 criteria; (4) the data cut off date for the second interim analysis for overall survival was September 1, 2022; (5) These estimates are based on management's current expectations and assumptions, including, without limitation, estimates of net price growth over time, adoption of ctDNA testing for GIST patients, estimates of on-label adoption, the potential for unpromoted off-label usage and reasonable expectations of the potential impact of the Inflation Reduction Act (the full impact of which is still under review) and an assumption of a second-line approval in GIST patients with mutations in KIT exon 11 + 17/18 ; estimates are inherently uncertain and are subject to a wide variety of assumptions, risks and uncertainties that can cause actual results to differ materially.

VIMSELTINIB

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TOP-LINE RESULTS FROM MOTION PHASE 3 STUDY EXPECTED IN 4Q 2023

- **Vimseltinib** is an oral, switch-control TKI specifically designed to selectively and potently inhibit CSF1R
- Positive Phase 1/2 data in TCGT strongly supports ongoing MOTION Phase 3 study[1]
- +$850MM TGCT market in U.S. with 90% of prescribers already targeted with GIST franchise[2]

Expected 2023 Milestones[3]

Completed (1Q 2023)	Complete enrollment in the MOTION Phase 3 study
4Q 2023	Announce top-line results from MOTION Phase 3 study
2H 2023	Present updated Phase 1/2 data in TGCT patients

Notes: CSF1R=colony-stimulating factor 1 receptor; GIST=gastrointestinal stromal tumor; KOL=key opinion leader; TGCT=tenosynovial giant cell tumor; TKI=tyrosine kinase inhibitor; (1) Data presented at the ESMO Congress 2022; (2) Based on HCP list match between Symphony Health IDV Claims; Analysis Period: 7/1/2020 – 6/1//2021 and internal Deciphera Targeted GIST HCP prescribers; (3) 2023 Corporate Goal.

AN INTERNATIONAL, MULTICENTER, OPEN-LABEL PHASE 1/2 STUDY

PHASE 1 (DOSE ESCALATION)

- A pharmacologically guided 3 + 3 design, to determine the recommended Phase 2 dose (RP2D) and the maximum tolerated dose
- Enrollment in Phase 1 dose escalation is complete

COHORT 5 (n=8)

Loading Dose	Dose
30 mg QD x 5 days	30 mg twice weekly

COHORT 8 (n=12)

Loading Dose	Dose
30 mg QD x 3 days	10 mg QD

COHORT 9 (n=12)

Loading Dose	Dose
20 mg QD x 3 days	6 mg QD

PHASE 2 (EXPANSION)

- Study to evaluate the safety, tolerability, and preliminary efficacy in two TGCT expansion cohorts
- Recommended Phase 2 dose of 30 mg twice weekly with no loading dose

RP2D
30 mg twice weekly

COHORT A (n=46)

TGCT patients with no prior anti-CSF1/CSF1R therapy (previous therapy with imatinib or nilotinib is allowed)

PHASE 2 (n=58)

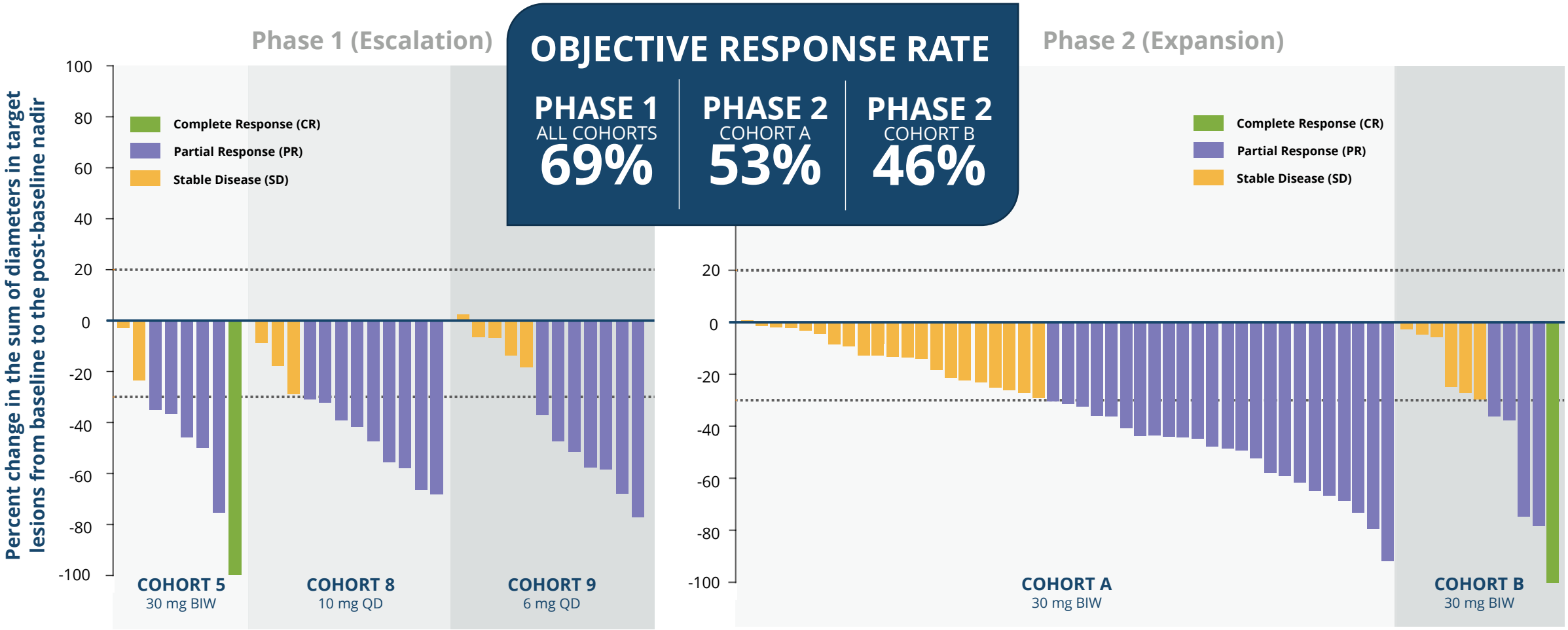
COHORT B (n=12)

TGCT patients with prior anti-CSF1/CSF1R therapy (previous therapy with imatinib or nilotinib alone would not be eligible)

Enrollment Ongoing in Cohort B

Notes: Data presented at the ESMO Congress 2022; CSF1=colony-stimulating factor 1; CSF1R=colony-stimulating factor 1 receptor; QD=once daily; RP2D=recommended Phase 2 dose; TGCT=tenosynovial giant cell tumor.

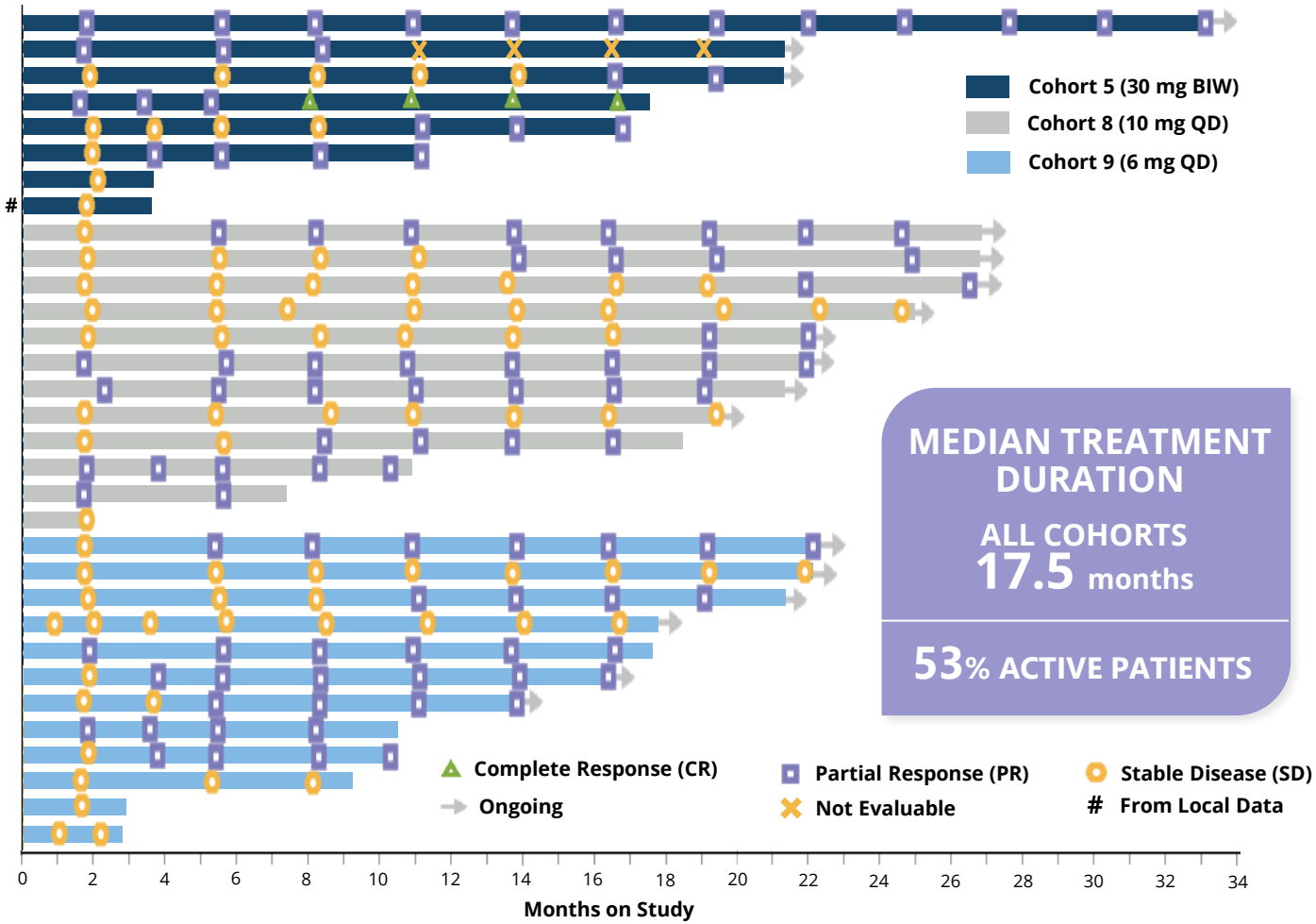
ENCOURAGING ANTI-TUMOR ACTIVITY REGARDLESS OF PRIOR CSF1R THERAPY



Notes: Data presented at the ESMO Congress 2022; results are reported for patients with TGCT with a data cutoff of May 6, 2022; ORR=objective response rate.

INCREASING DURATION OF THERAPY AND DURABLE RESPONSES

Phase 1 (Escalation)

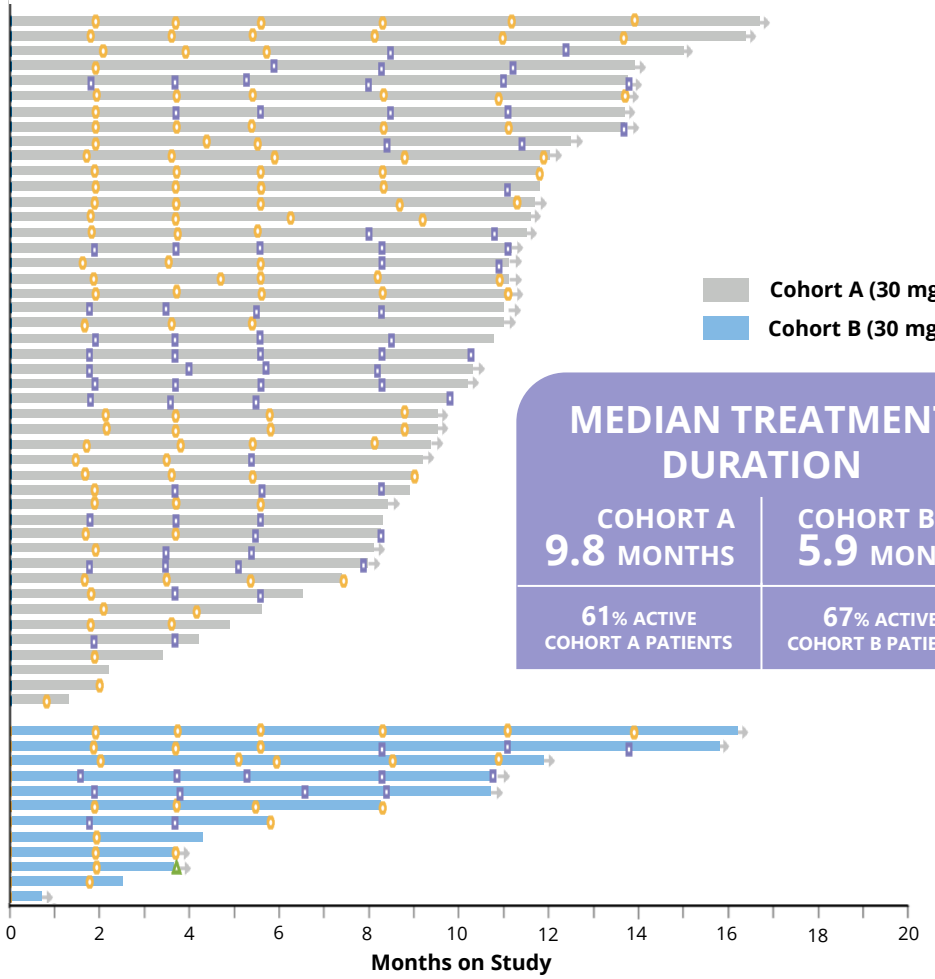


MEDIAN TREATMENT DURATION

ALL COHORTS
17.5 months

53% ACTIVE PATIENTS

Phase 2 (Expansion)



MEDIAN TREATMENT DURATION

COHORT A	COHORT B
9.8 MONTHS	5.9 MONTHS
61% ACTIVE COHORT A PATIENTS	67% ACTIVE COHORT B PATIENTS

Notes: Data presented at the ESMO Congress 2022; results are reported for patients with TGCT with a data cutoff of May 6, 2022.

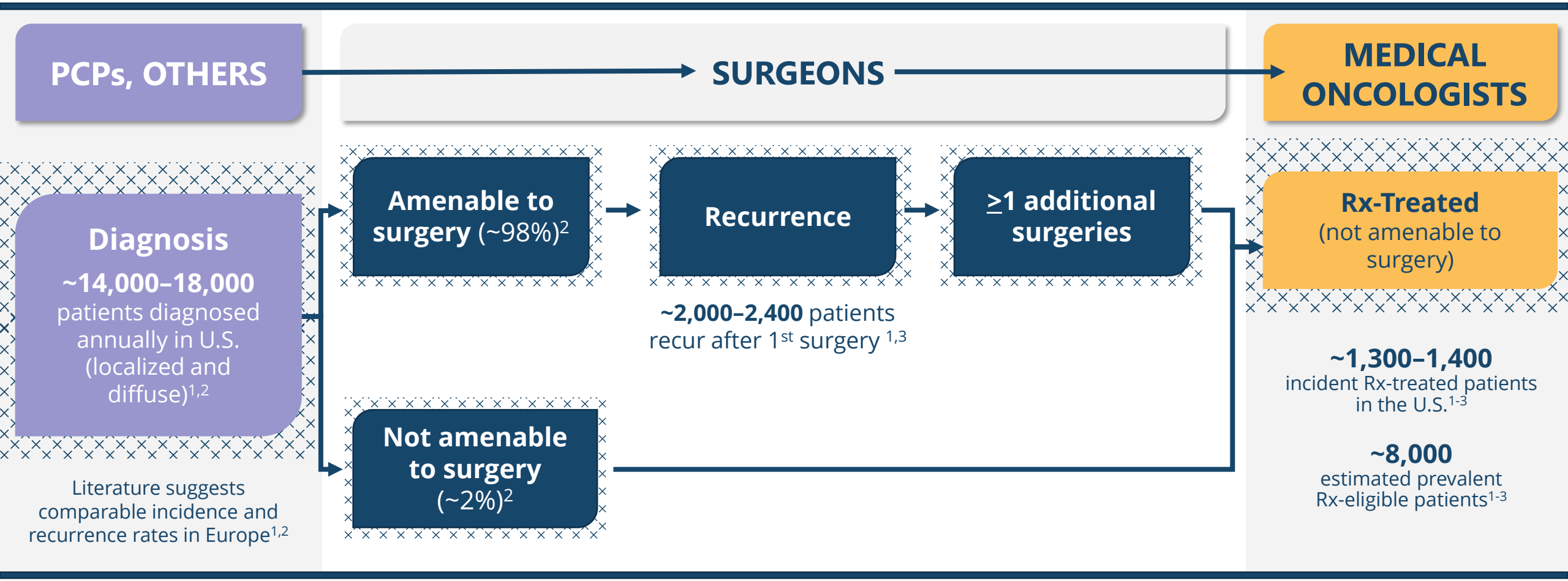
WELL-TOLERATED IN TGCT PATIENTS

TEAEs in ≥15% of Patients Receiving Vimseltinib

Preferred term	Phase 1 All Patients[1] (n = 32)		Phase 2 All Patients[1] (n = 58)		Phase 1/2 Combined All Patients (n = 90)	
	All grades	Grade 3/4	All grades	Grade 3/4	All grades	Grade 3/4
Blood CPK increased	20 (63%)	10 (31%)	34 (59%)	22 (38%)	54 (60%)	32 (36%)
Periorbital edema	17 (53%)	0	22 (38%)	0	39 (43%)	0
Fatigue	16 (50%)	0	16 (28%)	0	32 (36%)	0
AST increased	11 (34%)	4 (13%)	10 (17%)	0	21 (17%)	4 (4%)
Arthralgia	10 (31%)	1 (3%)	13 (22%)	1 (2%)	23 (26%)	2 (2%)
Face oedema	9 (28%)	0	10 (17%)	0	19 (21%)	0
Myalgia	9 (28%)	1 (3%)	15 (26%)	0	24 (27%)	1 (1%)
Oedema peripheral	9 (28%)	0	9 (16%)	0	18 (20%)	0
Pruritus	9 (28%)	0	0	0	9 (10%)	0
Headache	8 (25%)	0	27 (47%)	0	35 (39%)	0
ALT increased	7 (22%)	1 (3%)	0	0	7 (8%)	1 (1%)
Diarrhea	7 (22%)	1 (3%)	9 (16%)	0	16 (18%)	1 (1%)
Lipase increased	7 (22%)	3 (9%)	0	0	7 (7%)	3 (3%)
Generalized oedema	7 (22%)	0	0	0	7 (7%)	0
Hypertension	6 (19%)	2 (6%)	0	0	6 (7%)	2 (2%)
Nausea	6 (19%)	0	19 (33%)	0	25 (28%)	0
Rash	6 (19%)	0	0	0	6 (7%)	0
Amylase increased	5 (16%)	2 (6%)	0	0	5 (6%)	2 (2%)
Constipation	5 (16%)	0	0	0	5 (6%)	0
Dry skin	5 (16%)	0	0	0	5 (6%)	0
Paresthesia	5 (16%)	0	0	0	5 (6%)	0
Rash maculopapular	5 (16%)	0	13 (22%)	1 (2%)	18 (20%)	1 (1%)
Asthenia	0	0	15 (26%)	1 (2%)	15 (17%)	1 (1%)

Notes: Data presented at the ESMO Congress 2022; results are reported for patients with TGCT with a data cutoff of May 6, 2022; Data are presented as n (%) unless otherwise noted; percentages are rounded; ALT=alanine aminotransferase; AST=aspartate aminotransferase; CPK=creatine phosphokinase; TEAE=treatment-emergent adverse event; (1) TEAE cutoff of ≥15% based on all grades for total Phase 1 and Phase 2.

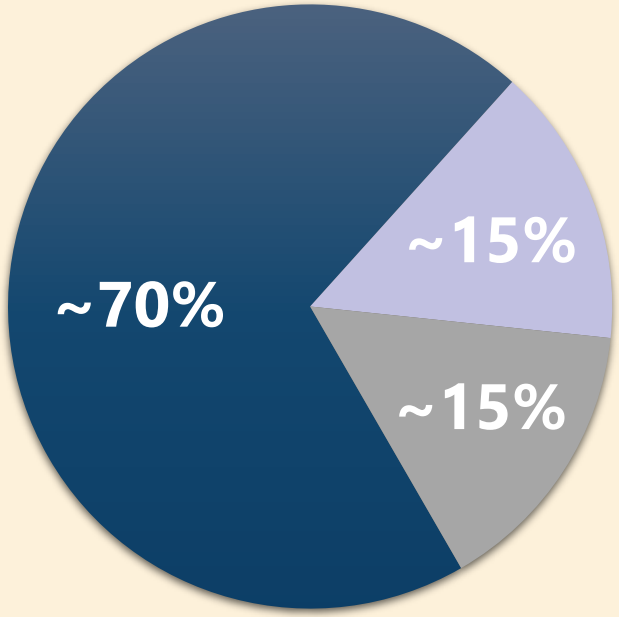
PATIENT JOURNEY OF TGCT PATIENTS NOT AMENABLE TO SURGERY



Notes: PCP=primary care physician; TGCT=tenosynovial giant cell tumor; (1) Mastboom et al. Acta Orthopaedica. 2017;88(6):688-694; (2) Internal Deciphera estimates based on market research and secondary data; estimates are inherently uncertain; (3) Ehrenstein. J Rheumatol. 2017;44(10):1476-1483.

TGCT MARKET LANDSCAPE OVERVIEW

U.S. TGCT Market Landscape: Imatinib is the Leading TKI Prescribed[1]



Imatinib **Pexidartinib** **Other TKI** (sunitinib or nilotinib)

Avg. Duration of Therapy
Imatinib: ~18 months, Pexidartinib: ~8 months[2]

Existing Product Profiles and Unmet Need

Imatinib

- Not FDA or EMA approved for TGCT
- Weak CSF1R inhibitor
- Guideline listed based on retrospective data showing 19% ORR[3,4]

Pexidartinib

- The only FDA approved agent for TGCT, not approved by EMA
- Inhibitor of CSF1R, KIT, FLT3, and PDGFRA/B
- Boxed warning for hepatoxicity, REMS, intensive liver monitoring

High Unmet Need

- Lifelong condition with significant morbidity
- HCPs and patients cite desire for effective therapy without having to sacrifice safety and tolerability[5]

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Notes: TGCT=tenosynovial giant cell tumor; TKI = Tyrosine Kinase Inhibitor. (1) Symphony Health IDV Claims; Analysis Period: 7/1/2020 – 6/1//2021; (2) Symphony Health IDV Claims; Analysis Period: 1/1/2008 – 2/1/2021; Patient Qualification: 1/1/2016 – 12/31/2020, Symphony Health IDV Claims; Analysis Period: Q3 2019 – Q32022 and Symphony Health Metys database (estimates calculated from pexidartinib data 2019 – 2022); (3) NCCN Guidelines Version 2.2022 Soft Tissue Sarcoma; (4) Cassier et al Cancer 2012:119:1649-1655; (5) Internal Deciphera market research.

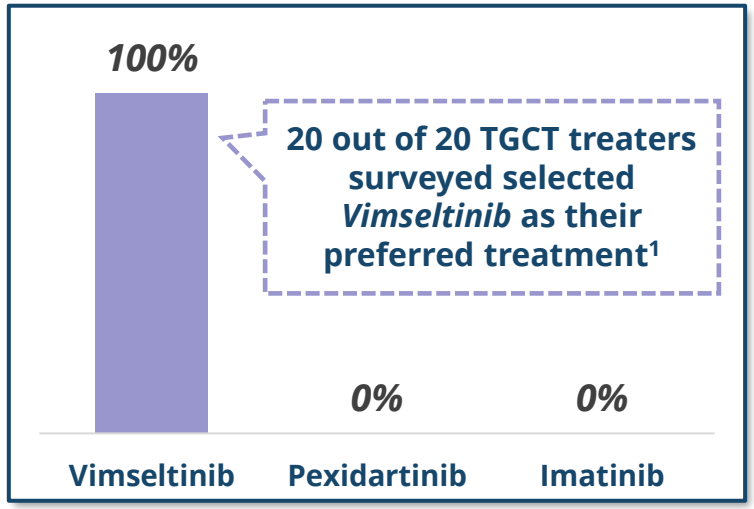
MARKET RESEARCH HIGHLIGHTS THE POTENTIAL FOR VIMSELTINIB TO DELIVER A BEST-IN-CLASS PROFILE AND ESTABLISH A NEW STANDARD OF CARE IN TGCT

Relative Scoring of Key Product Attributes

Clinical Attribute		Vimseltinib	Pexidartinib	Imatinib
Efficacy	***Tumor Response*** *(Objective Response, CBR)*			
	PROs *(Improvement in Pain & Stiffness)*			Limited Data
Safety	***Grade 3/4 AEs***			
	Hepatotoxicity			Not Reported in TGCT
	Discontinuation Rates *(Due to any TEAEs)*			

Highly Compelling | Moderately Compelling | Less Compelling

Preferred Systemic Treatment For TGCT



Clinical Profile: *"This is very impressive data. Well over half the patients responded to the medication with excellent outcomes. Pain measurements and mobility scores are quite remarkable compared to anything currently available" – Onc*

Efficacy: *"It's great to see that the best overall response numbers improve over long-term. In fact, this would probably lead me to keep some patients on it longer than I usually would." – Onc*

Treatment Choice: *"I would give [vimseltinib] to all my future TGCT patients" - Onc*

TGCT Treater Sentiments on Vimseltinib Profile

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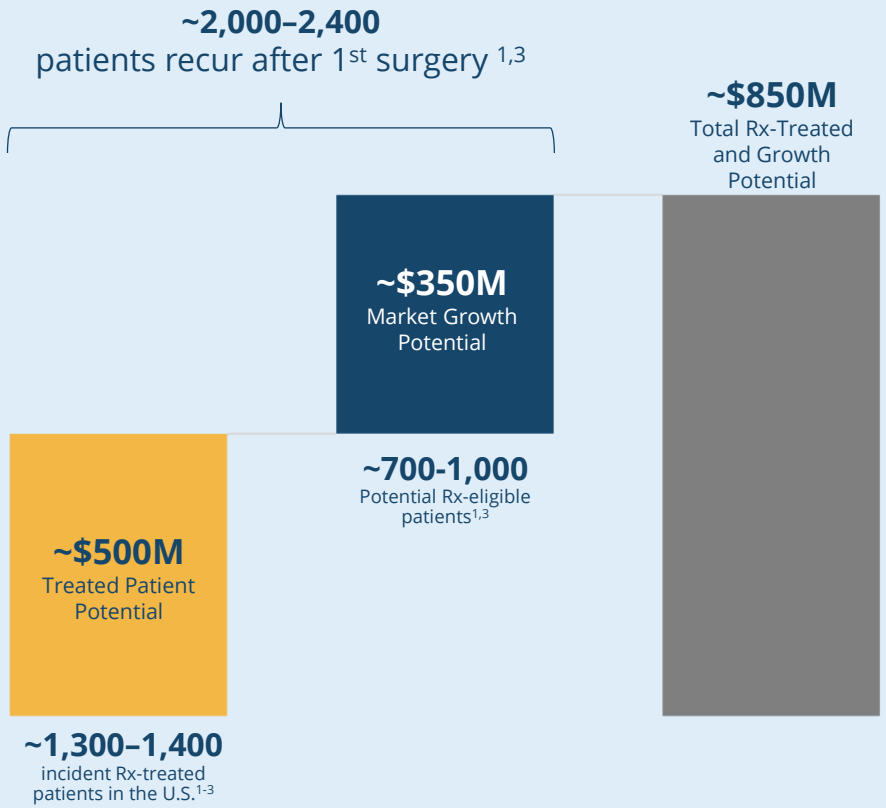
Notes: Qualitative market research conducted by Deciphera in Aug 2022 comparing target product profiles of vimseltinib (blinded), pexidartinib, and imatinib based on USPIs and published/presented literature for each product's efficacy and safety in TGCT diagnosed patients (n=20 HCPs). Hepatotoxicity as defined by increased AST/ALT rates. No head-to-head/comparative studies have been conducted. CBR = Clinical Benefit Rate, PROs = Patient Reported Outcomes, AEs = Adverse Events, TEAEs = Treatment-Emergent Adverse Events. (1) HCP asked to select their preferred (only 1) product to systemically treat TGCT patients based on current clinical profiles.

SIGNIFICANT OPPORTUNITY TO BENEFIT PATIENTS WITH TGCT GLOBALLY

U.S. Total Addressable Market Based on Incident Population



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U.S. Prevalent Population

~8,000
estimated prevalent Rx-eligible patients in the U.S.[1-3]

\+

E.U. Opportunity

- Comparable incidence and recurrence rates in Europe[1,2]
- No approved therapies for TGCT

Notes: TGCT=tenosynovial giant cell tumor; (1) Mastboom et al. Acta Orthopaedica. 2017;88(6):688-694; (2) Internal Deciphera estimates based on market research and secondary data; estimates are inherently uncertain; (3) Ehrenstein. J Rheumatol. 2017;44(10):1476-1483. Total Addressable Market calculated by estimated patient incidence x 18 months duration x current pexidartinib WAC price and assumes opportunity at steady state.

A MULTICENTER, RANDOMIZED, PLACEBO-CONTROLLED, DOUBLE-BLIND STUDY

Top-line Results Expected in 4Q 2023

International Study with ~40 Sites

DOUBLE-BLIND PERIOD

Part 1: Eligible study participants will be assigned to receive either vimseltinib or matching placebo for 24 weeks.

Vimseltinib (n = 80)
30 mg BIW (24 weeks)

2:1 Randomization
Enrollment Complete
(n=120)

Placebo (n = 40)
(24 weeks)

OPEN-LABEL PERIOD

Part 2: A long-term treatment phase in which all participants receive open-label vimseltinib.

Open-Label Study

Patients have the option to continue or cross over to vimseltinib 30 mg BIW

MOTION Phase 3 Study

will assess the efficacy and safety of vimseltinib for the treatment of patients with TGCT not amenable to surgery

Primary Endpoint

- Objective response rate (ORR) at week 25

Key Secondary Endpoints

- ORR per tumor volume score
- Range of motion (ROM)
- Patient-reported outcomes

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Notes: BIW=twice weekly; TGCT=tenosynovial giant cell tumor.

DCC-3116

HIGHLY SELECTIVE SWITCH-CONTROL INHIBITOR OF THE ULK KINASE

- **DCC-3116** is a potential first-in-class small molecule designed to inhibit cancer autophagy by targeting the ULK kinase
- The combination dose-escalation portion of the **DCC-3116** Phase 1 study is underway
- Pfizer supply agreement to support a new combination study evaluating **DCC-3116** + encorafenib/cetuximab in CRC

Expected 2023 Milestones[1]

Present preclinical data on new combinations

Completed (2Q 2023)

Present updated Phase 1 single agent and initial combination dose escalation data

2H 2023

Initiate escalation cohort for encorafenib/cetuximab

2H 2023

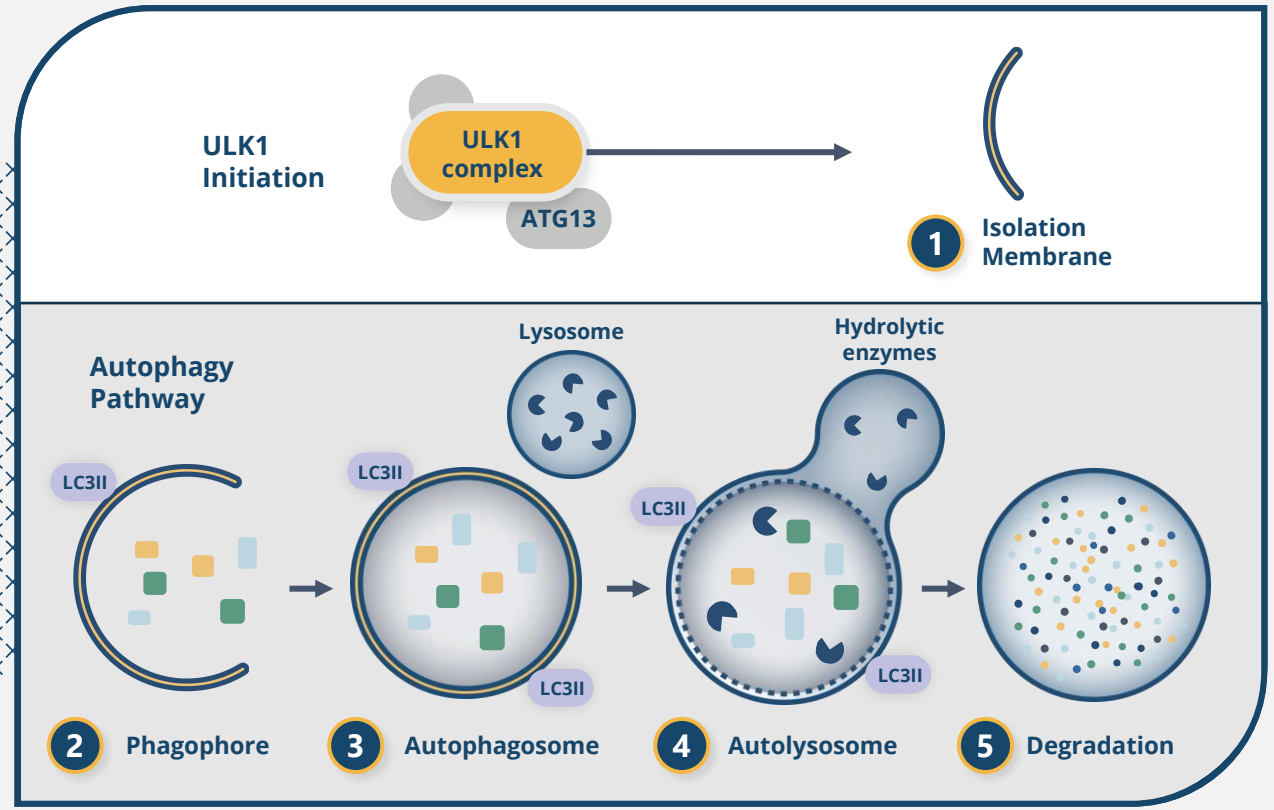
Initiate MEK/G12C expansion cohort(s)

2H 2023

Notes: CRC=colorectal cancer; G12C=single point mutation with a glycine-to-cysteine substitution at codon 12; MEK=mitogen-activated extracellular signal-regulated kinase; ULK=unc-51-like kinase; (1) 2023 Corporate Goal.

AUTOPHAGY: A BROAD RESISTANCE MECHANISM IN CANCER

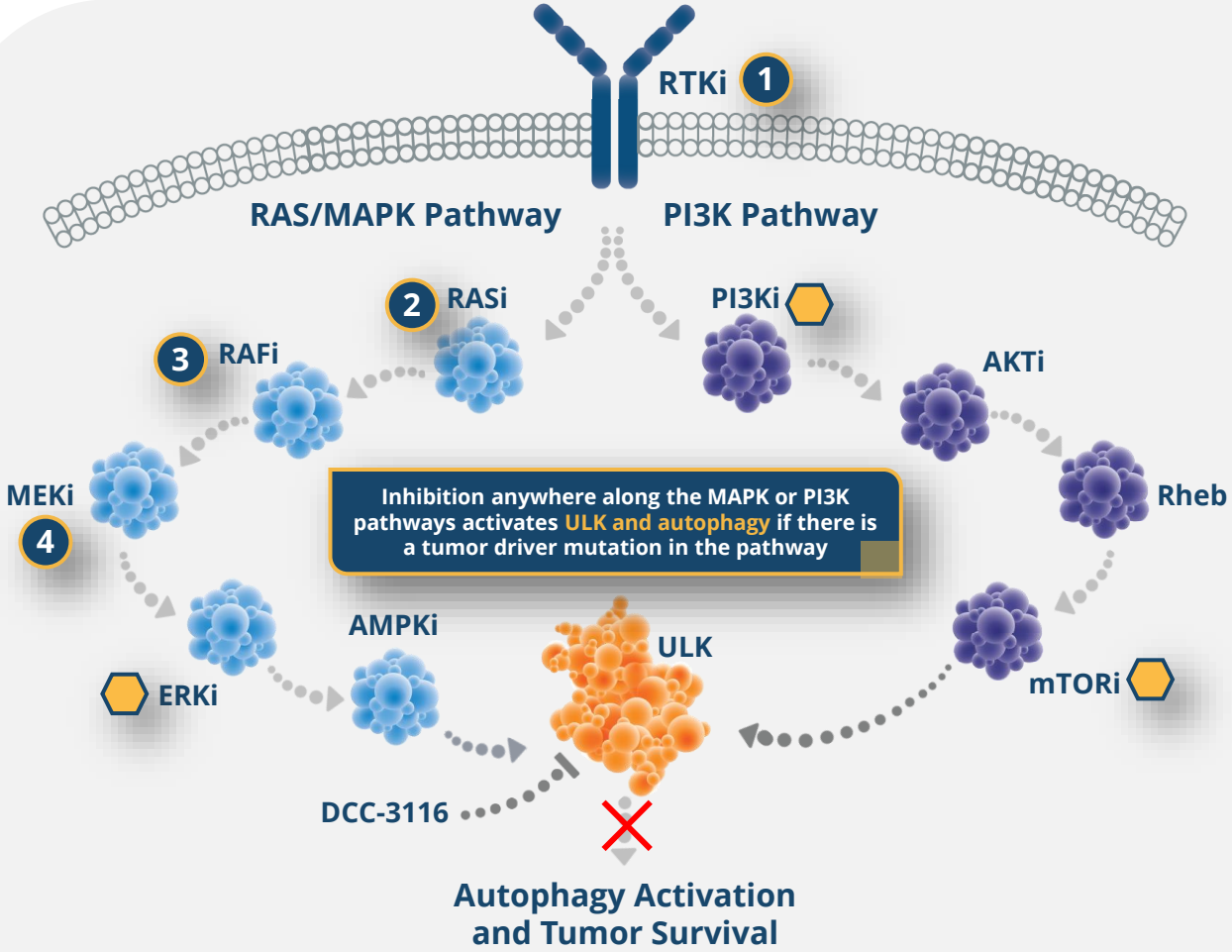
ULK: Initiating Factor for Autophagy



- Autophagy is a catabolic process in which cells recycle components to generate energy
- RTK-, RAS-, and RAF-mutant cancer cells activate autophagy in order to survive stresses or damage **due to anti-cancer therapeutic intervention** with MAPK and PI3K pathway inhibitors
- The **ULK kinase initiates the autophagy pathway** and provides a potential targeted approach to selectively inhibit autophagy in these cancers

Notes: G12C=single point mutation with a glycine-to-cysteine substitution at codon 12; MAPK=mitogen-activated protein kinase; PI3K=phosphatidylinositol-3 kinase; RAF=rapidly accelerated fibrosarcoma; RAS=rat sarcoma gene; RTK=receptor tyrosine kinase; ULK=unc-51-like kinase; ATG13= Autophagy-related protein 13.

CENTRAL ROLE FOR AUTOPHAGY IN THE RAS/MAPK AND PI3K PATHWAYS



GROWING PRECLINICAL VALIDATION FOR ROLE OF AUTOPHAGY IN CANCER

1 DCC-3116 In Combination with RTK Inhibition

- DCC-3116 exhibits synergy with ripretinib, osimertinib, and afatinib, resulting in tumor regression in EGFR-mutant NSCLC and GIST *in vivo*

2 DCC-3116 In Combination with KRAS^{G12C} Inhibition

- DCC-3116 exhibits synergy with sotorasib and adagrasib resulting in tumor regression in KRAS^{G12C}-mutant NSCLC *in vivo*

3 DCC-3116 In Combination with RAF Inhibition

- DCC-3116 exhibits synergy in combination with encorafenib in BRAFm CRC *in vivo*

4 DCC-3116 In Combination with MEK Inhibition

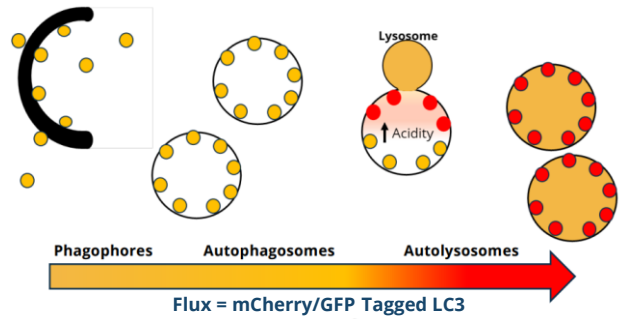
- DCC-3116 exhibits synergy in combination with trametinib and inhibited pancreatic, lung, and melanoma xenograft tumor growth

Other targets where therapeutic intervention activates ULK and autophagy

Notes: AKT=protein kinase B; AMPK=AMP-activated protein kinase; EGFR=epidermal growth factor receptor; ERK=extracellular signal-regulated kinase; G12C=single point mutation with a glycine-to-cysteine substitution at codon 12; GIST=gastrointestinal stromal tumor; KRAS=Kirsten rat sarcoma virus; LKB1=liver kinase B1; MAPK=mitogen-activated protein kinase; MEK=mitogen-activated extracellular signal-regulated kinase; mTOR=mammalian target of rapamycin; NSCLC=non–small-cell lung cancer; PI3K=phosphatidylinositol-3 kinase; RAF=rapidly accelerated fibrosarcoma; RAS=rat sarcoma gene; Rheb=ras homolog enriched in brain; RTK=receptor tyrosine kinase; ULK=unc-51-like kinase.

DCC-3116 OBSERVED PRECLINICALLY TO DURABLY AND POTENTLY INHIBIT AUTOPHAGY INDUCED BY MULTIPLE PATHWAY INHIBITORS

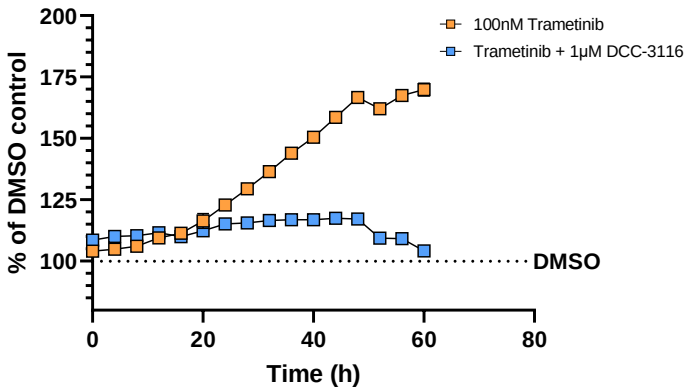
AUTOPHAGIC FLUX MATURATION



- RTK/RAS/MAPK induces significant autophagy flux
- DCC-3116 can sustainably inhibit this induction

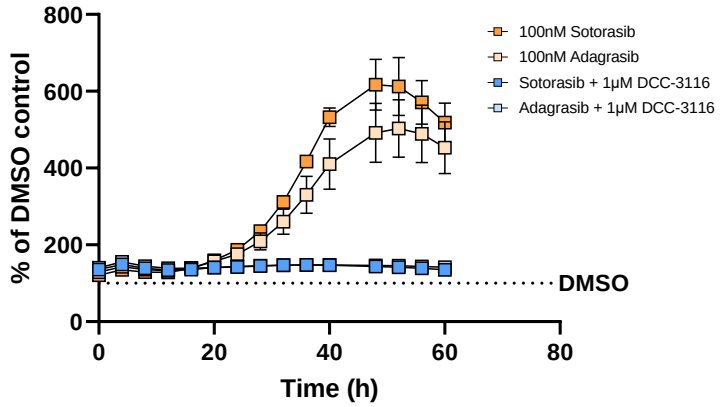
DCC-3116 + Trametinib

PDAC: Trametinib-induced Autophagy Induction Over Time (mCherry/GFP Tagged LC3)[1]



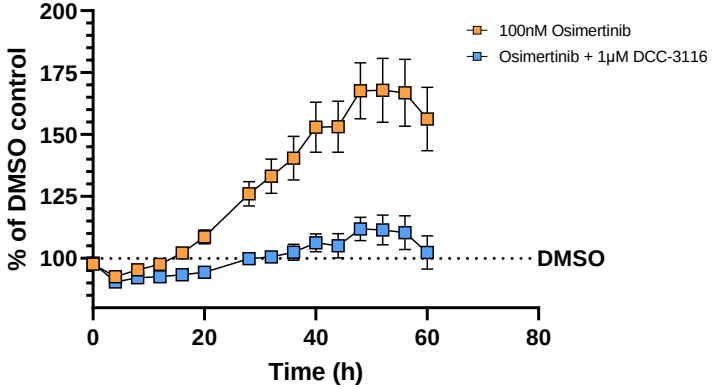
DCC-3116 + KRAS^{G12C} Inhibitor

NSCLC: Sotorasib vs. Adagrasib-induced Autophagy Induction Over Time (mCherry/GFP Tagged LC3)[2]



DCC-3116 + EGFR Inhibitor

NSCLC: Osimertinib-induced Autophagy Induction Over Time (mCherry/GFP Tagged LC3)[1]

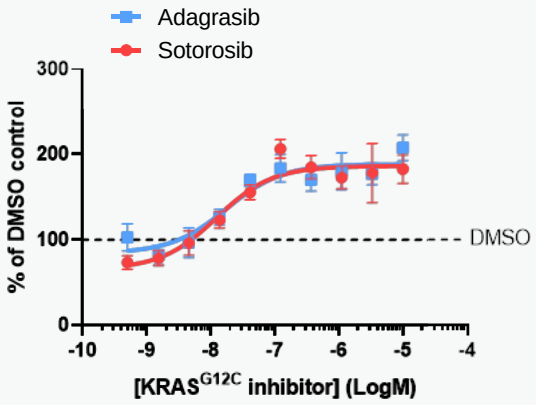


Notes: EGFR=epidermal growth factor receptor; G12C=single point mutation with a glycine-to-cysteine substitution at codon 12; KRAS=Kirsten rat sarcoma virus; MAPK=mitogen-activated protein kinase; NSCLC=non–small-cell lung cancer; PDAC=pancreatic ductal adenocarcinoma; RAS=rat sarcoma gene; RTK=receptor tyrosine kinase; (1) data published in Deciphera's Annual Report on Form 10-K for the year ended December 31, 2021; (2) data presented at the AACR Meeting 2022.

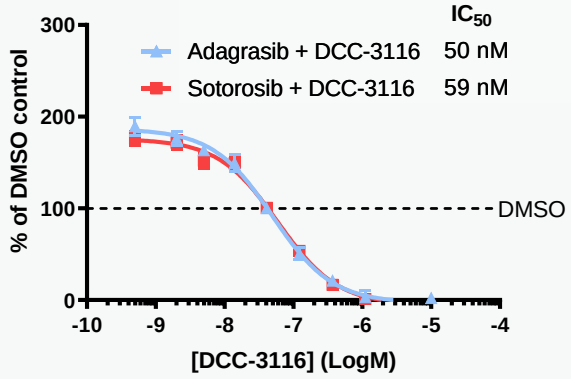
DCC-3116 INHIBITS RAS PATHWAY INHIBITOR-INDUCED ULK ACTIVITY

DCC-3116 Reverses KRAS^{G12C} Inhibitor-Induced ULK Activation

NSCLC: H358 pATG13 ELISA
KRAS^{G12C} Inhibitors Induce ULK Activity

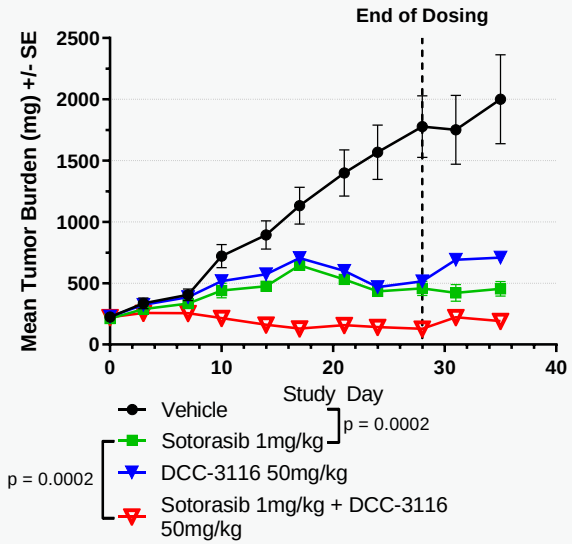


NSCLC: H358 pATG13 ELISA
DCC-3116 Inhibits KRAS^{G12C} Inhibitor-Induced ULK Activity

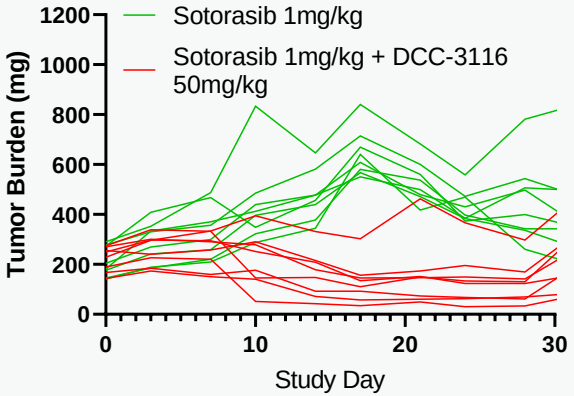


DCC-3116 Demonstrated Deeper and Longer Regressions in Combination with Sotorasib

NSCLC: H358 Tumor Growth
DCC-3116 + Sotorasib 1mg/kg



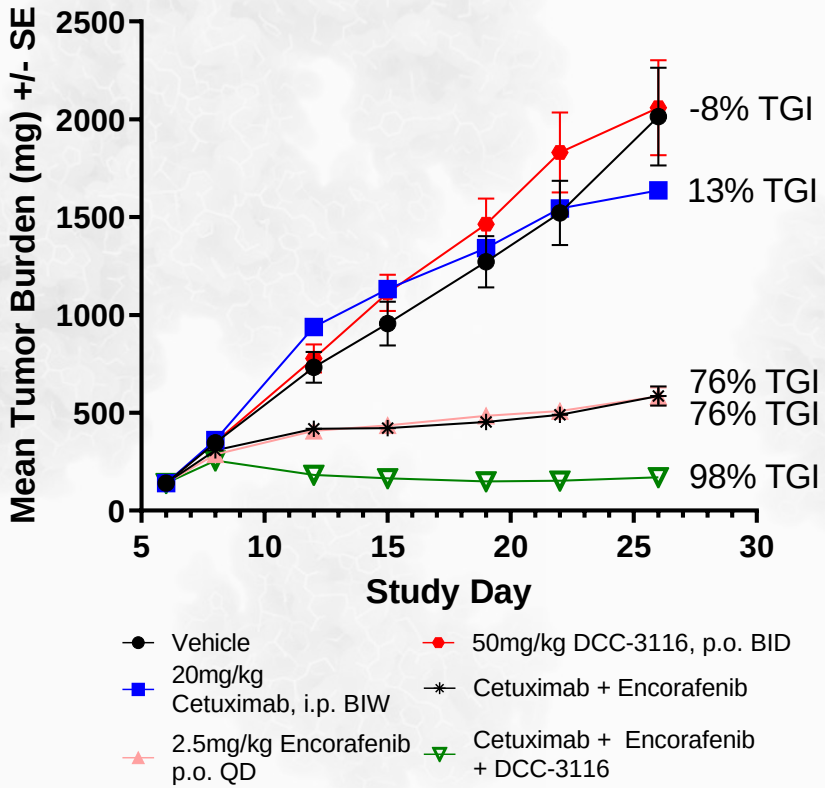
NSCLC: H358 Tumor Growth
DCC-3116 + Sotorasib 1mg/kg



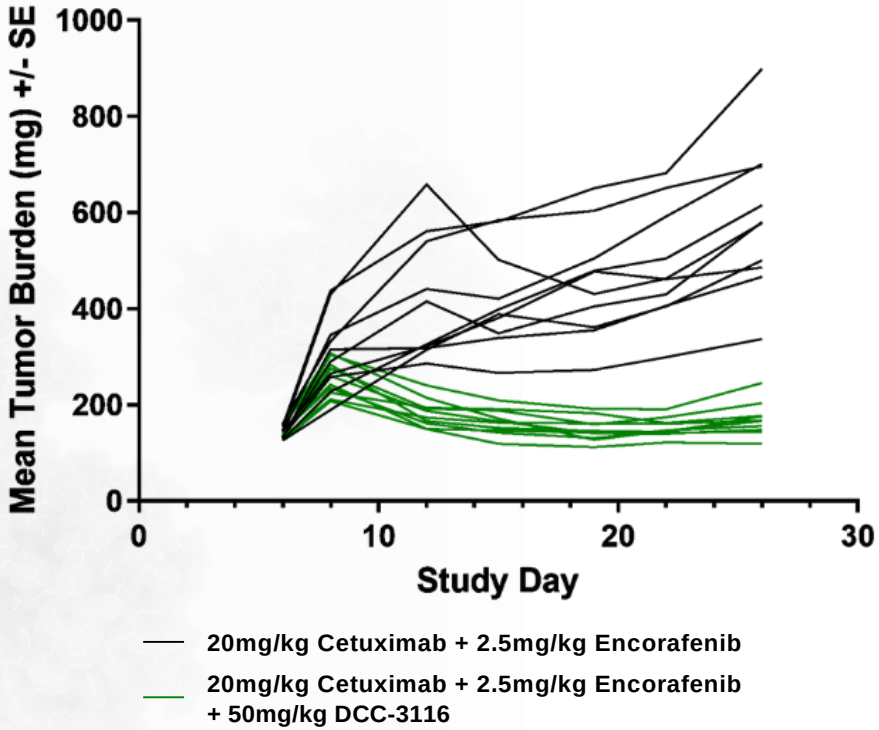
Notes: Data presented at AACR 2022; ATG13= Autophagy Related 13; ATG14=Autophagy Related 14: BID=twice daily; MEK=MAPK/ERK kinase; pATG13=phospho-ATG13; pATG14=phospho-ATG14; PBMCs=peripheral blood mononuclear cells; PD=pharmacodynamics; PK=pharmacokinetics; ULK=unc-51-like autophagy-activating kinase.

DCC-3116 INDUCES TUMOR REGRESSIONS IN COMBINATION WITH ENCORAFENIB AND CETUXIMAB *IN VIVO*

Colo-205: BRAF^{V600E} Colorectal Cancer Model



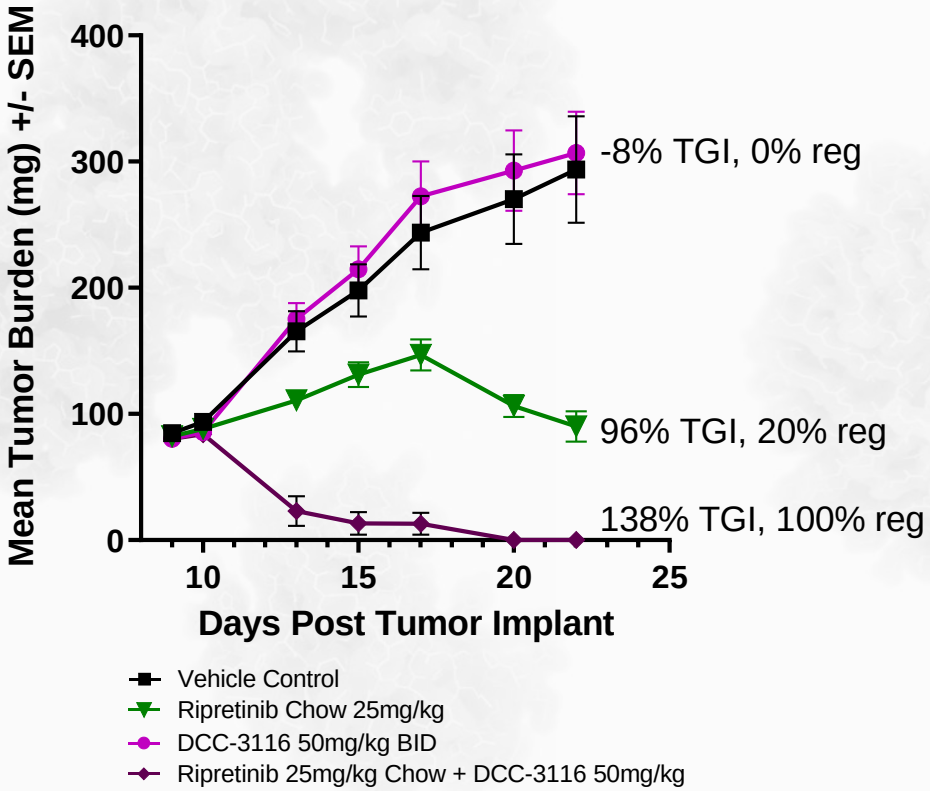
Colo-205: BRAF^{V600E} Colorectal Cancer Model



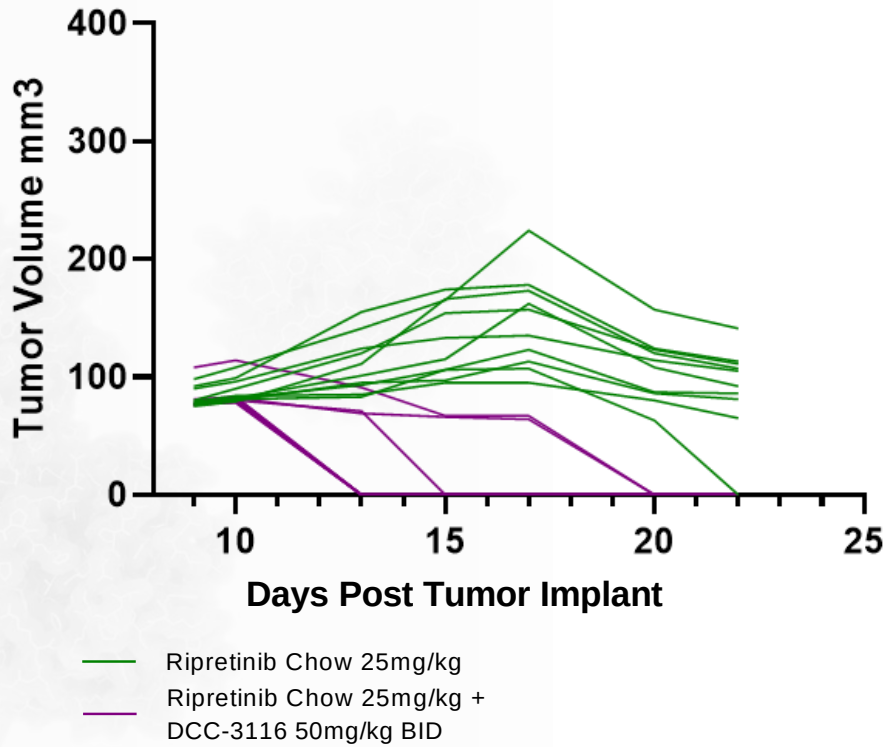
Notes: Data presented at the AACR Annual Meeting 2023; % reg is the percent of mice that had regressions. %TGI and %Reg were calculated at the last day of dosing; BID=twice daily; BIW=2 times a week; BRAF=proto-oncogene b-RAF; PO=orally; QD=once daily; Reg=regression; TGI=tumor growth inhibition

DCC-3116 SHOWS 100% TUMOR REGRESSIONS IN COMBINATION WITH RIPRETINIB IN KIT EXON 11 MUTATED GIST XENOGRAFT MODEL

GIST-T1: Tumor Growth Inhibition



GIST-T1: Tumor Volume



Notes: Data presented at the AACR Annual Meeting 2023; % reg is the percent of mice that had regressions; %TGI and %Reg were calculated at the last day of dosing; BID=twice daily; GIST=gastrointestinal stromal tumor; GIST-T1=Exon 11 del; KIT=KIT proto-oncogene receptor tyrosine kinase; Reg.=regression; TGI=tumor growth inhibition.

SUMMARY OF INITIAL SINGLE AGENT PHASE 1 STUDY RESULTS

- DCC-3116 was well tolerated at doses from 50 to 300 mg BID, which achieved exposure and ULK1/2 inhibition associated with anticancer efficacy with MEK inhibitors in preclinical studies
- Treatment-related TEAEs were mainly Grade 1/2, except for related asymptomatic and reversible Grade 3 ALT increases

DCC-3116 EXPOSURE APPEARED TO INCREASE DOSE PROPORTIONALLY ACROSS 50 – 300 mg BID

All DOSES ACHIEVED EXPOSURE AND ULK1/2 INHIBITION ASSOCIATED WITH EFFICACY IN PRECLINICAL STUDIES

NO DLTs OR TREATMENT-RELATED SAEs OBSERVED

MONOTHERAPY RESULTS DEMONSTRATED STABLE DISEASE AS BEST OVERALL RESPONSE

NOV '22 UPDATE

MAXIMUM TOLERATED DOSE NOT REACHED

50 mg BID SELECTED AS STARTING DOSE FOR COMBINATION DOSE ESCALATION

FIRST PATIENT TREATED IN COMBINATION DOSE ESCALATION

deciphera®

Notes: Data presented at the ESMO Congress 2022; data cutoff: June 9, 2022; ALT=alanine aminotransferase; BID=twice daily; DLTs=dose-limiting toxicities; MEK=MAPK/ERK kinase; PD=pharmacodynamics; PK=pharmacokinetics; SAE=serious adverse event; TEAE=treatment-emergent adverse event; ULK=unc-51-like autophagy-activating kinase.

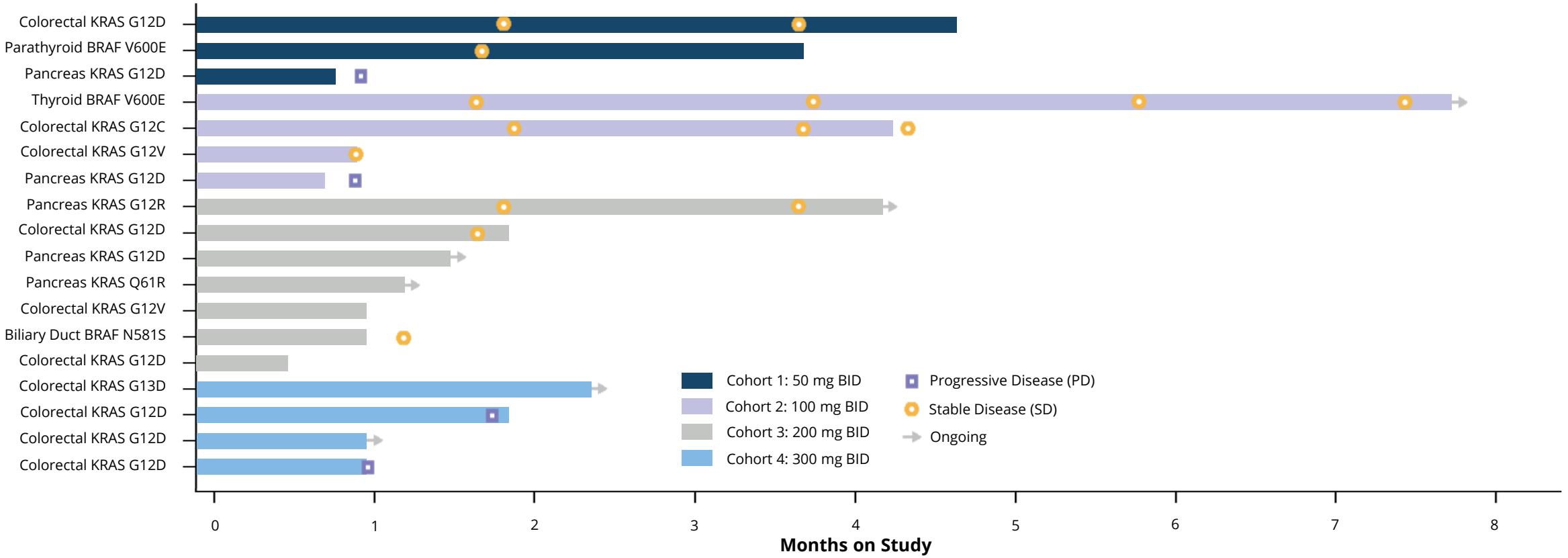
TEAEs REGARDLESS OF RELATEDNESS (≥15% OF PARTICIPANTS)

Preferred term	DCC-3116 Monotherapy Cohorts								All Participants
	Cohort 1 50 mg BID (n = 3)		Cohort 2 100 mg BID (n = 4)		Cohort 3 200 mg BID (n = 7)		Cohort 4 300 mg BID (n = 4)		n (%) N = 18
	Grade 1/2	Grade 3	Grade 1/2	Grade 3	Grade 1/2	Grade 3	Grade 1/2	Grade 3	All grades
Fatigue	2	0	1	0	3	0	1	0	7 (39%)
Dehydration	0	0	0	0	2	0	2	0	4 (22%)
ALT increased	0	0	0	0	0	1	1	1	3 (17%)
Anaemia	0	2	0	1	0	0	0	0	3 (17%)
AST increased	0	0	0	0	2	0	1	0	3 (17%)
Decreased appetite	1	0	1	0	0	0	1	0	3 (17%)
Hyponatraemia	1	0	0	0	1	0	1	0	3 (17%)
Nausea	0	0	1	0	0	0	2	0	3 (17%)
Vomiting	1	0	1	0	1	0	0	0	3 (17%)

- No DLTs or treatment-related serious TEAEs were observed
- Most treatment-related TEAEs were Grade 1/2; no treatment-related Grade 4 TEAEs
- Two related asymptomatic, reversible Grade 3 ALT increased TEAEs led to dose interruption and reduction

Notes: Data presented at the ESMO Congress 2022; data cutoff: June 9, 2022; ALT=alanine aminotransferase; AST=aspartate aminotransferase; BID=twice daily; DLTs=dose-limiting toxicities; TEAE=treatment-emergent adverse event.

TREATMENT DURATION AND RECIST v1.1 RESPONSE ASSESSMENTS



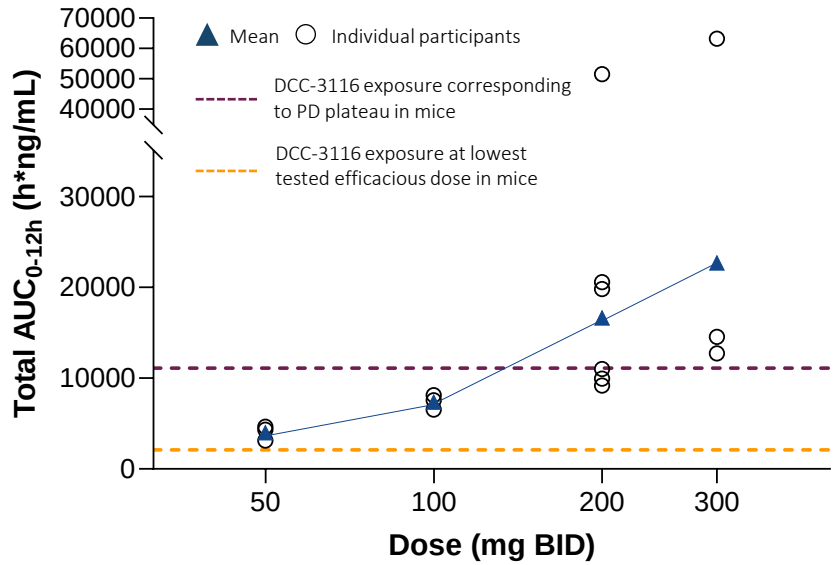
- Best overall response was stable disease
- Disease control rate at week 16 was 29% (4 of 14 response-evaluable participants)

Notes: Data presented at the ESMO Congress 2022; data cutoff: June 9, 2022; BID=twice daily; BRAF=proto-oncogene b-RAF; KRAS=Kirsten rat sarcoma virus.

INITIAL PK/PD DATA AT ALL DOSE LEVELS IS CONSISTENT WITH PREDICTED ACTIVITY BASED ON PRECLINICAL STUDIES

Pharmacokinetics

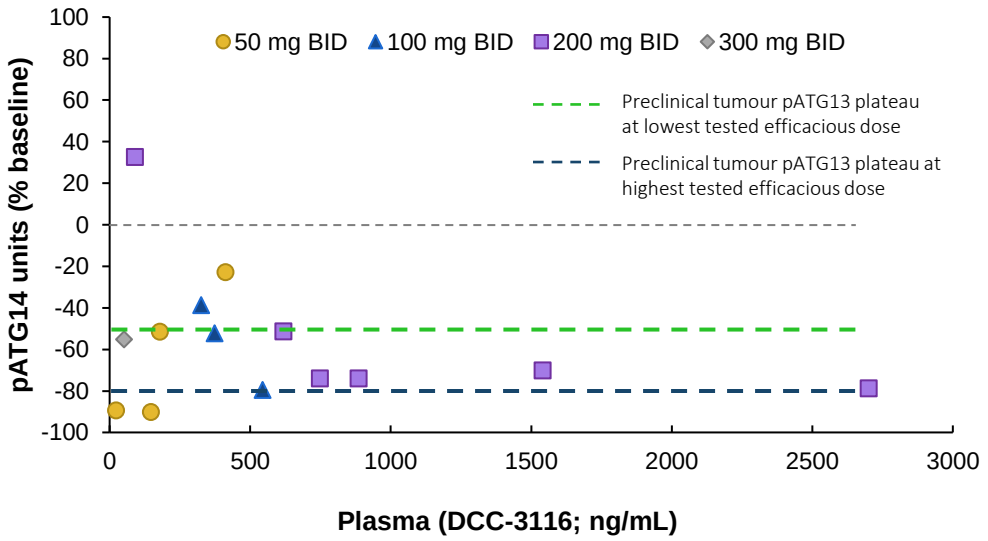
Total Individual and Mean $AUC_{0–12h}$ vs. Dose



- DCC-3116 AUC appeared to increase proportionally to dose from 50 to 300 mg BID
- DCC-3116 AUC at all dose levels at or above AUC of lowest tested dose that was effective in preclinical studies

Pharmacodynamics

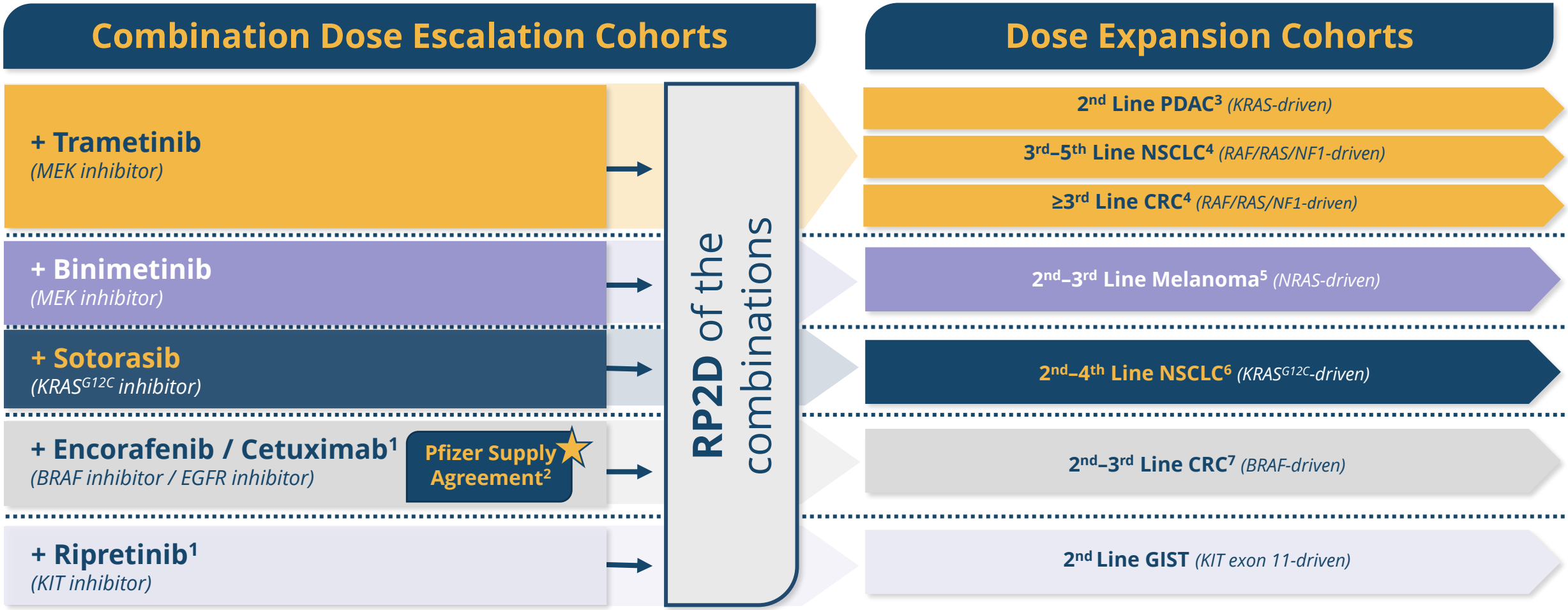
Inhibition of Phosphorylation of ULK1/2 Substrate ATG14 in PBMCs at Trough



- Decreases in phosphorylation of direct ULK1/2 substrates ATG14 and ATG13 both indicate inhibition of ULK1/2
- At all DCC-3116 dose levels, pATG14 in PBMCs was in range of preclinical pATG13 in tumors at doses that achieved anticancer effects with MEK inhibitor
- pATG14 inhibition in PBMCs reached maximum pATG13 inhibition observed preclinically in tumors

Notes: Data presented at the ESMO Congress 2022; data cutoff: June 9, 2022; ATG13= Autophagy Related 13; ATG14=Autophagy Related 14: BID=twice daily; MEK=MAPK/ERK kinase; pATG13=phospho-ATG13; pATG14=phospho-ATG14; PBMCs=peripheral blood mononuclear cells; PD=pharmacodynamics; PK=pharmacokinetics; ULK=unc-51-like autophagy-activating kinase.

PHASE 1 COMBINATION COHORTS EVALUATING MULTIPLE COMBINATIONS



Notes: CRC=colorectal cancer; G12C=single point mutation with a glycine-to-cysteine substitution at codon 12; KRAS=Kirsten rat sarcoma virus; MEK=MAPK/ERK kinase; MTD=maximum tolerated dose; NRAS=neuroblastoma RAS viral oncogene homolog; NSCLC=non–small-cell lung cancer; PDAC=pancreatic ductal adenocarcinoma; RAF=rapidly accelerated fibrosarcoma serine/threonine protein kinase; RAS=rat sarcoma gene; RP2D=recommended Phase 2 dose; (1) Corporate goal planned for 2H 2023; (2) Under the terms of the clinical trial collaboration and supply agreement with Pfizer, Inc., Deciphera will sponsor the trial and Pfizer will supply encorafenib at no cost; (3) with a documented mutation in KRAS; (4) with a documented mutation in KRAS, NRAS, NF1, or BRAF; (5) with a documented mutation in NRAS; (6) with a documented mutation in $KRAS^{G12C}$; (7) with a documented mutation in $BRAF^{V600E}$.

PROPRIETARY DRUG DISCOVERY PLATFORM

DECIPHERA'S PROPRIETARY DRUG DISCOVERY PLATFORM

DRIVING INNOVATION THROUGH OUR PROVEN DISCOVERY ENGINE

Fueled by our **proprietary drug discovery platform**, we intend to advance new drug candidates into clinical development to continue to fulfill our mission to defeat cancer

Expected 2023 Milestones

Nominate development candidate for pan-KIT Inhibitor

Completed (1Q 2023)

Present data on the preclinical profile of DCC-3084

Completed (1Q 2023)

Present new preclinical data from research programs

Completed (1Q 2023)

Submit IND to FDA for DCC-3084

2H 2023

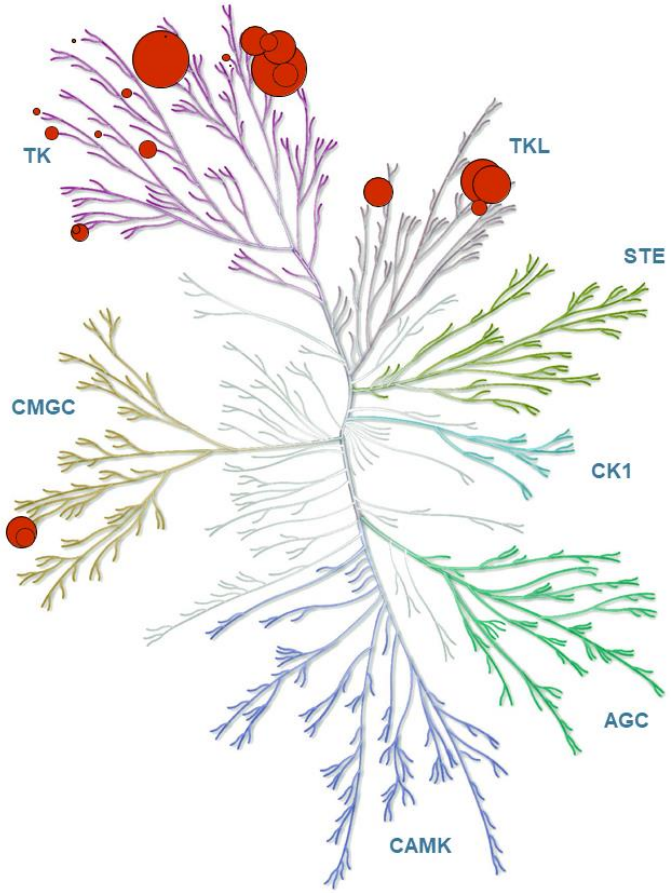
deciphera®

Notes: FDA=U.S. Food and Drug Administration; IND=Investigational New Drug Application; KIT=KIT proto-oncogene receptor tyrosine kinase.

DCC-3084

decīphera

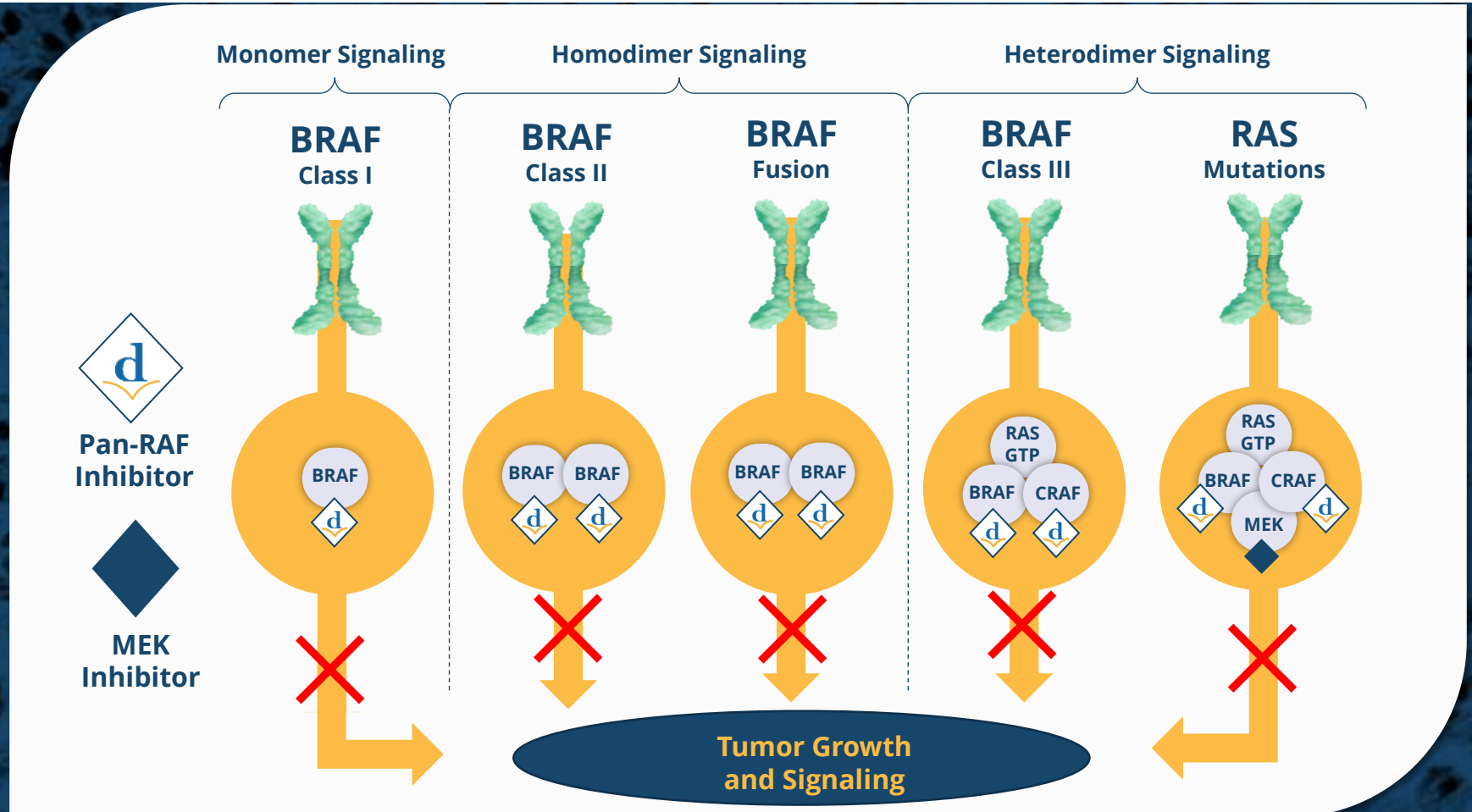
DCC-3084 IS A POTENT AND SELECTIVE PAN-RAF INHIBITOR



- DCC-3084 is a **potential best-in-class pan-RAF inhibitor** engineered using Deciphera's proprietary switch-control platform
- **Potent and selective inhibitor of BRAF and CRAF kinases**, shown preclinically to target all relevant aberrant signaling mechanisms (monomers, homodimers, heterodimers)
- **High permeability**, **CNS penetrance**, and **solubility** at gastric pH to facilitate tumor access
- **Long residency time**, **low efflux,** and **transporter inhibition** to enable durable efficacy
- **Strong pre-clinical data** supports single agent use in RAF and RAS mutant cancers, and deeper responses in combination with MEK inhibitors

Notes: BRAF=proto-oncogene b-RAF; CNS=central nervous system; CRAF=proto-oncogene c-RAF; MEK=mitogen-activated extracellular signal-regulated kinase; RAF=rapidly accelerated fibrosarcoma serine/threonine protein kinase; RAS=rat sarcoma gene.

POTENTIAL BEST-IN-CLASS BRAF/BRAF HOMODIMER AND BRAF/CRAF HETERODIMER INHIBITOR



Notes: BRAF=proto-oncogene b-RAF; CRAF=proto-oncogene c-RAF; GTP=guanosine triphosphate; MEK=mitogen-activated extracellular signal-regulated kinase; RAS=rat sarcoma gene.

KEY PROPERTIES FOR A BEST-IN-CLASS PAN-RAF INHIBITOR

Potency & Selectivity

- BRAF/CRAF inhibition of signaling via monomers, homodimers, and heterodimers
- Long on-target residency time
- Limited inhibition of off-target kinases

Pharmaceutical Properties

- High permeability, low efflux and inhibition of resistance transporters to maximize efficacy
- Improved solubility to enhance target inhibition

Tissue Distribution

- High accumulation in tumor cells
- CNS penetration for primary and secondary brain tumors

Notes: BRAF=proto-oncogene b-RAF; CNS=central nervous system; CRAF=proto-oncogene c-RAF; RAF=rapidly accelerated fibrosarcoma serine/threonine protein kinase.

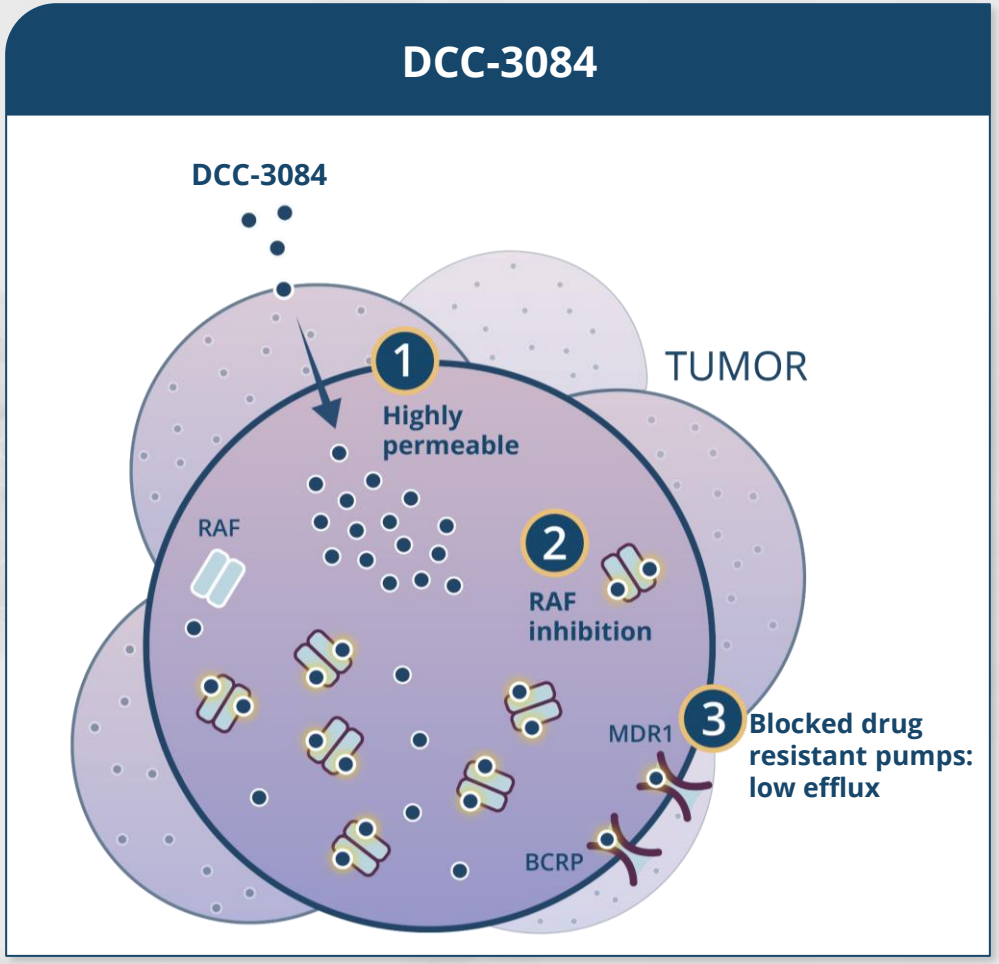
DCC-3084 EXHIBITS OVERALL BEST-IN-CLASS INHIBITION OF CLASS I, II, AND III BRAF-MUTANTS AND RAF FUSION CELL LINES

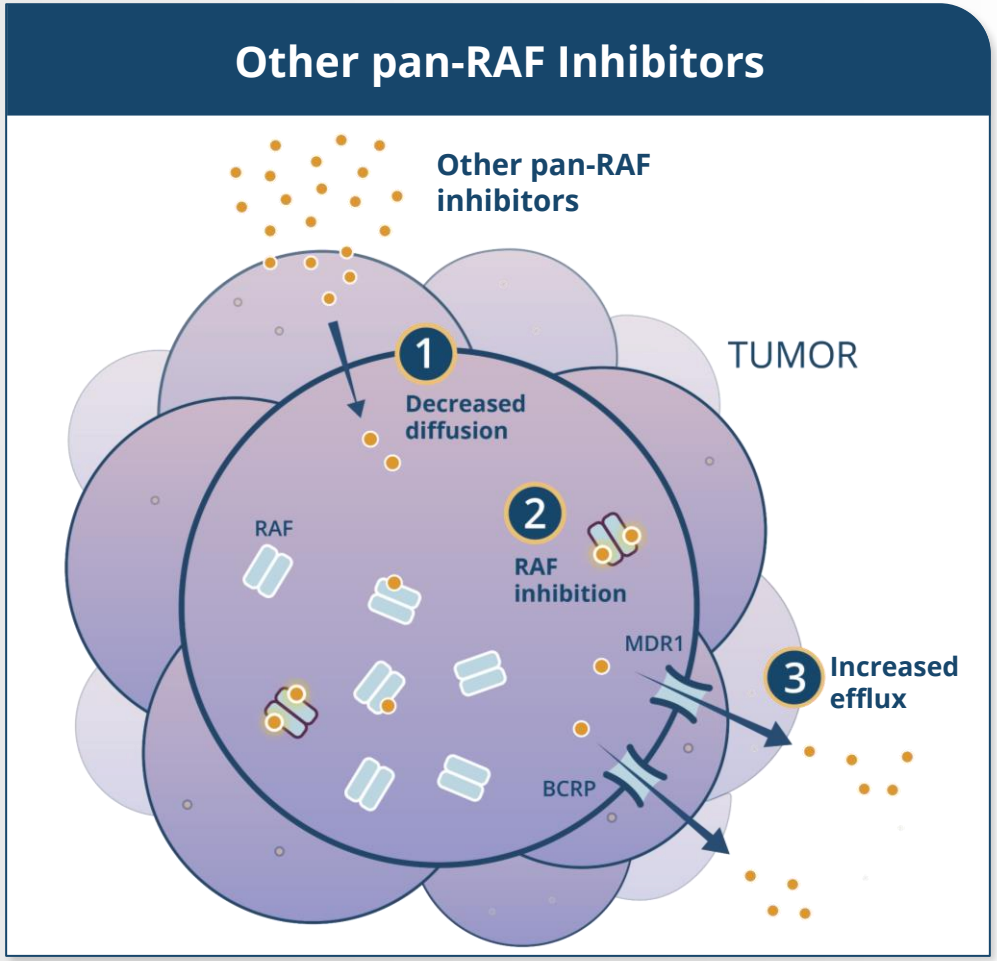
	Class I		Class II		Fusion	Class III + NRAS
Inhibitor	**A375**	**HT-29**	**BxPC-3**	**H2405**	**WM3928**	**WM3629**
DCC-3084	**54**	**13**	**61**	**74**	**42**	**3**
tovorafenib	3,000	5,270	1,100	603	669	305
naporafenib	438	228	19	465	90	3
belvarafenib	144	128	59	149	14	2
exarafenib	170	101	254	549	98	17
JZP815	141	47	200	47	133	2

IC_{50} (nM)

Notes: Data presented at the AACR Annual Meeting 2023; BRAF=proto-oncogene b-RAF; NRAS=neuroblastoma RAS viral oncogene homolog; RAF=rapidly accelerated fibrosarcoma serine/threonine protein kinase.

DCC-3084 HAS EXCELLENT PERMEABILITY, LOW EFFLUX, AND IS A STRONG INHIBITOR OF THE MDR1 AND BCRP DRUG RESISTANCE TRANSPORTERS



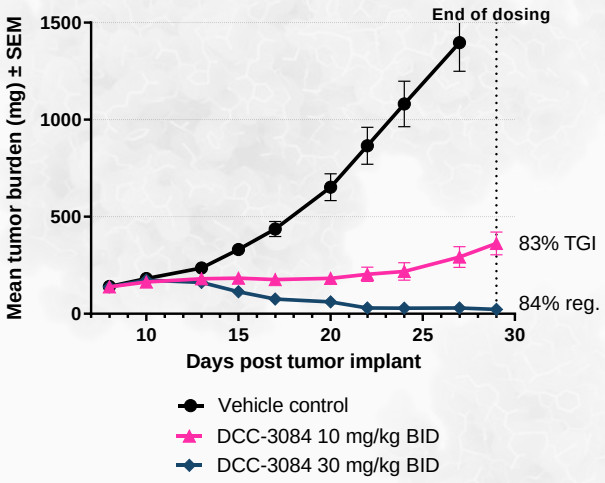


Notes: BCRP=breast cancer resistance protein transporter; MDR1=multidrug resistance mutation transporter; RAF=rapidly accelerated fibrosarcoma serine/threonine protein kinase.

DCC-3084 PRODUCES TUMOR REGRESSIONS IN BRAF MUTANT CANCER MODELS AS A SINGLE AGENT

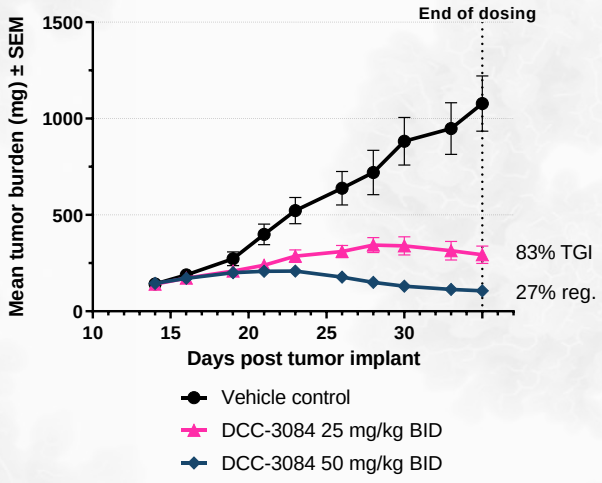
BRAF Class I

A375: BRAF Mutant Melanoma Model



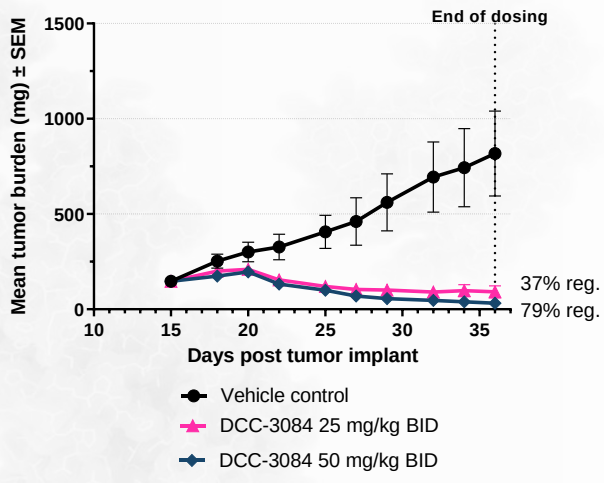
BRAF Class II

BxPC-3: BRAF Mutant Pancreatic Model



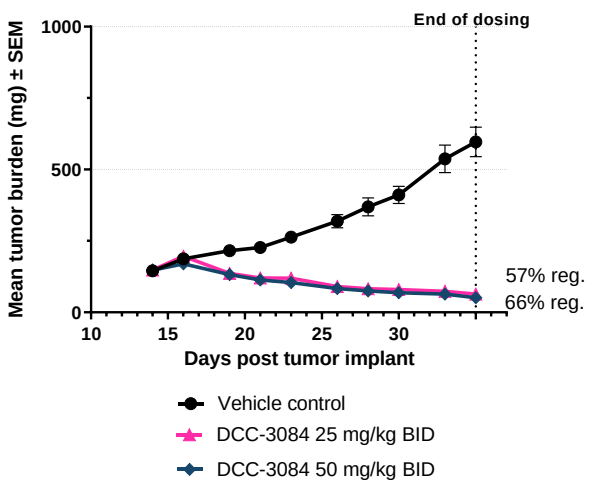
BRAF Fusion

WM3928: SKAP2-BRAF Fusion Melanoma Model



BRAF Class III

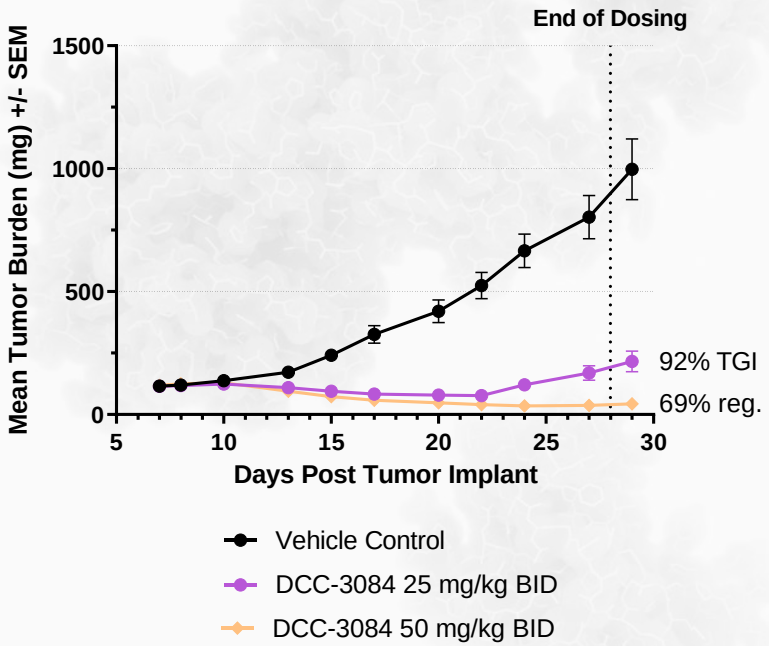
WM3629: BRAF plus NRAS G12D Melanoma Model



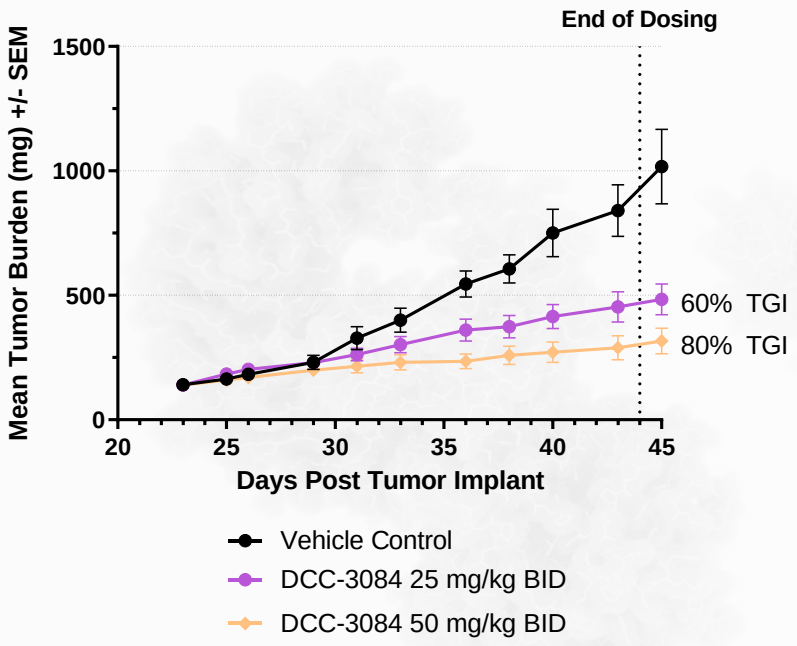
Notes: Data presented at the AACR Annual Meeting 2023; BID=twice daily; BRAF=proto-oncogene b-RAF; NRAS=neuroblastoma RAS viral oncogene homolog.

DCC-3084 PRODUCES SINGLE AGENT TUMOR REGRESSION OR TUMOR GROWTH INHIBITION IN MUTANT RAS MODELS DRIVEN BY BRAF/CRAF

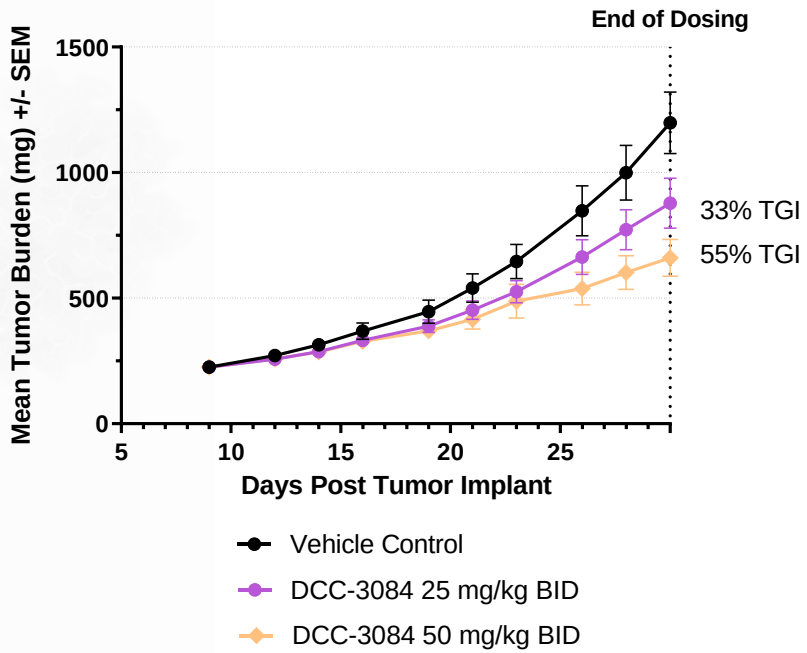
Calu-6: KRAS Q61K Lung Cancer



H358: KRAS G12C Lung Cancer



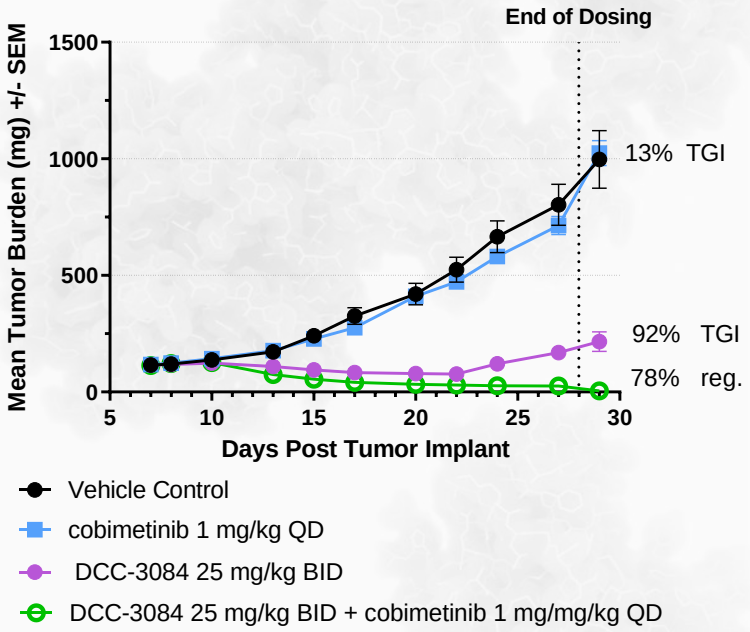
HPAF-II: KRAS G12D Pancreatic Cancer



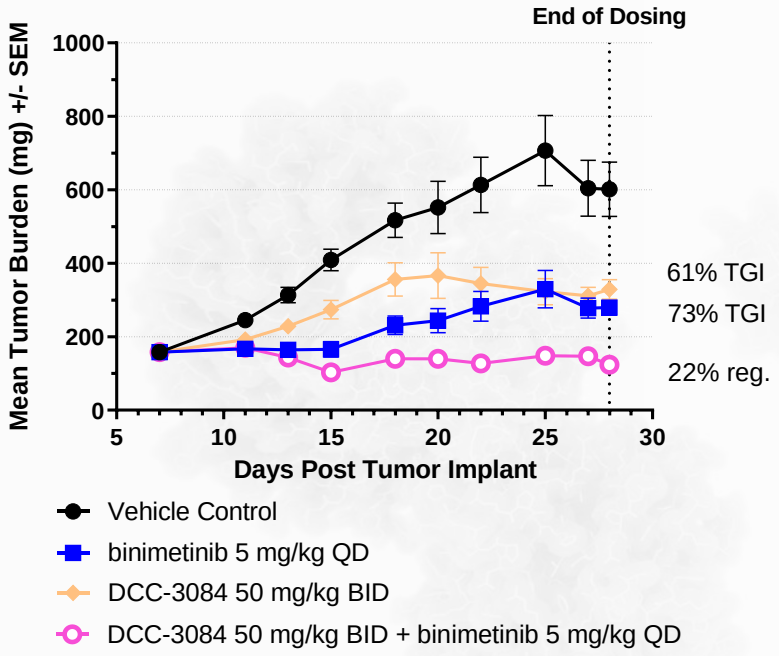
Notes: Data presented at the AACR Annual Meeting 2023; BID=twice daily; BRAF=proto-oncogene b-RAF; CRAF=proto-oncogene c-RAF; KRAS=Kirsten rat sarcoma virus; RAS=rat sarcoma gene; Reg.=regression; TGI=tumor growth inhibition.

DCC-3084 PRODUCES DEEPER TUMOR REGRESSION IN KRAS MUTANT CANCER MODELS IN COMBINATION WITH MEK INHIBITORS

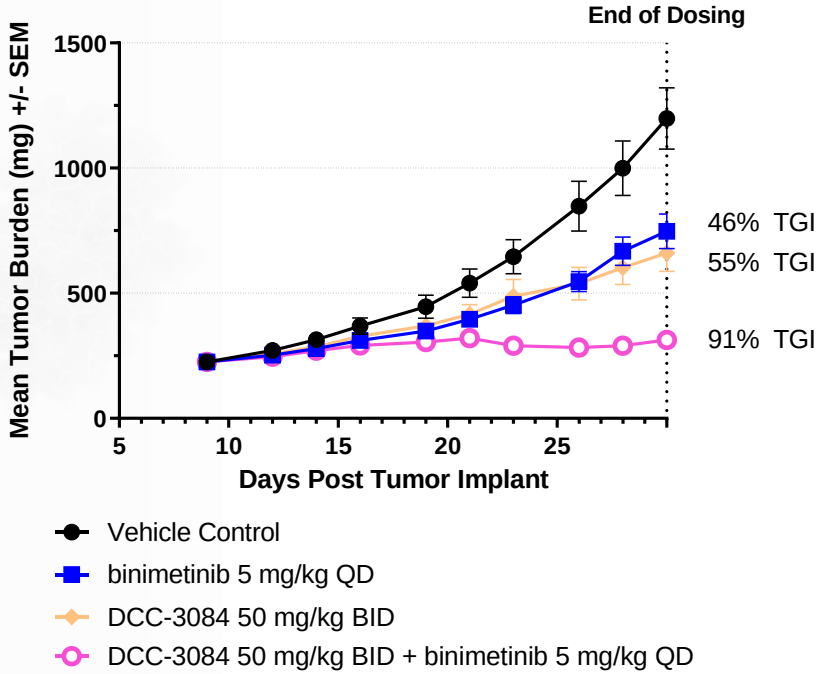
Calu-6: KRAS Q61K Lung Cancer



H358: KRAS G12C Lung Cancer



HPAF-II: KRAS G12D Pancreatic Cancer

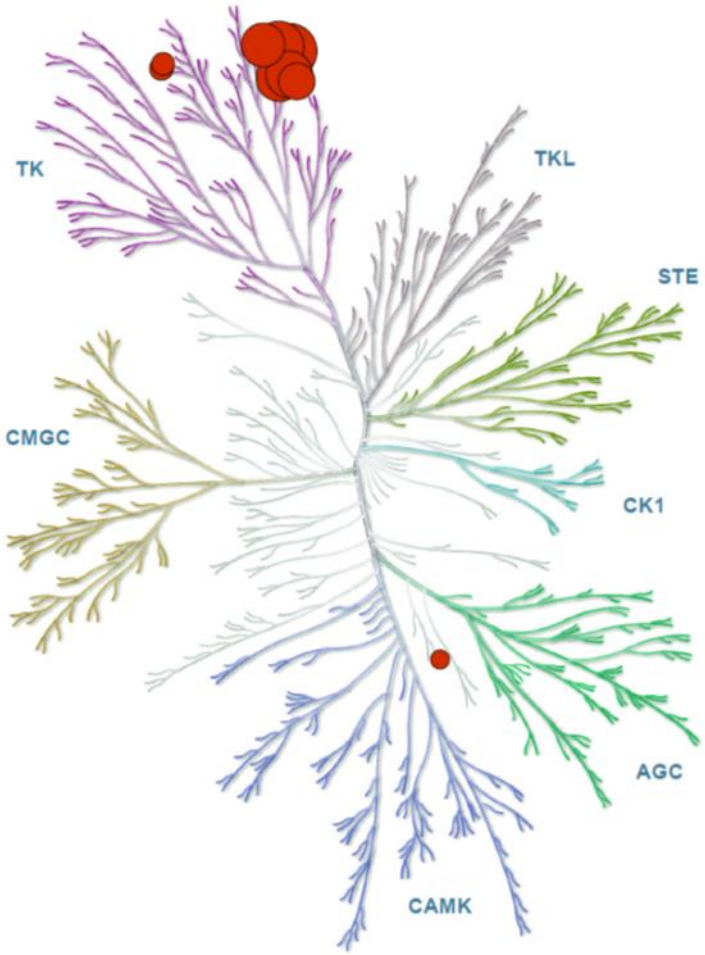


Notes: Data presented at the AACR Annual Meeting 2023; BID=twice daily; KRAS=Kirsten rat sarcoma virus; MEK=mitogen-activated extracellular signal-regulated kinase; QD=once daily; Reg.=regression; TGI=tumor growth inhibition.

DCC-3009

decīphera

DCC-3009 IS A POTENT AND SELECTIVE NEXT-GEN KIT INHIBITOR

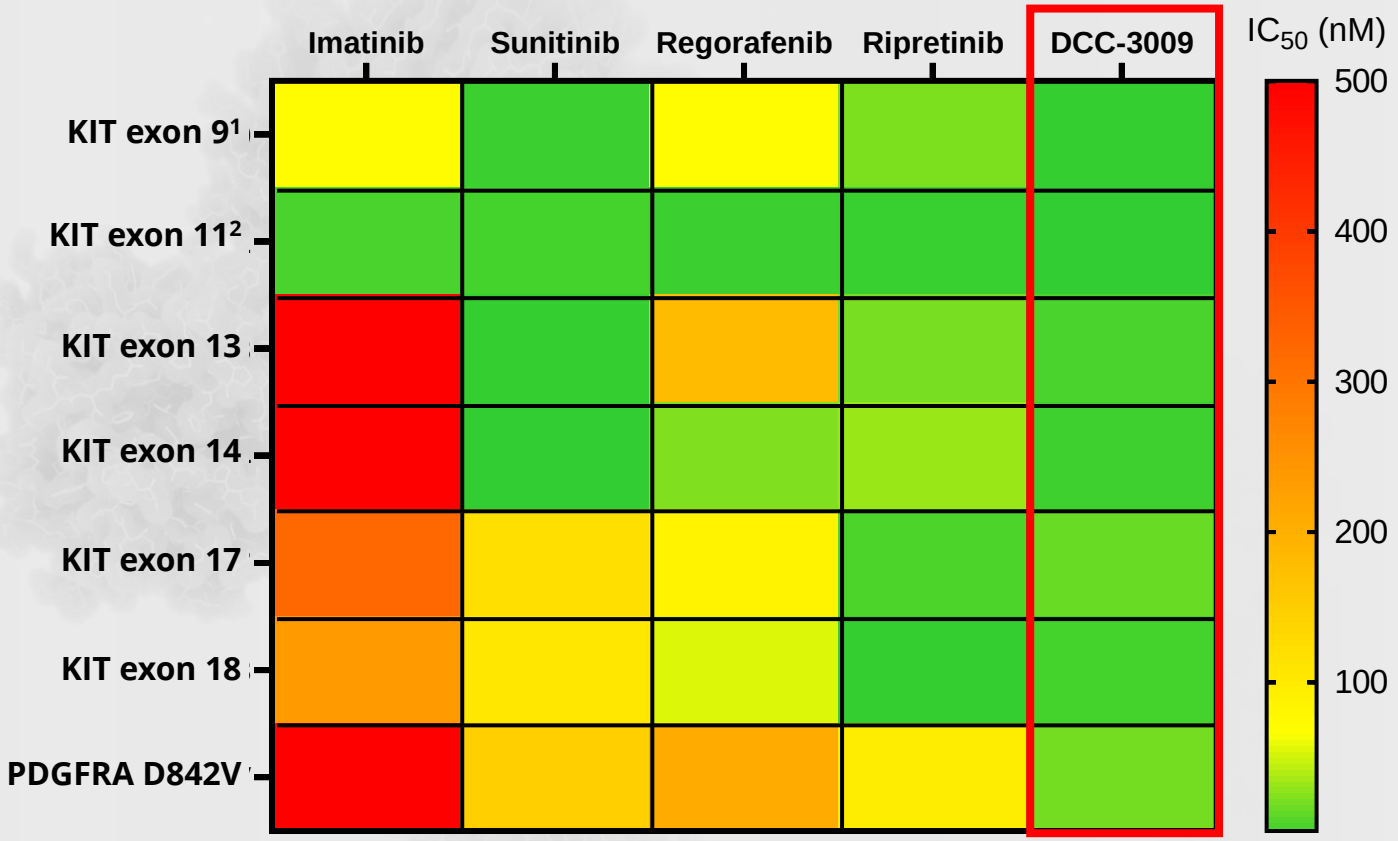


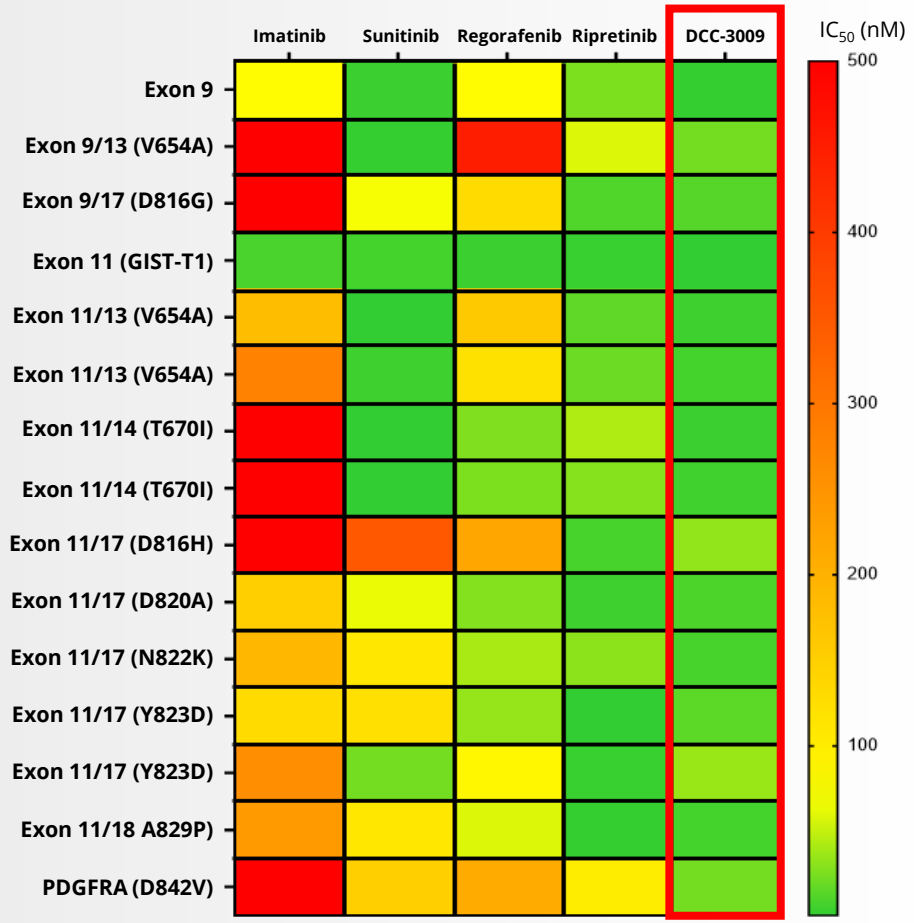
- DCC-3009 is a **potential best-in-class pan-KIT inhibitor** engineered using Deciphera's proprietary switch-control platform
- Unmet medical need remains for a **pan-KIT inhibitor** that can broadly and potently inhibit the **spectrum of KIT mutations** that drive GIST
- Potent inhibitor of primary KIT mutations in **exons 9 and 11 and secondary mutations across exons 13, 14, 17, and 18**
- Highly selective for KIT with **optimized pharmaceutical and ADME properties**
- **Strong pre-clinical efficacy data** in xenograft models driven by drug resistant KIT mutations

deciphera

Notes: ADME=absorption, distribution, metabolism, and excretion; GIST=gastrointestinal stromal tumor; KIT=KIT proto-oncogene receptor tyrosine kinase.

DCC-3009 POTENTLY INHIBITS THE SPECTRUM OF KNOWN PRIMARY AND SECONDARY DRUG-RESISTANT MUTATIONS IN GIST

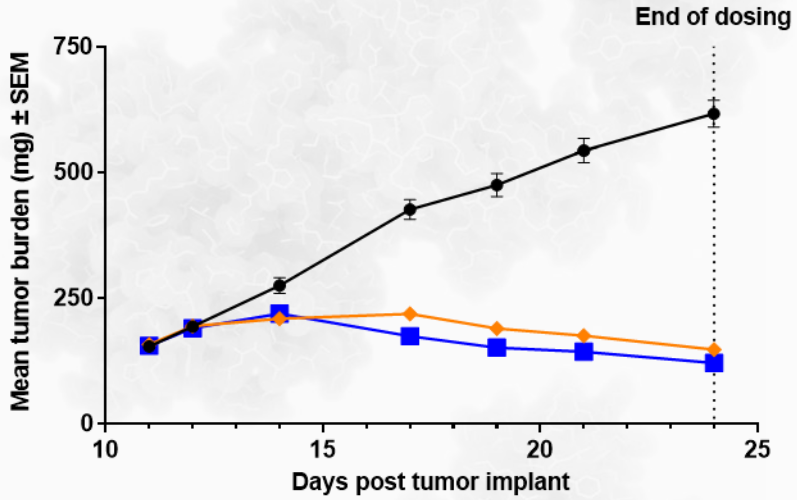




Notes: GIST=gastrointestinal stromal tumor; GIST-T1=exon 11 del; (1) exon 9 primary is A502/Y503 duplication; (2) exon 11 primary mutations include deletions or the V560D point mutation.

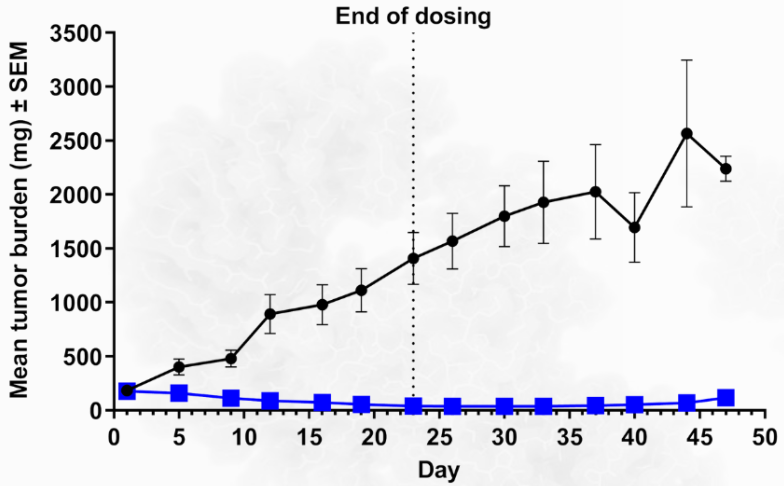
DCC-3009 RESULTED IN TUMOR REGRESSIONS IN GIST XENOGRAFT MODELS, INCLUDING KIT EXONS 9, 11, 13 AND 17 MUTATIONS

V654A: BaF3 KIT Exon 9 AY dup / Exon 13



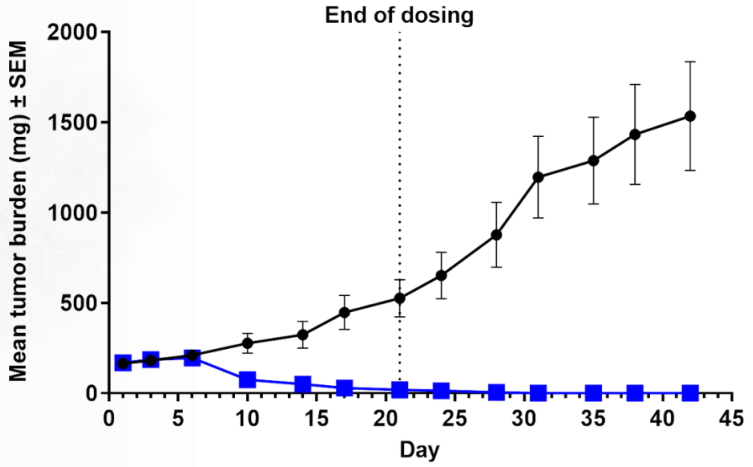
- Vehicle
- DCC-3009 50 mg/kg BID — 22% regression
- Sunitinib 20 mg/kg BID — 6% regression

V654A: GIST PDX KIT Exon 11 delWK / Exon 13



- Vehicle
- DCC-3009 50 mg/kg BID — 80% regression

Y823D: GIST PDX KIT Exon 11 delWK / Exon 17



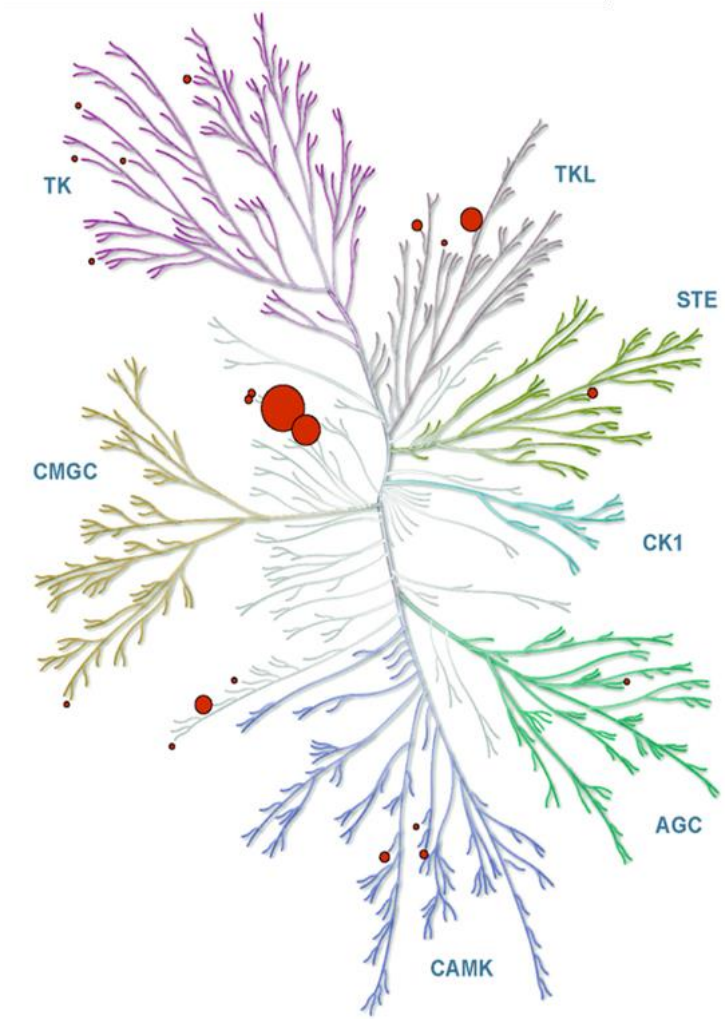
- Vehicle
- DCC-3009 50 mg/kg BID — 99.7% regression

Notes: Data presented at the AACR Annual Meeting 2023; BID=twice daily; GIST=gastrointestinal stromal tumor; KIT=KIT proto-oncogene receptor tyrosine kinase; PDX=patient-derived xenograft.

DP-9149 (GCN2 ACTIVATOR)

decīphera

DP-9149 IS A POTENT AND SELECTIVE ACTIVATOR OF THE GCN2 KINASE



Compelling Preclinical Data

- Potent and selective activator of GCN2 kinase
- Strong single agent activity in solid tumor models *in vivo*
- Tumor regressions in combination with standard of care agents *in vivo*

Novel Mechanism of Action

- Leveraging the cytotoxic arm of the Integrated Stress Response pathway enables the engagement of cancer cell death pathways
- GCN2 overexpression in solid tumors provides a favorable therapeutic window as evident by tolerability in preclinical models
- Synergizes with other stress-inducing therapies (anti-angiogenics/tumor driver-targeting agents) and effective in RAS/MAPK driven cancers

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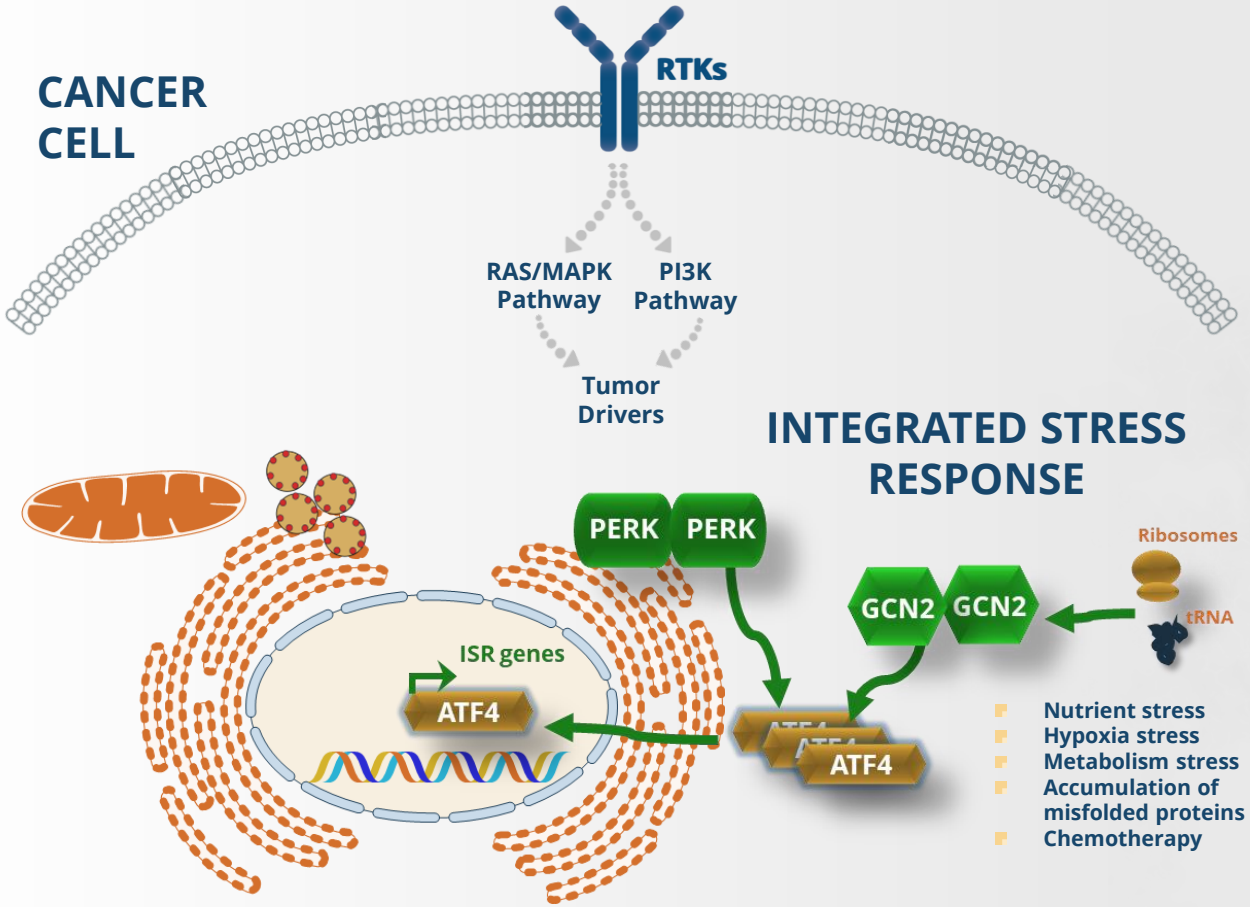
Notes: GCN2=general control nonderepressible 2; MAPK=mitogen-activated protein kinase; RAS=rat sarcoma gene.

THE INTEGRATED STRESS RESPONSE PATHWAY & GCN2 ACTIVATION



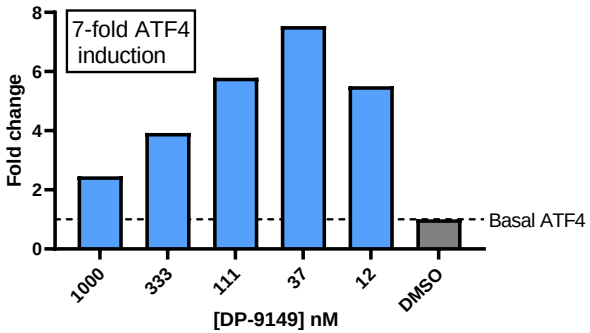
- The Integrated Stress Response (ISR) is a **major adaptive stress response pathway in cancer** and plays an important role in cell fate determination
- Oncogene addicted solid tumors are under high stress levels and are **dependent on a well-balanced ISR pathway** for accelerated growth
- Inhibition or stimulation of GCN2 in solid tumors can be pharmacologically leveraged to **induce anti-tumoral effects**
- Deciphera's GCN2 activator (DP-9149) has shown **anti-tumoral effects in solid tumors *in vitro* and *in vivo***

Notes: ATF4=activating transcription factor 4; GCN2=general control nonderepressible 2; ISR=Integrated Stress Response; MAPK=mitogen-activated protein kinase; PERK=protein kinase R–like endoplasmic reticulum kinas; PI3K=phosphatidylinositol-3 kinase; RAS=rat sarcoma gene; RTK=receptor tyrosine kinase.

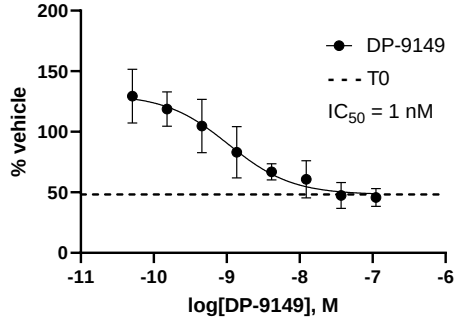
DP-9149 SELECTIVELY AND POTENTLY ACTIVATES GCN2 AND HAS AN OPTIMIZED PHARMACEUTICAL AND SELECTIVITY PROFILE

DP-9149 Upregulates the ISR Pathway and Potently Inhibits Cell Growth as a Single Agent

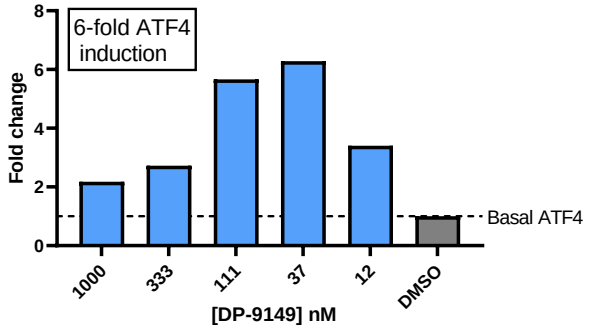
786-O: Renal Cell Carcinoma
ATF4 Upregulation



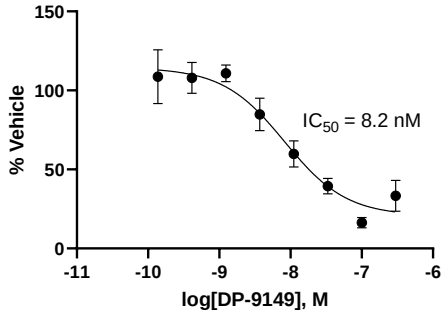
786-O: Renal Cell Carcinoma
Spheroid Growth Inhibition



LoVo: KRAS G13D Colorectal
ATF4 Upregulation



LoVo: KRAS G13D Colorectal
Spheroid Growth Inhibition



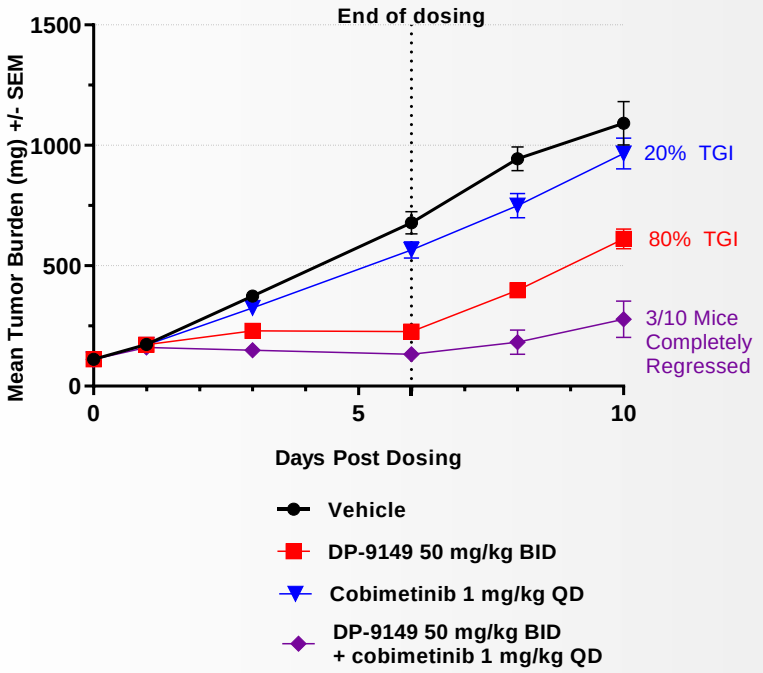
DP-9149 was Designed as a Potent and Selective Activator of GCN2

	Assay	DP-9149
Enzymatic Assays	GCN2 recombinant enzyme activation versus control	**2.5-fold activation**
Cellular Assays	ATF4 stimulation versus control 786-O (Renal; VHL-mut)	**7-fold activation**
	ATF4 Stimulation versus control LoVo (Colorectal; KRAS G13D)	**6-fold activation**
	Spheroid growth inhibition 786-O (Renal; VHL-mut)	**IC_{50} = 1 nM**
	Spheroid growth inhibition LoVo (Colorectal; KRAS G13D)	**IC_{50} = 8.2 nM**
Off-Target Profile	Kinome selectivity and safety (Cerep)	**High selectivity**
In Vivo	PK/PD	**Target engagement achieved**

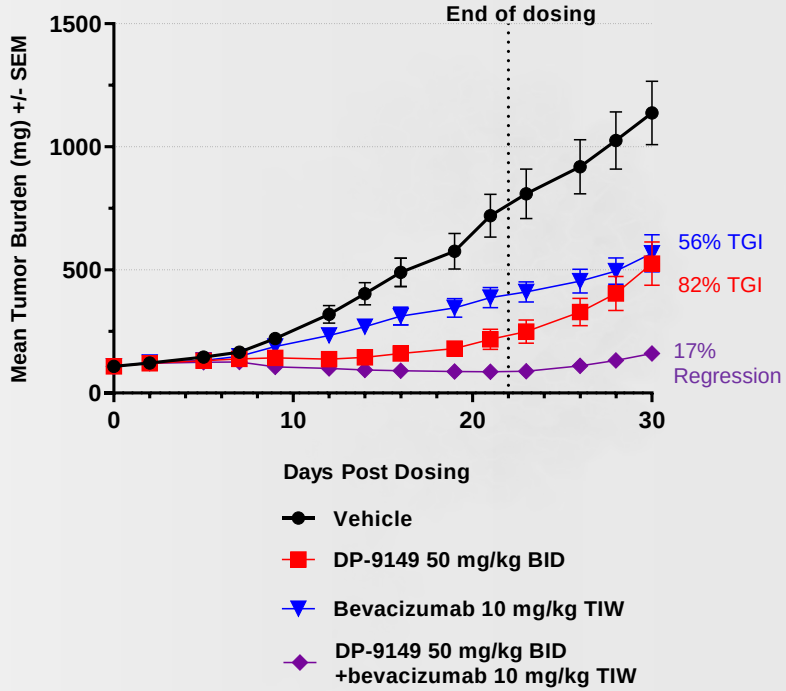
Notes: Data presented at the AACR Annual Meeting 2023; ATF4=activating transcription factor; DMSO=dimethyl sulfoxide; GCN2=general control nonderepressible 2; ISR=Integrated Stress Response; KRAS=Kirsten rat sarcoma virus; PD=pharmacodynamic; PK=pharmacokinetic; VHL=Von Hippel-Lindau.

DP-9149 RESULTS IN TUMOR GROWTH INHIBITION AS A SINGLE AGENT AND TUMOR REGRESSIONS IN COMBINATION *IN VIVO*

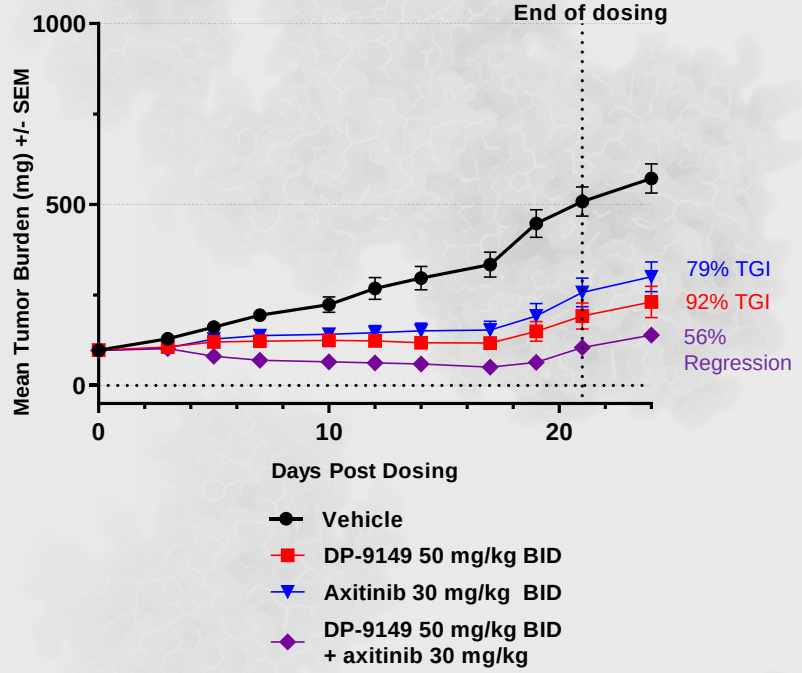
HT-1080: NRAS Fibrosarcoma Model
DP-9149 + Cobimetinib



LoVo: KRAS G13D CRC Model
DP-9149 + Bevacizumab



786-O: VHL Mutant RCC Model
DP-9149 + Axitinib



Notes: Data presented at the AACR Annual Meeting 2023; HT-1080 TGI was calculated on Day 6; LoVo TGI was calculated on Day 21; 786-8 TGI was calculated on Day 19; BID=twice daily; CRC=colorectal cancer; GCN2=general control nonderepressible 2; QD=once a day; BID= twice a day; RCC=renal cell carcinoma; TGI=tumor growth inhibition; TIW=3 times a week.

DECIPHERA

EXPECTED 2023 MILESTONES

QINLOCK®

- ✓ Present additional data from Phase 3 INTRIGUE ctDNA analysis at an ASCO Plenary Session
- Initiate INSIGHT Phase 3 study in 2L KIT exon 11+17/18 GIST patients **(2H 2023)**
- Continue geographic expansion with launches in key European markets **(2023)**

VIMSELTINIB

- ✓ Complete enrollment in the Phase 3 MOTION study
- Announce top-line results from MOTION study **(4Q 2023)**
- Present updated Phase 1/2 data in TGCT patients **(2H 2023)**

DCC-3116

- ✓ Present preclinical data on new combinations
- Present updated Phase 1 single agent and initial combination dose escalation data **(2H 2023)**
- Initiate MEK/G12C expansion cohort(s); initiate escalation cohort for encorafenib/cetuximab **(2H 2023)**

DCC-3084

- ✓ Present data on preclinical profile
- Submit IND to FDA **(2H 2023)**

PROPRIETARY DRUG DISCOVERY PLATFORM

- ✓ Nominate development candidate for pan-KIT inhibitor (DCC-3009 pan-KIT inhibitor)
- ✓ Present new preclinical data from research programs

deciphera®

Notes: 2L=second-line; ASCO=American Society of Clinical Oncology; ctDNA=circulating tumor deoxyribonucleic acid; FDA=U.S. Food and Drug Administration; G12C=single point mutation with a glycine-to-cysteine substitution at codon 12; GIST=gastrointestinal stromal tumor; IND=Investigational New Drug Application; KIT=KIT proto-oncogene receptor tyrosine kinase; MEK=mitogen-activated extracellular signal-regulated kinase; TGCT=tenosynovial giant cell tumor.

DECIPHERA

FINANCIAL HIGHLIGHTS

As of March 31, 2023

Weighted-Average Shares Outstanding[1]

82.7MM

Weighted average shares includes outstanding common stock and common stock issuable upon exercise of prefunded warrants

Cash, Cash Equivalents & Marketable Securities

$426.3MM

Cash Expected to Fund Operating Expenses and CapEx into 2026[2]

Notes: (1) As of March 31, 2023, there were 78.5MM outstanding shares, 6.4MM prefunded warrants, and 9.3MM options outstanding; (2) cash guidance based on current operating plan including proceeds from January 2023 public offering.

THANK YOU