One Mission, Inspired by Patients: Defeat Cancer.™

September 11, 2022

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OPENING REMARKS

Steve Hoerter

President and Chief Executive Officer

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This presentation may contain forward-looking statements that are based on our current expectations, estimates and projections about our industry 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. These statements include, without limitation, the commercialization of QINLOCK (ripretinib) for fourth-line GIST patients in the U.S., key European markets, and any other jurisdictions where we may receive marketing approval in the future, our expectations and timing regarding vimseltinib and the pivotal Phase 3 MOTION study in TGCT patients and the potential for vimseltinib to be a best-in-class treatment for TGCT, our Phase 1 study of DCC-3116 in patients with mutant RAS or RAF cancers, initial data from the dose escalation phase of the Phase 1 study of DCC-3116 and the potential for DCC-3116 to be a first-in-class ULK inhibitor, our expectations and timing for declaring a development candidate for our pan-RAF program, continuing to develop our in-licensed research stage VPS34 program, ex-U.S. strategies including in Europe (including developments, without limitation, in key markets such as Germany and France) and other geographies, 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 COVID-19, and 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 corporate strategy and restructuring plan, the success of our commercialization efforts with respect to QINLOCK, including our launch in Germany, 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 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; the ongoing effects of the COVID-19 pandemic, 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 Quarterly Report on Form 10-Q for the quarter ended June 30, 2022 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™

SUCCESSFUL COMMERCIAL FRANCHISE AND RESEARCH ENGINE TO FUEL PIPELINE

QINLOCK®

- Building on successful U.S. launch
- Now approved in nine territories, including the U.S., E.U., and China
- Launch in Germany underway

Leader in Autophagy

- DCC-3116, potential first-in-class ULK inhibitor for cancer in Phase 1
- Significant potential combination opportunity in 70% of cancers

Vimseltinib

- Potential best-in-class product profile
- Estimated $850M market opportunity in the U.S. for TGCT
- Phase 3 MOTION study enrolling

Pan-RAF Program

- Program targeting inhibition of BRAF and CRAF kinases
- Nomination of development candidate expected by 4Q 2022

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Notes: BRAF=proto-oncogene b-RAF; CRAF=proto-oncogene c-RAF; RAF=rapidly accelerated fibrosarcoma; TGCT=tenosynovial giant cell tumor; ULK=unc-51-like autophagy-activating kinase.

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ESMO CONGRESS 2022 INVESTOR EVENT AGENDA

Opening Remarks	**Steve Hoerter** *President and Chief Executive Officer*
Autophagy as a Resistance Mechanism In Cancer	**Matt Sherman, M.D.** *Executive Vice President and Chief Medical Officer*
Initial Results of Phase 1 DCC-3116 Monotherapy	**Anthony Tolcher, M.D., FRCPC** *CEO, Founder, and Director of Clinical Research at NEXT Oncology*
Phase 1 Combination Study Of DCC-3116	**Matt Sherman, M.D.** *Executive Vice President and Chief Medical Officer*
DCC-3116 Q&A	
Unmet Medical Need in TGCT and Phase 1 Update	**Matt Sherman, M.D.** *Executive Vice President and Chief Medical Officer*
Vimseltinib Phase 2 Update in TGCT	**Jean-Yves Blay, M.D., Ph.D.** *General Director of the Centre Léon Bérard Lyon*
Vimseltinib Phase 3 MOTION Study	**Matt Sherman, M.D.** *Executive Vice President and Chief Medical Officer*
TGCT Market Opportunity	**Dan Martin** *Senior Vice President and Chief Commercial Officer*
Vimseltinib Q&A	
Closing Remarks	**Steve Hoerter** *President and Chief Executive Officer*

AUTOPHAGY AS A RESISTANCE MECHANISM IN CANCER

Matt Sherman, M.D.

Executive Vice President and Chief Medical Officer

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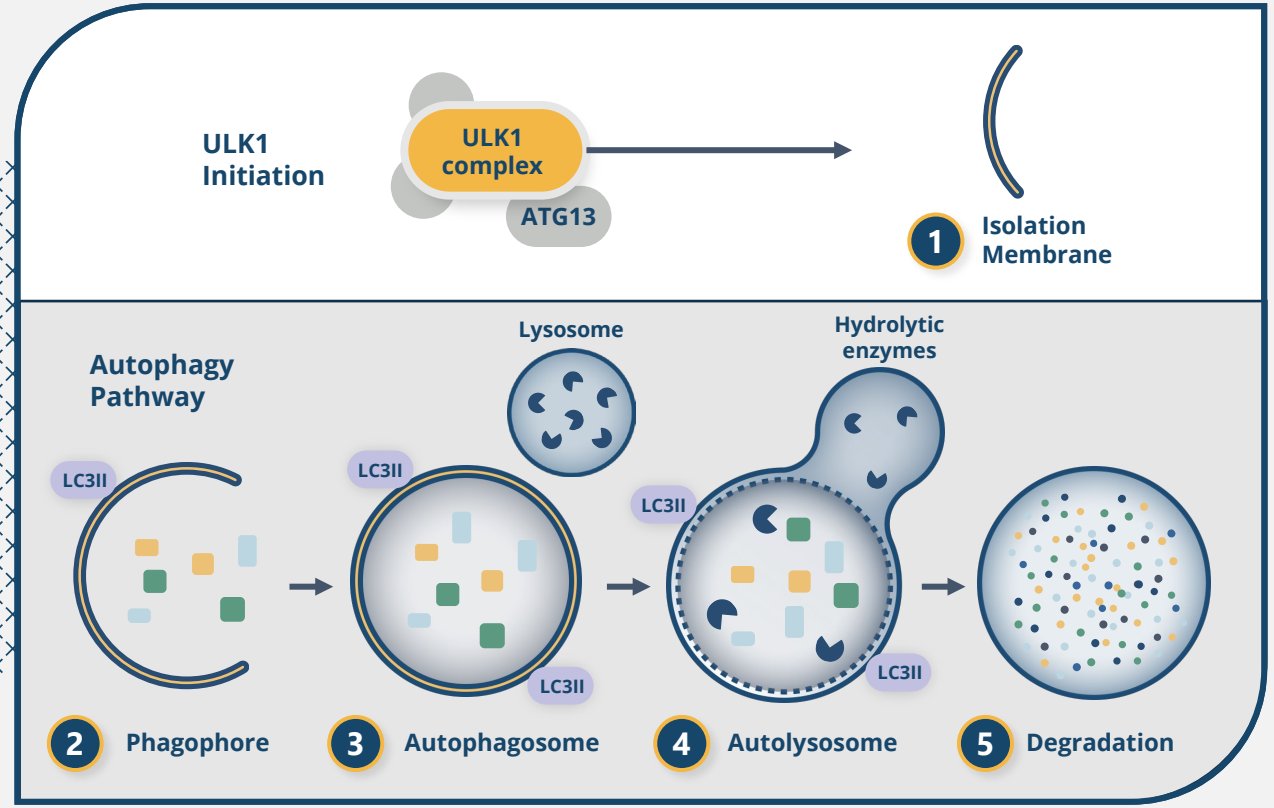
AUTOPHAGY: A BROAD RESISTANCE MECHANISM IN CANCER

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
- **DCC-3116 is a potential first-in-class small molecule** designed to inhibit cancer autophagy by inhibiting ULK kinase

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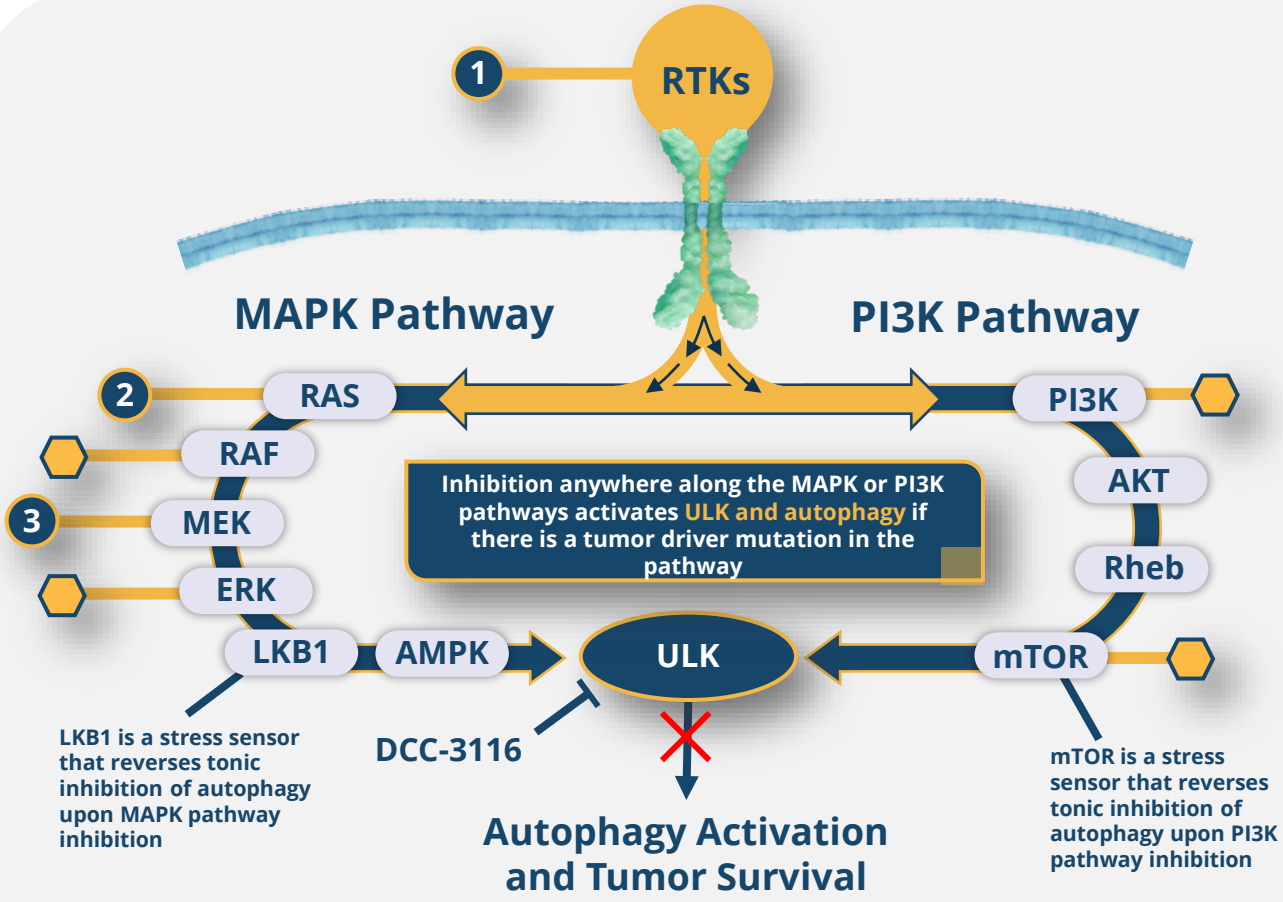
Notes: G12C=single point mutation with a glycine-to-cysteine substitution at codon 12; MAPK=mitogen-activated protein kinase; PI3K=phosphatidylinositol-3 kinase; RAF=rapidly accelerated fibrosarcoma; RAS=rat sarcoma gene; RTK=receptor tyrosine kinase; ULK=unc-51-like kinase; ATG13= Autophagy-related 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 osimertinib and afatinib resulting in tumor regression in EGFR-mutant NSCLC *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 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; 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.

POTENT AND SELECTIVE ULK INHIBITOR DESIGNED TO INHIBIT AUTOPHAGY

First-in-Class Switch-Control ULK Kinase Inhibitor

- Potent and selective inhibitor of ULK, the initiating factor in autophagy
- Strong preclinical data in combination with RTK/RAS/MAPK inhibitors
- Potential backbone combination agent across >70% of cancers

Highly Potent (Cellular IC_{50} values for ULK inhibition)

- ULK1 6 nM
- ULK2 9 nM

Highly Selective

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

Designed to Avoid CNS Exposure

- Low ratio $brain_{ff}/plasma_{ff}$ (4.3%) to avoid CNS autophagy

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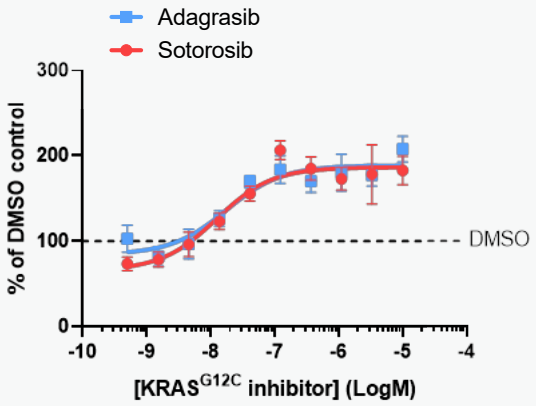
Notes: MAPK=mitogen-activated protein kinase; RAS=rat sarcoma gene; RTK=receptor tyrosine kinase; ULK=unc-51-like kinase.

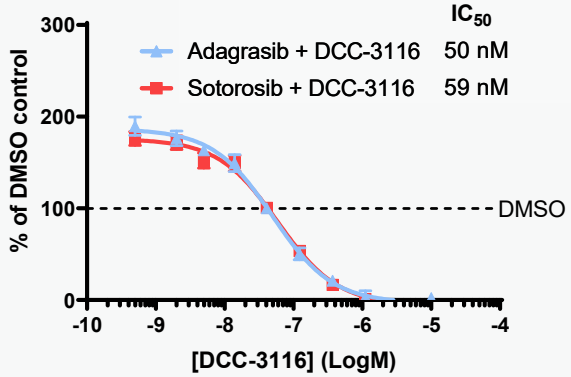
DCC-3116 INHIBITS RAS PATHWAY INHIBITOR-INDUCED ULK ACTIVITY

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

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

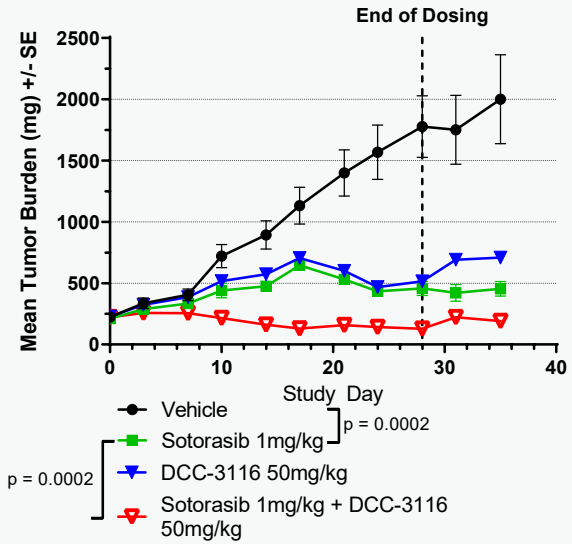


NSCLC: H358 pATG13 ELISA
DCC-3116 Inhibits KRAS^{G12C} Inhibitor-Induced ULK Activity



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

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



NSCLC: H358 Tumor Growth
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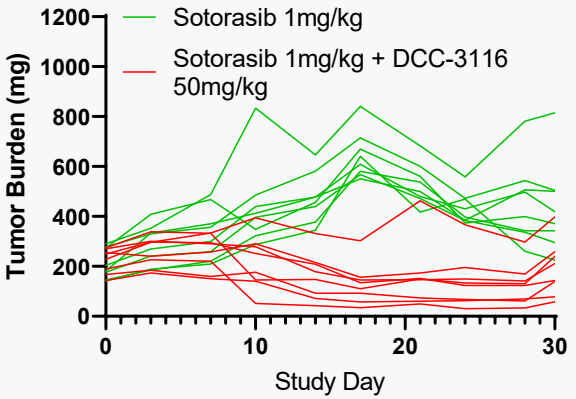

Notes: Data presented at AACR 2022; ATG13= Autophagy Related 13; ATG14=Autophagy Related 14: BID=twice daily; MEK=MAPK/ERK kinase; pATG13=phospho-ATG13; pATG14=phospho-ATG14; PBMCs=peripheral blood mononuclear cells; PD=pharmacodynamics; PK=pharmacokinetics; ULK=unc-51-like autophagy-activating kinase.

INITIAL RESULTS OF PHASE 1 DCC-3116 MONOTHERAPY

Anthony Tolcher, M.D., FRCPC

CEO, Founder, and Director of Clinical Research at NEXT Oncology

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PARTICIPANT DEMOGRAPHICS AND CLINICAL CHARACTERISTICS

	DCC-3116 Monotherapy Cohorts				All Participants n=18
	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	
Cancer type					
Colorectal	1 (33%)	2 (50%)	3 (43%)	4 (100%)	10 (56%)
Pancreas	1 (33%)	1 (25%)	3 (43%)	0	5 (28%)
Other[1]	1 (33%)	1 (25%)	1 (14%)	0	3 (17%)
Mutation type					
KRAS	2 (67%)	3 (75%)	6 (86%)	4 (100%)	15 (83%)
BRAF	1 (33%)	1 (25%)	1 (14%)	0	3 (17%)
Number of prior anticancer regimens					
Median (range)	2 (2–2)	3 (2–4)	3 (1–10)	3 (2–4)	3 (1–10)
1	0	0	1 (14%)	0	1 (6%)
2	3 (100%)	2 (50%)	1 (14%)	1 (25%)	7 (39%)
3	0	0	3 (43%)	2 (50%)	5 (28%)
≥4	0	2 (50%)	2 (29%)	1 (25%)	5 (28%)

Inclusion Criteria

- Histologically confirmed diagnosis of an advanced or metastatic solid tumor with a documented RAS, NF1, or RAF mutation

Primary Objectives

- Safety and tolerability
- Select DCC-3116 starting dose for combination with trametinib, binimetinib, and sotorasib escalation cohorts

Additional Objectives

- Antitumour activity per RECIST v1.1
- Pharmacokinetics
- Pharmacodynamics

Notes: Data presented at the ESMO Congress 2022; data cutoff: June 9, 2022. Data presented as n (%) unless otherwise indicated; BID, twice daily; BRAF, v-Raf murine sarcoma viral oncogene homolog B1; KRAS, Kirsten rat sarcoma small GTPase protein; SD, standard deviation; (1) Includes parathyroid, intrahepatic bile duct cancer, and thyroid cancer.

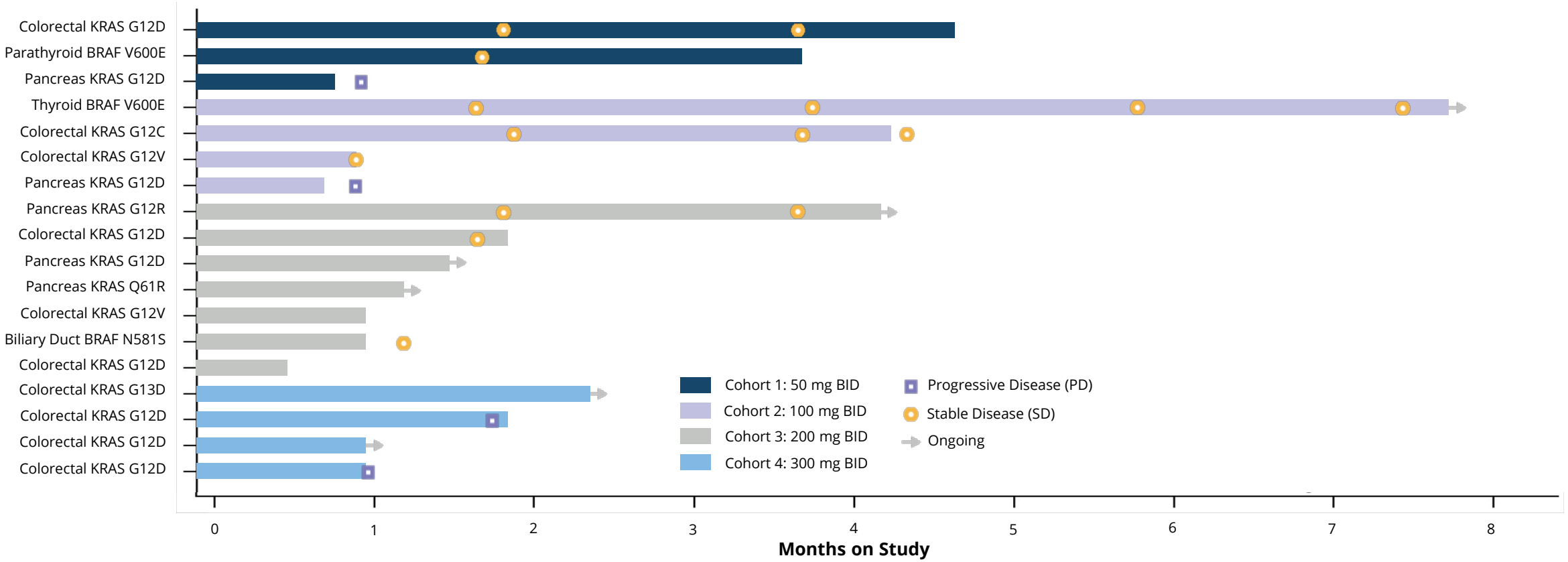
TEAEs REGARDLESS OF RELATEDNESS (≥15% OF PARTICIPANTS)

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



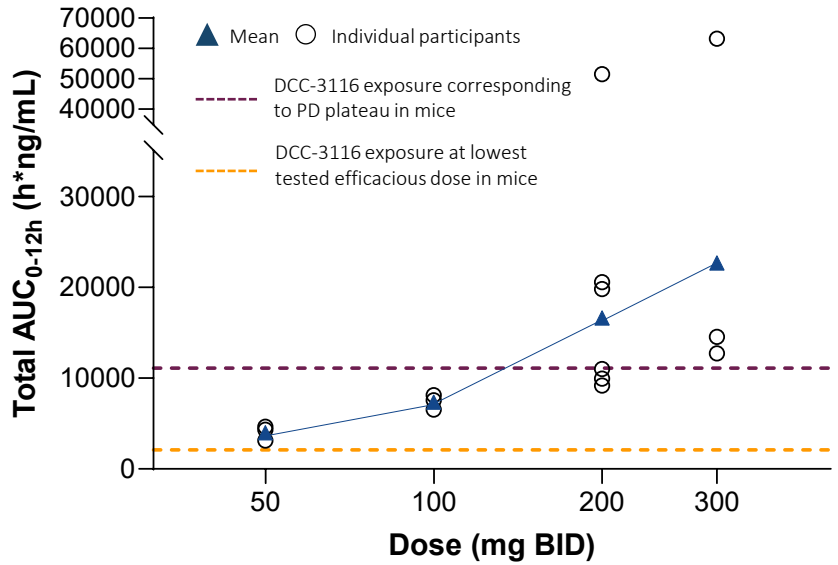
- Best overall response was stable disease
- Disease control rate at week 16 was 29% (4 of 14 response-evaluable participants)

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 EFFICACY BASED ON PRECLINICAL STUDIES

Pharmacokinetics

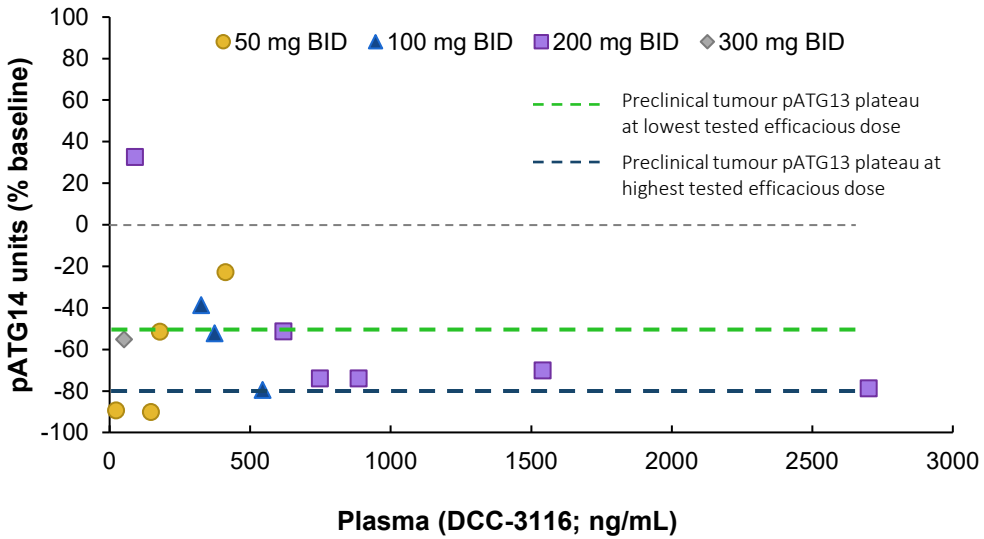
Total Individual and Mean $AUC_{0–12h}$ vs. Dose



- 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

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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 STUDY OF DCC-3116

Matt Sherman, M.D.

Executive Vice President and Chief Medical Officer

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SUMMARY OF INTERIM 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
- Dose cohorts 100 to 300 mg BID are being expanded to further characterize safety, PK, and PD
- In 4Q 2022, we expect to select the starting dose of DCC-3116 for, and initiate, dose escalation in combination with MEK and $KRAS^{G12C}$ inhibitors

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

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

MULTICENTER, OPEN-LABEL, FIRST-IN-HUMAN STUDY AS A SINGLE AGENT AND IN COMBINATION TRAMETINIB, BINIMETINIB, OR SOTORASIB

Part 1

Dose Escalation Phase (3 + 3 design)

DCC-3116

Single agent dose escalation cohorts

RP2D or MTD

of DCC-3116 as a single agent

- **DCC-3116 + Trametinib** *(MEK inhibitor)*
- **DCC-3116 + Binimetinib** *(MEK inhibitor)*
- **DCC-3116 + Sotorasib** *(KRAS^{G12C} inhibitor)*

RP2D of the combinations

Dose Escalation Phase Inclusion Criteria

- Histologically confirmed diagnosis of an advanced or metastatic solid tumor with a documented RAS, NF1, or RAF mutation

Part 2

Dose Expansion Phase

DCC-3116 + Trametinib

- **2nd Line PDAC[1]** *(KRAS-driven)*
- **3rd–5th Line NSCLC[2]** *(RAF/RAS-driven)*
- **≥3rd Line CRC[2]** *(RAF/RAS-driven)*

DCC-3116 + Binimetinib

- **2nd–3rd Line Melanoma[3]** *(NRAS-driven)*

DCC-3116 + Sotorasib

- **2nd–4th Line NSCLC[5]** *(KRAS^{G12C}-driven)*

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) with a documented mutation in KRAS; (2) with a documented mutation in KRAS, NRAS, NF1, or BRAF; (3) with a documented mutation in NRAS; (5) with a documented mutation in KRAS^{G12C}.

DCC-3116 Q&A

ANTHONY TOLCHER, M.D., FRCPC

STEVE HOERTER

MATT SHERMAN, M.D.

TUCKER KELLY

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UNMET MEDICAL NEED IN TGCT AND PHASE 1 UPDATE

Matt Sherman, M.D.

Executive Vice President and Chief Medical Officer

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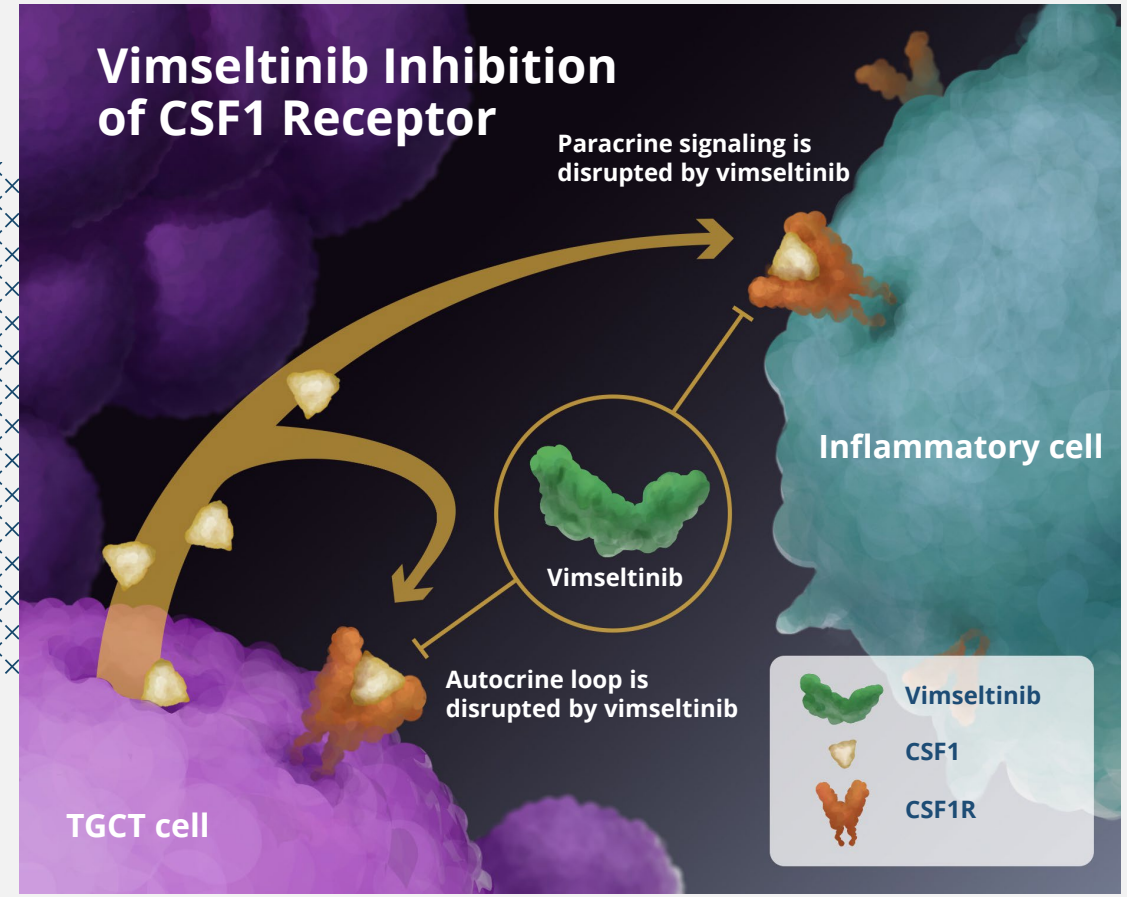
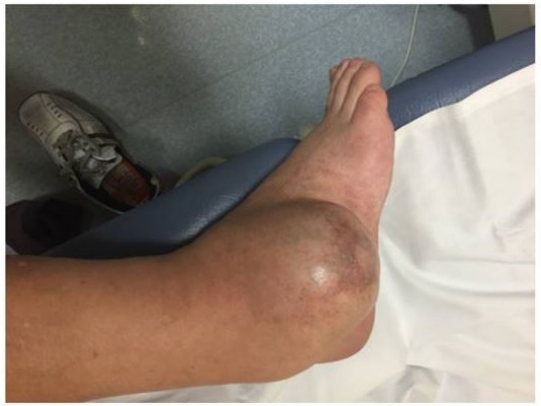
ENROLLMENT UNDERWAY FOR PHASE 3 MOTION STUDY IN TGCT

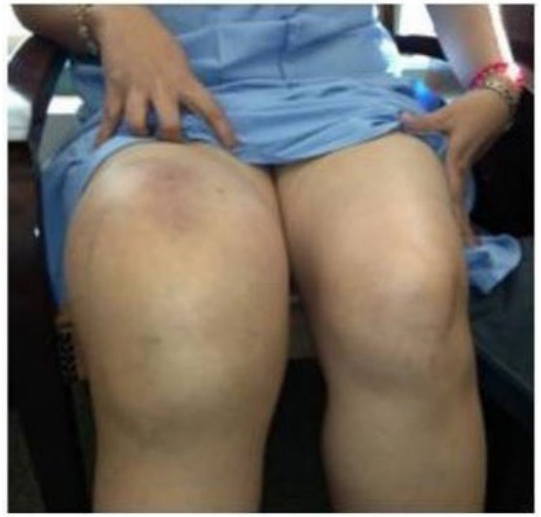


- Vimseltinib is an oral, switch-control TKI specifically designed to selectively and potently inhibit CSF1R
- TGCT caused by translocation in CSF1 gene, coding the ligand for CSF1R
- High unmet medical need in TGCT for effective therapy with improved safety profile
- Positive Phase 1/2 study updates provide strong support for ongoing Phase 3 MOTION study[1]
- Strong strategic fit with GIST based on overlapping KOLs and call-points

Notes: CSF1=colony-stimulating factor 1; CSF1R=colony-stimulating factor 1 receptor; GIST=gastrointestinal stromal tumor; KOL=key opinion leader; ORR=objective response rate; TGCT=tenosynovial giant cell tumor; TKI=tyrosine kinase inhibitor; (1) Data presented at the ESMO Congress 2022.

A LOCALLY AGGRESSIVE TUMOR ASSOCIATED WITH SUBSTANTIAL MORBIDITY





Disease Burden and Unmet Medical Need for TGCT Patients	
Diagnosis	Due to the slow-growing nature of TGCT and similar symptoms to other conditions like sports injuries and arthritis, patients may have a longer path to diagnosis
Patient burden	In the TOPP registry[1], patients at baseline commonly reported multiple symptoms, including pain (78%), limited function (64%), swelling (61%), stiffness (55%), and use of analgesics (15%)[2]

Unmet need	▪ Surgical resection is standard treatment
	▪ High rate of recurrence in diffuse TGCT
	▪ CSF1R inhibition has demonstrated clinical benefit in diffuse TGCT
	▪ Pexidartinib is only approved agent for TGCT patients no longer amenable to surgery (FDA approval in August 2019)
	  • FDA label includes boxed warning, REMS, and intensive liver monitoring due to off-target hepatotoxicity risks
	  • The EMA adopted the decision of refusal of the Turalio MAA in November 2020
	▪ Unmet medical need for effective CSF1R inhibitor with favorable safety/tolerability profile for TGCT patients

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Ankle image courtesy of Tap WD, et al. ASCO 2018, abstract 11502; Knee image courtesy of Tap WD, et al. N Engl J Med. 2015;373:428-437.
Notes: CSF1R=colony-stimulating factor 1 receptor; EMA=European Medicines Agency; FDA=U.S. Food and Drug Administration; MAA=Marketing Authorisation Application; REMS=Risk Evaluation and Mitigation Strategy; TGCT=tenosynovial giant cell tumor; (1) The TOPP registry was a multinational, multicenter, prospective observational study involving 12 tertiary sarcoma centers in 7 European countries, and 2 US sites; (2) Patients experienced more than or equal to 3 symptoms (52%).

STUDY DESIGN

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

PHASE 2 (n=58)

COHORT A (n=46)
TGCT patients with no prior anti-CSF1/CSF1R therapy (previous therapy with imatinib or nilotinib is allowed)

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.

SUMMARY OF LONG-TERM PHASE 1 RESULTS

- The Phase 1/2 study of vimseltinib is an open-label, multicenter study evaluating the safety, efficacy, pharmacokinetics, and pharmacodynamics of vimseltinib in patients with solid tumors and TGCT
- The Phase 1 data summary is based on the previously released abstract with a data cutoff date of February 18, 2022
- The Phase 1 poster presentation remains under embargo until September 12, 2022 and will include updated data based on a May 6, 2022 data cutoff date

OBJECTIVE RESPONSE RATE

69%

Across all dose cohorts of Phase 1

100%

CLINICAL BENEFIT RATE DEMONSTRATED ACROSS ALL PATIENTS[1]

16.4 months

MEDIAN TREATMENT DURATION

59% **ACTIVE PHASE 1 PATIENTS**

SAFETY AND TOLERABILITY CONSISTENT WITH PRIOR DISCLOSURES

Notes: Phase 1 results are based on the previously released abstract with a data cutoff date of February 18, 2022; results are reported for patients with TGCT with a data cutoff of Feb 18, 2022; TGCT= tenosynovial giant cell tumor; (1) complete response, partial response, and stable disease.

PHASE 2 UPDATE IN TGCT

Jean-Yves Blay, M.D., Ph.D.

General Director of the Centre Léon Bérard Lyon

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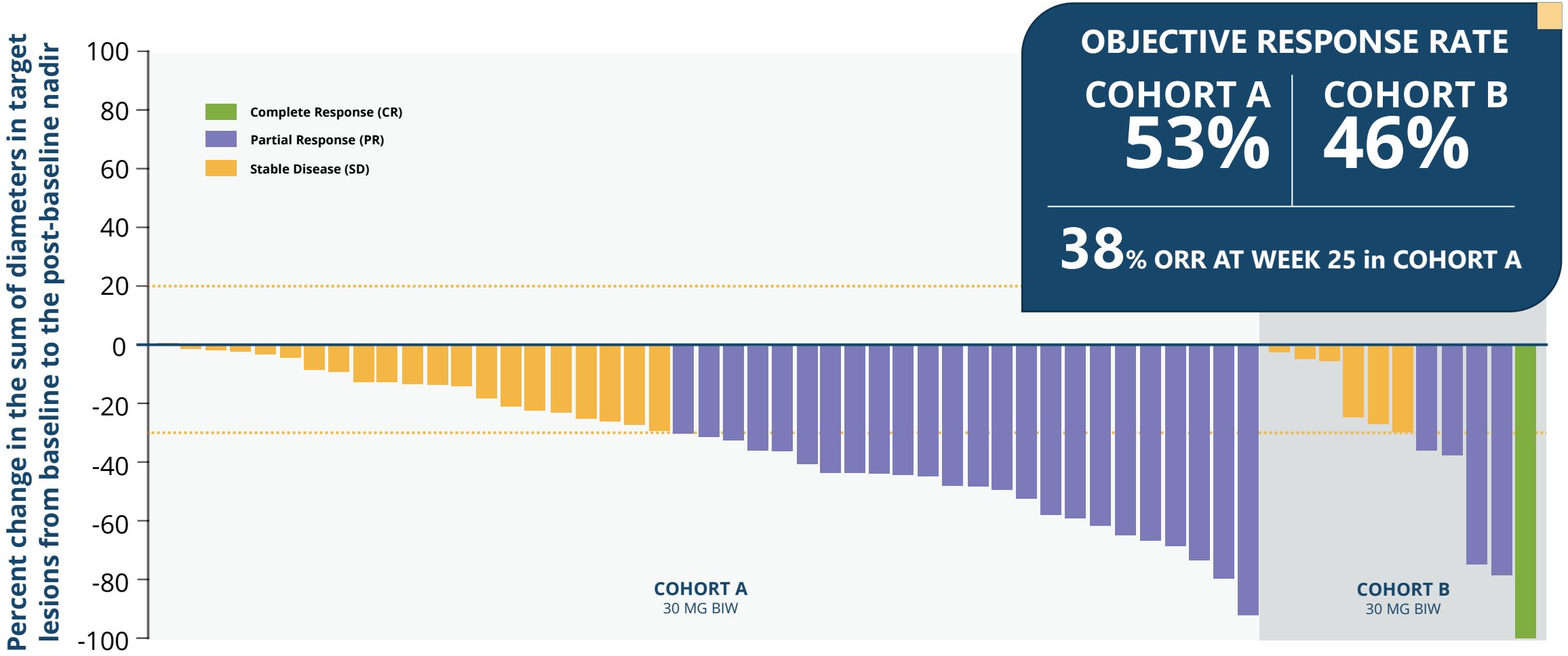
PHASE 2 BASELINE CHARACTERISTICS

	Cohort A (n=46)	Cohort B (n=12)
Median Age, years (range)	44 (21, 71)	47 (26, 65)
Sex		
Female	31 (67%)	7 (58%)
Male	15 (33%)	5 (42%)
Disease location		
Knee	26 (57%)	7 (58%)
Ankle	9 (20%)	1 (8%)
Foot	6 (13%)	0
Hand	0	1 (8%)
Other[1]	5 (11%)	3 (25%)
Patients with at least one prior surgery	31 (67%)	10 (83%)
Patients with at least one prior systemic therapy	3 (7%)	12 (100%)
Imatinib	3 (7%)	0
Pexidartinib	NA	7 (58%)
Imatinib and pexidartinib	NA	2 (17%)
Cabiralizumab and pexidartinib	NA	1 (8%)
Cabiralizumab	NA	1 (8%)
Vimseltinib	NA	1 (8%)

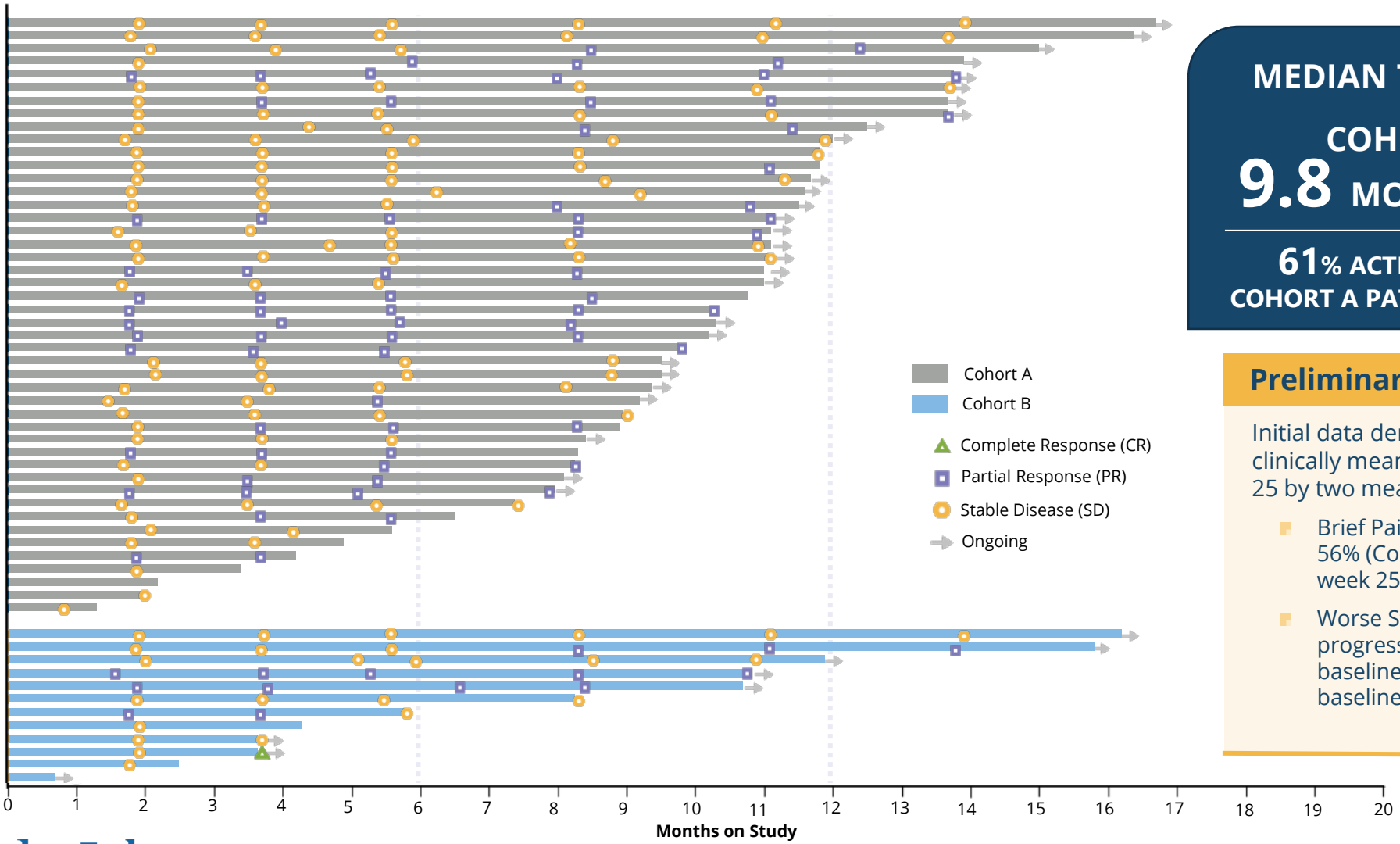
Notes: Data presented at the ESMO Congress 2022; Results are reported for patients with TGCT with a data cutoff of May 6, 2022; TGCT=tenosynovial giant cell tumor; Data are presented as n (%) unless otherwise noted; Percentages might not add up to 100% due to rounding; (1) Other locations include jaw, hip, shoulder, and thigh.

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



MEDIAN TREATMENT DURATION

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

Preliminary Patient-reported Outcomes

Initial data demonstrate that patients achieved clinically meaningful symptomatic benefit as of week 25 by two measures of patient-reported outcomes.

- Brief Pain Inventory (BPI): 48% (Cohort A) and 56% (Cohort B) of patients had a BPI response at week 25.
- Worse Stiffness NRS: Patients showed progressive improvements in stiffness from baseline to week 25, with mean changes from baseline of -2.0 (Cohort A) and -2.7 (Cohort B).

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Notes: Data presented at the ESMO Congress 2022; Results are reported for patients with TGCT with a data cutoff of May 6, 2022; BPI worst pain responder is defined as a patient who experiences a decrease of ≥30% in the mean BPI worst pain NRS item without experiencing a ≥30% increase in narcotic analgesic use; NRS=numeric rating scale.

WELL-TOLERATED IN TGCT PATIENTS

TEAEs in ≥15% of Patients Receiving Vimseltinib

Preferred term	Phase 2 Cohorts				Phase 2 All Patients	
	Cohort A (n = 46)		Cohort B (n = 12)		Total (N = 58)	
	All grades	Grade 3/4	All grades	Grade 3/4	All grades	Grade 3/4
Blood CPK increased	30 (65%)	20 (44%)	4 (33%)	2 (17%)	34 (59%)	22 (38%)
Headache	19 (41%)	0	8 (67%)	0	27 (47%)	0
Periorbital oedema	16 (35%)	0	6 (50%)	0	22 (38%)	0
Nausea	14 (30%)	0	5 (42%)	0	19 (33%)	0
Fatigue	9 (20%)	0	7 (58%)	0	16 (28%)	0
Asthenia	14 (30%)	1 (2%)	1 (8%)	0	15 (26%)	1 (2%)
Myalgia	13 (28%)	0	2 (17%)	0	15 (26%)	0
Arthralgia	10 (22%)	0	3 (25%)	1 (8%)	13 (22%)	1 (2%)
Rash maculopapular	10 (22%)	1 (2%)	3 (25%)	0	13 (22%)	1 (2%)
AST increased	8 (17%)	0	2 (17%)	0	10 (17%)	0
Face oedema	8 (17%)	0	2 (17%)	0	10 (17%)	0
Diarrhea	6 (13%)	0	3 (25%)	0	9 (16%)	0
Oedema peripheral	7 (15%)	0	2 (17%)	0	9 (16%)	0

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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; AST=aspartate aminotransferase; CPK=creatine phosphokinase; TEAE=treatment-emergent adverse event.

VIMSELTINIB
PHASE 3 MOTION STUDY

Matt Sherman, M.D.

Executive Vice President and Chief Medical Officer

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ENCOURAGING PHASE 1/2 RESULTS

- In patients with TGCT not amenable to surgical resection, vimseltinib demonstrated encouraging efficacy in all patients regardless of prior CSF1R therapy and at Week 25
- Vimseltinib was generally well-tolerated, with longer-term follow-up across all Phase 1/2 cohorts
- These results support further evaluation of vimseltinib in the MOTION study, a randomized, placebo-controlled, Phase 3 trial in patients with TGCT not amenable to surgical resection

OBJECTIVE RESPONSE RATE

PHASE 1 ALL COHORTS	PHASE 2 COHORT A	PHASE 2 COHORT B
69%	53%	46%

MEDIAN TREATMENT DURATION

PHASE 1 ALL COHORTS	PHASE 2 COHORT A	PHASE 2 COHORT B
16.4 months	9.8 months	5.9 months

59%	61%	67%
ACTIVE PHASE 1 PATIENTS	ACTIVE PHASE 2 COHORT A PATIENTS	ACTIVE PHASE 2 COHORT B PATIENTS

Preliminary PRO Data in Phase 2 Demonstrate Clinically Meaningful Improvements in Pain and Stiffness

Notes: Phase 1 results are based on the previously released abstract with a data cutoff date of February 18, 2022; Phase 2 results are based on the data presented at the ESMO Congress with a data cutoff of May 6, 2022; CSF1R=colony-stimulating factor 1 receptor; PRO=Patient-Reported Outcome; TGCT= tenosynovial giant cell tumor.

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

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
(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

Phase 3 Motion Study

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

Primary Endpoint:

- Objective response rate (ORR) at 25 weeks

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.

TGCT MARKET DYNAMICS AND VIMSELTINIB OPPORTUNITIES

Dan Martin

Senior Vice President and Chief Commercial Officer

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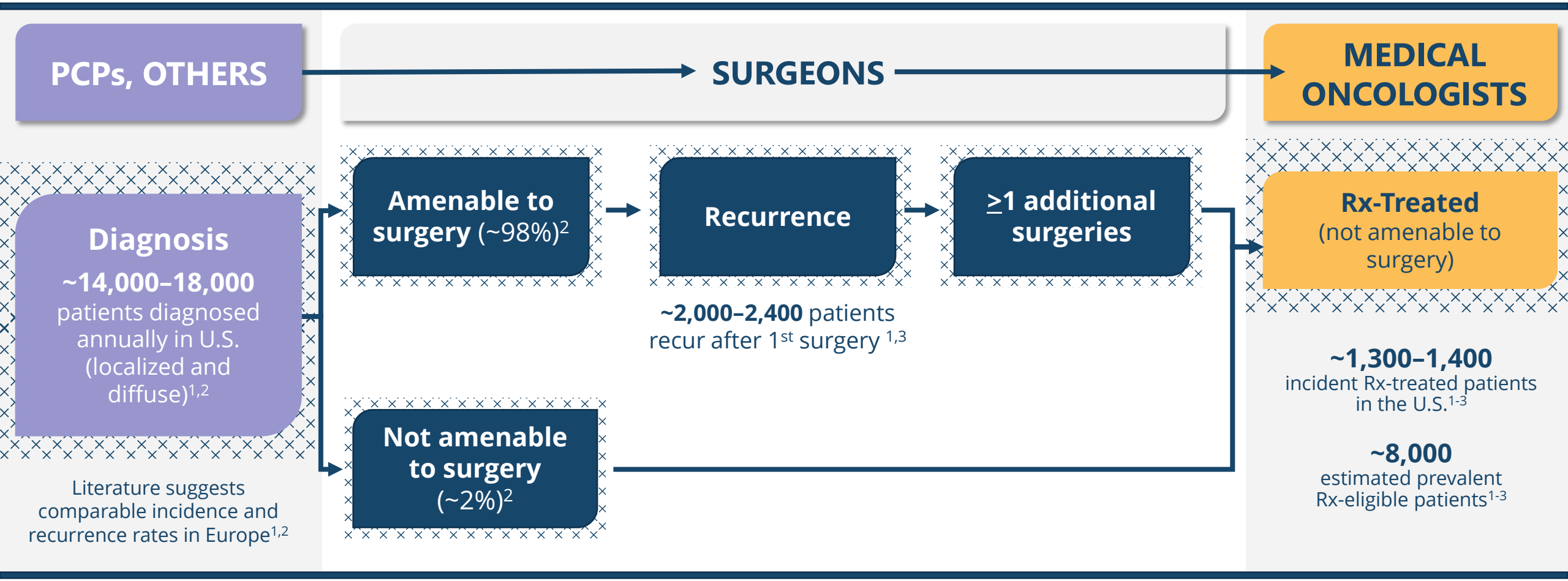
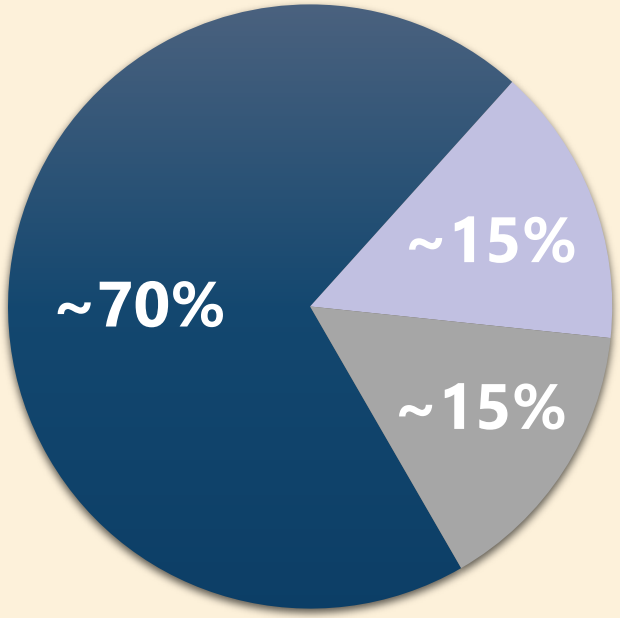
PATIENT JOURNEY OF TGCT PATIENTS NOT AMENABLE TO SURGERY



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

- FDA approved 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.

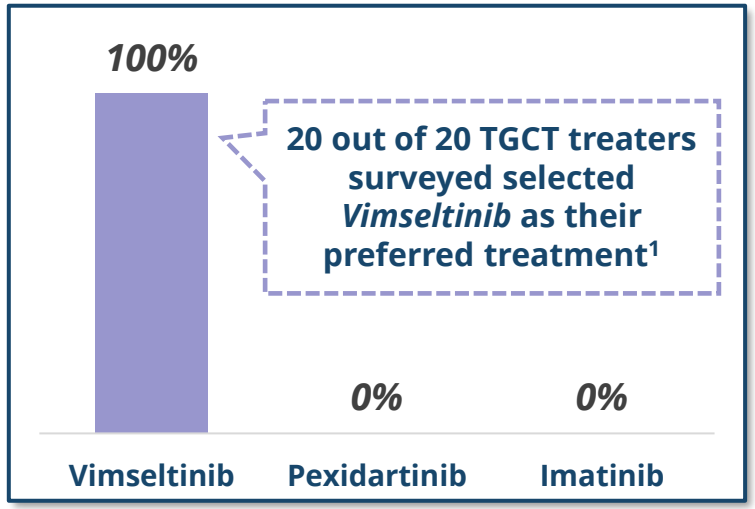
MARKET RESEARCH HIGHLIGHTS THE POTENTIAL FOR VIMSELTINIB TO DELIVER A BEST-IN-CLASS PROFILE AND ESTABLISH A NEW STANDARD OF CARE IN TGCT

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 The toxicity profile shows this is a very safe drug as well" – 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*

Safety: *"Black box warnings are usually at the top of the list of patient concerns. Not having one will be reassuring for them that this is safe to use in the short-term and the long-term." – Onc*

Treatment Choice: *"[Vimseltinib] is clearly superior to the other two products. It has better efficacy and safety data, which is key" – Onc*

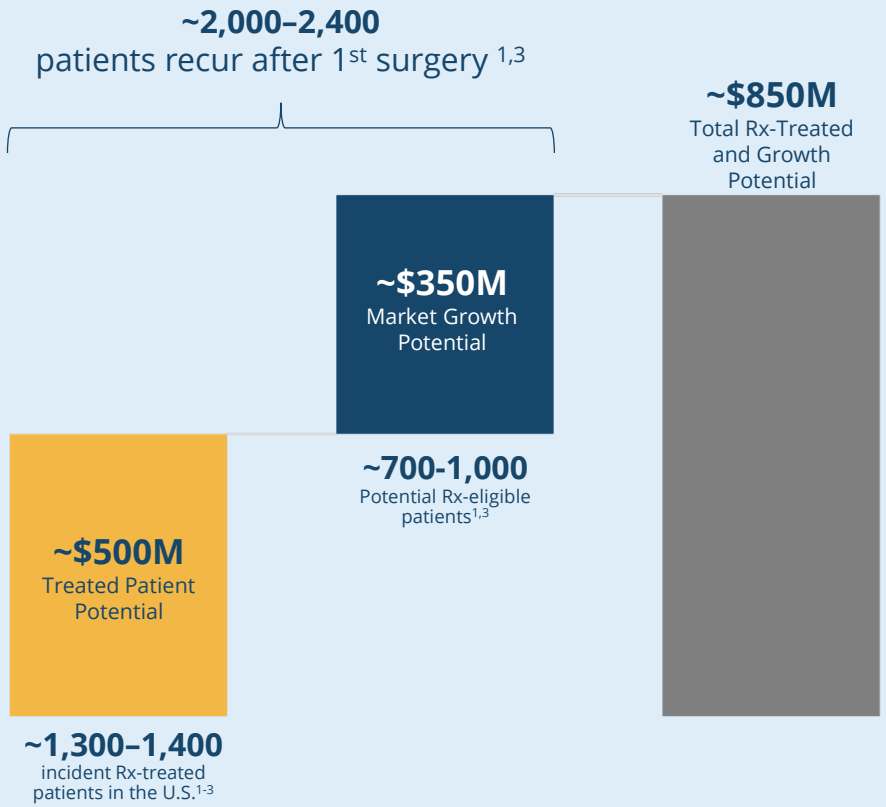
"I would give [vimseltinib] to all my future TGCT patients" - Onc

TGCT Treater Sentiments on Vimseltinib Profile

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. 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



+

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.

QINLOCK AND VIMSELTINIB OFFER THE POTENTIAL TO CREATE A SARCOMA FRANCHISE WITH SUBSTANTIAL OPERATIONAL SYNERGIES

~90% of TGCT prescribers are GIST prescribers already targeted by Deciphera[1]

- Ability to commercialize QINLOCK and vimseltinib with a single, sarcoma-focused field team
- Established relationships with sarcoma prescribers – DCPH sales force ranked highest by GIST treaters among all companies in GIST market[1]
- Existing commercial infrastructure can be leveraged at launch requiring only incremental investment
- Existing strong relationships with KOL and patient advocacy communities

Notes: DCPH=Deciphera; GIST=Gastrointestinal Stromal Tumor; TGCT=tenosynovial giant cell tumor; KOL=key opinion leader; (1) 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. Circles for TGCT and GIST prescribers are for illustrative purposes only.

VIMSELTINIB Q&A

JEAN-YVES BLAY, M.D., PH.D.

STEVE HOERTER

MATT SHERMAN, M.D.

DAN MARTIN

TUCKER KELLY

decīphera

CLOSING REMARKS

Steve Hoerter

President and Chief Executive Officer

decīphera®

THANK YOU