One Mission, Inspired by Patients: Defeat Cancer.™

September 11, 2022
OPENING REMARKS

Steve Hoerter
President and Chief Executive Officer
This presentation has been prepared by Deciphera Pharmaceuticals, Inc. for informational purposes only and not for any other purpose. Nothing contained in this presentation is, or should be construed as, a recommendation, promise or representation by Deciphera Pharmaceuticals, Inc. or any director, employee, agent, or adviser of Deciphera Pharmaceuticals, Inc. This presentation does not purport to be all-inclusive or to contain all of the information you may desire. Certain information contained in this presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and Deciphera Pharmaceuticals, Inc.’s own internal estimates and research. While Deciphera Pharmaceuticals, Inc. believes these third-party sources to be reliable as of the date of this presentation, it has not independently verified, and makes no representation as to the adequacy, fairness, accuracy or completeness of, any information obtained from third-party sources. While Deciphera Pharmaceuticals, Inc. believes its internal research is reliable, such research has not been verified by any independent source.

Forward-Looking Statements
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 (printinib) 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 UKI inhibitor, our expectations and timing for declaring a development candidate for our ran-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 may result from application of 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 completion 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 inaccuracy 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.

© 2022 Deciphera Pharmaceuticals. Deciphera®, the Deciphera logo, QINLOCK® and the QINLOCK logo are registered trademarks of Deciphera Pharmaceuticals, LLC. All rights reserved. This presentation may contain trade names, trademarks or service marks of other companies. Deciphera does not intend the use or display of other parties’ trade names, trademarks or service marks to imply a relationship with, or endorsement or sponsorship of, these other parties.
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.
DECIPHERA
ESMO CONGRESS 2022 INVESTOR EVENT AGENDA

Opening Remarks

Autophagy as a Resistance Mechanism In Cancer

Steve Hoerter
President and Chief Executive Officer

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
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.

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

**DCC-3116 | POTENTIAL BACKBONE OF COMBINATION THERAPY**

**GROWING PRECLINICAL VALIDATION FOR ROLE OF AUTOPHAGY IN CANCER**

**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

**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

**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.
DCC-3116 | OVERVIEW
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

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**

**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 Demonstrated Deeper and Longer Regressions in Combination with Sotorasib**

**NSCLC: H358 Tumor Growth**

- **Vehicle**
- **Sotorasib 1mg/kg**
- **Sotorasib 1mg/kg + DCC-3116 50mg/kg**

<table>
<thead>
<tr>
<th>Study Day</th>
<th>Tumor Burden (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>1000</td>
</tr>
<tr>
<td>10</td>
<td>800</td>
</tr>
<tr>
<td>20</td>
<td>600</td>
</tr>
<tr>
<td>30</td>
<td>400</td>
</tr>
<tr>
<td>40</td>
<td>200</td>
</tr>
</tbody>
</table>

**NSCLC: H358 pATG13 ELISA**

- **Adagrasib**
- **Sotorasib**
- **Adagrasib + DCC-3116**
- **Sotorasib + DCC-3116**

<table>
<thead>
<tr>
<th>[DCC-3116] (LogM)</th>
<th>% of DMSO control</th>
</tr>
</thead>
<tbody>
<tr>
<td>50 nM</td>
<td>100</td>
</tr>
</tbody>
</table>

**Adagrasib + DCC-3116 IC\(_{50}\) 59 nM**

**Sotorasib + DCC-3116 IC\(_{50}\) 50 nM**

**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
**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

---

### DCC-3116 Monotherapy Cohorts

<table>
<thead>
<tr>
<th>Cancer type</th>
<th>Cohort 1 (50 mg BID, n=3)</th>
<th>Cohort 2 (100 mg BID, n=4)</th>
<th>Cohort 3 (200 mg BID, n=7)</th>
<th>Cohort 4 (300 mg BID, n=4)</th>
<th>All Participants (n=18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colorectal</td>
<td>1 (33%)</td>
<td>2 (50%)</td>
<td>3 (43%)</td>
<td>4 (100%)</td>
<td>10 (56%)</td>
</tr>
<tr>
<td>Pancreas</td>
<td>1 (33%)</td>
<td>1 (25%)</td>
<td>3 (43%)</td>
<td>0</td>
<td>5 (28%)</td>
</tr>
<tr>
<td>Other¹</td>
<td>1 (33%)</td>
<td>1 (25%)</td>
<td>1 (14%)</td>
<td>0</td>
<td>3 (17%)</td>
</tr>
</tbody>
</table>

### Mutation type

<table>
<thead>
<tr>
<th>Mutation type</th>
<th>Cohort 1 (50 mg BID, n=3)</th>
<th>Cohort 2 (100 mg BID, n=4)</th>
<th>Cohort 3 (200 mg BID, n=7)</th>
<th>Cohort 4 (300 mg BID, n=4)</th>
<th>All Participants (n=18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>KRAS</td>
<td>2 (67%)</td>
<td>3 (75%)</td>
<td>6 (86%)</td>
<td>4 (100%)</td>
<td>15 (83%)</td>
</tr>
<tr>
<td>BRAF</td>
<td>1 (33%)</td>
<td>1 (25%)</td>
<td>1 (14%)</td>
<td>0</td>
<td>3 (17%)</td>
</tr>
</tbody>
</table>

### Number of prior anticancer regimens

<table>
<thead>
<tr>
<th>Number of regimens</th>
<th>Cohort 1 (50 mg BID, n=3)</th>
<th>Cohort 2 (100 mg BID, n=4)</th>
<th>Cohort 3 (200 mg BID, n=7)</th>
<th>Cohort 4 (300 mg BID, n=4)</th>
<th>All Participants (n=18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>0</td>
<td>1 (14%)</td>
<td>0</td>
<td>1 (6%)</td>
</tr>
<tr>
<td>2</td>
<td>3 (100%)</td>
<td>2 (50%)</td>
<td>1 (14%)</td>
<td>1 (25%)</td>
<td>7 (39%)</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>0</td>
<td>3 (43%)</td>
<td>2 (50%)</td>
<td>5 (28%)</td>
</tr>
<tr>
<td>≥4</td>
<td>0</td>
<td>2 (50%)</td>
<td>2 (29%)</td>
<td>1 (25%)</td>
<td>5 (28%)</td>
</tr>
</tbody>
</table>

---

**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.
### DCC-3116 | PHASE 1 STUDY

TEAEs REGARDLESS OF RELATEDNESS (≥15% OF PARTICIPANTS)

---

#### DCC-3116 Monotherapy Cohorts

<table>
<thead>
<tr>
<th>Preferred term</th>
<th>Cohort 1 50 mg BID (n = 3)</th>
<th>Cohort 2 100 mg BID (n = 4)</th>
<th>Cohort 3 200 mg BID (n = 7)</th>
<th>Cohort 4 300 mg BID (n = 4)</th>
<th>All Participants</th>
<th>n (%) N = 18</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fatigue</td>
<td>Grade 1/2: 2 0</td>
<td>Grade 1/2: 1 0</td>
<td>Grade 1/2: 3 0</td>
<td>Grade 1/2: 1 0</td>
<td>7 (39%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dehydration</td>
<td>Grade 1/2: 0 0</td>
<td>Grade 1/2: 0 0</td>
<td>Grade 1/2: 0 0</td>
<td>Grade 1/2: 0 1</td>
<td>4 (22%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td>Grade 3: 1</td>
<td>Grade 3: 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ALT increased</td>
<td>Grade 1/2: 0 0</td>
<td>Grade 1/2: 0 0</td>
<td>Grade 1/2: 0 0</td>
<td>Grade 1/2: 2 0</td>
<td>3 (17%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Grade 3: 0</td>
<td>Grade 3: 1</td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anaemia</td>
<td>Grade 1/2: 0 2</td>
<td>Grade 1/2: 0 1</td>
<td>Grade 1/2: 0 0</td>
<td>Grade 1/2: 0 0</td>
<td>3 (17%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AST increased</td>
<td>Grade 1/2: 0 0</td>
<td>Grade 1/2: 0 0</td>
<td>Grade 1/2: 0 0</td>
<td>Grade 1/2: 2 0</td>
<td>3 (17%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td>Grade 3: 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Decreased appetite</td>
<td>Grade 1/2: 1 0</td>
<td>Grade 1/2: 1 0</td>
<td>Grade 1/2: 0 0</td>
<td>Grade 1/2: 1 0</td>
<td>3 (17%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyponatraemia</td>
<td>Grade 1/2: 1 0</td>
<td>Grade 1/2: 0 0</td>
<td>Grade 1/2: 1 0</td>
<td>Grade 1/2: 1 0</td>
<td>3 (17%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nausea</td>
<td>Grade 1/2: 0 1</td>
<td>Grade 1/2: 0 1</td>
<td>Grade 1/2: 0 0</td>
<td>Grade 1/2: 2 0</td>
<td>3 (17%)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td>Grade 3: 0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vomiting</td>
<td>Grade 1/2: 1 0</td>
<td>Grade 1/2: 1 0</td>
<td>Grade 1/2: 1 0</td>
<td>Grade 1/2: 0 0</td>
<td>3 (17%)</td>
<td></td>
</tr>
</tbody>
</table>

---

- 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.
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.
**DCC-3116 | PHASE 1 STUDY**

**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

**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
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\textsuperscript{G12C} inhibitors.

Notes: Data presented at the ESMO Congress 2022; data cutoff: June 9, 2022; ALT=alanine aminotransferase; BID=twice daily; DLT=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.
**DCC-3116 | PHASE 1 STUDY**
**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<sup>G12C</sup> inhibitor)

**Part 2**
*Dose Expansion Phase*

- **DCC-3116 + Trametinib**
  - 2<sup>nd</sup> Line PDAC<sup>1</sup>
    - (KRAS-driven)
- **DCC-3116 + Binimetinib**
  - 3<sup>rd</sup>–5<sup>th</sup> Line NSCLC<sup>2</sup>
    - (RAF/RAS-driven)
  - ≥3<sup>rd</sup> Line CRC<sup>2</sup>
    - (RAF/RAS-driven)
- **DCC-3116 + Sotorasib**
  - 2<sup>nd</sup>–3<sup>rd</sup> Line Melanoma<sup>3</sup>
    - (NRAS-driven)
  - 2<sup>nd</sup>–4<sup>th</sup> Line NSCLC<sup>5</sup>
    - (KRAS<sup>G12C</sup>-driven)

---

**Dose Escalation Phase Inclusion Criteria**
- Histologically confirmed diagnosis of an advanced or metastatic solid tumor with a documented RAS, NF1, or RAF mutation

**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 RAF; (3) with a documented mutation in NRAS; (5) with a documented mutation in KRAS<sup>G12C</sup>.
DCC-3116 Q&A

ANTHONY TOLCHER, M.D., FRCPC

STEVE HOERTER

MATT SHERMAN, M.D.

TUCKER KELLY
UNMET MEDICAL NEED IN TGCT AND PHASE 1 UPDATE

Matt Sherman, M.D.
Executive Vice President and Chief Medical Officer
Vimseltinib inhibition of CSF1 receptor.

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.

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=tenoynovial giant cell tumor; TKI=tyrosine kinase inhibitor; (1) Data presented at the ESMO Congress 2022.
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

---

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%)
VIMSELTINIB | ONGOING PHASE 1/2 STUDY OF PATIENTS WITH TGCT

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

<table>
<thead>
<tr>
<th>COHORT 5 (n=8)</th>
<th>COHORT 8 (n=12)</th>
<th>COHORT 9 (n=12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loading Dose</td>
<td>Dose</td>
<td>Loading Dose</td>
</tr>
<tr>
<td>30 mg QD x 5 days</td>
<td>30 mg twice weekly</td>
<td>30 mg QD x 3 days</td>
</tr>
<tr>
<td>Dose</td>
<td>Loading Dose</td>
<td>Dose</td>
</tr>
<tr>
<td>30 mg twice weekly</td>
<td>10 mg QD</td>
<td>6 mg QD</td>
</tr>
</tbody>
</table>

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

<table>
<thead>
<tr>
<th>COHORT A (n=46)</th>
<th>COHORT B (n=12)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TGCT patients with no prior anti-CSF1/CSF1R therapy (previous therapy with imatinib or nilotinib is allowed)</td>
<td>TGCT patients with prior anti-CSF1/CSF1R therapy (previous therapy with imatinib or nilotinib alone would not be eligible)</td>
</tr>
</tbody>
</table>

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.
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.

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
### PHASE 2 BASELINE CHARACTERISTICS

**Cohort A**  
(n=46)  
**Cohort B**  
(n=12)  

<table>
<thead>
<tr>
<th><strong>Median Age, years (range)</strong></th>
<th>44 (21, 71)</th>
<th>47 (26, 65)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>31 (67%)</td>
<td>7 (58%)</td>
</tr>
<tr>
<td>Male</td>
<td>15 (33%)</td>
<td>5 (42%)</td>
</tr>
<tr>
<td><strong>Disease location</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knee</td>
<td>26 (57%)</td>
<td>7 (58%)</td>
</tr>
<tr>
<td>Ankle</td>
<td>9 (20%)</td>
<td>1 (8%)</td>
</tr>
<tr>
<td>Foot</td>
<td>6 (13%)</td>
<td>0</td>
</tr>
<tr>
<td>Hand</td>
<td>0</td>
<td>1 (8%)</td>
</tr>
<tr>
<td>Other(^1)</td>
<td>5 (11%)</td>
<td>3 (25%)</td>
</tr>
<tr>
<td><strong>Patients with at least one prior surgery</strong></td>
<td>31 (67%)</td>
<td>10 (83%)</td>
</tr>
<tr>
<td><strong>Patients with at least one prior systemic therapy</strong></td>
<td>3 (7%)</td>
<td>12 (100%)</td>
</tr>
<tr>
<td>Imatinib</td>
<td>3 (7%)</td>
<td>0</td>
</tr>
<tr>
<td>Pexidartinib</td>
<td>NA</td>
<td>7 (58%)</td>
</tr>
<tr>
<td>Imatinib and pexidartinib</td>
<td>NA</td>
<td>2 (17%)</td>
</tr>
<tr>
<td>Cabiralizumab and pexidartinib</td>
<td>NA</td>
<td>1 (8%)</td>
</tr>
<tr>
<td>Cabiralizumab</td>
<td>NA</td>
<td>1 (8%)</td>
</tr>
<tr>
<td>Vimseltinib</td>
<td>NA</td>
<td>1 (8%)</td>
</tr>
</tbody>
</table>

**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.
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.
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.
## TEAEs in ≥15% of Patients Receiving Vimseltinib

<table>
<thead>
<tr>
<th>Preferred term</th>
<th>Phase 2 Cohorts</th>
<th>Phase 2 All Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cohort A (n = 46)</td>
<td>Cohort B (n = 12)</td>
</tr>
<tr>
<td></td>
<td>All grades</td>
<td>Grade 3/4</td>
</tr>
<tr>
<td>Blood CPK increased</td>
<td>30 (65%)</td>
<td>20 (44%)</td>
</tr>
<tr>
<td>Headache</td>
<td>19 (41%)</td>
<td>0</td>
</tr>
<tr>
<td>Periorbital oedema</td>
<td>16 (35%)</td>
<td>0</td>
</tr>
<tr>
<td>Nausea</td>
<td>14 (30%)</td>
<td>0</td>
</tr>
<tr>
<td>Fatigue</td>
<td>9 (20%)</td>
<td>0</td>
</tr>
<tr>
<td>Asthenia</td>
<td>14 (30%)</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>Myalgia</td>
<td>13 (28%)</td>
<td>0</td>
</tr>
<tr>
<td>Arthralgia</td>
<td>10 (22%)</td>
<td>0</td>
</tr>
<tr>
<td>Rash maculopapular</td>
<td>10 (22%)</td>
<td>1 (2%)</td>
</tr>
<tr>
<td>AST increased</td>
<td>8 (17%)</td>
<td>0</td>
</tr>
<tr>
<td>Face oedema</td>
<td>8 (17%)</td>
<td>0</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>6 (13%)</td>
<td>0</td>
</tr>
<tr>
<td>Oedema peripheral</td>
<td>7 (15%)</td>
<td>0</td>
</tr>
</tbody>
</table>

**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
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

<table>
<thead>
<tr>
<th>Phase 1 All Cohorts</th>
<th>Phase 2 Cohort A</th>
<th>Phase 2 Cohort B</th>
</tr>
</thead>
<tbody>
<tr>
<td>69%</td>
<td>53%</td>
<td>46%</td>
</tr>
</tbody>
</table>

### Median Treatment Duration

<table>
<thead>
<tr>
<th>Phase 1 All Cohorts</th>
<th>Phase 2 Cohort A</th>
<th>Phase 2 Cohort B</th>
</tr>
</thead>
<tbody>
<tr>
<td>16.4 months</td>
<td>9.8 months</td>
<td>5.9 months</td>
</tr>
</tbody>
</table>

### Active Phase 1 Patients

<table>
<thead>
<tr>
<th></th>
<th>Phase 2 Cohort A</th>
<th>Phase 2 Cohort B</th>
</tr>
</thead>
<tbody>
<tr>
<td>59%</td>
<td>61%</td>
<td>67%</td>
</tr>
</tbody>
</table>

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.
VIMSELTINIB MOTION STUDY | PHASE 3 STUDY OF PATIENTS WITH TGCT
A MULTICENTER, RANDOMIZED, PLACEBO-CONTROLLED, DOUBLE-BLIND STUDY

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)

**Placebo (n = 40)**
(24 weeks)

2:1 Randomization
(N = 120)

**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

Notes: BIW=twice weekly; TGCT=tenosynovial giant cell tumor.
TGCT MARKET DYNAMICS AND VIMSELTINIB OPPORTUNITIES

Dan Martin
Senior Vice President and Chief Commercial Officer
VIMSELTINIB | TENOSYNOVIAL GIANT CELL TUMOR (TGCT)

PATIENT JOURNEY OF TGCT PATIENTS NOT AMENABLE TO SURGERY

PCPs, OTHERS

Diagnosis

~14,000–18,000 patients diagnosed annually in U.S. (localized and diffuse)\(^1,2\)

Literature suggests comparable incidence and recurrence rates in Europe\(^1,2\)

\[\text{PCP=primary care physician; TGCT=tenosynovial giant cell tumor.} \]


Amenable to surgery (~98\%)\(^2\)

Recurrence

~2,000–2,400 patients recur after 1\(^{st}\) surgery \(^1,3\)

SURGEONS

>1 additional surgeries

Not amenable to surgery (~2\%)\(^2\)

MEDICAL ONCOLOGISTS

Rx-Treated (not amenable to surgery)

~1,300–1,400 incident Rx-treated patients in the U.S.\(^1,3\)

~8,000 estimated prevalent Rx-eligible patients\(^1,3\)

~14,000–18,000 patients diagnosed annually in U.S. (localized and diffuse)\(^1,2\)

** 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 hepatotoxicity, 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\(^5\)

---

** U.S. TGCT Market Landscape: Imatinib is the Leading TKI Prescribed**\(^1\)

- \(~70\%\)
- \(~15\%\)
- \(~15\%\)

** Avg. Duration of Therapy **
- Imatinib: ~18 months, Pexidartinib: ~8 months\(^2\)

---

VIMSLETINIB | TENOSYNOVIAL GIANT CELL TUMOR (TGCT)

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

### Relative Scoring of Key Product Attributes

<table>
<thead>
<tr>
<th>Clinical Attribute</th>
<th>Vimseltinib</th>
<th>Pexidartinib</th>
<th>Imatinib</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Efficacy</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tumor Response (Objective Response, CBR)</td>
<td>Green</td>
<td>Green</td>
<td>Red</td>
</tr>
<tr>
<td>PROs (Improvement in Pain &amp; Stiffness)</td>
<td>Limited Data</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Safety</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Grade 3/4 AEs</td>
<td>Green</td>
<td>Red</td>
<td>Red</td>
</tr>
<tr>
<td>Hepatotoxicity</td>
<td>Red</td>
<td>Not Reported in TGCT</td>
<td>Red</td>
</tr>
<tr>
<td>Discontinuation Rates (Due to any TEAEs)</td>
<td>Green</td>
<td>Yellow</td>
<td>Red</td>
</tr>
</tbody>
</table>

- **Highly Compelling**
- **Moderately Compelling**
- **Less Compelling**

### Preferred Systemic Treatment For TGCT

- **20 out of 20 TGCT treaters surveyed selected Vimseltinib as their preferred treatment**

### TGCT Treater Sentiments on Vimseltinib Profile

**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

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

- ~2,000–2,400 patients recur after 1st surgery
- ~$850M Total Rx-Treated and Growth Potential
- ~$350M Market Growth Potential
- ~$500M Treated Patient Potential
- ~1,300–1,400 incident Rx-treated patients in the U.S.

U.S. Prevalent Population

- ~8,000 estimated prevalent Rx-eligible patients in the U.S.

E.U. Opportunity

- Comparable incidence and recurrence rates in Europe
- No approved therapies for TGCT

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

- 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

- 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 – 6/30/2021 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
CLOSING REMARKS

Steve Hoerter
President and Chief Executive Officer
THANK YOU