



Deciphera Presents Preclinical Data from DCC-3116 Program at the AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics

October 7, 2021

– DCC-3116 Shown to Inhibit EGFR and EGFR Family Inhibitor-induced Autophagy in Multiple EGFR-mutant Non-Small Cell Lung Cancer Cell Lines and Decrease Tumor Burden in Combination with Osimertinib and Afatinib in EGFR Mutant Xenograft Model –

WALTHAM, Mass.--(BUSINESS WIRE)--Oct. 7, 2021-- Deciphera Pharmaceuticals, Inc. (NASDAQ: DCPH), a commercial-stage biopharmaceutical company developing innovative medicines to improve the lives of people with cancer, today announced the presentation of preclinical data from the Company's first-in-class selective ULK kinase inhibitor, DCC-3116, in combination with EGFR inhibitors in non-small cell lung cancer (NSCLC) models at the AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics.

"The data presented today show that DCC-3116 inhibits the autophagy that develops as a resistance mechanism after treatment with EGFR inhibitors in multiple EGFR-mutant NSCLC cell lines and that DCC-3116 decreases tumor burden when combined with EGFR inhibitors. These findings are particularly important as EGFR is mutated in approximately 30% of NSCLC patients, the vast majority of whom develop resistance to EGFR inhibitors," said Matthew L. Sherman, M.D., Chief Medical Officer of Deciphera. "These results reinforce the broad potential of autophagy inhibition as a mechanism to address the challenge of drug resistance in the treatment of cancer."

Results from the study, presented in a poster titled "DCC-3116, a first-in-class selective inhibitor of ULK1/2 kinases and autophagy, synergizes with EGFR inhibitors osimertinib and afatinib in NSCLC preclinical models" are summarized below. The poster presentation is available on-demand via the meeting website and on the Company's website at www.deciphera.com/presentations-publications.

***In Vitro* Data Show Ability of DCC-3116 to Reduce Autophagy that Develops as a Resistance Mechanism after Treatment with EGFR Inhibitors in NSCLC Cell Lines**

- EGFR inhibitors gefitinib, erlotinib and osimertinib, and the ErbB-family inhibitor, afatinib, activated autophagy three to four-fold over basal levels as measured by pATG13, a cellular substrate of autophagy-initiating kinases ULK1/2, in the EGFR exon 19-deleted HCC827 NSCLC cell line. DCC-3116, a potent inhibitor of ULK1 and ULK2, was shown to inhibit both basal and EGFR-induced phosphorylation of pATG13.
- Treatment of the EGFR T790M-mutated NSCLC cell line H1975 with osimertinib or afatinib, which inhibit the T790M mutation, induced autophagy three-fold over basal levels while treatment with gefitinib or erlotinib, which are not able to inhibit the T790M mutation, did not induce ULK-mediated ATG13 phosphorylation. DCC-3116 potently inhibited osimertinib and afatinib-induced phosphorylation of ATG13 and inhibited the increase in autophagosomes induced by these agents.

***In Vivo* Data Show that Combination of DCC-3116 with EGFR Inhibitors Resulted in Significantly Greater Tumor Responses in NSCLC Xenograft Model**

- The combination of DCC-3116 with osimertinib or afatinib resulted in significantly greater tumor responses than single agent treatments in the H1975 EGFR-mutant xenograft model.

The clinical development plan for DCC-3116 will initially focus on documented RAS and RAF cancer mutations, which utilize autophagy for tumor growth and survival. DCC-3116 is currently being investigated in a Phase 1, multicenter, open-label, first-in-human study designed to evaluate the safety, tolerability, clinical activity, pharmacokinetics, and pharmacodynamics of DCC-3116 as a single agent and in combination with trametinib, a commercially available MEK inhibitor, in patients with advanced or metastatic tumors with a mutant RAS or RAF gene. Following the dose escalation phase, combination expansion cohorts are currently planned in patients with advanced or metastatic pancreatic ductal adenocarcinoma with KRAS or BRAF mutations, non-small cell lung cancer (NSCLC) with KRAS, NRAS, or BRAF mutations, colorectal cancer with KRAS, NRAS, or BRAF mutations, and melanoma with NRAS or BRAF mutations. Combination expansion cohorts are planned to evaluate DCC-3116 in combination with trametinib. Initial data from the Phase 1 dose escalation cohorts is expected in 2022.

About DCC-3116

DCC-3116 is an investigational first-in-class small molecule designed to inhibit cancer autophagy, a key tumor survival mechanism, by inhibiting the ULK kinase. DCC-3116 is currently being studied in a Phase 1, multicenter, open-label, first-in-human study as a single agent and in combination with trametinib, a commercially available MEK inhibitor, in patients with advanced or metastatic tumors with a mutant RAS or RAF gene.

About Deciphera Pharmaceuticals

Deciphera is a biopharmaceutical company focused on discovering, developing, and commercializing important new medicines to improve the lives of people with cancer. We are leveraging our proprietary switch-control kinase inhibitor platform and deep expertise in kinase biology to develop a broad portfolio of innovative medicines. In addition to advancing multiple product candidates from our platform in clinical studies, QINLOCK® is Deciphera's switch control inhibitor for the treatment of fourth-line GIST. QINLOCK is approved in Australia, Canada, China, Hong Kong, Taiwan, and the United States. For more information, visit www.deciphera.com and follow us on LinkedIn and Twitter (@Deciphera).

Cautionary Note Regarding Forward-Looking Statements

This press release contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, as amended,

including, without limitation, our expectations regarding the broad potential of autophagy inhibition as a mechanism to address the challenge of drug resistance in the treatment of cancer, ULK kinase inhibition's potential to represent a new treatment for cancers caused by RAS or RAF cancers, and our planned expansion cohorts. The words "may," "will," "could," "would," "should," "expect," "plan," "anticipate," "intend," "believe," "estimate," "predict," "project," "potential," "continue," "seek," "target" and similar expressions are intended to identify forward-looking statements, although not all forward-looking statements contain these identifying words. Any forward-looking statements in this press release are based on management's current expectations and beliefs and are subject to a number of risks, uncertainties and important factors that may cause actual events or results to differ materially from those expressed or implied by any forward-looking statements contained in this press release, including, without limitation, risks and uncertainties related to the severity and duration of the impact of COVID-19 on our business and operations, our ability to successfully demonstrate the efficacy and safety of our drug candidates and in additional indications for our existing drug, the preclinical or clinical results for our product candidates, which may not support further development of such product candidates, our ability to manage our reliance on sole-source third parties such as our third party drug substance and drug product contract manufacturers, comments, feedback and actions of regulatory agencies, our ability to commercialize QINLOCK and execute on our marketing plans for any drugs or indications that may be approved in the future, our ability to build and scale our operations to support growth in additional geographies, the inherent uncertainty in estimates of patient populations, competition from other products, our ability to obtain and maintain reimbursement for any approved product and the extent to which patient assistance programs are utilized, our ability to comply with healthcare regulations and laws, our ability to obtain, maintain and enforce our intellectual property rights, any or all of which may affect the initiation, timing and progress of clinical studies and the timing of and our ability to obtain additional regulatory approvals, and other risks identified in our Securities and Exchange Commission (SEC) filings, including our Quarterly Report on Form 10-Q for the quarter ended June 30, 2021, and subsequent filings with the SEC. We caution you not to place undue reliance on any forward-looking statements, which speak only as of the date they are made. We disclaim any obligation to publicly update or revise any such statements to reflect any change in expectations or in events, conditions or circumstances on which any such statements may be based, or that may affect the likelihood that actual results will differ from those set forth in the forward-looking statements. Any forward-looking statements contained in this press release represent our views only as of the date hereof and should not be relied upon as representing our views as of any subsequent date. We explicitly disclaim any obligation to update any forward-looking statements.

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