Deciphera Pharmaceuticals Presents Data Highlighting Kinase Inhibitor Pipeline including Proprietary and Partnered Programs at AACR 2015 Annual Meeting

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Preclinical data demonstrate robust and durable kinase inhibition to block key cancer signaling mechanisms targeting the tumor cell and the tumor microenvironment

Deciphera Pharmaceuticals, a clinical-stage biotechnology company focused on developing advanced kinase inhibitor treatments targeting the tumor cell and the tumor microenvironment, today announced that preclinical data on the company's growing pipeline of next generation kinase inhibitors were presented at the American Association for Cancer Research (AACR) Annual Meeting 2015. The presentations include data on Deciphera's most advanced proprietary drug candidates including altiratinib, a potent and balanced inhibitor of MET/TRK/TIE2 and VEGFR2 kinases and rebastinib, a TIE2 inhibitor, and on the Company's work with Eli Lilly and Company on pan-RAF inhibitors.

"We are excited to share the results from these studies of our novel kinase inhibitor drug candidates with the oncology community at AACR. These data demonstrate the unique attributes offered by our advanced small molecule kinase inhibitor therapeutics to block key cancer signaling mechanisms, preventing the ability of tumor cells to thrive and spread," said Michael D. Taylor, Ph.D., Deciphera's President and Chief Executive Officer.

In a poster presentation entitled “Altiratinib is a potent inhibitor of TRK kinases and is efficacious in TRK-fusion driven cancer models,” Deciphera researchers in collaboration with the Memorial Sloan Kettering Cancer Center demonstrated that altiratinib potently inhibited TPM3-TRKA and ETV6-TRKC fusion protein activation and cell proliferation in tumor cell lines. TRK kinases are implicated in a variety of cancers in which TRK gene fusions have been shown to drive tumor growth. Combined with its inhibition of key tumor microenvironment targets including MET, TIE2, and VEGFR2 kinases, altiratinib provides the potential to durably treat cancers in patients harboring these TRK fusions. Altiratinib is currently in a Phase 1 clinical trial in patients with solid tumors.

• In the KM-12 colorectal xenograft model, altiratinib dosed at 15 mg/kg altiratinib inhibited TPM3/TRKA fusion protein phosphorylation by >95% for more than 12 hours, and in a multi-day efficacy study led to significant tumor growth suppression. Similarly, in a ETV6/TRKC fusion protein xenograft study, altiratinib daily administration led to significant tumor regression.

In a poster presentation entitled, “Rebastinib potently inhibits function of perivascular TIE2 expressing macrophages in vitro and in vivo,” Deciphera researchers in collaboration with the Albert Einstein College of Medicine reported that rebastinib, a selective TIE2 kinase inhibitor with picomolar potency, completely blocked TIE-2 expressing macrophage (TEM)-induced tumor cell invasion. TEMs are a population of highly protumoral macrophages that facilitate tumor growth, angiogenesis, invasion, and immunosuppression. Rebastinib has completed a first-in-human study and will enter a Phase 1b study in the second half of 2015.

• When evaluated in vivo in a mouse breast cancer model, rebastinib dosed at 10 mg/kg orally twice weekly was sufficient to impair tumoral TEMs, resulting in significant reduction in tumor vascular permeability and ablation of tumor cell invasion as quantified by circulating tumor cells.

In a poster presentation entitled, “Mouse PDX trial suggests combination efficacy of Raf and EGFR inhibition for colorectal cancer with BRaf or KRas mutation,” preclinical results were presented on LSN3074753, a pan-RAF inhibitor developed by Deciphera in collaboration with Lilly. This program is currently in Phase 1 clinical development. Synergy was found in a subset of colorectal cancers by combining the pan-RAF

inhibitor, which has been shown to inhibit mutant KRAS and BRAF tumor cell drivers that occur in approximately 70% of colorectal cancer patients, with the anti-EGFR antibody cetuximab.

- When evaluated in a collection of 78 patient-derived xenograft models of colorectal tumors, the overall disease control rate (DCR) in the combination arm was 50% (39/78), while cetuximab or LSN3074753 alone had an overall DCR of 24 or 18%, respectively. Further statistical analyses revealed that BRaf mutations are the best predictor of combination synergy. BRaf or KRas mutations were also significantly associated with combination synergy.

About Deciphera Pharmaceuticals

Deciphera Pharmaceuticals seeks to improve treatment for patients with cancer by designing kinase inhibitor therapies that target the hallmarks of cancer biology. We specifically design our small molecule compounds to simultaneously block multiple cancer signaling mechanisms in the tumor cell and the tumor microenvironment to prevent growth and spread. Deciphera's unique approach represents an important advance over current therapies in the durability of kinase inhibition and resiliency to genetic mutations to provide greater benefit across a range of cancers. Deciphera's business strategy is to advance its drug candidates for genetically defined cancers and cancers that target the tumor microenvironment through both proprietary and partnered programs.

Deciphera's internal product pipeline includes altiratinib, a MET/TIE2/TRK/VEGFR2 kinase inhibitor currently in Phase 1 clinical development, a pan-KIT inhibitor (DCC-2618) currently in preclinical development and rebastinib, a TIE2 kinase inhibitor currently in Phase 1 clinical development. Our ultra-specific FMS inhibitor (DCC-3014), which blocks the actions of pro-tumoral macrophages in the tumor microenvironment, is currently in preclinical development. In addition, a pan-RAF inhibitor is currently in Phase 1 development with Lilly.

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