eFFECTOR's Zotatifin (eFT226) Downregulates Key Oncogenic Driver Proteins Including RTKs (HER2, FGFR1 and FGFR2) Providing Single Agent Activity and Key Combination Opportunities

SAN DIEGO, Oct. 26, 2020 (GLOBE NEWSWIRE) — eFFECTOR Therapeutics, Inc., a leader in the development of selective translation regulation inhibitors (STRIs) for the treatment of cancer, today announced that data presented over the weekend at the EORTC-NCI-AACR Molecular Targets and Cancer Therapeutics Symposium (ENA 2020), showed that zotatifin, the company's product candidate targeting eIF4A, downregulates key cancer-driving proteins including receptor tyrosine kinase (RTK) proteins (HER2, FGFR1, and FGFR2), as well as cell cycle protein Cyclin D1. These data also show that the downregulation of these oncoproteins correlates with apoptosis (programmed cell death) and tumor regression in vivo. Further, vertical inhibition of the PI3K-AKT-mTOR-eIF4F pathway through combination of zotatifin with other agents acting in the pathway provided a synergistic effect in RTK-driven tumors. The data were presented in a poster titled "Dissection of cancer therapy combinations in RTK driven tumors using Zotatifin (eFT226), a potent and selective eIF4A inhibitor," in the New Drugs poster session, abstract 196. The poster can be found in the on the company's website.

"These studies not only confirm zotatifin's specific activity of blocking expression of key oncoproteins and cell cycle targets leading to tumor regressions as a single agent, but also highlight the attractiveness of vertical inhibition within the very important PI3K-mTOR-eIF4F oncogenic pathway," said Steve Worland, Ph.D., president and chief executive officer of eFFECTOR. "These data informed our selection strategy for monotherapy and combination clinical expansion cohorts anticipated to start in early 2021."

A Phase 1/2 clinical trial of zotatifin in patients with KRAS- or RTK-mutant solid tumors is ongoing [NCT04092673]. The trial is enrolling patients with activating mutations, amplifications or fusions in HER2, ERBB3, FGFR1, or FGFR2 receptor tyrosine kinases, or any KRAS mutation subtype. The primary objectives of the trial include safety and tolerability of zotatifin as monotherapy. Secondary objectives include antitumor activity and survival, as well as pharmacokinetics of the drug. Exploratory objectives include pharmacodynamics of zotatifin.

About Zotatifin

Zotatifin is a potent and sequence-selective inhibitor of eukaryotic translation initiation factor 4A (eIF4A) mediated translation. Zotatifin is designed to inhibit the translation of mRNAs encoding several important oncogenes and survival factors, including several RTKS, KRAS, Cyclin D, CDK4/6, MYC, MCL1 and BCL-2 resulting in potent in vivo tumor regression in multiple tumor models dependent on these factors, including breast cancer, non-small cell lung cancer, colorectal cancer, hepatocellular carcinoma and B cell lymphomas. Since zotatifin inhibits the translation of mRNA encoding KRAS and RTK, it is not limited to any mutation subtypes. The product candidate is currently being evaluated in a Phase 1/2 clinical trial in patients with solid tumors.

Please visit www.clinicaltrials.gov for further information on ongoing clinical studies of tomivosertib

About eFFECTOR

eFFECTOR is a next-generation oncology company developing a new class of targeted therapies called selective translation regulator inhibitors (STRIs). Tomivosertib, eFFECTOR's MNK1/2 inhibitor is expected to enter KICKSTART, a randomized Phase 2 trial in non-small cell lung cancer (NSCLC) in combination with pembrolizumab, in Q4, 2020. Zotatifin, eFFECTOR's inhibitor of eIF4A, is in a dose-escalation Phase 1 trial, with expansion cohorts expected to open in H1 2021. The company also expects to initiate a clinical study of zotatifin in COVID-19 based on the dependence of SARS-CoV-2 on eIF4A to translate and replicate its RNA genome. eFFECTOR has a partnership with Pfizer addressing eIF4E.

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