Erasca Announces Four Poster Presentations at the Upcoming 34th EORTC-NCI-AACR Symposium

October 12, 2022

Initial Phase 1/1b patient data for ERK1/2 inhibitor ERAS-007 and SHP2 inhibitor ERAS-601 in advanced solid tumors show safe and tolerable profiles for combination development

CNS-penetrant EGFR inhibitor ERAS-801 demonstrates superior anti-tumor activity over osimertinib in preclinical model of EGFR mutant NSCLC with CNS metastases

Potent and selective KRAS G12D inhibitors identified with robust, dose-dependent anti-tumor activity

SAN DIEGO, Oct. 12, 2022 (GLOBE NEWSWIRE) -- Erasca, Inc. (Nasdaq: ERAS), a clinical-stage precision oncology company singularly focused on discovering, developing, and commercializing therapies for patients with RAS/MAPK pathway-driven cancers, today announced four poster presentations at the 34th EORTC-NCI-AACR (ENA) Symposium on molecular targets and cancer therapeutics taking place October 26-28 in Barcelona, Spain.

"Our pipeline continues to advance, and we are excited to share clinical data from our ERAS-007 ERK and ERAS-601 SHP2 programs showing well-behaved pharmacokinetic characteristics, favorable safety and tolerability profiles, and monotherapy efficacy activity that support the potential of both agents to be backbone therapies for the combination treatment of RAS/MAPK pathway-driven solid tumors. We look forward to reporting combination data in the first half of 2023 with ERAS-007 in gastrointestinal cancers and with ERAS-601 in triple wildtype colorectal cancer," said Jonathan E. Lim, M.D., Erasca's chairman, CEO, and co-founder. "Preclinical data characterizing our lead EGFR program further bolster the CNS penetration aspects and broad therapeutic potential of ERAS-801. Additionally, our RAS franchise continues to progress, and we are pleased to share new preclinical data showcasing promising compounds from our ERAS-4 program targeting the KRAS G12D oncogenic mutation."

Poster Presentation Details

Abstract 224: Preliminary results from HERKULES-1: A Phase 1b/2, Open-label, Multi-center Study of ERAS-007, an Oral ERK1/2 Inhibitor, in Patients with Advanced or Metastatic Solid Tumors
Presenter: Judy Wang, M.D., Florida Cancer Specialists, Sarah Cannon Research Institute
Session/Code: Molecular Targeted Agents 2/PP32

Inhibiting ERK, the most distal node of the RAS/MAPK pathway, has the potential to shut down oncogenic signaling. Preliminary HERKULES-1 Phase 1b data evaluating ERAS-007, an oral ERK1/2 inhibitor with prolonged target residence time and efficacy across broad mutational subtypes, demonstrated encouraging single agent activity and favorable safety and tolerability in patients with metastatic solid tumors refractory to standard therapies (NCT04866134).

- Well-behaved pharmacokinetic characteristics enable intermittent BID-QW dosing (*i.e.*, twice daily on a single day each week), offering a novel dosing regimen for combination development
- Monitorable and manageable adverse event (AE) profile with mostly grade 1 and 2 treatment-related AEs and no dose limiting toxicities (DLTs)
- Unconfirmed partial response in a patient with KRAS G12V mutated pancreatic ductal adenocarcinoma (PDAC) was observed following single agent treatment
- Initial Phase 1b combination data from HERKULES-2 in non-small cell lung cancer (NSCLC) are expected in 2023 and from HERKULES-3 in gastrointestinal malignances are expected in the first half of 2023

Abstract 95: Preliminary results from FLAGSHP-1: A Phase 1 dose escalation study of ERAS-601, a potent SHP2 inhibitor, in patients with previously treated advanced or metastatic solid tumors
Presenter: Meredith McKean M.D., M.P.H., Sarah Cannon Research Institute
Session/Code: New Drugs/PP22

Inhibition of SHP2, a convergent RTK signaling node governing cellular growth and survival, has the potential to suppress oncogenic signaling and limit the development of therapeutic resistance as part of a combination regimen. Preliminary Phase 1 data from the ongoing FLAGSHP-1 trial in advanced solid tumors demonstrated promising single agent safety and tolerability and supported the potential of ERAS-601, a potent and selective SHP2 inhibitor, to enhance efficacy in combination with other targeted therapies (NCT04670679).

- Well-behaved pharmacokinetic properties enable both twice daily (BID) and daily (QD) dosing; maximum tolerated doses for BID and QD dosing identified
- Treatment-related AEs were reversible, manageable, and consistent with the known mechanism of action for the SHP2 inhibitor class
- Confirmed partial response in a patient with a BRAF class III mutation was observed following single agent treatment
- Preliminary clinical data support continued development in combination with other anti-cancer therapies. Initial Phase 1b data from FLAGSHP-1 in combination with cetuximab in patients with triple wildtype (KRAS/NRAS/BRAF wildtype) CRC

are expected in the first half of 2023. Currently also evaluating ERAS-601 + sotorasib in patients with NSCLC in HERKULES-2 study

Abstract 90: The CNS-penetrant EGFR inhibitor, ERAS-801, shows promising nonclinical activity in a CNS metastases model of EGFR mutant NSCLC
Presenter: David Nathanson, Ph.D., University of California, Los Angeles
Session/Code: New Drugs/PP22

High central nervous system (CNS) penetration and comprehensive inhibition of both mutated and wildtype forms are limiting therapeutic factors of approved EGFR inhibitors. ERAS-801 is an oral, highly selective EGFR inhibitor with CNS penetration that is four times higher than approved EGFR inhibitors and comprehensive inhibition against oncogenic EGFRvIII alterations and wildtype.

- ERAS-801 is highly CNS penetrant and has *in vitro* activity against EGFR alterations observed in NSCLC
- Preclinical data characterizing the activity of ERAS-801 in a CNS metastases model of EGFR mutant NSCLC support superior anti-tumor activity in these lesions with ERAS-801 relative to osimertinib, the current standard of care treatment for patients with EGFR mutant NSCLC
- ERAS-801 is currently being evaluated in recurrent glioblastoma multiforme in the Phase 1 THUNDERBBOLT-1 trial (NCT05222802)

Abstract 54: Non-clinical identification and characterization of KRAS G12D inhibitors
Presenter: Alexei Brooun, Ph.D., Erasca, Inc.
Session: Molecular Targeted Agents 1/PP20

Currently there are no approved drugs that directly target KRAS G12D, the most prevalent KRAS oncogenic mutation that frequently occurs in PDAC (37%), CRC (12%), and NSCLC (4%).

- Erasca has developed novel compounds that preferentially bind to the GDP state of KRAS G12D as part of its ERAS-4 program
- Potent and selective KRAS G12D inhibitors have been identified with robust dose-dependent tumor growth inhibition and regression activity in a PDAC cell line-derived xenograft (CDX) model
- Lead optimization is ongoing with the aim of identifying a development candidate to advance into clinical studies

About Erasca
At Erasca, our name is our mission: To erase cancer. We are a clinical-stage precision oncology company singularly focused on discovering, developing, and commercializing therapies for patients with RAS/MAPK pathway-driven cancers. Our company was co-founded by leading pioneers in precision oncology and RAS targeting to create novel therapies and combination regimens designed to comprehensively shut down the RAS/MAPK pathway for the treatment of cancer. We have assembled what we believe to be the deepest RAS/MAPK pathway-focused pipeline in the industry. We believe our team's capabilities and experience, further guided by our scientific advisory board which includes the world's leading experts in the RAS/MAPK pathway, uniquely position us to achieve our bold mission of erasing cancer.

Cautionary Note Regarding Forward-Looking Statements
Erasca cautions you that statements contained in this press release regarding matters that are not historical facts are forward-looking statements. The forward-looking statements are based on our current beliefs and expectations and include, but are not limited to: our expectations regarding the potential therapeutic benefits of our product candidates and development programs, including ERAS-007, ERAS-601, ERAS-801, and ERAS-4; our expectations regarding the monotherapy data for ERAS-007 and ERAS-601 described in this press release being indicative of future clinical results; our beliefs regarding market sizes and opportunities; and the planned advancement of our development pipeline, including the anticipated timing of data readouts for our HERKULES-2, HERKULES-3, and FLAGSHP-1 clinical trials, and other upcoming development milestones. Actual results may differ from those set forth in this press release due to the risks and uncertainties inherent in our business, including, without limitation: preliminary results of clinical trials are not necessarily indicative of final results and one or more of the clinical outcomes may materially change as patient enrollment continues, additional reviews of the data occurs, and more patient data become available; our approach to the discovery and development of product candidates based on our singular focus on shutting down the RAS/MAPK pathway, a novel and unproven approach; delays in our preclinical and clinical development programs; our dependence on third parties in connection with manufacturing, research, and preclinical and clinical testing; unexpected adverse side effects or inadequate efficacy of our product candidates that may limit their development, regulatory approval, and/or commercialization, or may result in recalls or product liability claims; unfavorable results from preclinical studies or clinical trials; results from preclinical studies or early clinical trials not necessarily being predictive of future results; the inability to realize any benefits from our current licenses and acquisitions and any future licenses, acquisitions, or collaborations, and our ability to fulfill our obligations under such arrangements; regulatory developments in the United States and foreign countries; our ability to obtain and maintain intellectual property protection for our product candidates and maintain our rights under intellectual property licenses; our ability to fund our operating plans with our current cash, cash equivalents, and marketable securities; our ability to maintain undisrupted business operations due to the COVID-19 pandemic and global geopolitical events, such as the ongoing conflict between Russia and Ukraine; unstable market and economic conditions having serious adverse consequences on our business, financial condition and stock price; and other risks described in our prior filings with the Securities and Exchange Commission (SEC), including under the heading "Risk Factors" in our annual report on Form 10-K for the year ended December 31, 2021, and any subsequent filings with the SEC. You are cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date hereof, and we undertake no obligation to update such statements to reflect events that occur or circumstances that exist after the date hereof. All forward-looking statements are qualified in their entirety by this cautionary statement, which is made under the safe harbor provisions of the Private Securities Litigation Reform Act of 1995.

Contact:
Joyce Allaire
LifeSci Advisors, LLC

jallaire@lifesciadvisors.com

Source: Erasca, Inc.

Source: Erasca, Inc.