Erasca Reports Broad and Robust Phase 1 Clinical Efficacy and Favorable Safety Data for Pan-RAS Molecular Glue ERAS-0015

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ERAS-0015 at 32 mg QD achieved 44% confirmed ORR in 2L+ KRAS G12X PDAC, despite heavy pretreatment (84% in the 3L+ setting) and a high proportion of G12R mutations (44% of patients)

ERAS-0015 at 32 mg QD achieved 69% confirmed ORR in 2L+ KRAS G12D or G12V PDAC

ERAS-0015 at 32 mg QD achieved 60% confirmed ORR in post-ICI/platinum (2L/3L) KRAS G12X NSCLC

ERAS-0015 at 32 mg QD continued to be generally well tolerated, with mostly low-grade TRAEs and low rates of dose interruptions, reductions, and discontinuations

Early combination data with gemcitabine plus nab-paclitaxel, pembrolizumab, and panitumumab support ERAS-0015's combination backbone potential

ERAS-4001 showed preliminary Phase 1 antitumor activity, favorable tolerability, and encouraging PK; dose escalation of the ERAS-0015 + ERAS-4001 combination expected to initiate in 2027

Conference call and live webcast today at 8:00 a.m. Eastern Time

SAN DIEGO, Nov. 20, 2026 (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 positive updated U.S. Phase 1 monotherapy and initial combination data reinforcing the best-in-class potential of pan-RAS molecular glue ERAS-0015 in patients with RAS-mutant solid tumors. The Company confirms its plans to initiate a potentially registration-enabling trial in 2L+ lung cancer in the first half of 2027 and a Phase 3 trial in 1L lung cancer in the second half of 2027 to first half of 2028 timeframe; and is accelerating the planned initiation of a Phase 3 trial in 1L pancreatic cancer to the first half of 2027. The Company also announced encouraging initial U.S. Phase 1 monotherapy data for potential first-in-class pan-KRAS inhibitor ERAS-4001, supporting its combination potential with ERAS-0015.

"These compelling results highlight the strength and breadth of ERAS-0015's clinical activity across several tumor types," said Jonathan E. Lim, M.D., Erasca's chairman, CEO, and co-founder. "We are particularly encouraged by confirmed responses in common KRAS mutation subtypes, including G12D and G12V, which together occur in nearly 70% of patients with pancreatic cancer. Importantly, we believe the robust clinical activity observed in this heavily pretreated population, which included a high proportion of patients whose tumors harbored less sensitive mutations, compares favorably with reported activity for other pan-RAS and mutant-selective inhibitors, subject to the limitations of cross-study comparisons. Additionally, we believe these efficacy results, coupled with generally well-tolerated safety data and low rates of dose interruptions, reductions, and discontinuations at 32 mg once daily, support advancing ERAS-0015 toward multiple potentially registration-enabling trials."

Dr. Lim continued, "Early combination data demonstrating favorable tolerability results with chemotherapy, an immune checkpoint inhibitor, and an anti-EGFR antibody, together with preliminary clinical activity for ERAS-4001, strengthen our rationale for expanding ERAS-0015's combination potential. We expect 2027 to be an important year for our RAS-targeting franchise as we plan to initiate potentially registration-enabling clinical trials in pancreatic and lung cancers and advance combination dose-escalation trials, including the planned ERAS-0015 plus ERAS-4001 combination."

Key U.S. Phase 1 interim results for ERAS-0015 (AURORAS-1):

As of the data cutoff date, ERAS-0015 monotherapy at the pharmacologically active doses (PAD) of 16-32 mg once daily (QD) (N=122) was generally well-tolerated with favorable safety data, and demonstrated encouraging activity in key KRAS G12X tumors. Monotherapy responses shown are confirmed. All patients with responses at 32 mg QD in all tumor types remained on treatment.[1,2]

ERAS-0015 Monotherapy Showed Strong Anti-Tumor Activity Across Advanced KRAS-Mutant Cancers

- **KRAS G12X pancreatic ductal adenocarcinoma (PDAC) at 32 mg QD in the second-line or greater (2L+) setting[1,2]**
  - 44% confirmed objective response rate (ORR) across all patients (N=25) with KRAS G12X mutations; 84% were treated in the third-line or later (3L+) setting and 44% had G12R mutations
  - 69% confirmed ORR in patients (N=13) with KRAS G12D or G12V PDAC, which represent nearly 70% of patients with PDAC. Of these patients, 77% were treated in 3L+
    - 63% confirmed ORR (N=8) in patients with KRAS G12D
    - 80% confirmed ORR (N=5) in patients with KRAS G12V
  - All (11 of 11) patients with confirmed responses and 6 of 11 patients with stable disease remained on treatment

- **KRAS G12X non-small cell lung cancer (NSCLC)[1,2]**
  - 60% confirmed ORR (N=5) at 32 mg QD and 58% confirmed ORR (N=12) across the PAD range of 16-32 mg QD in the 2L or 3L setting following an immune checkpoint inhibitor and platinum-based chemotherapy (post-ICI/platinum)
  - Activity was observed across multiple KRAS mutation subtypes at 32 mg QD in the 2L+ setting:
    - 60% confirmed ORR (N=5) in patients with KRAS G12D mutations
    - 50% confirmed ORR (N=4) in patients with KRAS G12V mutations
  - At 32 mg QD in the 2L+ setting, all (5 of 5) patients with confirmed responses and all (5 of 5) patients with stable disease remained on treatment
- **KRAS G12X biliary tract cancer (BTC) at 32 mg QD in the 1L+ setting[1]**
  - 75% confirmed ORR (N=4)
  - All (3 of 3) patients with confirmed responses remained on treatment

ERAS-0015 Monotherapy Continued to Be Generally Well-Tolerated with Favorable Safety Data in Expanded Patient Population[1,5]

- Frequency and severity of treatment-related adverse events (TRAEs) remained consistent with the Company's April and July 2026 interim data disclosures
- Mostly low-grade TRAEs with no dose-limiting toxicities (DLTs) and low rate of dose interruptions, reductions, and discontinuations due to TRAEs
  - At 32 mg QD (N=54), TRAEs led to dose interruptions in 13.0% of patients and dose reductions in 16.7% of patients, and there were no dose discontinuations (0% of patients)
- 100% median relative dose intensity (RDI) at 32 mg QD

Early ERAS-0015 Combination Data Support Further Development and Potential for Differentiation with Standard-of-Care Therapies[3]

- **ERAS-0015 plus gemcitabine + nab-paclitaxel (Days 1 and 15)**:
  - Generally well tolerated with favorable safety data and no unexpected safety signals
  - The DLT window was cleared through the 16 mg QD ERAS-0015 cohort with no DLTs
  - Currently dosing the 24 mg QD ERAS-0015 dose escalation cohort
  - Efficacy data not yet available
  - Clinical data support further development in 1L PDAC
- **ERAS-0015 plus pembrolizumab:**
  - Generally well tolerated with favorable safety data and no unexpected safety signals
  - The DLT window was cleared through the 32 mg QD ERAS-0015 cohort with no DLTs
  - Multiple responses observed in multiple solid tumor types in dose escalation
  - Clinical data support further development in 1L NSCLC, including combination with carboplatin/pemetrexed /pembrolizumab (initiated in Q4)
- **ERAS-0015 plus panitumumab**:
  - 16 mg QD ERAS-0015 was generally well tolerated with favorable safety data and no unexpected safety signals
  - Multiple responses were observed in dose escalation in patients with KRAS G12X colorectal cancer (CRC), including 2 ongoing confirmed partial responses (PR) in 2 patients treated at 24 mg QD who then dose reduced to 16 mg QD, and 1 unconfirmed PR (uPR) in 4 patients treated at 16 mg QD
  - Combination dose exploration ongoing; plan to make dose selection in 2027

Encouraging Preliminary ERAS-4001 Phase 1 Data Support Planned Combination with ERAS-0015[4]

- Favorable safety and tolerability data with predominantly low-grade adverse events and no TRAEs leading to treatment discontinuation

- Pharmacokinetic (PK) results were encouraging and support continued dose escalation
- Preliminary monotherapy antitumor activity supports ERAS-4001's potential as a combination partner for ERAS-0015
- Nonclinical data demonstrated that the combination of ERAS-0015 and ERAS-4001 targets both active- and inactive-state KRAS and exhibits synergistic activity in KRAS G12X-driven models, providing a rationale for further evaluation of this combination

Key Anticipated Milestones

ERAS-0015: AURORAS-1 and Phase 3 Trials

- Potentially registration-enabling trial in patients with 2L+ RAS-mutant NSCLC expected to initiate in the first half of 2027
- Phase 3 trial in patients with 1L PDAC accelerated to initiate in the first half of 2027
- Phase 3 trial in patients with 1L RAS-mutant NSCLC expected to initiate in the second half of 2027 to the first half of 2028
- Combination dose escalation and expansion data expected in 2027

ERAS-4001: BOREALIS-1 Trial

- Initiation of combination dose escalation trial with ERAS-0015 planned for 2027

[1] Data cutoff date (DCO) September 1, 2026
[2] Includes responses confirmed after the data cutoff date
[3] DCO October 14, 2026
[4] As of November 9, 2026
[5] As disclosed by the Company in April 2026, one Grade 3 TRAE of pneumonitis progressed to Grade 5 after withdrawal of supportive care per patient decision. The patient was a 66 year-old male with heavily pretreated metastatic pancreatic adenocarcinoma who received 24 mg of ERAS-0015. The patient had pulmonary metastases, a history of right lung cryoablation and no history of lung radiation. The patient presented to the ER approximately a month after starting ERAS-0015 with Grade 3 pneumonitis that was treated aggressively with immediate discontinuation of ERAS-0015, high dose steroids and infliximab. The patient requested withdrawal of supportive care and ultimately died of the event. No additional Grade 5 TRAEs have occurred

Virtual Investor Event Webcast Information
Erasca will hold a virtual investor event today, Friday, November 20, 2026 at 8:00 a.m. Eastern Time. To register for the event please click here. The live webcast and replay may be accessed by referring to Erasca's website at Erasca.com/events.

About ERAS-0015
ERAS-0015 is an investigational, oral, highly potent pan-RAS molecular glue designed to inhibit RAS signaling with a potential best-in-class profile and has received Fast Track Designation (FTD) from the U.S. Food and Drug Administration (FDA) for the treatment of patients with metastatic pancreatic adenocarcinoma. The ongoing AURORAS-1 Phase 1 trial is evaluating ERAS-0015 as a monotherapy and in combination with other therapies in patients with RAS-mutant solid tumors. Clinical data have demonstrated robust monotherapy activity in pancreatic ductal adenocarcinoma (PDAC) and KRAS-mutant non-small cell lung cancer (NSCLC), and encouraging initial activity in KRAS-mutant colorectal cancer (CRC) in combination with an anti-EGFR antibody. ERAS-0015 has been generally well-tolerated with favorable safety data and with well-behaved pharmacokinetics (PK). Emerging tolerability data with chemotherapy, pembrolizumab, and panitumumab support combination development at ERAS-0015 doses within the pharmacologically active dose (PAD) range. ERAS-0015 is also designed to inhibit RAS wildtype variants, which may contribute to resistance to mutant-selective approaches. Together, these findings support its potential as both a monotherapy and backbone for combination regimens.

About ERAS-4001
ERAS-4001 is an investigational, oral, highly potent, and selective pan-KRAS inhibitor with a potential first-in-class and best-in-class profile. Erasca is evaluating ERAS-4001 in the BOREALIS-1 Phase 1 trial in patients with KRAS-mutant solid tumors. Preliminary clinical data have demonstrated favorable safety and tolerability across doses evaluated. Promising preliminary pharmacokinetic (PK) data have been observed. In nonclinical studies, ERAS-4001 showed potent activity against both guanosine triphosphate (GTP)-bound (active state) and GDP-bound (inactive state) KRAS and induced tumor regression in multiple KRAS-mutant models. ERAS-4001 also inhibited wildtype KRAS, which may contribute to resistance to mutant-selective approaches, while sparing wildtype HRAS and NRAS. Preliminary monotherapy antitumor activity supports ERAS-4001's potential as a combination partner for ERAS-0015.

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 patients with cancer. 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 for each of our product candidates, including ERAS-0015 and ERAS-4001; the planned advancement of our

development pipeline, including the anticipated timing of data readouts for the AURORAS-1 and BOREALIS-1 trials, and our expectations related to the initiation of our clinical trials and patient cohorts; our plans to initiate multiple potentially registration-enabling trials for ERAS-0015 in lung cancer and pancreatic cancer in the time periods set forth in this press release; characterizations of the clinical profile of ERAS-0015 and ERAS-4001; the potential for ERAS-0015 to be used in combination therapies, including in combination with ERAS-4001; the potential for ERAS-0015 to be best-in-class; and the potential for ERAS-4001 to be first-in-class and best-in-class. 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: potential delays in the initiation, enrollment, data readout, and completion of clinical trials, including the AURORAS-1 and BOREALIS-1 trials and the planned future trials discussed in this press release; our product candidates, including ERAS-0015 and ERAS-4001, may not demonstrate therapeutic benefits that we expect; interim, topline and preliminary results of a clinical trial are not necessarily indicative of final results and one or more of the clinical outcomes may materially change as patient enrollment continues, following more comprehensive reviews of the data and as more patient data becomes available, including the risk that an unconfirmed partial response to treatment may not ultimately result in a confirmed partial response to treatment after follow-up evaluations; the limitations of preliminary data based on small patient populations and limited follow-up, and of cross-study comparisons that are not based on head-to-head trials and may not be directly comparable due to differences in study designs, patient populations and assessment methods; FTD may not result in a more expedited development or regulatory review process, and such a designation does not increase the likelihood that ERAS-0015 will receive marketing approval in the United States; our approach to the discovery and development of product candidates is based on our singular focus on shutting down the RAS/MAPK pathway, a novel and unproven approach; results from preclinical studies not necessarily being predictive of future results; our assumptions around which programs may have a higher probability of success may not be accurate, and we may expend our limited resources to pursue a particular product candidate and/or indication and fail to capitalize on product candidates or indications with greater development or commercial potential; 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; our planned potentially registration-enabling trials may be delayed based on FDA feedback or requirements, as the FDA retains broad discretion to require additional clinical data prior to the conduct of a registrational trial or submission for regulatory approval; even if our planned trials are successful, they may not support regulatory approval; the inability to realize any benefits from our current licenses, acquisitions, and collaborations, 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, including our ability to successfully defend against allegations raised by, or any future litigation initiated by, Revolution Medicines (RevMed) that ERAS-0015 infringes patents held by RevMed or was derived from RevMed trade secrets; the sufficiency of our cash, cash equivalents, and marketable securities; we may use our capital resources sooner than we expect; 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, 2025, 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.

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