Edison Oncology and Senz Oncology Complete Enrollment in Phase 1–2a Trial of EO1001, Advancing Development in ErbB-Driven Cancers

- ***Early clinical activity observed across multiple ErbB-driven tumor types, including EGFR extracellular domain (ECD)-mutant cancers, for which no approved targeted therapies currently exist***
- ***Brain penetration and preliminary clinical findings support continued development of EO1001 in biomarker-selected populations, including patients with CNS-involved disease***
- ***Analysis of the complete safety, efficacy, pharmacokinetic and biomarker dataset underway ahead of planned presentation at a peer-reviewed medical meeting***

Menlo Park, CA and Melbourne, Australia — July 29, 2026 — Edison Oncology Holding Corp. ("Edison Oncology" or the "Company") and Senz Oncology Pty Ltd. ("Senz Oncology") today announced the completion of enrollment in the ongoing Phase 1–2a study of EO1001, Edison Oncology's oral, brain-penetrant pan-ErbB inhibitor, following the Phase 2 expansion cohorts (described as actively enrolling at the 2026 American Society of Clinical Oncology (ASCO) Annual Meeting). The trial (ANZCTR: ACTRN12620000583943), conducted in collaboration with Senz Oncology at Monash Health and The Alfred Hospital in Melbourne, Australia, is evaluating the safety, pharmacokinetics, and preliminary antitumor activity of EO1001 in patients with advanced ErbB-driven solid tumors, including those with CNS involvement.

Fifty-two patients were screened and thirty-four enrolled across seven ascending dose cohorts (2.5–90 mg once daily orally) and an expansion cohort. EO1001 was generally well tolerated; the most frequent treatment-related adverse events observed to date were diarrhoea, rash, and fatigue (predominantly Grade 1–2 and consistent with known class effects of ErbB inhibitors), with dose-limiting toxicity (diarrhoea) observed at higher dose levels. A full analysis of the safety dataset across the complete enrolled population is underway. In preclinical models, EO1001 achieved brain-to-plasma exposure ratios exceeding 4:1 and tumor-tissue accumulation more than 20-fold higher than erlotinib in EGFR-positive intracranial xenograft mouse tumor models, supporting its potential to address CNS-involved disease. This differentiator was reflected in the trial's inclusion of patients with brain metastases and primary CNS tumors such as glioblastoma.

In an earlier interim analysis of the dose-escalation phase, 10 of 14 evaluable patients treated at doses of 50 mg once daily or higher achieved stable disease or partial response (three partial responses). Disease control and tumor shrinkage were observed across a range of ErbB-positive tumor types including gastric, ovarian, cervical, colorectal, bladder, and glioblastoma (GBM). The companies plan to present updated efficacy, pharmacokinetic, and biomarker data reflecting the full enrolled population at a peer-reviewed medical meeting in the second half of 2026.

"Completing enrollment is a key milestone for the EO1001 program and for our biomarker-directed strategy more broadly," said Jeffrey A. Bacha, chief executive officer of Edison Oncology. "ECD mutations remain one of the most under-recognized mechanisms of resistance and disease progression in ErbB-driven cancers, and the early activity we have seen in this trial — including in heavily pretreated patients with EGFR ECD-altered glioblastoma — reinforces why EO1001 was designed to address this biology. It's fitting that our investigators chose to hold the final enrollment slot for a patient with this

mutation profile; this additional patient will provide further insight into how EO1001 behaves in ECD-mutant disease, and we plan to build on those observations as we advance the program. With enrollment now complete, our focus turns to a full analysis of the safety, efficacy, and biomarker dataset, which we look forward to sharing at a future peer-reviewed medical meeting."

*"*Patients whose tumors harbor ErbB extracellular domain mutations, often across a wide range of cancer types, frequently have few remaining options by the time they reach a trial like this one," said Dr. Sophia Frentzas, Monash Health, coordinating investigator for the study. "Preclinical data showing strong brain penetration for EO1001 was one of the reasons this trial was of real interest to us, given how few therapies reach CNS disease at meaningful concentrations. What's been encouraging clinically is that the disease control, and in some cases meaningful tumor shrinkage, that we've observed in patients with CNS involvement, including recurrent GBM, is consistent with that preclinical profile. Completing enrollment allows us to build a more complete picture of how the drug is performing across dose levels and tumor types, which is exactly what a trial at this stage is designed to do."

EGFR Extracellular Domain (ECD) Mutations: A Large and Underserved Patient Population

As Edison Oncology has previously reported, EGFR extracellular domain (ECD) mutations drive tumor growth through a mechanism that is structurally distinct from the kinase-domain mutations targeted by all currently approved EGFR inhibitors, including osimertinib, erlotinib, afatinib, and dacomitinib — leaving patients with these alterations no approved targeted therapy option.

EGFR ECD mutations occur across multiple tumor types representing large patient populations with high unmet need. In glioblastoma — the most common and lethal primary brain tumor, where EGFR alterations are found in approximately 40–50% of cases — ECD mutations including EGFRvIII and recurrent missense variants such as R108 and A289 substitutions collectively account for the majority of EGFR alterations, with ECD missense variants alone representing approximately 10–15% of all GBM cases. In colorectal cancer, acquired EGFR ECD mutations emerge as a clinically important resistance mechanism to anti-EGFR antibody therapy (cetuximab, panitumumab) in approximately 15–30% of patients who progress on these treatments. ECD-driven EGFR signaling has also been observed in subsets of neuroendocrine tumors and other solid tumor malignancies.

Because EO1001 is designed as a pan-ErbB inhibitor, the same ECD-driven mechanism of resistance extends beyond EGFR to HER2 and HER4, potentially broadening the addressable opportunity:

- **Non-small cell lung cancer (NSCLC)** — HER2 mutations occur in ~2–4% of NSCLC overall; S310F/Y accounts for approximately 11–15% of HER2-mutant NSCLC cases, making it one of the most common non-kinase-domain HER2 alterations in this tumor type.
- **Bladder/urothelial carcinoma, particularly the micropapillary variant** — HER2 ECD mutations (primarily S310F/Y) have been reported in up to 40% of micropapillary urothelial carcinoma (MPUC) cases, compared with roughly 9% in non-micropapillary urothelial carcinoma; a larger follow-up cohort found ECD mutations in ~4% of all urothelial bladder cancer, nearly 30% of which were MPUC.
- **Gastric/esophageal adenocarcinoma** — ERBB2 mutations occur in approximately 7–8% of advanced gastric cancers, with S310F/Y accounting for roughly 19–20% of those mutations; HER2-amplified disease has shown early tumor shrinkage with EO1001 in this trial.

- **Breast cancer, including HER2-nonamplified and invasive lobular subtypes** — ERBB2 mutations are enriched in invasive lobular carcinoma (ILC), occurring in approximately 5.7% of ILC cases versus 1.4% in invasive ductal carcinoma; ILC accounts for 10–15% of all breast cancers.
- **Prostate neuroendocrine carcinoma** — HER2 mutations occur in greater than 10% of cases, among the highest prevalence reported for any tumor type.

Cutaneous melanoma — ERBB4 mutations occur in approximately 19% of melanoma cases, with the majority of mutations concentrated in the receptor's extracellular domain. In the current trial, multiple patients with recurrent glioblastoma harboring EGFR ECD alterations — including EGFRvIII and A289 variants — experienced prolonged stable disease, and additional tumor shrinkage was observed in HER2-amplified esophageal cancer and EGFR-driven head and neck cancer. Across these settings, available EGFR- and HER2-targeted therapies, designed to block the intracellular kinase domain, have demonstrated limited activity, leaving patients with few options — underscoring the case for a biomarker-selected, tumor-agnostic approach.

About the EOHC-1001-01 Trial

EOHC-1001-01 is a two-stage, open-label, multi-centre Phase 1–2a study evaluating ascending single and multiple doses of EO1001 in adults with advanced or metastatic ErbB-positive solid tumors, including patients with CNS involvement, who have relapsed after or are unsuitable for approved therapies. The dose-escalation stage used an accelerated titration design transitioning to a standard 3+3 design upon the first Grade ≥2 treatment-related adverse event, followed by an expansion cohort of up to 20 patients at the recommended dose level. Primary objectives are safety and tolerability; secondary objectives include determination of a recommended Phase II dose, pharmacokinetic characterization, and preliminary antitumor activity by RECIST 1.1 and/or RANO criteria.

About EO1001

EO1001 is an investigational, orally administered, brain-penetrating, irreversible pan-ErbB inhibitor designed to target wild-type and structurally altered EGFR, HER2, and HER4. In preclinical models, EO1001 achieved brain-to-plasma exposure ratios exceeding 4:1 and tumor-tissue accumulation more than 20-fold higher than erlotinib in EGFR-positive intracranial xenograft mouse tumor models, supporting its potential in CNS-involved disease.

Edison Oncology holds exclusive worldwide rights to develop and commercialize EO1001 outside of China, Hong Kong, and Taiwan. With enrollment now complete, the Company is actively seeking development partnerships to advance EO1001 into biomarker-selected expansion and registration-directed studies across ErbB-driven tumor types, with a particular focus on the ECD mutation–defined patient population.

About Edison Oncology Holding Corp.

Edison Oncology is a clinical-stage biopharmaceutical company focused on developing small-molecule therapies for genetically defined cancers with significant unmet need. The Company’s approach centers on identifying compounds with established clinical safety profiles and demonstrated anticancer activity, then applying contemporary tools in genomics, biomarker science, and mechanistic biology to define the patient populations most likely to benefit — an approach made possible by advances in precision oncology that were not available when many of these compounds were first studied. The Company

advances programs through a capital-efficient model combining internal development with strategic co-development and licensing partnerships, retaining meaningful rights while enabling rapid clinical advancement. Edison Oncology's pipeline targets patient populations with no currently approved targeted therapy, including patients with EGFR extracellular domain mutations (EO1001), ARID1A-deficient cancers (EO3001), and CDA-high tumors (EO4426). To learn more, please visit www.edisononcology.com and follow us for updates on LinkedIn.

About Senz Oncology

Senz Oncology Pty Ltd, is a privately-held company based in Melbourne, Australia, is Edison Oncology's Australian clinical development partner and the trial sponsor for the EOHC-1001-01 study, conducted at Monash Health and The Alfred Hospital.

Forward-Looking Statements

[Standard forward-looking statements disclaimer — please provide each company's standard legal language, or confirm you'd like a template version covering both entities.]

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