Edison Oncology Presents New Preclinical Data on EO1001 and EO4426 Highlighting Differentiated Targeting of Cancer Resistance Pathways at AACR 2026

- **EO1001 demonstrates up to ~40-fold greater potency than osimertinib in EGFR ECD–mutant tumor models**
- **EO4426 (tezacitabine) demonstrates selective targeting of DNA replication machinery and resistance to CDA-mediated inactivation**

Menlo Park, CA – April 23, 2026 – Edison Oncology Holding Corp. ("Edison Oncology" or the "Company") today announced presentation of new preclinical data from its EO1001 and EO4426 programs at the American Association for Cancer Research (AACR) Annual Meeting 2026, that took place April 17–22, 2026 in San Diego, California.

"This work highlights two mechanistically distinct programs designed to address biologically defined resistance pathways in cancer," said Jeffrey A. Bacha, chief executive officer of Edison Oncology. "The EO1001 data support its potential relevance in EGFR extracellular domain–mutant tumors, where currently approved therapies have limited activity, while the EO4426 findings further define a selective, replication-directed mechanism with potential utility in CDA-high tumors. Together, these results underscore our strategy to build a precision oncology pipeline grounded in translational science."

EO1001 Demonstrates Activity Against Hard-to-Treat EGFR ECD Mutations Across Tumors

On April 21, 2026, Edison Oncology presented a poster entitled: *Preclinical evaluation of EO1001 in EGFR-ECD mutant solid tumor models: Toward biomarker-directed therapy* in the Experimental and Molecular Therapeutics session at the AACR annual meeting.

EGFR alterations are found across multiple solid tumors. A subset of these—extracellular domain (ECD) mutations—drive tumor growth through ligand-independent signaling and are not well addressed by existing therapies and represent an unmet medical need in cancer.

ECD mutations are particularly relevant in certain tumor types, including glioblastoma, where EGFR alterations occur in approximately 40–50% of cases and frequently involve the extracellular domain. They are also observed in colorectal cancer, where EGFR ECD mutations can emerge as a clinically relevant mechanism of acquired resistance to anti-EGFR therapies, reported in approximately 15–30% of resistant cases, as well as in subsets of neuroendocrine tumors.

EO1001 is an oral, brain-penetrant, irreversible pan-ErbB inhibitor designed to target both wild-type and structurally altered EGFR. Early clinical signals in patients with EGFR ECD–mutant tumors in

an ongoing Phase 1–2a study (ANZCTR: ACTRN12620000583943) support its potential as a biomarker-directed therapy in this patient population.

To further characterize its activity, EO1001 was evaluated in patient-derived tumor models harboring clinically relevant EGFR ECD mutations.

In these studies, EO1001 was tested in two patient-derived cell models carrying EGFR ECD mutations (R108K and A289T), with osimertinib as a comparator. Across both models, EO1001 demonstrated meaningful anti-tumor activity, with greater potency at earlier timepoints and sustained activity over time relative to osimertinib. In the R108K glioblastoma model, EO1001 showed approximately 40-fold greater potency at Day 5, while in the A289T neuroendocrine model, it maintained a consistent activity advantage through Day 7.

These findings demonstrate activity across distinct EGFR ECD mutation classes and tumor types, supporting the continued clinical development of EO1001 as a biomarker-directed therapy for patients with ECD-driven disease.

EO4426: A Targeted Strategy to Overcome Cytidine Analog Resistance in Cancer

On April 22, 2026, Edison Oncology presented a poster entitled *"Overcoming Cytidine Deaminase (CDA)–Mediated Resistance via EO4426 Dual DNA-Replication Targeting: Implications for CDA-High Solid Tumors and Mesenchymal GBM"* in the Experimental and Molecular Therapeutics – Novel Antitumor Agents session at the AACR Annual Meeting.

EO4426 (tezacitabine) is a brain-penetrant cytidine analog designed to disrupt DNA replication in cancer cells through a dual mechanism. It inhibits both DNA polymerase-α (Polα) and ribonucleotide reductase (RNR), leading to depletion of DNA building blocks, replication stress, and accumulation of DNA damage in rapidly dividing tumor cells. Published third-party studies have shown that EO4426 is resistant to deactivation by cytidine deaminase (CDA), an enzyme that inactivates many standard therapies such as gemcitabine.

CDA-mediated inactivation is a well-recognized mechanism of resistance and represents a significant unmet medical need across multiple tumor types. Tumors with high CDA expression—including mesenchymal glioblastoma, triple-negative breast cancer (TNBC), non-small cell lung cancer (NSCLC), and ovarian cancer—may be particularly susceptible to treatment with EO4426.

To further characterize its mechanism and selectivity, EO4426 was evaluated in a series of biochemical assays measuring activity across multiple DNA polymerases. These studies compared EO4426 to gemcitabine using purified enzyme systems to assess both target engagement and potential off-target effects.

In these experiments, EO4426 demonstrated no measurable activity against non-replicative DNA polymerases (POLβ, POLγ, and POLθ) across the tested concentration range, indicating selective targeting of the DNA replication machinery rather than broad polymerase inhibition. In contrast, gemcitabine showed off-target inhibition of select polymerases at higher concentrations.

This selective targeting supports a differentiated profile for EO4426 relative to gemcitabine, with a more focused impact on tumor cell replication and reduced off-target activity. Together, these results support future clinical development of EO4426 as a biomarker-directed therapy, particularly in CDA-high tumors and molecular subtypes such as mesenchymal or APOBEC-enriched glioblastoma.

About Edison Oncology Holding Corp.

Edison Oncology Holding Corp. is a clinical-stage biopharmaceutical company developing a pipeline of first-in-class, small-molecule, biomarker-driven therapies designed to overcome key resistance mechanisms and address critical unmet needs in aggressive and underserved cancers. Leveraging existing clinical data and a modern understanding of cancer biology, Edison Oncology focuses on genetically defined cancers, advancing its programs through a capital-efficient combination of internal development and strategic partnerships while retaining meaningful development and commercial rights. To learn more, please visit https://www.edisononcology.com/ and follow us for updates on LinkedIn.

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