

Edison Oncology Presents New Clinical and Preclinical Data for EO1001 in ErbB-Driven Tumors and EO-4426 in CDA-High Cancers at SNO 2025

Data highlight encouraging clinical activity of EO1001 and differentiated preclinical efficacy of EO-4426 in highly resistant, replication-stress—driven tumors

Menlo Park, CA — November 24, 2025 /Accessnewswire/ — Edison Oncology Holding Corp. ("Edison Oncology" or "the Company") today announced the presentation of new clinical and preclinical data for two of its precision-oncology drug candidates—EO1001, an irreversible pan-ErbB inhibitor, and EO4426 (tezacitabine), a dual DNA polymerase- α and ribonucleotide reductase inhibitor—at the 2025 Society for Neuro-Oncology (SNO) Annual Meeting.

Together, the presentations underscore Edison Oncology's leadership in developing differentiated therapies aimed at addressing major unmet needs in aggressive, treatment-resistant cancers, including those driven by complex genomic alterations and metabolic resistance mechanisms that limit the effectiveness of current treatments.

EO1001: Encouraging Early Clinical Activity and Durable Disease Control in ErbB-Driven Tumors

Interim results from the Phase 1 dose-escalation portion of the ongoing Phase 1–2a trial demonstrated that EO1001 was generally well tolerated across dose levels from 2.5 mg to 90 mg once daily, with mostly Grade 1–2 gastrointestinal and dermatologic adverse events consistent with the ErbB inhibitor class. Dose-limiting toxicities observed at higher dose levels were primarily gastrointestinal—specifically diarrhea—aligned with the known safety profile of FDA-approved ErbB inhibitors. Pharmacokinetic data showed dose-proportional exposure and identified 50 mg once daily as a biologically active dose aligned with preclinical efficacy targets, and both the 50 mg and 70 mg dose levels are currently being evaluated in an ongoing Phase 2a expansion cohort.

Among patients treated at ≥50 mg daily, 10 of 14 evaluable participants achieved stable disease or partial responses, including three partial responses. Several patients derived durable clinical benefit, with five continuing on therapy beyond six months in an extension protocol due to sustained tumor control.

Notably, patients with recurrent glioblastoma harboring EGFR extracellular domain mutations—including EGFRvIII and A289 hotspot variants—experienced prolonged stable disease, consistent with preclinical models showing rapid and efficient brain penetration with brain-to-plasma ratios exceeding 4:1 and durable tumor retention. These data support the potential of EO1001 in genetically defined tumors with limited effective treatment options.

Edison Oncology is conducting the Phase 1–2a clinical trial under the terms of its collaboration and licensing agreement with Apollomics, Inc., through which Apollomics is providing funding for the study while Edison retains responsibility for trial execution and oversight.

EO4426: A CDA-Resistant, Dual-Mechanism Agent Designed for Highly Resistant Solid Tumors

Edison Oncology also presented new mechanistic and preclinical findings for EO4426, a next-generation cytidine analog designed to overcome cytidine deaminase (CDA)-mediated inactivation, a key metabolic resistance mechanism driving poor outcomes in multiple solid tumors. EO4426 retains activation by deoxycytidine kinase and maintains its ability to inhibit both DNA polymerase- α and ribonucleotide reductase, sustaining replication-fork collapse even in highly resistant tumor environments.

In comparative preclinical biochemical studies, EO4426 was shown to be $^{\sim}$ 30-fold more resistant to CDA-mediated degradation than gemcitabine, enabling potent antitumor activity in CDA-high cancers such as non-small cell lung cancer (NSCLC), triple negative breast cancer (TNBC), ovarian cancer, and head and neck squamous cell carcinoma.

Across multiple intracranial and systemic tumor models—including GBM, neuroblastoma, and metastatic TNBC—EO4426 delivered significant survival extension, robust intracranial tumor control, and durable complete responses. In neuroblastoma models, EO4426 achieved up to 90% long-term survival, outperforming standard agents such as BCNU.

Particularly compelling was the drug's activity in mesenchymal (MES) GBM, a subtype associated with APOBEC3G-driven replication stress and high metabolic turnover. EO4426's brain penetration and resistance to CDA inactivation position it as a differentiated treatment option for tumors defined by genomic instability, replication stress, and metabolic resistance, where standard chemotherapies often fail.

Advancing In the Clinic

Edison Oncology is undertaking a coordinated development strategy for both programs:

- EO1001 dose expansion at 50 mg and 70 mg daily is underway to refine the recommended Phase 2 dose and deepen evaluation of early clinical activity.
- EO4426 is advancing toward clinical re-initiation with GMP manufacture of drug product. Planning is ongoing for Phase 1b/2 trials targeting CDA-high metastatic tumors.
- Translational studies across both programs will integrate biomarker analyses and combinationtherapy strategies.

"These data highlight the strength of our platform and the momentum behind our clinical and translational pipeline," said Jeffrey A. Bacha, CEO of Edison Oncology. "EO1001 continues to show early signs of durable clinical activity, including in genomically defined glioblastoma subtypes that have been historically insensitive to ErbB inhibitors. EO4426, with its dual mechanism and resistance to CDA inactivation, represents a differentiated approach for tumors driven by extreme replication stress.

Together, these programs are designed to address some of the most challenging and underserved segments of oncology."

About Edison Oncology

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, the company 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.

Contact

Brett Maas Hayden IR (646) 536-7331 brett@haydenir.com

James Carbonara Hayden IR (646)-755-7412 james@haydenir.com