

PRESS RELEASE

Enterome Phase 1/2 data show OncoMimics™ EO4010-induced CD8 T-cell expansion was associated with longer survival in patients with colorectal cancer

- **Faster and higher expansion of tumor-targeting CD8 T cells associated with longer overall survival in previously treated mismatch repair proficient (pMMR) metastatic colorectal cancer**
- **EO4010 was well tolerated and expanded tumor-targeting CD8 T cells in most patients, with expansion detectable after two to three administrations**
- **Association between CD8 T-cell expansion and clinical outcome, previously reported for EO2463 in lymphoma, now also observed with EO4010 in a solid tumor**
- **Data to be presented in a poster at the European Society for Medical Oncology (ESMO) Congress 2026 in Madrid**

Paris, France – 23 October 2026

Enterome SA, a clinical-stage company pioneering OncoMimics™, a new class of off-the-shelf, multi-targeted in vivo immune therapies, today announced final data from the Phase 1/2 AUDREY study of OncoMimics™ EO4010 in combination with nivolumab, with or without bevacizumab, in patients with mismatch repair proficient (pMMR) metastatic colorectal cancer (mCRC) previously treated with standard chemotherapy and anti-VEGF or anti-EGFR therapy. The data show that EO4010 expands CD8 T cells directed at the tumor-associated antigens it mimics in most patients, and that fast and high-level expansion of these tumor-targeting CD8 T cells is associated with longer overall survival.

The data are being presented on 25 October 2026 in a poster at the ESMO Congress 2026 in Madrid, Spain ([Abstract 859P](#)), by presenting author Dr. Elena Élez Fernandez, Vall d'Hebron University Hospital, Barcelona.

“pMMR metastatic colorectal cancer remains one of the most difficult settings for immunotherapy. The consistent expansion of tumor-directed CD8 T cells in these patients, and its association with survival, support continued investigation of this approach in a controlled study,” said **Dr. Elena Élez Fernandez, Vall d'Hebron University Hospital, Barcelona, Spain, presenting author.**

“In this pretreated population of patients with pMMR colorectal cancer, EO4010 rapidly expanded CD8 T cells recognizing the peptides of EO4010 in every patient, and in most patients, these CD8 T cells cross-reacted with the tumor-associated antigens that EO4010 mimics. The speed and level of CD8 T-cell expansion have now been shown to be associated with overall survival. This encouraging signal from a small Phase 1/2 trial, together with the data pointing to BIRC5 as the epitope driving it as well as the good tolerability we observed with EO4010 treatment, gives us clear support for the continued clinical evaluation of EO4010 in pMMR mCRC,” said **Jan Fagerberg, MD, Chief Medical Officer of Enterome.**

“Since the war on cancer began, researchers have been frustrated by the disease’s ability to evade the body’s immune system. These data presented at the ESMO conference confirm and extend to solid tumors what we reported this year with another OncoMimics™ candidate, EO2463, in blood cancer. CD8 T cells raised by OncoMimics™ are associated with patient outcomes. In short, we have shown that

OncoMimics™ therapeutic intervention is teaching the patients' immune systems how to recognize their cancer," said **Pierre Belichard, Chief Executive Officer of Enterome**.

"With clinical signals now in three programs across glioblastoma, lymphoma and colorectal cancer, we believe the OncoMimics™ platform has shown potentially disruptive therapeutic promise as it is now validated in both blood and solid cancers. Further studies are needed to determine the magnitude and duration of effect of OncoMimics™ and this is the basis of our ongoing discussions with partners and investors on the next stage of development for the portfolio."

The Phase 1/2 AUDREY trial treated 25 patients with unresectable pMMR mCRC whose disease had progressed after oxaliplatin, irinotecan, fluoropyrimidines and anti-VEGF or anti-EGFR therapy. Twenty patients received EO4010 plus nivolumab (Cohorts 1 and 2) and five patients received EO4010 plus nivolumab and bevacizumab (Cohort 3). Patients in Cohorts 1 and 2 had received a median of three prior lines of systemic treatment for metastatic disease. pMMR tumors account for the large majority of mCRC and generally do not respond to checkpoint inhibitors alone.

Key data presented at ESMO:

- **Tolerability:** EO4010 in combination with nivolumab, with or without bevacizumab, was well tolerated. The most common treatment-related adverse event was local administration site reaction (84% of patients). The only related grade 3 events were two local administration site reactions, and there were no grade 4 or 5 events.
- **Immune response:** EO4010-induced expansion of CD8 T cells specific for the EO4010 peptides and for their tumor-associated antigen targets was detected in 100% and 84% of patients, respectively. Expansion was fast, with 81% and 71% of patients positive after two to three administrations (weeks 5-6), and durable, with specific CD8 T cells still detectable at 10 months of follow-up.
- **Clinical activity:** Disease control was achieved in 6 of 25 patients (24%), with a median duration of disease control of 5.5 months, including one partial response with 47% shrinkage of liver metastases and 34% shrinkage of lung metastases lasting 8.7 months. Median overall survival was 11.3 months (95% CI 7.1–not reached) in Cohorts 1 and 2, with a median follow-up of 16.8 months, and was not reached in Cohort 3 (95% CI 9.7–not reached; median follow-up 12.0 months).
- **Association with survival:** In Cohorts 1 and 2, faster and higher expansion of tumor-targeting CD8 T cells was associated with longer overall survival in univariate analyses. Across all cohorts, patients with expansion at week 5 of CD8 T cells specific for BIRC5, or for the EO4010 peptide EO2317 that mimics BIRC5, lived longer than patients without such expansion (BIRC5: HR 0.27, 95% CI 0.08–0.91, log-rank $p=0.024$; EO2317: HR 0.15, 95% CI 0.03–0.69, log-rank $p=0.005$; $n=21$ evaluable patients). All p -values are nominal. Exploratory analyses indicate that anti-cancer treatments received after the study do not account for the survival signal.

Earlier this year, Enterome reported at the European Hematology Association (EHA) Congress and the Pan Pacific Lymphoma Conference (PPLC) that EO2463-induced CD8 T-cell expansion was associated with objective response, complete response and progression-free survival in patients with indolent non-Hodgkin lymphoma. Enterome expects to present further EO2463 data at the American Society of Hematology (ASH) Annual Meeting in December 2026.

ESMO Congress 2026 Presentation Details

Title: EO4010 (EO) multi-target peptide immunotherapy: tumor directed CD8 T cell expansion kinetics and association to survival in patients (pts) with treated mismatch repair proficient (pMMR) metastatic colorectal carcinoma (mCRC). Study EOCRC2-22/AUDREY (NCT05589597)

Abstract: 859P | Poster Display Session Colon Cancer, Sunday 25 October 2026, 12:00–12:45 PM CET, Poster Area Hall 5 – Copy of the poster is available [here](#).

Congress: ESMO Congress 2026, Madrid, Spain, 23–27 October 2026

Presenting author: Dr. Elena Élez Fernandez, Vall d’Hebron University Hospital, Barcelona, Spain

e-Poster: available to registered delegates on the ESMO 2026 Virtual Congress Platform from Friday 23 October 2026, 08:00 CEST

AUDREY (EOCRC2-22; NCT05589597) is an open-label, multicenter Phase 1/2 study conducted in Europe and the United States evaluating the safety, tolerability, immunogenicity and preliminary efficacy of EO4010 in combination with nivolumab (Cohorts 1 and 2) and with nivolumab plus bevacizumab (Cohort 3) in HLA-A2 positive patients with unresectable pMMR mCRC previously treated with oxaliplatin, irinotecan, fluoropyrimidines and anti-VEGF or anti-EGFR therapy. Twenty-five patients were treated; the data presented at ESMO are the final study data.

EO4010 is an off-the-shelf OncoMimics™ active immunotherapy composed of five synthetic microbial-derived peptides (EO2317, EO2318, OMP10, OMP11 and OMP12) designed to mimic CD8 T-cell epitopes of the tumor-associated antigens BIRC5 (survivin), FOXM1, UBE2C, CDC20 and KIF2C, which control the cell cycle and are upregulated in metastatic colorectal cancer, plus the helper peptide UCP2. This multi-target approach is intended to expand *in vivo* pre-existing memory CD8 T cells that cross-react with the tumor antigens, broaden target coverage and obviate antigen escape.

OncoMimics™ are designed to teach the immune system to recognize and defend against cancer. They consist of bacteria-derived peptide antigens that closely mimic tumor-associated antigens (TAAs) of solid tumors, or lineage markers (e.g. as observed in B cell lymphomas). These peptides induce a fast and potent *in vivo* expansion of effector-memory CD8 T-cells, naturally primed by gut bacteria, and cross-reactive with TAAs/B cell markers, thereby eliciting cytotoxic responses against tumor cells. Because they are recognized as foreign entities by the immune system, OncoMimics™ help overcome the self-tolerance that limits the ability of many cancer immunotherapies to trigger rapid, potent, and durable endogenous immune responses. The synthetically produced OncoMimics™ peptides are selected and designed *in silico* by mining Enterome’s proprietary database of 23 million commensal bacteria genes. Each product combines multiple highly immunogenic peptides specifically designed to broaden target coverage, mitigate tumor heterogeneity and obviate the cancer’s ability to escape the therapeutic intervention.

Enterome SA (www.enterome.com) is a privately held clinical-stage biopharmaceutical company developing breakthrough OncoMimics™ immunotherapeutics for cancer. The three most advanced product candidates have shown positive early data in Phase 2 clinical development, supporting the novel OncoMimics™ modality. The company’s pioneering approach to drug discovery is based on the unique and powerful bacterial Mimicry drug discovery platform, which allows it to discover OncoMimics™ with high similarity to tumor associated antigens (TAA) based on the big-data insights from millions of gut bacterial proteins that live in humans.

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