

How E0771 Breast Cancer Cells Revealed a Hidden Immune Escape Route in the Lung

By Pranitha Bhuthkuri

A landmark 2026 study from the Weizmann Institute of Science uses our [E0771 cells \(SKU: 94A001\)](#) murine breast adenocarcinoma cells to uncover a surprising mechanism by which metastatic cancer evades cytotoxic T cells — with major implications for adoptive cell therapy.

Publication: *Frontiers in Immunology*, 2026 | Cell line: [E0771 \(SKU: 94A001\)](#) | Host strain: C57BL/6 (syngeneic)

Researchers discovered that [E0771 cells \(SKU: 94A001\)](#) entering the lung vasculature **rapidly silence their neoantigen presentation machinery** within 24 hours — becoming functionally invisible to cytotoxic T cells. This lung-specific immune escape was absent at primary mammary tumors in the same animals, identifying it as a new organ-conditioned evasion mechanism.

About the study

Published in *Frontiers in Immunology* (Kizner, Levi et al., January 2026), this study addressed a central question in cancer immunotherapy: can tumor-antigen-specific cytotoxic T lymphocytes (CTLs), administered intravenously, effectively reach and destroy breast cancer micrometastases in the lungs?

The research team used ovalbumin (OVA) as a model neoantigen — engineering it into [E0771 cells \(SKU: 94A001\)](#) so that OT-I transgenic CD8 T cells (which specifically recognize the OVA-derived SIINFEKL peptide on H-2Kb MHC-I) could serve as a precise immunological probe, allowing real-time tracking of T cell encounters with tumor cells across different tissue compartments in immunocompetent mice.

Why [E0771 cells \(SKU: 94A001\)](#) cells?

The [E0771 cells \(SKU: 94A001\)](#) cell line (CH3 Biosystems, SKU 94A001) is a murine mammary adenocarcinoma line syngeneic with the C57BL/6 mouse background — the same genetic background as OT-I transgenic and wild-type recipient mice. This syngeneic compatibility is essential: it allows a functional immune system to be studied without rejection artifacts.

Syngeneic match	Perfectly matched to C57BL/6 hosts, enabling fully immunocompetent models without engraftment failure.
Reliable lung tropism	When injected intravenously, E0771 cells colonize the lung vasculature predictably — ideal for metastasis studies.
Lentiviral amenability	Readily transduced with OVA, RFP, mCherry-CaaX, and SIINFEKL constructs for fluorescent tracking and antigen engineering.
Orthotopic versatility	Well-validated in both mammary fat pad implantation and spontaneous and experimental lung metastasis models.

Key findings

Intravascular survival confirmed	Nearly all E0771 cells in the lung survived inside pulmonary blood vessels in both experimental and spontaneous metastasis settings, confirmed by CD31-labeled 3D light-sheet microscopy.	Complete CTL killing failure in lung	OT-I CTLs failed entirely to eliminate lung-residing E0771 cells, despite successfully clearing the same cells from mammary fat pad tumors in the same experimental system.
Antigen loss within 24 hours	SIINFEKL–H-2Kb pMHC complexes disappeared from the E0771 surface within 24 hours of lung entry. MHC-I protein was initially retained, then slowly declined by 72 hours.	No T cell recruitment	Lung-residing E0771 cells failed to upregulate VCAM-1 or ICAM-1 on nearby vessels. OT-I CTL density near E0771 cells was identical to tumor-free lung regions.
Melanoma cells were killed — ruling out CTL exhaustion	The same OT-I CTLs efficiently eliminated OVA-expressing B16 melanoma cells in the lungs, confirming the problem lies with E0771 antigen presentation, not CTL function.	Direct peptide expression rescues killing	E0771 cells engineered to present SIINFEKL directly (bypassing OVA processing) were effectively eliminated by OT-I CTLs in the lungs — confirming a processing-specific defect.

"Our results are a first demonstration that metastatic breast cancer cells accumulating inside the lung vasculature can evade CTL mediated killing by a rapid lung specific loss of neoantigen processing and pMHC presentation."

— Kizner, Levi et al., Frontiers in Immunology, 2026

FEATURED CH3 BIOSYSTEMS PRODUCT

E0771 Murine Breast Adenocarcinoma Cell Line

Catalog No. 94A001

Species:	Mus musculus
Syngeneic host:	C57BL/6
Tumor origin:	Medullary mammary adenocarcinoma
Growth medium:	DMEM + 10% FBS, 1 mM Na-pyruvate, 10 mM HEPES, 1% Pen-Strep-Amp
Key applications:	Metastasis models, CTL / immunotherapy studies, tumor-immune interaction, adoptive cell transfer (ACT)

Implications for cancer immunotherapy research

Systemic CTL therapies may be insufficient for intravascular lung metastases if antigen processing is shut down before T cell encounter. Even large doses of potent, cognate CTLs failed to clear E0771 cells once inside lung blood vessels.

Bypassing antigen processing — for example by engineering therapies targeting constitutively expressed surface antigens — may be necessary for effective clearance of intravascular lung metastases.

The lung vascular niche actively suppresses IFN-driven immune surveillance. Whether this reflects local IFN- γ depletion, stromal suppression, or intrinsic breast cancer transcriptional adaptation to the lung endothelial microenvironment remains an open and clinically important question.

CXCL12/CXCR4 as a survival axis: The dramatic upregulation of CXCL12 in lung-residing E0771 cells (775-fold) points to an autocrine pro-survival loop that may sustain these cells inside lung vessels — another potential therapeutic target.

Replicate and extend this research

If you are developing experimental lung metastasis models, studying adoptive cell transfer, or investigating organ-specific tumor-immune interactions in immunocompetent settings, [E0771 cells \(SKU: 94A001\)](#) from CH3 Biosystems provide a reliable, well-characterized, and extensively published backbone with proven compatibility with OT-I transgenic systems, lentiviral engineering, and both orthotopic and hematogenous metastasis models.

Citation: Kizner M, Levi N, Sochen C, Ryvkin J, Regev O, Zarbock A, Eisenbach L, Biton M and Alon R (2026). A lung specific escape of intravascular metastatic breast cancer cells from cytotoxic T cell killing. *Front. Immunol.* 16:1599751. doi: 10.3389/fimmu.2025.1599751

[E0771 cells \(SKU: 94A001\)](#) cells used in this study were sourced from CH3 Biosystems (SKU: 94A001) and maintained in DMEM supplemented with 10% FBS, 1 mM sodium pyruvate, 10 mM HEPES, and 1% Penicillin-Streptomycin-Amphotericin B solution, as specified by the original manuscript.