disclosed and claimed in the Licensed Patent Rights for Intraoperative fluorescence imaging to visualize the bile duct and small biliary structures, including gallbladder, during hepato-pancreato-biliary surgery in humans.''

The E–271–2014–0 and E–046–2019–0 patent families are primarily directed to a new class of stable heptamethine cyanine fluorophores and biomedical applications thereof, including near-infrared fluorescent contrast agents for intraoperative fluorescence imaging. The E–036–2018–0 patent family is primarily directed to heptamethine cyanines for use as fluorescent markers of the biliary and renal systems, including methods of intraoperative visualization of biliary structures using such agents.

The prospective exclusive license will be royalty bearing, and the prospective exclusive license may be granted unless within fifteen (15) days from the date of this published notice, the National Cancer Institute receives written evidence and argument that establishes that the grant of the license would not be consistent with the requirements of 35 U.S.C. 209 and 37 CFR part 404.

Complete applications for a license that are timely filed in response to this notice will be treated as objections to the grant of the contemplated exclusive patent license. In response to this Notice, the public may file comments or objections. Comments and objections, other than those in the form of a license application, will not be treated confidentially, and may be made publicly available.

License applications submitted in response to this Notice will be presumed to contain business confidential information and any release of information in these license applications will be made only as required and upon a request under the Freedom of Information Act, 5 U.S.C. 552.

''This Notice is made in accordance with 35 U.S.C. 209(e) and 37 CFR 404 Authority to grant exclusive licenses.''

Dated: August 7, 2026.

Richard U. Rodriguez,

Associate Director, Technology Transfer Center, National Cancer Institute.

[FR Doc. 2026–16444 Filed 8–11–26; 8:45 am]

BILLING CODE 4167–05–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government Owned Invention Available for License: MC38 B2m KO Cell Line

AGENCY: National Institutes of Health, HHS.

ACTION: Notice.

SUMMARY: The National Cancer Institute (NCI) seeks licensees for a CRISPR/Cas9-engineered MC38 B2m knockout murine colon cancer cell line that models tumor resistance to PD-1/PD-L1 checkpoint blockade caused by loss of MHC-I antigen presentation. This research tool provides an opportunity to study checkpoint-refractory tumors and evaluate alternative or combination immunotherapy strategies for cancers that evade conventional T-cell-mediated recognition.

FOR FURTHER INFORMATION CONTACT: Inquiries related to this license opportunity should be directed to: Michael Pollack, Ph.D., Unit Supervisor, NCI, Technology Transfer Center, Email: *PollackM@mail.nih.gov* or Phone: 240–276–5519.

SUPPLEMENTARY INFORMATION: Immune checkpoint blockade (ICB) is a type of cancer immunotherapy targeting proteins such as programmed cell death protein 1 (PD-1) and programmed death-ligand 1 (PD-L1), which tumors use to reduce T-cell immune activity. These therapies can be effective in some patients. However, ICB targeting PD-1/PD-L1 fails to provide clinical benefit for most cancer patients due to primary resistance. In such cases, tumors either do not respond from the outset or acquire resistance after initially responding. One important cause of resistance is defective antigen presentation, the process by which tumor cells display internal protein fragments on their surface using major histocompatibility complex class I (MHC-I) molecules for cancer-killing T cell recognition.

Researchers at the NCI have developed and validated an MC38 B2m knockout murine colon cancer cell line designed to reproduce a clinically relevant form of immunotherapy resistance. Using CRISPR/Cas9, NCI researchers eliminated B2m, a gene required for tumor cells to display MHC-I antigen-presenting molecules to cancer-killing T cells. The resulting MC38 B2m KO cell line produces tumors that lack this key immune-recognition signal and are resistant to anti-PD-1 and anti-PD-L1 therapy in syngeneic mouse models. This gives researchers a defined, practical preclinical model to: (1) study tumor immune escape and (2) evaluate new immunotherapy strategies in a checkpoint-resistant setting. This is a superior approach versus models only in tumors that remain responsive to checkpoint blockade. This model uses a clinically relevant checkpoint-resistance mechanism by deleting B2m, which causes loss of MHC-I antigen presentation and prevents conventional CD8+ T-cell recognition. It is based upon the MC38 colon cancer model, which has significant response to PD-1/PD-L1 immune checkpoint blockade before B2m knockout. MC38 B2m KO tumors show abrogated response to anti-PD-1 and anti-PD-L1 treatment *in vivo,* while wild-type MC38 tumors showed significant tumor growth reduction under the same treatment framework.

The Center for Immuno-Oncology seeks licensees interested in using this cell line as a research tool for immuno-oncology studies. This model may be useful for evaluating alternative or combination immunotherapy strategies for checkpoint-refractory tumors, including cancers that evade conventional T-cell-mediated recognition due to defective antigen presentation. It facilitates mechanistic studies of tumor immune escape, CD8+ T-cell recognition, and tumor microenvironment remodeling.

''This Notice is in accordance with 37 CFR 404.4 Authority to grant licenses.''

NIH Reference Number: E–123–2026–0.

Related Technologies: N/A.

Product Type: Research Tool.

Therapeutic Area(s): Oncology.

Development Stage: N/A.

Publications:

- Chariou, PL, et al. Generation of murine tumor models refractory to αPD-1/-L1 therapies due to defects in antigen processing/presentation or IFNγ signaling using CRISPR/Cas9 (PMID: 38427670)

Patens: N/A.

Potential Commercial Applications:

- Development of cancer therapeutics overcoming of PD-1/PD-L1 checkpoint blockade resistance.
- Development of alternative or combination immunotherapies for checkpoint-refractory tumors.
- Pre-clinical screening of alternative or combination immunotherapies for checkpoint-refractory tumors.
- Evaluation of cancer therapeutic strategies for cancers with defective antigen processing/presentation or loss of MHC-I expression.
- Companion model for comparing checkpoint-resistant MC38 B2m KO

tumors against checkpoint-responsive wild-type MC38 tumors.

Competitive Advantages:

- Uniquely models a clinically relevant checkpoint-resistance mechanism.
- Uses the MC38 colon cancer model, which is well-established and regulatorily de-risked.
- Unique model permitting the interrogation of abrogated response to anti-PD-1 and anti-PD-1 treatment.

Collaboration Opportunity: Licensing.

Dated: August 7, 2026.

Richard U. Rodriguez,

Associate Director, Technology Transfer Center, National Cancer Institute.

[FR Doc. 2026–16449 Filed 8–11–26; 8:45 am]

BILLING CODE 4167–05–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

NIH Owned Invention Available for License: Drug-Regulatable, Inducible Expression of Membrane-Bound Interleukin 12 (DRIM-IL-12) for Use in Adoptive Cell Therapy

AGENCY: National Institutes of Health, HHS.

ACTION: Notice.

SUMMARY: Scientists at the National Cancer Institute (NCI) have developed a novel tightly regulated drug-responsive, membrane-bound IL-12 cytokine platform, that enhances anti-tumor efficacy in adoptive cell therapy (ACT) with engineered T-cells (CAR, TCR, TILs) while improving safety. The NCI seeks research co-development partners and/or licensees to advance this technology toward clinical translation.

FOR FURTHER INFORMATION CONTACT: Inquiries related to this license/co-development opportunity should be directed to: Andrew Burke, Ph.D., Senior Technology Transfer Manager, Email: *burkear@mail.nih.gov* or Phone: 240–276–5484.

SUPPLEMENTARY INFORMATION: ACT offers hope for patients with refractory or metastatic cancers, but effectiveness is frequently undermined by the immunosuppressive tumor microenvironment and T-cell dysfunction. Interleukin-12 (IL-12), a powerful cytokine with strong anti-tumor properties, has long been recognized for its potential to invigorate T-cell responses within tumors. However, systemic administration of IL-12 results in severe toxicity. Further, prior gene therapy strategies failed to provide sufficient control over IL-12 expression. These two factors compromise safety and therapeutic performance.

This invention introduces a Nuclear Factor of Activated T cells (NFAT)-inducible, drug-regulatable, membrane-bound IL-12 (DRIM-IL-12) system that delivers spatiotemporally controlled cytokine expression within the engineered T cell therapy product. This platform ensures IL-12 is expressed only upon T-cell activation. Concurrently, the degron (D) sequence confers lenalidomide-dependent proteasome-mediated degradation—serving as a drug-controlled safety switch to limit systemic toxicity. A transmembrane (TM) domain anchors IL-12 in the plasma membrane, preventing unintended secretion and promoting localized immune modulation. When paired with tumor-specific TCRs or CARs (*e.g.,* anti-mutant p53 or KRAS TCRs, or CD19 CAR), this platform enhances tumor cell killing and long-term survival in preclinical models. In a mouse model, DRIM-IL-12 demonstrated substantially improved safety compared to the previous generation of NFAT-inducible IL-12. The inventors also demonstrate that DRIM-IL-12 expression can be dialed down or fine-tuned to prevent T-cell exhaustion or differentiation, which can occur with uncontrolled IL-12 expression.

''This Notice is in accordance with 37 CFR 404.4 Authority to grant licenses.''

NIH Reference Number: E–217–2023.

Related Technologies: N/A.

Product Type: Therapeutic.

Therapeutic Area(s): Immunology | Oncology.

Development Stage: Pre-clinical (*in vivo* validation).

Publications:

- Kim SP, et al. Drug-regulatable, inducible, and membrane-bound interleukin 12 (IL-12TM-D) for use in adoptive cell therapies against advanced cancers. *https://doi.org/10.1136/jitc-2024-SITC2024.0344.*

Patents: PCT/US2025/031121, filed May 28,2025.

Potential Commercial Applications:

- Solid tumors expressing p53 or KRAS mutations.
- Hematologic malignancies.
- Melanoma.

Competitive Advantages:

- Versatile platform for inducible cytokine regulation.
- Superior survival in mouse models compared with TCR-only T-cells.
- Enhanced tumor cell killing and long-term survival in murine models.
- Decreased IL-12-associated toxicity.
- Maintenance of higher IL-12 expression.
- Improved sensitivity to lenalidomide-mediated degradation.

Collaboration Opportunity: Researchers at the NCI seek licensing and/or co-development research collaborations for developing a novel tightly regulated drug-responsive, membrane-bound IL-12 cytokine platform, that enhances anti-tumor efficacy in adoptive cell therapy (ACT) with engineered T-cells (CAR, TCR, TILs).

Dated: August 7, 2026.

Richard U. Rodriguez,

Associate Director, Technology Transfer Center, National Cancer Institute.

[FR Doc. 2026–16438 Filed 8–11–26; 8:45 am]

BILLING CODE 4167–05–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government Owned Invention Available for License: DNA Methylation-Based Cancer Diagnostics for Accurate Tumor Classification

AGENCY: National Institutes of Health, HHS.

ACTION: Notice.

SUMMARY: This technology encompasses a DNA methylation-based diagnostic platform designed to improve the accuracy and consistency of cancer classification, with demonstrated utility for tumors of the central nervous system, kidney, and hematopoietic system. By identifying disease-specific methylation signatures, the approach reduces interobserver variability and enhances diagnostic confidence. The central nervous system (CNS) classifier is built from a curated reference set of 16,567 methylation profiles and organizes tumors into 22 families and 133 clinically relevant diagnostic classes, including 21 newly developed methylation classes not represented in other existing tools. Across multiple independent validation cohorts (n = 5,875), the classifier demonstrated robust performance, and in a clinical-impact analysis of 1,204 NIH validation cases, methylation profiling materially influenced final diagnosis in 74.4% of cases by refining, increasing precision, or reclassifying. The CNS classifier was deployed as a user-facing software tool, MethylScape Analysis, which streamlines methylation-based classification workflows for CNS tumors, *https://methylscape.ccr.cancer.gov/*. The development of multiple specialized classifiers supports a more granular