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