highly expressed in many solid tumors, such as mesothelioma, ovarian, and pancreatic cancers. MSLN is present at relatively low levels in mesothelial cells of healthy individuals, making it an ideal candidate for targeted therapeutics. However, the efficacy of MSLN-targeted agents is often reduced as a portion of the protein is shed from the cell surface and binds to available anti-MSLN antibodies. This reduces therapeutic engagement at the cell surface as shed MSLN acts as a decoy within tumor microenvironments, limiting antibodies from reaching and destroying tumor cells.

Researchers at the NCI developed a novel mAb, RO4, which specifically binds to the juxta-membrane region of MSLN to block shedding. RO4 binds to the same region of MSLN as a previously developed mAb, 15B6 (NCI Ref. #E–106–2017), but in a different conformation. This allows for increased binding affinity and specificity. CAR–T cells made with humanized RO4 demonstrated higher cytotoxicity both *in vitro* and *in vivo* compared to h15B6 CAR-Ts. Additionally, hRO4 exhibited broader binding across MSLN-positive cell types compared to h15B6, suggesting wider utility in patient populations.

Researchers at the NCI seek licensing and/or co-development research collaborations for further development of RO4 that can be used to treat MSLN-positive cancers.

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

NIH Reference Number: E–051–2024.

Related Technologies: E–106–2017 and E–033–2022.

Product Type: Therapeutic.

Therapeutic Area(s): Oncology | Immunology.

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

Publications:

- Onda M, et al. RO4, a high-affinity humanized antibody against the juxtamembrane region of mesothelin for targeted cancer therapy. (PMID41550974).
- Liu X, et al. Highly active CAR T cells that bind to a juxtamembrane region of mesothelin and are not blocked by shed mesothelin. (PMID35512094).

Patens: National Stage Applications.

Potential Commercial Applications:

- Treatment of various MSLN-positive cancers—including mesothelioma, ovarian and pancreatic cancer.
- Development of CAR T cells and bispecific antibodies to target MSLN and CD3.
- Development of Antibody-Drug Conjugates (ADCs) and Antibody-nanoparticle conjugates to deliver cytotoxic payloads to MSLN-positive tumors.

Competitive Advantages:

- Increased therapeutic effectiveness via avoiding shed decoy interference.
- Enhanced binding affinity and selectivity compared to earlier antibodies.
- Improved cytotoxicity across various MSLN-positive tumors.
- Versatility in use of CAR T cells and bispecific antibodies targeting MSLN and CD3.

Collaboration Opportunity: Researchers at the NCI seek licensing and/or co-development research collaborations for a novel monoclonal antibody (mAb), RO4, that can be used to treat MSLN expressing cancers.

Dated: August 7, 2026.

Richard U. Rodriguez,

Associate Director, Technology Transfer Center, National Cancer Institute.

[FR Doc. 2026–16443 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: Quantitative Particle Identification (QPID) Digital Autoradiography System

AGENCY: National Institutes of Health, HHS.

ACTION: Notice.

SUMMARY: The National Cancer Institute (NCI) seeks research co-development partners and/or licensees for a QPID digital autoradiography system with particle identification capabilities.

FOR FURTHER INFORMATION CONTACT: Inquiries related to this license opportunity should be directed to: Eric Cheng, Ph.D., Technology Transfer Manager, NCI, Technology Transfer Center, Email: *eric.cheng2@nih.gov* or Phone: 240–276–5978.

SUPPLEMENTARY INFORMATION: Autoradiography is a photographic process that exposes a medium, sensitive to radiation, to a sample emitting radiation. The current state of the art involves exposing a thin slice of tissue to an ionization-sensitive film for several hours or days and then scanning the film into an autoradiography image. This image is created using the sum of the ionizations from all the decays that occurred during the time of exposure on the ionization-sensitive medium. Imaging quality of these samples can be improved by collecting information from each individual decay to enhance the autoradiographic measurement.

Researchers at NCI developed a QPID digital autoradiography system to measure the energy deposition from charged particles for each individual radioactive decay. The QPID leverages the ionizing radiation detection features of the Timepix3 detector to generate autoradiograph images in units of dose per unit time and area. The high readout speed of the Timepix3 gives the detector the capability to measure separate decay ionizations. It also segregates the image into one generated only by alpha and the other only by beta particles. A gamma detector was interfaced with the Timepix3—allowing tagging charged particle ionization events in coincidence with a gamma emission during the isotope decay. This allows for a method to distinguish between alpha and positron emitting radioisotopes when imaged concurrently in the same pathology sample. This is the most unique feature of the QPID which will aid the development of new theranostic treatments in which two similar ligands are used.

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

NIH Reference Number: E–201–2023.

Related Technologies: N/A.

Product Type: Device.

Therapeutic Area(s): Oncology | Radiology.

Development Stage: Prototype.

Publications:

- Adler SS, et al. A Quantitative Particle Identification (QPID) spectral autoradiography system. (PMID40374923).

Patens: PCT Stage, filed May 29 2025.

Potential Commercial Applications:

- Improved radiotherapy dosing for cancer treatment.
- Improved radiotherapy guidance for cancer treatment.
- Improve accuracy of biopsy procedures through real-time tracer measurement and detection.
- Measuring dual radioligand pathology samples.

Competitive Advantages:

- QPID can measure charge particle activity for individual decays (alpha, beta, and gamma).
- QPID measures absolute time of decay relative to the start of data acquisition.
- QPID more accurately measures radioactive dose in a tissue sample.

Collaboration Opportunity: Researchers at the NCI seek licensing and/or co-development research collaborations for developing the QPID system for improved autoradiography imaging.

Dated: August 7, 2026.

Richard U. Rodriguez,

Associate Director, Technology Transfer Center, National Cancer Institute.

[FR Doc. 2026–16439 Filed 8–11–26; 8:45 am]

BILLING CODE 4167–05–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Center for Scientific Review; Notice of Closed Meetings

Pursuant to section 1009 of the Federal Advisory Committee Act, as amended, notice is hereby given of the following meetings.

The meetings will be closed to the public in accordance with the provisions set forth in sections 552b(c)(4) and 552b(c)(6), Title 5 U.S.C., as amended. The grant applications and the discussions could disclose confidential trade secrets or commercial property such as patentable material, and personal information concerning individuals associated with the grant applications, the disclosure of which would constitute a clearly unwarranted invasion of personal privacy.

Name of Committee: Center for Scientific Review Special Emphasis Panel; Health Promotion and Implementation.

Date: August 28, 2026.

Time: 9:00 a.m. to 3:30 p.m.

Agenda: To review and evaluate grant applications.

Address: National Institutes of Health, Rockledge II, 6701 Rockledge Drive, Bethesda, MD 20892.

Meeting Format: Virtual Meeting.

Contact Person: Andrea B. Kelly, Ph.D., Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD 20892, (301) 451–6339, *kellya2@mail.nih.gov.*

Name of Committee: Center for Scientific Review, Special Emphasis Panel; Fellowships: Synthetic and Medicinal Chemistry and Chemical Biology deferral panel.

Date: August 31, 2026.

Time: 2:00 p.m. to 5:00 p.m.

Agenda: To review and evaluate grant applications.

Address: National Institutes of Health, Rockledge II, 6701 Rockledge Drive, Bethesda, MD 20892.

Meeting Format: Virtual Meeting.

Contact Person: John J. Laffan, Ph.D., Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Bethesda, MD 20892, (301) 443–7154, *laffanjo@mail.nih.gov.*

Name of Committee: Risk, Prevention and Health Behavior Integrated Review Group; Biobehavioral Medicine and Health Outcomes Study Section.

Date: September 14–15, 2026.

Time: 9:30 a.m. to 6:00 p.m.

Agenda: To review and evaluate grant applications.

Address: National Institutes of Health, Rockledge II, 6701 Rockledge Drive, Bethesda, MD 20892.

Meeting Format: Virtual Meeting.

Contact Person: Mark A. Vosvick, Ph.D., Scientific Review Officer, Center for Scientific Review, National Institutes of Health, 6701 Rockledge Drive, Room 3110, Bethesda, MD 20892, (301) 402–4128, *mark.vosvick@nih.gov.*

(Catalogue of Federal Domestic Assistance Program Nos. 93.306, Comparative Medicine; 93.333, Clinical Research, 93.306, 93.333, 93.337, 93.393–93.396, 93.837–93.844, 93.846–93.878, 93.892, 93.893, National Institutes of Health, HHS)

Dated: August 7, 2026.

Margaret N. Vardanian,

Program Analyst, Office of Federal Advisory Committee Policy.

[FR Doc. 2026–16377 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: Soluble Tissue Factor, a Novel Target, and Antibodies, for Diagnosis, Prevention and Treatment of Thrombosis and Related Conditions

AGENCY: National Institutes of Health, HHS.

ACTION: Notice.

SUMMARY: Scientists at the National Cancer Institute (NCI) have discovered a novel therapeutic, diagnostic and prognostic target for thrombosis: Soluble Tissue Factor (sTF). NCI has generated first-in-class antibodies and platform selectively neutralizing pathological coagulation while preserving normal hemostasis. This platform technology can be used to prevent, diagnose and treat pathological thrombosis caused by a variety of clinical conditions-including cancer, sepsis, infectious diseases (*e.g.,* COVID), autoimmune disorders, trauma, heart conditions and inflammatory conditions. It offers a much more sensitive and specific test of thrombosis than the current D-dimer test. It may be applicable to any clinical condition which induces necroptosis, pyroptosis and NETtosis, such as cancer, neurodegenerative disease, ischemia-reperfusion and acute injuries, traumatic injuries, bacterial and viral infections, cardiovascular conditions, and atherosclerosis, inflammatory, autoimmune conditions, and others.

FOR FURTHER INFORMATION CONTACT: Inquiries related to this license/co-development opportunity should be directed to: Aida Cremesti, Ph.D., Senior Technology Transfer Manager, NCI, Technology Transfer Center, Email: *aida.cremesti@nih.gov* or Phone: 240–276–6641.

SUPPLEMENTARY INFORMATION: Investigators at the National Cancer Institute (NCI) have identified and therapeutically validated a distinctive, disease-restricted driver of thrombosis: soluble tissue factor (sTF). sTF is generated through proteolytic cleavage during inflammatory cell death. Mechanistically, the researchers demonstrated that sTF generation is not limited to necroptosis but represents a conserved outcome of multiple inflammatory cell death pathways, including pyroptosis and NETtosis.

Pathological thrombosis remains a leading cause of morbidity and mortality in sepsis, cancer, autoimmune diseases, viral infections, and inflammatory disorders. Current anticoagulant and thrombosis treatments options such as Heparins and Warfarin are effective at reducing clot formation, but act indiscriminately on the coagulation cascade, disrupt normal hemostasis, and are associated with significant bleeding risk. Current thrombosis diagnostics, D-dimer, a fibrin degradation by product, is a late stage markers that lacks sensitivity. This invention provides an alternative for early detection based on sTF that is more sensitive and specific.

To selectively target this mechanism, the team developed novel humanized monoclonal antibodies (*e.g.,* 58B3 and 56E5) that specifically recognize human soluble tissue factor (hsTF), but not full-length membrane-bound tissue factor (flTF). This selectivity preserves physiological hemostasis while inhibiting pathological coagulation. *In vivo* validation using human samples of various clinical diseases demonstrated that administration of sTF-specific antibodies prevents fibrinogen depletion and reduces thrombin–antithrombin (TAT) complex elevation, effectively normalizing coagulation parameters. These results establish proof of concept that selective sTF neutralization can block pathological thrombosis without systemic anticoagulation.

By establishing sTF as a unifying mechanistic link between inflammatory cell death and thrombosis across sepsis, cancer, infection, autoimmune disease, cardiovascular disorders, and metabolic inflammation, this antibody platform represents a first-in-class strategy to selectively inhibit pathological