

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]

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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]

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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