Science Advancement & Outreach
A DIVISION OF PETA

1536 16th St. N.W., Washington, DC 20036

October 27, 2026

Dear members of the National Diabetes and Digestive and Kidney Diseases Advisory Council,

I'm writing on behalf of Science Advancement and Outreach, the biomedical science policy division of People for the Ethical Treatment of Animals. **We continue to request that the Council advise the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) to stop conducting and funding sepsis research that uses other animals and redirect resources to support sepsis research and training focused exclusively on non-animal methods.**

As we included in our written comment to you on May 13, 2026, more than 150 drugs for human sepsis that appeared promising in preclinical animal testing have failed in clinical trials.[1,2] These repeated failures reflect major biological differences between humans and the species used in sepsis experiments,[3,4,5,6,7,8,9,10] as well as the inability of preclinical sepsis-inducing methods[11,12] and laboratory conditions[13,14,15,16] to accurately reproduce human disease onset and progression.

NIDDK must follow the National Institute of General Medical Sciences' lead in relegating rodent models of sepsis as "low priority,"[17] discouraging their use, and encouraging investigators to use non-animal methods.[18]

To develop safe, effective interventions for patients with sepsis, research must be grounded in human biology—not in approximations derived from fundamentally different species. Importantly, recent analyses have pointed out that "making animal studies more careful and rigorous tends to show that their results predict human outcomes even less well than was thought"[19] and "such efforts have not translated into lower overall clinical failure rates."[20]

NIDDK is currently funding at least 13 projects using mice in attempts to model human sepsis and sepsis-induced complications (see Appendix A for a list). NIDDK has awarded more than $34 million to these projects, which have little to no chance of advancing treatments for sepsis and related complications. <u>The Council must push for a change.</u>

Non-animal methods, including organoids,[21] microfluidic systems,[22,23] genomics,[24,25] and machine learning and other artificial intelligence tools,[26,27] are already advancing sepsis research. These tools offer greater translational relevance than animal models, but they remain dramatically underfunded and underutilized.

Recognizing the need to move away from animal use and the promise of human-based approaches, all relevant government agencies in the U.S. have initiated plans to reduce animal

testing and increase support of non-animal methods.[28,29] NIDDK can leverage this effort and align with the NIH Unified Strategy[30] to drive progress by expanding training and funding opportunities for NAMs so that researchers can better understand and use them in sepsis research. This could include:

- training grants and fellowships focused on human-based approaches,
- education programs developed in partnership with universities and research institutes,
- transition awards for researchers looking to switch from animal experiments to human-based methods,
- and early-career awards to support scientists who wish to begin a fruitful career in human-based sepsis research.

Targeted support from NIDDK would help address barriers to non-animal research and support wider adoption across the research community.

These recommendations are detailed further in our policy roadmap, Research Modernization NOW,[31] including a dedicated section on sepsis.[32] By implementing these recommendations, NIDDK will lead NIH in prioritizing human-based research, advancing predictive and human-relevant science, and accelerating progress toward effective sepsis treatments and improved patient outcomes.

Thank you for your time and consideration. We welcome the opportunity to discuss these recommendations further.

Sincerely yours,



Frances Cheng, PhD
Senior Scientist

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Appendix A: A list of NIDDK-funded projects on sepsis and sepsis-induced complications using mice that are active as of October 2026

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3. Janosevic D. Transcriptomic Diagnosis and Staging of Sepsis-Associated Acute Kidney Injury. https://reporter.nih.gov/search/8_R6OzqWrk2REnESmDyVLw/project-details/11289455
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5. Nakamura T, Zingarelli B. Exploring the Link Between Extracellular Vesicle-Associated Inflammation in Obesity and Sepsis Outcomes. https://reporter.nih.gov/search/8_R6OzqWrk2REnESmDyVLw/project-details/11360890
6. Odum JD. Renal endothelial cell signaling following viral priming in sepsis-associated acute kidney injury. https://reporter.nih.gov/search/8_R6OzqWrk2REnESmDyVLw/project-details/11371546
7. Parikh SM, Parikh CR. Cathepsin K-dependent Angiopoietin processing in sepsis associated AKI. https://reporter.nih.gov/search/8_R6OzqWrk2REnESmDyVLw/project-details/11390741
8. Sanders PW. Pre-Clinical Core. https://reporter.nih.gov/search/8_R6OzqWrk2REnESmDyVLw/project-details/11395335
9. Sanders PW. UAB-UCSD O'Brien Center for Acute Kidney Injury Research. https://reporter.nih.gov/search/8_R6OzqWrk2REnESmDyVLw/project-details/11395319
10. Star RA. Inhibition Of Renal Injury. https://reporter.nih.gov/search/8_R6OzqWrk2REnESmDyVLw/project-details/11410752
11. Susztak K. APOL1 associated disease spectrum. https://reporter.nih.gov/search/8_R6OzqWrk2REnESmDyVLw/project-details/10906257
12. Thom CS. Defining bone marrow factors that support neonatalhematopoiesis. https://reporter.nih.gov/search/8_R6OzqWrk2REnESmDyVLw/project-details/11445172
13. Wang X. Novel role of LSECs in hepatic immune and metabolic function during sepsis. https://reporter.nih.gov/search/8_R6OzqWrk2REnESmDyVLw/project-details/11290410