SEPSIS EXPERIMENTS ON ANIMALS

SCIENTIFICALLY OUTDATED, CLINICALLY USELESS

2025

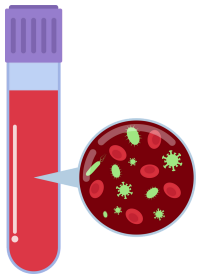
PETA
People for the Ethical Treatment of Animals

Summary

Sepsis, a life-threatening condition resulting from the body's response to infection, affects millions globally every year, leading to high mortality and substantial economic burden. Despite decades of research, advancements in sepsis therapies have been hindered—largely due to the poor translation of findings from experiments using animals, particularly mice. Mice are commonly used in sepsis research due to their low cost and ease of use. However, their biological responses to sepsis differ significantly from those of humans. These differences include genomic, immunological, and metabolic discrepancies, as well as issues with the reproducibility of sepsis induction methods. In 2019, the National Institute of General Medical Sciences (NIGMS), part of the National Institutes of Health (NIH) in the United States, acknowledged these shortcomings and announced plans to shift funding toward more clinically relevant research. Numerous scientists have also called attention to the limitations of using animals in sepsis research. Nevertheless, sepsis studies using animals continue to be conducted and published. Based on the critical scientific and ethical problems with using animals in sepsis research outlined in this report, we recommend that the use of animals in human sepsis research be prohibited by institutional, regional, and national legislative and ethical bodies. Funding agencies should also reject grant applications involving the use of animals in sepsis studies, and academic journals should adopt public policies refusing to publish manuscripts that report such experiments, as they are scientifically invalid.

What is sepsis?

Sepsis is defined as a "life-threatening organ dysfunction caused by a dysregulated host response to infection,"[1] and affects at least 48.9 million globally each year, and kills nearly 11 million of those.[2] It represents 20% of all global deaths and is one of the most expensive conditions to treat.[2,3] Most cases are caused by bacterial infections, but sepsis can also result from viral or fungal infections, or even traumatic injuries.[4] Vulnerable populations—such as infants, the elderly, and individuals with weakened immune systems—are at greater risk.[5] Treatment typically involves antibiotics, intravenous fluids, and sometimes oxygen or vasopressors.[6] When identified early, sepsis can be managed successfully, but survivors often suffer long-term effects, and in many cases it can lead to permanent organ damage or death.[7]



Translational failures in sepsis therapeutics

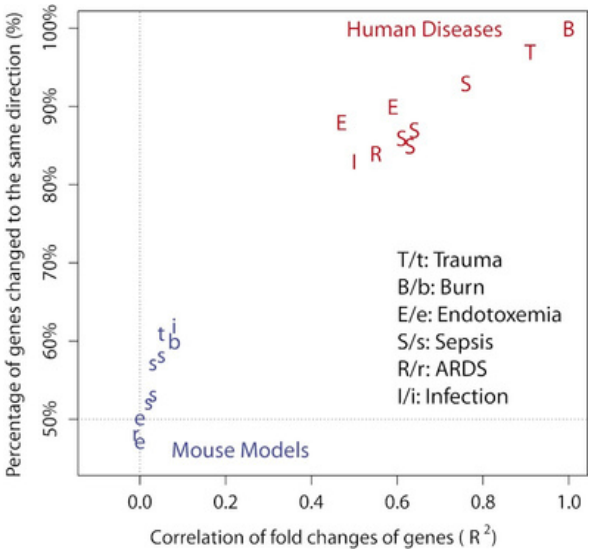
In 2014, Mitchell P. Fink, who is considered "one of the most inspiring and influential leaders in the field of intensive care medicine,"[8] published an article reviewing over 60 human clinical trials conducted since 1982 for the evaluation of pharmacological interventions for the treatment of sepsis. Of these, only eight showed any benefit to patients, and none resulted in a cure. Four trials caused further harm, while the remainder provided no clinical benefit. Furthermore, Fink detailed nine specific examples of pharmacological agents that had yielded beneficial results in several animal experiments but "negative results in one or more human clinical trials."[9] He concluded that "most animal models of human sepsis are flawed" and warned that "results from these preclinical studies never should be extrapolated directly to the problem of human sepsis."

Murine sepsis is not human sepsis

Mice are the most commonly used species in sepsis experiments, not because they make good "models" of human sepsis but because they are cheap, plentiful, small, and docile.[10] The difficulty in reliably translating results from mice to humans is believed to be the primary cause of the failure of practically all human trials of sepsis therapies.

In 2013, Proceedings of the National Academy of Sciences (PNAS) published a landmark study that had been 10 years in the making and involved the collaboration of 39 researchers from institutions across North America, including Stanford University and Harvard Medical School. Dr. Junhee Seok and his colleagues compared data obtained from hundreds of human clinical patients with results from experiments on animals to demonstrate that humans and mice are different in their genetic responses to serious inflammatory conditions such as sepsis, burns, and trauma.[11]

The PNAS paper reveals that in humans, many of the same genes are involved in recovery from sepsis, burns, and trauma, but that it was "close to random" which mouse genes might match these profiles.[11] Not only was the genomic response poorly correlated between mouse models and humans, but the genomic response timing was also different between the two species.[11]



"Comparison of the genomic response to severe acute inflammation from different etiologies in human and murine models." Figure 4 from Seok et al. 2013 PNAS DOI: 10.1073/pnas.1222878110

NIH Director at the time, Dr. Francis Collins, authored an article about these results, lamenting the time and resources spent developing 150 drugs that had successfully treated sepsis in mice but failed in human clinical trials. He called this failure "a heartbreaking loss of decades of research and billions of dollars."[12]

In addition to this landmark study, the criticism of mouse "models" of sepsis has been documented by more than 20 peer-reviewed scientific publications.[13–17] Some of these studies describe the numerous physiological differences between mice and humans, including variations in genetic responses and differences in immune responses, metabolic responses, and immune susceptibility.[11,13,15,18] Others discuss environmental and external variables that impact study outcomes. For example, the mice used in sepsis experiments are young, inbred, and of the same age and weight, and they live in settings that are mostly free of germs (other than those of their own feces). In contrast, it is mostly infant and elderly humans, who live in a variety of unsterilized, unpredictable environments and frequently have comorbidities such as diabetes or hypertension, who develop sepsis.[9,10,13,18,19] In laboratory settings, pathogens, dosage, and infection are controlled, but in human sepsis, the pathogenic bacteria are often unknown, and patients may not respond to antibiotic treatment when sepsis is caused by more than one type of microbe (polymicrobial).[18] When experimenters induce the condition in mice, the onset of

symptoms occurs within hours to days, whereas it often happens within days to weeks in humans.[14] Mice are not typically provided with the supportive therapy that human patients receive, such as fluids, vasopressors, and ventilators.[9,20,21] Another complicating factor is that, unlike humans, mice are rarely given pain relief.[22,23] This undermines data of already questionable value, as pain affects other physiological processes.

Other animals used in sepsis experimentation

Rats, dogs, cats, pigs, sheep, rabbits, horses, and primates, including baboons and macaques, have also been used in sepsis experimentation around the world. None of these species can reproduce all the physiological features of human sepsis. For example, the pulmonary artery pressure responses of pigs and sheep differ from those of humans, so this aspect of sepsis cannot be compared between species.[24] Pigs also differ from humans in key genes, immune cell types, microbiome composition, and protein expression related to inflammation.[11,25–27]

Baboons, like mice, are less sensitive than humans to the species of bacteria commonly used to induce sepsis in experimental settings.[9] This may be because animals are housed in feces-contaminated environments, allowing them to develop a level of resistance to pathogens that is not present in most humans.[18,20] One study found that rhesus macaques and baboons differ markedly in their innate immune response to pathogens compared to humans. Specifically, macaques and humans show different transcriptomic responses to infection, suggesting distinct regulatory mechanisms during the early stages of immune activation.[28] Additionally, both species differ in the number and composition of leukocytes, and the quantity of B and T cells, limiting their applicability to immunological human sepsis research.[29]

Sepsis induction methods

There are several ways by which experimenters induce sepsis or a sepsis-like condition in non-human animals. These can include infection of one animal with excrement from another, induction of sepsis via pneumonia or other organ infections, or the insertion of a stent allowing an animal's fecal matter to enter their abdominal cavity. The following sections focus on the most commonly used methods: endotoxemia, which involves injection of a toxin, usually lipopolysaccharide (LPS), and cecal ligation and puncture (CLP).

Cecal ligation and puncture

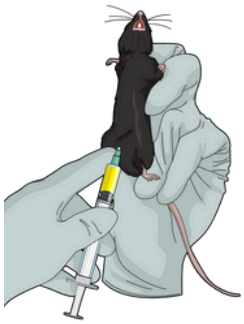
CLP is the most commonly used method for inducing sepsis in mice. Experimenters cut open the animals' abdomens and puncture their intestines with a needle so that fecal matter and bacteria will leak out. The mice then endure widespread pain and eventually become so sick that they are unable to move.

They experience fever, chills, diarrhea, difficulty breathing, lethargy, disorientation, septic shock (when the infection reaches their bloodstream, causing their blood pressure to plummet), which can then lead to, multiple organ failure and death (or euthanasia for moribund animals according to standard guidelines in place in some jurisdictions).[13] Abhorrently cruel, this method is also poor science. First, a mouse's response to CLP varies by age, sex, strain, laboratory, the size of needle used, and the size of the incision, which makes results between laboratories incomparable.[13,20,21,30,31] Second, the procedure can cause the formation of an abscess, whose effects may disguise or be disguised by the effects of the sepsis itself.[20] This means that an intervention that appears to be beneficial for sepsis may actually be beneficial only because of its effects on the abscess.

Endotoxemia does not induce sepsis

In endotoxic models, a mouse or other animal is injected with a bacterial toxin, typically LPS. As described by Riedemann, Guo, and Ward, "LPS, the main component of the Gram-negative bacterial cell wall, was known to stimulate release of inflammatory mediators from various cell types and induce acute infectious symptoms when injected into animals."[32] Endotoxic models have been criticized as a particularly poor model for human sepsis and perhaps not even an accurate model for murine sepsis.

The endotoxic method elicits a rapid and acute inflammatory response in mice, due to their higher resistance to the toxin, necessitating a much larger bacterial load than would be found in a septic human.[10,20] In contrast, sepsis patients typically experience a more prolonged inflammatory response at lower levels.[13] In addition, certain characteristics of human sepsis, such as hemodynamic changes (alterations in blood flow and pressure), never manifest in endotoxic models.[20] These discrepancies have led to skepticism regarding the efficacy of the endotoxic method and whether it genuinely induces sepsis in animals.[33]



Poor ethics in sepsis and trauma experimentation

In 2014, researchers from the University of Alberta surveyed 77 papers on animal studies that had been published in three high-impact critical-care journals between January and June 2012 and found that the "[r]eported … ethical quality" of this research was "poor."[23] The results of the analysis speak for themselves.

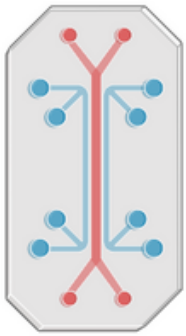
> *"Most studies did not report monitoring the level of anesthesia during invasive procedures, even when muscle paralytics were used, nor monitoring or treatment of expected pain. When euthanasia was used, the method was often not stated, and when stated, most methods were not appropriate for the species. A sample-size calculation was rarely used, and animal numbers were often poorly described. No studies performed a systematic review to ensure that the animal research would be useful and not simple repetition. … Most studies were funded with public funds (foundation or government funding). Sepsis models less often met the composite outcome of … using anesthesia and pain control, and stating the method of euthanasia."*[23]

The authors note that the disregard for the pain and distress experienced by the animals "may confound the study results, and may thus be a reason for the poor translation of [experiments on animals] to humans." In addition, the report states, "Alternatives to animal models were almost never explicitly considered"—even though consideration of non-animal methods is required by laws and policies. The chronic failure of experimenters who conduct sepsis research to adhere to even the minimum standards for the use of animals in laboratories causes animals to suffer, wastes public funds, and impedes a scientifically rigorous search for a treatment for sepsis in humans.

Non-animal methods for studying sepsis

Fortunately, researchers do not need to rely on animals to study and develop treatments for sepsis in humans. In 2015, an expert working group consisting of veterinarians, animal technologists, and scientists issued a report on the implementation of the 3Rs (replacement, reduction, and refinement) of the use of animals in sepsis research.[33] The group noted several methods that could be used instead of animals, such as *in vitro* cell-culture models for studying sepsis mechanisms, systems and computational biology for laying out the inflammatory processes occurring during sepsis, 3-dimensional cell-culture models for exploring human disease progression and infectious disease mechanisms, synthetic human models to recreate human disease-related cell types and tissues, and human genomic information to discover how sepsis affects individuals differently and which groups may be more at risk. The authors state that genomic information "will complement or even replace the need for mouse models in disease discovery and drug development."[33]

Some recent examples of human-relevant sepsis research are discussed below:

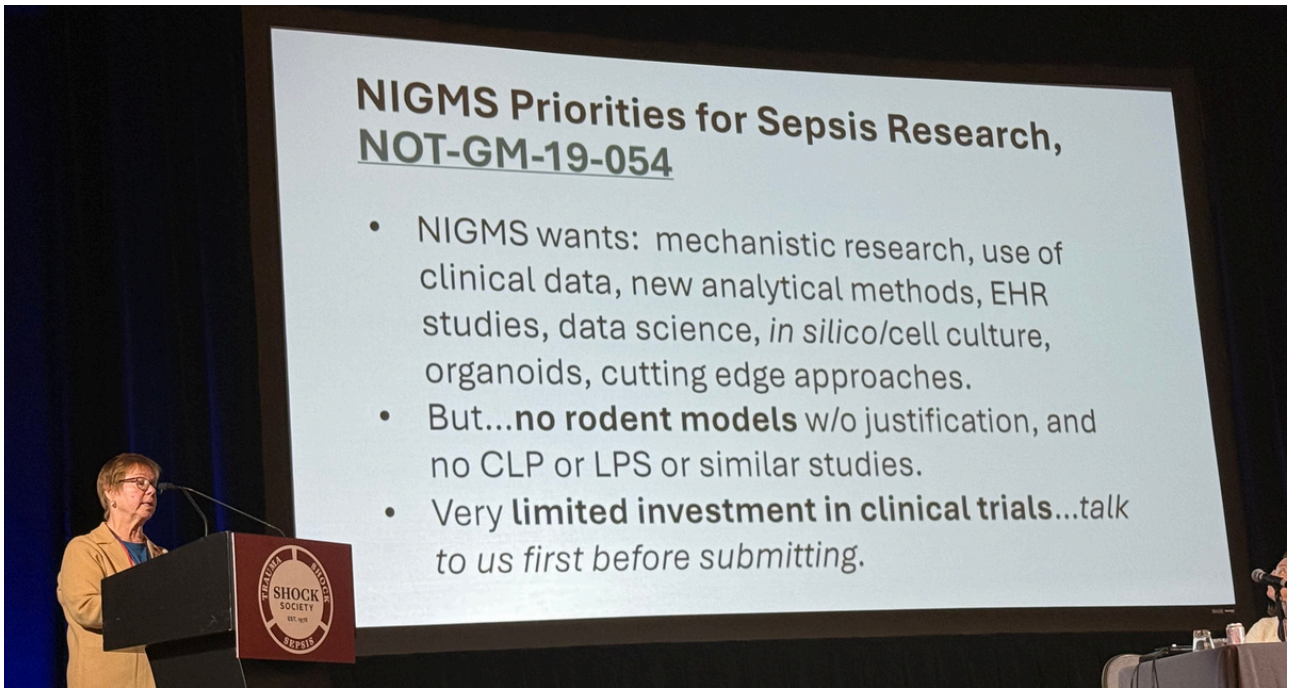


- Scientists in Tokyo used human induced pluripotent stem cell-derived liver organoids to model the pathological progression of sepsis-associated liver dysfunction and recovery following infection.[34]
- At Temple University in the US, a multidisciplinary team identified an association between neutrophil types and the severity of sepsis using a human lung-on-a-chip model, which can be used to determine the appropriate therapeutic intervention based on sepsis severity.[35]
- Researchers in Hefei, China, created a six-unit microfluidic device that comprehensively analyzes a sepsis patient's white blood cell activity to monitor disease progression and severity.[36]
- In the US, Massachusetts General Hospital scientists and physicians created a microfluidic device to accurately detect a biomarker of sepsis pathophysiology using a drop of blood, aiming to improve disease monitoring.[37]
- Because early detection of sepsis is likely the most critical factor in reducing mortality from this condition,[38] researchers around the globe are exploring various artificial intelligence and machine learning tools to aid in the early prediction and diagnosis of sepsis.[39–47]

Shift in sepsis priorities

A 2019 report by the NIH's National Advisory General Medical Sciences Council (NAGMSC) sepsis working group found that, despite decades of research and numerous clinical trials, no new drugs for sepsis had emerged. The report recommended that the NIGMS "rebalance" their sepsis research-funding portfolio to "include a more clinical focus."[48] Following this, NIGMS issued a Notice of Information indicating its intention to support more sepsis research that "uses new and emerging approaches, such as clinical informatics, computational analyses, and predictive modeling in patients, and new applications of high-resolution and high-throughput bioanalytical techniques to materials obtained from septic patients" and called the support of "[s]tudies using rodent models of sepsis" a "low priority."[49] At the 2024 and 2025 Shock Society Annual Conferences, NIGMS officials went further, notifying attendees that the institute does not intend to support research using LPS, CLP, and most other rodent models of sepsis, and it feels strongly that sepsis research should be conducted with human-based methods.[50] NIGMS division director

Dr. Rochelle Long encouraged conference attendees to seek access to clinical samples and collaborate with other researchers using non-animal methods instead of "just going back and doing something in a lab a way that's easy or was done that way in the past."[51]



NIGMS division director Rochelle Long, Ph.D. at the Shock Society Annual Conference; Boston, USA, June 2025.

The US Food and Drug Administration (FDA) has also published a roadmap to reduce animal testing in preclinical studies, acknowledging that the use of animals fails to provide adequate disease models, particularly in disease areas such as inflammatory disease (a disease category that encompasses sepsis). As a result, the FDA will promote the use of human cell-derived organoids and organs-on-chips in disease research.

NIH lawsuit

Because NIH has known—and acknowledged since at least 2013—that mice do not accurately model human sepsis, and because its mandate is to fund research that benefits human health, People for the Ethical Treatment of Animals U.S. (PETA US) challenged the agency's continued funding of animal-based sepsis studies under the U.S. Administrative Procedure Act in 2021. PETA US's case cited the lack of new pharmacological treatments for sepsis despite decades of animal experimentation, and NIH's ongoing support for methods that have repeatedly failed. PETA US's legal team argued that awarding these grants is arbitrary, capricious, an abuse of discretion, not in accordance with the law, and in violation of the agency's legal obligation to reduce the number of animals used in experiments and to minimize their suffering. NIH's motion to dismiss the case was denied and the case is ongoing.

Conclusions

Animals commonly used in sepsis research have significant immunological, genetic, and metabolic differences from humans, leading to poor predictivity. To date, animal experimentation has not produced a single, targeted, effective drug or treatment for sepsis. Despite this high failure rate in translation, researchers continue to subject mice, rats, pigs, and nonhuman primates to painful procedures that result in suffering, organ failure, and death. Sepsis experiments on animals waste public funds, animal lives, and research hours.

But the future of sepsis research can chart a new path. Advanced *in vitro* systems using human cells can reflect human-specific immune responses, cytokine production, and tissue interactions more accurately. Human cell-based systems can also be derived from individual patients, enabling personalized approaches to studying sepsis mechanisms and drug responses. Organ-on-a-chip and other microphysiological systems can simulate key aspects of human physiology, such as tissue interfaces, flow dynamics, and multi-organ interactions. These models increasingly approximate whole-body responses without involving animals. Likewise, computational models can integrate data from various sources to simulate complex sepsis dynamics, offering a whole-system perspective grounded in human data. Using integrated data from multiple human-relevant models can provide better translational value and help overcome the inherent reproducibility and validity issues associated with attempting to model sepsis in animals.

Recommendations for action

Institutional, regional, and national legislative and ethical bodies should prohibit the use of animals in human sepsis research.

Funders should reject grant applications that propose to use animals for sepsis research.

Journals should have a public policy declaring that they will not accept manuscripts that describe sepsis experiments on animals.

References

1. Singer M, Deutschman CS, Seymour CW, et al. The Third International Consensus definitions for sepsis and septic shock (Sepsis-3). *JAMA*. 2016;315(8):801.
2. Rudd KE, Johnson SC, Agesa KM, et al. Global, regional, and national sepsis incidence and mortality, 1990–2017: analysis for the Global Burden of Disease Study. *Lancet*. 2020;395(10219):200-211.
3. Torio CM, Moore BJ. National inpatient hospital costs: the most expensive conditions by payer, 2013. In: *Healthcare Cost and Utilization Project (HCUP) Statistical Briefs*. Agency for Healthcare Research and Quality (US); 2006. Accessed May 15, 2025. http://www.ncbi.nlm.nih.gov/books/NBK368492/
4. National Institute of General Medical Sciences. Sepsis. July 2024. Accessed May 15, 2025. https://www.nigms.nih.gov/education/fact-sheets/Pages/sepsis
5. Sepsis Alliance. Risk factors. Accessed May 15, 2025. https://www.sepsis.org/sepsis-basics/risk-factors/
6. MedlinePlus. Sepsis. November 13, 2023. Accessed May 15, 2025. https://medlineplus.gov/sepsis.html
7. Centers for Disease Control and Prevention. Managing recovery from sepsis. March 7, 2024. Accessed May 15, 2025. https://www.cdc.gov/sepsis/living-with/
8. Obituary: Mitchell P. Fink M.D. *Los Angeles Times*. https://www.legacy.com/us/obituaries/latimes/name/mitchell-fink-obituary?id=16504691. November 26, 2015. Accessed June 24, 2025.
9. Fink MP. Animal models of sepsis. *Virulence*. 2014;5(1):143-153.
10. Verma S. Laboratory animal models to mimic human sepsis: a review. *JZS*. 2016;4(2):34-39.
11. Seok J, Warren HS, Cuenca AG, et al. Genomic responses in mouse models poorly mimic human inflammatory diseases. *Proc Natl Acad Sci U S A*. 2013;110(9):3507-3512.
12. Collins F. Of mice, men, and medicine. NIH. February 19, 2013. Accessed May 15, 2025. https://us.pagefreezer.com/en-US/wa/browse/c530da90-f454-461b-9a86-959c53acb16c?url=https:%2F%2Fdirectorsblog.nih.gov%2F2013%2F02%2F19%2Fof-mice-men-and-medicine%2F&timestamp=2025-05-27T10:13:51Z
13. Rittirsch D, Hoesel LM, Ward PA. The disconnect between animal models of sepsis and human sepsis. *J Leukoc Biol*. 2007;81(1):137-143.
14. Raven K. Rodent models of sepsis found shockingly lacking. *Nat Med*. 2012;18(7):998-998.
15. Timmermans S, Libert C. Learning lessons in sepsis from the children. *Mol Syst Biol*. 2018;14(5):e8335.
16. Osuchowski MF, Ayala A, Bahrami S, et al. Minimum Quality Threshold in Pre-Clinical Sepsis Studies (MQTiPSS): an international expert consensus initiative for improvement of animal modeling in sepsis. *Shock*. 2018;50(4):377.
17. Huet O, De Haan JB. The ethical dimension in published animal research in critical care: the dark side of our moon. *Crit Care*. 2014;18(2):120.
18. Esmon CT. Why do animal models (sometimes) fail to mimic human sepsis? *Crit Care Med*. 2004;32(5 Suppl):S219-222.
19. Laudanski K, Stentz M, DiMeglio M, Furey W, Steinberg T, Patel A. Potential pitfalls of the humanized mice in modeling sepsis. *Int J Inflamm*. 2018;2018:1-9.
20. Buras JA, Holzmann B, Sitkovsky M. Animal models of sepsis: setting the stage. *Nat Rev Drug Discov*. 2005;4(10):854-865.
21. Ward PA. New approaches to the study of sepsis. *EMBO Mol Med*. 2012;4(12):1234-1243.
22. Nemzek JA, Hugunin KMS, Opp MR. Modeling sepsis in the laboratory: merging sound science with animal well-being. *Comp Med*. 2008;58(2):120-128.
23. Bara M, Joffe AR. The ethical dimension in published animal research in critical care: the public face of science. *Crit Care*. 2014;18(1):R15.
24. Redl H, Bahrami S. Large animal models: baboons for trauma, shock, and sepsis studies. *Shock*. 2005;24 Suppl 1:88-93.
25. Chalupova M, Horak J, Kramna L, et al. Gut microbiome diversity of porcine peritonitis model of sepsis. *Sci Rep*. 2022;12(1):17430.
26. Meurens F, Summerfield A, Nauwynck H, Saif L, Gerdts V. The pig: a model for human infectious diseases. *Trends Microbiol*. 2012;20(1):50-57.
27. Mair KH, Sedlak C, Käser T, et al. The porcine innate immune system: An update. *Dev Comp Immunol*. 2014;45(2):321-343.
28. Hawash MBF, Sanz-Remón J, Grenier JC, et al. Primate innate immune responses to bacterial and viral pathogens reveals an evolutionary trade-off between strength and specificity. *Proc Natl Acad Sci U S A*. 2021;118(13):e2015855118.
29. Bjornson-Hooper ZB, Fragiadakis GK, Spitzer MH, et al. A comprehensive atlas of immunological differences between humans, mice, and non-human primates. *Front Immunol*. 2022;13:867015.
30. Ruiz S, Vardon-Bounes F, Merlet-Dupuy V, et al. Sepsis modeling in mice: ligation length is a major severity factor in cecal ligation and puncture. *Intensive Care Med Exp*. 2016;4(1):22.
31. Joffre J. Preclinical model in sepsis: should we abandon the CLP? [Letter]. *J Inflamm Res*. 2023;Volume 16:1757-1759.
32. Riedemann NC, Guo RF, Ward PA. The enigma of sepsis. *J Clin Invest*. 2003;112(4):460-467.
33. Lilley E, Armstrong R, Clark N, et al. Refinement of animal models of sepsis and septic shock. *Shock*. 2015;43(4):304-316.
34. Li Y, Nie Y, Yang X, et al. Integration of Kupffer cells into human iPSC-derived liver organoids for modeling liver dysfunction in sepsis. *Cell Rep*. 2024;43(3):113918.
35. Yang Q, Langston JC, Prosniak R, et al. Distinct functional neutrophil phenotypes in sepsis patients correlate with disease severity. *Front Immunol*. 2024;15:1341752.
36. Yang X, Pu X, Xu Y, et al. A novel prognosis evaluation indicator of patients with sepsis created by integrating six microfluidic-based neutrophil chemotactic migration parameters. *Talanta*. 2024;281:126801.
37. Sakuma M, Wang X, Ellett F, et al. Microfluidic capture of chromatin fibres measures neutrophil extracellular traps (NETs) released in a drop of human blood. *Lab Chip*. 2022;22(5):936-944.
38. Marik PE, Farkas JD. The changing paradigm of sepsis: early diagnosis, early antibiotics, early pressors, and early adjuvant treatment. *Crit Care Med*. 2018;46(10):1690-1692.
39. Goh KH, Wang L, Yeow AYK, et al. Artificial intelligence in sepsis early prediction and diagnosis using unstructured data in healthcare. *Nat Commun*. 2021;12(1):711.
40. Rosnati M, Fortuin V. MGP-AttTCN: An interpretable machine learning model for the prediction of sepsis. *PloS One*. 2021;16(5):e0251248.
41. Honoré A, Forsberg D, Adolphson K, Chatterjee S, Jost K, Herlenius E. Vital sign-based detection of sepsis in neonates using machine learning. *Acta Paediatr Oslo Nor 1992*. 2023;112(4):686-696.
42. Sun B, Lei M, Wang L, et al. Prediction of sepsis among patients with major trauma using artificial intelligence: a multicenter validated cohort study. *Int J Surg Lond Engl*. 2025;111(1):467-480.
43. Gao J, Lu Y, Ashrafi N, Domingo I, Alaei K, Pishgar M. Prediction of sepsis mortality in ICU patients using machine learning methods. *BMC Med Inform Decis Mak*. 2024;24(1):228.
44. Hang Y, Qu H, Yang J, et al. Exploration of programmed cell death-associated characteristics and immune infiltration in neonatal sepsis: new insights from bioinformatics analysis and machine learning. *BMC Pediatr*. 2024;24(1):67.
45. Boussina A, Shashikumar SP, Malhotra A, et al. Impact of a deep learning sepsis prediction model on quality of care and survival. *NPJ Digit Med*. 2024;7(1):14.
46. Giacobbe DR, Signori A, Del Puente F, et al. Early detection of sepsis with machine learning techniques: a brief clinical perspective. *Front Med*. 2021;8:617486.
47. Steinbach D, Ahrens PC, Schmidt M, et al. Applying machine learning to blood count data predicts sepsis with ICU admission. *Clin Chem*. 2024;70(3):506-515.
48. NAGMSC. NAGMSC Working Group on Sepsis final report. May 17, 2019. Accessed May 15, 2025. https://www.nigms.nih.gov/sites/nigms/files/migrated/nagmsc-working-group-sepsis-report.pdf
49. National Institute of General Medical Sciences. Notice of information: NIGMS priorities for sepsis research. July 29, 2019. Accessed May 15, 2025. https://grants.nih.gov/grants/guide/notice-files/NOT-GM-19-054.html
50. Hays A. Major health agency slashes funding for sepsis experiments on animals after push from PETA. PETA. June 18, 2024. Accessed May 15, 2025. https://www.peta.org/blog/major-health-agency-slashes-funding-for-sepsis-experiments-on-animals/
51. Science Advancement and Outreach. SAO at Shock Con 2025. LinkedIn. June 9, 2025. Accessed June 16, 2025. https://www.linkedin.com/pulse/sao-shock-con-2025-petasao-y9ebe/