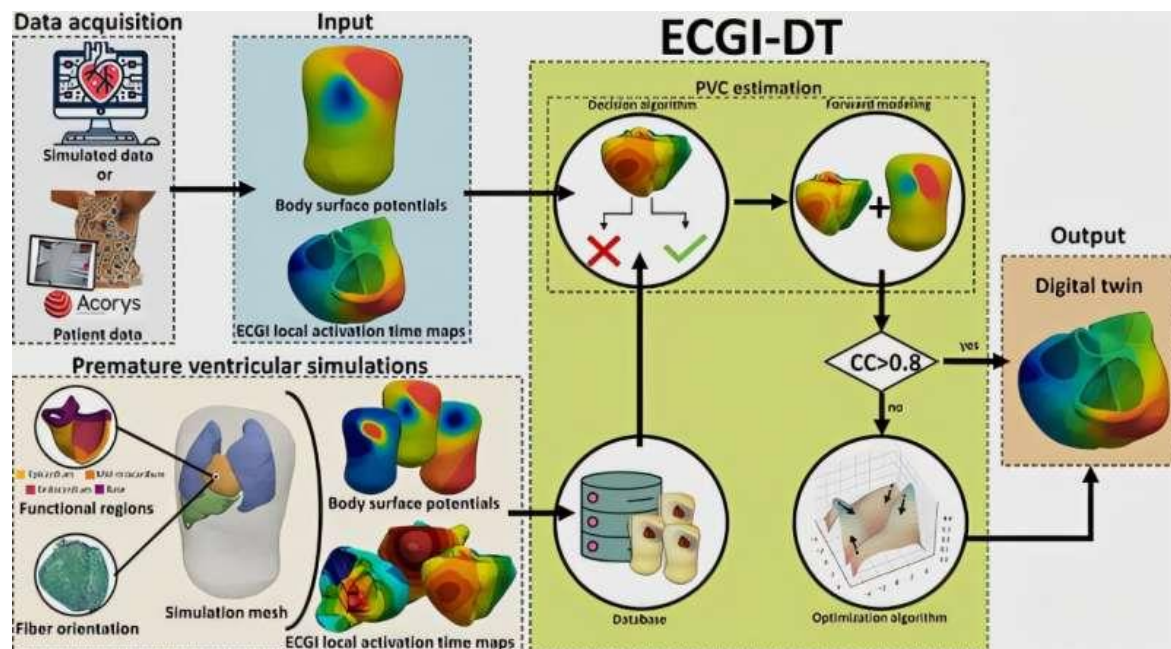


MEDICAL RESEARCH VOL 7

MARCH 31, 2025

Digital twin of the heart helps to improve the diagnosis and treatment of cardiac arrhythmias

by [Technical University of Valencia](#)



Credit: Technical University of Valencia

Millions of people around the world live with cardiac arrhythmias. Only in Spain, it is estimated that more than one million citizens suffer from them. Detecting and treating them accurately continues to be one of the great challenges of cardiovascular medicine.

ADVERTISING

Now, a team from the Universitat Politècnica de València (UPV), belonging to the COR-ITACA group, has launched a new noninvasive method to locate the origin of premature ventricular contractions (PVC), one of the most frequent [cardiac arrhythmias](#). The method combines electrocardiographic imaging (ECGI) with digital twins of the heart, which helps to improve the accuracy with which the focus where these arrhythmias originate can be identified.

The Gregorio Marañón General University Hospital, the spin-off Corify Care S.L., the Complutense University of Madrid, the Autonomous University of Barcelona, the Center for Biomedical Research Network in Cardiovascular Diseases (CIBERCV) and the Institute of Biomedical Engineering of the Karlsruhe Institute of Technology (Germany) have also participated in its development. Their work has been [published](#) in the journal *Computers in Biology and Medicine*.

Next week, the COR-ITACA team and the company Corify Care will present these results at the [EHRA 2025](#) (European Heart Rhythm Association) Congress.

Currently, conventional electrocardiography (ECG) is used to detect premature ventricular contractions (PVCs). However, as explained by the COR-ITACA group of the UPV, its results can be limited by anatomical differences between patients.

"Electrocardiographic imaging offers a more detailed view, but it also has certain limitations in accurately determining the exact point of origin of the arrhythmia. And this is what we achieve with our new method," says Andreu Climent.

The system, devised by the team of Spanish and German researchers and clinicians, integrates ECGI with personalized heart simulations. "With this, we have created a 'digital twin' capable of reproducing the electrical activity of the myocardium more faithfully," adds María S. Guillem.

How does it work?

To create the new method, the research team generated a database of more than 600 simulations of cardiac arrhythmias using detailed anatomical models of the torso and the heart. Using these simulations, they developed an algorithm that allows the arrhythmia focus to be located with an average accuracy of 7.8 mm, compared to the more than 30 mm error recorded with standard ECGI, all in a completely non-invasive way.

In addition to the simulations, the new method was applied in a real clinical case of a patient with an arrhythmia localized in the free wall of the left ventricle. The model based on ECGI and digital twins achieved an estimation of the focus with an error of 15.5 mm, well below the error of 36.7 mm recorded with conventional ECGI.

"Our method can facilitate planning interventions, such as catheter ablation, by reducing the need for invasive scans and shortening intervention times. It could be integrated as a support tool in the preoperative planning of ablations. And it would be especially useful in complex cases where other techniques are more limited, such as in arrhythmias originating in the septum or at the base of the ventricle," adds Jorge Sánchez.

EPICA+: a step forward in cardiac diagnosis

The COR-ITACA group at the UPV is currently leading a new project called EPICA+, whose ultimate goal is to apply this method to many more arrhythmias and to apply it in clinical

practice. To do this, they will integrate electrocardiographic images (ECGI) with [artificial intelligence](#) and digital heart twins. "The combination of these three technologies will help to improve the diagnosis and treatment of cardiac arrhythmias," says Guillem, project coordinator together with Climent.

The project brings together a multidisciplinary team of engineers, clinicians and data scientists, who will draw on a database of more than 1,000 patients and 20,000 computational models. Clinical trials involving 144 patients will validate the technology with the aim of doubling treatment success rates and reducing costs.

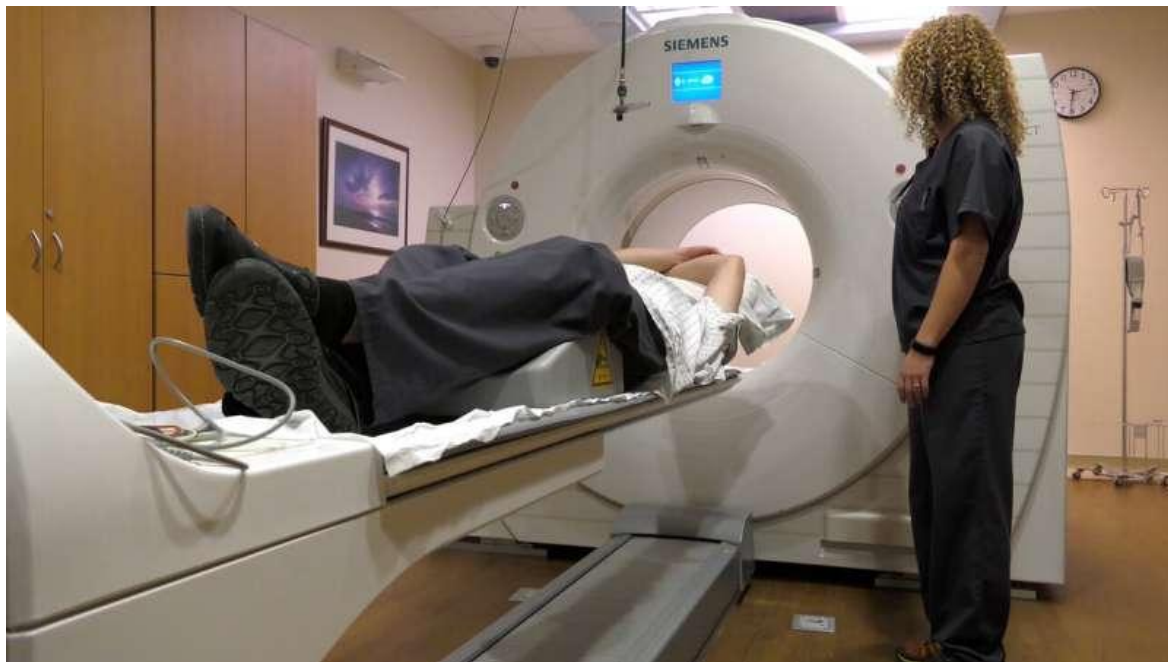
"EPICA+ represents a significant milestone in Spanish research. This project not only addresses one of the greatest challenges in cardiovascular medicine but also highlights the impact that integrating advanced technologies, such as artificial intelligence and computational modeling, can have on improving health care," says Guillem.

More information: Jorge Sánchez et al, Enhancing premature ventricular contraction localization through electrocardiographic imaging and cardiac digital twins, *Computers in Biology and Medicine* (2025). DOI: [10.1016/j.combiomed.2025.109994](https://doi.org/10.1016/j.combiomed.2025.109994)
Provided by [Technical University of Valencia](#)

MARCH 31, 2025

Low coronary artery calcium score associated with excellent prognosis regardless of age: New study

by Intermountain Healthcare



A large new study of more than 40,000 patients from heart researchers at Intermountain Health in Salt Lake City finds that a zero coronary artery calcium score continues to be an accurate indicator of a low risk for a coronary heart event even as a person ages. Moreover, researchers found that patients with a zero coronary artery calcium score also had a two to three times lower risk of death from any cause at any age, including in seniors. Credit: Intermountain Health

Having a coronary artery calcium (CAC) score of zero has generally been accepted as a marker of a very low risk of having a cardiac event within the next five years. However, age is a strong contributor to coronary risk, with risk increasing markedly as people age.

Whether age-related risk factors diminish the low risk predicted by a zero [coronary artery calcium](#) score has been uncertain—until now.

A large new study of more than 40,000 patients by heart researchers at Intermountain Health in Salt Lake City finds that a zero coronary artery calcium score continues to be an accurate indicator of a low risk for a coronary heart event even as a person ages.

Moreover, researchers found that patients with a zero coronary artery calcium score also had a two to three times lower risk of [death](#) from any cause at any age, including in seniors.

The findings of the new study are important because as cholesterol-laden plaques in the heart's coronary arteries age, they attract calcium.

"We found that a zero-plaque burden is not only highly prognostic of good heart health at any age, but also of excellent overall survival," said Jeffrey L. Anderson, principal investigator of the study and distinguished clinical and research physician at Intermountain Health. "Our findings show that having little or no coronary plaque predicts that people will live longer, healthier lives, including into their golden years."

A coronary artery calcium score of zero indicates essentially no advanced coronary plaques. A score of 1–99 indicates a mild case of plaque, a score of 100–299 a moderate case, and a score of 300 or more a high case.

The risk of a coronary event in the overall population generally parallels the coronary artery calcium plaque burden, said Dr. Anderson.

In the retrospective study, Intermountain Health researchers identified 40,820 symptomatic patients over a 10-year period who were at primary coronary risk and who underwent a PET/CT scan, which quantifies advanced, calcified plaque in the vessels of the heart.

Of that overall group, 8,170 patients had a coronary artery calcium score of zero, with 5,185 of those patients under age 65, and 2,782 patients 65 or older. They then assessed coronary prognosis during an average follow-up of over 2 years.

Intermountain researchers found that a zero coronary artery calcium score was predictive of a very low risk of coronary death or non-fatal heart attack and of death of any cause. These results were presented at the American College of Cardiology's annual scientific sessions conference ([ACC.25](#)) in Chicago on March 29.

Coronary events occurred in only 0.12% of younger patients and 0.25% of [older patients](#), a non-significant difference. Comparing overall death or heart attack rates in those with the presence of coronary artery calcium to CAC-zero patients showed two- to three-fold lower event rates with zero coronary artery calcium scores in both older and younger age groups, demonstrating the value of a zero coronary artery calcium score at any age.

"If you have disease in your coronary arteries, you may also have vascular complications in other organs of your body," said Dr. Anderson. "Given these results, a zero coronary artery calcium score may also be a predictor of not only very low coronary death but also of a lower risk of other causes of death."

The next step in the research is to further explore the relationship between a zero coronary artery calcium score and all-cause death, including vascular diseases in other organs and even cancer," Dr. Anderson added.

Provided by Intermountain Healthcare

APRIL 1, 2025 [REPORT](#)

The GIST
Editors' notes

Implant-derived metals found in cerebrospinal fluid

by Justin Jackson , Medical Xpress

Research led by Charité–Universitätsmedizin Berlin has found that metal particles from artificial joint implants can enter the central nervous system and accumulate in cerebrospinal fluid, raising concerns about potential neurological effects.

Joint replacement surgery has transformed orthopedic care, improving mobility and quality of life for millions of people. Modern implants, made from combinations of metals, are designed for durability and biocompatibility.

Over time, wear and corrosion of these materials can release microscopic particles into surrounding tissue. These byproducts have been linked to problems near the [implant](#) site, including inflammation, tissue damage, and loosening of the joint.

Emerging concerns point to the possibility of metal particles entering the bloodstream and affecting organs far from the implant. Case reports have described serious effects on the heart, thyroid, and nervous system in patients with elevated levels of certain metals, particularly cobalt and chromium. Neurological changes have been reported in some patients following joint replacement.

Previous research has largely focused on these two metals and has relied on blood and serum measurements, leaving open the question of whether such particles reach the [central nervous system](#).

In the study, "Metal Concentrations in Blood and Cerebrospinal Fluid of Patients With Arthroplasty Implants," [published](#) in *JAMA Network Open*, researchers conducted a single-site cross-sectional study to determine whether metals from joint implants can be found in cerebrospinal fluid and bloodstream.

A cohort was assessed of 204 adult participants, 102 with an existing large joint implant (median age 71.7) and 102 in a [control group](#) that had never received [joint replacement surgery](#) (median age 67.2).

Samples were collected during elective surgery under spinal anesthesia or during lumbar puncture for routine diagnostic or therapeutic reasons. Inductively coupled plasma mass spectrometry measured concentrations of ten metals in blood, serum, and cerebrospinal fluid, including cobalt, chromium, titanium, niobium, zirconium, and others known to be used in implant materials.

Cobalt levels in cerebrospinal fluid were significantly higher in patients with joint implants than in matched controls. Median cobalt concentrations were 0.03 µg/L in the implant group and 0.02 µg/L in the control group. Strong correlations were observed between cobalt levels in cerebrospinal fluid and those in serum and whole blood, suggesting systemic exposure may be reaching the central nervous system.

Patients with implants also exhibited higher levels of chromium, titanium, niobium, and zirconium in blood and serum. In cerebrospinal fluid, titanium, niobium, and zirconium levels were significantly elevated, but only when serum levels of these metals were also increased. This is an important finding as it supports the accuracy of less invasive blood sampling as an indicator of possible cerebrospinal fluid inundation.

Patients with implant components containing cobalt-chromium-molybdenum alloys had the highest cerebrospinal fluid concentrations of both cobalt and chromium. Cobalt levels in cerebrospinal fluid were significantly elevated even among patients with implants in place for less than ten years. Pain in the joint containing the implant was also associated with higher cobalt levels in cerebrospinal fluid.

No increase in cerebrospinal fluid metal levels was observed in patients with implants lacking cobalt-chromium-molybdenum components. Patients with cemented implants showed elevated levels of zirconium in blood and serum, though not in cerebrospinal fluid. Aluminum did not appear elevated in the implant group despite being present in certain implant alloys.

Blood-brain barrier integrity, assessed by serum S-100B levels, appeared unaffected and uncompromised in the implant group. Among those with elevated cerebrospinal fluid cobalt or zirconium, serum S-100B levels were lower than in matched controls.

Findings indicate that [metal particles](#) released from joint implants can accumulate in the central nervous system, especially those containing cobalt-chromium-molybdenum.

Results suggest that arthroplasty-related metal exposure is not confined to local tissues but extends systemically and may involve the brain. While blood-brain barrier dysfunction was not evident, the presence of these metals in [cerebrospinal fluid](#) raises questions about long-term neurological safety.

Further research is needed to determine whether the observed exposures can contribute to cognitive changes or neurodegenerative disease, particularly in those with cobalt-containing components or unexplained neurological symptoms following arthroplasty. As a pilot study with exploratory objectives, no causal link can be established from these data alone.

More information: Anastasia Rakow et al, Metal Concentrations in Blood and Cerebrospinal Fluid of Patients With Arthroplasty Implants, *JAMA Network Open* (2025). DOI:

[10.1001/jamanetworkopen.2025.2281](https://doi.org/10.1001/jamanetworkopen.2025.2281)

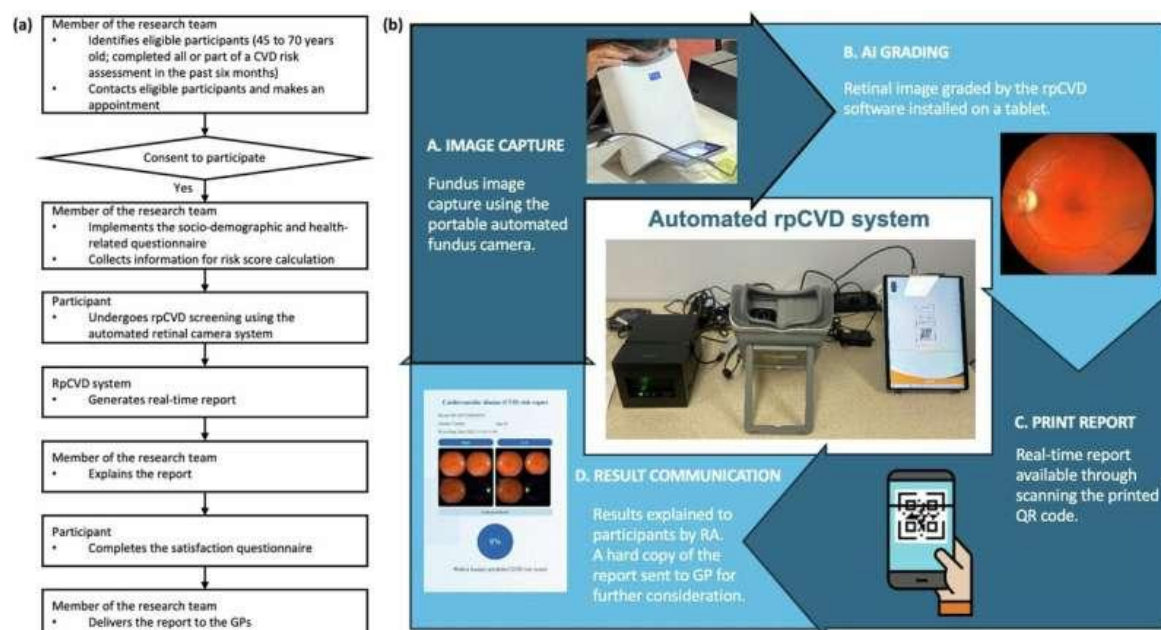
Journal information: *JAMA Network Open*

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APRIL 1, 2025

AI eye scans could help doctors identify heart disease and stroke risk

by Centre for Eye Research Australia



Overview of the study protocol and system. Credit: *npj Digital Medicine* (2025). DOI: [10.1038/s41746-025-01436-1](https://doi.org/10.1038/s41746-025-01436-1)

A new study has for the first time tested the use of an artificial intelligence–powered eye scan in general practice clinics to screen patients for cardiovascular disease.

Results of the research, [published](#) in *npj Digital Medicine*, found that the fast, noninvasive AI retinal scan could be readily integrated into primary care settings to screen for risk of heart attacks and stroke.

"The ease of use of the retinal camera and high acceptance by doctors and patients demonstrates that it could be integrated into clinic workflow to screen patients before appointments with their GP," says the study's first author Wenyi Hu.

"However, more work needs to be done to improve accuracy in some groups of patients, particularly men over 60."

About the study

Hu conducted the research as part of her University of Melbourne Ph.D. studies in CERA's Ophthalmic Epidemiology team, along with colleagues including her supervisor Associate Professor Lisa Zhuoting Zhu.

The research involved 361 people, aged 45–70, who were patients at two GP clinics and had received a full or partial cardiovascular risk assessment—such as a blood pressure or cholesterol test—in the previous six months.

Each patient had an eye scan taken with a desktop retinal camera, which scanned the blood vessels at the back of the eye, and received a real-time AI-generated report of their [cardiovascular disease](#) risk.

They were also assessed using a widely utilized clinical risk tool—World Health Organization's CVD risk chart—which considers age, sex, smoking status, [blood pressure](#), diabetes and total cholesterol.

The risks calculated from [retinal images](#) were then compared against the World Health Organization's CVD risk chart to measure the correlation between the two methods.

Both scoring approaches were then validated against more than 27,500 records from the UK Biobank, revealing that the retinal scan-based scores demonstrated similar accuracy to WHO CVD risk scores in predicting risk of heart attack or stroke over 10 years.

The patients and eight GPs from the participating clinics were also surveyed on their satisfaction with the technology and likelihood of using it in the future.

The study found:

- Moderate correlation between the accuracy of the retinal scan and the WHO risk scores—with 67.4% of participant results matching, 17.1% having their risk overestimated by the retinal scan and 19.5% having their risk underestimated.
- Similar ability between the retinal scan and WHO method to predict 10-year risk of coronary heart disease or stroke when compared to data from the UK Biobank

- A 93.9% imaging success rate, with the most patients receiving a retinal scan which could be graded for cardiovascular risk
- 92.5% of patients and 87.5% of GPs were satisfied with the technology.

General practitioner involvement

Camberwell general practitioner Dr. Malcolm Clark's clinic took part in the research and is a co-author on the paper.

He says retinal scanning has great potential to increase the number of Australians who are assessed for cardiovascular risks.

Dr. Clark says in the future retinal scanning could be used as an early triaging tool to alert GPs to patients who need further investigation or tests.

"In the future, I would see patients receiving an SMS, reminding them to have an eye scan which would send a report on their risk to their doctor, who could then follow up with further tests if needed.

"This could become part of regular health screenings like a cervical screening test or a fecal occult blood test."

A broader vision for clinical AI

Associate Professor Zhu says the integration of AI-based eye scans into general practice workflows marks a significant step toward precision public health.

"We are building a future pathway that could offer low-cost, scalable and equitable cardiovascular screening for everyone, including those in remote and underserved communities," she says.

"AI-powered eye checks could also provide insights into brain and kidney health and part of routine preventative health care."

More information: Wenyi Hu et al, Real-world feasibility, accuracy and acceptability of automated retinal photography and AI-based cardiovascular disease risk assessment in Australian primary care settings: a pragmatic trial, *npj Digital Medicine* (2025). DOI: [10.1038/s41746-025-01436-1](https://doi.org/10.1038/s41746-025-01436-1)

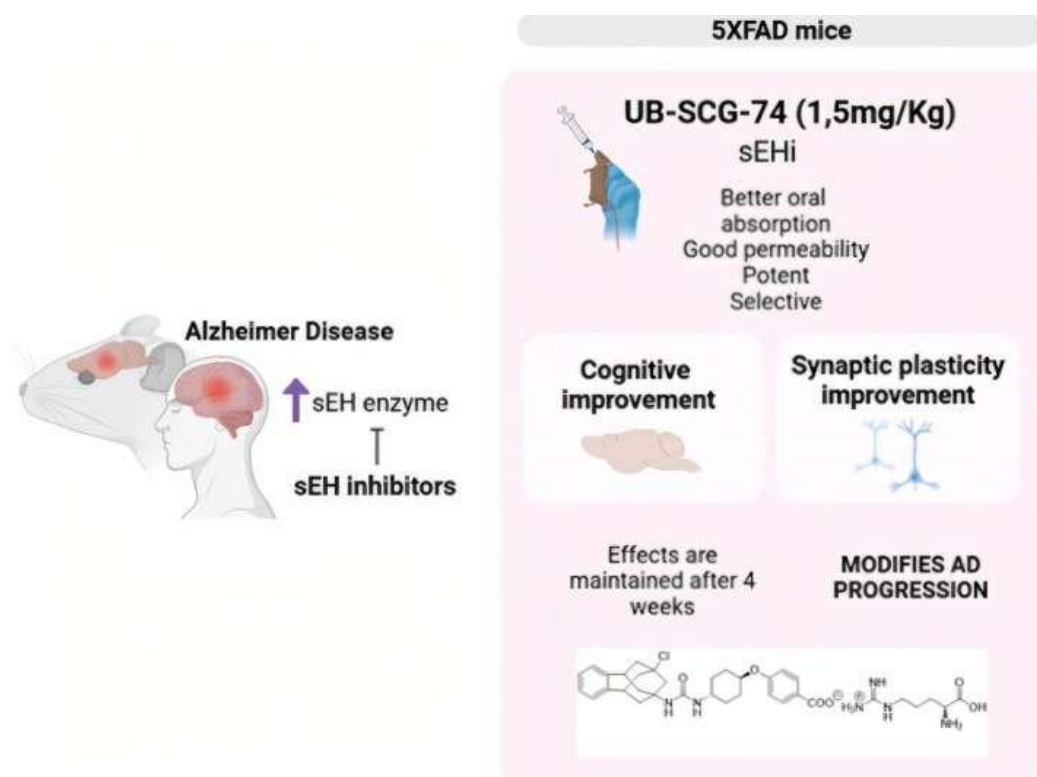
Journal information: [npj Digital Medicine](#)

Provided by Centre for Eye Research Australia

APRIL 1, 2025

Novel Alzheimer's drug candidate reduces inflammation and protects neurons

by [University of Barcelona](#)



Credit: *ACS Pharmacology & Translational Science* (2025). DOI: 10.1021/acsptsci.4c00629
Alzheimer's disease, the most common cause of dementia, is currently incurable. The current drugs available have very limited efficacy and only in mild stages of the disease. A team from the University of Barcelona has developed a promising therapeutic candidate to treat this disease, which affects more than 800,000 people in Spain.

According to their study [published](#) in the journal *ACS Pharmacology & Translational Science*, the new drug has been shown to have neuroprotective and anti-inflammatory effects in two murine models of the disease. The patent for the compound has been licensed to a pharmaceutical company to start the preclinical and [clinical trials](#) necessary for its approval.

This study was led by Mercè Pallàs, from the UB's Faculty of Pharmacy and Food Sciences and the Institute of Neurosciences (UBNeuro), and Santiago Vázquez, from the same faculty and the UB Institute of Biomedicine (IBUB) in collaboration with UB researchers Christian Griñán Ferré, Julia Jarne Ferrer, Javier Sánchez and Sandra Codony. Experts from

the Institute of Biomedical Research of Barcelona (IIBB)—a CSIC and the August Pi i Sunyer Biomedical Research Institute (IDIBAPS) center, the Neurodegenerative Diseases Area of the Center for Biomedical Research Network (CIBERNED) and the University of Bonn (Germany) have also participated.

A new approach against neuroinflammation

The study is the culmination of seven years of research in which researchers have used a new approach based on the inflammatory processes that contribute to triggering the disease and modulating its progression.

"Strategies that have been tried unsuccessfully over the past 10 years have specifically targeted beta-amyloid accumulation and plaque formation in the brain, but there is evidence that neuroinflammation is a major cause of Alzheimer's disease. Addressing [inflammatory processes](#) has therefore become a promising therapeutic strategy," the authors explain.

The new compound is an inhibitor of soluble epoxide hydrolase (sEH), an enzyme involved in the regulation of several physiological processes, including inflammation and pain response. "In the context of Alzheimer's disease, inhibition of this enzyme can increase levels of epoxyeicosatrienoic acids (EETs)—bioactive molecules that are endogenous anti-inflammatories—and thus reduce neuroinflammation and promote neuroprotection," explains Pallàs, who is also a researcher at CIBERNED.

The results of the study show that treatment with the new compound had neuroprotective effects in two mouse models of Alzheimer's disease, culminating in improved spatial and working memory and an improved neural network. "This could help preserve [neuronal function](#) and reduce neuronal death associated with Alzheimer's disease," says Vázquez.

These neuroprotective effects are due to the increase in EET acids, which are also involved in improving [cerebral blood flow](#), which is crucial for maintaining brain health. "Inhibition of sEH may therefore contribute to improved cerebral perfusion and protection against ischemic damage," the researchers note.



From left to right, Mercè Pallàs and Santiago Vázquez. Credit: University of Barcelona

Simultaneous effect on several anti-inflammatory pathways

The advantage of this new drug over other anti-inflammatory compounds—which have failed in clinical trials and have not reached patients due to ineffectiveness—is that TSE augmentation has been shown to reduce transcription and levels of multiple pro-inflammatory markers while enhancing anti-inflammatory cytokines.

"This global approach, which affects several inflammatory pathways simultaneously rather than acting on just one, results in a neuroprotective effect that is sufficient to ameliorate the symptomatology and pathology of Alzheimer's disease," the researchers stress.

In fact, in the study, the authors show that the compound is effective compared to ibuprofen—an anti-inflammatory drug—whose action is of little significance in the animal model of familial Alzheimer's disease in which it has been tested. For the researchers, this is a "very important" competitive advantage over other potential drugs.

Modifying disease progression

The study also shows that treatment with the sEH inhibitor not only prevents the progression of the disease, but is able to modify its course. According to the researchers, the treatment maintains the cognitive-enhancing effects even one month after the drug is stopped in the mice.

"More importantly, the integrity of the neuronal network and the dendrite count (a component of neurons) were preserved, suggesting that the treatment is not only symptomatic, but modifies disease progression in the mouse model."

Clinical translation challenges

Despite these encouraging results, the road to clinical application of this compound is still long and complex. The development of a new drug involves a rigorous process involving multiple stages: from initial research and pre-clinical testing to clinical trials with patients and, finally, approval by health authorities. During this process, the drug must pass stringent safety and efficacy regulations, a requirement that often requires decades of research and considerable financial investment.

In this sense, the patent for the compound has been licensed to a U.S. pharmaceutical company to promote its progress in the preclinical and clinical trials phase. Members of the research team, led by professors Vázquez and Pallàs, are following the project within the company as expert consultants.

"This collaboration will allow us to continue participating, as the UB, in the development of the new compound for the treatment of pathologies related to neuroinflammation," state the researchers.

More information: Júlia Jarne-Ferrer et al, Novel Soluble Epoxide Hydrolase Inhibitor: Toward Regulatory Preclinical Studies, *ACS Pharmacology & Translational Science* (2025). DOI: [10.1021/acsptsci.4c00629](https://doi.org/10.1021/acsptsci.4c00629)

Journal information: [ACS Pharmacology & Translational Science](#)

Provided by [University of Barcelona](#)

APRIL 1, 2025

Omega-3s have potential to counteract THC effects on fetal development, study finds

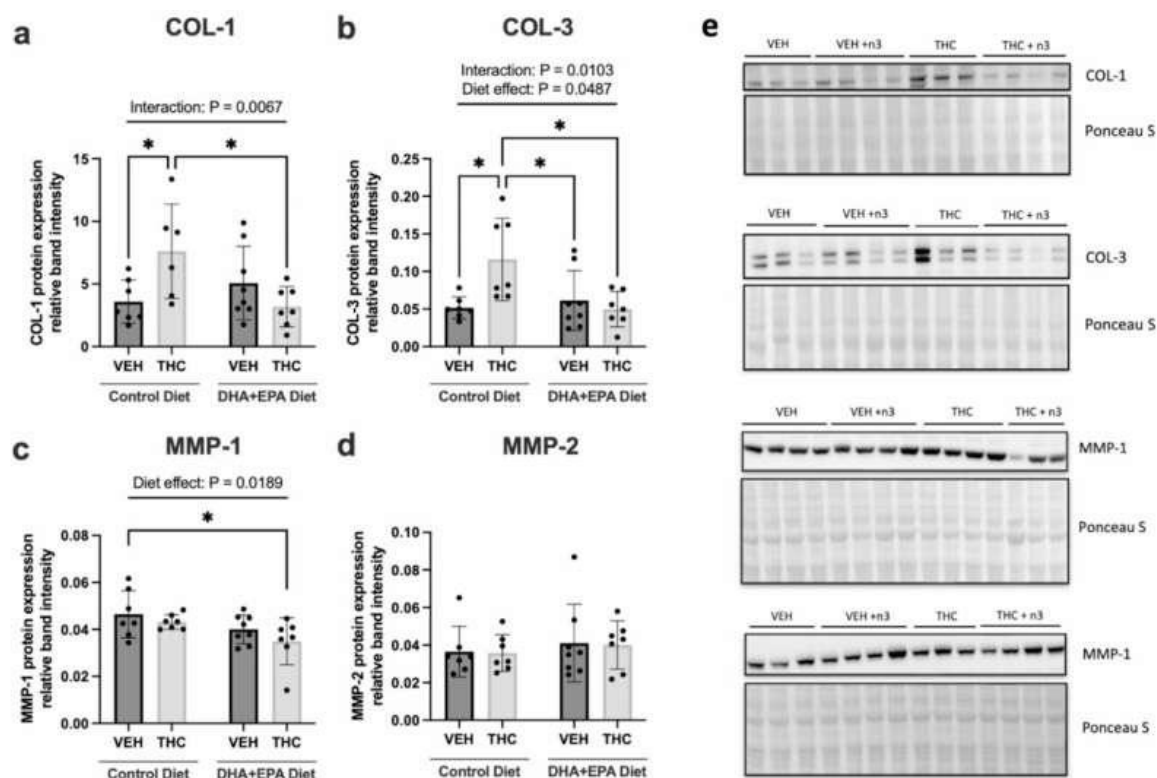
by Cynthia Fazio, [University of Western Ontario](#)

Previous studies show exposure to Tetrahydrocannabinol (THC)—the main psychoactive component in cannabis—while in utero can lead to lower birth weight and potential heart complications in newborn animal offspring. For the first time, researchers from Western's Schulich School of Medicine & Dentistry have found a potential way to prevent those effects.

A recent study, [published](#) in the journal *Scientific Reports*, is the first to uncover a potential intervention to remedy the decline in birth weight and compromised heart health in offspring due to THC exposure in utero. The study was led by Schulich Medicine & Dentistry researchers Dan Hardy and Kendrick Lee.

Using rat models, the researchers found heart deficits associated with THC were counteracted by Omega-3 supplementation. In this study, researchers were able to replicate the results from a 2019 study in which they demonstrated exposure to THC decreases stroke volume (volume of blood pumped out from the [left ventricle](#)), [cardiac output](#) (amount of blood your heart pumps out per minute) and [ejection fraction](#) (amount of blood your heart pumps each time it beats) in rat offspring.

"When we gave the animal models an Omega-3 diet during pregnancy and for three weeks postpartum, we found it prevented low birth weight outcomes," said Hardy, the study's lead author and professor in the departments of obstetrics and gynecology and physiology and pharmacology. "More importantly, it prevented the early decrease in cardiac function."



Maternal DHA + EPA significantly reduced collagen content in offspring hearts exposed to THC in gestation. Credit: *Scientific Reports* (2025). DOI: 10.1038/s41598-025-92844-6

According to Hardy, an Omega-3 diet consists of two important fatty acids, DHA and EPA, which play key roles in supporting fetal growth and heart health. Omega-3 has been proven to be a beneficial supplement during pregnancy in preventing [gestational diabetes](#) and protecting fetal development. It's also a feasible intervention as Omega-3 is widely accepted and available.

"These results were promising as they show that it is possible to prevent those long-term [adverse outcomes](#) in the offspring," said Lee, the study's first author who conducted this research while completing his Ph.D. at Western.

"Ultimately, the best way to prevent the adverse cardiac health outcomes in [offspring](#) is to stop using cannabis, but this study raises the exciting possibility that an Omega-3 diet could be beneficial to those children, whom without choice, were exposed to cannabis in utero."

More information: Kendrick Lee et al, Maternal dietary DHA and EPA supplementation ameliorates adverse cardiac outcomes in THC-exposed rat offspring, *Scientific Reports* (2025). DOI: [10.1038/s41598-025-92844-6](https://doi.org/10.1038/s41598-025-92844-6)

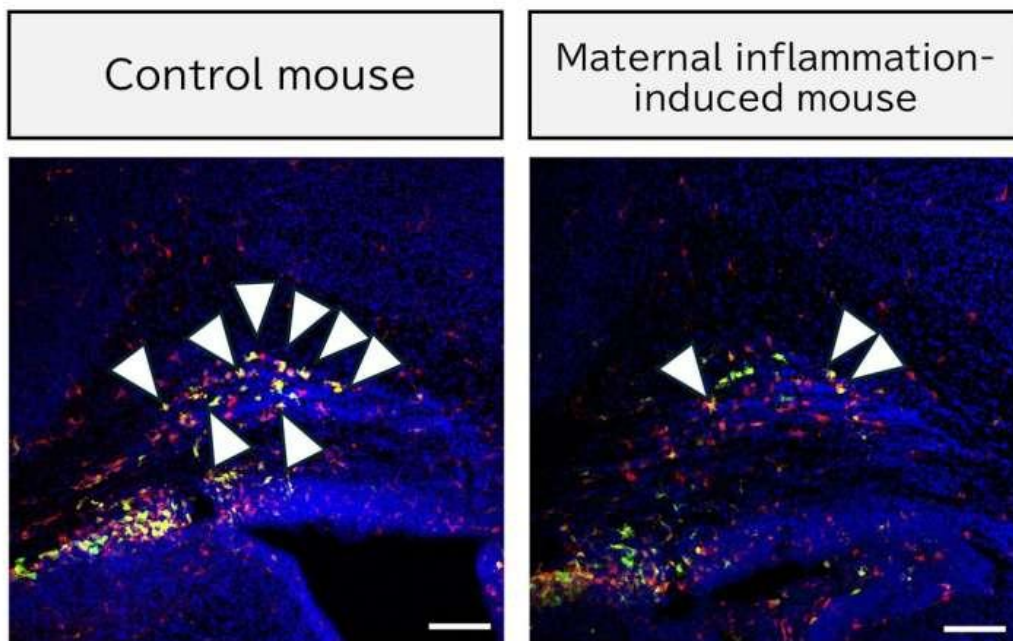
Journal information: [Scientific Reports](#)

Provided by [University of Western Ontario](#)

APRIL 1, 2025

An immune cell may explain how maternal inflammation causes neurodevelopmental disorder

by [Nagoya University](#)



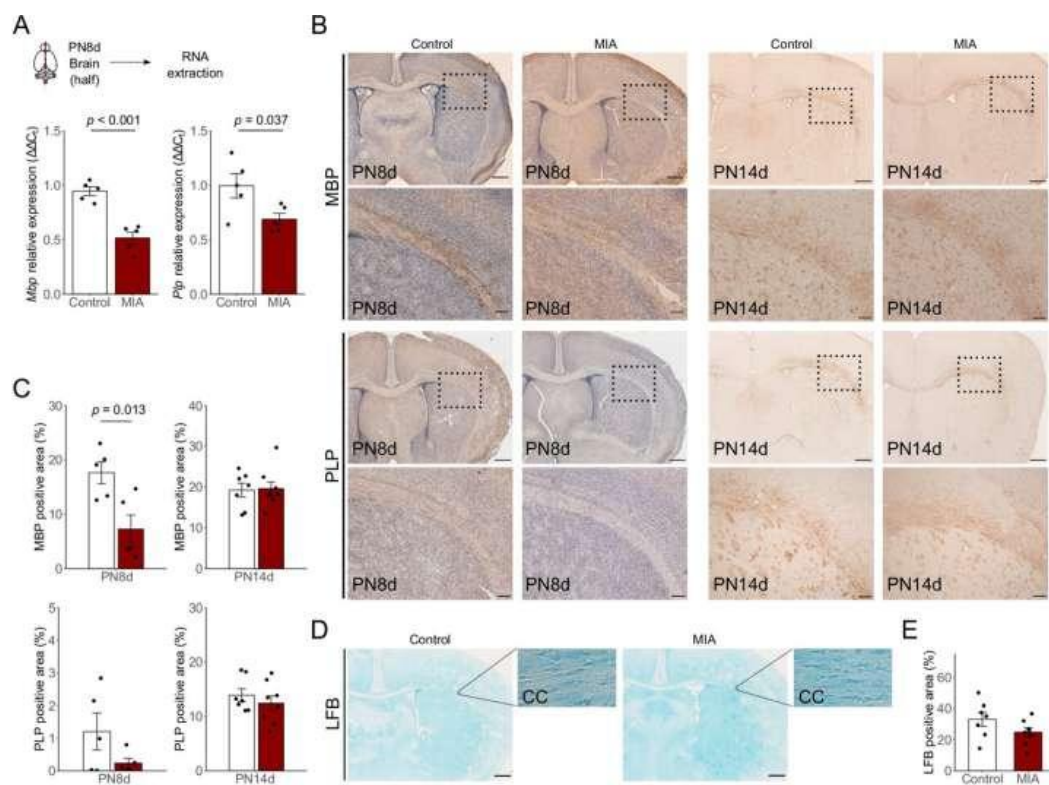
In neonatal mice exposed to maternal inflammation, the proportion of CD11c-positive microglia (yellow) was reduced compared to control mice. Microglia are shown in red, and CD11c-positive cells in green. Scale bar = 100 μ m. Credit: Kohei Nomaki

A research group led by Nagoya University Graduate School of Medicine in Japan has uncovered a potential mechanism linking maternal inflammation to delayed neurodevelopment in infants. The research suggests the role of CD11c-positive microglia—immune cells in the brain crucial for myelination—during infant brain development. The results, [published](#) in *Communications Biology*, suggest new strategies to mitigate the long-term neurodevelopmental effects of maternal inflammation.

Inflammation during pregnancy occurs when the mother's immune system becomes activated during pregnancy, typically due to an infection, autoimmune response, or environmental factors. It can have negative outcomes for the baby, potentially causing long-term cognitive and behavioral challenges.

Now, a research team led by Kazuya Fuma and Tomomi Kotani has discovered the role of CD11c-positive microglia in this process. Microglia, the brain's **immune cells**, play a key role in **myelination**, the process where **nerve fibers** are coated with myelin. Myelin is essential for making the **nerve cells** able to transmit electrical signals efficiently.

First, the researchers tested mice that were exposed to maternal inflammation. They found that the proliferation of these cells was reduced. Then, to determine if these findings were relevant to humans, the researchers analyzed cord blood from **preterm infants** exposed to chorioamnionitis, a condition that causes inflammation during pregnancy. They found lower levels of IGF-1, a protein linked to CD11c-positive microglia, in their samples. When they performed MRI of the infants, the scans confirmed that they had a higher incidence of delayed myelination.



Comparison of myelination-related mRNA and protein. Credit: *Communications Biology* (2025). DOI: 10.1038/s42003-025-07511-3

"Inflammation during pregnancy suppressed the increase in CD11c microglia that we usually see during typical infant development," Fuma said.

"CD11c microglia have been reported to be involved in myelination by being a major source of IGF-1. In the present study, both were decreased in inflammation during pregnancy, suggesting that this pathway is impaired in children with delayed neurodevelopment."

This study sheds light on the complex relationship between maternal inflammation and neurodevelopment. The researchers hope that understanding the role of CD11c-positive microglia in neurodevelopment will lead to new therapeutic strategies.

"If future studies confirm a decrease in these microglia in preterm infants exposed to inflammatory conditions, such as chorioamnionitis, early interventions could be developed to prevent or reduce the neurodevelopmental impact of maternal inflammation," Fuma said.

"By targeting CD11c-positive [microglia](#), it may be possible to protect infants from the long-term consequences of impaired myelination and improve their chances for healthy cognitive development."

More information: Kazuya Fuma et al, Prenatal inflammation impairs early CD11c-positive microglia induction and delays myelination in neurodevelopmental disorders, *Communications Biology* (2025). DOI: [10.1038/s42003-025-07511-3](https://doi.org/10.1038/s42003-025-07511-3)

Journal information: [Communications Biology](#)

Provided by [Nagoya University](#)

APRIL 1, 2025

GLP-1 drug use for weight loss has soared, costing billions

by Dennis Thompson



The number of Americans taking cutting-edge weight-loss drugs like Ozempic, Wegovy and Zepbound has skyrocketed in recent years, a new study says.

The number of people without diabetes taking a GLP-1 drug more than tripled between 2018 and 2022 in the U.S., researchers reported March 31 in the [Annals of Internal Medicine](#).

Spending on these drugs reached \$5.8 billion in 2022, with average prices consistently exceeding \$1,500 per prescription, results show.

"Despite this, [adoption](#) remained limited, as only 1 in 250 U.S. adults without diabetes used a GLP-1 (drug) in 2022," wrote the research team led by Michelle Dowsey, an epidemiologist with the University of Melbourne in Australia.

GLP-1 drugs mimic the GLP-1 hormone, which helps control insulin and blood sugar levels, decreases appetite and slows digestion of food.

Initially approved to control diabetes, the drugs' effectiveness at helping people lose weight led to their later approval for that purpose as well.

For this study, researchers analyzed U.S. federal research data in which people were asked about their GLP-1 prescriptions.

Results showed that among people without diabetes, GLP-1 drug use rose from 0.1% of the population in 2018 to 0.4% in 2022.

That doesn't seem like a lot, amounting to an estimated 854,000 Americans without diabetes using GLP-1 drugs to lose weight in 2022, but the drugs are costly, and can run close to \$1,000 or more a month, researchers found.

As a result, estimated annual spending on GLP-1 drugs among non-diabetics rose from \$1.6 billion between 2018 and 2021 to \$5.8 billion in 2022, the study showed.

On average, the drugs cost \$1,540 per prescription, but patients paid an average of just under \$35 out-of-pocket for each prescription.

The study provides the first characterization of nationwide GLP-1 use and spending among individuals without [diabetes](#) "to guide ongoing debates about balancing the sustainability of health care [spending](#) against improving accessibility and affordability of GLP-1 medications going forward," researchers conclude.

More information: Cade Shadbolt et al, National Trends in Glucagon-Like Peptide-1 Receptor Agonist Use in Adults Without Diabetes, 2018 to 2022, *Annals of Internal Medicine* (2025). DOI: [10.7326/ANNALS-24-02878](https://doi.org/10.7326/ANNALS-24-02878)

Journal information: [Annals of Internal Medicine](#)

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APRIL 1, 2025

4:3 intermittent fasting shows modestly greater weight loss than daily caloric restriction

by [American College of Physicians](#)

A randomized clinical trial compared the effects of 4:3 intermittent fasting versus daily caloric restriction on weight loss when combined with comprehensive behavioral support in adults with overweight or obesity. The data showed that 4:3 intermittent fasting resulted in

modestly greater weight loss over 12 months. The results are [published](#) in *Annals of Internal Medicine*.

Researchers from the University of Colorado School of Medicine and colleagues randomly assigned 165 adults with overweight or obesity to either 4:3 [intermittent fasting](#) or [calorie restriction](#) for 12 months to compare weight loss between interventions. Participants in the 4:3 intermittent fasting group undertook a modified fast to produce an 80% energy restriction for three nonconsecutive days per week.

On non-fast days, participants in the 4:3 intermittent fasting group did not have to restrict [energy intake](#), but were encouraged to make healthy food choices.

Participants in the calorie restriction group were prescribed a daily calorie goal designed to produce a 34.3% energy deficit. All participants were given a free gym membership and encouraged to exercise for at least 300 minutes per week. In addition to group-based behavioral support, participants received instruction in calorie counting and a guide to target dietary macronutrient content of 55% carbohydrates, 15% protein and 30% fat.

At 12 months, participants in the 4:3 intermittent fasting group had a –7.6% change in body weight compared to –5% in the calorie restriction group; 58% of those in the fasting group achieved weight loss of at least 5% at 12 months vs. 47% in the calorie restriction group.

Participants in the fasting group also achieved more favorable changes in cardiometabolic outcomes, including [systolic blood pressure](#), total and low-density lipoprotein cholesterol levels, and fasting [glucose levels](#).

According to the authors, the findings suggest that because fasting does not require participants to focus on counting calories and restricting food intake every single day, it may result in greater adherence and should be considered within the range of evidence-based dietary weight-loss approaches.

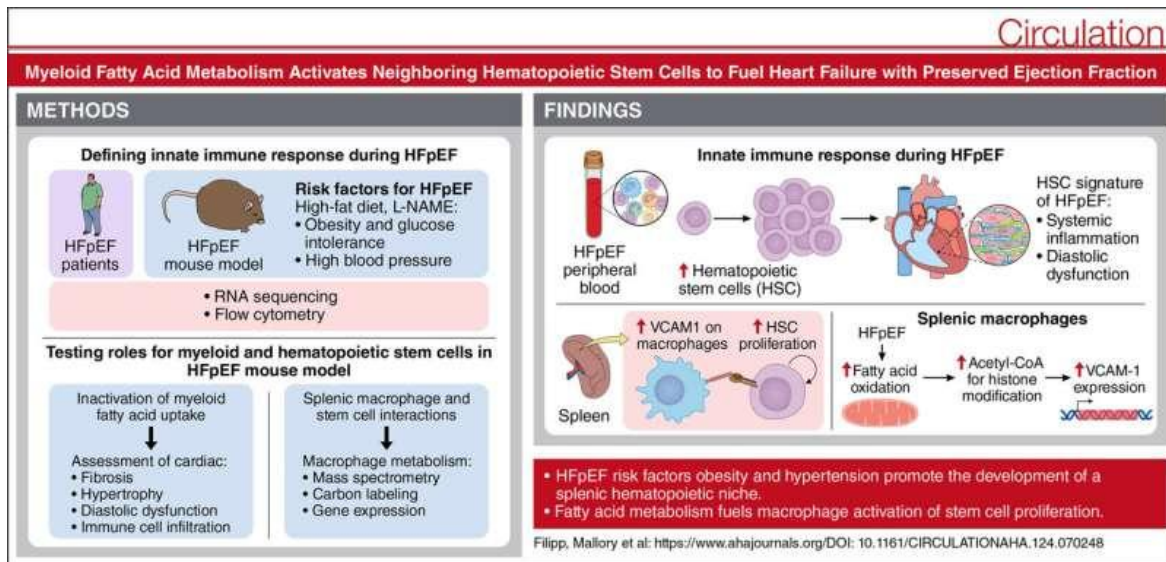
More information: Victoria A. Catenacci et al, The Effect of 4:3 Intermittent Fasting on Weight Loss at 12 Months, *Annals of Internal Medicine* (2025). DOI: [10.7326/ANNALS-24-01631](https://doi.org/10.7326/ANNALS-24-01631)

Journal information: [Annals of Internal Medicine](#)
Provided by [American College of Physicians](#)

APRIL 1, 2025

Exploring the link between inflammation and heart disease

by Olivia Dimmer, [Northwestern University](#)



Graphical Abstract. Credit: *Circulation* (2025). DOI: 10.1161/CIRCULATIONAHA.124.070248
A team of scientists led by Northwestern Medicine investigators have uncovered new details about a deadly heart condition with limited treatment options, according to a study [published](#) in the journal *Circulation*.

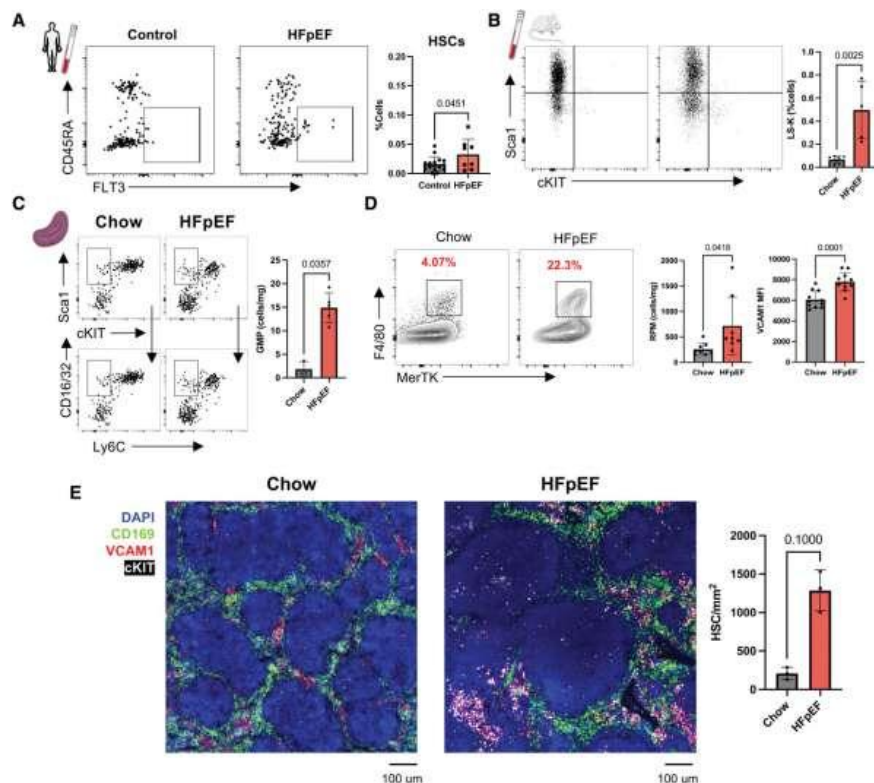
Heart failure with preserved [ejection fraction](#), or HFpEF, occurs when the heart pumps normally but is too stiff to fill properly. Current research suggests that HFpEF occurs when chronic medical conditions damage the heart and other organ systems in the body.

The condition can be deadly and [treatment options](#) remain limited, said Mallory Filipp, Ph.D., a postdoctoral fellow and first author of the current study, who was a former student in the laboratory of Edward Benjamin Thorp, Ph.D., the Frederick Robert Zeit Professor of Pathology.

"We've known for years that inflammation plays an important role in this kind of [heart failure](#)," Filipp said. "But the mechanisms and what exactly was happening was not well understood. So, we started by trying to figure out what immune cell subsets we could see within the heart."

In the study, Filipp and her collaborators used [flow cytometry](#) and single-cell RNA sequencing to identify immune cell populations within human and animal models of HFpEF. Next, investigators observed immune cell activity in mice with HFpEF and [high blood](#)

pressure that were fed a high-fat diet. They found that the mice had increased levels of blood stem cells and inflammatory proteins such as Vcam1, which has been linked to heart disease and inflammation.



RPMs orchestrate the development of a splenic hematopoietic niche during experimental HFpEF. Credit: *Circulation* (2025). DOI: 10.1161/CIRCULATIONAHA.124.070248

Finally, by administering medications that reduce blood glucose in the mice, investigators found that levels of Vcam1 could be partially lowered.

Taken together, the findings offer new insights into the relationship between inflammation and heart disease, as well as offering a potential therapeutic target for HFpEF.

"The findings are a good reminder that the heart exists within this whole body that's experiencing these systemic comorbidities," Filipp said. "It's not just about treating the heart individually, which is important, but treating the whole system: the whole patient."

Building on these findings, Filipp and her colleagues will study HFpEF and inflammation further in human patients to better understand how the disease impacts immune system responses.

"Now we're coming back full circle and asking, 'Once these cells are in the heart, what are they doing that's causing cardiac damage?'" Filipp said.

More information: Mallory Filipp et al, Myeloid Fatty Acid Metabolism Activates Neighboring Hematopoietic Stem Cells to Promote Heart Failure With Preserved Ejection Fraction, *Circulation* (2025). DOI: [10.1161/CIRCULATIONAHA.124.070248](https://doi.org/10.1161/CIRCULATIONAHA.124.070248)

Journal information: [Circulation](#)

Provided by [Northwestern University](#)

APRIL 1, 2025

3D-printed grafts: Shaping the future of bone and tissue regeneration

by Nieves Cubo Mateo, [The Conversation](#)



Credit: Unsplash/CC0 Public Domain

Over the past decade, 3D printing has gone from being a futuristic idea to a revolutionary tool. In medicine, its ability to produce custom-made, complex structures is changing the way doctors treat injuries and diseases—especially when it comes to rebuilding bones and other body tissues.

Additive manufacturing (as 3D printing is technically known) creates objects based on a digital model, building them layer by layer. In medicine, this technology is being used to

make inert objects like implants and prosthetics, but it can also create living tissues that help the body repair itself.

This exciting new development, known as bioprinting, uses tiny structures (called scaffolds) embedded with the patient's own cells to guide the growth of new tissue. This makes the printed structure more compatible with the body, and reduces the risk of rejection. It also helps the new tissue to heal faster and work more effectively.

In the future, this technology might even be used to print full organs for transplant, helping solve the worldwide shortage of donor organs.

Europe's pioneering efforts

Healing large or complex bone defects is one of the toughest challenges in surgery. Whether caused by accidents, cancer surgery or birth conditions, these defects often do not heal well with traditional bone grafts. One big problem is that the body struggles to grow [new blood vessels](#) inside the graft, which is essential for proper healing.

Researchers in Europe are currently at the forefront of developing innovative, groundbreaking new technology to tackle this problem, with initiatives spread across the continent.

Researchers at the [Institute for Bioengineering of Catalonia \(IBEC\)](#) have created 3D-printed scaffolds using polylactic acid and calcium phosphate that support bone growth and blood vessel formation. These have shown strong results in lab and animal tests, where scaffolds have [encouraged stem cells to grow](#) and release growth factors, successfully attracting blood vessels into the healing area.

At the University of Bergen, the [Tissue Engineering Group](#) is working on two major projects that use a patient's own [stem cells](#) to print bone replacements. These personalized constructs are designed to fit perfectly, reduce the chances of rejection, and improve the patient's recovery.

The EU-funded [Smart Bone Regeneration \(SBR\)](#) project is developing smart implants for rapid bone restoration with medical-grade polymers. The design also incorporates sensors to monitor implant performance, providing real-time data on bone growth and potential complications. *In vivo* studies in large animal models are currently underway to validate this approach.

The [Center for Translational Bone, Joint and Soft Tissue Research](#), in Dresden, is working on 3D-printed materials that support bone healing, with patients' own materials that help bone cells grow. They also combine bone cement with soft gels filled with living cells to create strong, custom implants. Their goal is to make bone implants that work well in the body, bringing these 3D-printed treatments closer to real use in hospitals, or even in outer space.

Another exciting example is from the company [BellaSeno](#), who work in partnership with the Julius Wolff Institute at the Charité hospital in Berlin. They are creating 3D-printed bone scaffolds that can support and guide new bone growth. Their custom-made system uses a high-speed, precise printing process that meets international medical manufacturing standards ([ISO 13485](#)). These implants are currently being evaluated for clinical use, offering hope for patients with large bone defects.

Such collaborations between academic institutions and private companies are essential. They help speed up the process of turning research discoveries into real-life treatments available in hospitals. These partnerships also ensure that safety, quality, and effectiveness remain a priority in every stage of development.

Overcoming challenges

The use of 3D printing in medicine is quickly moving from research labs to real [clinical use](#). The European cases mentioned above show how this technology is already helping patients heal better and faster. They are also paving the way for a new era of personalized and [regenerative medicine](#).

However, there are still some hurdles to clear. Printed implants need to stay strong and safe over time, so long-term studies and patient trials are essential. These help researchers understand how the materials perform in the body over the years. New medical tools must also meet strict safety rules, which takes time, but helps protect patients and build trust.

Progress will depend on close teamwork between scientists, doctors, engineers, and regulators. As research and trials move forward, 3D printing is likely to become a routine part of surgery. Personalized, cell-based implants could soon be a standard option for repairing [bone](#) and tissue, bringing us closer to a future where treatments are safer, faster, and made just for you.

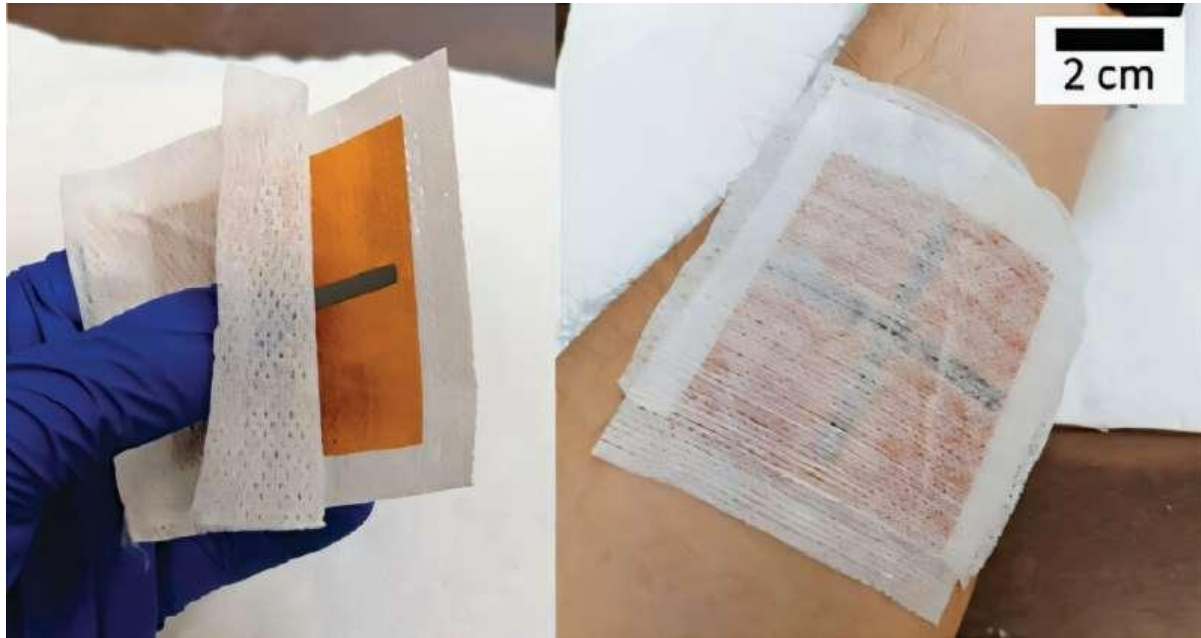
Provided by [The Conversation](#)

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APRIL 2, 2025

Smart skin sensor offers real-time infection detection

by [University of Rovira i Virgili](#)



The sensors (in the image) have thermoelectric properties and detect thermal variations without the need for batteries or external power supply. Credit: University of Rovira i Virgili

An international team led by researchers from the Universitat Rovira i Virgili (URV) has developed an innovative piece of equipment in the field of medical technology: an intelligent device capable of monitoring the temperature of the skin continuously and accurately. The device uses a thermoelectric system that works without batteries or an external power supply to detect temperature variations associated with inflammation and infection. The results of the project have been [published](#) in the journal *Advanced Science*.

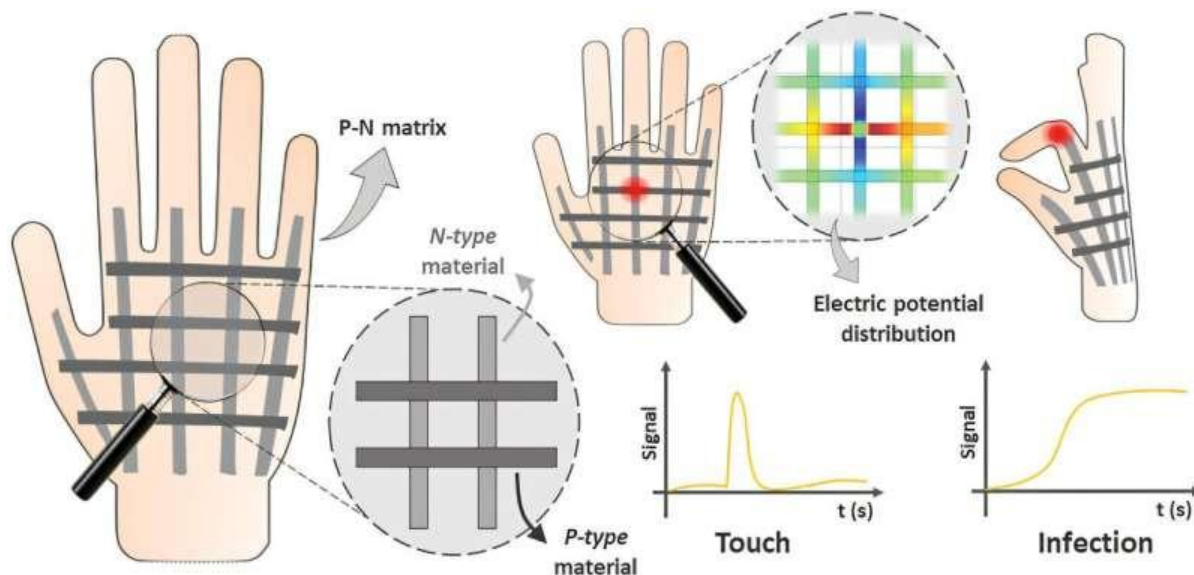
The device was created as part of a European project in collaboration with researchers from the University of Porto, who specialize in energy recovery systems. The initial aim of this research was to increase the autonomy of sensors used in medical and industrial environments. "The idea was to avoid dependence on batteries and to create [flexible devices](#) that could be adapted to the skin or other surfaces," explained Eduard Llobet, a researcher at URV's Department of Electronic, Electrical and Automatic Engineering.

Temperature as a key indicator

Temperature is an essential indicator for the early diagnosis of wounds and inflammatory processes in the skin. Local [temperature variations](#) can reveal infections, healing problems or other unhealthy alterations. Until now these diagnoses were carried out with infrared cameras or point sensors, but the problem with these techniques is that they require the patient to be immobilized and cannot monitor the entire skin surface.

This research marks a step forward. The device is able to precisely measure small changes in temperature and to identify specific points of inflammation thanks to its structure, which is based on a network of thermocouples. These are unions of semiconductor materials that generate an [electrical signal](#) when they detect differences in temperature. According to Llobet, "It is like creating a thermal map of the skin, with points that indicate where there is an affected area."

The device has been validated through [numerical simulations](#) and [experimental tests](#) on human skin, even under sweaty conditions using a saline solution to simulate perspiration. The tests have shown that it can detect temperature variations as small as 0.4°C and that it responds in less than three seconds to changes in temperature. This allows the evolution of a wound to be monitored in real time and any signs of infection to be detected quickly.



cheme of the thermoelectric configuration proposed in the current study for the detection of horizontal temperature gradients on the skin's surface. This configuration comprises p-type and n-type thermoelectric stripes transverse to each other, creating sensitivity points. Temperature fluctuations within the stripes lead to changes in the potential electrical distribution across the network, facilitating the location of these changes. Signal analysis enables differentiation between physical contact (touch) and infections. Credit: *Advanced Science* (2024). DOI: 10.1002/advs.202403775

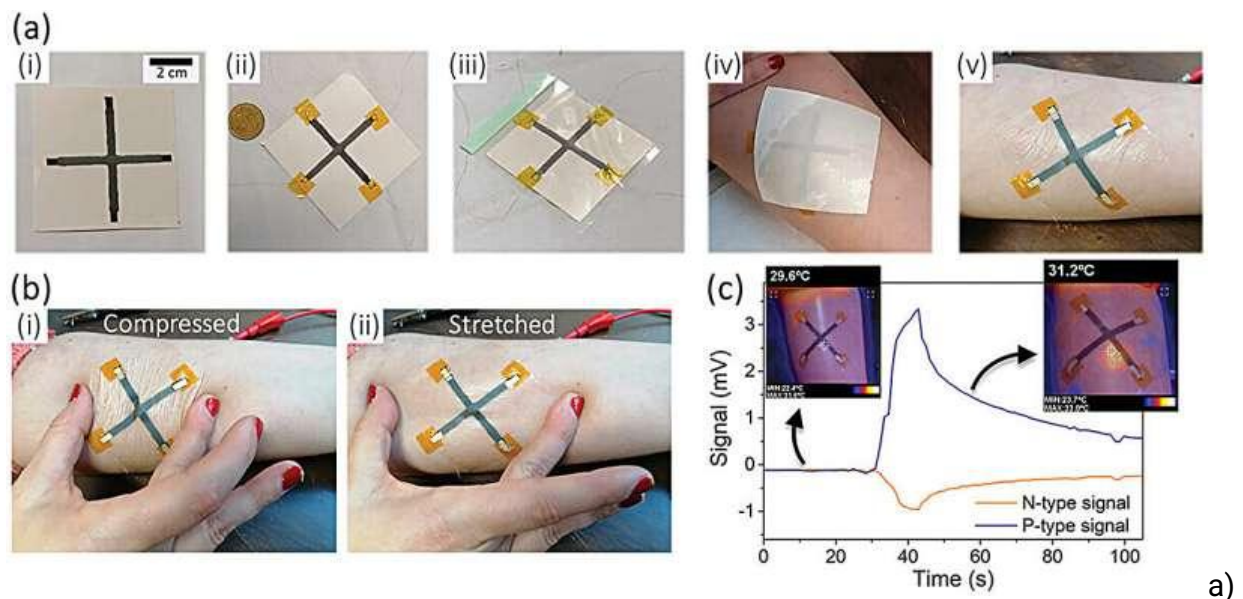
Pioneering and sustainable technology

The device is based on flexible thermoelectric materials combined with a conductive polymer printed using techniques similar to those used in the printing of t-shirts. This means it can be manufactured on flexible polymeric substrates that can adapt to the skin without difficulty. "This configuration is not only more precise than traditional sensors, but also more resistant to external interference, for example through sweat or the movement of the [skin](#)," said the researcher.

The researchers also tested the durability of the device. URV's Department of Mechanical Engineering subjected the sensors to hundreds of bending cycles to see if they deteriorated with use. "The results have been very promising, because we have shown that the sensor retains its functionality even after multiple deformations," said the head of department, Silvia de la Flor, who also participated in the research.

Easy to interpret and open to new applications.

Furthermore, the information provided by the sensor is easy to interpret: "The system can display a color map with temperature gradients, and this means that, without needing to be trained, health care staff can quickly identify the affected areas" said Llobet. This facilitates clinical decision-making and the proactive management of patient health.



Production of the sensor and transfer to the skin: i) screen-printing of the thermoelectric stripes and contacts, ii) placement of the contacts using 50 μm Cu wire, Ag ink for contacts and Kapton tape, iii) placement of the layer between the sensor and the skin, iv) placement of the tattoo paper in the skin and humidification, v) printed sensor obtained on the surface of the skin. b) Mechanical deformations applied to the sensor to confirm stability: i) Compressed, ii) Stretched. c) Thermoelectric signal obtained with the application of a

temperature gradient in the center of the configuration and respective thermal images.
Credit: *Advanced Science* (2024). DOI: 10.1002/advs.202403775

The device can be worn for several days before being replaced and in this regard it is similar to conventional techniques. "The idea is that it can be worn like any other device without affecting the person's comfort," said de la Flor.

This technological breakthrough opens the door to more personalized and accessible medicine. The sensor can help not only to prevent infections in surgical wounds and pressure ulcers, but can also be adapted to other applications such as the monitoring of inflammatory processes or for use in medical packs.

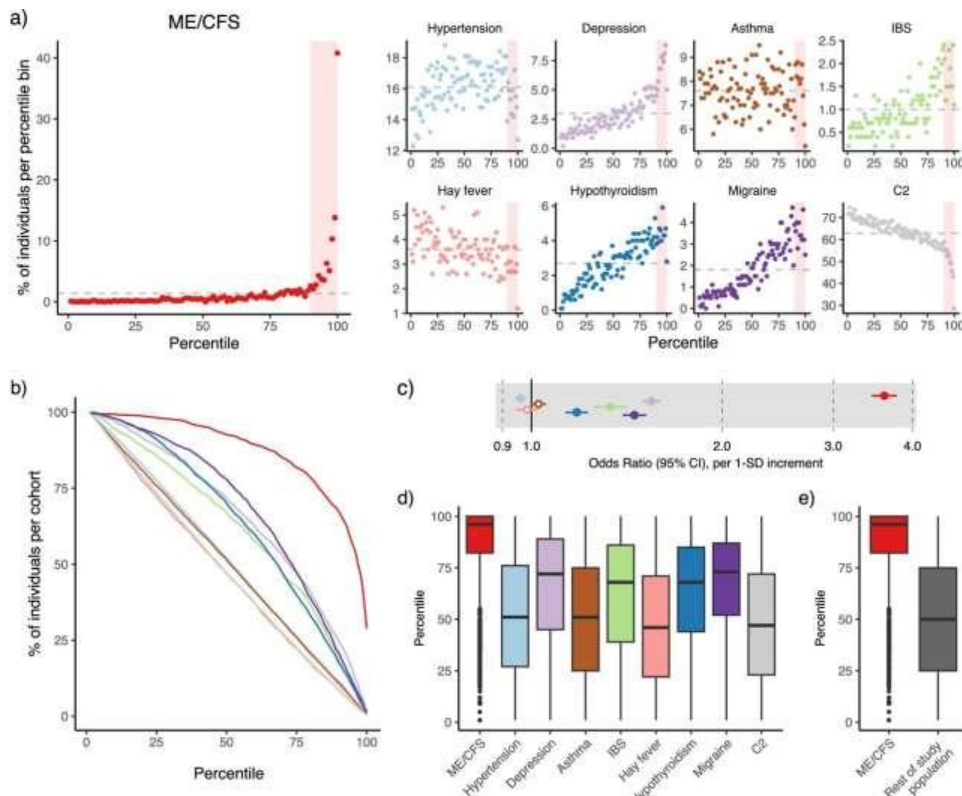
The research team is already working on new projects to expand the functionalities of the device, such as the incorporation of biosensors to measure other physiological variables, for example, levels of dissolved gases or specific biomarkers. "Our idea is to continue developing this technology to make it even more versatile and useful in the clinical setting," they concluded.

More information: M. A. S. Almeida et al, Touch Empowerment: Self-Sustaining e-Tattoo Thermoelectric System for Temperature Mapping, *Advanced Science* (2024). DOI: [10.1002/advs.202403775](https://doi.org/10.1002/advs.202403775)

Journal information: [Advanced Science](#)
Provided by [University of Rovira i Virgili](#)

APRIL 2, 2025

We don't yet know what causes chronic fatigue syndrome, but we're improving diagnosis by University of Melbourne



Stratified LightGBM score shows increased detection of ME/CFS cases in higher percentiles. Credit: *Communications Medicine* (2024). DOI: 10.1038/s43856-024-00669-7
As we come into cold and flu season, many of us will be hit by a virus once, twice or even more.

But imagine if one of these ailments dragged on for weeks, leaving you with that lingering sick feeling and fatigue.

Now, envision those symptoms persisting for months.

As the months stretched on, you would likely visit your doctor multiple times, and they might even mention long COVID—a term we've become familiar with through widespread media coverage.

But the concept of prolonged fatigue and ongoing symptoms after an illness is not exclusive to COVID-19.

Chronic fatigue syndrome, also called myalgic encephalomyelitis (ME/CFS), can occur following many types of infections—whether bacterial, viral, parasitic or fungal—or even after stress, surgery or pregnancy.

Regardless of the initial trigger, some individuals develop a long-term illness characterized by fatigue, brain fog, unrefreshing sleep and symptom flare-ups after exertion.

This illness isn't rare, with the Australian Government citing up to [250,000 suffering in Australia](#) and recent lifetime prevalence projections suggesting that 1 in 30 people will suffer.

ME/CFS doesn't discriminate

Decades before the emergence of COVID-19 and long COVID, ME/CFS was a recognized condition.

While it is often associated with young people who experience [prolonged symptoms after glandular fever](#), ME/CFS affects individuals of all ages and can occur at any time. The term '[chronic fatigue syndrome](#)' fails to give a true impression of the condition's severity.

Approximately one quarter of those with ME/CFS are [unable to leave their homes](#), and only [about 13% can maintain full-time employment](#).

The disease's fluctuating severity poses a significant challenge to everyday life.

When individuals with ME/CFS feel relatively well, they can engage in daily activities. However, during [symptom](#) flare-ups, they often [suffer in isolation](#) and can become bed-bound.

The challenge of diagnosis

One of the most troubling aspects of ME/CFS is the lack of clear answers for patients. Treatment options are limited, and none are currently approved for use in Australia.

The complexity of the disease adds to the challenge—patients experience varied symptoms, triggers and comorbidities (the simultaneous presence of two or more diseases or medical conditions in one patient), resulting in diverse responses to medication.

Obtaining a formal diagnosis is a major hurdle, as there is no diagnostic "[biological marker](#)" or biomarker, for example, a molecule found in blood that can be linked to ME/CFS symptoms.

The current diagnostic process requires ruling out other similar conditions, further complicated by the presence of multiple comorbidities for ME/CFS patients.

Toward diagnostic markers for ME/CFS

Our [research](#), published in the Nature journal *Communications Medicine*, is a large-scale data study that highlights the development of a differential diagnostic marker.

These markers help to distinguish one condition from others that share similar symptoms and, in this case, enable a faster diagnosis for ME/CFS patients.

In the study, we used the UK Biobank to identify biological markers significant to ME/CFS in this large cohort, which includes hundreds of thousands of individuals.

UK Biobank is a large-scale biomedical database and research resource containing de-identified genetic, lifestyle and health information and biological samples from half a million UK participants.

Traditional disease diagnostic markers depend on understanding what causes the disease, for example, a DNA mutation in certain types of cancers, but the cause remains unknown in ME/CFS.

However, we recognized that the current complicated diagnosis process for ME/CFS could be rapidly sped up by providing clinicians with the confidence to rule out similar conditions.

We looked for a pattern or signature of clinical and metabolite markers (products of metabolism) in more than 1,000 ME/CFS patients that distinguished them from more than 80,000 people with other similar or common comorbid diseases.

Our research identified a set of 19 basic health factors and nine blood markers that could differentiate ME/CFS from seven common comorbid conditions correctly 83% of the time.

The data revealed inflammation and abnormalities in cholesterol and triglycerides when comparing ME/CFS to other diseases. These findings could have significant implications for understanding the biological mechanisms underlying ME/CFS.

While the accuracy of this marker set is not perfect, this result is better than we expected, given that none of the UK Biobank data we identified was collected with ME/CFS in mind.

We are hopeful that this study provides a new path to being able to rapidly diagnose ME/CFS and to assess severity for treatment trial outcomes.

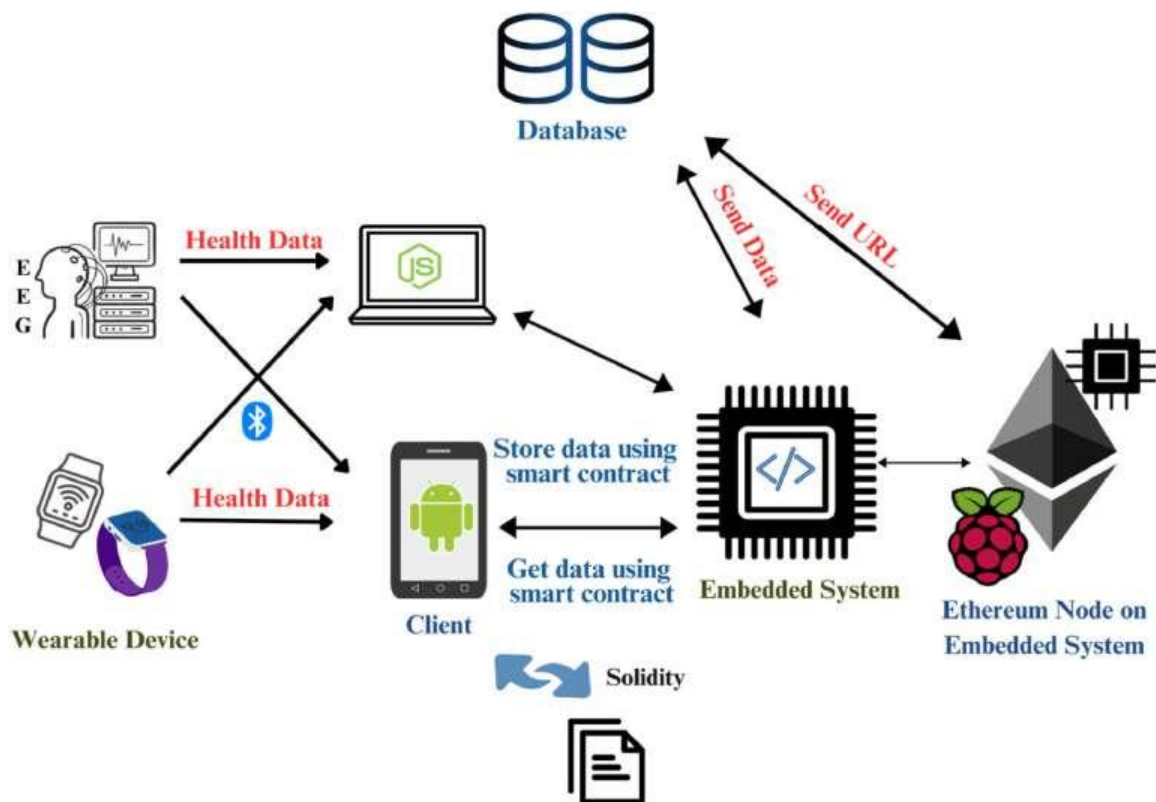
The next challenge we are working on is to translate this research to clinical use in the community.

More information: Katherine Huang et al, Discriminating Myalgic Encephalomyelitis/Chronic Fatigue Syndrome and comorbid conditions using metabolomics in UK Biobank, *Communications Medicine* (2024). DOI: [10.1038/s43856-024-00669-7](https://doi.org/10.1038/s43856-024-00669-7)
Journal information: [Communications Medicine](#)
Provided by [University of Melbourne](#)

APRIL 2, 2025

AI-driven smart devices to transform health care

by [University of Technology, Sydney](#)



Wearable devices are supported by a minimalist system (modified from [40]).

Credit: *Results in Engineering* (2024). DOI: [10.1016/j.rineng.2024.103787](https://doi.org/10.1016/j.rineng.2024.103787)

AI-powered, internet-connected medical devices have the potential to revolutionize health care by enabling early disease detection, real-time patient monitoring, and personalized treatments, a new study suggests.

They are already saving lives. Wearable devices can detect cardiac issues early, triggering emergency responses and preventing complications.

"The Internet of Medical Things (IoMT), powered by AI, connects [medical devices](#) for real-time monitoring and analysis—anything from a smartwatch to hospital monitors. For example, a wearable heart monitor can detect irregular rhythms and send alerts instantly," said Professor of Data Science Amir Gandomi, from the University of Technology Sydney (UTS).

"This study provides a comprehensive roadmap for integrating IoMT into health care, showing its potential to improve efficiency and reduce costs, while addressing key challenges like security and the ability to operate with other systems.

"For patients, it means better health management and fewer hospital visits. For families, it offers peace of mind, especially for seniors or those with chronic conditions. IoMT is making health care smarter, safer, and more responsive," he said.

While Gandomi's research covers a wide range of real-world engineering problems, he has a particular focus on the use of AI and [data analytics](#) to improve health care, including pandemic response and detection of diseases such as Parkinson's, diabetes, cancer and [heart disease](#).

The study, "[Transformative impacts of the internet of medical things on modern health care](#)," was led by Associate Professor Shams Forruque Ahmed from Sunway University in Malaysia, together with Professor Gandomi and an international research team, and published in the journal *Results in Engineering*.

"Our research highlights IoMT's potential to improve patient outcomes, reduce hospital strain, and reduce costs, making it essential for future health care systems," said Professor Gandomi.

It explores IoMT's full impact on health care—its benefits, challenges, and real-world applications, and highlights breakthrough results, such as AI-powered IoMT achieving 99.84% accuracy in heart disease diagnosis from [medical imaging](#), and [real-time](#) seizure detection.

The study also examines the challenges of integrating AI-powered IoMT technology into health care systems, including the need for strong data security, device compatibility, and better regulations to ensure patient trust and safety.

"For health care providers, investing in IoMT means upgrading digital infrastructure, training staff, and adopting remote monitoring for proactive care. IoMT also requires clear regulations and standards to ensure security and patient privacy," said Associate Professor Ahmed.

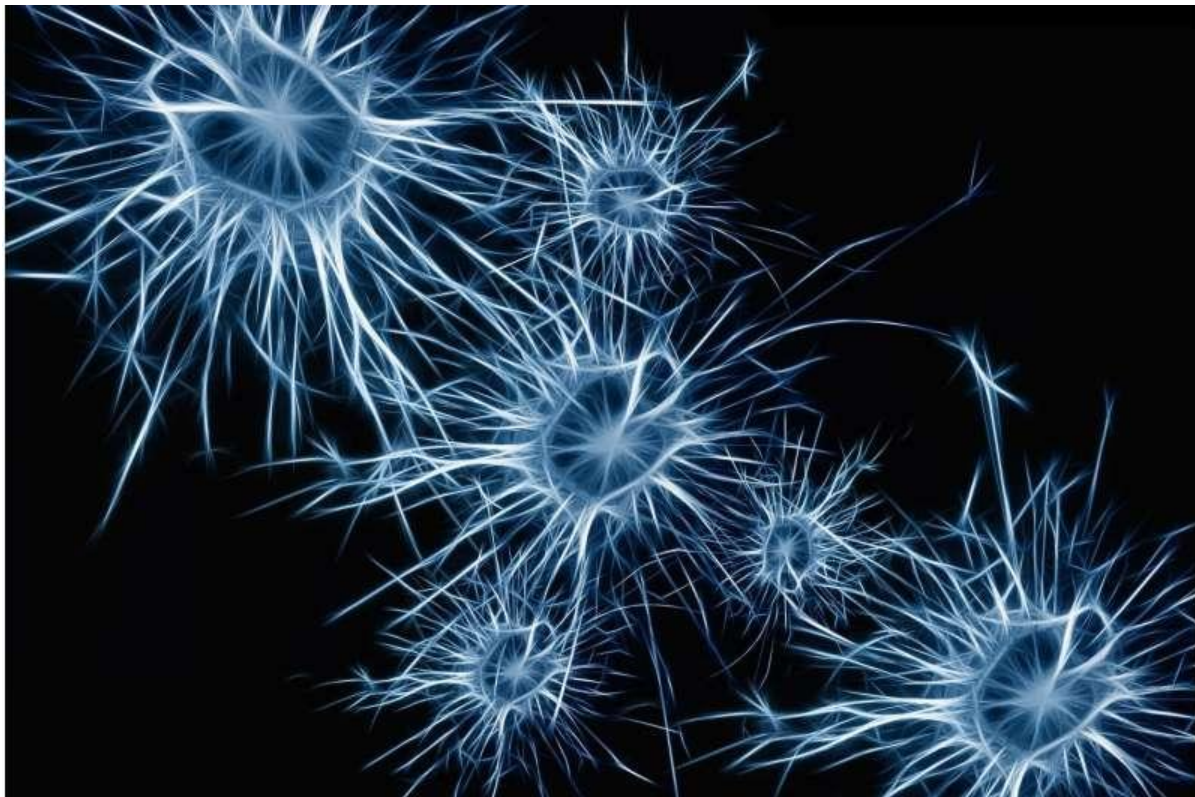
This study is relevant to patients, health care professionals, policymakers, and MedTech innovators, anyone interested in improving health care access, accuracy, and efficiency using AI and connected technologies.

More information: Shams Forruque Ahmed et al, Transformative impacts of the internet of medical things on modern healthcare, *Results in Engineering* (2024). DOI: [10.1016/j.rineng.2024.103787](https://doi.org/10.1016/j.rineng.2024.103787)
Provided by [University of Technology, Sydney](#)

APRIL 3, 2025

A new targeted approach could protect neurons against degeneration

by [Temple University](#)



Credit: CC0 Public Domain

Neurodegenerative conditions such as Parkinson's disease and Alzheimer's involve progressive neuronal loss due to disease-induced damage. An enzyme known as dual leucine-zipper kinase (DLK) plays a key role in this process, telling neurons that are damaged or unhealthy when they should cut their losses and self-destruct. Hence, sparing neurons from DLK is an attractive therapeutic strategy that could slow disease progression.

Past attempts to inhibit DLK's action in human patients, however, have led to unexpected side effects affecting the nervous system, suggesting that DLK also has beneficial effects on neurons and that blocking it indiscriminately is harmful. Now, in a study published

in *Nature Communications*, a group of scientists led by Gareth Thomas, Ph.D., Associate Professor of Neural Sciences in the Center for Neural Development and Repair at the Lewis Katz School of Medicine at Temple University, describes a more precise way to block DLK in damaged neurons, while preserving its function in healthy neurons.

The research reignites interest in DLK inhibition as a treatment strategy for neurodegenerative disease at a critical time, as cases of Parkinson's, Alzheimer's, and other conditions associated with neurodegeneration are poised to double by 2040.

"This study exemplifies the innovative spirit and collaborative strength of our research community at the Katz School of Medicine," said Amy J. Goldberg, MD, FACS, The Marjorie Joy Katz Dean of the Lewis Katz School of Medicine.

"By uncovering a more precise way to protect neurons, Dr. Thomas and his team are paving the way for treatments that could truly change the trajectory of neurodegenerative diseases."

In designing their breakthrough approach, Dr. Thomas's team considered the way damage affects axons—the long, thin projections on neurons that convey impulses within the brain and spinal cord and to other regions of the body. When axons are damaged, DLK sends signals from the site of injury in the axon back to the neuron's nucleus, which triggers the self-destruction process.

Previous attempts to completely block the enzymatic activity of DLK caused the development of severe sensory neuropathy in patients.

"This clinical finding suggested that the conventional DLK inhibitor might be disrupting the normal structure and function of axons," explained Dr. Thomas.

Confirming this idea, when his team treated cultured neurons with an existing DLK inhibitor, they indeed saw that axonal structure was rapidly disrupted. This inspired them to seek an alternative approach to more selectively block the enzyme.

"From some of our previous research, we knew that DLK initiates self-destruction signals from very specific locations in neurons," Dr. Thomas said. "We thought that if we could stop DLK getting to those locations, it wouldn't be able to initiate the self-destruction process."

Working with Dr. Wayne Childers at the Moulder Center for Drug Discovery in Temple's School of Pharmacy and with Dr. Margret Einarson at Fox Chase Cancer Center, Dr. Thomas's team sought to identify compounds that alter the location of DLK in cells.

"We screened more than 28,000 compounds and eventually hit on two in particular that protect neurons from DLK-driven damage," Dr. Thomas said.

The two compounds not only protected cultured [neurons](#) from degeneration but also reduced DLK signaling in animal models. Very importantly, they did not cause the axonal disruption that they saw with the conventional DLK inhibitor.

"Our findings reveal an exciting, novel way to block DLK-dependent signals," Dr. Thomas said.

Next steps involve working with medicinal chemists to make the compounds more potent and even more specific to minimize off-target effects.

"The current compounds also need to be made more stable if we want to move forward and develop them as drugs. We hope that moving this class of compounds toward the clinic may yield a valuable therapy for patients in the future," he added.

Other researchers who contributed to the study include Xiaotian Zhang, Heykyeong Jeong, Jingwen Niu, Sabrina M. Holland, and Brittany N. Rotanz, Center for Neural Development and Repair, Lewis Katz School of Medicine at Temple University; and John Gordon, Moulder Center for Drug Discovery, School of Pharmacy, Temple University.

More information: Inhibiting acute, axonal DLK palmitoylation is neuroprotective and avoids deleterious effects of cell-wide DLK inhibition, *Nature Communications* (2025). DOI: [10.1038/s41467-025-58036-6](https://doi.org/10.1038/s41467-025-58036-6)

Journal information: [Nature Communications](#)

Provided by [Temple University](#)

APRIL 3, 2025

A failed fragile X syndrome drug can reduce cocaine use, clinical trial finds

by Bob Yirka , Medical Xpress



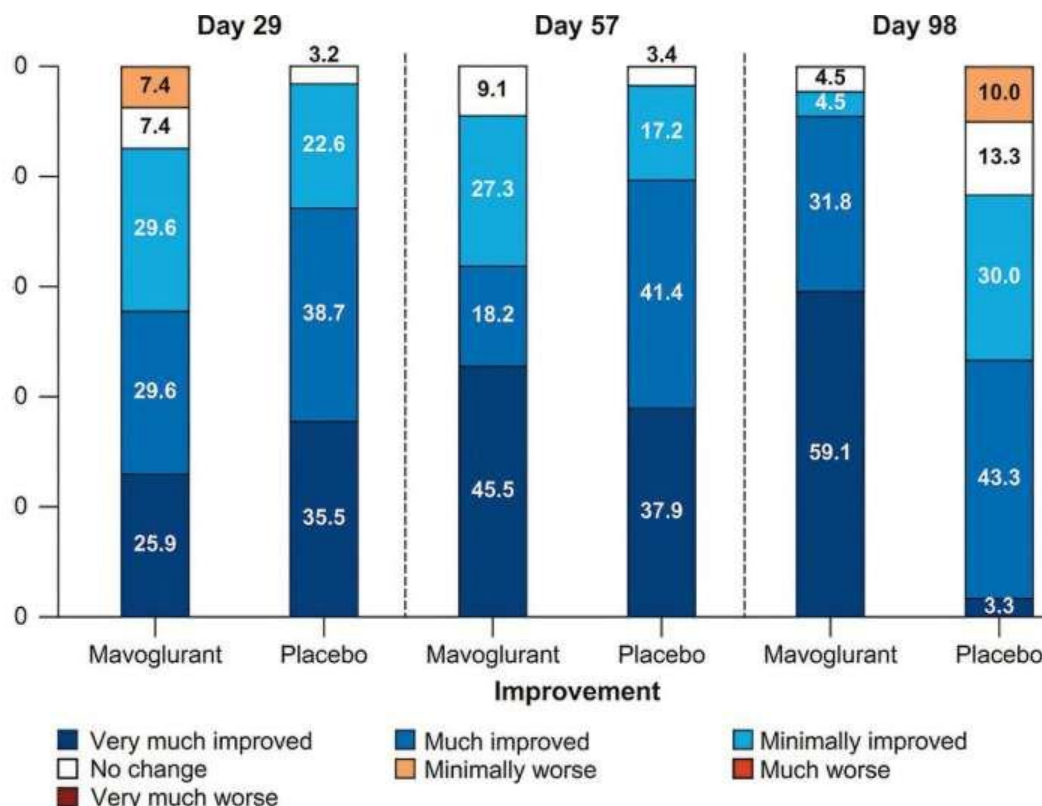
pile of cocaine hydrochloride. Credit: DEA Drug Enforcement Agency, public domain
A team of pharmaceutical researchers at Novartis Biomedical Research, working with an international team of associates, has found that the drug mavoglurant can reduce use in people with cocaine use disorder. In their study, [published](#) in the journal *Science Translational Medicine*, the group conducted a Phase II clinical trial with 68 people with a diagnosed cocaine use disorder.

Mavoglurant was first developed by a team at Novartis as a [treatment](#) for fragile X syndrome, which is a genetic neurodevelopmental condition that usually leads to some degree of intellectual disability. Trials for the drug were disappointing, prompting Novartis to explore other possible uses.

Testing explored its use as a treatment for Levodopa-induced dyskinesia (a type of dyskinesia associated with the prescription of L-Dopa for Parkinson's disease), reducing smoking [withdrawal symptoms](#) or even OCD. Eventually, the team hit on the idea of using it

to reduce craving symptoms in people with alcohol and cocaine use disorders. Initial testing showed some success, leading to a successful Phase I clinical trial.

In their Phase II trial, the research team recruited 68 people previously diagnosed with cocaine use disorders to take either a placebo or a single dose (one pill) of mavoglurant twice a day for 98 days. Use of cocaine by the participants was self-reported and tested via urine and hair throughout the trial.



Credit: *Science Translational Medicine* (2025). DOI: 10.1126/scitranslmed.adi4505

The research team found that use of the drug led to "significant reductions" in cocaine use compared to the placebo in most of the participants. The researchers also found that the medication led to a decrease in alcohol use by most of the participants, although this reduction in alcohol use was not statistically significant. Side effects included dizziness, nausea and headache, though the participants who experienced them described them as manageable. The researchers describe the testing outcome thus far as effective, safe and tolerable.

If the drug passes all its [trials](#) and at some point is approved by the FDA and other medical agencies, it would represent the first drug to be marketed as a therapy for treating cocaine abuse. Current treatments involve psychosocial sessions such as [cognitive-behavioral therapy](#) and counseling, which, to date, have proven to be far less effective than desired.

More information: Baltazar Gomez-Mancilla et al, Mavoglurant reduces cocaine use in patients with cocaine use disorder in a phase 2 clinical trial, *Science Translational Medicine* (2025). DOI: [10.1126/scitranslmed.adi4505](https://doi.org/10.1126/scitranslmed.adi4505)

APRIL 3, 2025

Study helps explain how laughing gas could fight treatment-resistant depression

by [University of Chicago Medical Center](#)



Credit: Unsplash/CC0 Public Domain

Although a plethora of medications and therapies now exist for major depression, [roughly one in three](#) diagnosed patients still suffer from treatment-resistant depression (TRD)—a form of the disorder that does not respond to first-line antidepressants. Even when traditional drugs work, they often take weeks to kick in, delaying relief for people in crisis.

In response to this need, scientists have recently begun investigating whether fast-acting substances with different neurological effects could serve as alternative antidepressants.

Encouraged by [initial successes with ketamine](#), researchers including Peter Nagele, MD, Professor of Anesthesia and Critical Care and Psychiatry and Behavioral Neuroscience at

the University of Chicago, turned to another anesthetic: [nitrous oxide](#), better known as "laughing gas."

Early clinical studies showed promise, so Nagele and his collaborators wanted to understand more about what exactly happens inside the brain during and after laughing gas treatment.

In a study published in [Nature Communications](#), they pinpoint a novel neurological mechanism that helps explain how a gas that exits the body in minutes can trigger lasting improvements in mood.

"Figuring out how the observed antidepressant effects work at a neural and molecular level is an important step toward clinical acceptance and implementation," said Nagele, the senior author of the new paper.

A new use for a familiar anesthetic

Most people associate laughing gas with dentists' offices, where it's used to ease anxiety and dull pain. While its nickname hints at euphoric effects, at the low doses used for depression research, it acts as a sedative, giving people a temporary feeling of calm rather than making them feel giddy.

"Nitrous oxide is the oldest anesthetic we've got—it's been used worldwide for over 180 years, costs about \$20 a tank, and yet we're still learning what it can do," said first author Joseph Cichon, MD, Ph.D., an assistant professor of Anesthesiology and Critical Care at the University of Pennsylvania. "I felt like Indiana Jones, going back in time to crack the mystery of this ancient drug."

In the [preliminary clinical trials](#) led by Nagele and researchers at Washington University in St. Louis, even a single inhalation session could bring positive change to patients who hadn't responded to other treatments, with effects lasting up to two weeks in some cases.

"The results were striking," Nagele said. "We saw people who had been struggling for years experience meaningful improvement within hours that lasted for weeks. It made us wonder what, exactly, was happening in the brain to cause this."

Defying expectations about brain activity

For years, scientists assumed the antidepressant effects of both nitrous oxide and ketamine were tied to the drugs' ability to block specific proteins on brain cells involved in memory and learning: N-methyl-D-aspartate (NMDA) receptors.

But while this theory was widely accepted, it had never been fully tested in living brain circuits. More importantly, it didn't fully explain why nitrous oxide, which leaves the body very quickly, could produce lasting effects.

To investigate, the researchers from UChicago, UPenn and WashU used advanced calcium imaging to observe [brain activity](#) in mice that inhaled nitrous oxide after being exposed to chronic stress—a common model for depression. Looking at the [cingulate cortex](#), a brain region associated with emotional regulation and mood, they zeroed in on a specific group of neurons known as layer V (L5) [pyramidal neurons](#).

"Particularly in stress-related depression, we usually see that these L5 neurons are underactive in both mice and humans," Nagele said.

In the experimental mice, however, the researchers saw that nitrous oxide quickly and selectively activated L5 neurons, pulling them out of their state of stress-induced inactivity even after the gas left mice's bodies. The previously-stressed mice almost immediately perked up and started doing more enjoyable activities like sipping sugar water.

"This 'disinhibition' effect—where the brain becomes less suppressed and more engaged—looks to be a crucial reason for the drug's antidepressant benefits," Nagele explained. "It helps reactivate [neural circuits](#) dulled by stress and depression without needing to form entirely new brain connections."

The key turned out to be specialized [potassium channels](#) found in L5 neurons, called SK2 channels. Under normal conditions, these channels help shut down neuron activity, but nitrous oxide blocks the SK2 channels, preventing them from silencing the L5 cells. As a result, the neurons remain active and the surrounding brain circuit shifts into a more excitable, energized state.

Steps toward a new generation of depression treatments

"These results show us there might be more than one path to the desired outcome in depression treatment," Nagele said.

"NMDA receptors matter, but what we're seeing with nitrous oxide suggests there's another way to spark the brain's circuitry back into action. It's an exciting discovery because it widens our understanding of how we can tackle depression from multiple angles.

The researchers caution that while the findings are promising, more studies are needed to understand how long the neurological effects of laughing gas last and whether they contribute to deeper, more permanent recovery.

However, this fresh understanding of how fast-acting antidepressants might work—without relying solely on NMDA-receptor mechanisms—opens the door to future drug development. For example, rather than relying on an inhaled gas that must be clinically administered, scientists might one day design oral medications that mimic this mechanism.

"This study brings us one step closer to understanding how nitrous oxide can help patients who haven't responded to anything else," Nagele said. "If we can isolate the exact pathways

involved, we could create new depression treatments that are more accessible and longer-lasting."

More information: Nitrous oxide activates layer 5 prefrontal neurons via SK2 channel inhibition for antidepressant effect, *Nature Communications* (2025). DOI: [10.1038/s41467-025-57951-y](https://doi.org/10.1038/s41467-025-57951-y). www.nature.com/articles/s41467-025-57951-y

Journal information: *Nature Communications*
Provided by [University of Chicago Medical Center](#)

APRIL 3, 2025

What stroke recovery looks like today

by Grace van Deelen, [Tufts University](#)



Credit: Anna Shvets from Pexels

You may be familiar with the common signs and symptoms of a stroke: loss of vision, drooping face, weak limbs, and slurred speech. But what happens during the recovery phase, once a patient has spent time in the hospital and their brain has begun to recover?

Lester Leung, a vascular neurologist at Tufts Medical Center and associate professor at Tufts University School of Medicine, Gabriele Moriello, a physical therapist and associate professor in the Department of Rehabilitation Sciences at the School of Medicine, and David Thaler, a vascular neurologist at Tufts Medical Center and professor at the School of

Medicine, recently shared how stroke patients recover and what factors may help or hinder the process.

The shape of recovery

There are two main categories of strokes: [ischemic strokes](#) and hemorrhagic strokes. Most strokes, about 87%, are ischemic strokes, caused by the blockage of a blood vessel in the brain. Hemorrhagic strokes, about 13% of strokes, are caused by bleeding in the brain.

Both types of strokes can damage healthy brain tissue. But the two types have slightly different trajectories of recovery. A patient with an ischemic stroke will see their [brain function](#) and mobility drop down after a stroke, then begin to improve starting as soon as 24 hours after their stroke.

A person with a hemorrhagic stroke also loses brain function quickly after a stroke. But because there is blood in the brain, hindering its function, it takes longer for that person's function to return. As blood reabsorbs into the brain, "there's a very steep potential incline in terms of regaining function, but it's delayed," Leung said.

For both ischemic and hemorrhagic strokes, the potential for improvement is high, Thaler said. The "shape" of recovery is also similar: People improve quickly early on, then their progress begins to slow. And while the severity of a stroke doesn't typically change the shape of recovery, it does affect the extent to which a person will ultimately improve, he said—a person with a mild stroke may regain all their functions, while someone with a severe stroke, such as one that paralyzed them, may not ever recover completely.

"Recovery is different for every single person," Moriello said.

Rebuilding the brain

As soon as 24 to 48 hours after a stroke, physical therapists like Moriello will begin to mobilize a stroke patient, asking them to get out of bed, stand up, and walk around. A person's long-term outcomes are better the sooner they're able to get up and moving, she said.

Next, people who can tolerate three hours of therapy per day typically head to inpatient rehab, which may include [physical therapy](#) (a method to improve [physical movement](#) and mobility), [occupational therapy](#) (similar to physical therapy, but based on the movements a person needs to carry out their role in life), speech therapy (which focuses on speech and swallowing), or a mix of all three.

After one to three weeks of inpatient rehab, the patient will usually head home, where they're able to continue therapy via [home visits](#) from therapists or by attending an outpatient therapy center. Home care and outpatient therapy usually last three to six months, after which insurance typically stops covering it, Leung said.

Therapies like these work because the brain is "plastic," or able to adapt its abilities, Moriello said. Therapies after a stroke use repetition to reinforce or even recreate neural pathways associated with movement that may have been lost or damaged during the stroke. "We have to make sure a patient is getting enough repetition that we're changing their brain, so that they can get back to doing some of the things they were doing before the stroke," she said.

"The brain needs to re-learn that these things are important," Leung said.

A stroke can also affect a patient's emotional health. About half of stroke patients experience an increase in symptoms of anxiety and depression, and some patients also deal with post-traumatic stress disorder. "Strokes are scary, and they make people introspective and concerned about a potential second stroke," Thaler said. Physicians typically refer patients struggling with anxiety and depression after a stroke to counseling or psychotherapy. In some cases, they also prescribe a patient medications meant to improve mental health.

Leung hopes that physicians who treat stroke patients begin to think of stroke recovery as a never-ending process. There's a misconception that stroke patients reach their maximum recovery within a few months after their stroke.

"That's the dominant narrative," Leung said. But research shows that a person can continue to regain function throughout their life. "We have to shift our understanding of stroke," he said. Leung also hopes that ongoing research will help physicians better personalize therapies for stroke patients based on the location of their stroke in the brain, among other factors.

Moriello and Leung also stressed the importance of counseling patients during recovery about how to avoid a second stroke. The risk factors for a second stroke are very similar to risk factors for a first stroke, with heart disease, diabetes, high blood pressure, and obesity at the top of the list.

Losing weight and improving cardiovascular health through diet and exercise can mitigate those risk factors. Counseling for prevention "should be involved in every single person's stroke recovery," Moriello said.

Healing factors

Many factors can either improve or hinder someone's recovery. How quickly someone can get medical care, and the type of care they're able to get, play a large role, Moriello said. People who are able to recognize stroke symptoms quickly and get to a comprehensive stroke center are more likely to recover well.

Two relatively new stroke treatments also provide a huge benefit to patients: intravenous medications that break up clots in the brain, called tissue plasminogen activator, or tPA, and a procedure called mechanical thrombectomy, in which a surgeon retrieves a clot using a

catheter. Both treatments improve patient outcomes significantly, Thaler said, but need to be done within the first few hours of symptoms.

Proximity to a comprehensive stroke center (a hospital with certified abilities to treat stroke cases) largely determines whether someone will be able to get appropriate care. But insurance also matters—research shows that patients with insurance that covers hospital care after a stroke have better outcomes and a lower risk of death.

Thaler pointed out that even without neurological improvement, there are ways to improve a patient's quality of life, such as teaching the patient how to navigate daily tasks with the abilities they still have. For example, a patient with a completely paralyzed arm might learn how to use their other limbs in new ways to supplement their mobility. Promising new technologies—some that stimulate the brain to amplify its signals—can also improve patients' lives, though research is ongoing, Leung said.

A person's support network can make a huge difference, too. For example, if someone recovering from a stroke has friends and family nearby, those loved ones can help them practice their therapies outside of a medical setting. "That helps us get in all the repetition that we need to make people better," Moriello said.

The landscape of stroke recovery and rehabilitation is evolving in a positive direction, allowing survivors to lead meaningful and enjoyable lives, Leung said. Nonetheless, he added, there is a critical need for further research to continue to progress rehabilitation strategies.

Provided by [Tufts University](#)

APRIL 3, 2025

Cognitive decline comes sooner for people with heart failure

by Noah Fromson, [University of Michigan](#)



Credit: Andrea Piacquadio from Pexels

There are over six million Americans with heart failure who are at greater risk of losing their cognitive abilities earlier in life, a study suggests.

The research team, led by Michigan Medicine, examined the cognitive abilities of nearly 30,000 adults over time, comparing those who did and did not develop heart failure.

The researchers found heart failure is associated with a significant decrease in cognition at the time of diagnosis.

Global cognition and executive functioning also declined more rapidly over the years after heart failure diagnosis, as people with the condition mentally aged the equivalent of 10 years within just seven years of a heart failure diagnosis.

The results are [published](#) in *Circulation: Heart Failure*.

"Heart failure is a disease that never goes away and treating it relies heavily on a patient's ability to follow specific instructions, monitor their symptoms and keep up with many

different medications," said Supriya Shore, MBBS, MSCS, first author and clinical assistant professor of internal medicine-cardiology at University of Michigan Medical School.

"Seeing this cognitive decline among patients, and how it worsens over time after a diagnosis of heart failure, should be a warning for providers to assess a patient's cognitive ability early and factor it into the care plan."

Typical risk factors for [cognitive impairment](#), such as [high blood pressure](#) and [heart attack](#), did not explain the accelerated decline observed in the participants with heart failure, according to the study.

The largest decrease in global cognition—a composite of several features of cognitive ability, including attention and problem solving—occurred among older adults, women, and white participants.

Investigators say, based on their findings, that adults with heart failure would reach the threshold for meaningful decline in global cognition nearly six years earlier than people without it.

Executive functioning would diminish around four-and-a-half years earlier.

"Regular cognitive monitoring of [older adults](#) with heart failure would help identify individuals with the earliest signs of cognitive decline who require supportive care," said senior author Deborah A. Levine, M.D., M.P.H., professor of internal medicine and neurology at U-M Medical School. "We need a better understanding of the mechanisms driving accelerated cognitive decline after heart failure to develop interventions that halt or slow the decline."

Decisions about treatment, Shore says, are complex and highly dependent on cognitive capacity.

These findings likely also explain the results of a 2024 qualitative study led by Shore which found that [most patients with heart failure displayed a poor understanding of their prognosis](#).

Caregivers, on the other hand, commonly identified when a patient was declining in health.

Some participants said their physicians avoided these conversations, while others said they were confused by the provider's medical jargon.

"Most people with heart failure, as well as their care partners, want discussions around prognosis to begin early in the course of the disease and to be repeated routinely using patient-centered language," Shore said.

"They want to speak candidly about quality of life and survival assessments. Even though these conversations are tough, people want to have it. With what we know about the rate

of [cognitive decline](#) in this patient population, there is a major need to meet patients where they are with clarity and compassion to improve overall care in heart failure."

More information: Supriya Shore et al, Trajectory of Cognitive Function After Incident Heart Failure, *Circulation: Heart Failure* (2025). DOI: [10.1161/CIRCHEARTFAILURE.124.011837](https://doi.org/10.1161/CIRCHEARTFAILURE.124.011837)
Provided by [University of Michigan](#)

APRIL 3, 2025

Your abdominal core: Expert shares tips for protecting a part of the body you may not think about

by Sharon Theimer, [Mayo Clinic](#)



Credit: CC0 Public Domain

It is a large part of the body that lies deep inside, out of sight and perhaps out of mind: your abdominal core. A new and rapidly developing area of medicine focuses on abdominal core health, including how people can incorporate it into a healthy lifestyle and how to address complex medical problems that arise when the core is compromised. Dr. Charlotte Horne, a metabolic and abdominal wall reconstructive surgeon at Mayo Clinic, explains what

abdominal core health is, how to protect it and risk factors for problems that may require surgery.

"The abdominal core is the outside muscular container of your abdominal wall," Dr. Horne says. "This starts at the diaphragm and goes all the way down to the pelvic support muscles. Most of the core is muscle and [connective tissue](#). It's a muscular container that holds your internal organs in. Every time you breathe, bend, bear down to have a bowel movement, you're using these muscles."

The abdominal core includes [abdominal muscles](#) that people may think of as the "six-pack," and oblique muscles and tissue that wrap all the way around the upper part of the abdomen and connect to the midline of the body, Dr. Horne adds. Many nerves lie between the layers of muscle and tissue, including those that extend to the groin, thighs, back and hips.

Those muscles function as a unit and that unit needs to operate well for you and your body to perform daily activities, Dr. Horne says. One way to strengthen the structural integrity of your abdominal core is to engage it during your normal activities, she explains. You do not have to do thousands of sit-ups or become a bodybuilder.

"One of the things we're realizing is that we need to educate people on how to appropriately engage those muscles when they do everything from going from lying to sitting and sitting to standing, lifting objects and other basic movements in their daily lives," she says. "When people do yoga or Pilates, they think about pulling their belly button into their spine. That helps stabilize the deeper muscles of the abdominal wall."

Improving abdominal core health involves conscious engagement of the abdominal muscles, "bringing everything in and holding it in," Dr. Horne says.

Risk factors for abdominal core problems are wide-ranging. They include [cancer treatment](#); inflammatory bowel disease; chronic or severe coughing; and complications from pregnancy (diastasis recti) and [surgery](#). The most common problems are hernias, when part of an organ or tissue bulges through a weak spot in muscle.

"Coughing can cause large hernias. When you're coughing, you're bearing down and there is a sudden, acute change in intra-abdominal pressure, almost like punching from the outside in or from the inside out," Dr. Horne explains. "When you're doing that all of the time, it causes significant stress to your abdominal wall."

Avoiding smoking and [excessive alcohol consumption](#) can help protect abdominal core health. Smoking increases the risk of chronic coughing, while heavy alcohol consumption to the point of cirrhosis can cause hormonal changes that in turn weaken the abdominal wall, Dr. Horne says.

As knowledge about abdominal core health grows, approaches to protecting it and healing it are advancing, Dr. Horne says. For example:

- Pregnancy causes muscles to expand to accommodate a baby, and sometimes those muscles do not go back to normal. Exercise regimens during and after pregnancy can help to stabilize them.
- Health care experts are realizing that restrictions on movement after surgery may not help and sometimes may be harmful. Rather than telling people not to lift anything, it may be more appropriate to explain how to safely reengage those muscles and tendons, Dr. Horne suggests.
- People with [inflammatory bowel disease](#) are likelier to have surgery and therefore are likelier to develop hernias. Surgeons now know that in those patients, mesh should be placed in different anatomic planes to prevent the mesh from touching the bowel and potentially causing problems later, Dr. Horne says.
- Pelvic floor [physical therapy](#) can help women experiencing urinary or [fecal incontinence](#) after pregnancy or menopause, she says.
- There is growing recognition that mesh used to repair hernias isn't one-size-fits-all. Dr. Horne's research focuses on hernia repair in women, including mesh and mesh techniques.

In her practice, Dr. Horne specializes in complex hernia surgeries.

"Most of the patients that I care for have more of their abdominal contents outside of their abdominal cavity than inside," she explains.

Even with hernias as large as 10 to 15 centimeters, surgeries can be performed with a robot to minimize the incision, or can be done with a mix of robotic surgery and minimal open surgery, Dr. Horne says. She uses 3D-printed models to help plan surgeries. Sometimes Botox is used to lengthen the abdominal wall muscles, she adds.

"The best part is that patients go from a dysfunctional abdominal wall to one that is functional within about a week in the hospital," Dr. Horne says.

"Seeing them at their checkup a year later is the best thing because they go from saying 'I couldn't do anything' to 'I've gone on all these trips, I've done all these things that I've wanted to do for years but haven't been able to.' They are so happy that they can put that whole thing behind them, which is great."

Provided by [Mayo Clinic](#)

APRIL 3, 2025

Successful therapy confirmed for newborns with fatal metabolic disorder

MoCD type A

by Eva Schissler, [University of Cologne](#)



Credit: Unsplash/CC0 Public Domain

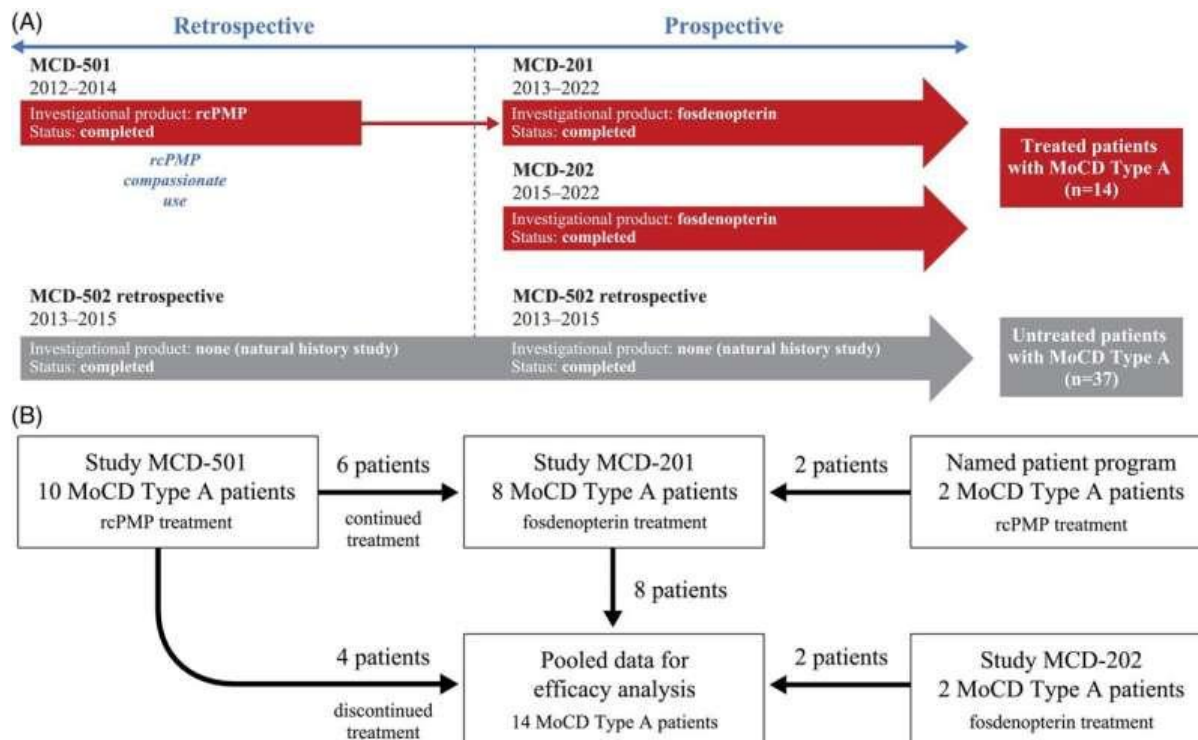
The drug fosdenopterin/rcPMP considerably improves the chances of survival and developmental progress in infants with molybdenum cofactor deficiency (MoCD type A), a rare and life-threatening genetic condition causing an inborn error in metabolism. This was shown in a clinical study recently published by an international research team led by Professor Dr. Günter Schwarz from the Institute of Biochemistry at the University of Cologne.

MoCD type A is a very rare disease. It affects around one in 200,000 to 500,000 newborns and is caused by [genetic mutations](#) that lead to the loss of the molybdenum cofactor (Moco). Ultimately, several enzymes in the newborn's metabolism are affected and are no longer able to function, thus causing very fast progressing and irreversible brain damage to affected infants, with childhood death as a typical outcome.

The clinical study has now shown that early [treatment](#) with fosdenopterin in the first days of life significantly reduces the risk of [early death](#) and promotes healthy brain development.

The study was [published](#) under the title "Treatment of molybdenum cofactor deficiency (MoCD) Type A with cyclic pyranopterin monophosphate (cPMP)" in the *Journal of Inherited Metabolic Disease*.

Without treatment, MoCD type A results in severe impairments such as seizures and severe developmental and [movement disorders](#), and often leads to early death within the first few months of life. Until recently, the only treatments available for patients with MoCD were symptomatic. They therefore targeted the symptoms and not the actual cause of the disease.



Studies used in comparison of cyclic pyranopterin monophosphate-treated versus untreated patients with MoCD Type A. Credit: *Journal of Inherited Metabolic Disease* (2025). DOI: 10.1002/jimd.70000

The present study now shows that treatment with fosdenopterin/rcPMP combats the underlying cause of MoCD type A by restoring the missing molecule cPMP. The researchers found that early administration of this drug can not only prolong life, but also promote important developmental milestones such as sitting, walking and eating behavior. The infants treated in the study showed significant improvements compared to untreated patients, with many achieving development equivalent to that of healthy infants.

Due to the rarity of the disease, it is difficult to conduct a large-scale, controlled clinical trial on the efficacy of the drug. The research group was able to demonstrate the drug's effectiveness in a mouse model back in 2004.

The first patients have been receiving treatment with the active substance fosdenopterin/rcPMP since 2008. The current publication therefore summarizes the results

of three [long-term studies](#) that tracked the development of 14 treated patients compared to 36 untreated patients in their first months of life.

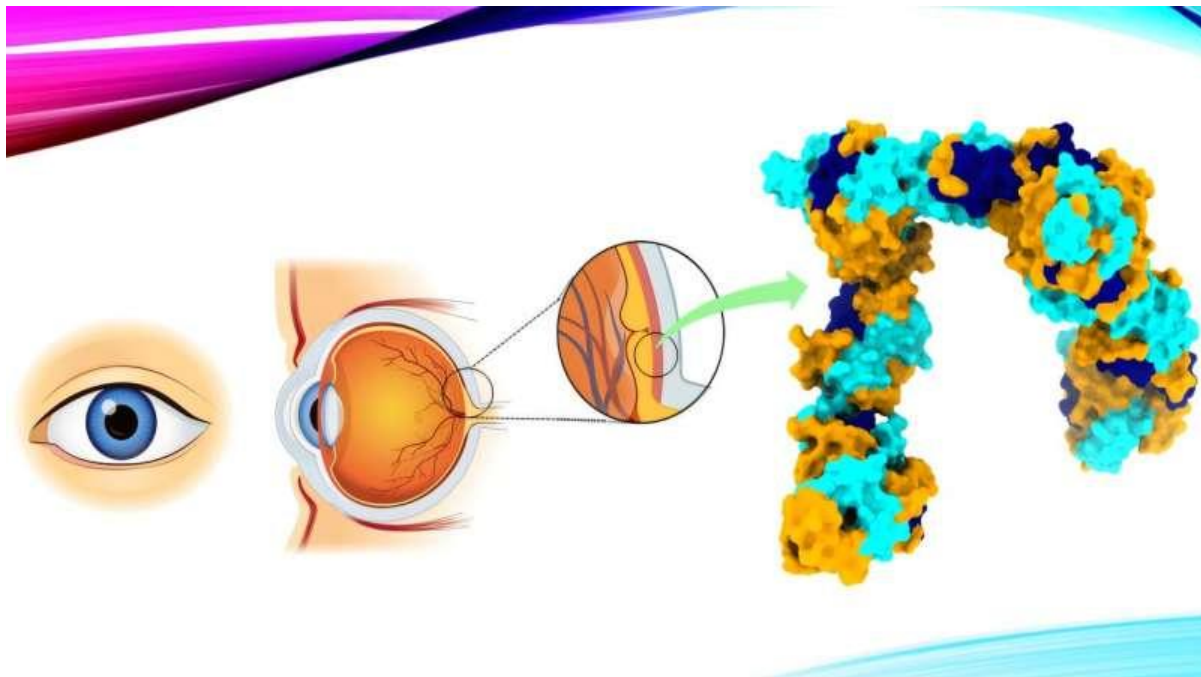
"The results show that fosdenopterin/rcPMP can significantly improve the prognosis of infants with MoCD type A," says Professor Dr. Schwarz, first author of the study. "Our results emphasize the importance of research into rare metabolic diseases, which allows us to lay the foundations for new treatment options and establish better prospects for patients."

More information: Guenter Schwarz et al, Increased Survival in Patients With Molybdenum Cofactor Deficiency Type A Treated With Cyclic Pyranopterin Monophosphate, *Journal of Inherited Metabolic Disease* (2025). DOI: [10.1002/jimd.70000](https://doi.org/10.1002/jimd.70000)
Provided by [University of Cologne](#)

APRIL 4, 2025

Molecular messenger of sight: Scientists investigate protein structure RBP3's role in vision

by [Polish Academy of Sciences](#)



Eye, retina, and surface rendering of cryoEM structure of RBP3. Credit: Nelam Kumar
Scientists have for the first time looked deep into the protein structure that may determine our vision—and discovered that it is much more dynamic than previously thought. RBP3 not only changes its shape depending on its load but may also play a role in protecting the retina from diseases ranging from diabetic retinopathy to retinitis pigmentosa.

Retinol-binding protein 3 (RBP3) is a glycoprotein of about 140 kDa, found in the intercellular space of the retina. It plays an important role in the transport of retinoids—molecules essential for properly functioning the vision process. Although the existence of RBP3 has been known for years, its structure and precise mechanisms of action have remained unknown until now. The lack of this information has been a significant gap in research on eye diseases, especially those leading to irreversible vision loss.

Retinitis pigmentosa (RP) is one such disease—insidious, progressive, and still incurable. It affects millions of people worldwide, leading to the gradual loss of photoreceptors and blindness. Previous studies suggested that abnormalities in RBP3 function may be one of the contributing factors to the development of the disease, but detailed knowledge of its structure and mechanism of action was lacking. The new findings shed light on this mystery, opening the way to potential therapies that could slow or even stop retinal degeneration.

An international team of scientists, including researchers from ICTER, used modern structural analysis methods to obtain for the first time an image of the native structure of RBP3 with high accuracy. The results are published in the journal *Open Biology* in a paper titled "[CryoEM structure and small-angle X-ray scattering analyses of porcine retinol-binding protein 3](#)."

"For the first time, we have managed to capture the full structure of native RBP3 in pigs with a resolution of 3.67 Å. This is an important step in understanding the function of this protein, especially in the context of its role in the transport of retinoids and [fatty acids](#) in the eye," says Dr. Humberto Fernandes from ICTER.

Why is RBP3 so important?

Vision is a process that begins with the conversion of light into an electrical signal by photoreceptors in the retina. A key element of this transformation is the visual cycle—a complex chain of chemical reactions in which retinoids, or derivatives of vitamin A, play an essential role. However, for them to effectively perform their function, they must be transported between different cells of the retina. This transport is the responsibility of the retinol-binding protein 3 (RBP3), which acts as a "courier" delivering retinoids to where they are needed.

RBP3 is found in the interphotoreceptor extracellular matrix (IPM), the space between the retinal pigment epithelium and the photoreceptors. This is where the transport of molecules necessary for the proper functioning of the eye takes place, including oxygen, nutrients, and retinoids.

RBP3 plays a key role in delivering all-trans-retinol (at-ROL) from photoreceptors to the retinal pigment epithelium, where it is converted into 11-cis-retinal (11c-RAL), a key molecule for vision. 11c-RAL then returns to the photoreceptors, where it binds to opsins to form light-sensitive pigments that enable vision. Without RBP3, this transport cycle would

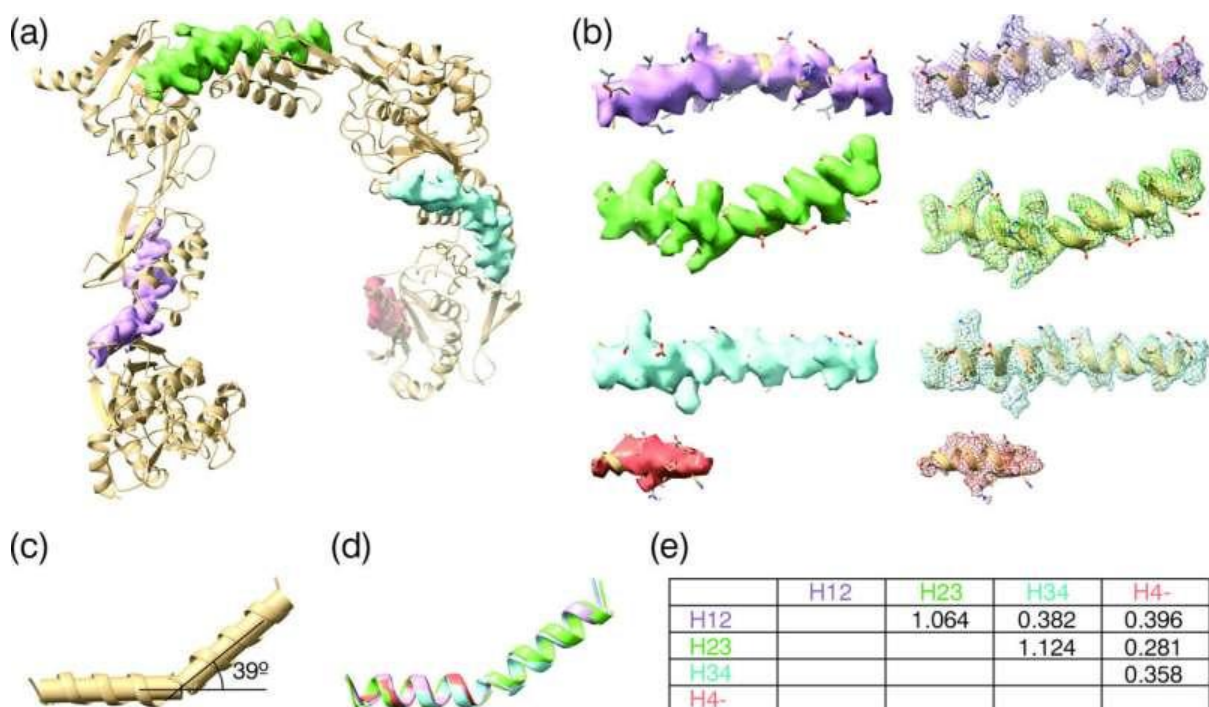
be much less efficient, which could lead to [retinoid](#) deficiencies in photoreceptors and, ultimately, retinal degeneration.

Structurally, RBP3 is a large glycoprotein consisting of four retinoid-binding modules. Each of these modules has a distinctive structure that allows it to interact with a variety of molecules, including retinoids and fatty acids such as docosahexaenoic acid (DHA). However, it has not been clear yet how exactly this protein interacts with its ligands and whether its structure changes depending on the type of molecule being transported.

However, it is known that RBP3 has additional protective functions. It protects retinoids from degradation under the influence of light by limiting their oxidation and disintegration. Its presence in the IPM stabilizes the biochemical environment of the retina, which is crucial for eye health. Moreover, mutations in the gene encoding RBP3 are associated with several eye diseases, including RP and some forms of myopia.

How was the study conducted?

To capture the three-dimensional structure of RBP3, the researchers used [cryo-electron microscopy](#) (cryoEM), a technique that allows for obtaining images of biomolecules in a nearly native state at cryogenic temperature. The study also used small-angle X-ray scattering (SAXS) analysis, which allowed for determining the conformational changes of the protein in solution.



Modules-connecting helices. (a) Cartoon representation of the refined atomic model of pRBP3 with the cryoEM map displayed for just the modules-connecting helices, with surface representation of the density. (b) Enlarged representation of the individual helices, and additional a mesh representation of the density. (c) Angle generated by the kink in the

helix (H12) joining Modules 1 and 2. (d) Overlap of the four helices to highlight the small rmsd and conserved kink position. (e) Rmsd in Å of the four helices that connect the four pRBP3 modules. Credit: *Open Biology* (2025). DOI: 10.1098/rsob.240180

The first step was to obtain the protein in its native form. To do this, the researchers isolated RBP3 from pig retinas obtained from local slaughterhouses. The tissues were stored in conditions that minimized protein degradation—on ice, in the dark. RBP3 was then purified using advanced chromatographic techniques, including affinity chromatography, ion exchange chromatography, and gel filtration. Each step of the procedure was aimed at obtaining a stable and functional form of the protein, which was crucial for further studies.

After obtaining pure samples of the protein, the researchers began experiments using cryo-electron microscopy. RBP3 samples were cooled to extremely low temperatures and placed in an electron beam, which allowed them to obtain hundreds of thousands of images of single molecules. Assembling these images into a three-dimensional model allowed the reconstruction of the protein structure at an unprecedented resolution of 3.67 Å.

In parallel, SAXS analysis was used, which provided additional information about the conformational plasticity of RBP3 in solution. These experiments allowed them to observe how the protein changes its shape when bound to different molecules, including retinoids and fatty acids. Thanks to this, the researchers discovered that RBP3 adopts different conformations depending on the type of cargo it transports, which may be crucial for its function as a dynamic retinoid carrier.

"This is one of the most detailed structural studies of RBP3 ever performed. By combining cryoEM and SAXS, we have gained unique insight into how this protein works," explains Dr. Fernandes.

What was found?

One of the key findings of this study is the ability of RBP3 to change shape depending on the type of molecule being bound. Experiments have shown that after binding 11-cis-retinal (11c-RAL) and all-trans-retinol (at-ROL), the protein assumes different conformations—from compact to open. SAXS analyses have shown that at higher concentrations of these retinoids, RBP3 is elongated, which may suggest the mechanism of its action as a dynamic retinoid transporter.

"This finding is particularly interesting because it suggests that RBP3 may act as a flexible retinoid carrier, changing shape to optimize the transport of these molecules between the [retinal pigment epithelium](#) and photoreceptors," says Dr. Vineeta Kaushik from ICTER.

Additional information on the plasticity of the protein was provided by molecular docking analyses, which indicated the presence of two main ligand binding sites. Interestingly, SAXS analysis showed that binding fatty acids such as DHA (docosahexaenoic acid) did not lead to significant changes in the structure of RBP3, indicating that its role in transporting these molecules may differ from that of retinoids.

This groundbreaking discovery changes our understanding of the role of RBP3 in the eye. Rather than being a passive transporter of retinoids, we are beginning to see it as an active, adaptive mechanism that can precisely regulate the delivery of key molecules to photoreceptors. Understanding these dynamics opens new avenues for research into the visual cycle and potential therapies for retinal degenerative diseases.

What's next?

The discovery of the full structure of RBP3 and its conformational plasticity opens a new chapter in research on the functioning of the visual cycle and the mechanisms leading to [retinal degeneration](#). The results may have key implications for the diagnosis and treatment of eye diseases, including diabetic retinopathy, retinitis pigmentosa, and high myopia.

Previous studies have suggested that reduced levels of RBP3 in the retina are associated with the progression of [diabetic retinopathy](#), and its stabilization could have a protective effect. Now, thanks to precise structural data, scientists can focus on developing new therapeutic strategies that would modulate RBP3 activity and could slow down the progression of this disease.

It is also possible to use RBP3 as a diagnostic biomarker, which could help identify patients at risk of vision loss at an early stage of the disease. The team plans to continue research on the dynamics of RBP3 function in both physiological and pathological conditions.

"This is just the beginning. Now that we have a 3D model of RBP3, we can study how exactly it interacts with other retinal proteins and how we can use this information to develop new treatments," concludes Dr. Fernandes.

More information: Vineeta Kaushik et al, CryoEM structure and small-angle X-ray scattering analyses of porcine retinol-binding protein 3, *Open Biology* (2025). DOI: [10.1098/rsob.240180](https://doi.org/10.1098/rsob.240180)

Journal information: [Open Biology](#)

Provided by [Polish Academy of Sciences](#)

APRIL 4, 2025

AI-driven clinical recommendations may aid physician decision making to improve quality of care

by [American College of Physicians](#)



Credit: CC0 Public Domain

A study comparing artificial intelligence (AI)-driven clinical recommendations to physician decision-making for common acute complaints in a virtual primary care setting found that AI support has the potential to improve quality of care when implemented responsibly.

In the study, AI recommendations were largely rated as superior to physicians by expert adjudicators. Trained on a massive amount of high-quality, real-world [clinical data](#), which is far beyond what any [physician](#) would see in their lifetime, AI uses modeling to calculate real probabilities for diagnosis and referrals and then derives treatment plans from these probabilities based on the latest medical guidelines and protocols.

Combined with human physician discernment, this approach could improve [patient care](#) and outcomes.

The findings, presented at the breaking news scientific plenary session "New in *Annals of Internal Medicine*: Hear it First from the Authors," held at the New Orleans Ernest N. Morial Convention Center during the [American College of Physicians' \(ACP\) Internal Medicine Meeting](#) 2025, are also published in *Annals of Internal Medicine*.

Researchers from Tel Aviv University, Cedars-Sinai Medical Center, and K Health, Inc. compared initial AI-driven clinical recommendations to final physician decision-making at Cedars-Sinai Connect, a virtual primary care clinic run by Cedars-Sinai, leveraging the K Health Clinical AI platform.

To evaluate AI versus physician performance, four expert adjudicators reviewed 461 real-life primary care visits involving acute urinary, vaginal, respiratory, eye, and dental chief complaints, conditions for which AI has shown high diagnostic accuracy, and used a checklist to rate both the AI and physician recommendations.

Patients described their symptoms through an AI-guided medical intake with their [medical records](#) automatically synced from the EHR, allowing physicians to review the AI-generated diagnosis, treatment recommendation, and any suggested prescriptions, labs, or referrals before the virtual visit where doctors would make the final clinical decision.

Physicians could access the initial AI decisions but the researchers were unable to determine whether they did before making their final care decisions. Overall, the expert adjudicators rated the AI's recommendations higher than physicians' decisions.

In about two in three cases, doctors made the exact same clinical decisions as the AI. In the remaining one in three cases, AI recommendations were rated as superior twice as often as they were inferior. AI received only about half as many "potentially harmful" ratings compared to physicians.

According to the researchers, these findings demonstrate the strengths of AI and how it complements human practice. AI is designed to follow medical guidelines much more strictly and can pick up on subtle details or medical record notes that doctors may miss. However, doctors have a clear advantage in clarifying symptoms during the visit.

Where AI and good doctors align is in their level of caution. If AI is not confident, it will not make a [recommendation](#), which is what happened in about 20% of cases.

The author of an accompanying editorial from Tufts University School of Medicine points to the study strengths while offering notes of caution about incorporating AI into medical practice.

A common-sense approach to using AI may include using it to confirm physician diagnoses and therapeutic recommendations rather than making them.

If discrepancies exist, then physicians should seek to determine why, which can be the seeds for improving the AI tool. The author emphasizes that to enhance clinical relevance, experienced clinicians should be involved in AI program development.

More information: Artificial Intelligence Recommendations versus Physician Decisions: Comparison in Remote Visits, *Annals of Internal Medicine* (2025). DOI: [10.7326/ANNALS-24-03283](https://doi.org/10.7326/ANNALS-24-03283)

Editorial: *Annals of Internal Medicine* (2025). www.acpjournals.org/doi/10.7326/ANNALS-25-00472

Journal information: *Annals of Internal Medicine*
Provided by [American College of Physicians](#)

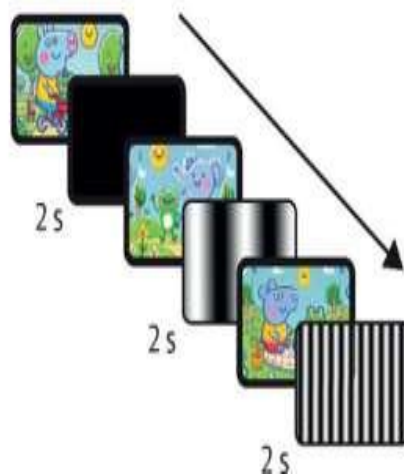
APRIL 4, 2025

'Curiosity-driven research' led to a recent major medical breakthrough, but it's under threat

A PopCSF test



B ssVEP



Methods to measure visual acuity and visual evoked potentials. Credit: *The Lancet* (2025). DOI: [10.1016/S0140-6736\(24\)02812-5](https://doi.org/10.1016/S0140-6736(24)02812-5)

Earlier this year, news broke about [doctors in London curing blindness in children with a rare genetic condition](#).

The genetic condition was a severe, albeit rare, form of retinal dystrophy. It causes severe sight impairment and can be caused by defects in many different genes.

In this case, the four [young patients](#) had mutations in the gene encoding AIPL1. This accounts for up to 5% of infants affected by this condition, and has no treatment.

In this study, published in [The Lancet](#), a team from the [Moorfields Eye Hospital and University College London Institute of Ophthalmology](#) injected a new copy of the gene AIPL1 into one eye of each patient to replace the defective one. The four children in the study showed improved functional vision without serious adverse effects.

The story of this incredible breakthrough actually begins 132 years ago. It highlights the importance of research done not for any clear application in the world—just curiosity. But around the world, this kind of research is [under threat](#).

Understanding the world—just for the sake of it

Curiosity-driven research is exactly what it sounds like: research driven by the goal of understanding nature without regard for application. It has many aliases. "Blue-sky research," "discovery science" and "basic science" are all terms commonly used to describe this approach.

This kind of research differs from "[mission-directed research](#)", which focuses primarily on practical applications and whose goals are set by governments and industry.

The logic behind curiosity-driven research is that understanding how things work will inevitably lead to discoveries that will fuel innovation.

Historically, this has led to transformational discoveries. Another recent example is the [2023 Nobel Prize in Physiology or Medicine](#), which was awarded to Katalin Karikó and Drew Weissman for discoveries that enabled the development of effective mRNA vaccines against COVID.

The recent study in *The Lancet* follows more than a century of curiosity-driven discoveries culminating in these four children receiving their life-changing injections.

Sketching the structure of the retina

The kind of medical intervention used on these patients is called a [gene therapy](#).

In this case, the cause of the condition is a defect in a single gene. This defect leads to the malfunction of an individual protein in the eye that is required for vision. The approach essentially is to provide a working copy of that gene to the eye, to restore function. This requires not only the technology to deliver the therapy, but the underlying knowledge of how AIPL1 functions in normal vision.

This knowledge dates back to 1893, when the pioneer of modern neuroscience, Santiago Ramon y Cajal, exquisitely sketched the structure of the retina—the light-sensitive tissue at the back of the eye.

In the 132 years since, our knowledge of how this tissue converts light into an electrical signal for our brain to interpret as vision has significantly advanced. We now understand a lot about how this works.

This foundational knowledge also means we know precisely why a dysfunctional AIPL1 gene leads to severe vision impairment. It also enables us to predict that providing a working version could improve vision. Armed with this knowledge, we have an engineering problem. How do we get a working copy into the eye?

In this case, the working copy of AIPL1 was delivered by an adeno-associated virus, or AAV. These were first discovered in the [mid-1960s](#), and without realizing their therapeutic potential, several research groups dedicated themselves to understanding their biology.

An AAV was first used in a human patient in 1995 for the treatment of [cystic fibrosis](#). Without this curiosity-driven research they would not have been developed into a [gene therapy](#) platform. This is how [most modern therapies have emerged](#).

Protecting curiosity-driven research

This is one of hundreds of therapies taking a similar approach. We will likely see many more stories like this in the coming decades. But I am certain we won't see any examples where we don't understand the underlying biology.

Curiosity-driven research, focused on understanding how biology works, is essential for the development of therapies to treat human disease. The history of medical advances shows us this time and time again.

Curiosity-driven breakthroughs include the discovery of X-rays as well as the antibiotic penicillin. The discovery of CRISPR/Cas9, an ancient bacterial defense, has enabled the editing of DNA with unprecedented precision. This has already led to an [FDA-approved therapy to treat sickle cell disease](#).

Australia has punched above its weight in this arena for many years. But this is [no longer the case](#).

Funding from the National Health and Medical Research Council, our largest funder of medical research, has been [falling since 2020](#). More broadly, this coincides with a decline in the [proportion of basic research](#) being funded in Australia and directly threatens our capacity for curiosity-driven innovation.

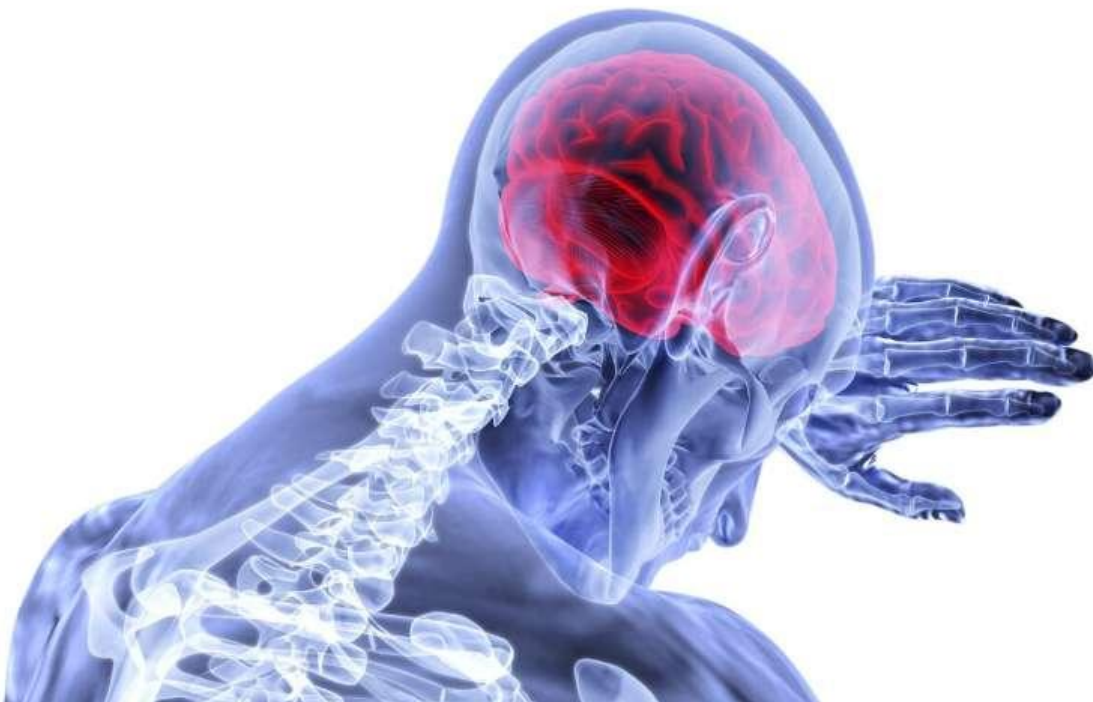
Internationally, this strong focus on practical application is repeated. For example, [83% of the European Union's €95.5 billion research funding program](#) supports mission-directed research.

In Australia, and globally, we must protect curiosity-driven research at all costs and not underestimate the vital contribution it will make to our future.

APRIL 2, 2025

Study finds nearly five-fold increase in hospitalizations for common cause of stroke

by [American Academy of Neurology](#)



Credit: CC0 Public Domain

Cervical artery dissection is a tear in an artery in the neck that provides blood flow to the brain. Such a tear can result in blood clots that cause stroke. A new study has found almost a five-fold increase in the number of U.S. hospitalizations for cervical artery dissection over a 15-year period.

The study [is published](#) online in *Neurology*.

A dissection of the artery wall is most often caused by trauma due to [motor vehicle accidents](#) but can also occur with smaller injuries. Heavy lifting has also been shown to cause dissection in some people.

"Cervical artery dissection is an important cause of [stroke](#), especially in people under 50, so it is crucial to detect it right away," said Shadi Yaghi, MD, of Brown University in Providence, Rhode Island.

"Strokes that are not fatal can lead to long-term disability, poor mental health and reduced quality of life. Our research found a dramatic increase in the number of hospitalizations for cervical artery dissection with rates rising steadily year over year."

For the study, researchers reviewed 15 years of U.S. health data to identify 125,102 people hospitalized for cervical artery dissection. Participants had an average age of 51, and just over half had a stroke at the same time as dissection.

Of all the participants, 65% were white, 10% were Black, 8% were Hispanic, 3% were Asian or Pacific Islanders, and 14% were of other racial groups.

Researchers compared the number of hospitalizations to U.S. Census data to determine the annual rate of cervical artery dissections. They then calculated the average annual percentage change in those rates.

Researchers found the number of dissections increased from 11 cases per one million people in 2005 to 46 cases per one million people in 2019, with an average annual increase of 10%. Results were similar for both female and male participants.

The average annual increase for Hispanic participants was 16%, for Black participants it was 13%, Asian participants, 12%, and white participants, 8%.

Researchers also found a greater average annual increase among people 65 and older at 12% compared to 8% for people under 65.

"Possible reasons for this nearly five-fold increase over 15 years include greater awareness of cervical artery dissection by [health care professionals](#), better access to imaging to help identify it and an overall increase in this condition for which a cause has yet to be determined," said Yaghi.

"Given the rising incidence of cervical artery dissection, our study underscores the importance of finding prevention strategies as well as new treatments to reduce the risk of stroke."

A limitation of the study was that the hospital admission data does not include undiagnosed or untreated cases, so the number of cases may be even higher.

More information: Liqi Shu et al, Incidence Trends and Risk of Recurrent Stroke of Cervical Artery Dissections in the United States Between 2005 and 2019, *Neurology* (2025). DOI: [10.1212/WNL.0000000000213548](https://doi.org/10.1212/WNL.0000000000213548) , dx.doi.org/10.1212/WNL.0000000000213548

Journal information: [Neurology](#)

Provided by [American Academy of Neurology](#)

APRIL 4, 2025

Alteplase increases frequency of functional independence after posterior circulation stroke

by Elana Gotkine



For patients with mainly mild posterior circulation ischemic stroke who do not receive thrombectomy, alteplase administered at 4.5 to 24 hours after onset results in higher frequency of functional independence at 90 days compared with standard medical care, according to a study [published](#) in the April 3 issue of the *New England Journal of Medicine*.

Shenqiang Yan, M.D., from the Second Affiliated Hospital of Zhejiang University in Hangzhou, China, and colleagues randomly assigned patients with posterior circulation [ischemic stroke](#), without extensive early hypodensity on computed [tomography](#) and with no planned [thrombectomy](#), to alteplase (0.9 mg/kg body weight; maximum dose, 90 mg) or standard medical [treatment](#) 4.5 to 24 hours after symptom onset (117 patients in each group). The primary outcome was functional independence (defined as a score of 0 to 2 on the modified Rankin scale; scores range from 0 to 6, with higher scores indicating greater disability) at 90 days.

The researchers found that the percentage of patients with functional independence at 90 days was higher in the alteplase group versus the standard treatment group (89.6% versus 72.6%; adjusted risk ratio, 1.16). The incidence of symptomatic intracranial hemorrhage

within 36 hours was 1.7% and 0.9% in the alteplase and standard treatment groups, respectively. At 90 days, 5.2% and 8.5% of patients in the alteplase and standard treatment groups, respectively, had died.

"These results support the use of alteplase in this extended time window if endovascular thrombectomy is not available," the authors write.

More information: Shenqiang Yan et al, Alteplase for Posterior Circulation Ischemic Stroke at 4.5 to 24 Hours, *New England Journal of Medicine* (2025). DOI: [10.1056/NEJMoa2413344](https://doi.org/10.1056/NEJMoa2413344)
Journal information: [New England Journal of Medicine](#)

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APRIL 7, 2025

Over 90% of women in sex work see it as their only means of survival, says study

by Teresa Bau and Rubén Permuy, [Open University of Catalonia](#)



Credit: Unsplash/CC0 Public Domain

Researchers from the Universitat Oberta de Catalunya (UOC) specializing in criminal law and criminology have published a study that breaks down stereotypes and shows the diverse and complex reality of the experiences of women (both cis and transsexual) who are sex workers. The research has been [published](#) in the journal *Revista de Derecho Penal*

y *Criminología* and is part of a project that includes several studies on the victimization suffered by sex workers, both women and men.

The project also examines the response of the criminal justice system to crimes related to sex work.

Josep Maria Tamarit, full professor of Criminal Law at the UOC and leader of the Empirical and Applied Victimology research group (VICRIM), who led the study, says that "there is great need for research in this area because it is especially sensitive to conceptions based on stereotypes or ideological positions. Political decisions about sex work must be based on an understanding of the social reality. It is also important because in Europe there are widely diverging national policies."

The diverse and complex experience of seventy-six women

Tamarit, together with Antonia Linde, professor of Criminology at the UOC and also a VICRIM researcher, and criminologists Patricia Martín Escribano and América Machado, interviewed 76 women who are or have been [sex workers](#) in the past in Barcelona and two other Catalan cities. The majority (84%) are of foreign origin, mainly from Latin America (more than 60%), although there are some from European countries, especially from Eastern Europe. The women are between 25 and 63 years old. 75% have at least secondary education, 20% have completed high school and almost 11% have a university education.

The study has addressed aspects such as the causes of their involvement in paid sex, the consequences it has for their physical and mental health and the violence they have suffered as a result. The main conclusion is that, despite the stereotypes surrounding sex work, the women's experience is very diverse, although there is a common denominator: being a sex worker has significant adverse consequences, with 90% of those interviewed having suffered violence.

The study also reveals the unique challenges faced by transsexual women, whose problems are different from those of other women, both in terms of the consequences of sex work for them and the way in which they are drawn into it and begin working.

Violence and drugs: Common denominators

Most of the women had suffered some form of violence—physical, sexual or psychological—from clients or pimps in the course of their work. But not only in this area, as Tamarit explains, "A very interesting aspect revealed by the research is that, in addition to the violence related to sex work, they have had other experiences of victimization, such as having suffered sexual abuse in childhood or violence in intimate relationships."

Regular drug use is also a factor: 67% had consumed drugs and 46% had suffered physical or psychological disorders that had affected their health as a result of their occupation.

Factors that motivate them to take drugs include the insistence of their clients, or using them as an escape mechanism or to manage the harm caused by their work.

"The use of alcohol and drugs, especially cocaine, is explained by the pressure exerted by some clients, who want to experience paying for sex and taking drugs. They may also find drugs are a way to escape from reality," said the UOC researcher.

Avoid making political decisions based on stereotypes

People who call for sex work to be banned claim that the majority of women who engage in it do so because they are victims of trafficking or exploited. However, the study shows that "only" 30% of those interviewed had been victims of trafficking or exploited; the others had started of their own free will.

"The belief that all women sex workers are exploited is based on stereotypes and reductionism, as is the belief that all women who engage in this work do so freely. Making [political decisions](#) based on this type of belief is a serious mistake," Tamarit said.

84% of the women in the study decided to become sex workers due to a lack of financial resources and 25% started because they were deceived by another person. What has been demonstrated is that women who engage in sex work "are at great risk of suffering significant consequences for their physical and [mental health](#), and public policies cannot ignore this. Nor can we ignore the importance of individual freedom as an essential principle of our political system. A serious, evidence-based debate is needed," said the UOC researcher.

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Transsexual women as sex workers: A different experience

26 of the women interviewed were transsexual, allowing the researchers to gain a different perspective on the experience of sex workers. One difference is their motivation for getting involved in this profession. In the case of cis women, financial need predominates, often after they have migrated, but they see their expectations frustrated when they arrive in Europe.

However, in the case of transsexual women, the decision is often related to a desire to affirm their sexual identity and to a sense of fun. In other cases, they experience difficulty finding work, due to the discrimination they suffer because of their condition.

A significant fact is that 61% of transsexual women began working in paid sex when they were minors, while for cis women the percentage was 20%. The percentage of transsexual

women who claim to have experienced health problems is also higher than that of cis women.

Lack of alternative employment, need for regulation

89% of women have tried to leave the profession without success and the vast majority, 94%, see it as their only option for survival, not as something they want to do permanently. Their reasons for continuing in sex work include the precarious nature of alternative types of work, or the impossibility of maintaining the income they receive from providing sexual services.

It is also significant that half of the interviewees believe that sex work should be regulated, while only 17% believe it should be banned. The reasons they give for regulation include "having greater protection and security," "so that the money belongs to the woman," and "so that society accepts it and sees us in a less negative light."

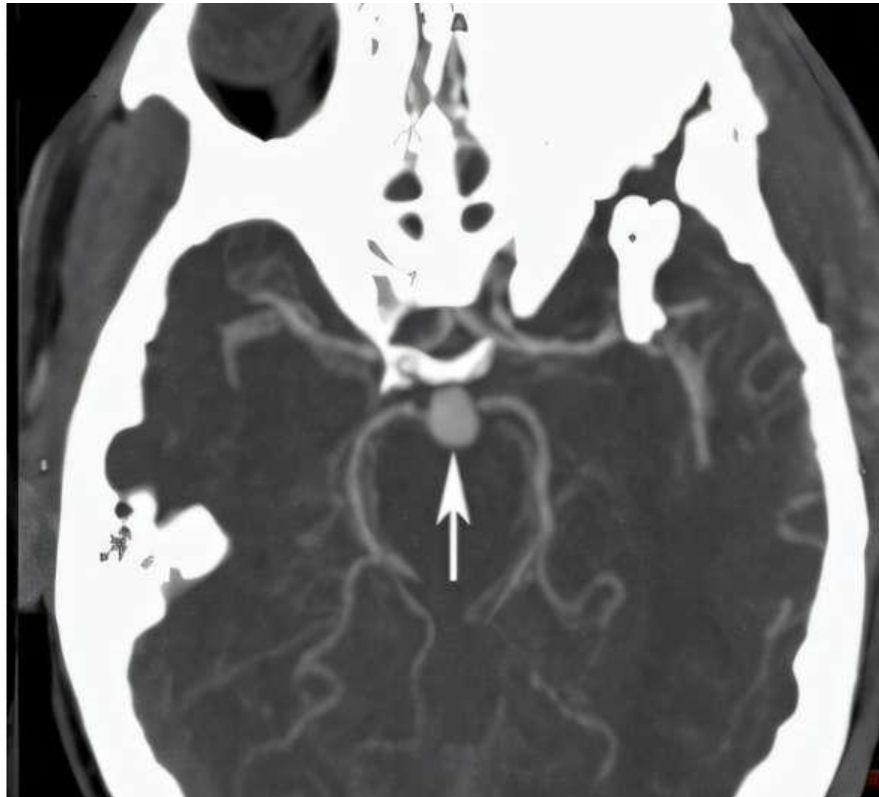
Professor Tamarit explained that "many women would like to stop doing this work, because they are aware of its negative effects on them, but they want to have the right to do it if they need to and to be recognized while they are engaged in it. They believe more legal obstacles would mean they would be forced to work in worse conditions."

The next steps in the project will be a study of men who pay for sex, an analysis of sentences in cases of homicide and crimes related to coercive sex work, and a study of criminal law. The objective is to generate reliable information for political decision-making that is based on evidence and free from the prejudices generated by stereotypes.

More information: Josep Maria Tamarit Sumalla et al, Mujeres Cis y Transexuales Que Practican Sexo de Pago: Involucración y Consecuencias, *Revista de Derecho Penal y Criminología* (2025). DOI: [10.5944/rdpc.JULIO.2024.42696](https://doi.org/10.5944/rdpc.JULIO.2024.42696)
Provided by [Open University of Catalonia](#)

APRIL 6, 2025

Brain aneurysms explained by Mayo Clinic News Network



CT angiography of basilar tip aneurysm Shazia Mirza and Sankalp Gokhale (2016-07-25). Credit: Wikimedia commons. Neuroimaging in Acute Stroke. [Attribution 4.0 International](#) (CC BY 4.0)

Each year, nearly half a million people worldwide die from brain aneurysms. In the U.S., an estimated 6.7 million people have an unruptured brain aneurysm, which means about one in 50 people might have one.

A [brain aneurysm](#), also called a [cerebral aneurysm](#), is a bulge or ballooning in a blood vessel in the brain. Dr. Chris Fox, a Mayo Clinic neurosurgeon, says there are two broad categories of aneurysms: ruptured aneurysms, which are neurosurgical emergencies, and unruptured aneurysms, where there is time to establish a treatment plan that may involve multiple options.

"For unruptured aneurysms, we have the ability to plan and make a decision on the best treatment upfront in a nonemergent situation," says Dr. Fox.

But when a brain aneurysm ruptures, prompt medical attention is required.

"Typically, when we see a patient with a [ruptured aneurysm](#), we treat it as quickly as possible. That's usually within a matter of hours because there's a risk that the aneurysm can rerupture," he says.

Symptoms

Symptoms of a ruptured aneurysm can include severe head pain, nausea, vomiting, confusion and loss of consciousness.

"The classic presentation for a ruptured aneurysm is that a patient has the worst headache of their life," Dr. Fox says.

Brain aneurysms are more common in women, and there may be a [genetic component](#) because aneurysms can run in families.

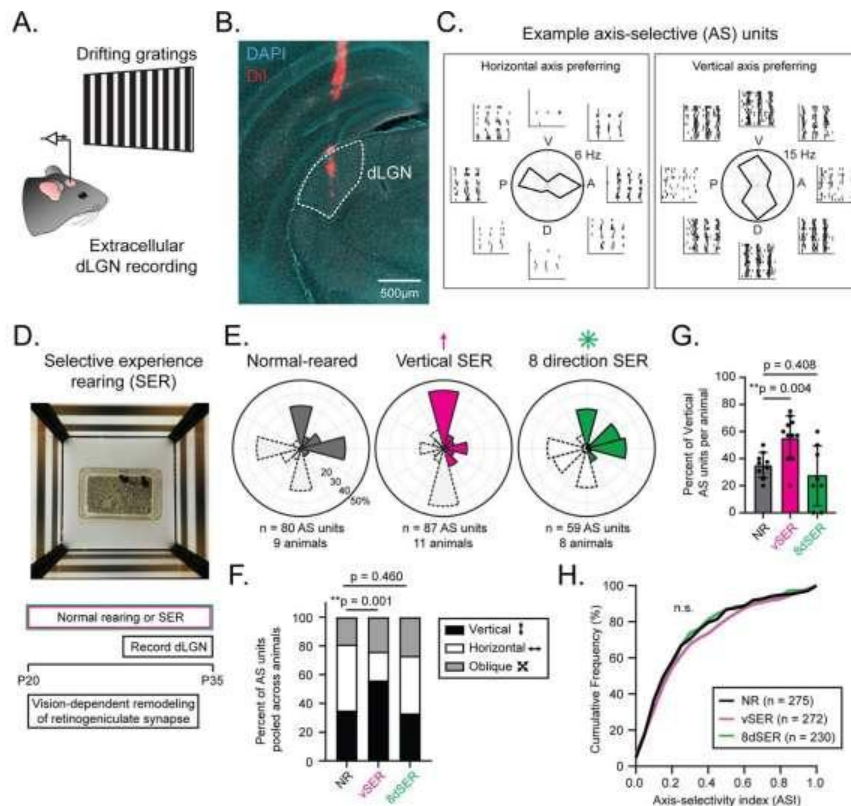
"But smoking and [hypertension](#) are two of the biggest risk factors for causing an aneurysm or having an aneurysm form," says Dr. Fox.

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APRIL 7, 2025

The thalamus: A potential therapeutic target for neurodevelopmental disorders

by [Children's Hospital Boston](#)



Selective experience rearing (SER) alters the tuning properties of dLGN neurons. Credit: Takuma Sonoda et al

Years ago, as a neurology resident, Chinfai Chen, MD, Ph.D., cared for a 20-year-old woman who had experienced a very small stroke, affecting only the thalamus.

"It was so tiny that she wouldn't have noticed any symptoms had the stroke been in any other area of the brain," says Chen, who is now an investigator at the F.M. Kirby Neurobiology Center at Boston Children's Hospital. "But she lost her ability to form [new memories](#). I could talk to her for hours, leave the room, come back, and she wouldn't remember me."

The [cerebral cortex](#) and hippocampus are traditionally viewed as the key structures in memory, but in this patient, these areas were undamaged. The experience made Chen curious to learn more about the thalamus.

Located deep in the brain, the thalamus is the brain's central relay hub, receiving information from the outside world and transmitting it to many areas of the cortex. Perhaps because it's harder to access, it's been underexplored. But Chen thinks the thalamus is critical to cognitive function, especially during early development when [brain connections](#) are first forming.

Neuronal connections—and the ability to update them as needed—have mainly been studied at the cortex level. In a study just [published](#) in the journal *Neuron*, Chen and her colleagues show that the thalamus can update its own connections, independently of the cortex.

Studying connections in the thalamus

Newer technologies that can selectively activate and monitor cells are now affording a better glimpse of the thalamus. "There's been an explosion of studies of smaller and deeper brain areas that were not well studied before," says Takuma Sonoda, Ph.D., in the Chen Lab, the study's first author.

The researchers placed mice in a controlled visual environment, presenting them with horizontal lines moving in different directions (vertically, horizontally, or diagonally). They then recorded the activity of neurons in the lateral geniculate nucleus (LGN), the part of the thalamus that relays [visual information](#) to the visual cortex.

The recordings showed that cell populations within the LGN changed their response patterns based on the animals' visual experience. This finding was unexpected as plasticity, the ability of neuronal connections to change, has traditionally been attributed to the cortex.

"Providing different experiences can change the connectivity of the thalamus, and these changes in connectivity can be long-lasting," explains Chen. "This was especially true during development, but we also saw plasticity in adult mice."

Notably, the circuits that changed were those connecting the retina to thalamus, not those connecting the cortex to thalamus—evidence that the thalamus was rewiring.

A new angle on neurodevelopmental disorders

Chen believes her team's findings are relevant to children with [neurodevelopmental disorders](#), which often involve altered brain connections. Some therapies—such as repetitively exposing children with autism to different situations to get them accustomed to them—may be changing these connections, strengthening certain pathways over others.

"If you want to alter connectivity across many cortical areas, it may be beneficial to intervene at the level of the thalamus, before signals are broadcast throughout the brain," says Chen. She suggests that one way to influence connectivity in the thalamus is by modulating neurotransmitters, using existing drugs.

"Neuromodulators could alter the processing of sensory information including visual processing, which can be affected in neurodevelopmental disorders," she says. "A lot of studies are looking at expression patterns of neuromodulatory receptors on thalamic neurons, and it's possible we could find pharmaceuticals that are more specific to the thalamus."

The thalamus may also be a more strategic target to focus on in improving cognitive function. "If you could change connectivity in the thalamus before information is transmitted throughout the rest of the brain, you could target one place rather than multiple areas of the brain. The [thalamus](#) is a very key area of the brain that shouldn't be neglected."

More information: Takuma Sonoda et al, Experience influences the refinement of feature selectivity in the mouse primary visual thalamus, *Neuron* (2025). DOI: [10.1016/j.neuron.2025.02.023](https://doi.org/10.1016/j.neuron.2025.02.023)

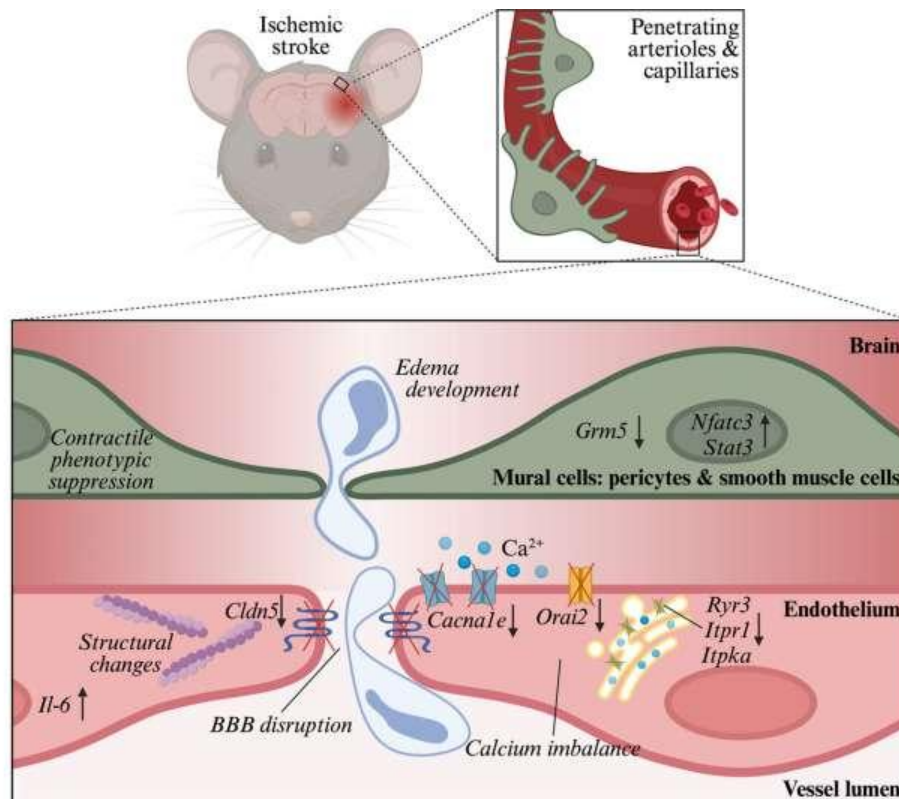
Journal information: [Neuron](#)

Provided by [Children's Hospital Boston](#)

APRIL 7, 2025

Brain blood vessel response after stroke mapped, suggesting better treatment outcomes

by [Aarhus University](#)



Graphical Abstract. Credit: *Stroke* (2025). DOI: 10.1161/STROKEAHA.124.048085
Every year, millions of people around the world suffer ischemic strokes that block blood flow to a region of the brain. Restoring blood flow rapidly is decisive and can save lives. But paradoxically, it can also lead to further damage to the blood vessels in the brain.

But thanks to new Danish research, we now have new and detailed insight into what happens in the [blood vessels](#) of the brain after [blood flow](#) is restored. The paper is [published](#) in the journal *Stroke*.

"This is the first time we've looked at how the brain's blood vessels react to a stroke at the [molecular level](#) in such detail. Our results show that the blood vessels become more permeable, which makes it easier for harmful substances to enter the brain," explains Line

Mathilde B. Hansen, a postdoctoral researcher at the Department of Biomedicine, who is one of the lead authors of the study.

A double-edged sword

The study also shows that reopening the blood vessels triggers a significant inflammatory response. Inflammation helps the body repair damage—but it can also exacerbate the [brain damage](#) and prolong the patient's rehabilitation.

"Inflammation is necessary, but it's about balance. If we can find a way to reduce the inappropriate effects of inflammation without interfering with the body's ability to repair itself, we can potentially help many patients to a better prognosis," explains Professor Vladimir Matchkov, the study's second lead author.

The treatment of the future

The research team behind the study has developed a publicly available web tool that researchers all over the world can use to identify significant changes in [gene expression](#) after a stroke.

"We hope that this resource will be an effective tool that will help quickly identify the key molecular signals involved and new, targeted therapies," Matchkov says.

He hopes that in the long term, researchers will be able to develop more effective and more precise forms of treatment for [stroke patients](#).

"The goal is clear: to improve the quality of life for millions of patients all over the world," Matchkov says.

More information: Line Mathilde Brostrup Hansen et al, Spatial Transcriptomics and Proteomics Profiling After Ischemic Stroke Reperfusion: Insights Into Vascular Alterations, *Stroke* (2025). DOI: [10.1161/STROKEAHA.124.048085](https://doi.org/10.1161/STROKEAHA.124.048085)

Journal information: [Stroke](#)
Provided by [Aarhus University](#)

APRIL 7, 2025

Medically tailored meal programs could yield significant health care savings across 49 US states

by [Tufts University](#)



Simulation predicts that prescriptions for foods tailored to support millions of Americans with diet-sensitive conditions would reduce related expenses. Credit: Alonso Nichols/Tufts University

While Food is Medicine programs are successfully helping patients manage their health, many initiatives are small-scale pilots, providing treatment to several hundred or several thousand patient.

Results of these studies have demonstrated that these nutrition-based food prescription programs improve a range of health outcomes for patients with diet-sensitive conditions, while also potentially saving in medical costs, suggesting wider implementation and investment would lead to long-term savings and a healthier population.

To quantify the potential impact of one such intervention, a team at the Food is Medicine Institute at the Gerald J. and Dorothy R. Friedman School of Nutrition Science and Policy at Tufts University developed a simulation model to predict how medically tailored meals—designed by registered dietitians to meet the nutritional needs of individuals with complex health conditions—would affect patients and health care systems across all 50 states.

[Their analysis](#), published in the journal *Health Affairs*, estimated that nationwide implementation could save approximately \$32.1 billion in health care costs in the first year alone and prevent more than 3.5 million hospitalizations annually related to complications from diabetes, heart disease, and cancer.

"The most striking finding is that medically tailored meals, assuming full uptake by eligible individuals, were cost-saving in 49 of 50 states, highlighting their potential to reduce both financial and health burdens," said first author Shuyue (Amy) Deng, a doctoral student at the Friedman School.

"These results emphasize the potential for policymakers to integrate medically tailored meals into health care coverage at scale."

The simulation was run 1,000 times to account for uncertainties. The state-by-state analysis showed a net health and cost benefit for medically tailored meal programs nationwide, with important variations based on each state's health care costs, infrastructure, policies, hospitalization patterns, and rates of diet-sensitive conditions.

Among all states, Connecticut showed the highest annual per-patient savings at \$6,299, followed by Pennsylvania (\$4,450) and Massachusetts (\$4,331). Alabama was the only state where implementing medically tailored meals would be cost-neutral but still yield health benefits for residents.

The researchers estimated that more than 7 million Americans across the country would qualify for receiving medically tailored meals—from as high as 1,221,000 in California to 18,000 in Alaska—based on having both a diet-sensitive condition like diabetes, cardiovascular diseases, or cancer and limitations in day-to-day activities, such as preparing meals or going shopping.

Among this population, the model calculated that the average annual health care expenditure is around \$30,900 per person, with each person experiencing an average of 0.53 hospitalizations per year.

The simulation model also evaluated how many patients would need to receive medically tailored meals to prevent one hospitalization. Maryland required the fewest (2.3 patients), and Colorado required the most (6.9 patients). The reasons for state-to-state differences varied, impacted by factors ranging from the health of each state's population to the infrastructure of their hospital payment systems. Nationally, these programs were estimated to avert over 3.5 million hospitalizations annually.

"Our findings suggest that medically tailored meals are not just good medicine—they're good economics," said senior author Dariush Mozaffarian, director of the Food is Medicine Institute at the Friedman School.

"States are excellent incubators for health care innovation. Investing in medically tailored meals could transform care for vulnerable patients in every state while creating substantial health care value."

Of the people considered in the [simulation model](#), nearly 90% were covered by Medicare and Medicaid services, and the rest by private insurers.

As of January 2025, 16 states have approved or proposed Medicaid section 1115 waivers (special permissions to test new health innovations) to cover Food is Medicine treatments, which can include pilots for medically tailored meals.

Further considerations will be needed to scale effectively, such as training providers, integrating screening and referral tools into the [electronic health records](#), and ensuring taste and nutritional quality of the meals.

This study is one of six papers by Food is Medicine Institute experts appearing in *Health Affairs* with the theme of food, nutrition, and health.

More information: *Health Affairs* (2025). DOI: [10.1377/hlthaff.2024.01307](https://doi.org/10.1377/hlthaff.2024.01307)

Journal information: [Health Affairs](#)

Provided by [Tufts University](#)

APRIL 7, 2025

Scientists discover the protein IL-17 that fights infection also acts on the brain, inducing anxiety or sociability

by [Massachusetts Institute of Technology](#)



Credit: Andrew Neel from Pexels

Immune molecules called cytokines play important roles in the body's defense against infection, helping to control inflammation and coordinating the responses of other immune cells. A growing body of evidence suggests that some of these molecules also influence the brain, leading to behavioral changes during illness.

Two new studies from MIT and Harvard Medical School, focusing on a cytokine called IL-17, now add to that evidence. The researchers found that IL-17 acts on two distinct brain regions—the amygdala and the somatosensory cortex—to exert two divergent effects. In the amygdala, IL-17 can elicit feelings of anxiety, while in the cortex it promotes sociable behavior.

These findings suggest that the immune and nervous systems are tightly interconnected, says Gloria Choi, an associate professor of brain and cognitive sciences, a member of MIT's Picower Institute for Learning and Memory, and one of the senior authors of the studies.

"If you're sick, there's so many more things that are happening to your internal states, your mood, and your behavioral states, and that's not simply you being fatigued physically. It has something to do with the brain," she says.

Jun Huh, an associate professor of immunology at Harvard Medical School, is also a senior author of both studies, which appear in *Cell*. One of the papers was led by Picower Institute Research Scientist Byeongjun Lee and former Picower Institute research scientist Jeong-Tae Kwon, and the other was led by Harvard Medical School postdoc Yunjin Lee and Picower Institute postdoc Tomoe Ishikawa.

Behavioral effects

Choi and Huh became interested in IL-17 several years ago, when they found it was involved in a phenomenon known as the fever effect. Large-scale studies of autistic children have found that for many of them, their behavioral symptoms temporarily diminish when they have a fever.

In a [2019 study](#) in mice, Choi and Huh showed that in some cases of infection, IL-17 is released and suppresses a small region of the brain's cortex known as S1DZ. Overactivation of neurons in this region can lead to autism-like behavioral symptoms in mice, including repetitive behaviors and reduced sociability.

"This molecule became a link that connects immune system activation, manifested as a fever, to changes in [brain function](#) and changes in the animals' behavior," Choi says.

IL-17 comes in six different forms, and there are five different receptors that can bind to it.

In their two new papers, the researchers set out to map which of these receptors are expressed in different parts of the brain. This mapping revealed that a pair of receptors known as IL-17RA and IL-17RB is found in the cortex, including in the S1DZ region that the researchers had previously identified. The receptors are located in a population of neurons that receive proprioceptive input and are involved in controlling behavior.

When a type of IL-17 known as IL-17E binds to these receptors, the neurons become less excitable, which leads to the behavioral effects seen in the 2019 study.

"IL-17E, which we've shown to be necessary for behavioral mitigation, actually does act almost exactly like a neuromodulator in that it will immediately reduce these neurons' excitability," Choi says.

"So, there is an immune molecule that's acting as a neuromodulator in the brain, and its main function is to regulate excitability of neurons."

Choi hypothesizes that IL-17 may have originally evolved as a neuromodulator, and later on was appropriated by the immune system to play a role in promoting inflammation.

That idea is consistent with previous work showing that in the worm *C. elegans*, IL-17 has no role in the immune system but instead acts on neurons. Among its effects in worms, IL-17 promotes aggregation, a form of social behavior. Additionally, in mammals, IL-17E is actually made by neurons in the cortex, including S1DZ.

"There's a possibility that a couple of forms of IL-17 perhaps evolved first and foremost to act as a neuromodulator in the brain, and maybe later were hijacked by the immune system also to act as immune modulators," Choi says.

Provoking anxiety

In the other *Cell* paper, the researchers explored another brain location where they found IL-17 receptors—the amygdala. This almond-shaped structure plays an important role in processing emotions, including fear and anxiety.

That study revealed that in a region known as the basolateral amygdala (BLA), the IL-17RA and IL-17RE receptors, which work as a pair, are expressed in a discrete population of neurons. When these receptors bind to IL-17A and IL-17C, the neurons become more excitable, leading to an increase in anxiety.

The researchers also found that, counterintuitively, if animals are treated with antibodies that block IL-17 receptors, it actually increases the amount of IL-17C circulating in the body. This finding may help to explain unexpected outcomes observed in a clinical trial of a drug targeting the IL-17-RA receptor for psoriasis treatment, particularly regarding its potential adverse effects on mental health.

"We hypothesize that there's a possibility that the IL-17 ligand that is upregulated in this patient cohort might act on the brain to induce suicide ideation, while in animals there is an anxiogenic phenotype," Choi says.

During infections, this anxiety may be a beneficial response, keeping the sick individual away from others to whom the infection could spread, Choi hypothesizes.

"Other than its main function of fighting pathogens, one of the ways that the immune system works is to control the host behavior, to protect the host itself and also protect the community the host belongs to," she says. "One of the ways the immune system is doing that is to use cytokines, secreted factors, to go to the brain as communication tools."

The researchers found that the same BLA neurons that have receptors for IL-17 also have receptors for IL-10, a cytokine that suppresses inflammation. This molecule counteracts the excitability generated by IL-17, giving the body a way to shut off anxiety once it's no longer useful.

Distinctive behaviors

Together, the two studies suggest that the immune system, and even a single family of cytokines, can exert a variety of effects in the brain.

"We have now different combinations of IL-17 receptors being expressed in different populations of neurons, in two different brain regions, that regulate very distinct behaviors. One is actually somewhat positive and enhances social behaviors, and another is somewhat negative and induces anxiogenic phenotypes," Choi says.

Her lab is now working on additional mapping of IL-17 receptor locations, as well as the IL-17 molecules that bind to them, focusing on the S1DZ region. Eventually, a better understanding of these neuro-immune interactions may help researchers develop new treatments for neurological conditions such as autism or depression.

"The fact that these molecules are made by the immune system gives us a novel approach to influence brain function as a means of therapeutics," Choi says. "Instead of thinking about directly going for the brain, can we think about doing something to the immune system?"

More information: Inflammatory and anti-inflammatory cytokines bidirectionally modulate amygdala circuits regulating anxiety, *Cell* (2025). DOI: [10.1016/j.cell.2025.03.005](https://doi.org/10.1016/j.cell.2025.03.005). [www.cell.com/cell/fulltext/S0092-8674\(25\)00278-8](https://www.cell.com/cell/fulltext/S0092-8674(25)00278-8)

Brain-wide mapping of immune receptors uncovers a neuro-modulatory role of interleukin-17E and the receptor IL-17RB., *Cell* (2025). DOI: [10.1016/j.cell.2025.03.006](https://doi.org/10.1016/j.cell.2025.03.006). [www.cell.com/cell/fulltext/S0092-8674\(25\)00279-X](https://www.cell.com/cell/fulltext/S0092-8674(25)00279-X)

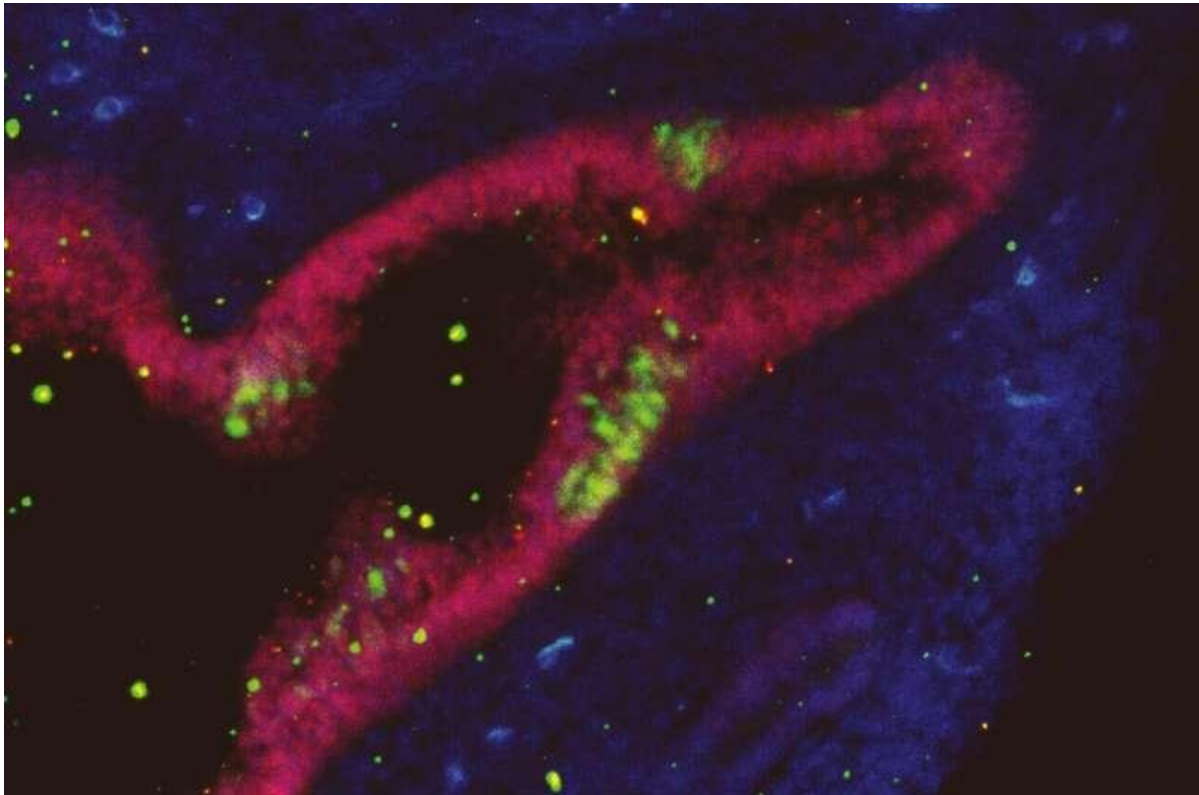
Journal information: *Cell*

Provided by [Massachusetts Institute of Technology](#)

APRIL 10, 2025

Neural stem cells outside the brain: Discovery opens new paths for regenerative medicine

By [Max Planck Society](#)



pNSCs distribute along the bronchia of mouse postnatal (image) and adult lung. These cells are specifically marked by Sox1 and co-express Sox2. Credit: MPI for Molecular Biomedicine / Dong Han

For decades, scientists assumed that neural stem cells (NSCs) only occur in the brain and spinal cord. A new international study, led by Hans Schöler of the Max Planck Institute for Molecular Biomedicine in Münster, has now refuted this assumption and discovered a new type of neural stem cell outside the central nervous system (CNS) that opens up enormous possibilities for the development of therapies for neurological diseases. The study is [published](#) in the journal *Nature Cell Biology*.

In 2014, an article titled "Stimulus-triggered fate conversion of [somatic cells](#) into pluripotency" was published in *Nature*. This publication initially caused quite a stir because it opened up a simple way to obtain [pluripotent stem cells](#). The induction of pluripotent

stem cells without the need for viral vectors, as Shinya Yamanaka had done and for which he received the Nobel Prize, would have been too good to be true.

Although the laboratory of Schöler at the Max Planck Institute for Molecular Biomedicine, like many others, tried to repeat the experiment that described the "stimulus-triggered acquisition of pluripotency" (STAP) based on treating somatic cells with low pH. However, the generation of pluripotent cells failed regardless of the culture conditions and tissues used—and the [corresponding paper](#) was eventually retracted several months after publication.

However, to the surprise of Dong Han and Schöler, they were able to use the STAP method to obtain a rare cell population from the periphery of the central nervous system that exhibits the properties of neural stem cells (NSCs). These NSCs, called peripheral neural stem cells (pNSCs), were found in several tissues of the mouse, including the lung and tail. Once the NSC population had been identified, it became clear that treatment with low pH was not necessary to cultivate them.

ADVERTISING

A team of researchers from more than 10 laboratories in Europe, Asia and North America then examined these newly identified pNSCs in great detail: pNSCs share important molecular and functional characteristics with NSCs of the brain.

pNSCs have the same cell morphology, self-renewal and differentiation capacity as NSCs of the brain. They express several NSC-specific markers and have genome-wide transcriptional and epigenetic profiles that are consistent with those of NSCs in the brain. Furthermore, many pNSCs that migrate out of the [neural tube](#) can differentiate into mature neurons and, to a limited extent, glial cells during embryonic and postnatal development.

The discovery of pNSCs not only provides new insights into the development of the mammalian nervous system. Their existence also challenges a long-standing hypothesis in neuroscience and, because they can be grown in substantial numbers in the petri dish, opens up new possibilities for regenerative medicine.

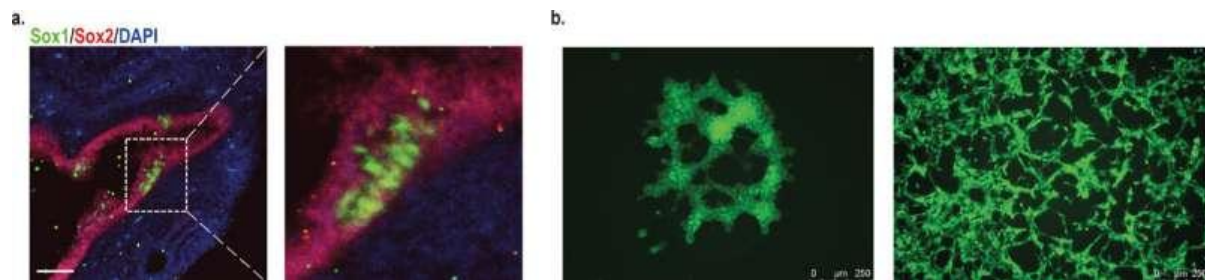
Furthermore, obtaining NSCs from the brain is not a favored method. By contrast, obtaining NSCs from other organs or tissues appears to be a viable and practical approach.

Schöler, the senior author of the study, looks back on the long road that led to this discovery: "This was the longest-running project in my career. Originally, we wanted to replicate the STAP results published in *Nature* that were published more than 10 years ago, namely, to induce pluripotent stem cells through low pH.

"Like other laboratories, we were unable to reproduce this. But fortunately, our attempts were not in vain: We found previously unknown peripheral neural stem cells, challenging the long-held dogma that neural stem cells do not exist outside the central nervous system.

Han, the lead researcher of the study, who carried out most of the experiments in this work as a member of Schöler's laboratory, emphasized the possible implications of this result: "If these cells exist in humans and can be propagated indefinitely as they can in mice, they could have enormous therapeutic potential.

"This is particularly exciting because accessible peripheral neural stem cells could provide a new avenue for neural repair and regeneration, bypassing many of the challenges associated with sourcing stem cells from the central nervous system."



Identification of pNSCs in the mouse lung: A) pNSCs line the bronchi in postnatal and adult lungs, marked by Sox1 and Sox2. B) They can be isolated and cultured in vitro for over 50 passages. Credit: MPI for Molecular Biomedicine / Dong Han

The discovery of pNSCs outside the CNS suggests a previously unrecognized level of cellular plasticity within the nervous system. In contrast to neural crest-derived stem cells, which have a limited self-renewal capacity, pNSCs closely resemble brain-derived NSCs and show the ability to sustain neurogenesis over an extended period of time.

Schöler emphasized the crucial role of interdisciplinary cooperation in making this discovery possible: "We involved many laboratories with different areas of expertise to ensure that this study is watertight. The combination of genetic lineage analysis, single-cell analysis and functional tests in vivo provides compelling evidence that these peripheral neural stem cells are a genuine and previously unrecognized component of the mammalian nervous system."

Potential impact on human health and medicine

The ability to harness pNSCs could have far-reaching implications for the treatment of neurodegenerative diseases and nerve cell repair strategies. If pNSCs exist in humans, they could provide an easily accessible source of [neural stem cells](#) that could be used in the future to treat diseases such as Parkinson's disease, spinal cord injury and other neurodegenerative disorders. Future studies will aim to establish the existence of pNSCs in humans and explore their full therapeutic potential.

The results, published in the journal *Nature Cell Biology*, thus pave the way for further research into the role of pNSCs in human biology and their potential application in the treatment of neurodegenerative diseases and in regenerative therapies.

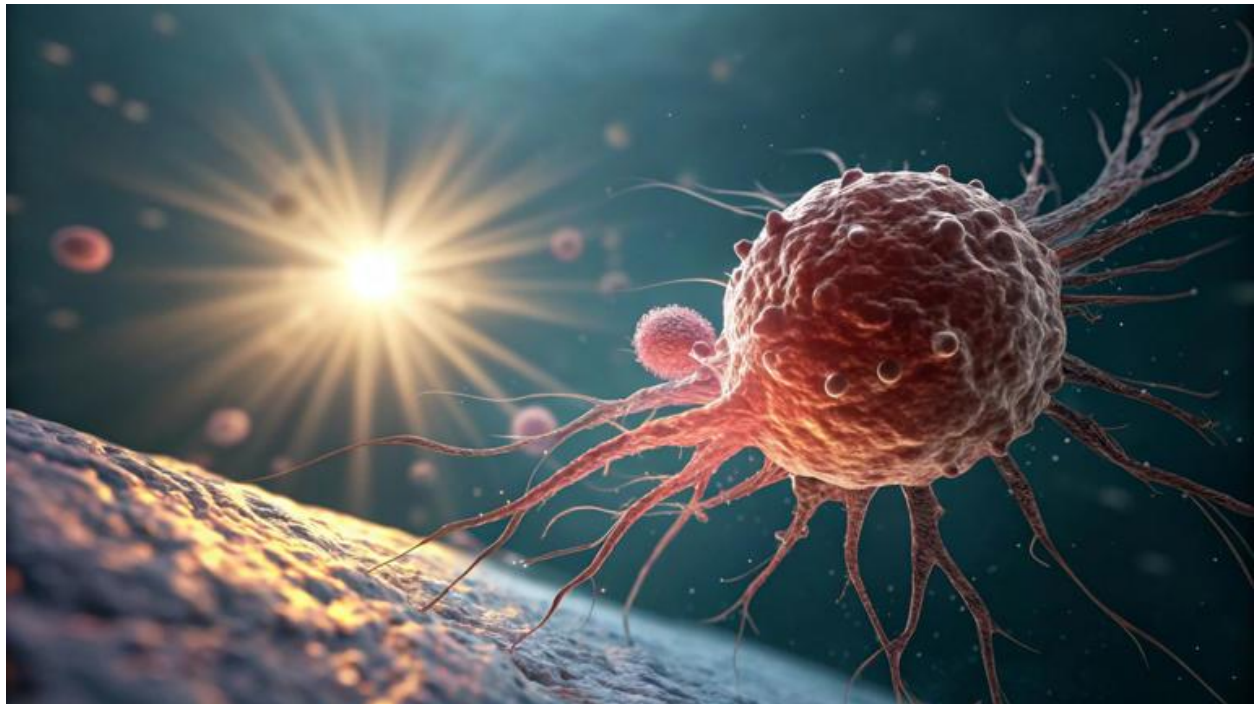
More information: Dong Han et al, Multipotent neural stem cells originating from neuroepithelium exist outside the mouse central nervous system, *Nature Cell Biology* (2025). DOI: [10.1038/s41556-025-01641-w](https://doi.org/10.1038/s41556-025-01641-w)

Journal information: [Nature](#) , [Nature Cell Biology](#)

Provided by [Max Planck Society](#)

Scientists create "smart bomb" to target cancer 🎯

Researchers have developed innovative compounds capable of precisely targeting cancer cells when activated by light. This approach, successfully tested on mice, offers promising prospects for aggressive cancers.



Scientists create "smart bomb" to target cancer 🎯

Photodynamic therapy (PDT), used for decades against certain cancers, could see a breakthrough thanks to this work. Scientists combined cyanine-carborane salts, molecules sensitive to infrared light, to eliminate tumors while preserving healthy tissue.

A precise and less toxic mechanism

The developed salts exploit a unique feature of cancer cells: the overexpression of OATP proteins. These proteins specifically capture the compounds, eliminating the need for additional targeting agents. Once activated by light, the salts generate destructive oxygen molecules for tumors.

Unlike conventional photosensitizers, these salts are rapidly eliminated from the body. Patients would no longer need to avoid light for months after treatment, a major drawback of current therapies. Tests on mouse models show a significant reduction in side effects.

Activation by infrared light also enables the treatment of deep-seated tumors. This wavelength penetrates tissues better than visible light, expanding the scope of PDT.

Potential for broader applications

Researchers are exploring ways to adapt these salts to other energy sources, such as ultrasound, to target even deeper cancers. This flexibility could extend their use to other hard-to-treat conditions.

Preclinical results, published in *Angewandte Chemie*, show complete eradication of metastatic breast tumors in mice. The compounds also act on cancer cell migration mechanisms, reducing the risk of spread.

The multidisciplinary team highlights the importance of this discovery for triple-negative breast cancers, often resistant to conventional treatments. Next steps include trials on other cancer types and protocol optimization.

Magnetism and biology join forces against cancer

Published by Adrien, April 11, 2025

Magnetism offers extremely promising perspectives in biology, particularly in mechanobiology and for various biomedical applications including cancer.

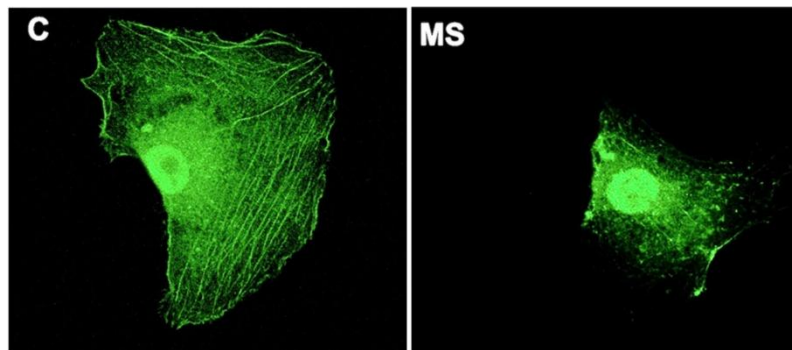
In vitro studies on three-dimensional cell assemblies have shown that the death of

different types of cancer cells (pancreatic, brain, kidney cancers, melanomas) can be triggered by magnetic stimulation of the cells. The effect is mechanically induced by applying mechanical stress to the cells through magnetic particles dispersed among them.

Researchers initially conducted studies on glioma cells (brain cancer) cultured in 2D at the bottom of culture dishes. However, these results vary significantly depending on the cellular microenvironment, which differs between 2D cultures and actual biological tissues.

Now, a new milestone has been reached by reproducing these effects on tumoroids—3D assemblies of cancer cells that are much closer to biological tissues. To maximize efficiency, it is crucial to readjust the magnetic field conditions to lower frequencies (2 to 5Hz instead of 20Hz) to adapt to the different texture of 3D environments.

A strong impact of magneto-mechanical stimulation on the cells' cytoskeleton has been demonstrated, leading to cell death.



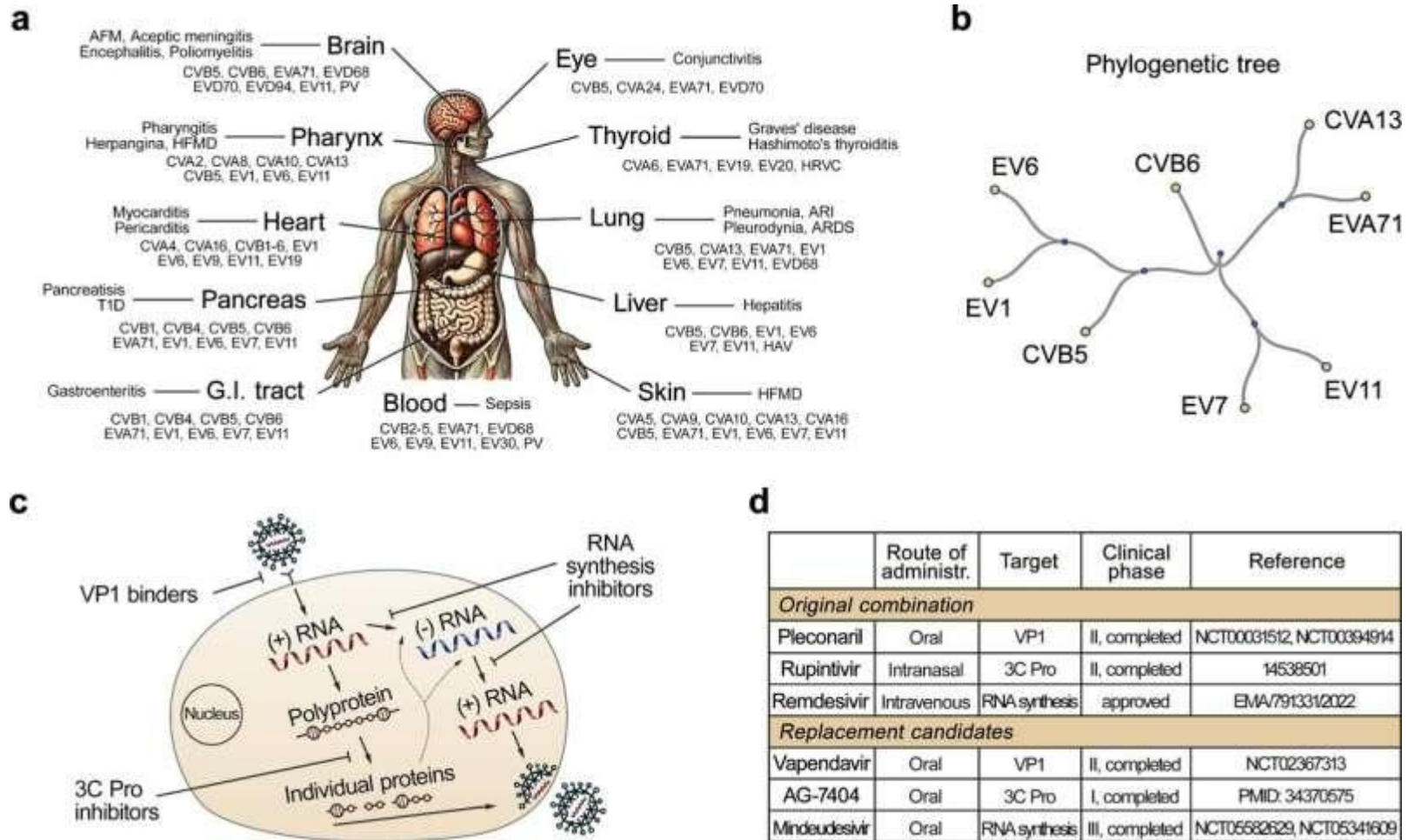
Impact of magneto-mechanical stimulation on the cytoskeleton of glioma cancer cells (left, C=control; right, MS=after magnetic stimulation). The actin fibers forming the cytoskeleton are clearly visible in the control. These fibers are destroyed after magnetic stimulation, leading to cell death.

This new study paves the way for *in vivo* testing of innovative cancer treatments using magneto-mechanical cell stimulation. This approach could be used alone or in synergy with chemotherapy.

APRIL 15, 2025

A cocktail of drugs may work against a whole family of viruses

by Steinar Brandslet, [Norwegian University of Science and Technology](#)



Orally available triple drug combination for potential treatment of picornavirus infections.

(a) Examples of picornaviruses, target organs and associated diseases. (b) Phylogenetic tree of enteroviruses used in our study. (c) The life cycle of picornavirus and inhibitors. (d) Example of published synergistic anti-enterovirus combination and orally available compound substitutes. Credit: *Cellular and Molecular Life Sciences* (2025). DOI: 10.1007/s00018-025-05581-4

Enteroviruses are a group of viruses that cause everything from the common cold to meningitis, polio, type 1 diabetes, and much more. Over 100 different types have been described by researchers, and millions of people are infected each year.

"Enteroviruses pose a significant global health problem," said Erlend Ravlo, a Ph.D. research fellow at the Norwegian University of Science and Technology (NTNU's) Department of Clinical and Molecular Medicine.

It may therefore sound a bit strange that we still haven't developed an approved general treatment or a vaccine for this group of viruses.

One explanation may be that infected patients usually recover well without medication. Sometimes, however, things can really take a turn for the worse, especially with children, which is why it is important to find a treatment method that can work against multiple enteroviruses.

Medication cocktail stops replication

Researchers are on the trail of a solution. Viruses replicate by making copies of themselves within the cells they infect, which makes for a good target where an intervention can be made.

"We have identified a combination of drugs that appears to prevent enteroviruses from replicating," said Aleksandr Ianevski at the Department of Clinical and Molecular Medicine.

The combination of drugs has not yet been tested on humans, but trials have been conducted on [human cells](#) and cultures of mini-organs in the laboratory—and the results are positive.

The work on enteroviruses has involved dozens of researchers in Norway and other European countries. The research results have now been [published](#) in the journal *Cellular and Molecular Life Sciences*.

The corresponding authors are professors Denis Kainov and Magnar Bjørås, both from the Department of Clinical and Molecular Medicine.



Erlend Ravlo, a Ph.D. research fellow at the Norwegian University of Science and Technology (NTNU) at work in the laboratory. Now he and colleagues have found promising solutions in the fight against enteroviruses. Credit: Idun Haugan, NTNU

Can be taken orally

One combination consists of three drugs already known to [health professionals](#): pleconaril, AG7404 and mindeudesivir. This combination of medicine can be taken orally.

These drugs have already been individually tested on humans, and safe doses of the combination appear to be effective against enteroviruses in cells and cultures of mini-organs.

"In addition, the combination does not alter glucose or insulin levels when tested on pancreatic cells in the laboratory," said Kainov.

This is good news for people who have or fear developing diabetes.

The researchers have also tested the combination on mini-organs that simulate hearts, and it does not appear to alter the [heart rate](#), which is, of course, also important.

"This cocktail of medicines is really promising," said Bjørås.

Tested several combinations

Finding the optimal combination of drugs took time. The NTNU researchers tried other possible treatments by testing several different mixtures.

"To find an effective treatment, we analyzed the toxicity and effectiveness of 12 known agents that can work against a wide range of enteroviruses. We tested them both

individually and in combination, and against different viruses in cell cultures," explained Kainov.

A promising combination is pleconaril, rupintrivir and remdesivir, which was effective against several viruses in lung cells and intestinal organoids. However, since rupintrivir and remdesivir cannot be taken as a single combined pill, the researchers replaced them with AG7404 and mindeudesivir. The new combination is still effective, while being more practical because it can be taken as a single pill.

"Our studies show great potential for finding broad-spectrum treatment methods against enteroviruses," said Kainov.

Bjørås emphasized that more studies are needed before we can confirm that these combinations of drugs are also effective in patients. These studies must involve several different enteroviruses, both in the laboratory and in clinical trials on humans.

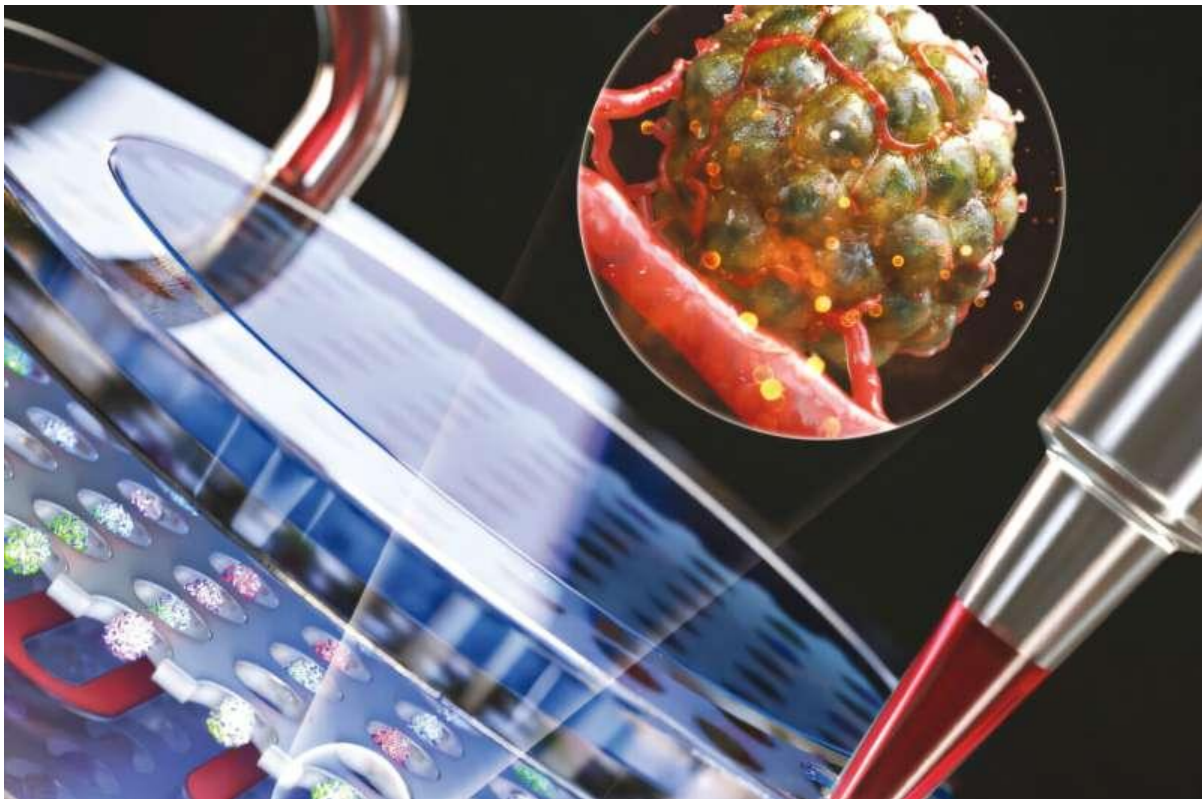
More information: Erlend Ravlo et al, Synergistic combination of orally available safe-in-man pleconaril, AG7404, and mindeudesivir inhibits enterovirus infections in human cell and organoid cultures, *Cellular and Molecular Life Sciences* (2025). DOI: [10.1007/s00018-025-05581-4](https://doi.org/10.1007/s00018-025-05581-4)

Journal information: [Cellular and Molecular Life Sciences](#)
Provided by [Norwegian University of Science and Technology](#)

APRIL 15, 2025

Microfluidic chip technology enables real-time study of cancer cell interactions with blood vessels

by JooHyeon Heo, [Ulsan National Institute of Science and Technology](#)



Credit: *Advanced Science* (2025). DOI: 10.1002/adv.202410659

Researchers have developed a cutting-edge chip technology capable of precisely mimicking the interactions between cancer cells and blood vessels, akin to the human body environment. This innovation is being hailed as a significant breakthrough in the quest for patient-specific cancer drug development.

On April 14, Professor Yoon-Kyoung Cho and her research team from the Department of Biomedical Engineering at UNIST unveiled the spheroid-endothelium interaction (ODSEI) chip, a microfluidic device designed for the large-scale real-time analysis of the interactions between [cancer cells](#) and [vascular cells](#). The research is [published](#) in the journal *Advanced Science* and was selected as a cover article.

Cancer cells require significantly more nutrients and oxygen than normal cells to proliferate rapidly. As they cannot produce these resources independently, they stimulate nearby vascular cells to draw the necessary supplies. Understanding the interactions between cancer and [blood vessels](#) is crucial for uncovering the dynamic mechanisms of cancer metastasis and drug resistance, and for formulating effective treatment strategies.

The ODSEI Chip enables the cultivation and analysis of more than 1,000 tumor spheroids alongside vascular cells. Unlike traditional closed systems, this open-structured chip allows researchers to retrieve specific spheroids at any moment for genetic analysis. This capability facilitates the tracking of how cancer cells acquire resistance through their interactions with the vasculature.

Using this technology, the research team investigated the mechanisms of resistance to tamoxifen, a breast cancer therapy. Through single-cell RNA sequencing and protein analysis, they identified IL-8 and TIMP-1 as biomarkers that could enhance drug delivery efficiency in blood vessels. They also elucidated how these signaling molecules activate survival signals in cancer cells, thus inhibiting their response to therapeutic agents and contributing to drug resistance.

Jooyoung Ro, a student who served as the first author of the study, remarked, "The chip is designed with a special coated double-layer porous membrane that allows it to maintain an open structure while simulating an environment similar to that of the human body. Although cancer cells and vascular cells are physically separated, signaling molecules can freely exchange between them, allowing for interactive dynamics. Additionally, since each cancer spheroid is cultivated in its own individual well, we can conduct independent observations and analyses without interference."

Professor Cho expressed optimism about the future applications of this technology, stating, "This opens up new avenues for effectively studying [drug resistance](#) under finely mimicked tumor microenvironments. It holds great potential as a crucial platform for the development of patient-specific treatments."

More information: Jooyoung Ro et al, ODSEI Chip: An Open 3D Microfluidic Platform for Studying Tumor Spheroid-Endothelial Interactions, *Advanced Science* (2025). DOI: [10.1002/advs.202410659](https://doi.org/10.1002/advs.202410659)

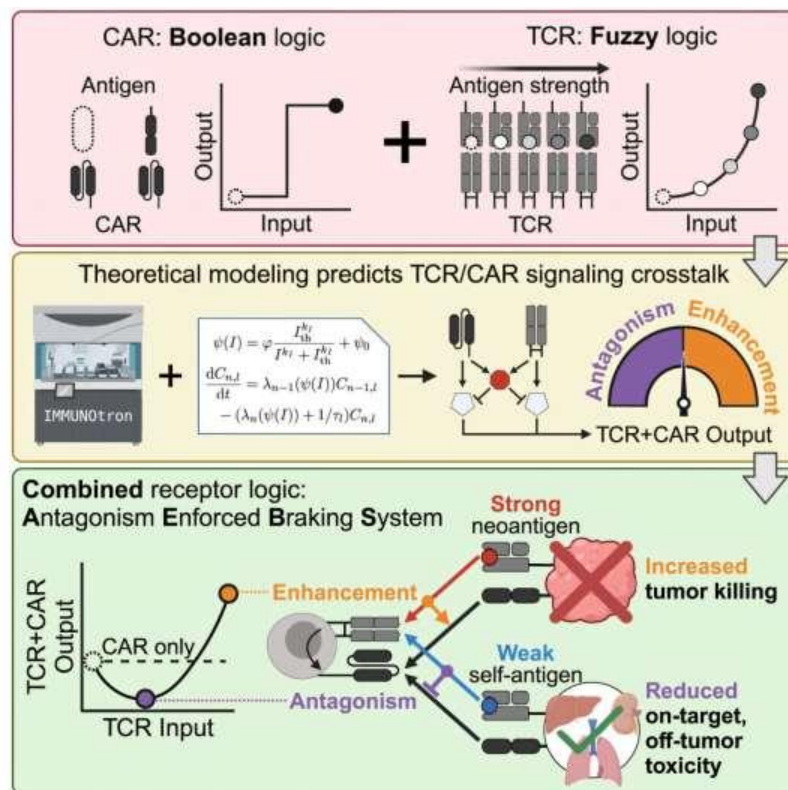
Journal information: [Advanced Science](#)

Provided by [Ulsan National Institute of Science and Technology](#)

APRIL 15, 2025

Engineered T-cells use natural receptors as 'brakes' to target cancer while sparing healthy tissue

by Catherine Couturier, [University of Montreal](#)



Credit: *Cell* (2025). DOI: 10.1016/j.cell.2025.03.017

A team of researchers has successfully created more effective immune cells that can fight cancer without damaging healthy tissue.

"This story goes back nearly 20 years," says Prof. Paul François. This biophysicist and bio-informaticist from UdeM's Department of Biochemistry and Molecular Medicine is interested in automatic learning, a field at the crossroads of various disciplines. "I'm mainly trying to develop a mathematical model for what's happening in biology in order to improve our understanding of it."

The story actually began when Prof. François attended a seminar on the immunity response detection model developed by Franco-American immunologist Grégoire Altan-Bonnet of the

U.S. National Cancer Institute. "From a mathematical viewpoint, I didn't understand what was going on. So I developed an interest in the immune system."

After many years of fruitful collaboration, the two researchers, joined by immunologist Naomi Taylor (also of the U.S. NCI) and their teams (including the other principal authors François Bourassa, Sooraj Achar and Taisuke Kondo), can now say "mission accomplished." Their findings were recently [published](#) in the journal *Cell*.

"It's really a team effort," Prof. François emphasizes. Thanks to the robotics platform developed in Dr. Altan-Bonnet's lab, in collaboration with Prof. François—"akin to an immune system microscope"—the scientists were able to study and compare the reactions of immune cells against cancer cells, and then design a mathematical model explaining and predicting the observations subsequently made in laboratory settings.

Immune system judo

In recent years, CAR-T cell immunotherapy (using chimeric antigen receptors) has revolutionized the fight against cancer. In combination with other tools (chemotherapy, radiotherapy), this new type of treatment reprograms the patient's T-cells (the "warriors" of the immune system) to attack cancer cells. "It's a bit like immune system judo," says Prof. François.

In the lab, the T-cells are modified by adding a synthetic chimeric antigen receptor (CAR). This receptor prompts the T-cells to actively destroy [cancer cells](#), although its mechanism for recognizing tumors is not very precise, so the T-cells often attack healthy tissue as well. In other words, CAR-T immunotherapy is a highly effective treatment for leukemia, where the lack of target specificity is not fatal. But it works less well with solid cancers, such as those affecting the ovaries.

"CAR-T cells attack tumors in the ovaries, but they also destroy the patient's healthy tissues, particularly the lungs. You attack the cancer, but you kill the patient," explains Prof. François.

Putting the brakes on

So what's the best way to modulate the [immune system's](#) response? The team of scientists made good use of the receptors naturally present in T-cells (TCRs), which can distinguish between healthy and cancerous cells by recognizing the different proteins on their surfaces. But TCRs are not very effective at combating tumors.

Nevertheless, the researchers successfully blended both strategies to engineer CAR cells that include TCRs. The team discovered how to induce the TCRs to act as a brake, thus mitigating the CAR cells' overly enthusiastic response. "It was long thought that the TCRs were exclusively 'accelerators,' but we showed they could be modulated to suppress the immune response instead of activating it," notes Prof. François.

Seeking a more nuanced response, Prof. François and his student François Bourassa designed a theoretical [mathematical model](#) to determine how to modulate the immune response using receptors naturally present on the surface of T-cells. "We came up with a mathematical explanation: To obtain a better immune response, you have to balance out the T-cell's brake and gas pedals."

Drawing on that idea, the team refined an immunotherapy concept known as AEBS (Antagonism-Enforced Braking System). "We're not the only ones who have tried to reprogram CAR cells to make them more effective on tumors, but we're the first ones to use natural receptors as the brake," says Prof. François.

The lab team went on to produce and test T-cells that not only ease up when they encounter healthy tissue, but also go into attack mode when they come across a tumor.

In light of such encouraging findings, a patent application is in the process of being filed; clinical trials may also be carried out. "We started off with a fairly theoretical problem and ended up using the idea to develop a treatment," says Prof. François.

More information: Taisuke Kondo et al, Engineering TCR-controlled fuzzy logic into CAR T cells enhances therapeutic specificity, *Cell* (2025). DOI: [10.1016/j.cell.2025.03.017](https://doi.org/10.1016/j.cell.2025.03.017)

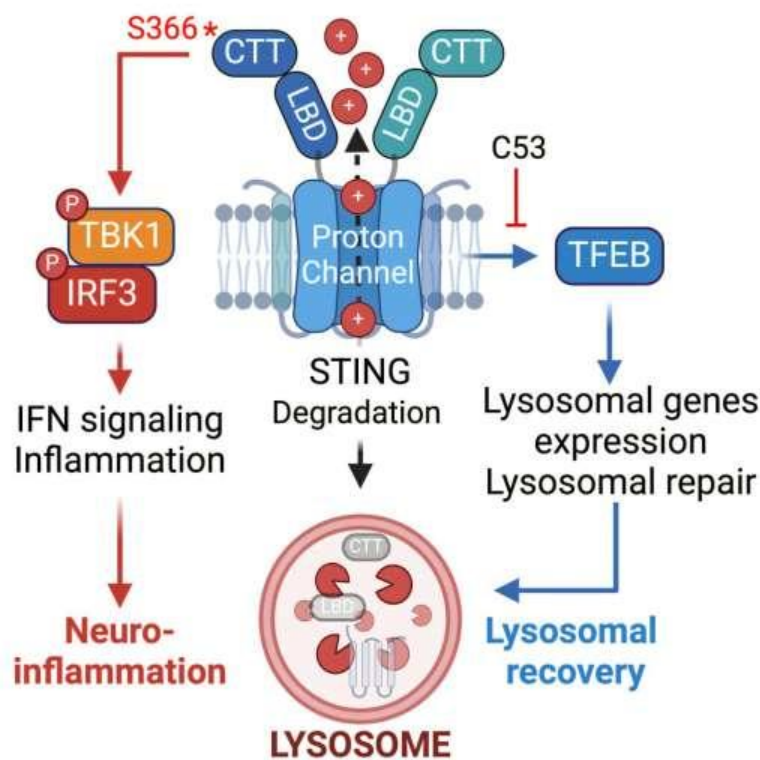
Journal information: [Cell](#)

Provided by [University of Montreal](#)

APRIL 15, 2025

STING protein aids lysosome repair, offering hope for neurodegenerative diseases

by [UT Southwestern Medical Center](#)



Graphical abstract. Credit: *Molecular Cell* (2025). DOI: 10.1016/j.molcel.2025.03.008

The STING protein, known for helping cells fight viral infections by generating inflammation, also appears to function as a quality control sensor for organelles that serve as cellular waste disposal systems, UT Southwestern Medical Center researchers found.

Their study, published in [Molecular Cell](#), helps explain critical features of diseases called [lysosomal storage disorders](#) and could eventually lead to new treatments for these and other [neurodegenerative diseases](#).

"STING is well known as an innate immune signaling protein. This study uncovered a new nonimmune function of STING," said study leader Nan Yan, Ph.D., Professor and Vice Chair of Immunology and Professor of Microbiology at UT Southwestern.

There are more than 70 known lysosomal storage disorders (LSDs). These rare neurodegenerative diseases are characterized by the dysfunction of lysosomes, cellular organelles that break down various substances ready for disposal, including proteins, nucleic acids, and even other organelles, by digesting them in acid. This dysfunction allows substances that would normally be broken down to accumulate to harmful levels.

Inflammation in the nervous system is a prevailing symptom of these disorders. But why lysosomal dysfunction causes neuroinflammation has been unclear. In a [2021 study](#), Dr. Yan and colleagues showed that STING—short for stimulator of interferon genes—drives this symptom for one LSD, Niemann-Pick disease type C1 (NPC1).

To see whether STING is involved in other LSDs, Dr. Yan's team at UTSW worked with a mouse model of an LSD called Krabbe disease with the same mutation found in human disease. In some of these animals, the researchers also deleted the gene for STING.

Compared with animals that carried neither genetic defect, those with only the Krabbe mutation had a substantial increase in the activity of inflammatory genes, particularly in a type of nervous system cell called microglia. Consequently, they developed severe neuroinflammation by about a month of age. However, in those also missing the STING gene, this increased gene activity and neuroinflammation was substantially reduced.

The researchers saw a similar phenomenon in mouse models for two other LSDs—palmitoyl-protein thioesterase 1 deficiency and lysosomal chloride channel deficiency. These and other LSD mouse models in the study were provided by Steven Gray, Ph.D., UTSW Professor of Pediatrics, in the Eugene McDermott Center for Human Growth and Development, of Molecular Biology, and of Neurology, who is an expert on gene therapy for LSD patients.

These results suggested STING drives neuroinflammation when lysosomes become damaged, a finding the researchers corroborated when they dosed [healthy cells](#) with a chemical that damages lysosomes.

A closer look at [gene expression](#) in cells derived from the animal models showed that STING also increased the activity of genes associated with lysosome repair and new lysosome generation. Additional experiments in collaboration with Lu Sun, Ph.D., Assistant Professor of Molecular Biology and an expert on glial cells, showed this phenomenon depends on a protein called transcription factor EB (TFEB), which acts as a master controller of several lysosome-related genes.

Because the STING protein has multiple functional regions spanning the [cell membrane](#), the researchers did additional experiments to determine which region might be responsible for

lysosome generation. They found that the region located squarely within the cell membrane was key for this function.

This "transmembrane" region is known to house a channel that helps membranous vesicles, such as lysosomes, regulate pH—a measure of acidity or alkalinity—by transporting pH-lowering protons across the membrane. Opening this channel on acidic vesicles releases protons, increasing the pH inside the vesicles and activating TFEB.

Dr. Yan and his colleagues hypothesize that because lysosomes normally degrade STING continuously, since it's perpetually generated in cells, STING accumulation signals cells to kickstart the lysosome repair and generation pathway. In LSDs, this accumulation also prompts STING to generate inflammation.

Thus, finding a way to dampen STING's inflammatory role while encouraging its lysosome repair and generation role could offer a new way to treat LSDs, Dr. Yan said.

Because [lysosome](#) dysfunction is also a prominent feature of other neurodegenerative diseases, including Alzheimer's, Parkinson's, and amyotrophic lateral sclerosis (ALS), this strategy eventually could be used to treat these conditions as well.

More information: Zhen Tang et al, STING mediates lysosomal quality control and recovery through its proton channel function and TFEB activation in lysosomal storage disorders, *Molecular Cell* (2025). DOI: [10.1016/j.molcel.2025.03.008](https://doi.org/10.1016/j.molcel.2025.03.008)

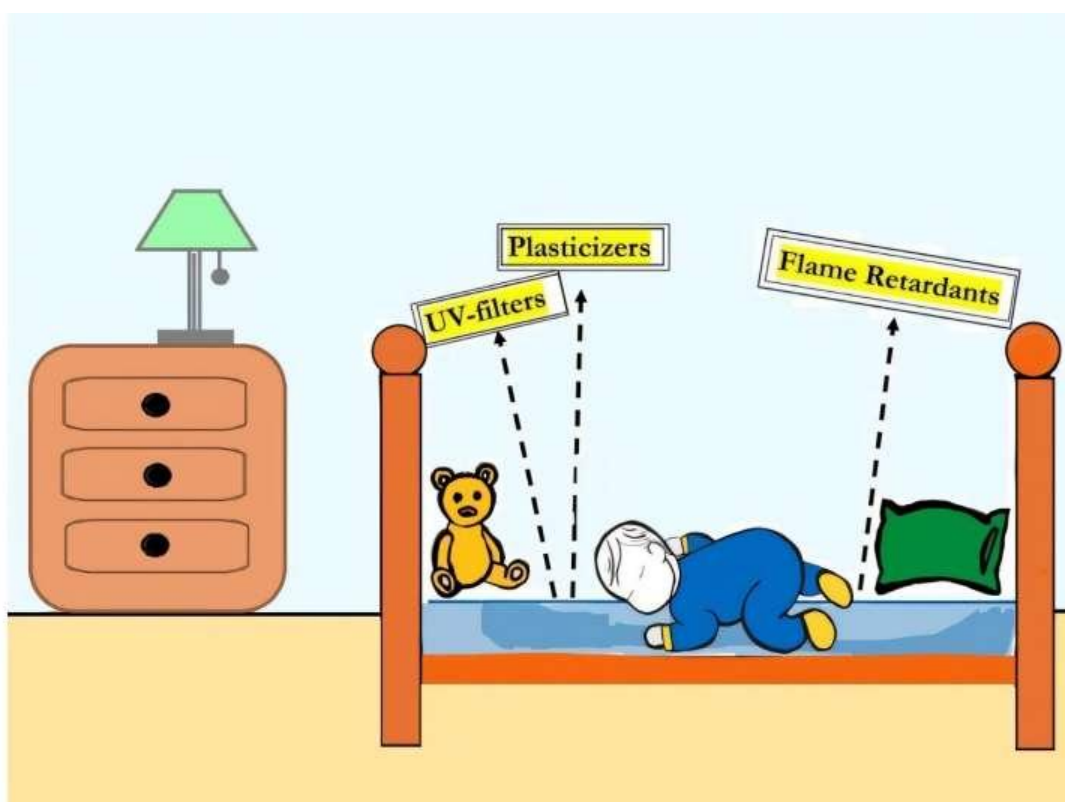
Journal information: [Molecular Cell](#)

Provided by [UT Southwestern Medical Center](#)

APRIL 15, 2025

Children exposed to brain-harming chemicals while sleeping: Scientists urge mattress manufacturers to act

by Green Science Policy Institute



Research suggests children may breathe and absorb plasticizers called phthalates, flame retardants, and other harmful chemicals from their mattresses while they sleep. Credit: University of Toronto

Babies and young children may breathe and absorb plasticizers called phthalates, flame retardants, and other harmful chemicals from their mattresses while they sleep, according to a pair of studies published by the University of Toronto in *Environmental Science & Technology* and *Environmental Science & Technology Letters*. These chemicals are linked to neurological and reproductive problems, asthma, hormone disruption, and cancer.

"Sleep is vital for brain development, particularly for infants and toddlers. However, our research suggests that many mattresses contain chemicals that can harm kids' brains," says senior author Miriam Diamond, professor at the University of Toronto.

"This is a wake-up call for manufacturers and policymakers to ensure our children's beds are safe and support healthy [brain development](#)."

In the [first study](#), researchers measured chemical concentrations in 25 bedrooms of children aged 6 months to 4 years. They detected concerning levels of more than two dozen phthalates, [flame retardants](#), and UV-filters in bedroom air, with the highest levels lurking around the beds.

In a [companion study](#), researchers tested 16 newly purchased children's mattresses and confirmed that they are likely the major source of these chemicals in children's sleeping environments. When the researchers simulated a child's body temperature and weight on the mattresses, chemical emissions increased substantially, as much as by several times.

The mattresses were purchased in Canada, but most contained materials originating from other countries, including the U.S. and Mexico. Because of integrated mattress markets and supply chains, the results are likely to apply to mattresses purchased throughout North America.

The phthalates and organophosphate ester flame retardants measured in this study are hormone disruptors and are linked to neurological harms, including learning disorders, reduced IQ scores, behavioral problems, and impaired memory. Some are also linked to childhood asthma and cancer. Several UV-filters are hormone disruptors.

Children are uniquely vulnerable to exposure, given that they are still developing, have hand-to-mouth behaviors, and have breathing rates ten times higher than adults. They also have more permeable skin and three times the skin surface area relative to their body weight than adults.

The high levels of flame retardants in most of the mattresses tested were puzzling, given these chemicals are not necessary to pass either Canadian or U.S. mattress flammability standards. Flame retardants are linked to neurological, reproductive, and hormonal harm as well as cancer, and also have no proven fire-safety benefit as used in mattresses.

"Parents should be able to lay their children down for sleep knowing they are safe and snug," said co-author Arlene Blum, Executive Director of the Green Science Policy Institute.

"Flame retardants have a long history of harming our children's cognitive function and ability to learn. It's concerning that these chemicals are still being found in children's mattresses even though we know they have no proven fire-safety benefit, and aren't needed to comply with flammability standards."

The researchers call for manufacturers to be more vigilant about the chemicals in children's mattresses through testing. Further, stronger regulations on the use of flame retardants and phthalate plasticizers in children's mattresses are needed.

In Canada, the authors recommend extending restrictions on certain plasticizers from toys and objects to be mouthed to include mattresses and other bedding items and passing long-proposed restrictions on two flame retardants.

Several mattresses had chemicals that are banned or restricted in Canada, suggesting that manufacturers need to test children's mattresses for these chemicals and other unnecessary chemicals before putting them on the market.

In the meantime, there are actions parents can take to reduce their children's exposure. The authors recommend decluttering your child's sleeping area by reducing the number of pillows, blankets, and toys.

They also recommend washing and refreshing your child's bedding and bed clothing frequently since these act as a protective barrier to reduce exposure. Finally, they say that undyed or neutral colored fabrics are likely safer since keeping strong colors requires the addition of UV-filters and other additives that may be harmful.

More information: *Environmental Science & Technology Letters* (2025). DOI: [10.1021/acs.estlett.5c00051](https://doi.org/10.1021/acs.estlett.5c00051)

Are Sleeping Children Exposed to Plasticizers, Flame Retardants, and UV-Filters from Their Mattresses?, *Environmental Science & Technology* (2025). DOI: [10.1021/acs.est.5c03560](https://doi.org/10.1021/acs.est.5c03560)

Journal information: [Environmental Science & Technology Letters](#) , [Environmental Science & Technology](#)

Provided by Green Science Policy Institute

APRIL 22, 2025

An AI tool grounded in evidence-based medicine outperforms other AI tools—and most doctors—on USMLE exams

by [University at Buffalo](#)



Credit: Pixabay/CC0 Public Domain

A powerful clinical artificial intelligence tool developed by University at Buffalo biomedical informatics researchers has demonstrated remarkable accuracy on all three parts of the United States Medical Licensing Exam (Step exams), according to a paper published in [JAMA Network Open](#).

Achieving higher scores on the USMLE than most [physicians](#) and all other AI tools so far, Semantic Clinical Artificial Intelligence ([SCAI](#), pronounced "sky") has the potential to become a critical partner for physicians, says lead author Peter L. Elkin, MD, chair of the Department of Biomedical Informatics in the Jacobs School of Medicine and Biomedical Sciences at UB and a physician with UBMD Internal Medicine.

Elkin says SCAI is the most accurate clinical AI tool available to date, with the most advanced version scoring 95.2% on Step 3 of the USMLE, while a GPT4 Omni tool scored 90.5% on the same test.

"As physicians, we are used to using computers as tools," he explains, "but SCAI is different; it can add to your decision-making and thinking based on its own reasoning."

The researchers tested the model against the USMLE, required for licensing physicians nationwide, which assesses the physician's ability to apply knowledge, concepts and principles, and to demonstrate fundamental patient-centered skills. Any questions with a visual component were eliminated.

Elkin explains that most AI tools function by using statistics to find associations in online data that allow them to answer a question. "We call these tools generative artificial intelligence," he says.

"Some have postulated that they are just plagiarizing what's on the internet because the answers they give you are what others have written." However, these AI models are now becoming partners in care rather than simple tools for clinicians to utilize in their practice, he says.

"But SCAI answers more complex questions and performs more complex semantic reasoning," he says. "We have created knowledge sources that can reason more the way people learn to reason while doing their training in [medical school](#)."

The team started with a [natural language](#) processing software they had previously developed. They added vast amounts of authoritative clinical information gleaned from widely disparate sources, ranging from recent medical literature and clinical guidelines to [genomic data](#), drug information, discharge recommendations, patient safety data and more. Any data that might be biased, such as clinical notes, were not included.

13 million medical facts

SCAI contains 13 million medical facts, as well as all the possible interactions between those facts. The team used basic clinical facts known as semantic triples (subject-relation-object, such as "Penicillin treats pneumococcal pneumonia") to create semantic networks. The tool can then represent these semantic networks so that it is possible to draw logical inferences from them.

"We have taught large language models how to use semantic reasoning," says Elkin.

Other techniques that contributed to SCAI include knowledge graphs that are designed to find new links in medical data as well as previously "hidden" patterns, as well as retrieval-augmented generation, which allows the [large language model](#) to access and incorporate information from external knowledge databases before responding to a prompt. This

reduces "confabulation," the tendency for AI tools to always respond to a prompt even when it doesn't have enough information to go on.

Elkin adds that using formal semantics to inform the large language model provides important context necessary for SCAI to understand and respond more accurately to a particular question.

'It can have a conversation with you'

"SCAI is different from other large language models because it can have a conversation with you and, as a human-computer partnership, can add to your decision-making and thinking based on its own reasoning," Elkin says.

He concludes, "By adding semantics to large language models, we are providing them with the ability to reason similarly to the way we do when practicing evidence-based medicine."

Because it can access such vast amounts of data, SCAI also has the potential to improve patient safety, improve access to care and "democratize specialty care," Elkin says, by making medical information on specialties and subspecialties accessible to primary care providers and even to patients.

While the power of SCAI is impressive, Elkin stresses its role will be to augment, not replace, physicians.

"Artificial intelligence isn't going to replace doctors," he says, "but a doctor who uses AI may replace a doctor who does not."

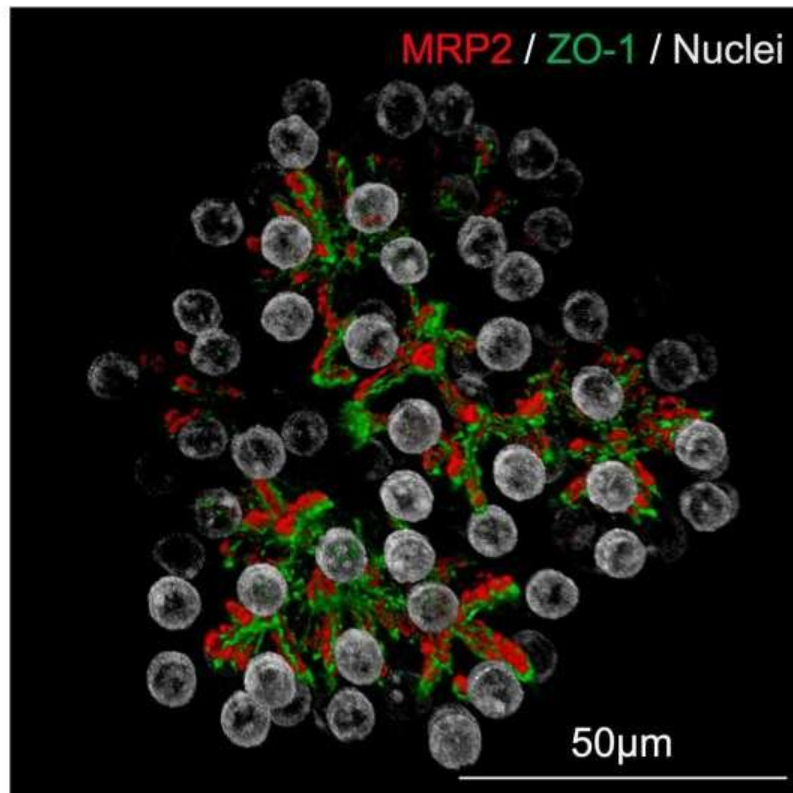
More information: Semantic Clinical Artificial Intelligence vs Native Large Language Model Performance on the USMLE, *JAMA Network Open* (2025). DOI: [10.1001/jamanetworkopen.2025.6359](https://doi.org/10.1001/jamanetworkopen.2025.6359). jamanetwork.com/journals/jamanetworkopen.2025.6359

Journal information: [JAMA Network Open](#)
Provided by [University at Buffalo](#)

APRIL 16, 2025

Scientists achieve record-breaking growth in miniature, functional liver models

by Keio University



Differentiated hepatocyte organoids formed networks of small canals that allowed bile acid to pass, in a manner similar to human tissue. Credit: Toshiro Sato from Keio University

The liver is the body's control tower for metabolism, powering vital functions like converting nutrients to glucose, storing fat and breaking down toxins. Over a third of the world, however, is thought to be affected by conditions including metabolic dysfunction-associated steatotic liver disease (MASLD), which jeopardize key liver functions as the condition progresses. Hepatocyte organoids—the miniature, 3D models of the organ—hold immense promise for accelerating drug development and advancing regenerative therapies.

In a study [published](#) in *Nature*, Keio University researchers unveiled a method to proliferate these hard-to-grow [organoids](#) by a million-fold in just 3–4 weeks while maintaining key liver functions. "These organoids are potentially the closest laboratory representations of the liver and its multifunctionality," says senior author Professor Toshiro Sato of the Keio University School of Medicine.

While organoids aim to mimic human organs, the liver's repertoire of complex functions—and thus the energy it needs to operate—have made it challenging for researchers to grow organoids that proliferate and fully function, says Sato. When prioritizing growth and survival in laboratory settings, hepatocytes, the liver's main cells, eventually transform into cells resembling cholangiocytes, which line the bile duct. Hepatocyte functions only last 1–2 weeks at most.

Now, the research team, led by Ryo Igarashi and Mayumi Oda at the Keio University School of Medicine, has generated hepatocyte organoids from cryopreserved human adult hepatocytes taken directly from patients. Treating these with oncostatin M, a [signaling protein](#) involved in inflammation, led to a million-fold proliferation of the organoids, in contrast to previous studies which saw little cell growth. The organoids continued growing for three months and survived half a year without losing the ability to differentiate.

The researchers also developed a new method for inducing differentiation using hormones that regulate hepatocyte functions. After the cells differentiated through this method, the organoids began to show all major functions of the liver, producing compounds including glucose, urea, bile acid, cholesterol and triglycerol.

The secretion of some compounds like albumin—a protein in the bloodstream for maintaining osmotic balance—exceeded previous studies, reaching levels comparable to hepatocytes in the human body. In addition, they formed networks of small canals that allowed bile acid to pass.

Discovering the role of oncostatin M in organoid development was a key breakthrough, says Sato.

"We only know a few molecules that unlock a stem cell's potential to grow into organoids and proliferate," he says. "This one is brand-new and opens up opportunities for developing new types of organoids that researchers have struggled to create."

3D reconstruction of dHHO. DHHO was stained with CDH1 (blue), ZO-1 (green) and MRP2 (red). Representative cropped images are in Fig. 3d and Extended Data Fig. 7a.

Credit: *Nature* (2025). DOI: 10.1038/s41586-025-08861-y

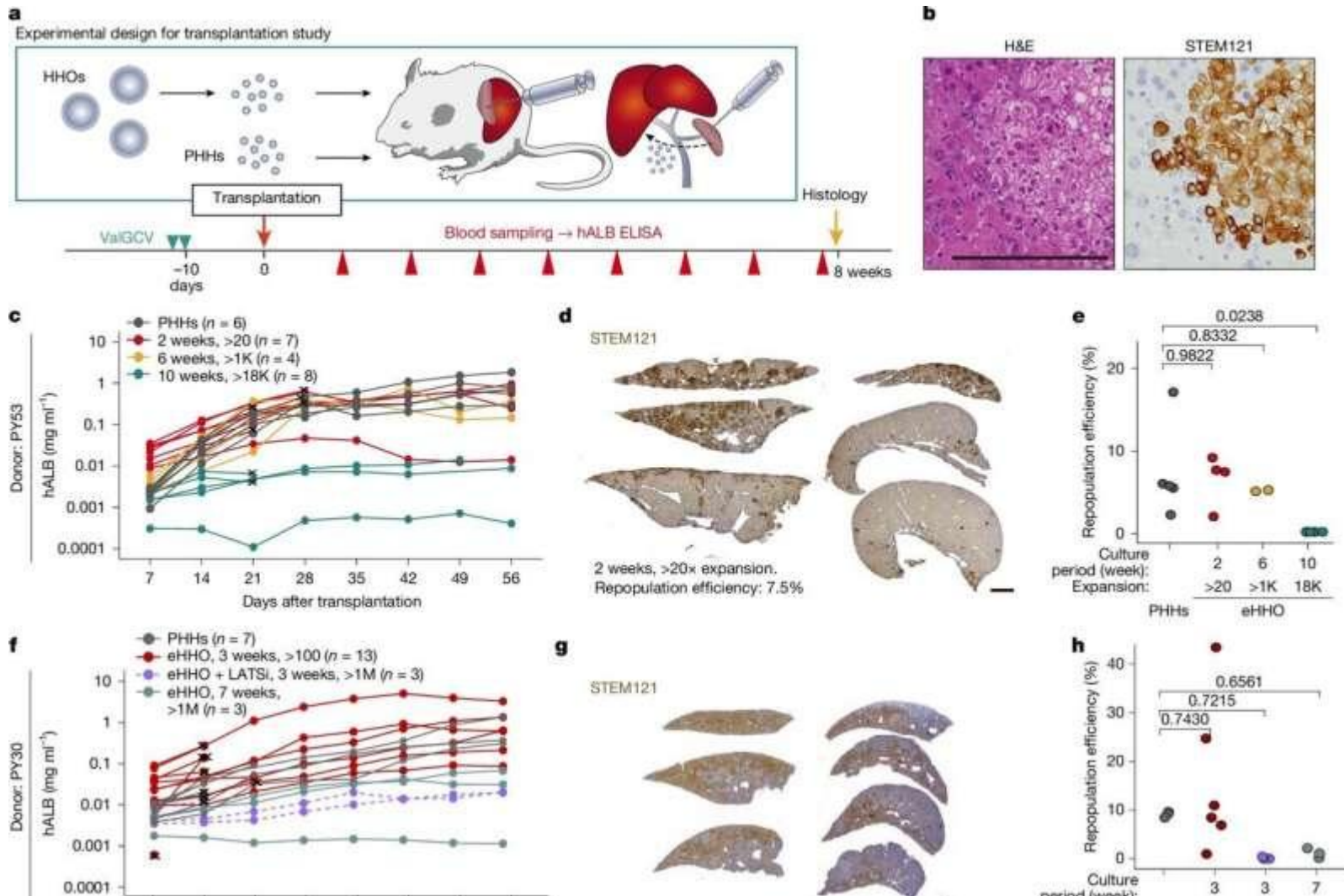
Accelerating liver research

When the team injected the human hepatocyte organoids into mice with compromised immune systems and dysfunctional livers, the cells eventually replaced the mice's own liver cells and restored liver function.

This has long-term implications on liver regeneration. Though the liver is one of the most sought-after organs for transplants, they are limited in availability as they degrade quickly after harvesting and must be transplanted within a short timeframe. While there are initiatives to extract and freeze hepatocytes for later transplantation, the method has had limited success.

Sato believes that turning frozen cells into organoids may revitalize their proliferation ability, which would make them better material for regeneration therapies.

"Our study demonstrated that liver organoid transplants can be successful in mice. But to regenerate a human liver, organoid growth has to scale up to thousands of millions, because the human body is larger," he says. "If realized, this approach could be a game-changer for patients awaiting transplantation."



Repopulation of a mouse liver with HHOs. Credit: *Nature* (2025). DOI: 10.1038/s41586-025-08861-y

In the more immediate future, the study helps streamline drug development for liver disease by cutting toxicity testing costs. Due to significant species differences in the liver, the standard pharmaceutical practice is to use human hepatocytes harvested directly from donors.

Though they lose their functions within days, these hepatocytes cost 100,000–300,000 Japanese yen (US \$670–2,000) per vial. In addition, there is high variability in a batch's

ability to survive and function. Meanwhile, [hepatocyte](#) organoids serve as more consistent research material.

The organoids also offer better models for liver disease. In the study, they generated their own lipids, which disappeared after being treated with drugs for MASLD. The approach better resembles conditions like MASLD compared to studies that artificially inject lipids.

Furthermore, the team succeeded in editing genes to replicate ornithine transcarbamylase deficiency, a genetic disorder disrupting the urea cycle. This achievement advances methods for modeling genetic liver diseases.

Sato says that work on proliferating organoids by a greater order of magnitude, as well as including other types of [liver cells](#) in the organoids, are crucial next steps for bolstering the effectiveness of organoids in medical research.

More information: Ryo Igarashi et al, Generation of human adult hepatocyte organoids with metabolic functions, *Nature* (2025). DOI: [10.1038/s41586-025-08861-y](https://doi.org/10.1038/s41586-025-08861-y). www.nature.com/articles/s41586-025-08861-y

Journal information: [Nature](#)

Provided by Keio University

UCLA discovers first stroke rehabilitation drug to repair brain damage in mice

Drug replicated recovery of the movement control produced by rehab in mice



Getty Images/iStockphoto

March 18, 2025

A NOTE TO PATIENTS

We have received inquiries about participating in clinical trials following the announcement of the research below. It is important to clarify that this research is in early stages and requires more laboratory research. Currently, no human clinical trials related to this research are being conducted and we are not aware of any planned in the immediate future. The transition from laboratory findings to human studies requires extensive additional research and regulatory approval. Should human trials become available in the future, information will be provided through clinicaltrials.gov(Link is external)

A new study by UCLA Health has discovered what researchers say is the first drug to fully reproduce the effects of physical stroke rehabilitation in model mice, following from human studies.

The [findings\(Link is external\)](#), published in Nature Communications, tested two candidate drugs derived from their studies on the mechanism of the brain effects of rehabilitation, of which one resulted in significant recovery in movement control after stroke in the mouse model.

Stroke is the leading cause of adult disability because most patients do not fully recover from the effects of stroke. There are no drugs in the field of stroke recovery, requiring stroke patients to undergo physical rehabilitation which has shown to be only modestly effective.

“The goal is to have a medicine that stroke patients can take that produces the effects of rehabilitation,” said Dr. [S. Thomas Carmichael](#), the study’s lead author and professor and chair of UCLA Neurology. “Rehabilitation after stroke is limited in its actual effects because most patients cannot sustain the rehab intensity needed for stroke recovery.

“Further, stroke recovery is not like most other fields of medicine, where drugs are available that treat the disease—such as cardiology, infectious disease or cancer,” Carmichael said.

“Rehabilitation is a physical medicine approach that has been around for decades; we need to move rehabilitation into an era of molecular medicine.”

In the study, Carmichael and his team sought to determine how physical rehabilitation improved brain function after a stroke and whether they could generate a drug that could produce these same effects.

Working in laboratory mice models of stroke and with stroke patients, the UCLA researchers identified a loss of brain connections that stroke produces that are remote from the site of the stroke damage. Brain cells that are located at a distance from the stroke site get disconnected from other neurons. As a result, brain networks do not fire together for such things like movement and gait.

The UCLA team found that some of the connections that are lost after stroke occur in a cell called a parvalbumin neuron. This type of neuron helps generate a brain rhythm, termed a gamma oscillation, which links neurons together so that they form coordinated networks to produce a behavior, such as movement. Stroke causes the brain to lose gamma oscillations. Successful rehabilitation in both laboratory mice and in humans brought gamma oscillations back into the brain, and in the mouse model, repaired the lost connections of parvalbumin neurons.

Carmichael and team then identified two candidate drugs that might produce gamma oscillations after stroke. These drugs specifically work to excite parvalbumin neurons. The researchers found one of the drugs, DDL-920, developed in the UCLA lab of Dr. Varghese John, who coauthored the study, produced significant recovery in movement control.

Further studies are needed to understand the safety and efficacy of this drug before it could be considered for human trials.

APRIL 23, 2025

Ashwagandha: This ancient herb is trending for its potential health benefits—but also comes with risks

by Dipa Kamdar, [The Conversation](#)



Credit: Unsplash/CC0 Public Domain

Depending on who you follow on social media, you may have come across talk of a little-known herb called ashwagandha over the past year. Celebrities including Meghan Markle, Gwyneth Paltrow and Jennifer Aniston are [reported](#) to use ashwagandha for its calming effects. [Ashwagandha](#) has been trending on social media with [data](#) showing #ashwagandha had more than 670 million views on TikTok in 2024.

Scientifically known as *Withania somnifera*, ashwagandha is a common herb used for thousands of years in Ayurvedic medicine—an ancient Indian system of healing based on the belief that health and wellness depend on a balance between the body, mind and spirit.

Ayurveda emphasizes a holistic approach to health using natural treatments, such as herbal remedies, dietary changes, physical therapy, meditation and yoga.

But beyond the social media buzz, what does science say about this herb's benefits?

Ashwagandha root has been used in traditional medicine as an [adaptogen](#). This means it could help people become more resilient to various types of stress, whether biological, physical, or chemical.

The strongest [evidence](#) available for ashwagandha is as a stress and anxiety reliever. A [review](#) looking at several small studies showed that ashwagandha can significantly reduce levels of perceived stress and anxiety in people. This may be partly due to its regulating effect on stress hormones such as cortisol.

Ashwagandha is also known for its ability to improve sleep quality. The "somnifera" part of its scientific name, meaning "sleep-inducing" hints at its effects. Some [trials](#) show it can help people fall asleep faster and enjoy deeper, more restful sleep, thus boosting energy levels. This may be beneficial for people suffering from insomnia. But there is no evidence showing whether it is better than taking sleeping tablets.

Possible benefits

Recently, this herb has been associated with other benefits. The Sanskrit word "ashwagandha" means "the smell of a horse," symbolizing its ability to give the strength and stamina of a horse. Athletes and fitness enthusiasts may benefit from ashwagandha's ability to enhance physical performance. Some [research](#) indicates that ashwagandha can improve strength, muscle mass and oxygen use during exercise.

For men, ashwagandha has been shown in some [small studies](#) to boost testosterone levels and improve fertility by increasing sperm count and motility. This may be linked to dehydroepiandrosterone ([DHEA](#))—a sex hormone that your body naturally produces. DHEA is used to make other hormones such as testosterone. This means men with prostate cancer sensitive to testosterone should avoid using this herb.

Ashwagandha has been linked to improved cognitive function, such as better memory and focus. [Small studies](#), involving older people who have some [cognitive impairment](#), suggest that ashwagandha may help to reduce [oxidative stress](#)—harmful molecules called free radicals that can damage cells in the body—and inflammation, which can negatively affect memory and thinking processes.

There are also ongoing [clinical trials](#) investigating whether ashwagandha may be effective in treating long COVID symptoms such as fatigue and cognitive dysfunction—having trouble with mental tasks such as thinking, remembering and making decisions—but there's no robust evidence yet.

Ashwagandha is rich in phytochemicals, including withanolides. [Withanolides are steroidal lactones](#)—they are structurally similar to steroids, with a lactone ring in their chemical structure—that are thought to help cells absorb glucose from the bloodstream. This can lower blood sugar in both healthy people and those with diabetes, although larger [studies](#) need to be done. In [animal studies](#), withanolides show anti-inflammatory activity.

Side effects

While ashwagandha may offer potential health benefits, it also has numerous [risks and side effects](#). The long-term safety of ashwagandha is not well-documented. Most studies have focused on short-term use, typically up to three months—but the benefits may take some weeks or months to appear. The most common side effects are mild stomach upsets and nausea.

Its use is not advised in people with some pre-existing health conditions such as [liver disease](#). Although rare, there have been [reports](#) of liver problems, including severe liver failure, associated with ashwagandha use. Ashwagandha may [stimulate](#) the immune system, potentially causing flare ups for people with autoimmune conditions such as multiple sclerosis and rheumatoid arthritis.

It's also possible that ashwagandha may interact with some medications, such as immunosuppressants, sedatives and thyroid hormone medications. [Research suggests](#) that ashwagandha may influence thyroid function, particularly by increasing thyroid hormone levels. It may also interact with thyroid medications, such as levothyroxine, possibly leading to overmedication.

Pregnant and breastfeeding women are advised to avoid ashwagandha, especially at higher doses. The herb may be linked to miscarriages and, although there's conflicting [evidence](#), it's best to be cautious.

Ashwagandha holds promise, then, as a stress reliever, sleep aid and even an energy booster. With growing interest and a large body of anecdotal evidence, it's no surprise that it's become a favorite among wellness enthusiasts. However, [scientific research](#) is still developing and more extensive clinical trials are needed to confirm the benefits, side effects and determine the safest, most effective doses.

If you're considering incorporating ashwagandha into your routine, especially for long-term use, do consult a health care professional first, especially if you have pre-existing health conditions or are taking other medications.

Provided by [The Conversation](#)

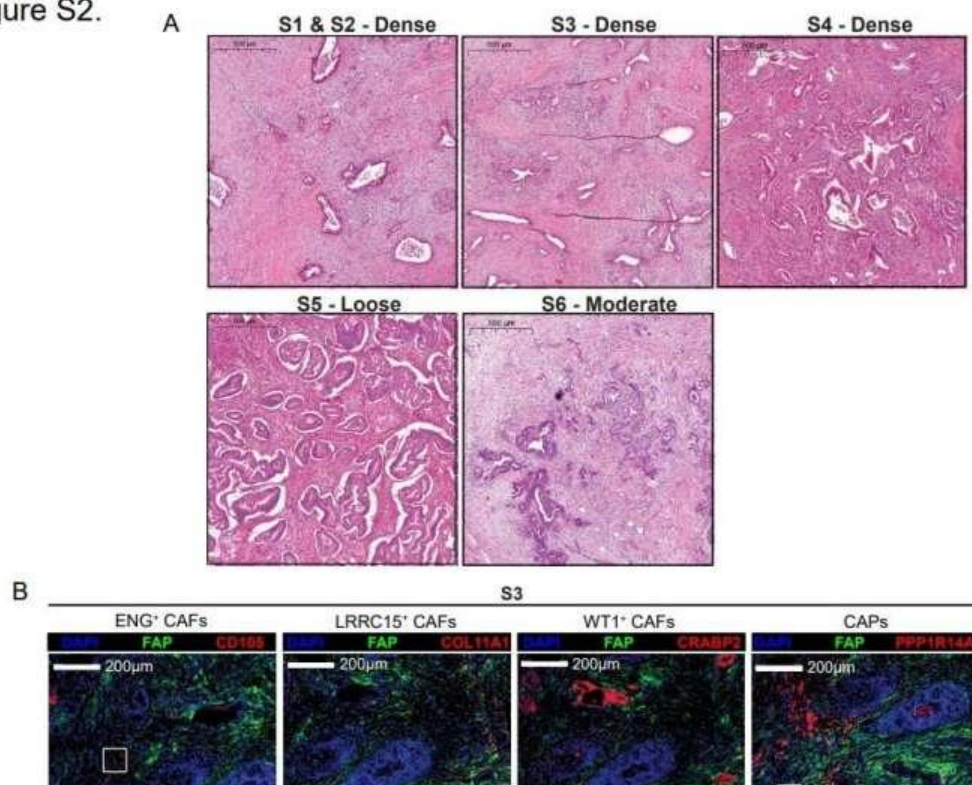
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APRIL 23, 2025

Newly discovered cells in connective tissue open up strategy against pancreatic cancer

by [Umea University](#)

Figure S2.



Validation of FAP+ CAF subpopulations in human PDAC. Credit: *Cancer Research* (2025). DOI: 10.1158/0008-5472.CAN-23-3252

Researchers at Umeå University, Sweden, have discovered a previously unknown type of connective tissue cells that surround cancer cells in pancreatic tumors. The newly discovered cells counteract tumor development and may therefore be a target for research into new treatments for pancreatic cancer, which is one of the deadliest forms of cancer. The work is [published](#) in the journal *Cancer Research*.

"We are still a long way from [treatment](#), but this may show a viable path for further research," says Daniel Öhlund, associate professor at Umeå University and leader of the current study.

Pancreatic cancer is one of the cancers with the worst prognosis for survival. This is partly because it is often detected late, and partly because it responds poorly to traditional treatments with chemotherapy.

Researchers at Umeå University have investigated the [connective tissue cells](#) that surround the cancer cells in [pancreatic cancer](#). These cells, called cancer-associated fibroblasts (CAF), play an important role in how the cancer develops. Several types of CAF stimulate the growth of cancer cells and contribute to the cancer cells being resistant to current chemotherapy treatments, making the cancer more aggressive and difficult to treat.

What the Umeå researchers have found is a previously unknown subgroup of CAF that has opposite properties. This group can instead hold back the cancer and open the way for the body's own immune cells to attack the cancer cells. However, this newly discovered group, called interferon response cancer-associated fibroblasts (ifCAF), is in the minority among different CAF around the cancer cells.

"The opening we see is whether it would be possible to stimulate the formation of this newly discovered and kinder cell type around the [cancer cells](#) at the expense of the connective tissue cells that make the cancer aggressive," says Öhlund.

The researchers at Umeå University are now proceeding to study potential drug substances that can promote the formation of ifCAF cells so that they increase in number and thus slow down the development of the disease. If successful, it could become a target for the development of future drugs.

About half a million people worldwide are diagnosed with pancreatic cancer every year.

More information: Joshua Cumming et al, Dissecting FAP+ Cell Diversity in Pancreatic Cancer Uncovers an Interferon-Response Subtype of Cancer-Associated Fibroblasts with Tumor-Restraining Properties, *Cancer Research* (2025). DOI: [10.1158/0008-5472.CAN-23-3252](https://doi.org/10.1158/0008-5472.CAN-23-3252)

Journal information: [Cancer Research](#)
Provided by [Umea University](#)

APRIL 24, 2025

Rethinking stroke risk in patients with atherosclerotic carotid stenosis

by University of Toyama



Clinical Features, Radiological Findings, and Outcome in Patients with Symptomatic Mild Carotid Stenosis: A MUSIC Study
Kashiwazaki et al. (2025) | Journal of Neurosurgery | DOI: 10.3171/2024.10.JNS241185

Researchers examined the clinical features, radiological findings, and treatment outcomes of patients with symptomatic mild carotid stenosis, seeking to improve current medical guidelines for the management of this patient population. Credit: Lecturer Daina Kashiwazaki from Toyama University, Japan

Ischemic stroke remains one of the leading causes of death and long-term disability worldwide, with narrowing of the carotid artery due to atherosclerosis contributing to up to 30% of all cases. For decades, medical practitioners have primarily relied on the degree of carotid narrowing (stenosis) to assess the risk of stroke and determine the best treatment options. However, mounting evidence suggests that this approach may be insufficient for patients with mild but symptomatic carotid stenosis.

Despite being classified as "low-risk" due to having less than 50% carotid artery narrowing, a significant number of [patients](#) with mild carotid stenosis continue to experience recurrent ischemic events, even when receiving appropriate medical therapy. This implies that factors beyond the degree of stenosis may play a crucial role in determining stroke risk for this patient population.

To address this gap, a team including Lecturer Daina Kashiwazaki and Dr. Satoshi Kuroda from Toyama University, Japan, aimed to tackle this knowledge gap via their "Mild but

Unstable Stenosis of Internal Carotid Artery (MUSIC)" study. This multicenter prospective cohort study, which was [published](#) online on February 21, 2025, in the *Journal of Neurosurgery*, investigated the clinical features, radiological findings, and treatment outcomes of patients with symptomatic mild carotid stenosis.

The researchers enrolled 124 patients who had experienced cerebrovascular or retinal ischemic events ipsilateral (same side) to mild carotid stenosis. While all participants received the best medical therapy (BMT) for their condition, carotid endarterectomy (CEA)—the surgical removal of plaque—or carotid artery stenting (CAS) was performed in 63 patients. Patients were followed up for two years, with the primary endpoint being the occurrence of ipsilateral ischemic stroke.

The findings were quite striking: approximately 81% of patients had radiologically unstable plaque, with 59.5% exhibiting intraplaque hemorrhage (IPH). This type of plaque composition was associated with a significantly higher risk of both primary and secondary endpoints, the latter of which included ocular symptoms, any type of stroke, and plaque progression requiring CEA. Additionally, the incidence of ipsilateral [ischemic stroke](#) was markedly higher in the group receiving only BMT compared to those who also underwent CEA (15.1% vs. 1.7%).

"The distinctive clinical and radiological features in high-risk patients strongly indicate that plaque composition, namely IPH, but not degree of stenosis, plays a key role in subsequent ischemic events in patients with symptomatic mild carotid stenosis," explains Kashiwazaki.

This study challenges current medical guidelines, which typically do not recommend CEA for patients with symptomatic mild carotid stenosis. The results demonstrated that CEA significantly reduced the incidence of both primary and secondary endpoints during the two-year follow-up period, with CEA emerging as a protective factor and IPH as a risk factor for recurrent events.

Moreover, it is also particularly noteworthy that approximately half of the study participants had been receiving antithrombotic therapy prior to enrollment but still experienced cerebrovascular or ocular events. This suggests that certain patients with mild carotid stenosis may be resistant to conservative medical therapy alone, calling for more proactive interventions.

Overall, the implications of this study could fundamentally alter how physicians approach stroke prevention. "In the very near future, the evaluation of plaque composition will be an essential examination to predict the risk of further events and to determine treatment strategies in each patient with symptomatic mild [carotid](#) stenosis," notes Kashiwazaki.

In this way, by shifting focus from stenosis degree to [plaque](#) composition, clinicians may be able to better identify high-risk patients who would benefit from surgical intervention. With any luck, this revised approach could potentially reduce stroke incidence and improve outcomes for a previously underrecognized at-risk population.

This research underscores the need for personalized stroke prevention strategies that go beyond measuring stenosis severity alone.

More information: Daina Kashiwazaki et al, Clinical features, radiological findings, and outcome in patients with symptomatic mild carotid stenosis: a MUSIC study, *Journal of Neurosurgery* (2025). DOI: [10.3171/2024.10.JNS241185](https://doi.org/10.3171/2024.10.JNS241185)

Journal information: [Journal of Neurosurgery](#)

Provided by University of Toyama

APRIL 24, 2025

Build muscle strength if you want to live longer and healthier, experts say

by Alena Kuzub , [Northeastern University](#)



Credit: CC0 Public Domain

Building and maintaining muscle strength is one of the most important factors to living a long and healthy life, according to Northeastern University experts.

"Muscle mass really is the key to longevity. It really is our [insurance policy](#) for how long we want to stay functional," says Elaina Manolis, assistant clinical professor of physical therapy, [human movement](#) and rehabilitation sciences at Northeastern University.

"To live a long and healthy life, you want to be as functional and independent as possible, and that's going to depend on how well you move."

Muscles not only move our bodies. They store glucose that can be used for energy and movement, Manolis says, rather than being stored as fat. Exercising our muscles helps regulate [blood sugar](#), which is especially important for those with insulin sensitivity.

However, our [muscle mass](#) typically peaks around age 30 and [decreases by 3% to 8%](#) per decade afterward.

A [sedentary lifestyle](#) especially depletes our muscle mass, Manolis says. Rebuilding lost muscle takes significantly more time than losing it.

Never too late to start

The good news is that it's never too late to start building your muscle mass, according to Carmen Castañeda Sceppa, professor of health sciences and dean of Bouvé College of Health Sciences at Northeastern.

"Muscle is very plastic and responsive to exercise," she says. "It's a very forgiving tissue. It will be lost if there is no activity or bed rest, or any condition like that, but it can very quickly get back on track with exercise."

While [strength training](#) is crucial, optimal health requires a well-rounded routine, Manolis says, including resistance training, cardio exercise and flexibility.

Bone and joint health

Resistance training strengthens bones, Manolis says, as they respond to stress and the pull of the muscles by putting down more tissue layers and reinforcing bone structure.

Weight-bearing exercises like walking, running, jumping and push-ups contribute to bone density.

Strong muscles also protect joints from excessive load. When muscles fail to properly support the body, Manolis says, joints degenerate faster, leading to knee or hip replacements.

Regular cyclical or elliptical movements help keep joints lubricated and healthy.

How to build muscle mass

How to start depends on your general health and your age.

Building muscles requires safely and progressively overloading your body, Manolis says, with external resistance. Body weight exercises or yoga alone won't be enough because our bodies are already accustomed to carrying that weight.

"Doing yoga is great, but you're not going to necessarily build strength and power because you don't have that external resistance," Manolis says.

That is why the terms "strength training" and "resistance training" are used interchangeably.

Any tools that add resistance can be used for strength training: dumbbells, kettlebells or resistance bands. Beginners should start with two days of training per week, which is enough to maintain muscle mass.

Building additional muscle requires four to five days of strength training, which could be split between upper and lower body days.

A good tool for beginners to establish a routine is resistance bands.

"You can adjust the tension by simply changing your position or how much you're pulling on them," Manolis says. "They're also easy to travel with."

Eventually, you'll need to progress to heavier weights—around 30 to 50 pounds for deadlifts.

"You have to lift heavy for your body to respond," Manolis says. "We totally underestimate our ability. Our legs are so strong and so powerful."

In general, resistance exercises should prepare muscles for functional movements such as push, pull, lift and squat.

For specific exercises, consider consulting a physical therapist who can recommend strength training programs tailored to everyday life.

Those interested in competitive powerlifting should work with a specialized personal trainer.

Benefits for chronic conditions

Age-related changes in muscle mass, Sceppa says, can get exacerbated by chronic conditions such as arthritis, autoimmune conditions, heart disease and diabetes.

Sceppa studied resistance exercise in people with diabetes who had significant muscle mass loss. She found that strength exercise not only reversed muscle loss but also improved blood sugar levels better than [diabetes medications](#) alone.

"In my research, I wanted to show that with [resistance training](#) I could not only revert the age-related change in muscle mass but also improve their [glucose levels](#) and, as a result, improve the outcomes of diabetes," she says.

Many participants of her research needed much less medication as a result, and some were able to stop taking medication entirely.

However, with the rise of weight-loss drugs like Ozempic, a popular diabetes medication, Sceppa says, she is concerned that many people will experience muscle mass loss as a side effect. These medications cause non-selective weight loss—reducing both fat and muscle.

To selectively target fat, she says, strength training must be added to the regimen.

Strength training in older adults

With age, people move less and are afraid of injuries, so they avoid strength training, Manolis says, which results in muscle wasting.

"It really just takes education to teach them actually how to move, and then they feel totally empowered," she says.

For seniors, even light resistance can be effective.

Sceppa's research on physical activity interventions for [older adults](#) demonstrates numerous benefits.

One successful approach involved training peer leaders to lead group exercises at community health centers and other community-based organizations.

"This can be done on a low budget, and it's an opportunity to bring people together to address loneliness, particularly in older adults who are living alone and not socializing as much," she says.

Being physically active makes older adults feel better, which motivates them to continue exercising. As people get stronger, they start taking walks or steps instead of an elevator. They cook healthier food and perform their daily activities better.

"Their outlook on life improves," Sceppa says. "So there is this psychosocial component of exercise that reduces loneliness, depression and anxiety."

Exercising with a family member or a friend creates a buddy system and puts some good "peer pressure," Sceppa says, encouraging regular workouts.

"My research over the years has been about lifestyle as preventative medicine, which can only happen at scale," she says. "If you have individuals, families and communities coming

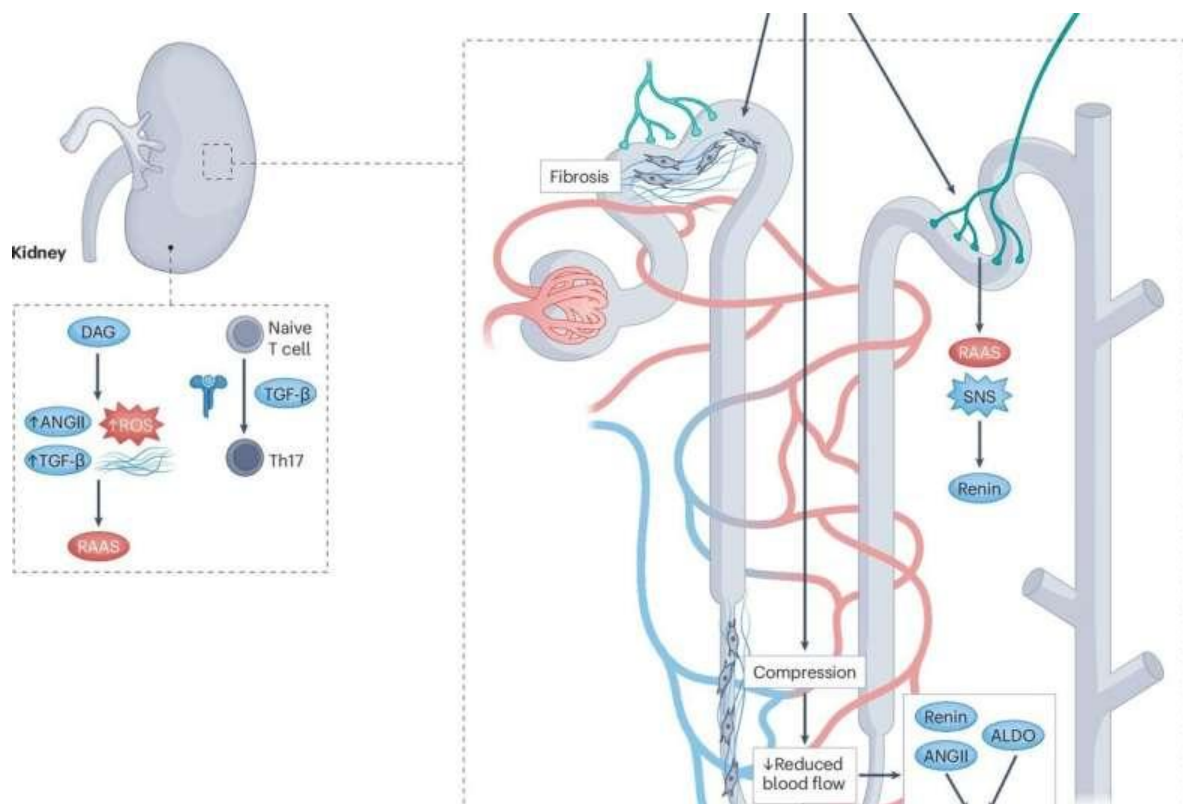
together to lead their own healthy lives, they can be an example for their children and grandchildren, role-modeling healthy behaviors."

Provided by [Northeastern University](#)

APRIL 24, 2025

Obesity triggers harmful signals that may worsen kidney disease, research finds

by [University of Kentucky](#)



Adipose tissue-derived factors that compromise the health and function of the kidney.

Credit: *Nature Reviews Nephrology* (2025). DOI: 10.1038/s41581-025-00950-5

A comprehensive new review by researchers at the University of Kentucky sheds light on the intricate and damaging relationship between obesity and kidney disease.

The review, recently [published](#) in *Nature Reviews Nephrology*, details the multiple ways in which excess [fat tissue](#) disrupts kidney function and even triggers changes in the brain that further exacerbate renal damage. Conversely, when the kidneys aren't working properly, that can also affect [adipose tissue](#) (or body fat).

"Our review shows that the link between obesity and [kidney disease](#) is far more complex than previously understood. This work underscores the urgent need for [new therapeutic strategies](#) to combat the growing epidemic of obesity-related kidney problems," said Analia S. Loria, Ph.D., an associate professor in the Department of Pharmacology and Nutritional Sciences in the UK College of Medicine and principal investigator of the grant from the National Heart, Lung, and Blood Institute.

The review emphasized that obesity doesn't just add extra weight—it triggers a cascade of harmful effects on the kidneys. These include direct physical compression, disruption of blood flow and increased pressure within the kidney, and the release of inflammatory signals and hormones from expanded fat tissue.

"Expanded fat tissue acts like an active neuroendocrine organ, sending signals that directly harm the delicate structures of the kidney. Our lab showed that adipose tissue also sends signals to the brain, increasing renal sympathetic outflow, and contributing to the development of chronic kidney disease," said Carolina Dalmasso, Ph.D., a research scientist in the Department of Pharmacology and Nutritional Sciences and one of the lead authors of the review.

"Investigating therapeutic targets to reduce these signals from adipose tissue may offer a promising strategy for mitigating obesity-related hypertension and [chronic kidney disease](#)."

The study also highlights the role of the communication network connecting adipose tissue, the brain and the kidneys via nerves, hormones and inflammatory factors.

"Notably, pre-existing kidney disease can contribute to the dysfunction of fat tissue through the accumulation of waste products in the body," said Nermin Ahmed, Ph.D., a [registered nurse](#), recent graduate of the Loria Lab and one of the lead authors. "These toxins can lead to fat tissue loss and the abnormal storage of fats, potentially worsening [kidney function](#)."

The researchers emphasize that a deeper understanding of all these interactions is crucial for developing effective treatments.

"Understanding the impact of fat-derived signals will pave the way for innovative therapies that can prevent or slow the progression of kidney disease in individuals with obesity," said Loria.

The review explored current and emerging therapeutic strategies, including drugs targeting a crucial hormonal pathway that regulates [blood pressure](#), fluid and electrolyte balance in the body—the renin-angiotensin-aldosterone system—along with inflammation, lipid metabolism, lifestyle interventions and bariatric surgery.

"It was a great experience to develop figures summarizing the most up-to-date information in the field and see them highlighted in such a prestigious journal," said Meghan Turner, a Ph.D. candidate in the Loria Lab.

This research provides critical insights into the dangerous connection between obesity and kidney disease, highlighting the need for continued investigation and the development of novel therapeutic interventions.

More information: Nermin Ahmed et al, From fat to filter: the effect of adipose tissue-derived signals on kidney function, *Nature Reviews Nephrology* (2025). DOI: [10.1038/s41581-025-00950-5](https://doi.org/10.1038/s41581-025-00950-5)
Provided by [University of Kentucky](#)

APRIL 24, 2025

Home care workers unaware of AI's role and potential benefits

by Grace Stanley , [Cornell University](#)



Credit: Kampus Production from Pexels

Artificial intelligence is increasingly being used in home health care—but home health care workers are generally unaware of that. Nor do they understand how AI works, why it may retain their information and that it could replicate bias and discrimination in their workplace.

A team of Cornell researchers investigated the implications of AI tools on the work of front-line home health care workers, such as personal care aides, home health aides and certified nursing assistants, in a [qualitative study](#). They'll present the work at the Association for Computing Machinery's Conference on Human Factors in Computing Systems ([CHI '25](#)), April 26–May 1 in Yokohama, Japan.

"Our study takes the first steps in a broader agenda that seeks to elevate the voices of frontline stakeholders in the design and adoption of safe and ethical AI systems in [home health care](#)," said Nicola Dell, co-author of the paper and associate professor of information science at Cornell Tech. She is also associate professor at the Jacobs Technion-Cornell Institute and at the Cornell Ann S. Bowers College of Computing and Information Science.

The researchers' interviews with 22 [home care workers](#), care agency staff and worker advocates revealed that home care workers lack an understanding of AI technology, its data usage and the reasons AI systems retain their information.

"Participants in the study recognized the significant efficiency gains AI tools can provide, especially in an industry facing labor shortages and increasing demand," said co-author Ian René Solano-Kamaiko, a doctoral student in computing and information science at Cornell Tech.

"However, we saw that agency participants often assumed these systems were trustworthy simply because they improved operational outcomes, despite acknowledging they have no idea if these tools are operating fairly."

The home care workers in the study generally did not realize that AI is already being implemented in their work, particularly through algorithmic shift-matching systems used by agencies that employ them. Home care workers receive shift assignments from agencies through a matching process designed to balance their availability, qualifications and geographic location with the needs and location of patients.

"We found a significant knowledge gap: Agency staff were generally more aware of AI's use in home care, while most home care workers—those directly affected by these systems—had little knowledge of AI and were often unaware it was already being used in their work," Solano-Kamaiko said.

This knowledge gap is troubling given that algorithmic rankers, which are similar to the shift-matching systems used in home care, have been shown to discriminate against groups who share the same demographic characteristics as home care workers: women, people of color, immigrants and individuals with other marginalized identities.

"While some participants acknowledged the risk of AI reinforcing existing inequalities, most were largely unaware of the potential for these technologies to reproduce racism, sexism and other forms of discrimination," Solano-Kamaiko said. "These findings underscore the urgent need for [greater transparency](#), critical oversight and awareness around the use of AI in home care settings."

To better support home care workers in the future, the researchers emphasize the need for equitable, participatory governance structures to regulate AI. They argue these structures should include important stakeholders at all levels, including patients and home care workers.

"Participatory approaches to developing AI governance will need to be constructed with care to ensure they center problems and potential solutions from the perspectives of stakeholders who are not only on the margins, but whose voices are critically excluded in current discourse on AI governance," Solano-Kamaiko said.

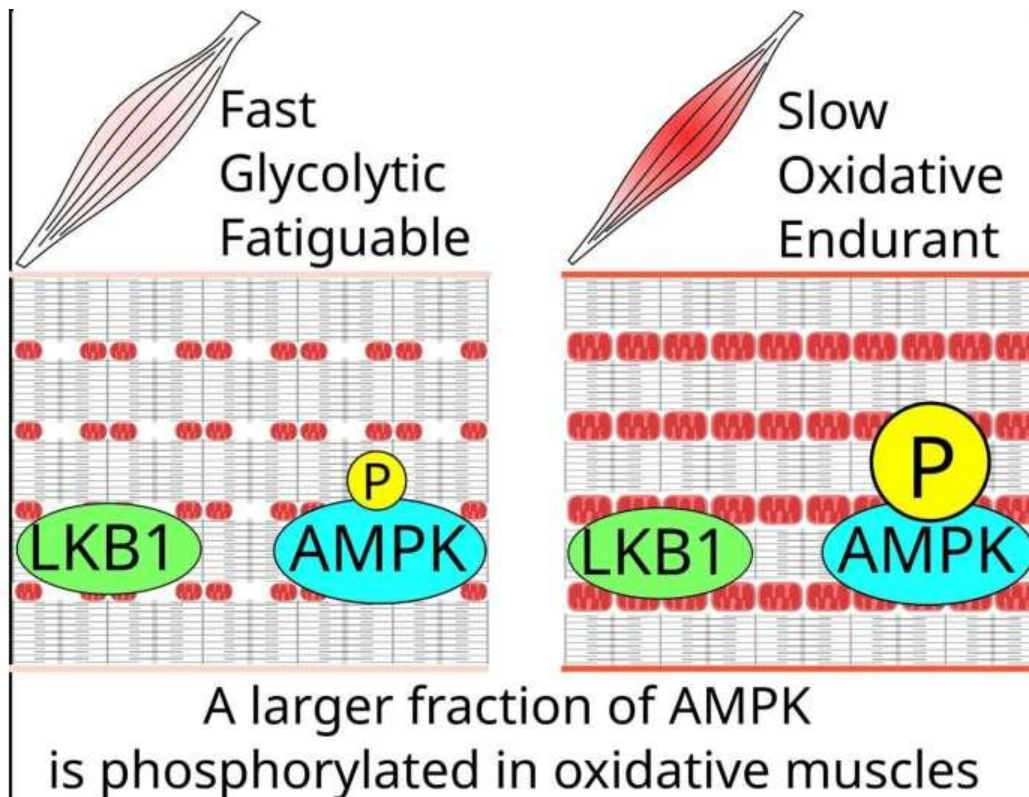
To ensure these stakeholders have the necessary AI knowledge to help govern AI structures, the researchers also advocate for "stakeholder-first" approaches to AI education.

"Instead of focusing AI literacy on the technology itself, the stakeholder-first approach shifts the emphasis from the content to be learned to the contexts in which AI systems are applied," Solano-Kamaiko said. "This approach helps workers better understand and reason about the implications of AI in their specific contexts without requiring technical skills like programming."

More information: Ian René Solano-Kamaiko et al, "Who is running it?" Towards Equitable AI Deployment in Home Care Work, *Proceedings of the 2025 CHI Conference on Human Factors in Computing Systems* (2025). DOI: [10.1145/3706598.3713850](https://doi.org/10.1145/3706598.3713850)
Provided by [Cornell University](#)

Activation of AMP-activated kinase found dependent on muscle type

by Estonian Research Council



A

larger fraction of AMPK is phosphorylated in oxidative muscles. Credit: TalTech
Nowadays, the prevalence of sedentary lifestyles and high-caloric diets leads to an imbalance between energy intake and spending. Hence, in the last decades, researchers have observed a significant rise in metabolic diseases such as obesity or diabetes.

Activation of AMP-activated kinase (AMPK) is a potential therapeutic target to treat metabolic diseases. Its activation shifts metabolism so that glucose and [fatty acids](#) from the diet are used to generate ATP rather than being stored as glycogen and fat.

The heart muscle burns more fat than the calf muscle

The heart and skeletal muscles represent 40–50% of the human body weight and are one of the main energy spending tissues in human bodies. When AMPK in the muscles is activated, it makes the cells take up glucose and fatty acids that are used to generate ATP.

In the long-term, AMPK changes [gene expression](#) to increase the number of mitochondria that can generate ATP.

AMPK-activating drugs are able to prevent obesity in mice that have diabetes or eat a [high-fat diet](#). However, researchers do not know all the details regarding the regulation of AMPK activation at the [cellular level](#), and this is still being intensely studied.

Researchers from the Laboratory of Systems Biology, Department of Cybernetics at Tallinn University of Technology (TalTech), Estonia, recently found that the extent of AMPK activation is not the same in all muscles. The muscles in the body are different, as they are adapted to the work they perform. Muscles performing continuous work, such as the heart and postural muscles that keep us upright all day, have more mitochondria that make ATP to fuel the work.

In comparison, muscles that perform intense work but tire out quickly, such as the leg muscles used for jumping, have fewer mitochondria but more myofibrils to generate force. In humans, the muscles are to some extent affected by training.

The work is [published](#) in the *American Journal of Physiology-Endocrinology and Metabolism*.



Main author of the science article, Romain Bernasconi Ph.D. student at TalTech, and K  rol Soodla, MA student at TalTech (left in the photo). Credit: TalTech

The more enduring the muscle, the greater the AMPK activation

The new study showed that whereas all muscles express almost the same amount of AMPK, a larger fraction of AMPK is activated in the heart and postural muscles. Thus, the AMPK activation in a muscle depends on what the muscle is used for. Muscles that work for longer periods of time and are more endurant have higher AMPK activation to ensure their uptake of glucose and fatty acids and to maintain their large number of mitochondria and ability to generate ATP.

The mechanism behind this is still under investigation. The study showed that different AMPK-activation did not correlate with the expression of the main activator of AMPK, liver kinase B1 (LKB1). The literature suggests that it does not correlate with the overall levels of AMP.

One way to explain the higher AMPK activation in heart and postural muscles is that the environment inside the cells is not uniform, i.e. muscle cells are not like bags with evenly mixed content. Instead, there are many structures that can form pockets in which the AMP concentration is different from the overall average concentration.

It is possible that more AMPK is activated in heart and postural muscles, because it sits in such pockets and senses AMP concentrations that are higher than average. However, further studies are needed to determine this.

These findings reveal a previously unappreciated complexity in cellular metabolic regulation. By building a more complete picture of basic AMPK regulation mechanisms, this research contributes to the foundation of knowledge upon which more applied studies can later build.

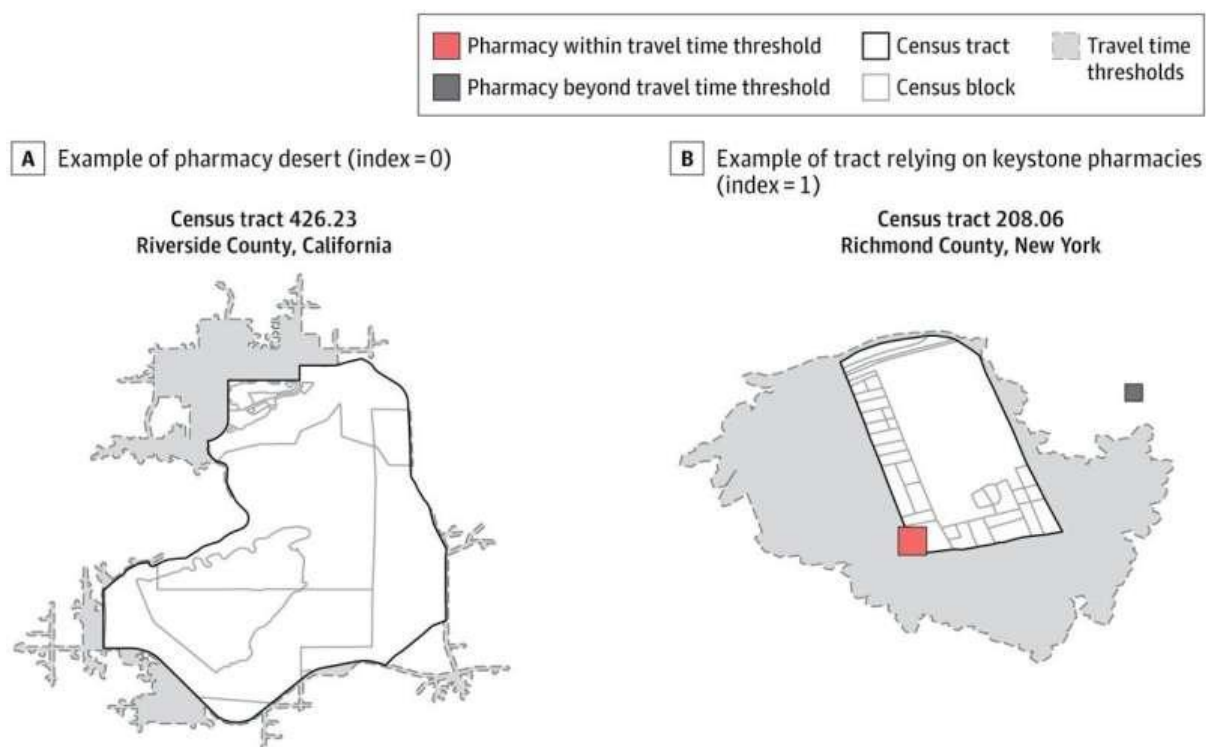
More information: Romain Bernasconi et al, Higher AMPK activation in mouse oxidative compared with glycolytic muscle does not correlate with LKB1 or CaMKK β expression, *American Journal of Physiology-Endocrinology and Metabolism* (2024). DOI: [10.1152/ajpendo.00261.2024](https://doi.org/10.1152/ajpendo.00261.2024)

Provided by Estonian Research Council

APRIL 24, 2025

New metric maps pharmacy deserts based on travel time, identifying at-risk communities

by Mallory Locklear , [Yale University](#)



Examples of Pharmacy Vulnerability Indices. Credit: *JAMA Network Open* (2025). DOI: 10.1001/jamanetworkopen.2025.0715

Local pharmacies fill more than 90% of prescriptions in the United States, making them essential for much of the population. They're also key providers of vaccines and services such as medication management.

Much like [food deserts](#)—areas where people lack adequate access to affordable, [healthy food](#)—pharmacy deserts have too few pharmacies to support the population, and they pose a threat to medication access and public health.

In a study [published](#) in *JAMA Network Open*, Yale researchers developed a way to identify pharmacy deserts—and communities at risk of becoming pharmacy deserts—based on travel time. Their approach accommodates [regional differences](#) in what's considered a reasonable amount of travel—[rural residents](#) may be used to longer travel times than city-dwellers, for instance—and by distinguishing areas at risk, it points to communities where additional resources could prevent a pharmacy desert from forming.

"Pharmacies are so much more than places to get prescription medications. They're frontline community health resources," says Walter Mathis, MD, assistant professor of psychiatry at Yale School of Medicine and senior author of the study. "And while there are some existing definitions of pharmacy deserts, we thought there was room for improvement. We wanted to develop a robust definition that was relevant on a national scale."

Calculating travel time

The researchers first collected location information for all of the pharmacies in the United States. They then developed travel time thresholds for each U.S. census tract. "We really felt like travel time, as opposed to distance, which previous definitions of pharmacy deserts have relied on, was a more important metric, as that's how most of us make decisions about travel day-to-day," says Mathis.

Supermarkets are among the most common resources for a given area. So travel time thresholds were determined by the minimum amount of time it would take for 80% of a community's population to get to a supermarket. If there were no pharmacies within that travel time, then a census tract was considered a pharmacy desert.

Among the 323.5 million people living in the United States at the time of the study, 17.7% lived in a pharmacy desert, the researchers found. And nearly 9% of people lived in an area served by a single pharmacy, which the researchers have dubbed "keystone pharmacies."

Within 18 states, more than 20% of the population lived in pharmacy deserts, and in five—New Hampshire, South Dakota, West Virginia, Maine, and Vermont—that number rose to more than 25%.

Further, the most rural settings were disproportionately reliant on keystone pharmacies, and independently owned pharmacies were more likely than chains to serve as keystones.

Supporting keystone pharmacies

Opening a new pharmacy requires substantial investment, but supporting a keystone pharmacy could be a more affordable, less resource-intensive option for communities. Therefore, directing [financial support](#) toward keystone pharmacies is one way that city, state, and federal government programs could prevent communities from becoming pharmacy deserts, say the researchers.

This could be particularly important for some regions where a single pharmacy serves as a keystone for multiple census tracts. For instance, a pharmacy in Oxnard, California, sits in the middle of several census tracts and serves as the keystone pharmacy for seven of them, possibly serving more than 34,000 people. And the researchers identified several pharmacies that were serving as the only one available to multiple neighborhoods.

"These keystone pharmacies—almost heroic in that if they were to go away, a neighborhood or neighborhoods would collapse into a dramatically underserved area—are really special cases," says Mathis. "Subsidizing them through grants or changing how they're reimbursed by insurance are a few options that could keep these essential pharmacies running. It comes down to helping them remain economically viable."

Because food deserts also exist in the United States, calculating reasonable travel thresholds through supermarket access could misrepresent [pharmacy](#) access in areas where food resources are inadequate, say the researchers. So context will be important when considering how to best target at-risk populations.

In that regard, the researchers have begun sharing their data with organizations in areas relying on keystone pharmacies.

"Hopefully, this will lead to locally generated ideas about how to support keystone pharmacies, augmenting smart new policies at the state and federal level," says Mathis.

More information: Walter S. Mathis et al, Vulnerability Index Approach to Identify Pharmacy Deserts and Keystone Pharmacies, *JAMA Network Open* (2025). DOI:

[10.1001/jamanetworkopen.2025.0715](https://doi.org/10.1001/jamanetworkopen.2025.0715)

Journal information: [JAMA Network Open](#)

Provided by [Yale University](#)

APRIL 24, 2025

Medically tailored meals improve nutrition, reduce readmissions for heart failure patients

by [University of Pennsylvania School of Nursing](#)



Credit: CC0 Public Domain

A study [published](#) in *BMC Nutrition* demonstrates that providing medically tailored meals (MTM) to patients with heart failure (HF) and malnutrition risk following hospital discharge significantly improves their nutritional status and keeps hospital readmission rates well below local and national averages.

The study compared the impact of delivering seven versus 21 MTM per week over a four-week period.

A research team, led by Penn Nursing's Charlene Compher, Ph.D., RD, LDN, FASPEN, the Shearer Chair of Healthy Community Practices, Professor of Nutrition Science, and Director of Nutrition programs in the Department of Biobehavioral Health Sciences, enrolled 46

patients discharged from the hospital with HF and [malnutrition](#) risk. The research team conducted telephone surveys at baseline, 30-, and 60-days post-discharge to assess changes in malnutrition risk, adherence to American Heart Association (AHA) [dietary guidelines](#), sarcopenia risk, and 30-day readmissions.

In collaboration with the Metropolitan Area Neighborhood Nutrition Alliance (MANNA) and the Clinical Nutrition Support Service at the Hospital of the University of Pennsylvania, the study revealed that patients receiving at least seven MTM per week experienced a significant reduction in malnutrition and sarcopenia risk.

Notably, adherence to AHA diet goals improved over time, regardless of whether patients received seven or 21 meals. The 30-day readmission rates were significantly lower than the national average, at 9% for those receiving 21 MTM and 12.5% for those receiving seven MTM.

"Our findings suggest that even a modest intervention of seven medically tailored meals per week can have a profound positive impact on the health and recovery of [heart failure](#) patients," said Compher. "This intervention not only improves nutritional outcomes and diet adherence but also keeps hospital readmissions low, which translates to better patient care and lower health care costs."

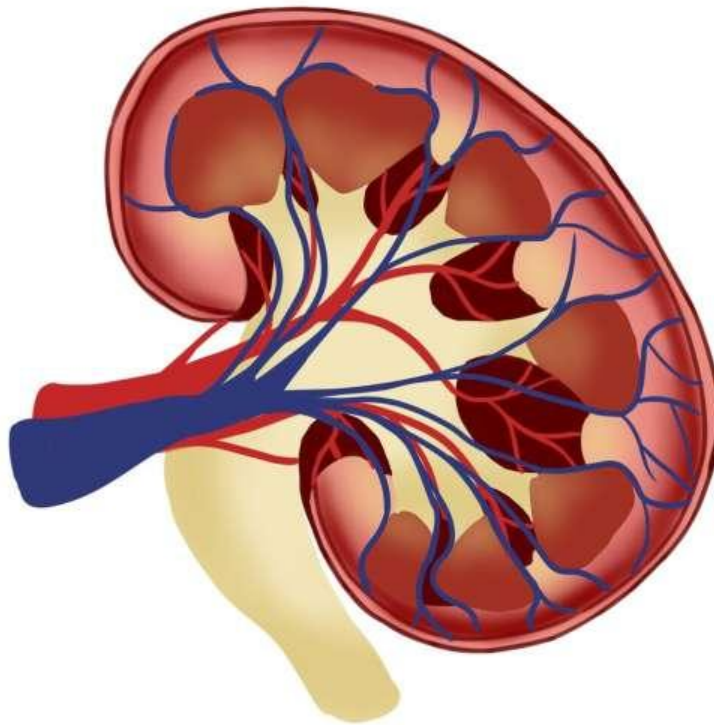
The researchers concluded that the provision of at least seven MTM per week in the immediate post-discharge period is a promising strategy to improve malnutrition and sarcopenia risk, enhance diet [adherence](#), and maintain readmission rates below national averages for patients with HF and malnutrition risk.

More information: Charlene Compher et al, The nutritional impact of 7 versus 21 home-delivered medically tailored meals in patients with heart failure and malnutrition risk: a random order crossover feeding trial (MEDIMEALS), *BMC Nutrition* (2025). DOI: [10.1186/s40795-025-01036-y](https://doi.org/10.1186/s40795-025-01036-y)
Provided by [University of Pennsylvania School of Nursing](#)

APRIL 24, 2025

Six ways to keep your kidneys healthy

by Taylor Hisey Pierson , [Rush University Medical Center](#)



Credit: Pixabay/CC0 Public Domain

More than 35 million adults in the U.S. have [chronic kidney disease](#), and just a fraction of them even know it. And the number of cases is growing.

Kidneys are vital organs that filter waste from the blood and produce hormones. Left untreated, CKD, a form of kidney failure, allows toxins to build up in the body and can lead to other health problems, like [heart disease](#) and [stroke](#). It also can progress to end-stage renal disease, which requires dialysis treatment or a [kidney transplant](#) to live.

The key, doctors say, is to look for symptoms of CKD early. "In many instances, there is treatment available for kidney disease but in only a few instances, unfortunately, is there a cure," says Vasil Peev, MD, a nephrologist at Rush. "That's why it's important to intervene early and engage in prevention efforts for the disease."

Here are six tips to follow to help keep your kidneys healthy:

1. Know the signs and symptoms

Unfortunately, the symptoms of kidney disease aren't always noticeable, and 40% of people with severely reduced [kidney function](#) are not aware they have CKD. Therefore, it's crucial to know whether you might be at risk.

Risk factors of CKD can include the following:

- Diabetes
- Obesity
- High blood pressure
- Heart disease
- Age (60 and older)
- Family history of CKD

Peev recommends keeping up with your annual physical exams with your primary care provider, who may order bloodwork or urinalysis tests that can detect kidney disease in its early stages and help slow down its progression.

2. Maintain a healthy diet and weight

Research shows that a kidney-friendly diet and regular exercise may help protect your kidneys from the risk factors that can lead to kidney damage.

"A kidney-healthy diet is low in sodium and carbohydrates and includes lots of fruits and vegetables," Peev says. "If you follow this type of diet consistently, it can help prevent weight gain and avoid obesity, high blood pressure and diabetes, which are major risk factors for kidney disease."

Additionally, incorporating 30 minutes of exercise each day, such as walking, jogging and biking, among other activities, as well as drinking plenty of water, can also keep your kidneys healthy.

"Dehydration reduces blood flow to your kidneys, which can damage them, so make sure to drink enough water throughout the day—around eight glasses," Peev says.

If you need additional support, Peev suggests working with a dietitian who can provide a kidney-healthy diet fit for your individual needs.

3. Monitor your diabetes, blood pressure and cholesterol levels

In the United States, diabetes and high blood pressure are the leading causes of kidney failure.

"Diabetes is vastly driven by obesity, which can cause high blood pressure and cholesterol," Peev says. "And diabetic kidney disease is the No. 1 reason for developing end-stage CKD, so it's important that you have it under control."

Further, a study by the National Kidney Foundation found that people with high cholesterol are twice as likely to develop CKD.

If you are overweight and can lose weight through changes in your diet and activity level, Peev says you can significantly reduce your risk of CKD.

4. Don't overuse over-the-counter drugs

Nonsteroidal anti-inflammatory drugs (NSAIDs) are a specific group of over-the-counter pain relievers that include ibuprofen and aspirin, that may cause an increased risk of sudden kidney failure and progressive kidney damage.

"Many common over-the-counter drugs contain certain toxins that affect our kidneys' ability to filter them, and an excess amount can be dangerous," Peev says. "Also, a lot of people already have preexisting kidney damage but are unaware. So, their ability to clear the toxins is even more limited and only adds to the potential damage."

The National Kidney Foundation recommends limiting the use of these over-the-counter drugs to no more than 10 days for [pain relief](#) or more than three days for reducing a fever. If your pain or fever lasts longer, consult with a doctor.

5. Limit your alcohol intake

Drinking too much alcohol can harm many parts of the body, including the kidneys.

The National Kidney Foundation warns that regular heavy drinking affects the kidneys and their function and doubles the risk for kidney disease. Consuming more than three drinks a day for women and more than four drinks a day for men is considered heavy drinking, according to the foundation.

"A small amount of alcohol—one or two drinks every now and then—tends not to have any serious effects," Peev says. "But it's important to remember that drinking in moderation is key."

6. Quit smoking

Smoking tobacco slows the blood flow to important organs, such as the kidneys, and can increase the risk for kidney disease. Smoking can also make medications used to treat [high blood pressure](#) less effective, and if poorly controlled, can lead to kidney disease.

A study found that a group of participants who went through a smoking cessation program saw less of a decline in their kidney function when compared to a group of active smoker participants.

Although quitting smoking for good can be a challenge, a [smoking cessation program](#) can help. When you are ready, talk to your primary care provider, who can provide a referral as you work to maintain the health of your kidneys.

APRIL 28, 2025

Global study links consumption of ultraprocessed foods to preventable premature deaths

by [Elsevier](#)



Credit: Unsplash/CC0 Public Domain

A study analyzing data from nationally representative dietary surveys and mortality data from eight countries (Australia, Brazil, Canada, Chile, Colombia, Mexico, United Kingdom, and United States) shows that premature deaths attributable to consumption of ultraprocessed foods (UPFs) increase significantly according to their share in individuals' total energy intake.

The new study, appearing in the *American Journal of Preventive Medicine*, reinforces the call for global action to reduce UPF [consumption](#), supported by regulatory and fiscal policies that foster healthier environments.

UPFs are ready-to-eat-or-heat industrial formulations that are made with ingredients extracted from foods or synthesized in laboratories, with little or no whole foods in their composition. These have gradually been replacing [traditional foods](#) and meals made from fresh and minimally processed ingredients.

Lead investigator of the study, Eduardo Augusto Fernandes Nilson, DSc, Oswaldo Cruz Foundation (Fiocruz), Brazil, says, "UPFs affect health beyond the individual impact of high content of critical nutrients (sodium, [trans fats](#), and sugar) because of the changes in the foods during industrial processing and the use of artificial ingredients, including colorants, artificial flavors and sweeteners, emulsifiers, and many other additives and processing aids, so assessing deaths from all-causes associated with UPF consumption allows an overall estimate of the effect of industrial food processing on health."

While previous studies focused on specific dietary risk factors instead of food patterns, the current study modeled data from nationally representative dietary surveys and [mortality data](#) from eight countries (Australia, Brazil, Canada, Chile, Colombia, Mexico, United Kingdom, and United States) to link dietary patterns, considering the extent and purpose of industrial food processing, to deaths from all causes.

Table 3: Premature deaths attributable to UPF consumption levels in 8 countries.

Country, year	UPF intake	Population Attributable Fraction	Premature deaths	
	(% of total energy)	(%)	Total	Attributable to UPF consumption
Colombia, 2015	15.0	3.9	72,940	2,813
Brazil, 2017-18	17.4	4.5	556,696	25,296
Chile, 2010	22.8	5.7	32,607	1,874
Mexico, 2016	24.9	6.3	272,293	17,110
Australia, 2011-12	37.5	9.4	34,994	3,277
Canada, 2016	43.7	10.9	70,994	7,735
UK, 2018-19	53.4	13.8	128,743	17,781
US, 2017-18	54.5	13.7	906,795	124,107

Premature deaths attributable to ultraprocessed food (UPF) consumption levels in 8 countries increase significantly according to its share in the total energy intake, varying

from 4% of the premature deaths in lower consumption settings, such as Colombia, to up to approximately 14% of the premature deaths in countries with the highest UPFs intake, such as the U.S. and the UK. Credit: *American Journal of Preventive Medicine* (2025). DOI:

10.1016/j.amepre.2025.02.018

Dr. Nilson explains, "We first estimated a linear association between the dietary share of UPFs and all-cause mortality, so that each 10% increase in the participation of UPFs in the diet increases the risk of death from all causes by 3%.

"Then, using the relative risks and the food consumption data for all countries (ranging from 15% of the total energy intake in Colombia, to over 50% of the calories in the United States), we built a model that estimated that the percentage of all-cause premature preventable deaths due to the consumption of UPFs can vary from 4% in countries with lower UPF consumption to almost 14% in countries with the highest UPF consumption. For example, in 2018, 124,000 premature deaths were attributable to the consumption of UPFs in the United States."

High consumption of UPFs has been associated with 32 different diseases, including cardiovascular disease, obesity, diabetes, some types of cancer, and depression. For the first time, this study has estimated the burden of UPF intake on premature deaths from all causes in different countries, showing that the attributable mortality is significant in all settings and that addressing UPF consumption should be a global public nutrition priority.

Dr. Nilson notes, "It is concerning that while in high-income countries UPF consumption is already high but relatively stable for over a decade, in low- and middle-income countries the consumption has continuously increased, meaning that while the attributable burden in high-income countries is currently higher, it is growing in the other countries. This shows that policies that disincentivize the consumption of UPFs are urgently needed globally, promoting traditional dietary patterns based on local fresh and minimally processed foods."

More information: *American Journal of Preventive Medicine* (2025). DOI:

10.1016/j.amepre.2025.02.018

Journal information: *American Journal of Preventive Medicine*

APRIL 28, 2025

Hospital-based outbreak detection system stops outbreaks, saves lives

by [University of Pittsburgh](#)



Credit: CC0 Public Domain

An infectious disease detection platform developed by University of Pittsburgh scientists working with UPMC infection preventionists proved over a two-year trial that it stops outbreaks, saves lives and cuts costs.

The results are [published](#) in the journal *Clinical Infectious Diseases*, making the case for adoption in hospitals nationwide and the development of a national early [outbreak](#) detection database.

"We saved lives while saving money. This isn't theoretical—this happened in a real [hospital](#) with real patients," said lead author Alexander Sundermann, Dr.P.H., assistant professor of infectious diseases in Pitt's School of Medicine. "And it could easily be scaled. The more hospitals implement this practice, the more everyone benefits, not just by stopping previously undetected outbreaks within the walls of the hospital, but by finding [medical device](#) or medication-linked outbreaks sweeping the nation."

The Enhanced Detection System for Healthcare-Associated Transmission (EDS-HAT) takes advantage of increasingly affordable [genomic sequencing](#) to analyze infectious disease samples from patients. When the sequencing detects that any two or more patients have near-identical strains of an [infection](#), it flags the results for the hospital's infection prevention team to find the commonality and stop the transmission.

Without genomic sequencing, hospital infection preventionists have no way of knowing if two hospitalized patients coincidentally have the same infection or if one of them was infected by the other. Because of this, patients with the same type of infection who don't have an obvious link—such as staying in the same inpatient unit—may unknowingly spread the infection, leading to an outbreak growing significantly before it is detected.

Conversely, infection preventionists may spend time and resources trying to avert a nonexistent outbreak when patients happen to have the same type of infection, but the transmission was from unrelated sources.

The study ran from November 2021 through October 2023 at UPMC Presbyterian Hospital. During that time, the analysis showed that EDS-HAT prevented 62 infections and five deaths, compared to if the system had not been running. It netted a savings of nearly \$700,000 in infection treatment costs—a 3.2-fold return on investment.

"These results are remarkable," said co-author Graham Snyder, M.D., M.S., medical director of infection prevention and hospital epidemiology at UPMC. "This project clearly illustrates how UPMC's academic partnership with Pitt is providing our patients with outstanding patient care while creating innovative solutions that pave the way for better patient care worldwide."

If health care facilities across the U.S. adopt EDS-HAT, a nationwide outbreak system could be developed, similar to PulseNet, the U.S. Centers for Disease Control and Prevention's network for detecting multistate outbreaks of foodborne illness. Sundermann and colleagues previously found that, had such a system existed, the 2023 outbreak of deadly bacteria linked to contaminated eye drops could have been stopped far earlier.

"It is a no-brainer to implement EDS-HAT at every health care facility nationwide," said senior author Lee Harrison, M.D., professor of infectious diseases at Pitt's School of Medicine and of epidemiology at Pitt's School of Public Health. "We hope these findings will contribute to ongoing conversations among U.S. health care leadership, payors and policymakers about the benefits of genomic surveillance as standard practice in health care."

Additional authors of this research are Praveen Kumar, Ph.D., Marissa P. Griffith, Kady D. Waggle, M.S., Vatsala Rangachar Srinivasa, M.P.H., Nathan Raabe, M.P.H., Emma G. Mills, Hunter Coyle, Deena Ereifej, M.P.H., Hanna M. Creager, Ph.D., Ashley Ayres, M.B.A., Daria Van Tyne, Ph.D., Lora Lee Pless, Ph.D., and Mark Roberts, M.D., all of Pitt, UPMC or both.

More information: Alexander J Sundermann et al, Real-Time Genomic Surveillance for Enhanced Healthcare Outbreak Detection and Control: Clinical and Economic Impact, *Clinical Infectious Diseases* (2025). DOI: [10.1093/cid/ciaf216](https://doi.org/10.1093/cid/ciaf216)

Journal information: [Clinical Infectious Diseases](#)

Provided by [University of Pittsburgh](#)

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APRIL 27, 2025

[The GIST](#)
[Editors' notes](#)

Heart donation after circulatory death technique expands transplant access

by International Society for Heart and Lung Transplantation

Researchers at the Annual Meeting and Scientific Sessions of the International Society of Heart and Lung Transplantation ([ISHLT](#)) marked the 10-year anniversary of modern heart donation after circulatory death (DCD), a technique that has significantly increased transplant volumes around the world.

Sarah Scheuer, MD, Ph.D., said that most centers that have started a DCD program experience an approximately 30 percent increase in their transplant volume.

"It's arguably the biggest shift in heart transplantation since the introduction of modern immunosuppression," she said.

In a DCD transplant, the heart comes from a donor whose circulatory and respiratory functions have stopped but who did not meet criteria for brain death.

"The first heart transplants performed in the '60s and '70s all preceded the legislation surrounding brain death, so in essence, they were DCD," said Dr. Scheuer, MD, Ph.D., a cardiothoracic surgeon trainee at St. Vincent's Hospital in Sydney, Australia.

After brain death criteria were established in 1981, donation after brain death (DBD) was the predominant source of donated hearts for nearly five decades.

The first modern DCD heart transplant was performed at St. Vincent's Hospital in 2014; the Royal Papworth Hospital in Cambridge, England, followed with their first in 2015. The modern DCD pathway was developed over a 10-year period of research and laboratory work conducted in Australia and the UK, both of which are challenged by the limited availability of donor organs.

In 2022, a randomized, controlled trial in the United States showed that transplantation outcomes with DCD hearts were comparable to traditional (DBD) hearts.

"The successful US trial led to a real explosion of the field within the U.S. with increasing numbers of centers starting modern DCD heart transplant programs," Dr. Scheuer said. "It's a huge impact on the availability of donor hearts."

Over the past decade, 123 DCD hearts have been transplanted as part of the St. Vincent's DCD program, and 134 DCD hearts have been transplanted at Royal Papworth Hospital.

"Papworth and St. Vincent's have both had a long history of innovation and research within our heart transplant programs," said Dr. Scheuer. "We're always pushing the boundaries,

partially out of necessity, and also out of a desire to offer transplantation to more people because, in many ways, it's the best treatment for end-stage heart failure."

Dr. Stephen Pettit, a [transplant](#) lead and consultant cardiologist at Royal Papworth Hospital, said DCD heart transplantation is gradually spreading worldwide, increasing the use of hearts that otherwise could not have been donated.

"Families of potential organ donors, who must give permission before organs are donated in the UK, have been very supportive of DCD heart transplantation," Dr. Pettit said. "The DCD process makes sense to families who sometimes struggle with the concept of [brain death](#)."

Researchers are looking for biomarkers to help identify whether DCD hearts are in good condition before transplantation and exploring less costly options for preserving the donor heart (called perfusion) during transportation. A shift toward regenerative medicine is also on the horizon.

"We may get to the point where we put a heart on a perfusion or preservation device and spend the next few hours effectively improving the function of that organ," said Dr. Scheuer. "I think we'll also get more clarity on the best method of preserving hearts during DCD transplantation."

For now, she's gratified by the many lives changed by transplantation, including a St. Vincent patient who received a DBD heart as a teenager and, more recently, a second DCD heart in his 40s.

"We're at a point with [heart transplantation](#) where some patients will live a relatively normal duration of life," she said. "I don't think we have any other treatment for heart failure that can offer that potential outcome."

Provided by International Society for Heart and Lung Transplantation

APRIL 28, 2025

Low doses of plastic chemical DEHP found to disrupt key hormones and fertility in females

by [Karolinska Institutet](#)

A study from the Institute of Environmental Medicine (IMM) at Karolinska Institutet [published](#) in *Toxicological Sciences* shows that the chemical di(2-ethylhexyl) phthalate (DEHP), which is found in many plastic products, has clear negative effects on hormones and fertility endpoints relevant to female fertility.

The results show that key reproductive processes, such as hormone balance and ovarian follicle count, are affected at lower doses than the effects often used in assessing the risks of chemicals for female fertility.

Endocrine-disrupting chemicals (EDCs), such as DEHP commonly found in plastics, can harm human reproductive health by disrupting the endocrine system. DEHP is already classified as an [endocrine disruptor](#) due to its negative effects on male [reproduction](#).

The current study, in which female mice were exposed to different doses of DEHP, shows that female reproduction is also affected. Parameters such as ovarian follicular count and progesterone levels were particularly sensitive to the exposure. These effects were dose-dependent and more pronounced than changes in [body weight](#) or estrous cycles, parameters often used as signs of reproductive toxicity in females.

"Current regulatory guidelines often overlook women-specific risks, mainly due to a lack of knowledge about sensitive effects. This underlines the need to update and strengthen today's safety assessments for chemicals," says Antero Vieira Silva, postdoctoral researcher at the Institute of Environmental Medicine (IMM).

Using advanced modeling, the researchers analyzed data from four animal studies in which adult [female mice](#) had been exposed to increasing doses of DEHP. The results show that female reproductive effects, such as progesterone levels and the number of follicles, are particularly sensitive.

Despite this, these effects are not included as in the tests required to assess the impact of chemicals on female reproduction—meaning that important effects can be overlooked. The researchers also reviewed safety data for DEHP from the European Chemicals Agency (ECHA) and found that the current risk assessments are mainly based on results from males.

"We need to get better at protecting female fertility," says Antero Vieira Silva, first author of the scientific article. "Our results show that important parts of female reproduction are more sensitive to chemicals than what current regulations take into account."

There is a large knowledge gap regarding how chemicals affect [female fertility](#), and the research team hopes that the study will contribute to an update of safety standards to protect female reproductive health from chemical exposure.

"Traditionally, there has been more focus on the effects of chemicals on male fertility, but our research shows that female reproduction can be at least as sensitive, or even more. It is therefore crucial to take this into account when assessing the risk of DEHP, phthalates and other chemicals," says Pauliina Damdimopoulou, senior lecturer and research group leader at the Department of Clinical Science, Intervention and Technology.

More information: Antero Vieira Silva et al, Bayesian benchmark dose modeling analysis and derivation of points of departure for female reproductive toxicity following exposure to di(2-ethylhexyl) phthalate (DEHP) – effects on reproductive hormones, folliculogenesis and estrous cyclicity, *Toxicological Sciences* (2025). DOI: [10.1093/toxsci/kfaf052](https://doi.org/10.1093/toxsci/kfaf052)

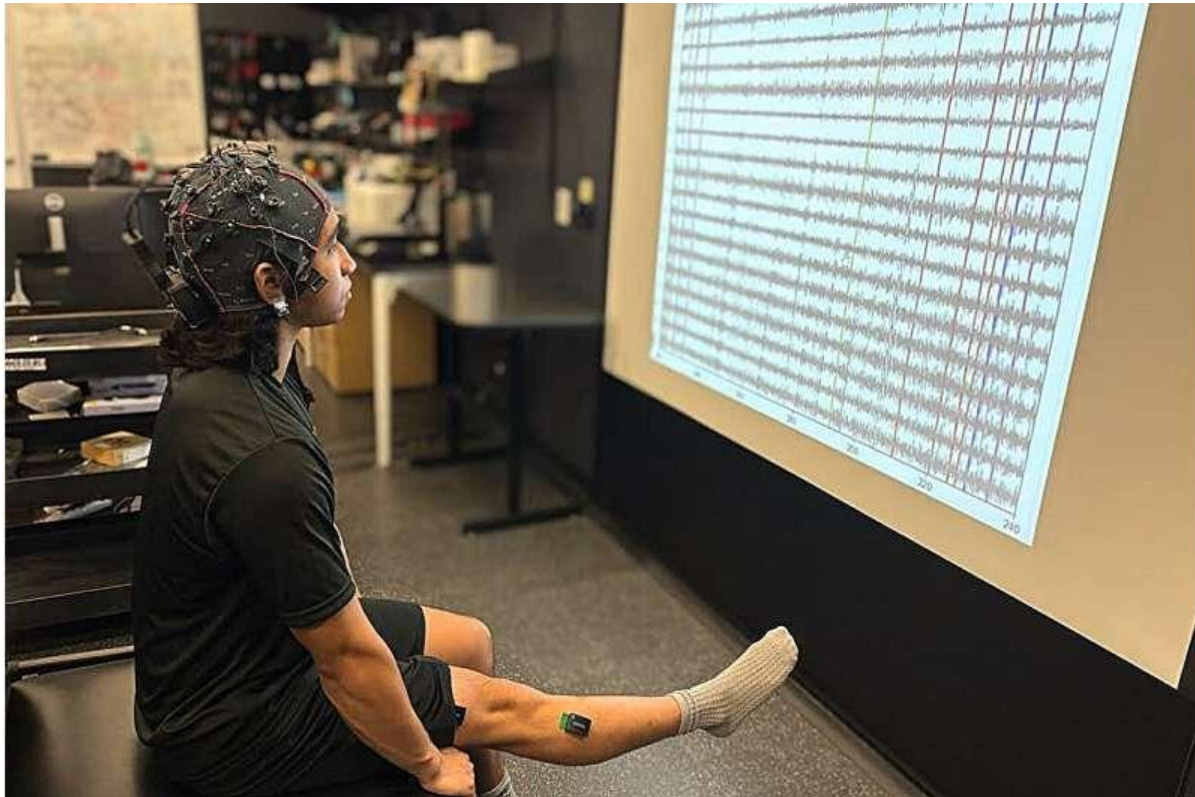
Journal information: [Toxicological Sciences](#)

Provided by [Karolinska Institutet](#)

APRIL 28, 2025

Brain wave decoder helps control spinal cord stimulation

by Beth Miller , [Washington University in St. Louis](#)



Researchers in Ismael Seáñez's lab have developed a type of decoder to restore communication between the brain and the area below a spinal cord injury. Through experiments in their lab with 17 human subjects without a spinal cord injury, they were able to cue movement in the lower leg with transcutaneous spinal cord stimulation, or noninvasive, external electrical pulses. Credit: Carolyn Atkinson

When a person sustains an injury to the spinal cord, the normal communication between the brain and the spinal circuits below the injury is interrupted, resulting in paralysis. Because the brain is functioning normally, as is the spinal cord below the injury, researchers have been working to re-establish communication to allow for rehabilitation and potentially restore movement.

Ismael Seáñez, assistant professor of biomedical engineering in the McKelvey School of Engineering at Washington University in St. Louis and neurosurgery at WashU Medicine, and members of his lab, including Carolyn Atkinson, a doctoral student, have developed a type

of decoder to restore that communication. Through experiments in their lab with 17 [human subjects](#) without a spinal cord injury, they were able to cue [movement](#) in the lower leg with transcutaneous spinal cord stimulation, or noninvasive, external electrical pulses.

Results of the research [appear](#) in the *Journal of NeuroEngineering and Rehabilitation*.

The team used a special cap fitted with noninvasive electrodes that measure brain activity through electroencephalography (EEG). While wearing the cap, seated volunteers were asked to extend their leg at the knee, then to only think about extending their leg—while keeping it still—so researchers could record the brain waves in both exercises.

The team provided the [neural activity](#) to the decoder, or algorithm, so it could learn how the brain waves act in both circumstances. They found that the actual movement and imagined movement used similar neural strategies.

"After we give the decoder this data, it learns to predict based on neural activity whenever there is movement or no movement," Seáñez said. "We show that we can predict whenever someone is thinking about moving their leg, even if their leg does not actually move."

The team used controls to ensure that the volunteers were truly imagining movement and not actually moving.

"Whenever people move, this can introduce signal noise, and we want to make sure that the signal noise is not what we're learning to predict," Seáñez said. "It's movement intention or [brain activity](#) that we want to predict, so we have people imagine that they're extending their leg, and use the same algorithm that has been trained on people moving to predict whether they were imagining or not."

Seáñez said this reveals two things: "One, that it's more likely that we're decoding movement intention and not an artifact, or noise; and second, whenever we employ this on people with spinal cord injury who will not have that ability to actually move their legs for us to label the data, we could use their imagination of moving a leg to train our decoder."

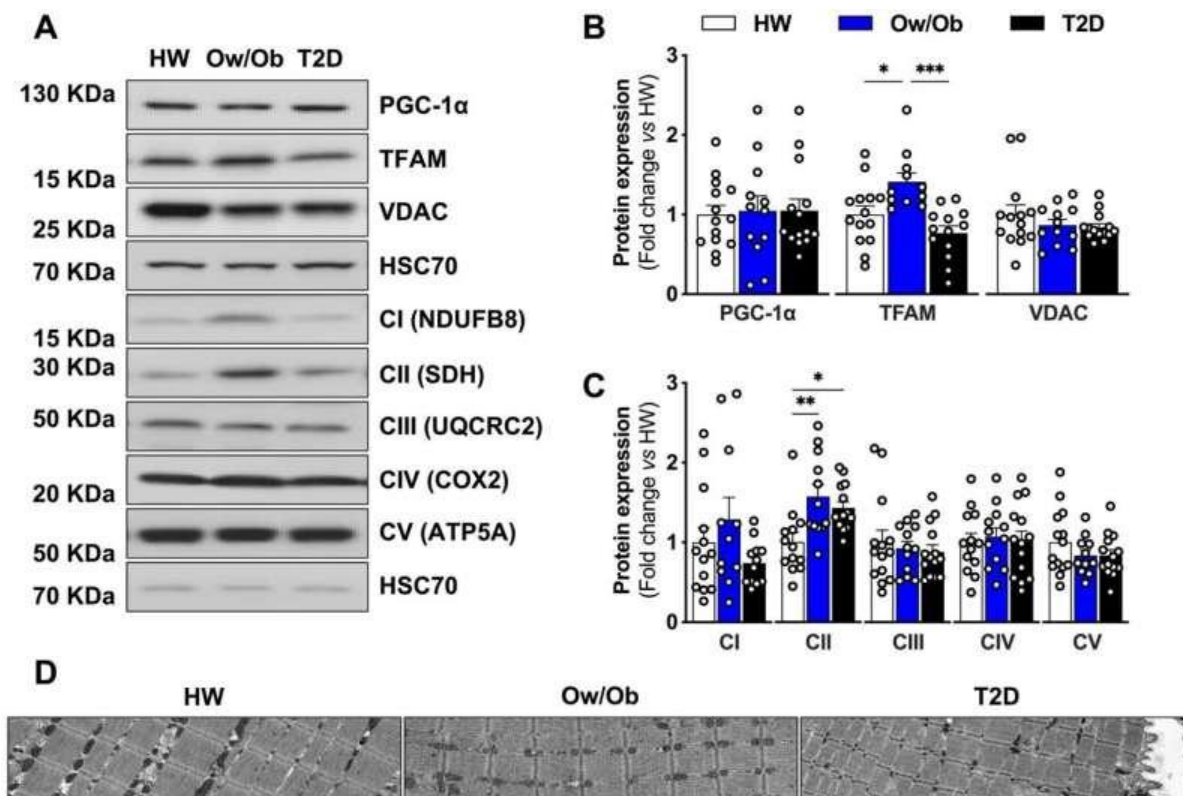
Seáñez said the proof-of-concept study is a first step toward developing a noninvasive brain-spine interface that uses real-time predictions to deliver transcutaneous spinal cord stimulation to reinforce voluntary movement in a single joint in rehabilitation in patients with a [spinal cord injury](#).

Going forward, the team plans to test a generalized decoder trained on data from all participants that could determine whether a universal decoder could perform as well as a personalized one and simplify its use in clinical settings.

More information: Carolyn Atkinson et al, Development and evaluation of a non-invasive brain-spine interface using transcutaneous spinal cord stimulation, *Journal of NeuroEngineering and Rehabilitation* (2025). DOI: [10.1186/s12984-025-01628-6](#)
Provided by [Washington University in St. Louis](#)

Cellular quality control linked to insulin resistance in type 2 diabetes

by [Pennington Biomedical Research Center](#)



Expression of proteins regulating mitochondrial biogenesis and content. Credit: *Journal of Cachexia, Sarcopenia and Muscle* (2025). DOI: 10.1002/jcsm.13763

Researchers at Pennington Biomedical Research Center have revealed critical insights into how impaired mitochondrial dynamics and quality control mechanisms in skeletal muscle influence insulin sensitivity in patients with type 2 diabetes, or T2D. The study, titled "Deubiquitinating Enzymes Regulate Skeletal Muscle Mitochondrial Quality Control and Insulin Sensitivity in Patients with Type 2 Diabetes," was recently [published](#) in the *Journal of Cachexia, Sarcopenia and Muscle*.

The research team, led by Pennington Biomedical Executive Director Dr. John Kirwan, focused on the significance of deubiquitinating enzymes, or DUBs, in regulating mitochondrial dynamics within skeletal muscle. Findings suggest that mitochondrial fragmentation can bypass defects in mitophagy, the process by which cells remove

damaged mitochondria, to sustain skeletal muscle quality control in patients with T2D. This adaptation may help maintain mitochondrial function despite impaired mitophagy.

In other words, the study shows that people with T2D have fewer healthy mitochondria, the parts of the cell that produce energy, because a certain protein—dynamin-related protein 1, or DRP1—is working too much. In addition, another group of proteins—DUBs—are shown to interfere with a process that helps the body clean up damaged mitochondria. This interference makes it harder for muscles to use insulin properly, which is a key issue in [diabetes](#).

The research findings advance the understanding of how impaired mitochondrial dynamics and quality control may contribute to [skeletal muscle](#) insulin resistance and the manifestation of T2D, and also provide key evidence that DUB antagonists may play an important role in preventing or treating T2D.

"Our research team examined how certain enzymes affect mitochondria in muscle cells of people with diabetes," said Dr. Kirwan, Executive Director and George A. Bray, Jr. Endowed Super Chair in Nutrition at Pennington Biomedical.

"We found that when the normal cleanup process for damaged mitochondria isn't working well, the cells adapt by breaking mitochondria into smaller pieces. This alternative approach helps maintain muscle function despite the metabolic challenges of type 2 diabetes. Our study highlights the complex interplay between [mitochondria](#) and insulin, paving the way for future interventions aimed at improving metabolic health."

Pennington Biomedical played a pivotal role in this study, with several key researchers from the Integrated Physiology and Molecular Medicine Laboratory contributing to the investigation. The Center's commitment to advancing metabolic research continues to drive discoveries that have significant implications for public health.

More information: Wagner S. Dantas et al, Deubiquitinating Enzymes Regulate Skeletal Muscle Mitochondrial Quality Control and Insulin Sensitivity in Patients With Type 2 Diabetes, *Journal of Cachexia, Sarcopenia and Muscle* (2025). DOI: [10.1002/jcsm.13763](https://doi.org/10.1002/jcsm.13763)
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APRIL 29, 2025

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Stroke in young people is not that rare, says stroke specialist

by Grace van Deelen , [Tufts University](#)

People typically think of stroke as an elderly person's ailment. Stroke risk does increase by age, but young adults (ages 18–55) can have strokes, too. In fact, the proportion of strokes affecting young adults in the United States has been growing for the past few decades.

At the Tufts Medical Center's Stroke and Young Adults Program (SAYA), Lester Y. Leung, a vascular neurologist, stroke specialist, and associate professor at Tufts University School of Medicine, works to improve [stroke recovery](#) for younger patients. Each day he sees firsthand how young stroke patients face a unique challenge.

"Stroke in young people is actually not that rare, but young stroke patients often end up being kind of invisible," Leung said.

A worrying trend

Globally, stroke in younger adults [now accounts for](#) about 10% of new cases of stroke each year. In the United States, stroke rates [increased](#) by about 64% between 1993 and 2015, and one [nationwide study](#) found that rates of hemorrhagic stroke—the second-most common type of stroke—among people 18-44 years old jumped by 38% between 2004 and 2018.

Notably, stroke is usually not a fatal disease, so young stroke survivors have to deal with the aftereffects of stroke for decades and likely represent a larger portion of stroke survivors than suggested by the 10% statistic.

Scientists are still studying why strokes may be on the rise among young people. In general, the rise indicates that young Americans aren't managing common [stroke risk](#) factors, like cardiovascular disease, [high blood pressure](#), and diabetes, well. But some factors more specific to young people, such as oral contraceptive use, pregnancy, migraine, and recreational drug use, can also contribute to the risk of stroke.

Helping young people cope

Overall, young adults tend to have less severe strokes, with less intense loss of function. "The main deficits of stroke may not be as severe," Leung said, adding that outwardly, it may not seem like a young stroke patient is struggling as much as an elderly one with language or movement. But strokes can cause other disabilities in young people that may be misunderstood.

For example, seizures are a concerning complication of stroke that rarely arises in stroke patients over 70 years old. But for young adults, the risk of developing seizures within five years after a stroke is much higher, around 20%, Leung said. Fatigue, depression, anxiety, and [chronic pain](#) are also [common](#) but invisible late complications after stroke among young people.

And young adults often suffer these complications in the midst of demanding life roles—they may be at the peak of their career, raising children, attending school, or working toward athletic goals. That can make stroke recovery much more frustrating, especially if employers or schools are unwilling to grant accommodations.

"The experience for a lot of young stroke survivors is very tough," Leung said. "The goals are set higher—I have patients who are surgeons, or computer programmers, or have hobbies that are very important to them that involve physical ability."

At the SAYA program, Leung tailors his care to the specific and sometimes ambitious goals of his young patients. He also checks in with them more frequently and over a longer time

period than typical providers, who might see a patient just once after they leave the hospital.

"It's becoming increasingly evident that younger folks and middle-aged folks need more help over longer periods of time," he said. "Besides preventing future strokes, we often focus on how to unlock more recovery potential and achieve their life goals that were interrupted by their strokes."

Provided by [Tufts University](#)

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APRIL 29, 2025

[The GIST](#)

Bridging the AI gap in medicine: New framework targets family doctor education

by JMIR Publications

A team of Canadian researchers has developed a curriculum framework to help train future family physicians in the use of artificial intelligence (AI), addressing a critical gap in medical training as digital tools become more common in patient care.

Published in *JMIR Medical Education*, the [study](#), "Curriculum Framework for AI Training in Postgraduate Family Medicine Education (AIFM-ed): Mixed Methods Study," introduces the AIFM-ed framework to guide the integration of AI into family medicine training programs.

As the health care system evolves, many [medical professionals](#) feel unprepared for the growing influence of AI in diagnostics, treatment, and patient management.

This newly published framework co-led by Dr. Samira A Rahimi and her trainee Raymond Tolentino offers a structured road map for medical educators to bridge that gap. Developed through a comprehensive review of existing AI education models and in-depth consultations with experts and residents from across Canada, the AIFM-ed framework outlines five core components: need and purpose of the curriculum, learning objectives, curriculum content, organization of curriculum content, and implementation aspects of the [curriculum](#).

With every [medical school](#) being different, the framework is designed to be flexible, so it can be adapted to each program's specific needs and resources.

"AI is rapidly transforming health care, and yet, most medical curricula haven't caught up. AIFM-ed framework provides a critical foundation to ensure that our future physicians are ready for the realities of digitally enabled care," said Dr. Samira A. Rahimi, Canada Research Chair of AI and Advanced Digital Primary Health Care at McGill University and Mila-Quebec AI Institute.

Raymond Tolentino, a recent master of science graduate of Dr. Rahimi's lab and co-lead on the study, emphasized how important this work is for preparing future doctors. "We're not just teaching new skills—we're making sure family doctors feel confident using AI to support better, safer [patient care](#)," he said.

The next step is for institutions to pilot the framework and assess its impact on postgraduate [medical training](#). By doing so, they can ensure that the next generation of family doctors is not only clinically competent but also technologically fluent.

As AI continues to reshape medicine, this framework represents a proactive step toward preparing doctors for the future of digital health.

More information: Raymond Tolentino et al, AIFM-ed Curriculum Framework for Postgraduate Family Medicine Education on Artificial Intelligence: Mixed Methods Study, *JMIR Medical Education* (2025). DOI: [10.2196/66828](https://doi.org/10.2196/66828)
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MAY 6, 2025

[The GIST](#)
[Editors' notes](#)

Half of US adults acknowledge health benefits of eating a plant-based diet

by Physicians Committee for Responsible Medicine



Credit: Unsplash/CC0 Public Domain

Half of U.S. adults say they know eating a plant-based diet can improve their health and help prevent chronic diseases, according to a new Physicians Committee for Responsible Medicine/Morning Consult [survey](#). However, the survey also showed that just 1 in 5 primary care practitioners discuss this lifesaving message with patients.

The poll included 2,203 U.S. adults surveyed April 15 to April 16, 2025.

Xavier Toledo, a registered dietitian with the Physicians Committee, a health advocacy group with 17,000 physician members, lauded the findings, which show most people recognize the health benefits of plant-based eating.

"What's missing," Mr. Toledo said, "is support and guidance from health care professionals. This represents a huge missed opportunity to turn interest into action—and to reduce the risk of [chronic diseases](#) that affect millions."

When asked if they'd be willing to try a [plant-based diet](#) if shown evidence of its benefits, 65% of survey respondents said they would.

A wealth of studies supports this, including:

- A [major 2023 meta-analysis](#) of more than 2 million people that found higher adherence to plant-based diets—especially those rich in fruits, vegetables, legumes, and whole grains—is associated with significantly lower risks of type 2 diabetes, [heart disease](#), cancer, and [early death](#). These findings reinforce the growing evidence that plant-based eating can help prevent many of the leading causes of death.
- A [large U.S. study](#) of more than 70,000 adults that found vegans and vegetarians have a lower risk of early death compared to people who eat meat. In the study, vegans saw up to a 15% lower risk overall, with especially strong results for heart disease. The findings come from the Adventist Health Study 2—one of the largest studies to directly compare plant-based and meat-based diets.
- A [2017 meta-analysis](#) that reviewed studies comparing vegetarian and vegan diets to meat-based diets and found that [vegetarian diets](#) were linked to a lower risk of heart disease and cancer. Vegans also had a lower risk of cancer, though fewer studies focused specifically on them. While most of the research looked at disease risk rather than lifespan, the findings support the broader conclusion that plant-based eating can reduce the risk of major chronic diseases.

To fill the gaps in medical schools, the Physicians Committee offers [plant-based education tools](#) to medical and nursing students to increase awareness of nutrition education in educational institutions.

And the organization recently praised New York legislators for introducing a [bill](#) to ensure physicians are kept informed about the latest continuing medical education opportunities in nutrition, which will help doctors in the state improve patient health outcomes and reduce racial disparities for the millions of New Yorkers struggling with diet-related diseases like type 2 diabetes and heart disease.

More information: Vegan Prevalence Survey: [pcrm.widen.net/s/l2kldfm9gs/pc ... lence-survey-4.28.25](https://pcrm.widen.net/s/l2kldfm9gs/pc-lence-survey-4.28.25)

For those ready to get started on a plant-based diet, the Physicians Committee has a free [Vegan Starter Kit](#) that contains recipes, links to resources, and more. The organization also publishes a [list](#) of plant-based health care providers.

Provided by Physicians Committee for Responsible Medicine

MAY 6, 2025

Nanoplastics are all around (and inside) us

by Renée Cho , State of the Planet



Credit: Unsplash/CC0 Public Domain

Each year, more than [400 million metric tons of plastic](#) are produced globally from petrochemicals derived from fossil fuels. Only [9% of this plastic is recycled](#) and 19% is incinerated; 72% goes to landfills, other dumpsites or ends up in our environment.

These plastics degrade into smaller and smaller pieces through exposure to sunlight, wind or waves, eventually becoming microplastics or even smaller nanoplastics.

What are nanoplastics?

Microplastics are plastic pieces or fibers less than 5 millimeters in size—smaller than a pencil eraser. As they degrade further, they become nanoplastics, which measure less than 1 micrometer—1/1,000 of a millimeter. Nanoplastics behave very differently from larger fragments of plastic because they are so tiny, have relatively large surface areas and are more reactive.

They cannot easily be observed, characterized or quantified. However, we do know that over 1,500 species, including humans, ingest these plastics. Nanoplastics have also been found in oceans, rivers, the Alps, Antarctic ice, foods and in bottled and tap water.

Nanoplastics in bottled water

A [2024 study](#) by Columbia University scientists revealed that a liter of [bottled water](#) (they tested three brands they did not identify) contained between 110,000 and 370,000 particles, 90% of which were nanoplastics; the rest were microplastics. The study's co-author, Beizhan Yan, an environmental chemist at Columbia Climate School's Lamont-Doherty Earth Observatory, said, "Previously, this was just a dark, uncharted area. Toxicity studies were just guessing what's in there."

But for this study, a new method called Raman scattering microscopy, developed by study co-authors Wei Min and chemistry graduate student Naixin Qian, hit plastic particles with laser beams, enabling the scientists to analyze their chemical structure and identify specific nanoplastics. Seven common plastics were recognized, including polyethylene terephthalate (PET), which [water bottles](#) are made of, and polyamide (PA), which is used to filter water before it is bottled.

Most of the nanoplastics appear to come from the bottle itself as well as from the reverse osmosis membrane filter used to purify the water before it is bottled. Other studies indicate that PET plastic particles can come from opening and closing the bottle cap, crushing the bottle or exposing it to heat, for example, in a car.

Nanoplastics in tap water

Tap water also contains nanoplastics, as microplastics and nanoplastics [are not completely removed](#) by wastewater and drinking [water treatment plants](#). Microfilters used in water treatment are effective at removing larger microplastics from raw water, but [studies have shown](#) that particles 1–20 micrometers in size remain after water is treated.

Reverse osmosis, which uses pressure to force water through a semi-permeable membrane, is used in many municipal water treatment systems, as well as in desalinization systems, to remove pollutants and other impurities in a third or fourth stage of treatment.

But reverse osmosis membranes degrade over time, releasing micro- and nanoplastics into the water. While one study found that reverse osmosis combined with other processes removed 93-98% of microplastics, nanoplastics have still been found in water after treatment.

The water distribution system itself—pipes, pumps and valves, especially old and worn ones—[can also be a source of microplastics and nanoplastics](#) through the corrosion of pipes and fittings. Pipes in drinking water distribution systems or households are often

made of polyethylene, polyvinylchloride, polypropylene or cast iron with PA fittings used to connect pipes in plumbing systems.

Where do nanoplastics originate?

Since [tires and laundry wastewater](#) have been identified as significant sources of microplastics, they are also likely the main sources of nanoplastics. It's estimated that 30% of a tire's weight is emitted into the environment from friction and braking.

Up to [279,972 metric tons](#) of acrylic, nylon and polyester microfibers come from the washing of synthetic materials and laundry wastewater around the world, of which about half makes its way into rivers, lakes or oceans. Yan and his Lamont colleague Joaquim Goes are working on ways to remove nanoplastics from laundry wastewater. Wastewater treatment plants and stormwater systems are major sources of nanoplastics that enter surface waters.

Microplastics are also found in sludge, a byproduct of the water treatment process. These accumulate from the PA and polyester in membranes, and polyacrylamide used during water treatment to facilitate the clumping of particles to make them easier to remove, and from the microplastics that remain from the original raw water. After sludge is treated to break down the organic matter within, it is often used as fertilizer or sent to landfills, and thus nanoplastics enter the soil.

How are humans exposed to nanoplastics?

We humans are exposed to nanoplastics throughout our lives, and because of the size of these particles, they can migrate through our bodies, penetrating tissues and cells. Their effects are not fully understood, because analytical techniques are not developed enough to quantify nanoplastics in the environment or in our bodies.

Most studies about the impact of nanoplastics on health have been conducted on animals or in cell cultures. And most have exposed organisms to very high concentrations of nanoplastics, not reflective of real-world environmental conditions. While the findings are worrisome, scientists do not yet know what effects real exposure levels might be having on human health, and therefore there are no established safe levels for micro- or nanoplastics in the body.

We are exposed to nanoplastics through what we eat. Nanoplastics in the ocean are consumed by microscopic organisms like plankton and algae; these organisms are eaten by small fish, which are then consumed by larger fish. As nanoplastics accumulate in these organisms, the tiny particles make their way up the food chain to humans. Human beings ingest nanoplastics in seafood, salt, chewing gum and many other foods, and through eating plants grown in soil and drinking water contaminated with nanoplastics.

We also breathe in nanoplastics. Car tires produce nanoplastics that enter the air around streets. Industrial plants that produce plastic emit aerosolized nanoplastics that enter the atmosphere, and eventually rain and water.

We are exposed through our skin by taking showers with water that carries nanoplastics, or using [personal care products](#) and cosmetics that contain nanoplastics.

Naixin Qian asserts, however, that humans are most directly exposed to nanoplastics through medical procedures. So far, this is an area that has not gotten much attention, but she and colleagues who are now researching this believe there is real urgency about exposure to nanoplastics in medical settings.

"If you go to the hospital and have an IV injection of a drug, that process actually delivers whatever plastic was in the liquid directly into your body," she said. "That is a much more dangerous process than, say, drinking bottled water. We know the package of the bag can introduce additional plasticizers, also in the form of nanoparticles, into the blood stream. And by imaging mice as a model, we see that the [nano]particles get distributed across the entire body to all varieties of organs."

Health care depends on [single-use plastics](#). For example, blood is collected in plastic bags for blood infusions. Dialysis involves plastic tubing and membranes for the entire dialysis process. Moreover, dialysis patients must have these procedures repeatedly.

"How many of those processes can introduce micro- and nanoplastics to the patients?" Qian asked. "And more importantly, these people already have bad health or an impaired immune system. So the toxicity implications from the nanoplastics are only going to be more significant and dangerous for them."

Impacts on human health

Ingested [nanoplastics can affect the microbiome](#), potentially leading to the development of inflammatory bowel disease, autoimmune diseases and cancer. Studies have shown that nanoplastics in the intestines also penetrate the intestinal barrier to enter the blood stream. The circulatory system can then carry nanoplastics to the neurological, musculoskeletal, reproductive and endocrine systems.

[According to studies of fish](#), nanoplastics caused an impaired heart rate, thrombosis, damage to the vascular structure, hormone changes, reduced immunity and altered blood biochemistry. A [2024 study](#) analyzed the plaque buildup removed from patients who had narrowing of the carotid arteries for micro- and nanoplastics. They found that 34 months later, patients with micro- and nanoplastics in their carotid artery plaque had a higher risk of heart attacks, stroke or death from any cause than patients in whom no plastic particles had been detected.

[Research](#) also shows that nanoplastics can cross the blood-brain barrier and cause brain damage in fish. [Mice fed nanoplastics](#) showed a decline in cognitive function and short-

term memory. One [recent study](#) analyzed the brains of people who had recently died and discovered 50% more plastic in them than brain samples from 2016.

The scientists also found that the brains of people with dementia contained far more [nanoplastic](#) in them than people without it, but they were not sure if this was because their brains had a less robust [blood-brain barrier](#).

Nanoplastics have also been found in the placenta, potentially causing harm to the [developing fetus](#), as well as in testes, semen and breast milk.

Inhaled nanoplastics can affect the respiratory tract, leading to chronic inflammation of the lungs, asthma, pulmonary fibrosis and increased risk of lung cancer.

Research has shown that almost every cell type in the body is affected if it comes into contact with nanoplastics, and generally, the interaction results in inflammation, which can cause other problems. However, scientists still do not understand the underlying mechanisms for these effects.

In addition to the impacts from the nanoplastic particles themselves, because of their large surface area, they can transport toxic chemicals such as the PCBs, dioxins, DDT, PAH, BPA and phthalates used in the manufacturing of plastic. Many of these chemicals are carcinogenic or endocrine disrupters. A new study has linked 356,238 deaths globally from cardiovascular disease to the phthalates typically used in personal care products, packaging and food containers.

Solutions to nanoplastic pollution

Researchers are trying to [develop better technologies](#) to remove nanoplastics from water. Physical methods include filtration using new membrane materials, and adsorption with various materials that can bind to pollutants and remove them from liquids. Magnetic particles are being developed that can be altered to attract nanoplastics and allow them to be removed by a magnetic field.

Electrochemical techniques apply electricity that enables certain materials to identify, separate and attract nanoplastics or break them into smaller, less harmful pieces. Biodegradable polymers that decompose into natural substances aided by microorganisms are being used to attract and bind to nanoplastics, allowing them to be easily removed.

Scientists at the University of Missouri developed a new nontoxic chemical solvent that captured nanoplastics from water, then rose back to the top, allowing for easy removal of 98% of the nanoplastics from both fresh and salt water in the lab.

Chinese researchers have experimented with boiling water and then filtering out the nanoplastics, [removing up to 90% of them](#). Qian said she herself boils her water before drinking it.

"Nanoplastics tend to aggregate and form larger particles at higher temperatures. By boiling the water, you can make small nanoparticles aggregate into larger, micro particles, thousands of them into one. That reduces their toxicity implications, because their larger size reduces their ability to cross the biological barrier and get into your cells," she said.

These removal techniques have shown promise in the lab, but more research is needed to see if they can be scaled up, and to determine their impact on the environment.

Qian believes it's also important to explore new materials to replace plastics and hopes to pursue post-graduate research in this area.

"There is a good chance we can build materials that will have friendly interactions with biological systems," she said. "There should be a way we can learn from nature and come up with solutions that are friendly to our health."

Right now, however, she hopes that the potential peril of micro- and nanoplastics in the medical area will be more widely acknowledged and that measures will be taken to address it.

"It's going to need collaborative efforts from the whole society, with manufacturing to reduce the exposure of micro- and nanoplastics in their products, agencies to impose regulations on products, and researchers from all different areas—material science, biology and chemistry—to understand their toxicity and provide alternatives to adopt," she said.

How to reduce your exposure to nanoplastics

Avoid:

- Bottled water
- Processed foods; their production and packaging convey nanoplastics
- Dishwasher or laundry pods
- Washing plastic products in hot water and microwaving plastics
- Single-use plastics, such as cutlery or cups
- Plastic wrap and packaging
- Cosmetics and personal care products containing microbeads, and silicone-based polymers used as thickeners; use bar soap and shampoo bars
- Clothes made from synthetic fibers such as polyester, nylon or spandex; choose natural fibers like cotton, wool or linen instead

Do:

- Buy and store products in glass if possible
- Filter your tap water or boil it before drinking.
- Opt for loose leaf teas; many tea bags shed nanoplastics
- Reduce seafood consumption

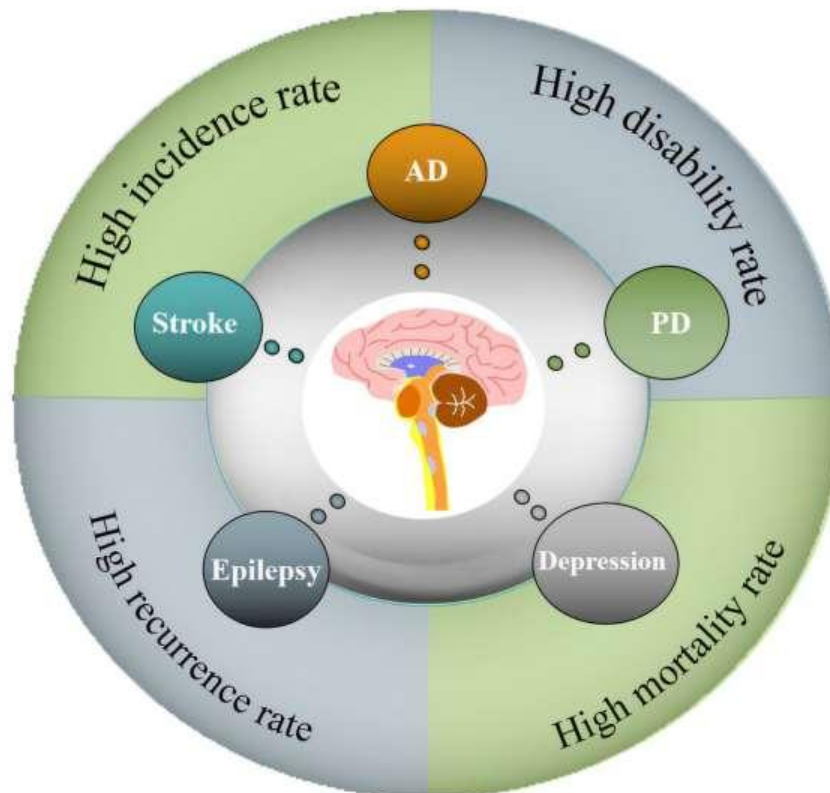
- Eat deeply colored fruits and vegetables. The antioxidants in them reduce microplastic-induced inflammation
- Be aware of plastics in medical procedures and avoid them if at all possible

Provided by State of the Planet

MAY 14, 2025

Traditional Chinese medicinal fungi might show potential for central nervous system diseases

by [Justin Jackson](#) , Medical Xpress



Central nervous system (CNS) diseases and characteristics (AD: Alzheimer's disease; PD: Parkinson's disease). Credit: *Journal of Food Science* (2025). DOI: 10.1111/1750-3841.70230

Shandong researchers suggest that edible and medicinal fungi contain bioactive components with therapeutic potential for central nervous system (CNS) diseases. Findings offer evidence for the potential of these fungi to serve as novel pharmaceuticals or functional foods in the treatment of CNS diseases.

Central nervous system (CNS) diseases involve complex pathophysiology, gradual symptom onset, and significant challenges in therapeutic evaluation. Aging populations and increasing life and work stress have contributed to a rising incidence of conditions such as stroke, Alzheimer's disease (AD), Parkinson's disease (PD), epilepsy, and depression, which are associated with high rates of morbidity and mortality.

Pharmacological development in CNS diseases remains limited, with existing treatments that do not halt disease evolution but only relieve symptoms. Adverse effects and incomplete mechanistic understanding further complicate drug discovery efforts in this area, resulting in lower success rates compared to other therapeutic fields.

Edible and medicinal fungi have been utilized in traditional Chinese medicine for centuries. Certain fungi contain polysaccharides, steroids, flavonoids, terpenoids, and alkaloids with documented antioxidant, anti-inflammatory, and neuroprotective effects. Recent studies have examined these [bioactive components](#) as potential therapeutic agents for CNS diseases, with specific compounds reported to target oxidative stress, neuroinflammation, and neuronal cell survival.

In the study, "New Dawn of Edible and Medicinal Fungi: Unlocking Central Nervous System Diseases," [published](#) in the *Journal of Food Science*, researchers conducted a comprehensive literature review of an untold number of previous studies to assess the therapeutic potential of bioactive components in edible and medicinal fungi for CNS diseases.

Studies on *Ganoderma lucidum*, *Cordyceps militaris*, and *Hericium erinaceus* were reviewed, focusing on reported neuroprotective effects, antioxidant properties, anti-inflammatory activities, and gut-brain axis interactions.

Neuroprotective, antioxidant, and anti-inflammatory effects were reported for bioactive components in *Ganoderma lucidum*, *Cordyceps militaris*, *Hericium erinaceus*, and *Antrodia camphorata*. Studies on *Ganoderma lucidum* addressed the role of deacetyl ganoderic acid F in regulating microglial and astrocytic activity. Selenium peptides from *Cordyceps militaris* were linked to modulation of inflammation and oxidative stress in preclinical models.

Antrodia camphorata alcohol extracts (AC-AE) were reported to reduce infarct size and increase antioxidant levels in ischemic stroke models. *Hericium erinaceus* was associated with regulation of monoamine neurotransmitters and anti-inflammatory pathways in studies of depression.

Fungi connected to traditional Chinese medicine were found to contain bioactive components that could serve as candidates for the development of pharmaceuticals and functional foods targeting CNS diseases. Neuroprotective, antioxidant, and anti-inflammatory effects suggest potential therapeutic applications in conditions such as Alzheimer's disease, Parkinson's disease, and epilepsy.

Researchers emphasize the need for further studies to validate the efficacy of specific compounds, and dosage optimization, under controlled experimental conditions. Standardization of extraction methods and comprehensive safety assessments are recommended to advance the clinical applicability of these fungi-derived bioactive components.

According to the study authors, "As the global health business continues to grow and human health awareness gradually increases, edible and medicinal fungi are becoming more and more popular due to their special benefits, increasing medicinal value, and steadily rising industrialization level."

As additional context from the ever-skeptical writer of this article with an admitted blindspot for believing supplement health claims (i.e., a medicine cabinet full of unverified mechanisms in edible wish form), the Chinese government continues to promote the strategic integration of the traditional Chinese medicine industry with Western pharmaceuticals under the "Healthy China 2030" initiative.

This policy emphasizes combining traditional Chinese medicine with Western pharmaceuticals as an adjunct treatment for chronic diseases, infectious diseases, and various other health conditions.

To reach this goal, a nationwide traceability system has been established, covering seeds, [raw materials](#), herbal production, documenting formulations and finished products along with advanced manufacturing and testing facilities to better adhere to Western Good Manufacturing Practice.

The National Administration of Traditional Chinese Medicine has put a great deal of support into research with a specific predetermined goal of finding applications for traditional Chinese medicine; essentially, starting with an existing compound, finding its Western therapeutic value, and linking it to a disease.

While traditional medicinal compounds cultivated for thousands of years will likely be found to contain useful bioactive components, it is just as inevitable that the research pipeline as constructed will include selection or confirmation biases. All research funded within this pipeline will require outside experimental verification, mechanistic understanding and registered clinical trials to confirm the existence of any potential effects.

More information: Xiaojin Liu et al, New Dawn of Edible and Medicinal Fungi Unlocking Central Nervous System Diseases, *Journal of Food Science* (2025). DOI: [10.1111/1750-3841.70230](https://doi.org/10.1111/1750-3841.70230)

Journal information: [Journal of Food Science](#)

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MAY 9, 2025

Cholesterol pill helps those at high risk of heart attack and stroke: Study

by [Monash University](#)

A Monash-led international study has found a cholesterol-lowering drug may offer a more effective and convenient way to protect people at high risk of heart attack and stroke.

The clinical trial "BROADWAY" tested a once-daily oral medication called Obicetrapib, and found it significantly lowered both LDL cholesterol and lipoprotein(a), [Lp(a)], two key contributors to [cardiovascular disease](#).

The BROADWAY phase 3 trial results were presented by Study lead Professor Stephen Nicholls, Director of Monash University's Victorian Heart Institute and Monash Health's Victorian Heart Hospital as a late-breaking clinical study at the European Atherosclerosis Society Congress in Glasgow, UK, and [published](#) in the *New England Journal of Medicine*.

Professor Nicholls said the findings marked an important step forward for patients who have struggled to reach their cholesterol targets with current therapies.

"We know that many people at high risk of heart attack or stroke don't get their [cholesterol levels](#) low enough, even on the best available treatments," Professor Nicholls said.

"Obicetrapib offers a promising new option—not only did it lower LDL cholesterol by over 30%, but we also saw a reduction in Lp(a), which is much harder to treat and has been linked to increased heart disease risk."

LDL cholesterol, often referred to as "bad cholesterol," builds up in [blood vessels](#) and increases the risk of heart attack and stroke. Lipoprotein(a), or Lp(a), is a lesser-known but inherited risk factor that can also accelerate artery damage—and unlike LDL, there are currently no widely approved treatments to lower it.

In the BROADWAY trial, more than 2,500 participants with established heart disease or genetic high cholesterol were given either Obicetrapib or a placebo, in addition to their regular cholesterol medications. After 12 weeks, those on Obicetrapib had dropped their LDL cholesterol by 32.6% and Lp(a) by 33.5% on average—many achieved guideline-recommended targets for the first time.

Obicetrapib was also well tolerated, with a safety profile similar to earlier trials.

"This could be a valuable tool in the fight against [heart disease](#)," Professor Nicholls said. "It's convenient, it's effective, and it may help close the gap for patients who've run out of options."

More information: Stephen J. Nicholls et al, Safety and Efficacy of Obicetrapib in Patients at High Cardiovascular Risk, *New England Journal of Medicine* (2025). DOI: [10.1056/NEJMoa2415820](https://doi.org/10.1056/NEJMoa2415820)

Journal information: [New England Journal of Medicine](#)
Provided by [Monash University](#)

MAY 9, 2025

Heart rhythm disorder traced to bacterium lurking in gums

by [Hiroshima University](#)

Immunohistochemical staining in mice shows *Porphyromonas gingivalis* (green) entering cardiac muscle through small blood vessels in the left atrium. Credit: Shunsuke Miyauchi/Hiroshima University

Tempted to skip the floss? Your heart might thank you if you don't. A new study from Hiroshima University (HU) finds that the gum disease bacterium *Porphyromonas gingivalis* (*P. gingivalis*) can slip into the bloodstream and infiltrate the heart. There, it quietly drives scar tissue buildup—known as fibrosis—distorting the heart's architecture, interfering with electrical signals, and raising the risk of atrial fibrillation (AFib).

Clinicians have long noticed that people with periodontitis, a common form of gum disease, seem more prone to cardiovascular problems. One recent meta-analysis has linked it to a 30% higher risk of developing AFib, a potentially serious heart rhythm disorder that can lead to stroke, heart failure, and other life-threatening complications.

Globally, AFib cases have nearly doubled in under a decade, rising from 33.5 million in 2010 to roughly 60 million by 2019. Now, scientific curiosity is mounting about how gum disease might be contributing to that surge.

Past research has pointed to inflammation as the likely culprit. When [immune cells](#) in the gums rally to fight infection, chemical signals they release can inadvertently seep into the bloodstream, fueling systemic inflammation that may damage organs far from the mouth.

But inflammation isn't the only threat escaping inflamed gums. Researchers have discovered DNA from harmful oral bacteria in heart muscle, valves, and even fatty arterial plaques. Among them, *P. gingivalis* has drawn particular scrutiny for its suspected role in a growing list of systemic diseases, including Alzheimer's, diabetes, and certain cancers. It

has previously been detected in the brain, liver, and placenta. But how it manages to take hold in the heart has been unclear.

This study, [published](#) in *Circulation*, provides the first clear evidence that *P. gingivalis* in the gums can worm its way into the [left atrium](#) in both animal models and humans, pointing to a potential microbial pathway linking periodontitis to AFib.

"The [causal relationship](#) between periodontitis and [atrial fibrillation](#) is still unknown, but the spread of periodontal bacteria through the bloodstream may connect these conditions," said study first author Shunsuke Miyauchi, assistant professor at HU's Graduate School of Biomedical and Health Sciences.

"Among various periodontal bacteria, *P. gingivalis* is highly pathogenic to periodontitis and some systemic diseases outside the oral cavity. In this study, we have addressed these two key questions: Does *P. gingivalis* translocate to the left atrium from the periodontitis lesion? And if so, does it induce the progression of atrial fibrosis and AFib?"

Examining the gum disease-AFib link

To simulate how *P. gingivalis* might escape the mouth and wreak havoc elsewhere, researchers created a [mouse model](#) using the bacterium's aggressive W83 strain. They divided 13-week-old male mice into two groups: one had the strain introduced into the tooth pulp, the other remained uninfected. Each was further split into subgroups and observed for either 12 or 18 weeks to track the cardiovascular risks of prolonged exposure.

Intracardiac stimulation—a diagnostic technique for arrhythmia—revealed no difference in AFib risk between infected and uninfected mice at 12 weeks. But by week 18, tests showed that mice exposed to the bacterium were six times more likely to develop abnormal heart rhythms, with a 30% AFib inducibility rate compared to just 5% in the control group.

To see if their model accurately replicated periodontitis, the researchers examined jaw lesions and found its telltale signs. They detected tooth pulp decay and microabscesses caused by *P. gingivalis*. But the damage did not stop there. They also spotted the bacterium in the heart's left atrium, where infected tissue had turned stiff and fibrous.

Using loop-mediated isothermal amplification to detect specific genetic signatures, the team confirmed that the *P. gingivalis* strain they had introduced was present in the heart. In contrast, the uninfected mice had healthy teeth and no trace of the bacterium in heart tissue samples.

Twelve weeks after infection, mice exposed to *P. gingivalis* already showed more heart scarring than their uninfected counterparts. At 18 weeks, scarring in the infected mice had climbed to 21.9% compared to the likely aging-related 16.3% in the control group, suggesting that *P. gingivalis* may not just trigger early heart damage, but also speed it up over time.

And this troubling connection was not only seen in mice. In a separate human study, researchers analyzed left atrial tissue from 68 AFib patients who underwent heart surgery. *P. gingivalis* was found there, too, and in greater amounts in people with severe gum disease.

Master of stealth assault

Past studies have shown that *P. gingivalis* can invade host cells and evade destruction by autophagosomes, the cellular garbage crew. This ability to hide inside cells suggests a way by which it can slip past immune defenses and trigger just enough inflammation to cause harm without being flushed out. Infected mice showed a spike in galectin-3, a biomarker for fibrosis, and higher expression of *Tgfb1*, a gene tied to inflammation and scarring.

The findings suggest that brushing, flossing, and regular dental checkups might do more than promote oral hygiene, they could also help protect the heart. Keeping gums healthy could block the gateway for a *P. gingivalis* invasion.

"*P. gingivalis* invades the circulatory system via the periodontal lesions and further translocates to the left atrium, where its bacterial load correlates with the clinical severity of periodontitis. Once in the atrium, it exacerbates atrial fibrosis, which results in higher AFib inducibility," Miyauchi said. "Therefore, periodontal treatment, which can block the gateway of *P. gingivalis* translocation, may play an important role in AFib prevention and treatment."

The team is now working to strengthen interdisciplinary collaboration between medical and dental professionals in Hiroshima Prefecture to improve cardiovascular care.

"For the next step, we're investigating the specific mechanisms by which *P. gingivalis* affects atrial cardiomyocytes," Miyauchi said.

"We're also now focusing on establishing a collaborative medical and dental system in Hiroshima Prefecture to treat cardiovascular diseases, including atrial fibrillation. We aim to expand this initiative nationwide in the future."

More information: Shunsuke Miyauchi et al, Atrial Translocation of *Porphyromonas gingivalis* Exacerbates Atrial Fibrosis and Atrial Fibrillation, *Circulation* (2025). DOI: [10.1161/CIRCULATIONAHA.124.071310](https://doi.org/10.1161/CIRCULATIONAHA.124.071310)

Journal information: [Circulation](#)
Provided by [Hiroshima University](#)

Antibiotic use in infancy linked to earlier puberty onset in girls

by European Society of Endocrinology

Girls given antibiotics during their first year of life, especially in the first three months, are more likely to enter puberty at an earlier age, according to research presented at the first [Joint Congress](#) between the European Society of Pediatric Endocrinology (ESPE) and the European Society of Endocrinology (ESE). The likelihood of early puberty was also higher among those exposed to a greater variety of antibiotic classes. The findings highlight the importance of using these medicines in infants appropriately and how early-life drug exposure could shape future health outcomes.

Early puberty, also known as central precocious puberty (CPP), is a condition characterized by the early onset of secondary sexual development in children. In girls this means before age 8 and, in boys, before age 9. Early puberty mostly affects girls and often has no obvious cause, while it is less common in boys. Over the past few decades, [early puberty](#) has been on the rise and researchers are continuously investigating the potential contributing factors.

In this study, researchers from Hanyang University Guri Hospital and Hanyang University Medical Center analyzed data on the antibiotic intake of 322,731 children, aged 0–12 months, in South Korea. They followed these children until the girls turned 9 years old and the boys were 10, and found that girls who were prescribed antibiotics before 3 months of age were 33% more likely to start puberty early. The risk was 40% higher in girls who received antibiotics before 14 days of age, and overall, the earlier the antibiotic exposure, the greater the risk of early puberty. Additionally, [girls](#) who used five or more classes of antibiotics had a 22% increased risk of early puberty compared to those who used two or fewer classes. No association was found between antibiotic intake and early puberty in boys.

"This population-based study is one of the first to explore this association between early-life [antibiotic use](#), including the timing, frequency and number of classes, and in such a large national cohort of children," said Dr. Yunsoo Choe at Hanyang University Guri Hospital in South Korea, who was involved in the study.

Dr. Choe added, "In our earlier research, we found that exclusive breastfeeding was associated with a lower risk of central precocious puberty (CPP), supporting the idea that early-life factors affecting the gut microbiome or endocrine-metabolic pathways may influence pubertal development. The current study builds on this by examining antibiotic exposure as a possible modifier of pubertal timing, using a much larger population and more detailed exposure data."

"Our results may encourage doctors and parents to consider the long-term effects of antibiotics when making treatment decisions for [young children](#)," said Dr. Choe.

Next, the researchers will explore how early antibiotic use may affect [pubertal development](#) and whether repeated or long-term intake during childhood is associated with other aspects of growth, metabolism or endocrine health. "Our study adds to growing concerns about how antibiotics given in infancy could affect children's long-term development—possibly by altering the [gut microbiome](#) or hormonal balance—but the reasons behind it are still unclear," said Dr. Choe.

"Understanding these biological mechanisms may help guide safer antibiotic use and inform early-life care strategies for children," she concluded.

Provided by European Society of Endocrinology

MAY 13, 2025

Is matcha a healthier alternative to coffee?

by Anthony Booker, [The Conversation](#)



Credit: Pixabay/CC0 Public Domain

Matcha, with its vibrant green hue and [centuries-old tradition](#), is often celebrated as a health-boosting superfood. But what exactly sets it apart from regular green tea, or even your morning coffee?

Like green and black tea, matcha comes from [the camellia sinensis plant](#). The difference lies in how it's grown and processed. While [black tea](#) is fermented and regular green tea is simply dried, matcha is shade-grown for several weeks before harvest.

This unique method [alters the plant's chemistry](#), boosting certain compounds like chlorophyll and [amino acids](#) and giving matcha its distinct flavor and rich green color. The leaves are then dried and finely ground into a powder—hence its name, which literally translates to "powdered tea" in Japanese.

Although widely associated with Japanese culture and Zen tea ceremonies, matcha actually [originated in China](#). It was brought to Japan in the 12th century by Buddhist monks, who used it to support meditation. Over time, it became a staple in Japanese tea culture, especially in formal tea ceremonies.

From a health perspective, matcha offers [many of the same benefits](#) as green tea—thanks to its [high content of polyphenols](#), including flavonoids, which are known antioxidants. However, because the leaves are consumed whole in powdered form, matcha may provide a [more concentrated dose](#) of these beneficial compounds.

Lots of potential, relatively little research

Matcha is touted for its wide [range of potential health benefits](#): antioxidant, antimicrobial, anti-inflammatory, [anti-obesity](#) and [even anti-cancer effects](#), as well as potential [improvements in brain function](#), stress relief, heart health and blood sugar regulation.

But there's a catch: [most of the evidence](#) supporting these claims comes from [lab studies](#) (on cells or animals), not robust clinical trials in humans. So while the early research is promising, it's far from conclusive.

One thing we do know: [matcha contains caffeine](#)—more than regular green tea, though typically less than coffee. Caffeine itself has [well-documented health benefits](#) when consumed in moderation, including improved focus, mood, metabolism and even reduced risk of certain diseases like Alzheimer's and Parkinson's.

But [high doses can cause side effects](#) like insomnia, anxiety and elevated blood pressure. The "more is better" approach doesn't apply here, and the optimal dose of caffeine [remains unclear](#).

When comparing matcha to coffee, both offer similar antioxidant properties and [cardiovascular benefits](#). However, coffee has been [studied more extensively](#), with clearer guidelines: [three to four cups a day](#) appears to be a safe upper limit for most people.

For matcha, the guidance is slightly more conservative, with sources suggesting [one to three cups a day](#), probably due to the higher levels of polyphenols.

Tannins and polyphenols in both tea and coffee [can interfere with](#) iron absorption, especially from plant-based foods. Drinking large amounts regularly, [particularly around mealtimes](#), may increase the risk of iron-deficiency anemia.

That's why it's recommended to enjoy these beverages at least two hours before or after meals, especially for people who follow a predominantly plant-based diet or are already prone to low iron levels.

Jitter-free

Another consideration: both coffee and matcha are mildly acidic and can [cause digestive discomfort or reflux](#) in people with sensitive stomachs. That said, matcha may be a better choice for some. Unlike coffee, it [contains L-theanine](#), an amino acid that promotes relaxation [and may counteract](#) the jittery effects of caffeine, making it a gentler alternative for people prone to anxiety.

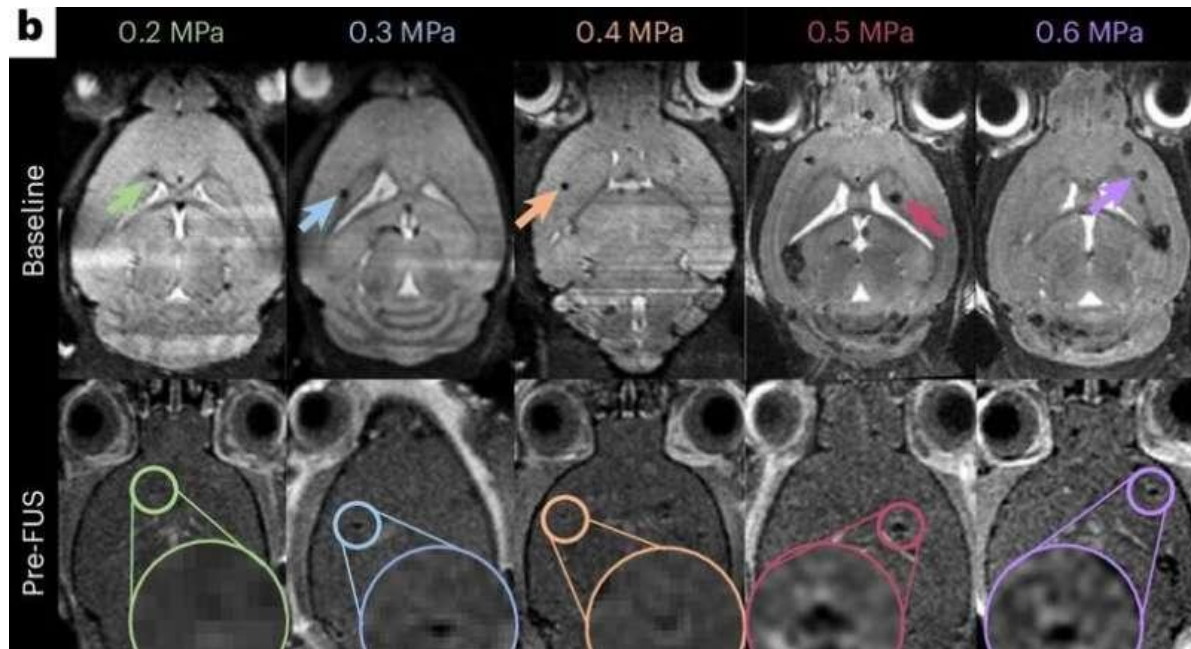
Both matcha and [coffee](#) have potential health benefits and the right choice depends on your personal needs and preferences. Coffee is better studied and may be ideal for those who tolerate caffeine well and enjoy several cups a day. Matcha, on the other hand, is a great option for those looking to consume less caffeine while still benefiting from antioxidants—and without the crash or jitters.

Just remember to enjoy either in moderation, especially if you're managing iron levels or digestive issues.

MAY 15, 2025

Focused ultrasound halts growth of debilitating brain lesions in early testing

by [University of Virginia](#)



FUS-MB treatment opens the BBB within the CCM microenvironment. Credit: *Nature Biomedical Engineering* (2025). DOI: 10.1038/s41551-025-01390-z

A new, incision-free technique developed at UVA Health to treat debilitating brain lesions called cerebral cavernous malformations, or cavernomas, has shown great promise in early testing, halting the growth of the lesions almost entirely.

The work is [published](#) in the journal *Nature Biomedical Engineering*.

The new approach could represent a paradigm shift in how the malformations, commonly called CCMs, are treated, the researchers say. The technique uses tiny, gas-filled "microbubbles" propelled by focused sound waves to open the brain's protective barrier and stunt the growth of the malformations.

"This is a clear example of serendipity in science. We were looking for something else—performing long-term safety studies of focused [ultrasound](#) as a tool for drug and gene delivery to CCMs—when we noticed that CCMs exposed to just focused ultrasound with microbubbles were being stabilized. After the initial observations, we spent years doing experiments to confirm the effect was real and reproducible," said researcher Richard J.

Price, Ph.D., co-director of UVA Health's Focused Ultrasound Cancer Immunotherapy Center.

"Because the focused ultrasound treatment is relatively simple and noninvasive and the necessary clinical devices are becoming more common, if proven safe in [clinical trials](#), I am hopeful it could eventually become a real treatment option."

Cavernomas are clusters of overgrown blood vessels that can sprout like weeds in the brain, spinal cord or other parts of the body. Most cases cause no symptoms, but they can, in some instances, cause headaches, seizures, muscle weakness and even death.

Treatment options for patients include brain surgery, often used when the CCM is at risk of causing a dangerous brain bleed, or [stereotactic radiosurgery](#), which uses radiation to destroy CCMs that are difficult or impossible for a surgeon to reach.

UVA's new approach could offer an alternative that avoids unwanted side effects associated with brain surgery and stereotactic radiosurgery, Price says. For example, traditional [brain surgery](#) comes with the risks of the surgery itself and also the possibility that the removed cavernomas could regrow.

Price and his collaborators were shocked at how well their microbubble treatment performed in lab tests. One month after treatment, the approach had halted the growth of 94% of CCMs in lab mice. During this same time, untreated CCMs grew seven-fold.

"One thing that really stands out is the magnitude of the effect. The mouse models of CCM are much more severe than human CCMs. Mouse CCMs grow exponentially. Yet despite their aggressive nature, CCMs in mice still respond completely to treatment," said Price, of UVA's Department of Biomedical Engineering.

"In some studies, we even saw that brain tissue exposed to focused ultrasound with microbubbles was less inclined to harbor new CCMs in the future. If translated to humans, this prophylactic effect could open the door to treatments for so-called 'familial' patients who are genetically predisposed to acquiring multiple new CCMs throughout their lifespan."

Further, simulated treatment plans for patients with CCMs (patients who have received stereotactic radiosurgery) revealed that the approach is already viable with existing technology, though clinical trials will be needed before the federal Food and Drug Administration would consider making it available for patients.

One notable aspect of the approach is that it doesn't involve the use of any drugs. Scientists at UVA and elsewhere have been exploring the potential of focused ultrasound to briefly breach the [blood-brain barrier](#)—the brain's natural defenses—to allow the targeted delivery of medications for Alzheimer's and other conditions.

But in both Alzheimer's and now cavernomas, the use of the sound-propelled microbubbles appears to have dramatic benefits even without drugs—benefits scientists can't fully explain.

The promising Alzheimer's results have already led to the launch of several clinical trials testing the approach in patients. Price hopes UVA's pioneering research will prompt the launch of similar trials soon for CCMs.

"We are very interested in understanding what is in the 'black box' that somehow connects focused ultrasound to the cessation of mutant cell expansion in the CCMs. We are also returning to our original ideas about drug and gene delivery to CCMs. Since the baseline effect stabilizes the lesions, perhaps we can now think of eradicating them entirely with additional therapies," Price said.

More information: Delaney G. Fisher et al, Focused ultrasound-microbubble treatment arrests the growth and formation of cerebral cavernous malformations, *Nature Biomedical Engineering* (2025). DOI: [10.1038/s41551-025-01390-z](https://doi.org/10.1038/s41551-025-01390-z)

Journal information: [Nature Biomedical Engineering](#)

Provided by [University of Virginia](#)

MAY 16, 2025

New genetic factor identified in worsening of MASLD, with vitamin B3 as the most effective therapeutic agent

by JooHyeon Heo, [Ulsan National Institute of Science and Technology](#)

Approximately 30% of the global population is affected by metabolic-associated fatty liver disease (MASLD), a condition that previously lacked targeted treatments. Researchers have now identified a genetic factor that exacerbates the disease, and remarkably, the FDA-approved drug that most effectively targets this factor is vitamin B3.

A collaborative research team led by Professor Jang Hyun Choi from the Department of Life Sciences at UNIST, in partnership with Professor Hwayoung Yun from the College of Pharmacy and Research Institute for Drug Development at Pusan National University (PNU), and Professor Neung Hwa Park from Ulsan University Hospital (UUH), has elucidated the role of microRNA-93 (miR-93), which is expressed in the liver, as a key genetic regulator in the development and progression of MASLD.

The paper is [published](#) in the journal *Metabolism*.

MiR-93 is a specialized RNA molecule expressed in hepatocytes that functions to suppress the expression of specific target genes. The team observed abnormally elevated levels of miR-93 in both patients with [fatty liver disease](#) and animal models. Through molecular analysis, they demonstrated that miR-93 promotes [lipid accumulation](#), inflammation, and fibrosis by inhibiting the expression of SIRT1, a gene involved in lipid metabolism within liver cells.

In experiments using gene-editing techniques to eliminate miR-93 production in mice, researchers observed a marked reduction in hepatic fat accumulation, along with significant improvements in insulin sensitivity and liver function indicators. Conversely, mice with overexpressed miR-93 exhibited worsened hepatic metabolic function.

Furthermore, screening of 150 FDA-approved drugs revealed that niacin (vitamin B3) most effectively suppresses miR-93. Mice treated with niacin showed a significant decrease in hepatic miR-93 levels and a notable increase in SIRT1 activity. The activated SIRT1 restored disrupted [lipid metabolism](#) pathways, thereby normalizing liver lipid homeostasis.

The research team explained, "This study precisely elucidates the molecular origin of MASLD and demonstrates the potential for repurposing an already approved vitamin compound to modulate this pathway, which has high translational clinical relevance. Given that niacin is a well-established and safe medication used to treat hyperlipidemia, it holds promise as a candidate for combination therapies targeting miRNA pathways in MASLD."

More information: Yo Han Lee et al, Hepatic miR-93 promotes the pathogenesis of metabolic dysfunction-associated steatotic liver disease by suppressing SIRT1, *Metabolism* (2025). DOI: [10.1016/j.metabol.2025.156266](https://doi.org/10.1016/j.metabol.2025.156266)

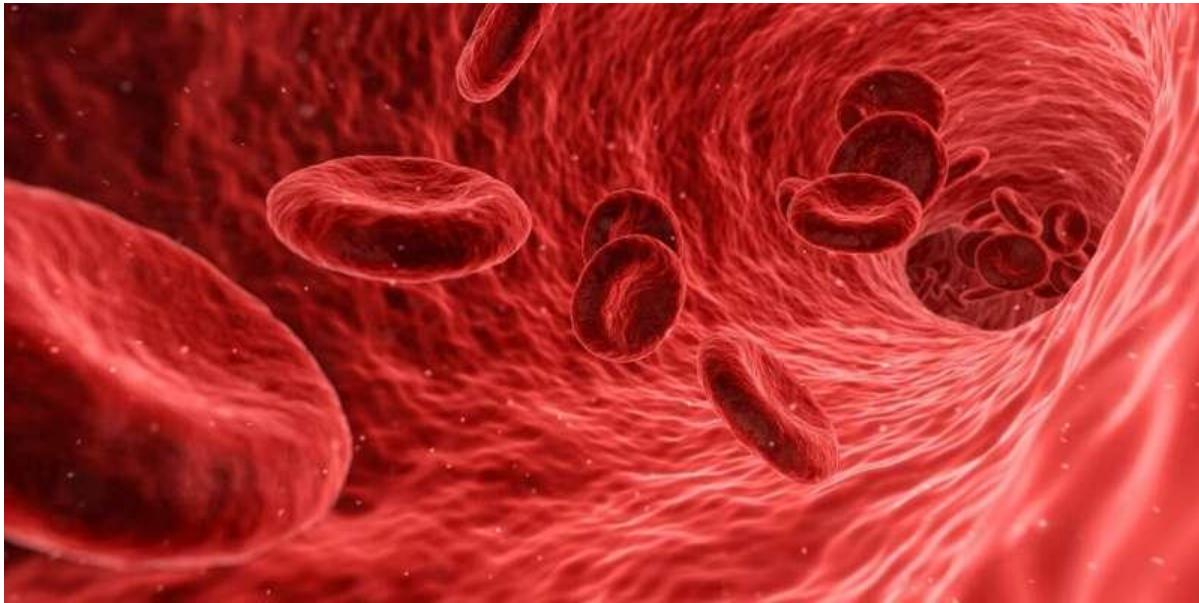
Journal information: [Metabolism](#)

Provided by [Ulsan National Institute of Science and Technology](#)

MAY 16, 2025

Iron deficiency anemia found to significantly increase ischemic stroke risk in young adults

by Ryan Livingston, [University of Cincinnati](#)



Credit: Pixabay/CC0 Public Domain

Findings from a recent study suggest iron deficiency anemia (IDA) independently and significantly increases the odds of ischemic stroke in young adults. The research is [published](#) in the journal *eJHaem*.

IDA is the most common form of anemia globally. It results from iron deficiency, when there are insufficient iron stores to support the production of red blood cells. IDA disproportionately affects women due to regular iron losses through menstruation and increased iron demands during pregnancy. Maternal anemia can lead to the child exhibiting [intellectual disability](#), [autism spectrum disorder](#), and attention-deficit/hyperactivity disorder.

Additionally, it is currently estimated that 10%–15% of all strokes occur in adults aged 18–50, a group that also exhibits an increased prevalence of IDA. However, little data has been collected regarding the association between IDA and stroke in [young adults](#).

"Although multiple studies have explored the association of IDA and thrombotic events in children, our study was the first to explore its role in ischemic stroke in a young adult

population, that is, most susceptible to IDA," wrote Jahnavi Gollamudi, MD, University of Cincinnati, and colleagues.

Investigators classified patients into two groups: those with a first-time ischemic stroke diagnosis were defined as Group A, and those without a history of ischemic stroke were defined as Group B.

Investigators queried an aggregate of health record data from more than 300 hospitals across the U.S., collecting information on patients 15–50 years of age with and without a history of ischemic stroke.

Patients with known diagnosis of thrombocytosis, malignancy, [atrial fibrillation](#), [antiphospholipid syndrome](#), previous history of stroke, pregnancy, hemoglobinopathies including [sickle cell trait](#), use of combined [oral contraceptives](#), thrombophilia, obesity, and use of anticoagulants one year prior to onset of stroke were excluded.

A total of 21,802,239 patients were initially identified; 36,989 were assigned to Group A and 21,765,250 were assigned to Group B. In Group A, 1880 (5.1%) had a diagnosis of antecedent IDA. In Group B, 241,110 participants had a first-time diagnosis of IDA (1.10%).

Investigators noted Group A exhibited greater proportions of hypertension, diabetes, dyslipidemia, smoking, and obesity compared to Group B. Those with antecedent IDA exhibited a substantially greater risk of stroke after multivariable analysis. IDA was associated with 39% increased odds of stroke ($P < .001$) independently of all other described risk factors.

A significant statistical interaction was detected between IDA, age, and non-Caucasian race on ischemic stroke ($P < .05$). Based on this association, investigators determined African American adults 20–39 years of age had a greater likelihood of ischemic stroke than Caucasian adults aged 40–50 years with IDA.

Additionally, the team makes note of possible external factors, such as access to a nutritional diet and fewer regular health care visits, that could lead to increased atherosclerotic risk factors and IDA.

Given the size and scope of the database utilized in the study, investigators also indicated that a general concept of IDA prevalence can be estimated. The overall population in the study exhibited a 1.11% prevalence, compared to the previously reported 5%, which the team attributes to underdiagnosis and underreporting of IDA outside of a laboratory setting.

"In summary, we demonstrate an association between antecedent IDA and [ischemic stroke](#) in young adults," wrote Gollamudi and colleagues. "Given the high prevalence of IDA in this population, an effective screening strategy is urgently needed to diminish its subsequent complications."

More information: Jahnavi Gollamudi et al, Iron Deficiency Anemia and Ischemic Stroke in Young Adults, *eJHaem* (2025). DOI: [10.1002/jha2.70008](https://doi.org/10.1002/jha2.70008)
Provided by [University of Cincinnati](#)

MAY 19, 2025

Inhaled microplastics inhibit key immune cell in the lungs, study finds

by [American Thoracic Society](#)

edited by [Gaby Clark](#), reviewed by [Andrew Zinin](#)

Credit: Alfo Medeiros from Pexels

Microplastics are ubiquitous in the environment, and we all routinely inhale these tiny pollutants. Now, new research published at the [ATS 2025 International Conference](#) has found that inhaled microplastics suppress pulmonary macrophages, a type of white blood cell found in the lungs that are critical to the immune system.

The findings shed new light on the mechanisms through which microplastic exposure leads to long-term disruption of immune function and increases risk of cancer and other diseases. These risks aren't limited to the lungs but can affect the entire body.

"For me, it's a bit eye opening that although microplastics aren't the most dangerous agent we may encounter, they are far from benign," said Adam Soloff, Ph.D., associate professor of cardiothoracic surgery at the University of Pittsburgh and first author of the study.

"Respiratory microplastics disseminate systemically after passing through the lung and negatively affect macrophage function. Overall, these may have detrimental effects on any and all organ systems and contribute to a number of diseases."

Macrophages are the most abundant immune cell of the lung and play a critical role. These cells work as immune housekeepers, devouring pathogens and maintaining tissue homeostasis. They also clear away dead lung cells, which can cause chronic inflammation when they're left to accumulate.

For the study, researchers cultured macrophages with polystyrene microplastics at different sizes and concentrations. They also exposed mice to inhaled microplastics and then measured the effects on the animals' macrophage function.

They found that within 24 hours of exposure to microplastics of any size, the macrophages showed a reduced ability to surround and absorb other bacteria, a critical process called

phagocytosis. Microplastic particles were readily detected in the liver, spleen and colon with trace amounts in the brain and kidney for up to a week after being inhaled.

Researchers also found that the drug Acadesine, an AMP kinase activator, was able to partially restore macrophage function after microplastic exposures.

Dr. Soloff said the results were surprising.

"When we first started to discuss these microplastic exposures, I was sure that the macrophages would just eat (phagocytose) and digest them (lysosomally process), and that would be the end of it," he said.

"I was really surprised to see that not only did the macrophages struggle to break down the plastics in vitro, but macrophages in the lung retained these particles over time as well."

In addition to driving [public health measures](#) to reduce the use of plastics overall, the findings could support the use of drugs like Acadesine in at-risk populations.

"Given the poor air quality in so many places around the world, you could imagine that developing a low-cost, low-side-effect therapeutic to restore pulmonary macrophage function may be an important tool to combat increasing rates of lung disease," Dr. Soloff said.

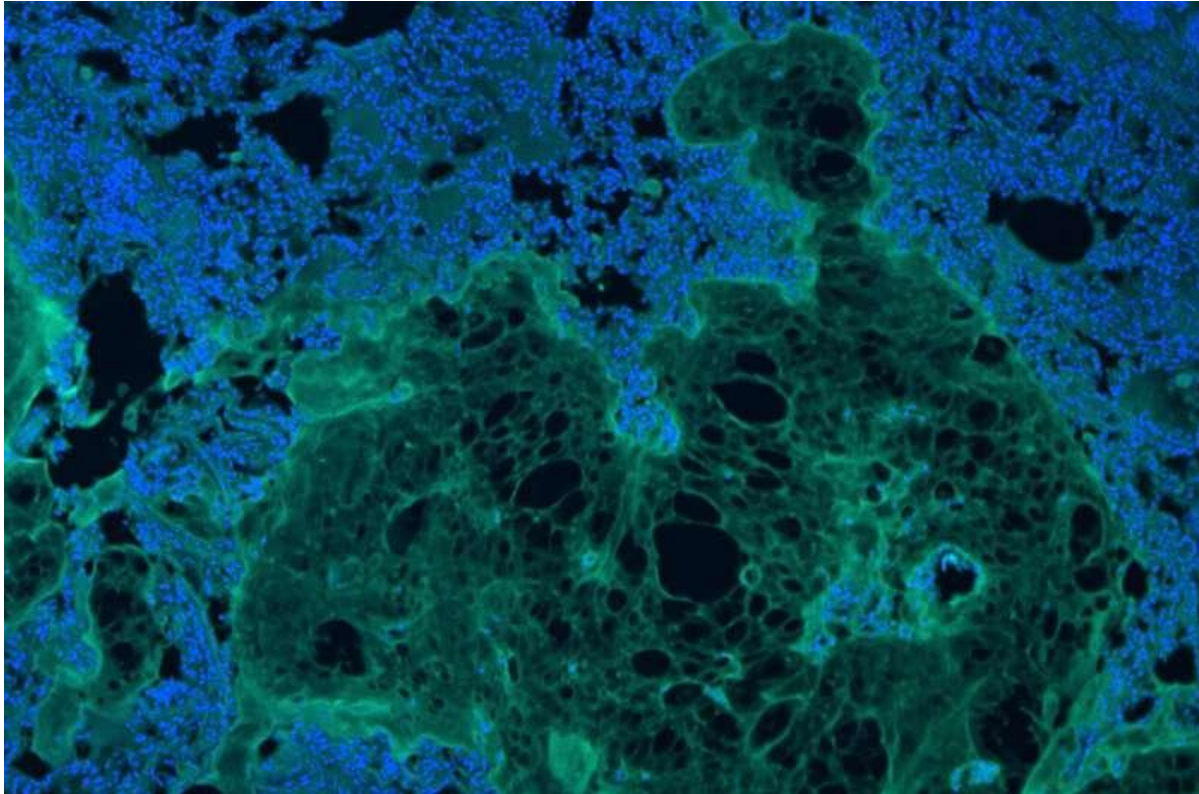
Next, the team will examine [microplastic](#) exposure in lung tissues from patients, with a long-term goal of establishing biomarkers for lung disease and [lung](#) cancer risk that could be used to trigger early screening or intervention.

More information: Inhaled Microplastics Inhibit Tissue Maintenance Functions of Pulmonary Macrophages, [ats2025.d365.events/directory/ ... 44-92e0-bb96b6bdc484](https://ats2025.d365.events.directory/.../44-92e0-bb96b6bdc484)
Provided by [American Thoracic Society](#)

MAY 17, 2025

Better than stitches: Researchers develop biocompatible patch for soft organ injuries

by [Justin Jackson](#) , Medical Xpress



Immunofluorescent staining for macrophages (CD68, green) and nuclei (DAPI, blue) on the hydrogel-tissue interface seven days after artery incisions in rats. Credit: Dr. Mahsa Ghovvati

University of California, Los Angeles and University of California, San Diego researchers developed an injectable sealant for rapid hemostasis and tissue adhesion in soft, elastic organs.

Formulated with methacryloyl-modified human recombinant tropoelastin (MeTro) and Laponite silicate nanoplatelets (SNs), the engineered hydrogel demonstrated substantial improvements in tissue adhesion strength and hemostatic efficacy in preclinical models involving lung and arterial injuries.

Injuries to [soft tissues](#) such as lungs, heart, and [blood vessels](#) complicate surgical closure due to their constant motion and elasticity. Sutures, wires, and staples are mechanically fixed, risking [blood loss](#) when applied to tissues that expand and contract with each breath

or heartbeat. Existing hemostatic agents, including fibrin-based sealants, aim to stem blood flow but may trigger intense coagulation responses in patients with clotting disorders.

Products like cyanoacrylates deliver strong adhesion yet introduce excessive stiffness that can disrupt natural tissue movement and cause cytotoxic effects. Natural polymer hydrogels are biocompatible but often lack the [mechanical strength](#) and adhesion needed for sealing wet or highly elastic surfaces. Progel and poly(ethylene glycol)-based FocalSeal demonstrate limited elasticity and require extended application times, reducing their effectiveness in dynamic tissue environments.

Soft tissue sealants that integrate hemostasis, flexibility, and adhesion remain limited. MeTro previously showed biocompatibility and mechanical properties resembling those of native elastic tissues.

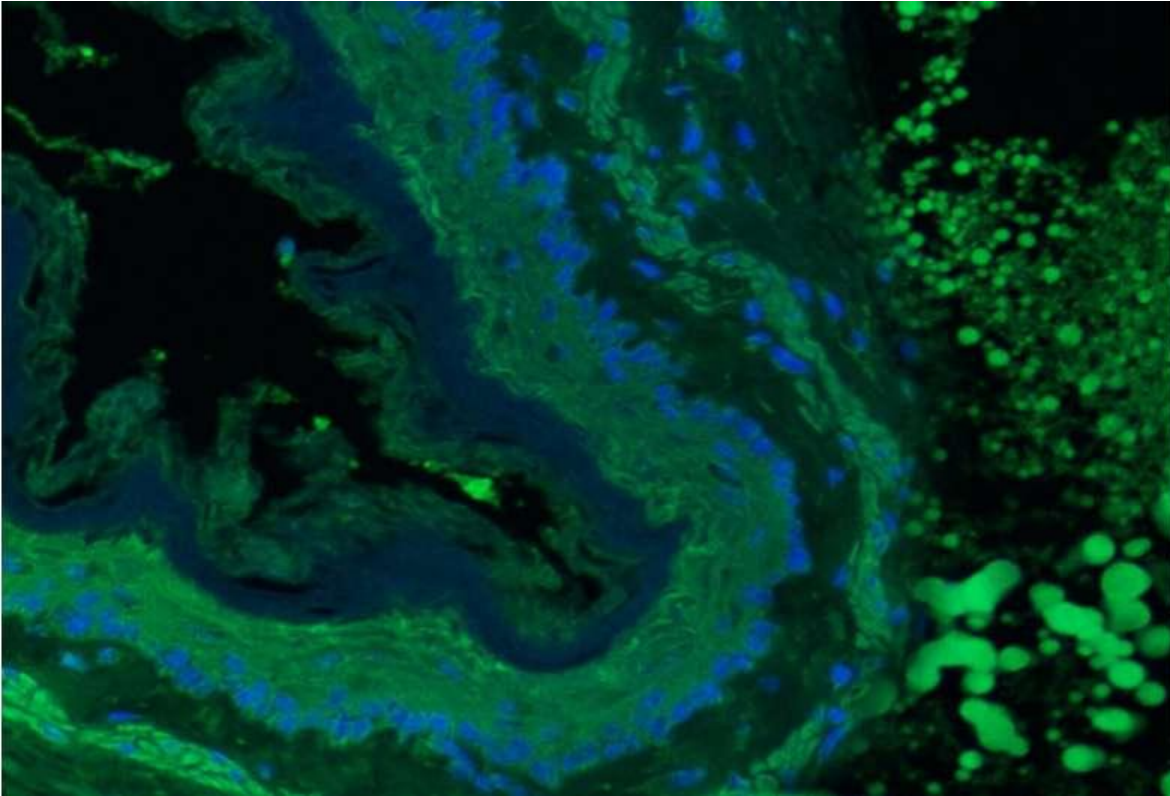
Prior modifications with [graphene oxide](#) increased toughness, while methacryloyl-modified gelatin enhanced nerve repair. Hemostatic capabilities, however, remained a gap. SNs have the potential for improving clotting response while maintaining the sealant's mechanical integrity.

In the study, "Rapid closure and hemostasis of ruptured soft tissues using a modified human tropoelastin-based sealant in preclinical models," [published](#) in *Science Translational Medicine*, researchers conducted preclinical testing of an engineered hydrogel combining MeTro and Laponite SNs to assess tissue adhesion and hemostatic efficacy in soft tissues.

Preclinical testing involved arterial injury models in rat and lung injury in pig subjects to assess the sealant's performance in soft, elastic organs. Researchers measured adhesion strength, clotting time, and tissue response to evaluate the effectiveness of the MeTro/SN formulation under dynamic physiological conditions.

Prepolymer solutions of MeTro and varying concentrations of SNs were prepared and cross-linked using visible light. Rat and pig subjects underwent injury procedures to assess the sealant's efficacy.

Testing involved ex vivo adhesion strength measurements on pig skin, lung, and heart tissues, with burst pressure evaluated on punctured collagen sheets. Hemostatic performance was assessed through clotting time analysis using fresh human blood treated with MeTro/SN hydrogels and commercial hemostatic agents.



Immunofluorescent staining for macrophages (CD68, green) and nuclei (DAPI, blue) on the hydrogel-tissue interface seven days after sealing lung incisions in rats. Credit: Dr. Mahsa Ghovvati

Biocompatibility assessments included live/dead staining and histological analysis of tissue surrounding MeTro/SN hydrogel implants. Additional testing included zeta potential analysis to evaluate electrostatic interactions and [nuclear magnetic resonance spectroscopy](#) to confirm cross-linking and chemical composition of the hydrogel matrix.

MeTro/SN hydrogels exhibited increased adhesion strength and burst pressure compared to MeTro alone and commercial sealants. On pig lung tissue, MeTro/SN achieved an adhesion strength of 23 kPa compared to 12 kPa for MeTro. Burst pressure of MeTro/SN hydrogels containing 1% SN reached 3.6 kPa, exceeding MeTro's 2.8 kPa.

In ex vivo heart burst pressure tests, MeTro/SN tolerated pressures of 47 kPa compared to 41 kPa for MeTro, indicating improved structural integrity. Collagen-sheet burst pressure tests also showed superior mechanical strength in MeTro/SN-treated samples.

Hemostatic testing demonstrated reduced clotting time in MeTro/SN-treated samples compared to untreated controls. Blood treated with MeTro/SN hydrogels containing 1% SN clotted within 12 minutes, while untreated blood required 15 minutes. No increase in hemolysis was observed, indicating minimal cell damage despite the faster clotting response.

In the rat tail amputation model, MeTro/SN hydrogels reduced blood loss by approximately 91% compared to MeTro and by 99% compared to untreated controls.

Histological analysis revealed no significant inflammatory response surrounding MeTro/SN implants after seven and 28 days in rat subcutaneous tissue. No fibrosis or lymphocyte infiltration was observed, indicating biocompatibility of the hydrogel formulation.

In the pig lung injury model, MeTro/SN hydrogels maintained seal integrity and prevented blood loss for 14 days under physiological pressures. No significant detachment or tissue damage was observed during pressure testing, confirming the hydrogel's adhesive stability in dynamic tissue environments.

Researchers conclude that MeTro/SN hydrogels provide effective sealing and hemostasis in soft, elastic tissues while maintaining biocompatibility in [preclinical models](#). Improved adhesion strength and burst pressure on lung and arterial tissues indicate the potential for clinical application in traumatic injuries where conventional sealants fail to adhere under dynamic conditions.

Integration of SNs reduced clotting time without inducing inflammatory responses, supporting the [hydrogel](#)'s potential for rapid intervention in hemorrhagic wounds. Biocompatibility analysis demonstrated minimal immune response and tissue damage after implantation, suggesting a low-risk profile for clinical translation.

More information: Mahsa Ghovvati et al, Rapid closure and hemostasis of ruptured soft tissues using a modified human tropoelastin-based sealant in preclinical models, *Science Translational Medicine* (2025). DOI: [10.1126/scitranslmed.adr6458](https://doi.org/10.1126/scitranslmed.adr6458)

Journal information: [Science Translational Medicine](#)

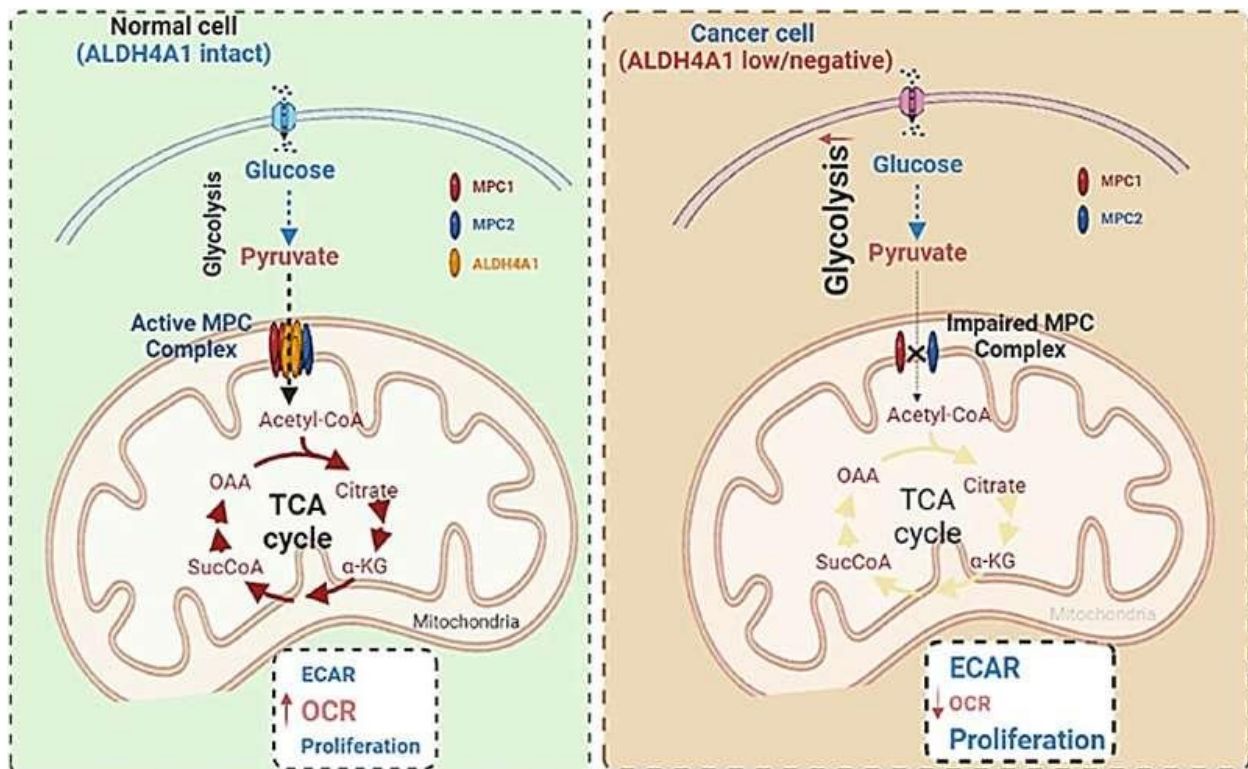
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MAY 19, 2025

A common enzyme takes on a surprising role in preventing cancer

by [Justin Jackson](#) , Medical Xpress

edited by [Sadie Harley](#), reviewed by [Robert Egan](#)



Working model revealing the role of ALDH4A1 in maintaining an active MPC complex for mitochondrial pyruvate import and TCA cycle entry. Credit: *Nature Cell Biology* (2025). DOI: 10.1038/s41556-025-01651-8

Researchers at Duke University Medical Center and Wake Forest University School of Medicine have identified ALDH4A1, a mitochondrial proline-metabolizing enzyme, as a third structural component of the mitochondrial pyruvate carrier (MPC) complex. Forming a trimeric assembly with MPC1 and MPC2, ALDH4A1 maintains MPC integrity and facilitates pyruvate import into mitochondria.

Mitochondrial [pyruvate](#) import serves as a critical step in [cellular energy metabolism](#), linking cytosolic glycolysis to mitochondrial oxidative phosphorylation. Disruptions in this pathway can promote cancer cell proliferation by increasing cytosolic pyruvate and driving glycolytic metabolism, a phenomenon often associated with the Warburg effect.

Understanding how the MPC complex regulates pyruvate import may reveal metabolic vulnerabilities in cancer.

MPC1 and MPC2, the core subunits of the mitochondrial pyruvate carrier complex, serve as the conduit for pyruvate entry into mitochondria. Identification of additional regulatory components had not been observed until now.

In the study, "ALDH4A1 functions as an active component of the MPC complex maintaining mitochondrial pyruvate import for TCA cycle entry and [tumor suppression](#)," [published](#) in *Nature Cell Biology*, researchers employed proteoliposome reconstitution, blue-native PAGE, and co-immunoprecipitation assays to investigate whether ALDH4A1 functions as a structural component of the MPC complex.

Mass spectrometry and co-immunoprecipitation assays identified interactions between ALDH4A1 and mitochondrial pyruvate carrier subunits MPC1 and MPC2. Pyruvate uptake was quantified, [oxygen consumption](#) revealed mitochondrial coupling efficiency.

Western blotting and immunofluorescence confirmed [protein expression](#) and mitochondrial localization. Subcutaneous xenograft models in nude mice evaluated tumor growth under conditions of ALDH4A1 knockdown and overexpression.

Loss of ALDH4A1 promoted in vitro cancer cell phenotypes, including proliferation and migration, and induced soft-agar transformation, indicating anchorage-independent growth.

Xenograft models further demonstrated ALDH4A1's tumor-suppressive function, with ALDH4A1 overexpression restricting tumor volume and knockdown promoting rapid [tumor growth](#).

Treatment with the MPC inhibitor UK5099 disrupted the interaction between ALDH4A1 and the MPC complex while leaving MPC1/2 dimers intact, suggesting potential druggability of the ALDH4A1-MPC interface.

Findings position ALDH4A1 as a structural component of the mitochondrial pyruvate carrier complex, expanding its role beyond proline metabolism to include pyruvate import and TCA cycle entry. By maintaining MPC integrity and pyruvate transport, ALDH4A1 limits cytosolic pyruvate accumulation and mitigates metabolic reprogramming associated with tumorigenesis.

Targeting ALDH4A1's regulatory activity within the MPC complex may hold promise for future metabolic interventions in cancer. Further research is required to explore therapeutic strategies and broader metabolic implications.

More information: Che-Chia Hsu et al, ALDH4A1 functions as an active component of the MPC complex maintaining mitochondrial pyruvate import for TCA cycle entry and tumour suppression, *Nature Cell Biology* (2025). DOI: [10.1038/s41556-025-01651-8](https://doi.org/10.1038/s41556-025-01651-8)

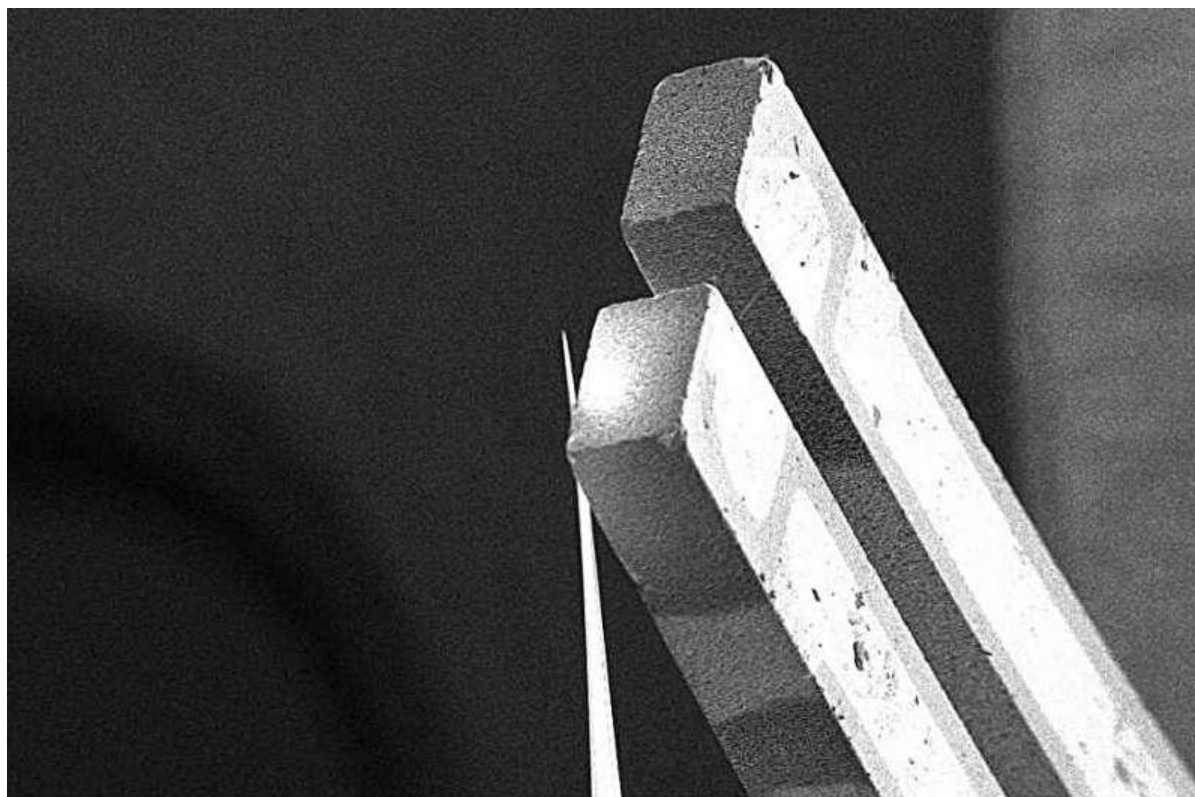
Journal information: [Nature Cell Biology](#)

MAY 19, 2025

MRI gets a nano-sized upgrade

by [Weizmann Institute of Science](#)

edited by [Lisa Lock](#), reviewed by [Robert Egan](#)



The Weizmann nano-MRI system viewed under an electron microscope. Top: A tuning fork that enables control of the distance from the quartz tip to the diamond sensor, thereby controlling the gradient of the magnetic field around the sensor. Scale: 300 nanometers.

Bottom: A closeup of the tip with a quartz center (the darker shade), coated with a gold conductor in the shape of a square arch (the lighter shade). Scale: 500 nanometers.

Credit: *Communications Physics* (2025). DOI: 10.1038/s42005-025-02019-y

Conventional MRI scans, familiar to us from hospitals, have a resolution of about one-tenth of a millimeter, which allows them to image incredibly thin slices of our bodies from head to toe, helping physicians diagnose a variety of medical conditions. Even this ultra-high resolution, however, is insufficient for researchers who want to study the structure of individual molecules.

New technology developed in Dr. Amit Finkler's laboratory at the Weizmann Institute of Science allows researchers to conduct MRI scans at a resolution of one nanometer (one-millionth of a millimeter, or one-billionth of a meter) or higher.

The new nano-MRI device, [described](#) recently in *Communications Physics*, may make it possible to distinguish all the tiny particles that make up a single molecule, generating the most detailed images of individual molecules ever produced. It represents a massive leap forward in the race to develop nano-MRI applications for research and for use in the materials and pharmaceutical industries.

MRI is based on a property of elementary particles within atoms called spin. It's a [magnetic property](#) that can be visualized as a rotation around an axis, similar to a spinning top, and it is characterized by a frequency—the number of "rotations" per second—known as the [resonance frequency](#). This resonance frequency is what the MRI device measures. It depends on the type of particle being measured and the strength of the surrounding magnetic field.

In conventional MRI, a gradient magnetic field, which varies in strength along the patient's body, causes the resonance frequency to vary as well, allowing the machine to differentiate between slices of tissue. A steeper gradient allows for thinner slices. The question that the Weizmann researchers posed was: Could a gradient magnetic field also be used to distinguish the individual particles within a [single molecule](#)?

Scientists in Finkler's laboratory in the Chemical and Biological Physics Department had already developed a method of magnetic resonance scanning based on a [synthetic diamond](#). Inside the diamond is a tiny defect the size of a single atom, called the nitrogen-vacancy center. This defect acts as a sensor that changes the intensity of the red light it emits, depending on the spin of the adjacent particles.

The advantage of the nitrogen-vacancy center is that it is highly sensitive to even the weakest signals—such as, for example, the presence of a single particle located 50 nanometers away. The problem, however, was that it didn't always differentiate effectively between adjacent particles, and the light it emitted was influenced by the average properties of all nearby particles. This meant that it was difficult to use this sensor to image the individual atoms that make up a molecule.

In their new study, led by doctoral candidate Leora Schein-Lubomirsky, the researchers developed a machine that generates a gradient magnetic field focused around an atomic magnetic sensor. This machine is based on a quartz tip coated with a gold conductor in the shape of a square arch. When an electric current was run through the wire, it generated a gradient magnetic field; the changes in the magnetic field were strongest near the corners of the rectangle and gradually weakened further away.

"The changes in the magnetic field led to changes in the resonance frequency of atoms, depending on their position in the molecule," Schein-Lubomirsky explains. "Previously, the sensor was incapable of distinguishing between several nearby hydrogen atoms and determining their locations, but now, within each region, a hydrogen particle will exhibit a different resonance frequency. We will then be able to assemble images showing these different locations into a complete picture of the molecule."

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'Don't look at the field, but at the change within it'

"The realization that led to the new development was that we could produce a very strong gradient of the magnetic field even if its absolute size remained small," Finkler explains. "Even though our magnetic field is significantly smaller than that of a commercial MRI machine, its gradient—the rate at which the magnetic field changes with distance from the device—is much greater.

"This is how we obtained a resolution of one nanometer, and we believe our device is capable of reaching even higher resolutions—meaning it would be capable of scanning the structure of an individual molecule."

The new device is also an improvement over previous diamond-sensor systems, since it can activate and deactivate the magnetic field on demand—and does so within just 0.6 millionths of a second. This is because the field is not produced by a magnet, but rather by means of an [electric current](#) that can be turned on or off. "The ability to quickly activate and deactivate the [magnetic field](#) ensures that there are fewer disruptions and that the scan is more accurate," Finkler adds.

High-resolution molecular imaging plays a crucial role in the materials and pharmaceutical industries. Today, every manufactured drug undergoes magnetic resonance testing to ensure that it contains only the desired substance in the correct molecular structure and arrangement that is safe for human use.

Current methods, however, require large sample quantities, which can be difficult to obtain, especially during early development stages. Additionally, these tests cannot be conducted at room temperature, and their resolution remains limited.

"The nano-MRI machine we are proposing can operate at [room temperature](#) and examine the structure of materials under exactly those conditions at which they are supposed to be used," Finkler emphasizes.

"The machine will also produce a more detailed image of the molecular structure and make it possible to test a small and significantly cheaper sample containing only a few molecules of the material. Moreover, this device could help reveal why substances sometimes behave unexpectedly in the real world, compared to the results of laboratory testing, and whether there are unknown differences between substances that appear to be identical."

More information: Leora Schein-Lubomirsky et al, Pulsed magnetic field gradient on a tip for nanoscale imaging of spins, *Communications Physics* (2025). DOI: [10.1038/s42005-025-02019-y](https://doi.org/10.1038/s42005-025-02019-y)

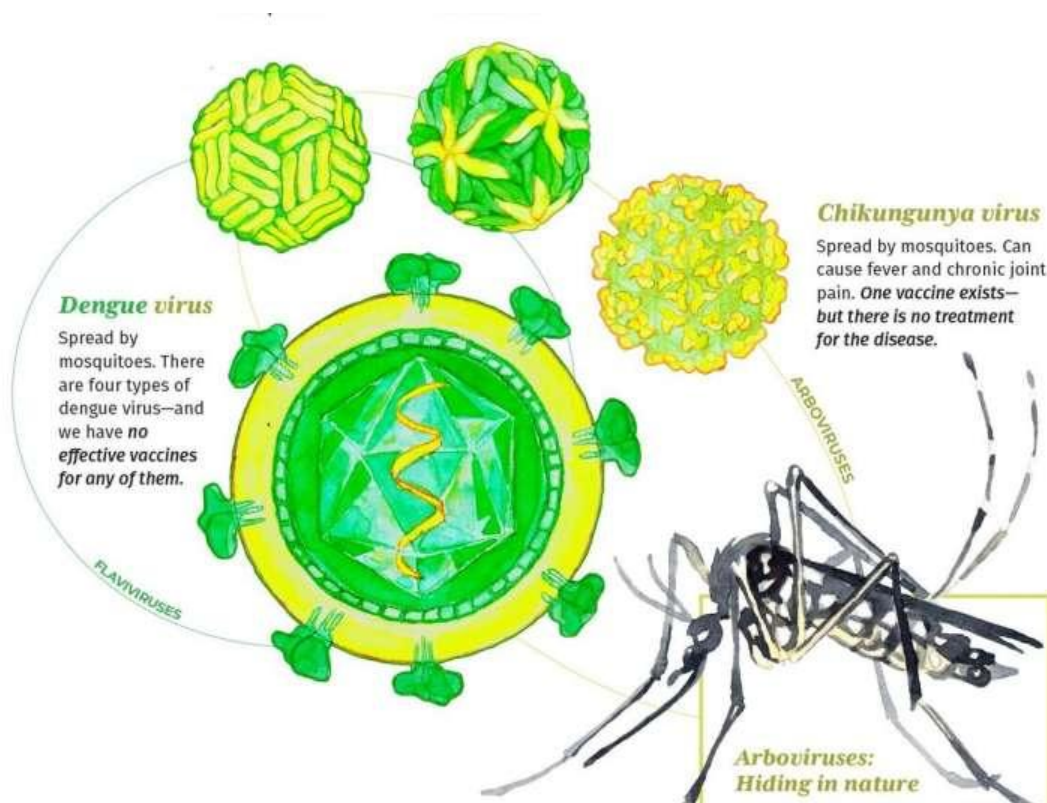
Journal information: [Communications Physics](#)

Provided by [Weizmann Institute of Science](#)

MAY 20, 2025

Scientists uncover key clues to how a viral infection can lead to arthritis-like disease

by [La Jolla Institute for Immunology](#) edited by [Sadie Harley](#), reviewed by [Robert Egan](#)



Chikungunya virus is one of several viruses spread by *Aedes aegypti* and *Aedes albopictus* mosquitos. Like Chikungunya virus, pathogens such as dengue virus and Zika virus continue to spread as climate change leaves more regions warmer and more habitable for these mosquito species. Credit: Patricia Pauchnick for La Jolla Institute for Immunology

Chikungunya virus (CHIKV) is a mosquito-borne pathogen that has been identified in more than 110 countries around the world. The virus typically causes flu-like symptoms, but it can also trigger chronic, severe joint pain in some people.

Researchers at La Jolla Institute for Immunology (LJI) are working to understand how a viral infection can cause persistent joint pain that closely resembles [rheumatoid arthritis](#), an autoimmune disease.

In a new study, LJI scientists share a critical first look at how the body's T cells target CHIKV. Their research suggests CD4⁺ T cells step up to fight the virus—and cause [chronic](#)

inflammation in the process. This discovery may help explain why some people infected with CHIKV develop severe joint pain.

"Autoimmune diseases like rheumatoid arthritis have exactly these parameters," says LJL Assistant Professor Daniela Weiskopf, Ph.D., senior author of the new study and member of LJL's Center for Vaccine Innovation.

The findings, published in *Cell Reports Medicine*, offer new clues to why some viral infections appear to trigger autoimmune disease. The research may also help guide the development of therapies to block harmful inflammation.

Testing immune cells vs. chikungunya virus

Weiskopf and her colleagues studied immune cells found in blood samples from a group of CHIKV patients in Colombia. The researchers tested how immune cells from these patients responded to small molecular chains, called peptides, from Chikungunya virus.

This experiment revealed which types of immune cells take the lead in fighting CHIKV infection. The researchers also captured the first-ever look at which sites on CHIKV, called viral epitopes, drew the strongest immune cell responses.

To their surprise, the researchers found that a type of T cell called CD4⁺ T cells showed a strong response to CHIKV. Although CD4⁺ T cells are a normal part of the body's anti-viral response, they are almost always accompanied by CD8⁺ T cells. Scientists have even nicknamed CD8⁺ T cells "killer" T cells because they play a very active role in fighting infections.

Yet the new study shows that CD4⁺ T cells are most active in fighting CHIKV virus. These same CD4⁺ T cells stay in the body as "memory" T cells after the infection is gone.

The researchers found that 87% of patients had detectable levels of CHIKV-specific memory CD4⁺ T cells in their blood six years after their initial infection. In contrast, only 13% of patients still had CHIKV-specific memory CD8⁺ T cells in their blood after six years.

According to Weiskopf, this kind of CD4⁺ T cell profile is more commonly seen in patients with autoimmune diseases. "I'm an infectious disease researcher, but I could see that this T-cell response looked awfully like what we see in autoimmune disease," says Weiskopf.

Not your typical T cells

This CD4⁺ T cell activity may help explain the association between CHIKV infection and chronic, autoimmune-like disease.

"There are many studies in mice showing that CD4⁺ T cells are pathogenic," adds study co-first author Rimjhim Agarwal, a UC San Diego Graduate Student and member of the Weiskopf Lab. "So we needed to know what CD4⁺ T cells are doing in people with CHIKV."

The researchers took a closer look at exactly how CD4⁺ T cells fought CHIKV infection. Normally, CD4⁺ T cells are "polyfunctional," meaning the cells can churn out many kinds of signaling molecules to help coordinate the body's immune response to a pathogen.

But CHIKV patients who developed severe joint pain had more "monofunctional" CD4⁺ T cells. Even years after initial infection, their T cells primarily produced an inflammatory molecule called TNF-alpha. This molecule helps direct immune cell activity during an infection, but TNF-alpha is not supposed to linger long after a virus has been cleared.

The new study offers evidence that these monofunctional CD4⁺ T cells may be the culprits behind joint pain and chronic inflammation following CHIKV infection. Although there's still a lot to learn, the researchers say future therapies that inhibit TNF-alpha may hold promise for treating arthritis-like symptoms in CHIKV patients.

Next steps for helping patients

The new study raises many big questions. Agarwal is currently working to explain a strange phenomenon—why are women in their 40s much more likely to develop chronic joint pain following CHIKV infection?

Last year, Agarwal won funding to investigate this sex-based difference. Her SPARK project may help shed light on whether CD4⁺ T cells are mistakenly attacking the body's own tissues when responding to CHIKV.

This work comes as more scientists today look at possible connections between viral infections and autoimmune disease. Other viruses, such as the mosquito-borne dengue virus, can also cause severe, chronic joint pain. Many people today are also dealing with "long COVID," chronic and debilitating autoimmune-like inflammation that often follows SARS-CoV-2 infection.

"More and more people are realizing, particularly after seeing the long-term effects of SARS-CoV-2, that viral infection can trigger autoimmune-like disease," says Agarwal. "We still have a lot more questions than answers right now, but we want to understand the relationship between viruses and [autoimmune diseases](#)."

More information: Chikungunya virus-specific CD4⁺ T cells are associated with chronic chikungunya viral arthritic disease in humans, *Cell Reports*

Medicine (2025). [dx.doi.org/10.1016/j.xcrm.2025.102134](https://doi.org/10.1016/j.xcrm.2025.102134) , On *bioRxiv*: DOI: [10.1101/2023.11.20.567688](https://doi.org/10.1101/2023.11.20.567688)

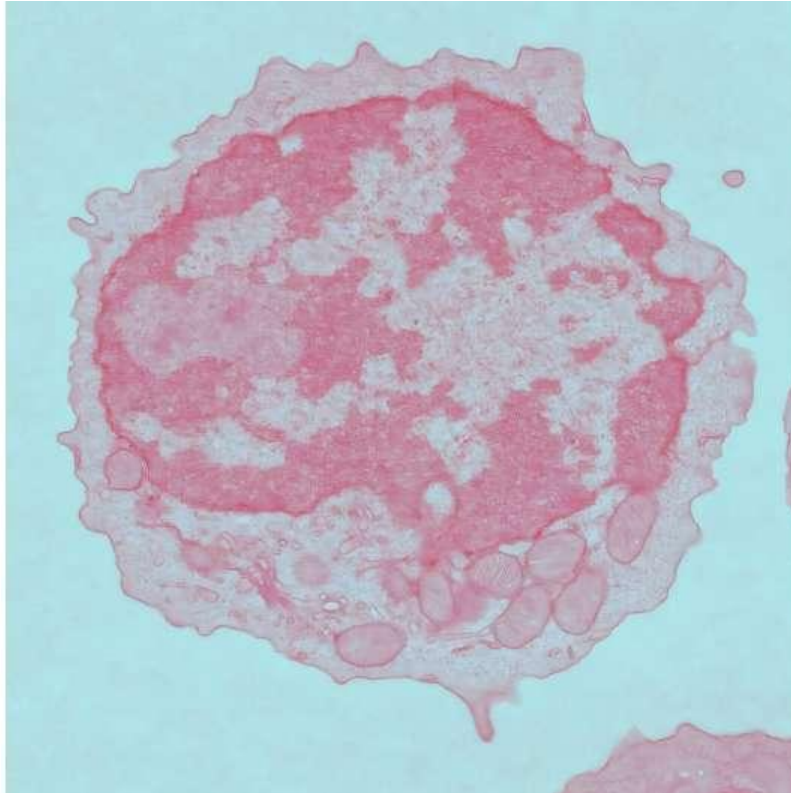
Journal information: [Cell Reports Medicine](#) , [bioRxiv](#)

Provided by [La Jolla Institute for Immunology](#)

Aging reduces CAR-T cell effectiveness by impairing metabolism, study shows

by University of Lausanne

edited by Stephanie Baum, reviewed by Robert Egan



Electron microscopy image of young naïve CD8⁺ T cells. Credit: Helen Hope / UNIL (2025)
As people age, their immune systems become less efficient, posing a challenge for cancer therapies that rely on harnessing immune cells.

In a new study published in *Nature Cancer*, researchers from the University of Lausanne (UNIL), the Lausanne University Hospital (CHUV), the Geneva University Hospitals (HUG) and the Ecole Polytechnique Fédérale de Lausanne (EPFL), show that this age-related immune decline has a measurable impact on CAR-T cell therapy, one of the most advanced forms of cancer immunotherapy.

CAR-T therapy works by engineering a patient's T cells to recognize and destroy cancer cells. But the study found that CAR-T cells from aged mice had poor mitochondrial function, lower "stemness," and reduced antitumor activity. The culprit: a drop in levels of nicotinamide adenine dinucleotide (NAD), a molecule essential for cellular energy and metabolism of mitochondria.

"CAR-T cells from older individuals are metabolically impaired and significantly less effective," said first author Dr. Helen Carrasco Hope. "What's exciting is that we were able to rejuvenate these aged cells by restoring their NAD levels—reviving their antitumor function in [preclinical models](#)."

"Our findings strengthen the growing recognition that aging fundamentally reshapes immune cell function and metabolism," she added. "They highlight the urgent need to model age more accurately in [preclinical studies](#), so that therapies are developed with the real-world cancer population in mind—where most patients are older adults."

The team used NAD-boosting compounds currently under [clinical investigation](#) for other conditions, demonstrating that this approach is translatable and potentially applicable in humans.

"This is a major step toward personalized and age-conscious immunotherapy," said senior author Dr. Nicola Vannini. "By correcting age-related metabolic defects, we could improve outcomes for a large segment of cancer patients."

The study adds to a growing body of work showing that age is not just a chronological number, but a biological factor that can shape therapy response. The authors call for age to be systematically considered in the development and evaluation of cell-based immunotherapies.

More information: Age-associated nicotinamide adenine dinucleotide decline drives CAR-T cell failure, *Nature Cancer* (2025). DOI: [10.1038/s43018-025-00982-7](https://doi.org/10.1038/s43018-025-00982-7)

Journal information: [Nature Cancer](#)
Provided by [University of Lausanne](#)

The silent injuries that shape our skeletons and an overlooked rescue enzyme

by Anik Tuladhar edited by [Sadie Harley](#), reviewed by [Robert Egan](#)

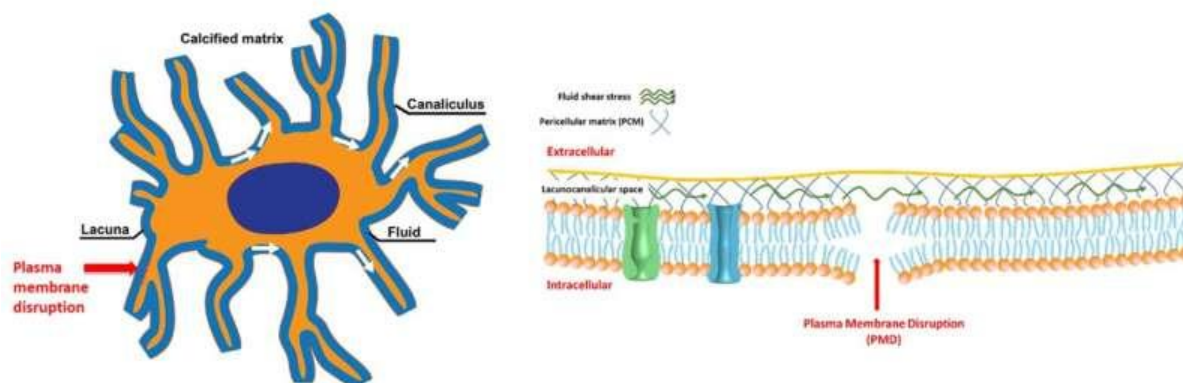


Illustration showing the formation of plasma membrane disruption (PMD) in osteocyte following exposure to fluid shear with mechanical loading.

Illustration showing the formation of plasma membrane disruption (PMD) in osteocyte following exposure to fluid shear with mechanical loading. Credit: Anik Tuladhar, with Biorender

Bone is not the inert structure we often imagine. It's alive, adapting daily to load-bearing and impact. But with over 200 million people affected by osteoporosis globally and therapies relying largely on antiresorptives and aging anabolic agents, the need for new regenerative strategies is critical.

Protein kinase D1 (Prkd1) is a relatively obscure enzyme with a surprising new role: facilitating plasma membrane repair in bone cells. A recent study led by Anik Tuladhar and colleagues at the Medical College of Georgia brings this overlooked protein into the spotlight, implicating it as a key regulator of osteocyte survival and bone mechanoadaptation.

The study is [published](#) in the journal *Bone*.

Osteocytes, the primary mechanosensory cells of bone, live embedded in a matrix they help shape. Their job is to detect strain and signal remodeling, but this signaling starts with micro-injuries. When bone is stressed, the osteocyte's dendritic processes develop plasma membrane disruptions (PMDs), which are transient "wounds" that allow calcium influx and trigger downstream gene expression. But for these cells to survive and adapt, they must quickly reseal these disruptions.

This is where Prkd1 steps in.

Dr. Tuladhar's team demonstrated that inhibiting or genetically deleting Prkd1 slowed membrane repair in osteocytes, increased [cell death](#) after [mechanical loading](#), and dampened bone's anabolic response. In other words, Prkd1 might be a linchpin in translating mechanical strain into healthy bone formation.

Rescuing the rescue enzyme

What makes the story even more compelling is the rescue attempt. The group tested Poloxamer 188, a synthetic membrane-stabilizing agent used in muscular dystrophy studies. They found that Poloxamer restored membrane repair and cell survival in Prkd1-deficient osteocytes but only partially salvaged the bone-building response in animals.

This duality raises key questions: Why does cell-level rescue not always translate to tissue-level repair? Could targeting Prkd1 open the door to new classes of osteoanabolic therapies or are we simply treating a symptom of a deeper signaling breakdown?

Despite its role in multiple tissues, Prkd1 hasn't yet been embraced by skeletal biology or pharmaceutical pipelines. It lacks the household name recognition of Wnt, BMPs, or RANKL. Yet this enzyme checks several critical boxes for therapeutic targeting:

- It's activated by mechanical stimuli
- It's druggable, with known small-molecule inhibitors
- It affects cell viability, calcium signaling, and [gene expression](#)
- It may act selectively in load-responsive [bone cells](#) without altering baseline architecture

In an [aging population](#) facing increased fracture risk, a treatment that enhances bone formation in response to activity rather than blindly increasing turnover has enormous appeal. For biotech, Prkd1 may represent a first-in-class opportunity for mechano-responsive bone therapeutics.

But why now? And why has it been ignored?

Historically, the focus in osteoporosis has been on preventing bone loss rather than enhancing formation. Prkd1's role in plasma membrane resealing might have seemed too downstream, too mechanical, or too niche. But in a post-GLP-1 world where metabolic, mechanical, and inflammatory pathways are increasingly cross-talking, the timing for such a target may finally be right.

Moreover, the rise of wearable health tech and [real-time](#) mechanical load tracking could dovetail with therapies that enhance load-induced bone formation. Imagine a future where [physical therapy](#) is augmented not only with training but with precision dosing of Prkd1-targeted drugs to maximize bone gain.

What's next: From cell to skeleton

The implications are profound, but hurdles remain. More studies are needed to define Prkd1's role across bone cell types. Its downstream targets remain partially mapped. And while Poloxamer 188 is FDA-approved, its bone-specific efficacy needs validation. Could Prkd1 activation synergize with known anabolic agents like PTH or sclerostin antibodies?

Additionally, [sex differences](#) and age-related expression of Prkd1 in human osteocytes remain underexplored. Translating findings from mice to humans will require not only new assays, but also bold clinical trial design.

Final thoughts

In the shadow of better-known bone pathways, Prkd1 has quietly controlled a vital decision point: whether a wounded osteocyte survives or dies. The work by Tuladhar and team cracks open a new window into the cellular logic of bone strength—and invites us to rethink what it means to regenerate from the inside out.

Sometimes, the most powerful breakthroughs don't come from discovering a new protein, but from realizing we misunderstood an old one.

This story is part of [Science X Dialog](#), where researchers can report findings from their published research articles. [Visit this page](#) for information about Science X Dialog and how to participate.

More information: Anik Tuladhar et al, Prkd1 regulates the formation and repair of plasma membrane disruptions (PMD) in osteocytes, *Bone* (2024). DOI: [10.1016/j.bone.2024.117147](https://doi.org/10.1016/j.bone.2024.117147)

Bio: I have a Ph.D. in Cellular Biology and Anatomy from Augusta University. My work spans from membrane level mechanotransduction in bone cells to preclinical safety evaluation in pharmaceutical R&D. I've trained as a researcher, toxicologic scientist, and scientific communicator—currently serving as a postdoctoral fellow at AbbVie. My specialty lies at the intersection of skeletal biology and translational toxicology, where cellular mechanics meet drug-induced liabilities an unclaimed space between regenerative medicine and safety pharmacology.

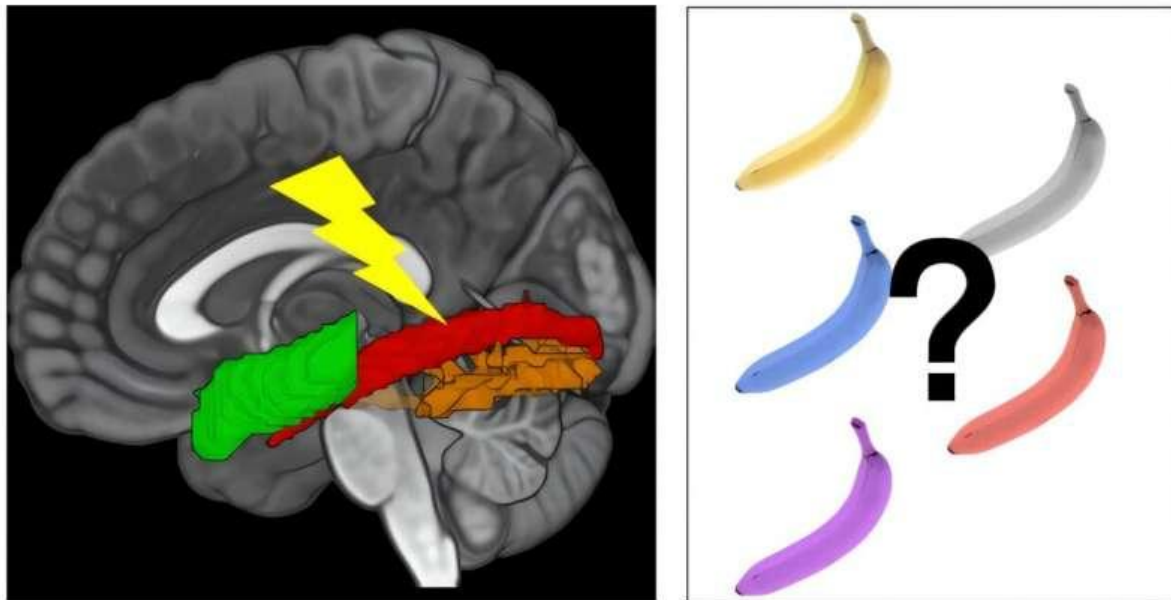
Journal information: [Bone](#)

MAY 20, 2025

A study in stroke patients shows the brain's vision-language connection shapes object knowledge

by [Public Library of Science](#)

edited by [Stephanie Baum](#), reviewed by [Robert Egan](#)



A schematic view of the main findings, adapting from a brain figure in the study.

Credit: *PLOS Biology* DOI: 10.1371/journal.pbio.3003161, CC-BY 4.0

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Our ability to store information about familiar objects depends on the connection between visual and language-processing regions in the brain, according to a study [published](#) in *PLOS Biology* by Bo Liu from Beijing Normal University, China, and colleagues.

Seeing an object and knowing [visual information](#) about it, like its usual [color](#), activate the same parts of the brain. Seeing a yellow banana, for example, and knowing that the object represented by the word "banana" is usually yellow, both excite the ventral occipitotemporal cortex (VOTC).

However, there's evidence that parts of the brain involved in [language](#), like the dorsal anterior temporal lobe (ATL), are also involved in this process—[dementia patients](#) with ATL

damage, for example, struggle with object color knowledge, despite having relatively normal visual processing areas.

To understand whether communication between the brain's language and sensory association systems is necessary for representing information about objects, the authors tested whether stroke-induced damage to the neural pathways connecting these two systems impacted patients' ability to match objects to their typical color.

They compared color-identification behavior in 33 [stroke patients](#) to 35 demographically matched controls, using fMRI to record brain activity and diffusion imaging to map the white matter connections between language regions and the VOTC.

The researchers found that stronger connections between language and visual processing regions correlated with stronger object color representations in the VOTC, and supported better performance on object color knowledge tasks.

These effects couldn't be explained by variations in patients' stroke lesions, related cognitive processes (like simply recognizing a patch of color), or problems with earlier stages of visual processing. The authors suggest that these results highlight the sophisticated connection between vision and language in the human brain.

The authors add, "Our findings reveal that the brain's ability to store and retrieve object perceptual knowledge—like the color of a banana—relies on critical connections between visual and language systems. Damage to these connections disrupts both [brain activity](#) and behavior, showing that language isn't just for communication—it fundamentally shapes how sensory experiences are neurally structured into knowledge."

More information: Bo Liu et al, Object knowledge representation in the human visual cortex requires a connection with the language system, *PLOS Biology* (2025). DOI: [10.1371/journal.pbio.3003161](https://doi.org/10.1371/journal.pbio.3003161)

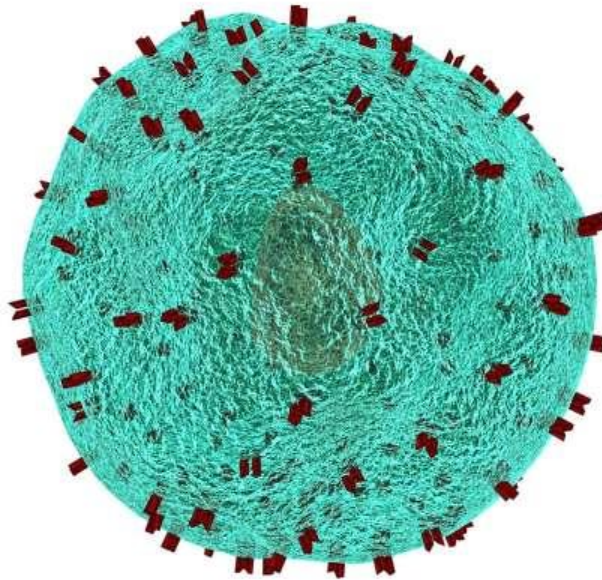
Journal information: [PLOS Biology](#)
Provided by [Public Library of Science](#)

MAY 20, 2025

CAR-T cell therapy is revolutionizing cancer treatment. Here's how it works

by Lydia Begoña Horndler Gil, [The Conversation](#)

edited by [Lisa Lock](#), reviewed by [Andrew Zinin](#)



Credit: CC0 Public Domain

Personalized medicine is already a reality in clinical practice, and [CAR-T cell therapy](#) is one of its most promising tools. This innovative approach, which involves genetically modifying the cells of the immune system, is transforming the way we treat not only cancer, but also other diseases.

The CAR-T revolution is widely documented, with more than 1,000 [clinical trials](#) currently underway worldwide according to the [ClinicalTrials platform](#). This reflects an enormous amount of scientific and medical interest.

To understand why it is of such interest, we first need to know what the function of a T-lymphocyte is. These cells belong to the [immune system](#), and are responsible for finding and eliminating cells that are infected or have become abnormal, such as [cancerous cells](#). We can think of them as the body's police force: they recognize when something is wrong, and help to maintain the balance in our body.

'Latest generation' cells

CAR-T cells are, in essence, T-lymphocytes equipped with a next-generation membrane receptor: a structure specifically designed to recognize and attack tumor cells with a precision and sensitivity far superior to a normal T-cell. The difference is huge, like going from a mid-2000s Nokia phone to the latest iPhone.

Thanks to this modification, CAR-T cells can generate an [immune response](#) up to [1,000 times stronger](#) than an ordinary lymphocyte. This makes it possible to eliminate [cancer cells](#) that do not respond to conventional treatments.

But what exactly is this alteration that proves so lethal to cancer cells? CAR-T cells are designed as "reconnaissance and attack" against cancer, and to do this, they have three main components. There is an external part that acts like a radar, identifying tumor cells; another part that passes through the cell membrane to transmit the signal inwards, and an internal part that triggers the T-cell response, activating its attack.

Although this basic design has already proven very powerful, researchers continue to improve it, both to make it even more effective and to prevent cells from becoming exhausted before they are able to kill off every last tumor cell.

Therapeutic options for many patients have multiplied thanks both to the ability to develop these cells *ex vivo*—in the laboratory—and to advances in gene editing. To date, there are [7 CAR-T therapies](#) which are authorized for use in patients.

Obstacles and limitations

Despite their efficacy, these therapies are not yet available to all patients due to their high cost and associated risks. This underlines the need to improve both their safety and accessibility.

Moreover, although CAR-T has been very successful in treating [leukemias and myelomas](#), its application in solid tumors remains a challenge. In these cases, the tumor microenvironment imposes additional barriers: T cells must pass through dense matrices that hinder their motility. The acidic environment also compromises their functionality, and nutrient shortages can lead to cellular exhaustion.

Such factors have so far limited the success of CAR-T therapies in solid tumors, but researchers continue to explore new ways to overcome these difficulties. One such method involves combining chemotherapy and radiotherapy to enable T-cells to reach tumor cells.

Combining CAR-T with [immune checkpoint inhibitors](#)—drugs that block certain signals used by tumor cells to avoid being attacked by the immune system—could also improve their effectiveness in such hostile environments.

However, it is not always possible to obtain lymphocytes from the patient, either due to limitations in quality or quantity or clinical conditions that make collection difficult. To address this, research is being done into [allogeneic therapies](#) that use "off the shelf" CAR-T cells. These use T-cells from healthy donors, and are stored ready for use when needed.

These methods not only reduce production times and costs, but also expand access to such treatments to a wider range of patients.

The biggest challenge: solid tumors

Although there are still several hurdles to overcome, the main challenge is the expanding the [application of CAR-T cells to other cancers](#), especially solid tumors, which could benefit from these therapies. Examples include glioblastoma and metastatic ovarian cancer.

In the case of glioblastoma (a type of brain tumor), specific targets such as [B7H3](#) or [EGFRvIII](#) are being investigated. These antigens are only expressed on tumor cells, improving the accuracy of these therapies.

Also under study are new delivery modes that [cross the blood-brain barrier](#) (a barrier that regulates the passage of what can and cannot reach the brain) and ways to modulate inflammation, which is critical in treating brain tumors.

For metastatic ovarian cancer, for which there is currently no curative treatment, CAR-Ts offer a promising alternative. Clinical trials have shown encouraging results by targeting certain [new tumor markers](#).

Moreover, CAR-T therapies are not only being applied in oncology. They are also being investigated for use in the treatment of autoimmune diseases, with trials underway for diseases such as [rheumatoid arthritis](#). This opens up new possibilities for the future use of these therapies.

Impact on the pharmaceutical industry

CAR-T's advances, both consolidated and those on the horizon, have led the pharmaceutical industry to boost its investment in these therapies. Currently, the CAR-T cell [therapeutics market](#) is estimated to grow from \$10.39 billion in 2024 to \$128.55 billion in 2032.

The true potential of these therapies is yet to be discovered, and may lie in their effective application against solid tumors. However, one thing is for certain: what until recently seemed a futuristic concept is now a reality. Personalized medicine is here to stay, and CAR-T therapies are leading the way.

Provided by [The Conversation](#)

MAY 26, 2025

Brain stimulation combined with cognitive training can curb impulsive eating

by [University of Granada](#) edited by [Gaby Clark](#), reviewed by [Robert Egan](#)



Fieldwork by the researchers in charge of the study Credit: *BMC Psychology* (2025). DOI: 10.1186/s40359-025-02556-9

Scientists at the Mind, Brain, and Behavior Research Center at the University of Granada (CIMCYC) have revealed how, when faced with highly appetizing and unhealthy food stimuli, many overweight people or those prone to binge eating keep their impulsive system hyperactive, while their reflective or conscious thinking system takes a back seat.

In response, researchers have combined [transcranial magnetic stimulation](#) (TMS) and inhibitory control training for the first time internationally. These techniques prepare the

brain to improve food-related outcomes by increasing the ability to make healthy decisions consciously. TMS has been tested in two projects.

"In our current research on neuroscientific intervention techniques, we are analyzing how, when applied alongside traditional guidelines (based on healthy eating and exercise), changes occur in the mechanisms that hinder the long-term maintenance of improvements in health habits in many people who are overweight or binge eat," explains Alfonso Caracuel, a researcher at CIMCYC working on these studies.

The CIMCYC's work has used the FoodTrainer mobile app, designed specifically to train inhibitory control in the context of eating through interactive exercises. This type of training has the potential to modify automatic behavior patterns and contribute to the adoption of healthier eating habits, helping people make decisions aligned with their wellness goals.

The findings are [published](#) in the journal *BMC Psychology*.

Identification of behavior

The research team describes brain functioning characterized by stimuli that lead to an urgent need to eat unhealthy foods. During decision-making about [food intake](#), the person's longer-term goals, such as maintaining a healthy diet, are relegated to a secondary level, according to the research.

At the same time, studies have been conducted on the functioning of the cognitive components that influence eating. Scientists have discovered differences in some cognitive and behavioral skills used by people with obesity. For example, a greater bias or tendency for attention to be captured by foods high in sugar and fat, as opposed to healthier foods, has been found.

This behavior is related to the activation of the so-called "reward circuit." This phenomenon can lead to decisions being made based on the hyperactivation of the reward system rather than on healthy goals when faced with food-related stimuli or feelings of hunger. In short, according to the results of brain imaging and cognitive performance tests, food responses can become semi-automatic, without conscious reflection, which allows for real and effective decision-making.

"Health care undoubtedly involves proper nutrition and regular exercise. However, although these principles are widely known, maintaining them over time is a challenge for many people," explains UGR researcher Raquel Vilar, who adds that, "when it comes to food, one of the reasons for this difficulty lies in the fast pace of modern life, where unhealthy food is easily accessible and inexpensive."

Despite living in environments saturated with food stimuli, not everyone experiences the same degree of difficulty in adopting and maintaining healthy habits, suggesting that individual factors play a decisive role in making it harder for some people than others.

Guidelines to correct this problem

Once some of the differences that may explain the difficulties in maintaining a [healthy diet](#) have been identified, researchers have initiated a series of studies aimed at determining which strategies help maintain health goals. In a recent study, they found four effective techniques, focused on reducing impulsivity and relearning how to direct attention, increasing inhibitory control, and efficiently managing behaviors related to eating and physical exercise.

Intervention with these guidelines has yielded positive results. Overweight participants have improved their anthropometric parameters and eating patterns. In a series of new studies, the mechanisms of action of these techniques are being determined to find out whether they are effective because they change brain networks and reduce cognitive biases.

"In addition, we are combining them with other novel applications to try to further increase their effectiveness. Individual variability is important. In this regard, it is well known that [body weight](#) is the result of the interaction of multiple factors, not only those we have mentioned at the contextual, brain, and cognitive levels, but also endocrine and genetic variables," explains Caracuel.

Measuring results from a neuroscientific approach encompasses measurements of brain, cognitive, emotional, and behavioral factors, as well as physiological and genetic parameters, representing a significant advance in understanding the mechanisms that influence food choices and, therefore, body weight management and feelings of loss of control.

What exactly is TMS?

Transcranial magnetic stimulation (TMS), tested by researchers, is a non-invasive and painless technique that uses magnetic pulses to stimulate specific areas of the brain. These pulses modify the neural activity of specific brain regions that are targeted. Depending on the frequency and intensity of the pulses, TMS can either increase or decrease neural activity, making it a useful tool for treating various neurological and psychological conditions.

In the context of the research carried out at the UGR, TMS is used to prepare the brain for specific training related to inhibitory control. In this way, the aim is to optimize the brain's response to food-related impulses, promoting more conscious decisions that are aligned with long-term health goals.

Inhibitory control is a fundamental cognitive function that allows us to curb impulses, distractions, or immediate desires, while helping us make more rational decisions. In the case of eating, inhibitory control plays a crucial role: it helps curb impulses such as the desire to eat [unhealthy foods](#), eat for reasons other than hunger, and choose options that are more beneficial to health.

Characteristics of the studies

Current research is aimed at people who are overweight or who experience episodes of binge eating. In addition to interventions, the projects include detailed cognitive and behavioral assessments to measure their effectiveness. MRI scans are also performed to observe possible brain changes after the intervention, along with an anthropometric and biochemical examination.

Participation in these studies requires attending the CIMCYC at the University of Granada for two weeks, with daily sessions lasting between 10 and 15 minutes. This research center has been distinguished as a María de Maeztu Unit of Excellence, one of the most prestigious awards at the national level.

"With these two current projects, we hope to continue contributing to the development of more comprehensive and effective interventions that promote the adoption of a healthy lifestyle, offering specific solutions for each person," concludes researcher Raquel Vilar.

For more than 15 years, this scientific team, in collaboration with researchers from the United Kingdom and Australia, has taken a neuroscientific approach, developing neuropsychological intervention projects for people who are overweight, obese, or binge eaters.

More information: Andrea Bernat-Villena et al, Intermittent theta burst stimulation (iTBS) and inhibitory control training for excess weight treatment: study protocol for a randomized controlled trial (InhibE), *BMC Psychology* (2025). DOI: [10.1186/s40359-025-02556-9](https://doi.org/10.1186/s40359-025-02556-9)

Journal information: [BMC Psychology](#)

Provided by [University of Granada](#)

MAY 25, 2025

Just three nights of poor sleep might harm your heart: New study

by Annie Curtis, [The Conversation](#) edited by [Lisa Lock](#), reviewed by [Andrew Zinin](#)



Credit: Andrea Piacquadio from Pexels

We've long known that a lack of sleep is [bad for the heart](#)—but scientists are now starting to understand exactly how it causes harm.

In a [new study](#) from Uppsala University in Sweden, researchers found that just three nights of restricted sleep—around four hours a night—triggered changes in the blood linked to a higher risk of heart disease.

The researchers looked at [inflammatory proteins](#) in the blood. These are molecules the body produces when it is under stress or fighting off illness. When these proteins stay high for a long time, they can damage blood vessels and raise the risk of problems like heart failure, coronary heart disease and atrial fibrillation (irregular heartbeat).

The study involved 16 healthy young men who spent several days in a lab, where everything from their meals to their activity levels and light exposure was carefully controlled.

The participants followed two routines: three nights of normal sleep (8.5 hours) and three nights of sleep restriction (4.25 hours). After each sleep phase, the men completed a short, high-intensity cycling workout, and their blood was tested before and after.

Researchers measured almost 90 different proteins in the blood samples. They found that [sleep deprivation](#) caused a clear rise in inflammatory markers linked to heart disease. And while exercise usually boosts healthy proteins such as interleukin-6 and BDNF (which support brain and heart health), these responses were weaker after poor sleep.

Even young adults

Strikingly, the changes happened even in young, [healthy adults](#), and after only a few nights of bad sleep. That's worrying given how common it is for adults to [experience poor sleep from time to time](#)—and around [one in four people](#) work shifts that disrupt sleep patterns.

The researchers also discovered that the time of day blood was taken mattered: protein levels varied between morning and evening, and even more so when sleep was restricted. This suggests that sleep affects not only what's in your blood, but when those changes are most visible.

Although [modern life](#) often encourages us to trade sleep for productivity, socializing or [screen time](#), studies like this remind us that the body keeps score—quietly, chemically and without compromise.

Science News

from research organizations

Weight loss linked to nerve cells in the brain

May 27, 2025

Date:

University of Gothenburg

Source:

A specific group of nerve cells in the brain stem appears to control how semaglutide affects appetite and weight -- without causing nausea.

Summary:

A specific group of nerve cells in the brain stem appears to control how semaglutide affects appetite and weight -- without causing nausea. The discovery, made at the University of Gothenburg, could pave the way for better drugs to treat obesity.

Semaglutide belongs to a group of drugs called GLP-1R agonists and has been shown to effectively reduce food intake and body weight. The drug is already well established as part of the treatment for obesity and type 2 diabetes, but can cause side effects such as nausea and muscle loss.

In a new study, researchers at the Sahlgrenska Academy at the University of Gothenburg have shown that it is possible to distinguish the nerve cells in the brain that control the beneficial effects -- such as reduced food intake and fat loss -- from those that contribute to side effects. The study is published in the journal *Cell Metabolism*.

Activated nerve cells

To investigate how semaglutide affects the brain, the researchers worked with mice. They tracked which nerve cells were activated by the drug and were then able to stimulate these cells -- without administering the drug itself.

The result was that the mice ate less and lost weight, just as they did when treated with semaglutide. When these nerve cells were killed, the drug's effect on appetite and fat loss instead decreased significantly. However, side effects such as nausea and muscle loss remained.

"This suggests that these nerve cells control the beneficial effects of semaglutide. We have therefore identified a specific group of nerve cells that is necessary for the effects that semaglutide has on weight and appetite, but which does not appear to contribute to any significant extent to side effects such as nausea. If we can target the treatment there, we may be able to maintain the positive effects while reducing side effects," says Júlia Teixidor-Deulofeu, first author of the study and PhD student at Sahlgrenska Academy at the University of Gothenburg.

The effect in the brain

The identified nerve cells are located in an area of the brain called the dorsal vagal complex. For the researchers, the result is not only an early step toward potentially improved treatment, it also provides new knowledge about how semaglutide works in the brain. The study also provides deeper insight into how the brain stem regulates our energy balance.

"Semaglutide and other GLP-1R agonists are currently being prescribed to more and more people and are also being investigated for other potential indications such as substance use disorders and neurodegenerative diseases. It is important to understand how these drugs actually work. The better we understand this, the greater the opportunity we have to improve them," says Linda Engström Ruud, researcher and supervisor to PhD students Júlia Teixidor-Deulofeu and Sebastian Blid Sköldheden, who both worked on the project.

30 percent weight loss in mice by restricting one amino acid

NYU Grossman School of Medicine researchers discover cysteine depletion through diet and genetic engineering triggers rapid fat loss.

From NYU Langone Health / NYU Grossman School of Medicine 27/05/25 (first released 21/05/25)

Illustration by Superinnovators

Mice genetically engineered to lack the ability to make the amino acid cysteine, and fed a cysteine-free diet, lost 30 percent of their body weight in just one week, a new study shows.

Published online May 21 in *Nature*, the work found that cysteine depletion disrupts the normal metabolic pathways used by mammalian cells to convert food into energy, forcing the animals to rapidly burn fat stores in a futile attempt to meet energy demands.

Led by researchers at NYU Grossman School of Medicine, the study reveals key details about how cells process fuels like carbohydrates and fats (metabolism), and how cysteine depletion affects tissues.

Experiments showed that lowering cysteine levels caused a drop in levels of the small molecule called coenzyme A (CoA), which rendered inefficient mechanisms that convert carbohydrates and fats into energy.

Despite CoA being involved in more than 100 intermediate metabolic reactions and serving as a partner (cofactor) for 4% of all enzymes in the body, scientists had previously been unable to study its function directly.

This is because mice with defective CoA synthesis typically do not survive beyond three weeks of age. The current findings detail, for the first time, how CoA shapes metabolism in adult mice.

“Our surprising findings reveal that low cysteine levels triggered rapid fat loss in our study mice by activating a network of interconnected biological pathways,” said co-senior study author [Evgeny Nudler, PhD](#), the Julie Wilson Anderson Professor in the [Department of Biochemistry and Molecular Pharmacology](#) at NYU Grossman School of Medicine, and an investigator with the Howard Hughes Medical Institute.

“While driving weight loss in the clinic remains a key future mission, we are most excited for the moment about the profound, fundamental aspects of metabolism revealed in this study,” added Dr. Nudler.

The current finding does not immediately suggest a new approach to weight loss, the authors caution, as cysteine is found in nearly all foods.

Achieving a truly cysteine-free diet would require patients to consume a specially formulated solution that would be challenging for most.

Moreover, because cysteine is involved in numerous cellular pathways, eliminating it – such as through a drug that inhibits cysteine production – could make organs more vulnerable to everyday toxins, including medications.

That said, the study authors say it is worth considering that fruits, vegetables, and legumes contain much lower levels of cysteine and its precursor, the sulfur-containing amino acid methionine, than red meat.

While earlier studies have linked low sulfur amino acid intake to health benefits, this study clarifies that these benefits are due to cysteine depletion specifically, and not methionine restriction.

“Given that achieving maximum cysteine deprivation weight loss in the mice was dependent on both diet and deletion of the gene, moving forward we can now restore cysteine production genetically in specific cells or tissues and determine the role of each in the dramatic weight loss we observed,” said co-senior author [Dan L. Littman, MD, PhD](#), the Helen L. and Martin S. Kimmel Professor of Molecular Immunology in the [Department of Pathology](#), and a professor in the [Department of Cell Biology](#), at NYU Grossman School of Medicine.

“We hope in the future to hijack parts of this process to induce a similar weight loss in humans but without completely removing cysteine,” added Dr. Littman, who is also an investigator with the Howard Hughes Medical Institute.

Overlapping Mechanisms

The study is the first to examine the effects of removing cysteine, or any of the nine of the essential amino acids, which must be obtained through diet and are required for building proteins that make up most of the body’s enzymes, tissues, and signaling molecules.

The findings revealed that eliminating cysteine from the mammalian body led to far greater weight loss than the removal of any other essential amino acid.

Specifically, cysteine deprivation disrupted oxidative phosphorylation, the main process for producing adenosine triphosphate (ATP), the molecule that serves as cells’ energy currency.

Oxidative phosphorylation is known to be tightly dependent on CoA.

As a result, sugar-derived intermediate molecules (carbon skeletons) such as pyruvate, orotate, citrate, and α -ketoglutarate were no longer used efficiently, and were instead lost in the urine.

In response, the body turned to stored lipids (fats) to make energy.

Further, the team found that cysteine restriction activates both the integrated stress response (ISR), a signaling network that restores cellular balance after stress, and the oxidative stress response (OSR), which is triggered by higher levels of reactive oxygen species (ROS) following depletion of glutathione, the body's primary antioxidant.

ROS can oxidize (take away electrons from) and damage sensitive cell parts like DNA.

Remarkably, this simultaneous activation of ISR and OSR—previously observed only in cancer cells—was shown to occur in normal tissues in mice in the cysteine-restriction group, with the two stress responses reinforcing each other.

The study also shows that ISR and OSR, acting independently of CoA depletion, increase production of the stress hormone GDF15, which contributes to food aversion and degradation of acetyl-CoA-carboxylase, a key enzyme in lipid synthesis.

This increased weight loss further in the study mice by preventing the replenishment of their fat stores.

Along with Drs. Evgeny Nudler and Dan Littman, the study's co-senior authors, co-first authors were Alan Varghese, a joint student in labs of Drs Littman and Nudler in the Department of Cell Biology, and Ivan Gusarov in the Department of Biochemistry and Molecular Pharmacology at NYU Grossman School of Medicine.

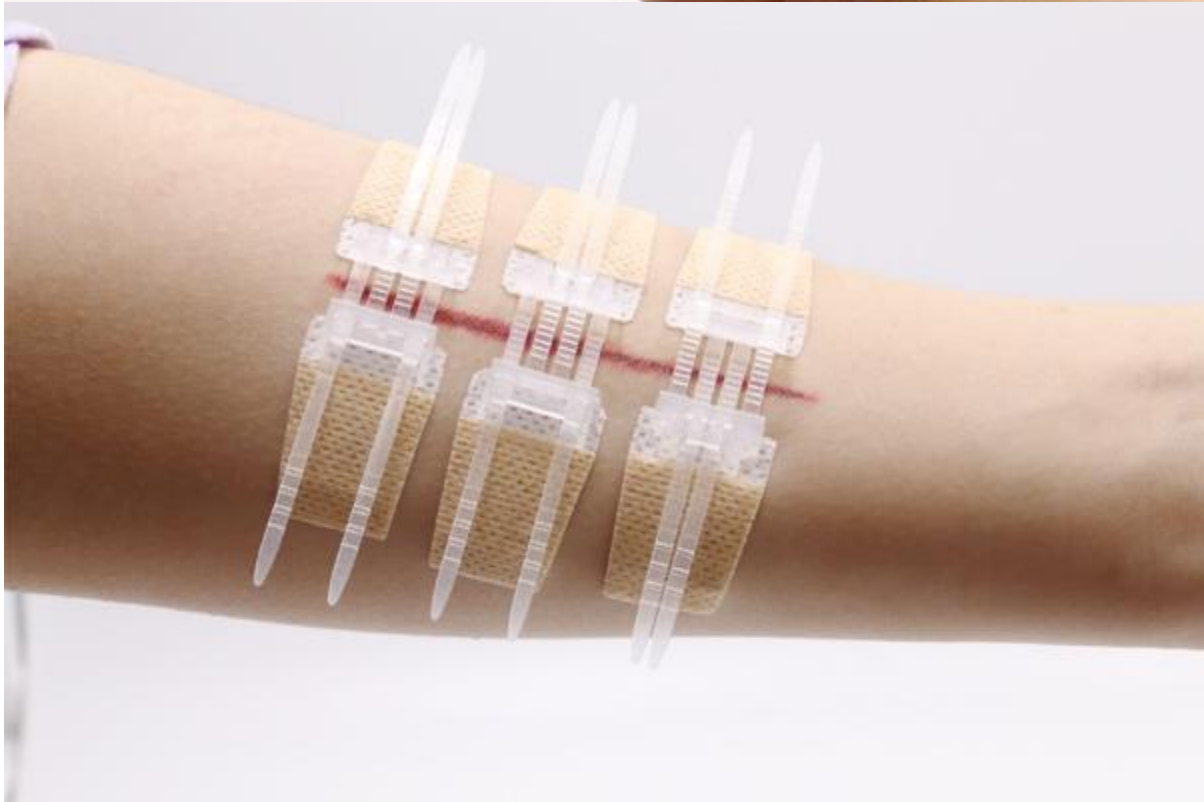
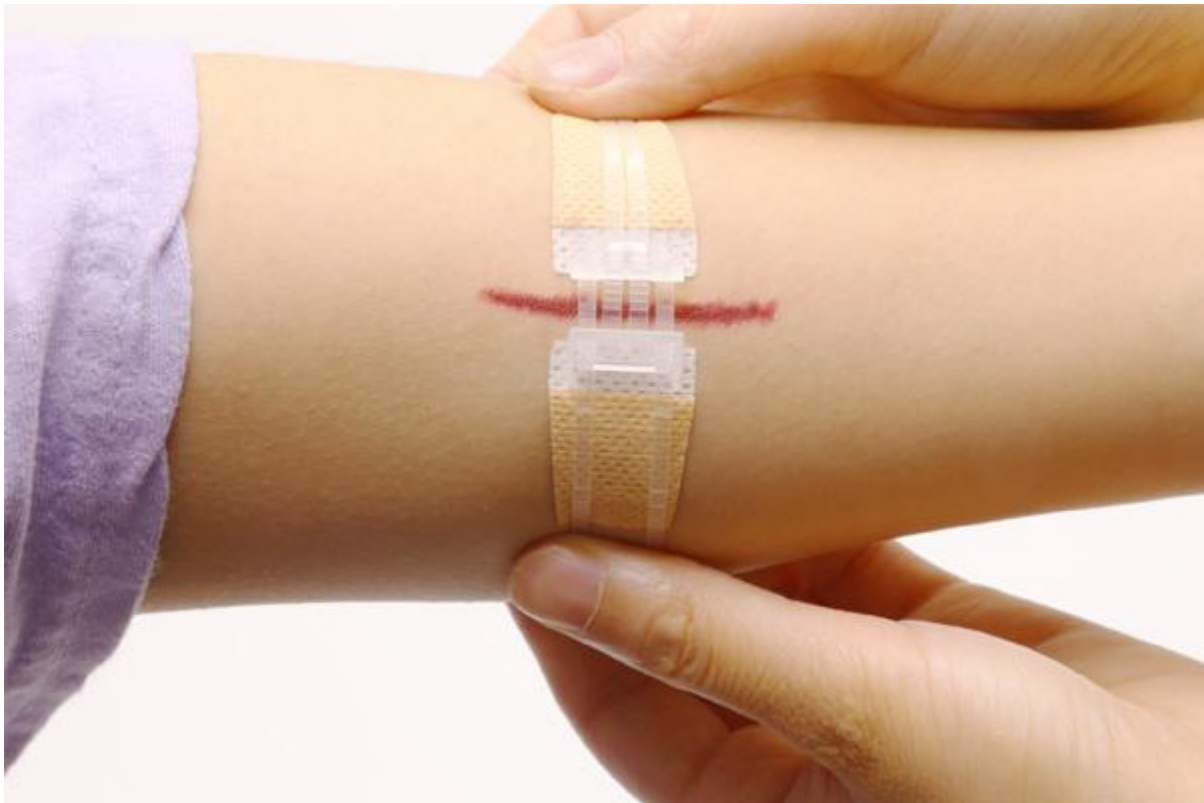
Additional NYU Langone contributors included Daria Dolgonos, Yatin Mankan, and Mydia Phan from the Department of Cell Biology; Ilya Shamovsky and Drew Jones from the Department of Biochemistry and Molecular Pharmacology; Begoña Gamallo-Lana and Adam Mar from the Department of Neuroscience; Rebecca Jones from the Division of Advanced Research Technologies; Thales Papagiannakopoulos from the Department of Pathology, and Michael Pacold from the Department of Radiation Oncology and Perlmutter Cancer Center.

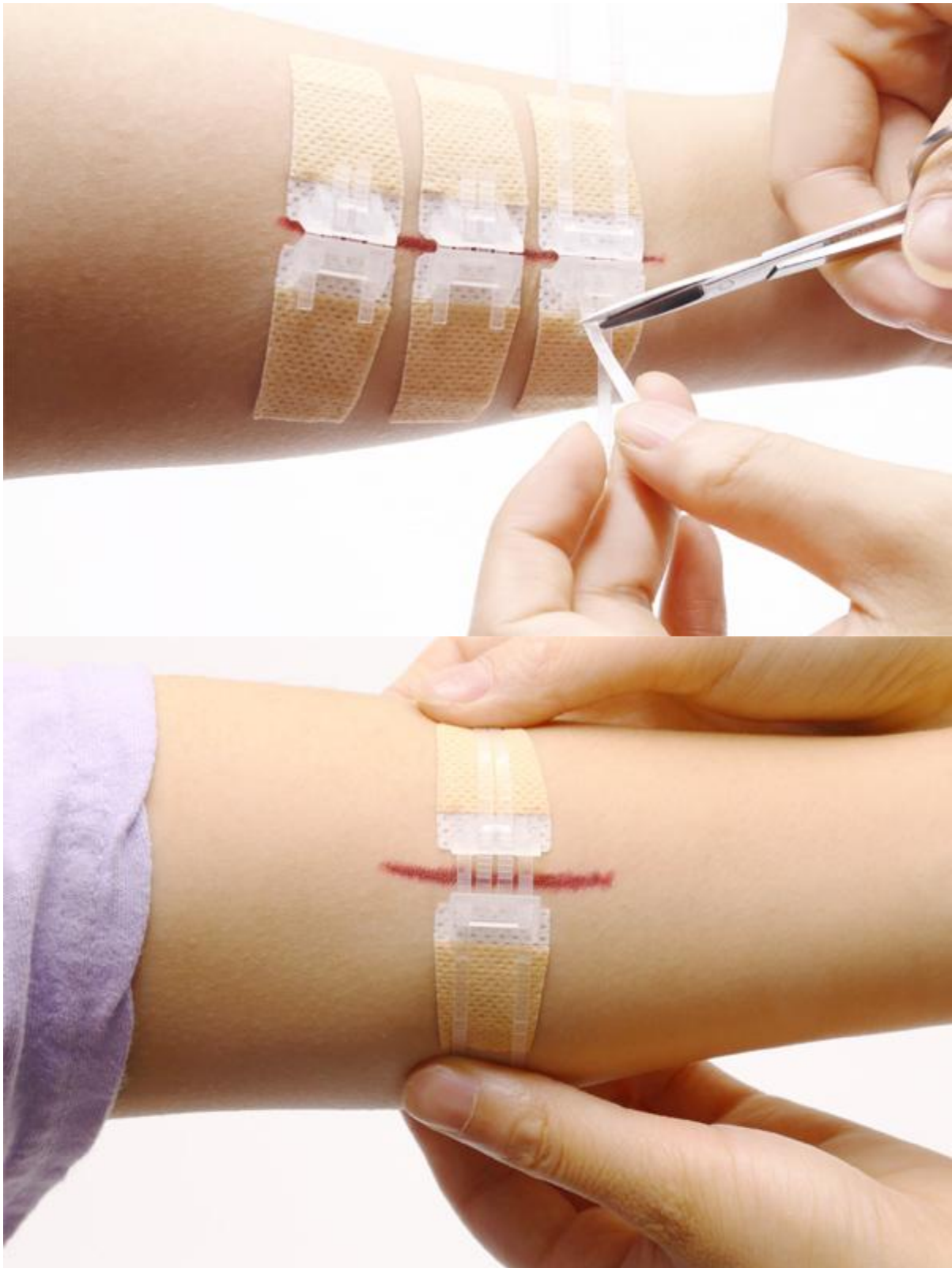
Other study authors were Maria Gomez-Jenkins and Eileen White of Rutgers Cancer Institute of New Jersey, and Rui Wang of the Department of Biology at York University in Toronto.

The study was supported by the long-term funding from the Howard Hughes Medical Institute (HHMI) and the Blavatnik Family Foundation.

Additional support was provided by the National Institutes of Health grants S10OD010584-01A1, S10OD018338-01, 1OT2CA278609-01, R35GM147119, and R01AI158687.

The research was also funded by the American Cancer Society (grant RSG-21-115-01-MM), the Natural Sciences and Engineering Research Council of Canada (grant RGPIN-2023-05099), and Cancer Research UK (grant CGCATF-2021/100022).





[Inquiry](#)
[WhatsApp](#)

[ZipStitch](#) Wound Closure Device is a surgical quality wound closure to help close minor lacerations quickly with no needles or puncturing of the skin. ZipStitch is the most advanced wound closure device available without a prescription. This is the exact same device some doctors are now using in surgery. The zip-tie strips enable micro-adjustment to precisely close the wound.

Zipstitch is an affordable option when you cut yourself and can't make it to the ER. An FDA cleared, highest quality, and hospital grade wound closure. Simple and easy to apply. Its technology has been clinically proven in over a dozen clinical studies to be safe and made with hypoallergenic materials. . Be prepared when accidents happen – anywhere. Needed in every first aid kit for emergency closure of wounds for the just in case incidents. Reduces risk of infection and avoids damaging any surrounding skin tissue. Evenly closes from both sides of the wound to provide a quality closure with less scarring.



Instruction for use: Application

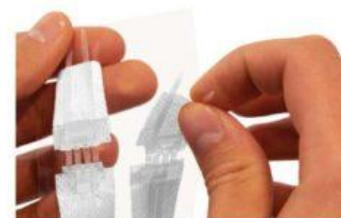
1. For patients with deep wounds, subcutaneous suture operation can be performed according to local clinical procedures, if necessary. See Figure 1



2. Clean and dry skin around the wound. See Figure 2



3. Select number of Zip Wound Closure hasps according to the wound size.



4. Take the Zip Wound Closure Device out of the package. See Figure 3



5. Hold three ends of the Zip Wound Closure Device, remove the liners. See Figure 4



6. Ensure the hasps cover both sides of the wound. Press adhesive tape with fingers gently along the direction of the ribbon and ensure that the adhesive tape sticks firmly on the skin. NOTE: Apply one pair of the Zip Wound Closure Device at every 2 cm of the wound. See Figure 6



7. Using both hands to hold the ribbon of the Zip Wound Closure Device, tighten the hasp gently to close the wound until the hasps are locked. See Figure 7



8. Cut off the long end of the ribbon. See Figure 8

9. Apply a suitable secondary dressing to protect the hasps and the site.

10. Observe the wound; apply appropriate dressing if necessary.

[Longmed](#) Wound Zip Tie Surgical Closure Device consists of medical adhesive tape and a pair of polypropylene hasps. The tape is porous and non-woven. The polypropylene hasps are designed to allow for a non-invasive zipper sutures wound closure of a wound and to encourage a tight closure of the wound for an early and successful healing.

It is a new technique that reduces any more tissue trauma, improves patient comfort, and may have less scarring after healing.

1. No nerve deadening injections, sutures, nor staples for skin closure.

2. No additional tissue damage which is caused by stitches and staples closures.
3. Precise and unique structure design ensures the sufficient closing force when pulling the two sides of skin together.
4. Holds the two sides together for quicker healing and less potential of infection.
5. Easy to apply with irregular lacerations.
6. Provides an even closure from both sides of the laceration to center the wound. It can be tightened gradually, especially for the edema wounds.
7. Consists of hypo-allergenic top grade medical adhesive.
8. The self-locked structure design ensures the ribbons will not be loosened during the healing process.
9. Decreases potential formation of hypertrophic and keloid scars.
10. Reduces surgical complexity and speeds up wound closure.
11. Simple & easy to apply when a trip to the ER is not an option.
12. Reduces risk of infection and avoids damaging of skin edges and adjacent tissue.
13. Allows to clearly observe the wound during the healing process and easy to apply daily an anti-bacterial medicine.
14. Easy to remove after the wound is healed.

What happened to zipstitch?

Zipstitch was designed as an alternative to traditional sutures or staples for closing certain types of wounds. ZipStitch consisted of a flexible adhesive strip with adjustable zip-like fasteners that could be used to close a wound without the need for stitches or staples. It helps reduce the wound tension and less scars.

Zipstitch wound closure device is typically used for minor to moderate lacerations or cuts that do not require extensive surgical intervention. ZipStitch is marketed as a consumer-friendly alternative for first aid treatment of certain wounds. Here are some common scenarios where ZipStitch could be used:

Home and First Aid Kits: ZipStitch is a convenient addition to first aid kits for use in emergencies, outdoor activities, or situations where immediate medical attention may not be readily available.

Minor Lacerations: It is suitable for small to medium-sized cuts or lacerations that would typically be treated with adhesive strips or butterfly bandages.

Low Tension Wounds: ZipStitch is designed for wounds in low-tension areas,

such as the arms, legs, or torso.

Post-Surgical Incisions: In some cases, Zip Stitches may be used to reinforce surgical incisions after minor surgical procedures.





longmed

Longmed Medical is a global manufacturer of medical product in CHINA with CE,FDA and ISO13485 certificates and its aim is to enable to protect the healthcare providers and help the patients recover soon.

Less pain¹, higher patient satisfaction² and less scarring¹

Trade in sutures, staples and glue for Zip's non-invasive, puncture-free skin closure³.
Zip's technology is designed with you in mind – so you can heal more comfortably.

How Zip works

Unlike sutures and staples, Zip helps improve your experience by creating a puncture-free skin closure³.





Allowing for movement ^{1,2}

But Zip's unique, flexible design allows you to move more freely during recovery.

Isolating the incision

Zip holds the edges of an incision together with a gentle adhesive, allowing for less pain¹, and uses plastic straps to keep the incision closed and protected – so you can focus on healing.

Benefits of Zip



Less pain, your gain

Significantly lower pain than staples⁴

It's designed with your comfort in mind.



Scar less, heal better

Significantly better cosmetic appearance

When it comes to scarring, you could call us minimalists. Zip helps minimize scarring, so you can say goodbye to "railroad" marks caused by staples and sutures^{1,5}.



Stay strong

12x stronger than sutures

Zip offers 12x stronger protection than sutures⁶, allowing your incision to heal quickly and safely.



Get moving

Flexible design allows movement

Zip moves with you, so you can have a better range of motion during recovery^{1,4}.



Zip in, bacteria out

Reduced risk of infection and complications

Zip lets you heal with reduced risk of infection and complications^{3,7}. It's good for your peace of mind, and even better for your body.



Better outcomes = Happier patients

Increased patient satisfaction²

Zip feels better and has better cosmetic outcomes, so it's no wonder that patients like it more than staples or sutures.

Zip can help create a better patient experience.



Feels better

Because Zip is puncture-free, it's gentler on the incision from the get go, and less painful after surgery¹.



Moves with you

Zip allows greater range of motion during healing¹, so you can recover confidently.



Removes with ease

Zip is less painful to remove than staples^{1,4} – and its non-invasive technology helps reduce the risk of infections and complications^{3,7}.

Lets you feel like you again

With less scarring and better cosmetic outcomes than staples or sutures^{1,2,5,8}, Zip lets you get back to focusing on the things you love instead of on how you look post-recovery.

Love Zip? Talk to a professional to learn more.

[FIND A SURGEON](#)

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*compared to staples

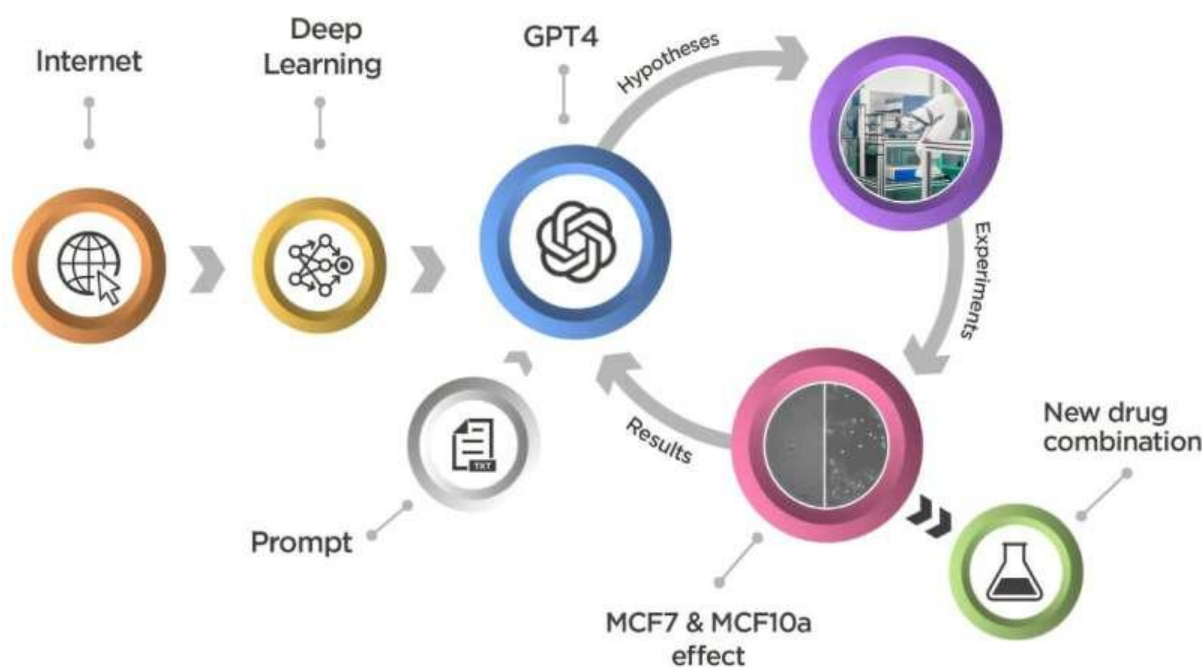
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Last Updated October/2024

JUNE 4, 2025

'AI scientist' suggests combinations of widely available non-cancer drugs can kill cancer cells

by [University of Cambridge](#)

edited by [Sadie Harley](#), reviewed by [Robert Egan](#)



The overall structure of our experiments. GPT4 was previously trained on data on a large fraction of the text on the Internet. Credit: *Journal of The Royal Society Interface* (2025). DOI: 10.1098/rsif.2024.0674.

An "AI scientist," working in collaboration with human scientists, has found that combinations of cheap and safe drugs—used to treat conditions such as high cholesterol and alcohol dependence—could also be effective at treating cancer, a promising new approach to drug discovery.

The research team, led by the University of Cambridge, used the GPT-4 large language model (LLM) to identify hidden patterns buried in the mountains of scientific literature to identify potential new cancer drugs.

To test their approach, the researchers prompted GPT-4 to identify potential new [drug combinations](#) that could have a significant impact on a breast cancer cell line commonly used in medical research. They instructed it to avoid standard cancer drugs, identify drugs that would attack cancer cells while not harming healthy cells, and prioritize drugs that were affordable and approved by regulators.

The drug combinations suggested by GPT-4 were then tested by human scientists, both in combination and individually, to measure their effectiveness against breast cancer cells.

In the first lab-based test, three of the 12 drug combinations suggested by GPT-4 worked better than current breast cancer drugs. The LLM then learned from these tests and suggested a further four combinations, three of which also showed promising results.

[The results](#), reported in the *Journal of the Royal Society Interface*, represent the first instance of a closed-loop system where experimental results guided an LLM, and LLM outputs—interpreted by human scientists—guided further experiments.

The researchers say that tools such as LLMs are not replacements for scientists, but could instead be supervised AI researchers, with the ability to originate, adapt and accelerate discovery in areas like [cancer research](#).

Often, LLMs such as GPT-4 return results that aren't true, known as hallucinations. But in scientific research, hallucinations can sometimes be a benefit, if they lead to new ideas that are worth testing.

"Supervised LLMs offer a scalable, imaginative layer of scientific exploration, and can help us as human scientists explore new paths that we hadn't thought of before," said Professor Ross King from Cambridge's Department of Chemical Engineering and Biotechnology, who led the research. "This can be useful in areas such as [drug discovery](#), where there are many thousands of compounds to search through."

Based on the prompts provided by the human scientists, GPT-4 selected drugs based on the interplay between biological reasoning and hidden patterns in the scientific literature.

"This is not automation replacing scientists, but a new kind of collaboration," said co-author Dr. Hector Zenil from King's College London. "Guided by expert prompts and experimental feedback, the AI functioned like a tireless research partner—rapidly navigating an immense hypothesis space and proposing ideas that would take humans alone far longer to reach."

The hallucinations—normally viewed as flaws—became a feature, generating unconventional combinations worth testing and validating in the lab. The human scientists inspected the mechanistic reasons the LLM found to suggest these combinations in the first place, feeding the system back and forth in multiple iterations.

By exploring subtle synergies and overlooked pathways, GPT-4 helped identify six promising drug pairs, all tested through lab experiments. Among the combinations, simvastatin

(commonly used to lower cholesterol) and disulfiram (used in [alcohol dependence](#)) stood out against breast [cancer cells](#). Some of these combinations show potential for further research in therapeutic repurposing.

These drugs, while not traditionally associated with cancer care, could be potential cancer treatments, although they would first have to go through extensive clinical trials.

"This study demonstrates how AI can be woven directly into the iterative loop of scientific discovery, enabling adaptive, data-informed hypothesis generation and validation in real time," said Zenil.

"The capacity of supervised LLMs to propose hypotheses across disciplines, incorporate prior results, and collaborate across iterations marks a new frontier in scientific research," said King.

"An AI scientist is no longer a metaphor without experimental validation: it can now be a collaborator in the scientific process."

More information: Abbi Abdel-Rehim et al, Scientific Hypothesis Generation by Large Language Models: Laboratory Validation in Breast Cancer Treatment, *Journal of The Royal Society Interface* (2025). DOI: [10.1098/rsif.2024.0674](https://doi.org/10.1098/rsif.2024.0674)

Journal information: [Journal of the Royal Society Interface](#)
Provided by [University of Cambridge](#)

JUNE 10, 2025

Why a fluttering heart could lead to stroke

edited by [Sadie Harley](#), reviewed by [Andrew Zinin](#)

Atrial fibrillation is a common type of heart rhythm disorder where the heart doesn't beat efficiently. This makes the heart unable to pump enough blood out to the body with each heartbeat. It's estimated 12.1 million people in the U.S. will be diagnosed with AFib by 2030.

Lowering stroke risk

People with AFib are at a higher risk of stroke— about one in seven strokes are AFib related—due to clots that can form in the upper chambers of the heart. Of the strokes resulting from atrial fibrillation, 90% occur from clots originating in the [left atrial appendage](#) in the left atrium of the heart.

Key risk factors for stroke in people with atrial fibrillation include:

- Age
- Gender
- High blood pressure
- Prior history of stroke
- Chronic health conditions, including [congestive heart failure](#), coronary artery disease and diabetes

Treating AFib

There are three main approaches to treating AFib. These are:

- Medical therapy for maintaining normal rhythm and preventing clots.
- Therapy to reset the heart rhythm, called cardioversion.
- Procedures to obtain and maintain normal heart rhythm.
- Medications.
- Medications for treating AFib are the front line for managing symptoms and preventing stroke. These medicines can:
 - Prevent blood clots.
 - Control the speed of the heartbeat.
 - Restore the heart rhythm.

This group of medications includes [beta blockers](#), [calcium channel blockers](#), digoxin, arrhythmics and anticoagulants. The cardiology team determines which medication will work best.

Cardioversion

Cardioversion is usually done in a hospital as a scheduled procedure. Patients may still need to take medicines for the rest of their lives to control their heart rhythm and prevent future episodes of atrial fibrillation. Even with medicine, AFib could return.

Procedural options

People who are candidates for procedural options are cared for by a team of cardiologists, including cardiac electrophysiologists and additional specialists as needed. Potential procedures include:

Left atrial appendage occlusion: The left atrial occlusion is a pouch-like extension of the heart. A minimally invasive procedure provides an alternative to long-term blood-thinner medications. It involves implanting a device, via a catheter, to close and seal off the left atrial appendage. This decreases the risk of blood clots entering the bloodstream, traveling to the brain or other body organs, and causing a stroke or organ damage. Patients typically leave the hospital the same day or the next day.

Ablation: Ablation stops the generation of abnormal electrical signals in the heart and keeps it in normal, or sinus, rhythm. During ablation, a catheter is fed into the heart through the groin area using a minimally invasive approach and delivers heat or cold to modify the tissues in the heart that are causing the arrhythmia.

Pulsed field ablation (PFA): PFA stands out from traditional atrial fibrillation (AFib) treatments due to its precision and safety. Unlike radiofrequency or cryoablation, which use heat or cold to destroy heart tissue, PFA uses short electrical pulses to target the myocardium, minimizing damage to the esophagus and nerves.

Benefits to patients include:

- Reduced procedure and anesthesia time
- Quicker recovery time
- Surrounding tissue protection

Hybrid ablation: For people who have long-standing, persistent atrial fibrillation, ablation alone is successful half of the time. Hybrid ablation is an option for people with atrial fibrillation that's hard to manage or who have been in [atrial fibrillation](#) for more than a year. It combines the best of the catheter lab ablation and an open surgical approach.

The procedure is done in two parts:

Part one is performed by surgeons with a scope inserted through a small incision under the breastbone to ablate the back side of the heart, followed by closure of the left atrial appendage.

During part two, a [catheter ablation](#) modifies the tissues on the inside of the heart.

This whole-heart approach allows patients to return to normal rhythm with excellent results.

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JUNE 10, 2025

AI used to analyze cancer samples to improve treatment outcomes

by Children's Medical Research Institute

edited by [Lisa Lock](#), reviewed by [Andrew Zinin](#)

A new way of using artificial intelligence (AI) to analyze thousands of patients' cancer samples has been developed by scientists at Children's Medical Research Institute (CMRI) in an international collaboration [published](#) in the journal *Cancer Discovery*.

The ProCan cancer research program at CMRI is analyzing thousands of different types of proteins (the proteome) in childhood and adult cancers to help cancer clinicians match their patients with the best treatment available. They are a step closer to that goal with this study that involves 30 collaborating research groups in six countries (Austria, Australia, Canada, Greece, Spain and the U.S.), and cancer proteomic data obtained by the ProCan team from 7,525 cancers, which is the largest set of cancer proteomes generated in a single center.

The reason that the size of the dataset matters is that predicting how a cancer will behave based on the proteome, including how the cancer will respond to treatment, requires advanced computational techniques. This includes AI, which needs to be trained on large datasets that include both the proteome and clinical information about the patient. However, data privacy regulations and other restrictions on the transfer of data across geographical boundaries make it challenging to assemble large sets of patient data, especially when multiple countries are involved.

The ProCan team have shown how this problem can be overcome by simulating the situation where permission is given to use proteomic and [clinical data](#), but with very restricted access.

Using an AI technique called federated deep learning, they trained AI models on datasets stored at several local sites held behind firewalls. Instead of sharing clinical data, these AI models were sent to a central server to update a global model. Repeating this process multiple times resulted in a [diagnostic test](#) that has essentially the same accuracy as when the data was all brought together in one centralized database.

Professor Roger Reddel, a senior author of the publication, said, "It was a very exciting moment when we first saw that the results from data with highly restricted access were just as accurate as the results obtained when the data was all stored in one place."

In addition, this work has overcome another problem regarding proteomic data that has made it very difficult to build large datasets. Different research institutions use different methods for obtaining proteomic data from cancer samples, and this is a major barrier to combining proteomic data from different research centers.

As part of the research reported in this publication, the team showed that federated [deep learning](#) made it possible to successfully combine the proteomic data generated at CMRI from the 7,525 cancer samples with proteomic data generated at other research institutions with different techniques, and that this further improved the accuracy of the cancer diagnosis.

These advances will speed up the achievement of ProCan's mission to use proteomic data to improve outcomes for cancer patients.

Professor Reddel said, "The purpose of CMRI's ProCan research program is to develop proteomic tests that will assist cancer clinicians to choose the best treatment available for each of their patients. By overcoming several major barriers to assembling and analyzing large [cancer proteomic](#) datasets, we have made a major step towards achieving this goal."

More information: Zhaoxiang Cai et al, Federated deep learning enables cancer subtyping by proteomics, *Cancer Discovery* (2025). DOI: [10.1158/2159-8290.CD-24-1488](https://doi.org/10.1158/2159-8290.CD-24-1488)

Journal information: [Cancer Discovery](#)

Provided by Children's Medical Research Institute

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JUNE 10, 2025

Ultra-processed foods are everywhere—and they're quietly raising health risks

by Angelina Baric and Anthea Christoforou, [The Conversation](#)

edited by [Lisa Lock](#), reviewed by [Andrew Zinin](#)

It's not exactly news that junk food isn't healthy.

What may be surprising is exactly how pervasive ultra-processed foods (UPFs) have become and what harm they're doing. This includes some foods that are specifically labeled and marketed as having nutritional value.

We are nutrition researchers, and the authors of a [new study](#) that identifies some of the specific negative effects of ultra-processed foods that are readily available, very popular and often hard to resist, especially when people are feeling pressed for time.

Our research group leads population-based studies that integrate nutrition epidemiology, [food policy](#), and dietary assessment to better understand how modern food environments and dietary patterns influence chronic disease risk.

While ultra-processed foods include obvious culprits like [potato chips](#), candy and frozen pizza, there are also some that people may believe are good for them, such as packaged granola bars, sports drinks and fruit-filled yogurt. Our study used the [Nova classification system](#) to define UPFs, which are industrial formulations made mostly or entirely from substances extracted from foods, derived from food constituents with little if any intact whole food remaining.

How UPFs harm health

Our research, based on diet questionnaires and personal medical data that Health Canada and Statistics Canada collected from more than 6,000 Canadians, shows that the effects of UPFs can pile up over time, adding to the risk of heart attack, stroke and other serious health issues by raising [blood pressure](#) and blood sugar levels, for example.

Even a person who is thin, active and free from illness might be accumulating risk by consuming UPFs that may seem innocuous or even healthy.

The ways ultra-processed foods harm our health aren't just about calories or individual nutrients like salt, sugar and fat, though those aren't making things better. It's also in the way they're made.

Take that seemingly healthy tub of yogurt. On its own, yogurt is indeed very healthy. The problem is when things like jam-like fruit with preservatives or artificial vanilla flavoring are added. They make yogurt taste better but can push it into unhealthy territory.

Even after we eliminated the impacts of influences such as the survey respondents' body mass index, age, exercise and smoking habits, the numbers showed a specific risk that may be related to the additives that give ultra-processed foods longer shelf life, brighter colors and enhanced flavors. For example, we know that the modern diet of highly processed food is associated with [distorted hormone levels](#).

Some products are so heavily processed that it appears our bodies [may not respond to them](#) as they would to more natural foods. [UPFs trigger inflammatory responses](#) that suggest the body regards them as stressors, rather than nutrition.

Substituting UPFs for healthy foods

We learned from survey respondents that consumers are increasingly using UPFs as substitutes for healthy staples such as vegetables and fruit. This is not surprising when the wrapper on a granola bar proclaims its contents to be a good source of fiber, or when a sports drink label says it's a good source of electrolytes, Vitamin D or some other single nutrient.

While these claims are factual, they don't represent the entire or even most significant effects of the products inside. For a long time, [food policies](#) have been very focused on single nutrients rather than thinking about the totality of our food supply.

Our complex food supply has come to be [heavily influenced by huge multinational companies](#) and their need for sales instead of our need for health, to the point where [marketing](#) and packaging have made it challenging to understand exactly what we are eating or drinking.

That is starting to change. Starting in January 2026, the Canadian government will require [food packaging to prominently declare](#) the presence of unhealthy amounts of sodium, sugar and saturated fat.

While that will be a significant and welcome improvement in transparency, it will not change the fact that a loaf of mass-manufactured [white bread](#), a package of bacon or even a tray of muffins may also be harming the people who eat them in ways they have not even considered.

Setting reduction targets

[Canada's food guide](#), produced by Health Canada, only suggests we limit the amount of processed food we eat, but it doesn't set any clear national target for how much we should cut our consumption. While most other countries also stop short of setting specific

limits, [France](#) has gone a step further by aiming to cut national consumption of ultra-processed foods by 20% over five years.

Setting a similar national reduction target in Canada could have a particularly significant, positive effect on people in [care homes](#), hospitals and schools that are required to use Canada's Food Guide in planning their menus.

Individual consumers know how easy it is to fall into the habit of eating too many ultra-processed foods. They are hard to resist because they are [heavily marketed](#), usually tasty, reasonably affordable and appear to make life easier by saving time and effort.

Understanding more about what these appealing products are really doing to people is an important step toward helping consumers make better, more informed choices. We are already working on more research to understand more about what's really inside those bright shiny packages that keep finding their way into shopping carts.

Provided by [The Conversation](#)

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JUNE 10, 2025

Rapid AI-assisted test developed for viral infection screening

by PNAS Nexus edited by [Gaby Clark](#), reviewed by [Robert Egan](#)

Structure of the herpes virus, nanopore module, and its waveform. Credit: *PNAS Nexus* (2025). DOI: 10.1093/pnasnexus/pgaf161

A non-DNA based test could identify viral infections in patients in minutes. When a clinician suspects a patient has a viral illness, the presence of specific virus types can be confirmed through a DNA sequencing test. However, the test takes several hours, even if a testing facility is available on site, and the test cannot discern whether the virus is viable.

In a study [published](#) in *PNAS Nexus*, Noriyasu Hashida and colleagues designed a test that confirms the presence of live virus by pushing particles through a nanopore, one at a time, and measuring their electrical conductivity, which varies with size and surface charge as well as the unique molecular structure of the virus.

A 2- μ m polystyrene bead passing through a 3- μ m diameter pore. Credit: NOK Corporation
Using an AI model, the device can use [electrical conductivity](#) waveforms to identify viruses and even distinguish viruses within the same family. The system was tested on herpesvirus that infect the eye, which can lead to blindness.

Samples of aqueous humor—the watery fluid around the eye—were analyzed with both PCR and the AI nanopore method. The sensitivity of the AI nanopore method was 100%, and the specificity was 71.4%, improving to over 90% when conditions were adjusted.

According to the authors, as the [technology](#) matures, it could help clinicians catch and treat viral ocular infections—and likely other types of [viral infections](#) as well.

More information: Noriyasu Hashida et al, Rapid diagnosis of ocular viral infections via single-virus detection using solid-state nanopore: A diagnostic evaluation study, *PNAS Nexus* (2025). DOI: [10.1093/pnasnexus/pgaf161](https://doi.org/10.1093/pnasnexus/pgaf161)

Journal information: [PNAS Nexus](#)

Provided by PNAS Nexus

JUNE 10, 2025

hMPV is likely one of the viruses making us sick this season. Here's what to know about human metapneumovirus

by Lara Herrero, [The Conversation](#)

edited by [Lisa Lock](#), reviewed by [Andrew Zinin](#)

Credit: Andrea Piacquadio from Pexels

As winter settles over Australia, it's not just the drop in temperature we notice—there's also a sharp rise in respiratory illnesses. Most of us are familiar with [the usual winter players](#) such as COVID, influenza and RSV (respiratory syncytial virus), which often dominate news headlines and public health messaging.

But scientists are now paying closer attention to another virus that's been spreading somewhat under the radar: [human metapneumovirus](#) (hMPV).

Although it's not new, hMPV is now being recognized as a significant contributor to [seasonal respiratory infections](#), especially among [young children](#), [older people](#), and people with weaker immune systems.

So what do you need to know about this winter lurgy?

What does a hMPV infection look like?

hMPV is a [close relative of RSV](#), and can cause infections in the upper or lower respiratory tracts.

Like other respiratory viruses, [hMPV infection causes symptoms](#) such as cough, fever, sore throat and nasal congestion. While most people experience relatively mild illness and recover in about a week, hMPV can lead to [serious illness](#)—such as bronchiolitis or pneumonia—in babies, older adults, and people with weakened immune systems.

hMPV spreads much like the flu or SARS-CoV-2 (the virus that causes COVID)—through [tiny droplets](#) from coughs and sneezes, and potentially by [touching surfaces](#) where the virus has landed and then touching your mouth, nose, or eyes.

Most people will catch it at some point in their lives, commonly [more than once](#). While an infection confers some immunity, this [waned over time](#).

hMPV generally [follows a seasonal pattern](#), tending to peak in [winter and spring](#).

hMPV around the world

By the end of 2024, China saw a surprising [spike in cases of hMPV](#)—enough to catch the attention of public health experts. While there were some suggestions hospitals were becoming overwhelmed, exact numbers were not clear.

The World Health Organization subsequently [issued a statement](#) in January indicating this rise in hMPV infections in China aligned with expected seasonal trends.

Other countries, such as the [United States](#), have also noted increases in hMPV infections since the COVID pandemic. Realizing hMPV might be playing [a more significant role](#) in seasonal illness than we'd previously thought, and with [improvements in diagnostic technology](#), global health agencies have ramped up their monitoring.

In Australia, comprehensive national data on hMPV is limited because hMPV is not one of the viruses [with mandatory reporting](#). In other words, if a patient is found to have hMPV (through a PCR swab sent to a pathology lab) there's no requirement for the doctor or the pathology lab to make a [public health](#) report of a positive result, as they would with another illness such as influenza, RSV or measles.

However, selected [medical clinics](#) voluntarily participate in [systematic data collection](#) on specific health conditions, which gives us an idea of the proportion of people who may be infected (though not the absolute numbers).

The Australian Sentinel Practice Research Network ([ASPREN](#)) is a national surveillance system funded by the federal department of health. In 2024, up to December 15, based on ASPREN data, 7.8% of patients presenting with fever and cough symptoms [tested positive for hMPV](#).

This year, to June 1, ASPREN data shows us hMPV has made up [4.2% of infections](#) among people with flu-like illness, behind RSV (7.7%), COVID (10.9%), influenza (19%) and rhinovirus (a virus which causes the common cold, 46.1%).

What about vaccines and treatments?

hMPV is likely to be part of the array of respiratory viruses circulating in Australia this winter. If you have a cold or flu-like illness and have done one of those at-home rapid tests for COVID, flu and RSV but came up all negative, it's possible hMPV is the culprit.

There's currently no specific treatment or vaccine for hMPV. Most cases are mild and can be managed at home with rest and symptom relief such as taking medication (paracetamol or ibuprofen) for pain and fever. But more serious infections may require hospital care.

If your baby or young child has a respiratory [infection](#) and is having trouble breathing, you should take them to the emergency department.

Researchers and companies such as [Moderna](#), Pfizer and [Vicebio](#) are actively working on vaccines for hMPV, however they're not yet available.

The best way to protect yourself and others against hMPV and other respiratory viruses is through [simple hygiene practices](#). These include washing your hands often, covering coughs and sneezes, staying home if you're sick, cleaning shared surfaces regularly, and considering wearing a mask in crowded indoor spaces during virus season.

Provided by [The Conversation](#)

JUNE 11, 2025

Pasteurizing fruit smoothies could improve digestion of beneficial polyphenols

by [American Chemical Society](#) edited by [Stephanie Baum](#), reviewed by [Robert Egan](#)

Smoothies pasteurized with pressure (left image) or heat (right image) have higher amounts of digestible polyphenols than raw smoothies. Credit: Izlar Ludwig

Drinking a smoothie is a popular way to consume fruits and vegetables, many of which are rich in micronutrients called polyphenols. If this beverage is purchased at a store, it's likely been pasteurized with heat or pressure to prevent harmful bacteria growth and extend shelf-life.

Now, a [preliminary study](#) in the *Journal of Agricultural and Food Chemistry* reports that processing [smoothies](#) with [high heat](#) could also make polyphenols easier for the [gut microbiome](#) to absorb.

Fruits and vegetables are key to a healthful human diet, partly because they contain polyphenols, which can protect against [heart disease](#) and neurodegenerative disorders. These beneficial components are released from food during digestion, making them available for absorption by the gut microbiome.

Previous research had found that food processing techniques like canning and boiling peppers and artichokes may increase the amount of polyphenols metabolized by the gut microbiota, potentially improving the health benefits of these foods. But gaps exist in scientists' knowledge of other food processing techniques. So, Izlar Ludwig and colleagues investigated how pasteurization could affect polyphenol digestion in the gut microbiome.

The researchers first prepared a smoothie made up of Granny Smith apples, green celery, green chicory, peppermint and lemon. They split up the smoothie into three parts: One was not treated, and the others underwent either high-pressure or high-temperature pasteurization.

Then, the smoothie samples were added to successive solutions meant to mimic the three stages of digestion—oral, gastric and intestinal. Post-digestion pressure- and heat-pasteurized smoothie samples had higher amounts of polyphenolic compounds (21% and 44%, respectively) available for absorption than the untreated sample (17%). The researchers attribute these results to changes in the plants' cell walls, such as softening or rupturing, induced by pasteurization that could favor the release of polyphenols into the body.

Finally, to analyze gut microbiome transformations of polyphenols, the digested samples went through a laboratory version of colonic fermentation in vials containing human feces as the microbiota source. The gut microbiota converted most [polyphenols](#) into smaller derivatives, such as phenylpropanoic acids. Some of these derivatives have previously demonstrated antidiabetic, anti-inflammatory and chemopreventive effects.

The researchers determined that the largest microbiota conversions happened in the high-temperature, post-digestion smoothie sample because it started fermentation with higher overall polyphenol levels. They say this work emphasizes how smoothie processing could lead to new beverage products with enhanced bioaccessibility.

More information: High-Pressure and Thermal Pasteurization Applied to Smoothies Enhances (Poly)Phenol Bioaccessibility along the Gastrointestinal Tract, *Journal of Agricultural and Food Chemistry* (2025). DOI: [10.1021/acs.jafc.4c09166](https://doi.org/10.1021/acs.jafc.4c09166)

Journal information: [Journal of Agricultural and Food Chemistry](#)
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