LabMedya®

HEALTH AND LABORATORY MAGAZINE

ISSN 2148-953X
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12

02

"BACTERIA SWIM" MAY PREVENT THE SPREAD OF DISEASES

04

AVOCADOS ARE LINKED TO LOWER RISK OF CARDIOVASCULAR DISEASES

16

OMEGA-3 SUPPLEMENTATION COULD BOOST IMMUNOTHERAPY'S CANCER-FIGHTING POWER

LabMedya
No: 07 April 2022
ISSN: 2148-953X

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DATE OF ISSUE
February 2022 - Ankara

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WHAT IS LABMEDYA ?
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"BACTERIA SWIM" MAY PREVENT THE SPREAD OF DISEASES

University of Minnesota researchers studied for the first time how bacteria move through fluids containing small solid particles.

For years, science fiction authors have written about the idea of using microswimmers that could perform surgeries or deliver medicines to humans. Now, a team led by University of Minnesota Twin Cities researchers discovered how bacteria swim through different complex fluids and environments, such as the human body.

Their findings could help scientists develop new treatments for bacteria-causing diseases and design bacteria-based systems for delivering drugs into the human body.

The study is published in Nature, the world's leading peer-reviewed, multidisciplinary science journal.

The University of Minnesota has a long history with swimming in fluids other than water. In 2004 Ed Cussler, then a professor in the Department of Chemical Engineering and Materials Science, compared how fast a competitive university athlete swam in water versus a thick, syrupy guar gum solution. It led to an unexpected discovery (and an IgNobel prize) that humans can swim just as fast in guar gum solutions as in water.

Almost two decades later, a multidisciplinary team at the University of Minnesota has revisited the problem, except the swimmers are now microscopic bacteria instead of university athletes. They found that bacteria swim even faster in thick solutions than in water.

"Bacterial swimming," as it's commonly known in the research community, has been studied intensively by scientists since the 1960s. Previous studies have found that bacteria swim faster in thick polymer solutions, namely fluids containing polymers, which are substances made up of long chain-like molecules. Researchers have theorized that this is because the bacteria can swim through the network formed by the chain molecules and can stretch the chains to assist their propulsion.

However, in this new study, the U of M team studied for the first time how bacteria move through solutions of small solid particles, instead of chain molecules. Despite vast differences in polymer and particle dynamics, they found that the bacteria still swam faster, suggesting that there must be a different explanation for how bacteria move through thick, complex fluids.

The U of M researchers have a possible answer. They believe that as the bacteria swim, the drag created from passing by particles allows their flagella—or the "tails" bacteria have that spin in order to propel them forward—to better align with their bodies, ultimately helping them move faster.

A bacterial cell "wobbles" in order to propel itself forward next to a micron-sized colloid particle. Video credit: Cheng Research Group, University of Minnesota

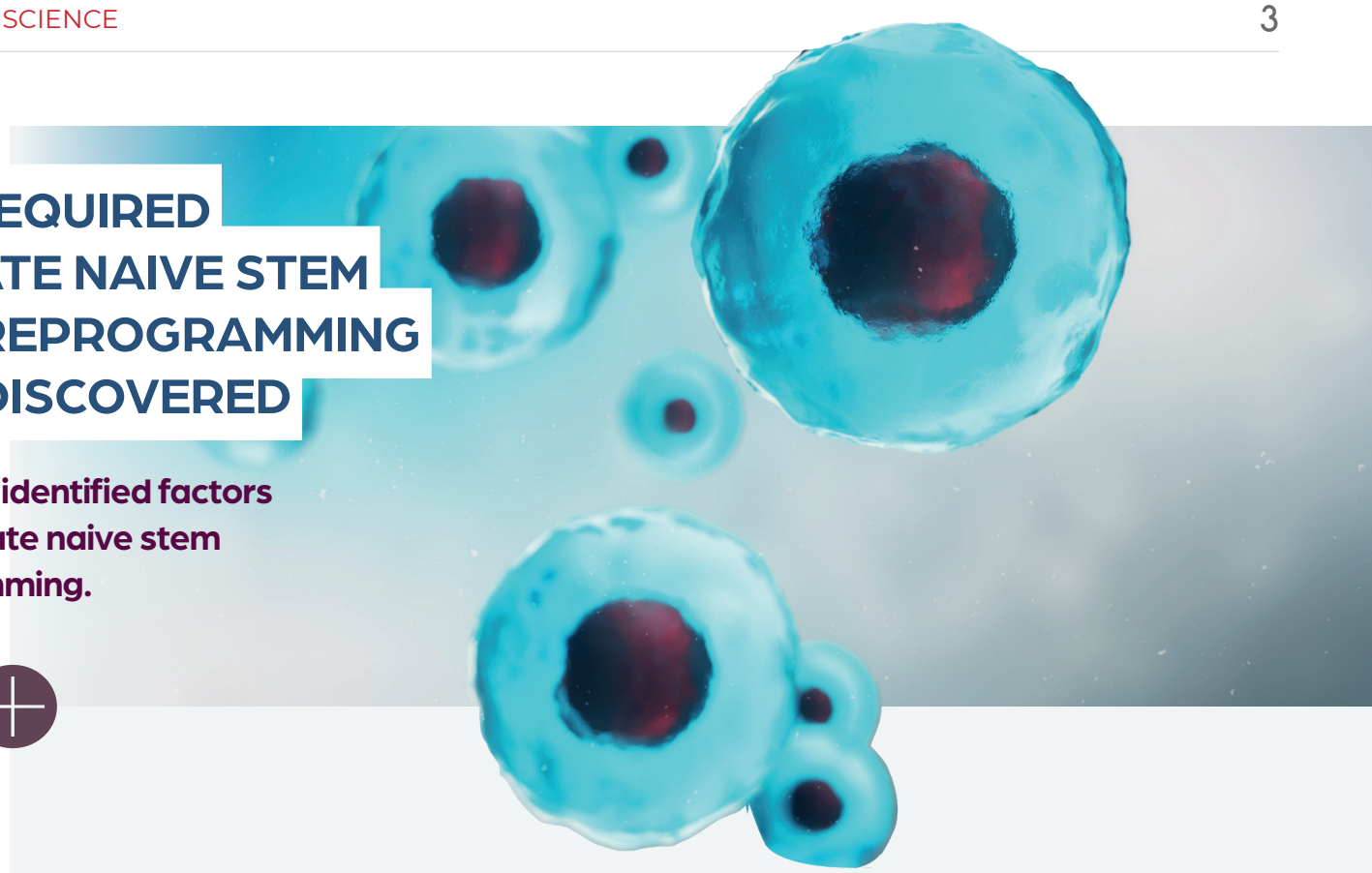
"People have been fascinated by the swimming of bacteria ever since the invention of microscopes in the 17th century, but until now, the understanding was mostly limited to simple liquids like water," explained Shashank Kamdar, lead author on the paper, a University of Minnesota chemical engineering graduate student, and a recipient of the PPG Research Fellowship. "But it is still an open question as to how bacteria are moving in real-life situations, like through soil and fluids in their own habitats."

Understanding how bacteria move through complex, viscous environments—the human body being one—can help scientists design treatments for diseases and even use bacteria as vessels for delivering medicines to humans.

"There are several mechanisms people have used to explain this phenomenon throughout the decades, but with this study, we provide a unified understanding of what happens when bacteria swim through complex solutions," said Xiang Cheng, senior author on the paper and an associate professor in the University of Minnesota Department of Chemical Engineering and Materials Science. "And it's important to understand how bacteria move in a complex environment. For example, a certain type of bacteria causes stomach ulcers. Stomach lining is a viscous environment, so studying how the bacteria move in these environments is important to understanding how the disease spreads."

"In the end, we should all learn from bacteria," Cheng added. "They keep moving forward despite opposition."

FACTORS REQUIRED TO GENERATE NAIVE STEM CELLS BY REPROGRAMMING HAS BEEN DISCOVERED



Researchers have identified factors required to generate naive stem cells by reprogramming.

Researchers from the Babraham Institute's Epigenetics research program have been able to learn more about naïve stem cell reprogramming following a genome wide functional screen. Their research, published today in Science Advances, describes the critical regulators of reprogramming and offers opportunities for a more efficient, faster way to generate human naïve pluripotent stem cells.

Human pluripotent stem cells (PSCs) are a useful tool for researchers investigating how cells specialize to make every tissue of our body. They come in two different states, primed and naïve. Both types of PSC can self-renew and differentiate into new cell types but they have distinct functions and molecular characteristics.

Group leader Peter Rugg-Gunn explained the importance of these cells: "Human PSCs in the naïve state replicate the key molecular and cellular characteristics of cells in a pre-implantation stage embryo. Importantly, when naïve PSCs are encouraged to self-organize in particular conditions, they form structures that resemble an early blastocyst stage of development. By growing these cells in the lab, we can learn about the key events that happen during human development, and they have potential uses in personalized medicine. But we need to create high-quality, stable stem cell populations to be able to conduct our experiments."

Immunofluorescent microscopy images show the different morphology of reprogrammed pluripotent stem cells (orange) and cells that were not reprogrammed (purple). Credit: Adam Bendall, PhD student, The Babraham Institute

Pluripotent stem cells are formed either from embryos or using Nobel Prize-winning methods to remove cell identity from specialized cells. The majority of reprogramming experiments generate primed PSCs, which are more developmentally advanced than naïve PSCs. Naïve PSCs can be collected directly from human pre-implantation embryos, or more commonly researchers expose primed PSCs to conditions that induces them to become naïve PSCs. Existing methods for reprogramming were inefficient and slow, preventing researchers' from quickly producing the numbers of high-quality stem cells they needed.

Adam Bendall, PhD student and a lead researcher on the study, said: "Very little was known about what genetic and epigenetic factors are required for naïve cell reprogramming, and this knowledge gap limited the design of reprogramming conditions."

The low efficiency of naïve reprogramming suggests the presence of barriers that limit cells in reaching the naïve state. Adam and his colleagues honed in on these barriers by performing a large-scale genetic screen to identify genes that hinder and help reprogramming. They were able to identify a large number of genes that have a crucial role in naïve PSC programming that had not been previously linked to the process.

The team focused on one epigenetic complex in particular, the PRC1.3 complex, that regulates gene expression without altering the underlying DNA sequence, and which they found to be essential for the formation of naïve PSCs. Without this complex, the cells undergoing reprogramming become a completely different type of cell rather than naïve PSCs. This suggests that the activity of PRC1.3 could encourage more cells to reprogram properly, in effect lowering the barrier.

After identifying factors that promote reprogramming, the researchers also looked at factors that impede reprogramming, exemplified in their study by an epigenetic protein called HDAC2. Dr. Amanda Collier, first author on the paper, explained: "Excitingly, when we inhibited one of these factors using selective chemicals, then naïve PSC reprogramming occurred more efficiently and rapidly. We're able to look at it from both sides; we can remove the barriers and introduce the factors that push cells towards state change."

Not only does this research improve scientists' ability to produce human naïve PSCs, it provides details on the molecular events that occur during the cell state transition itself, some of which are conserved in developmental regulation in human embryos.

The Rugg-Gunn lab are putting together the pieces of a bigger puzzle — the best understanding of the formation and control of naïve stem cells. Their previous research has identified molecular factors that help to maintain cells in a naïve stage. Group leader, Peter Rugg-Gunn said: "By building up our tools for manipulating pluripotent stem cells, we can spend more time asking important questions about the pre-implantation embryo. In the longer term, further improvements in working with naïve PSCs might open up the possibility for using these cells in personalized disease models or cell therapies, although this will require more research on how to differentiate naïve PSCs into specialized cell types."

AVOCADOS ARE LINKED TO LOWER RISK OF CARDIOVASCULAR DISEASE

Eating two servings of avocados a week linked to lower risk of cardiovascular disease

A 30-year study of more than 110,000 health professionals found that participants who ate at least two servings of avocado a week had a lower risk of cardiovascular disease compared to those who rarely ate avocados.

Replacing animal products like butter, cheese, or bacon with avocado was also associated with a lower risk of cardiovascular disease events.

Eating two or more servings of avocado weekly was associated with a lower risk of cardiovascular disease, and substituting avocado for certain fat-containing foods like butter, cheese, or processed meats was associated with a lower risk of cardiovascular disease events, according to new research published today in the Journal of the American Heart Association, an open access, peer-reviewed journal of the American Heart Association.

Avocados contain dietary fiber, unsaturated fats especially monounsaturated fat (healthy fats) and other favorable components that have been associated with good cardiovascular health. Clinical trials have previously found avocados have a positive impact on cardiovascular risk factors including high cholesterol.

Researchers believe this is the first, large, prospective study to support the positive association between higher avocado consumption and lower cardiovascular events, such as coronary heart disease and stroke.

"Our study provides further evidence that the intake of plant-sourced unsaturated fats can improve diet quality and is an important component in cardiovascular disease prevention," said Lorena S. Pacheco, Ph.D., Harvard T.H. Chan School of Public Health in Boston. "These are particularly notable findings since the consumption of avocados has risen steeply in the U.S. in the last 20 years, according to data from the U.S. Department of Agriculture."

For 30 years, researchers followed more than 68,780 women (ages 30 to 55 years) from the Nurses' Health Study and more than 41,700 men (ages 40 to 75 years) from the Health Professionals Follow-up Study. All study participants were free of cancer, coronary heart disease and stroke at the start of the study and living in the United States. Researchers documented 9,185 coronary heart disease events and 5,290 strokes during more than 30 years of follow-up. Researchers assessed participants' diet using food frequency questionnaires given at the beginning of the study and then every four years. They calculated avocado intake from a questionnaire item that asked about the amount consumed and frequency. One serving equaled half of an avocado or a half cup of avocado.

THE ANALYSIS FOUND:

- After considering a wide range of cardiovascular risk factors and overall diet, study participants who ate at least two servings of avocado each week had a 16% lower risk of cardiovascular disease and a 21% lower risk of coronary heart disease, compared to those who never or rarely ate avocados.
- Based on statistical modeling, replacing half a serving daily of margarine, butter, egg, yogurt, cheese or processed meats such as bacon with the same amount of avocado was associated with a 16% to 22% lower risk of cardiovascular disease events.
- Substituting half a serving a day of avocado for the equivalent amount of olive oil, nuts and other plant oils showed no additional benefit.

- No significant associations were noted in relation to stroke risk and how much avocado was eaten.

The study's results provide additional guidance for health care professionals to share. Offering the suggestion to "replace certain spreads and saturated fat-containing foods, such as cheese and processed meats, with avocado is something physicians and other health care practitioners such as registered dietitians can do when they meet with patients, especially since avocado is a well-accepted food," Pacheco said.

The study aligns with the American Heart Association's guidance to follow the Mediterranean diet – a dietary pattern focused on fruits, vegetables, grains, beans, fish and other healthy foods and plant-based fats such as olive, canola, sesame and other non-tropical oils.

"These findings are significant because a healthy dietary pattern is the cornerstone for cardiovascular health, however, it can be difficult for many Americans to achieve and adhere to healthy eating patterns," said Cheryl Anderson, Ph.D., M.P.H., FAHA, chair of the American Heart Association's Council on Epidemiology and Prevention.

"We desperately need strategies to improve intake of AHA-recommended healthy diets — such as the Mediterranean diet — that are rich in vegetables and fruits," said Anderson, who is professor and dean of the Herbert Wertheim School of Public Health and Human Longevity Science at University of California San Diego. "Although no one food is the solution to routinely eating a healthy diet, this study is evidence that avocados have possible health benefits. This is promising because it is a food item that is popular, accessible, desirable and easy to include in meals eaten by many Americans at home and in restaurants."

The study is observational, so a direct cause and effect cannot be proved. Two other limitations of the research involve data collection and the composition of the study population. The study analyses may be affected by measurement errors because dietary consumption was self-reported. Participants were mostly white nurses and health care professionals, so these results may not apply to other groups.

Reference: "Avocado Consumption and Risk of Cardiovascular Disease in US Adults" by Lorena S. Pacheco, Yanping Li, Eric B. Rimm, JoAnn E. Manson, Qi Sun, Kathryn Rexrode, Frank B. Hu and Marta Guasch Ferré30 March 2022, Journal of the American Heart Association.

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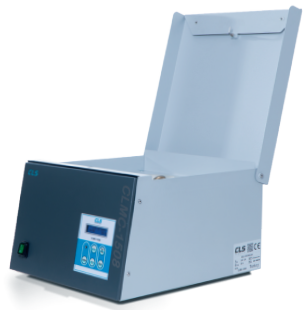

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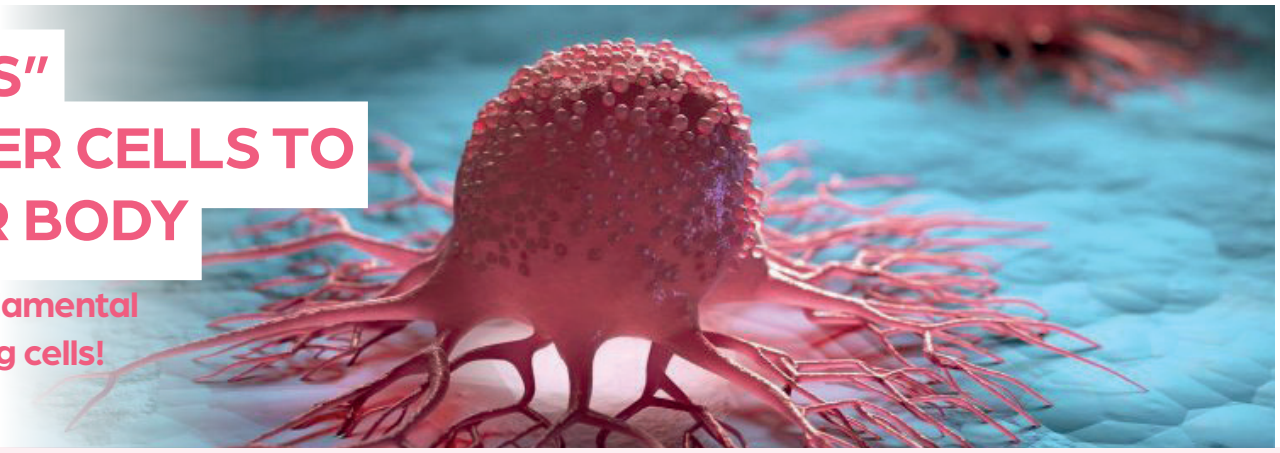


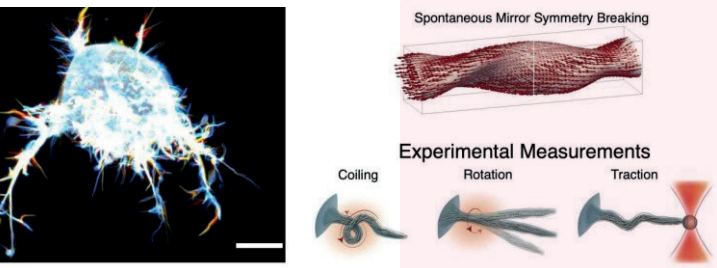
New insight on a fundamental mechanism in all living cells!

With help from the best tweezers in the world a team of researchers from the University of Copenhagen has shed new light on a fundamental mechanism in all living cells that helps them explore their surroundings and even invade tissue. Their discovery could have implications for research into cancer, neurological disorders, and much else.

Using octopus-like tentacles, a cell pushes toward its target, a bacterium, like a predator tracking down its prey. The scene could be playing out in a nature program. Instead the pursuit is being observed at the nano-scale through a microscope at the University of Copenhagen's Niels Bohr Institute. The microscope recording shows a human immune cell pursuing and then devouring a bacterium.

With their new study, a team of Danish researchers has added to the world's understanding of how cells use octopus-like tentacles called filopodia to move around in our bodies. This discovery about how cells move had never been addressed. The study is being published in the Nature Communications.

"While the cell doesn't have eyes or a sense of smell, its surface is equipped with ultra-slim filopodia that resemble entangled octopus tentacles. These filopodia help a cell move towards a bacterium, and at the same time, act as sensory feelers that identify the bacterium as a prey," explains Associate Professor Poul Martin Bendix, head of the laboratory for experimental biophysics at the Niels Bohr Institute.



The mechanical function of filopodia can be compared to a rubber band. Untwisted, a rubber band has no power. But if you twist it, it contracts. This combination of twisting and contraction helps a cell move directionally and makes the filopodia very flexible. The mechanism discovered by the Danish researchers appears to be found in all living cells. Besides cancer cells, it is also relevant to study the importance of filopodia in other types of cells, such as embryonic stem cells and brain cells, which are highly dependent on filopodia for their development.

Credit: Niels Bohr Institute / University of Copenhagen

The discovery is not that filopodia act as sensory devices – which was already well established – but rather about how they can rotate and behave mechanically, which helps a cell move, as when a cancer cell invades new tissue.

"Obviously, our results are of interest to cancer researchers. Cancer cells are noted for their being highly invasive and it is reasonable to believe that they are especially dependent on the efficacy of their filopodia, in terms of examining their surroundings and facilitating their spread. So, it's conceivable that by finding ways of inhibiting the filopodia of cancer cells, cancer growth can be stalled," explains Associate Professor Poul Martin Bendix.

For this reason, researchers from the Danish Cancer Society Research Center are a part of the team behind the discovery. Among other things, the cancer researchers are interested in whether switching off the production of certain proteins can inhibit the transport mechanisms which are important for the filopodia of cancer cells.

THE CELL'S ENGINE

According to Poul Martin Bendix, the mechanical function of filopodia can be compared to a rubber band. Untwisted, a rubber band has no power. But if you twist it, it contracts. This combination of twisting and contraction helps a cell move directionally and makes the filopodia very flexible.

"They're able to bend — twist, if you will — in a way that allows them to explore the entire space around the cell, and they can even penetrate tissues in their environment," says lead author, Natascha Leijnse.

The mechanism discovered by the Danish researchers appears to be found in all living cells. Besides cancer cells, it is also relevant to study the importance of filopodia in other types of cells, such as embryonic stem cells and brain cells, which are highly dependent on filopodia for their development.

STUDYING CELLS WITH THE BEST TWEEZERS IN THE WORLD

The project involved interdisciplinary collaboration at the Niels Bohr Institute, where Associate Professor Amin Doostmohammadi, who heads a research group that simulates biologically active materials, contributed with the modeling of filopodia behavior.

"It is very interesting that Amin Doostmohammadi could simulate the mechanical movements we witnessed through the microscope, completely independent of chemical and biological details," explains Poul Martin Bendix.

The main reason that the team succeeded in being the first to describe the mechanical behavior of filopodia is that NBI has unique equipment for this type of experiment, as well as skilled researchers with tremendous experience working with optical tweezers. When an object is extraordinarily small, holding onto it mechanically becomes impossible. However, it can be held and moved using a laser beam with a wavelength carefully calibrated to the object being studied. These are called optical tweezers.

"At NBI, we have some of the world's best optical tweezers for biomechanical studies. The experiments require the use of several optical tweezers and the simultaneous deployment of ultra-fine microscopy," explains Poul Martin Bendix.

Reference: "Filopodia rotate and coil by actively generating twist in their actin shaft" 28 March 2022, Nature Communications.

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RAMADAN FASTING BENEFICIAL FOR BLOOD PRESSURE

Fasting during the month of Ramadan can have beneficial effects on blood pressure independent of weight changes, fat mass and total body water.

The study sought to determine the health effects of the drastic change in lifestyle that millions of people undergo during Ramadan. The publication consisted of a systematic review of several previously conducted studies, as well as a longitudinal study conducted independently by the researchers in London.

"Although hundreds of millions of Muslims practice Ramadan fasting worldwide, the effect of this ritual on health is not adequately studied," Rami Al-Jafar, MSPH, lecturer and PhD candidate in epidemiology and biostatistics at Imperial College London, and colleagues wrote. "Blood pressure could be acutely affected by such changes in dietary intake and timing, physical activity and sleep patterns, including among individuals with hypertension. Studies on the effect of Ramadan fasting on blood pressure, however, are inconclusive."

LORANS STUDY

The London Ramadan Study (LORANS) assessed the systolic and diastolic BP of 85 participants from April 25 to June 16, 2019, before and immediately after Ramadan. The mean age of the participants was 46 years and 53% were men. The fasting time was 15.5 hours per day, and BP was calculated three times in 30-second intervals at each of the two visits (before and after Ramadan), with the researchers calculating the average of the three measurements. The researchers measured fat percentage/mass, fat free mass and total body water for each participant and had participants fill out questionnaires to determine basic lifestyle choices.

Results of LORANS found that systolic BP after Ramadan fasting was lower by 7.29 mm Hg (95% CI, −4.74 to −9.84) and diastolic BP was lower by 3.42 mm Hg (95% CI, −1.73 to −5.09).

SYSTEMATIC REVIEW

The researchers then conducted a systematic review and meta-analysis of 32 additional studies from around the world to investigate Ramadan's effects on systolic and diastolic BP outside of London. Among the cohort of 3,213 participants, 23.3% were healthy, 55.5% had type 2 diabetes, 3.5% had hypertension and 19.1% had chronic kidney disease.

"Although there were some previous reviews on this topic, each of them targeted studies on either healthy individuals or a specific disease group," Al-Jafar and colleagues wrote. "Our meta-analysis covered studies on healthy and nonhealthy individuals and included subgroup analysis. We included our own study (LORANS) in which we recruited a multicultural community-based sample."

Among participants in the systematic review, systolic BP was lower by 3.19 mm Hg (95% CI, −4.43 to −1.96; I2 = 48%) and diastolic BP by 2.26 mm Hg (95% CI, −3.19 to −1.34; I2 = 66%) after Ramadan.

The body undergoes a metabolic switch that starts between 8 and 12 hours of fasting, when the body switches from glucose to ketones for producing energy, resulting in an insulin drop that lowers BP, the researchers wrote.

According to the study, lower BP was observed largely in the healthy, hypertension and diabetes groups but not in patients with chronic kidney disease.

"Ramadan fasting appears to have a beneficial effect on BP independent of weight, total body water and fat mass," Al-Jafar and colleagues wrote.

GREY MATTER VOLUME FROM BRAIN MRI COULD INFORM TREATMENT DECISIONS FOR MENTAL HEALTH DISORDERS

The brain structure of patients with recent onset psychosis and depression can offer important biological insights into these illnesses and how they might develop.

Researchers at the University of Birmingham show that by examining structural MRI scans of the brain, it's possible to identify patients most susceptible to poor outcomes.

By identifying these patients in the early stages of their illness, clinicians will be able to offer more targeted and effective treatments.

"Currently, the way we diagnose most mental health disorders is based on a patient's history, symptoms, and clinical observations, rather than on biological information," says lead author Paris Alexandros Lalousis. "That means patients might have similar underlying biological mechanisms in their illness, but different diagnoses. By understanding those mechanisms more fully, we can give clinicians better tools to use in planning treatments."

In the study, the researchers used data from around 300 patients with recent onset psychosis and recent onset depression taking part in the PRONIA study. PRONIA is a European Union-funded cohort study investigating prognostic tools for psychoses which is taking place across seven European research centers including Birmingham.

The researchers used a machine learning algorithm to assess data from patients' brain scans and sort these into groups, or clusters. Two clusters were identified based on the scans, each of which contained both patients with psychosis and patients with depression. Each cluster revealed distinctive characteristics which related strongly to their likelihood of recovery.

In the first cluster, lower volumes of grey matter – the darker tissue inside the brain involved in muscle control and functions such as memory, emotions, and decision-making – were associated with patients who went on to have poorer outcomes. In the second group, in contrast, higher levels of grey matter signaled patients who were more likely to recover well from their illness.

A second algorithm was then used to predict the patients' condition nine months following the initial diagnosis. The researchers found a higher level of accuracy in predicting outcomes when using the biologically based clusters compared to traditional diagnostic systems.

Evidence also showed that patients in the cluster with lower volumes of grey matter in their brain scans may have higher levels of inflammation, poorer concentration, and other cognitive impairments previously associated with depression and schizophrenia.

Finally, the team tested the clusters in other large cohort studies in Germany and the US and were able to show that the same identified clusters could be used to predict patient outcomes.

"While the PRONIA study contained people who were recently diagnosed with their illness, the other datasets we used contained people with chronic conditions," explains Lalousis. "We found that the longer the duration of illness, the more likely it was that a patient would fit into the first cluster with lower grey matter volume. That really adds to the evidence that structural MRI scans may be able to offer useful diagnostic information to help guide targeted treatment decisions."

The next step for the team is to start to validate the clusters in the clinic, gathering patient data in real time, before planning larger scale clinical trials.

Reference: "Neurobiologically Based Stratification of Recent Onset Depression and Psychosis: Identification of Two Distinct Transdiagnostic Phenotypes" by Paris Alexandros Lalousis.

"30 YEARS TIME JUMP!"

Findings could lead to targeted approach for treating aging.

Research from the Babraham Institute has developed a method to 'time jump' human skin cells by 30 years, turning back the aging clock for cells without losing their specialized function. Work by researchers in the Institute's Epigenetics research program has been able to partly restore the function of older cells, as well as rejuvenating the molecular measures of biological age. The research is published in the journal eLife and whilst at an early stage of exploration, it could revolutionize regenerative medicine.

WHAT IS REGENERATIVE MEDICINE?

As we age, our cells' ability to function declines and the genome accumulates marks of aging. Regenerative biology aims to repair or replace cells including old ones. One of the most important tools in regenerative biology is our ability to create 'induced' stem cells. The process is a result of several steps, each erasing some of the marks that make cells specialized. In theory, these stem cells have the potential to become any cell type, but scientists aren't yet able to reliably recreate the conditions to re-differentiate stem cells into all cell types.

TURNING BACK TIME

The new method, based on the Nobel Prize-winning technique scientists use to make stem cells, overcomes the problem of entirely erasing cell identity by halting reprogramming part of the way through the process. This allowed researchers to find the precise balance between reprogramming cells, making them biologically younger, while still being able to regain their specialized cell function.

In 2007, Shinya Yamanaka was the first scientist to turn normal cells, which have a specific function, into stem cells that have the special ability to develop into any cell type. The full process of stem cell reprogramming takes around 50 days using four key molecules called the Yamanaka factors. The new method, called 'maturation phase transient reprogramming', exposes cells to Yamanaka factors for just 13 days. At this point, age-related changes are removed and the cells have temporarily lost their identity. The partly reprogrammed cells were given time to grow under normal conditions, to observe whether their specific skin cell function returned. Genome analysis showed that cells had regained markers characteristic of skin cells (fibroblasts), and this was confirmed by observing collagen production in the reprogrammed cells.

AGE IS NOT JUST A NUMBER

To show that the cells had been rejuvenated, the researchers looked for changes in the hallmarks of aging. As explained by Dr. Diljeet Gill, a postdoc in Wolf Reik's lab at the Institute who conducted the work as a PhD student: "Our understanding of aging on a molecular level has progressed over the last decade, giving rise to techniques that allow researchers to measure age-related biological changes in human cells. We were able to apply this to our experiment to determine the extent of reprogramming our new method achieved."

Researchers looked at multiple measures of cellular age. The first is the epigenetic clock, where chemical tags present

throughout the genome indicate age. The second is the transcriptome, all the gene readouts produced by the cell. By these two measures, the reprogrammed cells matched the profile of cells that were 30 years younger compared to reference data sets.

The potential applications of this technique are dependent on the cells not only appearing younger, but functioning like young cells too. Fibroblasts produce collagen, a molecule found in bones, skin tendons and ligaments, helping provide structure to tissues and heal wounds. The rejuvenated fibroblasts produced more collagen proteins compared to control cells that did not undergo the reprogramming process. Fibroblasts also move into areas that need repairing. Researchers tested the partially rejuvenated cells by creating an artificial cut in a layer of cells in a dish. They found that their treated fibroblasts moved into the gap faster than older cells. This is a promising sign that one day this research could eventually be used to create cells that are better at healing wounds.

In the future, this research may also open up other therapeutic possibilities; the researchers observed that their method also had an effect on other genes linked to age-related diseases and symptoms. The APBA2 gene, associated with Alzheimer's disease, and the MAF gene with a role in the development of cataracts, both showed changes towards youthful levels of transcription.

The mechanism behind the successful transient reprogramming is not yet fully understood, and is the next piece of the puzzle to explore. The researchers speculate that key areas of the genome involved in shaping cell identity might escape the reprogramming process.

Diljeet concluded: "Our results represent a big step forward in our understanding of cell reprogramming. We have proved that cells can be rejuvenated without losing their function and that rejuvenation looks to restore some function to old cells. The fact that we also saw a reverse of aging indicators in genes associated with diseases is particularly promising for the future of this work."

Professor Wolf Reik, a group leader in the Epigenetics research program who has recently moved to lead the Altos Labs Cambridge Institute, said: "This work has very exciting implications. Eventually, we may be able to identify genes that rejuvenate without reprogramming, and specifically target those to reduce the effects of aging. This approach holds promise for valuable discoveries that could open up an amazing therapeutic horizon."

Reference: "Multi-omic rejuvenation of human cells by maturation phase transient reprogramming" 7 April 2022, eLife.

AUTISM–ASSOCIATED BRAIN DIFFERENCES CAN BE OBSERVED IN PRENATAL MRI SCANS IN THE WOMB

Study is the first to analyze prenatal MRI scans of children later diagnosed with autism.

A new study using prenatal brain scans revealed significant differences in brain structures at around 25 weeks' gestation between children who were later diagnosed with ASD and those who were not. The study adds to mounting evidence that autism begins in early development and suggests possible opportunities to identify the disorder at an earlier age.

"Earlier detection means better treatment," said Alpen Ortug, PhD, a postdoctoral research fellow at Athinoula A. Martinos Center for Biomedical Imaging, Massachusetts General Hospital, Harvard Medical School, first author of the study. "Our results suggest that an increased volume of the insular lobe may be a strong prenatal MRI biomarker that could predict the emergence of ASD later in life."

ASD, diagnosed in 1 in 68 children in the U.S., is a complex neurodevelopmental disorder that can cause challenges with communication, cognitive processing, emotional awareness and perception. The causes of ASD are unknown but both genetic and environmental factors are thought to play a role. While early treatment has been shown to improve language and cognitive abilities, current diagnostic tools can only identify the disorder around 18 months of age.

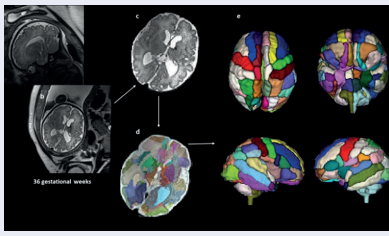
To find out if brain scans taken prenatally could help identify signs of ASD earlier, the researchers retrospectively analyzed 39 fetal MRI brain scans taken at Boston Children's Hospital. Nine of the children were later diagnosed with ASD, 20 were neurotypical and 10 did not have ASD but had other health conditions that were also observed in the children with ASD. The brain scans had been taken at about 25 weeks' gestation, on average.

After preprocessing, the researchers used an atlas-based automated anatomical labeling method to segment the brain scans and then compared the segmented brain regions between the different groups. The biggest differences were found in the brain's insular lobe, which had a significantly larger volume in the ASD group compared with the other three control groups. The insula is a region deep inside the brain that is thought to have a role in perceptual awareness, social behavior and decision-making, among other functions.

The findings align with other recent studies that have reported changes in the insular cortex in adults with autism and suggests these differences may begin in the womb. The researchers also found that the scans from children with ASD showed a significantly larger amygdala and hippocampal commissure compared with children who had other health conditions but not ASD.

"Given that many genetic and environmental factors could affect the emergence of ASD starting in the fetal stages, it is ideal to identify the earliest signature of brain abnormalities in prospective autism patients," said Ortug. "To the best of our knowledge, this is the first attempt to semi-automatically segment the brain regions in the prenatal stage in patients who are diagnosed with autism later and compare different groups of controls."

Ortug conducted the research while in a former position as a postdoctoral research fellow at Boston Children's Hospital. The study was led by Harvard Medical School Assistant Professor Emi Takahashi, PhD, whose lab recently moved from Boston Children's Hospital to the Athinoula A. Martinos Center for Biomedical Imaging at Massachusetts General Hospital.



Images representative of the process researchers used to analyze prenatal brain scans. (a–b) In-utero MRI images used in the study, (c) an MRI image after processing to mask the brain from the external tissue, (d) automatic segmentation of the brain structures, and (e) analysis of the segmented structures. The regional segmentation process was done in collaboration with Yangming Ou at Boston Children's Hospital. Credit: Alpen Ortug and Emi Takahashi, Harvard Medical School

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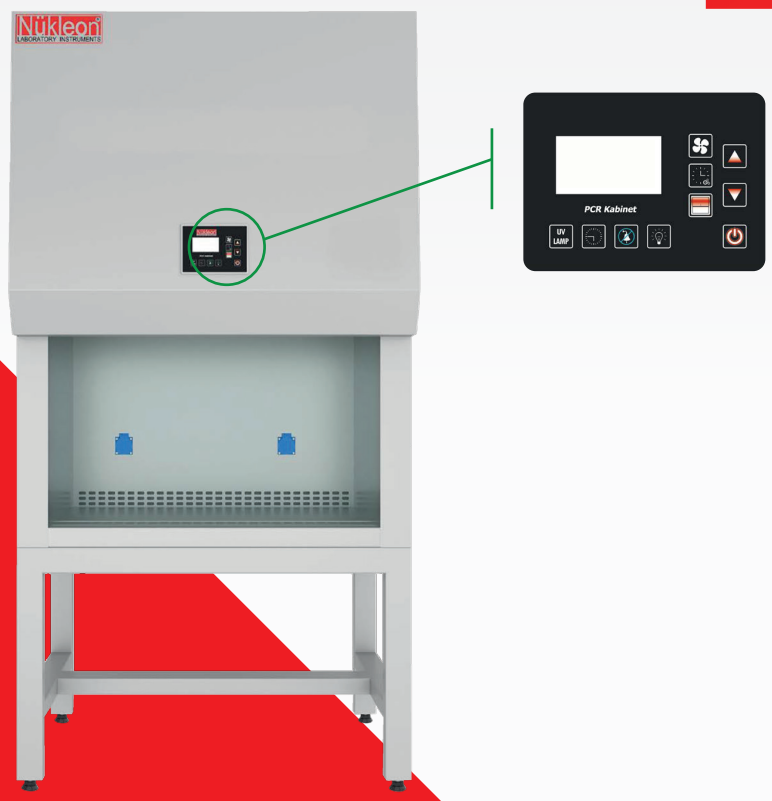

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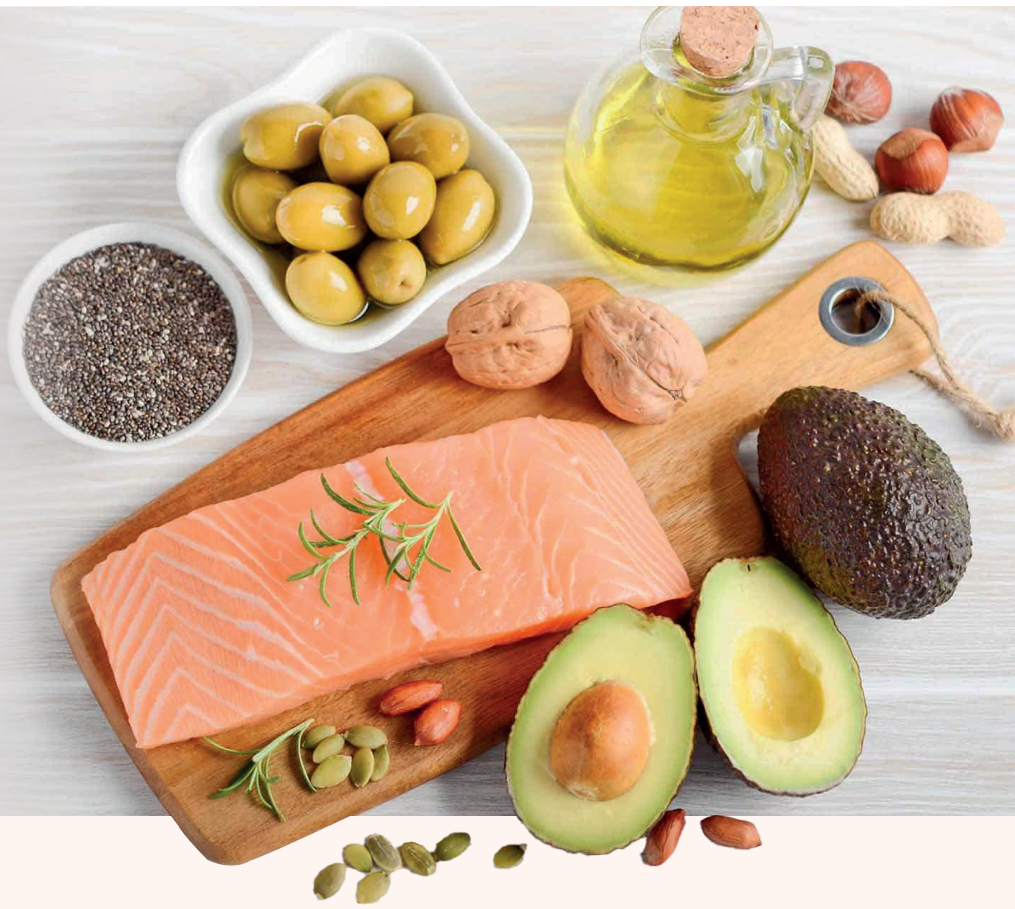
OMEGA-3 SUPPLEMENTATION COULD BOOST IMMUNOTHERAPY'S CANCER-FIGHTING POWER

Immunotherapy and anti-inflammatory therapy were more effective when mice consumed omega-3s.



Findings from a new study performed in mice suggest that omega-3 fatty acids could help immunotherapy and other treatments do a better job at fighting cancer. Immunotherapies, which stimulate the body's own immune system to attack cancer, have revolutionized cancer treatment, but they don't work for every patient.

"Dietary interventions can be powerful tools because they are relatively simple and inexpensive to implement," said Abigail Kelly, a research assistant at Harvard Medical School's Beth Israel Deaconess Medical Center in Boston. "Our findings show that omega-3 supplementation has the potential to broadly improve immunotherapy and other anti-cancer drugs in the clinical setting."

Research from various laboratories has suggested that omega-3 fatty acids can help reduce cancer risk whereas consuming too much omega-6 fatty acids can stimulate cancer. Sources of omega-3s include fish, nuts, and seeds while omegas-6s are found in meats, eggs, and other foods.

Omega-3 supplementation improves the efficacy of immunotherapy in subcutaneous murine MB49 bladder cancer tumors. Credit: Abigail Kelly, Beth Israel Deaconess Medical Center/Harvard Medical School

In the new studies, Kelly and senior author Dipak Panigraphy wanted to find out how diets supplemented with these fatty acids affected the anti-tumor activity of immune checkpoint blockade immunotherapy and an anti-inflammatory therapy that inhibits the enzyme soluble epoxide hydrolase (sEH). The immunotherapy has regulatory approval and is being used clinically while the anti-inflammatory therapy is undergoing clinical development.

The researchers used state-of-the-art mouse models of primary and metastatic tumors for the new study. They began by feeding the mice either a standard diet or a diet high in omega-3 or 6 for 10 days prior to tumor injection and for the duration of the studies. One week after the tumors were injected, mice in each diet group were started on immunotherapy, anti-inflammatory therapy, both therapies together, or no treatment.

Omega-3 supplementation in combination with inhibition of sEH improves the efficacy of immunotherapy in subcutaneous murine Lewis Lung Carcinoma tumors. Credit: Abigail Kelly, Beth Israel Deaconess Medical Center/Harvard Medical School

The researchers found that dietary omega-3 fatty acid supplementation blocked tumor growth in mice treated with immunotherapy, sEH inhibitor, or both treatments used together. In contrast, mice on the high-omega-6 diet and given immunotherapy experienced accelerated tumor growth in certain tumor types.

In mice receiving the high omega-3 diet and both cancer treatments, up to 67 percent of tumor growth was inhibited compared to mice receiving no treatment and a normal diet. This indicates possible synergistic anti-tumor activity, meaning that the combined effect may be greater than the sum of its parts.

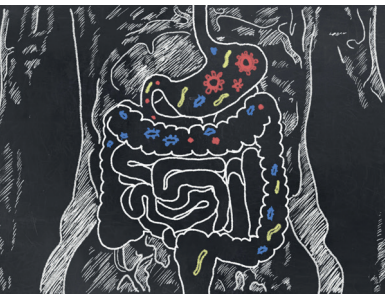
"We demonstrated, for the first time, that the combination of immunotherapy and anti-inflammatory treatment (sEHi) was more effective when mice were fed diets enriched with omega-3 fatty acids," said Kelly. "This is very promising because dietary supplementation is easy to implement for cancer patients and can be added for patients already on immunotherapy."

The researchers are now performing additional studies to determine the mechanism of action of the potentially synergistic anti-tumor activity imparted by omega-3 supplementation. They are conducting these studies with human cancer tissues and cells, human immune cells, and animal models to aid with translation to cancer patients. These new results from Kelly and colleagues may represent a new treatment approach that remains to be evaluated in humans.

GUT BACTERIAS ARE IMPORTANT FACTOR IN BLOOD PRESSURE MEDICINE RESISTANCE

Findings could lead to new options for hypertensive patients who don't respond to medication.

Almost half of the U.S. adult population has high blood pressure — or hypertension — and about 20% of these patients have treatment-resistant hypertension. The reason why some people are resistant to treatment has been a mystery, but new study results show that a certain gut bacterium may be an important factor.



"Today, doctors treat resistant hypertension by adding or substituting medications, which can contribute to overdoses, more side effects, and noncompliance," said Tao Yang, PhD, assistant professor at the University of Toledo. "A better understanding of the relationship between gut microbes and drug efficacy could lead to new treatment approaches for people who don't respond to blood pressure medication. This could include new drugs or modulating gut microbiota with probiotics, antibiotics, and other methods."

By studying the gut microbes of rats, Yang and colleagues discovered that a bacteria known as Coprococcus comes contributes to resistance to ACE inhibitors, one of the primary drug classes used to treat high blood pressure.

"Our ultimate goal is to find a link between gut microbial composition and enzymatic activity and drug response effectiveness because this will provide a foundation for applying precision medicine to treat resistant hypertension," Yang said.

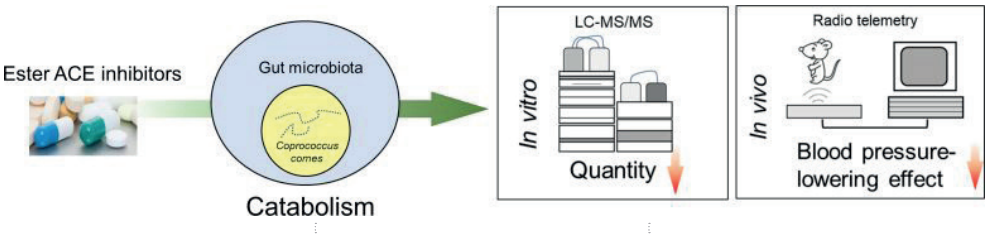
Research has shown that the microorganisms in our gut — collectively known as the gut microbiota — contain a variety of enzymes that can affect drug metabolism.

To find out if gut microbiota might play a role in resistance to blood pressure medicine, Yang and colleagues administered a single dose of the ACE inhibitor quinapril to rats with high blood pressure. They found that quinapril was more effective at lowering blood pressure in hypertensive rats with a lower gut microbiota load. When they analyzed the composition of the gut microbiota, C. comes emerged as an important player.

Through additional experiments, the researchers found that C. comes can actually break down quinapril. They also observed that giving C. comes and quinapril to hypertensive rats reduced blood pressure less than administering quinapril alone.

"We are still in the early stages of determining the interactions between gut bacteria and antihypertensive medications," said Yang. "However, our current findings suggest that the same drug may not be appropriate for everyone because each person has a unique gut microbial composition with a unique profile of enzymatic activities."

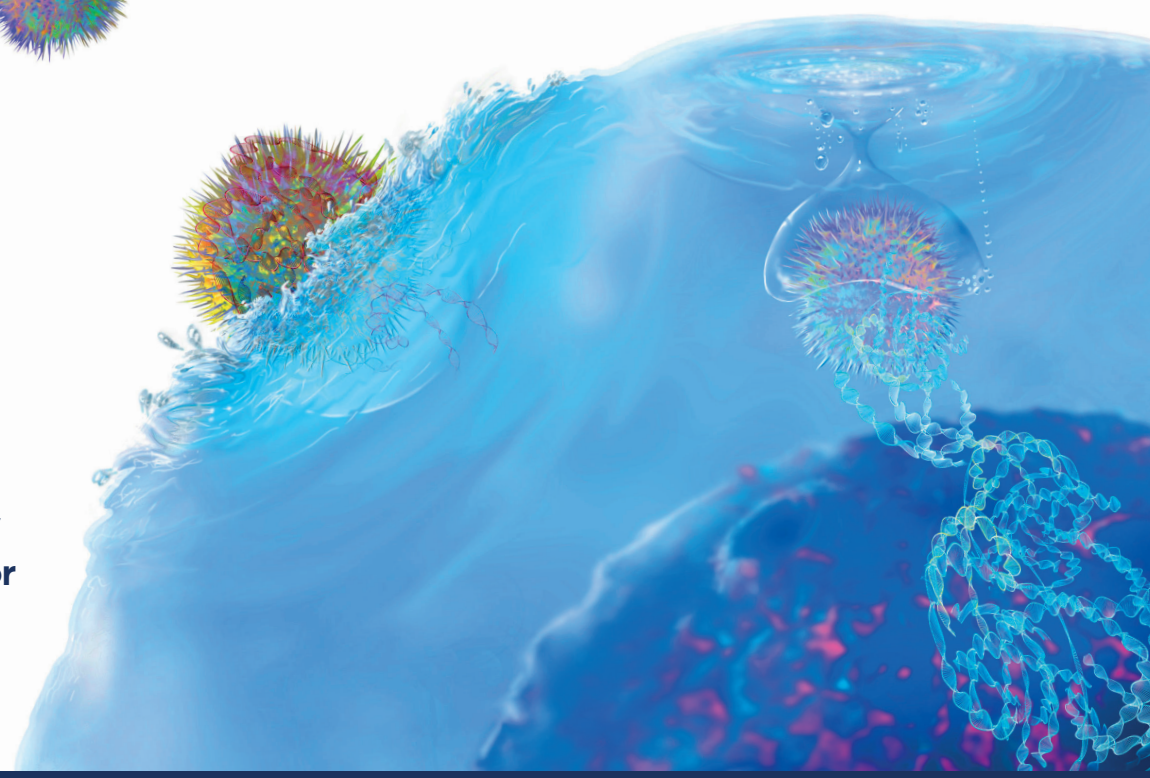
The researchers are now performing similar experiments using other types of gut bacteria and additional blood pressure medications to further explore how the gut microbiota modulates the effectiveness of antihypertensive drugs.



Researchers discovered that a bacterium known as Coprococcus comes may contribute to resistance to ACE inhibitors. They used studies involving liquid chromatography–mass spectrometry and blood pressure readings recorded via radio telemetry from a rat model of hypertension. Credit: Tao Yang, Department of Physiology and Pharmacology, University of Toledo

A NEW TECHNIQUE REVEALS HOW IMMUNE CELLS LOCATE THEIR TARGETS

MIT biological engineers have developed a simple way to identify B or T cells that interact with viral or bacterial proteins.



The human body has millions of unique B and T cells that roam the body, looking for microbial invaders. These immune cells' ability to recognize harmful microbes is critical to successfully fighting off infection.

MIT biological engineers have now devised an experimental tool that allows them to precisely pick out interactions between a particular immune cell and its target antigen. The new technique, which uses engineered viruses to present many different antigens to huge populations of immune cells, could allow large-scale screens of such interactions.

"This technique leads the way to understand immunity much closer to how the immune system itself actually works, will help researchers make sense of complex immune recognition in a variety of diseases, and could accelerate the development of more effective vaccines and immunotherapies," says Michael Birnbaum, an associate professor of biological engineering at MIT, a member of MIT's Koch Institute for Integrative Cancer Research, and the senior author of the study.

A SIMPLE SCREEN FOR A COMPLEX SYSTEM

Both B and T cells play critical roles in launching an immune response. When a T cell encounters its target, it starts proliferating to produce an army of identical cells that can attack infected cells. And B cells that encounter their target begin producing antibodies that help recruit other components of the immune system to clear the infection.

Scientists who study the immune system have several tools to help them identify specific antigen–immune cell interactions. However, these tools generally only allow for the study of a large pool of antigens exposed to one B or T cell, or a large pool of immune cells encountering a small number of antigens.

"In your body, you have millions of unique T cells, and they could recognize billions of possible antigens. All of the tools that have been developed to this point are really designed to look at one side of that diversity at a time," Birnbaum says.

The MIT team set out to design a new tool that would let them screen huge libraries of both antigens and immune cells at the same time, allowing them to pick out any specific interactions within the vast realm of possibilities.

To create a simple way to screen so many possible interactions, the researchers engineered a specialized form of a lentivirus, a type of virus that scientists often use to deliver genes because it can integrate pieces of DNA into host cells. These viruses have an envelope protein called VSV-G that can bind to receptors on the surface of many types of human cells, including immune cells, and infect them.

For this study, the researchers modified the VSV-G protein so that it cannot infect a cell on its own, instead relying on an antigen of the researchers' choosing. This modified version of VSV-G can only help the lentivirus get into a cell if the paired antigen binds to a human B or T-cell receptor that recognizes the antigen.

Once the virus enters, it integrates itself into the host cell's genome. Therefore, by sequencing the genome of all the cells in the sample, the researchers can discover both the antigen expressed by the virus that infected the cell and the sequence of the T or B-cell receptor that allowed it to enter.

"In this way, we can use viral infection itself as a way to match up and then identify antigen–immune cell parings," Birnbaum says.

INTERACTIONS IDENTIFIED

To demonstrate the accuracy of their technique, the researchers created a pool of viruses with antigens from 100 different viruses, including influenza, cytomegalovirus, and Epstein-Barr virus. They screened these viruses against about 400,000 T cells and showed that the technique could correctly pick out antigen–T-cell receptor pairings that had been previously identified.

The researchers also screened two different B-cell receptors against 43 antigens, including antigens from HIV and the spike protein of SARS-CoV-2.

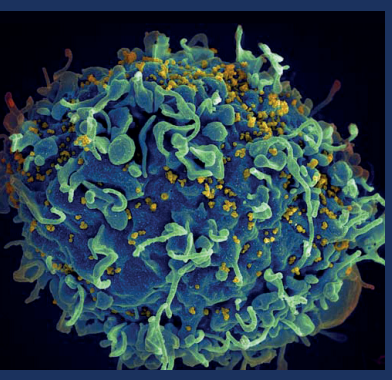
In future studies, Birnbaum hopes to screen thousands of antigens against B and T cell populations. "Our ideal would be to screen entire viruses or families of viruses, to be able to get a readout of your entire immune system in one experiment," he says.

In one study that is now ongoing, Birnbaum's lab is working with researchers at the Ragon Institute of MGH, MIT, and Harvard to study how different people's immune systems respond to viruses such

as HIV and SARS-CoV-2. Such studies could help to reveal why some people naturally fight off certain viruses better than others, and potentially lead to the development of more effective vaccines.

The researchers envision that this technology could also have other uses. Birnbaum's lab is now working on adapting the same viruses to deliver engineered genes to target cells. In that case, the viruses would carry not only a targeting molecule but also a novel gene that would be incorporated exclusively into cells that have the right target. This could offer a way to selectively deliver genes that promote cell death into cancer cells, for example.

"We built this tool to look for antigens, but there's nothing particularly special about antigens," Birnbaum says. "You could potentially use it to go into specific cells in order to do gene modifications for cell and gene therapy."



MIT biological engineers have devised a way to perform large-scale screens of how T cells such as this one recognize specific pathogens, such as the HIV viruses (yellow) show in this image. Credit: Seth Pincus, Elizabeth Fischer and Austin Athman, National Institute of Allergy and Infectious Diseases/NIH

A NEW TECHNIQUE REVEALS HOW IMMUNE CELLS LOCATE THEIR TARGETS

MIT biological engineers have developed a simple way to identify B or T cells that interact with viral or bacterial proteins.

A study in postmenopausal people suggests eating nutrient-rich prunes every day may be beneficial to bone health, reducing inflammatory factors that contribute to osteoporosis.

An estimated 13.6 million people in the U.S. over the age of 50 will develop osteoporosis—a loss of bone strength caused by reduced mineral density of the bones—by the year 2030. Osteoporosis increases the risk of fracture, especially in older adults. People who experience menopause have lower levels of estrogen, which trigger an increase in inflammation in the body, which can also contribute to bone loss.

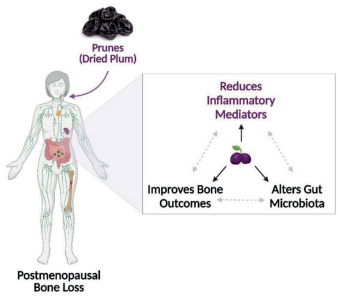
Previous research has shown that polyphenol extracts—plant compounds that act as antioxidants and reduce inflammation—in prunes promote lower levels of oxidative stress and inflammation in a type of bone cell called osteoclasts. In a new study, researchers from the Integrative and Biomedical Physiology Program and the Departments of Nutritional Sciences and Kinesiology at The Pennsylvania State University explored the effects of prunes on bone health after menopause

Postmenopausal women with a bone mineral density score that was defined as low—a marker of osteoporosis—were divided into three groups:

- One group ate 50 grams (g) of prunes (about six prunes) daily for 12 months.
- A second group ate 100 g of prunes (about 12 prunes) daily for 12 months.
- A control group ate no prunes.

The research team looked at blood samples taken from all volunteers before and after the trial and found significant reductions in inflammatory markers in both of the prune-eating groups compared to the control group.

"Our findings suggest that consumption of six to 12 prunes per day may reduce pro-inflammatory mediators that may contribute to bone loss in postmenopausal women. Thus, prunes might be a promising nutritional intervention to prevent the rise in inflammatory mediators often observed as part of the aging process," said Janhavi Damani, MS, first author of the study.



Prune study graphic. Credit: Janhavi J. Damani, MS; Nicole C.A. Strock, PhD; Mary Jane De Souza, PhD; Connie J. Rogers, PhD, MPH

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