ISPP INTERNATIONAL SOCIETY FOR PLANT PATHOLOGY

PROMOTING WORLD-WIDE PLANT HEALTH AND FOOD SECURITY

INTERNATIONAL SOCIETY FOR PLANT PATHOLOGY

ISPP NEWSLETTER

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ISPP PRESIDENT'S GREETINGS FOR 2026

YONG-HWAN LEE, ISPP PRESIDENT

Dear Members of the International Society for Plant Pathology,

The start of a new year always feels special. Many of us have small personal routines at this time of year - replying to messages from colleagues in different time zones, looking back on the past year, or making a few quiet resolutions that we hope will last beyond January. As I welcome 2026, I find myself thinking less about the calendar and more about the people who make our Society a strong and welcoming community.

I would like to begin by wishing all ISPP members and your families good health, happiness, and success in the year ahead. May 2026 bring you moments of satisfaction in your work, balance in your daily lives, and opportunities to reconnect with friends and colleagues across our global community.

ISPP is a Society built on the dedication of volunteers. Every activity, publication, and decision depend on people who generously give their time and energy because they care about our field and our community. At the start of this new year, I would like to offer my sincere thanks to all those whose work often happens quietly behind the scenes.

I am especially grateful to Dr. Serge Savary, Editor-in-Chief of Food Security, for his leadership and dedication, as well as to the Associate Editors and the many reviewers who generously contribute their expertise and time. Their commitment ensures that our journal continues to serve the community with quality, fairness, and integrity.

My thanks also go to Dr. Daniel Huberli for his continued work on the ISPP Newsletter for last 10 years. Each issue helps keep us informed and connected, and reminds us that we are part of a truly international Society.

I would like to acknowledge the members of Subject Matter Committee, who invest time and thoughtful discussion into addressing key issues and future directions within plant pathology. Their work reflects the collaborative spirit that defines ISPP.

I am equally thankful to all members of the Executive Committee and Secretariat for their guidance and steady support of the Society's activities. In addition, I wish to express my appreciation to Dr. Greg Johnson, widely recognised as the historian of ISPP, for his long-standing commitment to preserving the history and spirit of our Society.

Looking ahead, preparations for the ICPP 2028 on the Gold Coast, Australia, are progressing very well. I sincerely thank Dr. Andrew Geering, Chair of the Organising Committee, and all members of the local organising committee for their enthusiasm and hard work. Their efforts promise not only an excellent scientific meeting, but also a memorable gathering of our community.

Finally, 2026 will also be an important year as we begin the process of selecting the host location for ICPP 2032. This is a significant decision for ISPP, and information about the process will be shared with all members beginning in April 2026. I encourage your interest and participation as we move forward together.

As we step into the new year, I am reminded that the true strength of ISPP is not only science, but the people behind it - in the shared experiences, mutual support, and long-term friendships. I look forward to continuing this journey with you throughout 2026.

With my very best wishes for a happy, healthy, and rewarding New Year!

Yong-Hwan Lee

DUAL-MODE CRISPR SYSTEM ENABLES SIMULTANEOUS ON AND OFF GENE CONTROL

THE KOREA ADVANCED INSTITUTE OF SCIENCE AND TECHNOLOGY NEWS, 22 SEPTEMBER 2025

Turning genes on and off is like flipping a light switch, controlling whether genes in a cell are active. When a gene is turned on, the production of proteins or other substances is promoted; when it's turned off, production is suppressed. Korean researchers have gone beyond the limitations of existing CRISPR technology, which focused primarily on "off" functions, by developing the world's first innovative system that can simultaneously turn genes on and off, opening a new paradigm for the synthetic biology-based bio-industry.

(From left to right) Dr. Soo Young Moon, KAIST Institute of Life Science,Professor Ju Young Lee, Graduate School of Engineering Biology (Adjunct Professor of Biological Sciences),Dr. Myung Hyun Noh, Korea Research Institute of Chemical Technology (KRICT),Researcher Nan-Yeong An, Department of Biological Sciences (Photo credit: KAIST).

A joint research team led by Professor Ju Young Lee of The Korea Advanced Institute of Science and Technology (KAIST) Graduate School of Biological Engineering (Adjunct Professor of Biological Sciences) and Dr. Myung Hyun Noh of the Korea Research Institute of Chemical Technology (KRICT), an organisation under the National Research Council of Science & Technology (NST), announced that they have developed a new dual-mode CRISPR gene editing system that can simultaneously turn on and off desired genes in *E. coli*.

E. coli is a representative microorganism that is easy to experiment with and can be directly applied to industrial uses. Meanwhile, CRISPR technology is considered one of the most innovative tools in 21st-century biotechnology.

In particular, bacteria, which are the foundation of synthetic biology, have a simple structure and multiply rapidly, while also being able to produce a variety of useful substances. Therefore, gene activation in bacteria is a key technology for designing "microbial factories," and its industrial value is very high.

The core of synthetic biology is to design the genetic circuits of living organisms like programming a circuit board to perform a desired function. Just as switches are turned on and off in an electronic circuit, a technology is needed to optimize metabolic pathways by activating certain genes while suppressing others. The dual-mode gene scissors developed by the research team are the key tool that enables this precise gene regulation.

Existing CRISPR gene scissors were primarily specialized for the "off" function (repression) and were excellent at blocking gene expression, but their ability to turn genes on was very limited.

Furthermore, for CRISPR to work, a specific DNA recognition sequence (PAM, protospacer adjacent motif) is required, and the narrow range of PAM recognition in existing systems limited the scope of genes that could be controlled.

In addition, while CRISPR-based activation (CRISPRa) has been somewhat developed in eukaryotic cells (human, plant, and animal cells), there were limitations in bacteria where the "on" function did not work properly due to differences in their internal transcription regulation mechanisms.

To overcome these limitations, the research team expanded the target range to access more genes and significantly improved gene activation performance by utilising *E. coli* proteins. As a result, the gene scissors, which were previously "mainly for turning off," have evolved into a system that can simultaneously control both "on" and "off."

The performance verification results of the developed system were very impressive. In gene activation experiments, expression levels increased by up to 4.9 times, and in repression experiments, they could be suppressed by up to 83%.

Even more astonishing was the ability to control two different genes simultaneously. The team successfully activated one gene by 8.6 times while simultaneously repressing another by 90%.

To demonstrate the practicality of this technology, the research team challenged themselves to increase the production of 'violacein,' a purple pigment with anticancer properties. Through large-scale experiments on all genes of *E. coli*, they identified genes that help in violacein production.

As a result, production increased by 2.9 times when the 'rluC' gene, which helps protein production, was turned on, and by 3.0 times when the 'ftsA' gene, which helps cell division, was turned off. When both genes were controlled simultaneously, a greater synergistic effect was observed, achieving a remarkable 3.7-fold increase in production.

Dr. Myung Hyun Noh of KRICT stated, "Precise gene activation is now possible in bacteria," and "This will greatly contribute to the development of the synthetic biology-based bio-industry."

Professor Ju Young Lee said, "This research is a successful outcome of combining gene scissors with synthetic biology to significantly enhance the efficiency of microbial production platforms," and "The ability to control a complex genetic network with a single system presents a new research paradigm." He added, "This technology has also been confirmed to work in other bacterial species and can be utilised in various fields such as the production of biopharmaceuticals, chemicals, and fuels."

ARBUSCULAR MYCORRHIZAL FUNGI BOOST PLANT RESILIENCE BY REMODELLING THE PLANT-PATHOGEN MEMBRANE INTERFACE

SAINSBURY LABORATORY NEWS, 11 DECEMBER 2025

Scientists have discovered that beneficial root-dwelling fungi boost plant resilience to disease by remodelling the plant cell membrane at pathogen infection sites – offering critical new insights into how plants coordinate defences in complex natural environments. This new research, published in *Cell Reports*, reveals that the membrane interface between plant cells and invading pathogen microbes is not fixed. Instead, it can be reshaped by co-colonising symbionts, fundamentally altering how plants interact with pathogens and potentially improving resistance to disease.

More than 80% of land plants, including many crops, form partnerships with arbuscular mycorrhizal (AM) fungi to improve nutrient uptake. These mutualistic fungi are also known to help plants resist disease, but the mechanisms behind this protective effect have remained unclear.

Researchers at the Sainsbury Laboratory Cambridge University (SLCU) have now shown that when plant roots are pre-colonised by AM fungi, the plant remodels the membrane around subsequent cell-invading pathogens.

Instead of forming the usual pathogen-associated extrahaustorial membrane (EHM), the plant produces a membrane with properties similar to the membrane surrounding AM fungi, a transition that coincides with increased disease resistance.

The work highlights that AM fungi prime root defences at a cellular and molecular level by influencing the composition and identity of these specialised membrane interfaces.

The study used *Nicotiana benthamiana* (a species of tobacco), which can host the beneficial AM fungus *Funneliformis mosseae* and later be infected by the destructive filamentous oomycete *Phytophthora palmivora*.

PLANTS FORM DIFFERENT MEMBRANE INTERFACES FOR FRIENDS AND FOES

When microbes (beneficial or harmful) enter living plant cells they become enclosed within a host-derived membrane that separates them from the plant cell's cytoplasm. These membranes actively mediate nutrient exchange and molecular communication between plants and microbes, but their structure differs markedly between mutualistic and pathogenic interactions.

Mutualistic fungi form arbuscules inside roots cells encased in a periarbuscular membrane (PAM), while filamentous pathogens produce haustoria surrounded by an EHM.

FRIENDLY FUNGI FLIP THE PLAYBOOK BY CHANGING THE PLANT'S CELLULAR INTERFACE

This is the first demonstration that AM fungi can alter the EHM of a pathogen that arrives later. The study co-led by Dr Alex Guyon and Professor Sebastian Schornack, shows that AM fungi can effectively overwrite the normal distinction between mutualistic and pathogenic interfaces.

"We found that the pathogen's strategy completely fell apart in the presence of the mutualist," said Dr Guyon, who conducted the research as part of his PhD. "In these co-colonised roots, the pathogen's membrane identity was rewired and now contained a new signalling lipid and membrane-tethered proteins."

This transformation coincided with a significant reduction in pathogen colonisation.

"These findings fundamentally change our view of plant immunity and symbiosis," said Professor Sebastian Schornack, who is a group leader at SLCU. "They show that the long-established symbiotic process of enforcing a specific membrane composition may overrule the pathogen's attempts to manipulate the host cell. To fully understand this molecular mechanism, we must now look beyond single-microbe systems to understand how plants coordinate defences in the face of complex microbial communities that plants encounter in nature."

WHY THIS MATTERS

AM fungi not only improve plant nutrition, they also strengthen plant defences against pathogens. Understanding how they enhance disease resistance opens new possibilities for using beneficial fungi as natural biocontrol agents to improve crop resilience.

This research highlights a new front in the battle between plants and pathogens, showing that AM fungi can reprogramme membrane interfaces at infection sites. These insights offer promising avenues for developing future strategies to boost crop resilience by leveraging beneficial microbes.

HOW THE RESEARCH WAS DONE

To better reflect natural conditions where plants are colonised by diverse microbial communities and even individual cells can host multiple microbes, the researchers used *N. benthamiana*, a species of tobacco and model research plant that can simultaneously accommodate both AM fungi and pathogenic oomycetes.

To track changes in the host's cellular membranes in real time, the team developed and characterised phospholipid reporter lines in *N. benthamiana* by integrating phospholipid biosensors, which were previously established in *Arabidopsis thaliana* by Yvon Jaillais's group at ENS Lyon.

Researchers investigated the role of phosphoinositides (PIPs), which are a class of signalling lipids that act like molecular "identity tags" on cell membranes, dictating how the cell functions.

Under controlled laboratory conditions, the team confirmed that the two microbes established distinct membrane identities. The mutualistic AM fungus *F. mosseae* was surrounded by a membrane rich in PI4P. In contrast, the pathogenic oomycete *P. palmivora* successfully excluded key host components, including PI4P and a myristoylated protein (usually tethered to host plasma membranes) from its EHM. This exclusion may be a pathogen-driven strategy, possibly to suppress the host's immune response.

CO-COLONISATION FLIPS THE SCRIPT

The critical discovery came from simulating a more natural scenario where the plant roots were pre-colonised by the mutualist fungus before being attacked by the pathogen.

Strikingly, in roots pre-colonised by AM fungi, the pathogen's EHM composition was dramatically re-wired. PI4P and myristoylated proteins were now present around the pathogen's haustorium, making the EHM resemble the PAM of the mutualist.

This demonstrated that host membrane identity is not static and can be dynamically reshaped by co-colonisation, altering the properties of the pathogen's intracellular interface.

Crucially, this change in membrane identity coincided with enhanced resistance and reduced colonisation by *P. palmivora*.

Interestingly, neighbouring root cells, which may have recently, but not currently been colonised by AM fungi, also showed altered membrane composition at pathogen entry sites, although this effect was confined to roots and not observed in leaves.

EXPECTED EFFECTS OF A GLOBAL TRANSFORMATION OF AGRICULTURAL PEST MANAGEMENT

A paper by Niklas Möhring *et al.* titled "Expected effects of a global transformation of agricultural pest management" was published on 8 December 2025 by *Nature Communications* (Vol. 25, Article number 10901). The abstract is as follows:-

Ambitious policy goals to reduce pesticide use and risks have been established at global and regional levels. Here, we provide an assessment of the expected effects of such a global transformation of agricultural pest management. We develop a holistic assessment framework covering economic, human health, food security, social, and environmental effects and conduct a global survey with 517 experts from key disciplines and major agricultural production regions. This is an important step to identify leverage points for advancing pesticide policies and focusing future research efforts. Our results demonstrate that transforming agricultural pest management could be an important nexus for addressing multiple sustainability challenges. We find the highest expected benefits for the environmental and human health domains and the lowest for the economic and food safety domains. For regions with low income and low pesticide use, we find higher benefits and less trade-offs of the transformation than for intensive production systems in Europe and North America. Finally, a transformation is not free of costs and our results indicate that it will require a combination of new and locally adapted pest management solutions, research and support for their implementation on the ground, and an enabling policy environment.

Read paper.

COMMON MYCORRHIZAL NETWORKS FACILITATE PLANT DISEASE RESISTANCE BY ALTERING RHIZOSPHERE MICROBIOME ASSEMBLY

A paper by Xianhong Zhang *et al.* titled "Common mycorrhizal networks facilitate plant disease resistance by altering rhizosphere microbiome assembly" was published on 8 October 2025 by *Cell Host & Microbe* (Vol. 33, Issue 10, Pages 1765-1778.e7). The abstract is as follows:-

Arbuscular mycorrhizal fungi can interconnect the roots of individual plants by forming common mycorrhizal networks (CMNs). These symbiotic structures can act as conduits for interplant communication. Despite their importance, the mechanisms of signal transfer via CMNs and their implications for plant community performance remain unknown. Here, we demonstrate that CMNs act as a pathway to elicit defense responses in healthy receiver plants connected to pathogen-infected donors. Specifically, we show that donor plants infected by the phytopathogen Botrytis cinerea transfer jasmonic acid via CMNs, which then act as a chemical signal in receiver plants. This signal transfer to receiver plants induces shifts in root exudates, promoting the recruitment of specific microbial taxa (Streptomyces and Actinoplanes) that are directly linked to the suppression of B. cinerea infection. Collectively, our study reveals that CMNs act as interplant chemical communication conduits, transferring signals that contribute to plant disease resistance via modulation of the rhizosphere microbiota.

Read paper.

HOW HARMFUL BACTERIA HIJACK OUR CROPS

CHRIS WOOLSTON, WASHINGTON UNIVERSITY NEWS, 1 SEPTEMBER 2025

In a recent study, researchers at WashU have identified a tool that helps the bacteria *Pseudomonas syringae* turn a plant's fundamental biology against itself. The findings, published in the prestigious journal *mBio*, could eventually lead to new approaches to protecting crops, said co-author Barbara Kunkel, a professor of biology. "If we can understand the mechanism behind the infection, we can potentially stop it," she said.

The lead author of the study is Chia-Yun "Cynthia" Lee, a graduate student in Kunkel's lab at the time of the research. She is now a postdoctoral researcher in the Department of Biology. Maya Irvine, an undergraduate research assistant at the time of the research, is another co-author.

Plant-associated bacteria are known to exploit a crucial plant hormone called auxin, Kunkel said. The hormone, found in all land-based plants from mosses to trees, has a variety of functions, including promoting growth and, crucially, regulating responses to the environment.

The WashU team suspects *P. syringae* and other bacteria have developed a way to "listen" to the plant's auxin signaling process. When the germs notice that the plant is producing more auxin, they ramp up their attack. "The release of auxin tells the bacteria that the attack is working, so they multiply and become even more aggressive," Kunkel said. "It's a sneaky way to take advantage of and manipulate the plant's biology."

But a key question remained: How can bacteria pick up on a plant's chemical signal? To answer that question, the WashU team took a close look at the molecular and genetic machinery of *P. syringae* as it attacked thale cress (*Arabidopsis thaliana*), a plant from the mustard family used in many studies at WashU and elsewhere. "We started hunting for bacterial genes that could be involved in sensing auxin, and we found a good candidate," Lee said.

The team identified a particular protein called PmeR that seemed to fit the bill. Not only could it detect auxin — or, more specifically, a separate compound associated with auxin — it can also activate certain genes in bacteria that make the germs more aggressive and virulent. "Once bacteria sense indirectly that the auxin is there, they change their gene expression to set themselves up to better survive inside the plant," Kunkel said.

This insight into the complicated crosstalk between plants and their bacterial attackers could eventually lead to new approaches to protecting crops, Kunkel said. There's no obvious way to target the PmeR protein in wild bacteria, and it's certainly not possible to block or remove the vitally important hormone auxin from tomatoes or any other plant, but there may be another possible approach.

Kunkel's group is working with Joe Jez, the Spencer T. Olin Professor in Biology, to see if it might be possible to make pathogenic bacteria "blind" to auxin. "If we understood more about how bacteria sense auxin, we could potentially develop a compound that mimics auxin or auxin-related molecules and confuses the bacteria and blocks this system that makes them virulent," Kunkel said. "Perhaps we could spray fields with this compound."

Before any such compound could become a reality, researchers would need to better understand the physical structure of the molecules involved. "That's where Joe comes in," Kunkel says. "He has the tools and the insight to figure out even the most complicated structures."

Researchers are still a long way away from stopping P. syringae or other bacteria in their quest to find new plants to infect. But understanding the tools of the attack remains an important and exciting development, Lee said. "The communication between bacteria and plants is more complicated than we originally thought, but we're making progress."

DISRUPTING 'COMMUNICATION' WITH PLANTS COULD LIMIT SOYBEAN CYST NEMATODE INFECTIONS

IOWA STATE UNIVERSITY NEWS, 17 APRIL 2025

Targeting a newly discovered vulnerability in the signals that cyst nematodes use to infect plant roots could be a powerful method for reducing the damage the parasitic worms cause in crops such as soybeans, according to a study co-authored by an Iowa State University professor.

Researchers identified a single protein in cyst nematodes that triggers dozens of the chemical signals called effectors that the microscopic roundworms release inside roots to hijack plant cells and make themselves a home, said Thomas Baum, a distinguished professor of plant pathology, entomology and microbiology at Iowa State University.

QUICK LOOK

A new study co-authored by an Iowa State University professor opens new avenues for combatting soybean cyst nematodes, the most damaging pest for U.S. soybean crops.

Though the protein – a transcription factor that binds to genes, turning them on or off – is likely one of several that regulate effector production, researchers found that without it the nematode infection is severely reduced, according to the study recently published in Proceedings of the National Academy of Sciences. That could lead to major progress in reducing the destructiveness of soybean cyst nematodes, which cost U.S. farmers nearly 90 million bushels in production last year – by far the crop's most damaging pest.

"Now we have a validated target, a tangible molecular event involving a single transcription factor. It's a proof of concept that opens the door to various new ways of thinking about nematode management," said Baum, a co-author of the study.

A CHEMICAL CONVERSATION

For years, nematologists like Baum have been studying effectors in hope that targeting them could reduce infections by cyst nematodes, which also are prevalent in other crops such as sugar beets and potatoes. Those efforts have been a bit of a whack-a-mole game, Baum said. Nematodes produce hundreds of distinct effectors, providing redundancy and a capacity for rapid adaptation.

"You take one effector away and a nematode laughs and says, 'I've got 10 more,'" he said.

Yet effectors are the key molecular mechanism for nematode invasions in plant roots. The worms are sophisticated pathogens that after hatching in soil burrow their way into a root. Once inside, they look for a single cell to take over. When they find what they're looking for, they start not by feeding but by communicating.

"The language they use is chemicals, not words," Baum said. "Effectors deliver a message to a plant cell and it changes, turning into a cell type not usually found in a soybean root. Then all the neighboring cells change, and they fuse together to form a huge new organ whose sole function is feeding the worm."

FINDING SUGR-1

Since individual efforts to target individual effectors have little effect, researchers have had to dig deeper. Cyst nematodes produce their effectors in their esophageal glands. In prior research, Baum's lab sequenced both the whole genome of the soybean cyst nematode and the transcriptome of its gland cells, which documents RNA expression over time. Comparing the two sets of data, researchers can identify the genes responsible for nematode effectors and their production.

Baum generated the same sort of data for a close collaborator from the University of Cambridge in the United Kingdom who studies the sugar beet cyst nematode, which is nearly identical to the soybean cyst nematode. The collaborating research group, run by Sebastian Eves-van den Akker, built on the data, using a method of gene network analysis to identify a transcription factor in the nematode's glands that becomes active when effectors are produced. Their analysis showed 58 effector genes are triggered by this transcription factor, which they call the SUbventral-Gland Regulator (SUGR-1).

But what activates SUGR-1? Based on experiments by Eves-van den Akker's group, that's due to signals from the roots the researchers have termed effectostimulins. That explains why SUGR-1 activated when the Cambridge research team exposed it to root extracts from plants that are known nematode hosts.

"The most exciting thing for me about this paper is the picture it paints of a self-reinforcing cycle driving nematode infection of plants," Eves-van den Akker said. "They break plant cells, sense some signals released and respond in a way which increases their ability to break host cells."

HOW IT COULD WORK IN FIELDS

Currently, crop rotation and cultivars bred to have some nematode resistance are the main control measures for managing soybean cyst nematodes. The improved understanding of how nematodes trick plants into becoming willing hosts should eventually expand that toolbox, Baum said.

Breeding companies could develop soybean varieties genetically engineered to produce RNA that once consumed by nematodes, prevents SUGR-1 from activating. Blocking effectostimulins with chemical treatments, gene editing or breeding is another option. Methods wouldn't need to be comprehensive, as merely reducing virulence would be significant.

"With nematodes, you don't necessarily have to kill off every worm. If I reduce infection by 40%, that's a big deal. It would make a real dent in crop damages," Baum said.

The findings also hold potential for veterinary medicine and human medical treatment, as different types of nematodes are damaging parasites in animals.

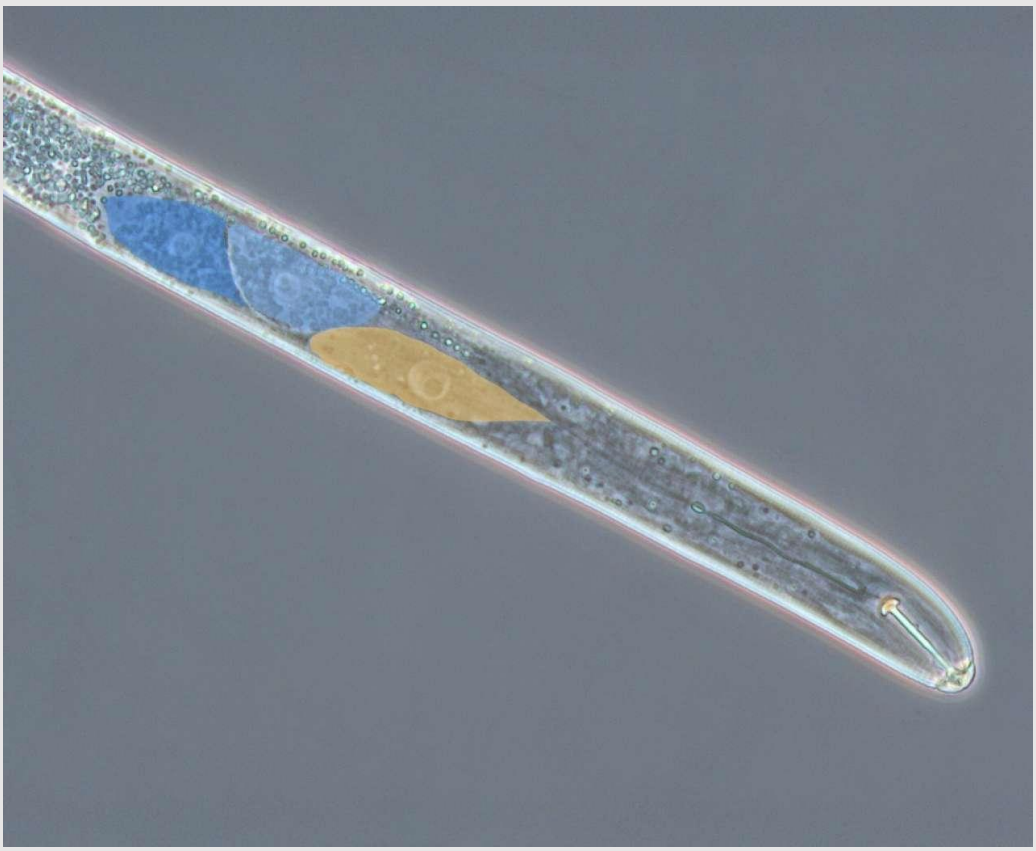
'JUST THE BEGINNING'

Baum and Eves-van den Akker plan to continue working together on cyst nematode research, as it has been a fruitful partnership for years, Baum said.

"If you were to draw up the way you want to interact with a scientific collaborator, it would be just like this. You build on each other and freely share to make advances that help both camps," he said.

Baum said he expects future studies will uncover more about the genetic and molecular mechanics of effector production, which should offer additional ways to disrupt the crucial signaling between cyst nematodes and their plant hosts.

"SUGR-1 is just the first one that jumped off the page. But we know there are others. This is only the beginning. There will be many targets," he said.



A photo of a cyst nematode's head viewed through a high-power microscope shows its esophageal glands, which produce effectors it uses to infect host plants. The image was digitally edited to highlight the glands. The subventral glands responsible for SUGR-1 are colored blue (Photo illustration credit: Tom Maier and Thomas Baum/Iowa State University).

BACTERIA REWIRE DIGESTIVE SYSTEMS TO TURN PLANT WASTE INTO POWER

AMANDA MORRIS, NORTHWESTERN UNIVERSITY NEWS, 2 SEPTEMBER 2025

For years, scientists have marveled at bacteria's ability to digest the seemingly indigestible, including carbon from lignin, the tough, woody material that gives plants their rigidity.

Now, a new Northwestern University study shows that *Pseudomonas putida*, a common soil bacterium, completely reorganizes its metabolism to thrive on these complex carbons. By slowing down some metabolic pathways while accelerating others, the bacterium manages to extract energy from lignin without exhausting itself.

The findings could have implications for the biomanufacturing industry, which has long sought to harness Pseudomonas putida to break down lignin and upcycle it into biofuels, plastics and other useful chemicals. The new information could help researchers build efficient and productive microbial factories.

The study, published in *Communications Biology*, marks the first quantitative blueprint of how bacteria coordinate carbon metabolism and energy production during digestion of lignin carbons.

"Lignin is an abundant, renewable and sustainable source of carbon that could potentially provide an alternative to petroleum in the production plastics and valuable chemicals," said Northwestern's Ludmilla Aristilde, who led the study. "Certain microbes naturally have an ability to make precursors to valuable chemicals that are lignin-based rather than petroleum-based. But if we want to take advantage of that natural ability to develop new biological platforms, we first need to know how it works. Now, we finally have a roadmap."

An expert in the dynamics of organics in environmental processes, Aristilde is a professor of civil and environmental engineering at Northwestern's McCormick School of Engineering and a member of the Center for Synthetic Biology, the International Institute of Nanotechnology and the Paula M. Trienens Institute for Sustainability and Energy.

NOTORIOUSLY TOUGH TO DIGEST

After cellulose, lignin is the second most abundant biopolymer on Earth. When broken down, it produces a mix of chemical compounds, including phenolic acids, which could be used as renewable feedstocks for valuable chemicals. But scientists have struggled to understand how bacteria manage to feed on these complex compounds.

These complex compounds are made up of a ring of six carbons with chains of carbons attached to the ring. Few organisms can process these compounds efficiently. In other words, it simply takes too much energy to digest.

"Before we eat food, we have to shop for it, cook it and eventually chew it up," Aristilde explains. "That whole process uses energy but consuming the food also gives us energy. There is a balance between the energy we exert to make the food versus the energy we derive from the food. It's the same for soil microbes."

SHIFTING FROM A MAJOR HIGHWAY TO BACK ROADS

To probe how bacteria strike this balance, Aristilde and her team grew *Pseudomonas putida* on four common, lignin-derived compounds. Then, they used a suite of "multi-omics" tools — including proteomics, metabolomics and advanced carbon-tracing techniques — to map exactly how the bacteria move carbon through their metabolism.

Aristilde likened this metabolic network to a collection of roads in a busy urban area.

"We wanted to see what happens on every street at very high resolution," she said. "We wanted to know where every 'stoplight' and 'traffic jam' might occur. That allowed us to determine which pathways are important to balance the energy in a way that is optimal for the cell."

The team discovered that, when faced with lignin, the bacterium rewires its metabolism into a high-energy mode. It ramps up the level of enzymes for certain metabolic reactions — sometimes by hundreds- to thousands-fold — to reroute digestive pathways, shifting carbon away from the "main highway" to backup metabolic "roads" to avoid bottlenecks. Due to this metabolic remodeling, the bacteria produced six times more ATP — a molecule that provides energy — compared to when it consumes easier-to-digest compounds.

A DELICATE BALANCE

While these strategies keep *Pseudomonas putida* balanced and functioning, the researchers also found the system is fragile. When they tried to relieve bottlenecks by overexpressing certain enzymes, the approach backfired, and the bacteria's metabolism fell out of its careful balance.

"Engineering strategies can often result in negative effects on the metabolism in a completely unexpected way," Aristilde said. "By speeding up the flow of one pathway, it can introduce an imbalance in energy that is detrimental to the operation of the cell."

This finding is especially important for biotechnology applications, where engineers often tweak bacteria's metabolism to produce bio-based fuels and chemicals. Aristilde says it's important to understand bacteria's natural energy rules before trying to push them to work harder. By revealing which pathways are speed bumps or energy boosters, the biotech industry can develop smarter strategies for harnessing bacteria to produce sustainable products from plant waste.

"Before this study, we could not explain exactly the coordination of carbon metabolism and energy fluxes important in the rational design of bacterial platforms for lignin carbon processing," Aristilde said. "We just had to figure it out as we went along. Now that we have an actual roadmap, we know how to navigate the network."

SWITCH ON, SWITCH OFF: THE DYNAMIC DEFENSE OF A DEADLY PLANT DISEASE

AARON CALLAHAN, BOYCE THOMPSON INSTITUTE NEWS, 10 SEPTEMBER 2025

The notorious pathogen that caused the Irish Potato Famine in the 1840s is still a major threat to potato and tomato crops worldwide. This oomycete water mold, *Phytophthora infestans*, can devastate entire fields, posing a constant threat to global food security. For decades, farmers have relied on fungicides like mefenoxam to manage the disease. But *P. infestans* is a cunning adversary.

Scientists have discovered that even strains considered sensitive to mefenoxam can rapidly develop resistance after a single exposure to a low dose of the chemical. Now, researchers at the Boyce Thompson Institute (BTI) and Universidad de los Andes have uncovered the dynamics of this defense mechanism, revealing a foe that is far more adaptable than previously thought.

"We usually think of resistance as a genetic change that happens over generations," says Silvia Restrepo, co-lead author of the study and president of BTI. "What we're seeing here is different. It's a temporary survival strategy, a kind of biological toggle switch that the pathogen can activate in the face of a threat and deactivate when the danger passes."

To understand how this switch works, the researchers conducted a series of elegant experiments, as reported in Plant Disease. They took sensitive strains of *P. infestans* and exposed them to a low dose of mefenoxam. As suspected, this single encounter was enough to "flip the switch," allowing the pathogen to survive and grow even when later moved to a dish with a very high, normally lethal, concentration of the fungicide.

Once the resistance was activated, it was robust. Repeatedly exposing the newly resistant pathogen to high levels of the fungicide didn't make it any stronger or weaker; the switch was simply "on". However, the researchers noted a crucial detail: while the pathogen could now grow in the presence of the fungicide, it didn't gain a reproductive advantage. It wasn't making significantly more spores (the tiny structures it uses to spread). It was surviving, not thriving.

The most remarkable discovery came when the researchers moved the resistant pathogen to a clean, fungicide-free environment. After just one transfer away from the chemical threat, it lost its resistance and became sensitive again. The switch had flipped "off." A subsequent low-dose exposure was all it took to re-activate the resistance, confirming that *P. infestans* possesses an incredibly flexible defense mechanism.

Such rapid, reversible behavior points away from slow genetic mutation and towards a more dynamic process known as an epigenetic change. Co-first author and BTI scientist Juliana González-Tobón explains, "Think of genetics as the permanent text in a cookbook, while epigenetics are like sticky notes you add or remove from the recipes. Rather than rewriting its DNA, the pathogen uses temporary 'notes' to quickly change its behavior in response to its environment."

The pathogen employs a defense mechanism known as pleiotropic drug resistance (PDR), which activates cellular pumps to eject the fungicide. However, this process requires significant energy, which likely explains why the pathogen readily abandons the resistance once it's no longer necessary.

This research sheds new light on the survival tactics of one of history's most devastating plant pathogens. The finding that resistance doesn't boost reproduction is good news for farmers, but the pathogen's ability to temporarily "hide" from fungicides complicates disease management. By unraveling the secrets of the pathogen's survival mechanisms, BTI scientists are paving the way for innovative disease management strategies, helping to protect our vital food crops and advance a more sustainable agricultural future.

CURRENT VACANCIES

There are no current vacancies.

ACKNOWLEDGEMENTS

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COMING EVENTS

Plant and Animal Genome Conference (PAG 33)
9 January – 14 January, 2026
San Diego California, USA
Website: https://intlpag.org/PAG33/

8th International Bacterial Wilt Symposium (IBWS)
22 March – 26 March, 2026
Wageningen, the Netherlands
Website: event.wur.nl/ibws2026

71st Annual Conference on Soilborne Plant Pathogens and the 56th California Nematology Workshop
24 March – 26 March, 2026
Kearney Agriculture Research and Extension Center in Parlier, CA, USA
Website: soilborneplantpathogens.org

21st Reinhardsbrunn Symposium 2026 – Modern Fungicides and Antifungal Compounds
19 April – 23 April, 2026
Friedrichroda, Germany
Website: https://reinhardsbrunn-symposium.de/de/

VIII International Symposium on Postharvest Pathology
18 May – 22 May, 2026
Ullensvang, Norway
Website:
https://nibio.pameldingssystem.no/isphpp2026#/contact-2228

36th Symposium of the European Society of Nematologists
1 June – 5 June, 2026
Egmond aan Zee, The Netherlands
Website: www.esn2026.nl/home

25th Annual Fusarium Laboratory Workshop
21 June – 26 June, 2026
Manhattan, Kansas, USA
Contact: John Leslie jfl@ksu.edu

Plant Health 2026
1 August – 4 August, 2026
Providence, Rhode Island, USA
Website:
www.apsnet.org/meetings/annual/PH2026/Pages/default.aspx

Plant Pathology 2026
8 September – 10 September, 2026
John Innes Centre Conference Centre, Norwich, UK
Website: Not yet available

13th Australasian Soilborne Diseases Symposium
14 September – 18 September, 2026
Melbourne, Australia
Website: www.asds-apps.com

International Plant Protection Congress
Dates not announced yet, 2027
Christchurch, New Zealand
Website: www.plantprotection.org

13th International Congress of Plant Pathology 2028
19 August – 25 August, 2028
Gold Coast, Queensland, Australia
Website: www.icpp2028.org


ICPP2028
13th International Congress of Plant Pathology
19-25 August, Gold Coast Convention & Exhibition Centre, Queensland, Australia

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WWW.ISPPWEB.ORG

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