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Editorial

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Mini review section – Fungal infections are increasingly recognized as a worldwide threat to human health. About 1.7 billion people worldwide suffer from a fungal infection, most of which are superficial infections of the skin and mucosa. Candida species are the predominant cause of nosocomial fungal infections and are the fourth leading cause of all hospital-acquired infections. Annually, there are approximately 400,000 bloodstream infections caused by Candida species globally, with mortality rates exceeding 40%.

Infections caused by *Candida. auris* have become a global threat due to its rapid emergence worldwide and multidrug resistance properties, with a crude mortality rate of 30-60%.

Current Trends section – Key elements for preventing infection in healthcare environments include hand hygiene, environmental cleanliness, isolation and barrier precautions, surveillance and antimicrobial stewardship. Individuals colonised with microorganisms can contaminate their environment, whereupon these microorganisms can be transferred to other sites, most commonly by peoples' hands. Microorganisms acquired from these sites may then be transferred to other patients.

In Profile Scientist – Robert Heinrich Hermann Koch was born on **December 11, 1843**, in Clausthal, a small mining town in the Kingdom of Hanover (now part of Germany). From a young age, Koch showed an extraordinary curiosity about nature and science. He excelled at school and pursued medicine at the **University of Göttingen**, where he studied under the guidance of the renowned anatomist **Jakob Henle**, one of the early proponents of the "germ theory" of disease. Henle's ideas—that microorganisms could cause illness—would strongly influence Koch's later work. Koch earned his medical degree in 1866, beginning his career as a physician in rural Germany.

Bug of the month – *Campylobacter* is a type of bacteria that can cause a diarrheal disease in people. Its name means "curved bacteria", as the germ typically appears in a comma or "s" shape. According to its scientific classification, it is a genus of gram-negative bacteria that is motile. The germ is common in nature and in domestic animals. It is frequently found in raw food of vegetable and animal origin. Its numbers can be very high in some foods, like raw poultry. Due to their diverse natural reservoir, some *Campylobacter* can also be detected in the air, although not at an epidemiologically significant level. The disease that some of the species of bacteria can cause is called campylobacteriosis.

Did You Know? The human body hosts trillions of microorganisms that form a vast ecosystem known as the microbiome. For years, scientists believed these microbes were confined largely to digestive processes. Today, however, mounting evidence suggests that the gut microbiome plays a profound role in conditions far beyond digestion, influencing immunity, metabolism, and even cardiovascular health. One of the most intriguing findings in this field is the discovery that certain gut bacteria produce a molecule capable of promoting plaque build-up in arteries, directly linking the microbiome to heart disease.

Best Practices – Magnesium oil, though not technically an oil but a concentrated solution of magnesium chloride and water, is gaining popularity for its transdermal application, offering a convenient way to potentially boost magnesium levels. Many users report benefits such as improved sleep quality due to magnesium's role in regulating neurotransmitters that calm the nervous system. It's also frequently used to soothe muscle aches, pains, and cramps, as magnesium is essential for muscle relaxation and proper function. This process, apart from being healthy, has no side effects. Doing these will result in a healthier, happier you.

Tickle yourself to enjoy the jokes in our **Relax Mood section**.

Our JHS team is thankful to all our readers for their ever-increasing appreciation that has served as a reward & motivation for us. Looking forward to your continuous support.

The most critically harmful fungi to humans: How the rise of *Candida. auris* was inevitable

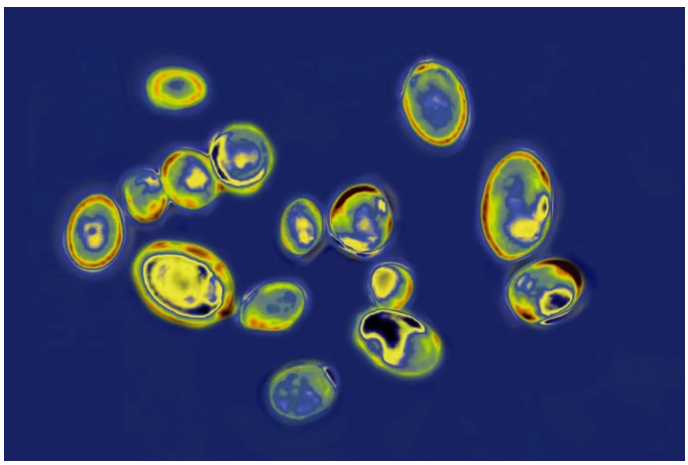
Fungal infections are increasingly recognized as a worldwide threat to human health. About 1.7 billion people worldwide suffer from a fungal infection, most of which are superficial infections of the skin and mucosa. Candida species are the predominant cause of nosocomial fungal infections and are the fourth leading cause of all hospital-acquired infections. Annually, there are approximately 400,000 bloodstream infections caused by Candida species globally, with mortality rates exceeding 40%.
Infections caused by *Candida. auris* have become a global threat due to its rapid emergence worldwide and multidrug resistance properties, with a crude mortality rate of 30-60%.
First identified in 2009, *C. auris* has been rapidly emerging to become a global risk in clinical settings and was declared an urgent health threat by the Centres for Disease Control and Prevention (CDC).

C. auris remains mostly resistant to the antifungal treatments, so once patients (and hospitals) become infected, it's nearly impossible to get rid of. A concerted global action is thus needed to successfully tackle the challenges created by this emerging fungal pathogen.

Populations at Risk
While healthy people are unlikely to develop *C. auris* infections, those with weakened immune systems are highly vulnerable. This includes:

- **Hospitalized patients**, especially those in intensive care units (ICUs).
- **Elderly individuals** in long-term care facilities.
- **Patients with invasive devices** such as central lines, feeding tubes, or ventilators.
- **People with chronic conditions** like diabetes, kidney disease, or cancer.
- **Recent surgical patients** or those receiving broad-spectrum antibiotics or antifungals.

Pathogenesis
Mechanisms of Infection and Virulence Factors
C. auris is incredibly **adhesive**. It can stick to human skin, plastic surfaces, and medical devices with remarkable tenacity. Once it latches on, it can form **biofilms** communities of microorganisms encased in a slimy matrix that protects them from antifungal agents and immune system attacks.



C. auris thrives in **warm, moist environments**, which makes the human body an ideal host—and hospital settings, with all their equipment, warm rooms, and constant patient turnover, a convenient playground. Unlike some other *Candida* species that prefer specific niches, *C. auris* appears to be more adaptable. It can colonize the skin, wounds, ears, and even respiratory tracts.
Another part of its survival strategy is **immune evasion**. *C. auris* seems to trigger a muted immune response, allowing it to slip under the radar and establish infection before the body mounts an effective defence. This fungus is remarkably good at **surviving hostile conditions**. It's been shown to tolerate high temperatures, salt concentrations, and disinfectants that would kill off many other microbes.

Resistance to Antifungal Treatments
C. auris is often resistant to three major classes of antifungal drugs:

- Azoles, such as fluconazole, which target the fungal cell membrane.
- Polyenes, like amphotericin B, a powerful but toxic option.
- Echinocandins, which inhibit fungal cell wall synthesis and are often used as last-resort treatments.

Some *C. auris* isolates are resistant to **all three classes**, creating what's known as **pan-resistant strains**. In these cases, there may be **no effective antifungal treatment available**. That's a chilling prospect, especially for patients with bloodstream infections, where timely and effective therapy is critical.

How did this resistance come about?
It's likely a combination of factors: overuse of antifungal medications, use of antifungals in agriculture, environmental pressures, and importantly the inherent genetic adaptability of *C. auris*. This fungus seems to evolve resistance more quickly than its relatives, and it's able to pass resistance traits along rapidly.

Clinical Manifestations

Common Signs and Symptoms

C. auris manifests as candidemia, or a bloodstream infection. Patients may experience fever and chills, but here's the twist: these symptoms frequently persist despite broad-spectrum antibiotic treatment. This can be a subtle but critical red flag, especially in patients with central lines or recent surgeries. When the usual antibiotics fail, clinicians should begin to suspect fungal involvement especially in high-risk environments like ICUs or long-term care facilities.

Candida auris isn't confined to the bloodstream. In some patients, particularly those who are colonized, the fungus can seed other body sites. For example, wound infections can develop in surgical sites or pressure ulcers, leading to delayed healing, localized inflammation, and tissue necrosis. In catheterized patients, *C. auris* may cause urinary tract infections that resist standard treatments, presenting as cloudy urine, discomfort, or even systemic symptoms. *C. auris* was first isolated from ear discharge, and though rare, otitis caused by the fungus can still occur. In ventilated patients, particularly those already colonized, *C. auris* may be isolated from tracheal aspirates.

The underlying problem is that *C. auris* infections are **non-specific in presentation** and **closely mimic bacterial infections**. *C. auris* often surfaces in **patients who aren't getting better on antibiotics**, especially when they've had recent surgeries, invasive devices, or long hospital stays.

Complications Associated with Infections

The complications of *C. auris* are not just medical they're systemic. Once the fungus reaches the bloodstream, it can rapidly spread to multiple organs. This can lead to septic shock, multi-organ failure, and death. The mortality rate from invasive *C. auris* infections has been reported to range from 30% to over 60%, especially when diagnosis is delayed or appropriate antifungal therapy isn't immediately available. Even when the infection is caught early, treatment is not always straightforward. Resistance to multiple antifungal drugs can complicate management and prolong the course of illness. In some cases, even with therapy, patients remain chronically colonized, which poses an ongoing risk both for themselves and for others around them.

Colonization itself may not cause symptoms, but it has serious implications. A colonized patient can unknowingly become a source of transmission within a facility, contaminating bedding, furniture, and even healthcare workers' clothing. If the patient's condition later deteriorates say, due to a new surgery or immune suppression *C. auris* can strike again, this time as a full-blown infection.

Patients who survive severe *C. auris* infections may face prolonged hospitalization, repeated courses of antifungal therapy, and even long-term complications like kidney damage or neurological deficits, depending on the organs affected. Moreover, these patients often require isolation and specialized care, which can limit access to rehabilitation services and prolong recovery.

Methods to evaluate environmental cleanliness in healthcare facilities

Key elements for preventing infection in healthcare environments include hand hygiene, environmental cleanliness, isolation and barrier precautions, surveillance and antimicrobial stewardship. Individuals colonised with microorganisms can contaminate their environment, whereupon these microorganisms can be transferred to other sites, most commonly by peoples' hands. Microorganisms acquired from these sites may then be transferred to other patients.

The potential for contaminated environmental surfaces to facilitate HAIs (Hospital Acquired Infections) depends on several factors, including:

- ✓ Ability of pathogens to remain viable on environmental surfaces
- ✓ Frequency by which organisms contaminate surface's location of reservoirs
- ✓ hand-touch frequency of surfaces
- ✓ Adequate contamination levels to present a transmission risk
- ✓ Pathogen infectivity index

Survival time for pathogens in the environment varies considerably and depends upon the characteristics of the organism. *Staphylococcus aureus* remains capable of causing infection for at least 10 days after inoculation onto a dry surface.

Unless adequate cleaning is undertaken, microorganisms in the healthcare environment may contaminate hands or be deposited onto a patient or surfaces near a patient by air currents.

Persistence of microorganisms in the environment leads to an increased risk
of infection in patients subsequently admitted to a room previously occupied by a patient colonised or infected with that organism.

Environmental contamination in conjunction with colonisation pressure is thought to encourage transmission of microorganisms. This explains the transmission of organisms such as methicillin-resistant *Staphylococcus aureus* (MRSA), *Clostridium difficile* and vancomycin-resistant enterococcus (VRE) in the healthcare environment and represents the best evidence for an environmental role in HAI (Healthcare Associated Infection).

Microorganisms are found on many surfaces in the healthcare environment. Items near the patient tend to be more heavily contaminated than more remote sites. It has been suggested that the greatest risk of acquiring pathogens arise from near-patient items such as bedrails and bedside tables, as contamination of these sites provides frequent opportunity for hands to touch and transfer organisms.

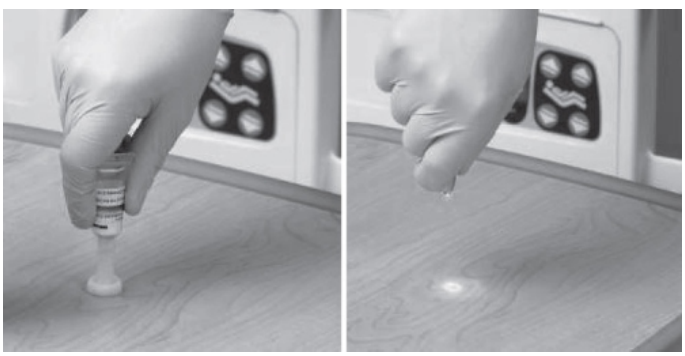
Process evaluation

Visual inspection

The primary method for assessing the cleanliness of healthcare environments is visual inspection. Visual inspection detects visible dirt, dust, rubbish, stains, soiling and moisture. Environmental cleanliness audits reliant upon visual inspection are generally undertaken by environmental cleaning staff, and the effectiveness of these is intermittently assessed by healthcare professionals such as infection control staff or trained monitoring consultants. Visual assessment was reported to perform poorly at identifying microbial load, typically passing between 17–93% more surfaces as clean than other assessment methods.

Fluorescent gel marker

This method employs an invisible transparent gel that dries on surfaces and resists dry abrasion but is easily removed with light abrasion after wetting. The gel is visible only under ultraviolet (UV) light so thoroughness of cleaning can be determined by using UV illumination for sites where the gel was applied before cleaning.



Outcome evaluation

Adenosine triphosphate bioluminescence

The measurement of ATP is the first of two methods commonly employed for sampling bioburden in the hospital environment. Sampling a surface for ATP measures the amount of organic soil present. This method uses a specialised swab to sample a standardised area. The swabs are placed in a detection device that uses the firefly enzyme and substrate luciferase and luciferin, respectively to catalyse a reaction with ATP. Light output from the reaction is proportional to the amount of ATP present and can be measured with a luminometer.

Adenosine triphosphate measurement has been used to evaluate cleanliness of food preparation surfaces for over 30 years. It is increasingly used in studies of hospital surface contamination where ATP data is gathered in addition to microbial swabbing, either to evaluate cleaning performance or to test the success of a cleaning intervention.

Adenosine triphosphate measurements provide quantification of organic material collected from a swab, including viable bacteria, but also including non-viable bacteria and organic debris such as food and liquids such as milk, blood or urine. Thus, an ATP result represents a quantitative indicator of all of these.

Microbial methods

Microbial methods for evaluating environmental cleaning have long been used to evaluate surface contamination and have been employed in hospitals to assess surface cleanliness. Colony counts, Rodac plate counts, and quantitative air sampling were all routinely used in the hospital environment, including screening of inanimate objects. Swabs, dip slides, sampling sponges and settle plates may be used to sample surfaces, and the choice of sampling method will affect microbial colony counts.

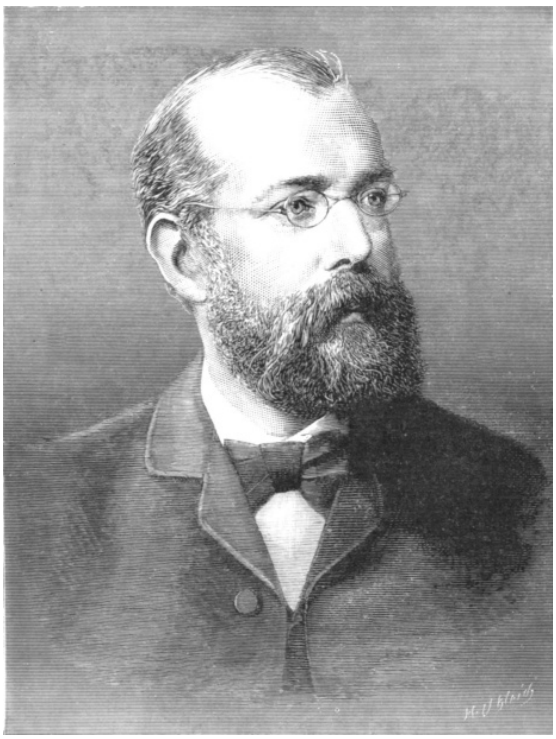
Dipslides have been evaluated as having superior sensitivity and consistency, particularly for dry surfaces, while swabs will not always accurately detect surface bioburden and may retain bacteria within the swab bud itself. Settle plates and air samplers have been used to measure airborne contamination caused by floor mopping and bed making, as well as routine cleaning.

Table 1. Summary of methodologies

	Advantages	Disadvantages[A]
	Assessing performance	
Visual inspection	Ease of use for large areas (wards, rooms)	Subjective
	Can be done with minimal training	Does not assess bio-burden
	Benchmarking possible	Does not correlate with bio-burden
	Simple and inexpensive	Can be confounded by clutter, fabric deficits and odours
Fluorescent gel	Quick	Does not assess bio-burden
	Provides immediate feedback on performance	Could be labour intensive as surfaces must be marked before cleaning and checked post cleaning
	Minimal training required	Potentially costly
	Objective	Emphasis on easily visible non-high-touch surfaces (walls, floors)
	Benchmarking possible	
	Assessing outcome	
Adenosine triphosphate bioluminescence	Quick	Expensive
	Provides immediate feedback	Low sensitivity and specificity
	Minimal training required	No current standardisation of tests
	Objective	Variable benchmarks
		Technology constantly changing
Microbial cultures	High sensitivity and specificity	Expensive
	Objective	Prolonged time for results
	Can identify screened pathogen	Requires accessible laboratory resources and trained personnel for interpreting results
	Provides quantitative data	Not supported for routine use by local and international guidelines
	May suggest or confirm environmental reservoir(s) and/or source of outbreak	Few laboratories NATA accredited to perform these tests
		Relies on standardised benchmark to assess infection risk

There are four main methods used to evaluate environmental cleanliness in healthcare facilities – ATP bioluminescence, microbiological methods, visual inspection and gel markers. Each of these methods has advantages and limitations. Methods that evaluate cleaning performance are useful in assessing adherence to cleaning protocols, whereas methods that sample bioburden provide a more relevant indication of infection risk.

Robert Koch



Early Life and Education

Robert Heinrich Hermann Koch was born on **December 11, 1843**, in Clausthal, a small mining town in the Kingdom of Hanover (now part of Germany). From a young age, Koch showed an extraordinary curiosity about nature and science. He excelled at school and pursued medicine at the **University of Göttingen**, where he studied under the guidance of the renowned anatomist **Jakob Henle**, one of the early proponents of the "germ theory" of disease. Henle's ideas—that microorganisms could cause illness—would strongly influence Koch's later work. Koch earned his medical degree in 1866, beginning his career as a physician in rural Germany.

Medical Practice and Scientific Curiosity

Koch initially worked as a country doctor. Although resources were limited, his determination and inventive spirit led him to set up a small laboratory in his home. There, he began experimenting with microscopes, culture methods, and animal models. His experience as a practicing physician exposed him to outbreaks of infectious diseases, and he became deeply interested in understanding their causes.

During the **Franco-Prussian War (1870–1871)**, Koch served as a medical officer. Witnessing epidemics of typhus and dysentery among soldiers fueled his interest in finding the microbial origins of disease.

Discovery of the Anthrax Bacillus

Koch's first major scientific breakthrough came in **1876**, while working in the provincial town of Wöllstein. He investigated **anthrax**, a deadly disease affecting cattle and sheep, which was devastating the local farming economy. Using microscopy and simple but ingenious techniques, Koch was able to isolate the **Bacillus anthracis**, the causative agent of anthrax.

He demonstrated the full disease cycle by growing the bacillus in pure culture, infecting healthy animals, and then recovering the same bacteria from their blood. This provided the first rigorous proof that a specific microorganism could cause a specific disease. His work offered powerful experimental support for the **germ theory**, helping to shift medical thinking away from vague "miasma" theories of disease.

Development of Koch's Postulates

Building on his anthrax research, Koch articulated a systematic framework to link microbes to diseases. These became known as **Koch's Postulates**, a set of four criteria:

1. The microorganism must be found in abundance in all organisms suffering from the disease, but not in healthy organisms.
2. The microorganism must be isolated from a diseased organism and grown in pure culture.
3. The cultured microorganism should cause disease when introduced into a healthy organism.
4. The microorganism must be re-isolated from the experimentally infected host and shown to be identical to the original.

Though later modified with modern microbiology's advances, Koch's Postulates remain a cornerstone of infectious disease research and have guided countless discoveries.

Work on Tuberculosis

Koch's most famous achievement came in **1882**, when he identified the bacterium responsible for **tuberculosis (TB)**, then one of the deadliest diseases worldwide. TB killed millions annually, and its cause was uncertain. Koch employed improved staining techniques and carefully examined infected tissues. On **March 24, 1882**, he announced his discovery of **Mycobacterium tuberculosis** before the Berlin Physiological Society.

This revelation had enormous medical significance. By proving TB was caused by a bacterium, Koch laid the groundwork for better diagnosis, treatment, and prevention. The day of his announcement is still commemorated annually as **World Tuberculosis Day**.

Cholera Research

In **1883**, Koch travelled to Egypt and later India to investigate outbreaks of **cholera**, a devastating diarrheal disease. There, he successfully isolated the bacterium **Vibrio cholerae**. He showed how contaminated water was a key vehicle for transmission, emphasizing the importance of sanitation and clean drinking water. This discovery directly influenced public health policies worldwide and reinforced the role of hygiene in preventing epidemics.

Contributions to Bacteriology and Laboratory Methods

Koch's impact was not limited to discovering pathogens. He and his students developed many laboratory techniques that shaped modern microbiology. Among these innovations were:

- **Solid culture media**: Koch introduced agar plates as a means to isolate pure bacterial colonies. This was a huge improvement over earlier liquid cultures.
- **Staining methods**: He refined ways to stain bacteria, making them more visible under the microscope.

- **Photomicrography**: Koch was one of the first to use photography to document microscopic images, ensuring accurate records of his findings.

His laboratory in Berlin became a training ground for a generation of microbiologists, spreading his methods across Europe, Asia, and America.

In **1905**, Robert Koch was awarded the **Nobel Prize in Physiology or Medicine** for his work on tuberculosis. By this time, he was internationally recognized as one of the greatest medical scientists of his era.

Later in his career, Koch also studied **sleeping sickness (trypanosomiasis)** in Africa and **malaria**. He contributed valuable observations about vector-borne diseases, though some of his ideas, such as the possibility of acquired immunity to tuberculosis, were debated and later revised.

Koch was known for his meticulous, methodical approach and strong commitment to experimental evidence. However, he sometimes clashed with other scientists, notably **Louis Pasteur**, the French microbiologist who had also advanced the germ theory through studies on fermentation, vaccination, and rabies. While their rivalry reflected national pride, their combined contributions built the foundations of modern bacteriology.

Legacy

Robert Koch died on **May 27, 1910**, in Baden-Baden, Germany. His influence on medicine and microbiology remains profound. Today, he is remembered not only for identifying deadly pathogens but also for pioneering a rigorous scientific method to study infectious disease.

Koch's work transformed public health, leading to improved sanitation, the development of vaccines, and the eventual creation of antibiotics. His postulates continue to guide scientists, even in the age of molecular biology and genomics. The **Robert Koch Institute** in Berlin, Germany's national public health institute, stands as a living tribute to his legacy.

Robert Koch was more than a physician—he was a trailblazer who changed the way humanity understood disease. By proving that microorganisms cause specific illnesses, he laid the foundation for modern microbiology, public health, and infectious disease medicine. His discoveries of **Bacillus anthracis, Mycobacterium tuberculosis, and Vibrio cholerae** directly saved millions of lives and inspired generations of scientists. More than a century after his death, Koch's name remains synonymous with scientific rigor, innovation, and the fight against infectious disease.

Jokes

A science teacher tells his class, "Oxygen is a must for breathing and life. It was discovered in 1773."

A blonde student responds, "Thank God I was born after 1773! Otherwise I would have died without it."

Wife: "The car isn't starting, I think there's water in the carburetor."

Husband: "How do you know? You don't even know where the carburetor is!"

Wife: "Well… the car is in the swimming pool."

Police: "Why are you driving so fast?"

Driver: "Because I'm in a hurry!"

Police: "Where are you going?"

Driver: "To get my driving license before you catch me!"

Teacher: "Why are you talking in the exam?"

Student: "Because I'm sharing knowledge."

Teacher: "Whose knowledge?"

Student: "Mostly yours, sir!"

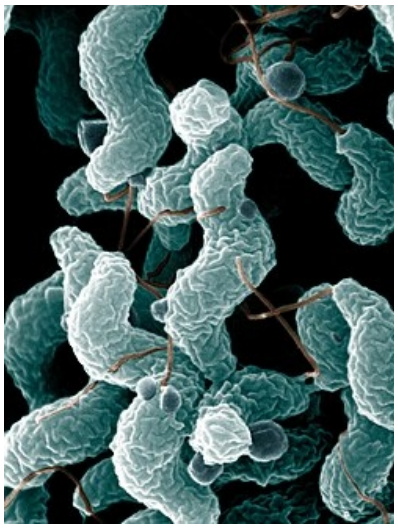
Wife: "I lost my car keys."

Husband: "Where did you see them last?"

Wife: "In your pocket… when you went out last night!"

Husband: "Oh… then we both lost them."

Campylobacter



Campylobacter is a type of bacteria that can cause a diarrheal disease in people. Its name means "curved bacteria", as the germ typically appears in a comma or "s" shape. According to its scientific classification, it is a genus of gram-negative bacteria that is motile.

The germ is common in nature and in domestic animals. It is frequently found in raw food of vegetable and animal origin. Its numbers can be very high in some foods, like raw poultry. Due to their diverse natural reservoir, some *Campylobacter* can also be detected in the air, although not at an epidemiologically significant level. The disease that some of the species of the bacteria can cause is called campylobacteriosis.

At least a dozen species of *Campylobacter* have been implicated in human disease, with *C. jejuni* (80–90%) and *C. coli* (5–10%) being the most common.*C. jejuni* is recognized as one of the main causes of bacterial foodborne disease in many developed countries. It is the number one cause of bacterial gastroenteritis in Europe, with over 246,000 cases confirmed annually. *C. jejuni* infection can also cause bacteremia in immunocompromised people, while *C. lari* is a known cause of recurrent diarrhea in children. *C. fetus* can cause spontaneous abortions in cattle and sheep, and is an opportunistic pathogen in humans.

The genomes of several *Campylobacter* species have been sequenced, beginning with *C. jejuni* in 2000. These genome studies have identified molecular markers specific to members of *Campylobacter*. *Campylobacter* spp. genomes are rather small compared to those of other gastrointestinal pathogens, with sizes ranging between 1.60 and 1.90 Mbp. A characteristic of most *Campylobacter* genomes is the presence of hypervariable regions, which can differ greatly between different strains.

Studies have investigated the genes responsible for motility in *Campylobacter* species. Some *Campylobacter* species contain two flagellin genes in tandem for motility, *flaA* and *flaB*. These genes undergo intergenic recombination, further contributing to their virulence. A single Type VI secretion system (T6SS) cluster was also predicted in approximately one-third of *Campylobacter* species, grouping into three distinct organisations and harbouring up to five *vgrG* genes.

Campylobacter can cause a gastrointestinal infection, campylobacteriosis. The incubation period is 24–72 hours after infection. This is characterized by an inflammatory, sometimes bloody diarrhea or dysentery syndrome, mostly including cramps, fever, and pain. The most common routes of transmission are fecal-oral, ingestion of contaminated food or water, and the eating of raw meat. Foods implicated in campylobacteriosis include raw or under-cooked poultry, raw dairy products, and contaminated produce.*Campylobacter* is sensitive to the stomach's normal production of hydrochloric acid: as a result, the infectious dose is relatively high, and the bacteria rarely cause illness when a person is exposed to less than 10,000 organisms. Nevertheless, people taking antacid medication (e. g. people with gastritis or stomach ulcers) are at higher risk of contracting disease from a smaller number of organisms, since this type of medication neutralizes normal gastric acid.

In humans, the sites of tissue injury include the jejunum, the ileum, and the colon. Most strains of *C. jejuni* produce cytolethal distending toxin, which inhibits cell division and impedes activation of the immune system. This helps the bacteria to evade the immune system and survive for a limited time inside intestinal cells. *Campylobacter* has, on rare occasions, been suggested to cause hemolytic uremic syndrome and thrombotic thrombocytopenic purpura, though no unequivocal case reports exist. *Campylobacter* infection is the most common trigger of Guillain–Barré syndrome. Gastrointestinal perforation is a rare complication of ileal infection.

Campylobacter has also been associated with periodontitis.

Campylobacter testing needs to be done to manage the risk of foodborne *Campylobacter* and reducing the level of foodborne *Campoboteriosis*, to protect people and to determine if a person is infected with *Campylobacter.*

In humans

Usually, detection of *Campylobacter* in humans is done by laboratory culturing a stool sample or swab of the rectum collected by a healthcare provider. Results take about 48–72 hours for preliminary results. Confirmation test and testing to determine the species of *Campylobacter* or drug sensitivities of the organism require additional time.

In livestock

Usually, detection of Campylobacter in livestock is done by laboratory culturing a faecal sample. Results take about 48–72 hours.

In meat

Usually, detection of Campylobacter in meat is done by laboratory culturing a homogenised sample. Results takes about 48–72 hours.

Treatment

The infection is usually self-limiting and, in most cases, symptomatic treatment by liquid and electrolyte replacement is sufficient to treat human infections. Symptoms typically last 5–7 days. Treatment with antibiotics has only a minor effect on the typical duration of the infection in non-complex cases, and is discouraged except in high-risk patients. Diagnosis of campylobacteriosis is made by testing a fecal specimen. Standard treatment in high-risk cases is azithromycin, a macrolide antibiotic, especially for *Campylobacter* infections in children, although other antibiotics, such as quinolones, tetracycline and other macrolides are sometimes used to treat gastrointestinal *Campylobacter* infections in adults. In case of systemic infection, other bactericidal antibiotics are used, such as ampicillin, amoxicillin/clavulanic acid, or aminoglycosides. Fluoroquinolone antibiotics, such as ciprofloxacin or levofloxacin, may no longer be effective in some cases, due to resistance. In addition to antibiotics, dehydrated patients may require intravenous fluid treatment in a hospital.

Gut bacteria make a molecule that promotes plaque build-up in arteries

The human body hosts trillions of microorganisms that form a vast ecosystem known as the microbiome. For years, scientists believed these microbes were confined largely to digestive processes. Today, however, mounting evidence suggests that the gut microbiome plays a profound role in conditions far beyond digestion, influencing immunity, metabolism, and even cardiovascular health. One of the most intriguing findings in this field is the discovery that certain gut bacteria produce a molecule capable of promoting plaque build-up in arteries, directly linking the microbiome to heart disease.

The molecule at the center of this story is **trimethylamine N-oxide (TMAO)**. Research over the past decade has established that this compound, generated through the metabolism of dietary nutrients by gut bacteria, contributes significantly to the process of atherosclerosis—the formation of fatty plaques in blood vessels that underlies many heart attacks and strokes. The connection between gut microbes, diet, and heart disease has added a new dimension to the way we understand cardiovascular risk.

The pathway leading to TMAO production begins with food. Nutrients such as choline, lecithin, and carnitine, abundant in red meat, eggs, liver, and some dairy products, serve as the raw material. Once ingested, these compounds reach the lower intestine, where certain groups of gut bacteria convert them into **trimethylamine (TMA)**. This intermediate product is then absorbed into the bloodstream and transported to the liver. There, enzymes known as **flavin-containing monooxygenases (FMOs)** oxidize TMA into TMAO, which circulates throughout the body.

Why is this molecule problematic? Studies have shown that TMAO interferes with normal cholesterol metabolism, hindering the body's ability to remove excess cholesterol from artery walls. It also promotes the formation of foam cells—cholesterol-laden immune cells that are central to plaque development. Beyond this, TMAO enhances the reactivity of platelets, making blood more prone to clotting, and stimulates inflammatory processes within blood vessels. Together, these effects create a perfect storm for arterial blockages and cardiovascular events.

The evidence linking TMAO to heart disease is compelling. Human studies have revealed that individuals with elevated TMAO levels in their blood face a higher risk of heart attack, stroke, and death, even after accounting for traditional risk factors like LDL cholesterol and hypertension. Animal studies further confirm the causal role: mice fed diets rich in choline or carnitine developed more arterial plaques, while treatment with antibiotics to suppress gut bacteria significantly reduced TMAO levels and plaque formation.

Interestingly, not everyone produces the same amount of TMAO from the same foods. This variability is strongly influenced by the composition of the gut microbiome. People whose intestinal bacteria harbor the genes for TMA lyase enzymes generate more TMA, and therefore more TMAO. Those with different microbial communities may produce little to none. Vegetarians and vegans, for instance, typically have lower baseline levels of TMAO, not only because they consume fewer animal-derived nutrients but also because their gut microbiota are less capable of converting those nutrients into TMA.

The recognition that gut microbes influence cardiovascular risk has opened new therapeutic possibilities. Dietary changes remain a cornerstone: reducing consumption of red meat, eggs, and processed animal products lowers the supply of TMAO precursors. Plant-based diets, rich in fiber, also foster microbial communities that are less inclined toward TMA production. Beyond diet, scientists are investigating targeted interventions such as probiotics to alter gut microbial balance, prebiotics to nourish beneficial species, and even specific inhibitors that block the microbial enzymes responsible for producing TMA. In parallel, pharmacological strategies aimed at reducing the liver's conversion of TMA to TMAO are under exploration.

The broader implication of this research is profound. Cardiovascular disease has traditionally been framed through the lens of cholesterol, blood pressure, and lifestyle choices such as smoking and exercise. While these remain crucial, the gut microbiome adds a new and independent factor to the equation. It suggests that future cardiovascular medicine may include not only cholesterol testing but also profiling of gut microbial activity and TMAO levels.

In conclusion, the discovery that gut bacteria contribute to plaque build-up in arteries marks a shift in our understanding of heart disease. The once-overlooked microbes in our intestines emerge as active players in the development of atherosclerosis, linking diet, microbial metabolism, and cardiovascular outcomes. As science continues to unravel these connections, strategies aimed at reshaping the gut microbiome could become a powerful tool in the fight against heart disease. The old adage "you are what you eat" now carries a new meaning: we are also what our gut bacteria make of what we eat.

6 reasons to rub magnesium oil on soles of feet 3 times a week

Magnesium oil, though not technically an oil but a concentrated solution of magnesium chloride and water, is gaining popularity for its transdermal application, offering a convenient way to potentially boost magnesium levels. Many users report benefits such as improved sleep quality due to magnesium's role in regulating neurotransmitters that calm the nervous system. It's also frequently used to soothe muscle aches, pains, and cramps, as magnesium is essential for muscle relaxation and proper function. This process, apart from being healthy, has no side effects. Doing these will result in a healthier, happier you.

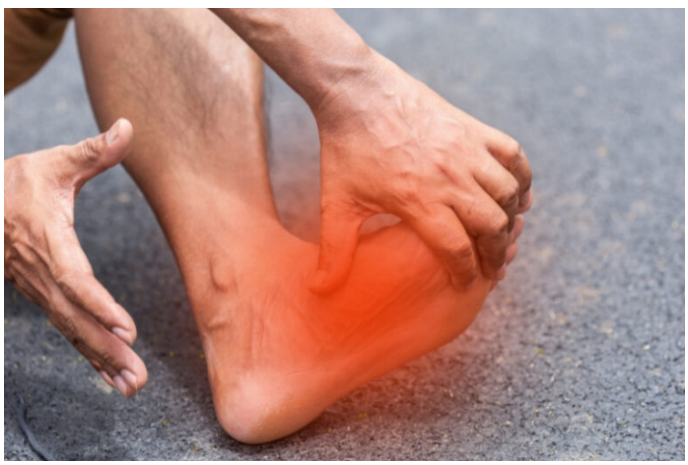
Why should people rub magnesium oil on their feet

Magnesium being an essential mineral, has an important role in over 300 bodily functions, many don't realise that a simple practice like applying magnesium oil to the soles of the feet a few times a week can lead to powerful changes in health. The soles of the feet have larger pores and thicker skin, which makes them a great spot for transdermal absorption, when magnesium oil is gently rubbed here.

1. Sleep starts to feel more restful and deeper

Research shows magnesium plays a role in regulating GABA, a neurotransmitter that calms the nervous system. When applied to the feet, magnesium oil is absorbed through the skin and enters the bloodstream, helping to ease nighttime restlessness. Within a couple of weeks of regular use, sleep quality may improve, and waking up might begin to feel more refreshing.

2. Unexplained foot cramps slowly fade away

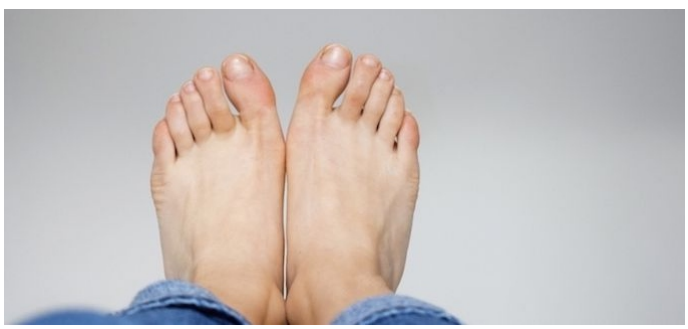


Magnesium helps muscles relax after they contract. Regularly applying magnesium oil on the feet can deliver this mineral directly to the area that often suffers the most—especially for those who stand a lot or walk extensively. Over time, the tightness or cramping, particularly in the arch or toes, may reduce significantly.

3. Stress begins to feel lighter without even noticing

Magnesium is known to influence the adrenal glands and nervous system. As magnesium levels in the body begin to stabilise with transdermal absorption, the response to stress often becomes more balanced.

4. Foot skin starts to heal and soften naturally



Magnesium has anti-inflammatory and antibacterial properties. When applied to the soles of the feet, it doesn't just nourish the inside of the body it helps from the outside too. Cracked heels, calluses, or dry patches often start to soften with regular application.

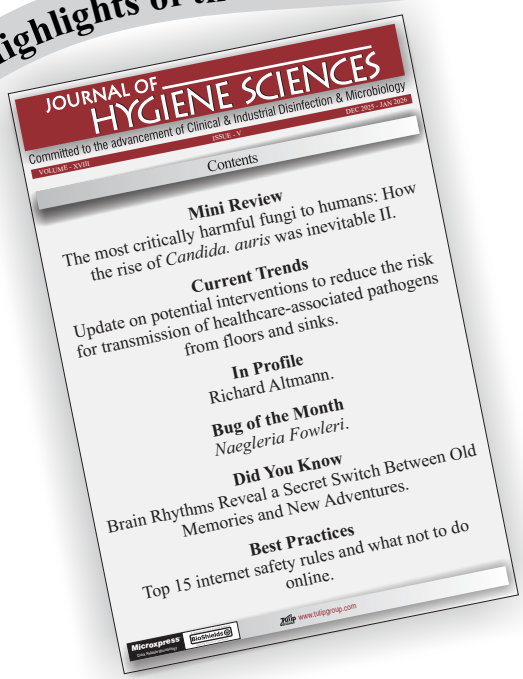
5. Digestive rhythms quietly improve

Magnesium does support digestion but through its calming effect on the nervous system and muscles, including those of the gut. Applying it to the feet doesn't directly stimulate digestion like an oral supplement might, but it encourages the parasympathetic nervous system also known as the "rest and digest" mode. This, in turn, may help with better bowel movements and reduced bloating over time.

6. Headaches and tension ease without medicine

Studies link magnesium deficiency to migraines and tension-type headaches. While applying magnesium oil on the feet doesn’t act as a painkiller, it slowly builds up magnesium levels in the body. With consistent use, some experience fewer headaches, reduced tension in the neck and shoulders, and a clearer headspace, especially when the body has been under stress or strain for long periods.

Highlights of the coming issue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