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Editorial

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Mini review section – The imbalance of microbial composition and diversity in favor of pathogenic microorganisms combined with a loss of beneficial gut microbiota taxa results from factors such as age, diet, antimicrobial administration for different infections, other underlying medical conditions, etc. Probiotics are known for their capacity to improve health by stimulating the indigenous gut microbiota, enhancing host immunity resistance to infection, helping digestion, and carrying out various other functions. The gut microbiome represents an intricate ecosystem where microorganisms and their metabolic products engage with host cells, exerting an influence on various bodily functions.

Current Trends section – An increased screentime is something that most of the people are experiencing day by day. Most of us can't even imagine going a full day without a mobile phone. From messages and video calls, from work to school assignments, and banking, our devices have become essential. But as convenient as they are, they emit electromagnetic radiation, which is a growing concern of health.

In Profile Scientist – The story of Frederick Banting is one of the most compelling narratives in the history of science, a tale of a man who possessed neither the pedigree nor the resources of a world-class researcher but who, through sheer force of will and a singular, inspired idea, managed to change the course of human history. Born on November 14, 1891, on a farm in Alliston, Ontario, Frederick Grant Banting was the youngest of five children. His upbringing was characterized by the rugged simplicity of rural life, shaped by the Methodist values of his parents, William Thompson Banting and Margaret Grant.

Bug of the month – *Trypanosoma cruzi* is a flagellated protozoan parasite of immense public health significance, serving as the etiological agent of Chagas disease, also known as American trypanosomiasis. Discovered in 1909 by the Brazilian physician and researcher Carlos Chagas, the parasite remains a major cause of morbidity and mortality across the Americas and, more recently, throughout the world due to global migration. It belongs to the order Kinetoplastida and is characterized by the presence of a kinetoplast, a specialized mitochondrial organelle containing a dense mass of circular DNA.

Did You Know? – Inflammation covertly rewires the bone marrow, enabling mutated stem cells to rise and setting the stage for future blood disease. Every moment, the bone marrow generates millions of fresh blood and immune cells. This nonstop renewal depends on a carefully balanced relationship between hematopoietic stem cells (HSCs), supportive stromal cells, and a network of immune signals.

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.

Role of probiotics in managing various human diseases, from oral pathology to cancer and gastrointestinal diseases

The imbalance of microbial composition and diversity in favor of pathogenic microorganisms combined with a loss of beneficial gut microbiota taxa results from factors such as age, diet, antimicrobial administration for different infections, other underlying medical conditions, etc.

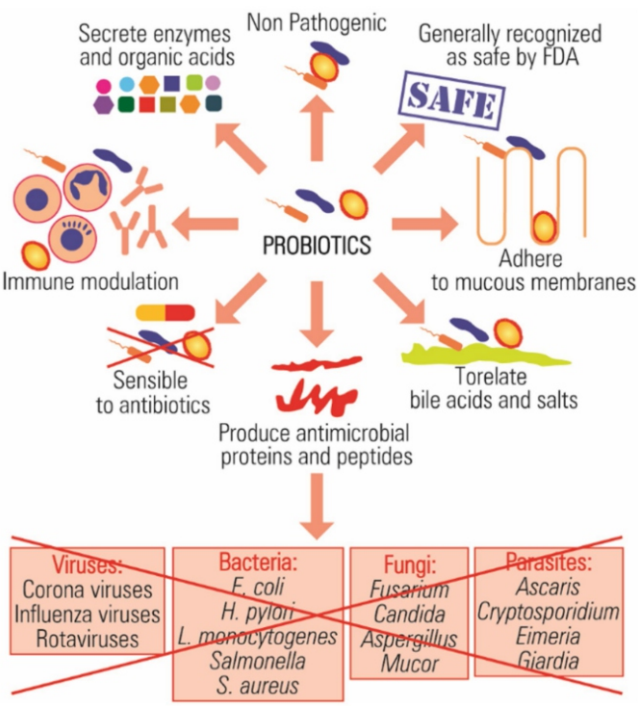
Probiotics are known for their capacity to improve health by stimulating the indigenous gut microbiota, enhancing host immunity resistance to infection, helping digestion, and carrying out various other functions.

The gut microbiome represents an intricate ecosystem where microorganisms and their metabolic products engage with host cells, exerting an influence on various bodily functions.

General characterization of probiotics

Probiotics are microorganisms that engage in a mutually beneficial relationship with the host organism, offering health advantages and performing crucial biological functions when administered in sufficient quantities.

Probiotic strains chosen for evaluation can be characterized by various vital properties, including their non-pathogenic nature, ability to withstand changes in the human gastrointestinal environment, capacity to adhere to and colonize the intestinal epithelium, antimicrobial properties, genetic and phenotypic stability, and immunomodulatory capabilities. Previous research has categorized probiotics into viable and active probiotics and viable inactive probiotics.



The most consumed probiotics in human nutrition belong to genera such as *Lactobacillus*, *Bacillus*, *Bifidobacterium*, and *Saccharomyces*. Lactic acid bacteria (LAB) are considered the main probiotic bacteria that are used as viable cells, including homofermentative lactobacilli, which are represented by three main groups, including *L. acidophilus*, *L. salivarius*, and *L. rhamnosus.* Probiotic lactic acid bacteria show significant promise as substitutes for antibiotics, serving as both preventive and curative treatments. Among producers of non-lactic acid are *B. cereus, E. coli Nissle 1917*, *Sporolactobacillus inulinus*, *Propionibacterium freudenreichii*, and *Saccharomyces cerevisiae.*

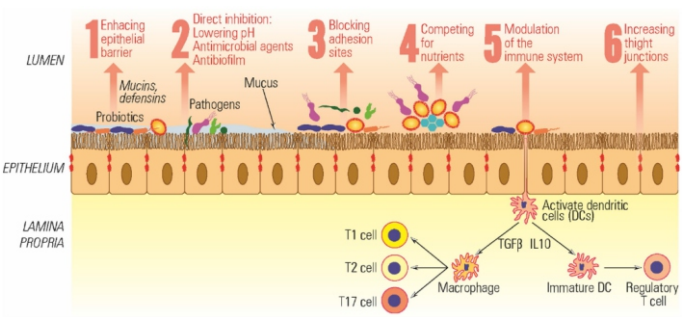
Properties and effect mechanisms of probiotics

-Effect mechanisms of probiotics

Probiotics serve a broad spectrum of health-related purposes. These health-promoting qualities are contingent on the specific probiotic strain in use, and each exerts its effects through distinct mechanisms.

Probiotics have several benefits:

(i) reduce vulnerability to infections.
(ii) reduce lactose intolerance, relieve allergic episodes and respiratory infections.
(iii) reduce serum cholesterol and blood pressure.
(iv) prevent the intestine from gastritis and diarrhoea.
(v) prevent urogenital and vaginal infections and
(vi) reduce the chances of colon cancer



-Anti-inflammatory properties

Probiotic bacteria have been found to significantly influence the immune system by releasing anti-inflammatory cytokines within the gut. They can potentially impact various types of immune cells, including dendritic cells, monocytes, natural killer (NK) cells, macrophages, lymphocytes, and epithelial cells. A key mechanism of action involves the activation of pattern recognition receptors (PRRs) found on both immune and non-immune cells.

Specific strains of *Lactobacillus* sp. have been found to influence cytokine production, while *Bifidobacterium* sp. strains have been associated with promoting immune tolerance.

Oral administration of probiotic strains like *L. plantarum*, *L. acidophilus*, *B. breve*, and *B. lactis* influence the release of proinflammatory cytokines through toll-like receptor signalling pathways.

Bifidobacterium sp. and Lactobacillus sp. play significant roles in modulating various aspects of the immune system, including

the humoral response, cell-mediated responses, and non-specific immunity.

The commensal microbiota maintains the epithelial environment's balance by stimulating the production of epithelial repair factors and regulating the immune response, safeguarding against epithelial damage. When administered orally, probiotics activate TLR signalling, producing cytokines that activate macrophages and influence intestinal epithelial cells (IEC) and immune cells within the lamina propria. This activation, in turn, stimulates regulatory T cells to release IL-10. Probiotics exhibit immunomodulatory effects that impact humoral immunity, cell-mediated immunity, and the non-specific immune response.

Among the commonly used probiotic strains are various *Lactobacillus* and *Bifidobacterium* sp., including *L. acidophilus*, *L. casei*, *L. rhamnosus*, *L. reuteri*, *L. johnsonii*, *L. gasseri*, *B. longum*, *B. bifidum*, and *B. infantis*. Several studies have highlighted the significance of *L. fermentum* and *L. gasseri* in healthy individuals, as they are responsible for inhibiting periodontal pathogens such as *Porphyromonas gingivalis*, *Prevotella intermedia*, and *Aggregatibacter actinomycetemcomitans*. This inhibition is achieved through various mechanisms, including the production of hydrogen peroxide, antibacterial substances like bacteriocins, and the generation of inorganic acids.

-Antimicrobial properties

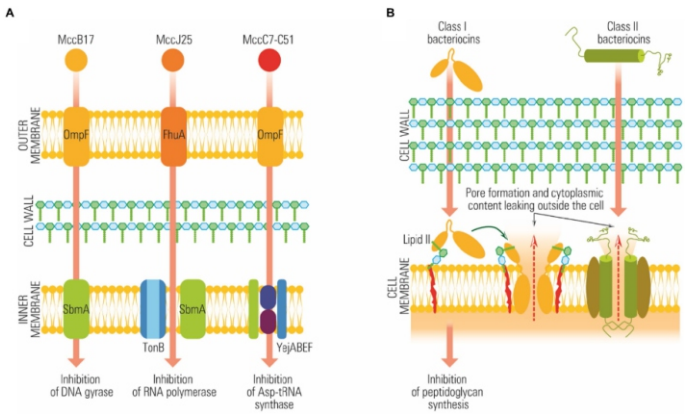
Probiotics present a promising approach to combat the rise of antibiotic-resistant bacteria. Probiotics employ several key antimicrobial mechanisms to achieve this goal, including competitive exclusion, intestinal barrier function improvement by enhancing mucin and tight junction protein expression, antimicrobial molecule secretion, and immune system regulation.

The antimicrobial activity of probiotics relies significantly on their ability to produce antimicrobial peptides, which play a central role in competitively excluding pathogens.

Lactobacillus strains have demonstrated significant antimicrobial activity against various pathogens, including *Klebsiella* sp., *Clostridium difficile*, *Shigella* sp., *E. coli*, *P. aeruginosa*, *S. mutans*, and *S. aureus.* They employ multiple strategies to outcompete and inhibit these pathogenic bacteria, including producing lactic acid, bacteriocin, and hydrogen peroxide, inhibiting the adhesion of pathogenic bacteria to the mucosa and improving the immune response.

Probiotics can improve and reinforce the integrity of the gut barrier through various mechanisms, including upregulating genes and protein expression involved in tight junction signalling. Probiotics can also regulate the apoptosis and proliferation of intestinal epithelial cells, contributing to barrier repair and maintenance. Probiotics can induce mucin expression and promote mucus secretion by goblet cells.

The primary method by which *Lactobacillus* strains exhibit antimicrobial activity is by releasing substances called bacteriocins. Bacteriocins are antimicrobial peptides that combat many bacteria, including Gram-positive and Gram-negative species. Both *Bifidobacterium* and *Lactobacillus* strains are known to be producers of bacteriocins. These antimicrobial peptides function through various means, which include inhibition of lipid II, a critical component of bacterial cell membranes, prevention of peptidoglycan synthesis, and pore formation. The pore formation process often involves a receptor known as the mannose-phosphotransferase system.



(A) The action of bacteriocins on Gram-negative targets, without the formation of pores. **(B)** The action of bacteriocins on Gram-positive targets, with the formation of membrane pores

-Antioxidant properties

Probiotics can chelate metal ions, such as iron, thereby reducing their availability for catalysing the production of harmful ROS. Certain probiotic strains can synthesize antioxidant metabolites, including vitamins like C and E, glutathione, and various ROS-scavenging enzymes, which help mitigate oxidative stress. Probiotics can stimulate the host's production of antioxidants or enhance dietary antioxidants' absorption, bolstering the body's overall antioxidant capacity. Probiotics can influence oxidative stress and inflammation by modulating signalling pathways, ultimately regulating ROS production. Some probiotic strains can downregulate enzymes responsible for ROS generation, such as NADPH oxidase, leading to decreased ROS levels and reduced oxidative stress. Probiotics also play a role in shaping the composition of the intestinal microbiota, indirectly impacting gut health and potentially contributing to the reduction of oxidative stress.

Oxidative stress arises from either the cumulative production of ROS or the inadequate scavenging activity of the antioxidant system, resulting in a disturbance in the body's redox balance.

Whether taken alone or alongside food, probiotic consumption has demonstrated the capacity to enhance antioxidant activity, thereby mitigating tissue damage caused by oxidative processes.

Among the various antioxidant activities exhibited by probiotic strains such as *Lactobacillus* sp., *Bifidobacterium* sp., and *Propionibacterium* sp., it has been observed that *P. freudenreichii* displays the highest antioxidant activity.

These probiotics release potent antioxidant compounds, including vitamin E, vitamin C, glutathione, beta-carotene, superoxide dismutase (SOD), polysaccharides, prototypical coenzyme I (NADH), and certain unidentified substances, all of which contribute to the promotion of gut health.

Various *Lactobacillus* strains, including *L. johnsonii*, *L. reuteri*, *L. brevis* and *L. fermentans* have been observed to activate the NF-κB pathway.

Probiotics exhibit antioxidant properties through several mechanisms, including scavenging free radicals, chelation of metal ions, regulation of antioxidant enzyme expression, and modulation of the gut microbiota. These effects are mediated at the molecular level by the influence of probiotics on various signalling pathways, such as Nrf-2, NF-κB, MAPK, and SIRTs, allowing them to exert their beneficial antioxidant effects.

Update on potential interventions to reduce the risk for transmission of healthcare-associated pathogens from floors and sinks II

Healthcare facility floors and sink drains and other wastewater drainage sites are universally contaminated with potential pathogens and it is plausible that organisms can be disseminated from these sites.

There are plausible mechanisms by which organisms in these sites can be disseminated to environmental surfaces that are commonly touched and to patients and personnel.

One factor that has limited progress in addressing floors and sinks has been the lack of practical and effective measures to reduce the risk for dissemination of organisms from these sites.

SINKS

Evidence that sinks and other wastewater drainage systems are a potential source of pathogen dissemination

Contaminated sink drains and other wastewater drainage sites have been linked to numerous outbreaks due to gram-negative bacilli. Wastewater drainage sites provide optimal conditions for biofilm formation and plasmid-mediated sharing of resistance genes. It is microbiologically plausible that organisms colonizing sink drains could be a source of infections: organisms can be dispersed from beneath the strainer to the sink bowl, countertop, and to patients or personnel by splashing of flowing water.

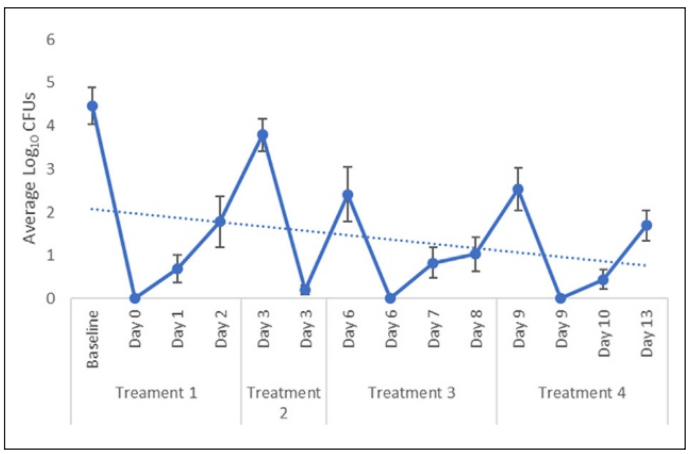
Potential interventions to reduce dispersal of microorganisms from sink drains pouring or instilling liquid disinfectants in sink drains

Addressing sink contamination is challenging because sink drains are not amenable to cleaning and disinfection. Sinks in healthcare facilities typically have fixed, narrow strainer holes that do not permit access by brushes that could be used to remove bioburden prior to application of disinfectants. Pouring liquid disinfectants into sink drains may have only a modest and transient effect on sink colonization. The limited impact of this approach may be that liquid disinfectants flow rapidly down the drain, providing inadequate contact time and poor penetration into many of the areas harbouring biofilm-associated microorganisms.

Use of as top valve or an inflated urinary catheter balloon to allow a 1-hour instillation of disinfectant throughout the proximal drainage system reduced proximal sink drain colonization for several days, whereas pouring disinfectants down drains had only a transient impact.

Foam disinfectants

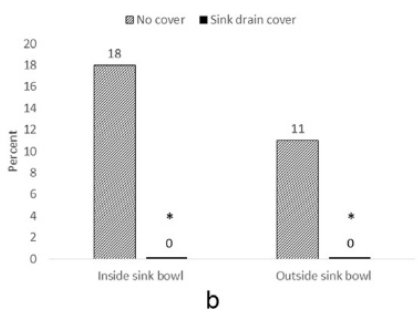
Effect of repeated treatment with a foam application of a disinfectant containing hydrogen peroxide 3.13%, peraceticacid 0.05%, and octanoic acid 0.099% on the burden of gram-negative bacilli recovered from sink drains.



It is plausible that disinfectants applied as a foam might be more effective than pouring liquid disinfectants for drain decontamination due to greater persistence and increased penetration into sites harbouring microorganisms. Intermittent application of an easy to apply foam product could potentially reduce the risk for dissemination of pathogens from sink drains. A hydrogen peroxide-based foam disinfectant was more effective than liquid bleach in decreasing sink drains bacterial counts at 24hours, but not 7 days, post-treatment.

Drain Covers

a



b

Sink drain cover placed in a sink and percentage of sink bowls and surfaces outside the bowls with positive cultures for gram-negative bacilli after running the water for 30seconds.

Given the challenges involved in cleaning and disinfection of sinks, one alternative strategy to prevent dispersal of organisms is to provide a cover for the sink strainer. This approach is like a recent intervention in which a reduction in Klebsiella pneumoniae carbapenemase (KPC) producing organisms was attributed to installation of covers on wastewater hoppers. Closing toilet covers while flushing has also been recommended to prevent dispersal of organisms.

The sink drain cover required approximately 15 seconds to install or remove. The device fit on all types of sinks tested and did not reduce water outflow.

The drain cover was effective in preventing dispersal of fluorescent gel to counter tops or other adjacent surfaces (0/30,0% vs 8/30,27%dispersal). There was evidence of splashing of fluorescent gel from the drain to the bottom surface of the drain cover, but no dispersal was noted outside the cover. The device also prevented dispersal of colonizing gram-negative bacilli to the sink bowl and to surfaces adjacent to the bowl.

Other approaches to reduce dispersal of microorganisms from sink drains

Variety of other approaches have been used to reduce dispersal of microorganisms from sink drains. Some of these approaches include programmable flushing with ozonated water, removal of sinks from units, replacement of drainage pipes and traps, and installation of heater vibrator devices for trap disinfection. The major limitation of many of these measures is that they are costly or labour intensive.

Using a laboratory sink model, it was recently demonstrated that bacterial persistence in sinks may be influenced by the frequency of water flushing and sink usage. A minimum of 2 flushes per week was necessary to significantly reduce establishment of colonization by a strain of Escherichia coli and keep bacterial counts undetectable at the sink drain.

Several studies have demonstrated that sink design may play a key role in determining the risk for dispersal of colonizing microorganisms. For example, placement of the faucet such that water flows directly onto the drain may increase the risk for organism dispersal.

The sink dispersed gram-negative bacteria to the environment and to the hands of personnel during handwashing. Organisms dispersed from the sink were like those recovered from hands of personnel and from infected wounds. The outbreak was controlled by infection control measures including discontinued use of the soiled utility sink.

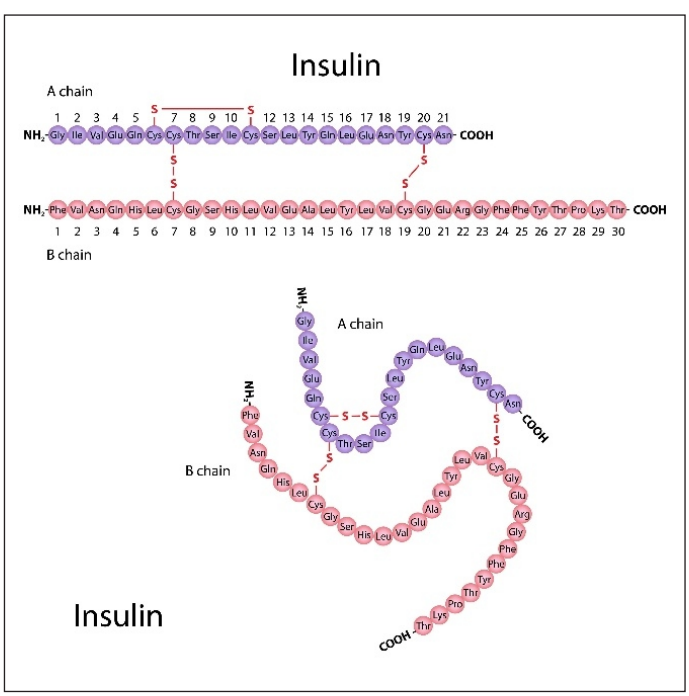
One factor that has hindered progress in addressing floors and sinks has been the lack of practical and effective measures to reduce the risk for dissemination of organisms from these sites. Some practical interventions that potentially could be used to address floor contamination include use of UV-C devices or electrostatic spray technologies to disinfect floors, use of UV-C devices to decontaminate the soles of shoes prior to entry into patient rooms or patient care areas, and having patients wear slippers when out of bed to reduce acquisition of pathogens on feet. Some practical interventions that could be used to address sink drains include intermittent use of foam disinfectants to reduce drain colonization, use of drain covers to reduce the dispersal of microorganisms, and modifications in sink design. Studies that include interventions to address floor and sink contamination may provide insights into the role of these sites in dissemination of healthcare associate pathogens.

Frederick Banting

The story of Frederick Banting is one of the most compelling narratives in the history of science, a tale of a man who possessed neither the pedigree nor the resources of a world-class researcher but who, through sheer force of will and a singular, inspired idea, managed to change the course of human history. Born on November 14, 1891, on a farm in Alliston, Ontario, Frederick Grant Banting was the youngest of five children. His upbringing was characterized by the rugged simplicity of rural life, shaped by the Methodist values of his parents, William Thompson Banting and Margaret Grant. It was in this environment that he developed the stoicism and tenacity that would later define his medical career. Though he initially struggled with his studies, even failing his first year at the University of Toronto's General Arts program, his determination eventually led him to the Faculty of Medicine. His education was accelerated by the tremors of the First World War, an event that would forge his character in the crucible of conflict. Graduating in a special class in 1916 to meet the demand for doctors, he immediately joined the Canadian Army Medical Corps. It was during the Battle of Cambrai in 1918 that Banting's legendary status began to take root. Despite being severely wounded by shrapnel, he refused to leave his post, spending seventeen hours tending to the wounded while under heavy fire. This act of bravery earned him the Military Cross, but the physical and mental scars of the war would stay with him forever, instilling in him a deep empathy for the suffering and a restless desire to find meaning in his post-war life.

Upon returning to Canada, Banting's journey was far from the triumphant return one might expect for a war hero. He struggled to establish a private practice in London, Ontario, finding himself in a quiet office with very few patients. It was during this period of professional stagnation that he took a part-time job teaching anatomy and physiology at the University of Western Ontario. On the night of October 31, 1920, while preparing a lecture on the pancreas, he read an article by Moses Barron concerning the relationship between the pancreas and diabetes. The prevailing medical knowledge suggested that the pancreas produced a mysterious "internal secretion" that regulated blood sugar, but no one had been able to isolate it because the organ's own digestive enzymes destroyed the secretion the moment the pancreas was handled. Banting, in a flash of insight, hypothesized that if he could tie off the pancreatic ducts, the digestive-enzyme-producing cells would atrophy, leaving the insulin-producing Islets of Langerhans intact to be harvested. This idea, scribbled in a notebook in the middle of the night, would become the foundation of one of the greatest medical breakthroughs in history.

Driven by this vision, Banting traveled to the University of Toronto to seek the help of Professor J.J.R. Macleod, a leading figure in carbohydrate metabolism. Macleod was initially unimpressed by the young, inexperienced surgeon who lacked a background in research, but Banting's persistence eventually wore him down. Macleod granted him a small, poorly ventilated laboratory, ten experimental dogs, and the assistance of a medical student named Charles Best. During the sweltering summer of 1921, while Macleod was away in Scotland, Banting and Best embarked on a grueling series of experiments. The conditions were miserable; the heat in the top-floor lab was stifling, and the work was physically and emotionally taxing. They faced numerous failures as their experimental dogs died, but they refused to give up. By late summer, they successfully isolated a crude extract from the shriveled pancreases of their dogs. When they injected this extract into a diabetic dog named Marjorie, her blood sugar levels dropped, and her lethargic state transformed into one of vitality. This was the first evidence that they had found the elusive internal secretion, which they initially called "Isletin."



As the results became undeniable, Macleod returned and brought in James Collip, a skilled biochemist, to help purify the extract for human testing. This period was marked by intense friction, particularly between Banting and Macleod, as Banting felt his contribution was being marginalized by the established professor. However, the scientific goal remained paramount. By early 1922, they were ready for the first human trial. At the Toronto General Hospital, a fourteen-year-old boy named Leonard Thompson was dying of diabetes. In that era, a diagnosis of diabetes was a slow death sentence, with children often placed on "starvation diets" that only briefly delayed the inevitable. Leonard was so weak he could barely move, weighing only sixty-five pounds. The first injection of the Toronto extract was only partially successful, but after Collip refined the process further, a second injection on January 23, 1922, produced miraculous results. Leonard's blood sugar returned to normal levels, his urine became clear of ketones, and he began to regain his strength. The "sugar death" had finally been conquered.

The success of insulin brought Banting instant international fame, but it also brought a sense of duty that transcended personal gain. In 1923, when the Nobel Prize in Physiology or Medicine was awarded to Banting and Macleod, Banting was outraged that Charles Best had been excluded. In a remarkable show of loyalty and character, he immediately shared his prize money with Best, prompting Macleod to do the same for Collip. Perhaps even more significant was Banting's decision regarding the patent for insulin. He believed that a life-saving discovery should belong to the world, not to an individual or a corporation. He famously sold the patent to the University of Toronto for a mere one dollar, ensuring that the treatment would be accessible to as many people as possible. He famously stated that insulin did not belong to him, but to the world. This act of altruism solidified his reputation as a humanitarian. He spent the following years as a public figure, though he often felt uncomfortable with the limelight, preferring the quiet solitude of his laboratory or his hobby of painting. He was an accomplished artist, often traveling with members of the Group of Seven to capture the stark beauty of the Canadian wilderness.

As the world moved toward another global conflict in the late 1930s, Banting once again felt the call to serve. He turned his scientific mind toward aviation medicine, recognizing that the pilots of the new generation of fighter planes would face physiological challenges never before seen. He was instrumental in developing the first anti-gravity suit, known as the Franks G-suit, which prevented pilots from blacking out during high-speed maneuvers. He frequently put his own life at risk, acting as a human subject for his own experiments, including tests in high-pressure chambers and exposures to chemical agents. Tragically, it was this dedication to military research that led to his death. On February 21, 1941, while flying to England for secret research consultations, his plane crashed in a remote region of Newfoundland. Banting survived the initial crash but died of his injuries and exposure before help could arrive. He was only forty-nine years old. Sir Frederick Banting's legacy lives on in the millions of people who lead full lives today because of a midnight thought in 1920. He was a man who combined the practical hands of a surgeon with the visionary mind of a researcher and the heart of a soldier, proving that a single individual, armed with an idea and the courage to pursue it, can change the world forever.

Jokes

.A man takes his dog to the vet and says, "Doctor, I'm worried. I think my dog is dead."
The vet puts the dog on the table, then brings in a Labrador Retriever. The Lab sniffs the dog from head to toe and shakes its head.
Then the vet brings in a cat. The cat sniffs the dog, meows sadly, and walks away.
The vet says, "I'm sorry, he's gone. That'll be $500."
The man cries, "Rs. 500 just to tell me he's dead?!"
The vet replies, "Well, it was Rs. 50 for the office visit, Rs. 200 for the **Lab report**, and Rs. 250 for the **Cat scan**."

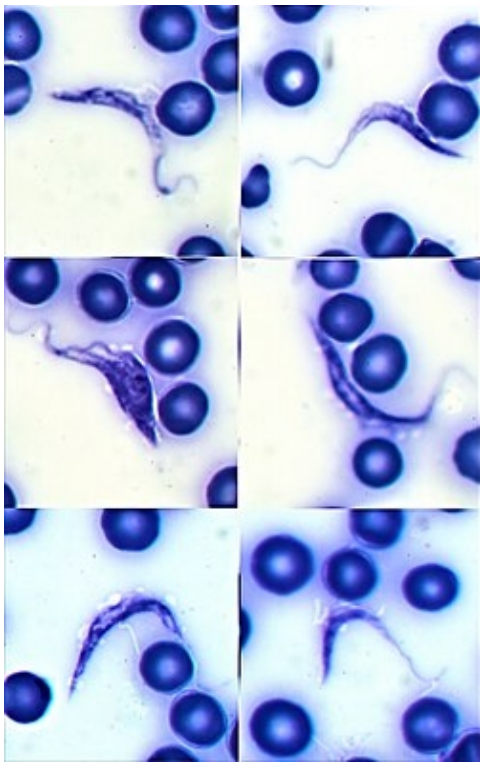
A guy buys a parrot, but it has a terrible habit of swearing constantly.
He tries everything to stop it, but nothing works. Finally, he gets fed up and puts the parrot in the freezer to cool off for a minute.
After a moment of silence, he opens the door. The parrot walks out, shivering, and says, "I sincerely apologize for my behaviour and will never swear again."
Then the parrot adds, "By the way... can I ask what the **chicken** did?"

An interviewer is talking to a candidate for a high-level position.
"What would you say is your greatest weakness?" the interviewer asks.
The candidate replies, "I can be uncomfortably honest sometimes."
The interviewer smiles and says, "I actually don't think honesty is a weakness at all."
The candidate looks him in the eye and says, "**I don't give a damn what you think.**"

A police officer pulls over a man who has a penguin sitting in his passenger seat.
The officer says, "Sir, you can't drive around with a penguin! Take him to the zoo immediately."
The next day, the officer sees the same man with the penguin again and says, "I thought I told you to take him to the zoo!"
The man replies, "**I did! We had a great time, so today we're going to the movies.**"

A guy finds a magic lamp and a genie pops out to grant him three wishes.
First, the guy says, "I want a billion dollars." *Poof!* He's rich.
Second, he says, "I want a brand-new Ferrari." *Poof!* A red car appears.
For his third wish, he says, "I want to be irresistible to women."
Poof! The genie turns him into a giant box of chocolates.

Trypanosoma cruzi



Trypanosoma cruzi is a flagellated protozoan parasite of immense public health significance, serving as the etiological agent of Chagas disease, also known as American trypanosomiasis. Discovered in 1909 by the Brazilian physician and researcher Carlos Chagas, the parasite remains a major cause of morbidity and mortality across the Americas and, more recently, throughout the world due to global migration. It belongs to the order Kinetoplastida and is characterized by the presence of a kinetoplast, a specialized mitochondrial organelle containing a dense mass of circular DNA. Unlike many other pathogens, *T. cruzi* is a generalist, capable of infecting over 150 species of mammals, including humans, domestic animals, and various sylvatic reservoirs like opossums and armadillos. This broad host range makes the eradication of the parasite nearly impossible, as it can persist in diverse ecological niches outside of human intervention.

The life cycle of *Trypanosoma cruzi* is complex and involves two distinct hosts: a triatomine insect vector, often referred to as the "kissing bug," and a mammalian host. The cycle begins when a triatomine bug takes a blood meal from an infected mammal, ingesting bloodstream trypomastigotes. Once inside the insect's midgut, these parasites transform into epimastigotes, which are the primary replicative stage within the vector. As they migrate toward the insect's hindgut, they differentiate into metacyclic trypomastigotes, the non-replicative but highly infectious form. Transmission to humans occurs uniquely; the bug typically feeds on a person at night, often biting near the eyes or mouth. While feeding, the bug defecates, and the metacyclic trypomastigotes present in the feces enter the human body through the bite wound or through mucous membranes when the person inadvertently rubs the site.

Once inside the mammalian host, the metacyclic trypomastigotes invade various cell types, particularly macrophages, fibroblasts, and muscle cells. Inside the host cell, the parasite transforms into an amastigote, a round, intracellular stage that lacks a visible external flagellum. The amastigotes multiply by binary fission within the host cell's cytoplasm until the cell is physically overwhelmed. At this point, the amastigotes transform back into trypomastigotes, which possess a flagellum and an undulating membrane, allowing them to burst the host cell and enter the bloodstream. These bloodstream trypomastigotes do not replicate; instead, they circulate through the body to invade new tissues or wait to be ingested by another triatomine vector to continue the cycle. This constant cycle of cell invasion and rupture leads to significant tissue damage over time, particularly in the heart and digestive organs.

While vector-borne transmission is the most prevalent route, *T. cruzi* can be transmitted through several other pathways that have become increasingly relevant in the modern era. Oral transmission is a significant concern, often occurring through the consumption of food or drink contaminated with infected triatomines or their feces, such as raw sugarcane or acai juice. These oral outbreaks are often more severe than vector-borne cases because the initial parasite load is much higher. Furthermore, the parasite can be transmitted congenitally from an infected mother to her fetus across the placental barrier, leading to a new generation of infected individuals even in regions where the vector has been eliminated. Blood transfusions and organ transplants from infected donors also pose a risk, necessitating rigorous screening protocols in blood banks and hospitals worldwide.

The pathogenesis of Chagas disease is driven by both the direct damage caused by the parasite and the host's subsequent immune response. *T. cruzi* has evolved sophisticated mechanisms to evade the host's immune system, such as the production of trans-sialidase enzymes. These enzymes allow the parasite to "steal" sialic acid from host glycoproteins and incorporate it into its own surface, essentially masking itself from the host's antibodies. Additionally, the parasite produces cruzipain, a cysteine protease that helps it degrade host tissues and modulate the immune environment. Over time, the persistent presence of the parasite and the chronic inflammation it triggers lead to the destruction of the autonomic nervous system in the heart and gut, resulting in the characteristic organ enlargements associated with the disease.

Clinically, Chagas disease is divided into two phases: the acute and the chronic. The acute phase occurs immediately after infection and lasts for about two months. In many cases, it is asymptomatic or presents with mild symptoms like fever, headache, and lymph node swelling. A classic diagnostic sign during this phase is Romaña's sign, characterized by painless unilateral swelling of the eyelid, which occurs when the parasite enters through the conjunctiva. If the acute phase is not treated, the infection enters the chronic phase, which begins with an "indeterminate" stage. During this period, which can last for decades, individuals appear healthy and show no symptoms, yet they remain carriers. Roughly $30–40\%$ of these individuals will eventually progress to the symptomatic chronic stage,

developing life-threatening conditions such as Chagasic cardiomyopathy or digestive megasyndromes like megaesophagus and megacolon.

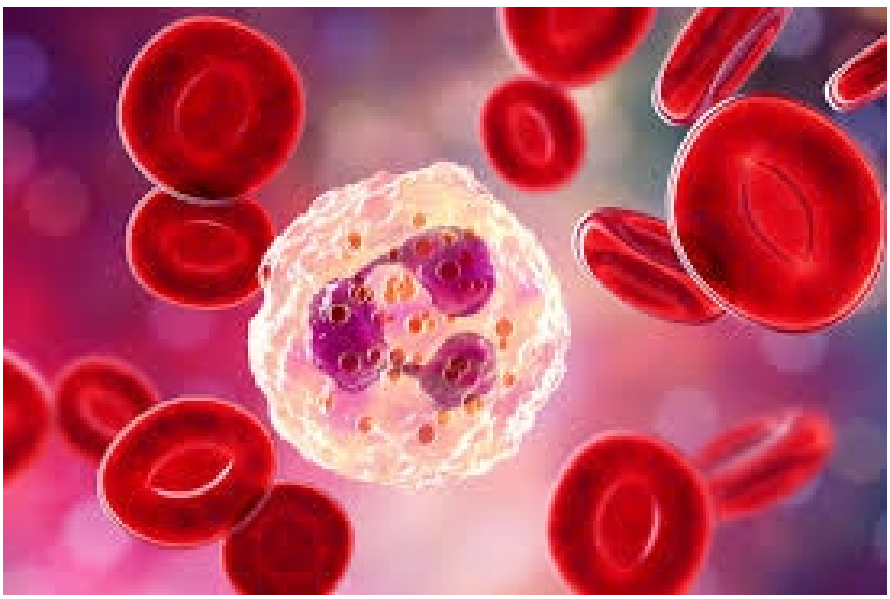
Diagnosis of *T. cruzi* infection varies depending on the stage of the disease. In the acute phase, when parasite levels in the blood are high, direct microscopic observation of blood smears or the use of polymerase chain reaction (PCR) to detect parasite DNA is effective. However, once the disease enters the chronic phase, the parasites retreat into deep tissues, making them nearly impossible to find in the blood. Therefore, chronic diagnosis relies on serological tests, such as ELISA or indirect immunofluorescence, to detect IgG antibodies against *T. cruzi*. Because of the potential for cross-reactivity with other parasites like *Leishmania*, international guidelines typically require two different serological tests to confirm a positive diagnosis.

Treatment for Chagas disease is currently limited to two nitroheterocyclic compounds: Benznidazole and Nifurtimox. These drugs are highly effective when administered during the acute phase or in congenital cases, often achieving a 100% cure rate in newborns. However, their efficacy decreases significantly as the infection progresses into the chronic stage. Furthermore, both medications are associated with a high frequency of adverse side effects, including severe dermatitis, peripheral neuropathy, and gastrointestinal distress, which often lead patients to discontinue treatment. In 2026, research continues to focus on finding more effective, less toxic alternatives and exploring whether combination therapies can better clear the parasite in chronic patients.

The epidemiology of *T. cruzi* has shifted dramatically over the last century. Once confined to rural, impoverished areas of Latin America where people lived in mud-walled, thatched-roof houses that harbored triatomine bugs, the disease is now a global concern. Urbanization and international migration have brought the parasite to non-endemic regions including the United States, Europe, and parts of the Western Pacific. In these areas, the primary risks are congenital transmission and blood safety. Public health efforts now emphasize a multifaceted approach: improving housing conditions to exclude vectors, implementing universal screening for pregnant women and blood donors, and increasing awareness among healthcare providers in non-endemic countries to identify the "silent" chronic cases before they reach end-stage organ failure.

Trypanosoma cruzi is a resilient and complex parasite that poses a unique challenge to global health. Its ability to hide within host cells for decades and its diverse modes of transmission make it a formidable pathogen. While significant progress has been made in controlling the triatomine vectors in many South American countries, the millions of people currently living with chronic infections require ongoing medical support and better therapeutic options. As we move forward, the integration of molecular research, vector control, and social policy remains essential to reducing the burden of Chagas disease and eventually breaking the cycle of transmission for *T. cruzi*.

INFLAMMATION TURNS BONE MARROW INTO A BREEDING GROUND FOR DISEASE



Inflammation covertly rewires the bone marrow, enabling mutated stem cells to rise and setting the stage for future blood disease.

Every moment, the bone marrow generates millions of fresh blood and immune cells. This nonstop renewal depends on a carefully balanced relationship between hematopoietic stem cells (HSCs), supportive stromal cells, and a network of immune signals.

Over the years, this balance becomes vulnerable. Aging, chronic inflammation, or somatic mutations can disrupt communication among these cell groups, reducing normal stem-cell renewal and allowing mutated HSCs to expand unnoticed. This process leads to clonal hematopoiesis of indeterminate potential (CHIP), which appears in about 10 to 20% of adults over 60 and nearly 30% of those over 80

Although people with CHIP typically have no symptoms, the condition increases the risk of blood cancers by tenfold and doubles the likelihood of cardiovascular disease and early death. Myelodysplastic syndrome (MDS), a related disorder involving clonal HSCs, causes inefficient blood-cell production and gradual failure of the bone marrow. It affects up to 20 in every 100,000 adults over 70, and around 30% of cases advance to acute myeloid leukemia (AML), an aggressive and often fatal cancer.

Despite the seriousness of these disorders, the contribution of the bone marrow microenvironment to their development has remained unclear.

Mapping Hidden Changes in the Bone Marrow Microenvironment

To better understand how mutated HSC clones gain dominance, an international research team co-led by Judith Zaugg from EMBL and University of Basel and Borhane Guezguez from UMC Mainz carried out an extensive molecular and spatial analysis of human bone marrow. The samples came from the BoHemE cohort study in collaboration with Uwe Platzbecker at the National Center for Tumor Diseases (NCT) Dresden.

Using single-cell RNA sequencing, biopsy imaging, proteomics, and co-culture models, the researchers created a detailed map of the bone marrow microenvironment in healthy donors (including those with CHIP) and in patients with MDS. Their analysis revealed an unexpected cellular shift that begins long before clinical signs appear. The team found that a population of inflammatory stromal cells gradually replaces the usual mesenchymal stromal cells (MSC) that support stem-cell function.

"I was surprised to observe such pronounced remodeling of the bone marrow microenvironment already in individuals with CHIP, although the underlying cause-and-effect relationships remain unclear," said Zaugg, co-senior author, EMBL Group Leader, and Professor at Basel University.

Unlike healthy stromal cells, these inflammatory MSCs (iMSC) produce large amounts of interferon-induced cytokines and chemokines. These molecules attract and activate interferon-responsive T cells, which then intensify the inflammatory activity. This creates a feed-forward loop that maintains chronic inflammation, disrupts normal blood formation, and contributes to vascular changes in the marrow.

Identifying What Drives Bone Marrow Inflammation

Interestingly, the researchers did not find signs that mutated hematopoietic cells in MDS directly trigger this inflammatory response. They were able to separate mutated from non-mutated cells using SpliceUp, a computational method developed by co-lead author and EMBL alumnus Maksim Kholmatov in collaboration with Pedro Moura and Eva Hellström-Lindberg from Karolinska Institute. SpliceUp identifies mutated cells in

single-cell datasets by detecting abnormal RNA-splicing patterns. In MDS, the inflammatory network within the microenvironment becomes dominant and replaces much of the marrow's normal regenerative structure.

"Another striking observation was that MDS stem cells couldn't trigger stromal cells to produce CXCL12, an important signal that triggers blood cells to settle in the bone marrow. This failure may help explain why the bone marrow stops working properly," said Karin Prummel, co-lead author and EMBL postdoc.

"It was quite surprising to see the lack of a direct inflammatory effect that we could attribute to the mutant cells," said Maksim Kholmatov, co-lead author and EMBL alumnus. "However, when viewed in the context of changes in the T cell and stromal compartments, it underlines the importance of the bone marrow microenvironment in shaping disease progression."

Inflammation as an Early Driver of Blood Disease

These findings indicate that inflammation plays a central role in the earliest phases of disease and highlight the bone marrow microenvironment (also called the bone marrow niche) as a key therapeutic focus. By directing attention to the ecosystem that supports mutated stem cells rather than the mutated cells alone, the research points to new opportunities for early treatment and prevention.

Anti-inflammatory drugs or therapies that adjust interferon signaling may help preserve marrow function in older adults with CHIP. Combining targeted treatments with therapies that act on the microenvironment could slow or prevent the transition from CHIP to MDS or AML. The specific molecular features of iMSCs and interferon-responsive T cells may also serve as early biomarkers for people at elevated risk.

"Our findings reveal that the bone marrow microenvironment actively shapes the earliest stages of malignant evolution," said Guezguez, Principal Investigator in the Department of Hematology at UMC Mainz and co-senior author. "As advances in molecular profiling allow us to detect pre-leukemic states years before clinical onset, understanding how stromal and immune cells interact provides a foundation for preventive therapies that intercept disease progression before leukemia develops."

Inflammaging and the Wider Impact on Age-Related Disease

Beyond blood disorders, the results contribute to a broader understanding of 'inflammaging', the low-level, chronic inflammation that supports many age-related conditions, including cancer and cardiovascular and metabolic disease. The bone marrow, once considered only a site of blood production, now appears to be both affected by and responsible for systemic inflammatory aging. By showing how interactions between immune and stromal cells drive these changes, the study offers a model for exploring inflammatory remodeling in other myeloid malignancies and advanced leukemia.

“It will be crucial to study these processes over time; our current findings are based on cross-sectional data," Zaugg said. "This has important implications for therapies that replace malignant cells but leave the bone marrow niche intact, such as blood stem cell transplantation. We are now investigating to what extent the niche retains a 'memory' of disease, which could shape how it responds to new, healthy stem cells."

The Radiation Risk That's Hiding in Your Pocket Right Now and How to Stop it

An increased screentime is something that most of the people are experiencing day by day. Most of us can't even imagine going a full day without a mobile phone. From messages and video calls, from work to school assignments, and banking, our devices have become essential. But as convenient as they are, they emit electromagnetic radiation, which is a growing concern of health.

Importance of Reducing Radiation Exposure

Cell phones give off a type of low-level radiation whenever they send or receive signals. This radiation, while not as strong as X-rays, can still be absorbed by your body, especially when the phone is close to your head or body, which can be harmful in the long run.

Some research has suggested potential links between long-term exposure to RF radiation and issues such as headaches, sleep disturbances, reduced sperm quality, and, in some cases, more serious conditions.

Many experts suggest it's smart to take simple steps to reduce your exposure, to be on a safer side. That's why adopting simple daily habits can go a long way in minimizing your radiation exposure.

Tips To Reduce Cell Phone Radiation

1. Use Speakerphone or Wired Headphones

Holding your phone to your ear exposes your brain and surrounding tissues to the highest level of RF radiation. To remedy that, you can use speaker mode or wired headphones to keep the phone away from your head during calls. Bluetooth devices also emit radiation, so it is a good idea to go wired when possible.

2. Keep Your Phone Away from Your Body

Avoid keeping your phone in your pockets or under your pillow. You can use a bag, table, or desk instead. Even when not in use, phones emit small amounts of radiation as they search for a signal.

3. Text More, Talk Less

Texting reduces exposure significantly compared to making voice calls, since you are not holding the phone to your head or body. You can choose to communicate through messaging apps or SMS instead of calling whenever possible.

4. Limit Call Time and Frequency

When it is important for you to be on calls, try to make it shorter. Shorter calls mean less exposure. If you need to talk for a long time, alternate ears, take breaks, or use a landline. Avoid prolonged use in low signal areas, where your phone boosts its power output.

5. Avoid Calls When Signal Is Weak

When your phone shows one or two bars of signal, it works harder to connect, emitting more radiation. Try to wait until you are in a better signal zone before placing or receiving calls.

6. Don't Sleep Next to Your Phone

This one is common among many. You can adopt the habit of keeping your phone at least 3 feet away from your bed while you sleep. Better yet, switch off your Mobile Data or Wi-Fi for the duration. This minimizes unnecessary nighttime radiation exposure.

7. Limit Radiation Exposure for Children

Children's bodies absorb more radiation due to their thinner skulls and developing tissues. Parents must encourage limited phone time for kids.

8. Disable Unused Connectivity

Turn off Wi-Fi, Bluetooth, and GPS when you are not using them. These functions emit radiation even when idle and contribute to your overall exposure.

9. Avoid Using Phones in Enclosed Spaces

Elevators, cars, buses, and trains often have poor signal reception. In these cases, your phone emits higher radiation within enclosed environments to maintain a connection. And so, it is better to wait until you are in open space to use your phone.

10. Use Radiation-Protection Solutions

There are various solutions out in the market to protect yourself from unwanted radiation, especially from the usage of a phone.

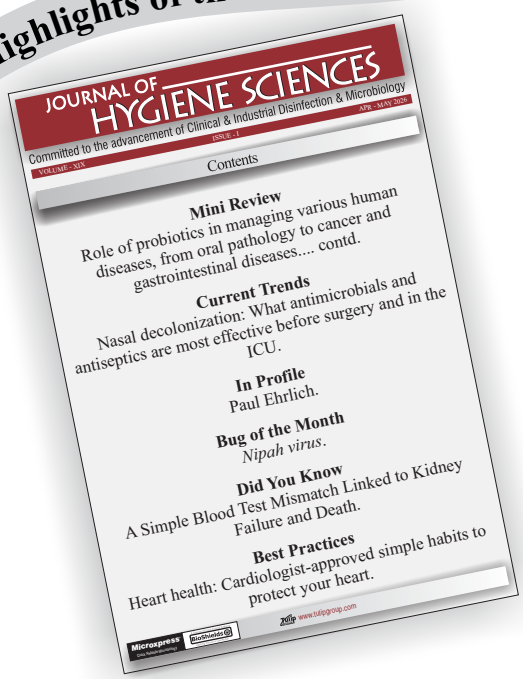
Choose a Smarter Approach to Reduce Phone Radiation

Cell phones have become an essential part of modern life. However, the radiation emitted by them does not mean that we should completely avoid using them. The goal is not to eliminate technology, but to use it in a smart manner.

There is no need to ditch your phone. But with small, conscious changes, you can significantly reduce radiation exposure.

Whether it is using a speakerphone, avoiding low-signal areas, or applying a tested radiation shield, every step counts toward protecting your long-term health.

Highlights of the coming issue



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