INFINITY

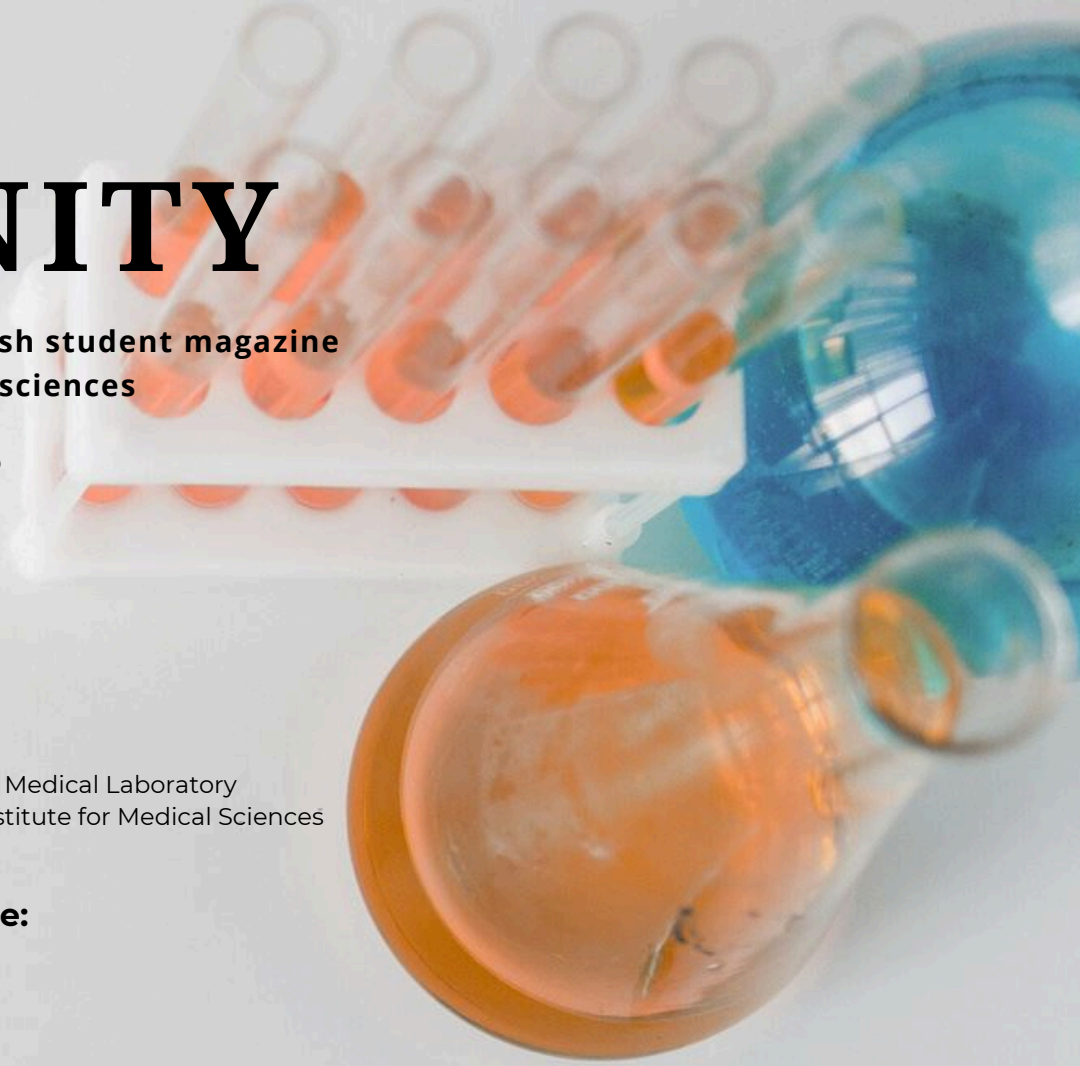
SCIENTIFIC ASSOCIATION OF MEDICAL LABORATORY SCIENCES, VARASTEGAN INSTITUTE FOR MEDICAL SCIENCES

No.10- December 2025

INFINITY

The first and only English student magazine for medical laboratory sciences

No.10- December 2025



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DIRECTOR-IN-CHARGE:

We are honored to present the tenth edition of our English-language journal in laboratory sciences — an achievement made possible through the dedication of our contributors and the unwavering support of the academic community.

As our readership continues to grow, our commitment to providing relevant, accurate, and forward-thinking content grows even stronger. Laboratory science is advancing at an unprecedented pace, and our goal is to provide a platform that not only informs but also inspires helping professionals navigate complex questions, adopt innovative approaches, and remain at the forefront of discovery.

Our editorial board, proudly supports the insights and ambition of students in related fields. Whether your path leads you to writing, editing, reviewing, or simply exploring new scientific perspectives, there is a meaningful place for you within this evolving community.

May this edition serve as a graceful reminder of what students can accomplish together guided by curiosity, discipline, and a shared devotion to the pursuit of science.
Sincerely,

Yeganeh Khazaei
Director-in-charge of Infinity

EDITOR-IN-CHIEF:

In every laboratory, there is a moment when the invisible becomes visible, and that moment is where Infinity begins. It is my honor to welcome you all to a publication shaped not only by science but also by intellect, commitment, and the genuine curiosity of our scientific community.

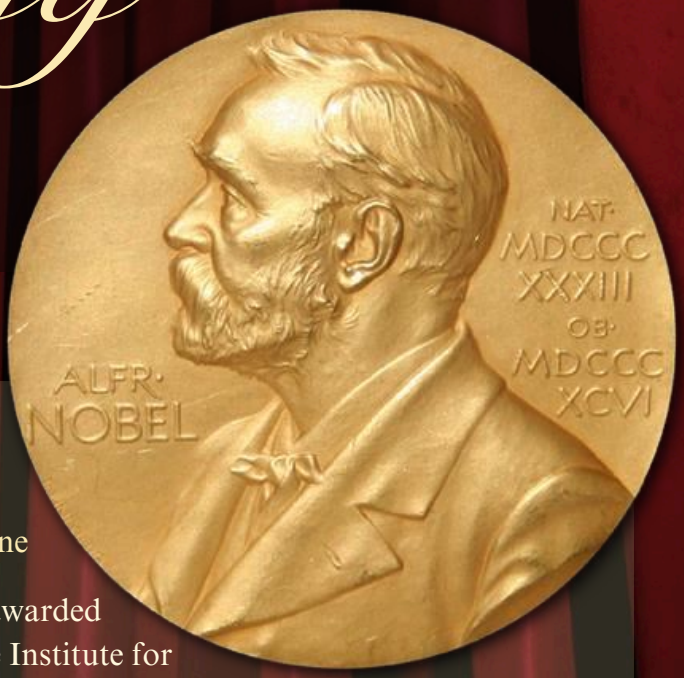
Infinity stands as a gathering point for those who seek more than facts to discover the mechanisms, patterns, and phenomena that define modern laboratory science.

This magazine is a reflection of the collaboration of authors who transform complex concepts into clear insight. Whether you are here to learn or to be inspired, we invite you with pleasure to explore this issue and even be part of it.

May this issue remind you that discovery is a continuous process without limits, and each step is valuable.

Melika Hosseinpour Mashhadi
Editor-in-chief of Infinity

The Nobel Prize in *Physiology* or Medicine 2025



How does the body protect itself from immune system attack?

A look at the 2025 Nobel Prize in Physiology or Medicine

In 2025, the Nobel Prize in Physiology or Medicine was awarded to three outstanding researchers—Mary A. Branco of the Institute for Biosystems in Seattle, Fred Ramsdell of Sonoma Biotherapeutics in San Francisco, and Shimon Sakaguchi of Osaka University in Japan.

The three scientists' joint achievement provides a deeper understanding of the immune system's amazing mechanism: how the body learns not to attack itself.

The secret to balance in the body's defense army

Every day, millions of immune cells engage with foreign agents such as viruses and bacteria. But this defense army, if left unchecked, can turn against the body itself.
Research by Branko, Ramsdel, and Sakaguchi showed that this delicate balance is the result of the action of a group of cells called "regulatory T "cells—cells that act as sentinels to keep the immune system from going astray.

From hypothesis to discovery

In the 1990s, most scientists believed that immune tolerance (the body's ability to prevent itself from being attacked) was formed only in the thymus, during the maturation of immune cells.
But in 1995, Sakaguchi challenged this belief with a bold discovery.

He identified a new type of immune cell that inhibited the activity of other immune cells even after leaving the thymus. These cells were later called “regulatory T cells.”

A few years later, in 2001, Branko and Ramsdel found the missing piece of the puzzle when they discovered a gene called Foxp3. They found that disruption or mutation of this gene disables regulatory T cells, resulting in severe autoimmune diseases such as IPEX.
Two years later, Sakaguchi showed that the Foxp3 gene in humans plays the same role as it did in mice final confirmation of the existence of a universal mechanism of “environmental tolerance” in the body.

New doors for treatment

Several therapies based on regulating the function of regulatory T cells are currently in clinical trials and could offer a different future for patients with type 1 diabetes, multiple sclerosis, and lupus.

Conclusion

The discovery of the concept of "environmental safety tolerance" has not only illuminated the secret of the body's self-control but has also shown that in science, as in life, sometimes power lies in the ability to restrain oneself.

Mary E. Brunkow

Fred Ramsdell

Shimon Sakaguchi

Amirali Ebrahimpour

Department of Medical Laboratory Sciences. Varastegan Institute for Medical Sciences, Mashhad, Iran

World Cervical Cancer Elimination Day, commemorated on November 17, was instituted by

Cervical cancer

AWARENESS DAY

November 17 has been a World Cervical Cancer Elimination Day for promoting awareness and efforts toward the elimination of cervical cancer worldwide.

Significance of the Day

World Cervical Cancer Elimination Day, commemorated on November 17, was instituted by the WHO to put a spotlight on the elimination of cervical cancer as a public health problem. This day will usher in the WHO's global strategy to accelerate the process of elimination of this type of cancer, which is one of the most common cancers among women in the world.

Plans and Strategies

The day highlights the WHO's 90-70-90 goals, which are to:

90: By the age of fifteen, 90% of girls had received all recommended doses of the HPV vaccine.

70: By the age of 35 and again by the age of 45, 70% of women were screened using a high-performance test.

90: 90% of women with invasive cancer were managed, while 90% of women with pre-cancer received treatment.

Activities and Awareness

This day is marked globally with various activities such as educational campaigns, health screenings, and advocacy to guarantee that all women have access to prevention, screening, and treatment services. The day acts as a time for reflection, education, and reiterated political commitment in combating cervical cancer.
Apart from World Cervical Cancer Elimination Day, Cervical Cancer Prevention Day is observed on May 21 to create awareness and stress the urgent need for effective prevention strategies against cervical cancer.
This will definitely let participation and contribution come from all levels- individuals and organizations-toward the worldwide effort of eradicating cervical cancer and improving women's health worldwide.

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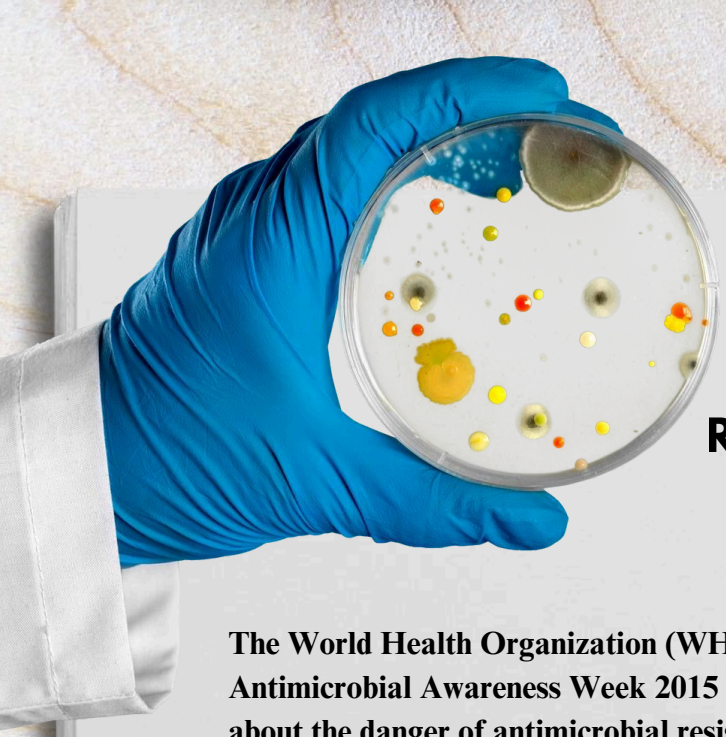
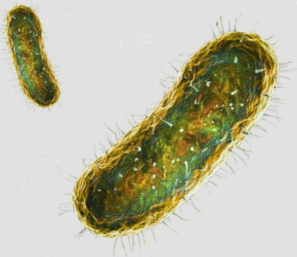
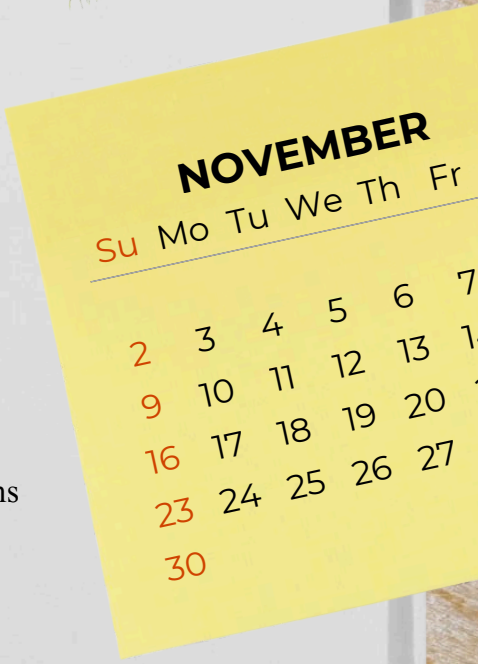


ANTIMICROBIAL RESISTANCE DAY



The World Health Organization (WHO) launched the World Antimicrobial Awareness Week 2015 to raise global awareness about the danger of antimicrobial resistance (AMR) and the growing threat of resistant infections.

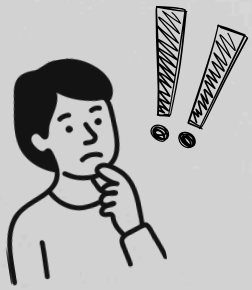
Since then, every year has emphasized educating the public, policymakers, and health professionals about the risks of misusing antibiotics and other antimicrobial medicines. This week also provides an opportunity to reflect on the progress made in combating AMR and to identify areas where further work is needed.



Antimicrobial Resistance

Antimicrobial Resistance (AMR) refers to the ability of microorganisms such as bacteria, viruses, parasites, and fungi to resist the effects of drugs that were originally designed to kill them or inhibit their growth. One of the main causes of this type of drug resistance is the self-medication or excessive use of antibiotics.

AMR is one of the most serious threats to public health today. With the excessive and improper use of antimicrobial drugs in humans and animals, as well as insufficient prevention of infections, this issue has worsened.



Numerous studies have shown a link between the use of antimicrobial drugs and resistance. Surveillance programs on antimicrobial drugs, and decrease AMR. When implemented alongside infection control measures-especially hygiene practice, these antimicrobial stewardship (AS) programs are more effective than when carried out alone. Coordinated, targeted strategies to prevent the spread of resistance organisms are extremely important for stopping the spread of resistance to multiple drugs.

Conclusion

Drug resistance to antibiotics is one of the main causes of death worldwide and is more frequently observed in low-income regions. The threats posed by antimicrobial resistance are global. To gain insight on the precise nature of the resistance predictors for the formation of drug resistance on different pathogens and combinations of pathogens, crucial correlations and patterns must be elucidated to assist in the development and the right potential measures for containment, and in the case of infection, to provide the right antibiotics, and in the prevention, to obtain new antibiotics and vaccines. There are still serious gaps in basic data, especially in low-income regions of the world, which underscores the urgency of broadening the collection of microbiology labs and data systems that are crucial to provide understanding on this fundamental aspect of the potential to improve the threats to human health.

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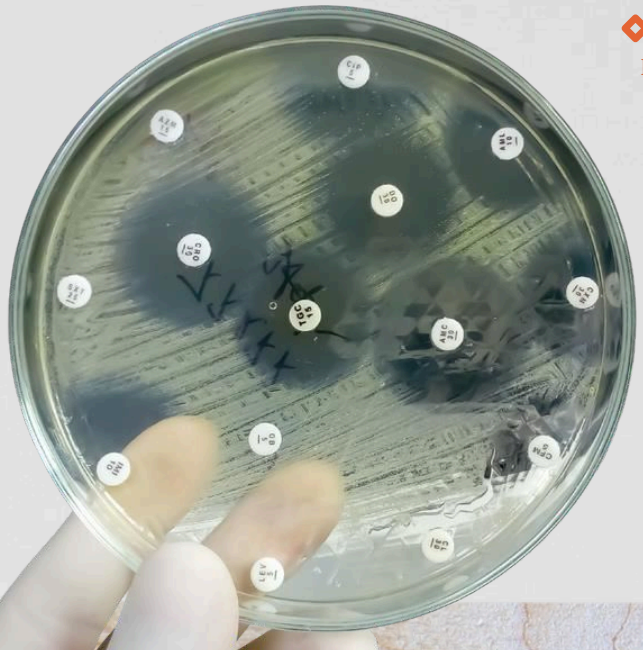
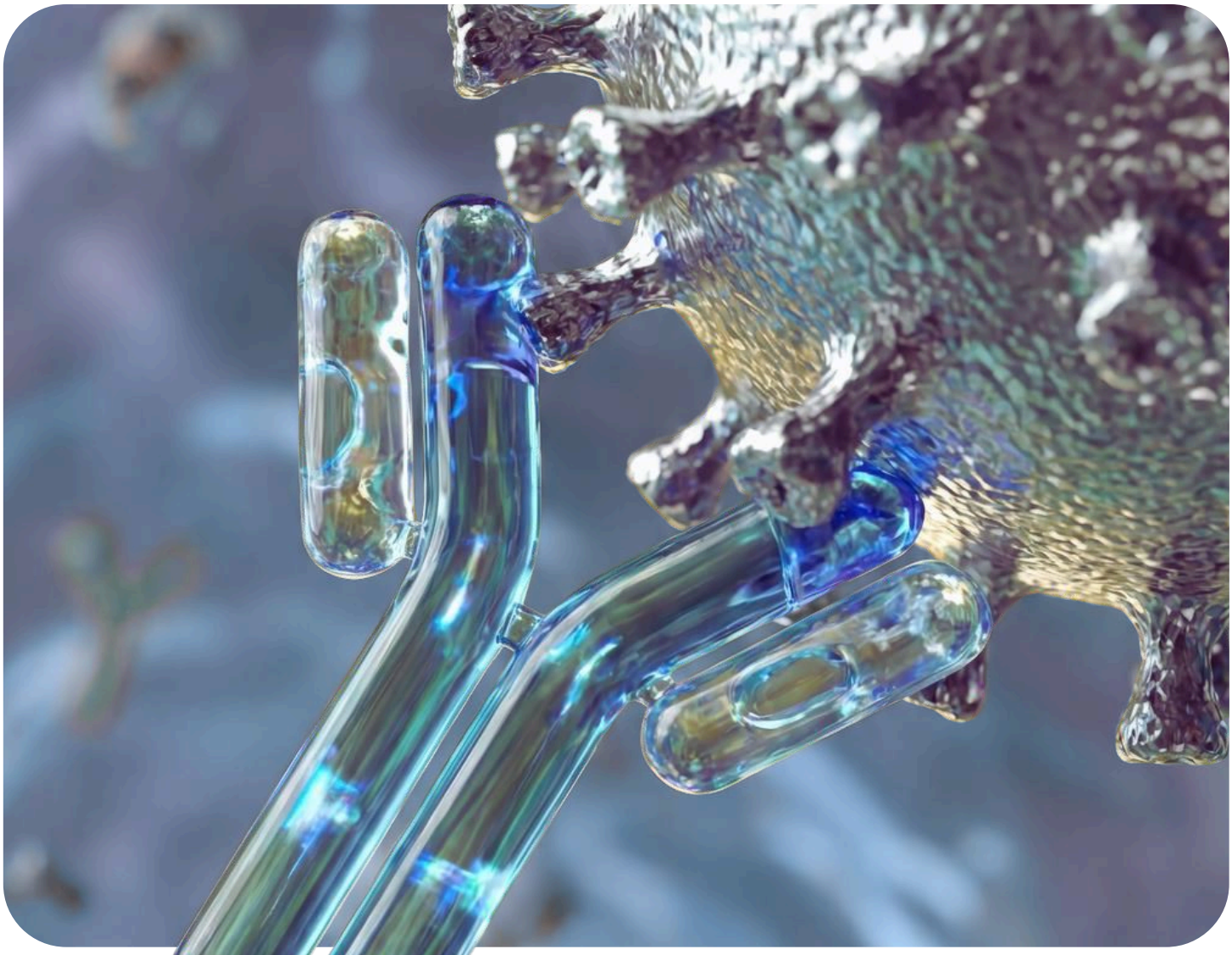




IMMUNOLOGY

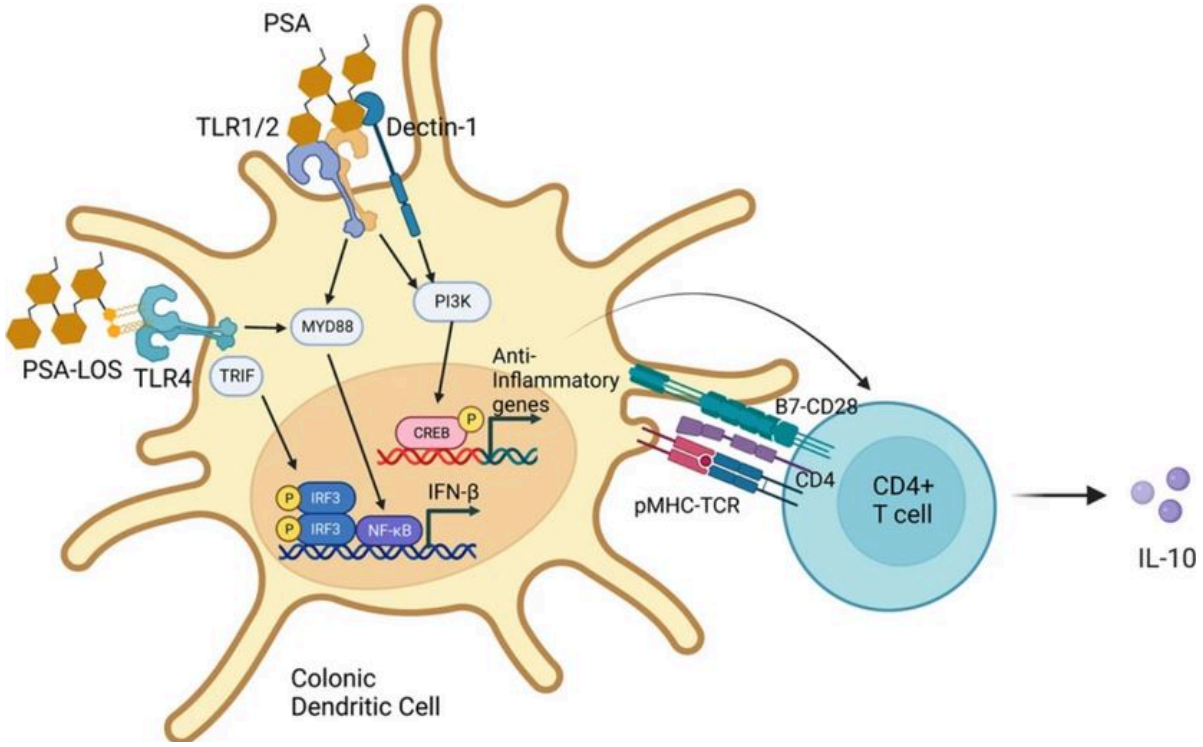
- Microbiome-driven Modulation of Macrophages and Dendritic Cells
- Nano Vaccine and Adjuvants Strategies for Enhanced Immunity

MICROBIOME DRIVEN MODULATION OF

MACROPHAGES AND DENDRITIC CELLS

The microbiome's impact on the immune system

Recently, the gut microbiome has emerged to play critical roles in regulating both innate and adaptive immune responses. Microbial metabolites may, in fact, condition the signaling and functional pathway of various immune cells, such as macrophages and dendritic cells (DC), toward functional polarizations in some cases, conditions that may be required to maintain mucosal homeostasis or for orchestration of proinflammatory responses or anti-tumoral immune responses.



Bacterial PSAs transduce signals through innate receptors to modulate immune responses:

TLR1/2 and Dectin-1 mediate PSA-induced PI3K activation, which leads to CREB-dependent transcription of anti-inflammatory genes in dendritic cells. This signaling pathway might drive dendritic cells to differentiate T cells into IL-10-producing cells. The LOS portion of PSA can bind to TLR4 and activate the MyD88-dependent NF-kB signaling pathway and the TRIF-dependent IRF3 signaling pathway, leading to the transcription of proinflammatory cytokines (e.g., IFN-β).

Immunomodulatory Effects of Short Chain Fatty Acids on Macrophage Polarization

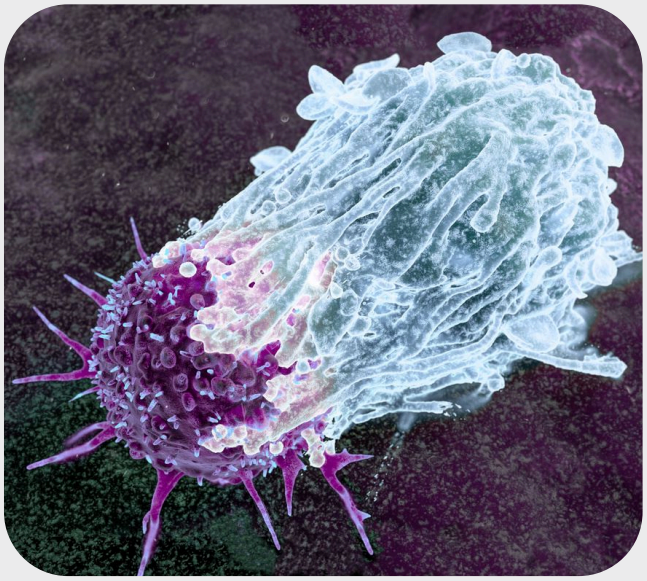
Short-chain fatty acids are likely potent immunomodulatory SCFAs produced from commensal bacteria, and in particular, these are butyric, propionic, and acetic acids. In addition, by SCFAs, the inhibition of NF-kappa B as well as STAT3 has shifted the activation of macrophages from pro-inflammatory M1 to non-inflammatory or wound-healing M2.

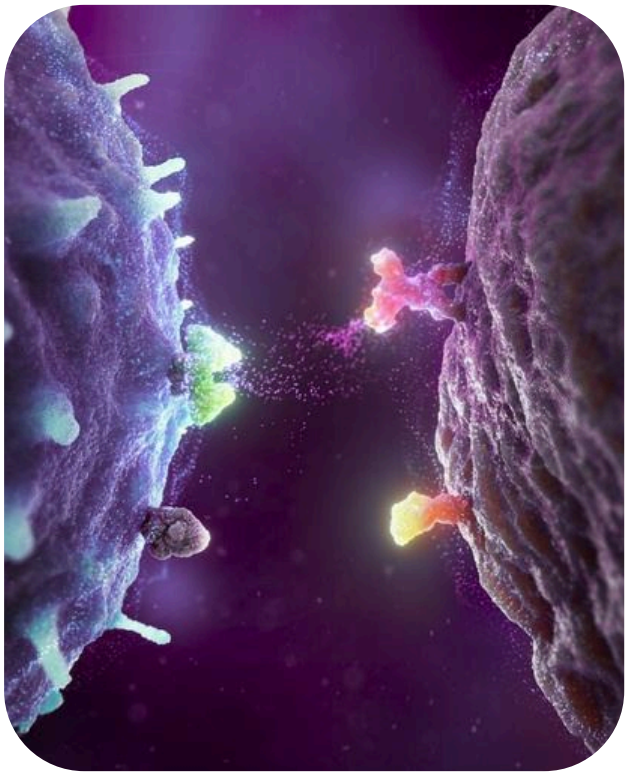
LPS-Induced Proinflammatory Activation of Macrophages in Dysbiosis

The bacterial LPS on the surfaces of the cells thus stimulates a pro-inflammatory pathway in macrophages by TLRs, the most-expressed being TLR4. Henceforth, the dysbiosis state is deemed associated with such overstimulation in several chronic diseases, including IBDs and metabolic syndrome.

Microbial Signaling Pathways Regulating Dendritic Cell Tolerogenicity

Dendritic cells is able to respond to microbial signals. G-coupled protein receptors in GPR43 and GPR109A would get activated, allowing tolerogenic DC phenotypes to develop as they will increase IL-10 secretion upon stimulation of regulatory T cells. Dysbiosis would cause DC maturation as it upregulates antigen presentation and initiates effector T cell differentiation, leaving the scene set for an inflammatory process.





Both modes illustrate the ambivalent role of microbiomes on the one hand with tolerance, and on the other hand with triggering of immune-mediated diseases.

It would, however, mean that knowledge of cell mechanisms would further drive development of therapeutics centered on the microbiome that perhaps inhibit chronic inflammation, restore immunity, and even create strong anti-tumor immunity.

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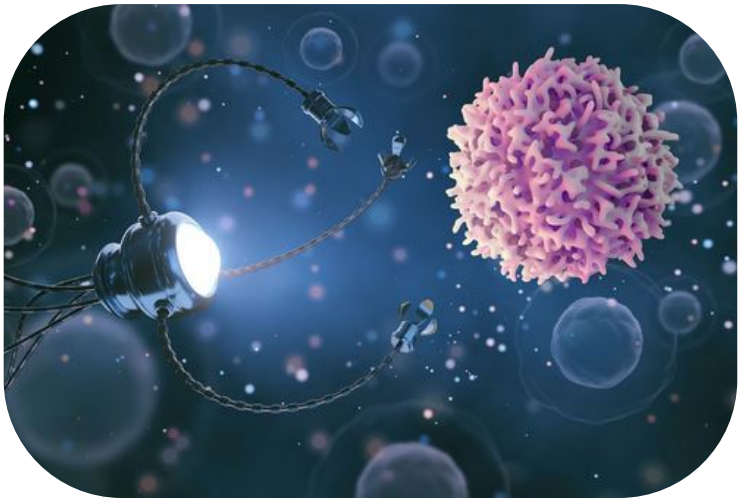
NANO VACCINE AND ADJUVANT STRATEGIES

FOR ENHANCED IMMUNITY

Nanotechnology increasingly plays a significant role in vaccine development. In recent years, much attention has been brought to the nanovaccines strategy due to their possibilities of enhancing and tailoring immune responses with higher efficiency compared with traditional vaccines. Unlike conventional vaccines, which rely on bulk antigen delivery, nanovaccines protect antigens from degradation, facilitate cellular uptake, and promote targeted delivery to immune cells by using nanoscale carriers.



The carriers can be represented by lipid nanoparticles, polymeric particles, virus-like particles, or inorganic nanomaterials. Due to the nanoscale size, they can efficiently interact with dendritic cells and lymphoid tissues, providing more precise and controlled activation of immunity compared to standard formulations.

The role of Nanoadjuvants

In many nanovaccine formulations, the nano-adjuvant nanostructured substance that strengthens the immune response without altering the antigen itself, plays a very fundamental role. For instance, recent mRNA vaccines have demonstrated how lipid-based nanoparticles could improve the stability of mRNA vaccines and allow for efficient cytosolic delivery of the vaccine.

Moreover, a nano-adjuvant was recently identified that could dynamically integrate two waves of innate immune stimuli into effective antitumour immunity without immune-cell exhaustion.

Moreover, research has been performed on a multi-functional nanoadjuvant that couples manganese with Toll-Like 9 agonist, stimulating potent innate and adaptive anti-tumor immunity.

This tunability makes nanovaccines a promising platform for a variety of emerging infectious diseases, cancer immunotherapies, and personalized medicine.

Overall, nanovaccines and nano-adjuvants represent versatile and potent strategies that can enhance vaccine performance. Given their potential to optimize antigen delivery, control immune activation, and reduce side effects, they represent one of the key next-generation vaccination approaches.

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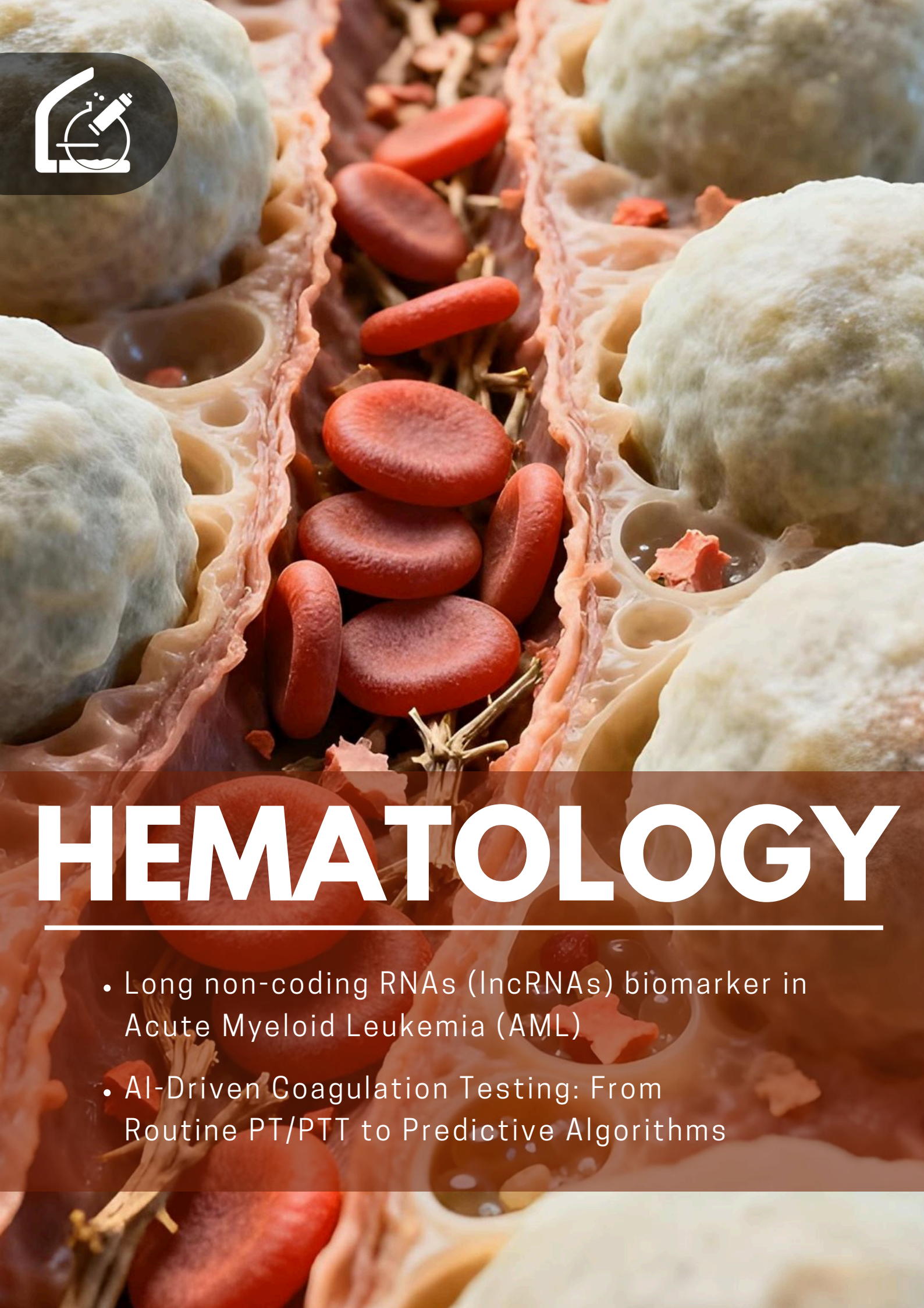
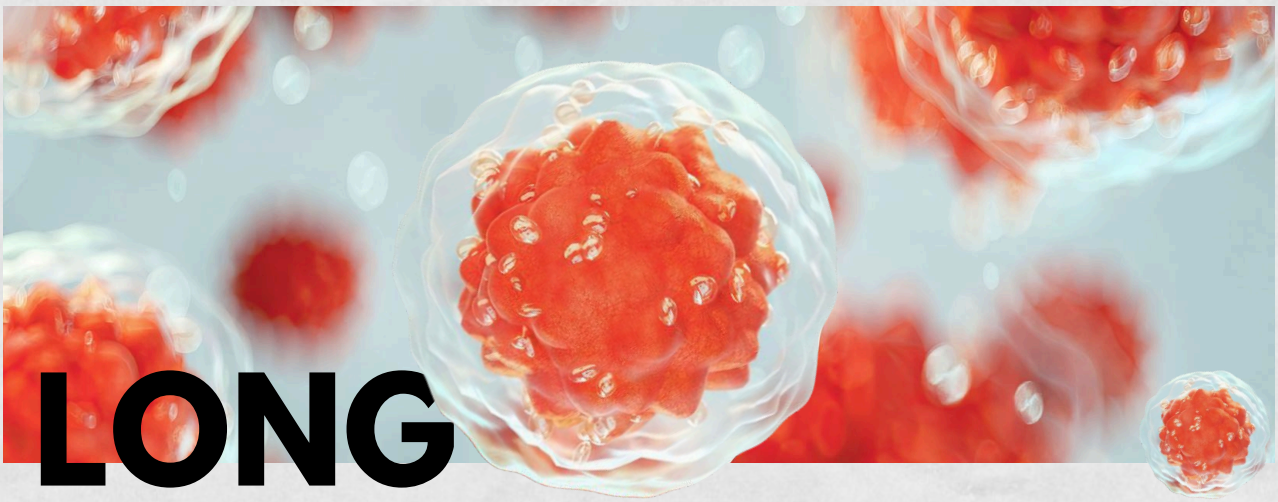


HEMATOLOGY

- Long non-coding RNAs (lncRNAs) biomarker in Acute Myeloid Leukemia (AML)
- AI-Driven Coagulation Testing: From Routine PT/PTT to Predictive Algorithms



LONG NONCODING RNAS (LNCRNAS)

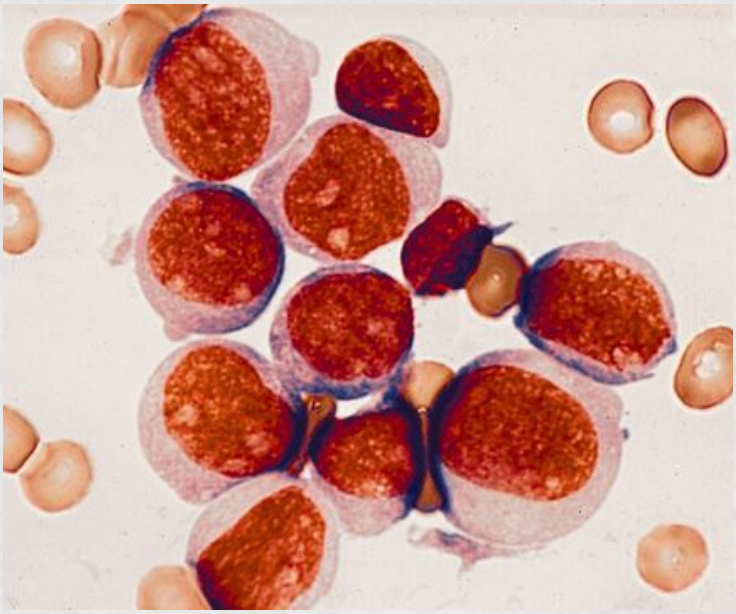
biomarker in acute myeloid leukemia (AML)

Introduction:

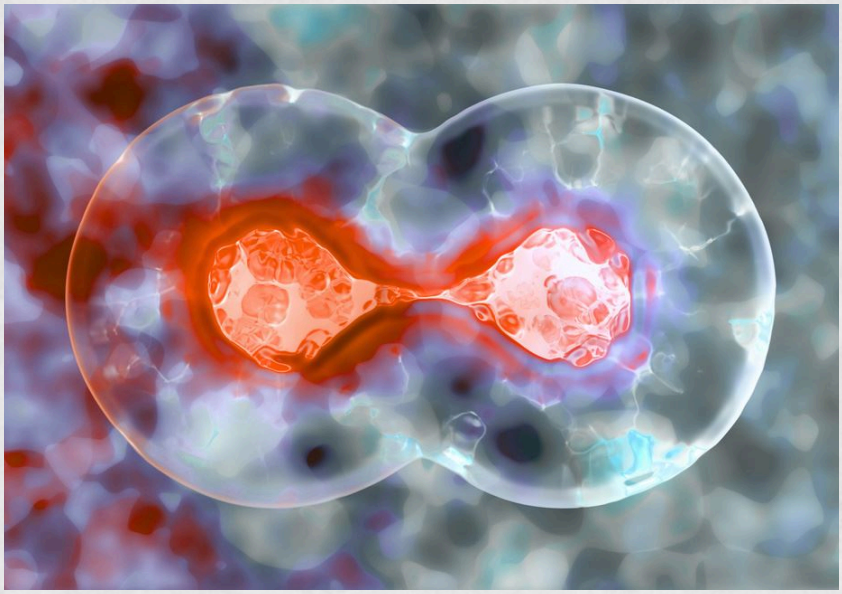
Acute myeloid leukemia AML is a heterogeneous hematologic cancer characterized by blocked myeloid differentiation and accumulation of immature myeloid cells. Various genetic, cytogenetic, and epigenetic alterations have been implicated in its pathogenesis over the past decades. Of late, lncRNAs have been increasingly recognized to be implicated in AML as well as other cancers. These molecules play a role in key biological processes that lead to the differentiation of normal haematopoietic stem cells to leukaemic blasts. LncRNAs participate in crucial signaling pathways, including IGF-1R, FLT3, c-KIT, Wnt, and PI3K/AKT, and control actions like apoptosis, autophagy, as well as glucose metabolism. Due to the critical biological functions, lncRNAs are becoming a novel indicator for diagnosis, prognosis, and drug resistance prediction of AML, which might contribute to the development of precision therapy for AML.

Roles of lncRNAs in Acute Myeloid Leukemia:

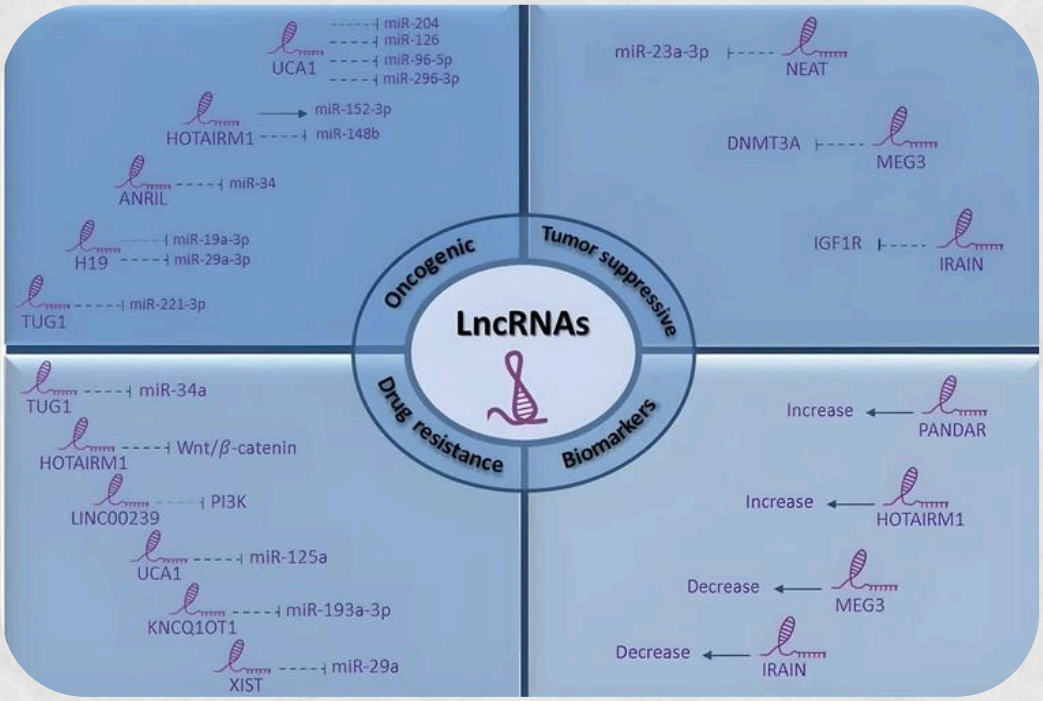
Long noncoding RNAs (lncRNAs) have been implicated in the pathophysiology of acute myeloid leukemia (AML), functioning as essential modulators of different cellular functions. LncRNA expression profiles correlated with distinct genetic AML subtypes. The observations that distinctive lncRNAs were predominantly dys-regulated in different AML subtypes have implications for the biological complexity of such subtype-specific genotypes. Collectively, such molecules affect key signaling pathways and can either constitute prognostic signatures to categorize AML patients or 'order' the biology of leukemic stem cells.



lncRNAs play crucial roles in proliferation, differentiation, and therapy resistance, and are emerging as promising biomarkers and therapeutic targets. They are misregulated through genetic mutations, chromosomal translocations, and epigenetic modifications. In AML, lncRNAs also influence differentiation by modifying the expression of transcription factors, genome stability, and the regulation of oncogenes and tumor suppressors at an epigenetic level.



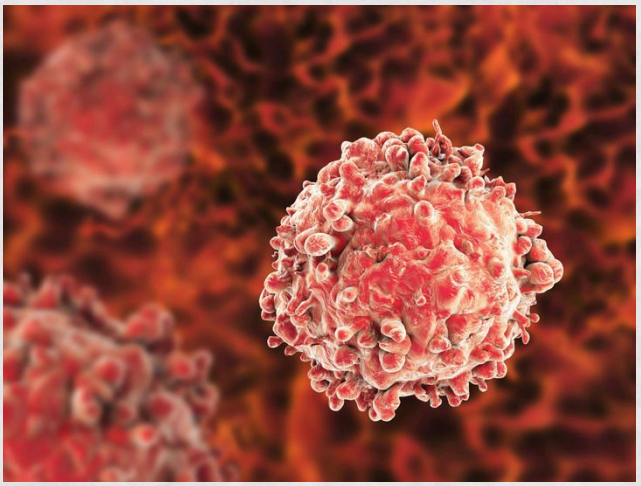
Therapy targeting lncRNAs may offer a selective therapeutic approach with lower toxicity towards normal dividing cells in comparison to conventional chemotherapy. Despite that the majority of studies is being conducted on ex vivo models, and many mechanisms have not been unraveled yet, the potential of lncRNAs as promising diagnostic and prognostic biomarkers in AML is growing.



LncRNAs in acute myeloid leukemia. Well-known examples of lncRNAs and how they influence key genes and processes, are involved in tumor development and tumor suppression. The solid lines show the results of lncRNA expression, while the dashed lines identify pathways that are decreased upon lncRNA expression

In summary:

lncRNAs are involved in the regulation of cell differentiation, proliferation, and survival in AML. Their distinctive expression patterns make them promising biomarkers for AML diagnosis, prognosis, and therapy resistance, providing promising potential for refining precision medicine in AML treatment.



Parisa Nezafati

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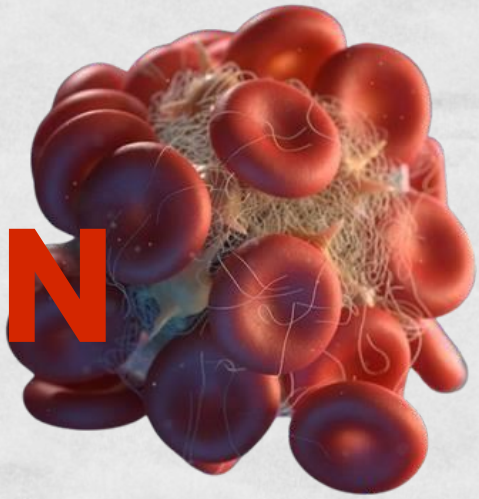
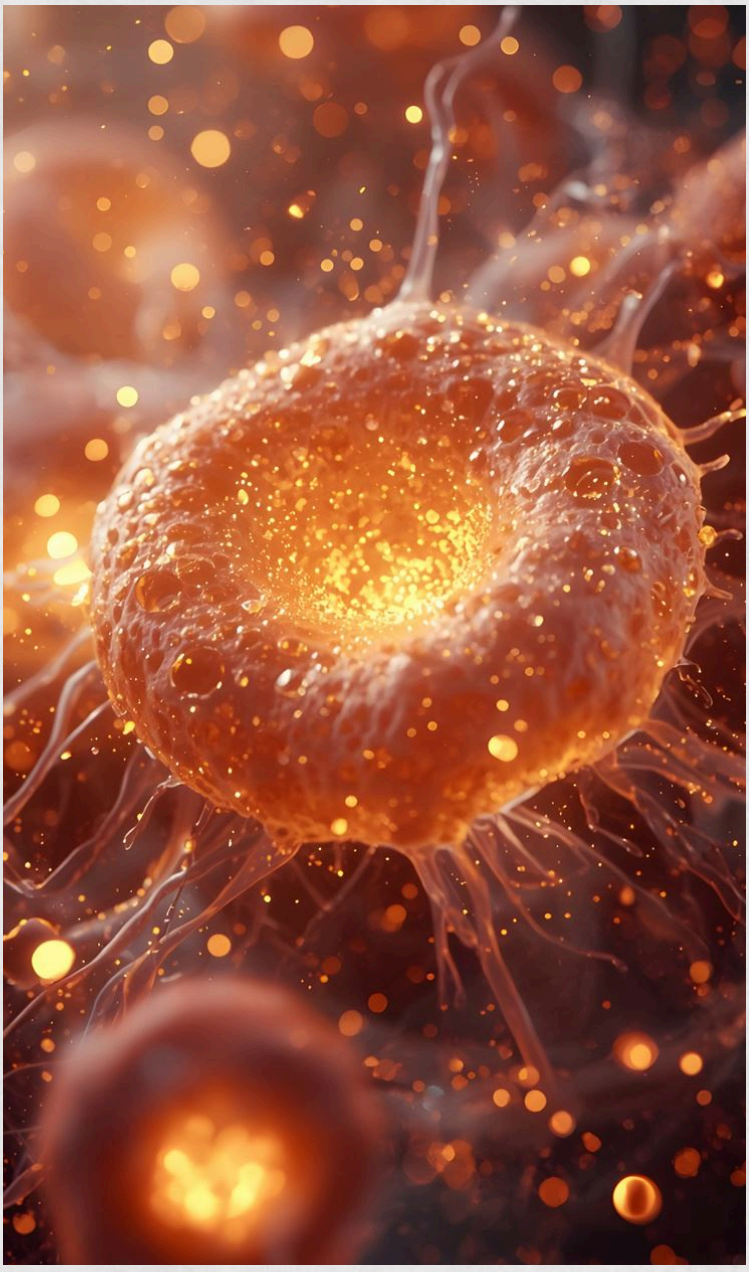
AI DRIVEN COAGULATION TESTING



From Routine PT/aPTT to Predictive Algorithms

Abstract

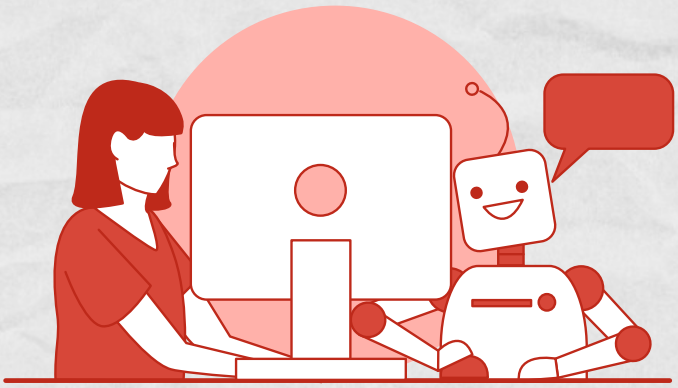
The accurate evaluation of coagulation function is a key factor in the prevention of not only bleeding but also thrombotic events. Coagulation tests such as PT and aPTT are helpful, but still cannot depict the characteristics and the complexity of coagulation dynamics in detail. Over the last ten years, AI/ML technologies have opened up a new way of diagnosing disorders of coagulation through their ability to amalgamate and analyze large quantities of clinical and laboratory data, giving better predictions of the disorders.



Introduction

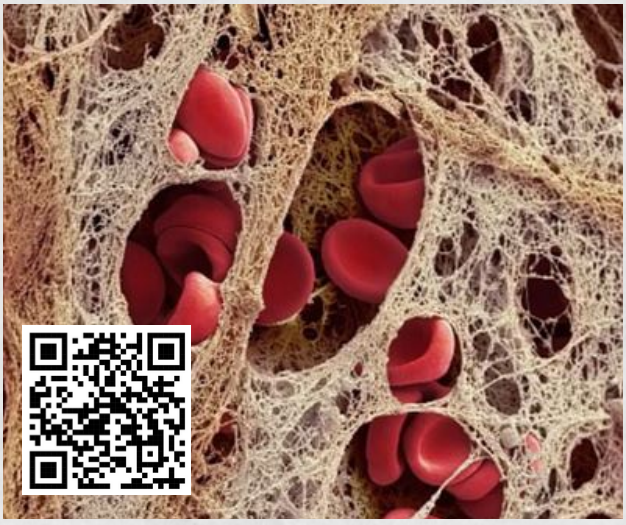
The most common method of testing coagulation is the time measurement of the clot formation in plasma samples. However, the provision of a mere trifle of the situation of hemostasis is the only output of these tests, and in addition, they are usually affected by the pre-analytical and patient-specific factors.

The traditional techniques for evaluating coagulation disorders have been altered by AI-based systems, as they have started to play an important role in this area with the advent of precision medicine.



Applications of AI in Coagulation Testing

Algorithms based on machine learning are utilizing variables such as the number of platelets, levels of fibrinogen, inflammatory markers, and clinical history to create predictive models for disorders like DIC and heparin resistance. Different AI models, such as random forests and neural networks, have shown that their implementation can lead to better results than traditional statistical methods in terms of identifying abnormal coagulation patterns and predicting patient outcomes.



Clinical Significance

One of the most significant is heparin monitoring; that is the case for machine learning. By monitoring the trends in the levels of aPTT and Anti-Xa, the machine can provide the necessary dosage. That will help to avoid the risk of bleeding or thrombosis in patients who are critically ill. AI algorithms are already being developed.

Conclusion

Despite such progress, AI tools still need to be validated across populations, and standardization is required before they replace conventional diagnostic practices. However, AI-based coagulation testing is a major step toward personalized patient management and rapid, evidence-based decision-making in laboratory medicine.

Aylin Nobari

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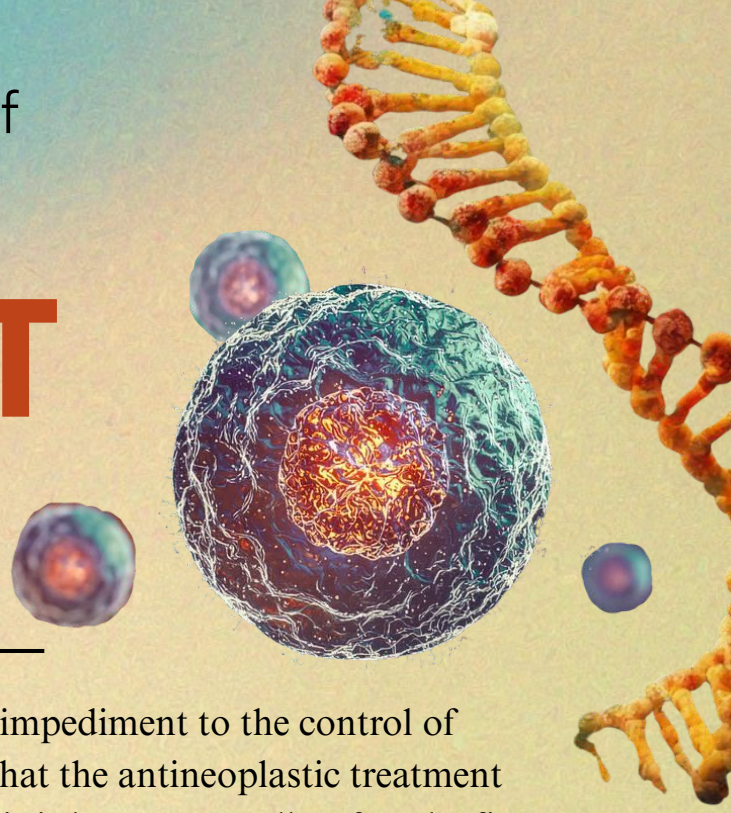
GENETICS

- The Persistent challenges of Treatment-Resistant Cancers
- Revolutionizing Personalized Gene Therapy with AI

The Persistent Challenge of

TREATMENT RESISTANT CANCERS



The scenario of resistant cancers is still a major impediment to the control of many cancers despite the revolutionary change that the antineoplastic treatment has undergone over the last forty years or so. This is because usually, after the first treatment, a patient gets better, but then the cancer turns on some resistance mechanisms and continues its growth.

From Genetic Mutations to Epigenetics: A New Focus in Drug Resistance

At first, researchers were looking at genetic mutations as the main culprit for resistance. Recently, however, the field of epigenetics has attracted the most attention. The latter includes changes that result in the modulation of the gene's activity without the necessity of altering the nucleotide sequence or DNA.

Epigenetic Regulators: Reversible Switches That Drive Cancer Survival

Epigenetic regulators, which consist of proteins and enzymes, are responsible for the change in the DNA's structure within the cell and thereby controlling the expression of the genes by turning them on or off.

A misplacement of such regulators can cause the tumor suppressor genes to be turned off, or it can activate the pathways that support the cancer cells in surviving the treatment. The good thing about these changes is that they can be reverted; hence, drawing attention to epigenetic regulators might open the potential of "reprogramming" the cancer cell and making it sensitive to treatment again.

Epigenetic Therapies: DNMT and HDAC Inhibitors in Overcoming Resistance

The combination of epigenetic treatment with standard ones has been reported by several clinical trials to result in a general enhancement of patient outcomes in hematological malignancies (especially AML and MDS) and is showing highly promising results in solid tumors (lung, breast, colorectal, and melanoma).

These agents could bring back the dormant genes and thus make the cancer cells more sensitive to chemotherapy, immunotherapy, or other targeted therapies.

Clinical Success of Combining Epigenetic Drugs with Standard Treatments

There are already some drugs that are aimed at the epigenome. DNMT inhibitors (e.g., azacitidine, decitabine) and HDAC inhibitors (e.g., vorinostat, romidepsin, panobinostat) are possibly the most studied agents among epigenetic drugs.

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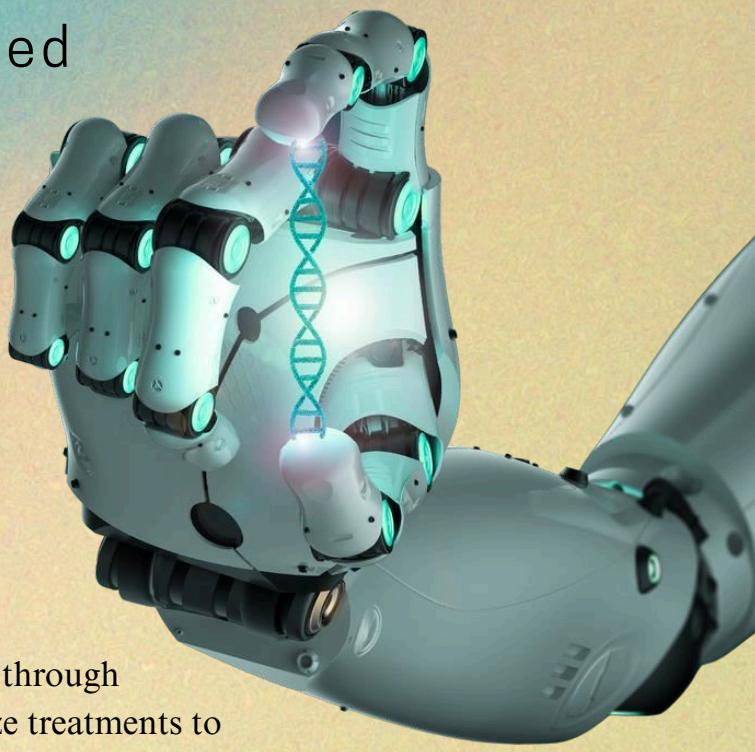
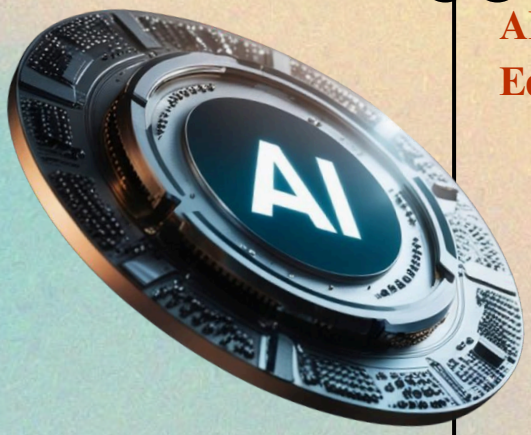
Revolutionizing Personalized

GENE THERAPY WITH AI



Personalized gene therapy is being revolutionized through Artificial Intelligence (AI) that can help personalize treatments to a patient's individual genetic needs. Such integration promotes higher specificity, better therapeutic effect, and fewer safety issues than those of conventional gene therapies. The use of AI will transform patient health from the time a clinical diagnosis is determined through the process of developing targeted treatments, marking a paradigmatic shift in how genetic diseases are monitored.



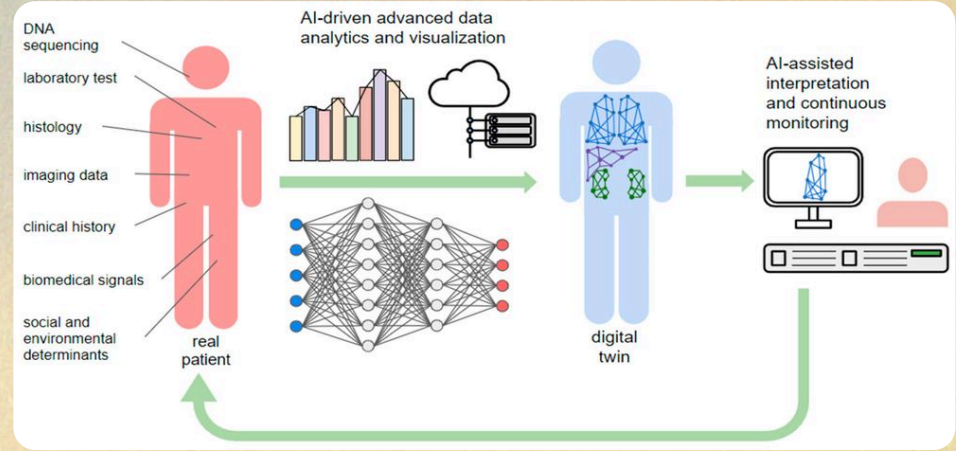
AI in Detecting Genetic Variants and Enhancing Gene Editing

AI is essential in the detection of disease-related genetic variants, as it uses various ML techniques applied to large genomic data sets. AI's ability to analyze large quantities of information helps in the development and perfection of gene-editing tools like CRISPR-Cas9, ensuring highly targeted changes would be made to a person's DNA with minimal off-target effects.

The Role of Digital Twins in Advancing Gene Therapy

One particularly exciting area of progress in this field is the development and application of digital twins 3D, virtual models of patients created using computer learning algorithms. Such a digital twin would mirror the genetic and biological context of an individual, giving clinicians and researchers a testing platform to try out gene therapies before moving ahead in real patients.

Digital twins predict by modeling the effects on a patient's biology of different gene edits, and can also help design treatment strategies that work best.

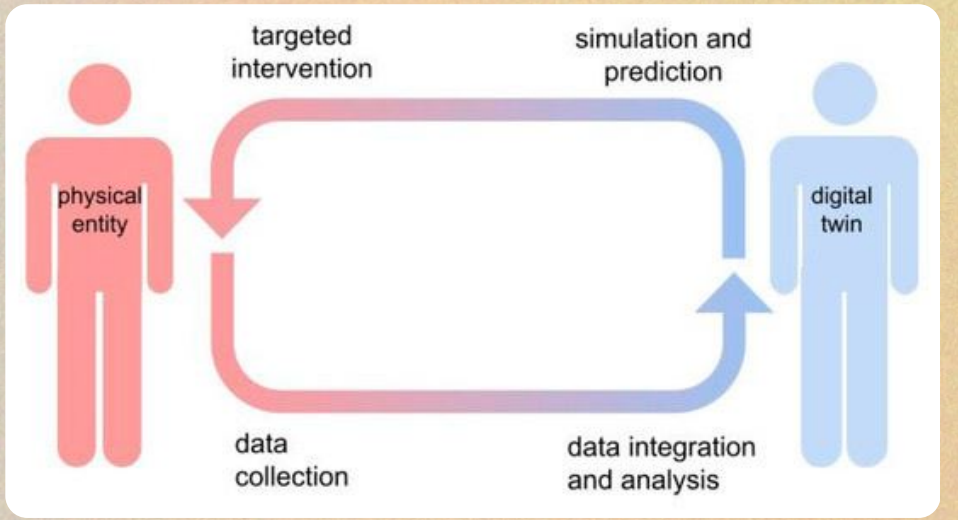


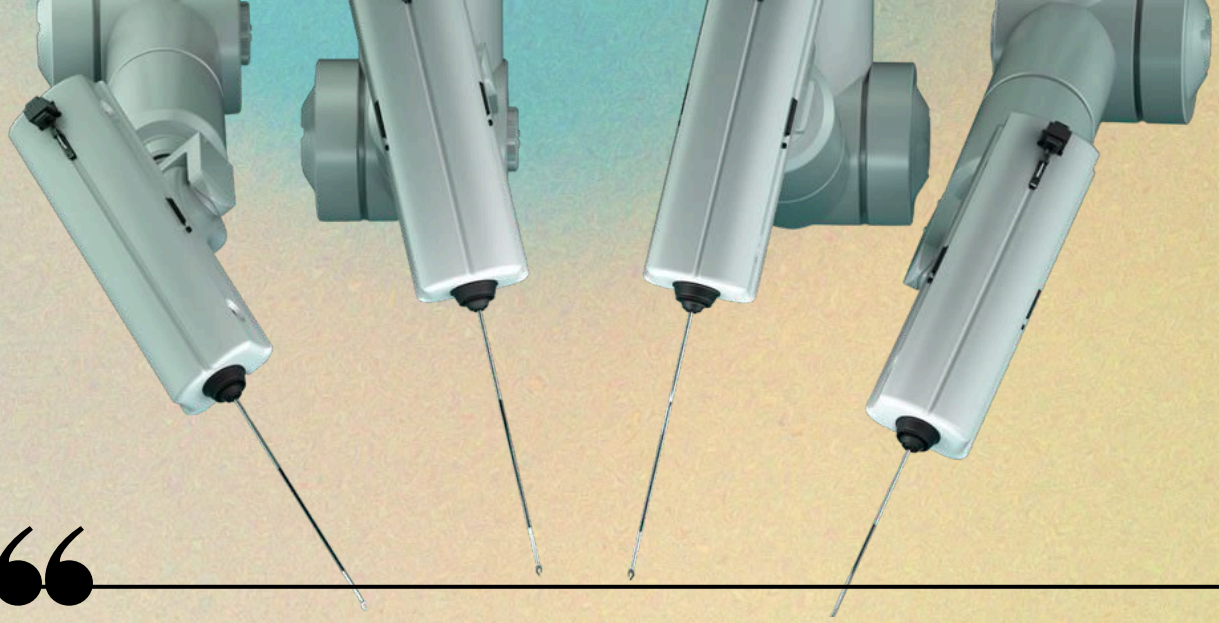
This method dramatically speeds the drug development process, avoiding expensive and time-consuming trial and error testing on real patients.

Real-Time Monitoring of Gene Therapy with AI and Digital Twins

In addition, AI is crucial in the real-time tracking of gene therapy effects. Combining the digital twin with wearable sensors and AI analysis enables doctors to monitor a patient's response to therapy from the molecular level. This feedback loop in real-time would mean that treatments would be able to be dynamically targeted for maximum benefit and minimal harm.

This fine level of surveillance allows for personalized medicine, where the next treatment step is adapted in a continuous manner according to each patient's individual genetic situation and therapeutic prospect.





The Future of Personalized Gene Therapy with AI

In Conclusion, the use of AI and digital twins in individualized gene therapy will revolutionize the management of genetic diseases. By fine genetic editing, the best gene delivery, and real-time monitoring, AI helps ensure more effective and safe therapy. The future of gene therapy is becoming more personalized by the day, with opportunities to finally treat and cure genetic diseases with pinpoint accuracy.

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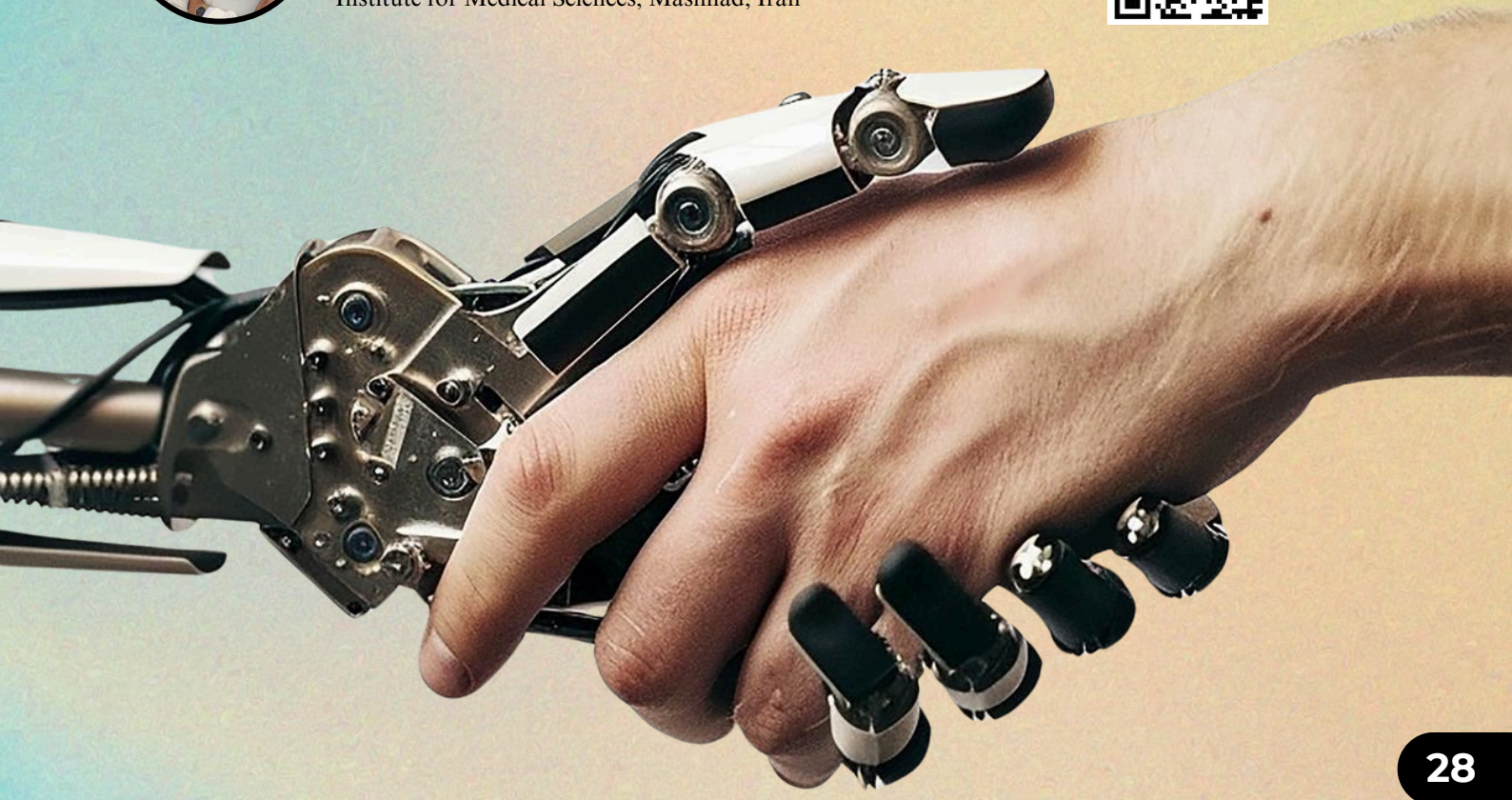
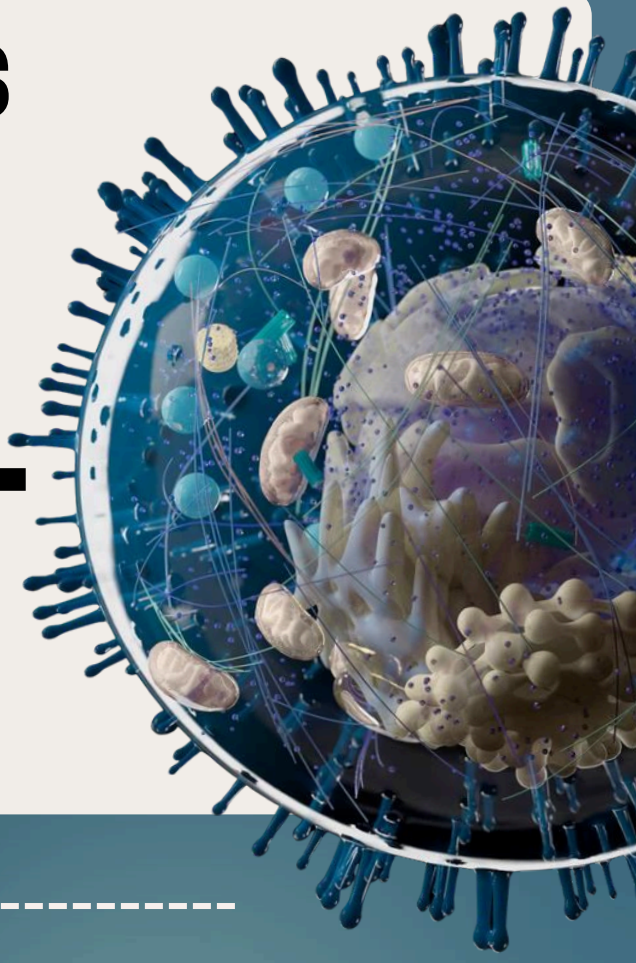


MICROBIOLOGY

- The Persistent challenges of Treatment-Resistant Cancers
- Revolutionizing Personalized Gene Therapy with AI

NANOPARTICLES A NEW APPROACH TO COMBAT

Antibiotic Resistance and the Challenges Ahead

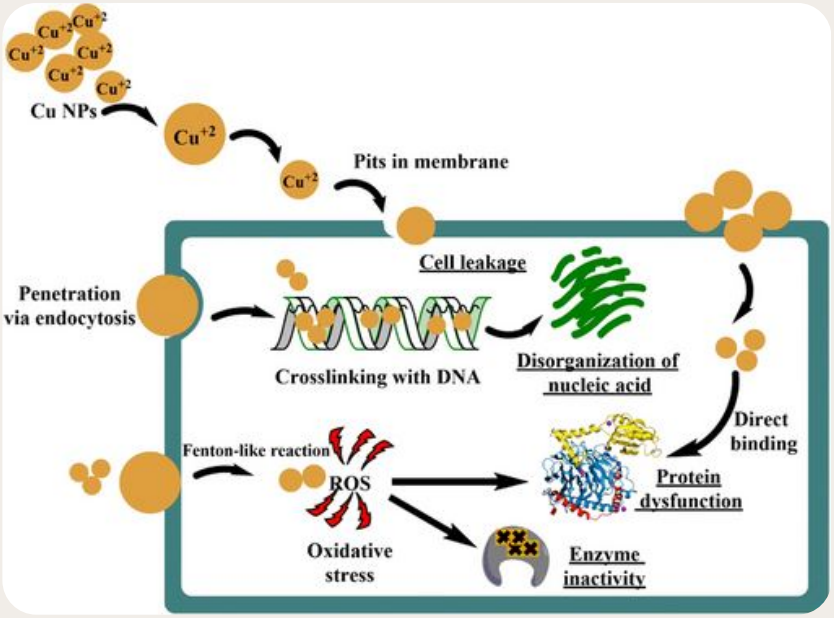


Abstract

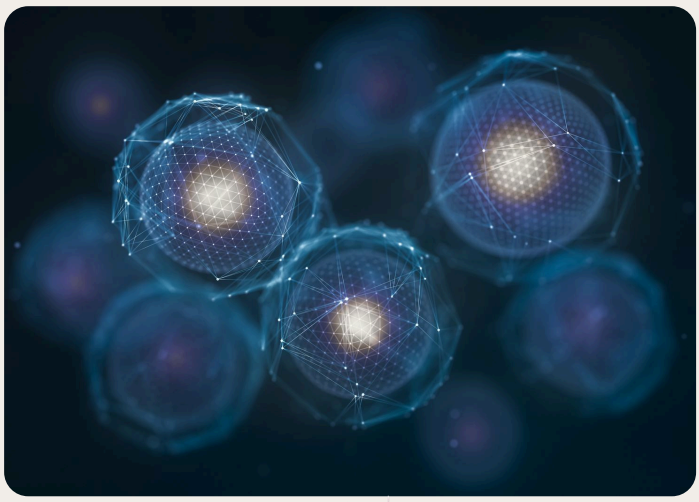
The rapid and universal development of antibiotic resistance in pathogenic bacteria is causing increases in morbidity and mortality rates; the multidrug-resistant pathogens have rendered many conventional antibiotics ineffective against infections that were manageable in the past. Under such circumstances, nanotechnology, specifically metallic and metal oxide nanoparticles, has shown potential as an effective treatment regimen due to physicochemical and antimicrobial properties.

Antimicrobial Mechanisms of Nanoparticles

Nanoparticles can invoke an antimicrobial effect through more than one mechanism. Plasmonic nanoparticles can utilize this property to manifest surface plasmon resonance, whereby gold and silver plasmons in authorities can also be rendered in conjunction with antibodies, phages, or aptamers in bacterial detection.

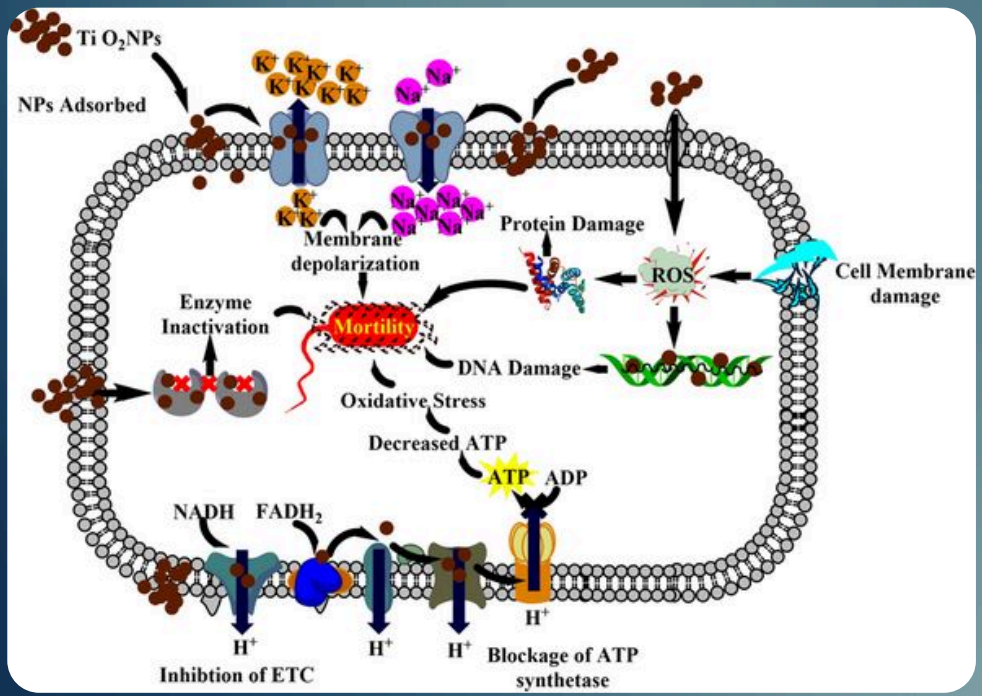


ZnO nanoparticles are most often cited as being effective under action, antibacterial, and other antioxidant conditions. They are effective against many Gram-positive and Gram-negative bacteria, depending on the size and concentration of particles

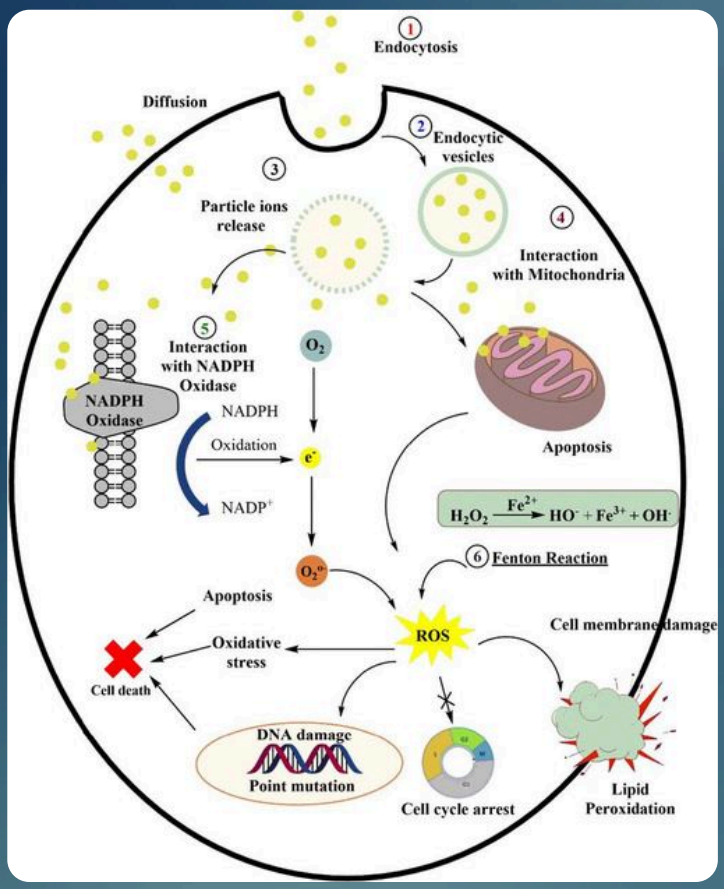


These nanoparticles disrupt cell membranes, induce reactive oxygen species, and interfere with critical biomolecules. Copper nanoparticles (CuNPs) display antimicrobial action against several important foodborne pathogens through the release of copper ions that can bind to the functional groups in proteins and create membrane pores, thereby ultimately inducing DNA damage.mbrane, with or without UV irradiation

Green synthetic methods can suppress oxidation in blue-colored CuNPs. Other metal oxide nanoparticles, such as Cr_2O_3, Co_3O_4, and Mn_2O_3, induce oxidative stress by oxidizing cytochrome c and NADPH. On the other hand, TiO_2 nanoparticles produce size- and zeta potential-dependent bactericidal actions by generation of ROS while destabilizing the plasma me



Below is a brief summary of the antibacterial efficacy against E. coli, S. aureus, and Klebsiella pneumoniae through cobalt nanoparticles enabled via the quantum size effect and catalytic properties



Challenges and Toxicological Considerations

Although these nanoparticles have great therapeutic promise, in their synthesis, application, and disposal, there is immense potential for environmental accumulation along with promoting microbial mutation and the horizontal spread of antibiotic resistance genes (ARGs).

Continuous exposure to nanoparticles could activate bacterial competence and SOS pathways, possibly facilitating resistance development. All the toxicological outcomes vary in a way dependent on the dose, time exposed, and the route used in introduction, which can be dermal, inhalation of gases, oral, or the intravenous route, with the last being the most dangerous.

Conclusion

Biogenic metal nanoparticles are tunable in size, shape, and surface characteristics. The nanoparticles produce very strong antibacterial efficacy through membrane disruption, ROS-mediated intracellular injury, and metabolic inhibition. More interdisciplinary research is needed for sustainable nanotechnology use against antibiotic resistance.

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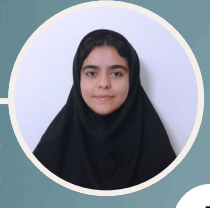
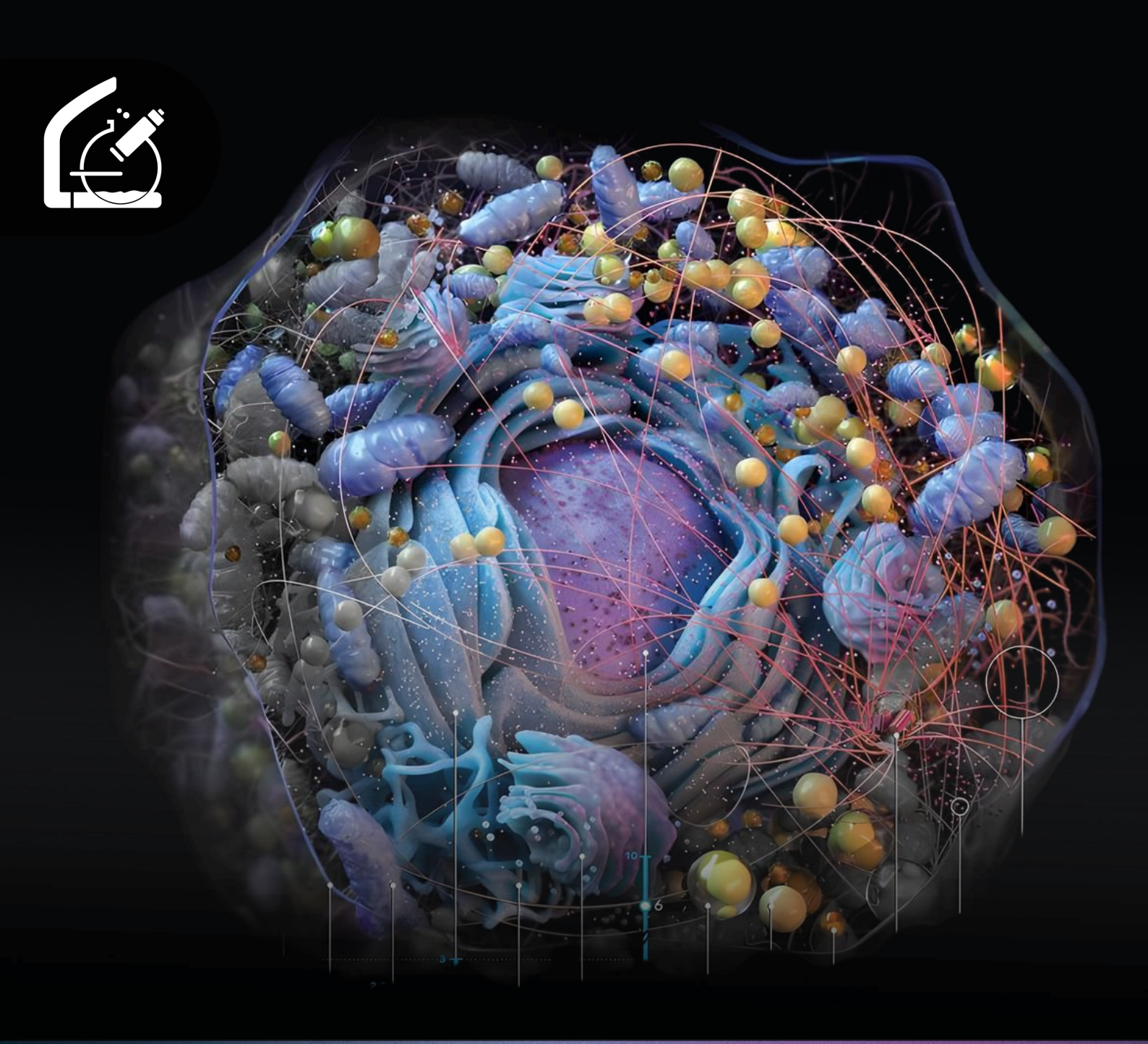
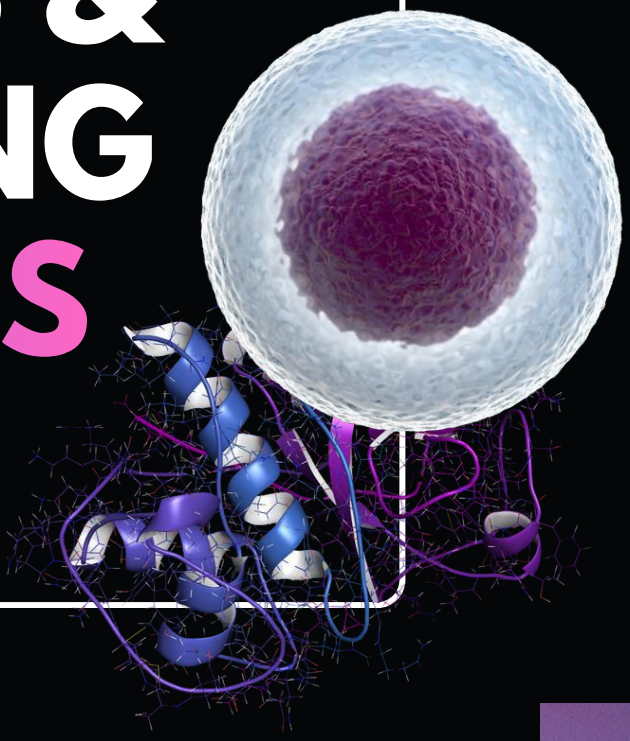




BIOCHEMISTRY

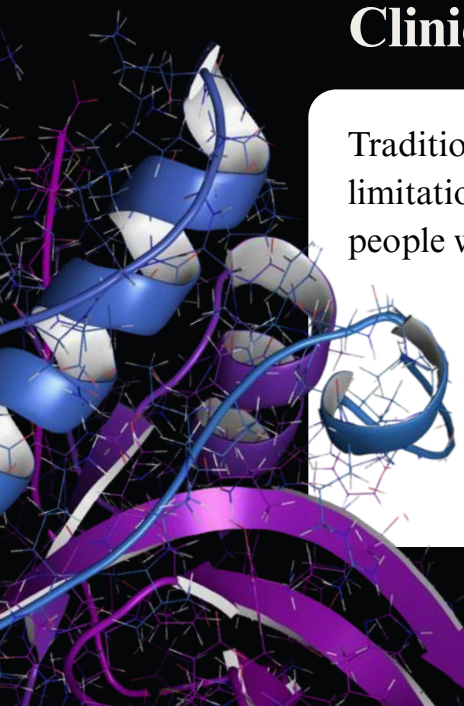
- Chemical Biomarkers in Diagnosis & Monitoring of Diabetes Mellitus

CHEMICAL BIOMARKERS IN DIAGNOSIS & MONITORING OF DIABETES MELLITUS



Diabetes Mellitus, Diagnosis and Monitoring

Diabetes mellitus is a complex, chronic illness characterized by elevated blood glucose levels that occurs when there is cellular resistance to insulin action, pancreatic β-cells do not produce sufficient insulin, or both. Implementing successful and cost-effective strategies for systematic screening of diabetes mellitus is imperative to ensure early detection, lowering patients' risk of developing life-threatening disease complications. Therefore, identifying new biomarkers and assay methods for diabetes mellitus to establish robust, non-invasive, painless, highly sensitive, and precise screening techniques is essential.



Clinically Validated Biomarkers

Traditional glycemic markers, such as glucose and HbA1c, present several limitations that can lead to underdiagnosis and poor disease prognosis in people with Type 2 diabetes(T2DM).

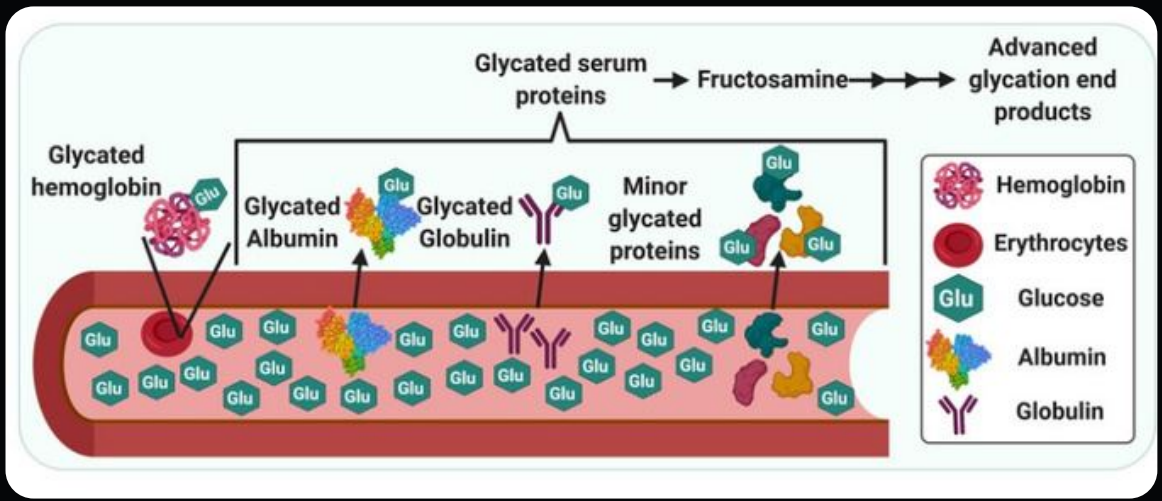
Evidence from studies comparing the performance of new glycemic markers such as glycated albumin (GA), fructosamine (FA), and 1,5-anhydroglucitol (1,5-AHG) has shown that they provide independent clinical information and can improve the prognostic value of conventional markers.

These intermediate markers can be used to determine the risk of T2DM and its complications independently of fasting blood glucose and HbA1c values.

The moderate correlation and clinical variations between nontraditional markers such as GA, FA, and 1,5-AHG and conventional markers might be due to the fact that they are more strongly influenced by postprandial excursions than HbA1c, which is more affected by long-term glycemia as well as by the differential effect of oxidative stress.

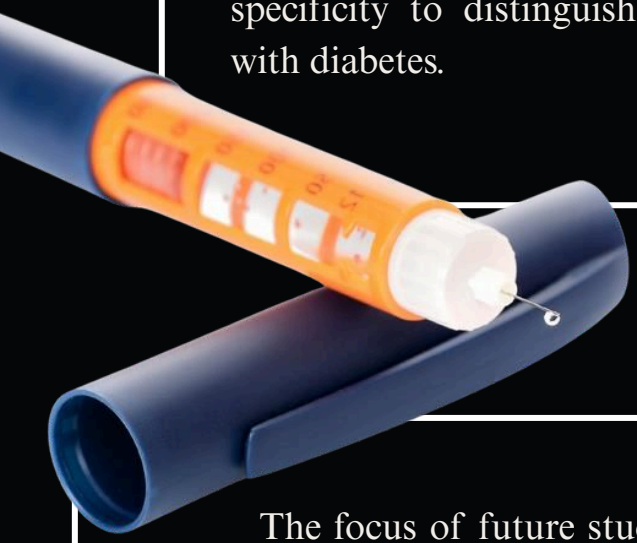
Fructosamine

Fructosamine refers to all stable keto amines produced through the non-enzymatic glycation of circulating serum proteins. The concentration of FA in serum increases in T2DM due to the higher sugar concentration in the blood. Therefore, it could be useful as a glycemic marker that allows discrimination between normoglycemic individuals and those with diabetes. Also, its application as a biomarker for screening or diagnosis of gestational diabetes mellitus compared against the OGTT has been reported.



A Graphical representation of the mechanism by which glycated proteins and fructosamine correlate to hyperglycemia.

Unlike the determination of HbA1c, which measures long-term changes because of the longer circulating lifetime of hemoglobin, FA reflects glucose levels over 2 to 3 weeks. Several studies have shown strong correlations between FA and HbA1c in T2DM with high sensitivity and specificity to distinguish between normoglycemic individuals and those with diabetes.



The focus of future studies should be on observing the relationship between FA and the onset of diabetes complications to assess the marker's potential as a risk indicator in already diagnosed patients.

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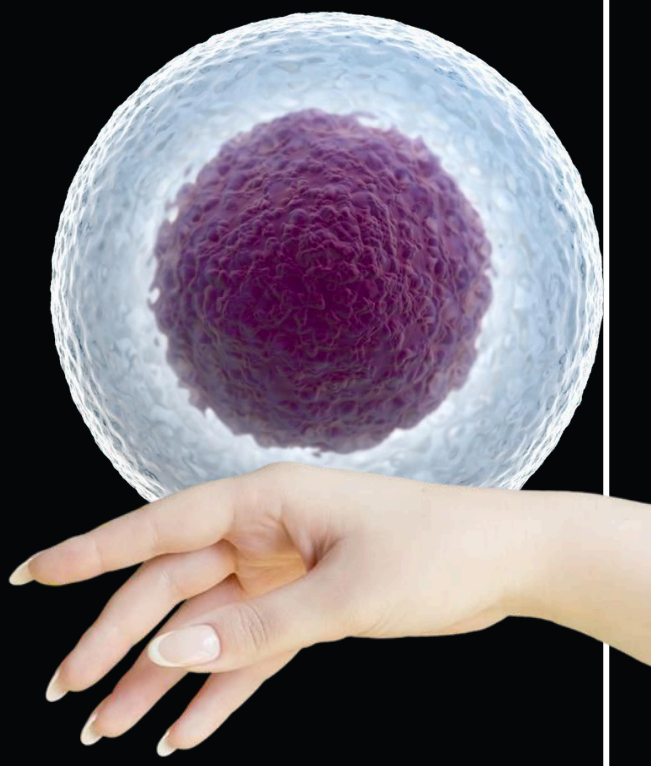
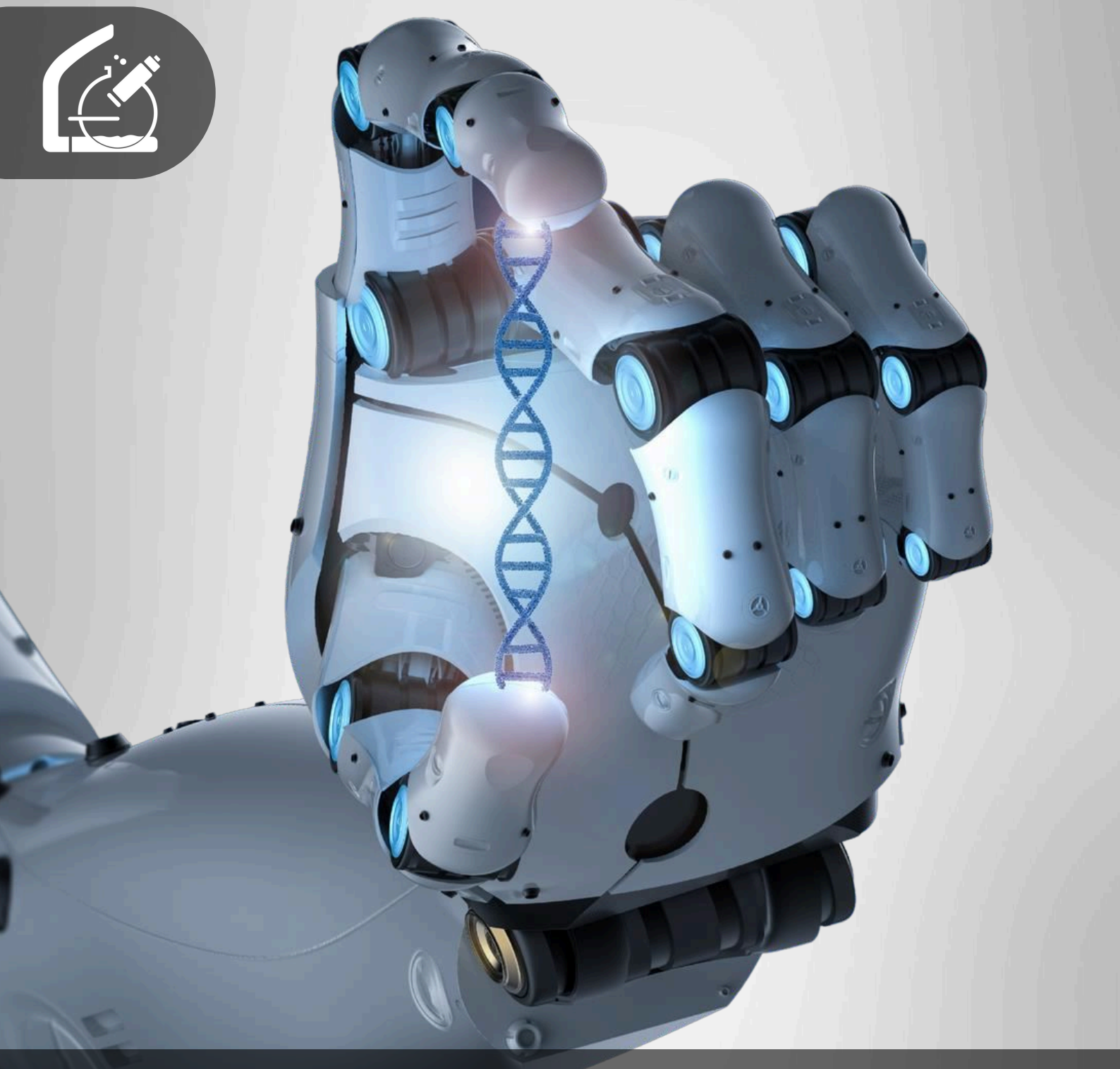


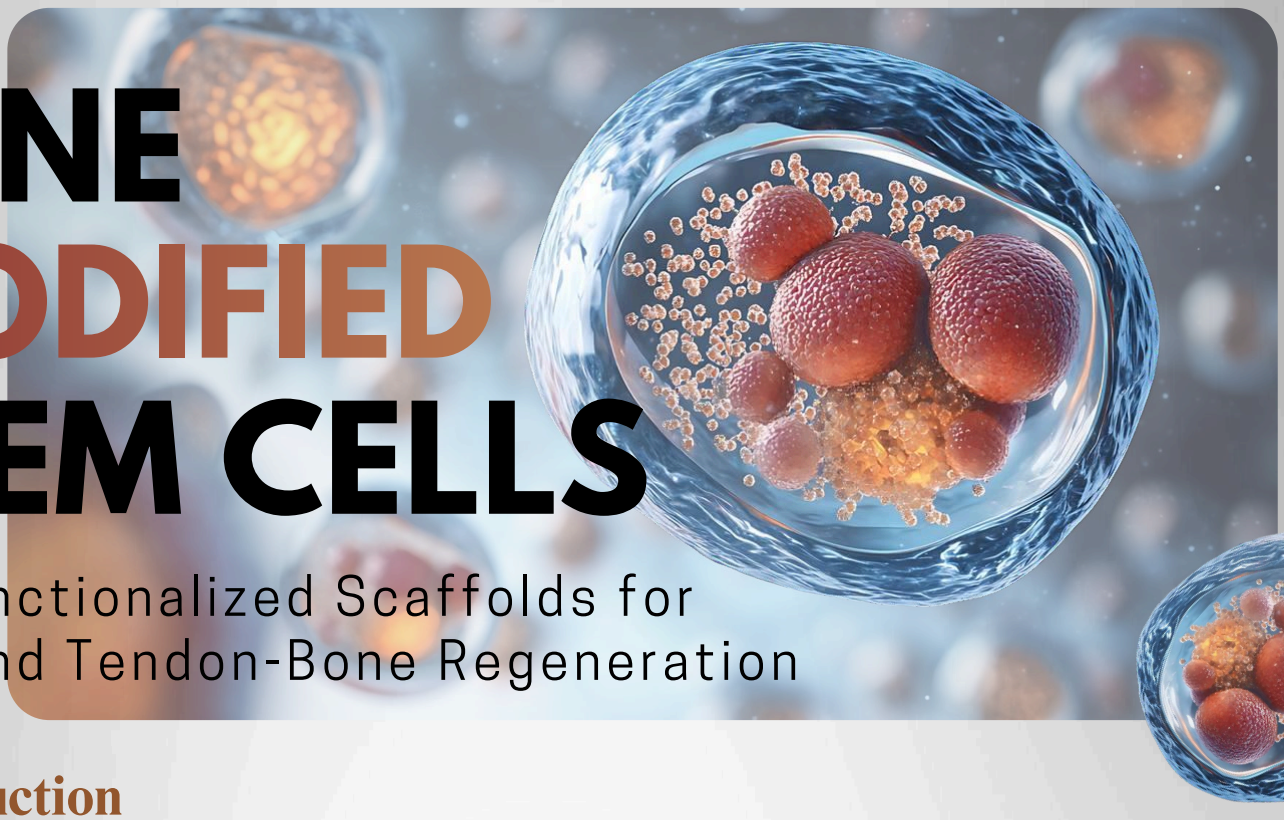


BIOTECHNOLOGY

- Gene-Modified Stem Cells and Functionalized Scaffolds for Bone and Tendon-Bone Regeneration

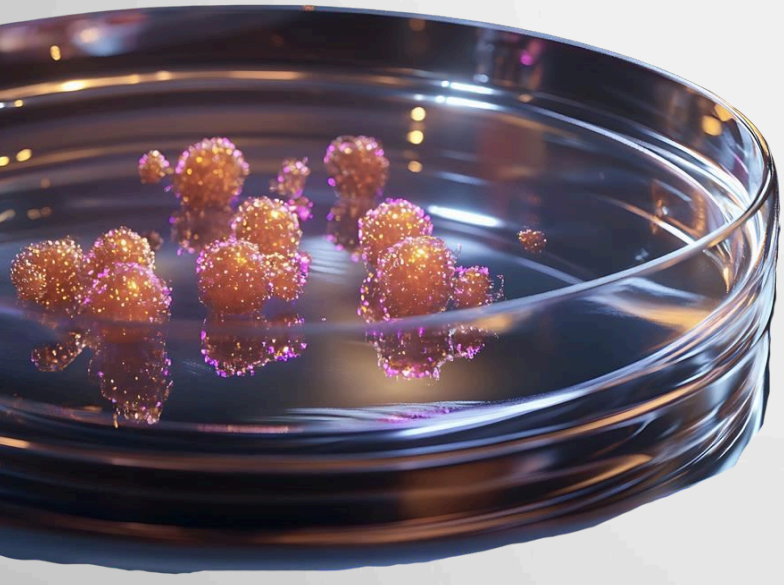
GENE MODIFIED STEM CELLS

And Functionalized Scaffolds for Bone and Tendon-Bone Regeneration



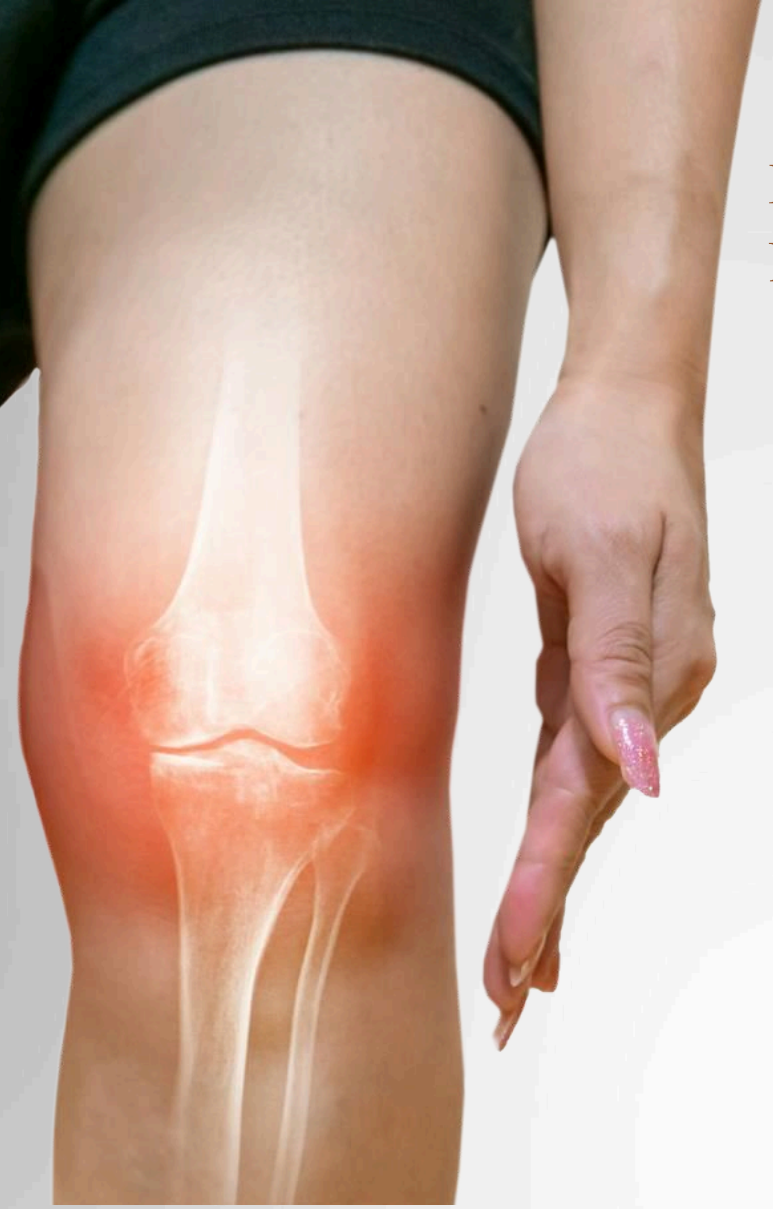
Introduction

Bone injuries and problems at the tendon bone interface like the complications that often appear after ACL reconstruction are still pretty hard to treat. These tissues don't naturally heal well, and the usual clinical approaches rarely deliver fully satisfying results. Because of that, mesenchymal stem cells (MSCs) have become a big focus in regenerative medicine. They can differentiate into bone, calm down inflammation, and move toward injured areas, which makes them promising. Still, once they're actually transplanted, they often struggle: many don't survive long, some fail to integrate into surrounding tissue, and others end up distributed in places where they're not really useful. That's why recent research is trying to "upgrade" MSCs by genetically modifying them, combining them with engineered scaffolds, preconditioning them chemically, or delivering them through more targeted systems.



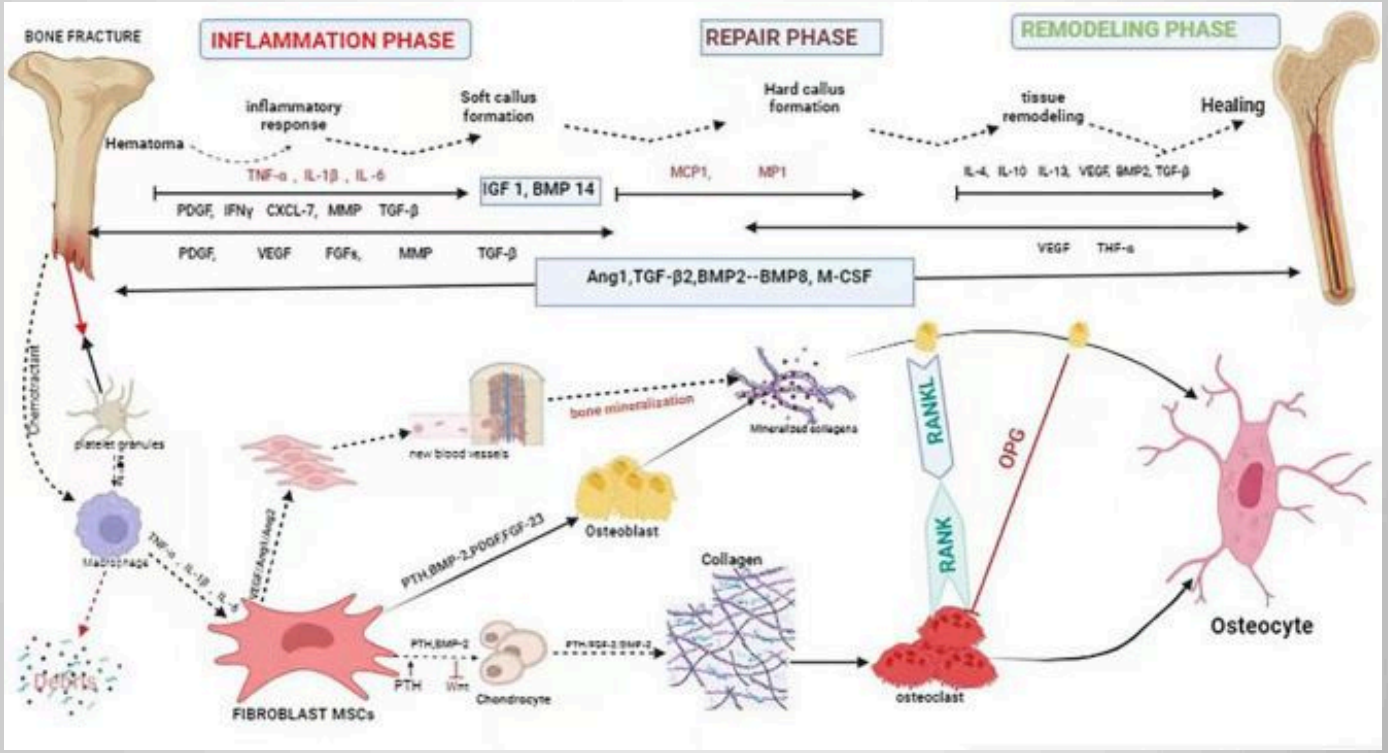
CRISPR-TMSCs for Enhanced Bone Repair

One example is CRISPR/Cas9-engineered tonsil-derived MSCs that were edited to overexpress BMP2 and VEGF. When these cells were combined with a vitamin D–D-loaded PLGA scaffold, they showed stronger osteogenic and angiogenic behavior in vitro, and they also displayed better immunomodulatory effects. Interestingly, this type of multifunctional scaffold helps the cells survive longer in vivo and shifts macrophages from the inflammatory M1 state toward the regenerative M2 state, which is essential for successful bone repair.



BMP-2 MSCs Improve ACL Healing

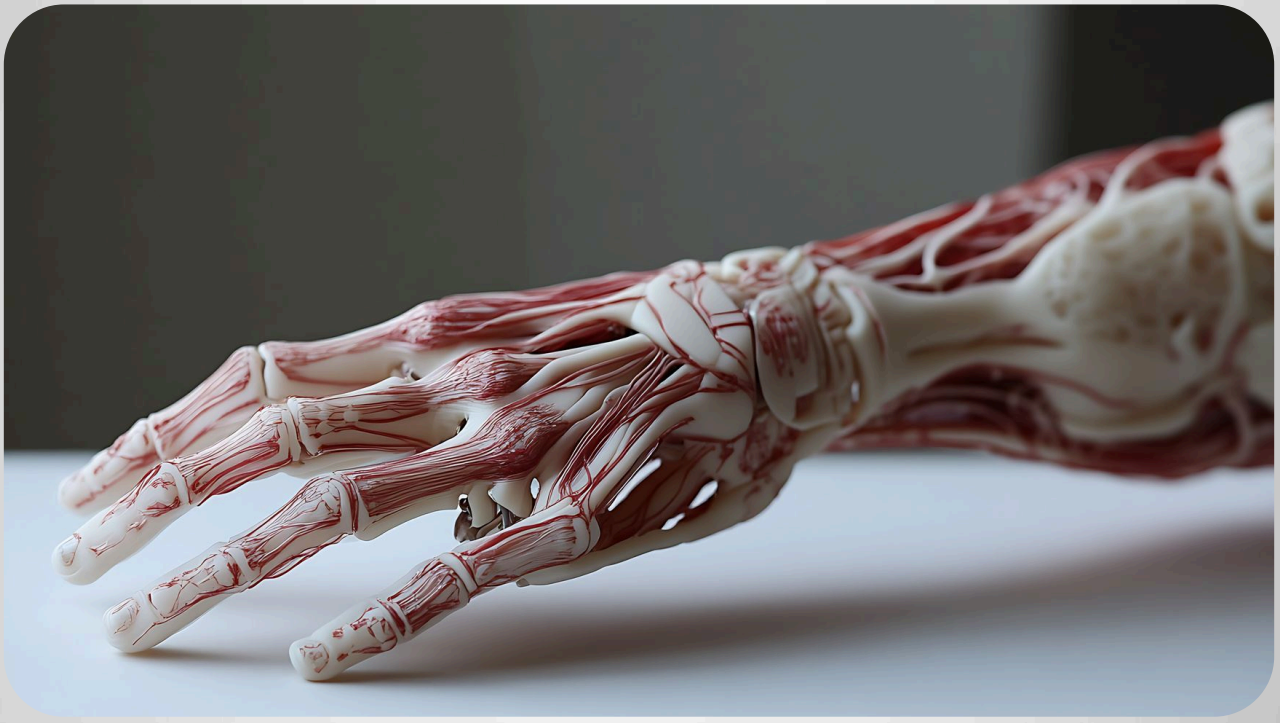
Another study looked at BMP-2-modified bone marrow MSCs in a rabbit ACL reconstruction model. These engineered cells improved tendon bone integration much more effectively than adenovirus-transfected MSCs or standard controls. Micro-CT scans and biomechanical tests showed increases in bone volume, trabecular thickness, trabecular number, and overall mechanical strength. Of course, moving from these promising animal studies to real clinical use comes with its own complications manufacturing consistency, immune compatibility, and large-scale production are still major hurdles. But newer approaches like precisely engineered implants and AI-supported scaffold design are opening the door to more reliable and customizable therapies.



Conclusion

Overall, combining gene-modified MSCs with multifunctional biomaterial scaffolds seems to be one of the most convincing strategies for regenerating bone and repairing the tendon bone interface.

By enhancing osteogenesis, promoting vascularization, and improving local immune regulation, these approaches create a much more supportive environment for healing. Scaffold-based systems—whether injectable hydrogels, 3D-printed structures, or bioactive coatings also help maintain localized and sustained cell activity, addressing some of the biggest weaknesses of traditional cell therapy. Together, these modified cells activate key pathways like BMP and Wnt, boost VEGF-driven angiogenesis, and help control inflammation, ultimately speeding up bone formation and improving tendon–bone integration.



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CASE REPORT

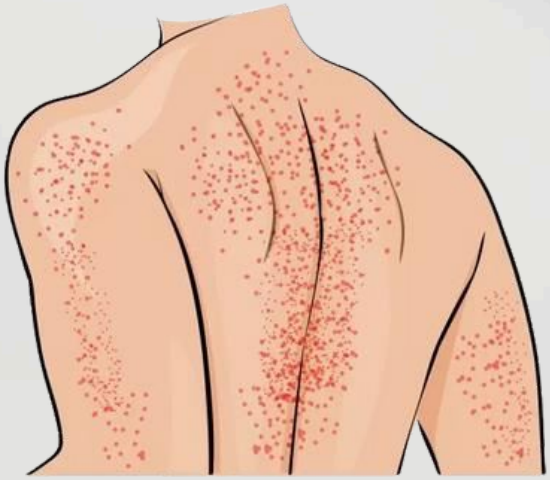
THROMBOTIC PURPURA ⊛ INFECTIVE ENDOCARDITIS

1 Thrombotic thrombocytopenic PURPURA IN PEDIATRICS

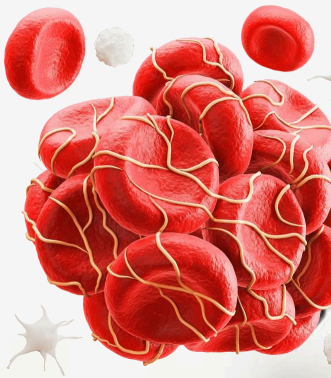
Introduction

Thrombotic thrombocytopenic purpura (TTP) is an uncommon yet fatal obstructive microangiopathy as a consequence of a profound deficiency of ADAMTS13 (<10%).

TTP is characterized by microangiopathic hemolytic anemia, thrombocytopenia, and, depending on the presence or absence of neurological, cardiac, and gastrointestinal symptoms. TTP has a high mortality rate in untreated patients; therefore, TTP must always be considered when TMA is present.



Case Presentation



A healthy 15-year-old girl developed right hemiparesis and paresthesia followed by asthenia, vomiting, and abdominal pain over 5 days. On admission, she was afebrile and well-looking with jaundice, petechiae, and a right ankle hematoma; splenomegaly was palpable. The rest of the systemic examinations were unremarkable.

Laboratory and Diagnostic Findings (on admission)

Parameter	Finding	Comment
Hemoglobin	5.4 g/dL	Severe normocytic, normochromic, regenerative anemia
MCV / MCH	88 fL / 29 pg	Normal indices
Platelet count	12,800/µL	Severe thrombocytopenia
WBC count	Normal	---
Liver function tests	Elevated AST and LDH; unconjugated hyperbilirubinemia	Consistent with hemolysis
Kidney function	Normal	Excludes hemolytic uremic syndrome pattern
Coombs test	Negative	Non-immune hemolysis
Peripheral smear	Schistocytes, microangiopathy	Suggestive of TMA
Immunologic tests	Normal	Autoimmune disorders ruled out
Infectious screening	HIV (-), HCV (-), HBV (-), CMV IgG (+), HSV-1/2 IgG (+), VDRL (-), Chagas (-)	No active infection
Imaging (US, CT, MRI, CNS angio MRI)	Normal	No organ-specific lesions
ADAMTS13 activity	<10%, inhibitor positive	Confirms autoimmune acquired TTP

Management and Outcome

The findings suggested TTP, so plasma exchange with high-dose methylprednisolone was started immediately. An ADAMTS13 activity level of less than 10% confirmed the diagnosis of autoimmune acquired TTP. After achieving remission, the patient was treated with rituximab for a relapse and continues to be monitored regularly.

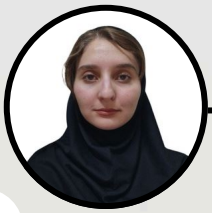
Discussion

Thrombotic thrombocytopenic purpura (TTP) is an uncommon and life-threatening microangiopathy due to severe deficiency of von Willebrand factor-cleaving protease (ADAMTS13). In the absence of this enzyme, platelets clump together to form microthrombi, which are followed by intravascular hemolysis and tissue ischemia. Approximately 90% of TTP patients have an ADAMTS13 antibody-mediated acquired form. This form is characterized by fluctuating neurological and cardiac manifestations, but mild renal involvement. It is diagnosed with the presence of microangiopathic hemolytic anemia, thrombocytopenia, and an ADAMTS13 level less than 10%.

Differential diagnoses consist of hemolytic uremic syndrome, drug induced anemia , and complement-mediated microangiopathy. The main therapies are plasma exchange and glucocorticoids, which have now reduced the TTP mortality to less than 20%, from about 90%. For relapse and refractory cases, rituximab is very effective. In our case, the patient responded with total remission to this therapy.

Conclusion

Acquired TTP is an uncommon but life-threatening disorder. Recognition at an early stage and immediate treatment are important for a higher survival rate and prevention of recurrence.



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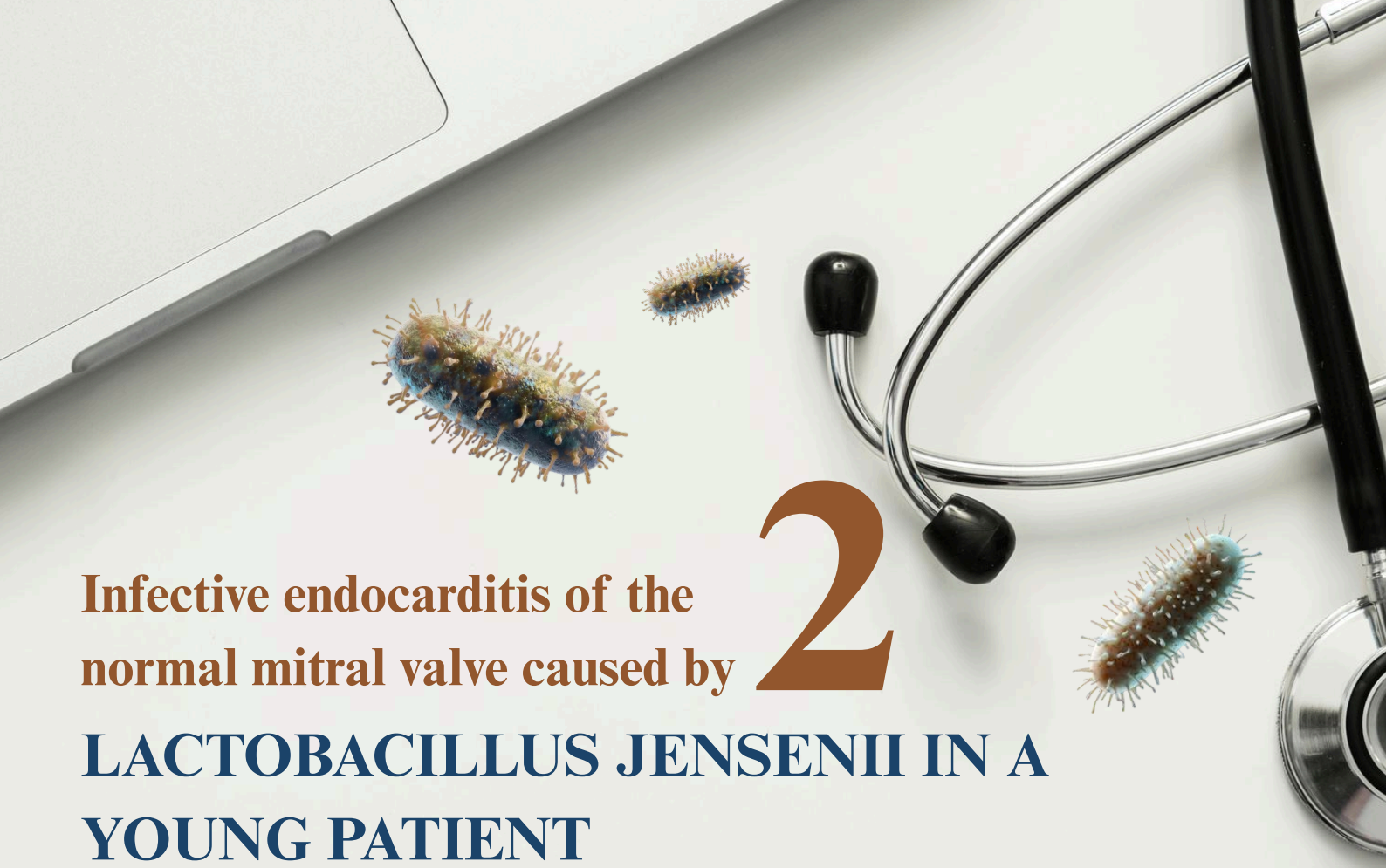


2 Infective endocarditis of the normal mitral valve caused by LACTOBACILLUS JENSENII IN A YOUNG PATIENT

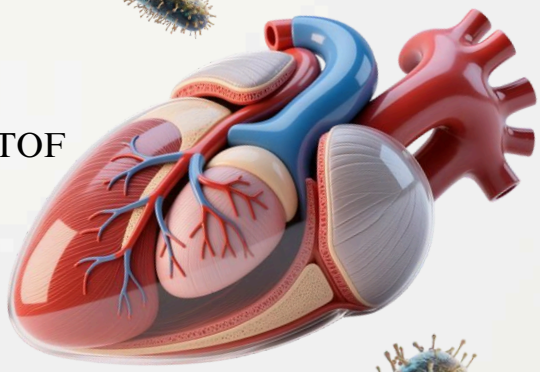
Introduction

Lactobacillus jensenii is a gram-positive, rod-shaped, facultative anaerobic bacterium that is part of the normal female genital tract flora and is found in fermented foods and probiotics. Infective endocarditis (IE) caused by L. jensenii is extremely rare and usually occurs in patients with specific underlying risk factors. This report presents a rare case of IE involving a normal mitral valve in a previously healthy adolescent girl.

Clinical Case

- A 15-year-old Thai girl presented with progressive exertional dyspnea and intermittent low-grade fever for two months. She had no prior medical history, menstrual abnormalities, sexual activity, tobacco or alcohol use, or genitourinary complaints.
- Vital signs were notable for a temperature of 38.5°C, blood pressure 114/59 mmHg, heart rate 61 beats/min, and respiratory rate 22 breaths/min. Skin, hair, and dentition were normal. Cardiac auscultation revealed a pansystolic murmur at the apex and bilateral pitting edema. No skin lesions or neurological deficits were observed.

- Laboratory findings showed leukocytosis (WBC 14,870/µL, 81% neutrophils), elevated ESR (59 mm/hr), and mildly increased troponin T (31 pg/mL). Transthoracic echocardiography revealed severe mitral regurgitation due to ruptured chordae tendineae of the anterior mitral leaflet and a 0.7 × 0.9 cm oscillating mass. Other cardiac valves and ventricular function were normal.



- After four days, L. jensenii was detected by MALDI TOF mass spectrometry with a confidence score of 2.30 in three consecutive blood cultures. Gentamicin (120 mg daily), cloxacillin (3 g every 6 hours), and intravenous ampicillin (3 g every 6 hours) were the first treatments administered in accordance with ESC 2023 criteria. A 28-mm Cosgrove-Edwards annuloplasty ring was used to reconstruct the mitral valve 11 days after the procedure. If you misspell something, make a grammatical error, or misuse a punctuation mark, Grammarly will suggest ways to fix it.

- Histopathology showed torn chordae tendineae at A2–A3 and perforation at P2, with mild chronic inflammation, fibrosis, and calcium deposition. No microorganisms were identified, likely due to prior antibiotics.

- Postoperatively, ceftriaxone (2 g daily) and vancomycin (1,500 mg daily, titrated to pre-dose levels 10–15 mg/L) were administered alongside warfarin. She completed a six-week antibiotic course at a rehabilitation center. At the three-month follow-up, she was asymptomatic with normal cardiac function. Gynecologic evaluation, including pelvic ultrasound, was unremarkable.

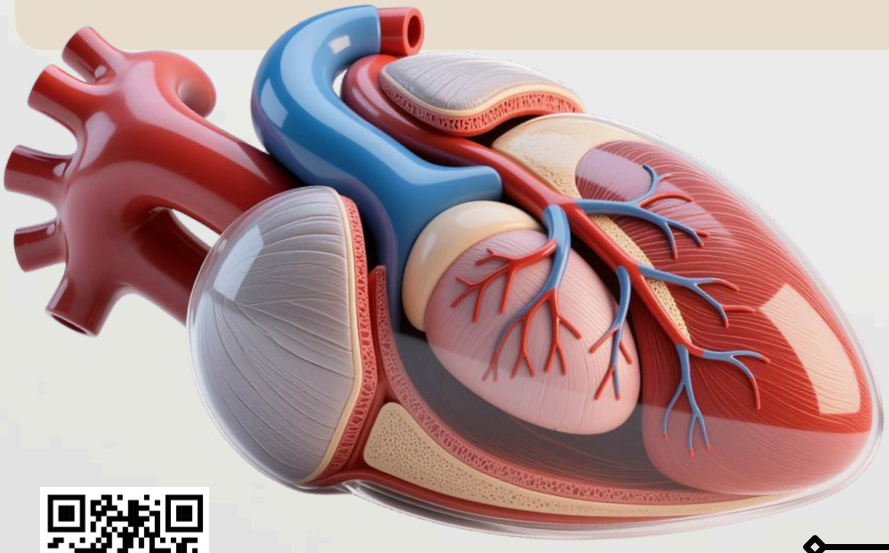
Discussion

- L. jensenii is a dominant vaginal commensal that protects against infections. IE, due to this species being rare, it occurs mostly in patients with dental disease, immune compromise, or structural heart abnormalities. Symptoms include fever, malaise, and heart failure, typically with a subacute onset. Unlike other Lactobacillus species, L. jensenii IE is more frequently reported in women.

- The bacterium can infect both native and prosthetic valves, with a predilection for native valves. Entry pathways are often unclear, though poor dentition and invasive procedures may contribute. Lactobacillus species are generally susceptible to penicillin, ampicillin, erythromycin, and clindamycin, but resistant to gentamicin, vancomycin, and most aminoglycosides; L. jensenii is an exception and remains vancomycin-sensitive. Combination therapy with ampicillin and gentamicin for 4–6 weeks is effective, with favorable outcomes and low recurrence.

Conclusion

L. jensenii IE is extremely rare and may present diagnostic and therapeutic challenges. Despite being part of normal vaginal flora and present in probiotics, it can infect native or prosthetic valves. Ampicillin plus gentamicin for 4–6 weeks is effective, though further studies are needed to optimize management strategies due to the scarcity of cases.



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