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# Gratitude to Our Guiding Lights

## Dr G MATHAN

Coordinator-BioMed Futura

*His unwavering faith in student potential, his encouragement to question boldly, and his wisdom to guide without restricting are the threads that have stitched this vibrant scientific community together.*

*Dr. Mathan's leadership is not measured in milestones alone, but in the minds he's inspired — to think deeply, act ethically, and dream audaciously.*

*We thank him not only for his direction, but for the belief he instills in us every day.*

Dr K PREMKUMAR
Professor, BMS

Dr S D SARASWATHY
Associate Professor, BMS

Dr P SHANMUGAPRIYA
Associate Professor, BMS

*You are the voice that challenges our assumptions, the guide that nurtures resilience, and the spark that turns curiosity into inquiry. Whether in lecture halls, labs, or brief moments between practical, your presence has shaped how we think, how we learn, and how we dream. We honor your unwavering support from behind us.*

# About Biomed Futura

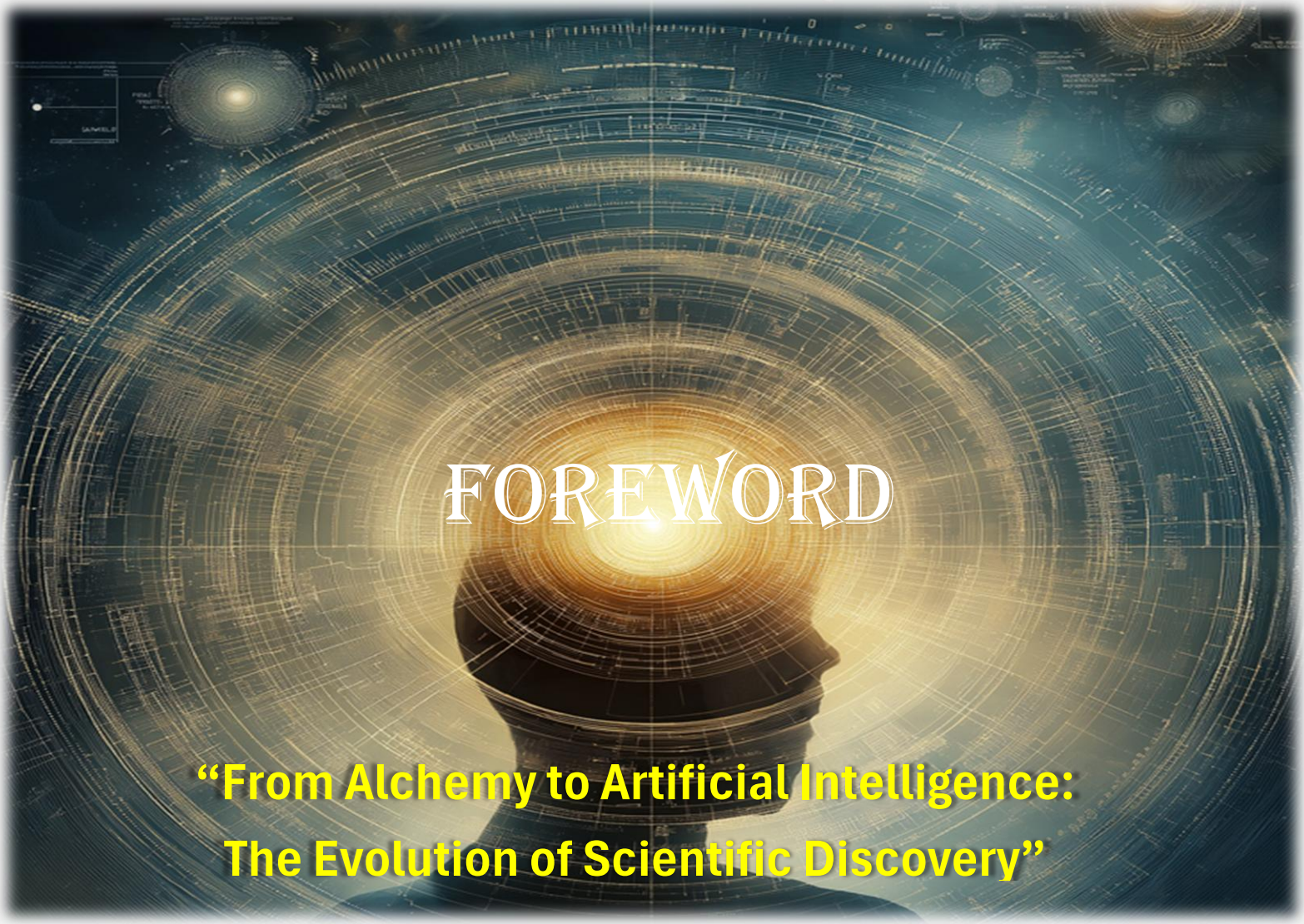
*Biomed Futura isn't just a club — it's a movement, where science meets spirit*

**Established in 2017, Biomed Futura is the in-house academic club of the Department of Biomedical Science. It serves as a dynamic platform where students transform curiosity into contribution, and learning into leadership. Rooted in the vision of inspiring bold thinking beyond classroom boundaries, the club cultivates globally competent individuals who combine scientific excellence with social responsibility.**

**From its inception, Biomed Futura has pulsed with creative energy — organizing thought-provoking events, weekly meetings, and hands-on activities that encourage students to think beyond convention. Members of the club, elected by peers, drive initiatives with passion and purpose, fostering a strong sense of ownership and teamwork.**

**Every year, the club invites distinguished speakers from diverse scientific domains to spark dialogue, nurture intellectual growth, and promote interdisciplinary collaboration — empowering students to tackle complex challenges with holistic insight.**

**Through debates, quizzes, speeches, and student-led research magazines, Biomed Futura validates the voices and visions of emerging scientists. It stands as a vital force within the department, championing liberal education, ethical inquiry, and a culture of innovation.**




Welcome to the inaugural edition of *Biomed Futura Digest*. As we embark on our editorial journey, we begin with a theme close to every curious mind — the evolution of scientific exploration.

From mystical cures to molecular interventions, the story of biomedical science is a tale of transformation. Ancient healers spoke through herbs and intuition, while modern researchers decode pathogens in nanoscale chambers. What unites them? Curiosity. Courage. A relentless pursuit of understanding.

This issue maps the metamorphosis, tracing milestones from medieval medicine to microfluidic marvels. At Biomed Futura, we embrace both retrospective wonder and futuristic ambition, believing that innovation blooms when we honor the legacy of questions asked long ago.

Let us journey together — across centuries, across cells, and into the heart of discovery.

Warmly,
**Meiyamai S P & Janani R**
**Editorial chiefs, BioMed Futura.**

## Contents

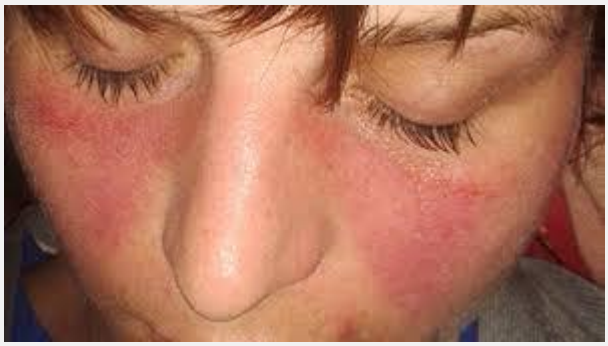
# HEALING FROM WITHIN:
## ROLE OF STEM CELLS IN FIGHTING LUPUS"

Kodiarasy P - 3rd year BMS

-

**Despite advances in immunosuppressive drugs and biologics, a significant number of patients continue to suffer from relapses and organ damage. This has led to the exploration of stem cell therapy as a novel treatment avenue. Two main types of stem cells are being investigated:**

- **Hematopoietic Stem Cells (HSCs) –** Responsible for generating new blood and immune cells.
- **Mesenchymal Stem Cells (MSCs) –** Known for their immune-modulating and tissue-repair properties.



SOURCE: https://www.healthline.com/health/systemic-lupus-erythematosus

However, many patients still experience persistent disease activity, relapses, or adverse effects from long-term drug use. **HSCT** aims to "reset" the immune system by first wiping out the patient's faulty immune cells and replacing them with new, healthy hematopoietic stem cells. Some studies report long-term remission in severe cases of SLE. However, HSCT is associated with high risks, including infections and treatment-related complications. It is also expensive and not widely accessible.

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**MSCs** can be derived from bone marrow, umbilical cord blood, or adipose (fat) tissue. They modulate immune responses, reduce inflammation, and help regenerate damaged tissue. Clinical trials in China and elsewhere have shown promising results: up to 60% of patients experienced improvement in symptoms, reduced proteinuria (a sign of kidney damage), and better kidney function. Importantly, MSC therapy is relatively safe with fewer side effects and no significant risk of graft-versus-host disease (GVHD).

Stem cell therapies offer a promising new approach for treating refractory or severe SLE. While hematopoietic stem cell transplantation can be effective, MSC therapy is gaining attention due to its lower risks, regenerative properties, and immunomodulatory effects. Ongoing research is crucial to establishing its place in standard clinical practices.

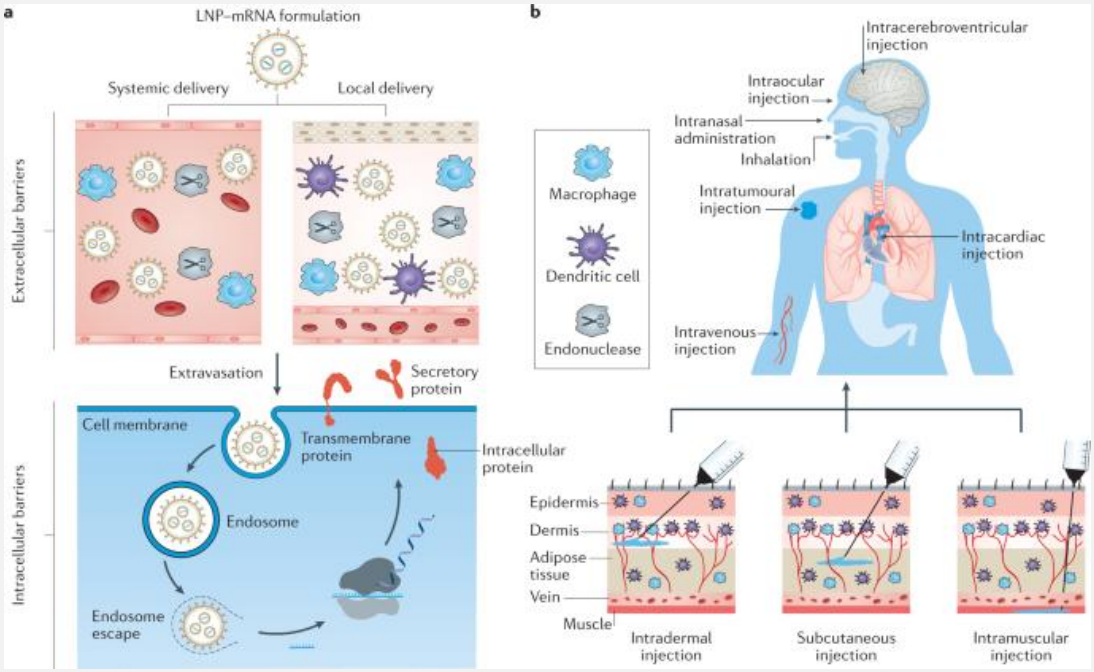
# MODULAR TARGETING:

## Bispecific-Antibody-Guided mRNA-LNPs

Shafiq Ahamed Mohamed Riaz- 4th Year BMS

## Introduction:

Messenger RNA (mRNA) therapeutics have transformed biomedicine by enabling cells to produce therapeutic proteins directly from genetic instructions. However, conventional lipid nanoparticles (LNPs) predominantly accumulate in the liver, limiting their utility for other tissues and cell types. Overcoming this delivery bottleneck is essential to fully realize mRNA's potential beyond vaccines.




SOURCE: Cheetham S.J., et al. (2024) *Targeted mRNA delivery with bispecific antibodies that tether LNPs to cell-surface markers*. **BioRxiv.**

*This schematic highlights why off-target uptake and endosomal escape remain key hurdles for non-hepatic delivery.*

## The Challenge:

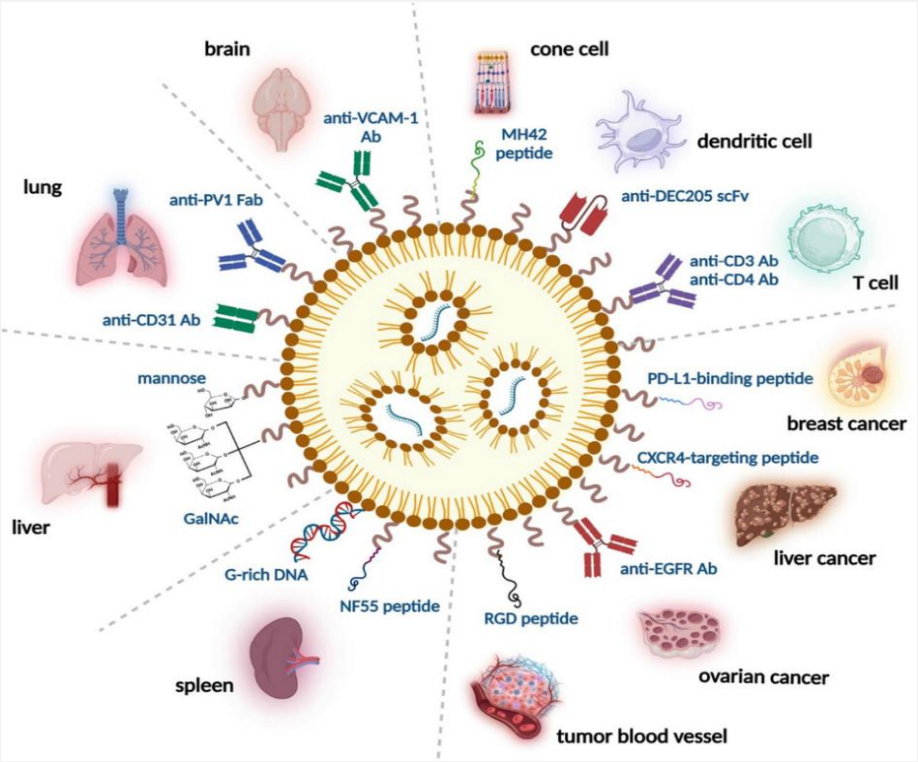
Despite the remarkable success of mRNA vaccines, broad adoption of mRNA drugs has been constrained by off-target delivery. Traditional LNP formulations lack intrinsic selectivity, causing most of the injected dose to localise in hepatocytes. This not only reduces efficacy against non-hepatic targets but also raises safety concerns due to unintended protein expression. Moreover, tailoring LNP composition for each new target cell type adds complexity and cost, slowing the path from bench to bedside.

## The Breakthrough:

Researchers led by Seth Cheetham's team at the Australian Institute for Bioengineering and Nanotechnology pioneered a **bispecific-antibody (Bs Ab) guided LNP** platform that modularly directs

mRNA payloads to chosen cell-surface markers. In this approach, a Bs Ab is engineered with one arm binding polyethylene glycol (PEG) on the LNP surface and the other arm recognising a cell-specific antigen (e.g., EGFR on tumor cells or PSMA on prostate tissue).

Preclinical in vitro assays demonstrated that Bs Ab-decorated LNPs preferentially bind and enter antigen-positive cells, dramatically increasing mRNA uptake compared to untargeted controls. In mouse models, systemic administration of EGFR-targeted Bs Ab-LNPs resulted in robust protein expression in EGFR-expressing tumors, while sparing non-target tissues (Cheetham et al., 2024). This "plug-and-play" design requires no reformulation of the core LNP; simply swapping the BsAb redirects the same mRNA cargo to any antigen of interest.

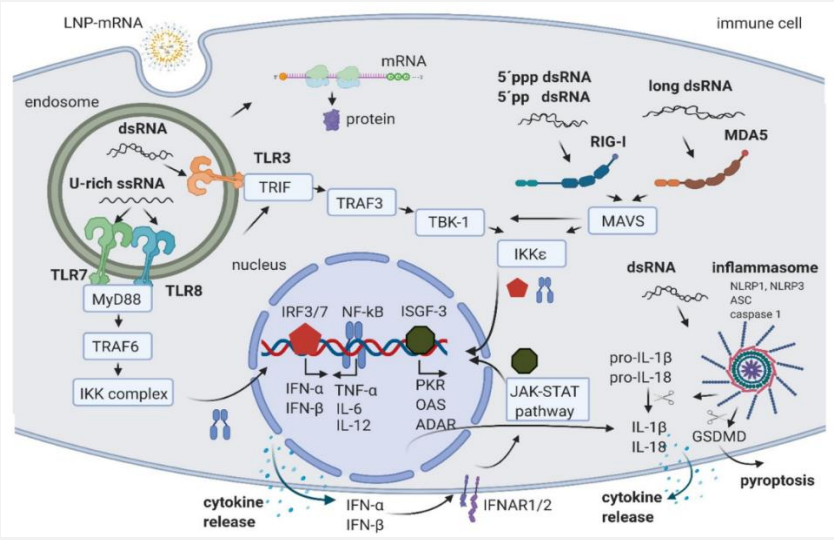



SOURCE: Cheetham S.J., et al. (2025) *Bispecific-antibody guided mRNA-LNPs for targeted protein expression*. **Molecular Therapy – Nucleic Acids**.

*In vitro and in vivo studies showed marked increases in target-cell protein expression with negligible off-target delivery.*

## Why It Matters:

This modular targeting strategy addresses two major obstacles in mRNA therapeutics. First, it decouples delivery specificity from nanoparticle chemistry, allowing researchers to use well- characterized, clinically validated LNP backbones while achieving cell-type precision. Second, by attaching Bs Abs ex vivo, manufacturing remains streamlined: a single GMP-grade LNP stock can serve multiple indications. These advantages promise to accelerate development timelines and reduce production costs, crucial factors for scalable personalized medicine.




SOURCE**:** Kulkarni, J.A., et al. (2020). *The current landscape of lipid nanoparticles for nucleic acid delivery*. **Frontiers in Chemistry**, 8, 589959.

*This general schematic emphasises the modularity: just as antibodies or peptides can be grafted onto LNPs, BsAbs serve as a universal "plug" for any antigen target.*

## Broader Implications:

Beyond cancer, BsAb-guided LNPs could revolutionise treatments for a spectrum of diseases. In rare genetic disorders, targeting patient-specific cell types (e.g., neurons or muscle fibres) becomes feasible without bespoke nanoparticle development. Autoimmune conditions might benefit from directing tolerogenic proteins to dendritic cells, retraining the immune system. Furthermore, the platform's modularity aligns perfectly with next-generation cancer vaccines: mRNA sequences encoding tumour neoantigens can be delivered selectively to antigen-presenting cells, enhancing immune priming while minimising systemic inflammation.

## Conclusion:

Bispecific-antibody-guided mRNA-LNPs represent a pivotal advance in biomedicine's toolkit. By uniting the versatility of mRNA with precise, antibody-mediated targeting, this approach transforms generic delivery vehicles into bespoke therapeutics. As this technology progresses toward clinical evaluation, it holds the promise of truly personalised, tissue-specific mRNA treatments and ushering in a new era of precision medicine.

### References:


1. Cheetham S.J., et al. (2024) *Targeted mRNA delivery with bispecific antibodies that tether LNPs to cell-surface markers*. **bioRxiv**.
2. Cheetham S.J., et al. (2025) *Bispecific-antibody guided mRNA-LNPs for targeted protein expression*. **Molecular Therapy – Nucleic Acids**.
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4. Cheng, Q., et al. (2021) *Selective organ targeting (SORT) nanoparticles for mRNA delivery and CRISPR/Cas gene editing*. **Nature Nanotechnology**, 16, 214–224.
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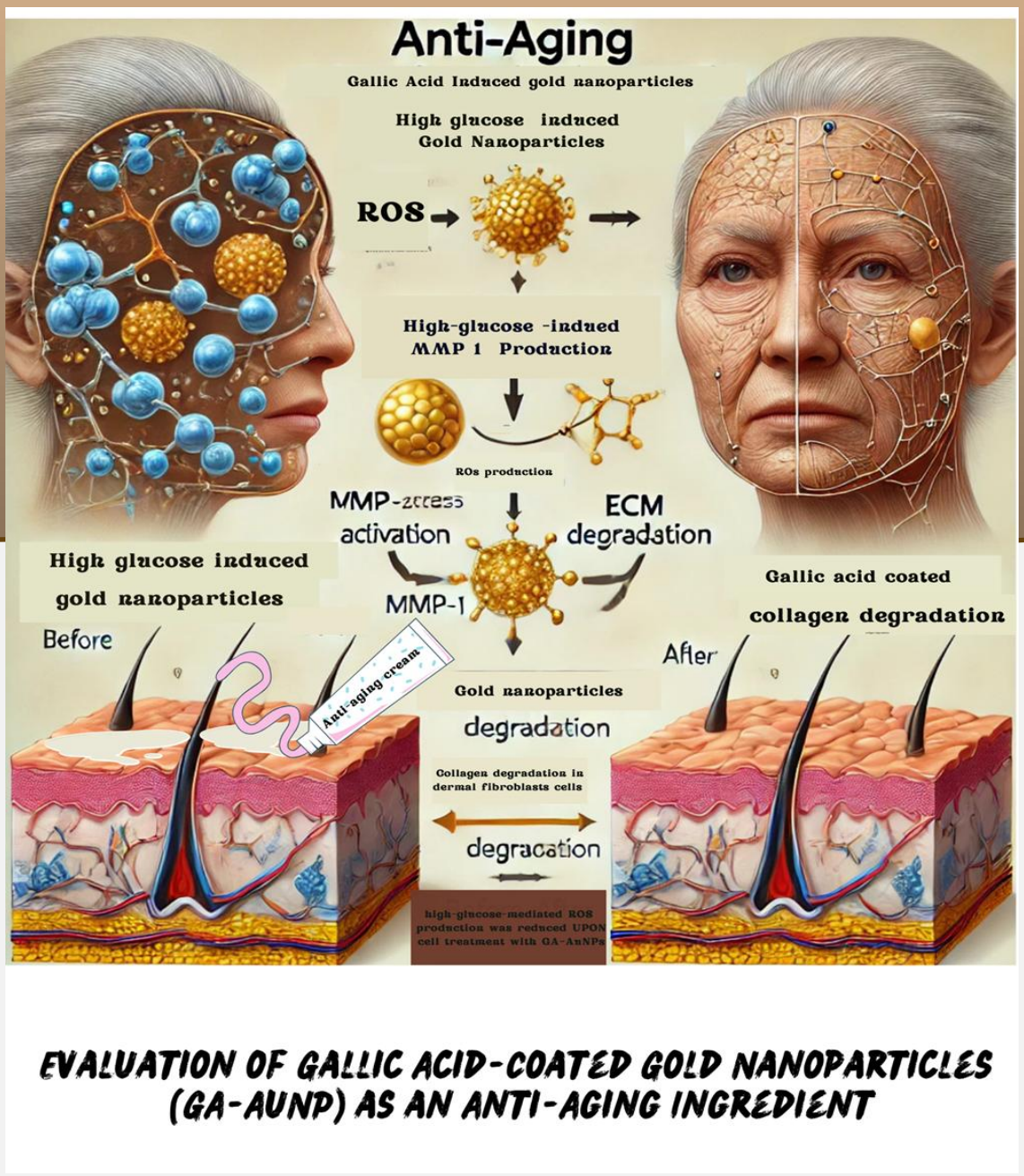
# High glucose induced Gold Nanoparticles
# A Illustrate

**Megavarshini- 4th year BMS**



# "THE URGENCY - MARBURG THERAPEUTICS"

Sathya N - 3rd year BMS

**A virus is an extremely small infectious agent (much smaller than a bacterium) that can only replicate within the living cells of a host organism. Marburg virus disease (MVD) is a rare but deadly hemorrhagic fever that is caused by the Marburg virus, a filovirus of the family Filoviridae and a close relative of the Ebola virus. The illness has a case fatality ratio of 24–88%, varying with outbreak and access to treatment.**

## Marburg Virus:

Marburg Virus Disease (MVD), a most lethal viral fever, was first detected in 1967 in Germany and Yugoslavia not Africa. The incident started in laboratory, the work done with African green monkeys imported from Uganda for the production of polio vaccines. Laboratory workers contracted fever, vomiting, hemorrhaging, and organ malfunction, developing 31 cases and 7 fatalities. The virus was named so because it first caused fatal cases in the town of Marburg.

## Case Description:

A 38 years old male farmer from Guinea, had the symptoms of High fever, Severe headache, Diarrhea and vomiting, Bleeding, internal and external.

Patient had recent contact with a bat-infested cave—the main natural reservoir Egyptian fruit bat (Rousettus aegyptiacus). The patient died within 10 days despite supportive treatment in an isolation unit, showing rapid course and virulence of the disease. Contact tracing of more than 90 people were affected. WHO has sent Rapid Response teams, and supportive therapy (IV fluids, oxygen, analgesics) is still the cornerstone, as there is no licensed vaccine or antiviral therapy. Experimental vaccines are in Phase II trials.

Being oral, it's simpler to distribute and give in resource-scarce environments, facilitating outbreak control particularly in far-flung areas like rural Africa. In January 2025, WHO initiated a Collaborative Open Research Consortium (CORC) on filoviruses, including Marburg World Health Organization. The initiative supports fair global research, accelerating rapid sharing of data, therapeutics, and vaccine trials between nations. In March 2025, a genomic survey found 70+ full Marburg genomes and highlighted hotspots, bat reservoir dynamics, and cross-border spread patterns. This enhances global surveillance, supporting targeted control zone delineation and prevention of future zoonotic spillovers.

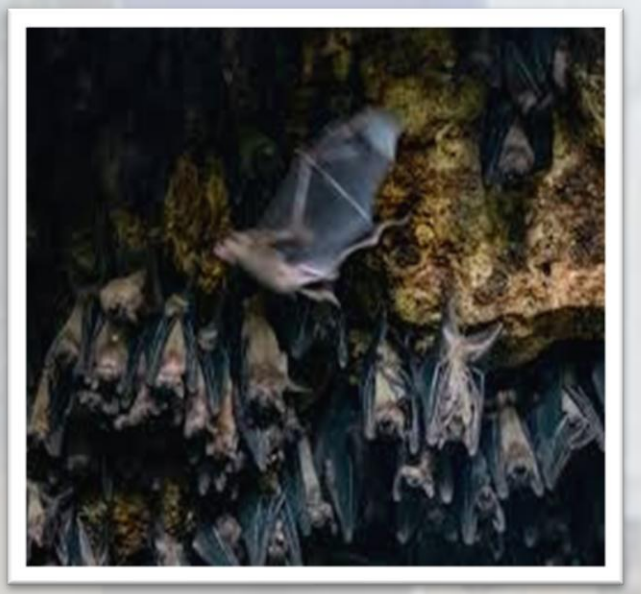
BMC Medicine (May 20, 2025) highlighted that Rwanda's initial 10-day response was characterized by speedy testing, health worker protection, and coordination with global partners. The 15th World Health Summit & Global Preparedness Monitoring Board prioritized Marburg, Mpox, and H5N1 as the top threats for 2024 and encouraged investment in healthcare resilience, digital data systems, and One-Health Cooperation. The ongoing research on Marburg therapeutics: Vaccine development, Monoclonal Antibodies, Antiviral Drugs – Remdesivir, Galidesivir, Favipiravir, Combination Therapies, and Novel therapies.

## Challenges And Future Directions:

Despite promising research, several challenges remain in developing and distributing Marburg therapeutics, including:

Lack of clinically approved treatments, pathogenicity, Geographic dispersion of outbreaks, and Equitable access

The development and deployment of safe and effective Marburg therapeutics require a concerted global effort, including increased funding, collaborative research, and robust public health preparedness strategies. This will pave the way for a more effective response to future Marburg outbreaks and strengthen global health security.



SOURCE: https://theconversation.com/the-first-human-case-of-marburg-virus-in-west-africa-is-no-surprise-heres-why-166694

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# CASE STUDY REPORT: HUTCHINSON-GILFORD PROGERIA SYNDROME (HGPS)

Pradeep P - 4th year BMS

## Background:

Hutchinson-Gilford Progeria Syndrome (HGPS) is an extremely rare genetic disorder characterized by dramatic, rapid ageing beginning in childhood. It affects approximately 1 in 20 million people worldwide.The disorder is caused by a de novo point mutation in the **LMNA gene**, leading to the production of an abnormal protein called **progerin.**

## Patient Profile:

• Name: Aryan (Pseudonym) • Age: 7 years • Gender: Male • Location: Mumbai, India
• Diagnosis Age: 2 years

## Clinical Presentation:

Aryan was born full-term with normal birth weight and no apparent abnormalities. Around 18 months of age, he exhibited growth delays, hair thinning, prominent scalp veins, and scleroderma-like skin changes. By age 3, signs of aged appearance, joint stiffness, and loss of subcutaneous fat were noted.

## Symptoms Observed:

• Failure to thrive and growth retardation
• Alopecia and prominent eyes
• Tight, shiny skin and joint contractures
• Visible scalp veins and aged facial features
• High-pitched voice and hip dislocations

SOURCE: https://healthwire.pk/healthcare/rare-diseases-in-the-world/

## Diagnosis:

Molecular genetic testing confirmed a heterozygous mutation (c.1824C>T) in the LMNA gene, validating the diagnosis of HGPS. Aryan's karyotype was normal.

## Management:

Currently, there is no cure for HGPS. Aryan is under a multidisciplinary care plan involving pediatric cardiologists, dermatologists, orthopaedic specialists, and nutritionists. He is enrolled in a clinical trial for lonafarnib, a farnesyltransferase inhibitor, which has shown promise in improving cardiovascular outcomes.

## Conclusion:

This case illustrates the classical clinical and molecular presentation of HGPS. Early diagnosis and symptomatic management, including access to emerging therapies, are essential to improve the quality of life and potentially extend survival in children.

# PUZZLE- CANCER BIOLOGY

**Abarna B - 2nd Year BMS**

1. **Step in the cell cycle where replicated chromosomes are separated to opposite Poles of the cell**

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2. **Programmed cell death.**

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3. **Gene located on chromosome 9 that codes for a proto-oncogene**

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4. **A germ cell tumor containing multiple tissue types, not usually associated with the site of tumor formation**

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5. **Process in which glucose is converted into lactate in the presence of oxygen, also known as the Warburg effect**

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6. **Transmembrane protein that is a receptor for members of the epidermal growth factor family**

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7. **Gene that codes for tumor suppressor protein**

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8. **Series of events leading to duplication of cell (DNA, organelles, cytoplasm, etc)**

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9. **Cells that produce the major components of the Extra cellular matrix**

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10. **Control mechanism of cell cycle**

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**ANSWERS:**

1. Anaphase
2. Apoptosis
3. ABL Gene.
4. Teratoma
5. Aerobic glycolysis
6. EGFR
7. P53
8. Cell cycle
9. Fibroblasts
10. Check points

# WHEN THE WORLD STOOD STILL:

## A Reckoning with Infectious Diseases

Meiyamai S P- 4th year BMS

**For centuries, pathogens have shaped human evolution and history—from the Plague that reshaped medieval Europe to influenza pandemics that rewrote global geopolitics. Yet despite our scientific progress, antimicrobial resistance rises, zoonotic spillovers increase, and the social systems meant to protect us remain fragmented. Diseases like Chikungunya, Dengue, and Nipah simmer in regional pockets, each one a potential spark for the next global crisis.**

The pathogens are coming, whether we're ready or not.
Over the past decade, the biomedical research landscape has tilted heavily toward **oncology, metabolic diseases**, and other non-communicable conditions. While these areas deserve continued attention, the relative decline in **infectious disease immunology publications**, funding, and academic investment creates a knowledge gap that is increasingly dangerous in the face of **global outbreaks, mutation-driven immune escape, and neglected tropical diseases**. though there exists global contribution by various organizations such as WHO in ensuring preparedness for various influenza-like threats, this field requires more eye to protect the human community from suffering.

## Expanding this research domain is vital because:

- **Immunological responses shape everything from disease progression to vaccine efficacy**. Without continuous study, especially in varied populations, we risk designing immunotherapies that falter when new variants arise or in regions with distinct genetic and environmental immune profiles.
- **Geographic inequality in research output** means that areas most affected by infectious diseases—like sub-Saharan Africa, Southeast Asia, and Latin America—often lack tailored insights. By increasing global investment, we empower **locally relevant immune studies** that foster sustainable interventions.
- **Emerging pathogens such as Nipah, Ebola, and drug-resistant tuberculosis** require immunological expertise to develop **host-directed therapies** that minimize tissue damage, control inflammation, and improve survival rates—especially when vaccines or antivirals aren't yet available.
- **Cross-cutting technologies**, like **droplet microfluidics**, **single-cell immunogenomics**, and **AI-guided antigen discovery**, are redefining what's possible—but they remain underutilized in infectious disease contexts. Promoting this field invites innovation in **real-time immunological surveillance**, **rapid response platforms**, and **universal vaccine development**.
- **Ethical imperatives** demand scientific equity. We must correct the imbalance wherein cancer research receives disproportionately more resources than diseases that cause preventable deaths in under-resourced settings. Funding infectious disease immunology isn't just science—it's justice.

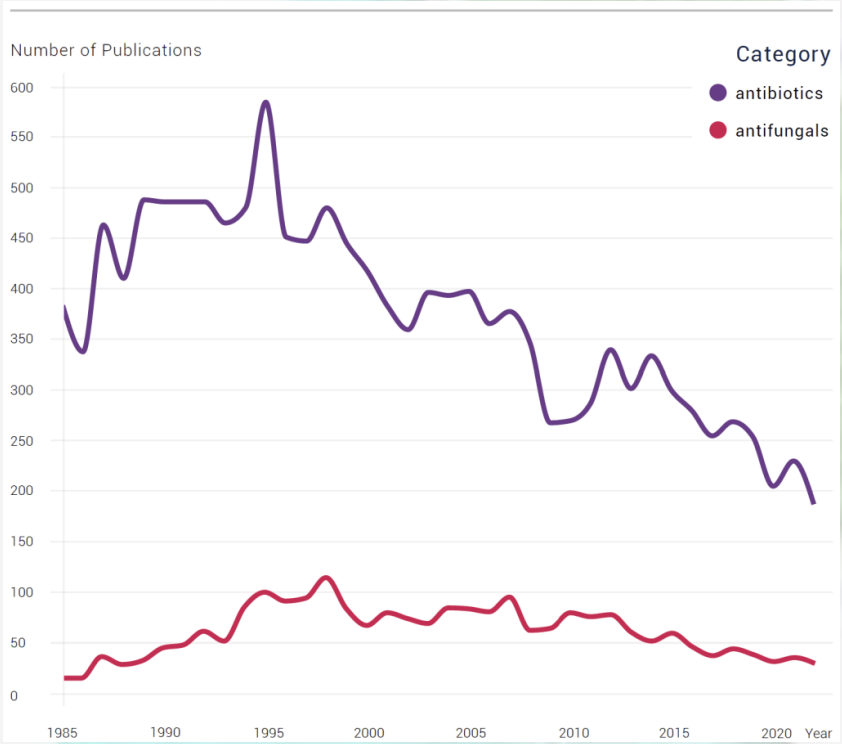
# Global Contributions in Readiness and Resilience:

The **World Health Organization (WHO)** leads the charge, unifying nations under the **International Health Regulations (IHR)** and launching the **One Health Framework**, which surveils human, animal, and environmental health as an integrated system. Its **Pandemic Influenza Preparedness (PIP)** Framework ensures equitable sharing of virus samples and vaccines, while its **Collaborative Open Research Consortiums (CORCs)** accelerate research on emerging pathogens.

The **United Nations** established the **Pandemic Fund**, jointly administered with the World Bank, pledging over **$10 billion** to fortify health systems and workforce capabilities. Their 2023 **Political Declaration on Pandemic Preparedness** commits to transparency, trust, and data equity, essential for countering misinformation and fostering global solidarity.

The pandemic redefined the pace of research—and expanded its horizons.

**As researchers, we hold more than data; we hold responsibility—to pursue knowledge that doesn't just advance careers but safeguards communities. If we do not recalibrate our priorities now, we risk entering the next outbreak with sharper tools but dulled vision. Let this review stand as a voice to make the language of immunity be spoken fluently and fearlessly across every lab, every border, and every discipline."**




SOURCE:
https://amr.solutions/2024/02/08/leaving-the-lab-the-decline-in-amr-rd-professionals/

# ENVIRONMENTAL FORENSICS-

## ADVANCEMENTS & APPLICATIONS

Swetha M P- 2nd year BMS.

Environmental forensics is defined as the systematic and scientific evaluation of physical, chemical and historical information for the purpose of developing defensible scientific and legal conclusions regarding the source or age of a contaminant release into the environment. In other words, Environmental forensics is a specialized field that employs scientific principles to investigate and solve environmental crimes. It bridges the gap between environmental science and the legal system, aiming to identify the source, timing, and responsible parties for environmental contamination.

It is a multidisciplinary scientific approach involving the application of principles from various fields such as chemistry, biology, geology, and environmental science to solve environmental problems.

The **historical background** and origins of environmental forensics can be traced back to the early environmental concerns prior to the 1960s. During this period, there was limited public awareness about pollution, and contamination events were often perceived as isolated or accidental occurrences, lacking systematic investigation. However, the environmental movement that emerged during the 1960s and 1970s brought a significant shift in public consciousness. Influential works like Rachel Carson's *Silent Spring* (1962) highlighted the dangers of unregulated chemical use and sparked widespread environmental awareness. This period also witnessed several major pollution incidents, such as the Love Canal disaster in 1978 in the United States, which exposed the severity of industrial contamination and its health impacts.

In response, a series of **landmark environmental laws** were introduced to regulate and monitor environmental quality. The National Environmental Policy Act (NEPA) in 1970, the Clean Water Act in 1972, and most notably the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), commonly known as the Superfund Act, in 1980, marked crucial milestones.

Applications of environmental forensic includes: Investigation of environmental impact of illegal waste disposal on soil, water, and air quality; Investigating the pollution; Analysing environmental data to understand the impacts of climate change

and identify sources of greenhouse gas emissions; Assessing the environmental impact of industrial activities and ensuring compliance with environmental regulations; Providing scientific evidence to support remediation plans and monitor the effectiveness of cleanup efforts; also significantly used in investigating the origin of ground water contamination, environmental impact of illegal waste disposal on soil, water, and air quality.

There are some commendable advancements in the field of environmental forensics like usage of High-Resolution Mass Spectrometry (HRMS) and Compound-Specific Isotope Analysis (CSIA) for accuracy in identifying and quantifying even trace levels of contaminants and for more detailed investigations into the origin and fate of pollutants respectively.

Also, in the recent days there is a significant Integration of Data Science and Artificial Intelligence (AI) like application of machine learning algorithms which can analyze vast datasets from various sources to identify patterns, predict contamination events, and optimize investigative efforts.

SOURCE: http://www.science-scene.org/blog/environmental-forensics

# VOICES THAT SHAPED OUR VISION

*From the Frontlines of Discovery, Biomed Futura has welcomed some truly inspiring voices and trailblazers whose work reshaped how we see biology, technology, and the future of healthcare. These events weren't just talks but they were moments of deep connection, spirited debate, and visionary exploration. Below is a curated look at the brilliant minds who joined us this semester, each one adding a new lens to our evolving journey.*

**Dr P CHELLAPANDI**

*23rd July,2024*

PROFESSOR & HEAD
DEPARTMENT OF BIOINFORMATICS
BHARATHIDASAN UNIVERSITY, TRICHY

**"MOLECULAR VIRULENCE MECHANISM OF *CLOSTIRIDIUM BOTULINUM* – A LOCHE MAREE ON HUMAN GI TRACT "**

*30th July,2024*

**Dr RAMASWAMY BABU RAJENDRAN**

SENIOR PROFESSOR
DEPARTMENT OF ENIRONMENTAL BIOTECHNOLOGY
BHARATHIDASAN UNIVERSITY, TRICHY

**"ENVIRONMENTAL FORENSICS"**



**Dr R SENTHILKUMAR**

*6th August, 2024*

ASSOCIATE PROFESSOR
DEPARTMENT OF BIOCHEMISTRY
THANTHAI PERIYAR GOVERNMENT ARTS &SCIENCE COLLEGE, TRICHY

**"ACTIVATION OF INNATE IMMUNE CELLS THROUGH TTNF & TNFR LIGAND MEDIATED REVERSE SIGNALING MECHANISMS"**

*13th August,2024*

**Dr MAHARAJA PONNAIAH**

DATA SCIENTIST & HEAD
ICAN I/O – DATA SCIENCE
SORBENNE UNIVERSITY, GERMANY

**"AI FOR PERSONALIZED MEDICINE "**

**Dr N AMSAVENI**

*13th August,2024*

NODAL OFFICER, RAGGING PREVENTION COMMITTEE,
BHARATHIDASAN UNIVERSITY, TRICHY

**"ANTIRAGGING WEEK CELEBRATION"**

*28th August,2024*

**Dr M ANUSUYADEVI JEYACHANDARN**

ASSISTANT PROFESSOR
DEPARTMENT OF BIOCHEMISTRY
BHARATHIDASAN UNIVERSITY, TRICHY

**"RESVERATOL: A MULTI THERAPEUTIC DRUG TARGET AND A POTENTIAL NEURO PROTECTANT AGAINST AGE ASSOCIATED NEURO DEGENERATION. WITH SPECIAL EMPHASIS ON AGING AND ALZEIMER'S DISEASE "**

**Dr A UMAMAHESHWARI**

*5th September,2024*

ASSISTANT PROFESSOR
DEPARTMENT OF PHARMACEUTICAL TECHNOLOGY
ANNA UNIVERSITY, TRICHY

**"DRUG DESIGNING & BIOINFORMATICS - REVOLUTIONIZING PHARMACEUTICAL"**

# BASE EDITING EXTENDS LIFESPAN

## in Prion Disease Mouse Model

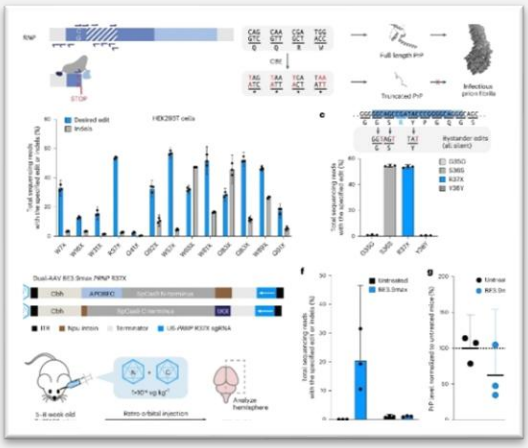
Nivetha J - 4th year BMS


## Abstract:

Prion diseases are a group of fatal neurodegenerative disorders characterized by the misfolding and aggregation of prion proteins (PrP). Currently, no effective treatments exist. Recent research has demonstrated the potential of base editing, a precise gene-editing technology, to reduce PrP levels and extend lifespan in a mouse model of prion disease. This review summarizes these findings and discusses the implications for developing future therapies for this devastating condition.


## Introduction:

Prion diseases, including Creutzfeldt-Jakob disease (CJD), fatal familial insomnia (FFI), and Gerstmann-Sträussler-Scheinker syndrome (GSS), are 1 caused by the misfolding of the cellular prion protein This misfolded protein acts as a template, into a pathogenic isoform converting normal leading to a cascade of protein aggregation,neuronal dysfunction, and ultimately, death. The lack of effective treatments highlights the urgent need for novel therapeutic strategies.



SOURCE: An, M., Davis, J.R., Levy, J.M. et al. In vivo base editing extends lifespan of a humanized mouse model of prion disease.

## Base Editing:

A Precise Gene-Editing Tool: Base editing is a revolutionary gene-editing technique that enables precise single-base conversions in DNA without inducing double-strand breaks. This approach utilizesa Cas enzyme fused to a deaminase, which directly converts one base to another (e.g., C to T, A to G) within a targeted DNA sequence. This precision offers significant advantages over traditional gene editing methods like CRISPR-Cas9, which can introduce unintended insertions or deletions.

## Targeting PrP with Base Editing:

A recent study published in Nature Medicine by researchers at the Broad Institute of MIT and Harvard demonstrated the therapeutic potential of base editing for prion disease. The research team, led by Sonia Vallabh, Eric Minikel, and David Liu,

focused on reducing PrP levels by introducing a specific mutation (R37X) known to be protective in humans. This naturally occurring mutation, identified through population databases like gnomAD, reduces PrP levels without causing adverse effects.

**Experimental Findings:** The study employed adeno-associated viruses to deliver the base-editing machinery to the brains of mice infected with a human version of the prion protein. The results were compelling:

Reduced PrP levels: The base editing system successfully installed the R37X edit in a significant proportion of targeted genes, leading to a substantial reduction (up to 63% with improved systems) in PrP levels in the brain.

Extended lifespan: Mice treated with the base editing therapy exhibited a significant increase in lifespan, approximately 50% longer compared to untreated controls.

Improved delivery and efficiency: The researchers iteratively improved their system, enhancing editing efficiency and minimizing off-target effects and delivery to other tissues.

**Implications for Prion Disease Therapy:** These findings represent a significant advancement in the search for prion disease treatments. The study provides the first direct evidence that lowering PrP levels can significantly impact disease progression and extend survival in a relevant animal model. Key advantages of this approach include:

**Potential for a one-time treatment**: Base editing offers the possibility of a single-administration therapy due to the permanent nature of the DNA edit.

**Broad applicability**: The strategy targets the PrP gene itself, making it potentially effective for all forms of prion disease, regardless of the underlying genetic mutation or origin (genetic, sporadic, or acquired).

**Inspiration from human genetics:** The use of a naturally occurring protective mutation (R37X) strengthens the therapeutic rationale and increases confidence in its potential safety.

## Future Directions:

Optimization of delivery: Further improvements in AAV delivery are needed to maximize editing efficiency and minimize off-target effects.

Cargo size reduction: Reducing the size of the base-editing cargo is crucial for efficient packaging into AAVs, particularly when using dual AAV systems.

Exploration of alternative editing strategies: Future research will explore prime editing to introduce more complex protective mutations that modify PrP function rather than simply reducing its production.

Translation to human clinical trials: Rigorous preclinical studies,safety assessments are essential before translating this approach to human clinical trials.

# அமேசானின் நெகிழி உண்ணும் பூஞ்சை: உயிரியல் பெஸ்டலோசியோப்சிஸ் மைக்ரோஸ்போரா (Pestalotiopsis microspora) பற்றிய ஒரு ஆய்வு

ரா.ஜனனி.நான்காம் ஆண்டு

## சுருக்கம்:

நெகிழி மாசுபாடு (plastic pollution) என்பது நமது காலத்தின் மிக அவசரமான சுற்றுச்சூழல் நெருக்கடிகளில் ஒன்றாகும். இதற்கு வழக்கமான மறுசுழற்சி முறைகளுக்கு அப்பால் புதுமையான தீர்வுகள் தேவை. நெகிழியைச் சிதைக்கும் திறன் கொண்ட புதிய நுண்ணுயிரிகளின் கண்டுபிடிப்பு, உயிர்ப்புத்தாக்கத்திற்கு (bioremediation) ஒரு நம்பிக்கைக்குரிய வழியை வழங்குகிறது. இந்த ஆய்வு, அமேசான் மழைக்காடுகளில் இருந்து பிரித்தெடுக்கப்பட்ட ஒரு அகவுயிரி பூஞ்சையான (endophytic fungus) பெஸ்டலோசியோப்சிஸ் மைக்ரோஸ்போராவை மையமாகக் கொண்டுள்ளது. இது பாலியூரித்தேனை (polyurethane) சிதைக்கும் தனித்துவமான திறனுக்காக அறியப்படுகிறது. கழிவு மேலாண்மை மற்றும் நெகிழி மாசுபாடு இல்லாத எதிர்காலத்திற்கான அதன் மகத்தான திறனை எடுத்துரைக்கும் அதே வேளையில், சுற்றுச்சூழல் சுத்திகரிப்பு முயற்சிகளில் அதன் பெரிய அளவிலான பயன்பாட்டிற்கு முக்கியமான எதிர்கால ஆராய்ச்சி திசைகளையும் இந்த ஆய்வு குறிப்பிடுகிறது.

## அறிமுகம்:

நெகிழி கழிவுகளின் இடைவிடாத குவிப்பு, நிலப்பரப்பு மற்றும் நீர்வாழ் சூழல் அமைப்புகளை உலகளவில் பாதிக்கும் ஒரு பரவலான சுற்றுச்சூழல் அச்சுறுத்தலாக மாறியுள்ளது. மறுசுழற்சி மற்றும் குப்பைக் கிடங்குகளில் சேகரிப்பது போன்ற வழக்கமான கழிவு மேலாண்மை உத்திகள், செயற்கைப் பாலிமர்களின் அளவு மற்றும் நிலைத்தன்மையைக் கையாள பெரும்பாலும் போதுமானதாக இல்லை. நெகிழிகள் சுற்றுச்சூழலில் நூற்றுக்கணக்கான முதல் ஆயிரக்கணக்கான ஆண்டுகள் வரை நீடித்து, பரவலான மாசுபாடு, வனவிலங்குகளுக்கு தீங்கு மற்றும் மனித ஆரோக்கியத்திற்கு சாத்தியமான அபாயங்களை ஏற்படுத்துகின்றன. இந்த அவசர நெருக்கடி, உயிர்ப்புத்தாக்கம் எனப்படும் உயிரியல் ரீதியாக இயக்கப்படும் புதிய தீர்வுகளை ஆராய அறிவியல் முயற்சிகளைத் தூண்டியுள்ளது. நுண்ணுயிரிகள், குறிப்பாக பூஞ்சைகள் மற்றும் பாக்டீரியாக்கள், சிக்கலான கரிமச் சேர்மங்களை உடைக்கக்கூடிய பலவிதமான நொதி பொறிமுறைகளைக் கொண்டுள்ளன. இது நெகிழிகள் போன்ற எளிதில் சிதையாத மாசுபடுத்திகளைச் சிதைப்பதற்கு சிறந்த வேட்பாளர்களாக அமைகின்றன. இந்தத் துறையில் மிக அற்புதமான கண்டுபிடிப்புகளில் ஒன்று பெஸ்டலோசியோப்சிஸ் மைக்ரோஸ்போரா ஆகும். இது பூமியின் மிகப்பல உயிரியல் பன்முகத்தன்மை கொண்ட பகுதிகளில் ஒன்றான அமேசான் மழைக்காடுகளில் காணப்படும் ஒரு பூஞ்சை. நெகிழி உயிர்ப்புத்தாக்கத்தில் பெஸ்டலோசியோப்சிஸ் மைக்ரோஸ்போராவின் பங்கு, அதன் பண்புகள், செயல்பாட்டு முறை மற்றும் எதிர்கால திசைகள் பற்றிய தற்போதைய புரிதலை ஒருங்கிணைப்பதே இந்த ஆய்வின் நோக்கமாகும்.

பெஸ்டலோசியோப்சிஸ் மைக்ரோஸ்போராவின் கண்டுபிடிப்பு மற்றும்

## பண்புகள்:

பெஸ்டலோசியோப்சிஸ் மைக்ரோஸ்போராவின் நெகிழி-சிதைக்கும் திறன்களின் திருப்புமுனையான கண்டுபிடிப்பு 2011 இல் நிகழ்ந்தது. யேல் பல்கலைக்கழக இளங்கலை மாணவர்களின் ஒரு குழு, ஆண்டுதோறும் நடைபெறும் மழைக்காடு ஆய்வு மற்றும் ஆய்வகப் பாடத்திட்டத்தில் பங்கேற்று, ஈக்வடார் அமேசான் மழைக்காடுகளுக்குள் உள்ள யாசுனி தேசிய வனத்தில் உள்ள மரத்தாலான தாவரங்களின் தண்டுகளில் இருந்து இந்த அகவுயிரி பூஞ்சையைப் பிரித்தெடுத்தது. அகவுயிரி பூஞ்சைகள், தாவர திசுக்களுக்குள் வெளிப்படையான நோயை ஏற்படுத்தாமல் வாழும் நுண்ணுயிரிகள் ஆகும். இவை பெரும்பாலும் கூட்டு வாழ்க்கை உறவுகளை உருவாக்குகின்றன.இந்த பிந்தைய பண்பு, குப்பைக் கிடங்குகள் போன்ற ஆக்ஸிஜன் மிகவும் குறைவாக உள்ள சூழல்களில் உயிர்ப்புத்தாக்கப் பயன்பாடுகளுக்கு மிகவும் முக்கியமானது.

## நெகிழி சிதைவின் பொறிமுறை:

பெஸ்டலோசியோப்சிஸ் மைக்ரோஸ்போரா, குறிப்பிட்ட நொதிகளை சுரப்பதன் மூலம் பாலியூரித்தேனை சிதைக்கிறது. இந்தச் செயல்பாட்டில் செரின் ஹைட்ரோலேஸ் (serine hydrolase) ஒரு முக்கிய நொதியாக அடையாளம் காணப்பட்டுள்ளது. இந்த நொதிகள் பூஞ்சையால் உற்பத்தி செய்யப்பட்டு, பின்னர் பாலியூரித்தேனின் நீண்ட பாலிமர் சங்கிலிகளில் செயல்படுகின்றன. இந்த நொதிகள் சிக்கலான செயற்கைப் பாலிமர்களை சிறிய, எளிய கரிமச் சேர்மங்களாக (ஒலிகோமர்கள் மற்றும் மோனோமர்கள்) உடைக்கின்றன. பூஞ்சை பின்னர் இந்த சிறிய மூலக்கூறுகளை உறிஞ்சி, ஆற்றல் மற்றும் வளர்ச்சிக்கு கார்பன் மூலமாகப் பயன்படுத்திக் கொள்ள முடியும். இது நெகிழியை பூஞ்சை உயிரி நிறை, கார்பன் டை ஆக்சைடு மற்றும் நீராக திறம்பட மாற்றுகிறது. இந்த நொதி அடிப்படையிலான சிதைவு, உயிர்ப்புத்தாக்கச் செயல்பாட்டில் ஒரு முக்கியமான படியாகும். இது நிலைத்திருக்கும் நெகிழி கழிவுகளை குறைந்த தீங்கு விளைவிக்கும் அல்லது பயனுள்ள கரிமப் பொருளாக மாற்றுகிறது.

## முடிவுரை:

பெஸ்டலோசியோப்சிஸ் மைக்ரோஸ்போராவின் கண்டுபிடிப்பு, நெகிழி மாசுபாட்டிற்கு உயிரியல் தீர்வுகளைத் தேடும் பயணத்தில் ஒரு குறிப்பிடத்தக்க மைல்கல்லைக் குறிக்கிறது. பாலியூரித்தேனை, குறிப்பாக காற்றில்லா சூழ்நிலைகளில், சிதைக்கும் அதன் தனித்துவமான திறன், குப்பைக் கிடங்கு சீர்திருத்தத்திற்கும், மற்ற கழிவு மேலாண்மை சூழ்நிலைகளுக்கும் ஒரு நம்பிக்கைக்குரிய கருவியாக அமைகிறது. எதிர்கால ஆராய்ச்சி முயற்சிகள் முக்கியமானவை. . இது இயற்கையின் அற்புதமான சுய-குணப்படுத்தும் திறனை வெளிப்படுத்துகிறது. இது ஒரு முழுமையான தீர்வு இல்லாவிட்டாலும், அதன் தனித்துவமான பண்புகள் ஒரு சக்திவாய்ந்த உயிரியல் கருவியை வழங்குகின்றன. இது தொடர்ச்சியான அறிவியல் கண்டுபிடிப்புகள் மற்றும் கவனமான பரிசீலனையுடன், உலகளாவிய நெகிழி நெருக்கடியைக் குறைப்பதிலும், மேலும் நிலையான எதிர்காலத்தை வளர்ப்பதிலும் முக்கியப் பங்காற்ற முடியும்.

# CASE STUDY ON SPINAL MUSCULAR ATROPHY

## (SMA) Type 2

Joan C - 3rd year BMS

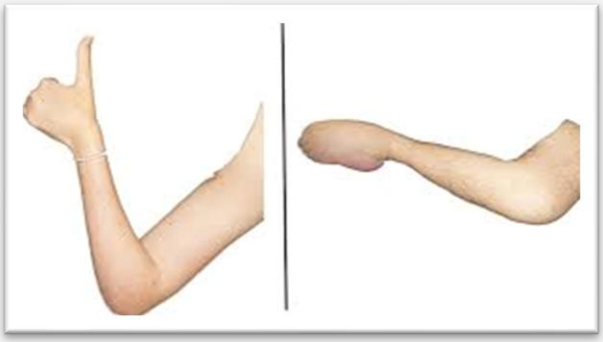
**A 2-year-old child, Shreenika, was a case with complaints of inability to sit unsupported for extended periods without support and cannot stand or walk, even with assistance. It was suspected to be a genetic disorder, and the primary test is to identify mutations in the SMN1 gene. Additionally, a clinical evaluation electromyography (EMG), nerve conduction studies, was used to confirm the diagnosis and assess the severity. After diagnosis the Spinal Muscular Atrophy (SMA) Type II, a genetic disorder was identified that causes progressive muscle weakness. This SMA Type 2 is an intermediate form of SMA and appears between six months and 18 months of age.**

The disease is inherited and is caused by a mutation in the survival motor neuron-1 (SMN1) gene. The SMN protein is critical to the health and survival of the nerve cells in the spinal cord responsible for muscle contraction (motor neurons). This disease is diagnosed at an early stage of an infant. The SMA disease has 4 types: **Type 1**(most common and severe), **Type 2**(intermediate), **Type 3**(milder), and **Type 4**(very rare). Around 70% of people with type 2 will survive until 25, with some surviving into their 30s. Respiratory issues are the major cause of death.

## PREVENTION AND CONTROL METHODS:

Physical therapy, can help improve posture, prevent joint immobility, and slow muscle weakness.

- ➢ Occupational therapy can improve your ability to perform daily tasks.
- ➢ Assistive devices, like ortho-paedic braces, crutches, walkers, and wheelchairs.
- ➢ Therapy for speech and swallowing difficulties.
- ➢ A feeding tube if swallowing is too difficult and/or dangerous.



SOURCE: https://www.researchgate.net/figure/Pattern-of-muscle-atrophy-in-SMA-Patient-with-SMA-type-2-showing-the-typical-pattern-of_fig3_383469674

## TREATMENTS:

- ✓ **Gene replacement therapy:** Children younger than 2 may benefit from a one-time intravenous (IV) infusion of a medication called onasemnogene abeparvovec-xioi

(Zolgensma). This therapy replaces a missing or faulty SMN1 gene with a functioning gene.

Current research in SMA includes expanding **Zolgensma gene therapy** to older patients, testing **muscle-enhancing drugs** like Apitegromab, and using **SMN2 splicing modifiers** such as Nusinersen and Risdiplam. Other promising areas include **epigenetic therapies**, **neuroprotection using biomarkers**, and **spinal implants for electrical muscle stimulation**.

# HAEMODIALYSIS

Pooja Priyadharshini G – 2nd year BMS

**Key Components & Functions**

1. Blood Removal: Blood is drawn from the patient via a vascular access.
2. Arterial Pressure Monitor: Checks blood pressure before the pump.
3. Blood Pump: Controls blood flow through the system.
4. Heparin Pump: Prevents clotting by injecting anticoagulant.
5. Dialyzer (Filter): Filters waste and excess fluid via a semi-permeable membrane.
6. Dialyzer Inflow Pressure Monitor: Ensures proper pressure before filtering.
7. Air Trap & Detector: Removes and detects air bubbles in returning blood.
8. Venous Pressure Monitor: Checks pressure before returning blood to body.
9. Air Detector Clamp: Stops flow if air is detected.
10. Filtered Blood Returned: Clean blood is sent back into the body.

**Applications of Hemodialysis**

1. Chronic Kidney Disease (CKD):

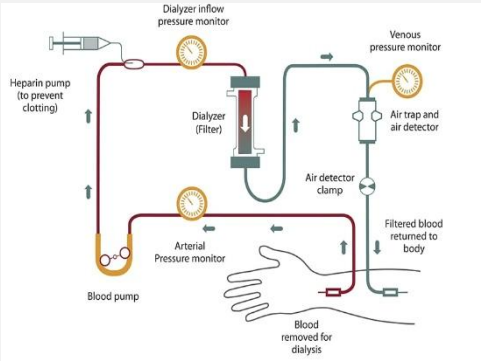
Maintains life in patients with end-stage renal disease (ESRD).

2. Acute Kidney Injury (AKI):

Temporarily supports kidney function in critically ill patients.

3. Toxin Removal:

Used in cases of poisoning or overdose (e.g., lithium, methanol).




SOURCE: https://franciscokidneycentre.com/haemodialysis-vs-peritoneal-dialysis/

# MEDICAL BREAK THROUGH

## THE DISCOVERY OF A NEW DRUG FOR "CROHN'S DISEASE"

Nishanthini K - 3rd year BMS.

## Introduction:

Crohn's disease is a chronic inflammatory bowel disorder characterized by inflammation of the gastrointestinal tract, leading to symptoms such as abdominal pain, diarrhea, weight loss, and fatigue. Over the years, advancements in medical research have led to the development of various therapeutic agents aimed at managing this condition. This overview highlights some of the notable drugs introduced for Crohn's disease, their mechanisms, and the scientists behind their discovery.

## Some recently developed drugs for Crohn's disease are:

- Upadacitinib
- Duvakitug
- Mirikizumab
- Ustekinumab biosimilars
- Etrasimod

**1. Upadacitinib (Rinvoq)**

Upadacitinib is an oral Janus kinase (JAK) inhibitor that modulates immune responses by targeting specific pathways involved in inflammation. In May 2023, the U.S. Food and Drug Administration (FDA) approved upadacitinib for adults with moderately to severely active Crohn's disease who have had an inadequate response or intolerance to one or more tumor necrosis factor (TNF) blockers. Clinical trials demonstrated that patients receiving upadacitinib experienced significant improvements in clinical remission and endoscopic response compared to placebo.

**2. Duvakitug**

Duvakitug is an investigational human monoclonal antibody targeting the TL1A protein, which is implicated in inflammatory processes. Developed collaboratively by Teva Pharmaceuticals and Sanofi, duvakitug has shown promising results in Phase 2b clinical trials. In studies reported in December 2024, patients with ulcerative colitis and Crohn's disease receiving duvakitug achieved higher clinical remission rates compared to those receiving a placebo. Specifically, remission rates for Crohn's disease patients were 26.1% and 47.8% for low and high dosages, respectively, outperforming the placebo group. These encouraging outcomes have led to plans for Phase 3 trials to further evaluate the efficacy and safety of duvakitug.

**3. Mirikizumab (Omvoh)**

Mirikizumab, marketed as Omvoh, is a monoclonal antibody that targets interleukin-23 (IL-23), a cytokine involved in inflammatory responses. Initially approved for the treatment of moderate-to-severe ulcerative colitis, mirikizumab received FDA approval in January 2025 for treating moderate-to-severe Crohn's disease in adults. Clinical trials indicated that 53% of patients achieved remission after one year of treatment with mirikizumab, compared to 36% with a placebo. This approval expands the therapeutic options available for Crohn's disease, particularly for patients who have not responded adequately to existing treatments.

**4. Ustekinumab Biosimilars**

Ustekinumab is a monoclonal antibody that inhibits interleukins 12 and 23, playing a significant role in managing Crohn’s disease. In December 2024, the FDA approved several biosimilar versions of ustekinumab, including products marketed under names such as Wezlana and Imuldosa. These biosimilars offer more treatment options and may improve accessibility for patients requiring biologic therapies for Crohn's disease.

**5. Etrasimod (Velsipity)**

Etrasimod, sold under the brand name Velsipity, is an oral selective sphingosine-1-phosphate (S1P) receptor modulator that modifies immune activity. While primarily approved for the treatment of moderate-to-severe ulcerative colitis in October 2023, ongoing clinical trials are investigating its potential efficacy in Crohn’s disease. Etrasimod works by preventing immune cells from migrating to inflamed tissues, thereby reducing inflammation. If successful in trials, etrasimod could offer another oral therapeutic option for patients.

## Conclusion:

The evolution of therapeutic strategies for Crohn’s disease underscores the relentless efforts of the scientific community to address this debilitating condition. From the advent of TNF-α inhibitors like infliximab and adalimumab to the development of integrin blockers and JAK inhibitors, each advancement offers a more targeted approach to managing the disease. The recent introduction of agents like duvakitug and Omvoh highlights the ongoing commitment to expanding and refining treatment options, aiming to improve the quality of life for those affected by Crohn’s disease.

# GERIATRIC PATIENT WITH CORONARY ARTERY DISEASE-ASSOCIATED DIABETIC FOOT ULCER

Udhayakumar K- 4th year, BMS.

Diabetes mellitus and cardiovascular diseases are leading causes of morbidity in elderly populations. Diabetic foot ulcers (DFUs), especially when compounded by coronary artery disease (CAD), pose significant challenges due to delayed wound healing, infection risk, and high mortality rates. This case study highlights a geriatric patient diagnosed with a diabetic foot ulcer in the presence of advanced coronary artery disease, emphasizing the importance of multidisciplinary care in complex disease associations.

## Case Overview:

A 66-year-old male was admitted to the cardiology ward with complaints of chest pain, breathlessness, sweating, and cough. He also reported a loss of sensation over his foot. His medical history included type 2 diabetes mellitus and hypertension. He had an ulcer on his right big toe that progressed to gangrene following trauma 20 days prior. Echocardiography revealed moderate left ventricular systolic dysfunction (EF: 38%). Coronary angiogram showed calcified triple-vessel disease. He was diagnosed with a diabetic foot ulcer associated with coronary artery disease and was hospitalized for further management.

## Treatment and Recovery:

Cardiovascular Management:
Antiplatelet agents (aspirin/clopidogrel)
Statins for dyslipidemia
Beta-blockers for myocardial oxygen demand
ACE inhibitors/ARBs to improve cardiac output and control blood pressure

## Foot Ulcer Management:

Wound debridement
Culture-specific antibiotics
Advanced wound dressing techniques
Offloading methods to reduce pressure on the affected area

## Discussion:

The presence of both diabetic foot ulcer and coronary artery disease in a geriatric patient requires a tailored and comprehensive care approach. Such patients are at high risk for poor healing and recurrent hospitalizations. This case demonstrates the effectiveness of early cardiovascular stabilization and aggressive ulcer management. Importantly, the clinical pharmacist's role was central in educating the patient, managing medications, and ensuring adherence to lifestyle changes—thereby improving treatment outcomes.

## Future Perspective:

Early screening for peripheral vascular disease and foot complications
Routine cardiovascular evaluation in diabetic patients
Multidisciplinary care teams involving physicians, nurses, pharmacists, and dietitians
Use of digital health tools for monitoring and follow-up

# BIRKBAREL SYNDROME

**Vedha Valli T- 4th year BMS.**

## Introduction:

Birk-Barel syndrome, also known as KCNK9 imprinting syndrome, is a rare neurodevelopmental disorder first described in 2008. It is inherited in an autosomal dominant manner with paternal imprinting and is caused by a specific missense mutation in the maternal copy of the KCNK9 gene. To date, approximately 21 cases have been reported in the literature, with 15 originating from the original Arab-Israeli family described by Barel et al. This report presents the first known case of Birk-Barel syndrome in a patient of Puerto Rican ethnicity, who showed clinical improvement in central apneas with the use of non-invasive ventilation.

## Case Presentation:

A 16-month-old female of Puerto Rican was admitted to a community hospital with cough and respiratory distress that progressed to acute respiratory failure requiring PICU(Pediatric Intensive Care Unit) admission. She is the first child of healthy, non-consanguineous parents and was born prematurely at 36 weeks via vaginal delivery, with a birthweight and length below the 3rd percentile.

## Mechanisms:

This disease is attributed primarily to a genetic mechanism involving a heterozygous missense mutation (p.Gly236Arg) in the maternal allele of the KCNK9 gene, located on chromosome 8q24.3. It is caused by a specific missense mutation 770G> A in exon 2, replacing glycine at position 236 by arginine (G236R) in the maternal copy of KCNK9 within this locus. This gene encodes the TASK3 potassium channel, which is crucial for regulating neuronal excitability, maintaining resting membrane potential, and supporting normal brain development—especially in the cerebellum and brainstem. The mutation disrupts channel function, leading to impaired neural signaling and altered control of respiratory drive, which explains the severe central sleep apneas observed in the patient.

## Symptoms:

These include congenital hypotonia, cleft palate, bilateral hand contractures, talipes , face, short philtrum, and epicanthal folds. She also experienced recurrent respiratory infections and developed acute respiratory failure requiring PICU admission. Legends of the Picturistic representation of symptoms are given below as A, B, C & D

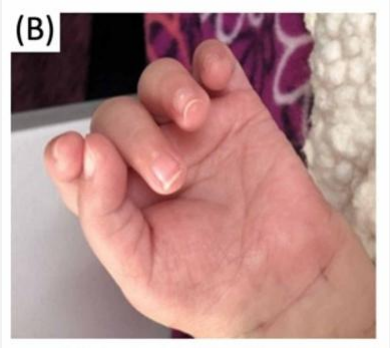
## Diagnosis:

Diagnosis was established through clinical evaluation and confirmed by whole-exome sequencing, which revealed a heterozygous missense mutation (p.Gly236Arg) in the KCNK9 gene, consistent with KCNK9 imprinting syndrome (Birk-Barel syndrome). Additional diagnostic tests which revealed severe central sleep apnea with an apnea-hypopnea index (AHI) of 35.9 events/hour initially, later worsening to 117.3 at age 3.
Chest x-ray shows bilateral perihilar opacities with right upper lobe atelectasis. Abnormal curvature of spine consistent with severe dextroscoliosis

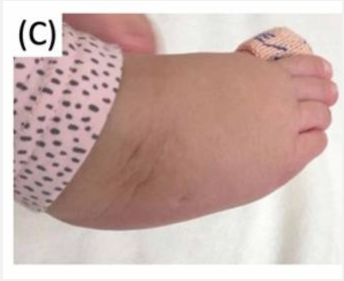
## Treatment:

Treatment included non-invasive ventilation (NIV) with a nasal mask and BiPAP (Bi-level Positive Airway Pressure) settings during sleep which helped maintain airway patency and improve oxygenation, supplemental oxygen at 1 LPM was provided to keep oxygen saturation above 92%, and an airway clearance regimen with albuterol, hypertonic saline nebulization, and chest physiotherapy was implemented to reduce airway secretions and improve respiratory function. The patient's feeding difficulties were managed with a gastrostomy tube for long-term nutrition. Following 15 days of NIV therapy, significant improvements in motor skills, alertness, and social interaction were observed. A multidisciplinary approach was established upon discharge, involving specialists in neurology, pulmonology, gastroenterology, rehabilitation, speech therapy, and ophthalmology to support her complex needs. Despite initial improvements, follow-up polysomnography at age three revealed worsening apnea severity, underscoring the need for ongoing respiratory monitoring and care.


**Premature baby with bilateral feet deformalities**




**Tapered fingers**




**Talipes Equinovarus**


**Face with deformalities**

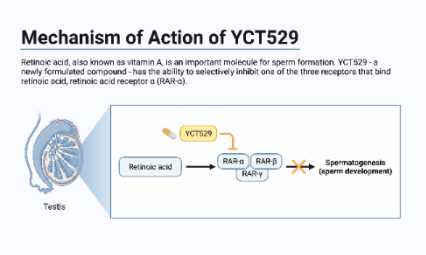
Source:
https://pmc.ncbi.nlm.nih.gov/articles/PMC8301725/

# YCT-529: THE FIRST NON-HORMONAL MALE CONTRACEPTIVE PILL

**Hari hara sudhan T - 4th year BMS**

**Context :**

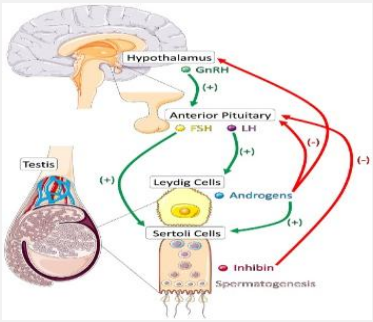
For decades, contraceptive responsibility has largely been shouldered by women through hormonal pills, IUDs, and implants while men had only two main options: condoms or permanent vasectomy(reversals exist but are expensive, costing up to $7,000, and are not guaranteed to work) Research from the University of Minnesota College of Pharmacy, published in **Communications Medicine**, laid the groundwork for the first hormone-free male birth control pill to enter clinical trials. Despite it has modern advances in reproductive health, the development has become safe, reversible male birth control has remained elusive. A team of scientists introduced a new option just for men: **A nonhormonal-contraceptive pill** that does not mess with testosterone, does not reduce sex drive and best among this is reversible. This game changing pill is called **YCT-529.**




SOURCE: https://www.biorender.com/template/mechanism-of-action-of-yct529

## The Breakthrough of YCT-529:

In July 2025, researchers successfully completed Phase 1a human trials for YCT-529, a non-hormonal oral male contraceptive. The pill acts by targeting the retinoic acid receptor alpha (RAR-α), which plays a key role in the production of sperm. In early 2025, it was successfully tested in healthy man during a phase 1 clinical trial.




SOURCE: https://www.researchgate.net/figure/Hypothalamic-pituitary-gonadal-axis-Figure-modified-with-text-markings-and-annotation_fig1_369108832

## The Science behind YCT-529:

YCT-529 is a selective antagonist of the **RAR-α receptor**, a nuclear receptor essential for sperm development. RAR-α is activated by **retinoic acid** (a form of vitamin A) which drives the differentiation of spermatogonia into mature sperm cells.

By blocking RAR-α, YCT-529 halts sperm production at the source, preventing new sperm from forming while leaving hormones like testosterone unaffected. Because the drug targets a specific receptor only involved in testicular function, it avoids many of the side effects seen in hormone-based therapies.

**Supporting Data & Results:**

Preclinical studies in mice: 99% effective in preventing pregnancies with no hormonal disruption. Full fertility returned within 4–6 weeks after stopping the drug. Primate studies: Similar sperm suppression observed, with recovery in 10–15 weeks post-treatment.

Phase 1a human trial (2025):

- 16 healthy men (mostly post-vasectomy) took single ascending doses up to 180 mg.
- No significant side effects, no mood changes, and no effect on testosterone or libido.

## Impact of the Breakthrough:

YCT-529 represents a revolution in male contraception. If future trials confirm its effectiveness in suppressing sperm production over weeks or months, it could:

- Provide men with safe, reversible contraceptive control. Relieve contraceptive burden on women, promoting reproductive equality
- Reduce reliance on permanent or barrier-based methods. Encourage broader shared responsibility in family planning

This breakthrough could also transform public health, especially in countries where access to female contraceptives is limited.

**Limitations & Next Steps:**

While the early results are promising, YCT-529 is still under testing. Key limitations and upcoming steps include:

| Trial Phase | Timeline |
|---|---|
| Phase 1a (safety) | Completed 2024 |
| Phase 1b/2a (efficacy) | 2025 (ongoing) |
| 90- day mid-stage trial | Late 2025 |
| Phase 3 & Approval | Likely by 2027-2030 |

- Larger Phase 2 trials in men who have not had vasectomies, to evaluate actual sperm suppression. Long-term safety and reversibility studies
- Public education and social acceptance, which may influence adoption. Regulatory approval and production scalability

## Conclusion:

The success of YCT-529 signals a paradigm shift in contraception. For the first time, a safe, non-hormonal, reversible pill for men is moving through clinical trials with real potential. If fully validated, YCT-529 will not only balance the scales of reproductive responsibility but also pave the way for next-generation biomedical solutions in fertility control.

# BIOREMEDIATION IN POLLUTION CONTROL

**Varshini S - 4th year BMS**

## Introduction:

Growing rate of industrialization and urbanization has led to increase in several types of pollution caused by release of chemicals like cyanide, sulphuric acid,etc... Remediation is the process to remove the contaminant from pollutant sites. Therefore there is a need for the remediation for the pollutants using physical, chemical, biological methods.

**Bioremediation:**

Biological remediation also known as bioremediation. It employs the use of biological agents such as fungi, bacteria and archaea are used as bioremediators to remove the pollutants.

## Microbes Used In Bioremediation:

In 1968 George M.Robinson, was the first to use bioremediation on large scale to clean up an oil spill in Santa Barbara, California. In 1970 Dr.Ananda M.Chakrabarty and his colleagues discovered a strain of bacteria isolating from the pseudomonas that is able to degrade some components in crude oil.The other microbes includes: Bacterium *Deinococcus radiodurans* has been used to digest toluene and ionic mercury from highly radioactive nuclear waste. Cyanobacteria, Mycobacteria, Green algae and molds and yeast can remove nearly 80% of hydrocarbon from oil spills within short span of one year.

**Strategies Of Bioremediation:**

Two stratagies i.e; in situ and ex situ

In situ bioremediation involves the biological degradation of contaminants to benign product onsite.

Ex situ process pollutants are removed from the contamination site and treated.

## Recent Advancement:

Recent advancements in microbial bioremediation includes: microbial biofilms, composed of polysaccharides extracellular DNA , proteins are used as bioremediation of organic pollutants. Bioelectrochemical systems combine biological and electrochemical method to control pollution.Microbial glycoconjugates from *Scedosporium sp*.and *Acinetobacter sp*. in biodegradation of petroleum hydrocarbons.

Therefore necessary to carry out further research to unravel technologies and mechanism to improve efficiency of bioremediation.

## Conclusion:

Bioremediation is the afforadable, sustainable, safe remediation process. It is a powerful tool to clean up thecontaminated sites. The process has been successfully applied to degrade pollutants, heavy metals, industrial chemicals.

# சூப்பர் ஸ்ட்ரெயினர்

பாக்டீரியாவின் தந்தை

Nivetha C – 4th year BMS

சுமார் 50 ஆண்டுகளுக்கு முன்பு பெரிய கண்டெய்னர்களில் கச்சா எண்ணெய்யை ஏற்றுமதி செய்தார்கள். அப்போது எண்ணெய் கசிவால் கடல் மாசு பட்டது. இதனால் பல்வேறு கடல்வாழ் உயிரினங்கள் பாதிப்படைந்தன. இதை தடுக்க என்ன செய்யலாம் என உலகமே யோசித்தது இப்படி கடல் மாசடைவதை சரி செய்ய ஒரு செயற்கை பாக்டீரியாவை கண்டறிந்தவர்தான் ஏ.எம்.சக்கரவர்த்தி

இவர் 1938 ஆம் ஆண்டு ஏப்ரல் 4-ம் தேதி கொல்கத்தாவில் பிறந்தார். அங்கு இருக்கும் செயின்ட் சேவியர்ஸ் கல்லூரியில் படித்தார். கொல்கத்தா பல்கலைக்கழகத்தில் வேதியலில் பி எச் டி செய்தார். சக்கரவர்த்தியின் அறிவியல் ஆர்வம் பேராசிரியர் எஸ்.சி.ராயை வெகுவாக கவர்ந்தது பயோ கெமிஸ்ட்ரியில் இருந்து மூலக்கூறு மரபணுத் துறைக்கு சக்கரவர்த்தியின் லட்சியத்தை மடைமாற்றினார் ராய்.

கார்பனும் ஹைட்ரஜனும் ஒன்றிணைந்து தான் ஹைட்ரோ கார்பன் உருவாகிறது. அப்படிப்பட்ட ஒரு கலவைதான் குருடு ஆயில் எனப்படும். கச்சா எண்ணெய் ஹைட்ரோ கார்பனின் வெவ்வேறு கலவைகளை சாப்பிட "சுடோமொனாஸ்" (Pseudomonas) செல்கின்ற பாக்டீரியாவில் முடிந்தது. இந்த பாக்டீரியாவால் கடல் நீரை அசுத்தப்படுத்தும் குருடு ஆயிலை (crude oil) உட்கொள்ள முடியும் . ஒவ்வொரு வித பாக்டீரியாவும் ஒவ்வொரு வித ஹைட்ரோ கார்பன் கலவைகளை மட்டுமே சாப்பிடும். மாறாக வேறு விதமானதை சாப்பிட்டால் இறந்து போகும் எனவே பல்வேறு விதமான ஹைட்ரோ கார்பன் கலவைகளை ஜீரணிக்கும் வல்லமையுள்ள பாக்டீரியா இனம் ஒன்று தேவை என்பதை உணர்ந்த சக்கரவர்த்தி, "சூப்பர் ஸ்ட்ரெயின்" பாக்டீரியாவை உருவாக்கினார்.

இது ஹைட்ரோ கார்பனை விழுங்கி ஜீரணம் செய்தது. விழுங்கப்பட்ட இந்த ஹைட்ரோ கார்பன்கள் சத்து நிறைந்த புரோட்டீனாக வெளியேறி கடல் வாழ் உயிரினங்களுக்கு உணவாகவும் மாறியது. தொடர் ஆராய்ச்சியின் முடிவில் மிதமான தட்பவெப்ப நிலையில் ஹைட்ரோ கார்பனை சாப்பிடும் நான்கு வித்தியாசமான பாக்டீரியாக்களை ஒரே இனமாக மாற்ற முடியும் என்பதை கண்டறிந்தார்.மேலும் இந்த சூப்பர் ஸ்ட்ரெயின் பாக்டீரியா தனக்குத்தானே இனவிருத்தி செய்து கொள்ளும் தன்மையும் பெற்றிருந்தது. கூடுதல் வரம் சக்கரவர்த்தியின் சேவையை பாராட்டி அவருக்கு மத்திய அரசு 2007-ஆம் ஆண்டு பத்மஸ்ரீ விருது கொடுத்து கவுரவித்தது. நம்முடன் வாழ்ந்து ,பல அரிய கண்டுபிடிப்புகளை நிகழ்த்திய சக்கரவர்த்தி 2020-ஆம் ஆண்டு மறைந்தார்.

# CASE STUDY ON ALUMINUM PHOSPHIDE POISONING

Srikanth V S -4th year BMS

## Introduction:

The patient is confused with a Glasgow coma scale(GCS) of 10 and his BP is 80/60 mmHg. Pulse 116/min and RR 24/min. His temperature is 37˚C and pupils are normal. Peripheral cyanosis is present with cold extremities. His chest is clear and his abdomen is soft with normal bowel sounds.

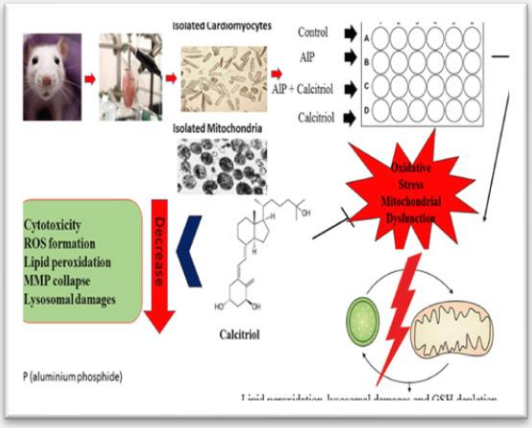
## Diagnosis:

The diagnosis finding of a rotten fish-like odour in the breath, shock, absent fasciculcation and with severe ALP poisoning. The discovery of the empty tube of ALP nearby confirms this. The investigations of the elevated levels of hemoglobin is 14g/L and total and differential counts are normal. The arterial blood gas analysis reveals a severe metabolic acidosis with hypoxia. ECG shows sinus tachycardia and occasional ventricular ectopics. Serum sodium 140mmol/L, potassium 3.9mmol/L, urea 6.8mmol/L, creatinine 88.4mmol/L and blood glucose 3.9mmol/L.

## Disease confirmation:

To confirm **Aluminium phosphide poisoning**, the presence of phosphine can be detected by using a filter paper impregnated with freshly prepared silver nitrate solution and holding it close to mouth and the colour will change to black if phosphine is present in the breath or in stomach contents.

**ALUMINIUM PHOSPHIDE POISONING** is a fumigant used to control insects and rodents in a variety of settings. Pain in the abdomen, vomiting and diarrhea are the initial symptoms and continue to remain conscious though anxious and confused, until coma develops as a result of cerebral hypoxia due to shock.




SOURCE: https://www.sciencedirect.com/science/article/abs/pii/S0045653522002934

## Treatment:

There is no specific antidote and the treatment is supportive. Patient should be managed with intensive care unit. Two intravenous lines should be secured, one for infusion of saline(0.9% sodium chloride) and the other for sodium bicarbonate.

A recent study suggests that careful correction of acidosis leads to reduced mortality but needs to confirmed with further investigations. Patient has severe shock, noradrenaline may also be administered. Urinary output should be measured hourly and glucocorticoids should be administered 100mg intravenously for every 6 hours.

# AVIAN INFLUENZA

Salai Meivazhvu S V – 4th year BMS

## Introduction:

On 2nd April 2025, the World Health Organization of the laboratory confirmed human infection with the AVIAN INFLUENZA A (H5N1)- MEXICO virus for the child under the age of 10 years from the state of Durango who tested positive for influenza A(H5N1) and did not have any underlying medical conditions, had not received seasonal influenza vaccination, and had no history of travel.

## Situation at a glance:

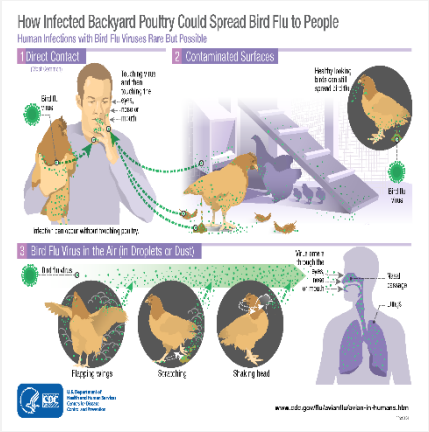
Symptoms began on 7 March 2025 with fever, malaise, and vomiting. On 13 March, the case was admitted to hospital due to respiratory failure and antiviral treatment was initiated and the next day, were transferred to a tertiary care hospital on 16 March and on 18 March, a nasopharyngeal swab was collected, and processed by real-time polymerase chain reaction (RT-PCR).

## Diagnosis:

The result was influenza A, non-subtypable complications. On 1 April, the sample was tested, where the positive result for influenza A(H5N1) was confirmed by RT-PCR and characterized as avian influenza A(H5N1) virus and died on 8 April due to respiratory complications. During contact tracing, 91 individuals were identified, including 21 household contacts, 60 healthcare workers, and 10 individuals from a childcare center. Pharyngeal and nasopharyngeal swab samples collected from 49 contacts tested negative.

## Disease transmission:

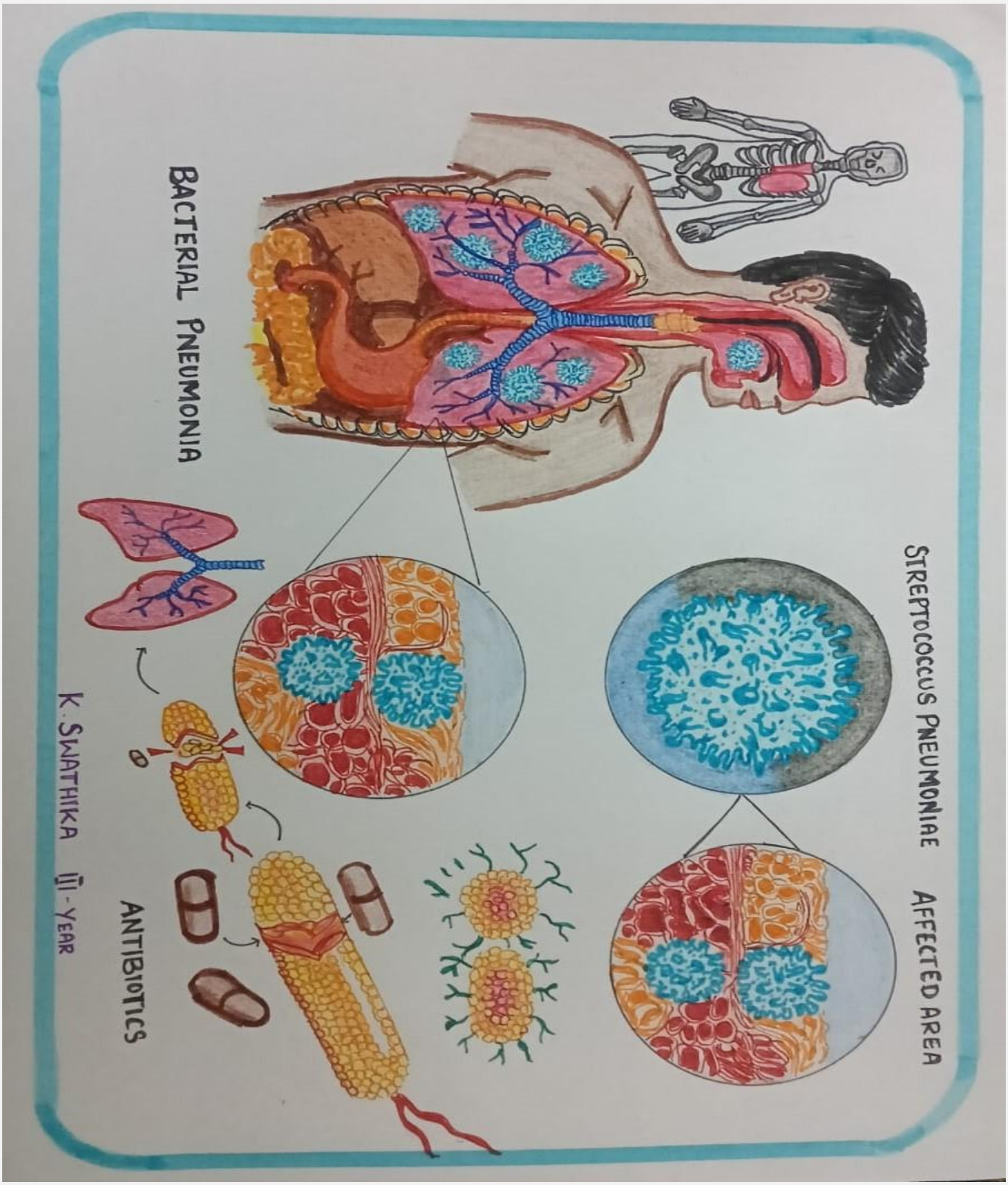
To date, no further cases of human infection with influenza A(H5N1) linked to this case have been identified. Ongoing investigations are focused, On to date, no additional human cases of A(H5N1) virus have been identified in relation to this case, nor have any been detected through routine influenza surveillance. When these virus circulate in poultry populations, there is an inherent risk of human infection through exposure to infected birds or contaminated environments.




SOURCE:www.cdc.gov/flu/avianflu/avian-in-humans.html

## Conclusion:

Based on current information, the WHO assesses the overall public health risk associated with A(H5) viruses as low. There are several vaccines licensed for preventing influenza A(H5) virus infection in humans, although their availability is limited. Candidate vaccine viruses for pandemic preparedness have been selected to protect against A(H5) disease in humans based on circulating strains.

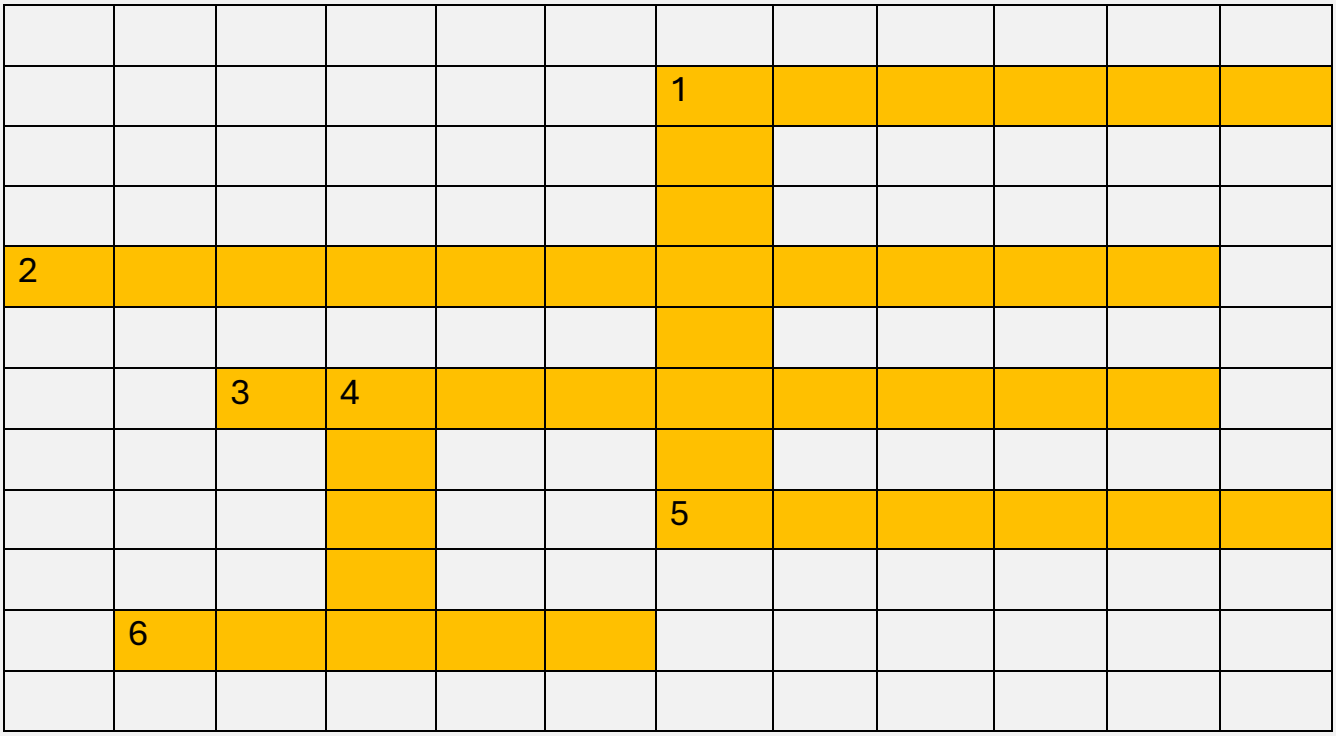



# STREPTOCOCCUS PNEUMONIAE

Swathika K- 3rd year BMS

# CROSS WORD PUZZLE

Nithish N – 1st year BMS




**PUZZLE:**

1) Another name for this is “Voice box “
1) Tissue that holds joint together
2) The process where blood moves throughout the body
3) The layer of skin on the outside of the body
4) The opening in the eye that light passes through
5) The tissue that connects bones with muscle
6) The throbbing of arteries in the body as blood is pumped through them

**Answer :**

1) Larynx
1) Ligament
2) Circulation
3) Epidermis
4) Pupil
5) Tendon
6) Pulse

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# THANK YOU!

Cover art by,

**DENCY EVA -** 3rd year BMS

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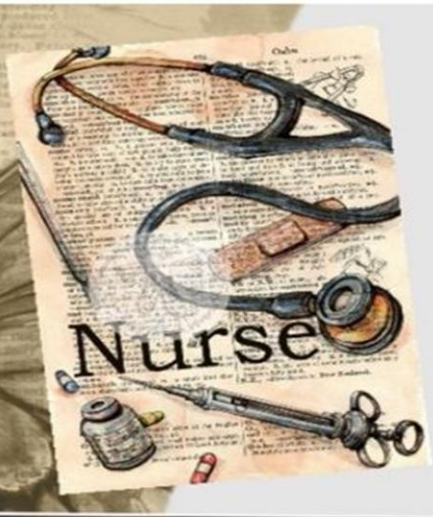
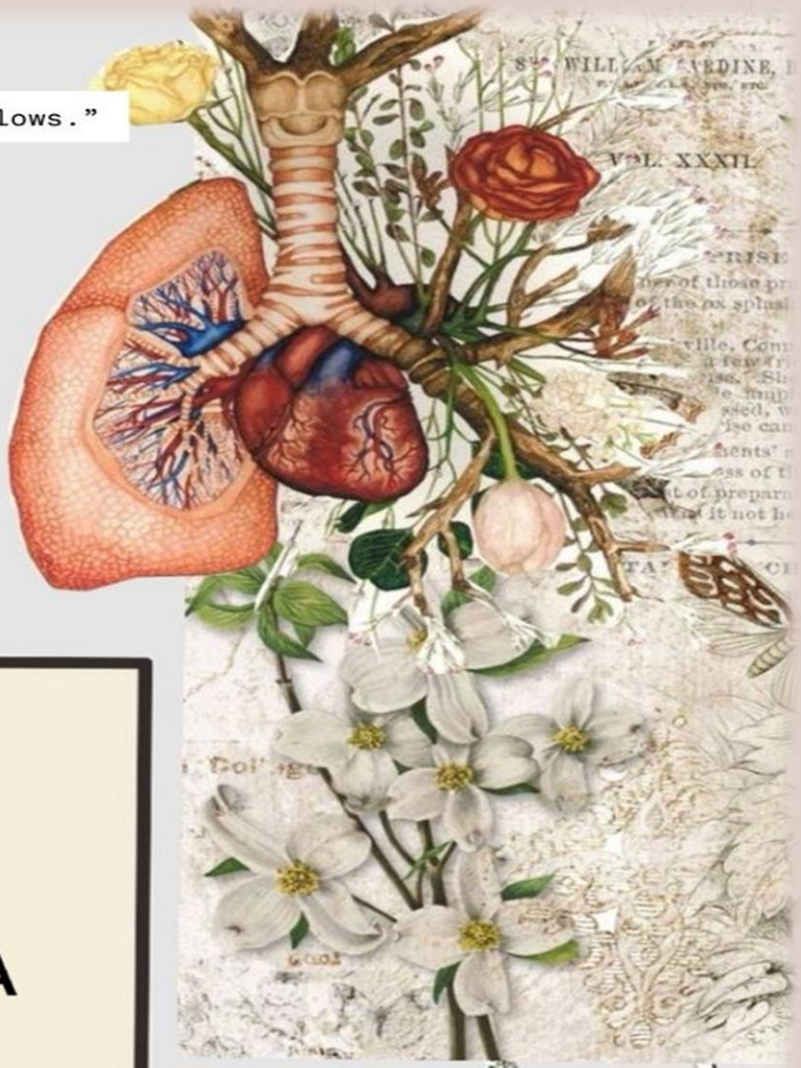
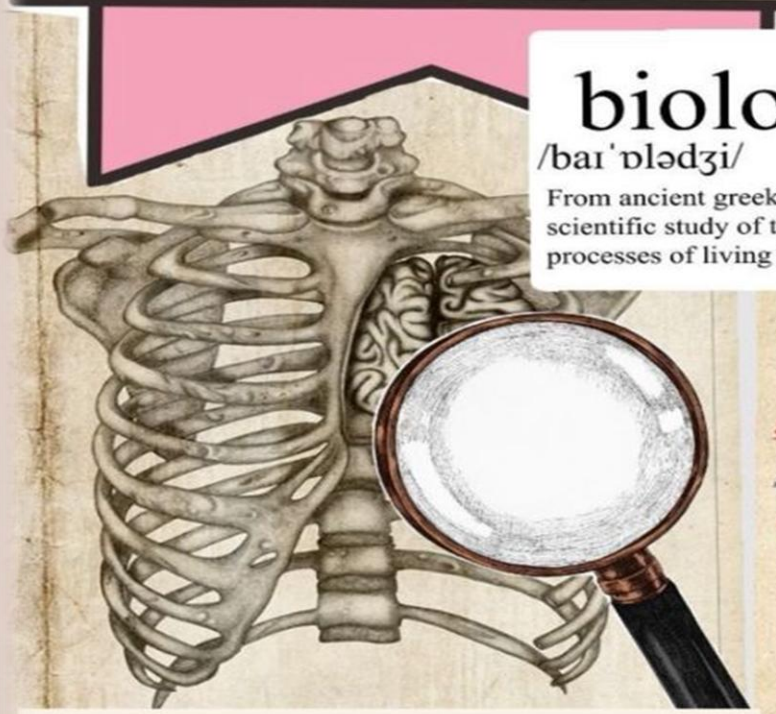
Where curiosity leads, discovery follows.”



# BIOMED FUTURA

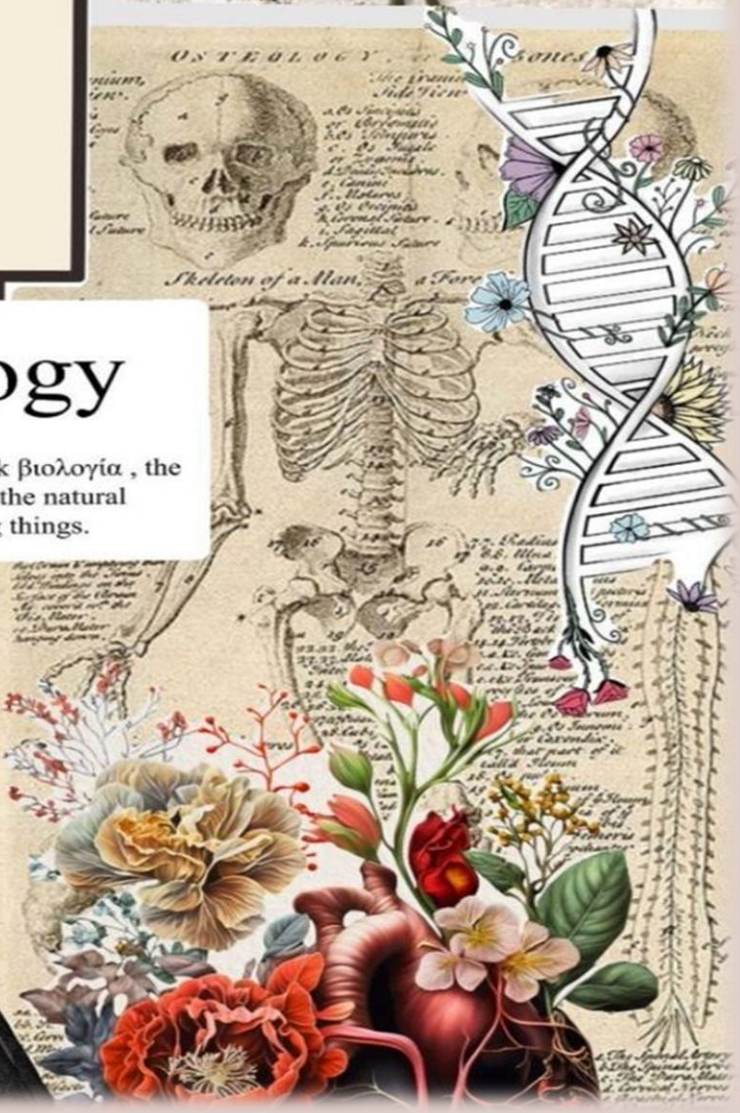
*Decode.*
*Discover.*
*Define the Future.*



## biology

/baɪˈɒlədʒi/

From ancient greek βιολογία , the scientific study of the natural processes of living things.




“ The future is in our genes."

SARÆLAN illustration