ZOOSPECTRA

প্রাণীজগতের বৈচিত্র্য

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DEPARTMENT OF ZOOLOGY

ANANDA MOHAN COLLEGE

KOLKATA, WEST BENGAL

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ANANDA MOHAN COLLEGE

Ananda Mohan College has a rich history of education and social values, with its roots going back to the vision of Ananda Mohan Bose and the Sadharan Brahmo Samaj in 1881. In 1961, it became a separate institution from City College and began its journey as Ananda Mohan College.

Since then, the college has continued to inspire students through quality education, discipline, and progressive values. Today, it proudly combines its glorious heritage with a vibrant academic environment, helping students learn, grow, and prepare for a brighter future.

Message From Principal:

Heartiest congratulations to the Department of Zoology, its revered teachers, non-teaching staff and loving students, of Ananda Mohan College on the launch of your radiant wall magazine, **'ZOOSPECTRA'**.

A Zoology wall magazine is a gateway into the natural world rather than merely a show. May it celebrate the wonderful tapestry of life, start discussions, and stir passions for conservation.

I keenly hope, with every iota of my deepest sense of desire, that your illuminating pages will be filled with scientific knowledge, wonder, and curiosity. Cheers to an enlightening and prosperous voyage ahead!

Dr. Debarshi Bhattacharya

Principal

Ananda Mohan College, Kolkata

Message From IQAC Coordinator:

It gives me immense pleasure to present ZOOSPECTRA, the departmental magazine of the Department of Zoology, Ananda Mohan College. The magazine reflects the academic enthusiasm, creativity and scientific curiosity of our students and teachers.

Zoology is a diverse and fascinating discipline encompassing areas such as cell biology, biochemistry, genetics, molecular biology, physiology, ecology, evolution and biodiversity. ZOOSPECTRA provides a valuable platform for exploring these varied dimensions of biological science and encourages students to develop scientific thinking and effective communication skills.

I congratulate the faculty members and students for their sincere efforts in bringing out this publication. I hope ZOOSPECTRA will continue to inspire young minds to explore, learn and appreciate the wonders of life sciences.

Dr. Attreyee Mukherjee

IQAC Coordinator

Ananda Mohan College, Kolkata

Message From HOD, Department of Zoology:

In the past decade, science and scientists have played an important role in the invention of distinct biological things. The two main factors are observation and analysis. ZOOSPECTRA, published by the Department of Zoology, contains different aspects of zoology, such as biochemistry, immunology, classical zoology, ecology, cell and molecular biology, etc. Each constituent has a specific impact on living organisms. This magazine proceeds to assist as an indispensable casement into the steadfast research and conservation endeavors of our Department of Zoology. The relationship between our life, culture and the environment is greatly described by poet Jibananda Das in his poem "Abar Ashibo Phire"

হয়তো ভোরের কাক হয়ে এই কার্তিকের নবান্নের দেশে
কুয়াশার বুকে ভেসে একদিন আসিব এ কাঁঠাল ছায়ায়।
হয়তো বা হাঁস হব- কিশোরীর- ঘুঙুর রহিবে লাল পায়
সারাদিন কেটে যাবে কলমীর গন্ধভরা জলে ভেসে ভেসে।

Dr. Sanjay Dey

Head Of the Department

Department of Zoology

Ananda Mohan College, Kolkata

Names Of the Current Zoology Faculties:

1. Dr. Goutam Das (ASSOCIATE PROFESSOR)
2. Dr. Ujjal Roy (ASSOCIATE PROFESSOR)
3. Dr. Pallab Ray (ASSISTANT PROFESSOR)
4. Shampa Bag (ASSISTANT PROFESSOR)
5. Dr. Jesmin Mondal (ASSISTANT PROFESSOR)
6. Dr. Sanjay Dey (ASSISTANT PROFESSOR; **Head of the Department**)
7. Anirban Basu (SACT)
8. Dr. Swati Sinha (SACT)

Student's Involvement:

1. Utsov Mondal (Semester 5)

2. Himadri Roy (Semester 7)

3. Biswanath Ghosh (Semester 7)

4. Nishith Naga (Semester 7)

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Immunization for Senior Citizens

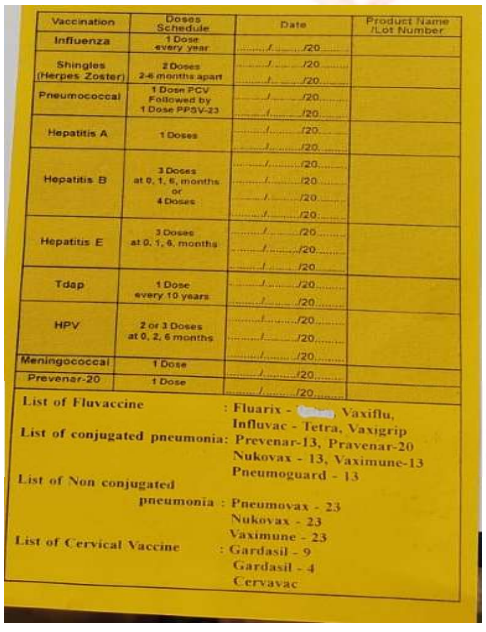
Dr. Goutam Das

Associate Professor
Department of Zoology, Ananda Mohan College Kolkata

Pneumonia and Flu Vaccine:

It is advisable for those above 50years to take pneumonia and flu vaccine as per recommendation by their house physician.

Upstream process (USP) of vaccination is directly proportional with the preservation of vaccine in the cold room not in ordinary refrigerator present in medicine shop and follow the proper immunization schedule which is as follows.



Vaccination	Doses Schedule	Date	Product Name /Lot Number
Influenza	1 Dose every year	...../...../20.....	
Shingles (Herpes Zoster)	2 Doses 2-6 months apart	...../...../20..... ...../...../20.....	
Pneumococcal	1 Dose PCV Followed by 1 Dose PPSV-23	...../...../20..... ...../...../20.....	
Hepatitis A	1 Doses	...../...../20..... ...../...../20.....	
Hepatitis B	3 Doses at 0, 1, 6, months or 4 Doses	...../...../20..... ...../...../20..... ...../...../20..... ...../...../20.....	
Hepatitis E	3 Doses at 0, 1, 6, months	...../...../20..... ...../...../20..... ...../...../20.....	
Tdap	1 Dose every 10 years	...../...../20..... ...../...../20.....	
HPV	2 or 3 Doses at 0, 2, 6 months	...../...../20..... ...../...../20.....	
Meningococcal	1 Dose	...../...../20.....	
Prevenar-20	1 Dose	...../...../20.....	

List of Fluvaccine : Fluarix - [illegible] Vaxiflu, Influvac - Tetra, Vaxigrip

List of conjugated pneumonia: Prevenar-13, Pravenar-20 Nukovax - 13, Vaximune-13 Pneumoguard - 13

List of Non conjugated pneumonia : Pneumovax - 23 Nukovax - 23 Vaximune - 23

List of Cervical Vaccine : Gardasil - 9 Gardasil - 4 Cervavac

Animals That Made History

Dr. Pallab Ray
Assistant Professor
Department of Zoology, Ananda Mohan College Kolkata

Human history is full of kings, generals, inventors, and explorers, but animals have been standing in the background the whole time, pulling sleds, carrying messages, guiding people through danger, and changing what we thought was possible. Some became famous because they saved lives in a single desperate moment. Others mattered because they pushed science, war, medicine, or culture in a new direction. They did not ask to become symbols, but people kept telling their stories because the stories were too useful, moving, or strange to forget. Here are five animal heroes who carved their own place in human history.

1) Laika- On November 3, 1957, Laika (meaning "barker" in Russian) travelled aboard the Soviet spacecraft Sputnik 2, becoming the first animal to orbit Earth and paving the way for human spaceflight. The canine cosmonaut made her historic mission just one month after the launch of the Soviet Union's unmanned artificial satellite, Sputnik 1, which signalled the world's entrance into the space age.

Laika

A small, female mutt Laika was a stray before being captured and trained along with other potential space dogs. Although it was initially claimed Laika survived aboard Sputnik 2 for about a week before perishing, in 2002 it was revealed she died a few hours after blast off due to overheating and stress.

2) Dolly- In 1996 the first clone of an adult mammal was born, a female Finn Dorset sheep named Dolly. British developmental biologist Ian Wilmut and colleagues of the Roslin Institute, near Edinburgh, had created her by using electrical pulses to fuse the mammary cell of a ewe with an unfertilized egg cell, the nucleus of which had been removed. The announcement in 1997 of Dolly's birth marked a milestone in science, dispelling decades of presumption that adult mammals could not be cloned and igniting a debate concerning the many possible uses and misuses of mammalian cloning technology. Dolly remained alive and well long after her birth, with a functional heart, liver, brain, and other organs. In 2003 she was euthanized after being found to suffer from progressive lung disease.

Dolly

3) Martha, passenger pigeon- At the end of the 8th century, perhaps two in every five birds in North America were passenger pigeons. There may have been six billion of them, split between a handful of flocks so vast that they blackened the sky for days as they passed overhead. To hunters, these nomadic birds seemed a limitless resource. Guns, nets, poison and fire were deployed against the pigeons on an industrial scale, precipitating a catastrophic decline in their numbers from 1870. Emergency legislation to protect the species came too late, and captive breeding failed. Martha, the Passenger Pigeon, passed away on September 1, 1914, in the Cincinnati Zoo. She was believed to be the last living individual of her species after two male companions had died in the same zoo in 1910. Martha was a celebrity at the zoo, attracting long lines of visitors. When she was found dead on the floor of her cage that afternoon, she was immediately frozen into a 300-pound block of ice and shipped by fast train to the Smithsonian Institution in Washington, DC, where her body was carefully preserved as a taxidermy mount and an anatomical specimen. Her death helped to create the conservation movement, and she is still a conservation icon today.

Martha, passenger pigeon

4. David Greybeard- Until 1960, scientists believed that only humans could make and use tools. But that changed when Jane Goodall observed a chimpanzee, she named David Greybeard using a grass stalk to extract termites from a mound. Later, she watched him and another chimp make fishing tools by stripping the leaves off twigs. "[David] was the first chimpanzee who let me come close, who lost his fear," Goodall told Bill Moyers in 2011. "He helped introduce me to this magic world out in the forest." His actions helped overturn the long-held belief that only humans were capable of using tools, making him one of the most famous animals in the field of behavioural science. David, in addition to all that he taught Dr. Goodall about primate behaviour, also helped her by bringing other chimps with him when he visited her camp. Without David's helpful introductions, Jane may not have been able to meet the other Gombe chimps. Jane believes that David Greybeard died during a pneumonia epidemic in 1968. Time Magazine named David one of the 15 most influential animals that ever lived.

David Greybeard

5) Elsa The Lioness- George Adamson lived and worked in Kenya as a game warden. In 1956, together with his wife Joy, they adopted a lion cub who they called Elsa. While Elsa lived in many ways like a domesticated pet when she was small, Joy Adamson, whom Elsa trusted the most, considered her relationship with Elsa to be that of equals. Indeed, after sending the other two to a zoo, Joy was fiercely determined to give Elsa the education she needed to hunt and live in the wild. Her efforts paid off, earning Elsa worldwide fame at the time, when her early life's story was published in the book Born Free. The life of Elsa and her cubs is covered in the book Living Free, published not long afterwards.

Elsa the Lioness

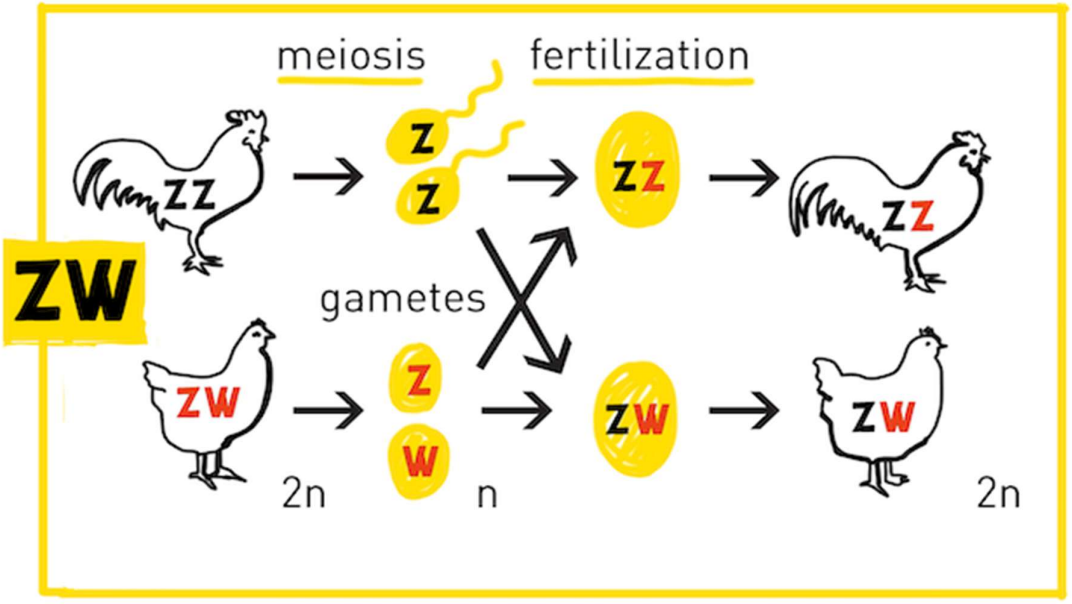
ZW- ZZ method of bird sex determination

Dr. Sanjay Dey

Assistant Professor, Head of the Department,

Department of Zoology, Ananda Mohan College, Kolkata

Sex determination in bird is responsible for the two-chromosome Z and W. Insects involving lepidoptera (butterfly) also exhibit this ZW- ZZ sex chromosome. In male bird, two homologous sex chromosomes of Z are present (ZZ). In case of male bird, 100% male sperm have one Z chromosome. So, the male bird is homogametic. In female bird, two different sex chromosomes are present one is Z and another is W (ZW). Female bird produces two ovum, one is Z and another is W. In general, W chromosome contains 50% gynoovum and Z chromosome contains 50% Andro ovum. So female bird is heterogametic. Double sex and mab 3 related transcription factor (DMRT1) is present on Z chromosome that is responsible for male bird. DMRT1 protein reduction hampers genetically male embryo (ZZ). Partial sex reversal is observed in affected male. In the W chromosome, SPIN-W, ASW, ATP5A1-W, EAVH 2 , CHD-W are female specific genes. FET1 and ASW genes present on the W chromosome are responsible for female bird.



In Vivo Animal Models: Unlocking the Secrets of Life

Dr. Swati Sinha

State Aided College Teacher

Department of Zoology, Ananda Mohan College Kolkata

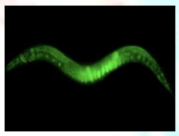
From tiny insects to aquatic and fossorial creatures, animals have been silent partners in scientific discovery. Animal models are vital for researchers to understand biological processes, disease, development, evolution and responses to potential treatments because many fundamental genes and cellular pathways are conserved across evolution. Fruit Fly, Nematode Worm, Zebrafish, African Clawed Frog and Laboratory Mouse are particularly valuable because they combine genetic tractability with different levels of physiological complexity. They act as living laboratories, bridging the gap between basic research and real-world applications in biotechnology, gene therapy, targeted drug delivery, agriculture, biomedical innovations, especially, in the field of oncology and even in space-biology research.

Table 1. The comparative table provides a concise overview of the five important animal models.

Animal models	Key features	Approx. genetic similarity with humans
Drosophila melanogaster **(Fruit fly)**	An insect with very short generation time; high reproduction; powerful genetic tool; **"Cinderella of Genetics."**	~**75%** of human disease-associated genes have functional counterparts.
Caenorhabditis elegans **(nematode worm)**	Transparent, 1 mm body; **non-parasitic**, rapid life cycle; easy to culture; exactly 302 neurons in the adult hermaphrodite; powerful RNAi/genetic tool.	~**40%** to ~**65%** of C. elegans genes have human orthologues; first animal whose whole genome was sequenced.
Danio rerio **(Zebrafish)**	Small, transparent embryos; rapid development; high fertility; organs and physiological systems comparable with vertebrates; amenable to CRISPR and transgenesis.	~**70%** of human genes have at least one zebrafish orthologue; about **82%** of human disease-associated genes have zebrafish orthologues

***Xenopus laevis* (African clawed frog)**	Amphibian model, large eggs and embryos; excellent for studying embryonic development, organ formation and cell signaling; CRISPR and other genetic tools available.	About **79%** of identified human disease genes have *Xenopus* counterparts.
***Mus musculus* (Laboratory mouse)**	**"Gold standard" Mammalian model**; complex organs and immune system; transgenic and knockout strains; strong physiological similarity to humans.	Mouse and human protein-coding regions are **~85%** identical on average, while **~99%** of human genes have a mouse counterpart.

Table 2. A comparison table, complemented by images, highlights the implications in Biotechnology

Animal models	Images	Implications in Biotechnology & Medicine
***Drosophila melanogaster* (Fruit fly)**	1. 	Genetic screening, developmental biology, cancer and neurological-disease studies, toxicity testing and drug screening. Its genetic tractability makes it useful for identifying drug targets and disease pathways.
***Caenorhabditis elegans* (Nematode worm)**	2. 	Excellent for gene-function analysis, RNAi screening, ageing, neurobiology, toxicology and high-throughput drug screening. Its transparency permits observation of targeted drug delivery in nano-biotechnology.
***Danio rerio* (Zebrafish)**	3. 	Used for disease modeling, developmental biology, toxicity testing, regenerative medicine and high-throughput drug screening.
***Xenopus laevis* (African clawed frog)**	4. 	Useful for developmental biology, gene-function studies, human disease modeling, regenerative biology and testing genetic variants. Large embryos make experimental manipulation and imaging comparatively convenient.
***Mus musculus* (Laboratory mouse)**	5. 	Central to medical research, cancer, immunology, infectious disease, cardiovascular research, drug development, toxicology and gene therapy. It permits testing of therapies in a whole mammalian system before clinical studies. COVID-19 vaccines were tested on it by Pfizer, Moderna, AstraZeneca before human clinical trials.

Picture courtesy: 1. https://images.indianexpress.com/2017/10/fruit-fly.jpg. 2. https://wi.mit.edu/unusual-labmates-how-c-elegans-wormed-its-way-science-stardom 3.https://cdn1.byjus.com/wp-content/uploads/2021/06/Zebra-Fish-Danio-Zebrafish-

700x467.jpg 4.https://riverparkaquatics.co.uk/african-clawed-frog-xenopus-laevis/
5..https://scihigh.ca/wpcontent/uploads/2017/05/shutterstock_85771906.jpg

All these five models were sent to space by USA, Russia and China to investigate the effects of microgravity, effect of radiation, bone, muscle, immunity and metabolic changes, ageing and spaceflight stresses on astronaut health. Research using these five models has contributed to dozens of Nobel Prize-winning discoveries in diverse fields. The five animal models provide complementary platforms for modern biotechnology and biomedical research. *Drosophila* and *C. elegans* enable rapid genetic and drug studies, while Zebrafish and *Xenopus* support vertebrate development and disease research. *Mus musculus* closely models human physiology. Together, these models accelerate discoveries and support safer, more effective medical applications.

Role of H^+ and Na^+ in Chemiosmotic system in bioenergetics of intracellular respiration:

Utsov Mondal

Semester 5

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Picture your cells as billions of tiny power stations, each running a battery made not of electrons but of ions. Let that charge flow back through the right turbine, and out comes ATP — the currency that pays for everything a cell does. This is the chemiosmotic story, one of biology's most elegant plot twists [1], [2].

1. The Heretic Who Was Right

For years biochemists searched for a chemical middleman linking respiration to ATP synthesis. It never turned up — because it doesn't exist. In 1961, Peter Mitchell proposed instead that energy is stored as a proton gradient across a membrane [1]. Electron transport complexes I, III and IV pump H^+ from the mitochondrial matrix into the intermembrane space, building the proton-motive force ($\Delta p = \Delta\Psi + \Delta pH$) [2], [3]. ATP synthase is the only gate built to let H^+ back through, and every proton that passes spins its rotor a little further, forging ATP from ADP and phosphate. The theory was dismissed for nearly a decade before Mitchell won the 1978 Nobel Prize in Chemistry for it [4].

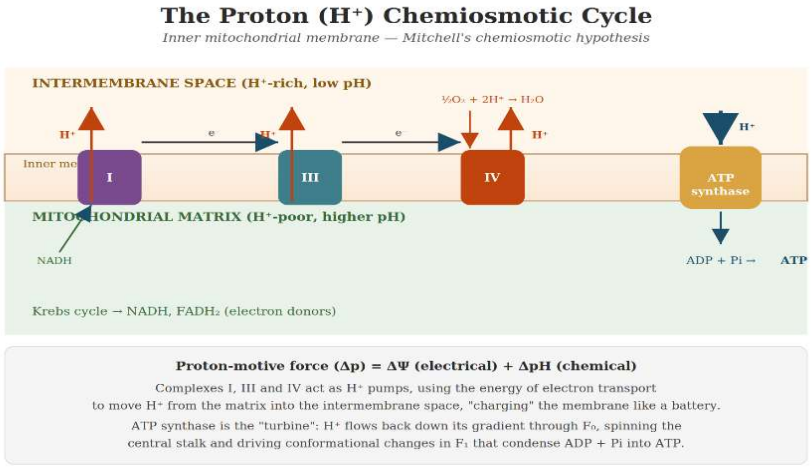

Figure 1. The proton (H^+) chemiosmotic cycle in the inner mitochondrial membrane [1,2]

2. Meet the Understudy: The Sodium Cycle

Mitchell's proton circuit is not the only ionic economy in the living world. In the 1980s, Vladimir Skulachev, and separately Hajime Tokuda and Tsutomu Unemoto studying the marine bacterium *Vibrio alginolyticus*, found organisms running the same circuit with sodium instead of hydrogen [5], [7]. The pump, Na^+-NQR, oxidises NADH to expel Na^+, building a sodium-motive force ($\Delta\tilde{p}Na = \Delta\Psi + \Delta pNa$) that spins Na^+-driven ATP synthases, powers flagellar motors, and drives Na^+/solute symporters [6]. Marine and alkaliphilic bacteria favour Na^+ because, where external pH is high or protons are scarce, a sodium battery is cheaper to maintain than a proton one [8]. This also matters medically: *Vibrio cholerae* relies on Na^+-NQR as its main respiratory entry point, making the enzyme a target for new antibiotics [6].

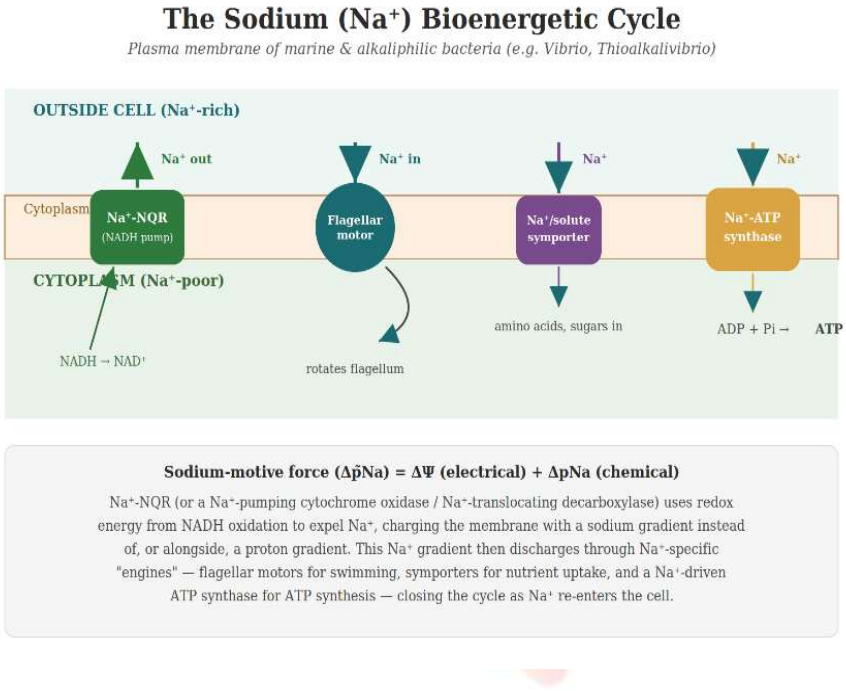

Figure 2. The sodium (Na^+) bioenergetic cycle in marine bacteria [5,7].

3. One Blueprint, Two Currencies

Strip away the ion label and both systems share the same three parts: a pump that spends redox energy to charge the membrane, a gradient that stores it, and an engine that spends it. Mitchell called this a proton circuit [2]; Skulachev extended the same logic to sodium, and some organisms even run mixed economies, interconverting the two gradients with Na^+/H^+ antiporters [7].

Feature	H^+ System	Na^+ System
Pumps	Complex I, III, IV	Na^+-NQR, Na^+-cytochrome oxidase
Engine	F_1F_o-ATP synthase	Na^+-ATP synthase, flagellar motor
Habitat	Mitochondria, most bacteria	Marine/alkaliphilic bacteria, pathogens
Key source	Mitchell, 1961/1966	Skulachev; Tokuda & Unemoto

4. Why This Still Matters

- Medicine: targeting Na^+-NQR could fight cholera without harming human (H^+-based) mitochondria [6].
- Evolution: a parallel sodium circuit hints that chemiosmotic coupling itself, not any one ion, is the ancient universal trick [7].
- Everyday physiology: every mitochondrion in your body is running Mitchell's proton circuit right now, turning food into the ATP that powers thought and movement [3].

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Mutation of Caspase-8 Causes Pleiotropic Defects in Human Lymphocytes

Disha Adhikari

Semester 5

Department of Zoology

Ananda Mohan College

1. Introduction

Apoptosis, controlled by a family of cysteine proteases called caspases, keeps lymphocyte numbers in check and prevents autoimmunity [1,2]. Caspase-8 is unusual among caspases because it is not only the initiator of death-receptor-mediated apoptosis but is also required for normal lymphocyte activation and proliferation [2,3]. Consequently, human mutations that inactivate caspase-8 do not simply block cell death — they cause a broad, *pleiotropic*, pattern of immune dysfunction. This review summarises the molecular role of caspase-8 and the lymphocyte defects seen in caspase-8 deficiency.

2. Structure and Normal Function of Caspase-8

Caspase-8 contains two death effector domains (DEDs) and a catalytic protease domain. Via the adaptor FADD, it is recruited to death receptors such as CD95 (Fas), where receptor clustering triggers its autocatalytic activation and formation of the death-inducing signalling complex (DISC) [1,4]. Active caspase-8 then cleaves downstream effector caspases to execute apoptosis. Complete caspase-8 loss is embryonically lethal in mice, reflecting additional roles in suppressing necroptosis, showing that this protein is essential well beyond apoptosis alone [5].

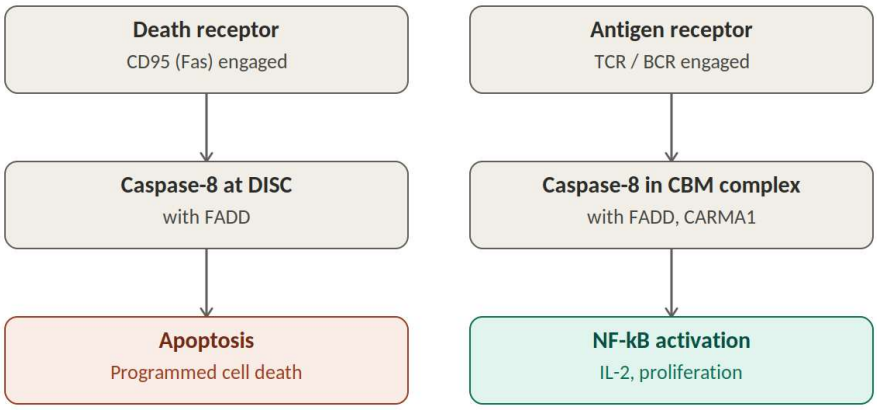

Figure 1. Caspase-8 acts in the DISC after death-receptor engagement (apoptosis) and in the CBM complex after antigen-receptor engagement (NF-kB activation and proliferation).

3. Discovery of Human Caspase-8 Deficiency

In 2002, Chun et al. described two siblings with lymphadenopathy, splenomegaly, and recurrent sinopulmonary and herpes simplex infections who carried a homozygous *CASP8* mutation that destabilised the protein and abolished its enzymatic activity [1]. Unlike classical autoimmune lymphoproliferative syndrome (ALPS), caused by heterozygous CD95/CD95L/caspase-10 mutations, these patients were clinically immunodeficient rather than autoimmune [1,6], defining a distinct entity now called caspase-8 deficiency state (CEDS) [7].

4. Pleiotropic Defects in Lymphocytes

Caspase-8 mutation produces defects across every major lymphocyte lineage:

• **Apoptosis:** Lymphocytes resist Fas-induced apoptosis, causing mild lymphadenopathy and splenomegaly [1,4].

• **T cells:** Proliferation and cytotoxic responses to antigen and mitogen stimulation are markedly reduced, despite normal T-cell numbers; this was confirmed by RNAi knock-down and rescue experiments and in T-cell-specific caspase-8-deficient mice [1,3].

• **B cells:** Activation and immunoglobulin production after stimulation are impaired, contributing to poor vaccine responses [1,7].

• **NK cells:** Cytotoxic activation is diminished, weakening early antiviral defence [1].

Mechanistically, caspase-8 also acts downstream of the antigen receptor to help activate NF-κB and drive IL-2 gene expression, linking it directly to lymphocyte proliferation rather than only to cell death [6,8]. Barnhart and Peter described this as the "two faces of caspase-8" — a death-inducing face at the DISC and an activation-promoting face in stimulated lymphocytes [2].

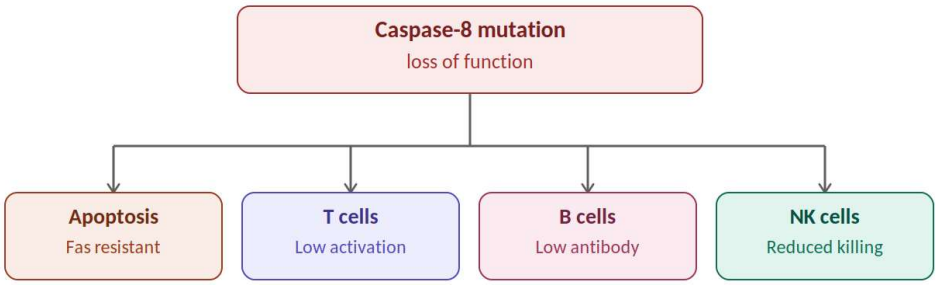

Figure 2. Loss-of-function caspase-8 mutation produces defects across apoptosis, T-cell, B-cell, and NK-cell function simultaneously.

5. Clinical Picture and Comparison with ALPS

Patients with CEDS present with lymphadenopathy/splenomegaly, recurrent bacterial and viral infections, and poor immunisation responses, reflecting combined apoptosis and activation defects [1,7,9]. This distinguishes CEDS from classical ALPS, where apoptosis alone is impaired while activation remains intact, producing autoimmunity rather than immunodeficiency [1,6].

6. Conclusion

Human caspase-8 mutations show that a single enzyme, once thought to function purely in apoptosis, is also essential for T-, B-, and NK-cell activation [1]. Loss of caspase-8 therefore causes a unique combined phenotype of mild lymphoproliferation and immunodeficiency (CEDS), highlighting how apoptotic and immune-activation pathways are molecularly intertwined [7,9].

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Molecular Genetics of Bacteria

Sweety k Mondal

Sem-5

Department of Zoology, Ananda Mohan College Kolkata

1. Genetic Material

- The main genetic material of bacteria is DNA.

- Bacterial DNA is usually a single circular chromosome.

- It is present in a region called the nucleoid.

- Bacteria may also have small circular DNA called plasmids.

- Plasmids can carry useful genes, such as genes related to antibiotic resistance.

2. DNA Replication

DNA replication means making a copy of DNA.

Before a bacterial cell divides, its DNA is copied so that each new cell receives a copy of the genetic information.

3. Gene Expression

Gene expression means using the information stored in DNA to make proteins.

It mainly occurs in two steps:

DNA → RNA → Protein

- Transcription: DNA information is copied into RNA.

- Translation: RNA information is used to make a protein.

4. Operon

An operon is a group of genes that are controlled together.

Two important examples are:

- Lac operon: helps bacteria use lactose as a food source.

- Trp operon: controls the production of tryptophan.

5. Mutation

A mutation is a change in the DNA.

Mutation can produce new characteristics in bacteria.

6. Transfer of Genetic Material

Bacteria can get DNA from other bacteria in three main ways:

Transformation: Bacteria take up free DNA from their surroundings.

Transduction: DNA is transferred from one bacterium to another by a virus called a bacteriophage.

Conjugation: DNA is transferred directly from one bacterial cell to another through cell-to-cell contact.

Importance

Molecular genetics helps us understand:

- How bacteria grow and function

- How bacteria develop new characteristics

- Antibiotic resistance

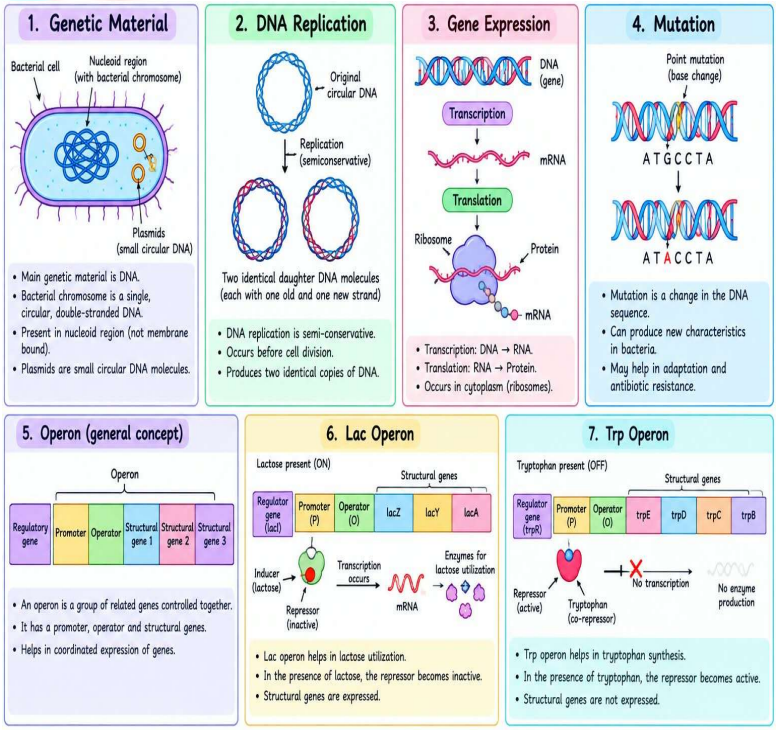
- Gene regulation

- Genetic engineering

- Bacterial diseases

In short

Molecular genetics of bacteria is the study of bacterial DNA, how DNA makes proteins, how genes are controlled, and how genetic information can change or move between bacteria.



METAGENOMICS APPROACH IN SYSTEMS MICROBIOLOGY

Sabahat Farheen
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• **Introduction to Metagenomics in Systems Microbiology: -**

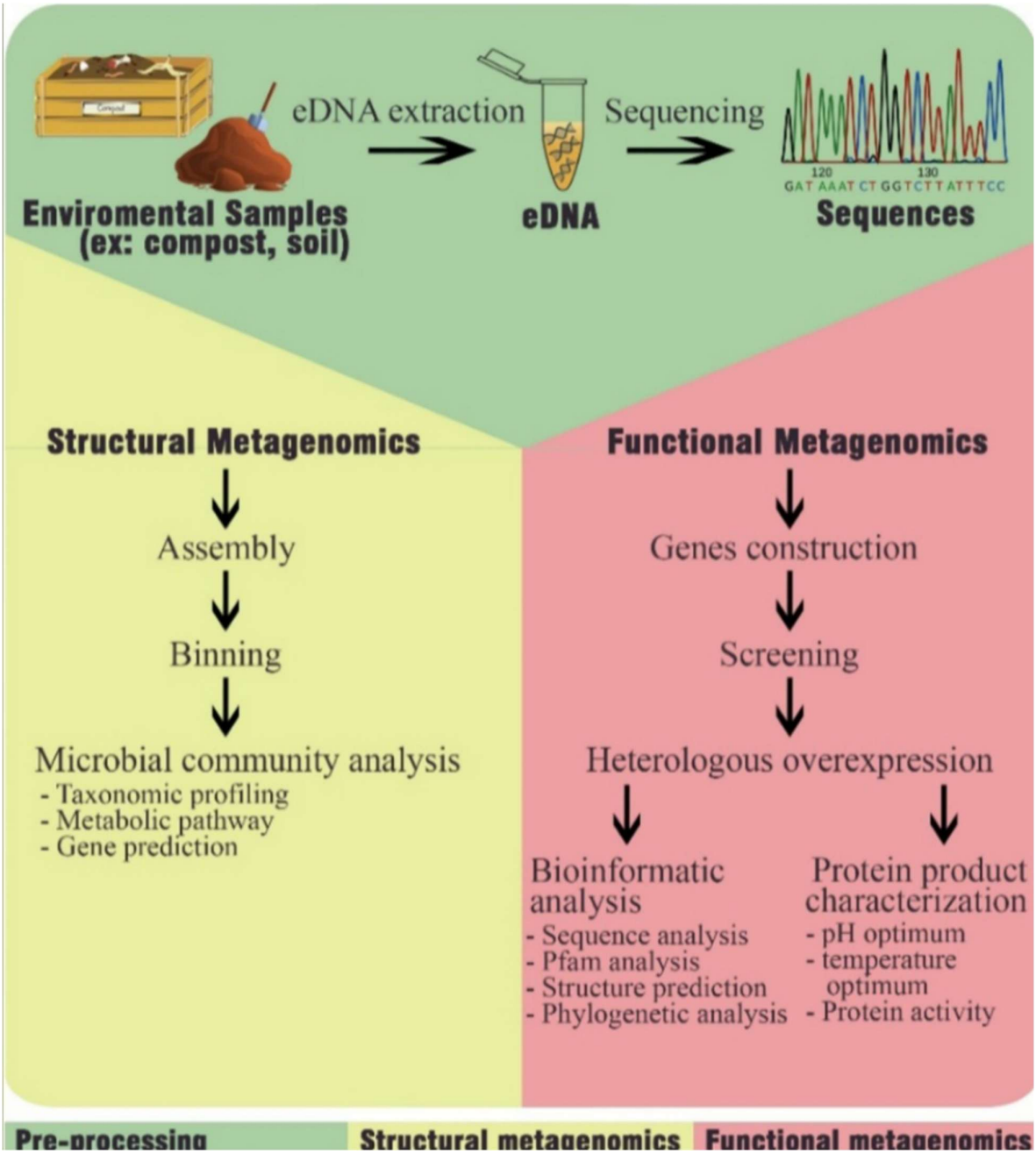
Systems microbiology aims to understand the complex interactions within microbial communities and their functional roles in various ecosystems. Traditional microbiological methods rely heavily on culturing microorganisms in laboratory media; however, over 99% of environmental and host-associated microbes cannot be easily cultured using standard techniques. Metagenomics overcomes this fundamental limitation by directly analyzing total genomic DNA extracted directly from environmental or host biological samples.

• **Metagenomics Workflow & Sequencing Approaches:-**

The core pipeline of metagenomics involves environmental DNA (eDNA) extraction, followed by high-throughput sequencing and advanced bioinformatic processing to reconstruct community profiles and genomic functional pathways.

• **Targeted (Amplicon) Sequencing:** Focuses on specific marker genes (such as 16S rRNA for bacteria or ITS for fungi) to profile microbial taxonomic diversity and composition across samples.

• **Shotgun Metagenomics:** Sequences all accessible DNA fragments randomly, providing a comprehensive genomic blueprint to reveal both taxonomic identity (*who is there*) and functional metabolic potential (*what they can do*).



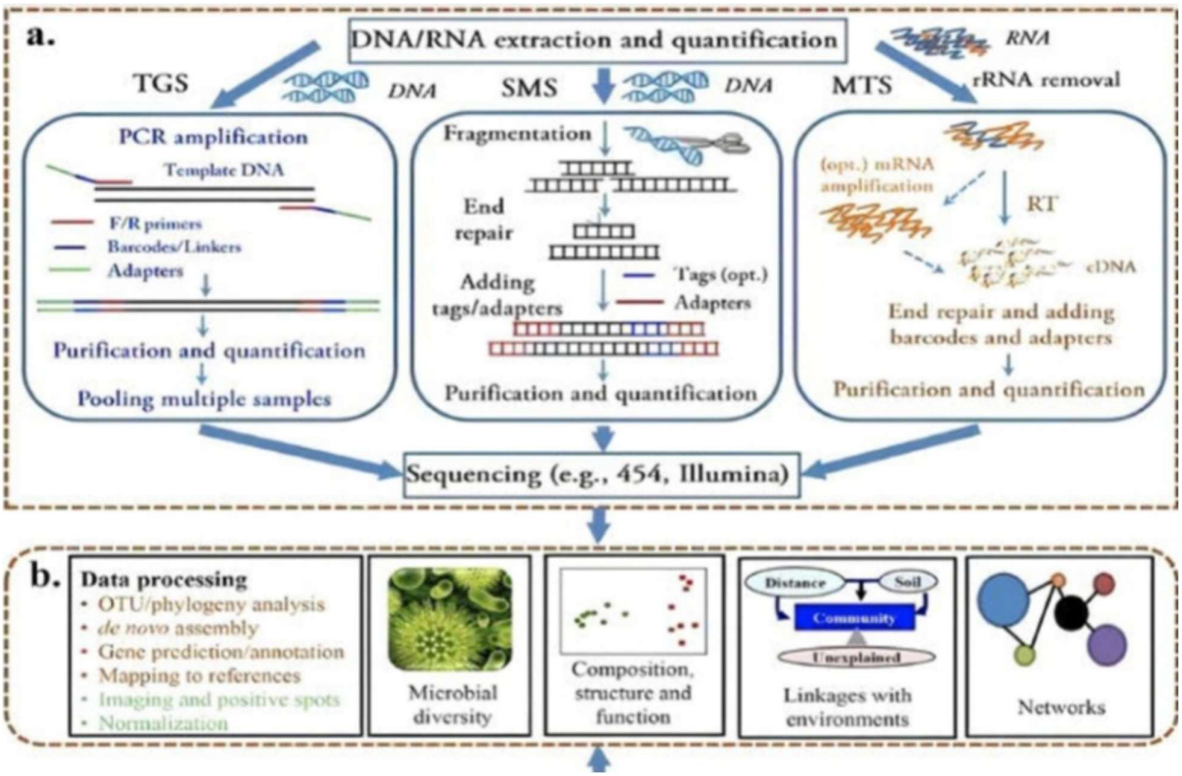


Figure 1: High-throughput Metagenomic Extraction, Sequencing & Data Processing Workflow.

• **Structural vs. Functional Metagenomics:-**

Metagenomics approaches in systems microbiology are broadly categorized into structural and functional strategies based on the primary analytical objective:

• **Structural Metagenomics:** Focuses on sequence assembly, binning into Metagenome-Assembled
Genomes (MAGs), and bioinformatic mapping to characterize community composition, phylogenetic
structures, and evolutionary relationships.

• **Functional Metagenomics:** Involves cloning eDNA into expression vectors, constructing gene
libraries, host screening, and heterologous overexpression to discover novel bioactive compounds,
enzymes, and metabolic activities without prior sequence knowledge.

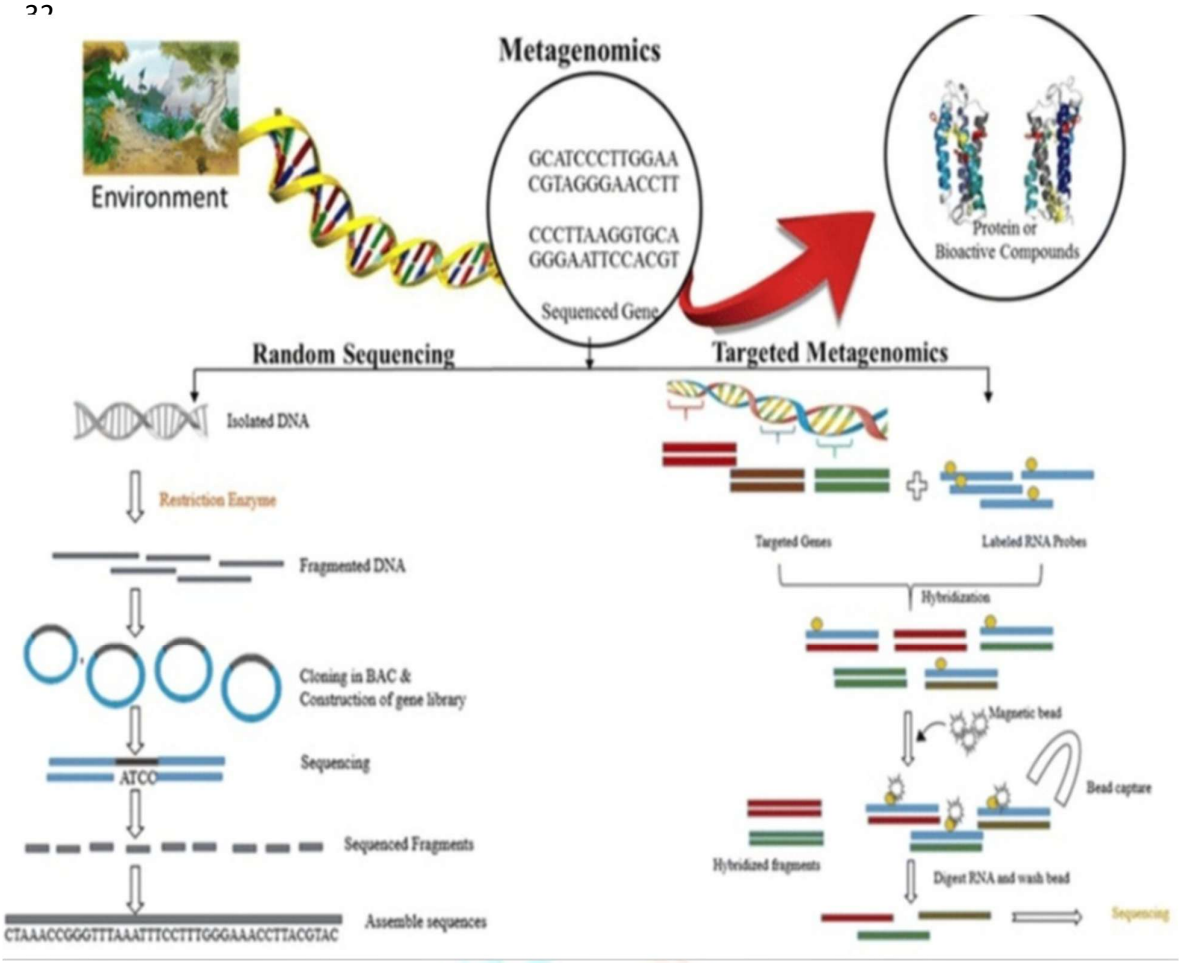


Figure 3: Random Sequencing vs Targeted Metagenomics for Bioactive Discovery.

• **Systems-Level Integration and Applications in Zoology:-**

In systems microbiology, metagenomic datasets are integrated with other omics approaches (metatranscriptomics, metaproteomics, and metabolomics) to construct systemic network models of host-microbiome interactions:

• **Microbial Community Networks:** Maps complex host-microbe and microbe-microbe metabolic interaction networks and cross-feeding dynamics.

• **Host Health & Physiological Impact:** Evaluates the physiological roles of animal-associated microbiomes in digestive efficiency, metabolic synthesis, immune system modulation, and disease resistance.

Biotechnological prospect of metagenomics

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Metagenomics is the culture-independent genomic analysis of microbial communities.

The term "metagenomics" was coined in the late 1990s, as researchers began applying genomic sequencing methods directly to microbial communities in environmental samples. The term derived from the statistically combining separate analysis) and genomics (the comprehensive analysis of an organism's genetic material).

Metagenomics can be used to study microorganisms that live in the natural environment but cannot be grown in the laboratory. Metagenomics helps scientists to study their DNA directly from the environmental samples. In some environment these difficult to grow microorganisms create more than 99% of microbial population.

A

B

Metagenomics uses modern techniques in microbial genomics, polymerase chain reaction (PCR) amplification and cloning of genes that share sequence similarity.

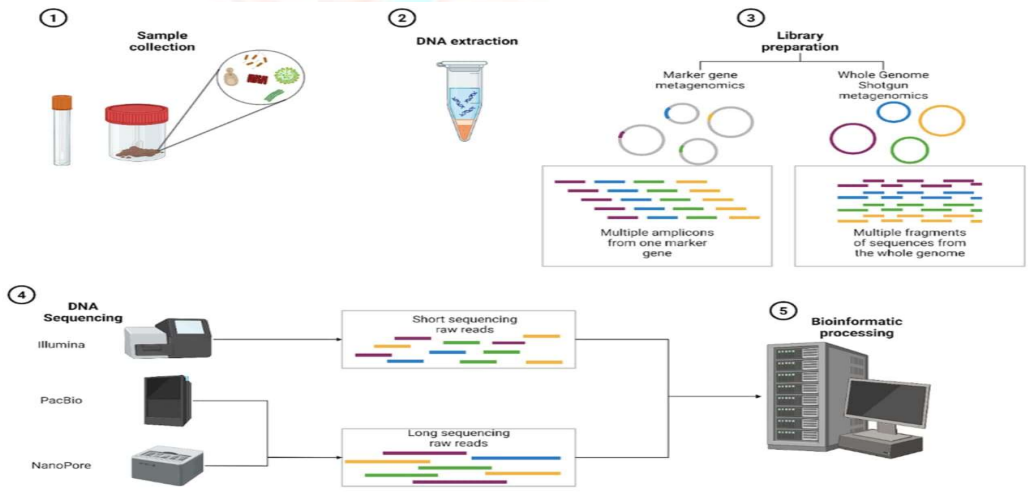
Direct extraction and cloning of DNA can help researchers to find genes even if their sequences or proteins are unknown, whereas PCR amplification requires prior knowledge of the sequence of the gene to design primers for amplification.

Metagenomics can help in biotechnology through both basic research and the search for useful biological product to discover new antibiotics, industrial enzymes and other useful substances.

Two approaches, the function-driven analysis and the sequence-driven analysis, have emerged to extract biotechnological information from metagenomic libraries.

- **Malacidins** and **turbomycin** are notable examples of **antibiotics** discovered through the metagenomics analysis of environmental microbial communities.
- Streptomycin can be mentioned as an example when discussing the broader biotechnological potential of **microbial metagenomics**. But, streptomycin itself was discovered before modern metagenomics, from the cultured soil bacterium ***Streptomyces griseus***.
- Metagenomics helps discover **industrially useful enzymes** from microorganisms that are difficult to culture. Examples include amylase for starch processing, protease for detergents.
- Isolate microbial genes capable of breakdown stubborn pollutants like plastics, pesticides, oils and hydrocarbons. **Alkane Monooxygenase (al kB)** encodes an enzyme that starts the breakdown of medium to long – chain alkanes found in oil and petroleum spills.

Metagenomic workflow –



The first process is **sample collection**, where microbial specimen are gathered, followed by **DNA extraction** to isolate their genetic material.

In the library preparation, researcher choose between marker gene metagenomics to target a specific gene or whole genome shotgun metagenomics to fragment entire genomes.

In the DNA sequencing step, where technologies like **Illumina** generate short raw reads, while **PacBio** and **Nanopore** deliver long raw reads.

Finally, the workflow concludes with bioinformatic processing, where powerful computational systems assemble and analyse the resulting data to identify and understand the microbial community.

Metagenomics gives us a chance to explore the huge microbial world that we cannot see or easily grow in the lab. It can help us find new antibiotics, enzymes, useful genes and other valuable products. It contributes to medical, agriculture, industry and environmental field.

Bengal's Wildlife: A Heritage at Risk

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When we think of West Bengal, we often think of the high Himalayan peaks in the north, through lush green river plains in the middle and down to the tidal mangrove forests of the Sundarbans in the South. But alongside these landscapes, animals have always been an important part of Bengal's story. From the Himalayan mountains in north, where animals such as the red panda are found, to the Sundarbans in the south, home of the famous Royal Bengal Tiger. For generations, people and animals have shaped the forests, rivers and villages of West Bengal.

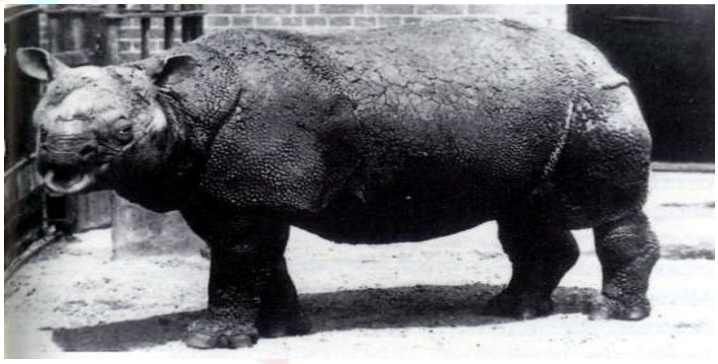
Not only Bengal's story, but they are also an important part of Bengal's culture, traditions, religion and daily life. They represent nature, faith and the identity of Bengal. The Royal Bengal Tiger is perhaps the most famous example. It is not simply an animal found in the Sundarbans; it has become a symbol of Bengal itself. It is also the national animal of India. Elephants, deer, crocodiles, birds and countless other animals have also been closely connected with the natural life of the state.

Many animals that were once seen in different parts of Bengal have now become rare or have disappeared because of hunting, habitat loss and changes in the environment.

The Pink-headed duck -

The Pink-headed duck (*Rhodonessa caryophyllacea*) was once found in the wetlands of India, Bangladesh and Myanmar. Now, the Pink-headed duck is officially listed as Critically Endangered on the IUCN Red List, but experts fear it is actually extinct in the wild because there have been no confirmed sightings since the mid-20th century. Pink-headed ducks were found historically across the Ganges and Brahmaputra River plains in India, Bangladesh, Nepal rand the swamps of Myanmar. They preferred small ponds surrounded by dense bushes and high grass, occasionally moving to larger rivers and lagoons during colder winter month. The primary drivers behind the extinction of the Pink-headed duck were extensive habitat destruction and intense hunting.

The Javan Rhinoceros - The Javan Rhinoceros (*Rhinoceros sondaicus*) is one of the world's most critically endangered large mammals, currently hovering on the very brink of extinction. The Javan rhinoceros historically inhabited a vast range of tropical woodland forests, dense rainforests, marshlands and mud-filled river deltas across South and Southeast Asia. Historically, this species shared a notable geographic and ecological relationship with the Bengal region (modern-day Bangladesh and West Bengal, India).

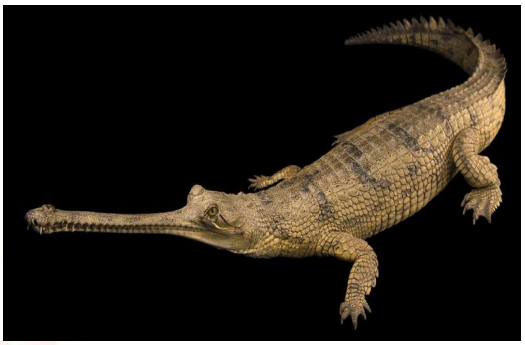


The entire species is critically endangered and restricted to a single population in Indonesia's Ujung Kulon National Park. The primary reasons for the Javan rhino's catastrophic decline and near-extinction across its former range include relentless trophy hunting and poaching for its highly prized horn.

The Bengal florican -The Bengal florican (*Houbaropsis bengalensis*) is a rare grassland bird listed as Critically Endangered on the IUCN Red List, due to a rapidly shrinking population of fewer than 1000 to 1500 mature individuals globally. I t inhabits dense, lowland riverine and alluvial grasslands, particularly in subtropical flood plains and seasonally inundated plains that are 50 to 100 cm high. The Bengal florican is facing extinction primarily due to rapid habitat loss and fragmentation, alongside human-induced pressure.

The Gharial

The gharial (*Gavialis gangeticus*) is a critically endangered crocodilian species in West Bengal, facing a severe risk of extinction. They are fish-eating crocodiles, and their wild populations have crashed due to sand mining, river damming and fishing nets.



The Wild Water Buffalo -

The wild Water Buffalo (*Bubalus arnee*) is locally extirpated (regionally extinct) in West Bengal, mirroring its disappearance from much of its historical Indian range.

Historically found in parts of the northern Duars contiguous with Bhutan and Assam, true wild populations are functionally absent or non – viable. Habitat loss of alluvial grasslands, conversion of wetlands and genetic swamping through interbreeding with domestic and feral livestock.

Role of FOX Gene in Human Hind Gut Development

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What are FOX gene family?

The Fork head box (Fox) family of transcription factors, which originated in unicellular eukaryotes, has expanded over time through multiple duplication events, and sometimes through gene loss, to over 40 members in mammals. Fox genes have evolved to acquire a specialized function in many key biological processes. Mutations in Fox genes have a profound effect on human disease, causing phenotypes as varied as cancer, glaucoma and language disorders.

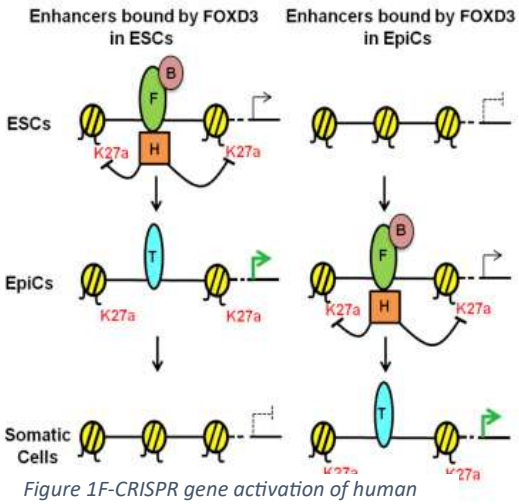
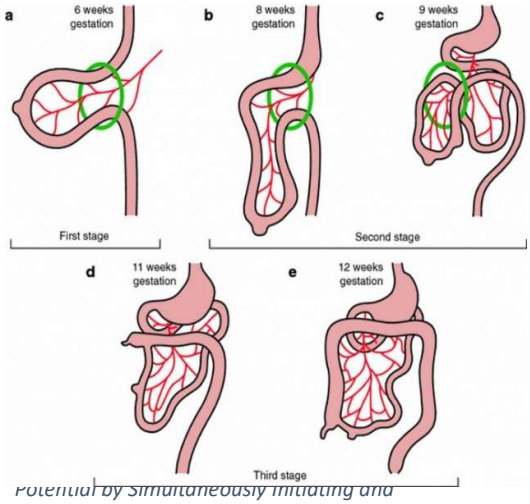


Figure 1F-CRISPR gene activation of human FOXD4

Family of FOX gene that play a vital role in human hindgut development:

Human FOXD3 and FOXD4 is an important member of the FOX gene family and is located on 1p31.3 and 9p24.3. It has been shown that, acting as an important transcription factor at the embryonic stage, FOXD3/FOXD4 is indispensable for embryonic development and maintenance of embryonic stem cells in mammals.3,4 The translation product of FOXD3 is a transcriptional regulatory factor that activates or inhibits the expression of target genes, which not only activates gene cascades during the embryonic development stage but also regulates the differentiation of select embryonic stem cell populations.



Potential by Simultaneously Initiating and Repressing Enhancer Activity

However, Fork head box (FOX) gene family is divided into 19 subfamilies (or families) in humans and mice.

Role of FOXD3 and FOXD4 genes in human Hind Gut Development:

A properly functioning hindgut requires a complex web of nerves—known as the enteric nervous system (ENS)—to manage peristalsis and digestion. Along the anterior-posterior axis of the developing embryo, FOXD3 and FOXD4 function cooperatively to regulate structural development of the intestinal wall. Both the FOXD3 and FOXD4 regulates the generation, migration and the differentiation of neural crest cells. During development of embryo, the vaginal neural crest cells enter the foregut and migrate in a rostro-to-caudal direction, colonizing the entire gastrointestinal tract and generating the majority of the enteric nervous sytem (ENS). Foxd3 was strongly expressed in ENPs as they colonize the gastrointestinal tract and was progressively restricted to enteric glial cells.FOXD3 recruits SWI/SNF Complex subunit BRG1 to clear nucleosomes while simultaneously recruiting histone deacetylases (HDAC1/2) to repress maximal activation until the appropriate developmental stage. Using a novel Ednrb-iCre transgene to delete Foxd3 after vagal neural crest cells migrate into the midgut. Foxd3 is directly expressed within the epithelial cells of the rectum and the anus so it's disruption can fail the structural development of hindgut.

In case of FOXD4 it has both C and N -terminal end and both acts to specific domain. FOXD4 uses its N-terminal **acidic blob (AB) domain** to upregulate genes that support early cell proliferation and keep embryonic precursors in an immature state and uses its C-terminal **Eh1 domain** (which interacts with corepressors like Groucho) to downregulate genes that trigger premature differentiation. FOXD4 is heavily expressed in the embryonic epithelial cells of the rectum and anus.

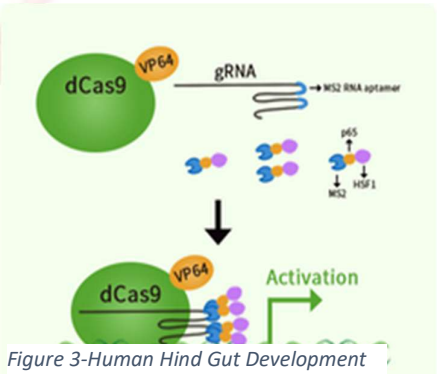


Figure 3-Human Hind Gut Development

The FOXD3/FOXD4 signalling axis functions to **maintain intestinal epithelial growth** and regulate wall development explicitly along the A-P axis, ensuring the hindgut forms distinctly from the foregut and midgut.

Aftereffects of Irregular Gene Regulations of FOXD3 and FOXD4 Gene:

1. **Embryonic Development & Neural Tube Defects:**
   Both FOXD3 and FOXD4 control cell pluripotency (the ability of a cell to develop into multiple cell types) and dictate early head, brain, and structural development and due to their irregular gene regulation Anorectal Malformations, Embryonic Lethality, Neural Tube & Craniofacial Malformations.
2. **Neural Crest Cell Disruption (Ocular & Facial Defects):**
   Neural crest cells (NCCs) are a temporary group of cells that migrate during development to form facial bones, the peripheral nervous system, and parts of the eye as for a irregular gene regulation anterior segment dysgenesis and congenital glaucoma.
3. *FOXD3* in the neural crest causes premature cell death among progenitors, leading to a severe reduction in the peripheral nervous system (PNS) and completely blocking the formation of the enteric nervous system (ENS) in the gut.

Preferences:

- **https://pubmed.ncbi.nlm.nih.gov/29307282/**
- **https://maayanlab.cloud/Harmonizome/gene/FOXD4**
- **https://www.sciencedirect.com/science/article/pii/S0012160612001376?_cf_chl_tk=jfUQXNrSu04Ve71V91Cz_rxu9w.69OFz88HSX0yQGlM-1789447714-1.0.1.1-CmyVikvbrubUgHOxMWEDnw6cHAa0CjmqXaVa6R0309w**
- **https://pubmed.ncbi.nlm.nih.gov/29307282/**
- **https://pubmed.ncbi.nlm.nih.gov/27868055/**

Jack-stat signalling pathway in human immune system

Nishith naga

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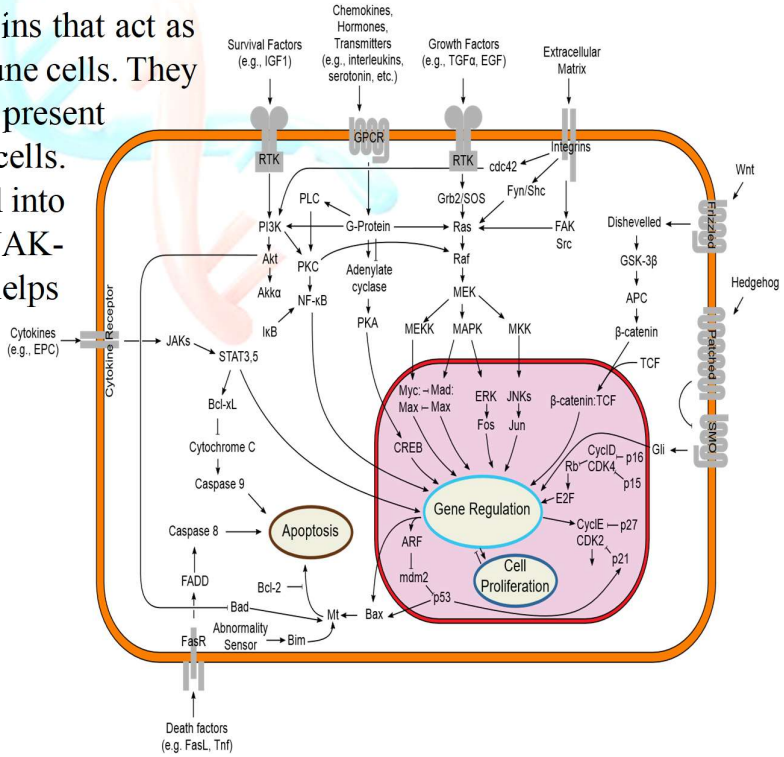
- **<u>Cytokines bind to receptors on immune cells:-</u>**

Cytokines are small proteins that act as messengers between immune cells. They bind to specific receptors present on the surface of immune cells. This binding sends a signal into the cell and starts the JAK-STAT pathway. It helps immune cells respond to infections and regulate inflammation.



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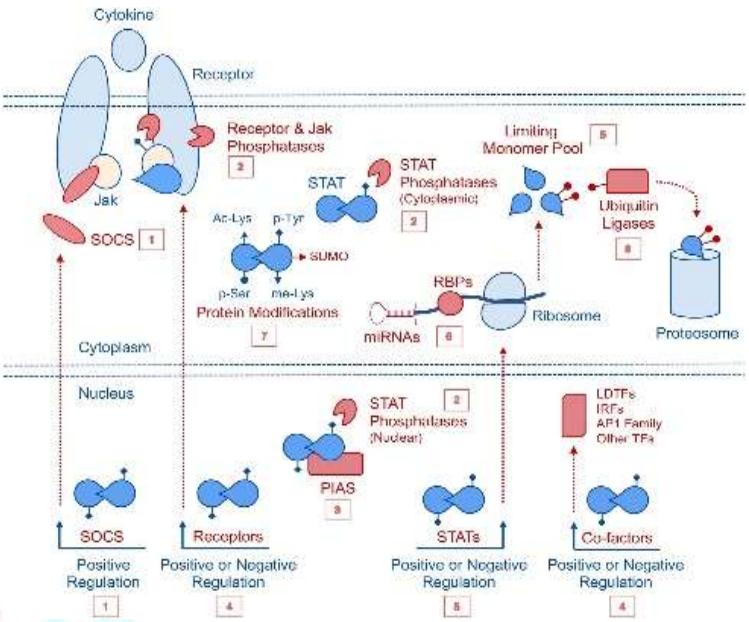
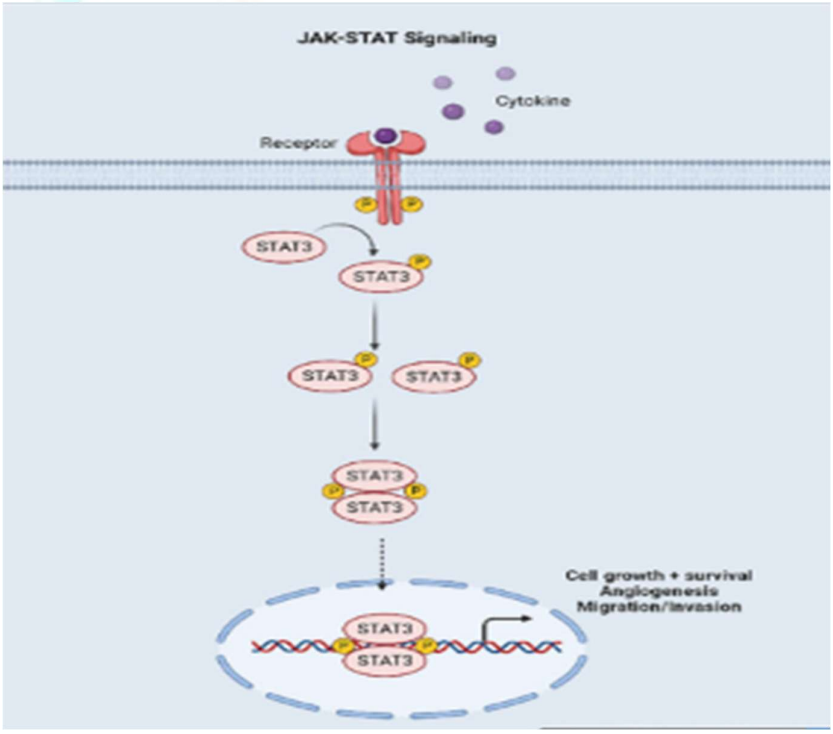
This activates JAK proteins inside the cell :-

JAK proteins are enzymes attached to the inside of cytokine receptors. When a cytokine binds to the receptor, JAK proteins become activated. Activated JAKs add phosphate groups to the receptor and STAT proteins.This starts the next step of the JAK-STAT signaling pathway, sir.



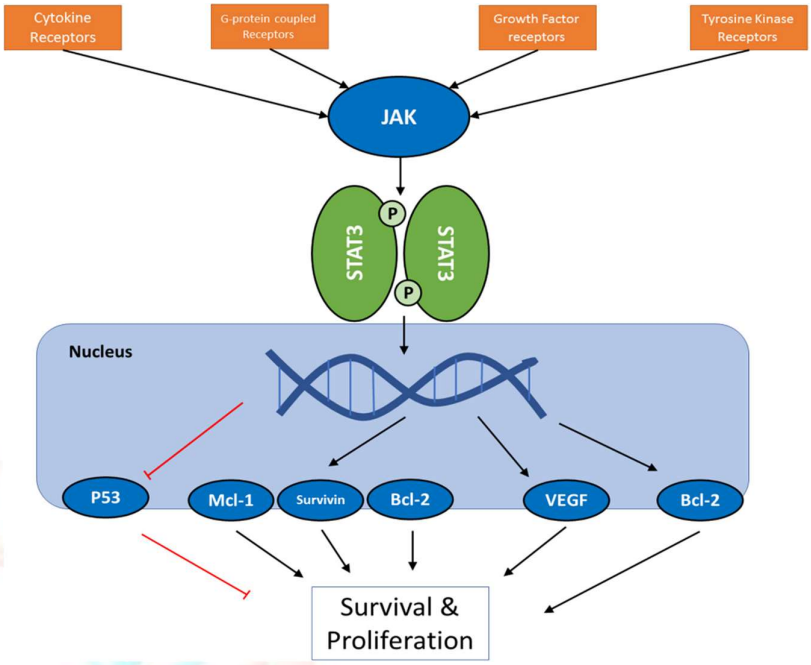
STAT proteins enter the nucleus and turn on immune-response genes:-

Activated JAK proteins add phosphate groups to STAT proteins. This phosphorylation activates the STAT proteins. Activated STAT proteins join together and form a pair. The STAT pair enters the nucleus and helps turn on specific immune-response genes.



STATs control genes that regulate immunity and inflammation:-

STAT proteins enter the cell nucleus after activation. They bind to specific DNA regions and switch certain genes on or off. These genes help immune cells grow, communicate, and respond to infections. STATs also regulate inflammation to help maintain a balanced immune response.



Applications of Recombinase Polymerase Amplification

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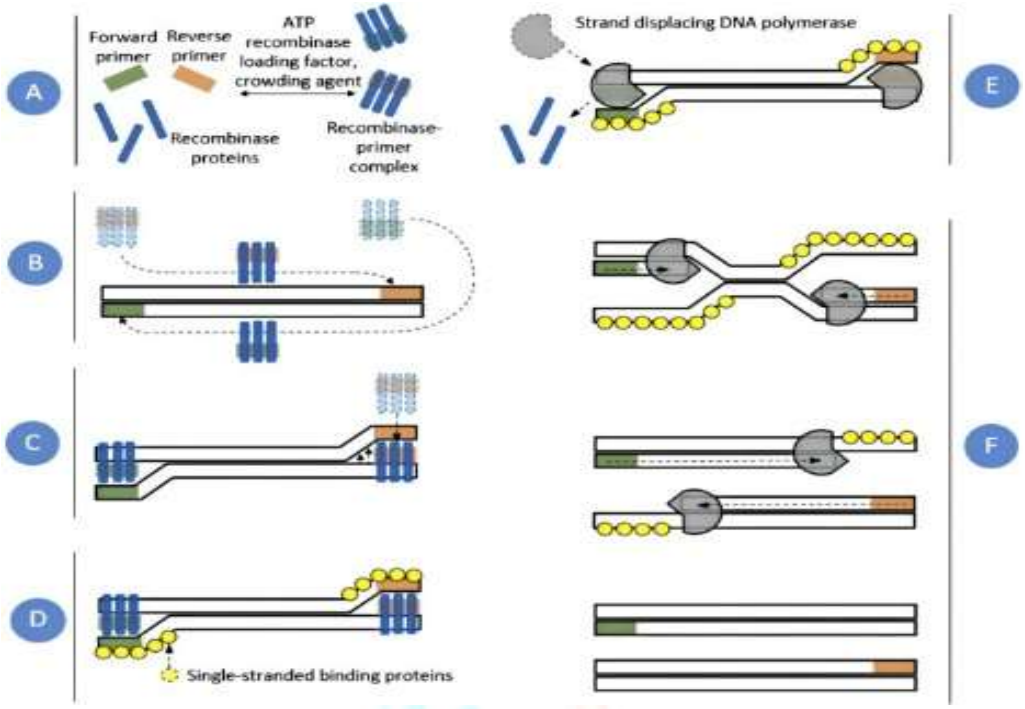
Recombinase Polymerase Amplification (RPA) is a rapid and innovative molecular technique that has gained considerable attention in biotechnology and molecular diagnostics.

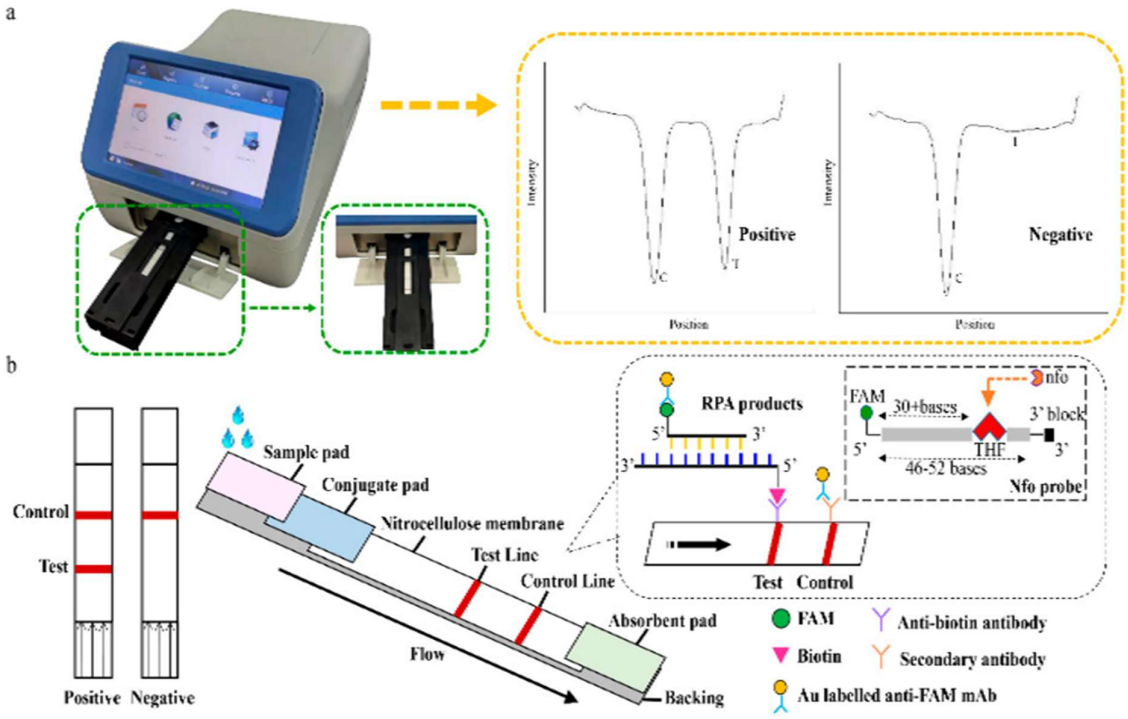
It is an isothermal nucleic acid amplification method that can produce large amounts of a specific DNA sequence at a relatively low and nearly constant temperature. Unlike conventional Polymerase Chain Reaction (PCR), RPA does not require repeated heating and cooling cycles, making it suitable for rapid and portable testing. The technique uses recombinase proteins, primers, single-stranded DNA-binding proteins and a strand-displacing DNA polymerase to recognize and amplify the target genetic sequence.

One of the major applications of RPA is **medical diagnosis**, particularly the rapid detection of infectious diseases caused by bacteria, viruses, fungi and parasites. When RNA targets are involved,

Reverse Transcription-RPA (RT-RPA) can be used for RNA detection. RPA is also valuable in **food safety**, where it can help detect foodborne pathogens such as *Salmonella*, *Listeria* and *Escherichia coli*, allowing potentially contaminated food products to be identified rapidly. In **agriculture**, RPA can be used to detect plant pathogens and diseases at an early stage, helping in timely disease management and reducing crop losses. Another important application is **environmental monitoring**, where RPA-based methods can assist in detecting microorganisms and specific genetic markers in samples such as water and soil. The technique is particularly promising for **point-of-care and field-based testing** because it can operate with relatively simple equipment and can be combined with portable detection systems. RPA products can also be detected using methods such as lateral-flow strips and fluorescence-based systems, making results easier to obtain outside conventional laboratories. Recent research has further explored combining RPA with **CRISPR-based detection, microfluidic devices and paper-based platforms**, creating opportunities for compact and decentralized diagnostic systems. Although RPA has several advantages, challenges such as nonspecific amplification, primer design, sample preparation and the need for further validation remain important considerations. Overall, Recombinase Polymerase Amplification is a promising molecular technology with applications extending from healthcare and food safety to agriculture and

environmental monitoring. Its rapid amplification and compatibility with portable detection systems may help make molecular testing more accessible in both laboratory and field settings.





Understanding the Transcriptome through RNA structure

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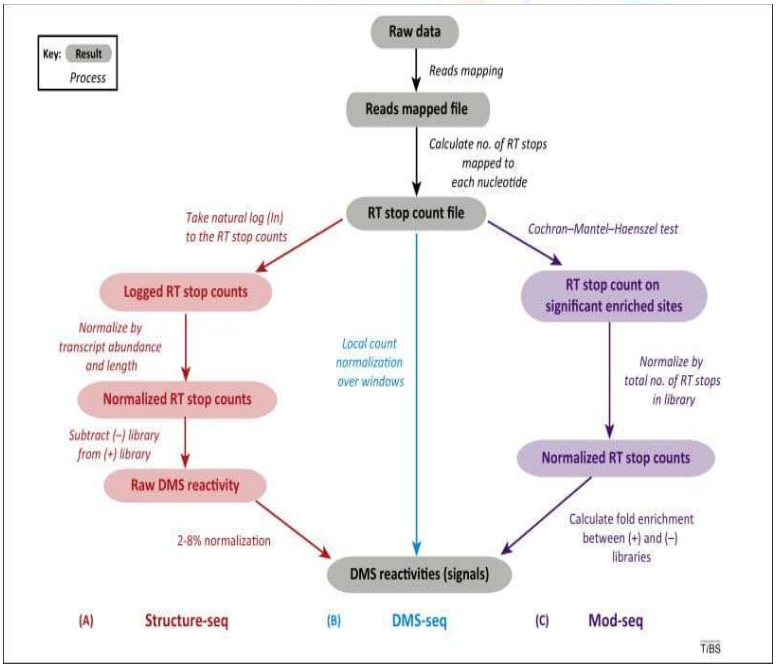
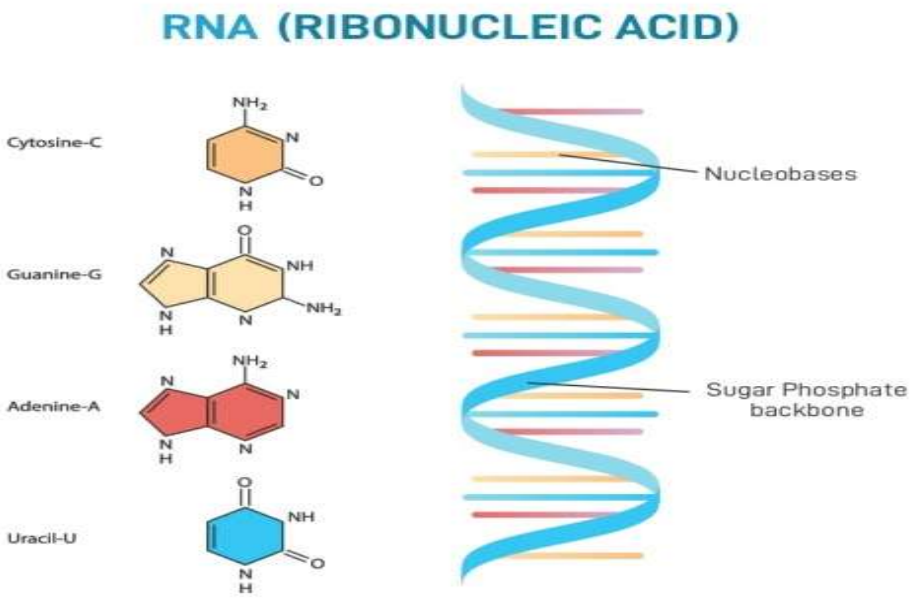
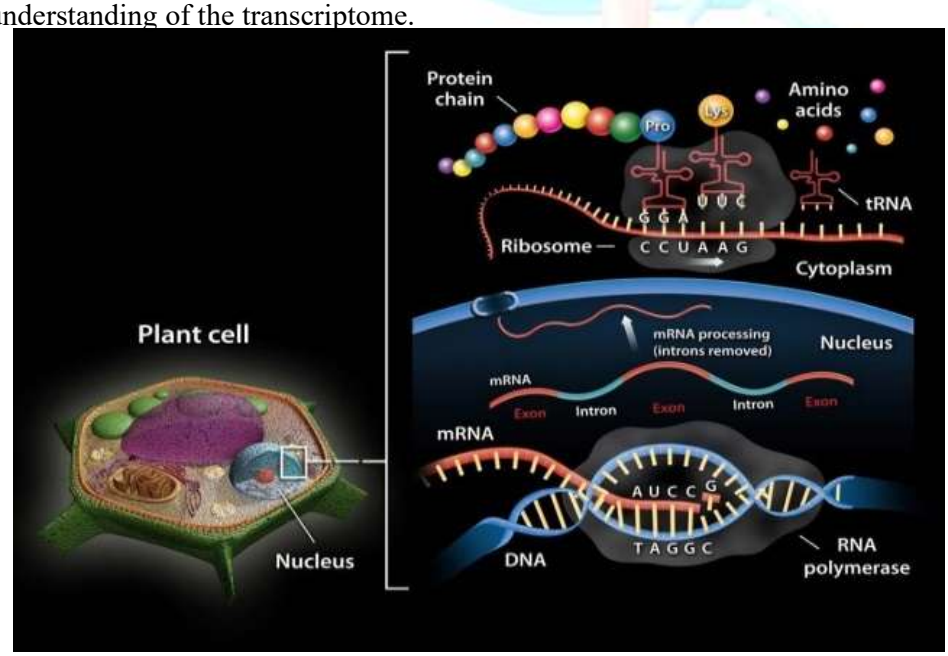
The transcriptome is the complete set of RNA molecules produced by a cell or organism at a particular time and under specific conditions. It mainly consists of mRNA, rRNA, tRNA, and other non-coding RNAs. The transcriptome reflects which genes are active in a cell.





RNA structure helps us understand the transcriptome because the structure and sequence of RNA are closely related to its functions. RNA is usually single- stranded and can fold into complex structures through base pairing, forming stem-loops, hairpins, and other secondary structures. These structures influence RNA stability, processing, transport, and translation.

Thus, studying RNA sequences along their structures helps researchers understand gene expression, RNA processing and cellular regulation, providing a more complete understanding of the transcriptome.



DETECTION AND ANALYSIS METHODS OF PROTEIN–PROTEIN INTERACTION

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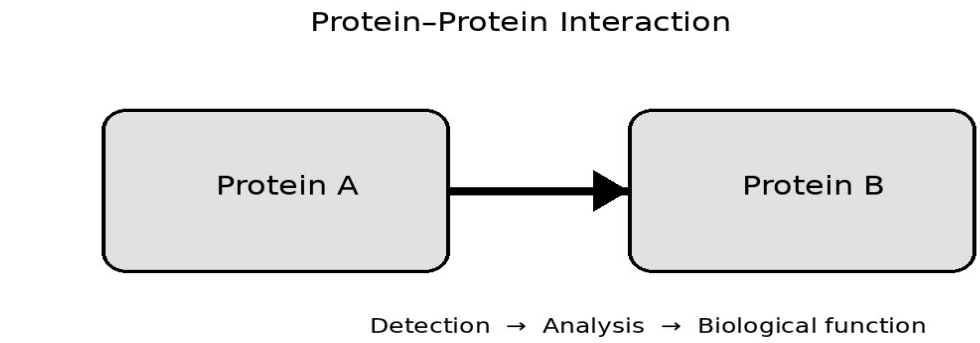
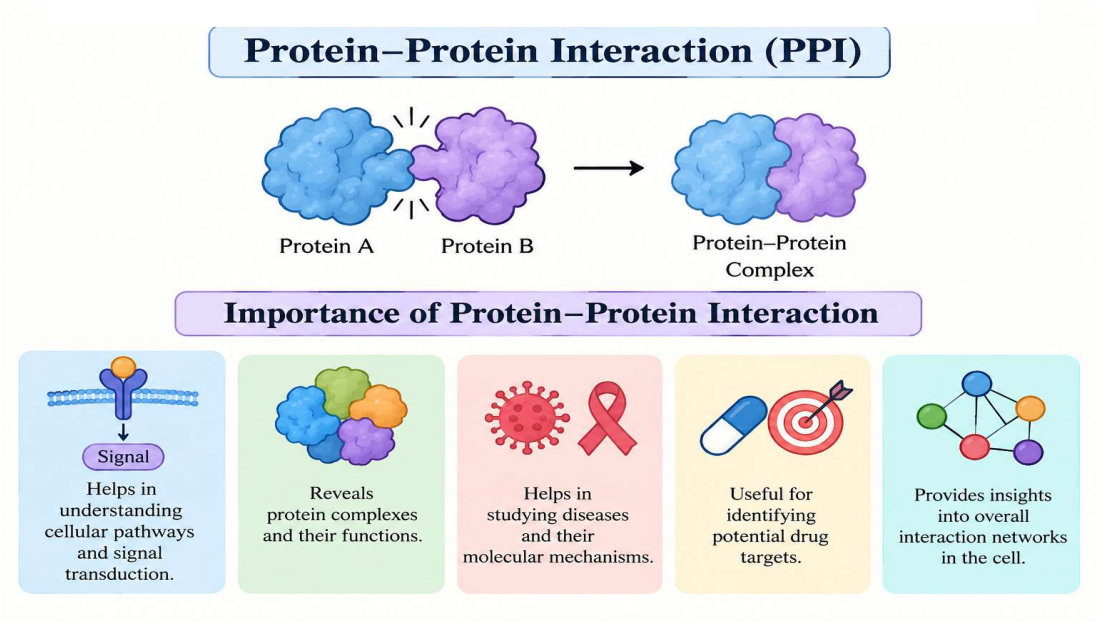
Topic Illustration





FLOWCHART

PROTEIN–PROTEIN INTERACTION (PPI)

Physical or functional association between proteins

↓

IMPORTANCE OF PROTEIN-PROTEIN INTERACTION

Protein–protein interactions are important for understanding cellular pathways, signal transduction, protein complexes, and various biological processes.

↓

DETECTION METHODS

↓

Yeast Two-Hybrid (Y2H) **Detects direct interaction using a reporter system.**	**Co-immunoprecipitation (Co-IP)** **Isolates protein complexes using an antibody.**
Pull-down Assay **Uses a bait protein to capture an interacting protein.**	**AP–MS** **Purifies complexes and identifies proteins by mass spectrometry.**
FRET **Detects close proximity of fluorescently labelled proteins.**	**Protein Microarray** **Screens many possible interactions simultaneously.**

↓

DATA ANALYSIS

Experimental controls → validation → reliable interactions → interaction network

↓

BIOLOGICAL APPLICATIONS

Cell signalling • Protein complexes • Disease studies • Drug-target discovery

↓

CONCLUSION

Combining complementary methods gives a more reliable understanding of PPI networks.

ATP SYNTHESIS BY OXIDATIVE PHOSPHORYLATION

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Definition: Oxidative phosphorylation is the final stage of aerobic respiration. It occurs mainly on the inner mitochondrial membrane and produces ATP using energy released from electrons.

1. Electron Transport Chain: NADH and $FADH_2$ donate electrons to the ETC. Electrons pass through protein complexes and release energy.

2. Proton Gradient: The released energy pumps H^+ into the intermembrane space, creating a proton gradient.

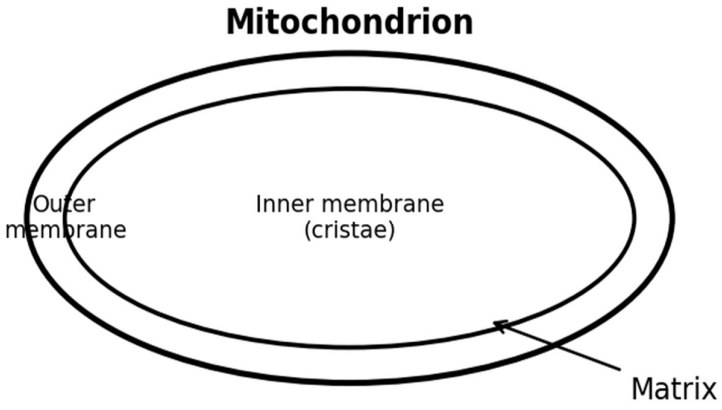
3. ATP Formation: H^+ flows back into the matrix through ATP synthase. This energy converts ADP + Pi into ATP.

4. Role of Oxygen: Oxygen is the final electron acceptor and combines with electrons and H^+ to form water.

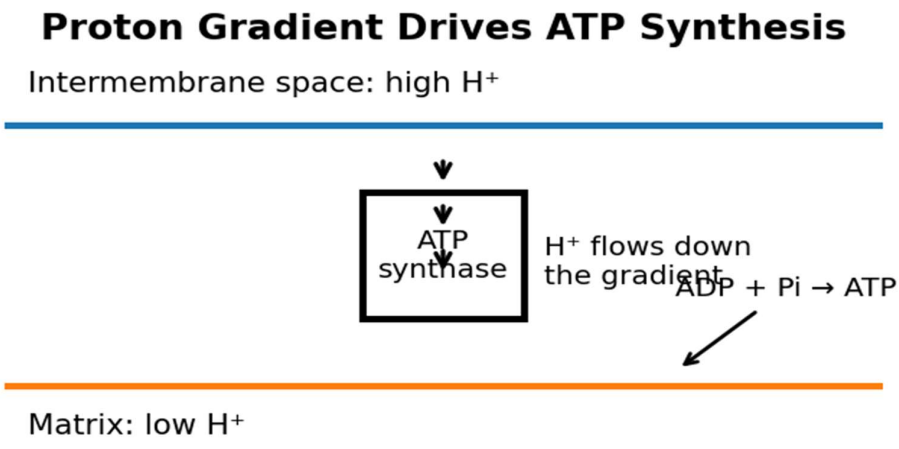
FLOWCHART

NADH / $FADH_2$ → Electron Transport Chain → H^+ Gradient → ATP Synthase → ATP

Importance: It produces most of the ATP used by aerobic cells for cellular activities.



Oxidative phosphorylation occurs mainly at the inner membrane


Proton Gradient Drives ATP Synthesis
Intermembrane space: high H+
ATP synthase
H+ flows down the gradient
ADP + Pi → ATP
Matrix: low H+

THE ENZYMATIC STEPS OF GLYCOLYSIS

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Glycolysis is the fundamental metabolic pathway operating within the cytosol of the cell, responsible for the anaerobic catabolism of carbohydrates. Over a series of ten sequentially coupled enzymatic steps, a single six-carbon glucose molecule undergoes oxidative degradation to yield two molecules of three-carbon pyruvate. The overall thermodynamic and chemical process is structured into two phases: the preparatory energy-investment phase and the subsequent energy-payoff phase.

PHASE I: PREPARATORY (ENERGY-INVESTMENT) PHASE

Step1: Hexokinase Catalysed Phosphorylation
Reaction: Glucose + ATP -> Glucose-6-Phosphate + ADP + H+
Details: The entry of glucose into the glycolytic sequence is initiated by the transfer of a terminal phosphoryl group from ATP to the C-6 hydroxyl group of glucose. This reaction is catalyzed by hexokinase, an enzyme requiring Mg2+ as a cofactor to stabilize the nucleotide substrate. The addition of a charged phosphate group effectively traps the carbohydrate within the cellular membrane, preventing its efflux. This step represents a key irreversible thermodynamic checkpoint.

Step 2: Isomerization by Phosphoglucose Isomerase
Reaction: Glucose-6-Phosphate <=> Fructose-6-Phosphate
Details: The second step involves an aldose-to-ketose conversion where glucose-6-phosphate undergoes reversible isomerization to form fructose-6-phosphate. The reaction is driven by phosphoglucose isomerase. This structural rearrangement alters the ring symmetry, exposing the C-1 carbon to facilitate a secondary phosphorylation event in the subsequent step.

Step 3: Phosphofructokinase-1 (PFK-1) Mediated Regulation
Reaction: Fructose-6-Phosphate + ATP -> Fructose-1,6-Bisphosphate + ADP + H+
Details: Phosphofructokinase-1 (PFK-1) transfers a phosphoryl group from a second ATP molecule onto the C-1 position of fructose-6-phosphate. This forms fructose-1,6-bisphosphate. Because this step is highly exergonic under intracellular conditions, it is virtually irreversible. PFK-1 serves as the primary allosteric regulatory control center of the entire pathway, sensitive to the energy charge of the cell (inhibited by elevated ATP and stimulated by AMP).

Step 4: Cleavage by Aldolase
Reaction: Fructose-1,6-Bisphosphate <=> Dihydroxyacetone Phosphate + Glyceraldehyde-3-Phosphate
Details: The enzyme aldolase splits the six-carbon fructose-1,6-bisphosphate via an aldol cleavage mechanism. The reaction yields two distinct triose phosphate intermediates: one molecule of dihydroxyacetone phosphate (an aldotriose) and one molecule of glyceraldehyde-3-phosphate (a ketotriose).

Step 5: Triose Phosphate Isomerase Interconversion
Reaction: Dihydroxyacetone Phosphate <=> Glyceraldehyde-3-Phosphate
Details: Since glyceraldehyde-3-phosphate is the only substrate capable of entering downstream metabolic steps, dihydroxyacetone phosphate must be rapidly converted into its isomer. This reversible reaction is efficiently managed by triose phosphate isomerase. Following this step, the preparatory phase concludes, and all subsequent metabolic yields are doubled per starting glucose molecule.

PHASE II: ENERGY-PAYOFF PHASE

Step 6: Dehydrogenation by Glyceraldehyde-3-Phosphate Dehydrogenase
Reaction: Glyceraldehyde-3-Phosphate + NAD+ + Pi <=> 1,3-Bisphosphoglycerate + NADH + H+
Details: Glyceraldehyde-3-phosphate dehydrogenase (GAPDH) coordinates a multi-step reaction involving both the oxidation of the aldehyde group and the structural addition of an inorganic phosphate. The high-energy electrons released during oxidation reduce the coenzyme NAD+ to form NADH. The final molecular product, 1,3-bisphosphoglycerate, contains an acyl-phosphate bond with significant group-transfer potential.

Step 7: Substrate-Level Phosphorylation by Phosphoglycerate Kinase
Reaction: 1,3-Bisphosphoglycerate + ADP <=> 3-Phosphoglycerate + ATP
Details: Phosphoglycerate kinase directs the transfer of the high-energy phosphoryl group from the carboxyl end of 1,3-bisphosphoglycerate over to a molecule of ADP. This leads to the direct biosynthesis of ATP. Since two triose units are processed simultaneously, this phase generates two ATP molecules, functionally breaking even with the initial energy investment of Phase I.

Step 8: Phosphoglycerate Mutase Mutation
Reaction: 3-Phosphoglycerate <=> 2-Phosphoglycerate
Details: The ester linkage of the remaining phosphate group undergoes a structural shift. The

enzyme phosphoglycerate mutase facilitates the internal migration of the phosphoryl group from the C-3 position to the C-2 hydroxyl position of the glycerate skeleton, producing 2-phosphoglycerate.

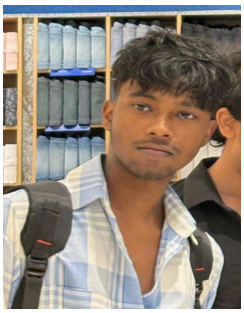
Step 9: Enolase Catalyzed Dehydration
Reaction: 2-Phosphoglycerate <=> Phosphoenolpyruvate + H2O
Details: Enolase drives a reversible dehydration reaction by removing a molecule of water from 2-phosphoglycerate. This introduces a double bond into the molecule, reorganizing electron density to generate phosphoenolpyruvate (PEP). PEP possesses an exceptionally high phosphate group-transfer potential, setting the stage for the final exergonic reaction.

Step 10: Pyruvate Kinase Mediated Final Energy Yield
Reaction: Phosphoenolpyruvate + ADP + H+ -> Pyruvate + ATP
Details: In the final step of glycolysis, pyruvate kinase catalyzes the irreversible transfer of the high-energy phosphate group from PEP to ADP, producing a molecule of ATP and converting the substrate into pyruvate. The reaction represents the second substrate-level phosphorylation checkpoint and marks the completion of the cytoplasmic pathway.

SUMMARY OF INTRA-CELLULAR MATERIAL BALANCES

Net Metabolic Inputs: 1 Glucose molecule, 2 oxidized NAD+ cofactors, 2 Adenosine Diphosphates (ADP), and 2 inorganic free phosphates (Pi).
Net Metabolic Outputs: 2 Pyruvate molecules, 2 reduced NADH units, 2 protons (H+), 2 cellular water molecules, and 4 gross ATP molecules.
Thermodynamic Energy Yield: A net gain of 2 functional ATP and 2 NADH per single oxidized molecule of glucose.

REPLISOME MEDIATED DNA REPLICATION



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DNA Double Helix

KEY COMPONENTS OF THE REPLISOME

DNA Helicase

Unwinds the parental DNA double helix at the replication fork.

Primase

Synthesizes short RNA primers needed to begin DNA synthesis.

DNA Polymerase

Adds nucleotides to the growing DNA strand in the 5′→3′ direction.

Sliding Clamp

Keeps DNA polymerase attached to DNA and increases its processivity.

Single-Strand Binding Proteins

Stabilize separated DNA strands and prevent them from reannealing.

DNA Ligase

Seals breaks between adjacent DNA fragments, especially on the lagging strand.

MECHANISM

The replisome is a coordinated molecular machine that functions at the replication fork. Helicase opens the DNA, primase lays down primers, and DNA polymerase extends the new strands. Because DNA strands are antiparallel, the leading strand is synthesized continuously while the lagging strand is synthesized as Okazaki fragments. Primer removal, gap filling and ligation complete the process.

BIOLOGICAL SIGNIFICANCE

Replisome-mediated replication ensures that genetic information is accurately copied before cell division. Proper coordination among its components is essential for genome stability and normal cellular function.

CONCLUSION

Replisome-mediated DNA replication is a highly coordinated process involving several proteins working together at the replication fork. Accurate completion ensures faithful transmission of genetic information to daughter cells.

FLOWCHART

free 3’-OH group.

↓

DNA SYNTHESIS

Leading strand → continuous synthesis

Lagging strand → discontinuous synthesis as Okazaki REPLISOME MEDIATED DNA REPLICATION

DNA replication is the process through which a cell accurately copies its DNA before division.

↓

INITIATION

Origin of replication → helicase unwinds DNA → replication fork is formed

↓

REPLISOME ASSEMBLY

Helicase + primase + DNA polymerase + sliding clamp + accessory proteins

↓

PRIMER SYNTHESIS

Primase synthesizes a short RNA primer to provide a fragments

↓

MATURATION

RNA primers are removed → gaps are filled with DNA → DNA ligase joins fragments.

↓

TERMINATION

Replication is completed and two daughter DNA molecules are produced.

↓

IMPORTANCE

Accurate DNA duplication → transmission of genetic information → cell growth and division

REPLISOME MEDIATED DNA REPLICATION

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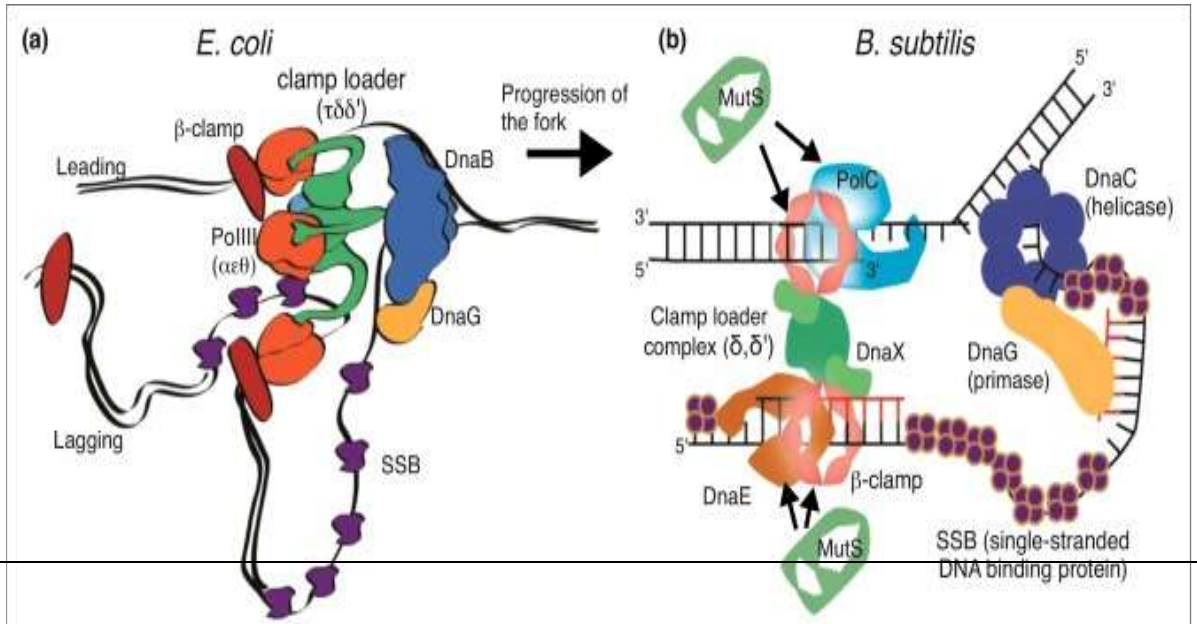
Topic Illustration

DNA Double Helix



FLOWCHART

REPLISOME MEDIATED DNA REPLICATION

DNA replication is the process by which a cell copies its DNA before cell division.

↓

INITIATION

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Helicase + primase + DNA polymerase + sliding clamp + accessory proteins

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PRIMER SYNTHESIS

Primase synthesizes a short RNA primer to provide a free 3′-OH group.

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DNA SYNTHESIS

Leading strand → continuous synthesis

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↓

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RNA primers are removed → gaps are filled with DNA → DNA ligase joins fragments.

↓

TERMINATION

Replication is completed and two daughter DNA molecules are produced.

↓

IMPORTANCE

Accurate DNA duplication → transmission of genetic information → cell growth and division

Molecular docking as a tool for Drug Design

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Introduction: Drug Design is the process of discovering and development molecules that con be modify a biological target to procedure a desired therapeutic effect.

What is Molecular Docking?

Molecular Docking is the computational method used to predict.

- How a ligand binds to a biological target.
- The strength of the ligand acid involved in binding.
- Target: Usually, a protein or enzyme.

An update on molecular docking in drug discovery.

Basic principle of Molecular Docking

Molecular Docking involves searching for the most favourable interaction between a ligand and the target.

Basic Principle

Target +Ligand→ Binding process →Scoring →Best pose.

Flow Chat

Target Selection

↓

Protein Preparation

↓

Binding site Identification

↓

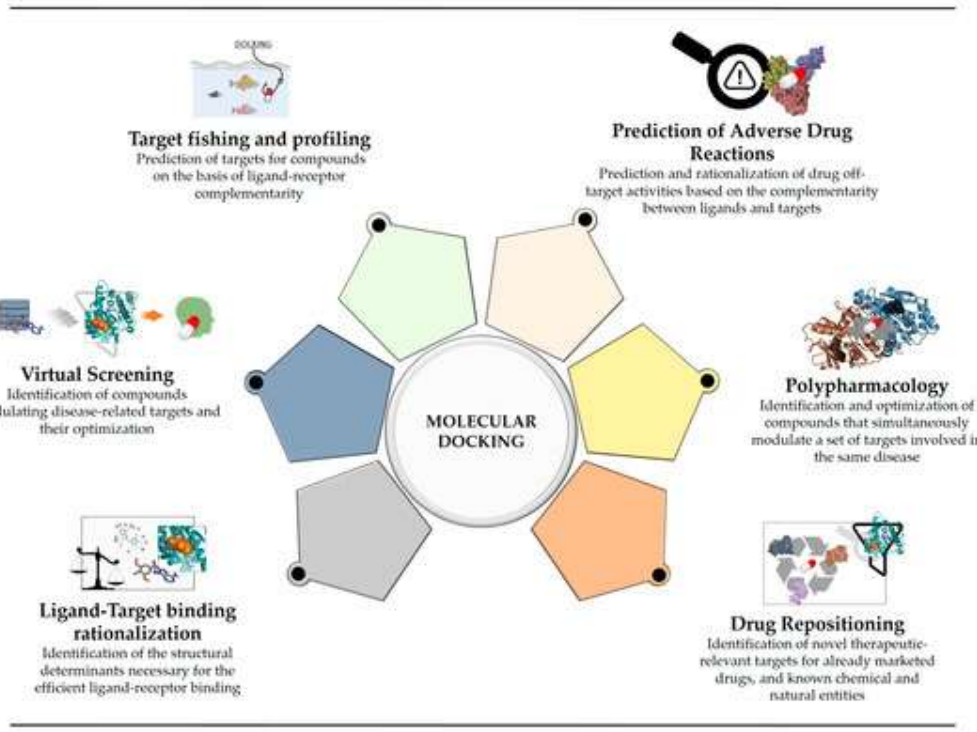
Ligand Selectin and Preparation

↓

Docking and stimulation

↓

Scoring and Ranking → Inter action Analysis → Lead Selection →Experimental Validation



Role of $CD8^+$ T cell and $FOXP^{3+}$ Cell in Relation to Breast Cancer

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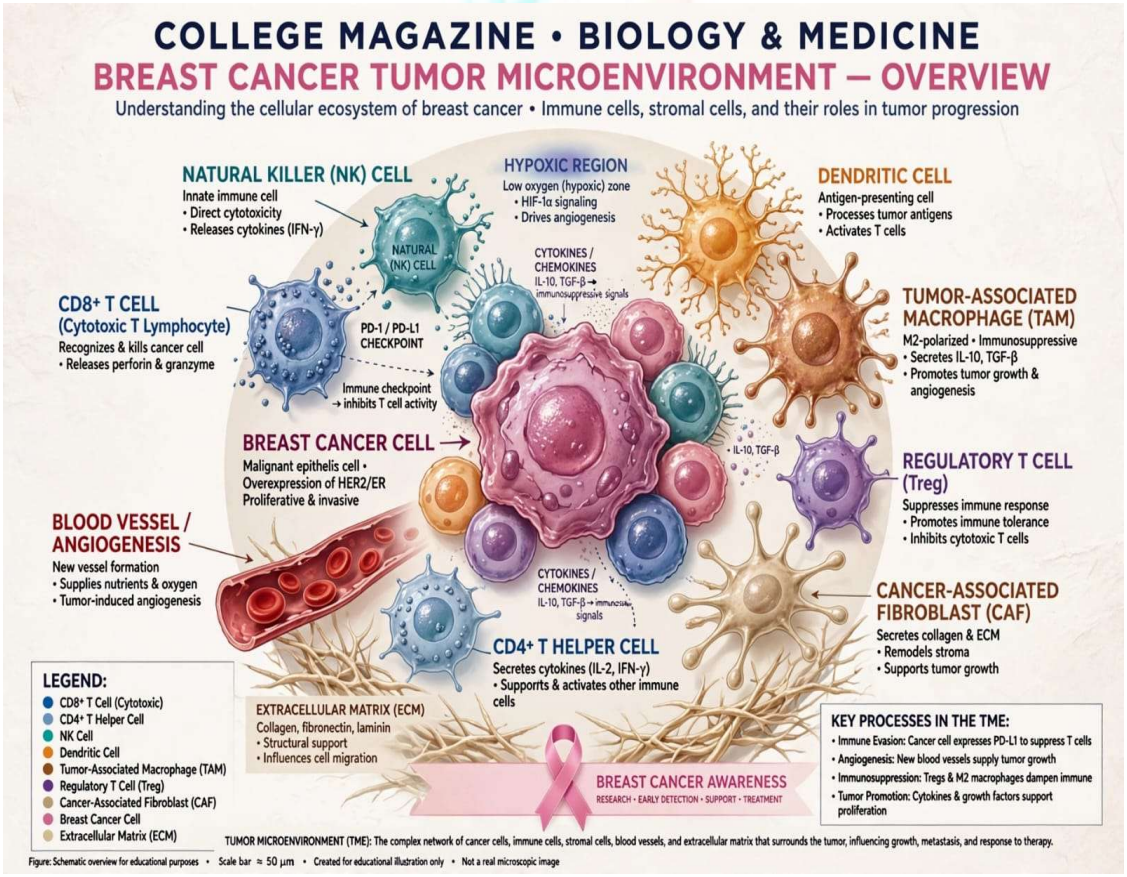
Introduction: Breast Cancer is Immunogenic. Breast Cancer is no longer considered a non-immunogenic tumour. Tumour infiltrating lymphocytes are now recognized as a critical prognostic factor. Among TILs, two subsets play opposite roles: $CD8^+$ cytotoxic T Lymphocytes are the killers, and **$FOXP3^+$** Regulatory T Cells are the suppressors.



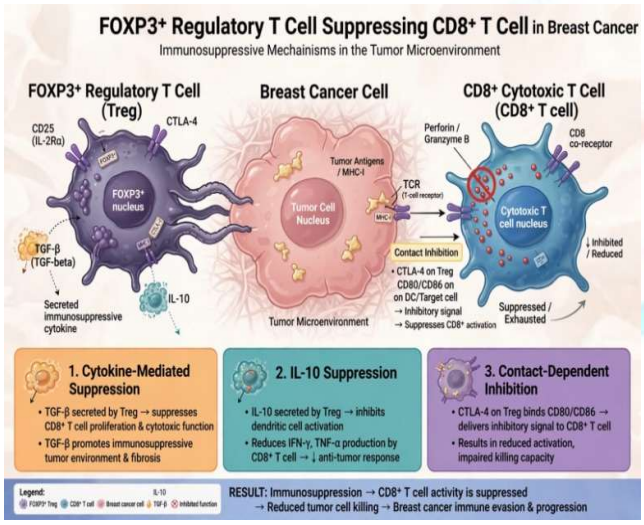
The hero - CD8^{+} TCELL: Who are they?

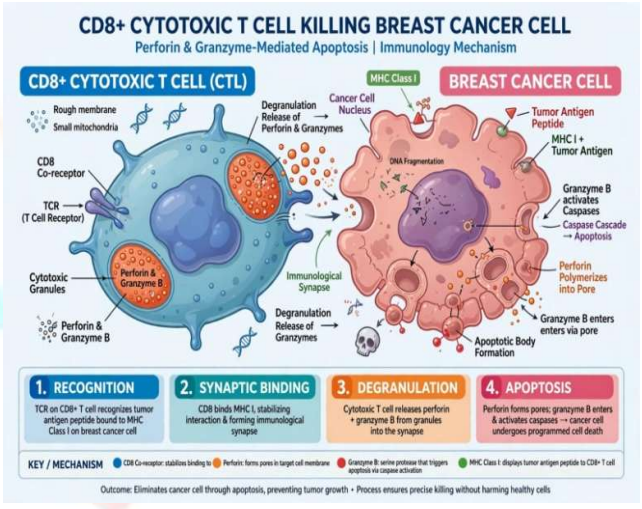
CD8^{+} Tells are cytotoxic T lymphocytes. They recognized tumor antigens via MHC class I and kill cancer cells directly.

Mechanism of Action:

1. Induces tumour cells cryostasis and apoptosis.
2. Releases Interferon – gamma which activates M1 macrophages.
3. Causes angiostatins (stops blood supply to tumour)

Clinical significance: High infiltration of **CD8^{+} T**ells is correlated with improved overall survival, better response to neoadjuvant chemotherapy, and lower incidence of lymph node metastasis. Patients with lymphocytes predominant Breast Cancer show up to 40% pathological complete response.





The villain FOXP^{3+} REGULATORY T CELL: Who are they?

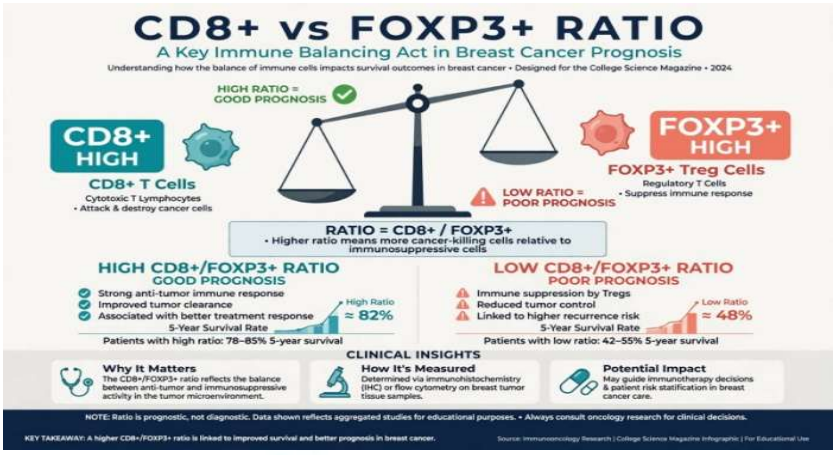
FOXP^{3+} is the master transcription factor and specific marker for Tregs. They are responsible for suppression of immune responses.

Mechanism of Action:

They suppress **CD8^{+}** Cells in to ways

- Contact dependent inhibition.
- Releasing suppressive cytokines like TGF - beta .

Clinical significance: Elevated **FOXP^{3+} Tregs** often correlate with poorer out comes due to immune escape and tumor progression.



THE MOST IMPORTANT CD8^{+} / FOXP^{3+} RATIO:

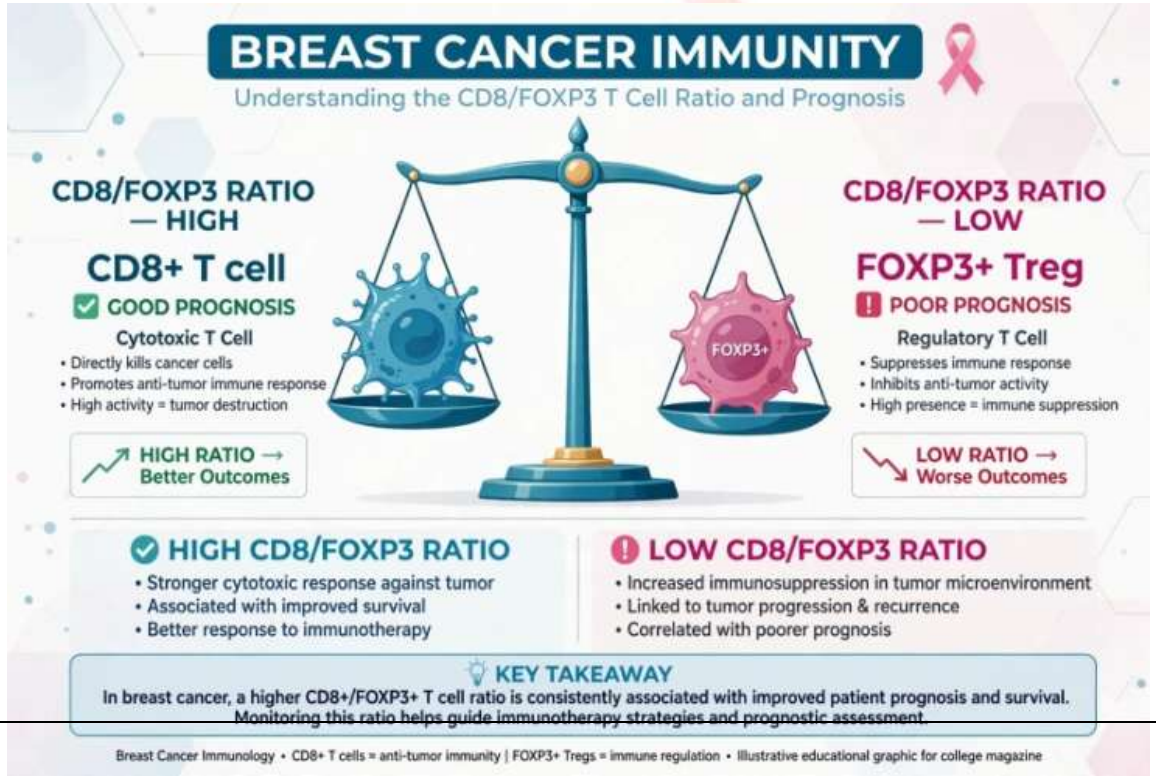
Single markers are controversial, but the ratio is the real prognostic marker.

High CD8/FOXP3 Ratio = Good Prognosis

Low CD8/FOXP3 Ratio = High Chance of Relapse.

Studies show:

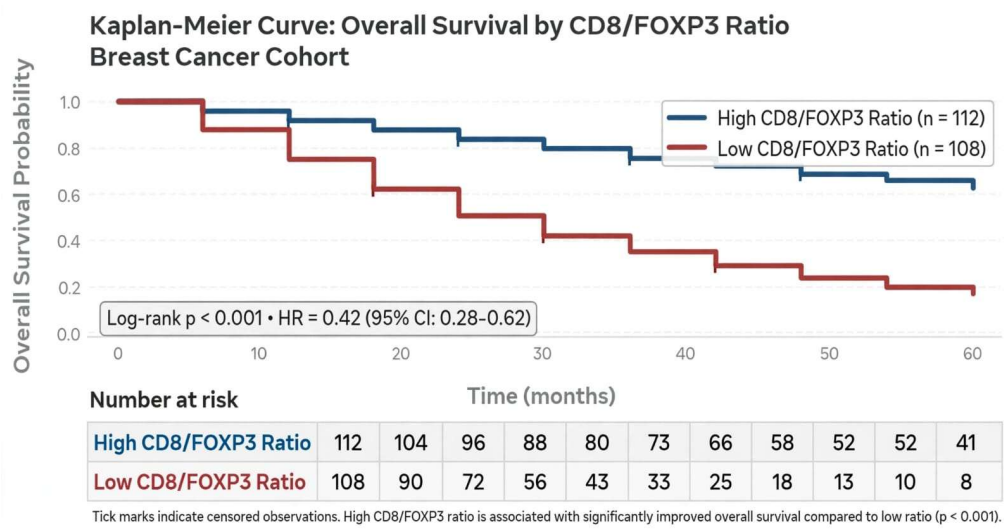
- High ratio patients have 39.6% per rate vs 13.3% in low ratio in triple negative Breast Cancer.
- High ratio in tumour stroma is associated with improved survival in non metastatic TNBC.

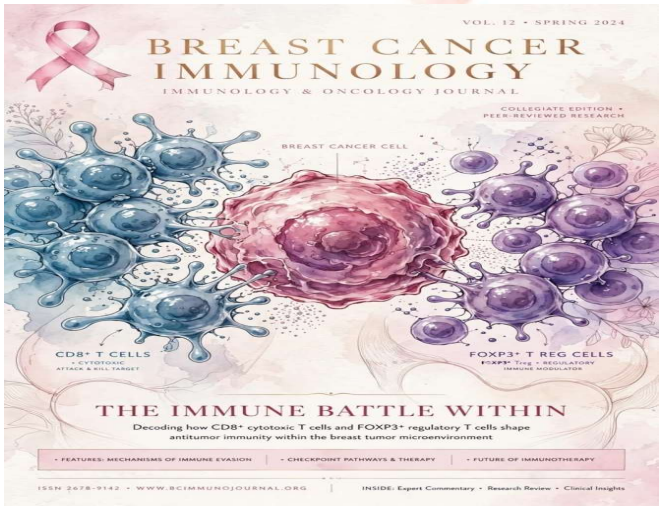


Therapeutic Implications:

1. Depletion of Tregs by blocking IL2 pathway can reprogram the microenvironment.
2. Immune checkpoint inhibitors work better when CD8/FOXP3 Ratio is high.
3. BRCA – mutated Breast Cancers have high immune infiltration and may benefit most from immunotherapy.

Conclusion: Breast cancer prognosis is not decided by the tumour alone, but by the battle between $CD8^+$ Attackers and $FOXP3^+$ suppressors. The $CD8^+$ / $FOXP3^+$ Ratio is emerging as a powerful predictive biomarker for personalized immunotherapy.





Cardiac Arrest in Young Age

ZEBA PARWEEN

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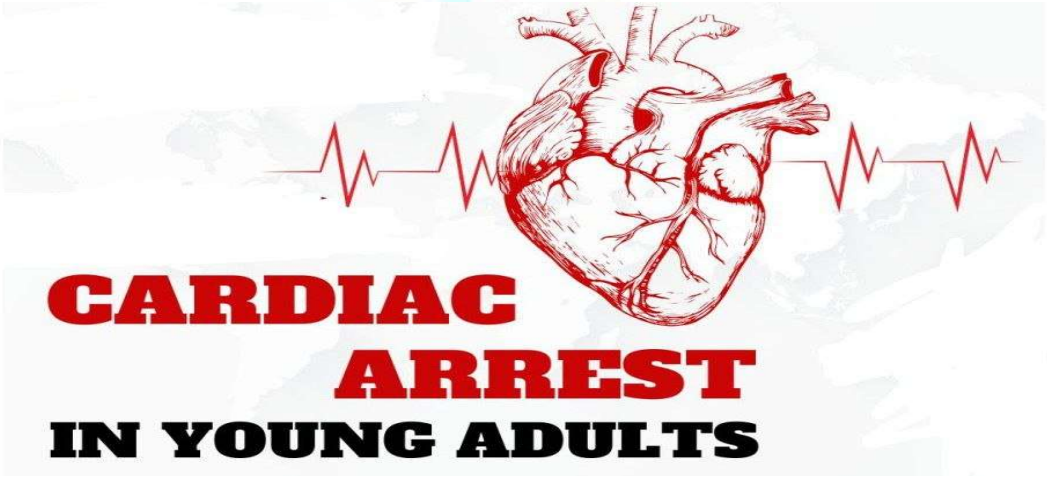
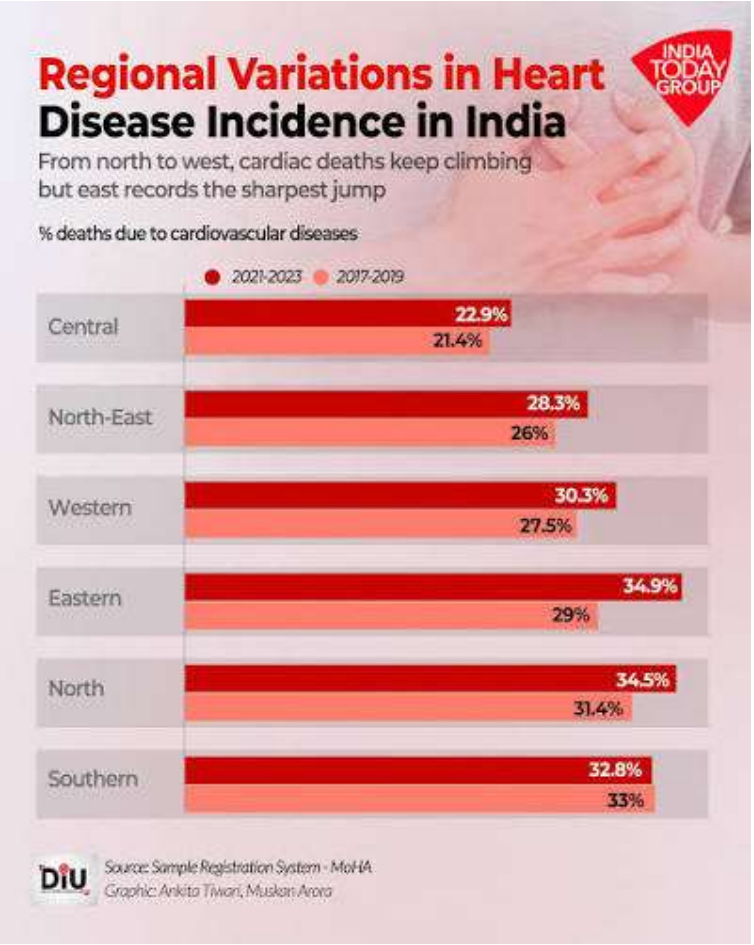


Cardiac arrest in young adults is an abrupt and severe cardiovascular crisis characterized by the sudden cessation of effective mechanical cardiac function due to lethal electrical instability. While ischemic heart disease predominates in older demographics, sudden cardiac arrest in young populations is primarily precipitated by silent inherited or structural abnormalities, such as hypertrophic cardiomyopathy, congenital coronary anomalies, and cardiac channelopathies. These underlying substrate defects predispose the myocardium to fatal ventricular arrhythmias, particularly during periods of intense physical exertion. Because

premorbid manifestations—including exertional syncope or chest tightness—are frequently subtle or unnoticed in otherwise healthy individuals, early athletic screening, prompt bystander CPR, and rapid defibrillation via an Automated External Defibrillator (AED) are essential to mitigate mortality.

Preventing sudden cardiac arrest relies on early clinical screening—such as ECGs and cardiac evaluations—to detect silent inherited heart conditions or structural defects before symptoms occur. High-risk individuals may require an Implantable Cardioverter-Defibrillator (ICD) to automatically shock and correct lethal rhythms.



The Guardian of the Genome: How p53 Fails in Liver Cancer

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Every day, our cells face a barrage of insults — ultraviolet light, toxins, replication errors, and viral infections — any of which can scar the DNA that carries our genetic instructions. Standing guard over this fragile molecule is a single protein: p53. Nobel laureate and cancer biologist **Sir David Lane** famously dubbed it the "guardian of the genome", and few nicknames in biology have proven so apt. Nowhere is p53's importance — and the consequences of its failure — clearer than in hepatocellular carcinoma (HCC), the most common form of primary liver cancer and one of the deadliest cancers worldwide.

What Does p53 Normally Do?

The TP53 gene, located on chromosome 17, encodes a protein that acts as a transcription factor — a molecular switch that turns other genes on or off. Under normal, unstressed conditions, p53 is kept at very low levels in the cell, largely through continuous degradation by a partner protein called MDM2. But the moment a cell experiences DNA damage or other stress, p53 is rapidly stabilised and activated.

Once switched on, p53 forms a tetramer (a cluster of four p53 molecules) and binds specific DNA sequences to switch on a set of protective genes. Depending on the severity of the damage, this triggers one of three outcomes:

- Cell-cycle arrest — pausing the cell cycle (largely via the gene p21) to allow time for DNA repair machinery to fix the damage.

- Senescence or repair — permanently retiring a damaged cell from the dividing pool, or coordinating repair enzymes.
- Apoptosis — if the damage is too severe to fix, p53 activates pro-death genes such as BAX and PUMA, instructing the cell to self-destruct for the good of the organism.

This decision-making role is why p53 is often compared to a genomic "emergency brake": it prevents cells carrying dangerous mutations from ever dividing and passing those mutations on. It is estimated that TP53 is altered in over half of all human cancers, underscoring how central this single gene is to preventing malignancy.

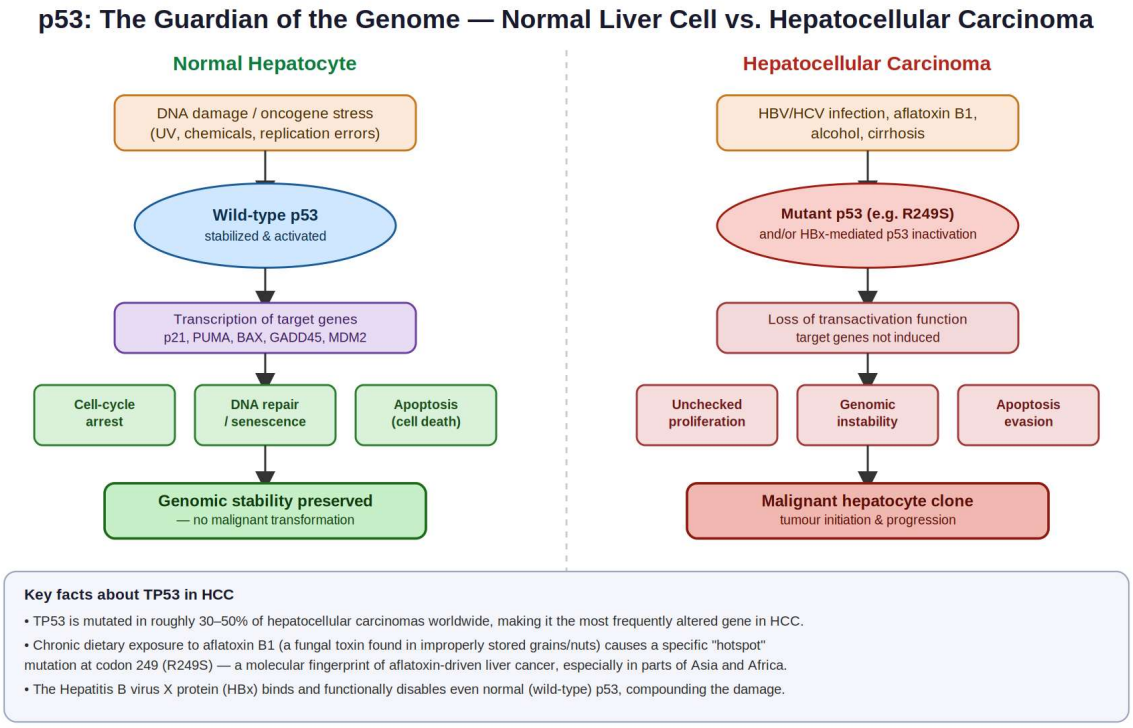

Figure 1. Simplified schematic comparing the p53 stress-response pathway in a healthy hepatocyte (left) with its disruption in hepatocellular carcinoma (right).

Why the Liver Is a Special Battleground

Hepatocellular carcinoma accounts for the large majority of primary liver cancers and remains one of the leading causes of cancer death globally, largely because it is often diagnosed at an advanced stage. Three risk factors converge on the liver in a way that makes p53 disruption especially common and especially damaging:

- Chronic hepatitis B or C infection, which causes ongoing liver inflammation and cell turnover, and, in the case of hepatitis B, produces a viral protein (HBx) that physically binds and inactivates p53.
- Dietary exposure to aflatoxin B1, a toxin produced by Aspergillus moulds that can contaminate improperly stored grains, maize and groundnuts in warm, humid climates.
- Chronic alcohol use, metabolic (fatty liver) disease and cirrhosis, all of which increase the rate of liver cell division and, with it, the odds of an uncorrected mutation.

The R249S Hotspot: A Molecular Fingerprint of Aflatoxin

One of the most striking discoveries in HCC research is that aflatoxin B1 does not damage TP53 randomly. After being metabolised by liver enzymes into a reactive epoxide, aflatoxin forms a DNA adduct that preferentially strikes a single codon: position 249 of the TP53 gene, converting an arginine to a serine (a mutation known as R249S).

This mutation is now recognised as a molecular fingerprint of aflatoxin exposure, and its prevalence tracks closely with regional dietary aflatoxin levels — reaching very high frequencies in parts of East and Southeast Asia and sub-Saharan Africa where aflatoxin contamination and hepatitis B infection are both common. Because hepatitis B and aflatoxin act synergistically, a person exposed to both carries a substantially higher risk of developing HCC than exposure to either alone.

When the Guardian Falls: Consequences for the Liver Cell

Across all forms of HCC, TP53 is estimated to be mutated in roughly a third to half of cases, making it the single most frequently altered gene in this cancer. When p53 is mutated or otherwise inactivated (for instance, by HBx binding), the consequences cascade through the cell:

- Loss of cell-cycle checkpoints allows cells with damaged DNA to keep dividing instead of pausing for repair.
- Failure to trigger apoptosis lets pre-malignant cells evade the “self-destruct” instruction that would normally eliminate them.

- Accumulating genomic instability compounds over successive cell divisions, producing the chromosomal chaos characteristic of advanced tumours.
- Some mutant p53 proteins acquire new, cancer-promoting "gain-of-function" activities, actively helping tumour cells invade tissue and resist chemotherapy, rather than simply losing their protective function.

Clinical and Research Significance

Because TP53 status shapes so much of a tumour's behaviour, it has become an important area for prognosis and drug development in HCC. Tumours carrying TP53 mutations, and especially the R249S variant, tend to be associated with more aggressive disease and poorer outcomes, making TP53 sequencing a candidate biomarker in liver cancer research. Public-health measures such as hepatitis B vaccination and improved crop storage to limit aflatoxin contamination directly reduce the two major insults that drive p53 mutation in the liver — making "protecting the guardian" a matter of prevention as much as treatment.

Meanwhile, researchers are exploring compounds that can restore normal folding and function to mutant p53 proteins, as well as therapies that exploit the vulnerabilities created when p53 is lost, illustrating how a decades-old discovery in basic cell biology continues to shape the search for new liver cancer treatments today.

Conclusion

The story of p53 in hepatocellular carcinoma is really the story of a single molecular guardian overwhelmed by a combination of chronic viral infection, dietary toxin exposure, and long-term liver injury. Studying exactly how and where this guardian fails — down to the level of a single altered amino acid at codon 249 — has given researchers precise molecular fingerprints of cancer-causing exposures and concrete targets for prevention and future therapy. It is a powerful reminder that some of the most important battles against cancer are fought, and sometimes lost, at the level of a single gene.

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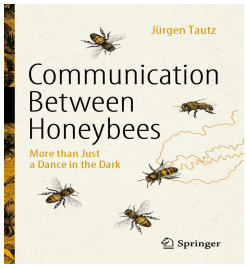
THE SECRET LANGUAGE OF BEES

(How Bees Communicate Without Words)



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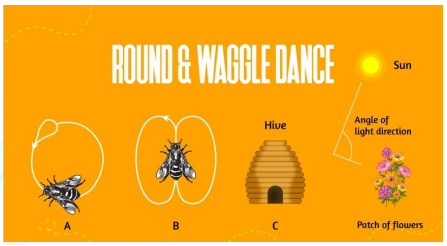
HOW DO BEES COMMUNICATE?

Bees cannot use human language, but they have a fascinating communication system. Honey bees communicate using body movements, chemical signals, sounds and vibrations. These signals help the colony find food, respond to danger and work together efficiently.



1. The Waggle Dance

The most famous bee communication method is the waggle dance. A worker bee that has found a useful food source can return to the hive and perform a patterned dance.

DANCE	DIRECTION	DISTANCE
The bee moves in a figure-eight-like pattern.	The angle of the waggle helps indicate the direction of the food source relative to the Sun.	The duration and characteristics of the waggle provide information related to distance.

Why is this amazing?

A bee returning from a food source can give other foragers useful information without taking them directly to the flowers. The dance acts like a biological information system.

2. Chemical Communication

Bees also communicate through chemical substances called pheromones. Different pheromones can influence colony activities such as queen recognition, alarm responses and coordination.

3. Sounds and Vibrations

Inside a busy hive, bees also use body movements, sounds and vibrations. These signals can help communicate at close range and support coordination among colony members.

WHY BEE COMMUNICATION MATTERS

A honey-bee colony works like a highly organized society. Communication allows thousands of individual bees to coordinate their activities even though each bee has a relatively simple role.

🌼	Finding Food
	Foragers can share information about productive food sources.
🚨	Responding to Danger
	Chemical signals can help trigger defensive behaviour.
👑	Maintaining the Colony
	Queen-related chemical signals contribute to colony organization.
🤝	Coordinating Work
	Signals help bees adjust their activities to the needs of the colony.

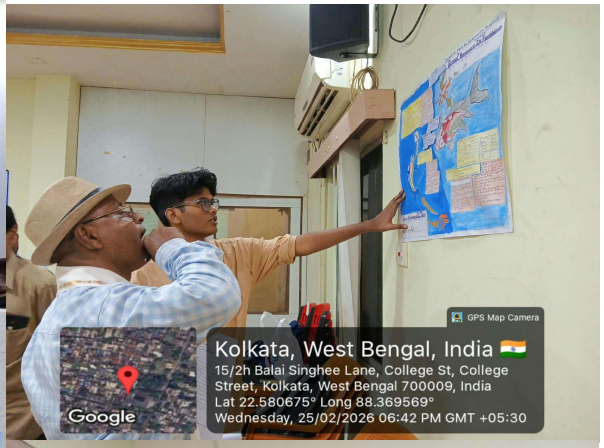
Fun Facts About Bees

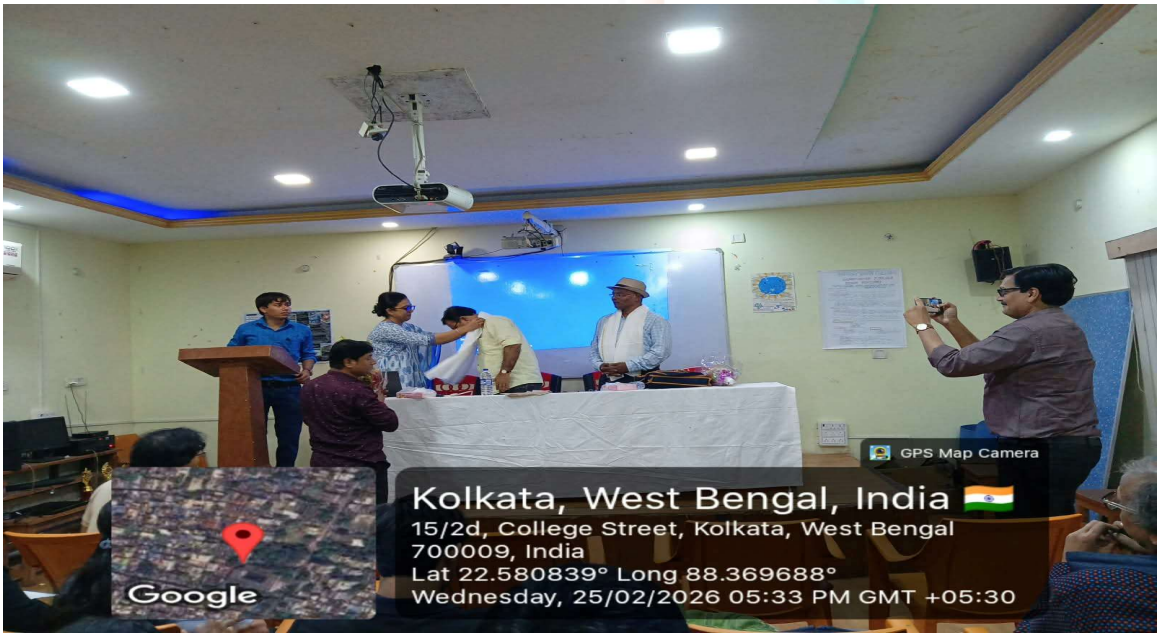
- 🐝 Honey bees live in highly social colonies.
- 🌞 The Sun can act as an important reference for the waggle dance.
- 🌸 A successful forager can help other bees locate rewarding flowers.
- 🧠 Bee communication is an important example of animal behaviour studied in zoology.

Conclusion

Bees show that communication does not require words. Through dance, chemical signals, sounds and vibrations, they exchange information and coordinate life in the hive. Their communication system is one of the most fascinating examples of animal behaviour.

Seminar On Fisheries and Aquaculture, Organized by Department of Zoology





Students Participating in International Seminar

Students Participating in Educational Excursion with Faculty Members: