Genomic Horizons

Second issue | July 2026

# Genomic Horizons

Quarterly e- Newsletter
of the
All India Congress of Genetics and Genomics


Advancing Science | Uniting Community | Inspiring Innovation | Shaping the Future

*Quarterly e-Newsletter of the*

# *All India Congress of Genetics and Genomics*

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# Recent Advances in Genetics and Genomics in plant, animal, human, microbial, environmental systems

***Second issue***

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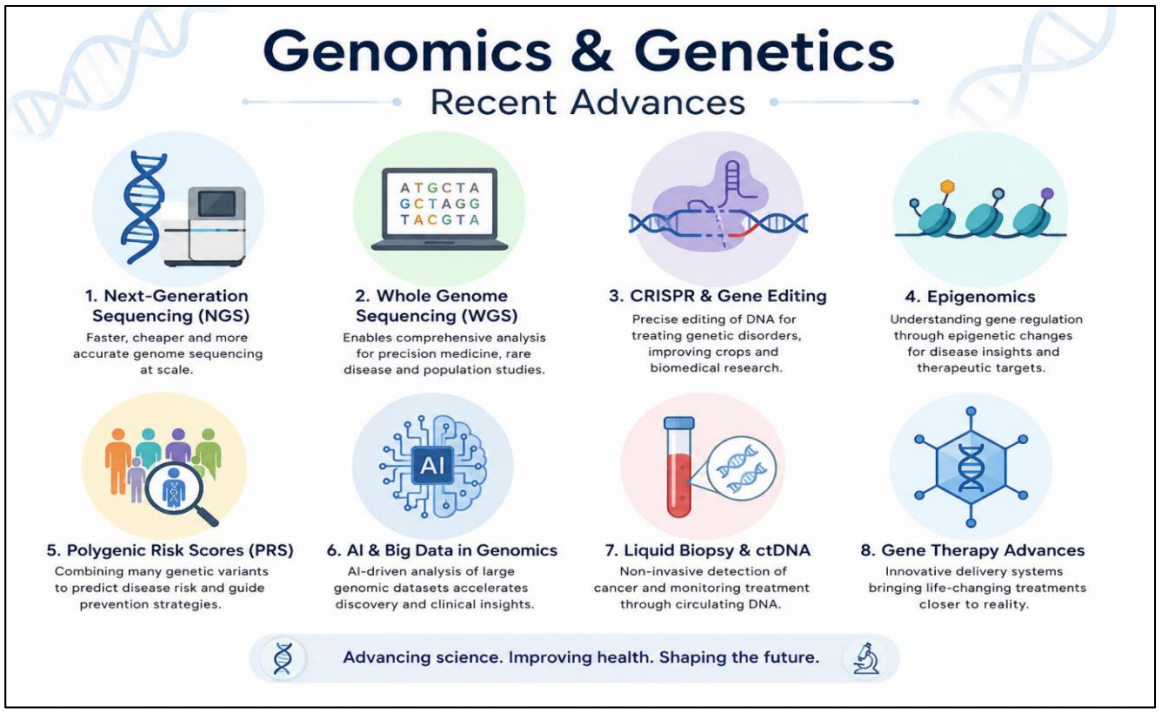
# Contents





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# Editorial

## The Genomic Renaissance: Advances Across Living Systems

Genetics and genomics are no longer locked away in research labs. Today, they are shaping medicine, farming, animal breeding, and even ecology in ways that were unimaginable a generation ago. With tools like **CRISPR gene editing, long-read sequencing, artificial intelligence, and synthetic biology**, we are entering a new era. Genomes are not just being read—they are being rewritten, interpreted, and used to solve real-world problems. This editorial aims to look at the latest advances across living systems, and the opportunities and challenges they bring.

### *Human Health and Medicine*

The biggest breakthroughs have come in human health. **CRISPR-Cas systems**, once experimental, are now being tested in patients. In 2025, trials showed that **base editing delivered with lipid nanoparticles** could safely treat conditions like hemophilia B and hereditary angioedema. Unlike older gene therapies, base editing makes precise changes to single DNA letters, lowering the risk of unwanted side effects. A newer method, **prime editing**, is being tested for more complex mutations. This could open the door to treating diseases that were once thought untreatable. Another major advance is in **genomic sequencing**. Long-read technologies such as PacBio HiFi and Oxford Nanopore give doctors a much clearer view of the genome. They can detect rare diseases faster, especially in newborns who need urgent care. Hospitals are now using whole-genome sequencing in neonatal intensive care units to save lives. Genomics is also changing how drugs are prescribed. **Pharmacogenomics** helps doctors choose cancer treatments that match a patient's genetic profile, reducing harmful side effects. **Polygenic risk scores** are being used to predict heart disease before symptoms appear, allowing preventive care. Artificial intelligence is also playing a role. **Liquid biopsies**, which test blood for traces of tumor DNA, are being improved with machine learning. These tests can detect cancers like liver and lung disease earlier, giving patients a better chance of survival.

### *Plant Genomics and Agriculture*

Farming is being reshaped by genomics. Traditional breeding relied on visible traits, but now scientists can directly adjust genetic pathways. In rice, genomics is helping improve yield, nutrition, and stress tolerance. Wheat research is focusing on resilience to drought and salty soils—key challenges in a warming climate. **Multi-omics approaches** are being used to improve forage crops for livestock. Fruits and vegetables are also benefiting, with genomics helping fight viral diseases and reduce pesticide use. Advances in **fungal genomics** are allowing scientists to engineer symbiotic fungi that help crops absorb nutrients and withstand stress. Beyond farming, fungi are being used in biotechnology to produce enzymes and other useful compounds.

### *Animal Genomics*

Animal breeding has entered the genomic era. **Genomic selection**, which uses DNA markers to predict traits, is speeding up progress in cattle, poultry, and fish farming. Farmers can now breed animals that are healthier, more productive, and better suited to changing climates. Genomics is also vital for disease control. Sequencing pathogens in livestock helps track and prevent outbreaks, reducing the risk of diseases spreading to humans. This kind of surveillance is crucial in a world still wary of pandemics.

### *Microbial and Environmental Genomics*

Genomics is revealing the hidden diversity of human populations. A major study in Pakistan found thousands of naturally "switched-off" in microbial genes, offering new insights into resilience and disease risk. Such studies are important for making sure genomic research represents all populations, not just those in wealthy countries. New techniques like **spatial CRISPR screening** are mapping how genes work inside tissues, showing how they interact in complex environments like tumors or

microbial communities. Genomics is also central to fighting **antibiotic resistance**. By identifying resistance genes in bacteria, scientists can design better drugs and guide public health strategies.

***AI and Synthetic Biology***

Artificial intelligence is now being trained on DNA sequences, much like language models are trained on text. These **genomic language models** can predict how genes function and what mutations might mean. They are speeding up discoveries in functional genomics. Synthetic biology is pushing boundaries further. Scientists are designing **gene circuits** that can sense and respond to their environment. These could be used in cancer therapies or industrial processes. Chimeric RNAs and engineered proteins are expanding what genomes can do.

***Challenges and Ethics***

Despite progress, challenges remain. Genomic research is still heavily focused on people of European descent. This lack of diversity risks leaving out important genetic insights and widening health gaps. Privacy and consent are also major concerns. As genomic data is linked with health and social information, questions arise about surveillance, discrimination, and personal rights. Finally, cost is a barrier. While sequencing is cheaper than before, advanced therapies remain expensive. Without policies to ensure access, genomic medicine could become a privilege of wealthy nations. Thus, it might be said that, the advances in genetics and genomics across living systems represent a true **renaissance in biology**. From CRISPR cures for genetic diseases to AI-driven crop improvement, the possibilities are vast. But the real test will be whether these breakthroughs are being used responsibly. Genomics is not just about reading the book of life—it is about rewriting it. As we enter this new era, the challenge is clear: to harness the power of genomes for the collective good, ensuring that the benefits of precision biology reach all species, ecosystems, and societies.

This issue (**Theme: Recent Advances in Genetics and Genomics in Plant, Animal, Human, Microbial, and Environmental Systems)** of the journal highlights the remarkable breadth of genetics and genomics research across biological systems. The exploration of microbial genomics in enzyme discovery demonstrates how fundamental science can accelerate biocatalyst innovation. The study of waterborne toxicants and their epigenomic imprint on early life underscores the environmental determinants of long-term metabolic health. Insights into the genetic regulation of efflux pumps in *Pseudomonas aeruginosa* remind us of the urgent need to confront antibiotic resistance through molecular understanding. Finally, the integrative perspective on Polyendocrine Metabolic Ovarian Syndrome (PMOS) exemplifies how One Health, genomics, and epigenomics converge to address complex human disorders in the context of environmental and clinical factors.Together, these contributions reflect the dynamic advances in genetics and genomics across plant, animal, human, microbial, and environmental systems—showcasing how interconnected approaches are shaping the future of science and medicine.

**References**


1. Anzalone, A. V., et al. (2020). *Prime editing*. Nature.
2. Appels, R., et al. (2018). *Wheat genome research*. Science.
3. Cristiano, S., et al. (2019). *Liquid biopsy in cancer*. Nature.
4. Gillmore, J. D., et al. (2025). *CRISPR clinical trials*. NEJM.
5. Logsdon, G. A., et al. (2020). *Long-read sequencing*. Nat Rev Genet.
6. Popejoy, A. B., & Fullerton, S. M. (2016). *Genomics and diversity*. Nature.
7. Saleheen, D., et al. (2017). *Human knockouts and phenotypic consequences*. Nature.


***Editor-duo***

**Dr. Nilanjana Banerjee & Dr. Vikas Srivastava**

## Article 1

# From Genomics to Biocatalysts: How Microbial Genomics is Accelerating Enzyme Discovery

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Over about 4 billion years, life on Earth evolved and shaped the once-hostile planet into a biosphere rich in remarkable biological diversity. The transition from prebiotic chemistry to protocells, followed by the emergence of the first cellular organism, marked the beginning of Earth's most long-lasting occupant: a microorganism. Microbes have consistently adapted to changing environmental conditions throughout their evolutionary journey, acquiring new genetic traits and developing novel metabolic capacities. This enormous genetic diversity provides a platform for the discovery of novel enzymes, metabolic pathways, and biological functions, many of which remain unexplored. Modern genomic technologies have enabled researchers to access vast amounts of genetic information, leading to new opportunities to discover novel biomolecules and biocatalysts for use in biotechnology, medicine, agriculture, and sustainable industrial processes.

Numerous technological developments that advanced the study of biological systems, from individual genes to whole genomes, gave rise to the genomics era. Modern genomics began with the launch of large-scale genome sequencing programs in the 1990s. However, the foundations were laid by the discovery of DNA structure and the development of DNA sequencing tools, culminating in the landmark completion of the first complete bacterial genome in 1995. Millions of microbial genome sequences are now available in public repositories, including NCBI, IMG, EMBL-ENA, MGnify, GTDB, and DDBJ, and next-generation sequencing has reduced the time and cost of genome sequencing. Despite the remarkable potential of genomic databases, an obstacle remained posed by unculturable microorganisms.

Metagenomics, a culture-independent method that permits the direct recovery and analysis of genetic material from environmental samples, emerged as a transformative culture-independent technology that revolutionizes human access to biological information. This revolution has increased our understanding of the human microbiome and its role in health and illness, accelerated the development of new enzymes, antibiotics, and bioactive substances, and enabled the development of new instruments for bioremediation and environmental monitoring. It provides direct access to the genetic blueprints of whole microbial ecosystems. As a result, billions of hitherto unidentified genes are now accessible for investigation; determining which of these genes encode biologically relevant and industrially valuable functions is a major challenge nowadays. To anticipate function before experimental confirmation, the genome-mining approach is widely helpful. The discovery of biosynthetic gene clusters linked to the manufacture of natural products is made easier by genome-mining tools such as anti-SMASH, BAGEL, and PRISM. When combined with bioinformatic tools such as Pfam, InterPro, and HMMER, and carbohydrate-active enzyme identification using dbCAN, these resources enable researchers to identify promising candidates for experimental validation and to deduce gene function. These approaches provide the initial step in transforming genomic information into functional enzymes and biocatalysts.

Advancements in metagenomics have created an un-rivalled opportunity to investigate microbial activities at the community level in ways previously not possible. Researchers are increasingly integrating comparative genomics and metagenomic approaches to correlate genomic insights with microbial communities across diverse geographical and ecological niches. These approaches have enabled scientists to discover new metabolic pathways, track the evolution of infections, improve agricultural and livestock production, and identify the molecular basis of diseases. Comparative genomics combined with functional genomics has expedited the identification of genes involved in specialized metabolic processes, antibiotic resistance, and environmental adaptation. Such discoveries expand understanding of biological systems and enable the development of novel biotechnology solutions by identifying putative enzyme-coding genes, valuable targets for pathway engineering, sustainable farming practices, and innovative therapeutic applications.

As aforementioned, technological advancements are continuously contributing towards the rapid increase in the volume of genomic data, and challenges associated with data validation, annotations, and management are becoming significant. Consequently, the identification and functional characterization of genes remain labor-intensive and time-consuming processes. The artificial intelligence (AI) and machine learning (ML) revolution has significantly impacted nearly every aspect of scientific research, improving the efficiency of genomic data analysis and interpretation. Notably, tools such as AlphaFold have enabled structural prediction for proteins that previously lacked experimental structural information. Various models developed for specialized tasks are trained using large-scale sequence datasets, experimental enzyme data, and curated biological databases. These models or tools are used to establish patterns and relationships among sequence, structure, and function, thereby improving annotation and gene-discovery pipelines, among many other applications.

Beyond identifying novel enzymes and providing structural information, genomics, combined with multi-omics techniques, aids in reconstructing metabolic pathways and discovering regulatory mechanisms. This systems-level understanding enables researchers not only to identify genetic attributes but also to optimize metabolic flux and biosynthetic pathways. This kind of information is critical for enhancing microbial strains used in industrial production and for metabolic engineering to develop industrially compatible biosynthetic systems. For example, a metagenome-mined thermostable novel D-allulose 3-epimerase variant can be used to develop microbial cell factories for the industrial production of valuable rare sugars. AI, functional genomics, and genomics are converging to transform research into practical applications. Various such metagenome-mined enzyme variants are used in food processing, pharmaceutical synthesis, agriculture, and environmental biotechnology applications. Microbial enzymes help to produce rare sugars, amino acids, and fermentation products in the food industry. Biocatalysts are ecologically friendly substitutes for sophisticated chemical synthesis in pharmaceutical manufacture. Biofertilizers, microorganisms that promote plant development, and biomolecules that reduce stress are examples of agricultural uses. Enzymes that enable waste valorization, detoxify contaminants, and break down polymers are examples of environmental uses. Together, resource-efficient manufacturing processes and advancements in biomanufacturing are strengthening the establishment of a sustainable bioeconomy.

Despite this significant progress, technological limitations still exist. Many genomes lack functional annotations due to limitations in sequencing. Similarly, many biosynthetic pathways remain poorly understood and lack scientific evidence. The integration of genomics, metagenomics, synthetic biology, artificial intelligence, and automated bio-foundries holds potential for future developments. This integrated approach is helpful in reducing the time required for scientific discoveries to translate into industrial applications of biocatalytic processes. Simultaneously, improved access to previously unexplored microbial communities is likely to uncover new classes of enzymes and processes relevant to industry. Thus, the journey from early sequencing techniques to modern multi-dimensional genomic approaches, encompassing genome mining, functional genomics, AI/ML-driven

analytics, and systems biology, has significantly accelerated the discovery, characterization, and engineering of novel biocatalysts (Figure 1). Microbial genomics has evolved as a powerful method for discovering enzymes and developing biocatalytic systems. By harnessing the potential of microbial diversity, researchers are opening new possibilities in agriculture, food production, healthcare, and environmental sustainability. Genome mining will remain essential for the development of next-generation biocatalysts as genomic resources and computational techniques continue to evolve.

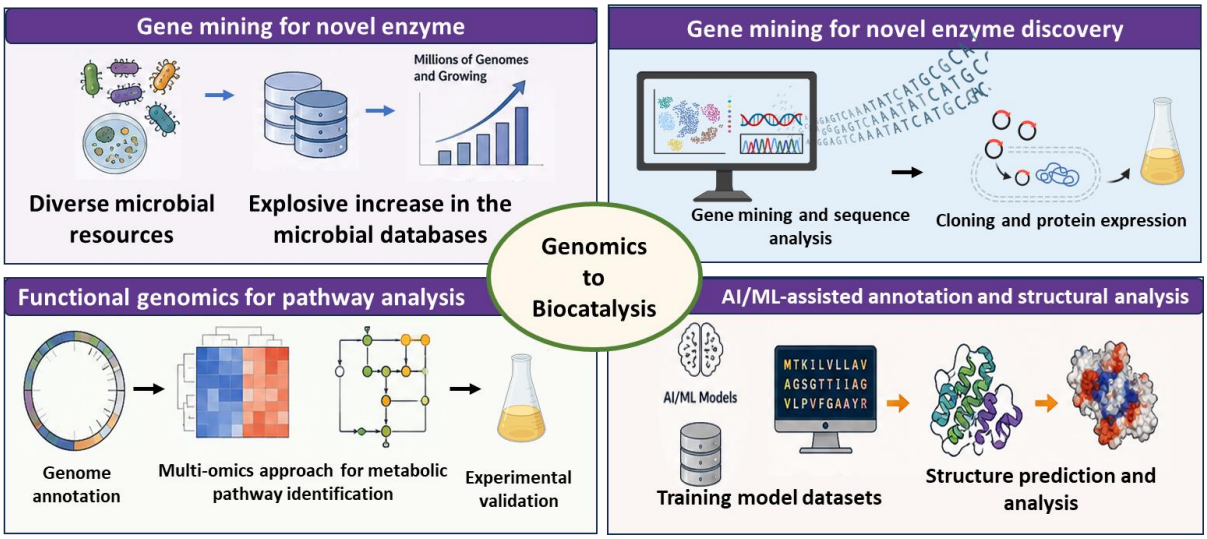


Figure 1: Integrated workflow for genome mining, functional genomics, and AI-assisted analyses highlights the significance of multi-layered attributes of microbial genomics studies in biocatalyst development.

## References


1. Woese CR, Kandler O, Wheelis ML. Towards a natural system of organisms: proposal for the domains Archaea, Bacteria, and Eucarya. Proceedings of the National Academy of Sciences of the USA. 1990;87(12):4576-4579.

2. Woese CR. The universal ancestor. Proceedings of the National Academy of Sciences of the USA. 1998;95(12):6854-6859.

3. Venter JC, et al. The sequence of the human genome. Science. 2001;291(5507):1304-1351.

4. Handelsman J. Metagenomics: application of genomics to uncultured microorganisms. Microbiology and Molecular Biology Reviews. 2004;68(4):669-685.

5. Patel SN, Kaushal G, Singh SP. D-allulose 3-epimerase of *Bacillus* sp. origin manifests profuse heat-stability and noteworthy potential of D-fructose epimerization. *Microbial Cell Factories*. 2021;20(1):60

6. Simon C, Daniel R. Metagenomic analyses: past and future trends. Applied and Environmental Microbiology. 2011;77(4):1153-1161.

7. Van der Oost J, et al. New opportunities for genome mining and biocatalyst discovery. *Current Opinion in Biotechnology*. 2014;29:1-8.

8. Blin K, Shaw S, Steinke K, et al. antiSMASH 6.0: improving cluster detection and comparison capabilities. Nucleic Acids Research. 2021;49(W1):W29-W35.

9. Choi KR, et al. Systems metabolic engineering strategies: integrating systems and synthetic biology. *Trends in Biotechnology*. 2019;37:817-837.

10. Jumper J, Evans R, Pritzel A, et al. Highly accurate protein structure prediction with AlphaFold. Nature. 2021;596(7873):583-589.

## Article 2

# Early-Life Exposure: How a Waterborne Toxicant Modifies the Epigenome to Seed Metabolic Diseases

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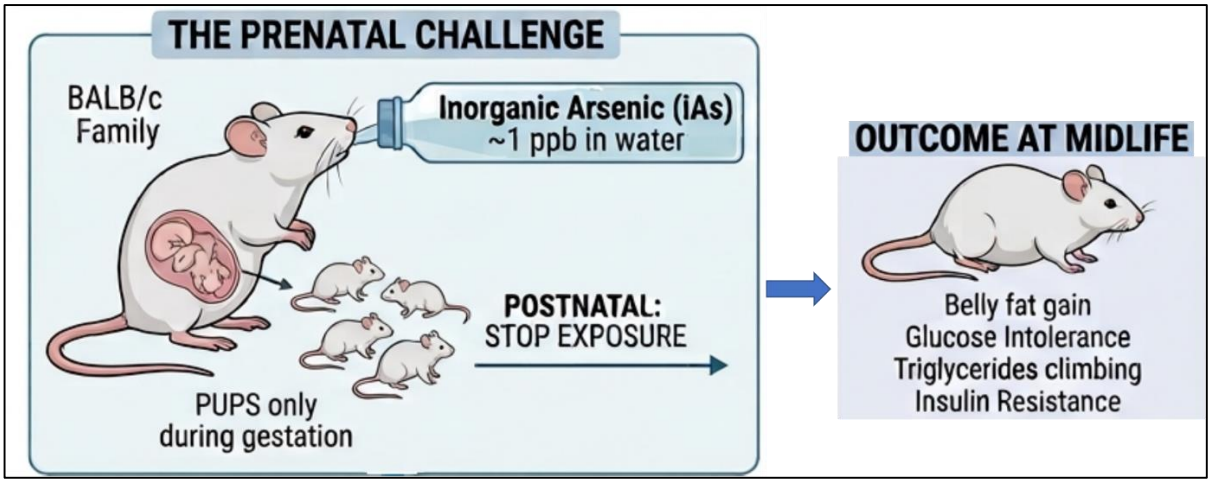
*The studies trace metabolic syndrome back to the womb — and to two separately corrupted layers of epigenetic control.*

Metabolic syndrome looks like a disease of midlife: belly fat that won't shift, fasting glucose that creeps up, triglycerides climbing year on year. The studies here make a different case. For some individuals the opening move is played before birth, and the culprit, may have been sitting in their mother's drinking water.

Arsenic contaminates groundwater across the Indian subcontinent and much of the world; more than 230 million people drink water above the WHO safety limit of 10 ppb. Most toxicology has studied arsenic at high doses in already-born adults but the studies here did the opposite. Female pregnant BALB/c mice received arsenic at a dose modelled on what people actually drink from contaminated wells. Their offspring were never exposed to arsenic. By 20 weeks, roughly midlife for a mouse, the offspring were heavier, fatter, glucose-intolerant and insulin-resistant. They had assembled a full metabolic syndrome on the strength of a prenatal exposure alone. The effect was male-specific, with female littermates largely protected, consistent with the known buffering role of estrogen. It also appeared in BALB/c, a strain not predisposed to metabolic disease, which tells us the programming is robust rather than a quirk of susceptible genetics.




This is the ***Developmental Origins of Health and Disease*** principle in action. But the principle raises an immediate question for anyone who works in genetics: if the offspring carry no new mutations and no ongoing exposure, what is doing the remembering? In both studies the answer is the epigenome. And arsenic corrupts it in two entirely different ways, in two different cell populations, within the same fat pad.

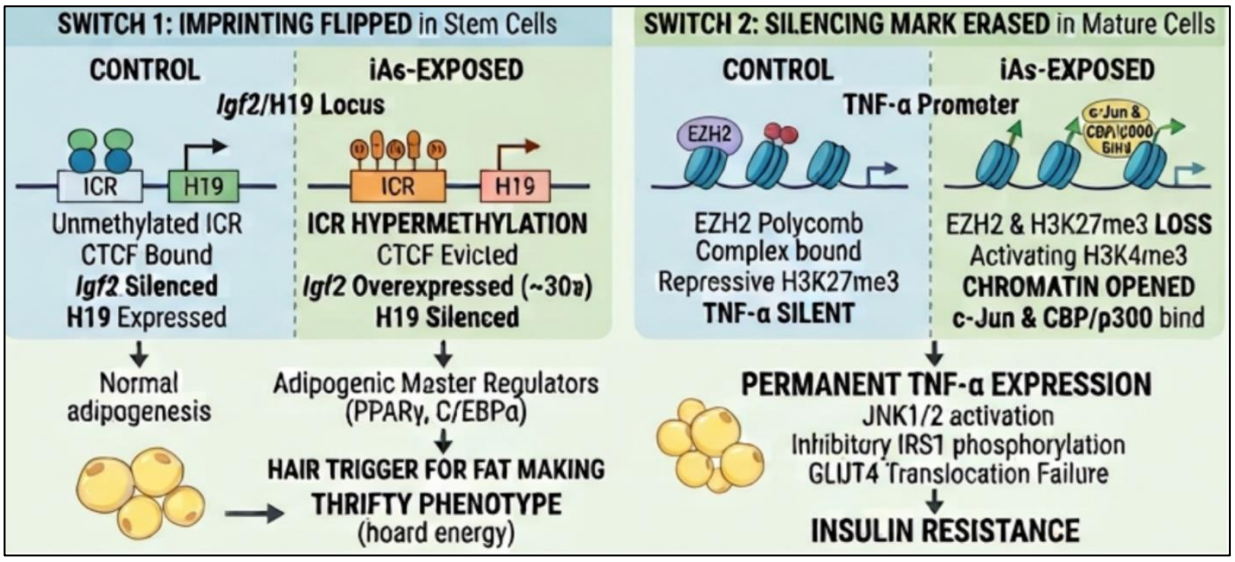
***An imprinting switch, flipped in the stem cells***

The first study follows the adipose-derived stem cells — the progenitor pool that renews fat tissue across the lifespan. Stem cells taken from arsenic-exposed pups divided faster and turned into lipid-loaded adipocytes far sooner than control cells, as though set on a hair trigger for making fat. At the centre sat IGF2 — a growth factor and one of the most studied imprinted genes in the mammalian genome. The Igf2/H19 locus is governed by an imprinting control region (ICR) whose methylation state decides which of two genes the shared enhancers reach. When the insulator protein CTCF binds the unmethylated ICR, it shields Igf2 and allows H19 to be expressed instead. Arsenic drove DNA hypermethylation across this control region, evicting CTCF and switching Igf2 on — overexpressed roughly thirty-fold in the stem cells, with H19 correspondingly silenced. Downstream, real-time gene expression profiling confirmed the breadth of transcriptional reprogramming: adipogenic master regulators PPARγ, CEBPα, FABP4, and AdipoQ were all significantly upregulated, sketching a genome-wide shift in stem cell identity toward a constitutively adipogenic state. The consequence was constitutive firing of Akt, ERK and p38 MAPK pathways: a stem cell pre-committed to a lifetime of excess adipogenesis — in the vocabulary of metabolic programming, cells that had taken on a thrifty phenotype, built to hoard energy against a famine that never came.

***A silencing mark, erased in the mature cells***

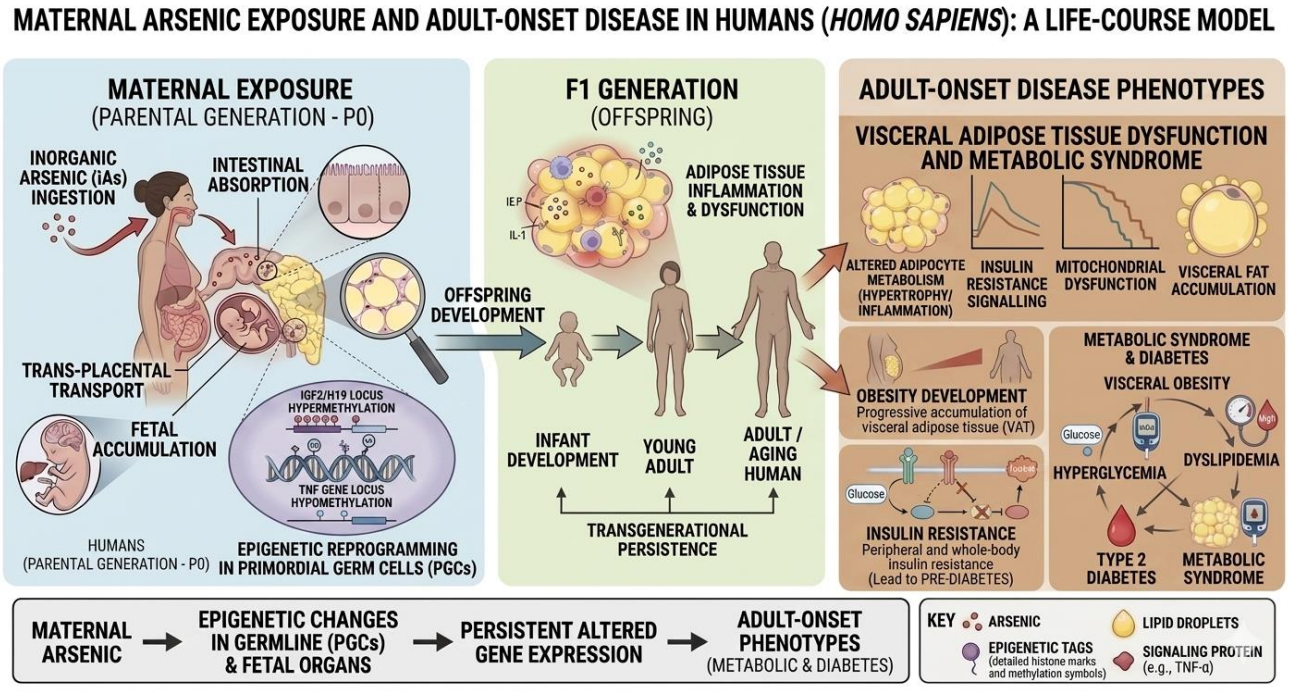
The second study turns to working adipocytes and to inflammation. The offending gene was TNF-α, persistently overexpressed from gestational day 15.5 onward and still elevated at six and twenty weeks. Chronic TNF-α drove JNK1/2 activation, inhibitory phosphorylation of IRS1, and failure of GLUT4 to translocate to the membrane — the textbook molecular route to insulin resistance. Blocking TNF-α pharmacologically restored insulin signalling, identifying the cytokine as the central driver.The mechanism belonged to a different epigenetic system. TNF-α is normally held silent by Polycomb Repressive Complex 2 through its enzyme EZH2, which lays down the repressive histone mark H3K27me3. Arsenic stripped EZH2 from the TNF-α promoter; H3K27me3 fell while the activating mark H3K4me3 rose simultaneously — a transition from poised repression to stable activation that

represents the collapse of a bivalent chromatin domain. Into that opened chromatin came c-Jun and the co-activator CBP/p300, assembling an enhanceosome that locked TNF-α into permanent expression.




***Two lesions, one disease***

For a genetics and genomics audience, the pairing is the part worth lingering on. One study shows a gain of DNA methylation dismantling an imprinting insulator; the other shows a loss of histone methylation dismantling Polycomb silencing. Different machinery, opposite directions of change, different cell types — yet both converge on the same outcome: a depot that grows too eagerly and broadcasts an inflammation it cannot switch off.




For our community specifically, both loci — the Igf2/H19 ICR methylation landscape and the EZH2-H3K27me3 axis at the TNF-α promoter — represent tractable candidate regions for epigenome-wide association studies in arsenic-exposed human cohorts. Their stability across developmental time makes them viable candidates as accessible biomarkers of prenatal arsenic burden

and metabolic risk, a question that now demands urgent investigation in India's arsenic-endemic regions. There is a measure of hope in that picture. ***Mutations are permanent; epigenetic marks are not.*** CTCF occupancy, DNA methylation, EZH2 activity and histone modifications are all, in principle, druggable — which is why the same lesions that explain the disease also nominate targets for preventing or reversing it.

**The studies**


- Koshta K, Chauhan A, Singh S, Gaikwad AN, Kumar M, Srivastava V. *Altered Igf2 imprint leads to accelerated adipogenesis and early onset of metabolic syndrome in male mice following gestational arsenic exposure.* Chemosphere. 2024;352:141493.
- Koshta K, Chauhan A, Singh S, Srivastava V. *Prenatal arsenic exposure alters EZH2–H3K27me3 occupancy at TNF-α promoter leading to insulin resistance and metabolic syndrome in a mouse model.* Environment International. 2024;190:108929.


## Article 3

# Genetic control of Major Efflux Pump Proteins in Antibiotic-Resistant and Susceptible *Pseudomonas aeruginosa*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*Pseudomonas aeruginosa* is an opportunistic Gram-negative pathogen responsible for a wide range of healthcare-associated infections, especially in immunocompromised individuals and patients with cystic fibrosis. Lister et al. (2009) and Hancock and Speert (2000) state that *P. aeruginosa* is becoming increasingly difficult to treat due to its impressive ability to acquire resistance to many classes of antibiotics including aminoglycosides, quinolones, β-lactams and carbapenems. As a result, the World Health Organization has classified carbapenem-resistant *P. aeruginosa* as a critical priority pathogen for the development of new antimicrobial agents (Tacconelli et al., 2018). *P. aeruginosa* has intrinsic resistance through multiple mechanisms such as the production of AmpC β-lactamase, reduced outer membrane permeability, and multidrug efflux pump systems. Antibiotic exposure can also induce chromosomal mutations that upregulate the expression of efflux pumps, inactivate the OprD porin, and modify the targets of antibiotics leading to multidrug resistance. Efflux pumps are a major contributor to antimicrobial resistance but the degree to which structural variations in these proteins contribute to resistance is poorly understood. This study was to understand the mechanism of developing resistant strain from susceptible one or *vice versa* among the selected strains of *P. aeruginosa*: three wild susceptible (M18, LESB58, and UCBPP-PA14) and three resistant strains (PAO1, DHSO1, PA7). 44 common and 8 uncommon antibiotic resistant genes were identified among these strains. Phylogenetic tree of these uncommon genes showed that an AmpC variant,

*Pseudomonas*-derived cephalosporinases (PDC-1) from strain PAO1 was diversified among the strains of *P. aeruginosa* by few specific amino acid substitutions: PDC-1→PDC-3 by a common single substitution from threonine to alanine (Figure 1). Beta-lactamase AmpC is considered the main source of intrinsic resistance of *Pseudomonas aeruginosa* against antipseudomonal penicillins and cephalosporins (DSH01). Strain LESB58 with PDC-3 known as marker gene for susceptibility was resistant only to ceftazidime (R ≥ 32 mg/L) (Barnes et al., 2018). APH (3) IIa of PA7 had close identity with APH(3)IIb of PA14 and LESB58 (Figure 1). Therefore, phylogenetic tree validated the facts that these genes among these resistant and susceptible strains were transformed from one to another by amino acid substitution during course of evolution.

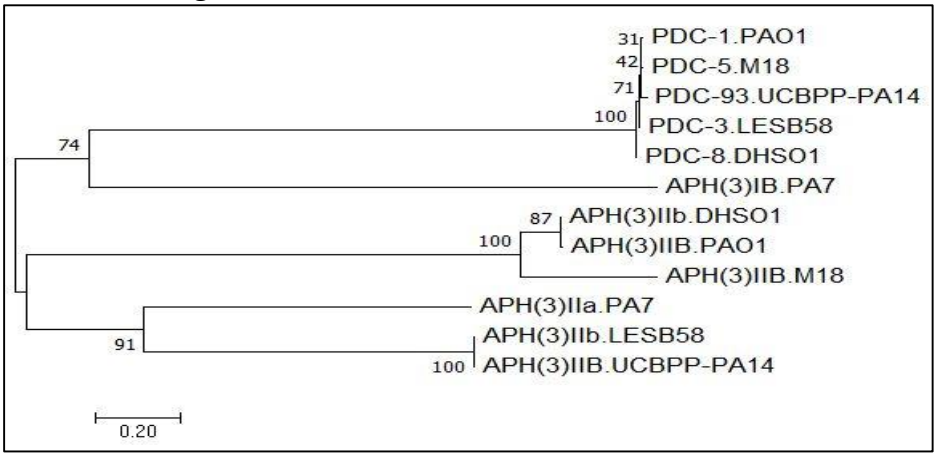


**Figure 1:** Phylogenetic tree of 8 uncommon proteins by Neighbor joining method

All these common proteins from susceptible strains (M18, LESB58 and PA14) and resistant strain (PAO1) showed ≥99% identity and similarity with each other. Out of these 44 common proteins, total 23 proteins from PA7, DSHO1 and LESB58 showing <99% identity each other were selected for considering modeling to view their structural changes in resistant and susceptible strains. Configuration of all these protein models including efflux pump proteins MexA, MexB, MexE, MexF, OprJ, and OprM (Identity > 99%) almost remained unchanged as indicated in table 1. MexE, MexG, MexH, MexL, and MexP., MexE., and MexE, MexF and MexR between PA7 vs. DHSO1., PA7 vs. LESB58., and DHSO1 vs. LESB58, respectively had zero RMSD value.

**Table 1: Identity and similarity percentage of 23 common proteins with RMSD value**

| Protein name | Strain name | | | Identity (%) | Similarity (%) | Score | RMSD |
|---|---|---|---|---|---|---|---|
| | PA7 | DHS01 | LESB58 | | | | |
| *Bcr -1* | Yes | Yes | | 90.1 | 91.8 | 1867.0 | 0.556 |
| *emrE* | Yes | Yes | | 95.5 | 98.2 | 508.0 | 0.002 |
| *MexA* | Yes | Yes | | 99.7 | 100 | 1886.0 | 0.002 |
| *MexB* | Yes | Yes | | 99.2 | 99.6 | 5215.0 | 0.496 |
| *MexC* | Yes | Yes | | 87.6 | 94.8 | 1683.0 | 0.257 |
| *MexD* | Yes | Yes | | 91.0 | 94.5 | 4727.0 | 0.148 |
| *MexE* | Yes | Yes | | 100 | 100 | 2065.0 | 0.00 |
| | Yes | | Yes | 100 | 100 | 2065.0 | 0.00 |
| | | Yes | Yes | 100 | 100 | 2065 | 0.00 |
| *MexF* | Yes | Yes | | 99.3 | 99.4 | 5274 | 0.031 |
| | Yes | | Yes | 99.3 | 99.4 | 5274 | 0.031 |
| | | Yes | Yes | 100 | 100 | 5313 | 0.00 |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| *MexG* | Yes | Yes | | 96.6 | 98.0 | 710.0 | 0.00 |
| *MexH* | Yes | Yes | | 94.1 | 98.9 | 1735 | 0.00 |
| *MexL* | Yes | Yes | | 94.8 | 96.2 | 1017.0 | 0.00 |
| *MexP* | Yes | Yes | | 89.1 | 94.3 | 1721 | 0.00 |
| *MexR* | Yes | Yes | | 95.9 | 97.3 | 712 | 0.043 |
| | | Yes | Yes | 98.6 | 98.6 | 739 | 0.010 |
| | Yes | | Yes | 95.9 | 97.3 | 714 | 0.043 |
| *MexS* | Yes | Yes | | 97.9 | 98.8 | 1714 | 0.317 |
| *MexT* | Yes | Yes | | 99.3 | 99.3 | 1513 | 0.014 |
| *MexV* | Yes | Yes | | 95.2 | 97.1 | 1767 | 0.021 |
| *MexX* | Yes | Yes | | 94.9 | 98.0 | 1862 | 0.049 |
| *MexY* | Yes | Yes | | 96.7 | 98.3 | 5062 | 0.012 |
| *opmB* | yes | yes | | 97.2 | 98.0 | 2425.0 | 0.527 |
| *opmH* | yes | yes | | 96.3 | 98.3 | 2323.0 | 0.026 |
| *oprJ* | yes | yes | | 99.6 | 100.0 | 2352.0 | 0.078 |
| *oprM* | yes | yes | | 99.6 | 100.0 | 2409.0 | 0.001 |
| *oprN* | yes | yes | | 97.5 | 99.6 | 2280.0 | 0.002 |

## Conclusion

Major efflux pump proteins of *Pseudomonas aeruginosa* were highly conserved in resistant and susceptible strains. Minor changes in the sequence did not result in major changes in the structure, suggesting that evolutionary mutations do not affect the integrity of efflux pumps. The results suggest that antibiotic resistance is more likely to be driven by regulatory and functional mechanisms rather than structural changes.

## References


Barnes, MD. et al. (2018) Inactivation of the Pseudomonas-derived cephalosporinase-3 (PDC-3) by relebactam. Antimicrobial agents and chemotherapy **62**, e02406-17

Berrazeg, M., Jeannot, K., Enguéné, V. Y. N., Broutin, I., Loeffert, S., Fournier, D., &Plésiat, P. (2015). Mutations in β-lactamase AmpC increase resistance of *Pseudomonas aeruginosa* isolates to antipseudomonal cephalosporins. Antimicrobial agents and chemotherapy, 59(10), 6248-6255.

Hancock, R. E., & Brinkman, F. S. (2002). Function of Pseudomonas porins in uptake and efflux. Annual Reviews in Microbiology, 56(1), 17-38.

Lister, P. D., Wolter, D. J., & Hanson, N. D. (2009). Antibacterial-resistant *Pseudomonas aeruginosa*: clinical impact and complex regulation of chromosomally encoded resistance mechanisms. *Clinical microbiology reviews*, *22*(4), 582-610.

Petitjean, M. et al., (2017).Genomic characterization of a local epidemic *Pseudomonas aeruginosa* reveals specific features of the widespread clone ST395. Microbial Genomics 3, 1-10

Roy PH. et al. (2010).Complete Genome Sequence of the Multiresistant Taxonomic Outlier *Pseudomonas aeruginosa* PA7. PLoS ONE 5, e8842

Tacconelli, E., Carrara, E., Savoldi, A., Harbarth, S., Mendelson, M., Monnet, D. L., ... & Ouellette, M. (2018). Discovery, research, and development of new antibiotics: the WHO priority list of antibiotic-resistant bacteria and tuberculosis. *The Lancet Infectious Diseases*, *18*(3), 318-327.

## Article 4

# Polyendocrine Metabolic Ovarian Syndrome (PMOS), One Health, Genomics, and Epigenomics: Integrating Clinical, Environmental, and Molecular Perspectives

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### *Introduction*

Polyendocrine Metabolic Ovarian Syndrome (PMOS) extends the conventional concept of polycystic ovary syndrome (PCOS) by recognizing reproductive, endocrine, and metabolic disorders as interconnected manifestations of a complex systems disorder. It integrates ovarian dysfunction with metabolic, thyroid, adrenal, inflammatory, and environmental factors through shared mechanisms including insulin resistance, oxidative stress, mitochondrial dysfunction, immune dysregulation, altered steroidogenesis, and genetic susceptibility (Azziz et al., 2024; Teede et al., 2023; Goodarzi et al., 2023). Genomic and epigenomic evidence highlights interactions between genetic predisposition and environmental exposures, supporting precision medicine and prevention within the One Health framework (Melin et al., 2024; Destoumieux-Garzón et al., 2021).

### *Conceptualizing Polyendocrine Metabolic Ovarian Syndrome*

PMOS is a systemic endocrine-metabolic syndrome characterized by ovarian dysfunction, insulin resistance, obesity, thyroid abnormalities, chronic inflammation, and metabolic dysregulation rather than isolated organ disease (Azziz et al., 2024). Clinically, it combines reproductive abnormalities—including anovulation, infertility, hyperandrogenism, and polycystic ovarian morphology—with metabolic complications such as impaired glucose tolerance, dyslipidemia, hypertension, obesity, non-alcoholic fatty liver disease, and thyroid dysfunction (Teede et al., 2023). Insulin resistance and chronic inflammation promote hyperandrogenism, mitochondrial dysfunction, endothelial damage, and cardiovascular risk (Goodarzi et al., 2023; Melin et al., 2024). Disease heterogeneity reflects interactions among genetic, epigenetic, environmental, and lifestyle factors, supporting personalized diagnosis and management.

### *Genomics and Epigenomics in PMOS*

High-throughput genomic technologies have transformed the understanding of Polyendocrine Metabolic Ovarian Syndrome (PMOS), which is now recognized as a complex polygenic disorder. Genome-wide association studies (GWAS) have identified susceptibility loci involved in ovarian steroidogenesis, gonadotropin signaling, insulin secretion, adipogenesis, inflammation, and energy metabolism. Key genes, including *DENND1A, THADA, LHCGR, FSHR, INSR, YAP1, RAB5B,*

and *HMGA2,* are consistently associated with reproductive and metabolic phenotypes across diverse populations (Goodarzi et al., 2023). Although individual variants confer only modest risk, polygenic risk scores (PRS) integrate cumulative genetic information to improve early risk stratification and support preventive interventions.

Genetic susceptibility alone does not explain the clinical heterogeneity of PMOS. Epigenetic mechanisms—including DNA methylation, histone modifications, chromatin remodeling, and non-coding RNAs—mediate the effects of environmental exposures on gene expression throughout life. Because these changes are potentially reversible, they represent promising targets for preventive and therapeutic interventions (Melin et al., 2024). Maternal obesity, gestational diabetes, poor prenatal nutrition, chronic psychological stress, and exposure to endocrine-disrupting chemicals (EDCs) have been linked to persistent epigenetic alterations affecting insulin signaling, inflammation, ovarian steroidogenesis, mitochondrial function, and adipocyte differentiation. These developmental influences may predispose individuals to metabolic dysfunction long before clinical manifestations emerge, supporting the Developmental Origins of Health and Disease (DOHaD) hypothesis (Barker, 2007; Destoumieux-Garzón et al., 2021).

Among epigenetic mechanisms, DNA methylation is the most extensively studied. Altered methylation profiles in ovarian granulosa cells, adipose tissue, skeletal muscle, liver, and peripheral blood are associated with hyperandrogenism, insulin resistance, ovulatory dysfunction, and impaired mitochondrial function (Melin et al., 2024). Likewise, dysregulated microRNAs—including **miR-21, miR-93, miR-146a,** and **miR-222**—play important roles in post-transcriptional regulation of endocrine pathways and show promise as minimally invasive biomarkers for diagnosis, prognosis, and therapeutic monitoring.

Overall, genomic and epigenomic evidence demonstrates that PMOS results from lifelong interactions between inherited genetic susceptibility and environmental influences rather than isolated genetic defects. This integrated understanding supports the development of precision endocrinology through individualized risk prediction, early prevention, biomarker-guided diagnosis, and targeted therapeutic strategies (Goodarzi et al., 2023; Melin et al., 2024).

### *Mechanistic Pathways, Precision Medicine, and Clinical Translation*

PMOS results from continuous interactions between inherited genetic susceptibility and environmental exposures throughout life. While genetic variants establish baseline risk, environmental factors determine disease onset, severity, and clinical heterogeneity (Goodarzi et al., 2023). The **Developmental Origins of Health and Disease (DOHaD)** hypothesis explains how fetal and early-life exposures induce long-lasting endocrine changes through epigenetic programming (Barker, 2007). Maternal obesity, gestational diabetes, prenatal androgen excess, nutritional deficiencies, chronic stress, and placental dysfunction alter fetal metabolic programming, increasing susceptibility to insulin resistance, obesity, reproductive dysfunction, and cardiometabolic disease in adulthood (Teede et al., 2023). Prenatal androgen exposure further disrupts hypothalamic regulation, pancreatic β-cell

function, adipocyte differentiation, ovarian folliculogenesis, and hepatic glucose metabolism, with epigenetic changes persisting across the lifespan (Melin et al., 2024).

Environmental endocrine-disrupting chemicals (EDCs), including bisphenol A (BPA), bisphenol S (BPS), phthalates, per- and polyfluoroalkyl substances (PFAS), pesticides, and heavy metals, interfere with estrogen, androgen, thyroid, and glucocorticoid signaling, inducing oxidative stress, mitochondrial dysfunction, chronic inflammation, and abnormal DNA methylation (Heindel et al., 2022). Since environmental exposures occur as complex mixtures, the cumulative **exposome** has become a key determinant of endocrine health, highlighting the importance of integrating exposomics with genomics and epigenomics in PMOS research (Melin et al., 2024).

Several interconnected molecular pathways underpin PMOS. **Insulin resistance** is the central metabolic abnormality linking reproductive dysfunction with obesity, hypertension, cardiovascular disease, and type 2 diabetes. Hyperinsulinemia increases ovarian androgen production while reducing hepatic sex hormone-binding globulin, resulting in elevated free testosterone and impaired follicular maturation (Azziz et al., 2024). **Chronic low-grade inflammation**, characterized by elevated IL-6, TNF-α, CRP, MCP-1, and adipokines, further impairs insulin signaling, promotes oxidative stress, disrupts steroidogenesis, and contributes to endothelial dysfunction and cardiovascular risk (Escobar-Morreale, 2018). Simultaneously, **oxidative stress and mitochondrial dysfunction** impair ATP production, granulosa cell viability, oocyte quality, and systemic metabolism, while neuroendocrine dysregulation of the hypothalamic–pituitary–ovarian axis exacerbates ovarian androgen excess (Goodarzi et al., 2023; Melin et al., 2024).

Advances in multi-omics technologies are advancing **precision medicine** by integrating genomic, epigenomic, transcriptomic, metabolomic, and clinical data for individualized diagnosis and treatment (Melin et al., 2024). Polygenic risk scores, epigenetic biomarkers, and artificial intelligence may improve early risk prediction, disease stratification, and therapeutic monitoring (Goodarzi et al., 2023). Nevertheless, multidisciplinary management combining lifestyle modification with individualized pharmacotherapy, including metformin, GLP-1 receptor agonists, anti-androgen therapy, and fertility treatment, remains the cornerstone of PMOS care (Teede et al., 2023). Genomic testing should complement comprehensive clinical evaluation, while greater inclusion of diverse populations is essential for equitable implementation of precision endocrinology.

### *One Health Perspective, Environmental Epigenomics, Public Health Implications, and Research Priorities*

The **One Health** framework provides an integrated perspective on Polyendocrine Metabolic Ovarian Syndrome (PMOS) by recognizing that endocrine health is shaped by interactions among humans, animals, and the environment. Beyond genetics and lifestyle, environmental degradation, pollution, biodiversity loss, climate change, and changing food systems influence endocrine homeostasis across species (Destoumieux-Garzón et al., 2021). Endocrine-disrupting chemicals (EDCs), including bisphenols, phthalates, persistent organic pollutants (POPs), per- and polyfluoroalkyl substances (PFAS), pesticides, dioxins, and heavy metals, are associated with ovarian dysfunction, thyroid

disorders, insulin resistance, obesity, infertility, and adverse pregnancy outcomes, highlighting the need for stronger environmental policies (Heindel et al., 2022).

Environmental factors contribute to PMOS through epigenetic mechanisms—including DNA methylation, histone modification, chromatin remodeling, and microRNA regulation—that alter gene expression without changing DNA sequences (Melin et al., 2024). Cross-species evidence demonstrates that wildlife and livestock exposed to EDCs develop reproductive and metabolic abnormalities comparable to those in humans, supporting environmental monitoring as an early warning system (Heindel et al., 2022). Furthermore, prenatal EDC exposure may induce persistent, potentially transgenerational epigenetic changes affecting endocrine and metabolic regulation. Together, these findings underscore that PMOS results from interactions among genetic susceptibility, environmental exposures, and developmental programming, emphasizing environmental protection alongside precision medicine (Heindel et al., 2022; Melin et al., 2024).

**Public Health Implications -** The rising global prevalence of obesity, metabolic syndrome, insulin resistance, infertility, and endocrine disorders highlights the need for life-course, population-based prevention of PMOS. Prevention should begin before conception by optimizing maternal nutrition, preventing obesity, managing gestational diabetes, promoting smoking and alcohol cessation, ensuring adequate micronutrient intake, and maintaining healthy gestational weight gain to improve offspring metabolic programming. Lifestyle medicine remains the cornerstone of prevention, emphasizing Mediterranean-style diets, regular physical activity, adequate sleep, stress management, and smoking cessation, which improve insulin sensitivity while reducing inflammation, oxidative stress, and epigenetic alterations (Teede et al., 2023). Reducing exposure to endocrine-disrupting chemicals through healthier environmental practices further supports endocrine health. Comprehensive screening should integrate reproductive, metabolic, and endocrine assessment, enabling early detection of insulin resistance, obesity, thyroid dysfunction, cardiovascular risk factors, and reproductive abnormalities, thereby facilitating personalized management and reducing fragmentation of care.

**Research Priorities -** Despite substantial advances, important questions regarding PMOS remain unresolved. Future research should integrate **multi-omics** approaches—including genomics, epigenomics, transcriptomics, proteomics, metabolomics, lipidomics, microbiomics, and exposomics—with systems biology and artificial intelligence to identify novel disease subtypes, pathogenic pathways, and therapeutic targets. Large longitudinal birth cohort studies are needed to evaluate the long-term effects of maternal nutrition, obesity, endocrine-disrupting chemical exposure, psychosocial stress, microbiome composition, and socioeconomic factors on endocrine health. Precision medicine will depend on validating biomarkers such as DNA methylation signatures, circulating microRNAs, extracellular vesicles, metabolomic, inflammatory, and mitochondrial markers for early diagnosis and treatment monitoring. Artificial intelligence may further improve disease stratification, risk prediction, and personalized therapy. Expanding genomic studies to underrepresented populations and strengthening implementation science—including cost-effectiveness, healthcare infrastructure, clinician education, ethical governance, and policy integration—will be essential for translating precision endocrinology into routine PMOS care.

## References


1. Azziz, R., Carmina, E., Chen, Z., Dunaif, A., Laven, J. S. E., Legro, R. S., Lizneva, D., Natterson-Horowtiz, B., Teede, H. J., & Yildiz, B. O. (2016). Polycystic ovary syndrome. *Nature Reviews Disease Primers, 2*, 16057. https://doi.org/10.1038/nrdp.2016.57
2. Barker, D. J. P. (2007). The origins of the developmental origins theory. *Journal of Internal Medicine, 261*(5), 412–417. https://doi.org/10.1111/j.1365-2796.2007.01809.x
3. Destoumieux-Garzón, D., Mavingui, P., Boetsch, G., Boissier, J., Darriet, F., Duboz, P., Fritsch, C., Giraudoux, P., Le Roux, F., Morand, S., Paillard, C., Pontier, D., Sueur, C., & Voituron, Y. (2021). The One Health concept: 10 years old and a long road ahead. *Frontiers in Veterinary Science, 8*, 676852. https://doi.org/10.3389/fvets.2021.676852
4. Escobar-Morreale, H. F. (2018). Polycystic ovary syndrome: Definition, aetiology, diagnosis and treatment. *Nature Reviews Endocrinology, 14*(5), 270–284. https://doi.org/10.1038/nrendo.2018.24
5. Goodarzi, M. O., Dumesic, D. A., Chazenbalk, G., & Azziz, R. (2023). Polycystic ovary syndrome: Etiology, pathogenesis and diagnosis. *Nature Reviews Endocrinology, 19*, 321–339. *(Use the final published bibliographic details for your target journal format if required.)*
6. Heindel, J. J., Blumberg, B., Cave, M., Machtinger, R., Mantovani, A., Mendez, M. A., Nadal, A., Palanza, P., Panzica, G., Sargis, R., Vandenberg, L. N., & vom Saal, F. S. (2022). Metabolism disrupting chemicals and metabolic disorders. *Nature Reviews Endocrinology, 18*(6), 353–367. https://doi.org/10.1038/s41574-022-00683-0
7. Melin, J. M., Forslund, M., Alesi, S. J., et al. (2024). Effects of different insulin sensitisers in the management of polycystic ovary syndrome: A systematic review and meta-analysis. *Clinical Endocrinology, 100*(2), 149–163. https://doi.org/10.1111/cen.14983
8. Stener-Victorin, E., Teede, H. J., Norman, R. J., Legro, R. S., Goodarzi, M. O., Dokras, A., Laven, J. S. E., Hoeger, K. M., & Piltonen, T. T. (2024). Polycystic ovary syndrome. *Nature Reviews Disease Primers, 10*, Article 27. https://doi.org/10.1038/s41572-024-00511-3
9. Teede, H. J., Tay, C. T., Laven, J. S. E., Dokras, A., Moran, L. J., Piltonen, T. T., Costello, M. F., Boivin, J., Redman, L. M., Boyle, J. A., Norman, R. J., Mousa, A., Joham, A. E., & International PCOS Network. (2023). Recommendations from the 2023 International Evidence-based Guideline for the Assessment and Management of Polycystic Ovary Syndrome. *Human Reproduction, 38*(9), 1655–1679. https://doi.org/10.1093/humrep/dead156
10. Teede, H. J., Mousa, A., Tay, C. T., Costello, M. F., Brennan, L., Norman, R. J., Peña, A. S., Boyle, J. A., Joham, A. E., Berry, L., & Moran, L. J. (2024). Summary of the 2023 international evidence-based guideline for the assessment and management of polycystic ovary syndrome: An Australian perspective. *Medical Journal of Australia, 221*(7), 389–395. https://doi.org/10.5694/mja2.52432

# Announcements

**1. Webinar Series**

- The **Second webinar** was held on **April 15th, 2026**, with **Prof. (Dr) Ashwin Dalal, Head & Staff Scientist VII,** Diagnostic Division, BRIC-Centre for DNA Fingerprinting & Diagnostics (CDFD), Hyderabad, India delivering a lecture entitled *"***Harnessing Multi-Omics Approaches in Diagnostics and Research on Rare Genetic Diseases***"*.

**2. Membership of AICGG**

## i. List of New Life members of AICGG (April 2026 on wards)

| Name | Year of Joining | Designation and Affiliations |
|---|---|---|
| **DR. MAINAK SENGUPTA** | **2026** | Assistant Professor, Grade III,<br>Dept. of Genetics, University of Calcutta.<br>sengupta.mainak@gmail.com,<br>msgntcs@caluniv.ac.in |
| **DR. SUBHALAKSHMI GHOSH** | **2026** | Founder & Director, SUBHAMI BIOPHARMA, Co-Founder & Director, Alona LS subhalakshmi.ghosh@gmail.com |
| **DR. ANJALIKA ROY** | **2026** | Assistant Professor<br>Visva Bharati University<br>Email ID: roy.anjalika@rediffmail.com |
| **PROF. HOSSAIN ALI MONDAL** | **2026** | Department of Genetics and Plant Breeding at the College of Post graduate Studies in Agricultural Sciences, Meghalaya under the Central Agricultural University,Imphal.<br>E mail : hossainalimondal@gmail.com |
| **DR. ASMITA SAMADDAR** | **2026** | Assistant Professor<br>Kalyani University<br>Email ID:- asmitazoo19@klyuniv.ac.in |
| **DR. KRISHNENDU GHOSH** | **2026** | Senior Post Doctoral Research Scholar,<br>University of Iowa<br>krishnendughosh110@gmail.com |

## ii. Many Young researchers joined the AICGG family as Annual members as a part of our membership drive

| Sl No | Name | Joining on | Organization & Email Address |
|---|---|---|---|
| 1. | **Ms Sonu Sharma** | May,2026 | CSIR-Indian Institute of Toxicology Research sousharma967082@gmail.com |
| **2.** | **Mr Reetish Raj Sahoo** | April,2026 | University of Calcutta reetishrajsahoo16@gmail.com |
| **3.** | **Ms Nilanjana Saha** | April,2026 | University of Calcutta nilanjanaphdcu@gmail.com |
| **4.** | **Ms Anwesha Acharyya** | April, 2026 | University of Calcutta asmii1396@gmail.com |
| **5.** | **Mr Indranil Rakshit** | April, 2026 | University of Calcutta Irenvs_rs@caluniv.ac.in |
| **6.** | **Ms Sudeshna Mandal** | May, 2026 | University of Calcutta sudeshnamandal2023@gmail.com |
| **7.** | **Ms Sunandini Ghosh** | May, 2026 | University of Calcutta sunandini09ghosh@gmail.com |
| **8.** | **Mr Gobardhan Halder** | May, 2026 | University of Calcutta gobardhanhalder@gmail.com |
| **9.** | **Mr Samir Makhal** | May, 2026 | University of Calcutta searchsamir@gmail.com |
| **10.** | **Mr Soumen Dey** | May, 2026 | University of Calcutta deysofficial.me@gmail.com |
| **11.** | **Dr Mahasweta Chatterjee** | May, 2026 | Manovikas Kendra, Kolkata mahasweta@manovikaskendra.org |
| **12.** | **Dr Sharmistha Saha** | May, 2026 | Manovikas Kendra, Kolkata sharmistha@manovikaskendra.org |
| **13.** | **Ms Pallabi Adak** | May, 2026 | Manovikas Kendra, Kolkata pallabiadak007@gmail.com |
| **14.** | **Mr Samidh Sankar Chakraborty** | May, 2026 | Manovikas Kendra, Kolkata samidhschakraborty@gmail.com |
| **15.** | **Ms Nilanjana Dutta** | May, 2026 | Manovikas Kendra, Kolkata nilanjanadutta@gmail.com |
| **16.** | **Ms Chandrani Mukherjee** | May, 2026 | University of Calcutta mukherjeechandrani2015@gmail.com |
| **17.** | **Ms Saumya Pramanik** | May, 2026 | University of Calcutta saumyapramanik05@gmail.com |

| | | | |
|---|---|---|---|
| **18.** | **Ms Pritha Chakraborti** | May,2026 | University of Calcutta<br>pritha0603pc@gmail.com |
| **19.** | **Mr Ushnish Roy** | May, 2026 | University of Calcutta<br>ushnishroyy@gmail.com |
| **20.** | **Ms Devendri Khantwal** | June, 2026 | CSIR-Indian Institute of Toxicology Research<br>Kantwaldevendri01@gmail.com |
| **21.** | **Km Priya** | June, 2026 | CSIR-Indian Institute of Toxicology Research<br>priarair1102@gmail.com |
| **22.** | **Mr Sandip Chatterjee** | May, 2026 | CSIR-Indian Institute of Toxicology Research<br>sandip365g@gmail.com |
| **23.** | **Ms Debava Chaudhuri** | May, 2026 | St. Xavier's College (Autonomous), Kolkata<br>debavachaudhuri@sxccal.edu |
| **24.** | **Ms Nikkita Das** | May, 2026 | St. Xavier's College (Autonomous), Kolkata<br>dasnikkita44@gmail.com |
| **25.** | **Dr. Souradip Basu** | May, 2026 | University of Engineering and Management, Kolkata<br>souradip.basu@iem.edu.in |
| **26.** | **Mr. Wrick Chakraborty** | May, 2026 | St. Xavier's College (Autonomous), Kolkata<br>wrickchakraborty@sxccal.edu |
| **27.** | **Mr. Indranil Biswas** | May, 2026 | St. Xavier's College (Autonomous), Kolkata<br>indranilbiseas.academic@gmail.com |

**We warmly welcome all new members into the AICGG family.**

## Acknowledgement

We gratefully acknowledge the generous contribution of **Dr. Ashok K. Giri,** former Secretary-Convenor of AICGG, who has kindly donated a sum of **₹ 3,00,000 (INR Three Lacs**) to support the organization. His thoughtful gesture will greatly strengthen our mission and activities.

# Invitation for Articles

**Invitation for the quarterly e-Newsletter AICGG**

We warmly invite eminent researchers, scientists, professors, and distinguished members to contribute scholarly articles aligned with the theme of our newsletter ***Genomic Horizons***. Contributions may take the form of concise scientific articles, **typically one to two pages in** length (**1000 to 1500 words**), that reflect ongoing research, insights, or perspectives relevant to our field.

**Format for articles (Word Doc, Times New Roman, Font size 12)**

***Theme:*** **"Genetic Frontiers in Disease Prevention and Cure"**

***Title:***

***Name of author (s):***

***Affiliations:***

***Email Address of Corresponding author:***

***Article Body:*** In addition to the text, figures/images/tables/schematic diagrams may be included as relevant

***References: 10 (maximum)***

**Articles should reach us by 15th September 2026** for the next issue to be published in **October 2026.**

**Email address for sending articles**: 2006.nilanjana@gmail.com and vikas.srivastava@csir.res.in

**"We are all 99.9% genetically identical, yet that 0.1% makes us unique." – Francis Collins**