

Review Article

Beyond the Powerhouse – Unveiling Hidden Influence of Mitochondria on Reproductive Health and Fertility

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ABSTRACT

Mitochondria, recognised mainly for their role in adenosine triphosphate (ATP) production, are now understood to be pivotal regulators of reproductive function, extending their influence to gametogenesis, fertilisation, and early embryonic development. In males, mitochondrial dysfunction and excessive reactive oxygen species (ROS) generation compromise sperm motility, deoxyribonucleic acid (DNA) integrity, and fertilisation potential, contributing to infertility. Similarly, in females, proper mitochondrial activity is essential for oocyte maturation and embryonic viability, with mitochondrial DNA (mtDNA) abnormalities increasingly associated with ovarian ageing and diminished fertility. Emerging evidence also highlights the role of mitochondrial epigenetics, such as mtDNA methylation, non-coding ribonucleic acid (RNA) regulation, and retrograde signalling, in modulating reproductive outcomes. Disruptions to these pathways from ageing, metabolic disorders, environmental stressors, or assisted reproductive technologies (ART) can impair fertility in both sexes. Promising therapeutic strategies, including mitochondrial transfer, antioxidant supplementation, and modulation of mitochondrial dynamics, are still under investigation. A deeper understanding of mitochondrial function and its epigenetic interactions offers novel avenues for the diagnosis and treatment of infertility.

Keywords: Epigenetics, Infertility, Mitochondria, Oocyte, Spermatozoa

INTRODUCTION

Mitochondria are double-membrane organelles composed of an outer mitochondrial membrane (OMM) and an inner mitochondrial membrane (IMM), which encloses the intermembrane space and the mitochondrial matrix. The IMM forms invaginations known as cristae, which are specialised structures crucial for adenosine triphosphate (ATP) production through oxidative phosphorylation. In mature mammalian spermatozoa, ~72-80 mitochondria are concentrated in the midpiece, reflecting their essential role in energy supply and sperm motility.^[1] The morphology and abundance of mitochondria vary significantly across different cell types and are closely aligned with their metabolic requirements.^[2]

Historically viewed as mere cellular powerhouses, mitochondria are now recognised as multifunctional organelles that extend their influence well beyond energy metabolism. Recent

advances in reproductive biology have unveiled their critical involvement in gametogenesis, fertilisation, and early embryogenesis. In addition to ATP synthesis, mitochondria serve as signalling centres, regulators of calcium homeostasis, and epigenetic modulators influencing gene expression.^[3] These organelles are remarkably dynamic, capable of movement, fusion, and fission, and they communicate with each other via structures such as mitochondrial nanotunnels, regulated by proteins like mitofusin-2 and dynamin-related proteins, to meet fluctuating cellular energy demands. Furthermore, mitochondria influence nuclear gene expression through metabolite-mediated epigenetic modifications, underscoring their role in cellular signalling and developmental regulation.^[4-6]

Mitochondrial dysfunction has been implicated in various reproductive disorders, affecting both male and female fertility. This evolving understanding marks a paradigm shift, emphasising that infertility may arise not merely from energy deficiency but also from complex mitochondrial dysregulation. As such, mitochondria represent promising targets for the development of diagnostic biomarkers and therapeutic strategies aimed at improving reproductive outcomes.

Mitochondria's role in male fertility

Mitochondrial functionality, particularly the maintenance of an intact mitochondrial membrane potential, is a prerequisite for optimal sperm function and overall semen quality. Mitochondria supply the ATP necessary for sperm motility, with the dynein protein's ATPase activity confirming the essential role of ATP in driving flagellar movement. In addition to powering motility, mitochondria contribute to key reproductive processes such as sperm capacitation and fertilisation.^[7]

Mitochondrial quality is preserved through tightly regulated quality control mechanisms, including fission and fusion processes, collectively referred to as mitochondrial dynamics. These dynamic events allow mitochondrial populations to mix their contents, ensuring functional homogeneity and adaptability to metabolic demands. Mitochondrial dynamics are particularly critical during spermatogenesis and meiosis, where energy needs and metabolic states fluctuate rapidly.^[8,9]

Additionally, mitophagy, the selective degradation of damaged mitochondria via autophagy,^[10] serves as a vital process to maintain cellular homeostasis and prevent the accumulation of dysfunctional mitochondria in developing germ cells.

The coordination of mitochondrial dynamics and turnover is intricately linked to the metabolic requirements of germ cells as they navigate the compartmentalised architecture of the seminiferous epithelium.^[11] Any disruption to these finely tuned processes, whether from genetic disorders, metabolic diseases, or environmental toxins, can lead to mitochondrial

dysfunction and consequent infertility in males.

Reactive oxygen species (ROS) formation occurs primarily in mitochondrial complexes I and II residing in the mid-piece of spermatozoa. Although ROS plays a role in normal cellular signalling, an imbalance, characterised by excessive ROS production alongside insufficient antioxidant defences, can be detrimental to sperm cells.^[12] Due to the high concentration of polyunsaturated fatty acids in sperm membranes, excessive ROS readily induces lipid peroxidation, resulting in impaired spermatogenesis, mitochondrial dysfunction, and apoptosis.^[13] These effects significantly compromise male fertility.

Mitochondrial DNA (mtDNA) is especially vulnerable to oxidative damage, given its proximity to the electron transport chain and lack of protective histones. Oxidative stress not only disrupts mitochondrial integrity but also leads to reduced sperm motility, lower fertilising capacity, and increased susceptibility to programmed cell death.^[14] Protective mechanisms, such as the expression of mitochondrial uncoupling protein 2 (UCP2), help modulate ROS levels and maintain sperm quality under stress.^[15] However, when mitochondrial function is severely impaired or mtDNA accumulates mutations, there is often an increase in mtDNA copy number alongside reduced mtDNA integrity, both of which are associated with poor sperm quality.^[16]

Such mitochondrial impairments can manifest clinically as asthenozoospermia (reduced sperm motility), oligospermia (low sperm count), and teratozoospermia (abnormal sperm morphology), ultimately contributing to male infertility.^[17-20]

Mitochondria's role in female fertility

Among all cell types, the oocyte contains the highest number of mitochondria and mitochondrial DNA (mtDNA) copies per cell.^[21] These organelles are vital for fulfilling the substantial energy requirements of the oocyte, particularly during processes such as meiotic maturation, fertilisation, and early embryonic development. During oocyte growth and maturation, mtDNA copy number increases dramatically to support the energy needs of subsequent developmental stages.^[22] Proper mitochondrial function is essential for oocyte maturation and the acquisition of developmental competence.^[23] Disruptions in mitochondrial dynamics or deviations in mtDNA content and expression are associated with diminished ovarian reserve, impaired oocyte quality, and poor reproductive outcomes. Dysfunctional mitochondria or abnormal mtDNA expression are directly linked to conditions such as ovarian insufficiency and implantation failure. Oocytes harbouring deleterious mtDNA mutations are often eliminated via follicular atresia, a process believed to contribute to reduced ovarian reserve in some individuals.^[24]

With age, the accumulation of mtDNA mutations in oocytes

contributes significantly to ovarian ageing and a decline in fertility.^[25,26] A low mtDNA copy number within oocytes has been associated with fertilisation failure and aberrant embryonic development.^[21,27,28]

To address mitochondrial dysfunction and improve oocyte quality, especially in advanced age or those experiencing recurrent *in vitro* fertilization (IVF) failure, various mitochondrial transfer techniques are under investigation. These include both autologous and heterologous approaches, though the optimal strategy remains under refinement.^[26,29] In pigs, mitochondrial transfer from developmentally competent oocytes has been shown to enhance oocyte competence significantly.^[30] Similarly, the transfer of mitochondria derived from surrounding follicular cells has shown promise in improving oocyte quality in bovine and human models. However, the efficacy and safety of mitochondrial transfer techniques are not yet fully established, and additional research is needed to determine their long-term outcomes and clinical viability.^[31]

Mitochondrial epigenetics

Mitochondrial DNA (mtDNA), though structurally distinct from nuclear DNA, being circular, maternally inherited, and histone-free, undergoes epigenetic-type modifications similar to those seen in the nucleus. These include DNA methylation, hydroxymethylation, and alterations in nucleoid-associated protein structure, all of which influence mitochondrial transcription and replication.^[32,33] Such modifications are critical, as they directly affect mitochondrial functions like energy production, redox balance, and apoptosis, processes fundamental to gametogenesis and early embryonic development.^[25]

Factors such as ageing, excess body weight, and metabolic disorders can compromise mtDNA integrity in oocytes, leading to mutations, a reduced mtDNA copy number, and impaired ATP synthesis. These mitochondrial defects may result in abnormal spindle formation and diminished embryo viability.^[34,35] Additionally, mitochondrial dysfunction in granulosa cells has been shown to alter mitochondrial gene expression and biogenesis, impairing folliculogenesis and ovulatory processes.^[32]

Emerging evidence suggests that assisted reproductive technologies (ART) may influence mitochondrial epigenetic landscapes, potentially exerting long-term effects on the health of offspring.^[35] In sperm, epigenetic alterations in mtDNA, such as methylation patterns, oxidative stress-induced damage, and dysregulation of non-coding RNAs, have been associated with male infertility conditions like asthenozoospermia and oligo-astheno-zoospermia, both of which impair fertilisation capacity.^[36]

Although paternal mitochondria are typically eliminated after fertilisation, their epigenetic state prior to this event may still influence embryonic development through retrograde signalling. This mitochondria-to-nucleus communication can modify nuclear gene expression by altering DNA methylation, histone modifications, and non-coding RNA activity in germ cells, thereby disrupting transcriptional regulation.^[37,38]

Environmental stressors, including toxins, poor nutrition, and metabolic imbalances, can exacerbate mitochondrial dysfunction, adversely impacting fertility in both males and females.^[39]

Current research is increasingly focused on therapeutic interventions aimed at restoring mitochondrial function. These include the use of antioxidants such as Coenzyme Q10, sirtuin (SIRT) pathway modulators, and mitochondrial transfer techniques, all of which hold promise for enhancing reproductive outcomes.^[35,40] Figure 1 shows the involvement of mitochondria in sperm physiology, oocyte maturation, and early embryonic development. The mitochondrial production of ATP and the electron transport chain have roles in sperm motility, capacitation, and meiotic maturation, while overproduction of ROS due to ageing, stress, and metabolic disease damages mtDNA, causing apoptosis and impairing embryo development and fertilisation.

Understanding the complex interplay between mitochondrial and nuclear epigenetics opens promising avenues for diagnosing, preventing, and treating infertility. As research in this area deepens, it may unlock targeted therapeutic strategies to improve reproductive health.

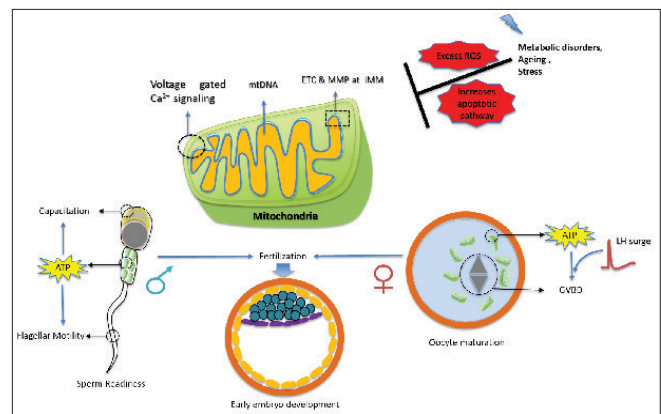


Figure 1: Schematic representation of mitochondrial role in gamete functioning, fertilisation, and embryo development. ETC: electron transport chain, ATP: adenosine triphosphate, IMM: inner mitochondrial membrane, MMP: mitochondrial membrane potential, mtDNA: mitochondrial DNA, ROS: reactive oxygen species, GVBD: germinal vesicle breakdown, LH: luteinising hormone

Clinical significance

From a clinical fertility perspective, mitochondrial integrity

is central to oocyte quality. Age-related mitochondrial dysfunction, characterised by accumulated mtDNA mutations, reduced ATP production, altered calcium homeostasis, and oxidative stress, contributes directly to poor oocyte competence, increased aneuploidy, implantation failure, and miscarriage. This explains why advanced maternal age remains one of the strongest predictors of reduced IVF success and adverse reproductive outcomes.^[41]

Clinically, this understanding has led to novel therapeutic strategies, particularly mitochondrial replacement therapy (MRT). Techniques such as spindle transfer and pronuclear transfer allow replacement of defective mitochondrial cytoplasm while preserving the patient's nuclear genome. This has dual significance: Prevention of transmission of mtDNA disorders, which otherwise have no curative treatment and can cause severe multisystem disease in offspring. Restoration of cytoplasmic competence in infertile patients, especially older women with repeated IVF failure, offering the possibility of genetically related but metabolically rejuvenated embryos. MRT represents a major clinical advancement aimed at preventing the transmission of mtDNA-based diseases and improving fertility outcomes. Techniques such as spindle transfer (ST), pronuclear transfer (PNT), and polar body transfer (PBT) enable the replacement of defective mitochondria while preserving the nuclear genetic identity of the parents. Another critical clinical implication lies in embryo viability and developmental programming. Even low levels of mitochondrial dysfunction can impair early embryonic metabolism, influencing blastocyst quality and long-term health. Experimental and preclinical studies demonstrate that minimising mtDNA carryover below disease thresholds (<2%) is achievable and likely clinically safe, reinforcing the feasibility of MRT as a preventive and therapeutic intervention in reproductive medicine.^[42] Finally, these insights reshape reproductive counselling and ethics. Mitochondria are not passive energy units but active determinants of fertility, inheritance, and offspring health. Clinicians must therefore integrate mitochondrial assessment, age-related mitochondrial decline, and emerging mitochondrial therapies into infertility management and genetic counselling.

Future direction

Future research must pivot from correlation to mechanistic causality, focusing on four interconnected pillars. First, the development of standardised, non-invasive mitochondrial biomarkers, such as cell-free mtDNA mutations in follicular fluid and sperm telomere-mtDNA copy number ratios, is essential for clinical adoption. Second, deciphering the precise bidirectional signalling between mitochondrial metabolites (e.g., acetyl-CoA, α -ketoglutarate) and nuclear epigenetic machinery during gametogenesis will identify

novel therapeutic targets for epigenetic dysregulation. Third, advancing mitochondria-specific therapeutic delivery, including next-generation antioxidants (MitoQ analogues) and safe autologous mitochondrial transfer protocols, requires rigorous preclinical validation to ensure efficacy and long-term safety. Finally, longitudinal clinical studies must integrate these biomarkers and interventions to establish causative links between mitochondrial dysfunction, ART outcomes, and offspring health. This integrated approach will transition mitochondrial science from a diagnostic adjunct to a central pillar of personalised reproductive medicine, enabling targeted interventions that address the root causes of idiopathic infertility.

CONCLUSION

Mitochondria play an indispensable role in reproductive physiology, far beyond their traditional function as cellular powerhouses. Their influence spans from energy metabolism to redox balance, epigenetic regulation, and apoptosis, all of which are critical to the success of gametogenesis, fertilisation, and early embryonic development. Mitochondrial dysfunction, whether due to genetic mutations, oxidative stress, or epigenetic dysregulation, is increasingly recognised as a key contributor to infertility in both males and females. Advancements in our understanding of mitochondrial biology, particularly in the context of reproduction, open new frontiers for diagnostics and therapeutic interventions. Continued research into mitochondrial function and its interplay with nuclear epigenetics is essential for developing innovative, effective strategies to combat infertility and improve reproductive health.

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REFERENCES

1. Freitas MJ, Vijayaraghavan S, Fardilha M. Signaling mechanisms in mammalian sperm motility. *Biology of Reproduction*. 2017;96:2-12.
2. Vafai SB, Mootha VK. Mitochondrial disorders as windows into an ancient organelle. *Nature*. 2012;491:374-83.
3. Friedman JR, Nunnari J. Mitochondrial form and function. *Nature*. 2014;505:335-43.
4. Gut P, Verdin E. The nexus of chromatin regulation and

- intermediary metabolism. *Nature*. 2013;502:489-98.
5. Chandel NS. Mitochondria as signaling organelles. *BMC Biology*. 2014;12:34.
 6. Rossi A, Pizzo P, Filadi R. Calcium, mitochondria and cell metabolism: A functional triangle in bioenergetics. *Biochimica et Biophysica Acta – Molecular Cell Research*. 2019;1866:1068-78.
 7. Barbagallo F, La Vignera S, Cannarella R, Aversa A, Calogero AE, Condorelli RA. Evaluation of sperm mitochondrial function: A key organelle for sperm motility. *Journal of Clinical Medicine*. 2020;9:363.
 8. Chan DC. Fusion and fission: Interlinked processes critical for mitochondrial health. *Annual Review of Genetics*. 2012;46:265-87.
 9. Varuzhanyan G, Rojansky R, Sweredoski MJ, Graham RL, Hess S, Ladinsky MS, *et al.* Mitochondrial fusion is required for spermatogonial differentiation and meiosis. *eLife*. 2019;8:e51601.
 10. Youle RJ, Narendra DP. Mechanisms of mitophagy. *Nature Reviews Molecular Cell Biology*. 2011;12:9-14.
 11. Mishra P, Chan DC. Metabolic regulation of mitochondrial dynamics. *Journal of Cell Biology*. 2016;212:379-87.
 12. Koppers AJ, De Juijls GN, Finnie JM, McLaughlin EA, Aitken RJ. Significance of mitochondrial reactive oxygen species in the generation of oxidative stress in spermatozoa. *Journal of Clinical Endocrinology and Metabolism*. 2008;93:3199-207.
 13. Aitken RJ, Drevet JR. The importance of oxidative stress in determining the functionality of mammalian spermatozoa: A two-edged sword. *Antioxidants*. 2020;9:111.
 14. Nowicka-Bauer K, Nixon B. Molecular changes induced by oxidative stress that impair human sperm motility. *Antioxidants*. 2020;9:134.
 15. Toda C, Diano S. Mitochondrial UCP2 in the central regulation of metabolism. *Best Practice and Research Clinical Endocrinology and Metabolism*. 2014;28:757-64.
 16. Durairajanayagam D, Singh D, Agarwal A, Henkel R. Causes and consequences of sperm mitochondrial dysfunction. *Andrologia*. 2021;53:e13666.
 17. Costa J, Braga PC, Rebelo I, Oliveira PE, Alves MG. Mitochondria quality control and male fertility. *Biology*. 2023;12:827.
 18. Nakada K, Sato A, Yoshida K, Morita T, Tanaka H, Inoue SI, *et al.* Mitochondria-related male infertility. *Proceedings of the National Academy of Sciences of the United States of America*. 2006;103:15148-53.
 19. Park YJ, Pang MG. Mitochondrial functionality in male fertility: From spermatogenesis to fertilization. *Antioxidants*. 2021;10:98.
 20. Mai Z, Yang D, Wang D, Zhang J, Zhou Q, Han B, *et al.* A narrative review of mitochondrial dysfunction and male infertility. *Translational Andrology and Urology*. 2024;13:2134.
 21. Santos TA, El Shourbagy S, John JCS. Mitochondrial content reflects oocyte variability and fertilization outcome. *Fertility and Sterility*. 2006;85:584-91.
 22. Cotterill M, Harris SE, Collado Fernandez E, Lu J, Huntriss JD, Campbell BK, *et al.* The activity and copy number of mitochondrial DNA in ovine oocytes throughout oogenesis in vivo and during oocyte maturation in vitro. *Molecular Human Reproduction*. 2013;19:444-50.
 23. Kirillova A, Smitz JE, Sukhikh GT, Mazunin I. The role of mitochondria in oocyte maturation. *Cells*. 2021;10:2484.
 24. Colnaghi M, Pomiankowski A, Lane N. The need for high-quality oocyte mitochondria at extreme ploidy dictates mammalian germline development. *eLife*. 2021;10:e69344.
 25. Tesarik J, Mendoza-Tesarik R. Mitochondria in human fertility and infertility. *International Journal of Molecular Sciences*. 2023;24:8950.
 26. Cecchino GN, Seli E, da Motta ELA, García-Velasco JA. The role of mitochondrial activity in female fertility and assisted reproductive technologies: Overview and current insights. *Reproductive Biomedicine Online*. 2018;36:686-97.
 27. Reynier P, May-Panloup P, Chretien MF, Morgan CJ, Jean M, Savagner F, *et al.* Mitochondrial DNA content affects the fertilizability of human oocytes. *Molecular Human Reproduction*. 2001;7:425-29.
 28. El Shourbagy SH, Spikings EC, Freitas M, St John JC. Mitochondria directly influence fertilisation outcome in the pig. *Reproduction*. 2006;131:233-45.
 29. Rodriguez-Varela C, Labarta E. Role of mitochondria transfer in infertility: A commentary. *Cells*. 2022;11:1867.
 30. Cagnone GL, Tsai TS, Makanji Y, Matthews P, Gould J, Bonkowski MS, *et al.* Restoration of normal embryogenesis by mitochondrial supplementation in pig oocytes exhibiting mitochondrial DNA deficiency. *Scientific Reports*. 2016;6:23229.
 31. May-Panloup P, Boguenet M, El Hachem H, Bouet PE, Reynier P. Embryo and its mitochondria. *Antioxidants*. 2021;10:139.
 32. Boucret L, Tramon L, Sifer C, Ferré-LHotellier V, Lornage J, Bouet PE, *et al.* Relationship between diminished ovarian reserve and mitochondrial biogenesis in granulosa cells. *Fertility and Sterility*. 2015;104:452-59.
 33. May-Panloup P, Boucret L, Chao de la Barca JM, Desquirit-Dumas V, Ferré-LHotellier V, Morinière C, *et al.* Ovarian ageing: The role of mitochondria in oocytes and follicles. *Human Reproduction Update*. 2016;22:725-43.
 34. Shukla P, Melkani GC. Mitochondrial epigenetic modifications and nuclear-mitochondrial communication: A new dimension towards understanding and attenuating the pathogenesis in women with PCOS. *Reviews in Endocrine and Metabolic Disorders*. 2023;24:317-26.
 35. Ge H, Tollner TL, Hu Z, Dai M, Li X, Guan H, *et al.* The importance of mitochondrial metabolic activity and mitochondrial DNA replication during oocyte maturation in vitro on oocyte quality and subsequent embryo developmental competence. *Molecular Reproduction and Development*. 2012;79:392-401.
 36. Boguenet M, Bouet PE, Spiers A, Reynier P, May-Panloup P. Mitochondria: Their role in spermatozoa and in male infertility. *Human Reproduction Update*. 2021;27:697-719.
 37. Chan DC, Chandel NS, Wallace DC, Germain M. Mitochondrial retrograde signaling and its role in epigenetic reprogramming of gametes. *Reproductive Sciences*. 2020;27:535-45.
 38. Demain LA, Conway GS, Newman WG. Genetics of mitochondrial dysfunction and infertility. *Clinical Genetics*. 2017;91:199-207.
 39. Erdoğan K, Karaer İK, Aydın M, Tatar M, Koç O. Are epigenetic mechanisms and nutrition effective in male and female infertility? *Journal of Nutritional Science*. 2023;12:e32.

40. García-Rodríguez A, Gosálvez J, Agarwal A, Roy R, Sharma R, Gutiérrez-Adán A. Therapeutic strategies targeting mitochondrial dysfunction and epigenetic regulation in male infertility. *Antioxidants*. 2020;9:222.
41. Rasool A, Sachin, Manzoor I, Patil A. Reproductive aging in farm animals: Impacts on fertility and strategies for mitigation: A narrative review. *Asian Pacific Journal of Reproduction*. 2025;14:1-6.
42. Wolf DP, Mitalipov N, Mitalipov S. Mitochondrial replacement therapy in reproductive medicine. *Trends in Molecular Medicine*. 2015;21:68-76.

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