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High glycemic diet-induced mitochondrial dysfunction and female fertility: emerging molecular links between nutrition and reproductive health

Abstract

Female fertility is highly dependent on optimal mitochondrial function, as mitochondria regulate ATP generation, calcium homeostasis, redox signaling, and apoptosis in oocytes (female egg cells).[1,2] Emerging evidence suggests that chronic consumption of high glycemic index (HGI) diets contributes to insulin resistance, oxidative stress, and mitochondrial dysfunction, ultimately impairing reproductive outcomes.[3,4] Elevated postprandial glucose and hyperinsulinemia increase reactive oxygen species (ROS) production, disrupt mitochondrial membrane potential, damage mitochondrial DNA (mtDNA), and impair oocyte maturation.[4,5] These metabolic disturbances are strongly associated with reproductive disorders such as polycystic ovary syndrome (PCOS), anovulation, poor embryo quality, and infertility.[1,6] Furthermore, mitochondrial dysfunction activates apoptotic pathways in granulosa cells and compromises follicular development.[5] Recent clinical and experimental studies indicate that low glycemic dietary interventions may improve insulin sensitivity, androgen profiles, and reproductive outcomes in women with metabolic infertility.[6,7] This review summarizes current evidence linking high glycemic diets to mitochondrial dysfunction and female infertility and highlights potential nutritional strategies targeting mitochondrial health to improve reproductive outcomes.

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Dr Palak Sangal
Department of Nutrition, ECSL Nutrition Consultancy, India

Correspondence: Dr Palak Sangal PhD, Department of Nutrition, ECSL Nutrition Consultancy, India, Tel +7906843294

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Introduction

Infertility is a major global health concern affecting nearly 10–15% of couples worldwide, with female reproductive dysfunction contributing to approximately half of all cases.[8] In recent decades, increasing prevalence of obesity, insulin resistance, metabolic syndrome, and Polycystic Ovary Syndrome has coincided with rising infertility rates, suggesting a strong association between metabolic health and reproductive function.[1,8] While infertility has traditionally been evaluated from hormonal and anatomical perspectives, growing evidence indicates that cellular metabolism and mitochondrial function are equally critical determinants of female reproductive health.[1,9] Oocyte maturation, fertilization, and early embryonic development are highly energy-dependent processes that require efficient mitochondrial activity and adequate ATP production.[9] Oocytes contain exceptionally high mitochondrial content, reflecting their dependence on tightly regulated oxidative phosphorylation and cellular energy homeostasis.[9,10]

Dietary habits have emerged as important modulators of reproductive physiology.[11] Modern diets rich in refined carbohydrates, processed foods, and rapidly absorbable sugars are associated with repeated postprandial glucose and insulin fluctuations, contributing to chronic metabolic stress.[6,12] High glycemic index (HGI) dietary patterns have been linked with obesity, insulin resistance, systemic inflammation, and endocrine dysfunction, all of which may negatively influence ovarian function and fertility.[6,8] Increasing evidence suggests that chronic hyperglycemia and metabolic imbalance can impair mitochondrial integrity and promote oxidative stress within reproductive tissues, thereby affecting oocyte quality and follicular development.[4,13] Mitochondrial dysfunction is increasingly recognized as a key molecular feature underlying several metabolic reproductive disorders.[1,4] Excessive reactive oxygen species (ROS) production, impaired ATP synthesis, altered redox balance, and activation of inflammatory and apoptotic pathways may collectively compromise reproductive competence.[13,14] Understanding the molecular relationship between high glycemic diets, mitochondrial dysfunction, and female fertility may therefore provide important insights into the pathophysiology of infertility and the role of nutrition in reproductive health.

High glycemic diets and metabolic stress

High glycemic index (HGI) diets are characterized by frequent consumption of rapidly absorbable carbohydrates including refined flour products, sugary beverages, desserts, processed cereals, and ultra-processed foods.[6] These foods cause rapid elevations in circulating blood glucose levels, resulting in repeated stimulation of pancreatic β-cells and excessive insulin secretion.[12] While insulin initially facilitates glucose uptake into peripheral tissues, chronic exposure to elevated glucose and insulin levels gradually reduces insulin receptor sensitivity, leading to insulin resistance and compensatory hyperinsulinemia.[8,15] Persistent hyperinsulinemia significantly disrupts endocrine and metabolic homeostasis in women. Within the ovary, insulin acts synergistically with luteinizing hormone (LH) to stimulate ovarian theca cells, enhancing androgen biosynthesis through upregulation of steroidogenic enzymes including CYP17A1 and 3β-hydroxysteroid dehydrogenase.[8] concurrently, insulin suppresses hepatic synthesis of sex hormone-binding globulin (SHBG), increasing free circulating androgen levels.[8] Elevated androgens impair normal follicular recruitment and dominant follicle selection, leading to arrested follicular growth, anovulation, and menstrual irregularities commonly observed in metabolic reproductive disorders such as Polycystic Ovary Syndrome.[1,8] Chronic metabolic dysregulation associated with HGI diets is also strongly linked with central obesity, dyslipidemia, systemic inflammation, and metabolic syndrome, all of which negatively influence reproductive function and fertility outcomes.[6,8]

At the cellular level, excessive glucose availability induces profound metabolic stress by overwhelming normal mitochondrial energy metabolism.[4] Increased intracellular glucose enhances glycolytic activity and tri-carboxylic acid (TCA) cycle flux,

producing large amounts of NADH and FADH2 that donate electrons to the mitochondrial electron transport chain (ETC).[4,15] Under chronic nutrient excess, the ETC becomes overloaded, particularly at complexes I and III, resulting in incomplete electron transfer and leakage of electrons to molecular oxygen.[4] This process generates excessive reactive oxygen species (ROS), including superoxide radicals and hydrogen peroxide.[13,16] Although physiological levels of ROS are required for processes such as ovulation and cellular signaling, sustained overproduction creates a pro-oxidative intracellular environment.[13] Simultaneously, chronic hyperglycemia activates alternative glucose metabolic pathways including the polyol pathway, protein kinase C (PKC) pathway, and advanced glycation end product (AGE) formation, all of which further amplify oxidative stress and inflammatory signaling.[15] Accumulation of AGEs within ovarian tissues promotes receptor for AGE (RAGE)-mediated activation of NF-κB signaling, increasing production of pro-inflammatory cytokines and perpetuating metabolic inflammation.[15]

As oxidative stress intensifies, endogenous antioxidant defense mechanisms such as superoxide dismutase (SOD), catalase, glutathione peroxidase, and reduced glutathione become insufficient to neutralize excess ROS.[13] The resulting oxidative damage affects multiple cellular components essential for reproductive competence. Lipid peroxidation damages mitochondrial and cellular membranes, reducing membrane fluidity and disrupting ion transport and intracellular signaling. Oxidative modification of proteins impairs enzyme activity, receptor function, and structural protein integrity, while ROS-induced DNA damage affects both nuclear DNA and mitochondrial DNA (mtDNA), the latter being particularly vulnerable due to limited repair capacity and close proximity to the ETC.[1,4] Damage to mtDNA impairs expression of mitochondrial respiratory proteins, further reducing ATP synthesis and exacerbating mitochondrial dysfunction in a self-perpetuating cycle.[4] In ovarian tissues, these alterations compromise granulosa cell function, disrupt follicular microenvironment homeostasis, impair oocyte metabolic competence, and reduce embryonic developmental potential.[5,17] Collectively, chronic consumption of high glycemic diets may therefore create a sustained state of metabolic and oxidative stress that directly impacts ovarian physiology and female fertility through interconnected endocrine, inflammatory, and mitochondrial mechanisms.

Mitochondrial dysfunction in oocyte biology

Mitochondria are essential regulators of female reproductive physiology and play a critical role in maintaining oocyte quality and developmental competence.[9] During oogenesis and fertilization, mitochondria provide the large amounts of ATP required for energy-intensive processes such as meiotic spindle assembly, chromosomal alignment and segregation, calcium homeostasis, cytoplasmic maturation, and early embryonic cleavage.[9] Proper mitochondrial distribution within the oocyte is also necessary for localized energy supply and intracellular signaling. Oocyte competence is therefore closely associated with mitochondrial number, mitochondrial membrane potential, ATP-generating capacity, and overall mitochondrial integrity.[9,10] Alterations in mitochondrial function can disrupt these tightly coordinated processes and negatively affect fertilization and embryo development.

Exposure to chronic high glucose environments may induce mitochondrial abnormalities through several interconnected mechanisms.[1,4] Excess intracellular glucose increases metabolic flux into the mitochondrial electron transport chain, resulting in excessive reactive oxygen species (ROS) generation and oxidative damage to mitochondrial components.[4,13] Mitochondrial DNA (mtDNA), due to its close proximity to the electron transport chain and limited repair capacity, is particularly susceptible to oxidative mutations, which may impair expression of proteins involved in oxidative phosphorylation.[10] High glucose conditions have also been associated with reduced mitochondrial membrane potential, impaired oxidative phosphorylation efficiency, and decreased ATP synthesis, ultimately compromising the energy supply required for normal oocyte maturation.[1,3] In addition, metabolic stress may alter mitochondrial biogenesis and mitochondrial dynamics, affecting mitochondrial replication, distribution, and turnover within developing oocytes and granulosa cells.[2,3] Mitochondrial dysfunction may further activate stress-responsive and apoptotic signaling pathways within ovarian tissues.[5] Disturbances in mitochondrial membrane integrity can promote release of pro-apoptotic factors and disrupt cellular homeostasis, contributing to granulosa cell dysfunction and impaired follicular support.[5] These alterations collectively compromise oocyte developmental competence and embryonic viability. Experimental and clinical studies have demonstrated associations between mitochondrial dysfunction and meiotic abnormalities, chromosomal instability, follicular atresia, poor embryo quality, and reduced fertilization potential, emphasizing the importance of mitochondrial health in female fertility.[9–17] The central relationship can be summarized as in Figure 1.

Oxidative stress and apoptotic pathways

Oxidative stress represents a critical molecular mechanism linking high glycemic diets to impaired female fertility.[13,14] Under physiological conditions, low levels of reactive oxygen species (ROS) are necessary for normal ovarian functions such as follicular development, ovulation, and oocyte maturation.[13] However, chronic consumption of high glycemic foods leads to repeated episodes of hyperglycemia and hyperinsulinemia, which significantly increase intracellular ROS production through mitochondrial electron leakage and activation of oxidative enzymes such as NADPH oxidase.[4,13] Excessive ROS overwhelms endogenous antioxidant defenses including superoxide dismutase (SOD), catalase, and glutathione peroxidase, resulting in oxidative damage to lipids, proteins, and nucleic acids.[13,16] Oocytes are particularly vulnerable to oxidative stress because of their high metabolic activity and dependence on mitochondrial ATP production.[9] Elevated ROS levels disrupt mitochondrial membrane potential, impair oxidative phosphorylation, and induce mitochondrial DNA (mtDNA) damage, thereby compromising oocyte competence and embryonic developmental potential.[1,4] Oxidative stress further contributes to infertility through activation of intrinsic apoptotic pathways in ovarian and granulosa cells.[5] Mitochondrial dysfunction promotes permeabilization of the outer mitochondrial membrane, leading to the release of cytochrome c into the cytoplasm and subsequent activation of caspase-9 and caspase-3 mediated apoptosis.[5] Increased expression of pro-apoptotic proteins such as Bax, along with reduced levels of anti-apoptotic proteins including Bcl-2, has been observed in hyperglycemic and insulin-resistant conditions.[5] Granulosa cell apoptosis disrupts the bidirectional communication between granulosa cells and oocytes, impairing nutrient exchange, steroidogenesis, and follicular maturation.[17] Persistent activation of apoptotic signaling may therefore lead to follicular atresia, reduced ovarian reserve, poor oocyte quality, and decreased fertilization potential.[14,17] These findings collectively suggest that oxidative stress-induced mitochondrial apoptosis serves as an important mechanistic bridge between metabolic dysfunction and reproductive failure in women.

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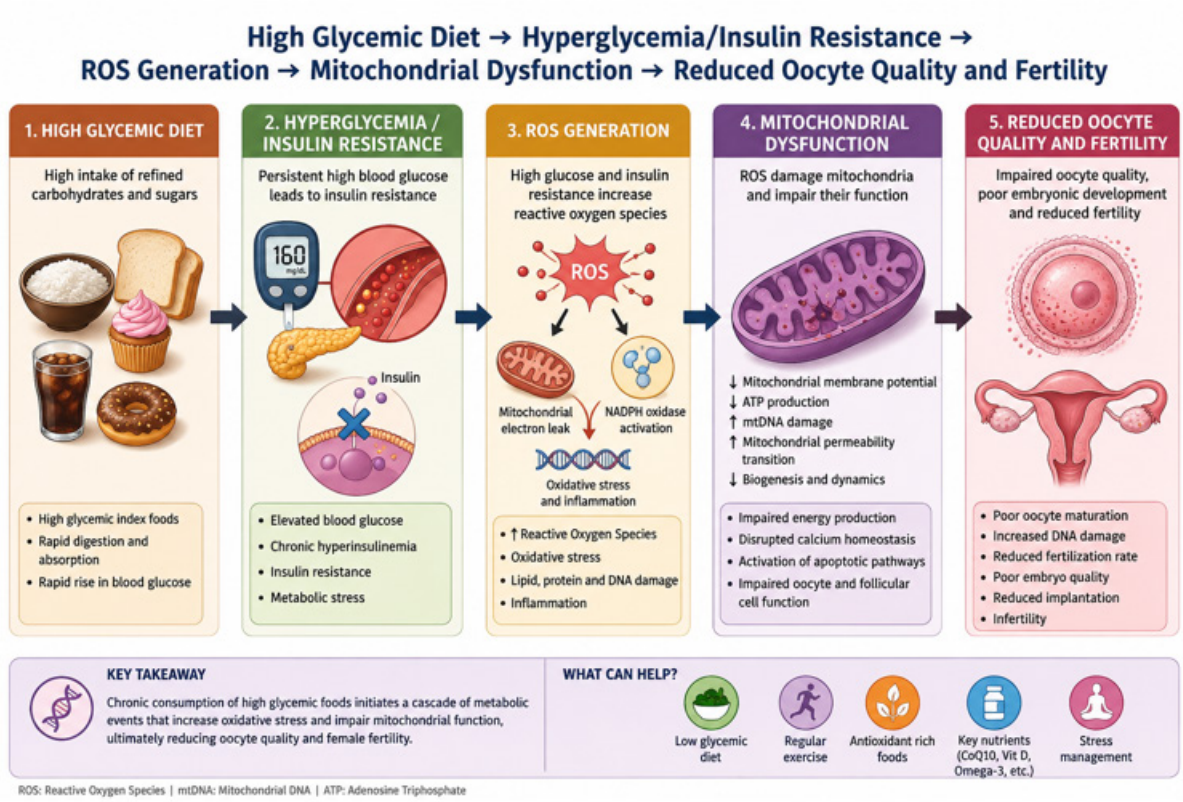

Figure 1 High Glycemic Diet-Induced Mitochondrial Dysfunction and Its Impact on Female Fertility. A high glycemic diet leads to hyperglycemia, insulin resistance, oxidative stress, mitochondrial dysfunction, reduced oocyte quality and female fertility.

Clinical relevance in pcos and infertility

Polycystic Ovary Syndrome is among the most prevalent endocrine-metabolic disorders associated with female infertility and is strongly linked to insulin resistance, oxidative stress, chronic inflammation, and mitochondrial dysfunction.[1,4,8] At the molecular level, insulin resistance in PCOS results in compensatory hyperinsulinemia, which not only disrupts systemic glucose homeostasis but also directly affects ovarian physiology.[8] Elevated insulin concentrations stimulate ovarian theca cells to increase androgen synthesis through activation of insulin and insulin-like growth factor-1 (IGF-1) signaling pathways.[8] Hyperandrogenism subsequently interferes with normal follicular maturation and ovulation.[8] Simultaneously, chronic hyperglycemia increases glucose flux through glycolysis and the tri-carboxylic acid (TCA) cycle, generating excessive reducing equivalents (NADH and FADH2) that overload the mitochondrial electron transport chain.[4] This promotes electron leakage primarily from complexes I and III, leading to overproduction of reactive oxygen species (ROS) such as superoxide anions and hydrogen peroxide.[4,13] Elevated ROS levels overwhelm endogenous antioxidant systems including superoxide dismutase, catalase, and glutathione peroxidase, thereby creating a persistent oxidative environment within ovarian tissues.[13] Mitochondrial dysfunction induced by oxidative stress has profound consequences on oocyte quality and follicular development in PCOS.[1,2] Several studies have demonstrated altered mtDNA copy number, impaired mitochondrial membrane potential, abnormal mitochondrial morphology, and dysregulated expression of mitochondrial biogenesis genes such as PGC-1α and TFAM in granulosa cells and oocytes from women with PCOS.[2,3] Excessive ROS additionally activates stress-sensitive inflammatory pathways including NF-κB, MAPK, and JNK signaling, increasing production of inflammatory cytokines such as TNF-α and IL-6, which further worsen insulin resistance and ovarian dysfunction.[4,5] Oxidative stress also triggers intrinsic apoptotic pathways through mitochondrial outer membrane permeabilization and release of cytochrome c, followed by activation of caspase-9 and caspase-3 mediated apoptosis in granulosa cells.[5] Since granulosa cells are essential for nutrient transport, steroidogenesis, and metabolic support of the developing oocyte, their apoptosis disrupts folliculogenesis and contributes to follicular arrest, anovulation, and infertility commonly observed in PCOS.[5,17]

Clinical studies demonstrating improvement in reproductive outcomes following low glycemic index or low glycemic load dietary interventions may therefore be explained through modulation of these molecular pathways.[6,7] Reduction in postprandial glucose excursions decreases mitochondrial ROS generation and improves insulin sensitivity, thereby reducing hyperinsulinemia-driven androgen excess and inflammatory signaling.[6] Improved metabolic homeostasis may partially restore mitochondrial bioenergetics, reduce oxidative stress-mediated apoptosis, and support healthier follicular development and ovulatory function [6,7] These findings collectively support the emerging concept that PCOS is not solely a reproductive endocrine disorder, but also a disorder of cellular metabolism and mitochondrial dysfunction influenced significantly by dietary glycemic patterns.

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Nutritional strategies targeting mitochondrial health

Emerging evidence suggests that nutritional modulation may play a significant role in improving female reproductive health by targeting mitochondrial dysfunction, oxidative stress, and insulin resistance.[11] Since mitochondrial integrity is highly dependent on substrate availability, redox balance, and cellular metabolic status, dietary composition can directly influence ovarian bioenergetics and oocyte quality.[11] High glycemic dietary patterns characterized by refined carbohydrates, sugar-sweetened beverages, bakery products, and ultra-processed foods promote repeated postprandial glucose spikes and hyperinsulinemia, which increase mitochondrial ROS production and inflammatory signaling.[6,15] In contrast, low glycemic dietary approaches help maintain glucose homeostasis, reduce oxidative burden, and improve mitochondrial efficiency.[6] Slower glucose absorption decreases excessive electron influx into the mitochondrial electron transport chain, thereby limiting electron leakage and ROS generation.[4] This may help preserve mitochondrial membrane potential, ATP synthesis, and mitochondrial DNA integrity in ovarian cells.[1,4] Clinically, low glycemic diets have been associated with improved insulin sensitivity, reduced androgen excess, improved ovulatory cycles, and better reproductive outcomes, particularly in women with insulin resistance and PCOS.[6,7] Beyond glycemic regulation, several dietary components may provide mitochondrial support through antioxidant and anti-inflammatory mechanisms.[11] Diets rich in colorful fruits, vegetables, legumes, nuts, seeds, whole grains, and healthy fats provide bioactive compounds that regulate oxidative stress and cellular metabolism.[11] Polyphenols such as curcumin, quercetin, and resveratrol have been shown to modulate mitochondrial biogenesis pathways including PGC-1α, activate antioxidant defense systems, and suppress NF-κB-mediated inflammatory signaling.[11,18] Omega-3 fatty acids may improve mitochondrial membrane fluidity and reduce production of pro-inflammatory cytokines, while vitamin D has been implicated in regulation of mitochondrial oxidative phosphorylation and insulin signaling pathways.[11] Coenzyme Q10, an essential component of the electron transport chain, may support ATP production and reduce oxidative damage by acting as an intracellular antioxidant.[9,11] Adequate intake of micronutrients including magnesium, selenium, zinc, B vitamins, iron, and folate is also critical because these nutrients function as cofactors for mitochondrial enzymes involved in ATP generation, DNA repair, and antioxidant defense.[11] Deficiencies in these nutrients may impair cellular energy metabolism and exacerbate oxidative stress within reproductive tissues.

From a practical nutritional perspective, dietary strategies supporting mitochondrial health should emphasize long-term metabolic stability rather than short-term caloric restriction.[11] Meals combining complex carbohydrates with protein, fiber, and healthy fats may help attenuate postprandial glucose excursions and reduce insulin spikes.[12] Inclusion of minimally processed carbohydrates such as millets, oats, quinoa, lentils, beans, brown rice, and whole fruits may provide sustained glucose release while supporting gut microbial diversity and metabolic regulation.[11] Adequate protein intake is particularly important because amino acids are required for cellular repair, antioxidant synthesis, and hormone production.[11] Excessive intake of trans fats, refined sugars, and highly processed foods should be minimized due to their association with increased oxidative stress and mitochondrial injury.[6,11] Additionally, chronic under nutrition and overly restrictive dieting may themselves impair mitochondrial function and reproductive hormone synthesis, highlighting the importance of balanced and sustainable nutritional interventions in reproductive health management.

Potential mitochondrial-supportive mechanisms of these nutritional strategies include:

i. Enhanced ATP production and mitochondrial bioenergetic efficiency

ii. Reduced ROS accumulation and oxidative damage

iii. Improved insulin sensitivity and glucose utilization

iv. Stabilization of mitochondrial membranes and mtDNA integrity

v. Suppression of inflammatory signaling pathways

vi. Reduced apoptosis in granulosa cells and ovarian tissues

vii. Improved follicular development and oocyte competence

viii. Better hormonal and metabolic homeostasis within the ovarian microenvironment

Conclusion

Emerging evidence increasingly supports the concept that female fertility is profoundly influenced by metabolic and nutritional status at the cellular level.[1,4] High glycemic dietary patterns may impair reproductive health through interconnected mechanisms involving chronic hyperglycemia, insulin resistance, oxidative stress, inflammation, and mitochondrial dysfunction.[4,13] Since oocyte maturation, follicular development, fertilization, and early embryogenesis are highly energy-dependent processes, mitochondrial integrity is critical for maintaining reproductive competence.[9] Excessive ROS generation and impaired mitochondrial bioenergetics can disrupt ATP production, damage mitochondrial DNA, activate apoptotic pathways, and compromise granulosa cell function, ultimately contributing to poor oocyte quality, anovulation, and infertility.[5,13] These molecular disturbances appear particularly relevant in metabolic reproductive disorders such as Polycystic Ovary Syndrome, where insulin resistance and oxidative stress are central pathological features.[1,4] Importantly, the growing understanding of nutrition-mediated mitochondrial regulation highlights diet not merely as a lifestyle factor, but as a potential modulator of reproductive cellular physiology.[11] Dietary glycemic control and nutrient-dense eating patterns may influence reproductive outcomes by reducing oxidative stress, improving metabolic homeostasis, and preserving mitochondrial function within ovarian tissues.[6,11] This evolving evidence strengthens the emerging paradigm that reproductive health and metabolic health are deeply interconnected.[19] Future research should focus on identifying sensitive molecular biomarkers of mitochondrial dysfunction, oxidative stress, and ovarian bioenergetics,[20] while also evaluating targeted nutritional interventions as adjunct strategies for improving fertility and reproductive outcomes in women with metabolic dysfunction.

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None.

Conflicts of interest

The author declares there is no conflict of interest.

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