Review Article

Phytochemicals Targeting Oxidative Stress in Male Infertility: Molecular Mechanisms, Cellular Protection, and Translational Perspectives

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ABSTRACT

Male infertility is a heterogeneous reproductive disorder associated with impaired spermatogenesis, abnormal sperm maturation, endocrine dysfunction, varicocele, infection, environmental exposures, metabolic disease, and idiopathic factors. Oxidative stress is an important mechanism linking several of these conditions to impaired sperm function. Excessive reactive oxygen species can cause lipid peroxidation, protein oxidation, mitochondrial dysfunction, DNA damage, apoptosis, altered autophagy, and ferroptosis, whereas physiological reactive oxygen species are required for normal sperm signaling and fertilization. Phytochemicals have attracted interest because they may restore redox homeostasis through direct antioxidant activity and modulation of endogenous antioxidant, inflammatory, mitochondrial, and cell-death pathways. This review summarizes primary research investigating phytochemicals targeting oxidative stress in male reproductive dysfunction, with emphasis on chlorogenic acid, lycopene, curcumin, astaxanthin, flavonoids, epigallocatechin-3-gallate, resveratrol, Morinda officinalis polysaccharides, and apigenin. Human and human-sperm studies provide evidence for effects on sperm motility, viability, oxidative damage, DNA integrity, and cryopreservation outcomes, while animal and cellular studies provide additional evidence involving NRF2, mitochondrial function, SIRT1/PGC-1α, apoptosis, inflammation, and the GSH–GPX4 ferroptosis pathway. However, most mechanistic evidence remains preclinical, and findings vary according to compound, concentration, formulation, and experimental model. Phytochemicals should therefore be considered promising experimental redox modulators rather than established treatments for male infertility. Future clinical studies require standardized preparations, biologically achievable dosing, oxidative-stress phenotyping, rigorous mechanistic assessment, and fertility-relevant outcomes.

Key words: Male Infertility, Oxidative Stress, Reactive Oxygen Species, Spermatozoa, Antioxidants, Phytochemicals, Sperm Motility, DNA Damage, Mitochondria, Ferroptosis, NRF2, Cryopreservation.

Male infertility is biologically heterogeneous. Abnormal spermatogenesis, varicocele, endocrine dysfunction, infection, environmental exposures, metabolic disease, gonadotoxic treatment, and idiopathic mechanisms can converge on cellular pathways that are not captured fully by routine semen analysis. Oxidative stress is one such pathway. In infertile men, higher seminal ROS and oxidative markers have been associated with poorer motility and greater sperm DNA fragmentation, while lower antioxidant activity has been reported in comparison with fertile controls [1-2]. In men with varicocele, seminal ROS and oxidative stress measures have likewise been associated with sperm nuclear DNA damage [3]. ROS should not, however, be regarded as intrinsically pathological. Controlled redox signaling contributes to sperm capacitation and other maturation events.

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The therapeutic problem is therefore an imbalance between oxidant generation and antioxidant defenses rather than the complete elimination of ROS. This distinction provides a mechanistic basis for the variable dose-response relationships observed with antioxidant compounds and for the possibility that excessive antioxidant exposure may interfere with physiological signaling. Spermatozoa are particularly susceptible to oxidative injury because their membranes are rich in polyunsaturated fatty acids and mature sperm have limited cytoplasmic antioxidant reserves. Lipid peroxidation can alter membrane fluidity and generate reactive aldehydes, while mitochondrial injury can reduce ATP availability and further increase ROS generation. Oxidative damage to sperm DNA may compromise fertilization and embryo development even when conventional semen parameters are relatively preserved [1-3].

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Plant-derived phytochemicals have attracted interest because their actions extend beyond direct radical scavenging. Depending on structure and concentration, they can influence endogenous antioxidant defenses, inflammatory signaling, mitochondrial function, apoptosis, and regulated cell-death pathways. The present review focuses on primary research evaluating these effects in human sperm or patients, animal models, and reproductive-cell systems. Particular attention is given to evidence that can be connected to measurable sperm or testicular outcomes rather than to pathway expression alone.

2. OXIDATIVE STRESS AND MOLECULAR INJURY IN THE MALE REPRODUCTIVE SYSTEM

Excessive ROS can initiate lipid peroxidation, protein oxidation, and oxidative DNA damage. In sperm, these processes are linked to impaired motility, membrane dysfunction, abnormal chromatin integrity, and altered mitochondrial activity. A prospective study of 101 infertile men found that men with higher seminal ROS had poorer motility and a greater incidence of sperm DNA fragmentation; ROS was negatively correlated with several motility measures [1]. Another study comparing fertile and infertile men reported higher seminal malondialdehyde and lower superoxide dismutase and glutathione peroxidase activities in infertile men, with oxidative markers correlating with sperm DNA fragmentation [2].

Mitochondria occupy a central position in this process because they are both targets and sources of ROS. Oxidative damage to the mitochondrial membrane and respiratory machinery can reduce ATP production required for flagellar movement. In parallel, mitochondrial dysfunction can facilitate apoptotic signaling through changes in BCL-2-family proteins and caspase activation. In varicocele models, lycopene has been investigated against a background of hypoxia, hyperthermia, and apoptosis, illustrating how several forms of testicular stress can converge on sperm dysfunction [4].

Ferroptosis adds another layer to the redox biology of the testis. This iron-dependent regulated cell death pathway is characterized by phospholipid peroxidation and failure of antioxidant defenses centered on glutathione and GPX4. Recent primary work in mice linked astaxanthin treatment with changes in iron handling, lipid metabolism, and GPX4-associated pathways while improving sperm concentration and forward motility in a cyclophosphamide-induced model [5]. These findings are mechanistically relevant but remain preclinical. Autophagy and mitochondrial quality control also interact with oxidative stress. Changes in autophagy markers can accompany testicular injury, but expression of LC3-II or Beclin-1 alone does not establish increased autophagic flux. Similarly, an increase in an antioxidant protein does not by itself demonstrate pathway causality. The strongest mechanistic experiments combine molecular measurements with functional inhibition, genetic manipulation, sperm phenotyping, histology, or fertility outcomes.

3. PHYTOCHEMICALS WITH HUMAN OR HUMAN-SPERM EVIDENCE

Chlorogenic acid provides a recent example of direct human-sperm evidence. In an in vitro study using human semen, 100 µM chlorogenic acid increased rapid progressive motility and reduced seminal F2-isoprostanes. Under hydrogen-peroxide-induced oxidative stress, chlorogenic acid reduced DNA damage and restored motility while increasing NRF2 mRNA expression [6]. These results support redox modulation in human sperm, but they were obtained ex vivo and do not establish that oral chlorogenic acid treatment improves pregnancy or live birth.

Lycopene has stronger clinical exposure data. In a randomized, double-blind, placebo-controlled trial, 44 infertile men with oligozoospermia received 25 mg/day lycopene or placebo for 12 weeks. The intervention was associated with increases in total sperm count and concentration, with additional within-group changes in several semen measures [7]. Experimental work complements this finding: in rats with induced varicocele, lycopene improved sperm viability, membrane integrity, and testicular histology and reduced expression of pro-apoptotic markers [4]. The clinical and animal studies address different questions and should not be treated as interchangeable evidence. Curcumin illustrates both the potential and limitations of translation. A randomized clinical trial in 56 completing infertile men reported that 80 mg/day curcumin nanomicelles for 10 weeks was associated with improvements in sperm count, concentration and motility, together with changes in total antioxidant capacity, malondialdehyde, C-reactive protein and TNF-α [8]. Human sperm cryopreservation studies have also reported lower ROS and DNA fragmentation and higher GPX4 expression after curcumin exposure [9]. However, another human cryopreservation study of curcumin with coenzyme Q10 did not uniformly improve endpoints and reported increased DNA fragmentation in curcumin-containing groups [10]. These contrasting findings reinforce the importance of dose, formulation, and experimental context.

Astaxanthin has been studied primarily as a cryoprotective additive in human sperm. In one study of human semen, 1 µM astaxanthin during freeze-thawing improved motility and viability and reduced ROS, phosphatidylserine externalization, and lipid peroxidation [11]. A separate study in 30 normozoospermic samples found improved post-thaw motility and reduced ROS with 0.5 µM astaxanthin in both vitrification and vapor-freezing protocols, although morphology and DNA fragmentation were not changed [12]. Another human study reported favorable effects on post-thaw motility with astaxanthin, but concentration-dependent differences

underscore that a single optimal dose cannot be generalized across protocols [13].

4. FLAVONOIDS, CAROTENOIDS AND CATECHINS: MECHANISTIC EVIDENCE

Flavonoids have been investigated in both human-sperm toxicant models and whole-animal reproductive injury. In an experimental human-sperm study, exposure to aluminum, cadmium and lead reduced motility; rutin, naringin and kaempferol attenuated some of these effects, while quercetin and catechin did not improve motility under the tested conditions [14]. This result is useful because it demonstrates that the antioxidant label does not predict a uniform biological response across compounds.

Lutein has been evaluated in an alcohol-induced reproductive injury model in male rats. Chronic alcohol exposure reduced sperm quality, testosterone, and glutathione peroxidase while increasing malondialdehyde and inflammatory mediators. Lutein, particularly at the highest tested dose, improved reproductive and antioxidant measures and was associated with higher Nrf2/HO-1 and lower NF-κB, Bax, and caspase-3 signaling [15].

The study supports a combined antioxidant, anti-inflammatory, and anti-apoptotic mechanism but does not establish efficacy in human infertility. Kaempferol has similarly been tested against toxicant-induced testicular injury. In lead-exposed rats, kaempferol reduced oxidative and inflammatory abnormalities and improved sperm-related and steroidogenic outcomes; effects were also observed when combined with vitamin E [16]. The interpretation should remain model-specific because lead toxicity is a defined experimental insult and differs biologically from idiopathic infertility. Naringin has recently been examined in a heat-stress model. In male rats exposed to repeated heat stress, naringin reduced ROS and malondialdehyde, improved glutathione and superoxide dismutase, and partially restored sperm and hormonal measures and seminiferous architecture [17].

Again, this supports biological plausibility rather than a clinical recommendation. The model is particularly relevant to the temperature-sensitive testicular environment but does not reproduce all causes of human infertility. Epigallocatechin-3-gallate (EGCG) has been evaluated in semen cryopreservation. In human sperm exposed to oxidative stress, EGCG was investigated in relation to DNA damage and subsequent embryo-related outcomes [18]. In buffalo and bovine semen, EGCG supplementation improved selected post-thaw motility, antioxidant enzyme activity and oxidative-stress measures at specific concentrations [19-20]. These findings support dose-dependent redox protection, but animal semen cryopreservation cannot be used as direct evidence for clinical benefit in infertile men.

5. RESVERATROL, MITOCHONDRIAL SIGNALING AND REGULATED CELL DEATH

Resveratrol has been studied across several experimental reproductive-injury models. In human sperm and rat germ cells exposed to lipid-peroxidation stress, trans-resveratrol reduced lipid peroxidation at concentrations below those producing substantial cytotoxicity and preserved sperm chromatin and membrane characteristics [21]. In rats exposed to malathion, resveratrol improved sperm and reproductive parameters and reduced testicular malondialdehyde [22]. Other toxicant models have implicated p38 MAPK, ATF2, and iNOS signaling in testicular injury and shown that resveratrol can preserve steroidogenic function and reduce apoptosis [23].

More recent work has connected resveratrol with ferroptosis-related pathways. In a model of anti-PD-1-associated testicular toxicity, treatment with resveratrol was associated with activation of NRF2 and preservation of the SLC7A11-GPX4 system, together with reduced lipid peroxidation and improved reproductive measures [24]. The study is mechanistically relevant to ferroptosis but concerns drug-induced reproductive toxicity rather than idiopathic male infertility. The broader implication is that phytochemicals may act through endogenous stress-response systems rather than simply neutralizing ROS. Such pathway effects are potentially advantageous because they can amplify cellular defenses, but they also make dose selection more complex. A compound that activates a stress-response pathway at one concentration may produce different effects at another, particularly in cells with different basal redox states.

6. EMERGING PHYTOCHEMICALS AND DELIVERY STRATEGIES

Morinda officinalis polysaccharides illustrate a strategy centered on mitochondrial protection and Leydig-cell function. In H2O2-treated TM3 Leydig cells and an ethane dimethanesulfonate-induced rat model, these polysaccharides reduced oxidative and mitochondrial injury, improved testosterone secretion and sperm measures, and activated the SIRT1/PGC-1α pathway. Pharmacological inhibition of SIRT1 attenuated the protective effect, providing stronger pathway evidence than simple protein-expression correlations [25].

Apigenin has been investigated mainly in semen cryopreservation. In rooster sperm, 100 μM apigenin improved post-thaw motility, viability, membrane and mitochondrial function and reduced ROS and apoptosis [26]. Earlier animal-sperm work similarly found concentration-dependent cryoprotective effects [27]. These findings are useful for understanding redox control during sperm handling but should not be equated with treatment of infertility in men. Nanoformulations may alter the pharmacological behavior of poorly soluble phytochemicals. Curcumin-containing

nanoliposomes have improved post-thaw parameters in ram sperm, while other curcumin nanoformulations have been evaluated in goat and buffalo semen [28]. Such systems may increase local exposure, but the formulation itself becomes a new source of variability. Particle size, composition, biodistribution, reproductive toxicity, and long-term clearance require independent characterization.

Astaxanthin also illustrates the emergence of mechanistically integrated phytochemical research. A mouse study linked astaxanthin with mitochondrial oxidative-stress reduction, altered fatty-acid metabolism, and ferroptosis-related proteins including GPX4, while improving sperm concentration and motility [5]. These data extend the discussion beyond conventional antioxidant capacity toward metabolic regulation, although clinical relevance remains unproven.

7. EVIDENCE HIERARCHY AND LIMITATIONS

Several recurring limitations should shape interpretation. First, endpoint inflation is common: a study reporting simultaneous changes in ROS, MDA, SOD, CAT, BAX and NRF2 has multiple correlated measurements rather than multiple independent demonstrations of improved fertility. Second, pathway labels can be overinterpreted. Increased NRF2 protein does not necessarily demonstrate increased transcriptional activity; increased LC3-II does not prove autophagic flux; and increased GPX4 does not alone prove that ferroptosis caused the reproductive phenotype. Third, toxicant and cryopreservation models test protection against a defined insult and therefore answer a narrower question than treatment of spontaneous male infertility.

Dose is another central limitation. Human-sperm studies often expose cells directly to micromolar concentrations that are not equivalent to oral dosing. Curcumin, EGCG and several flavonoids have limited or complex bioavailability, and formulation can materially change exposure. Conversely, the finding that a compound works at a particular concentration in vitro does not mean that a higher concentration will be more effective. The human curcumin cryopreservation literature illustrates this issue particularly well [9-10].

8. THE ANTIOXIDANT PARADOX AND TRANSLATIONAL PRIORITIES

The therapeutic objective should be restoration of redox homeostasis rather than maximal antioxidant activity. Physiological ROS contribute to sperm signaling, and excessive antioxidant exposure may theoretically suppress useful redox processes. This may help explain concentration-dependent effects observed with several phytochemicals and argues against assuming that combinations of multiple antioxidants will be additive. Future clinical studies should therefore define the population biologically rather than treating all male infertility as a single oxidative-stress phenotype. Baseline oxidative status, sperm DNA fragmentation, mitochondrial measures and conventional semen parameters could be used to characterize participants before intervention. Trials should use standardized chemical preparations, report actual doses and formulation, monitor adherence and exposure, and distinguish effects on semen quality from effects on pregnancy, ongoing pregnancy and live birth.

Primary research should also improve mechanistic rigor. Studies claiming NRF2 activation should measure downstream transcriptional targets; studies invoking autophagy should assess flux; studies claiming ferroptosis should evaluate lipid peroxidation together with GPX4/GSH and iron-dependent rescue or inhibition; and studies of mitochondrial protection should incorporate functional measurements rather than membrane-potential staining alone. These approaches would reduce pathway overinterpretation and make cross-study comparison more reliable.

CONCLUSION

Oxidative stress is an important biological component of male reproductive dysfunction, but it is neither the sole cause of infertility nor a process that should be suppressed indiscriminately. Primary research supports a range of phytochemical effects on sperm motility, oxidative damage, DNA integrity, mitochondrial function, inflammation, apoptosis and, increasingly, ferroptosis. Human evidence is available for selected compounds, particularly lycopene, curcumin, chlorogenic acid and astaxanthin-related sperm cryopreservation approaches, whereas many mechanistic claims remain confined to animal or cell models.

The current evidence therefore supports phytochemicals as research candidates rather than established universal treatments for male infertility. Translation will depend on standardized preparations, biologically credible dosing, careful phenotyping of oxidative stress, rigorous mechanistic assays and adequately powered clinical trials using fertility-relevant endpoints.

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