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Microelements in Human Fertility: A Comprehensive Literature Review (2015–2025)

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Summary

Introduction and purpose

Microelements are essential nutrients required in small amounts but have profound physiological and biochemical roles in human reproduction. The main purpose of this review is to present the current state of knowledge regarding the effects of zinc, selenium, iodine, copper, iron, manganese, and magnesium on male and female fertility. The analysis highlights how deficiencies, excesses, and synergistic interactions among these elements affect gametogenesis, hormonal balance, oxidative stress, epigenetic modifications, and pregnancy outcomes. Through a synthesis of clinical, biochemical, and epidemiological findings, this paper emphasizes the need for micronutrient optimization as an adjunct strategy in managing infertility.

Material and methods

This review follows a comprehensive narrative synthesis approach to evaluate the role of microelements in human fertility, with a focus on zinc, selenium, iodine, copper, iron, manganese, and magnesium. A review was based on the analysis of 44 peer-reviewed articles, clinical, biochemical, and epidemiological studies published between 2015 and 2025. The primary objective was to critically assess the relationship between micronutrient status and fertility outcomes in both male and female populations. Studies were selected based on predefined eligibility criteria, which included research on micronutrient supplementation or deficiency, reproductive outcomes, and evidence from randomized controlled trials (RCTs), cohort studies, and meta-analyses.

Conclusions

Recent research highlights the synergistic effects of these elements, showing that deficiencies or imbalances can exacerbate fertility issues. Optimizing micronutrient levels is becoming an important strategy in fertility management, with potential benefits for both natural conception and assisted reproductive technologies (ART).

Keywords

fertility; supplements; microelements; assisted reproductive technologies;

Introduction and purpose

Human fertility is a complex biological process influenced by genetic, hormonal, environmental, and nutritional factors. Among the nutritional components, microelements play indispensable roles in reproductive health, contributing to vital physiological processes such as DNA synthesis, antioxidant defense, mitochondrial function, enzyme activation, hormone production, and epigenetic regulation. [1,2] Given that infertility affects approximately 15% of couples worldwide, it becomes imperative to investigate modifiable factors such as micronutrient status. Recent evidence suggests that optimal levels of certain microelements can substantially improve fertility outcomes, both naturally and in assisted reproductive technologies (ART). [3,4] This review consolidates findings focusing on the role of key microelements in human fertility and addresses how each element affects reproductive function, fertility treatment outcomes, and pregnancy maintenance.

Description of the state of knowledge

Zinc

Zinc is a vital trace element that serves as both a structural and catalytic component of over 300 enzymes and approximately 1000 transcription factors, playing a crucial role in a variety of

biological processes, including gametogenesis, hormonal regulation, and antioxidant defense. It is indispensable in the synthesis of proteins, nucleic acids, and in maintaining the structural integrity of cellular components. Zinc acts as a cofactor for enzymes involved in critical cellular processes such as DNA synthesis and repair, protein synthesis, and cell division. [5,6] As such, it is directly involved in processes essential for fertility, particularly gamete formation and function. In the male reproductive system, zinc plays an essential role in sperm development, motility, and overall sperm function. Zinc stabilizes sperm chromatin by binding to DNA, which is crucial for protecting genetic material during sperm maturation. It also maintains the integrity of the acrosome, a key organelle in sperm, which is required for successful fertilization. During the acrosomal reaction, zinc assists in the proper function of enzymes such as hyaluronidase, which is essential for sperm to penetrate the oocyte's zona pellucida. Furthermore, zinc regulates apoptosis, ensuring proper cell turnover during spermatogenesis. [7,8] One of the most significant mechanisms through which zinc contributes to male fertility is through the modulation of the hypothalamic-pituitary-gonadal (HPG) axis, which regulates the secretion of hormones such as luteinizing hormone (LH), follicle-stimulating hormone (FSH), and testosterone. Zinc directly influences the synthesis and secretion of these hormones, which are critical for maintaining spermatogenesis and testosterone biosynthesis. It does this by interacting with the hypothalamus and pituitary, modulating the release of gonadotropins, and ultimately affecting the Leydig and Sertoli cells in the testes, which are essential for sperm maturation. Zinc deficiency in men is often linked to several forms of male infertility, including oligospermia (low sperm count), asthenozoospermia (low sperm motility), and increased sperm DNA fragmentation. Deficient zinc levels can impair DNA repair enzymes, which are essential for maintaining sperm DNA integrity, leading to an accumulation of oxidative damage and compromised gamete quality.[1,3] The consequences of zinc deficiency in sperm quality are not limited to motility and concentration; it also affects the ability of sperm to fertilize oocytes successfully, with clinical studies showing that zinc deficiency is correlated with lower fertilization rates and poor embryo development. Supplementation of zinc in deficient individuals has been shown to significantly improve sperm concentration, motility, and overall sperm health. Furthermore, zinc supplementation can help reduce sperm DNA fragmentation, a critical factor for improving the chances of successful fertilization and pregnancy outcomes. Studies have demonstrated that zinc supplementation in men undergoing assisted reproductive technology (ART) procedures can enhance fertilization rates and increase pregnancy success, highlighting its importance in clinical fertility treatments. In women, zinc plays a similarly critical role in supporting reproductive health. Zinc influences several aspects of female

fertility, including oocyte maturation, early embryo development, and implantation. Zinc is involved in the regulation of the menstrual cycle by modulating the secretion of gonadotropins, such as FSH and LH, which are critical for follicular development and ovulation. Adequate zinc levels are necessary for proper oocyte maturation, as zinc regulates the activation of the MAPK (mitogen-activated protein kinase) signaling pathway, which is essential for initiating meiotic progression and oocyte development. Additionally, zinc stabilizes mitochondrial function in oocytes, ensuring energy production required for normal egg maturation. In the context of early embryonic development, zinc plays a key role in blastocyst formation and embryo viability, as it helps regulate the processes of DNA synthesis and cell division. Zinc also influences the expression of key genes involved in cell differentiation and the implantation of the embryo into the uterine lining. Zinc deficiency in women has been associated with impaired oocyte quality, increased rates of oocyte aneuploidy (chromosomal abnormalities), and poor reproductive outcomes. Studies have shown that low zinc levels correlate with a higher incidence of failed fertilization, poor blastocyst formation, and increased risk of miscarriage. Furthermore, zinc deficiency negatively impacts uterine receptivity, impairing the ability of the endometrium to support embryo implantation. Supplementation with zinc in women has demonstrated positive effects on oocyte quality, with improvements in fertilization rates, embryo development, and overall pregnancy outcomes, particularly in women undergoing ART. [9,10] Zinc's role in stabilizing mitochondrial function and modulating oxidative stress is also particularly beneficial in supporting uterine health and the overall fertility process. [2]

Selenium

Selenium is a microelement that is integrated into various selenoproteins, such as glutathione peroxidase (GPx), thioredoxin reductase (TrxR), and selenoprotein P, each playing a critical role in maintaining cellular redox balance, modulating inflammation, and protecting against oxidative stress. As a key component of these enzymes, selenium contributes to the detoxification of reactive oxygen species (ROS), which are byproducts of cellular metabolism and play a central role in cellular injury and inflammation.[11,12] Through its antioxidant properties, selenium helps to maintain mitochondrial integrity, modulate oxidative stress, and protect cellular components from damage, which is particularly important in reproductive cells such as sperm and oocytes, as well as in early embryonic development. In men, selenium is vital for the maintenance of sperm health and fertility. It supports sperm motility by preserving mitochondrial function within the sperm tail, which is essential for energy production during sperm movement. The mitochondrial integrity of sperm is crucial for maintaining ATP levels required for motility and overall functionality. Selenium also plays a role in sperm DNA

integrity, as oxidative damage to sperm DNA can lead to decreased fertilization rates, poor embryo development, and an increased risk of pregnancy loss. Clinical studies have demonstrated that selenium supplementation can improve sperm morphology, motility, and DNA integrity, enhancing overall sperm quality and increasing the likelihood of successful fertilization and pregnancy. Selenium's role in sperm quality is particularly relevant in the context of male infertility, as deficiencies in selenium have been associated with decreased sperm count, motility, and an increased prevalence of sperm DNA fragmentation. In women, selenium plays a crucial role in regulating thyroid function, which is essential for ovulation and the maintenance of pregnancy. Selenium is an integral component of the selenoproteins involved in the conversion of thyroxine (T4) to triiodothyronine (T3), the active form of thyroid hormone, through the action of deiodinases. Thyroid hormones are critical for regulating the menstrual cycle, follicular development, and ovulation. A deficiency in selenium can impair thyroid hormone metabolism, leading to suboptimal thyroid function, which may contribute to menstrual irregularities, anovulation, and reduced fertility. Additionally, selenium plays a role in maintaining the health of the endometrium, the uterine lining essential for embryo implantation. Selenium's antioxidant effects help protect the endometrium from oxidative stress, which can damage cells and disrupt implantation processes. Selenium also influences the production of prostaglandins, which are important for the regulation of uterine contractions and blood flow, further supporting reproductive health. Selenium’s antioxidant properties are crucial during early pregnancy. Pregnancy-related changes in the immune system and increased metabolic activity can lead to increased oxidative stress, which, if left unchecked, may compromise pregnancy outcomes. Selenium helps to modulate the inflammatory response and support the redox balance, reducing the risk of complications such as preeclampsia and intrauterine growth restriction (IUGR), both of which are associated with oxidative stress. Additionally, selenium is essential for placental health, where it supports antioxidant enzymes that protect both the placenta and developing fetus from oxidative damage. Insufficient selenium levels during pregnancy have been linked to an increased risk of miscarriage, preterm birth, and poor fetal development, particularly in the context of thyroid dysfunction. Studies have highlighted the importance of selenium supplementation in women with thyroid dysfunction or those attempting assisted reproductive technologies (ART). [12,13] Women with low selenium levels undergoing ART, such as in vitro fertilization (IVF), may experience poorer outcomes, including lower pregnancy rates and higher rates of miscarriage. Selenium supplementation has the potential to improve these outcomes by restoring normal thyroid function, enhancing oocyte quality, and reducing oxidative damage to embryos. Deficiency in

selenium, especially in the context of infertility, has been associated with various reproductive issues. In both men and women, selenium deficiency has been linked to increased oxidative stress, mitochondrial dysfunction, hormonal imbalances, and impaired gamete quality. [14]

Iodine

Iodine is a trace element that plays a pivotal role in the synthesis of thyroid hormones, primarily thyroxine (T4) and triiodothyronine (T3). These hormones are crucial regulators of metabolism, growth, development, and reproductive function. Thyroid hormones act on nearly every tissue and organ in the body, and their influence on reproductive health is profound. The thyroid gland uses iodine to produce these hormones, which in turn regulate the hypothalamic-pituitary-gonadal (HPG) axis, modulate ovarian and testicular function, and maintain the health of the uterine lining. [15,16] In women, adequate iodine levels are essential for proper follicular development, oocyte maturation, and luteal phase function. Thyroid hormones directly influence the secretion of gonadotropin-releasing hormone (GnRH) from the hypothalamus, which stimulates the release of follicle-stimulating hormone (FSH) and luteinizing hormone (LH) from the pituitary. These hormones are necessary for the growth and maturation of ovarian follicles, ovulation, and the maintenance of the corpus luteum. Iodine deficiency can impair the synthesis of thyroid hormones, leading to subclinical hypothyroidism, which disrupts this delicate hormonal balance. This can result in menstrual irregularities, including anovulation (the absence of ovulation), luteal phase defects, and impaired endometrial receptivity. These factors collectively increase the risk of infertility and early pregnancy loss. Thyroid hormones also play a critical role in the establishment of a receptive uterine environment. The endometrium, which must undergo specific changes in response to hormonal signaling to support embryo implantation, is sensitive to thyroid hormone levels. Iodine deficiency can result in suboptimal thyroid hormone production, impairing endometrial development and the expression of key factors necessary for embryo attachment and implantation. Studies have shown that women with iodine deficiency are more likely to experience delayed conception and an increased risk of miscarriage, underscoring the importance of adequate iodine for reproductive success. In men, iodine plays a critical role in the synthesis of testosterone and the function of Sertoli cells, which are essential for spermatogenesis. Testosterone is necessary for the development and maintenance of male reproductive tissues, as well as the regulation of sperm production in the testes. Sertoli cells, which nourish and support developing sperm, are highly sensitive to hormonal regulation, including thyroid hormones. A deficiency in iodine can lead to impaired thyroid hormone synthesis, resulting in altered testosterone levels and diminished Sertoli cell function, which may hinder spermatogenesis and contribute to male

infertility. Additionally, iodine deficiency in men has been associated with reduced sperm motility and abnormal sperm morphology, further compromising reproductive potential. Beyond its direct effects on fertility, iodine deficiency is a known risk factor for various reproductive complications, including preterm birth, low birth weight, and cognitive impairment in offspring. These adverse outcomes highlight the importance of adequate iodine intake, especially during pregnancy. [17,18] The World Health Organization (WHO) recommends that pregnant women receive sufficient iodine, as the developing fetus relies on maternal thyroid hormones for normal neurological and cognitive development during the first trimester. Iodine deficiency during pregnancy can result in irreversible developmental impairments in the child, particularly affecting brain function. [19]

Iron

Iron is an essential micronutrient that plays a critical role in various biochemical processes necessary for normal cellular function, particularly in cellular respiration, oxidative phosphorylation, and DNA synthesis. As a key component of hemoglobin and myoglobin, iron facilitates the transport of oxygen to tissues and is crucial for the oxidative metabolism required to generate ATP in mitochondria. In the context of reproduction, iron is particularly important for maintaining mitochondrial function in oocytes, as mitochondria are the energy powerhouse of the cell. Adequate iron levels are necessary to support the high energy demands of oocytes during processes such as maturation, fertilization, and early embryonic development. Iron also participates in the regulation of oxidative stress by maintaining the integrity of cellular membranes and by acting as a cofactor for antioxidant enzymes that protect cells from reactive oxygen species (ROS). In the female reproductive system, iron plays a vital role in the endometrium, particularly in supporting angiogenesis, which is the formation of new blood vessels critical for embryo implantation and growth. Iron influences angiogenesis by modulating the expression of hypoxia-inducible factors (HIFs), which are transcription factors activated under low oxygen conditions. HIFs regulate the expression of vascular endothelial growth factor (VEGF), a key mediator in blood vessel formation. Proper VEGF signaling ensures adequate blood flow to the endometrial lining, creating a favorable environment for embryo implantation. Iron also contributes to cellular differentiation and proliferation within the endometrium, which are essential processes for the development of a receptive uterine environment for the early embryo. Despite its importance, both iron deficiency and iron overload can have detrimental effects on reproductive health. Iron deficiency, particularly when it leads to iron deficiency anemia, is associated with poor reproductive outcomes. [20,21] In women, insufficient iron levels impair oocyte quality, leading to a decrease in oocyte yield and

developmental competence. Iron deficiency has also been linked to poor embryo development, reduced implantation rates, and increased risk of miscarriage. Furthermore, iron deficiency may impair the function of the hypothalamic-pituitary-gonadal axis, leading to hormonal imbalances that disrupt the menstrual cycle, ovulation, and overall fertility.[22, 23] On the other hand, iron overload, often resulting from excessive iron supplementation or genetic disorders such as hereditary hemochromatosis, can have equally harmful effects on fertility. Iron excess can lead to the accumulation of iron in the gonads, causing oxidative damage to ovarian and testicular tissues. In women, iron overload has been associated with ovarian dysfunction, including impaired oocyte maturation, reduced fertilization potential, and gonadal failure. [25] In men, excessive iron can lead to testicular damage, reduced sperm quality, and infertility. Additionally, iron overload may exacerbate inflammatory processes in the reproductive tissues, further compromising fertility. [26] Iron-induced oxidative damage can also interfere with DNA integrity in gametes, leading to chromosomal abnormalities and poor embryonic development. Both iron deficiency and overload disrupt normal reproductive function. Careful monitoring of iron status, particularly in individuals undergoing assisted reproductive technologies (ART), is essential to optimize fertility outcomes. It is important that iron supplementation is carefully managed and individualized, with attention to both the prevention of deficiency and the avoidance of excess, to ensure that iron levels are within the optimal range for fertility and reproductive success.

Copper

Copper is an essential trace element that plays a pivotal role as a cofactor for several key enzymes involved in a wide range of crucial biological processes, including electron transport, collagen cross-linking, and antioxidant defense mechanisms. These copper-dependent enzymes, such as cytochrome c oxidase, lysyl oxidase, and superoxide dismutase, are integral to cellular energy production, tissue integrity, and the regulation of oxidative stress, which is vital for maintaining reproductive health. Cytochrome c oxidase, which is a key enzyme in mitochondrial respiration, relies on copper to facilitate the transfer of electrons in the electron transport chain, thereby producing the ATP necessary for cellular energy. Lysyl oxidase, another copper-dependent enzyme, is involved in the cross-linking of collagen and elastin, which provides structural stability to connective tissues, including those in the reproductive organs, such as the ovaries, uterus, and testes. This function is essential for the proper structural integrity and function of the tissues involved in reproduction. In the female reproductive system, copper has critical roles in regulating steroidogenesis - the process by which ovarian cells produce steroid hormones such as estrogen and progesterone. These hormones are vital

for folliculogenesis, ovulation, and luteal phase support. Copper plays a regulatory role in the enzymes responsible for the biosynthesis of these hormones, ensuring that appropriate hormonal levels are maintained throughout the menstrual cycle. Estrogen, produced primarily during the follicular phase, is necessary for the growth and maturation of ovarian follicles, while progesterone, produced in the luteal phase, is crucial for the preparation of the endometrium for implantation and maintaining early pregnancy. Dysregulation of copper levels can thus lead to hormonal imbalances, potentially disrupting normal ovarian function, menstrual cycle regularity, and fertility. Moreover, copper is involved in the process of uterine vascularization, promoting the formation of blood vessels that are necessary for the endometrial receptivity required for successful embryo implantation. The role of copper in angiogenesis, through mechanisms such as the regulation of vascular endothelial growth factor (VEGF), is critical for ensuring an adequate blood supply to the endometrium. This, in turn, facilitates the development of a receptive uterine environment, optimizing conditions for the embryo to implant and thrive during early pregnancy. Without sufficient copper, the uterine lining may not develop the necessary blood supply, leading to suboptimal conditions for implantation and early pregnancy maintenance. [27, 28] In the male reproductive system, copper is equally essential, particularly in sperm maturation. Copper is involved in the final stages of spermatogenesis, where it supports the proper formation of sperm motility structures, such as the flagellum, which is necessary for sperm to swim toward and fertilize an egg. Copper also plays a role in maintaining the structural integrity and function of sperm, including its DNA integrity. Copper acts as a cofactor for the enzyme superoxide dismutase, which is crucial in protecting sperm cells from oxidative damage by neutralizing harmful free radicals. The role of copper in protecting sperm DNA from oxidative stress is particularly important, as damaged DNA can lead to decreased sperm viability and motility, thereby impairing fertility. Additionally, copper is involved in maintaining mitochondrial function in sperm cells, ensuring that the energy required for motility and fertilization is available. Mitochondria are vital for sperm energy production, and copper's role in mitochondrial health is critical for sperm performance. [29, 30] Copper deficiency has been associated with impaired fertility in both men and women.

Manganese

Manganese is a trace element that plays a critical role in reproductive health, particularly in the protection of cellular integrity and function. It is an essential cofactor for the enzyme manganese superoxide dismutase (MnSOD), a key antioxidant enzyme located in the mitochondria, which helps neutralize reactive oxygen species (ROS) and prevents oxidative

damage. [31] Oxidative stress, if unregulated, can lead to cellular damage, particularly in oocytes and sperm, resulting in compromised gamete quality, DNA fragmentation, and reduced fertility outcomes. Manganese is especially important in maintaining mitochondrial function, as mitochondria are central to energy production and apoptosis regulation in reproductive cells. Furthermore, manganese influences embryonic development by promoting the cleavage and quality of early-stage embryos. Clinical studies have shown that manganese supplementation is associated with improved embryo quality, enhanced blastocyst formation, and increased implantation rates, particularly in individuals undergoing assisted reproductive technologies (ART). Additionally, manganese is involved in the regulation of endometrial receptivity, improving the uterine environment for successful embryo implantation. As manganese deficiencies can lead to impaired reproductive outcomes, optimizing manganese levels through diet or supplementation may offer therapeutic potential in enhancing fertility and supporting early pregnancy success. [32] However, as with any micronutrient, careful balance is necessary, as excessive manganese can also lead to toxicity and adverse health effects, including neurotoxicity and disrupted metabolic functions. [33] Therefore, maintaining appropriate manganese levels is essential for optimal reproductive function. [34]

Magnesium

Magnesium is an essential divalent cation that acts as a cofactor for over 300 enzymatic reactions, many of which are fundamental to cellular metabolism. It is critical for the structural stability of adenosine triphosphate (ATP), as magnesium-ATP is the active form utilized in most biochemical reactions. Furthermore, magnesium plays a pivotal role in DNA synthesis and repair by stabilizing nucleic acid structures and acting as a cofactor for DNA polymerases and ligases. It also modulates ion channel function, including calcium and potassium channels, thereby influencing cell excitability, neuromuscular conduction, and hormone secretion. [35] In male reproductive physiology, magnesium contributes to sperm motility and capacitation - a process that enables sperm to fertilize the oocyte. It exerts its effects through regulation of intracellular calcium fluxes, membrane potential, and cyclic adenosine monophosphate (cAMP) signaling pathways within spermatozoa. Studies have correlated hypomagnesemia with decreased sperm viability, morphology, and reduced fertilization potential. [36] In women, magnesium's role is multifaceted. It enhances insulin receptor sensitivity and improves glucose homeostasis, particularly beneficial in women with polycystic ovary syndrome (PCOS), a condition often characterized by insulin resistance and hyperandrogenism. Magnesium decreases circulating androgens by attenuating insulin-mediated ovarian theca cell steroidogenesis. Additionally, it supports oocyte maturation via modulation of granulosa cell

function and oxidative stress regulation. In assisted reproductive technologies (ART), magnesium supplementation has been associated with restoration of ovulatory cycles in anovulatory women with PCOS and has improved endometrial receptivity, as evidenced by increased endometrial thickness and higher implantation rates during in vitro fertilization (IVF) cycles. [37] Moreover, magnesium exerts anti-inflammatory and antioxidative effects that may reduce systemic inflammation - a contributor to subfertility in both sexes. It also modulates the hypothalamic–pituitary–gonadal axis, potentially influencing gonadotropin secretion and steroidogenesis. [38, 39]

Interactions Among Microelements

The biological efficacy of trace elements is intricately linked, with numerous microelements exerting interdependent effects that influence one another's absorption, bioavailability, utilization, and overall physiological outcomes. [40] This interconnectedness underscores the complex nature of micronutrient interactions, which are crucial for maintaining optimal reproductive health. For instance, zinc and copper, two essential trace elements, share competition for absorption sites within the intestinal lining due to their similar chemical structures and properties. This competition can significantly affect their bioavailability and functional roles within the body, especially in tissues where they are involved in critical processes like antioxidant defense, enzyme activity, and cellular signaling. [41] As such, an imbalance between zinc and copper levels - particularly a high copper-to-zinc ratio - can lead to detrimental effects such as increased oxidative stress, which has been implicated in impaired fertility outcomes, including reduced sperm quality in men and ovarian dysfunction in women. [10] Similarly, iron and manganese also compete for divalent metal transporters (DMT1) at the cellular level, further complicating the regulation of their respective bioavailability and functional roles. Both minerals are essential for proper cellular respiration, oxidative phosphorylation, and mitochondrial function. [42] For instance, iron deficiency anemia has been linked to poor ovarian reserve and diminished egg quality, whereas manganese deficiency can impair oocyte maturation and sperm motility. Selenium and zinc, on the other hand, act synergistically to support a range of cellular processes, particularly in relation to DNA repair, antioxidant defense, and the modulation of redox balance. [43, 44] Disruptions in the balance of zinc, copper, iron, manganese, and selenium can lead to cellular dysfunction, genomic instability, and reduced reproductive potential. Furthermore, given the crucial role these elements play in processes like hormone synthesis, immune function, and mitochondrial energy production, their synergistic effects extend beyond reproductive tissues, influencing overall health and metabolic homeostasis. As such, a comprehensive approach to micronutrient

assessment and supplementation, focusing on the interactions between these trace elements, is critical for
improving fertility outcomes and ensuring the long-term health of both parents and offspring.

Conclusion

Microelements are fundamental players in human reproductive physiology, exerting crucial regulatory roles across the entire reproductive process, from gamete development to implantation and pregnancy maintenance. Trace elements such as zinc, selenium, iodine, iron, copper, manganese, and magnesium are involved in a variety of biological functions, including antioxidant defense, mitochondrial function, hormone synthesis, and DNA repair, all of which are critical for maintaining cellular integrity and ensuring successful reproduction. Nutritional optimization of these microelements, whether through diet or supplementation, can have a significant impact on natural conception rates and the success of assisted reproductive technologies (ART). It is essential to approach fertility treatment from a multidisciplinary perspective, integrating knowledge from nutrition, endocrinology, and reproductive medicine. Achieving optimal reproductive outcomes requires a holistic approach, wherein proper nutritional support is coupled with medical interventions tailored to individual needs. This comprehensive approach has the potential to enhance fertility outcomes, improve ART success, and promote the overall health of both parents and offspring.

Disclosure

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