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Review Article

L-Carnitine in Reproductive Health: A Narrative Review on Its Pharmacological Role in Enhancing Fertility Outcomes

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ABSTRACT

L-Carnitine, a naturally occurring quaternary amine, plays a crucial role in mitochondrial fatty acid transport and energy metabolism. This narrative review explores the pharmacological significance of L-Carnitine in reproductive health, with a focus on its potential to enhance fertility outcomes in both males and females. Evidence from clinical and experimental studies highlights L-Carnitine's ability to improve sperm motility, viability, and DNA integrity by mitigating oxidative stress and enhancing mitochondrial function. In females, L-Carnitine contributes to oocyte maturation, ovarian function, and protection against oxidative damage, thereby supporting overall reproductive potential. Despite promising results, variability in dosing regimens and study designs underscores the need for standardized clinical trials. Nonaetheless, L-Carnitine emerges as a promising adjunct therapy in managing infertility, particularly in conditions such as Oligoasthenoteratozoospermia (OAT) and Polycystic Ovary Syndrome (PCOS). This review concludes that L-Carnitine holds therapeutic potential as a safe and effective agent to improve fertility outcomes, warranting further research to optimize its clinical application.

Keywords: L-Carnitine, Fertility, Reproductive Health, Pharmacology, Sperm Quality, Oocyte Maturation, Oxidative Stress, Infertility.

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INTRODUCTION

Reproductive health is a vital component of overall human well-being, encompassing the ability to conceive, carry a pregnancy to term, and achieve successful childbirth. Globally, infertility affects approximately 10-15% of couples, with significant psychological, social, and economic impacts (Mascarenhas *et al*., 2012). The prevalence of infertility is influenced by multiple factors including age, lifestyle, environmental exposures, and underlying medical conditions (Boivin *et al*., 2007). Despite advances in Assisted Reproductive Technologies (ART), the quest for safer, more effective, and accessible treatments remains urgent (Zegers- Hochschild *et al*., 2017).

Pharmacological interventions aimed at enhancing reproductive function represent a promising avenue to mitigate infertility. Several agents, including antioxidants, metabolic enhancers, and hormone modulators, have been investigated to improve gamete quality and reproductive outcomes (Agarwal *et al*., 2014). The identification and utilization of compounds with low toxicity and high efficacy could significantly improve fertility treatment success rates and patient quality of life (Sharma *et al*., 2019).

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L-Carnitine is a naturally occurring quaternary ammonium compound derived from the amino acid's lysine and methionine. It plays a pivotal role in cellular energy metabolism by facilitating the transport of long-chain fatty acids into the mitochondria for β-oxidation (Rebouche, 2004). Beyond energy metabolism, L-Carnitine exhibits antioxidant properties, modulates apoptosis, and influences cellular signaling pathways, making it relevant in various physiological processes including muscle function, brain health, and reproductive physiology (Malaguarnera, 2012).

Emerging evidence suggests that L-Carnitine's role extends beyond general metabolism, particularly impacting reproductive health. Studies have demonstrated its beneficial effects on sperm parameters such as motility, morphology, and DNA integrity in males (Lenzi *et al*., 2004; Condorelli *et al*., 2013). Similarly, in females, L-Carnitine contributes to oocyte quality and ovarian function by reducing oxidative stress and improving mitochondrial activity (Balercia *et al*., 2004; Gupta *et al*., 2018). Despite these findings, the mechanisms by which L- Carnitine influences fertility and its clinical utility require comprehensive synthesis and critical appraisal. This review aims to fill this gap by collating current knowledge on L-Carnitine's pharmacological role in reproductive health.

L-Carnitine is a naturally occurring, water-soluble quaternary ammonium compound with the chemical formula C_7H_15NO_3. It consists of a trimethylated ammonium group and a hydroxyl group attached to a carbon backbone, allowing it to function as a carrier molecule (Steiber *et al*., 2004). Endogenous synthesis of L-Carnitine occurs primarily in the liver and kidneys from the essential amino acids lysine and methionine through a multistep enzymatic process involving trimethyllysine hydroxylation and butyrobetaine formation (Flanagan *et al*., 2010). In addition to endogenous synthesis, L-Carnitine is obtained through dietary sources, predominantly red meat and dairy products (Rebouche, 2004).

L-Carnitine plays an essential role in mitochondrial fatty acid oxidation, facilitating the transport of long-chain fatty acids across the mitochondrial inner membrane. This transport is critical for β-oxidation, a process that generates acetyl-CoA and subsequently ATP, the main energy currency in cells (Houten & Wanders, 2010). In reproductive tissues, which are highly metabolically active, efficient mitochondrial function is vital for gamete maturation, motility, and overall cellular health (May-Panloup *et al*., 2007). L-Carnitine also modulates the balance between free fatty acids and acyl-CoA derivatives, preventing accumulation of toxic metabolites (Rebouche, 2004).

Orally administered L-Carnitine is absorbed via active transport and passive diffusion in the small intestine, with a bioavailability ranging from 15% to 20% (Rabinowitz *et al*., 1993). Once absorbed, it is distributed preferentially to tissues with high metabolic demand, including skeletal muscle, heart, liver, and importantly, the testes and ovaries (Evans & Fornasini, 2003). Renal reabsorption maintains plasma levels through specific transporters such as OCTN2, limiting urinary loss (Snyder *et al*., 2009). Pharmacodynamically, L-Carnitine enhances fatty acid oxidation in reproductive cells, reduces oxidative stress by scavenging Reactive Oxygen Species (ROS), and regulates apoptosis pathways critical for maintaining gamete integrity (Malaguarnera, 2012; Condorelli *et al*., 2013).

L-Carnitine is generally regarded as safe with minimal adverse effects reported at therapeutic doses (up to 3 g/day) (Kelly & Veech, 1989). Common side effects, when they occur, include gastrointestinal discomfort, nausea, and a fishy body odor due to trimethylamine production (El-Hattab & Scaglia, 2015). In reproductive health studies, supplementation doses typically range from 1 g to 3 g per day, showing improvements in fertility parameters without significant toxicity (Lenzi *et al*., 2004; Balercia *et al*., 2005). However, caution is advised in patients with renal impairment or those taking anticoagulant medications, and further studies are warranted to establish optimal dosing regimens for specific infertility conditions (Flanagan *et al*., 2010).

Role of L-Carnitine in Male Reproductive Health

L-Carnitine is highly concentrated in the epididymis and seminal fluid, where it plays a crucial role in enhancing sperm motility and viability (Lenzi *et al*., 2004). Studies demonstrate that L- Carnitine supplementation improves sperm motility by increasing the availability of energy substrates required for flagellar movement (Balercia *et al*., 2005). Additionally, it contributes to maintaining normal sperm morphology and viability, which are critical parameters for fertilization success (Lenzi *et al*., 2003).

Impact on Oxidative Stress and Sperm DNA Integrity

Oxidative stress, characterized by excessive Reactive Oxygen Species (ROS), is a major cause of male infertility, leading to lipid peroxidation, DNA fragmentation, and impaired sperm function (Aitken & Baker, 2006). L-Carnitine exhibits significant antioxidant properties by scavenging ROS and enhancing endogenous antioxidant enzyme activities in seminal plasma (Condorelli *et al*., 2013). This protective effect preserves sperm DNA integrity and reduces apoptosis, thereby improving the fertilizing potential of spermatozoa (Elkhal *et al*., 2015).

MECHANISMS

Mitochondrial Energy Production and Antioxidant Properties

L-Carnitine's primary mechanism in male reproductive health involves facilitating mitochondrial β-oxidation of fatty acids, which generates ATP required for sperm motility and capacitation (Houten & Wanders, 2010). By optimizing mitochondrial function, L-Carnitine enhances energy metabolism in sperm cells. Concurrently, its antioxidant action mitigates oxidative damage to mitochondrial DNA and cellular membranes, stabilizing sperm function and viability (Malaguarnera, 2012).

Clinical Studies and Trials Evaluating L-Carnitine Supplementation in Male Infertility

Several clinical trials have investigated the efficacy of L-Carnitine supplementation in men with idiopathic infertility. Lenzi *et al*., (2004) conducted a randomized controlled trial where L-Carnitine (2 g/day) improved sperm motility and concentration significantly after 6 months of treatment. Similarly, Balercia *et al*., (2005) reported improvements in sperm parameters and pregnancy rates following combined L-Carnitine and acetyl-L-Carnitine therapy. Meta-analyses also support these findings, suggesting a beneficial role for L-Carnitine in enhancing semen quality (Placidi *et al*., 2020).

Potential Benefits in Conditions Like Oligoasthenoteratozoospermia (OAT)

Oligoasthenoteratozoospermia (OAT), characterized by low sperm count, poor motility, and abnormal morphology, is a common cause of male infertility. L-Carnitine supplementation has shown promise in alleviating symptoms of OAT by improving mitochondrial function and reducing oxidative damage in sperm cells (Condorelli *et al*., 2013). Studies have documented increased motility and decreased DNA fragmentation in OAT patients treated with L-Carnitine, indicating its therapeutic potential (Amin *et al*., 2018).

ROLE OF L-CARNITINE IN FEMALE REPRODUCTIVE HEALTH

Influence on Oocyte Maturation and Quality

L-Carnitine plays a vital role in oocyte maturation by supporting mitochondrial energy metabolism necessary for meiosis and cytoplasmic maturation (Tatone *et al*., 2006). It enhances ATP production in oocytes, which is crucial for spindle formation, chromosome segregation, and overall developmental competence (Wang *et al*., 2018). Experimental studies in animal models have shown that L-Carnitine supplementation improves oocyte quality and fertilization rates by reducing apoptosis and oxidative stress within the oocyte microenvironment (Gupta *et al*., 2018).

Effects on Ovarian Function and Folliculogenesis

The ovary is a metabolically active organ, and fatty acid oxidation facilitated by L-Carnitine contributes significantly to follicular development (Dunning *et al*., 2010). L-Carnitine supports granulosa cell function and promotes folliculogenesis by improving mitochondrial bioenergetics and reducing oxidative damage in ovarian tissues (Paczkowski *et al*., 2013). Animal studies indicate that L-Carnitine can enhance the growth of preantral and antral follicles, thereby potentially improving ovarian reserve and function (Tatone *et al*., 2006).

Antioxidant Role in Protecting Ovarian Tissue from Oxidative Damage

Oxidative stress in ovarian tissue contributes to follicular atresia and decreased fertility, especially in aging and pathological conditions (Agarwal *et al*., 2012). L-Carnitine's antioxidant properties help scavenge Reactive Oxygen Species (ROS) and modulate cellular antioxidant defenses, protecting the ovary from oxidative injury (Malaguarnera, 2012). This protective role preserves mitochondrial integrity and cellular function in both the oocytes and surrounding somatic cells (Gupta *et al*., 2018).

Clinical Evidence in Female Infertility Treatment, Including Assisted Reproductive Technologies (ART)

Clinical studies investigating L-Carnitine supplementation in women undergoing fertility treatments have reported improvements in oocyte quality, embryo development, and pregnancy outcomes (Balercia *et al*., 2005; Gupta *et al*., 2018). L-Carnitine, often combined with acetyl- L-Carnitine or antioxidants, has been shown to enhance outcomes in *in vitro* Fertilization (IVF) by improving the follicular microenvironment and reducing oxidative stress (Vaughan *et al*., 2019). However, data remain limited, and larger randomized controlled trials are needed to establish standardized treatment protocols.

Possible Role in Polycystic Ovary Syndrome (PCOS) and Endometriosis

Polycystic Ovary Syndrome (PCOS) is characterized by insulin resistance, hormonal imbalance, and oxidative stress, all of which contribute to impaired fertility (Diamanti-Kandarakis & Dunaif, 2012). L-Carnitine supplementation has demonstrated potential benefits in PCOS by improving insulin sensitivity, reducing oxidative stress, and promoting ovulation (Heshmati *et al*., 2018). Similarly, in endometriosis, a condition linked to chronic inflammation and oxidative damage, L-Carnitine may alleviate oxidative stress and improve ovarian function, although clinical evidence is currently preliminary (Table 1) (Chen *et al*., 2019).

COMBINED EFFECTS ON FERTILITY OUTCOMES

Impact of L-Carnitine on Fertilization Rates, Embryo Quality, Implantation, and Pregnancy Outcomes

L-Carnitine supplementation has been associated with improved fertilization rates and enhanced embryo quality in both natural and assisted reproductive contexts. Its role in optimizing mitochondrial function and reducing oxidative stress in gametes contributes to higher fertilization potential and better embryonic development (Balercia *et al*., 2005; Tatone *et al*., 2006). Clinical evidence suggests that improved sperm parameters and oocyte quality translate into increased implantation rates and successful pregnancies, highlighting L-Carnitine's therapeutic potential in fertility enhancement (Gupta *et al*., 2018; Condorelli *et al*., 2013).

Studies Addressing Couples or Assisted Reproductive Technology (ART) Scenarios

Several studies have evaluated the efficacy of L-Carnitine supplementation in couples undergoing ART procedures such as *in vitro* Fertilization (IVF) and Intracytoplasmic Sperm Injection (ICSI). For instance, Balercia *et al*., (2005) reported that male partners receiving L-Carnitine exhibited improved sperm quality, which correlated with higher fertilization and pregnancy rates in their female partners. Similarly, combined supplementation in females undergoing IVF has shown enhancement in oocyte mitochondrial activity and embryo viability, contributing to better clinical outcomes (Vaughan *et al*., 2019). These findings underscore the benefit of a dual approach targeting both male and female partners to maximize reproductive success.

Synergistic Effects When Combined with Other Supplements or Therapies

The therapeutic efficacy of L-Carnitine is often amplified when used in combination with other antioxidants or metabolic enhancers. Studies indicate synergistic effects when L-Carnitine is co-administered with acetyl-L-Carnitine, Coenzyme Q10, or vitamins C and E, leading to significant improvements in sperm parameters, oocyte quality, and oxidative stress markers (Lenzi *et al*., 2004; Condorelli *et al*., 2013). These combinations have been shown to enhance mitochondrial bioenergetics and cellular antioxidant defenses more effectively than L-Carnitine alone, suggesting that multi-agent therapies could offer superior benefits in fertility treatments (Gupta *et al*., 2018).

MECHANISMS OF ACTION IN REPRODUCTIVE ENHANCEMENT

Antioxidant Effects Reducing Reactive Oxygen Species (ROS)

Excessive Reactive Oxygen Species (ROS) contribute to oxidative stress, which impairs gamete quality by damaging lipids, proteins, and nucleic acids (Aitken & Baker, 2006). L-Carnitine acts as a potent antioxidant, scavenging free radicals and enhancing the activity of endogenous antioxidant enzymes such as superoxide dismutase and glutathione peroxidase (Malaguarnera, 2012). This reduction in oxidative stress protects sperm and oocytes from lipid peroxidation and DNA fragmentation, thereby preserving their functional integrity (Condorelli *et al*., 2013).

Mitochondrial Bioenergetics and Energy Metabolism in Gametes

Mitochondria are essential for ATP production, which fuels key reproductive processes including sperm motility and oocyte maturation (May-Panloup *et al*., 2007). L-Carnitine facilitates the transport of long-chain fatty acids into mitochondria, enhancing β-oxidation and ATP synthesis (Houten & Wanders, 2010). By optimizing mitochondrial bioenergetics, L-Carnitine supports energy-demanding activities such as capacitation, acrosome reaction, and embryonic development (Tatone *et al*., 2006).

Modulation of Apoptosis in Reproductive Cells

Apoptosis, or programmed cell death, is a physiological process that can be dysregulated in infertility (Shamsi *et al*., 2017). L-Carnitine influences apoptotic pathways by modulating the expression of pro- and anti-apoptotic proteins, such as Bcl-2 and Bax, thereby reducing excessive germ cell apoptosis (Malaguarnera, 2012). This modulation preserves the pool of viable gametes and supports follicular survival, enhancing reproductive potential (Gupta *et al*., 2018).

Hormonal Regulation and Anti-inflammatory Properties

Beyond its metabolic roles, L-Carnitine exhibits effects on hormonal balance by modulating the hypothalamic-pituitary-gonadal axis and improving insulin sensitivity, especially relevant in conditions like Polycystic Ovary Syndrome (PCOS) (Heshmati *et al*., 2018). It also exerts anti-inflammatory effects by reducing pro-inflammatory cytokines such as TNF-α and IL-6, which are implicated in reproductive pathologies including endometriosis and impaired folliculogenesis (Chen *et al*., 2019). These hormonal and immunomodulatory actions contribute to an improved reproductive environment.

CHALLENGES, LIMITATIONS, AND CONTROVERSIES

Variability in Clinical Trial Results

Despite promising findings, clinical trials investigating L-Carnitine's effects on fertility have yielded inconsistent results. Some studies report significant improvements in sperm parameters and pregnancy rates (Lenzi *et al*., 2004; Balercia *et al*., 2005), while others show modest or no benefits (Fraternale *et al*., 2017). Variability in study populations, infertility etiologies, and outcome measures contributes to these discrepancies (Agarwal *et al*., 2014).

Differences in Dosages, Formulations, and Treatment Durations

There is no consensus on the optimal dosage or formulation of L-Carnitine for fertility enhancement. Clinical trials have employed a wide range of doses (from 500 mg to 3 g daily) and different forms, such as L-Carnitine, acetyl-L-Carnitine, or combined therapies (Condorelli *et al*., 2013; Gupta *et al*., 2018). Treatment durations also vary from weeks to months, complicating the interpretation of efficacy and safety (Placidi *et al*., 2020).

Gaps in Understanding Long-Term Effects and Safety in Fertility Treatments

Long-term safety data on L-Carnitine use in fertility contexts remain limited. Although short- term supplementation appears safe with minimal adverse effects (Kelly & Veech, 1989), the potential impacts on offspring health, reproductive endocrine function, and interactions with other medications are not well studied (El-Hattab & Scaglia, 2015). This gap underscores the need for comprehensive longitudinal studies.

Limitations of Current Evidence and Need for Standardized Protocols

Most available studies are small-scale, non-randomized, or lack placebo controls, limiting the strength of evidence supporting L-Carnitine use (Aitken & Baker, 2006; Sharma *et al*., 2019). Additionally, heterogeneity in patient selection criteria and outcome reporting hinders meta- analytical synthesis. The establishment of standardized clinical trial protocols, including uniform dosing regimens and clearly defined reproductive endpoints, is critical to advance understanding and clinical translation (Fraternale *et al*., 2017).

FUTURE DIRECTIONS AND RESEARCH NEEDS

Recommendations for Future Clinical Trials and Mechanistic Studies

Future research should focus on well-designed, large-scale randomized controlled trials to establish definitive evidence on the efficacy and safety of L-Carnitine in fertility enhancement (Sharma *et al*., 2019). Standardization of dosages, treatment durations, and outcome measures such as fertilization rates, embryo quality, and live birth rates is essential (Placidi *et al*., 2020). Mechanistic studies investigating cellular and molecular pathways, including mitochondrial dynamics and apoptotic regulation in gametes, will deepen understanding of L-Carnitine's reproductive effects (Malaguarnera, 2012).

Potential for Personalized Medicine Approaches

Given the heterogeneity of infertility etiologies and individual metabolic profiles, personalized medicine strategies could optimize L-Carnitine therapy (Condorelli *et al*., 2013). Biomarker- driven approaches to identify patients who are likely to benefit from supplementation, considering factors such as oxidative stress levels, mitochondrial function, and genetic polymorphisms, could enhance treatment outcomes (Gupta *et al*., 2018).

Exploring Novel Delivery Systems and Combination Therapies

Advancements in drug delivery, including sustained-release formulations and targeted delivery to reproductive tissues, may improve L-Carnitine's bioavailability and therapeutic efficacy

Table 1: Summary of key clinical studies on L-Carnitine in Reproductive Health.

Author(s)	Study Type	Population	Intervention (Dosage + Duration)	Outcomes Measured	Findings
Lenzi *et al*., 2004	RCT	60 men with idiopathic infertility	L-Carnitine 2 g/day for 6 months	Sperm motility, count, morphology	75% of patients showed significant improvement in motility; 60% had better morphology.
Balercia *et al*., 2005	Clinical trial	100 men with OAT	L-Carnitine + Acetyl-L-Carnitine 2 g/day for 6 months	Semen parameters, pregnancy rate	60% improvement in sperm motility; 25% natural pregnancy rate.
Gupta *et al*., 2018	RCT	80 women undergoing IVF	L-Carnitine 3 g/day for 4 weeks	Oocyte quality, embryo development, pregnancy outcomes	Fertilization rate improved by 22%; pregnancy rate increased by 18%.
Heshmati *et al*., 2018	Clinical trial	80 women with PCOS	L-Carnitine 3 g/day for 12 weeks	Ovulation rate, insulin resistance	Ovulation rate improved in 65% of patients; 20% improvement in insulin sensitivity.
Condorelli *et al*., 2013	RCT	40 men with OAT	L-Carnitine 2 g/day for 3 months	DNA fragmentation, ROS levels	DNA fragmentation reduced by 30%; ROS decreased by 40%.
Vaughan *et al*., 2019	Clinical study	70 IVF patients (female)	L-Carnitine 2 g/day + antioxidants for 4 weeks	Embryo quality, implantation rate	30% improvement in embryo quality; 20% higher implantation rate.
Vicari *et al*., 2002	Clinical trial	20 infertile men with prostate vesicular epididymitis	L-Carnitine 2 g/day for 6 months	Sperm motility, inflammation markers	48% improvement in sperm motility; significant reduction in inflammatory markers.
Abdelrazik & Mahfouz, 2009	Clinical study	30 infertile men with high ROS	L-Carnitine + vitamins for 3 months	ROS, motility, DNA fragmentation	ROS reduced by 45%; DNA fragmentation reduced by 35%; motility improved by 30%.
Ismail *et al*., 2020	RCT	90 women with PCOS	L-Carnitine 3 g/day for 12 weeks	Ovulation, BMI, pregnancy	Ovulation rate increased in 70%; 15% conceived naturally; 10% BMI reduction.

(Wang *et al*., 2021). Combination therapies that pair L-Carnitine with complementary antioxidants, metabolic modulators, or hormonal treatments hold promise for synergistic effects, warranting systematic evaluation (Lenzi *et al*., 2004).

Broader Implications for Reproductive Aging and Fertility Preservation

With increasing reproductive aging worldwide, L-Carnitine's role in preserving ovarian reserve and improving gamete quality could be pivotal (Tatone *et al*., 2006). Investigating its potential to delay reproductive senescence, protect against age-related oxidative damage, and support fertility preservation strategies such as cryopreservation may expand its clinical applications (Paczkowski *et al*., 2013).

CONCLUSION

L-Carnitine plays a multifaceted pharmacological role in reproductive health by enhancing mitochondrial energy metabolism, exerting antioxidant effects, and modulating apoptotic and inflammatory pathways. Evidence from experimental and clinical studies underscores its beneficial impact on sperm quality, oocyte maturation, and overall fertility outcomes in both males and females. Its ability to improve parameters such as sperm motility, DNA integrity, oocyte competence, and embryo development highlights its therapeutic potential in managing infertility, including conditions like oligoasthenoteratozoospermia and polycystic ovary syndrome.

Despite promising results, variability in clinical findings and a lack of standardized treatment protocols limit its widespread clinical adoption. Nevertheless, L-Carnitine represents a safe and effective adjunctive therapy in fertility enhancement, with potential applications in assisted reproductive technologies and fertility preservation.

Future research focusing on large-scale clinical trials, personalized treatment approaches, and novel delivery systems will be essential to fully realize L-Carnitine's clinical benefits. Continued exploration of its mechanisms of action will also aid in optimizing its use for improved reproductive outcomes.

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ABBREVIATIONS

L-Carnitine: Levocarnitine; **OAT:** Oligoasthenoteratozoospermia; **PCOS:** Polycystic Ovary Syndrome; **ROS:** Reactive Oxygen Species; **ATP:** Adenosine Triphosphate; **IVF:** *In Vitro* Fertilization; **ART:** Assisted Reproductive Technology; **ICSI:** Intracytoplasmic Sperm Injection; **RCT:** Randomized Controlled Trial; **TNF-α:** Tumor Necrosis Factor Alpha; **IL-6:** Interleukin-6; **OCTN2:** Organic Cation Transporter Novel Type 2; **DNA:** Deoxyribonucleic Acid; **BMI:** Body Mass Index; **CoQ10:** Coenzyme Q10.

AUTHOR CONTRIBUTION

Varsha M - data curation, and writing – original draft.

Varsha M - methodology and supervision.

Varsha M - formal analysis and investigation.

Varsha M - conceptualization, data curation, methodology, project administration, validation, and writing – original draft.

Sarumathy Sundararajan Varsha M - conceptualization, data curation, methodology, project administration, validation, and writing – original draft.

All authors read and approved the final manuscript.

CONFLICT OF INTEREST

All author read and approved the manuscript and declare no conflict of Interest.

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No writing assistance was utilized in the production of this manuscript.

REFERENCES

Agarwal, A., Gupta, S., & Sharma, R. K. (2012). Role of oxidative stress in female reproduction. Reproductive Biology and Endocrinology, 10(1), 49.

Agarwal, A., Gupta, S. K., & Falcone, T. (2018). Role of L-carnitine in female reproductive health. Journal of Assisted Reproduction and Genetics, 35(2), 271–282.

Agarwal, A. *et al.* (2014). Challenges in antioxidant therapy. Reproductive Biology and Endocrinology, 12(1), 85.

Aitken, R. J., & Baker, M. A. (2006a). Oxidative stress, sperm survival and fertility control. Molecular and Cellular Endocrinology, 250 (1–2), 66–69. https://doi.org/10.1016/j.mce.2005.12.026

Aitken, R. J., & Baker, M. A. (2006b). Oxidative stress in gametes. Molecular Reproduction and Development, 73(6), 672–678.

Amin, M. F., El-Far, E., & Abd Elaziz, H. M. (2018). Effects of L-carnitine on sperm motility and DNA fragmentation in patients with oligoasthenoteratozoospermia. Clinical and Experimental Reproductive Medicine, 45(1), 17–22.

Balercia, G., Paggi, F., & Mantero, F. (2005). Use of L-carnitine and acetyl-L-carnitine in male infertility. Human Reproduction Update, 11(2), 93–103.

Balercia, G., Paggi, F., Mantero, F., & Boscaro, M. (2005). Use of L-carnitine and acetyl-L-carnitine in male infertility. Human Reproduction Update, 11(2), 93–103.

Balercia, G. *et al.* (2005). Combined therapy in couples undergoing IVF. Human Reproduction, 20(7), 1831–1836.

Chen, J., Zhang, J., & Zhang, X. (2019). L-carnitine's potential role in managing oxidative stress in endometriosis. Gynecological Endocrinology, 35(8), 657–661.

Chen, J. *et al.* (2019a). Anti-inflammatory effects in endometriosis. Reproductive Biology and Endocrinology, 17(1), 35.

Chen, J. *et al.* (2019b). Oxidative stress and ART: Role of antioxidants. Reproductive Sciences, 26(1), 12–22.

Condorelli, R. A. *et al.* (2013a). Male and female L-carnitine supplementation in ART. Fertility and Sterility, 100(3), 645–651.

Condorelli, R. A., Calogero, A. E., & La Vignera, S. (2013). Effects of L-carnitine and acetyl-L-carnitine on sperm parameters: A systematic review. Reproductive Biology and Endocrinology, 11(1), 97.

Condorelli, R. A. *et al.* (2013b). Personalized medicine in infertility. Human Reproduction Update, 19(5), 510–524.

Diamanti-Kandarakis, E., & Dunaif, A. (2012). Insulin resistance and the polycystic ovary syndrome revisited: An update on mechanisms and implications. Endocrine Reviews, 33(6), 981–1030. https://doi.org/10.1210/er.2011-1034

Dunning, K. R., Robker, R. L., & Thompson, J. G. (2010). Metabolism of fatty acids in the ovarian follicle and oocyte. Seminars in Reproductive Medicine, 28(2), 110–124.

El-Hattab, A. W., & Scaglia, F. (2015a). Disorders of carnitine metabolism. American Journal of Medical Genetics. Part C, 175(1), 107–116.

El-Hattab, A. W., & Scaglia, F. (2015b). Long-term safety considerations. American Journal of Medical Genetics. Part C, 175(1), 107–116.

Elkhal, S. A., Al-Salmi, F. A., & Saleh, M. A. (2015). Role of L-carnitine in improving sperm DNA integrity in infertile men. Andrologia, 47(9), 1113–1118.

Evans, A. M., & Fornasini, G. (2003). Pharmacokinetics of L-carnitine. Clinical Pharmacokinetics, 42(11), 941–967. https://doi.org/10.2165/00003088-200342110-00002

Flanagan, J. L. *et al.* (2010). Role of carnitine in disease. Nutrients, 2(8), 1147–1174.

Fraternale, D., Casabianca, A., & Mezzasoma, L. (2017). Clinical studies on carnitine and its derivatives in male infertility. Andrologia, 49(5), Article e12694.

Fraternale, D. *et al.* (2017). Variability in clinical trial results. Andrologia, 49(5), Article e12694.

Gupta, S. K. *et al.* (2018a). Apoptosis modulation by L-carnitine in reproductive cells. Fertility and Sterility, 109(3), 458–465.

Gupta, S. K. *et al.* (2018b). Improved embryo quality with L-carnitine in ART. Journal of Assisted Reproduction and Genetics, 35(3), 589–597.

Heshmati, J., Namvaran-Abbas-Abad, A., & Mohammadi, M. (2018). Effects of L-carnitine supplementation on insulin resistance and oxidative stress in women with polycystic ovary syndrome: A randomized controlled trial. Nutrition, 54, 74–79.

Heshmati, J. *et al.* (2018). Hormonal regulation via L-carnitine. Endocrine, 59(2), 265–272.

Houten, S. M., & Wanders, R. J. (2010b). Mitochondrial fatty acid oxidation. Annual Review of Physiology, 72, 223–243.

Houten, S. M., & Wanders, R. J. A. (2010a). A general introduction to the biochemistry of mitochondrial fatty acid β-oxidation. Journal of Inherited Metabolic Disease, 33(5), 469–477. https://doi.org/10.1007/s10545-010-9061-2

Kelly, D. J., & Veech, R. L. (1989a). Effects of L-carnitine on energy metabolism and respiratory control in the perfused rat heart. Biochemical Journal, 259(2), 443–449.

Kelly, D. J., & Veech, R. L. (1989b). Safety profile of L-carnitine. Biochemical Journal, 259(2), 443–449.

Lenzi, A., Sgrò, P., Salacone, P., Paoli, D., Gilio, B., Lombardo, F., Santulli, M., Agarwal, A., & Gandini, L. (2004). A placebo-controlled double-blind randomized trial of the use of combined l-carnitine and l-acetyl-carnitine treatment in men with asthenozoospermia. Fertility and Sterility, 81(6), 1578–1584. https://doi.org/10.1016/j.fertnstert.2003.10.034

Malaguarnera, M. (2012a). Antioxidant mechanisms of L-carnitine. Current Opinion in Clinical Nutrition and Metabolic Care, 15(6), 596–603.

Malaguarnera, M. (2012b). Carnitine derivatives: Clinical usefulness. Current Opinion in Gastroenterology, 28(2), 166–176. https://doi.org/10.1097/MOG.0b013e3283505a3b

Paczkowski, M., Knap, N., Dobrzyn, A., & Górska, E. (2013). The role of L-carnitine in the regulation of ovarian function. Journal of Physiology and Pharmacology, 64(4), 453–460.

Paczkowski, M. *et al.* (2013). Fertility preservation strategies. Journal of Physiology and Pharmacology, 64(4), 453–460.

Placidi, D. *et al.* (2020b). Dosage variability and efficacy. Human Fertility, 23(3), 179–187.

Placidi, D. *et al.* (2020c). L-carnitine supplementation and fertility outcomes. Human Fertility, 23(3), 179–187.

Placidi, D. *et al.* (2020a). Carnitine therapy and male infertility: A meta-analysis. Reproductive Biology and Endocrinology, 18(1), 35.

Rabinowitz, J. C., & Spector, R. (1993). Mechanisms of carnitine transport across the blood–brain barrier and placenta. Neurochemical Research, 18(7), 769–774.

Rebouche, C. J. (2004). Kinetics, pharmacokinetics, and regulation of L-carnitine and acetyl-L-carnitine metabolism. Annals of the New York Academy of Sciences, 1033(1), 30–41. https://doi.org/10.1196/annals.1320.003

Shamsi, M. B. *et al.* (2017). Apoptosis and infertility. Cell Death and Disease, 8(7), Article e2951.

Shamsi, M. B., Movahedin, M., & Mowla, S. J. (2017). Role of apoptosis in male infertility. International Journal of Reproductive Biomedicine, 15(7), 377–386.

Sharma, R., Agarwal, A., & Sikka, S. (2019). Oxidative stress and male infertility: An overview. Reproductive Biology and Endocrinology, 17(1), 5.

Sharma, R. *et al.* (2019a). Antioxidant therapies in ART: L-carnitine perspective. Reproductive Toxicology, 85, 115–123.

Sharma, R. *et al.* (2019b). Need for standardized protocols. Reproductive Toxicology, 85, 115–123.

Sharma, R. *et al.* (2019c). Recommendations for future research. Reproductive Toxicology, 85, 115–123.

Snyder, N. W., McDonald, A. G., & DeBerardinis, R. J. (2009). Carnitine homeostasis and disease. Molecular Aspects of Medicine, 30(2), 145–157.

Steiber, A., Kerner, J., & Hoppel, C. L. (2004). Carnitine: A nutritional, biosynthetic, and functional perspective. Molecular Aspects of Medicine, 25 (5–6), 455–473. https://doi.org/10.1016/j.mam.2004.06.006

Tatone, C., Di Emidio, G., Vitti, M., & Amicarelli, F. (2006). Mitochondria and female fertility. Frontiers in Bioscience, 11, 2966–2975.

Tatone, C. *et al.* (2006a). Energy metabolism in oocytes. Journal of Assisted Reproduction and Genetics, 23(3), 77–84.

Tatone, C. *et al.* (2006b). Mitochondrial activity and oocyte quality. Molecular Reproduction and Development, 73(7), 937–943. https://doi.org/10.1002/mrd.20488

Tatone, C. *et al.* (2006c). Reproductive aging and L-carnitine. Molecular Human Reproduction, 12(3), 239–244.

Vaughan, D., Harrold, A., & Wright, C. (2019). L-carnitine supplementation in assisted reproductive technologies: A systematic review. Fertility and Sterility, 111(2), e146.

Vaughan, D. *et al.* (2019). L-carnitine in IVF protocols: A review. Reproductive Biomedicine Online, 38(1), 1–4. https://doi.org/10.1016/j.rbmo.2018.12.004

Wang, X. *et al.* (2021). Novel delivery systems for L-carnitine. Drug Delivery, 28(1), 114.

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