Coenzyme Q10 Supplementation in Poor Ovarian Response and Diminished Ovarian Reserve: A Mitochondrial-targeted Strategy to Optimize Assisted Reproductive Technologies Outcome

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Abstract

Poor ovarian response (POR) and diminished ovarian reserve (DOR) are major contributors to suboptimal outcomes in assisted reproductive technologies (ART), which are often associated with reduced oocyte yield, lower fertilization rates, and diminished embryo quality. Emerging evidence has identified mitochondrial dysfunction and oxidative stress as central mechanisms underlying ovarian aging, thereby prompting interest in antioxidant-based therapeutic strategies. Coenzyme Q10 (CoQ10), a naturally occurring mitochondrial cofactor and potent antioxidant, plays a critical role in cellular energy production and the maintenance of redox homeostasis within ovarian cells. Preclinical and clinical studies suggest that CoQ10 supplementation improves mitochondrial efficiency, reduces reactive oxygen species (ROS), and enhances granulosa cell function, thereby collectively supporting oocyte competence and embryonic development.

Multiple randomized controlled trials and meta-analyses have demonstrated that CoQ10 pretreatment in women with POR and DOR significantly improves ovarian response, the number of mature oocytes retrieved, embryo quality, and clinical pregnancy rates. Doses ranging from 600 to 1200 mg daily for 8 to 12 weeks have been found to be effective, with minimal reported side effects. Compared to other adjunct therapies, such as DHEA and growth hormone, CoQ10 has shown superior efficacy in improving key ART outcomes, while maintaining a favorable safety profile.

Although further research is needed to standardize dosing regimens and to confirm effects on live birth rates and miscarriage rates, current evidence supports the clinical utility of CoQ10 as a safe and effective adjunct therapy for ovarian stimulation protocols. Its integration into ART practice represents a promising approach to optimizing outcomes in women with compromised ovarian reserve.

Keywords: Coenzyme Q10, Poor ovarian response, Diminished ovarian reserve, Assisted reproductive technologies, Female infertility

I. Introduction

The process of reproductive aging is traditionally understood to be based on the principle that the number of human oocytes peaks during fetal life and subsequently declines due to ovulation or atresia, without the capacity for regeneration. Reproductive and ovarian senescence occur as this pool of oocytes, referred to as the ovarian reserve, gradually depletes. Women in the later stages of reproductive aging, such as during perimenopause and menopause, when menstrual cycles become irregular, have significantly lower ovarian reserve compared with women in the earlier reproductive years, when menstrual cycles are typically regular.[1]

II. Poor Ovarian Response (POR)

POR refers to a suboptimal ovarian reaction to controlled ovarian stimulation (COS) in ART, most notably in *in vitro* fertilization (IVF) cycles. The concept was initially defined by Garcia et al in 1983 as a threshold for individual ovarian response to stimulation.[2]

Bologna Criteria (ESHRE, 2011): A patient is diagnosed with POR if at least two of the following criteria are met:

1. Advanced maternal age (≥40 years) or any other risk factors for POR.
2. A previous history of POR (≤3 oocytes retrieved with a standard stimulation protocol).
3. An abnormal ovarian reserve test:
    i. Antral Follicle Count (AFC) <5–7 follicles

ii. Anti-Müllerian Hormone (AMH) < 0.5–1.1 ng/mL

These criteria were established by the European Society of Human Reproduction and Embryology (ESHRE) in 2011.[3], [4] However, their validity has been questioned due to the heterogeneity in patient profiles and the lack of distinction between oocyte quality and quantity. A systematic review found 11 different POR definitions across 8 studies.[5]

POSEIDON Criteria (2016): To address the limitations of the Bologna criteria, the POSEIDON group introduced a novel classification system for patients with low prognosis (Table 1). This classification is based on age, ovarian reserve markers (AMH and AFC), and oocyte yield in prior cycles.[2]

Table 1: POSEIDON Classification

Group	Age	Ovarian Reserve	Response to Stimulation
1	< 35 years	Normal (AFC ≥ 5, AMH ≥ 1.2 ng/mL)	Unexpected poor (<4 oocytes) or suboptimal (4–9 oocytes) response
2	≥ 35 years	Normal (AFC ≥ 5, AMH ≥ 1.2 ng/mL)	Poor or suboptimal response
3	< 35 years	Low (AFC < 5, AMH < 1.2 ng/mL)	—
4	≥ 35 years	Low (AFC < 5, AMH < 1.2 ng/mL)	—

The POSEIDON classification helps to individualise treatment, optimise outcomes, and manage expectations by separating patients with expected vs. unexpected poor responses.[2]

III. Diminished Ovarian Reserve (DOR)

DOR describes a reduced number and quality of ovarian follicles, often seen in women in their mid 40s; however, it can also affect younger women. It is characterized by poor fertility outcomes, even with ART. The diagnosis is not uniformly standardized but commonly involves low AMH, low AFC, elevated basal FSH, and poor response to ovarian stimulation.[6]

DOR is sometimes confused with primary ovarian insufficiency (POI) and POR.[6] POI is characterized by the onset of amenorrhea in women under 40 years of age, accompanied by elevated FSH levels (>25 IU/L on at least two occasions) and low estradiol.[7] In contrast, POR often overlaps with DOR but specifically describes an inadequate response to ovarian stimulation in previous assisted reproduction cycles.[6]

POR and DOR are clinically significant as they are critical considerations in ART, particularly IVF. Early identification using ovarian reserve markers such as AMH and AFC enables tailored treatment strategies, optimizing outcomes.[3] It is estimated that approximately 10% of women undergoing IVF may be poor responders, although the true prevalence is likely higher due to underdiagnosis.[4]

Understanding and correctly diagnosing POR and DOR are crucial for selecting optimal ovarian stimulation protocols and for improving patient counseling and outcomes in reproductive medicine.[6]

IV. Epidemiology and Risk Factors

The reduction in both the quantity and quality of oocytes with advancing age, typically in the mid-40s, is a normal physiological process that results in DOR. However, some women experience DOR much earlier, resulting in premature infertility, which is considered pathological. Approximately 10% of women seen at infertility clinics in the USA (about 275,000 women) are diagnosed with DOR.[6] Recent data from the Society for Assisted Reproductive Technology (SART) indicate that approximately 32% of IVF cycles, around 66,000 annually, are attributed to DOR; although definitions vary and are not standardized, which limits accurate prevalence estimates and research.[6]

Globally, the prevalence of DOR appears to be rising and affecting younger women. For example, a large Korean study involving over 13,000 women in 2022 showed that the prevalence of DOR increases markedly with age: from 3.8% in women aged 20–24 years to 95% in those aged 45–49 years. The overall and age-adjusted prevalence rates were approximately 37%–38%, with small proportions showing elevated FSH alone without low AMH.[8]

POR often overlaps with DOR and is typically identified during assisted reproduction when women demonstrate a suboptimal response to controlled ovarian stimulation despite adequate treatment. The incidence of POR has also been reported to be increasing, ranging from 19% to 26% in different studies.[9]

POR and DOR are influenced by a combination of genetic predisposition, natural aging, and various lifestyle and environmental factors. Advancing age remains the most significant risk factor, but conditions such as thyroid dysfunction, polycystic ovarian syndrome (PCOS), chronic inflammation, and prolonged use of oral contraceptives can also contribute to reduced ovarian reserve. Additionally, modifiable factors such as poor diet, nutrient deficiencies (e.g., low folate or vitamin D), and exposure to environmental toxins may accelerate ovarian aging and impair fertility potential.[1], [9], [10]

V. Current Treatment Limitations and Need for Novel Interventions

Existing treatments for POR and DOR often yield suboptimal results, with limited success in restoring adequate oocyte quantity and quality. Conventional approaches such as hormonal stimulation and IVF may not sufficiently overcome the underlying mitochondrial dysfunction, oxidative stress, or age-related decline in oocyte competence.[10] CoQ10 is a naturally occurring, fat-soluble antioxidant that is widely present in cell membranes. It is a key component of the mitochondrial respiratory chain and is essential for efficient ATP production in oocytes and granulosa cells.[11] By enhancing mitochondrial function, CoQ10 supports the high-energy demands of oocyte maturation and embryo development. Additionally, it acts as a powerful antioxidant, scavenging reactive oxygen species (ROS) and protecting ovarian cells from oxidative stress and age-related decline.[12], [13]

VI. Pathophysiology of POR & DOR

The deterioration of ovarian function with age is primarily driven by increased oxidative stress and mitochondrial dysfunction, both of which are key factors in ovarian aging. Mitochondria play a crucial role in ATP production, which is essential for oocyte maturation, meiotic spindle assembly, chromosome segregation, fertilization, and early embryo development. With aging, mitochondrial efficiency declines, leading to reduced ATP production and impaired oocyte competence. Concurrently, the excessive generation of ROS damages mitochondrial DNA and other cellular components, triggering a cycle of oxidative stress that further compromise's mitochondrial function. Elevated ROS levels contribute to DNA double-strand breaks, cellular senescence, and an altered ovarian microenvironment, thereby accelerating follicular atresia and reducing both oocyte quality and ovarian reserve.[12]

Although antioxidants are widely used in gynecological practice to mitigate oxidative stress and improve reproductive outcomes, their specific benefit for women with ovarian aging remains unclear due to inconsistent evidence and a lack of standardized treatment protocols. Existing studies focus mainly on general infertility populations without addressing the unique pathophysiology of ovarian aging, which includes the critical roles of mitochondrial dysfunction and ROS-induced follicular loss.[14] Consequently, there is no consensus on which antioxidants, dosages, or patient profiles are optimal for this purpose. To address this gap, robust meta-analyses and well-designed randomized controlled trials are urgently needed to clarify the effectiveness and safety of antioxidant therapies. Evidence-based recommendations could help to prolong the reproductive lifespan, counteract the decline in ATP production, and prevent ROS-driven follicular depletion in this challenging patient group.[4]

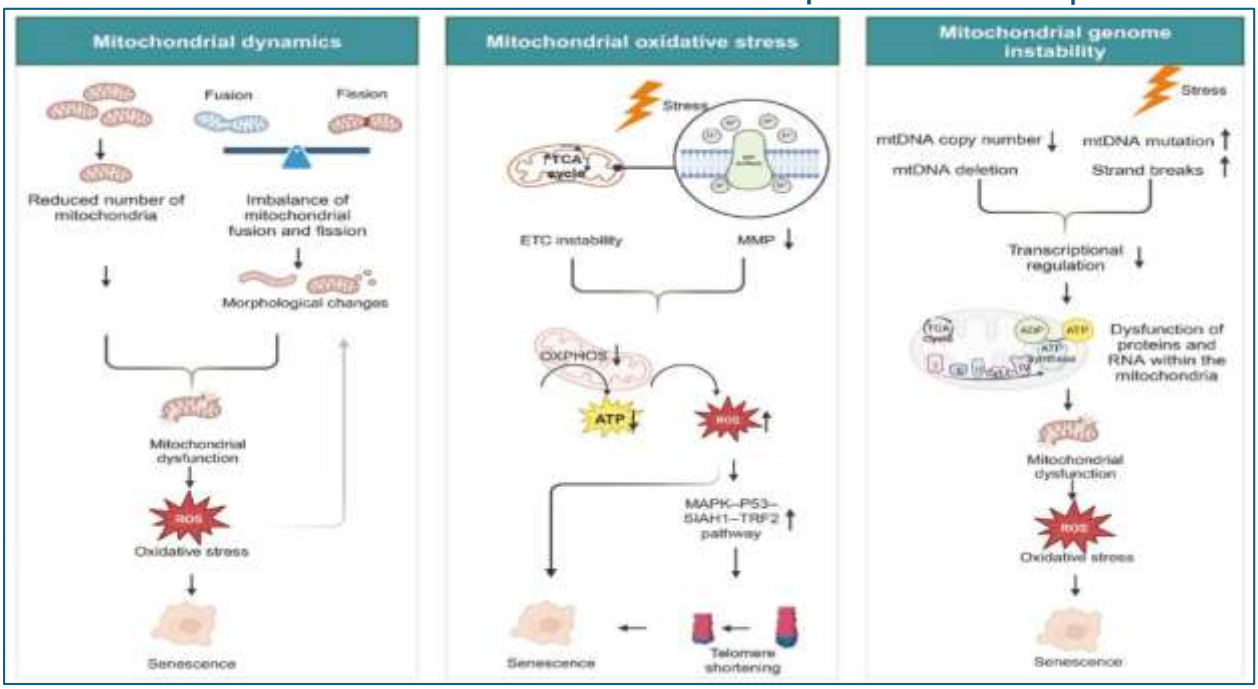

Figure 1. Mechanisms of mitochondrial dysfunction in ovarian cellular senescence. Adapted from Tang et al 2025[15]

VII. Mechanism of Action of Coenzyme Q10 in Ovarian Function

The mechanism of action of mitochondrial dysfunction in ovarian cellular senescence is shown in Figure 1. CoQ10 plays an important role in mitigating this effect. CoQ10 enhances ovarian function through the following specific mechanisms:

Role in Mitochondrial Bioenergetics and ATP Production:

CoQ10 is a vital component of the mitochondrial respiratory chain, acting as an electron and proton carrier within the inner mitochondrial membrane. It facilitates efficient electron transport, which is essential for generating the proton gradient that drives ATP synthesis. Adequate CoQ10 levels help to maintain optimal mitochondrial respiration, ensuring that oocytes and granulosa cells have sufficient ATP to support energy-demanding processes such as meiotic spindle assembly, chromosome segregation, fertilization, and early embryo development.[12], [13]

Antioxidant Properties and Reduction of Oxidative Stress:

CoQ10 functions as a potent antioxidant by directly scavenging ROS and protecting cellular and mitochondrial membranes from lipid peroxidation. This dual role helps to maintain the balance between ROS generation and elimination, preventing oxidative stress-induced damage that would otherwise accelerate oocyte aging and follicular atresia. By mitigating oxidative damage, CoQ10 interrupts the vicious cycle between mitochondrial dysfunction and ROS accumulation.[13], [15]

I*nfluence on Granulosa Cell Function, Follicular Development, and Hormone Regulation:*

CoQ10's support of mitochondrial function and redox balance directly benefits granulosa cells, which are crucial for nurturing the oocyte and producing steroid hormones. By maintaining healthy mitochondria and reducing ROS levels, CoQ10 helps preserve granulosa cell viability, supports proper follicular development, and ensures balanced steroidogenesis, all of which are key to optimal ovarian response during assisted reproduction.[13], [15]

Effects on Immune Modulation in Ovarian Health:

Emerging evidence suggests that CoQ10 may also contribute to modulating immune responses within the ovarian microenvironment. By lowering oxidative stress and supporting cellular health, it may indirectly dampen chronic inflammation and cellular senescence pathways, which are known to disrupt ovarian function and accelerate aging.[15]

VIII. Clinical Evidence on CoQ10 in POR & DOR

Impact of CoQ10 supplementation on ovarian reserve markers (AMH, FSH, AFC)

CoQ10 supplementation has demonstrated promising effects on ovarian reserve in animal studies, where it reversed ovarian toxicity, increased serum AMH levels, improved AMH-positive follicle counts, and reduced atretic follicles. In women with POR, a study found that basal day-3 FSH levels significantly decreased after 60 days of CoQ10 treatment (from 12.3 [9.4, 15.5] to 10.5 [9.2, 12.6] IU/mL, $P = 0.006$), suggesting a possible benefit for ovarian function. However, AMH and AFC values remained unchanged (AMH: 0.6 [0.4, 0.8] ng/mL before vs. after, $P = 0.91$; AFC: 5 [3, 6] vs. 5 [3, 7], $P = 0.94$), indicating that short-term supplementation may not be sufficient to impact early follicular pool dynamics. Overall, while CoQ10 may lower FSH and support mitochondrial function, longer or more intensive regimens might be necessary to observe consistent improvements in AMH and AFC in women with diminished ovarian reserve or POR.[16], [17] Another retrospective study evaluated whether adding CoQ10 to DHEA could benefit women with decreased ovarian reserve undergoing intrauterine insemination (IUI) and IVF.[18] The combination therapy significantly increased AFC compared to DHEA alone in both types of cycles. A higher AFC indicates a better pool of recruitable follicles, which is crucial for successful ovarian stimulation. In IVF cycles, patients who received CoQ10 required a lower total dose of gonadotropins, demonstrating better ovarian responsiveness. Despite these favorable effects on AFC and ovarian response, there was no significant difference in pregnancy and delivery rates between the groups. Overall, CoQ10 appears to enhance ovarian reserve markers such as AFC and potentially AMH, supporting its role in improving stimulation outcomes in women with diminished reserve.[18]

Effect on Follicular Fluid Content and Oocyte Maturity

A study evaluated the effect of oral CoQ10 supplementation on the follicular fluid environment and its potential role in supporting oocyte maturation in women undergoing IVF-ET.[19] Fifteen women aged 31–46 years received 200 mg/day of CoQ10 and were compared with unsupplemented patients. The results showed that CoQ10 supplementation significantly increased CoQ10 levels within the follicular fluid and improved its oxidative status. Notably, in follicles containing mature oocytes, the total antioxidant capacity was significantly lower in the CoQ10 group, indicating a more balanced redox state favorable for oocyte maturation. A similar improvement in oxidative status was observed in follicles with immature oocytes, suggesting a general enhancement of the follicular environment. These findings support the hypothesis that oral CoQ10 may help optimize follicular fluid oxidative metabolism and improve oocyte quality, particularly in women over 35 years of age, who are more prone to reduced oocyte competence with advancing age.[19] Similar arguments have been supported by Nie et al 2023 in in vitro studies, stating that CoQ10 may promote oocyte maturation by enhancing energy metabolism and regulating gene expression in both oocytes and granulosa cells (GCs). By improving mitochondrial function and reducing oxidative stress, CoQ10 helps create a more favorable microenvironment for follicular development, ultimately supporting better oocyte quality and maturation.[20]

Effects on In Vitro Fertilization (IVF) Outcomes

In a large multicenter, real-world evidence study conducted in India, Agarwal et al (2024) found that pre-treatment with CoQ10 at a dose of 300 mg twice daily for approximately 74 days led to significant improvements in embryo quality, oocyte yield, fertilization rate, and clinical pregnancy rate, with a median of five high-quality day-3 embryos, a median of eight oocytes retrieved, a mean fertilization rate of 73%, and a clinical pregnancy rate of 82.53%.[21] These findings are consistent with those of Xu et al (2018), who reported that CoQ10 pre-treatment in young women with poor ovarian reserve not only improved ovarian response and embryological parameters but also increased fertilization rates (67.49% vs. 45.06% in controls) and the number of high-quality embryos, although differences in clinical pregnancy and live birth rates did not reach statistical significance.[17] In this study, no improvement was observed in other ovarian reserve markers, specifically AMH and AFC, despite CoQ10 supplementation. This discrepancy compared to animal studies may be due to differences in treatment protocols and physiological variations between species.[17]

Comparison with Adjunct Therapies

Further supporting evidence comes from systematic reviews and network meta-analyses: Zhu et al (2023) demonstrated that CoQ10 was superior to other adjuvant therapies, such as DHEA and growth hormone in improving live birth and clinical pregnancy rates, with a surface under the cumulative ranking curve (SUCRA) value of 89.9% for live birth rate and a notable reduction in cycle cancellation rates.[22] Similarly, Zhang et al (2020) found that among ten different adjuvant strategies for poor ovarian responders, CoQ10 was associated with the lowest global cancellation rate and significant improvements in clinical pregnancy rates and the number of oocytes retrieved.[23] Collectively, these studies suggest that CoQ10, especially when sourced from natural origins such as *Rhodobacter sphaeroides*, acts as a mitochondrial energizer and antioxidant, making it a promising and well-tolerated pre-treatment option to optimize IVF/ICSI outcomes by enhancing both oocyte and embryo quality.[21]

Table 2: Comparison of CoQ10 with Adjunct Therapies in Female Infertility

Supplement	Mechanism of Action	Fertility Benefits	Reference
CoQ10	Mitochondrial energy production, antioxidant scavenger of ROS	Improved clinical pregnancy rate (CPR); no significant effect on live birth or miscarriage rates in ART settings	Florou et al, 2020[16]
DHEA	Androgen precursor; may improve ovarian environment	Increases AFC and improves ovarian responsiveness during IUI and IVF; no difference in clinical outcome	Gat et al, 2016[18]
Melatonin	Antioxidant; regulates circadian rhythm and supports oocyte maturation	Reduces oxidative damage in oocytes and supports follicular health	Saleh et al, 2025[14]
Vitamin C & E	Nonenzymatic antioxidants that reduce oxidative stress	Shown to reduce DNA damage; evidence for ART outcome improvement is limited	Alexandru et al, 2025[24]
Kirin Pill	Traditional Chinese medicine formula; may include herbs with antioxidant properties	Limited high-quality clinical evidence; used anecdotally to enhance reproductive function	Zheng et al, 2025[25]
NAC	Boosts glutathione, direct ROS scavenger	Lowers testosterone and improves insulin resistance in PCOS; may improve ovulation	Saleh et al, 2024[14]
Myo-inositol	Enhances oocyte quality	Improves ovulation and CPR in PCOS, especially with folic acid	Shang et al, 2024[12]
Vitamin D	Supports steroidogenesis and immune modulation	No significant benefit over CoQ10; helps regulate ovarian function	Florou et al, 2020[16]
Folic Acid	Essential for DNA synthesis and methylation	Supports embryo development; may enhance live birth rates with other antioxidants	Saleh et al, 2024[14]
Resveratrol	Polyphenol with antioxidant and anti-inflammatory properties	Experimental use in POR; may support follicular health	Shang et al, 2024[12]
Zinc & Selenium	Cofactors for antioxidant enzymes and hormone regulation	Improved oocyte quality and PCOS outcomes; may reduce miscarriage risk	Saleh et al, 2024[14]

Integration with Traditional Chinese Medicine (TCM)

A study investigated the effectiveness of combining Kirin pills with vitamin C and E and CoQ10 in treating DOR in 145 patients.[25] Participants received either vitamin C and E plus CoQ10 alone (control) or in combination with Kirin pills (study group). Although baseline characteristics were similar between groups, the combination treatment significantly improved ovarian reserve markers: it lowered serum FSH and FSH/LH ratios, increased AMH levels, and improved LH and estradiol (E2) levels compared with CoQ10 and vitamins alone. Additionally, the study group showed better menstrual recovery and a higher number of ovarian basal sinus follicles. These findings suggest that adding Kirin pills to vitamin C and E and CoQ10 may further enhance ovarian reserve function in women with DOR, although more clinical trials are needed to confirm these results.[19], [25] Another study explored the effects of CoQ10 alone and in combination with transcutaneous electrical acupoint stimulation (TEAS), a form of Traditional Chinese Medicine (TCM), in patients with POR undergoing IVF/ICSI-ET.[26] A total of 330 patients with POR were divided into three groups: CoQ10 alone, CoQ10 plus TEAS, and a control group without pretreatment. Both CoQ10 and the combination with TEAS significantly increased AFC, the number of retrieved oocytes, mature (MII) eggs, and high-quality embryos compared with the control group. The addition of TEAS, a TCM technique, further improved endometrial thickness, the number of MII eggs, and excellent-quality embryos compared to CoQ10 alone. Although CoQ10 alone enhanced ovarian reactivity and follicle development, it did not significantly increase implantation, clinical pregnancy, or live birth rates. In contrast, combining CoQ10 with TEAS significantly improved implantation rates, clinical pregnancy rates, and live birth rates compared to no pretreatment. These results suggest that CoQ10, combined with TCM-based TEAS provides a more effective adjuvant strategy than CoQ10 alone for improving ovarian response and pregnancy outcomes in patients with POR.[26]

IX. CoQ10 in ovarian stimulation protocols for assisted reproductive technologies (ARTs)

A recent meta-analysis quantitatively evaluated the impact of CoQ10 pretreatment on IVF/ICSI outcomes in women with DOR by analyzing six randomized controlled trials involving 1,529 participants.[27] The findings demonstrated that CoQ10 significantly improved key reproductive outcomes: it increased the clinical pregnancy rates, the number of high-quality embryos, the number of oocytes retrieved, and E2 levels on the day of hCG administration. Additionally, CoQ10 pretreatment reduced cycle cancellation rates, miscarriage rates, total gonadotropin (Gn) dosage, and days of Gn use. Sensitivity analyses confirmed the robustness of these results. Despite these encouraging findings, the meta-analysis highlighted the need for larger, high-quality RCTs to further validate CoQ10's benefits in patients with DOR undergoing assisted reproduction.[27] A subgroup analysis in a systematic review by Shang et al 2024, revealed that CoQ10 showed significant benefits across various fertility outcomes.[12] Taking 30 mg of CoQ10 daily for 3 months before the controlled ovarian stimulation cycle significantly improved clinical pregnancy rates. This effect was especially notable in women with diminished ovarian reserve, particularly those of younger reproductive age. The analysis suggests that CoQ10 supplementation enhances ovarian function and fertility outcomes in this patient group. Furthermore, the data indicated a dose-dependent relationship, with lower doses of CoQ10 tending to produce better effects. These findings highlight the importance of optimizing dosage and treatment duration to maximize benefits. Overall, CoQ10 appears to be a promising antioxidant treatment for improving clinical pregnancy rates in women undergoing fertility treatment.[12]

X. Safety, Dosage, and Recommendations

In reproductive medicine, optimal dosing strategies for CoQ10 vary depending on the patient profile, but evidence from Florou *et al* (2020) suggests that higher doses over a sustained period are most effective. In women with POR, CoQ10 has been administered at doses such as 600 mg once daily for 8 weeks, 600 mg twice daily for 12 weeks, and 200 mg three times daily for 8 weeks, with ART procedures typically initiated in the menstrual cycle following completion of supplementation. In women with PCOS, a lower dose of 60 mg three times daily was administered until the day of hCG administration. These dosing regimens, particularly in the POR population, were associated with improvements in ovarian response, fertilisation rates, and embryo quality, highlighting the importance of both adequate dosage and treatment duration.[16]

Regarding the safety profile of CoQ10, clinical evidence shows that CoQ10 is generally well tolerated, and no serious side effects or adverse events have been reported.[14], [16] Occasional mild gastrointestinal symptoms, such as nausea or abdominal discomfort, may occur as noted in the broader antioxidant literature, but these effects were not commonly observed in fertility-focused trials. Importantly, no participants discontinued CoQ10 due to side effects; discontinuation was limited to personal preference or adherence issues. Although Saleh et al 2024 noted the theoretical risk of reductive stress with excessive antioxidant use, this risk has not been documented in CoQ10 fertility trials.[14]

Regarding clinical recommendations, CoQ10 is emerging as a valuable adjunct in the management of POR and DOR. According to Florou et al (2020), CoQ10 supplementation was associated with a significant improvement in clinical pregnancy rates (28.8% vs. 14.1% with placebo; OR 2.44, $P = 0.006$), as well as improvements in several reproductive parameters, including the number of mature oocytes retrieved, fertilization rates, day-3 high-quality embryos, and embryo cryopreservation rates.[16] Although CoQ10 did not significantly improve live birth or miscarriage rates, its beneficial impact on early ART outcomes supports its use as a pre-treatment strategy. Saleh *et al* (2024) also stated that CoQ10 is widely prescribed by clinicians for female infertility, despite the absence of standardized dosing protocols. Therefore, a daily dose of 600–1200 mg for 8–12 weeks prior to ART appears to be a clinically appropriate approach, particularly for women with POR or DOR.[14]

XI. Conclusion

Coenzyme Q10 has emerged as a promising adjunctive therapy for women with poor ovarian response and diminished ovarian reserve. These conditions that pose significant challenges in ART. CoQ10 supplementation is supported by its dual role as a mitochondrial bioenergetic enhancer and potent antioxidant, which supports oocyte maturation, reduces oxidative damage, and improves granulosa cell function. Clinical studies and meta-analyses consistently show improved reproductive outcomes, including oocyte yield, embryo quality, fertilization rates, and clinical pregnancy rates. These benefits are especially noted when CoQ10 is administered at higher doses (600–1200 mg/day) for a duration of 8 –12 weeks prior to ART.

Despite variability in study protocols and the lack of standardisation in treatment guidelines, CoQ10 is well tolerated, with minimal side effects, and has shown superior efficacy compared to other adjunct therapies such as DHEA and growth hormone. Although more high-quality, large-scale randomised controlled trials are needed to confirm its impact on live birth and miscarriage rates, current evidence supports its use as a safe, cost-effective, and biologically plausible intervention to optimise ART outcomes in women with compromised ovarian reserve. Therefore, CoQ10 supplementation represents a valuable and evidence-based strategy to enhance reproductive success in this difficult-to-treat patient population.

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XIII. References

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