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Unlocking Fertility Potential in Women 40+; Can Q10 Improve Egg Quality?

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Abstract

Could CoQ10 hold unexpected promise for ovarian rejuvenation? Could it be the "bridge" between the "bench" and the "bed" for millions navigating the intersection of biology and time." Reproductive aging in women over 40 is marked by declining oocyte quality driven by mitochondrial dysfunction, oxidative stress, and chromosomal instability. At the heart of this decline lies aneuploidy—a leading cause of age-related infertility and miscarriage—rooted in failures of meiotic cohesion, spindle assembly, and DNA repair. Emerging preclinical research now spotlights Coenzyme Q10 (CoQ10), a mitochondrial electron transporter and antioxidant, as a potential mitigator of these age-associated defects. It offers the potential to restore ATP fuel to spindles, shield cohesion from oxidative sabotage, and perhaps even slow the telomeric clock. Could this molecule hold a key to rewriting the narrative of reproductive aging? The data, though nascent, ignite a question; could CoQ10 bridge the gap between the "bench" and the "bed" for women over 40?

Keywords: CoQ10; Coenzyme Q10; Advanced maternal age

CoQ10 has attracted global interest due to its crucial role in improving oocyte quality and counteracting oocyte aging (1-4). Xu Y, et al showed that pretreatment with CoQ10 significantly increased the number of high-quality embryos in women with poor ovarian response (POR) during the IVF-ICSI cycle (3).Turi et al. reported that human follicular fluid CoQ10 correlated with embryo quality (5).

Several animal studies indicated that CoQ10 opposed ovarian aging and enhanced ovarian reserve through different mechanisms (6–9). However, biomarkers and biological pathways through which CoQ10 counteracts oocyte aging are not understood. A network pharmacology-based approach was successfully used to reveal all candidate targets, functions, and potential therapeutic mechanisms of melatonin in women with diminished ovarian reserve (10). System network pharmacology combined with molecular docking revealed the multiple mechanisms involved in the anti-aging effect of CoQ10 on oocytes. It provided evidence that CoQ10 may act on these hub targets to fight against oocytes aging (11). Diminished expression of the enzymes responsible for CoQ10 production, Pdss2 and Coq6, was observed in oocytes of older females in both mouse and human.

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Editorial

Oocyte specific disruption of Pdss2 induced reduction in ATP production and increased meiotic spindle abnormalities, resulting in infertility. Ovarian reserve was also diminished, leading to premature ovarian failure which could be prevented by maternal dietary administration of CoQ10 (12).

Previously published microarray studies comparing old and young oocytes revealed altered expression of genes responsible for mitochondrial function, oxidative stress responses, chromosome alignment, and ubiquitination Hamatani et al., 2004 (13); Pan et al., 2008 (14). Ultrastructural examination of cellular organelles in aging oocytes confirmed the defects in mitochondrial architecture Kujjo et al., 2013 (15). Similar findings are reported in bovine oocyte models, where oxidative stress disrupts spindle proteins (16) Tatone et al., 2010). ROS destabilizes tubulin polymerization. CoQ10's antioxidant effects may protect microtubule-organizing centers (MTOCs) in mouse oocytes which resemble in function the role of centrosomes in human oocytes and keep kinetochore-microtubule attachments (16) Ben-Meir et al., 2015).

Telomere attrition is linked to aging and genomic instability, though its direct role in oocyte aneuploidy is less clear. Telomeres are highly susceptible to oxidative damage. CoQ10 may slow telomere shortening by reducing ROS-induced DNA breaks (17) Blackburn et al., 2015). In mice, CoQ10 delays ovarian aging and improves mitochondrial health, though telomere length was not directly measured (18) (Rodríguez-Nuevo et al., 2022). Proper crossover formation ensures homologous chromosomes segregate correctly. ROS can disrupt meiotic recombination, leading to non-disjunction. CoQ10's antioxidant properties may protect proteins (e.g., BRCA1, MLH1) and DNA structures involved in crossover regulation, though direct evidence is lacking (19) (Hunt & Hassold, 2008).

The hallmark of this editorial was to shed a light on CoQ10's preclinical portfolio, from restoring mitochondrial bioenergetics in aged mouse oocytes (12)(Ben-Meir et al., 2015) to shielding cohesion complexes from oxidative decay (16) (Tatone et al., 2010), offers a compelling blueprint.Yet, the leap from bench to bedside demands rigorous human trials to confirm whether these cellular repairs translate to fewer chromosomal errors, higher live birth rates and healthier pregnancies. While CoQ10 is not a panacea, its story underscores that aging ovaries are not inert, but dynamic systems "potentially" responsive to metabolic and antioxidant intervention.

The way forward is a path between skepticism and empathy, recognizing that every molecular insight could provide a hope in a situation which is already disparate. Evidence based, patient oriented and personalized medicine are the borders guiding our practice. For women over 40 the future of fertility may lie not in defying age, but in redefining its limits."

Key Takeaways

There is indirect biological plausibility that CoQ10 could mitigate aging effect on oocyte aneuploidy. CoQ10's benefits for chromosomal stability are indirect, mediated via mitochondrial support and antioxidant effects. Direct evidence in human oocytes/embryos is lacking, particularly for processes like cohesion stabilization and crossing over. A call for action: Clinical trials measuring aneuploidy rates (e.g., PGT-A) in CoQ10-supplemented women over 40 are critical next steps.

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