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EDITORIAL

Coenzyme Q10 at the Crossroads of Migraine and Fertility Care

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Migraine and female subfertility are typically managed as separate clinical entities, yet both conditions share a compelling biological substrate: mitochondrial dysfunction and elevated oxidative stress. Coenzyme Q10 (CoQ10), a lipid-soluble molecule integral to mitochondrial ATP synthesis and a potent scavenger of reactive oxygen species, sits at this mechanistic intersection [1, 2].

The interplay between migraine and the female reproductive lifespan is well recognized, with hormonal fluctuations influencing migraine susceptibility, frequency, and severity across puberty, pregnancy, and the perimenopausal transition. Hormonal investigations further show that women with migraine demonstrate dysregulated sex hormone profiles compared with controls, confirming that reproductive biology and migraine pathophysiology are more intertwined than conventional specialty boundaries acknowledge [3].

The mitochondrial hypothesis of migraine is increasingly supported. Brain energy deficits, neuronal hyperexcitability, and elevated markers of oxidative stress, including malondialdehyde, matrix metalloproteinase-9, and nitric oxide, are consistently documented in women with migraine [4]. Multi-omics analyses further confirm that mitochondrial dysfunction, inflammatory responses, and oxidative stress are central to migraine pathophysiology [5]. These metabolic disturbances promote calcitonin gene-related peptide (CGRP) release and neuroinflammatory signaling, the downstream mediators of migraine pain, placing CoQ10 in a mechanistically coherent preventive position [2].

Clinical evidence increasingly supports this rationale. A ded-

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icated clinical trial demonstrated the efficacy of CoQ10 as a prophylactic agent in episodic migraine [6]. A 2025 systematic review and meta-analysis of double-blinded randomized controlled trials (RCTs) further suggested that CoQ10, alone or combined with nutraceuticals, significantly reduces monthly migraine frequency, duration, and severity, with optimal benefit requiring at least three months of supplementation [7].

A complementary dose-response meta-analysis documented significant reductions across all three clinical parameters [8]. Biochemical analyses further show that CoQ10 supplementation significantly lowers serum CGRP and TNF-α levels, providing mechanistic corroboration for its clinical effects [4]. A head-to-head RCT comparing CoQ10 with amitriptyline reported comparable long-term clinical outcomes with fewer adverse effects, although the trial was conducted in children. This safety profile may still be clinically relevant when considering CoQ10 for reproductive-age women, for whom many standard migraine preventive medications carry hormonal or teratogenic safety concerns [9].

In reproductive medicine, human oocytes harbor exceptional mitochondrial density to sustain the energy demands of meiosis, fertilization, and early embryonic development. However, this high metabolic activity may predispose them to oxidative-stress-induced damage, which can adversely affect fertility [10]. Declining endogenous CoQ10 with aging coincides with deterioration in oocyte quality, increased chromosomal errors, and age-related subfertility [11]. A 2024 systematic review and meta-analysis of six RCTs encompassing 1,529 women with diminished ovarian reserve suggested that CoQ10 pretreatment significantly elevated clinical pregnancy rates, oocyte yield, and optimal embryo counts, while reducing cycle cancellation and miscarriage rates [12]. A broader meta-analysis of antioxidants in women with ovarian aging ranked CoQ10 as superior to melatonin, myo-inositol, and vitamins in improving reproductive outcomes and, based on subgroup analysis, suggested that 30 mg/day for three months before controlled ovarian stimulation may be the optimal CoQ10 regimen for improving clinical pregnancy rates [13].

Network meta-analysis further identifies CoQ10 as the leading adjuvant for improving live birth and clinical pregnancy rates in poor ovarian responders undergoing IVF [14]. A 2025 meta-analysis of 16 studies (2,773 participants) additionally suggested that CoQ10 supplementation sustained for more than two months significantly improves FSH, AMH, antral follicle counts, and pregnancy rates in women with diminished ovarian reserve [15].

CoQ10 has a consistently favorable safety and tolerability profile across both neurological and reproductive populations [2, 7], unlike conventional migraine preventives such as valproate and topiramate, which carry significant reproductive safety concerns. Nevertheless, current evidence is limited by heterogeneous dosing, variable formulations (ubiquinol vs. ubiquinone), and the near-complete absence of trials enrolling women with coexisting migraine and subfertility as a unified target population. This gap reflects a systemic problem in women's health: the shared mitochondrial substrate underpinning these two conditions goes largely unrecognized when they are evaluated in isolation within separate specialties.

In conclusion, CoQ10 occupies a clinically meaningful crossroads between migraine prevention and fertility care. Its dual mechanism, enhancing mitochondrial bioenergetics and limiting oxidative damage, offers a biologically coherent and well-tolerated strategy applicable to two burdensome, potentially overlapping conditions in women of reproductive age. We call for integrated clinical trials targeting this population and for closer collaboration between neurologists, gynecologists, and reproductive specialists, as recognizing this shared biology may help shift the paradigm toward a more holistic, patient-centered model of women's health care.

ETHICAL DECLARATIONS

- **Ethics Approval and Consent to Participate**

  Not applicable.

- **Consent for Publication**

  Not applicable.

- **Availability of Data and Material**

  Not applicable.

Competing Interests

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Authors' Contributions

Both authors contributed to the conception, drafting, critical revision, and final approval of the editorial.

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