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Estrogen–Mitochondrial Interactions in Cellular Resilience: Biological Implications for Female Military Personnel

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ABSTRAK

Operasi militer menghadapkan personel pada stres fisiologis dan psikologis yang tinggi, yang dapat meningkatkan beban allostatik serta mengganggu fungsi mitokondria. Estrogen, khususnya 17β-estradiol, diketahui berperan penting dalam menjaga homeostasis sel melalui regulasi fungsi mitokondria. Tinjauan naratif sistematis ini bertujuan untuk mengkaji interaksi antara estrogen dan mitokondria sebagai dasar biologis ketahanan seluler perempuan dalam menghadapi stres operasional. Penelusuran literatur dilakukan pada basis data PubMed, Scopus, dan ScienceDirect untuk publikasi tahun 2004–2024. Sebanyak 44 artikel yang memenuhi kriteria inklusi dianalisis secara kualitatif. Hasil kajian menunjukkan bahwa estrogen memodulasi fungsi mitokondria melalui reseptor ERα, ERβ, dan GPER. Estrogen meningkatkan biogenesis mitokondria melalui jalur PGC-1α/NRF-1/TFAM, mendukung dinamika mitokondria melalui protein MFN2 dan OPA1, memperkuat sistem antioksidan melalui peningkatan aktivitas MnSOD dan katalase, serta menekan proses apoptosis. Selain itu, estrogen berperan dalam regulasi mitofagi dan menjaga stabilitas DNA mitokondria (mtDNA). Kesimpulannya, interaksi estrogen–mitokondria merupakan faktor penting dalam mendukung ketahanan metabolik dan neurokognitif perempuan. Temuan ini memberikan dasar ilmiah bagi pengembangan strategi kesehatan yang lebih spesifik gender untuk mengoptimalkan performa perempuan dalam lingkungan militer yang menuntut.

Kata kunci: Estrogen; Mitokondria; Ketahanan Seluler; Bioenergetika; Stres Oksidatif; Personel Militer Wanita

ABSTRACT

Military operations expose personnel to intense physiological and psychological stressors that can increase allostatic load and impair mitochondrial function. Estrogen, particularly 17β-estradiol, plays a crucial role in maintaining cellular homeostasis through the regulation of mitochondrial activity. This systematic narrative review aimed to examine estrogen–mitochondrial interactions as a biological basis for female cellular resilience under operational stress. A literature search was conducted using PubMed, Scopus, and ScienceDirect databases for publications from 2004 to 2024. A total of 44 eligible articles were qualitatively analysed. The findings indicate that estrogen modulates mitochondrial function through ERα, ERβ, and GPER signalling pathways. Estrogen promotes mitochondrial biogenesis via the PGC-1α/NRF-1/TFAM pathway, supports mitochondrial dynamics through MFN2 and OPA1 proteins, enhances antioxidant defence by increasing MnSOD and catalase activity, and suppresses apoptotic processes. In addition, recent evidence suggests that estrogen contributes to the regulation of mitophagy and the maintenance of mitochondrial DNA (mtDNA) stability. In conclusion, estrogen–mitochondrial interactions play a critical role in supporting metabolic and neurocognitive resilience in women. These findings provide a scientific foundation for the development of gender-specific health strategies aimed at optimising female performance and adaptability in demanding military environments.

Keywords: Estrogen; Mitochondria; Cellular Resilience; Bioenergetics; Oxidative Stress; Female Military Personnel

INTRODUCTION

Modern military operations subject personnel to extreme stress conditions involving intense physical exertion, sleep deprivation, exposure to hypoxia, chronic psychological strain, and environmental uncertainty. These conditions increase the production of reactive oxygen species (ROS), disrupt oxidative phosphorylation, and increase the risk of mitochondrial dysfunction as the cell's bioenergetic centre (Picard et al., 2018). Mitochondria not only play a role in ATP production, but also regulate redox balance, calcium homeostasis, inflammatory responses, and apoptosis.

Sexual dimorphism in mitochondrial function is increasingly recognised as an important biological factor. Women generally exhibit higher mitochondrial respiratory efficiency and lower basal ROS production compared to men, partly mediated by estrogen (Junker et al., 2022). Estrogen is known to modulate energy metabolism, mitochondrial membrane stability, and antioxidant pathways through estrogen receptor interactions at both the nuclear and mitochondrial levels.

Although estrogen–mitochondrial mechanisms have been extensively discussed in cardiovascular and neurodegenerative contexts, there is no comprehensive synthesis explicitly linking these molecular pathways to female cellular resilience in a military operational environment. This translational gap is the primary focus of this review.

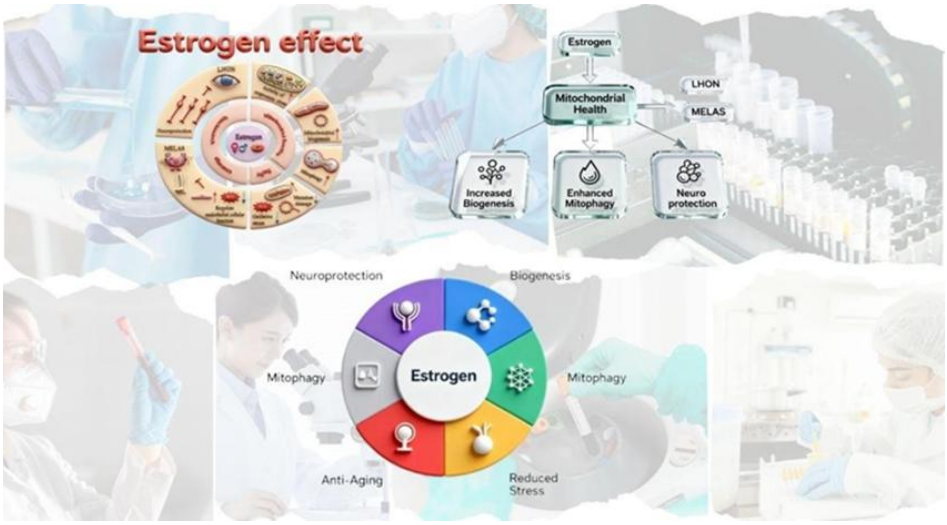

Figure 1. The effects of estrogen on mitochondria.

METHODS

This study employs a systematic narrative review approach with a structured literature search strategy.

Search Strategy

Searches were conducted on PubMed, Scopus, and ScienceDirect for publications from January 2004 to 2024. Emphasis was placed on literature from 2020 to 2024 to ensure up-to-date relevance. The keywords used were: ("estrogen" OR "17β-estradiol" OR "ERα" OR "ERβ") AND ("mitochondria" OR "mitochondrial biogenesis" OR "oxidative stress" OR "mitophagy" OR "apoptosis") AND ("sex differences" OR "female physiology" OR "stress adaptation").

Inclusion Criteria

1. Original research articles and peer-reviewed reviews.
2. Studies discussing the molecular or cellular mechanisms of estrogen–mitochondrial interactions.
3. Human, animal, or in vitro studies with translational relevance.
4. Full text available in English.

Exclusion Criteria

1. Articles not focusing on mitochondria.
2. Non-peer-reviewed literature.
3. Conference abstracts without a full paper.

Study Selection

A total of 67 articles were identified; following screening of titles, abstracts, and full texts, 44 articles were included in the final synthesis. Study selection was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines to ensure transparency and reproducibility of the literature selection process (Page et al., 2021).

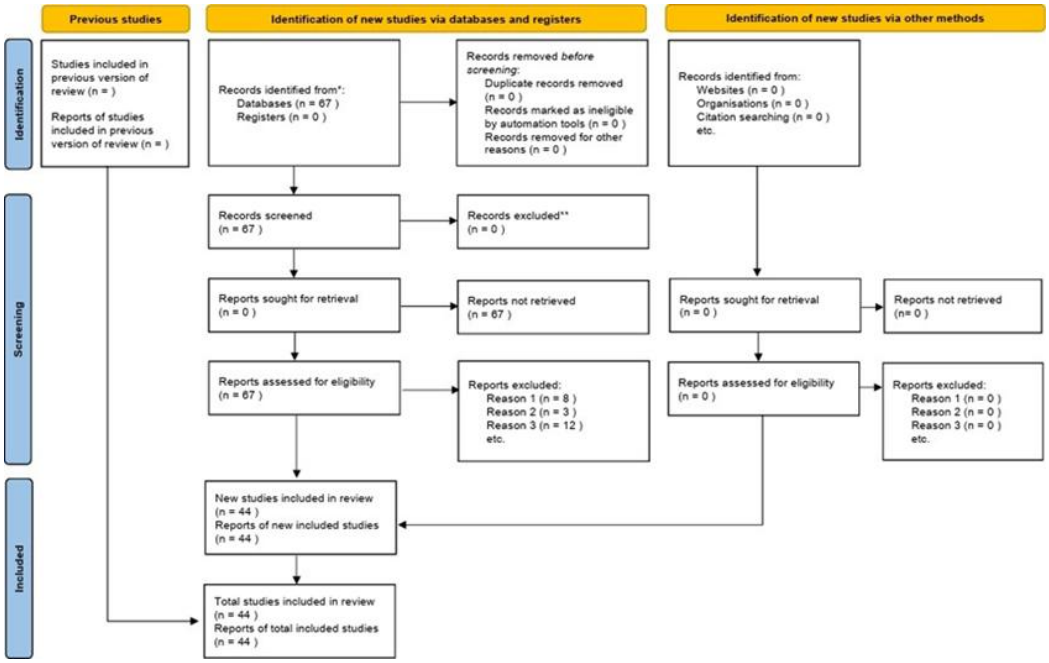

Figure 2. Flowchart of study selection based on the PRISMA 2020 guidelines.

RESULTS AND DISCUSSION

Estrogen Receptors and Mitochondrial Localization

Estrogen acts via ERα, ERβ, and GPER, with strong evidence indicating that ERβ is localised to the mitochondria and plays a role in the direct regulation of mtDNA expression (Chen et al., 2004; Klinge, 2020). This nuclear–mitochondrial communication is increasingly regarded as a central node through which estrogen coordinates organelle function and cellular adaptation (Guajardo-Correa et al., 2022). Experimental studies show that ERβ activation increases transcription of electron transport chain genes, such as COX I and ND1, thereby contributing to mitochondrial respiratory efficiency. However, recent studies emphasise that the distribution of these receptors is tissue-specific and influenced by physiological conditions (Arnold et al., 2017; Junker et al., 2022). This explains the variability in results across studies and constitutes a limitation in generalising findings to the human population, particularly in the context of extreme stress such as military service.

Regulation of Bioenergetics and Mitochondrial Biogenesis

Several recent studies have shown that estrogen consistently activates the PGC-1α–NRF-1–TFAM pathway as the primary regulator of mitochondrial biogenesis (Klinge, 2020; Krishnan et al., 2018). This effect not only increases mitochondrial numbers but also enhances the efficiency of oxidative phosphorylation and metabolic flexibility. Consistent with this, estrogen receptor α signalling in skeletal muscle has been shown to be critical for maintaining mitochondrial function and metabolic homeostasis in females (Ribas et al., 2016), and 17β-estradiol can directly improve bioenergetic function by lowering mitochondrial membrane microviscosity (Torres et al., 2018; Ventura-Clapier et al., 2019).

Within the context of military operations, these physiological adaptations may contribute to improved aerobic capacity, greater tolerance to hypoxic conditions, and more efficient recovery of cellular energy following stress exposure. Despite these promising findings, the translational relevance to human populations remains limited, as most of the current evidence derives from in vitro experiments and animal studies (Arnold et al., 2017; Wilson, 2017).

Modulation of Oxidative Stress and mtDNA Stability

Estrogen has been shown to upregulate the expression of key antioxidant enzymes, including manganese superoxide dismutase (MnSOD), catalase, and glutathione peroxidase (GPx), thereby strengthening the cellular antioxidant defence system (de Bari et al., 2020; Tower et al., 2020; Viña et al., 2013). In addition to enhancing antioxidant capacity, emerging evidence suggests that estrogen contributes to the reduction of electron leakage within the mitochondrial electron transport chain, supports the maintenance of

mitochondrial membrane potential, and mitigates mitochondrial DNA damage induced by reactive oxygen species.

Under conditions of chronic stress, mitochondrial dysfunction plays a central role in increasing allostatic load (Picard et al., 2018). In this context, estrogen appears to function as a metabolic stabiliser, particularly in environments characterised by sleep deprivation, hypoxic exposure, and sustained psychological stress, thereby supporting cellular resilience under extreme conditions.

Mitochondrial Dynamics and Mitophagy

Estrogen plays a critical role in regulating mitochondrial dynamics by maintaining the balance between fusion and fission processes through the modulation of key proteins such as mitofusin-2 (MFN2) and optic atrophy 1 (OPA1) (Beikoghli Kalkhoran & Kararigas, 2022). In addition, emerging evidence indicates that estrogen is involved in the regulation of the PINK1/Parkin-mediated mitophagy pathway, which is essential for the selective removal of damaged or dysfunctional mitochondria (Jiang, 2025).

However, the regulatory effects of estrogen on mitochondrial dynamics are not uniform and appear to be influenced by multiple physiological factors. These include interactions with other hormones, particularly progesterone and cortisol, as well as the nature and duration of stress exposure, such as acute versus chronic conditions. Collectively, these factors suggest that mitochondrial regulation by estrogen is highly dynamic and context-dependent.

Regulation of Apoptosis and Neuroprotective Resilience

Estrogen has been shown to exert anti-apoptotic effects by modulating the intrinsic apoptotic pathway, primarily through the upregulation of anti-apoptotic proteins such as Bcl-2 and the suppression of cytochrome c release from the mitochondria (Lewis-Wambi & Jordan, 2009; Simpkins et al., 2008, 2010). These mechanisms contribute to the preservation of mitochondrial integrity and cellular survival under stress conditions and extend to ischemia–reperfusion injury, in which estrogen mediates sex-dependent cardioprotection (Wang et al., 2019).

Furthermore, the concept of "healthy cell bias" suggests that the protective effects of estrogen are more pronounced in cells that have not yet experienced severe or irreversible damage. This phenomenon is particularly relevant in conditions associated with high physiological and psychological stress, such as blast-related trauma and chronic psychological stress, where mitochondrial stability and neuronal survival are critical for maintaining functional resilience. This interpretation is supported by evidence that the uncoupling action of 17β-estradiol underlies the greater resilience of female-derived mitochondria to damage following experimental traumatic brain injury (Kalimon et al., 2024), a finding with direct relevance to blast-related neurotrauma in operational settings.

Translational Implications for Female Military Personnel

Fluctuations in estrogen throughout the menstrual cycle have the potential to influence antioxidant capacity, metabolic efficiency, and neurocognitive resilience. This review suggests that estrogen is not merely a reproductive hormone, but also a key regulator of mitochondrial resilience. However, field evidence in military populations remains limited and heterogeneous, so there is as yet no strong scientific basis for regulating operational deployments based on the phase of the menstrual cycle. Longitudinal research based on mitochondrial biomarkers is required to support evidence-based policy.

Beyond the question of scientific sufficiency, translating these findings into operational policy raises practical and ethical considerations that must be resolved before any implementation is contemplated. From a practical standpoint, the menstrual cycle is highly variable both between and within individuals, and a substantial proportion of female service members use hormonal contraceptives that suppress or flatten the natural 17β-estradiol fluctuation on which any phase-based scheduling would depend. Because inter-individual variability may exceed the average phase-level differences in mitochondrial efficiency, duty rosters constructed around cycle phase risk being operationally unreliable, logistically demanding, and incompatible with the unpredictable tempo of field operations.

Ethically, using reproductive and hormonal information to inform deployment decisions intersects directly with personnel privacy, autonomy, and the principle of non-discrimination. If inadequately governed, phase-based assignment could stigmatise female personnel, reinforce long-standing assumptions about women's suitability for demanding or combat roles, and be misused to justify exclusion rather than targeted support; mandatory hormonal

monitoring would additionally raise concerns regarding informed consent and the secure handling of sensitive medical data. The responsible interpretation of the present evidence is therefore that estrogen–mitochondrial physiology should inform individualised and voluntary health optimisation—such as recovery, nutrition, and countermeasure strategies—rather than serve as a deterministic criterion for restricting opportunity. Any prospective policy should be evidence-led, protective of reproductive privacy, and explicitly designed to widen rather than narrow the operational roles available to women.

Table 1
Summary of estrogen-regulated mitochondrial mechanisms and their operational relevance.

Mitochondrial Pathway / Function	Molecular Targets	Estrogenic Effects	Cellular Implications	Military Operational Relevance
Biogenesis	PGC-1α, NRF-1, TFAM	Increased mitochondrial biogenesis and oxidative phosphorylation efficiency	Greater bioenergetic capacity and metabolic flexibility	Improved aerobic capacity and tolerance to hypoxic conditions
Antioxidants / Redox	MnSOD, catalase, GPx	Increased enzyme activity and reduced ROS production	Maintains redox homeostasis and prevents membrane damage	Reduces oxidative fatigue from sleep deprivation and high stress
Dynamics (Fusion / Fission)	MFN2, OPA1, PINK1/Parkin	Regulation of mitochondrial fusion, fission, and mitophagy	Maintains mitochondrial network quality	Rapid adaptation to extreme environmental stress (altitude, blast)
Intrinsic Apoptosis	Bcl-2 family, cytochrome c	Increased Bcl-2; decreased release of cytochrome c	Suppresses programmed cell death	Enhances neurocognitive resilience under chronic psychological stress
mtDNA Stability	Pol γ, mt-ERα receptor	Stabilisation and repair of mtDNA	Reduces stress-induced mutations	Prevents long-term performance decline in female personnel

Note. Operational relevance: in extreme field operations, hypoxia, chronic stress, and sleep deprivation consistently increase ROS production and mitochondrial dysfunction; whereas testosterone confers sex-dimorphic protection, estrogen in women is associated with higher respiratory efficiency. Implications: a potential, evidence-pending consideration of menstrual-cycle-aware recovery scheduling during the follicular phase (when estrogen levels are high) to reduce the risk of neurocognitive fatigue, subject to military field validation. Strategy: selective estrogen receptor modulator (SERM) supplementation or monitoring of mtDNA biomarkers to enhance resilience.

Contributions and Novelty of the Review

This review presents a systematic integration of the estrogen–mitochondrial molecular pathway and its implications in the context of military operational stress, an area that has not previously been comprehensively studied. By linking the basic literature on bioenergetics with issues in women's military health, this article offers a new conceptual framework regarding gender-specific "mitochondrial resilience."

Limitations and Future Directions

Variability in experimental methodologies further complicates interpretation. Differences in the assessment of reactive oxygen species (e.g., fluorometric assays versus Western blot techniques) and mitochondrial DNA (e.g., sequencing versus quantitative PCR) limit cross-study comparability and hinder the development of unified conclusions. In parallel, the current literature rarely integrates physiological hormonal dynamics, such as menstrual cycle phases or menopausal status, with operational stress exposure. The interplay between estrogen and other endocrine mediators, including cortisol and progesterone, also remains insufficiently characterised. In addition, the limited number of field-based investigations and the possibility of publication bias toward positive outcomes constrain the strength of current conclusions.

To address these limitations, future research should prioritise:

1. Prospective longitudinal studies in female military personnel with serial measurements of mitochondrial biomarkers (mtDNA copy number, ROS levels, and OXPHOS efficiency via high-resolution respirometry);
2. Field studies during simulated operations (e.g., high-altitude training, 72-hour sleep deprivation) for translational validation;
3. Analysis of the menstrual cycle on metabolic and neurocognitive performance using

wearable biosensors and saliva hormone profiling;
4. Clinical trials of selective estrogen receptor modulators (SERMs) or phytoestrogens to enhance resilience;
5. Multi-omics approaches (mtDNA genomics, metabolomics, mitochondrial proteomics) combined with machine learning for the identification of predictive biomarkers.

Collectively, this agenda will help bridge the gap from the laboratory to the battlefield, supporting evidence-based and sex-specific military health policies.

CONCLUSION

Estrogen–mitochondrial interactions constitute a fundamental biological mechanism in maintaining women's metabolic and neurocognitive resilience under extreme stress. The regulation of biogenesis, mitochondrial dynamics, the antioxidant system, mitophagy, and apoptosis forms the basis of cellular adaptation to physical and psychological stress.

Although the mechanistic evidence is strong, translational research in military populations remains limited. Longitudinal studies based on mitochondrial biomarkers, as well as intervention trials based on hormonal phases, are needed to integrate these findings into gender-specific military health strategies, provided that such applications remain voluntary, privacy-protective, and non-discriminatory.

AUTHOR CONTRIBUTIONS

Conceptualisation and study design: C.M. and S.T.; literature search, screening, and data extraction: C.M., L.D.P., and N.T.S.; qualitative synthesis and analysis of molecular mechanisms: C.M. and S.T.; writing—original draft preparation: C.M.; writing—review and editing: S.T., L.D.P., and N.T.S. All authors have read and agreed to the final version of the manuscript.

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ETHICAL APPROVAL

Not applicable. This study is a systematic narrative review of previously published literature and did not involve direct experimentation with human participants or animals. All analysed data were derived from experimental, clinical, and review articles available in public databases. Therefore, formal ethical approval was not required for this study.

CONFLICTS OF INTEREST

The authors declare that they have no known competing financial interests, institutional affiliations, or personal relationships that could have appeared to influence the work reported in this study. No funding bodies or external parties had any role in the study design, data collection, analysis, interpretation of data, manuscript preparation, or the decision to submit the article for publication.

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