JOURNAL OF MULTIDISCIPLINARY SCIENCES AND INNOVATIONS
VOLUME 05, ISSUE 01
MOPNTHLY JOURNALS
ISSN NUMBER: 2751-4390
IMPACT FACTOR: 9,08

THE ROLE OF BEHAVIORAL AND METABOLIC FACTORS IN THE DEVELOPMENT OF ADAPTIVE DISORDERS IN WOMEN DURING MENOPAUSE

Mukhitdinova X.S.
Asia International University

Abstract: Menopause is a biologically determined stage of female aging accompanied by profound endocrine, metabolic, and neuroregulatory transformations. Although it represents a physiological transition, menopause is frequently associated with the development of adaptive disorders that compromise metabolic stability, psychoemotional balance, and cardiovascular health. Considerable interindividual variability in clinical manifestations suggests a decisive influence of modifiable behavioral and metabolic determinants.

This narrative review analyzes current scientific evidence regarding the mechanisms through which metabolic alterations and lifestyle-related factors contribute to maladaptive responses during the menopausal transition and early postmenopause. Particular emphasis is placed on estrogen deficiency–mediated metabolic remodeling, visceral adipose tissue accumulation, insulin resistance, circadian rhythm disruption, chronic low-grade inflammation, and behavioral patterns affecting energy homeostasis.

Available data indicate that menopause constitutes a critical period of increased metabolic sensitivity, during which unfavorable lifestyle factors may accelerate biological aging and disease progression. Conversely, timely behavioral modification and metabolic correction appear capable of significantly improving adaptive capacity and long-term health outcomes.

Keywords: menopause, adaptive disorders, metabolic remodeling, lifestyle factors, insulin resistance, visceral adiposity, circadian dysregulation

Introduction

Menopause is defined as the irreversible cessation of ovarian activity resulting from depletion of the follicular reserve and permanent termination of menstrual function. This transition marks a pivotal stage in female aging and is accompanied by extensive hormonal, metabolic, and neuroendocrine reorganization.

While menopause is universally experienced by women, the intensity and spectrum of associated clinical manifestations vary widely. Vasomotor instability, metabolic disturbances, sleep disorders, mood alterations, and increased cardiovascular risk are among the most frequently reported complications. Such heterogeneity cannot be fully explained by estrogen deficiency alone, indicating the involvement of additional pathogenic mechanisms.

In recent decades, menopause has increasingly been conceptualized as a period of reduced adaptive reserve, during which the ability of regulatory systems to maintain homeostasis becomes limited. Under these conditions, metabolic and behavioral factors may exert a disproportionate influence on health trajectories, predisposing women to metabolic syndrome, type 2 diabetes mellitus, and cardiovascular disease.

Understanding the interplay between endocrine changes, metabolic remodeling, and lifestyle-related determinants is therefore essential for the development of preventive strategies and personalized interventions in menopausal medicine.

Endocrine Background of Menopausal Adaptation

Estrogens exert pleiotropic effects on carbohydrate and lipid metabolism, vascular tone, mitochondrial function, and central nervous system regulation. Their decline during menopause leads to disruption of multiple metabolic pathways.

At the central level, estrogen deficiency alters hypothalamic regulation of appetite, thermoregulation, and circadian rhythms. Peripherally, reduced estrogen signaling impairs insulin sensitivity, enhances adipocyte differentiation, and weakens antioxidant defense mechanisms.

These endocrine changes create a metabolic environment characterized by increased energy storage, reduced metabolic flexibility, and heightened susceptibility to external stressors, thereby lowering overall adaptive capacity.

Metabolic Remodeling and Visceral Adiposity

One of the most prominent metabolic consequences of menopause is the redistribution of adipose tissue toward the visceral compartment. This shift occurs independently of total body weight gain and reflects altered hormonal regulation of adipocyte metabolism.

Visceral adipose tissue is metabolically active and functions as an endocrine organ. It secretes proinflammatory cytokines, adipokines, and free fatty acids that promote systemic inflammation, endothelial dysfunction, and insulin resistance.

The accumulation of visceral fat is strongly associated with cardiometabolic risk and represents a central mediator linking menopause with chronic non-communicable diseases.

Insulin Resistance as a Core Pathophysiological Mechanism

Insulin resistance plays a pivotal role in menopausal metabolic maladaptation. Reduced responsiveness of peripheral tissues to insulin leads to compensatory hyperinsulinemia, which further aggravates lipid metabolism disorders and promotes adipogenesis.

At the cellular level, insulin resistance disrupts glucose transport, mitochondrial oxidative phosphorylation, and intracellular signaling pathways. These alterations contribute to cellular energy deficiency, increased oxidative stress, and impaired tissue repair.

The progression of insulin resistance is significantly influenced by behavioral determinants, particularly physical inactivity, excessive caloric intake, and weight gain during midlife.

Chronic Inflammation and Oxidative Stress

Menopause is associated with the development of a state of chronic low-grade inflammation. Visceral adiposity, insulin resistance, and endocrine imbalance collectively contribute to increased production of inflammatory mediators.

Proinflammatory cytokines activate intracellular signaling cascades that enhance oxidative stress and promote cellular damage. Reactive oxygen species accumulation disrupts membrane integrity, protein structure, and genomic stability, accelerating biological aging processes.

Chronic inflammation therefore represents a critical mechanism through which metabolic disturbances impair adaptive responses in menopausal women.

Circadian Rhythm Disruption

Age-related decline in melatonin secretion and alterations in sleep architecture are common features of the menopausal period. Circadian rhythm disruption affects hormonal secretion patterns, glucose metabolism, appetite regulation, and autonomic nervous system balance.

Sleep deprivation and circadian misalignment increase cortisol secretion and sympathetic activity, further exacerbating insulin resistance and visceral fat accumulation.

Accumulating evidence suggests that circadian dysregulation acts as an independent risk factor for metabolic disorders and significantly contributes to adaptive failure during menopause.

Behavioral Determinants of Menopausal Health

Nutritional Patterns

Dietary behavior plays a decisive role in modulating metabolic outcomes during menopause. Diets high in refined carbohydrates, trans fats, and ultra-processed foods promote inflammation and insulin resistance. In contrast, dietary patterns rich in fiber, antioxidants, and unsaturated fatty acids demonstrate favorable effects on metabolic stability.

Physical Activity

Regular physical activity improves insulin sensitivity, preserves skeletal muscle mass, and enhances mitochondrial function. Both aerobic and resistance training have been shown to reduce visceral adiposity and improve cardiometabolic markers in menopausal women.

Psychoemotional Factors

Chronic stress, anxiety, and depressive symptoms are common during menopause and exert profound neuroendocrine effects. Activation of the hypothalamic–pituitary–adrenal axis intensifies metabolic disturbances and further compromises adaptive capacity.

Clinical Implications and Preventive Perspectives

The convergence of endocrine decline, metabolic remodeling, and unfavorable behavioral patterns significantly increases long-term morbidity in menopausal women. Early metabolic maladaptation may remain clinically silent while progressively increasing cardiovascular and diabetic risk.

Implementation of preventive strategies during the menopausal transition represents a critical opportunity to modify disease trajectories. Integrated interventions combining lifestyle modification, metabolic screening, and psychosocial support appear most effective.

Such an approach aligns with modern concepts of personalized and preventive medicine.

Conclusion

Menopause constitutes a complex biological transition characterized by reduced adaptive reserve and heightened metabolic sensitivity. Estrogen deficiency initiates metabolic vulnerability, while behavioral and lifestyle-related factors largely determine clinical outcomes.

Visceral adiposity, insulin resistance, circadian disruption, chronic inflammation, and psychoemotional stress collectively contribute to the development of adaptive disorders in menopausal women.

Early identification of risk factors and implementation of comprehensive lifestyle-based interventions may substantially reduce long-term morbidity and improve quality of life during and after the menopausal transition.

References

1. Alberti, K. G. M. M., Eckel, R. H., Grundy, S. M., Zimmet, P. Z., Cleeman, J. I., Donato, K. A., … Smith, S. C. (2009). Harmonizing the metabolic syndrome: A joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention. Circulation, 120(16), 1640–1645. https://doi.org/10.1161/CIRCULATIONAHA.109.192644

2. Azziz, R., Carmina, E., Chen, Z., Dunaif, A., Laven, J. S. E., Legro, R. S., & Yildiz, B. O. (2016). Polycystic ovary syndrome. Nature Reviews Disease Primers, 2, 16057. https://doi.org/10.1038/nrdp.2016.57
3. Barber, T. M., Hanson, P., Weickert, M. O., & Franks, S. (2019). Obesity and polycystic ovary syndrome: Implications for pathogenesis and novel management strategies. Clinical Medicine Insights: Reproductive Health, 13, 1–9. https://doi.org/10.1177/1179558119874042
4. Bednarska, S., & Siejka, A. (2017). The pathogenesis and treatment of polycystic ovary syndrome: What's new? Advances in Clinical and Experimental Medicine, 26(2), 359–367. https://doi.org/10.17219/acem/59380
5. Bucci, D., Muscogiuri, G., Colao, A., & Savastano, S. (2020). Obesity, metabolic syndrome and infertility: A new therapeutic approach. Journal of Endocrinological Investigation, 43(7), 843–852. https://doi.org/10.1007/s40618-020-01214-8
6. Carter, S., & Davies, M. (2012). Metabolic syndrome and reproductive health. Best Practice & Research Clinical Obstetrics & Gynaecology, 26(1), 1–12. https://doi.org/10.1016/j.bpobgyn.2011.08.004
7. Diamanti-Kandarakis, E., Dunaif, A., & Katsikis, I. (2020). Insulin resistance and the polycystic ovary syndrome revisited: An update on mechanisms and implications. Endocrine Reviews, 41(2), 1–32. https://doi.org/10.1210/endrev/bnaa001
8. Duleba, A. J., & Dokras, A. (2012). Is PCOS an inflammatory process? Fertility and Sterility, 97(1), 7–12. https://doi.org/10.1016/j.fertnstert.2011.11.023
9. Escobar-Morreale, H. F. (2018). Polycystic ovary syndrome: Definition, aetiology, diagnosis and treatment. Nature Reviews Endocrinology, 14(5), 270–284. https://doi.org/10.1038/nrendo.2018.24
10. Fenkci, S. M., Fenkci, V., Yilmazer, M., & Serteser, M. (2003). Decreased total antioxidant status and increased oxidative stress in women with polycystic ovary syndrome. Gynecological Endocrinology, 17(4), 283–288. https://doi.org/10.1080/0951359031000111871
11. Gambineri, A., Pelusi, C., Vicennati, V., Pagotto, U., & Pasquali, R. (2002). Obesity and the polycystic ovary syndrome. International Journal of Obesity, 26(7), 883–896. https://doi.org/10.1038/sj.ijo.0801994
12. González, F. (2012). Inflammation in polycystic ovary syndrome: Underpinning of insulin resistance and ovarian dysfunction. Steroids, 77(4), 300–305. https://doi.org/10.1016/j.steroids.2011.12.003
13. Grundy, S. M. (2016). Metabolic syndrome update. Trends in Cardiovascular Medicine, 26(4), 364–373. https://doi.org/10.1016/j.tcm.2015.10.004
14. Ibrahim, M. A., & Abdel-Gadir, A. (2020). Ovarian morphology and metabolic syndrome: Pathophysiological correlations. Reproductive Biology and Endocrinology, 18, 1–10. https://doi.org/10.1186/s12958-020-00645-1