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Review Article

Diet–Gene Interactions Across the Female Hormonal Cycle: Implications for Personalized Nutrition

Amber Tehseen[1], Kainat Ijaz[1], Rania Faryan Yousaf[1], Malaika Amjad[1], Haleema Qadir[1], Abida Liaquat[2], Nimra Ather[1], Iqra[1], Anum Nazir[1]

[1] Department of Nutrition and Dietetics, The University of Faisalabad, Faisalabad, Pakistan
[2] Department of Nutritional Sciences, Government College Women University Faisalabad, Faisalabad, Pakistan

***Corresponding author: Amber Tehseen, ambertehseen.dnd@tuf.edu.pk; Anum Nazir, anumnazir.dnd@tuf.edu.pk**

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ABSTRACT

Background: Personalized nutrition increasingly recognizes that dietary response varies between individuals, yet sex-specific physiology and the cyclical hormonal environment of women remain underaddressed, leaving a gap in understanding how the menstrual cycle shapes nutrient metabolism and gene expression. Objective: This narrative review evaluates current evidence on diet–gene interactions across the female hormonal cycle and appraises its implications for personalized nutrition in women of reproductive and menopausal age. Methods: Literature was identified through a structured, systematic-style search of PubMed, Scopus, Web of Science, and Google Scholar, combining terms for personalized nutrition, the female hormonal cycle, and associated disorders; from 946 records identified and 800 screened after de-duplication, 96 sources informed a thematic qualitative synthesis, without formal risk-of-bias appraisal. Results: Estrogen- and progesterone-driven phase transitions modulate insulin sensitivity, substrate use, appetite, and inflammation, interacting with polymorphisms in FTO, VDR, MTHFR, ESR1/ESR2, PPAR-γ, and COMT to influence nutrient metabolism and disease risk; the estrogen-dominant follicular phase favours carbohydrate-oriented metabolism and the luteal phase lipid oxidation and relative insulin resistance. Nutrigenomic evidence is most developed for polyendocrine metabolic ovarian syndrome, whereas epigenetic and gut-microbiome mechanisms across premenstrual syndrome, endometriosis, and menopause remain the principal evidence gaps. Conclusion: Cycle-aware, genotype-informed dietary strategies show promise for improving metabolic and reproductive health, but current evidence is largely associative and non-representative; longitudinal, diverse, multi-omics research is needed to enable equitable, evidence-based personalized nutrition for women. Keywords: personalized nutrition; nutrigenomics; menstrual cycle; diet–gene interactions; endometriosis; menopause; polyendocrine metabolic ovarian syndrome

INTRODUCTION

Personalized nutrition has emerged as a central paradigm in preventive and therapeutic medicine, reflecting a shift away from population-level dietary guidance toward recommendations calibrated to an individual's genetic makeup, metabolism, and environmental and lifestyle context (1). Conventional guidelines implicitly assume a relatively uniform response to a given diet, yet accumulating evidence demonstrates that metabolic and inflammatory responses to identical nutrient exposures vary substantially between individuals (2, 3). This variability is increasingly understood through the complementary disciplines of nutrigenomics, which examines how nutrients and bioactive compounds modulate gene expression, and nutrigenetics, which examines how inherited genetic variation shapes the metabolic handling of nutrients (1, 4). Advances in high-throughput omics technologies, genomics, transcriptomics, metabolomics, and microbiome profiling, have accelerated this field, extending its

reach from basic mechanistic research into preventive medicine and disease management (3, 5). Despite this progress, sex-specific physiology remains insufficiently addressed within personalized nutrition research, and women in particular are underrepresented in the evidence base (6, 7). Across the menstrual cycle, cyclical fluctuations in estrogen and progesterone influence insulin sensitivity, appetite regulation, substrate utilization, lipid handling, inflammation, and energy expenditure, producing physiological states that differ meaningfully from one phase to the next (7, 8). During the follicular phase, when estrogen predominates, women tend to exhibit improved insulin sensitivity and greater reliance on carbohydrate oxidation, whereas the progesterone-dominant luteal phase has been associated with increased fat oxidation, heightened energy intake and cravings, and reduced metabolic flexibility (8). These shifts imply that dietary requirements are unlikely to remain constant across the cycle and that guidance derived predominantly from male or mixed-sex cohorts may inadequately serve women of reproductive age.

The interface between female endocrinology and the nutrigenomic response adds a further layer of complexity. Fluctuating hormone concentrations can alter the expression of genes governing inflammation, oxidative stress, neurotransmitter synthesis, reproduction, and energy metabolism, offering a partial explanation for why individuals respond differently to comparable diets (5, 9). Several nutrients and bioactive molecules, including omega-3 fatty acids, phytoestrogens, polyphenols, vitamin D, magnesium, and iron, interact with these hormonal and metabolic pathways, and their effects may themselves be conditioned by cycle phase. Hormonal status further shapes the gut microbiota and epigenetic regulation, mechanisms that in turn feed back on metabolism and immune function (2, 4). Taken together, these observations position the female hormonal cycle as a biologically meaningful, yet largely neglected, axis of interindividual variation in nutritional response.

The breadth and heterogeneity of this evidence, spanning endocrinology, molecular nutrition, epigenetics, microbiome science, and clinical medicine, lend themselves to narrative synthesis rather than to a single quantitative pooling, since the underlying studies differ widely in design, populations, and outcome measures and are not readily comparable through meta-analysis. A narrative approach permits integration across these disparate domains while foregrounding mechanistic plausibility and clinical relevance. An updated synthesis is also timely: recent methodological guidance for research involving women, the rapid maturation of multi-omics and wearable technologies, and evolving clinical frameworks for female metabolic and reproductive disorders have collectively reshaped the field over a short period, yet no accessible synthesis has connected cycle-specific hormonal physiology to diet–gene interactions and their downstream clinical implications (6, 10, 11). Accordingly, this review is organized around four interrelated threads: the metabolic characteristics of each menstrual cycle phase; the fundamentals of diet–gene interaction, including nutrient regulation of gene expression and epigenetic mechanisms; the hormone-related genes, among them ESR1 and ESR2, MTHFR, FTO, VDR, and COMT, alongside key metabolic loci, that shape nutritional response; and the female health disorders in which these interactions are clinically consequential, including polyendocrine metabolic ovarian syndrome, premenstrual syndrome, endometriosis, and menopause-related metabolic change. Within this framework, the objective of this narrative review is to evaluate current evidence on diet–gene interactions across the female hormonal cycle and to appraise its implications for personalized nutrition in women of reproductive and menopausal age (3, 12, 13). In doing so, the review also seeks to identify methodological limitations and research gaps and to outline directions for cycle-aware, genotype-informed nutritional strategies that may ultimately support metabolic health, reproductive outcomes, and chronic disease prevention.

MATERIALS AND METHODS

This review was conducted as a narrative synthesis of the literature on diet–gene interactions across the female hormonal cycle and their implications for personalized nutrition. A narrative approach was chosen deliberately, because the relevant evidence spans several methodologically distinct domains,

endocrinology, molecular nutrition, epigenetics, microbiome science, and clinical medicine, whose constituent studies differ markedly in design, populations, exposures, and outcome measures and are therefore not amenable to quantitative pooling. Narrative synthesis permits integration across these heterogeneous fields while preserving mechanistic reasoning and clinical interpretability. To enhance transparency and reduce arbitrariness in source selection, the literature was nonetheless identified and screened through a structured, systematic-style process, the flow of which is summarized in Figure 1.

A comprehensive literature search was performed across four electronic databases, PubMed, Scopus, Web of Science, and Google Scholar, to capture peer-reviewed evidence relevant to female-specific precision nutrition and nutrigenomics. The search combined controlled and free-text terms addressing three conceptual axes: personalized nutrition ("personalized nutrition," "nutrigenomics," "nutrigenetics," "diet–gene interactions," "epigenetics"), the female hormonal cycle ("menstrual cycle," "female hormone cycle," "estrogen," "progesterone"), and associated clinical conditions ("PCOS," "PMOS," "endometriosis," "premenstrual syndrome," "menopause"). These terms were combined using Boolean operators to construct database-specific queries, and the search emphasized contemporary peer-reviewed literature while retaining foundational studies that established key mechanistic or genetic concepts.

Eligible sources comprised clinical trials, observational studies, systematic reviews, and molecular nutrition studies involving women of reproductive or menopausal age, with a focus on hormonal fluctuation, nutrient metabolism, gene expression, and female health disorders relevant to personalized nutrition. Studies were excluded if they were restricted to male participants, examined unrelated metabolic conditions, were conducted solely in animal or in vitro models, or lacked sufficient methodological detail, and conference abstracts, editorials, and case reports were not considered. The database search yielded 865 records, supplemented by 81 records identified through other sources, for a total of 946 records; after removal of 146 duplicates, 800 unique records remained. Title and abstract screening excluded 634 records, leaving 166 articles for full-text assessment, of which 70 were subsequently excluded on the basis of non-female populations, animal or in vitro designs, insufficient nutrigenomic data, or unrelated outcomes. A final set of 96 sources informed the qualitative synthesis.

The retained literature was organized thematically rather than chronologically, structured around the metabolic characteristics of successive menstrual cycle phases, the fundamentals of nutrient–gene interaction and epigenetic regulation, the specific hormone-related genes that modulate nutritional response, the role of the gut microbiome, and the female health disorders in which these interactions are clinically consequential. Consistent with the nature of a narrative review, formal risk-of-bias assessment and quantitative quality appraisal were not undertaken, and the thematic selection and interpretation of evidence carry an inherent potential for selection bias; findings are therefore presented as an integrative synthesis of current understanding rather than as pooled effect estimates, and are interpreted with corresponding caution.

RESULTS AND SYNTHESIS

Female Hormonal Cycle and Metabolic Changes

The menstrual cycle comprises four physiologically distinct phases, menstrual, follicular, ovulatory, and luteal, governed principally by cyclical variation in estrogen and progesterone, which in turn modulate metabolism, appetite, insulin sensitivity, and nutrient handling (14). These hormonal transitions, together with their ovarian, endometrial, and metabolic correlates, are summarised in Figure 2. During the menstrual and early follicular phases, both hormones are low; as the follicular phase advances, estrogen rises while progesterone remains suppressed. Elevated estrogen is associated with improved insulin sensitivity, more stable glucose handling, and efficient carbohydrate metabolism, and women in this window tend to report lower appetite and energy intake than in the luteal phase (14). Estrogen appears to support metabolic regulation by promoting lipid oxidation, limiting visceral fat deposition, and optimising mitochondrial function, effects mediated in part by estrogen receptors expressed in

adipose tissue, skeletal muscle, liver, and the hypothalamus, where they regulate genes governing glucose transport and energy balance (15).

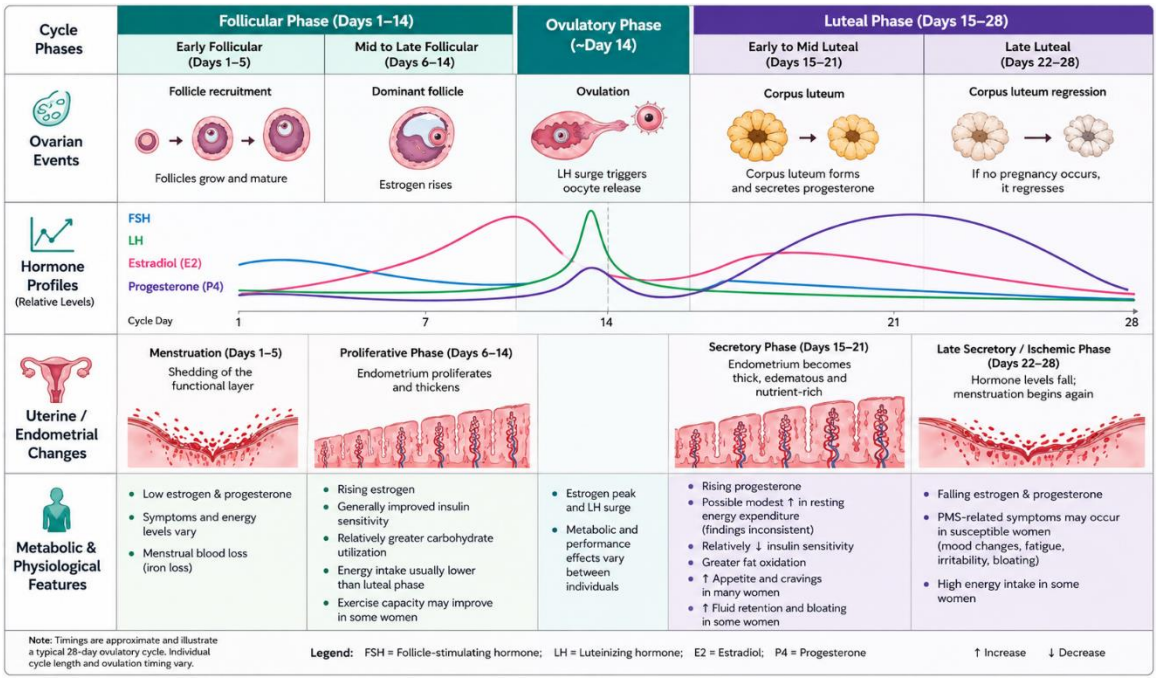

Figure 1 illustrates the ovarian, hormonal, endometrial, and metabolic changes occurring across an illustrative 28-day menstrual cycle, highlighting phase-related variation in insulin sensitivity, substrate utilization, energy expenditure, appetite, and premenstrual symptoms.

The ovulatory phase is characterised by peak estradiol and a luteinising hormone surge, representing a transitional metabolic state of relative insulin sensitivity and efficient energy utilisation, with some evidence of enhanced lipid oxidation (14). In the luteal phase, progesterone predominates alongside moderate estrogen and is associated with a rise in resting energy expenditure; some studies report increases of approximately 100–300 kcal per day, although findings across the literature are inconsistent, with other reports describing negligible change (17). This phase is also marked by relative insulin resistance and less efficient central insulin signalling, which may contribute to increased hunger, cravings for energy-dense foods, and greater fluctuation in blood glucose, accompanied by a shift toward lipid oxidation and glycogen sparing (18). These phase-dependent shifts translate into differing nutritional priorities across the cycle, summarised in Table 1.

Table 1. Nutrition strategies across the principal menstrual cycle phases

Cycle Phase	Hormonal Characteristics	Metabolic Changes	Nutrition Strategies	Reference
Menstrual	Low estrogen and progesterone	Fatigue, iron loss, reduced energy, inflammation	Iron-rich foods, vitamin C, anti-inflammatory foods, hydration	(14, 19)
Follicular	Rising estrogen	Improved insulin sensitivity and carbohydrate metabolism, increased energy	Complex carbohydrates, lean protein, fibre-rich foods	(14, 17)
Ovulatory	Peak estrogen and luteinising hormone	Increased metabolic activity, oxidative stress	Omega-3 fatty acids, healthy fats, antioxidant-rich foods, hydration	(14, 20)
Luteal	Rising progesterone with subsequent hormonal decline	Increased appetite and cravings, fluid retention, PMS symptoms	Higher protein intake, magnesium, calcium, vitamin B6, omega-3 fatty acids	(17, 19, 20)

Fundamentals of Diet–Gene Interactions

Diet–gene interactions describe the reciprocal relationship in which nutrients influence gene expression while inherited genetic variation shapes how nutrients are metabolised, providing the conceptual foundation of personalized nutrition (23). This foundation rests on two complementary disciplines. Nutrigenomics examines how nutrients and food-derived bioactive molecules alter gene expression, protein synthesis, and cellular function, often by modulating transcription factors, signalling pathways, and metabolic regulators (23). Nutrigenetics, conversely, examines how genetic polymorphisms,

variations in DNA sequence at defined loci, condition individual responses to nutrients; the MTHFR polymorphism influencing folate metabolism and methylation, and the FTO variant linked to obesity and appetite, are illustrative examples (24, 25).

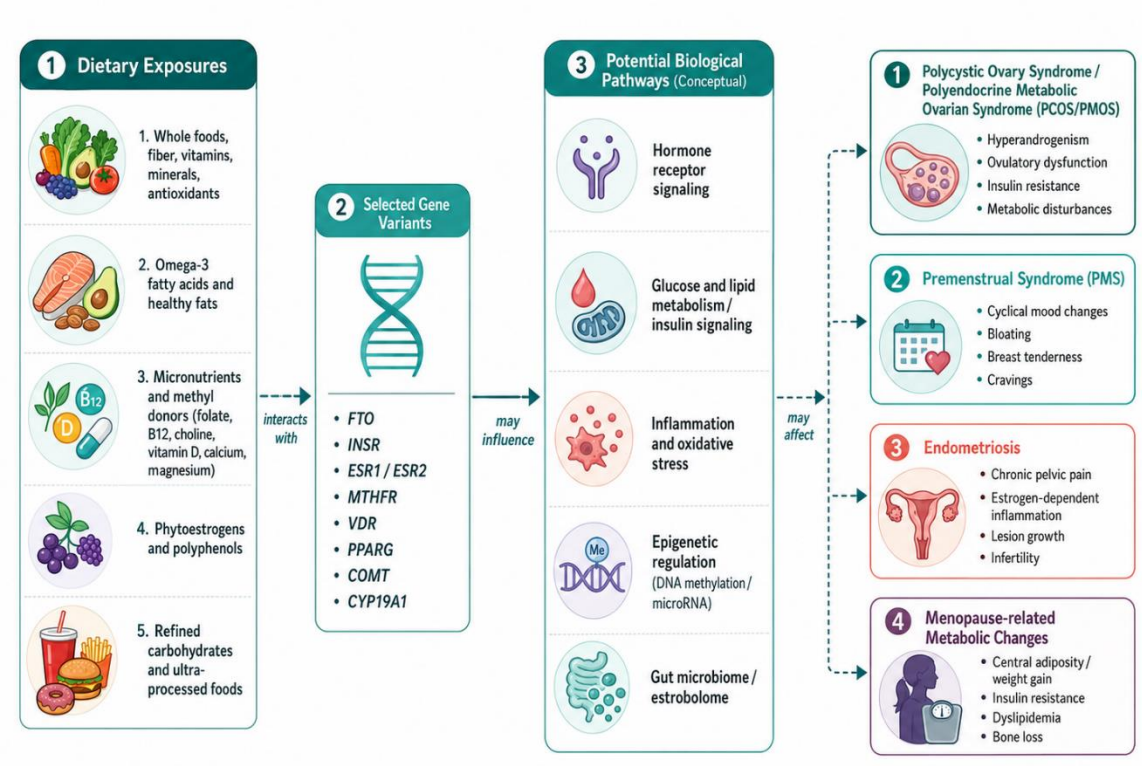

Figure 2 presents a conceptual framework showing how dietary exposures may interact with selected genetic variants and influence hormonal signalling, metabolism, inflammation, epigenetic regulation, and the gut microbiome in female reproductive and metabolic conditions.

Beyond their role as energy substrates, nutrients act as signalling molecules capable of regulating transcription. Macronutrients and micronutrients modulate transcription factors such as peroxisome proliferator-activated receptors (PPARs), sterol regulatory element-binding proteins (SREBPs), and nuclear receptors that govern lipid and glucose metabolism (26). Omega-3 polyunsaturated fatty acids can influence inflammatory gene expression through NF-κB and PPAR signalling, vitamins A and D bind their respective nuclear receptors to regulate immune and metabolic genes, and amino acids such as leucine affect genes involved in glucose metabolism and protein synthesis (27). Nutrition also acts through epigenetic mechanisms that alter gene expression without changing the underlying sequence. DNA methylation, dependent on one-carbon metabolism nutrients including folate, vitamin B12, choline, and methionine, is a central pathway, and aberrant methylation has been implicated in obesity, insulin resistance, cardiovascular disease, and infertility (43). MicroRNAs, short non-coding RNAs that regulate gene expression post-transcriptionally, are similarly responsive to dietary constituents such as polyphenols, omega-3 fatty acids, and phytochemicals, with downstream effects on inflammation, oxidative stress, and hormonal regulation (29). Together, these mechanisms explain how nutrition modulates the metabolic and hormonal processes central to female physiology.

Hormone-Related Genes Influencing Nutritional Response

Polymorphisms in hormone-related genes shape nutrient metabolism, energy homeostasis, and hormonal regulation, and understanding them is central to designing phase-aware nutritional strategies (30). Key genes, their functions, and their relevance to the female hormonal cycle are presented in Table 2. The estrogen receptor genes ESR1 and ESR2 encode ERα and ERβ, which regulate genes controlling glucose and lipid metabolism, inflammation, and reproduction, and are abundantly expressed in metabolically active tissues (26). Variants in these genes are associated with altered body-fat distribution,

insulin sensitivity, cardiovascular risk, and bone mineral density, and may influence how individuals metabolise dietary fats and phytoestrogens, such that women carrying particular variants may respond differently to plant-derived estrogens (40, 41). The MTHFR gene encodes an enzyme central to folate metabolism and one-carbon transfer required for DNA synthesis and methylation; carriers of reduced-activity variants may have greater requirements for folate, vitamin B12, and choline, particularly during periods of hormonal change and pregnancy, since methylation insufficiency has been linked to infertility, neural tube defects, cardiovascular disease, and hormone-related conditions (42, 43).

Table 2. Key genes and their relevance to the female hormonal cycle and associated health outcomes

Gene	Primary Function	Relationship with the Hormonal Cycle	Nutritional Relevance	Associated Outcomes	Reference
FTO	Appetite and energy balance	Hormonal shifts may modify appetite via estrogen-related pathways	High-calorie/high-fat diets may amplify obesity risk in variant carriers	Obesity, weight gain, insulin resistance	(31)
APOE	Lipid transport and cholesterol metabolism	Estrogen affects lipid metabolism and cardiovascular responses	Genotype influences response to dietary fat and cholesterol	Dyslipidaemia, cardiovascular disease	(32)
TCF7L2	Glucose metabolism and insulin signalling	Estrogen and progesterone alter insulin sensitivity and glucose regulation	Carbohydrate intake may modify diabetes risk in variant carriers	Type 2 diabetes, impaired glucose tolerance	(33)
PPAR-γ	Fat and adipocyte metabolism	Cyclical hormones may affect fat utilisation and adipose metabolism	Omega-3 and unsaturated fats may improve responses via PPAR-γ	Obesity, metabolic syndrome	(34)
VDR	Vitamin D metabolism and calcium regulation	Estrogen influences calcium balance and bone metabolism	Variants may affect vitamin D absorption and supplementation response	Bone health, osteoporosis, immune dysfunction	(35, 36)
MTHFR	Folate metabolism and methylation	Folate metabolism supports reproductive health and hormonal balance	Variant carriers may require higher folate intake	Hyperhomocysteinaemia, reproductive complications	(37)
ESR1 / ESR2	Estrogen receptor signalling (ERα/ERβ)	Directly mediate estrogen activity across reproductive phases	Dietary phytoestrogens interact with estrogen receptor pathways	Menopausal symptoms, bone health, hormonal disorders	(38)
SLC6A4	Serotonin transport and neurotransmitter regulation	Hormonal shifts affect serotonin activity and mood	Magnesium, vitamin B6, and omega-3 may modulate serotonergic pathways	Premenstrual syndrome, mood disorders	(39)
COMT	Catechol-estrogen and catecholamine metabolism	Modulates estrogen degradation and neurotransmitter regulation	Flavonoids and methyl donors may influence enzyme activity	PMS, mood disturbance, hormone-related cancer risk	(48)

The FTO gene, strongly associated with obesity, appetite, and energy balance, appears to interact with hypothalamic satiety and reward mechanisms, and women carrying risk variants may be more susceptible to weight gain during hormonally dynamic periods such as the luteal phase and menopause, though higher fibre and protein intake and physical activity may attenuate this predisposition (44, 45). The vitamin D receptor gene VDR mediates vitamin D action across calcium absorption, immunomodulation, and insulin secretion, and its polymorphisms may affect bone mineral density, osteoporosis risk, and susceptibility to metabolic dysfunction, with adequate vitamin D and calcium intake potentially offsetting unfavourable genotypes, an interaction of particular relevance around menopause given estrogen's role in bone remodelling (46, 47). Finally, COMT degrades catechol estrogens and catecholamines, and reduced enzyme activity, as in the Val158Met variant, may slow estrogen clearance and permit accumulation of reactive estrogen metabolites, promoting oxidative stress and inflammation; flavonoids and methyl donors may modulate this pathway, with possible relevance to premenstrual and mood-related symptoms (48).

Nutrient Requirements across the Hormonal Cycle

Nutritional requirements in women reflect physiological, neurological, and hormonal changes across the life course, and effective nutrition policy must accommodate this variation rather than focusing solely on caloric adequacy (50, 51). Because endogenous sex hormones shift across the menstrual cycle, single-timepoint assessments of intake or status may misrepresent true requirements and can confound intervention research when dietary exposure is inadequately controlled (51, 52). Resting metabolic rate is influenced by these hormonal fluctuations, though the literature is inconsistent, reporting lower rates in the follicular phase, higher rates in the luteal phase, or no significant difference, partly reflecting design limitations and the underrepresentation of women with obesity (53). Ovarian hormones and local

lipid mediators also remodel the endometrium across the cycle, with prostaglandins and endocannabinoid signalling contributing to endometrial receptivity, implantation, and early embryonic development (54, 55, 56).

Iron requirements are of particular importance during menstruation. Heavy menstrual bleeding is more common than generally recognised and is a major contributor to the widespread and underappreciated burden of iron deficiency, which affects quality of life and, in pregnancy, carries adverse obstetric and neurodevelopmental consequences (57, 58). Micronutrient status is likewise relevant to premenstrual symptoms; a clinical study of women with documented premenstrual syndrome found that magnesium citrate combined with vitamin B2 reduced neurological symptoms more effectively than vitamin B2 alone, and magnesium therapy is described as safe, well tolerated, and inexpensive, though the relationship between magnesium deficiency and premenstrual syndrome remains incompletely understood (59, 60, 61). Essential fatty acids, linoleic and alpha-linolenic acid, must be obtained from the diet and are metabolised differently across the female life course, such that supporting balanced intake through diet, breastfeeding, and physical activity is important for lipid homeostasis, particularly as menopause disrupts lipogenic and lipolytic pathways (62, 63). Calcium and vitamin D are similarly critical, with deficiency linked to osteoporosis and a range of metabolic disorders, and requirements are heightened by the greater calcium turnover of pregnancy and lactation (64).

Gut Microbiome, Hormones, and Gene Expression

The gut microbiome interacts bidirectionally with female sex hormones to influence host metabolism, and experimental models indicate that estrogen loss combined with high-fat feeding exacerbates intestinal permeability, inflammation, and metabolic dysfunction, accompanied by shifts in microbial composition and increased faecal β-glucuronidase activity (65, 66). Central to this relationship is the estrobolome, the collection of microbial genes encoding β-glucuronidases and β-glucosidases that deconjugate and reactivate estrogens, thereby regulating enterohepatic circulation and circulating estrogen bioavailability; alterations in estrobolome activity have been associated with estrogen-related conditions including polyendocrine metabolic ovarian syndrome, endometrial cancer, and breast cancer (67). Diet-induced changes in the microbiome are further shaped by sex-specific biological factors, including hormone action, immune divergence, and epigenetic influences, which together produce sex-differential patterns of disease susceptibility and treatment response (68). Through this gut–reproductive axis, the microbiome participates in the neuroendocrine, immune, and metabolic signalling that supports hormonal balance and reproductive health (69).

Personalized Nutrition Across Menstrual Phases

Because energy levels, hormonal status, and symptom burden vary across the cycle, nutritional needs differ meaningfully by phase, motivating cycle-aware dietary guidance (70). During menstruation, when fatigue, iron loss, and discomfort are common, iron- and antioxidant-rich, anti-inflammatory dietary patterns are appropriate (71). The follicular and peri-ovulatory phases, characterised by low then rising estradiol, are associated with a comparatively carbohydrate-oriented metabolic profile early on and greater lipid oxidation and metabolic efficiency as estrogen peaks (72). The luteal phase, marked by elevated progesterone and moderate estradiol, induces a distinct state of altered substrate use, increased energy expenditure, and reduced insulin sensitivity; the accompanying decline in serotonin may heighten appetite and increase intake of fats and carbohydrates, providing a physiological rationale for phase-specific attention to protein, magnesium, calcium, and mood-supporting nutrients (73, 74).

Diet–Gene Interactions in Female Health Disorders

Diet–gene interactions are clinically consequential in several hormone-related disorders, in which genetic susceptibility, endocrine function, and dietary exposure jointly influence onset and progression (19, 75). These relationships, and the dietary factors, pathways, and outcomes involved, are depicted in Figure 3. Polyendocrine metabolic ovarian syndrome, the recently adopted designation for polycystic

ovary syndrome, reflecting its multisystem endocrine and metabolic nature, is among the most common hormonal disorders of reproductive-age women and is strongly linked to insulin resistance, obesity, hyperandrogenism, and chronic low-grade inflammation (75, 77). Nutrigenomic evidence indicates that dietary patterns can influence gene expression in insulin signalling, steroidogenesis, inflammation, and lipid metabolism, and polymorphisms in FTO, INSR, PPAR-γ, and CYP19A1 have been associated with increased metabolic risk under high-calorie or high-glycaemic-index diets (75, 76). Interventions emphasising omega-3 fatty acids, low-glycaemic-index carbohydrates, and antioxidant and anti-inflammatory foods may improve insulin resistance and inflammation through effects on gene expression and epigenetic regulation, and emerging evidence implicates gut microbiota and dietary polyphenols in modulating hormonal dysregulation (76, 78, 79, 80).

Premenstrual syndrome comprises recurrent physical, emotional, and behavioural symptoms in the luteal phase, in which fluctuating estrogen and progesterone modulate neurotransmitter function, inflammation, and micronutrient metabolism (19, 81). Nutrients including magnesium, calcium, vitamin B6, vitamin D, and omega-3 fatty acids may regulate serotonin synthesis, stress responses, and inflammation, while polymorphisms in SLC6A4, ESR1, and cytokine genes may influence symptom severity and dietary responsiveness; conversely, high intake of processed sugar and saturated fat may aggravate symptoms through oxidative and inflammatory gene–nutrient interactions (81, 82). Endometriosis, a chronic estrogen-dependent inflammatory condition defined by ectopic endometrial tissue, arises from genetic predisposition, immune dysregulation, oxidative stress, and environmental factors, with angiogenesis, mediated in part by vascular endothelial growth factor, contributing to lesion establishment (83, 84). Dietary patterns rich in omega-3 fatty acids, antioxidants, fibre, and vegetables may suppress inflammatory signalling through pathways such as NF-κB and COX-2, whereas high intake of trans fats, processed foods, and red meat may enhance estrogenic activity and inflammatory gene expression, with epigenetic and microbiome-mediated effects increasingly recognised (84, 85, 86). Finally, menopause is accompanied by declining estrogen and a propensity toward central adiposity, insulin resistance, dyslipidaemia, oxidative stress, bone loss, and cardiovascular risk; nutritional interventions incorporating phytoestrogens, calcium, vitamin D, omega-3 fatty acids, and antioxidants may influence gene expression related to lipid and glucose metabolism, inflammation, and osteogenesis, with individual responses conditioned by variation in ESR1, ESR2, VDR, and lipid-metabolism genes (87, 88, 89).

DISCUSSION

This narrative review set out to evaluate current evidence on diet–gene interactions across the female hormonal cycle and to appraise its implications for personalized nutrition in women of reproductive and menopausal age. The synthesis indicates that cyclical variation in estrogen and progesterone constitutes a biologically meaningful axis of interindividual variability in nutritional response, acting through phase-dependent shifts in insulin sensitivity, substrate utilization, appetite, and inflammation, and interacting with polymorphisms in hormone- and metabolism-related genes to shape both physiological requirements and disease susceptibility (7, 8, 14). The estrogen-dominant follicular and peri-ovulatory phases are broadly associated with improved insulin sensitivity and carbohydrate-oriented metabolism, whereas the progesterone-dominant luteal phase favours lipid oxidation, heightened appetite, and relative insulin resistance, a pattern that provides a coherent, if not yet clinically standardized, rationale for cycle-aware dietary guidance (14, 17, 18).

These findings align with and extend prior work in several respects. The role of estrogen receptor signalling, folate-cycle enzymes, obesity-associated loci, and the vitamin D receptor in conditioning nutritional response is consistent with the broader nutrigenetic literature, and the recognition that nutrients function as signalling molecules capable of modulating transcription and epigenetic state situates female-specific findings within established mechanistic frameworks (26, 27, 43). Where this review advances beyond earlier syntheses is in integrating these gene- and nutrient-level mechanisms

with the temporal structure of the menstrual cycle and with the gut microbiome, particularly the estrobolome, whose regulation of enterohepatic estrogen circulation offers a plausible link between diet, microbial composition, and hormone-dependent disease (65, 67). At the same time, the synthesis surfaces genuine contradictions in the literature. The magnitude and even direction of cycle-related change in resting metabolic rate remain unsettled, with reports of luteal increases of approximately 100–300 kcal per day counterbalanced by studies finding negligible difference; this inconsistency likely reflects heterogeneity in cycle-phase verification, small samples, variable control of hormonal contraceptive use, and the underrepresentation of women with obesity, and it cautions against overinterpreting phase-specific energy prescriptions (17, 53).

The clinical implications differ by disorder, and the evidence gap map (Figure 4) makes the unevenness explicit. For polyendocrine metabolic ovarian syndrome, the recently adopted designation reflecting the condition's multisystem endocrine and metabolic nature, the nutrigenomic evidence is comparatively mature, with variants such as FTO, INSR, PPAR-γ, and CYP19A1 linked to metabolic risk that may be modifiable through low-glycaemic-index, anti-inflammatory, and omega-3-rich dietary patterns (75, 76, 77). For premenstrual syndrome, interventional signals for magnesium, vitamin B6, and calcium coexist with sparse epigenetic and microbiome data, and for endometriosis much of the dietary evidence remains observational and susceptible to confounding (59, 81, 84). Menopause-related metabolic change is supported by moderate nutrigenomic and interventional evidence centred on phytoestrogens, calcium, vitamin D, and estrogen-receptor and lipid-metabolism genes, yet its epigenetic and microbial dimensions are comparatively underdeveloped (87, 88). Across all four conditions, the epigenetic and gut-microbiome domains emerge as the principal evidence gaps, indicating that DNA-methylation, microRNA, and estrobolome-mediated pathways are more often hypothesised than empirically established in women.

Several limitations specific to this review warrant emphasis. As a narrative synthesis, it did not employ formal risk-of-bias appraisal or quantitative pooling, and although literature was identified through a structured, systematic-style search across four databases, the thematic selection and interpretation of sources carry an inherent potential for selection bias. The absence of a validated, fully reproducible search string and of a predefined date limit further constrains reproducibility, and the reliance on heterogeneous primary studies, differing in design, cycle-phase definition, and outcome measurement, means that many synthesized relationships remain associative rather than causal. The evidence density represented in Figure 4 is a qualitative appraisal derived from this synthesis rather than a quantitative count, and should be read as an interpretive map of the field's maturity, not as a measured effect. Finally, much of the underlying evidence derives from populations that may not reflect the genetic, dietary, and sociocultural context of women in South Asia and other under-studied regions, limiting direct generalizability.

These limitations point toward concrete future directions. Priority should be given to longitudinal, ethnically diverse studies that verify cycle phase objectively, control for hormonal contraceptive use, and integrate genotype with repeated within-cycle assessment of intake and metabolic response, thereby moving beyond single-timepoint designs that risk misrepresenting requirements (7, 8, 52). Multi-omics approaches combining genomics, epigenomics, metabolomics, and microbiome profiling are particularly needed in the epigenetic and estrobolome domains identified here as sparse, and continuous glucose monitoring, wearable sensors, and cycle-tracking applications offer scalable means to capture individual metabolic variability in real time (5, 10). Realizing the clinical promise of this field will require not only stronger mechanistic evidence but also attention to equity, data privacy, and the credibility of commercial personalized-nutrition offerings, alongside interdisciplinary collaboration among nutrition scientists, endocrinologists, geneticists, and public health specialists to translate cycle-aware, genotype-informed strategies into validated practice (4, 11, 96).

CONCLUSION

Diet–gene interactions across the female hormonal cycle represent an important and rapidly evolving dimension of personalized nutrition, in which cyclical estrogen and progesterone fluctuations interact with polymorphisms in genes such as FTO, VDR, MTHFR, ESR1/ESR2, PPAR-γ, and COMT to shape nutrient metabolism, appetite, insulin sensitivity, and susceptibility to obesity, polyendocrine metabolic ovarian syndrome, premenstrual syndrome, endometriosis, and menopause-related metabolic change. Current evidence supports phase-aware dietary strategies, adequate iron, omega-3 fatty acids, calcium, magnesium, vitamin D, antioxidants, and balanced macronutrients, as a plausible means of improving hormonal balance, metabolic flexibility, and reproductive outcomes, while nutrigenomic characterization is most mature for polyendocrine metabolic ovarian syndrome and epigenetic and gut-microbiome mechanisms remain the field's principal gaps. Recognizing that these conclusions rest on associative, heterogeneous, and often non-representative evidence, a clearer understanding of these interactions, advanced through longitudinal, diverse, multi-omics research and enabled by wearable and microbiome technologies, may ultimately support the development of equitable, evidence-based personalized nutrition strategies that improve women's metabolic and reproductive health, quality of life, and chronic disease prevention.

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