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THE INTERPLAY BETWEEN DIET AND HORMONAL REGULATION: A NARRATIVE REVIEW

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ABSTRACT

Diet is one of the key external factors that can modify hormonal activity across the major endocrine axes and affect metabolic and appetite-regulating hormones including insulin, leptin, ghrelin, and cortisol. A literature search conducted between October and November 2025 using PubMed, Scopus, Web of Science, and Google Scholar identified peer-reviewed studies examining the effects of caloric restriction, macronutrient distribution, glycemic load, micronutrient intake, and structured dietary interventions on endocrine function. The evidence shows that diets high in glycemic load, saturated fats, and low in fiber can disrupt insulin sensitivity, alter leptin and ghrelin signaling, affect cortisol responses, and contribute to thyroid and reproductive hormone imbalances. In contrast, Mediterranean-style and low-glycemic diets, time-restricted feeding, and adequate intake of iodine, selenium, and zinc appear to support metabolic health and more stable hormonal activity. Diet also plays a therapeutic role in endocrine-related conditions including polycystic ovary syndrome, Hashimoto's disease, acne vulgaris, endometriosis, and thyroiditis, with improvements seen in inflammation, insulin regulation, and hormonal homeostasis. Overall, the review emphasizes that diet can both disturb and restore endocrine balance. Well-structured, nutrient-dense dietary strategies may serve not only as preventive measures but also as valuable tools in supporting long-term hormonal health.

KEYWORDS

Diet And Endocrine Regulation, Hormonal Homeostasis, Metabolic and Appetite Hormones, HPA/HPT/HPG Axes, Dietary Interventions, Insulin Resistance

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Introduction

The human body is strongly influenced by a wide range of hormones it produces. Sensory and neural signals are converted into hormonal responses, with hormones released into the bloodstream to act on distant targets. Hormones are molecules produced by organs such as the hypothalamus, which secretes releasing and inhibiting hormones that regulate the pituitary gland and stimulate its hormone release. Some of them then influence other glands to release their own hormones or directly affect their target organs. Other glands that produce hormones are the adrenal glands, which primarily produce cortisol; the gonads (i.e., ovaries and testes), which produce sex hormones; the thyroid, which produces thyroid hormone; the parathyroid, which produces parathyroid hormone; and the pancreas, which produces insulin and glucagon. Each of them serves a specific function and influences the others. The main functions comprise growth, development, and metabolism of the body, the electrolyte composition of bodily fluids, and reproduction. Hormones can be divided into classes such as steroids, amino acid derivatives, and polypeptides and proteins. Due to differences in molecular structure, the mechanisms of action differ. From mundane functions such as eating to complex fertility processes, it all depends on the balance between the axes - ie. the hypothalamic-pituitary-adrenal axis (HPA), the hypothalamic-pituitary-gonadal axis (HPG), and the hypothalamic-pituitary-thyroidal axis(HPT).

The HPA axis begins with the release of CRH, which stimulates the anterior pituitary to produce ACTH, which in turn triggers cortisol production by the adrenal glands. Cortisol regulates carbohydrate, protein, and lipid metabolism. It increases blood glucose concentration by stimulating gluconeogenesis in the liver. In high-stress situations, it protects the body from the harmful effects of trauma.

The release of TRH from the hypothalamus commences the HPT axis. TRH stimulates TSH production in the pituitary, which then allows the thyroid gland to release thyroxine (T4) and triiodothyronine (T3). These hormones are crucial for cellular metabolism throughout the human body.

The HPG axis controls the release of sex hormones. In both men and women, GnRh is released from the hypothalamus and stimulates the production of FSH and LH from the anterior pituitary. In men, LH stimulates Leydig cells in the testes to release testosterone. FSH and testosterone regulate Sertoli cells, which support the sperm cells. In women, LH and FSH take part in the menstrual cycle. They stimulate the ovarian follicle to produce estradiol. The estradiol peak triggers a surge in LH, which promotes ovulation. After ovulation, the corpus luteum releases progesterone, which, through negative feedback, lowers FSH and LH levels. Once the corpus luteum breaks down, progesterone levels drop, and the cycle begins again. For the whole system to run smoothly, there must be continuous feedback from target organs to the hypothalamus and pituitary gland.

Other hormones, such as insulin, glucagon, ghrelin, and leptin, are influenced by different stimuli. Insulin is released from the pancreas beta cells in response to a glucose peak in the blood. However, it can also be stimulated by other hormones, such as glucagon and those that influence blood glucose levels (e.g., GH, glucocorticoids, and thyroid hormones). Glucagon acts antagonistically by increasing blood glucose levels when they are low[1]. Ghrelin and leptin act as opposites: ghrelin increases hunger and promotes food intake, while leptin suppresses appetite and reduces food intake. They act as a result of binding to the satiety receptors in the brain[2].

Certain factors can influence hormone production, including diet. It can have a negative impact, ie, be the cause of the development of a disease (such as obesity)[3], or can be used as a therapeutic option in some conditions (such as infertility)[4]. Given its potential to treat various ailments, it is worth investigating how different diets affect human hormones.

Methodology

This narrative review was conducted to synthesize current evidence on the relationship between dietary patterns and hormonal regulation in humans. The review aimed to integrate findings from endocrinology, nutrition science, and metabolism to provide an updated overview of how specific nutrients, dietary behaviors, and structured dietary interventions influence major hormonal axes, including the hypothalamic–pituitary–adrenal (HPA), hypothalamic–pituitary–gonadal (HPG), and hypothalamic–pituitary–thyroid (HPT) axes, as well as appetite-regulating and metabolic hormones.

The literature search was performed between October 2025 and November 2025 using electronic databases including PubMed, Scopus, Web of Science, and Google Scholar. The search strategy combined Medical Subject Headings (MeSH) with relevant free-text terms to identify studies examining the effects of diet and dietary patterns on hormonal regulation. Core MeSH terms included concepts related to diet (*Diet*, *Caloric Restriction*, *Diet, Mediterranean*, *Diet, Carbohydrate-Restricted*), major hormonal systems (*Insulin*, *Leptin*, *Ghrelin*, *Hydrocortisone*, *Thyroid Hormones*, *Gonadal Steroid Hormones*), and endocrine-related disorders (*Polycystic Ovary Syndrome*, *Hashimoto Disease*, *Endometriosis*, *Acne Vulgaris*). These were combined using Boolean operators to capture a broad range of experimental and clinical evidence.

Studies were considered eligible if they met the following criteria:

1. published in peer-reviewed journals;
2. written in English;
3. focused on human participants or relevant translational models;
4. addressed dietary influences on endocrine pathways, hormonal secretion, or hormonal disorders.

Randomized controlled trials, meta-analyses, systematic reviews, observational studies, and mechanistic studies were included. Case reports, conference abstracts, non–peer-reviewed sources, and studies lacking clear methodological description were excluded.

Two authors independently screened titles and abstracts, followed by full-text assessment. Any discrepancies were resolved through discussion with the wider research team. Reference lists of selected articles were scanned to identify additional relevant publications.

Given the heterogeneity of study designs, populations, and dietary exposures, a narrative synthesis approach was chosen rather than a quantitative meta-analysis. Extracted data were organized into thematic sections corresponding to key hormonal systems: glucose and insulin homeostasis, appetite-regulating hormones, cortisol and the stress response, thyroid hormones, sex hormones, and diet-related therapeutic implications for endocrine disorders.

The narrative approach allowed for integrating mechanistic insights with clinical outcomes, highlighting both established evidence and emerging research gaps. The final synthesis aimed to provide a comprehensive overview of diet–endocrine interactions and to identify potential directions for future research.

Insulin and Glucose Homeostasis

Following carbohydrate ingestion, the rise in blood glucose stimulates insulin secretion from pancreatic β-cells and suppresses glucagon release from α-cells. This coordinated endocrine response rapidly reduces hepatic glucose output, allowing the liver to take up a substantial portion of the absorbed glucose, much of which is directed toward glycogen formation during the first pass. Substantial hepatic extraction of insulin moderates its entry into the systemic circulation, preventing excessive peripheral hyperinsulinemia. The insulin that reaches peripheral tissues suppresses adipose tissue lipolysis, lowering circulating non-esterified fatty acid (NEFA) levels and thereby further supports hepatic glucose regulation. At the same time, insulin enhances skeletal muscle glucose uptake, where glucose is either oxidized or stored as glycogen. This ensures controlled postprandial glucose disposal. When circulating glucose exceeds the oxidative and glycogenic capacities of skeletal muscle, some of the excess glucose is diverted to alternative metabolic pathways. Muscle cells convert it into intermediates such as lactate, alanine, and glutamine. These metabolites are exported into the circulation and subsequently taken up by the liver, where their carbon skeletons are reconverted into glycogen, providing an additional route for disposing of surplus carbohydrate [5].

Dietary patterns that moderate the glycemic impact of meals appear to be especially important for managing postprandial glucose handling and supporting insulin sensitivity. Diets that emphasize low-glycemic foods—particularly when they are combined with protein or fat—tend to blunt the rise in glucose and insulin after a meal, which in turn favors better insulin action. Limiting large swings in blood glucose is particularly relevant, as they contribute to oxidative stress and β-cell strain. In addition to nutrient composition, everyday eating behaviors, such as the speed of meal consumption, may influence glycemic responses, with rapid

consumption linked to greater postprandial glucose peaks. Meals that slow carbohydrate absorption by providing high soluble fiber, modest amounts of digestible carbohydrate, and higher protein content can lessen the insulin requirement of a meal and may also help moderate appetite. Since carbohydrate quality and quantity are the dominant drivers of glycemic responses, factors like the proportion of rapidly absorbed carbohydrates, the overall glycemic load, accompanying macronutrients, and even the timing of carbohydrate intake all shape the resulting metabolic profile [5]. A comprehensive meta-analysis comparing nine dietary approaches in adults with type 2 diabetes found that, while all patterns improved HbA1c and fasting glucose, the magnitude of benefit varied. Low-carbohydrate and Mediterranean-style diets produced the largest reductions in HbA1c, whereas Mediterranean and vegetarian diets were particularly effective at lowering fasting glucose. Age also appeared to influence outcomes: individuals ≥60 years showed greater HbA1c improvements with low-carbohydrate diets, while those <60 years tended to benefit more from Mediterranean-style, moderate-carbohydrate, high-protein, and low-fat patterns. Overall, the Mediterranean-style diet stood out as the most consistent dietary strategy for improving postprandial hyperglycemia and insulin resistance across the interventions examined [6]. Intermittent fasting appears to reduce plasma insulin levels and improve insulin sensitivity, yet the specific physiological pathways driving these effects remain unclear, underscoring the need for further research on β-cell function, insulin clearance, and related regulatory mechanisms [7]. Very restrictive versions of low-carbohydrate diets, including ketogenic diets, may temporarily worsen hepatic insulin action, highlighting the importance of monitoring carbohydrate restriction. Still, dietary patterns that reduce the glycemic burden of meals and limit large postprandial spikes generally yield superior outcomes in glycemic control and insulin sensitivity compared with high-glycemic eating patterns [5].

Leptin and ghrelin-appetite-regulating hormones

Leptin and ghrelin are two major hormones that exert opposite effects in controlling appetite and energy homeostasis. Ghrelin enhances hunger and food consumption by binding to its receptors and activating orexigenic neurons in the arcuate nucleus. Leptin, in contrast, has been shown to inhibit appetite and decrease food intake. Disruptions in leptin and ghrelin signaling have been associated with obesity and other metabolic conditions [2].

Leptin is a 146-amino-acid peptide hormone secreted by white adipose tissue, mammary epithelial cells, and bone marrow, and it readily crosses the blood–brain barrier. Initially, leptin was believed to promote satiety, lower food intake, increase energy expenditure, and contribute to weight loss, but newer findings indicate that sensitization, desensitization, and leptin resistance influence its physiological effects. During fasting, both stored and circulating triglycerides fall, leading to reduced leptin release [8].

CR (calorie reduction) and weight loss are recognized modulators of leptin. CR decreases leptin levels independently of body-weight changes and disrupts its circadian rhythm. This decline in leptin has been linked to reduced subjective appetite and diminished compensatory eating; however, it remains unclear whether the drop in leptin directly corresponds to the extent of caloric compensation [9]. TRF (time-restricted feeding) — whether based on 6-h, 8-h, or 12-h eating windows or Ramadan-style fasting — without intentional CR or weight loss has been shown to lower fasting leptin concentrations [10]. Conversely, studies applying TRF without CR, regardless of weight change, have reported no significant alterations in leptin levels [11]. After a period of CR, leptin concentrations return to pre-restriction levels once energy intake is restored.

Ghrelin is a 28-amino-acid peptide hormone predominantly produced by X/A cells in the stomach, especially in the gastric fundus. It is released before meals, with circulating levels peaking just prior to eating and dropping within 1 h after food intake. Ghrelin stimulates growth hormone secretion from the anterior pituitary and also affects homeostatic mechanisms of appetite, taste perception, and reward-related behaviors [12].

Circulating ghrelin follows a diurnal rhythm in both humans and rodents, driven by sleep-related suppression and independent of meal timing. Ghrelin rises during fasting, while refeeding reduces its plasma concentration [13]. Across studies, inconsistencies exist regarding whether active (acylated) or total ghrelin is measured. Despite this variability, CR has consistently led to significant increases in fasting ghrelin levels. The capacity of TRF to modify ghrelin concentrations remains uncertain.

Cortisol and the Stress Response

Cortisol is the principal glucocorticosteroid (GC) synthesized by the adrenal glands, specifically within the zona fasciculata of the adrenal cortex, and is widely referred to as the "stress hormone." It plays a central role in the body's stress response system, which is regulated by the hypothalamic–pituitary–adrenal (HPA) axis. Beyond this, cortisol participates in numerous physiological functions, including metabolism, regulation of blood glucose levels, immune function, growth, cardiovascular health, mood, cognition, reproduction, and development [14].

Over recent decades, studies have shown that low-carbohydrate (LC) diets have several beneficial health effects, including promoting weight loss, lowering triglyceride levels, and raising high-density lipoprotein cholesterol [15].

The rise in resting cortisol observed during short-term—but not long-term—LC diets is likely linked to glucocorticoids' role in maintaining glucose balance. Cortisol, glucagon, and gluconeogenesis all increase in the early stages of LC diets, suggesting that the initial cortisol elevation may partly drive a temporary rise in gluconeogenesis. Cortisol may also increase to preserve glucose for the brain, which cannot rely on fatty acids for substantial energy. Glucocorticoids reduce glucose uptake and oxidation in skeletal muscle and adipose tissue, thereby reserving glucose for cerebral use. It has also been noted that cortisol elevations during exercise are more pronounced when following LC diets [16].

Diet composition can additionally influence cortisol patterns. Shively et al. showed in non-human primates that following a Mediterranean (MED) diet led to lower cortisol responses to acute stress and ACTH stimulation, along with enhanced resilience to stress. Moreover, Carvalho et al. found that the MED diet mitigated associations between various cortisol markers and inflammatory processes [17,18]. Specific polyphenol-rich foods, such as *Hibiscus sabdariffa,* have also been linked to cortisol regulation, suggesting that these compounds may help modulate the body's stress response [19]. According to Alufer et al., long-term adherence to a Green-MED diet (rich in vegetables and emphasizing poultry and fish) and high in polyphenols may primarily lower fasting morning cortisol levels in individuals with obesity, which, in turn, could improve metabolic health indicators [20].

Thyroid Hormones and Metabolism

Adequate iodine intake is critical throughout life, particularly during pregnancy, infancy, and adolescence, as it is essential for thyroid hormone synthesis. Iodine is absorbed in the small intestine primarily as iodide and transported to the thyroid gland, where it contributes to the production of triiodothyronine (T3) and thyroxine (T4). Maternal iodine deficiency impairs thyroid hormone synthesis in both mother and fetus, and severe or chronic deficiency during early gestation can result in irreversible neurodevelopmental deficits. Even mild to moderate iodine insufficiency has been associated with lower cognitive performance in childhood, including reduced literacy and numeracy scores at age nine. In adolescents, inadequate iodine intake increases the risk of nodular goiter, whereas excessive intake may predispose to thyroid autoimmunity. Age-specific intake recommendations range from 70 μg/day in young children to 150 μg/day in late adolescence, and universal salt iodization remains the most effective population-level intervention to prevent iodine deficiency [21].

Selenium plays a central role in thyroid hormone regulation by being incorporated into selenoproteins, which include enzymes involved in antioxidant defense, thyroid hormone activation and deactivation, and steroid hormone synthesis. Selenium-dependent deiodinases modulate thyroid hormone metabolism and support proper functioning of the hypothalamic-pituitary-thyroid (HPT) axis. Additionally, selenium enhances the activity of antioxidant enzymes such as glutathione peroxidases and thioredoxin reductases, protecting hormone-producing tissues from oxidative stress. To avoid toxicity, supplementation should remain within the recommended range of 80–400 μg/day [21].

Zinc is also vital for thyroid function, particularly for the activity of thyroid peroxidase (TPO), which catalyzes the iodination of thyroglobulin and coupling of iodotyrosine residues to form T4 and T3. Zinc possesses antioxidant properties and is essential for proper thyroid hormone signaling. Deficiency can impair hormone action, contribute to hypothyroidism, and affect hair growth, as it is a cofactor for metalloenzymes involved in pigmentation and follicular function. Zinc deficiency has also been associated with increased thyroid size in children and adults. Clinical evidence suggests that zinc supplementation may support thyroid function in hypothyroid patients and reduce levothyroxine requirements [21].

Caloric restriction (CR) has significant effects on thyroid hormone homeostasis, primarily by reducing circulating T3 [22,23,24]. These changes appear to result from energy limitation itself rather than from

reductions in fat mass [22,23]. In a randomized trial, both CR and exercise led to comparable fat loss, yet only the CR group experienced a marked decrease in plasma T3, while TSH, T4, and free T4 levels remained stable, indicating an adaptive response to reduced caloric intake [22]. Long-term CR in healthy, lean adults with adequate protein and micronutrient intake produces sustained lower T3 concentrations without affecting T4, reverse T3, or TSH, paralleling findings from animal studies [23]. Baseline thyroid hormone levels also appear to influence the early response to energy restriction, with higher initial free T3 and T4 associated with greater weight loss during the initial phases of dieting [25]. Very low-calorie ketogenic diets have similarly been reported to reduce circulating T3, particularly in women with obesity, suggesting that both the magnitude and type of energy restriction shape thyroid physiology [24].

Undernutrition triggers adaptive modifications in the HPT axis to conserve energy. Circulating T3 declines early during fasting or short-term caloric restriction and remains suppressed during prolonged starvation, whereas T4 and TSH are generally maintained or only slightly reduced. These reductions reflect central suppression at the hypothalamic and pituitary levels rather than primary thyroid dysfunction and are often accompanied by elevated reverse T3. Notably, T3 levels recover with weight restoration, supporting the concept that these changes represent a physiological adaptation to prolonged energy deficiency [26].

In contrast, obesity is associated with heightened HPT axis activity, typically manifesting as modestly elevated TSH and circulating T3 or free T3. Chronic overnutrition and hyperleptinemia can induce leptin resistance, which reduces the HPT axis's responsiveness to nutritional signals. This resistance disrupts energy balance, weakens leptin-mediated stimulation of TRH and thyroid hormone production, and may contribute to the maintenance of obesity despite elevated leptin levels [27].

Sex Hormones and Diet

High-fat, low-fiber dietary patterns are increasingly recognised as contributing to disturbances in sex steroid hormone homeostasis. Such diets promote expansion of adipose tissue (particularly visceral fat), which enhances peripheral aromatization of androgens to estrogens and may alter the entero-hepatic recirculation of estrogens via changes in gut microbiota diversity and fibre-mediated bile acid metabolism. This mechanism is implicated in elevated circulating estrogens and sometimes androgens in people consuming Western-type diets. In women of reproductive age, observational data show that poor diet quality (including high fat and low fibre content) correlates with insulin resistance, increased free androgen index, and ovarian dysmorphology, via mediating influences of body mass index (BMI), waist circumference, and metabolic markers [28].

The role of phytoestrogens in modulating estrogen metabolism remains controversial. A recent expanded meta-analysis of randomized controlled trials in men found that neither soy protein nor isoflavone intake produced significant changes in total testosterone, free testosterone, estradiol, or estrone levels, regardless of dose or duration [29]. While this suggests a limited effect on male steroid profiles, extrapolation to female reproductive endocrinology is not straightforward; the influence of phytoestrogens on estrogen metabolism in women—particularly in the context of insulin resistance or obesity—remains to be investigated.

In conditions characterised by hyperandrogenism and metabolic dysfunction (e.g., polycystic ovary syndrome [PCOS] and obesity), interventions that result in weight loss and improved insulin sensitivity frequently lead to normalization of testosterone and estrogen levels, restoration of sex-hormone-binding globulin (SHBG) concentrations, and improvements in ovulatory function. Data suggest that improved insulin sensitivity is a key mediator of these hormonal changes [30]. Accordingly, lifestyle interventions aimed at reducing adiposity, enhancing dietary quality, increasing fibre intake, and improving metabolic health should be considered as integrative approaches to modulate steroid hormone balance and support reproductive function.

Recent literature has increasingly emphasized the roles of diet, metabolic health, and lifestyle factors in regulating sex hormone balance and fertility outcomes in both men and women. Evidence from a comprehensive analysis of clinical trials demonstrates that neither soy intake nor isolated isoflavones exerts clinically meaningful effects on circulating total or free testosterone, estradiol, or sex hormone–binding globulin in men, challenging the persistent notion that phytoestrogens induce feminizing hormonal changes[29]. Similarly, contemporary reviews examining dietary protein intake suggest that the impact on testosterone concentrations is dose-dependent: extremely high protein intake, expressed relative to body weight, may lead to reductions in total testosterone, whereas moderate or conventionally "high" protein diets do not consistently demonstrate adverse endocrine effects [30]. These findings highlight the importance of quantitatively defining protein intake when interpreting or formulating dietary recommendations.

In women, particularly those with polycystic ovary syndrome (PCOS) or obesity-related metabolic dysfunction, growing evidence indicates that weight reduction and improved insulin sensitivity are central to the restoration of reproductive endocrine homeostasis. Interventions such as increased physical activity, caloric restriction, Mediterranean-style dietary patterns, and insulin-sensitizing therapies are frequently associated with normalization of androgen and estrogen levels, improved ovulatory function, and enhanced fertility outcomes. Mechanistically, these improvements appear to be mediated through reduced systemic inflammation, enhanced insulin signaling, and favorable shifts in the gut microbiome, all of which contribute to the reestablishment of hypothalamic–pituitary–ovarian axis function [4].

Therapeutic and Lifestyle Implications

Polycystic Ovary Syndrome (PCOS)

Dietary patterns are a critical determinant in the management of disorders arising from hormonal dysregulation. Excessive fructose consumption has been shown to exacerbate endocrine, but not metabolic, alterations in polycystic ovary syndrome (PCOS), suggesting its potential to worsen endocrine-related phenotypic manifestations. Evidence from a recent review indicates that reducing total energy intake, together with adopting a low-calorie, low–glycaemic index (GI) diet, represents an important therapeutic strategy. Furthermore, the adoption of a low-GI diet, caloric restriction, and/or regular physical activity, combined with omega-3 supplementation, has been associated with increased high-density lipoprotein (HDL) concentrations, enhanced synthesis of sex hormone-binding globulin (SHBG), and reduced adipose tissue mass. In addition, adherence to a ketogenic diet has been reported to improve menstrual cycle regularity, reduce blood glucose levels, facilitate weight loss, and improve hepatic function. Overall, a well-balanced diet that supports appropriate insulin regulation constitutes a primary therapeutic approach in the management of PCOS. The consumption of herbal infusions—such as aloe vera, cinnamon, green tea, chamomile, and white mulberry—may serve as a beneficial adjunct to conventional therapy [31].

Hashimoto's Disease

Diet can also serve as a complementary treatment in Hashimoto's disease by influencing thyroid function and providing anti-inflammatory benefits. Anti-inflammatory nutrients such as vitamin D, antioxidants, fatty acids, magnesium, and zinc are important for reducing thyroid inflammation. Among patients with Hashimoto's disease, celiac disease is more prevalent; therefore, in such cases, it is advisable to eliminate gluten from the diet [32].

Acne

Genetic predispositions, hormones, the skin and gut microbiome, psychological stress, air pollution, harsh facial products, and certain medications have been reported to influence the development of acne. Diet is widely recognized as directly related to specific biochemical markers and gene transcription associated with sebaceous gland function, as well as to bacterial proliferation and inflammatory processes contributing to the disease. An imbalance between omega-6 and omega-3 fatty acids is believed to play a significant role in acne development. A diet rich in dairy products, carbohydrates, chocolate, and saturated fats may exacerbate acne by activating metabolic signals from these foods. Other products, including alcohol, salty foods, gluten, eggs, biscuits, corn, fruits, sweets, cola, or soft drinks, have also been reported to contribute to the promotion and worsening of acne lesions. Saturated fatty acids, primarily from hydrogenated vegetable fats found in margarine, sweets, or fast food, negatively affect skin affected by acne vulgaris [33].

Endometriosis

In patients with endometriosis, adherence to diets such as FODMAP, gluten-free, Mediterranean, or anti-inflammatory diets helps reduce pain perception and overall endometriosis symptoms. The review remains unclear whether a single dietary or nutritional intervention is the most appropriate adjuvant therapy. A personalized approach that considers the patient's comprehensive clinical history and lifestyle may be beneficial. Additionally, curcumin inhibits endometrial cell proliferation by lowering E2 levels. Treating endometriotic stromal cells with curcumin also significantly suppresses TNF-α-induced secretion of IL-6 and IL-8, as well as TNF-α expression. Vitamin C is an essential micronutrient that humans cannot synthesize and must obtain from the diet. It plays a key role in enhancing cellular functions in both the innate and adaptive immune systems and acts as a powerful antioxidant. Furthermore, the severity of painful menstruation and dyspareunia was significantly reduced in the group treated with vitamins C and E, suggesting their potential in reducing reactive oxygen species (ROS) in endometriosis [34].

Thyroiditis

According to the study, after approximately 6 months of a gluten-free diet (GFD), there was a tendency toward decreased anti-thyroid antibodies, including anti-Tg (TgAb) and anti-TPO (TPOAb). The TSH (thyroid-stimulating hormone) level decreased significantly. There was also an improvement in the absorption of nutrients important for thyroid function. The effect of a GFD on enhanced absorption of selenium, zinc, vitamin D, and iodine—all essential micronutrients for thyroid function—is still under investigation [35].

Conclusions

Diet plays an important role in shaping the endocrine system. Hormones responsible for growth, development, metabolism, and reproductive health are influenced by the nutrients we consume and the eating habits we adopt. The review demonstrates that diet can both disorganize and repair hormonal balance. Excessive caloric intake, high-glycemic-load diets, low fiber consumption, and Western-type nutrition patterns contribute to insulin resistance, dysregulated appetite hormones, chronic inflammation, and impaired reproductive and thyroid axes. On the other hand, dietary strategies such as low-glycemic or Mediterranean-style diets, time-restricted feeding, balanced macronutrient distribution, adequate intake of critical micronutrients, and targeted weight reduction can significantly improve hormonal profiles and metabolic health.

Moreover, conditions such as PCOS, Hashimoto's disease, acne vulgaris, endometriosis, and thyroiditis display clinical improvement when patients apply custom diets to their therapeutic plan. It shows that diet should not be viewed only as a preventative measure but also as a therapeutic tool capable of restoring hormonal balance.

Future research should aim to explore our understanding of the influence on endocrine-related conditions. By understanding the interplay between the two, there is a promise of incorporating personalized nutrition as a therapeutic tool in treatment plans. Overall, integrating high-quality nutrition, appropriate meal timing, and balanced nutrient intake may be one of the most effective and accessible ways to promote hormonal stability and prevent endocrine disease.

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