Obesity Medicine 40 (2023) 100495

Contents lists available at ScienceDirect

Obesity Medicine

journal homepage: www.elsevier.com/locate/obmed

Impact of junk food on obesity and polycystic ovarian syndrome: Mechanisms and management strategies

Rukaiah Fatma Begum, Ankul Singh S, Sumithra Mohan *

Department of Pharmacology, SRM College of Pharmacy, SRM Institute of Science and Technology, Kattankulathur, 603209, Tamil Nadu, India

ARTICLE INFO

Keywords:
Obesity
Polycystic ovarian syndrome
Junk food
insulin resistance
Transgenerational inheritance

ABSTRACT

The prevalence of obesity among women worldwide has escalated to 26%, and among adolescent girls, it is 18%. An elevated BMI is closely associated with metabolic and gynecological issues in women. PCOS is a serious and frequently prevalent obesity-related comorbidity that manifests in girls and women genetically prone to it. A cross-sectional study examined the intake of several types of junk food in 200 girls with and without menstrual abnormalities by investigating their menstrual patterns, anthropometric measures, and eating frequency. It found that junk food consumption was substantially related to menstrual difficulties. Junk food slows down the body's metabolism and reduces the calories it burns, making it challenging to maintain a healthy weight. Junk food indirectly affects androgen levels through IR. Elevated insulin levels cause the decline of sex hormone-binding globulin (SHBG), a regulatory protein that suppresses the activity of androgens in females and causes hyperandrogenism when cytokines cause IR. There is a correlation between the current young society and junk food which lead to obesity and its complications. Its already been proven that consuming junk food rapidly and frequently results in binge and overeating without reaching satiety and limiting the amount of energy consumed. Obesity and junk eating are inherently connected with hormones. In the globalized era, when there is an abundance of fast food and sedentary lifestyles foster weight gain, polygenic obesity is the most prevalent sort of obesity. A highly integrated gut-to-brain neuroendocrine system controls appetite and body weight by monitoring both short- and long-term fluctuations in energy intake and expenditure. Several diet regimens, like the ketogenic diet, DASH diet, low GI diet, etc, make it easier to cut portion sizes and extra sugar and fat drastically. Provided our knowledge of the underlying mechanisms behind obesity and reproductive diseases, certain strategies should emphasize nutrition and lifestyle for treatment and management.

1. Introduction

Obesity is a prevalent cause of all metabolic and endocrine issues (Kurylowicz, 2021; Powell-Wiley et al., 2021). Obesity is the term for excessive fat deposition, which increases the probability of every complication. Through the body mass index (BMI), which is calculated using weight, height, and age, obesity-related metabolic diseases determine our health. An elevated BMI is closely associated with metabolic and gynecological issues in women, such as polycystic ovarian syndrome (PCOS), abnormal uterine bleeding (AUB), miscarriage, and infertility (Douchi et al., 2002; Venkatesh et al., 2022; Wei et al., 2009). The prevalence of obesity among women worldwide has escalated to 26%, and among adolescent girls, it is 18% (Mizgier et al., 2020). Since the 21st century, obesity has been called a global pandemic as its spread from adolescent to adults due to junk food are evident. Junk food is defined as calorie

* Corresponding author.
E-mail address: sumithrm@srmist.edu.in (S. Mohan).

https://doi.org/10.1016/j.obmed.2023.100495
Received 30 January 2023; Received in revised form 3 April 2023; Accepted 19 April 2023
Available online 20 April 2023

dense foods which are high in fat, salt, cholesterol, and sugar content but low in nutritional content. It also contains high amount of refined carbohydrates and sodium along with preservatives. For instance, Chocolate, candy, potato chips, trans-fats, refined grains, salt, high fructose corn syrup etc. (Chapman and Maclean, 1993; Shankar et al., 2014) Junk food has low nutrient density, vitamins, or minerals and provides calories through refined carbohydrates, fats, and added sugar in contrast to regular food which has high nutritional value. This is unhealthy food that contains high levels of saturated fats, trans-fatty acids, and refined sugar, which is the underlying cause of obesity and affects the body physically, psychologically, and socially (Ertz and Le Bouhart, 2022).

According to an analysis of epidemiological data, 38–88% of women with PCOS are overweight or obese (Jaacks et al., 2019). This demonstrates how closely junk food, obesity, PCOS, and many other gynecological complications are interrelated. PCOS is a serious and frequently prevalent obesity-related comorbidity that manifests in girls and women genetically prone to it (Barber et al., 2006). PCOS affects women or adolescents at an early age, although it is rarely effectively diagnosed at the beginning (Koivuaho et al., 2019). It presents the classic clinical signs of hyperandrogenism, such as oligo-amenorrhea and concomitant subfertility, as well as acne, hirsutism, and male-pattern alopecia (Barber and Franks, 2021). Although there is no cure for PCOS, the condition must be appropriately controlled because failure to comply could result in infertility. Considering a description of insulin resistance, androgen excess, and other PCOS-related concerns, we investigate the relationship involving PCOS and junk food and obesity, including strategies to effectively manage it through dietary and lifestyle modifications for overweight and obese women with or without PCOS.

2. Junk food in adolescent girls

According to a study, the prevalence of junk food intake was high among schoolgirls. A correlation between intake and obesity was largely dependent on BMI depending on the type of junk food consumed, which included high calorie, sugar, and salt content (Table 1) in adolescents, which can contribute to early cardiovascular disorders (CVD) and other complications (Mohammadbeigi et al., 2019).

Junk food consumption and decreased physical activity can cause premenstrual symptoms, dysmenorrhea, and irregular menstrual cycles, which can subsequently affect a woman's health and quality of life. A cross-sectional study examined several types of junk food intake in 200 girls with and without menstrual abnormalities by investigating their menstrual patterns, anthropometric measures, and eating frequency. It found that junk food consumption was substantially related to menstrual difficulties (Latif et al., 2022). Menstrual abnormalities are common in teenage girls and can sometimes be normal but can lead to different conditions such as amenorrhea, dysmenorrhea, and polycystic ovaries. BMI plays an important role in menstrual disorders, which are affected mainly due to junk food, psychological stress, and lifestyle factors (Singh et al., 2019). Overeating, food cravings, and a change in appetite are other premenstrual symptoms that may be observed in 70–80% of women. These desires are caused by the progesterone influence throughout the menstrual cycle, which boosts hunger and junk food intake in the presence of oestrogen (Hirschberg, 2012). According to research on cravings for junk food during menstruation, 86% of youngsters had increasing appetites before and after their cycle, contrary to 70% of them during. Therefore, a large intake of junk food in younger generations could be attributed to premenstrual symptoms (Yukie et al., 2020).

Table 1
Metabolic disorders due to junk food in adolescents.

S. No.	Junk Food	Composition	Metabolic disorders
1	Burger, hotdog, bacon, sausage	Sodium nitrite and sodium nitrate	Blood pressure and metabolic syndrome, including PCOS, diabetes mellitus, and infertility. (Mattila et al., 2020; Oghbaei et al., 2020)
2	Maida (pasta, noodles, doughnut, white bread, paratha, pizza)	Monosodium glutamate (MSG), Azodicarbanamide	PCOS, impaired ovary and uterus, infertility. (Mondal et al., 2018) Endocrine disrupter, infertility, thyroid diseases. (Gafford et al., 1971; Ganga et al., 2020; Maranghi et al., 2010)
3	High sugar content sweets (cake, muffins, sweet juices, carbonated drinks, pastry, ice creams)	Sulfites, 2- methyl imidazole and 4- methylimidazole, Ponceau	Type 2 diabetes mellitus, PCOS, urinary incontinence (Douglas et al., 2006; Ringel et al., 2022; Schulze et al., 2004)
4	Fried foods (chicken, French fries, chips)	Butylated hydroxyanisole (BHA)	Carcinogenic (Adeyemi, 2021; Römsing et al., 2003)
5	Sauces (soya, fish, tomato)	Monosodium glutamate	PCOS and infertility (Mondal et al., 2018)
6	Mayonnaise	Butylated hydroxyanisole (BHA), trans and saturated fats	Carcinogenic (Felter et al., 2021)
7	Canned foods (beans, fish, meat, vegetables,	Monosodium glutamate, nitrites, sulphites, benzene, Butylated Hydroxyanisole 320 (BHA), Butylated Hydroxytoluene 321(BHT), Bisphenol A (BPA)	Uterine leiomyoma and breast cancer (Chiang et al., 2021; Engin and Engin, 2021; Kwon, 2022)
8	Ready-to-eat processed foods (Maggie, frozen snacks)	Monosodium glutamate, Benzoates, nitrites, sulphites	Diabetes mellitus, cardiovascular diseases (Banerjee et al., 2021; Brial et al., 2022)
9	Cheese processed foods (macaroni)	Tartrazine	Precocious puberty, endocrine disrupter, embryotoxic, and teratogenic (Hashem et al., 2019; Mindang et al., 2022)

3. Junk food, binge disorder and PCOS in underweight girls

Body weight is a constant concern, whether for slender or fat girls. In adolescent girls, underweight and junk food consumption are correlated because of their dysfunctional eating habits, which have been discussed in the literature and are influenced by ethnocultural variables. These variables encourage teenagers to develop strategies to change their body weight (Fogelkvist et al., 2020). These strategies increase the intake of junk food and household confectionaries in skinny adolescent girls, which can affect them psychologically and lead to eating disorders (EDs). EDs are mental illnesses that cause aberrant eating patterns that are detrimental to both physical and mental health. The most prevalent ED among teens is binge eating disorder (BED), which is attributed to high caloric intake over a specified time period and a sensation of eating-related helplessness (Alvero-Cruz et al., 2020). These can cause premenstrual symptoms and increase the risk of PCOS in lean and slim girls, despite the fact that they are intended for overweight and obese girls.

PCOS initially appears in adolescence, when physiological, psychological, and hormonal changes begin to emerge. However, optimizing lifestyle variables at a young age is the primary treatment strategy for PCOS. A case-control study of adolescent girls (13–18 years) with and without PCOS reported that binge eating was not substantially distinct between the two groups, despite the fact that only 13% of the girls were overweight or obese (Lidaka et al., 2022). Even in sports, binge eating has been common; a study was undertaken to evaluate athletes' dysfunctional eating habits with a questionnaire examining psychological and nutritional components owing to the primary risk factor of body image disturbance which led them to risk factors including premenstrual symptoms, PCOS, and other associated issues (Ramona et al., 2021). This demonstrates that BED is prominent, including lean, slender, and fit individuals as well as overweight and obese teenagers, putting the entire community at risk for metabolic and endocrine issues.

4. Impact of junk food on obesity

The frequency of overweight and obesity in the younger generation is developing as the variety of junk foods and consumption increases. This contributes to the increased complications and risks in public health (Nixon and Doud, 2011; Singh S et al., 2021). The affordability, flavor, accessibility, and variety of junk food on the market are the key factors contributing to this rise. Due to peer pressure in their daily lives, students find it easier to satiate with junk food without realizing the impact of the latter on their bodies (Shah, 2014). However, this is not the sole cause of obesity; there are other lifestyle choices that also play an impact, which is discussed in Table 2. Obesity, individually or with other factors, leads to non-communicable diseases like precocious puberty, early menopause, PCOS, infertility, cardiovascular complications, hypertension, and diabetes mellitus (Hassanzadeh et al., 2012). A study conducted on medical and science students reported that 67.4% of females, of which 21% were obese, consumed fast food every day. Therefore the consumption of junk food does not depend upon the BMI or the background of the person (Mohammadbeigi et al., 2018).

5. Impact of junk food on insulin resistance

Large amounts of junk food consumption have an impact on metabolism. All the chemical processes that happen within our bodies are referred to as the metabolism. It is closely related to the number of calories expended each day. Junk food slows down the body's metabolism and reduces the amount of calories it burns, which makes it challenging to maintain a healthy weight. The energy required to metabolize food is termed the thermic effect of food (Westerterp, 2004). Due to the high content of refined carbohydrates and partially hydrogenated oils in junk food, it needs less energy from the body to digest. As a result, eating a high amount of junk food with high glucose-salt fat content causes the body to deposit more fat. Insulin resistance is closely correlated with extra fat accumulation around abdominal organs. According to a long-term prospective study, there is a clear correlation between junk food consumption, body weight, and insulin resistance (IR), which also enhances the likelihood of developing type 2 diabetes (Pereira et al., 2005).

Based on an experimental investigation, junk food may cause obesity in women by interfering with the hormonal processes that control hunger and appetite. Junk food includes free fatty acids (FFAs), which are produced from adipocytes and are associated with lipotoxicity, and the enhancement of IR. Adiponectin, a hormone generated from adipose tissue, improves insulin sensitivity. In comparison to individuals with normal BMI, obese people secrete less adiponectin (Goldfine and Kahn, 2003). Adipocytes, generally alluded to as lipocytes or fat cells, produce a number of cytokines that trigger IR, which is associated with PCOS and other metabolic syndromes (Rangwala et al., 2004).

Obesity and junk eating are inherently connected with hormones. Despite health harm and the dysregulation of mediators of the neuroendocrine, autonomic, immunological, and metabolic systems, junk food and environmental stress increase rates of obesity and metabolic syndrome. Cognitive impairment is mediated by the inactivation of the insulin receptor in the hippocampus of the brain,

Table 2
Obesity due to lifestyle factors.

Lifestyle factors	Pathological conditions
Increased stress levels	Obesity, PCOS, and Cardiovascular (CV) risk (Daiber and Chlopicki, 2020; Shan et al., 2022)
Insomnia or difficulty sleeping.	Weight gain, PCOS, hypertension, diabetes mellitus (Demir Çaltekin et al., 2021; Hachul et al., 2019; Li et al., 2021)
Fatigue	Obesity, PCOS (Norris et al., 2017; Vgontzas and Calhoun, 2009)
Overeating (binge eating)	Obesity, CV risk, and metabolic syndrome (Hudson et al., 2010; Martinez-Avila et al., 2020; McCuen-Wurst et al., 2018)
Sedentary lifestyle (due to technology)	Obesity, PCOS, diabetes mellitus, CV diseases (Bakrania et al., 2016; BUOITE STELLA et al., 2021; Figueiró et al., 2019; Tay et al., 2020)

whereas IR, PCOS, and dyslipidemia are prompted by the inactivation of the hypothalamic insulin receptor (Grillo et al., 2015). Insulin encourages energy storage; however, in cases of obesity and aging, insulin-sensitive cells are disturbed and produce IR by overproducing adenosine triphosphate (ATP), which causes mitochondrial malfunction. Through a feedback mechanism, ATP blocks the synthesis of the mitochondria, causing enzyme activity and ATP production. In pancreatic islets, there is an overproduction of ATP, which leads to the hypersecretion of insulin in beta cells and promotes IR through intracellular and extracellular activities. When food and lifestyle are altered, mitochondrial function reverts, allowing cells' insulin sensitivity to function normally (Ye, 2021). This IR will cause major complications, including PCOS, infertility, type 2 diabetes, CVD, and other illnesses if it is not corrected in a timely approach (Łakoma et al., 2023).

6. Impact of junk food on androgen hormones

The hormones known as androgens, which mostly include testosterone and androstenedione, are responsible for maintaining masculine traits in both men and women. A minimal quantity of testosterone is released in females, mostly for reproductive development. A rise in testosterone can result in a number of issues for women's bodies, including acne, hirsutism, alopecia, menstrual irregularities, insulin resistance, hypertension, obesity, an increase in muscle mass, low libido, mood changes, and a deep husky voice (Skiba et al., 2019). Junk food indirectly affects androgen levels through IR. Elevated insulin levels cause the decline of sex hormone-binding globulin (SHBG), a regulatory protein that suppresses the activity of androgens in females and causes hyperandrogenism when cytokines cause IR. Hyperinsulinemia also contributes to PCOS ovarian sensitivity (Qu and Donnelly, 2020). This is one of the processes by which junk food causes hyperandrogenemia; additional mechanisms are possible but have yet to be investigated. Unfortunately, there is scant evidence of the link between junk food and androgen excess. This may be the focus of the researchers' forthcoming study. Although it is evident in the studies of PCOS that junk food is one of the etiology for the metabolic syndromes (Pramodh, 2020; Vidya Bharathi et al., 2017).

7. Insights of the mechanism on junk food on obesity and PCOS

There is a correlation between the current young society and junk food which lead to obesity and its complications (Sharma et al., 2022). Its already been proven that consuming junk food rapidly and frequently results in binge and overeating without reaching satiety and limiting the amount of energy consumed (Andrade et al., 2012). As mentioned above, carbonated drinks are available more easily than pure water nowadays due to technology and development (Magni et al., 2009). The major concern of modern society is consuming too much junk containing artificial sweeteners, preservatives, MSG, and much more chemicals that are harmful to our body and organs, making it develop complications at an early stage of life (Malik et al., 2006). With the changes in the global environment, the prevalence of obesity is increasing, mainly in the urban population (Blauw et al., 2017). Some of the possible mechanisms that exist and are well understood are discussed.

7.1. Microbiota

Junk foods have a direct correlation with the gut microbiota and determine the microbiome composition. The microbiomes of individuals that reside in the same environment are not related genetically but have a significant amount of similarities (Corrie et al., 2023). Both independent factors are linked to food, medications, and anthropometric measures and account for about 20% of interpersonal heterogeneity in microbiota (Rothschild et al., 2018). Microbes that live in the gastrointestinal (GI) tract are important in human health as they have an influence on the genes and metabolites which have the ability to maintain body weight (Greathouse et al., 2017). Studies have revealed that the gut microbiota's microbiome composition transmits afferent signals to the sympathetic (SNS) and parasympathetic (PNS) nervous system that has an impact on energy homeostatic systems, regulating both intake and energy balance, including eating behavior, whether normal and excessive energy reserves (Rosenbaum et al., 2015). Human exposure to microbiota may alter a person's genetic predisposition towards obesity and have an impact on weight management (Greathouse et al., 2017). Furthermore, the gut microbiota increases the body weight as it influences the parts of CNS which regulates energy and decreases the expression of brain-derived neurotrophic factor (BDNF) and glucagon-like peptide (GLP-1) as it is the factors responsible for anti-obesity in the body of an individual. This reduction mediates the obesogenic effect in the gut microbiota (Schéle et al., 2016).

7.2. Neurobiological evidence

The Nucleus Accumbens (NAc) area in the hypothalamus's basal forebrain is important for controlling behaviors like eating and craving. Medium spiny neurons (MSNs) present in the NAc core control obesity and binge eating. A study in rodents revealed that junk food alters MSN excitability by manipulating voltage-gated potassium channels, which disrupts the function of the dopamine receptor and increases adiposity and fat accumulation (Oginsky and Ferrario, 2019). Another clinical study demonstrated that binge eating triggers the NAc substantially even before obesity appears (Demos et al., 2012) and slowly influences the mesolimbic function and receptor expression, which leads to obesity. However, it is still unknown how drastically the consumption of junk food and the development of obesity and its problems damage these neurochemical systems (Robinson et al., 2015).

7.3. Genetic mechanism

In the globalized era, when there is an abundance of fast food and sedentary lifestyles foster weight gain, polygenic obesity is the most prevalent sort of obesity. In comparison between obese and normal people, there are more than 100 BMI-associated loci. These genes are linked to obesity using the genome-wide association (GWA) technique called fat-mass obesity-associated genes (FTO) (Woo et al., 2022). Multiple predisposing factors, such as gene variations, are believed to interact with a lifestyle to boost the likelihood of

obesity. This interplay between genes and environmental variables is believed to contribute to obesity. FTO alleles regulate food intake and are directly associated with binge eating, reduced satiety, dietary fat, and energy (Albuquerque et al., 2017). Studies were conducted to examine the risk of FTO gene polymorphism carriers with a high-fat diet that elevates the risk of obesity. The greatest genetic factor for obesity presently recognized is the FTO gene. A major environmental factor, dietary fat, may work in concert with genes to raise the risk of obesity and metabolic syndrome, and CVD (Phillips et al., 2012). Since there are billions of obese people around the globe, a comprehensive understanding of the mechanism underpinning the above findings may promote the therapy of precision medicine using a nutritional approach to alleviate the complications associated with obesity.

8. Transgenerational inheritance of PCOS (pre and clinical data)

The primary PCOS symptom, hyperandrogenism, is frequently prevalent in PCOS-affected females. Dehydroepiandrosterone (DHEA), dehydroepiandrosterone sulfate (DHEAS), and androstenedione are the most frequently elevated androgens expressed in excess in 60% of females. First-degree relatives are more likely to have PCOS in a family aggregation. Due to this, PCOS is categorized as an autosomal dominant genetic illness with peri-pubertal onset (Crespo et al., 2018). According to preclinical research, excessive androgen exposure may contribute to chronic problems in both the exposed group and the succeeding generation. The incidence of metabolic and endocrine abnormalities in PCOS offspring was also demonstrated to be significantly correlated with the prenatal and early postnatal environments of PCOS women's pregnancies (Zhang et al., 2020). The pathophysiology of human PCOS, which causes PCOS and other metabolic complications throughout development, is related to these transgenerational consequences (Subramaniam et al., 2019). Numerous animal experiments have shown that prenatal androgen exposure impacts IR, gonadotrophin secretion, hyperandrogenism, and ovulatory disruption (Sanchez-Garrido and Tena-Sempere, 2020). Since the placenta plays a significant role in maternal-fetal androgen metabolism, determining the function of the predisposing state is complicated. However, it has been shown that the term placenta of women with PCOS exhibits impaired placental aromatization due to higher placental inflammation of maternal androgens, which could be the cause of increased fetal exposure to maternal androgens (Koster et al., 2015; Kumar et al., 2018).

In a study conducted on pregnant women, High anogenital distance (AGD) values were calculated using PCOS symptoms before conception. AGD is measured as the distance between the base of the genital tubercle and the caudal extremity of the fetus, and this distance is influenced by both fetal sex and testosterone. AGD represents fetal testosterone exposure in the female fetus of PCOS-pregnant mothers (BAYRAKTAR and TANER, 2023). In some clinical pieces of evidence, Anti-mullerian hormone (AMH) expression was determined in umbilical cord blood, which was found to be higher in neonates born to PCOS mothers as AMH plays a critical part in PCOS pathogenesis and inhibits folliculogenesis in the fetal stage (Zhou et al., 2022).

9. Management and therapeutics of obesity and PCOS

A highly integrated gut-to-brain neuroendocrine system controls appetite and body weight by monitoring both short- and long-term fluctuations in energy intake and expenditure (Schwartz et al., 2017). For survival, the body's ability to store surplus energy as fat is essential. Both the body's resting metabolic rate and the energy used during physical activity can be reduced by 20% or more when there is a food shortage. Furthermore, humans must reinforce the neuroendocrine regulation of body weight with behavioral and cognitive attempts to regulate their eating and physical activity (Hawley et al., 2020). Lifestyle and diet modifications are crucial first steps to resolve this. Restricting calories is necessary to cause clinically significant weight reduction. According to US Dietary guidelines, 10% or less of daily calories ought to be from saturated fat, 20%–35% of daily calories must be brought from fat, and the remaining calories must come from carbohydrates, especially from fresh fruits, vegetables, and grains (Table 3). Several diet regimens, like the ketogenic diet, Dietary approaches to stop hypertension (DASH) diet, low GI diet, etc., make it easier to drastically cut portion sizes as well as extra sugar and fat (Ludwig, 2020; Wadden et al., 2020).

Typically, lifestyle change programmes recommend 150–180 min per week of moderately intense aerobic activity, such as brisk walking or cycling. Regular aerobic exercise includes various advantages, including enhancing physical and mental health (blood pressure, cholesterol, etc.). (e.g., anxiety, depression). Strengthening fitness via physical exercise is also linked to a significantly reduced mortality risk from obesity (Powell et al., 2019).

10. Conclusion

The prevalence of junk food, obesity, and its linked complications like PCOS is projected to rise as the world's technological growth accelerates. Correlation between current young society lifestyles associated with junk food leads to obesity and significantly triggers the NAc even before obesity appears to affect hormonal balance. It eventually leads to a worsening of the PCOS disorder. Provided our knowledge of the underlying mechanisms behind obesity and reproductive diseases, there should be certain strategies that emphasize nutrition and lifestyle for treatment and management. The competence to therapeutically modulate the expression of genes and transgenerational inheritance that contribute to the tendency to become obese and, eventually, to promote effective weight reduction and weight maintenance by providing each patient with precision medicine and an empathetic approach.

Funding

This research received no grant from any funding agency.

Table 3
Management of obesity.

Types of diet	Food included in the diet	Significance of the diet for obesity and PCOS
Dietary approaches to stop hypertension (DASH) diet	Whole wheat bread and pasta, pita bread, cereals, oatmeal, brown rice, green vegetables, fruits, dry fruits, fat-free milk products, frozen yogurt, lentils, kidney beans	Effective tool to reduce body weight and manage obesity by limiting high-calorie intake. (Perry et al., 2019)
Low glycemic index (GI) diet	High protein foods such as lean meat and fish, dairy products such as milk and yogurt, unsweetened soy milk; vegetables such as broccoli, green peas, and leafy greens; low sugar fruits such as apples, oranges, and blueberries, oatmeal, legumes, and pulses	Delays the absorption of carbohydrates and improve metabolic pathways and insulin resistance (IR), and weight loss in women with or without PCOS. (Shishehgar et al., 2019)
Anti-inflammatory diet	Vegetables: broccoli, cabbage, cauliflower, fruits: pomegranates, grapes, cherries, healthy fats: olive oil, avocado oil, fish, nuts, bell peppers, spices: fenugreek, turmeric, cinnamon, green tea, dark chocolate	Improvement in body composition, anthropometry, liver parameters, reducing visceral adiposity, metabolic and inflammatory biomarkers. Also reduces PCOS symptoms and the risk of getting PCOS (Kenđel Jovanović et al., 2021; Panjeshahin et al., 2020)
Mediterranean diet	Vegetables, fruits, nuts, legumes, potatoes, whole grains, herbs, seafood, olive oil, spices, fish, rarely eat eggs, cheese, and yogurt.	Improves BMI, anthropometric parameters, nutrition, quality of sleep, and protection against IR-related diseases such as obesity, T2DM, CVD, CKD, PCOS, and breast cancer. (Mirabelli et al., 2020; Muscogiuri et al., 2020)
Ketogenic diet	Low carb diet includes eggs, chicken, fish, dairy, nuts, seeds, healthy fat oil such as olive oil, avocado oil, and sesame oil, non-starchy vegetables such as greens, broccoli, tomatoes, mushrooms and pepper, lemon juice, fresh herbs, and spices.	Reduce BMI, body weight, and waist circumference. Improves HBA1c, triglycerides, lipid and liver parameters, and PCOS. (Adeyemi, 2021; McPherson and McEneny, 2012)
Pulses based diet	Lentils, Kidney beans, chickpeas, dry peas, beans	Weight loss, reduce waist circumference, blood pressure, triglyceride, cholesterol (total, low-density lipoprotein (LDL)), glycaemic load (Marinangeli and Jones, 2012; Venn et al., 2013)
Low carb diet	Meat, fish, eggs, non-starchy vegetables: spinach, broccoli, cauliflower, carrots, asparagus, tomatoes, low carb fruits: oranges, blueberries, strawberries, nuts, seeds, dairy products, avocado oil, olive oil, coconut oil	Obesity, and T2DM improve glycemic control, and hyperinsulinemia, improve hepatic parameters, and reduce risk of CVD, and PCOS (Avolio et al., 2020; Bolla et al., 2019; Goss et al., 2020)

CRediT authorship contribution statement

Rukaiah Fatma Begum: Methodology, Writing – original draft, Investigation, Resources. **Ankul Singh S:** Data curation, Writing – review & editing. **Sumithra Mohan:** Writing – review & editing, Conceptualization, Supervision.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgement

We would like to express our sincere gratitude to the faculty of SRM college of Pharmacy, SRMIST for their endeavour and support in completing this work.

References

Adeyemi, K.D., 2021. Comparative effect of dietary Morinda lucida leaf and Butylated hydroxyanisole (BHA) on carcass traits, meat quality, and oxidative stability of broiler chickens. J. Food Sci. Technol. 58, 4359–4369. https://doi.org/10.1007/S13197-020-04916-2/TABLES/6.

Albuquerque, D., Nóbrega, C., Manco, L., Padez, C., 2017. The contribution of genetics and environment to obesity. Br. Med. Bull. 123, 159–173. https://doi.org/10.1093/bmb/ldx022.

Alvero-Cruz, J.R., Mathias, V.P., García-Romero, J.C., 2020. Somatotype components as useful predictors of disordered eating attitudes in young female ballet dance students. J. Clin. Med. 9, 1–12. https://doi.org/10.3390/JCM9072024.

Andrade, A.M., Kresge, D.L., Teixeira, P.J., Baptista, F., Melanson, K.J., 2012. Does eating slowly influence appetite and energy intake when water intake is controlled? Int. J. Behav. Nutr. Phys. Activ. 9, 135. https://doi.org/10.1186/1479-5868-9-135.

Avolio, E., Ferraro, S., Mocciaro, R., De Pergola, G., Buzzanca, F., Venturella, R., Pecorella, C., Palma, G., Romano, L. & K, Mahata, S., De Lorenzo, A., Marchetti, M., 2020. The effect of ketogenic and low carb diet (Cyclic Alternation) on the ovarian morphology and insulin metabolism in polycystic ovary syndrome patients. Clin. Obstet. Gynecol. Reprod. Med. 6. https://doi.org/10.15761/COGRM.1000307.

Bakrania, K., Edwardson, C.L., Bodicoat, D.H., Esliger, D.W., Gill, J.M.R., Kazi, A., Velayudhan, L., Sinclair, A.J., Sattar, N., Biddle, S.J.H., Khunti, K., Davies, M., Yates, T., 2016. Associations of mutually exclusive categories of physical activity and sedentary time with markers of cardiometabolic health in English adults: a cross-sectional analysis of the Health Survey for England. BMC Publ. Health 16. https://doi.org/10.1186/S12889-016-2694-9.

Banerjee, A., Mukherjee, S., Maji, B.K., 2021. Worldwide flavor enhancer monosodium glutamate combined with high lipid diet provokes metabolic alterations and systemic anomalies: an overview. Toxicol Rep 8, 938–961. https://doi.org/10.1016/J.TOXREP.2021.04.009.

Barber, T.M., Franks, S., 2021. Obesity and polycystic ovary syndrome. Clin. Endocrinol. 95, 531–541. https://doi.org/10.1111/cen.14421.

Barber, T.M., McCarthy, M.I., Wass, J.A.H., Franks, S., 2006. Obesity and polycystic ovary syndrome. Clin. Endocrinol. 65, 137–145. https://doi.org/10.1111/j.1365-2265.2006.02587.x.

Bayraktar, B., Taner, C.E., 2023. The effect of polycystic ovary syndrome history on neonatal anogenital distance: a prospective study in Turkish population. J. Heal. Sci. Med. 6, 30–34. https://doi.org/10.32322/jhsm.1189938.

Blauw, L.L., Aziz, N.A., Tannemaat, M.R., Blauw, C.A., de Craen, A.J., Pijl, H., Rensen, P.C.N., 2017. Diabetes incidence and glucose intolerance prevalence increase with higher outdoor temperature. BMJ Open Diabetes Res. Care 5, e000317. https://doi.org/10.1136/bmjdrc-2016-000317.

Bolla, A.M., Caretto, A., Laurenzi, A., Scavini, M., Piemonti, L., 2019. Low-Carb and ketogenic diets in type 1 and type 2 diabetes. 2019. Nutrition 11, 962. https://doi.org/10.3390/NU11050962. Page 962 11.

Brial, F., Matsuda, F., Gauguier, D., 2022. Diet dependent impact of benzoate on diabetes and obesity in mice. Biochimie 194, 35–42. https://doi.org/10.1016/J.BIOCHI.2021.12.010.
Buoite Stella, A., Ajčević, M., Furlanis, G., Cillotto, T., Menichelli, A., Accardo, A., Manganotti, P., 2021. Smart technology for physical activity and health assessment during COVID-19 lockdown. J. Sports Med. Phys. Fit. 61. https://doi.org/10.23736/S0022-4707.20.11373-2.
Chapman, G., Maclean, H., 1993. Junk food" and "healthy food": meanings of food in adolescent women's culture. J. Nutr. Educ. 25, 108–113. https://doi.org/10.1016/S0022-3182(12)80566-8.
Chiang, Y.F., Chen, H.Y., Ali, M., Shieh, T.M., Huang, Y.J., Wang, K.L., Chang, H.Y., Huang, T.C., Hong, Y.H., Hsia, S.M., 2021. The role of cell proliferation and extracellular matrix accumulation induced by food additive butylated hydroxytoluene in uterine leiomyoma. 2021. Nutrition 13, 3074. https://doi.org/10.3390/NU13093074. Page 3074 13.
Corrie, L., Singh, S.K., Gulati, M., Awasthi, A., Khursheed, R., Kaur, J., Vishwas, S., 2023. Improvement in polycystic ovarian syndrome using pharmacological and non-pharmacological approaches: current perspectives and principles. Curr. Women s Heal. Rev. 19. https://doi.org/10.2174/1573404818666220329123334.
Crespo, R.P., Bachega, T.A.S.S., Mendonça, B.B., Gomes, L.G., 2018. An update of genetic basis of PCOS pathogenesis. Arch. Endocrinol. Metab. 62, 352–361. https://doi.org/10.20945/2359-3997000000049.
Daiber, A., Chlopicki, S., 2020. Revisiting pharmacology of oxidative stress and endothelial dysfunction in cardiovascular disease: evidence for redox-based therapies. Free Radic. Biol. Med. 157, 15–37. https://doi.org/10.1016/J.FREERADBIOMED.2020.02.026.
Demir Çaltekin, M., Hamamcı, M., Onat, T., Aydoğan Kırmızı, D., Başer, E., Yalvaç, E.S., 2021. Evaluation of sleep quality, restless legs syndrome, anxiety and depression in polycystic ovary syndrome. J. TURKISH SLEEP Med. UYKU TIBBI Derg. 8, 243–249. https://doi.org/10.4274/JTSM.GALENOS.2021.85057.
Demos, K.E., Heatherton, T.F., Kelley, W.M., 2012. Individual differences in nucleus accumbens activity to food and sexual images predict weight gain and sexual behavior. J. Neurosci. 32, 5549–5552. https://doi.org/10.1523/JNEUROSCI.5958-11.2012.
Douchi, T., Kuwahata, R., Yamamoto, S., Oki, T., Yamasaki, H., Nagata, Y., 2002. Relationship of upper body obesity to menstrual disorders. Acta Obstet. Gynecol. Scand. 81, 147–150. https://doi.org/10.1034/j.1600-0412.2002.810210.x.
Douglas, C.C., Norris, L.E., Oster, R.A., Darnell, B.E., Azziz, R., Gower, B.A., 2006. Difference in dietary intake between women with polycystic ovary syndrome and healthy controls. Fertil. Steril. 86, 411–417. https://doi.org/10.1016/J.FERTNSTERT.2005.12.054.
Engin, A.B., Engin, A., 2021. The effect of environmental Bisphenol A exposure on breast cancer associated with obesity. Environ. Toxicol. Pharmacol. 81, 103544. https://doi.org/10.1016/J.ETAP.2020.103544.
Ertz, M., Le Bouhart, G., 2022. The other pandemic: a conceptual framework and future research directions of junk food marketing to children and childhood obesity. J. Macromarketing 42, 30–50. https://doi.org/10.1177/02761467211054354.
Felter, S.P., Zhang, X., Thompson, C., 2021. Butylated hydroxyanisole: carcinogenic food additive to be avoided or harmless antioxidant important to protect food supply? Regul. Toxicol. Pharmacol. 121, 104887. https://doi.org/10.1016/J.YRTPH.2021.104887.
Figueiró, T.H., Arins, G.C.B., Santos, C.E.S. dos, Cembranel, F., Medeiros, P.A. de, D'Orsi, E., Rech, C.R., 2019. Association of objectively measured sedentary behavior and physical activity with cardiometabolic risk markers in older adults. PLoS One 14, e0210861. https://doi.org/10.1371/journal.pone.0210861.
Fogelkvist, M., Gustafsson, S.A., Kjellin, L., Parling, T., 2020. Acceptance and commitment therapy to reduce eating disorder symptoms and body image problems in patients with residual eating disorder symptoms: a randomized controlled trial. Body Image 32, 155–166. https://doi.org/10.1016/J.BODYIM.2020.01.002.
Gafford, F.H., Sharry, P.M., Pittman, J.A., 1971. Effect of azodicarbonamide (1,1′-azobisformamide) on thyroid function. J. Clin. Endocrinol. Metab. 32, 659–662. https://doi.org/10.1210/JCEM-32-5-659.
Ganga, S., Mathiyoli, P.M., Naachimuthu, K.P., 2020. Darkside of the white flour – maida. Indian J. Heal. Wellbeing 11. https://doi.org/10.15614/IJHW.V11I01.20.
Goldfine, A.B., Kahn, C.R., 2003. Adiponectin: linking the fat cell to insulin sensitivity. Lancet 362, 1431–1432. https://doi.org/10.1016/S0140-6736(03)14727-7.
Goss, A.M., Gower, B., Soleymani, T., Stewart, M., Pendergrass, M., Lockhart, M., Krantz, O., Dowla, S., Bush, N., Garr Barry, V., Fontaine, K.R., 2020. Effects of weight loss during a very low carbohydrate diet on specific adipose tissue depots and insulin sensitivity in older adults with obesity: a randomized clinical trial. Nutr. Metab. 17, 64. https://doi.org/10.1186/s12986-020-00481-9.
Greathouse, K.L., Faucher, M.A., Hastings-Tolsma, M., 2017. The gut microbiome, obesity, and weight control in women's reproductive health. West. J. Nurs. Res. 39, 1094–1119. https://doi.org/10.1177/0193945917697223.
Grillo, C.A., Piroli, G.G., Lawrence, R.C., Wrighten, S.A., Green, A.J., Wilson, S.P., Sakai, R.R., Kelly, S.J., Wilson, M.A., Mott, D.D., Reagan, L.P., 2015. Hippocampal insulin resistance impairs spatial learning and synaptic plasticity. Diabetes 64, 3927–3936. https://doi.org/10.2337/db15-0596.
Hachul, H., Polesel, D.N., Tock, L., Carneiro, G., Pereira, A.Z., Zanella, M.T., Tufik, S., Togeiro, S.M., 2019. Sleep disorders in polycystic ovary syndrome: influence of obesity and hyperandrogenism. Rev. Assoc. Med. Bras. 65, 375–383. https://doi.org/10.1590/1806-9282.65.3.375.
Hashem, M.M., Abd-Elhakim, Y.M., Abo-El-Sooud, K., Eleiwa, M.M.E., 2019. Embryotoxic and teratogenic effects of tartrazine in rats. Toxicol. Res. 35, 75–81. https://doi.org/10.5487/TR.2019.35.1.075.
Hassanzadeh, J., Mohammadbeigi, A., Eshrati, B., Moemenbellah-Fard, M.J., 2012. Estimation of the regional burden of non-communicable diseases due to obesity and overweight in Markazi province, Iran, 2006–2007. J. Cardiovasc. Dis. Res. 3, 26–31. https://doi.org/10.4103/0975-3583.91600.
Hawley, J.A., Sassone-Corsi, P., Zierath, J.R., 2020. Chrono-nutrition for the prevention and treatment of obesity and type 2 diabetes: from mice to men. Diabetologia 63, 2253–2259. https://doi.org/10.1007/s00125-020-05238-w.
Hirschberg, A.L., 2012. Sex hormones, appetite and eating behaviour in women. Maturitas 71, 248–256. https://doi.org/10.1016/j.maturitas.2011.12.016.
Hudson, J.I., Lalonde, J.K., Coit, C.E., Tsuang, M.T., McElroy, S.L., Crow, S.J., Bulik, C.M., Hudson, M.S., Yanovski, J.A., Rosenthal, N.R., Pope, H.G., 2010. Longitudinal study of the diagnosis of components of the metabolic syndrome in individuals with binge-eating disorder. Am. J. Clin. Nutr. 91, 1568–1573. https://doi.org/10.3945/ajcn.2010.29203.
Jaacks, L.M., Vandevijvere, S., Pan, A., McGowan, C.J., Wallace, C., Imamura, F., Mozaffarian, D., Swinburn, B., Ezzati, M., 2019. The obesity transition: stages of the global epidemic. Lancet Diabetes Endocrinol. 7, 231–240. https://doi.org/10.1016/S2213-8587(19)30026-9.
Kenđel Jovanović, G., Mrakovcic-Sutic, I., Pavičić Žeželj, S., Benjak Horvat, I., Šuša, L., Rahelić, D., Klobučar Majanović, S., 2021. Metabolic and hepatic effects of energy-reduced anti-inflammatory diet in younger adults with obesity. Chin. J. Gastroenterol. Hepatol. 2021, 1–12. https://doi.org/10.1155/2021/6649142.
Koivuaho, E., Laru, J., Ojaniemi, M., Puukka, K., Kettunen, J., Tapanainen, J.S., Franks, S., Järvelin, M.-R., Morin-Papunen, L., Sebert, S., Piltonen, T.T., 2019. Age at adiposity rebound in childhood is associated with PCOS diagnosis and obesity in adulthood—longitudinal analysis of BMI data from birth to age 46 in cases of PCOS. Int. J. Obes. 43, 1370–1379. https://doi.org/10.1038/s41366-019-0318-z.
Koster, M.P.H., de Wilde, M.A., Veltman-Verhulst, S.M., Houben, M.L., Nikkels, P.G.J., van Rijn, B.B., Fauser, B.C.J.M., 2015. Placental characteristics in women with polycystic ovary syndrome. Hum. Reprod. dev 265. https://doi.org/10.1093/humrep/dev265.
Kumar, S., Gordon, G.H., Abbott, D.H., Mishra, J.S., 2018. Androgens in maternal vascular and placental function: implications for preeclampsia pathogenesis. Reproduction R155–R167. https://doi.org/10.1530/REP-18-0278.
Kurylowicz, A., 2021. Endocrine disorders accompanying obesity - effect or cause? In: Role of Obesity in Human Health and Disease. IntechOpen. https://doi.org/10.5772/intechopen.98793.
Kwon, Y., 2022. Potential pro-tumorigenic effect of bisphenol A in breast cancer via altering the tumor microenvironment. 2022. Cancers 14, 3021. https://doi.org/10.3390/CANCERS14123021. Page 3021 14.
Łakoma, K., Kukharuk, O., Śliż, D., 2023. The influence of metabolic factors and diet on fertility. Nutrients 15, 1180. https://doi.org/10.3390/nu15051180.
Latif, S., Naz, S., Ashraf, S., Ahmed Jafri, S., 2022. Junk food consumption in relation to menstrual abnormalities among adolescent girls: a comparative cross sectional study. Pakistan J. Med. Sci. 38. https://doi.org/10.12669/pjms.38.8.6177.
Li, X., Sotres-Alvarez, D., Gallo, L.C., Ramos, A.R., Aviles-Santa, L., Perreira, K.M., Isasi, C.R., Zee, P.C., Savin, K.L., Schneiderman, N., Wassertheil-Smoller, S., Sofer, T., Daviglus, M., Redline, S., 2021. Associations of sleep-disordered breathing and insomnia with incident hypertension and diabetes: the hispanic community health study/study of latinos. Am. J. Respir. Crit. Care Med. 203, 356–365. https://doi.org/10.1164/RCCM.201912-2330OC/SUPPL_FILE/DISCLOSURES.PDF.
Lidaka, L., Lazdane, G., Kivite-Urtane, A., Gailite, L., Dzivite-Krisane, I., Stokenberga, I., 2022. Health-related quality of life and binge eating among adolescent girls with PCOS. Clin. Exp. Obstet. Gynecol. 49, 57. https://doi.org/10.31083/j.ceog4903057.
Ludwig, D.S., 2020. The ketogenic diet: evidence for optimism but high-quality research needed. J. Nutr. 150, 1354–1359. https://doi.org/10.1093/jn/nxz308.

Magni, P., Dozio, E., Ruscica, M., Celotti, F., Masini, M.A., Prato, P., Broccoli, M., Mambro, A., Morè, M., Strollo, F., 2009. Feeding behavior in mammals including humans. Ann. N. Y. Acad. Sci. 1163, 221–232. https://doi.org/10.1111/j.1749-6632.2008.03627.x.
Malik, V.S., Schulze, M.B., Hu, F.B., 2006. Intake of sugar-sweetened beverages and weight gain: a systematic review1–3. Am. J. Clin. Nutr. 84, 274–288. https://doi.org/10.1093/ajcn/84.1.274.
Maranghi, F., Tassinari, R., Marcoccia, D., Altieri, I., Catone, T., De Angelis, G., Testai, E., Mastrangelo, S., Evandri, M.G., Bolle, P., Lorenzetti, S., 2010. The food contaminant semicarbazide acts as an endocrine disrupter: evidence from an integrated in vivo/in vitro approach. Chem. Biol. Interact. 183, 40–48. https://doi.org/10.1016/J.CBI.2009.09.016.
Marinangeli, C.P.F., Jones, P.J.H., 2012. Pulse grain consumption and obesity: effects on energy expenditure, substrate oxidation, body composition, fat deposition and satiety. Br. J. Nutr. 108, S46–S51. https://doi.org/10.1017/S0007114512000773.
Martinez-Avila, W.D., Sanchez-Delgado, G., Acosta, F.M., Jurado-Fasoli, L., Oustric, P., Labayen, I., Blundell, J.E., Ruiz, J.R., 2020. Eating behavior, physical activity and exercise training: a randomized controlled trial in young healthy adults. Nutrients 12, 1–14. https://doi.org/10.3390/NU12123685.
Mattila, M., Niinistö, S., Takkinen, H.M., Tapanainen, H., Reinivuo, H., Åkerlund, M., Suomi, J., Ahonen, S., Ilonen, J., Toppari, J., Knip, M., Veijola, R., Virtanen, S.M., 2020. Maternal nitrate and nitrite intakes during pregnancy and risk of islet autoimmunity and type 1 diabetes: the DIPP cohort study. J. Nutr. 150, 2969–2976. https://doi.org/10.1093/JN/NXAA250.
McCuen-Wurst, C., Ruggieri, M., Allison, K.C., 2018. Disordered eating and obesity: associations between binge-eating disorder, night-eating syndrome, and weight-related comorbidities. Ann. N. Y. Acad. Sci. 1411, 96–105. https://doi.org/10.1111/nyas.13467.
McPherson, P.A.C., McEneny, J., 2012. The biochemistry of ketogenesis and its role in weight management, neurological disease and oxidative stress. J. Physiol. Biochem. 68, 141–151. https://doi.org/10.1007/S13105-011-0112-4.
Mindang, E.L.N., Awounfack, C.F., Ndinteh, D.T., Krause, R.W.M., Njamen, D., 2022. Effects of tartrazine on some sexual maturation parameters in immature female wistar rats. Int. J. Environ. Res. Publ. Health 19, 10410. https://doi.org/10.3390/ijerph191610410.
Mirabelli, M., Chiefari, E., Arcidiacono, B., Corigliano, D.M., Brunetti, F.S., Maggisano, V., Russo, D., Foti, D.P., Brunetti, A., 2020. Mediterranean diet nutrients to turn the tide against insulin resistance and related diseases. Nutrients 12, 1066. https://doi.org/10.3390/nu12041066.
Mizgier, M., Jarząbek-Bielecka, G., Opydo-Szymaczek, J., Wendland, N., Więckowska, B., Kędzia, W., 2020. Risk factors of overweight and obesity related to diet and disordered eating attitudes in adolescent girls with clinical features of polycystic ovary syndrome. J. Clin. Med. 9, 3041. https://doi.org/10.3390/jcm9093041.
Mohammadbeigi, A., Asgarian, A., Ahmadli, R., Fara-Shirazi, S.Z., Moshiri, E., Ansari, H., Khazaei, S., Afrashteh, S., 2019. Prevalence of junk food consumption, overweight/obesity and self-rated health and fitness in high school adolescent girls: a cross sectional study in a deprived area of Qom. Sri Lanka J. Child Heal. 48, 208–214. https://doi.org/10.4038/sljch.v48i3.8754.
Mohammadbeigi, A., Asgarian, A., Moshir, E., Heidari, H., Afrashteh, S., Khazaei, S., Ansari, H., 2018. Fast food consumption and overweight/obesity prevalence in students and its association with general and abdominal obesity. J. Prev. Med. Hyg. 59, E236–E240. https://doi.org/10.15167/2421-4248/jpmh2018.59.3.830.
Mondal, M., Sarkar, K., Nath, P.P., Paul, G., 2018. Monosodium glutamate suppresses the female reproductive function by impairing the functions of ovary and uterus in rat. Environ. Toxicol. 33, 198–208. https://doi.org/10.1002/TOX.22508.
Muscogiuri, G., Barrea, L., Aprano, S., Framondi, L., Di Matteo, R., Laudisio, D., Pugliese, G., Savastano, S., Colao, A., 2020. Chronotype and adherence to the mediterranean diet in obesity: results from the opera prevention project. 2020. Nutrition 12, 1354. https://doi.org/10.3390/NU12051354. Page 1354 12.
Nixon, H., Doud, L., 2011. Do fast food restaurants cluster around high schools? A geospatial analysis of proximity of fast food restaurants to high schools and the connection to childhood obesity rates. J. Agric. Food Syst. Community Dev. 181–194. https://doi.org/10.5304/jafscd.2011.021.007.
Norris, T., Hawton, K., Hamilton-Shield, J., Crawley, E., 2017. Obesity in adolescents with chronic fatigue syndrome: an observational study. Arch. Dis. Child. 102, 35–39. https://doi.org/10.1136/ARCHDISCHILD-2016-311293.
Oghbaei, H., Hamidian, G., Alipour, M.R., Alipour, S., Keyhanmanesh, R., 2020. The effect of prolonged dietary sodium nitrate treatment on the hypothalamus-pituitary-gonadal axis and testicular structure and function in streptozotocin-induced diabetic male rats. Food Funct. 11, 2451–2465. https://doi.org/10.1039/C9FO00974D.
Oginsky, M.F., Ferrario, C.R., 2019. Eating "junk food" has opposite effects on intrinsic excitability of nucleus accumbens core neurons in obesity-susceptible versus -resistant rats. J. Neurophysiol. 122, 1264–1273. https://doi.org/10.1152/jn.00361.2019.
Panjeshahin, A., Salehi-Abargouei, A., Anari, A.G., Mohammadi, M., Hosseinzadeh, M., 2020. Association between empirically derived dietary patterns and polycystic ovary syndrome: a case-control study. Nutrition 79–80, 110987. https://doi.org/10.1016/j.nut.2020.110987.
Pereira, M.A., Kartashov, A.I., Ebbeling, C.B., Van Horn, L., Slattery, M.L., Jacobs, D.R., Ludwig, D.S., 2005. Fast-food habits, weight gain, and insulin resistance (the CARDIA study): 15-year prospective analysis. Lancet 365, 36–42. https://doi.org/10.1016/S0140-6736(04)17663-0.
Perry, C.A., Van Guilder, G.P., Kauffman, A., Hossain, M., 2019. A calorie-restricted DASH diet reduces body fat and maintains muscle strength in obese older adults. 2020. Nutrition 12, 102. https://doi.org/10.3390/NU12010102. Page 102 12.
Phillips, C.M., Kesse-Guyot, E., McManus, R., Hercberg, S., Lairon, D., Planells, R., Roche, H.M., 2012. High dietary saturated fat intake accentuates obesity risk associated with the fat mass and obesity–associated gene in adults. J. Nutr. 142, 824–831. https://doi.org/10.3945/jn.111.153460.
Powell-Wiley, T.M., Poirier, P., Burke, L.E., Després, J.-P., Gordon-Larsen, P., Lavie, C.J., Lear, S.A., Ndumele, C.E., Neeland, I.J., Sanders, P., St-Onge, M.-P., 2021. Obesity and cardiovascular disease: a scientific statement from the American heart association. Circulation 143. https://doi.org/10.1161/CIR.0000000000000973.
Powell, K.E., King, A.C., Buchner, D.M., Campbell, W.W., DiPietro, L., Erickson, K.I., Hillman, C.H., Jakicic, J.M., Janz, K.F., Katzmarzyk, P.T., Kraus, W.E., Macko, R.F., Marquez, D.X., McTiernan, A., Pate, R.R., Pescatello, L.S., Whitt-Glover, M.C., 2019. The scientific foundation for the physical activity guidelines for Americans, 2nd edition. J. Phys. Activ. Health 16, 1–11. https://doi.org/10.1123/jpah.2018-0618.
Pramodh, S., 2020. Exploration of lifestyle choices, reproductive health knowledge, and polycystic ovary syndrome (PCOS) awareness among female Emirati university students. Int. J. Womens. Health 12, 927–938. https://doi.org/10.2147/IJWH.S272867.
Qu, X., Donnelly, R., 2020. Sex hormone-binding globulin (SHBG) as an early biomarker and therapeutic target in polycystic ovary syndrome. Int. J. Mol. Sci. 21, 8191. https://doi.org/10.3390/ijms21218191.
Ramona, F., Sara, G., Carlo, P., 2021. Risk of developing eating disorders through the misperception of the body image and the adoption of bad eating habits in a sample of young volleyball athletes. Arch. Food Nutr. Sci. 5, 7–17. https://doi.org/10.29328/journal.afns.1001027.
Rangwala, S.M., Rich, A.S., Rhoades, B., Shapiro, J.S., Obici, S., Rossetti, L., Lazar, M.A., 2004. Abnormal glucose homeostasis due to chronic hyperresistinemia. Diabetes 53, 1937–1941. https://doi.org/10.2337/diabetes.53.8.1937.
Ringel, N.E., Hovey, K.M., Andrews, C.A., Mossavar-Rahmani, Y., Shadyab, A.H., Snetselaar, L.G., Howard, B.V., Iglesia, C.B., 2022. Artificially Sweetened Beverages and Urinary Incontinence—A Secondary Analysis of the Women's Health Initiative Observational Study. Menopause Publish Ah. https://doi.org/10.1097/GME.0000000000002129.
Robinson, M.J., Burghardt, P.R., Patterson, C.M., Nobile, C.W., Akil, H., Watson, S.J., Berridge, K.C., Ferrario, C.R., 2015. Individual differences in cue-induced motivation and striatal systems in rats susceptible to diet-induced obesity. Neuropsychopharmacology 40, 2113–2123. https://doi.org/10.1038/npp.2015.71.
Römsing, S., Ulfberg, J., Bergqvist, Y., 2003. Determination of melatonin in human plasma with solid-phase extraction, high-performance liquid chromatography and fluorescence detection. Scand. J. Clin. Lab. Invest. 63, 81–88. https://doi.org/10.1080/00365510310000529.
Rosenbaum, M., Knight, R., Leibel, R.L., 2015. The gut microbiota in human energy homeostasis and obesity. Trends Endocrinol. Metabol. 26, 493–501. https://doi.org/10.1016/j.tem.2015.07.002.
Rothschild, D., Weissbrod, O., Barkan, E., Kurilshikov, A., Korem, T., Zeevi, D., Costea, P.I., Godneva, A., Kalka, I.N., Bar, N., Shilo, S., Lador, D., Vila, A.V., Zmora, N., Pevsner-Fischer, M., Israeli, D., Kosower, N., Malka, G., Wolf, B.C., Avnit-Sagi, T., Lotan-Pompan, M., Weinberger, A., Halpern, Z., Carmi, S., Fu, J., Wijmenga, C., Zhernakova, A., Elinav, E., Segal, E., 2018. Environment dominates over host genetics in shaping human gut microbiota. Nature 555, 210–215. https://doi.org/10.1038/nature25973.
Sanchez-Garrido, M.A., Tena-Sempere, M., 2020. Metabolic dysfunction in polycystic ovary syndrome: pathogenic role of androgen excess and potential therapeutic strategies. Mol. Metabol. 35, 100937. https://doi.org/10.1016/j.molmet.2020.01.001.
Schéle, E., Grahnemo, L., Anesten, F., Hallén, A., Bäckhed, F., Jansson, J.-O., 2016. Regulation of body fat mass by the gut microbiota: possible mediation by the brain. Peptides 77, 54–59. https://doi.org/10.1016/j.peptides.2015.03.027.

Schulze, M.B., Manson, J.A.E., Ludwig, D.S., Colditz, G.A., Stampfer, M.J., Willett, W.C., Hu, F.B., 2004. Sugar-Sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women. JAMA 292, 927–934. https://doi.org/10.1001/JAMA.292.8.927.
Schwartz, M.W., Seeley, R.J., Zeltser, L.M., Drewnowski, A., Ravussin, E., Redman, L.M., Leibel, R.L., 2017. Obesity pathogenesis: an endocrine society scientific statement. Endocr. Rev. 38, 267–296. https://doi.org/10.1210/er.2017-00111.
Shah, T., 2014. Assessment of obesity, overweight and its association with the fast food consumption in medical students. J. Clin. DIAGNOSTIC Res. https://doi.org/10.7860/JCDR/2014/7908.4351.
Shan, H., Luo, R., Guo, X., Li, R., Ye, Z., Peng, T., Liu, F., Yang, Z., 2022. Abnormal endometrial receptivity and oxidative stress in polycystic ovary syndrome. Front. Pharmacol. 13. https://doi.org/10.3389/FPHAR.2022.904942/FULL.
Shankar, B., Mazzocchi, M., Traill, W.B., 2014. Macroeconomic causes and effects of noncommunicable disease: the case of diet and obesity. In: Encyclopedia of Health Economics. Elsevier, pp. 160–164. https://doi.org/10.1016/B978-0-12-375678-7.00611-8.
Sharma, P., Kaur, M., Kumar, S., Khetarpal, P., 2022. A cross-sectional study on prevalence of menstrual problems, lifestyle, mental health, and PCOS awareness among rural and urban population of Punjab, India. J. Psychosom. Obstet. Gynecol. 43, 349–358. https://doi.org/10.1080/0167482X.2021.1965983.
Shishehgar, F., Mirmiran, P., Rahmati, M., Tohidi, M., Ramezani Tehrani, F., 2019. Does a restricted energy low glycemic index diet have a different effect on overweight women with or without polycystic ovary syndrome? BMC Endocr. Disord. 19, 1–11. https://doi.org/10.1186/S12902-019-0420-1/FIGURES/2.
Singh, M., Rajoura, O., Honnakamble, R., 2019. Menstrual patterns and problems in association with body mass index among adolescent school girls. J. Fam. Med. Prim. Care 8, 2855. https://doi.org/10.4103/jfmpc.jfmpc_474_19.
Singh S, A., Dhanasekaran, D., Ganamurali, N., L, P., Sabarathinam, S., 2021. Junk food-induced obesity- a growing threat to youngsters during the pandemic. Obes. Med. 26, 100364. https://doi.org/10.1016/j.obmed.2021.100364.
Skiba, M.A., Bell, R.J., Islam, R.M., Handelsman, D.J., Desai, R., Davis, S.R., 2019. Androgens during the reproductive years: what is normal for women? J. Clin. Endocrinol. Metab. 104, 5382–5392. https://doi.org/10.1210/jc.2019-01357.
Subramaniam, K., Tripathi, A., Dabadghao, P., 2019. Familial clustering of metabolic phenotype in brothers of women with polycystic ovary syndrome. Gynecol. Endocrinol. 35, 601–603. https://doi.org/10.1080/09513590.2019.1566451.
Tay, C.T., Moran, L.J., Harrison, C.L., Brown, W.J., Joham, A.E., 2020. Physical activity and sedentary behaviour in women with and without polycystic ovary syndrome: an Australian population-based cross-sectional study. Clin. Endocrinol. 93, 154–162. https://doi.org/10.1111/cen.14205.
Venkatesh, S.S., Ferreira, T., Benonisdottir, S., Rahmioglu, N., Becker, C.M., Granne, I., Zondervan, K.T., Holmes, M.V., Lindgren, C.M., Wittemans, L.B.L., 2022. Correction: obesity and risk of female reproductive conditions: a Mendelian randomisation study. PLoS Med. 19, e1004095. https://doi.org/10.1371/journal.pmed.1004095.
Venn, B.J., Perry, T., Green, T.J., Skeaff, C.M., Aitken, W., Moore, N.J., Mann, J.I., Wallace, A.J., Monro, J., Bradshaw, A., Brown, R.C., Skidmore, P.M.L., Doel, K., O' Brien, K., Frampton, C., Williams, S., 2013. The Effect of Increasing Consumption of Pulses and Wholegrains in Obese People: A Randomized Controlled Trial, pp. 365–372. https://doi.org/10.1080/07315724.2010.10719853 29. 10.1080/07315724.2010.10719853.
Vgontzas, A.N., Calhoun, S., 2009. Polycystic ovary syndrome, sleep apnea, and daytime sleepiness and fatigue. Diagnosis Manag. Polycystic Ovary Syndr 143–154. https://doi.org/10.1007/978-0-387-09718-3_12/COVER.
Vidya Bharathi, R., Swetha, S., Neerajaa, J., Varsha Madhavica, J., Janani, D.M., Rekha, S.N., S., R., B., U., 2017. An epidemiological survey: effect of predisposing factors for PCOS in Indian urban and rural population. Middle East Fertil. Soc. J. 22, 313–316. https://doi.org/10.1016/j.mefs.2017.05.007.
Wadden, T.A., Tronieri, J.S., Butryn, M.L., 2020. Lifestyle modification approaches for the treatment of obesity in adults. Am. Psychol. 75, 235–251. https://doi.org/10.1037/amp0000517.
Wei, S., Schmidt, M.D., Dwyer, T., Norman, R.J., Venn, A.J., 2009. Obesity and menstrual irregularity: associations with SHBG, testosterone, and insulin. Obesity 17, 1070–1076. https://doi.org/10.1038/oby.2008.641.
Westerterp, K.R., 2004. Diet induced thermogenesis. Nutr. Metab. 1. https://doi.org/10.1186/1743-7075-1-5.
Woo, S., Yang, H., Kim, Y., Lim, H., Song, H.J., Park, K.H., 2022. Sedentary time and fast-food consumption associated with weight gain during COVID-19 lockdown in children and adolescents with overweight or obesity. J. Kor. Med. Sci. 37. https://doi.org/10.3346/jkms.2022.37.e103.
Ye, J., 2021. Mechanism of insulin resistance in obesity: a role of ATP. Front. Med. 15, 372–382. https://doi.org/10.1007/s11684-021-0862-5.
Yukie, M., Aoi, I., Mizuki, K., Toshiyuki, Y., 2020. Change in appetite and food craving during menstrual cycle in young students. Int. J. Nutr. Metab. 12, 25–30. https://doi.org/10.5897/IJNAM2019.0264.
Zhang, H.-L., Yi, M., Li, D., Li, R., Zhao, Y., Qiao, J., 2020. Transgenerational inheritance of reproductive and metabolic phenotypes in PCOS rats. Front. Endocrinol. 11, 601–603. https://doi.org/10.3389/fendo.2020.00144.
Zhou, S., Lu, D., Wen, S., Sheng, Y., Kang, D., Xu, L., 2022. Elevated anti-müllerian hormone levels in newborns of women with polycystic ovary syndrome: a systematic review and meta-analysis based on observational studies. Reprod. Sci. 29, 301–311. https://doi.org/10.1007/s43032-021-00652-w.