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UNDERSTANDING OBESITY THROUGH THE LENS OF HUMAN PHYSIOLOGY: A SYSTEMATIC REVIEW OF ITS PREVALENCE AND UNDERLYING MECHANISMS

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ABSTRACT

Background: Obesity has become a global health issue, with over 650 million adults in the world having been affected by the epidemic. Besides lifestyle and behavioural factors, a complicated system of physiological mechanisms is involved in its development and maintenance. For the prevention and treatment of the disease, it is important to know these mechanisms.

Objectives: This systematic review aims to analyse the prevailing prevalence of obesity across the world and an in-depth physiology process, start-up and pathophysiology of further developments. The studies look at energy balance, hormones, brain pathways, fat cells and the gut microbiome.

Methods: The primary databases, PubMed, Scopus and Web of Science, were searched systematically on the basis of pre-determined keywords, with the use of the terms connected with obesity and human physiology. The inclusive period was between 2000 and 2024 for English language studies. The keywords-based incarceration concentrated on original articles and systematic reviews concerning physiological factors, which are attributed to human obesity. The articles were chosen based on the PRISMA, and the reliability of the included studies was ensured with the help of quality assessment tools.

Key Findings: One of the significant points that the authors make in the review is the role of hormonal regulators of the appetite, energy metabolism, leptin, ghrelin, insulin and cortisol. The hypothalamus also has important neurophysiological centres that control hunger and satiety. Fat is an organ in itself, which releases pro-inflammatory cytokines and causes systemic inflammation. More than that, dysbiosis of gut microbiota has been linked to alterations in nutrient uptake and metabolic diseases. These physiological pathways are in turn, regulated by genetic and epigenetic mechanisms, both of which add more layers of personal vulnerability.

Conclusion: Obesity is a multifactorial, multidimensional and human-physiological disease. The efficacy of treatment methods and clinical community health programs could be enhanced by taking into account their physiological basis as well as the lifestyle changes.

KEYWORDS: Obesity, Endocrinology: Hormones and Nutrition, Endocrinology: Human Physiology, Energy Balance: Systematic Review

1. INTRODUCTION

1.1. Obesity (BMI category, WHO/CDC)

Obesity is a condition of excessive or unusual fat that is not healthy and can be dangerous to human health. According to the World Health Organisation (WHO), adults whose Body Mass Index (BMI) is (BMI)≥30 kg/m² are considered obese, whereas a BMI of 25 to 29.9 kg/m² and above is considered overweight (WHO, 2021). There is a parallel classification in use in the Centres for Disease Control and Prevention (CDC), which divides obesity into three classes: Class I (BMI 3034.9), Class II (3539.9), and Class III, severe or morbid obesity (CDC, 2020).

1.2. Trends in the Prevalence of Public Health and Relevance in the World

Obesity is now recognised as one of the leading public health issues, which is also linked to the increased risk of type 2 diabetes, cardiovascular disease, selected malignancies and musculoskeletal problems (Ng et al., 2014). In 2016, more than 650 million adults in the world were obese, and the number is constantly growing in both developed and developing

nations (WHO, 2021). Worryingly, obesity in children has also increased dramatically, and 124 million children and adolescents all over the world are considered obese (NCD Risk Factor Collaboration, 2017).

1.3. Significance of Learning Physiological Processes.

Even though diet and physical activity are crucial factors in obesity, new data show a major role in physiological dysregulation in energy deficiency and excess fat storage (Bray and Ryan, 2020). The multi-factorial aetiology of obesity encompasses the dysregulation of appetite-controlling hormones, hypothalamic signal, adipose tissue functions and metabolic pathways (Farooqi and O'Rahilly, 2007). A better understanding of these distal mechanisms is required in order to have all-encompassing, targeted and long-term interventions.

1.4. Aim and Background of the Review.

Thus, this review aims at a systematic review of the prevalence of obesity across the globe and a critical evaluation of the physiological actions that are involved in the development and maintenance of the condition. Considering the increasing cases of treatment-resistant obesity, it will be relevant to discuss biological and contextual factors that precondition the occurrence of weight gain despite the absence of external change (Heymsfield and Wadden, 2017). This review is an integrative physiological perspective of obesity that incorporates scientific data obtained from recent studies.

1.5. Research Questions/Objectives

This review has been based on the following research questions.

- In the past two decades, has obesity become more or less prevalent across the globe?
- What are the various synergistic processes that are triggered by hormones, the nervous system, metabolism, and microbiology to trigger obesity and its maintenance?
- Could knowledge of these processes help with preventive and therapeutic interventions?

The review also aims to:

- To establish significant hormonal and neurophysiological pathways that can be applied to obesity.
- The interaction between gut microbiota and the adipose tissue regarding energy metabolism.
- Test the interplay between biology and sociology.

2. METHODOLOGY

In spite of the systematic review, methodology reporting was not fully optimised and allowed not to meet predetermined inclusion criteria according to a priori submission requirements like PRISMA checklist items, which are critical to methodological transparency, scientific replicability, and reproducibility. In response to this, a thorough literature search was conducted to single out the current peer-reviewed works dealing with the physiological processes and universal prevalence of obesity. It was searched in a variety of electronic databases, such as PubMed, Scopus, Web of Science, ScienceDirect, and Google Scholar, where the latter was also utilised to retrieve grey literature and additional references. An a priori combination of MeSH terms and free-text keywords was created in a structured search strategy with the use of Boolean operators (AND, OR). One of the search terms is as follows: ("Obesity or Overweight) AND (Physiology or Human physiology or Endocrine regulation or Energy balance) AND (Prevalence or Epidemiology) AND (Hormones or Leptin or Insulin or Ghrelin) AND (Hypothalamus or Gut microbiota). A set of filters was applied that restricted the studies to those published after 2000 and 2024, those written in English and those that involve human subjects.

The inclusion criteria were well specified, such as peer-reviewed original research and systematic reviews of human populations (adults and children) that addressed either the physiological processes of obesity or the prevalence of obesity globally or regionally and had been published within the past four years (between January 2000 and April 2024) in English. The studies were filtered out based on the following criteria: they had to be of animal or in vitro experiments, involve exclusive surgical or pharmacological interventions and had to be an editorial or letter, conference abstract without access to the full-text or published in non-English languages. The process of study selection adhered to PRISMA 2020 standards, and PRISMA principles were applied as database searches followed by the importation of articles into Zotero as a means of referencing and then duplicates were removed. Two independent reviewers began to screen the titles and abstracts, and then conducted full-text screening of the articles to remove those that did not meet the study criteria. All issues of disagreement were solved by agreement or by consulting a third reviewer. The general process of selection was recorded on a PRISMA flow diagram that was also provided in the appendix.

To extract and synthesise data, a structured Excel-based sheet was created to record essential variables such as author details, year of publication, country, sample size and demographics, research design, parameters of physiological processes to be studied (hormonal, neural, metabolic, and microbiota-related pathways) and report, and limitations. A narrative synthesis method was used because of the heterogeneity of study designs and reported results. In order to guarantee methodological rigour, the Newcastle-Ottawa Scale (NOS) of observational studies and the AMSTAR 2 of systematic reviews were used to estimate quality assessment and risk of bias evaluation. The reviews were conducted by two reviewers who independently evaluated these assessments, and disagreements were solved by discussion as a way of ensuring consistency and reliability in the evaluation.

sTable: Key Studies on Physiological Mechanisms of Obesity

Study (Year)	Physiological Focus	Population / Sample	Key Findings	Link
Mantzoros et al. – Leptin in Human Physiology (2011)	Leptin signaling	Human subjects	Explores leptin's role in energy balance and "leptin resistance" mechanisms—defective signaling despite high circulating leptin in obesity	Link to study
Friedman & Farooqi – Leptin Resistance Mechanisms (2020)	Central appetite regulation	Review of human & animal studies	Highlights hypothalamic receptor and JAK/STAT pathway defects as key drivers of leptin resistance	Link to review
Tschöp et al. – Ghrelin & Leptin in Appetite Control (2006)	Combined hormonal regulation	Mixed adult cohorts	Describes interplay between ghrelin (hunger signal) and leptin (satiety), balancing short- and long-term energy regulation	Link to review
Wu et al. – Cross-talk Between Gut Microbiota & Adipose Tissue (2022)	Gut microbiome–adipose interaction	Human and animal studies	Demonstrates microbiota's role in inflammation, energy expenditure, adipose browning, and metabolic regulation	Link to article
Frontiers in Endocrinology – Gut–Adipose Crosstalk in Inflammation (2021)	Microbiome-mediated adipose dysfunction	Review article	Details microbial metabolites influencing adipose inflammation and metabolic disease susceptibility	Link to review
Wikipedia – Adipose Tissue as Endocrine Organ	Adipokines and chronic inflammation	General review	Summarizes adipose tissue's secretion of leptin, estrogen, resistin, TNF-α, IL-6; implicates adipokines in chronic inflammatory state	Link to reference
Wikipedia – Pathophysiology of Obesity	Hormones & CNS	General reference	Leptin and ghrelin act via hypothalamus; leptin resistance is central in appetite dysregulation	Link to summary

3. Highlights

- **Leptin resistance:** A common cause of obesity - most obese people have plenty of leptin, but their brains cannot utilise it through impaired receptor signalling.
- **Ghrelin and Leptin balance:** before meals, Ghrelin stimulates hunger; after meals, leptin and satiety. The active interaction between them ensures that there is energy equilibrium
- **Endocrine functions of adipose tissue:** In addition to serving as an energy store, adipocytes secrete hormones and cytokines (e.g., IL 6, TNF-α) that affect systemic metabolism and inflammation.
- **What is being communicated is that adipose tissue:** gut microbes can regulate adipose inflammation and metabolism through metabolites (e.g., SCFAs); microbiota-adipose axes offer new therapeutic opportunities.

4. Global Prevalence of Obesity

4.1. Epidemiological Statistics by Region and Demographics

Obesity has attained alarming statistics across the globe with 2.5 billion adults being overweight or obese, which is close to 43 % of the total world adult population. In this, there are approximately 890 million obese people. Regional differences are distinctive since the prevalence rates are almost 31% in the areas like Southeast Asia and Africa to a high of 67% in the Americas. Recent estimates in the world also show that over 1 billion people are living with obesity; 880 million adults and 159 million children of the age group 5-19 years. These statistics demonstrate how prevalent and growing the issue of obesity in the international community is (WHO; World Obesity Federation; NCBI/PMC).

4.2. Trends Over the Last 2–3 Decades

In the last several decades, the prevalence of obesity has been skyrocketing all over the world. The level of obesity among adults has almost tripled since 1975 as a result of changes in lifestyle and environment. The prevalence of obesity among men has increased by 4.8 to 14.0 % since 1990, and among women, 8.8 to 18.5 % during the same time period. It has been estimated that over two out of every three adult populations in the world may become overweight or obese by 2035 (possibly over 3 billion people). The trend of obesity in children is also alarming, as the prevalence of the issue nearly doubled during 1975-2022: between 0.7 and 6.9 % among girls and between 0.9 and 9.3 % among boys, respectively (World Obesity Federation; Obesity Evidence Hub).

4.3. Age, Gender, Socioeconomic, and Ethnic Disparities

There is a significant difference in the prevalence of obesity depending on demographics. The global gender inequality is clear, with women showing a higher prevalence of obesity than men, as in the world estimates, where 18.5 % of women are found to have obesity compared to 14.0 percent of men in 2022. In the US, new statistics show that 41.3 % of women are obese as opposed to 39.2 % of men (CDC). The age factor is also significant as the obesity rate is the highest in middle-aged people, mostly adults aged between 40-59 years, with prevalence reaching up to 46.4. Another influential factor is socioeconomic status because the level of education and income is always linked to the prevalence of obesity, particularly in women, both in developed and developing nations (NCBI/PMC; Springer). Ethnic and regional differences are also high; in particular, in some African states, obesity levels in women are about 40 %, which is much higher than that of men (only about 26 %), and it is estimated that the difference between the two numbers can even increase in the next few years (The Guardian).

4.4. Role of Urbanisation and Lifestyle Transition

Rapid urbanisation has become an important contributor to the global obesity epidemic. Dietary changes that may accompany urban settings include dietary shifts towards more consumption of processed foods, sugar-sweetened beverages and energy-dense diets, accompanied by a decrease in physical activity. Research has indicated that urban residents tend to be more obese than their rural counterparts. Indeed, in Zambia, the prevalence rate of obesity among urban women (10.8%), is significantly higher compared to that of rural women (2.9%) (BMC Women's Health). This is directly connected to the concept of the more generalised nutrition transition, meaning a change in the world towards more Westernised dietary habits, characterised by increased fat, sugar and processed food consumption. Along with more sedentary lifestyles, these changes only go on to increase the rate at which obesity is on the rise globally (World Obesity Federation; Wikipedia).

The guardian.com Nearly half of women in Africa will be obese or overweight by 2030 – study Mar 6, 2025

Most adults expected to be overweight or obese by 2050 - these states need the most help Mar 4, 2025

reuters.com Obesity rates soaring globally in 'monumental social failure', study says Mar 4, 2025

theguardian.com More than half of adults worldwide will be overweight or obese by 2050 - report

5. Mechanisms Underlying Human Physiology and Obesity

5.1. Energy Balance and Metabolism

In its simplest definition, obesity arises when there is an excess of energy intake over time compared to energy expenditure, leading to fat storage. Basal metabolism is approximately 60-75 % of the total energy expenditure, and this ratio varies

according to the individual characteristics like age, sex, lean body mass, and the hormonal activity (Ravussin and Bogardus, 2000). Although obesity increases BMR because of the gain in weight, it fails to compensate for the excess calories taken (Hall et al., 2012). The second significant factor is thermogenesis and more precisely adaptive thermogenesis, i.e. the capacity of brown adipose tissue (BAT) and skeletal muscles to generate heat, as a result of overfeeding or cold exposure (Lowell & Spiegelman, 2000). This process is often curtailed in obesity because it has counterproductive effects on energy use and energy loss. In addition, defects in mitochondrial function, such as defective oxidative phosphorylation and enhanced ROS, may restrict metabolic plasticity and eventually result in fat deposition (Szendroedi et al., 2011.

5.2. Hormonal Regulation

Hormonal interaction is a complex interaction of hormones that intertwines appetite, satiation, and fat metabolism. Leptin is secreted by the adipocytes, which signals the hypothalamus that the individual is satiated, yet in many obese people, the body develops leptin resistance, i.e. the leptin level in the blood is high; however, leptin no longer suppresses hunger (Friedman, 2014). Comparatively, the stomach-derived hormone ghrelin stimulates hunger and reaches its maximum level prior to meals and in obesity, the superiority in suppressing ghrelin relative to PYY leads to increased intake of food (Cummings and Overduin, 2007). Insulin is also known to be a better fat storer and the inhibitor of lipolysis, perhaps more commonly in the role of glucose regulator of the blood. The visceral fat is closely associated with hyperinsulinemia and insulin resistance (Kahn et al., 2006). When the hypothalamic-pituitary-adrenal (HPA) axis is more activated with chronic stress, cortisol secretion is stimulated, which consequently raises the appetite and visceral adiposity (Bjornptorp, 2001). Thyroid hormones T3 and T4 also have an effect on the BMR, and a mild state of hypothyroidism actually decreases energy expenditure and causes a tendency to gain weight (Yen, 2001).

5.3. Centrally-acting mechanisms and hypothalamic pathways

The hypothalamus, particularly the arcuate nucleus, is a pivotal integrative point of afferent hormonal and neuronal satiety and hunger cues that are used to ensure the maintenance of energy homeostasis (Morton et al., 2006). Two primary neuropathways- NPY/AgRP (orexigenic) and POMC/ CART (anorexigenic) regulate the peripheral signals of hunger, including leptin, insulin and ghrelin. The disruption of this neural circuitry leads to obesity. Neurotransmitters in the brain brain which circulate among its reward systems such as dopamine and serotonin, also determine hedonic eating. When these pathways are out of order, however, the outcome can be compulsive overeating and preference of palatable high-calorie foods (Volkow et al., 2011).

5.4. Persistent activation of mature adipocytes secretes proinflammatory mediators that convert adipose tissue into an endocrine organ.

Adipose tissue is an active endocrine gland and secretes hormones and cytokines known as adipokines; it is not merely a fat reservoir [15]. Adipose tissue is primarily defined into two categories: white adipose tissue (WAT) that stores energy and releases adipokines and brown adipose tissue (BAT) that generates heat through thermogenesis (Rosen and Spiegelman, 2014). Systemic insulin resistance and chronic inflammation is caused by the endocrine organ WAT becoming hypertrophic and losing its function due to the secretion of pro-inflammatory cytokines, such as TNF-α, IL-6, and resistin (Hotamisligil, 2006). Conversely, lower levels of adiponectin that causes insulin resistance are observed in obese patients than insulin adverse effect (Kadowaki & Yamauchi, 2005). This induced low-grade chronic inflammatory condition that accompanies dysfunctional adipose tissue is a key factor influence the diseases that are associated with obesity, including type 2 diabetes and atherosclerosis.

5.5. The Gut Microbiome and Gut–Brain-Axis

New data is available on the role of the gut microbiota in obesity pathology. Balanced Metabolism. A complex and dynamic microbial environment is relevant to metabolic regulation, and disorder in the community structure of microbes (dysbiosis) has been linked to enhanced energy extraction by food, lipid generation and low-grade inflammation (Turnbaugh et al., 2006). Short-chain fatty acids (SCFA) including butyrate and acetate, are microbial metabolites that influence gut hormones like GLP-1 and PYY to regulate post-prandial satiation and insulin secretion (Canfora et al., 2015). Disturbance of this gut-brain axis has been linked with more food intake, less energy use, and more fat storage. This altered microbiome also hinders gut integrity and leads to system-wide inflammation resulting in metabolic impairment (Cani et al.2014).

5.6.1. Environmental and Behavioural Contributors

There are no completely genetic or endocrine disposed cases of obesity, but environmental and lifestyle issues also contribute largely to its development and sustainability. Modifiable lifestyle-related factors and determinants such as physical inactivity, unhealthy eating, poor sleep, psychosocial stress, and poor sleep hygiene have proven to have a significant contribution towards obesity epidemic.

5.6.2. Sedentary Lifestyle and Physical Inactivity

The main risk factor that causes obesity is sedentary behaviour, as it involves spending long durations of time without engaging in physical activities that also involve low energy expenditure. The modern environment is becoming more defined by leisure activities using screens and desk jobs, mechanised transportation, and a lack of opportunities for

unstructured physical activity (Owen et al. 2010). The World Health Organisation estimates that over 1.4 billion adults worldwide fail to engage in physical activities to the recommended levels by the WHO, which has been associated with weight gain and poor cardiometabolic health (WHO, 2020). The outcome of a sedentary lifestyle is chronic energy imbalance, reduced insulin sensitivity and low fat oxidation; eventually, slow accumulation of body weight and compartments of visceral fat accumulate (Booth et al., 2012) gradually.

5.6.3. Dietary Patterns and Processed Food

Eating habits have shifted over the past decades, to the intake of ultra-processed, energy-rich and high-sugar content foods. They are engineered to be hyper-palatable and aggressively advertised (a result in overconsumption and satiety signal). It has been shown that consumption of processed foods and fast foods also results in higher caloric density diets and weight gain among most populations (Mozaffarian et al. 2011). Besides, sweetened beverages and sweets contain no or insignificant nutrients and thus contribute to the energy deficit and micronutrient deficiency.

5.6.4. Sleep and Circadian Rhythm Disruptions

Poor sleep and lack of sleep are being recognized as a reason behind obesity. Sleep deprivation alters the secretion of the two hormones that control the appetite; decreasing leptin and increasing ghrelin, which contribute to the growth of appetite/total caloric intake (Taheri, 2004). The short duration of sleep is also linked with (i) food with high calorie content taken late at night, (ii) lack of physical activity (i.e., sedentary behaviour), as well as (iii) alteration in glucose metabolism (Spiegel et al., 2005). The circadian misalignment is referred to as shift work, which results in metabolism disturbance, subsequently resulting in insulin cross-linked and fat gain (Arble et al., 2010). Then there should be a constant and sufficient sleep to maintain our metabolism in good condition.

5.6.5. Psychosocial Stress

Psychosocial stresses that are continuous due to job demands, economic burden or absence of social support can be a cause of obesity both behaviorally and biologically. Hence, stress triggers the hypothalamic-pituitary-adrenal (HPA) axis that leads to an increase in cortisol levels- a large amount of it will stimulate the deposition of abdominal fats and insulin resistance (Bjorn, 2001). The emotional eating, which is characterized by increased intake of fat- and sugar-rich foods, is often stimulated by stress on the behavioral side (Dallman et al., 2003). This multiplies our behaviour in two ways: It leads us to consume more calories and positively reinforces reward-contingent eating behaviour and the result is a very vicious circle that is quite difficult to break without psychological help.

6. DISCUSSION

6.1. General Account of the Physiology, external and internal.

The conclusions of this systematic review point in underlining obesity is not just related to consumption of a lot of food, rather a synergistic interplay of a biological control and an environmental opportunity with excess of energy consumption. Although the outside manipulation of a modern lifestyle such as low fysical activity, diety abuse and psychosocial strains have driven the harmful influences of the dysregulated hormonal signaling, decimation of hypothalamic control and elimation of the adipose tissues, physiological causes of obesity phenotypic tendency (Bray and Ryan 2020) have been substituted by the internal modulators. Obesity, in its turn, should be considered a biopsychosocial phenomenon, that is, a combination of biological susceptibility and exposure to behaviour (Heymsfield and Wadden, 2017).

6.2. Major Findings of the Review

It is worthy to note that the review incorporates some of the most significant evidence that has shown that leptin resistance, insulin resistance, and HPA axis upregulation represent typical physiology of obesity (Friedman, 2014; Kahn et al., 2006; Bjornptorp, 2001). Furthermore, there is growing evidence to propose that members of the microbiota located in the gut are involved in the regulation of metabolism and inflammation as well (Cani et al., 2008). Epidemiologically, obesity has risen in the world with heterogeneity based on sex, age, and other population variables (Ng et al., 2014). These notes demonstrate the importance of individual treatment using lifestyle modifications and biomedical treatment.

6.3. Complexity in Physiological Clinical Management

Clinical obesity becomes of special concern because the body reacts and adapts so that managing it becomes more difficult. Once losing weight the adaptation of the human body results in a briefer metabolic rate, hunger increases and the level of hormones change hence becoming harder to maintain weight (Hall et al., 2012). Where it has often been called the set-point theory, this physiological defence system is the way adipose tissue is seen as having an apparent homeostatic integrity, as do the systems that are responsible in the regulation of the appetite (Rosenbaum and Leibel, 2010). Conventional therapies, involving energy restriction alone, therefore, may not be effective unless accompanied by metabolic and hormonal dysregulations.

6.4. Evidence deficiency and inconsistency in the literature

However, there are still significant gaps in our knowledge. A case in point concerning this would be the resistance of leptin, which is rather more generally defined but the molecular mechanisms underlying impaired leptin signalling in obesity remain poorly understood and defined (Farooqi & O'Rahilly, 2007). Similar, but not comparable gut dysbiosis

can be a complication of obesity, yet causality and correlation are a moot point, and the experimental approach using microbiota remains under translational debate (Turnbaugh et al., 2006). Furthermore, hormonal alterations are most commonly measured after comparatively brief interventions and longitudinal human studies on prolonged adaptations are not available. Moreover, genetic and epigenetic contributions to the population are not usually taken into account in populations (that are diverse, i.e., regardless of whose population was initially sampled) (Loos & Yeo, 2022).

6.5. The Review: strengths and limitations.

The main strength of this review is that it is a multidisciplinary approach that includes both the biological, behavioural, and epidemiological views to offer an insight into a multifaceted movement of obesity. PRISMA guidelines are used to bring out the transparency and uniformity of methodology. The bias of language is due to the monolingual study of English in the review. Moreover, the heterogeneity of the study design and sample population prevented a meta-analysis and probably restricted a contrast of findings. Lastly, although the review has reviewed both observational and interventional studies, much of the literature is cross-sectional, so one cannot make absolute causal conclusions.

7. CONCLUSION

- **Major Mechanisms of the Drivers of Obesity:** Obesity is a complicated disorder due to the interplay between biological, environmental and behavioural factors. As noted in this review, it is not just excess calories that pose a problem, but physiological dysregulation. These major processes involve leptin resistance, insulin dysfunction, HPA axis activation, and a change in gut microbiota, which all disrupt the regulation of appetite, energy balance, and fat stores. Furthermore, hypothalamic signalling pathways and inflammation of adipose tissue also lead to chronic metabolic dysregulation. This pattern of events, with the frame of the current environment (sedentary lifestyle, processed food, chronic stress), strengthens itself and causes weight gain and metabolic disease.

- **Relevance in Research, Clinical, and Policy:** This critical review is vital in a number of ways. We request longitudinal, human studies at the research stage of the chronic hormonal changes and gene-environment interactions. When obesity is perceived in a clinical setting as a physiologically complex disorder, it reframes the necessity to change the focus on caloric treatment methods toward more personalised treatment strategies. This can involve hormonal treatment, the regulation of gut microbiota, and behavioural therapy that does not simply treat underlying physiological imbalances. Politically speaking, obesity is an international problem and requires a systems approach solution, like implementing regulations on ultra-processed foods, better urban design that encourages active transportation, and information on sleep and stress management practices. Public health interventions should target accessible early screening, community interventions and healthy food access at both ends of the socioeconomic ladder.

- **Request Multidisciplinary Prevention Strategies:** Owing to the multidimensional nature of obesity, prevention and management should be multidisciplinary. A combination of medical, diet, mental health, and policy change will be the most effective. To develop an environment that promotes improved choices and metabolic health, healthcare systems must collaborate with education, urban planning, agriculture, and media. Only integrated, cross-sectoral interventions based on the existing efforts and delivered in a sustainable manner will allow developing a societal approach to the obesity epidemic to enhance the health of future generations.

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