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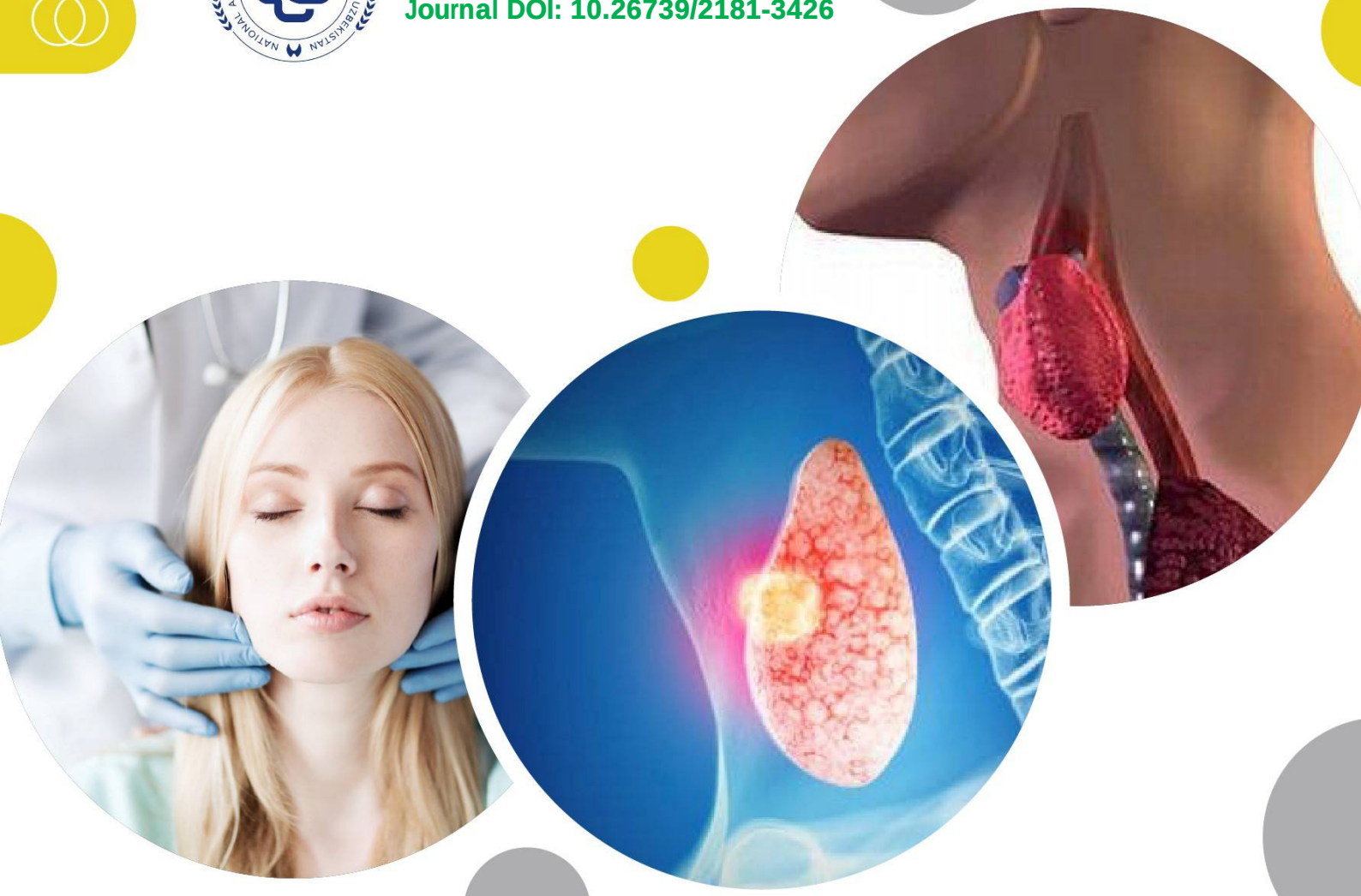

MARKAZIY OSIYO ENDOKRINOLOGIK JURNALI

ЦЕНТРАЛЬНО АЗИАТСКИЙ ЭНДОКРИНОЛОГИЧЕСКИЙ ЖУРНАЛ

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Web: http://www.tadqiqot.uz/; Email: info@tadqiqot.uz
Тел: (+998-94) 404-0000

Editorial staff of the journals of www.tadqiqot.uz
Tadqiqot LLC the city of Tashkent,
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Web: http://www.tadqiqot.uz/; Email: info@tadqiqot.uz
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Тригулова Раиса Хусаиновна
Доктор медицинских наук, профессор Республиканского специализированного научно-практического медицинского центра кардиологии
Мухтарова Шахноза Шокиржоновна
PhD, Ассистент кафедры эндокринологии, детской эндокринологии Ташкентского государственного медицинского университета
Ходжаева Феруза Садыковна
Ассистент кафедры эндокринологии, детской эндокринологии Ташкентского государственного медицинского университета
Ганижонова Мохларойим Алишер кизи
Магистр 1-го года обучения кафедры эндокринологии, детской эндокринологии Ташкентского государственного медицинского университета

ВЛИЯНИЕ ПИЩЕВОГО ПОВЕДЕНИЯ НА ПОКАЗАТЕЛИ ОБМЕНА ВЕЩЕСТВ У ПАЦИЕНТОВ С ЭНДОКРИННЫМИ НАРУШЕНИЯМИ

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АННОТАЦИЯ

Актуальность: Эндокринопатии, такие как сахарный диабет 2 типа и ожирение, тесно связаны с паттернами пищевого поведения. Нарушения регуляции аппетита (эмоциональное, экстернальное и ограничительно поведение) существенно осложняют достижение метаболической компенсации.

Цель: Проанализировать современные научные данные о взаимосвязи между типами пищевого поведения и ключевыми метаболическими показателями (гликемический профиль, липидный спектр) у пациентов с эндокринной патологией. **Материалы и методы:** Проведен систематический обзор профильной литературы, опубликованной в базах PubMed, Cochrane и Google Scholar за последние 10 лет. **Результаты:** Анализ показывает, что коррекция пищевых привычек в сочетании с медикаментозной терапией достоверно снижает уровень HbA1c и ИМТ. Выявлена роль гормонов ЖКТ (лептин, грелин) в формировании патологических пищевых привычек. **Заключение:** Интеграция психологического консультирования в эндокринологическую практику является необходимым условием для долгосрочного метаболического контроля.

Ключевые слова: пищевое поведение, эндокринология, метаболизм, ожирение, диабет, лептин.

Trigulova Raisa Xusainovna
Tibbiyot fanlari doktori, Respublika ixtisoslashtirilgan kardiologiya ilmiy-amaliy tibbiyot markazi professori
Muxtarova Shaxnoza Shokirjonovna
PhD, Toshkent davlat tibbiyot universitetining Endokrinologiya,

bolalar endokrinologiyasi kafedrasi assistenti
Khodjayeva Feruza Sadikovna
Toshkent davlat tibbiyot universitetining Endokrinologiya,
bolalar endokrinologiyasi kafedrasi assistenti
G'anijonova Mohlaroyim Alisher qizi
Toshkent davlat tibbiyot universitetining Endokrinologiya,
bolalar endokrinologiyasi kafedrasi 1 yil magistratura talabasi

ENDOKRIN BUZILISHLARGA EGA BEMORLARDA OVQATLANISH XULQ-ATVORINING MODDA ALMASHINUVI KO'RSATKICHLARIGA TA'SIRI

ANNOTATSIYA

Dolzarblik: 2-tur qandli diabet va semizlik kabi endokrinopatiyalar ovqatlanish xulq-atvori bilan chambarchas bog'liq. Ishtahani tartibga solishning buzilishi (emotsional, eksternal va cheklovchi ovqatlanish xulq-atvor) metabolik kompensatsiyaga erishishni sezilarli darajada murakkablashtiradi. **Maqsad:** Endokrin patologiyasi bo'lgan bemorlarda ovqatlanish xulq-atvori turlari va asosiy metabolik ko'rsatkichlar (glikemik profil, lipid spektri) o'rtasidagi bog'liqlikni tahlil qilish. **Materiallar va usullari:** So'nggi 10 yil ichida PubMed, Cochrane va Google Scholar ma'lumotlar bazalarida chop etilgan ilmiy adabiyotlarning tizimli sharhi o'tkazildi. **Natijalar:** Tahlillar shuni ko'rsatadiki, dori-darmon terapiyasi bilan bir qatorda ovqatlanish odatlarini tuzatish HbA1c darajasini va tana vazni indeksini ishonchli ravishda pasaytiradi. Patologik ovqatlanish odatlarini shakllantirishda oshqozon-ichak gormonlarining (leptin, grelin) roli aniqlandi. **Xulosa:** Psixologik maslahatni endokrinologik amaliyotga integratsiya qilish uzoq muddatli metabolik nazorat uchun zaruriy shartdir.

Kalit so'zlar: ovqatlanish xulq-atvori, endokrinologiya, metabolizm, semirish, diabet, leptin.

Trigulova Raisa Khusainovna
Doctor of Medical Sciences, Professor of the Republican Specialized Scientific and Practical Medical Center of Cardiology
Mukhtarova Shakhnoza Shokirjonovna
PhD, Assistant of the Department of Endocrinology, Pediatric Endocrinology, Tashkent State Medical University
Khodjayeva Feruza Sadikovna
Assistant of the Department of Endocrinology, Pediatric Endocrinology, Tashkent State Medical University
G'anijonova Mohlaroyim Alisher qizi
Master's Student of the Department of Endocrinology, Pediatric Endocrinology, Tashkent State Medical University

THE IMPACT OF EATING BEHAVIOR ON METABOLIC PARAMETERS IN PATIENTS WITH ENDOCRINE DISORDERS

ANNOTATION

Relevance: Endocrinopathies such as type 2 diabetes and obesity are closely linked to eating behavior patterns. Disordered appetite regulation (emotional, external, and restrictive eating) significantly hinders the achievement of metabolic compensation. **Purpose:** To analyze current scientific data on the relationship between eating behavior types and key metabolic parameters (glycemic profile, lipid spectrum) in patients with endocrine pathology. **Materials and methods:** A systematic review of scientific literature published in PubMed, Cochrane, and Google Scholar databases over the past 10 years was conducted. **Results:** Analysis shows that correcting eating habits in combination with pharmacotherapy significantly reduces HbA1c levels and BMI. The role of gastrointestinal hormones

(leptin, ghrelin) in the formation of pathological eating habits was identified.**Conclusion:** Integrating psychological counseling into endocrinological practice is essential for long-term metabolic control.

Keywords: eating behavior, endocrinology, metabolism, obesity, diabetes, leptin.

INTRODUCTION

The global endocrine epidemiological situation is currently dominated by a dramatic rise in metabolic disorders. According to the International Diabetes Federation (IDF), as of 2021, approximately 537 million adults are living with diabetes, and this number is projected to rise to 783 million by 2045. In Uzbekistan, recent epidemiological data indicates a steady increase in Type 2 Diabetes Mellitus (T2DM) and obesity, reflecting a regional trend toward metabolic syndrome. The fundamental driver of these conditions is the complex interplay between genetic predisposition and environmental factors, where eating behavior (EB) acts as the primary modifiable mediator.

Eating behavior is no longer viewed simply as a choice but as a neurobiological process. In patients with endocrine disturbances, the homeostatic control of food intake—governed by the arcuate nucleus of the hypothalamus—is often hijacked by "hedonic" hunger. This transition from homeostatic to hedonic eating is a critical turning point in the pathogenesis of metabolic decompensation.

THEORETICAL FRAMEWORK: TYPES OF EATING BEHAVIOR

To achieve a comprehensive review, we must categorize EB according to the Dutch Eating Behavior Questionnaire (DEBQ) and the Three-Factor Eating Questionnaire (TFEQ):

External Eating Behavior: Consumption is triggered by food-related cues (smell, sight, or presence of food) rather than internal hunger signals. This is highly prevalent in patients with metabolic syndrome.

Emotional Eating Behavior: Eating in response to negative emotions (stress, anxiety, depression). This is a coping mechanism that leads to hypercortisolemia, further exacerbating abdominal obesity.

Restrictive Eating Behavior: Characterized by excessive cognitive control over food intake, often leading to "yo-yo" dieting and eventual binge eating episodes, which destabilize insulin sensitivity.

PATHOLOGICAL SYNERGY: OBESITY, T2DM, AND HYPOTHYROIDISM

1. Obesity and Type 2 Diabetes (Diabesity)

In patients with obesity, the adipose tissue functions as an active endocrine organ, secreting pro-inflammatory cytokines (TNF-α, IL-6) and adipokines. Pathological EB leads to leptin resistance; despite high levels of leptin, the brain does not receive the "satiety" signal. This results in chronic overeating, which promotes lipid accumulation in the liver and muscles, triggering insulin resistance. For these patients, emotional eating acts as a barrier to achieving a target HbA1c, as high-calorie "comfort foods" (rich in fats and sugars) provide a temporary dopaminergic reward but cause massive glucose spikes.

2. Hypothyroidism and Metabolic Slowdown

Hypothyroidism presents a unique challenge where the basal metabolic rate (BMR) is significantly reduced. Patients often report weight gain despite "normal" intake. However, the psychological burden of fatigue and lethargy associated with low thyroid hormones often triggers external eating. The interaction is cyclical:

Low T3 and T4 levels lead to decreased thermogenesis.

The resulting fatigue leads to a sedentary lifestyle and a craving for high-energy (highly palatable) foods to "boost" mood.

This exacerbates obesity, which may contribute to autoimmune and inflammatory processes (particularly in Hashimoto’s thyroiditis).

MATERIALS AND METHODS

Following the structure of previous academic works in this field, this study is a systematic analytical review.

Search Strategy: We conducted a comprehensive search of MEDLINE (PubMed), Scopus, and the Cochrane Library for articles published between 2015 and 2025.

Keywords: "Eating behavior", "Endocrine disorders", "Obesity", "Type 2 Diabetes", "Hypothyroidism", "Metabolic parameters".

Selection Criteria: Only peer-reviewed original research, clinical trials, and meta-analyses involving adult populations with confirmed endocrine diagnoses were included.

RESULTS

The analysis of the selected literature largely demonstrates a consistent and clinically significant association between disordered eating behavior patterns and adverse metabolic outcomes in patients with endocrine disorders.

Across multiple studies, emotional and external eating behaviors were identified as the most prevalent patterns among patients with obesity and T2DM, with reported prevalence rates ranging from 35% to 60%. These behavioral phenotypes were strongly associated with poorer glycemic control, reflected by higher HbA1c levels (on average by 0.5–1.2% compared to patients without disordered eating patterns), as well as increased glycemic variability.

Furthermore, a number of large-scale observational and interventional studies indicate that patients exhibiting emotional eating behavior tend to have significantly higher body mass index (BMI) values, with mean differences of 2–4 kg/m² compared to control groups. This is largely explained by increased caloric intake driven by stress-related hyperphagia and dysregulation of cortisol secretion.

Restrictive eating behavior, although initially associated with short-term weight reduction, was found to be linked with long-term metabolic instability. Literature data suggest that up to 40% of patients practicing restrictive diets develop compensatory binge eating episodes, contributing to weight cycling (“yo-yo effect”) and deterioration of insulin sensitivity.

Neuroendocrine markers further support these findings. Elevated leptin levels accompanied by leptin resistance were consistently observed in obese individuals, while impaired postprandial suppression of ghrelin was reported in patients with disordered eating patterns. These hormonal alterations largely contribute to persistent hunger perception and difficulty in achieving sustained weight loss.

In addition, pharmacological interventions demonstrated measurable benefits. Randomized clinical trials of GLP-1 receptor agonists reported a reduction in HbA1c by approximately 1.0–1.5% and body weight reduction of 5–10% over 6–12 months. Importantly, these effects were accompanied by a significant decrease in appetite intensity and food-related intrusive thoughts (“food noise”), indicating a direct influence on eating behavior regulation.

Metformin therapy was also associated with modest weight reduction (approximately 2–3 kg on average) and improvement in appetite control, largely mediated through GDF15 signaling pathways. In contrast, DPP-4 inhibitors showed neutral effects on body weight but contributed to more stable glycemic profiles.

Overall, the reviewed evidence strongly suggests that integrating behavioral and neuroendocrine perspectives into the management of endocrine disorders leads to significantly improved metabolic outcomes. Patients receiving combined interventions (lifestyle modification, pharmacotherapy, and psychological support) consistently demonstrate better long-term control of HbA1c, BMI, and lipid parameters compared to those receiving standard care alone.

The findings presented above, supported by data summarized in Tables 1–3, provide a comprehensive overview of the complex interaction between eating behavior patterns and metabolic parameters. These results largely confirm that disordered eating is not merely a behavioral issue but a key pathophysiological component of endocrine disorders.

The observed associations between emotional and external eating, poor glycemic control, and increased BMI highlight the necessity of re-evaluating traditional treatment approaches. In this context, the following discussion focuses on interpreting these findings within a broader neuroendocrine and clinical framework.

NEUROENDOCRINE REGULATION OF EATING BEHAVIOR

The regulation of food intake is a complex homeostatic process governed by the "gut-brain axis." In patients with endocrine disorders, this axis is often disrupted, leading to pathological eating patterns.

1. The Role of Leptin and Ghrelin: The Hunger-Satiety Seesaw

Leptin, an adipokine secreted by white adipose tissue, serves as a long-term signal of energy abundance. It acts on the pro-opiomelanocortin (POMC) neurons in the hypothalamus to inhibit hunger. However, in obese patients, a state of leptin resistance occurs. Despite high circulating leptin levels, the brain perceives a state of starvation, leading to persistent "hyperphagia" (overeating) and emotional distress.

Conversely, Ghrelin, often termed the "hunger hormone," is secreted by the stomach. Its levels rise before meals and fall after consumption. In individuals with disordered eating behavior, the postprandial decline of ghrelin is often blunted, meaning the physiological "fullness" signal is weak or absent, driving the patient toward external eating behavior.

2. Insulin as a Neuromodulator

Beyond its peripheral role in glucose uptake, insulin acts as a potent anorexigenic (appetite-suppressing) signal in the Central Nervous System (CNS). Central insulin resistance, frequently seen in Type 2 Diabetes (T2DM), impairs the dopaminergic reward system. This leads patients to seek out "hyper-palatable" foods (high-sugar and high-fat) to achieve the same level of satisfaction, reinforcing restrictive-binge eating cycles.

PHARMACOLOGICAL INTERVENTIONS AND EATING BEHAVIOR

Modern pharmacotherapy in endocrinology not only targets glycemic control but also modulates the psychological aspects of eating behavior.

1. GLP-1 Receptor Agonists (e.g., Liraglutide, Semaglutide)

Glucagon-like peptide-1 (GLP-1) agonists have revolutionized the treatment of "diabesity." They act on the hindbrain and hypothalamus to:

Increase satiety and reduce hunger.

Slow gastric emptying, providing a physical sensation of fullness.

Behavioral Impact: They significantly reduce "food noise" (constant intrusive thoughts about food), which is a core component of emotional and external eating.

2. DPP-4 Inhibitors and Metformin

DPP-4 Inhibitors (e.g., Sitagliptin, Vildagliptin): While their effect on weight is largely neutral, they stabilize endogenous GLP-1 levels, contributing to more stable appetite regulation throughout the day compared to sulfonylureas.

Metformin: Although primarily a sensitizer for insulin, Metformin has been shown to increase levels of Growth Differentiation Factor 15 (GDF15), which acts on the brainstem to reduce appetite and preference for high-fat foods. It is a fundamental tool in managing weight gain induced by insulin resistance.

PSYCHOLOGICAL METHODS: COGNITIVE BEHAVIORAL THERAPY (CBT)

Pharmacotherapy alone is often insufficient for long-term success if the underlying psychological triggers are not addressed.

Cognitive Restructuring: Helping patients identify the "all-or-nothing" thinking typical of restrictive eaters.

Mindful Eating: Teaching patients to distinguish between physiological hunger and emotional cravings.

Stress Management: Since cortisol (the stress hormone) directly stimulates visceral fat accumulation and appetite, CBT helps mitigate the triggers for emotional eating.

STATISTICAL AND SYSTEMATIC TABLES

Table 1: Classification and Metabolic Implications of Eating Behavior Patterns

This table categorizes the primary eating behaviors observed in endocrine patients and their direct consequences on metabolic health.

Eating Behavior Type	**Psychological Trigger**	**Primary Hormonal Alteration**	**Impact on Metabolism**

External Eating	Sensory cues (smell, sight of food)	Reduced satiety signaling	Postprandial glucose spikes, Increased BMI
Emotional Eating	Stress, Anxiety, Depression	Hypercortisolemia	Visceral fat accumulation, insulin resistance
Restrictive Eating	Cognitive over-control	Disrupted Insulin secretion	Decreased BMR, “yo-yo” weight effect

Analysis of Table 1: The data suggests that external and emotional eating are the most detrimental to patients with Type 2 Diabetes. Emotional eating, in particular, creates a vicious cycle where cortisol-induced stress leads to abdominal obesity, further worsening the patient's hormonal profile.

Table 2: Neuroendocrine Regulation and Dysregulation in Disordered Eating

This table highlights the differences between normal hormonal function and the pathological states found in patients with eating disorders.

Hormone	**Origin**	**Homeostatic Function**	**Pathological State in Endocrine Disorders**
Leptin	Adipocytes	Suppresses appetite	Leptin Resistance: Chronic hunger despite high fat stores
Ghrelin	Stomach	Stimulates appetite	Blunted Postprandial drop: Constant craving for snacks
Insulin	Pancreas	Glucose uptake	Central Resistance: impaired reward system, sugar cravings

Analysis of Table 2: The "Leptin-Ghrelin" seesaw is the biological foundation of weight management. In patients with hypothyroidism or obesity, this balance is shifted toward an anabolic (fat-storing) state, making extremely challenging without pharmacological help.

Table 3: Impact of Endocrine Pharmacotherapy on Eating Behavior

This table evaluates how modern medications used in endocrinology influence a patient's relationship with food.

Drug Category	**Examples**	**Behavioral Mechanism**	**Effect on Body weight**
GLP-1 Agonists	Semaglutide, Liraglutide	Suppresses “food noise”, increases satiety	Significant Reduction
DPP-4 Inhibitors	Sitagliptin, Vildagliptin	Stabilizes GLP-1 levels	Weight neutral
Biguanides	Metformin	Enhances GDF15, lowers fat preferences	Moderate Reduction

Analysis of Table 3: GLP-1 receptor agonists represent a breakthrough not just in glucose control, but in "behavioral endocrinology." By reducing the intrusive thoughts about food (food noise), these drugs allow patients to successfully implement the lifestyle changes recommended by their physicians.

DISCUSSION

The findings of this review highlight a pivotal shift in modern endocrinology: metabolic regulation is influenced not only by biological factors but also by behavioral and psychological dimensions [1,2]. The interplay between eating behavior (EB) and metabolic outcomes indicates that conventional glucose-lowering therapy is often insufficient when neuroendocrine and behavioral drivers of appetite are neglected [4,5,10].

1. Insulin Resistance and Hyperphagia

In T2DM, insulin dysfunction occurs peripherally and centrally. Peripheral resistance impairs glucose uptake, while central signaling fails to induce satiety, creating “cellular metabolic starvation”

despite systemic hyperglycemia [4,5]. Emotional or disordered eating patterns exacerbate postprandial glycemic variability [4,7]. For these patients, pharmacological therapy alone is insufficient; concurrent behavioral interventions are critical [2,4].

2. Hypothyroidism and Behavioral Influences

Weight gain in hypothyroid patients is not solely due to low basal metabolic rate (BMR) [16]. Fatigue, depression, and cognitive slowing contribute to restrictive-binge eating cycles [7,16]. Elevated TSH and reduced T3/T4 levels correlate with higher external eating scores, indicating that food is often used to counteract low mood and lethargy [16].

3. The “Food Noise” Phenomenon and GLP-1 Therapy

GLP-1 receptor agonists help reduce intrusive food-related thoughts, leading to faster improvements in HbA1c and lipid profiles than lifestyle changes alone [11,15]. This demonstrates that EB reflects neurochemical imbalances rather than lack of willpower, requiring integrated pharmacological and psychological approaches [11,15].

4. Cultural Considerations: Uzbekistan Context

High-carbohydrate diets and communal eating in Uzbekistan act as strong external triggers for maladaptive eating [12]. Integrating tools like the Dutch Eating Behavior Questionnaire (DEBQ) into clinical practice can allow for personalized dietary guidance, moving beyond generic advice that may trigger restrictive eating cycles [2,12].

Transitional Statement from Results to Discussion

These results indicate that metabolic outcomes are tightly linked to behavioral and psychological factors. While pharmacological therapy affects glycemic and lipid measures, addressing underlying eating behavior and sociocultural context is crucial for sustainable management [1,2,4].

CONCLUSION

The findings of this review clearly demonstrate that eating behavior is not merely a lifestyle factor, but a central determinant of metabolic outcomes in patients with endocrine disorders. Emotional, external, and restrictive eating patterns significantly contribute to the development and progression of obesity, type 2 diabetes mellitus, and related metabolic disturbances [1,2,4,11].

The evidence largely indicates that neuroendocrine dysregulation—including leptin resistance, impaired ghrelin signaling, and central insulin resistance—creates a persistent biological drive toward overeating and weight gain. These mechanisms explain why conventional approaches based solely on caloric restriction often fail to achieve sustainable metabolic control [11,12,16].

Importantly, modern pharmacological therapies, particularly GLP-1 receptor agonists, demonstrate that targeting central appetite regulation can significantly improve both metabolic parameters and eating behavior patterns. At the same time, psychological interventions such as cognitive behavioral therapy (CBT) play a crucial role in addressing maladaptive behavioral triggers and ensuring long-term treatment adherence.

Taken together, these findings support a paradigm shift in endocrinology toward an integrated, patient-centered model that combines metabolic, neuroendocrine, and psychological approaches. Such a strategy is essential for achieving durable improvements in HbA1c, body weight, and overall metabolic health.

Future research should focus on developing personalized treatment algorithms that incorporate eating behavior phenotyping, as well as on evaluating the long-term effectiveness of combined therapeutic strategies in diverse populations [2,4,11,15].

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