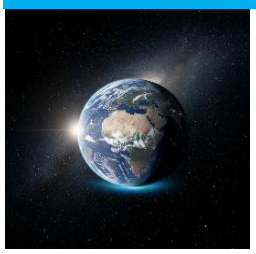
NICOLAUS COPERNICUS UNIVERSITY IN TORUŃ



Journal of Education, Health and Sport. eISSN 2391-8306.

Journal Home Page
https://apcz.umk.pl/JEHS/index

JANIK, Dominika, SOCHOCKA, Agata, SIKORSKA, Alicja, MIAN, Emil, ZAPART, Zuzanna, SZEPIETOWSKA, Martyna, NOWICKA, Nina, BUNDYRA, Jakub, GAŃSKA, Oliwia Zuzanna and PARDA, Julia. Deciphering 'Food Noise' in the Era of GLP-1 Receptor Agonists: A Comprehensive Narrative Review of Neurobiological Mechanisms and Clinical Implications for Health Education. Journal of Education, Health and Sport. 2026;88:69532. eISSN 2391-8306. https://doi.org/10.12775/JEHS.2026.88.69532

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).
Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).
The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 04.03.2026. Revised: 16.03.2026. Accepted: 17.03.2026. Published: 21.03.2026.

Deciphering 'Food Noise' in the Era of GLP-1 Receptor Agonists: A Comprehensive Narrative Review of Neurobiological Mechanisms and Clinical Implications for Health Education

Authors:

Dominika Janik

Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland

https://orcid.org/0009-0000-8527-7428

dominika.janik121@gmail.com

Agata Sochocka
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland
https://orcid.org/0009-0004-5384-2014
agatasochocka@gmail.com

Alicja Sikorska
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland
https://orcid.org/0009-0009-6274-6944
s082697@student.wum.edu.pl

Emil Mian
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland
https://orcid.org/0009-0006-1538-7190
emilmian@icloud.com

Zuzanna Zapart
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland
https://orcid.org/0009-0007-0466-4508
pczuzanna@gmail.com

Martyna Szepietowska
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland
https://orcid.org/0009-0000-2423-4563
mszepietovska@gmail.com

Nina Nowicka
Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland
https://orcid.org/0009-0004-1942-0045
nina.en@autograf.pl

Jakub Bundyra

Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland

https://orcid.org/0009-0008-7042-1990

kubabundyra4@gmail.com

Oliwia Zuzanna Gańska

Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland

https://orcid.org/0009-0007-9024-0209

oliwiaganska12@gmail.com

Julia Parda

Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warsaw, Poland

https://orcid.org/0009-0006-9079-5936

parda.julka@gmail.com

Corresponding author: Dominika Janik

E-mail: dominika.janik121@gmail.com

Abstract

Background: Obesity is a complex metabolic disease where impaired brain-gut communication often manifests as "food noise"—intrusive thoughts about food that drive overeating and hinder weight loss.

Aim: The aim of this study is to analyze the phenomenon of "food noise" as a key cognitive pathology in obesity and to assess the effect of GLP-1 receptor agonists on its suppression.

Materials and methods: A review of scientific sources (2003–2026) was conducted, covering including PubMed, , Google Scholar

Results: Food noise was identified as a measurable cognitive pathology linked to hyperactivity in the default mode network (DMN) and dysregulation in the dopaminergic reward system. Clinical and neuroimaging data confirm that incretin-based therapies, notably GLP-and dual GIP/GLP-1 receptor agonists, effectively silence these signals. They act centrally on the hypothalamus (modulating POMC and AgRP neurons), suppressing both homeostatic and hedonic eating drives. This neurological silencing directly correlates with substantial and sustained weight loss, leading to long-term obesity remission.

Conclusions: Effective treatment of obesity requires a multidisciplinary approach aimed at eliminating food noise through pharmacotherapy, CBT-OB psychotherapy, and health education.

Keywords: obesity; food noise; GLP-1; semaglutide; tirzepatide; health education.

1. Introduction: Nutritional noise as the basis of obesity pathology

1.1. The contemporary approach to obesity defines it as a complex multidisciplinary challenge, in which disturbed communication between the brain and gut plays a central role, manifesting itself in the subjective experience of "food noise" [1].This noise, clinically defined as intrusive and often unwanted thoughts about food, is a major barrier to maintaining a calorie deficit, as it constantly reinforces physiological satiety signals [2]. In a world dominated by high-calorie stimuli, food noise becomes a mechanism that drives consumption, forcing patients to constantly expend mental energy in attempts to inhibit their impulses [3]. The understanding that silencing this noise is the key to therapeutic success is changing the goals of modern metabolic medicine [4].

1.2. The evolution of the perception of hunger has led to the identification of "food noise" as a measurable psychological construct that can be studied using specialized tools such as the Food Noise Questionnaire [5]. This noise is not uniform; it includes meal planning, rumination about calories, and intense cravings for specific flavors, which drastically reduces the patient's quality of life [2]. Health education must therefore emphasize that food noise has a biological basis, which allows the patient to be relieved of guilt for "lack of willpower" and move on to effective pharmacological or surgical treatment [6].

2. The neurobiology of food noise: Neural networks and the blood-brain barrier

2.1. At the root of food noise lies a dysfunction of the resting state network [7], which in obese individuals is constantly focused on food-related projections. This state of "faulty prospecting" causes the brain to generate food cravings at every spare moment, simulating short-term rewards at the expense of long-term health goals [7]. Neuroimaging studies confirm that this noise is the result of overactivity of the reward system, particularly the striatum, and weakened inhibitory control from the prefrontal cortex [8]. To effectively silence food cravings, it is necessary to interfere with these circuits, which is possible thanks to modern incretin drugs [9].

2.2. A key aspect of silencing food noise with drugs such as semaglutide is their interaction with the blood-brain barrier (BBB). Although GLP-1 receptor agonists (GLP-1RAs) are large molecules, their noise-silencing effect is achieved through access to periventricular organs, such as the far field, which do not have a tight blood-brain barrier [10]. From there, the signal that suppresses food cravings spreads to deeper structures of the hypothalamus, modulating the activity of neurons responsible for appetite [11]. Understanding the relationship between the BBB and the central action of GLP-1 drugs explains why patients report such a rapid reduction in feeding noise almost immediately after starting therapy [10].

3. Advanced pharmacotherapy: The incretin breakthrough in noise control

3.1. The silencing of food noise has been made possible by the introduction into clinical practice of GLP-1 receptor agonists (GLP-1RAs), which mimic the action of natural incretins secreted after a meal. These drugs, such as liraglutide and semaglutide, are redefining standards of care, offering patients not only weight loss but, above all, cognitive peace of mind by stabilizing satiety signals [12].

3.2. This mechanism involves prolonging the action of the hormone, which is rapidly degraded under physiological conditions, allowing for sustained suppression of food cravings around the clock [13]. As a result, patients are no longer slaves to intrusive thoughts about food, which is a key factor distinguishing this class of drugs from older pharmacological methods [14].

3.3. Semaglutide, whose effectiveness has been confirmed in the STEP program, occupies a special place in the hierarchy of food noise suppression. It has been shown that weekly administration of semaglutide leads to a radical change in eating behavior, which patients subjectively describe as "turning off the speaker" that generates constant food noise [4]. This silencing is associated with a decrease in preference for high-fat and sweet foods, which were previously the main source of hedonic food noise [15]. This phenomenon allows patients to regain their freedom of choice, and food ceases to be the central focus of their cognitive activity [16].

4. Tirzepatide: Synergy of GIP and GLP-1 in eliminating food noise

4.1. Tirzepatide, a breakthrough dual agonist of GIP and GLP-1 receptors, offers even deeper silencing of feeding noise. The synergy of these two signaling pathways allows for effects similar to bariatric surgery, resulting in almost complete elimination of feeding noise in most patients [17]. GIP (glucose-dependent insulinotropic polypeptide) additionally acts on receptors in the brain, which enhances the effect of suppressing feeding signals generated by metabolic centers [18]. The results of the SURMOUNT study showed that such strong suppression of food noise allows for weight loss exceeding 20%, which revolutionizes our view of the possibilities of pharmacological suppression of food noise [17,19].

4.2. An additional advantage of tirzepatide in the context of food noise is its effect on glycemic stability and insulin resistance, which prevents rapid fluctuations in appetite that cause metabolic noise [12]. Patients on this therapy report that food noise disappears almost completely and their relationship with food becomes neutral, which is essential for a lasting lifestyle change [18]. Tirzepatide is so effective at silencing food noise that it is now considered the most powerful "suppressor" of pathological thoughts about food available [13]. Such a high level of control over food noise gives patients the chance for long-term remission from obesity without a constant feeling of struggle [19,20].

5. Cellular mechanisms: How POMC and AgRP neurons respond to noise

5.1. At the molecular level, food noise is silenced through precise control of neurons in the hypothalamus. GLP-1 agonists activate POMC (proopiomelanocortin) neurons, which act as "silence transmitters," sending satiety signals to the brain via while inhibiting AgRP/NPY neurons, which are the main generators of food noise and hunger [21]. This shift in hormonal balance causes the biological drive to eat to lose its strength, and food noise ceases to dominate the patient's consciousness [22]. When AgRP neurons are silenced, the brain stops scanning the environment for calories, which radically reduces the level of food noise [23].

5.2. The silencing of food noise is also associated with the direct action of drugs on the brain stem and limbic system. Semaglutide and liraglutide modify the activity of reward-related areas, which suppresses food noise generated by hedonic stimuli (e.g., the sight of cake) at its source [11].

5.3. Studies in animal models confirm that these dispersed neural pathways are critical for the complete extinction of food noise and control of energy intake [23]. As a result, patients experience "neural silence," which allows them to rebuild healthy eating patterns without the interference of intrusive food noise [9].

6. Neuroanatomy of noise: fMRI and parietal cortex activity

6.1. The precise mechanisms by which pharmacotherapy silences food noises are evident in neuroimaging studies using fMRI. Liraglutide has been shown to alter neural activity in response to images of high-calorie foods not only in the hypothalamus, but also in the parietal cortex and brainstem, which patients experience as a reduction in the intensity of food noise [9]. The parietal cortex plays an important role in attention processes, and its modulation by GLP-1RAs suggests that these drugs physically "distract" the brain from food stimuli, silencing food cravings at the cognitive level [9]. Such a wide distribution of GLP-1 receptors in the human brain confirms that food cravings are a phenomenon involving entire neural networks, not just individual hypothalamic nuclei [11].

6.2. Disorders in the dopaminergic system, especially in the striatum, are directly correlated with the intensity of food noises and the tendency to seek sensations [24]. People with a higher BMI show altered dopamine D2 receptor density, which makes their food noise louder and their satisfaction with food lower, driving a vicious cycle [8]. Silencing this noise therefore requires stabilization of neurotransmission, which is possible thanks to modern targeted therapies [25]. Knowledge of the neuroanatomical stations that process food noise allows for a better understanding of why some patients require higher doses of medication to achieve the desired effect of "silence in the head" [26].

7. GIP and GABAergic neurons: A new level of noise suppression

7.1. Recent discoveries in neurobiology indicate that dual agonists such as tirzepatide use GABAergic neurons to silence feeding noise. GIP (glucose-dependent insulinotropic polypeptide)

receptors located on GABAergic neurons in the brain have been shown to play a key role in regulating body weight and inhibiting food intake, which directly translates into a reduction in food cravings [27]. The synergy between GLP-1 and GIP allows for a "double suppression" effect, where food noise is attacked from two different hormonal pathways simultaneously. This precise approach minimizes side effects while maximizing the suppression of pathological food noise [27].

7.2. The role of GABAergic neurons in suppressing food noise is important because they act as natural "brakes" in the nervous system [27]. When dual agonists activate these cells, the food noise generated by orexigenic neurons is rapidly suppressed, allowing the patient to regain control over their impulses [23]. This molecular explanation for the phenomenon in which patients on tirzepatide report an almost complete disappearance of thoughts about food redefines the concept of "metabolic silence" [17,19,20]. The future of metabolic medicine will be based on further exploration of these specific subpopulations of neurons to optimize the processes of silencing food noise [27].

8. Physical noise vs. food noise: A multisensory perspective.

8.1. An interesting and often overlooked aspect is the influence of environmental noise on the intensity of internal food noise. External noise, for example in crowded restaurants or school cafeterias, can mask satiety signals and intensify [17,20] food noise, leading to faster consumption [28]. Studies in Italian school cafeterias have shown that noise negatively affects meal perception and may increase food waste, indirectly indicating that external noise disturbs natural appetite control mechanisms [29]. In this context, reducing food noise also requires attention to the acoustic hygiene of the environment in which we eat [28].

8.2. This phenomenon is even more evident in noisy grocery stores, where noise can act as a stressor that increases impulsivity and intensifies internal food noise in shoppers [30]. Environmental noise affects the neurophysiology of hunger, hindering the cognitive regulation of cravings, which makes food noise more difficult to control [31]. Therefore, modern obesity therapy should teach patients how to avoid environments where external noise interacts with their internal food noise, creating a cycle of uncontrolled eating [31].

9. Beyond obesity: Silencing the noise in substance addictions

9.1. An extremely promising area of research is the ability of GLP-1 agonists to silence the noise associated with other addictions, particularly alcohol. It turns out that alcohol craving and food noise share the same pathways in the reward system, which can be blocked by semaglutide [32]. These drugs reduce dopamine release in the nucleus accumbens in response to alcohol, which leads to the suppression of cravings for psychoactive substances [33]. Patients using these drugs for obesity often report the disappearance of alcohol cravings as a side effect, which opens up new perspectives in the treatment of alcohol use disorders [34].

9.2. This mechanism is based on central modulation of GABAergic neurotransmission, which stabilizes the hyperactive reward system that generates the urge [33]. Semaglutide has been shown to reduce the relaxing and rewarding effects of alcohol, which subjectively translates into a reduction in the urge to drink [32]. This phenomenon suggests that food noise and addiction noise share a common neurobiological matrix that can be effectively targeted with GLP-1 agonists [34]. Such a universal noise-reducing effect confirms that we are entering a new era of precision medicine, capable of "turning off" pathological signals in the brain [35].

10. Personalized CBT-OB therapy: Psychological noise management

10.1. Even with strong pharmacological support, food noises have a behavioral and cognitive component that requires intervention in the CBT-OB (Cognitive-Behavioral Therapy for Obesity) model. This therapy does not focus solely on calories, but on identifying psychological barriers that generate food noises in response to stress or low self-esteem [36]. Patients are taught thought monitoring techniques, which allow them to recognize early on when food cravings begin to dominate rational meal planning [36]. The integration of biology-calming pharmacology with CBT-OB, which calms the psyche, allows for lasting effects, preventing patients from giving up treatment due to unrealistic expectations [1]. The effectiveness of CBT-OB in reducing food noise is also based on addressing so-called "emotional noise," or the tendency to regulate mood through eating [36]. Research shows that obese patients often experience food noise as a defense mechanism against anxiety, which therapy helps to replace with healthier coping strategies [7]. As a result, food noise ceases to be the only available response to difficult life situations, which drastically reduces the risk of relapse [16]. Health education based on this model teaches patients that food cravings are a signal that can be understood and controlled, rather than a sentence [36].

11. Stigmatization in healthcare and the intensity of food noises

11.1. Another factor determining the intensity of food noises in patients is stigmatization by medical staff. Negative prejudices against people with higher BMIs cause them stress, which, through the hypothalamic-pituitary-adrenal axis, intensifies food cravings and pushes them to compensatory eating [6]. Patients, feeling judged, internalize the stigma, which leads to hatred of their own bodies and even louder dietary noise [6]. Patients' perceptions of medical care indicate that a lack of empathy on the part of doctors is a major barrier to effectively silencing dietary noise and building a therapeutic relationship [37].

11.2. Silencing the dietary noise therefore requires a change in attitudes in healthcare and a shift to inclusive, non-judgmental language [6]. Educating medical staff on the biological basis of nutritional noise is crucial so that patients do not feel guilty for the symptoms of their disease [38]. Only in an atmosphere of acceptance can patients focus on the substantive aspects of silencing nutritional noise [37].

12. Nutritional noise in pregnancy and in women of childbearing age

12.1. Obesity in women of childbearing age is associated with adipose tissue dysfunction, which generates hormone-related nutritional noise, hindering conception and the normal course of pregnancy [39].

12.2. Modern GLP-1 pharmacotherapy can reduce this noise and optimize fertility before conception by improving oocyte quality and insulin sensitivity [39]. However, pharmacological treatment is limited during pregnancy itself, which means that nutritional noise must be controlled through strict adherence to weight gain guidelines [40].

12.3. Excessive weight gain in obese pregnant women is often the result of an inability to control nutritional noise, leading to metabolic complications in both mother and child [40]. Silencing nutritional noise during this period is critical for achieving optimal birth weight in the newborn and avoiding gestational diabetes [41–43].

12.4. The diagnosis of food hype in pregnant women should take into account specific nutritional requirements, such as folic acid, whose deficiencies may be masked by excessive consumption caused by hype [44]. Interventions based on diet and physical activity, although more difficult to implement in the presence of strong nutritional noise, remain the foundation of perinatal care [44]. Health education must support women in consciously managing nutritional noise to ensure the safety of both organisms [40].

13. Alternative dietary models and noise reduction: ADF and TRE

13.1. An interesting perspective in the non-pharmacological silencing of nutritional noise are the models of alternate day fasting (ADF) and time-restricted eating (TRE). Studies on ADF in non-obese individuals show that periodic metabolic fluctuations can improve cardiovascular parameters and reduce visceral fat, which indirectly affects nutritional noise [45]. In turn, the TRE model combined with resistance training in overweight individuals allows for the reduction of fat mass while maintaining muscle tissue, which stabilizes satiety signals and reduces food noises [46]. However, for many obese patients, overly strict fasting may initially exacerbate food noises, leading to compensatory mechanisms [47].

13.2. Long-term success in silencing food cravings after weight loss requires an understanding of the "energy gap" phenomenon, in which appetite increases and demand decreases [47]. Metabolic adaptations to weight loss, including a decrease in leptin and an increase in ghrelin, act as amplifiers that generate food cravings, which is the main cause of the yo-yo effect [47].

13.3. Strategies such as increasing energy expenditure through exercise can help close this gap and silence nutritional noise [48]. Understanding that nutritional noise is a biological driver of weight regain allows for better design of weight maintenance programs [47].

14. The behavioral-economic aspect of noise: Reinforcer Pathology

14.1. Food noises can also be interpreted through the lens of the Reinforcer Pathology Model, which assumes excessive motivation to eat while lacking patience in waiting for delayed rewards [49]. People with obesity exhibit so-called steep discounting, which means that food noise commanding immediate consumption wins out over long-term health benefits [49]. Food cue reactivity is directly related to the impulse that intensifies internal food cravings in the presence of highly rewarding foods [50]. Calming these cravings therefore requires strengthening inhibition processes, which is possible through executive function training [51].

14.2. Food noise in children is particularly susceptible to the influence of marketing of highly processed products, which reinforces pathological consumption patterns [52]Advertisements for healthy foods, using cartoon characters, for example, can be an effective tool in silencing the noise directed towards sweets in favor of fruits and vegetables [52]. Health education should take into account the fact that food noise is shaped by the environment from an early age, and promoting healthy habits through games and play can protect children from developing food noise in adulthood [52]. Reducing food noise in the population therefore requires systemic changes in the way food is promoted.

15. Bariatric surgery as a mechanical and hormonal way to reduce food noise

15.1. For patients with morbid obesity, in whom food noise reaches pathological levels that prevent any control over energy intake, bariatric surgery remains the most effective form of intervention [53]. Long-term studies, such as SLEEVEPASS, show that both sleeve gastrectomy and gastric bypass (Roux-en-Y) lead to permanent weight loss and remission of comorbidities by radically silencing nutritional noise at its hormonal source [53]. This surgery acts as a kind of "wake-up call" for appetite regulation systems, forcing the brain to abandon noisy hunger signals in favor of metabolic homeostasis [54]. Such a profound modification of the digestive tract allows patients to maintain weight reduction of more than 20% even after a decade, which is the result of stable suppression of nutritional noise [53].

15.2. However, it should be emphasized that bariatric surgery is not a method free from the risk of recurrence, and weight regain is often heralded by a return of food noise [54].

15.3. In severe cases, pharmacotherapy with GLP-1 analogues can serve as essential support, helping to re-silence the feeding noise that has not been completely eliminated by the scalpel [54]. Innovative surgical approaches, combined with modern medications, are creating a new roadmap for the treatment of obesity, in which the overriding goal is to maintain lasting "silence" in the hunger center [25]. Understanding that feeding noise can evolve even after surgery is crucial to providing patients with lifelong multidisciplinary care [1].

16. Response variability and "Cognitive Enhancement"

16.1. An important challenge in the multidisciplinary treatment of obesity is the fact that silencing food noise does not work the same way for every patient. Studies on drugs that affect cognitive function indicate enormous individual variability in response to therapy, which means that the degree of food noise silencing may depend on the initial neurochemical state of the brain [26]. For some patients, eliminating food noise acts as a kind of "cognitive enhancement," freeing up brain resources previously absorbed by food noise and allowing for better focus on other aspects of life [26]. Understanding these differences is crucial for health education so that patients do not feel discouraged if their food noise reduction process is slower. This phenomenon is also evident in discussions on social media platforms, where patients share their experiences of the impact of GLP-1RA on food noise, libido, and other impulsive behaviors [35]. Analysis of social media data confirms that reducing food cravings is often seen as the most revolutionary aspect of therapy, changing patients' sense of agency [35]. At the same time, variability in the experience of side effects, such as nausea, can affect how patients perceive the process of reducing their food cravings [35]. This is why it is so important to personalize treatment and maintain constant dialogue with patients experiencing a transformation of their internal cognitive noise [37].

17. The patient's perspective in the context of chronic diseases

17.1. The treatment of obesity and the silencing of food noise often takes place in the shadow of other chronic diseases, which requires a tremendous effort of adaptation on the part of the patient. Patients' perceptions indicate that obesity management is difficult when the care of other conditions dominates the therapeutic process and food noise is ignored by physicians of other specialties [37]. Patients emphasize the need for an integrated approach in which silencing food noise would be treated as an integral part of the treatment of diabetes or heart disease. The healthcare system's lack of understanding of the phenomenon of food noise leads to a feeling of isolation and may exacerbate emotionally-based noise [37].Multidisciplinary support must therefore take into account the fact that reducing food noise improves the control of all comorbidities [1]. When patients are freed from intrusive thoughts about food, it is easier for them to follow recommendations regarding the intake of other medications or regular physical activity [37]. Health education should promote a partnership model in which the patient is an active participant in the process of silencing their food noise, rather than just a passive recipient of therapy [37]. Such synergy of actions is the only way to achieve lasting success in the fight against the obesity epidemic [1].

Discussion

The phenomenon of “food noise” has gained well-deserved attention in recent years as a key element in the pathophysiology of obesity. For decades, failure to lose weight was attributed to a lack of willpower on the part of patients, ignoring the powerful biological drive generated by overactive neural networks, including the reward system and the hypothalamus. The emergence of modern incretin drugs such as semaglutide and tirzepatide on the market is completely changing this paradigm. As demonstrated in this analysis, these drugs not only regulate peripheral mechanisms, but more importantly, they cross the blood-brain barrier (including through the paraventricular organs), directly targeting central appetite signals. This “neurological silencing” opens up a cognitive space for patients that was previously blocked by constant, intrusive ruminations about food and calories.However, it should be strongly emphasized that there is no miracle pill, and pharmacotherapy does not work in a vacuum. The effectiveness of treatment depends on the integration of biological actions with psychological interventions. Cognitive-behavioral therapy (CBT-OB) is essential for addressing the emotional and behavioral sources of dietary noise that medications may not be able to cope with. Furthermore, individual variability in response to GLP-1 and GIP/GLP-1 agonist drugs suggests that noise suppression does not proceed at the same rate in every patient. This requires tremendous empathy on the part of medical staff and a departure from stigmatization, which is itself a powerful stressor that exacerbates pathological food noise. Given the chronic nature of obesity, experts increasingly point out that pharmacotherapy to suppress food noise should be treated as a long-term treatment, analogous to the treatment of hypertension or diabetes. The influence of environmental factors, such as physical noise or aggressive marketing of highly processed products, further complicates the picture, pointing to an urgent need for broad systemic and educational interventions.

Conclusions

In conclusion, "food noise" represents a central, neurobiological driver of obesity rather than a mere behavioral deficit. Modern incretin-based therapies, specifically GLP-1 and dual GIP/GLP-1 receptor agonists, offer a highly effective pharmacological tool to centrally silence this drive, effectively bridging the therapeutic gap between lifestyle modifications and bariatric surgery.

However, achieving sustainable obesity remission requires a paradigm shift towards a truly multidisciplinary care model. Integrating advanced pharmacotherapy with specialized psychological interventions, such as CBT-OB, is imperative to address the complex cognitive and emotional facets of the disease. Ultimately, long-term clinical success depends not only on medical and psychological tools but also on cultivating a stigma-free, empathetic healthcare environment that recognizes obesity as a chronic, treatable neuro-metabolic condition. Future efforts must prioritize this holistic approach to improve both weight outcomes and the overall quality of life for patients.

Author's contribution:

Conceptualization: Dominika Janik
Methodology: Oliwia Gańska, Agata Sochocka
Software:Jakub Bundyra, Julia Parda
Check:Dominika Janik, Martyna Szepietowska, Alicja Sikorska, Nina Nowicka
Formal analysis: Zuzanna Zapart, Emil Mian
Investigation:Jakub Bundyra, Julia Parda
Resources:Dominika Janik, Nina Nowicka
Data curation:Emil Mian, Zuzanna Zapart, Alicja Sikorska
Writing -rough preparation:Dominika Janik, Agata Sochocka
Writing -review and editing: Dominika Janik, Oliwia Gańska, Martyna Szepietowska,
Visualization:Nina Nowicka, Zuzanna Zapart
Supervision:Dominika Janik, Nina Nowicka
Project administration:Dominika Janik

All authors have read and agreed with the published version of the manuscript.

Funding Statement: The study did not receive external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Acknowledgments: Not applicable.

Conflict of Interest Statement: The authors declare no conflicts of interest.All authors have read and agreed with the published version of the manuscript.

Statement on the use of generative artificial intelligence and AI-assisted technologies in the writing process. In preparing this work, the authors used Gemini (Google) to ensure linguistic, punctuation, grammatical, stylistic, and readability correctness. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the substantive content of the publication.

References

1. Mazur A, Sajewicz K, Dulęba J, Strzałkowska P, Kin F, Koszałko J, et al. Obesity as a Multidisciplinary Challenge. Quality in Sport. 2026;49. doi:10.12775/qs.2026.49.66725

2. Dhurandhar EJ, Maki KC, Dhurandhar N V., Kyle TK, Yurkow S, Hawkins MAW, et al. Food noise: definition, measurement, and future research directions. Nutrition and Diabetes. 2025. doi:10.1038/s41387-025-00382-x

3. Hayashi D, Edwards C, Emond JA, Gilbert-Diamond D, Butt M, Rigby A, et al. What Is Food Noise? A Conceptual Model of Food Cue Reactivity. Nutrients. 2023. doi:10.3390/nu15224809

4. Singh G, Krauthamer M, Bjalme-Evans M. Wegovy (Semaglutide): A New Weight Loss Drug for Chronic Weight Management. Journal of Investigative Medicine. 2022. doi:10.1136/jim-2021-001952

5. Diktas HE, Cardel MI, Foster GD, LeBlanc MM, Dickinson SL, Ables EM, et al. Development and validation of the Food Noise Questionnaire. Obesity. 2025;33(2). doi:10.1002/oby.24216

6. Talumaa B, Brown A, Batterham RL, Kalea AZ. Effective strategies in ending weight stigma in healthcare. Obesity Reviews. 2022. doi:10.1111/obr.13494

7. Cook G. Quieting “Food Noise”: How GLP-1s and Mindfulness Rewire the Default Mode Network (DMN) and Reward Circuits. Cureus. 2026. doi:10.7759/cureus.100818

8. Ziauddeen H, Alonso-Alonso M, Hill JO, Kelley M, Khan NA. Obesity and the neurocognitive basis of food reward and the control of intake. Advances in Nutrition. 2015. doi:10.3945/an.115.008268

9. Farr OM, Sofopoulos M, Tsoukas MA, Dincer F, Thakkar B, Sahin-Efe A, et al. GLP-1 receptors exist in the parietal cortex, hypothalamus and medulla of human brains and the GLP-1 analogue liraglutide alters brain activity related to highly desirable food cues in individuals with diabetes: a crossover, randomised, placebo-controlled trial. Diabetologia. 2016;59(5). doi:10.1007/s00125-016-3874-y

10. Dong M, Wen S, Zhou L. The Relationship Between the Blood-Brain-Barrier and the Central Effects of Glucagon-Like Peptide-1 Receptor Agonists and Sodium-Glucose Cotransporter-2 Inhibitors. Diabetes, Metabolic Syndrome and Obesity. 2022. doi:10.2147/DMSO.S375559

11. Baggio LL, Drucker DJ. Glucagon-like peptide-1 receptors in the brain: Controlling food intake and body weight. Journal of Clinical Investigation. 2014. doi:10.1172/JCI78371

12. Rabenda M, Słomczyński A, Zatyka P, Pawłowska A, Opala D, Osuch D, et al. Glucagon-like peptide-1 receptor agonists and derivatives in the treatment of obesity - review. Quality in Sport. 2024;20. doi:10.12775/qs.2024.20.53995

13. Abdi Beshir S, Ahmed Elnour A, Soorya A, Parveen Mohamed A, Sir Loon Goh S, Hussain N, et al. A narrative review of approved and emerging anti-obesity medications. Saudi Pharmaceutical Journal. 2023. doi:10.1016/j.jsps.2023.101757

14. Wang JY, Wang QW, Yang XY, Yang W, Li DR, Jin JY, et al. GLP−1 receptor agonists for the treatment of obesity: Role as a promising approach. Frontiers in Endocrinology. 2023. doi:10.3389/fendo.2023.1085799

15. Blundell J, Finlayson G, Axelsen M, Flint A, Gibbons C, Kvist T, et al. Effects of once-weekly semaglutide on appetite, energy intake, control of eating, food preference and body weight in subjects with obesity. Diabetes Obes Metab. 2017;19(9). doi:10.1111/dom.12932

16. Chong MC, Ko TYL, le Roux PL, le Roux CW. Changes in Eating Behaviour During Treatment With Obesity Medications. Clin Obes. 2026;16(1). doi:10.1111/cob.70065

17. Jastreboff AM, Aronne LJ, Ahmad NN, Wharton S, Connery L, Alves B, et al. Tirzepatide Once Weekly for the Treatment of Obesity. New England Journal of Medicine. 2022;387(3). doi:10.1056/nejmoa2206038

18. Hanslik A, Mendak M, Białek A, Walczak A, Hovagimyan A, Olszanecka M, et al. Tirzepatide - role in obesity, health and physical wellness. Information overview. Quality in Sport. 2025;38. doi:10.12775/qs.2025.38.58191

19. Aronne LJ, Sattar N, Horn DB, Bays HE, Wharton S, Lin WY, et al. Continued Treatment with Tirzepatide for Maintenance of Weight Reduction in Adults with Obesity: The SURMOUNT-4 Randomized Clinical Trial. JAMA. 2024;331(1). doi:10.1001/jama.2023.24945

20. Aronne LJ, Sattar N, Horn DB, Bays HE, Wharton S, Lin WY, et al. Continued Treatment With Tirzepatide for Maintenance of Weight Reduction in Adults with Obesity (SURMOUNT-4). Diabetologie und Stoffwechsel. 2024;19(S 01). doi:10.1055/s-0044-1785403

21. Kabahizi A, Wallace B, Lieu L, Chau D, Dong Y, Hwang ES, et al. Glucagon-like peptide-1 (GLP-1) signalling in the brain: From neural circuits and metabolism to therapeutics. British Journal of Pharmacology. 2022. doi:10.1111/bph.15682

22. Secher A, Jelsing J, Baquero AF, Hecksher-Sørensen J, Cowley MA, Dalbøge LS, et al. The arcuate nucleus mediates GLP-1 receptor agonist liraglutide-dependent weight loss. Journal of Clinical Investigation. 2014;124(10). doi:10.1172/JCI75276

23. Gabery S, Salinas CG, Paulsen SJ, Ahnfelt-Rønne J, Alanentalo T, Baquero AF, et al. Semaglutide lowers body weight in rodents via distributed neural pathways. JCI Insight . 2020;5(6). doi:10.1172/jci.insight.133429

24. Kessler RM. Obesity, dopamine, and reward behaviors. Acta Neuropsychiatrica. 2013. doi:10.1017/neu.2013.46

25. Wach D, Głaz A, Wiśniewska J, Radziejowska Z. Current innovative treatments of obesity. Journal of Education, Health and Sport. 2023;21(1). doi:10.12775/jehs.2023.21.01.020

26. Husain M, Mehta MA. Cognitive enhancement by drugs in health and disease. Trends in Cognitive Sciences. 2011. doi:10.1016/j.tics.2010.11.002

27. Liskiewicz A, Khalil A, Liskiewicz D, Novikoff A, Grandl G, Maity-Kumar G, et al. Glucose-dependent insulinotropic polypeptide regulates body weight and food intake via GABAergic neurons in mice. Nat Metab. 2023;5(12). doi:10.1038/s42255-023-00931-7

28. Spence C. Noise and its impact on the perception of food and drink. Flavour. 2014;3(1). doi:10.1186/2044-7248-3-9

29. Piochi M, Franceschini C, Fassio F, Torri L. A large-scale investigation of eating behaviors and meal perceptions in Italian primary school cafeterias: The relationship between emotions, meal perception, and food waste. Food Qual Prefer. 2025;123. doi:10.1016/j.foodqual.2024.105333

30. Olutumise AI, Oparinde LO, Oloruntoba MM, Oluwafemi SA, Oluwasanmi FA, Akinrotimi AF, et al. Assessing the socioeconomic and heterogeneous impacts of noise pollution on food markets in Akure metropolis, Nigeria. Discover Social Science and Health. 2025;5(1). doi:10.1007/s44155-025-00263-9

31. Peng-Li D, Alves Da Mota P, Correa CMC, Chan RCK, Byrne DV, Wang QJ. "Sound" Decisions: The Combined Role of Ambient Noise and Cognitive Regulation on the Neurophysiology of Food Cravings. Front Neurosci. 2022;16. doi:10.3389/fnins.2022.827021

32. Aranäs C, Edvardsson CE, Shevchouk OT, Zhang Q, Witley S, Blid Sköldheden S, et al. Semaglutide reduces alcohol intake and relapse-like drinking in male and female rats. EBioMedicine. 2023;93. doi:10.1016/j.ebiom.2023.104642

33. Czach Z, Bachurska D, Ignarska M, Ignarska J, Grygorczuk O, Krzyżanowska K. GLP-1 Receptor Agonists in Alcohol Use Disorder: Neurobiological Mechanisms and Therapeutic Potential. Journal of Education, Health and Sport. 2025;85. doi:10.12775/jehs.2025.85.66493

34. Wajda K, Mika W, Słowik J, Sieradzka I. Semaglutide and Other GLP-1 Receptor Agonists as Potential Therapies for Addiction – Mechanisms, Evidence, and Clinical Implications. Journal of Education, Health and Sport. 2025;86. doi:10.12775/jehs.2025.86.66921

35. Arillotta D, Floresta G, Papanti Pelletier GD, Guirguis A, Corkery JM, Martinotti G, et al. Exploring the Potential Impact of GLP-1 Receptor Agonists on Substance Use, Compulsive Behavior, and Libido: Insights from Social Media Using a Mixed-Methods Approach. Brain Sci. 2024;14(6). doi:10.3390/brainsci14060617

36. Dalle Grave R, Sartirana M, Calugi S. Personalized cognitive-behavioural therapy for obesity (CBT-OB): Theory, strategies and procedures. BioPsychoSocial Medicine. 2020. doi:10.1186/s13030-020-00177-9

37. Patton I, Salas XR, Hussey B, Poddar M, Sockalingam S, Twells L, et al. Patient perceptions about obesity management in the context of concomitant care for other chronic diseases. Obesity Pillars. 2023;8. doi:10.1016/j.obpill.2023.100089

38. Norman K, Burrows L, Chepulis L, Lawrenson R. GP and client perspectives of the barriers to obesity healthcare in New Zealand general practice: a metaethnography review. New Zealand Medical Journal. 2022;135(1549).

39. Duah J, Seifer DB. Medical therapy to treat obesity and optimize fertility in women of reproductive age: a narrative review. Reproductive Biology and Endocrinology. 2025. doi:10.1186/s12958-024-01339-y

40. Handzlik I, Myszkowska B, Zawada-Wiśniewska P, Wieczór R, Olejarczyk A. Pregnant Woman with Obesity: How to Diagnose and Treat Obesity During Pregnancy? A Review of Current Guidelines and Scientific Data. Journal of Education, Health and Sport. 2025;80. doi:10.12775/jehs.2025.80.60100

41. Banafshe E, Javadifar N, Abbaspoor Z, Ghanbari S, Karandish M. Implementation and Evaluation of a Weight Management Programme for Overweight or Obese Pregnant Women: A Randomised Controlled Trial. Sultan Qaboos Univ Med J. 2025;25(1). doi:10.18295/2075-0528.2878

42. Opie RS, Neff M, Tierney AC. A behavioural nutrition intervention for obese pregnant women: Effects on diet quality, weight gain and the incidence of gestational diabetes. Australian and New Zealand Journal of Obstetrics and Gynaecology. 2016;56(4). doi:10.1111/ajo.12474

43. Syafitasari J, Putri PS, Afriannisyah E. THE INFLUENCE OF NUTRITION MANAGEMENT EDUCATION ON MOTHERS KNOWLEDGE AND ATTITUDES IN FULFILLING NUTRITION FOR OBESE PREGNANT WOMEN. Media Gizi Indonesia. 2024;19(1SP). doi:10.20473/mgi.v19i1SP.52-57

44. Effect of diet and physical activity based interventions in pregnancy on gestational weight gain and pregnancy outcomes: meta-analysis of individual participant data from randomised trials. BMJ. 2017;358. doi:10.1136/bmj.j3119

45. Stekovic S, Hofer SJ, Tripolt N, Aon MA, Royer P, Pein L, et al. Alternate Day Fasting Improves Physiological and Molecular Markers of Aging in Healthy, Non-obese Humans. Cell Metab. 2019;30(3). doi:10.1016/j.cmet.2019.07.016

46. Kotarsky CJ, Johnson NR, Mahoney SJ, Mitchell SL, Schimek RL, Stastny SN, et al. Time-restricted eating and concurrent exercise training reduces fat mass and increases lean mass in overweight and obese adults. Physiol Rep. 2021;9(10). doi:10.14814/phy2.14868

47. Melby CL, Paris HL, Foright RM, Peth J. Attenuating the biologic drive for weight regain following weight loss: Must what goes down always go back up? Nutrients. 2017. doi:10.3390/nu9050468

48. King N, Byrne N, Hills A. Exercise, appetite and weight management. ACHPER Active & Healthy Magazine. 2010;17(3/4).

49. Musquez M, Rasmussen EB. Food Cue Reactivity Meets the Reinforcer Pathology Model: Behavioral Economic Measures of Cue-Induced Changes in Food Reinforcer Efficacy. Perspect Behav Sci. 2024;47(2). doi:10.1007/s40614-024-00409-1

50. van den Akker K, Stewart K, Antoniou EE, Palmberg A, Jansen A. Food Cue Reactivity, Obesity, and Impulsivity: Are They Associated? Current Addiction Reports. 2014. doi:10.1007/s40429-014-0038-3

51. Verbeken S, Braet C, Goossens L, van der Oord S. Executive function training with game elements for obese children: A novel treatment to enhance self-regulatory abilities for weight-control. Behaviour Research and Therapy. 2013;51(6). doi:10.1016/j.brat.2013.02.006

52. Folkvord F, Hermans RCJ. Food Marketing in an Obesogenic Environment: a Narrative Overview of the Potential of Healthy Food Promotion to Children and Adults. Current Addiction Reports. 2020. doi:10.1007/s40429-020-00338-4

53. Salminen P, Helmio M, Ovaska J, Juuti A, Leivonen M, Peromaa-Haavisto P, et al. Effect of laparoscopic sleeve gastrectomy vs laparoscopic roux-en-y gastric bypass onweight loss at 5 years among patients with morbid obesity the SLEEVEPASS randomized clinical trial. JAMA - Journal of the American Medical Association. 2018;319(3). doi:10.1001/jama.2017.20313

54. Cohen R V., Cummings DE. Weight Regain After Bariatric/Metabolic Surgery: A Wake-Up Call. Obesity. 2020. doi:10.1002/oby.22822