Obesity

WILEY | THE OBESITY SOCIETY

LETTER TO THE EDITOR OPEN ACCESS

The Voice of a Disease: Why Food Noise Can No Longer Be Ignored!

Abdulhameed Alhazmi[1,2] | Carel W. le Roux[2,3]

[1]Internal Medicine, Jazan University Hospital, Jazan, Saudi Arabia | [2]Diabetes Complications Research Centre, University College Dublin, Dublin, Ireland | [3]Diabetes Research Centre, Ulster University, Belfast, UK

Correspondence: Abdulhameed Alhazmi (abdulalhazmimd@gmail.com)

Received: 17 November 2025 | **Revised:** 15 December 2025 | **Accepted:** 6 January 2026

Diktas et al. [1] present a rigorous study establishing the Food Noise Questionnaire (FNQ) as the first validated tool to quantify intrusive food-related thoughts in individuals with overweight and obesity. The authors demonstrate psychometric reliability and validity, providing a practical measure to the neurobehavioral construct. Their findings represent a step toward operationalizing food noise (FN) as a measurable phenomenon rather than a subjective experience. However, while the FNQ's validation is a major advance, the clinical interpretation of FN and its potential role as a disease activity marker remain to be explored.

The struggle with obesity is not simple as a battle of willpower. The disease of obesity is complex and has a much deeper brain-gut relation than thought [2]. The hypothalamus regulates adipocyte mass by integrating hormonal and neural signals from the gut and adipose tissue to maintain energy homeostasis [2]. In obesity, this homeostasis becomes dysregulated, leading to persistent hunger and intrusive thoughts about eating that reflect the brain's defended higher adipocyte mass [2]. These thoughts tend to be persistent, undesirable, or distressing in a noisy pattern patients call "food noise" [1]. Rather than dismissing this phenomenon as a psychological lack of discipline, we must ask: could FN be a symptom, a clinical signifier of aberrant neuroendocrine signaling?

1 | Symptomatic Urge is a Real Deal

This concept of relying on "symptomatic urges" is no stranger to the field of endocrinology. A patient with primary adrenal insufficiency has overwhelming salt craving, which represents a behavioral adaptation to the critical, homeostatic drive stemming from aldosterone deficiency and sodium wasting [3]. Thus, the symptom is so characteristic that its emergence in a patient treated with mineralocorticoid replacement signals that the treatment is insufficient.

Similarly, the unquenchable thirst with compulsive water drinking is a pathognomonic feature of central diabetes insipidus (CDI) and indicated vasopressin deficiency [4]. While the presence of thirst is an important symptom during the management of patients with CDI, the absence of the symptom can lead to "adipsic diabetes insipidus" resulting in unrecognized hypernatremia [4]. The urge to drink water is a logical, physiological response to the failure of the posterior pituitary and the resulting unregulated diuresis [4]. When clinicians treat the underlying deficiency of vasopressin, the pathological thirst resolves [4].

2 | Why Should We Consider FN as a Pathological Phenomenon?

What is the pathophysiological basis for FN in obesity? The current hypothesis is that FN is a symptom indicating a dysregulation of adipocyte mass. In many forms of obesity, adipocyte mass is regulated at an elevated mass resulting in the complications of obesity, a state of abnormal or excess adiposity [2]. FN may represent the subcortical areas of the brain trying to increase the adipocyte mass to achieve homeostasis. The brain, starved of the appropriate satiety signals, such as GLP-1, oxyntomodulin, peptide YY, and amylin, remains in a persistent state of perceived energy deficit, driving foraging behavior and constant cognitive engagement with food [1, 2]. This is not a failure of will; it is a failure of internal biochemical communication. Supporting this, neuroimaging studies and intracranial electrophysiology show heightened neural reactivity to food cues in the reward circuits

TABLE 1 | Effects of obesity therapy modalities on food noise.

Mechanism	GLP-1 receptor agonists	Bariatric surgery
Enhances GLP-1 signaling	Pharmacologic activation of GLP-1 R	Increased endogenous GLP-1 secretion
Reduces appetite and cravings	Via gut–brain signaling	Via gut–brain signaling
Dampens reward drive and food cue reactivity	↓ Dopaminergic drive	↓ Food reward after surgery
Leads to food noise reduction	Reported as "mental quiet"	Reported as "loss of food obsession"

Abbreviations: GLP-1, glucagon-like peptide-1; GLP-1 R, glucagon-like peptide-1 receptor.

of individuals with obesity, suggesting a neurobiological substrate for the constant preoccupation [5, 6]. The classical test in endocrinology to understand causation is "if something is high, suppress it, and if something is low, stimulate it," then evaluate the change in symptoms and signs of the disease.

3 | Therapeutic Evidence Supporting FN as a Disease Marker

Just as adequate treatment of primary adrenal insufficiency and CDI results in the resolution of salt craving and excessive thirst, effective treatment of the disease of obesity may likewise lead to the attenuation or remission of FN. Both GLP-1 receptor agonists (GLP-1 RAs) and bariatric surgery appear to silence the noisy food thoughts [1, 2]. However, it is important to acknowledge that not all patients treated with GLP-1–based therapies experience complete silencing of FN, nor is this maintained in all patients over time [6].

Semaglutide and tirzepatide as examples of effective treatments for the disease of obesity act centrally on receptors in the hypothalamus and hindbrain to adjust the regulation of adipocyte mass [6, 7]. Similar neural mechanisms are triggered after bariatric surgery, which substantially elevates postprandial GLP-1, oxyntomodulin, and peptide YY levels, reinforcing gut–brain satiety signaling and reducing cravings and FN intensity [8]. As highlighted by Purnell and le Roux, the hypothalamus integrates hormonal and reward inputs to regulate adipocyte mass; disruption of this system underlies pathological appetite drive in obesity [2]. Theoretically, restoration of GLP-1 signaling pharmacologically or surgically would reestablish hypothalamic inhibitory tone, attenuating FN and promoting durable weight loss. Table 1 shows that FN reduction may therefore represent a measurable indicator of successful central adipocyte mass recalibration following GLP-1 therapy or bariatric surgery.

There are two complementary instruments currently available to quantify FN. The Ro Allison Indiana Dhurandhar Food Noise Inventory (RAID-FN), a detailed 29 item measure founded through Dhurandhar et al. to assess the full behavioral structure of FN across four domains: cognitive burden, persistence, dysphoria, and self-stigma [9, 10]. Although conceptually robust, RAID-FN is yet undergoing psychometric refinement, making it more suited for research settings where comprehensive behavioral profiling is needed. In contrast, the FNQ is a concise five item tool that has already demonstrated excellent internal consistency, strong test–retest reliability, and clear discriminant validity [1]. It has low respondent burden, making it highly applicable for routine clinical practice, longitudinal monitoring, and therapeutic response assessment in both GLP-1 therapy and bariatric procedures. Together, RAID-FN offers depth while the FNQ provides validated clinical applicability, forming a complementary framework for integrating FN measurement into obesity bedside care and academic research.

In conclusion, although GLP-1-based therapies and bariatric surgery can alleviate FN for many individuals, this effect is not universal, and not all patients experience the same magnitude of change in the longer term [6]. Structured instruments such as the FNQ and future refined questionnaires may thus play a central role. Current data support a correlation between FN and the disease of obesity, yet much like salt craving in primary adrenal insufficiency or pathological thirst in CDI, FN is best interpreted as part of a broader clinical assessment rather than a stand-alone diagnostic marker. FN-related questionnaires may be very impactful as they are poised to help quantify disease severity, track therapeutic response, and identify early signals of relapse after treatment.

Acknowlegments

This manuscript is part of the Stratification of Obesity Phenotypes to Optimize Future Therapy (SOPHIA) project. SOPHIA has received funding from the Innovative Medicines Initiative 2 Joint Undertaking under grant agreement No. 875534. This Joint Undertaking received support from the European Union's Horizon 2020 research and innovation program and EFPIA and T1D Exchange. The communication reflects the authors' view and neither the IMI nor the European Union, EFPIA, or any Associated Partners are responsible for any use that may be made of the information contained therein.

Funding

The authors have nothing to report.

Conflicts of Interest

Abdulhameed Alhazmi declares no conflicts of interest. Carel W. le Roux has received personal fees from Boehringer Ingelheim, GI Dynamics, Herbalife, Johnson and Johnson, Keyron, Eli Lilly, and Novo Nordisk outside the submitted work.

Data Availability Statement

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Abdulhameed Alhazmi
Carel W. le Roux

References

1. H. E. Diktas, M. I. Cardel, G. D. Foster, et al., "Development and Validation of the Food Noise Questionnaire," *Obesity (Silver Spring)* 33 (2025): 289–297.

2. J. Q. Purnell and C. W. le Roux, "Hypothalamic Control of Body Fat Mass by Food Intake: The Key to Understanding Why Obesity Should Be Treated as a Disease," *Diabetes, Obesity & Metabolism* 26, no. Suppl. 2 (2024): 3–12.

3. M. J. Morris, E. S. Na, and A. K. Johnson, "Salt Craving: The Psychobiology of Pathogenic Sodium Intake," *Physiology & Behavior* 94, no. 5 (2008): 709–721.

4. M. Tomkins, S. Lawless, J. Martin-Grace, M. Sherlock, and C. J. Thompson, "Diagnosis and Management of Central Diabetes Insipidus in Adults," *Journal of Clinical Endocrinology and Metabolism* 107, no. 10 (2022): 2701–2715.

5. E. Stice, S. Spoor, J. Ng, and D. H. Zald, "Relation of Obesity to Consummatory and Anticipatory Food Reward," *Physiology & Behavior* 97, no. 5 (2009): 551–560.

6. W. Choi, Y. H. Nho, L. Qiu, et al., "Brain Activity Associated With Breakthrough Food Preoccupation in an Individual on Tirzepatide," *Nature Medicine* 31 (2025): 4038–4043, https://doi.org/10.1038/s41591-025-04035-5.

7. J. Aaseth, S. Ellefsen, U. Alehagen, T. M. Sundfør, and J. Alexander, "Diets and Drugs for Weight Loss and Health in Obesity - An Update," *Biomedicine & Pharmacotherapy* 140 (2021): 111789.

8. A. M. Koball, G. E. Ames, A. J. Fitzsimmons, K. J. Kallies, and B. A. Bennie, "Food Cravings After Bariatric Surgery: Comparing Laparoscopic Sleeve Gastrectomy and Roux-En-Y Gastric Bypass," *Eating and Weight Disorders* 29 (2024): 7.

9. E. J. Dhurandhar, K. C. Maki, N. V. Dhurandhar, et al., "Food Noise: Definition, Measurement, and Future Research Directions," *Nutrition & Diabetes* 15, no. 1 (2025): 30, https://doi.org/10.1038/s41387-025-00382-x.

10. E. J. Dhurandhar, K. C. Maki, N. V. Dhurandhar, et al., "Development and Rigorous Multistep Validation of a Psychometric Tool to Measure Food Noise," *Appetite* 217 (2026): 108339, https://doi.org/10.1016/j.appet.2025.108339.