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Article

Exploring personalized diagnosis and intervention in binge eating disorder: Five case reports

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Abstract: Background: Binge Eating Disorder (BED) has gained attention in recent years due to its complexity and the challenges it poses in diagnosis and treatment. Unlike other eating disorders such as anorexia nervosa and bulimia, BED has been less researched, particularly regarding the influence of genetic factors and biomechanical on eating behaviors. This study introduces a novel approach by individually analyzing how genetic predisposition and biomechanical factors impacts the diagnosis of BED. The primary objective of this research was to demonstrate the diagnostic variability and intervention possibilities in patients with BED, highlighting the importance of an interdisciplinary approach that integrates biomechanical principles. Additionally, it aimed to identify individual factors—clinical, psychosocial, biomechanical and genetic—that influence the presence of the disorder, and to evaluate the efficacy of personalized treatments that include psychological, psychiatric, and nutritional interventions tailored to each patient's unique needs. **Case presentation:** The main concerns of the patients were how to manage their food-related anxiety, which was often exacerbated by biomechanical stressors. Many felt stigmatized by their weight and guilty for being unable to control their binge eating, which they initially attributed to a lack of self-control. However, upon learning about their genetic profile and the biomechanical underpinnings of their condition, patients began to better understand their eating behaviors, allowing them to reduce the associated guilt. Clinically, it was observed that after receiving interdisciplinary treatment, which included both psychological interventions and precision nutritional management, and biomechanical modulation, binge eating episodes significantly decreased. In four out of five cases, episodes disappeared. **Conclusions:** This reinforces the importance of tailoring treatments to the genetic and psychosocial, and biomechanical specifics of each patient. By incorporating biomechanical insights into therapeutic strategies, new research opportunities are opened, and the therapeutic approach for BED is significantly improved. This interdisciplinary framework not only addresses the psychological and nutritional aspects of BED but also leverages biomechanical principles to optimize treatment outcomes, offering a more holistic and effective approach to managing this complex disorder.

Keywords: case report; BED; biomechanics; personalized diagnosis; personalized intervention; nutrigenetics

1. Background

Binge Eating Disorder (BED), officially recognized in the Diagnostic and

Statistical Manual of Mental Disorders (DSM-5) in 2013 [1], is a relatively new diagnostic entity. Its late recognition means many aspects of BED remain underexplored, posing a challenge for the scientific and medical communities. It is often confused with other eating behaviors, particularly those linked to obesity. Unlike more well-known disorders like anorexia nervosa (AN), BED is harder to detect due to its subtle nature, making diagnosis and treatment more challenging.

1.1. Definition and prevalence of binge eating disorder

BED is characterized by recurring episodes of the consumption of large quantities of food in a short period of time, along with a feeling of losing control during these episodes [1]. As opposed to bulimia nervosa (BN), individuals with BED do not perform compensatory behaviors regularly, which can lead to a significant increase in weight and health complications, such as cardiovascular diseases and Type 2 diabetes [2]. Presently, it is estimated that BED affects approximately 1.9% of adults worldwide, with higher rates in women than men [2]. However, its prevalence could be underestimated in specific populations, such as athletes, for whom BED can be confused with normal dietary control practices [3].

1.2. Detection and diagnosis challenges

One of the main obstacles for the management of BED is its detection. Although the DSM-5 provides a clear framework, the condition can easily be confused with feeding behaviors associated with obesity [1]. As opposed to AN, which has evident physical symptoms, BED can manifest in normal weight or obese individuals, which makes its visual diagnosis more difficult. Also, BED episodes tend to occur in private, which complicates its identification [4]. To improve its diagnosis, tools such as the Binge Eating Scale (BES) [5] and the Yale Food Addiction Scale (YFAS) have been developed [6,7]. However, these self-report measures have limitations, such as the possibility that the symptoms are reported in an inexact manner, complicating its diagnosis [8,9,10].

Recently, genetic and environmental factors that contribute to BED have been explored. Genome Wide Association Studies (GWAS) seek to identify genetic markers related with BED, such as in the project BEGIN [11]. Although conclusive results are lacking, it is suggested that genetic pre-dispositions can exist, which along with environmental influences, contribute towards the development of the disorder [11–13]. However, the studies that have examined these interactions are even more limited [14].

1.3. Treatment approaches for eating disorders (ED)

Eating disorder (ED) treatments generally involve psychological and psychiatric interventions, with cognitive behavioral therapy (CBT) being the most common and efficient approach. CBT helps patients challenge negative thinking patterns and develop healthy coping mechanisms [15]. It has also been shown that dialectical behavior therapy (DBT) is efficient, especially for those who have co-existing conditions such as depression or anxiety [16].

Pharmacological treatments, such as selective serotonin reuptake inhibitors (SSRIs), are frequently used, although their efficacy varies between individuals [17].

In addition, the FDA has approved lisdexamfetamine for the treatment of BED, which has been demonstrated to be efficient in the reduction of BED episodes [18,19]. However, the stigma associated with binge eating, and the perception that it is a less severe disorder as compared to AN or BN, make it difficult for individuals to seek treatment [4].

Given the above, the aim of the present study is to demonstrate the diagnostic variability and the possibilities of intervention for each patient with BED, underlining the need for an interdisciplinary approach. The secondary objectives include analyzing the differences in clinical, psychosocial, and genetic patterns, as well as how to assess the efficacy of the interventions that include psychological, psychiatric, and nutritional treatments.

2. Method

2.1. Procedure

Case reports were obtained through the recruitment of individuals with suspected compulsive eating behavior via the nutrition and dietetics clinic of one of the co-authors, after receiving approval from the Ethics Committee for Non-Invasive Research Involving Humans. All participants provided written informed consent before participating in the screening process.

Initially, all volunteer subjects completed a screening questionnaire assessing food dependency and binge eating behavior: YFAS [20] and BED [5]. Subjects who scored over 5 out of 11 on the YFAS 2.0 and 18 out of 46 on the BES participated in a clinical interview conducted a few days later by a psychologist specialized in eating disorders (ED), in which their eating behavior was investigated, and it was determined whether they met the preliminary diagnostic criteria established by the DSM-V [1]. Only those who met the criteria were considered as compulsive eaters and participated in the rest of the study. The five potential participants were asked to provide written informed consent for further involvement. The five participants selected, all suspected of having BED, displayed different facets of BED-related eating behavior to demonstrate its variability.

In the nutritional planning, first, the references for food consumption from the EFSA [21] were considered, and second, necessary variations based on the analyzed genetic polymorphisms were accounted for. Nutrient intake was specified using Dietopro® software.

The process of interdisciplinary communication between psychologist and nutritionist was carried out continuously. Initially, the professionals met to define the key areas to address: the patient's emotional and cognitive patterns on one hand, and eating habits on the other. The psychologist focused on modifying dysfunctional thoughts related to food, while the nutritionists designed a personalized meal plan.

During the sessions, psychologist provided information on the patient's emotional progress, such as the emotions that triggered binge eating, allowing nutritionist to adjust the dietary recommendations. In turn, nutritionists offered strategies to integrate the meal plan into the patient's daily routines, which was discussed during the psychotherapy sessions.

Meetings between professionals were held every two weeks, coinciding with the

patient's consultations, which facilitated constant feedback and adjustments in the treatment. This research has been developed in strict adherence to ethical and scientific guidelines set by various international and national frameworks. First, the study followed the principles of the Case Report Guidelines (CARE), which provide guidelines for presenting detailed and accurate clinical reports [22]. Additionally, the study complies with the ethical principles set out in the Declaration of Helsinki, a key reference in medical research that advocates for the protection of human participants' rights, dignity, and well-being [23]. The research was also reviewed and approved by the Ethics Committee of the University of Almería (UALBio2024/022), ensuring compliance with ethical research regulations involving humans.

It is important to note that the protection of personal data and participants' privacy was ensured in accordance with the General Data Protection Regulation (GDPR) of the European Union (2016/679), maintaining patient anonymity and confidentiality of the information provided.

Furthermore, the genetic analysis and biological samples were conducted at a specialized center for clinical and genetic studies, ensuring the reliability and accuracy of the obtained results.

2.2. Sample

Table 1 shows the descriptive data of the five cases. The three women and two men were aged between 27 and 51 years, with a mean age of 40 years (SD = 11.113). Their body mass index (BMI) ranged from 25.4 kg/m^2 to 57.7 kg/m^2, with a mean of 34.74 kg/m^2 (SD = 13.113).

Table 1. Sociodemographic characteristics of the participants.

Case	A	B	C	D	E
Age (years)	50	42	51	30	27
Gender	Male	Female	Female	Male	Female
BMI kg/m^2	25.4	30	27.9	57.7	32.7
Eating habits*	Intermittent Fasting	Low carbohydrate diet	Diet for weight loss	High protein diet	Not specified
Sport Habits*	Competitive athlete	Engages in physical activity (group classes 3 times per week)	Ex-competitive athlete. Currently sedentary.	Engages in physical activity (bodybuilding) 5 days a week.	Does not engage in nor has previously engaged in regular physical activity.

*Self-description of the participant.

2.3. Materials

A sociodemographic questionnaire was used to assess age, gender, height, weight, and eating habits as well as physical exercise or sports practices.

The YFAS 2.0 scale [20], based on the original scale by Gearhardt et al. [6] used to measure food addiction, consists of 35 items with responses on a Likert scale ranging from 0 to 4 (never, once a month, 2–3 times a week, and 4 or more times a week). For interpretation, each question is part of a set related to the criteria established in the DSM-V for BED. If the response is "2–3 times a week" or "4 or more times a week," it is scored 1 (all other responses are scored 0). If the count of all questions for

each criterion is ≥1, the overall criterion score is 1 (if not, it is 0). This scale has a reliability of $\alpha = 0.94$ for the Spanish population [24].

The Binge Eating Scale (BES) [5] helps in detecting and diagnosing binge eating. The purpose of the instrument is to determine whether a Binge Eating Disorder exists and assess its severity. The scale consists of 16 items grouped into two dimensions: 1) cognitive manifestations and 2) behavioral manifestations. Each question has three to four response options, assigned a numerical value from zero to three points. The total score ranges from 0 to 46. Respondents are categorized into three levels of severity: those scoring 17 or less do not have binge eating, those scoring between 18 and 26 exhibit moderate binge eating symptoms, and those scoring 27 or higher have severe disorder severity. A cutoff score of 17 is typically used, with a score of 18 or higher indicating the presence of binge eating [5]. This scale has a reliability of $\alpha = 0.89$ for the European population [20].

Using the DSM-V diagnostic criteria, a semi-structured interview was designed to assess whether participants could be identified with BED. Twelve questions were developed to reflect the 4 primary criteria and 3 secondary criteria related to Criterion A (see **Table 2**), allowing the researchers to assess the extent to which the behavior described by the individuals met the preliminary criteria. To include an individual in the sample with suspected compulsive eating behavior (BED), responses to at least 4 of the 8 questions for Criterion A and 2 questions for Criteria B, C, and D had to indicate this diagnosis.

Table 2. DSM-V diagnostic criteria and corresponding questions used in the interview to assess BED.

	Criteria	Interview questions
A	Recurrent episodes of binge eating associated with 3 (or more) of the following factors	
A_1	Eating more quickly than usual.	How long does it take you to eat the main meals? When comparing yourself to others, do you think you eat faster?
A_2	Eating until feeling uncomfortably full.	Can you stop when you start eating, or do you eat until you feel uncomfortably full?
A_3	Eating large amounts of food when not physically hungry	At what time of day do you usually experience binge episodes? When they happen, do you feel hungry? (e.g., stomach growling, many hours without eating, etc.)
A_4	Eating alone due to feeling embarrassed about the quantity consumed.	Do you tend to seek moments of solitude to eat? Do you feel embarrassed and/or avoid eating in public because of the amount of food you consume?
A_5	Feeling disgusted with oneself, depressed, or ashamed after the binge episode.	Do you feel ashamed or experience feelings of distress after binge episodes?
B	Intense distress regarding the binge eating episodes.	Could you define what you feel regarding the situation you experience with binge episodes?
C	The binges occur, on average, at least once a week for three months.	Could you count the binge episodes you've had in the last month? Do you consider them to be many?
D	The binge eating is not associated with the recurrent presence of inappropriate compensatory behaviors, such as in bulimia nervosa, and does not occur exclusively during the course of bulimia nervosa or anorexia nervosa.	Do you usually engage in compensatory behaviors after binge episodes? (e.g., vomiting, excessive exercise, prolonged fasting, etc.)

In addition, several questions related to eating behavior and habits, as well as related behaviors, were asked to obtain a general overview of the individual's eating

behavior and physical exercise and/or sports practices in order to assess the criteria in a broader context. These questions included: "How would you describe your eating behavior?", "What draws your attention about your way of eating?", "Do you engage in physical exercise?", "How many times a week?", "Do you engage in any other activity in your daily life that you might think is done in an excessive way?", "Since when do you remember this behavior with food?" These questions were ordered and formulated in a way that allowed for a smooth conversation about the participants' eating behavior. The interviews were conducted in the participants' native language.

Finally, information was gathered on the presence of genetic polymorphisms associated with BED in the scientific literature [14], as well as others not directly related to BED but linked to overeating or binge episodes in conditions such as obesity, AN, or BN. The polymorphisms were detected through a laboratory test at the "Echevarne Laboratory" (see Appendix A, B, C and D). The procedure involves taking a DNA sample from the patient through a blood test. This sample is analyzed using microarray technology, which allows for the identification of genetic variants associated with obesity and metabolism. Microarrays are an advanced tool that enables the simultaneous analysis of thousands of genetic variants, providing a detailed genetic profile. From this data, a genetic profile is obtained that can provide relevant information about genetic predisposition to obesity. This predisposition has been linked to BED in the review conducted by Monserrat-Hernández and Jiménez-Rodríguez [14]. Specifically:

1) DRD2 rs1800497 (Dopamine Receptor D2). Associated with a decrease in glucose metabolism in the striatum, this polymorphism has been linked to eating disorders and may influence compulsive behaviors, such as binge eating [25–27].
2) FTO rs1558902, rs1421085, rs1121980, rs9939609 (Fat Mass and Obesity-Associated Gene). Associated with obesity, variations in this gene can affect appetite regulation and increase the risk of dysregulated behaviors, such as binge eating [28–30].
3) MC4R rs17782313 (Melanocortin 4 Receptor). Affects satiety signaling, promoting excessive intake and binge episodes [27].
4) TAS1R2 rs120338382 (Taste Receptor Type 1 Member 2). Can alter the sweet taste detection threshold, predisposing individuals to greater consumption of sugary foods [31].
5) GLUT2 rs5400 (Glucose Transporter Type 2). Associated with increased intake of sugar-rich foods due to a reduced ability to detect blood glucose levels. It may influence the consumption of ultra-processed foods and compulsive behaviors [32–34].
6) TCF7L2 rs7903146 (Transcription Factor 7-Like 2). Interferes with the activation of GLP-1 (glucagon-like peptide-1), a hormone crucial for regulating appetite and the metabolism of carbohydrates and fats [35].
7) FABP2 rs1799883 (Fatty Acid-Binding Protein 2). The A allele of the SNP rs1799883 in the FABP2 gene has been associated with elevated leptin levels and insulin resistance. These metabolic factors may promote compulsive eating [8,36].
8) CLOCK rs1801260 (Clock Circadian Regulator). Affects the regulation of circadian rhythms and the secretion of insulin, leptin, and ghrelin, key hormones

in appetite control. It can influence eating patterns, increasing the likelihood of binge episodes at specific times of day, such as at night [37].

2.4. Analysis

The total scores of the questionnaires were calculated according to the instructions in the respective manuals. BMI was calculated using the formula: weight in kg/height in m^2. Scores for the scales and subscales are reported.

Regarding the genetic profile, it is indicated whether or not the polymorphism detected in the scientific literature related to compulsive eating behavior is present.

The description of each case follows a narrative design. Since there is no consensus on the severity of BED [1,24,38], the score based on the criteria met was used as an approximation. Due to the study design, inferential statistics were not calculated.

3. Cases presentation

3.1. Descriptive data

For each case, the individual scores are shown in **Tables 3–5**.

Table 3 shows that the participants obtained high scores in the scales used, with female Patient B obtaining the highest score in BED and female Patient C the highest in YFAS. In general, the women obtained higher scores than the men in the YFAS.

Table 3. Scores and range of results obtained on the administered scales (YFAS 2.0 and BES).

	Case A	**Case B**	**Case C**	**Case D**	**Case E**	**Rango**
YFAS total Scale/ scoring for 12 criterions	**86/9**	**96/10**	**113/11**	**84/9**	**100/10**	**0–136/0–11**
Consumed more than planned	10/1	12/1	12/1	12/1	10/1	0–12/0–1
Unable to cut down or stop	12/1	12/1	14/1	12/1	12/1	0–16/0–1
Great deal of time spent	12/1	12/1	12/1	12/1	12/1	0–12/0–1
Important social, occupational, or recreational activities given up or reduced	6/0	10/1	12/1	8/0	12/1	0–16/0–1
Use despite physical/emotional consequences	8/1	8/1	8/1	8/1	8/1	0–8/0–1
Tolerance (marked increase in amount; marked decrease in effect)	8/1	6/1	8/1	8/1	8/1	0–8/0–1
Failure in role obligation (e.g., work, school, home)	0/0	4/0	8/1	4/1	6/1	0–8/0–1
Withdrawal	0/0	10/1	15/1	0/0	6/0	0–20/0–1
Use in physically hazardous situation	8/1	0/0	0/0	0/0	6/0	0–12/0–1
Use despite interpersonal/social consequences	8/1	8/1	8/1	6/1	6/1	0–8/0–1
Craving or a strong desire or urge to use	6/1	6/1	8/1	6/1	6/1	0–8/0–1
Impairment or distress	8/1	8/1	8/1	8/1	8/1	0–8/0–1
Binge Eating Scale	26	31	28	19	26	0–46
Cognitive manifestations	11	17	16	9	8	0–23
Behavioral manifestations	15	14	12	10	18	0–23

After analyzing the subscales of the YFAS, it was observed that men obtained a significantly lower score than women in concealment, as well as avoidance in social events.

When analyzing the BES subscales, it was observed that female patient B and female patient C obtained higher scores with respect to the cognitive manifestations. The remaining participants scored higher in the behavioral manifestations.

Table 4 shows the data that refers to the results observed according to the clinical psychologist in the individual interview and its relationship with diagnostic criteria of the DSM-V [1]. According to these data, it was observed that all the participants recognized having BED episodes at least once a week, and these were composed by large quantities of food consumed fast until feeling unpleasantly full. Male participants A and D were not ashamed of these binge eating episodes, and did not seek to perform them in private, as opposed to female participants A, B, and E.

Table 4. Diagnostic criteria met according to DSM-V after the interview.

	A	B	C	D	E
A1	XX	X	XX	X	X
A2	X	XX	X	X	X
A3	XX	XX	XX	XX	XX
A4	-	XX	XX	-	X
A5	-	XX	XX	-	XX
B	X	XX	XX	-	X
C	XX	XX	XX	XX	XX
D	X	X	X	XX	X

XX clearly fulfilled; x, partially fulfilled; - not fulfilled or insufficient information.

Table 5 shows the existence of polymorphisms associated with BED according to the literature, or the presence of binge eating in other pathologies related with eating disorders (obesity, AN, BN), in each of the participants. As shown, there is no repeated pattern in any of the participants, and therefore, the responses with respect to their physiological relationship with food should be different.

Table 5. Genetic polymorphisms detected in the participants.

	Case A	Case B	Case C	Case D	Case E
DRD2 rs1800497	No	Yes	No	No	Yes
FTO rs1558902 rs1421085 rs1121980 y rs9939609	Yes	Yes	No	No	No
MCR4 rs17782313	No	No	No	No	No
TAST1R2 rs12033832 rs35874116x	No	No	No	Yes	No
GLUT2 rs5400	Yes	No	Yes	No	No
TCF7L2 rs7903146	No	Yes	Yes	No	Yes
FABP2 rs1799883	Yes	No	No	No	No
CLOCK rs1801260	Yes	No	No	No	Yes

3.2. Case reports

Male participant A: Competition-level athlete and BED. With a BMI considered obesity type I.

Specific information with respect to eating behavior: Male participant A describes his eating behavior as healthy and disciplined when preparing for a competition, but

in the rest months, recognized developing a compulsive behavior towards food composed of refined flour, sweet in taste, and beer, either accompanied or alone, with a mean of 3 times per week. He performs intermittent fasting (from 20:00 to 11:00 the following day) as a method to lose weight and compensate for the binge eating performed, or the knowledge that they will occur during the week.

Main worries and symptoms of the patient: Male participant A commented that he was afraid of gaining weight in light of the competitions, but at the same time, he could not stop binge eating, and confessed that he was aware that this eating behavior was harmful for his health, as he sometimes substituted the meals planned for his sport (vegetable dish with rice and fish) for a "binge of carbohydrates, sweets, and beer". He also considered that this sports activity was addictive, and that during competition season, he "changed eating for running compulsively".

Medical, family, and psychosocial history including genetic information: Male patient A was diagnosed with Barret's esophagus in 2010, with a history of obesity until 2008, when he started to compete in Marathons. With respect to the preliminary diagnosis criteria (**Tables 3–5**), it is observed that he clearly meets the BED diagnostic criteria, i.e., the presence of recurring episodes of binge eating, as well as distress due to their occurrence (criteria B, C, and D, as well as 3 sub-criteria of the five from A). It must be noted that despite performing an intermittent fast, this was considered by the specialist as a routine practice, and not sporadic episodes or purging due to the binges. The YFAS scores were high in all the subscales analyzed, except for those related with the reductions in their social obligations and relationships due to their addiction. He obtained a BES score considered to be high. In the genetic study, polymorphisms were found related with the difficulty in reaching satiety and preference for products with a high content of simple sugars (**Table 5**).

Relevant past interventions and their results: Male patient A was never treated as having BED. Before 2008, when he suffered from obesity, he performed weight loss treatments, but they were never centered on working on anxiety for food. He never followed treatment with medication and/or guided by a psychologist.

Type of therapeutic intervention: A two-dimensional intervention (psychological and nutritional) was performed. At the psychological level, work was performed with cognitive behavioral therapy [15]. The recommendations at the nutritional level, based on his genetic profile, included planning of schedules and menus depending on his sporting activity, avoiding the reduction of calories especially before training and/or competitions, as well as compensating for the provision of nutrients for an adequate recovery after the training efforts. The practice of intermittent fasting was eliminated. In general, the consumption of foods with a medium or low glycemic index (GI) was encouraged, and those with a high GI were avoided to reduce binge eating episodes promoted by blood glucose imbalances. Also, the consumption of protein and foods rich in fiber in all the meals was increased, to work on the feeling of satiety (**Figure 1**). In general, the macronutrients consumed were: 50% CHO (32% polysaccharides and 18% sugars), 23% protein (16% animal and 7% plant), 27% fats (4% saturated, 6% polyunsaturated, and 17% monounsaturated). In addition, the level of micronutrients (**Table 6**), the consumption of foods rich in B vitamins, vitamin D, Calcium, Magnesium and Chromium was promoted, due to their involvement in the health of the nervous system, mental health, and the regulation of sugar levels in the

blood [39,40].

Table 6. Micronutrient recommendations based on genetic analysis. Case A, B, C, D, E.

	Case A	**Case B**	**Case C**	**Case D**	**Case E**
Nutrient	**Input (% Deviation*)**	**Input (% Deviation*)**	**Input (% Deviation*)**	**Input (% Deviation*)**	**Input (% Deviation*)**
Iodine (µg)	133.9 (−4.4)	123.5 (−11.8)	123.5 (−11.8)	133.9 (−4.4)	110.6 (−1.0)
Potasium (mg)	5086.1 (45.3)	4367.5 (24.8)	4367.5 (24.8)	5697.4 (62.8)	4611.7 (31.8)
Calcium (mg)	191.78 (91.8)	1904.3 (90.4)	1904.3 (90.4)	2262.5 (126.2)	2020.1 (102.0)
Magnesium (mg)	569 (intake >UL)	698 (intake > UL)	728.7 (intake > UL)	844.5 (intake > UL)	528 (intake > UL)
Phosphorus (mg)	2705 (286.5)	2622.8 (274.7)	2622.8 (274.7)	3046.2 (335.2)	2183.7 (212.0)
Iron (mg)	26.9 (169.3)	25.3 (152.9)	28 (179.6)	31.4 (214.5)	23.5 (30.4)
Selenium (mg)	202.3 (189)	95.8 (36.9)	105.3 (50.4)	115.5 (64.9)	98.2 (0.2)
Zinc (mg)	16.8 (76.7)	13.9 (46.5)	15.7 (65.8)	16.8 (76.7)	13.8 (15.1)
Vit. B1 (mg)	2.9 (143.1)	2.1 (87)	2.7 (128)	4.2 (283.5)	2.4 (96.7)
Vit. B2	2.6 (42.7)	3.2 (89)	3.3 (80.7)	3.0 (76.6)	2.3 (28.9)
Vit. B6	5 (176.2)	4 (120.4)	5 (175.8)	4.4 (144.7)	3 (69.4)
Vit. B12 (µg)	15 (649)	16.6 (730.1)	12.5 (526.7)	14.9 (645.7)	9.2 (358.7)
Folate (µg)	542 (35.5)	605 (51.3)	648.4 (62.1)	672.8 (68.2)	469.7 (17.4)
Niacin (mg)	52.8 (164.1)	508 (2573.9)	54.9 (174.4)	740.4 (3797.0)	31.8 (58.8)
Vit. C (mg)	253 (321.6)	175.1 (191.8)	305.9 (409.8)	248.4 (314.0)	202.1 (236.9)
Vit. A (µg)	1843.9 (84.4)	2194.7 (119.5)	2194.7 (119.5)	2980.9 (198.1)	2559.3 (155.9)
Vit. D (µg)	26.7 (78)	20.6 (37)	29.6 (97.1)	22.0 (46.6)	20.6 (37)
Vit. E (mg)	22.4 (86.8)	12.9 (7.7)	12.9 (7.7)	17.8 (48.4)	16.7 (4.3)

* Deviation from the Dietary Reference Intakes (DRIs) [21].

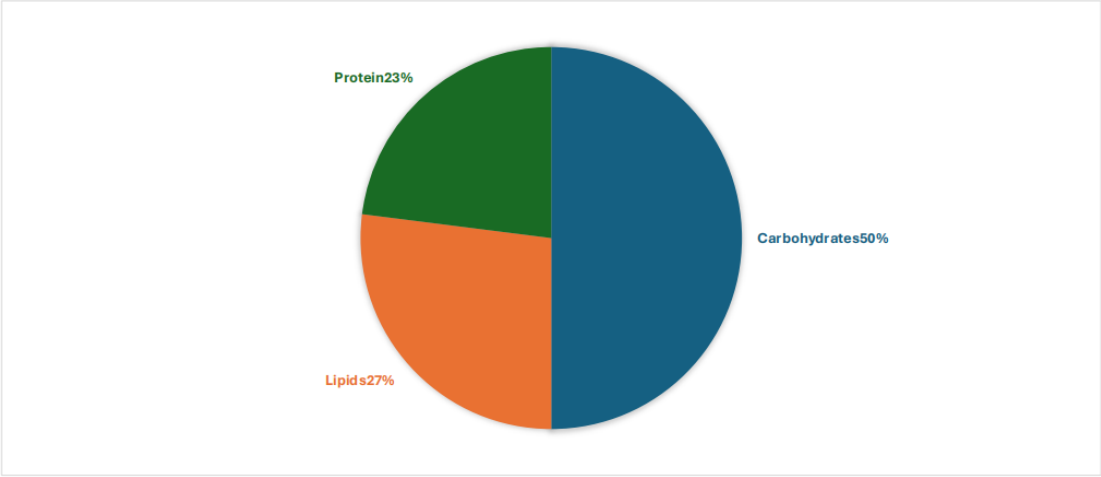

Figure 1. Macronutrient recommendations based on genetic analysis. case A.

Administration of the intervention (duration, dose): The intervention lasted 6 months, with follow-up and check-up consultations every 15 days. No changes were made during the intervention, as the results were positive. The timeline with respect to the changes in the number of binging episodes, as well as BMI (can be observed at the end of this section). The BMI figure shows the progressive reduction in BMI throughout the treatment, being more pronounced from month 5 to 6. In addition, the

binging episodes were reduced to zero in the fourth month, and remained so until the end of the treatment.

The assessment of male patient A with respect to the intervention was positive. The following quote can be highlighted: "at the start, I thought that I could still cheat with respect to the food, but after the first month, I observed that the anxiety for my favorite foods started to disappear, and now I save them for special occasions, and sometimes I don't even crave them". The adherence to the intervention was satisfactory, mainly starting in the third month, when the patient became aware of the need to become involved in the process of change and recognized the need for treatment after the interviews with the specialists.

No adverse or unforeseen events occurred during the treatment.

Female patient B: Active and BED. With a BMI considered obesity type II. Clinical history of depression and anxiety.

Specific information with respect to eating behavior: Female patient B describes her current eating behavior as "not very healthy". In the past few years, she followed various diets in order to lose weight, but "rebound effects" always took place, and they caused anxiety that led to an increase in binge eating episodes, and for this reason, had given up on "counting calories". Nevertheless, she recognizes "being afraid of carbohydrates", and due to this, tries to follow a diet low in carbohydrates. She recognizes binge eating any type of food, although she prefers fried foods and snacks, especially in the period before menstruation and in periods of work-related stress, and mainly seeks moments alone to perform them.

Main worries and symptoms of the patient: Female patient B, during the interview, recognized that she didn't care about weight anymore, and that all she needed was "to reduce the unpleasantness caused by the binge eating episodes". She also mentioned that she was worried about her physical and mental health. She openly described that she needed "a diet that makes her feel good inside and out".

Medical, family, and psychosocial history, including genetic information: Female patient B has diagnosed anxiety with intake of fluoxetine since two months ago. Also, she has been taking oral contraceptives in the last year. With respect to the preliminary diagnosis criteria (**Tables 3–5**), it is observed that she clearly meets the diagnostic criteria for BED, i.e., presence of recurring episodes of binge eating, as well as distress due to their occurrence and involvement in social relations (criteria A, B, C, and D). The YFAS scores were high in all the subscales analyzed. In the BES, a score considered to be very high was observed. In the genetic study, polymorphisms were observed, with these related with a difficulty to reach satiety, appetite, preference for products with a high fat content, and also, emotional regulation (reinforcement/reward stimuli) (**Table 5**).

Relevant past interventions and their results: Female participant B had never been treated for BED. As previously commented, she had followed many weight loss diets, but all of them based on caloric restriction, and some of the including avoidance/elimination of specific foods (mainly carbohydrates), but never centered on working on her anxiety for food. She is presently undergoing a psychiatric medical treatment to reduce her level of stress and anxiety, but not related with food.

Type of therapeutic intervention: a two-dimensional intervention was conducted (psychological and nutritional), in addition to the fluoxetine treatment, which had

already been prescribed. At the psychological level, work was performed with cognitive-affective therapy, with a main interest in the moments before and after the binge eating episodes, establishing recodification guidelines of the feeling of well-being through other means different than food (music, practice of meditation, and social relations) [15]. The therapeutic process began with a detailed assessment of the automatic emotions and thoughts that arose before binge eating, such as the belief that food was the only way to alleviate emotional distress. The therapist and the patient worked together to identify and question these cognitive patterns, teaching the patient to replace them with more adaptive thoughts. Cognitive replacement techniques were implemented, where the patient learned to substitute food with healthy alternatives, such as meditation, listening to music, or relaxation exercises, during moments of anxiety or stress. Additionally, elements of acceptance and commitment therapy were integrated, promoting the acceptance of emotions without the need to alter them through food, and encouraging self-compassion to reduce the self-criticism that often led to more binge eating. Therapy sessions lasted 50 min and were conducted every 15 days for six months. During this time, the patient kept a record of their emotions and responses to high-risk situations, allowing for the assessment of progress and the adjustment of therapeutic strategies as needed.

The recommendations at the nutritional level, based on her genetic profile, included the planning of schedules and menus, avoiding caloric restriction, and the promotion of the consumption of foods rich in monounsaturated fats (17% as compared to 5% of saturated fats, and 6% polyunsaturated fats), and proteins with satiety effects and rich in amino acids, with repercussions at the level of mental health (tryptophan, tyrosine, phenylalanine, and methionine) [41]. In general, the macronutrients provided were: 46% CHO (29% polysaccharides and 17% sugars, 26% proteins (19% animal and 7% plant), 28% fats (**Figure 2**). Also, with respect to the micronutrients (**Table 6**), the consumption of foods rich in B vitamins, vitamin D, Calcium, Magnesium, and Zinc were promoted, due to their considerable benefits at the level of the central nervous system [42,43].

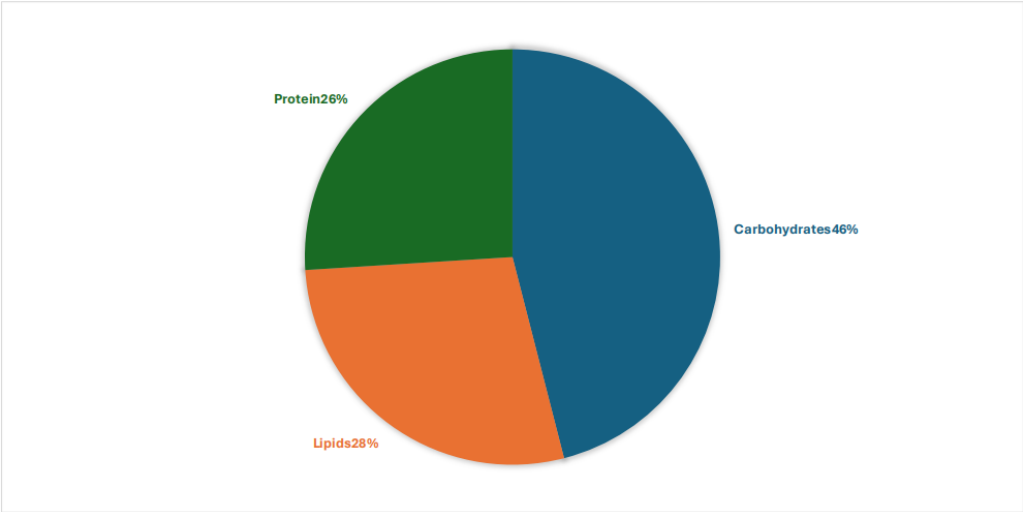

Figure 2. Macronutrient recommendations based on genetic analysis. case B.

Administration of the intervention (duration, dose): The intervention lasted 6

months, with follow-up and check-up consultations every 15 days. No changes were made during the intervention, as the results were positive. However, due to the improvement of the symptoms, the medication with Fluoxetine was reduced after 4 months of participation in the program (as indicated by the referred psychiatrist). The timeline with respect to the changes in the number of binge eating episodes, as well as the BMI (can be observed at the end of this section). These show that BMI was stable throughout the treatment. However, the binging episodes were reduced to zero in the fourth month, increasing to one a half per week in month 5 (perhaps influenced by the reduction in medication), to again decrease to zero in the last month.

The assessment by female participant B, with respect to the intervention, was positive, as indicated by the following quote: "I don't care that I didn't lose as much weight. I've observed that I no longer fear eating in public, and due to this, I'm enjoying my social life; learning how my body works at the physical and mental level, gives me strength to want it more. And, stopping the medication makes me have more self-esteem and with the capacity to be able to continue on this road by myself". The adherence to the intervention was satisfactory, and throughout the process, she was a very active and participative person.

No adverse or unforeseen events occurred during the treatment.

Female participant C: ex-athlete and BED.

Specific information with respect to eating behavior: Female patient C describes her current eating behavior as "healthy". She reports following a weight loss diet since she stopped competing, but without achieving the expected results on her figure. She recognized developing binge eating episodes once or twice a week, "sweet or salty", mainly at night when she is "relaxing at home".

Main worries and symptoms of the patient: Female participant C expressed that she feels uncomfortable with her current body, and "does not feel comfortable with the person she sees in the mirror". Also, she was worried for her health, due to the existence of a glaucoma (the reason why she left the sport in 2001), and therefore, she knows that the diet can help in preventing other derived illnesses. However, in spite of this, she cannot avoid her preference for those foods she calls "unhealthy".

Medical, family, and psychosocial history including genetic information: Female participant C reports coming from a family of overweight individuals. Since she was little, her family had always instilled the need to restrict eating "to not gain weight", and has the well-established idea about "good foods and bad foods", and "sometimes I would eat secretly from my family, I was hungry but they did not allow me to eat". She was diagnosed and underwent surgery for glaucoma 23 years ago. She is not presently taking any medication. With respect to preliminary diagnostic criteria (**Tables 3–5**), it is observed that she clearly meets the diagnostic criteria for BED, i.e., presence of recurring episodes of binge eating, and distress due to their occurrence and involvement in social relations (criteria A, B, C, and D). The YFAS scores were high in all the subscales analyzed. She also obtained a BES scored considered very high. In the genetic study, she showed polymorphisms related with the preference towards foods that are excessively sweet and fatty (**Table 5**).

Relevant past interventions and their results: Female participant C had never been treated for BED. During her childhood, she had food restrictions implemented by her family "for fear of becoming fat", and recognizes that the binge eating episodes began

during her period as an athlete, "which were camouflaged by excessive exercise" and that after the glaucoma operation, as she was not able to exercise, she began to gain weight, and was aware for the first time that she had a bad relationship with food. In the interview, she indicated many times that her relationship with food could be influenced by her family's behavior, especially her mother and aunt.

Type of therapeutic intervention: a two-dimensional intervention was conducted (psychological and nutritional). At the psychological level, work was conducted with cognitive-affective therapy [15] focused on identifying and modifying the thought patterns and emotions that preceded binge eating episodes. The process began with a detailed evaluation to detect dysfunctional beliefs, such as the idea that food was the only way to alleviate emotional distress. From there, the work focused on questioning these thoughts and replacing them with more adaptive ones. Cognitive restructuring techniques were used to replace food with healthy activities, such as meditation, listening to music, or relaxation exercises, during moments of anxiety or stress. Additionally, elements of Acceptance and Commitment Therapy (ACT) were integrated, helping the patient to accept her emotions without turning to food to modify them, promoting self-compassion and reducing self-criticism. Therapy sessions lasted 50 min and were conducted every 15 days for six months. During this period, the patient kept a record of her emotions and responses to high-risk situations, allowing adjustments to the therapeutic strategies as necessary. In addition to participation in the anonymous compulsive eaters organization (https://www.comedorescompulsivos.es/reuniones/presenciales/andalucia/) with the main objective being the elimination of the stigmatizing effect of overweightness she had inherited since her childhood. At the nutritional level, recommendations were given based on her genetic profile: an increase in daily food intakes to 7, to avoid episodes of prolonged fasting, an increase in the consumption of monounsaturated fats and carbohydrates (mainly polysaccharides, rich in fiber, and a medium to low GI to improve satiety). Caloric and food restrictions were avoided. In general, the macronutrients provided were (**Figure 3**): 55% CHO (34% polysaccharides and 21% sugars), 20% proteins (15% animal and 5% plant), 25% fats (15% monounsaturated, 5% saturated, and 5% polyunsaturated). Also, with respect to micronutrients (**Table 6**), the consumption of foods rich in B vitamins, Vitamin D, Calcium, Magnesium, Zinc, and Chromium were promoted, due to their considerable benefits to the central nervous system and regulation of the blood sugar levels [42,43].

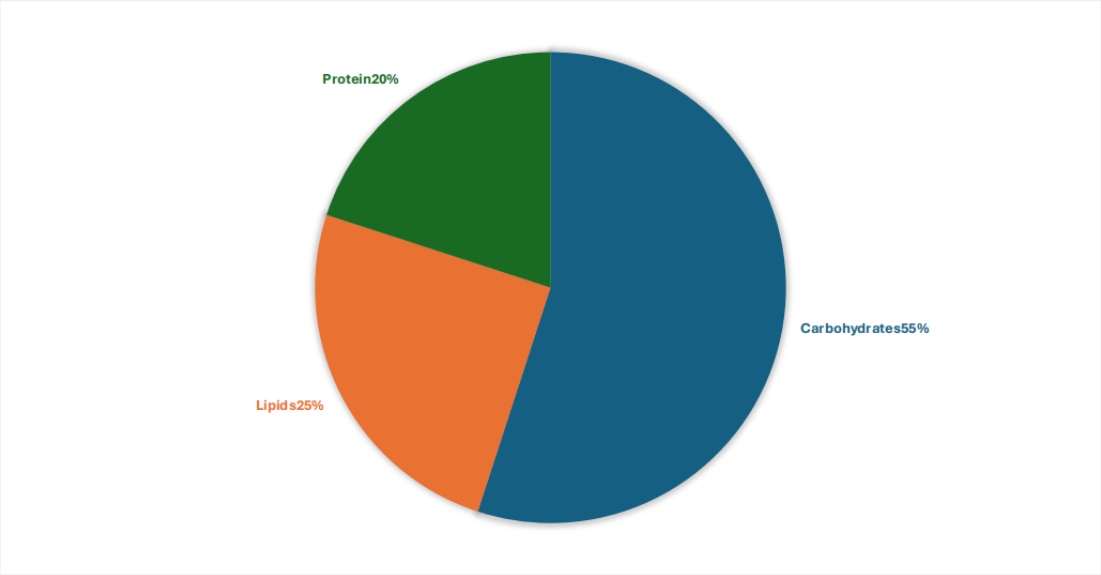

Figure 3. Macronutrient recommendations based on genetic analysis. case C.

Administration of the intervention (duration, dose): The intervention lasted 6 months, with follow-ups and check-up consultations every 15 days. No changes were made during the intervention as the results were positive. The timeline with respect to the changes in the number of binge eating episodes, as well as BMI (can be observed at the end of this section). It is observed that BMI progressively decreased until month 4, after which it remained stable, and with respect to the binge eating episodes, despite beginning with a high number of them (every day of the week), they decreased significantly until month 4 when they stopped, remaining stable until the end of the study.

The assessment of Female participant C of the intervention was positive, as described in the following quote: "I know that I have to keep on working on my relationship with food, I still believe that the binge eating episodes could come back. Discovering that there a genetic factor in me that incites me to eat like this makes me feel less guilty for not knowing how to control my impulses. Also, being part of a group of anonymous eaters have made me feel better about who I am and how I am little by little, so that losing weight did not become an objective, although I achieved it, …, I know that what I experienced in my childhood promoted what I could be predisposed for genetically."

Adherence to the intervention was satisfactory. No adverse or unforeseen events occurred during the intervention.

Male participant D: BED. Body building and a diet high in protein. With a BMI considered obesity type IV.

Specific information with respect to eating behavior: Male participant D describes his eating behavior as "normal", assuming that it is not something that specially worries him, and follows a diet centered on gaining muscle mass. The diet followed is hyperproteic, including 6 meals a day. Although he ensures not feeling hungry, he states that there are days when he needs "to eat a lot" without feeling satiated, and that this behavior is performed in the company of others or in private: "on some Saturdays, I have dinner with friends, they order a family-sized pizza for

three, and I order one for myself, I finish fast, and then, I continue eating whatever is around, nuts, sweets, snacks, ice-cream…I think I can eat more than 3000 calories without a problem in a short period of time".

Main worries and symptoms of the patient: Male patient D did not have worries with respect to his weight or the binge eating episodes, until a medical analysis reported high levels of cholesterol, triglycerides, and transaminases with respect to reference values. It was then that he began to think that the way he was eating may be harmful to his health.

Medical, family, and psychosocial history including genetic information: Male participant D has hypertriglyceridemia, high LDL cholesterol, and high levels of transaminases (diagnosed a year ago). With respect to preliminary diagnostic criteria (**Tables 3–5**), it is observed that he clearly meets the diagnostic criteria for BED, with respect to the number and frequency of the binge eating episodes, but not to the posterior distress (criteria A4, A5, and B). The YFAS score was the lowest from all the case studies analyzed, with scores of zero obtained in the subscales "Important social, occupational, or recreational activities given up or reduced", "Withdrawal" and "Use in physically hazardous situation". As for the BES, scores slightly higher than the cut-off value were obtained (cut-off value of 18, score of 19). In the genetic study, he only had a polymorphism of risk, the one related with difficulty for satiety, and problems in the detection of the sweet taste (**Table 5**).

Relevant past interventions and their results: Male participant D has never been treated for BED. He has not followed weight loss diets, despite suffering from obesity as a child until 2020, when he took part in an experimental study in which Saxenda was administered with the objective of losing weight. With respect to it, he states that: "I was the only participant who gained weight". After this, he started bodybuilding, and gained muscle mass, despite maintaining a similar weight.

Type of therapeutic intervention: A one-dimensional intervention (nutritional) was conducted. The recommendations at the nutritional level, based on his genetic profile, included scheduling and menus according to his sporting activity, avoiding caloric restriction, especially before training, as well as compensating the provision of nutrients for an adequate recovery after the training effort. In general, the consumption of foods with a medium and/or low glycemic index was promoted, and foods with a high GI were avoided in order to reduce the binge eating episodes promoted by the blood glucose imbalance. In addition, the consumption of protein and food rich in fiber was increased in all the meals, to work on the feeling of satiety (**Figure 4**). In general, the macronutrients provided were: 50% CHO (32% polysaccharides and 18% sugars), 17% proteins (10% animal and 7% plant), 33% fats (4% saturated, 5% polyunsaturated, and 20% monounsaturated). Also, at the level of micronutrients (**Table 6**), the consumption of foods rich in B vitamins, vitamin D, Calcium, Magnesium, and Chromium was promoted, due to their involvement in the health of the central nervous system, mental health, and regulation of blood sugar levels [42,43].

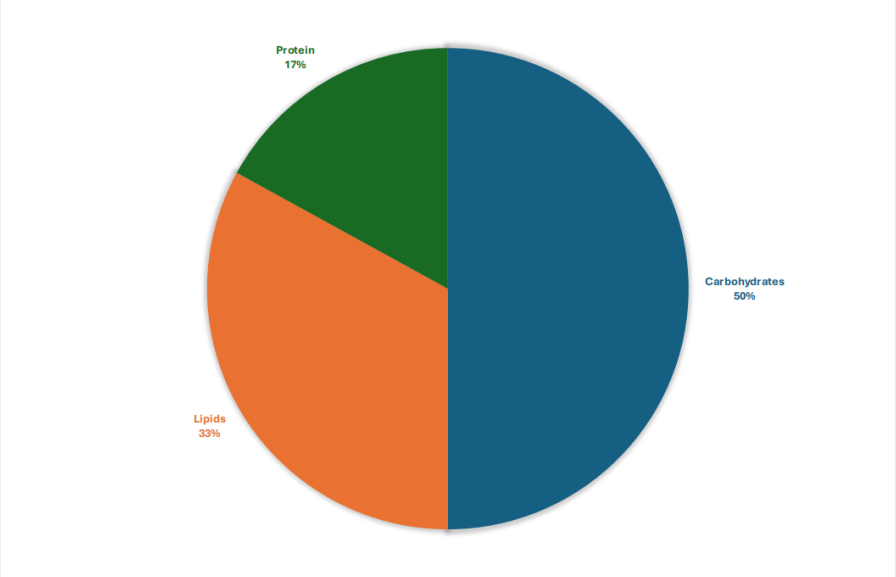

Figure 4. Macronutrient recommendations based on genetic analysis. case D.

Administration of the intervention (duration, dose): The intervention lasted 6 months, with follow-up and check-up consultations every 15 days. No changes were made during the intervention, as the results were positive. The timeline with respect to the changes in the number of binge eating episodes, as well as BMI (can be observed at the end of this section). The figures show that BMI was stable during the entire program, and with respect to the binge eating episodes, they significantly decreased until month 5, when they occurred 1 week per week in the last month on average.

The assessment of male participant D with respect to the intervention was positive, as shown by the following quote: "I noticed that with the nutritional planning, I don't have the need to eat in excess, I feel satiated, and I even perform better in my gym sessions, …, I like to eat, and on the weekends, I relaxed quite a bit in the last month".

Adherence to the intervention was satisfactory. No adverse or unforeseen events occurred during the intervention.

Female patient E: Sedentary and with BED. With a BMI considered obesity type I. Nocturnal compulsive eater.

Specific information with respect to eating behavior: Female participant E describes her eating behavior as "not very healthy". In the last few years, she has followed many diets, but without results. She recognizes that it is not difficult for her to "not eat for an entire day", but at night, independently of having eaten or not, anxiety takes over, and she "attacks the fridge", looking for foods that are excessively fatty, such as ice-creams and salty snacks. She currently has problems with her partner, and work makes it so that she has a "greater need" for binge eating.

Main worries and symptoms of the patient: Female participant E feels worried about her compulsive behavior towards food, especially because she says that she wakes up at night to eat compulsively. She also commented that she felt worried about her health, as the impossibility of losing weight makes it so that it affects her everyday life when trying to perform basic tasks such as cleaning or going to work.

Medical, family, and psychosocial history including genetic information: Female

participant E has a family history of obesity (parents and siblings), although she reports that: "they do not eat that much to be so fat". With respect to the preliminary diagnostic criteria (**Tables 3–5**), it is observed that she clearly meets the diagnostic criteria for BED, i.e., presence of recurring episodes of binge eating, and discomfort due to their occurrence and involvement in social relations (criteria A, B, C, and D). The YFAS scores were high in all the subscales analyzed, except for "Withdrawal", and "Use in physically hazardous situation". In the BES, she obtained a scored considered high. In the genetic study, she showed polymorphisms related with the difficulty for reaching satiety, appetite, preference for products high in fats, and also, emotional regulation and alteration of the circadian rhythm (**Table 5**).

Relevant past interventions and their results: Female participant E has never been treated for BED. As previously mentioned, she had followed weight loss diets, but all of them based on caloric restriction, but never centered on working on her anxiety for food.

Type of therapeutic intervention: a two-dimensional intervention (psychological and nutritional) was performed. At the psychological level, work was conducted with cognitive-behavioral therapy applied to the patient focused on managing nocturnal binge eating, establishing guidelines to improve the circadian rhythms, such as performing relaxation activities before nocturnal resting, and avoiding the use of mobile devices. The therapy began by identifying the triggers and patterns associated with the patient's late-night eating episodes. The therapeutic process included establishing guidelines to improve her circadian rhythms, such as performing relaxation activities before bedtime, like deep breathing exercises and progressive muscle relaxation. Additionally, the patient was encouraged to avoid using mobile devices or engaging in stimulating activities close to bedtime, as they could contribute to disrupted sleep patterns and emotional distress. Cognitive-behavioral therapy also included cognitive restructuring to challenge the beliefs and thoughts that led to the desire to binge eat at night. The therapist helped the patient develop coping strategies to manage stress or emotional discomfort without resorting to food. Therapy sessions lasted 50 minutes and were conducted every 15 days for six months. The patient was also asked to keep a daily log of her eating habits, sleep patterns, and emotional triggers, which allowed for continuous monitoring and adjustment of the therapeutic approach.

The recommendations at the nutritional level, based on her genetic profile, included the planning of schedules and menus, avoiding caloric restriction, as well as promoting the consumption of foods rich in tryptophan and magnesium, and reducing the intake of CHO with a high GI and fats to favor resting. In addition, with respect to the micronutrients (**Table 6**), the consumption of foods rich in B vitamins, Vitamin D, Calcium, Magnesium, and Zinc were promoted, due to their considerable benefits at the level of central nervous system [42,43]. In general, the macronutrients provided were: 49% CHO (33% polysaccharides and 16% sugars), 23% proteins (16% animal, and 7% plan), 28% fats (17% monounsaturated, 5% saturated, and 6% polyunsaturated) (**Figure 5**).

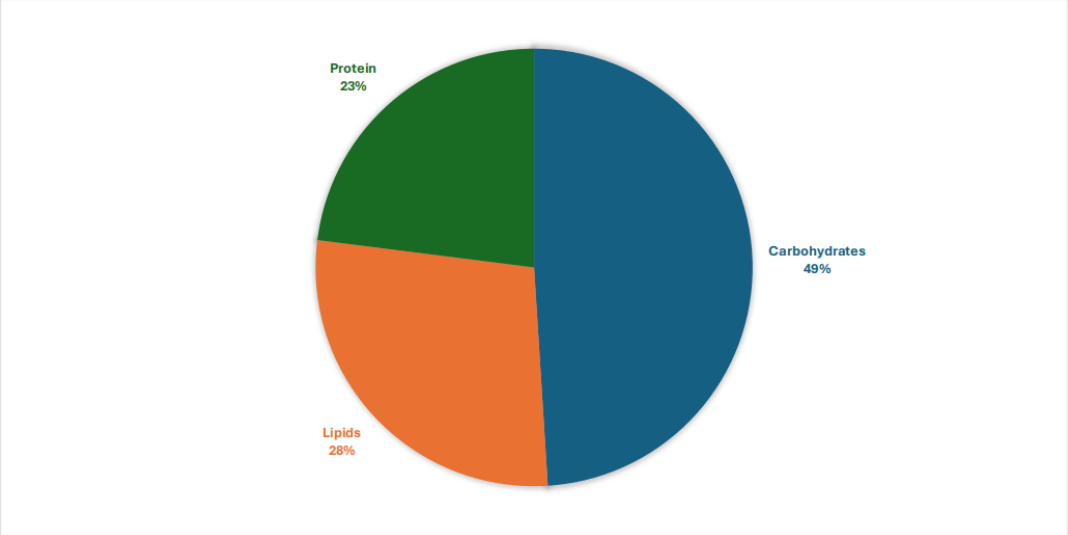

Figure 5. Macronutrient recommendations based on genetic analysis. case E.

Administration of the intervention (duration, dose): The intervention lasted 6 months, with follow-up and check-up consultations every 15 days. No changes were made during the intervention, as the results were positive. The timeline with respect to the changes in the number of binge eating episodes, as well as BMI, can be observed in **Figures 6** and **7** the evolution of all participants. The figures show that the BMI remained stable until month 5, when it slightly decreased. With respect to the binge eating episodes, they decreased from 4 to 2 a week from the first to the second month of the treatment, although they increased in the third month to then decrease to 2 during months 4 and 5, after which they were reduced to zero in the sixth month.

The assessment of female participant E with respect to the intervention was positive, as shown by the following quote: "sleeping at night without waking up to eat has improved my self-esteem, and makes me feel like following these guidelines. Good rest is important, as well as a good organization in feeding to avoid binge eating". Adherence to the intervention was satisfactory. No adverse or unforeseen events occurred during the intervention.

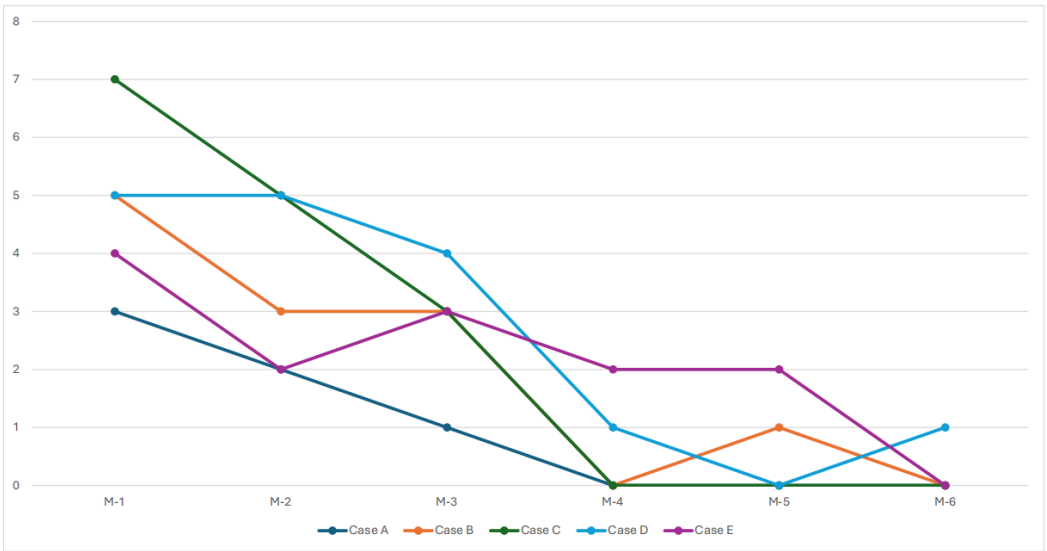

Figure 6. Change in the number of binge eating episodes per week during the treatment. Monthly measurement.

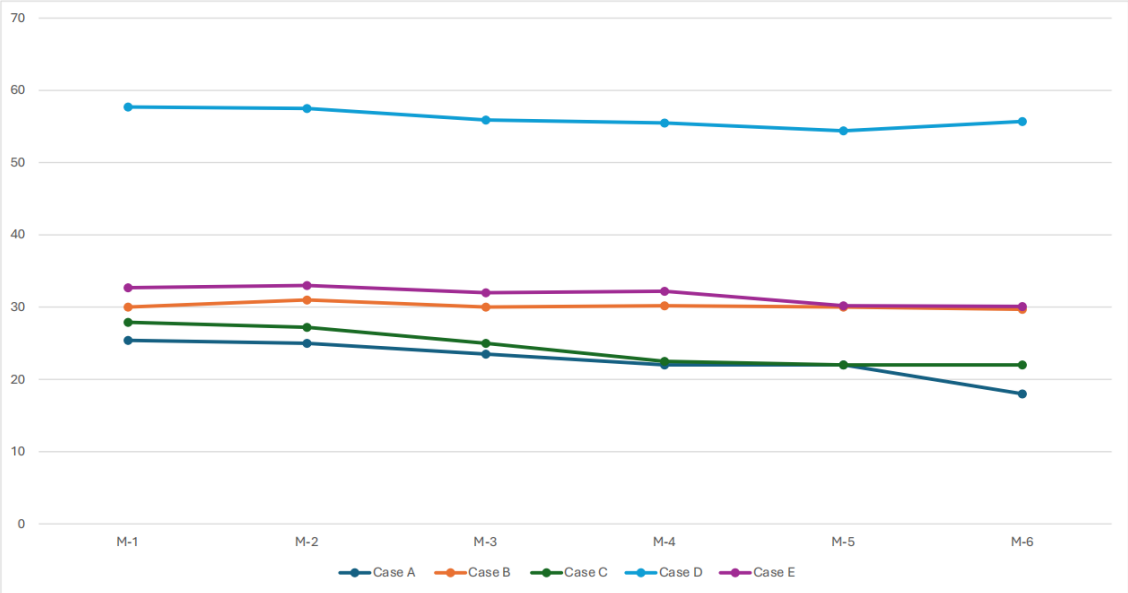

Figure 7. Changes in monthly BMI during the treatment.

4. Discussion

To determine whether BED (Binge Eating Disorder) should be addressed in a personalized manner based on genetic profiles that influence eating behavior and from a multidisciplinary perspective, it is essential not only to conduct empirical studies with large samples on the characteristic traits of BED-related eating behavior. Case studies are also necessary to understand the behavior of patients with BED. Given that no case series studies have been published addressing BED using preliminary diagnostic criteria, genetic analyses, and a multidisciplinary approach, the aim of this study was to present five case reports of patients with suspected BED, applying preliminary diagnostic criteria established in the DSM-5 [1] and performing genetic studies to tailor therapeutic interventions based on the individual needs of each patient. The cases presented aimed to demonstrate the variability in behavior and the need for personalized interventions based on the genetic profiles of patients with BED.

The main objective of the present study was to demonstrate the variability in the diagnosis and the possibilities of intervention of each patient with Binge Eating Disorder (BED), emphasizing the need for an interdisciplinary approach for its treatment. Despite the fact that none of the participants had been diagnosed with BED before their inclusion in their study, all of them showed feelings of guilt and confusion with respect to their relationship with food, attributing their binging to a supposed lack of self-control. This finding underlines the lack of visibility of this disorder in clinical practice, where many individuals mistake their dysfunctional eating behaviors with problems of willpower, which can result in a significant delay in the access to adequate care [44,45].

4.1. Lack of visibility and confusion with feeding control

The lack of a previous diagnosis of the participants is a common phenomenon among those who have BED: the erroneous belief that their eating problems are simply the result of a lack of self-control. This misunderstanding is perpetuated by social and

cultural stigmas, which on many occasions portray people who battle their eating habits as weak in character or lacking discipline [46]. This erroneous perception can result in individuals experiencing an additional emotional load, intensifying their feelings of guilt and shame related with their eating behavior. The social pressure of adhering to the ideals of a thin body, and the culture of dieting also contributes to this confusion, where BED is often misinterpreted as a simple desire to "not wanting to stop eating" [47].

4.2. Gender differences in the diagnosis and eating behavior

The results from the study show that women obtained higher scores than men in the Yale Food Addition Scale (YFAS). In contrast, men obtained lower scores in aspects related to concealment and avoidance of social events. This finding suggests that men can experience BED in a manner that it allows them to be more open about their behavior, while women tend to feel a stronger social pressure to conceal their binging [48].

The participants (men) A and D did not show shame in binging in public, while they women did so, which can be explained by the social pressure faced by women in terms of body image and eating behaviors [48]. Gender stereotypes can have an influence on how each gender perceives and faces their eating problems, where women are often socialized to be more aware about their body image, which results in feelings of shame and concealment [49].

4.3. Need for personalized and interdisciplinary treatments

The variability of clinical and psychosocial patterns observed among participants reinforces the importance of personalizing treatments. Participants (women) B and C obtained higher scores in the cognitive dimensions, while on the remaining participants, higher scores were observed in the behavioral dimensions. This suggests that the interventions must address both behavioral manifestations and psychological aspects of the disorder. The literature supports this need, emphasizing that a comprehensive approach that combines psychological, psychiatric, and nutritional therapies can be more effective in the personalization of each treatment for each individual [50,51]. In addition, the interventions must be tailored to the individual differences to maximize their efficacy, considering the variations in the symptomology observed [15,45].

An interdisciplinary approach is crucial in the treatment of Binge Eating Disorder (BED), given that this disorder not only involves biological factors, but also psychological, social, and environmental aspects. Although cognitive-behavioral therapy (CBT) has been shown to be efficient in the reduction of binge eating episodes, some studies indicate that the complete remission of the disorder cannot be achieved in many cases, given the interaction of multiple factors that lead to the maintenance of BED [4]. This highlights the need for a more comprehensive approach that also considers the social and emotional context of the patient.

Various studies have found that CBT is effective in the short term to reduce the number of binge eating episodes and improve general well-being, but it cannot always address the underlying factors that perpetuate the disorder, such as chronic stress,

family dysfunction, past traumatic events, social support, or cultural pressures [15,51]. For example, traumatic life events, such as child abuse, have been identified as common triggers of eating disorders, which suggests that the treatment must address not only eating behaviors, but also the emotional consequences of these experiences [50].

The integration of the management of eating with psychological therapy in the treatment for Binge Eating Disorder (BED) has shown to be essential for improving clinical results, especially in patients with dysfunctional eating patterns and emotional difficulties. The study by Dalle Grave et al. [50] highlights the efficacy of the "Enhanced Cognitive Behavioral Therapy" (E-CBT), which combines a specific nutritional intervention with addressing dysfunctional beliefs about eating and the body, achieving sustained improvements in the results of patients with BED [3].

One of the crucial aspects of current diet-based interventions is to ensure an adequate and balance energy state to prevent binge eating, which on many occasions can be related with nutritional deficiencies, of both macronutrients and micronutrients. The lack of essential nutrients, such as in restrictive or unbalanced diets, can lead to a metabolic imbalance that increases the propensity towards binge eating episodes [4].

Nevertheless, nutritionists are not always considered an integral part of the multidisciplinary team for the treatment of BED. The interventions tend to be monopolized by psychologists and psychiatrists, who push aside the relevance of the management of nutrition, limiting the potential impact of a truly comprehensive treatment. This exclusion could be due to the perception that the disorder is principally psychological, ignoring the fact that eating plays a crucial role in the triggering of the binge eating episodes, as well as the general improvement of the patient [15]. In fact, many studies have highlighted the need to include nutritionists in the treatment team, as their contribution could be a determinant factor in the avoidance of energy shortages that perpetuate the cycles of binge eating and shame [51]. In addition, as the present study shows, the nutritional approach must further evolve and work within a precise approach to tailor the intake not only to the general needs, but according to the genetic predispositions. In this sense, "precision nutrition" is a promising field that could significantly improve BED treatment, especially in those cases in which traditional approaches are not able to completely eliminate the binge eating episodes. Integrating nutrigenetics in meal planning will allow creating more personalized diets that take into account not only the caloric needs of patients, but also their genetic predisposition to the development of dysfunctional eating behaviors as a response to certain types of foods [52].

4.4. Analysis of the cases and genetic considerations

The analysis of each case in the study reveals unique patterns that reinforce the idea that a single model of BED does not exist.

In Case A (genetic predisposition to foods high in sugar content and high glycemic index), it was observed that the genetic predisposition to foods high in sugars and simple carbohydrates could have an influence on the manifestation of BED, in agreement with studies that associated genetics with eating behavior [53]. This relationship highlights the importance of performing genetic assessments to identify

predisposing factors, which could ease the creation of personalized interventions that address biological and behavioral triggers of the disorder [54]. In addition, the relationship between the lack of macro and essential micronutrients, due to the practices of intermittent fasting and long training cycles, could illustrate binge eating behavior that emerges as a response to extreme dietary restrictions. The literature suggests that intermittent fasting can increase the risk of binge eating episodes, especially in people predisposed to a ED [55]. These findings underline the need to address not only the eating behavior, but also the physical and nutritional health of the patients, as part of comprehensive treatment.

In Case B (predisposition in dopaminergic genes, satiety, and preference for fatty foods), it was evidenced that the emotional load and personal discomfort pushed female participant B to seek comfort in food. This finding is consistent with the theory that BED is often manifested as a coping mechanism when dealing with stressful or emotional situations [49]. In this case, the combination of cognitive-affective therapy and nutritional planning helped female participant B to improve her relationship with food and herself, reaffirming the importance of addressing the emotional dimension in the treatment of BED.

Case C (genetic predisposition towards foods rich in simple CHO, and sweet and fatty foods) highlighted how the family environment can incite binge eating from an early age. This observation reinforces the importance of considering social and environmental factors in the manifestation of BED. Various studies have documented that a dysfunctional family environment can contribute to the appearance of eating disorders, suggesting that social and family support can be fundamental for recovery [56]. In this context, it has been proposed that the inclusion of group therapies and joint sessions with others who are facing similar problems can offer a crucial support for those who suffer from BED.

Case D (genetic predisposition to sweet foods, as well as difficulty metabolizing them) stands out for his high body mass index (BMI), and relatively low scores in the YFAS (Yale Food Addition Scale) and BES (Binge Eating Scale), despite his condition. These scores suggest a lower involvement of factors related with addiction to food or the severity of the binge eating episodes, which is aligned to the inherent complexity of Binge Eating Disorder (BED). In agreement with previous studies, BED is still under active investigation, particularly with respect to the identification of specific genetic polymorphisms. The GWAS and BEGIN studies have indicated a genetic predisposition to BED, but no definite conclusions have been drawn about the exact genes that have an influence on the onset and course of the disorder [57]. In the case of subject D, the lack of a strong correlation between his genetic polymorphisms and the severity of the binge eating episodes suggests that there could be other variables aside from the genetic ones that have an influence on the development of the disorder. At the emotional level, the patient does not have significant factors that may have an influence on his binge eating episodes, which is unusual, given that most of the patients with BED experience some type of emotional distress before or after the episodes [50]. In addition, he did not show shame when binge eating in public, which contrasts with what has been observe in many studies about the behaviors of individuals with BED, especially among women, who tend to feel a greater social pressure and shame with respect to eating behavior [57].

As for the treatment, the approach followed by patient D was exclusively centered on nutritional planning. This type of intervention has shown improvements until a certain point, achieving a significant reduction in the number of binge eating episodes in the first five months. However, during the sixth month, the patient recognized having at least one binge eating episode per week. This pattern suggests that although the nutritional intervention was useful, it was not sufficient for completely eliminating the binge eating episodes. In this context, it is important to underline that nutrition, even though it is a fundamental pillar in the treatment of BED, by itself it is insufficient for resolving the disorder without addressing the underlying psychological factors [15].

The intervention in Case E (dopamine, fats, and alteration of the circadian rhythm), in which binge eating episodes were primarily observed at night, underlines the relationship between the circadian rhythm and eating behaviors, sleeping disorders are becoming increasingly associated with eating problems, suggesting that the improvement in sleeping patterns can be an effective strategy for reducing the binge eating episodes [58]. The efficacy of the guidelines implemented to improve sleep patterns and avoid waking up at night is aligned with previous studies [38].

4.5. Implications for clinical practice

The findings of the present study emphasize the importance of adopting an interdisciplinary approach in the treatment of BED. The integration of genetic assessment, psychological therapy, and nutritional planning, can not only personalize the treatment, but also improve the long-term results of the patients. Also, the attention given to psychosocial and emotional factors is crucial for helping individuals develop a healthier relationship with food and with themselves.

Health professionals must be capacitated to recognize BED signs and symptoms, even those that do not fit the typical stereotype. Continuous education on the disorder and its manifestations can help improve early detection and access to an adequate treatment. Likewise, it is vital to promote a greater awareness about the existence of this disorder, to reduce stigmatization and to promote an environment of support for those who suffer from it.

4.6. Strengths

The present study is an innovation in the field of BED, as it is the first to systematically integrate the assessment of individuals with different profiles through a combination of semi-structured interviews, established diagnostic criteria, and genetic analyses. According to previous studies, it is estimated that up to 40% of the predisposition to BED is associated with genetic factors [13]. Despite the GWAS studies identifying some polymorphisms associated with BED, the genetic relationship has not been conclusively established, and more research is needed to obtain firm conclusions of the genetic influence on the appearance and progression of the pathology [8].

In all the cases assessed in the present study, despite the differences in the diagnosis and treatment approaches, it was concluded that an adequate therapeutic management of BED must be necessarily integrate the genetic information of the

patient, as certain predisposition factors can have an influence on the severity and course of the disorder. In addition, it is crucial to count with an exhaustive analysis of the psychosocial situation of the patient, both past and present, to identify triggering factors that could have an influence on the appearance of binge eating episodes.

4.7. Limitations

The nutritional planning was based on the references from the related scientific literature. In the future, as more information about polymorphisms and BED is obtained, more specific plans will be made.

It is still unknown if the binge eating results will be stable, or if they will reappear after the study. Despite providing information and guidelines for the patients to independently follow, it is still unknown if the reduction in the binge eating episodes can be produced by continuous follow-up with a specialist. Further follow-ups must be performed to be able to observe these results in the future.

The case series study implies that inferential statistical analysis cannot be performed nor generalizations to a broader population. This type of study focuses on the detailed observation of individual cases and does not allow for statistical inferences that extrapolate the results to other groups and/or contexts.

5. Conclusions

- Nutrigenetic interventions are found to be fundamental as a co-adjuvant of psychological therapies.
- Nutritional interventions provide nutrients that complete the nutritional profile of patients and prepares them at the physiological level for a better therapy at the psychological level.
- Knowledge about the genetic profile of patients and their predisposition towards a type of food is necessary.

Author contributions: Conceptualization, MMH and LGP; methodology, MMH, LGP and MJGM; software, DSG and JTP; validation, MMH, LGP, DSG and JTP; formal analysis, MMH and LGP; investigation, MMH, LGP and MJGM; resources, MMH and GAM; data curation, LGP and GAM; writing—original draft preparation, MMH and LGP; writing—review and editing, MJGM and GAM; visualization, DSG; supervision, LGP and GAM; project administration, MMH and LGP; funding acquisition, DSG and JTP. All authors have read and agreed to the published version of the manuscript.

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Availability of data and materials: All data regarding the participants' results are included in the manuscript or as a supplementary document.

Consent for publication: This research has received consent for publication from the five participants involved in the study. Signed consent forms from each participant are attached.

Ethical approval: The study was conducted in accordance with the Declaration of Helsinki, and approved by the Ethics Committee of the University of Almería (Ref: UALBIO2024/022). All participants signed an informed consent form in compliance with current ethical and legal regulations. Strict procedures were followed to ensure the confidentiality and privacy of the collected data.

Conflict of interest: The authors declare no conflict of interest.

Abbreviations

DRD2	Dopamine Receptor D2
FTO	Fat mass and Obesity associated gene
MC4R	Melanocortin 4 Receptor
TAS1R2	Taste receptor type 1 member 2
GLUT2	Glucose transporter type 2
TCF7L2	Transcription factor 7-like 2
FABP2	Fatty Acid Binding Protein 2
CLOCK	Clock circadian regulator
BMI	Body Max Index
ED	Eating Disorder
BED	Binge Eating disorder
AN	Anorexia Nervosa
BN	Bulimia Nervosa

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Appendix A

GEN	FUNCIÓN	POLIMORFISMO		GENOTIPOS DE RIESGO	SU GENOTIPO
ADRB2	Termogénesis, lipólisis y β-oxidación	Rs1042713/ Rs1042714	Haplotipo	GG/GG, GG/CG, GA/GG, GA/CG	GG/CG
ADRB3	Termogénesis, lipólisis y β-oxidación	Rs4994		CC, TC	TT
APOA2	Transporte de grasas	Rs5082		CC	TT
APOA5	Transporte de grasas	Rs662799		CC, TC	TT
CLOCK	Ritmo circadiano	Rs1801260		CC, TC	TC
DRD2	Gula	Rs1800497		AA, GA	GG
FABP2	Captación de grasas, estrés oxidativo e inflamación	Rs1799883		AA, GA	GA
FTO	Detección de proteínas	Rs9939609		AA, TA	TA
GLUT2	Detección de azúcares	Rs5400		TT, CT	CT
MC4R	Detección de grasas, termogénesis y lipólisis	Rs17782313		GG, AG	AA
PPARG	Adipogénesis, termogénesis y lipólisis	Rs1801282		GG, CG	CC
TAS1R2	Detección de azúcares	Rs12033832/ Rs35874116	Haplotipo	CC/AA, CT/AA	CC/AG
TCF7L2	Detección de grasas y adipogénesis	Rs7903146		TT, CT	CC
TNFA	Estrés oxidativo e inflamación	Rs1800629		AA, GA	GG
PLIN1[1]	Termogénesis y lipólisis, β-oxidación, estrés oxidativo e inflamación	Rs2289487/ Rs894160	Haplotipo	AA/AA, AA/GA AA/GG	GA/GA
PLIN1[2]	Termogénesis y lipólisis, ritmo circadiano, β-oxidación, estrés oxidativo e inflamación	Rs1052700		TT	TA

LEYENDA

En rojo y amarillo se destaca el resultado de su genotipo asociado a los genes y polimorfismos analizados.

Homocigoto: 2 alelos sin riesgo

Heterocigoto: 1 alelo sin riesgo y 1 alelo de riesgo

Homocigoto: 2 alelos de riesgo

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Figure A1. Genetic report Mr.A.

Appendix B

GEN	FUNCIÓN	POLIMORFISMO		GENOTIPOS DE RIESGO	SU GENOTIPO
ADRB2	Termogénesis, lipólisis y β-oxidación	Rs1042713/ Rs1042714	Haplotipo	GG/GG, GG/CG, GA/GG, GA/CG	GA/CC
ADRB3	Termogénesis, lipólisis y β-oxidación	Rs4994		CC, TC	TT
APOA2	Transporte de grasas	Rs5082		CC	TT
APOA5	Transporte de grasas	Rs662799		CC, TC	TT
CLOCK	Ritmo circadiano	Rs1801260		CC, TC	TT
DRD2	Gula	Rs1800497		AA, GA	GG
FABP2	Captación de grasas, estrés oxidativo e inflamación	Rs1799883		AA, GA	GG
FTO	Detección de proteínas	Rs9939609		AA, TA	TA
GLUT2	Detección de azúcares	Rs5400		TT, CT	CC
MC4R	Detección de grasas, termogénesis y lipólisis	Rs17782313		GG, AG	AA
PPARG	Adipogénesis, termogénesis y lipólisis	Rs1801282		GG, CG	CC
TAS1R2	Detección de azúcares	Rs12033832/ Rs35874116	Haplotipo	CC/AA, CT/AA	CT/AG
TCF7L2	Detección de grasas y adipogénesis	Rs7903146		TT, CT	CT
TNFA	Estrés oxidativo e inflamación	Rs1800629		AA, GA	GA
PLIN1[1]	Termogénesis y lipólisis, β-oxidación, estrés oxidativo e inflamación	Rs2289487/ Rs894160	Haplotipo	AA/AA, AA/GA AA/GG	GA/GG
PLIN1[2]	Termogénesis y lipólisis, ritmo circadiano, β-oxidación, estrés oxidativo e inflamación	Rs1052700		TT	TA

LEYENDA

En rojo y amarillo se destaca el resultado de su genotipo asociado a los genes y polimorfismos analizados.

Homocigoto: 2 alelos sin riesgo

Heterocigoto: 1 alelo sin riesgo y 1 alelo de riesgo

Homocigoto: 2 alelos de riesgo

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Figure B1. Genetic report Ms.B.

Appendix C

GEN	FUNCIÓN	POLIMORFISMO		GENOTIPOS DE RIESGO	SU GENOTIPO
ADRB2	Termogénesis, lipólisis y β-oxidación	Rs1042713/ Rs1042714	Haplotipo	GG/GG, GG/CG, GA/GG, GA/CG	GG/GG
ADRB3	Termogénesis, lipólisis y β-oxidación	Rs4994		CC, TC	TT
APOA2	Transporte de grasas	Rs5082		CC	TC
APOA5	Transporte de grasas	Rs662799		CC, TC	TT
CLOCK	Ritmo circadiano	Rs1801260		CC, TC	TT
DRD2	Gula	Rs1800497		AA, GA	GG
FABP2	Captación de grasas, estrés oxidativo e inflamación	Rs1799883		AA, GA	GG
FTO	Detección de proteínas	Rs9939609		AA, TA	TT
GLUT2	Detección de azúcares	Rs5400		TT, CT	CT
MC4R	Detección de grasas, termogénesis y lipólisis	Rs17782313		GG, AG	AA
PPARG	Adipogénesis, termogénesis y lipólisis	Rs1801282		GG, CG	CC
TAS1R2	Detección de azúcares	Rs12033832/ Rs35874116	Haplotipo	CC/AA, CT/AA	TT/AG
TCF7L2	Detección de grasas y adipogénesis	Rs7903146		TT, CT	TT
TNFA	Estrés oxidativo e inflamación	Rs1800629		AA, GA	GG
PLIN1[1]	Termogénesis y lipólisis, β-oxidación, estrés oxidativo e inflamación	Rs2289487/ Rs894160	Haplotipo	AA/AA, AA/GA AA/GG	AA/GG
PLIN1[2]	Termogénesis y lipólisis, ritmo circadiano, β-oxidación, estrés oxidativo e inflamación	Rs1052700		TT	TA

LEYENDA

En rojo y amarillo se destaca el resultado de su genotipo asociado a los genes y polimorfismos analizados.

Homocigoto: 2 alelos sin riesgo

Heterocigoto: 1 alelo sin riesgo y 1 alelo de riesgo

Homocigoto: 2 alelos de riesgo

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Figure C1. Genetic report Ms.D.

Appendix D

GEN	FUNCIÓN	POLIMORFISMO		GENOTIPOS DE RIESGO	SU GENOTIPO
ADRB2	Termogénesis, lipólisis y β-oxidación	Rs1042713/ Rs1042714	Haplotipo	GG/GG, GG/CG, GA/GG, GA/CG	AA/CC
ADRB3	Termogénesis, lipólisis y β-oxidación	Rs4994		CC, TC	TT
APOA2	Transporte de grasas	Rs5082		CC	TC
APOA5	Transporte de grasas	Rs662799		CC, TC	TT
CLOCK	Ritmo circadiano	Rs1801260		CC, TC	TC
DRD2	Gula	Rs1800497		AA, GA	GA
FABP2	Captación de grasas, estrés oxidativo e inflamación	Rs1799883		AA, GA	GG
FTO	Detección de proteínas	Rs9939609		AA, TA	TT
GLUT2	Detección de azúcares	Rs5400		TT, CT	CC
MC4R	Detección de grasas, termogénesis y lipólisis	Rs17782313		GG, AG	AA
PPARG	Adipogénesis, termogénesis y lipólisis	Rs1801282		GG, CG	CG
TAS1R2	Detección de azúcares	Rs12033832/ Rs35874116	Haplotipo	CC/AA, CT/AA	TT/AA
TCF7L2	Detección de grasas y adipogénesis	Rs7903146		TT, CT	TT
TNFA	Estrés oxidativo e inflamación	Rs1800629		AA, GA	GA
PLIN1[1]	Termogénesis y lipólisis, β-oxidación, estrés oxidativo e inflamación	Rs2289487/ Rs894160	Haplotipo	AA/AA, AA/GA AA/GG	AA/GG
PLIN1[2]	Termogénesis y lipólisis, ritmo circadiano, β-oxidación, estrés oxidativo e inflamación	Rs1052700		TT	TT

LEYENDA

En rojo y amarillo se destaca el resultado de su genotipo asociado a los genes y polimorfismos analizados.

Homocigoto: 2 alelos sin riesgo

Heterocigoto: 1 alelo sin riesgo y 1 alelo de riesgo

Homocigoto: 2 alelos de riesgo

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Figure D1. Genetic report Mr.D.

Appendix E

GEN	FUNCIÓN	POLIMORFISMO		GENOTIPOS DE RIESGO	SU GENOTIPO
ADRB2	Termogénesis, lipólisis y β-oxidación	Rs1042713/ Rs1042714	Haplotipo	GG/GG, GG/CG, GA/GG, GA/CG	GA/CC
ADRB3	Termogénesis, lipólisis y β-oxidación	Rs4994		CC, TC	TT
APOA2	Transporte de grasas	Rs5082		CC	TC
APOA5	Transporte de grasas	Rs662799		CC, TC	TT
CLOCK	Ritmo circadiano	Rs1801260		CC, TC	TT
DRD2	Gula	Rs1800497		AA, GA	GG
FABP2	Captación de grasas, estrés oxidativo e inflamación	Rs1799883		AA, GA	GG
FTO	Detección de proteínas	Rs9939609		AA, TA	TT
GLUT2	Detección de azúcares	Rs5400		TT, CT	CC
MC4R	Detección de grasas, termogénesis y lipólisis	Rs17782313		GG, AG	AA
PPARG	Adipogénesis, termogénesis y lipólisis	Rs1801282		GG, CG	CC
TAS1R2	Detección de azúcares	Rs12033832/ Rs35874116	Haplotipo	CC/AA, CT/AA	CC/AA
TCF7L2	Detección de grasas y adipogénesis	Rs7903146		TT, CT	CC
TNFA	Estrés oxidativo e inflamación	Rs1800629		AA, GA	GG
PLIN1[1]	Termogénesis y lipólisis, β-oxidación, estrés oxidativo e inflamación	Rs2289487/ Rs894160	Haplotipo	AA/AA, AA/GA AA/GG	GA/GA
PLIN1[2]	Termogénesis y lipólisis, ritmo circadiano, β-oxidación, estrés oxidativo e inflamación	Rs1052700		TT	TA

LEYENDA

En rojo y amarillo se destaca el resultado de su genotipo asociado a los genes y polimorfismos analizados.

Homocigoto: 2 alelos sin riesgo

Heterocigoto: 1 alelo sin riesgo y 1 alelo de riesgo

Homocigoto: 2 alelos de riesgo

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Figure E1. Genetic report Ms.E.