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RESEARCH ARTICLE

# Eating disorders in narcolepsy type 1: Evidence from a cross-sectional Italian study

Valentina Baldini[1,2] | Noemi Venezia[1] | Anna Iriti[1] | Silvia Quattrocchi[1] | Corrado Zenesini[3] | Francesco Biscarini[1] | Anna Rita Atti[1] | Marco Menchetti[1] | Christian Franceschini[4] | Giorgia Varallo[2] | Diana De Ronchi[1] | Giuseppe Plazzi[2,3] | Fabio Pizza[1,3]

[1]Department of Biomedical and Neuromotor Sciences, University of Bologna, Bologna, Italy

[2]Department of Biomedical, Metabolic and Neural Sciences, University of Modena and Reggio Emilia, Modena, Italy

[3]IRCCS Istituto Delle Scienze Neurologiche di Bologna (ISNB), Bologna, Italy

[4]Department of Medicine and Surgery, University of Parma, Parma, Italy

**Correspondence**
Giuseppe Plazzi, IRCCS Istituto delle Scienze Neurologiche di Bologna, Via Altura 3, 40139 Bologna, Italy.
Email: giuseppe.plazzi@isnb.it

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## Summary

Narcolepsy type 1 is a chronic central disorder of hypersomnolence, and it is frequently accompanied by overweight, but the association between narcolepsy type 1 and eating disorders is controversial. Our study aims to compare patients with narcolepsy type 1 and controls on the symptomatology of eating disorders and to evaluate the association between clinical factors. This is a cross-sectional study, with consecutive recruitment of patients with narcolepsy type 1 attending the Outpatient Clinic for Narcolepsy at the IRCCS Istituto delle Scienze Neurologiche di Bologna (Italy) for routine follow-up visits. Healthy subjects from general populations were recruited as controls. Patients underwent a questionnaire-based assessment using the Eating Disorder Examination Questionnaire (EDE-Q), Binge Eating Scale (BES), Italian Night Eating Questionnaire (I-NEQ), Epworth Sleepiness Scale (ESS), and Narcolepsy Severity Scale (NSS). One hundred and thirty-eight patients with narcolepsy type 1 and 162 controls were enrolled. This study showed that individuals with narcolepsy type 1 reported higher scores on the EDE-Q, I-NEQ, and a higher body mass index (BMI) than the controls. The logistic regression analysis results, with EDE-Q positivity as a dependent variable, demonstrate a significant association with antidepressant drugs, female sex, and the use of sodium oxybate. We found an association between antidepressant drug consumption, the NSS total score, and female sex with BES positivity as the dependent variable. The logistic regression analysis for I-NEQ positivity found an association with antidepressant drug use. This study shows that patients with narcolepsy type 1 frequently present with comorbid eating disorder symptomatology, mainly night eating syndrome. Investigating the possible presence of eating disorders symptomatology through questionnaires is fundamental during the assessment of patients with narcolepsy type 1.

KEYWORDS
eating attack, eating behaviour, eating disorder, narcolepsy

## 1 | INTRODUCTION

Narcolepsy type 1 is a chronic central disorder of hypersomnolence that clinically manifests with excessive daytime sleepiness (EDS), cataplexy, sleep paralysis, hypnopompic or hypnagogic hallucinations, and disrupted nocturnal sleep (Golden & Lipford, 2018; Sateia, 2014). Cataplexy, the pathognomonic symptom of narcolepsy type 1, is defined as a sudden and brief loss of muscle tone, ranging from partial weakness to generalised paralysis, typically triggered by intense emotional stimuli (Dauvilliers et al., 2014; Wang et al., 2018). Narcolepsy type 1 is associated with a deficiency of the hypothalamic neuropeptide hypocretin-1 (hcrt-1) in the cerebrospinal fluid (a value <110 pg/mL is diagnostic), and the disorder is thought to be caused by a selective defect of the hypocretin-producing neurons in the lateral hypothalamus.

The management of narcolepsy type 1 comprises pharmaceutical intervention to ease symptoms. Given the distinct effects of available medications, selecting a first-line approach that includes sodium oxybate, modafinil, or pitolisant necessitates careful consideration of several factors, including the patient's overall medical complexity, the severity of individual symptoms, and the presence of potential comorbidities (Franceschini et al., 2021). Antidepressants, although not registered in Europe for narcolepsy type 1, have been used and they are still commonly prescribed in clinical practice for cataplexy, despite the indication for sodium oxybate first, and pitolisant lately. The recent European Guidelines include antidepressants within the pharmacological treatment for cataplexy (Bassetti et al., 2021).

Hypocretins, also known as orexins, were initially described as regulators of feeding behaviour and are known to play a role in several essential physiological functions, including sleep–wake cycle regulation, energy homeostasis, neuroendocrine function, glucose metabolism, stress-adaptive responses, and reward-seeking behaviour (Kotagal et al., 2004; Schuld et al., 2000). The complex role of hypocretins makes the well-known association between narcolepsy type 1 and overweight/obesity intriguing (Krhan et al., 2001; Poli et al., 2013).

Patients with narcolepsy type 1 have 3–4 body mass index (BMI) points more than healthy controls (American Psychiatric Association, 2013; Nishino et al., 2001; Swanson et al., 2011). This association may be related to aberrant hypocretin function, but the mechanisms underlying the higher BMI in individuals with narcolepsy type 1 are not fully understood, and we do not know why patients with hypocretin deficiency may present differently with obesity.

Studies investigating the presence of eating disorders in patients with narcolepsy type 1 produced contradictory results. Dahmen and colleagues did not find an increased prevalence of eating disorders in patients with narcolepsy type 1 compared with the general population data (Dahmen et al., 2008). In contrast, another case–control study found that patients with narcolepsy type 1 tend to develop eating disorders more frequently than healthy controls (Droogleever Fortuyn et al., 2008). Accordingly, two additional studies highlighted that individuals with narcolepsy type 1 had more frequently mild eating disorders, such as eating disorder not other specified (EDNOS), and a higher prevalence of sleep-related-eating disorder (SRED) (Chabas et al., 2007; Palaia et al., 2011). Interestingly, a case–control and a cross-sectional study revealed an association between narcolepsy type 1 and binge eating disorder (BED) (Dimitrova et al., 2011; Fortuyn et al., 2008). Therefore, the evidence from the literature about the relation between narcolepsy type 1 and eating disorders is inconclusive.

Eating disorders present with alterations in eating behaviours, emotional and cognitive dysfunctions, and dysregulated body weight (Hoek, 2006). Anorexia nervosa (AN), bulimia nervosa (BN), and binge-ED represent the most common eating disorders in the general population, and these disorders frequently show an early onset during adolescence (Call et al., 2013). Anorexia nervosa has a prevalence of 0.3% and is the most common cause of weight loss in youth (Hartman, 1995), while bulimia nervosa affects 1.5% of the population and involves the uncontrolled eating of an abnormally large amount of food in a short period, followed by compensatory behaviours such as self-induced vomiting or laxative abuse (Bulik et al., 2010).

Binge-ED affects an estimated 1–3% of the general population, making it the most frequent eating disorder. Individuals with BED report consuming a large amount of food quickly with a feeling of losing control (Vamado et al., 1997).

Further, eating disorders have been defined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) as an "other specified feeding or eating disorder", such as night-eating syndrome (NES), which is characterised by recurrent episodes of night-eating (Allison et al., 2010; American Psychiatric Association, 2016).

This study aimed to assess the prevalence of eating disorders in narcolepsy type 1 versus unaffected controls, their clinical association among patients with narcolepsy type 1, and the possible association between eating disorders and obesity in narcolepsy type 1.

## 2 | METHODS

### 2.1 | Population of the study and ethical committee

Individuals aged 18 to 65 years old, with an established diagnosis of narcolepsy type 1 according to current criteria (American Academy of Sleep Medicine, 2014), were consecutively recruited between March and June 2023 among patients attending the Outpatient Clinic of the Narcolepsy Centre of IRCCS Istituto delle Scienze Neurologiche di Bologna (Italy) for routine follow-up visits. Individuals from the general population were enrolled during the same period among the acquaintances of the recruited patients, excluding family members. All participants' medical history was evaluated through clinical interviews, and psychiatric comorbidities were identified according to DSM-5 diagnostic criteria by a psychiatrist. Patients with a history of psychotic disorder, bipolar disorder, and major depressive disorder were excluded from the study.

During the clinical interview performed by a medical doctor trained in sleep medicine, we collected anthropometric data (BMI) as

well as the presence of a diagnosis of cardiovascular comorbidities (including myocardial infarction, arterial hypertension, stroke, and dyslipidaemia) gastrointestinal disease, anxiety disorder, obstructive sleep apnea syndrome (OSAS), REM sleep behaviour disorder (RBD), restless leg syndrome, and neurological disease. Regarding BMI, the World Health Organization cut-off was used to identify participants with obesity i.e., 25.0–29.9 kg/m$^2$ for obesity grade 1, 30.0–39.9 kg/m$^2$ for obesity grade 2, and 40.0 kg/m$^2$ or more for obesity grade 3 (World Health Organization, 1995). Patients were also requested to disclose any drug therapies they were undergoing, including antihypertensives, thyroid medications, antidepressants, and drugs for the symptomatic treatment of narcolepsy type 1.

Socio-demographic data were also collected for each subject enrolled.

We administered five questionnaires investigating narcolepsy type 1 and ED symptoms to assess disease severity, estimated the prevalence of eating disorders, and their possible association.

The study was approved by the Independent Ethics Committee of Area Vasta Emilia Centro (CE-AVEC) (number 851-2022/AUSLBO). A signed informed consent form was obtained from the individuals included in the study. Each participant was free to withdraw his/her consent for inclusion in the study at any time.

## 2.2 | Tool measures

### 2.2.1 | Eating disorder examination questionnaire

The eating disorder examination questionnaire (EDE-Q) is a self-report questionnaire to explore several symptoms of eating disorders. The latest version, EDE-Q 6.0 consists of 22 items grouped into four dimensions: eating concern, shape concern, weight concern, and dietary restraint (Calugi et al., 2016). The validity of EDE-Q scores has been helpful for discriminating between individuals with and without eating disorders. Specifically, a score of ≥2.8 effectively distinguished clinical cases from non-cases with a sensitivity of 85% and a specificity of 75% (Mond et al., 2008).

### 2.2.2 | Binge eating scale

The binge eating scale (BES) is a 16-item self-report measure investigating the typical behavioural manifestation and emotions preceding or following a binge. Research has shown that BES is particularly accurate in the identification of non-binge eaters, making it ideal for screening purposes (Greeno et al., 1995). Based on the BES total score, subjects can be characterised as binge eaters with scores greater than or equal to 17 with a sensitivity value of 81.8% and a specificity value of 97.8% (Marcus et al., 1988).

### 2.2.3 | Night eating questionnaire

The most common version of the night eating questionnaire (NEQ) comprises 14 items measured on a five-point Likert-type scale. The NEQ comprises four dimensions: nocturnal ingestion, evening hyperphagia, morning anorexia, and mood and sleep disturbance (Allison et al., 2008). A total score of 25 or more indicates the presence of NES with a sensitivity of 50% and a specificity of 98.5%. The Italian version of this questionnaire has been validated to assess the presence of NES in different types of populations (Innamorati et al., 2018).

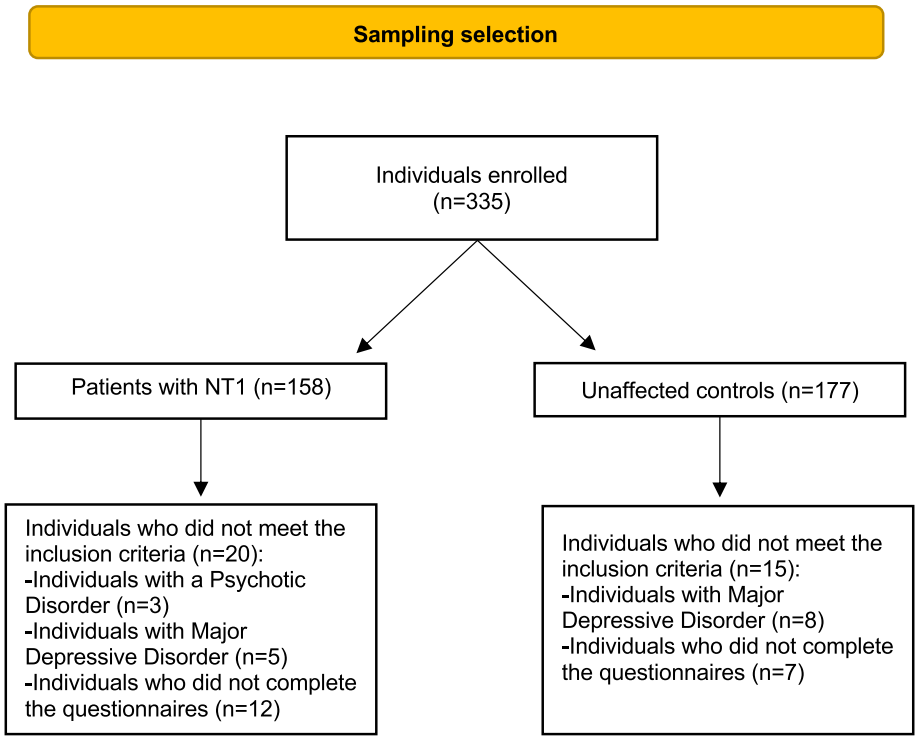


**FIGURE 1** Flow-chart of sampling selection.

### 2.2.4 | Epworth sleepiness scale

The Italian version of the Epworth sleepiness scale (ESS) assessed subjective sleepiness based on the self-rated likelihood of falling asleep in eight different situations, with possible scores of 0 (would never doze) to 3 (high chance of dozing) on each question (Vignatelli et al., 2003). A total score ≥ 11/24 indicates pathological subjective EDS (Moller et al., 2006).

### 2.2.5 | Narcolepsy severity scale

The narcolepsy severity scale (NSS) is a self-administered 15-item scale that evaluates the severity, frequency, and impact of the five main narcolepsy symptoms (EDS, cataplexy, hypnagogic hallucinations, sleep paralysis, and disturbed nocturnal sleep). The total score (0–57) is the sum of the scores of each item, and higher scores indicate more severe symptoms. However, the number of items per symptom is not identical, and six items assess the symptom frequency, while nine items describe the symptom's effects on daily life (Dauvilliers et al., 2017; Dauvilliers et al., 2020).

## 2.3 | Statistical analysis

The descriptive analysis was presented as median and interquartile range (IQR), while categorical variables were absolute (*n*) and relative frequency (%). The comparison between groups (patients with narcolepsy type 1 vs. unaffected controls) was evaluated with the

**TABLE 1** Sociodemographic features of narcolepsy type 1 (NT1) patients and unaffected controls.

| | NT1 patients ($N = 138$) | Unaffected controls ($N = 162$) | *p* value |
|---|---|---|---|
| Sex, *n* (%) | | | |
| Males | 64 (46.4) | 77 (45.5) | 0.84 |
| Age, median (IQR) | 33.5 (22–49) | 32 (24–51) | 0.34 |
| Civil status, *n* (%) | | | |
| Single | 82 (59.4) | 49 (30.00) | **<0.001** |
| In a relationship | 56 (40.5) | 113 (70.00) | |
| Education, *n* (%) | | | |
| Primary school | 4 (2.9) | 1 (0.6) | **0.034** |
| Secondary school | 28 (20.3) | 18 (11.1) | |
| ≥ High school | 106 (76.8) | 143 (88.2) | |
| Employed, *n* (%) | | | |
| No | 71 (51.4) | 57 (35.2) | **0.005** |
| Yes | 67 (48.6) | 105 (64.8) | |
| BMI | 25.3 (22.6–29.7) | 23.1 (21.1–26.3) | **<0.001** |
| Orexin, median (IQR) | 14.3 (0–61.1) | - | - |
| Narcoleptic therapy | | | |
| Sodium oxybate, *n* (%) | 99 (71.7) | - | - |
| CNS stimulants, *n* (%) | 101 (73.2) | - | - |
| Antidepressants, *n* (%) | 43 (31.2) | 2 (1.3) | **<0.001** |
| Drug therapy | | | |
| Antihypertensive, *n* (%) | 18 (13.00) | 13 (8.00) | 0.15 |
| Thyroid drugs, *n* (%) | 5 (3.6) | 8 (4.9) | 0.58 |
| Comorbidity, *n* (%) | | | |
| Cardiovascular disease | 28 (20.3) | 16 (9.9) | **0.011** |
| Gastrointestinal disease | 9 (6.5) | 5 (3.1) | 0.16 |
| Neurological disease | 17 (12.3) | 3 (1.9) | **<0.001** |
| RBD | 10 (7.3) | 0 (0) | **<0.001** |
| Anxiety disorder | 9 (6.5) | 0 (0) | **0.001** |
| OSAS | 23 (16.7) | 2 (1.2) | **<0.001** |
| Restless leg syndrome | 14 (10.1) | 1 (0.6) | **<0.001** |

Abbreviations: BMI, body mass index; CNS, central nervous system; RBD, REM sleep behaviour disorder; OSAS, obstructive sleep apnea syndrome.

Mann–Whitney test for continuous variables and with the chi-square test for categorical variables. A value of $p$ <0.05 was considered significant.

Following the cut-off scores of EDE-Q (i.e., total score ≥2.8), BES (i.e., total score ≥ 17), and I-NEQ (i.e., total score ≥ 25) participants were categorised into "clinically significant ED symptomatology" and "no clinically significant ED symptomatology". Three multivariable logistic regression models were performed, using the dichotomised categories identified based on EDE-Q, I-NEQ, and BES positivity scores as dependent variables in relation to the significant factors in the univariable analysis. The best multivariable models were selected using a stepwise procedure with the likelihood ratio test for model comparison.

A multivariable linear regression model was performed, using BMI as a dependent variable (after logarithmic transformation), in relation to the significant factors in the univariable analysis. The results were presented as beta coefficients with 95% CI.

Statistical analysis was performed using Stata SE version 14.2.

## 3 | RESULTS

### 3.1 | Sociodemographic and clinical characteristics

Of the 335 individuals who expressed an initial interest in the study, the final sample that met the inclusion criteria included 300 subjects, 138 individuals with narcolepsy type 1, and 162 unaffected controls (Figure 1).

Most of the individuals in the sample were female (55%), and the two groups did not differ in age ($p = 0.34$), with a mean age of the sample of 32.7 years old. Patients with narcolepsy type 1 were more frequently single ($p < 0.001$) and unemployed ($p = 0.05$), and typically they had a lower education level ($p = 0.034$) than the controls. In the NT1 group, there was a higher rate of sleep-related comorbidities, such as RBD, OSAS, and restless leg syndrome ($p < 0.001$), cardiovascular diseases ($p = 0.011$), neurological diseases ($p < 0.001$), and anxiety disorder ($p = 0.001$). Patients with narcolepsy type 1 had a higher BMI than the control group ($p < 0.001$).

No significant differences were found in the consumption of antihypertensive therapies ($p = 0.15$) or thyroid drugs ($p = 0.58$). Among the NT1 group, sodium oxybate had been prescribed in 71.7% of cases, and other wake-promoting agents available in Italy for the treatment of narcolepsy in 73.2% of cases.

The characteristics of individuals included in the study are reported in Table 1.

### 3.2 | Eating disorders and subjective sleepiness assessment

As expected, the ESS scores appeared significantly higher in narcolepsy type 1 than in controls ($p < 0.001$).

Concerning the assessment of eating disorders, the value of EDE-Q was higher in the patient group ($p = 0.040$) with significantly higher scores only in the eating concern subscale ($p = 0.001$). For the BES score, we did not see significant differences between the two groups ($p = 0.26$). Conversely, the NT1 group presented more severe scores in I-NEQ ($p < 0.001$) and in all related subscales: morning anorexia, evening hyperphagia, and nocturnal ingestion.

Eating and sleeping evaluations are reported in Table 2.

### 3.3 | Characteristics of NT1 individuals with high scores of EDE-Q

The percentage of male individuals with narcolepsy type 1 with abnormal scores of EDE-Q (i.e., ≥ 2.8) was lower than in patients with narcolepsy type 1 and normal EDE-Q scores (17.3% vs. 52.7%, $p = 0.002$). The BMI showed a non-significant trend toward higher values in patients with narcolepsy type 1 and positive EDE-Q than in patients with normal EDE-Q ($p = 0.093$). Other significant differences concern comorbidity with anxiety disorders, which appeared more frequent in individuals with high EDE-Q scores (26.09% vs. 2.61%, $p < 0.001$) as well as the consumption of antidepressant medications (56.5% vs. 26.1%, $p = 0.004$). Patients with narcolepsy type 1 with altered EDE-Q complained of more severe narcolepsy symptoms expressed by higher scores in the NSS ($p = 0.007$) and were less frequently treated with sodium oxybate than patients with normal EDE-Q (76.5% vs. 47.8%, $p < 0.005$). No differences were found in age, ESS scores, CSF hrct-1 levels, and prescribed CNS stimulants (Table 3).

**TABLE 2** Questionnaire assessment of patients and controls.

| | NT1 patients ($N = 138$) median (IQR) | Unaffected controls ($N = 162$) median (IQR) | $p$ value |
|---|---|---|---|
| EDE-Q | 3 (0–12) | 3 (0–6) | **0.040** |
| Eating concern | 0 (0–1) | 0 (0–0.5) | **0.001** |
| Weight concern | 0.5 (0–2.5) | 0.5 (0–2) | 0.56 |
| Shape concern | 0.75 (0–1.75) | 0.5 (0–1.5) | 0.29 |
| Restraint | 0.9 (0–2.4) | 0.7 (0–1.6) | 0.12 |
| BES | 4 (1–10) | 3 (2–7) | 0.26 |
| I-NEQ | 11 (9–17) | 9 (7–13) | **<0.001** |
| Morning anorexia | 2 (2–3) | 1.5 (0–2) | **<0.001** |
| Evening hyperphagia | 5 (4–6) | 4 (4–5) | **<0.001** |
| Nocturnal ingestion | 1 (0–6) | 0 (0–1) | **<0.001** |
| Mood/Sleep | 2 (1–4) | 3 (1–5) | **0.029** |
| ESS | 9 (6–13) | 5 (3–8) | **<0.001** |
| NSS | 18 (13–25) | - | - |

Abbreviations: BES, binge eating scale; EDE-Q, eating disorder examination questionnaire; ESS, Epworth sleepiness scale; I-NEQ, Italian night eating questionnaire; NSS, narcolepsy severity scale.

The multivariable logistic model with EDE-Q positivity as a dependent variable confirms a significant positive association with antidepressant drug consumption (OR 3.24, 95% CI 1.09–9.61, $p = 0.034$) and a negative association with male gender (OR 0.20, 95% CI 0.06–0.749, $p = 0.017$) and the use of sodium oxybate (OR 0.24, 95% CI 0.08–0.73, $p = 0.012$). On the other hand, the

**TABLE 3** Comparison between narcolepsy type 1 (NT1) patients with positive and negative EDE-Q, BES, and I-NEQ.

| | NT1 patients with EDE-Q high score (N = 23) | NT1 patients with EDE-Q low score (N = 115) | *p* value |
|---|---|---|---|
| Male sex, *n* (%) | 4 (17.3) | 60 (52.7) | 0.002 |
| Age, median (IQR) | 33 (22–48) | 34 (21–49) | 0.90 |
| BMI, median (IQR) | 26.3 (23.8–30.1) | 24.78 (21.7–29.7) | 0.093 |
| Anxiety disorder, *n* (%) | 6 (26.09) | 3 (2.61) | <0.001 |
| Restless leg syndrome, *n* (%) | 5 (21.74) | 9 (7.83) | 0.04 |
| OSAS, *n* (%) | 3 (13.04) | 20 (17.39) | 0.61 |
| Antidepressant therapy, *n* (%) | 13 (56.5) | 30 (26.1) | 0.004 |
| ESS, median (IQR) | 11 (7–16) | 9 (6–12) | 0.081 |
| NSS, median (IQR) | 30 (14–37) | 17 (12–24) | 0.007 |
| Orexin, median (IQR) | 20.1 (0–45.8) | 13.1 (0–62.9) | 0.47 |
| Sodium oxybate, *n* (%) | 11 (47.8) | 88 (76.5) | 0.005 |
| CNS stimulants, *n* (%) | 18 (78.3) | 83 (72.2) | 0.54 |
| | **NT1 patients with BES high score (*N* = 17)** | **NT1 patients with BES low score (*N* = 121)** | ***p* value** |
| Male sex, *n* (%) | 2 (11.7) | 62 (51.2) | **0.002** |
| Age, median (IQR) | 27 (22–40) | 34 (22–49) | 0.38 |
| BMI, median (IQR) | 27.3 (23.8–33.5) | 25.2 (22.2–29.4) | 0.12 |
| Anxiety disorder, *n* (%) | 5 (29.4) | 4 (3.3) | **<0.001** |
| Restless leg syndrome, *n* (%) | 1 (5.88) | 13 (10.74) | 0.53 |
| OSAS, *n* (%) | 1 (5.88) | 22 (18.18) | 0.20 |
| Antidepressant therapy, *n* (%) | 10 (58.8) | 33 (27.3) | **0.009** |
| ESS, median (IQR) | 10 (7–14) | 9 (6–12) | 0.16 |
| NSS, median (IQR) | 30 (16–37) | 17 (12–25) | **0.003** |
| Orexin, median (IQR) | 20.7 (0–45.8) | 13.62 (0–61.8) | 0.60 |
| Sodium oxybate, *n* (%) | 10 (58.8) | 89 (73.6) | 0.20 |
| CNS stimulants, *n* (%) | 13 (76.5) | 88 (72.7) | 0.74 |
| | **NT1 patients with I-NEQ high score (*N* = 13)** | **NT1 patients with I-NEQ low score (*N* = 125)** | ***p* value** |
| Male sex, *n* (%) | 3 (23.1) | 61 (48.8) | 0.077 |
| Age, median (IQR) | 31 (22–45) | 34 (22–49) | 0.54 |
| BMI, median (IQR) | 24.8 (22.7–30.1) | 25.30 (22.6–29.5) | 0.94 |
| Anxiety disorder, *n* (%) | 1 (7.7) | 8 (6.4) | 0.85 |
| Restless leg syndrome, *n* (%) | 1 (7.69) | 13 (10.40) | 0.75 |
| OSAS, *n* (%) | 3 (23.08) | 20 (16.00) | 0.51 |
| Antidepressant therapy, *n* (%) | 10 (76.9) | 33 (26.4) | **<0.001** |
| ESS, median (IQR) | 9 (7–12) | 9 (6–13) | 0.89 |
| NSS, median (IQR) | 25 (15–37) | 18 (13–25) | 0.12 |
| Orexin, median (IQR) | 13.35 (0–28.5) | 14.83 (0–62.9) | 0.44 |
| Sodium oxybate, *n* (%) | 8 (61.5) | 91 (72.8) | 0.39 |
| CNS stimulants, *n* (%) | 10 (76.9) | 91 (72.8) | 0.74 |

Abbreviations: BES, binge eating scale; BMI, body mass index; CNS, central nervous system; EDE-Q, eating disorder examination questionnaire; ESS, Epworth sleepiness scale; I-NEQ, Italian night eating questionnaire; NSS, narcolepsy severity scale; OSAS, obstructive sleep apnea syndrome.

**TABLE 4** Multivariable logistic regression models for EDE-Q, I-NEQ, and BES scores; multivariable linear regression model for BMI in association with the other variables in narcolepsy type 1 (NT1) patients.

| **EDE-Q score-dependent variable** | | |
|---|---|---|
| **Independent variables** | **OR (95% CI)** | ***p*-value** |
| Male sex | 0.20 (0.06–0.749) | 0.017 |
| Anxiety disorders | 3.27 (0.54–19.77) | 0.197 |
| Sodium oxybate | 0.24 (0.08–0.73) | 0.012 |
| Antidepressants | 3.24 (1.09–9.61) | 0.034 |
| Restless leg syndrome | 1.54 (0.36–6.56) | 0.563 |
| NSS score | 1.04 (0.99–1.1) | 0.158 |
| **I-NEQ score-dependent variable** | | |
| **Independent variables** | **OR (95% CI)** | ***p*-value** |
| Male sex | 0.28 (0.07–1.12) | 0.072 |
| Antidepressants | 9.97 (2.53–39.3) | 0.001 |
| **BES score-dependent variable** | | |
| **Independent variables** | **OR (95% CI)** | ***p*-value** |
| Male sex | 0.14 (0.03–0.70) | 0.016 |
| NSS score | 1.07 (1.01–1.13) | 0.022 |
| Antidepressants | 3.78 (1.21–11.8) | 0.022 |
| **BMI-dependent variable** | | |
| **Independent variables** | **Beta coefficient (95% CI)** | ***p*-value** |
| Male sex | 0.073 (0.001–0.145) | 0.046 |
| EDE-Q score | 0.12 (0.028–0.21) | 0.011 |
| Antihypertensive | 0.10 (−0.003–0.21) | 0.056 |
| OSAS | 0.11 (0.006–0.21) | 0.038 |
| Orexin | −0.00085 (−0.00162 – −0.00007) | 0.046 |

Abbreviations: BES, binge eating scale; EDE-Q, eating disorder examination questionnaire; I-NEQ, Italian night eating questionnaire; NSS, narcolepsy severity scale; OSAS, obstructive sleep apnea syndrome.

association with the presence of anxiety disorders resulted in a high variability at the limit of significance (OR 3.27, 95% CI 0.54–19.77, $p = 0.197$) (Table 4).

## 3.4 | Differences between NT1 individuals with high and low BES scores

The majority of individuals with narcolepsy type 1 with positive BES scores (i.e., total score ≥ 17) were females compared with the NT1 group with normal BES results (88.3% vs. 48.8%, $p = 0.002$). Anxiety disorders (29.4% vs. 3.3%, $p < 0.001$) and the use of antidepressants (58.8% vs. 27.3%, $p < 0.009$) were more frequent in patients with narcolepsy type 1 and altered BES than in the group with normal scores. While age, BMI, subjective sleepiness, and CSF-hrct-1 levels were comparable, the NSS scores were higher in patients with altered BES results ($p = 0.003$). No differences emerged in therapy with sodium oxybate and CNS stimulants ($p = 0.20$ and $p = 0.74$ respectively) (Table 3).

The multivariable logistic model confirmed the association with antidepressant drug consumption (OR 3.78, 95% CI 1.21–11.8, $p = 0.022$) and the NSS total score (OR 1.07, 95% CI 1.01–1.13, $p = 0.022$) with BES positivity as a dependent variable, and a protective role of male gender (OR 0.14, 95% CI 0.03–0.70, $p = 0.016$) (Table 4).

## 3.5 | Differences between NT1 individuals with high and low I-NEQ scores

Participants with narcolepsy type 1 and a positive score of the I-NEQ (i.e., total score ≥ 25) did not significantly differ in sex distribution from those with normal I-NEQ, despite a trend toward a higher female prevalence (76.9% vs. 51.2%, $p = 0.077$). Significant differences were

**TABLE 5** Differences between narcolepsy type 1 (NT1) patients with high and low levels of BMI.

| | **NT1 patients with high level of BMI ($N = 33$)** | **NT1 patients with low level of BMI ($N = 105$)** | ***p* value** |
|---|---|---|---|
| Male sex, *n* (%) | 21 (63.6) | 43 (40.9) | **0.023** |
| Age, median (IQR) | 44 (22–48) | 33 (22–49) | 0.97 |
| Anxiety disorder, *n* (%) | 2 (6.1) | 7 (6.7) | 0.90 |
| Antidepressant therapy, *n* (%) | 14 (42.4) | 29 (27.6) | 0.10 |
| ESS, median (IQR) | 7 (6–12) | 9 (7–13) | 0.27 |
| NSS, median (IQR) | 17 (13–25) | 18 (12–25) | 0.88 |
| Orexin, median (IQR) | 0 (0–21.0) | 21.1 (0–68.6) | **0.041** |
| Sodium oxybate, *n* (%) | 49 (68.1) | 50 (75.8) | 0.31 |
| CNS stimulants, *n* (%) | 48 (66.7) | 53 (80.3) | 0.071 |

Abbreviations: BMI, body mass index; CNS, central nervous system; ESS, Epworth sleepiness scale; NSS, narcolepsy severity scale.

**TABLE 6** Differences between narcolepsy type 1 (NT1) patients with mild (i.e., <14) and moderate-to-very severe (i.e., <15–57) scores in narcolepsy severity scale.

| | NSS mild (0–14) ($N = 45$) | NSS moderate-to-very severe (15–57) ($N = 93$) | *p*-value |
|---|---|---|---|
| Male sex, *n* (%) | 24 (53) | 40 (43) | 0.25 |
| Age, median (IQR) | 30 (21–48) | 34 (22–49) | 0.83 |
| BMI, median (IQR) | 25 (22–29) | 25 (22–29) | 0.79 |
| Orexin, median (IQR) | 21 (0–79) | 13 (0–54) | 0.41 |
| ESS, median (IQR) | 6 (3–8) | 10 (7–15) | **<0.001** |
| EDE-Q, median (IQR) | 0 (0–10) | 3 (0–12) | 0.14 |
| Eating concern, median (IQR) | 0.80 (0–2.80) | 1 (0–2.40) | 0.96 |
| Weight concern, median (IQR) | 0 (0–0.50) | 0 (0–1.50) | **0.02** |
| Shape concern, median (IQR) | 0.50 (0–1) | 1 (0–3) | **0.03** |
| Restraint, median (IQR) | 0.50 (0–1.38) | 1 (0.25–1.75) | **0.04** |
| BES, median (IQR) | 2 (1–5) | 5 (2–11) | **0.01** |
| I-NEQ, median (IQR) | 10 (8–13) | 13 (9–18) | **0.01** |
| Morning anorexia, median (IQR) | 2 (2–3) | 2 (2–3.5) | 0.30 |
| Evening hyperphagia, Median (IQR) | 5 (4–6) | 5 (4–6) | 0.63 |
| Nocturnal ingestion, median (IQR) | 1 (0–4) | 3 (1–4.50) | **0.002** |
| Mood/Sleep, median (IQR) | 0 (0–4) | 1 (0–8) | 0.18 |

Abbreviations: BES, binge eating scale; EDE-Q, eating disorder examination questionnaire; I-NEQ, Italian night eating questionnaire; NSS, narcolepsy severity scale.

detected in the consumption of antidepressant drugs, more frequently in patients with altered I-NEQ (76.9% vs. 26.4%, $p < 0.001$). No significant differences were observed in age, BMI, anxiety disorder, CSF-hrct1 levels, ESS, NSS, or consumption of drugs for narcolepsy symptoms (Table 3).

With the multivariate logistic regression analysis, I-NEQ positivity emerged strongly associated with antidepressant drug consumption (OR 9.97, 95% CI 2.53–39.3, $p = 0.001$), also including male gender as a possible confounder (OR 0.28, 95% CI 0.07–1.12, $p = 0.072$) (Table 4).

### 3.6 | Differences between NT1 individuals with high and low levels of BMI

Patients with high levels of BMI were more frequently male (63.6% vs. 40.9%, $p = 0.023$), with lower CSF-hrct1 values ($p = 0.041$). No other significant differences were found in anxiety disorder, consumption of antidepressants, or use of narcoleptic medications, Table 5 reports the differences in BMI between individuals with narcolepsy type 1.

### 3.7 | Differences between NT1 individuals with high and low NSS scores

Most of the patients had moderate-to-very severe rather than low NSS scores (67.3% vs. 32.6%). Patients with moderate-to-very severe NSS presented high scores in EDE-Q subscales as weight concern ($p = 0.02$), shape concern ($p = 0.03$), and restraint ($p = 0.04$) and higher scores at BES ($p = 0.01$) and I-NEQ ($p = 0.01$), respectively (Table 6).

## 4 | DISCUSSION

In this cross-sectional study, we investigated the association between narcolepsy type 1 and eating disorders and evaluated the factors associated with the symptomatology of eating disorders in narcolepsy type 1 as well as their possible connection with BMI.

According to our results, patients with narcolepsy type 1 have more severe eating disorders symptoms than controls. Our study showed that, within an NT1 cohort, there were more female individuals with eating disorders, confirming that women have a greater risk of eating disorders than men (Smink et al., 2012). Moreover, patients with narcolepsy type 1 with eating disorders often have a comorbid anxiety disorder, mirroring evidence from the literature showing that eating disorders and anxiety disorder often co-occur (Pallister & Waller, 2008). This association could also explain why patients with narcolepsy type 1 with eating disorders take more antidepressant medication than the group without eating disorders.

We confirm that narcolepsy type 1 patients have a higher BMI, 3–4 BMI points more than the control population and the Italian national average (Dahmen et al., 2001; Mohammadi et al., 2021).

Sodium oxybate was prescribed in the majority of patients, both with higher and lower BMI, in line with the literature, which reports

that narcolepsy type 1 medication status has no demonstrable association with the BMI of patients with narcolepsy type 1 with obesity despite the well-known association of sodium oxybate treatment with weight loss (Date et al., 1999). Interestingly, treatment with sodium oxybate appeared to be a protective factor on clinically relevant symptoms of eating disorders, expressed by a pathological score in EDE-Q.

We also found that patients with narcolepsy type 1 with high levels of BMI have lower CSF-hrct1 levels compared with the normal BMI group, suggesting that elevated BMI could be an intrinsic manifestation of the pathophysiology of narcolepsy type 1 (Branson et al., 2003).

Our findings demonstrate that eating disorders are more prevalent in individuals with narcolepsy type 1 than in healthy controls. The former, indeed, develop NES more often than the latter (the presence of NES in individuals with narcolepsy type 1 has not been previously established), also suggested by previous research demonstrating that patients with NES experience higher levels of insomnia and poorer sleep quality (Brion et al., 2012).

Another interesting evidence was the highest score at EDE-Q among patients with narcolepsy type 1, with a higher score in the eating concern subscale, which represents the impact of eating symptomatology in an individual's life.

Indeed, symptoms of eating disorders further add to the already significant burden of illness of narcolepsy type 1. We did not appreciate significant differences in BES total score between the two groups, however, patients with narcolepsy type 1 had higher scores.

The hypocretin system is intrinsically related to the dopaminergic system, both being fundamental for many types of motivated behaviour. Specifically, hypocretin neurons project to several reward-associated regions, with interaction with impulsivity, which is a typical trait present in various types of eating disorders, such as BES and NES (Jennum et al., 2017). Moreover, preliminary evidence highlighted that patients with narcolepsy type 1 might display higher levels of impulsivity, furthermore, stimulants are known to increase impulsivity (Aston-Jones et al., 2009).

Based on our findings, we speculate that individuals diagnosed with narcolepsy type 1 exhibiting more pronounced narcoleptic symptoms may be predisposed to manifest BES, NES, and have heightened concerns of weight, shape, and restraint. Consequently, it may be clinically useful to assess the presence of these eating disorders in patients with narcolepsy type 1 presenting with severe clinical manifestations.

Lastly, we confirm different sociodemographic aspects already known from the literature, such as that individuals from the general population have more relationships and a higher employment rate than patients with narcolepsy type 1. The literature reported that patients suffering from narcolepsy are more unemployed due to work-related difficulties, such as falling asleep at work (Teixeira et al., 2004).

Patients with narcolepsy type 1 more often have clinical comorbidities, particularly OSAS, restless leg syndrome, cardiovascular disease, and anxiety disorder. Also, this evidence confirms a previous study reported in the literature, in which individuals with narcolepsy type 1 frequently have psychiatric comorbidities such as depression and anxiety disorders (Abenza-Albidua et al., 2023).

Our study has several limitations. In particular, we investigated only symptoms that were present during or close to the time of the clinical interview, and thus, it cannot be ruled out that eating disorders might manifest at some point during the life of an individual with narcolepsy type 1.

Moreover, the I-NEQ scale had low sensibility, therefore, future studies might implement more reliable measures to identify NES symptoms.

Furthermore, we did not evaluate with a specific questionnaire the possible presence of depressive symptoms, although the previous diagnosis of major depressive disorder was a criterion of exclusion in our study.

Finally, the sole administration of a questionnaire cannot represent a diagnosis, but a clinical examination by a psychiatrist must follow it.

Several epidemiological research studies were carried out to identify the comorbidities of narcolepsy type 1, however, none of these studies explored the presence of eating disorders (Ruoff et al., 2017), therefore, future epidemiological studies should investigate this association.

Nevertheless, our work suggests that in the multidisciplinary approach to patients with narcolepsy type 1, questionnaires for eating disorders (EDE-Q, BES, I-NEQ) should be administered to identify those cases to be addressed by an eating behaviour disorders specialist.

## 5 | CONCLUSION

This study assesses eating disorders symptoms in patients with narcolepsy type 1 using a cross-sectional design through validated questionnaires in a large population. To our knowledge, this is the first study to show the presence of eating disorders symptomatology in narcolepsy type 1 and a specific type of eating disorder behaviour, such as NES.

Individuals with narcolepsy type 1 more frequently have high BMI, but according to the evidence from our study, subjects suffering from narcolepsy type 1 reported higher scores on EDE-Q and I-NEQ than the general population. These data are extremely important for clinicians, indeed, a multidisciplinary team with a psychiatry specialist, dietician, and nutritionist is necessary to identify the possible presence of eating disorders in narcoleptic patients. Future polysomnographic studies may unravel the relation between nocturnal sleep features and eating disorders in narcolepsy type 1.

### AUTHOR CONTRIBUTIONS

**Valentina Baldini:** Conceptualization; investigation; writing – original draft; methodology; validation; visualization; writing – review and editing; data curation; software. **Noemi Venezia:** Investigation; methodology; validation; visualization; data curation; software. **Anna Iriti:** Investigation; methodology; validation; visualization; data curation; software. **Silvia Quattrocchi:** Investigation; writing – original draft; methodology; validation; visualization; data curation. **Corrado Zenesini:** Writing – review and editing; formal analysis; data curation;

software. **Francesco Biscarini:** Validation; writing – review and editing. **Anna Rita Atti:** Validation; writing – review and editing; supervision; methodology; conceptualization. **Marco Menchetti:** Validation; writing – review and editing; supervision. **Christian Franceschini:** Conceptualization; validation; writing – review and editing; supervision. **Giorgia Varallo:** Writing – review and editing; validation. **Diana De Ronchi:** Validation; writing – review and editing; supervision. **Giuseppe Plazzi:** Conceptualization; investigation; funding acquisition; methodology; validation; visualization; writing – review and editing; project administration; supervision; resources; data curation; formal analysis; software. **Fabio Pizza:** Conceptualization; investigation; writing – original draft; methodology; validation; visualization; writing – review and editing; project administration; formal analysis; software; data curation; supervision; resources.

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Fabio Pizza has received honoraria for presentations from Jazz Pharmaceuticals, for participation in the advisory board by Tadeka, and meeting attendance support from Bioprojet. Giuseppe Plazzi has received honoraria for advisory board and consulting fees from Bioprojet, Jazz, Takeda, and Idorsia. Valentina Baldini, Noemi Venezia, Anna Iriti, Silvia Quattrocchi, Corrado Zenesini, Francesco Biscarini, Anna Rita Atti, Marco Menchetti, Christian Franceschini, Giorgia Varallo, and Diana De Ronchi have nothing to disclose. Non-financial disclosure: none.

## DATA AVAILABILITY STATEMENT

Research data are not shared.

## ORCID

*Christian Franceschini* https://orcid.org/0000-0002-9307-5209

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