

ORIGINAL RESEARCH ARTICLE

Gender, cultural, and geographical differences in
feeding and eating disorders

Giovan Battista Ivan Polichetti*

Department of Mental Health and Addiction, Local Health Trust of Romagna, Italy

Abstract

Feeding and eating disorders have had a complex and multifaceted history, leading to the current classification in the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition*, which recognizes eight distinct disorders. Although simple obesity is not classified as a feeding and eating disorder, it shares important clinical and biological features with several of these conditions. This study examines the influence of gender, sociocultural, and geographical factors on the epidemiology, severity, and prognosis of feeding and eating disorders based on previous investigations. The first study compared anorexia nervosa, bulimia nervosa, and eating disorders not otherwise specified across male patient cohorts from two geographically and culturally distinct countries. The second study evaluated the psycho-neuro-endocrine-immuno-biological aspects of obesity by assessing relative risk and odds ratios among male and female patients receiving specialist treatment. The third study explored the conceptualization of feeding and eating disorders as addictive phenomena within an experimental program for pathological addictions. Statistical, neurobiological, psychodynamic, social, and clinical aspects are integrated to provide a comprehensive understanding of how gender and sociocultural context shape the presentation and progression of feeding and eating disorders. Treatment of feeding and eating disorders cannot be based solely on classification criteria and reference standards, but must be carefully personalized, case by case, based on gender, geographic characteristics, and the individual peculiarities of the affected subjects, to the point of considering treatment, in unsuccessful or resistant cases, as a form of addiction.

***Corresponding author:**
Giovan Battista Ivan Polichetti
(giovanbattista.polichetti@
auslromagna.it)

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1. Introduction

Drawing on more than 25 years of research on feeding and eating disorders, this study integrates and compares findings from three independently conducted investigations to identify similarities and differences and to evaluate whether meaningful distinctions exist between males and females, as well as among the disorders themselves. The initial objective was to establish whether the epidemiological and clinical differences in eating disorders between males and females were coincidental or explained by certain factors. The “discovery” of substantial differences between Italy and the United States, for example, across multiple factors, was later confirmed in a study dedicated solely to obesity. The

study also explores the hypothesis that certain feeding and eating disorders may be conceptualized as addictive disorders. Overall, feeding and eating disorders appear to exhibit broadly similar characteristics across geographically and socioculturally diverse settings, including healthcare systems, particularly with respect to the age at which they typically develop, manifest clinically, or come to medical attention. Furthermore, only patients classified as anorexic may exhibit almost entirely overlapping body mass fluctuations, in line with current considerations on the disease and current therapeutic options in a broad sense; all other values predicted by the parameters considered show variations and fluctuations too large to be compared to any extent. Eating disorders appear to occur more frequently in certain age groups and are generally more prevalent in females.¹ However, their clinical presentation is highly heterogeneous and unpredictable, making it difficult to identify consistent patterns or exclude specific patient groups. Anamnestic factors also appear to differ by sex. In particular, a family history of overweight or obesity was associated with an increased risk of treatment failure among females undergoing therapy for excessive body weight, whereas no such association was observed in males.² These findings suggest that additional, as-yet-unidentified factors may influence treatment outcomes in females beyond the established risk factors. In females, this factor may interact with established risk factors, either amplifying or mitigating their effects on treatment outcomes. In males, it may be a more prominent, or even primary, determinant of treatment response.² Subsequent analysis of the available data highlighted how the level of participation, treatment compliance, and the results at the six-monthly follow-up showed that the approach based on the interpretation of disordered eating behavior as an addictive phenomenon ensured more frequent, better, and longer-lasting results than treatment as usual (seven cases of binge eating disorder with morbid obesity, two cases of anorexia nervosa (AN), one case of bulimia nervosa (BN) with purging behavior, and five cases of *obesitas simplex*).³ Consistent with the other investigations included in the tripartite analysis, this study also identified substantial epidemiological and clinical differences between males and females.

2. Methodology

2.1. Study population

The populations analyzed in this study comprised a combined dataset derived from three groups previously examined in separate investigations: (i) the first group considered two subgroups of male patients affected by eating disorders (135 in the United States and 31 in Italy);

(ii) the second group consisted of 54 subjects aged between 5 and 75 years (32 female subjects and 22 male subjects, all observed at the same Italian clinic); and (iii) the third group consisted of 35 patients (23 females and 12 males) who were recruited from an Italian Addiction Service, as well as from the Mental Health Service, private practice, and the general population.

2.2. Procedure and analysis of the first investigation

A 2002 study provided an in-depth comparison of anthropometric, medical, nutritional, and psychological characteristics data from two cohorts of male patients with eating disorders. The first cohort comprised 135 patients treated at an American center between 1980 and 1994, as reported by Carlat *et al.*⁴ The second cohort included 31 male patients treated at the Naples Polyclinic between 1992 and 1998.⁵ In both cohorts, patients were categorized into four diagnostic groups: all patients (ALL), AN, BN, and eating disorders not otherwise specified (EDNOS). For each of the resulting eight groups, seven data groupings were established: age at onset (years); age at first treatment (years); interval between onset and first treatment (years); body weight (kg); maximum body weight (kg); minimum body weight (kg); and the difference between maximum and minimum body weight (kg).

By processing the raw data for each of the 56 groupings, seven parameters were calculated: mean, variance, standard deviation, standard error, 95% confidence intervals, standard error of the difference, and degrees of freedom. Comparisons were conducted by making analogies at different relative levels through direct comparison or by developing more complex functions, such as statistical significance tests.

2.3. Procedure and analysis of the second investigation

In a study conducted on patients recruited between 2003 and 2008 from a dedicated private practice, all known risk factors for the development and chronicity of obesity were carefully considered.² All cases of “genetic” obesity were excluded from the study, while all those with medical and surgical comorbidities were considered, provided they had never undergone psychotherapy. The key criterion for inclusion in the study was at least one initial positive outcome (i.e., significant weight loss after treatment). A significant and sufficiently homogeneous sample was thus identified and selected: 54 subjects aged between 5 and 75 years, of whom 32 were female and 22 were male. Thirteen patients were initially overweight, and 41 were frankly obese. Among them, 33 had negative outcomes, and 21 had positive outcomes. A positive outcome was defined as maintenance of the achieved clinical outcome at the two-

year follow-up in subjects who remained free from specific therapy, irrespective of age, sex, or initial body weight. The aim of the study was ultimately to determine whether the presence of numerous relatives with obesity in one's medical history served as a risk factor for treatment failure and for the persistence or recurrence of obesity.

An increasing number of relatives affected by overweight (NAR) was associated with a progressively higher risk of an unfavorable outcome at follow-up, even after successful treatment, primarily due to relapse and/or recurrence. However, gender emerged as a confounding factor. When the sample was stratified by sex, this association remained evident only among females, whereas no significant relationship was observed in males, suggesting that family history of overweight had little influence on their clinical outcome.

2.4. Procedure and analysis of the third investigation

The Addiction Service of Cesena (Italy) implemented a multidisciplinary program entitled *It Depends on How You Eat* between March 20 and April 10, 2017.³ A total of 35 participants enrolled in the initiative. The Service had extensive clinical experience in the diagnosis and management of feeding and eating disorders, with approximately 15 patients receiving specialized dietary, pharmacological, and psychotherapeutic treatment in the preceding two years. The program's admission criteria also included subjects from different territorial realities, such as patients undergoing treatment for mental disorders and addictions, privately treated patients, and ordinary citizens.

The study aimed to evaluate whether individuals who had previously failed to respond to conventional treatments (treatment as usual) could benefit from an addiction-based therapeutic approach. All participants had undergone standard treatment programs, primarily dietary and/or psychiatric interventions, without achieving satisfactory outcomes. A preliminary analysis of the available data suggested that conceptualizing disordered eating as an addictive behavior was associated with higher treatment participation, better adherence, and more favorable outcomes at the six-month follow-up than treatment as usual. These apparent benefits were observed across a range of diagnoses, including binge eating disorder with morbid obesity ($n = 7$), AN ($n = 2$), BN with purging behaviors ($n = 1$), and simple obesity (*obesitas simplex*; $n = 5$).

2.5. Statistical analyses

All statistical analyses were performed manually without the use of any software.

2.5.1. Statistical analysis for the first investigation

In this case, the class of critical ratios was used: the one-tailed Student's t -test and the two-tailed Student's t -test. Finally, the p -value was calculated for each aspect examined, using an alpha level of 0.05 for the null hypothesis. The null hypothesis was that the observed differences were merely random variations. All available data and parameters were carefully analyzed in this regard.

2.5.2. Statistical analysis for the second investigation

The slope of the increasing risk for males was negligible (although present), whereas it was more significant for females (exposure risk, relative risk, linear interpolation, and odds ratio).

2.5.3. Statistical analysis for the third investigation

Among the 15 participants with favorable outcomes, females constituted the majority. However, this finding does not align with the female predominance typically reported in the literature, as the baseline cohort had a male-to-female ratio of 1:2. Consequently, the treatment success rate was approximately 50% in both sexes, indicating no meaningful gender difference in outcomes.

3. Results

3.1. Results for the first investigation

Despite the apparent heterogeneity of the study cohorts, several notable similarities emerged. Although the number of patients with AN differed substantially between the two studies (30 in the US cohort vs. 7 in the Italian cohort), their relative frequencies were remarkably similar (22.0% vs. 22.6%, respectively). In contrast, the number of patients with BN differed considerably in both absolute terms (62 vs. 7) and relative frequency (46.0% vs. 22.6%). Nevertheless, the mean age at onset was nearly identical between the two cohorts (19.5 vs. 19.9 years), and this similarity was further supported by comparable standard errors and overlapping 95% confidence intervals.

The EDNOS groups were also heterogeneous in both size and relative frequency (43 patients [32.0%] in the US cohort vs. 17 patients [54.8%] in the Italian cohort). Nevertheless, it was remarkable that the mean age at onset was identical in the two cohorts (19.1 years in both). Furthermore, the standard errors were nearly identical (1.7 vs. 1.6), and the corresponding 95% confidence intervals showed substantial overlap (15.8–22.4 years in the US cohort vs. 16.0–22.2 years in the Italian cohort), indicating a striking similarity despite the differences in sample composition.

Beyond these preliminary descriptive observations, more sophisticated statistical analyses were performed, yielding more robust and informative findings. Mean ages of onset (years) showed no statistically significant differences within the parallel diagnostic categories: ALL ($0.15 < p < 0.20$); AN ($0.05 < p < 0.10$); BN ($0.20 < p < 0.30$); and EDNOS ($p > 0.40$). In the cases of AN and EDNOS, the differences observed in age at first treatment, expressed in years, were also not statistically significant: AN ($0.05 < p < 0.10$) and EDNOS ($0.05 < p < 0.10$). Similarly, for AN, the differences in the mean interval between disease onset and first treatment were also not statistically significant ($0.10 < p < 0.15$).

Similarly, the differences in body weight range, expressed as the difference between maximum and minimum body weight achieved (kg), were also not statistically significant ($0.10 < p < 0.15$). The p -values for the means of ages recorded at the first treatment were largely non-significant in the cases of AN and EDNOS ($0.05 < p < 0.10$). All other values for the parameters evaluated in both groups were statistically significant at the predefined alpha threshold, in agreement with current international literature ($p < 0.05$). The use of a two-tailed Student's t -test did not substantially alter the overall findings; however, two relevant differences should be noted. In the case of EDNOS, the interval between treatment and onset lost statistical significance (whereas, in the one-tailed Student's t -test, this was observed only for the AN group; $p > 0.05$). Moreover, in the case of EDNOS, the difference between the maximum body weight and the minimum body weight achieved was no longer statistically significant (while in the one-tailed analysis this finding was limited to the AN group; $p > 0.05$). These results are presented in [Tables 1 and 2](#).

3.2. Results for the second investigation

The anamnestic dimension of eating disorders appears to have a stronger influence among females, whereas males seem largely unaffected. A family history characterized by multiple relatives with overweight increases the risk of unsuccessful outcomes in individuals undergoing treatment for excess weight; however, this association appears to be limited to females. In contrast, males did not exhibit a comparable relationship, suggesting that this risk factor is specifically associated with females. It was therefore necessary to further interpret the results by considering all known factors. It could be argued that there is an additional yet unexplored etiological factor that influences treatment outcomes more than all the known factors combined. In females, this factor may act in combination with other known risk factors, potentially attenuating their effects and even eliminating the associated risk. In males, however, this factor may represent the predominant determinant itself ([Table 3](#)).

3.3. Results for the third investigation

The reflections made, together with the studies and experiments conducted, suggest a significant, modern, and innovative role for Addiction Services and addiction specialists in the study and management of feeding and eating disorders, especially those resistant to standard treatments or treatments as usual, as well as complex or comorbid cases. These findings also support the potential development and coordination of dedicated multidisciplinary and multiprofessional programs ([Table 4](#)).

4. Discussion

The analysis of the three investigations presented in this article, although substantially different from one another, reveals a common objective: to improve understanding of feeding and eating disorders. Considered collectively, these studies allow broader conclusions to be drawn, providing a potential unified interpretation of the underlying mechanisms and offering possible contributions toward improved management and resolution of these disorders. Overall, the general epidemiology of feeding and eating disorders has remained substantially unchanged in the literature, with a female-to-male ratio of approximately 10:1, as reported when these studies were initiated. Nevertheless, from an epidemiological point of view, cases of feeding and eating disorders in males, although still less prevalent than in females, have shown a steady increase in recent years.⁶ Studies on this topic often report conflicting findings, with some confirming a 10:1 female-to-male ratio, particularly for AN⁷, while others suggest lower ratios (approximately 4:1 for AN and 2:1 or less for BN) and greater variability across other feeding and eating disorders.⁸ Such discrepancies may contribute to underestimating male susceptibility and reinforcing the misconception that eating disorders primarily affect adolescent girls.⁹

The current classification, according to the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5-TR)¹⁰ categorizes eating disorders into (i) pica (in children or adults); (ii) rumination disorder; (iii) avoidant/restrictive food intake disorder; (iv) AN (restricting type or binge-eating/purging type); (v) BN; (vi) binge eating disorder; (vii) other specified feeding or eating disorder; and (viii) unspecified feeding or eating disorder. Each disorder can be specified according to remission status (partial or full) and severity level (mild, moderate, severe, or extreme). Obesity is not included in DSM-5-TR as a mental disorder. Obesity (excess body fat) results, instead, from the long-term excess of energy intake relative to energy expenditure. A wide range of genetic, physiological,

Table 1. Statistical comparison between American and Italian patients based on the first investigation

Variables	Mean	Variance	SD	SE	95% CI
USA: ALL (<i>n</i> = 135, 100%)					
Age at onset (years)	19.3	56.25	7.5	0.6	18.1–20.5
Age at first treatment (years)	25.9	73.96	8.6	0.7	24.5–27.3
Δ in age of onset and treatment (years)	6.7	64.0	8.0	0.7	5.3–8.0
Body weight (kg)	108.0	1,089.0	33.0	2.8	102.5–113.5
Maximum body weight achieved (kg)	137.0	1,296.0	36.0	3.1	131–143.1
Minimum body weight achieved (kg)	88.0	361.0	19.0	1.6	84.9–91.1
Δ between maximum and minimum body weight (kg)	48.0	841.0	29.0	2.5	43.1–52.9
USA: AN (<i>n</i> = 30, 22%)					
Age at onset (years)	19.0	31.36	5.6	1.0	17–21
Age at first treatment (years)	20.3	36.0	6.0	1.1	18.1–22.5
Δ in age of onset and treatment (years)	2.1	11.56	3.4	0.6	0.9–3.3
Body weight (kg)	79.0	100.0	10.0	1.8	75.5–82.5
Maximum body weight achieved (kg)	106.0	484.0	22.0	4.0	98.2–113.8
Minimum body weight achieved (kg)	70.0	100.0	10.0	1.8	66.5–73.5
Δ between maximum and minimum body weight (kg)	35.0	484.0	22.0	4.0	27.2–42.8
USA: BN (<i>n</i> = 62, 46%)					
Age at onset (years)	19.5	30.25	5.5	0.7	18.1–20.9
Age at first treatment (years)	28.0	36.0	6.0	0.8	26.4–29.6
Δ in age of onset and treatment (years)	8.4	67.24	8.2	1.0	6.4–10.4
Body weight (kg)	118.0	576.0	24.0	3.0	112.1–123.9
Maximum body weight achieved (kg)	145.0	900.0	30.0	3.8	137.5–152.4
Minimum body weight achieved (kg)	98.0	196.0	14.0	1.8	94.5–101.5
Δ between maximum and minimum body weight (kg)	49.0	676.0	26.0	3.3	42.5–55.5
USA: EDNOS (<i>n</i> = 43, 32%)					
Age at onset (years)	19.1	121.0	11.0	1.7	15.8–22.4
Age at first treatment (years)	26.9	121.0	11.0	1.7	23.6–30.2
Δ in age of onset and treatment (years)	7.6	82.81	9.1	1.4	4.9–10.3
Body weight (kg)	115.0	1,681.0	41.0	6.2	102.8–127.2
Maximum body weight achieved (kg)	144.0	1,936.0	44.0	6.7	130.9–157.1
Minimum body weight achieved (kg)	88.0	400.0	20.0	3.0	82.1–93.9
Δ between maximum and minimum body weight (kg)	57.0	3,249.0	57.0	5.6	46–68

(Cont'd...)

Table 1. (Continued)

Variables	Mean	Variance	SD	SE	95% CI
Italy: ALL (<i>n</i> = 31, 100%)					
Age at onset (years)	18.3	31.36	5.6	1.0	16.34–20.26
Age at first treatment (years)	20.9	33.64	5.8	1.0	18.94–22.86
Δ in age of onset and treatment (years)	2.6	3.24	1.8	0.3	2.012–3.188
Body weight (kg)	61.0	302.76	17.4	3.1	55–67
Maximum body weight achieved (kg)	80.0	316.84	17.8	3.2	73.7–86.3
Minimum body weight achieved (kg)	55.4	114.49	10.7	1.9	51.7–59.1
Δ between maximum and minimum body weight (kg)	24.8	219.04	14.8	2.7	19.5–30.1
Italy: AN (<i>n</i> = 7, 22.6%)					
Age at onset (years)	14.9	19.36	4.4	1.7	11.6–18.2
Age at first treatment (years)	16.0	18.49	4.3	1.6	12.9–19.1
Δ in age of onset and treatment (years)	1.1	0.81	0.9	0.3	0.5–1.7
Body weight (kg)	45.0	15.21	3.9	1.5	42–47.9
Maximum body weight achieved (kg)	70.7	313.29	17.7	6.7	57.6–83.8
Minimum body weight achieved (kg)	45.0	15.21	3.9	1.5	42–47.9
Δ between maximum and minimum body weight (kg)	25.7	196.0	14.0	5.3	15.3–36
Italy: BN (<i>n</i> = 7, 22.6%)					
Age at onset (years)	19.9	4.84	2.2	0.8	18.3–21.5
Age at first treatment (years)	22.9	7.29	2.7	1.0	20.9–24.9
Δ in age of onset and treatment (years)	3.0	2.89	1.7	0.6	1.8–4.2
Body weight (kg)	62.4	81.0	9.0	3.4	55.7–69
Maximum body weight achieved (kg)	82.4	110.25	10.5	4.0	74.6–90.2
Minimum body weight achieved (kg)	58.9	57.76	7.6	2.9	53.2–64.6
Δ between maximum and minimum body weight (kg)	21.2	47.61	6.9	2.6	16.1–26.3
Italy: EDNOS (<i>n</i> = 17, 54.8%)					
Age at onset (years)	19.1	43.56	6.6	1.6	16–22.2
Age at first treatment (years)	22.1	40.96	6.4	1.5	19.2–25
Δ in age of onset and treatment (years)	3.0	3.61	1.9	0.5	2.0–4.0
Body weight (kg)	67.1	380.25	19.5	4.7	57.9–76.3
Maximum body weight achieved (kg)	83.0	388.09	19.7	4.8	73.6–92.4
Minimum body weight achieved (kg)	58.3	127.69	11.3	2.7	53–63.6
Δ between maximum and minimum body weight (kg)	24.6	320.41	17.9	4.3	16.2–33

Abbreviations: ALL: All patients; AN: Anorexia nervosa; BN: Bulimia nervosa; CI: Confidence intervals; Δ: Difference or variation; EDNOS: Eating disorders not otherwise specified; SD: Standard deviation; SE: Standard error; USA: United States of America.

Table 2. Student's *t*-test comparison between American and Italian patients based on the first investigation

Variables	MDSE	df	<i>t</i> (1)	<i>t</i> (2)	<i>p</i>
ALL					
Age at onset (years)	1.196	164.0	0.914	<>	0.15 < <i>p</i> < 0.20
Age at first treatment (years)	1.277	164.0	1.979	<>	<0.05
Δ in age of onset and treatment (years)	0.387	164.0	3.255	<>	<0.05
Body weight (kg)	4.224	164.0	3.336	<>	<0.05
Maximum body weight achieved (kg)	4.452	164.0	3.578	<>	<0.05
Minimum body weight achieved (kg)	2.522	164.0	3.595	<>	<0.05
Δ between maximum and minimum body weight (kg)	3.647	164.0	2.522	<>	<0.05
AN					
Age at onset (years)	1.954	35.0	1.449	<>	0.05 < <i>p</i> < 0.10
Age at first treatment (years)	1.960	35.0	1.481	<>	0.05 < <i>p</i> < 0.10
Δ in age of onset and treatment (years)	0.714	35.0	1.183	<>	0.10 < <i>p</i> < 0.15
Body weight (kg)	2.345	35.0	3.808	<>	<0.05
Maximum body weight achieved (kg)	7.803	35.0	2.127	<>	<0.05
Minimum body weight achieved (kg)	2.345	35.0	3.265	<>	<0.05
Δ between maximum and minimum body weight (kg)	6.643	35.0	1.183	<>	0.10 < <i>p</i> < 0.15
BN					
Age at onset (years)	1.086	67.0	0.607	<>	0.20 < <i>p</i> < 0.30
Age at first treatment (years)	1.273	67.0	2.002	<>	<0.05
Δ in age of onset and treatment (years)	1.221	67.0	2.103	<>	<0.05
Body weight (kg)	4.567	67.0	3.489	<>	<0.05
Maximum body weight achieved (kg)	5.502	67.0	3.373	<>	<0.05
Minimum body weight achieved (kg)	3.378	67.0	3.402	<>	<0.05
Δ between maximum and minimum body weight (kg)	4.207	67.0	2.571	<>	<0.05
EDNOS					
Age at onset (years)	2.317	58.0	0.000	<>	>0.40
Age at first treatment (years)	2.285	58.0	1.449	<>	0.05 < <i>p</i> < 0.10
Δ in age of onset and treatment (years)	1.463	58.0	1.773	#	<0.05
Body weight (kg)	5.126	58.0	3.057	<>	<0.05
Maximum body weight achieved (kg)	8.237	58.0	2.721	<>	<0.05
Minimum body weight achieved (kg)	4.1	58.0	2.691	<>	<0.05
Δ between maximum and minimum body weight (kg)	9.716	58.0	1.826	#	<0.05

Note: *t*(2) refers to the two-tailed Student's *t*-test. Using the two-tailed test produces essentially the same results; however, two important differences should be noted, as indicated by # in the table.

Abbreviations: ALL: All patients; AN: Anorexia nervosa; BN: Bulimia nervosa; df: Degrees of freedom; EDNOS: Eating disorders not otherwise specified; MDSE: Mean deviation.

Table 3. Obese/overweight patients and their family history of obesity/overweight based on the second investigation

ID	Sex	Age (years)	Number of affected relatives	Diagnosis	Outcome
AA	Male	39	3	OB	–
AC	Female	57	0	OW	+
ADM	Female	39	0	OW	+
MD'O	Male	35	2	OB	–
MF	Female	42	0	OB	–
FF	Male	57	0	OB	–
AF	Male	36	0	OB	–
LG	Male	5	0	OB	+
PG	Female	28	0	OB	–
MG	Female	35	0	OB	–
GL	Male	45	0	OB	–
UL	Male	32	1	OB	–
RML	Female	64	0	OB	–
AM	Male	40	1	OW	+
MM	Female	38	3	OW	+
PP	Male	25	0	OB	–
DP	Female	33	3	OB	+
CP	Male	43	0	OB	+
FR	Male	36	2	OB	–
MCS	Female	63	0	OB	–
AS	Male	42	2	OW	–
AS	Female	40	0	OB	–
AMS	Female	31	4	OB	+
MLV	Female	8	1	OB	+
AA	Male	28	15	OB	+
AMC	Female	44	0	OB	–
TD'A	Female	37	1	OB	+
MTDR	Female	20	10	OB	+
RDR	Female	17	2	OB	–
IF	Female	59	9	OB	–
AI	Male	24	6	OB	–
AI	Female	25	7	OB	+
RL	Female	35	1	OB	–
MM	Female	75	0	OB	–
DM	Female	21	3	OW	+
GP	Male	17	9	OB	–
SP	Male	14	12	OB	–
LP	Male	13	12	OB	–

(Cont'd...)

Table 3. (Continued)

ID	Sex	Age (years)	Number of affected relatives	Diagnosis	Outcome
TP	Female	47	3	OB	–
GP	Female	46	3	OB	–
ER	Female	38	10	OW	–
AR	Female	48	2	OB	+
BS	Male	6	7	OB	–
GS	Female	20	2	OW	+
CS	Female	49	2	OB	–
LDM	Male	21	10	OB	–
GL	Male	32	7	OB	+
ML	Female	27	7	OW	+
RDM	Female	34	5	OW	–
AS	Female	46	1	OB	–
GS	Male	36	4	OB	–
RB	Female	26	1	OW	+
RMM	Male	36	3	OW	+
LP	Female	38	1	OW	+

Note: A positive outcome was considered to be maintenance of the achieved objective at two years of follow-up for subjects free from specific therapy, regardless of age, sex, and initial body weight; otherwise, all other cases were considered negative.

Abbreviation: OB: Obese; OW: Overweight.

Table 4. Patients who have successfully completed the addiction treatment process based on the third investigation

ID	Provenance	Registration date	1	1a	2	2a	3	3a	3b	4	4a	4b
GA	A. Ser.	March 3 rd , 2017			*	*						
WHM	A. Ser. / MHS	March 9 th , 2017	*	(*)	*	*			*	*	*	
CS	PP	March 16 th , 2017	*	*	*	*	*	*	*	*	*	*
GM	A. Ser.	March 17 th , 2017	*	*								
PP	PP	March 17 th , 2017	*	*	*	*	*	*		*	*	*
ZS	A. Ser.	March 17 th , 2017	*	*	*	*	*	*	*	*	*	*
GP	MHS	March 20 th , 2017	*	*	*	*						
MA	A. Ser. / MHS	March 20 th , 2017	*	*								
PF	Cit.	March 20 th , 2017	*	*	*	*						
SD	A. Ser.	March 20 th , 2017	*	*	*	*	*	*	*	*	*	(*)
CML	Cit.	March 23 rd , 2017			*	*			♦	♦	*	
SB	PP	March 23 rd , 2017			*	*	*	*				
TR	A. Ser.	March 23 rd , 2017			*	*			♦	♦	*	
BI	MHS	March 31 st , 2017					*	*		*	*	*
LA	MHS	March 31 st , 2017					*	*		*	*	*

Notes: 1: First meeting; 1a: Sub-project 1; 2: Second meeting; 2a: Sub-project 2; 3: Third meeting; 3a: Sub-project 3a; 3b: Sub-project 3b; 4: Fourth meeting; 4a: Sub-project 4a; 4b: Sub-project 4b. * indicates presence/participation; (*) indicates partial/incomplete presence/participation.

Abbreviations: A. Ser.: Addiction service; Cit.: Common citizen; MHS: Mental health service; PP: Private practice.

behavioral, and environmental factors that vary across individuals contribute to the development of obesity; hence, it is not considered a mental disorder in the DSM-5-TR.

The first study, which analyzed confounding factors in groups of males who were entirely similar and homogeneous, even if in two different geographical settings, demonstrated that eating disorders in males are comparable to those in females, albeit with significantly lower incidence and prevalence. However, complete overlap of multiparametric findings was observed only for AN, likely because its diagnostic criteria are more stringent. For other diagnoses requiring broader criteria, differences were evident across multiple parameters. Obesity, according to the dictates of the DSM-5-TR, has a wide range of causes and symptoms. The second investigation analyzed here demonstrates this, despite the author's belief that a psychological, if not strictly psychiatric, component is also present in this pathological condition. There are several theories attempting to explain the etiology of obesity, including genetic, familial, environmental, biopsychosocial, and psycho(patho)logical models, since Banting¹¹ published a work dedicated exclusively to the problem of excess weight. The third investigation further highlights these differences by treating individuals with treatment-resistant ("non-responder") feeding and eating disorders through an addiction-based approach. This strategy showed markedly different success rates and revealed variability in both causal factors and symptom presentation beyond the strictly nutritional or food-related context. The statistical dimension is undoubtedly predominant; however, neurobiological considerations and arguments are equally compelling. According to Balestrieri¹², for example, ethology can be properly defined only in epistemological terms, as the application of a fundamental hypothesis within a specific model to animal behavior, investigated through objective and comparative methods while considering the organism's natural relationship with its environment.^{13–16} Lambertini¹⁷ even stated: "How many strange inconsistencies and contradictions emerge as soon as we apply our principles of animal biology to the study of man!"¹⁷(p2) However, caution is necessary, as Biondi emphasizes: these comparisons and analogies concern only the type and form of behavior, and do not imply equivalence in the identity or meaning of the behaviors being compared.¹⁸ Although the ultra-elemental representation of living matter coexists with the systemic instance that assumes the organism as a dynamic, self-regulating "whole," it may initially appear difficult to reconcile the global properties of living systems within modern biology. These properties were traditionally recognized primarily through processes such as nutrition,

metabolism, and biological purpose. It appears that the qualities once considered intrinsic to each species and to each individual within a species are now largely attributed to the knowing subject: the human mind, which, according to various criteria, classifies and differentiates what may appear identical at the level of elementary structure.¹⁹

Sigmund Freud²⁰ appears to have addressed the problem primarily in females; however, Breuer, who had also observed similar cases (including the hysterical manifestations of Anna O.), described and analyzed a male pediatric case: a 12-year-old boy who was already affected by *pavor nocturnus*.²¹ He also reported observations that could be interpreted as challenging certain modern psychiatric theories suggesting that the loss of "appetite" occurs only during the terminal stages of the illness.²¹ The "hysterical nature" of Anna O., diagnosed by Breuer, was not limited to classic anorexia for food, but implied "motivations" for an obstinate refusal even to drink, and not for water or the liquid itself; this is a fact of extreme interest. This line of investigation began with Freud's correspondence with Fliess in the late 1880s but received little further attention after 1895.²² The following century, however, was characterized by extensive research: in 1903, Janet revisited the theory of the female sexual role while studying "psychasthénie" and describing a case of "obsession de la honte du corps." Almost 20 years later, studying sadism, Abraham²³ introduced the concept of "ambivalence" into the study of the genesis of eating disorders, also starting from Freud; very important concepts were developed such as the castration complex, penis envy, and a topic of extreme interest: the nutrition–defecation relationship. For the most part, and with some notable exceptions, the path initiated by Freud was continued and further developed. His daughter, Anna Freud, examined eating disorders first in relation to ego defense mechanisms, then in the drives, impulses, and fantasies of the id, and ultimately in the simple "existence" of love objects associated with past experiences, including both Oedipal and pre-Oedipal dimensions. From the concept of the male castration complex, through the somato-pathological influences on the psychic life of the child²⁴, she arrived, in the mid-1940s, at the analysis of renunciation behaviors, developing a huge amount of clinical material on eating disorders.

During approximately the same period, Klein focused primarily on anxiety-related defense mechanisms, which she distinguished into a schizophrenic modality (the body experienced as fragmented) and a depressive modality (associated with the loss of the object). Anorexia, for example, was attributed by Klein to the former, conceptualized as a form of "intrapsychic paranoia".²⁵ The split, in fact, would affect the separation between "good" and "bad" objects, between the "loving" ego and the

“hating” ego, and in Freudian terms, between the “life” instinct and the “death” instinct (or drive).²⁶ More recently, the most accredited theories were proposed by Bruch (for her, the inevitable change linked to adolescent development could be experienced as a major narcissistic trauma²⁷; Freud considered it paradoxical as a “victory” experienced with “very painful” sacrifice), by Palazzoli-Selvini²⁸ (for whom the extreme sexual components should be scaled down), and by Minuchin²⁹ (who supports the importance of development in the family “corpus”). However, the merit of highlighting the importance of the narcissistic component in the relationship between bodily control and ego adaptation belongs to Kestenberg.³⁰ In anorexia, this need for control may assume a megalomaniacal quality. The “typical” condition was defined as a set of eating disorders that usually manifest in otherwise healthy young women, conditioned by an obsessive fear of gaining weight.³¹ Thus defined, these disorders apparently predominate among the white race and, within certain limits, a middle-class population. The scarcity of cases among the poorest classes was compared with the prevalence among males; however, actual estimates of incidence and prevalence are not yet available.

Specific studies aimed at identifying a potential population at risk have highlighted an age range of 16–18 years among young adolescents attending private schools, in whom prevalence may reach up to 1%. Of great psychological importance is the fact that samples with the same characteristics but attending public schools showed prevalence estimates hovering around 0.1%. A study conducted in Rochester, Minnesota, recorded similar prevalence rates (0.2%) in the male population (0.02%).³² According to particularly authoritative scholars in this field, such as Bruch²⁷, Palazzoli-Selvini²⁸, and Ushakov³³, this rate could be increased up to 10%, 14%, and 20%, respectively. In Ushakov’s experience³³, which involved certain stages of prepubertal development in males, this trend was reversed, with a slight predominance among male subjects. Subsequently, a large-scale study involving a substantial sample ($n = 6,728$) identified disordered eating (“binge–purge cycling”) in 13% of females and 7% of males, while an eating disorder diagnosis was reported in 24% of females and 15% of males.³⁴ The incidence, adjusted for sex and age, in the aforementioned Rochester study would amount to approximately 7.3 cases per 100,000 inhabitants per year.³²

Although extremely rare, cases of anorexia have been reported in the post-adolescent phase, among prepubescent girls (consistent with Ushakov’s observations), and even in infants. These presentations are generally associated with a poorer prognosis. Infantile anorexia, in particular,

was studied by Debré *et al.*³⁵, Doumic³⁶, and Lelong and Kreisler³⁷ and described as “opposition anorexia.” Doumic³⁶, in particular, described very severe cases in early childhood. According to Kreisler³⁸, the forms of late childhood and adolescence would be less severe and more accessible to psychotherapy. In newborns, as well as in infants, it is possible that a generic eating disorder represents, nothing less, a “symptom” of arterial hypertension.³⁹ Data relating to BN are less confirmed. Studies conducted in Ontario, Canada, have reported prevalence estimates of approximately 1% in females and 0.1% in males.⁴⁰ In the United States, however, recent studies indicate that the prevalence of bulimia in high school and university students may vary between 3.8% and 9%.⁴¹ The same authors reported an age of onset of between 16 and 20 years, therefore later than that of AN, and a gender preference of around 85% of female subjects.³⁷ Furthermore, the estimates relating to the incidence rates are very disturbing (18% of female students in an American college reportedly presented post-prandial vomiting: suffice it to say that in Italy, some authors have preferred to include a form of “schoolboy vomiting” among the psychosomatic disorders “determined” by “psychic suffering”⁴²). Rather summarily, it is believed that bulimia is slightly more frequent than anorexia; in any case, the comparison with previous data reported in the literature shows an increase in both the incidence and prevalence of both disorders. In summary, the literature still appears to be influenced by the marked predominance of the disorder among females. Nevertheless, the available data can be considered broadly comparable, and it may reasonably be assumed that the male-to-female ratio is approximately 1:10, as consistently reported, at least when using the diagnostic tools and interpretative frameworks currently available.

These findings provide a useful point of reflection for future clinical studies: among males affected by eating disorders, there appears to be a relative predominance of atypical presentations. The paradoxical peculiarity of these forms must be correctly indicated, and precisely, on the basis of “atypicality”⁴³, as the literature may contain potentially erroneous adjectives such as “partial” or “subclinical”.⁴⁴ Cassano and Pancheri⁴⁵ also reported other possible expressions found in the literature, including “soft,” “subsyndromic,” “subthreshold,” “with limited symptomatic expression,” “incomplete,” and “frustrated.” Other expressions, such as “subliminal” and similar terms, can also be found. The study conducted by Canton and Giannini⁴⁶ on “personality factors” is also interesting. The pathological situation was defined on the basis of “incorrect eating behavior disorders,” while the “personality traits” allowed for the establishment of two

large groups of obese patients: the “restrictive eaters” and the “free eaters.”⁴⁶ The findings of a study conducted in a sample of 124 Italian women diagnosed with feeding and eating disorders are particularly noteworthy.⁴⁶ Participants were assessed using a psychometric battery during observation and semi-residential treatment. Patients with BN exhibited greater psychological distress than those with other eating disorder diagnoses or obesity; however, this difference was not significantly attributable to comorbidity with personality disorders. Across all disorder subtypes, negative affectivity, emotional detachment, and purging behaviors were significantly associated with psychological distress. Furthermore, emotional dysregulation was found to indirectly influence the relationship between self-esteem and purging behaviors.⁴⁷ Equally interesting is the conclusion reached in another very recent randomized controlled trial: intensive lifestyle modification, which includes calorie-carbohydrate restriction and time-restricted feeding, improved the psychopathology of eating disorders.⁴⁸

Regarding the interpretation of feeding and eating disorders as addictive disorders, one of the most significant contributions, following Freud’s early insights and providing a basis for the present discussion, is the work of Therese Benedek.⁴⁹ In 1934, she published a seminal paper entitled “Dominant Ideas and Their Relation to Morbid Cravings”⁴⁹, which already included extensive consideration of food consumption as a potential source of addictive behavior. Otto Fenichel, in his famous *The Psychoanalytical Theory of Neurosis*⁵⁰, wrote that the mechanisms and symptoms of pathological cravings can also occur without the use of any drugs, and therefore without the complications due to the chemical effects of drugs. A special category is represented by the above-mentioned food cravings, which include many different types. In severe cases, eating remains the only interest that connects the person to reality. And then, a little further on, he even claims that there is a characteristic relationship between the desire for food and phobias concerning eating or certain types of anorexia: here, evidently, he already resolves the apparent paradox by intuiting how the solution to food cravings is found precisely in the denial of the craved substance.

Kohut^{51,52}, developing the concepts of a psychoanalysis of addictions, emphasized that the central role of narcissism is equally implicated in eating disorders, especially those with body dysmorphic disorder. Additionally, more than 15 years ago, following a series of public presentations entitled “Old and New Addictions as Two Sides of the Same Coin” held in several schools in the province of Salerno (Italy), the concept was introduced—and subsequently published—

that eating disorders represent a highly complex and heterogeneous group of phenomena. These disorders can be classified as follows: obesity, anorexia nervosa, bulimia nervosa, binge eating disorder, rumination disorder, and pica. The various behaviors associated with each disorder recognize a common denominator: food. Everyone understood it as profoundly disconnected from the world of diet, food, and nutrition; rather, it was attributed a substitutive, or at least coextensive, meaning, elevating it to the highest level of emotions and even thoughts. In this sense, it became perceived as irreplaceable, indispensable, and therefore inevitable—unless interpreted through its very absence, as a path from dependence to freedom through fasting, vomiting, purging, and restriction. Gradually, the circuits feed on themselves, leading the poor subject into a state of mortifying chronicity: this, therefore, is the key to understanding the dimension of addiction. All of this, which is not illegal, nevertheless appears to be potentially life-threatening. Added to these considerations is the complex issue of the body.⁵³

The same author later presented, at a national conference in 2017, the relationship between substance addiction and anorexia in rare syndromic conditions, which was subsequently published in a specialized journal.⁵⁴ Contemporary neurobiology has demonstrated, through positron emission tomography, that dopaminergic neurotransmission in the corpus striatum, due to anomalous expression of the receptors involved, is practically superimposable in subjects with cocaine, methamphetamine, or alcohol addiction and morbid obesity.⁵⁵ These findings have justified, over time, the proposal of food addiction for some eating disorders. Pacifici and Palmi⁵⁶, at the Italian Higher Institute of Health, have argued that narcotics and foods with a high salt, sugar, and fat content act in a very similar way: once ingested, they follow the same neurological circuits to reach the brain areas responsible for gratification. However, amino acids and proteins, through supplement abuse and in specific populations, have also been shown to generate pathological addiction. Other authors have simultaneously argued, invoking the lack of evidence of foods with properties sufficient to cause addiction, that it is more appropriate to consider the behavior, rather than the substance, underlying the actual addiction, thus proposing the concept of “eating addiction.”⁵⁷ In this way, they also seem to lean toward the inclusion of all pathological eating behaviors in the spectrum of addictions and not just overeating. The ability to “cause pathological addiction” would not be the exclusive prerogative of foods as substances but would also extend to behavior itself, in direct analogy with gambling and other behavioral addictions. Studies conducted using functional

magnetic resonance imaging have also demonstrated that stimuli from drugs and those from foods activate the same brain regions (amygdala, insula, orbitofrontal cortex, and corpus striatum). These experiments, in humans, have been summarized by Blumenthal and Gold.⁵⁸ The analysis of BN, in fact, has shown similarities and correlations, at the behavioral level, with what is observed in addictions, especially those to substances: concern about the lack of the object of desire, loss of control, and craving, above all. The suggestive comorbidity between disorders of this type and substance abuse in general cannot be underestimated: high percentages of overlap have also been widely documented (a recent review by Harrop and Marlatt estimates them at up to 46%).⁵⁹

Eating disorders, just like substance use disorders, once again, in general, involve the opioid-type neurotransmitter systems; increasingly evident analogies appear between the personality of patients with eating disorders and those with substance abuse (propensity to impulsivity, for example). According to von Ranson and Cassin⁶⁰, who fully shared the concepts illustrated above, approximately one third of treatment programs for eating disorders are based on principles already effective in the treatment of addictions. The increased daily use of technological devices (digital addictions) appears to be more frequently associated with risky eating behaviors. Almost simultaneously, Szasz⁶¹ and Glatt⁶² introduced, by analogy with drug addiction, the concepts of “food mania” and the distinction between the “compulsive overeater” and the “simple” or “social” obese individual. Shortly thereafter, related concepts such as “dietomania” and “satiomania” also became widely discussed.^{61,62}

Marazziti *et al.*⁶³ included certain behavioral deviations, such as orthorexia in eating practices and overtraining syndrome in sports activities, in their article. In this regard, the phenomenon of doping and its related addictive patterns should also be considered, together with specific conditions such as bigorexia, vigorexia, and compulsive anabolic behavior. The implications of reward deficiency syndrome are also relevant in this context. Furthermore, some authors have suggested that alcohol may be considered a form of food and approached accordingly, a perspective that would have significant hermeneutic and therapeutic implications. Grilo's group has underlined⁶⁴ precisely the evident analogies between problematic behaviors in the food sphere and those related to alcohol. There is also no shortage of bold studies on animals with complicated ethanol extracts capable of producing anorectic effects⁶⁵; similarly, the molecular mechanisms implicated in the opioid system and in the pharmacology of cocaine have been studied.⁶⁶ Finally, suggestively, there is a global association known as “Overeaters Anonymous,” a striking

analogy to “Narcotics Anonymous” and “Alcoholics Anonymous.” For them, food is a drug; they lose control; in half an hour, they can ingest 30,000 calories, the equivalent of seven panettone cakes, declared the director of an Eating Disorders Center in Umbria in an interview.⁶⁷ Furthermore, the evolution observed in the field of eating disorders resembles that previously seen in substance use disorders, where severe and pervasive dependence has gradually been accompanied by more episodic patterns of use, such as “Saturday night” consumption of even potent substances. Although these patterns may appear less destructive, they can be highly insidious and carry significant risks.⁶⁸

Most of the drugs once used to treat obesity, BN and binge eating disorder, almost all of which were subsequently withdrawn from the market, were amphetamine-based. More recently, the orexigenic effects of tetrahydrocannabinoids have been demonstrated.⁶⁹ Rimonabant, however, in the very short time it remained on the market, was used to reduce appetite, food cravings, and treat complex obesity. It was, in fact, a cannabinoid receptor antagonist. Similarly, the effects of cocaine in increasing tolerance to hunger and fatigue are well documented. A review of recent therapeutic developments also highlights that some of the newest drugs approved for the management of complicated obesity have mechanisms with notable similarities to those of psychotropic medications used in the treatment of substance addictions.⁷⁰ For example, Mysimba* contains a combination of bupropion, a medication widely used in smoking cessation therapy, and naltrexone, an agent commonly used in the treatment of opioid and alcohol dependence. Topiramate, certainly effective in alcohol use disorder and currently being tested for the treatment of cocaine abuse, shows widely recognized positive effects in the treatment of complicated overweight, alone and in combination with phentermine. Samidorphan (ALKS33), a selective antagonist of the μ -opioid receptors, is gaining indications for the treatment of opioid, alcohol, and cocaine addiction, as well as Binge eating disorder.⁷¹ Similarly, interventions developed over time to promote orexigenic effects have generally involved centrally acting drugs, although through different receptor mechanisms, often involving dopaminergic pathways. As discussed above, the reward circuit can be artificially activated by drugs or stimulated through implanted electrodes; however, it can also be activated by naturally rewarding behaviors, such as eating. The answer is clearly affirmative.⁷² It follows that multimodal and integrated approaches, typical of addiction clinics, are probably more fruitful than mono-modal approaches. We could therefore say that there is an isomorphism of therapeutic models and approaches.

It follows that Addiction Services could contribute to a potential network of public facilities dedicated to the management and treatment of eating disorders. This perspective also invites consideration of concepts such as harm reduction, traditionally applied to addiction disorders, in relation to eating disorders. Addiction Services that adopt these models may be particularly well positioned to assume such a role, given the multidisciplinary expertise of their teams and their established practice of integrating multiple therapeutic approaches, which is characteristic of addiction care.⁷³ During the Alcohol Information Groups periodically organized at the Addiction Service of Cesena, the psychiatric contribution did not omit the discussion of alcohol as a food.⁷⁴

5. Conclusion

Once a diagnosis of feeding and eating disorder is made, the effectiveness of treatment, the relapse rate, and the prognosis do not depend on the diagnosis itself or the appropriateness of the treatment, but rather on chronological and geographical factors, often still unclear, rather than on how much and what one eats or how one behaves. The differences, often substantial, concern mainly gender and cultural background. Specialist treatments, given the same diagnosis and severity of the diagnosed disorder, do not currently appear to be standardizable, but can be effectively personalized on the basis of these differences.

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Further disclosure

Parts of or the entire set of findings have been presented at the following conferences, academic meetings, and congresses:

- (i) Polichetti GBI. *I disturbi del comportamento alimentare nei maschi* [Eating disorders in males]. Degree Thesis in Medicine and Surgery, University of Naples “Federico II”, June 28, 2002 [in Italian].
- (ii) Polichetti GBI. *I disturbi alimentari* [Eating Disorders]. Presentation for schools. Chieti, May 3, 2005 [in Italian].
- (iii) Polichetti GBI. *Dipendenza e legalità: progetto per le scuole* [Addiction and Legality: A Project for Schools]. Salerno: January 14, March 17, and April 22, 2008 [in Italian].
- (iv) Polichetti, GBI. *Genesi, epigenetica, entanglement e psico(pato)logia nel disordine alimentare* [Genesis, epigenetics, entanglement and psycho(patho)logy in eating disorders]. Thesis, Advanced University Training Course in Psycho-neuro-endocrine-immunology, Bologna, July 7, 2010 [in Italian].
- (v) Polichetti GBI. *Terapia psico-comportamentale, psiconeuroendocrinoimmunobiologia dell'obesità e aspetti psico(pato)logici peculiari* [Psychobehavioral therapy, psychoneuroendocrinoimmunology of obesity and specific psychopathological aspects]. Presentation at the Maurizio Bufalini Hospital in Cesena. October 16, 2010 [in Italian].
- (vi) Polichetti GBI. *Dipende da come mangi* [It depends on how you eat]. Presentation at the XVIII National Course of the ADI (Italian Association of Dietetics and Clinical Nutrition). October 24-26, 2019 [in Italian].

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