

Systematic Review

Core Symptoms of Eating Disorders and Heart Rate Variability: A Systematic Review

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Abstract

(1) Eating disorders (EDs), characterized by symptoms such as restrictive eating, binge eating, compensatory behaviors, and emotional dysregulation, are associated with autonomic nervous system dysregulation, which may contribute to cardiovascular complications. This review systematically examines the relationship between heart rate variability (HRV) and core ED symptoms to identify specific autonomic patterns linked to behaviors like fasting, binge eating, and emotional dysregulation. (2) A total of 16 cross-sectional and longitudinal studies were narratively synthesized following PRISMA guidelines. All studies were selected from the Science Direct, PubMed, Web of Science, and Scopus databases. (3) Findings indicate that individuals with anorexia nervosa exhibit blunted sympathetic reactivity and reduced parasympathetic flexibility, particularly during stress or physical activity, with HRV measures normalizing after weight restoration. In contrast, binge eating and loss-of-control eating are associated with lower resting HRV, which correlates with the severity of eating behaviors. Reactive HRV also varies with food cues and emotional states, showing complex autonomic responses in individuals with EDs. Emotional dysregulation, consistently marked by reduced high-frequency HRV, is a key feature across these disorders. (4) These results suggest that HRV patterns may serve as physiological markers of ED symptomatology, offering insights for targeted interventions aimed at improving both emotional regulation and cardiovascular health in affected individuals.

Keywords: eating disorders; heart rate variability; emotional regulation; stress; autonomic imbalance



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1. Introduction

A contemporary dimensional framework highlights the value of pinpointing transdiagnostic traits and core behavioral symptoms that transcend traditional diagnostic categories, as advocated by the Research Domain Criteria initiative [1]. Eating disorders (EDs) are a group of psychiatric conditions characterized by an excessive preoccupation with body weight and shape—manifesting in core symptoms such as restrictive eating, binge eating (BE), compensatory behaviors (e.g., purging), and pervasive body dissatisfaction—which together reflect a dysfunctional and emotional relationship with food [2,3]. These disorders are associated with a range of medical complications—particularly severe and life-threatening cardiovascular problems—where core symptoms such as restrictive eating, binge eating, and emotional dysregulation may contribute to arrhythmias, hypertension, systolic dysfunction, and, in the most critical cases, sudden cardiac death [4,5].

In anorexia nervosa (AN), core features include severe food restriction and compulsive exercise. Restrained eating refers to self-imposed dieting rules and heightened self-control to regulate eating behavior with the goal of weight control [6] (for a review, see [7]). Additionally, elevated physical activity may reflect both intentional weight control and a compulsive drive for activity [8]. In contrast, binge eating disorder (BED) and bulimia nervosa (BN) are characterized by the presence of recurrent BE episodes, which involve two fundamental elements: the experience of being unable to stop eating, known as loss-of-control (LOC) eating, and the consumption of unusually large amounts of food, referred to as overeating [2,9]. Furthermore, LOC has been associated with negative health outcomes, serving as a key indicator of the severity of overeating episodes [10] and contributing to weight gain [11], as well as to the development of disordered eating attitudes over time [12]. Individuals with binge-type EDs exhibit heightened responsivity to food-related cues [13] and impairments in inhibitory control [14].

Beyond their distinct behavioral manifestations, EDs share transdiagnostic features that may play a central role in both their development and persistence. Two particularly relevant dimensions are emotion dysregulation [15–17] and stress [18,19], both of which appear to be shared vulnerabilities across ED subtypes. On the one hand, regarding emotion dysregulation, individuals with EDs commonly exhibit difficulties in identifying, accessing, and applying adaptive emotional regulation strategies [16,20–22]. Instead, they often rely on maladaptive strategies [23] such as suppression, avoidance, or rumination [19], which can perpetuate disordered eating behaviors [22,24]. On the other hand, stressful life events and prolonged psychosocial stress are frequently reported prior to the onset of the disorders and are known to exacerbate core symptoms [25,26]. In fact, stress is closely linked to emotional dysregulation and LOC behaviors, as it can disrupt self-regulatory capacity and modulate eating patterns through changes in physiological arousal and self-regulatory capacity [19,27].

Taken together, the high incidence of cardiovascular complications in EDs, along with the prominent role of emotion dysregulation and stress, points to a potential shared underlying mechanism: an autonomic nervous system (ANS) dysfunction. Dysfunction in the sympathetic and parasympathetic branch systems—such as autonomic imbalance—can contribute to the onset of both psychiatric and cardiovascular disorders [28,29] and may persist despite normalized weight [28]. For instance, the role of psychosocial stress in the onset and maintenance of AN, as well as its effects on ANS functionality, has been frequently thematized [30,31]. Additionally, patients with AN and BN have shown a blunted sympathetic activity under resting [32] and acute stress conditions [33]. Moreover, patients with EDs have shown dysregulations of the hypothalamic–pituitary–adrenal (HPA) axis [34], as occurred in underweight patients who showed an exaggerated cortisol awakening response, whereas weight-restored patients had normalized HPA axis activity [35].

In recent years, heart rate variability (HRV) has emerged as a pivotal non-invasive biomarker for neuropsychiatric disorders, capturing the dynamic interplay between sympathetic and parasympathetic activity and offering unique insight into heart–brain connectivity and emotional regulation capacity [36,37]. Specifically, high resting HRV is associated with parasympathetic dominance and greater flexibility in emotional and physiological regulation, whereas low resting HRV has been linked to sympathetic dominance, emotional rigidity, and increased risk for psychiatric and somatic disorders [38,39]. Studies have demonstrated that extreme HRV levels are associated with various psychopathologies, including EDs (for a review, see [40]). Thus, elevated HRV—indicative of heightened parasympathetic tone—has been reported in individuals with AN and BN [41] and may be related to behaviors such as fasting and purging [40,42]. In contrast, other studies have documented reduced HRV in EDs [43,44]. Moreover, some studies did not find differences

in resting-state HRV between individuals with and without BED [5,45,46]. In addition, cue-elicited HRV changes have been linked to disordered eating behaviors [47,48]. Nevertheless, the evidence remains inconsistent. For instance, Nederkoorn et al. [49] reported that exposure to palatable food increased low-frequency (LF) HRV in normal-weight women without affecting vagal measures, whereas Udo et al. [50] found that obese individuals exhibited higher high-frequency (HF) HRV when viewing food images. Similarly, resistance to high-calorie foods has been associated with increases in RMSSD [51], underscoring the complexity and context-dependence of HRV responses in eating-related paradigms. Therefore, HRV offers a promising physiological marker aligned with Research Domain Criteria principles, particularly within the arousal and regulatory systems domain (basic in EDs). Thus, reduced HRV has been consistently associated with poor emotional regulation, heightened anxiety, and impaired cognitive flexibility commonly observed in ED symptomatology. By linking HRV metrics to these transdiagnostic domains, researchers can move beyond categorical diagnoses and better characterize the autonomic correlations of individual differences in ED symptoms.

Although existing reviews have confirmed ANS dysregulation in individuals with EDs, they have not clarified whether this dysregulation is driven by specific ED symptoms [40]. For example, fasting—a core feature of AN and BN—has been implicated in the parasympathetic dominance observed in these populations. A symptom-focused approach could reveal how the nuclear symptoms of EDs map onto distinct patterns of autonomic activation—such as heightened sympathetic arousal in response to food cues or parasympathetic suppression during exposure to body image- or control-related stimuli. HRV captures these patterns, enabling us to probe the physiological mechanisms underlying clinical symptoms. By examining resting HRV in relation to specific behaviors—restrictive eating, binge eating, compensatory behaviors, and emotional dysregulation—this review moves beyond categorical diagnoses to identify which core symptoms most strongly predict ANS dysfunction. Ultimately, a transdiagnostic, symptom-driven HRV framework may not only clarify the links between disordered eating behaviors and autonomic imbalance but also open new avenues for assessing cardiovascular risk and tailoring interventions to target the mechanisms that underlie dysregulation, thereby improving clinical outcomes.

This review systematically examines the empirical literature on HRV in relation to the core symptoms of EDs—restrictive eating, binge eating, compensatory behaviors, and emotional dysregulation—with the goal of uncovering the specific symptom(s) that drive autonomic nervous system dysregulation. By moving beyond descriptive associations between baseline HRV and diagnostic categories, we aim to elucidate the physiological mechanisms linking disordered eating behaviors to cardiovascular risk and to develop an integrative framework that addresses both the psychological and physiological dimensions of these disorders. Ultimately, identifying symptom-specific HRV patterns will support more targeted, individualized interventions and improve clinical and cardiovascular outcomes.

2. Materials and Methods

2.1. Search Strategy

A systematic literature search was conducted in accordance with PRISMA guidelines [52] across four major databases: PubMed, Web of Science, Scopus, and ScienceDirect. The following keywords were used to identify relevant studies: “bulimia”, “bulimia nervosa”, “anorexia”, “anorexia nervosa”, “binge eating”, “binge eating disorder”, and “eating disorder”, combined with “HRV” and “heart rate variability” to retrieve research addressing eating disorders and heart rate variability. Boolean operators were used to interlink terms effectively.

The comprehensive literature search was conducted in May 2023. An updated search was performed in April 2025 to ensure the inclusion of the most recent studies. Additionally, a manual screening of reference lists and other sources was carried out to identify any possibly eligible studies that may not have been retrieved through database indexing.

2.2. Study Selection

The inclusion criteria considered empirical studies published between 2013 and 2023, written in either English or Spanish. Eligible studies included participants diagnosed with an eating disorder (e.g., AN, BN, or BED) or presenting subclinical symptoms consistent with disordered eating behaviors. Only studies that examined HRV as a primary or secondary outcome were included. Both cross-sectional and longitudinal designs were considered, provided they included human participants and were published in peer-reviewed journals.

Exclusion criteria comprised animal studies, theoretical or review articles (including systematic reviews and meta-analyses), single-case reports, book chapters, and manuals. Studies were also excluded if they did not include HRV measures, or if the population under study did not present any current or past symptoms related to eating disorders. Articles published in languages other than English or Spanish, or those without access to the full text, were excluded unless sufficient data were available from the abstract or supplementary sources.

Two independent reviewers (A.A. and M.A.S.) independently screened the search results from each database, evaluating studies based on their abstracts and full-text content. Studies were included only when both reviewers reached a consensus. In cases of disagreement, a third reviewer (N.S.M.) was consulted to mediate and resolve discrepancies through discussion.

2.3. Data Extraction

After the initial selection of studies, data extraction was carried out by the first author (A.A.). For each article that met the final inclusion and exclusion criteria, the following information was systematically collected: (1) authorship and publication details; (2) type of ED addressed and symptomatology described; (3) characteristics of the sample, including size and population type; (4) HRV assessment details, including the specific parameters measured; and (5) main findings related to HRV and eating disorder symptomatology.

2.4. Risk-of-Bias Assessment

The risk-of-bias assessment was conducted by the authors (N.S.M. and M.A.S.) for the included studies using the revised critical appraisal tools developed by the Joanna Briggs Institute (JBI). Different JBI checklists were applied depending on the study design. Comparative cross-sectional studies were evaluated using the Checklist for Analytical Cross-Sectional Studies. Quasi-experimental studies—including those with pre-existing groups and embedded experimental manipulations—were assessed using the Checklist for Quasi-Experimental Studies. Experimental studies with within-subject designs and no control group were also evaluated using this checklist, due to the lack of a more specific tool for this design. The randomized controlled trial was appraised using the Checklist for Randomized Controlled Trials. Finally, studies employing longitudinal observational designs or ecological momentary assessment were evaluated using the Checklist for Cohort Studies.

3. Results

A comprehensive database search yielded a total of 7768 bibliographic records, with 203 from PubMed, 5246 from ScienceDirect, 1862 from Scopus, and 457 from Web of Science. After removing duplicates, 7372 records remained for screening. Based on title and abstract

review, 6786 articles were excluded due to lack of relevance to the topic or population of interest.

Subsequently, 330 full-text articles were assessed for eligibility. Of these, 233 were excluded for not including all variables of interest, 49 for evaluating unrelated variables, and 16 for not reporting HRV measures adequately or failing to specify them. As a result, 16 studies met the final inclusion criteria and were included in the systematic review. The full selection process is detailed in Figure 1.

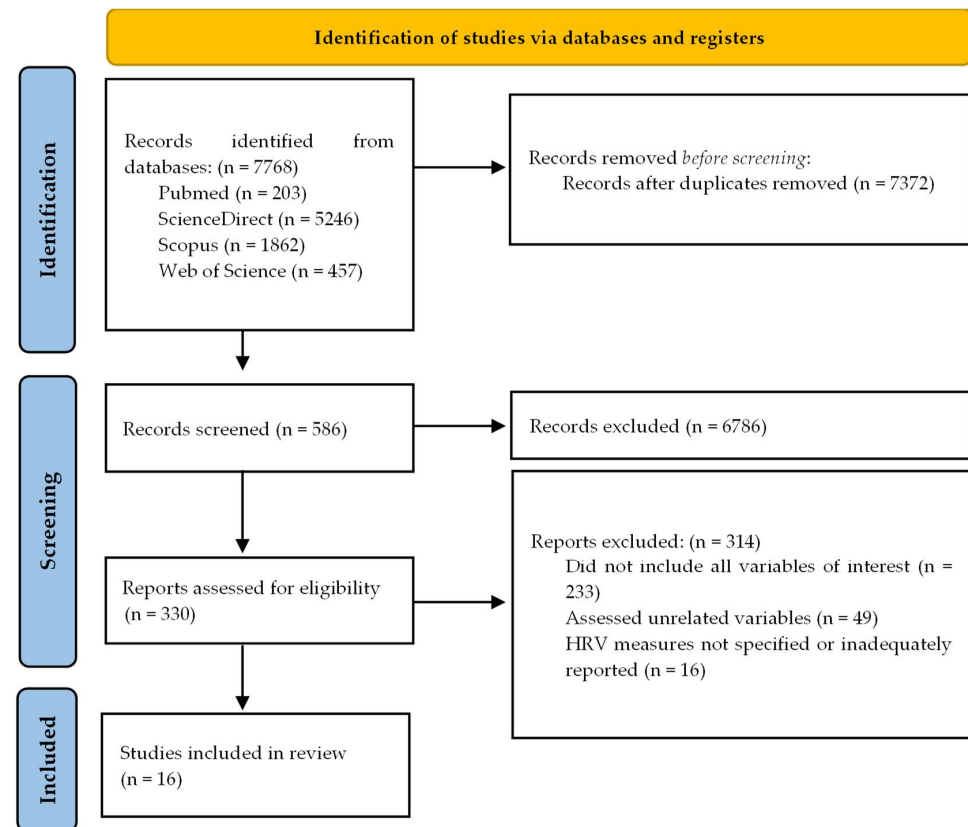


Figure 1. PRISMA 2020 flow diagram for new systematic reviews which included searches of databases and registers only.

3.1. Symptomatology Measurements

Among the 16 included studies, 9 examined HRV in relation to emotional regulation, including those by Bottera et al. [53], Godfrey et al. [54], Het et al. [55], Juarascio et al. [56], Mehak et al. [57], Ortmann et al. [58], Ranzenhofer et al. [59,60], and Rommel et al. [61]. Four studies explored HRV in connection to LOC eating, specifically Godfrey et al. [54], Parker et al. [62], and Ranzenhofer et al. [59,60]. Three studies focused on weight regain and nutritional status, particularly Jenkins et al. [28], Paysal et al. [63], and Schmalbach et al. [64]. Billeci et al. [65] evaluated physical activity, and Mehak et al. [57] addressed the somatic experience of being overweight. Finally, Chang et al. [66] focused on food cue reactivity, while Geisler et al. [67] assessed restrained eating.

Furthermore, three studies specifically evaluated stress response using standardized induction tasks. Godfrey et al. [54] examined physiological reactivity to an acute cognitive stressor, applying a mental math task to elicit stress. Het et al. [55] implemented the Trier Social Stress Test (TSST) to evaluate autonomic and hormonal responses to psychosocial stress in patients with EDs. Similarly, Schmalbach et al. [64] utilized the same task to investigate ANS reactivity to social-evaluative threat in individuals with AN.

Table 1 summarizes the sixteen studies included in the systematic review.

Table 1. A summary of the sixteen studies included.

Study	Sample Characterization					Diagnosis Criteria	ED Symptoms	Main HRV Results	Effect Size
	Type	N	% Female	Age (M ± SD)	BMI (M kg/m ² ± SD)				
Billeci et al. [65]	Adolescent girls	40	AN-RT (<i>n</i> = 23)			DSM-IV and DSM-5 criteria: AN-RT	Physical activity	AN-RT: RMSSD/SDNN stable; LFn ↑ slightly during task, ↓ at recovery; HFn ↓ during task, ↑ at recovery; LF/HF ↑ during task	Moderate to large
			100%	15.2 ± 1.9	15.7 ± 1.6				
			HC (<i>n</i> = 17)						
Bottera et al. [53]	Males and females with or without OBE	86	100%	15.7 ± 2.1	21.7 ± 2.8	DSM-5 criteria: Modified EDDS to assess past-month and lifetime OBE and EDE-Q	Emotional regulation and BE	HC: RMSSD/SDNN ↑ during task, ↓ at recovery; LFn ↓; HFn ↑; LF/HF stable	Small
			OBE+ (<i>n</i> = 34)						
			73.50%	22.26 ± 5.89	24.01 ± 5.36				
Chang et al. [66]	General non-clinical sample individuals	99	OBE- (<i>n</i> = 52)			BITE and EDEQ	Reactivity to food	No SDNN/HF response to food cues; LF/HF reactive to high-caloric vs. neutral stimuli and inversely correlated with symptom severity	Not reported
			74.50%	20.48 ± 2.92	23.27 ± 3.95				
			61.61%	28.77 ± 6.04	23.06 ± 4.59				
Geisler et al. [67]	Undergraduate students	111			Ego depletion (<i>n</i> = 55)	German Restraint Scale	Restrained eating, self-control in response to food cues	↑ RMSSD linked to eating restriction; restraint score predicted HRV post-ego depletion only	Moderate
			77%	23.06 ± 4.50	23.67 ± 3.88				
					Non-ego depletion (<i>n</i> = 56)				
Godfrey et al. [54]	Adults, BMI >30 kg/m ² , ≥4 LOC/overeating episodes in 4 weeks, PSS ≥2	28				DSM-5: EDE-Q (LOC and overeating episodes, past 4 weeks), DEBQ and BES for self-reported ED symptoms	Emotional regulation, stress response, and BE severity	Post-stress: RMSSD ↓ trend; SDNN stable; LFn/LF-HF ↑ during stress; HFn ↓ (math vs. rest); no link between HRV and overeating/LOC	Moderate
			78.6%	41.1 ± 16.5	BED (<i>n</i> = 15)				
					37.5 ± 5.1				
Het et al. [55]	AN/BN female patients (pre-/post-treatment)	50				DSM-5: EDE-Q	Emotional regulation and stress response	ED > HC in negative affect (pre/post); post-TSST, HF blunted in ED	Not reported
			ED (<i>n</i> = 13)						
			100%	21 ± 1.3	BMI pre-treatment				
Jenkins et al. [28]	Female participants with AN and AN-WR	37	HC (<i>n</i> = 22)			DSM-5: Current AN diagnosis; AN-WR = past AN diagnosis, BMI >18.5 for ≥12 months	Weight regain	AN: ↓ HR and ↓ HRV (SDNN, SD rate); LF/HF ↔ across groups; no HRV differences in AN-WR vs. HC	Moderate to large
			100%	23 ± 1.1	21.9 ± 0.60				
			AN (<i>n</i> = 10)						
Juarascio et al. [56]	Participants with clinically significant emotional eating (≥4 episodes/28 days)	21	100%	31.64 ± 11.25	15.57 ± 1.69	Unspecified: EOQ	Emotional eating	↓ SDANN/RMSSD and ↑ SDNN linked to ↑ emotional eating; HF/LF showed greater fluctuation pre-episode vs. control	Moderate to large
			AN-WR (<i>n</i> = 17)						
			100%	25.07 ± 4.87	21.64 ± 2.14				
Juarascio et al. [56]	Participants with clinically significant emotional eating (≥4 episodes/28 days)	21	HC (<i>n</i> = 10)			Unspecified: EOQ	Emotional eating	↓ SDANN/RMSSD and ↑ SDNN linked to ↑ emotional eating; HF/LF showed greater fluctuation pre-episode vs. control	Moderate to large
			100%	27.44 ± 6.07	21.40 ± 1.84				
			Initial BMI: 27.79 ± 6.92						

Table 1. *Cont.*

Study	Sample Characterization					Diagnosis Criteria	ED Symptoms	Main HRV Results	Effect Size
	Type	N	% Female	Age (M ± SD)	BMI (M kg/m ² ± SD)				
Mehak et al. [57]	Women with ≥1 OBE in past 3 months	82	BE (n = 41) 100%	24.73 ± 6.95	26.24 ± 6.73	Unspecified: Adapted SCID-5-RV and ED examination; ≥1 OBE in past 3 months	Emotional regulation and somatic experience of being overweight	No effects on RMSSD, RSA, or HF	Not reported
			HC (n = 41) 100%	24.83 ± 7.65	23.35 ± 3.95				
Ortmann et al. [58]	Women with recurrent BE vs. without BE or ED history	56	BE (n = 28) 100%	35.2 ± 16.5	29.2 ± 8.0	DSM-5: EDI-2, EDIP-Q, and DEBQ	Emotional regulation in BE	BE: ↓ HR, ↑ HFn; ↑ negative affect vs. HC	Moderate to large
			HC (n = 28) 100%	32.9 ± 14.1	24.2 ± 4.2				
Parker et al. [62]	Non-clinical youth, BMI >5th percentile, good health	209	LOC-E (n = 19) 73.7%	13.7 ± 2.7	0.88 ± 1.02 (z-score)	Unspecified: EDE interview (child version for <13 y)	LOC	RMSSD/pNN50/HF ↔ LOC severity ↑ HRV → ↑ perceived LOC (recent group only)	Not reported
			No LOC-E (n = 19) 52.6%	12.5 ± 2.7	0.49 ± 1.02 (z-score)				
Paysal et al. [63]	AN patients without and with WR	46	AN without WR (n = 26) 100%	13.9 ± 1.6	13.9 ± 1.6	DSM-5: AN	Weight regain	AN without WR: ↑ parasympathetic activity (↑ RMSSD, pNN50, HF), stable LF; ↑ SDNN vs. AN-WR and HC	Moderate to large
			AN-WR (n = 10) 100%	15.7 ± 1.9	16.9 ± 2.2				
			HC (n = 33): 100%	14.1 ± 2.0	19.2 ± 2.3				
Ranzenhofer et al. [59]	Females, BMI ≥85th percentile, ≥2 LOC episodes/month	17	100%	14.77 ± 1.55	2.17 ± 0.48 (z-score)	Unspecified: EDE	Emotional regulation and LOC	↑ HR and ↓ RMSSD predicted ↑ LOC; HRV lower before high- vs. low-LOC episodes	Small to moderate
Ranzenhofer et al. [60]	Adolescents, BMI >70th percentile, ≥2 LOC episodes/month	24	66.7%	15.6 ± 1.7	30.4 ± 5.6	Unspecified: EDE	Emotional regulation and LOC	↑ HR (30 min pre-eating) → ↑ LOC ratings; RMSSD/pNN50 ↓ with ↑ LOC	Small
Rommel et al. [61]	AN-RT patients (BMI ≈15)	40	AN-RT (n = 16): 100%	19.5	15.2	DSM-IV: AN-RT	Emotional regulation	AN-RT: ↓ HF, ↑ WT-HRV during emotional induction	Large
			HC (n = 24) 100%	19	21				

Table 1. Cont.

Study	Sample Characterization					Diagnosis Criteria	ED Symptoms	Main HRV Results	Effect Size
	Type	N	% Female	Age (M ± SD)	BMI (M kg/m ² ± SD)				
Schmalbach et al. [64]	Patients with AN	38	AN (n = 19) 89.5%	26.05 ± 5.49	18.70 ± 3.30	DSM-IV: AN	Weight regain and stress response	AN: ↑ stress appraisal; ↓ overall HRV reactivity (↓ SDNN, LF; ↑ HF); SDNN ↑ at rest (low-stress subgroup); blunted HR/LF response	Moderate to large
			HC (n = 19) 89.5%	24.21 ± 5.54	24.23 ± 3.04				

Note: Symbols are interpreted as follows: ↑ indicates a significant increase, ↔ indicates stability (no significant change), and ↓ indicates a significant decrease. AN = Anorexia Nervosa; AN-RT = Anorexia Nervosa Restrictive Type; AN without WR = Anorexia Nervosa without Weight Recovery; AN-WR = Anorexia Nervosa with Weight Recovery; BE = Binge Eating; BES = Binge Eating Scale; BITE = Bulimic Investigatory Test, Edinburgh; BMI = Body Mass Index; DEBQ = Dutch Eating Behavior Questionnaire; DSM- IV = *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition; DSM-5 = *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition; ED = Eating Disorder; EDDS = Eating Disorder Diagnostic Scale; EDE = Eating Disorder Examination Interview; EDE-Q = Eating Disorder Examination Questionnaire; EDI-2 = Eating Disorder Inventory-2; HC = Healthy Control; HF = High Frequency; HFn = normalized High Frequency; HRV = Heart Rate Variability; LF/HF = Ratio of Low Frequency to High Frequency; LFn = normalized Low Frequency; LOC-E = Loss-of-Control Eating; No LOC-E = No Loss-of-Control Eating; OBE- = Without Objective Binge Eating Episodes; OBE+ = With Objective Binge Eating Episodes, pNN50 = Percentage of consecutive NN intervals that differ by more than 50 ms with respect to the total number of NN intervals; RMSSD = Root Mean Square of Successive Differences; RSA = Respiratory Sinus Arrhythmia; SCID-5-RV = Structured Clinical Interview for DSM-5, Research Version; SD = Standard Deviation, SDNN = Standard Deviation of NN intervals.

3.2. HRV Measurement

Across the included studies, HRV was assessed using a combination of time-domain, frequency-domain, and, in fewer cases, nonlinear metrics. Commonly reported time-domain measures included RMSSD and SDNN, pNN50, SDANN, and SD rate, which capture short-term and overall variability in heart rate intervals. Frequency-domain parameters such as HF, LF, LF/HF ratio, LFn, and HFn were also frequently used to differentiate between parasympathetic and sympathetic influences on cardiac activity. Some studies, such as those by Rommel et al. [61] and Paysal et al. [63], additionally employed more advanced techniques like wavelet transform HRV (WT-HRV) or nonlinear indices such as SD1 and SD2. This variety in HRV metrics reflects the diverse methodological approaches to study this topic.

3.3. Risk-of-Bias Analysis Results

Risk-of-bias assessment was conducted for the included studies using the JBI revised critical appraisal tools (Supplementary Materials). Different JBI checklists were applied depending on the study design. Cross-sectional comparative studies [28,58,63,65] were evaluated using the Checklist for Analytical Cross-Sectional Studies (Supplementary Materials, Table S1). Quasi-experimental studies [53,55,57,61,64], including those with pre-existing groups and embedded experimental manipulations, were assessed using the Checklist for Quasi-Experimental Studies (Supplementary Materials, Table S2). Experimental studies with within-subject designs and no control group [54,66] were also evaluated using the quasi-experimental checklist, given the lack of a more specific tool for this design. The only randomized controlled trial [67] was appraised using the Checklist for Randomized Controlled Trials (Supplementary Materials, Table S3). Finally, studies with longitudinal observational or ecological momentary assessment designs [56,59,60,62] were assessed using the Checklist for Cohort Studies (Supplementary Materials, Table S4).

Overall, the risk of bias across studies was judged to be low to moderate. Analytical cross-sectional studies generally showed high methodological quality, with all studies clearly identifying confounding factors and applying appropriate outcome measures. However, one study [65] had an unclear response for one domain related to confounding control.

Among the quasi-experimental studies, most showed adequate internal validity, particularly in the administration of interventions and outcome measurement. Nevertheless, consistent weaknesses were observed regarding participant retention, with several studies failing to report strategies for handling dropouts [53,57,61]. Two studies [54,66] also lacked clarity regarding temporal precedence and participant selection, increasing the potential for bias.

The only randomized controlled trial [67] exhibited limitations in allocation concealment, blinding, and participant retention, suggesting a moderate risk of bias despite otherwise appropriate procedures.

Finally, cohort studies varied in quality. While some demonstrated robust methodologies [56,59], others [60,62] showed moderate to high risk of bias due to unclear temporal relationships and insufficient strategies to address confounding and missing data.

3.4. Overall Findings

To present the information on how HRV relates to different eating disorder-related phenomena, we organized the findings into five interrelated thematic areas: AN, BE behavior, dietary restraint, weight status, and emotional eating/LOC. While each theme is presented separately to highlight distinct patterns in the literature, we acknowledge that these domains are not independent. This thematic structure allows a focused analysis and a

broader understanding of how HRV may serve as a transdiagnostic marker across multiple, interacting features of eating pathology.

3.4.1. Anorexia Nervosa and Autonomic Regulation

First, relating to BMI and physical exercise, Billeci et al. [65] examined the effect of physical exercise on HRV in adolescents with AN, finding a slight increase in HRV during the task, followed by a return to low levels during recovery in the AN group. Importantly, differences emerged based on BMI: among individuals with a BMI below 17.5, interbeat interval (IBI) was positively correlated with BMI, whereas a negative correlation was observed in those with a BMI above 17.5. These associations reflected moderate to strong effect sizes, with a negative (though nonsignificant) correlation between BMI and heart rate in the more underweight subgroup ($r = -0.44$) and a strong positive correlation in the less underweight subgroup ($r = 0.66$), suggesting distinct patterns of cardiovascular-autonomic coupling depending on illness severity. In terms of time-domain measures, RMSSD and SDNN remained stable in the AN group throughout exercise, whereas in healthy controls both indices increased during the task and decreased during recovery. Together, these results suggest altered autonomic reactivity and recovery dynamics in AN patients compared to healthy peers.

Along the same lines, a study by Rommel et al. [61] examined the relationship between emotional induction and HRV, investigating whether individuals diagnosed with restrictive anorexia would have greater difficulties in emotional regulation, evaluating HRV after an emotional induction task. The results showed that AN patients experienced a decrease in HRV after emotional induction, with recovery being slower than in the control group, with a large effect size ($R^2 = 0.658$), which indicates a greater difficulty in emotional regulation.

In relation to stress reactivity, Schmalbach et al. [64] measured HRV during the TSST in AN patients. The findings indicated a reduction in sympathetic activity in the AN group, as reflected by lower LF HRV, without a corresponding compensatory increase in parasympathetic indices. Specifically, HF, LF/HF ratio, RMSSD, and SDNN remained comparable to those of the control group. Moreover, overall HRV reactivity was attenuated in AN patients, indicating blunted sympathetic responsiveness in the absence of parasympathetic dominance. These alterations were supported by moderate to large effect sizes, with significant group-by-time interactions for HR ($\eta p^2 = 0.206$), HF ($\eta p^2 = 0.140$), LF ($\eta p^2 = 0.179$), and LF/HF ratio ($\eta p^2 = 0.166$), and a particularly large effect for perceived stress reactivity ($\eta p^2 = 0.72$).

Finally, with regard to weight restoration, Jenkins et al. [28] expanded upon previous findings by comparing individuals suffering from AN with weight-restored AN patients and healthy controls. The results showed that individuals with AN exhibited significantly lower HR and reduced time-domain HRV (SDNN and SD rate), whereas frequency-domain measures did not differ across groups. Importantly, HRV measures in AN-WR participants were comparable to those of healthy controls, suggesting that autonomic function may normalize following sustained weight restoration. Additionally, the study reported moderate associations between autonomic markers and physiological indices across groups, with muscle sympathetic nerve activity positively correlated with BMI ($r = 0.485$) and diastolic blood pressure ($r = 0.361$), reflecting moderate to large effect sizes.

3.4.2. Binge Eating and Emotional Dysregulation

Five studies investigated BE and emotional dysregulation, focusing on guilt and LOC. Bottera et al. [53] explored HRV in relation to emotional responses after OBE. The authors found an inverse relationship between HRV and guilt—individuals reporting greater post-binge guilt exhibited lower HRV. Notably, during the binge episode itself,

individuals with high guilt levels showed a paradoxical increase in HRV ($\eta^2 = 0.139$), alongside accelerated eating rates, highlighting complex and potentially dysregulated autonomic–affective interactions during binge episodes.

Similarly, Godfrey et al. [54] examined HRV at rest and during a cognitive stressor in individuals with obesity and LOC eating. At baseline, lower SDNN correlated with greater LOC severity, while autonomic imbalance—reflected in higher LF and lower HF components—was linked to more severe overeating. During the stress task, HRV reactivity patterns emerged (increased LF and LF/HF ratio and decreased HF), but these were not associated with LOC or overeating severity. The authors concluded that resting HRV reflects traits like autonomic flexibility related to disinhibited eating, whereas HRV reactivity to acute stress may be more indicative of transient emotional regulation capacity.

Additionally, Mehak et al. [57] investigated physiological and subjective responses to experimental inductions in women with and without a recent history of BE. When participants imagined consumption of high-calorie foods, a significant increase in self-reported somatic feelings of being overweight was exclusively identified in the binge eating group. In contrast, negative affect induction did not elicit changes in this experience in either group, challenging the traditional displacement hypothesis. HRV, measured via RMSSD, remained stable across conditions and groups. These findings suggest that the somatic experience of feeling overweight may be specifically linked to cognitive–affective processes related to eating, rather than to general emotional distress.

Ortmann et al. [58] further explored emotional experience in women with and without BE, focusing on ANS activity. Compared to healthy controls, individuals with BE exhibited reduced HR alongside elevated HF-HRV ($d = 1.13$), a marker of parasympathetic activity. These findings suggest a shift toward parasympathetic dominance and reduced sympathetic arousal in the BE group.

Finally, Ranzenhofer et al. [59,60] examined whether autonomic indices could prospectively predict LOC eating in adolescents with a history of LOC and elevated BMI. A higher pre-meal heart rate was associated with greater post-meal LOC ratings at the intra-individual level, suggesting that acute elevations in physiological arousal may precede episodes of disinhibited eating. In addition, HRV—measured via RMSSD and pNN50—was inversely associated with LOC severity in the 30 min preceding food intake, indicating that reduced parasympathetic activity may contribute to heightened vulnerability for LOC episodes. Together, these findings underscore the relevance of momentary autonomic markers in capturing short-term fluctuations in emotional and physiological regulation in individuals with BE.

Taken together, these results point to altered autonomic profiles in individuals with binge eating, potentially reflecting distinctive patterns of interoceptive processing and emotional regulation in this population.

3.4.3. Dietary Restraint and Self Control

Geisler et al. [67] assessed how dietary restraint—defined as the adherence to self-imposed dieting rules—interacts with HRV during food exposure. Although HRV rose during exposure for all participants, restrained eaters only showed higher HRV (adjusted $R^2 = 0.34$) when they had experienced prior ego depletion, suggesting that the capacity for autonomic self-regulation in response to food cues is contingent on both trait restraint and available cognitive resources.

3.4.4. Weight Status and Food Cues

In relation to weight status, Chang et al. [66] investigated HRV responses to high-calorie food images across individuals varying in BMI. Overall, exposure to food cues

increased the LF/HF ratio, indicating heightened sympathetic activation. However, BMI was negatively correlated with this ratio, suggesting that individuals with higher BMI may exhibit impaired sympathovagal balance and reduced cognitive control in response to appetitive stimuli. These findings point to altered autonomic reactivity as a potential mechanism contributing to dysregulated eating behavior in individuals with elevated weight status.

3.4.5. Emotional Eating and Locus of Control

Finally, in relation to emotional eating and HRV, Juarascio et al. [56] investigated whether emotional eating risk could be predicted by five-minute HRV intervals. The authors found that decreases in high-frequency HRV preceded episodes of emotional eating, confirming HF HRV as a marker of poor emotion regulation. Unexpectedly, higher LF also predicted greater emotional eating risk, suggesting that elevated sympathetic activation may index dysregulated eating responses. The study employed machine learning models to classify emotional eating episodes based on HRV features, achieving high accuracy (77.99%), sensitivity (78.75%), and specificity (75.00%), indicating strong predictive power and practical relevance for detecting real-time autonomic patterns linked to dysregulated eating. Consistent with other work, lower SDANN and RMSSD increased emotional eating risk, whereas higher SDNN was associated with a greater likelihood of emotional eating.

Finally, Parker et al. [62] demonstrated a positive correlation between pre-meal HRV and internal locus of control, but only among participants with recent LOC episodes, suggesting that autonomic flexibility before eating may bolster perceived control in vulnerable individuals.

Collectively, these results underscore that HRV indices—both in time and frequency domains—are differentially associated with restrictive and binge-type behaviors, stress reactivity, self-control, BMI, and perceived control overeating.

4. Discussion

This review aimed to explore the relationship between HRV and the core symptoms of EDs, particularly focusing on AN, BN, BED, and related disordered eating behaviors. Across the literature, findings have painted a complex picture. Some studies point to higher HRV levels, which indicate greater parasympathetic activity [42,55,68,69], while others reveal more ambiguous outcomes, showing no clear alterations in ANS activity. While this review primarily addresses the impact of HRV on the nuclear symptoms of EDs—such as emotional dysregulation, autonomic reactivity to stress, and disordered eating behaviors—these findings have significant implications for understanding the role of autonomic function in EDs more broadly. The inconsistencies across studies do not necessarily undermine the relevance of HRV; rather, they suggest that autonomic dysregulation in EDs may manifest differently depending on clinical subtype, illness stage, or contextual factors. Paradoxically, this very heterogeneity points to the potential of HRV as a dynamic and sensitive biomarker—capable of capturing subtle physiological shifts over the course of the disorder. Analyzing the results, one recurrent theme is the disruption of autonomic regulation, with HRV frequently used as a physiological index of this imbalance. Billeci et al. [65] observed that AN patients displayed less adaptive HRV responses during physical activity compared to healthy controls. Beyond showing diminished recovery, their results also pointed to an intriguing link between body mass index (BMI) and interbeat interval (IBI): while individuals with higher BMI showed a positive correlation, those with lower BMI showed the reverse. This pattern suggests that extreme underweight status may amplify autonomic dysfunction, while partial weight restoration might contribute to modest improvements in HRV. Supporting this, Jenkins et al. [28] found that individuals

with AN who had achieved weight restoration exhibited HRV levels closer to those of healthy peers.

These findings imply that at least some of the physiological disruptions observed in AN, particularly those linked to autonomic regulation, may not be irreversible. Weight restoration, in this sense, becomes a turning point in the recovery process. However, the HRV patterns observed during emotional induction and stress tasks point to persistent vulnerabilities in autonomic function. Rommel et al. [61] found that AN patients exhibited slower HRV recovery following emotional induction, indicating a diminished ability to regulate emotional responses. This is consistent with the findings of Schmalbach et al. [64], who documented blunted sympathetic reactivity to stress in AN participants, without the expected parasympathetic compensation. These findings integrate well, suggesting that the AN phenotype is marked by a combination of attenuated sympathetic responsiveness and insufficient parasympathetic flexibility, further compromising the emotional regulation capacity that is central to the disorder.

In the context of BED, HRV is similarly linked to emotional regulation difficulties. Bottera et al. [53] identified a complex context-dependent pattern: after binge episodes, HRV decreased in association with heightened guilt, a finding that reflects poor post-binge regulation. Paradoxically, during the binge episodes themselves, individuals with high levels of guilt showed increased HRV, which may point to an autonomic compensatory mechanism during high-arousal states, only to be overwhelmed by subsequent emotional dysregulation. This underscores the idea that HRV changes may not only reflect a stable trait of autonomic dysfunction but also an adaptive or maladaptive response to specific emotional or behavioral triggers.

Other studies have further explored the relationship between HRV and behaviors like LOC eating. Godfrey et al. [54] examined LOC eating behaviors and found that lower resting HRV was associated with greater LOC severity, suggesting that diminished baseline autonomic flexibility may reflect chronic difficulties in self-regulation. In line with this, Ortmann et al. [58] reported that individuals with BE behaviors exhibited reduced HR alongside elevated high-frequency HRV, pointing to atypical parasympathetic dominance. Complementing these findings, Ranzenhofer et al. [59,60] demonstrated that momentary autonomic indices—specifically elevated pre-meal HR and reduced HRV—prospectively predicted LOC eating episodes in adolescents with elevated BMI, highlighting the relevance of short-term physiological arousal in the onset of disinhibited eating. This relationship reflects how chronic low HRV might serve as a trait marker for the difficulty in regulating eating behaviors, aligning with prior evidence [38,39], while HRV reactivity to stress was more indicative of momentary emotional regulation capacity rather than the severity of disordered eating. Together, these findings suggest that reduced HRV may contribute to vulnerability to both emotional dysregulation and compulsive eating behaviors, with an important distinction between baseline HRV as a trait indicator and reactive HRV as a state marker during emotional or stressor challenges.

This dynamic interplay between trait and state HRV becomes even more apparent when considering cognitive control. Geisler et al. [67] examined how dietary restraint interacts with HRV during food exposure, finding that HRV was higher among restrained eaters only after they experienced prior ego depletion. This interaction suggests that self-regulation in response to food cues is not solely a function of an individual's baseline autonomic flexibility but is contingent on cognitive resources that facilitate restraint. This interplay between autonomic flexibility and cognitive self-control aligns with the findings of Chang et al. [66], who reported that individuals with higher BMI exhibited impaired sympathovagal balance (as indicated by lower HRV), suggesting that autonomic flexibility may be compromised in individuals with greater weight and poorer dietary control. Taken

together, these studies emphasize the importance of considering both trait and state factors in understanding the autonomic regulation of eating behaviors.

A key theme emerging across several studies is the connection between emotional eating and LOC. Juarascio et al. [56] found that decreased HF-HRV predicted emotional eating episodes, a finding that suggests that poor emotional regulation, as reflected in HRV, is a precursor to dysregulated eating. This finding was complemented by Parker et al. [62], who observed a positive correlation between pre-meal HRV and internal locus of control in individuals with recent LOC episodes. This suggests that higher HRV before eating may help bolster a sense of control, thereby reducing vulnerability to emotional eating. Together, these studies illustrate that autonomic flexibility—evidenced by higher HRV—may serve as a protective factor against disordered eating, particularly in contexts where emotional regulation is key.

Therefore, the collective evidence from these studies highlights a crucial link between HRV and the core symptoms of EDs, particularly emotional dysregulation, stress reactivity, and disordered eating behaviors. In both AN and BED, altered autonomic function appears to underline difficulties in regulating emotional responses and food-related behaviors. While the blunted autonomic reactivity observed in AN patients [33,55], especially in response to stress and emotional stimuli, suggests a chronic dysregulation that may persist despite weight restoration [44], the studies on BED point to a more dynamic, context-dependent relationship between HRV and emotional eating. For example, the paradoxical HRV increase during binge episodes followed by a sharp decrease in recovery may indicate a cyclical pattern of autonomic dysfunction tied to emotional and behavioral responses.

Altogether, these findings suggest that HRV not only reflects important physiological differences across eating disorder profiles but may also offer a promising entry point for therapeutic innovation. One such avenue is HRV biofeedback as a method to be taken into account in the treatment of EDs, for example, using biofeedback intervention in HRV to improve emotional regulation by decreasing HRV [70]. Supporting this, Meule et al. [71] showed that HRV biofeedback reduced food-related cognitions and cravings, highlighting the need to combine autonomic interventions with cognitive strategies to enhance clinical outcomes. This type of intervention has been tested and has proven to be useful in the population with AN, so it would be interesting to incorporate it into current intervention procedures [72]. Beyond its therapeutic role, HRV may also serve a broader function as a clinical marker. Given the known cardiovascular risks associated with eating disorders, including bradycardia and autonomic instability [73,74], monitoring HRV could provide valuable information about both psychological functioning and physical health. As such, HRV occupies a unique position at the intersection of emotion, behavior, and physiology—one that merits deeper exploration in future research and clinical practice.

4.1. Practical Implications

These findings emphasize the need for interventions that not only target the symptoms of EDs but also focus on enhancing autonomic regulation. Strategies aimed at improving HRV—such as HRV biofeedback, mindfulness-based interventions, yoga, slow-paced breathing, and vagal nerve stimulation techniques—could complement traditional therapeutic approaches by fostering better emotional regulation and reducing vulnerability to dysregulated eating behaviors. These methods can be integrated alongside cognitive-behavioral therapy or emotion-focused therapy, enhancing their impact by addressing physiological self-regulation. Moreover, understanding the complex relationship between HRV, emotional regulation, and eating behaviors could help inform personalized treatment plans, particularly for individuals with BED or those with chronic AN who may benefit from targeted interventions aimed at improving autonomic flexibility.

An example of a protocol combining RV biofeedback with non-invasive vagus nerve stimulation to support emotional and autonomic regulation in individuals with anorexia nervosa could be developed as follows. Participants take part in the intervention over the course of 8 weeks, attending two sessions per week, each lasting about 45 to 60 min. During each session, they begin with HRV biofeedback. First, a 5 min baseline is recorded. Then, participants are guided to breathe slowly and evenly, usually around 6 breaths per minute, while watching visual feedback that shows how well their heart rhythms are becoming more stable and “coherent.” This part lasts about 20 min. The goal is to train the body to improve parasympathetic activity and manage emotional states more effectively. In addition to biofeedback, participants receive transcutaneous auricular vagus nerve stimulation. The stimulation typically lasts 15–20 min, at a frequency of around 25 Hz, and is kept at a comfortable level just below the threshold of discomfort. It can be applied either before or during the biofeedback session. Throughout the program, measures such as HRV readings, eating disorder symptoms, anxiety levels, and emotion regulation capacity are tracked to monitor progress.

4.2. Limitations

Limitations of this review include the heterogeneity of HRV measurement protocols, variability in study designs, and the lack of standardized quality assessments across the included studies. Additionally, the narrative synthesis approach, while appropriate given the diversity of methods, may introduce interpretive bias. Another limitation is the restriction to studies published in English or Spanish, which may have excluded relevant findings reported in other languages. These limitations highlight the need for more rigorous, standardized, and internationally inclusive methodologies in future research examining HRV in eating disorders.

5. Conclusions

In summary, while this review focused on the core symptoms of EDs in relation to HRV, it is evident that HRV plays a central role in both the physiological and emotional aspects of these disorders. However, some limitations of this review include variability in HRV measurement protocols and a lack of standardized quality assessments across the included studies. Future research should continue to investigate how autonomic regulation interacts with the full spectrum of ED symptoms and how HRV can be leveraged as both a diagnostic tool and a target for therapeutic intervention.

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Abbreviations

AN	Anorexia Nervosa
AN-RT	Anorexia Nervosa Restrictive Type
ANS	Autonomic Nervous System
BE	Binge Eating
BED	Binge Eating Disorder
BL	Baseline
BMI	Body Mass Index
BN	Bulimia Nervosa
DSM-5	<i>Diagnostic and Statistical Manual of Mental Disorders</i> , 5th Edition
EDs	Eating Disorders
HC	Healthy Control
HF	High Frequency
HF _n	normalized High Frequency
HPA	Hypothalamic–pituitary–adrenal
HR	Heart Rate
HRV	Heart Rate Variability
IBI	Interbeat interval
LF	Low Frequency
LF _n	normalized Low Frequency
LF/HF	Ratio of Low Frequency to High Frequency
LOC	Loss of Control
OBE	Objective Binge Episode
pNN50	Percentage of consecutive NN intervals that differ by more than 50 ms with respect to the total number of NN intervals
RMSSD	Root Mean Square of Successive Differences
RS	Restrained Scale
RSA	Respiratory Sinus Arrhythmia
SDANN	Standard Deviation of the Average NN intervals
SDNN	Standard Deviation of NN intervals
SD rate	Standard Deviation of Heart Rate
TSST	Trier Social Stress Test
WR	Weight Restriction

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