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Uncovering the association between broad emotional dysregulation and emotional eating: A meta-analysis

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ABSTRACT

Emotional eating refers to the consumption of food in response to pleasant or unpleasant emotions. This behavior has frequently been linked to emotional dysregulation; however, the results obtained are inconsistent. Therefore, the aim of this meta-analytic study was to conduct a meta-analysis to synthesize the available evidence on the relationship between broad emotional dysregulation and emotional eating, as well as to identify possible moderators of this association. A systematic search was conducted in March 2025 in the following databases: Scopus, Web of Science, APA PsycInfo, and APA PsycArticles. Inclusion criteria comprised quantitative studies published in English or Spanish, analyzing the broad emotional dysregulation-emotional eating relationship, and reporting sufficient data to extract effect sizes. This systematic search yielded 40 studies and 42 samples (N = 14,481; M_{age} = 26.8; SD_{age} = 11.6; 67.8% females). The results revealed a positive, moderate association between broad emotional dysregulation and emotional eating (r_{pooled} = .337 [.279, .396]). The valence of emotional eating and sample type emerged as relevant moderators. The association was stronger when focusing on studies that exclusively analyzed negative emotional eating (r_{pooled} = .286 [.226, .345]) and became non-significant for studies addressing positive emotional eating (r_{pooled} = .055 [-.113, .220]). Moreover, the links were stronger for non-clinical samples as compared to clinical ones. This review helps to better understand the association between broad emotional dysregulation and emotional eating and will contribute to preventing and intervening in this problem by developing new and more effective preventive and therapeutic strategies.

Emotional eating (EE) refers to eating in response to emotions rather than physiological hunger (Arnow et al., 1995; van Strien, 2018). This behavior is not only observed in clinical populations (e.g., Braden et al., 2016), but also relatively common in community samples (e.g., Sultson & Akkermann, 2019). Approximately 20% to 45% of adults (Gibson, 2012; Scarmozzino & Visioli, 2020) and 30% of adolescents (Kidwell et al., 2024) in the general population engage in EE, with rates rising to nearly 60% among those with overweight or obesity (Péneau et al., 2013; Wong et al., 2020). EE is a significant predictor of difficulties in losing weight (Risica et al., 2021), unhealthy eating patterns (e.g., Barnhart, Braden, & Dial, 2021), and mental health issues such as eating disorders (Reichenberger et al., 2020) or depression and anxiety (Marchena-Giráldez et al., 2024). Therefore, it is crucial to deepen our understanding of EE, particularly by exploring the underlying mechanisms, to establish a foundation for preventive and therapeutic interventions.

The idea that eating functions as a regulatory mechanism for emotions is central to contemporary theories of EE and forms the basis for several psychotherapeutic approaches to eating disorders (e.g., Macht, 2008; Safer et al., 2009). According to affect regulation models, individuals experiencing negative emotional states may turn to food consumption as a coping strategy (Burton et al., 2007), as eating can provide a temporary sense of comfort. However, the tendency to eat in response to emotions varies among individuals (e.g., Reichenberger

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et al., 2021). Current trends in research focus on identifying psychological variables indicating greater vulnerability to EE in certain individuals and on examining the antecedents and mechanisms that contribute to its development (e.g., Ouwens et al., 2009; Van Strien & Ouwens, 2007).

From a transdiagnostic perspective, which emphasizes the potential for broad psychological interventions (for a review, see Carlucci et al., 2021), broad emotional dysregulation (ED) is considered a factor associated with EE. Following Gratz and Roemer's (2004) conceptualization, ED is understood as the difficulties individuals face in: (a) emotional awareness and understanding; (b) emotional acceptance; (c) the ability to engage in goal-directed behavior and inhibit impulsive actions when experiencing negative emotions; and (d) access to effective emotion regulation strategies. Some empirical evidence supports this association (e.g., Braden et al., 2021; Zijlstra et al., 2012), suggesting the potential value of transdiagnostic interventions targeting ED. However, existing literature indicates that the strength of this relationship is not consistently clear. For instance, Braden et al. (2021) found a strong correlation (i.e., $r = .61$) between ED and EE among adults, while Zijlstra et al. (2012) reported a much weaker one (almost absent: $r = .07$) in a sample of adults, highlighting the need for more detailed examination, for example, by assessing different moderating factors. Understanding these factors is crucial because they may condition the relationship between ED and EE. When the association between ED and EE is stronger, transdiagnostic approaches addressing general ED (e.g., Cludius et al., 2020; Lattimore, 2020; Sloan et al., 2017) may be beneficial. Conversely, when the relationship is weaker or absent, more specific interventions that focus directly on the use of food for emotion regulation might be more appropriate, rather than broader transdiagnostic approaches.

To address this significant gap in the literature, the objective of this study is to conduct a meta-analysis of the association between ED and EE in adolescents and adults. This analysis will examine various factors that could serve as relevant moderators of this relationship. Potential moderating factors are outlined in the following subsection.

1. Possible moderators in the link between ED and EE

In this study, the potential moderating effects of the following variables were examined: type of EE (positive *vs*. negative emotions), sample type (non-clinical *vs*. clinical), age, Body Mass Index (BMI), type of emotion (boredom, depression, or anxiety/anger), and percentage of women. The inclusion of the type of EE as a potential moderator is grounded in recent evidence highlighting the distinct underlying psychological and behavioral mechanisms (Manchón et al., 2021). Positive EE involves eating in response to positive emotions, such as joy or excitement, and it has been associated with reward-driven eating patterns (Barnhart, Braden, & Price, 2021; Loxton & Tipman, 2017; van Bloemendaal et al., 2015; van Strien et al., 2016). Negative EE refers to the consumption of food in response to negative emotions such as sadness or anxiety, and it has been consistently linked to ED and maladaptive coping strategies (Braden et al., 2018; Deroost & Cserjési, 2018; Evers et al., 2018). The different effects of these two types of EE on ED suggest that the pathways connecting ED and EE may vary depending on whether eating is used to cope with negative emotions or to enhance positive experiences (Braden et al., 2018; van Strien, 2018). Since negative EE is more strongly linked to maladaptive coping strategies, higher stress levels, and emotional instability, we hypothesize that the association between ED and EE will be stronger for negative EE compared to positive EE.

Regarding the type of population (non-clinical *vs*. clinical), previous studies have found that populations with greater health problems, such as obesity (Klatzkin et al., 2018), eating disorders (Haedt-Matt & Keel, 2011; Svaldi et al., 2012), or other mental disorders (Aldao et al., 2010; Edmond et al., 2016) tend to experience greater emotional instability and difficulty managing negative emotions. These studies also highlight that difficulties in emotion regulation are clearly associated with the use of EE as a coping strategy in clinical groups. Furthermore, in relation to the aim of our meta-analytic review, it can be suggested that the strength of the association may intensify for clinical groups. For instance, Demirci (2018) observed that the association between ED and EE was notably higher ($r = .76$) in adolescents with post-traumatic stress disorder symptoms who had been victims of child sexual abuse as compared to adolescents without clinical problems ($r = .25$). Regarding eating-related problems, in a series of consecutive studies, Vandewalle et al. (2014, 2016) observed that the association between ED and EE was also stronger in a sample of adolescents in a treatment center for obesity ($r = .39$) than for a community sample of the same age ($r = .27$). Therefore, we hypothesize that clinical status will function as a moderator, with the association between ED and EE expected to be stronger in clinical than in general populations.

The relationship between ED and EE across ages has not been systematically examined. Emotion regulation strategies develop with age, and during adolescence individuals typically have a more limited repertoire compared to later stages (Zimmermann & Iwanski, 2014). With fewer options for managing emotions, adolescents may more readily turn to accessible behaviors like eating savory comfort food, potentially strengthening the ED-EE association at this stage. In contrast, older individuals are often more aware of the long-term health risks associated with unhealthy eating (Lockenhöff & Carstensen, 2004). This awareness may reduce their reliance on EE when ED occurs.

Although BMI, overweight, and obesity have been assessed in studies on ED and EE, their potential moderating roles are rarely addressed. Most studies treat BMI as a covariate (e.g., Barnhart, Braden, & Dial, 2021; Echeverri-Alvarado et al., 2020; Ferrell et al., 2020), assuming an association with either ED or EE. While some evidence supports links between BMI and ED or EE (e.g., Czepczor-Bernat and Brytek-Matera, 2021; Guerrini-Usubini et al., 2023; Spinner et al., 2024), other studies report no such associations (e.g., Guerrini-Usubini et al., 2021; Mohorić et al., 2023; Yang et al., 2023), suggesting that more complex interactions—such as moderation—may be involved. To the best of our knowledge, only one study has directly tested this association: Shriver et al. (2021) found that the link between ED and EE was significant among adolescents with normal weight, but not among those with overweight or obesity. Zhou et al. (2025), in a recent meta-analysis, found no moderating effect of BMI on the link between ED and disordered behaviors, but noted that the limited representation of individuals with obesity precluded firm conclusions. They emphasized the need for studies including adolescents and obese samples. Given the current gaps and limited evidence, we do not propose a formal hypothesis but instead explore BMI.

Regarding the type of emotion experienced prior to EE, it is worth noting that not all negative emotions are associated with EE to the same extent. In this sense, the association between high-activation emotions, such as anxiety/anger ($r = .62$; Barnhart, Braden, & Dial, 2021) and ED, was stronger than for low-activation emotions, such as boredom ($r = .12$; Braden et al., 2023) or depression ($r = .14$; Ferrell et al., 2020). Therefore, we hypothesize that the relationship between ED and negative EE will be stronger for high arousal emotions (anxiety/anger) than for low arousal emotions (boredom and depression).

Regarding gender, a few studies support the possibility of being a moderator. For example, Zhang et al. (2021) observed differential associations between ED and EE in their study with university students. Stronger links were observed for both the total scores of ED and all its components (i.e., non-acceptance of emotions, limited emotion regulation strategies, and impulsiveness) in female students than in male students. In other types of samples, such gender-differential associations have also been noted. This is the case of Saccaro et al.'s (2023) study; in a sample of bariatric surgery candidates, they did not find gender differences for ED, but women showed more EE than men. Consequently, we hypothesize that the relationship between ED and EE will be stronger in women than in men.

2. Methods

2.1. Sources and literature search strategy

The present meta-analysis was reported considering the guidelines established by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement (Page et al., 2021). The systematic search was conducted in March 2025 in the following databases: Scopus, Web of Science, APA PsycInfo, and APA PsycArticles. We used a search strategy combining the terms "emotional eating" AND "emotion* regulation" OR "affect regulation" OR "mood regulation".

To ensure a more comprehensive search that included grey literature and reduced possible publication bias, two specific measures were taken. First, the peer-review filter was not selected in the searched databases, which yielded one PhD thesis. Second, e-mail requests were sent through group lists; however, no additional records were obtained through this route.

2.2. Eligibility criteria

The following inclusion criteria were established: 1) quantitative studies that included analysis linking ED and EE, or that could be provided by authors upon request; 2) published in English or Spanish; and 3) reported sufficient information to allow extraction of effect sizes. We excluded papers with the following characteristics: 1) theoretical articles or secondary data (i.e., systematic reviews or meta-analyses); 2) studies that did not provide a total ED score; 3) studies applying or evaluating a prevention program or intervention; 4) single-case studies; 5) inaccessible papers.

2.3. Study selection

The initial search yielded 638 documents that were reduced to 311 after eliminating those that were duplicated. Next, after reading the titles and abstracts, 220 documents were excluded because they did not meet the previously indicated inclusion criteria. Three authors reviewed all the remaining records ($k = 91$). Six documents were excluded as they could not be located. Subsequently, a fourth author conducted a thorough review of the documents flagged for discrepancies in the previous step, resulting in elimination of 50 documents. Finally, a fifth author, outside the entire selection process, thoroughly reviewed the studies initially excluded ($k = 220$), ultimately including a total of 40 documents comprising 42 samples (see Fig. 1). The description of the included studies can be found in Table 1.

2.4. Study codification

According to Lipsey and Wilson (2001), a coding scheme was developed in which one author recorded all the relevant information for each of the studies included: authors, year of publication, objective of the study, sample size, information related to the age of the participants (mean, standard deviation, and range), percentage of women, origin of the sample, measurement instruments, reliability indices, type of design, type of EE, mean Body Mass Index (BMI), and effect sizes. Another author independently coded 30% of the data. The agreement percentage was 90%. An additional author not involved in the previous phases resolved discrepancies among the coders until consensus was reached.

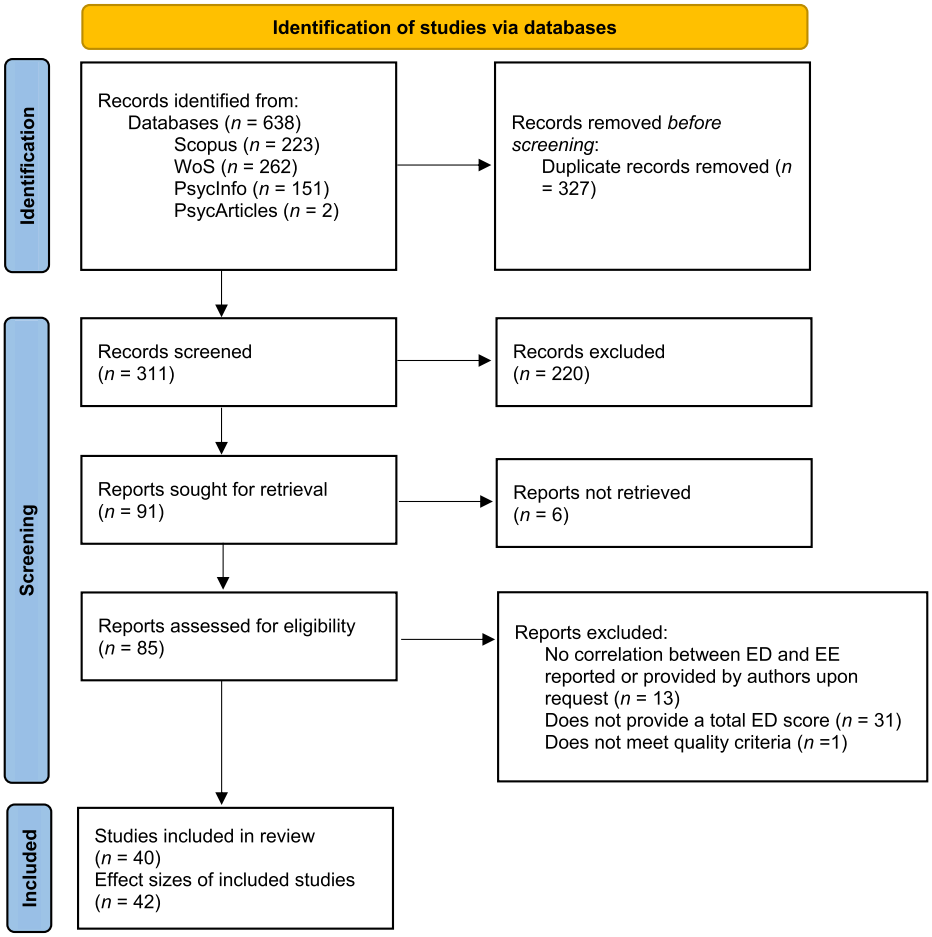

Fig. 1. The flowchart only includes database searches (PRISMA 2020).

Table 1
Study description.

Author (Year)	Country	*N*	% Female	Age: *M (SD)*	Sample type	ED measure	EE measure
Ansari et al. (2018)	Iran	700	51.7	29.81 (9.43)	Non-clinical	DERS	DEBQ
Barnhart et al. (2021-S1)	United States	258	50.38	36.5 (11)	Non-clinical	DERS	EES
Barnhart et al. (2021-S2)	United States	77	79	20.3 (2.5)	Non-clinical	DERS	DEBQ
Braden et al. (2018)	United States	189	64.9	41.78 (13.61)	Clinical	DERS	nr
Braden et al. (2021)	United States	258	50	36.52 (11.02)	Non-clinical	DERS	EES
Braden et al. (2023)	United States	63	96.8	47 (12.5)	Clinical	DERS	EES
Cella et al. (2022)	Italy	973	45.9	14.17 (1.25)	Non-clinical	DERS	DEBQ
Crockett et al. (2015)	United States	552	60.5	19.25 (2.15)	Non-clinical	DERS	nr
Czepczor-Bernat & Brytek-Matera (2021)	Poland	298	nr	34.08 (9.5)	Non-clinical	DERS	TFEQ
da Fonseca et al. (2023)	Brazil	34	100	29 (nr)	Clinical	DERS	TFEQ
Debeuf et al. (2018)	Belgium	109	52.3	13.49 (1.64)	Non-clinical	FEEL-KJ	DBEQ
Demirci (2018-S1)	Türkiye	33	66.7	14.3 (2.3)	Non-clinical	DERS	DEBQ
Demirci (2018-S2)	Türkiye	27	59.3	12.5 (3.4)	Clinical	DERS	DEBQ
Demirci (2018-S3)	Türkiye	25	68	13.9 (2.6)	Clinical	DERS	DEBQ
Echeverri-Alvarado et al. (2020)	United States	360	50.6	36.6	Clinical	DERS	DEBQ
Ferrell et al. (2020)	United States	77	79	20.25	Non-clinical	DERS	EES
Fisher et al. (2017)	United Kingdom	632	72.31	34 (14.2)	Non-clinical	DERS	TFEQ
Gouveia et al. (2019)	Portugal	245	49.38	14.49 (1.71)	Clinical	DERS	DEBQ
Guerrini-Usubini et al. (2023)	Italy	600	48.7	25.4 (5.14)	Non-clinical	DERS	DEBQ
Guerrini-Usubini et al. (2021)	Italy	437	51.3	25.2 (5.12)	Non-clinical	DERS	DEBQ
Jones & Herr (2018)	United States	103	83	19.3 (1.4)	Non-clinical	DERS	EMAQ
Jones et al. (2019)	United States	136	52.2	42.25 (11.24)	Clinical	DERS	DEBQ
Kocak & Cagatay (2024)	Türkiye	400	65	42 (6.91)	Non-clinical	DERS	EES
Lyvers et al. (2022)	Australia	532	71	24.77	Non-clinical	NMRS	DEBQ
McAtamney et al. (2021)	United Kingdom	136	64.7	32	Non-clinical	DERS	nr
Michopoulos et al. (2015)	United States	1110	80.4	39.6 (nr)	Non-clinical	EDS	DEBQ
Mikhail et al. (2022)	United States	311	100	22.09 (3.21)	Non-clinical	DERS	DEBQ
Mohoric et al. (2023)	Croatia	482	51.03	15 (0.31)	Non-clinical	DERS	TFEQ
Munguia et al. (2021)	Mexico	160	75.62	20.1 (1.7)	Non-clinical	DERS	DEBQ
Naeimijoo et al. (2021)	Iran	292	58.21	16.11 (0.99)	Non-clinical	DERS	DEBQ
Prefit & Szentágotai -Tătar (2018)	Romania	269	100	23.87 (6.98)	Non-clinical	DERS	TFEQ
Richmond et al. (2025)	United States	739	61.7	53.45 (16.61)	Non-clinical	DERS	DEBQ
Spinner et al. (2024)	United States	163	57.7	14.5 (1.6)	Non-clinical	CBCL	EES
Sultson et al. (2019)	Estonia	605	100	29.8 (9.6)	Non-clinical	DERS	PNEES
Taube-Schiff et al. (2015)	United States	1383	79.17	44.72 (10.59)	Clinical	DERS	EES
Vandewalle et al. (2014)	Belgium	110	57.3	13.59 (1.64)	Clinical	FEEL-KJ	DEBQ
Vandewalle et al. (2016)	Belgium	81	55.6	12.86 (1.65)	Non-clinical	FEEL-KJ	DEBQ
Vervoort et al. (2022)	Belgium	572	49	nr (nr)	Clinical	FEEL-KJ	DEBQ
Watford et al. (2019)	United States	189	64.2	41.87 (13.64)	Clinical	DERS	EES
Williams (2017)	United States	144	83.2	45.9 (nr)	Clinical	DERS	DEBQ
Yang et al. (2023)	China	494	62.8	20.1	Non-clinical	DERS	EES
Yilmaz Kafali et al. (2021)	Türkiye	123	55.28	15.24 (1.85)	nr	DERS	TFEQ

Note. DERS = Difficulties in Emotion Regulation Scale; FEEL-KJ = Questionnaire to Assess Children's and Adolescents' Emotion Regulation Strategies; NMRS = Negative Mood Regulation Scale; EDRS = Emotional Dysregulation Scale; CBCL: Child Behavior Checklist; DEBQ = Dutch Eating Behavior Questionnaire; EES = Emotional Eating Scale; TFEQ = Three-Factor Eating Questionnaire; EMAQ = Emotional Appetite Questionnaire; PNEES = Positive-Negative Emotional Eating Scale; nr = not reported.

2.5. Quality assessment

An adapted version of the Newcastle–Ottawa Scale (NOS) for cross-sectional studies (Carra et al., 2025; Nieto-Retuerto et al., 2025) was employed to evaluate the quality of the methodology used in the studies included in the meta-analysis. The NOS is one of the tools most used worldwide to assess quality and risk of bias in observational studies included in systematic reviews and meta-analyses (Luchini et al., 2017). Three scoring ranges are proposed: low risk of bias (7 to 9), moderate risk (4 to 6), and high risk (0 to 3) (Carra et al., 2025; Zeng et al., 2015). In the absence of clear guidelines for this procedure in meta-analysis including cross-sectional studies (Luchini et al., 2017), the authors determined a cutoff criterion of a minimum score of seven (Drukker et al., 2021). The risk of bias of all 42 included studies was assessed independently by the third and sixth authors. The overall inter-rater agreement between the two evaluators was substantial, with a Cohen's kappa of .70 (see Table S1 and Table S2 of Supplementary Online Materials; SOM). A third researcher, experienced with the scoring procedure, resolved any discrepancies between the two primary rates. A final agreed-upon rating was assigned to each study (see Table S3 of SOM). Since the variability of NOS scores was minimal (all studies scored between 8 and 9), the risk of bias was not used as a moderator, despite initial intentions.

2.6. Data analyses

Data analyses were conducted in R (R Core Team, 2014) with RStudio (Posit team, 2025), and the R package *metafor* (Viechtbauer, 2010). To analyze the relationship between ED and EE, we fitted random-effects models following the guidelines of Rosenthal (1979), Hunter and Schmidt (2004), and Cumming (2013). First, we calculated the relationship between the variables, including both positive ($k = 7$) and negative ($k = 35$) aspects of EE. This total measure was also used to conduct a moderation analysis based on the valence of EE (i.e., positive vs. negative). Subsequently, however, we focused on negative EE and conducted the following moderation analyses and meta-regressions.

2.6.1. Effect sizes

To report the magnitude of the relationship between negative/positive ED and EE, Pearson's *r* was used as the effect size. If studies did not report *r* as the effect size, relevant transformations were performed using Psychometrica online calculator (Lenhard & Lenhard, 2016). The effect sizes were computed using Fisher's z-transformation (*r*-to-Z) and corresponding variances, following the procedure described by Borenstein

(2022). Specifically, we used the "escalc" function (with the measure "COR", using correlations, as can be seen in the database) from the "metafor" package (Viechtbauer, 2010). As specified in the software, this function applies this transformation automatically. Results were then back-transformed to *r* for ease of interpretation. Additionally, when studies did not report sufficient data to extract an effect size, we contacted the authors to request the necessary data. For some studies (e.g., Braden et al., 2018), more than one effect size was provided; therefore, to avoid multicollinearity issues, we either selected the effect sizes corresponding to a total ED score or conducted separate meta-analyses for specific emotions associated with EE (boredom, depression, anxiety/anger).

2.6.2. Publication bias

Publication bias refers to the inclination to publish more studies with statistically significant effects, as opposed to those with null effects. Such bias results in both the literature and subsequent meta-analyses being biased. To address publication bias, two different analyses were conducted: the correlation rank test (Begg & Mazumdar, 1994) and regression test (Sterne & Egger, 2005). Both tests are based on the standard error of the observed results to verify the asymmetry of the funnel plot. Publication bias can be considered absent when both tests are not significant, whereas if at least one test is significant, the risk of publication bias is considered possible; if both are significant, the risk is considered high. Furthermore, following the recommendations of Viechtbauer (2015), an analysis of standardized residuals and Cook's distances (Cook & Weisberg, 1982) was also carried out to identify outliers (when the standardized residual is greater than ± 2.914) or overly influential values (when the Cook's distance exceeds a certain threshold in relation to the number of observations).

To ensure the robustness of the effects, Fail-safe *N* tests (Rosenthal, 1979) were performed. This test refers to how many additional—or missing—studies with zero effect size would be necessary to convert a significant *p*-value into a non-significant result. If the fail-safe *N* value is small, it would be very likely that the true effect is actually zero (Borenstein, 2022). Although it is considered that the larger this value, the greater the confidence that the observed effect size estimate is reliable, Rosenthal (1979) proposed a conservative criterion for Fail-safe *N*, suggesting that it can be considered safe if it exceeds $5k + 10$.

2.6.3. Heterogeneity analysis

To examine possible heterogeneity, the *Q* test for heterogeneity, the I^2 and τ^2 were used. The *Q* test assesses whether the distribution of effect sizes around the mean is wider than expected from sampling error alone, thus suggesting that a random-effects model is more appropriate. When the value of *Q* is statistically significant, this assumption is confirmed. On the other hand, the I^2 statistic describes the percentage of between-study variation that is due to heterogeneity rather than chance (i.e., the percentage of true variability). A percentage greater than 75% would indicate a high level of heterogeneity and, therefore, the need to perform moderation analyses that would clarify such heterogeneity. Finally, τ^2 together with its standard error, indicates the absolute value of the true variance (i.e., heterogeneity), expressed in terms of the effect size scale (in this case, in Pearson's *r*).

2.6.4. Effect size interpretation

To establish comparable criteria for reported effects, we adopted the following standards: Small effects were defined as $r < .11$, medium effects as $r = .12$-$.19$, moderate effects as $r = .20$-$.29$, and large effects as $r > .29$. These criteria were based on the review by Gignac and Szodorai (2016). In this way, the effects identified in this meta-analysis can be evaluated with greater precision and compared to findings from other studies in the individual differences literature.

2.6.5. Moderation analysis

Different moderation analyses were carried out with the aim of examining how effect sizes varied according to certain variables, as well as the way in which these variables helped to explain the heterogeneity of effect sizes. The variables and levels explored were the following: Type of EE (positive *vs*. negative emotions), sample type (non-clinical *vs*. clinical), and type of emotion (boredom, depression, or anxiety/anger). In the case of quantitative moderator variables (age mean, percentage of females, and BMI mean), meta-regression analyses were performed.

3. Results

3.1. Descriptives

As shown in Table 2, the studies primarily focused on young adults ($M_{age} = 26.8$; $SD_{age} = 11.6$; % of females = 67.8%; $M_{BMI} = 27.2$). Of the total sample, 31% were classified as clinical, defined as samples explicitly recruited on the basis of a diagnosed medical or psychiatric condition or ongoing treatment (e.g., trauma-related disorders, substance use, obesity, or bariatric surgery candidates). Non-clinical samples accounted for 66.7% and consisted of community or convenience samples not selected based on clinical status. One study (2.38%) did not report sufficient information to determine clinical status. Classification was based only on the recruitment criteria and sample description provided in each study. Most studies were conducted in North American (42.10%) or European (31.57%) countries.

3.2. Main effects

The main results showed a positive and moderate relationship between ED and general EE (see Fig. 2): $r_{pooled} = .337$ (95% CI [.279, .396]; $k = 42$, $N = 13{,}185$) and, as observed, the studies exhibited high heterogeneity ($Q_{(45)} = 450.73$, $p < .001$; $I^2 = 93.44\%$). The Fail-safe *N* value was 31597, indicating a high reliability in the average effect size observed. We found a non-significant value in the rank correlation (i.e., $p = .153$), but a significant one in the Egger's regression test ($p = .002$),

Table 2
Moderation analyses for the relationship between negative EE and ED.

Moderator levels	*k*	*N*	r_{pooled} [95% CI]	$QM_{(df)}$	I^2	τ^2(*SE*)
Type of EE[a]						
Negative	35	11071	.385 [.330, .440]	$QM_{(1)} = 16.67$***	91.10%	.023 (.006)
Positive	7	1484	.093 [-.037, .222]			
Sample Type						
Clinical	10	1842	.344 [.280, .407]	$QM_{(1)} = 5.99$*	91.30%	.022 (.006)
Non-clinical	24	9736	.492 [.392, .592]			
Type of Emotion[b]						
Boredom	6	1327	.369 [.223, .502]	$QM_{(2)} = .299$	89.20%	.022 (.011)
Depression	5	1969	.422 [.227, .567]			
Anxiety/ Anger	5	1969	.404 [.259, .549]			

Note. EE = Emotional Eating.
[a] For this analysis, we used both positive and negative EE.
[b] For this analysis, we included only those studies that identified particular types of emotions when measuring ED ($k = 16$, $N = 5{,}265$). $QM_{(df)}$ refers to the *Q* test comparing effects across the levels of each moderator and their corresponding degrees of freedom. *$p < .05$, **$p < .01$, ***$p < .001$.

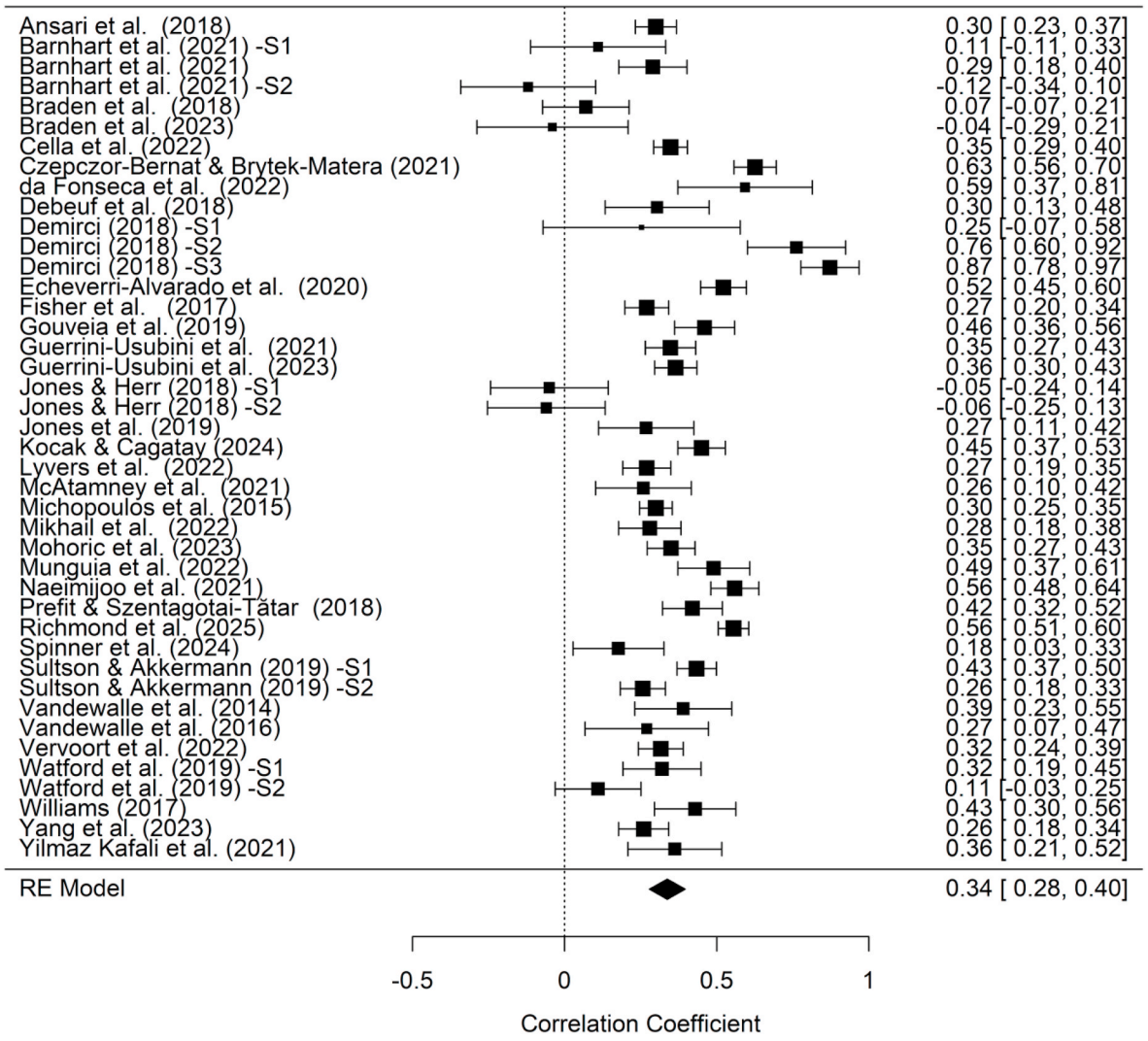

Fig. 2. Forest plot showing the observed outcomes and the estimate of the random-effects model for the relationship between emotional dysregulation and emotional eating (general).

indicating asymmetry in the funnel plot (see Fig. S1 in the SOM), suggesting publication bias. Finally, an examination of studentized residuals (i.e., residuals from a regression model divided by an estimate of their standard deviation) showed that the value reported in Demirci (2018, Study 3), exceeded 3.241 and could represent a potential outlier. Nonetheless, a replication of the effect without it revealed a similar overall trend.[1]

When the types of EE are separated, a positive and moderate main effect is found between ED and negative EE: $r_{pooled} = .385$ [.330, .440]; $Q_{(37)} = 346.07$, $p < .001$; $I^2 = 91.76\%$ ($k = 35$, $N = 11701$). Fail-safe N value was 28,990 and the non-significant values of the rank correlation and the Egger regression test ($p = .978$ and $p = .119$, respectively) indicated the absence of publication bias. In this case, Demirci's (2018) Study 3 was evaluated again as an outlier and deemed influential.[2] On the other hand, in the case of the relationship between ED and positive EE, the main effect was positive but non-significant: $r_{pooled} = .100$ [-.074, .274]; $Q_{(6)} = 26.97$, $p < .001$; $I^2 = 75.49\%$ ($k = 7$, $N = 1484$). Fail-safe N value indicated a less robust association (i.e., 50) and significant rank correlation and the Egger regression test values ($p = .014$ and $p < .001$, respectively) suggested the presence of publication bias (see Figs. S2–S5 in the SOM).

3.3. Moderation analyses

Based on the previously described hypotheses and explorations, we examined the first moderator (i.e., valence of EE) considering ED and positive and negative EE. Yet, considering the large difference in the total number of studies between positive and negative EE (i.e., $k = 7$ *vs.* 35), the remaining analyses were conducted only for negative EE.

Among the tests conducted (see Table 2), the moderation analyses reduced the heterogeneity to varying degrees. The portion of heterogeneity accounted for (i.e., in terms of R^2) varied from 0% to 30.29%. Additionally, τ^2 values were generally low, suggesting minimal true variation among studies in terms of measurement; however, in all cases, a large portion of heterogeneity remained non-random (i.e., high I^2 values).

When analyzing the differences in the levels of each moderator, differences were observed in the relationship between ED and EE. As expected, this relationship is conditioned by the type of EE, which was non-existent when positive EE was measured. Considering the sample

[1] Results obtained after removing the study showed an overall similar trend in the relationship between variables (i.e., $r_{pooled} = .326$, [.273, .378]), and similar levels of heterogeneity ($Q_{(40)} = 348.61$, $p < .001$; $I^2 = 91.60\%$).

[2] The results without the study again revealed a similar overall trend ($r_{pooled} = .372$, [.325, .418]) and subsequent levels of heterogeneity ($Q_{(33)} = 251.91$, $p < .001$; $I^2 = 88.19\%$).

types, the relationship between ED and EE was stronger among non-clinical participants (compared to clinical participants). Finally, it should be noted that the relationship between ED and EE did not vary depending on the emotion experienced (see the forest and funnel plots in Figs. S6–S11 of SOM).

As for the meta-regressions, although the remaining heterogeneity was reduced, none of the variables significantly moderated the relationship between ED and negative EE. These variables include mean age, percentage of women, and mean BMI (see Table 3).

4. Discussion

The present study aimed to conduct a meta-analysis of the association between ED and EE in adolescents and adults. To the best of our knowledge, this is the first effort to summarize empirical findings on the subject and explore possible moderators that may influence this association. The results revealed a positive and robust relationship between ED and EE (r_{pooled} = .337). Notably, this association is stronger when focusing solely on studies examining the influence of negative emotional states on EE (r_{pooled} = .385). Comparing these findings with a meta-analysis on a similar field that explores the relationship between stress and food intake in adults (Hill et al., 2022), the magnitude of the association observed in the present meta-analysis is substantially larger. Specifically, Hill et al. (2022) reported a smaller effect size (Hedges'g = .114, approximately $r \approx .057$ when converted), highlighting the comparatively stronger link between ED and EE.

The larger effect size in our meta-analysis compared to Hill et al.'s (2022) may be due to both conceptual and methodological factors. Conceptually, stress is a more general, variable environmental pressure with different levels and intensities, whereas ED is a more specific, individual-specific factor (Gratz & Roemer, 2004). Stress impacts eating differently depending on individual and contextual factors (e.g., Pool et al., 2015; Sproesser et al., 2014), while ED directly relates to feeling emotionally overwhelmed, which can trigger the use of food for regulation. Methodologically, our meta-analysis focused on self-report measures of ED and EE. These instruments assess individuals' perceived difficulties and habitual tendencies rather than experimentally induced responses, whereas Hill et al.'s (2022) meta-analysis also included experimental paradigms involving stress induction and behavioral outcomes (e.g., dietary restraint). Therefore, the greater similarity between the self-report measures used in our study may partly explain the larger effect size observed.

As mentioned above, our results showed a positive and moderate association between ED and EE. A tentative explanation for this result may be that people who have difficulty managing unpleasant emotions, such as anxiety or sadness, are more vulnerable to using food as a strategy to alleviate emotional distress (Braden et al., 2018; Crockett et al., 2015). A maladaptive pattern would therefore be generated in which, in the short term, food provides temporary relief from emotional distress; however, in the long term, the causes of the distress would still be present, so that the distress tends to recur if the source is not resolved. Following this reasoning, these dynamics would not only perpetuate difficulties in emotion regulation, but also maintain dependence on food as a coping strategy. In the long term, this dependence is linked to weight gain, feelings of guilt, and other negative outcomes (Frayn et al., 2018; Konttinen, 2020).

The high heterogeneity observed highlights the variability among the included studies. This emphasizes the need to explore variables that may influence the magnitude of the association between ED and EE. The first moderator examined the emotional valence associated with the desire to eat: ED showed a stronger association with EE in studies examining food intake driven by negative emotions, compared to those focusing on positive emotions. This finding suggests that unpleasant emotions may play a larger role in using food as a coping strategy (Braden et al., 2018). Conversely, the lack of a significant link between ED and positive EE may reflect the tendency for positive food intake to be driven by pleasant emotions, rather than maladaptive emotion regulation (Braden et al., 2018).

We theorized that sample type (non-clinical *vs*. clinical) would influence the association between ED and EE. However, contrary to our hypothesis, the relationship between ED and EE was stronger in non-clinical compared to clinical samples. One possible explanation is that the clinical subgroup may have high heterogeneity between studies. This could dilute the magnitude of the association between ED and EE (Riley et al., 2011). Furthermore, in clinical samples EE could be related to many factors (e.g., comorbidity with other disorders), so EE may not only be the result of a deficit in emotion regulation, but also reflect a much more complex psychological dynamic (Macht, 2008; van Strien, 2018).

Contrary to our expectations, age did not emerge as a significant moderating variable. Notwithstanding, this absence of moderating effect was also observed in Hill et al.'s (2022) meta-analysis. One possible explanation is that the association between ED and negative EE truly remains stable in adolescence and adulthood. With samples of older adults (which could not be addressed in this work due to the current lack of studies within those older age groups), a greater variability in the magnitude of the relationship between ED and EE could have been observed between age groups (Hill et al., 2018; Samuel & Cohen, 2018). Determining which alternative accounts for the lack of moderating effect in our meta-analysis can only be achieved when new studies examining the association between these variables in older samples are available.

Regarding BMI, this variable had no significant impact on the effect size estimates of our meta-analysis. Similarly, BMI did not function as a moderator either in the meta-analysis by Hill et al. (2022). Following the previous line of argument, it is possible that EE is such a common regulatory strategy that no significant differences emerge based on BMI. This pattern was also observed during the COVID-19 pandemic (e.g., Cecchetto et al., 2021; McAtamney et al., 2021). Additionally, BMI does not differentiate between muscle mass and body fat, does not account for the distribution of body fat, and does not consider individual variables, such as age or gender (Sweatt et al., 2024). Future research could explore other, more relevant variables related to body weight, such as body dissatisfaction (e.g., Halliwell & Dittmar, 2006), which has also been linked to EE.

With respect to the types of emotion that trigger EE, it should be noted that no difference was found between levels (boredom, depression, or anxiety/anger). This could suggest that EE does not respond to a specific type of emotion, but it may follow in response to any type of emotional distress. It is also worth noting that the emotions analyzed are

Table 3
Meta-regression analyses.

Predictor			95% CI		Model test		
	b	*SE*	*LL*	*UL*	$QM_{(3)}$	I^2	$\tau^2(SE)$
(Intercept)	.366	.155	.062	.671	.173	85.21%	.012 (.006)
Age (mean)	.002	.004	−.007	.010			
Women (percentage)	.000	.002	−.003	.004			
BMI (mean)	−.002	.007	−.016	.012			

Note. BMI = Body Mass Index. The test of residual heterogeneity indicates a substantial amount of unexplained variability ($QE_{(19)} = 71.85$, $p < .001$).

not mutually exclusive (for example, it is possible to experience boredom and sadness simultaneously), so it is plausible that this fact reduces the ability of the moderation analysis to detect meaningful differences (Mill et al., 2018).

Finally, as for the absence of moderating role of gender, our findings lead us to conclude that the impact of ED on negative EE is similar across genders. The mechanisms involved in triggering negative EE seem to be common in women and men. This aligns with the findings of the meta-analytic review of Hill et al. (2022), where no significant moderating effect of gender was observed on the link between stress and unhealthy eating behaviors. Based on these findings, prevention and interventions targeting broad emotion dysregulation and use of food as an emotion regulator may be applicable to both women and men, although conclusions regarding the necessity of gender-tailored approaches require confirmation using longitudinal or intervention designs.

4.1. Limitations and future directions

This work makes significant contributions, but it has limitations that should be considered when interpreting the findings. Firstly, a limitation of this meta-analysis is that only total ED scores were included, which means that associations with specific ED dimensions were overlooked. Future studies should examine specific ED dimensions to clarify their relationships with EE. Secondly, despite the analysis of potential moderators, high heterogeneity between studies persists, suggesting that other variables may better explain the examined relationship and should be further explored (e.g., the analysis of specific unpleasant emotions). Thirdly, the stronger association observed between ED and EE in non-clinical samples should be interpreted cautiously, as it may be influenced by uneven sample sizes or methodological differences rather than genuine population differences. Likewise, the predominance of studies conducted with female participants and within Western countries limits the generalizability of the results to men and other cultural contexts. The indirect approach of analyzing the link with gender (i.e., through the percentage of females in each study) may be less effective. A more robust design, such as an Individual Participant Data (IPD) meta-analysis, could provide better insights. In an IPD meta-analysis, original data are directly obtained from the researchers of each study and re-analyzed centrally, allowing for more accurate and combined results. Future studies could benefit from this approach. Methodologically, the scarcity of longitudinal studies poses a challenge to drawing robust conclusions about the causal direction of the relationship between ED and EE, as is the case with other meta-analyses in the field (e.g., Hill et al., 2022). Future research should prioritize developing more longitudinal studies to clarify the dynamic interplay between these variables. A final limitation is that the grey literature search did not include dedicated databases for unpublished academic work, and therefore some relevant studies may have been missed. Nevertheless, given that the included studies were predominantly peer-reviewed and that effect estimates were consistent across study designs, this limitation is unlikely to have substantially affected the overall conclusions. Future systematic reviews could address this by expanding the search strategy to further reduce the risk of publication bias.

Finally, regarding future directions, it would be valuable for future studies to focus on the moderating role of specific types of clinical samples (samples associated with feeding problems *vs.* samples involving individuals with other types of problems). Due to the limited number of investigations currently available focusing on clinical samples, this analysis was not possible here. We believe that this distinction could offer relevant nuances for understanding the mechanisms underlying the relationship between ED and EE. Furthermore, future studies may benefit from searching thesis and dissertation databases to locate more grey literature.

4.2. Practical implications

This meta-analysis offers practical implications for clinical and public health professionals. The findings highlight the need for preventive and therapeutic strategies focused on promoting adaptive emotion regulation. Approaches such as Dialectical Behavior Therapy (Linehan, 1993) could be effective. Previous studies suggest that working on distress tolerance, ED, and mindfulness can significantly reduce EE levels (Braden et al., 2022).

It is important to note that public health campaigns integrating emotional education with the promotion of healthy eating habits could help reduce the prevalence of EE. They may also lower related issues, such as obesity (e.g., Braet et al., 2008). Overall, this work provides a foundation for developing and implementing more effective preventive and therapeutic strategies.

5. Conclusion

This meta-analysis makes a valuable contribution by identifying a significant link between broad ED and EE in adolescents and adults. These findings stress the importance of developing adaptive emotion regulation strategies through preventive and therapeutic interventions. These strategies aim to address EE and its associated consequences. This presents a challenging yet essential task for professionals working at the intersection of emotional well-being and eating behaviors.

CRediT authorship contribution statement

Jara Mendia: Writing – original draft, Funding acquisition, Formal analysis, Conceptualization. **Yolanda Quiles:** Writing – review & editing, Writing – original draft, Conceptualization. **Aitziber Pascual:** Writing – review & editing, Funding acquisition, Conceptualization. **Álvaro Ruiz:** Writing – review & editing, Writing – original draft, Conceptualization. **Javier Manchón:** Writing – review & editing, Writing – original draft, Conceptualization. **Susana Conejero:** Writing – review & editing, Funding acquisition, Conceptualization. **José J. Pizarro:** Writing – review & editing, Formal analysis, Conceptualization. **Itziar Alonso-Arbiol:** Writing – review & editing, Writing – original draft, Funding acquisition, Conceptualization, Supervision.

Ethical statement

This meta-analysis was conducted in accordance with ethical guidelines for research synthesis. As it is a secondary analysis of previously published studies, no new data were collected from human or animal subjects, and therefore, no ethical approval was required.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.appet.2026.108490.

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