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REVIEW



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Do eating behavior traits predict energy intake and body mass index? A systematic review and meta-analysis

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Summary

At present, it is unclear whether eating behavior traits (EBT) predict objectively measured short-term energy intake (EI) and longer-term energy balance as estimated by body mass index (BMI). This systematic review examined the impact of EBT on BMI and laboratory-based measures of EI in adults (≥ 18 years) in any BMI category, excluding self-report measures of El. Articles were searched up until 28th October 2021 using MEDLINE, PsycINFO, EMBASE and Web of Science. Sixteen EBT were identified and the association between 10 EBT, EI and BMI were assessed using a random-effects meta-analysis. Other EBT outcomes were synthesized qualitatively. Risk of bias was assessed with the mixed methods appraisal tool. A total of 83 studies were included (mean $BMI = 25.20 \text{ kg/m}^2$, mean age = 27 years and mean sample size = 70). Study quality was rated moderately high overall, with some concerns in sampling strategy and statistical analyses. Susceptibility to hunger (n = 6) and binge eating (n = 7) were the strongest predictors of EI. Disinhibition (n = 8) was the strongest predictor of BMI. Overall, EBT may be useful as phenotypic markers of susceptibility to overconsume or develop obesity (PROSPERO: CRD42021288694).

KEYWORDS eating behavior, energy intake, obesity

INTRODUCTION 1

Obesity is a common, serious, and costly condition with significant health care and societal costs.¹ There is current interest in identifying psychological and physiological markers that characterize individuals who are susceptible to weight gain. Eating behaviors influence amount and types of foods eaten and hence energy intake (EI).² Eating behavior traits (EBT) are considered to be reliable, acquired indices of food-related behaviors.³ EBT have been extensively studied in an attempt to identify potential markers that detect tendency to overconsume.⁴ This has led to the development of several constructs and

psychometric measures, which aim to capture important individual differences in eating behavior and disordered eating symptomology, for example, the Three Factor Eating Questionnaire (TFEQ) with subscales for cognitive restraint, susceptibility to hunger and disinhibition.⁵ Many EBT have been reported to be associated with excess food intake.⁶⁻⁹ However, 'overconsumption' is often assessed by other self-report measures which are prone to misreporting and not truly representative of actual food intake.¹⁰ For example, in a systematic review on the associations between emotions and eating behavior, the majority of studies associated self-reported emotions with another self-reported EBT as a proxy of food intake.¹¹ Associations

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between EBT and EI have yet to be extensively reviewed and little is known about which EBTs influence objectively measured EI.

Previous systematic reviews have begun to answer these questions indirectly by examining associations between EBT and BMI.^{9,11,12} BMI is an index of long-term energy balance status. Vainik, Dagher, Dubé and Fellows¹² found that several different personality measures were consistently associated with BMI. However, very few measures included were eating-related. French, Epstein, Jeffery, Blundell and Wardle⁹ reviewed seven eating behavior dimensions and found that most of the available data showed positive cross-sectional associations with BMI. However, very few studies reported measures of EI and only four measures were eating-related. Overall, disinhibition had the largest empirical support to link it prospectively with weight gain and a narrative review also found that disinhibition plays a significant role in obesity, diet quality and uncontrolled eating patterns.¹³ As few measures associated with psychological eating behavior have been reviewed, this systematic review aimed to assess all possible psychological EBT.

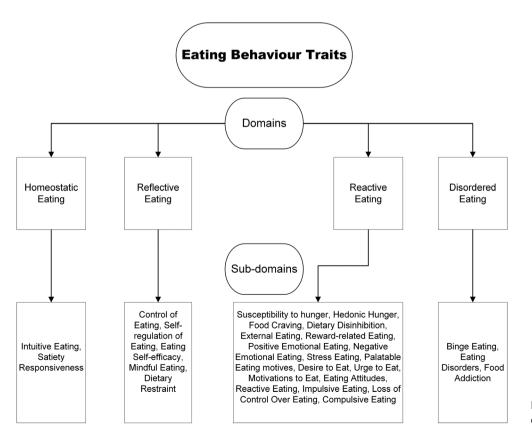
EBT are theoretical constructs operationalized through eatingrelated measures. There is currently no database of EBT, and studies often refer to these traits with different names, for example, overeating measures, eating behavior dimensions, appetite measures and eating attitudes. Consequently, a conceptual diagram was developed to capture the scales used to assess EBT and to a-priori allocate all of the collected scales to theoretical domains (see Figure 1). Firstly, a scoping exercise was conducted to create EBT domains, then subdomains were identified, which formed the basis for the search strategy. Secondly, this systematic review was conducted to identify studies that included measures of both El and BMI in the same population. This systematic review included only laboratory-based measures of El. Use of a controlled environment allows for more standardized, objective measurement of El.¹⁴ This is more accurate and precise than self-report El.¹⁵

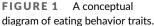
The primary aim of this systematic review was to examine whether EBT predicts short-term EI and whether there is evidence that this effect translates into longer term energy balance, as estimated by BMI. The specific research questions of the review were: (1) What measures are used to capture EBT? (2) How well do these measures predict short term EI? (3) How well do these measures predict BMI? (4) Do some EBT and questionnaires used to measure EBT better predict EI or BMI than others?

2 | METHODS

2.1 | Information sources and search strategy

Four electronic databases were searched: MEDLINE (Ovid), PsycINFO (Ovid), EMBASE (Ovid) and Web of Science (core collection). The MEDLINE, Embase and PsychInfo strategies were run simultaneously as a multi-file search in Ovid on 28th October 2021. Searches for all databases are included in Table S1. The conceptual diagram, alongside the identification of key databases formed the basis for the search strategy (see Figure 1). All proposed EBT and their domains were





included in the search. Key databases such as the UCL database for eating behavior questionnaires,¹⁶ NIH obesity measures,¹⁷ and Arab psychology scales¹⁸ were used to find relevant eating-related guestionnaires to include in the search. Because many studies use EBT but not in relation to measured eating behavior, the search also required studies to include key words related to eating behavior or eating traits (see Table S1). The two outcome measures were EI and BMI (and their related terms, see Table S1). Limits were set to include articles published in English (as translating studies into English could lead to relevant information being mistranslated) and humans. The search strategies were peer reviewed by three experts in the field (KB, JS, GF) prior to execution using the PRESS checklist.¹⁹ Duplicates were removed using Screenatron's 'deduplication' tool and were checked to ensure all duplicates found were actual duplications. This systematic review followed PRISMA guidelines²⁰ and was pre-registered with PROSPERO (registration number = CRD42021288694).

2.2 | Study selection, inclusion, and exclusion

Articles were included if they recruited adults (≥18 years including older adults) in any BMI category. The presence of the following was not excluded: type 2 diabetes, pre-menopausal/post-menopausal women, cardiovascular diseases. Those with the following comorbidities were excluded: Parkinson's disease, anorexia, bulimia nervosa, binge eating disorder, cancers, inflammatory bowel diseases, intellectual deficiency, psychiatric conditions, sleep disorders, those having undergone weight loss surgery (gastric bypass or bariatric surgery) and those who were pregnant. As binge eating was identified as an EBT in the scoping exercise, any study that used a binge eating scale (e.g., binge eating scale²¹) to classify binge eaters was included. However, if studies identified participants with binge eating disorder using a version of the DSM, for example, DSM-5,²² this was not recognized as an EBT and these studies were excluded. Studies that measured psychological variables, for example, stress or mood, were excluded unless an EBT was also measured, for example, emotional eating. In cases where studies measured other variables alongside EBT, the reviewers discussed whether these variables would be considered potential contaminates of the outcomes. For example, an alcohol preload or a food craving protocol were considered potential contaminates and were excluded if no data was given for a control condition. Self-report measures of EI were excluded as this systematic review focused on laboratory-based measures of EI but were saved for a future review. Laboratory-based measures of EI also included studies where EI was measured in a laboratory and weighed food was given to participants to take home and bring back to be re-weighed, as this measure of EI is considered in the literature to be ad-libitum EI.

All titles and abstracts were assessed for eligibility by the main author (CD) and were also independently screened by three researchers (MH, JS, GF), with uncertainty regarding eligibility discussed among the researchers. Reference lists from the resulting reviews were also screened to identify additional articles. Full texts were retrieved and assessed for eligibility by CD and were also OBESITY Reviews -WILEY-

independently assessed by JS and GF. Intervention studies that included results for a control condition or baseline measures were included. Studies that only measured one of the outcomes variables (El or BMI) were excluded. If a study measured both outcome variables but the results for one of the outcome measures was missing, the study authors were contacted for the missing outcome results. In this case, the study was included for data extraction as both outcome variables were measured. Thirty-three authors were contacted about missing data. Five authors responded with the necessary data.

2.3 | Data extraction and synthesis

Data was extracted by one author (CD) and was cross-checked for data extraction errors by JS and GF. Data was extracted using the data collection form for intervention reviews (randomized control trials and non-randomized control trials) from the Cochrane group.²³ The characteristics of each included article consisted of title, study ID. publication type, study design, aim of the study, ethical information, number of participants, population characteristics (age, BMI, % female, ethnicity, population description, method of recruitment, inclusion/ exclusion criteria), description of EBT, description of outcomes (method, definition, unit of measurement and validation information for the collection and measurement of BMI and EI), study funding sources, possible conflicts of interests, data and analysis (results, including number of participants, means and standard deviations where possible, unit of analysis, statistical methods used and appropriateness of these), key conclusions of study authors and references to other available studies. Where means and standard deviations were not reported, correlations, F-values or t-values were extracted, and standard errors were converted to standard deviations. Effects on El and BMI were examined using a random-effects meta-analysis because it was anticipated that there would be considerable betweenstudy heterogeneity. All analyses were carried out in the R environment, version 2021.09.0.24 The following R packages were used: dmetar,²⁵ esc,²⁶ tidyverse,²⁷ and meta.²⁸

Meta-analysis of the effect of each EBT on BMI and EI was performed when ≥2 effect sizes were available for each trait. For each analysis, effect sizes were pooled using the r value from correlations and were transformed to Fisher's Z.²⁹ Where correlations were not present, the available raw data were transformed into Fisher's Z. Where one study provided data for more than one questionnaire (e.g., all participants completed the DEBQ and TFEQ), the sample size was halved to prevent 'double counting', which can artificially inflate effect sizes and distort results.^{30,31} If studies provided data for multiple types of El outcome, for example, sweet, and savory El, or provided data for multiple preloads, the selected outcomes were pooled to provide a single 'total El' correlation, to prevent overpowering of a single study. Effect sizes based on Fisher's Z were interpreted as very small (≤0.05), small (>0.05-<0.10), moderate (>0.10-<0.30), large (>0.30-<0.40), and very large (≥0.40).³² The restricted maximum likelihood estimator was used to calculate heterogeneity variance and heterogeneity was also evaluated using the I-squared

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statistic (l^2), with values inferred as low (<25%), moderate (25%– 75%), and high (>75%). However, the l^2 statistic was interpreted cautiously when the number of studies in each meta-analysis was <7. In this case, it is advised that confidence intervals should supplement the l^2 statistic; therefore, confidence intervals were also provided for each meta-analysis.³³ Knapp-Hartung adjustments³⁴ were also used to calculate the confidence interval around the pooled effect. The Knapp-Hartung adjustment can reduce the chance of false positives, especially when the number of studies is small.³⁵ The results of the meta-analyses are presented as forest plots, which include the authors, standard error (SE), questionnaire type, correlation (COR), 95% confidence intervals, weight and Hedges G³⁶ that is converted from Fisher's *Z*.

Further analysis of outliers and influential cases was conducted using a leave-one-out analysis in addition to identifying influential cases, and results are reported for both the main meta-analyses and further analyses where outliers or influential cases were removed. Publication bias was assessed by visually inspecting the funnel plot and Egger's regression test when the number of included studies was <10. Duval and Tweedie's trim-and-fill method was also used. Two subgroup analyses were conducted to test whether the effect of restraint on BMI and EI was influenced by the type of questionnaire used because the questionnaires used to measure restraint varied across studies. Additionally, a subgroup analysis tested whether the use of a preload influenced the correlation between restraint and EI. Studies were split into 'no preload' and 'preload' categories. For the preload studies, effect sizes were conducted for both no preload and preload groups, and sample size was halved to prevent double counting.^{30,31} Where studies included multiple preload conditions, the largest preload condition was used to calculate the effect size. Subgroup analyses were also conducted to test the influence of meal type (1 test meal vs. multiple test meals) where there were ≥10 effect sizes available for each EBT and ≥ 2 per category.

2.4 | Quality assessment

To assess risk of bias, the excel version of the mixed method appraisal tool (MMAT) for appraising studies was used.³⁷ It is a critical appraisal tool designed for systematic mixed studies reviews (including qualitative, quantitative, and mixed methods studies). The MMAT has been pilot tested and has high content validity.³⁸ The following questions were answered to determine the risk of bias: is the sampling strategy relevant to address the research question, is the sample representative of the target population, are the measurements appropriate, is the risk of nonresponse bias low and is the statistical analysis appropriate to answer the research question? The four assessment items represented flaws if answered "No/cannot tell." It is discouraged to calculate an overall score from the ratings of each criterion; therefore, a detailed discussion of the ratings follows. Quality assessment was completed by the main reviewer (CD) and was cross-checked by JS and GF.

3 | RESULTS

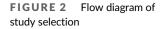
2,760 articles were retrieved from the four databases. After deduplication, 1955 articles remained for title and abstract screening. The reasons for excluding any studies that did not meet the inclusion criteria during full text screening are shown in the flow diagram in Figure 2. Seventy-seven papers met the inclusion criteria and were included in this systematic review, with some papers consisting of several studies. In total, 83 studies were included. Several studies could not be synthesized for each EBT meta-analysis; therefore, the number of studies included in each meta-analysis varies. Where metaanalysis was not possible, associations between the EBT and outcome variables are qualitatively summarized. Table 1 gives the included questionnaires used to measure EBT and their abbreviations.

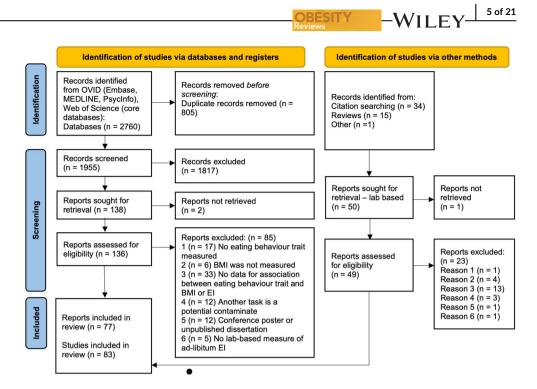
3.1 | Study characteristics

Study characteristics are summarized in Table 2 (see Table S2 for more detailed characteristics). The studies were published between 1975 and 2021. All data from the studies are cross-sectional. The following EBT were identified, restraint (n = 50), disinhibition (n = 17), emotional eating (n = 12), binge eating (n = 8), external eating (n = 7), susceptibility to hunger (n = 6), hedonic hunger (n = 4), satiety responsiveness (n = 3 via Satiety Quotient, SQ) and n = 1 via AEBQ), intuitive eating (n = 3), food craving (n = 1), mindful eating (n = 1), eating disorders (n = 1), food addiction (n = 1), eating self-efficacy (n = 1), control of eating (n = 1), reward-related eating (n = 1). The mean (range) of the total sample size was 70 (18-273). The mean (range) age of participants was 27 years (19-59 years); BMI was 25.20 kg/m² (21.0-36.8 kg/m²). Males and females were included in 25 studies, females only in 56 studies, males only in 1 study and no data was given for 1 study. BMI was objectively measured in 52 studies, self-reported in 10 studies, and in 21 studies, there was no information for how BMI was measured. Measures of EI varied widely across studies. EI was measured over one test meal in 69 studies and across multiple test meals in 14 studies. Studies also used various types of food to measure El. Overall, snack foods were the most popular El measure (n = 29). Multiple food items (n = 21), pasta (n = 8), cookies (n = 5), ice cream (n = 5), popcorn (n = 3), sandwiches (n = 3), vending machines (n = 2), beef casserole (n = 2), risotto (n = 1), pizza (n = 1), milkshake (n = 1), Boost (a nutritionally complete liquid supplement, n = 1) and breakfast foods (n = 1) were also used.

3.2 | Study quality

Overall, studies scored relatively high on study quality for four out of five quality criteria. Studies scored low for sampling strategies because none used probability sampling and most recruited via opportunity sampling. Studies did define their target population by presenting inclusion and exclusion criteria, and some included a-priori sample size calculations. Respondents and the target population were well





matched and the reasons why certain individuals were excluded were made clear. All questionnaires have been validated and in 51 studies, BMI was measured objectively in the laboratory. The measurements of EI were, for most studies, not previously validated and the most frequently used type of food was snacks (n = 28). The extent to which findings of eating behavior, using energy dense snacks can generalize to wider and more typical eating behavior is unknown. The majority of studies also attempted to reduce possible confounders by providing participants with a standardized breakfast, testing during normal lunch hours and keeping testing times between participants consistent.

The risk of non-response bias in studies included in this systematic review was low, no studies reported high drop-out rates. Lastly, study quality was high for the appropriateness of statistical analysis used in studies. However, in some studies, the method used to split participants into groups was a limitation. Using a median split to identify low or high scorers could have misclassified individuals and lead to null findings.¹¹ Indeed, van Strien, Herman, Anschutz, Engels and de Weerth¹¹⁹ found that if a median split was used, their results would have become non-significant. Additionally, in Dalton, Hollingworth, Blundell and Finlayson,⁶⁰ 40% of the participants were unclassified based on their SQ score. Therefore, any findings cannot be generalized to a wider population if almost half of the sample could not be classified into a level of satiety responsiveness.

3.3 | Study findings

3.3.1 | Intuitive eating

A meta-analysis of 2 studies (n = 262) found no significant correlation between intuitive eating and EI (r = 0.32 [-0.30, 2.00], p = 0.096). A meta-analysis of 3 studies (n = 317) found that intuitive eating was negatively correlated with BMI (r = -0.26 [-0.45, -0.04], p = 0.036, Figure S1) and this represents a moderate effect size. Heterogeneity was low ($l^2 = 0.0\%$, Q = 1.66, p = 0.436). One study was identified as an influential case.⁵⁷ With this study removed from the meta-analysis, the overall effect size increased; however, the effect became non-significant (r = -0.30 [-0.86, 0.58], p = 0.156). The trim-and-fill method suggested no missing studies.

3.3.2 | Mindful eating

Anderson, Reilly, Schaumberg, Dmochowski and Anderson⁵⁸ found no significant association between mindful eating and EI (r = 0.15) or mindful eating and BMI (r = 0.01).

3.3.3 | Satiety responsiveness

Only one effect size was available; therefore, meta-analysis could not be conducted. However, of the four studies that measured satiety responsiveness, three reported that higher satiety responsiveness as associated with reduced $El^{60,62,63}$ and one reported no significant effect.⁶² A meta-analysis of 3 studies (n = 197) found a moderate negative correlation between satiety responsiveness and BMI (r = -0.18 [-0.31, -0.04], p = 0.031, Figure S2). Heterogeneity was low ($l^2 = 0.0\%$, Q = 0.41, p = 0.815). No studies were identified as outliers or influential cases. Two missing studies were suggested to the right (adjusted r = -0.15 [-0.24, -0.06], p = 0.009).

TABLE 1 Table of questionnaires included and their abbreviations

Questionnaire	Abbreviation	Subscales	Reference paper
Intuitive Eating Scale	IES	Reliance On Hunger and Satiety (RHSC), Unlimited Permission to Eat (UPE), Eating for Physical Rather Than Emotional Reasons (EPR)	Tylka ³⁹
Intuitive Eating Scale-2	IES-2	RHSC, UPE, EPR, Body-Food Choice Congruence (B-FCC)	Tylka and Kroon Van Diest ⁴⁰
Mindful Eating Questionnaire	MEQ	Disinhibition, Awareness, External Cues, Emotional Response, Distraction	Framson, Kristal, Schenk, Littman, Zeliadt and Benitez ⁴¹
Satiety Quotient	SQ	Desire To Eat, Hunger, Fullness, Satiety, Prospective Food Consumption (PFC)	Green, Delargy, Joanes and Blundell ⁴²
Adult Eating Behavior Questionnaire	AEBQ	Hunger (H), Food Responsiveness (FR), Emotional Over-Eating (EOE), Enjoyment of Food (EF), Satiety Responsiveness (SR), Emotional Under- Eating (EUE), Food Fussiness (FF), Slowness in Eating (SE)	Hunot, Fildes, Croker, Llewellyn, Wardle and Beeken ⁴³
Three Factor Eating Questionnaire	TFEQ	Disinhibition, Dietary Restraint, Susceptibility to Hunger	Stunkard and Messick ⁵
Restraint Scale	RS		Herman and Mack ⁴⁴
Revised Restraint Scale	RRS		Herman, Polivy and Silver ⁴⁵
Dutch Eating Behavior Questionnaire	DEBQ	Restraint Eating, Emotional Eating, External Eating	van Strien, Frijters, Bergers and Defares ⁴⁶
Control of Eating Questionnaire	COEQ	Craving Control, Positive Mood, Craving for Sweet, Craving for Savory	Dalton, Finlayson, Blundell and Hill ⁴⁷
Power of Food Scale	PFS	Food Available, Food Present, Food Tasted	Lowe, Butryn, Didie, Annunziato, Thomas, Crerand, Ochner, Coletta, Bellace and Wallaert ⁴⁸
Food Craving Inventory	FCI	High fats, Sweets, Carbohydrates, Fast- Food Fats and Subjective, Behavioral	White, Whisenhunt, Williamson, Greenway and Netemeyer ⁴⁹
Binge Eating Scale	BES		Gormally, Black, Daston and Rardin ²¹
Eating Disorders Examination Questionnaire	EDE-Q	Restraint, Eating Concern, Shape Concern, Weight Concern	Fairburn, Cooper and O'Connor ⁵⁰
Eating Disorders Assessment Scale	EDAS	Restrained Eating, Binge Eating, Purging, Preoccupation with Body Image, and Body Weight.	Akkermann, Herik, Aluoja and Järv ⁵¹
Eating Disorders Diagnostics Scale	EDDS	Anorexia Nervosa, Bulimia Nervosa, Binge Eating Disorder, Noneating Disordered	Stice, Telch and Rizvi ⁵²
Eating Disorders Inventory	EDI-2	EBT related subscales: Drive for Thinness, Bulimia, Body Dissatisfaction	Garner ⁵³
Eating Self-Efficacy	WEL	Negative Emotions, Availability, Social Pressure, Physical Discomfort, Positive Activities	Clark, Abrams, Niaura, Eaton and Rossi ⁵⁴
Reward-based Eating Drive Questionnaire	RED-9, RED-13, RED-X5	Loss of Control Over Eating, Lack of Satiety, Preoccupation with Food	Vainik, Eun Han, Epel, Janet Tomiyama, Dagher and Mason ⁵⁵
Questionnane			-

3.3.4 | Restraint

Meta-analysis of 49 studies (n = 2,594) showed a small but significant, negative correlation between restraint and El (52 study arms, r = -0.07, [-0.12, -0.01], p = 0.015, Figure 3). Heterogeneity was moderate ($l^2 = 41.8\%$, Q = 87.70, p = 0.001). Four outliers were identified^{68,74,78,79} and the one-study-removed procedure identified

one influential case.⁷⁴ With these outliers removed, the effect size slightly increased (r = -0.09 [-0.14, -0.05], p < 0.001). Heterogeneity was low ($l^2 = 10.2\%$) and the Q test was non-significant (Q = 52.32, p = 0.275). The funnel plot did not suggest any evidence of publication bias (Figure S3), no missing studies were suggested, and Egger's test was non-significant (p = 0.680). The association between restraint and El was influenced by the type of questionnaire used to

TABLE 2 Eating behavior traits: Summary of included studies

	Questionnaire used		Associatio	ns with
	to assess eating			
Eating behavior trait	behavior trait	Cross-sectional study reference	EI	BMI
Intuitive eating (IE)	IES	Anderson, Schaumberg, Anderson and Reilly ⁵⁷	NS and \uparrow	\downarrow
	IES	Anderson, Reilly, Schaumberg, Dmochowski and Anderson ⁵⁸	NS	NS
	IES-2	Ruzanska and Warschburger ⁵⁹	NS and \uparrow	\downarrow
		Total	3 NS 2 ↑	1 NS 2 ↓
Mindful eating	MEQ	Anderson, Reilly, Schaumberg, Dmochowski and Anderson ⁵⁸	NS	NS
		Total	1 NS	1 NS
Satiety Responsiveness	SQ	Dalton, Hollingworth, Blundell and Finlayson ⁶⁰	\downarrow	NS
	SQ desire to eat SQ for PFC SQ for fullness SQ for hunger	Drapeau, Blundell, Therrien, Lawton, Richard and Tremblay ⁶¹	NS NS ↓ NS	NS NS NS NS
	SQ	Drapeau, Jacob, Panahi and Tremblay ⁶²	NS	NS
	AEBQ	Zuraikat, Roe, Smethers, Reihart and Rolls ⁶³	\downarrow	NS*
		Total	4 NS 3 ↓	7 NS
Dietary restraint	TFEQ	Anderson, Reilly, Schaumberg, Dmochowski and Anderson ⁵⁸	NS	Î
	TFEQ	Ard, Desmond, Allison and Conway ⁶⁴	\downarrow	
	TFEQ	Bellisle, Dalix, Airinei, Hercberg and Péneau ⁶⁵	NS	NS
	TFEQ	Bryant, Caudwell, Blundell, Hopkins and King ⁶⁶	NS	
	TFEQ	Chambers and Yeomans ⁶⁷	NS	NS
	RS	Coelho, Polivy, Herman and Pliner ⁶⁸	↑	Î
	RRS	Cools, Schotte and McNally ⁶⁹	\downarrow	
	RS	de Witt Huberts, Evers and de Ridder ⁷⁰ study 1	NS	Î
	RS	de Witt Huberts, Evers and de Ridder ⁷⁰ study 2	NS	Î
	RS	de Witt Huberts, Evers and de Ridder ⁷⁰ study 3	NS	NS
	DEBQ	Dweck, Jenkins and Nolan ⁷¹		Î
	RRS	Fedoroff, Polivy and Herman ⁷²	\downarrow	
	TFEQ	Finlayson, Blundell, Bordes, Griffioen-Roose and de Graaf ⁷³	NS	NS
	DEBQ	Finlayson et al. ⁷³	NS and \uparrow	
	RS	Guerrieri, Nederkoorn, Schrooten, Martijn and Jansen ⁷⁴	↑	Î
	TFEQ	Haynes, Lee and Yeomans ⁷⁵	\downarrow	NS
	DEBQ	Herhaus and Petrowski ⁷⁶	NS	NS
	RS	Herman and Mack ⁴⁴	\uparrow and \downarrow	NS
	RS	Herman, Polivy and Silver ⁴⁵	↑	
	DEBQ	Higgs, Williamson and Attwood ⁷⁷	NS	NS
	TFEQ	Hofmann and Friese ⁷⁸	Ļ	
	DEBQ	Hopkins et al. ¹¹³	Ļ	NS
	DEBQ	Jansen ⁷⁹ study 1	↑	NS
		Jansen ⁷⁹ study 2	↑	Î
	RS	Jansen, Merckelbach, Oosterlaan, Tuiten and Van Den Hout ⁸⁰	NS	Î

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(Continues)

TABLE 2 (Continued)

Eating b

	Questionnaire used		Associations with		
pehavior trait	to assess eating behavior trait	Cross-sectional study reference	EI	BMI	
	RS	Lattimore and Maxwell ⁸¹	NS		
	TFEQ	Martin, Williamson, Geiselman, Walden, Smeets, Morales and Redmann Jr ⁸²	NS	NS	
	TFEQ	McNeil, Lamothe, Cameron, Riou, Cadieux, Lafreniere, Goldfield, Willbond, Prud'homme and Doucet ⁸³	NS		
	RRS	Myhre, Buchwald, Kratz, Goldberg, Polivy, Melhorn, Schur and Cummings ⁸⁴	NS	NS	
	DEBQ	Oliver, Wardle and Gibson ⁸⁵	NS		
	DEBQ	Ouwens, van Strien and van der Staak ⁸⁶	NS		
	TFEQ	Ouwens, van Strien and van der Staak ⁸⁶	NS		
	RS	Ouwens, van Strien and van der Staak ⁸⁶	NS		
	TFEQ	Peluso ⁸⁷	NS	NS	
	TFEQ	Rideout, McLean and Barr ⁸⁸	\downarrow	NS	
	DEBQ	Robinson and Haynes ¹¹⁵	NS	NS	
	TFEQ	Rolls, Castellanos, Shide, Miller, Pelkman, Thorwart and Peters ⁸⁹	NS	NS	
	TFEQ	Ruddock, Field and Hardman ⁹⁰	↓	NS	
	TFEQ	Schoch and Raynor ⁹¹	\downarrow	Î	
	RRS	Schotte, Cools and McNally ⁹²	NS	1	
	RS	Shapiro and Anderson ⁹³	NS		
	DEBQ	Sim, Lee and Cheon ⁹⁴	NS		
	TFEQ	Smith, Geiselman, Williamson, Champagne, Bray and Ryan ⁹⁵	NS	Ţ	
	RS and TFEQ	Stice, Fisher and Lowe ⁹⁶ study 1	NS	NS	
	TFEQ and EDEQ-R	Stice, Fisher and Lowe ⁹⁶ study 2	NS		
	TFEQ	Stice, Sysko, Roberto and Allison ⁹⁷	NS		
	TFEQ	Stinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁸	\downarrow	NS	
	DEBQ	van Strien and Ouwens ⁹⁹	NS		
	TFEQ	Visona and George ¹⁰⁰	NS	NS	
	DEBQ	Wallis and Hetherington ¹⁰¹	NS	NS	
	DEBQ	Wallis and Hetherington ¹⁰²	Ļ	Ť	
	DEBQ	Wardle and Beales ¹⁰³	↑ 1	NS	
	TFEQ	Westenhoefer, Broeckmann, Münch and Pudel ¹⁰⁴	NS	Ť	
	RRS	Westenhoefer, Broeckmann, Münch and Pudel ¹⁰⁴	↑	1 1	
	TFEQ	Yeomans and Coughlan ¹⁰⁵	NS	NS	
	TFEQ	Yeomans, Tovey, Tinley and Haynes ¹⁰⁶		NS	
	TFEQ	Zambrowicz, Schebendach, Sysko, Mayer, Walsh and Steinglass ¹⁰⁷	Ļ		
	EDE-2	Zambrowicz, Schebendach, Sysko, Mayer, Walsh and Steinglass ¹⁰⁷	NS		
		Total	36 NS	24	
			9 ↑	NS	
			13↓	1 4 ↑	

TABLE 2 (Continued)

	Questionnaire used to assess eating		Associat	ions wit
Eating behavior trait	behavior trait	Cross-sectional study reference	EI	BMI
Control of eating	COEQ Craving control Positive mood Lack of Craving for sweet ^a	Dalton, Finlayson, Blundell and Hill ⁴⁷	$\downarrow \\ \downarrow$	$\downarrow \\ \downarrow \\ \downarrow$
		Total	3 ↓	3 ↓
Hedonic hunger/food reward sensitivity	PFS	Appelhans, Liebman, Woolf, Pagoto, Schneider and Whited ¹⁰⁸	NS	NS
	PFS	Ely, Howard and Lowe ¹⁰⁹	NS and \uparrow	¢
	PFS	Finlayson et al. ⁷³	NS	NS
	PFS	Nolan-Poupart, Veldhuizen, Geha and Small ¹¹⁰	NS	
		Total	4 NS 1 ↑	2 NS 1 ↑
Food craving	FCI	Martin, O'Neil, Tollefson, Greenway and White ¹¹¹	Ť	
		Total	1 ↑	
Dietary disinhibition	TFEQ	Ard, Desmond, Allison and Conway ⁶⁴	NS	
	TFEQ	Bryant, Caudwell, Blundell, Hopkins and King ⁶⁶	NS	
	TFEQ	Chambers and Yeomans ⁶⁷	NS and \uparrow	
	TFEQ	Epstein, Lin, Carr and Fletcher ¹¹²	↑	Î
	TFEQ	Finlayson, Blundell, Bordes, Griffioen-Roose and de Graaf ⁷³	NS and ↑	Ť
	TFEQ	Haynes, Lee and Yeomans ⁷⁵	↑	NS
	TFEQ	Higgs, Williamson and Attwood ⁷⁷	NS	NS
	TFEQ	Martin, Williamson, Geiselman, Walden, Smeets, Morales and Redmann Jr ⁸²		NS
	TFEQ	McNeil, Lamothe, Cameron, Riou, Cadieux, Lafreniere, Goldfield, Willbond, Prud'homme and Doucet ⁸³	NS	
	DEBQ	Ouwens, van Strien and van der Staak ⁸⁶	Ť	
	TFEQ	Ouwens, van Strien and van der Staak ⁸⁶	Ť	
	TFEQ	Ruddock, Field and Hardman ⁹⁰	Ť	NS
	TFEQ	Smith, Geiselman, Williamson, Champagne, Bray and Ryan ⁹⁵	Î	Ť
	TFEQ	Stinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁸	NS	î
	TFEQ	Westenhoefer, Broeckmann, Münch and Pudel ¹⁰⁴	↑	Ť
	TFEQ	Yeomans and Coughlan ¹⁰⁵	NS	Ť
	TFEQ	Yeomans, Tovey, Tinley and Haynes ¹⁰⁶	↑	Î
	TFEQ	Zambrowicz, Schebendach, Sysko, Mayer, Walsh and Steinglass ¹⁰⁷	NS	
		Total	9 NS 10 ↑	4 NS 7 ↑
Susceptibility to Hunger	TFEQ	Bryant, Caudwell, Blundell, Hopkins and King ⁶⁶	Ť	
	TFEQ	Finlayson, Blundell, Bordes, Griffioen-Roose and de Graaf ⁷³	NS	NS
	TFEQ	McNeil, Lamothe, Cameron, Riou, Cadieux, Lafreniere, Goldfield, Willbond, Prud'homme and Doucet ⁸³	Ť	
	TFEQ	Stinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁸	NS	Ŷ
	TFEQ	Westenhoefer, Broeckmann, Münch and Pudel ¹⁰⁴	Ť	Ŷ

(Continues)

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TABLE 2 (Continued)

Eating behavior trait behavior trait Cross-sectional study reference EI MI TEQ Zambrowicz, Schobendich, Sysko, Mayer, Walsh and NS Stitelinglins ²⁰ NS 31 External eating DEBQ Dueck, Jenkins and Nokan ²¹ I NS 31 External eating DEBQ Dueck, Jenkins and Nokan ²¹ I NS NS DEBQ Bellog Robinson and Haynes ¹²¹ NS NS NS DEBQ Robinson and Haynes ¹²¹ NS NS NS NS DEBQ Walsrine, Donker and Okuwen ²¹⁴ NS NS NS NS DEBQ walsrine, Donker and Okuwen ²¹⁴ NS NS NS NS DEBQ walsrine, Donker and Okuwen ²¹⁴ NS NS NS NS DEBQ DeBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 4 NS NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 4 NS NS NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 4 NS NS NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁵ study 3 NS <		Questionnaire used to assess eating		Associati	ons with
Steinglass ¹⁰⁷ Steinglass	Eating behavior trait		Cross-sectional study reference	EI	BMI
External exitingDEBQDeckQ, Lenkins and Notan ¹² NBDEBQFinlayson et al. ³ NSNSDEBQRokosche, Kernos and Tiggennam ^{11,4} NSNSDEBQRokosche, Kernos and Tiggennam ^{11,4} NSNSDEBQWan Strien, Donker and Ouversi ¹⁴ NSNSDEBQEvers, de Ridder and Adriaanst ¹¹⁵ study 3NSNSDEBQEvers, de Ridder and Adriaanst ¹¹⁵ study 4NSNSDEBQEvers, de Ridder and Adriaanst ¹¹⁵ study 4NSNSDEBQEvers, de Ridder and Adriaanst ¹¹⁵ study 5NSNSDEBQElequeFinlayson et al. ³ NSNSDEBQRokosche, Myhbrow, Horgan and Stubbs ¹¹³ NSNSDEBQRokosche, Myhbrow, Horgan and Stubbs ¹¹⁴ NSNSDEBQRokosche, Myhbrow, Horgan and Stubbs ¹¹⁵ NSNSDEBQRokosche, Myhbrow, Horgan and Stubbs ¹¹⁴ NSNSDEBQRokosche, Myhbrow, Horgan and Stubbs ¹¹⁵ NSNSDEBQRokosche, Myhbrow, Horgan and Stubbs ¹¹⁴ NSNSDEBQRokosche, Myhbrow, Horgan and Stubbs ¹¹⁵ NSNSDEBQRokosche, Myhbrow, Horgan and Stubbs ¹¹⁵ NSNSDEBQRokosche, Myhbrow, Horgan and Stubbs ¹¹⁵ NSNSDEBQNationa		TFEQ		NS	
DEBQFinlayson et al. ⁷³ (1NSDEBQHopkins, Michalowska, Whybrow, Horgan and Stubbis ¹¹³ NSNSDEBQKakoschke, Kemps and Tiggenann ^{11,4} (1NSDEBQRobinson and Haynes ^{11,12} NSNSDEBQvan Strien, Donker and Ouwens ¹⁶ (1NSDEBQvan Strien, Donker and Ouwens ¹⁶ NSNSEmotional eatingDEBQEvers, de Ridder and Adriaanse ^{11,7} study 3NSNSDEBQEvers, de Ridder and Adriaanse ^{11,7} study 4NSNSNSDEBQEvers, de Ridder and Adriaanse ^{11,7} study 5NSNSNSDEBQEvers, de Ridder and Adriaanse ^{11,7} study 5NSNSNSDEBQEvers, de Ridder and Adriaanse ^{11,7} study 5NSNSNSDEBQEvers, de Ridder and Adriaanse ^{11,7} study 5NSNSNSDEBQEDEQCiver, de Ridder and Adriaanse ^{11,7} study 5NSNSNSDEBQEDEQOiter, Wardle and Gibson ^{12,1} NSNSNSNSDEBQNa Strien, Hornay Adviseon and Anisman ^{11,8} NSNSNSNSNSNSDEBQvan Strien, Hornay Adviseon and Anisman ^{11,8} NS <td></td> <td></td> <td>Total</td> <td></td> <td></td>			Total		
DEBQ Haykins, Michalowska, Whybrow, Horgan and Stubist ¹¹³ NS NS DEBQ Kakockke, Kemps and Tiggemann ¹³⁴ I I DEBQ Robinson and Haynes ¹¹⁵ NS NS NS DEBQ wan Strine, Dorker and Ouwens ¹²⁶ NS NS S DEBQ van Strine, Dorker and Ouwens ¹²⁶ NS NS NS Emotional eating DEBQ DeEQ Vers. de Ridder and Adriaanse ¹¹⁷ study 3 NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 3 NS NS NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 3 NS NS NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 3 NS	External eating	DEBQ	Dweck, Jenkins and Nolan ⁷¹		NS
DEBQ Kakoschke, Kenps and Tiggenann ^{14.} 1 DEBQ Robinson and Haynes ^{11.9} NS NS DEBQ van Strien, Donker and Ouwers ^{12.9} NS NS DEBQ wan Strien and Ouwers ^{12.9} NS NS Total 3 NS 4 NS Emotional eating DEBQ Dveck, Jenkins and Nolan ⁷¹ NS NS DEBQ Dveck, Jenkins and Adrianse ^{11.9} study 3 NS NS DEBQ Evers, de Ridder and Adrianse ^{11.9} study 4 NS NS DEBQ Evers, de Ridder and Adrianse ^{11.9} study 4 NS NS DEBQ Evers, de Ridder and Adrianse ^{11.9} study 4 NS NS DEBQ Evers, de Ridder and Adrianse ^{11.9} study 4 NS NS DEBQ Oliver, Wardle and Gibson ^{11.9} NS NS DEBQ Rabpopow, Abizid, Matheson and Anisman ^{11.8} NS NS DEBQ van Strien, Donker and Ouwers ^{12.9} NS NS DEBQ Valis and Hetherington ^{10.1} NS NS DEBQ Walis and Hetherington ^{10.1} NS NS DEBQ Walis and Hetherington ^{10.2} NS NS DEBQ Walis and Hetherington ^{10.2} NS NS DEBQ		DEBQ	,	↑	NS
DEBQ Robinson and Haynes ¹¹⁵ NS NS NS DERQ van Strien, Donker and Ouwens ¹¹⁰ (1		DEBQ	· · · · · ·	NS	NS
DEBQ van Strien, Donker and Ouwens ³⁴⁴ i DEBQ van Strien and Ouwens ³⁴⁴ NS Total 31 NS NS Emotional eating DEBQ Deveck, Jenkins and Nolar ⁷¹ NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 3 NS NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 5 NS NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 5 NS NS NS DEBQ Finalyson et al. ⁷² NS and 1 T NS		DEBQ		Î	
DEBQ van Strien and Duwers ⁸⁹ NS Total 3 NS 3 NS 3 NS 3 NS 3 NS 3 NS 3 NS 3 NS		DEBQ		NS	NS
Fordal \$\begin{aligned}{3} & \begin{aligned}{3} & aligne		DEBQ		Î	
Enotional eating DEBQ Dveck, Jenkins and Nolan ²⁴ NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 3 NS NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 5 NS NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 5 NS NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 5 NS NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 5 NS NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 5 NS NS NS DEBQ Hopkins, Michalowska, Whybrow, Horgan and Stubbs ¹¹³ NS NS NS DEBQ Raspopow, Abizidi, Matheson and Anisman ¹¹⁸ NS NS NS DEBQ van Strien, Donker and Ouvens ¹²⁶ 1 T T DEBQ van Strien and Ouvens ¹²⁶ NS NS NS DEBQ van Strien and Ouvens ¹²⁶ NS NS NS DEBQ Walis and Hetherington ¹⁶¹ NS NS NS DEBQ Walis and Hetherington ¹⁶² NS NS NS DEBQ Alger, Seagle and Ravusin ¹²⁰ I NS NS BES Dalton, Blundell and Fin		DEBQ	van Strien and Ouwens ⁹⁹	NS	
DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 3 NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 4 NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 5 NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 5 NS NS DEBQ Hopkins, Michalowska, Whybrow, Horgan and Stubbs ¹¹⁰ NS NS DEBQ Hopkins, Michalowska, Whybrow, Horgan and Stubbs ¹¹⁰ NS NS DEBQ Oliver, Wardle and Glison ⁶⁵ NS NS NS DEBQ Raspopow, Abizaid, Matheson and Anisman ¹¹⁸ NS NS NS DEBQ van Strien, Donker and Ouwens ¹¹⁶ NS NS NS DEBQ van Strien and Ouwens ⁷⁷ T T NS NS DEBQ van Strien and Ouwens ⁷⁹ T NS NS NS DEBQ van Strien and Ouwens ⁷⁹ T T NS NS NS DEBQ van Strien and Ouwens ⁷⁰ T T NS NS NS DEBQ van Strien and Ouwens ⁷⁰ T T NS NS NS NS DEBQ Valis and Hetherington ¹⁰² NS Alger, Sceagle and Ravussin ¹²⁰ T NS <t< td=""><td></td><td></td><td>Total</td><td></td><td>4 NS</td></t<>			Total		4 NS
DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 4 NS NS DEBQ Evers, de Ridder and Adriaanse ¹¹⁷ study 5 NS NS DEBQ Finlayson et al. ⁷³ NS and 1 T DEBQ Hopkins, Michlaowska, Whybrow, Horgan and Stubbs ⁴¹³ NS NS DEBQ Hopkins, Michlaowska, Whybrow, Horgan and Stubbs ⁴¹³ NS NS DEBQ Raspopov, Abizaid, Matheson and Anisman ¹¹⁸ NS NS DEBQ Raspopov, Abizaid, Matheson and Anisman ¹¹⁸ NS NS DEBQ van Strien, Donker and Ouwens ¹¹⁴ T T DEBQ van Strien, Merman, Anschutz, Engels and de Weerth ¹¹⁹ T T DEBQ van Strien and Ouwens ¹²⁴ T T T DEBQ van Strien and Duwens ¹²⁰ T NS NS DEBQ van Strien and Ravussin ¹²⁰ NS S 1 S DEBQ Valitis and Hetherington ¹²⁰ T NS S 1 BES DEBQ Animae, Kreegipuu and Vainik ¹²¹¹ NS I	Emotional eating	DEBQ	Dweck, Jenkins and Nolan ⁷¹	NS	NS
DEBQEvers, de Ridder and Adriaanse ¹¹⁷ study 5NSNSNSDEBQFinlayson et al.73NS and 11DEBQHopkins, Michalowska, Whybrow, Horgan and Stubbs ¹¹³ NSNSDEBQOliver, Wardle and Gibson ¹⁵ NSNSDEBQRaspopow, Abizaid, Matheson and Anisman ¹¹⁴¹ NSNSDEBQRobinson and Haynes ¹¹⁵ NSNSDEBQvan Strien, Donker and Ouwens ¹¹⁶ 11DEBQvan Strien, Herman, Anschutz, Engels and de Weerth ¹¹⁹ 11DEBQvan Strien and Ouwens ¹¹⁶ 111DEBQWallis and Hetherington ¹⁰¹ NS3 (1)3 (1)DEBQWallis and Hetherington ¹⁰² 1NS3 (1)DEBQWallis and Hetherington ¹⁰² 1NS3 (1)DEBQWallis and Hetherington ¹⁰³ 1NS3 (1)DEBQMager, Seagle and Ravussin ¹²⁰ 1NS3 (1)DEBQDaton, Blundell and Finlayson ¹²³ 1NS1BESDaton, Blundell and Finlayson ¹²³ 1NS1BESElsonDaton, Slundell and Finlayson ¹²³ 1NS3 (1)BESElsonStinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁸ NS11BESStinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁸ NS4 NS3 (1)BESElsonStinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁸ NS4 NS3 (1)BESElsonson ³⁴ Anderso		DEBQ	Evers, de Ridder and Adriaanse ¹¹⁷ study 3	NS	NS
DEBQFinlayson et al.73NS and 1NDEBQHopkins, Michalowska, Whybrow, Horgan and Stubbs113NSNSDEBQOliver, Wardle and Gibson55NSNSDEBQRaspopow, Abizaid, Matheson and Anisman118NSNSDEBQRobinson and Haynes115NSNSDEBQvan Strien, Donker and Ouwens116NSNSDEBQvan Strien, Horman, Anschutz, Engels and de Weerth119NSNSDEBQvan Strien and Ouwens670NSNSDEBQvan Strien and Ouwens670NSNSDEBQWallis and Hetherington101NSNSDEBQWallis and Hetherington102NSNSDEBQWallis and Hetherington102NSNSDEBQWallis and Hetherington102NSNSDEBQWallis and Hetherington102NSNSDEBQWallis and Hetherington102NSNSDEBQWallis and Hetherington102NSNSDEBQDatton, Blundell and Finlayson1221NSBESDatton, Blundell and Finlayson1231NSBESDatton, Slundell and Finlayson123NS1BESStanson, Votruba, Venti, Krakoff, Gluck and Perez98NS1BESStanson, Votruba, Venti, Krakoff, Gluck and Perez98NS1BESEnderson, Stanson, Votruba, Venti, Krakoff, Gluck and Perez98NS1BESStanson, Votruba, Venti, Krakoff, Gluck and Perez98NS1Anderson39Cong, Meyer,		DEBQ	Evers, de Ridder and Adriaanse ¹¹⁷ study 4	NS	NS
DEBQ Hopkins, Michalowska, Whybrow, Horgan and Stubbs ¹¹³ NS NS DEBQ Oliver, Wardle and Gibson ⁸⁵ NS NS DEBQ Raspopov, Abizaid, Matheson and Anisman ¹¹⁸ NS NS DEBQ Robinson and Haynes ¹¹⁵ NS NS DEBQ van Strien, Donker and Ouwens ¹¹⁶ ↑ ↑ DEBQ van Strien, Herman, Anschutz, Engels and de Weerth ¹¹⁹ ↑ ↑ DEBQ van Strien, Herman, Anschutz, Engels and de Weerth ¹¹⁹ ↑ ↑ DEBQ van Strien, Herman, Anschutz, Engels and de Weerth ¹¹⁹ ↑ ↑ DEBQ van Strien, Herman, Anschutz, Engels and de Weerth ¹¹⁹ ↑ ↑ DEBQ van Strien, Herman, Anschutz, Engels and Rouwens ⁹ ↑ NS \$ DEBQ Wallis and Hetherington ¹⁰¹ NS \$ \$ \$ \$ DEBQ Wallis and Hetherington ¹⁰² 10 NS \$ \$ \$ \$ BES Alger, Seagle and Ravussin ¹²⁰ 10 NS \$ \$ \$ \$ \$ \$		DEBQ	Evers, de Ridder and Adriaanse ¹¹⁷ study 5	NS	NS
DEBQOliver, Wardle and Gibson*5NSDEBQRaspopov, Abizaid, Matheson and Anisman*1*8NSNS*DEBQRobinson and Haynes*1*5NSNSDEBQvan Strien, Donker and Ouwens*1*6↑↑DEBQvan Strien, Herman, Anschutz, Engels and de Weerth*1*9↑↑DEBQvan Strien and Ouwens*1*6↑↑DEBQvan Strien and Ouwens*0*↑↓DEBQValitis and Hetherington*10*NSNSDEBQWallis and Hetherington*10*NS§ NSDEBQWallis and Hetherington*10*NS§ NSDEBQWallis and Hetherington*10*10 NS§ NSDEBQWallis and Hetherington*10*10 NS§ NSDEBQWallis and Hetherington*10*10 NS§ NSDEBQWallis and Hetherington*10*10 NS§ NSDEBQNager, Scagle and Ravussin*10*10 NS§ NSBESDaton, Blundell and Finlayson*2*↑NS*BESDaton, Blundell and Finlayson*2*↑NS*BESFinlayson et al.*3↑NS↑BESFinlayson, Arlotti, Daton, King and Blundelf*NS↑BESStinson, Votruba, Venti, Krakoff, Gluck and Perez*0*NS↑BESEnlayson, Arlotti, Daton, King Gluck and Perez*0*NS↑BESEnlayson, Arlotti, Schaumberg, Dmochowski and Anderson***NS↓Eating disordersEDDSAnderson***NS↓ED1-2Long, Meyer, Le		DEBQ	Finlayson et al. ⁷³	NS and \uparrow	Ŷ
DEBQRaspopow, Abizaid, Matheson and Anisman ¹¹⁸ NSNS*DEBQRobinson and Haynes ¹¹⁵ NSNSDEBQvan Strien, Donker and Ouwens ¹¹⁶ IIDEBQvan Strien and Ouwens ⁹⁷ TIDEBQvan Strien and Ouwens ⁹⁷ TIDEBQvan Strien and Ouwens ⁹⁷ NSNSDEBQWallis and Hetherington ¹⁰¹ NSNSDEBQWallis and Hetherington ¹⁰² NSNSDEBQWallis and Hetherington ¹⁰² NSNSDEBQWallis and Hetherington ¹⁰² NSNSDEBQKager, Seagle and Ravussin ¹²⁰ NSNSEDASArumae, Kreegipuu and Vainik ¹²¹ NSNSBESDalton, Blundell and Finlayson ¹²² INSBESDalton, Slundell and Finlayson ¹²³ INSBESDalton, Slundell and Finlayson ¹²³ NSIBESStinson, Arlotti, Dalton, King and Blundell ⁴ IIBESStinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁹ NSIBESStinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁹ NSIStinson, Notruba, Venti, Krakoff, Slunck and Perez ⁹⁹ NSIEnting disordersEDDSAnderson ⁸⁹ NSIEDI-2Log, Meyer, Leung and Wallis ¹²⁵ NSITota2NSJIITota2NSIIIFood addictionYFAS 2.0Schule, Sonneville and Gearhardt ¹²⁶ NS		DEBQ	Hopkins, Michalowska, Whybrow, Horgan and Stubbs ¹¹³	NS	NS
DEBQRobinson and Haynes ¹¹⁵ NSNSDEBQvan Strien, Donker and Ouvers ¹¹⁶ 11DEBQvan Strien, Herman, Anschutz, Engels and de Weerth ¹¹⁹ 11DEBQvan Strien and Ouvers ⁹⁹ 11DEBQVallis and Hetherington ¹⁰¹ NSNSDEBQWallis and Hetherington ¹⁰² NSNSDEBQWallis and Hetherington ¹⁰² 1NSDEBQVallis and Hetherington ¹⁰² NSNSDEBQKagr, Seagle and Ravussin ¹²⁰ 1NSBinge eatingBESAlger, Seagle and Ravussin ¹²⁰ 1NSBESDaton, Blundell and Finlayson ¹²² 1NSNSBESDaton, Blundell and Finlayson ¹²³ 1NSNSBESFinlayson et al. ⁷³ NS1NSNSBESStinson, Votruba, Venti, Krakoff, Gluck and Perez ⁷⁸ NS11BESStinson, Votruba, Venti, Krakoff, Gluck and Perez ⁷⁸ NS11Fating disordersEDDSAnderson, Reilly, Schaumberg, Dmochowski and Anderson ⁸⁸ NS11Fold addictionYEA 2.0Stoute, Sonewille and Gearbardt ¹²⁶ NS11		DEBQ	Oliver, Wardle and Gibson ⁸⁵	NS	
DEBQvan Strien, Donker and Ouwens ⁹¹⁴ 1DEBQvan Strien, Herman, Anschutz, Engels and de Weerth ¹¹⁹ 1DEBQvan Strien and Ouwens ⁹⁹ 1DEBQWallis and Hetherington ¹⁰¹ NSDEBQWallis and Hetherington ¹⁰² NSDEBQWallis and Hetherington ¹⁰² NSDEBQWallis and Hetherington ¹⁰² NSDEBQMaler, Seagle and Ravussin ¹²⁰ 10 NSBinge eatingBESAlger, Seagle and Ravussin ¹²⁰ BESDaton, Blundell and Finlayson ¹²² 1BESDaton, Blundell and Finlayson ¹²³ 1BESDaton, Blundell and Finlayson ¹²³ 1BESFinlayson, Arlotti, Daton, King and Blundell ⁴ 1BESStinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁹ NSBESStinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁹ NS <trr>BES<td< td=""><td></td><td>DEBQ</td><td>Raspopow, Abizaid, Matheson and Anisman¹¹⁸</td><td>NS</td><td>NS*</td></td<></trr>		DEBQ	Raspopow, Abizaid, Matheson and Anisman ¹¹⁸	NS	NS*
DEBQvan Strien, Herman, Anschutz, Engels and de Weerth ¹¹⁹ iDEBQvan Strien and Ouwens ⁹⁹ iDEBQWallis and Hetherington ¹⁰¹ NSNSDEBQWallis and Hetherington ¹⁰² NS3 rDEBQWallis and Hetherington ¹⁰² no3 rDEBQWallis and Hetherington ¹⁰² 10 NS3 rBinge eatingRESAlger, Seagle and Ravussin ¹²⁰ iBESDalton, Blundell and Finlayson ¹²² iNS*BESDalton, Blundell and Finlayson ¹²² iNS*BESDalton, Blundell and Finlayson ¹²² iNS*BESFinlayson, Arlotti, Dalton, Kring and Blundell ⁴ iiBESSinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁹ NSiBESEtinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁹ NSiBESEtinson, Notruba, Schaumberg, Dunochowski and Anderson ⁹⁸ NSiFating disordersEDDSAnderson ⁹⁸ NSiEDI-2Long, Meyer, Leung and Walisl ¹²⁵ NSiiFood addictionYFAS 2.0Schule, Sonneville and Geahardt ¹²⁶ NSi		DEBQ	Robinson and Haynes ¹¹⁵	NS	NS
DEBQvan Strien and Ouwens ⁹⁹ iDEBQWallis and Hetherington ¹⁰¹ NSNSDEBQWallis and Hetherington ¹⁰² NS2Total10 NS2 nS2Bige eatingBESAlger, Seagle and Ravussin ¹²⁰ iNSBESDaton, Blundell and Finlayson ¹²² 1NS*NS*BESDaton, Blundell and Finlayson ¹²³ 1NS*NS*BESDaton, Blundell and Finlayson ¹²³ 1NS*1BESFinlayson rat.73NS11BESStinson, Votruba, Venti, Krakoff, Gluck and Perez.98NS1BESStinson, Votruba, Venti, Krakoff, Gluck and Perez.98NS1EDDSAnderson Si ⁶⁹ NS11CollEDDSAnderson Si ⁶⁹ NS1Ford addictionVFAS 2.0Stolute, Soneville and Gearhardt ¹²⁴ NS1Food addictonKFAS 2.0Stolute, Soneville and Gearhardt ¹²⁴ NS1Food addictonKDSAnderson Si ⁶⁹ 111Food addictonKDSStolute, Soneville and Gearhardt ¹²⁴ NS1Food addictonKFAS 2.0Stolute, Soneville and Gearhardt ¹²⁴ NS1Food addictonKFAS 2.0Stolute, Soneville and Gearhardt ¹²⁴ NS1Food addictonKFAS 2.0Stolute, Soneville and Scilla Anderson SiNS1Food addictonKFAS 2.0Stolute, Soneville and Scilla Anderson Si11 <td></td> <td>DEBQ</td> <td>van Strien, Donker and Ouwens¹¹⁶</td> <td>Ť</td> <td></td>		DEBQ	van Strien, Donker and Ouwens ¹¹⁶	Ť	
DEBQ Walis and Hetherington ¹⁰¹ NS NS DEBQ Walis and Hetherington ¹⁰² NS NS BEQ Total 10 NS 2 NS Bige eating BES Alger, Seagle and Ravussin ¹²⁰ 1 NS BES Arumae, Kreegipuu and Vainik ¹²¹ 1 NS NS BES Dalton, Blundell and Finlayson ¹²² 1 NS NS BES Dalton, Blundell and Finlayson ¹²³ 1 NS NS BES Dalton, Blundell and Finlayson ¹²³ 1 NS NS BES Dalton, Blundell and Finlayson ¹²⁴ NS NS NS BES Nasser, Gluck and Geliebter ¹²⁴ NS NS NS BES Stinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁸ NS 1 BES Stinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁸ NS 1 BES EDDS Anderson ⁵⁸ NS 1 Anderson ⁵⁸ Long, Meyer, Leung and Walls ¹²⁵ NS 1 EDI-2 Long, Meyer, Leung and Walls ¹²⁵ NS 1 Total 201 21NS 1 Food addiction YES 2.0 Schule, Sonneville and Genhardt ¹²⁶		DEBQ	van Strien, Herman, Anschutz, Engels and de Weerth ¹¹⁹		Ŷ
DEBQ Walis and Hetherington ¹⁰² NS Total 10 NS 2 Binge eating BES Alger, Seagle and Ravussin ¹²⁰ EDAS Arumae, Kreegipuu and Vainik ¹²¹ 1 BES Dalton, Blundell and Finlayson ¹²² 1 BES Dalton, Blundell and Finlayson ¹²³ 1 BES Naser, Gluck and Geliebter ¹²⁴ NS BES Naser, Gluck and Geliebter ¹²⁴ NS BES Stinson, Votruba, Venti, Krakoff, Gluck and Preze ⁷⁸⁰ NS BES Stinson, Votruba, Venti, Krakoff, Gluck and Preze ⁷⁸⁰ NS BES Stinson, Votruba, Venti, Krakoff, Gluck and Preze ⁷⁸⁰ NS BES Stinson, Votruba, Venti, Krakoff, Gluck and Preze ⁷⁸⁰ NS BES Stinson, Nortuba, Venti, Krakoff, Gluck and Preze ⁷⁸⁰ NS BES Stinson, Votruba, Venti, Krakoff, Gluck and Preze ⁷⁸⁰ NS BES Stinson, Votruba, Venti, Krakoff, Gluck and Preze ⁷⁸⁰ NS BES Stinson, Nortuba, Venti, Krakoff, Gluck and Preze ⁷⁸⁰ NS EDDS Anderson Reilly, Schaumberg, Dunchowski and Anderson ⁵⁸⁰ <td></td> <td>DEBQ</td> <td>van Strien and Ouwens⁹⁹</td> <td>↑</td> <td></td>		DEBQ	van Strien and Ouwens ⁹⁹	↑	
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Binge eating BES Alger, Seagle and Ravussin ¹²⁰ I Binge eating EDAS Arumae, Kreegipuu and Vainik ¹²¹ ↑ NS BES Dalton, Blundell and Finlayson ¹²² ↑ NS BES Dalton, Blundell and Finlayson ¹²³ ↑ NS* BES Dalton, Blundell and Finlayson ¹²³ ↑ NS* BES Finlayson, Arlotti, Dalton, King and Blundell ⁴ ↑ ↑ BES Finlayson et al. ⁷³ NS ↑ BES Sinson, Voruba, Venti, Krakoff, Gluck and Perez ⁹⁸ NS ↑ BES Sinson, Voruba, Venti, Krakoff, Gluck and Perez ⁹⁸ NS ↑ Pote Total Alderson ⁵⁸ NS ↑ Pote Long, Meyer, Leung and Wallis ¹²⁵ NS ↓ Food addiction YFA 2.0 Schulte, Sonneville and Gearbardt ¹²⁶ NS ↓		DEBQ	Wallis and Hetherington ¹⁰²		NS
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BESDalton, Blundell and Finlayson ¹²² 1NS*BESDalton, Blundell and Finlayson ¹²³ 1NS*BESFinlayson, Arlotti, Dalton, King and Blundell ⁴ 11BESFinlayson et al. ⁷³ NS1BESNasser, Gluck and Geliebter ¹²⁴ NSNSBESStinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁸ NS1BESTotal3 NS4 NSAtherson, Seilly, Schaumberg, Dmochowski and Anderson ⁵⁸ NS1EDDSAnderson, Seilly, Schaumberg, Dmochowski and Anderson ⁵⁸ NS1EDI-2Long, Meyer, Leung and Wallis ¹²⁵ NS1Total2 NS1 1Food addictionYFAS 2.0Schulte, Sonneville and Gearhardt ¹²⁶ NSNS	Binge eating	BES	Alger, Seagle and Ravussin ¹²⁰		
BESDalton, Blundell and Finlayson1231NS*BESFinlayson, Arlotti, Dalton, King and Blundell411BESFinlayson et al.73NS1BESNasser, Gluck and Geliebter124NSNSBESStinson, Votruba, Venti, Krakoff, Gluck and Perez28NS1BESStinson, Votruba, Venti, Krakoff, Gluck and Perez28NS4 NSAt 1Total3 NS4 NSEating disordersEDDSAnderson, Reilly, Schaumberg, Dmochowski and Anderson58NS1ED1-2Long, Meyer, Leung and Wallis125NS1TotalTotal2 NS1 1Food addictionYFAS 2.0Schulte, Sonneville and Gearhardt126NS		EDAS	Arumae, Kreegipuu and Vainik ¹²¹	↑	NS
BESFinlayson, Arlotti, Dalton, King and Blundell ⁴ ↑↑BESFinlayson et al. ⁷³ NS↑BESNasser, Gluck and Geliebter ¹²⁴ NSNSBESStinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁸ NS↑Total3 NS 4 ↑3 ↑EDDSAnderson, Reilly, Schaumberg, Dmochowski and Anderson ⁵⁸ NS↑EDI-2Long, Meyer, Leung and Wallis ¹²⁵ NS1 ↑Total2 NS1 ↑Food addictionYFAS 2.0Schulte, Sonneville and Gearhardt ¹²⁶ NSNS		BES	Dalton, Blundell and Finlayson ¹²²	↑	NS*
BESFinlayson et al. ⁷³ NS1BESNasser, Gluck and Geliebter ¹²⁴ NSNSNSBESStinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁸ NS1TotalSanSa3 13EDDSAnderson, Reilly, Schaumberg, Dmochowski and Anderson ⁵⁸ NS1ED1-2ED1-2Long, Meyer, Leung and Wallis ¹²⁵ NS1Total2 NS1 1Food addictionYFAS 2.0Schulte, Sonneville and Gearhardt ¹²⁶ NSNS		BES	Dalton, Blundell and Finlayson ¹²³	↑ (NS*
BESNasser, Gluck and Geliebter124NSNSBESStinson, Votruba, Venti, Krakoff, Gluck and Perez98NS↑Total3 NS 4 ↑3 ↑Eating disordersEDDSAnderson, Reilly, Schaumberg, Dmochowski and Anderson58NS↑ED1-2Long, Meyer, Leung and Wallis125NS↓Total2 NS1 ↑Total2 NS1 ↑		BES	Finlayson, Arlotti, Dalton, King and Blundell ⁴	↑	Î
BESStinson, Votruba, Venti, Krakoff, Gluck and PerezNS1Total3 NS4 NSEDDSAnderson, Seilly, Schaumberg, Dmochowski and Anderson ⁵⁸ NS1ED1-2Long, Meyer, Leung and Wallis ¹²⁵ NS1Total2 NS11Food addictionYFAS 2.0Schulte, Sonneville and Gearhardt ¹²⁶ NSNS		BES	Finlayson et al. ⁷³	NS	Î
Total3 NS 4 1 3 1Eating disordersEDDSAnderson, Reilly, Schaumberg, Dmochowski and Anderson ⁵⁸ NS1EDI-2Long, Meyer, Leung and Wallis ¹²⁵ NS1Total2 NS1 1Food addictionYFAS 2.0Schulte, Sonneville and Gearhardt ¹²⁶ NS		BES	Nasser, Gluck and Geliebter ¹²⁴	NS	NS
k f d f d f d f d f d f d f d f d f d f		BES	Stinson, Votruba, Venti, Krakoff, Gluck and Perez ⁹⁸	NS	Ť
Anderson ⁵⁸ EDI-2 Long, Meyer, Leung and Wallis ¹²⁵ NS Total 2 NS 1 ↑ Food addiction YFAS 2.0 Schulte, Sonneville and Gearhardt ¹²⁶ NS			Total		
Total 2 NS 1 ↑ Food addiction YFAS 2.0 Schulte, Sonneville and Gearhardt ¹²⁶ NS NS	Eating disorders	EDDS		NS	†
Food addictionYFAS 2.0Schulte, Sonneville and Gearhardt126NSNS		EDI-2	Long, Meyer, Leung and Wallis ¹²⁵	NS	
			Total	2 NS	1 ↑
Total 4 NG 4 NG	Food addiction	YFAS 2.0	Schulte, Sonneville and Gearhardt ¹²⁶	NS	NS
iotai 1 NS 1 NS			Total	1 NS	1 NS

TABLE 2 (Continued)

	Questionnaire used to assess eating		Associa	tions with
Eating behavior trait	behavior trait	Cross-sectional study reference	EI	BMI
Eating self-efficacy	WEL	Vijayvargiya, Chedid, Wang, Atieh, Maselli, Burton, Clark, Acosta and Camilleri ¹²⁷	Ļ	
		Total	1↓	
Reward-related eating	RED-X5 RED-9	Vainik, Eun Han, Epel, Janet Tomiyama, Dagher and Mason ⁵⁵	Î	Î
	RED-13	Total	1↑	1 ↑
Total scores	TFEQ	Bellisle and Dalix ¹²⁸	NS	NS
	DEBQ	Bellisle and Dalix ¹²⁸	NS	NS
	TFEQ	Nolan-Poupart, Veldhuizen, Geha and Small ¹¹⁰	NS	
		Total	3 NS	2 NS

Note: blank = outcome was measured but the data was not available as authors were contacted but did not reply, NS = non-significant, $NS^* = significant$ association with body fat or body weight, $\uparrow = significance$ positive association, $\downarrow = significant$ negative association,

^a= reverse coded. If two results are presented for one outcome (e.g., NS and \uparrow), the effect differed depending on the group or condition tested.

measure restraint (p < 0.001, Table S3). For the TFEQ, RRS and EDE-Q, restraint was negatively correlated with El. Whereas, for the RS and DEBQ, restraint was positively correlated with El. A randomeffects meta-regression was undertaken to test for differences in effect sizes between questionnaires used to measure restraint and El. The meta-regression was significant (p < 0.05); however, TFEQ was the only variable that was significantly associated with El (p < 0.01). Further subgroup analyses revealed that the effect of restraint on El was not significantly influenced by the number of test meals (one vs. multiple test meals, p = 0.103) or BMI group (healthy, overweight, obese, p = 0.553). Lastly, the association between restraint and El was influenced by a preload. Consuming a preload was associated with increased El (p = 0.040, see Table S4).

Meta-analysis of 32 studies (n = 1869) showed that restraint was positively correlated with BMI, and this effect was moderate (r = 0.20[0.12, 0.28], p < 0.001, Figure 4). Heterogeneity was moderate ($l^2 = 61.2\%$, Q = 79.86, p < 0.001). Four outliers were identified.^{58,79,89,100} Without these studies included, there was no change in the overall effect size (r = 0.20 [0.13, 0.26], p < 0.001). However, heterogeneity decreased ($l^2 = 34.8\%$, Q = 41.38, p = 0.038). Using the leave-one-out method, no studies were identified as influential. The funnel plot (Figure S4) suggested little evidence of publication bias, no missing studies were identified, and Egger's test was nonsignificant (p = 0.753). The effect of restraint on BMI did not depend on the type of questionnaire used to measure restraint (p = 0.103).

3.3.5 | Control of eating

Dalton, Finlayson, Blundell and Hill⁴⁷ found that craving control and positive mood were negatively associated with El (r = -0.20, p < 0.05, r = -0.21, p < 0.05) and BMI (r = -0.31, p < 0.001, r = -0.23, p < 0.01) respectively, whereas craving for sweet foods

was positively associated with EI (r = 0.40, p < 0.001) and BMI (r = 0.23, p < 0.01).

3.3.6 | Hedonic hunger

A meta-analysis of 4 studies (n = 183) found no significant correlation between hedonic hunger and El (r = 0.13 [-0.08, 0.33], p = 0.147). Additionally, a meta-analysis of 2 studies (n = 92) also found a nonsignificant association between hedonic hunger and BMI (r = 0.05[-1.00, 1.00], p = 0.868).

3.3.7 | Food craving

Martin et al.¹¹¹ reported that scores on the FCI were associated with increased total EI (r = 0.22, p < 0.05); however, there was no data for the effect of FCI on BMI.

3.3.8 | Susceptibility to hunger

A meta-analysis of 6 studies (n = 362) found susceptibility to hunger was positively associated with El, and this effect was moderate-to-large (r = 0.27 [0.19, 0.35], p < 0.001, see Figure S5). Heterogeneity was low ($l^2 = 0.0\%$, Q = 2.05, p = 0.843). No outliers or influential cases were identified. Three missing studies were identified to the left (adjusted r = 0.22 [0.13, 0.31], p < 0.001). A meta-analysis of 3 studies (n = 245) found a moderate positive correlation between susceptibility to hunger and BMI (r = 0.20 [0.11, 0.28], p = 0.011; Figure S6). Heterogeneity was small ($l^2 = 0.0\%$, Q = 0.20, p = 0.903). No outliers or influential cases were identified. Two missing studies were identified to the left (adjusted r = 0.17 [0.11, 0.23], p = 0.002).

Author	g	SE	Questionnaire	Correlation	COR	95% - Cl	Weight
Hofmann and Friese (2008)	-0.51	0.1961	TFEQ		-0.47	[-0.71; -0.13]	1.4%
Peluso (2017)	-0.38	0.2182	TFEQ		-0.36	[-0.67; 0.05]	1.2%
Zambrowicz et al. (2019)	-0.33	0.1782	TFEQ		-0.32	[-0.59; 0.02]	1.6%
Hopkins et al. (2021)	-0.32	0.1336	DEBQ		-0.31	[-0.52; -0.06]	2.3%
Schoch and Raynor (2012)	-0.31	0.1690	TFEQ		-0.30	[-0.57; 0.02]	1.7%
Rideout et al. (2004)	-0.31	0.1302	TFEQ			[-0.51; -0.05]	2.3%
Stinson et al. (2018)	-0.30	0.1125	TFEQ		-0.29	[-0.48; -0.08]	2.7%
Yeomans and Coughlan (2009)	-0.28	0.1857	TFEQ		-0.27	[-0.57; 0.08]	1.5%
Haynes et al. (2003)	-0.27	0.1140	TFEQ		-0.27	[-0.46; -0.05]	2.6%
Ruddock et al. (2017)	-0.26	0.1325	TFEQ			[-0.48; -0.01]	2.3%
Fedoroff et al. (1997)	-0.24	0.2182	RRS		-0.23	[-0.58; 0.19]	1.2%
Zambrowicz et al. (2019)	-0.24	0.1782	EDE		-0.23	[-0.53; 0.11]	1.6%
Ard et al. (2006)	-0.23	0.0825	TFEQ		-0.23	[-0.38; -0.07]	3.4%
Martin et al. (2005)	-0.23	0.2085	TFEQ			[-0.56; 0.18]	1.3%
Schotte et al. (1990)	-0.21	0.1925	RRS		-0.21	[-0.53; 0.17]	1.5%
Oliver et al. (2000)	-0.19	0.1796	DEBQ			[-0.50; 0.16]	1.6%
Stice et al. (2004) study 2	-0.18	0.1943	EDE		-0.18	[-0.51; 0.20]	1.4%
Visona and George (2002)	-0.18	0.1741	TFEQ		-0.17	[-0.48; 0.16]	1.7%
de Witt Huberts et al. (2013) study 2	-0.16	0.1581	RS		-0.16	[-0.44; 0.15]	1.9%
Bryant et al. (2012)	-0.15	0.1348	TFEQ			[-0.39; 0.11]	2.2%
Stice et al. (2004) study 1	-0.14	0.2338	DEBQ		-0.14	[-0.54; 0.31]	1.1%
Stice et al. (2010)	-0.14	0.1043	TFEQ		-0.14	[-0.33; 0.06]	2.9%
Anderson et al. (2016)	-0.13	0.0905	TFEQ		-0.13	[-0.30; 0.05]	3.2%
Herman et al. (1979)	-0.13	0.1644	RRS		-0.13	[-0.42; 0.19]	1.8%
Sim et al. (2018)	-0.11	0.2357	DEBQ		-0.11	[-0.52; 0.33]	1.1%
Rolls et al. (1997)	-0.09	0.1250	TFEQ		-0.09	[-0.33; 0.15]	2.4%
de Witt Huberts et al. (2013) study 3	-0.09	0.1601	RS		-0.09	[-0.38; 0.22]	1.8%
Herhaus and Petrowski (2021)	-0.05	0.1459	DEBQ		-0.05	[-0.32; 0.23]	2.1%
Wallis and Hetherington (2004)	-0.05	0.1690	DEBQ		-0.05	[-0.36; 0.28]	1.7%
Robinson and Haynes (2021)	-0.03	0.0941	DEBQ			[-0.21; 0.16]	3.1%
Stice et al. (2004) study 1	-0.02	0.2338	RS		-0.02	[-0.44; 0.41]	1.1%
Jansen et al. (1988)		0.1644	RS			[-0.32; 0.30]	1.8%
Stice et al. (2004) study 1		0.2338	TFEQ			[-0.44; 0.42]	1.1%
de Witt Huberts et al. (2013) study 1		0.1361	RS			[-0.26; 0.26]	2.2%
Bellisle et al. (2009)		0.1644	TFEQ			[-0.31; 0.31]	1.8%
van Strien and Ouwens (2003)		0.1890	DEBQ			[-0.35; 0.36]	1.5%
Stice et al. (2004) study 2		0.1943	TFEQ			[-0.35; 0.38]	1.4%
Lattimore and Maxwell (2004)		0.1925	RS			[-0.32; 0.40]	1.5%
Westenhoefer et al. (1994)		0.1245	TFEQ			[-0.18; 0.30]	2.4%
Higgs et al. (2008)		0.1195	DEBQ			[-0.17; 0.29]	2.5%
Ouwens et al. (2003)		0.1225	TFEQ			[-0.17; 0.30]	2.5%
Herman and Mack (1975)		0.1361	RS			[-0.18; 0.33]	2.2%
Myhre et al. (2014)		0.1857	RRS			[-0.27; 0.42]	1.5%
Wardle and Beales (1987)		0.1459	DEBQ			[-0.20; 0.35]	2.1%
Ouwens et al. (2003)		0.1225	RS			[-0.15; 0.32]	2.5%
Ouwens et al. (2003)		0.1225	DEBQ			[-0.13; 0.34]	2.5%
McNeil et al. (2017)		0.1387	TFEQ			[-0.14; 0.38]	2.2%
Finlayson et al. (2012)		0.1925	DEBQ			[-0.21; 0.50]	1.5%
Jansen (1996) study 2		0.1601	DEBQ		0.29	[-0.01; 0.55]	1.8%
Coelho et al. (2009)		0.1443	RS		0.32	[0.05; 0.55]	2.1%
Jansen (1996) study 1		0.1925	DEBQ		0.33	[-0.03; 0.62]	1.5%
Guerrieri et al. (2009)	0.54	0.1525	RS		0.49	[0.23; 0.68]	2.0%
Random effects model				÷	-0.07	[-0.12; -0.01]	100.0%
Prediction interval						[-0.32; 0.19]	
Heterogeneity: / ² = 42%, <i>p</i> < 0.01							
				06 04 02 0 02 04 06			

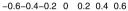


FIGURE 3 Forest plot of the effect of restraint on El

3.3.9 External eating

A meta-analysis of 6 studies (n = 583) found that external eating was positively correlated with EI, and this effect was moderate (r = 0.17[0.07, 0.27], p = 0.007, see Figure S7). Heterogeneity was low $(I^2 = 0.0\%, Q = 4.46, p = 0.485)$. Two influential studies were identified.115,116 With these studies removed, the effect size slightly increased (r = 0.18 [0.01, 0.34], p = 0.044). Heterogeneity was low $(l^2 = 0.0\%, Q = 2.21, p = 0.531)$. One missing study was identified to the left (adjusted r = 0.16 [0.06, 0.26], p = 0.008). A meta-analysis of 4 studies (n = 269) found no significant correlation between external eating and BMI (r = -0.08 [-0.28, 0.12], p = 0.285).

3.3.10 Emotional eating

A meta-analysis of 10 studies (n = 608) found a small-to-moderate, positive correlation between emotional eating and EI (r = 0.12[0.00, 0.24], p = 0.042, see Figure S8). Heterogeneity was moderate ($l^2 = 32.1\%$, Q = 13.26, p = 0.151). No influential studies were identified. The funnel plot did not suggest any publication bias (Figure S9), one missing study was identified to the left (adjusted r = 0.11 [-0.02, 0.23], p = 0.088) and the Egger's regression test was non-significant (p = 0.800). The effect of emotional eating on El was not influenced by the number of test meals (p = 0.342). A meta-analysis of 10 studies (n = 737) found that emotional eating

Author	g	SE	Correlation	COR	95%–Cl	Weight
Visona and George (2002)	-0.42	0.1741			[-0.64; -0.08]	2.6%
Rolls et al. (1997)	-0.15	0.1250		-0.15	[-0.38; 0.09]	3.5%
Hopkins et al. (2021)	-0.13	0.1336		-0.13	[-0.37; 0.13]	3.3%
Myhre et al. (2014)	-0.13	0.1857			[-0.45; 0.23]	2.5%
de Witt Huberts et al. (2013) study 3	0.02	0.1601			[-0.29; 0.32]	2.9%
Yeomans and Coughlan (2009)	0.02	0.1857		0.02	[-0.33; 0.37]	2.5%
Herhaus and Petrowski (2021)	0.04	0.1459		0.04	[-0.24; 0.31]	3.1%
Smith et al. (1998)	0.05	0.0909		0.05	[-0.13; 0.22]	4.2%
Yeomans and Coughlan (2004)	0.07	0.1644		0.07	[-0.25; 0.37]	2.8%
Wallis and Hertherington (2004)	0.08	0.1690	•	0.08	[-0.24; 0.39]	2.7%
Haynes et al. (2003)	0.09	0.1644		0.09	[-0.23; 0.39]	2.8%
Robinson and Haynes (2021)	0.10	0.0941		0.09	[-0.09; 0.27]	4.1%
Stinson et al. (2018)	0.10	0.1125		0.10	[-0.12; 0.31]	3.7%
Jansen (1996) study 1	0.17	0.1925		0.17	[-0.21; 0.50]	2.4%
Wardle and Beales (1987)	0.18	0.1459		0.18	[-0.10; 0.44]	3.1%
Westenhoefer et al. (1994)	0.18	0.0877	— ,	0.18	[0.01; 0.34]	4.2%
Bellisle et al. (2009)	0.22	0.1644		0.22	[-0.10; 0.49]	2.8%
Rideout et al. (2004)	0.22	0.1302		0.22	[-0.03; 0.44]	3.4%
Higgs et al. (2008)	0.23	0.1195		0.23	[0.00; 0.44]	3.6%
Finlayson et al. (2012)	0.25	0.1925		0.24	[-0.13; 0.55]	2.4%
Martin et al. (2005)	0.26	0.1741		0.25	[-0.08; 0.54]	2.6%
Dweck et al. (2014)	0.32	0.1280		0.31	[0.07; 0.52]	3.4%
Guerrieri et al. (2009)	0.34	0.1525		0.33	[0.05; 0.57]	3.0%
Coelho et al. (2009)	0.36	0.0941	· · · ·	0.35	[0.18; 0.50]	4.1%
de Witt Huberts et al. (2013) study 2	0.38	0.1581		0.36	[0.07; 0.60]	2.9%
Schotte et al. (1990)	0.38	0.1325	· · · ·	0.36	[0.12; 0.56]	3.4%
Schoch and Raynor (2012)	0.43	0.1690		0.40	[0.09; 0.64]	2.7%
Wallis and Hertherington (2009)	0.44	0.2085		0.42	[0.04; 0.69]	2.2%
Anderson et al. (2016)	0.47	0.0905		0.44	[0.29; 0.57]	4.2%
de Witt Huberts et al. (2013) study 1	0.54	0.1361		0.49	[0.26; 0.67]	3.3%
Jansen et al. (1988)	0.56	0.1644		0.51	[0.24; 0.71]	2.8%
Jansen (1996) study 2	0.67	0.1601		0.59	[0.35; 0.76]	2.9%
Random effects model			\diamond	0.20	[0.12; 0.28]	100.0%
Prediction interval					[-0.16; 0.51]	
Heterogeneity: $I^2 = 61\%$, $p < 0.01$					- · ·	
			-0.6 -0.2 0 0.2 0.4 0.6			

FIGURE 4 Forest plot of the effect of restraint on BMI

was positively correlated with BMI (r = 0.19 [0.13, 0.25] p < 0.001). This effect was moderate (Figure S10). Heterogeneity was low ($l^2 = 0.0\%$, Q = 5.17, p = 0.819). No influential cases were identified. The funnel plot did not suggest any publication bias (Figure S11), No missing studies were suggested, and the Egger's test was non-significant (p = 0.860).

3.3.11 | Disinhibition

A meta-analysis of 14 studies (n = 1,172) found a moderate positive correlation between disinhibition and El (r = 0.19 [0.14, 0.24], p < 0.001, see Figure S12). Heterogeneity was low ($l^2 = 0.0\%$, Q = 9.74, p = 0.715). Influence diagnostics identified one influential case.¹¹² With this study removed, the overall effect size increased (r = 0.21 [0.14, 0.27], p < 0.001). Heterogeneity was low ($l^2 = 0.0\%$, Q = 8.88, p = 0.713). The funnel plot showed little evidence of publication bias (Figure S13), one missing study was suggested to the right (adjusted r = 0.20 [0.14, 0.25], p < 0.001), and Egger's test was non-significant (p = 0.973). The effect of disinhibition on El was not influenced by the number of test meals (p = 0.341). Meta-analysis of

8 studies (n = 618) showed a moderate-to-large, positive correlation between disinhibition and BMI (r = 0.28 [0.19, 0.38], p < 0.001, Figure S14). Heterogeneity was low ($l^2 = 13.8\%$, Q = 8.12, p = 0.322). One influential case was identified.⁹⁵ Without this study included, there was a small decrease in the overall effect size (r = 0.25 [0.16, 0.33], p < 0.001). Heterogeneity was low ($l^2 = 0.0\%$, Q = 3.68, p = 0.720). No missing studies were suggested.

3.3.12 | Binge eating

A meta-analysis of 7 studies (n = 249) found a moderate, positive correlation between binge eating and El (r = 0.26 [0.08, 0.43], p < 0.001, see Figure S15). Heterogeneity was low ($l^2 = 0.0\%$, Q = 3.68, p = 0.720). Two influential studies were identified.^{98,123} With these studies removed, the overall effect size increased slightly (r = 0.28 [0.11, 0.43], p = 0.010). Heterogeneity was low ($l^2 = 0.0\%$, Q = 2.02, p = 0.731). Two missing studies were suggested to the left (adjusted r = 0.17 [-0.04, 0.37], p = 0.098). A meta-analysis of 7 studies (n = 277) found a moderate positive correlation between binge eating and BMI (r = 0.21 [0.07, 0.34],

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p = 0.011, see Figure S16). Heterogeneity was low ($l^2 = 0.0\%$, Q = 5.30, p = 0.506). No influential studies were identified. One missing study was identified to the right (adjusted r = 0.23 [0.09, 0.36], p = 0.006).

3.3.13 | Disordered eating

A meta-analysis of 2 studies (n = 152) was conducted to test the correlation between eating disorder symptomology and El. The analysis was non-significant (r = -0.17 [-0.98, 0.96], p = 0.499).

Only one effect size was available to test the effect of eating disorders on BMI; therefore, a meta-analysis could not be conducted. However, Anderson, Reilly, Schaumberg, Dmochowski and Anderson⁵⁸ did report that increased scores on the EDDS were associated with increased BMI (r = 0.40, p < 0.05). The YFAS 2.0 was not significantly associated with EI or BMI.¹²⁶

3.3.14 | Other EBT

Eating self-efficacy was negatively associated with EI (r = -0.26, p < 0.05).¹²⁷ Three measures of reward-related eating were positively correlated with EI (RED-X5: r = 0.31, p < 0.05, RED-9: r = 0.35, p < 0.05, RED-13: r = 0.32, p < 0.05) and BMI (RED-X5: r = 0.18, p < 0.05, RED-9: 0.17, p < 0.06, RED-13: r = 0.18, p < 0.05).

3.3.15 | Summary of meta-analyses findings

Figure 5 displays the overall effect sizes for each EBT on EI and BMI. A positive effect size indicates that the EBT is positively correlated with EI or BMI. A negative effect size indicates that the EBT is negatively correlated with EI or BMI. Intuitive eating and eating disorder symptomology are not displayed in Figure 5 due to the small number of studies included in these meta-analyses and the large variability in their correlations with EI and BMI. Figure 6 displays the effect sizes for the subgroup analysis testing the correlation between restraint and EI influenced by the questionnaire used to measure restraint. Error bars in both figures reflect standard error.

4 | DISCUSSION

The aims of this systematic review and meta-analyses were to (1) identify which measures are used to capture EBT, (2) assess how well these measures associate with short term EI and (3), BMI, (4) and evaluate whether some EBT and questionnaires used to measure EBT are better at predicting EI or BMI than others. Sixteen EBT were identified. However, the majority of EBT included were restraint, disinhibition, emotional eating, external eating, and binge eating. Considering the >45 years of research on human eating behavior, this systematic review demonstrates that relatively few studies on EBT have been conducted in relation to indices of objectively measured short-term EI as well as long-term energy balance status (BMI). For most of the EBT

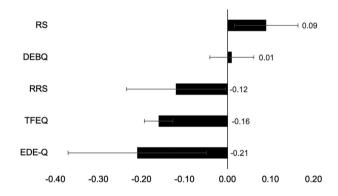


FIGURE 6 Bar graph of the subgroup analysis results, testing the correlation between restraint and El influenced by the questionnaire used to measure restraint

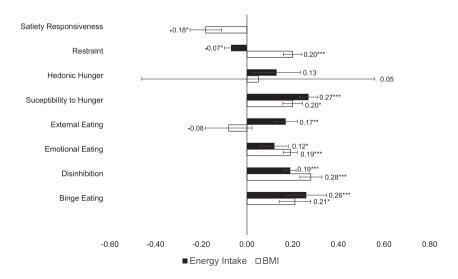


FIGURE 5 Summary of meta-analyses findings. *Note*: black line = Energy Intake, white line with black border = BMI, error bars indicate standard error, *p < 0.05, **p < 0.01, ***p < 0.001.

identified (aside from restraint), this research has been accomplished in only a small number of studies. Additionally, the majority of traits were measured using a single questionnaire (see Table 1), which were often older measures such as TFEQ or RS. This means that there are additional, typically newer questionnaires with no research that met the inclusion criteria for this systematic review, for example, the Positive and Negative Emotional Eating Scale.¹²⁹ Future research should assess the validity of these traits in relation to EI and BMI.

Overall, the meta-analyses demonstrated that EBT do significantly predict both EI and BMI. Effect sizes were generally moderate, with some analyses reaching larger effects. Susceptibility to hunger and binge eating were the strongest predictors of EI, whereas disinhibition was the strongest predictor of BMI. Although intuitive eating was moderately correlated with BMI, this meta-analysis was based on a very small number of studies. If there are not at least two adequately powered studies per meta-analysis, there may not be enough information to contribute an accurate conclusion of evidence about that EBT.¹³⁰ Therefore, drawing conclusions about intuitive eating and satiety responsiveness requires caution. Below, the main outcomes of the systematic review are discussed.

4.1 | Restraint

Restraint as a concept relates to a cognitive intention to restrict food intake, which is not necessarily reflected in actual reduction in El or adherence to a weight reducing diet.¹⁰³ This meta-analysis found a positive association between restraint and BMI, which is consistent with previous cross-sectional studies.^{131,132} However, negative findings between restraint and BMI have also emerged in the literature.¹³³ The finding of this systematic review could suggest that although restrained individuals attempt to restrict their food intake, increased restraint puts them at a higher risk of overeating (disinhibition), leading to a positive energy balance and consequently an increased BMI. However, the results of this systematic review do not agree with this prediction. Increased restraint was associated with decreased shortterm EI. This finding is more consistent with the interpretation that people with a higher BMI invoke strategies of dietary restraint to manage their weight. Approximately 40% of the adult population report at least one weight management attempt in the preceding 12 months.¹³⁴ It is also evident from the weight management literature that the majority of weight management attempts have limited success and are subject to weight regain.¹³⁵

Previous literature examining associations between restraint and EI have also been inconsistent, which is demonstrated in this systematic review as both positive and negative associations were found between restraint and EI. Research has highlighted that the different measures used to assess restraint could be influencing the different outcomes (research question 4). Heatherton, Herman, Polivy, King and McGree¹³⁶ suggest that the TFEQ may be better able to identify individuals who are successful in restricting their food intake, whereas the RS is better able to identify individuals who are unsuccessful in dieting. Indeed, the subgroup analysis found a significant effect of OBESITY -WILEY-

questionnaire type, such that the TFEQ was associated with decreased EI, whereas the RS was associated with increased EI (see Appendix). The EDE-Q and TFEQ had the largest effect size therefore suggesting these measures may be better at predicting El. This analysis suggests that each questionnaire may not be measuring exactly the same construct and generates questions about which scale is best at measuring restraint. The RS, for example, could be contaminated by other constructs such as disinhibition, as items on this scale refer to overeating, for example, "Do you have feelings of guilt after overeating?" This may explain why the RS was positively associated with El.¹⁰³ Moreover, there is also a potential need to re-evaluate what is meant by the construct of restraint. Is restraint a measure of concern controlling intake and its consequences, or could it be a measure of motivation or success in restraining intake like the TFEQ appears to measure or perhaps a combination of these considerations? There is a need to clearly define and achieve consensus about what restraint is and what it is not

A prediction of restraint theory is that the likelihood of a binge/ overeating episode should be increased after consuming a preload only in high restraint individuals. This is because dieters who have just broken their diet are predicted to subsequently overeat.^{103,137} In the current analysis, the correlation between restraint and EI was influenced by a preload such that the use of a preload was associated with increased EI, whereas no preload was associated with decreased EI (see Supporting Information). Nevertheless, Ouwens, van Strien and van der Staak⁸⁶ reported no significant effect of restraint on El influenced by a preload. The researchers highlight that not all dieters will show this disinhibition effect; but a particular subgroup might. Restrained eaters can therefore be split into two subpopulations: successful dieters and unsuccessful dieters. Successful dieters are characterized by high restraint and low levels of tendency toward overeating. Unsuccessful dieters are characterized by high restraint and are prone to overeating, meaning they are more likely to show a disinhibition effect after a preload. Westenhoefer, Broeckmann, Münch and Pudel¹⁰⁴ provided evidence for this idea and found that overeating only occurred in participants who displayed high scores on both restraint and disinhibition, as measured by the TFEQ.

More recent research such as Bryant, Caudwell, Blundell, Hopkins and King⁶⁶ and Chambers and Yeomans⁶⁷ have tested this effect by splitting participants into four groups based on their restraint (high vs. low; HR vs. LR) and disinhibition (high vs. low; HD vs. LD) scores using the TFEQ (HD/HR, HD/LR, LD/HR, LD/LR). A limitation of the current systematic review is that effect sizes were pooled so that high restraint included high and low disinhibition and low restraint included both high and low disinhibition. By not keeping the four groups separate, the analysis may have been insensitive to the effects of differing disinhibition levels. Consequently, future research could conduct a multi-level meta-analysis of restraint on EI, which can account for the influence of other variables such as disinhibition and emotional eating. Based on these findings, one must consider that restraint itself may not be a homogenous construct. Westenhoefer, Broeckmann, Münch and Pudel¹⁰⁴ proposed that restraint can be split into rigid and flexible control, with rigid control

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relating to an all-or-nothing approach. In contrast, flexible control reflects a less stringent approach to restriction of intake. These subscales could reflect successful and unsuccessful dieters and may better account for the differing effects of restraint on El. Unfortunately, only two studies in this review^{66,104} split restraint into flexible and rigid control and therefore testing the validity of this concept is currently not possible. Overall, this systematic raises questions about the current definition of restraint and whether it should be altered. If restraint is associated with increased BMI, then attempting to restrain intake as a dietary approach to weight loss may not be effective and other approaches should be utilized.

4.2 | Emotional eating

Another interesting finding of this systematic review was that emotional eating was positively correlated with short term EI and BMI, although there were two studies in the meta-analysis that found a negative correlation between emotional eating and El.^{71,101} The small-to-moderate effect size could have been influenced by the inclusion of only control conditions in this systematic review. Conditions that attempted to influence the emotional state of participants were excluded to reduce potential contaminates of the specific emotional eating trait effect. However, studies have shown that emotional eaters increased their food intake after a negative mood induction, compared with a neutral or positive mood induction.^{119,138} A limitation of this systematic review was that only one study presented data for the positive and negative subscales of emotional eating,¹¹⁶ and therefore the influence of positive or negative emotional eating could not be assessed. Further research should look to test systematically whether emotion valence, as well as emotion induction conditions increase EI in high emotional eaters versus low emotional eaters.

4.3 | Disinhibition

The overall finding that disinhibition was positively associated with short-term EI and BMI supports previous research^{6,9} and suggests that associations between uncontrolled eating and food intake are more consistently measured by scales capturing disinhibition, potentially because disinhibition is a 'pure' measure of uncontrolled eating. These findings suggest that increased disinhibition is an indicator of risk for potential weight gain and could be targeted in weight loss interventions characterize participants at risk of weight regain. Disinhibition could influence weight regain¹³⁹ and research has found that participants who maintained weight loss were characterized by lower disinhibition scores.¹⁴⁰ Niemeier, Phelan, Fava and Wing¹⁴¹ found that higher internal disinhibition predicted less successful weight loss and studies have consistently shown that successful weight loss is associated with a decrease in disinhibition.¹⁴²⁻¹⁴⁴ If weight loss interventions can successfully target and reduce participants' uncontrolled eating behavior (disinhibition), this approach may improve weight outcomes.

Furthermore, binge eating and susceptibility to hunger were also consistent predictors of EI and BMI and may be useful constructs to target in weight loss interventions. Assessing whether other traits are associated with behavior change is another important direction for future research. For example, studies have found that higher baseline emotional eating was associated with more weight gain over time,¹⁴⁵ meaning emotional eating could also be targeted in weight loss interventions. Mindfulness-based interventions have also been used to help participants decrease their tendency to eat in response to negative emotions.¹⁴⁶ Future research should assess which traits are prospectively associated with weight change or weight maintenance, as well as investigating the long-term efficacy of interventions utilizing EBT. Ultimately, disinhibition is a robust EBT that should be used in weight loss interventions and further research should strive to find ways to reduce levels of disinhibition in those scoring high on this subscale.

4.4 | Methodological comments

This systematic review included only laboratory-based measures of El because there is substantial evidence that self-report measures are susceptible to misreporting.¹⁰ However, laboratory-based measures of EI are highly susceptible to experimental design and demand characteristics. For example, Long, Meyer, Leung and Wallis¹²⁵ highlight that eating in a laboratory is an unnaturalistic setting and could distract and impede a participant's focus on internal signals of hunger and satiety, which could influence their El. Another concern relates to how well one single eating episode can generalize to usual eating behavior.⁵⁹ Snacking once in a test meal (often involving unusual foods) may not be an accurate reflection on a participants normal EI. Fourteen studies did test EI over multiple test meals, which helps to reduce this limitation. However, there were still 68 studies that tested a single eating episode and therefore, multiple test meal methods are not commonplace in eating behavior research. There are also issues relating to the 'observer effect', whereby participants behavior is biased by the awareness that they are being observed and that they are participating in a food-related study.¹⁴⁷ Studies rarely attempt to disguise that a meal is being used to measure food consumption and this transparency may alter the way a participant eats.¹⁴ Laboratory measures of food intake may have limited generalizability to normal every day eating behavior. However, the current meta-analyses generalize over multiple test designs and test meals which helps to overcome the role of specific research designs.

Another limitation of many studies included in this systematic review is the potential for other variables measured to contaminate the El outcome. Although studies that included potential contaminates were excluded, there were still some tasks which may have influenced the outcomes. For example, Coelho, Polivy, Herman and Pliner,⁶⁸ included a control condition that required participants to complete an arithmetic problem-solving task and a word-recall task. The researchers found that the participants could not complete the

test within the time provided and could have exposed the participants to task failure or ego threat which may have affected the participant mood/emotions.

4.5 | Conceptual comments

An important consideration of EBT is uncertainty over the extent to which individuals are capable of accurately assessing their own eating behavior, for example, emotional eating. Evers, de Ridder and Adriaanse¹¹⁷ suggests a 'triple recall bias', whereby people are generally unable to perceive their own behavior, they underreport their EI and personality disturbances, and retrospective emotional ratings are known to be highly sensitive to recall bias. Consequently, questionnaires that measure emotional eating are asking participants to recall their negative or positive emotions, food intake and association between both, all three of which may be biased. It is therefore possible that in some populations, for example, the elderly and young children, recall methods may not yield accurate findings and as such, should not be used. However, recommendations for specific population where EBT assessments should not be measured is beyond the scope of this analysis. The triple recall bias can also be extended to other psychological EBT, which raises concerns over their construct validity and questions whether EBT measuring what they were designed to measure? Interestingly, new developments in real time digital tracking of energy balance behaviors, 148-152 digital ecological momentary assessments and mobile video recording¹⁵³ could be a new avenue for more ecological assessments of El to capture EBT.

Most studies test the validity of EBT against very short-term EI, often over one test day. In order to relate EBT to long-term outcomes, EI and BMI need to be measured longitudinally. This also holds true for studies that aim to assess how EBT change in weight management interventions. In the short term, EBT are not expected to change because they are long-term processes. Therefore, to examine how EBT are associated with behavior change, they also need to be measured over a longer period of time.

5 | CONCLUSIONS

The outcome of this review has demonstrated that many EBT are associated with short-term EI and BMI, with disinhibition, susceptibility to hunger and binge eating having the largest effect sizes for EI and BMI. These findings highlight the potential use of these EBT as phenotypic markers of susceptibility to overconsume, develop obesity, or to influence outcomes in weight management interventions. Dietary restraint is evidently a complex construct and further research with preload conditions and separate subscales are needed to fully understand its effect on EI. The effect of emotional eating may have been limited by not including mood or stress induction conditions. However, this review still indicates it plays a role in EI and BMI. Importantly, there are methodological and conceptual issues with EBT that need to be stressed when utilizing these traits in eating behavior research. This does not mean they should not be used but a greater focus on objective indices of what such traits should predict (food intake and El, BMI or weight change) might improve their use in explaining eating behavior and energy balance in the wider population.

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CONFLICTS OF INTEREST

Pr. James Stubbs consults for Slimming World UK through The University of Leeds. Slimming World was not involved in the analysis or interpretation of this review.

AUTHOR CONTRIBUTIONS

CD, JS, GF, and MH performed the literature search, study selection, data extraction, and quality assessment. CD performed the metaanalysis and drafted the manuscript. All authors critically revised the manuscript.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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