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Predictors of relapse in eating disorders: A meta-analysis

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ABSTRACT

Introduction: Eating disorders (EDs) have high rates of relapse. However, it is still not clear which factors are the strongest predictors of ED relapse, and the extent to which predictors of relapse may vary due to study and individual differences.
Objective: We conducted a meta-analysis to quantify and compare which factors predict relapse in EDs and evaluate various potential moderators of these relations (e.g., ED subtype, sample age, length of follow-up, timing of predictor assessment, relapse operationalization).
Methods: A total of 35 papers (effects = 315) were included. We used a multilevel random-effects model to estimate summary study-level effect sizes, and multilevel mixed-effects models to examine moderator effects.
Results: Higher level of care, having psychiatric comorbidity, and higher severity of ED psychopathology were associated with higher odds of relapse. Higher leptin, higher meal energy density/variety, higher motivation for change, higher body mass index/weight/body fat, better response to treatment, anorexia nervosa-restricting (vs. anorexia nervosa-binge purge) subtype diagnosis, and older age of ED onset were associated with lower odds of relapse. Several moderators were identified.
Discussion: A variety of variables can predict ED relapse. Furthermore, predictors of ED relapse vary among ED subtypes, sample ages, lengths of follow-up, timing of predictor assessments, and relapse operationalization. Future research should identify the mechanisms by which these variables may contribute to relapse.

1. Introduction

EDs are highly recurrent in nature, with high rates of relapse. On average, relapse rates when followed for up to 10 years have been approximately 40–50% for anorexia nervosa (AN; Carter et al., 2012; McCormick et al., 2009; Herzog et al., 1999; Keel et al., 2005), 40% for other specified feeding and eating disorder (OSFED; Grilo et al., 2007; McFarlane et al., 2008), and 30% for bulimia nervosa (BN; Herzog et al., 1999; Keel et al., 2005; Olmsted et al., 1994; Olmsted et al., 1996; Olmsted et al., 2015) and binge eating disorder (BED; Safer et al., 2002; Ricca et al., 2010).

1.1. Predictors of relapse in EDs

Despite high relapse rates of EDs, it is still not clear which factors are most strongly predictive of relapse. Identifying predictors of ED relapse is critical for the understanding of key factors that may precipitate relapse and to inform relapse prevention interventions. Thus far, researchers have identified several factors predictive of ED relapse, including patient demographics (e.g., Berends et al., 2016; Deter and Herzog, 1994; Kahn et al., 2020), ED characteristics (e.g., Castellini et al., 2011; Halmi et al., 2002), psychiatric comorbidity (Deter and Herzog, 1994; Ricca et al., 2010), and treatment processes (e.g., Berends et al., 2016; Richard et al., 2005). However, some findings have been mixed, and the relative contribution (i.e., the size of the effects) of these predictors is currently unclear.

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Demographic characteristics. Demographic variables have been studied as potential predictors of ED relapse. However, findings have been mixed for both age (Berends et al., 2016; Deter and Herzog, 1994; Olmsted et al., 1994) and gender (Kahn et al., 2020; Castellini et al., 2011).

ED characteristics. ED characteristics have been examined as potential predictors of ED relapse. Research has produced mixed findings regarding whether age of onset, duration of ED (Halmi et al., 2002; Richard et al., 2005; Safer et al., 2002), and diagnostic subtype of ED are predictive of relapse (e.g., Castellini et al., 2011; Herzog et al., 1999; Keel et al., 2005; Carter et al., 2012; Eddy et al., 2002).

Other ED characteristics have been found to be predictive of ED relapse. First, most studies have concluded that lower pre-and post-treatment BMI/weight/body fat (Mayer et al., 2007; Steinhausen et al., 2008; Castro-Fornieles et al., 2007; Schebendach et al., 2008) is predictive of higher likelihood of relapse in individuals with AN/BN. Second, more severe ED psychopathology has been found to predict a higher likelihood of relapse across EDs (McFarlane et al., 2008; Castro-Fornieles et al., 2007; Safer et al., 2002; Halmi et al., 2002; Castellini et al., 2011; Freeman et al., 1986; Keel et al., 2005; Olmsted et al., 1994, 2015; Steinhausen et al., 2008; Carter et al., 2004, 2012; Richard et al., 2005; Kuipers et al., 2017). However, a few studies have found that more severe ED psychopathology is associated with a lower likelihood of relapse (Olmsted et al., 2015; Richard et al., 2005). Third, post-treatment dietary intake (e.g., energy density, dietary variety, protein, calories, etc.) predicts relapse (Schebendach et al., 2008, 2012).

Psychiatric comorbidity (Deter and Herzog, 1994; Ricca et al., 2010) and worse overall psychosocial/global functioning (Keel et al., 2005) have been found to be predictive of relapse across EDs. Specific comorbidities associated with higher likelihood of relapse have included exposure to traumatic events (Rodríguez et al., 2005; Carter et al., 2012), obsessive and compulsive symptoms (Carter et al., 2004), depressive symptoms (Castro-Fornieles et al., 2007; Ricca et al., 2010), a history of suicide attempts (Carter et al., 2004), and post-partum depression (Morgan et al., 1999). However, other research has not found a significant relationship between psychiatric comorbidity (e.g., depression) and likelihood of relapse (Romano et al., 2002).

Treatment processes. Predictors of relapse across EDs have also included treatment process related variables, such as stronger level of care (e.g., inpatient vs. outpatient treatment; Berends et al., 2016), longer duration of treatment (Berends et al., 2016), and slower and/or poorer response to treatment (Olmsted et al., 1996, 2015; Richard et al., 2005; Halmi et al., 2002; McFarlane et al., 2008).

1.2. Previous reviews

Berends et al. (2018) conducted a systematic review which identified several predictors of relapse in individuals with AN, including: more severe ED symptoms, AN-binge purge (AN-BP) subtype, higher psychiatric comorbidity, lower self-esteem, lower motivation to recover, older age, longer duration of illness, and various treatment process variables (e.g., longer-duration of treatment, a higher level of care, additional medical treatment). However, Berends et al. (2018) only conducted a meta-analysis of the rates of relapse, and did not meta-analyze or compare the strength of each predictor's effect sizes, and it is thus currently unclear which of these factors are the strongest predictors of relapse in AN. Understanding the strongest predictors of relapse in AN is critical to maximally improve current AN relapse prevention interventions. Furthermore, to our knowledge, no study to date has synthesized predictors of relapse in BN, BED, and OFSED, which are some of the most common EDs. We therefore have limited guidelines on relapse prevention for BN, BED, and OFSED.

1.3. Possible moderators in predictors of relapse

The extent to which various predictors are related to likelihood of relapse may vary because of differences across sample types. Prior ED literature highlights that some of these differences may include: (a) ED subtype; (b) sample age; (c) length of follow-up assessment; (d) timing of predictor assessment; and (e) relapse conceptualization.

ED subtype. It remains uncertain whether certain predictors of relapse are only relevant to certain ED diagnoses. For example, whereas *older* age has emerged as a predictor of relapse in AN (Berends et al., 2016; Deter and Herzog, 1994), *younger* age has emerged as a predictor of relapse in BN (Olmsted et al., 1994). Similarly, whereas female gender has emerged a predictor of relapse in AN (Kahn et al., 2020), male gender has emerged as a predictor of relapse in BED (Castellini et al., 2011).

Sample age. It remains uncertain whether certain predictors of relapse are only relevant in younger vs. older individuals. For example, whereas many studies in adults have found that factors such as an AN-BP diagnosis (e.g., Carter et al., 2012) and longer duration of illness (Richard et al., 2005) predict a higher likelihood of relapse among individuals with AN, Castro-Fornieles et al. (2007) found that this was not the case in a younger sample of patients, where duration of illness was short. Identifying which predictors of relapse are most relevant to specific age groups will allow for the development of tailored relapse prevention strategies to the needs of distinct subpopulations.

Length of follow-up. It is possible that certain relapse predictors may be relevant to certain time-periods following recovery. For example, Richard et al. (2005) found that whereas high motivation predicts a higher likelihood of relapse in individuals with AN six months following remission, it predicts a lower likelihood of relapse two years following remission. Knowing the time-points in which certain factors may be most predictive of relapse would allow for optimal timing in relapse prevention delivery.

Timing of assessment. The timing of when the predictor is assessed may influence the strength with which it is related to likelihood of relapse. ED characteristics that are assessed immediately post-treatment may not robustly predict likelihood of relapse, because some ED treatment (e.g., inpatient, residential care) is conducted in a controlled environment. Therefore, the ability to abstain from engaging in ED behaviors during treatment may not predict the ability to do so outside of treatment. For example, some research suggests that whereas indicators of severity of ED pathology assessed before treatment predict likelihood of relapse, indicators of the severity of ED pathology assessed immediately after treatment do not (Olmsted et al., 2015).

Relapse operationalization. The operationalization of relapse may influence findings. Various disparate operationalization of relapse have been used in the literature. For example, operationalizations of relapse have included body mass index (BMI), percentage of ideal body weight, and/or psychiatric symptoms (Khalsa et al., 2017). In addition, several studies have operationalized relapse as re-entry into treatment (e.g., Steinhausen et al., 2008; Kahn et al., 2020). The variables included in relapse operationalizations, and the comprehensiveness of these relapse definitions, may influence findings.

1.4. The present study

Overall, although previous research has identified some vulnerability factors for relapse in EDs, findings have been mixed, and a quantitative synthesis of factors that predict ED relapse is needed. The current systematic review and meta-analysis sought to: (1) quantify and compare factors that predict relapse in EDs; and (2) determine how these associations vary by (a) ED subtype; (b) sample age; (c) the length of follow-up; (d) timing of predictor assessment; and (e) relapse operationalization.

2. Methods

2.1. Study selection

This meta-analysis followed the guidelines for Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA; Moher et al., 2009). Searches were performed in Pubmed and PsycInfo through December 2021. Search terms were: "*anorexia nervosa*" OR "*bulimia nervosa*" OR "*binge eating disorder*" OR "*eating disorder*" AND "*relaps **. To maximize finding eligible articles, the citations and references of included articles and related reviews were also investigated.

The initial search yielded 722 abstracts. Many abstracts were duplicates, and 618 unique abstracts were screened. One hundred twenty-three full texts were assessed for eligibility, and 35 articles were ultimately included in the review (Table 1; Fig. 1).

2.2. Selection criteria

Inclusion criteria. Criteria for studies to be included in the review were (1) Presence of an ED (e.g., AN, BN, BED, OFSED): for all participants at some point during or before the start of the study, verified by either clinician diagnosis or a self-report scale; (2) At some point during the study, participants needed to be in (partial) remission or recovery; (3) Relapse or recurrence was diagnosed *or* participants re-entered treatment at some point (see Table 1 for how the included studies defined relapse); (4) The study design was longitudinal and prospective; (5) The relapse predictor was assessed before relapse or recurrence of the ED; (6) There was sufficient information to calculate effect size; and (7) The study was empirical and quantitative. Our search was restricted to peer-reviewed articles written in English.

Primary and secondary screening. The first and last authors developed and discussed the inclusion criteria. Both the second and fourth author reviewed the research reports to determine their eligibility. There was a 93% inter-rater agreement for study inclusion (kappa = .78). All disagreements were discussed among authors until consensus was reached.

2.3. Data extraction

We coded the following variables: authors, title, publication year, sample size, ED type, age of study sample, percentage of women in sample, percentage of White individuals in sample, mean BMI of the study sample, length of follow-up, relapse operationalization, percentage of participants who experienced relapse, description of predictor, timing of predictor assessment, the type of effect size information provided (e.g., frequency tables, etc.), and the magnitude of the effect size. Both the second and third author coded the studies, and all disagreements were discussed among the first three authors until consensus was reached.

Relapse predictors were classified into one of the following categories: (1) age; (2) age of ED onset; (3) duration of illness (4) AN-R (vs. AN-BP); subtype; (5) BMI/weight/body fat percentage; (6) severity of ED psychopathology, which was further subclassified into severity of (a) global ED psychopathology (e.g., global Eating Disorder Examination-Questionnaire scores), (b) over-exercise, (c) BN symptoms, (d) body dissatisfaction, (e) restraint, (f) binge eating and (g) other ED symptoms (e.g., any other ED symptoms that did not fit into the categories above, such as Eating Disorder Inventory maturity features, interoceptive awareness, perfectionism, ineffectiveness, interpersonal distrust, etc.); (7) meal energy density and variety (e.g., caloric intake relative to quantity of food consumed; calories consumed; dietary variety; consumption of non-caloric beverages, etc.); (8) psychiatric comorbidity, which was further subclassified into comorbid (a) depression, (b) anxiety, (c) personality disorder, and (d) general psychiatric comorbidity; (9) level of care (e.g., inpatient vs. outpatient, additional psychopharmacological treatment); (10) treatment duration; (11) response to treatment (e.g., weight rate increase, rapid response to meal plan, etc.); (12) a variety of potential ED risk factors (e.g., stress vulnerability, impulsiveness, family problems, etc.); (13) self-esteem; (14) motivation for change; (15) leptin levels; and (16) brain-derived neurotrophic factor (BDNF; See Table 2 for examples). Several predictors (e.g., type of ED diagnosis, various biomarkers) could not be classified into a category of analysis due to a low number of such predictors ($n < 4$).

2.4. Calculation of effect size estimates

The logarithmic odds ratio (log OR) effect sizes for all outcome measures with their estimated variances were calculated using reported statistics for each study. Log OR effects were back transformed to OR for easier interpretation. The odds for each predictor represent the increased or decreased likelihood of relapse.

2.5. Statistical analyses

Analyses were conducted using the R package *metafor* (Viechtbauer, 2010).

Dependence of data. Most (90.5%) studies examined multiple predictors of ED relapse. Including multiple effect sizes from a given study can pose risk of dependencies because within-study effects are nested within the same group of participants and are thus intercorrelated (Lipsey and Wilson, 2000). Therefore, we used multilevel modeling. Multilevel modeling is a method for modeling dependence among effect sizes that avoids violating the assumption of independent effect sizes (Hox et al., 2010) and is therefore useful when multiple effect sizes are nested within the same study designs and samples. Given the varied nature of the included predictors, we conducted separate models for each category of predictors to estimate the summary effects for each category of predictors.

Publication bias. We examined the likelihood of publication bias by visually inspecting the symmetry in a funnel plot. We also conducted Egger's regression tests (Egger et al., 1997). We also conducted trim-and-fill analyses for any category of predictors with a statistically significant Egger's regression test (Duval and Tweedie, 2000).

Weighted-mean effect size estimate. We used a random-effects model to calculate the overall weighted mean effect size, and the 95% confidence intervals around this summary effect.

Study heterogeneity. We used Cohran's Q homogeneity test, the I^2, and τ^2 statistics as indicators of heterogeneity.

Moderator analyses. We used a mixed-effects model to evaluate the extent to which the moderators affected between-study variability in the relationships between predictors and odds of relapse. ED subtype and the timing of predictor assessment were examined separately as categorical predictors of the relationship between the various predictors and likelihood of relapse. We also included sample age and length of study follow-up as continuous moderators.

3. Results

3.1. Descriptive information

A total of 35 articles and 36 independent samples met our inclusion criteria. Some articles included multiple samples, while other articles overlapped in their use of samples. From the 36 samples, a total of 359 effect sizes were coded, and 315 were classified into a category (with 44 unable to be classified due to a low number of similar types of predictors that could be grouped into one category). The included samples had between 1 and 48 effect sizes. The mean sample size was 72 ($SD = 55$). Samples were predominantly female [average percentage 98.1% ($SD = 3.9$)], White [average percentage 92.9% ($SD = 5.7$)], and the mean age of the samples was 26.16 years (Range = 14.4–49.20, $SD = 8.8$). In terms of diagnostic distribution, 18 samples included only individuals with AN, 6 only included individuals with BN, 3 only individuals with BED,

Table 1
Summary of included studies.

Authors	Year	ED	ED Diagnosis	Diagnostic criteria	Study Type	Setting	Relapse operationalization	N	Age Category	Relapse Rate	Follow-up Length	Significant Predictors of Relapse (+= higher likelihood of relapse - = lower likelihood of relapse)
Avnon et al.	2018	AN	Not explicitly specified	DSM IV & DSM 5	Longitudinal	Inpatient	Re-entry into in-patient treatment	44	Children & adolescents	34%	1 year	Weight gain lability during treatment(+) Percent from target weight(−)
Berends et al.	2016	AN	Interview	DSM IV	Longitudinal	Outpatient and inpatient	Full: BMI <18.5 for adults and SD BMI < −1 for adolescents; full recurrence of core DSM sx Partial: re-occurrence of one or more core diagnostic symptoms of AN	83	Adolescents & adults	Full: 11% Partial: 19%	18 months	Age 19+ (vs. < 18) (+) Inpatient + outpatient tx (vs. outpatient only)(+) Longer duration of treatment (+)*
Carter et al.	2004	AN	Interview	DSM IV	Longitudinal	Inpatient	BMI <17.5 for 3 consecutive months.	51	Mixed	35%	6–27 months	History of suicide attempts(+)* Specialized ED treatment(+)* More severe OCD at presentation(+)* Excessive exercise immediately after discharge (+)* Concern about shape and weight at discharge(+)*
Carter et al.	2012	AN	Interview	DSM IV	Longitudinal	Day and inpatient	BMI less than or equal to 17.5 for 3 consecutive months or at least one episode of BP behavior per week for 3 consecutive months during the 1-year follow-up period	100	Mixed	41%	1-year	BP subtype of AN(+) History of childhood physical abuse(+)* Higher pre-treatment eating concern(+)* Higher pre-treatment checking behavior(+)* Decrease in motivation during treatment(+)* Increase in shape concern during treatment(+)* Increase in weight concern during treatment(+)* Decrease in self-esteem during treatment(+)*
Castellini et al.	2011	AN, BN, & BED	Interview	DSM IV & DSM 5	Longitudinal	Outpatient	The return to a full syndromal ED or EDNOS criteria as per the DSM-IV or DSM V Criteria	793	Adults	AN: 26% BN: 18% BED: 11%	6 years	AN: Higher subjective binge eating frequency(+) BN: Higher subjective

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Table 1 (*continued*)

Authors	Year	ED	ED Diagnosis	Diagnostic criteria	Study Type	Setting	Relapse operationalization	N	Age Category	Relapse Rate	Follow-up Length	Significant Predictors of Relapse (+= higher likelihood of relapse - = lower likelihood of relapse)
												binge eating frequency(+) Absence of substance abuse(+) <u>BED:</u> Male gender(+) Higher subjective binge eating frequency(+)
Castro-Fornieles et al.	2007	AN	Not explicitly specified	DSM IV	Longitudinal	Inpatient	Readmission into treatment	40	Children & adolescents	38%	9 months	Higher BMI at admission(−) Higher motivation to change(−) Higher abnormal eating attitudes(+) Higher depressive symptoms(+)
Freeman et al.	1985	BN	Not explicitly specified		Longitudinal	Outpatient	Pre-eminence of body image dissatisfaction; no further information was provided	39	Adults	41%	6 months	Higher weight(+) Post-tx body image distortion(+)
Eddy et al.	2002	AN	Interview	DSM III & DSM IV	Longitudinal	Mixed	The return of full criteria symptomatology for at least 1 week	39	Adolescents & adults	40%	8–12 years	.
Grilo et al.	2007	BN (25%) & EDNOS (75%)	Interview	DSM IV	Longitudinal	Mixed	Variables were defined in terms of remission, not relapse: Remission from eating disorders (bulimia nervosa and EDNOS) was defined as 8 consecutive weeks with PSR ratings on the LIFE of less than 2 (for any type of eating disorder diagnosis)	74	Adults	BN: 47% EDNOS: 42%	5 years	.
Halmi et al.	2002	BN	Interview	DSM III	Longitudinal	Outpatient	Re-engagement with any quantity of binge eating and purging	48	.	44%	4 months	Shorter illness duration(+) Higher preoccupation and ritualization of eating(+) Lower motivation for change(+) Higher post-treatment restraint (+) Shorter abstinence from purge behavior (+)
Herzog et al.	1999	AN (36%) & BN (64%)	Interview	DSM III & DSM IV	Longitudinal	Mixed	Return of full criteria symptoms for at least 8 weeks	120	Adolescents & adults	AN: 40% BN: 35%	7.5 years	.
Kahn et al.	2020	AN	Interview	DSM 5	Longitudinal	Inpatient	Re-hospitalization	56	Children and Adolescents	38%	1 year	Female gender(+)
Keel et al.	2005	AN (34%) & BN (66%)	Interview	DSM III	Longitudinal	Mixed		125	Adolescents & adults	AN: 36% BN: 35%	8–10.5 years	<u>AN:</u> Misperception of

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Table 1 (*continued*)

Authors	Year	ED	ED Diagnosis	Diagnostic criteria	Study Type	Setting	Relapse operationalization	N	Age Category	Relapse Rate	Follow-up Length	Significant Predictors of Relapse (+= higher likelihood of relapse - = lower likelihood of relapse)
							Return to full syndromal criteria after a period of remission for either anorexia nervosa or bulimia nervosa (PSR 5 or 6)					body weight and shape (+)* Fear of gaining weight or becoming fat (+)* Worse psychosocial functioning (+)* Receiving individual psychotherapy(+)* BN: Worse psychosocial functioning (+)* Overconcern with weight and shape (+)* Worse global functioning(+)*
Kuipers et al.	2017	AN & BN	Interview	DSM IV	Longitudinal	Day	Fulfilling the criteria for an eating disorder diagnosis according to the structured clinical interview for DSM axis I disorders (SCID-I)	11	Adults	18%	18 months	More severe ED symptoms (+)
Mayer et al.	2007	AN	Interview	DSM IV	RCT	Inpatient	"Treatment failure" was categorized as "poor" on the Morgan-Russell classification	26	Adults	38%	8+ weeks	Higher percent body fat(−)
McCormick et al.	2009	AN	Not explicitly specified	DSM IV	Longitudinal	Inpatient	Readmission to a partial or inpatient unit for eating disorder-related treatment; and/or a drop in BMI below 17.5 kg/m2	15	Adults	47%	1 year	Higher health concern(−) MMPI negative treatment indicator improvement during hospitalization(−) Self-esteem improvement during hospitalization(−)
McFarlane et al.	2008	AN (28%), BN (31%), & EDNOS (41%)	Interview	DSM IV	Longitudinal	Day	Consistent with DSM-IV diagnostic criteria: more than 8 binge eating and/or purging episodes per month for three consecutive months, or BMI below 18.5 for three consecutive months,or eating less than 1000 calories per day for three consecutive months	58	.	AN: 50% BN: 28% EDNOS: 42%	2 years	Severe pre-treatment caloric restriction(+) Higher weight-related self-evaluation(+) Higher weight-related self-esteem (+) Higher residual symptoms at end of treatment(+) Early adherence to meal plan during treatment(−)
Mueller et al.	2017	BN	Interview	DSM IV	RCT	Unclear	At least 1 binge eating or purging episode	16	Adults	31%	18–42 months	Shorter time in remission (+) AMPT induced increases in

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Table 1 (*continued*)

Authors	Year	ED	ED Diagnosis	Diagnostic criteria	Study Type	Setting	Relapse operationalization	N	Age Category	Relapse Rate	Follow-up Length	Significant Predictors of Relapse (+= higher likelihood of relapse - = lower likelihood of relapse)
												depression(−) CBF reduction in the right hippocampus (−) CBF reduction in the right inferior parietal lobe(−)
Olmstead et al.	1994	BN	Interview	DSM III	Longitudinal	Day	Meeting DSM-III criteria for at least 2 weeks	48	Adults	31%	2 years	Younger age(+) Higher pre-treatment vomiting frequency (+) Higher post-treatment vomiting frequency(+) Worse pre-treatment BN symptoms(+) Higher amount below highest weight(+) Higher post-treatment interpersonal distrust (+)
Olmstead et al.	1996	BN	Self-report & Interview	DSM III	Longitudinal	Day	A change from full or partial remission to the full BN syndrome	41	Adults	30%	2 years	Rapid response to treatment(−)
Olmstead et al.	2015	BN (78%) & OSFED (22%)	Interview	DSM 5	Longitudinal	Day	Meeting DSM-IV-TR criteria	116	Mixed	28%	6 months	Pre-treatment binge eating frequency(+) Pre-treatment vomiting frequency (+) Pre-treatment body avoidance(−) Slow response to treatment(+)*
Ricca et al.	2010	BED	Interview	DSM IV	RCT	Outpatient	BED or s-BED diagnosis (as per the DSM IV)	36	Adults	33%	3 years	History of amphetamine derivatives consumption(+) Higher depression symptoms (+)
Richard et al.	2005	AN BN	Interview	DSM III	Longitudinal	Inpatient	Return to full symptomatic status as per DSM III-R	233 422	Adults	33% 37%	2.5 years 2.5 years	Lower desired weight (+) Longer duration of illness (+) Low severity of ED symptoms (+) Non-specialized treatment (+) Additional psychological

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Table 1 (*continued*)

Authors	Year	ED	ED Diagnosis	Diagnostic criteria	Study Type	Setting	Relapse operationalization	N	Age Category	Relapse Rate	Follow-up Length	Significant Predictors of Relapse (+= higher likelihood of relapse - = lower likelihood of relapse)
												treatment (+) Additional medical treatment (+) Partial (vs. full) remission status (+) Lower motivation for treatment(+) Additional inpatient treatment(+)
Rodriguez et al.	2005	AN (37%), BN (39%), & BED (24%)	Interview	DSM IV	Longitudinal	Outpatient	Return to baseline binging or vomiting frequency in purging sub-types, for at least 2 consecutive weeks, weight loss or a return to low weight baseline, or restrictive intake in AN	160	Mixed	34%	1 year	History of exposure to traumatic events (+)
Romano et al.	2002	BN	Self-report	DSM IV	RCT	Outpatient	Return to the baseline vomiting frequency that persisted for 2 consecutive weeks	150	Adults	33–51%	1 year	None
Safer et al.	2002	BED	Interview	DSM IV	Longitudinal & RCT	Outpatient	No definition provided	32	Adults	28%	6 months	Early onset binge eating (+) Post-treatment restraint (+) Post-treatment emotional eating(+) Post-treatment self-esteem(+)
Schedenbach et al.	2008	AN	Not explicitly specified	DSM IV	Longitudinal	Inpatient	Morgan-Russell criteria	47	Adults	30%	1 year	Dietary energy density(−) Dietary variety(−) Fat intake(−) Fat intake as % of kcals(−) Longer duration of illness(−) Post-treatment BMI (−) Protein intake as % of kcals(+) Noncaloric beverage consumption(+)
Schebendach et al.	2012	AN	Interview	DSM IV	Longitudinal	Inpatient	Morgan-Russell criteria	16	Adults	50%	.	Dietary energy density(−) Energy/calorie consumption(−) Noncaloric beverage consumption(+)
Steinhausen et al.	2008	AN	Interview	ICD-10	Longitudinal	Inpatient	Re-hospitalization	212	Children/ Adolescents	45%	8.3 years (average)	Pattern alcoholism (+) History of AN in family(+) Eating disorder in infancy(+)

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Table 1 (*continued*)

Authors	Year	ED	ED Diagnosis	Diagnostic criteria	Study Type	Setting	Relapse operationalization	N	Age Category	Relapse Rate	Follow-up Length	Significant Predictors of Relapse (+= higher likelihood of relapse - = lower likelihood of relapse)
												Periodic overactivity (+) Lower weight increase at first admission(+) Lower BMI at discharge(+)
Cooper et al.	2021	AN	Interview	DSM 5	Longitudinal	Partial & inpatient	No longer meeting weight restoration at follow-up	107	Adolescents and Adults	31%	6 months	Longer illness duration(+)
Kim et al.	2021	AN	Interview	DSM IV	Longitudinal	Inpatient	BMI less than 18.5 kg/m2 for the 8 weeks leading up to the 1-year follow-up	41	Adults	51%	12 months	Higher leptin levels after weight restoration (−) Higher body fat % after weight restoration (−)
Borsford et al.	2021	AN	Interview	DSM IV	Longitudinal	Inpatient	Scores on the SIAB-EX and Morgan–Russell Outcome Assessment Schedule measures.	114	Children & Adolescents	28–35%	1–2.5 years	None
Dardennes et al.	2021	AN	Interview	DSM IV	Longitudinal	Inpatient	Weight status	26	Adults	27%	2 months	Higher leptin levels at discharge (−)
Makino et al.	2020	AN & BN	Self-report	DSM 5	Longitudinal	Outpatient	No definition of relapse provided	24	Adults	50–67%	.	Younger age at remission(+) Younger age at pregnancy(+) ED relapse during pregnancy(+) ED relapse after delivery(+)
Solid et al.	2021	AN (69%), BN (23%), BED (3%), & Subthreshold ED (5%)	Interview	ICD-10	Longitudinal	Unclear	Onset of recurrent ED behavior, that is, at least once a week over a period of a month, closely corresponding to ICD-10 level including atypical AN and BN	122	Adults	25%	.	None

Notes: ED = Eating Disorder; N=Number of participants included in relapse analysis; F/U = follow-up; AN = anorexia nervosa; Tx: Treatment; OCD = Obsessive Compulsive Disorder; BP =Binge-purge; BN = bulimia nervosa; BED = Binge eating disorder; EDNOS = Eating Disorder Not Otherwise Specified; BMI = Body Mass Index; MMPI = Minnesota Multiphasic Personality Inventory; AMPT = Alpha-methyl-*p*-tyrosine; CBF =Cerebral blood flow; OSFED = Other Specified Feeding and Eating Disorder; * = not included in meta-analysis because there was not enough information to calculate effect size; DSM = Diagnostic and Statistical Manual of Mental Disorders; ICD= International Classification of Diseases; RCT = Randomized Controlled Trial; SIAB-EX = The structured interview for anorexic and bulimic disorders; EAT = Eating Attitudes Test; PSR= Psychiatric Status Rating Scale.

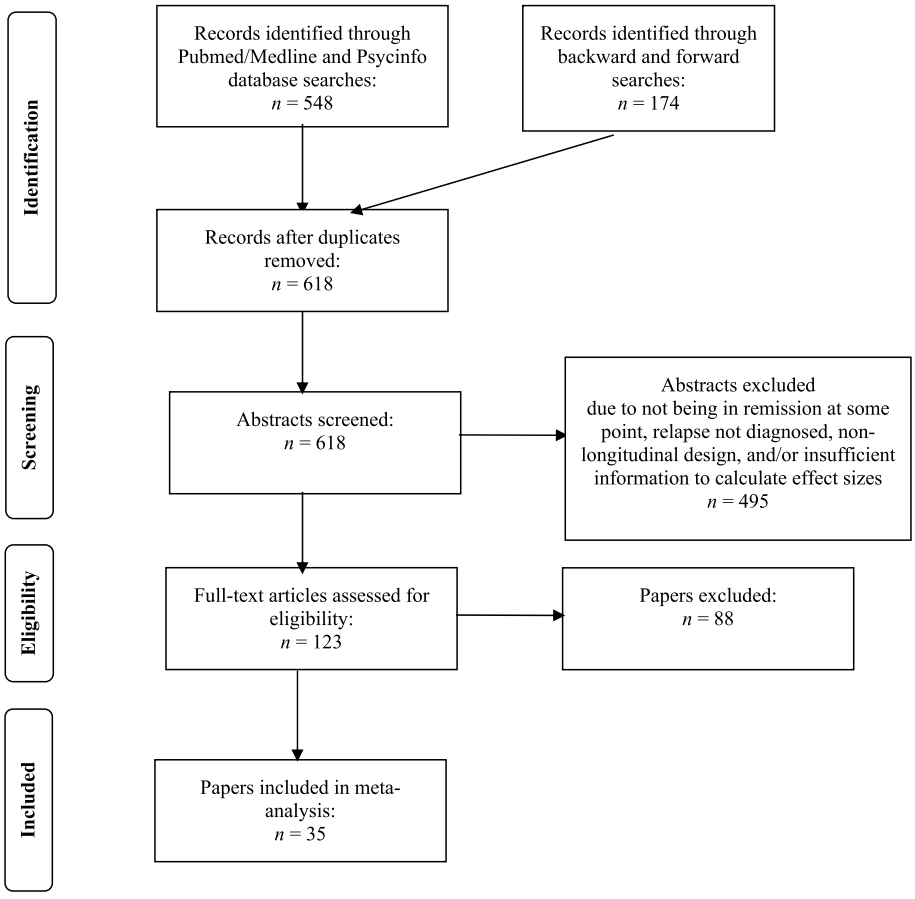

Fig. 1. Prisma flow diagram.

and 9 of individuals with mixed EDs (e.g., a combination of AN, BN, BED, and/or OFSED). The average length of follow-up was 29.0 months (*SD* = 34.06). The average rate of relapse was 36.1% (*SD* = 9.0). The relapse rate for AN only samples was 37.1% (*SD* = 7.5), for BN only samples was 34.9% (*SD* = 5.9), for BED only samples was 31.1% (*SD* = 2.6), and for mixed ED samples was 36.3% (*SD* = 13.8).

3.2. Publication bias

The Egger's regression test indicated that the funnel plots for our data were not asymmetric for age ($t = -0.50$, $p = .623$), age of ED onset ($t = -1.60$, $p = .212$), illness duration ($t = -0.45$, $p = .660$), BMI/weight/body fat ($t = 0.94$, $p = .361$), levels of ED psychopathology ($t = 0.97$, $p = .334$), mean energy density/variety ($t = -0.86$, $p = .405$), psychiatric comorbidity ($t = 0.50$, $p = .623$), level of care ($t = -2.17$, $p = .071$), response to treatment ($t = 0.46$, $p = .654$), levels of potential ED risk factors ($t = -0.35$, $p = .730$), self-esteem ($t = -0.27$, $p = .809$), motivation ($t = -0.30$, $p = .781$), leptin ($t = 0.70$, $p = .527$), and BDNF ($t = -3.09$, $p = .091$), suggesting that, for these predictor categories, study effect sizes were not related to respective sample sizes (see Fig. 2).

The funnel plot of our data was asymmetric for AN-R (vs. AN-BP) diagnosis ($t = 3.00$, $p = .022$) and treatment duration ($t = 3.26$, $p = .023$), suggesting the possibility of publication bias. Because the funnel plots were asymmetric for these predictors, we conducted trim and fill analyses. Using the observed and imputed effect sizes, AN-R (vs. AN-BP) continued to be associated with significantly lower likelihood of relapse (OR = 0.43, 95% CI = 0.26–0.69), and treatment duration continued to not be significantly associated with likelihood of relapse (OR = 1.13, 95% CI = 0.76–1.68).

3.3. Predictors of relapse

Table 3 summarizes the odds ratios linking each predictor to odds of ED relapse and tests of heterogeneity. The Q homogeneity test was significant for age, illness duration, BMI/weight/body fat, severity of ED psychopathology, meal energy density/variety, psychiatric comorbidity, response to treatment, and leptin, indicating between-study heterogeneity for studies involving these predictors. The I^2 and τ^2 were highest for age, illness duration, and response to treatment, suggesting that the highest heterogeneity occurred for these predictors (see Table 3).

Increased likelihood of relapse. Higher level of care, having a comorbid psychiatric disorder, and more severe ED psychopathology were associated with higher likelihood of relapse (see Table 3).

We further examined which specific psychiatric comorbidities were predictive of ED relapse, and found that only comorbid depression was significantly associated with higher likelihood of ED relapse. Comorbid anxiety, general comorbid psychiatric disorder, and comorbid personality disorder were not significantly associated with likelihood of relapse. There were not enough effects to test the extent to which post-traumatic stress disorder, obsessive compulsive disorder, and substance use were associated with likelihood of ED relapse (see Table 3).

We also examined which specific ED symptoms were predictive of ED relapse, and found that more severe restraint, binge eating, bulimic symptoms, body dissatisfaction, and other ED symptoms (e.g., EDI maturity fears, interpersonal distrust, perfectionism, ineffectiveness, etc.) were significantly associated with higher likelihood of relapse. Global ED symptoms and over-exercise were not significantly associated with likelihood of ED relapse (see Table 3).

Reduced likelihood of relapse. Higher leptin, higher meal energy density/variety [e.g., higher motivation for change, better response to treatment, AN-R (vs. AN-BP) diagnosis, higher BMI/weight/body fat,

Table 2
Categories definition.

Predictor	Examples
Older age	Age 19+ (vs. < 18); Age (years)
Older age of ED onset	Age of ED onset
Illness duration	Illness duration
AN-R (vs. AN-BP)	AN-R (vs. AN-BP) subtype
BMI/weight/body fat	Percent from target weight; BMI; percent body fat; weight; waist-to-hip ratio
Severity of ED psychopathology	
Restraint	EDE/EDE-Q restraint; EDI drive for thinness
Binge eating	Binge eating frequency
Bulimic symptoms	EDI bulimic symptoms; frequency of purging; frequency of vomiting; has vomiting; has laxative abuse
Body dissatisfaction	EDI body dissatisfaction; EDE/EDE-Q weight concern; EDE/EDE-Q shape concern; body checking; body avoidance
Other ED symptoms	EDI maturity fears; EDI interoceptive awareness; EDI perfectionism; EDI ineffectiveness; EDI interpersonal distrust
Global ED symptoms	EAT total score; EDI total scores
Overexercise	Has periodic overactivity; self-reported IPAQ physical activity; SIAB physical activity
Meal energy density and variety	Dietary energy density; fat (g and %kcal); protein (g and %kcal); carbohydrates (g and %kcal); non-caloric beverage; caloric intake; diet variety score assessed via food records
Comorbid psychiatric disorder	
Depression	BDI scores; MMPI-2 depression scores; has current major depressive episode; NEO personality inventory depression scores; HDRS scores
Anxiety	MMPI-2 anxiety scores; NEO anxiety scores; STAI scores
General psychiatric disorder	Comorbid psychiatric comorbidity (vs. no comorbidity); greater amount of persisting psychopathology
Personality disorder	Has a personality disorder; Has borderline personality disorder; has obsessive compulsive personality disorder
Level of care	Inpatient + outpatient tx (vs. outpatient only); specialized ED treatment; additional psychopharmacological treatment; additional medical treatment
Treatment duration	Length of stay; length of hospitalization period; inpatient length of stay; partial hospitalization length of stay; treatment duration
Response to treatment	Weight gain lability during treatment; rate of weight gain during treatment; response rate to treatment (rapid vs. slow vs. partial); weight increase during treatment; response rate to meal plan; weekly weight gain rate
Levels of various potential ED risk factors	History of childhood physical abuse; history of AN in the family; NEO vulnerability to stress; MMPI impulsiveness; MMPI obsessiveness; MMPI neuroticism; MMPI type A behavior; MMPI anger; MMPI health concerns; parental substance use disorder
Self-esteem	RSES scores
Motivation for change	ANSOCQ motivation to change
Leptin	Fasting leptin
BDNF	BDNF

Note. AN-R = Anorexia Nervosa – Restricting Subtype; AN-BP = Anorexia Nervosa – Binge Purge Subtype; BMI = Body Mass Index; ED = Eating Disorder; EDE = Eating Disorder Examination; EDI = Eating Disorder Inventory; EAT = Eating Attitudes Test; IPAQ= International Physical Activity Questionnaire; SIAB= Structured Inventory for Anorexic and Bulimic Symptoms; BDI = Beck Depression Inventory; MMPI = Minnesota Multiphasic Personality Inventory; HDRS = Hamilton Depression Rating Scale; STAI = State Trait Anxiety Inventory; Tx = Treatment; RSES = Rosenberg Self-Esteem Scale; ANSOCQ = Anorexia Nervosa Stages of Change Questionnaire; BDNF= Brain Derived Neurotrophic Factor.

and older age of ED onset] were associated with reduced likelihood of relapse (see Table 3).

No association with relapse. Age, illness duration, levels of various potential ED risk factors (e.g., stress vulnerability, impulsiveness, family problems, etc.), self-esteem, treatment duration, and BDNF were not significantly associated with likelihood of ED relapse.

3.4. Sources of variability

Type of ED. Moderation analysis revealed that several effects were only significant among samples consisting solely of individuals with AN and/or BN (see Table 4 for all statistics). Specifically, higher BMI, and a comorbid psychiatric disorder significantly predicted a higher likelihood of relapse only in samples comprising solely individuals with AN, but not in samples of individuals with other EDs (e.g., BN, mixed EDs). More severe ED psychopathology was only significant in predicting relapse among samples consisting solely of individuals with BN, but not in samples of individuals with other EDs (e.g., AN, BED, mixed EDs). Better response to treatment only predicted a lower likelihood of relapse in samples of solely individuals with AN and BN, but not in samples of individuals with BED and mixed EDs.

The association between age and the likelihood of relapse differed depending on ED type, such that older age only predicted lower likelihood of relapse in samples consisting of individuals with mixed EDs, but not in samples of solely individuals with AN or BN. There were not sufficient effects to examine the extent to which age predicted likelihood of relapse in samples of individuals with BED.

ED type did not moderate the relationship between age of ED onset, illness duration, levels of various potential ED risk factors, self-esteem, treatment duration, and odds of relapse. There was not enough variability in ED type among the effect sizes to test the extent to which ED type moderates the relationship between AN-R (vs. AN-BP) subtype, meal energy density/variety, level of care, motivation, leptin, and BDNF with odds of relapse.

Age. Moderation analysis revealed that age moderated the relationship between various predictors and likelihood of relapse (see Table 5). Age moderated the relationship between having a comorbid psychiatric disorder, as well as various levels of potential ED risk factors and odds of relapse, such that higher age was associated with lower odds of relapse from having a comorbid psychiatric disorder. For example, for a sample mean age of 15, having a comorbid psychiatric disorder was associated with 2.35 increased odds of relapse, whereas for a sample mean age of 30, having a comorbid psychiatric disorder was associated with only 1.25 increased odds of relapse. Similarly, higher age was associated with lower odds of relapse from potential ED risk factors. For example, for a sample mean age of 15, having higher levels of various potential ED risk factors was associated with 2.69 increased odds of relapse, whereas for a sample mean age of 30, having higher levels of various potential ED risk factors was associated with 1.16 increased odds of relapse. Age moderated the relationship between treatment response and odds of relapse, such that higher age was associated with higher odds of relapse from better treatment response. For example, for a sample mean age of 15, a better treatment response was associated with 0.31 odds of relapse, whereas for a sample mean age of 30, a better treatment response was associated with 0.61 odds of relapse.

Sample age did not moderate the relationship between age, age of ED onset, illness duration, AN-R (vs. AN-BP) diagnosis, BMI, levels of ED psychopathology, level of care, treatment duration, self-esteem, motivation, BDNF, and odds of relapse. There was not enough variability in sample age to test the extent to which age moderates the relationship between meal energy density/variety and leptin with odds of relapse.

Length of Study Follow-Up. Moderation analysis revealed that length of follow-up moderated the relationship between various predictors and likelihood of relapse (see Table 6). Length of follow-up also moderated the relationship between better response to treatment and odds of relapse, such that higher length of follow-up was associated with

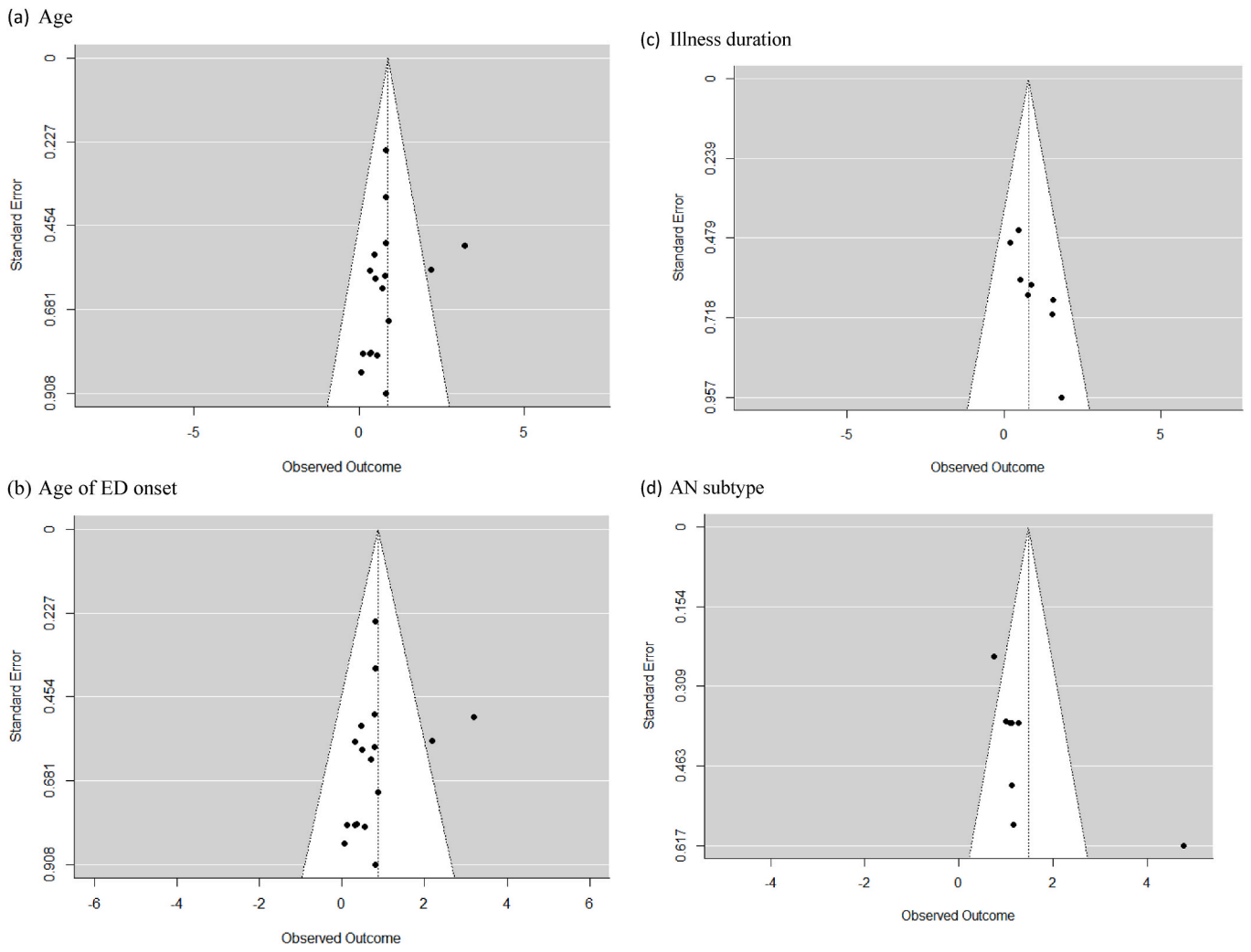

Fig. 2. Funnel plots showing precision of the following predictors of ED relapse: (a) age, (b) age of ED onset, (c) illness duration, (d) AN subtype, (e) BMI/weight/body fat, (f) severity of ED psychopathology, (g) meal energy density/variety, (h) psychiatric comorbidity, (i) treatment intensity, (j) treatment duration, (k) response to treatment, (l) levels of various potential ED risk factors, (m) self-esteem, (n) motivation for change, (o) leptin, and (p) BDNF. Filled circles represent observed studies in each study, and line represent the observed summary effects.

lower odds of relapse from better treatment response. For example, at 24-months follow-up, a better response to treatment was associated with 0.51 odds of relapse, while at 36-months follow-up, a better response to treatment was associated with 0.46 odds of relapse. Length of follow-up also moderated the relationship between levels of various potential ED risk factors and odds of relapsing, such that a longer length of follow-up was associated with higher odds of relapsing from higher levels of various potential ED risk factors. For example, at 24-month follow-up, higher levels of various potential ED risk factors were associated with 1.35 odds of relapsing, and at 36-months follow-up the odds increased to 1.56.

The length of follow-up did not moderate the relationship between age, age of ED onset, illness duration, AN-R (vs. AN-BP) diagnosis, BMI/weight/body fat, ED psychopathology, comorbid psychiatric disorder, level of care, treatment duration, self-esteem, motivation, leptin, BDNF and odds of relapse. There was not enough variability in length of follow-up among the effect sizes to test the extent to which length of follow-up moderates the relationship between meal energy density/variety and odds of relapse.

Timing of Predictor Assessment. The timing of the predictor assessment moderated the relationship between several variables and odds of relapse such that several predictors were only significantly associated with likelihood of relapse when they were assessed at discharge (see Table 7). Specifically, having a comorbid psychiatric disorder, various potential ED risk factors, and higher levels of leptin were only significantly associated with relapse when the predictor was assessed at discharge, but not when it was assessed at admission. A higher BMI/weight/body fat was associated with lower odds of relapse when it was assessed at discharge than when assessed at admission.

Other predictors were only significantly associated with likelihood of relapse when they were assessed at admission. Specifically, higher motivation was only significantly associated with lower odds of relapse when motivation was assessed at admission, but not when motivation was assessed at discharge. More severe ED psychopathology was associated with slightly higher odds of relapse when ED psychopathology was assessed at admission than when it was assessed at discharge.

The timing of the predictor assessment (i.e., admission vs. discharge) did not moderate the relationship between age, illness duration, AN-R vs. AN-BP subtype, level of care, self-esteem, and BDNF with odds of relapse. There was not enough variability in the timing of predictor assessments to test the extent to which the timing of predictor assessment moderates the relationship between age of onset, meal energy density/variety, treatment duration, response to treatment, and odds of relapse.

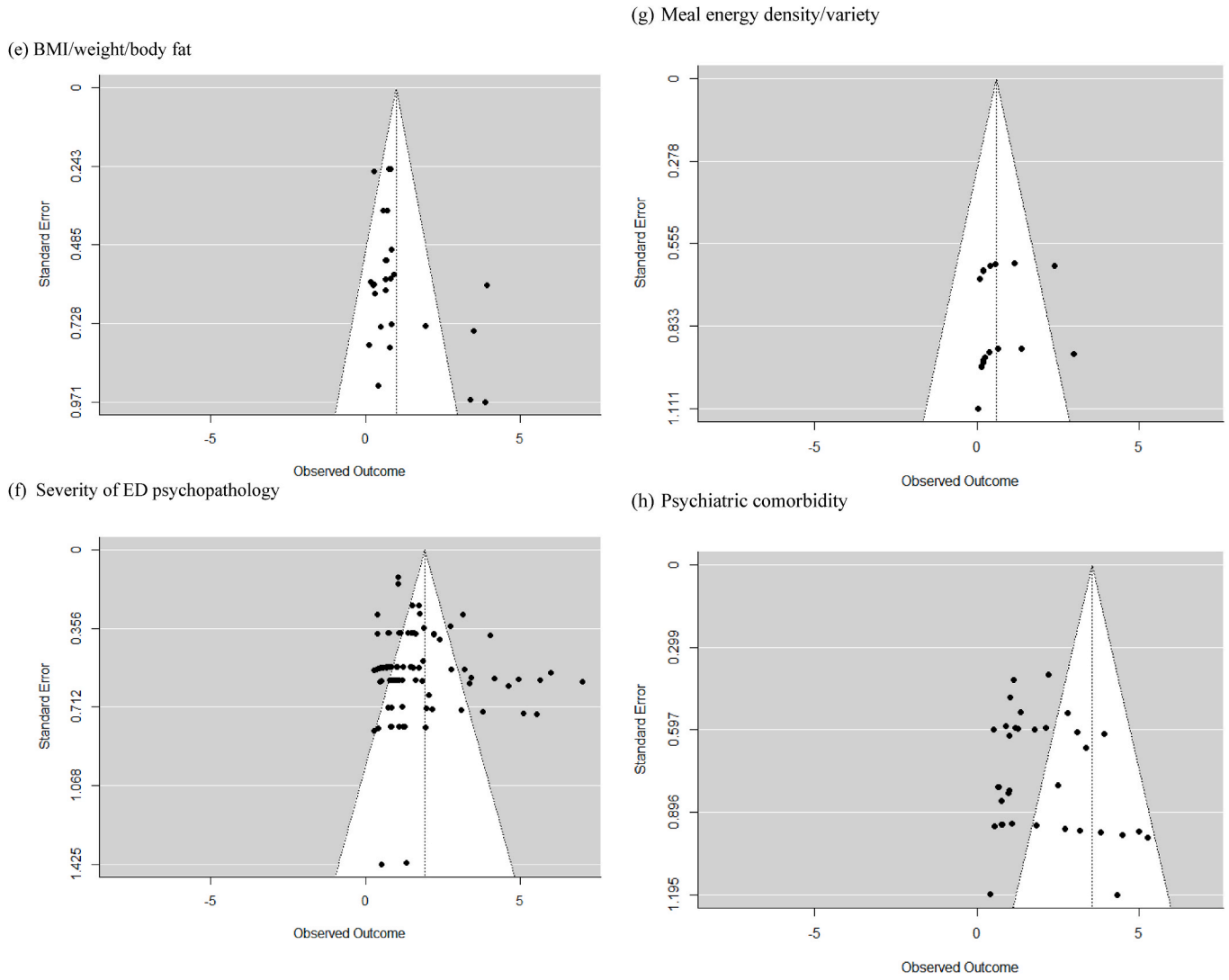

Fig. 2. (*continued*).

Relapse Operationalization. Moderation analysis revealed that several effects varied depending on the relapse operationalization used (see Table 8 for all statistics). Specifically, older age predicted higher likelihood of relapse when relapse was operationalized based on BMI + psychiatric symptoms, but lower likelihood of relapse when relapse was operationalized based on psychiatric symptoms only. Longer illness duration only predicted a higher likelihood of relapse when the relapse operationalization included BMI (e.g., BMI only or BMI + behavior + psychiatric), but not when relapse was operationalized via behavior only, psychiatric symptoms only, or re-entry into treatment. Stronger treatment intensity only predicted higher odds of relapse when the operationalization included psychiatric symptoms (e.g., BMI + psychiatric, BMI + psychiatric + behavior), but not when it only included BMI or behavioral symptoms. Higher BMI/weight/body fat, as well as having a comorbid psychiatric disorder only predicted lower odds of relapse when relapse was operationalized via BMI only or re-entry into treatment. Severity of ED psychopathology predicted higher odds of relapse only when relapse was operationalized via re-entry into treatment or via behaviors only. Better response to treatment only predicted lower odds of relapse when relapse was operationalized with re-entry into treatment or via BMI + psychiatric symptoms + behaviors, but not when operationalized via BMI only or behavior only.

The relapse operationalization did not moderate the relationship between age of ED onset, AN subtype, treatment duration, levels of various potential ED risk factors, and self-esteem. There was not enough variability in relapse operationalizations used among the effect sizes to test the extent to which the relapse operationalization moderates the relationship between motivation, meal energy density/variety, leptin, and BDNF with odds of relapse.

4. Discussion

Across ED diagnoses, approximately one third of individuals experienced relapse. This rate of relapse is high and problematic, as relapse can lead to cycles of continuous readmission and discharge from treatments among ED patients (Stewart et al., 2014; Vall and Wade, 2017). We identified several predictors of relapse and moderators, which could guide future intervention research to prevent relapse among individuals with EDs, as well as future research on predictors of relapse.

4.1. Predictors of ED relapse

ED characteristics. Findings that AN-BP is associated with higher likelihood of relapse suggest that AN-BP may reflect a later and more severe stage of illness (Franko et al., 2018; Serra et al., 2021). This finding may also be because patients with AN-R only have one route to relapse (i.e., weight loss), whereas patients with AN-BP have two routes to relapse (i.e., weight loss or binge/purge behaviors; Carter et al.,

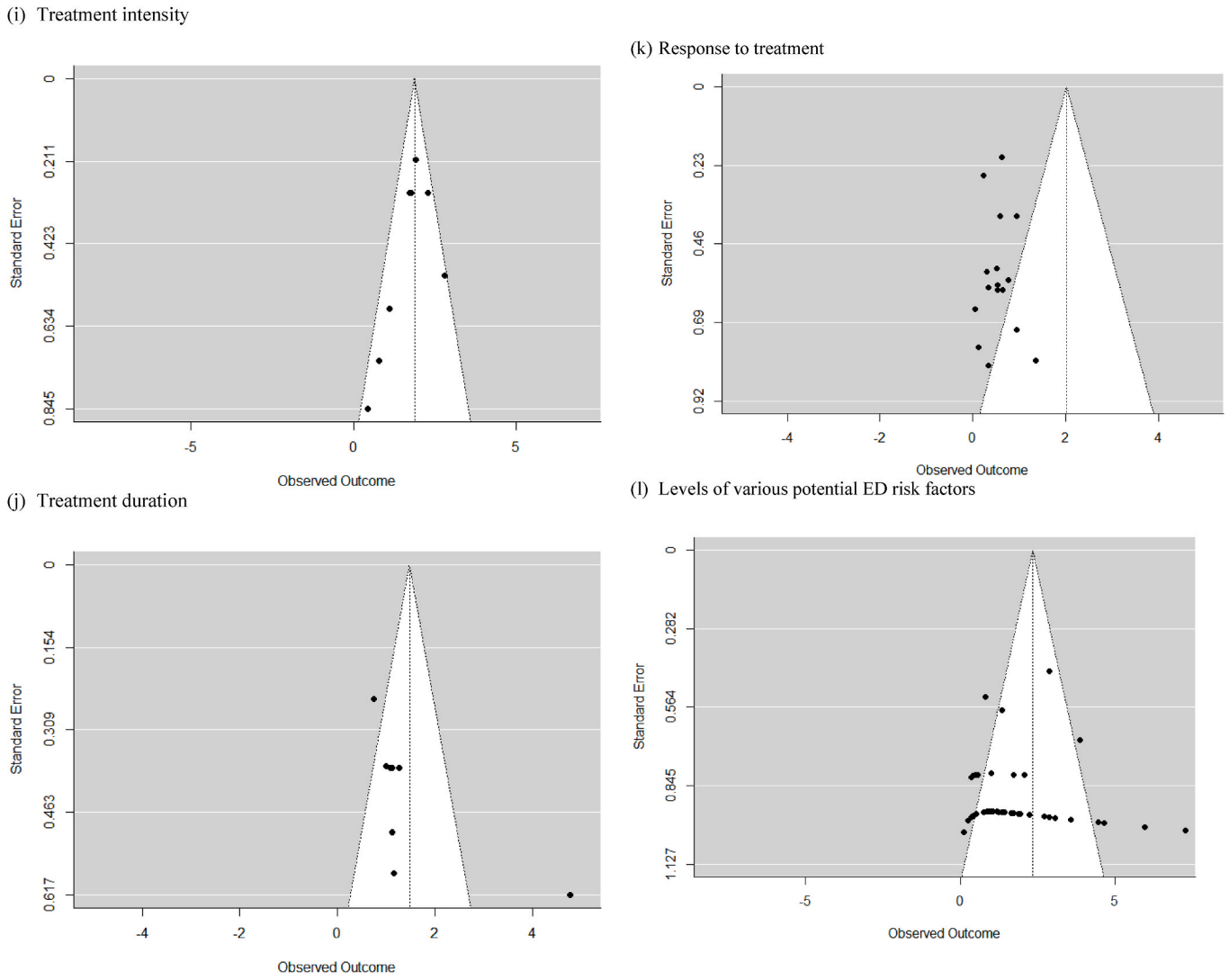

Fig. 2. (*continued*).

2004). These possibilities should be explored in future research.

Consistent with Berends et al. (2018), more severe ED psychopathology predicted higher likelihood of ED relapse. Almost all ED symptoms (except over-exercise) were significantly associated with ED relapse, and dietary restraint was most strongly associated with relapse, suggesting that although all ED symptoms should be targeted, it may be particularly important to target restraint in treatment and ensure that it ameliorates prior to discharge. Moderator analyses revealed that severity of ED psychopathology only predicted risk of relapse in samples of individuals with BN, suggesting that future research should examine if targeting ED psychopathology during treatment and ensuring abstinence from ED behaviors is particularly important to prevent relapse among individuals with BN specifically. Notably, severity of ED psychopathology was only predictive of relapse when relapse was operationalized via re-entry into treatment or via behaviors, but not when the relapse operationalization included BMI, suggesting that severity of ED psychopathology may be predictive of the return of ED behaviors and re-entry into treatment, but not of BMI changes.

Normalized and varied eating behaviors were associated with lower likelihood of relapse. Notably, most of the included effect sizes analyzing the predictive effect of meal energy density/variety were in AN samples. Future AN treatments and relapse prevention research should examine the efficacy of having a strong emphasis on helping patients learn to eat a larger variation of energy dense meals.

Findings suggest that trying to maintain a weight that is too low is associated with higher odds of relapse, potentially because individuals who try to maintain a weight that is too low may be engaging in a variety of ED behaviors to do so. Accepting a body appropriate weight may improve a patient's prognosis. Furthermore, patients who leave treatment prematurely with lower BMIs are likely to relapse, emphasizing the need for providers and financial supporters to ensure that patients can stay in treatment until fully weight-restored. Moderator analyses revealed that these findings are only applicable to individuals with AN - perhaps because AN is the ED that is characterized by low body weight) - and when relapse was operationalized as re-entry into treatment or with. It makes sense that a lower BMI at discharge would predict a higher likelihood of returning to an underweight BMI.

Older age of ED onset predicted lower odds of ED relapse, suggesting that chronicity is more likely when EDs begin at a younger age. However, illness duration was not significantly associated with odds of ED relapse. This latter finding suggests that it is possible to avoid relapse despite longer illness duration, which should be communicated to patients and clinicians to increase their confidence regarding the patient's abilities to remain in recovery. However, it is important to note that longer illness duration did predict higher odds of relapse when the relapse operationalization included BMI (but did not when the relapse operationalization only included psychiatric symptoms, behavior symptoms, or re-entry into treatment), suggesting that individuals who

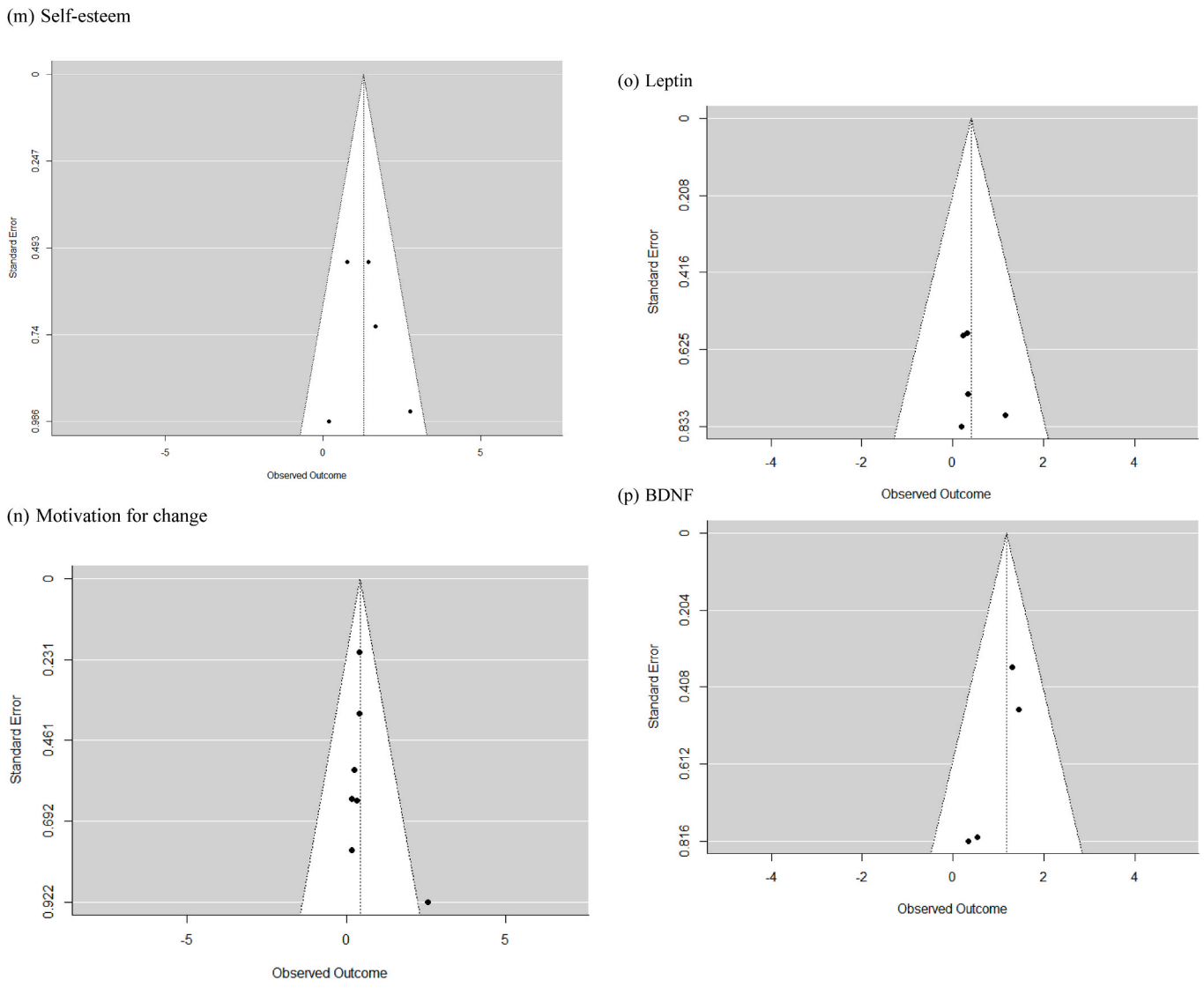

Fig. 2. (*continued*).

have had a longer illness duration may have a harder time maintaining weight restoration specifically.

Comorbid psychiatric disorder Having a comorbid psychiatric disorder was associated with higher odds of relapse, highlighting the importance of effectively treating comorbid psychopathology. The predictive effect of having a comorbid psychiatric disorder on odds of relapse was stronger for younger samples, revealing the importance of addressing psychiatric comorbidity especially in younger individuals with EDs. Moderator analyses revealed that having a comorbid psychiatric disorder only increased odds of relapse in samples of individuals with AN, suggesting that future research should explore if a focus on treating psychiatric comorbidities may be particularly helpful in preventing relapse among individuals with AN. Moderator analyses also revealed that having a comorbid psychiatric disorder only increased odds of relapse when relapse was operationalized via BMI only or re-entry into treatment, but not when the relapse operationalization included psychiatric symptoms or behavior.

Having a comorbid psychiatric disorder only increased odds of relapse when it was assessed at discharge. This result may be because many individuals with EDs present with psychiatric comorbidity at the beginning of treatment (e.g., potentially due to malnourishment, medical complications, psychosocial ramifications of intensive ED treatment, etc.; Berends et al., 2016), and there may therefore be a low amount of variability in psychiatric comorbidity at the beginning of treatment, reducing its predictive power. There may be more variability in the extent to which having a comorbid psychiatric disorder resolves/improves during treatment. It may be that only patients who are unable to experience improvements in psychiatric comorbidity during treatment are at increased likelihood of relapse (Kahn et al., 2020), underscoring the importance of including treatment of comorbid psychopathology in ED treatment planning and that it is important to target co-occurring psychiatric illnesses before discharging from treatment.

Further analysis revealed that only depression symptoms specifically were associated with increased likelihood of relapse. However, this may have been the case due to the low number of effects for other disorders.

Treatment process. Receiving a higher level of care predicted higher odds of relapse. Of note, moderator analyses revealed that a higher level of care predicted a higher odds of relapse only when the operationalization of relapse included psychiatric symptoms (e.g., BMI + psychiatric symptoms or BMI + behavior + psychiatric symptoms), suggesting that needing a higher level of care may be related to higher levels of ED psychiatric symptoms, which in turn could be related to a higher likelihood of a return of those symptoms. Future research should examine the extent to which the subgroup of patients who necessitate more intense treatment should be offered intensive monitoring and extra attention after being discharged from treatment.

Table 3
Predictors of ED relapse.

Predictor	OR (95% CI)	*k*	*Q*	I^2	τ^2
Older age	.71 (.48–1.06)	17	31.79**	48.3	.28
Older age of ED onset	.63 (.42–.94)**	5	2.82	0	0
Longer illness duration	1.08 (.72–1.64)	13	27.32***	55.5	.27
AN-R (vs. AN-BP)	.58 (.36–.93)**	8	9.68	21.2	.10
Higher BMI/weight/body fat	.62 (.52–.75)***	26	52.01***	0	0
Severity of ED psychopathology	1.57 (1.19–2.06)***	89	209.38***	1.96	.21
Restraint	3.29 (1.89–5.72)***	9			
Binge eating	1.97 (1.11–3.50)*	11			
Bulimic symptoms	1.81 (1.16–2.82)**	11			
Body dissatisfaction	1.57 (1.03–2.39)*	20			
Other ED symptoms	2.14 (1.28–3.58)**	12			
Global ED symptoms	1.01 (.71–1.70)	13			
Overexercise	1.82 (.93–3.59)	5			
Higher meal energy density and variety	.34 (.24–46)***	21	37.87***	0	0
Comorbid psychiatric disorder	1.59 (1.20–2.10)***	36	51.69*	1.7	.07
Depression	1.57 (1.15–2.13)**	19			
Anxiety	1.40 (.75–2.60)	6			
General psychiatric disorder	1.43 (.88–2.30)	5			
Personality disorder	1.07 (.49–2.34)	4			
Stronger level of care	1.81 (1.43–2.29)***	8	6.52	0	0
Longer treatment duration	1.06 (.81–1.39)	8	8.32	0	0
Better response to treatment	.52 (.35–.76)***	17	49.50***	10.9	.24
Higher levels of various potential ED risk factors	1.28 (.80–2.05)	46	52.36	2.8	.15
Higher self-esteem	1.07 (.59–1.92)	5	5.14	0	0
Higher motivation for change	.37 (.27–.50)***	7	8.32	0	0
Higher leptin	.33 (.18–.61)***	5	3.13**	0	0
Higher BDNF	1.00 (.53–1.87)	4	3.49	15.6	.06

Note. k = number of observed studies, *OR* = odds ratio, 95% CI = 95% confidence intervals.; *AN-R* = anorexia nervosa – restricting subtype; *AN-BP* = anorexia nervosa – binge purge subtype * $p < .05$, ** $p < .01$, *** $p < .001$. ED = Eating Disorder; AN-BP = Anorexia Nervosa- Binge Purge Subtype; BMI= Body Mass Index; BDNF= Brain Derived Neurotrophic Factor.

Table 4
Eating disorder type as a moderator.

	OR	95% CI	$Q_{between}$
Older age			8.84***
AN	0.99	[0.63, 1.56]	
BN	0.49	[0.23, 1.05]	
Mixed EDs*	0.45	[0.23, 0.88]	
Older age ED Onset			6.40
Longer illness duration			5.23
Higher BMI/weight/body fat			26.22***
AN***	0.60	[0.49, 0.74]	
BN	0.86	[0.48, 1.54]	
Mixed EDs	0.57	[0.27, 1.21]	
Severe ED psychopathology			23.76***
AN	1.33	[0.98, 1.80]	
BN***	2.67	[1.68, 4.25]	
BED	2.10	[0.92, 4.80]	
Mixed EDs	1.10	[0.69, 1.78]	
Comorbid psychiatric disorder			17.78***
AN***	1.86	[1.35, 2.54]	
BN	1.16	[0.75, 1.81]	
Mixed EDs	2.04	[0.85, 4.87]	
Stronger level of care			23.99**
AN***	1.81	[1.34, 2.45]	
BN**	1.79	[1.22, 2.63]	
Longer treatment duration			0.23
Better response to treatment			12.63**
AN*	0.54	[0.32, 0.89]	
BN*	0.41	[0.20, 0.84]	
BED	0.95	[0.18, 5.15]	
Mixed EDs	0.59	[0.17, 1.96]	
Higher levels of various potential ED risk factors			1.73
Higher self-esteem			0.52
Higher motivation for change			41.07**
AN***	0.35	[0.21, 0.58]	
BN***	0.38	[0.26, 0.55]	

Note: * $p < .05$, ** $p < .01$, *** $p < .001$.
AN = Anorexia Nervosa; BN= Bulimia Nervosa; ED = Eating Disorder; BED= Binge Eating Disorder; BMI= Body Mass Index.

Individuals who made better progress during treatment were less likely to relapse. This finding highlights the potential importance of engaging patients in treatment and motivating them to adhere to prescribed strategies and attain full recovery. Interestingly, a better response to treatment appears to be even more predictive of relapse odds with longer-follow-up, potentially because, for most patients, relapse only becomes an issue after a few months have passed since they received treatment and recovered (Berends et al., 2016; Carter et al., 2004). Moderator analyses also revealed that response to treatment may be most relevant to AN and BN. Furthermore, the protective effect of better response to treatment was strongest in younger samples, highlighting the possible importance of engaging younger patients in treatment that aims for amelioration of symptoms rather than just some improvement in symptoms. Finally, a better response to treatment was only predictive of relapse when relapse was operationalized via a comprehensive measure of symptoms (BMI + behavior + psychiatric) or re-entry to treatment, but not when operationalized via BMI only or behavior only, suggesting that response to treatment likely predicts a full return to ED symptoms, rather than just certain symptoms of ED pathology.

Other variables. On the aggregate, higher motivation was one of the most robust predictors of lower likelihood of relapse. Moderator

Table 5
Age as a moderator.

	b	SE	Q_M
Older age	−0.02	0.03	0.39
Older age ED Onset	0.04	0.09	0.21
Longer illness duration	−0.02	0.05	0.22
AN-R (vs. AN-BP)	−0.10	0.06	3.34
Higher BMI/weight/body fat	0.01	0.02	0.31
Severe ED psychopathology	0.00	0.02	0.02
Comorbid psychiatric disorder	−0.05	0.02	4.79*
Stronger level of care	0.02	0.05	0.26
Longer treatment duration	−0.01	0.05	0.76
Better response to treatment	0.05	0.02	7.68*
Higher levels of various potential ED risk factors	−0.06	0.03	3.97*
Higher self-esteem	0.02	0.04	0.44
Higher motivation for change	0.06	0.05	1.15
Higher BDNF	0.08	0.46	0.03

Note: *$p < .05$.
ED = Eating Disorder; AN-R = anorexia nervosa – restricting subtype; BMI= Body Mass Index; BDNF= Brain Derived Neurotrophic Factor.

Table 6
Length of study follow-up as a moderator.

	b	SE	Q_M
Older age	0.00	0.00	0.00
Older age ED Onset	0.00	0.00	0.50
Longer illness duration	0.00	0.00	0.03
AN-R (vs. AN-BP)	0.00	0.01	0.03
Higher BMI/weight/body fat	0.00	0.00	0.95
Severe ED psychopathology	0.00	0.01	0.06
Comorbid psychiatric disorder	0.00	0.01	0.70
Stronger level of care	0.03	0.02	2.72
Longer treatment duration	−0.01	0.00	2.91
Better response to treatment	−0.01	0.01	4.95*
Higher levels of various potential ED risk factors	0.01	0.01	7.96**
Higher self-esteem	−0.04	0.08	0.22
Higher motivation for change	0.01	0.01	0.50
Higher leptin	−0.05	0.07	0.63
Higher BDNF	0.03	0.02	1.72

Note: *$p < .05$, **$p < .01$.
ED = Eating Disorder; AN-R = Anorexia Nervosa Restricting Subtype; AN-BP = Anorexia Nervosa Binge/Purge Subtype; BMI= Body Mass Index; BDNF= Brain Derived Neurotrophic Factor.

Table 7
Timing of predictor assessment as a moderator.

	OR	95% CI	Q_M
Older age			1.47
Older age ED Onset			0.21
Longer illness duration			5.22
AN-R (vs. AN-BP)			0.94
Higher BMI/weight/body fat			29.80***
Admission	0.70**	[0.53, 0.91]	
Discharge	0.43***	[0.30, 0.61]	
Severe ED psychopathology			10.15**
Admission	1.65**	[1.18, 2.29]	
Discharge	1.56**	[1.14, 2.13]	
Comorbid psychiatric disorder			15.31***
Admission	1.04	[0.68, 1.58]	
Discharge	2.16***	[1.47, 3.18]	
Stronger level of care			4.37
Higher levels of various potential ED risk factors			9.27***
Admission	0.83	[0.55, 1.27]	
Discharge	1.71**	[1.15, 2.55]	
Higher self-esteem			0.06
Higher motivation for change			8.21*
Admission	0.41*	[0.19, 0.87]	
Discharge	0.45	[0.18, 1.13]	
Higher leptin			13.55**
Admission	1.15	[0.24, 5.55]	
Discharge	0.26***	[0.12, 0.53]	
Higher BDNF			0.65

Note: *$p < .05$, **$p < .01$, ***$p < .001$.
ED = Eating Disorders; AN-R = Anorexia Nervosa Restricting Subtype; AN-BP = Anorexia Nervosa Binge/Purge Subtype; BDNF= Brain Derived Neurotrophic Factor.

analyses revealed that motivation only predicted lower odds of ED relapse when it was assessed at admission, but not when it was assessed at discharge. Individuals with EDs are often ambivalent about recovery (Williams and Reid, 2010). Higher motivation at admission may be key for increasing treatment engagement and achieving changes in cognitions and behaviors during treatment that may ultimately help in contributing to sustained change and recovery (Wade et al., 2009; Castro-Fornieles et al., 2011). This finding has potential important treatment implications that should be examined in future research because it suggests that motivation for change needs to be addressed soon after beginning treatment.

On the aggregate, higher leptin was one of the most robust predictors of lower likelihood of ED relapse. Of note, all studies examining leptin as a predictor of relapse were in AN samples. Therefore, these findings only apply to individuals with AN, and no conclusions can be made to the extent to which leptin predicts relapse in individuals with other EDs. Moderator analyses revealed that higher leptin was only predictive of lower odds of relapse when it was assessed at discharge, suggesting that leptin levels at discharge may be a biomarker of AN relapse. Leptin levels may be indicative of fat mass or could reflect the reward status of food-restriction (Dardennes et al., 2021). However, this finding should be interpreted with caution given the small number of effect sizes included.

On the aggregate, levels of various potential ED risk factors did not predict ED relapse. However, moderator analyses revealed that when these various potential risk factors were assessed at discharge (vs. at admission), higher levels of them were associated with higher likelihood of relapse. Furthermore, moderator analyses suggested that the predictive effect of various potential risk factors increased with longer follow-up. This finding highlights that future research should examine the importance of addressing factors such as stress, impulsivity, and family problems during ED treatment.

4.2. Limitations

First, certain characteristics of the included studies limit our conclusions. Of particular concern, there was a lack of consistency in how recovery and relapse were defined (see Table 1), which limits the validity of the current meta-analysis. Notably, the relapse operationalization used moderated several of the findings (see Table 8). Researchers should continue collaborating to establish a consensus definition of recovery and relapse (Bardone-Cone et al., 2010; Khalsa et al., 2017). Relatedly, most studies classified relapse as a dichotomous rather than continuous outcome. While dichotomous characterization of disorders is clinically relevant, it has its limitations (e.g., decrease of statistical power; Fitzsimons, 2008; MacCallum et al., 2002; Maxwell and Delaney, 1993; McClelland et al., 2015; Rucker et al., 2015). Second, we were unable to calculate the predictive ability of several effects due to insufficient numbers to classify them into categories. Other important predictors of ED relapse may exist that we were unable to identify.

Table 8
Relapse operationalization.

	OR	95% CI	$Q_{between}$
Older age			29.22***
BMI only	0.85	[0.48, 1.54]	
Psychiatric only***	0.17	[0.07, 0.38]	
BMI + Psychiatric*	3.19	[1.18, 8.68]	
Re-entry into treatment	0.80	[0.51,1.24]	
Behavior only	0.63	[0.39,0.99]	
Older age ED Onset			9.45
Longer illness duration			14.48**
BMI only*	1.56	[1.01, 2.38]	
Psychiatric only	0.67	[0.14, 3.17]	
Re-entry into treatment	0.51	[0.07, 3.64]	
Behavior only	0.68	[0.40, 1.16]	
BMI + behavior + psychiatric**	2.26	[1.27, 4.02]	
AN Subtype			13.75
Higher BMI/weight/body fat			26.22***
BMI only***	0.51	[0.35, 0.75]	
Psychiatric only	0.82	[0.20, 3.45]	
Re-entry into treatment***	0.59	[0.45, 0.77]	
Behavior only	0.73	[0.46, 1.16]	
Severity of ED psychopathology			25.42***
BMI only	1.47	[0.93, 2.32]	
Re-entry into treatment*	1.84	[1.03, 3.30]	
Behavior only***	2.08	[1.49, 2.92]	
BMI + behavior + psychiatric	1.08	[0.51, 2.27]	
Comorbid psychiatric disorder			25.95***
BMI only***	2.56	[1.57, 4.19]	
Psychiatric only	1.95	[0.58, 6.64]	
BMI + psychiatric	1.35	[0.68, 2.70]	
Re-entry into treatment*	2.20	[1.01, 4.79]	
Behavior only	1.26	[0.89, 1.83]	
Treatment intensity			29.66**
BMI only	0.59	[0.21, 1.76]	
BMI + psychiatric*	2.80	[1.04, 7.53]	
Behavior only	1.11	[0.35, 3.54]	
BMI + behavior + psychiatric***	1.91	[1.48, 2.57]	
Longer treatment duration			2.84
Better response to treatment			64.04***
BMI only	0.92	[0.59, 1.43]	
Re-entry into treatment***	0.23	[0.15,0.36]	
Behavior only	0.46	[0.28, 0.74]	
BMI + behavior + psychiatric*	0.62	[0.41, 0.93]	
Higher levels of various potential ED risk factors			1.73
Higher self-esteem			0.68

Note: *$p < .05$, **$p < .01$, ***$p < .001$.
BMI= Body Mass Index; ED = Eating Disorder; AN = Anorexia Nervosa.

Third, some predictors had fewer included effect sizes than others. The small group sizes for some of the included predictors reduce our confidence in the precision of these effects. Furthermore, groups were even smaller when conducting moderator analyses, and there were discrepancies in group sizes in moderator analyses. Fourth, all studies examined heterogenous EDs or AN/BN/BED exclusively and no study examined OFSED exclusively. Therefore, we do not know the extent to which these predictors may predict relapse in OFSED specifically, despite the fact that OSFED is the most common ED diagnosis (Micali et al., 2017). Fifth, two studies used exclusively self-report measures to establish ED diagnosis, which is of concern due to the limited validity of diagnostic ED self-report measures. Relatedly, due to the low number of studies that used exclusively self-report measures to establish ED diagnosis, we were unable to conduct moderator analyses comparing results when measures were self-report vs. interview-based. Finally, we had to include some broad categories (e.g., various potential ED risk factors) due to low numbers of effect sizes to form narrower categories. Some of these potential ED risk factors are quite variable and would likely need to be addressed differently in treatment.

5. Conclusion

This study helps to clarify prognostic factors of ED relapse. Overall, these findings have important implications for the identification of characteristics that may lead to relapse, shedding light on key factors maintaining EDs, and identifying areas in greater need of assessment before and during treatment to minimize the risk of relapse. Findings have important implications in guiding treatment development research, including in-depth assessment and monitoring of various factors that are predictive of relapse, developing treatments that target these factors, and evidence-based guidelines for providers on when to discharge individuals with EDs from treatment.

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Declarations of competing interest

None.

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