A Systematic Review and Meta-Analysis On The Longitudinal Relationship Between Eating
Pathology and Depression

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LONGITUDINAL RELATIONSHIP OF EP AND DEPRESSION

Abstract

Objective: Undertake a meta-analysis to provide a quantitative synthesis of longitudinal studies that assessed the direction of effects between eating pathology and depression. A second aim was to use meta-regression to account for heterogeneity in terms of study-level effect modifiers. **Method:** A systematic review was conducted on 42 studies that assessed the longitudinal relationship between eating pathology and depression. Of these 42 studies, multilevel random-effects meta-analyses were conducted on 30 eligible studies. **Results:** Meta-analysis results showed that eating pathology was a risk factor for depression ($r_m = 0.13$) and that depression was a risk factor for eating pathology ($r_m = 0.16$). Meta-regression analyses showed that these effects were significantly stronger for studies that operationalized eating pathology as an eating disorder diagnosis versus eating pathology symptoms, and for studies that operationalized the respective outcome measure as a categorical variable (e.g., a diagnosis of a disorder or where symptoms were “present”/“absent”) versus a continuous measure. Results also showed that in relation to eating pathology type, the effect of an eating disorder diagnosis and bulimic symptoms on depression was significantly stronger for younger participants. **Discussion:** Eating pathology and depression are concurrent risk factors for each other, suggesting that future research would benefit from identifying factors that are etiological to the development of both constructs.

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Individuals with an eating disorder are at elevated risk for experiencing comorbid major depressive disorder (1). This comorbidity is especially prominent for those suffering from bulimia nervosa, with estimates showing the comorbidity rate to range from 31% to 50% (1, 2). The consequences of eating disorders and major depressive disorder are significant and include suicide (3, 4), economic burden (5, 6), and severe role impairment (2, 7). Despite the potentially deleterious effects of both disorders, it remains unclear why this comorbidity rate is so high and whether these factors are causally related.

In an attempt to elucidate the direction of effects between eating disorders and major depressive disorder, researchers have assessed whether eating pathology (e.g., an eating disorder or disordered eating symptoms) and depression (e.g., a depressive disorder diagnosis or depressive symptoms) are related longitudinally. Despite the considerable number of studies that have assessed evidence for a longitudinal relationship between eating pathology and depression (8, 9) there is no clear consensus regarding the direction of effects between the two constructs, or whether they are potentially bi-directionally related (10, 11). Given the inconsistencies between individual studies, the present study used meta-analysis to quantify the average effect size of eating pathology predicting depression and depression predicting eating pathology, and to also account for this heterogeneity in terms of study-level effect modifiers.

Studies that have assessed the direction of effects for the comorbidity between the two constructs have collectively tested three different models. The first model assessed whether eating pathology predicts depression (12), the second model assessed whether depression predicts eating pathology (13, 14) and the third model investigated whether eating pathology and depression are bi-directionally related (i.e., whether each construct assessed at baseline

predicts the other construct assessed at follow-up) (10, 15). Researchers (16) that have examined whether the constructs are uni-directionally related have proposed that eating pathology predicts depression due to feelings of shame and guilt that are generated from the distress associated with failing to adhere to strict dietary restraint and in turn failure to achieve an idealized and unrealistic physical ideal. Additionally, habitual loss of control over eating (e.g., binge-eating), as well as the possible effects of caloric deprivation on mood that result from dietary restraint are also thought to generate mood difficulties.

Other researchers (17) have typically advocated, consistent with the affect-regulation model of binge-eating (18) that depression predicts eating pathology. According to this affect-regulation model, individuals who experience depression binge-eat because binge-eating is a compensatory mechanism to reduce depression via distraction and/or comfort from aversive mood. Additionally, it has been suggested that individuals might engage in dietary restraint or compensatory behaviors, such as purging, in order to reduce negative feelings associated with weight gain that result from binge-eating and/or the belief that compensatory behaviors are emotionally cathartic (19). These two viewpoints - that eating pathology predicts depression and that depression predicts eating pathology - are not mutually exclusive of course, and thus some researchers (20) have investigated whether the two constructs are risk factors for each other.

To our knowledge, only two reviews have investigated the longitudinal relationship between eating pathology and depression. The first review by Stice (19) assessed whether mood difficulties, operationalized as a composite of depressive symptoms, negative-affect, and self-esteem, are a risk factor for eating pathology. Stice (19) found that mood difficulties were a small yet significant risk factor for eating pathology and that dietary restraint was a small yet significant risk factor for negative affect. Stice (19) also revealed that neither the age of participants nor the length of follow-up moderated the effect of mood difficulties on

eating pathology. The second review by Jacobi and colleagues (21) assessed whether psychiatric morbidity, psychopathology and negative emotionality were a predictor of eating pathology. Their review concluded that these higher order constructs placed individuals at risk for developing an eating disorder. However this second review did not examine any pooled effect-sizes. Further, the review was only able to include seven longitudinal studies, none of which assessed the specific relationship between eating pathology and depression.

The results of Stice's (19) meta-analysis provided evidence for the view that mood difficulties and eating pathology might be risk factors for each other, however, the paucity of studies at that time prevented Stice from conducting separate analyses to determine the unique effects of depression, negative-affect and self-esteem on eating pathology. Since Stice's and Jacobi et al.'s reviews, a considerable number of studies have investigated the longitudinal relationship between eating pathology and depression (22, 23). Existing evidence suggests a potential bi-directional relationship (10), however, a meta-analysis is required to quantify this possibility and to explore reasons for heterogeneous results observed in past studies.

Regarding study-level effect modifiers, no meta-analysis has investigated whether eating pathology type [e.g., overall disordered eating symptoms versus bulimic symptoms (i.e., binge-eating combined with compensatory behaviors such as purging) versus binge-eating symptoms versus an eating disorder diagnosis] is a factor in determining the magnitude of effect sizes on depression and vice versa despite considerable diversity in how eating pathology has been assessed (24-26). In his meta-analysis, Stice (19) examined whether the effect of mood difficulties on eating pathology differed between studies that assessed general eating pathology versus the pooled effect of studies that assessed binge-eating or bulimic symptoms. His results showed that the effect of mood difficulties was significantly stronger for studies that assessed the pooled effect of binge-eating or bulimic

symptoms ($r_m = 0.10$) compared with overall eating pathology ($r_m = 0.07$), however, given the small number of studies at that time, Stice (19) was unable to examine whether the relationship between mood difficulties differed as a function of eating pathology type (i.e., overall disordered eating symptoms versus bulimic symptoms versus binge-eating symptoms versus an eating disorder diagnosis). Regarding the effect of time-lag (i.e., the length of time between baseline and follow-up assessment), Stice (19) found that the time-lag utilized by studies in his review did not influence the effect of mood difficulties on eating pathology; however, a considerable number of studies have been published since Stice's (19) review, and we therefore now have a better opportunity to see the effect of quite disparate time-lags and number of waves of assessment on the effect of eating pathology on depression and vice versa. In addition to the moderators proposed by Stice (19), we also argue that there is a need to consider distinguishing between the effects in which eating pathology and depression have been assessed as a continuous measure versus a categorical measure. Continuous variables examine change in symptoms, whereas categorical variables investigate change in symptom status, results between studies may differ. Finally, past research (14) has illustrated that the trajectory for eating pathology symptoms differs as a function of age; for example, binge-eating symptoms in females have shown to be stable from 14 years to 17 years of age and then increase significantly from 17 years to 20 years, whereas overall disordered eating symptoms have been shown to increase from 14 years of age and peak at 17 years of age. Given these differences, it is possible that the interaction between age and eating pathology type might also be a factor in influencing the relationship between eating pathology and depression. In light of this, the present study will examine the following factors as possible moderators of the relationship between eating pathology and depression: participants' age, eating pathology type (i.e., overall disordered eating symptoms versus bulimic symptoms versus binge-eating symptoms versus an eating disorder diagnosis), the interaction between

participants' age and eating pathology type, eating pathology and depression assessment type (i.e., continuous versus categorical), time-lag assessment interval and number of waves of assessment.

This study aimed to identify and summarize the available literature that has examined the longitudinal relationship between eating pathology and depression and to quantify the size and direction of their effects by conducting a meta-analysis on available data. A second aim of the current meta-analysis was to use meta-regression to determine whether the above moderators conferred influence on the relationship between eating pathology and depression. Understanding whether the relationship between eating pathology and depression is uni- or bi-directional will help us better understand how and when eating pathology and depression influence each other. This, in turn, may have significant clinical implications that extend to early intervention for both constructs (if the relationship is bi-directional, clinicians would be prudent to screen for eating pathology in individuals with depression), as well as more effective prevention modalities (if the relationship is uni-directional, prevention interventions designed to target the construct that confers greater risk may prove to be more efficacious in attenuating symptoms of both constructs). Similarly, gaining insight into the direction of effects between eating pathology and depression may inform etiological models that explain their high comorbidity. Research has shown that biological and sociocultural influences (27) as well as psychological factors such as impulsivity (28, 29) and body dissatisfaction (30) are implicated in the etiology of both eating pathology and depression. Thus, it is possible that shared risk factors predispose individuals towards developing both eating pathology and depression, and that once symptoms of each construct are experienced, they amplify the other in a bi-directional feedback loop.

2. METHOD

2.1. Search Strategies

Search strategies followed PRISMA guidelines (31). A systematic search was undertaken by utilizing three international databases; PsycINFO, MEDLINE and Web of Science. Two researchers (FP and DO) searched all papers written in English and published in peer-reviewed journals until September 2015. The search terms for eating pathology and depression were combined with keywords for longitudinal study designs: ("eating disorder*" OR anorexi* OR bulimi* OR binge* OR purg* OR diet* OR "disordered eating") AND (depress* OR dysthymi* OR "low* affect" OR "MDD" OR "affective disorder" OR mood) AND (longitudinal OR prospective). Studies that assessed the longitudinal relationship between eating pathology and depression were included in the review. Finally, a manual search of references cited in the selected papers was performed and relevant papers were included in the review. A total of 1877 papers were retrieved as illustrated in Figure 1.

2.2. Systematic Review Selection Criteria

Inclusion criteria for the review were that studies: (1) were longitudinal; (2) employed a self-report methodology or clinical interview where scores were utilized to assess the predictive relationship between eating pathology and depression; (3) tested either *i*) a unidirectional model (e.g., that eating pathology measured at baseline was utilized as a predictor for depression measured at follow-up or that depression measured at baseline was assessed as a predictor of eating pathology measured at follow-up) or *ii*) a bi-directional model (e.g., that each construct was assessed at the same points in time to determine if eating pathology, controlling for depression at baseline, predicted both eating pathology and depression at follow up, and if depression, controlling for eating pathology at baseline, predicted both depression and eating pathology at follow up concurrently). A study was excluded from the review if it assessed specific groups (e.g., sports groups) because the mechanism(s) that link eating pathology and depression might be quantitatively different for

selected populations relative to community samples. Studies using an ecological momentary assessment design were also excluded because this review focused on trait/stable-level relationships rather than relationships from moment-to-moment, as the former more clearly links to diagnostic criteria. Studies that assessed negative-affect (presented in Figure 1) were excluded since negative-affect is a heterogeneous measure of general negative mood and this review examined the unique relationship between eating pathology and depression. Finally, studies that only assessed eating pathology compensatory behaviors (e.g., purging, extreme exercise) were excluded due to a dearth of studies on the topic (32, 33).

3. SYSTEMATIC REVIEW RESULTS

3.1 Selection of Studies for Systematic Review

Of the 98 studies that were retrieved for close reading, the following assessed the same longitudinal dataset: a) (34-36); b) (17, 37, 38); c) (39, 40); d) (41-43); e) (44-47); f) (48-50); g) (20, 51). The following studies reported the greater number of waves of data regarding the relationship between eating pathology and depression and were therefore selected for the review: (17, 20, 36, 40, 43, 44, 49). Only seven studies (9, 12, 14, 40, 52-54) included in the review assessed dietary restraint. We were only able to obtain data for dietary restraint predicting depression for six of these studies (9, 12, 14, 40, 52, 53) and only two studies (52, 54) assessed whether depression predicted dietary restraint. Hence, due to low power, the effect of dietary restraint was not examined in this review. 42 studies (8-17, 20, 22-26, 36, 40, 43, 44, 49, 52-72) (presented in Figure 1) met selection criteria and were included in the systematic review. Table 1 presents an overview of each study and their relevant findings.

---- Include Figure 1 about here -----

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4. Meta-Analysis

4.1. Selection of Studies for Meta-Analyses

As seen in Figure 1, 30 studies met selection criteria and were included in the meta-analysis. For inclusion in the meta-analysis, when the outcome was continuous, partial correlations (controlling for Time 1 scores on the outcome variable) were utilized, so that the effect reflected the ability of the independent variable to predict change in symptoms in the outcome variable. In cases where the outcome was conceptualized as the onset of the outcome variable, odds ratios (ORs) were used and converted to correlations. In these instances, a study either had a measurement of a diagnosis at baseline and follow-up [in which case, we were interested in participants who had no diagnosis at baseline and either had a diagnosis at follow-up (target group) or remained diagnosis free at follow-up (reference category)]. In other studies that utilized ORs, all participants were diagnosis free at Time 1, and hence follow-up scores reflected onset of the construct of interest. If insufficient information was reported in papers to calculate an effect size between eating pathology and depression, the corresponding author was contacted and asked to provide the correlations between and within the eating pathology and depression constructs of interest. If these coefficients were not reported, and the authors of a study were unable to provide them, the paper (12 in total presented in Figure 1) was excluded from the meta-analysis. We were not able to obtain estimates of effects of eating pathology on depression and depression on eating pathology for all studies, and hence unable to test a bi-directional model using the entire sample. Meta-analysis and meta-regression was therefore performed on two separate unidirectional models where a) eating pathology predicted depression and b) depression predicted eating pathology.

4.2. Analytic Decisions for Meta-Analysis

Meta-analysis was conducted on r values. Effect sizes and relevant demographics were extracted from each paper and tabled in SPSS (see Table 2). Although studies varied in the effect size metric used, all effects were converted to r values for the present analyses as an easily interpretable metric with good statistical properties (73). In instances where non-significant effect sizes were unavailable (from papers or contact with authors), r values were set to 0 (74). A multilevel modeling (MLM) approach was used to derive an estimate of average effect size across all studies and estimates, whilst controlling for non-independence due to multiples estimates within the same study (75). Random-effects modeling was undertaken within the MLM framework to assess the extent to which effect sizes were heterogeneous across papers. Intra-class correlations (ICCs) were used to quantify the extent of heterogeneity, and ICC values greater than .25 (indicating that at least 25% of the variance in effect sizes occurred across papers) were followed up with meta-regression analyses that examined the moderated effect of participants' age, eating pathology type (i.e., overall disordered eating symptoms versus bulimic symptoms versus binge-eating symptoms versus an eating disorder diagnosis), the interaction between participants' age and eating pathology type, eating pathology and depression assessment type (i.e., continuous versus categorical), time-lag assessment interval and number of waves of assessment.

---- Include Table 2 about here -----

5. RESULTS

5.1. Synthesis of Results for Meta-Analyses

5.1.1. Eating Pathology Predicting Depression

Overall, the effect of eating pathology on depression showed a significant prediction $r = 0.13$ (95% CI: .09 to .17), $p < 0.001$. The effect sizes did not reliably differ across studies;

$t = 1.26, p = 0.21$. However, since the amount of heterogeneity as assessed by the ICC (ICC = .66) was substantial, the non-significant result was likely due to low sample size/power, and we therefore proceeded with meta-regression. Results of the meta-regression analyses are presented in Table 3. Results showed that neither the effect of age, time-lag, or number of waves of assessment moderated the relationship when eating pathology predicted depression. Similarly, there was no statistical difference between studies that assessed eating pathology as a categorical versus a continuous measure. By contrast, when eating pathology predicted depression, studies that operationalized depression as a diagnosis of a depressive disorder showed significantly stronger effects sizes. Regarding the moderated effect of eating pathology type, when all types (i.e., overall disordered eating, bulimic symptoms, binge-eating symptoms and a diagnosis of an eating disorder) were entered into the model, results showed that an eating disorder diagnosis exerted a significantly stronger effect relative to other types of eating pathology. It was also found that overall disordered eating symptoms exerted a significantly stronger effect on depression relative to bulimic symptoms and binge-eating symptoms. The analyses that examined the interaction terms for age $\times$ eating pathology type revealed that relative to older participants, the effects of age $\times$ eating disorder diagnosis and age $\times$ bulimic symptoms were significantly greater for younger participants.

5.1.2. Depression Predicting Eating Pathology

Overall, the effect for depression predicting eating pathology was significant, $r = 0.16$ (95% CI: .10 to .22), $p < .001$. There was evidence of heterogeneity in this effect size, $t = 2.77, p = 0.006$. The amount of heterogeneity measured by the ICC was 85%. As such, a meta-regression analysis was conducted. Results showed that neither the effect of age, time-lag, or number of waves of assessment moderated the relationship when depression predicted eating pathology. Regarding the effect of assessment type, results indicated no significant difference when the depression predictor variable was assessed as a categorical opposed to a

continuous variable. However, studies that operationalized eating pathology as a categorical outcome (i.e., the “presence”/“absence” of symptoms or an eating disorder diagnosis) showed a significantly stronger effect relative to studies that measured eating pathology as a continuous outcome. Regarding the moderated effect of eating pathology type, the adjusted difference indicated that eating disorder diagnosis had a significantly greater influence on depression relative to the three other types of eating pathology. The analyses that examined the interaction terms for age $\times$ eating pathology type revealed one significant finding; the effect of bulimic symptoms on depression was significantly stronger for older participants.

---- Include Table 3 about here -----

6. DISCUSSION

6.1. Summary of Findings

To our knowledge this is the first systematic review and meta-analysis of the relationship between eating pathology and depression since Stice (19) and Jacobi et al.'s (21) research. The aim of this study was to synthesize the findings of a disparate body of longitudinal literature to determine the size and direction of effects between eating pathology and depression. Meta-analysis summary effects on 30 studies showed that eating pathology significantly predicted depression and depression significantly predicted eating pathology. This is therefore the first meta-analysis to show that the eating pathology - depression relationship is bi-directional.

The results of our meta-analysis provide initial support for the affect-regulation model which proposes that individuals who experience depression develop eating pathology because eating pathology is thought to be a mechanism that reduces negative mood (17, 18). Results also provide initial support for the view that eating pathology is a risk factor for depression;

potentially because failure to control eating behaviors (e.g., dietary restraint and/or binge-eating), and in turn, failure to achieve an idealized physical ideal, as well possible effects of caloric deprivation, might generate depression (16, 46).

The effect sizes for eating pathology predicting depression and depression predicting eating pathology were both small. These findings are consistent with the results of the meta-analysis conducted by Stice (19) who showed that the estimated effect of mood difficulties on eating pathology was $r_m = 0.07$. Indeed, these small effects raise the possibility that the relationship between eating pathology and depression might be subserved by shared risk factors such as genetic, environmental (76) and psychological factors (27-29). Shared risk factors might predispose individuals towards developing both eating pathology and depression, and initial symptoms of each construct might amplify the other in a bi-directional feedback loop. Future research assessing the causal relationship between eating pathology and depression would therefore benefit from examining the influence of shared risk factors on both constructs.

6.2. Moderated Effect of Participant Age and Age $\times$ Eating Pathology Type

A post hoc meta-regression that assessed participant age as a moderator of the effect between eating pathology and depression revealed a null finding. However, the results from this meta-analysis indicated quantitative differences as a function of age $\times$ eating pathology type, suggesting that age does indeed influence the eating pathology - depression relationship. Specifically, the results showed that of the different types of eating pathology that were assessed, when eating pathology predicted depression, the interaction between age $\times$ bulimic symptoms and age $\times$ eating disorder diagnosis was significantly stronger for *younger* participants. By contrast, the results found that when depression predicted eating pathology, the interaction between age $\times$ bulimic symptoms was significantly stronger for *older*

participants. The baseline mean age of participants in this meta-analysis was approximately 16 years and the average time-lag between points of assessment was two years. Accordingly, on average, participants would have been 18 years of age or older at assessment points subsequent to baseline.

Past research has found that overall disordered eating symptoms increase from 14 years of age and peak at 17 years of age whereas binge-eating peaks around 20 years of age (14). Other research (77) has found that the age range of onset for meeting criteria for bulimia nervosa is between 20 to 24 years of age, higher than that of anorexia nervosa, which is 15 to 19 years of age. Similarly, research has shown that the prevalence of depressive symptoms increases from childhood to early adolescence (78). Hence, extant research indicates that different facets of eating pathology peak at different ages. It is currently unclear why the results of this meta-analysis show that the moderated effect of age $\times$ eating pathology type differed according to whether eating pathology predicted depression or depression predicted eating pathology. These results are further complicated by the heterogeneous trajectory of the development and maintenance of eating pathology within individuals; for example, research has shown a high degree of diagnostic cross-over between anorexia nervosa and bulimia nervosa such that an individual with anorexia nervosa will likely develop symptoms of bulimia nervosa at some stage along the pathogenesis of the disorder (79, 80). Thus, the course of eating pathology symptoms might vary both within and between individuals. This heterogeneity in symptom trajectory and the possible interactions that such differences might have with age and gender arguably impact the risk that eating pathology confers to depression and vice versa.

Gender differences have been shown to influence the trajectory of disordered eating such that boys and girls aged nine to 11 years have been shown to have similar levels of disordered eating but girls' symptom level increased at around 14 years of age while

boys remained stable (81). Research has also shown that the trajectory of depressive symptoms varies as a function of gender and age (78). For example, a meta-analysis (78) of the relationship between age, gender and depression showed that the level of depressive symptoms between boys and girls was similar until the age of 12 years, after which girls experienced a significant increase in symptoms, peaking at age 15 years. By contrast, boys' level of depressive symptoms remained constant regardless of age. Indeed, the relationship between eating pathology and depression in boys and girls has been shown to increase from seven years to 12 years of age and plateau from 12 to 16 years for boys but increase for girls. Hence, while the current meta-analysis provides preliminary results to suggest that the interaction between age $\times$ eating pathology type influences the effect of eating pathology on depression and vice versa, further research is required to understand how age and gender interact with eating pathology and depression.

6.3. Moderated Effect of Eating Pathology Assessment and Type

Another finding of our review was that the effect of eating pathology on depression and vice versa was significantly greater when the outcome measure was operationalized as a categorical, rather than a continuous, variable. This suggests that the effect of eating pathology on depression and vice versa was stronger for individuals who were either diagnosed with a disorder or where core symptoms were "present" relative to individuals who were classified as having a continuous measure of symptoms.

Results also indicated a significant effect of moderation for eating pathology type. The effect of eating pathology on depression was significantly greater for individuals diagnosed with an eating disorder, and in turn, overall disordered eating symptoms showed a significantly greater effect relative to bulimic symptoms and binge-eating symptoms. The effect of depression on eating pathology was strongest for individuals with an eating disorder. In contrast to our findings, Stice (19) found that the effect of mood difficulties on eating

pathology was significantly larger for studies that assessed bulimic symptoms and binge-eating relative to overall disordered eating. This contradictory finding might reflect the fact that unlike Stice's (19) meta-analysis, our meta-analysis was able to examine the influence of depression on four different types of eating pathology. Further, Stice operationalized mood difficulties as a composite of negative mood states (e.g., negative-affect, depressive symptoms and self-esteem) whereas the present study examined the unique relationship between eating pathology and depression. Future research would benefit from disambiguating which facet(s) of eating pathology confer greater risk for depression and vice versa.

6.4. Moderated Effect of Participant Time-Lag and Number of Waves of Assessment

Regarding the variability around the length of time between assessments, results indicated no significant effect of moderation for any of the models that were tested. One explanation for this null result is that the time-lags that were utilized might not have captured optimal intervals between assessment points; recent research has highlighted that optimal time-lags for cross-panel designs is short (e.g., within the vicinity of months) (82). While not all studies in this review employed a cross-panel design, the average time-lag of studies was around two years. The observed null effect might therefore reflect sub-optimal selection of time-lag intervals in that the choice of time-lags might have meant that eating pathology and depression were assessed at intervals where these variables were stable (e.g., no marked change in symptom levels from baseline to follow-up), and hence, the longitudinal effects that were assessed in this study might not have detected a moderated influence of time-lag. This null finding could also reflect the possibility that there may be an increase in the effect of eating pathology over time, such that initial exposure to depression leads to a worsening of eating pathology, and that eating pathology remains constant for years afterwards and vice versa. If this is true, the present result suggests that the length of time between baseline

assessment and follow-up is irrelevant, and that the effect of one construct on the other holds equally regardless of whether the follow-up is seven days (66) or seven years (9).

It is also possible that the underlying structure of the relationship between eating pathology and depression is non-linear, for example, the relationship might be a threshold-based one, where the influence of the predictor variable on the outcome is negligible until a threshold has been reached for the predictor. Indeed, research has shown that sub-optimal time-lag intervals can underestimate the magnitude of observed effects (83). The time-lags that were utilized in this study were arbitrary since none of the studies provided an empirical justification for their selection of intervals; most likely due to a deficit in empirically based guidelines on the topic (83). Hence, this raises the possibility that the small effect sizes obtained in this meta-analysis might also have been influenced by the relatively long time-lags utilized. Thus, further research into optimal time-lag intervals between eating pathology and depression is required before firm conclusions can be made regarding a) the influence of time-lag and b) the magnitude of the effect of eating pathology on depression relationship and vice versa.

The results of this study showed that the moderated effect of the number of waves of assessment had no significant influence on eating pathology predicting depression or vice versa. This result was surprising given that an increased number of waves of assessment should theoretically be associated with increased power to detect an effect. Again, this null result might reflect the view that the number of waves of assessment utilized by studies in this meta-analysis was arbitrary.

6.5. Limitations and Strengths

The current review comprised a few limitations, which need to be acknowledged. Previous research has shown that dietary restraint had been implicated as a symptom of

eating pathology (12); however, the present study was unable to examine the unique causal relationship between dietary restraint and depression due to insufficient data to test these links. Similarly, this meta-analysis was unable to examine the moderated effect of gender on the relationship between eating pathology and depression due to the paucity of research on males (13, 14). As outlined above, the interaction of age and gender has been shown to influence the relationship between eating pathology and depression, hence future studies would benefit from examining the respective facets of eating pathology and their relationship with depression for both genders. Notwithstanding these limitations, the current review had a number of strengths, which included assessing for the first time, whether the eating pathology – depression relationship is uni- or bi-directional. Another strength of this review was that it was able to examine for the first time, whether a range of modifiers influence the eating pathology – depression relationship, thus providing insight into factors that potentially amplify the effects of eating pathology on depression and vice versa.

6.6. Future Directions

This review has highlighted a dearth of research for studies that have assessed uni-directional and bi-directional relationships between specific eating pathology types and depression. Future studies that assess evidence for a bi-directional relationship might also benefit from employing cross-panel designs to test for both simultaneous (cross-sectional) and cross-lagged (longitudinal) relationships to ensure valid conclusions are drawn. Cross-lagged statistical procedures enables researchers to assess nonrecursive relationships (i.e., a hypothesized relationship between two or more variables assessed at two or more time points that are thought to be reciprocally causal) and enable researchers to determine which variable(s) in the hypothesized

model is the strongest temporal predictor of the other (84). It has been suggested that a failure to utilize a cross-panel approach when attempting to elucidate the temporal relationship between two constructs precludes valid conclusions from being drawn regarding (i) the direction of effects between two constructs and (ii) whether two constructs are bi-directionally linked (85). Finally, future research into understanding the optimal time-lags between assessment points would help ensure that the conclusions derived from longitudinal studies into the relationship between eating pathology and depression are as accurate as possible.

6.7. Clinical Implications

The results of this meta-analysis underscore the importance of assessing for eating pathology in individuals with depression and vice versa. In addition, the small effect sizes observed in this study raise the possibility that the comorbidity between eating pathology and depression might be subserved by shared risk factors, and prevention/early intervention designed to attenuate symptoms of either construct might benefit from targeting shared risk factors. In addition, the results of this study suggest that etiological models of eating pathology should consider the relative risk that depression confers and vice versa when conceptualizing the direction of effects between symptoms.

6.8. Conclusions

Significant results were observed for the bi-directional effects between eating pathology and depression. Meta-regression indicated considerable heterogeneity between studies such that the interaction between age $\times$ eating pathology type significantly influenced the eating pathology – depression relationship. Results also

indicated that the classification of eating pathology and depression assessment type (categorical/continuous) significantly moderated the effects of eating pathology on depression and vice versa, suggesting that the observed longitudinal effects were greater when the outcome variable was categorical. Our findings suggest that prevention and early intervention designed to attenuate symptoms of eating pathology and depression should target both dysfunctional eating attitudes and behaviors as well as symptoms of depression. The results of this analysis also suggest that future research would benefit from examining other factors that confer risk to the development of both eating pathology and depression.

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Nil.

Accepted Article

Table 1
Overview of Each Study Included in the Systematic-Review including Authors, Country of Sample, Sample Size, Mean Age of Participants at Baseline, Number of Assessment Points, Time-Lag, Eating Pathology Measure and Assessment Type, Depression Measure and Assessment Type and Relevant Findings.

Author(s)	Country	Sample Size at Baseline	Mean Age in years at Baseline (SD)	Number of assessment points	Time-Lag (months)	Eating Pathology Measure	Eating Pathology Assessment	Depression Measure	Depression Assessment	Finding
Direction of effects: Eating Pathology predicting Depression										
*Bearman & Stice (2008)	America	428 females = 247	13.6	3	12	EDDI	Continuous	K-SADS	Continuous	Bulimic symptoms → DS for females and males.
Fairweather-Schmidt et al. (2015)	Australia	13,715	45-50	6	33.6	Study-devised scale	Categorical	CESD-10	Continuous	Overall DE symptoms→DS.
Johnson, Cohen, Kasen et al. (2002)	America	717	13.8 (2.6)	3	24	DISC-I	ED AN = 1 BN = 14 BED = 2 EDNOS = 23	DISC-I	MDD, DYS DD = 45	ED →MDD and DYS.

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Author(s)	Country	Sample Size at Baseline	Mean Age in years at Baseline (SD)	Number of assessment points	Time-Lag (months)	Eating Pathology Measure	Eating Pathology Assessment	Depression Measure	Depression Assessment	Finding
*Micali et al. (2015)	England	6,140 females = 3,416	14	3	24	, MRFS, -DAW- BA, DSM-5, Study-devised scales to assess binge-eating, purging, and fasting	ED AN = 153, BN = 16, BED = 30, PD = 26,	SMFQ	Categorical	AN, BN, BED and DP →DS respectively for boys and girls.
Tanofsky-Kraff et al. (2011)	America	118	10.25 (.04)	2	60	EDE, SPEEI	Categorical	CDI	Continuous	Binge-eating → DS.

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Direction of effects: Depression predicting Eating Pathology										
*Abebe et al. (2012)	Norway	3844 females = 1,729	16.3 (0.3)	3	48	BITE	Continuous	DMI	Continuous	DS →bulimic symptoms for males and females.
*Allen et al. (2013)	Australia	1383 females = 703	14.01 (.19)	3	36	ChEDE EDE-Q	Categorical	BDI-Y	Categorical	DS ⇏ binge-eating for males or females.
Berg et al. (2009)	America	324	Range = 18 - 21	2	2	EDI-2 EDDS	Continuous	CES-D	Continuous	DS →binge-eating. DS ⇏ bulimic symptoms.
Bodell et al. (2012)	America	119	6.2 (0.3)	2	12	EDI-B	Continuous	DISC-IV	MDD	MDD →bulimic symptoms.

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Author(s)	Country	Sample Size at Baseline	Mean Age in years at Baseline (SD)	Number of assessment points	Time-Lag (months)	Eating Pathology Measure	Eating Pathology Assessment	Depression Measure	Depression Assessment	Finding
Cooley et al. (2007)	America	339	18	2	20	EDI	Continuous	BDI-II	Continuous	DS →overall DE symptoms.
Dobmeyer & Stein (2003)	America	80	-	2	48	EDI	Continuous	SCID-DSM III- R	Continuous	DS → bulimic symptoms or overall DE symptoms.
Ferriter et al. (2010)	America	134	18.29 0.48	5	12	SCID-DSM III-R	ED AN = 2 BN = 3	BDI	Continuous	DS →ED.
*Gardner et al. (2000)	America	216 females = 104	7.06	3	12	EDI-C EDI-DFT, EDI-B ChEat	Continuous	CDI	Continuous	DS →overall DE symptoms for females and males.
Gilbert & Meyer (2005)	England	197	18.7 (1.67)	2	8	EDI-B, EDI-DFT	Continuous	HADS	Continuous	DS →bulimic symptoms.

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Author(s)	Country	Sample Size at Baseline	Mean Age in years at Baseline (SD)	Number of assessment points	Time-Lag (months)	Eating Pathology Measure	Eating Pathology Assessment	Depression Measure	Depression Assessment	Finding
*Goldschmidt et al. (2012)	America	1827 females = 1,040	12.8 (0.7)	3	60	2 item devised scale	Continuous	DSS	Continuous	DS →binge-eating for males and females.
Hautala et al. (2011)	Finland	722	14.9 (0.6)	2	49.2	SCOFF	Categorical	BDI	Continuous	DS →overall DE symptoms.
Jacobi et al. (2011)	America	215	20.8 (2.6)	4	12	EDE EDE-Q EDI-B, EDI-DFT	ED ED = 6	CES-D	MDD	Current MDD ⇄ ED. Lifetime history of MDD →ED.
Johnson, Cohen, Kotler et al. (2002)	America	726	13.8 (2.6)	3	36	DISC-I	ED AN = 1 BN = 5	DISC-I	MDD, DYS	MDD/DYS →ED. MDD/DYS →binge-eating symptoms.

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Author(s)	Country	Sample Size at Baseline	Mean Age in years at Baseline (SD)	Number of assessment points	Time-Lag (months)	Eating Pathology Measure	Eating Pathology Assessment	Depression Measure	Depression Assessment	Finding
							EDNOS = 12			
*Keel et al. (1997)	America	204 females = 102	5 th and 6 th grade	3	12	EAT-26	Continuous	CDI	Continuous	DS → overall DE symptoms for males or females.
*Le Grange et al. (2014)	Australia	1300 females = 668	11-12	2	24	EDI	Continuous	SMFQ	Continuous	DS → overall DE symptoms for males and females.
Liechty & Lee (2013)	America	14,322	15.9 (1.8)	2	84	2 item study- devised scale	Categorical	CES-D	Categorical	DS → binge-eating and ED diagnosis for males and females.

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Author(s)	Country	Sample Size at Baseline	Mean Age in years at Baseline (SD)	Number of assessment points	Time-Lag (months)	Eating Pathology Measure	Eating Pathology Assessment	Depression Measure	Depression Assessment	Finding
*Pearson et al. (2015)	America	1,906 females = 949	10.86	3	6	EDE-Q	Categorical	CES-D	Continuous	DS →binge-eating symptoms for males and females.
Perez et al. (2004)	America	1709	16.6 (1.2)	3	13	K-SADS	ED BN = 17	K-SADS	MDD and DYS	MDD → BN. DYS →BN.
Salafia & Gondoli (2011)	America	85	10.52 (0.52)	2	12	EDI-B	Continuous	CDI	Continuous	DS →bulimic symptoms.
*Sihvola et al. (2009)	Finland Females = 671	1,318	14.19	2	42	C-SSAGA-A	ED AN = 6 BN = 1 BR ED = 73	C-SSAGA-A	MDD = 71	MDD →ED for males and females.

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Author(s)	Country	Sample Size at Baseline	Mean Age in years at Baseline (SD)	Number of assessment points	Time-Lag (months)	Eating Pathology Measure	Eating Pathology Assessment	Depression Measure	Depression Assessment	Finding
Stice, Presnell, & Spangler (2002)	America	231	14.9	3	10	EDE-Q	Continuous	BDC	Continuous	DS →binge-eating.
Vogeltanz-Holm et al. (2000)	America	709	34.7	2	60	EDE-Q	Categorical	DIS	Lifetime MDD	MDD → binge-eating.
Wertheim et al. (2001)	Australia	435	12.82 – 15.75	2	8	EDI-B, DFT,	Continuous	BDI	Continuous	DS →bulimic symptoms.
*Wichstrøm (2000)	Norway	9,690 females = 4,899	15.56	2	24	EAT-12	Categorical	DSS	Categorical	DS → overall DE for males or females.

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Author(s)	Country	Sample Size at Baseline	Mean Age in years at Baseline (SD)	Number of assessment points	Time-Lag (months)	Eating Pathology Measure	Eating Pathology Assessment	Depression Measure	Depression Assessment	Finding
Direction of effects: Bi-Directional										
Boujut & Gana (2014)	France	359	18.7 (1.3)	3	6	EAT 26	Continuous	BDI	Continuous	Bi-directional relationship between overall DE and DS.
*Ferreiro et al. (2014)	Spain	942 females = 465	10.83 0.75	4	12	ChEAT	Continuous	CDI	Continuous	DS at T1 →overall DE symptoms at T2 for males and females.
*Herpertz-Dahlmann et al. (2014)	Germany	771 females = 420	14.3 (2.0)	2	72	SCOFF	Continuous	CES-DC	Continuous	Overall DE symptoms →DS for males and females.
Hilbert et al. (2013)	Germany	112	10.72 1.48	5	6	ChEDE ChEDE -Q	Categorical	CDI	Continuous	DS →overall DE symptoms.

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Author(s)	Country	Sample Size at Baseline	Mean Age in years at Baseline (SD)	Number of assessment points	Time-Lag (months)	Eating Pathology Measure	Eating Pathology Assessment	Depression Measure	Depression Assessment	Finding
Leung & Steiger (1991)	Canada	543	13-17	2	6	EAT-26	Continuous	8 item self-devised scale	Continuous	Null findings between DS and overall DE.
Mackinnon et al. (2011)	Canada	200	19.86 (3.02)	3	0.25	EDDS-BE	Continuous	PoMS	Continuous	Bi-directional relationship between DS and binge-eating.
Marmorstein et al. (2008)	America	754	11.7 (0.4)	3	36	MEBS	Continuous	DISC-1 SCID-DSM III-R	Continuous	Overall DE at T1 and T2 →DS at T2 and T3 respectively.
Measelle et al. (2006)	America	493	14.48 (0.67)	4	12	EDE	Continuous	K-SADS	Continuous	DS →overall DE symptoms.
Presnell et al. (2009)	America	496	13.5	8	12	EDDI	Continuous	K-SADS	Continuous	Bi-directional relationship between DS and bulimic symptoms.

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Procopio et al. (2006)	America	150	45.19 10.4	2	30	EDI-B	Continuous	BDI	Continuous	Null findings between DS and bulimic symptoms.
Skinner et al. (2012)	America	4798	14.9	3	24	2 item self-devised scale	Categorical	MRFS	Continuous	Bi-directional relationship between DS and binge-eating.
Spoor et al. (2006)	Netherlands	143	19.6	2	12	EDI II-B	Continuous	SCL	Continuous	DS →bulimic symptoms.
Zaider et al. (2002)	America	201	16.3 1.08	2	10	PHQ-A	ED BN = 5 BED = 6 S-ED = 30	PHQ-A	DYS = 20 MDD = 29	MDD and DYS →ED. S-ED symptoms → DYS. S-ED symptoms ↔ MDD.

Table 1

Overview of Each Study Included in the Systematic-Review including Authors, Country of Sample, Sample Size, Mean Age of Participants at Baseline, Number of Assessment Points, Time-Lag, Eating Pathology Measure and Assessment Type, Depression Measure and Assessment Type and Relevant Findings.

Note. * = males and females were assessed; DE = disordered eating; DS = depressive symptoms; BITE = Bulimic Investigatory Test, Edinburgh; ChEDE = Child Eating Disorder Examination; EDE = Eating Disorder Examination; EDE-Q = Eating Disorder Examination-Questionnaire; EDI = Eating Disorder Inventory; EDI-2 = Eating Disorder Inventory-2; EDDS = Eating Disorders Diagnostic Scale; EDI-B = Eating Disorder Inventory-Bulimia Subscale; SCID-DSM-III-R = Structured Clinical Interview for DSM-III-R; EDI-C = Eating Disorder Inventory for Children; EDI-DFT = Drive for Thinness subscale of the EDI; EDI-B = Bulimia subscale of the EDI; ChEat = Children's Eating Attitudes Test; DISC-1 = Diagnostic Interview Schedule for Children; EAT-26 = Eating Attitudes Test-26; K-SADS = Schedule for Affective Disorders and Schizophrenia for School-Age Children; EAT-12 = Eating Attitudes Test-12; EDDI = Eating Disorders Diagnostic Interview; YRBSS = Youth Risk Behavior Surveillance System questionnaire; ChEDE-Q = Eating Disorder Examination-Questionnaire adapted for Children; PHQ-A = Patient Health Questionnaire for Adolescents; SPEEI = Standard Pediatric Eating Episode Interview; EDDS-BE = Binge Eating subscale of the Eating Disorder Diagnostic Scale; C-SSAGA-A = Finnish translation of the adolescent Semi-Structured Assessment for the Genetics of Alcoholism; DMI = Depressive Mood Inventory; BDI-Y = Beck Depression Inventory-Youth; BDI = Beck Depression Inventory; BDI-II = Beck Depression Inventory-II; CES-D = Centre for Epidemiological Studies-Depression; CESD-10 = Centre for Epidemiological Studies-Depression short form; DISC-IV = Diagnostic Interview Schedule for Children-IV; CDI = Children's Depression Inventory; HADS = Hospital Anxiety and Depression Scale; DSS = Depressive Symptoms Scale; SMFQ = Short Mood and Feelings Questionnaire; BDC = Burns Depression Checklist; DAW-BA = Development and Wellbeing Assessment; DIS = Diagnostic Interview Schedule; DSM-5 = Diagnostic and Statistical Manual of Mental Disorders Fifth Edition; DSM-III-R = Diagnostic and Statistical Manual of Mental Disorders, Third Edition- Revised; CES-DC = Centre for Epidemiological Studies Depression for Children; PoMS = Depression subscale of the Profile of Mood States; MRFS = McKnight Risk Factor Survey; SCL = Hopkins Symptom Checklist; MEBS = Minnesota Eating Behavior Survey; MDD = Major Depressive Disorder diagnosis; DYS = Dysthymic Disorder diagnosis; DD = any Depressive Disorder; ED = measured as a diagnosis of an Eating Disorder; Categorical = construct measured as a dichotomized variable where symptoms were "present" or "absent"; Continuous = measured as a continuous variable; AN = Anorexia Nervosa; BN = Bulimia Nervosa; BED = Binge Eating Disorder; ENDOS = Eating Disorder Not Otherwise Specified; BR ED = Broadly defined ED where 2/4 DSM IV criteria for AN or BN were met; S-ED = Sub-threshold BN or BED symptoms; T1 = Time 1; T2 = Time 2; → = significantly predicted; ↗ = did not significantly predict; - = data not reported in study.

Accepted Article

Table 2

Relation of Initial Depressive Scores to Subsequent Change in Eating Pathology Scores (and Vice Versa) Expressed as r Values and Descriptive Statistics for Moderator Variables (Sample Size, Time-Lag, Age, Number of Waves of Assessment and Eating Pathology and Depression Assessment Type)

Authors	Time Point	Sample Size	Time-Lag (months)	Mean Age (Months)	Number of Waves	Eating Pathology Assessment	Depression Assessment	Eating Pathology Type	Effect Size D→EP	Effect Size EP→D
Abebe et al. (2012)	T1→T2	2923	52	17.34	3	Continuous	Continuous	Bulimic	.126	.077
	T2→T3	2890							.074	.079
	T1→T2	2923		17.34		Continuous	Continuous	Bulimic	.053	.084
	T2→T3	2890							.072	.088
Allen et al. (2013)	T1→T2	1383	36	14.00	3	Continuous	Continuous	Overall DE	-.085	-.029
	T2→T3	1383							.098	.109
	T1→T2	1383		14.00		Continuous	Continuous	Overall DE	-.001	.173
	T2→T3	1383							-.124	.139
Bearman et al. (2008)	T1→T2	428	12	13.60	3	Continuous	MDD	Bulimic	-	.082
Boujut et al. (2014)	T1→T2	359	6	18.70	3	Continuous	Continuous	Overall DE	.054	.085
	T2→T3	359							.010	.140
Cooley et al. (2007)	T1→T2	117	20	18.00	2	Continuous	Continuous	Overall DE	.214	-

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Authors	Time Point	Sample Size	Time-Lag (months)	Mean Age (Months)	Number of Waves	Eating Pathology Assessment	Depression Assessment	Eating Pathology Type	Effect Size D→EP	Effect Size EP→D
Ferreiro et al. (2014)	T1→T2	882	12	10.83	4	Continuous	Continuous	Overall DE	.130	.040
	T2→T3	748							.050	.050
	T3→T4	476							.169	.145
Gardner et al. (2000)	T1→T2	189	12	7.06	3	Continuous	Continuous	Overall DE	.144	.083
Goldschmidt et al. (2012)	T1→T2	1827	60	12.80	3	Continuous	Continuous	Binge	.155	.076
	T2→T3	1827							.154	.008
Hautala et al. (2011)	T1→T2	722	49.1	14.90	2	Categorical	Continuous	Overall DE	.502	.457
Herpertz-Dahlmann et al. (2014)	T1→T2	771	72	14.30	4	Continuous	Continuous	Overall DE	.062	.060
	T2→T3	771							.130	.160
	T3→T4	771							.090	.170
Hilbert et al. (2013)	T1→T2	112	6	10.72	5	Categorical	Continuous	Binge	.007	.026
	T2→T3	112						Overall DE	.196	-

Table 2

Relation of Initial Depressive Scores to Subsequent Change in Eating Pathology Scores (and Vice Versa) Expressed as r Values and Descriptive Statistics for Moderator Variables (Sample Size, Time-Lag, Age, Number of Waves of Assessment and Eating Pathology and Depression Assessment Type)

Authors	Time Point	Sample Size	Time-Lag (months)	Mean Age (Months)	Number of Waves	Eating Pathology Assessment	Depression Assessment	Eating Pathology Type	Effect Size D→EP	Effect Size EP→D
Johnson, Cohen, Kotler et al. (2002)	T1→T2	726	24	13.80	3	Categorical	MDD, DYS	Binge	.580	-
	T1→T2	726	24	13.80	3	ED diagnosis	MDD, DYS	AN, BN, EDNOS	.510	-
Johnson, Cohen, Kasen et al. (2002)	T1→T2	726	24	13.80	3	ED diagnosis	MDD, DYS	AN, BN, EDNOS	-	.370
Leung et al. (1991)	T1→T2	543	6	17.34	2	Continuous	Continuous	Overall DE	.049	.065
Liechty et al. (2013)	T1→T2	14322	84	15.90	2	Categorical	Continuous	Binge	.290	-
Mackinnon et al. (2011)	T1→T2	200	0.25	19.86	3	Continuous	Continuous	Overall DE	.028	.200
	T2→T3	200							.090	.080
Marmorstein et al. (2008)	T1→T2	715	36	14.8	3	Continuous	Continuous	Overall DE	.040	.130
	T2→T3	644		18					.040	.090

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Authors	Time Point	Sample Size	Time-Lag (months)	Mean Age (Months)	Number of Waves	Eating Pathology Assessment	Depression Assessment	Eating Pathology Type	Effect Size D→EP	Effect Size EP→D
Micali et al. (2015)	T1→T2	5069	24	16	3	ED diagnosis	Categorical	AN	-	.09
	T1→T2	5069	24	16	3	ED diagnosis	Categorical	BN	-	.319
	T1→T2	5069	24	16	3	ED diagnosis	Categorical	BED	-	.188
	T1→T2	5069	24	16	3	ED diagnosis	Categorical	PD	-	.251
Pearson et al. (2015)	T1→T2	1906	6	10.86	3	Continuous	Continuous	Binge	.121	.064
	T2→T3	1906							.108	.018
Perez et al. (2004)	T1→T2	1507	13	16.60	3	ED diagnosis	DYS	BN	.090	-
	T2→T3	941					MDD		.030	-
Presnell et al. (2009)	T1→T2	496	12	13.50	8	Continuous	Continuous	Bulimic	.080	.110
	T2→T3	496							.090	.080
	T3→T4	496							.140	.150
	T4→T5	496							.130	.090
	T5→T6	496							.120	.130

Table 2

Relation of Initial Depressive Scores to Subsequent Change in Eating Pathology Scores (and Vice Versa) Expressed as r Values and Descriptive Statistics for Moderator Variables (Sample Size, Time-Lag, Age, Number of Waves of Assessment and Eating Pathology and Depression Assessment Type)

Authors	Time Point	Sample Size	Time-Lag (months)	Mean Age (Months)	Number of Waves	Eating Pathology Assessment	Depression Assessment	Eating Pathology Type	Effect Size D→EP	Effect Size EP→D
Procopio et al. (2006)	T6→T7	496							.140	.080
	T7→T8	496							.030	.080
	T1→T2	150	30	45.19	2	Continuous	Continuous	Bulimic	.150	-.010
Sihvola et al. (2009)	T1→T2	1,318	42	14.19	2	ED Diagnosis	MDD	AN, BN, AED	.392	-
Spoor et al. (2006)	T1→T2	143	12	19.60	2	Continuous	Continuous	Bulimic	.172	.100
Stice, Presnell, & Spangler (2002)	T1→T2	231	10	14.90	3	Continuous	Continuous	Binge	.190	-
Tanofsky-Kraff et al. (2011)	T1→T2	118	60	10.25	2	Continuous	Continuous	Binge	-	.185
Vogeltanz-Holm et al. (2000)	T1→T2	709	60	34.70	2	Categorical	MDD	Binge	.030	-
Wertheim et al. (2001)	T1→T2	435	8	14.09	2	Continuous	Continuous	Bulimic	.155	-
Wichstrøm (2000)	T1→T2	7751	24	15.56	4	Continuous	Continuous	Overall DE	.080	.119
	T2→T3	7751							.095	.090
	T3→T4	7751							.101	.084

Table 2
Relation of Initial Depressive Scores to Subsequent Change in Eating Pathology Scores (and Vice Versa) Expressed as r Values and Descriptive Statistics for Moderator Variables (Sample Size, Time-Lag, Age, Number of Waves of Assessment and Eating Pathology and Depression Assessment Type)

Authors	Time Point	Sample Size	Time-Lag (months)	Mean Age (Months)	Number of Waves	Eating Pathology Assessment	Depression Assessment	Eating Pathology Type	Effect Size D→EP	Effect Size EP→D
Zaider et al. (2002)	T1→T2	201	10	16.30	2	ED diagnosis	MDD DYS	AED	.390	.150
	T1→T2	201							.560	.380

Note. A dash indicates a missing effect size. EP = eating pathology; D = depression; ED diagnosis = diagnosis of any type of eating disorder; Binge = binge eating symptoms; Bulimic = bulimic symptoms; AN = anorexia nervosa diagnosis; BN = bulimia nervosa diagnosis; EDNOS = Eating Disorder Not Otherwise Specified diagnosis; AED = any eating disorder diagnosis; BED = Binge Eating Disorder diagnosis; PD = Purging Disorder diagnosis; DE = disordered eating; MDD = Major Depressive Disorder diagnosis; DYS = Dysthymic Disorder diagnosis; DS = depressive symptoms; Continuous = measured as a continuous variable; Categorical = measured as a dichotomized variable where symptoms were “present” or “absent”; *sr* = part correlation; T1 = time point 1; T2 = time point 2; T3 = time point 3; T4 = time point 4; T5 = time point 5; T6 = time point 6; T7 = time point 7; T8 = time point 8.

Table 3

Results of Meta-Regression Analyses for Eating Pathology Predicting Depression and Depression Predicting Eating Pathology

	Moderator	b weight	SE	t-value	p (two tailed)
Direction of Effects					
Eating Pathology Predicting Depression	Lag	.001	.001	.792	.428
	Age	-.002	.002	-1.225	.220
	Wave	-.032	.017	-1.878	.060
	Eating Pathology Assessment Type (Categorical Versus Continuous)*	.194	.072	2.699	.007
	Depression Assessment Type (Categorical Versus Continuous)*	.173	.100	1.728	.084
	Bulimic Symptoms Versus Overall Disordered Eating Symptoms*	-.014	.05	-.283	.777
	Binge-Eating Versus Overall Disordered Eating Symptoms*	-.059	.087	-.671	.502
	Eating Disorder Diagnosis Versus Overall Disordered Eating Symptoms*	.223	.112	2.002	.045
	Age × Bulimic Symptoms	.011	.005	2.119	.034
	Age × Binge-Eating Symptoms	.010	.008	1.286	.198
	Age × Eating Disorder Diagnosis	-.102	.055	-1.862	.063
Depression Predicting Eating Pathology	Lag	.001	.001	.724	.469
	Age	-.003	.002	-1.318	.188

Table 3
Results of Meta-Regression Analyses for Eating Pathology Predicting Depression and Depression Predicting Eating Pathology

Moderator	b weight	SE	t-value	p (two tailed)
Wave	-.016	.013	-1.247	.212
Eating Pathology Assessment Type (categorical/continuous)*	.160	.195	.823	.411
Depression Assessment Type (categorical/continuous)*	.128	.031	4.185	<.001
Bulimic Symptoms Versus Overall Disordered Eating Symptoms*	-.033	.013	-2.585	.010
Binge-Eating Versus Overall Disordered Eating Symptoms*	-.060	.023	-2.618	.009
Eating Disorder Diagnosis Versus Overall Disordered Eating Symptoms*	.132	.033	3.977	<.001
Age × Bulimic Symptoms	-.006	.003	-2.388	.017
Age × Binge-Eating Symptoms	-.013	.014	-.895	.371
Age × Eating Disorder Diagnosis	-.060	.008	-7.304	<.001

Note. b weight = unstandardized beta weight; SE = standard error; Wave = The number of points of assessment; Lag = the length of time (months) between points of assessment; * = For these models, the reference group (the first factor entered in the analysis) was coded as 0 and the comparison group (the second factor entered in the analysis) was coded as 1. The coefficients for these models indicate the difference in effect sizes between the two groups. A positive coefficient indicates that the reference category exhibited a stronger effect relative to the comparison group and a negative coefficient indicates that the comparison group exhibited a stronger effect relative to the reference group.

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Submission ID: IJED-15-0271

Title: A Systematic Review and Meta-Analysis On The Longitudinal Relationship Between Eating Pathology and Depression

Responses to the reviewers

The present version of the manuscript includes modifications in response to the concerns raised by the reviewers. For the sake of clarity and brevity, we refer to each comment in the same order as listed by the reviewers. We thank the reviewers and Editor for their balanced and detailed feedback, and feel that the revised manuscript is greatly improved as a result.

Editor's Comments:

Comment 1: The text on pp. 9-12 that describe the data prior to the meta-analysis is long and difficult to wade through. I see the value in the information provided, but given that the manuscript is quite long, I would encourage you to add a table with the information in this text. A table would allow for a more concise presentation of the information and a shorter manuscript.

Reply 1: We thank the reviewer for this suggestion. The text on pp. 9-12 has been omitted from the text and is now included in Table 1. We hope that this will make the review easier to read.

Comment 2: The title of Table 1 states that the table entries are grouped according to the direction of effects model examined, but it looked to me as if the studies were grouped alphabetically. I think it would be very useful to add subheadings and to group the studies by the direction of effects examined, rather than just alphabetically. It is possible that I missed some sort of grouping in my read - if so, please feel free to clarify in your revision.

Reply 2: Studies have now been grouped according to their direction of effects and then alphabetised thereafter. Studies have been reordered so that those that assessed eating pathology predicting depression are presented first, depression predicting eating pathology second, and bi-directional studies are presented third.

Submission ID: IJED-15-0271

Systematic Review and Meta-Analysis

Reviewer #1 Comments:

Comment 1: In the first paragraph the authors suggest that the reason for the comorbidity between depression and eating disorders is unknown, but one reason is the shared genetic risk factors both between depression and bulimia nervosa, and depression and anorexia nervosa. This information should be included in the paragraph. While discussion on this issue and other factors influencing comorbidity are mentioned in the Discussion, it would be better to place this in the Introduction, as it was not clear to me how the results of this study suggested a multiplicity of factors explaining comorbidity.

Reply 1: We have now included potential reasons for the high comorbidity between eating pathology and depression in the Introduction on pp. 7-8:

“Research has shown that biological and sociocultural influences (27) as well as psychological factors such as impulsivity (28, 29) and body dissatisfaction (30) are implicated in the etiology of both eating pathology and depression. Thus, it is possible that shared risk factors predispose individuals towards developing both eating pathology and depression, and that once symptoms of each construct are experienced, they amplify the other in a bi-directional feedback loop.”.

Comment 2: On page 5 the differentiation between “bulimic symptoms” and “binge eating” is not immediately clear – it is unclear whether the former refers to purging and binge eating or simply purging, and this needs to be clarified.

Reply 2: We appreciate the reviewer’s comment, and have now provided a definition of bulimic symptoms on p. 5:

“Regarding study-level effect modifiers, no meta-analysis has investigated whether eating pathology type [e.g., overall disordered eating symptoms versus bulimic symptoms (i.e., binge-eating combined with compensatory behaviors such as purging) versus binge-eating symptoms versus an eating disorder diagnosis] is a factor in determining the magnitude of effect sizes on depression and vice versa despite considerable diversity in how eating pathology has been assessed (24-26)”.

Comment 3: Also, the use of the term time lag could be clarified – between initial and final measures?

Reply 3: We have now provided a definition for time lag on p. 6:

“Regarding the effect of time-lag (i.e., the length of time between baseline and follow-up assessment)...”

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Systematic Review and Meta-Analysis

Comment 4: Given the lack of evidence to suggest twins differ from the general population, omission of these studies seems questionable. Or else the authors may need to cite evidence showing them to be different to justify this exclusion. Otherwise removal of a variety of groups could be justified on the basis of subjective interpretation of the literature.

Reply 4: Thank you for pointing this out. We have now included studies that assessed twins into the systematic review and meta-analysis and have deleted the section that specified that twin studies were omitted from the review on p. 9:

“A study was excluded from the review if it assessed specific groups (e.g., sports groups) because the mechanism(s) that link eating pathology and depression might be quantitatively different for selected populations relative to community samples”.

Comment 5: Figure 1 does not need to be referenced twice in this section, remove the first mention.

Reply 5: We apologise for duplicating this information. The first mention of Figure 1 has now been removed.

Comment 6: The exclusion of negative affect studies is understandable but does form an interesting sub group given that negative affect is likely to be a higher order construct that explains comorbidity between eating disorders and depression. It would therefore be of interest to know how many studies were included in this excluded group.

Reply 6: The 11 studies that assessed negative-affect that were not included in the initial submission are now listed in Figure 1.

Comment 7: Given that specific groups were excluded (page 7) and only one study of a clinical group (anorexia nervosa) was included in the meta-analysis, with the remainder being community samples, it does appear that this study (47) should be excluded from the meta-analysis.

Reply 7: Study 47 has now been omitted from the paper.

Comment 8: Given that the number of waves is associated with more power and stability of results, I wonder if N waves should also be examined as a moderator (e.g., 2 versus >2)?

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Systematic Review and Meta-Analysis

Reply 8: We thank the reviewer for this important suggestion. The number of waves of assessment has now been included as a moderator in the meta-regression. This variable was treated as a continuous measure. Interestingly, the number of waves of assessment showed no significant effect of moderation.

Comment 9: Page 8: concerning those studies that examined the same longitudinal data set, I wonder if this information is better suited to being a footnote for Figure 1 - and then perhaps just bold which of these included?

Reply 9: We are thankful for this recommendation and have tried putting this information in a footnote and bolding the studies that were included as suggested by reviewer 1, however, we think that the current presentation of this information (as presented on p. 10) is easier/simpler for the reader to understand:

“Of the 98 studies that were retrieved for close reading, the following assessed the same longitudinal dataset: a) (34-36); b) (17, 37, 38); c) (39, 40); d) (41-43); e) (44-47); f) (48-50); g) (20, 51). The following studies reported the greater number of waves of data regarding the relationship between eating pathology and depression and were therefore selected for the review: (17, 20, 36, 40, 43, 44, 49)”.

Comment 10: Page 8: 40 studies are listed in Table 1 but only 20 are referred to in the “study characteristics” section in terms of numbered studies. Please reconcile this information. Also, it is not really accurate to say that the majority of studies were conducted in America (10%) – actually the majority were conducted outside of America (90%).

Reply 10: The editor has suggested that we synthesise the information on pp. 9-12 in a Table 1 (which we have done), therefore this information in-text has been omitted.

Comment 11: Page 9: I don't think listing of all self-report questionnaires measuring ED are required – perhaps just mention the most commonly used one, the percentage of studies it is used in, and reference the questionnaire.

Reply 11: The editor has suggested that we synthesise the information on pp. 9-12 in a Table 1 (which we have done), therefore this information in-text has been omitted.

Comment 12: Table 1: it would be informative to notate studies indicating if they included female only or male/females, and I think the final column could be omitted if the column named “direction of effects” had more specific acronyms for DE (i.e., bulimic symptoms, DE) and relevance to males and females could also be noted in this column.

Reply 12: Table 1 now provides a description of all the studies that assessed males and females and their findings as a function of gender. The column labelled “ direction of effects” now specifies the type of eating pathology that was assessed.

Comment 13: The last sentence on page 17 (end of first paragraph in section 6.3) did not make sense to me. How can adolescents already having DS be at risk for DS?

Reply 13: We apologise for this confusion. This sentence has been omitted as the new results did not support this conclusion. (The original statement that we made that “DS was a risk factor for DS” was a typo – the second “DS” should have been “DE”).

Comment 14: I would like to see more contextualising and explanation from the existent literature with respect to the age as a moderator finding.

Reply 14: Once we included the number of waves as a moderator, age no longer had a significant effect of moderation, however the interaction of age*eating pathology symptom did have an effect. In the results section, on p. 12 of the review, when eating pathology predicted depression, we reported the following: *“The analyses that examined the interaction terms for age × eating pathology type revealed that relative to older participants, the effects of age × eating disorder diagnosis and age × bulimic symptoms were significantly greater for younger participants”*.

On p. 13 of the review, when depression predicted eating pathology, we reported the following: *“The analyses that examined the interaction terms for age × eating pathology type revealed one significant finding; the effect of bulimic symptoms on depression was significantly stronger for older participants”*.

In the Discussion section, these results have been contextualised in relation to extant research on pp. 14-16.

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Systematic Review and Meta-Analysis

Comment 15: Given the lack of power to examine dietary restraint, I wonder if this should be just stated up front and excluded from further analyses?

Reply 15: Thank you for this suggestion. Studies that assessed dietary restraint have now been omitted from the paper and an explanation for this omission is provided (low power):

“Only seven studies (9, 12, 14, 40, 52-54) included in the review assessed dietary restraint. We were only able to obtain data for dietary restraint predicting depression for six of these studies (9, 12, 14, 40, 52, 53) and only two studies (52, 54) assessed whether depression predicted dietary restraint. Hence, due to low power, the effect of dietary restraint was not examined in this review”.

Reviewer #2 Comments:

Comment 1a: The paper claims to address “disordered eating” and “depressive symptoms”. This is a crucial methodological point that needs more clarification in the several parts of the paper and also in the analyses. When reading the paper, the focus seems to be on DE and DS as continuous outcomes. This probably makes sense, since the number of longitudinal studies using ED (diagnoses) and depression as categorical outcomes will be much smaller. On the other hand, the authors aim to discriminate between DE and DE assessed as continuous vs. categorical measures as part of their moderator analyses and also use the term “eating disorders” as part of their search strategy which I found somewhat confusing. I don’t think it is clear enough whether categorical DE outcomes in the current paper do include diagnoses of ED and depression or other categorical outcomes of DE (e.g., DE present/absent yes/no). The distinction between dichotomous outcomes of DE and diagnoses however, seems an important one since risk factors for these outcomes might be different.

Reply 1a: We thank the reviewer for this suggestion. Table 1 now lists whether a study assessed eating pathology or depression as either i) a continuous measure, ii) a categorical measure (i.e., symptoms present/absent) and iii) a diagnosis (i.e., and ED or a depressive disorder).

Comment 1b: I also think that diagnoses (as categorical outcomes) should be kept separate from other DE categorical outcomes in the analyses (or addressed specifically by the

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Systematic Review and Meta-Analysis

moderator analyses to examine potential differences between these conditions) since they represent different clinical conditions.

Reply 1b: The moderator “eating pathology type” now includes a separate category for an ED diagnosis. Thus, the moderator “eating pathology type” now has four levels; overall disordered eating, bulimic symptoms, binge-eating symptoms, and an eating disorder diagnosis. We have now also been able to assess whether the effect of eating pathology on depression (and vice versa) differed between studies that assessed symptoms versus an ED diagnosis.

Comment 1c: This would however, lead to another important point: If categorical outcomes in the present review include diagnoses, one would want to know what the exact number of cases in these studies was.

Reply 1c: We thank the review for this suggestion. For the studies that assessed a diagnosis of an eating disorder, the number of cases per disorder are now included in Table 1.

Comment 1d: Since ED are disorders of low prevalence, studies with very few cases (e.g., below N=10) might be underpowered and results from these studies should be considered with caution (meaning they should not be mixed with adequately powered studies). For example, the Ivarsson et al. 2000 study included AN as outcomes – how many cases? Enough to predict meaningful associations?

Reply 1d: Of the studies that have assessed an ED diagnosis that are now included in the meta-analysis, no study has an N below 10. The Ivarsson et al. (2000) study has been omitted from the review as per Reviewer #1’s suggestion (to delete the Ivarsson et al. paper as it was the only paper to assess a clinical group).

Comment 1e: Finally, if ED diagnoses were included, in addition to the N of cases the reader would want to know what kind of diagnoses were found (AN, BN, BED, OSFED/EDNOS) and the results should be presented by diagnostic group in addition to the overall results (i.e., categories of the moderator variable “type of DE” should be changed).

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Systematic Review and Meta-Analysis

Reply 1e: Thank you. The N of cases of each ED (where available) are now presented in Table 1. Due to a lack of statistical power, we were unfortunately unable to examine the effect of diagnostic group (e.g., BN, AN, BED) as a moderator variable. We have now also included (where available) the number of cases of individuals with a depression diagnosis in Table 1.

Comment 2a: Apart from the meta-analysis by Stice et al., (2002) there is another meta-analysis (Jacobi et al. 2004; update Jacobi & Fittig, 2010) that is missing in the present review.

Reply 2a: The review by Jacobi et al. (2004) has now been included in the meta-analysis on p. 5:

“The second review by Jacobi and colleagues (21) assessed whether psychiatric morbidity, psychopathology and negative emotionality were a predictor of eating pathology. Their review concluded that these higher order constructs placed individuals at risk for developing an eating disorder. However this second review did not examine any pooled effect-sizes. Further, the review was only able to include seven longitudinal studies, none of which assessed the specific relationship between eating pathology and depression”.

Comment 2b: In this meta-analysis, several other longitudinal studies addressed the relation between depression/depressive symptoms and eating disorders but are not included in the current meta-analysis. However, unlike the meta-analysis by Stice et al. (2002) these authors included eating disorders (=mostly categorical outcomes) rather than the (much broader) construct of eating pathology (Stice et al., 2002). Examples of missing longitudinal studies in the current meta-analysis are studies by Leon et al., 1999; Killen et al., 1996; The McKnight Investigators, 2003; Moorhead et al., 2003. In the latter (missing) studies, depressive symptoms or depression were sometimes part of a broader construct of negative emotionality or negative affectivity or part of a composite measure of general psychopathology which leads to another major critical point. I find it debatable to exclude studies using negative affect as predictors or outcome measures. Depending on the measure used, the focus is often depression as part of negative affect. In addition, in the theoretical models this distinction is not made. To my knowledge, both Stice and Heatherton and Baumeister discuss negative affect equivalent with depression as precursor (or consequence) of DE. Because both

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constructs are highly correlated, I would not exclude negative affect because one would lose important information.

Reply 2b: We thank the reviewer for this important suggestion. We agree that the theoretical models proposed by Stice and Heatherton and Baumeister discuss negative affect as equivalent with depression as a precursor/consequence of eating pathology. We also agree that there are several longitudinal studies that have incorporated depression in their measure of negative-affect/general psychopathology that we have omitted after careful consideration (e.g., Killen et al., 1996; Leon et al., 1999). Our rationale for omitting that group of studies was that they did not assess the unique relationship between eating pathology and depression; the aim of our meta-analysis was to examine the unique relationship between eating pathology and depression as to date, no study has examined this bi-directional relationship in isolation. Also, negative-affect is a higher order construct (e.g., it encompasses feelings of sadness, anxiety, depression etc) and hence, is a more heterogeneous measure of negative mood relative to depression. We think that the clinical implications of examining the unique relationship between eating pathology and depression link more directly to existing treatment interventions and mental disorder classification systems.

Comment 3: The authors of the current meta-analysis state that “...no meta-analysis has investigated whether DE symptom type (...) is a factor in determining the magnitude of the effect size...”. This is not quite true. The meta-analysis by Jacobi et al. (including its update) does determine effect sizes at least for one direction of the relationship (ED -> DS). Therefore, this statement should be rephrased.

Replay 3: We appreciate this comment. The statement that we had made “no meta-analysis has investigated whether DE symptom type (...) is a factor in determining the magnitude of the effect size...” has now been qualified to reflect the idea that no previous meta-analysis has looked at the exclusive effects of eating pathology on depression (and vice versa) on p. 6 (as presented below). Past research has investigated whether higher order constructs, such a negative-affect, general psychopathology, and/or negative emotionality are risk factors for EDs/mood difficulties, but no study to date has looked exclusively at the bi-directional relationship between eating pathology and depression.

“Regarding study-level effect modifiers, no meta-analysis has investigated whether eating pathology type [e.g., overall disordered eating symptoms versus bulimic symptoms (i.e., binge-eating combined with compensatory behaviors such as purging) versus binge-eating symptoms versus an eating disorder diagnosis] is a factor in determining the magnitude of effect sizes on depression and vice versa despite considerable diversity in how eating pathology has been assessed”.

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Comment 4: In the result section, the authors make a distinction between results related to the systematic review and meta-analytical results. To my opinion, this seems somewhat arbitrary (if not confusing) since a meta-analysis will always also include search strategies and present some descriptive results of included studies and participants. Also, it seems like in sections 3.3.6. and 3.3.7. results are based on box score method counting (counts of significant and non-significant results based on reports of these studies) rather than analyses conducted by the authors themselves. If this was the case, I don't understand why 14 studies were excluded from this more descriptive report because they did not provide more data information (e.g., correlations). This seems like an unnecessary loss of information.

Reply 4: The editor has suggested that we synthesise the information on pp. 9-12 in a Table 1 (which we have done), therefore this information in-text has been omitted. We apologise for any confusion regarding the comment "*I don't understand why 14 studies were excluded from this more descriptive report because they did not provide more data information (e.g., correlations)*". These 14 studies were included in the systematic review box score method of the previous version of this review, however, we might not have made this explicit in the previous version.

Comment 5: The rationale for including dietary restraint as separate outcome (or predictor) category is not clear enough. While it would be interesting to see whether there are differences between the relationship of different constructs of DE and DS I would assume that many of the included measures of DE do also include restraint as construct (e.g., if an EDE total score is used, restraint is one of the subscales). Therefore, the seemingly different outcomes might be collinear. I would recommend to skip restraint or add a table including measures of disordered eating vs. dietary restraint to demonstrate that independent constructs have been addressed.

Reply 5: We appreciate this important comment. The other reviewer of this paper made the comment below (followed by our reply):

Comment 15: Given the lack of power to examine dietary restraint, I wonder if this should be just stated up front and excluded from further analyses?

Reply 15: Thank you for this suggestion. Studies that assessed dietary restraint have now been omitted from the paper and an explanation for this omission is provided (low power):

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“Only seven studies (9, 12, 14, 40, 52-54) included in the review assessed dietary restraint. We were only able to obtain data for dietary restraint predicting depression for six of these studies (9, 12, 14, 40, 52, 53) and only two studies (52, 54) assessed whether depression predicted dietary restraint. Hence, due to low power, the effect of dietary restraint was not examined in this review”.

Comment 6: Apart from studies addressing a unidirectional relationship between DE and DS (and vice versa), studies testing a bidirectional model between DE and DS were also included. The authors specifically state that they included studies in which “...DS and DE were measured at the same time points in time to determine whether each construct predicted the other concurrently” (2.2, p.7). However, to test a bidirectional relationship, the two outcomes would also need to be controlled for each other at baseline, i.e., two models would need to be tested as follows: 1. DS (controlled for DE) predicting both DE and DS; 2. DE (controlled for DS) predicting both DE and DS.

Comment 6: We have now specified that the two outcomes needed to control for each other at baseline on pp. 8-9 (below). All of the studies that were included in the meta-analysis controlled for baseline symptoms of the outcome variable.

“ii) a bi-directional model (e.g., that each construct was assessed at the same points in time to determine if eating pathology, controlling for depression at baseline, predicted both eating pathology and depression at follow up, and if depression, controlling for eating pathology at baseline, predicted both depression and eating pathology at follow up concurrently)”.

Comment 7: I would change the order in which results are presented (DE predicting DS first) throughout the paper since this is more consistent with the title of the paper.

Reply 7: The order in which the results are presented have now been reversed so that eating pathology predicting depression is presented first.

Comment 8: When describing effects of studies (sections 3.3) the total number of studies (which will vary from subsection to subsection) should always be added (e.g., of the 11

studies that examined whether....seven found....). In some sections, this information is missing.

Reply 8: The editor has suggested that we synthesis the information on pp. 9-12 in a Table 1 (which we have done), therefore this information in-text has been omitted.

Comment 9: In section 4.1. the authors state that *“For inclusion in the meta-analysis, when the outcome was continuous, partial correlations (controlling for T1 scores on the outcome variable) were utilized so that the effect reflectedIn cases where the outcome was conceptualized as the onset of the outcome variables, odds ratios were used.”* The latter sentence does not really fit with the previous one. Information on whether and how T1 scores were controlled for is missing here and should be added.

Reply 9: We have now clarified on p. 10 that for studies that presented odds ratios, these odds ratios were converted to correlations. We have now described that all studies included in the meta-analysis controlled for initial symptoms of the outcome variable.

“In cases where the outcome was conceptualized as the onset of the outcome variable, odds ratios (ORs) were used and converted to correlations. In these instances, a study either had a measurement of a diagnosis at baseline and follow-up [in which case, we were interested in participants who had no diagnosis at baseline and either had a diagnosis at follow-up (target group) or remained diagnosis free at follow-up (reference category)]. In other studies that utilized ORs, all participants were diagnosis free at Time 1, and hence follow-up scores reflected onset of the construct of interest”.

Comment 10: Section 4.2 could be more precise in terms of the separate analyses for unidirectional models and bidirectional models (statistical methods for the latter are missing or mixed with the further).

Reply 10: We thank the reviewer for this suggestion. We have now clarified on pp. 10-11 that we tested two uni-directional models (eating pathology on depression and depression on eating pathology). For the studies that assessed a bi-directional model, we were able to obtain estimates from the same sample of the effect of eating pathology on depression and depression on eating pathology. However, for the group of studies that assessed a uni-

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directional relationship, we were not able to obtain estimates of both direction of effects for all studies. Hence, we were unable to test a bi-directional model for the entire sample and therefore opted to test two uni-directional models.

“Because we were not able to obtain estimates of effects of eating pathology on depression and depression on eating pathology for all studies, and hence unable to test a bi-directional model using the entire sample, meta-analysis and meta-regression was performed on two separate uni-directional models where a) eating pathology predicted depression and b) depression predicted eating pathology”.

We believe we have addressed the issues raised by the reviewers as comprehensively as possible. We thank them for their suggestions and believe that attention to their concerns has improved the quality of this work. We hope that our manuscript now meets the required standards to be published in the IJED.

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