REVIEW

A Systematic Review and Meta-Analysis on the Longitudinal Relationship Between Eating Pathology and Depression

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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.

Resumen

Objetivo: Llevar a cabo un meta-análisis para proporcionar una síntesis cuantitativa de los estudios longitudinales que evaluaron la dirección de los efectos entre la alimentación patológica y la depresión. Un segundo objetivo fue utilizar la meta-regresión para dar cuenta de la heterogeneidad en términos de modificadores del efecto a nivel de estudio.

Método: Una revisión sistemática se llevó a cabo en 42 estudios que evaluaron la relación longitudinal entre la alimentación patológica y la depresión. De estos 42 estudios, se realizaron meta-análisis de multinivel de efectos aleatorios en 30 estudios elegibles.

Resultados: Los resultados del meta-análisis mostraron que la alimentación patológica era un factor de riesgo para depresión (rm=0.13) y que la depresión era un factor de riesgo para la alimentación patológica (rm=0.16). Los análisis de meta-regresión mostraron que estos efectos eran significativamente más fuertes para estudios que operacionalizaban la alimentación patológica como un diagnóstico de trastorno de la conducta alimentaria versus síntomas de alimentación patológica, y para los estudios que operacionalizaban la medida respectiva de resultado como una variable categórica (e.g., un diagnóstico de trastorno o cuando los síntomas estaban "presentes"/"ausentes") versus una medida continua. Los resultados mostraron que en relación al tipo de alimentación patológica, el efecto de un diagnóstico de trastorno de la conducta alimentaria y síntomas bulímicos en la depresión era significativamente más fuerte para participantes más jóvenes.

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Discusión: La alimentación patológica y la depresión son factores de riesgo concurrentes uno para el otro, lo que sugiere que la investigación futura se beneficiaría de identificar factores que son etiológicos al desarrollo de ambos constructos. © 2015 Wiley Periodicals, Inc.

Keywords: disordered eating; eating disorders; depressive symptoms; depression; systematic review; meta-analysis; longitudinal

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Introduction

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, this 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] who 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 effect. 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 et al.[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, and therefore 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 to 17 years of age and then increase significantly from 17 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, this 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 toward 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.

Method

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 1,877 papers were retrieved as illustrated in Figure 1.

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 Fig. 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]

Systematic Review Results

Selection of Studies for Systematic Review

Of the 98 studies that were retrieved for close reading, the following assessed the same longitudinal dataset: (a) Refs. 34–36; (b) Refs. 17, 37, 38; (c) Refs. 39, 40; (d) Refs. 41–43; (e) Refs. 44–47; (f) Refs. 48–50; and (g) Refs. 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: Refs. 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. Forty-two studies[8–17,20,22–26,36,40,43,44,49,52–72] (presented in Fig. 1) met selection criteria and were included in the systematic review. Table 1 presents an overview of each study and their relevant findings.

Meta-Analysis

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

FIGURE 1. Flow diagram of eligibility screening of studies. [Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.]

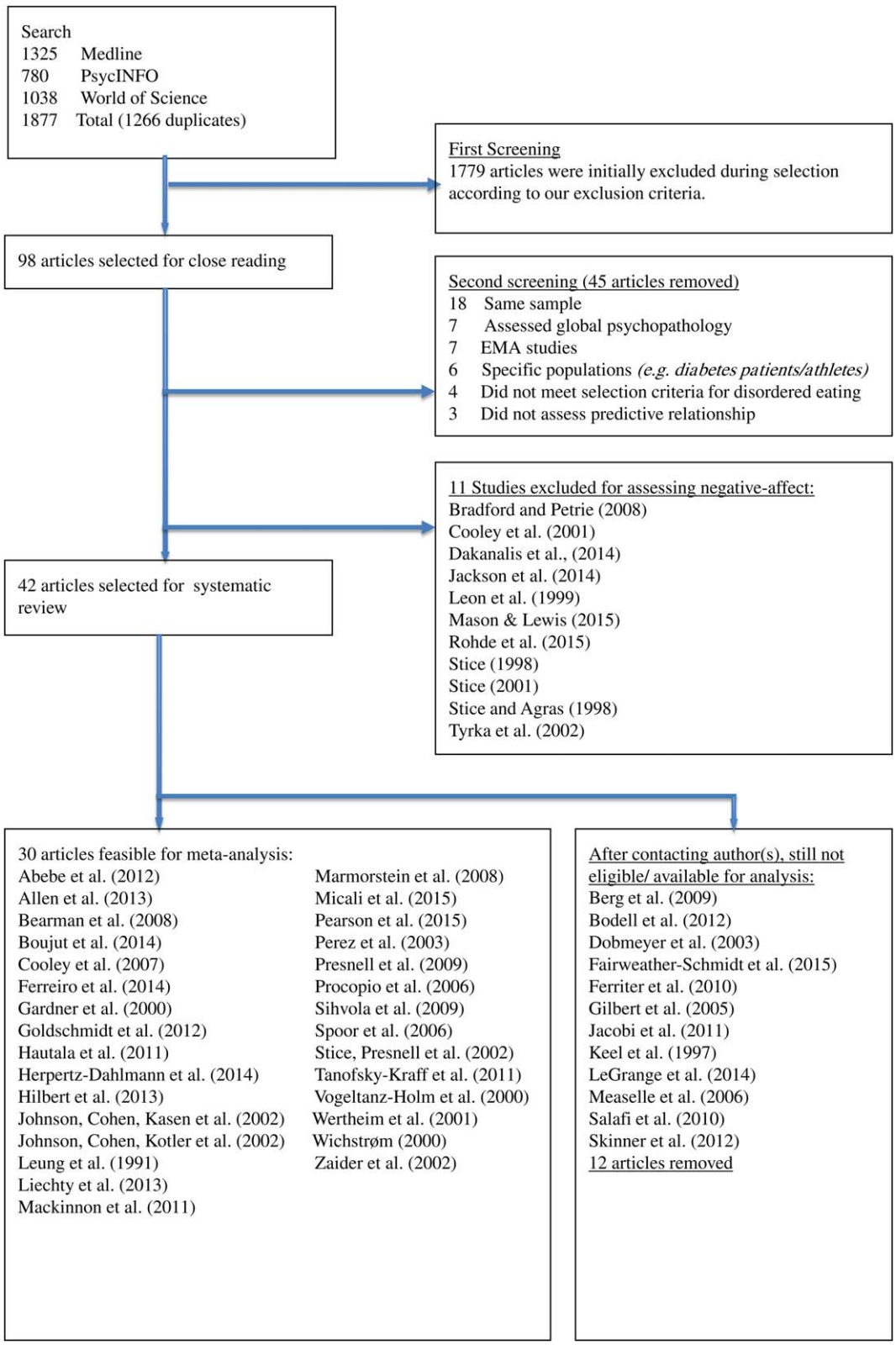

TABLE 1. Overview of each study included in the systematic-review including authors, country of sample, sample size, mean age of participants at baseline, number of assessment points, time-lag, eating pathology measure and assessment type, depression measure and assessment type, and relevant findings

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.
*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 (0.04)	2	60	EDE, SPEEI	Categorical	CDI	Continuous	Binge eating → DS.
Direction of Effects: Depression Predicting Eating Pathology										
*Abebe et al. (2012)	Norway	3,844 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	1,383 females = 703	14.01 (0.19)	3	36	ChEDE EDE-Q	Categorical	BDI-Y	Categorical	DS ↛ binge eating for males or females.
Berg et al. (2009)	America	324	Range = 8–21	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.
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 SCID-DSM III-R	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.
*Goldschmidt et al. (2012)	America	1,827 females = 1,040	12.8 (0.7)	3	60	2 item devised scale	Continuous	DSS	Continuous	DS →binge eating for males and females.

TABLE 1. *Continued*

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
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 EDNOS = 12	DISC-I	MDD, DYS	MDD/DYS →ED. MDD/DYS →binge eating symptoms.
*Keel et al. (1997)	America	204 females = 102	5th and 6th grade	3	12	EAT-26	Continuous	CDI	Continuous	DS ↛ overall DE symptoms for males or females.
*Le Grange et al. (2014)	Australia	1,300 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.
*Pearson et al. (2015)	America	1,906 females = 949	10.86	3	6	EDE-Q	Categorical	CES-D	Continuous	DS →binge eating symptomsfor males and females.
Perez et al. (2004)	America	1,709	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	1,318 females = 671	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.
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.
Direction of Effects: Bi-Directional										
Boujut & Gana (2014)	France	359	18.7 (1.3)	3	6	EAT 26	Continuous	BDI	Continuous	Bi-directional relationship betweenoverall 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 symptomsat T2 for males and females.

TABLE 1. *Continued*

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
*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.
Leung & Steiger (1991)	Canada	543	13–17	2	6	EAT-26	Continuous	8 item self-devised scale	Continuous	Null findings between DS andoverall 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.
Procopio et al. (2006)	America	150	45.19 10.4	2	30	EDI-B	Continuous	BDI	Continuous	Null findings between DS andbulimic symptoms.
Skinner et al. (2012)	America	4,798	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.

* = 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.

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 Fig. 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 uni-directional models where (a) eating pathology predicted depression and (b) depression predicted eating pathology.

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 (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, while 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.

Results

Synthesis of Results for Meta-Analyses

Eating Pathology Predicting Depression. Overall, the effect of eating pathology on depression showed a significant prediction $r = 0.13$ (95% CI: 0.09 to 0.17), $p < 0.001$. 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 $= 0.66$) 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.

Depression Predicting Eating Pathology. Overall, the effect for depression predicting eating pathology was significant, $r = 0.16$ (95% CI: 0.10 to 0.22), $p < 0.001$. 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-

TABLE 2. Relation of initial depression 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	2,923	52	17.34	3	Continuous	Continuous	Bulimic	0.126	0.077
	T2→T3	2,890							0.074	0.079
	T1→T2	2,923		17.34		Continuous	Continuous	Bulimic	0.053	0.084
	T2→T3	2,890							0.072	0.088
Allen et al. (2013)	T1→T2	1,383	36	14.00	3	Continuous	Continuous	Overall DE	−0.085	−0.029
	T2→T3	1,383							0.098	0.109
	T1→T2	1,383		14.00		Continuous	Continuous	Overall DE	−0.001	0.173
	T2→T3	1,383							−0.124	0.139
Bearman et al. (2008)	T1→T2	428	12	13.60	3	Continuous	MDD	Bulimic	-	0.082
Boujut et al. (2014)	T1→T2	359	6	18.70	3	Continuous	Continuous	Overall DE	0.054	0.085
	T2→T3	359							0.010	0.140
Cooley et al. (2007)	T1→T2	117	20	18.00	2	Continuous	Continuous	Overall DE	0.214	-
Ferreiro et al. (2014)	T1→T2	882	12	10.83	4	Continuous	Continuous	Overall DE	0.130	0.040
	T2→T3	748							0.050	0.050
	T3→T4	476							0.169	0.145
Gardner et al. (2000)	T1→T2	189	12	7.06	3	Continuous	Continuous	Overall DE	0.144	0.083
Goldschmidt et al. (2012)	T1→T2	1,827	60	12.80	3	Continuous	Continuous	Binge	0.155	0.076
	T2→T3	1,827							0.154	0.008
Hautala et al. (2011)	T1→T2	722	49.1	14.90	2	Categorical	Continuous	Overall DE	0.502	0.457
Herpertz-Dahlmann et al. (2014)	T1→T2	771	72	14.30	4	Continuous	Continuous	Overall DE	0.062	0.060
	T2→T3	771							0.130	0.160
	T3→T4	771							0.090	0.170
Hilbert et al. (2013)	T1→T2	112	6	10.72	5	Categorical	Continuous	Binge	0.007	0.026
	T2→T3	112						Overall DE	0.196	-
Johnson, Cohen, Kotler et al. (2002)	T1→T2	726	24	13.80	3	Categorical	MDD, DYS	Binge	0.580	-
	T1→T2	726	24	13.80	3	ED diagnosis	MDD, DYS	AN, BN, EDNOS	0.510	-
Johnson, Cohen, Kasen et al. (2002)	T1→T2	726	24	13.80	3	ED diagnosis	MDD, DYS	AN, BN, EDNOS	-	0.370
Leung et al. (1991)	T1→T2	543	6	17.34	2	Continuous	Continuous	Overall DE	0.049	0.065
Liechty et al. (2013)	T1→T2	14,322	84	15.90	2	Categorical	Continuous	Binge	0.290	-
Mackinnon et al. (2011)	T1→T2	200	0.25	19.86	3	Continuous	Continuous	Overall DE	0.028	0.200
	T2→T3	200							0.090	0.080
Marmorstein et al. (2008)	T1→T2	715	36	14.8	3	Continuous	Continuous	Overall DE	0.040	0.130
	T2→T3	644		18					0.040	0.090
Micali et al. (2015)	T1→T2	5,069	24	16	3	ED diagnosis	Categorical	AN	-	0.090
	T1→T2	5,069	24	16	3	ED diagnosis	Categorical	BN	-	0.319
	T1→T2	5,069	24	16	3	ED diagnosis	Categorical	BED	-	0.188
	T1→T2	5,069	24	16	3	ED diagnosis	Categorical	PD	-	0.251
Pearson et al. (2015)	T1→T2	1,906	6	10.86	3	Continuous	Continuous	Binge	0.121	0.064
	T2→T3	1,906							0.108	0.018
Perez et al. (2004)	T1→T2	1,507	13	16.60	3	ED diagnosis	DYS	BN	0.090	-
	T2→T3	941					MDD		0.030	-
Presnell et al. (2009)	T1→T2	496	12	13.50	8	Continuous	Continuous	Bulimic	0.080	0.110
	T2→T3	496							0.090	0.080
	T3→T4	496							0.140	0.150
	T4→T5	496							0.130	0.090
	T5→T6	496							0.120	0.130
	T6→T7	496							0.140	0.080
	T7→T8	496							0.030	0.080
Procopio et al. (2006)	T1→T2	150	30	45.19	2	Continuous	Continuous	Bulimic	0.150	−0.010
Sihvola et al. (2009)	T1→T2	1,318	42	14.19	2	ED Diagnosis	MDD	AN, BN, AED	0.392	-
Spoor et al. (2006)	T1→T2	143	12	19.60	2	Continuous	Continuous	Bulimic	0.172	0.100
Stice, Presnell, & Spangler (2002)	T1→T2	231	10	14.90	3	Continuous	Continuous	Binge	0.190	-
Tanofsky-Kraff et al. (2011)	T1→T2	118	60	10.25	2	Continuous	Continuous	Binge	-	0.185
Vogeltanz-Holm et al. (2000)	T1→T2	709	60	34.70	2	Categorical	MDD	Binge	0.030	-
Wertheim et al. (2001)	T1→T2	435	8	14.09	2	Continuous	Continuous	Bulimic	0.155	-
Wichstrøm (2000)	T1→T2	7,751	24	15.56	4	Continuous	Continuous	Overall DE	0.080	0.119
	T2→T3	7,751							0.095	0.090
	T3→T4	7,751							0.101	0.084
Zaider et al. (2002)	T1→T2	201	10	16.30	2	ED diagnosis	MDD DYS	AED	0.390	0.150
	T1→T2	201							0.560	0.380

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	0.001	0.001	0.792	0.428
	Age	−0.002	0.002	−1.225	0.220
	Wave	−0.032	0.017	−1.878	0.060
	Eating Pathology Assessment Type (Categorical versus Continuous)*	0.194	0.072	2.699	0.007
	Depression Assessment Type (Categorical versus Continuous)*	0.173	0.100	1.728	0.084
	Bulimic Symptoms versus Overall Disordered Eating Symptoms*	−0.014	0.05	−0.283	0.777
	Binge Eating versus Overall Disordered Eating Symptoms*	−0.059	0.087	−0.671	0.502
	Eating Disorder Diagnosis versus Overall Disordered Eating Symptoms*	0.223	0.112	2.002	0.045
	Age × Bulimic Symptoms	0.011	0.005	2.119	0.034
	Age × Binge Eating Symptoms	0.010	0.008	1.286	0.198
	Age × Eating Disorder Diagnosis	−0.102	0.055	−1.862	0.063
Depression Predicting Eating Pathology	Lag	0.001	0.001	0.724	0.469
	Age	−0.003	0.002	−1.318	0.188
	Wave	−0.016	0.013	−1.247	0.212
	Eating Pathology Assessment Type (categorical/continuous)*	0.160	0.195	0.823	0.411
	Depression Assessment Type (categorical/continuous)*	0.128	0.031	4.185	<0.001
	Bulimic Symptoms versus Overall Disordered Eating Symptoms*	−0.033	0.013	−2.585	0.010
	Binge Eating versus Overall Disordered Eating Symptoms*	−0.060	0.023	−2.618	0.009
	Eating Disorder Diagnosis versus Overall Disordered Eating Symptoms*	0.132	0.033	3.977	<0.001
	Age × Bulimic Symptoms	−0.006	0.003	−2.388	0.017
	Age × Binge Eating Symptoms	−0.013	0.014	−0.895	0.371
	Age × Eating Disorder Diagnosis	−0.060	0.008	−7.304	<0.001

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.

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 × eating pathology type revealed one significant finding; the effect of bulimic symptoms on depression was significantly stronger for older participants.

Discussion

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 toward 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.

Moderated Effect of Participant Age and Age × 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 × 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 × bulimic symptoms and age × eating disorder diagnosis was significantly stronger for younger participants. By contrast, the results found that when depression predicted eating pathology, the interaction between age × bulimic symptoms was significantly stronger for older participants. The baseline mean age of participants in this meta-analysis was approximately16 years and the average time-lag between points of assessment was 2 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 and 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 × 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 9–11 years have been shown to have similar levels of disordered eating but girls' symptom level increased at around 14 years of age girls 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 7 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 × 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.

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 this 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.

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 2 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 afterward 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 7 days[66] or 7 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.

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, this 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 to 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.

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.

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.

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 × 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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