

Det Helsevitenskapelige Fakultet – Institutt for Psykologi

Effects of Cognitive-behavioral and Psychodynamic-interpersonal Treatments for Eating Disorders: A Meta-analytic Inquiry Into the Role of Patient Characteristics, and Change in Eating Disorder-specific and General Psychopathology in Remission.

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Hovedoppgave i Profesjonsstudiet i psykologi, PSY-2901, Mai 2020

Preface

The study was conducted in fulfilment of course PSY 2901, and represents the candidates' master thesis in clinical psychology for the cand.psychol. degree at the University of Tromsø - UiT. Both candidates find themselves having a pronounced interest in eating disorders and in research on therapeutic processes and treatment effects, which generated the focus of the study. Additionally, both candidates have over the last year gained clinical experience in treating eating disorders.

Due to the meta-analytical design of the study and need for methodological supervision, the candidates contacted associate professor Rannveig Grøm Sæle at the department of Psychology in october 2018. Later on, doctoral research fellow Anna Dahl Myrvang was contacted, as the candidates needed an assistant supervisor with a specific interest in eating disorders. Rannveig has also contacted other relevant professionals, associate professor Mattias Mittner and senior academic librarian Torstein Låg, to answer questions about systematic literature search, statistical method/design and interpretation of results. The candidates have proposed variables and study design. Research questions were exploratory, thus formulated in advance without having any hypotheses about the results. The supervision has focused on moderating the scope of the study, discussing operationalization of the variables regarding eating disorders, clarifying the research question, discussing the results and feedback on drafts. Most of the supervision has occurred via skype meetings or e-mail.

The meta-analysis is performed by both candidates. However, only Leif Tore had access to the Comprehensive Meta-Analysis Software. Full-text review and coding were completed by both candidates. Disagreement was solved through discussion. The study was submitted for pre-

registration in Prospero in February 2020, and is still being evaluated for eligibility. Given approval of the submission, the candidates will try to publish the study.

Abstract

Background: Knowledge about the outcomes of different psychotherapeutic approaches for eating disorders in terms of remission is limited. Also, knowledge is limited about how therapies and patient characteristics interact to affect outcomes, and by which therapeutic processes. **Method:** Reports on the psychotherapeutic treatment of eating disorders by CBT and PIT were searched. Rates of remission and changes in eating disorder specific and general psychopathology were computed and meta-analytically synthesized. Regression models were made to predict summary event rates by patient characteristics and changes in specific and general psychopathology. **Results:** Only CBT produced remission rates (34.2%) significantly different from waitlist conditions, and only CBT led to significantly greater change in specific psychopathology than waitlist/nutritional counseling conditions. However, CBT and PIT were equally effective in changing general psychopathology. Reduction in general psychopathology predicted higher remission for PIT. For CBT, change in specific psychopathology predicted remission only when controlling for differences between diagnostic categories. Change in general psychopathology predicted remission only for PIT. The presence of comorbid personality disorder decreased the effect of CBT. **Discussion:** A subgroup of eating disorder patients may require therapy aimed at strengthening deficits in self functions not easily ameliorable by cognitive behavioral techniques alone. However, although effective in changing specific and general psychopathology, PIT is not effective in producing behavioral change. Further research should be aimed at identifying treatment interventions that effectuate both behavioral change and strengthening self-functions to substitute eating-disordered behavior as a means to meet psychological needs.

Eating disorders are multifaceted psychiatric states that bring about several aspects to psychotherapy that complicate the process of recovery. Rates of dropout from treatment range from 20 % to 73 %, and patients often have chronic courses of illness (Lock & Fitzpatrick, 2009; Steinhausen & Weber, 2009). Eating disorders are associated with several medical complications (Westmoreland, Krantz & Mehler, 2016) and increased mortality (Arcelus, Mitchell Wales & Nielsen, 2011). There are different theoretical understandings of eating disorders, pertaining to their etiology and maintaining factors, and thus how best to address the difficulties patients face in treatment.

Eating disorders, as defined by the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5; American Psychiatric Association [APA], 2013), are "characterized by persistent disturbance of eating or eating-related behavior that results in the altered consumption or absorption of food and that significantly impairs health or psychosocial functioning". DSM-5 (APA, 2013) recognizes three different eating disorders; anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED). These diagnostic categories intend to reflect independent manifestations of eating disorders, relating to the expression and severity of diagnose-specific core cognitions and behavior. Eating-disordered cognitions are characterized by thoughts about and over-evaluation of body weight, shape and eating. Eatingdisordered behavior, such as subjective or objective episodes of binge-eating, and dietary restriction and compensatory strategies (e.g., use of laxatives, vomiting and excessive exercise) aimed at controlling body weight, shape and eating. These cognitive and behavioral aspects represent the specific psychopathology, distinctive of eating disorders. In the DSM-5, AN is defined by significant weight loss following dietary restriction, lasting three months. BN is defined by the number of compensatory behaviors per week, and BED is defined by the number

of uncontrolled bingeing episodes per week. For BN and BED, the eating-disordered behavior has to be present once a week for a three months period to satisfy diagnostic criteria.

Furthermore, in addition to the eating disorder specific psychopathology, the clinical presentations of eating disorders are accompanied by interpersonal difficulties and comorbidity. Personality disorders have meta-analytically been estimated to be comorbid with eating disorders in 50% of the cases (Martinussen, Friborg, Schmierer, et al., 2017). Also, a large comorbidity survey for eating disorders by Hudson, Hiripi, Pope & Kessler (2007) showed high rates of comorbid disorders (depressive, anxiety and substance use) across eating disorder diagnoses (56% for AN, 94% for BN and 78% for BED). For patients with eating disorders, interpersonal difficulties (Jones, Lindekilde, Lubeck & Clausen, 2015) and psychiatric comorbidities (Berkman, Lohr & Bulik, 2007) are associated with poor outcomes of psychotherapy, and persistence of eating-disordered symptoms.

Two historically prominent theoretical frameworks for understanding the psychopathology present in eating disorders are (a) The cognitive-behavioral schools of thought based off Becks (1979) cognitive-behavioral model of depression and adapted to the symptomatology and clinical presentations of EDs (e.g., Garner & Bemis, 1982; Fairburn, Marcus & Wilson, 1993) and (b) The psychodynamic (Gabbard, 2014) or interpersonal (Klermann, Weissmann, Rounsaville & Chevron, 1984) models converging on the emphasis of the role of others in the development of the self, and relating the development of psychopathology to deficits or difficulties in self-other relations. Regarding the treatment of EDs, these two traditions are distinguished by the extent to which they target eating-disordered cognitions and behaviors - what makes eating disorders special, or deficiencies in the development of the self - what eating disorders share with other mental disorders. The focus of

this meta analytic review was the therapeutic processes and treatment effects of these different theoretical treatment approaches.

In line with the cognitive-behavioral model of eating disorders (Fairburn, Cooper & Shafran, 2003), and the transdiagnostic understanding of eating disorders, all eating-disordered behaviors are assumed to be driven by the core eating-disordered cognitions, i.e., the overevaluation of shape and weight, and their control. For eating disorder patients, self-evaluation is based largely on the extent to which they can control their shape and weight, a feature commonly associated with a subjective experience of low self worth. The over-evaluation is hypothesized to lead to dietary restraint; understood as attempts to restrict food intake. The cognitive and behavioral traits are assumed to be mutually reinforcing and self-perpetuating maintaining mechanisms seen in AN, BN and BED. Thus, the different categories of eating disorders do not necessarily represent independent disorders; rather, they may all reflect different manifestations of the patient's attempts to restore the emotional and cognitive unbalance associated with eating disorders. Fairburn et al., (2003) proposes that the main difference between the eating disorder diagnoses is reflected by the relative balance between under-eating and over-eating. Thus, eating disorders can be understood transdiagnostically as different manifestations of similar cognitivebehavioral mechanisms.

Cognitive behavioral therapies aim to break the cognitive triangle consisting of thoughts, emotions and behavior (Beck, 1979) by restructuring dysfunctional thoughts, beliefs and attitudes pertaining to eating, body shape and weight (Fairburn, 2008) The patient is helped to identify the over-evaluation, and to reflect upon the consequences for emotional and social functioning. The patient is then able to decide whether behavior should be guided by the over-evaluation (i.e., eating-disordered behavior), or not. Self-monitoring of eating- and compensatory

behaviors, and the thoughts and emotions preceding and following these behaviors, are established (Fairburn, 2008; Waller et al., 2018). Early on in treatment, the patient is encouraged to establish regular eating, and abstain from bingeing, and compensatory behaviors.

Psychoeducation about the consequences of eating disorders and the relevant cognitive-behavioral mechanisms is used to increase patients' understanding of their psychological processes and help them to make an informed choice of behavior (Fairburn, 2008). In cognitive behavior theory the specific eating-disordered psychopathology and behaviors are mutually reinforcing (Fairburn, 2003). By simultaneously challenging the specific psychopathology of eating disorders, and deciding not to engage in eating-disordered behaviors, the cognitive-behavioral links that maintain the disorders are gradually dissolved.

Furthermore, eating disorders have been conceptualized as disorders of the self, stemming from a deficiency in the organization of the personality. The lack of sense of self can be regarded as the core psychological deficiency leading to difficulties in emotion regulation and interpersonal difficulties (Schore, 2002; Tasca & Balfour 2014). The deficiency in the sense of self is apparent in the patients experience of self-cohesion, and evaluation of self-worth and self-agency (Beasten & Touyz, 2019). The sense of self is a transdiagnostic construct that has been shown to play important roles in the development of borderline personality disorder, depression and eating disorders (Beasten & Touyz, 2019).

Psychodynamic and interpersonal theories have to various extents emphasised that development of the personality and psychopathology is dependent upon the relation of the self to others. With the advent of attachment theory (Ainsworth 1970; Bowlby, 1958) and the empirical research it inspired (Schore, 2009), the role of deficiencies in self-other relations were increasingly recognized. In light of attachment theory, the development of the personality can be

understood as being dependent upon the quality of the early attachment relationships. Through being emotionally mirrored and regulated by caregivers, the individual develops a cohesive sense of self, capable of containing emotional experiences and needs as real and legitimate. Also, this development underpins an individual's understanding of the affective, motivational and cognitive states of the self and of others, referred to as reflective functioning/mentalization ability (Allen, Fonagy & Bateman, 2008; Fonagy, Gergely, Jurist & Target, 2002). The ability to mentalize make up important areas of functioning of the self, and is closely related to the person's ability to regulate emotions and to form meaningful interpersonal bonds (Tasca & Balfour, 2014).

Studies have shown associations between impaired mentalization skills and insecure attachment styles (Kuipers & Bekker, 2012) in patients with eating disorders. While good mentalization skills are influenced by a fundamental trust in oneself and others; impaired mentalization is often congruent with a fundamental distrust, and thus misconceptions regarding other's intentions. The ability to effectively mentalize is dynamic, and depends on attachment style, emotional arousal and situation.

The deficit in self-cohesion and functioning in adolescence and adulthood can be seen as a result of dissociation of the needs of the self (i.e., the need for self esteem, control and agency, and soothing of aversive emotional states) when frustration of these needs in the past became exceedingly distressing (Schore 2009). However, this defence comes at a high price. In the absence of a cohesive self that can contain basic needs as expressed in a differentiated set of emotions and motivations, it makes the person susceptible to undifferentiated states of general discomfort, which is often expressed as general psychiatric symptoms of anxiety and depressive states, that the self can not contain and modulate (Schore, 2002). A developmentally based lack

of self-cohesion and self esteem has been associated with a higher degree of eating-disordered symptoms in nonclinical college samples (Perry, Silvera, Neilands, Rosenvinge & Hanssen, 2007), the severity of symptoms in AN samples (Bacher-Melman, Zohar, Ebstein & Bachar 2007) and it prospectively predicted the onset of eating disorders in a large sample of schoolgirls (Bachar, Gur, Canetti, Berry & Stein, 2010).

The eating-disordered behaviors may thus serve as surrogate strategies for restoring the patients' sense of self-worth and self-agency, and regulating relations and emotions (Gabbard, 2014). In this sense they are expressions of psychological needs of the self that are disavowed through the defence of dissociation. For instance, research has shown both through qualitative (Espeset, Gulliksen, Nordbø, Skårderud, & Holte, 2012) and quantitative (Greeno, Wing & Shiffman 2000; Meule et al., 2019; Steinberg Tobin & Johnson, 1990) methods that eating-disordered behaviors (i.e., purging, bingeing, dietary restriction, exercising and body checking) reduce negative affect, thus fulfilling the need for emotion regulation. Furthermore, the sense of self-worth and relations may be regulated through the exertion of power and discipline over one's own body, and eliciting care from others (Serpell, Treasure, Teasdale & Sullivan, 1998; Serpell & Treasure, 2002).

To summarize, impairment of different features of the self (e.g., poor mentalizing ability, emotion regulation and interpersonal functioning) is associated with general psychopathology such as anxious and depressive features in patients with EDs (Ivanova, Tasca, Proulx & Bissada, 2015; Tasca et al., 2009). Experienced emotional distress may thus be an important maintaining factor, linking deficiency in the patients self-functions with the specific cognitive and behavioral features of EDs, and psychiatric disorders in general.

The aim of psychodynamic-interpersonal therapy is to heighten the patient's awareness, acceptance and tolerance of affective and motivational experiences. Furthermore, the aim is to help the patient to integrate and contain previously disavowed affective and motivational content into her sense of self (Gabbard, 2014). Several areas of focus that are characteristic of PIT (e.g., mentalization-based therapy, interpersonal therapy, psychodynamic therapy) are thought to be conducive to this process; (a) affect and expression of emotions; (b) exploration of avoidance related to facilitation of the therapy process; (c) identifying patterns in the patient, relating to actions, thoughts, feelings, experiences and relationships; (d) past experiences; (e) interpersonal experiences; (f) therapeutic relationship; and/or (g) exploration of wishes, dreams or fantasies (Blagys & Hilsenroth, 2000).

The National Institute for Clinical Excellence (2004) recommends outpatient psychotherapy as a first-line treatment for EDs. Symptom-focused CBT is recommended for AN, BN and BED. For AN, it is also recommended using psychodynamic or interpersonal therapy approaches, but no specific therapy is recommended over another.

Some meta-analytic evidence has been found for specific treatment effects for CBT relative to other treatment conditions. CBT has been found to be more effective in reducing eating-disordered cognitions (Linardon, 2018a), depressive symptoms (Linardon, Wade, Garcia & Brennan, 2017b) and increasing quality of life (Linardon & Brennan 2017). Furthermore, reduction of eating disorder psychopathology predicted the reduction of behavioral symptoms for BN and BED samples (Linardon, 2018a), and reduction of binge/purge symptoms have been found to predict greater reduction of depressive symptoms in BN samples receiving CBT, compared to other treatments (Linardon et al., 2017b). These findings lend preliminary support

for the cognitive-behavioral model of eating disorders, and thus the core behavioral and cognitive symptoms as principal targets of therapeutic interventions.

However, inferences as to the effect of different specified therapeutic approaches have been difficult to make from meta-analytic inquiries (e.g., Couturier, Kimber & Szatmari 2013; Linardon 2018a, Linardon & Brennan, 2017; Linardon et al., 2017b; Murray et al., 2018; Vocks et al., 2010). This difficulty is due to heterogeneity within the treatments under study and their comparator-treatments with regards to therapeutic approach employed (e.g., different combinations of active psychotherapies, treatment as usual, or wait-list conditions). In addition, outcomes have mainly been focusing on the degree to which eating disorder psychopathology has changed due to treatment, instead of remission. Thus the clinical relevance of differences in treatment effects have been difficult to assess.

Rates of remission, in terms of abstinence from the core behavioral symptoms have, however, been synthesized meta-analytically for BN (Linardon & Wade, 2017) and BED (Linardon, 2018b). For BN the rate was 30% and for BED 45%, for all patients who started therapy. Such figures are to date missing for AN. Comparisons made between treatments included in the studies were specifically defined in terms of their therapeutic foci and yielded mixed results. Interpersonal therapy produced the highest rates of remission for BED. For BN, CBT was most effective. In a direct comparison with interpersonal therapy, CBT led to higher remission rates across eating disorder diagnoses (Linardon, Wade, Garcia & Brennan 2017a).

Knowledge is to date incomplete, as to how different psychotherapeutic foci act on different aspects of eating disorders, as well as the relative contributions of reduction of specific and general psychopathology to remission. Meta-analysis could inform on these issues by basing the assignment of different therapeutic interventions to treatment-arms based on their common

therapeutic focus more consistently and examine their effects on different clinical features presented.

The aim of this meta-analytic review is to shed light on how two specific and conceptually different treatment approaches work for patient samples with varying presentations of eating disorders. Given the high rates of interpersonal problems and psychiatric comorbidity in eating disorder presentations, it is important also to understand the extent to which changes in specific and general psychopathology influence the outcome of therapy in terms of remission. For example, anxious and depressive features often constitute a considerable part of the clinical presentations of eating disorders. These features may be linked to, and maintain the symptoms. The specific and the general psychopathology may also respond differently to different therapeutic approaches. Thus, knowing the way in which these treatment constructs are modified by specific psychotherapeutic approaches would help inform which treatment construct to target and how to target them. To this end we raise three research questions:

- 1. Do CBT and psychodynamic-interpersonal therapies differ in treatment efficacy, and what are their respective effects with regards to remission, and change in specific and general psychopathology?
- 2. To what extent are rates of remission, for each of the two treatment approaches, dependent upon changes in specific and general psychopathology?
- 3. How does patient characteristics, such as eating disorder diagnosis, comorbid personality disorder, and mean patient age affect remission rates for each of the treatment approaches?

Method

This meta-analytic review was submitted for pre-registration in February 2020 at International Prospective Register of Systematic Reviews (PROSPERO). All analyses were planned before the systematic searches, literature review and data extraction were performed.

Search strategy

This review was conducted and reported according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines (Moher, Liberati, Tetzlaff, & Altman, 2009). Electronic databases that were searched are PsycInfo, Embase, Medline, Proquest Dissertations and Theses, and Cinahl. Three search strings were constructed to represent the constructs "eating disorders", "cognitive behavior therapy", and "psychodynamic-interpersonal therapy". The search strings representing the treatment approaches were first combined with the operator OR, then combined with the construct "eating disorder" with the operator AND, in the respective databases. The complete search strategy is attached in appendix A. In order to maximize sensitivity and specificity, a broad spectrum of established practiced forms of therapy within the theoretical traditions of interest were included in the searches. Thesauruses that appeared in the searches were screened, and used if deemed relevant.

Study selection and data management

Reports were pooled in a common library, across databases, using EndNote X9. The references were reviewed, and data from included studies were extracted by the two authors independently. As eating disorders first was included as an independent chapter in DSM-3 in 1980, papers older than 1980 were excluded. Additionally, cognitive behavioral therapy first became a mainstream treatment option for eating disorders in the same period of time. A pilot-review was conducted to ensure inter-rater agreement on the extracted data. Discrepancy was

solved by discussion. When calculating effect sizes, data based on intention to treat analyses were prioritized over data based on completers analysis.

Eligibility criteria

During screening, all references to original clinical trials on the treatment of eating disorders were considered for full text review. To be considered eligible for final inclusion, the reports had to provide information to calculate a between-group or within-groups effect size for the proportion of patients in remission, or changes specific or general psychopathology across at least two time-points; for at least one psychotherapeutic intervention that has a cognitive-behavioral focus or a psychodynamic-interpersonal focus; and directed to outpatients with a clinical eating disorder. Waitlist/nutritional counseling (WL/NC) conditions were included if present in reports that met other inclusion criteria.

Exclusion criteria in the fulltext review included multimodal therapies combining, e.g., milieu therapy, medication, exercise; treatments combining aspects of CBT and PIT, e.g., emotion focused therapy; purely behavioral interventions not targeting the cognitive or psychodynamic-interpersonal aspects of eating disorders, e.g., exposure and response prevention, dietary advice or specialist supportive care; treatments that target some of the treatment constructs targeted by CBT and PIT, but can not be categorized as such because they are broader in scope than either of these therapies, or does in part target different treatment constructs entirely, e.g., dialectical behavior therapy, and acceptance and commitment therapy.

Data extraction and coding

Outcome variables

All outcome variables were coded across two time-points; pre-treatment (t0) and 12 months follow-up (t1). Because relapse rates are high for eating disorders, 12 months follow up

was used to assess treatment effects that can be said to be stable over time. If outcome assessments were available for several time-points after the end of treatment, the time-point closest to 12 months was prioritized. If follow-up assessment was unavailable, end of treatment assessment were used and coded as 0.

The number of patients intended to be treated at t0, and the number of patients in remission at t1 were extracted. For the continuous outcomes, standardized between group and within group changes in the form of Cohen's *d*, were computed based on means and standard deviations at t0 and t1, or from correlations or p-values for pre-post changes. Computing Cohen's *d*, the correlation between the pre and post measures are needed, but rarely reported in primary studies. It is recommended that this correlation is an approximation of the test-retest reliability of the relevant measures (Lipsey & Wilson, 2001). We followed convention and set this correlation to .70 for all measures, which is considered sufficiently close to the test-retest reliability of many psychometric scales (Lipsey & Wilson, 2001).

Primary outcome variable. Remission was defined as the proportion of patients in the treated sample that has undergone weight normalization (AN-samples), cessation of compensatory behaviors (AN- and BN-samples), and cessation of bingeing at t1 (BN- and BED-samples).

Secondary outcome variables. Furthermore, two secondary outcome variables were coded. First, specific psychopathology was coded for t0 and t1. Scales such as the Eating Disorder Examination (EDE) were preferred if primary studies reported several measures. These instruments consist of the subscales; restraint, eating concerns, shape concerns, and weight concerns, assumed to encompass the specific psychopathology. In studies where other instruments were used for measuring specific psychopathology, each subscale were evaluated in

terms of relevance to its core features, e.g., "the drive for thinness" and "body dissatisfaction" subscale in the Eating Disorders Inventory (Garner, Olmstead & Polivy, 1983). The remaining subscales of the EDI were not used because they targeted non-specific aspects of eating disorder psychopathology, or purely behavioral aspects. Second, change in general psychopathology from t0 to t1 was quantified using assessment scales for depressive (e.g., BDI, HAM-D) or anxious (e.g., STAI-S, STAI-T, HAM-A) symptomatology. In cases where several subscales were reported, composite change scores were made from subscale scores measuring specific or general psychopathology. Each sample contributed only with one effect size per outcome measure.

Predictors variables

Treatment approaches were coded categorically as either cognitive-behavioral therapy or psychodynamic-interpersonal therapy. The CBT approach was included and coded based on the focus on dysfunctional thoughts, beliefs and attitudes regarding eating, body shape and weight, and how these relate to behavior and emotions. The PIT approach was included and coded according to the definition by Blagys & Hilsenroth (2000).

Standardized change scores for specific and general psychopathology were also used as predictors of remission in the analyses.

Three patient variables/moderator variables were coded. First, eating disorder diagnosis were coded as either AN, BN, BED or mixed samples. Second, personality disorder was coded as the number of patients in the treated sample with a personality disorder diagnosis. Third, mean patient age was coded as a continuous variable.

Additional study-level predictors. To examine the potential moderating role of followup time on treatment effect, the number of months from end of treatment to follow up was coded

as a continuous variable. This variable will allow an assessment of the stability of treatment effects over time.

Data synthesis and meta-analysis

Meta analyses were performed by using the Comprehensive Meta Analysis Software version 3. All meta-analytic models were constructed with effect sizes weighted by their inverse variance, assuming random effects, as is recommended when the true treatment effects reported by studies are expected to vary (Borenstein, Hedges, Higgins & Rothstein, 2011).

To answer question 1, computation of main effects of treatment approach were planned in separate between- and within-groups analyses. Synthesis of between-group effect sizes were planned for the relative effects of CBT and PIT versus WL/NC in direct comparisons using odds ratios for remission and Cohen's d for standardized differences in changes in specific and general psychopathology. Within-group summary effect sizes were calculated for individual treatment arms where CBT, PIT or WL/NC were delivered, using event rates for remission and Cohen's d for pre-post changes in psychopathology. Because effect sizes were derived from studies with different designs and patient samples, significant statistical heterogeneity were expected and were subjected to examination.

To answer question 2, the impact of change in specific and general psychopathology on remission was assessed. The value of change scores were centered as is recommended for continuous variables used in multiple regression with categorical variables (Frazier, Tix & Barron, 2015). Regression models were made for each treatment approach, where remission rates were independently predicted by change scores. Additional regression models were planned where remission was regressed on change scores and treatment approach compared to WL/NC as simultaneous covariates. In accordance with the conceptual model described by Baron & Kenny

(1986) the effect of one variable on another is dependent upon, i.e., mediated by a third variable to the extent it is accounted for by that variable. A mediated effect would be indicated if the regression weights for the direct relationship between treatment approach and remission decrease to a nonsignificant level when controlling for the effect of purported mediators. Furthermore, the relative importance of each hypothesized mediator was examined by comparing their respective regression coefficients, the variance explained by, and significance of the addition of this variable to the model.

To answer question 3, regression analyses of remission on patient-characteristics (eating disorder diagnosis, age and comorbidity), for each treatment approach were performed. The strength and direction of relationships, significance-levels, as well as variance explained by the models were examined. Regression analyses of remission rates on treatment approach and patient variables as simultaneous predictors were planned.

Analyses were performed to assess the possible significance of design characteristics, i.e., follow-up time and allocation to treatment conditions.

Publication bias assessment

One vulnerability of meta-analyses is the potential presence of publication bias, i.e., if studies with weak or non-significant effects are not published and therefore not included in the analysis. Publication bias has been identified as a problem in both psychological and medical research (van Aert, Wicherts, van Assen, 2019), but is unreliable to test with one method only. The use of several methods is therefore recommended (Coburn & Vevea, 2015). To test for publication bias we used funnel plots to visually assess publication presence of publication bias and Egger's regression for examining correlations between sample size and estimated effect sizes.

Results

Study characteristics

Table D1-D4 in appendix D contains complete descriptions of characteristics for all included studies, and complete reference list of included studies is attached in appendix B. After removal of irrelevant reference types (e.g., qualitative studies, books, reviews, comments, editorials, and papers in other languages than English). 3089 references were screened for eligibility. After fulltext review, 110 studies, with 153 samples met the inclusion criteria. Studies included had sample sizes ranging from 4 to 327 participants. The most common treatment condition was CBT (k = 119). PIT was represented in 25 samples, and WL/NC in 9 samples. Mean age of participants in the included samples ranged from 14.7 to 48.7 years. BED samples had a notably higher mean age compared to mixed, AN and BN samples. Follow-up length ranged from 0 to 24 months.

The studies included varied in study-design, with an even distribution of RCTs and single-arm studies. Out of 363 coded outcomes, 209 were based on intention-to-treat sample size and 136 were based on completer sample size. The remaining 18 outcomes were not specified whether based on ITT or completer analyses. All effect sizes for remission were calculated on ITT sample size.

Definitions of remission varied in the sample of studies. A post-hoc decision was made to include only studies that reported remision as abstinence bingeing and compensatory behavior for 28 days (for BN/BED); or weight restoration to a minimum BMI of 17.5 (for AN). For specific psychopathology the most common measure was EDE/EDE-Q. When the EDI subscales were reported, only the "drive for thinness" and "body dissatisfaction" subscales were included. Other measures included were EAT-26 and the "restraint" subscale of the TFE-Q. The most

common measures of general psychopathology were BDI, BAI and SCL-90. All scales measuring anxious and depressive symptoms were included.

Treatment effects

Direct comparison/between-group. Eight studies met inclusion criteria for direct comparisons of CBT (k = 5) or PIT (k = 3) to waitlist/nutritional counseling conditions. However, because comparisons could not be made across similar outcomes, direct comparison analyses were not performed.

Indirect comparison/within-group. Table 1 shows treatment effects for primary and secondary outcome variables. Rates of remission (k = 81) were significantly predicted by treatment approach (Q(2) = 14.25, p < .001). Remission rates for the CBT condition were significantly higher compared to WL and PIT. However, PIT was not significantly different from WL. Weighted averaged remission rates were 34.2 % for CBT (k = 66), 21.6 % for PIT (k = 10) and 15.9 % for WL/NC (k = 5). The within-group heterogeneity was significant (Q(78) = 320.46, p < .001). The amount of variance explained by the model, indicated by R^2 analog, was 9 %.

Changes in specific psychopathology (k = 100) were significantly predicted by treatment condition (Q(2) = 15.59, p < .001). Only CBT differed significantly from WL/NC. However, there was no significant difference between CBT and PIT in regard to change in specific psychopathology. The standardized differences in means were -.940 for CBT (k = 80), -.737 for PIT (k = 15) and -.145 for WL (k = 5). The within-group heterogeneity was significant (Q(97) = 939.73, p < .001). The amount of total variance explained by the model was 5 %.

Changes in general psychopathology (k = 92) were significantly predicted by treatment condition (Q(2) = 20.02, p < .001). Both CBT and PIT were significantly different from WL. However, there was no significant difference between CBT and PIT in regard to change in

general psychopathology. The standardized differences in means were -.700 for CBT (k = 68), -.710 for PIT (k = 17) and -.162 for WL (k = 7). The within-study heterogeneity was significant (Q(89) = 408.08, p < .001). The amount of total variance explained by the model was 15 %.

Table 1Effect Sizes for Primary and Secondary Outcomes by Treatment Condition.

		Rem	ission		
	k	Event rate	Lower	Upper	I^2
CBT	66	.342	.308	.377	77 %
PIT	10	.216	.153	.297	66 %
WL/NC	5	.159	.087	.275	64 %
		Specific			
	k	Standard difference means	Lower	Upper	I^2
CBT	80	940	-1.039	840	91 %
PIT	15	737	971	502	85 %
WL/NC	5	145	541	.251	12 %
		General			
	k	Standard difference means	Lower	Upper	I^2
CBT	68	700	772	627	81 %
PIT	17	710	864	557	67 %
WL/NC	7	162	389	.064	0 %

Note. k = number of samples; $I^2 =$ the percentage of between-study heterogeneity not due to sampling error; CBT = cognitive behavior therapy; PIT = psychodynamic-interpersonal therapy; WL/NC = wait list/nutritional counseling condition.

Table 2 displays results of meta regression of logit event rates of remission on change in specific and general psychopathology. For CBT samples, remission rates were not significantly predicted by change in specific psychopathology (Q(1) = 3.71, p = .054), leaving significant heterogeneity (Q(34) = 133.06, p < .001). The amount of total variance explained by the model was 4 %. Remission rates were not significantly predicted by change in general psychopathology (Q(1) = .00, p = .946), leaving significant heterogeneity (Q(29) = 114.43, p < .001). The amount

of total variance explained by the model was 0 %. Neither specific or general change predicted remission for either of the diagnostic subgroups. Figure 1 and Figure 2 displays the role of specific and general psychopathology, respectively, in predicting remission for each diagnostic group.

Table 2

Meta Regression of Logit Event Rates of Remission on Change in Psychopathology

	Change in general psychopathology					Change in specific psychopathology						
	95 % CI					95 % CI						
	k	\boldsymbol{B}	Lower	Upper	R^2	p	K	\boldsymbol{B}	Lower	Upper	R^2	p
CBT	31	03	870	.812	.00	.946	36	.42	073	.844	.04	.054
AN	4	54	-2.678	1.605	.00	.623	7	1.36	093	2.814	.37	.067
BN	9	.97	074	2.015	.46	.069	8	.40	444	1.252	.00	.350
BED	8	.03	-1.141	1.192	.00	.965	9	.11	527	.750	.00	.733
Mixed	10	.73	624	2.082	.13	.291	12	.41	053	.863	.13	.083
PIT	6	-2.07	.381	3.756	.67	<.05	-	-	-	-	-	-

Note. k = number of samples; B = unstandardized regression coefficients; $R^2 =$ variance explained; p = significance level; AN = anorexia nervosa; BN = bulimia nervosa; BED = binge eating disorder; Mixed = samples consisting of more than one eating disorder diagnose; CBT = cognitive behavior therapy; PIT = psychodynamic-interpersonal therapy.

Because of nonsignificant coefficients for change in psychopathology and significant unexplained variance, the simple regression models were followed up with hierarchical regressions, testing ED-diagnosis as a potential moderator of the effect. A second regression model was made including diagnostic subgroup as a covariate. Adding diagnostic subgroups to the model, specific change significantly predicted remission (B = .40, p < .001), and model fit was significantly increased (Q(3) = 42.05, p < .001). The model (Q(4) = 48.54, p < .001), explained 70 % of the variance in remission. A third model was made, testing interaction effect. Inclusion of interaction terms did not improve model fit (Q(3) = 1.97, p = .579).

A second model was also constructed for general change. Adding diagnostic subgroups to the model, general change did not significantly predict remission (B = .61, p = .073). However, model fit significantly increased (Q(3) = 31.21, p < .001), explaining 63 % of the variance in remission. A third model was made, testing interaction effect. Inclusion of interaction terms did not improve model fit (Q(3) = 2.02, p = .569).

For PIT samples, regression of remission on specific psychopathology could not be performed due to an insufficient number of studies. Remission rates were strongly predicted by change in general psychopathology (k = 6, Q(1) = 5.77, b = -2.07, p < .05). The remaining unexplained variance was not significant (Q(4) = 6.54, p = 0.162). Change in general psychopathology explained 67 % of the total variance in remission, for PIT. For PIT there was not a sufficient number of studies to conduct multiple regression analyses.

A formal statistical test of mediation was not performed because assumptions for mediated effects were not met for either CBT or PIT. For CBT the purported mediators (i.e., specific and general psychopathology) did not independently predict remission rates. For PIT, the treatment did not significantly predict remission over and above WL/NC.

Table 3 displays point estimates for remission rate by treatment conditions and eating disorder diagnosis. In a multiple hierarchical regression analysis, treatment condition and eating disorder diagnosis significantly predicted the total variance in remission (Q(4) = 34.95, p < .001), and explained 32 % of the between study variance. For CBT, eating disorder diagnosis was a significant independent predictor of logit event rate for remission (Q(3) = 25.53, p < .001), explaining 29 % of total variance in remission. BED had the highest remission rates. Higher mean patient age (k = 56) significantly predicted higher remission rates for CBT (B = .03, Q(1) = 9.97, p < .05), and explained 13 % of the variance. Higher frequency of comorbid personality

disorders in the sample (k = 7) significantly predicted lower remission rates for CBT (B = -.02, Q(1) = 7.90, p < .01), and explained 81 % of the total variance in remission.

Table 3Point Estimates for Remission Rate by Treatment Conditions and Eating Disorder Diagnosis

			CBT					
	95 % CI							
	k	Event rate	Lower	Upper	I^2			
AN	17	.33	.276	.395	77 %			
BN	15	.28	.224	.339	29 %			
BED	15	.50	.424	.568	0 %			
Mixed	19	.30	.251	.352	83 %			
			PIT					
		95 % CI						
	k	Event rate	Lower	Upper	I^2			
AN	3	.24	.120	.431	52 %			
BN	4	.18	.092	.321	28 %			
BED	2	.27	.129	.492	0 %			
Mixed	1	.15	.043	.416	0 %			
			WL/NC					
		95 % CI						
	k	Event rate	Lower	Upper	I^2			
AN	1	.10	.013	.481	0 %			
BN	0	-	-	-	-			
BED	4	.16	.068	.338	71 %			
Mixed	0	-	-	-	-			

Note. k = number of samples; $I^2 =$ the percentage of between-study heterogeneity not due to sampling error; AN = anorexia nervosa; BN = bulimia nervosa; BED = binge eating disorder; Mixed = samples consisting of more than one eating disorder diagnose; CBT = cognitive behavior therapy; PIT = psychodynamic-interpersonal therapy; WL/NC = wait-list/nutritional counseling condition.

For PIT, eating disorder diagnosis did not significantly predict logit event rate for remission (Q(3) = 1.26, p = .739), and did not explain any of the variance. Significant weighted averaged remission rates were found for all diagnose samples (p < .05). Mean patient age (k = 9) did not significantly predict treatment effects of PIT (B = .02, Q(1) = .28, p = .599), and did not

explain any of the variance. For PIT, there was not enough studies to do a regression analysis of comorbid personality disorder.

The significance of design characteristics on treatment effects

Allocation to study (RCTs vs. non-RCTs) did not significantly predict logit event rate for remission (Q(1) = .47, b = -.102, p = .495), and did not explain any of the variance. Follow-uptime did not significantly predict logit event rate for remission (Q(1) = 1.88, b = .010, p = .171), and explained 6 % of the variance.

Publication bias assessment

Visual inspection of funnel plots (Figure C1-C3) were performed for remission, change in specific psychopathology and general psychopathology across all treatment conditions. For remission, the studies were symmetrically distributed around the weighted mean effect sizes, not indicative of small study bias (Borenstein et al., 2011). For remission, Egger's regression test was not significant (t(79) = 1.36, p = .178), indicating that sample size did not predict effect size. However, the funnel plot indicated that high-precision studies varied significantly in effect size around the weighted mean.

For specific psychopathology, visual inspection of the funnel plot suggests asymmetry with effects higher than the mean being more frequently reported. Eggers regression showed a significant association (t(98) = 3.72, p < .001) between sample size and effect size, indicating that sample size predicted effect sizes.

For general psychopathology, visual inspection of the funnel plot suggests asymmetry with higher effect sizes being more frequently reported in smaller studies. Eggers regression showed a significant association (t(90) = 3.55, p < .001) between sample size and effect size, indicating that sample sizes predicted effect sizes.

Discussion

This meta-analysis examined three research questions regarding differences in treatment effects between CBT and PIT on remission rates, and specific and general psychopathology; the predictive value of change in psychopathology for remission; and the role of patient characteristics for remission.

The results can be summarized as follows for question 1: For remission rates, only the CBT condition differed significantly from WL/NC. Only CBT produced changes in specific psychopathology significantly greater than WL/NC. However, the treatments were equally effective in changing general psychopathology. According to convention for interpreting Cohen's *d*, (Lipsey & Wilson, 2001), CBT had a large effect in reducing specific psychopathology and a medium effect in reducing general psychopathology. PIT had a medium effect in reducing both specific and general psychopathology.

For question 2, reduction in specific psychopathology was not significantly associated with higher rates of remission for CBT. When controlling for different remission rates across different diagnoses, specific change emerged as a significant predictor of remission. There was, however, no interaction effects between eating disorder diagnosis and specific change. Although not a significant predictor of remission for either of the eating disorder diagnoses, change in specific psychopathology tended to be a more important predictor of change in AN compared to the other diagnostic subgroups, explaining 37% of the variance in remission. Reduction in general psychopathology was strongly associated with higher remission rates for PIT, but not for CBT. When controlling for differences in remission rates across diagnoses there was still no effect of change in general psychopathology for CBT, and there were no interaction effects

between the variables. Although not a significant predictor, change in general psychopathology tended to be a more important predictor in BN than in the other diagnostic subgroups.

For question 3, eating disorder diagnosis was associated with differences in remission rates only for CBT. BED had the highest rate of remission, with 50% achieving abstinence from eating-disordered behavior for 28 days. The results further indicated that 33 % of AN patients achieved weight normalization. Among BN and mixed diagnoses patients, 28 % and 30 % respectively, achieved remission from behavioral symptoms for 28 days. For CBT, higher mean sample age was associated with higher remission rates. However, this association may possibly be confounded by eating disorder diagnosis due to consistently higher mean age among BED samples. The results showed a strong negative relationship between the proportion of patients with comorbid personality disorder in the samples and remission rates, indicating poorer prognosis for achieving remission for patients with personality disorder. Due to an insufficient number of studies, regression analyses of remission rates on these variables could not be performed for PIT.

The treatment effects described in this study are in line with treatment effects identified by Linardon et al. for CBT in BN (2018a) and BED (2018b), when using the same criteria for remission (i.e., 28 days abstinence from bingeing and purging). The present study is, however, the first meta-analytic estimation of the proportion of AN patients achieving weight restoration in outpatient samples receiving pure psychotherapeutic treatment.

In order to allow comparisons across treatment conditions, variables relevant to inclusion criteria (i.e., diagnosis, treatment approach, psychopathology and remission) in this study were strictly operationalized, either predefined based on relevant literature or the DSM-V. Remission was consistently defined, and only treatment effects in accordance with these definitions were

included. Treatments included were consistently delivered on an outpatient basis with clinicianled psychotherapy as the only intervention. Thereby the confounding effect of multimodal
interventions was avoided, and allowing more valid inferences about the effect of different
psychotherapeutic processes. Only samples consisting of patients with a diagnosed DSM-IV or
DSM-V eating disorder diagnosis were included in the study, allowing generalization to clinical
eating disorder populations. In addition, samples in this study represent a broad spectrum of
patients with different eating disorders that received treatment both in the context of clinical
research and in the setting of regular clinical practices, increasing the ecological validity of the
study.

No systematic evaluation of primary study risk of bias was performed. However, measures were taken to circumvent the effect of attrition bias for remission, as effect sizes were always based on intention-to-treat samples. Furthermore the impact of detection bias was reduced by using objective criteria for remission.

The questions sought to be answered were theoretically motivated, and due to submission for pre-registration, the research questions of the study were pre-determined. Such research practice is encouraged because it reduces the amount of reporting bias in the scientific literature (Stewart, Moher, Shekelle, 2012).

Clinical implications

CBT consistently showed better treatment effect on remission, but eating-disordered behavior persists in many patients even after receiving this treatment. Although results provided by the present study shows that decrease in specific psychopathology is related to remission, the importance of change in specific psychopathology in remission is not ubiquitously clear, and may vary significantly for different eating disorder diagnoses. Such a finding for the significance

of change in specific psychopathology is in contrast to what may be expected by symptomfocused CBT, because of its emphasis on addressing the core cognitive symptoms of eating disorders.

Results of the present study show an apparent disjoint between decrease in general psychopathology, i.e., anxious and depressive features, and remission for CBT. Linardon et al. (2017b), however, found (a) that CBT was more effective in direct comparisons to other active psychotherapies in reducing symptoms of depression in bulimia nervosa; and (b) that the reduction of eating-disordered behavior predicted the superiority of CBT in alleviating symptoms of depression. Although causal inferences can not be made as to the mechanisms of change in CBT from either the present study or the one by Linardon et al. (2017b), these findings may jointly indicate that given that the patient succeeds in reducing eating-disordered behavior during treatment, depressive symptoms are likely to decrease. This finding may indicate that the superior effect of CBT on the behavioral symptoms of eating disorders (e.g., dietary restriction, bingeing and purging) found in the present study may be conveyed through other mechanisms than change in, and general psychopathology.

A question about similarities shared among eating disorder patients in remission may thus be warranted. Among patient factors that have previously been identified as important in enhancing rates of remission from eating disorders, are higher motivation for change, lower degree of depressive features, fewer comorbidities and better interpersonal functioning (Vall & Wade, 2015). Furthermore, important within-treatment factors predictive of good outcomes of therapy is early change in eating-disordered behavior and cognitions. These within-treatment factors have been identified meta-analytically as strong predictors of a favorable treatment response (Linardon, Brennan & Garcia, 2016). Furthermore, early behavioral change, defined as

a reduction of eating-disordered behavior of 65-75% early in treatment was found to mediate outcome across several clinical trials of CBT for eating disorders that examined this relationship (Linardon, Garcia & Brennan 2016).

Taken together with the present results, indicating no association between change in general psychopathology and remission; change in general psychopathology does not seem to be sufficient in itself to effectuate behavioral change in CBT. Although change in specific psychopathology did predict remission rates, the relationship was only significant when controlling for differences between diagnostic subgroups. This association may imply that the maintaining mechanisms and thus relevant targets for treatment interventions may not be uniform across all eating disorder presentations. Thus the patients' motivation for change and actual change early in treatment may be the driving factor in achieving symptom abstinence and weight restoration. In a clinical setting this means that general psychopathology may persist in the absence of behavioral symptoms of eating disorders, and vice versa.

Regarding the effects of PIT, results of the present study suggest that the gradual change of specific and general psychopathology throughout the course of treatment is often not sufficient to achieve remission as defined by behavioral criteria. To the extent that PIT is effective in treating the behavioral symptoms of eating disorders, this effect seems to be conveyed by the reduction of general psychopathology. A different mechanism of action may thus be working in PIT, decreasing both general psychopathology and the dependence on maladaptive emotion regulation strategies expressed as eating-disordered behavior. The therapeutic strategy of PIT, as opposed to symptom-focused CBT, is to ameliorate developmentally acquired deficiency in self-functions that are associated with maladaptive emotion regulation strategies. These deficiencies are reflected in one's sense of self,

mentalization abilities and relational skills, which cause emotional distress. In line with the theoretical assumptions for PIT, the results of this study indicate that remission following PIT seems, to a greater extent than for CBT, to depend on helping the patient develop adaptive regulation strategies to alleviate emotional distress.

Despite indications of fairly uniform remission rates across diagnoses, with the exception of BED, it is clinically relevant to identify what makes patients achieving remission different from patients who do not. This is especially important since only about one third of patients receiving even the most consistently effective treatment achieve remission.

Several factors may complicate the treatment of eating disorders. Patients may experience significant ambivalence towards the prospect of change due to the ego-syntonic nature of the eating-disordered symptoms (Vitousek, 1998). The present study identified a negative association between comorbid personality disorder and remission rate for CBT, perhaps pointing to difficulties engaging these patients in treatment. This finding is in line with the findings in the meta-analysis by Vall and Wade (2015), who found that a higher degree of personality disorders, and other comorbid psychiatric states and poor interpersonal functioning is detrimental to the results of treatment. This may indicate that patients with more severe deficiencies in their sense of self, may be more ambivalent to give up their symptoms, because they function as a means to enhance their sense of self-worth, and regulating emotions and relationships. Therefore, the prospect of change may be threatening. Roncero, Belloch, Perina & Treasure (2013) found that eating-disordered behavior is mainly governed by thoughts that are ego-syntonic, i.e., eatingdisordered thoughts may be valued by the patients and may be in line with the patients' selfimage. However, eating disorders are also characterized by thoughts that are ego-dystonic, i.e., they are unwanted by the patients, not in line with the patients' self-image and unwanted by the

patient, so that they are experienced as intrusive and demeaning. Thus, ego-syntonic thoughts can be understood as a strategy, to handle the ego-dystonic thoughts through eating-disordered behavior (Roncero et al., 2013).

One interpretation of these results may be that the strength of CBT, relative to PIT, is that this therapy is able to offer a therapeutic framework that helps patients utilize their motivation to relieve symptoms that they find bothersome (e.g., intrusive thoughts, loss of control over eating) and seek to eliminate. However, PIT seems to be effective to the extent it alleviates the underlying emotional distress associated with eating disorders. In this, it is as effective as CBT, but in achieving remission results are significantly poorer. Thus, the focus on rapid behavioral change in CBT seems to be effective for patients who have a subjective experience of suffering because of their eating disorder and who are able to commit to treatment. However, in patients where the sense of self is pervasively impaired (i.e., where there is significant lack of self-cohesion, and doubt in self-worth and self-efficacy), internal motivation to work on the behavioral aspects of the disorders may be lacking. PIT may thus offer a more favorable approach and understanding of the subjective sources of suffering for the patient.

Therefore, in line with findings of the present study, evaluation of the extent to which the patient is motivated for change; the patient's sense of self and self-efficacy; and the patient's ability to tolerate emotional distress, may be suggestive of which treatment approach the patient is most likely to benefit from. Patients who are lacking in the aspects of self-functioning may require interventions aimed at examining what needs are fulfilled through the eating-disordered behavior and working with strengthening different features of the self (e.g., mentalization abilities, emotion regulation).

Limitations of the present study

Several features of this study may warrant caution in interpreting the results. Due to lack of randomization, both within and across studies included, this meta analytic inquiry does not demonstrate causal relationships between the variables assessed. The results serve only to represent the degree to which the variables relate to each other, at a meta level.

Furthermore, our sample of studies are characterized by a disproportionate distribution of CBT and PIT samples; CBT being more frequently represented than PIT and WL/NC. Thus, distribution of statistical variance is skewed towards CBT, with less variance contained within the PIT and WL/NC samples, decreasing statistical power. This makes statistical inferences about differences between groups of samples more prone to type 1 and type 2 error (Field, 2013).

There was evidence of publication bias for change in specific and general psychopathology, but not in rates of remission. The effect of publication bias on remission rates in this study may have been attenuated because strict criteria for remission were applied and estimates for remission were consistently based on intention-to-treat sample sizes. However, for specific and general psychopathology, all studies meeting inclusion criteria were included, and consisted of estimates based on both completers and intention-to-treat samples. Thus, publication bias may have been more pronounced in the continuous outcomes and purported mediators than in remission rates, decreasing the association between these variables.

A significant amount of unexplained variance remained for several of the models tested by subgroup and meta-regression analyses. The models indicate differences in remission rates across levels of included variables. However, these effect may be confounded by association to several variables, both included in the model and not included (i.e., diagnostic subgroups, treatment setting, comorbidity, sex, education). The models predicting remission by change in

general psychopathology for PIT and personality disorder indicated good model fit, but were based on few studies.

Directions for future research

During the fulltext review and data extraction, we identified aspects of the previous research that can be more thoroughly investigated in future research. The present results show that the presence of personality disorder is detrimental to the effectiveness of CBT for eating disorders. This result is, however, based on a limited number of studies. It should be made a priority to screen for personality disorders in clinical studies on CBT and PIT for eating disorders. In the pursuit of finding out what therapy works for whom, this is important, especially since meta-analytic evidence (Martinussen et al., 2017) indicates a high rate of comorbid personality disorders in eating disorders. Further research on the role of personality disorders in the treatment of eating disorders could enhance clinicians' ability to match treatment to the needs of the individual patients, and thereby providing effective treatment for a higher number of patients.

However, to make PIT a legitimate treatment alternative to CBT there is a need for more knowledge about active mechanisms of change. Specifically, further research should focus on the extent to which the reduction of general psychopathology seen in PIT is sufficient to bring about satisfying rates of abstinence from eating-disordered behavior. The present results, although based on a limited number of studies, suggest that PIT is not sufficiently effective in bringing about behavioral change. However the therapeutic focus provided by PIT may be a valuable contribution to treatment of eating disorders for targeting aspects of psychological functioning underlying the eating-disordered behavior.

In order to achieve this knowledge, there is a need for more consistent definitions of remission and agreement on relevant outcome measures, across different treatment approaches. Studies on remission in PIT for eating disorders tend to use remission definitions pointing to those aspects of psychological functioning underlying eating-disordered behavior instead of behavioral definitions, as used by CBT studies. Perhaps, primary studies should use both definitions independently of treatment approach, making the outcomes possible to compare directly to each other.

Conclusion

The results of the present study confirm that eating disorders are multifaceted and complex psychiatric states with need for a thorough understanding about precipitating and maintaining factors. It also seems that a therapeutic focus on the eating disorder specific behavior, which is characteristic CBT is necessary to reliably produce behavioral remission. However, the effect of change in eating disorder psychopathology was in itself not sufficient to explain rates of remission. The importance of change in general psychopathology in PIT, suggests that for some patients improvements in self-worth, self-efficacy and self-cohesion may be necessary to obtain remission from eating disorder symptoms. Although the results of the present study suggest that CBT has the best effect on remission and specific psychopathology, the observations made in this study suggest that for some patients the treatment may need a wider focus, and also target aspects of the sense of self.

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Appendix A: Search strategy

For the construct "eating disorders", the terms "eating disorder*", "anorexia nervosa", "bulimia" and "binge eating disorder*" were used.

For the construct "cognitive behavior therapy", the terms "cognitive behavior psychotherapy", "cognitive behaviour psychotherapy", "cognitive behaviour therapy", "cognitive behavior therapy", "cognitive behavior treatment", "cognitive behavior approach", "cognitive behavior approach", "CBT-E", "CBT-ED", "CBT-AN", "CBT-BN" and "CBT-T" were used.

For the construct "psychodynamic-interpersonal therapy", the terms "psychodynamic psychotherapy", "psychodynamic treatment", "psychodynamic approach", "mentalization based psychotherapy", "mentalization based therapy", "mentalization based treatment" "mentalization based approach", "interpersonal psychotherapy", "interpersonal therapy", "interpersonal treatment", "interpersonal approach", "expressive psychotherapy", "expressive therapy", "expressive treatment", "expressive approach", "supportive psychotherapy", "supportive therapy", "supportive treatment", "supportive approach", "analytic psychotherapy", "analytic therapy", "analytic treatment", "analytic approach", "psychoanalytic psychotherapy", "psychoanalytic therapy", "psychoanalytic treatment", "psychoanalytic approach" and "psychoanalysis" were used.

Appendix B: References for primary studies included in the meta-analyses.

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Appendix C: Funnel plots

Figure C1

Funnel Plot for Logit Event Rate for Remission by Standard Error of Measurement

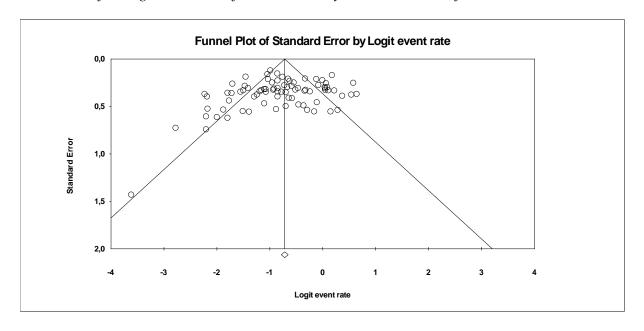


Figure C2

Funnel Plot for Standardized Difference in Means for Specific Psychopathology by Standard

Error of Measurement

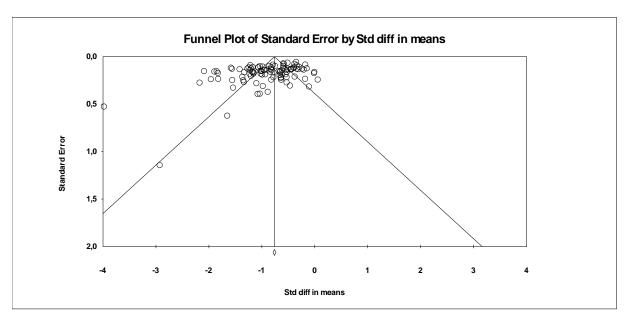
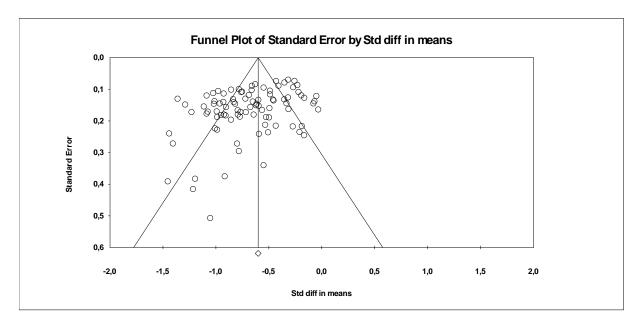


Figure C 3

Funnel Plot for Standardized Difference in Means for General Psychopathology by Standard

Error of Measurement



Appendix D: Primary study characteristics sorted by diagnose subgroup

Table D1.Study Characteristics for AN Samples (k = 25)

Study name	Treatment condition	ITT (n)	Completers (n)	Specific	General	Remission (n)	Mean age	Design
Ball 2004	CBT	13		EDI-BD; EDE-global	BDI; STAI	7	18.5	RCT
Castellini 2013	CBT		27	EDE-Q-global	BDI; STAI		25.6	nRCT
Castellini 2015	CBT	32				21		nRCT
Dalle Grave 2013	CBT	46		EDE-Q-global	SCL-90	13	15.5	nRCT
Dalle Grave 2019	CBT	49		EDE-Q-global	BSI	14	15.5	nRCT
Dare 2001	PIT	21				3	26.7	RCT
Fairburn 2013	CBT	99		EDE-Q-global	GSI	34	24.0	nRCT
Fernandez 1998	CBT		22	EDI-BD; EDI-DT	BDI		20.8	nRCT
Frostad 2018a	CBT	17				5		nRCT
Frostad 2018b	CBT	44				17	23.3	nRCT
Gowers 1994	PIT	20				4	21.2	RCT
Gowels 1994	WL	20				2	21.9	RCT
Hay 2018	CBT	78		EDE-global	Kessler-10	22	28.6	RCT
Jenkins	CBT	63		EDE-global		9	23.6	nRCT
La Mela 2013	CBT	18				6		nRCT
Lelli 2019	CBT	34				15		nRCT
Lock 2013	CBT	23		EDE-global		11	14.7	RCT
Raykos 2013	CBT	17				7		nRCT
Ricca 2010a	CBT (sample 1)	53				19	14.7	RCT
Ricca 2010a	CBT (sample 2)	50				12	23.0	RCT
Sauro 2013	CBT	134				73	27.2	nRCT
Touyz 2013	CBT	31		EDE-global	BDI		34.6	RCT
Watson 2012	CBT	34		EDE-Q-global			25.2	nRCT
				~ 0				

Zipfel 2014	PIT	80	28	28.0	RCT
	CBT	80	15	27.4	RCT

Note. CBT = cognitive behavior therapy; PIT = psychodynamic-interpersonal therapy; ITT = intention-to-treat sample size; RCT = randomized controlled trial design; nRCT = non-randomized/controlled trial design; EDI = eating disorder inventory; BD = body dissatisfaction subscale in EDI; DT = drive for thinness subscale in EDI; EDE = eating disorder examination; EDE-Q = EDE questionnaire; BDI = Beck's depression inventory; SCL-90 = symptom checklist including 90 items; STAI = Stait Trait Anxiety Inventory; Kessler-10 = Kessler psychological disstress scale including 10 items; GSI = global severity index for SCL-90.

Table D2.Study Characteristics for BED Samples (k = 37)

Study name	Treatment condition	ITT(n)	Completers (n)	Specific	General	Remission (n)	Mean age	Design
Abiles 2013	CBT		40	EDE-Q-w; EDE-Q-s;	CHO - CHO 1		10.6	, D.C.T.
Aguera 2013		0.7	49	EDE-Q-e; EDE-Q-r	GHQ-a; GHQ-d	44	42.6	nRCT
· ·	CBT	87				41	34.1	nRCT
Castellini 2011a	CBT	133		EDE-Q-global	BDI; STAI			nRCT
de Zwaan 1997	CBT		80	EDE	BDI		42.7	RCT
Dingemans 2007	CBT	30		EDE-global	BDI	19	38.8	RCT
Dingemans 2007	WL	22		EDE-global	BDI	4	36.4	RCT
Duchesne 2007	CBT	21			BDI		37.2	nRCT
Fahy 1993	CBT	39		EAT				nRCT
				EDE-Q-w; EDE-Q-s;				
Fischer 2014	CBT		33	EDE-Q-e; EDE-Q-r;			45.6	RCT
Fossati 2004	СВТ	13	33	EDE-Q-ows	HAAD; HADD		45.6	nRCT
1 033411 2004		13	22		*			
Gorin 2003	CBT		32		BDI		45.2	RCT
G !! 2011	WL		31		BDI		45.2	RCT
Grilo 2011	CBT	45	37	EDE-global	BDI	23	44.8	RCT
	CBT (sample 1)	14	12	EDE-Q-w; EDE-Q-s; EDE-Q-e; EDE-Q-r	BDI	6	42.1	RCT
Hilbert 2004	CD1 (sample 1)	14	12	EDE Q c, EDE Q 1 EDE-Q-w; EDE-Q-s;	DDI	O	72.1	RCI
	CBT (sample 2)	14	12	EDE-Q-e; EDE-Q-r	BDI	8	38.6	RCT
Hilbert 2020	CBT	37		EDE-global	BDI	19	15.3	RCT
Timbert 2020	WL	36		EDE-global	BDI	12	15.3	RCT
Hill 2015	PIT		75		CES-D		44.2	nRCT
Maxwell 2018	PIT		44		BDI		44.3	nRCT
McIntosh 2016	CBT	36	28	EDE-global	SCL-90-d	19		RCT
Munach 2007		20		EDE-Q-w; EDE-Q-s;	~			
Munsch 2007	CBT	44	16	EDE-Q-e; EDE-Q-r	BDI; BAI	23	44.4	RCT
Olmsted 1989	CBT	30				9	23.7	RCT

Peterson 2009	CBT	60		EDE-Q-global	IDS-SR	31	47.1	RCT
reterson 200)	WL	69		EDE-Q-global	IDS-SR	7	47,1	RCT
Quilty 2019	CBT	27		EDE-global		16	32.8	RCT
Schlup 2009	CBT	18				7	47.1	RCT
Semap 2009	WL	18				0	41.2	RCT
Schlup 2010	CBT (sample 1)	40				15	44.6	nRCT
CBT (sample 2)	36				20	44.4	nRCT	
	CBT		37		CES-D		42.8	RCT
Tasca 2006	PIT		37		CES-D		42.8	RCT
	WL		40		CES-D		42.8	RCT
Tasca 2013	PIT (sample 1)	52	31		BDI	13	46.2	nRCT
1 useu 2013	PIT (sample 2)	50	24		BDI	15	24.2	nRCT
Wilfley 2002	CBT		67	EDE-Q-w; EDE-Q-s; EDE-Q-e; EDE-Q-r EDE-Q-w; EDE-Q-s;	SCL-90-d		45.6	RCT
	PIT		71	EDE-Q-e; EDE-Q-r	SCL-90-d		44.9	RCT
Wilson 2010	PIT	75		EDE-global			48.7	RCT

Note. CBT = cognitive behavior therapy; PIT = psychodynamic-interpersonal therapy; ITT = intention-to-treat sample size; RCT = randomized controlled trial design; nRCT = non-randomized/controlled trial design; EDI = eating disorder inventory; EDE = eating disorder examination; EDE-Q = EDE questionnaire; EDE-Q-w = weight concern subscale in EDE-Q; EDE-Q-s = shape concern subscale in EDE-Q; EDE-Q-e = eating concern subscale in EDE-Q; EDE-Q-r = restraint subscale in EDE-Q; EDE-Q-ows = overevaluation of shape and weight scale in EDE-Q; BDI = Beck's depression inventory; SCL-90-d = symptom checklist including 90 items depression subscale; STAI = Stait Trait Anxiety Inventory; GHQ-a = General health questionnaire anxiety subscale; GHQ-d = General health questionnaire depression subscale; HAAD = Hospital anxiety and depression scale anxiety subscale; HADD = Hospital anxiety and depression scale depression subscale; BAI = Beck's anxiety inventory; IDS-SR = Inventory of Depressive Symptomatology Self Report; CES-D = Center for Epidemiological Studies depression scale.

Table D3.Study Characteristics for Mixed Samples (k = 45).

Study name	Treatment condition	ITT(n)	Completers (n)	Specific	General	Remission (n)	Mean age	Design
Allen 2012	CBT	43		EDE-global		11	26.4	nRCT
Arcelus 2009	PIT	59		EDE-global	BDI	9	28.1	nRCT
Bachar 1999	PIT		14	EAT-26	SCL-90			RCT
Bachar 1999	WL		10	EAT-26	SCL-90			RCT
Back 2017	PIT	16		REDS	PHQ-9		25.9	nRCT
Balestrieri 2015	PIT (sample 1)		7	EDI-BD; EDI-DT	HAM-A; HAM-D			nRCT
Balestrieri 2015	PIT (sample 2)		6	EDI-BD; EDI-DT	HAM-A; HAM-D			nRCT
Bandini 2006	CBT	67				43	20.9	nRCT
Byrne 2011	CBT	125		EDE-global	DASS-A; DASS-D	40	26.0	nRCT
Carter 2016	CBT		42	EDE-Q-global	CC-total		19.7	nRCT
Craig 2019	CBT	22		EDE-Q-global				nRCT
Dalle Grave 2015	CBT	68		EDE-Q-global	SCL-90	25	16.5	nRCT
Fairburn 2009	CBT	77		EDE-global	BSI		26.2	RCT
Fairburn 2015	PIT	65		EDE-global	BDI		26.8	RCT
Fairburn 2015	CBT	65		EDE-global	BDI		25.9	RCT
Fassino 2005	PIT (sample 1)		38	EDI-BD; EDI-DT				nRCT
Fassino 2005	PIT (sample 2)		19	EDI-BD; EDI-DT				nRCT
Fernandez 2009	CBT (sample 1)		150	EDI-BD; EDI-DT			26.7	nRCT
Fernandez 2009	CBT (sample 2)		19	EDI-BD; EDI-DT EDE-Q-w; EDE-Q-s;			22.4	nRCT
Knott 2015	CBT	246		EDE-Q-e; EDE-Q-r	BDI		28.7	nRCT
Mathisen 2020	CBT		39		BDI		27.7	RCT
McIntosh 2016	CBT	38	30	EDE-global EDE-Q-w; EDE-Q-s;	SCL-90-dep	16	34.4	RCT
Mitchell 2008	CBT	66	25	EDE-Q-e; EDE-Q-r	HAM-D	13	29.6	RCT
Navarro-Haro 2018	CBT	47			BDI			nRCT

Palavras 2020	CBT	48		EDE-global			40.9	RCT
Pellizzer 2018	CBT	26		EDE-global	DASS-21	9	28.7	nRCT
Pellizzer 2019	CBT	52		EDE-Q-global	DASS	25	26.4	nRCT
Pendleton 2002	CBT	17			BDI		45.0	RCT
Prestano 2008	PIT		6	EDI-BD; EDI-DT			16.0	nRCT
Raykos 2013	CBT	38				23		nRCT
Riesco 2018	CBT (sample 1)	82				8	25.2	nRCT
Riesco 2018	CBT (sample 2)	57				10	27.4	nRCT
Riesco 2018	CBT (sample 3)	37				8	27.4	nRCT
Rigaud 2011	CBT	51				13	28.0	RCT
Rose 2017	CBT	47		EDE-Q-global	PHQ-9	11	27.1	nRCT
Schapmann-William	CBT		4	EDE-Q-global EDE-Q-w; EDE-Q-s;			16.4	
Shafran 2008	CBT	31		EDE-Q-e; EDE-Q-r			25.9	RCT
Signorini 2018	CBT	111	108	EDE-Q-global	DASS-A; DASS-D		26.1	nRCT
Tomba 2017	CBT	195	185	EAT		51	28.9	nRCT
Turner 2015a	CBT		77	EDE-Q-global			28.4	nRCT
Turner 2015b	CBT	179		EDE-Q	HADS-A; HADS-D	34	27.6	nRCT
Wade 2017	CBT	39		EDE-global		4	23.9	nRCT
Waller 2014	CBT	78			BDI	39	27.8	nRCT
Waller 2018	CBT	93		EDE-Q	GAD-A; PHQ-9		27.4	nRCT
Watson 2012	CBT	75					25.6	nRCT

Note. CBT = cognitive behavior therapy; PIT = psychodynamic-interpersonal therapy; ITT = intention-to-treat sample size; RCT = randomized controlled trial design; nRCT = non-randomized/controlled trial design; EDI = eating disorder inventory; BD = body dissatisfaction subscale in EDI; DT = drive for thinness subscale in EDI; EDE = eating disorder examination; EDE-Q = EDE questionnaire; EDE-Q-w = weight concern subscale in EDE-Q; EDE-Q-s = shape concern subscale in EDE-Q; EDE-Q-e = eating concern subscale in EDE-Q; EDE-Q-r = restraint subscale in EDE-Q; BDI = Beck's depression inventory; SCL-90 = Symptom checklist including 90 items; SCL-90-d = SCL-90 depression subscale; HADS-A = Hospital anxiety and depression scale anxiety subscale; GAD-A = General anxiety disorder assessment; PHQ-9 = Patient health questionnaire major depression module; DASS-A = Depression anxiety stress scale anxiety subscale; DASS-D = Depression anxiety stress scale depression subscale; HAM-A = Hamilton anxiety rating scale; HAM-D = Hamilton depression rating scale; BSI = Brief symptom inventory; CC-total = Cognitive checklist for anxiety and depression.

Table D 4.Study Characteristics for BN Samples (k = 44).

Study name	Treatment condition	ITT (n)	Completers (n)	Specific	General	Remission (n)	Mean age	Design
Agras 2000a	CBT	194				58	28.4	RCT
Agras 2000b	PIT	110	64	EDE-global	SCL-90	17	27.9	RCT
Agras 2000b	CBT	110	65	EDE-global	SCL-90 SCL-90-A; SCL-90-	29	28.3	RCT
Aguera 2012	CBT		100	EDI-BD; EDI-DT	D		23.9	nRCT
Aguera 2013	CBT (sample 1)	327				89	26.2	nRCT
Aguera 2013	CBT (sample 2)	40				12	27.2	nRCT
Arcelus 2012	PIT	10		EDE-global	BDI		28.8	nRCT
Bailer 2004	CBT	41	26	EDI-BD; EDI-DT	BDI	6	24.2	RCT
Barga 2004	CBT	6		EDE-w; EDE-s				nRCT
Blouin 1994	CBT		69	EDI-BD; EDI-DT			23.6	nRCT
Boutacoff 1996	CBT (sample 1)		33	EDI-BD; EDI-DT	BDI		25.8	RCT
Boutacoff 1996	CBT (sample 2)		41	EDI-BD; EDI-DT	BDI		25.6	RCT
Boutacoff 1996	CBT (sample 3)		35	EDI-BD; EDI-DT	BDI		26.4	RCT
Boutacoff 1996	CBT (sample 4)		34	EDI-BD; EDI-DT	BDI		25.7	RCT
Castellini 2011a	CBT	85		EDE-Q-global	BDI; STAI			nRCT
Castellini 2013	CBT		31	EDE-Q-global	BDI; STAI BDI; STAI-S;		30.1	nRCT
Chen 2003	CBT (sample 1)	30		EDE-global	STAI-T BDI; STAI-S;	4	25.8	RCT
Chen 2003	CBT (sample 2)	30		EDE-global	STAI-T BDI; STAI-S;	3	25.8	RCT
Cooper 1995	CBT	13	12	EDE-r; EDE-s; EDE-w	STAI-T	6	23.8	RCT
Fahy 1993	CBT		39		MADRS		23.8	nRCT
Fassino 2005	PIT	38		EDI-DT				nRCT
Freeman 1988	CBT	32		EDI-BD; EDI-DT	IDA-a; IDA-d		24.2	RCT
Garner 1993	CBT	25		EDE-r; EDE-s; EDE-w	BDI	9	23.7	RCT

Garner 1993	PIT	25		EDE-r; EDE-s; EDE-w	BDI	3	24.6	RCT
Ghaderi 2006	CBT	26		EDE-Q-global	BDI		27.2	RCT
Goldbloom 1997	CBT	24	13	EDE-Q-s; EDE-Q-w	BDI	6	25.8	RCT
Le Grange 2015	CBT	58	40		BDI	19	15.7	RCT
Leitenberg 1994	CBT	6		EAT-26	BSI		26.7	RCT
Poulsen 2014	CBT	36				15	25.8	RCT
Poulsen 2014	PIT	34				2	25.8	RCT
Raykos 2013	CBT	50				17		nRCT
Raykos 2014	CBT		78	EDE-Q-global				nRCT
Ricca 1997a	CBT		19	EDE-Q-global	STAI; BDI		23.4	RCT
Stefini 2017	CBT	39		EDE-global	SCL-90		18.8	RCT
Stefini 2017	PIT	42		EDE-global	SCL-90		18.6	RCT
Thiels 1998	CBT		24	EDE-r; EDE-s; EDE-s	BDI		28.7	RCT
Thompson-Brenner 2016	CBT		21	EDE-Q-global	BDI		25.6	RCT
Valbak 2001	PIT	19	10		SCL-90	9		nRCT
Watson 2012	CBT	87		EDE-Q-global			26.8	nRCT
Wilson 1991	CBT		8		BDI		19.8	RCT
Wolf 1985	CBT	15		EDI-BD; EDI-DT	SCL-90-A; SCL-90- D		25.8	RCT
Won 1903	CDI	13		EDI DD, EDI DI	SCL-90-A; SCL-90-		23.0	IC1
Wolf 1985	WL	11		EDI-BD; EDI-DT	D		27.8	RCT
Wonderlich 2014	CBT	40		EDE-global	BDI; STAI	9	28.8	RCT
Zerwas 2016	CBT	90		1.1. TTT '		27	27.5	RCT

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