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**Experiential Avoidance in Depression, Anxiety,
Obsessive-compulsive related, and Posttraumatic stress disorders:
A Comprehensive Systematic Review and Meta-Analysis**

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The data and code used in this manuscript is been attached as appendix file.

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Abstract

Although numerous studies on experiential avoidance and its relationship to psychopathology have been conducted, systematic summaries of this research are lacking. The current systematic review and meta-analysis evaluated the transdiagnostic role of experiential avoidance across depression, anxiety and related disorders (obsessive-compulsive and related disorders [OCRDs] and post-traumatic stress disorder [PTSD]) as well as potential moderators of these relations. A total of 441 eligible studies including 135,347 participants (66.16% female, mean age = 31.53) and 899 effect-sizes were summarized. Results indicated a moderate-to-large association of experiential avoidance with anxiety ($r = .506$) and depressive symptoms ($r = .562$), major depressive disorder ($r = .453$), worry ($r = .516$), generalized anxiety disorder ($r = .588$), social anxiety disorder ($r = .461$), panic and agoraphobia ($r = .340$), specific phobias ($r = .431$), OCRDs ($r = .406$), and PTSD ($r = .489$). Anxiety sensitivity moderated the relationship of experiential avoidance to anxiety and depression. Moreover, depression moderated the relationship of experiential avoidance to generalized anxiety disorder and OCRDs. Correlations varied by mean experiential avoidance value, suggesting a potentially nonlinear relationship of experiential avoidance to psychological symptoms. Other potential moderators including type of population, type of measure, comorbidity, and clinical status were investigated. Results support the hypothesized role of experiential avoidance as a transdiagnostic and transcultural process relevant to depression, anxiety, OCRDs, and PTSD. However, experiential avoidance has largely been measured as a generalized trait; future research would be enhanced by measuring experiential avoidance as a dynamic and contextualized process.

Keywords: experiential avoidance; psychological inflexibility; depression; obsessive-compulsive and related disorders; posttraumatic stress disorder; acceptance and commitment therapy.

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The classification of mental disorders, guided primarily by the field of psychiatry, has changed notably over time (Surís et al., 2016). A topographical and syndromal approach to classification has historically dominated the field, as exemplified by the Diagnostic and Statistical Manual of Mental Disorders (DSM), including its most recent 5th version (DSM-5; APA, 2013). This approach to classification has not helped to elucidate the etiology of mental illness (Frances & Widiger, 2012) and its utility in informing treatment is contested (e.g., Jablensky, 2016; Wakefield, 2016).

Critics of the syndromal approach in the DSM proposed as an alternative a functional dimensional approach that is organized first by treatment utility and subsequently guides research into etiology (Hayes et al., 1996). Unlike syndromal classification, this approach is concerned with specifying underlying functional processes that maintain syndromes and considers symptoms as visible signals of these clinically useful processes. In recent years, calls have grown for the field of clinical psychology to be reorganized around researching such functional psychological processes, that are potentially relevant to psychopathology broadly, rather than protocols matched to syndromes (Hofmann & Hayes, 2018).

One maladaptive functional dimensional process that has been the subject of a large body of research is *experiential avoidance* (EA), defined as a rigid pattern of attempting to avoid or escape unwanted internal experiences such as distressing thoughts, emotions, or physical sensations (Hayes et al., 1996). EA has clear utility in informing treatment (e.g., by fostering acceptance and related psychological flexibility processes as an alternative) and can help integrate research across distinct symptoms and theories (Hayes et al., 1996). Indeed, a vast body of research supports the proposition that EA is the most critical functional process in psychopathology, relevant to depression, anxiety, and other related disorders such as obsessive-

compulsive related disorders (OCRDs) and posttraumatic stress disorder (PTSD; Bluett et al., 2014; Chawla & Ostafin, 2007).

The avoidance of painful or threatening external stimuli is an adaptive part of our evolutionary heritage with clear survival value (Kenrick & Shiota, 2008). Avoiding distressing internal stimuli may also be adaptive in specific contexts; for example, taking a few minutes to down regulate heightened anger before continuing a conversation may lead to a more positive interaction. However, EA becomes problematic when it is pervasive, insensitive to contingencies, and interferes with valued living (Hayes et al., 1996), leading to paradoxically increased suffering and maintenance of symptoms (Chawla & Ostafin, 2007; Cobb et al., 2017; Jacob et al., 2013).

This problematic EA is made more likely due to the bidirectional (Hayes & Wilson, 1993) and combinatorial (Hayes, 1992) nature of language and cognition, which makes it aversive to report, remember, or imagine an aversive experience. While nonverbal organisms merely contact an experience as it is (Hayes et al., 1996), humans interact symbolically with an event, causing the function of the event to be transferred to these symbols (Hayes et al., 2001). As a result, the symbol itself can provoke all thoughts, emotions, and action urges linked with the event. For example, merely hearing the word “chocolate” may make one’s mouth water—and merely thinking about one’s stress may increase it. Thus, EA becomes ubiquitous, pervasive, and dysfunctional through relational learning (Hayes & Wilson, 1993; Hayes et al., 2001), leading to its broad role in psychopathology.

In this way, EA may contribute to a range of psychological disorders as individuals narrow their behavior in order to rigidly avoid unwanted internal experiences, which may actually increase the frequency of those experiences (e.g., paradoxical effects of thought suppression; Abramowitz et al., 2001) and inhibit effective behaviors (e.g., avoidance of exercise due to unwanted physical sensations in those with panic disorder; Sardinha et al.,

2011). The content and manner of avoidance varies depending on the disorder and individual.

In depressive disorders, behaviors such as social withdrawal and loss of interest may be forms of EA toward unwanted private events. EA may occur as nonacceptance of somatic symptoms in panic disorder, of situational anxiety or worries about social performance in social anxiety disorder (SAD), and of worry, thoughts about feared outcomes, or negative contrast (i.e., a steep negative change in affect; Newman & Llera, 2011) in generalized anxiety disorder (GAD). In obsessive-compulsive disorder (OCD), which shares functional similarities with and historically has been included as part of anxiety disorders, situational avoidance and compulsive rituals serve as an attempt to avoid unwanted thoughts. In PTSD, another related disorder historically included with anxiety disorders, avoidance of unwanted trauma memories is a core symptom that exemplifies EA. In addition to clear theoretical relationships between EA and a broad range of disorders, there is empirical support for EA contributing to depression (e.g., Cookson et al., 2020; Ruiz & Odriozola-González et al., 2015; Spinhoven et al., 2014), anxiety disorders (e.g., Bluett et al., 2014; Cookson et al., 2020; Kashdan et al., 2014; Spinhoven et al., 2014), OCRDs (e.g., Begotka et al., 2004; Bluett et al., 2014), and PTSD (Maack et al., 2012; Orcutt et al., 2020; Seligowski et al., 2015; Serrano-Ibáñez et al., 2021).

Although there are many potential psychopathological processes, EA is a particularly promising target of research because it can be reduced through acceptance and mindfulness-based treatments (e.g., Bluett et al., 2014; Kocovski et al., 2013; Lappalainen et al., 2015) as well as other types of treatment (e.g., Eustis et al., 2016; Kocovski et al., 2013). Moreover, EA has been consistently found to mediate treatment effects in clinical trials for anxiety and depression (e.g., Bohlmeijer et al., 2011; Eustis et al., 2016; Forman et al., 2007; Potts et al., 2016). If EA is indeed highly relevant to a broad spectrum of disorders, characterizing this relationship with precision may help to develop and employ effective, efficient treatment procedures that are widely applicable.

However, measurement of EA has had notable challenges, in part due to its transdiagnostic nature. The most common measure of EA, the Acceptance and Action Questionnaire-II (AAQ-II; Bond et al., 2011), has been critiqued for excessive overlap with measures of negative affect (e.g., Tyndall et al., 2019), although early factor analytic research suggested AAQ-II items are distinguishable from items measuring distress (Bond et al., 2011). In addition, the AAQ-II has sometimes been described as measure of EA, and sometimes of the broader construct of psychological inflexibility (a pattern in which EA as well as other processes like inflexible attention and unclear values interfere with meaningful living; Bond et al., 2011). Newer measures have attempted to more precisely distinguish between the multiple, interrelated components of psychological inflexibility (e.g., Francis et al., 2016; Rolffs et al., 2018) or to measure EA more specifically (e.g., Multidimensional Experiential Avoidance Questionnaire [MEAQ]; Gamez et al., 2011). In addition, general measures of EA may not effectively assess EA in a specific domain (Ong et al., 2019), leading to the development of disorder-specific or otherwise context-specific measures such as the Social Anxiety Acceptance and Action Questionnaire (SA-AAQ; MacKenzie & Kocovski, 2010). Despite these measurement limitations, an extensive body of research has been conducted with measures of EA like the AAQ-II and its variants as well as newer, targeted measures of EA like the MEAQ.

Past reviews of the literature have summarized earlier empirical research on EA across multiple disorders (Boulanger et al., 2010; Chawla & Ostafin, 2007; Hayes et al., 1996), and some systematic reviews and meta-analyses have been conducted evaluating the relationship of EA with anxiety disorders (Bluett et al., 2014) and posttraumatic stress symptoms (Seligowski et al., 2015). However, a systematic review and meta-analysis of EA that summarizes its relationship to depression, anxiety and related disorders (i.e., OCRDs and

PTSD) is lacking. This is a particularly major gap given that this body of research has continued to grow rapidly in recent years, without any up-to-date, comprehensive reviews.

A summary of the literature including depression, anxiety, OCDs and PTSD can help to further clarify the transdiagnostic role of EA. Depression, anxiety, OCDs and PTSD are prevalent forms of psychological disorders that frequently co-occur (Kessler et al., 2005), suggesting potential shared transdiagnostic risk factors (Wilamowska et al., 2010), including EA (Levin et al., 2014). Such findings have led to transdiagnostic treatments for these disorders (Ellard et al., 2010; Harvey et al., 2004), including acceptance and commitment therapy (ACT; Hayes et al., 2012), which focuses on EA as a key pathological process (Bluett et al., 2014; Twohig & Levin, 2017). Based on Harvey et al.'s (2009) suggested criteria, a process may be considered transdiagnostic if it is linked to 1) at least four different mental disorders and both 2) clinical (disordered form) and 3) non-clinical (symptomatic form) populations. Therefore, the primary goal of this meta-analysis is to examine the transdiagnostic role of EA across these symptom domains.

In addition, meta-analytic research is needed to evaluate potential moderators of the relationship between EA and psychological symptoms, such as demographics, methodological quality, and psychometrics. Such analyses are now possible with the size of the research literature on EA, allowing for further examination of whether these relations are uniform or vary based on methodological factors or sample/participant-level characteristics. For example, moderation analyses can test whether EA-outcome correlations vary across geographic locations and demographic categories (testing generalizability across populations and potential cultural moderators), measurement factors (type of EA measure, Cronbach's alpha, data collection method), and a variety of psychological variables that have theoretical and clinical implications (diagnostic categories and comorbidities, related constructs like anxiety sensitivity, average level of EA and psychological symptoms). A large scale review that

examines a variety of moderators could further clarify sources of heterogeneity across studies in a way that provides valuable insights for furthering our understanding of how and when EA contributes to psychopathology and methodological factors to consider in future research.

Thus, this study aims to provide an up-to-date systematic review and meta-analysis of the relationship between EA and depressive, anxiety and related disorders. Specifically, the aims of this study are to 1) estimate the association of EA with depressive disorders, anxiety disorders, OCDs and PTSD, and 2) evaluate potential moderators of this relationship. This review will provide a comprehensive summary of a large research literature on the relationship between EA and these disorders, which can clarify the transdiagnostic role of EA, further inform treatment, and guide more precise research into how EA occurs across different contexts.

Method

Literature Search

This review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (Moher et al., 2009). Four databases (PsycINFO, PubMed, Scopus, and Google Scholar) were searched independently by two authors to locate relevant peer-reviewed studies. A comprehensive search strategy was used to capture all potential studies up to October 29, 2020 that examined the correlation of EA with any disorders categorized as depression and related disorders, anxiety and related disorders (OCDs and PTSD), as described in the Diagnostic and Statistical Manual of Mental Disorders, Fifth (DSM-5) and Fourth (DSM-IV-TR) Editions.

A set of keywords were used to detect relevant studies. We searched for *experiential avoidance* OR *psychological inflexibility*. Next, we combined these terms with keywords for depressive disorders, anxiety disorders, obsessive-compulsive and related disorders, and PTSD (see Appendix A for additional details).

This search yielded 10,331 papers; of these, 374 studies were retrieved through the cross-reference technique (Rosenthal, 1991), and four were received by contacting corresponding authors. After removing 1,404 duplicate records, 9,301 were then screened based on title and abstract, but if eligibility was unclear the full text was reviewed. During this screening, 6,533 records were rejected, and 2,768 records were identified as potentially eligible. Two independent reviewers completed the entire screening process, and disagreements were resolved by consensus and consultation with the first author.

Inclusion and Exclusion Criteria

Eligible studies had to meet the following inclusion criteria: 1) used a measurement focused mainly on EA (i.e., AAQ and its variants for specific domains, MEAQ, Brief Experiential Avoidance Questionnaire [BEAQ]), 2) reported a zero-order correlation between EA and a relevant outcome (symptoms of depression, anxiety disorder, OCD, or PTSD) or otherwise provided sufficient data to calculate the effect size, 3) were published in a peer-reviewed journal, 4) had at least ten participants, and 5) were written in English. Studies were excluded if they: 1) used other psychological flexibility process measures that are not specific to EA (e.g., the Comprehensive Assessment of Acceptance and Commitment Therapy Processes scale; Francis et al., 2016), 2) reported only a partial or adjusted correlation, 3) reported only a Spearman (r) correlation coefficient, or 4) had a sample that overlapped with other included studies.

Studies were included across a broad range of disorders including depression, anxiety disorders, OCDs, and PTSD. OCDs and PTSD were included in the review given they have historically been included with anxiety disorders (e.g., DSM-IV-TR), demonstrate a high comorbidity with depression and anxiety disorders (e.g., Kessler et al., 2005), and are regularly included in transdiagnostic theories and treatments for depression and anxiety disorders (e.g., Bluett et al., 2014; Ellard et al., 2010). OCDs and PTSD were also included to further test the

transdiagnostic role of EA across several prevalent disorders. Other trauma and stress-related disorders (e.g., Reactive Attachment Disorder, Disinhibited Social Engagement Disorder) were excluded due to a lack of studies focused specifically on these disorders.

After a full-text review of the remaining 2,768 records, 2,379 were excluded, leaving 389 records included in the meta-analysis. Studies using multiple samples were considered as independent studies; therefore, 441 studies were included in the final analysis. See the PRISMA flow diagram in Figure 1 for an overview of the screening process and Appendix B for a full list of included studies.

Coding Procedures

Authors coded for each study the title, first author, year of publication, journal name, study design, name and mean score for EA and outcome measures, sample size, whether it measured depression, anxiety, OCDs, PTSD, or/and any specific disorder symptomatology that was measured. Study design was coded as cross-sectional, longitudinal, or randomized controlled trial (RCT). To characterize the overall relationship between EA and symptoms, we focused on cross-sectional studies. Baseline, pre-intervention correlations were extracted from longitudinal studies and RCTs. When effect sizes were reported in the opposite direction (i.e., reverse scoring the AAQ-II as a measure of acceptance), the direction was reversed for consistency. All studies reported full data needed to extract effect sizes; authors were otherwise contacted (30 of the corresponding authors) to gather relevant missing data.

Some studies used several EA measures, effectively reporting more than one effect size per sample. To prevent these studies from being overrepresented in the meta-analysis, we calculated and used the combined effect size for these samples in the main analysis. Separate effect sizes by measure were retained and used only for measure-specific comparisons. Some studies reported correlations between EA and OCD symptom subscales (such as washing,

checking, obsessing, ordering and symmetry; Abramowitz et al., 2009; Blakey et al., 2016; Reuman et al., 2018). In these studies, a combined effect size was used in the main analysis.

Moderator Variable Coding

The list of variables to extract was determined by author consensus in order to capture all relevant variables. Two authors independently coded the variables and disagreements were resolved through discussion (see Appendix C for details on extracted data). Four types of moderator variables were coded: 1) methodological, 2) demographic, 3) clinical status, and 4) psychological variables. If moderator variable information was missing from an article, authors were contacted and data that could be retrieved were added to the analysis. If missing data could not be obtained, the study was excluded from the relevant moderation analysis.

Coding of Methodological Variables

Studies were classified into five categories based on EA measure type: using the first version of the AAQ (AAQ-I; Hayes, Strosahl et al., 2004), AAQ-II (Bond et al., 2011), Chronic Pain Acceptance Questionnaire (CPAQ; McCracken et al., 2004), other context-specific AAQ variant, and MEAQ/BEAQ (Gamez et al., 2011). The CPAQ was separated from other context-specific AAQ variants due to its frequency of use (i.e., 28 samples). The included studies were separated into four categories based on the method of data collection: 1) in-person, 2) online, 3) both in-person and online, and 4) over telephone. Cronbach's alphas were extracted for EA measures and outcomes of interest. In addition, sample size and year of publication were extracted and analyzed as potential moderators.

We used criteria provided by Kmet et al. (2004) to assess the quality of quantitative studies. We used only criteria suitable for assessing correlational studies (items 5, 6,7,10 and 12 were removed). Items were scored as 0, 1, and 2 that indicating criteria were not met, partially met, or completely met. Disagreements between authors were resolved through discussion. Scores for acceptable quality range from 10 to 18 in raw score (or 0.56 to 1 if

transformed to a 0-to-1 scale). When studies used multiple samples, they also received multiple quality ratings when relevant.

Coding of Demographic Variables

Samples were coded into three types: 1) general, 2) psychological treatment seekers, and 3) physical health treatment seekers. Geographic region of studies was coded by continent (with North and South America combined). Mean age, years of educational attainment, percent White, percent female, and rates of marriage and employment were also extracted.

Coding of Clinical Status

The samples that met diagnostic interview criteria based on DSM were coded as a clinical sample. Participants selected based on a high score of self-report cut-off criteria were coded as an at-risk sample.

Studies were coded for types of comorbidities: anxiety-related symptoms or disorders, depression-related symptoms or disorders, another psychological symptom or disorder type, multiple psychological problems, no comorbidity, or comorbid with a physical condition. Comorbid substance use disorders were coded as: alcohol use disorder, drug use disorder, smoking, multiple substance use disorders, not specified, or study excluded those with substance use disorder. Medication status was coded as: using psychiatric medication, not using psychiatric medication, or unspecified.

Coding of Psychological Variables

Mean score for EA, outcomes, depression, and anxiety were extracted and analyzed as potential psychological moderators. In addition, the more frequently reported psychological variables across the included studies were coded with the name of their measurement and mean score. These variables, including a brief definition and their relationship with EA are as follows:

Cognitive fusion, the process of rigidly engaging with thoughts in a literal manner (Hayes et al., 1999). This cognitive process may foster avoidance of internal experiences due to perceiving them as more aversive, dangerous, or otherwise needing to be avoided despite the adverse consequences of doing so (Hayes, Strosahl et al., 2004).

Mindfulness, the process of non-judgmental observation of experiences (Robins et al., 2004). Being more mindful can facilitate less EA as an alternative, accepting response to aversive internal experiences (Hooper et al., 2010).

Anxiety sensitivity, the fear of anxiety and its related sensations supported by a belief that such sensations have harmful consequences (Reiss et al., 1988). Anxiety sensitivity is distinct from EA in referring to a specific set of beliefs and attentional biases, while EA describes a broader functional process; however, they are related in that EA towards anxiety is likely among those with high anxiety sensitivity.

General distress, measured by the General Health Questionnaire (Goldberg & Hillier, 1979) refers to impaired functioning and distress due to somatic symptoms, anxiety, insomnia, social dysfunction, and depression.

Negative affect, the extent to which a person feels negative emotions (i.e. upset, guilty, angry, hostile, ashamed, nervous, and afraid (Watson et al., 1988). One major aspect of EA is avoidance of negative affect (Luoma et al., 2020).

Stress, the presence of tension, agitation, and negative affect (Lovibond & Lovibond, 1995). EA may lead to increased stress as it prevents effective coping and behaviors. However, stress may also make anxiety more persistent even in the context of acceptance (Hofmann et al., 2009).

Meta-analytic Procedures

The extracted data were analyzed using version three of the Comprehensive Meta-Analysis software (CMA; Borenstein et al., 2013). A random-effects model was employed as

studies were conducted in a wide range of contexts and thus between-study variance was expected (Borenstein et al., 2009).

Following recommended procedures to increase reliability (Hedges & Olkin, 1985), the effect sizes were weighted by inverse variance weights, which give greater weight to studies with more precise results, for example due to larger samples (Lipsey & Wilson, 2001). Effect sizes (r) were converted to Fisher's Z scale for analysis then converted back to r correlation coefficients for interpretation using the conventional rule of .10, .30, and .50 as small, medium, and large effect sizes, respectively (Cohen, 1988). This also follows expert recommendations (Borenstein et al., 2009).

The pooled correlation coefficients, confidence intervals, and the Tau index were reported. The Tau index is a standard deviation of the true effect sizes across studies. For a meta-analytic effect size of .50, if Tau is 0.10, then most of the effects (95%) fall in the approximate range of 0.30 to 0.70 (Borenstein et al., 2009). We further report the I^2 test, which indicates the proportion of the observed variation that is not simply due to sampling error. Proportions of 25%, 50%, and 70% are considered low, moderate, and high heterogeneity, respectively (Higgins & Thompson, 2002).

Outlier Detection and Sensitivity Analysis

The distribution of effect sizes was evaluated for outliers using multiple methods, including inspection of funnel plots and the one-sample-removed analysis (Borenstein et al., 2009). Sensitivity analyses, which evaluate the effect of using different inclusion criteria (Higgins & Green, 2011), were also conducted. We evaluated the impact of sample size ($N \geq 100$), study quality ($Q \geq 14$), acceptable Cronbach's alpha for EA and outcomes ($\alpha \geq .70$) and recency (publication year ≥ 2010) as methodological factors and a high percentage of women (% women ≥ 60), restriction to adults ($18 \leq M \leq 60$), and % White, as demographic factors on

the robustness of the meta-analytic effect sizes under different decisions. Whenever results were sensitive to a particular variable, it has been reported.

Moderation Analyses

Potential moderators were selected and evaluated based on the purpose of this study and the data obtained from studies included in this meta-analysis. For categorical moderators the combined effect size (r) and homogeneity statistic (Q) were calculated separately for each category and subgroup analysis was used to test whether effect sizes differed significantly. To ensure reliable estimates for subgroup analyses, only categories with five or more studies ($k \geq 5$) were included. Meta-regression was used for continuous moderator variables. Based on expert recommendations (Borenstein et al., 2009), continuous moderators were only analyzed if reported at least by ten studies. All meta-regression was done using Fisher's Z scores within random-effects models with method of moment's estimation.

Publication Bias

Since dissertations and unpublished papers are not included in the present meta-analysis, the assessment of publication bias is critical. Therefore, the likely impact of publication bias on the results was evaluated in several ways, including funnel plot inspection (Light & Pillemer, 1984), Egger's test (Egger et al., 1997), and trim and fill (Duval & Tweedie, 2000).

Results

Study Characteristics

The meta-analysis included 389 articles, 441 distinct studies, 899 effect sizes and 135,347 participants. Among the studies that reported demographic characteristics, the average percent female was 66.16%, percent married was 35.43%, percent White was 67.1%, percent employed was 55.02% and the mean age of participants was 31.53. In total, 77.81% of studies were published between 2014 and 2020. The transformed quality score of included studies

ranged from 0.56 to 1, and 85.67% of studies were rated as high quality. The majority of the studies were conducted in North and South America (63.84%). Data collection was conducted online in 65.07% of studies that reported data collection method (see Table 1).

Overall, 273 studies including 300 independent samples reported Cronbach's alpha for EA measures. The means for the 315 alphas reported were as follows: AAQ-I ($k = 70$, $m_\alpha = .69$), AAQ-II ($k = 173$, $m_\alpha = .88$), CPAQ ($k = 23$, $m_\alpha = .84$), context-specific AAQ variants ($k = 26$, $m_\alpha = .87$) and MEAQ/BEAQ ($k = 23$, $m_\alpha = .86$).

A total of 899 effect sizes were obtained from 441 studies. There were 377 effect sizes for depression and MDD, 359 for anxiety and related disorders, 90 for OCDs, and 73 for PTSD.

Outlier Detection and Sensitivity Analysis

Before conducting any further analysis of the effect sizes, potential outliers were inspected. Eleven outliers were detected from seven different outcomes (a single outlier for MDD, depressive symptoms, SAD, panic and agoraphobia, specific phobia, worry, and PTSD and four outliers for GAD). Only removing the four most influential outliers impacted effect size estimates and reduced heterogeneity significantly, so these four were excluded in subsequent analyses: Esteve et al., 2012 (specific phobia); Flynn et al., 2019 (SAD); Ghazanfari et al., 2018 (MDD); Gloster et al., 2011 (panic and agoraphobia).

Sensitivity analysis only found one potentially sensitive effect. Specifically, the pooled effect size for anxiety symptoms increased significantly ($r = .538$, 95% CI [.513, .562]) when including only studies that reported an acceptable Cronbach's alpha for outcomes, but high heterogeneity was still evident ($Q(105) = 839.136$, $I^2 = 87.606$, $p < .001$).

Effect Size by Outcome

The overall effect size for depression symptom based on 330 studies was $r = .562$, 95% CI [.546, .577] with high heterogeneity ($Q = 4203.635$, $I^2 = 92.173$, $p < .001$) and for MDD the effect size was $r = .456$, 95% CI [.314, .578] with low heterogeneity ($Q = 4.952$, $I^2 = 0.000$, $p > .05$) based on 6 studies. Overall effects and heterogeneity were obtained for anxiety symptoms ($k = 209$, $r = .506$, 95% CI [.485, .525]; $Q = 1806.577$, $I^2 = 88.487$, $p < .001$), SAD ($k = 34$, $r = .461$, 95% CI [.408, .511]; $Q = 217.172$, $I^2 = 84.805$, $p < .001$), GAD ($k = 21$, $r = .580$, 95% CI [.522, .631]; $Q = 219.003$, $I^2 = 90.868$, $p < .001$), panic and agoraphobia ($k = 11$, $r = .348$, 95% CI [.235, .451]; $Q = 17.125$, $I^2 = 41.607$, $p > .05$), specific phobia ($k = 18$, $r = .426$, 95% CI [.348, .498]; $Q = 50.367$, $I^2 = 66.248$, $p < .001$) and worry ($k = 29$, $r = .516$, 95% CI [.461, .568]; $Q = 328.798$, $I^2 = 91.484$, $p < .001$). The average weighted effects and heterogeneity for OCRDs ($k = 28$, $r = .406$, 95% CI [.343, .466]; $Q = 137.191$, $I^2 = 80.319$, $p < .001$), and PTSD ($k = 65$, $r = .489$, 95% CI [.452, .525]; $Q = 597.248$, $I^2 = 89.284$, $p < .001$) were also significant. A forest plot summarizing the effect sizes is presented in Figure 2.

A significant overall difference was found between the effect sizes of the major outcomes (effect sizes from largest to smallest were GAD, depression symptoms, worry, anxiety symptoms, PTSD, SAD, MDD, specific phobia, OCRDs, and panic and agoraphobia; $Q = 77.473$, $df = 9$, $p < .001$). The greatest and smallest pooled effects were .580 for GAD and .348 for panic and agoraphobia respectively. Pairwise comparisons indicated the pooled effect for the EA-panic and agoraphobia correlation was significantly smaller than all other correlations except the EA-OCRDs correlation ($Q = 1.705$, $df = 1$, $p > .05$). The pooled effect size for EA-OCRDs was also significantly smaller than EA-GAD ($Q = 37.060$, $df = 1$, $p < .001$), EA-depression symptoms ($Q = 24.406$, $df = 1$, $p < .001$), EA-worry ($Q = 7.969$, $df = 1$, $p < .01$), EA-anxiety symptoms ($Q = 10.565$, $df = 1$, $p < .01$), and EA-PTSD ($Q = 5.684$, $df = 1$, $p < .05$). Also, the pooled effect size of EA-specific phobia was significantly weaker than EA-depression symptoms ($Q = 12.570$, $df = 1$, $p < .001$) and EA-anxiety symptoms ($Q = 4.599$,

$df = 1, p < .05$). The average weighted effect for EA-GAD was significantly larger than all other correlations except EA-depression symptoms.

The aggregated effect size for EA-depression symptoms ($r = .562, 95\% \text{ CI } [.547, .576]$) was significantly stronger than EA-anxiety symptoms ($r = .506, 95\% \text{ CI } [.485, .525]; Q = 18.376, df = 4, p < .001$). However, this observed difference was no longer significant after considering publication bias (see publication bias section for more details).

Moderator Analyses

There was substantial heterogeneity in the effect sizes, warranting moderator analyses to attempt to identify sources of heterogeneity. Analyses were conducted with categorical moderators to test for differences between subgroups on effect sizes within each symptom area. Meta-regression analyses tested whether continuous variables moderated the relation between EA and each symptom areas. Significant moderator results are presented for categorical moderators in Table 2 and for continuous moderators in Table 3 as well as described further in the following section. Potential moderators without sufficient studies were not analyzed and are omitted from the results. Non-significant moderator results are listed in Appendix D.

EA and Depressive Symptoms

EA assessment type moderated effects such that effects were observed from largest to smallest with the AAQ-II, AAQ-I, CPAQ, MEAQ/BEAQ, and finally context-specific AAQ variants (Table 2). No significant differences were found based on data collection method ($Q = 3.511, df = 2, p > .05$), population type ($Q = 4.808, df = 2, p > .05$) or region ($Q = 4.354, df = 4, p > .05$). Similarly, no differences were found based on MDD versus depressive symptoms ($Q = 2.418, df = 1, p > .05$), medication status ($Q = 1.342, df = 1, p > .05$), drug use ($Q = 0.023, df = 1, p > .05$) or comorbidity ($Q = 8.491, df = 4, p > .05$).

Continuous methodological variables significantly moderated relations such that the EA-depression correlation strengthened as Cronbach's alpha increased for EA and depression

measures as well as for more recent publications relative to earlier ones. Similarly, the EA-depression correlation was greater when severity of general distress and the proportion employed increased. In contrast, the EA-depression symptoms correlation weakened as age increased, and as mean EA and anxiety sensitivity increased (Table 3).

EA and Anxiety

The effects were classified into six classes (anxiety symptoms, SAD, GAD, panic and agoraphobia, specific phobia, and worry) and subgroup analysis indicated significant heterogeneity ($Q = 59.974$, $df = 4$, $p < .001$). Because of high heterogeneity in the anxiety classes, moderator analysis was conducted for each class (except for panic and agoraphobia) independently.

EA and Anxiety Symptoms. Results indicated significant differences between EA assessment types such that effects were largest for the AAQ-II, followed by AAQ-I, MEAQ, CPAQ, and finally other context-specific AAQs (Table 2). The EA-anxiety symptoms correlation was also moderated by both population type and geographic region, such that effects were strongest for North and South American samples and general samples (Table 2). Other categorical variables did not significantly moderate EA-anxiety symptom effect sizes including method of data collection ($Q = 3.967$, $df = 2$, $p > .05$) and comorbidity ($Q = 5.691$, $df = 3$, $p > .05$).

Among the tested continuous moderators, the EA-anxiety symptoms correlation significantly increased based on higher Cronbach's alphas for EA and outcome, increased education, and an increase in general distress. Meanwhile, the EA-anxiety symptoms correlation significantly decreased as age, mean EA, and anxiety sensitivity increased (Table 3).

EA and SAD. There were no significant categorical moderators including type of EA measure ($Q = 4.178$, $df = 2$, $p > .05$), data collection method ($Q = 0.061$, $df = 1$, $p > .05$),

population type ($Q = 0.537$, $df = 1$, $p > .05$) or region ($Q = 0.205$, $df = 1$, $p > .05$). The only significant continuous moderator of the EA-SAD correlation was mean EA, such that greater mean EA weakened this correlation (see Table 3). Mean age, percent female, percent White, mean outcome score, and mean depression score were not statistically significant moderators.

EA and GAD. Data collection method and population type were significant categorical moderators. The pooled effect sizes for online data collection were larger than pooled effects for correlations obtained through in-person report and the correlation between EA and GAD in general samples was stronger than in psychological treatment seeker samples (Table 2).

The EA-GAD correlation was strengthened by increased study quality, increased Cronbach's alphas for EA and outcome measures, and , more recent publication year, but was decreased as percent married, mean EA score, and mean depression score increased (Table 3). Mean age, percent female, percent White and mean outcome score were not significant moderators.

EA and Worry Symptoms. Population type moderated the correlation between EA and worry such that effect sizes for general samples were larger than psychological treatment seeker samples (Table 2). Other categorical variables did not significantly moderate EA-worry including type of EA measure (AAQ-I or AAQ-II; $Q = 0.756$, $df = 1$, $p > .05$), data collection method ($Q = 0.046$, $df = 1$, $p > .05$), or region ($Q = 0.001$, $df = 1$, $p > .05$).

Only Cronbach's alpha for outcome measures was a significant continuous moderator for EA-worry, such that this correlation increased as Cronbach's alpha increased (Table 3). Mean age, percent female, percent White, mean EA, mean outcome, and mean depression score were non-significant.

EA and Specific Phobia. The EA-specific phobia correlation in physical health treatment seeker samples was larger than other populations (general and psychological treatment seekers; Table 2). The EA-phobia correlation was also stronger in samples with

comorbid physical problems than in samples with no comorbidity (Table 2). There were no significant differences in effect size based on EA measure type ($Q = 7.462$, $df = 3$, $p > .05$) or region ($Q = 1.450$, $df = 1$, $p > .05$).

Publication year, sample size, study quality, and percent female were not significant predictors. Increasing age strengthened the EA-specific phobia correlation (Table 3) with an R-squared of 0.98, indicating mean age explains 98% of the observed variance in the EA-specific phobia correlation. However, it should be noted that R-squared estimates for meta-regression can be unstable with a tendency toward overestimation when the number of included studies is small (López-López et al., 2014).

EA and OCRDs

This category included 19 effect sizes for OCD, 5 for trichotillomania, 3 for hoarding, and 1 for skin-picking (i.e., only OCD and trichotillomania had sufficient effect sizes for subgroup analysis). The correlation with EA was significantly larger for OCD compared to trichotillomania (Table 2).

When combining effect sizes for all OCRDs, the EA-OCRDs correlation was significantly larger in general than psychological treatment seeker samples, in European compared to North and South American samples, and in at-risk versus clinical samples (Table 2). Neither EA measure type ($Q = 4.286$, $df = 2$, $p > .05$) nor data collection method ($Q = 0.567$, $df = 1$, $p > .05$) moderated effects.

Three continuous moderators were significant for the EA-OCRDs correlation. Higher Cronbach's alpha for the EA measure as well as higher outcome and depression scores reduced the correlation between EA and OCRDs (Table 3). Cronbach's alpha for outcome measure, mean age, percent female, percent White and mean EA score were not significant moderators.

EA and PTSD

EA measure type significantly explained high heterogeneity, with larger effects for the AAQ-II relative to the AAQ-I, and MEAQ (Table 2). The EA-PTSD correlation was also significantly moderated by region, such that North and South American samples had larger effect sizes than European samples (Table 2). Other testable moderators were not significant including data collection method ($Q = 1.301, df = 1, p > .05$), population type ($Q = 5.296, df = 2, p > .05$), clinical status ($Q = 0.000, df = 1, p > .05$), and comorbidity ($Q = 0.549, df = 1, p > .05$).

The EA-PTSD correlation strengthened with higher Cronbach's alphas for EA, with a larger percent employed, with increased education, for more recent publications, and for greater outcome severity (Table 3). Higher mean EA diminished the size of EA-PTSD correlation. Percent female, percent White, mean age, percent married, and mean depression score were not significant moderators.

Publication Bias

Funnel plots are presented in Appendix E for all estimates derived from $k > 10$ studies. Results of Egger's regression, trim and fill and analysis are reported in Table 4.

Regression coefficients from Egger's regression were significant only for depressive symptoms ($p < .001$), GAD ($p < .001$) and worry ($p < .05$). Therefore, the possible effect of publication bias on these results was further assessed by trim and fill analysis, which showed that the adjusted effects were still significant for all outcomes.

However, results did indicate that publication bias may be relevant to the size of the EA-depression correlation. Based on the trim and fill analysis, 59 studies would need to be added to the left side of the distribution of effect sizes (i.e., small effects are missing) to make the funnel symmetrical, resulting in a new estimate of $r = .518$, 95% CI [.498, .537], significantly smaller than the original estimate (.562, 95%CI [.546, .577]) although still indicating a large correlation. We repeated the comparison between the effect size for the

depressive and anxiety symptoms correlations using the adjusted correlation for depressive symptoms, and the difference was no longer significant.

Discussion

This systematic review and meta-analysis sought to estimate the magnitude of the relationship of EA with depression, anxiety, OCDs and PTSD as well as identify potential moderators. Studies span 18 years of research from 2002 to 2020, composed of 135,347 participants and including effect sizes ranging from medium ($r = .406$) to large ($r = .560$). The results support the functional dimensional (Hayes et al., 1996) and transdiagnostic (Boulanger et al., 2010) nature of EA across depression, generalized anxiety disorder, social anxiety disorder, panic disorder, specific phobia, PTSD, and OCDs. The size and consistency of these effects also support the conceptualization of EA as a core pathological process and treatment target.

Comparison of Effect Sizes

While the pooled weighted correlation between EA and depressive symptoms was larger than that of anxiety symptoms prior to accounting for publication bias, this difference was no longer significant after adjusting for publication bias, suggesting a similar, large relationship of EA with anxiety and depression.

In more fine-grained analyses, the effect size was also similar for MDD relative to depressive symptoms, suggesting that EA makes a similar contribution to depression regardless of its severity. Similarly, there was not a significant difference when comparing the correlation of EA with anxiety symptoms to that of EA with worry, social anxiety, or posttraumatic stress symptoms, suggesting EA may share a similar role in these disorders. However, the correlation of EA with anxiety symptoms was significantly larger than that of EA with specific phobia, panic/agoraphobia, and OCDs. Specific phobia, panic, and OCDs tended to have smaller effect sizes relative to other disorders. This disparity may be due to EA serving as a maintaining

factor rather than initial cause of anxiety in some presentations (Hayes et al., 1996; Orsillo et al., 2004), or to differences in how well EA is captured by existing measures as anxiety disorders vary in exactly what content is avoided (e.g., intrusive thoughts in OCD, large changes in negative affect in GAD). The smallest effect size, although still medium in magnitude, was for EA with panic and agoraphobia.

The relationship between EA and OCD was significantly different based on the level of symptoms, with a smaller correlation for clinical OCD than at-risk OCD. This suggests that the explanatory power of EA may decrease as OCD symptoms become more intense. This surprising result is consistent with findings that show EA measures are too general to explain more specific symptoms of OCD, above and beyond variables such as obsessive beliefs and general distress (Abramowitz et al., 2009). It is possible that EA contributes to OCD symptoms in the early stages, leading to the development of dysfunctional cognitive patterns, which then become dominant as severity increases.

Generally, more research is needed to identify processes that alter the relationship of EA to anxiety and depression across their specific manifestations. Therefore, we examined potential moderators in depth. Notable findings are discussed hereafter.

Moderators

Key Demographic and Clinical Status Moderator Findings

Eighty-seven percent of studies were conducted in North and South America and Europe; of this, sixty-three percent were done in the former, and the rest done in other regions. There were no significant differences across region in the effect sizes for depressive symptoms, SAD, specific phobias, and worry (GAD and panic/agoraphobia lacked sufficient studies for comparison), but there was a significant difference for anxiety symptoms and OCDs. The correlation was smaller in North and South America compared to Europe for OCD, but the reverse was true for PTSD. The effect size for anxiety symptoms was also larger in North and

South America relative to Asia. Broadly, the lack of consistent differences supports the role of EA as a process relevant across cultures and languages.

Eighty-six percent of the participants were aged 18 to 45 years, and the mean age was 31.53. Age was a significant moderator for anxiety and depressive symptoms indicating that the correlations weakened as age increased. This is potentially consistent with findings that older people report lower psychopathology (Erskine et al., 2007) or that acceptance increases with age (Shallcross et al., 2013). These findings overall suggest a consistent role for EA across age groups for psychological disorders, albeit less so for psychological symptoms.

Among the studies where education was reported, eighty-one percent were undergraduates. It was not possible to investigate years of education as a moderator for most outcomes, but it did moderate the EA-anxiety symptoms and EA-PTSD correlations such that the correlation was stronger among those more highly educated.

Comorbidity did not significantly moderate the EA-anxiety, EA-depressive symptoms, or EA-PTSD relationships, although it did moderate the EA-specific phobia correlation such that effects were larger for those with a comorbid physical condition. Overall, this supports the transdiagnostic role of EA, suggesting that comorbidity does not impact its fundamental relationship to psychopathology; however, several outcomes could not be examined due to insufficient studies.

Key Methodological Moderator Findings

EA measurement types did not yield significantly different effect sizes across OCRDs, specific phobia, anxiety symptoms, worry, or SAD. However, the AAQ-II had greater correlations with PTSD, anxiety, and depressive symptoms compared to other measures. While it is reasonable for the AAQ-II to have larger correlations than the AAQ-I due to improved measurement, the AAQ-II also had larger effects than newer EA measures like the MEAQ. Context-specific AAQs also had significantly lower effect-sizes for correlations with anxiety

and depressive symptoms compared to general AAQs. Although context-specific AAQ measures typically have stronger correlations than the AAQ-II with targeted outcomes of interest (Ong et al., 2019), it may be that the AAQ-II is already sensitive to assessing effects with depression and anxiety symptoms.

Cronbach's alpha for EA moderated the relationship with PTSD, GAD, anxiety, depressive symptoms, and worry, such that higher internal consistency was related to stronger relations with EA. However, internal consistency for EA actually had the reverse relationship with OCRDs, with lower effect-sizes when internal consistency was higher. Overall these findings highlight the importance of using EA measures with strong psychometrics given the impact of internal consistency on observed relations with several symptom categories.

Data collection method was not a significant moderator in six of seven analyses (with the EA-GAD correlation being the lone exception). This suggests that relationships with EA can be accurately measured using both in-person and online methods.

Psychological Moderators

Several findings suggested a nonlinear relationship between EA and psychological symptoms across different levels of the severity of symptoms or of EA. General distress moderated the EA-anxiety and EA-depressive symptom correlations, such that higher distress was associated with stronger correlations. Higher PTSD severity strengthened the EA-PTSD correlation, while higher OCD severity weakened the EA-OCD correlation. Such findings suggest that the impact of EA varies across contexts, such as the degree of distress or symptom severity. This moderation effect could be due to differing impacts of EA at different stages in the trajectory of symptom development or recovery. When examining mean EA as a moderator, the strength of the effects weakened in anxiety and depressive symptoms, SAD, GAD, and PTSD as mean EA increased. This could be explained, for example, by EA being particularly relevant to mild or early psychological problems, while other processes such as cognitive

fusion or loss of contact with values are more relevant to more severe psychological problems. Alternatively, this could be related to issues of insight (e.g., highly avoidant individuals could underreport symptomatology). Future research should investigate potential explanations, including measurement issues and the trajectory of EA across the development and maintenance of these disorders.

Anxiety sensitivity negatively moderated the EA-anxiety and EA-depressive symptom correlations, such that higher anxiety sensitivity was related to weaker correlations. Given the positive relationship anxiety sensitivity has with EA (Epkins, 2016) and anxiety and depressive symptoms (Hovenkamp-Hermelink et al., 2019; Olthuis et al., 2014), this moderation effect is surprising. One possible explanation is that general EA measures may not adequately assess avoidance of somatic cues, which is particularly relevant among those with high anxiety sensitivity.

Limitations and Future Directions

Several limitations should be noted. First, this meta-analysis was not pre-registered. Pre-registration would have increased transparency in how the meta-analysis was conducted and whether changes in the approach were made during data collection and analysis in such a way that might increase the potential for Type I errors. That said, we aimed to provide a comprehensive summary of all of our moderation analyses and detailed supplementary information in the appendices to aid in transparency in analyses that were conducted and moderators that were tested.

There are also limitations in the literature that was analyzed. Young adults and undergraduate students are overrepresented in studies identified. Concerns about generalizability are well warranted; indeed, age and education status did moderate the relationship between EA and symptoms in several analyses. Additionally, current research lacks geographic diversity and much more research is needed outside of the USA in particular.

Indeed, findings from this review signal the importance of considering cultural context. For example, the weaker association between anxiety symptoms and EA for studies conducted in Asia relative to those in North and South America could be due to difficulties effectively measuring EA in this context (for example, due to cultural conceptualizations of anxiety; Hinton et al., 2009) or due to a meaningful difference in the relationship between EA and anxiety symptoms across cultures. It is essential to conduct research on EA among populations that are diverse in terms of culture, age, race, and other aspects of identity in order to ensure generalizability of findings, as well as to identify relationships between social and individual context and EA.

Another caveat of note is that correlations with EA were larger for studies that used the AAQ-II. Given concerns that the AAQ-II may have notable overlap with negative affect (Tyndall et al., 2019), and that studies using the AAQ-II make up more than half of the included studies, it is possible that the effect sizes identified in this analysis are larger than they might be if EA were measured with greater precision. Given that several measures of EA or psychological flexibility have now shown superior divergent validity such as the BEAQ (Tyndall et al., 2019), MEAQ (Rocheffort et al., 2018) Multidimensional Psychological Flexibility Inventory (MPFI; Landi et al., 2021) and CompACT (Francis et al., 2016), the quality of research on EA and psychological inflexibility more broadly may be improved through use of such measures.

Moderation findings identified a number of areas where more targeted research is needed to confirm and clarify variables influencing the impact of EA. It would be particularly valuable to investigate why the relationship between EA and psychopathology appears to vary depending on the degree of symptoms and/or EA. As EA is a dynamic, contextual process it may have a non-linear (e.g., quadratic) relationship to psychopathology over time. This finding highlights a notable limitation of this review, and of much of the research conducted on EA:

cross-sectional and group-level analysis of EA is inherently limited in its ability to examine the impact of EA as a contextual process. As advocates for process-based therapy have suggested (Hayes et al., 2019), using innovative methods to examine intra-individual relations between EA and symptoms, in a manner that is both longitudinal and context-sensitive, is sorely needed.

The pattern of findings in OCRDs also highlights the importance of ongoing research on EA within specific areas. The relationship between EA and symptoms was lower in clinical OCD, even in studies with lower measurement error. This may suggest that EA is not being effectively measured in OCRDs using general measures, and points to broader issues in measurement. With general self-report measures, EA is effectively being measured as a trait rather than a process. Even using disorder-specific measures is insufficient, because EA is fundamentally a contextually bound, dynamic process, which varies as context varies. Therefore, we need measurements that evaluate EA in a context-sensitive manner. Measures that assess psychological flexibility across a specific timeframe (Gloster et al., 2021) or in the pursuit of specific, personalized values (Kashdan et al., 2020; Akbari, Disabato et al., 2021; Akbari, Seydavi et al., 20) are a step forward. Such measures can also help shift research on EA to be more consistent with the framework of process-based therapy (Hofmann & Hayes, 2018).

As a whole, the results of this meta-analysis support the importance of EA and its transdiagnostic role across depression, anxiety and related disorders. However, the moderator findings do suggest that the role of EA depends both on measurement and on contextual factors that may vary between and within each disorder. Thus, more fine-grained research into the role of EA may be valuable. For example, what individuals avoid and how they avoid varies between and within disorders. Identifying stimuli and responses classes that have distinct relations to problem severity, as well as the contexts that govern when they are more or less problematic, may help explain some variability in the role of EA psychopathology. Identifying

such variability would not change the functional definition of EA (Hayes et al., 1996), but would provide additional guidance for assessing, conceptualizing and treating these disorders.

Conclusions

In sum, eighteen years of research support a moderate-to-large relationship of EA with depression, anxiety and related disorders, suggesting that the attention it has drawn as a pathological process and treatment target is well-merited. More studies are needed to examine how EA is associated with anxiety and depression, not as a trait but as a process in context. While the results support the transdiagnostic nature of EA, moderator analyses suggest variability in how EA develops and operates within disorders, and research into these interactions may help shed light on EA more broadly. The variability in effect sizes across measurement types, and the high correlations EA measures share with trait anxiety and distress measures, suggest serious concerns in whether EA is being measured as intended. A new wave of state EA measures (Kashdan et al., 2014) and contextualized EA measures (Kashdan et al., 2020) may lead to a more precise understanding of the impact of EA. Measuring and analyzing EA as a dynamic process and considering non-linear relationships between EA and psychopathology may also lead to new breakthroughs.

Although more precise and contextualized research is needed, these findings support the role of EA as a transdiagnostic, functional contextual process consistently linked to depression and anxiety disorders. As such, it is a strong candidate both for ongoing research and as a clinical treatment target, as psychology shifts away from the syndromal system of the DSM and towards a process-based therapy model.

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Table 1*Study Characteristics*

Variables	<i>k</i>	Reported Demographics		Mean/%
		<i>S</i>	<i>N</i> (%participants)	
Final sample	389	441	135,347 (100)	
Publication date (2002-2020)	389	441	135,347 (100)	
2002-2008	29	37	6,137	4.80%
2008-2014	101	118	27,151	20.42%
2014-2020	259	286	102,059	77.81%
Quality (10-18)	389	441	135,347 (100)	
10-12	2	2	400	0.29%
12-14	18	24	2,292	1.69%
14-16	98	115	16,698	12.34%
16-18	271	300	115,957	85.67%
% female		414	123,732 (91.42%)	66.16%
Age (13.6-82.5 years)		385	119,613 (88.38%)	31.53%
13.6-18		4	731 (0.54%)	0.61%
18-30		148	65,319 (48.26%)	54.61%
30-45		153	37,661 (27.83%)	31.49%
45-60		62	12,108 (8.95%)	10.77%
60-82.5		18	3,794 (2.80%)	3.17%
% married		145	54,563 (40.31%)	35.43%
% employed		104	24,559 (18.15%)	55.02%
Education level		183	72,056 (53.24%)	
Undergraduate %		123	58,748(43.41%)	81.52%
Geographic region		441	135,347 (100%)	
North and South America		242	86,413 (63.84%)	63.84%
Europe		132	32,981 (24.37%)	24.37%
Asia		32	9,869 (7.29%)	7.29%
Australia		22	2,422 (1.79%)	1.79%
Africa		1	65 (0.048%)	0.048%
International		12	3,597 (2.66%)	2.66%
Race/Ethnicity		380	118,321 (87.42%)	
White			79,393 (58.66%)	67.1%
Black			17,594 (12.99%)	14.87%
Latino			1,775 (1.31%)	1.5%
Asian			13,457 (9.94%)	11.37%
Mixed			6,105 (4.51%)	5.16%
Data collection method		314	101,638 (75.09%)	
In-person		155	32,232 (23.81%)	31.71%
Online		146	66,134 (48.86%)	65.07%
Mixed		12	3,225 (2.38%)	3.17%
Telephone		1	47 (0.035%)	0.046%
Population type		441	135,347 (100%)	
General		253	96,434 (71.25%)	71.25%
Physical health treatment seekers		100	23,667 (17.48%)	17.48%
Psychological treatment seekers		88	15,246 (11.26%)	11.26%

Note. *k* = number of studies; *S* = number of samples; *N* = number of participants.

Table 2

Significant Categorical Moderator Results

Variables	<i>k</i>	<i>N</i>	<i>r</i>	95% CI		<i>Z</i>	<i>Q</i>	<i>I</i> ² (%)	τ^2
				Lower	Upper				
Depressive Symptoms									
EA measure type (<i>Q</i> = 128.153, <i>df</i> = 4, <i>p</i> = .000)									
AAQ-I	64	12,650	.500	.466	.532	24.485***	411.904***	84.705	0.029
AAQ-II	186	76,588	.623	.608	.638	58.024***	1,672.918***	88.941	0.021
CPAQ	51	11,125	.497	.461	.532	22.789***	348.027***	85.633	0.028
Context-specific	36	8,114	.428	.380	.473	15.746***	313.652***	88.841	0.037
MEAQ/BEAQ	15	4,549	.488	.421	.550	12.313***	18.375	23.808	0.001
Anxiety Symptoms									
EA measure type (<i>Q</i> = 33.843, <i>df</i> = 4, <i>p</i> = .000)									
AAQ-I	42	11,265	.474	.428	.518	17.435***	254.908***	83.916	0.020
AAQ-II	119	32,398	.551	.527	.575	35.850***	1,151.918***	89.756	0.033
CPAQ	24	5,472	.420	.357	.480	11.770***	133.113***	82.721	0.022
Context-specific	25	4,584	.413	.350	.473	11.572***	167.205***	85.646	0.034
MEAQ/BEAQ	7	1,589	.461	.344	.564	6.975***	12.195	50.801	0.005
Population type (<i>Q</i> = 6.065, <i>df</i> = 2, <i>p</i> = .048)									
General	120	35,167	.523	.497	.547	33.382***	1199.714***	90.081	0.032
Psychological treatment seekers	40	8,392	.504	.455	.549	17.135***	196.643***	80.167	0.021
Physical health treatment seekers	49	9,050	.461	.417	.503	17.887***	341.026***	85.925	0.034
Geographic region (<i>Q</i> = 11.096, <i>df</i> = 4, <i>p</i> = .026)									
North and South America	88	22,577	.536	.507	.564	29.473***	664.512***	86.908	0.026
Europe	81	21,650	.485	.453	.516	25.290***	699.946***	88.571	0.030
Asia	17	4,603	.443	.371	.510	10.727***	202.464***	92.097	0.044
Australia	14	1,426	.469	.381	.550	9.190***	35.395***	63.272	0.018
International	9	2,353	.556	.462	.638	9.643***	51.444***	84.449	0.022
GAD									
Data collection method (<i>Q</i> = 15.001, <i>df</i> = 1, <i>p</i> = .000)									
In person	5	1,327	.517	.453	.576	13.413***	1.564	0.000	0.000
Online	11	26,178	.645	.614	.673	29.312***	74.209***	86.525	0.005
Population type (<i>Q</i> = 8.708, <i>df</i> = 1, <i>p</i> = .003)									
General	11	26,121	.615	.574	.653	22.151***	121.027***	91.737	0.009
Psychological treatment seekers	6	1,067	.492	.413	.564	10.579***	4.274	0.000	0.000

<i>Worry</i>									
(<i>Q</i> = 5.624, <i>df</i> = 1, <i>p</i> = .018)									
Population type									
General	18	6,496	.564	.496	.625	13.240***	213.342***	92.032	0.037
Psychological treatment seekers	9	2,181	.405	.277	.519	5.793***	48.209***	83.406	0.029
<i>Specific Phobia</i>									
(<i>Q</i> = 6.760, <i>df</i> = 2, <i>p</i> = .034)									
Population type									
General	7	1,424	.386	.315	.452	9.882***	13.302*	54.896	0.006
Psychological treatment seekers	5	475	.384	.280	.479	6.780***	8.816	54.628	0.015
Physical health treatment seekers	6	1,992	.493	.434	.547	14.054***	9.963	49.817	0.003
Comorbidity									
(<i>Q</i> = 8.750, <i>df</i> = 1, <i>p</i> = .003)									
No comorbidity	9	1,585	.377	.317	.433	11.484***	13.842	42.203	0.004
Physical condition	6	1,992	.493	.441	.542	15.858***	9.963	49.817	0.003
<i>OCRDs</i>									
(<i>Q</i> = 7.482, <i>df</i> = 1, <i>p</i> = .006)									
Population type									
General	13	3,482	.457	.404	.507	14.759***	48.865***	75.442	0.012
Psychological treatment seekers	15	3,358	.347	.286	.405	10.479***	34.861**	59.840	0.008
Geographic region									
(<i>Q</i> = 8.242, <i>df</i> = 1, <i>p</i> = .004)									
North and South America	19	5,061	.361	.308	.411	12.524***	81.057***	77.793	0.014
Europe	6	1,268	.507	.422	.583	10.121***	4.137	0.000	0.000
Clinical status									
(<i>Q</i> = 21.309, <i>df</i> = 1, <i>p</i> = .000)									
Clinical	7	1,268	.269	.189	.345	6.413***	6.041	0.671	0.000
At-risk	18	4,299	.464	.426	.501	20.818***	43.554***	60.968	0.007
Disorder									
(<i>Q</i> = 7.432, <i>df</i> = 1, <i>p</i> = .006)									
OCD	19	4,000	.417	.370	.462	15.556***	65.984***	72.721	0.013
Trichotillomania	5	2,101	.268	.166	.365	5.011***	1.831	0.000	0.000
<i>PTSD</i>									
(<i>Q</i> = 52.093, <i>df</i> = 2, <i>p</i> = .000)									
EA measure type									
AAQ-I	22	3,609	.370	.313	.424	11.816***	66.579***	68.458	0.014
AAQ-II	34	9,800	.590	.555	.622	26.143***	199.541***	83.462	0.018
MEAQ/BEAQ	8	2,209	.419	.331	.500	8.525***	16.610*	57.856	0.006
Geographic region									
(<i>Q</i> = 3.938, <i>df</i> = 1, <i>p</i> = .047)									
North and South America	56	14,157	.500	.460	.539	20.511***	509.825***	89.212	0.034
Europe	7	1,418	.374	.242	.492	5.272***	24.077***	75.080	0.016

Note. k = number of studies; N = number of participants; r = mean weighted effect size; CI= confidence interval; z = z value of the significance test; Q = ratio of variation to within-study error; I^2 = proportion of total observed variation attributable to between-study effects; τ^2 = between-study variance.

* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 3

Continuous Moderator Results

Moderators	<i>b</i>	<i>SE</i>	95% CI		<i>z</i>	τ^2	<i>Q_M</i>	<i>R</i> ²	<i>k</i>
			Lower	Upper					
Depressive Symptoms									
EA α	0.943	0.135	0.680	1.207	7.01	0.0283	49.12 ^{***}	.27	197
Outcome α	1.295	0.260	0.785	1.805	4.97	0.0305	24.75 ^{***}	.10	180
Publication year	0.010	0.003	0.005	0.016	3.71	0.0331	13.73 ^{***}	.12	330
Mean age	-0.002	0.001	-0.004	0.000	-2.01	0.0316	4.05 [*]	.06	283
% Employed	0.002	0.001	0.000	0.003	2.45	0.0291	6.02 [*]	.09	82
Mean EA	-0.052	0.010	-0.071	-0.033	-5.34	0.0289	28.49 ^{***}	.22	216
GHQ	0.066	0.021	0.024	0.107	3.09	0.0208	9.53 ^{***}	.26	10
ASI	-0.015	0.005	-0.024	-0.006	-3.34	0.0206	11.17 ^{***}	.63	14
Anxiety Symptoms									
EA α	0.583	0.165	0.260	0.906	3.53	0.0303	12.49 ^{***}	.07	117
Outcome α	0.879	0.234	0.420	1.338	3.76	0.0234	14.10 ^{***}	.11	109
Mean age	-0.002	0.001	-0.004	-0.001	-2.58	0.0241	6.66 [*]	.06	176
Years of education	0.027	0.007	0.013	0.041	3.90	0.0088	15.22 ^{***}	.38	26
Mean EA	-0.052	0.012	-0.074	-0.029	-4.44	0.0229	19.69 ^{***}	.13	134
GHQ	0.056	0.021	0.016	0.097	2.72	0.0230	7.39 ^{**}	.17	11
ASI	-0.008	0.003	-0.014	-0.002	-2.71	0.0196	7.35 ^{**}	.13	25
SAD									
Mean EA	-0.054	0.023	-0.099	-0.009	-2.36	0.0179	5.57 [*]	.21	23
GAD									
EA α	1.498	0.283	0.943	2.053	5.29	0.0042	28.00 ^{***}	.62	16
Outcome α	4.23	1.22	1.85	6.62	3.48	0.0055	12.10 ^{***}	.23	14
Study quality	0.066	0.022	0.023	0.109	3.03	0.0104	9.19 [*]	.15	21
Publication year	0.029	0.006	0.018	0.041	4.97	0.0054	24.67 ^{***}	.56	21
% Married	-0.007	0.002	-0.011	-0.002	-2.71	0.0057	7.37 ^{**}	.13	10
Mean EA	-0.055	0.023	-0.099	-0.010	-2.41	0.0102	5.81 [*]	.07	17
Depression score	-0.055	0.016	-0.086	-0.023	-3.44	0.0043	11.83 ^{***}	.37	15
Specific phobia									
Mean age	0.01	0.00	0.00	0.01	3.53	0.0001	12.46 ^{***}	.98	14
Worry									
Outcome α	1.456	0.527	0.423	2.49	2.76	0.0286	7.63 [*]	.34	21
OCRDs									
EA α	-0.901	0.441	-1.765	-0.036	-2.04	0.0034	4.17 [*]	.58	12
Outcome mean	-0.044	0.016	-0.075	-0.012	-2.73	0.0109	7.45 ^{**}	.47	17
Depression score	-0.087	0.032	-0.149	-0.025	-2.76	0.0096	7.60 ^{**}	.45	12
PTSD									
EA α	0.961	0.167	0.635	1.288	5.77	0.0171	33.26 ^{***}	.46	47
Publication year	0.015	0.005	0.005	0.024	2.95	0.0304	8.73 ^{**}	.12	65
% Employed	0.005	0.001	0.002	0.007	3.61	0.0105	13.05 ^{***}	.52	14

Years of education	0.025	0.006	0.014	0.036	4.48	0.014	20.04***	0.50	25
Mean EA	-0.072	0.024	-0.119	-0.024	-2.96	0.0331	8.78**	.16	40
Outcome mean	0.053	0.017	0.019	0.087	3.06	0.0334	9.38**	.09	51

Note. b = regression coefficient; SE = standard error; CI = confidence interval; z = z value of the significance

test; R^2 = proportion of variance explained; k = number of samples.

* $p < .05$, ** $p < .01$, *** $p < .001$

Table 4*Results of Publication Bias Analyses*

Variable	Trim & Fill						Egger's reg.
	Observed			Imputed			
	<i>k</i>	<i>Obs. r</i>	95% CI	<i>N</i> (Direction)	<i>Adj. r</i>	95% CI	
MDD	6	.453	[.395,.508]	1 (L)	.434	[.367,.497]	0.193
Dep. Sym.	330	.562	[.546,.577]	59 (L)	.518	[.498,.537]	-2.271***
Anx. Sym.	209	.506	[.486,.525]	0	.506	[.486,.525]	-0.568
SAD	34	.461	[.416,.505]	1 (R)	.467	[.422,.511]	1.55
GAD	21	.588	[.551,.623]	4 (R)	.608	[.574,.640]	-3.13***
Panic & agoraphobia	11	.340	[.276,.401]	1 (L)	.332	[.265,.397]	0.203
Specific phobia	18	.431	[.383,.478]	1 (R)	.440	[.391,.487]	-1.26
Worry	29	.516	[.457,.570]	4 (R)	.555	[.500,.605]	-2.38*
OCRDs	28	.406	[.357,.453]	0	.406	[.357,.453]	1.448
PTSD	65	.489	[.450,.526]	5 (R)	.510	[.471,.546]	-1.139

Note. *Obs. r* = observed average effect size; *Adj. r* = adjusted average effect size. CI = confidence interval;

Egger's Reg = Egger's Regression; Dep. Sym. = Depression symptoms; Anx. Sym. = Anxiety symptoms; L = Left; R = Right.

* $p < .05$, *** $p < .001$.

Figure 1

PRISMA Flow Diagram

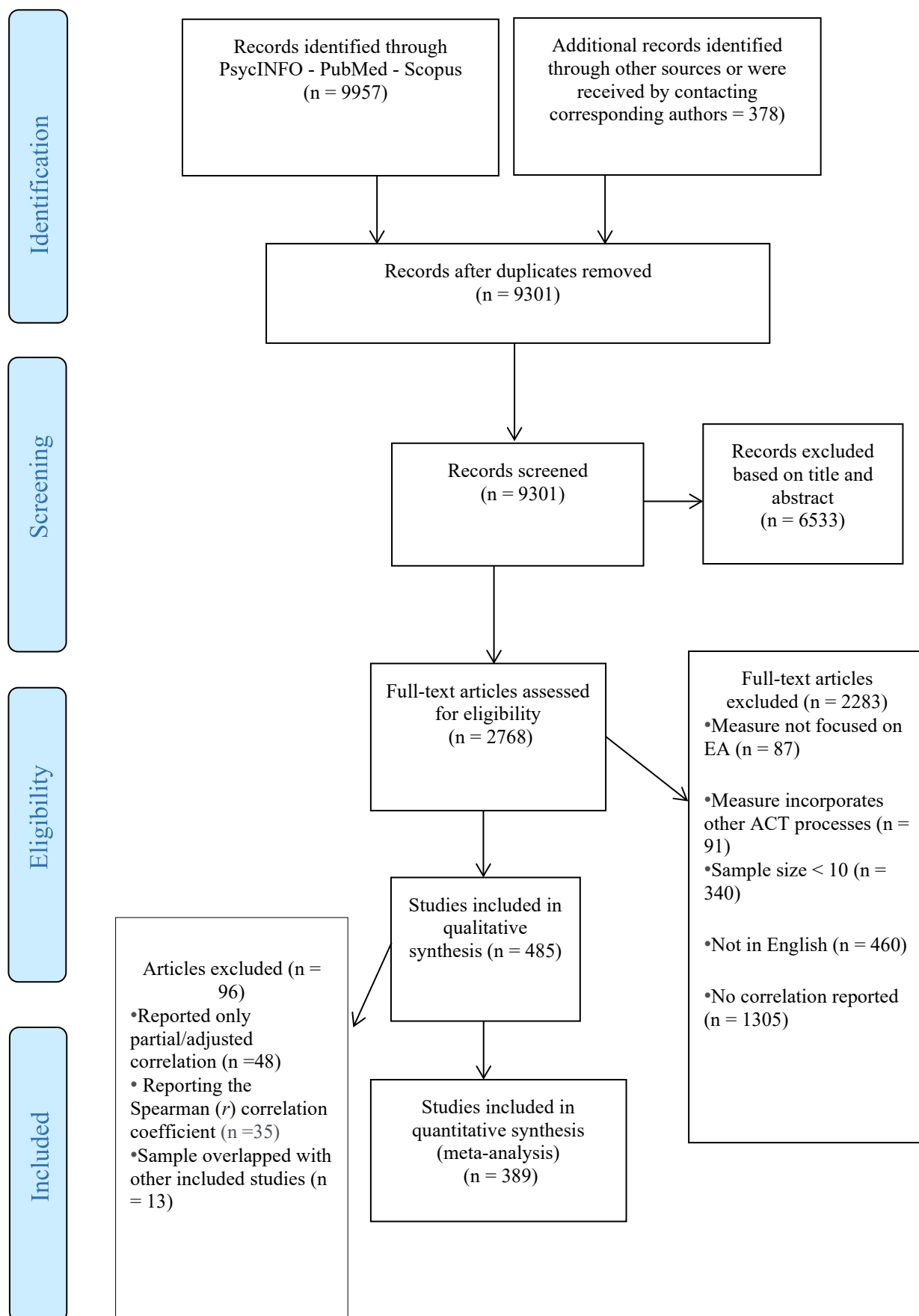


Figure 2

Forest Plot

