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Emotion Regulation Strategies in Cognitive Behavioral Therapy for Panic Disorder

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Emotion regulation (ER) has been incorporated into many models of psychopathology, but it has not been examined directly in cognitive behavioral therapy (CBT) for panic disorder with agoraphobia (PD/A). In this study, a preliminary model of ER in CBT for PD/A is proposed based on existing theories, and several propositions of the model are tested. We hypothesized that increases in cognitive reappraisal would precede decreases in biased cognitions, decreases in expressive suppression would follow decreases in biased cognitions, and a reduction in symptom severity would follow decreases in expressive suppression. Twenty-nine patients who received CBT for PD/A completed weekly self-report measures of symptom severity, anxiety sensitivity, reappraisal and expressive suppression. In addition, patients were compared to a matched normal sample. Cross-lagged analyses partially supported the hypotheses. Reappraisal did not change until late stages of therapy and was generally not associated with treatment outcome. Suppression decreased significantly and exhibited a reciprocal relationship with biased cognitions. Symptom reduction followed decreases in suppression as hypothesized. However, patients did not differ in ER from matched controls at either pre- or posttreatment. Results suggest the important distinction between reappraisal and appraisal, and stress the role of session-by-session decreases in suppression as a predictor of symptom reduction.

Keywords: panic disorder and agoraphobia; cognitive behavioral therapy; emotion regulation; reappraisal; expressive suppression

PANIC DISORDER AND AGORAPHOBIA (PD/A) is characterized by recurrent unexpected panic attacks and anxiety about experiencing additional attacks, resulting in avoidance of related situations (American Psychiatric Association, 2013). PD/A is prevalent, debilitating (Kessler, Chiu, Demler, & Walters, 2005), and can have significant negative consequences on the physical and mental health of patients and their environment, as well as a significant economic burden (Kroenke, Spitzer, Williams, Monahan, & Löwe, 2007). Cognitive-behavioral therapy (CBT) is considered the gold standard for treating PD/A, but effects are still smaller than those of CBT for other anxiety disorders (Carpenter et al., 2018). In addition, effects in naturalistic settings are somewhat smaller than those obtained in more controlled conditions (Stewart & Chambless, 2009). Identifying mechanisms of change of CBT for PD/A has the potential to provide insights that could be used for improving existing protocols. The current study utilized an intensive measurement design with advanced data analytic techniques to study emotion regulation (ER) as a proposed mechanism of change in CBT for PD/A.

In recent years, emotion dysregulation (difficulties in effectively managing one's emotions; Gross, 2015) has received increasing theoretical and empirical attention as a possible central impairment across many forms of psychopathology in general (e.g., Campbell-Sills & Barlow, 2007; Sheppes,

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Suri, & Gross, 2015) and specifically in anxiety disorders (e.g., Hofmann, Sawyer, Fang, & Asnaani, 2012). Indeed, the role of emotion dysregulation has been extensively studied in some anxiety disorders such as social anxiety disorder (for a review, see Jazaieri, Morrison, Goldin, & Gross, 2015). Related processes that are indicative of the role emotion regulation plays in PD/A have been examined in numerous studies. For example, studies have examined the role of catastrophic cognitions (Clark, 1986; McNally, 2002; Teachman, Smith-Janik, & Saporito, 2007), as well as engagement in safety-seeking behaviors (Salkovskis, Clark, Hackmann, Wells, & Gelder, 1999) in the maintenance of PD/A. In addition, CBT includes empirically supported techniques that are hypothesized to operate by promoting adaptive emotion regulation skills such as cognitive restructuring and interoceptive exposure (Arntz, 2002; Bouchard et al., 1996). These studies provide support to the idea that emotion regulation plays an important role in PD/A and its treatment, but a direct examination of emotion regulation strategies as they unfold over time is still needed in order to establish the effects of active use of such strategies. Few studies have examined emotion dysregulation in PD/A, and to the best of our knowledge, no studies have examined changes in self-reported use of emotion regulation strategies during treatments of PD/A.

Two emotion regulation strategies are of particular relevance for PD/A: cognitive reappraisal and expressive suppression. Cognitive reappraisal, reinterpreting emotional stimuli in less threatening ways in order to regulate negative emotions, is considered an adaptive strategy (Gross, 2015). Reappraisal is a very similar concept to cognitive restructuring, and thus reduced use of reappraisal might explain some of the catastrophic interpretations of anxiety-provoking situations (e.g., "catastrophic thinking") that play an important role in cognitive theories of PD/A (Clark, 1986; McNally, 2002; Teachman et al., 2007).

Expressive suppression, inhibiting the expression of one's emotions, is generally considered maladaptive (Gross, 2015). Overreliance on suppression may play a role in PD/A as means of control over physical sensations, given that many individuals with PD/A are concerned that others might notice their symptoms of anxiety and therefore unsuccessfully try to hide them, which often results in further exacerbation of those symptoms (Salkovskis et al., 1999).

Additional support for the possible role of reappraisal and suppression in PD/A comes from the unified model for the treatment of emotional disorders and the studies that have empirically tested the model (Barlow, Allen, & Choate, 2016). According to the model, emotional disorders (i.e., anxiety and depressive disorders) are characterized with specific emotion regulation impairments, which include, among others, impaired reappraisal and emotional avoidance, broadly defined (excessive attempts to avoid the experience and expression of negative emotions). Studies informed by the unified model have demonstrated the contribution of these impairments to emotional disorders (e.g., Campbell-Sills, Barlow, Brown, & Hofmann, 2006; Sloan & Telch, 2002). Collectively, these findings lend indirect support to specific emotion regulation impairments in PD/A, but direct examination of these processes in PD/A is still warranted.

The few studies that examined the use of reappraisal and suppression among individuals with PD/A provide inconclusive evidence. Breuninger, Sláma, Krämer, Schmitz, and Tuschen-Caffier (2017) found that PD/As endorsed less habitual use of reappraisal compared to healthy controls (HC) on a self-report measure, although Manber Ball, Ramsawh, Campbell-Sills, Paulus, and Stein (2013) did not find such differences using the same measure (based on our own statistical analyses of descriptive statistics reported in the original paper). In addition, no differences were found in spontaneous (uninstructed) use of lab-based reappraisal. Similar to Breuninger et al. (2017), Manber Ball et al. (2013) and Reinecke et al. (2015) showed that PD/As were able to down-regulate their emotions using reappraisal in response to negative images during a lab-based task and that this reduction was similar to that observed among HCs. Thus, it seems like reappraisal reduces negative emotional responses in PD/A (albeit to a similar extent to HCs), which points to the salutary effect of reappraisal among PD/As and to the potential importance of increasing use of frequency of reappraisal in treatment.

In terms of suppression, two studies found greater self-reported use of habitual suppression among individuals with PD/A compared to HCs (Baker, Holloway, Thomas, Thomas, & Owens, 2004; Manber Ball et al., 2013) whereas one study failed to find such differences (Breuninger et al., 2017). Finally, Breuninger et al. did not find differences between PD/As and HCs in spontaneous use of lab-based suppression. Therefore, the findings regarding the habitual use of and actual success in reappraisal and suppression among individuals with PD/A compared to HCs are still inconclusive and merit additional research.

Treatment studies that examine changes in reappraisal and suppression during treatment of PD/A and their role in treatment outcome may potentially inform the question of whether emotion

regulation is impaired in PD/A, but we are not aware of any studies that directly examined these questions. Smits, Julian, Rosenfield, and Powers (2012) reviewed studies that examined reductions in threat appraisals (i.e., catastrophic thinking) as mediators of outcome in treatments of anxiety disorders, including PD/A. They concluded that although these reductions are indeed related to better outcome, few studies have attempted to establish them as a specific *cause* of outcome. However, the studies included in the Smits et al. (2012) review examined the role of threat *appraisals* and not *reappraisal*. Thus, the review deals with the possible mediating role of reduction in catastrophic thinking (and not *reappraisal* per se) in CBT for PD/A. Such a mediating role could be due to various processes such as exposure or an increase in use of other emotion regulation strategies (cf. Lorenzo-Luaces, Keefe, & DeRubeis, 2016, for the differences between techniques and mechanisms). A direct examination of threat *reappraisal* is still needed to establish the impact of the conscious, active process of emotion regulation via reappraisal and whether it underlies the reduction in catastrophic thinking.

In sum, additional studies are needed to clarify the role of emotion regulation in CBT for PD/A given the absence of such studies to date. However, findings from studies examining the role of emotion regulation in CBT for related anxiety disorders can serve as a basis for forming hypotheses about CBT for PD/A. For example, studies have shown that reappraisal increases in CBT for social anxiety disorder and that these increases generally predict subsequent reductions in anxiety symptoms, though findings regarding suppression are less conclusive (Goldin et al., 2014; Moscovitch et al., 2012). In addition, given the benefits of reappraisal and the costs of suppression in general as well as in terms of anxiety (e.g., Gross & John, 2003), and the specific role of catastrophic cognition and attempts to conceal panic symptoms in the maintenance of PD/A and its treatment (Gallagher et al., 2013), individuals with PD/A may benefit from interventions targeting these strategies.

Several techniques of CBT for PD/A seem of particular relevance for promoting adaptive emotion regulation including psychoeducation, cognitive restructuring, interoceptive exposures and in-vivo exposures. Specifically, the cognitive restructuring module might be particularly beneficial in increasing the use of reappraisal because it directly targets patients' biased catastrophic cognitions regarding their panic symptoms via reappraisal techniques. (Due to the conceptual overlap between these constructs, and to avoid possible confusion, we chose to refer to cognitive reappraisal and not cognitive restructuring throughout the manuscript. The only exception is when we refer to the cognitive restructuring module, which is the name of the module as it appears in many CBT protocols for PD/A.) To the degree that psychoeducation, interoceptive exposure, and in-vivo exposure challenge biased cognitions, they are expected to facilitate greater use of reappraisal as well. At the same time, interoceptive exposure likely decreases reliance on suppression, as patients learn to acknowledge that their symptoms are not necessarily dangerous and therefore need not be concealed. Similarly, other modules possibly decrease the use of suppression to the degree that patients learn that warding off one's symptoms is futile and an unnecessary endeavor.

Based on the abovementioned considerations and findings, we propose a preliminary model of the role of ER in CBT. The model is not intended to replace other existing models of PD/A but rather to integrate existing models and findings with contributions from emotion regulation theory in order to guide theory and research on the role of emotion regulation in PD/A. Figure 1, panel A, illustrates the hypothesized temporal course of ER and symptoms in the course of CBT. Early in treatment, patients acquire reappraisal skills through psychoeducation and cognitive restructuring. The acquisition of these skills then leads to a reduction in catastrophic cognition (i.e., decreased fear of panic symptoms and reduced anxiety sensitivity). These reductions are further enhanced by interoceptive and *in vivo* exposures, which subsequently lead to reductions in suppression, as patients develop tolerance of their physical symptoms and become less concerned about experiencing panic attacks. Finally, these combined changes in emotion regulation and cognitions then lead to reduced symptoms of panic disorder, namely, reductions in panic attacks and in avoidance symptoms (Gallagher et al., 2013).

The aim of the present study is to provide a preliminary test for some of the main propositions of the ER model of CBT for PD/A as well as several alternative hypotheses that may help to disconfirm and revise parts of it. These hypotheses were examined in a sample of 29 patients who received manualized CBT for PD/A. An intensive measurement design was utilized in attempt to characterize the dynamic relationships between ER and the outcome of CBT using advanced data-analytic techniques that have not been widely applied in the study of CBT for PD/A to date.

Specifically, we examined the following hypotheses: use of reappraisal will increase throughout the course of CBT (H1) and especially following the cognitive restructuring module (H2); use of

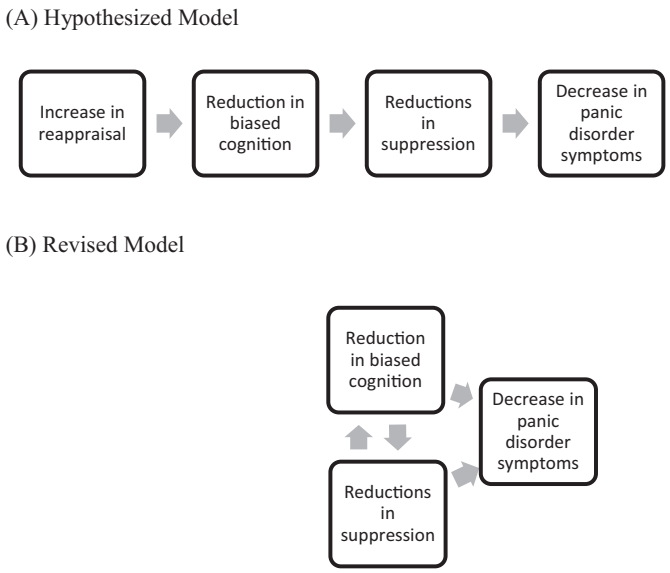

FIGURE 1 The Hypothesized (Panel A) and Revised (Panel B) Models of Temporal Course of Reappraisal, Suppression and PD/A Symptom Variables During CBT

suppression will decrease throughout the course of CBT (H3); increases in reappraisal will predict subsequent reduction in biased cognition in CBT (H4); reductions in biased cognitions will predict subsequent reductions in suppression (H5); reductions in suppression will predict subsequent reductions in panic symptoms (H6). On an exploratory basis, we examined whether measures of ER at pretreatment moderated changes in PD/A symptoms during treatment (H7). Finally, we also examined differences in reappraisal and suppression between patients and matched individuals from the normal population (NPs) at the beginning and end of treatment (H8). Given the inconclusive findings in the literature, we did not have a priori hypotheses regarding the direction of these differences.

Method

PARTICIPANTS

The study was conducted at the Hebrew University of Jerusalem in Israel. Twenty-nine patients were recruited between 2009–2014 via advertisements in central locations in Jerusalem and referrals. The sample was therefore a community sample, though students from the university were able to participate. Inclusion criteria included age of 18 or older, PD/A as the primary diagnosis according to the Mini International Neuropsychiatric Interview 4.5 (MINI 4.5; Sheehan et al., 1998), and score equal or above 10 on the Panic Disorder Severity Scale–Independent Evaluator Version (PDSS-IE, Shear et al., 2001) at intake. Both the MINI and the PDSS-IE were administered in the initial intake meeting, in the presence of two clinicians, to strengthen the reliability of the diagnoses: the patient's future therapist and the therapist's supervisor (the last author).

Exclusion criteria included history of CBT for PD/A, history of psychosis, current diagnosis of bipolar disorder, severe suicidality and active substance abuse/dependence. Comorbidities were allowed as long as PD/A was the primary diagnosis. Eleven participants had one or more comorbid diagnoses (2 met criteria for major depressive disorder, 1 dysthymic disorder, 4 obsessive-compulsive disorder, 6 generalized anxiety disorder, 4 social anxiety disorder, 1 posttraumatic stress disorder). Demographics, pretreatment severity scores, and pretreatment ER scores are presented in Table 1. Participants received psychotherapy for free and did not receive additional compensation for their involvement in this study. The university ethics board approved the study. Informed consent was obtained from all participants prior to participation. Additional findings based on this sample are reported elsewhere (Weiss, Kivity, & Huppert, 2014; Zalaznik, Weiss, & Huppert, 2017) but the analyses presented here are novel.

THERAPISTS AND TREATMENT

Twenty-nine therapists, master's students in clinical psychology, participated in this study as part of a research seminar on psychotherapy process and outcomes at the university. All therapists had experience treating other patients in their practicum sites. Treatment was based on Craske and Barlow's (2007) protocol for treating PD/A with elaborations

Table 1
Descriptive Statistics for Demographic, Baseline Symptoms, Biased Cognition and Emotional Regulation Measures for Patients in Study ($N = 29$)

	Percent	
Female	62%	
	M	*SD*
Age	34.48	11.13
Symptom severity		
PDSS	12.55	4.53
Biased cognitions		
ASI	42.17	12.78
Emotion regulation		
CR	4.46	0.97
ES	3.78	1.24

Note. PDSS = panic disorder severity scale; ASI = anxiety sensitivity index; CR = cognitive reappraisal; ES = expressive suppression.

from Huppert and Baker-Morissette (2004) and included 12 sessions. The initial sessions were dedicated to psychoeducation and the cognitive restructuring module, the next sessions were focused on interoceptive exposures, and the later sessions were dedicated to in-vivo exposures (sometimes combined with interoceptive exposures). Finally, the last session (s) focused on consolidation of gains and relapse prevention. Patients received homework after each session. Therapists were trained and supervised by the last author, a clinical psychologist with extensive expertise in treating and supervising CBT of PD/A. All in-office treatment sessions were videotaped for supervision and adherence monitoring. An independent review of each of the patient files revealed that all patients received psychoeducation, cognitive restructuring, interoceptive exposure, and in vivo exposure. Although patients progressed through identical treatment phases, some variation was allowed when moving from stage to stage and when determining the total length of treatment. For instance, 11 sessions were permitted if patients improved sufficiently earlier, and 13 sessions if an additional session was needed. The average number of sessions was 11.34 with only 4 patients finishing prematurely and receiving less than 10 sessions (minimum 6 sessions). The exact number of patients at each timepoint can be found in Section 1, Table S1 in the supplementary material.

MEASURES

Mini International Neuropsychiatric Interview 4.5 (MINI 4.5)

The MINI 4.5 is a structured interview of Axis-I disorders of the DSM-IV-TR (Sheehan et al., 1998).

Panic Disorder Severity Scale–Independent Evaluator Version (PDSS-IE)

The PDSS-IE is a valid and reliable measure of the severity of the panic disorder (Shear et al., 2001). The internal reliability for the PDSS-IE in our sample was very good (Cronbach's $\alpha = .87$).

Panic Disorder Severity Scale–Self-Report Version (PDSS-SR)

The PDSS-SR is a self-report version of the respective independent evaluator measure, which has been shown to have good psychometrics and sensitivity to change (Houck, Spiegel, Shear, & Rucci, 2002). The PDSS-SR has 7 items, rated on a 5 point Likert scale (0–4). The internal reliability for the PDSS-SR in our sample was very good (α range across timepoints = .84–.95).

Anxiety Sensitivity Index-3 (ASI-3)

The ASI-3 served as a measure of biased catastrophic cognitions in the current study. The ASI-3 is an 18-item self-report scale used to evaluate somatic, cognitive, and social facets of beliefs regarding panic-related bodily sensations. Each item is rated on a 5-point Likert scale (0–4). It has been used extensively in research on panic disorder, and has been found to have strong psychometrics (Taylor et al., 2007). The internal reliability for the ASI in our sample was excellent (α across timepoints = .90–.98).

Emotion Regulation Questionnaire (ERQ)

The ERQ was developed to measure the frequency of use of two emotion regulation strategies: cognitive reappraisal and expressive suppression. Six items pertain to reappraisal (e.g., "When I want to feel less negative emotion, I change the way I'm thinking about the situation") and 4 items pertain to suppression (e.g., "I control my emotions by not expressing them") rated on a 7-point Likert scale. The ERQ has been used extensively and has been found to exhibit good psychometric properties (e.g., Gross & John, 2003). The internal reliability for both reappraisal and suppression was good (α across timepoints for reappraisal = .86–.95; for suppression = .78 –.96).

NORMAL POPULATION SAMPLE

To compare our patients to a normal population sample, we used a matched subsample taken from a student sample from a recently completed study (Kivity, Tamir, & Huppert, 2016). In the original study, 267 participants (202 females, age: 19–64, $M = 26.91$, $SD = 6.64$) completed a series of questionnaires online, including the ERQ. In the current study, subjects from the normal sample were matched to PD/As on gender and age (±3

years). When more than one match was available in the normal sample, we randomly assigned a matching subject from the available subjects. We were unable to match three older patients, and therefore only 26 patients are compared to this normal subsample (15 females, age: 23–64, M = 32.42, SD = 12.01).

PROCEDURE

Patients were interviewed by the therapist and the last author who supervised both the interview and therapy. Measures were all translated into Hebrew and then backtranslated into English by a separate individual to ensure valid translations. All reported measures were administered on a computer in the clinic at baseline and before each session. The ASI and the ER measures were collected also after each session. Because this article does not focus on differentiating within- and between-session processes, these post measures were not analyzed and therefore not reported here.

DATA PREPARATION AND DATA ANALYTIC APPROACH

Data were analyzed using R software version 3.4. First, data were screened to assess violations of normality and existence of outliers. This was done by visually examining histograms of study variables at each timepoint, as well as computing skewness and kurtosis indexes and examining z scores of outliers for each timepoint separately. No violations of normality or existence of systematic outliers were apparent. Hypotheses 1 and 3 examined changes in ER measures during treatment. For this analysis we used full intent-to-treat longitudinal mixed effects models (LMLM) via 'nlme' package in R (Pinheiro, Bates, DebRoy, & Sarkar, 2016). Models were adjusted for repeated measures with restricted maximum likelihood estimation (REML), a first-order autoregressive covariance structure at the time level and random intercepts and slopes at the patient level. Linear, quadratic and log-linear curves were estimated and compared in terms of model fit. Hypothesis 2 examined changes in reappraisal during the cognitive restructuring module. To determine which sessions were dedicated to the module, treatment sessions were coded separately for each patient based on process notes provided by therapists regarding the content of each session. We compared reappraisal scores measured before the cognitive restructuring module with the reappraisal scores measured before the subsequent meeting using paired t-test.

Hypotheses 4–6 examined the cross-lagged, time-varying relationship between ER and PD/A symptoms. LMLM methods were used following similar procedures to those recommended by Wang and Maxwell (2015). Consistent with their recommendations, time varying predictors were person-mean centered. Also, we did not control for time effects in our data because the variables of interest were expected to show changes during treatment and controlling for time might have resulted in a removal of the very phenomena that we were attempting to capture. Two models were fitted for each ER-symptom measures pair to examine the relationship between their changes over time. In the first model, we examined the prediction of symptoms from ER in the previous timepoint (i.e., let t be the index for the session number, level of symptoms at session t were predicted by level of ER scores at session t-1). In the second model, we reversed the roles and examined the prediction of ER from symptoms in the previous timepoint (level of ER scores at session t were predicted by level of symptom scores at session t-1). The reader is referred to the supplementary material for a graphical representation (Fig. S1 in Section 2) as well as equations for these models (Section 3). This approach can facilitate a better understanding of the directionality of the relationship between the variables, and also potentially establishes temporality. Although it is likely that levels of the dependent variable have causal effects on subsequent levels of the dependent variable, we did not include the lagged dependent variable as a predictor, following the recommendations of Falkenström, Finkel, Sandell, Rubel, and Holmqvist (2017) because such an approach introduces a dependency between the lagged dependent variable and the error term, thus violating one of the assumptions of regression-based models (the "endogeneity" problem). However, we partly accounted for the possible lagged effects of the dependent variable by including a first-order autoregressive (AR1) residual structure. The AR1 structure accounted for the correlations among observations of the dependent variable that are close in time and at the same time avoided the problem of endogeneity because the correlation between two adjacent observations of the dependent variable was accounted for at the residual level.

Hypothesis 7 examined whether pretreatment levels of ER measures moderated change in symptoms during treatment. This was achieved by entering pretreatment levels of both ER measures (group-mean centered) as level 2 predictors of the slope of symptom change during treatment. Finally, to examine Hypothesis 8, we compared scores on ER measured before and after treatment to a matched normal sample using t-tests.

For all LMLMs, effect sizes were calculated as semi-partial r (r_s; Jaeger, Edwards, Das, & Sen, 2016; Nakagawa & Schielzeth, 2013) using

package 'r2glmm' in R (Jaeger, 2016). These effect sizes represent the unique contribution of the predictor above and beyond the contribution of all other predictors in the model. For *t*-tests, effect sizes were calculated as Hedges's *g*. The 29 participants were treated in a total of 329 sessions. Out of these sessions, there were a total of 6 missing datapoints for PDSS, 3 for ASI, 4 for reappraisal, and 4 for suppression. In addition, 4 patients dropped out of treatment prematurely. As a result, we were missing a total of 11 sessions for the 4 patients who dropped out. Given this level of missingness (~ 1% of the data missing at random and another 3–4% of those who dropped out and therefore were missing all measures), we did not impute missing values, but rather relied on the robustness of REML models in handling missing data (Shin, Davison, & Long, 2017).

Results

CHANGES IN PD/A SYMPTOMS MEASURES DURING TREATMENT

First, changes in PD/A severity and biased cognitions during the course of treatment were examined by exploring linear, quadratic, and log-linear changes. Both measures significantly decreased linearly during treatment, as expected (PDSS: b = -0.57, t_{293} = -6.27, p < .001, r_s = .36[.27, .45]; ASI: b = -2.05, t_{296} = -6.74, p < .001, r_s = .46[.38, .53]). No quadratic model was found significant (all p's > .570) and no log-linear model was found to better fit the data (Δ AIC and Δ BIC > 8 for both model comparisons). Therefore, we chose the linear model as the best-fitting model.

CHANGES IN REAPPRAISAL DURING TREATMENT (HYPOTHESIS 1)

Figure 2 presents the mean levels of reappraisal and suppression during treatment. Reappraisal did not display a linear significant increase throughout treatment (b = 0.04, t_{295} = 1.57, p = .117, r_s = .11 [.01, .21]), nor a log-linear significant increase (b = 0.31, t_{295} = 1.36, p = .175, r_s = .09[.01, .19]). However, reappraisal did display a significant quadratic increase (b = 0.01, t_{294} = 2.01, p = 0.045, r_s = .07[.00, .17]), corresponding to the increase visible in the late sessions in Figure 2. Therefore, the quadratic model was chosen as the best fitting model.

In the chosen quadratic model, the linear slope at the beginning of treatment was not significant (b = -0.04, t_{295} = -1.10, p = .272, r_s = .03[.00, .14]). This indicates that there was no significant increase in reappraisal at the beginning of treatment but that the rate of change per session increased as treatment progressed, eventually becoming significant. To determine the inflection point of the quadratic pattern we generated separate estimates for each session, allowing for an examination of the linear

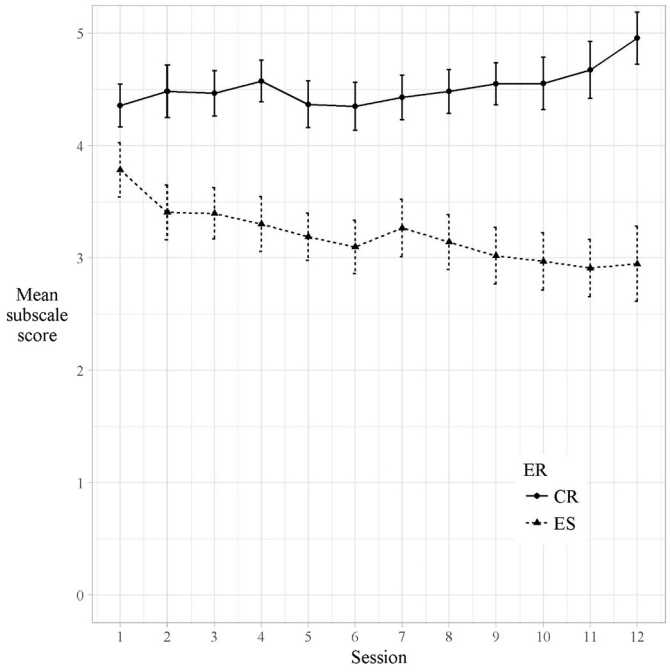

FIGURE 2 Mean Scores for the Emotion Regulation Questionnaire (ERQ) Subscales Measured Before Each Session: Cognitive Reappraisal (CR) and Expressive Suppression (ES)

change attributed to that session. We found that by Session 7, the changes became positive and significant (b = 0.05, t_{294} = 1.99, p = .048) and remained significant until the end of treatment. These findings are consistent with the shape of the growth curve visible in Figure 2.

CHANGES IN REAPPRAISAL DURING THE COGNITIVE RESTRUCTURING MODULE (HYPOTHESIS 2)

For all patients except one, only a single session was devoted primarily to reappraisal of panic-related cognitions as part of the cognitive restructuring module. This session was either the second, third, or fourth session (for 9, 17, and 2 patients, respectively). For one patient this session spanned over two sessions (second and third). Contrary to Hypothesis 2, there were no significant changes in reappraisal from the start of the cognitive restructuring module to the start of the session after it, during which the patients were requested to practice reappraisal at home (mean difference = -0.05, t_{27} = -0.33, p = .744, g = 0.04 [-0.48, 0.56]).

CHANGES IN SUPPRESSION DURING TREATMENT (HYPOTHESIS 3)

In accordance with Hypothesis 3, suppression displayed a significant linear decrease (b = -0.07, t_{295} = -3.13, p = .002, r_s = .19[.09, .29]), whereas the quadratic slope was not significant (b < 0.01, t_{294} = 1.07, p = .285, r_s = .03[.00, .14]). Contrary to our predictions, the log-linear model for suppression) b = -0.74, t_{295} = -3.03, p = .003, r_s = .18[.08, .28]) performed much better than the linear model (Δ AIC and Δ BIC = −25.8). Therefore, the log-linear model was chosen as the best-fitting model, indicating that suppression decreased to a greater extent early in treatment compared to later in treatment. This pattern is also observable in Figure 2.

CROSS-LAGGED ANALYSES: EXAMINING THE BI-DIRECTIONAL RELATIONSHIP BETWEEN ER AND PD/A SYMPTOMS (HYPOTHESES 4–6)

As noted in the methods section, all cross-lagged analyses use symptom and ER levels over time as indications of change in each variable. Table 2 presents estimates of fixed-effects from cross-lagged analyses of ER and symptom measures. The full results of all cross-lagged models are included in supplemental material, section 4 (Tables S2-S4). Contrary to Hypothesis 4, levels of reappraisal were not significantly associated with subsequent levels of cognition (reappraisal ➔ ASI; b = 0.42, t_{264} = 0.59, p = .556, r_s = .02[.00, .13]). The opposite direction (ASI ➔ reappraisal) was also not significant (see Table 2). In accordance with Hypothesis 5, levels of ASI were associated with subsequent levels of suppression (ASI ➔ suppression; b = 0.01, t_{264} = 2.20, p = .029, r_s = .10 [.01, .21]). However, a significant association was also found in the opposite direction: levels of suppression predicted subsequent levels of ASI (suppression ➔ ASI; b = 1.77, t_{264} = 2.14, p = .033, r_s = .06[.00, .17]), suggesting a reciprocal relationship between these two constructs. Finally, in accordance with Hypothesis 6, levels of suppression were significantly associated with subsequent levels of PDSS (suppression ➔ PDSS; b = 1.54, t_{261} = 2.97, p = .003, r_s = .16[.05, .27]). Levels of symptoms did not predict subsequent levels of suppression (PDSS ➔ suppression; b = 0.00, t_{262} = -0.17, p = .865, r_s = .00[.00, .13]) suggesting a temporal precedence of the suppression in relation to PDSS (Table 2).

Table 2
Mean Regression Coefficients (Fixed Effects) and 95% Confidence Interval for Cross-Lagged Models for Symptoms Severity, Biased Cognitions and Emotion Regulation During Treatment

Outcome	ER	ER predicting outcome	Outcome predicting ER
PDSS	CR	.40 [-.42, 1.22]	-.02 [-.05, .02]
ASI		.42 [-.98, 1.82]	-.01 [-.03, .00]
PDSS	ES	1.54 [.52, 2.56]**	.00 [-.02, .02]
ASI		1.77 [.14, 3.41]*	.01 [.00, .03]*

Note. PDSS = panic disorder severity scale; ASI = anxiety sensitivity index; ER = emotion regulation; CR = cognitive reappraisal; ES = expressive suppression.
* / ** significantly different from zero at the p < .05 / .01 level.

CROSS-LAGGED ANALYSES: EXAMINING THE BI-DIRECTIONAL RELATIONSHIP BETWEEN ER AND PD/A SYMPTOMS IN DIFFERENT PHASES OF THERAPY

As reported above, both reappraisal and suppression changed nonlinearly during treatment (reappraisal changed in a quadratic fashion, whereas suppression changed in a log-linear fashion). Therefore, we further examined (post-hoc) the bi-directional relationship between ER and PD/A symptoms separately in two different phases of therapy: until (and including) the initial interoceptive exposure session (1st phase) and after the interoceptive exposure session (2nd phase). Clinical considerations guided our decision: at the end of the interoceptive exposure session, both core cognitive and behavioral skills have been introduced and practiced in therapy, allowing for a transition to the second phase, where the patient is required to practice and incorporate these skills in the context of in-vivo exposures and real-life. Phases were determined for each patient individually in similar fashion described above regarding the coding of the

cognitive restructuring module. On average the first phase was 4.65 sessions (*SD* = 0.90) and the second phase was 6.93 sessions (*SD* = 1.33).

As seen in Table 3, significant associations between ER and symptoms measures were apparent only in the second phase of treatment. Levels of PDSS significantly predicted levels of reappraisal (PDSS ➔ reappraisal; b = -0.05, t_{158} = -2.27, p = .025, r_s = .13[.01, .27]), but not in the opposite direction (reappraisal ➔ PDSS; b = 0.06, t_{158} = 0.11, p = .913, r_s = .01[.00, .16]), establishing temporal precedence of PDSS in relation to reappraisal in the later sessions of therapy. Regarding the association between PD/A symptoms and suppression, suppression temporally preceded levels of PDSS (suppression ➔ PDSS: b = 2.00, t_{158} = 2.93, p = .004, r_s = .16[.03, .30], but not in the opposite direction: b = -0.02, t_{158} = -1.64, p = .103, r_s = .04[.00, .19]), and also temporally preceded levels of ASI (suppression ➔ ASI: b = 2.82, t_{159} = 2.03, p = .044, r_s = .07[.00, .21] but not in the opposite direction: b = 0, t_{159} = 0.91, p = .364, r_s = .03[.00, .17]). The findings for suppression and the ASI were unidirectional, whereas in the whole treatment, results suggested bi-directionality.

PRETREATMENT ER PREDICTING CHANGES IN PD/A SYMPTOMS DURING TREATMENT (HYPOTHESIS 7)

Pretreatment reappraisal scores did not significantly predict changes in PD/A severity or biased cognitions (predicting PDSS: b = -0.07, t_{292} = -0.66, p = .510, r_s = .04[.00, .15]; predicting ASI: b = -0.50, t_{295} = -1.53, p = .130, r_s = .10[.01, .20]). Similar results were found regarding pretreatment suppression scores, which did not significantly predict changes in either PD/A severity or biased cognitions (predicting PDSS: b = -0.03, t_{292} = -0.43, p = .670, r_s = .02[.00, .13]; predicting ASI: b = 0.01, t_{295} = 0.02, p = .982, r_s = .00[.00, .12]).

COMPARING PD/A PATIENTS' PRE- AND POSTTREATMENT ER SCORES TO MATCHED NP SAMPLE (HYPOTHESIS 8)

The average reappraisal pretreatment score in our patient sample was 4.46 (*SD* = 1.01), similar to NP who scored on average 4.37 (*SD* = 1.13; t_{49} = 0.28, p = .781, g = 0.08 [-0.46, 0.61]). At posttreatment patients scored higher than NP, though differences were not significant (*M* = 4.72, *SD* = 1.08, t_{49} = 1.13, p = .264, g = 0.31 [-0.23, 0.85]). The average pretreatment suppression score in our patient sample was 3.83 (*SD* = 1.29), insignificantly higher than NP (t_{50} = 1.18, p = .244; g = 0.32 [-0.21, 0.86]) who scored on average 3.40 (*SD* = 1.28). At posttreatment patients' average score (*M* = 2.92, *SD* = 1.36) decreased significantly compared to pretreatment scores (t_{25} = 3.71, p = .001; g = 0.69 [0.14, 1.24]), and were lower compared to NP, though not significantly (t_{33} = -1.31, p = .199, g = 0.36 [-0.18, 0.90]).

POST HOC POWER ANALYSES

Given the sample size in the current study, it is important to conduct power analyses of the effects of interests. Post hoc power analyses for primary multilevel models were conducted using Monte Carlo simulations in Mplus 8.1, as outlined by Bolger and Laurenceau (2013) and Lane and Hennes (2018). An elaborated description of the procedure and findings for these analyses is included in supplemental material, section 5. As the results show, across model types, power was, for the most part, excellent for small-medium effects or larger, but suboptimal for small effects. One exception was power for models for crossed-lagged prediction of ASI from reappraisal or suppression, which was adequate only for large effects.

Discussion

The goal of this study was to examine the role of reappraisal and suppression as predictors of change in

Table 3
Mean Regression Coefficients (Fixed Effects) and 95% Confidence Interval for Cross-Lagged Models for Symptoms Severity, Biased Cognitions and Emotion Regulation During Two Treatment Phases Separately

		1st treatment phase (until first *in vivo* exposure)		2nd treatment phase (from first *in vivo* exposure)	
Outcome	ER	ER predicting outcome	Outcome predicting ER	ER predicting outcome	Outcome predicting ER
PDSS	CR	.80 [-.30, 1.91]	.02 [-.02, .06]	.06 [-1.06, 1.18]	-.05 [-.09, -.01]*
ASI		.55 [-1.11, 2.21]	-.01 [-.03, .02]	.85 [-1.15, 2.85]	-.01 [-.03, .00]
PDSS	ES	.34 [-1.19, 1.88]	.02 [-.02, .05]	2.00 [.65, 3.34]**	-.02 [-.04, .00]
ASI		2.02 [-.80, 4.85]	.02 [-.01, .04]	2.82 [.07, 5.56]*	.00 [-.01, .02]

Note. PDSS = panic disorder severity scale; ASI = anxiety sensitivity index; ER = emotion regulation; CR = cognitive reappraisal; ES = expressive suppression.
* / ** significantly different from zero at the p < .05 / .01 level.

CBT for PD/A. A temporal model was suggested to describe the unfolding of the use of these two emotion regulation strategies and symptom change over the course of CBT. The model (Figure 1, panel A) predicted that increases in reappraisal would precede decreases in biased cognitions (i.e., anxiety sensitivity), which, in turn, would precede decreases in suppression, which would be followed by symptom reduction. A test of some of the propositions of the model provided partial support: we found that reappraisal was generally not associated with treatment outcome. However, suppression exhibited a reciprocal relationship with biased cognitions. Symptom reduction followed changes in suppression as hypothesized (see Figure 1, panel B, for the revised model).

THE ROLE OF REAPPRAISAL

As expected, reappraisal increased significantly in treatment, though unexpectedly, this increase occurred in late and not early stages of therapy (from Session 7 onwards), exhibiting a quadratic change over time. To our surprise, reappraisal did not change immediately after the cognitive restructuring module. This appears contrary to the finding by Kivity and Huppert (2016), that training in reappraisal in social anxiety increased use of reappraisal 1 week later. Contrary to the proposed model, changes in biased cognitions did not appear to be preceded by changes in reappraisal. This is not surprising considering the lack of change in reappraisal until later stages of treatment and the linear changes in biased cognitions. Pretreatment measures of reappraisal also did not significantly moderate changes in PD/A symptoms during treatment. In addition, comparing our patient sample to matched subjects from a normal population revealed no significant differences between groups either before or after treatment. Finally, post-hoc analysis of two different stages of treatment found that reappraisal changes occurred after symptom reduction in the later phase of therapy. Although caution is needed in terms of interpreting null results in modest sample sizes, taken together these findings are inconsistent with our predictions.

These findings are consistent with previous findings which failed to find significant differences in habitual and lab-based use of reappraisal between PD/As and HCs (e.g., Breuninger et al., 2017; Manber Ball et al., 2013; Reinecke et al., 2015). Our findings regarding reappraisal are quite surprising given that reevaluating negative cognitions related to bodily sensations is an important concept in CBT for PD/A (Clark, 1986), and reappraisal is thought to be a key aspect of CBT in general (e.g., Campbell-Sills & Barlow, 2007).

It should be noted that whereas the frequency of the use of reappraisal was not a significant predictor of symptom change, biased cognitions decreased linearly throughout treatment. Given the findings that show that reductions in threat appraisals predict subsequent symptom reduction in CBT for anxiety (Gallagher et al., 2013; Smits et al., 2012), there are a number of potential explanations for the findings of the current study. First, the discrepancy between the change in biased cognition compared to lack of change in reappraisal stresses the important distinction between *appraisal* and *reappraisal*. In most models of emotion, appraisals influence the initial emotional experience whereas reappraisal is an attempt to modify the experience (Gross, 2015). Changes in cognitions (appraising sensations or situations in a nonanxious manner) may occur without deliberately engaging in reappraisal. For example, psychoeducation may correct erroneous thinking without the patient needing to subsequently, explicitly utilize reappraisal. Alternatively, patients might have a very specific deficit in reappraisal in relation to panic cognitions, which does not generalize to their overall style of emotion regulation. Thus, the ERQ may not be sensitive to this specific deficit. Further studies are needed to examine specific panic-related reappraisal, potentially by modifying the ERQ to specific panic-related cognitions (cf. Kivity & Huppert, 2016, for a similar modification for social anxiety). A third explanation is related to the multifaceted nature of ER. There is evidence (Goldin et al., 2014; Kivity & Huppert, 2018) that the efficacy of using reappraisal is more strongly associated with symptom severity or outcomes compared to frequency, but only the latter was measured in this study. Patients might be learning to utilize reappraisal more efficiently rather than to use it more frequently.

Thus, future studies should measure efficacy as well as frequency of reappraisal. Despite these possibilities, the findings question whether reappraisal is an important emotion regulation strategy that is necessary to promote effective treatment of PD/A. Models incorporating reappraisal as an important factor leading to cognitive change may need to be revised to focus more on appraisals or interpretations (cf. Clark, 1986) or self-efficacy (instead of frequency) of reappraisal (cf. Gallagher et al., 2013), if these findings are replicated and extended.

THE ROLE OF SUPPRESSION

As expected, suppression decreased significantly during treatment, displaying a greater decrease in the initial stages of therapy, thereby exhibiting a log-linear change over time. In partial support of the hypothesized model, changes in biased cognitions preceded

changes in suppression, though changes in suppression predicted subsequent changes in biased cognitions as well, indicating a reciprocal relation between the two variables. These findings suggest that as patients suppress their emotions less frequently, their panic cognitions tend to be less catastrophic, and vice versa. Different findings emerged when examining two different phases of treatment post-hoc. Decreases in suppression were found to precede changes in biased cognitions significantly only in the latter phase of treatment, though effects were of similar size in the early phase of treatment as well. These findings were counter to our hypothesis, which predicted that changes in cognitions would precede changes in suppression. Our findings that across all of treatment, there was a bi-directional relationship between cognitions and suppression whereas in early and late phases examined separately, there was an apparent unidirectional relationship requires further study in larger samples.

In contrast, as predicted, changes in suppression preceded changes in symptoms. These findings suggest that decreasing one's tendency to inhibit the expression of emotions is an important skill learned via CBT. How should we understand the association between suppression and panic symptoms? First, as noted, many PD/A patients are concerned that others might notice their symptoms of anxiety and therefore unsuccessfully try to hide them, often resulting in further exacerbation of those symptoms. Learning not to hide symptoms of anxiety is therefore an important step in decreasing the fear of fear, and eventually is followed by a decrease in symptom severity. Second, whereas what was measured in our study was expressive suppression, this form of suppression may be considered a proxy to other forms of suppression, which theoretically may be related to PD/A too, such as a general tendency to suppress the *experience* of anxiety, which is an important part of the CBT model for PD/A. Future studies might explore the role of other forms of suppression as well.

Suppression changed more rapidly at the beginning of treatment, compared to later stages. We did not have a specific hypothesis for this finding. If replicated, future studies can examine if this process is associated with starting treatment per se, or to techniques that are typically introduced in the initial sessions such as psychoeducation and cognitive work.

Whereas changes in suppression preceded symptom change, pretreatment measures of suppression did not moderate changes in PD/A symptoms during treatment. In addition, comparing our patient sample to matched subjects from the normal population revealed no significant differences before or after treatment, even though suppression decreased significantly via treatment. The fact that the effects were found *within* patients rather than *between* patients stresses the importance of measuring ER repeatedly throughout treatment, allowing for disaggregation of within- and between-patient effects. Our findings indicate that the importance of suppression comes from its association with changes in treatment measures over time, rather than from differences between patients and controls. In this manner, our findings do not contradict null findings regarding differences between HCs and individuals with PD/A (Breuninger et al., 2017), but instead suggest a refocus on within-patients processes.

LIMITATIONS AND CONCLUSION

Several limitations of the study are important to note. Despite the multiple assessment points for each patient (allowing for detection of within-patient effects), our sample was moderate in size for detecting between-patient effects and comparisons with the matched sample, thus limiting the interpretation of null results. In addition, power analyses showed that our power was mostly adequate for small to medium effects but not for small effects. Thus, it is possible that some of the nonsignificant findings would have been significant with a larger sample size. It is important to replicate the findings in a larger sample, ideally along with Bayesian estimates in order to be more confident of the significant and nonsignificant findings.

The use of multilevel modeling has some strengths and limitations. Whereas Restricted Maximum Likelihood (REML) handles missing data adequately even in small samples (Shin et al., 2017), endogeneity is only partly handled via the AR1 covariance structure. Structural equation models (SEM), on the other hand, are more flexible, allow for testing the proposed model all at once and can overcome the endogeneity problem (Falkenström et al., 2017), but require a large number of participants, and thus were not feasible in the current sample. Additionally, multiple models as were fitted in this analysis are at risk of Type 1 error inflation. We have reported effect size and confidence intervals, and, most important, tested hypotheses derived based on *a priori* theory. Although we believe that such theory-driven approaches help guard against capitalization on chance, this risk was not controlled for in our analysis. Thus, it is important to replicate and extend these findings on larger samples, employing other forms of analysis.

Other noteworthy limitations include the absence of interrater reliabilities for diagnosis and the use of self-report measures. In addition, whereas phases were coded on an individual basis, their timing and length were not completely identical across patients, thereby

introducing additional noise to analyses pertaining to specific phases. Last, our matched sample was taken from an undiagnosed population. Comparing patients to healthy controls free of diagnoses would have been preferable. Therefore, our report should be considered as preliminary.

In conclusion, this is the first study we are aware of exploring emotion regulation strategies in CBT for PD/A. Our findings only partially support the hypothesized model of emotion regulation and instead suggested a limited role of reappraisal as it was measured in our study. In addition, our results suggest that suppression tends to precede changes in symptoms and appraisals, emphasizing the role of expressive suppression in PD/A treatment. Whereas our study questions the active role of reappraisal as an emotion regulation technique acquired via treatment, it stresses the importance of evaluating other means in which cognitions change during treatment. In addition, our study highlights the role of expressive suppression as promoting symptom change during therapy, suggesting its importance when incorporating emotion regulation in models of treatment of PD/A.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.beth.2018.10.005.

References

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders (DSM-5)*. Washington, DC: Author.

Arntz, A. (2002). Cognitive therapy versus interoceptive exposure as treatment of panic disorder without agoraphobia. *Behaviour Research and Therapy*, *40*(3), 325–341. https://doi.org/10.1016/S0005-7967(01)00014-6

Baker, R., Holloway, J., Thomas, P. W., Thomas, S., & Owens, M. (2004). Emotional processing and panic. *Behaviour Research and Therapy*, *42*(11), 1271–1287. https://doi.org/10.1016/j.brat.2003.09.002

Barlow, D. H., Allen, L. B., & Choate, M. L. (2016). Toward a unified treatment for emotional disorders–republished article. *Behavior Therapy*, *47*(6), 838–853. https://doi.org/10.1016/j.beth.2016.11.005

Bolger, N., & Laurenceau, J. P. (2013). *Intensive longitudinal methods: An introduction to diary and experience sampling research*. New York, NY: Guilford.

Bouchard, S., Gauthier, J., Laberge, B., French, D., Pelletier, M. H., & Godbout, C. (1996). Exposure versus cognitive restructuring in the treatment of panic disorder with agoraphobia. *Behaviour Research and Therapy*, *34*(3), 213–224. https://doi.org/10.1016/0005-7967(95)00077-1

Breuninger, C., Sláma, D. M., Krämer, M., Schmitz, J., & Tuschen-Caffier, B. (2017). Psychophysiological reactivity, interoception and emotion regulation in patients with agoraphobia during virtual reality anxiety induction. *Cognitive Therapy and Research*, *41*(2), 193–205. https://doi.org/10.1007/s10608-016-9814-9

Campbell-Sills, L., & Barlow, D. H. (2007). Incorporating emotion regulation into conceptualizations and treatments of anxiety and mood disorders. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 542–559). New York: Guilford Press.

Campbell-Sills, L., Barlow, D. H., Brown, T. A., & Hofmann, S. G. (2006). Acceptability and suppression of negative emotion in anxiety and mood disorders. *Emotion*, *6*(4), 587–595. https://doi.org/10.1037/1528-3542.6.4.587

Carpenter, J. K., Andrews, L. A., Witcraft, S. M., Powers, M. B., Smits, J. A., & Hofmann, S. G. (2018). Cognitive behavioral therapy for anxiety and related disorders: A meta-analysis of randomized placebo-controlled trials. *Depression and Anxiety*, *35*(6), 502–514. https://doi.org/10.1002/da.22728

Clark, D. M. (1986). A cognitive approach to panic. *Behaviour Research and Therapy*, *24*(4), 461–470. https://doi.org/10.1016/0005-7967(86)90011-2

Craske, M. G., & Barlow, D. H. (2007). *Mastery of your anxiety and panic: Therapist guide*. New York: Oxford University Press.

Falkenström, F., Finkel, S., Sandell, R., Rubel, J. A., & Holmqvist, R. (2017). Dynamic models of individual change in psychotherapy process research. *Journal of Consulting and Clinical Psychology*, *85*(6), 537–549. https://doi.org/10.1037/ccp0000203

Gallagher, M. W., Payne, L. A., White, K. S., Shear, K. M., Woods, S. W., Gorman, J. M., & Barlow, D. H. (2013). Mechanisms of change in cognitive behavioral therapy for panic disorder: the unique effects of self-efficacy and anxiety sensitivity. *Behaviour Research and Therapy*, *51*(11), 767–777. https://doi.org/10.1016/j.brat.2013.09.001

Goldin, P. R., Lee, I., Ziv, M., Jazaieri, H., Heimberg, R. G., & Gross, J. J. (2014). Trajectories of change in emotion regulation and social anxiety during cognitive-behavioral therapy for social anxiety disorder. *Behaviour Research and Therapy*, *56*, 7–15. https://doi.org/10.1016/j.brat.2014.02.005

Gross, J. J. (2015). Emotion regulation: Current status and future prospects. *Psychological Inquiry*, *26*(1), 1–26. https://doi.org/10.1080/1047840X.2014.940781

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology*, *85*(2), 348–362. https://doi.org/10.1037/0022-3514.85.2.348

Hofmann, S. G., Sawyer, A. T., Fang, A., & Asnaani, A. (2012). Emotion dysregulation model of mood and anxiety disorders. *Depression and Anxiety*, *29*(5), 409–416. https://doi.org/10.1002/da.21888

Houck, P. R., Spiegel, D. A., Shear, M. K., & Rucci, P. (2002). Reliability of the self-report version of the panic disorder severity scale. *Depression and Anxiety*, *15*(4), 183–185. https://doi.org/10.1002/da.10049

Huppert, J. D., & Baker-Morissette, S. L. (2004). Beyond the manual: The insider's guide to panic control treatment. *Cognitive and Behavioral Practice*, *10*(1), 2–13. https://doi.org/10.1016/S1077-7229(03)80003-2

Jaeger, B. (2016). r2glmm: Computes R squared for mixed (multilevel) models (LMMs and GLMMs) [Software]. https://cran.r-project.org/web/packages/r2glmm/.

Jaeger, B. C., Edwards, L. J., Das, K., & Sen, P. K. (2016). An R^2 statistic for fixed effects in the generalized linear mixed model. *Journal of Applied Statistics*, *44*(6), 1086–1105. https://doi.org/10.1080/02664763.2016.1193725

Jazaieri, H., Morrison, A. S., Goldin, P. R., & Gross, J. J. (2015). The role of emotion and emotion regulation in social

anxiety disorder. *Current Psychiatry Reports*, *17*(1), 531. https://doi.org/10.1007/s11920-014-0531-3

Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-Month DSM-IV Disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 617–627. https://doi.org/10.1001/archpsyc.62.6.617

Kivity, Y., & Huppert, J. D. (2016). Does cognitive reappraisal reduce anxiety? A daily diary study of a micro-intervention with individuals with high social anxiety. *Journal of Consulting and Clinical Psychology*, *84*(3), 269–283. https://doi.org/10.1037/ccp0000075

Kivity, Y., & Huppert, J. D. (2018). Emotion regulation in social anxiety: A systematic investigation using self-report, behavioral, and event-related potentials measures.*Cognition & Emotion*. https://doi.org/10.1080/02699931.2018.1446414 Advance online publication

Kivity, Y., Tamir, M., & Huppert, J. D. (2016). Self-acceptance of negative emotions: the positive relationship with effective cognitive reappraisal. *International Journal of Cognitive Therapy*, *9*(4), 279–294. https://doi.org/10.1521/ijct_2016_09_10

Kroenke, K., Spitzer, R. L., Williams, J. B. W., Monahan, P. O., & Löwe, B. (2007). Anxiety disorders in primary care: Prevalence, impairment, comorbidity, and detection. *Annals of Internal Medicine*, *146*(5), 317–325. https://doi.org/10.7326/0003-4819-146-5-200703060-00004

Lane, S. P., & Hennes, E. P. (2018). Power struggles: Estimating sample size for multilevel relationships research. *Journal of Social and Personal Relationships*, *35*(1), 7–31. https://doi.org/10.1177/0265407517710342

Lorenzo-Luaces, L., Keefe, J. R., & DeRubeis, R. J. (2016). Cognitive-behavioral therapy: Nature and relation to non-cognitive behavioral therapy. *Behavior Therapy*, *47*(6), 785–803. https://doi.org/10.1016/j.beth.2016.02.012

Manber Ball, T., Ramsawh, H. J., Campbell-Sills, L., Paulus, M. P., & Stein, M. B. (2013). Prefrontal dysfunction during emotion regulation in generalized anxiety and panic disorders. *Psychological Medicine*, *43*(07), 1475–1486. https://doi.org/10.1017/S0033291712002383

McNally, R. J. (2002). Anxiety sensitivity and panic disorder. *Biological Psychiatry*, *52*(10), 938–946. https://doi.org/10.1016/S0006-3223(02)01475-0

Moscovitch, D. A., Gavric, D. L., Senn, J. M., Santesso, D. L., Miskovic, V., Schmidt, L. A., & Antony, M. M. (2012). Changes in judgment biases and use of emotion regulation strategies during cognitive-behavioral therapy for social anxiety disorder: Distinguishing treatment responders from nonresponders. *Cognitive Therapy and Research*, *36*(4), 261–271. https://doi.org/10.1007/s10608-011-9371-1

Nakagawa, S., & Schielzeth, H. (2013). A general and simple method for obtaining R^2 from generalized linear mixed-effects models. *Methods in Ecology and Evolution*, *4*(2), 133–142. https://doi.org/10.1111/j.2041-210x.2012.00261.x

Pinheiro, J. C., Bates, D. M., DebRoy, S., & Sarkar, D. (2016). nlme: Linear and Nonlinear Mixed Effects Models [Software]. https://cran.r-project.org/web/packages/nlme/.

Reinecke, A., Filippini, N., Berna, C., Western, D. G., Hanson, B., Cooper, M. J., & Harmer, C. J. (2015). Effective emotion regulation strategies improve fMRI and ECG markers of psychopathology in panic disorder: implications for psychological treatment action. *Translational Psychiatry*, *5*(11), e673. https://doi.org/10.1038/tp.2015.160

Salkovskis, P. M., Clark, D. M., Hackmann, A., Wells, A., & Gelder, M. G. (1999). An experimental investigation of the role of safety-seeking behaviours in the maintenance of panic disorder with agoraphobia. *Behaviour Research and Therapy*, *37*(6), 559–574. https://doi.org/10.1016/S0005-7967(98)00153-3

Shear, M. K., Rucci, P., Williams, J., Frank, E., Grochocinski, V., Vander Bilt, J., & Wang, T. (2001). Reliability and validity of the Panic Disorder Severity Scale: Replication and extension. *Journal of Psychiatric Research*, *35*(5), 293–296. https://doi.org/10.1016/S0022-3956(01)00028-0

Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., Weiller, E., & Dunbar, G. C. (1998). The Mini-International Neuropsychiatric Interview (M.I.N.I): The development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *Journal of Clinical Psychiatry*, *59*(Suppl 20), 22–33.

Sheppes, G., Suri, G., & Gross, J. J. (2015). Emotion regulation and psychopathology. *Annual Review of Clinical Psychology*, *11*(1), 379–405. https://doi.org/10.1146/annurev-clinpsy-032814-112739

Shin, T., Davison, M. L., & Long, J. D. (2017). Maximum likelihood versus multiple imputation for missing data in small longitudinal samples with nonnormality. *Psychological Methods*, *22*(3), 426–449. https://doi.org/10.1037/met0000094

Sloan, T., & Telch, M. J. (2002). The effects of safety-seeking behavior and guided threat reappraisal on fear reduction during exposure: An experimental investigation. *Behaviour Research and Therapy*, *40*(3), 235–251. https://doi.org/10.1016/S0005-7967(01)00007-9

Smits, J. A., Julian, K., Rosenfield, D., & Powers, M. B. (2012). Threat reappraisal as a mediator of symptom change in cognitive-behavioral treatment of anxiety disorders: A systematic review. *Journal of Consulting and Clinical Psychology*, *80*(4), 624. https://doi.org/10.1037/a0028957

Stewart, R. E., & Chambless, D. L. (2009). Cognitive–behavioral therapy for adult anxiety disorders in clinical practice: A meta-analysis of effectiveness studies. *Journal of Consulting and Clinical Psychology*, *77*(4), 595–606. https://doi.org/10.1037/a0016032

Taylor, S., Zvolensky, M. J., Cox, B. J., Deacon, B., Heimberg, R. G., Ledley, D. R., & Stewart, S. H. (2007). Robust dimensions of anxiety sensitivity: Development and initial validation of the Anxiety Sensitivity Index-3.*Psychological Assessment*, *19*(2), 176. http://psycnet.apa.org/doi/10.1037/1040-3590.19.2.176

Teachman, B. A., Smith-Janik, S. B., & Saporito, J. (2007). Information processing biases and panic disorder: Relationships among cognitive and symptom measures. *Behaviour Research and Therapy*, *45*(8), 1791–1811. https://doi.org/10.1016/j.brat.2007.01.009

Wang, L.(. P.)., & Maxwell, S. E. (2015). On disaggregating between-person and within-person effects with longitudinal data using multilevel models. *Psychological Methods*, *20*(1), 63–83. https://doi.org/10.1016/j.genhosppsych.2016.01.003

Weiss, M., Kivity, Y., & Huppert, J. D. (2014). How does the therapeutic alliance develop throughout cognitive behavioral therapy for panic disorder? Sawtooth patterns, sudden gains, and stabilization. *Psychotherapy Research*, *24*(3), 407–418. https://doi.org/10.1080/10503307.2013.868947

Zalaznik, D., Weiss, M., & Huppert, J. D. (2017). Improvement in adult anxious and avoidant attachment during cognitive behavioral therapy for panic disorder. *Psychotherapy Research*, 1–17. https://doi.org/10.1080/10503307.2017.1365183

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