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Psychopathology and thought suppression: A quantitative review

Joshua C. Magee [a,b,*], K. Paige Harden [c], Bethany A. Teachman [d]

[a] Centers for Behavioral and Preventive Medicine, the Miriam Hospital, Providence, RI, USA
[b] The Alpert Medical School of Brown University, Providence, RI, USA
[c] University of Texas, Austin, TX, USA
[d] University of Virginia, Charlottesville, VA, USA

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ABSTRACT

Recent theories of psychopathology have suggested that thought suppression intensifies the persistence of intrusive thoughts, and proposed that difficulty with thought suppression may differ between groups with and without psychopathology. The current meta-analytic review evaluates empirical evidence for difficulty with thought suppression as a function of the presence and specific type of psychopathology. Based on theoretical proposals from the psychopathology literature, diagnosed and analogue samples were expected to show greater recurrence of intrusive thoughts during thought suppression attempts than non-clinical samples. However, results showed no overall differences in the recurrence of thoughts due to thought suppression between groups with and without psychopathology. There was, nevertheless, variation in the recurrence of thoughts across different forms of psychopathology, including relatively less recurrence during thought suppression for samples with symptoms of Obsessive-Compulsive Disorder, compared to non-clinical samples. However, these differences were typically small and provided only mixed support for existing theories. Implications for cognitive theories of intrusive thoughts are discussed, including proposed mechanisms underlying thought suppression.

Contents

* Corresponding author at: Centers for Behavioral and Preventive Medicine, The Miriam Hospital, Coro East, Suite 1B, 167 Point St., Providence, RI 02903, USA. Tel.: +1 401 793 8332; fax: +1 401 9793 8059.
E-mail address: jmagee2@lifespan.org (J.C. Magee).

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1. Introduction

Persistent intrusive thoughts and images are a key source of distress and dysfunction across many forms of psychopathology (Clark & Rhyno, 2001). Understandably, attempts to keep these distressing thoughts and images out of mind are nearly as ubiquitous as the thoughts themselves. However, a growing body of evidence suggests that attempts to keep intrusive thoughts out of mind, a process termed 'thought suppression,' can unintentionally heighten the recurrence of intrusive thoughts under certain circumstances (Abramowitz, Tolin, & Street, 2001; Wenzlaff & Wegner, 2000), and lead to various forms of distress. Perceived deficiencies in control over thoughts are a basic component of many mental disorders (American Psychiatric Association, 2000), so it is not surprising that psychopathology researchers have sought to integrate thought suppression into their disorder-specific theories, including theories of anxiety disorders (Ehlers & Clark, 2000; Salkovskis, 1996; Thorpe & Salkovskis, 1997), depression (Wegner, 1994; Wenzlaff & Luxton, 2003), and eating disorders (Polivy & Herman, 2002), among others. However, to date, evidence that thought suppression enhances the recurrence of intrusive thoughts in psychopathology has been mixed, and there is likely variability across disorders. The current meta-analysis seeks to quantify differences in difficulties with thought suppression (i.e., the extent to which intrusive thoughts recur during or after suppression attempts) to determine whether groups with psychopathology experience more difficulty with thought suppression than groups without psychopathology, and to examine variation in difficulty across different types of psychopathology. The meta-analysis focuses on the recurrence of intrusive thoughts during and following suppression because increased intrusive thoughts are predicted to lead to extreme distress in psychopathology (Purdon, 1999; Reynolds & Salkovskis, 1992). Further, the recurrence of intrusive thoughts is the most universally measured outcome of thought suppression attempts, although it can be assessed in different ways (e.g., thought frequency, thought duration, and retrospective estimates of thought recurrence).

While many ideas concerning thought suppression can be traced back to William James (see Clark, 2001), Daniel Wegner has spurred much of the recent interest in thought suppression research (Wegner, Schneider, Carter, & White, 1987). In their seminal work, Wegner et al. (1987) found that participants attempting to suppress thoughts of a white bear ironically later experienced *more* white bear thoughts than participants not instructed to suppress. The effects of thought suppression are usually divided into two components: the enhancement of intrusive thoughts during suppression, known as the "initial enhancement effect," and the enhancement occurring *after* suppression has ceased, termed the "rebound effect" (Wenzlaff & Wegner, 2000). Across studies of largely non-clinical samples, it seems that people are generally successful at suppressing initially (i.e., experiencing little amplification of thoughts), but show a small to medium rebound effect of increased intrusive thoughts after suppression (compared to control instructions; Abramowitz et al., 2001).

Models of thought suppression have generally converged on two independent, but not mutually exclusive, proposals concerning how the thought suppression process could function differently among persons with psychopathology (Magee & Zinbarg, 2007). One possibility is that thought suppression attempts may result in greater recurrence of thoughts for some samples with psychopathology because of differences in how much thought suppression is attempted, without there necessarily being differences in initial enhancement or rebound effects per thought suppression experience between samples with and without psychopathology. Along these lines, there is evidence that samples with psychopathology attempt to suppress their thoughts more frequently than non-clinical samples (Magee & Zinbarg, 2007).

A second possibility, and the focus of the current study, is that when an individual with psychopathology attempts to suppress a thought, the thought is more likely to return than for a person without psychopathology (Magee & Zinbarg, 2007). Reviews have come to conflicting conclusions about the level of empirical support for this idea: Purdon (1999) and Najmi and Wegner (2008) concluded that studies examining samples with depression and generalized anxiety disorder (GAD) do not seem to show amplification of unsuccessful thought suppression (i.e., greater recurrence of intrusive thoughts) compared to non-clinical samples, whereas there is mixed evidence in obsessive-compulsive disorder (OCD). Purdon (1999) suggested there is stronger evidence for amplification in post-traumatic stress disorder (PTSD), although in a later review Najmi and Wegner (2008) interpreted the evidence as being unclear. Rassin, Merckelbach, and Muris (2000) concluded that evidence for an amplification of thought suppression failures in psychopathology is generally weak, with the possible exception of substance abuse disorders. Additionally, a previous meta-analysis focusing mainly on thought suppression difficulties in non-clinical samples did not support the thought suppression amplification hypothesis, finding that thought suppression was equally problematic among samples with and without psychopathology. However, this meta-analysis only included five diagnosed and six analogue samples with psychopathology, and the authors called for future research involving a larger number of studies as well as disorder-specific comparisons (Abramowitz et al., 2001).

Taken together, it is not clear that thought suppression is more difficult for samples with (versus without) psychopathology, despite the predictions of many disorder-specific theories. Unfortunately, the data have been too limited to evaluate this question quantitatively in prior reviews. Since then, a substantial number of studies involving samples with psychopathology have examined thought suppression, allowing the present computation of general and specific comparisons between groups with and without psychopathology. Additionally, more studies have begun to employ stringent control instructions that do not artificially heighten suppression effects (Rassin, Muris, Jong, & de Bruin, 2005), resulting in less measurement error. It is now possible to evaluate more rigorously whether thought suppression difficulties are amplified among groups with psychopathology compared to non-clinical samples.

2. Proposed mechanisms underlying thought suppression difficulties

There is little direct empirical evidence to speak to the mechanisms underlying thought suppression attempts and failures, and

mechanisms have rarely been measured in the studies reviewed here. Notwithstanding, it is important to consider how psychopathology might lead to difficulty when trying to manage an intrusive thought, because these considerations can lead to competing hypotheses. Thus, we outline three plausible mechanisms in order to provide a theoretical framework to guide expectations about when to expect difficulties with suppressing thoughts across different types of psychopathology.

2.1. Available cognitive resources

The first mechanism, initially proposed by Wegner (1994), emphasizes available cognitive resources as a main determinant of thought suppression difficulty. In Wegner's model, thought suppression is believed to involve two processes: one conscious, effortful 'operating' process that involves attempts to stop thinking about intrusive thoughts, and a second, automatic 'monitoring' process that monitors for further thought instances (Wegner, 1994). Suppression is purported to be successful as long as the conscious process is active and possesses sufficient cognitive resources. Supporting this dual-process account, non-clinical individuals are generally successful during thought suppression (when the conscious process is presumably active), but experience a small to moderate rebound effect of intrusive thoughts after strategic suppression effort has ceased and the automatic process continues to unintentionally activate the target thought (Abramowitz et al., 2001). Additionally, it seems that individuals have more difficulty during thought suppression when encountering other simultaneous cognitive demands (for a review, see Wenzlaff & Wegner, 2000), or when possessing lower levels of fluid intelligence (Brewin & Beaton, 2002). Further, recent imaging work has revealed a neural correlate of thought suppression performance located in the anterior cingulate, a region linked to effortful processing of stimuli (Mitchell et al., 2007; Wyland, Kelley, Macrae, Gordon, & Heatherton, 2003).

According to this resources account, individuals should experience a greater initial enhancement effect when cognitive resources are limited. If depletion of cognitive resources occurs especially frequently for individuals struggling with psychopathology, this could result in increased recurrence of intrusive thoughts during thought suppression attempts. In favor of this account, elevated negative mood, which is common across various forms of psychopathology (Ormel, Rosmalen, & Farmer, 2004), has been suggested to deplete cognitive resources during thought suppression, particularly among anxious and depressed persons (Wenzlaff & Wegner, 2000). This would suggest that individuals with psychopathology may show an increased initial enhancement effect, because high levels of negative mood are active and depleting the cognitive resources needed during thought suppression. This prediction should be distinguished from work by Bless (2001) and Gasper and Clore (2002), which suggests that mild or moderate levels of negative mood may promote a detailed processing style that can be *helpful* during the conscious, effortful part of suppression. Indeed, there is evidence among non-clinical individuals that negative mood is associated with more successful suppression compared to positive mood (Wyland & Forgas, 2007). However, as these authors note, it is likely these findings do not extend to individuals with psychopathology, who tend to have more chronic, higher levels of negative mood.

Taken together, the connection between depleted cognitive resources in specific types of psychopathology and increased difficulties with thought suppression is provocative, but empirical support is limited. If the connection is accurate, it leads to the hypothesis that depleted resources (due to negative mood or other disorder-specific deficits that deplete resources) may be a general vulnerability characterizing most forms of psychopathology, and will lead to a greater initial enhancement effect on intrusive thoughts during suppression attempts than non-clinical samples. For the rebound thinking period, there is not as clear a link between depleted cognitive resources and recurrence of thoughts (Wegner, 1994).

2.2. Motivation to suppress intrusive thoughts

A second mechanism that may help account for possible psychopathology group differences in suppression success is the motivation to suppress unwanted thoughts. Theoretically, thought suppression effort can be initially successful, but will predict greater subsequent rebound (Wenzlaff & Wegner, 2000). Along these lines, there is some evidence that when participants are instructed to suppress thoughts over longer time periods, they reduce their suppression effort and are less successful (Abramowitz et al., 2001), suggesting a role for motivation in predicting suppression success. Importantly, it is likely that motivation to suppress differs across psychopathology groups based on variable conceptions of the acceptability of, and consequent resistance to, intrusive thoughts. For example, a man with OCD will likely be highly motivated to suppress ego-dystonic thoughts about harming his child, whereas a woman with depression might feel ambivalent about suppressing self-critical intrusive thoughts. This leads to the prediction that persons suffering from the types of psychopathology that are associated with a high motivation to suppress should experience less of an initial enhancement effect with intrusive thoughts (due to substantial effort spent suppressing), but possibly a greater subsequent rebound effect (due to the greater automatic activation resulting from prior suppression effort as well as sustained, less successful suppression efforts; Wenzlaff & Wegner, 2000).

OCD and PTSD are likely on the higher end of motivation to suppress intrusive thoughts, given these disorders are partially defined by their persistent, intense resistance to intrusive thoughts and images (American Psychiatric Association, 2000). On the lower end, depression and GAD stand out as possible prototypes. In depression, general motivation deficits are well established (American Psychiatric Association, 2000), and cognitive theories of depression such as Beck's (1967, 1987) can be interpreted to imply a low drive to combat or suppress self-critical intrusive thoughts. In other words, a depressed person who perceives a self-critical intrusive thought as being consistent with their (low) self-worth would be less likely to resist it with suppression efforts (Wenzlaff, 2001). Depressed people may instead be prone to elaborate upon their negative intrusive thoughts (Clark & Purdon, 2009) and put less effort into tasks, such as suppression, that can require intensive cognitive effort (Scheurich et al., 2007). In GAD, the influential avoidance theory of worry (Borkovec, Alcaine, & Behar, 2004) suggests that worry may already serve the purpose of avoiding more affectively-laden information. In so far as worries are utilized as an avoidance strategy, there may be less motivation to suppress the worries. Finally, motivation to suppress may be more idiosyncratic in other forms of psychopathology, such as eating disorders and social anxiety disorder. For example, a negative thought about one's appearance or a memory of a negative social interaction may elicit high motivation to suppress at one time but persistent rumination at another. Overall, due to motivation to suppress, it follows that persons with OCD and PTSD may be expected to show greater success during the initial enhancement thinking period, followed by greater difficulty during the rebound thinking period. Depression and GAD might be expected to show the opposite pattern: equivalent or greater difficulty than non-clinical groups during the initial enhancement thinking period, followed by equivalent or less difficulty during the rebound thinking period. This mechanism offers competing hypotheses to the available cognitive resources mechanism, described earlier, in that there is reason to expect disorder-specific variation, whereas the strongest evidence with cognitive resources was for general psychopathology-wide suppression difficulties during the initial enhancement thinking period.

2.3. Metacognition about unsuccessful thought suppression

The third mechanism is derived from cognitive models of OCD, which state that simply having intrusive thoughts is not necessarily a problem; instead, the way an individual interprets their intrusive thoughts is expected to determine their consequences (Rachman, 1997). These models are in line with empirical evidence suggesting that unsuccessful thought suppression may be a common experience (Abramowitz et al., 2001). Instead, the way an individual interprets unsuccessful thought suppression is thought to be crucial (Purdon, 2004; Purdon & Clark, 1999; Wenzlaff & Wegner, 2000). Supporting this notion, in previous research we found that maladaptive attributions about unsuccessful thought suppression predicted increased intrusive thoughts during and following suppression (Magee & Teachman, 2007), and these attributions mediated group differences between persons high versus low in OCD symptoms in the recurrence of intrusive thoughts during and following thought suppression. Additionally, in a non-clinical sample, Förster and Liberman (2001) found that manipulating the meaning of unsuccessful thought suppression led to elimination or amplification of the rebound effect.

These findings converge on the hypothesis that groups who tend to make maladaptive metacognitions about unsuccessful thought suppression may paradoxically have greater difficulty during and after thought suppression attempts. For example, OCD may be a prototypical example of a disorder in which unsuccessful thought suppression is often met with maladaptive metacognition that could exacerbate the recurrence of the intrusive thought (e.g., "This thought of my sister being in a car accident keeps returning when I try to get rid of it because I'm an evil person who really wants her to be in an accident!"). While OCD is often the focus of this literature, one can easily speculate about other possible disorder-specific metacognitions (e.g., depression: "I can't control this thought because I'm weak"). We expect that metacognition about thought suppression is likely to occur and cause difficulty with thought suppression attempts during both the initial enhancement and rebound thinking periods.

2.4. Overview

In the current study, we evaluate the existing evidence for differential effects of thought suppression attempts on recurrence of intrusive thoughts across many forms of psychopathology. We also examine the consistency of the evidence with the predictions of three theoretical mechanisms proposed in the thought suppression literature. According to the first mechanism, available cognitive resources, diagnosed and analogue samples are expected to experience greater initial enhancement effects during suppression attempts than non-clinical samples. According to the second mechanism, motivation to suppress, samples with symptoms of OCD and PTSD are expected to experience lessened initial enhancement effects followed by a greater rebound effects, whereas samples with symptoms of GAD and depression are expected to demonstrate the opposite pattern. According to the third mechanism, maladaptive metacognition about thought suppression, samples with symptoms of OCD are expected to experience greater initial enhancement and rebound effects compared to non-clinical samples. Overall, this study provides an empirical test to refine existing theories of thought suppression in psychopathology.

3. Method

3.1. Literature search and study selection

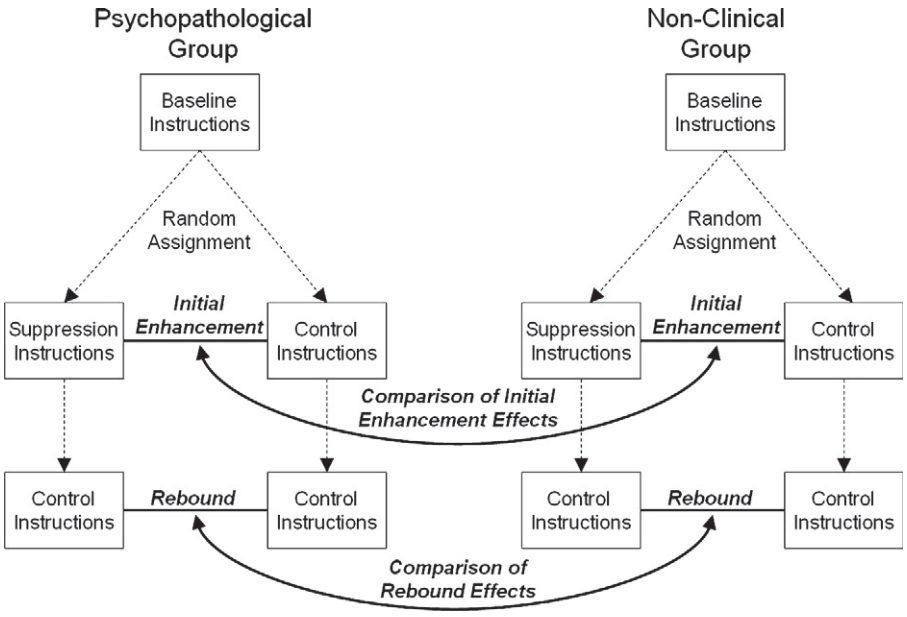
Studies on thought suppression and psychopathology available prior to April 2010 were identified using literature searches on PsycInfo, PubMed and Dissertation and Theses databases, reference lists from publications, and searches of individual journal issues for *Behaviour Research and Therapy*, *Cognitive Therapy and Research*, *Journal of Abnormal Psychology*, and *Journal of Anxiety Disorders*. All studies were required to be written in English and to contain sufficient information to permit the calculation of effect sizes for at least one measure of thought recurrence. We restricted the search to studies involving diagnosed or analogue samples corresponding to Axis I disorders, because it is not clear that the proposed theoretical differences in mechanisms underlying thought suppression extend to Axis II disorders. We also included studies using high trait anxiety samples; while this characteristic is not necessarily specific to any disorder, we believe it is informative about psychopathology, negative mood, and anxiety disorders in general. For any studies lacking sufficient data for the calculation of effect sizes, the data were requested from the study author(s).

For the primary analyses, we included two types of controlled studies in the final sample. The first type of controlled studies included at least one group with psychopathology, and one non-clinical group, both of which were randomly assigned to follow either thought suppression or control instructions. These studies possessed ideal controls for our primary research question, because both groups followed identical procedures, allowing us to better isolate difficulties due to thought suppression while holding methodological variations constant across the mental health groups being compared. Fig. 1 displays a visual guide for understanding the primary controlled comparisons. In the Figure, dashed lines indicate the flow that individual participants follow during a study, and the solid lines denote the between-subject comparisons that are used to draw inferences about the effects of thought suppression. The bolded straight lines for the "initial enhancement" and "rebound" effects of thought suppression refer to comparisons of suppression and control instructions *within* psychopathology and non-clinical groups. The bolded curved arrows reflect the main comparisons of initial enhancement and rebound effects *between* psychopathological and non-clinical groups. These latter comparisons offer the crucial tests for theories of thought suppression and psychopathology.

While this first type of studies offered ideal control, we also included a second type of controlled studies. This type included samples with psychopathology (but not non-clinical samples) randomly assigned to receive suppression or control instructions. We included these studies as a means of increasing the accuracy and reliability of the estimates of effect sizes for the various types of psychopathology (by increasing power). Further, adding these studies did not change the overall pattern of results, and made only small differences for psychopathology-specific comparisons.

For more descriptive, secondary analyses, we also examined studies that compared multiple samples receiving suppression instructions (e.g., a sample with OCD symptoms versus a non-clinical sample both following suppression instructions). These uncontrolled comparisons are less informative about the effects specifically due to thought suppression, but can estimate general differences in rates of intrusive thoughts. We included these studies due to a secondary interest in comparing rates of intrusive thoughts between groups with and without psychopathology. Thus, we used only controlled comparisons for the primary analyses, and included studies with lower levels of control for secondary estimates of more general intrusive thought differences across groups. Finally, we included studies with crossover designs (e.g., a group monitoring then suppressing compared to a group suppressing then monitoring), but only used data recorded prior to the crossover (i.e., in the above example, only the initial between-groups comparison was included).

A total of 59 studies (51 published and 8 unpublished) met criteria for the review. While we were unable to obtain complete data for every study, 52 studies (44 published and 8 unpublished) contributed data either from the manuscript, through correspondence with the study author(s), or based upon effect sizes reported in alternate manuscripts (e.g., Abramowitz et al., 2001). Of these 52 studies, 33 studies



Note. Words in bold print indicate the comparisons used in the analyses. "Initial Enhancement"and "Rebound" refer to the difference between thought suppression and control instructions within psychopathological and non-clinical groups individually

Fig. 1. Controlled Comparisons of thought suppression and control instructions between samples with and without psychopathology.

provided data permitting controlled comparisons of thought suppression difficulties within psychopathological samples, and 25 studies also provided data for controlled comparisons within non-clinical samples. Nineteen studies provided data only for uncontrolled comparisons between psychopathology and non-psychopathology samples.

3.2. Study variables

For each study, diagnosed or analogue classification of participants was coded, along with the publication status. It was not possible to record comorbidity within samples with psychopathology because too few studies provided this information. We also coded a series of demographic (e.g., age) and methodological (e.g., valence of thought) variables; analyses for these variables can be found in the Appendix.

3.3. Calculation of effect sizes and analysis plan

We used Cohen's *d* as the common metric for comparison across studies (following procedures described in Hunter & Schmidt, 2004), because Cohen's *d* has been recommended for combining studies that primarily report the results of ANOVAs and t-tests (Johnson & Eagly, 2000). All Cohen's *d*s were calculated by subtracting the mean of participants following control instructions from the mean of those following suppression instructions, then dividing by the pooled standard deviation (Cohen, 1988). When means and standard deviations were not provided in the study, the results of statistical tests (e.g., *t* or *F* values) were used to estimate effect size values (Ray & Shadish, 1996). For several older studies for which sufficient data could not be obtained, estimates from a previous meta-analysis were used (i.e., Abramowitz et al., 2001). For all effect sizes, we used Hedges (1982) to adjust effect size estimates according to the sample size. According to Cohen's (1988) guidelines, Cohen's *d*s of .2, .5, and .8 correspond to small, medium and large effect sizes respectively.

The studies used numerous types of dependent measures, including thought frequency, thought duration, reaction time tasks measuring thought activation, and retrospective estimates of thought recurrence. To increase ease of interpretation, all effect sizes were calculated so that positive numbers reflect greater activation of an intrusive thought. For the comparisons of thought suppression to control instructions, effect sizes are reported so that positive numbers always correspond to more intrusive thoughts associated with suppression relative to control instructions. For example, an effect size of $d = .30$ would indicate that participants randomly assigned to suppress experienced a small to medium increase in intrusive thoughts over participants randomly assigned to control instructions.

For studies that provided more than one comparison to a reference suppression group (e.g., comparisons for two different types of thoughts, or comparisons to two types of control groups following different control instructions), effect sizes were averaged to provide one estimate for that study. This avoids potentially biasing results in favor of the studies using a greater number of dependent measures. However, studies including more than one type of psychopathology group were permitted to contribute one effect size for every diagnosed or analogue sample available.

To combine effect sizes across studies (e.g., within a given category of psychopathology), we weighted each effect size using inverse variance weights. These weights are computed by dividing one by the squared standard error of the effect size, and are considered to provide optimal, efficient summaries of population effect sizes (Hedges & Olkin, 1985; Sánchez-Meca & Marín-Martinez, 1998). Weighted effect sizes are denoted by d_+, and for all effect size statistics, N_S refers to the number of samples combined to provide the mean effect size. All analyses were conducted using SPSS macros (Wilson, 2006) designed for aggregating across effect sizes and conducting analogs to multiple regression analyses with effect sizes as dependent variables (Lipsey & Wilson, 2001).

Meta-analyses generally use either fixed or random effects models to estimate effect sizes. Fixed effects models are used when the study effect sizes are assumed to be randomly distributed around one common population effect size, and random effects models allow for the possibility that study effect sizes may be distributed around different population effect sizes. While fixed effects models tend to have more power, random effects models better generalize to the entire set of studies (both those in existence and yet to be conducted) on the topic (Hedges & Vevea, 1998). Because we were interested in wider generalization, and expected heterogeneity across types of psychopathology and methodological factors, we used random effects models

for all analyses (Hedges & Vevea, 1998). Given the resulting reduced power, and to maximize interpretability and statistical validity, we focus on the magnitude of effects (i.e., effect sizes), and deemphasize significance testing. We believe that the focus on effect sizes is necessary for ascertaining meaningful differences that could be obscured by Type II error when focusing on significance testing. This is evident particularly when considering that a small number of samples may lead to a wide confidence interval around an effect size estimate, but still represents many participants (e.g., 5 samples of participants with GAD totaling 222 participants).

With this approach, we primarily focused on the size of differences in initial enhancement or rebound effect sizes (i.e., d_+) between groups (e.g., psychopathology vs. non-clinical). For example, within samples with depression, an effect size of $d_+ = .30$ during the initial enhancement thinking period would indicate that the depressed participants randomly assigned to suppress experienced a small to medium increase in intrusive thoughts over depressed participants randomly assigned to control instructions. A comparison of the same effect size calculation within corresponding non-clinical samples (e.g., $d_+ = .10$) would reveal a raw difference of $d_+ = .20$ between the groups, a small effect according to Cohen (1988). In this case, one would conclude that thought suppression may be associated with greater intrusive thought recurrence compared to control instructions for the depressed sample relative to the non-clinical sample, with the magnitude of the effect being small. This method of examining mean differences in Cohen's *d* between groups is widely used (e.g., Abramowitz et al., 2001).

As a secondary, complementary effect size, we also calculated the percentage of heterogeneity across a set of studies explained by psychopathology group status (Lipsey & Wilson, 2001). This approach is less informative about the magnitude of differences between psychopathology and non-clinical groups. Instead, this effect size scales the effect size difference between psychopathology and non-clinical groups by the total variation across the studies being combined in effect sizes (i.e., the total heterogeneity). For each analysis, the heterogeneity in effect sizes across combined studies was assessed by computing the *Q* statistic, which has a chi-square distribution with $k - 1$ degrees of freedom, where *k* signifies the number of effect sizes being combined. The heterogeneity accounted for by the predictor variable (i.e., psychopathology status) was then divided by the total heterogeneity across studies to provide an analog R^2 value. This analog R^2 value can be interpreted according to Cohen's (1988) guidelines that values of approximately 1, 9, and 25% correspond to small, medium and large effects respectively. For instance, in the previous example we might find that whether or not a sample was depressed explains $R^2 = 30\%$ of the heterogeneity in effect sizes among the studies that were combined for the comparison. We chose to include this method to quantify heterogeneity due to the many methodological variations present in thought suppression research (Purdon, 2004).

The analysis involved four stages: first, we conducted preliminary descriptive statistics for the entire sample. Second, we examined the controlled comparisons of initial enhancement and rebound effects *between* psychopathological and non-clinical samples. Third, we conducted controlled comparisons *within* different forms of psychopathology. Fourth, we conducted uncontrolled comparisons between psychopathology and non-clinical samples to examine differences in intrusive thought activation. We report confidence intervals in the text for comparisons conducted across types of psychopathology, and report these in the tables for all other results.

4. Results

4.1. Preliminary analyses

The 33 studies that permitted at least one within-psychopathology group comparison of thought suppression to control instructions provided 58 samples (33 psychopathological and 25 non-clinical). This final pool included 2344 participants, had an average age of 25.94 (range 18.70–47.00), and was 69% female (range 22–100%) among those studies reporting age or gender. Published studies demonstrated a medium sized difference of a greater initial enhancement effect (difference of $d_+ = .50$, $N_S = 32$) than unpublished studies, but did not show much difference for the rebound thinking period ($d_+ = .05$, $N_S = 22$).

To replicate the general findings from Abramowitz et al. 's (2001) examination of thought suppression effects in primarily non-clinical samples, we first examined the effects of thought suppression for all samples (including those with and without psychopathology). In accordance with Abramowitz and colleague's previous meta-analysis, during the initial enhancement thinking period participants showed a small to medium negative effect of thought suppression ($d_+ = -.36$, $N_S = 56$, 95% CI: −.52, −.20), indicating less recurrence during thought suppression compared to control instructions. Also in accordance with their previous findings, participants showed a small positive rebound effect ($d_+ = .15$, $N_S = 40$, 95% CI: .04, .27), indicating increases in intrusive thoughts after suppression compared to control instructions. Given the minimal overlap of the current studies with those included in the previous meta-analysis (12 studies), the similarity of the current estimates to those in Abramowitz et al. (i.e., $d_+ = -.35$ for the initial enhancement effect, $d_+ = .30$ for the rebound effect) suggests that the general pattern of findings for thought suppression is robust.

Next we calculated the correlation between initial enhancement and rebound effect sizes, finding a medium, positive relationship ($r = .43$, $p < .01$, $N_S = 38$ samples included both measurements). The correlation was similar in psychopathological ($r = .49$, $p < .03$, $N_S = 21$) and non-clinical groups ($r = .36$, $p = .16$, $N_S = 17$). While these correlations do not explain processes occurring at the participant level, they suggest that studies with greater initial enhancement effects also tend to have greater rebound effects.

4.2. Difficulty with thought suppression according to the presence or absence of psychopathology

Next, we tested the proposal that samples with psychopathology would experience greater difficulties with suppression than non-clinical samples (the curved arrow comparisons bolded in Fig. 1). These comparisons involved separately computing initial enhancement and rebound effects within all psychopathology and non-clinical samples, then comparing the groups using analog weighted ordinary least squares regression (Lipsey & Wilson, 2001). See Table 1 for a summary of the differences. Results indicated that during the initial enhancement thinking period, difficulty with thought suppression attempts was similar between psychopathological ($d_+ = -.39$, $N_S = 32$) and non-clinical samples ($d_+ = -.31$, $N_S = 24$). Contrary to expectations, there was also little difference for the rebound thinking period comparison between samples with psychopathology ($d_+ = .12$, $n = 22$) and the non-clinical samples ($d_+ = .19$, $n = 18$). These comparisons yielded only minimal differences in the magnitude of effects between psychopathology and non-clinical samples, and suggest that thought suppression is associated with similar recurrence of thoughts (compared to control instructions) in samples with and without psychopathology.

4.3. Variation in the effects of thought suppression among psychopathology groups

Next we examined variation among the specific types of psychopathology represented in the studies. First, we broke down the overall sample into diagnosed and analog groups. For the initial

Table 1
Differences between psychopathological and non-clinical samples during the initial enhancement and rebound thinking periods.

	Initial enhancement thinking period						Rebound thinking period					
Type of psych.	d_+	95% CI	R^2	*Q*	N_S	N_P	d_+	95% CI	R^2	*Q*	N_S	N_P
OCD	−.17	−.60, .26	4.1%	14.47	16	600	−.13	−.49, .23	5.4%	9.54	13	486
PTSD	−.02	−1.09, 1.05	0.0%	4.91	7	219	−.14	−.87, .60	4.5%	2.88	5	136
GAD	.09	−2.89, 3.08	0.1%	3.21	5	222	.12	−.56, .80	13.3%	.91	4	150
Depression	.39	−.64, 1.42	22.8%	2.41	4	164					0	0
Specific	−.24	−.88, .41	8.6%	6.11	8	310	−.05	−.66, .56	0.6%	3.89	6	226
Trait anxiety	−.42	−1.24, .40	14.0%	7.12	8	414	−.25	−1.02, .53	8.0%	4.90	6	340
Other	.03	−.75, .80	0.1%	7.03	8	375	−.03	−1.02, .96	0.1%	4.31	6	314
Total	−.08	−.41, .24	0.4%	57.41	56	2304	−.07	−.31, .16	1.1%	38.70	40	1653

Note. $^*p<.05$. Psych. refers to psychopathology. d_+ refers to the weighted Cohen's *d*, and represents the mean effect size difference between groups with and without psychopathology for a particular comparison. R^2 refers to the analog R^2 value reflecting the heterogeneity accounted for by psychopathology status divided by the total heterogeneity among combined studies. *Q* refers to the heterogeneity statistic. N_S refers to the total number of samples with and without psychopathology included in a comparison. N_P refers to the total number of participants included in a comparison.

enhancement thinking period, we found that the difference in effect sizes was a small to medium effect in the direction of a greater initial enhancement effect for diagnosed samples ($d_+ = -.23$, $N_S = 11$) than for analogue samples ($d_+ = -.48$, $N_S = 21$). For the rebound thinking period, the effect sizes for diagnosed ($d_+ = .17$, $N_S = 6$) and analogue samples ($d_+ = .11$, $N_S = 16$) showed minimal differences. These comparisons supported the available cognitive resources mechanism, as the greater initial enhancement effect by diagnosed samples was consistent with a greater depletion of cognitive resources than in analogue samples.

We then examined initial enhancement and rebound effects for each specific type of psychopathology. All types of psychopathology for which there were fewer than two psychopathology and two non-clinical samples for comparison were combined into an "other" category. For the initial enhancement effect comparisons, this "other" category included two social anxiety samples, one sample each with symptoms of nicotine dependence, insomnia, and eating disorder, and three non-clinical comparison groups. For rebound effect comparisons, the same samples were used, except data for the nicotine dependence sample and one non-clinical comparison group were unavailable. We focus here on the differences *between* psychopathology versus non-clinical groups, but Fig. 2 also breaks down initial enhancement and rebound effect sizes *within* psychopathology and non-clinical groups (e.g., suppression relative to control instructions for depressed samples). It should be noted that even non-clinical control groups show considerable heterogeneity that is likely due to methodological variations across studies.

4.4. OCD and PTSD

For the initial enhancement thinking period, consistent with the proposal that individuals with OCD would be highly motivated to suppress their intrusive thoughts, participants with OCD symptoms showed less of an initial enhancement effect relative to non-clinical participants (difference of $d_+ = -.17$, $N_S = 16$), although the effect was small. This difference indicated that to a small degree, OCD samples experienced less amplification of intrusive thoughts while suppressing (compared to monitoring their thoughts) than did corresponding non-clinical groups. For the rebound thinking period, participants with OCD symptoms unexpectedly showed a small effect size difference ($d_+ = -.13$, $N_S = 13$) in the direction of a smaller rebound effect than non-clinical participants.

For participants with PTSD symptoms, suppression difficulties were similar to non-clinical samples ($d_+ = -.02$, $N_S = 7$) during the initial enhancement thinking period, which did not appear to be consistent with an increased motivation to suppress. For the rebound thinking period, participants with PTSD symptoms showed a small difference ($d_+ = -.14$, $N_S = 5$) in the direction of a smaller rebound effect than non-clinical samples. Together, the results for participants with symptoms of OCD and PTSD offered partial support for the motivation to suppress mechanism, but did not appear to support the other two possible mechanisms.

4.5. GAD and depression

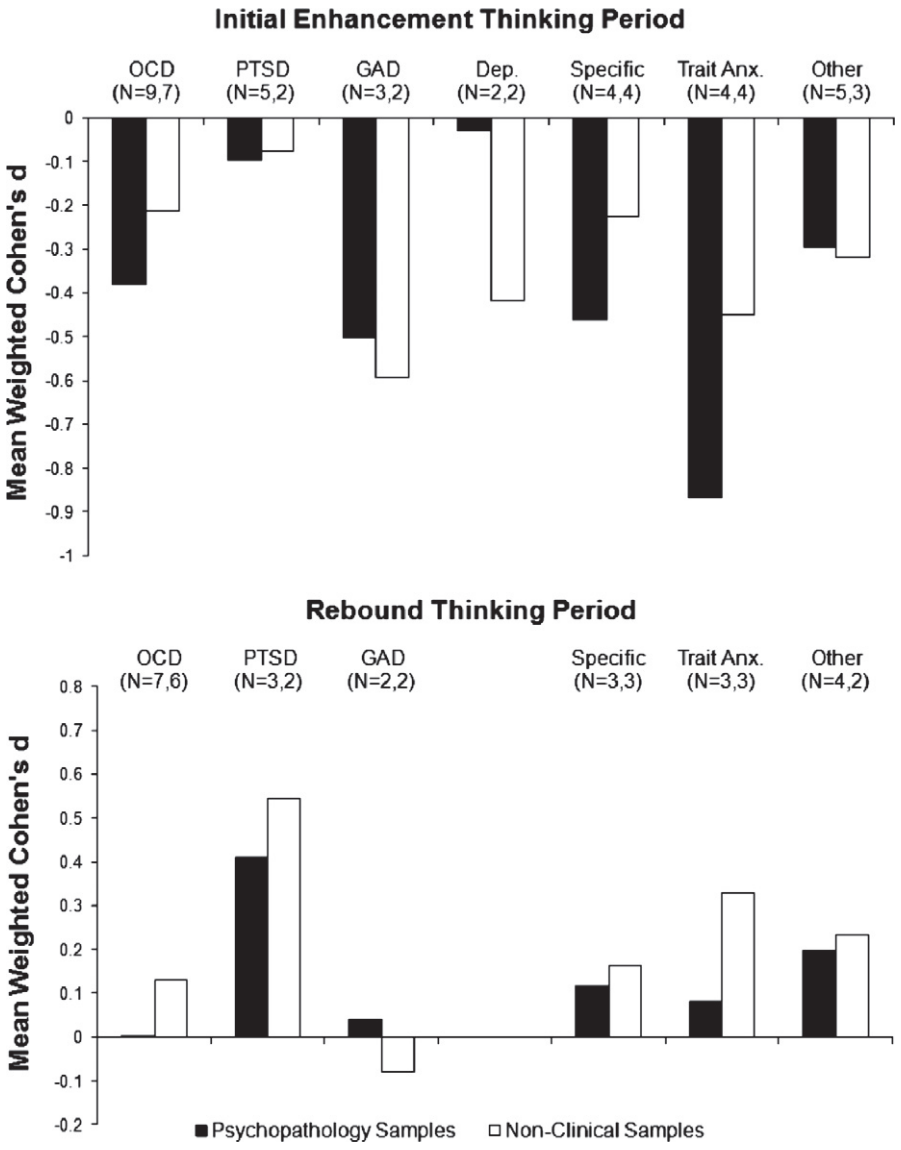
In line with a decreased motivation to suppress, participants with GAD symptoms showed a similar initial enhancement effect compared to non-clinical groups (difference of $d_+ = .09$, $N_S = 5$) during the initial enhancement thinking period. For the rebound thinking period, participants with GAD symptoms showed a small effect size difference from non-clinical groups ($d_+ = .12$, $N_S = 4$) in the direction of a greater rebound effect. Also consistent with a decreased motivation to suppress, participants with symptoms of depression showed a small to medium effect of greater initial enhancement compared to non-clinical participants (difference of $d_+ = .39$, $N_S = 4$). There were not sufficient samples to estimate the magnitude of differences for the rebound thinking period. Together, the findings for GAD and depression largely supported the motivation to suppress mechanism, as we expected that less motivation to suppress would result in an equivalent or greater initial enhancement effect. However, the small rebound effect for the sample with GAD symptoms indicated that this support did not extend to the follow-up rebound thinking period, in which decreased motivation to suppress would be expected to result in an equivalent or decreased rebound effect compared to non-clinical samples.

4.6. Specific phobias, trait anxiety, and other forms of psychopathology

For the remaining forms of psychopathology, the available cognitive resources mechanism predicted greater difficulties with suppression than non-clinical samples, but there were not obvious expectations based upon the other mechanisms. Results indicated that participants with specific phobia symptoms experienced a small to medium effect in the direction of a smaller initial enhancement effect ($d_+ = -.24$, $N_S = 8$) than non-clinical participants. Participants with specific phobia symptoms showed minimal differences ($d_+ = -.05$, $N_S = 6$) for the rebound thinking period when compared to the corresponding non-clinical samples.

Next, participants with high trait anxiety showed a small to medium effect in the direction of a smaller initial enhancement effect than non-clinical participants ($d_+ = -.42$, $N_S = 8$), indicating that participants with high trait anxiety experienced fewer intrusive thoughts associated with thought suppression (compared to monitoring) than non-clinical groups. For the rebound thinking period, participants with trait anxiety continued the pattern of fewer intrusive thoughts associated with thought suppression, showing a small to medium difference in the direction of a *smaller* rebound effect ($d_+ = -.25$, $N_S = 6$) than participants without trait anxiety.

Finally, for the combined "other" types of psychopathology, the psychopathology group was similar to non-clinical samples for both



Note. The sample sizes in parentheses refer to the number of samples for the psychopathology and corresponding non-clinical groups respectively. Positive effect sizes refer to greater activation of thoughts during thought suppression compared to control instructions. OCD refers to Obsessive-Compulsive Disorder, PTSD refers to Post-Traumatic Stress Disorder, GAD refers to Generalized Anxiety Disorder, Dep. refers to Depression, Specific refers to Specific Phobias, Trait Anx. refers to trait anxiety, and Other refers to all other forms of psychopathology analyzed.

Fig. 2. Effect sizes for the initial enhancement and rebound thinking periods according to type of psychopathology.

the initial enhancement ($d_+ = .03$, $N_S = 8$) and rebound ($d_+ = -.03$, $N_S = 6$) thinking periods. Together, among specific phobias, trait anxiety, and other forms of psychopathology, trait anxiety stood out by showing the greatest differences from non-clinical samples of any type of psychopathology measured in this meta-analysis. Interestingly, these differences were in the direction of less difficulty during both the initial enhancement and rebound thinking periods.

Across the types of psychopathology measured in this meta-analysis, the overall pattern of results suggested few differences between psychopathological and non-clinical samples in intrusive thoughts associated with thought suppression. Initial enhancement effects showed slightly greater differences than rebound effects, and appeared to be most consistent with predictions based upon the motivation to suppress mechanism. Nonetheless, the majority of these effects were small.

4.7. Differences between initial enhancement and rebound effects

Another possible way to examine thought suppression outcomes that may more closely parallel the individual experience of thought suppression is to examine the differences between thought recurrence associated with thought suppression during the initial enhancement and rebound thinking periods (Wenzlaff & Wegner, 2000). For example, if thought suppression is successful during the initial enhancement thinking period, then shows a major rebound, the subjective experience of thought suppression would be different (and perhaps more alarming) than if the effects of thought suppression remained rather steady across occasions. To examine this question, we estimated initial enhancement and rebound effects separately within each type of psychopathology, then calculated the difference in magnitude between the two (this allowed us to include

studies that did not include both initial enhancement and rebound effects in the same study). All differences between initial enhancement and rebound effects were positive, indicating greater intrusive thoughts relative to control instructions for the rebound effect compared to the initial enhancement effect. We then compared the size of the difference between the initial enhancement and rebound effects according to each type of psychopathology. These comparisons are reported so that positive numbers indicate greater increases (or smaller decreases) in intrusive thoughts for samples with psychopathology than the corresponding non-clinical samples.

As with the within-disorder comparisons for specific types of psychopathology above, there were not substantial differences between psychopathology and non-clinical samples. Participants with OCD symptoms showed differences similar to non-clinical participants ($d_+ = .04$), as did participants with symptoms of GAD ($d_+ = .03$), and the combined "other" group ($d_+ = -.06$). Participants with symptoms of PTSD showed slightly smaller differences than non-clinical samples ($d_+ = -.12$), whereas participants with symptoms of specific phobias ($d_+ = .19$) and trait anxiety ($d_+ = .17$) showed slightly larger differences than non-clinical groups. None of these comparisons yielded differences greater than a small effect size.

4.8. Presence of psychopathology and recurrence of intrusive thoughts

As a secondary question, we were interested in examining differences between psychopathological and non-clinical samples in the rate of intrusive thoughts experienced during uncontrolled thinking periods. In other words, these comparisons were between psychopathological and non-clinical samples where all participants received one type of instruction (i.e., baseline, control, or suppression) for a given thinking period, rather than comparing participants randomly assigned to control or suppression instructions, as occurred in the controlled comparisons. In addition to the initial enhancement and rebound thinking periods, we also examined comparisons for baseline thinking periods, which a subset of studies included prior to the initial enhancement thinking period (see Fig. 1). Together, these uncontrolled comparisons are not informative about the effects specifically due to thought suppression, but rather detect general rates of intrusive thoughts. Given the lack of major differences for the controlled comparisons, this comparison estimated the degree to which samples with psychopathology experience greater intrusive thoughts than non-clinical samples, independent of thought suppression instructions.

The uncontrolled comparisons revealed consistently higher rates of intrusive thoughts for samples with psychopathology across each type of thinking period. For the baseline thinking period, samples with psychopathology experienced more intrusive thoughts than non-clinical samples ($d_+ = .43$, $N_S = 12$, 95% CI: .24, .63). For the initial enhancement thinking period, samples with psychopathology experienced more intrusive thoughts than non-clinical samples regardless of whether they were following control ($d_+ = .46$, $N_S = 20$, 95% CI: .25, .67) or suppression ($d_+ = .31$, $N_S = 43$, 95% CI: .20, .42) instructions. Further, for the rebound thinking period, samples with psychopathology experienced more intrusive thoughts than non-clinical samples whether they had received control ($d_+ = .42$, $N_S = 14$, 95% CI: .21, .62) or suppression ($d_+ = .32$, $N_S = 28$, 95% CI: .12, .52) instructions. The small to medium magnitude of differences was consistent across all thinking periods and indicates that samples with psychopathology experience greater levels of intrusive thoughts during experimental thinking periods. When these findings are combined with the results of the controlled comparisons above, a picture emerges suggesting that while groups with psychopathology may consistently exhibit higher levels of intrusive thoughts during thought suppression, very little of this difference appears to be attributable to actual thought suppression efforts.

5. Discussion

The current study reviewed evidence for the link between thought suppression and the persistent intrusive thoughts evident among many populations with psychopathology. As expected, samples with psychopathology consistently exhibited higher levels of intrusive thoughts than healthier samples during thought suppression. However, a similar difference in intrusive thoughts was evident when participants followed control thinking instructions, making it unlikely that there is any special amplification of intrusive thoughts due to thought suppression for samples with psychopathology in general. This finding challenges the assumption that thought suppression uniquely enhances the recurrence of persistent intrusive thoughts across clinical disorders and highlights the importance of using proper control group comparisons in thought suppression research.

In comparisons examining specific types of psychopathology, most types demonstrated small or no differences relative to non-clinical samples. The greatest difference for both the initial enhancement and rebound thinking periods occurred for samples with high trait anxiety; these samples experienced smaller initial enhancement and rebound effects to a small to medium degree compared to non-clinical samples. Samples with OCD symptoms reported slightly smaller initial enhancement effects than non-clinical samples, consistent with a higher motivation to suppress. However, samples with PTSD did not differ during the initial enhancement thinking period, in line with a predicted higher motivation to suppress, and both groups showed smaller rebound effects to a small degree compared to non-clinical samples, inconsistent with predictions of the reviewed mechanisms. Finally, as expected by the motivation to suppress mechanism, samples with symptoms of GAD and depression both showed similar or greater initial enhancement effects than non-clinical samples. Unexpectedly, the samples with GAD symptoms experienced slightly greater rebound effects than corresponding non-clinical groups. Overall, these results suggest some heterogeneity among types of psychopathology that might be accounted for by the proposed mechanisms; however, most effects were small, and it appeared that the support for the motivation to suppress mechanism was more consistent for the initial enhancement thinking period than the rebound thinking period.

5.1. Proposed mechanisms underlying thought suppression

There was mixed support for hypotheses based upon the three mechanisms proposed to underlie thought suppression ability. First, there appeared to be no evidence of generally increased difficulty with thought suppression in psychopathology, contrary to the hypothesis that available cognitive resources would be limited in samples with psychopathology compared to non-clinical samples, leading to difficulties inhibiting intrusive thoughts (Wegner, 1994). These results contradict expectations that the high levels of negative affect in groups with psychopathology would be associated with worsened thought suppression performance through the mechanism of depleted cognitive resources. There are several explanations that could account for these findings, including insufficient group differences in cognitive resources to have an impact for individuals with psychopathology, or insufficient manipulation of cognitive resources in previous studies. Indeed, few of the studies included in this meta-analysis added additional cognitive demands during thought suppression (Wegner, Erber, & Zanakos, 1993). However, an alternative possibility is that the effortful (resource-demanding), conscious thought suppression process may become more automatic and effortless over time (Wegner, 1994). Given the frequency of thought suppression attempts among individuals with psychopathology, this could explain how cognitive resources between individuals with and without psychopathology could differ but not lead to divergences in measured thought suppression performance. In either case, future

work directly measuring and manipulating cognitive resources will be useful for disentangling the contributions of available cognitive resources to theories of thought suppression in psychopathology.

The second proposed mechanism, motivation to suppress, received the strongest (albeit indirect) support from the data in its predictive power during the initial enhancement thinking period. Results for samples with symptoms of OCD, GAD, and depression all followed expectations according to this mechanism for the initial enhancement thinking period. For samples with OCD symptoms, there was a reduced initial enhancement effect relative to healthier participants, consistent with the prediction of increased suppression effort in this group. Depression and GAD showed equivalent or greater initial enhancement effects than non-clinical samples, perhaps reflecting similar or less intensive suppression effort compared to non-clinical participants. The only type of psychopathology which did not fit the predicted pattern for the initial enhancement effect was PTSD. Individuals with PTSD symptoms were expected to show high motivation to suppress, leading to a reduced initial enhancement effect, but surprisingly there was no difference compared to non-clinical samples. Thus, while the estimated differences were generally small, the motivation to suppress mechanism predicted heterogeneity fairly successfully during the initial enhancement thinking period.

Based upon prominent theories of thought suppression, samples expending greater suppression effort were expected to experience higher subsequent rebound effects (Wenzlaff & Wegner, 2000). However, the rebound effects did not follow the predicted pattern of an increased rebound effect for OCD and PTSD and an equivalent or decreased rebound effect for GAD. Instead, the current results suggest that other factors in addition to motivation or consequent suppression effort are needed to predict rebound effects (though it should be noted that suppression effort was not measured directly in most studies so this is an inference).

For the third mechanism, meta-cognition about unsuccessful thought suppression, the evidence was difficult to interpret, as there was little preexisting empirical evidence to indicate which disorders other than OCD would have been expected to show differences according to meta-cognition. However, the results for OCD did not support this mechanism, as samples with OCD symptoms demonstrated only small differences in the direction of smaller enhancement and rebound effects than non-clinical samples. It may be that the tendency to experience high levels of intrusive thoughts and apply maladaptive meanings is elevated for samples with OCD symptoms whether they are suppressing or following control instructions. As a result, the influence of maladaptive metacognition would be less apparent using the thought suppression paradigm.

The overall pattern of results did not support a relationship between maladaptive meta-cognition and increased recurrence of intrusive thoughts as a general characteristic of psychopathology. This is unsurprising, as the content of meta-cognition is likely to vary tremendously for different types of psychopathology. However, this is a critical factor to examine for individual types of psychopathology, as meta-cognition is a crucial component of theories of intrusive thoughts (e.g., Clark, 2001; Wells & Matthews, 1996). It is also important to acknowledge that it is unclear whether meta-cognition about unsuccessful thought suppression differs from meta-cognition about intrusive thought recurrence in general. It is plausible that maladaptive meta-cognition could lead to increased intrusive thoughts whether a person is suppressing or monitoring. It is also critical to test whether meta-cognition about unsuccessful thought suppression might have a greater impact on distress following suppression than on the recurrence of intrusive thoughts (Purdon, Rowa, & Antony, 2005).[1] It may be that individuals with psychopathology suffer increased distress due to their beliefs about the meaning of unsuccessful thought suppression, even when their suppression efforts result in recurrence levels similar to non-clinical samples.

While these three possible mechanisms (available cognitive resources, motivation to suppress, maladaptive metacognition) were described independently, it is likely that in practice they are interactive. For example, an unsuccessful thought suppression attempt that is interpreted as a sign of weakness could lead to increased negative affect and effort suppressing (followed by consequent resource depletion), which in turn could lead to an increased rebound effect. Disorder-specific theories that delineate specific sequences and combinations of these mechanisms will help to build more nuanced understandings of thought suppression in psychopathology.

5.2. Trait anxiety and negative mood

Turning to the other specific forms of psychopathology tested, there were not strong hypotheses concerning trait anxiety as a unique predictor of thought suppression effects. However, results indicated that trait anxiety was associated with smaller initial enhancement and rebound effects compared to non-clinical groups, suggesting that perhaps milder levels of state anxiety or negative affect are actually helpful when suppressing, at least in the short-term period reflected by these studies. Previous work by Wyland and Forgas (2007) has suggested that among non-clinical samples, mild negative mood is associated with less of an initial enhancement effect (although a greater rebound effect) than positive mood. Additionally, other work focusing on predictors of the rebound effect has suggested that trait anxiety is associated with less of a rebound effect (Rutledge, Hancock, & Rutledge, 1996). Perhaps this occurs because mild or even moderate negative mood can promote focused, detail-oriented information processing that is helpful for focusing on distracters. For example, focusing on a single, specific distracter tends to be helpful during suppression (Wegner et al., 1987), and negative mood tends to promote localized processing that should be useful for focusing on an individual target (Bless, 2001; Gasper & Clore, 2002). Meanwhile, positive mood is associated with a global, assimilative style (seeing the forest versus the trees) that may be detrimental to focusing on a distracter during suppression (Wyland & Forgas, 2007). Notwithstanding, the comparisons between diagnosed and analogue samples suggest there may be limits to the benefits of negative mood with thought suppression. In these comparisons, diagnosed samples experienced greater enhancement than analogue samples, possibly indicating that diagnosed samples are above the critically high level of negative affect at which negative affect becomes unhelpful. It should be noted that the differences between diagnosed and analogue samples could also be due to differences in attentional control, neuroticism, or other factors that tend to vary in intensity between the two types of samples. It will be important in future research to measure negative affect and thought suppression strategy use (e.g., focusing on a distracter) more consistently in thought suppression paradigms to determine why analogue and diagnosed samples differ, and why trait anxious persons experience low recurrence.

5.3. Advancing research on psychopathology and thought suppression

The current review suggests that focusing exclusively on thought recurrence as the outcome of thought suppression is likely to yield only small differences for samples with psychopathology. Further, there is increasing evidence that this focus misses the larger context in which thought suppression occurs - the subjective emotional experience of thought suppression. This shift in focus parallels advances in cognitive theories of intrusive thoughts, which suggest that having intrusive thoughts is a normative phenomenon; instead, the way an individual interprets those thoughts is expected to lead to benign versus serious outcomes (Obsessive Compulsive Cognitions Working

[1] We appreciate an anonymous reviewer for bringing this possibility to our attention.

Group, 2001; Rachman, 1997). Similarly, having difficulties with thought suppression is a common experience (Magee & Teachman, 2007); it is the way an individual interprets that experience that may be key. Previous discussions of thought suppression have frequently implied that people having difficulties with thought suppression often ascribe negative meaning to their difficulties. For instance, in the classic "white bear" study, Wegner et al. (1987) wrote: "The person finds the thought hard to suppress and may soon wonder why this particular thought is so insistent"…"It is at this point that the person becomes alarmed, noticing that an unusual degree of preoccupation is underway" (p. 11–12). One infers from these descriptions that the outcome would be different if the individual instead interpreted the thought recurrence more benignly: e.g., "This thought keeps returning, but thoughts do this occasionally. It doesn't mean anything about me." Initial evidence from studies of thought suppression in OCD supports this idea: interpretations about thought suppression failure differ between OCD and non-clinical samples (Tolin, Abramowitz, Hamlin, Foa, & Synodi, 2002), and can mediate subsequent differences in intrusive thought recurrence and distress (Magee & Teachman, 2007). Interpretations about thought suppression can also be shifted among people with psychopathology (Najmi et al., 2010) and without psychopathology (Förster & Liberman, 2001), although it is unclear the degree to which these specific belief shifts generalize to broader beliefs about intrusive thoughts (Najmi, Riemann, & Wegner, 2009). Nonetheless, shifting beliefs about thought suppression has received limited empirical attention compared to studies of recurrence of thoughts and distress (Wenzlaff & Wegner, 2000). The proposed mechanisms in this review identify areas that can be more directly assessed and manipulated within specific forms of psychopathology.

One finding that is frequently overlooked in the thought suppression literature is that individuals can successfully suppress thoughts for short periods of time (Abramowitz et al., 2001). The current study replicated this finding among both psychopathological and non-clinical samples, each of whom reported less recurrence during suppression instructions than control instructions during the initial enhancement thinking period. It will be helpful for future research to examine the conditions under which this short term suppression success may contribute to positive or negative long-term consequences for individuals with or without psychopathology.

The studies surveyed also raise several methodological considerations for future research. As these psychopathology studies are quasi-experimental, they do not allow one to determine why or how the use of thought suppression developed in the first place. While a few pioneering studies have begun to examine such vulnerabilities in longitudinal designs (e.g., Wenzlaff & Luxton, 2003), more work is needed. Another issue is the difficulty of interpreting comparisons of thought recurrence for different types of psychopathology. The reason for this difficulty is that the samples may follow control instructions differently, or may respond differently to standardized versus ideographic intrusive thoughts; for example, in disorders in which motivation to suppress is high, such as OCD, participants following control instructions usually spontaneously suppress their thoughts to a higher degree than participants with other disorders, or non-clinical samples (Purdon & Clark, 2000). Frequent intraindividual assessments using multiple target thoughts and over longer time periods, in which an individual could be tracked while sometimes suppressing and sometimes using monitoring or other strategies, such as distraction, may be a useful supplement to the current designs. These variations could help mitigate the tradeoffs that are present with each design choice.

6. Limitations and conclusion

While the current meta-analysis included sufficient studies to estimate effect size differences between samples with specific types of psychopathology and non-clinical samples, the power to estimate the possible range of these effect sizes was limited. Further, several types of psychopathology had to be combined into a less informative 'other' category due to their limited individual sample sizes. Nonetheless, mean effect size estimates for individual types of psychopathology outside of this 'other' category were based on a large number of participants for each psychopathology comparison, ranging from 136 participants (the rebound effect comparison for PTSD) to 600 participants (the initial enhancement effect comparison for OCD), with an average of 297 participants per comparison. Additionally, while the proposed mechanisms were used to frame expectations and directions for future research, the relationships between the mechanisms and specific types of psychopathology are clearly complex and must be tested in direct examinations.

The current study found no general differences in the recurrence of thoughts associated with thought suppression between psychopathology and non-clinical groups, and minimal group differences for several types of psychopathology that have been considered prime candidates for difficulties with thought suppression, including OCD. Future research should carefully consider pre-existing differences in thought activation when interpreting the results of thought suppression paradigms. Further, integration of cognitive theories of intrusive thoughts that describe 'subjective' reactions to thought suppression, such as distress and interpretations about unsuccessful thought suppression, are needed to understand the complex relationship between thought suppression and intrusive thoughts in psychopathology.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at doi:10.1016/j.cpr.2012.01.001.

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** Indicates data were available for controlled comparisons of samples with psychopathology.

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