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Mechanisms of change during group metacognitive therapy for repetitive negative thinking in primary and non-primary generalized anxiety disorder

Peter M. McEvoy[1,2], David M. Erceg-Hurn[1,3], Rebecca A. Anderson[1,2], Bruce N. C. Campbell[1], & Paula R. Nathan[1,3]

[1] *Centre for Clinical Interventions, Perth, Australia*

[2] *School of Psychology and Speech Pathology, Curtin University, Perth, Australia*

[3] *University of Western Australia, Perth, Australia*

Accepted, *Journal of Anxiety Disorders*

Correspondence concerning this article should be addressed to Peter M McEvoy, Ph.D., School of Psychology and Speech Pathology, Curtin University, GPO Box U1987, Perth, Western Australia, 6845, Australia. Phone: +618 9266 5110. Fax: +618 9226 2464. Email: peter.mcevoy@curtin.edu.au

Abstract

Repetitive negative thinking (RNT) is a transdiagnostic process that serves to maintain emotional disorders. Metacognitive theory suggests that positive and negative metacognitive beliefs guide the selection of RNT as a coping strategy which, in turn, increases psychological distress. The aim of this study was to test the indirect effect of metacognitive beliefs on psychological distress via RNT. Patients (N = 52) with primary and non-primary generalized anxiety disorder attended a brief, six-week group metacognitive therapy program and completed measures of metacognitive beliefs, RNT, and symptoms at the first and final treatment sessions, and at a one-month follow-up. Prospective indirect effects models found that negative metacognitive beliefs (but not positive metacognitive beliefs) had a significant indirect effect on psychological distress via RNT. As predicted by metacognitive theory, targeting negative metacognitions in treatment appears to reduce RNT and, in turn, emotional distress. Further research using alternative measures at multiple time points during therapy is required to determine whether the absence of a relationship with positive metacognitive beliefs in this study was a consequence of (a) psychometric issues, (b) these beliefs only being relevant to a subgroup of patients, or (c) a lack of awareness early in treatment.

Key Words: Metacognitive therapy, repetitive negative thinking, indirect effects, mechanisms, generalized anxiety disorder

1.0 Introduction

Generalized anxiety disorder (GAD) is characterized by excessive and uncontrollable worry and hyperarousal (e.g., restlessness, irritability, muscle tension; American Psychiatric Association [APA], 2013). GAD has a lifetime prevalence of around 6% (Kessler et al., 2005; McEvoy, Grove, & Slade, 2011) and is highly comorbid with other anxiety disorders and depression (Brown, Campbell, Lehman, Grisham, & Mancill, 2001). In a large treatment-seeking sample (N = 1127), Brown et al. (2001) found that GAD was the most common comorbid diagnosis for individuals with major depression. GAD was also the second most common comorbid anxiety disorder for those with another primary anxiety disorder. Studies investigating treatment effects and mechanisms of change in primary and non-primary GAD therefore have the potential to have a large impact by increasing the generalizability of findings to many real world clients. A recent trial of metacognitive therapy in a sample with primary and non-primary GAD found large effect sizes that were comparable to studies using samples with primary GAD (McEvoy, Erceg-Hurn, Anderson et al., 2015). The aim of the current study was to extend these findings by examining mechanisms of change during group metacognitive group therapy for primary and non-primary GAD.

Worry has been defined as “a chain of thoughts and images, negatively affect-laden, and relatively uncontrollable” (Borkovec, Robinson, Pruzinsky, & DePree, 1983, p. 10). Worry is the cardinal feature of, but not exclusive to, GAD (APA, 2013; Harvey, Watkins, Mansell, & Shafran, 2004). Worry is elevated in a range of emotional disorders, including depression, social anxiety disorder, and panic disorder (McEvoy, Watson, Watkins, & Nathan, 2013). Worry also shares many features with other forms of repetitive negative thinking (RNT), including rumination in depression and post-event processing in social anxiety (Ehring & Watkins, 2008; McEvoy, Mahoney, & Moulds, 2010; Watkins, Moulds, & Mackintosh, 2005). These findings have lead clinical researchers to conclude that the

processes driving RNT are likely to be common across emotional disorders even though the cognitive content and associated emotional responses may differ (Watkins, 2008). Prospective and experimental research suggests that RNT contributes to the onset and maintenance of a range of emotional disorders and negative emotional states, and thus it is an important target for intervention (McLaughlin & Nolen-Hoeksema, 2011; Nolen-Hoeksema, 2000; Nolen-Hoeksema & Morrow, 1991; Park, Goodyer, & Teasdale, 2004). The common underlying pathological processes of RNT across emotional disorders, along with high rates of comorbidity, provide a strong rationale for targeting elevated RNT regardless of an individual's primary emotional disorder.

Wells and Matthews' (1996) Self-Regulatory Executive Function (S-REF) model provides a metacognitive account of processes that drive RNT across various emotional disorders. Wells (2004) defined metacognition as "…cognitive processes, strategies, and knowledge that are involved in the regulation and appraisal of thinking itself (p. 167)". Within the metacognitive model, positive and negative metacognitive beliefs are thought to drive the initiation and maintenance of RNT, respectively. Positive metacognitions are beliefs that RNT is helpful in some way, such as increasing the likelihood that one can prevent, prepare for, or cope with adverse events. The more strongly an individual adheres to these beliefs the more motivated they will be to further engage in RNT as a coping strategy. Once individuals engage in RNT, Wells (2013) argues that negative metacognitive beliefs are then activated in individuals who are vulnerable to emotional disorder. Common negative metacognitions include beliefs that RNT is uncontrollable and dangerous, which lead to maladaptive and counterproductive attempts to reduce RNT, such as thought suppression, further threat monitoring, and avoidance (known as the cognitive affective syndrome). The escalation of RNT and negative affect resulting from these counterproductive coping

strategies serves to confirm the negative metacognitive beliefs (i.e., RNT appears even less controllable and potentially more dangerous), thereby continuing the cycle.

Metacognitive therapy (MCT) is based on the S-REF model (Wells & Matthews, 1996) and aims to modify positive and negative metacognitive beliefs in order to reduce reliance on RNT as a coping strategy, and to reduce maladaptive responses to any occurrence of RNT. Trials of MCT across a range of emotional disorders have demonstrated large effects on RNT and symptoms (Bailey & Wells, 2014; Dammen, Papageorgiou, & Wells, 2014; McEvoy, Erceg-Hurn, Anderson et al., 2015; Papageorgiou & Wells, 2015; Rees & van Koesveld, 2008; Wells et al., 2012; van der Heiden & Melchior, 2014; van der Heiden, Melchior, & de Stigter, 2013; van der Heiden, Muris, & van der Molen, 2012; see Matthews, 2015). However, most MCT studies have used small samples and few have directly tested the theoretical mechanisms of change. Most of the evidence supporting the role of metacognitive beliefs in driving RNT to date is cross-sectional and correlational.

In a clinical sample with GAD van der Heiden et al. (2010) evaluated a cross-sectional hierarchical model and found that negative metacognitive beliefs mediated the relationship between the vulnerability factor of neuroticism and worry. McEvoy and Mahoney (2013) replicated and extended this model in a mixed-diagnosis sample and found that negative metacognitive beliefs mediated the relationship between neuroticism and a transdiagnostic measure of RNT. Other cross-sectional studies have found that the relationship between rumination and depression is partially mediated by negative beliefs about rumination (Papageorgiou & Wells, 2003). Roelofs et al. (2010) used structural equation modelling in a depressed sample and found that both positive and negative metacognitions lead to rumination and worry and, in turn, symptoms of emotional disorder.

Longitudinal and treatment studies have also found support for an association between metacognitive beliefs, RNT, and emotional disorder symptoms. In a healthy sample

of university students Weber and Exner (2013) found that positive metacognitive beliefs predicted rumination two months later, even after controlling for baseline rumination. Weber and Exner (2013) also found a significant indirect effect of positive beliefs about rumination on depressive symptoms via rumination. A recent trial of internet-based cognitive behavioural therapy in a mixed sample with anxiety and depression investigated the relationship between positive metacognitive beliefs, RNT, and symptom change (Newby, Williams, & Andrews, 2014). These researchers found that early reductions in positive metacognitive beliefs and RNT mediated improvements in depression, and early reductions in positive metacognitive beliefs mediated improvements in anxiety, thereby demonstrating that changes in metacognitive beliefs temporally preceded changes in emotional disorder symptoms. This study did not assess negative metacognitive beliefs. Findings from these cross-sectional, longitudinal, and treatment studies suggest that metacognitive beliefs are associated with RNT and may play a causal role in translating a temperamental vulnerability for emotional disorders into increased engagement in RNT, which, in turn, increases emotional symptoms. However, further prospective treatment studies investigating both positive and negative metacognitive beliefs are required to build the case for these causal relationships, particularly from a transdiagnostic perspective in mixed-diagnosis samples.

The main aim of this study was to examine whether changes in metacognitive beliefs during group MCT are associated with changes in emotional distress via changes in RNT in a mixed-diagnosis sample. Consistent with the S-REF model (Wells & Mathews, 1996), the first hypothesis was that positive and negative metacognitive beliefs would be positively associated with RNT and symptoms of emotional distress at pre-treatment, post-treatment, and follow-up. The second hypothesis was that negative and positive metacognitive beliefs would demonstrate significant indirect effects on emotional distress via RNT.

2.0 Method

2.1 Participants and Recruitment

Patients (N = 52) participated in a recent benchmarking study reporting outcomes from group MCT for individuals with primary and non-primary GAD (McEvoy, Erceg-Hurn, Anderson et al., 2015). Mean age was 38 years (SD = 14.3) and 60% (n = 31) were women. Most were employed (63%, n = 33), around half had a university qualification (52%, n = 27), and one-quarter had completed high school or less (25%, n = 13), and a similar proportion had a technical or trade certificate (23%, n = 12). Half were married or lived with their partner (52%, n = 27), with the remainder being single (40%, n = 21) or widowed/separated/divorced (8%, n = 4).

Patients were referred by general practitioners, psychiatrists, or clinical psychologists to an Australian specialist community mental health clinic for psychological treatment of anxiety disorders and/or depression. All patients completed a structured diagnostic interview (Mini International Diagnostic Interview; Lecrubier et al., 1997; Sheehan et al. 1997a, b, 1998) at their initial assessment and principal diagnoses were nominated based on the most debilitating disorder. Patients completed a battery of questionnaires prior to their initial assessment (see Measures section), which were reviewed by the assessing clinician and patient as part of a comprehensive assessment. A collaborative treatment plan was developed with patients being allocated to the MCT group if they met criteria for GAD. One exception was patients with principal social anxiety disorder (SAD), who were referred to a specialist SAD group program within the clinic. Patients were drawn from 11 groups with between 3 and 7 participants each.

GAD (77%, n = 40) and recurrent major depressive disorder (MDD, 17%, n = 9) were the most common principal disorders, with one patient (2%) each having principal agoraphobia, panic disorder with agoraphobia, and obsessive compulsive disorder. The

majority (63%) were diagnosed with more than one mental disorder. All patients with a principal diagnosis of MDD were given a secondary diagnosis of GAD, while a quarter of patients with a principal GAD diagnosis were given a secondary diagnosis of MDD. The most common other secondary or tertiary diagnosis was social phobia (n = 11), followed by recurrent MDD in remission (n = 4), hypochondriasis, simple phobia (n = 2 each), and agoraphobia, OCD, dysthymic disorder, and bulimia nervosa (n = 1 each). About two-thirds of patients were taking psychiatric medication (*N* = 35, 67%) for an extended period of time (median 1 year; interquartile range 6 months to 4 years) without responding adequately. More sociodemographic details are reported in McEvoy, Erceg-Hurn, Anderson et al. (2015).

2.2 Outcome Measures

2.2.1 Repetitive thinking questionnaire (RTQ-10; McEvoy et al., 2010; McEvoy, Thibodeau, & Asmundson, 2014). The RTQ is a transdiagnostic measure of RNT that was developed from the Penn State Worry Questionnaire (Meyers, Miller, Metzger, & Borkovec, 1990), Ruminative Response Scale (Nolen-Hoeksema & Morrow, 1991), and revised Post-Event Processing Questionnaire (McEvoy & Kingsep, 2006) by removing diagnosis-specific content such as references to worry and depression symptoms. Example items include “Once I start thinking about the situation, I cannot stop” and “I think about the situation all the time”. Items are answered on a 5-point scale qualified by not at all true (1), somewhat true (3), and very true (5). The 10-item trait version of the RTQ used in this study has a robust unidimensional structure, is able to distinguish between clinical and non-clinical populations, is highly sensitive to change and has high internal consistency (αs ≥ .90 at all time points in this study; McEvoy, Erceg-Hurn, Anderson et al., 2015; McEvoy et al., 2010; McEvoy et al., 2014; Newby et al., 2014). RTQ total scores can fall between 10 and 50.

2.2.2. Metacognitions questionnaire-30 (MCQ-30, Wells & Cartwright-Hatton, 2004). Positive and negative metacognitions were assessed using the positive (MCQ-POS)

and uncontrollability and dangerousness (MCQ-NEG) subscales from the MCQ-30. The MCQ-POS is a measure of positive beliefs about worry (e.g., “Worrying helps me cope”, “Worrying helps me to avoid problems in the future”), while the MCQ-NEG measures negative beliefs about the uncontrollability and dangerousness of worry (e.g., “When I start worrying I cannot stop”, “My worrying is dangerous for me”). Items are answered on a 4-point scale qualified by do not agree (1), agree slightly (2), agree moderately (3), and agree very much (4). Both scales comprise 6 items, and scores can range between 6 and 24. Cronbach’s alphas in this study were > .80 for both MCQ subscales across all time points, except for the MCQ-NEG at pretreatment ($\alpha = .73$).

2.2.3 Kessler psychological distress scale-10 (K10, Kessler et al., 2002). The K10 is a widely used general measure of psychological distress (e.g., Dear et al., 2011; Farchione et al., 2012; Newby et al., 2014). It comprises 10 items assessing common depressive and anxiety symptoms. Example items include “About how often did you feel tired out for no good reason?”, “About how often did you feel nervous?”, and “About how often did you feel hopeless?” Items are answered on a 5-point scale qualified by none of the time (1), a little of the time (2), some of the time (3), most of the time (4), and all of the time (5). Total scores can range between 10 and 50. Cronbach’s alphas for the K10 exceeded .89 at each time point in the current study.

2.3 Procedure & Treatment

Patients completed a questionnaire battery prior to their initial assessment session, at which the MINI was administered by a Clinical Psychologist experienced in both the assessment and treatment of emotional disorders. All assessing and treating clinicians had completed masters or doctorate degrees in clinical psychology, and all cases were presented at weekly supervision meetings where diagnoses, treatment plans, and patient progress were

discussed. All measures were completed prior to the first session, and following session 6 (post-treatment) and session 7 (follow-up).

The group MCT program (Anderson & Campbell, 2011) was modified from Wells (2009) and comprised six two-hour sessions (Session1-6) plus a one-month follow-up (Session 7). Treatment fidelity was supported by the use of a treatment manual that included detailed therapist notes, worksheets, and client handouts. All groups were co-facilitated by one (7 groups) or two senior therapists (1 group), or one senior therapist plus one trainee therapist (3 groups). All senior therapists had extensive experience providing psychological interventions in previous trials (McEvoy, Erceg-Hurn, Saulsman, & Thibodeau, 2015; McEvoy & Nathan, 2007; McEvoy, Nathan, Rapee, & Campbell, 2012). The study was approved by the Health Services' Human Research Ethics Committee (#QI 2014_04) and all patients provided informed written consent for their data to be used for quality improvement and research purposes.

Session 1 included psychoeducation about the nature of worry and rumination (RNT), including maintaining factors (i.e., negative beliefs about RNT, unhelpful behaviors aiming to stop RNT, attentional biases, and positive beliefs about RNT). Session 2 began with a homework review, whereas the remaining sessions began with an attention training exercise followed by a homework review, before discussing the new content and finally setting homework for the following week. Session 2 targeted uncontrollability metacognitions and introduced attention training skills. Attention training skills included exercises to sustain attention on present moment activities and mindfulness exercises. Session 3 focused on identifying and challenging dangerousness metacognitions and patients were taught questions to determine the veracity of the information they gathered with respect to their negative beliefs about RNT. Session 4 focused on challenging positive metacognitions. Sessions 2 to 4 included an evidence-testing exercise with the whole group, which involved identifying

evidence for and against the target metacognitive beliefs. Each session also involved behavioural experiments to test the beliefs. For instance, uncontrollability beliefs were challenged using a postponement experiment, whereby RNT was delayed to a scheduled 'worry time'. Dangerousness beliefs and positive beliefs were tested using 'worry up' and 'worry down' days, where patients observed whether their predictions were more, less, or equally likely when they worried more or less. Session 5 introduced active coping (structured problem-solving) as a technique for constructively managing solvable problems that required action, as an alternative to responses indicative of the Cognitive Affective Syndrome (e.g., avoidance, repeated checking). Session 6 involved a review of the key principles and the development of self-management plans for relapse prevention. The one-month follow-up session served the dual purposes of reviewing progress and self-management plans in an attempt to reduce relapse and collecting maintenance data. A more detailed description of the protocol is provided in McEvoy, Erceg-Hurn, Anderson et al. (2015).

2.4 Data Analyses

2.4.1 Bivariate associations. Cross-sectional relationships between metacognitions, RNT and symptoms were explored by creating scatterplots between each pair of variables at pre-treatment, post-treatment, and follow-up. Linear and non-linear regression lines were superimposed on the scatterplots, to inspect whether relationships between variables were linear or more complex. The strength of linear associations was quantified by computing Pearson correlation coefficients. Associations between change scores (pre- to post-treatment, and from pre-treatment to follow-up) for metacognitions, RNT and symptoms were examined using the same methods as for the cross-sectional data.

2.4.2 Indirect effects models. Four longitudinal models were used to test whether changes in metacognitions during treatment (the independent variable, IV) had an indirect

effect on changes in symptoms (the dependent variable, DV) via changes in RNT (the mediator). The only differences between the models were (i) in models 1 and 2 the IV was negative metacognitions whereas in models 3 and 4 the IV was positive metacognitions, and (ii) models 1 and 3 were fit using pre-to-post treatment change scores, whereas models 2 and 4 were fit using pre-treatment to follow up change scores.

The path between the IV and mediator (*a*) quantifies the number of units the mediator changes for each 1 unit increase in the IV. The *b* path quantifies the relationship between mediator and DV, controlling for the IV. Multiplying the *a* and *b* paths gives the *indirect (or 'mediated') effect* of metacognitions on symptoms via RNT. The c' path is the *direct effect* of metacognitions on symptoms, controlling for the indirect effect. The c' path represents the extent to which metacognitions cause symptom change through mechanisms other than RNT.

2.4.3 Significance and magnitude of indirect effects. Indirect effects models were tested using a generalization of the bootstrap approach popularized by Preacher and Hayes (2004; see also Hayes, 2013). Bootstrap tests of indirect effects use computer simulations to calculate a 95% confidence interval for the *ab* coefficient. An interval that *does not* include zero is interpreted as evidence of an indirect effect. There was a small amount of missing data ranging from $n = 1$ (2%) on the MCQ at pre-treatment to $n = 15$ (29%) on the K10 at follow up. The missing scores were handled using multiple imputation (MI), which is one of the gold standard methods for handling missing data recommended in expert statistical guidelines (e.g., National Research Council Panel on Handling Missing Data in Clinical Trials, 2010). In order to conduct bootstrap tests of indirect effects using the imputed data, we used the MI(BOOT) procedure described in Wu and Jia (2013).

The *ab* coefficient is an effect size, but it can be hard to interpret because it is unstandardized. Therefore, two additional standardized indices for quantifying the magnitude of indirect effects are recommended by Preacher and Kelley (2011) and Hayes (2013). The

first of these, ab_{cs}, is a completely standardized version of *ab* that measures what impact a 1 *SD* change in the IV has on the DV, via the mediator. For example, if ab_{cs} = .3, it would mean that a 1 *SD* reduction in metacognitions during treatment would cause symptoms to reduce by 0.3 *SD*, due to the indirect effect metacognitions have on symptoms via RNT.

The second standardized effect is kappa squared (κ^2), which is the ratio of the *observed* indirect effect (*ab*) to the *maximum possible* indirect effect ($ab_{\max}$). The maximum possible indirect effect can be determined using the standard deviations of the variables in the indirect effects model and their intercorrelations (see Preacher & Kelly, 2011). For example, the maximum possible value of *ab* for a certain indirect effects model may be 5 units and the observed value of *ab* might be 1. In this case, $\kappa^2 = ab / ab_{\max} = 1 / 5 = 0.2$. Preacher and Kelley suggested that κ^2 values of .01, .09 and .25 could be loosely interpreted as small, medium, and large effects. All analyses were conducted using the statistical software *R* version 3.1.1 (R Core Team, 2014).

3.0 Results

3.1 Cross-sectional associations

Scatterplots revealed that the relationships between the variables were linear. Cross-sectional correlations are displayed in Table 1. Consistent with the first hypothesis, negative metacognitions, RNT, and symptoms were strongly correlated at all time points. Contrary to the first hypothesis, relationships between positive metacognitions and the other constructs were weak and inconsistent. At pre-treatment, there were no associations between positive metacognitions and RNT, symptoms, or negative metacognitions, respectively. Positive metacognitions were significantly correlated with RNT and symptoms at post-treatment and follow up, but the size of the associations were numerically smaller than the magnitude of the relationships between negative metacognitions, RNT and symptoms at the same time points.

Positive metacognitions were only significantly associated with negative metacognitions at post-treatment.

Table 1.

Cross-sectional correlations between psychological distress (K10), repetitive negative thinking (RNT), positive metacognitions (PMC) and negative metacognitions (NMC) at pre-treatment, post-treatment, and follow-up

	K10	RNT	PMC
Pre-Treatment			
RNT	**.53**		
PMC	-.07	.00	
NMC	**.56**	**.67**	.10
Post-Treatment			
RNT	**.69**		
PMC	**.33**	**.32**	
NMC	**.56**	**.56**	**.35**
Follow-up			
RNT	**.70**		
PMC	**.35**	**.35**	
NMC	**.72**	**.60**	.21

Note. Bolded values are statistically significant ($p < .05$).

3.2 Change score associations

Correlations between change scores are displayed in Table 2. Changes in RNT during treatment were associated with changes in negative metacognitions and symptoms. Neither

positive nor negative metacognitions were associated with symptom change. The same pattern of results was observed for pre-treatment to follow up change scores.

Table 2

Longitudinal correlations between psychological distress (K10), repetitive negative thinking (RNT), positive metacognitions (PMC) and negative metacognitions (NMC)

	K10	RNT	PMC
Pre- to Post-treatment			
RNT	**.44**		
PMC	-.01	.19	
NMC	.24	**.47**	**.52**
Pre-treatment to Follow-up			
RNT	**.45**		
PMC	.00	.02	
NMC	.07	**.38**	**.54**

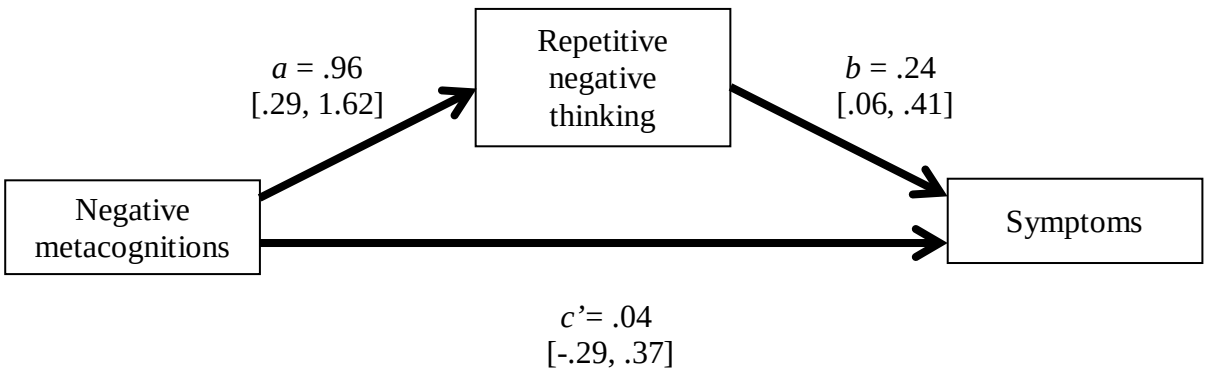
Note. Bolded values are statistically significant ($p < .05$).

3.3 Indirect Effects Models

3.3.1 Negative metacognition models. In Model 1 the IV was negative metacognitions and the model was fit using pre-to-post treatment change scores. The *a*, *b*, and *c'* path coefficients for this model are displayed in Figure 1. There was evidence that negative metacognitions had an indirect effect on symptoms via RNT, *ab* = .23, 95% CI [.05, .52]. The value of ab_{cs} was .20, indicating that a 1 *SD* reduction in negative metacognitions indirectly results in a 0.2 *SD* drop in symptoms, and $\kappa^2 = .19$, which according to Preacher and Kelley's (2011) guidelines is a relatively large effect. McEvoy, Erceg-Hurn, Anderson et

al. (2015) reported that, on average, patients scores fell during treatment by 6.94 points on the MCQ-NEG and 6.26 points on the K10. Approximately 25% (1.57 out of 6.26) of the change in symptoms during treatment can be attributed to the indirect effect of the negative metacognitions via RNT. The small and non-significant (p = .82) *c'* path suggests that negative metacognitions did not have a significant impact on symptom change through mechanisms other than RNT.

Figure 1. Simple mediational model for negative metacognitions (model 1)



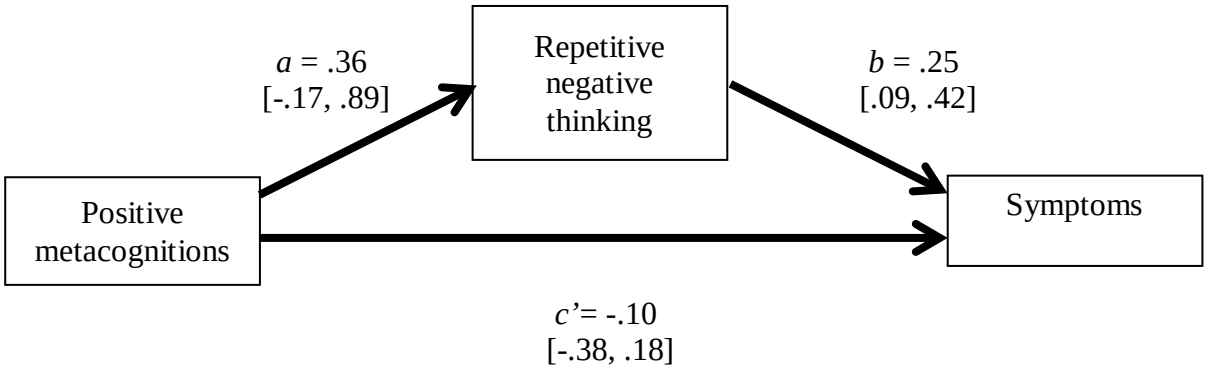
Note. Values in brackets are 95% confidence intervals for each coefficient calculated using heteroskedasticity-corrected standard errors.

The same pattern of results held for model 2 (using pre-treatment to follow up scores). The values of the *a*, *b* and *c'* coefficients were .66, .35 and -.15, and the size of the mediated effect was the same as for model 1 when rounded to the second decimal place, *ab* = .23, 95% CI [.05, .46]. The standardized effect sizes were also nearly identical, ab_{cs} = .19, and κ^2 = .19. Twenty six percent of the reduction in symptoms between pre-treatment and follow up could be attributed to the indirect effect.

3.3.2 Positive metacognition models. In Model 3 the IV was positive metacognitions and the model was fit using pre-to-post treatment change scores. The *a*, *b*, and *c'* path coefficients for this model are displayed in Figure 2. There was no evidence of an indirect

effect. The coefficient for the indirect effect was less than half that of model 1, *ab* = .09, 95% CI [-.03, .25], and the confidence interval included zero. The standardized effect sizes were also much smaller than for model 1 (ab_{cs} = .08, and κ^2 = .09) and only 4% of change in K10 symptoms during treatment could be attributed to the indirect effect. For model 4 (using pre-treatment to follow up change scores) there was also no evidence of an indirect effect, with the effect sizes all being close to zero, *ab* = .01, 95% CI [-.15, .19], ab_{cs} < .01, and κ^2 = .02. The values of the path coefficients were *a* = .02, *b* = .32 and *c'* = -.01.

Figure 2. Simple mediational model for positive metacognitions (model 3)

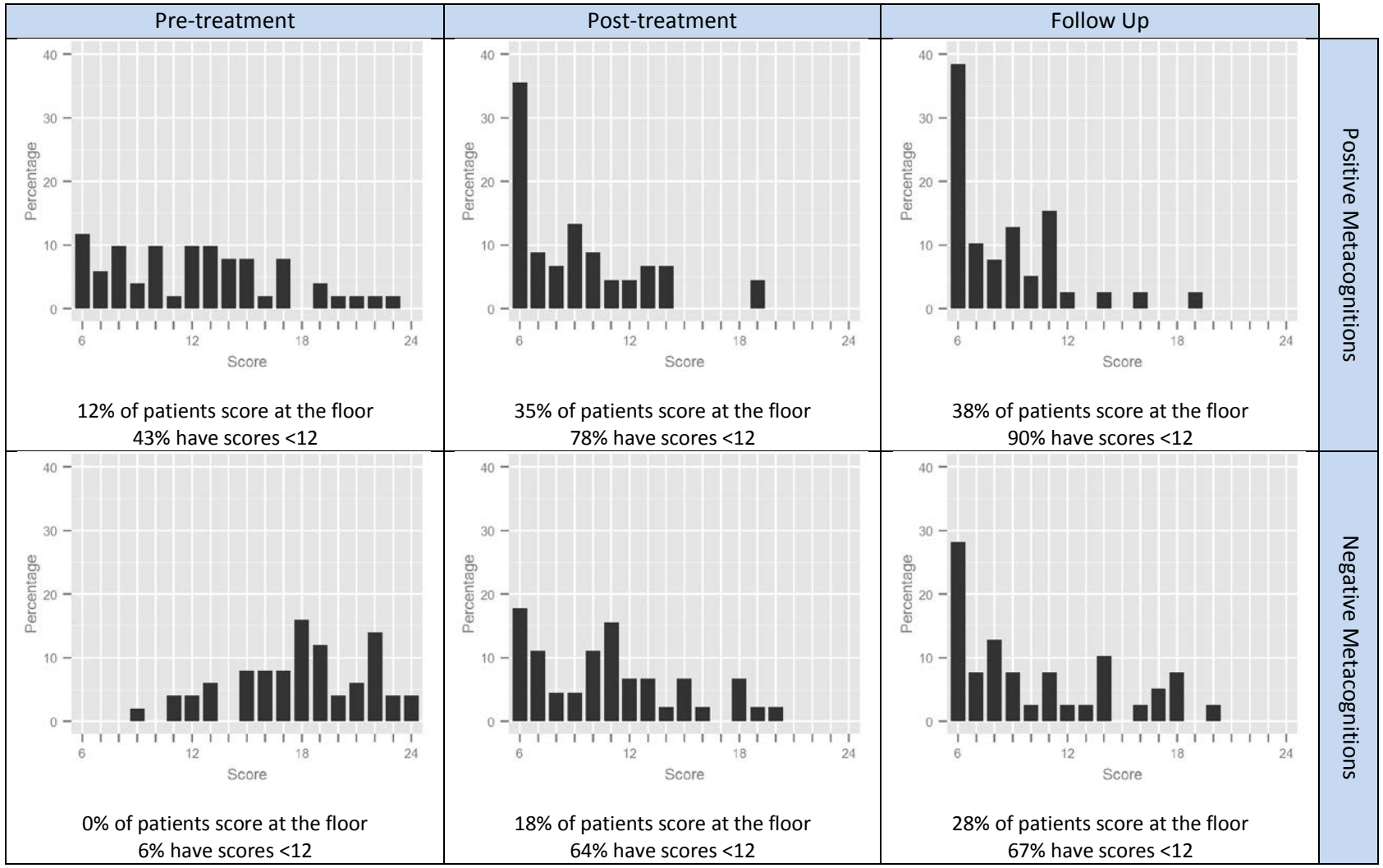


Note. Values in brackets are 95% confidence intervals for each coefficient calculated using heteroskedasticity-corrected (HC4) standard errors.

3.4. Floor Effects

There was evidence of floor effects for both metacognition measures, which was particularly pronounced for positive metacognitions (see Figure 3). Nearly half the sample had low scores (≤ 25th percentile) on the MCQ-POS at pre-treatment, and large proportion of patients had low scores at post-treatment and follow up. For the negative metacognitions subscale there were no floor effects apparent at pre-treatment, but there were problems at both post-treatment and follow up. There were no floor effects apparent for the RTQ and

Figure 3. Distributions of positive and negative metacognition scores illustrating floor effects



K10, with scores being distributed over a range of scale points and away from the floor at pre-treatment, post-treatment, and follow up.

4.0 Discussion

The main aim of this study was to investigate mechanisms of change during group metacognitive therapy for repetitive negative thinking (RNT) in a sample with primary and non-primary generalized anxiety disorder. The first hypothesis, that positive and negative metacognitive beliefs would be significantly and positively associated with RNT and psychological distress at pre-treatment, post-treatment, and follow-up, was partially supported. Consistent with metacognitive theory, negative metacognitive beliefs were positively associated with RNT and distress at each time point. Positive metacognitive beliefs were associated with RNT and distress at post-treatment and follow-up, but not at pre-treatment. The second hypothesis, that positive and negative metacognitions would have an indirect effect on psychological distress via RNT, was also partially supported. A significant indirect effect was found for negative metacognitive beliefs but not positive metacognitive beliefs. These findings suggest that reductions in negative metacognitive beliefs were associated with reductions in RNT which, in turn, were associated with reductions in distress. This was not the case for positive metacognitions.

The lack of association between positive metacognitive beliefs, RNT, and emotional distress is inconsistent with metacognitive theory. The floor effect and resulting limited range of scores on the positive metacognitive beliefs subscale, compared to the other measures, may have attenuated its associations with RNT and distress. Attenuation of these associations would have limited our ability to detect direct and indirect effects of positive metacognitive beliefs on RNT and distress.

One explanation for the relatively low average endorsement of positive beliefs, and lack of cross-sectional associations with other measures at pre-treatment, is that patients may

have poorer insight into their positive beliefs prior to treatment. Patients may instead be more preoccupied with the negative consequences of their RNT that brought them into treatment and thus find it difficult to acknowledge any perceived benefits (McEvoy, Erceg-Hurn, Anderson et al., 2015). It is noteworthy that the measures were administered prior to the first session, at which patients are socialized to the formulation and metacognitive beliefs are Socratically elicited. Future research should investigate whether administering measures of positive metacognitive beliefs more frequently will capture increases in insight (and thus greater endorsement) after the model is presented in session 1 and throughout therapy. For example, once uncontrollability and dangerousness metacognitions have been successfully challenged and modified it may become clearer to patients that they are opting to engage in excessive RNT on some occasions due to residual positive metacognitions.

Wells (2013) argues that whilst positive metacognitive beliefs are commonly held to some degree by most individuals, negative metacognitive beliefs "…are the most pervasive and powerful influences in psychological disorder…(p. 188)". Thus, another explanation for the floor effect may be that whilst negative beliefs are ubiquitous and invariably pathological in individuals with elevated RNT, positive beliefs may be genuinely held at a problematic level by only a subset of these individuals. Consistent with this possibility, there was a strong correlation between pre-treatment positive metacognitions subscale scores and change scores (e.g., $r = .78$ between pre-treatment positive beliefs and pre-treatment to follow-up change scores, compared to rs $\leq .32$ across all other measures at the same time points). Therefore those who endorsed positive beliefs more strongly at pre-treatment changed more on this measure to follow-up, whereas change on other measures were less strongly associated with degree of endorsement at pre-treatment. Modules targeting positive metacognitions may only be required for those who endorse these beliefs at pre- or post-treatment, rather than being a standard component of MCT.

Yet another explanation for the floor effect on the positive metacognitions scale is that key positive beliefs have been omitted from the relatively brief MCQ-30 subscale. The positive beliefs subscale from the original MCQ (Cartwright-Hatton & Wells, 1997) contains 19 items, so may also provide a more comprehensive and sensitive assessment than the short version used in this study. However, previous cross-sectional studies using the longer positive beliefs subscale have also found relatively weak associations with RNT and symptoms (van der Heiden et al., 2010). Other research using versions of the Positive Beliefs about Rumination Scale (PBRS, Papageorgiou & Wells, 2001; Watkins & Moulds, 2005) has found significant and stronger associations between positive metacognitive beliefs and RNT (Papageorgiou & Wells, 2003; Roelofs et al., 2010; Weber and Exner, 2013), and that early changes in positive beliefs are associated with subsequent reductions in RNT (Newby et al., 2014). It is noteworthy that there is little overlap in content between the MCQ-30 positive beliefs subscale and the PBRS, suggesting that the item content could potentially explain discrepancies in findings between studies using the alternative measures.

Interestingly, changes in positive and negative metacognitive beliefs were not directly associated with changes in emotional distress. One possible explanation for this finding is that the focus of the program is on modifying RNT by challenging metacognitive beliefs, rather than immediate emotional symptom reduction. McEvoy, Erceg-Hurn, Anderson et al. (2015) reported very large effect sizes on measures of RNT and negative metacognitive beliefs during the program (Cohen's *d*s ~ 2.0), suggesting that these cognitive processes were effectively modified. Effect sizes on measures of anxiety, depression, and psychological distress were smaller, although still moderate to large (*d*s = .66 for anxiety, .98 for depression, and 1.02 for psychological distress). Additional strategies may be required if the goal is to more rapidly reduce affective symptoms of emotional disorder. Relatedly, the intervention was relatively brief compared to other trials of MCT and, although this did not

adversely impact the magnitude of change of the key cognitive mechanisms, affective symptoms may reduce more slowly. Most patients reported chronic anxiety and depression, so it may be unrealistic to expect these symptoms to resolve further over a 6-week intervention and 1-month follow-up. Longer-term follow-up would be useful to evaluate whether the treatment effects on symptoms endured or even improved over time.

This study was one of the first treatment studies to examine indirect effects of both positive and negative metacognitive beliefs on symptoms via RNT, and therefore the findings have important theoretical implications. Although our study is correlational so cannot establish causality, the major theoretical contribution is that our findings are consistent with the S-REF model's (Wells & Matthews, 1996) contention that RNT is one important pathway through which modifying negative metacognitive beliefs can reduce emotional disorder. In fact, the lack of a direct association between negative metacognitive beliefs and emotional distress in this study suggests that RNT may be the *only* pathway through which changing metacognitive beliefs influenced emotional distress. However, it is important to note that several components of the S-REF model (Wells & Mathews, 1996) and processes targeted by the MCT program were not assessed in this study. For instance, the S-REF model suggests that inflexible self-focused attention and counterproductive cognitive (e.g., thought suppression) and behavioural (e.g., alcohol use) coping strategies also contribute to RNT and emotional distress. If these factors were assessed they may have contributed additional explanatory power to symptom reduction.

Findings from this study need to be considered within the context of several limitations. First, larger sample sizes would enable more rigorous evaluations of multiple mediational pathways whilst correcting for measurement error (e.g., structural equation modelling). Second, administering the measures on multiple occasions during therapy would enable a more nuanced assessment of the temporal precedence of changes in metacognitive

beliefs relative to changes in RNT and emotional disorder symptoms (see Newby et al., 2014). Multiple assessment points would also shed light on whether patients develop greater awareness of existing metacognitive beliefs during therapy, and at which points they are successfully modified. Third, future research should assess additional elements of the Cognitive Affective Syndrome so that the S-REF model (Wells & Mathews, 1996) can be more comprehensively evaluated. Fourth, the absence of a control group means that we cannot definitively rule out the possibility that the reductions in RNT and symptoms were the consequence of the passage of time, or other non-specific factors. However, McEvoy, Erceg-Hurn, and Anderson et al. (2015) demonstrated that this program achieved similar effect sizes to previous controlled trials of MCT and that waitlist groups remain virtually unchanged on measures of RNT. Finally, although the intervention was associated with medium to large improvements across a large array of outcome measures including negative metacognitive beliefs, RNT, and distress (McEvoy et al., 2015), the lack of data on protocol adherence introduces a potential threat to internal validity.

This study was one of the first treatment studies to assess the influence of both positive and negative metacognitive beliefs on emotional distress via RNT. The findings suggest that modifying negative metacognitive beliefs is a particularly effective strategy for reducing engagement in the pernicious process of RNT which, in turn, is associated with a reduction in emotional distress. The influence of positive metacognitive beliefs appears to be less critical to treatment outcome, although further research is required to determine whether psychometric issues obscured this relationship.

Acknowledgements

The authors wish to gratefully acknowledge Amanda Swan, Lisa Saulsman, and Mark Summers for facilitating groups.

References

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

Anderson, R.A., Campbell, B.N.C. 2011. Working with worry and rumination: a cognitive behavioural group treatment programme for repetitive negative thinking. Perth: Centre for Clinical Interventions. ISBN: 0975799584.

Bailey, R., Wells, A. 2014. Metacognitive therapy in the treatment of hypochondriasis: a systematic case series. Cog. Ther. Res. 38, 541-550.

Borkovec, T.D., Robinson, E., Pruzinsky, T., DePree, J.A., 1983. Preliminary exploration of worry: some characteristics and processes. Behav. Res. Ther. 21, 9–16.

Brown, T.A., Campbell, L.A., Lehman, C.L., Grisham, J.R., Mancill, R.B., 2001. Current and lifetime comorbidity of the DSM-IV anxiety and mood disorders in a large clinical sample. J. Abnorm. Psychol. 110, 585–599.

Cartwright-Hatton, S. Wells. A. 1997. Beliefs about worry and intrusions: the Meta-Cognitions Questionnaire and its correlates. J. Anx. Dis. 11, 279-296.

Dammen, T., Papageorgiou, C., Wells. A. 2014. An open trial of group metacognitive therapy for depression in Norway. Nord. J. Psychiatry. 69, 126-131. DOI: 10.3109/08039488.2014.936502

Dear, B.F., Titov, N., Schwencke, G., Andrews, G., Johnston, L., Craske, M.G., & McEvoy, P. 2011. An open trial of a brief transdiagnostic internet treatment for anxiety and depression. Behav. Res. Ther. 49, 830-837. doi: 10.1016/j.brat.2011.09.007

Ehring, T., Watkins, E.R. 2008. Repetitive negative thinking as a transdiagnostic process. Int. J. Cog. Therapy. 1, 192-205.

Farchione, T.J., Fairholme, C.P., Ellard, K.K., Boisseau, C.L., Thompson-Hollands, J., Carl, J.R., Gallagher, M.W., Barlow, D.H. 2012. Unified protocol for transdiagnostic treatment of emotional disorders: A randomized controlled trial. Behav. Ther., 43, 666-678.

Harvey, A., Watkins, E., Mansell, W., Shafran, R. 2004. Cognitive behavioural processes across psychological disorders: a transdiagnostic approach to research and treatment. Oxford: Oxford University Press.

Hayes, A. F. 2013. Introduction to mediation, moderation, and conditional process analysis. New York: The Guilford Press.

Kessler, R. C., Andrews, G., Colpe, L.J., Hiripi, E., Mroczek, D.K., Normand, S.-L.T., et al. 2002. Short screening scales to monitor population prevalences and trends in non-specific psychological distress. Psychol. Med. 32, 959-976.

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. Arch. Gen. Psychiatry, 62, 617-627.

Lecrubier, Y., Sheehan, D., Weiller, E., Amorim, P., Bonora, I., Harnett-Sheehan, K., et al. 1997. The Mini International Neuropsychiatric Interview (MINI). A short diagnostic structured interview: Reliability and validity according to the CIDI. Euro. Psychiatry, 12, 224–231.

Matthews, G. 2015. Advancing the theory and practice of metacognitive therapy: a commentary on the special issue. Cogn. Ther. Res. 39, 81-87.

McEvoy, P.M., Erceg-Hurn, D.M., Anderson, R.A., Campbell, B.N.C., Swan, A., Saulsman, L.M., Summers, M., Nathan, P.R. 2015. Group metacognitive therapy for repetitive negative thinking in primary and non-primary generalized anxiety disorder: an effectiveness trial. J. Affect. Disord. 175, 124-132. DOI: 10.1016/j.jad.2014.12.046.

McEvoy, P.M., Erceg-Hurn, D.M., Saulsman, L.M., Thibodeau, M.A. 2015. Imagery enhancements increase the effectiveness of cognitive behavioural group therapy for social anxiety disorder: a benchmarking study. Behav. Res. Ther. 65, 42-51. DOI: 10.1016/j.brat.2014.12.011

McEvoy, P.M., Grove, R., Slade, T. 2011. Epidemiology of anxiety disorders in the Australian general population: findings of the 2007 Australian National Survey of Mental Health and Wellbeing. Aust. New Zealand J. Psychiatry, 45, 957-967.

McEvoy, P. M., Kingsep, P. 2006. The post-event processing questionnaire in a clinical sample with social phobia. Behav. Res. Ther. 44, 1689-1697. doi: http://dx.doi.org/10.1016/j.brat.2005.12.005

McEvoy, P.M., Mahoney, A.E.J. 2013. Intolerance of uncertainty and negative metacognitive beliefs as transdiagnostic mediators of repetitive negative thinking in a clinical sample with anxiety disorders. J. Anx. Dis. 27, 216-224. DOI:10.1016/j.janxdis.2013.01.006

McEvoy, P.M., Mahoney, A.E.J., Moulds, M.L. 2010. Are worry, rumination, and post-event processing one and the same? Development of the repetitive thinking questionnaire. J. Anx. Dis. 24, 509-519.

McEvoy, P.M., Nathan, P. 2007. Effectiveness of Cognitive-Behavioral Therapy for Diagnostically Heterogeneous Groups: A Benchmarking Study. J. Consult. Clin. Psychol. 75, 344-350.

McEvoy, P.M., Nathan, P., Rapee, R.M., Campbell, B.N.C. 2012. Cognitive behavioural group therapy for social phobia: Evidence of transportability to community clinics. Behav. Res. Ther. 50, 258-265.

McEvoy, P.M., Thibodeau, M., Asmundson, G.J.G. 2014. Trait repetitive negative thinking: A brief transdiagnostic assessment. J. Exp. Psychopath., 3, 382-398. DOI:10.5127/jep.037813

McEvoy, P.M., Watson, H., Watkins, E., Nathan, P. 2013. The relationship between worry, rumination, and comorbidity: Evidence for repetitive negative thinking as a transdiagnostic construct. J. Affect. Dis. 151, 313-320. DOI: 10.1016/j.jad.2013.06.014i

McLaughlin, K.A., Nolen-Hoeksema, S. 2011. Rumination as a transdiagnostic factor in depression and anxiety. Beh. Res. Ther. 49, 186-193.

Meyers, T., Miller, M., Metzger, R., Borkovec, T. 1990. Development and validation of the Penn State Worry Questionnaire. Behav. Res. Ther. 28, 487-495.

National Research Council Panel on Handling Missing Data in Clinical Trials. 2010. The Prevention and Treatment of Missing Data in Clinical Trials. Washington, DC: The National Academies Press.

Newby, J.M., Williams, A.D., Andrews, G. 2014. Reductions in negative repetitive thinking and metacognitive beliefs during transdiagnostic internet cognitive behaviour therapy (iCBT) for mixed anxiety and depression. Behav. Res. Ther. 59, 52-60.

Nolen-Hoeksema, S. 2000. The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. J. Abnorm. Psychol. 109, 504-511. doi:10.1037/0021843X.109.3.504.

Nolen-Hoeksema, S., 1991. Responses to depression and their effects on the duration of depressive episodes. J. Abnorm. Psychol. 100, 569–582.

Papageorgiou, C., Wells, A. 2001. Positive beliefs about depressive rumination: development and preliminary validation of a self-report scale. Beh. Ther. 32, 13–26. doi:10.1016/S0005-7894(01)80041-1.

Papageorgiou, C., Wells, A. 2003. An empirical test of a clinical metacognitive model of rumination and depression. Cog. Ther. Res, 27, 261-273.

Papageorgiou, C., Wells, A. 2015. Group metacognitive therapy for severe antidepressant and CBT resistant depression: a baseline-controlled trial. Cogn. Ther. Res. 39, 14-22. DOI 10.1007/s10608-014-9632-x.

Park, R.J., Goodyer, I.M., Teasdale, J.D. 2004. Effects of induced rumination and distraction on mood and overgeneral autobiographical memory in adolescent Major Depressive Disorder and controls. J. Child. Psychol. Psychiatry. Allied Disc. 45, 996-1006.

Preacher, K., Hayes, A. 2004. SPSS and SAS procedures for estimating indirect effects in simple mediation models. Behav. Res. Meth. 36, 717-731.

Preacher, K.J., Kelley, K. 2011. Effect size measures for mediation models: Quantitative strategies for communicating indirect effects. Psychol. Meth., 16, 93-115.

R Core Team. 2013. R: A language and environment for statistical computing. Vienna, Australia: R Foundation for Statistical Computing.

Rees, C.S., van Koesveld, K.E. 2008. An open trial of group metacognitive therapy for obsessive-compulsive disorder. J. Behav. Ther. Exp. Psychiatry, 39, 451-458.

Roelofs, J., Huibers, M., Peeters, F., Arntz, A., van Os, J. 2010. Positive and negative beliefs about depressive rumination: a psychometric evaluation of two self-report scales and a test of a clinical metacognitive model of rumination and depression. Cog. Ther. Res. 34, 196-205.

Sheehan, D.V., Lecrubier, Y., Sheehan, K.H., Amorim, P., Janavs, J., Weiller, E., et al. 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. J. Clin. Psychiatry, 59(suppl. 20), 22–33.

Sheehan, D.V., Lecrubier, Y., Sheehan, K.H., Janavs, J., Weiller, E., Keskiner, A., et al. 1997a. The Mini InternationalNeuropsychiatric Interview (MINI). A short diagnostic structured interview: Reliability and validity according to the CIDI. Europ. Psychiatry, 12, 224–231.

Sheehan, D.V., Lecrubier, Y., Sheehan, K.H., Janavs, J., Weiller, E., Keskiner, A., et al. 1997b. The validity of the Mini International Neuropsychiatric Interview (MINI) according to the SCID-P and its reliability. Europ. Psychiatry, 12, 232–241.

van der Heiden, C., Melchior, K. 2014. A 30-month follow-up of generalized anxiety disorder: status after metacognitive therapy and intolerance of uncertainty therapy. Europ. J. Pers. Cent. Healthcare, 2, 434-438.

van der Heiden, C., Melchior, K., Muris, P., Bouwmeester, S., Bos, A.E.R., van der Molen, H.T. 2010. A hierarchical model for the relationships between general and specific vulnerability factors and symptom levels of generalized anxiety disorder. J. Anx. Dis, 24, 284-289.

van der Heiden, C., Melchior, K., de Stigter, E. 2013. The effectiveness of group metacognitive therapy for generalised anxiety disorder: A pilot study. J. Contemp. Psychotherapy, 43, 151-157.

van der Heiden, C., Muris, P., van der Molen, H.T. 2012. Randomized controlled trial on the effectiveness of metacognitive therapy and intolerance-of-uncertainty therapy for generalized anxiety disorder. Behav. Res. and Ther. 50, 100-109.

Watkins, E. 2008. Constructive and unconstructive repetitive thought. Psych. Bull. 134, 163-206.

Watkins, E., Moulds, E. 2005. Positive beliefs about rumination in depression: a replication and extension. Person. Indiv. Diff. 39, 73-82.

Watkins, E., Moulds, M., Mackintosh, B. 2005. Comparisons between rumination and worry in a non-clinical population. Behav. Res. Ther. 43, 1577–1585.

Weber, F., Exner, C. 2013. Metacognitive beliefs and rumination: a longitudinal study. Cog. Ther. Res. 37, 1257-1261.

Wells, A. 2004. A cognitive model of GAD: Metacognitions and pathological worry. In. R. G. Heimberg, C. L. Turk, and D. S. Mennin's (Eds), Generalized Anxiety Disorder: Advances in Research and Practice (pp. 164-186). New York: The Guilford Press.

Wells, A. 2009. Metacognitive therapy for anxiety and depression. New York: Guildford Press.

Wells, A. 2013. Advances in Metacognitive Therapy. Int. J. Cog. Ther. 6, 186-201.

Wells, A. Carter, K. 2001. Further tests of a cognitive model of generalize anxiety disorder: metacognitions and worry in GAD, panic disorder, social phobia, depression, and nonpatients. Beh. Ther. 32, 85-102.

Wells, A., Cartwright-Hatton, S. 2004. A short form of the metacognitions questionnaire: properties of the MCQ-30. Behav. Res. Ther. 42, 385-396.

Wells, A., Fisher, P., Myers, S., Wheatley, J., Patel, T., Brewin, C. 2012. Metacognitive therapy in recurrent and persistent depression: A platform trial. Behav. Res. Ther. 50, 367-373.

Wells, A., Matthews, G. 1996. Modelling cognition in emotional disorder: the S-REF model. Behav. Res. Ther. 34, 881–888.

Wu, W. Jia, F. 2013. A new procedure to test mediation with missing data through nonparametric bootstrapping and multiple impulation. Multivar. Behav. Res. 48, 663-691.