



# Mechanisms of change in metacognitive and cognitive behavioral therapy for treatment-resistant anxiety: The role of metacognitive beliefs and coping strategies

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## Abstract

Metacognitive therapy (MCT) has shown promising outcomes across disorders, but, currently, little is known about the mechanisms of change in MCT as well as their specificity compared to those of cognitive behavioral therapy (CBT). The main purpose of this study was to examine the within-person relationships between features of the cognitive attentional syndrome (CAS)—the thinking style supposed to maintain clinical disorders according to the MCT model—and anxiety over the course of MCT and CBT for comorbid anxiety disorders. Seventy-four inpatients had been randomized to either MCT or CBT and actually started treatment. CAS features and anxiety were assessed weekly during treatment. These measures were disaggregated to their within- and between-person components and used as predictors in mixed models. All CAS features—coping activities, negative metacognitive beliefs, and positive metacognitive beliefs—decreased over the course of treatment. Negative and positive beliefs decreased more in MCT than in CBT. Time-specific changes in positive metacognitive beliefs predicted variations in subsequent anxiety across the two treatments (within-person effect). The finding of a within-person relationship between positive metacognitive beliefs and subsequent anxiety has the clinical implication that reduction in these beliefs may be important for treatment response.

## Keywords

Cognitive attentional syndrome, cognitive behavioral therapy, comorbid anxiety disorders, metacognitive therapy, within-person mechanism outcome relationships

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## Introduction

Metacognitive therapy (MCT) has shown promising outcomes across disorders including generalized anxiety disorder (van der Heiden, Muris, & van der Molen, 2012), major depressive disorder (Wells et al., 2012), and posttraumatic stress disorder (PTSD; Wells, Walton, Lovell, & Proctor, 2015). A recently conducted meta-analysis indicated that MCT was superior to both wait-list and cognitive behavioral therapy (CBT) for anxiety and depression (Normann, van Emmerik, & Morina, 2014). However, currently, little is known about the mechanisms of change in MCT as well as their specificity compared to those of CBT. This is of particular importance as identification of mechanisms may help to refine treatment.

MCT is based on the self-regulatory executive function model (S-REF; Wells and Matthews, 1996), which offers an account of first-order cognitive and metacognitive factors involved in the maintenance of emotional disorder. The model locates processing within a three-level cognitive architecture: a low level involving automatic and reflexive processing; an intermediate level involving strategic, capacity limited processing; and a high level consisting of knowledge or beliefs that are metacognitive in nature and stored in long-term memory. A core principle of the self-regulatory executive function (S-REF) model is that psychological disorder is linked to the activation of a particular maladaptive style of thinking called the cognitive attentional syndrome (CAS). The CAS consists of cognitive perseveration, a thinking style that takes the form of worry or rumination, attentional focusing on threat, and unhelpful behaviors that backfire (e.g., thought suppression and situational avoidance). The CAS is conceptualized as arising from metacognitive knowledge and beliefs. Two categories of beliefs are important: (1) positive beliefs about the need to engage in aspects of the CAS (e.g., “Worrying helps me cope”) and negative beliefs about the uncontrollability, dangerousness, or importance of thoughts and feelings (e.g., “Worrying too much could harm me”).

Consistent with CBT approaches, the content of beliefs and thoughts is considered to determine the type of disorder experienced. Thoughts about danger lead to anxiety, and self-devaluative thoughts give rise to depression. However, MCT posits that this content does not cause disorder because most people have thoughts like this and for most emotion is transitory. Emotional disorder is caused by the CAS that

is activated in response to negative thoughts, that is, by sustained negative processing which is under the influence of metacognition. Thus, MCT focuses on the thought processes and underlying metacognitive beliefs but does not address the content of symptom-related thoughts. While more recently CBT addresses thought processes such as thought suppression and rumination (Ehlers & Wild, 2015) and selective attention (Rapee, Gaston, & Abbott, 2009), it remains different from MCT, as CBT emphasizes the content of symptom-related thoughts and metacognitive beliefs are seldom addressed.

The CAS (and metacognitive beliefs) can be described as a common pathway to most psychological disorders, with specific features of CAS being related to specific disorders. Thus, the S-REF model forms the basis for a trans-diagnostic MCT. In this generic version of MCT (Wells, 2009, p. 250), challenging metacognitive beliefs, driving the CAS, and removing the CAS activities (worry, rumination, threat monitoring, maladaptive coping behavior) are central elements. Techniques such as detached mindfulness (DM) and worry/rumination postponement are used to reduce the amount of CAS activity, to enhance executive control, and to modify metacognitive beliefs.

Few studies have examined the role of CAS and metacognitions in therapy. Solem, Håland, Vogel, Hansen, and Wells (2009) found that change in metacognition in exposure and response prevention treatment for obsessive-compulsive disorder (OCD) predicted outcome over and above change in OCD-related first-order cognitions. However, this finding was based on between-subject differences in outcomes and does not capture covariation in outcome at both within- and between-subject levels. This is important because therapy theories (and therapists) primarily focus on within-person relationships, that is, how change in a mechanism variable influences change in outcome within the same patient (Curran & Bauer, 2011). Only under very strict statistical conditions can within-person relationships be inferred from between-person findings (Molenaar, 2004). Usually, they are different. In an earlier randomized controlled trial (Johnson, Hoffart, Nordahl, & Wampold, 2017), we measured anxiety-related cognitions, metacognitions, and anxiety repeatedly during trans-diagnostic MCT and disorder-specific CBT for comorbid anxiety disorders (Johnson et al., 2018). Using repeated measurements gave us the opportunity to disaggregate the within-person components of the

scores from the between-person components and thus to study the therapy-relevant within-person relationships between the mechanism variables and anxiety. By focusing on within-person variability, we could also rule out any stable patient characteristics as a rival explanation for the process–outcome relationships (Falkenström, Finkel, Sandell, Rubel, & Holmqvist, 2017). We found that time-specific changes both in metacognitions and anxiety-related cognitions predicted subsequent changes in anxiety 4 days later.

In this study, we wished to extend our previous study by focusing on both the CAS and associated metacognitions and to explore patterns of covariation with symptoms over a longer time lag, that is, 1 week. In addition, we aimed to deconstruct the CAS and examine the contribution of its constituent parts to changes in symptoms. With this set of aims in mind, we tested a series of specific hypotheses:

1. Over the course of therapy, features of the CAS will decrease more in MCT than in CBT.
2. Time-specific changes in features of a patient's CAS over the course of therapy will be positively related to subsequent change in that patient's anxiety symptoms assessed a week later (within-person effect). That is, when a CAS feature for a given patient is higher than is expected for that patient, subsequent anxiety will be higher.

We also wanted to explore whether there were reversed relationships between anxiety and the CAS features, whether treatment condition moderated the within-person relationships between the CAS features and anxiety and whether the between-person component of a CAS feature moderated the within-person relationships between that feature and anxiety (cross-level interaction).

## Materials and methods

### Participants

Participants were referred for treatment to the Department of Anxiety Disorders at Modum Bad Psychiatric Center in Norway. Modum Bad is a specialist in National hospital running an inpatient program for treatment-resistant patients with anxiety disorders from the entire Norwegian population. Recruitment was designed to be liberal using the clinical criteria for treatment used at the department. The Anxiety

Disorders Interview Schedule (IV) (Brown, Di Nardo, & Barlow, 1994) was used to diagnose patients. Participants had to meet criteria for a principal DSM-IV disorder, equal to or greater than 4 on the clinical severity rating, of PTSD, social anxiety disorder (SAD), or panic disorder with or without agoraphobia (PD/A). In addition, participants had to have failed at least one structured psychological treatment, be 18 years or older, able to speak Norwegian, and provide informed consent. Following the intake procedures at the department, patients were excluded if (a) they would have required immediate or simultaneous treatment that could interact with the treatment in unknown ways or (b) had current DSM-IV diagnosis of organic mental disorders, current suicidal risk, or current substance abuse. All participants had to terminate the use of psychotropic medications before treatment. All participants provided informed consent and the study was approved by the Norwegian regional ethical committee (2013/209/REK South-East).

Ninety patients were included in the trial and randomized to treatment stratified by principal disorder. The 74 patients ( $n = 38$  in CBT,  $n = 36$  in MCT) who actually started treatment were included in the present study. These patients—45 women and 29 men—had a mean age of 42.0 years ( $SD = 12.8$ ) and a mean duration of anxiety disorder of 16.1 years ( $SD = 11.8$ ). They had on average 3.7 diagnoses at the start of treatment, 40 had PTSD, 51 had PD/A, and 45 had SAD. Only four (5%) participants were working full time when entering therapy. Seven of the 74 patients did not complete the treatment program, leaving 67 who completed all treatment sessions ( $n = 33$  in CBT,  $n = 34$  in MCT). For more information on the participants, see Johnson, Hoffart, Nordahl, and Wampold (2017).

### Treatments

The treatments lasted 8 weeks. The mean number of individual sessions for completers was 9.4 ( $SD = 1.7$ ) and the number of sessions was equal in the two conditions. However, the individual sessions in CBT for SAD and PTSD lasted longer—90 min—due to the protocols. Mean session time was 71.5 min ( $SD = 18.8$ ) in CBT and 51.8 min ( $SD = 13.3$ ) in MCT. The patients received treatment in a ward with other anxiety patients and participated in the ward's common activity, consisting of one ward meeting and one physical exercise session per week.

### **Random assignment**

Following diagnostic assessment at evaluation, participants were randomly assigned using a randomization sequence generated by <http://www.random.org>. Patients were stratified on primary diagnosis (i.e., PD/A, SAD, or PTSD) and randomly assigned to either MCT or CBT.

### **Metacognitive therapy**

MCT followed the manualized treatment protocol of Wells (2009). This trans-diagnostic protocol deemphasizes diagnostic labels and focuses instead on challenging positive and negative metacognitions that drive the use of worry, rumination, self-focused attention, and coping behaviors to regulate emotions. Session 1 began with generation of the generic case formulation, thought-suppression experiment, and practice of DM. Homework involved applying DM and postponing worry and rumination experiments. In Session 2, the focus was continued socialization to the treatment model and the use of metaphors and verbal reattribution to weaken negative metacognitions about uncontrollability of worry/rumination. Postponement of worry and rumination was given as homework. In Session 3, remaining beliefs concerning negative metacognitions about danger and control were the main theme. Postponement of worry and rumination was given as homework as well as banning unhelpful coping strategies. Sessions 4–7 emphasized the remaining elements of the CAS, mainly threat monitoring and unhelpful coping behaviors. Remaining positive metacognitions regarding the use of worry and rumination were challenged. Homework was related to implementing a new thinking style. In the two last sessions (7 and 8), the old and the new plan for handling negative thought triggers was developed as well as repeated implementation of the new thinking style.

### **Cognitive behavioral therapy**

Treatments in the diagnosis-specific CBT condition were different depending on the patient's primary diagnosis. In this trial, CBT therapists used Clark's (1986) panic disorder model for the treatment of PD/A, Clark and Wells' (1995) model for social phobia, and Foa, Hembree, and Rothbaum's (2007) prolonged exposure treatment for PTSD. The protocols were chosen because they are widely used and well-documented. For PD, Session 1 consisted of mapping out a panic circle for a recent attack, focusing on the

range of misinterpretations and safety behaviors. Homework was related to filling out the panic diary and listening to the tape of the therapy session. Session 2 started with mapping out a cycle for a recent panic attack and introducing the concept of safety behaviors for the patient. Homework was given to continue to listen to tapes of the therapy session but also dropping safety behaviors. Sessions 3–7 would include behavioral experiments like hyperventilation or other experiments to challenge specific beliefs in catastrophic misinterpretations. Sessions 8–12 focused on challenging remaining beliefs and avoidance and developing a therapeutic blueprint.

For social phobia, Session 1 included reviewing a recent social episode and drawing out a cognitive case formulation. Session 2 included a behavioral experiment with increased/decreased safety behaviors and testing specific negative automatic thoughts. Homework consisted of specific exposure, dropping safety behaviors, and shifting to external focus of attention. Session 3 included videotaping a performance in an analogue feared situation and using video feedback to correct distorted images of the self. Homework included tests of specific predictions. Sessions 4–9 included further specific test of predicted social catastrophes as well as exposure to increase the patient's understanding of what is socially acceptable. Sessions 10–14 consisted of work on residual negative thoughts and development of a therapy blueprint.

Prolonged exposure for PTSD consists usually of 9–12 sessions of 90 min (Foa Hembree, & Rothbaum, 2007). However, due to the limited length of the program, the number of sessions was adjusted to 7–9. Session 1 consisted of presenting the rationale for the therapy and beginning breathing training. Session 2 included discussing common reactions to trauma, presenting rationale for in vivo exposure, creating an exposure hierarchy, and introducing the Subjective Units of Distress Scale. Session 3 focused on presenting the rationale for imaginal exposure and conducting it in the session. Sessions 4–9 included more imaginal exposure as well as wrapping up the treatment and giving an overview of what had been learned. Between each session, patients were given assignments to work on, consisting of in vivo exposure and listening to audio recordings of the last session.

### **Comparative description of the treatments**

There are surface similarities between MCT and CBT, mainly that both therapies aim to change

various aspects of cognitions and are goal directed, short term, and structured treatments. However, the mechanisms of change targeted in the two approaches are different. CBT works mainly on the level of content of cognitions and reality tests them, while MCT works on regulating cognitive processes and challenging metacognitive beliefs. For example, in CBT treatments, the focus is on catastrophic misinterpretations and challenging the content of these cognitions; for example, the therapists help patients reality-test thoughts such as “I am having a heart attack” (PD/A), “everyone is looking at me” (SAD), and “the world is dangerous” (PTSD). MCT, in contrast, focuses exclusively on metacognitive beliefs and processes. Therapy is not focused on thoughts such as “I am having a heart attack” but on the processes such as worry as a response to such thoughts. In MCT, the goal is to change how patients directly experience and respond to thoughts by changing the underlying metacognitions that drive worry and rumination. Further differences between CBT and MCT can be found in the use of exposure. In the treatment for PTSD, Foa et al. (2007) use multiple exposures to the traumatic memory or event, while in MCT exposure to the trauma memory and related situations is not considered necessary. In general, MCT may be considered a trans-diagnostic framework in the approach to psychological disorders as the CAS is identified in all disorders (Wells, 2009). It is also evident that CBT has common elements across disorders that form the basis for a trans-diagnostic approach (e.g., Norton & Barrera, 2012), but it is most typically implemented based on content-specific models (e.g., memory exposure in PTSD versus challenging catastrophic misinterpretations in panic). Given the research questions of the present trial and the established efficacy of CBT for specific disorders, disorder-specific CBT treatments were used.

### Therapists

Four clinical psychologists and a psychiatrist available at the department at the time of the trial served as study therapists. They were nested within treatment conditions and provided only one of the treatments. They were allocated to the patients according to the decisions of a clinical leader. All the therapists had 2 years of formal clinical training in CBT or MCT. In the CBT group, the average length of clinical experience was higher ( $M = 10.0$  years,  $SD = 10.8$ ), while it

was lower in the MCT group ( $M = 2.5$  years,  $SD = 0.7$ ). A CBT expert (first author), and an MCT expert (second author), supervised the therapists weekly and was responsible for maintaining competence and adherence to the models. The mean overall competence and adherence ratings were 4.00 or above in both conditions (0–6 scales), reflecting adequate integrity levels (Johnson et al., 2017).

### Diagnostic interviews

*Anxiety Disorder Interview Scale IV* (ADIS-IV) is a semi-structured diagnostic interview designed to assess the presence, nature, and severity of DSM-IV anxiety and mood disorders (Brown et al., 1994). The interviews were conducted by postgraduate clinical psychology students with satisfactory inter-rater reliability (Johnson et al., 2017).

*Structured Clinical Interview for Diagnosis of DSM-IV Axis I* (SCID-I) is a semi-structured diagnostic interview designed to assess the presence, nature, and severity of DSM-IV personality disorders (First, Gibbon, Spitzer, Williams, & Benjamin, 1997). The interviews were conducted by postgraduate clinical psychology students with satisfactory inter-rater reliability (Johnson et al., 2017).

### Competence and adherence

To address competence and adherence in the two treatment conditions, all the videotapes were evaluated using the MCT Competency Scale (Nordahl & Wells, 2009) for MCT and the Cognitive Therapy Scale (Vallis, Shaw, & Dobson, 1986; Young & Beck, 1980) for CBT. Postgraduate clinical psychology students rated all the available videos from the trial ( $n = 595$ ) with satisfactory inter-rater reliability (Johnson et al., 2017).

### Weekly outcome measure

*Beck Anxiety Inventory* (BAI; Beck, Epstein, Brown, & Steer, 1988) is an instrument with 21 items, measuring anxiety symptoms the last week. The severity of symptoms is rated on a scale from 0 to 3. BAI has been found reliable and valid for measuring anxiety symptoms (Steer, Ranieri, Beck, & Clark, 1993). Cronbach's  $\alpha$  derived from the first assessment scores for BAI was .89.

### Weekly mechanism measure

Cognitive Attentional Syndrome 1 (CAS-1; Wells, 2009) is a 16-item measure purported to assess CAS activation. The first 8 items assess coping activities to deal with negative feelings or thoughts (worry, threat monitoring, avoidance of situations, asking for reassurance, suppressing thoughts, controlling emotions, use alcohol/drugs, and controlling symptoms). Worry and threat monitoring are rated on 0–8 scales in terms of amount of time used; the other coping activities are rated on 0–8 scales in terms of frequencies. The next 8 items measure metacognitive beliefs. Four of these items are negative beliefs (worrying too much could harm me, strong emotions are dangerous, I cannot control my thoughts, and some thoughts could make me lose my mind), and 4 are positive (worrying helps me cope, focusing on possible threat can keep me safe, it is important to control my thoughts, and analyzing my problems will help me find answers). Subjects rate their degree of conviction in each of them on 0–100 scales. Relevant items were averaged to subscales for coping activities, negative meta-beliefs, and positive meta-beliefs. Cronbach's  $\alpha$ s derived from the first assessment scores for these three subscales were .84, .86, and .87, respectively.

### Procedure

The ADIS-IV interviews were conducted at intake, posttreatment, and 1-year follow-up. The SCID-II interviews were conducted at intake and 1-year follow-up. The weekly outcome and mechanism measures were administered to patients every Monday.

### Statistical analyses

Our first hypothesis was tested by examining the time by treatment interaction on the CAS-1 subscales in linear mixed-effects models. These models lead to less biased estimates of statistical tests by adjusting for the interdependence of the repeated observations within individuals that is typical in multilevel longitudinal data (Fitzmaurice, Laird, & Ware, 2004). This dependency is accounted for by introducing individual-specific random effects and by modeling the covariance structure of the residuals. For each of the dependent variables in the analyses, the combination of random effects and covariance structure of the residuals that gave the best fit was chosen. Maximum likelihood was used as the estimation method (Fitzmaurice et al., 2004). Akaike's information criterion

was used to compare the fit of different models. For BAI, a random intercept and slope and a one-lag autoregressive (AR(1)) covariance structure for the residuals turned out to have the best fit. For coping activities, a random intercept and slope and an AR(1) covariance structure for the residuals turned out to have the best fit. For negative meta-beliefs, a random intercept and an AR(1) covariance structure for the residuals were best. For positive meta-beliefs, a random intercept and slope, a random interaction of intercept and slope, and a diagonal covariance structure for the residuals were best.

The main purpose of this study was to examine how within-person changes in mechanism variables affected subsequent within-person changes in anxiety from week to week during treatment. The intervention in one of the treatment conditions was designed to cause these within-person effects. Also as a result of the interventions, all these variables were expected to change over time. For this situation, where change is deliberately sought for, Wang and Maxwell (2015) argue that person-mean centering of the time-varying predictors is the proper disaggregation method of the within-person and between-person effects. Any kind of de-trending—which is controlling for the effect of time—would remove the purposefully designed experimental manipulation and could prevent the discovery of between- and within-person effects of interest. However, as recommended by Falkenström, Finkel, Sandell, Rubel, and Holmqvist (2017), we included time in a second set of analyses to explore the robustness of the findings.

We proceeded with the mean-centered approach in a series of mixed models using anxiety (BAI) as a dependent variable and the person-mean-centered mechanism variables (CAS-1) and the person-mean of the mechanism variables as independent variables. A separate analysis was conducted for each mechanism variable (coping activities, negative meta-beliefs, positive meta-beliefs). To establish a temporal sequence between mechanism and outcome, the mechanism variables were lagged. Thus, we estimated the effect of mechanism on anxiety in the following model:

$$\begin{aligned} \text{BAI}_{it} = & \beta_{00} + \beta_{01}\text{MeanCAS} - 1_i \\ & + \beta_{10}(\text{CAS} - 1_{i,t-1} - \text{MeanCAS} - 1_i) \\ & + u_{0i} + u_{1i}\text{Week} + e_{it} \end{aligned}$$

Anxiety score at time point  $t$  for person  $i$  is a function of a fixed intercept,  $\beta_{00}$ ; a fixed effect of the

person's mean on the mechanism variable,  $\beta_{01}$  (between-person effect); a fixed effect of the time-specific deviation from the person's mean on the mechanism variable,  $\beta_{10}$  (within-person effect); a person-specific random intercept,  $u_{0i}$ ; a person-specific random effect of week,  $u_{1i}$ ; and a week- and person-specific residual,  $e_{it}$ . Then, as mentioned above, we added time as a predictor in a second set of analyses.

Next—to explore the possibility of reversed causation—the CAS-1 variables and BAI switched roles in the models. That is, lagged person-mean-centered BAI and person-mean of BAI were the independent variables, whereas the CAS-1 variables were the dependent variables in the models.

There were no missing data, except for those due to drop out from treatment. To correct for the possibility of Type I error, a sequential rejective approach to the study hypotheses was applied (Holm, 1979). The most extreme  $p$ -level was compared to the alpha significance level of .05 (two-tailed) divided by the number of tested hypotheses (6), yielding a level of .0083. Then, the next most extreme  $p$ -level was compared to  $.05/5 = .010$  and so forth. For the exploratory comparisons, a liberal  $p$ -level of .05 (two-tailed) was used. The program SPSS 23.0 was used.

## Results

### Preliminary analyses

For patients' raw CAS-1 and BAI scores, we calculated intraclass correlations (ICCs) to estimate the proportion of the variation in the scores that were accounted for by between-patient variability. The ICCs were .60 for coping activities, .68 for negative meta-beliefs, .71 for positive meta-beliefs, and .64 for anxiety. This means that within-person variation accounted for 40% in coping activities, 32% in negative meta-beliefs, 29% in positive meta-beliefs, and 40 and 36% in anxiety of the total variance across the scales.

### Changes over the course of treatment

The weekly scores on the BAI and the CAS-1 subscales were regressed on time (week) and treatment (MCT vs. CBT) in mixed models. As previously reported (Johnson et al., 2017), there was a significant effect of time on the BAI scores,  $B = -.87$ ,  $SE = .13$ ,  $t(71.2) = -6.48$ ,  $p < .001$ . Moreover, adding treatment and time by treatment interaction as predictor

**Table 1.** WP and BP effects of CAS-1 variables on BAI.

Predictors	<i>B</i>	<i>SE</i>	<i>df</i>	<i>T</i>
WP coping activities	0.39	.32	417.7	1.25
BP coping activities	5.42	.57	64.1	9.95*
WP negative meta-beliefs	0.04	.02	449.0	2.08*
BP negative meta-beliefs	0.21	.04	64.6	5.19*
WP positive meta-beliefs	0.07	.02	407.7	3.15 <sup>a,*</sup>
BP positive meta-beliefs	0.16	.04	64.0	3.85*

Note. Results of separate mixed models for each process variable. CAS-1 = cognitive attentional syndrome I; BAI = Beck Anxiety Inventory (Beck, Epstein, Brown, & Steer, 1988); WP = within-person; BP = between-person.

<sup>a</sup>Significant according to a sequential rejective approach to the study hypotheses for WP effects (Holm, 1979) with overall  $\alpha = .05$  (two-tailed tests).

\* $p < .05$  (two-tailed).

showed a trend toward a significant interaction effect,  $B = -.51$ ,  $SE = .26$ ,  $t(69.4) = -1.95$ ,  $p = .055$ , the negative sign of the coefficient suggests that the MCT patients improved more than the CBT patients (coding: CBT = 0, MCT = 1). There were time effects on all three CAS-1 subscales (all  $t$ s absolute values were higher than 5.00). Our first hypothesis that the CAS-1 features would decrease more in MCT than in CBT was supported by the significant time by treatment interaction effects for negative meta-beliefs,  $B = -1.55$ ,  $SE = .60$ ,  $t(90.4) = -2.57$ ,  $p < .012$ , and positive meta-beliefs,  $B = -1.71$ ,  $SE = .67$ ,  $t(60.8) = -2.55$ ,  $p < .013$ . Again, the negative sign of the coefficients suggests that the MCT patients changed more than the CBT patients. However, our first hypothesis was not supported for coping variables as there was no interaction for this variable,  $B = -.02$ ,  $SE = .04$ ,  $t(61.6) = -.44$  ns.

### Within-person relationships

The disaggregated within- and between-person component of the CAS-1 was included as predictors of BAI (see equation). Table 1 presents both the significances for the within-person relationships when corrected for multiple tests and the significances for the individual tests. There was a within-person relationship between positive meta-beliefs and subsequent anxiety but not between coping activities and subsequent anxiety. There was a trend that negative meta-beliefs predicted subsequent anxiety ( $p = .039$  compared to  $p = .017$  according to Holm's correction procedure). To examine the sensitivity of our findings, time was included in the three

models. Now, none of the within-person relationships were significant; for coping activities,  $B = -.10$ ,  $SE = .33$ ,  $t(457.9) = -.31$  ns; for negative meta-beliefs,  $B = .03$ ,  $SE = .02$ ,  $t(447.9) = 1.23$  ns; and for positive meta-beliefs,  $B = .03$ ,  $SE = .02$ ,  $t(424.8) = 1.62$  ns. As also presented in Table 1, there were between-person relationships between all the CAS-1 variables and anxiety, that is, the levels on the CAS-1 variables over the course of treatment were related to level of anxiety.

Examining the reversed within-person relationships, it turned out that the within-person component of anxiety predicted subsequent positive meta-beliefs,  $B = .30$ ,  $SE = .09$ ,  $t(374.0) = 3.50$ ,  $p < .001$ , but not subsequent coping activities,  $B = .01$ ,  $SE = .01$ ,  $t(466.5) = 1.59$  ns. Anxiety did not predict subsequent negative meta-beliefs,  $B = .18$ ,  $SE = .10$ ,  $t(456.7) = 1.92$ ,  $p = .056$ .

Finally, there were no significant interactions between the CAS-1 variables and treatment condition or between the within-person and between-person components of the CAS-1 variables in predicting anxiety. On the other hand, examining the interaction between the within- and between-person effects of anxiety on subsequent positive meta-beliefs and treatment condition revealed that the within-person effect was stronger in CBT than in MCT,  $B = .34$ ,  $SE = .17$ ,  $t(386.5) = 2.01$ ,  $p < .05$ , whereas the between-person effect was weaker in CBT than in MCT,  $B = -1.46$ ,  $SE = .61$ ,  $t(65.8) = -2.40$ ,  $p < .05$ . Analyzing separate treatment groups revealed a significant within-person relationship between anxiety and subsequent positive meta-beliefs in CBT,  $B = .49$ ,  $SE = .13$ ,  $t(163.9) = 3.95$ ,  $p < .001$ , but not in MCT,  $B = .15$ ,  $SE = .12$ ,  $t(245.6) = 1.26$  ns, and a significant between-person relationship between level of anxiety and level of positive meta-beliefs in MCT,  $B = 1.66$ ,  $SE = .39$ ,  $t(33.7) = 4.29$ ,  $p < .001$ , but not in CBT,  $B = .42$ ,  $SE = .45$ ,  $t(33.5) = .93$  ns.

## Discussion

In summary, the results show that (1) negative and positive metacognitive beliefs decreased more in MCT than in CBT over the course of treatment; (2) also coping activities decreased, but they did not change more in MCT than in CBT; (3) time-specific changes in positive metacognitive beliefs predicted variations in subsequent anxiety across the two treatments (within-person effect); (4) there was a trend for a predictive relationship between time-specific

changes in negative metacognitive beliefs and subsequent anxiety; and (5) coping activities did not appear to be predictive. In terms of our original hypotheses, we found evidence to support the role of the CAS (especially metacognitive beliefs) as mechanism of change in MCT. Positive meta-beliefs exhibited a within-person relationship to anxiety across treatments, suggesting that these meta-beliefs also work on anxiety in CBT. CAS coping activities appeared to change in a similar degree in both treatments. This is not surprising as this is the area of most overlap in the approaches. For example, both MCT and CBT aim to reduce unhelpful coping behaviors such as avoidance or unhelpful self-control strategies (safety behaviors). However, only MCT aims to modify metacognitive beliefs, and we found that such beliefs did indeed change more in MCT than CBT. We found clearer effects for positive than for negative metacognitive beliefs in determining anxiety change. This is somewhat surprising, given the greater emphasis in the MCT model on negative beliefs as the most important causal factor. However, it may reflect aspects of therapy as delivered in this study because positive metacognitive beliefs are often easier to challenge in therapy than negative ones and the MCT therapists in this study had relatively little experience.

Our results “replicate” the findings of our previous analyses of data from the same sample with a different measure—the CAS-1 used currently, instead of the Meta-Cognition Questionnaire—and a different time lag—1 week instead of 4 days—between mechanism variables and anxiety. Thus, the mechanistic role of metacognitions was corroborated.

We also explored whether there were reversed within-person relationships between the CAS variables and anxiety and found that anxiety predicted subsequent positive metacognitions but only in CBT. Thus, an interpretation could be that the MCT interventions have interrupted a causal influence of anxiety on meta-beliefs. This interpretation must be taken with care as an influence of anxiety on metacognitions was limited to MCT in our previous study (Johnson et al., 2018), although a different measure of metacognitions and a different time lag were investigated here. Time-specific changes in anxiety did not predict coping activities or negative metacognitive beliefs.

The hypothesized finding of a within-person relationship between positive meta-beliefs and anxiety was not robust, however, in that it disappeared when time was controlled. This pattern of findings may be interpreted in several ways. Unobserved confounders



external to the treatments (e.g., positive life events, internal healing processes) may have caused improvements in metacognitions as well as anxiety (Falkenström et al., 2017) and the inclusion of time may have controlled for this external cause. For example, in the metacognitive model of PTSD, time is thought to be associated with a reflexive adaptation process in which acute stress symptoms spontaneously decay as metacognitions form a rudimentary plan for regulating cognition under threat (Wells, 2009). On the other hand, if the trends in the two variables result from the circular causal relationship between positive meta-beliefs and anxiety, controlling for time will—as shown by Wang and Maxwell (2015)—artificially diminish the within-person effects. Given that decisive treatment-external changes are less likely to occur during a short inpatient phase, we favor the last interpretation.

Several study limitations should be noted. First, we did not include a control group, which may introduce a possible confounding effect of time. Second, the two treatments contained several shared elements, and the study was carried out at one department, which suggests that there may have been contamination of the therapies between the patients during the treatment phase. However, patients were explicitly given instructions not to talk about the treatment outside the therapy room and instead focus on other aspects related to the inpatient setting. Third, even though all the therapists had documented competency in CBT and MCT, there was clear difference in experience in favor of the CBT group. Fourth, the within-person relationships were studied on a weekly level, and other time levels could have produced different results. Fifth, although we could rule out any stable third variables as alternative explanations of the within-person relations we identified, we still cannot rule out the possibility that an unknown time-varying variable accounted for the within-patient relation of positive meta-beliefs and subsequent anxiety. Sixth, although the three studied CAS-I subscales proved to have satisfactory internal consistencies, they were constructed for the present study based on the face validity of the items and have not undergone a systematic psychometric investigation. Thus, the item groups may not reflect the intended latent constructs equally well, and this may have influenced the findings.

## Conclusions

The present results indicate a within-person relationship between positive metacognitive beliefs and subsequent

anxiety. Because within-person relationships are the focus of therapy models and therapists, this finding can be rather directly translated to a recommendation for clinical practice. It suggests that anxiety can be reduced through a reduction of positive metacognitive beliefs. Future studies should examine the potential influence of coping activities on a smaller time scale, for instance, using experience sampling methods.

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