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Behavior Therapy xxx (2023) xxx–xxx

Behavior Therapy

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Prospective Relations Between Dysfunctional Metacognitive Beliefs, Metacognitive Strategies, and Anxiety: Results From a Four-Wave Longitudinal Mediation Model

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The metacognitive model of psychological disorders suggests that emotional disorders are related to maladaptive metacognitive strategies corresponding to underlying dysfunctional metacognitive beliefs. There is substantial empirical evidence supporting a role of metacognition in psychopathology, but fewer studies have evaluated the metacognitive model using longitudinal data and taken into consideration its differentiation between components and how they are hypothesized to be related to each other. Thus, more specific model evaluation is important as it relates to identifying mechanisms of disorder with a potential to provide clinical advances. In the present study, 868 participants took part in a 4-wave survey and reported on metacognitive beliefs and strategies and anxiety symptoms. Two longitudinal mediation models (forward and reversed causation) were run to test temporal precedence and bidirectional relations. The results indicated that metacognitive beliefs significantly predicted metacognitive strategies, which further predicted anxiety symptoms and mediated the indirect effect in the relationship between metacognitive beliefs and anxiety over time. The relationship between metacognitive beliefs and anxiety symptoms over time were bidirectional, but this relationship was not accounted for by metacognitive strategies. These findings largely support central predictions set forward by the metacognitive model and indicate that metacognitions play a preceding and maintaining role in anxiety.

Keywords: anxiety; metacognition; S-REF model; metacognitive; cognitive attentional syndrome

THE METACOGNITIVE MODEL of psychological disorders (Wells, 2019; Wells & Matthews, 1994) emphasizes a set of maladaptive metacognitive strategies, named the *Cognitive Attentional Syndrome* (CAS), as the proximal cause of emotion disorders. The CAS consists of worry/rumination, threat monitoring, and maladaptive coping strategies, and is directed by underlying metacognitive beliefs (i.e., beliefs about cognition). Negative beliefs about cognitive control (e.g., "worry is uncontrollable") are considered the most influential, with a smaller contribution from other metacognitive belief domains such as positive metacognitive beliefs (e.g., "worry helps me avoid mistakes") and cognitive confidence beliefs (e.g., "I have a poor memory") (Nordahl, et al., 2022a; Sun et al., 2017; Wells, 2019). Based on this perspective, formulation and treatment of anxiety should focus on metacognitive strategies (e.g., worrying) and corresponding metacognitive beliefs (e.g., "I must worry to be prepared"; "I cannot stop worrying") as they interact in maintaining and intensifying symptoms (Wells, 2019). Metacognitive therapy (MCT; Wells, 2009) was specifically designed to reduce dysfunctional metacognitive strategies and modify dysfunctional metacognitive beliefs and has shown promising results as a treatment of anxiety disorders and depression (Normann & Morina, 2018). Nonethe-

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.
The authors declare no conflicts of interest.
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Please cite this article as: Nordahl, Anyan and Hjemdal, Prospective Relations Between Dysfunctional Metacognitive Beliefs, Metacognitive Strategies, and Anxiety: Results From a Four-Wave Longitudinal Mediation Model, Behavior Therapy, https://doi.org/10.1016/j.beth.2023.02.003

less, more research is needed on the metacognitive model as it has the potential to transform our understanding of psychological disorders if its central predictions hold (i.e., shifting from a therapeutic focus on the content of thoughts or reflexive networks to top-down cognitive regulation).

The suggested link between dysfunctional metacognition and emotion disorder has been reliably established (Sun et al., 2017), but there is still a need to evaluate the relationships between the different components specified and distinguished in the metacognitive model. Key questions arising from the model include the following: whether metacognitive beliefs are linked to metacognitive strategies (i.e., the CAS); if the CAS is linked to emotional distress symptoms; and to establish whether metacognitive factors have a preceding and/or contributory role in emotion disorder symptoms. These specific questions are important as they relate to identifying underlying mechanisms of disorder with the potential to provide significant clinical advances (Capobianco & Nordahl, 2021; Schweiger et al., 2019; Wells, 2019).

Longitudinal data is a necessity to address questions about temporal precedence, and according to the metacognitive model (Wells & Matthews, 1994), metacognition should precede metacognitive strategies (CAS) and emotion disorder symptoms in time-series data. A unidirectional relationship where symptoms only gave rise to later metacognition would not be consistent with the model. However, the model allows for reciprocal relationships in which emotion can also impact on metacognition. For example, emotion disorder symptoms may strengthen dysfunctional beliefs about cognition if they are interpreted as a sign of loss of cognitive functioning (Wells & Matthews, 1994). In addition, metacognitive strategies may also impact on metacognition. For example, thought suppression may increase the frequency of intrusions and thereby strengthen beliefs about lack of cognitive control (Wells, 1995). Hence, the metacognitive model makes specific predictions about how emotion disorders and symptoms are initiated and maintained that are well-suited for empirical evaluation.

There are several prospective studies on the relationship between metacognitive factors and anxiety that support the role of metacognitive beliefs (e.g., Capobianco et al., 2019; Nordahl et al., 2022b) and metacognitive strategies (Ebrahimi et al., 2022) as precedents to anxiety symptoms. Experimental studies that have manipulated metacognitive beliefs have demonstrated their causal impact on symptoms (Capobianco et al., 2018; Myers & Wells, 2013). Consistent with a contributory and maintaining role of metacognitive beliefs in disorder, metacognitive change correlates with (McEvoy et al., 2015; Nordahl et al., 2017; Solem et al., 2009) and precedes (Hoffart et al., 2018; Johnson et al., 2018; Sunde et al., 2021) symptom improvement in psychotherapy. Other studies have reported that metacognitions prospectively predict metacognitive strategies in nonclinical individuals (Fergus & Wheless, 2018; Thielsch, Andor, et al., 2015; Weber & Exner, 2013) and in patients with anxiety disorders (LaFreniere & Newman, 2019; Thielsch, Ehring, et al., 2015; Wahlund et al., 2021).

Fewer prospective studies have taken into consideration the differentiation between metacognitive beliefs, metacognitive strategies, and symptoms, and evaluated their hypothesized temporal order as specified by the metacognitive model (Wells, 2009). Ryum and colleagues (2017) reported that worry (a metacognitive strategy) and metacognitions made unique contributions to anxiety over three time points but did not report on the temporal relations between worry and metacognitions. Thielsch and colleagues (2015) reported that negative metacognitive beliefs caused worry (but not vice versa) and that worry had a reciprocal relationship with sleep problems. Hallard et al. (2021) found that metacognitions prospectively predicted a thought control strategy that further predicted suicidal ideation. Johnson and Hoffart (2018) reported within-person connections between metacognition and metacognitive strategies, and that change in these mechanisms influenced anxiety symptoms over time in patients receiving MCT.

Although these studies have tested key hypotheses and contributed with evidence for the metacognitive model with longitudinal data, these previous studies are limited in that they either do not evaluate the temporal relations between all levels distinguished in the metacognitive model, or in that they have included very few variables as indicators in evaluating the model. Thus, prospective studies that clarify the relationships between metacognitions, metacognitive strategies, and symptoms in a broader sense are largely lacking, and further studies are needed to provide better understanding of mechanisms at different levels in emotional distress and disorder.

In the present study, we therefore aimed to evaluate central predictions set forward by the metacognitive model, namely that metacognitions precede metacognitive strategies, prospectively predict anxiety symptoms, and that metacognitive

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strategies will mediate the relationship between metacognitions and anxiety symptoms. To achieve this aim and test the specific hypotheses outlined below we gathered data over four time points that were analyzed using a four-wave longitudinal mediation model including evaluation of reversed causation to test for bidirectional relations and to evaluate temporal precedence among the variables. In line with the metacognitive model, our hypotheses were as follows: (1) metacognitive beliefs, metacognitive strategies, and anxiety symptoms will be positively intercorrelated; (2) metacognitive beliefs will prospectively predict metacognitive strategies; (3) metacognitive strategies will prospectively predict anxiety symptoms; and (4) metacognitive strategies will mediate the relationship between metacognitive beliefs and anxiety symptoms. In addition, we were interested in the reversed causal relationships as, for example, a unidirectional relationship where anxiety symptoms prospectively predict metacognition but not vice versa, would not be consistent with the metacognitive model.

Material and Methods

PROCEDURE AND PARTICIPANTS

The current study used data from an online self-report survey with four measuring points each 6 weeks apart using a program provided by the Norwegian University of Science and Technology. Participants were invited through advertisement on social media assisted by several Norwegian voluntary organizations for mental health and were offered participation in a lottery to win a laptop if they completed the survey at all four time points. Participants were gathered at convenience but had to be 18 years old or above and had to be able to read Norwegian. The research was conducted in accordance with the Declaration of Helsinki and was approved by the Norwegian Regional Committee for Medical and Health Research Ethics (REC; reference: REK-Midt, 2016/705). Upon entering the survey portal, participants were presented with an information sheet that was approved by REC and were informed that proceeding to the main survey would be regarded as a signed informed consent.

A total of 868 participants participated in the study and in the total sample, the mean age was 33.90 (*SD* = 12.92) years and 660 (76.0%) of the participants were female. Two hundred and sixty (30.0%) reported they were single, 138 (15.9%) were in a relationship, 418 (48.2%) cohabiting or married, 42 (4.8%) separated or divorced, four (0.5%) reported being widowed, and six (0.7%) did not report their marital status. In terms of occupational status, 299 (34.4%) were students, 367 (42.2%) reported to be working, 25 (2.9%) were unemployed, 26 (3.0%) reported being on short-term sick leave, 115 (12.7%) were on long-term sick leave (> 1 year), 37 (4.3%) reported being retired, while 4 (0.5%) did not report their occupational status. In all, 339 (39.1%) endorsed having a higher education (completed 3 years or more at a university or equivalent). The prevalence of anxiety symptoms based on the Generalized Anxiety Disorder (Kroenke, et al., 2001) in the sample were as follows: minimal symptoms (35.4% scoring from 0 to 4), mild symptoms (31.8% scoring from 5 to 9), moderate symptoms (19.5% scoring from 10 to 14) and severe symptoms (13.3% scoring from 15 to 21), with moderate to severe indicating clinical significance.

MEASURES

The Metacognitions Questionnaire 30 (MCQ-30; Wells & Cartwright-Hatton, 2004) is a 30-item self-report scale measuring beliefs about cognition (i.e., metacognitive beliefs). Responses are required on a 4-point scale ranging from 1 ("do not agree") to 4 ("agree very much"). MCQ-30 has a replicable five-factor structure: (1) positive beliefs about worry; (2) negative beliefs about the uncontrollability and danger of worry; (3) cognitive confidence; (4) need to control thoughts; and (5) cognitive self-consciousness. Higher scores reflect stronger endorsements of the belief domains in question. The measure has shown good internal consistency with α for the subscales ranging from .72 to .93 and a retest correlation for the total scale of .75 (Wells & Cartwright-Hatton, 2004). In the current study, the internal consistency ranged from .81 to .89.

The CAS-1 (Wells, 2009) is a 16-item self-report scale which was developed to assess metacognitions and the cognitive attentional syndrome in clinical practice. In the current study, the first 8 items were used as they assess metacognitive strategies: item 1 ("How much time in the last week have you found yourself dwelling on or worrying about your problems?"), 2 ("How much time in the last week have you been focusing attention on the things you find threatening?"), 3A ("avoided situations"), 3B ("asked for reassurance"), 3C ("tried not to think about things"), 3D ("tried to control my emotions"), 3E ("used alcohol/drugs"), and 3F ("controlled my symptoms"). Items are scored on a scale from 0 ("none of the time") to 8 ("all the time"). The CAS-1 has shown good internal consistency in previous stud-

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ies (Nordahl & Wells, 2019). In the current study, the internal consistency ranged from .89 to .91.

The Generalized Anxiety Disorder (GAD–7; Kroenke et al., 2001; Spitzer et al., 2006) scale is a 7-item self-report measure that assesses anxiety symptoms during the past 2 weeks. All items are answered using a 4-point Likert-type scale format ranging from 0 ("not at all") to 3 ("nearly every day") with total scores from 0 to 21. Higher scores indicate more anxiety symptoms (Kroenke et al., 2001; Spitzer et al., 2006). It was developed as a screener for GAD in primary care setting but is increasingly used as a measure for anxiety in general (Johnson et al., 2019; Magnúsdóttir et al., 2022) and in anxiety disorder research (Dear et al., 2011). In the current study, the internal consistency ranged from .90 to .92.

STATISTICAL ANALYSES

All analyses were performed in Mplus version 8.6 (Muthén & Muthén, 1998–2021), and the Mplus output and codes can be found in the Open Science Framework platform at osf.io/4gw9c. The full-information maximum likelihood (FIML) method was used with robust estimation (MLR) to make use of all available data and account for non-normality. Structural equation modelling (SEM) was applied to test the longitudinal mediation model following the recommendations by Cole and Maxwell (2003). All variables (*Metacognitive beliefs, MCQ; Metacognitive strategies, CAS; and Anxiety symptoms, ANX*) were conceptualized as latent to address attenuating effects due to measurement error problems. The metacognitive beliefs latent factor was specified by the 5 subscales of the MCQ-30, the latent metacognitive strategies factor by item 1-3 from the CAS-1, and the latent anxiety symptoms factor by the 7 items from the GAD-7. This approach supports stronger inferences about direction of causation and reduces potential problems of parameter biases (Selig & Preacher, 2009). Model fit was evaluated with the following indices: Standardized Root Mean Square Residual (SRMR; Browne & Cudeck, 1993) and Root Mean Square Error of Approximation (RMSEA; Hu & Bentler, 1999) values less than .08 and values equal to or less than .06 (upper 90% CI close to or <.08) respectively, a Comparative Fit Index (CFI) and a non-Normed Fit index (NNFI; aka TLI) greater than .90 as acceptable and .95 as good model fit (Hu & Bentler, 1999).

Testing the direction of causation between variables in the longitudinal mediation model proceeds sequentially. The first step of the analyses involved the test of measurement model and its equivalence across time by imposing strong invariance restrictions in the longitudinal factorial invariance framework. The stability of individual differences in each construct from one occasion to the next (i.e., within-domain stability) was tested. To define the baseline model, three sets of structural paths were included following recommendations by Cole and Maxwell (2003): (a) direct effects were estimated for each latent variable to predict subsequent follow-up assessment of itself; (b) correlations between all latent variables at Time 1 were freely estimated; (c) all residuals of latent endogenous variables were freely estimated within each occasion of measurement (from Time 2 to Time 4). To account for the short time interval between occasions of measurements and the assumptions of stability and stationarity in cross-lagged panel models, autoregressive and cross-lagged paths were constrained equal (Cole & Maxwell, 2003; Little et al., 2007; Selig & Preacher, 2009). The tenability of these constraints was tested using a chi-square difference test. If the constrained model fits the data significantly worse, the imposed constraints are untenable (Little et al., 2007).

The second step of the analyses involved estimating a cross-domain longitudinal model. Here, cross-lagged paths were estimated from metacognitive beliefs on to metacognitive strategies, and from metacognitive strategies to anxiety symptoms (Panel A, Fig. 1). In the third step of the analyses, direct effects from metacognitive beliefs to anxiety symptoms were included (Panel B, Fig. 1). Here, longitudinal mediation by CAS strategies can now be formally assessed in the relation between metacognitive beliefs and anxiety symptoms (i.e., *forward causation model*). Finally, a follow-up reverse causation model was tested by including direct paths from anxiety symptoms to metacognitive beliefs and cross-domain paths from anxiety symptoms to the metacognitive strategies as well as from metacognitive strategies to metacognitive beliefs (Panel C, Fig. 1).

Results

MISSING DATA DIAGNOSTICS

From the total of 868 participants, 387 participants participated in all four waves of the survey, while 133 participated in three, 128 participated in two, and 220 participated once. Data were available for 804 participants while 64 participants had incomplete data on all variables and were excluded from analyses. Prior to performing the analysis, we conducted missing data analysis to rule out any pattern of systematic missingness in the variables that might be dependent on previ-

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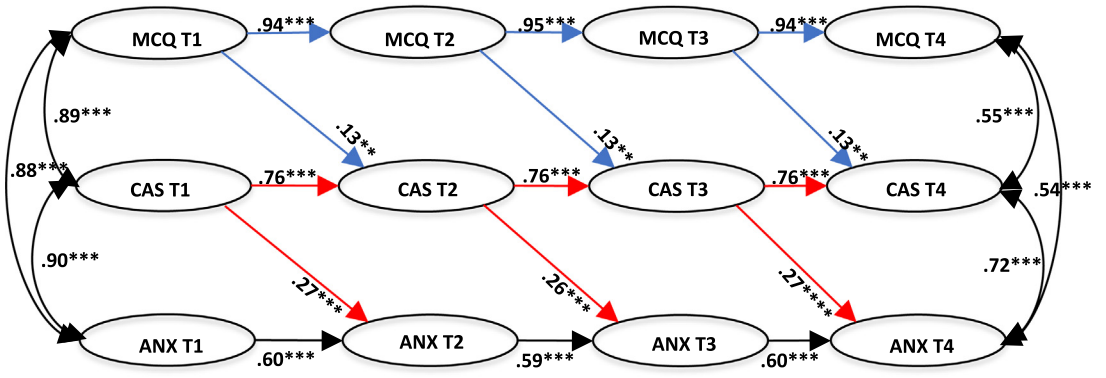
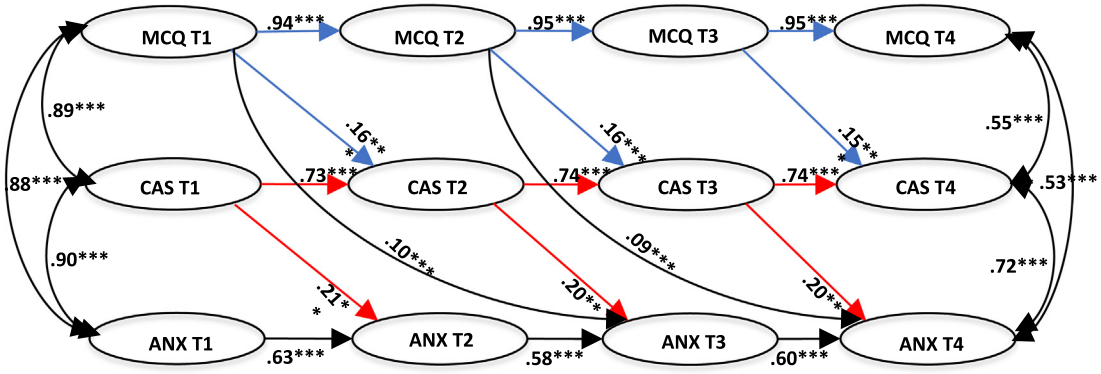
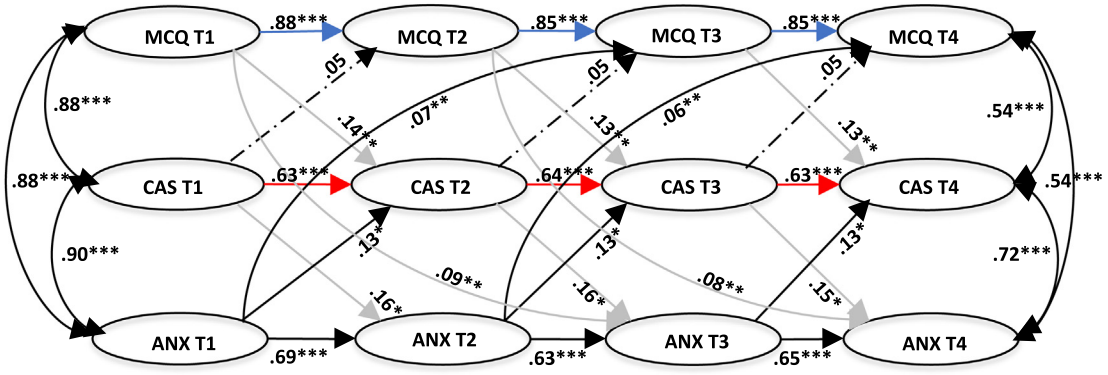


Panel A: Longitudinal Cross-domain Model



Panel B: Longitudinal Mediation Model



Panel C: Reverse Causation Model

FIGURE 1 MCQ = Metacognitive beliefs (i.e., metacognition); CAS = Cognitive attentional syndrome (i.e., metacognitive strategies); ANX = Anxiety symptoms. Statistically significant effects are solid lines, whereas non-significant effects are dotted lines. Within-wave, cross-sectional associations, and autocorrelations of indicator uniqueness for latent variables were estimated but not displayed.

ous values, hence violating the assumption of missing at random (MAR) or missing completely at random (MCAR). First, we systematically analyzed missing data patterns by investigating whether any differences exist between completers and non-completers at each wave with their initial levels of metacognition, metacognitive strategies and anxiety symptoms. These systematic analyses revealed that there were no differences between completers and noncompleters in their initial levels of metacognition, metacognitive strategies and anxiety symptoms (See Supplementary Material Table S1). Additionally, Little's Missing Completely at Random (MCAR) test was conducted to identify patterns of missing values across waves, testing the null hypothesis that missingness is com-

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pletely at random. The result did not reject the null hypothesis: $\chi^2 = 335.51$, $df = 340$, $p = .559$. With both analyses supporting random missingness in the data, it is then plausible to rely on the full information maximum likelihood (FIML). FIML is regard as a state-of-the-art missing data technique because it improves the accuracy and the power of the analyses relative to other missing data handling methods (Schafer & Graham, 2002).

LONGITUDINAL FACTORIAL INVARIANCE OF LATENT CONSTRUCTS

Table 1 shows the results from testing the longitudinal factorial invariance of all latent variables used in this study—metacognitive beliefs, metacognitive strategies, and anxiety symptoms.

Going by the recommended cut-off values (Chen, 2007; Cheung & Rensvold, 2002; Rutkowski & Svetina, 2014), the results show support for strong invariance of the constructs over time. This implies that the relation of the observed variables to the latent variables was constant over time and that there was no change in the meaning of the constructs across the course of the study.

WITHIN-DOMAIN LONGITUDINAL MODEL

This model tested the hypothesis that individual differences in the latent variables were relatively stable over time. The full model showed an acceptable model to data consistency ($\chi^2 = 5706.177$, $df = 3047$, $p < .001$; SRMR = .074; RMSEA = .033 [90% CI = 0.032, 0.034]; CFI = .928; TLI = .926). Within-domain stability coefficients were: (i) metacognitive beliefs (Time 1→ Time 2: .94, $p < .001$; Time 2 → Time 3: .94, $p < .001$; Time 3 → Time 4: .94, $p < .001$), CAS strategies (Time 1→ Time 2: .88, $p < .001$; Time 2 → Time 3: .87, $p < .001$; Time 3 → Time 4: .84, $p < .001$), and anxiety symptoms (Time 1→ Time 2: .86, $p < .001$; Time 2 → Time 3: .84, $p < .001$; Time 3 → Time 4: .82, $p < .001$). High stability coefficients means that the change in individual differences was relatively small, or that individual differences in the latent variables were relatively stable across time. Within-wave positive correlations were found between all variables, supporting Hypothesis 1.

CROSS-DOMAIN LONGITUDINAL MODEL

The cross-domain longitudinal model tested the longitudinal associations between metacognitive beliefs and the metacognitive strategies (Hypothesis 2) as well as between metacognitive strategies and anxiety symptoms (Hypothesis 3). Equality constraints on the autoregressive and cross-

Table 1
Evaluations of Longitudinal Factorial Invariance ($N = 868$)

Model	Type of test	Compared with	χ^2	*df*	*RMSEA*	*CFI*	*TLI*	*Δdf*	*ΔCFI*	*ΔRMSEA*	Decision
Metacognitive beliefs (MCQ)											
M1	Configural invariance		252.746	134	.033[0.027, 0.039]	.987	.982				
M2	Metric invariance	M1	276.451	146	.033[0.031, 0.039]	.986	.982	12	−.001	.000	Accept
M3	Scalar invariance	M2	331.580	158	.037[0.031, 0.043]	.981	.978	12	−.005	.004	Accept
Cognitive attentional syndrome (CAS)											
M1	Configural invariance		1066.959	416	.045[0.041, 0.048]	.940	.928				
M2	Metric invariance	M1	1108.461	437	.044[0.041, 0.048]	.938	.930	21	−.002	−.001	Accept
M3	Scalar invariance	M2	1166.090	458	.045[0.041, 0.048]	.935	.929	21	−.003	.001	Accept
Anxiety symptoms (ANX)											
M1	Configural invariance		628.574	302	.037[0.033, 0.041]	.969	.962				
M2	Metric invariance	M1	641.999	320	.036[0.032, 0.040]	.970	.964	18	.001	−.001	Accept
M3	Scalar invariance	M2	680.526	338	.036[0.032, 0.040]	.968	.964	18	−.002	.000	Accept

Note: χ^2 = Chi-square statistic, *df* = degrees of freedom, *RMSEA* = Root Mean Square Error of Approximation, *CFI* = Comparative Fit Index, *TLI* = Tucker Lewis Index, Δ = Change in statistical values.

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lagged paths $\Delta\chi^2(10) = 9.484$, $p = .487$ did not degrade model fit. The fit of the model (Model 2B) to the data was acceptable ($\chi^2 = 5657.433$, $df = 3051$, $p < .001$; SRMR = .059; RMSEA = .033 [90% CI = 0.031, 0.034]; CFI = .930; TLI = .927). As shown in Panel A, Fig.1 high levels of metacognitive beliefs significantly predicted high levels of metacognitive strategies over time, which also predicted high levels of anxiety symptoms over time, thus, supporting Hypotheses 2 and 3.

LONGITUDINAL MEDIATION AND REVERSE CAUSATION MODEL

The longitudinal mediation and reverse causation models tested whether metacognitive beliefs predicted anxiety symptoms over time (*forward causation model*), or anxiety symptoms predicted metacognitive beliefs over time (*reverse causation model*) and how metacognitive strategies were involved in either the forward or the reverse causal direction (Panel C, Fig. 1). When direct effects were included, the fit of the model was acceptable ($\chi^2 = 5643.593$, $df = 3050$, $p < .001$; SRMR = .056; RMSEA = .033 [90% CI = 0.031, 0.034]; CFI = .930; TLI = .928), and the comparison between a model with freely estimated and constrained direct effects was not significant $\Delta\chi^2(1) = .677$, $p = .410$, indicating that model fit was not degraded by constraining direct effects equal over time. The two direct effects were: (i) Metacognitive beliefs at Time 1 → Anxiety symptoms at Time 3 ($\beta = .10$, $p < .001$) and (ii) Metacognitive beliefs at Time 2 → Anxiety symptoms at Time 4 ($\beta = .09$, $p < .001$). The best estimate of mediation in a longitudinal panel model design is the overall (total) indirect effect of metacognitive beliefs at Time 1 on anxiety symptoms at Time 4. This is the sum of all time-specific indirect effects in a longitudinal mediation model. The total indirect effect was [$\beta = .22$, (95% CI: 0.135, 0.296); $p < .001$], indicating that metacognitive beliefs predicted higher levels of metacognitive strategies, which in turn predicted higher levels of anxiety symptoms. This supports the hypothesis that metacognitive strategies act as the mechanism by which metacognitive beliefs exert their effect on anxiety symptoms—Hypothesis 4.

When estimating a longitudinal mediation model, Cole and Maxwell (2003) recommend testing the bidirectional associations or reverse causal directions in the model. The reverse causation model also showed acceptable fit to the data ($\chi^2 = 5619.815$, $df = 3047$, $p < .001$; SRMR = .053; RMSEA = .019 [90% CI = 0.031, 0.034]; CFI = .931; TLI = .928). In the bidirectional associations, metacognitive beliefs predicted higher levels of anxiety symptoms and anxiety symptoms also predicted higher levels of metacognitive beliefs over time. Although the size of effect of metacognitive beliefs on anxiety symptoms was slightly bigger, it was not significantly different from the effect of anxiety symptoms on metacognitive beliefs χ^2 (1) = 0.652, $p = .419$.

Despite the bidirectional associations between metacognitive beliefs and anxiety symptoms over time, there was no indirect effect of anxiety symptoms through metacognitive strategies on metacognitive beliefs $\beta = .01$, (95% CI: −0.002, 0.011); $p > .05$. (Panel C, Fig. 1).

SENSITIVITY ANALYSES

We assessed the robustness or variations in the results across two sample scenarios namely – will the results change in a complete case analysis using listwise deletion ($n = 295$) versus when using FIML ($n = 804$) to account for missing data. The results pertaining to the longitudinal mediation model (Fig. 1: Panel B) in the main analyses were replicated, and the indirect effect of metacognitive beliefs through metacognitive strategies to anxiety symptoms was [$\beta = .26$, (95% CI: 0.155, 0.371); $p < .001$]. However, the results in the reverse causation model (Fig. 1: Panel C) slightly deviated. First, replicating the main analyses, no indirect effects from anxiety through metacognitive strategies to metacognitive beliefs was observed $\beta = .02$, (95% CI: −0.003, 0.011); $p > .05$. Second, the bidirectional associations between anxiety symptoms and metacognitive beliefs over time was not observed. As this was the only deviating result from the main analyses, and given the substantial drop in samples, the disappearance of the bidirectional effect in the reverse causation model could be due to low power. Since the FIML improves the accuracy and the power of the analyses (Schafer & Graham, 2002), we focus on the main analyses.

Discussion

The primary aim of this study was to test central predictions set forward by the metacognitive model using a four-wave longitudinal mediation model. This approach allowed us to distinguish the different levels and components specified in the theoretical model and to evaluate bi-direct relationships and temporal precedence. Specifically, we were interested to test if dysfunctional metacognitions preceded metacognitive strategies and anxiety symptoms, and if the CAS (i.e., metacognitive strategies) mediated the relationship between dysfunctional metacognitions and anxiety

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symptoms, as predicted by metacognitive theory (Wells, 1995; Wells & Matthews, 1994).

In accordance with our first hypothesis, positive and significant intercorrelations were observed between dysfunctional metacognition, metacognitive strategies and anxiety. Using a cross-domain longitudinal model, we observed that metacognitive beliefs significantly predicted the CAS over time, which further predicted anxiety symptoms over time, in line with our second and third hypotheses. In line with our fourth hypothesis, we observed that metacognitive strategies mediated the indirect effects in the relationship between metacognitive beliefs and anxiety over time, suggesting that the CAS is a mechanism by which dysfunctional metacognitions exerts its effect on anxiety symptoms. However, metacognitive strategies did not fully mediate the relationship between metacognitions and anxiety, indicating a unique contribution from metacognitions that is not accounted for by the CAS, at least as it was measured in the current data. In the main analysis, we found a bidirectional relationship between dysfunctional metacognitions and anxiety symptoms over time, indicating that higher levels of anxiety led to later higher levels of dysfunctional metacognitions. This relationship was not accounted for by the CAS. However, in secondary analysis including only completers, the relationship between dysfunctional metacognitions and anxiety over time was not bidirectional and only the direction from metacognitions to anxiety was significant.

These findings bring further support to the metacognitive model of psychological disorders (Wells, 2019; Wells & Matthews, 1994) in longitudinal data by highlighting a consistent preceding role of dysfunctional metacognitions on maladaptive self-regulatory strategies (i.e., the CAS) and thereby to anxiety symptoms—a putative causal relationship in which the CAS mediates the indirect effect between dysfunctional metacognitions on anxiety symptoms but that also indicates a role for metacognitions in anxiety beyond metacognitive strategies. The relation between dysfunctional metacognitions and anxiety appeared reciprocal in the main analysis, meaning that anxiety symptoms might also influence dysfunctional metacognitions, a mutual prospective association that could constitute a maintenance process in which the CAS was found to be involved in the forward, but not the reverse, relationship. However, in secondary analysis using only completers, the relationship between dysfunctional metacognitions and anxiety was not bidirectional with only the direction from metacognitions to anxiety remaining. Nonetheless, dysfunctional metacognitions and metacognitive strategies do not merely represent an effect of anxiety symptoms and are likely important mechanisms for anxiety that should be addressed in interventions.

Our findings are in line with previous prospective studies that have reported prospective associations between dysfunctional metacognitions and anxiety symptoms (e.g., Ryum et al., 2017), between metacognitive strategies and anxiety symptoms (Ebrahimi et al., 2022), and between dysfunctional metacognitions and metacognitive strategies (e.g., Johnson & Hoffart, 2018). Similar to our findings, others have reported that negative metacognitive beliefs cause worry (i.e., CAS) (but not vice versa) and that worry has a reciprocal relationship with sleep problems (Thielsch et al., 2015). In a different study, metacognitions prospectively predicted thought control (i.e., CAS), which further predicted suicidal ideation (Hallard et al., 2021). There is also some evidence indicating that dysfunctional metacognitions maintain metacognitive strategies and lead to fear of cancer recurrence in patients treated for cancer (Ng et al., 2019). In a recent study, Hoffart et al. (2022) reported that belief in uncontrollability of worry and threat monitoring (i.e. CAS) were central "nodes" in an anxiety network at the within-person level during the COVID-19 pandemic. Adding to previous research, we distinguished between the different levels and components specified according to the metacognitive model (Wells, 2019) using appropriate measures to construct latent variables and we were able to test longitudinal mediation repeatedly through a forward and a reverse causation model. The results add evidence to the notion that metacognition is central to understanding how emotion disorder symptoms are developed and maintained.

In our main analysis, the relationship between dysfunctional metacognitions and anxiety was reciprocal, a finding that has also been reported by others (e.g., Capobianco et al., 2019) and that is consistent with the metacognitive model (Wells & Matthews, 1994). However, metacognitive strategies did not account for the influence from anxiety symptoms to dysfunctional metacognitions over time, suggesting that a different mechanism than the one measured in this study may account for this effect. While others have reported similar findings (Thielsch, Ehring, et al., 2015), we should be careful in interpreting these findings as the timescale of the assessed relationships must be considered and as the effect from anxiety to metacognitions over time did not exist in the sample consisting of only completers. A more robust test of these relations would require experimental

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manipulations of the specified components in evaluating their respective causal effects. In addition, how constructs are operationalized and measured is important. For example, using a latent CAS variable or just a few indicators of this construct can potentially conceal relationships between more specific subdomains at different levels. Some metacognitive strategies are likely more important in reinforcing dysfunctional metacognitions than others. By extension, the unique contribution from metacognitions to later anxiety not fully accounted for by metacognitive strategies indicates a more direct role of metacognition and could be a result of *meta-worry* (i.e., worry about worry; Wells, 1995), not specifically assessed and distinguished in the current study—but which according to the metacognitive perspective is a cognitive state-manifestation of underlying negative metacognitive beliefs that will strongly increase state anxiety. In line with the metacognitive model of generalized anxiety (Wells, 1995), a recent study found that meta-worry is a stronger influence on anxiety compared to trait-worry in a sample of highly anxious individuals (Nordahl, Vollset, et al., 2022).

The clinical implication of our findings is that it is important to formulate and address metacognitive beliefs and strategies (i.e., CAS) in treatment of anxiety. According to the metacognitive model (Wells, 2019), metacognitive strategies are processes operating in a cognitive system that is controlled and directed by a *metacognitive control system* containing metacognitive beliefs and factors necessary for control and regulation of cognition. In psychological disorder it is primarily the metacognitive control system that is the cause of dysfunction and therefore treatment should focus on formulating and modifying the content, strategies, and regulatory influence of the metacognitive system as the most important source of disorder. Targeting the content of cognition, as in cognitive therapies or aiming to change reflexive networks through prolonged exposure techniques as in behavioral therapies, may not be necessary to effectively treat anxiety. Metacognitive therapy (Wells, 2009) was specifically developed to create metacognitive change and is a highly effective treatment for anxiety and depression (Normann & Morina, 2018). There is some evidence indicating that MCT may produce better treatment outcomes than current recommended treatments (Callesen et al., 2020; Nordahl et al., 2018), and we may speculate that the rapid and strong improvements associated with MCT are a result of its more direct impact on psychological mechanisms (i.e., metacognitive beliefs and strategies) in psychopathology compared to other psychological interventions. Meta-analytic evidence indicates that MCT is associated with strong metacognitive change (Normann & Morina, 2018), and in trials comparing the effectiveness of MCT with other types of psychotherapy for generalized anxiety, more metacognitive change and symptom change are reported in MCT compared to competing interventions (Nordahl et al., 2018; van der Heiden et al., 2012; Wells et al., 2010).

The current study has several limitations that must be acknowledged. Participants were recruited at convenience, and we had no information about prevalence of psychopathology or treatment history. The sample consisted of an overrepresentation of females compared to males, which may limit the generalizability of our findings. We relied on a 6-week lag between timepoints for practical purposes to enable a time-interval within which the study could be completed. With regards to modeling stability and change, the time lag between timepoints is very crucial since this may have implications for when hypothesized autoregressive or cross-lagged effects may be significant or not significant (Anyan et al., 2020). Equally spaced, fixed lag schedules between measurement occasions suggests that the cross-lagged effects between the variables occur simultaneously. This may be untenable and introduces limitations to the study as it cannot be ruled out that we may have underestimated the strength of relationships that are more short-lived (e.g., effects of metacognitive strategies on metacognitions and anxiety symptoms). This is because cross-lagged effects can emerge at different lag schedules than fixed lag schedules (Selig & Little, 2012). Furthermore, the GAD-7 was the only indicator of symptoms in the current study. While the GAD-7 is increasingly used as a measure of anxiety not specific to generalized anxiety disorder (Dear et al., 2011; Johnson et al., 2019; Magnúsdóttir et al., 2022), it could be that using this measure favors the metacognitive model as several of its items assess cognitive symptoms and symptoms related to worrying. However, others have tested the metacognitive model of anxiety using different measures, for example, the Beck Anxiety Inventory, which includes more items that address physiological symptoms in addition to cognitive symptoms, and they also report that there are significant relationships between metacognition and anxiety cross-sectionally (Nordahl, Vollset, et al., 2022) and longitudinally (Ryum et al., 2017), supporting the notion that metacognitive factors are related to anxiety in general. Testing the dynamic relationships between trait (i.e., metacognitions) and state (i.e., metacognitive strategies, symptoms) factors

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and their expected influence on each other over time is methodologically challenging, but the design of the current study adds to previous evaluations of the metacognitive perspective. It adds to the literature in evaluating the relevance of mechanisms in psychopathology derived from a promising theory with potential major implications for formulation and treatment.

Future studies should evaluate the metacognitive model as we did in the current study but in the context of depression symptoms and other symptom domains, as well as in clinical samples. For example, there are only a few prospective studies on dysfunctional metacognitions and depression symptoms, and these mostly do not take the different levels and components separated in the metacognitive model into consideration (Cano-López et al., 2022). This area of research is important as it will further test the transdiagnostic nature of the metacognitive model at a mechanistic level with important implications for treatment, as we know that anxiety, depression, and other mental health problems are highly comorbid.

Furthermore, the constructs used in this study ("metacognition" and "CAS") reflect broad domains that may obscure specificity in the relations between more specific subdomains or constructs. For example, negative metacognitive beliefs are considered more important to anxiety than positive metacognitive beliefs, as are some metacognitive strategies (e.g., worrying) considered more central in anxiety than others (e.g., reassurance seeking) (Wells, 2009). It could be that some metacognitions precede symptoms while others play a maintaining role. Thus, future studies should therefore examine relations between even more specific and central constructs than in the current study and also consider incorporating the joint between-person and within-person change to disaggregate the variations at both analyses' levels. Experimental manipulations of metacognition, metacognitive strategies, and symptoms is needed to evaluate their true causal effects more precisely.

In conclusion, the current study adds to previous prospective studies by demonstrating that dysfunctional metacognitions exert a prospective effect on anxiety symptoms repeatedly mediated by metacognitive strategies. While the most compelling test of causal and mediational hypotheses derive from randomized experimental designs, the longitudinal mediation model in a longitudinal panel design is "perhaps the strongest basis for inferring causation" (Belsky et al., 2007, p. 1239) within the context of nonexperimental designs by relying on strict statistical controls (Cole & Maxwell, 2003), as implemented in this study. Thus, our findings imply that dysfunctional metacognitive beliefs and strategies should be considered as important targets in treatment of anxiety and bring further support for the metacognitive model of psychological disorders.

Supplementary data to this article can be found online at https://doi.org/10.1016/j.beth.2023.02.003.

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Received: August 15, 2022
Accepted: February 9, 2023
Available online: xxxx

Please cite this article as: Nordahl, Anyan and Hjemdal, Prospective Relations Between Dysfunctional Metacognitive Beliefs, Metacognitive Strategies, and Anxiety: Results From a Four-Wave Longitudinal Mediation Model, Behavior Therapy, https://doi.org/10.1016/j.beth.2023.02.003