Graduate thesis

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The role of metacognitive change in treatment outcome: A systematic review of the literature to evaluate a central prediction of the S-REF model

Graduate thesis in Clinical Psychology
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To Arne Bendelorm
For teaching us how to appreciate the beauty
of walruses falling down a cliff

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ACKNOWLEDGEMENTS

We never expected to write about metacognitive beliefs or even the metacognitive model. Since the beginning of our friendship we have wandered off in contemplation of whatever caught our interest. After many late nights since the beginning of 2019, through COVID and up until we started working with this thesis, we were shamelessly keen to develop our own theories of what exactly constitutes effective therapy. We did not of course know the exact details of our theories, but we liked the idea of spending even more time together with something theoretical. So we walked into Henrik Nordahl's office at NTNU and asked what the chances were of us conjuring up our own theories of psychopathology writing this thesis. "Well…" he said. "There is Beck, and there is Wells, and perhaps there are you". We knew right there and then that we had to bring ourselves back to earth - or should we say academia. We did however get caught by the idea of writing a theoretical comment on an existing theory of psychopathology. Along the way we realized that it would take years to wrap our heads around all the relevant data we wanted to include. So finally, and luckily, we decided to do a systematic review, keeping our area of interest close to heart. Although writing this review has been far from easy, we are really happy with what we have learned and produced.

We would like to thank our supervisor, Henrik Nordahl, for keeping us clear headed with invaluable remarks and proper guidance. From the very first time we stepped into your office you have cheered us forward and we are truly grateful for that. We would also like to thank Gunhild Elisabet Berget for statistical and emotional aid when what felt like endless hours of reading through articles made our eyes squint.

ABSTRACT

Psychotherapies have yet to demonstrate sufficient empirical grounding in their purported mechanisms of change. Yet, identifying and verifying mechanisms of change could be considered one of the main priorities of therapeutic research. In this review, we set out to synthesize the current empirical data of a relatively new therapeutic model: the metacognitive model – also known as the S-REF model. Our aim was to evaluate the claim that changing dysfunctional metacognitive beliefs would be associated with change in symptom outcomes across different psychological disorders and treatments. We did this by conducting a systematic literature review, which yielded twenty-six articles with a statistical analysis of the relationships between changes in metacognitive beliefs and symptom change across different treatment interventions. We hypothesized that all effective therapeutic interventions change metacognitions following treatment regardless of disorder, and that this change was associated with changes in treatment outcome.

Our synthesis showed that at least some metacognitive change occurred following treatment in all the articles testing for metacognitive change. This was also the case for interventions that did not directly target metacognitive beliefs. Metacognitive therapy (MCT) was, when compared to other treatments, superior in decreasing dysfunctional metacognitions. Overall, we found that changes in metacognitive beliefs were related to symptom change across all mental disorders included in this review, although the contribution of specific metacognitions varied between studies, treatments, and disorders. Change in "negative beliefs about the uncontrollability and danger of worry" appeared as the most relevant metacognitive domain related to change in a variety of symptoms.

This review gives further support to the metacognitive model, reflecting a shift in the understanding of psychopathology from content-oriented to an emphasis on mental regulation. We urge future research to investigate the relations between metacognitions and symptom outcome further, with study designs appropriate for testing causality. Nonetheless, our results show that metacognitive change is a relatively robust correlate of symptom change with the implication that further evaluation of metacognitive mechanisms in psychopathology is warranted.

SAMMENDRAG

Psykoterapier har ennå ikke klart å demonstrere tilstrekkelig empirisk evidens for deres påståtte endringsmekanismer. Å identifisere og verifisere endringsmekanismer kan likevel anses som en av hovedprioriteringene i terapeutisk forskning. I denne litteraturgjennomgangen har vi syntetisert tilgjengelig forskning på en relativt ny terapeutisk modell: den metakognitive modellen, også kjent som S-REF-modellen. Målet vårt var å evaluere påstanden om at endring av dysfunksjonelle metakognitive antagelser er assosiert med endringer i symptomutfall - på tvers av psykiske lidelser og behandlingstilnærminger. Vi gjorde dette ved å gjennomføre en systematisk litteraturgjennomgang, hvor tjueseks artikler ble inkludert på grunnlag av at de hadde en statistisk analyse av sammenhengen mellom endring i metakognisjoner og endring i symptomer på tvers av ulike behandlingsintervensjoner. Vi hypotetiserte at alle effektive terapeutiske intervensjoner endret metakognisjoner gjennom behandlingsløpet, uavhengig av lidelse, og at denne endringen var assosiert med endringer i symptomutfall.

Endring i metakognisjoner ble observert i alle inkluderte artikler som testet for dette. Denne endringen var også tilstede i intervensjoner som ikke adresserte metakognitive antakelser direkte. Metakognitiv terapi (MCT) viste størst reduksjon av dysfunksjonelle metakognitive antakelser i artikler som sammenlignet MCT med andre terapeutiske intervensjoner. Samlet sett var endringer i metakognitive antakelser relatert til endringer i symptomutfall på tvers av alle psykiske lidelser inkludert i denne artikkelen, selv om bidraget til spesifikke metakognisjoner varierte avhengig av studiedesign, behandling og lidelse. Endring i "negative antakelser om at bekymring er farlig og ukontrollerbart" viste seg å være den mest relevante metakognitive antakelsen relatert til symptomsendring.

Denne litteraturgjennomgangen gir ytterligere støtte til den metakognitive modellen, og underbygger et skifte i forståelse av psykopatologi fra innholdsorientert til vektlegging av mental regulering. Våre funn viser at metakognitiv endring kan anses som et robust korrelat til symptomendring, noe som berettiger en ytterligere evaluering av metakognisjoner som en mulig endringsmekanisme i psykopatologi. Vi oppfordrer derfor fremtidig forskning til å undersøke videre forholdet mellom metakognisjoner og symptomutfal med studiedesign som gir mulighet for å teste kausalitet.

1 INTRODUCTION

Psychological disorders refer to a range of conditions where thoughts, feelings, behavior and/or social function is disturbed (WHO, 2022a). They are commonly classified according to the international diagnostic criteria of DSM or ICD, which are revised regularly in concordance with new scientific evidence. The World Health Organization (WHO, 2022a) estimates that in 2019 about 12.5 percent of children and adults worldwide live with a mental disorder, and in Norway it is estimated that 16-22 percent of the adult population fulfills the criteria of a mental disorder within a year (Reneflot et al., 2018). In 2020 alone, these numbers rose due to the COVID-19 pandemic, with an estimated increase of 25-28 percent in depression and anxiety disorders worldwide (Santomauro et al., 2022). Undoubtedly, these conditions can have substantial effects on all areas of life, including performance in school or work, relational issues, and the ability to participate in the community. Mental disorders thus constitute consequential economic expenses, considering depression and anxiety disorders alone cost the global economy 1 trillion US dollars each year (WHO, 2022b).

One of the most important roles of research in psychopathology is to find ways to treat patients effectively. Norcross (2005) estimates that there are more than 500 therapies in use. Although not all of these have been scrutinized with scientific rigor, there has historically been a lasting claim, called the Dodo bird verdict, that all bona fide treatments produce equivalent outcomes regardless of their specific components (Budd & Hughes, 2009). Wampold & colleagues' (1997) famous meta-analysis gives support to this notion, demonstrating that the effect sizes between therapies were homogeneously distributed around zero, indicating that different therapies have more or less the same effect. Similarly, two of the most commonly used treatments, psychodynamic therapy (PDT) and cognitive-behavioral therapy (CBT), has shown to be equally effective in treating depression (Driessen, et al. 2015), anxiety disorders (Keefe et al., 2014) and within a sample of studies on different mental disorders (Steinert et al., 2017). They have also shown to be equally effective compared to pharmacotherapy across different disorders (Huhn, et al., 2014). However, the dodo bird verdict is not in harmony with the literature as a whole. As González-Blanch and Carral-Fernández (2017) have pointed out, there is growing evidence for the superiority of different treatments for specific mental disorders. For instance, exposure and response prevention (ERP) has long been considered the best treatment for obsessive compulsive disorder (OCD; Marks & O'Sullivan, 1988; NICE, 2005). Similarly, cognitive therapy (CT) based on the model by Clark and Wells (1995), has shown large effect sizes compared to

other treatments for patients with social phobia in Mayo & Wilson (2014), and should according to the authors , and in accordance with the NICE guidelines (2013), be regarded as the treatment of choice.

Furthermore, a relatively new treatment for psychological disorders, metacognitive therapy (MCT; Wells, 2009) has shown very promising results for a range of mental disorders, also when compared to other treatments. Two meta-analyses have found MCT to be superior to CBT in treating anxiety and depression with large between-group effect sizes pre- to post-treatment (Normann et al., 2014; Normann & Morina, 2018). Another meta-analysis found that MCT showed large within-group effect sizes in treating PTSD (Brown et al., 2022). Promising results are also found for OCD, schizophrenia, body dysmorphia, grief, and hyposexual desire disorder (Normann & Morina, 2018). Yet, studies of MCT are still relatively scarce, lacking for instance randomized controlled trial-designs. However, two randomized controlled trial- (RCT-) studies have found that MCT showed large effect sizes in treating generalized anxiety disorder (GAD), and produced results superior to CBT (Nordahl et al., 2018) and intolerance-of-uncertainty therapy (UIT; Heiden et al., 2012). Recently a RCT-study by Callesen & colleagues (2020) showed MCT to be superior to CBT in treating depression. Another RCT by Johnson et al (2017) yielded the same results for comorbid anxiety disorders. This may further challenge the Dodo birds claim, as more effective therapeutic interventions could arise in the future.

Evidently, it seems that some treatments could be more effective than others in treating specific mental disorders, and psychotherapies such as MCT could potentially be more effective than other bona fide treatments in general. But to understand and develop better treatments we should know precisely what the effective components are, or to put it differently, what the mechanisms of change are in psychopathology.

1.1 Mechanisms of change

Mechanisms of change refer to the causal steps or process through which therapy (or some independent variable) unfolds and produces change (Kazdin, 2007). As mentioned above, there are a wide variety of different psychotherapies, which are based on different theoretical frameworks for mechanisms of change in therapy. Until now there has been extensive research on the effectiveness of different therapies. Here the results are clear: we know therapy works (Kazdin, 2007). Yet little is known about the mechanisms that cause

symptom change (Moldovan, 2015). And although the validity of the Dodo bird verdict is still debated, it illustrates two important points. Firstly, it may indicate that the theoretical assumptions of mechanisms of change in bona fide treatments are inaccurate, false or that there is equifinality for the purported mechanisms of change in therapy. Today, we lack the evidence to support the current theoretical framework of the leading psychotherapies. For example, while considered effective (Steinert et al., 2017), psychodynamic therapies have so far not been able to overcome the problem of operationalizing their purported mechanisms of change (PDT: Hoffart and Johnson, 2017; ISTDP: Hoviatdoost et al., 2020). Psychodynamic therapies are heterogeneous, but according to (intensive) short term dynamic psychotherapy (Frederickson, 2013, p. 4-5; McCullough et al., 2003, p. 13-25), psychopathology is regarded as a result of anxiety, maladaptive feelings and defense mechanisms blocking adaptive feelings, in which an internal conflict arises. By this account, systematic desensitization of adaptive feelings will dissolve the psychodynamic conflict and thus alleviate the patients problems. In contrast, cognitive behavior therapy states that mental disorders are a result of internal dysfunctional schemas, and therefore all psychotherapies work by altering dysfunctional cognitions, either directly or indirectly (Clark, 1995). In CBT these cognitions are subjected to logical analysis and hypothesis-testing, which is thought to lead to changes in behavior and emotion. Yet, a growing literature has pointed out that the effects of CBT seem to be related to other factors than change in cognition (Longmore & Worrell, 2007; Hayes 2004). For example, component analyses show that CBT does not seem to be more efficacious than one of its core behavioral components alone, behavioral activation, in treating depression - indicating that cognitive interventions do not add additional value to therapy (Longmore & Worrell, 2007). In addition, it may seem that improvements in symptoms most likely occur before the implementation of cognitive techniques (Longmore & Worrell, 2007). Similarly, measures of cognitive mediators are inconclusive, and cognitions do not seem to precede changes in symptoms, which indicate that changes in symptoms may cause the change in cognition (Longmore & Worrell, 2007). These findings in turn contradicts the theoretical framework of CBT, and the notion of changing cognition as a means to ameliorate symptoms.

Secondly, the Dodo bird verdict may indicate that the real mechanisms of change are in fact common factors shared between treatment modalities. Yet, even if we find common factors, we lack the specificity to understand the mechanisms of change. For instance, we know that therapeutic alliance is an important transtheoretical predictor of treatment outcome,

but the specific mechanisms that explain how therapeutic alliance and treatment outcome are related are yet not clear (Kazdin, 2007; Baier et al. 2020).

These considerations are important because in search for effective treatment we need to understand mechanisms of change, which require theoretical frameworks that match the empirical data on mediators and moderators of treatment outcome (Kazdin, 2007). Evaluating predictions from theoretical frameworks should therefore be a driving factor of research on mechanisms of therapeutic change (Kazdin, 2007). As mentioned, the metacognitive model, more precisely referred to as the S-REF model, may be a good candidate for such a framework, as its proposed change mechanisms are possible to evaluate and measure.

1.2 The Self-Regulatory Executive Function model

Adrian Wells began to search for mechanisms and underlying concepts of psychological disorder in the mid-1980s (Capobianco & Nordahl, 2021). He argued that contrary to several findings of its importance, attentional processes and thinking styles such as worry, self-attention or threat monitoring, were not given enough attention in psychotherapy. Contemporary clinical models of disorder, such as CBT and Rational Emotive Behavior Therapy (REBT), were centered on the *content* of cognitions and schemas (Beck, 1976; Ellis & Grieger, 1986), and attentional biases were considered to be bottom-up phenomena, arising from the result of emotion, personality dispositions or environmental factors (e.g., Mathews & Macleod, 1985; Williams et al., 1988). This in turn did not make it a target for therapeutic change (Capobianco & Nordahl, 2021). However, Wells and Matthews (1994) argued that attentional bias and cognitive thinking styles are generated by an interaction between higher-level controlled processing and lower-lever automatic processing and can therefore be altered directly through the higher-level processes. This led them to develop the metacognitive model, which they considered to be a transdiagnostic framework for understanding psychopathology.

The core principles of the metacognitive model are presented by Wells and Matthews' (1994, 1996) Self-Regulatory Executive Function (S-REF) model, which was a new model of the cognitive architecture, including how self-regulation is conducted as a function of the processes and knowledge contained at different levels of cognition. The S-REF model offers an account of how cognitive and metacognitive factors are involved in the top-down control or maintenance of psychological disorders. It states that there are three interacting levels of

processing: automatic and reflexive processing (low-level processing), conscious processing, which is attentionally demanding (cognitive style), and a level of stored knowledge and self-beliefs (meta-level; Wells, 2009). These levels construct reciprocal feedback loops in concordance with input from the world. According to this framework, psychopathology is maintained by the activation of a maladaptive and universal cognitive style called the Cognitive Attentional Syndrome (CAS), which locks people in prolonged negative emotions or appraisals in response to internal events (CAS; Wells, 2009). In essence, CAS is a state of processing where negative self-relevant information is prioritized and becomes perseverative. The CAS consists of both conceptual and attentional processes. The conceptual component is characterized by excessive worry and rumination, while the attentional component constitutes self-focused attention and threat monitoring. In addition, this cognitive style inadvertently maintains distress through dysfunctional coping strategies, such as avoidance, thought suppression, or substance abuse (Wells, 2009). Although this model has recently been revised and expanded (Wells, 2019), its main components remain.

The state of CAS is, according to the S-REF model, considered to be a conscious processing style. In contrast to the cognitive model (Beck & Haigh, 2014), persistent worry and rumination are therefore seen not as a result of schemas, but as a strategic response to internal events, such as negative automatic thoughts and emotions (Wells, 2009, p. 197). The metacognitive model thus makes an emphasis on the difference between experiencing negative emotions and thoughts, which all people do from time to time, and the development and maintenance of psychopathology through prolonged CAS-strategies. These strategies are according to the S-REF model influenced by the beliefs at the meta-level, namely metacognitive beliefs.

Metacognitive beliefs are defined as beliefs about cognition itself and dysfunctional metacognitive beliefs function to sustain CAS (Wells, 2009). Moreover, there are several types of dysfunctional metacognitive beliefs. Positive metacognitive beliefs manifest themselves declaratively as thoughts about the usefulness of the CAS. This could appear as statements such as: “Focusing on danger will keep me safe” (Wells, 2009, *p*. 15). In contrast, negative metacognitive beliefs concern the uncontrollability of thoughts and the danger and importance of them, such as “I have no control over my worrying/rumination” or “Worrying can damage my body” (Wells, 2009, *p*. 16). Wells argues that negative metacognitive beliefs are more relevant to psychopathology than positive beliefs (Wells, 2019). These beliefs are thought to be related to procedural metacognitive beliefs, which operate as implicit instructional information that guides cognition (Wells, 2019). This makes the content of

cognition an ineffective therapeutic target, as negative thoughts and feelings in CAS are maintained not by their symbolic value but by our beliefs about them and our self-regulatory strategies. From the perspective of the S-REF model, dysfunctional metacognitive beliefs are the mechanism of change in mental disorders (excluding neurological disorders such as ADHD, autism, dementia and schizophrenia), and should therefore occur regardless of treatment modalities.

1.3 Metacognition and psychopathology

Previous research has supported the notion that metacognitive beliefs are related to psychological disorders. A meta-analysis by Sun & colleagues (2017) found elevated metacognitive beliefs across different psychological disorders, including eating disorders, major depressive disorder, generalized anxiety disorder, schizophrenia spectrum, and obsessive-compulsive disorder, compared to healthy controls. Of particular importance were "negative beliefs concerning the uncontrollability and danger of worry", and "beliefs about the need to control thoughts", which showed large and robust effect sizes across patients (Sun et al., 2017). Similarly, systematic reviews have found metacognition to be related to social anxiety (Gkika et al., 2018), addictive behaviors (Hamonniere & Varescon, 2018) and emotional distress in those with physical illnesses (Lenzo et al., 2020). Interestingly, Keen & colleagues (2022) found in their review on health anxiety and somatic distress, that metacognitions, in addition to being related to emotional distress, were also related to physical symptoms. These findings suggest that metacognitive beliefs are related to a range of psychopathology, emotional distress symptoms and physical symptoms.

1.4 Aims

In order to find "true" mechanisms of change one has first to find a causal relationship between the mechanism variable, metacognitions, and treatment outcome (Kazdin, 2007). Studies should include several measures points to ensure temporal precedence of metacognitions, and there should be solid experimental design studies. While these types of studies founded on the S-REF model are still largely lacking (Wells, 2019), a preliminary step forward is to establish whether there is an association between change in the mechanism variable and treatment outcome during treatment. Although an increasing amount of research

has shown that metacognitions are related to psychological disorders, it has not yet been reviewed if the change in metacognitive beliefs is correlated with symptom change during treatment. In addition, if metacognitive change is universally relevant for symptom change as suggested by the S-REF model, then these beliefs must change across different therapeutic interventions proven to be efficacious, even those that do not address these beliefs directly. The aim of this study is therefore to conduct a systematic literature review to evaluate whether change in metacognitive belief is associated with symptom change across different psychological disorders and across different therapeutic interventions. We hypothesize that all effective therapeutic interventions change metacognitions following treatment regardless of disorder, and that this change is associated with changes in treatment outcome. In studies with appropriate design, we would also expect to find that change in metacognitive beliefs act as a mediator of symptom change. In addition, we hypothesize that MCT shows larger changes in metacognitions compared to other treatments as MCT was specifically designed to create metacognitive change.

By summarizing the evidence of the importance of metacognitive beliefs in therapeutic change, we evaluate a central prediction of the S-REF model. Thus, our systematic review serves to evaluate an innovative model of psychological disorders. Further, identifying potentially influential metacognitions to specific disorders may contribute to better therapeutic targets in the future.

2 METHOD

We conducted a systematic literature search by the standards of Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) statement (Page et al., 2021). The PRISMA statement provides evidence-based recommendations designed primarily to encourage transparent and complete reporting of systematic reviews.

2.1 Search strategy

To reduce data omission, we identified studies through a triangulation of peer-reviewed databases using Scopus, PubMed, and PsycINFO. Two searches were included in the preliminary data extraction on February 2nd, 2022. These were "metacog* AND (therap* OR treatment)" in the title, keywords, and abstract, and "metacog* AND (MCQ* OR MWQ OR CAS-* OR TFI) AND (treatment OR therap*)" in full text. Whereas the first search intended to be broad, finding treatment studies related to metacognitive change in treatment, the intention of the second search was to specify and include the relevant questionnaires measuring metacognitive beliefs using their acronyms. Later in the screening process, another two suitable measures for metacognitive beliefs were identified and therefore included in a third search on March 28th, 2022: "metacog* AND (PBRS OR NBRS) AND (treatment OR therap*)" in full text. In our search specifications, we also excluded non-articles, such as books and pamphlets, publications in any language other than English, and publications dated prior to the publication of the first measure of metacognitive beliefs in 1997 (Cartwright-Hatton & Wells, 1997). In addition, articles only in the final publication (i.e. published or “in press”) stage were included.

2.2 Inclusion and exclusion criteria

Our inclusion criteria were articles that 1) measured both metacognitions and treatment outcome pre- and post-therapeutic intervention, 2) reported a relevant statistical analysis of the relationship between metacognitions and treatment outcome, such as change score correlations, regression analysis, mediation/moderation analysis, or a network analysis. Since we wanted the treatment outcome to be broad, we defined this as any primary outcome related to psychopathology. This includes for example neuroticism traits, which when high, are considered vulnerability markers for psychopathology.

We excluded articles that 1) did not measure metacognitions with MCQ-30/65, CAS-1, TFI, MWQ, PBRS, NBRS, or any other measurement that did not explicitly refer to the metacognitive model developed by Wells and Matthews (1994; 1996), 2) were case studies, 3) were conducted with children and adolescents. Although case studies may be relevant as they may show relationships between metacognitive change and symptom change over the course of treatment at the individual level, they do not report on statistics between variables and were therefore excluded here.

2.3 Procedure for study selection

Articles from our search were imported into the screening tool DistillerSR (www.evidencepartners.com). Duplicates were removed using the Artificial Intelligence for DistillerSR, and manually for the duplicates the AI did not detect. To reduce biases and errors in the process, both authors independently screened the abstracts of the remaining articles. In the abstract screening process, we included articles that mentioned metacognitions in relation to research on therapeutic interventions, which indicated a measurement of symptoms over the course of treatment. Articles we disagreed upon were discussed separately, and agreement was made with consensus. Although DistillerSR has many functions, we decided not to automate any part of the screening process. However, we used AI for a reverse search to detect omissions, where the AI searched for articles similar to those we had included in the screening process. After the exclusion of articles in the abstract screening process both authors did a full-text screening following the inclusion and exclusion criteria outlined above. The remaining articles were included in the review.

2.4 Qualitative synthesis

Our findings were synthesized with respect to two questions: 1) do metacognitive beliefs change over the course of different treatment modalities and disorders?, and 2) are these changes in metacognitive beliefs associated with changes in treatment outcome? Pre- and post-metacognitive belief scores change, and its association to treatment outcome variable(s) were extracted from the articles, including study design, therapeutic intervention(s) used, diagnoses, sample characteristics, measuring instruments for both metacognitive beliefs and outcome variable(s), and statistical analyses used in the studies.

For data extraction, articles were divided in two between both authors, where the data extraction process was followed by a correction process by the other author. Statistical data is reported in table 2 in the appendix. In text we report effect sizes of Pearson's and Spearman's correlations according to norms for psychological studies: 0.1 < weak < 0.4 < moderate < 0.7 < strong (Dancey & Reidy, 2007).

2.5 Quality Assessment

To evaluate the quality and possible limitations of our data material, we conducted a quality assessment of each article using The Quality Assessment Tool for Studies of Diverse Designs (Sirriyeh et al., 2012). It has 16 components scored on a 4-point scale, where two of them were excluded as they are only relevant for qualitative designs. Components in the assessment tool are focused on transparency and clarity in each step of the research process, considering themes such as theoretical frameworks, research setting, recruitment and representativeness of the sample, fit between aim, method of data collection and analysis, and self-reported limitations. The assessment tool has shown good inter-rater reliability (k = 71.5%) and test-retest reliability (Sirriyeh et al., 2012). Scores over 30 indicate good methodological robustness. Both authors did the assessment individually using the scoring guidelines provided by Sirriyeh et al. (2012) which were compared for inter-rater reliability with weighted kappa. To preserve the validity of our assessment, we decided to individually reassess subscales scores that showed a score discrepancy of 2 or above between raters. We found four such incidents. The scoring guidelines and complete assessment by both raters can be found in the appendix.

2.6 Included instruments for the assessment of metacognitive beliefs

We included instruments for assessing metacognitive beliefs that correspond to the S-REF model, of which all have been developed by Adrian Wells and colleagues. These instruments include general and diagnosis-specific measures of metacognitions. Measures described below that were not present in our initial search strategy were included after the screening process in line with the inclusion and exclusion criteria.

2.5.1 Metacognition Questionnaire (MCQ-30/-65)

The metacognitions questionnaire (MCQ) is considered the golden standard of self-reported measurement of general metacognitive beliefs related to the metacognitive model of psychopathology. There are two different versions, one consisting of 65 items (Cartwright-Hatton, S., & Wells, A. 1997) and a shorter version consisting of 30 items (Wells & Cartwright-Hatton, 2004). The MCQ is composed of five subscales: 1) positive beliefs about worry (PB; e.g. “Worrying helps me cope”), 2) negative beliefs about the uncontrollability and danger of worry (NB; e.g. “When I start worrying I cannot stop”), 3) cognitive confidence (assessing confidence in memory and attention; CC; e.g. “I have poor memory”), 4) need to control thoughts (NCT; e.g. “Not being able to stop my worrying is a sign of weakness”), and 5) cognitive self-consciousness (CS; e.g. “I pay close attention to the way my mind works”). Scores range from 1 ("I do not agree") to 4 ("I agree very much"). The MCQ-65 has shown good internal consistency, with Cronbach's alpha ranging from 0.72 to 0.89 and test-retest reliabilities ranging from 0.76 to 0.89 (Cartwright-Hatton, S., & Wells, A. 1997). The short version, MCQ-30, has shown similar internal consistency and validity, with Cronbach's alpha ranging from 0.72 to 0.93 and test-retest correlations ranging from 0.75 to 0.87 (Wells & Cartwright-Hatton, 2004).

2.5.2 CAS-1

Cognitive attentional Syndrome questionnaire (CAS-1) is a self-descriptive measurement for assessing the severity of CAS (Wells, 2009). The questionnaire consists of 16 items scored on a scale from 0 ("None of the time") to 8 ("All of the time"), loading onto three subscales: 1) metacognitive strategies, such as worry/rumination, threat monitoring and coping behavior, 2) positive metacognitive beliefs, and 3) negative metacognitive beliefs (Wells, 2009, *p.* 268). The latter two subscales are included as indicators of dysfunctional metacognitions in our thesis. Here, the items regarding negative metacognitive beliefs are "Worrying too much could harm me," "Strong emotions are dangerous," "I cannot control my thoughts," and "Some thoughts could make me lose my mind." Items regarding positive metacognitive beliefs are "Worrying helps me cope," "Focusing on possible threats can keep me safe," "It is important to control my thoughts," and "Analyzing my problems will help find my answer" (Wells, 2009, *p.* 268). CAS-1 has shown good internal consistency (Cronbach's $\alpha = .87$) and validity (Nordahl & Wells, 2019).

2.5.3 Negative Beliefs about Rumination Scale (NBRS)

Negative Beliefs about Rumination Scale measures negative metacognitive beliefs about the uncontrollability and harm of rumination (Papageorgiou & Wells, 2001a). NBRS has 13 items scored on a 4 point-likert scale. NBRS includes statements such as "Rumination can make me physically ill" or "If I did not ruminate about my feelings, I wouldn't be able to control them/I could end up harming myself.". It also has questions centering metacognitive beliefs about the interpersonal implications of rumination, such as "Only weak people ruminate" and "Nobody wants to be with people who ruminate all the time". NBRS shows good internal consistency with Cronbach's alphas ranging from 0.80 to 0.83 (Luminet et al., 2004).

2.5.4 Positive Beliefs about Rumination Scale (PBRS)

Positive Beliefs about Rumination scale (PBRS) assess metacognitive beliefs about the benefits of rumination (Papageorgiou & Wells, 2001b). PBRS has 9 items scored on a 4 point-likert scale. PBRS includes questions such as "Ruminating about my feelings helps me to recognize the trigger of my depression," "I need to ruminate about the bad things that have happened in the past to make sense of them," or "I need to ruminate about my problems to find answers to my depression". Luminet et al. (2004) document good psychometric properties with a Cronbach's alpha of 0.89.

2.5.5. OCD-specific measures of metacognitive beliefs

The Thought-Fusion Instrument (TFI) is a 14-point inventory in which each item is scored from zero ("I do not believe this at all") to a hundred ("I am completely convinced that it is true"; Wells et al., 2001). TFI is designed to measure fusion beliefs about the meaning, significance, and danger of intrusive thoughts. This includes beliefs of what power thoughts may inherent, such as "If I think about an unpleasant event, it is more likely to happen" or "If I think about harming someone, it will harm him or her" (Wells, 2009, *p.* 265). TFI has shown good internal consistency (α = .89; Melchior et al., 2021).

The Beliefs about Rituals Inventory (BARI) is a 12-items questionnaire that assesses positive beliefs about the necessity of performing rituals (McNicol & Wells, 2012). The items consist of questions that include “I need to perform rituals otherwise…”. This can be “... I will never have peace of mind”, or “I will lose control of my thoughts" (McNicol & Wells., 2012). Recent studies found BARI to have excellent internal consistency in a clinical population (Cronbach's α = 96; Melchior et al., 2021).

The Stop Signals Questionnaire (SSQ) assesses the importance of certain criteria in deciding to stop carrying out rituals (Myers, 2009). It consists of 12 questions that are investigating how important each of the signals are for stopping their rituals on a likert scale from 0 ("Not at all important") to 4 ("Extremely important"). The questions start with "An important signal of when I can stop my rituals is when…" and end with for example "... I have performed my rituals in the correct order" or "I have replaced the intrusive thoughts with a positive image." Internal consistency for SSQ was considered good ($\alpha = 0.89$; Myers, 2009).

3. RESULTS

The study selection process is depicted in figure 1. Overall, the searches yielded a total of 4230 studies. One-thousand two-hundred and ten duplicates were removed which resulted in 3020 articles for abstract screening. This process resulted in 2788 articles being excluded. As for the reverse AI search mentioned above, no new articles were detected. In the final step, screening the full text of 232 articles, 206 articles were excluded for several reasons: 9 articles were not available through NTNU's license; 35 studies did not have appropriate design; 96 studies lacked appropriate measurement of metacognitive beliefs; and 66 studies did not have the appropriate statistical analysis of the relationship between metacognitive beliefs and symptom outcome. This resulted in 26 articles that were included in the review.

We sorted studies by diagnosis to provide a comparable overview of the relative importance of metacognitive beliefs on treatment outcome. Of the 26 articles included, three articles were related to unipolar depression disorder (Hjemdal et al., 2019; Jelinek et al., 2017; Sürig et al., 2021); six articles were related to anxiety disorders (Hoffart et al., 2018; Johnson & Hoffart, 2018; Johnson et al., 2020; McEvoy & Perini, 2009; McEvoy et al., 2009; Nordahl et al., 2017); seven articles were related to obsessive-compulsive disorder (Besiroglu et al., 2011; Grøtte et al., 2015; Hansmeier et al., 2021; Park et al., 2020; Solem et al., 2009; Solem et al., 2015; Sunde et al., 2021); four were related to fatigue (Brugnera et al., 2021; Fernie et al., 2015; Jacobsen et al., 2016; Jacobsen et al., 2020); one was related to high risk of developing psychosis (Parker et al., 2020); one was related to alcohol abuse (Spada et al., 2009); one was related to trait anxiety (McEvoy et al., 2017); one was related to fear of recurring cancer (Sharpe et al., 2019); and two were related to mixed anxiety and depression (Corpas et al., 2021; Newby et al., 2014).

As the studies were too diverse to conduct a meaningful quantitative synthesis, we adjusted the PRISMA statement to fit a literature synthesis, excluding guidelines for statistical assessments (e.g. meta-analytical methods).

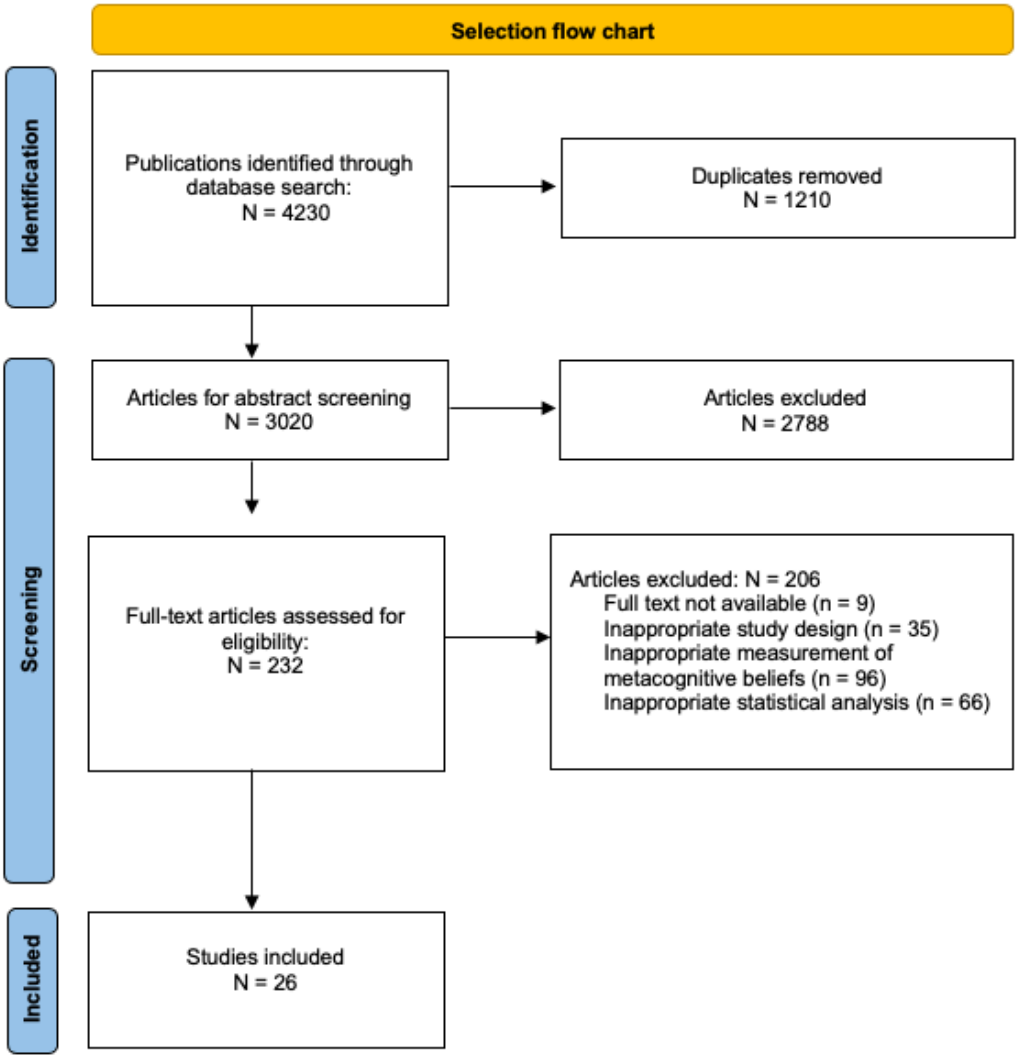

Figure 1: Selection flow chart

3.1 Quality assessment

In general, the studies were limited by low sample size, and few had evidence of sample size considered in terms of analysis. The included articles' quality scores varied from 20 to 36 out of a total score of 42. Most of the scores varied around 30, indicating that the studies were generally of high quality. We identified one article in particular, by Besiroglu et al (2011), that both authors rated significantly lower than the rest. This was mainly due to the lack of transparency of the recruitment process, data collection and research setting, lack of representative sample of reasonable size and insufficient discussion of limitations. There was

a strong consistency between the author's quality assessment scores, with a weighted kappa of .89.

3.2 Primary depression

Three articles investigated the association between change in metacognitive beliefs and symptom outcome in patients with a primary unipolar depression diagnosis.

#1 Jelinek and colleagues (2017) assessed metacognitive training for depression (D-MCT; Jelinek et al., 2015, for english version see Jelinek et al., 2022) in a RCT study. Eighty-four participants diagnosed with major depressive episode, recurrent depression, or dysthymia (74 % female) were randomly allocated to D-MCT or health training (HT) as an active control group. The MCQ-30 subscales MCQ-30-PB, MCQ-30-NB, and MCQ-30-NCT were included, as they were assumed to be most related to depression. Hamilton Depression Rating Scale (HDRS; Hamilton, 1960) and Beck Depression Inventory (BDI; Beck et al., 1961) were used to measure depressive symptoms. They found a significant decrease in all three subscales following D-MCT compared to the active control group, with effect sizes from medium to large. The authors conducted a mediation analysis using Hayes' PROCESS (Hayes, 2009). In the mediation analysis, change in MCQ-30-NCT were the only subscale that were a significant mediator of long-term reduction in depressive symptoms. The mediation was partial with medium effect size.

#2 Hjemdal and colleagues (2019) did a one-year follow-up from an RCT study in which thirty-nine participants (59 % female) diagnosed with depression had been divided into two conditions: ten sessions of manualized MCT for depression (Wells, 2009) or waiting list. MCQ-30, NBRS, and PBRS were used to measure metacognitive beliefs, BDI were used to assess depressive symptoms, and Beck Anxiety Inventory (BAI; Beck & Steer, 1990) were used to assess anxiety symptoms. There was a significant change in metacognitive beliefs from pre- to post-treatment, six-month and 1-year follow-up. They found a moderate significant correlation between change in MCQ-30 total score and change in depressive symptoms at 1-year follow-up. Both PBRS and NBRS also showed a significant moderate correlation with BDI. Similarly, weak to moderate correlations between MCQ-30 total score, PBRS and NBRS paired with BAI was found. They also conducted a multiple hierarchical

regression analysis, where change in MCQ-30 total score emerged as a significant predictor for change in BDI from pre-treatment to 1-year follow-up. Subscales of MCQ-30 were not addressed.

#3 Sürig & colleagues (2021) assessed the association between metacognitive belief change and change in depressive symptoms in two treatment groups. Ninety participants admitted from a day treatment program for depression were given either Cognitive Behavioral Analysis System of Psychotherapy (CBASP; Schweiger et al., 2019; 41 % female) or MCT (Wells, 2009; 43 % female). The choice of treatment was a shared decision between the patients and clinicians. Metacognitive beliefs were measured using MCQ-30, and depressive symptoms by using Quick Inventory of Depressive Symptomatology (QIDS-SR16; Rush et al., 2003). They found a significant decrease in metacognitive beliefs in both treatment groups during the course of treatment. The largest decrease of metacognition was found in the MCT group. The authors conducted a hierarchical multiple regression analysis to investigate the relationship between metacognitive beliefs and depressive symptoms. Improvement in MCQ-30 total score did not significantly predict symptom change. Rerunning the analysis with only MCQ-30-NB yielded the same results.

3.3 Primary anxiety

A total of six studies investigated the relationship between metacognitive beliefs and symptom change for patients diagnosed with a primary anxiety disorder.

#4 A study by Hoffart and colleagues (2018) investigated the within-person relationship between metacognitive beliefs over the course of either MCT or CBT treatment. Seventy-four participants (61 % female) referred for treatment were recruited in the study. The participants had to meet the inclusion criteria for a principal DSM-IV disorder, equal to or greater than four on the clinical severity rating, of PTSD, social anxiety disorder, or panic disorder with or without agoraphobia. There was a weekly assessment of metacognitive beliefs using CAS-1 and anxiety symptoms using BAI. The weekly scores of BAI and CAS-1 were regressed on time and treatment in mixed models. Their results showed that positive and negative metacognitive beliefs decreased more throughout treatment in the MCT group than in the CBT group. Using linear mixed-effects models, the authors found a within-person

relationship between positive metacognitive beliefs and subsequent anxiety. There was also a trend that negative metacognitive beliefs predicted subsequent anxiety. When examining reversed within-person relationships, it turned out that anxiety predicted subsequent positive metacognitive beliefs but not negative metacognitive beliefs. Analyzing separate treatment groups revealed a significant within-person relationship between anxiety and subsequent positive metacognitive beliefs in CBT, but not in MCT, and a significant between-person relationship between the level of anxiety and level of positive metacognitive beliefs in MCT, but not in CBT.

#5 Based on data from the same trial as used by Hoffart and colleagues (2018) described above, Johnson and Hoffart (2018) also conducted a network analysis to investigate the potential mechanisms involved in treating anxiety with MCT and CBT. Here, the relationships between anxiety and metacognitions following MCT and CBT was investigated using a Multilevel vector autoregressive (mlVAR) model. This analysis' primary function is to address how different concepts are interrelated, viewing symptoms as mutually interacting, often reciprocally reinforcing, elements of a complex network. Specific items were selected to capture key processes related to CBT, MCT and symptoms of anxiety and depression. Symptoms of anxiety were selected from BAI, symptoms of depression were selected from Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001) while metacognitions were selected from CAS-1.

The study produced three different networks: The temporal network, which shows the average within-person effects from one week to the next, the contemporaneous network which captures the average within-person association at the same measurement point, and the between-person network which shows the partial correlation between person-means. In the MCT temporal network, but not the CBT temporal network, "negative belief about the uncontrollability of thoughts" predicted "threat-monitoring". In the MCT contemporaneous network, "beliefs about the uncontrollability of thought" is a central node, connected to “feeling depressed”, “taking little interest in things”, "threat monitoring" and negatively connected to “shakiness”. In the CBT contemporaneous network, "negative beliefs about the uncontrollability of thought" was a less central node but connected to “fear of dying” and “sleeping issues”. In the between-person MCT network, "negative belief about uncontrollability of thoughts” was connected to "threat monitoring", whereas it was a central node in the between-person CBT network, connecting to “feeling depressed”, “fear of losing

control" and negatively to "sleeping issues". "Worry" and "threat-monitoring" from CAS-1 were also central nodes across all three types of networks.

#6 Johnson & colleagues (2020) investigated if cytokines could be a moderator between metacognitive beliefs and anxiety over the course of therapy in comorbid anxiety disorders. Cytokines constitute a group of small messenger molecules that may alter the metabolism of neurotransmitters, hence potentially influencing psychological changes. The study included thirty-seven patients (54 % female), which were given either MCT or CBT. Metacognitive beliefs were measured using MCQ-30, and anxiety was measured using BAI. Peripheral circulating cytokines were measured in pre-, middle, and end-of-treatment. In their statistical analyses treatments were not separated, thus results encompass both. Changes in metacognitions from pre- to post-treatment were not reported. MCQ-30 total score predicted anxiety symptoms on the between-person and the within-person level. At the within-person level, a decrease in MCQ-30 total score in a given week was associated with reduction in BAI the following week. At the between-person level, lower levels of metacognitive beliefs predicted lower levels of overall anxiety symptoms. Cytokine levels did not moderate these effects. Subscales of MCQ-30 were not addressed.

3.2.1 Social Anxiety

#7 McEvoy & Perini (2009) investigated whether supplementing standard Cognitive Behavioral Group Therapy (CBGT; McEvoy, 2007) with attention training technique (ATT; Wells, 1990; vs. relaxation training (RT)) resulted in greater changes in patients with social phobia. Measures of symptom outcome were BDI, Social Phobia Scale (SPS; Mattrick & Clarke, 1998) and Social Interaction and Anxiety Scale (SIAS; Mattrick & Clarke, 1998). Eighty-one participants (37 % female) with social phobia were included in the study. All subscales on MCQ-30 except MCQ-PB changed during treatment, but the condition by time interaction was insignificant for all scores, indicating that metacognitions did not change more with ATT than with RT. Change scores in MCQ-30-NB were significantly correlated with reductions in all three symptom measures. This correlation was weak for change scores in SPS and SIAS, and moderate for change scores in BDI. MCQ-30-CC and MCQ-30-NCT showed significant weak correlations with change scores in BDI, but not with SPS or SIAS.

#8 In a similar study, McEvoy & colleagues (2009) examined the relationship between social anxiety, post-event processing (PEP), depression and metacognitive beliefs in

a clinical sample with social phobia following CBGT. Sixty-one participants (34 % female) were included in the study. Metacognitive beliefs were measured with MCQ-30, social anxiety with SPS and SIAS, and depression with BDI. MCQ-30-total- and subscale scores were moderately reduced from pre- to post-treatment, except for MCQ-30-CC in which the effect was considered weak, and MCQ-30-PB in which no significant change was found. Change scores in MCQ-30 total or subscale scores were not significantly correlated with change scores in SPS, and only change in MCQ-30-NB was weakly correlated with change in SIAS. Changes in the subscores in MCQ-30-NCT, MCQ-30-CC and MCQ-30-NB were all moderately significant correlated with a reduction in BDI. There was a weak significant correlation between reduction in the subscales MCQ-30-NB and MCQ-30-NCT and reduction in PEP. The study concludes that metacognitive beliefs were generally associated with a reduction in depression and not social anxiety.

#9 Nordahl & colleagues (2017) investigated the association between negative metacognitive beliefs in forty-six participants (48 % female) diagnosed with social anxiety disorder treated with CT based on the Clark and Wells model (1995), SSRI, or the combination of both treatments. Metacognitive beliefs were measured with MCQ-30-NB as this was regarded as the most important in social anxiety disorder. Symptoms of social anxiety was operationalized using the Fear of Negative Evaluation Scale (FNE; Watson & Friend, 1969), the Liebowitz Social Anxiety Scale (LSAS; Liebowitz, 1987), the Social Avoidance and Distress Scale (SAD; Watson & Friend, 1969), and the Social Interaction Anxiety Scale (SIAS). The study combined all three treatment conditions in the analysis. Paired sample *t*-tests showed a medium effect size, indicating that MCQ-30-NB changed during treatment. Hierarchical multiple linear regression analyses were conducted, in which negative metacognitive beliefs explained a significant additional 15.9 % of the variance in FNE, 5.9 % of the variance in LSAS, 12.9 % in SAD, and 10.3 % of the variance in SIAS. MCQ-30-NB were the only consistent predictor across all outcome variables.

3.4 Primary obsessive-compulsive disorder

We identified seven articles with samples of OCD as the primary diagnosis, which examined the relationship between metacognitive beliefs and symptom change.

#10 Solem et al. (2009) examined metacognitions using MCQ-30 in an OCD sample of eighty-three outpatients (71 % female) undergoing Exposure and Response Prevention (ERP) treatment (Foa & Kozak, 1997). OCD-symptoms were assessed using Yale-Brown Obsessive compulsive scale (Y-BOCS; Goodman et al., 1989), and "clinical significant change" in OCD-symptoms were defined as having post-treatment score below 14 and a change of at least ten on Y-BOCS, and "reliable change" were defined as having post-treatment score above 14, but a change of at least ten on Y-BOCS. They found that all metacognitive beliefs decreased significantly following treatment, where MCQ-30-NB and MCQ-30-NCT had the highest change scores. Patients who achieved clinical significant change in OCD symptoms had significantly lower MCQ-30 total score at post-treatment compared to patients who achieved reliable change and patients who did not achieve change. Furthermore, there were weak to moderate correlations between change in all MCQ-30 subscores and Y-BOCS. This relationship was moderate for MCQ-30 total score. Subsequent regression analysis showed that MCQ-30 total score and MCQ-30 subscales NCT and PB predicted change in OCD symptoms following treatment.

#11 Besiroglu et al. (2011) investigated the effect of 12-16 weeks SSRI-treatment on depressive and OC-specific symptoms and metacognitions in a sample of fifty-five patients (58 % female) with OCD. OCD-symptoms were measured with Y-BOCS and depressive symptoms were measured with BDI. MCQ-30 was used to measure metacognitive beliefs, and Thought-Action Fusion scale (TAF; Shafran et al., 1996) was used to measure OC-specific metacognitions. Although not developed with reference to the S-REF model, it has similarities with TFI, and assesses the beliefs that having unwanted intrusive thoughts would increase the likelihood of adverse effects (TAF-Likelihood) or the belief that these intrusive thoughts would be morally equivalent to carrying out the act (TAF-Morality). They found that MCQ-30 total score and TAF-morality were reduced following SSRI-treatment, but not TAF-likelihood. Change in MCQ-30 total score correlated moderately with change in the subscale Y-BOCS-obsessions and BDI. Both TAF change scores showed a significant weak correlation with Y-BOCS-obsessions change scores, while only changes in TAF-morality correlated with changes in BDI, which was moderate. Only weak trends emerged for changes in metacognitions and changes in Y-BOCS-compulsion. In their final regression analysis, change in metacognitions did not predict changes in Y-BOCS after treatment. Subscales of MCQ-30 were not mentioned.

#12 Grøtte et al. (2014) included one hundred and eight OCD patients (69 % female) in their study, who completed an intensive 3 week multimodal treatment package consisting of behavioral, cognitive, and metacognitive elements, including ERP as the main ingredient. OCD-specific metacognitive beliefs were measured with Thought Fusion Instrument (TFI) and Beliefs about Rituals Inventory (BARI), OCD-symptoms were measured with self-report Y-BOCS and The Obsessive-Compulsive Inventory-Revised (OCI-R; Foa et al., 2002). "Clinical significant" and "reliable change" in Y-BOCS were assessed with the same cut-offs and change index as previously referred to in Solem & colleagues (2009). In OCI-R, clinical cutoff was set to 21 and reliable change index was set to 12. Their results showed that OCD-specific metacognitive beliefs significantly reduced after treatment. OCD-specific metacognitive beliefs were also correlated with both OCD symptoms and depressive symptoms at both pre-and post-treatment, which were moderate to strong at post-treatment. However, change scores correlations were not reported. In their logistic regression analysis with a clinical significant change in Y-BOCS as the dependent variable, TFI and BARI did not emerge as significant predictors. When using reliable change indices, reliable change in TFI and BARI emerged as significant predictors of Y-BOCS score following treatment. This analysis was also repeated, measuring OCD-symptoms with OCI-R as the dependent variable. Here, TFI and BARI emerged as predictors for clinical significant change in OCI-R, while only BARI emerged as a predictor of reliable change indices in OCI-R.

#13 In a RCT study by Solem et al. (2015), health anxiety symptoms were examined in relation to OCD in a sample of three hundred and thirteen patients (65 % female) with a community control of 382 (55 % female). Health anxiety symptoms were assessed using The Whiteley Index (WI; Pilowsky, 1967), OCD-symptoms were assessed with Y-BOCS and depressive symptoms with BDI. Metacognitions were also examined by using MCQ-30. Results showed medium to large effect sizes in reducing metacognitions following ERP treatment, where MCQ-30-NB and MCQ-30-CC showed the greatest reduction. Changes in MCQ-30 total were moderately correlated with both changes in health anxiety symptoms as well as changes in OCD-symptoms, and changes in depressive symptoms. All MCQ-30 subscores showed the same pattern, ranging from weak to moderate correlations, where MCQ-NB showed the strongest correlation with OCD-symptoms and health anxiety symptoms. In the regression analysis, changes in MCQ-30-CC were the only predictor of health anxiety post-treatment. Regression analyses with Y-BOCS or BDI as dependent variables were not assessed.

#14 In a study by Park et al. (2020) metacognitions were investigated in relation to the early response (4 weeks) to SSRI treatment in a sample of one hundred and thirty-two OCD patients (46 % female). Y-BOCS and MCQ-65 were used to measure OCD-symptoms and general metacognitions respectively. Early responders were defined as more than 20 percent improvement on Y-BOCS. Results showed that early responders following SSRI-treatment showed a significantly lower score on MCQ-65-PB compared to non-responders but not on other subdimensions of MCQ-65. Their regression analysis revealed MCQ-65-PB as a significant predictor of early treatment response and for predicting Y-BOCS reduction. Partial correlation analysis with age as a covariate was also conducted between the subdimensions of change scores in MCQ-65 and Y-BOCS. Again, only MCQ-65-PB showed significant results, where a weak correlation with the improvement of Y-BOCS was found.

#15 In a longitudinal study by Sunde et al. (2021) forty patients (78 % female) were assessed over eight years following treatment for OCD. Participants received group Exposure and Response Prevention (ERP; Himle et al., 2001). Metacognitions were measured using MCQ-30 and OCD-symptoms with Y-BOCS. They found that MCQ-30 total score significantly reduced over the course of treatment and follow-up. MCQ-30 total score were also assessed at both between- and within-person levels to determine if they could predict Y-BOCS. Results showed a significant between-person but not within-person effect. They also did a reversed analysis indicating that Y-BOCS were significantly predictive of MCQ-30-total score at the between-person level but not at the within-person level, indicating a reciprocal relationship between Y-BOCS and MCQ-30 total score at the between-person level. The same analysis was conducted by adding OCD obsessive cognitive beliefs measured with Obsessive beliefs questionnaire (OBQ-44; OCCWG, 2005) to the analysis. Here, MCQ-30 total score at the between-person level still emerged as a predictor of Y-BOCS, while OBQ-44 did not. Subsequently, the subscales of MCQ-30 were investigated as predictors of change in Y-BOCS over the course of therapy and follow-up. Whereas MCQ-30-PB and MCQ-30-NB predicted Y-BOCS at the between-person level, only MCQ-30-NCT predicted Y-BOCS at both between- and within-person level. The MCQ-30-CC and MCQ-30-CS did not emerge as significant predictors of Y-BOCS.

#16 In a pilot RCT, Hansmeier and colleagues (2021) compared the effect of MCT and exposure and response prevention (ERP) in the treatment of twenty-four OCD patients

(63 % female). In their study, they used clinician-rated Y-BOCS and self-rated Palatine Revision of the Padua Inventory (PI-PR; Gönner et al., 2010) to measure OCD-symptoms. OCD-specific metacognitions were measured using TAF, BARI, and the Stop Signals Questionnaire (SSQ), in addition to MCQ-65 which were used to assess general metacognitive beliefs. They found that both treatments significantly changed all OCD-specific metacognitions from pre- to post-treatment, while changes in MCQ-65 were not reported. Only TAF showed a significant group by time interaction, showing a stronger reduction in TAF following MCT than ERP. Furthermore, they assessed correlations between change scores in metacognitions and OCD-symptoms. At post-treatment there was a significant moderate correlation between change in BARI and change in Y-BOCS, but not for other measures of metacognitive beliefs. There was also moderate significant correlations between change in both SSQ and MCQ-65-total score and change in PI-PR. The correlation between change in SSQ and change in PI-PR was also significant at follow-up. There was no significant correlation between BARI change scores and change in PI-PR. In their final analysis, they included significant variables into a regression model. In the Y-BOCS model, there was only a trend for the additional block of changes in BARI at post-treatment and follow-up. In the PI-PR model, SSQ emerged as the only predictor of OCD symptoms at post-treatment and follow-up.

3.5 Other categories of diagnoses or syndromes

3.5.1 Chronic fatigue

We identified four articles related to chronic fatigue, one of which also included patients with chronic pain and/or mental distress (Jacobsen et al., 2020).

#17 Fernie et al. (2015) compared CBT with Graded Exercise Therapy (GET; Fulcher, 1997) as treatment of patients with chronic fatigue syndrome in one hundred and forty-eight patients (gender distribution N/A). Fatigue severity was measured with Chalder Fatigue Questionnaire (CFQ; Chalder et al., 1993), and metacognitions were measured with MCQ-30. Differences in metacognitions following treatment were not reported. In their regression model, changes in both MCQ-NB and MCQ-CC significantly predicted fatigue levels, regardless of treatment modality. These metacognitions were according to the authors chosen based on a correlation matrix that explored the relationship between change in fatigue scores

and change in metacognitions between pre-treatment and follow-up, but this matrix was not reported in the article.

#18 Jacobsen et al. (2016) investigated subjective memory problems, a hallmark symptom of chronic fatigue, and its relation to metacognitive beliefs. In a rehabilitation center in Norway, one hundred and thirty-seven patients (80 % female) on sick leave due to chronic fatigue received a 3.5-week inpatient return-to-work program with ACT as the overarching treatment model (Fimland et al., 2014). Subjective memory problems were assessed with The Everyday Memory Questionnaire-Revised (EMQ; Royle & Lincoln, 2008). Metacognitive beliefs were assessed with MCQ-30. Their results showed a significant decrease in MCQ-30-CC following treatment. However, MCQ-30-NB showed a significant increase following treatment. In their regression analysis, reductions in MCQ-30 total score were a significant predictor of reduced post-treatment score of EMQ, controlling for pre-treatment MCQ-30 total score and pre-post scores of fatigue, pain, insomnia severity, anxiety, and depression. As pre-treatment MCQ-30-CC was associated with pre-treatment and post-treatment scores on EMQ, they repeated the analysis by looking at this subscale. Post-treatment MCQ-30-CC then emerged as an independent predictor of post-treatment scores on EMQ. Regression analyses with other symptoms of chronic fatigue were not included.

#19 Jacobsen et al. (2020) conducted a study at the same occupational rehabilitation center with a 3.5-week return-to-work inpatient program with ACT. This study included one hundred and thirty-seven long-term sick leave patients (81 % females) with chronic fatigue, chronic pain, and/or mental distress. Primary outcome was a dichotomous evaluation of (re)entry to the ordinary workforce from baseline and up to 56 weeks after discharge. Metacognitions were measured with MCQ-30. They found a significant reduction in MCQ-30 subscores for both those who returned to work and those who did not return to work after treatment, except for MCQ-30-PB and MCQ-30-CS. Changes in MCQ-30 total score were significantly associated with an increased odds ratio for returning to work. When looking at the subscales, only the MCQ-30-NCT was significant, in which a 1-point change in score was associated with a 20 % higher odds ratio for returning to work.

#20 Another study was conducted by Brugnera et al. (2021) at the rehabilitation center. They conducted a longitudinal study with one hundred and ninety-five patients

diagnosed with chronic fatigue (81 % female) where they received a 3.5-week inpatient return-to-work program with ACT. Follow-up was assessed 6 and 12 months post-treatment. A range of variables were included. Chronic fatigue symptoms were measured with CFQ; anxiety and depression symptoms were assessed with Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983); health-related quality of life were measured with SF-8 Health Survey (SF-8; Ware et al., 2001); and metacognitions were assessed with MCQ-30. Other process variables such as avoidance and psychological inflexibility, along with cognitive and behavioral responses to chronic fatigue, were included. Work status was also addressed. In their study, metacognition scores decreased significantly from pre- to post-treatment. When process variables were entered one at a time in different regression models, a greater decrease in metacognitive beliefs predicted a steeper decline in fatigue symptoms and lower levels of anxiety and depression symptoms. Furthermore, MCQ-30-NCT predicted a steeper increase in mental health-related quality of life. In addition, MCQ-30-PB predicted the odds of working more hours per week at the end of treatment. When all process variables were entered together in the regression models, only one metacognitive predictor of symptom change remained significant, namely MCQ-30-NB as a predictor of anxiety.

3.5.2 High risk psychosis

#21 We found one article, a pilot study by Parker and colleagues (2020), investigating the role of metacognitive change in worry for patients with risk of developing psychosis. Ten participants (40 % female) who met the criteria for ultra high risk of developing psychosis were included in the study and were given 12 sessions of MCT treatment. General metacognitions were measured using MCQ-30, while psychosis-specific metacognitions were assessed with Interpretations of Voices Inventory (IVI; Morrison et al., 2002) and Beliefs about Paranoia Scale - short form (BAPS; Gumley et al., 2011). Levels of worry and threat-monitoring were included using CAS-1. MCQ-30 total scores were significantly reduced after 12 weeks of treatment and at six months follow-up, as were the subscales MCQ-30-NB MCQ-30-CC, and MCQ-30-NCT. With clustering at the participant level, regression analysis suggested that the strength of metacognitive beliefs significantly predicted levels of worry across sessional measures. No analysis of the association between metacognitive beliefs and psychotic symptoms was included in the study.

3.5.3 Alcohol addiction

#22 We included one article which investigated metacognitive change and change in symptoms of alcohol addiction by Spada and colleagues (2009). Seventy individuals (31 % female) seeking treatment for problem drinking were given CBT and assessed at pre-treatment and 3, 6, and 12-month post-treatment follow-up. Alcohol consumption levels were measured using Quantity Frequency Scale (QFS; Cahalan et al., 1969), and metacognitions were measured using MCQ-65. Weak to moderate correlations were found between MCQ-65-NCT and weekly alcohol use at 3, 6, and 12 months. They also found a weak correlation between MCQ-65-CC and weekly alcohol use at 3 months, a weak correlation between MCQ-65-NB and levels of alcohol use at 6 months, and a moderate correlation with MCQ-65-CC and alcohol use at 6 months. In their regression analysis MCQ-30-NCT was the only significant predictor for drinking status (absence or presence of drinking) at both 3- and 6 months follow-up, and for a level of weekly alcohol use at 3, 6, and 12 months.

3.5.4 Trait anxiety

#23 In a randomized controlled study, McEvoy et al. (2017) compared Attention Training Technique (ATT; Wells., 2005) deriving from metacognitive therapy with Mindfulness-Based Progressive Muscle Relaxation (MB-PMR; Orsillo & Roemer, 2011) to a thought wandering control (TWC). They wanted to examine each condition regarding their impact on anxiety and mechanisms of change, including the subscale MCQ-30-NB. The sample consisted of eighty-one high-trait anxious participants (80 % female) evenly divided between the groups. The intervention lasted for 12 minutes in each condition. Consequently, outcome measures of MCQ-30-NB were adjusted to a modified version (UTS) to assess MCQ-30-NB in a short time frame, asking patients how much they agreed on each item if they were to start worrying "right now". State anxiety symptoms were assessed with State-Trait Inventory for Cognitive and Somatic Anxiety (STICSA-S; Ree et al., 2008). Their results showed that both ATT and MB-PMR decreased metacognitions post-manipulation, along with anxiety levels. There were no significant differences between the two groups, and these groups were thus combined for subsequent analysis. A series of indirect effects models were conducted, which showed that UTS were significantly associated with lower anxiety post-manipulation.

3.5.5 Fear of recurring cancer

#24 One study by Sharpe et al. (2019) investigated the effect of ConquerFear treatment versus relaxation training control in a sample of one-hundred and fifty-two survivors (98 % female) of breast cancer, colorectal cancer and melanoma with fear of cancer recurrence. ConquerFear consists of elements from metacognitive therapy and acceptance and commitment therapy, including attention training technique and challenging metacognitions. Fear of cancer recurrence was measured with Fear of Cancer Recurrence Inventory (FCRI; Simard & Savard, 2009) and metacognitions were assessed with MCQ-30. Results showed that patients in the ConquerFear group reported greater reductions in MCQ-30 total score compared to the control group. In order to assess the relationship between metacognitive beliefs and treatment efficacy on fear of cancer recurrence, a moderation and mediation analysis was conducted. They found that metacognitions did not moderate treatment efficacy in terms of FCRI. However, the mediation model showed that MCQ-30 total score predicted FCRI at follow-up. Treatment group still predicted FCRI when mediators were included, which indicated that metacognitive beliefs as a mediator on treatment outcome were partial. Subscales on MCQ-30 were not reported.

3.5.6 Mixed symptoms of anxiety and depression

#25 A RCT study by Newby & colleagues (2014) explored the role of metacognitive beliefs in a mixed anxiety and depression sample. Ninety-nine participants (78 % female) with a generalized anxiety disorder (GAD) and/or major depressive disorder (MDD) received either a transdiagnostic internet-delivered CBT (iCBT; see article) or were placed in waitlist control. Metacognitive beliefs were measured using PBRS, and outcome variables were PHQ-9 and GAD-7 (Spitzer et al., 2006). Repetitive negative thinking was also addressed using the Repetitive Negative Thinking Questionnaire (RTQ; McEvoy et al., 2010). PBRS were significantly lower at post-treatment and at 3-month follow-up. In order to establish temporal precedence of mediators on treatment outcome, mediation models tested the pre- to mid-treatment scores of PBRS and RNT against post-treatment outcome. Results indicated that reductions in PBRS mediated the impact of iCBT on post-treatment depression symptoms by influencing reductions in RNT frequency. For anxiety symptoms, the results suggested that a reduction in PBRS improved anxiety symptoms but not via a reduction in RNT.

#26 Corpas & colleagues (2022) investigated if negative metacognitive beliefs predicted therapeutic change in a population of participants with mild to moderate symptoms of somatoform anxiety and/or depression. One-hundred and five participants (67 % female) were randomly allocated to either receiving brief group transdiagnostic therapy (BGTP; see article) or pharmacological therapy with antidepressants and anxiolytics. Outcome variables consisted of GAD-7 for GAD-symptoms, PHQ-9 for depression symptoms, PHQ-PD for panic disorder symptoms (Spitzer et al., 1999), PHQ-15 for somatoform disorder symptoms (Kroenke et al., 2002) and Structured Clinical Interview for DSM-5 (First et al., 2015) with focus on the relevant disorders just mentioned. Negative metacognitive beliefs were measured with the subscale MCQ-30-NB, while other process variables included measures of emotion regulation and worry. BGTP showed a significant decrease in MCQ-30-NB, while pharmacological treatment did not. When all process variables were included in their regression model, MCQ-30-NB predicted change in panic disorder symptoms, but was not a significant predictor of the other outcome variables.

4. DISCUSSION

In this systematic literature review, we set out to evaluate the claim proposed by the S-REF model (Wells & Matthews, 1994; 1996) that metacognitive change are important factors of therapeutic change across psychological disorders and irrespective of treatment modality. We had two hypotheses: 1) all effective therapeutic interventions would change metacognitive beliefs following treatment, across all psychological disorders, and 2) change in metacognitions would be associated with improvement in treatment outcome. Furthermore, we expected that MCT would show larger changes in metacognitions compared to other treatments. We were able to identify and assess twenty-six eligible articles which reported on correlational and predictive relationships between metacognitive change and change in outcome.

4.1 Change in metacognitive beliefs following treatment intervention

Our findings suggest that at least some metacognitive beliefs are indeed reduced across all the therapeutic interventions included in this review. These interventions are MCT (Hansmeier et al., 2021; Hjemdal et al., 2019; Hoffart et al., 2018; Johnson et al., 2020; Johnson et al., 2021; Parker et al., 2020; Sürig et al., 2021;), CBASP (Sürig et al., 2021), CBT (Fernie et al., 2015; Johnson & Hoffart., 2018; Johnson et al., 2020; Newby et al, 2014; Spada et al., 2009), CBGT (McEvoy & Perini, 2009; McEvoy et al., 2009), ERP (Grøtte et al., 2015; Hansmeier et al., 2021; Solem et al., 2009; Solem et al., 2015; Sunde et al., 2021), SSRI (Besiroglu et al., 2011; Park et al., 2020), combined data of CT, SSRI and CT with SSRI (Nordahl et al., 2017), D-MCT (Jelinek et al., 2017), ATT and MB-PMR (McEvoy et al., 2017), multimodal treatment consisting of behavioral, cognitive, and metacognitive elements, including ERP (Grøtte et al., 2015), RTW rehabilitation with ACT (Brugnera et al., 2021; Jacobsen et al., 2016; Jacobsen et al., 2020), ConquerFear (Sharpe et al., 2019) and BGTP (Corpas et al., 2021). Four of these studies did not explicitly report pre-post changes in metacognitions (Fernie et al., 2015; Johnson et al., 2020; Sharpe et al., 2019; Spada et al., 2009), but these studies did report significant findings of a relationship between changes in metacognitions and symptom outcome in subsequent analyses which indicates that some change in metacognitions during treatment occurred.

However, we did find seven articles reporting non-significant change in one or more subscales of MCQ-30/65. “Cognitive self-consciousness” and “positive beliefs about the

usefulness of worry" did not change in MCT for patients with high risk psychosis (Parker et al., 2020); positive metacognitive beliefs did not change in CBGT for social phobia (McEvoy & Perini, 2009; McEvoy et al., 2009); in Park et al. (2020; SSRI for OCD) only change in "positive beliefs about worry" were significant; and in RTW rehabilitation with ACT for patients on sick leave due to chronic fatigue, Jacobsen et al. (2016) found all but "cognitive confidence" to be insignificant, whereas Jacobsen et al. (2020) found cognitive self-consciousness and positive metacognitive beliefs to be insignificant. Also, in Sunde et al. (2021), scores of "cognitive confidence" were found to increase during group ERP-treatment, but the statistical significance of these was not reported. It is not clear as to why the particular metacognitions did not change in these studies. However, for some it may be due to small sample sizes (e.g. Parker et al., 2020) and/or weak intervention effects (e.g. Park et al., 2020).

In the studies evaluated we found three comparative studies of MCT versus other treatments, in which two showed MCT to be superior in reducing general metacognitive beliefs (vs. CBASP, depression: Sürig et al., 2021; vs. CBT, anxiety disorder, Hoffart et al., 2018) and one showed MCT to be superior to ERP in reducing thought-fusion beliefs, but not other OC-specific metacognitions in OCD patients (Hansmeier et al., 2021). In this study, differences in change of general metacognitions were not reported. Since targeting metacognitions is an explicit goal of MCT, these studies may support the claim of the S-REF model that metacognitions can effectively be targeted directly in therapy.

Other treatments, which applied metacognitive interventions as elements of treatment, may also target metacognitions directly. For instance, the ConquerFear intervention is composed of elements from both MCT and ACT, where one of the techniques is to challenge the usefulness of worry (Sharpe et al., 2019). And although the main goal of D-MCT is to address content of thought, and is thus according to Jelinek & colleagues (2022), closer to CBT than MCT, changing metacognitions is also an integral part of D-MCT (for example the usefulness of worry; see their module 6). However, these treatments were not compared to other interventions prohibiting any inferences about metacognitive change in these interventions compared to others.

4.1.1 Short summary and considerations in regards to the S-REF model

With regards to the S-REF model, it seems that metacognitions are indeed reduced across different treatments, although not all metacognitions seem to change in all studies. Changes in the MCQ subscales "negative beliefs about the uncontrollability and danger of worry" and the "need to control thoughts" appear to be more frequently significant following

treatment than “cognitive self-consciousness”, “positive beliefs about the usefulness of worry” and “cognitive confidence”. Moreover, metacognitions seem to change more in MCT, where metacognitions are targeted directly than in other effective treatments, but comparative studies are limited in the evaluated studies in this review.

However, the fact that metacognitions were reduced consistently across treatments, indicates that metacognitions could change indirectly through different pathways. Indirect change of metacognitions seems to be the case in most of the studies evaluated. It is beyond this review to speculate how each treatment may change metacognitions specifically, but three general points could be made. Firstly, metacognitions may not just reflect the explicit and consciously held beliefs about cognition, but also the underlying phenomena of cognitive processing itself. Changes in the underlying phenomena of cognitive processing could thus influence the appraisal of metacognitive beliefs explicitly held. In other words, one could “feel” the “need to control thoughts” subside even without conscious reflection of the validity of the belief. For example, cognitive or attentional change may naturally occur after exposure, behavioral activation, changing content of thought, or through acceptance of events, which in turn alter metacognitive beliefs. The S-REF model (1994) claims that the attentional biases and thinking style of CAS is a result of an interaction between bottom-up and top-down processes. Metacognitions are according to this model the top-down pathway to changing CAS. But if CAS were to change in an indirect way through lower-level processing, this may also influence the metacognitive level by means of integration. In the evaluated studies we found two studies of SSRI-treatment without any cognitive components, in which treatment-responders showed reductions in metacognitions (Besiroglu et al., 2011; Park et al., 2020).

Secondly, metacognitions could be changed by top-down processes which are procedurally or semantically linked. In short, any adaptive adjustments to conscious processes such as beliefs, goals and values may change metacognitive beliefs indirectly. Procedurally, one could have a thought about the importance (goal) of attending to something in the external world, which may weaken the relative value of, for example, the need to control thought, since they may be competing in a hierarchy of importance. Similarly, a patient can start to believe that she is safe in her environment, which may semantically be linked to the reduction of “negative beliefs about the uncontrollability and danger of thoughts”, and thereby contribute to procedural change.

And thirdly, metacognitions could be changed as a result of changes in symptoms, as feeling better may naturally produce changes in metacognitions. In a test of temporal

precedence of metacognitions on symptoms by Capobianco et al. (2019), they found that although metacognitions preceded anxiety symptoms, there was also reciprocal relations. Yet, changes in metacognitive beliefs could be due to different explanations than those mentioned here. Nonetheless, these observations seem to indicate that metacognitions are potentially important variables over the course of treatment.

4.2 Metacognitive belief change and change in treatment outcome

The main objective in our review was to investigate the relationship between change in metacognitive beliefs and change in treatment outcome. To do this we identified articles that investigated this relationship by means of correlation analysis, regression analysis, mediation- and moderation analysis, and network analysis. Studies of within-person and between-person effects were also identified and distinguished. Our hypothesis was that change in metacognitions are associated with change in treatment outcome. We expected to find negative metacognitions to be most important, as Wells has proposed (Wells, 2019). In the following we will sort our findings by treatment outcome rather than diagnosis to interpret the findings according to our aim.

4.2.1 Depressive symptoms

Of eleven studies that measured the relationship between change in metacognitions and change in depression symptoms, nine studies indicated a significant association (Besiroglu et al., 2011; Brugnera et al.,2021; Hjemdal et al., 2019; Jelinek et al., 2017; Johnson & Hoffart, 2018; McEvoy & Perini, 2009; McEvoy et al., 2009; Newby et al., 2014; Solem et al., 2015).

Four of these found an association between change in MCQ-30 total score and change in depressive symptoms (Besiroglu et al., 2011; Brugnera et al., 2021; Hjemdal et al., 2021; Solem et al., 2015). Of those studies assessing specific metacognitions, two found that change in the MCQ-30 subscales “negative beliefs about the uncontrollability and danger of worry", “need to control thoughts”, and “cognitive confidence” were significantly correlated with change in depressive symptoms, whereas change in “negative beliefs of uncontrollability and danger of worry” showed the largest effect size (McEvoy & Perini, 2009; McEvoy et al., 2009). They did not find any association with changes in “positive beliefs about worry” and depressive symptoms. Conversely, Newby and colleagues (2014) only assessed “positive

beliefs about rumination and worry", but found that changes in positive beliefs mediated the impact of treatment on depressive symptoms (via repetitive negative thinking). In Jelinek et al. (2017), only the MCQ-30 subscale "need to control thoughts" mediated depressive symptoms. And in Johnson and Hoffart (2018), "negative beliefs about the uncontrollability of thoughts" are associated with feeling depressed and taking little interest in things.

We did however find two studies, by Sürig and colleagues (2021) and Corpas and colleagues (2021), that did not find a significant association between change in metacognitive beliefs and depressive symptoms. For Sürig and colleagues (2021), this inconsistency may be due to the fact that CBASP, which did not target metacognitive beliefs and showed less metacognitive change, was more effective than MCT in treating depression symptoms in this study. Yet, patients were free to choose whether to participate in CBASP or MCT, where those struggling with interpersonal difficulties were recommended CBASP, and those struggling with worry were recommended MCT. Therefore the possibility that performance biases may have influenced the data, in which differences occur as a result of knowledge of interventions, cannot be ruled out. In Corpas et al (2021), several other process variables were included, i.e. measures of emotional regulation and worry, whereas only the subscale MCQ-30-NB were used for metacognitive beliefs, which may have deflated or omitted relations to metacognitions in their analysis.

Our findings indicate that metacognitive belief change seems to be related to change in depressive symptoms, whereas these effects vary from weak to moderate depending on the metacognitive belief domain tested. "Negative beliefs about the uncontrollability and danger of thoughts", "need to control thoughts", "positive beliefs about worry" and "cognitive confidence" all seem to be related to changes in depressive symptoms, whereas "cognitive self-consciousness" did not significantly relate to changes in symptoms in the studies that investigated this variable.

4.2.2 Anxiety symptoms

Changes in metacognitions were associated with changes in levels of anxiety symptoms following treatment, but to a varying degree in subscales on MCQ and other measurements. We found seven studies measuring general anxiety symptoms, all but one reporting a significant association with change in metacognitive beliefs. Johnson et al. (2020) found that changes in metacognitions predicted anxiety symptoms both at the within- and between-level, but did not test for subscales; in Brugnera et al. (2021), total MCQ-30 score predicted anxiety symptoms, but only "negative beliefs about the uncontrollability and danger

of worry" remained as a predictor of anxiety when all process variables were included in their regression model; negative metacognitive beliefs were also found to be a central node in several networks across MCT and CBT, notably in relation to threat monitoring, fear of dying and fear of losing control in Johnson & Hoffart (2018); in Hoffart et al. (2018) change in positive metacognitive beliefs predicted anxiety symptoms at the within-level, and in MCT at the between-level, but only a trend was found between negative metacognitive beliefs and anxiety symptoms; Hjemdal et al. (2019) found correlations between change in MCQ-30 total score, PBRS and NBRS paired with change in anxiety symptoms; while in Newby et al. (2014) change in "positive beliefs about rumination" predicted anxiety symptoms, but other metacognitions were not measured. As with the case for depression symptoms, Corpas et al. (2021) did not find an association between metacognition (MCQ-30-NB) and GAD-symptoms when controlling for emotional regulation, worry and rumination. MCQ-30-NB did however emerge as the only predictor of panic disorder symptoms in their study.

Similar findings were found for other specific anxiety symptoms. In Sharpe et al. (2019), changes in MCQ-30 total score (subscores N/A) predicted fear of cancer recurrence, and in Solem et al. (2015), changes in all MCQ-30 subscales correlated with changes in health anxiety. Here, "negative beliefs about the uncontrollability and danger of worry" showed the strongest association, but only "cognitive confidence" predicted health anxiety in their regression analysis. For state anxiety symptoms measured in McEvoy et al. (2017), change in "negative beliefs about the uncontrollability and danger of worry" predicted state anxiety symptoms, although they did not measure other metacognitions.

Furthermore, we found three studies on the relationship between change in metacognitive beliefs and social anxiety symptoms. McEvoy & Perini (2009) and McEvoy et al. (2009) found a correlation between changes in "negative beliefs about the uncontrollability and danger of worry" and changes in social phobia symptoms, but for other metacognitions or total MCQ-30 score were not found. These findings were modest in comparison to Nordahl & colleagues (2017), where changes in "negative beliefs about the uncontrollability and danger of worry" explained 6-16 percent of the variance in social anxiety symptoms even after controlling change in cognitive beliefs and self-focused attention, depending on how symptoms were measured.

Overall, our findings suggest that changes in metacognitions seem to be influential predictors of change in anxiety symptoms, such as general anxiety symptoms, panic disorder symptoms, health anxiety symptoms, fear of cancer and social phobia symptoms. "Negative

beliefs about the uncontrollability and danger of worry" seem to be particularly important across anxiety symptoms. Also, three studies also suggest positive metacognitive beliefs to be important, specifically to general anxiety symptoms.

However, these findings should be interpreted with caution. Four of the studies mentioned above only assessed "negative beliefs about the uncontrollability and danger of worry", one study only assessed positive beliefs about rumination, while two studies assessed total MCQ-30, but not subscores. Thus, there could have been potential relationships omitted. In addition, one study reversed the analysis, showing that anxiety symptoms also predicted positive metacognitions (Hoffart et al., 2018). This suggests a bidirectional relationship between symptoms and metacognition. Also, the degree to which metacognitions are associated with anxiety symptoms varied. For example, for social anxiety symptoms, there was found only a relation to "negative beliefs about the uncontrollability and danger of worry", whereas for health anxiety, changes in all subscales of MCQ-30 correlated with symptoms.

4.2.3 OCD-symptoms

Our review found an association between change in OCD symptoms and change in metacognitions in all the OCD studies included (Besiroglu et al., 2011; Grøtte et al., 2014; Hansmeier et al., 2021; Park et al., 2020; Solem et al., 2009; Solem et al., 2015; Sunde et al., 2021). Solem and colleagues (2009) found correlations between all MCQ-30 subscores and OCD-symptoms, where positive metacognitive beliefs and "need to control thoughts" showed largest effects. These subscores also emerged as predictors of OCD-symptoms. Solem et al. (2015) found correlations in change on all MCQ-30 scores and OCD-symptoms, but in this study, "negative beliefs about the uncontrollability and danger of worry" showed the largest effect. In Sunde et al. (2021) MCQ-30 total score predicted OCD-symptoms at between-person level, but not on the within-person level. When assessing subscores, MCQ-30-PB, MCQ-30-NB and MCQ-30-NCT predicted OCD-symptoms at the between-person level, but only MCQ-30-NCT did so at the within-person level. Hansmeier et al. (2021) found that for clinician-rated OCD-symptoms, MCQ-65 or SSQ did not correlate with symptoms, but BARI did. For self-rated OCD-symptoms, both changes in MCQ-65 and SSQ correlated with symptoms, but not for BARI. The only significant predictor found was between SSQ and self-rated OCD-symptoms. In Grøtte et al. (2014) MCQ was not assessed but they found that both TFI and BARI predicted change in OCD-symptoms. In Besiroglu et al. (2011) changes in MCQ-30 and TAF correlated with OCD-obsession symptoms, but not

for OCD-compulsion symptoms, where only trends were found. Park et al (2020) found "positive beliefs about worry" to correlate and predict changes in OCD-symptoms, but not for other metacognitions.

In sum, both general and OC-specific metacognitions seem to be consistently associated with change in OCD-symptoms. As with anxiety and depression, "negative beliefs about the uncontrollability and danger of worry" and "positive beliefs about worry" stand out as particularly important, but also "need to control thoughts", which could reflect a general need for control these patients may have. Based on the studies evaluated, we do not have strong indices of whether OC-specific metacognitions are better at explaining symptom change than general metacognitions, or if they add to different aspects of changes in OCD-symptoms. The data varied in which OC-specific metacognitions were most important, and different results were found for different measures of OCD-symptoms, for example in Hansmaier et al. (2021).

4.2.4 Other symptom domains

In chronic fatigue and/or sick leave patients, metacognitive beliefs change were related to change in fatigue symptoms (Brugnera et al., 2021; Fernie et al., 2015), subjective memory problems (Jacobsen et al., 2016), increased odds of returning to work (Jacobsen et al., 2020), and quality of life (Brugnera et al., 2021). Specifically, "negative beliefs about the uncontrollability and danger of worry" and "cognitive confidence" predicted fatigue levels in Fernie et al. (2015); "cognitive confidence" predicted subjective memory problems in Jacobsen et al. (2016); "need to control thoughts" predicted the odds of returning to work in Jacobsen et al. (2020); and in Brugnera et al. (2021), general metacognitions predicted fatigue symptoms, "need to control thoughts" predicted mental health quality of life, and "positive beliefs about worry" predicted odds of working more hours - but none of these effects were significant when other process variables were included, such as avoidance, psychological inflexibility, and cognitive and behavioral responses to chronic fatigue. It should be noted that three of the four studies on fatigue symptoms were conducted at the same rehabilitation center in Norway, and with the same treatment.

In patients with alcohol addiction, changes in all subscales except "positive beliefs about worry" of MCQ-30 correlated with changes in alcohol consumption, whereas "need to control thoughts" remained as a significant predictor of alcohol consumption when depression symptoms and state anxiety was controlled for (Spada et al. 2009). And finally, in patients with high risk of psychosis, treatment outcome was assessed as the CAS-variable

worry, where clustering at the participant level suggested that metacognitions significantly predicted levels of worry across sessional measures (Parker et al., 2020).

4.2.5 Short summary and considerations in regards to the S-REF model

In sum, our findings indicate that changes in metacognitive beliefs seem related to changes in various symptom outcomes. As expected, "negative beliefs about the uncontrollability and danger of worry" appear to be the most relevant subscale of general metacognitions, as it was related to symptoms of depression (Johnson & Hoffart, 2018; McEvoy & Perini, 2009; McEvoy et al., 2009), anxiety (Brugerna, 2021; Johnson & Hoffart, 2018; McEvoy et al., 2017), panic disorder (Corpas et al., 2021), social phobia (McEvoy & Perini, 2009; McEvoy et al., 2009; Nordahl et al., 2017), health anxiety (Solem et al. 2015), OCD (Solem et al., 2015; Sunde et al., 2021), fatigue (Fernie et al., 2015), and alcohol consumption (Spada et al., 2009). Change in "positive beliefs about worry" or "positive beliefs about rumination" (PBRS) was also reported as relevant for change in depressive and anxiety symptoms, and OCD-symptoms (Hoffart et al., 2018; Newby et al., 2014 Park et al., 2020; Solem et al., 2009; Sunde et al., 2021). Furthermore, it may seem that "need to control thoughts" are important for depressive symptoms (Jelinek et al., 2017) and OCD-symptoms (Solem et al., 2009; Sunde et al., 2021), but also for work status in sick leave patients (Jacobsen et al., 2020). "Cognitive confidence" seem to be important for fatigue and health anxiety symptoms (Fernie et al., 2015; Jacobsen et al., 2016; Solem et al., 2015), whereas "cognitive self-consciousness" did not stand out as particularly important in any of our studies.

With regards to the S-REF model, these findings seem to give support to the claim that changing metacognition is a universal mechanism of change. Yet, the studies in this review do not demonstrate causality. To do so requires robust isolation of cause and several points of measurement to establish temporal precedence in the relationship between metacognitions and symptoms. In the majority of our studies, metacognitions were only measured at pre- and post-treatment. Some of these used bivariate correlation analysis, which only demonstrates covariation. Other studies conducted prediction analyses, which controls for other correlational variables, yet assumes a unidirectional path between metacognitions as an independent variable and symptom outcome as a dependent variable. Four studies did however conduct prediction analyses at the within-person level, which is more robust as it shows the aggregated change within each person. We also found two studies that reversed the

analysis, in which one of them demonstrated that changes in symptom outcome could predict metacognitions as well.

However, the findings in the articles investigated seem to indicate that the S-REF model may be too reductionistic to capture the maintenance and change across all mental disorders. Although metacognitions may play an important role in changing symptoms of mental disorders, there seem to be other factors at play. This could reflect an inherent problem with operationalizing theories of psychopathology. György Buzsáki (2019) argues that there is poor overlap between mental constructs and the actual functioning of the brain. This is to say that one should first understand how the brain functions before assuming that the same constructs have the same underlying mechanisms, either between or within a person, or over time. Similar arguments have been made by others, such as Lisa Feldman Barrett, who argues that our nomenclature of emotions are actually not reflected in our brain or body, but are contextualized to our environment (Barrett, 2017). This could imply that metacognitions, or in a larger context - any psychotherapeutic theory of mechanism of change, face the same problem of imprecision in regards to what the concepts actually refer to, and thus what is actually going on when patients improve. If for example metacognitions are umbrella terms reflecting the conscious interpretation of several different processes at both conscious and non-conscious levels in the brain, it is hard to justify true causality, since the term could encompass different things. Yet, imprecision does not necessarily imply falsity. Even if metacognitions do not reflect one true mechanism, there could be true mechanisms at play that could be meaningfully described through metacognitions.

Interestingly however, the S-REF model seems to reflect a shift in the understanding of psychopathology, which is highly relevant for therapy. This is the question of whether psychological disorders are primarily results of stored content of thoughts and feelings, or as a result of the accumulation of maladaptive processing styles. The former would imply that one should target the content of thoughts and feelings, while the latter, argued by the S-REF model, would imply that one should target the processing styles itself. Although the specificity of metacognitions are somewhat inconsistent in our studies, there seems to be broad support to the notion that targeting cognitive processing styles through metacognitions could change symptoms of a range of psychopathology. Also, metacognitions seem to reflect a universal factor associated with symptom change, even when not targeted directly. Most of the studies in this review show evidence at the between-person level, but some studies also found evidence of the importance of metacognitions at the within-person level, which could be considered strong evidence. Thus, when S-REF is seen in light of the overall field of

psychotherapy, which bare little empirical grounds for their theoretical models of therapeutic change, these findings could be seen as a substantial step forward.

4.3 Strengths and limitations

A strength in this review is that we followed the PRISMA statement for conducting systematic reviews, providing transparency in each step. In addition, both authors completed the screening process and quality assessment separately to ensure omissions and biases were reduced.

Our review has several limitations that should be acknowledged. First, we collected studies primarily to assess the relationship between changes in metacognitions and changes in symptom outcome. Thus assessing whether metacognitions change during treatment, and if metacognitions change more in MCT, was a secondary goal. As such, there may be other studies of this sort, which were excluded on the basis of not having sufficient statistical analysis of the relationship between change in metacognitive beliefs and symptom outcome. Our data on these issues are not exhaustive and could lead to biases, particularly if authors see changes in metacognitions as a prerequisite to proper statistical analyses of the relationship between these changes and changes in symptom outcome.

Secondly, the included studies present an amalgam of different treatments modalities, different measurements of treatment outcome and metacognitions, different statistical methods, and across different diagnoses and designs. This lack of homogeneity makes interpretation of data challenging and vulnerable for biases. For example, whereas some studies show that one measure of metacognition is associated with symptom outcome, others show that this may not be the case, but simultaneously showing that another measure of metacognition is associated with the same treatment outcome. Or alternatively, some studies show that metacognitions were associated with symptom change measured with one measurement tool, but not for another. Our aim was to test if metacognitions are indeed associated with treatment outcome across different interventions, but the specifics of these relationships are somewhat inconsistent at this point. The fact that symptom measures were compared between samples with different characteristics also adds to this point, although we did not detect any mentionable differences in data, comparing for example same symptoms with different diagnoses. A quantitative synthesis (e.g. meta-analysis) could have solved some of these limitations, providing a more robust analysis that is less dependent on the

author's subjective interpretation of the articles included. However, the identified studies were too diverse in diagnoses and treatments to make a meaningful analysis.

Thirdly, but closely related, we did not evaluate risk of bias for each of these studies. This could have caused the results to be evaluated as more or less influential, giving us an inaccurate interpretation of the field (Cochrane Collaboration, 2011). These biases include: selection bias, in which experimental groups in the study differ in their baseline characteristics; performance bias, which reflect different factors that would affect one group but not another group in terms of treatment outcomes; detection bias, which occur when outcome variables were collected and analyzed differently in the groups, or when measurements are affected by characteristics of some participants; reporting bias, in which there is a bias in how the data is reported, for example omitting some findings; and attrition bias, where withdrawals from the study may result in incomplete data in one group compared to the other. We did comment on a possible performance bias in Surig et al. (2021), in regards to their lack of findings, but there may be other such potential biases undetected in our data that could weaken the reliability of our analysis.

Fourthly, neither of the authors of this review are trained in quality assessment of studies. A full analysis of each article's limitations were therefore beyond our limits. However, we did our best to have a common understanding of the practical score guidelines seen in the appendix, which may explain the high inter-rater reliability. This of course could also have led to coexisting biases in our interpretation of these guidelines.

Finally, we did not pre-registered our review to databases such as PROSPERO (Booth et al., 2012). Registering in such a database would contribute to avoid duplication and reduce bias by comparing the completed review with what was planned in a protocol.

4.4 Suggestions for further research based on the existing literature

At present, more studies with comparable design and measurements are needed to conduct meaningful systematic quantitative analyses (e.g. meta-analyses). Our review however, indicates that metacognitions are promising factors in psychotherapeutic change. Research should therefore further investigate metacognitions as a potential mechanism of change. Overall, the current studies lack statistical power and/or do not show temporal precedence of metacognitions over symptoms. To overcome this, one should conduct studies with measures at several time points and larger sample sizes. There is also a need for more RCT-studies to isolate metacognitions as a change variable by controlling for other possible

confounding variables. In addition, studies at the within-person level will provide more robust findings than studies at the between-person level. Different measures of metacognitions should also be evaluated simultaneously to identify the unique contributions of specific and general metacognitions.

As metacognitions did not appear as a perfect fit to explain the variance of symptom change, future research should also investigate other proposed change mechanisms. By controlling for these variables, one can compare the relative importance of metacognitions versus other specific mechanism variables, such as cognitive or affective variables. Also, as both symptoms and mechanism variables may have mutually interactive and reciprocal relations with other symptoms and mechanism variables, investigating these relations with a network approach is recommended. Finally, any findings should be replicated across different treatment modalities and across different symptoms to evaluate the universality of these potential mechanisms. Of particular notice, we did not find any studies investigating the role of metacognitions in studies with a psychodynamic approach, or any studies investigating symptoms of PTSD, eating disorders or insomnia. Further research should also include these domains.

5. CONCLUSION

The primary aim of this study was to conduct a systematic literature review to evaluate whether change in metacognitive belief is associated with symptom change across different psychological disorders and across different therapeutic interventions. Based on the S-REF model (Wells & Matthews, 1994) we hypothesized that all effective therapeutic interventions change metacognitions following treatment regardless of disorder, and that this change was associated with changes in treatment outcome. In addition, we hypothesized that MCT would show larger changes in metacognitive change than other treatment interventions. Our results show that all therapeutic interventions did indeed change metacognitions during treatment. Broadly speaking, changes in metacognitions were also associated with changes in treatment outcome across all disorders investigated in this review, although the specific metacognitions did not always overlap between studies, treatment, or disorders. As expected, change in "negative beliefs about the uncontrollability and danger of worry" appeared as the most relevant metacognition in regards to symptom change. These results give further support to the claim of the S-REF model that metacognition is a universal factor associated with symptom change, and in particular uncontrollability beliefs. Furthermore, the fact that MCT showed the largest change in metacognitions following treatment could be an indication as to why MCT has been associated with strong and positive treatment effects compared to other interventions. If dysfunctional metacognition is central to disorder, effective modification of them should produce improvement.

To our knowledge, this is the first systematic review to synthesize the relationships of changes in metacognitive beliefs and changes in symptoms during treatment. Our synthesis builds on the current research showing that the S-REF model is a promising model for understanding psychopathology. Hence, the metacognitive model has substantial empirical support of one of its most central tenets, which is arguably a consequential step forward in regards to the search for effective treatments. Yet, showing an association between changes in metacognition and changes in symptoms during treatment is only a preliminary step and must not be confused with causality. We urge future research to investigate these relations further with proper experimental designs and sufficient samples. If metacognitive change is identified as causal in improvement of psychopathology – using the term "paradigm shift" would for the first time in many years come to its right within the field of clinical psychology.

APPENDIX

Table 1: Study characteristics

	(Year) Authors	Intervention	Diagnosis / symptoms	Metacognition / outcome variables	QA Score, R1 / R2	Sample Characteristics	Statistical analysis
#1	(2017) Jelinek, van Quaquebeke & Moritz	Metacognitive training (D-MCT) group therapy or Health training (active control)	Depression	MCQ-30-PB, MCQ-30-NB, MCQ-30-NFC / HDRS, BDI	30/30	N = 84 (M age = 45.5 (*SD* = 9.9), 74% female), D-MCT group = 41, HT group = 43, diagnosed with major depressive episode, recurrent depression, or dysthymia	Simple linear regression analysis, mediation analysis
#2	(2019) Hjemdal, Solem, Hagen, Kennair, Nordahl & Wells	MCT or waiting list	Depression and anxiety symptoms	MCQ-30, NBRS, PBRS / BDI, BAI	30/29	N = 39 (M age = 33.7 (*SD* = 10.4), 59% female), diagnosed with depression	Bivariate Pearson's correlation, multiple hierarchical regression analyses
#3	(2021) Sürig, Ohm, Grave, Glanert, Herzog, Fassbinder, Borgwardt & Klein	MCT or CBASP	Depression	MCQ-30 / QIDS-SR16	32/32	N = 90, CBASP group = 37, age (M age = 28.7, 41% female), MCT group = 53, (M age = 44.7, 43% female), diagnosed with depression	Hierarchical multiple regression analysis
#4	(2018) Hoffart, Johnson, Nordahl & Wells	MCT or CBT	Anxiety disorders	CAS-1 / BAI, PHQ-9	33/33	N = 67 (M age = 42 (*SD* = 12.8), 61% female) CBT group = 33, MCT group = 34, who met DSM-IV inclusion criteria for either PTSD, social anxiety disorder, or panic disorder with or without agoraphobia	Mixed models
#5	(2018) Johnson & Hoffart	MCT or CBT	Anxiety disorders and depressive symptoms	CAS-1 / BAI, PHQ-9	29/29	N = 67 (M age = 42 (*SD* = 12.8), 61% female) CBT group = 33, MCT group = 34, who met DSM-IV inclusion criteria for either PTSD, social anxiety disorder, or panic disorder with or without agoraphobia	Multilevel vector autoregressive (mlVAR) model
#6	(2020) Johnson, Hoffart, Tilden, Toft, Neupane, Lien & Bramness	MCT and CBT mixed in analysis	Anxiety disorders	MCQ-30 / BAI	32/33	N = 37 (M age = 43.6 (*SD* = 11.0) 54% female) MCT group = 15, CBT group = 22, diagnosed with GAD, PTSD, Social phobia, and/or panic disorder.	Longitudinal multilevel modeling

#7	(2009) McEvoy & Perini	CBGT with or without ATT	Social phobia and depressive symptoms	MCQ-30 / SPS, SIAS, BDI, ACS, AP	29/28	N = 81 (M age = 30.7 (*SD* = 9.4), 37% female), diagnosed with social phobia	Mixed model ANOVAS
#8	(2009) McEvoy, Mahoney, Perini & Kingsep	CBGT	Social phobia and depressive symptoms	MCQ-30 / PEP, SPS, SIAS, BDI	26/26	N = 61 (M age = 30.9 (*SD* = 9.5), 34% female), diagnosed with social phobia	Pearson's bivariate correlation
#9	(2017) Nordahl, Nordahl, Hjemdal & Wells	CT, SSRI or a combination were mixed in the analysis	Social phobia	MCQ-30-NB / FNE, LSAS, SAD, SIAS	31/30	N = 46 (M age = 30.02 (*SD* = 9.2), 48% female), diagnosed with social anxiety disorder	Hierarchical multiple linear regression analysis
#10	(2009) Solem, Tellefsen, Vogel, Hansen & Wells	ERP treatment or CBGT (mainly ERP-based)	OCD	MCQ-30 / Y-BOCS	31/29	N = 83 (M age = 34.2 (*SD* = 11.9), 71% female), individual ERP = 50, group ERP = 33, diagnosed with OCD	Correlation and regression analysis
#11	(2011) Besiroglu, Cetinkaya, Selvi & Atli	SSRI	OCD and depressive symptoms	MCQ-30, TAF / Y-BOCS, BDI	29/29	N = 55 (M age = 27.7 (*SD* = 8.5), 58% female), diagnosed with OCD	Linear regression analysis
#12	(2015) Grøtte, Solem, Vogel, Güsey, Hansen & Myers	Multimodal treatment with ERP, and behavioral, cognitive and metacognitive elements	OCD	TFI, BARI / Y-BOCS-SR, OCI-R	31/30	N = 108 (M age = 34.1 (*SD* = 12.1), 69% female), diagnosed with OCD	Logistic regression analysis
#13	(2015) Solem, Borgejordet, Haseth, Hansen, Håland & Bailey	ERP treatment or control group	OCD and health anxiety- and depressive symptoms	MCQ-30 / Y-BOCS, WI, BDI	30/28	N = 695, ERP group = 313 (M age 34.6 (*SD* = 12.0), 65% female), control group = 382 (M age 26.2 (*SD* = 5.7), 55% female). OCD sample and community control	Correlation and regression analysis
#14	(2020) Park, Kim, Jeon, Hwang, Kang & Kim	SSRI	OCD	MCQ-65 / Y-BOCS, MADRS	20/20	N = 132 (M age = 27.6 (*SD* = 8.3), 46% female), diagnosed with OCD	Binary logistic regression analysis, partial correlation analysis
#15	(2021) Sunde, Johnson, Himle, Bertelsen, Haaland, Vogel, Walseth & Haaland	Group ERP	OCD	MCQ-30 / Y-BOCS	27/27	N = 40 (M age = 33.4 (*SD* = 12.4), 78% female), diagnosed with OCD	Longitudinal multilevel modeling
#16	(2021) Hansmeier, Haberkamp, Glombiewski & Exner	MCT or ERP	OCD	MCQ-65, TAF, BARI, SSQ / PI-PR, Y-BOCS	32/31	N = 24 (M age = 30.5 (*SD* = 10.4), 63% female), diagnosed with OCD	Hierarchical regression analysis
#17	(2015) Fernie, Murphy, Wells, Nikcevic & Spada	CBT or GET	Chronic fatigue	MCQ-30 / CFQ	33/34	N = 171 (M age = 40.8 (*SD* = 12.5, gender distribution N/A), CBT group = 116, GET group = 55, diagnosed with	Regression analysis

						chronic fatigue	
#18	(2016) Jacobsen, Aasvik, Borchgrevink, Landrø & Stiles	RTW program with ACT	Chronic Fatigue and Subjective memory problems	MCQ-30 / EMQ	35/36	N = 137 (M age = 43.6 (*SD* = 9.3), 80% female) on sick leave due to chronic fatigue	Hierarchical linear regression
#19	(2020) Jacobsen, Glette, Hara & Stiles	RTW program with ACT	Long term sick leave due to chronic pain, chronic fatigue and common psychological disorders	MCQ-30 / Work status	28/31	N = 137 (M age = 43.0 (*SD* = 8.6), 81% female) patients on long-term sick leave	Linear mixed-modeling regression analysis
#20	(2021) Brugnera, Jacobsen, Woodhouse, Compare & Jacobsen	RTW program with ACT	Chronic fatigue and anxiety- and depressive symptoms	MCQ-30 / CFQ, HADS-A, HADS-D, SF-8, Work status	31/30	N = 195 (M age = 43.6 (*SD* = 9.3), 81% female), diagnosed with chronic fatigue	Hierarchical linear models
#21	(2020) Parker, Mulligan, Milner, Bowe, & Palmier-Claus	MCT	High risk psychosis and worry	MCQ-30 / CAS-1	30/30	N = 10 (M age = 22.8 (SD = 4.0), 40% female), at risk of developing psychosis	Regression analysis
#22	(2009) Spada, Caselli & Wells	CBT	Alcohol addiction	MCQ-65 / QFS	28/28	N = 70 (M age = 47.6 (*SD* = 9.1), 31% female), diagnosed with alcohol abuse	Logistic- and hierarchical regression
#23	(2017) McEvoy, Graville, Hayes, Kane, & Foster	ATT or MB-PMR or Thought wandering control	High-trait anxious symptoms	UTS (MCQ-30-NB) / STICSA	30/28	N = 81 (M age = 23.6 (*SD* = 7.7), 80% female), ATT group = 27, MB-PMR group = 27, TWC group = 27. Nonclinical sample	Generalized linear mixed models
#24	(2019) Sharpe, Turner, Fardell, Thewes, Smith, Gilchrist, Beith, Girgis, Tesson, Day, Grunewald. & Butow	ConquerFear or Control group with relaxation training	Fear of cancer recurrence	MCQ-30 / FCRI	33/34	N = 152 (M age = 52.8 (*SD* = 10.1), 98% female) with fear of cancer recurrence	Mediation and moderator analysis
#25	(2014) Newby, Williams & Andrews	Transdiagnostic internet-delivered CBT (iCBT)	GAD and depressive symptoms	PBRS-A / RNT, PHQ-9, GAD-7	28/28	N = 99 (M age = 44 (*SD* = 12.2), 78% female). iCBT group = 46, WLC = 53, mixed anxiety and depression sample	Mediation analysis
#26	(2021) Corpas, Moriana, Venceslá & Gálvez-Lara	Brief group transdiagnostic therapy (BGTP) or pharmaco-logical therapy	Symptoms of GAD, depression, panic disorder and somatoform disorder	MCQ-30-NB / GAD-7, PHQ-15, PHQ- PD, PHQ-9	32/32	N = 105 (M age = 39.6 (*SD* = 11.2), 67% female), BGTP = 53, TAU = 52, with mild to moderate symptoms of somatoform anxiety and/or depression.	Linear regression analysis

Table 2: Study results with statistics

	(Year) Authors	Results
#1	(2017) Jelinek, Van Quaquebeke & Moritz	**1.** Significant change in MCQ-30-PB ($F(1, 81) = 12.041$, $p = .001$, $\eta p\ 2 = .129$), MCQ-30-NB ($F(1, 81) = 8.327$, $p = .005$, $\eta p\ 2 = .093$) and MCQ-NCT ($F(1, 81) = 11.445$, $p = .012$, $\eta p\ 2 = .075$) in the D-MCT group compared to control group. **2.** MCQ-30-NCT was the only significant mediator of long term reduction in HDRS ($b = .27$, $SE = .21$) and BDI ($b = .40$, $SE = 11$). For HDRS there was an indirect effect of treatment on change in depression via MCQ-30-NCT of .15 ($SE = .10$; BootLLCI = .02, BootULCI = .44; K^2 = .09). Indirect effect of treatment on change in BDI via MCQ-30-NCT was .22 ($SE = .11$; LLCI = .05, ULCI = .52; K^2 = .13).
#2	(2019) Hjemdal, Solem, Hagen, Kennair, Nordahl & Wells	**1.** Significant ($p < .001$) change in MCQ-30 pre- to post-treatment ($d = 1.82$), 6-month ($d = 1.75$) and 1-year follow-up ($d = 1.74$, F = 59.73). Significant ($p < .001$) change in NBRS pre- to post-treatment ($d = 1.54$), 6-month (d = 1.59) and 1-year follow-up ($d = 1.61$, F = 51.80). Significant ($p < .01$) change in PBRS pre- to post-treatment ($d = 1.34$), 6-month ($d = 1.27$) and 1-year follow-up ($d = 1.23$, F = 43.50). **2.** Significant ($p < .01$) correlation between ΔMCQ-30 (r. = .62; .59), ΔPBRS (r.= .48; .34), ΔNBRS (r.= .45; .33) paired with ΔBDI and ΔBAI respectively was found from pre-treatment to follow up. **3.** Change in MCQ-30 showed to be a significant ($p < .000$) predictor for change in BDI ($\beta = .58$, $t = 4.07$, $p = .000$) from pre-treatment to 1-year follow-up.
#3	(2021) Sürig, Ohm, Grave, Glanert, Herzog, Fassbinder, Borgwardt & Klein	**1.** Significant ($p < .001$) change in MCQ-30 from pre- to post-treatment (t = 7.56, d = -.80 (95% CI (-1,06 to -.45)) in both treatments, where MCT was superior (t = 6.21, d = -.86; CBASP = t = 4.61, d = -.76) **2.** Change in MCQ-30 was not a significant ($p = .26$) predictor of QUIDS scores. Neither was MCQ-30-NB (B = 1.74, SE = .18, $p = .33$).
#4	(2018) Hoffart, Johnson, Nordahl & Wells	**1.** CAS-1 decreased more in MCT than in CBT for both positive- (B = -1.71, SE = .67, t(60.8) = -2.55, $p < .013$) and negative meta-beliefs (B = -1.55, SE = .60, t(90.4)) = -2.57, $p < .012$). **2.** There was a within-person relationship between positive meta-beliefs and BAI ($B = .07$, dF = 407.7, T = 3.15, $p < .05$). There was also a trend that negative meta-beliefs predicted BAI ($B = .04$, dF = 449, T = 2.08, $p = .039$). When the variable time was entered into the model, none of the within-person relationships were significant (NMB = $B = .03$, $SE = .02$, $t(447.9) = 1.23$, PMB = $B = .03$, $SE = .02$, $t(424.8) = 1.62$). **3.** There was a reversed within-person relationships between BAI and subsequent positive meta-beliefs ($B = .30$, $SE = .09$, $t(374.0) = 3.50$, $p < .001$), but not in negative meta-beliefs ($B = .18$, SE = .10, t(456.7) = 1.92, p = .056). Significant ($p < .001$) within-person relationship between BAI and subsequent positive meta-beliefs in CBT ($B = .49$, $SE = .13$, $t(163.9) = 3.95$), but not in MCT, and a significant between-person relationship between level BAI and level of positive meta-beliefs in MCT ($B = .42$, $SE = .45$, $t(33.7) = 4.29$ $p < .001$), but not in CBT ($p = .93$).
#5	(2018) Johnson & Hoffart	In the MCT temporal network, belief about uncontrollability of thoughts predicts threat-monitoring, and threat monitoring is also predicted by fear of losing control. In the contemporaneous network, beliefs about uncontrollability of thoughts is central, as well as worry and threat monitoring. For the between person network in MCT, worry is again a central node, and connected to threat monitoring, and threat monitoring to the belief about uncontrollability of thoughts. In the CBT networks some MCT typical nodes were prominent. Fear of losing control predicts the cognition fear of dying, which predicts heart pounding and racing. Beliefs about uncontrollability of thoughts is central as well as worry and threat-monitoring. Beliefs about uncontrollability of thoughts is also central in the between-person network in CBT.
#6	(2020) Johnson, Hoffart, Tilden, Toft, Neupane, Lien & Bramness	**1.** Changes in metacognitions pre-post-treatment were not reported. **2.** Metacognitions predicted anxiety over the course of therapy both on and within-person effect (.25(.05), (95%CI = .16, .34), $p < .001$) and on a between-person level (.51(.07),(95%CI .4, .7), $p < .001$). **3.** Cytokines did not significantly ($p > .05$) function as a moderator between metacognition and anxiety.
#7	(2009) McEvoy & Perini	**1**. MCQ-30-subscores, except for positive beliefs, changed in both treatments, but no differences between treatment conditions as the condition by time were significant ($p > .05$). **2**. Reductions in MCQ-30-NB were significantly ($p < .05$) correlated with reductions on all three symptom measures (BDI: r. = .53, SIAS: r. = .26, SPS r. = .22). Reduction in MCQ-30-CC and MCQ-30-NCT were significantly ($p < .05$) correlated with reductions in BDI (MCQ-30-CC: r. = .31, MCQ-30-NCT: r. = .34).

#8	(2009) McEvoy, Mahoney, Perini & Kingsep	**1.** MCQ-30 total and subscale scores was significantly ($p < .05$) reduced from pre- to post treatment (MCQ-30 total: $d = .61$, MCQ-30-CC: $d = .33$, MCQ-30-CS: $d = .27$, MCQ-30-NB: $d = .66$, MCQ-30-NCT: $d = .68$), with the exception of MCQ-30-PB ($d = .01$). **2.a** Reduction in metacognitions were not significantly ($p > .05$) correlated with reduction in SPS, and only the MCQ-30-NB correlated with SIAS (r. = .29). **2.b** ΔMCQ-30-NCT (r. = .38), ΔMCQ-30-CC (r. = .43)" and ΔMCQ-30-NB (r. = .54) was significantly ($p < .01$) correlated with reduction on BDI. **2.c** Reduction in MCQ-30-NB (r. = .37) and MCQ-30-NCT (r = .31) were associated with reduction in PEP.
#9	(2017) Nordahl, Nordahl, Hjemdal & Wells	**1.** MCQ-30-NB changed during the course of treatment (t = 3.92 $d = .62$, $p < .01$**) 2.** MCQ-30-NB explained a significant ($p < .01$) additional 15.9% of the variance in FNE (β = .41 t(3.283), 5.9% of the variance in LSAS (β = .25 t = (2.101), 12%9% of variance in SAD (β = .38 t(2.958), and 10.3% of the variance in SIAS (β = .32 t(2.817).
#10	(2009) Solem, Tellefsen, Vogel, Hansen & Wells	**1**.Significant ($p < .001$) decrease in MCQ-30 total score ($t = 8.01$, $d = .78$) following treatment. Patients who achieved clinical significant change on Y-BOCS had significant ($p < .001$) lower MCQ-30 total scores at post-treatment compared to patients who were improved (45.51 [$SD = 9.77$] vs. 59.66 [$SD = 13.60$], $t(49) = 3.37$) as well as patients who did not achieve change (60.06 [$SD = 17.91$], $t(74) = 4.54$, $p < .0001$). Greater change in MCQ-30 total score on responders (M = 15.75 ($SD = 12.93$) compared to nonresponders (M = 4.26 ($SD = 10.20$) **2.** ΔMCQ-30 subscales were all significantly ($p < .01$) moderately (.35 (ΔMCQ-30-CS) - .39 (ΔMCQ-30-PB)) correlated with both the Y-BOCS post-treatment scores and ΔY-BOCS. **3.** Regression analysis showed that MCQ-30 total score (B =−.18 ($SE = .07$), β =−.38, $t = -2.63$, $p < .001$), MCQ-30-NB (B = −.49 ($SE = .18$), β = −.31, $t = -2.67$, $p < .01$) and MCQ-30-PB (B = −.57 (SE = .28), β = −.22, $t = -2.07$, $p < .05$) predicted change in Y-BOCS following treatment.
#11	(2011) Besiroglu, Cetinkaya, Selvi & Atli	**1.** MCQ-30 was reduced during SSRI-treatment ($t = 2.92$, dF = 54, $p < .01$). TAF was reduced during SSRI treatment (TAF morality: $t = 3.38$, dF = 54, $p < .005$), TAF total: $t = 2.71$, dF = 54, $p < .01$) except TAF morality ($p > .05$ **2.** ΔMCQ-30 correlated with ΔY-BOCS obsession (r = .41, $p < .005$) and Y-BOCS insight (r = .27, $p < .05$), but not in Y-BOCS compulsion ($p > .05$). ΔMCQ-30 was also correlated with ΔBDI (r = .45, $p < .001$). **3** MCQ-30 did not predict Y-BOCS during treatment (statistics N/A).
#12	(2015) Grøtte, Solem, Vogel, Güsey, Hansen & Myers	**1.** TFI ($t = 9.37$, $d = 1.18$) and BARI ($t = 17.15$, $d = 2.09$) was significantly ($p < .000$) reduced after treatment. **2.** When using reliable change indices reliable TFI (B = 1.46, $SE = .61$, $p = .02$) and BARI (B = 1.47, $SE = .68$, $p = .02$) emerged as significant predictors of recovery (Y-BOCS-SR). Achieving reliable change (or no beliefs) on the TFI at post-treatment was associated with an increase in odds of recovery by a factor of 4.32 (95% CI [1.32, 14.16], and similar results for reliable change (or no beliefs) on the BARI with a factor of 4.35 (95% CI [1.15, 16.41]. **3.** When OCI-R was the dependent variable, TFI (B = .00, $SE = .00$, $p = .04$) and BARI (B = .14, $SE = .05$, p = .00) emerged as significant predictors Y-BOCS-SR. When using reliable change indices only BARI (B = 1.83, $SE = .63$, $p = .00$) emerged as a significant predictor on the OCI-R. Achieving reliable change on the BARI (or no beliefs) were associated with an increase in odds of recovery by a factor of 6.21 (95% CI [1.81, 21.25]).
#13	(2015) Solem, Borgejordet, Haseth, Hansen, Håland & Bailey	**1.** Results showed significant ($p < .05$) decrease in MCQ-30 total score ($d = 1.20$) following treatment. Significant ($p < .001$) change in MCQ-30-NB ($d = 1.39$), MCQ-30-NCT ($d = 1.17$), MCQ-30-PB ($d = .56$), MCQ-30-CC ($d = .57$) and MCQ-30-CS ($d = .92$) following treatment. **2.** ΔMCQ-30-NB were significantly ($p < .01$) correlated with both ΔWI (health anxiety symptoms) (r = .48), ΔY-BOCS (r = .38) and ΔBDI (r = .46). **3.** In the regression analysis MCQ-30-CC was the only significant ($p = .049$) metacognitive predictor of WI post-treatment.
#14	(2020) Park, Kim, Jeon, Hwang, Kang & Kim	**1.** Early responders on SSRI showed a significant lower score on MCQ-30-PB, compared to non-responders, but not for other MCQ-30-subscores (statistics N/A). **2.** ΔMCQ-30-PB was associated with ΔY-BOCS (r. = -.262, $p = .002$). **3.** The regression analysis revealed PB as a significant independent variable for predicting early treatment response (explained 11.4% of variance) and for predicting Y-BOCS reduction (B = − .566, $p = .001$.)

#15	(2021) Sunde, Johnson, Himle, Bertelsen, Haaland, Vogel, Walseth & Haaland	**1.** MCQ-30-total scores were significantly (p < .001) reduced over the course of treatment and follow-up (β = -2.81, *SE* = .49, t(33.6) = 5.77) **2.** There was a significant between-person effect of MCQ-30 total score on Y-BOCS (β = .19, SE = .06, t(38.91) = 3.4, p = < .01), but not a within person effect (β = .05, SE = .04, t(36.50) = 1.51, p = .14). In the reversed analysis, OCD symptoms were predictive of MCQ-30 total score at the between-person level (β = 1.69, SE = .44, t(38.57) = 3.80, p < .01*),* but not at the within-person level (β = .13, S = .013, t(56.66) = .99, p = .33). **3.** MCQ-30 at the between-person level emerged as a predictor when obsessive beliefs were entered into the model (β = .25, SE = .09, t(36.94) = 29, p < .01). **4.** MCQ-30-PB (β = .79, *SE* = .21, t(38.04) = 3.63, p = .001), MCQ-30-NB (β = .99, *SE* = 21, t(36.84) = 4.77, p < .001 had a between-person effect on Y-BOCS, but not a within person effect. MCQ-30-NCT had both an between-person- (β = .68, *SE* = .28, t(39.60) = 2.40, p = .021) and within-person effect (β = .29, *SE* = .12, t(39.30) = 2.49, p < .017). MCQ-30-CC (β = .24, *SE* = .22, t(39.34) = 1.08, p = .287), and MCQ-30-CS (β = .46, *SE* = .23, t(39.14) = 1.97, p = .055), was not significant predictors at between-person level. MCQ-30-CC (β = .10, *SE* = .10, t(60.31) = .66, p = .509) and MCQ-30-CS (β = .21. *SE* = .13, t*(*43.79*)* = 1.62*,* p = .013) were not significant predictors at within person-level.
#16	(2021) Hansmeier, Haberkamp, Glombiewski & Exner	**1.** Both MCT (Pre-post: TAF: 2.42, BARI: 2.91, SSQ: .57) and ERP (Pre-post: TAF: .04, BARI: 1.28, SSQ: .70) changed OC-specific metacognition. **2.** SSQ pre-post significantly (p = .047) predicted PI-PR post-treatment (Δr^2 = .13) and follow-up at three months (Δr^2 = .30, p = .011). There was only a trend for MCQ-30 total score in predicting PI-PR post-treatment outcome (Δr^2 = .22, p = .060). **3.**The association between change in PI-PR and change in SSQ was also significant (.047) at follow-up (B = -.38.). No significant prediction between the rest of the variables pre- or post treatment (p < .1).
#17	(2015) Fernie, Murphy, Wells, Nikcevic & Spada	**1.**MCQ-30 pre-post scores were not reported. **2.** When entered in the regression analysis along with HADS-A and HADS-D, MCQ-30-NB (estimation: -.26 t = -2.4) and MCQ-30-CC (estimation: .17 t = 1.68) had a significant (p < .001) effect on levels of fatigue regardless of treatment modalities.
#18	(2016) Jacobsen, Aasvik, Borchgrevink, Landrø & Stiles	**1.** MCQ-CC was the only significant subsore that decreased during treatment (t = 3.2, g = .15, p = .002). **2.** Reduction in MCQ-30 total score was significantly (p < .000) associated with a reduced post-treatment score on EMQ (B = .347). **3.** Post-treatment MCQ-30-CC then emerged as an independent predictor of post-treatment scores on EMQ (p < .0001; t = 6.0; B = 1.2; CI = .8, 1.7).
#19	(2020) Jacobsen, Glette, Hara & Stiles	**1.** Significant (p < .05) reduction in MCQ-30-total score . MCQ-30-PB (p > .27) and MCQ-30-CS (p > .27) was the only subscale that did not significantly change from pre-post treatment in both groups. **2.** MCQ-30 was significantly associated with a 5% increased odds ratio for returning to work per 1-point change in MCQ-30. When looking at the subscales, only MCQ-30-NCT was significant, in which 1-point change in score was associated with 20% OR for RTW.
#20	(2021) Brugnera, Jacobsen, Woodhouse, Compare & Jacobsen	**1.** Significant decrease in MCQ-30 total score from pre- to post-treatment (β10 = -2.91 (*SE* = .68), t = -4.31(*df* = 194), p < .001). **2.** When entered one at a time: a greater decrease in MCQ-30-CC (β = 289 (*SE* = .118), t = 2.449(*df* = 139 p = .016), MCQ-30-NB (β = *.032* (SD = .238), t = *2.*526(df = 136), p = .013) and MCQ-30-N*CT* (β = .032 (S*D* = .152), t = 2.079(df = 136), p = .040) predicted a steeper decline in fatigue symptoms (CFQ) post treatment. **2a.** MCQ-30-CS (β = .933 (*SE* = .294), t = 3.175 (*df* = 139, p = .002) and MCQ-30-NB (β = 1.13 (*SD* = .321), t = 3.510 (*df* = 137), p < .001) predicted HADS-A scores post treatment. **2b.** MCQ-30-CC (β = .431 (*SD* = .210), t = 2.050 (*df* = 140), p = .042), MCQ-30-CS (β = .587 (*SD* = .271), t = 2.168 (*df* = 139), p = .032) and MCQ-30-NB (β = .659 (*SD* = .282), t = 2.334 (*df* = 137, p = .021) predicted HADS-D scores post treatment. **2c.** MCQ-30-NCT (β = -.345 (SD = .124, t = -2.782 (*df* = 133), p = .006) predicted steeper increase in SF-8. **2d.** MCQ-30-PB (B = -.396 (*SE* = .187), *wald* = 4.487, p = .034) predicted Work Status **3.** When variables entered together: Only MCQ-30-NB (β = .651 (*SD* = .252) t = 2.586 (*df* = 133), p = .011) predicted anxiety levels (HADS-A).
#21	(2020) Parker, Mulligan, Milner, Bowe, & Palmier-Claus	**1**.Significant lower scores on MCQ-30 total (p = .018) and in three out of five subscales, MCQ-30-NB (p = .018), MCQ-30-CC (p = .018) and MCQ-30-NCT (p = .027), after 12 weeks treatment. Significant reduction in MCQ-30 total score (p = .028) and in three out of five subscales, MCQ-30-NB (p = .026), MCQ-30-CC (p = .026) and MCQ-30-NCT (p = .027), at six months. **2**. Metacognitive beliefs significantly predicted levels of worry across sessional measures (β = .73, *SE* = .07, p < .001, CI: .58-.89)

#22	(2009) Spada, Caselli & Wells	**1.** MCQ-65 pre-post scores were not reported. **2.** Significant ($p < .01$) positive correlation between ΔMCQ-65-NCT and ΔQFS at 3 (.r = .37), 6 (.r = .45) and 12 (.r = .39) months. Significant ($p < .01$) positive correlation between ΔMCQ-65-CC and ΔQFS at 3 months (.r = .54). Significant ($p < .05$) positive correlation between ΔMCQ-65-NB (.r = .27) and ΔMCQ-65-CS (.r = .25) and ΔQFS at 6 months. **3**. MCQ-65-NCT were a significant ($p < .01$) predictor for drinking status at both 3- ($B = 1.11$) and 6 months ($B = 1.13$) follow-up. **4.** In the hierarchical regression analysis, MCQ-65-NCT accounted for a significant variance in level of weekly alcohol use at all time points (3 months: β = .31, t, $p = .01$, 6 months: β = .39, t, $p = .00$, 12 months: β = .31, t = 2.6, $p = .01$).
#23	(2017) McEvoy, Graville, Hayes, Kane, & Foster	**1.** Both ATT ($F(1, 156) = 41.43$, $p < .001$, d = .86) and MB-PMR ($F(1, 156) = 46.93$, $p < .001$, d = .91) decreased (UTS) post-manipulation (no significant difference between the two groups). TWC had also a small but significant ($F(1, 156) = 5.63$, $p = .019$, d = .32.) effect. **2.** Lower UTS scores were associated with lower STICSAcog (anxiety) scores at post treatment (β = .477, SE = .126, 95% CI [.231;.718]).
#24	(2019) Sharpe, Turner, Fardell, Thewes, Smith, Gilchrist, Beith, Girgis, Tesson, Day, Grunewald. & Butow	**1.** Statistics of MCQ-30 change pre-post were not reported. **2.** In the mediation model, the ConquerFear group reported more reductions in unhelpful metacognitions than control ($F(6,136) = 2.337$, $p = .0353$). **3.** Reductions in metacognitions significantly ($p = .008$) predicted (β = -.204) FCRI at follow-up. **4**. MCQ-30 total score did not moderate treatment efficacy ($F(6,123) = .0701$, $p = .792$).
#25	(2014) Newby, Williams & Andrews	**1.** PBRS-A was significantly ($p < .001$) reduced during treatment at both a between- ($F(1, 95.51) = 14.60$) and within- ($t(96.28) = 5.87$) person level. The main effect of time was not significant for PBRS-A at 3-months follow-up ($F(1,38.39) = .80$, $p = .37$, $r = .64$) **2.** The indirect effect of treatment on PHQ-9 via PBRS was statistically different from zero (95% CI = -.88 to -.04. 3), also when repeated with gender (95% CI: −.42 to −.02). The indirect effect of treatment on GAD via change in PBRS was statistically different from zero (95%CI = -1.23 to -.05), but not when baseline levels of RNT and degree of PB was controlled for. **3.** Reduction of PBRS mediated the impact of iCBT on PHQ-9 (95% CI: −.72 to −.01). Indirect effect of iCBT on GAD-7 via reduction of PBRS and reduction in RNT scores was statistically different from zero (95% CI = −.73 to −.02). The indirect effect of treatment via PBRS was not statistically different from zero (95% CI = −.71 to .85).
#26	(2021) Corpas, Moriana, Venceslá & Gálvez-Lara	**1.** The BGTP group showed significant decrease ($p < .001$) in MCQ-30-NB ($t = 6.50$, $d = .90$ (CI = .61–1.20), while TAU (pharmacological treatment) did not (p = .884). MCQ-30-NB predicted PHQ-PD in both treatment group (B = .42 ($SE = .10$) (CI = .22–.61), $p = .000$) and in the intention-to-treat group (B = .42 ($SE = .09$ (CI = .04–0.59), $p <$ .000), but did not predict GAD-7, PHQ-9 or PHQ-15 ($p > .05$).

Appendix 1: Quality assessment scores

	Jelinek et al. (2017)		Hjemdal et al. (2019)		Sürig et al. (2021)		Hoffart et al. (2018)		Johnson & Hoffart (2018)		Johnson et al. (2020)		McEvoy & Perini (2009)		McEvoy et al. (2009)		Nordahl et al. (2017)	
Criteria	R1	R2	R1	R2	R1	R2	R1	R2	R1	R2	R1	R2	R1	R2	R1	R2	R1	R2
Explicit theoretical framework	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3
Statement of aims/objectives in main body of report	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3
Detailed recruitment data	2	2	3	3	2	3	3	3	3	3	3	2	3	2	3	2	3	3
Clear description of research setting	3	3	2	2	3	3	3	3	3	3	2	2	2	2	3	2	2	3
Description of procedure for data collection	3	3	3	3	1	1	3	2	3	2	3	3	3	2	2	2	2	2
Rationale for choice of data collection tool(s)	2	2	1	1	2	2	2	2	2	2	2	2	2	2	3	2	3	2
Statistical assessment of reliability and validity of measurement tool(s)	3	2	3	3	3	3	2	2	1	1	2	2	3	3	3	3	3	3
Evidence of sample size considered in terms of analysis	1	1	1	1	1	0	0	1	1	2	1	1	1	0	0	0	1	1
Representative sample of target group of a reasonable size	1	2	2	2	1	1	2	2	2	2	1	1	1	2	1	2	1	0
Good justification for analytic method selected	2	2	1	1	2	1	3	3	3	3	2	2	2	2	2	2	2	2
Fit between stated research question and method of data collection	3	3	3	3	3	3	3	3	3	3	2	3	2	3	3	3	2	2
Fit between research question and method of analysis	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3
Evidence of user involvement in design	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Strengths and limitations critically discussed	3	2	2	2	3	3	3	3	2	2	2	2	3	3	2	2	1	2
Total Score	32	31	30	30	30	29	33	33	32	32	29	29	31	30	31	29	29	29

	Solem et al. (2009)		Besiroglu et al. (2011)		Grøtte et al. (2015)		Solem et al (2015)		Park et al. (2020)		Sunde et al. (2021)		Hansmeier et al. (2021)		Fernie et al. (2015)		Jacobsen et al. (2016)	
Criteria	R1	R2	R1	R2	R1	R2	R1	R2	R1	R2	R1	R2	R1	R2	R1	R2	R1	R2
Explicit theoretical framework	3	3	2	2	3	3	3	3	3	3	3	3	3	3	3	3	3	3
Statement of aims/objectives in main body of report	3	3	2	2	3	3	3	3	3	3	3	3	3	3	3	3	3	3
Detailed recruitment data	2	2	2	1	2	2	3	3	3	3	2	2	2	2	2	2	3	3
Clear description of research setting	3	2	1	1	2	2	3	3	2	2	2	3	2	2	3	3	2	2
Description of procedure for data collection	2	2	2	2	1	1	2	2	3	3	3	2	2	2	2	2	1	2
Rationale for choice of data collection tool(s)	1	2	1	1	2	2	2	2	1	1	2	2	2	2	3	2	1	1
Statistical assessment of reliability and validity of measurement tool(s)	3	3	1	1	3	3	3	2	0	0	3	3	3	3	3	2	3	3
Evidence of sample size considered in terms of analysis	2	2	0	0	1	1	1	1	0	0	1	2	1	1	1	1	1	1
Representative sample of target group of a reasonable size	1	2	1	2	2	2	2	2	1	2	2	2	1	1	1	2	2	2
Good justification for analytic method selected	1	2	1	1	2	2	1	1	2	2	3	3	2	1	3	2	2	1
Fit between stated research question and method of data collection	3	3	3	3	3	3	3	3	3	3	2	3	3	3	2	3	3	2
Fit between research question and method of analysis	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3
Evidence of user involvement in design	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Strengths and limitations critically discussed	1	2	1	1	3	3	2	2	3	2	3	2	2	2	2	2	3	2
Total Score	28	31	20	20	30	30	31	30	27	27	32	33	29	28	31	30	30	28

	Jacobsen et al. (2020)		Brugnera et al. (2021)		Parker et al. (2020)		Spada et al. (2009)		McEvoy et al. (2017)		Sharpe et al. (2019)		Newby et al. (2014)		Corpas et al. (2021)	
Criteria	R1	R2	R1	R2	R1	R2	R1	R2	R1	R2	R1	R2	R1	R2	R1	R2
Explicit theoretical framework	3	3	3	3	3	3	3	3	3	3	3	3	3	3	1	1
Statement of aims/objectives in main body of report	3	3	3	3	2	2	3	3	3	3	3	3	3	3	3	2
Detailed recruitment data	2	3	2	2	2	2	3	3	3	3	2	2	2	3	2	3
Clear description of research setting	2	2	3	3	2	2	2	2	3	3	2	3	3	3	3	3
Description of procedure for data collection	2	2	2	2	2	2	2	2	3	3	2	2	3	3	3	3
Rationale for choice of data collection tool(s)	1	1	1	1	3	3	2	2	3	2	3	3	3	3	2	2
Statistical assessment of reliability and validity of measurement tool(s)	3	2	2	3	2	2	3	2	3	3	1	1	3	3	3	3
Evidence of sample size considered in terms of analysis	1	1	3	3	1	1	1	0	3	3	1	1	2	2	3	3
Representative sample of target group of a reasonable size	2	2	2	3	1	1	1	2	1	2	2	2	1	2	2	3
Good justification for analytic method selected	2	2	3	3	1	2	2	1	3	3	1	1	3	3	2	2
Fit between stated research question and method of data collection	3	3	3	3	2	2	3	3	1	2	2	2	3	2	2	2
Fit between research question and method of analysis	3	3	3	3	2	2	3	3	3	3	3	3	3	3	3	3
Evidence of user involvement in design	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Strengths and limitations critically discussed	1	1	3	2	3	2	2	2	3	3	3	2	1	1	3	2
Total Score	28	28	33	34	26	26	30	28	35	36	28	28	33	34	32	32

Appendix 2: Quality assessment scoring guidelines

Criteria	0 = Not at all	1 = Very slightly	2 = Moderately	3 = Complete
Explicit theoretical framework	No mention at all.	Reference to broad theoretical basis.	Reference to a specific theoretical basis.	Explicit statement of theoretical framework and/or constructs applied to the research.
Statement of aims/objectives in main body of report	No mention at all.	General reference to aim/objective at some point in the report including abstract.	Reference to broad aims/objectives in main body of report.	Explicit statement of aims/objectives in main body of report.
Detailed recruitment data	No mention at all.	Minimal recruitment data, e.g. no. of questionnaire sent and no. returned.	Some recruitment information but not complete account of the recruitment process, e.g. recruitment figures but no information on strategy used.	Complete data regarding no. approached, no. recruited, attrition data where relevant, method of recruitment.
Clear description of research setting	No mention at all.	General description of research area and background, e.g. 'in primary care'.	General description of research problem in the target population, e.g. 'among GPs in primary care'.	Specific description of the research problem and target population in the context of the study, e.g. nurses and doctors from GP practices in the east midlands.
Evidence of sample size considered in terms of analysis	No mention at all.	Basic explanation for choice of sample size. Evidence that size of the sample has been considered in study design.	Evidence of consideration of sample size in terms of saturation/information redundancy or to fit generic analytical requirements.	Explicit statement of data being gathered until information redundancy/saturation was reached or to fit exact calculations for analytical requirements.
Representative sample of target group of a reasonable size	No statement of target group.	Sample is limited but represents some of the target group or representative but very small.	Sample is somewhat diverse but not entirely representative, e.g. inclusive of all age groups, experience but only one workplace. Requires discussion of target population to determine what sample is required to be representative.	Sample includes individuals to represent a cross section of the target population, considering factors such as experience, age and workplace.
Statistical assessment of reliability and validity of measurement tool(s) (Quantitative only)	No mention at all.	Reliability and validity of measurement tool(s) discussed, but not statistically assessed.	Some attempt to assess reliability and validity of measurement tool(s) but insufficient, e.g. attempt to establish test–retest reliability is	Suitable and thorough statistical assessment of reliability and validity of measurement tool(s) with reference to the quality of evidence as a result of the measures used.

			unsuccessful but no action is taken.	
Description of procedure for data collection	No mention at all.	Very basic and brief outline of data collection procedure, e.g. 'using a questionnaire distributed to staff'.	States each stage of data collection procedure but with limited detail, or states some stages in details but omits others.	Detailed description of each stage of the data collection procedure, including when, where and how data were gathered.
Rationale for choice of data collection tool(s)	No mention at all.	Very limited explanation for choice of data collection tool(s).	Basic explanation of rationale for choice of data collection tool(s), e.g. based on use in a prior similar study.	Detailed explanation of rationale for choice of data collection tool(s), e.g. relevance to the study aims and assessments of tool quality either statistically, e.g. for reliability & validity, or relevant qualitative assessment.
Fit between stated research question and method of data collection (Quantitative)	No research question stated.	Method of data collection can only address some aspects of the research question.	Method of data collection can address the research question but there is a more suitable alternative that could have been used or used in addition.	Method of data collection selected is the most suitable approach to attempt answer the research question
Fit between research question and method of analysis	No mention at all.	Method of analysis can only address the research question basically or broadly.	Method of analysis can address the research question but there is a more suitable alternative that could have been used or used in addition to offer greater detail.	Method of analysis selected is the most suitable approach to attempt answer the research question in detail, e.g. for qualitative IPA preferable for experiences vs. content analysis to elicit frequency of occurrence of events, etc.
Good justification for analytical method selected	No mention at all.	Basic explanation for choice of analytical method	Fairly detailed explanation of choice of analytical method.	Detailed explanation for choice of analytical method based on nature of research question(s).
Evidence of user involvement in design	No mention at all.	Use of pilot study but no involvement in planning stages of study design.	Pilot study with feedback from users informing changes to the design.	Explicit consultation with steering group or statement or formal consultation with users in planning of study design.
Strengths and limitations critically discussed	No mention at all.	Very limited mention of strengths and limitations with omissions of many key issues.	Discussion of some of the key strengths and weaknesses of the study but not complete.	Discussion of strengths and limitations of all aspects of study including design, measures, procedure, sample & analysis.

Appendix 2: Scoring guidelines for rating The Quality Assessment Tool for Studies of Diverse Designs (Sirriyeh et al., 2012).

Appendix 3: Cohen's Kappa of inter-rater reliability on quality assessment

Cohen’s Kappa correlation coefficients and confidence boundaries

	Lower	Estimate	Upper
Unweighted kappa	.62	.68	.75
Weighted kappa	.89	.89	.89

Number of subjects = 338

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