

# Early Maladaptive Schemas in Anxiety Disorders

*An Investigation of Schemas' Relation to  
Symptoms in a Complex Sample*

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Cand. psychol. thesis  
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# Summary

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**Title:** Early Maladaptive Schemas in Anxiety Disorders - An Investigation of Schemas' Relation to Symptoms in a Complex Sample

**Supervisors:** Asle Hoffart and Sverre Urnes Johnson

**Objective:** Anxiety disorders are wide-spread and often long-lasting and highly debilitating to sufferers. Therefore, it is important to make efficient therapeutic efforts to alleviate them. Therapeutic efforts may be more efficient if directed at mechanisms underlying anxiety disorders. The main objective of this longitudinal study is to elucidate the relation between schemas, a proposed underlying cognitive structure, and symptoms in complex anxiety disorders. More specifically, the relation of schemas to symptoms are investigated concurrently and prospectively. It is examined whether the relationship between schemas and symptoms is still significant when including another proposed cognitive foundation, namely metacognition, in the analyses. Lastly, the relative efficiency of the current treatment conditions in producing changes in metacognitions, schemas and symptoms is investigated.

**Methods:** Data in the current study are part of an RCT comparing the effects of metacognitive therapy (MCT) and cognitive behavioural therapy (CBT) for complex anxiety disorders. Participants were recruited from the Department of anxiety disorders at Modum Bad in Norway. The participants were randomised to either diagnosis-specific CBT or transdiagnostic MCT. Current analyses are based on computer-administered self-report questionnaires at pre-treatment, post-treatment, and 1-year follow-up. Schemas, metacognitions and symptoms were assessed by the Young Schema Questionnaire-Short Form (YSQ-S1), Metacognitions Questionnaire-30 (MCQ-30), and Beck Anxiety Inventory (BAI), respectively. Multilevel models were used to analyse the data. Data were disaggregated into within- and between person effects when applicable. The current study is the first to compare between- and within-person effects of schemas and metacognitions on anxiety symptoms.

**Results and conclusions:** Schemas are related to anxiety symptoms in complex anxiety disorders. There were significant concurrent between- and within-person relationships between symptoms and schema-endorsement. The within-person relationship of schema-endorsement and concurrent anxiety symptoms upheld even when between- and within

person effects of metacognitions were included in the analysis. The within-person effect of metacognitions was also significant. This indicates that within-person changes in both EMSs and metacognitions uniquely impact concurrent anxiety symptoms. Treatment condition significantly impacted the degree of decline in symptoms and metacognitions during treatment, and showed a trend towards affecting decline in EMSs in the same direction. In these analyses, patients receiving MCT showed greater declines than those receiving CBT. However, this effect was not apparent in analyses across treatment and follow-up.

In conclusion, the significant concurrent within-person relationships in this study indicate that changing both schemas and metacognitions can cause symptom-relief. The lack of lasting difference between treatment conditions imply that both EMSs and metacognitions can be changed by different interventions.





# Preface

I would like to thank my supervisors for continuing to challenge, direct, redirect, and guide me until the very end. Thank you for installing the confusion in me that sparked desire and extensive efforts to fully comprehend the analyses I were to carry out. After the first few sessions, I came home with a list of miss-spelled words I had no idea what meant. Now, a bright new world of disaggregation, heteroscedasticity and autoregressive variance structures has opened up to me.

Asle, thank you for your mainly concentrated presence, but also your occasional digressions such as discussing segments from the 90s hit-show “Lille Lørdag”. Sverre, thank you for your limitless availability via e-mail and phone-conferences from your car at 07:30 AM, and for drawing my attention to the fact that there are not 100 I’s in thesis.

I would also like to thank Sverre for giving me the opportunity to work as a research assistant in his project. In doing so, I gained valuable insight into psychotherapy research, and extensive experience in diagnostics and rating of adherence and competence in CBT-sessions.

Gratitude is also due towards the participants in the study. Thank you for spending hours completing questionnaires, and answering the hundreds of questions I and the other research assistants posed during the three rounds of diagnostics. Your openness and your willingness to contribute to research in a distressing time of your lives has been humbling and inspiring.

Thank you, thesis. Coupled with my tendency to procrastinate (a possible avoidant coping response to a Failure schema, or a CAS-directed behaviour to stop worrying about the thesis?), you have led to several positive outcomes. Thank you for leading me to dance more, play more guitar, refurbishing a table, looking at all the ceiling lamps on the internet twice before ordering, and much, much more. I am forever grateful.

Lastly, I would like to thank my husband for being even more patient than normal towards my occasionally strange behaviour in working with this thesis. Thank you for your love, compassion, support, grocery shopping, pizza-baking, and numerous bags of Hockypucker and Pulverpadder. Thank you for being there for me whenever I needed you.



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# 1 Introduction

Anxiety disorders are characterized by prolonged internal agitation, tension and fear that is exaggerated relative to the objective danger at hand (Malt & Malt, 2012). Key cognitive features are a fixation on danger and an underestimation of ability to cope with danger (Wells, 1997). Anxiety disorders are highly prevalent, with an approximated global point prevalence of 7.3% (Baxter, Scott, Vos, & Whiteford, 2013). The life-time prevalence of different anxiety disorders was found to be 4.5-14.4% in a Norwegian sample (Kringlen, Torgersen, & Cramer, 2001), and 2.3–15.6% in an American sample (Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). Anxiety disorders have debilitating effects on sufferers, resulting in 26 million disability-adjusted life-years<sup>1</sup> worldwide (Whiteford et al., 2013). Therefore, giving effective treatment for anxiety disorders is a priority. Identifying and targeting underlying elements could lead to more focused and efficient therapeutic efforts, with more lasting effects. In this thesis, two proposed underlying cognitive elements, namely *schemas* and *metacognitions*, will be examined.

Within the cognitive branch of psychology, schemas have been recognized as central to the occurrence and recurrence of psychopathology since Beck's (1963, 1964) writings on their role in depression. Beck claimed that schemas were the source of the stream of negative thoughts and concomitant depressed mood characterizing individuals with depressive disorders (Beck, 1963). Despite being prescribed a central role to pathology for more than fifty years (Riso & McBride, 2007), it is not clear how or to what extent schema-change relates to symptom relief in anxiety disorders. Therefore, one cannot conclude that one should target schemas in treatment of anxiety disorders. Investigation of the relationship between schemas and psychopathology is a main focus of this thesis.

The idea that metacognition underlies psychopathology is more recent, primarily entering the field of clinical psychology with the writings of Wells and Matthews on the self-regulatory executive function (S-REF) model (1994). Although metacognitions' role in psychopathology had been written about earlier as well (e.g., Hartman, 1983), it was not made into a separate psychological theory and therapy until the works of Wells and Matthews. In metacognitive theory, metacognitions are considered the cognitive foundation of psychopathology. Schema-beliefs are mainly considered consequences of activity directed by metacognitions, as are symptoms of anxiety. Based on this assumption, treatment of anxiety disorders should be effective if one focuses on, and manages to change

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<sup>1</sup> A combination of years lost due to premature mortality and years lived with disability

metacognitions. There is some support for this claim, as metacognitively focused treatment has proved to be highly efficacious in treating anxiety and depressive disorders (Normann, van Emmerik, & Morina, 2014). However, one cannot conclude that metacognitions are “more core” and should be primary focus in treatment until one has directly compared the influence of schemas and metacognitions. This has not yet been done in a clinical anxiety sample<sup>2</sup>.

The main focus of this thesis will be on elucidating the relation between anxiety symptoms and maladaptive schemas, as measured by self-reported endorsement of schema-relevant statements. According to schema theory (Young, Klosko, & Weishaar, 2003), this relationship is especially relevant in long-standing, treatment-refractory cases. Therefore, the current sample of complex anxiety patients (see [Table 2](#)) should be well-suited to examine how schemas relate to anxiety symptoms. The current study is also suited to explore whether schemas play the vital role stated in traditional cognitive psychology, or a peripheral role to the core concept of metacognitions, as assumed in metacognitive theory

## 1.1 Outline

The thesis begins with an introduction to the schema-concept in psychology, to situate the subject matter in a larger context. Ensuing, I focus on the specific construct of Early Maladaptive Schemas (EMSs), as defined by Young (2003). To set EMSs apart from other cognitive constructs, I will account for their specific developmental origin, how they are maintained and changed, and also how they relate to pathology. Lastly, the role designated to schemas and potential schema-changing processes in the treatments in the current study are accounted for. This is done to examine whether there is reason to expect that EMSs will change during the current treatment, a matter that of significance to what hypotheses will be stated. After the presentation of EMSs, the concept of metacognition and how it relates to pathology is introduced. Based on the theoretical background, Chapter 2 culminates in the statement of the hypotheses and research questions that will be investigated throughout the rest of the thesis. Methods and analytical procedures are accounted for in Chapter 3 and results are stated in Chapter 4. Chapter 5 is devoted to discussion of the findings, limitations of this study and directions for future research.

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<sup>2</sup> As of February 2017, I found only two studies that considered metacognitions and schemas simultaneously (Amouzadeh & Aghili, 2016; Zirakbash, Naderi, & Enayati, 2015). However, these were published in dubious journals not contained in the Directory of Open Access Journals (DOAJ) database and neither investigated the relative importance of schemas and metacognition to anxiety longitudinally in clinical samples.

# 2 Theoretical Background

## 2.1 An Introduction to Schemas

*“The cautious and the rash; the student and the man of affairs; the subject doubting and the same subject confident never perceive alike, though they may all be faced by exactly the same situation, so far as external features go.”* (Bartlett, 1932)

The above quote speaks to the perception of simple stimuli in laboratory experiments. Yet, it is likewise applicable to the perception process in complex real-life situations. A researcher-duo giving a talk at a conference can serve as an example. The external conditions are exactly the same for both, but their perceptions of the situation are far apart. One researcher perceived denouncing glances during the talk, and the ensuing questioning as aggressively critical. Taunted by thoughts of inadequacy he feels dismal and concludes, as he usually does, that he is a failure. The other researcher perceived keen glances during the talk and great interest in the subject through the audience’s extensive questioning. Her perception of the situation leaves her in high spirits. Supported by contented thoughts about her performance she concludes that she is a talented person. As the different perceptions in this case do not emanate from the objective stimuli, they must stem from subjective factors. Within cognitive psychology, there is a longstanding tradition of turning to the notion of schemas to explain such subjective differences. There are numerous definitions of the term schema, one of which is that schemas are networks of knowledge that enable the person to understand and act upon its surroundings (James, Southam, & Blackburn, 2004).

When a schema-relevant situation occurs, the schema is activated and stored knowledge of what stimuli to attend to, how to understand it and what to do with it, is used to guide perception and action. Having these templates in place provides us with a quick, and often sufficiently close to correct, interpretation of the situation without expending a lot of mental energy (James et al., 2004). This is a major advantage in routine situations, such as when the researchers were driving to the conference. Their “driving-schemas” guided perception to relevant features, enabling them to navigate safely while simultaneously going over the pending talk in their heads. However, the schema-guided efficiency comes at a cost. It can misguide your perception and leave you with a schema-consistently distorted impression of a given situation (Wenzel, 2012). If we assume that the talk objectively went well, the first researcher’s impression can be said to be schema-consistently distorted. By

looking at the way his information processing was distorted, we can infer what kind of schema might have been at the handles during the time of the talk. It seems likely that being faced with a performance situation activated his “failure-schema”. This activation in turn led him to pay attention to schema-consistent stimuli (peoples’ denouncing glances) and to interpret the questioning as aggressively critical rather than engaged and spirited. The result of the schema-consistent perception and evaluation is nagging self-critical thoughts, negative emotions, a desire for future avoidance, and a reinforcement of the schema itself.

As can be seen in the above, schemas significantly impact how we think, feel and act. For more than fifty years, schemas have been recognized as important contributors to disturbances in thinking, emotion and behaviour. In 1964, Beck defined a schema as “a structure used for screening, coding, and evaluating impinging stimuli” (p. 562). Schemas were theorized to be stable structures making up the fundamental layer of cognition, a layer that mostly operates outside of conscious awareness (Riso & McBride, 2007). These fundamental structures are thought to give rise to intermediate beliefs and, at the most “superficial” and conscious level, automatic thoughts (Riso & McBride, 2007). Negative and maladaptive schemas lead to negative automatic thoughts (NATs). NATs, in turn, influence the person’s mood and behaviour in response to their surroundings (Wells, 1997).

Being relatively stable components of the cognitive organisation, schemas were designated an especially important role in the maintenance and recurrence of mental disorders such as depression (Beck, 1964). Beck’s cognitive model can be said to be a diathesis-stress model in which schemas make up the individual’s diathesis to pathology. As such, it was proposed that treatment should be aimed at altering schemas as it would lead to a reduction in NATs and in the recurrence of depressive episodes (Beck, 1964). However, the cognitively focused therapies that developed in the ensuing years mainly focused on challenging NATs and less core cognitive structures such as attributional styles (Riso & McBride, 2007). In the late eighties and early nineties, however, there was a renewed interest in working on the more “fundamental” layer, with schemas. One of the leading proponents of this renewed focus was Jeffrey Young (Riso & McBride, 2007).

## **2.2 Young’s Early Maladaptive Schemas**

In 1990, Young published his first clinical guide on a schema-focused approach to treating complex pathology. Through clinical experience, he found that complex patients with personality disorders and longstanding symptom disorders rarely fulfilled prerequisites of



successful short-term cognitive therapy. They had trouble accessing emotions, thoughts and beliefs, and engaging in a good therapeutic alliance in a few sessions. They could not easily circumscribe a problem-focus, and were not motivated to do homework and learn self-control strategies. In addition, the problems were often ego-syntonic and very resistant to change (Young et al., 2003). To face these challenges, Young developed schema therapy as clinical guideline. Schema therapy builds on Beck's (1967) theory of depression, integrating elements from amongst others gestalt, attachment and psychodynamic theory to better meet the needs of the complex patients not responding to short-term cognitive therapy (Young et al., 2003).

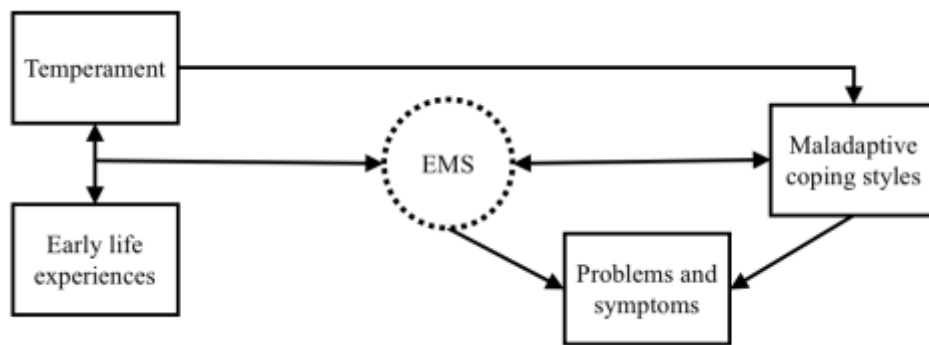
The core of Young's schema therapy is the construct of Early Maladaptive Schemas (EMS; Young et al., 2003). An EMS is defined as a "broad, pervasive theme or pattern comprised of memories, emotions, cognitions, and bodily sensations regarding oneself and one's relationship with others" (Young et al., 2003, p. 7). EMSs are considered organizing principles for the person's emotional, cognitive, behavioural and interpersonal functioning (Young et al., 2003). However, EMSs do not guide functioning at all times. They are only activated when the person encounters schema-relevant situations. An example is the male researcher, mentioned above, whose failure-schema was activated when faced with a performance situation. In non-performance situations, his failure schema may be dormant and some other schemas guide the way he perceives himself and others.

EMSs constitute a specific type of schemas. One of the defining features is that their etiology is clearly specified and situated early in life. This separates EMSs from other cognitive schema-constructs, such as Beck's (1964) schemas. Thus, an account of their origins and organization is necessary to understand the unique characteristics of EMSs.

### **2.2.1 Development of Early Maladaptive Schemas**

EMSs develop through an interaction between innate temperament and early life experiences (Figure 1). These experiences include not having one's core emotional needs met by getting too little or too much of a good thing (e.g., rules and limits), being traumatized, or selectively internalizing or identifying with abusive significant others (Young et al., 2003). Certain temperamental features make children more vulnerable to developing EMSs as they have more extensive emotional needs (Lockwood & Perris, 2012). In other cases, features such as a child's temperament can make it less likely that caregivers will satisfy their needs (Philipsen et al., 2016). For example, a withdrawn, easily upset and angry child may evoke less affection and warmth from caregivers, thus frustrating the child's need for nurturance.

Figure 1. *The origin of schemas.*



Note. Adapted from van Genderen, Rijkeboer, & Arntz, 2012.

Young postulated five core emotional needs whose frustration can lead to EMSs: (1) secure attachment, (2) autonomy, competence and sense of identity, (3) freedom to express valid needs and emotions, (4) spontaneity and play, and (5) realistic limits and self-control (Young et al., 2003). When the child's needs are consistently not met, either within the family or with their peers, schemas form on what to expect from others and what to think about oneself. To begin with, the schemas are useful guides because they concur with the child's environment. But as the environment changes, the rigid EMSs become misrepresentative and maladaptive. EMSs guide the person to perceive, think and respond in ways that lead to a continued experience of the noxious atmosphere from which the EMSs originated.

The five core emotional needs make up five schema domains, which in turn have several EMSs subsumed under them. EMSs are commonly measured by the Young Schema Questionnaire (YSQ). The most recent revision (YSQ-3) contains 18 EMSs clustered under the five domains (Young et al., 2003). As three of these are not measured in the current study, only the 15 relevant EMSs are included in the overview in [Table 1](#). Even though all the EMSs are assumed to form fundamental cognitive structures, there are some differences between them. Generally, the earlier formed EMSs are very potent and rigid as they are fully unconditional (Young et al., 2003). Beliefs from the Defectiveness schema about being intrinsically unlovable, holds no hope of ever having meaningful and loving relationships. EMSs that develop later in life, such as the schema Unrelenting Standards, holds some hope because they are conditional (Young et al., 2003). If the person manages to perform perfectly in every way, he or she could be worthy of love. Later developed EMSs are more amenable to change, although they are still rigid and hard to alter. To understand the rigidity of EMSs, one needs to know what processes underlie their maintenance. The next section pertains to schema-maintenance.

Table 1. *EMSs Organized Under Schema Domains<sup>a</sup> with Means and Standard Deviations<sup>b,c</sup>*

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***Disconnection and Rejection (DR)***

1. *Abandonment/Instability*: Perceives those available for support and connection as unstable and unreliable. ( $M = 2.77, SD = 1.33$ )
2. *Mistrust/Abuse*: Expectation that one will be intentionally taken advantage of, lied to, manipulated and hurt by others. ( $M = 2.14, SD = 1.09$ )
3. *Emotional Deprivation*: Expectation that one's need for normal emotional support will not be met. ( $M = 2.69, SD = 1.29$ )
4. *Defectiveness/Shame*: Feeling of being invalid or inferior in important respects or that one would be unlovable to significant others if exposed. ( $M = 2.22, SD = 1.24$ )
5. *Social Isolation*: Feeling of being isolated, different and/or not part of any group or community. ( $M = 2.67, SD = 1.35$ )

***Impaired Autonomy and Performance (IA)***

6. *Dependence/Incompetence*: Belief that one is unable to competently handle everyday responsibilities without considerable help. ( $M = 2.03, SD = 0.98$ )
7. *Vulnerability to Harm or Illness*: Exaggerated fear that disaster will strike at any time and that one is unable to prevent it. ( $M = 2.24, SD = 1.12$ )
8. *Enmeshment*: Excessive emotional involvement and closeness with significant others at the expense of full individuation. ( $M = 1.92, SD = 1.02$ )
9. *Failure*: Belief that one has failed, will fail, or is fundamentally inadequate relative to others when it comes to achievement. ( $M = 2.37, SD = 1.29$ )

***Impaired Limits (IL)***

10. *Entitlement*: Belief that one is superior, entitled to special rights and privileges, or not bound by socially normed rules of reciprocity. ( $M = 1.99, SD = 0.82$ )
11. *Insufficient Self-Control*: Pervasive difficulty or refusal to exercise sufficient self-control to achieve goals and restrict impulses. ( $M = 2.51, SD = 1.08$ )

***Other-directedness (OD)***

12. *Subjugation*: Excessive surrender of control to others, and subjugation of feelings or needs, because one feels coerced. ( $M = 2.66, SD = 1.25$ )
13. *Self-Sacrifice*: Excessive focus on voluntarily meeting the needs of others at the expense of fulfilling one's own, to avoid guilt, and maintain connection. ( $M = 3.29, SD = 1.16$ )

***Over-Vigilance and Inhibition (OI)***

14. *Emotional Inhibition*: Excessive inhibition of spontaneous action, communication and feeling to avoid disapproval, feelings of shame and losing control. ( $M = 2.53, SD = 1.18$ )
15. *Unrelenting Standards*: Belief that one must meet very high internalized standards of behaviour and performance to avoid criticism. ( $M = 3.32, SD = 1.20$ )

***Total score*** ( $M = 2.50, SD = 0.85^d$ )

---

*Note.* <sup>a</sup> Shortened version of Young and colleagues' (2003, p.14-17) list. <sup>b</sup> Scores for each EMS from YSQ range from 1-6. <sup>c</sup> Means and standard deviations are average scores on YSQ of 1291 patients from five Norwegian studies (Hoffart et al., 2005; Haaland et al., 2011; Lishaugen, 2007; Thimm, 2010, 2013) <sup>d</sup> SD for total scores only reported in one study (Haaland et al., 2011)

### **2.2.2 Maintenance of Early Maladaptive Schemas**

Schemas are maintained by the schema coping styles of avoidance, surrender and over-compensation (van Genderen, Rijkeboer, & Arntz, 2012; Young et al., 2003). The coping styles correspond to the three basic reactions all organisms have when faced with threat; flight, freeze and fight, respectively. In the case of EMSs, the threats are (1) not having one's core emotional needs met and (2) the intense negative affect that accompanies schema-activation (Young et al., 2003). An individual will often use more than one coping style over time, but there will be some consistency as temperamental features affect the propensity towards the different styles (Young et al., 2003). For example, an inhibited and passive temperament disposes the person to avoiding and surrendering coping efforts.

Schema avoidance encompasses behavioural, cognitive and emotional avoidance. That is, the individual tries to avoid triggering schema-relevant thoughts and feelings, oftentimes by repression. In addition, the person avoids behaviour that could lead to activation of a schema. Avoidance can be an effective strategy, but it has potentially high costs. Cognitive avoidance can in extreme cases lead to depersonalisation and compulsive behaviour in an attempt to gain control over one's thoughts (Young et al., 2003). Affective avoidance shelters the person from the extreme levels of affect caused by schema-activation, but it will often lead to more chronic, diffuse emotions and psychosomatic symptoms (Young et al., 2003). Behavioural avoidance can be very effective, but often entails limiting one's life. An example is a person with a Failure scheme who avoids all performance situations, in order to avoid activating beliefs regarding inevitable failure. Another downside to schema avoidance is that as the schemas are largely inactivated, they are not amenable to revision.

The coping style of schema surrender entails giving in to the schema. Emotionally, the person feels the full range of negative emotion elicited by schema-activation (Young et al., 2003). Cognitively, the person engages in selective attention to and overgeneralisation of schema-consistent information (Young et al., 2003). Human beings have an innate strive for consistency and a corresponding discomfort with inconsistency and unpredictability, especially in personally meaningful matters (Hart et al., 2009; Metin & Camgoz, 2011). The discomfort of inconsistency can lead people to prefer maintaining a predictably awful view of oneself and others over challenging these beliefs. Therefore, one automatically searches for information that fits one's schemas and emphasises its validity and generalizability (Young et al., 2003). Rather than changing our schemas to fit reality, we construe reality to fit our schemas (Beck, 1964). This also holds true for behavioural surrender in which the person acts

in ways that confirm the schema. An example is the man with a subjugation schema who marries a controlling and domineering wife. In doing so he recreates the subjugating setting from which the schema originated, leaving him with a predictably dismal inter- and intrapersonal world.

Schema overcompensation involves fighting schemas by behaving, thinking and feeling the opposite of what the schema dictates (Young et al., 2003). Working against the schemas can be healthy, but overcompensation is usually unproductive and takes little regard to how the compensation affects oneself and others (Young et al., 2003). In addition, overcompensation is rarely successful all the time. When it fails, the schemas resurface with tremendous emotional strength. An example is a person with a Defectiveness schema who constantly criticizes others while appearing to be perfect. When he gets in return what he dishes out, he breaks down and the schema resurfaces with intense feelings of shame, sadness and inferiority. This coping style does not incite schema-change as it involves shifting between denying the existence of the schema and being fully overwhelmed by it. The different ways in which schemas can be changed are accounted for in the ensuing section.

### **2.2.3 Change of Early Maladaptive Schemas – Schema Healing**

Achieving schema healing is the main goal of schema therapy (Young et al., 2003). Schema healing involves a reduction of intensity of the memories, emotions, bodily reactions and cognitions of the schema (Young et al., 2003). This leads schemas to be activated less often, and the remaining activation is less overwhelming and enduring. Young and colleagues (2003) propose utilizing a mix of cognitive, experiential, interpersonal and behavioural pattern-breaking techniques to promote schema healing.

Cognitive techniques are used to challenge the schema-distorted view of self and others, and the usefulness of the current coping strategies, by presenting contrary objective evidence. Achieving a more sympathetic understanding of oneself has proved to be important in order to achieve schema healing and symptom-relief (Hoffart, Versland, & Sexton, 2002). Often, the therapist will use guided discovery to examine and challenge the evidence the patient has for the schema (Hoffart et al., 2002). The contrary evidence can then be used to write a schema flash-card that the patient carries with her and repeats, especially when a schema is activated (Young et al., 2003). The various cognitive exercises are intended to improve the way patients habitually process information, so that the gains made in therapy can be maintained (Young et al., 2003). Experiential techniques are mainly used after the

cognitive interventions to make sure that the patient's new understanding is emotional as well as cognitive (Young et al., 2003).

The interpersonal interventions entails using the therapeutic relationship as an active vessel to promote change. The main interventions are empathic confrontation and limited reparenting (Young et al., 2003). In empathic confrontation, the therapist shows understanding and empathy for the patient whilst at the same time confronting the patient with the need to change. The degree of therapist empathic confrontation has been showed to be related to symptomatic distress in schema therapy (Hoffart et al., 2002). In limited reparenting, the therapist provides the patient with corrective experiences in which the patient's core emotional needs are met to a reasonable degree (Young et al., 2003).

Behavioural pattern-breaking consists of repeated training to replace the maladaptive schema-driven behaviour-patterns of avoidance, surrender and overcompensation (Young et al., 2003). The goal is to replace these behaviours with more adaptive ways of responding to the schemas. Behavioural change is important although behaviour is not part of the EMS itself, as maladaptive behavioural patterns are strong schema-maintaining factors.

Thus far, an account has been given of what EMSs are, how they develop, what makes them rigid and how they can be changed. With this background in mind, we turn to EMSs' relationship to psychopathology.

## **2.3 EMSs and Psychopathology**

There is a significant body of research supporting a relationship between the degree of endorsement of different EMSs and various forms of psychopathology. Links have been found between schema-endorsement and symptoms of eating disorders (Cullum, 2009; Damiano, Reece, Reid, Atkins, & Patton, 2015; Dingemans, Spinhoven, & van Furth, 2006; Jones, Leung, & Harris, 2007), personality disorders (Barazandeh, Kissane, Saeedi, & Gordon, 2016; Hoffart et al., 2005; Kriston, Schäfer, Jacob, Härter, & Hölzel, 2013), maladaptive personality and character traits (Halvorsen et al., 2009; Thimm, 2010), and measures of anxiety and depression in clinical and non-clinical samples (e.g., Baranoff, Oei, Cho, & Kwon, 2006; Calvete, Estévez, López de Arroyabe, & Ruiz, 2005; Calvete, Orue, & Hankin, 2013; Cui, Lin, & Oei, 2011; Glaser, Campbell, Calhoun, Bates, & Petrocelli, 2002; Hawke & Provencher, 2012; Hoffart et al., 2005; Saritaş & Gençöz, 2011; Schmidt, Joiner, Young, & Telch, 1995; Welburn, Coristine, Dagg, Pontefract, & Jordan, 2002). Research has also demonstrated that changes in schema beliefs are related to symptom-relief in samples of

patients with mixed psychiatric disorders (van Vreeswijk, Spinhoven, Eurelings-Bontekoe, & Broersen, 2014; Welburn, Dagg, Coristine, & Pontefract, 2000), personality disorders (Nordahl, Holthe, & Haugum, 2005; Schaap, Chakhssi, & Westerhof, 2016), eating disorders (Cullum, 2009), depression (Halford, Bernoth-Doolan, & Eadie, 2002; Wegener, Alfter, Geiser, Liedtke, & Conrad, 2013) and anxiety (Borge et al., 2008; Cockram, Drummond, & Lee, 2010; Halford et al., 2002). Randomized controlled trials finding that schema therapy is efficient in treating personality disorders speak to the link between EMSs and long-standing, hard to treat pathology (Dickhaut & Arntz, 2014; Farrell, Shaw, & Webber, 2009; Giesen-Bloo, van Dyck, Spinhoven, & et al., 2006; Schaap et al., 2016).

There is also research supporting the claim that EMSs are stable diathetic entities to psychopathology, not reducible to temporary cognitions accompanying current symptoms. In a sample of depressed individuals, the relative stability of most EMSs was found to be moderate to high over a 2.5-5-year interval, even when controlling for depression at both measurements (Riso et al., 2006). In addition, most of the EMSs were as stable as, or even more so, than the well-established personality trait neuroticism (Riso et al., 2006). Another study found significant moderate relative stability of most EMSs over a 9-year interval (C. E. Wang, Halvorsen, Eisemann, & Waterloo, 2010). Furthermore, EMSs were found to be higher in previously depressed currently asymptomatic individuals than in never depressed individuals (C. E. Wang et al., 2010).

In summary, EMSs have been found to be related to symptomatic distress across a wide range of psychological disorders, and changes in EMSs appear to be related to symptom-relief. In addition, there is evidence to support the claim that schemas are not merely by-products of current pathology, but rather stable diathetic structures that can lie dormant and be reactivated to cause recurrence of pathology. Next, the focus is narrowed in on the relationship between EMSs and symptoms in anxiety disorders.

### **2.3.1 EMSs in Anxiety Disorders**

Patients with anxiety disorders have been found to score higher than healthy controls on most EMSs, as measured by different versions of the YSQ (Hawke & Provencher, 2011). EMSs from the schema domains Impaired Autonomy (IA), Disconnection and Rejection (DR), Other-Directedness (OD) and Over-Vigilance and Inhibition (OI) have all been found to be related to anxiety symptoms general (Glaser et al., 2002; Halford et al., 2002; Hawke & Provencher, 2011; Hovland, 2007; McGinn, Cukor, & Sanderson, 2005; Welburn et al.,

2002). The EMSs most strongly related to symptoms varies somewhat across the individual anxiety disorders. The patients in the current sample have primary diagnoses of panic disorder with or without agoraphobia (PD/A), post-traumatic stress disorder (PTSD) and social anxiety disorder (SAD). In the following, the literature on EMSs associated with each disorder will be briefly reviewed in order to evaluate what EMSs may be most relevant in the current sample.

The Vulnerability schema in the IA domain has consistently been found to be strongly related to symptoms of PDA (Hedley, Hoffart, & Sexton, 2001; Hinrichsen, Waller, & Dhokia, 2007; Hinrichsen, Waller, & Emanuelli, 2004; Hoffart et al., 2005; Kwak & Lee, 2015). This connection makes sense, as the schema content closely resembles the diagnostic features of PD/A of sudden, unexpected onset of panic, and fear of the next time catastrophe will strike, listed in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 2000).

Two studies examining the relationship between EMSs and PTSD coincided in finding that Emotional Inhibition, Social Isolation and Mistrust were significantly related to PTSD-symptoms (Cockram, 2009; Dutra, Callahan, Forman, Mendelsohn, & Herman, 2008). The Defectiveness schema was also related to symptoms in two studies (Cockram, 2009; Price, 2007). In addition, in only one study the Vulnerability-schema (Cockram, 2009), and Enmeshment, Dependence and Failure schemas (Price, 2007) have been found to be strongly related to PTSD-symptoms. The combination of seeing the world as dangerous (Mistrust, Vulnerability) and oneself as unable to cope (Dependence, Failure) is a cognitive trademark of PTSD (Schnyder et al., 2015). The Social Isolation schema resembles the diagnostic feature of feeling estranged and detached (American Psychiatric Association, 2000). The schema-endorsement seen in people with PTSD could be pre-existing, and the trauma could exacerbate their influence on the person's life, as Young suggests (2003). It is also possible that pre-existing EMSs lead to maladaptive coping after trauma (such as not seeking social support), thus increasing risk of PTSD. As none of the above studies measured EMSs before trauma and development of PTSD, one cannot ascertain whether EMSs lead to PTSD, or whether pathology leads to temporarily elevated EMSs.

The following schemas have been found to be most consistently associated with SAD; Defectiveness (Hinrichsen et al., 2007; Lishaugen, 2007; Pinto-Gouveia, Castilho, Galhardo, & Cunha, 2006), Emotional Inhibition (Hinrichsen et al., 2004; Lishaugen, 2007) and Abandonment (Hinrichsen et al., 2007; Hinrichsen et al., 2004). These findings indicate that beliefs about being unlovable and fundamentally defect (Defectiveness), that others cannot be



counted on for support (Abandonment), and that one needs to rigorously control expression of emotion and impulses to gain acceptance (Emotional Inhibition) are central SAD. In addition, Mistrust (Pinto-Gouveia et al., 2006) and Social Isolation (Lishaugen, 2007) have been found to be associated with SAD in one study each. However, as none of these studies were longitudinal, one cannot know whether the schema-endorsement preceded the onset of SAD, or if the beliefs developed as a consequence of the disorder.

Except for the association of Vulnerability and PD/A, the EMS-profiles are not unanimous. For PTSD and SAD, a multitude of EMSs have emerged as significant predictors of symptomatic distress. In sum, most EMSs have been connected to anxiety symptoms in general and to symptoms of the three current disorders. Therefore, there is reason to assume that most EMSs will be related to anxiety symptoms in highly comorbid samples of patients with anxiety disorders, such as the current sample (see [Table 2](#)).

### **2.3.2 Treatment of Anxiety Disorders and Change in EMSs**

A few studies have investigated changes in EMSs during treatment and their relation to changes in anxiety symptoms. Decline in schemas in the Impaired Autonomy domain explained 26% of the variance in PTSD-symptom reduction in patients with PTSD receiving schema therapy (Cockram et al., 2010). Changes in EMSs were also found to be related to changes in anxiety symptoms in a mixed clinical sample given CBT (Halford et al., 2002). Changes in schemas relevant to SAD<sup>3</sup> concurred with changes in symptoms in a sample of patients with SAD receiving either cognitive or interpersonal therapy (Borge et al., 2008). However, no analyses were made as to the impact of change in EMSs on symptom-change (Borge et al., 2008). In a study of patients with PD/A given 10 weeks of CBT, no significant changes in EMSs were found at posttreatment<sup>4</sup>, despite full recovery of anxiety symptoms (Rusinek, Graziani, Servant, Hautekeete, & Deregnacourt, 2004). It can thus seem that EMSs are related to symptomatic distress, but that it is not necessary to change EMSs in order to achieve improvement in symptoms. Within schema theory, the notion of a dormant schema can explain how distress-levels can change without change in schema-endorsement. However, as the schemas have not been challenged and changed, they can be reactivated to cause symptoms at a later stage. Unfortunately, Rusinek and colleagues' (2004) study did not include a follow-up measurement that could enlighten this issue. As this study contains a

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<sup>3</sup> Large changes in most EMSs in the DR and IA domains, reported in Lishaugen (2007).

<sup>4</sup> There was a significant change in Emotional Deprivation for the men in the study. However, the same change was found in a control group not receiving treatment. Thus, the change cannot be attributed to the treatment.

follow-up measurement, it can allow for such investigation. An important presupposition for detection of a relationship between change in EMSs and change in symptoms is that there are changes in the EMSs. Is there reason to expect schema-change in the treatment conditions in the current study?

### **2.3.3 EMSs in the Current Treatment Conditions**

Schema therapy was developed because of a perceived lack of a therapeutic framework to treat complex patients with strong EMSs. Yet, several studies have showed that EMS-endorsement declines during non-schema-therapy treatments (Borge et al., 2008; Fitzsimmons et al., 2008; Wegener et al., 2013; Welburn et al., 2002). As schema therapy is an integrative therapy based on elements from several other treatment directions, it is reasonable to assume that these other therapies contain some potentially schema-changing elements. In the following, the role designated to schemas and processes that can be expected to affect schema-endorsement in the current treatment protocols are presented.

In the PTSD-protocol, Prolonged Exposure (PE), by Foa, Hembree and Rothbaum (2007), erroneous beliefs regarding the dangerousness of the world and one's incompetence are assigned a central role in the development and maintenance of PTSD (Schnyder et al., 2015). These beliefs are not EMSs, but there is a resemblance in content to schema-beliefs in the Impaired Autonomy and Disconnection and Rejection domains. Changing these beliefs constitutes an important vessel for change in PE (Zalta et al., 2014). The beliefs are worked on through exposure (activation of emotions and beliefs) and subsequent cognitive restructuring processing (Foa et al., 2007). As such, PE can be said to utilize both cognitive and experiential methods that are potentially schema-changing. In addition, the reversal of avoidance, resembling behavioural pattern-breaking, is a key element in PE (Foa et al., 2007). Hence, the PTSD-protocol can be expected to cause schema-change by working directly with beliefs through experiential and cognitive techniques, and by challenging maladaptive coping strategies of cognitive, emotional and behavioural avoidance.

The conceptualisation of PD/A in Clark's (1986) protocol does not emphasize the role of schema-like beliefs. The focus is mainly on modifying catastrophic misinterpretations of the significance of bodily symptoms (e.g., "Heart palpitations mean that I am having a heart attack"; "Feeling light-headed means I will faint") (Clark, 1986). Nevertheless, there are some elements of this protocol that can also affect schema beliefs. As explicated by Wells (1997), challenging the notion that disaster can strike at any moment, and that the individual

will lose control and not be able to handle it, is a main focus of the protocol. This emphasis can be expected to affect schemas in the Impaired Autonomy domain, as they revolve around a fear of disaster that one will not be able to cope with. Both verbal, behavioural and experiential techniques are utilized to this end (Wells, 1997). In sum, there are some potentially schema-changing elements in the protocol, even though schemas are not considered central.

In the SAD-protocol, schema beliefs are incorporated as a target of intervention at later stages of the treatment (Wells, 1997, pp. 197-199). It is recommended to use verbal reattribution, behavioural experiments, and a number of schema-theoretical interventions such as using schema-flashcards, to achieve schema-change (Wells, 1997). Because schema-beliefs are part of the conceptualisation of the disorder and there are schema-specific interventions in the protocol, this treatment can be expected to reduce endorsement of the EMSs targeted in therapy.

In metacognitive therapy (MCT), there is no emphasis on schema beliefs. However, there is focus on modifying attentional allocation, reversing avoidance behaviours and reducing rumination and worry (Wells, 2009). Attentional fixation has some similarities with the attentional bias in the “surrendering” coping response to schemas. Increasing attentional flexibility may contribute to alteration of schemas, as it could widen the person’s information-processing beyond what is schema-consistent. Worry is considered a surrendering coping strategy in response to the Vulnerability schema (Young et al., 2003, p. 150). Therefore, work on reducing worry could affect maintenance, and consequently endorsement, of the Vulnerability schema. And, as previously mentioned, reversing avoidance can be seen as behavioural pattern-breaking which is considered to be schema-changing.

In conclusion, both MCT and CBT treatments work on processes that may affect schemas. Therefore, some EMS-decrease can be expected to ensue from therapy. However, given that previous studies have found modest decreases in EMSs after short-term therapy and based on the supposed rigidity of EMSs, there is reason to expect a rather small decrease.

Thus far, the focus of this thesis has been on schemas and schema-beliefs. That is, on how thoughts about oneself, others and the world shape how one functions and affect one’s psychological well-being. In the next section, the focus will be on how thoughts about cognition can impact on one’s well-being and functioning. This includes giving an account of what metacognitions are and how they impact on processes and symptoms of pathology.

## **2.4 Contending Core Cognition - Metacognitions**

Metacognition is defined as “the internal cognitive factors that control, monitor, and appraise thinking” (Wells, 2009, p. viii). The concept of metacognition can be subdivided into three interdependent aspects: knowledge and beliefs, experiences, and strategies. Metacognitive knowledge comprises beliefs about cognition in the form of declarative statements (e.g., “Worrying is harmful to me”) and implicit plans for processing (e.g., attentional allocation and memory search) (Wells, 2009). A metacognitive experience is based on the individual’s assessment of one’s own cognitive states and events (e.g., being worried about one’s worrying) (Wells, 2009). Metacognitive strategies comprise attempts to control the activity in the cognitive system to obtain cognitive and emotional self-regulation (e.g., trying to control one’s stream of consciousness to avoid frightening thoughts) (Wells, 2009).

According to the metacognitive S-REF model of psychological disorders, psychopathology arises from maladaptation and rigidity in the different aspects of metacognition (Wells & Matthews, 1994). This maladaptation consists of (1) beliefs about the usefulness, controllability and harmfulness of certain cognitive processes, (2) processing plans that allocate disproportionate attention to potential threats and (3) unpleasant metacognitive experiences based on negative appraisal of cognitive activity (Wells, 2009). The strategies to deal with cognitions typically involve arduous endeavours to suppress certain thoughts, efforts to find answers to the current state through thorough analysis, and attempts at avoiding future discomforting experiences by preparing for all possible outcomes through worrying. These maladaptive metacognitive coping strategies do not achieve the goal of cognitive and emotional self-regulation, but instead maintain the sense of threat that they were devised to resolve (Wells, 2009).

### **2.4.1 Metacognition and the Cognitive Attentional Syndrome (CAS)**

Maladaptive beliefs about the advantages of engaging in certain cognitive processes (i.e., positive metacognitive beliefs), and beliefs about the uncontrollability and potential danger of such processes (i.e., negative metacognitive beliefs), give rise to a maladaptive cognitive style named the Cognitive Attentional Syndrome (CAS; Wells, 2009). The CAS is characterized by repetitive thinking in the form of worry and rumination, an attentional bias towards threat-related stimuli, and efforts to control thoughts and emotions through thought-suppression and cognitive, emotional and behavioural avoidance (Wells, 2009). The CAS is theorized to be responsible for maintaining and intensifying distressing emotions in all

psychiatric disorders (Hjemdal, Hagen, Nordahl, & Wells, 2013). Worrying can maintain a sense of threat and consequent anxiety, and the perception that the world is a dangerous place that one is not equipped to cope with. In addition, worry disturbs self-regulation as it interferes with emotional processing (Stokes & Hirsch, 2010; Wells & Papageorgiou, 1995). Ruminating on questions without clear answers maintains a state of uncertainty and a discrepancy between what the person knows, and what the person thinks he should know (Wells, 2009). An example is a person with obsessive-compulsive disorder asking himself “Why do I have these thoughts that scare me?”. Worrying and rumination also claim a lot of attentional resources, leaving less resources for decision-making and task-relevant processing (Wells, 2009). Engaging in threat-monitoring also takes a toll on attentional resources and lowers the person’s threshold for detecting potential threats. This leads to an increase in subjectively experienced danger, and it reinforces beliefs regarding the need to focus on dangers in order to stay safe, thus forming a vicious cycle (Wells, 2009). Attempts at controlling thoughts and emotions through suppression and avoidance deprives the person of the opportunity to discover that they are able to cope and that their feelings and thoughts are, in fact, not dangerous.

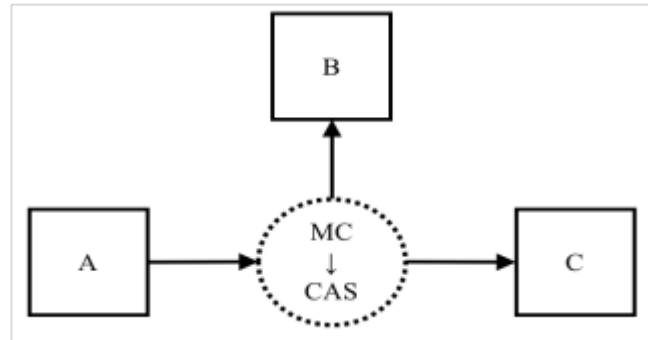
#### **2.4.2 Metacognition, Cognitive Beliefs and Psychopathology**

There are two forms of metacognitive theory with different implications for the focus of therapy. In the “soft” version, metacognitions are thought to underlie the processes of pathology along with core beliefs about oneself and the world, closely resembling EMSs (Wells, 2009). Therefore, therapy could be aimed at changing both metacognitive and cognitive beliefs. In the “hard” form, metacognitive beliefs are considered the sole base of psychopathology (see [Figure 2](#)). Schema-related beliefs are seen as the conclusions people draw about themselves and the world based on CAS-activity (Wells, 2009). As such, schemas do not cause symptoms, but are products of the same processes that produce symptoms. Therefore, schema-beliefs’ impact on symptoms is expected to be of little importance compared to metacognitions’ impact. On that premise, therapy should be aimed at modifying metacognitions. The focus of metacognitive therapy in the current study is in line with the “hard” form.

Research has shown support for the claim that metacognitions, as measured by the Metacognitions Questionnaire (MCQ), are related to psychopathology in the form of anxiety

disorders, depression, psychotic disorders and alcohol abuse (Wells, 2009). Metacognitions about the danger and uncontrollability of cognition has been found to be especially important contributors to anxiety symptoms (Bailey & Wells, 2013, 2016; Cho, Jahng, & Chai, 2012; Gkika, 2011; Spada, Mohiyeddini, & Wells, 2008; Yilmaz, Gencoz, & Wells, 2008) and pathological worry (Martin et al., 2014; McEvoy, Erceg-Hurn, Anderson, Campbell, & Nathan, 2015; Yilmaz et al., 2008), which is a central feature in many anxiety disorders (Wells, 2009).

Figure 2. “Hard” Metacognitive Theory.



Note. Based on Wells, 2009, p. 256-257. A = Antecedent (internal trigger), MC = Maladaptive Metacognitive Beliefs; CAS = Cognitive Attentional Syndrome, B = Beliefs; C = Emotional Consequences.

There is some support for a relationship between changes in metacognitions and changes in measures related to anxiety disorders. Studies in samples with obsessive-compulsive disorder (OCD) have found that changes in metacognitions are significantly related to changes in OCD-symptoms (Grøtte et al., 2015; Solem, Haland, Vogel, Hansen, & Wells, 2009). The relationship is significant even when controlling for initial symptom level and changes in depression (Solem et al., 2009), and cognitive belief-change (Grøtte et al., 2015). In a review of the effect of MCT for depression and anxiety disorders, MCT was found to produce large changes in both maladaptive metacognitive beliefs, and symptoms of anxiety and depression (Normann et al., 2014). Changes in primary outcome measures were significantly larger for patients undergoing MCT than patients in CBT or in wait-list control groups (Normann et al., 2014). Normann and colleagues’ review also showed that changes in metacognitions are maintained over time, alongside with maintenance of symptom relief. A recent study found support for the stability of metacognitive beliefs, and the role of negative metacognitive beliefs as a stable vulnerability factor for depression (Solem et al., 2017). All this taken together builds a strong case for metacognitions as fundamental cognitive units that should be targeted in order to achieve lasting symptom-relief.

The information presented thus far in this chapter about EMSs and schema theory, and metacognitions and metacognitive theory, form the foundation for the hypotheses and research questions that are posed in the next section.

## 2.5 Research Questions and Hypotheses

The primary aim of this study is to examine the role of EMSs in anxiety disorders. That entails an investigation of whether EMSs are related to anxiety symptoms and whether changes in EMSs effect changes in anxiety symptoms. Another concern regarding the role of EMSs, is whether they are to be considered central, as in schema theory, or peripheral as in the “hard” metacognitive theory. Informed by aspects of schema theory and metacognitive theory presented in this chapter, the following hypotheses and exploratory questions are posed:

Hypothesis 1: There will be a significant decrease in EMS-endorsement across pre-treatment, post-treatment and follow up.

Hypothesis 2: Schema-endorsement will account for a significant amount of the variance in anxiety symptoms. There will be significant within- and between-person relationships between schemas and symptoms. That is:

- a) Higher average level of EMS-endorsement across pre-treatment, post-treatment and follow-up will be related to higher symptom-level (between-person relationship).
- b) When EMSs for a given patient is higher than the patients average, the concurrent symptoms will also be higher (within-person relationship).

Hypothesis 3: Larger decreases in schema-endorsement during treatment will be related to lower levels of and larger decreases in anxiety symptoms in the follow-up period.

Exploratory 1: Are EMSs still significantly related to anxiety symptoms, both concurrently and prospectively, when metacognitions are added to the analyses? Based on the S-REF model, it is hypothesized that inclusion of metacognitions will lead to less significant relationships between EMSs and symptoms.

Exploratory 2: According to metacognitive theory, the MCT-condition should lead to greater decline in maladaptive metacognitive beliefs than the CBT-condition, resulting in greater decline in both schema-beliefs and anxiety symptoms. Are there significant interactions between treatment condition and decline in metacognitions, symptoms and EMSs?

## 3 Method

### 3.1 Ethics

The study from which the data in this thesis emanate (Johnson, Hoffart, Nordahl, & Wampold, 2016) has been approved by the Norwegian Regional Committees for Medical and Health Research and Ethics. All participants provided written informed consent as to the use of their scores on Internet-administered questionnaires and diagnostic interviews performed by research assistants.

### 3.2 Participants

The participants in the study took part in a randomized controlled trial (RCT) comparing the effect of CBT vs. MCT in the treatment of complex anxiety disorders (Johnson et al., 2016). The participants were recruited from the Department of anxiety disorders at Modum Bad in Norway. The department has an 8-week inpatient treatment program for people with treatment-resistant anxiety disorders. All patients at the department have failed to benefit from previous treatment. To be included in the study, the participants had to speak Norwegian, be 18 years of age or older and meet the DSM-IV criteria (American Psychiatric Association, 2000) for one or more of the following disorders at pre-treatment: SAD, PTSD or PD/A. Diagnostic assessment was performed by advanced clinical psychology students using the Anxiety Disorder Interview Schedule for DSM-IV (ADIS-IV; DiNardo, Brown, & Barlow, 1994), a reliable and valid instrument for Axis I disorders (Antony & Rowa, 2005). The assessors were blind to treatment conditions. Exclusion criteria were concurrent DSM-IV diagnosis of organic mental disorders or other comorbid conditions that interfered with treatment, current substance abuse, present suicidal risk and non-compliance to the requirement to bring their use of psychotropic medication to cessation prior to treatment.

A total of 90 participants were randomized to treatment, 45 to each condition. Six of these lost eligibility from evaluation to pre-treatment, six did not show up to treatment and four were excluded because their therapists had not completed the specific CBT or MCT training. The intention-to-treat (ITT) sample consisted of 74 patients (CBT:  $n = 38$ ; MCT:  $n = 36$ ). See [Table 2](#) for characteristics of the ITT sample. Seven patients (9.5%) dropped out prematurely due to lack of motivation (CBT:  $n = 4$ ; MCT:  $n = 1$ ) and use of alcohol (CBT:  $n = 1$ ; MCT:  $n = 1$ ). 67 participants completed the entire treatment program (CBT:  $n = 33$ ; MCT:  $n = 34$ ). 11 additional participants were lost to follow up due to withdrawal from trial



(CBT:  $n = 1$ ; MCT:  $n = 1$ ) and unknown reasons (CBT:  $n = 5$ ; MCT:  $n = 4$ ). The analyses in this thesis is based on the ITT-sample.

Table 2. *Characteristics of the ITT sample at pre-treatment.*

Characteristic	CBT ( $n = 38$ )	MCT ( $n = 36$ )	Total ( $N = 74$ )
Age in years, $M$ ( $SD$ )	40.2 (12.3)	44.7 (13.7)	42.3 (13.0)
Female, <i>number</i> (%)	25 (65.8)	20 (55.6)	45 (60.8)
Occupational status past 6 months, <i>number</i> (%) <sup>a</sup>			
Disabled	8 (21.1)	10 (27.8)	18 (24.3)
Unemployed	17 (44.7)	13 (36.1)	30 (40.5)
Sick leave	7 (18.4)	4 (11.1)	11 (14.8)
Partly employed	2 (5.3)	5 (13.9)	7 (9.5)
Employed	2 (5.3)	2 (5.6)	4 (5.4)
Other <sup>b</sup>	1 (2.6)	2 (5.6)	3 (4.1)
Education, <i>number</i> (%)			
Upper secondary school	15 (39.5)	14 (38.9)	29 (39.2)
University college	7 (18.4)	12 (33.3)	19 (25.7)
University	8 (21.1)	1 (2.8)	9 (12.2)
Other	5 (13.2)	9 (25.0)	14 (18.9)
Primary diagnosis, <i>number</i> (%)			
PD/A	12 (31.6)	16 (44.4)	28 (37.8)
SAD	12 (31.6)	10 (27.8)	22 (29.7)
PTSD	14 (36.8)	10 (27.8)	24 (32.4)
Former treatment, <i>number</i> (%) <sup>c</sup>			
Outpatient psychiatric treatment	32 (84.2)	31 (86.1)	63 (85.1)
Inpatient psychiatric treatment	18 (47.3)	12 (33.3)	30 (40.5)
Duration of illness, $M$ ( $SD$ )	14.1 (11.1)	17.6 (12.2)	16.1 (11.8)
Number of diagnoses at start of treatment, $M$ ( $SD$ )	3.76 (1.60)	3.67 (1.58)	3.72 (1.58)

*Note.* <sup>a</sup> Occupational status missing for one person. <sup>b</sup> Other = Funded by spouse/ living on old funds.

<sup>c</sup> Numerous patients had both inpatient and outpatient former treatment.

### **3.3 Procedure**

After diagnostics were done at the evaluation stay, participants were stratified on primary diagnosis (PD/A, PTSD, SAD) prior to randomisation. The participants were randomised to one of two treatment conditions: trans-diagnostic MCT or diagnosis-specific CBT, based on random computer-generated numbers ([www.random.org](http://www.random.org)). All questionnaires were computer-administered and scored automatically through Check Ware (<http://checkware.com>). The questionnaires were completed at evaluation, pre-treatment, post-treatment and follow-up.

### **3.4 Treatment and Therapists**

Participants had weekly 1-hr individual therapy sessions of CBT and MCT based on detailed treatment manuals. In the CBT condition, treatment focused on the primary diagnosis by utilizing diagnosis-specific manuals for PD/A (Clark, 1986), PTSD (Foa et al., 2007) and SAD (Clark & Wells, 1995). In the MCT condition, a generic MCT-manual developed by Wells (2009) was followed.

The therapists in the study were three clinical psychologists, a psychiatrist and a junior registrar who were all employed as therapists at the Department of anxiety disorders at Modum Bad at the time of the RCT. The therapists were trained in CBT or MCT, and subsequently supervised by Asle Hoffart and Hans Nordahl who are highly experienced in CBT and MCT, respectively.

### **3.5 Measures**

#### **3.5.1 Beck Anxiety Inventory (BAI)**

Anxiety symptoms were measured using the Norwegian version of Beck Anxiety Inventory (BAI), a 21 item self-report inventory designed to measure the severity of anxiety in psychiatric populations (Beck, Epstein, Brown, & Steer, 1988). Each item is scored on a four-point Likert-scale from 0 = “Not at all” to 3 = “Severely” according to how much each symptom has bothered the person the past week (Beck et al., 1988). This yields an item score of 0-3 and a total score of 0-63. The scoring is criterion-referenced with a recommended clinical cut-off of 16 points (Bardhoshi, Duncan, & Erford, 2016). A recent meta-analysis shows that BAI has good internal consistency in clinical and non-clinical samples ( $\alpha=.91$ ,  $k = 117$ ,  $n = 43\ 932$ ). Considering that BAI is a measure of state-anxiety, it showed satisfactory test-retest reliability in a clinical population over a median of six weeks ( $r_{tt} = .66$ ,  $n = 699$ )

(Bardhoshi et al., 2016). BAI also shows strong convergent validity across 33 different measures of anxiety (Bardhoshi et al., 2016). The Norwegian version has been found to possess good psychometric properties, similar to the original version (Nordhagen, 2001). In the current study, BAI showed good internal consistency at intake ( $\alpha = .89$ ), and satisfactory relative test-retest stability from evaluation to intake ( $r_{tt} = .62$ ,  $n = 63$   $M = 110$  days,  $SD = 51$  days)<sup>5</sup>.

### 3.5.2 Metacognitions Questionnaire–30 (MCQ-30)

Metacognitions were measured by the 30-item version of the Metacognitions Questionnaire (MCQ-30; Wells & Cartwright-Hatton, 2004). MCQ-30 gives a measure of metacognitive beliefs, judgements and monitoring propensities considered important within the metacognitive understanding of psychopathology (Wells & Cartwright-Hatton, 2004). The MCQ-30 contains five subscales with six items each; (1) Positive beliefs about worry, (2) Negative beliefs about worry (regarding its danger and uncontrollability), (3) Low cognitive confidence, (4) Need to control thoughts, and (5) Cognitive self-consciousness (Wells & Cartwright-Hatton, 2004). Each item each scored on a 4-point Likert-scale ranging from 1 = “Do not agree” to 4 = “Agree very much”. This gives a subscale score of 6-24, and a total score of 30-120. There is no set cut-score for the MCQ-30.

The factor structure of the MCQ-30 has been cross-culturally replicated in translated forms in samples in Korea (Cho et al., 2012), the UK (Spada et al., 2008), Turkey (Yilmaz et al., 2008), Spain (Martin et al., 2014) and Norway (Grøtte et al., 2015). Test-retest reliability estimates for the total scale are satisfactory at  $r_{tt} = .75$  over an interval of 22-118 days (Wells & Cartwright-Hatton, 2004) and  $r_{tt} = .80$  for 5-7 weeks (Spada et al., 2008) in non-clinical samples. The scale has demonstrated good internal consistency in both clinical and non-clinical samples (e.g., Grøtte et al., 2015; Martin et al., 2014; Solem et al., 2009; Yilmaz et al., 2008). In previous studies with the Norwegian version, translated by Hansen, Vogel and Nordahl, Cronbach’s alphas have ranged between .71-.87 for the subscales (Grøtte et al., 2015; Solem et al., 2017; Solem et al., 2009). In the current study, alphas were high, ranging between .83-.89 for the subscales and .92 for the total scale at intake. MCQ-30 has shown good convergent validity with established measures of worry, trait-anxiety, obsessive-

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<sup>5</sup> An MLM with BAI as dependent variable and context (evaluation, intake), time lapsed (number of days from evaluation to intake) and context\*time lapsed as covariates revealed no significant main effect of time lapsed or any moderating effect of time lapsed on the relationship between context and BAI-scores. Therefore, all participants scores could be analysed together, despite a large range in time lapsed.

compulsive symptomatology and depression (Cho et al., 2012; Grøtten et al., 2015; Martin et al., 2014; Solem et al., 2009; Spada et al., 2008; Yilmaz et al., 2008).

### **3.5.3 Young Schema Questionnaire – Short Form (YSQ-S1)**

The Young Schema Questionnaire (YSQ) is a self-report inventory developed by Young and Brown in 1989 (cited in Young, 1990) to assess EMSs subordinated under schema domains. The items in the questionnaire were produced by Young and other therapists based on clinical experience with complex psychotherapy patients (Schmidt et al., 1995). Each item is rated on a 6-point Likert-scale ranging from 1 = “Completely untrue of me” to 6 = “Describes me perfectly”. Commonly, mean scores are used when scoring the individual EMSs, schema domains and the total score (Rijkeboer, 2012; Sheffield & Waller, 2012). There are no definite cut-scores on the YSQ, but Rijkeboer (2012) suggests that individual EMS-scores of over 2,5 are in the clinical range, with the exception of Defectiveness for which 2 serves as a cut score, and the schemas Self-Sacrifice and Unrelenting Standards that are rarely in the clinical range until they surpass a mean value of around 3. Scores in Norwegian samples (see [Table 1](#)) have generally been found to be somewhat lower than these international cut-scores.

Numerous revisions of the YSQ have been published, varying in both item- and EMS-count (Hawke & Provencher, 2012). In this study, the Norwegian 75-item YSQ short-form (YSQ-S1; Young, 1998) was utilized. The item pool is comprised of the five items with the highest loadings on each of the 15 EMSs (see [Table 1](#)) found in in the first psychometric evaluation of the YSQ (Schmidt et al., 1995). The Norwegian version has been back translated to English and the items were found to not differ significantly in meaning (Hoffart et al., 2005). The YSQ-S1 has shown good to excellent internal consistency for both total scores and individual EMS scores across cultures and translations in samples in Australia (Baranoff et al., 2006), Britain (Waller, Meyer, & Ohanian, 2001), Canada (Welburn et al., 2002), China (Cui et al., 2011), France (Lachenal-Chevallet, Mauchand, Cottraux, Bouvard, & Martin, 2006), Norway (Hoffart et al., 2005; Thimm, 2013), Spain (Calvete et al., 2005) and South-Korea (Baranoff et al., 2006). In this study, Cronbach’s alpha at the beginning of treatment was .97 for the total scale and .82-.94 ( $M = .88$ ) for the individual schemas. The test-retest reliability of the YSQ-S1 has been found to be moderate to high. In a non-clinical sample ( $N = 351$ ), relative stability of the 15 EMSs were found to be .48-.71 ( $M = .62$ ) over a six-month interval (Calvete, Orue, & González-Diez, 2013). Test-retest correlations for EMSs in clinical samples are generally moderate to high:  $r_{tt} = .63-.87$  over an average time

period of 72 days (Hoffart et al., 2005), median  $r_{tt} = .75$  over 2.5-5 years (Riso et al., 2006) and mean  $r_{tt} = .54$  over a 9-year interval (C. E. Wang et al., 2010). In this study, test-retest correlations were computed from evaluation to pre-treatment ( $n = 63$ ). The mean time-interval was 110 days ( $SD = 50$  days)<sup>6</sup>. Test-retest correlations were high for total score ( $r_{tt} = .81$ ) and moderate to high for individual EMSs ( $r_{tt} = .68-.80$ ,  $M = .76$ ).

Several psychometric studies have explored the factor structure of the YSQ-S1 (e.g., Baranoff et al., 2006; Calvete et al., 2005; Cui et al., 2011; Hoffart et al., 2005; Lachenal-Chevallet et al., 2006; Welburn et al., 2002). These studies have yielded differing results as to the number of EMSs and schema domains. In this study, the analyses are based on the total score of the YSQ-S1. This is in part due to lack of consistency in the factor structure in previous factor analytical studies, and also due to an expectation of a multitude of relevant EMSs as the current sample is highly comorbid.

The YSQ-S1 has been found to possess similar validity and other psychometric properties to the long-form (Waller et al., 2001). The schemas and domains measured in the YSQ-S1 have been found converge with symptoms of a number of disorders, and maladaptive personality traits (see [Section 2.3](#)). YSQ-scores have also shown discriminant validity, with significantly higher scores in clinical than non-clinical samples (Hawke & Provencher, 2012; Rijkeboer & van den Bergh, 2006; Waller et al., 2001; C. E. Wang et al., 2010) and significantly higher scores in more complex and chronic cases such as personality disorders (Hawke & Provencher, 2013; Lee, Taylor, & Dunn, 1999; Nilsson, Jørgensen, Straarup, & Licht, 2010).

### 3.6 Statistical Procedures

The current analyses are based on the scores on BAI, MCQ-30 and YSQ-S1 measured at pre-treatment, post-treatment and the 1-year follow-up<sup>7</sup>. This allows for investigation of changes in the variables over time and the relation between changes in the different variables. The time variable was coded to reflect the number of months between assessments (Pre-treatment = 0, Post-treatment = 2, 1-year follow-up = 14). Doing so gives a meaningful intercept (i.e., the score of a person at pre-treatment) and a more correct presentation of time than would be

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<sup>6</sup> Same analysis as in footnote 5, only with YSQ-score as dependent variable. There was no significant main effect of time lapsed or any moderating effect of time lapsed on the relationship between context and YSQ-scores. Therefore, all participants scores could be analysed together, despite a large range in time lapsed.

<sup>7</sup> Evaluation was not included, due to a significant moderating effect of time lapsed on the change in MCQ-30 from evaluation to intake, and lack of relevance to the hypotheses relating to therapy-relevant change.

achieved by simply coding it 0, 1 and 2. This lead to a skewed time-variable with regards to the numerical distance between the measurement points. MLMs consider time as linear unless otherwise specified. In this study, there is reason to believe that change in the dependent variables are not linear across time. There is reason to believe that there will be a steep decrease during treatment, followed by a less steep decrease over the subsequent time points (the follow-up period). This was confirmed by visual inspection of scatter plots of patients' scores on the dependent variables across time. Therefore, the time variable was transformed logarithmically ( $0 = 0$ ;  $2 = 0.4771$ ;  $14 = 1.1761$ ). This significantly improved the linearity of trajectories and the model fit, indicated by decreases in the -2 Log Likelihood fit statistics at equal degrees of freedom for BAI, MCQ, and YSQ across time.

### **3.6.1 The Multilevel Modelling Framework**

All analyses were performed in IBM Statistical Package for Social Sciences (SPSS) version 24. The dataset was organized vertically in SPSS to allow for multilevel modelling (MLM; Heck, Thomas, & Tabata, 2010). MLM is a well suited framework for modelling hierarchical data (Tasca & Gallop, 2009). In the current study, there is a 2-level hierarchical data structure with repeated measurements (Level 1) nested within individuals (Level 2). As there are repeated measures, the data likely violate the assumption of uncorrelated residuals, leading to possible inflation of Type 1 errors (Tasca & Gallop, 2009). By utilizing MLM, the dependence of the residuals over time can be modelled, thus avoiding this fallacy. Being able to model changes in residual variance across time points is beneficial because the error variances in psychotherapy research are often reduced after treatment as a result of decline in scores for previously high-scorers (Tasca & Gallop, 2009).

Another advantage of MLM is that one can utilize all the participants' measurements, regardless of their completer-status, so long as the missing data are missing at random (Gallop & Tasca, 2009). Traditional analytical methods based on estimation of group means and group variances offer less robust methods for dealing with missing data. Within the traditional frameworks, one has to either impute missing values or remove all scores of participants with incomplete score-sets, leading to substantial loss of statistical power (Tasca & Gallop, 2009). This is a major disadvantage as psychotherapy research is often riddled with drop-outs (Field, 2013). Using MLM will render more power to the statistical analyses.

Furthermore, in MLM the intercepts and slopes can be allowed to vary across individuals (random coefficients), in addition to estimating the average intercepts and slopes

(fixed coefficients). Modelling a random intercept can uncover whether there are substantial differences between individuals in level of the dependent variable when predictors equal zero. Modelling a random slope can uncover individual differences in the growth of the dependent variable. By modelling the covariance between the random intercept and random slope, one can assess whether the individuals' starting level has any systematic impact on their subsequent growth.

The multilevel models were developed through building from an empty model, to more complex models, as recommended by Tasca and Gallop (2009). Increasing model complexity entails building from an empty model with no predictor, to adding a number of fixed and random effects, and modelling residual variance structures on the within- and between-person levels. Equations for model-building are given in Appendix A. For each of the outcome variables, it was investigated whether a random effect for slopes and intercepts, and an interaction between random slopes and intercepts improved model fit.

Model fit for the growth was evaluated by checking whether a first- or second order polynomial best fit the data (Field, 2013). Having found the best fitting model, the error structures of the variances were examined. On Level 1, the residuals were examined to determine whether they were best modelled as homo- or heteroscedastic. On Level 2, it was examined what covariance structure for the random effects' variances gave the best fit. Maximum likelihood was used as the estimation method, and Chi-square tests of change in the -2 Log Likelihood (-2LL) fit index, calculated in SPSS, were used to assess model fit (Field, 2013). To assess degree of predicted variance by the models, a Pseudo-R<sup>2</sup> based on the change in Level 1 and Level 2 residuals from the base model to the more complex models was calculated (Calculation in Appendix B). Due to the risk of Type 1 error in performing several statistical tests, the significance was set to a more stringent level of  $p < .01$ .

### **3.6.2 Disaggregation of Within- and Between-Person Effects**

In order to best model the impact of individuals' change in the predictor variables on the outcome variable, the predictor variables were disaggregated into between- and within-person effects. It is important to disaggregate these effects, as the aggregate effect is "generally an uninterpretable blend" (L. Wang & Maxwell, 2015, p. 67). This because the between and within-person effects can be different in magnitude and even direction. An easily understandable example is cited in Curran and Bauer (2011): Although size of is predictive of longevity between mammal species, the relationship does not apply within species. An

average human being lives longer than an average rat, but a 500-pound person does not, on average, live longer than a 150-pound person. Aggregating the within and between subject effects in such a case would obscure the actual relationship between size and longevity. In the current study, person mean centring was utilized to disaggregate the within- and between-person effects, as is recommended by Wang and Maxwell (2015). As the person mean only varies between individuals it is a between-person (BP) effect. The person mean centred score varies within persons, making it a within-person (WP) effect. The BP variable is an expression of the individual's average level of the predictor variable across time. The WP-variable indicates whether the individual scores above or below its own average at a given time. The BP- and WP-scores were computed by equations 1 and 2, respectively. No detrending (control for the effect of time) was performed on any of the variables because it is counter-indicated when the study design involves interventions to change both predictor and outcome variables (L. Wang & Maxwell, 2015), as is the case in the current study.

$$(1) \quad BP_{X_i} = \frac{\sum_t X_{it}}{T_i}$$

$$(2) \quad WP_{X_{it}} = x_{it} - BP_{X_i}$$

### 3.6.3 Statistical Analyses

Relevant equations and syntaxes for hypothesis-testing are found in Appendix B and C, respectively. Decline in EMSs across time (Hypothesis 1), was examined by regressing YSQ-scores on Time in an MLM. The within- and between-person relationship between EMSs and concurrent symptoms (Hypothesis 2), was examined by entering BP-YSQ and WP-YSQ as covariates and BAI-score as the dependent variable. To estimate the contribution of YSQ-scores to the explanation of variance in BAI-scores, the increase in pseudo- $R^2$  ( $\sim R^2$ ; see appendix B for calculation) from adding the YSQ-effects was calculated.

In order to test Hypothesis 3, that changes in EMSs during treatment would relate to level and change of symptoms from post-treatment to follow-up, a variable representing change in YSQ-scores was calculated (YSQ-change). The change-score was calculated by subtracting YSQ-score at pre-treatment from YSQ-score at post-treatment (YSQ-change =  $YSQ_2 - YSQ_0$ )<sup>8</sup>. As such, a positive score indicates that YSQ-scores increased from pre- to

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<sup>8</sup> If there were more time-points, the same disaggregation procedure as described above would have been utilized. However, with only two time-points, the WP-variable is not well-suited to establish temporal sequence, as the effect of WP-predictor on BAI would likely be negative at one point and positive at the other due to the concurrent correlation between the measurements. The change-score is not a within-person variable, as it does not vary within persons.



post-treatment, while a negative change-score indicates a decline in scores from pre- to post-treatment. The YSQ-change, Time and the interaction of YSQ-change\*Time, were added as covariates in the analysis, and BAI-score at post-treatment and follow-up was the dependent variable. This allowed for investigation of the effect of change in YSQ during treatment on the level of BAI (main effect of YSQ-change) and change in BAI from post-treatment to follow-up (interaction of YSQ-change\*Time).

The exploration of the effect of metacognitions on symptoms, and the consequences of adding metacognitions in analyses of EMSs' relationship to symptoms (Exploratory 1) was conducted by the following procedure: First, separate models wherein the BP and WP-effects of YSQ and MCQ on concurrent BAI-scores were compared. Then, all four covariates were entered into the same model to investigate their relative unique relations to BAI-scores. Next, the separate prospective models were compared (i.e., Time, YSQ-change, Time\*YSQ-change vs. Time, MCQ-change, Time\*MCQ-change). Then, the change-scores' relative contribution to the prediction of BAI-scores was examined by entering Time, YSQ-change, MCQ-change, Time\*YSQ-change and Time\*MCQ-change as covariates in the same model.

To explore whether MCT led to greater decrease in MCQ than CBT, resulting in greater decrease in both YSQ and BAI (Exploratory 2), separate analyses for each measure were conducted. First, the main effects of Treatment Condition (TC) and Time were added as covariates to examine whether there was change across time, and whether TC was associated with differences in level of the dependent variable. In the subsequent interaction-analyses, Time and the interaction of Time\*TC were entered as covariates, as the aim is to analyse differences in *change* between the conditions. The analyses were carried out during treatment alone, and then across all three time-points. This was done to investigate whether potential differences in changes between the groups were evident at post-treatment, and whether they upheld across the follow-up period.

Finally, to explore the possibility of reversed causation, YSQ and MCQ were entered as dependent variables, and BP-BAI, WP-BAI and BAI-change were entered as covariates. These analyses were conducted concurrently and prospectively, like the above analyses.

# 4 Results

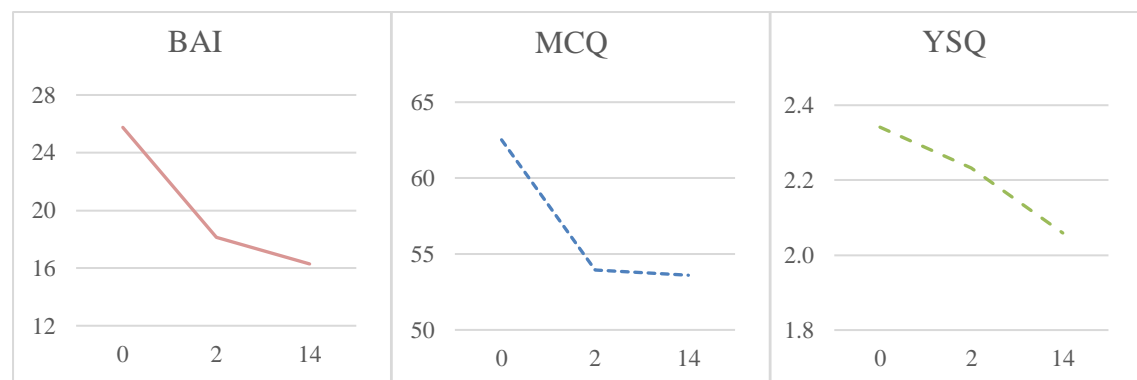
## 4.1 Descriptive Statistics

Table 3. *Total scores across time*

	Pre-treatment M (SD)	Post-treatment M (SD)	1-year follow-up M (SD)
BAI	25.73 (10.84)	18.09 (11.48)	16.32 (10.92)
MCQ	62.49 (16.17)	53.83 (16.86)	53.54 (16.83)
YSQ	2.34 (0.73)	2.23 (0.80)	2.06 (0.68)
N	74 <sup>a</sup>	67 <sup>b</sup>	56 <sup>c</sup>

*Note.* BAI=Beck Anxiety Inventory, MCQ=Metacognitions Questionnaire-30, YSQ= Young Schema Questionnaire-S1. <sup>a</sup> 1 missing YSQ-score. <sup>b</sup>1 missing YSQ and MCQ score. <sup>c</sup>1 missing YSQ-score.

Figure 3. *Total scores across time*



*Note.* 0 = Pre-treatment; 2 = Post-treatment; 14 = 1-year follow-up

As seen in Table 3 and Figure 3, there are substantial declines in BAI and MCQ from pre- to post-treatment (7.64 and 8.66, respectively), followed by small declines from post-treatment to follow-up (1.77 and 0.29, respectively). The decline of YSQ is more linear. Nevertheless, the decline from pre- to post-treatment (0.11) is smaller than from post-treatment to follow-up (0.17). The mean BAI-scores are still slightly above the clinical cut-score of 16 at follow-up (16.32). An average MCQ-score of 53.54 at follow-up indicates that the sample still endorse maladaptive metacognitive beliefs to some extent. The average YSQ-score at follow-up of 2.06 implies that EMS-endorsement is mainly below the internationally recommended cut-scores (Rijkeboer, 2012). However, it is worth noting that this sample mainly scored below the international cut-scores of YSQ at pre-treatment as well.

## 4.2 Preliminary Analyses

First, the impact of completer-status<sup>9</sup> was investigated. Completer-status did not significantly impact pre-treatment BAI scores ( $p = .60$ ) or YSQ scores ( $p = .45$ )<sup>10</sup>. There was an almost a significant effect of completer status on MCQ scores ( $\gamma = 5.93$ ,  $SE = 2.82$ ,  $t(74) = 2.1$ ,  $p = .04$ ), indicating that initial MCQ scores tended to be higher among completers. There were no significant differences in the change in scores on BAI, MCQ or YSQ from pre-treatment to post-treatment between those who were lost to follow-up and those who completed the follow-up (all  $p > .52$ ). In addition, completer status did not moderate the relationship between the within- or between-persons' effects of YSQ or MCQ on BAI. Therefore, the statistical prerequisite of data missing at random are considered to be met.

For each dependent variable in the analyses (meaning all of the variables due to reversal of predictor and outcome), the combination of random effects and covariance structure of the residuals that gave the best fit was chosen. None of the variables modelled in the current analyses showed increased model fit when allowing for heteroscedastic residuals on Level 1. This means that, contrary to what can be expected in psychotherapy research (Tasca & Gallop, 2009), the variance of the Level 1 residuals was fairly equal across time-points. For all analyses, including a random intercept improved model fit, whereas the additional inclusion of a random slope or random effect of a time-varying covariate (TVC), did not. The significant increase in model fit when including a random intercept, indicates significant between-person variance in outcome when predictors equal zero.

For YSQ across three time-points, time was best modelled by a first-order polynomial growth curve. BAI scores and MCQ scores across three time points were significantly better modelled by a second order polynomial according to a chi square test of model fit when including a squared time-term, Time-Squared (BAI:  $\chi^2(1) = 7.04$ ,  $p = .008$ , MCQ:  $\chi^2(1) = 16.95$ ,  $p < .001$ ). For the prospective analyses, time was modelled by a first-order polynomial growth curve for all variables because there were only two time-points (Field, 2013).

There were three outliers ( $\pm 3$  SD) in the data. All analyses were re-ran without the outliers to investigate their impact on the analyses. There were no substantial differences in the magnitude or significance of results when removing the outliers, therefore analyses were completed with all scores. The residuals of the analyses were fairly normally distributed, indicated by skewness and kurtosis-statistics and visual inspection of their distribution.

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<sup>9</sup> (0 = only pre-treatment; 1 = pre- and post-treatment; 2 = pre, post and follow-up)

<sup>10</sup> Investigated by a multilevel model with completer status as predictor and BAI and YSQ at pre-treatment as dependent variables.

## 4.3 Hypotheses

### 4.3.1 Hypothesis 1

Mixed model analyses including Time as a covariate showed that YSQ declined significantly over time ( $\gamma = -0.20$ ,  $SE = 0.06$ ,  $t(125.43) = -3.36$ ,  $p = .001$ ).

### 4.3.2 Hypothesis 2

The YSQ-scores accounted for a substantial amount of variance in BAI scores (Residual  $\sim R^2 = .34$ ; Intercept  $\sim R^2 = .17$ ). Both the time- and individual-specific WP-YSQ ( $\gamma = 15.77$ ,  $p < .001$ ) and the individual-specific BP-YSQ ( $\gamma = 6.13$ ,  $p < .001$ ) were significantly related to BAI-scores (top part of Table 5).

### 4.3.3 Hypothesis 3

YSQ-change (top part of Table 6) during treatment was related to level of BAI-scores at post-treatment and follow-up ( $\gamma = 8.83$ ,  $p = .02$ ). The main effect of time ( $p = .16$ ) and the interaction effect between YSQ-change and Time ( $p = .45$ ) was not statistically significant. Thus, change in YSQ during treatment was not related to change in BAI scores from post-treatment to follow-up.

### 4.3.4 Exploratory 1

Table 5. Comparison of the effect of MCQ and YSQ on concurrent BAI-scores

-2LL (df)	Fixed effects	Estimate	SE	df	t	Sig.
1425.38 (5)	BP-YSQ	6.13	1.27	71.77	4.81	.000
	WP-YSQ	15.77	1.94	122.14	8.14	.000
1437.25 (5)	BP-MCQ	0.31	0.06	67.89	5.45	.000
	WP-MCQ	0.56	0.07	121.78	7.92	.000
1400.79 (7)	BP-YSQ	2.68	1.85	72.00	1.45	.151
	WP-YSQ	10.11	2.20	119.88	4.60	.000
	BP-MCQ	0.21	0.09	69.53	2.47	.016
	WP-MCQ	0.36	0.08	120.08	4.50	.000

In the separate YSQ-analysis, there was a significant concurrent effect of BP-YSQ ( $\gamma = 6.13$ ,  $p < .001$ ) and WP-YSQ ( $\gamma = 15.77$ ,  $p < .001$ ) on BAI. In the separate MCQ-analysis, there was a significant concurrent effect of BP-MCQ ( $\gamma = 0.31$ ,  $p < .001$ ) and WP-MCQ ( $\gamma = 0.56$ ,

$p < .001$ ) on BAI-scores. The YSQ-model showed better fit than the MCQ-model, indicated by a lower -2LL number at equal degrees of freedom ( $\chi^2(0) = 11.87$ ). When all four covariates were entered into the same model, there was a significant fixed effect WP-MCQ ( $\gamma = 0.36, p < .001$ ) and WP-YSQ ( $\gamma = 10.11, p < .001$ ), and a close to significant effect of BP-MCQ ( $\gamma = 0.21, p = .016$ ). The fixed effect of BP-YSQ was no longer significant ( $p = .15$ ), indicating that the inclusion of MCQ-effects impacted the magnitude of a YSQ-effect. The model fit for the combined model is significantly better than for either separate model (MCQ:  $\chi^2(2) = 36.46, p < .001$ ; YSQ:  $\chi^2(2) = 24.59, p < .001$ ), indicating unique contributions of both MCQ and YSQ to BAI-scores.

Table 6. Comparison of the effect of YSQ and MCQ on prospective BAI-scores

-2LL (df)	Fixed effects	Estimate	SE	df	t	Sig.
911.77 (6)	Time	-3.19	2.23	63.29	-1.43	.157
	YSQ-change	8.83	3.74	101.18	2.36	.020
	Time*YSQ-change	-2.98	3.93	62.07	-0.76	.451
914.55 (6)	Time	-3.63	2.83	65.85	-1.29	.203
	MCQ-change	0.30	0.15	102.00	1.98	.050
	Time*MCQ-change	-0.10	0.16	64.93	-0.60	.550
910.53 (8)	Time	-3.55	2.91	67.37	-1.22	.227
	MCQ-change	0.15	0.18	100.79	0.81	.420
	Time*MCQ-change	-0.05	0.20	65.92	-0.27	.792
	YSQ-change	6.61	4.60	100.69	1.44	.153
	Time*YSQ-change	-2.15	4.86	62.58	-0.44	.659

In the separate prospective models (Table 7), Change-scores, Time and Time\* Change-scores were covariates, and the dependent variable was BAI-score at post-treatment and follow up. The effect of YSQ-change on level of BAI-scores was nearly significant ( $p = .02$ ), while the other effects were not significant. In the separate prospective MCQ-model, the effect of MCQ-change on BAI-scores was likewise approaching significance ( $p = .05$ ), while the other effects were not. Again, the YSQ-model shows a somewhat better model fit than the MCQ-model ( $\chi^2(0) = 2.78$ ). In the comparison-model, there were no significant effects. The effect that is closest to achieving significance is the YSQ-change effect ( $p = .15$ ). The model fit for the combined model was not significantly better than either of the separate models (YSQ:  $\chi^2(2) = 1.24, p = .54$ ; MCQ:  $\chi^2(2) = 4.04, p = .13$ ).

### 4.3.5 Exploratory 2

The initial analyses consisted of investigating the main effects of Treatment Condition (TC) and Time-variables on the dependent variables. In the initial analyses of main effects of Time and TC on the dependent variables at pre- and post-treatment, the main effect of TC was insignificant (all  $p > .38$ ) for all dependent variables. This means that there were no significant differences in level of the dependent variables between the groups at pre-treatment (as can be seen in Figure 4). The main effect of Time was significant on MCQ ( $\gamma = -20.23, p < .001$ ) and BAI ( $\gamma = -15.64, p < .001$ ), and approached significance for YSQ ( $\gamma = -0.26, p = .07$ ). The same pattern was found in the analyses of main effects across pre-treatment, post-treatment and follow-up. The main effect of TC was insignificant for all variables (all  $p > .39$ ). The main effects of the time variables were significant for MCQ (Time:  $\gamma = -28.85, p < .001$ ; Time-squared:  $\gamma = 17.61, p < .001$ ), BAI (Time:  $\gamma = -20.85, p < .001$ ; Time-squared:  $\gamma = 10.91, p = .008$ ) and YSQ (Time:  $\gamma = -0.20, p = .001$ ). The aim of the following analyses is to investigate differences in change-rate between treatment conditions. As the main effect of TC was insignificant in all initial analyses, it is excluded from the subsequent analyses in order to retain a more parsimonious model.

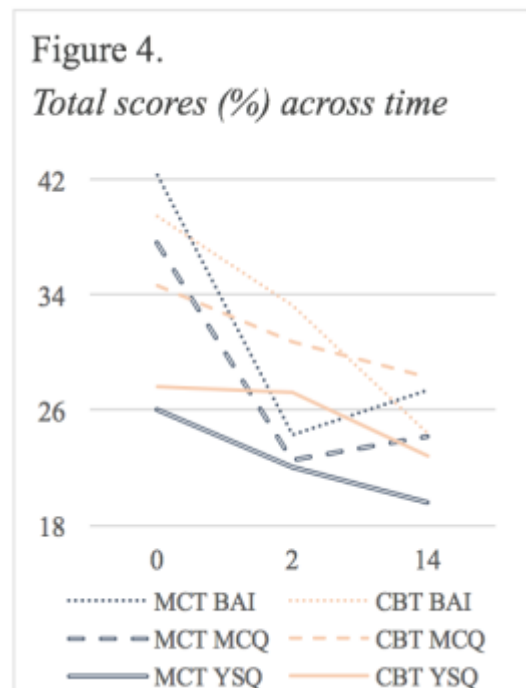


Table 7. Treatment condition (TC) impact on change in total scores during treatment

Dependent	Fixed effects	Estimate	SE	df	t	Sig.
MCQ	Time	-11.81	4.70	78.67	-2.51	.014
	Time*TC	-16.56	6.28	92.42	-2.64	.010
BAI	Time	-8.97	4.12	92.56	-2.18	.032
	Time*TC	-13.16	5.24	124.64	-2.51	.013
YSQ	Time	-0.08	0.19	76.89	-0.44	.664
	Time*TC	-0.34	0.26	86.72	-1.31	.193

In the analyses including pre- and post-treatment measurements (Table 7), the interaction effect of Time and TC is significant on MCQ ( $\gamma = -16.56, p = .01$ ) and BAI-scores ( $\gamma = -13.16, p = .01$ ). These interaction effects indicate that the decrease over time is larger for MCT (Condition 1) than CBT (Condition 0). For YSQ-scores, the direction of the relationships is the same as for BAI and MCQ, but the effect is not statistically significant ( $\gamma = -0.34, p = .19$ ).

Table 8. *Treatment condition (TC) impact on change in total scores across all time-points*

Dependent	Fixed effects	Estimate	SE	df	t	Sig.
MCQ	Time	-15.96	6.89	136.80	-2.32	.022
	Time-sq	8.42	5.65	130.39	1.49	.138
	Time*TC	-25.21	9.34	149.85	-2.70	.008
	Time-sq*TC	17.97	7.75	137.01	2.32	.022
BAI	Time	-9.27	6.52	152.95	-1.42	.157
	Time-sq	0.67	5.46	141.72	0.12	.902
	Time*TC	-22.76	8.50	179.19	-2.68	.008
	Time-sq*TC	20.07	7.37	154.52	2.72	.007
YSQ	Time	-0.17	0.08	138.50	-2.07	.040
	Time*TC	-0.06	0.11	152.90	-0.50	.617

In the analyses of changes across treatment and follow-up (Table 8), the interaction effect of Time and TC on MCQ was significant ( $\gamma = -25.21, p = .008$ ). The interaction effect of Time-Squared and TC approaches significance ( $\gamma = 19.33, p = .02$ ). This indicates that patients receiving MCT decrease more than those receiving CBT initially, but that this effect is then reversed as the decline in MCQ slows down more substantially for MCT than for CBT. For the BAI-scores, there are significant fixed interaction effects of Time and TC ( $\gamma = -22.76, p = .008$ ) and Time-Squared and TC ( $\gamma = 20.07, p = .007$ ). Again, this indicates larger initial decrease in the MCT-condition, which is reversed by less decrease in MCT relative to CBT. For the YSQ-scores, treatment condition does not affect the decline over pre-treatment, post-treatment and follow-up.

#### 4.3.6 Examination of Reversed Causation

When BP- and WP-BAI were entered as covariates and concurrent YSQ-score was the dependent variable, there was a significant effect of BP-BAI ( $\gamma = 0.04, p < .001$ ) and WP-

BAI ( $\gamma = 0.02, p < .001$ ), explaining a substantial proportion of the variance in YSQ-scores (Residual  $\sim R^2 = .35$ ; Intercept  $\sim R^2 = .21$ ). In the analysis with concurrent MCQ-scores as dependent variable, there were significant effects of BP-BAI ( $\gamma = 0.84, p < .001$ ) and WP-BAI ( $\gamma = 0.61, p < .001$ ), explaining a substantial proportion of the variance in MCQ-scores (Residual  $\sim R^2 = .33$ ; Intercept  $\sim R^2 = .25$ ).

In the prospective analyses, wherein Time, BAI-change and Time\*BAI-change were entered as covariates, the effect of BAI-change was significant in the prediction of YSQ-score-level ( $\gamma = 0.03, p = .002$ ) and MCQ-score level ( $\gamma = 0.49, p = .007$ ) from post-treatment to follow-up. The effect of Time was insignificant on the MCQ, but approached significance for the YSQ ( $\gamma = -0.25, p = .022$ ). The interaction of Time and BAI-change showed trends towards larger declines over time for those with larger BAI-change for both MCQ ( $\gamma = -0.19, p = .13$ ) and YSQ ( $\gamma = -0.01, p = .08$ ). The three covariates accounted for 8.6% of the residual variance on Level 1, and 11.7 % of the variance in intercepts between persons on the YSQ, and 4.6% and 7.7% of the respective variances in the MCQ.



# 5 Discussion

The primary aim of this thesis is to examine the role of EMSs in anxiety disorders. In sum, the results largely imply that EMSs are related to symptoms in complex anxiety patients. There were significant between- and within person relationships between YSQ and concurrent BAI-scores. The prospective analysis partially supported the link between EMSs and symptoms, as there was a trend towards YSQ-changes during treatment affecting subsequent level of BAI-scores. The exploratory analyses of the relative unique contributions of YSQ and MCQ to BAI-scores were not equivocal as to the importance of EMSs relative to the importance of metacognitions. The hypothesis that the MCT-condition would lead to greater decline in metacognitions, and consequently greater decline in symptoms and schema-beliefs was supported from pre- to post-treatment. However, the effect did not uphold across all three time-points.

## 5.1.1 Hypothesis 1 – Decline in EMSs over Time

The decline in YSQ-scores over time was statistically significant, as was expected due to the potentially schema-changing elements in the current protocols (see [Section 2.3.3](#)). The current numerically small, statistically significant declines are in line with previous findings of modest significant declines during non-schema-therapy such as dynamic group therapy (Wegener et al., 2013), CBT (Borge et al., 2008; Halford et al., 2002) and a mixed group-treatment program (Welburn et al., 2000). Given the assumptions in schema theory regarding the rigidity of EMSs and that they require longer-term therapy to change (Young et al., 2003), it is unsurprising that the reduction in YSQ-scores is small following merely 8 weeks of treatment. The pattern of a slight increase in the steepness of decline from post-treatment to follow-up in this study was also found in the only other study of changes in EMSs in treatment of anxiety disorders with a long-term follow-up (Borge et al., 2008). A possible explanation of this effect is that the controlled treatments cause small initial changes in EMSs that in turn allows for less biased information-processing and more adaptive coping, which in turn can dampen schema-endorsement after being repeated over a longer period of time. It is also possible that continued improvement after treatment comes from patients' continuing therapy in the follow-up period. As there was no control of whether or what kind of therapy the patients received after treatment-completion in the current or previous study (Borge et al., 2008), one cannot know which mechanism more likely underlies the change-pattern.

### **5.1.2 Hypothesis 2 – EMSs and Concurrent Symptoms**

The significant positive between-person effect of YSQ on the level of symptoms indicates higher level of symptoms at higher mean levels of YSQ. The significant positive within-person relationship between YSQ and symptoms signifies that scoring above the person-mean at a given time was associated with higher BAI-scores at that time. This offers support to the contention that EMS-endorsement is related to symptomatic distress, and that within-person change in EMSs relates to symptom-relief. These findings are in accordance with previous research on the relationship between EMS-endorsement and symptoms in anxiety disorders (Cockram et al., 2010; Dutra et al., 2008; Glaser et al., 2002; Halford et al., 2002; Hawke & Provencher, 2013; Hedley et al., 2001; Hinrichsen et al., 2007; Hinrichsen et al., 2004; Hoffart et al., 2005; Hovland, 2007; Kwak & Lee, 2015; McGinn et al., 2005; Pinto-Gouveia et al., 2006; Price, 2007; Welburn et al., 2002; Welburn et al., 2000). The current study holds two important advantages over many of the previous studies. Firstly, the relationship between symptoms and EMSs are measured over time. Secondly, the current study is based on disaggregated scores, which none of the previous longitudinal studies were (Cockram et al., 2010; Halford et al., 2002; Hedley et al., 2001; Welburn et al., 2000).

Finding that differences between persons in predictor change-scores on average are related to between-person differences in outcome, does not necessarily mean that the relationship is the same on the within-person level. Therefore, the findings do not automatically imply that a person will achieve symptom-relief by increasing level of change in the predictor variable. Assuming such a relationship could be a case of the ecological fallacy of misattributing the between-person effects to the within person level (Curran & Bauer, 2011). By disaggregating scores through person-mean centring in this study, such misattribution is avoided and the results speak to what is of most interest to therapists: how change within the person in the predictor relates to that person's outcome. The within-person effect is easily translatable to information that is useful in psychotherapy (Hoffart, 2017). The current study shows that within-person changes in EMSs are related to the person's anxiety symptoms. The finding of this within-person relationship represents an extension of the previous knowledge base on the relationship between EMSs and symptoms.

### **5.1.3 Hypothesis 3 – Prospective Effects of EMS-Changes on Symptoms**

There was a trend ( $p = .02$ ) of the effect of change in EMSs from pre- to post-treatment on the level of symptoms from post-treatment to follow-up. The positive relationship between change in EMSs and subsequent symptom-level indicates that between persons, decline in

EMSs during treatment is related to lower level of symptoms from post-treatment to follow-up. None of the previous studies on the longitudinal relationship between EMSs and anxiety have investigated the prospective effect of changes in EMSs on subsequent symptom-level (Cockram et al., 2010; Halford et al., 2002; Hedley et al., 2001; Welburn et al., 2000). As such, the current finding extends the corpus of knowledge on the relationship between EMSs and anxiety symptoms. There was no significant effect of time or of the interaction of time and EMS-change during treatment on subsequent symptoms. This implies that there were no significant changes in symptoms from post-treatment to follow-up. The lack of change in symptoms strongly limits the possibility of finding a significant interaction of EMS-change and time. The direction of the insignificant effects indicates that there was a small decline in symptoms over time that was exacerbated at higher levels of YSQ-change. The direction of these effects are in line with what could be expected from schema theory. However, if EMSs are core sources of pathology and its recurrence, the reduction of EMSs should lead to maintenance of or continued decline in symptoms in the follow-up period. The current study did not show the expected prospective effects of change in EMSs on subsequent change in symptoms.

#### **5.1.4 Exploratory 1 – Comparison of Effect of EMSs and Metacognitions**

The separate analyses showed that there were significant within- and between-person relationships between symptoms and both YSQ and MCQ. In the combined model, the between-person effect of the YSQ on symptoms was no longer significant, while the between-person effect of MCQ was nearly significant ( $p = .016$ ). This supports the predictions based on metacognitive theory, as the inclusion of metacognitions led a YSQ-effect to contribute less uniquely to prediction of symptoms. However, the within-person scores' effect on concurrent symptoms remained significant for both EMSs and metacognitions in the comparison model. This signifies that a reduction relative to the persons' mean on both YSQ and MCQ is significantly related to lower concurrent symptoms.

In a schema-theoretical framework, this can be understood by looking at the influence of EMSs and coping styles to symptomatic distress (see [Figure 1](#)). According to schema theory, symptoms and problems are caused by the EMSs themselves, and by the maladaptive coping responses to schema-threat. A coping response to the Vulnerability-schema, is to surrender to excessive worry (Young et al., 2003, p. 150). The Vulnerability-schema is one of the schemas most consistently associated to symptoms in anxiety disorders (Hawke &

Provencher, 2011). Therefore, it can be assumed that a reduction in the symptom-inducing coping strategy would lead to symptom-relief. The MCQ can be understood to affect symptoms through its effect on the coping response of excessive worry. That is, lower levels of and decline in metacognitive beliefs can lead to decline in worry (a maladaptive coping strategy), which in turn is associated with lower levels of and decline in symptoms. As such, the unique contributions of MCQ and YSQ to symptoms make sense from a schema-theoretical standpoint. From the standpoint of “hard” MCT, the continued unique contribution of the within-person effect of YSQ in the comparison model is not as easily incorporated. In “hard” MCT, it is theorized that metacognition is more substantially related to symptomatic distress than are schema-beliefs. As such, the within-person effect of metacognitions should overshadow the within-person effect EMSs. This was not the case in the current study, as the magnitude of the within-person effects of EMSs and metacognitions decreased similarly when entered as covariates in the same model<sup>11</sup>. The current analysis, being the first comparison of between- and within-person effects of YSQ and MCQ on symptoms, indicates that symptom-relief can be achieved through within-person changes in both metacognition and EMSs.

In the prospective models, the YSQ- and MCQ-change variables’ impact on the level of BAI-scores showed a nearly significant trend in the separate models ( $p = .02$  and  $p = .05$ , respectively). The effects of time and the interaction between time and change-variables were insignificant in the separate analyses. This signifies that BAI-scores did not change over time from post-treatment to follow-up, and that the cognitive variables did not moderate changes in BAI. In the combined analysis, there were no significant effects, indicating that neither of the change-variables are uniquely related to level of BAI or to subsequent change in BAI-scores. Previous studies on the relationship between anxiety symptoms and changes in MCQ (e.g., Grøtte et al., 2015; Solem et al., 2009) and EMSs (Cockram et al., 2010; Halford et al., 2002) have investigated the effect of changes concurrently. That is, the effect of change in the predictor from time A to time B on the outcome at time B, or change in outcome from time A to time B, was investigated. These studies found significant relationships between changes in the predictors and corresponding symptom-scores. Previous research has largely found that declines in both metacognitions and EMSs are maintained or further amplified across follow-up periods (Borge et al., 2008; Cockram et al., 2010; van der Heiden, Muris, & van der

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<sup>11</sup> The decline in t-value from the separate to the comparison model was from  $t = 8.14$  to  $t = 4.60$  for WP-YSQ, and from  $t = 7.92$  to  $t = 4.50$  for WP-MCQ

Molen, 2012; Wells et al., 2012; Wells et al., 2010). Therefore, the effect of changes in the predictors were expected to be maintained across the follow-up period. However, this was not the case as the current results indicate that changes in metacognitions and EMSs during treatment are not significantly related to subsequent level of or change in symptoms.

### **5.1.5 Exploratory 2 – Treatment Condition Effects**

During treatment, treatment condition significantly impacted the effect of time on MCQ-scores and BAI-scores. The negative interaction effect between treatment condition and time signified that the MCT group decreased more per unit of time than the CBT-group (for further discussion of these treatment effects, see Johnson et al., 2016). The interaction effect of condition and time on YSQ-scores was not significant ( $p = .19$ ). However, the direction of the insignificant interaction was the same, indicating that decrease over time was larger in MCT than CBT. Thus, MCT caused larger declines than CBT during treatment in both metacognitive beliefs and anxiety symptoms, and showed a trend towards causing larger reductions in EMSs. This finding can be understood as support for the metacognitive conceptualisation depicted in [Figure 2](#). As YSQ decreases more in the condition with more decline in MCQ, it is plausible that schema-beliefs are by-products of metacognitively driven CAS-activity.

When the follow-up measure was included in the analysis, the advantage of MCT over CBT was no longer apparent (see [Figure 4](#)). The relationship between MCQ- and YSQ-decline was also more ambiguous as the patients in the MCT-condition continue to decline on YSQ, while they slightly increased on MCQ and BAI. This finding runs contrary both to the posited relationship between metacognitions and EMSs, and to the proposed relationship between EMSs and symptoms. The schema-theoretical framework can accommodate the finding of no EMS-change despite significant symptom-improvements (Rusinek et al., 2004) by turning to the notion of “dormant schemas” (Young et al., 2003). It is more challenging to incorporate the opposite finding of EMS-change without improvement in symptoms ([Figure 4](#)). However, in the CBT-condition, the expected relationship between EMSs and symptoms appear to hold true, as larger declines in symptoms coincided with larger declines in EMSs. The seeming differences in the relationship between the cognitive variables and symptoms in the two conditions can possibly be attributed to the patients’ implicit theories (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003). Through the treatments, the MCT-patients learn that metacognitions are central to symptoms, while the CBT-patients learn that the content of

their thoughts are related to symptom-relief. Perhaps the patients' implicit theories led the improved patients to score themselves as improved on the cognitive variables they have been taught are most strongly connected to symptom-improvement? As there is no control of implicit theories or any measure to capture the belief in the relationship between cognitive variables and symptoms, this remains an open question.

## **5.2 Clinical Implications**

The finding that within-person change in YSQ was strongly and significantly related to symptoms, supports the potential usefulness of clinical interventions that affect endorsement of EMSs. Thus, a clinician who manages to modify EMSs could expect reductions in anxiety at the end of treatment. The current study does not indicate what type of treatment interventions are most efficient at effecting EMS-change, but rather demonstrates that a range of different interventions from different theoretical frameworks can be used to achieve schema-change.

Finding that symptoms are also strongly related to within-person changes in metacognitive beliefs indicates that this too is a potentially beneficial focus for treatment. The comparative analysis shows that within-person changes in both metacognitions and EMSs make unique contributions to variance in symptoms. This points to potential additive beneficial effects of inducing change in both forms of cognition.

## **5.3 Limitations**

There are numerous limitations to consider when interpreting the results of this study. First, there are limits to the generalizability due to factors related to sample characteristics, time-frame of measurements, treatment-characteristics and limited number measurements per participant. As the sample consists of highly comorbid and chronic anxiety patients, the findings may not be applicable to less complex cases of anxiety. This concern is especially relevant with regards to the main focus of this thesis, as EMSs are assumed to be more central in more complex cases. However, as associations between EMSs and anxiety symptoms have been found in healthy samples as well (e.g., Calvete, Orue, & Hankin, 2013; Gonzalez-Diez, Calvete, Riskind, & Orue, 2015; Orue, Calvete, & Padilla, 2014; Saritaş & Gençöz, 2011), this might not present a serious issue. The time-frame of measurement (2-12 months between assessments) challenges the generalization of within-person relationships to recommendations for therapy from week to week. The effects may not be directly translatable

across these different time-frames. The generalizability of the effects of the treatments on EMSs and metacognitions are limited by the fact that the treatment occurred within a unique context of intensive inpatient treatment. It is therefore unclear whether the results generalize to outpatient-treatment. The utility and generalizability of the findings are also limited by there being only three observations per person. With only three time-points, the establishment of causal relationships between variables is troublesome. This especially limits the analyses based on change-scores. With only three time-points, there is only one period of change to predict one other period of change. That equates to drawing conclusions about a relationship based on only two observations. Doing so strongly limits generalizability and precludes conclusions regarding the shape of the relationship (linear, quadratic).

Secondly, the use of self-report poses considerable limitations. Self-report questionnaires are subject to a number of possible biases, including false reports in order to self-present as better or worse than what is the case (Podsakoff et al., 2003). Moreover, as all variables were measured by questionnaires completed at the same place and time and through the same medium (computer-scoring), the risk of inflated relationships due to shared method variance is substantial (Podsakoff et al., 2003). The relationships between the variables may be heavily influenced by method-variance, thus overstating the actual relationship between the variables. Another challenge relating to the use of questionnaires is especially salient with regards to the YSQ. While EMSs are thought to be unconscious structures (Young et al., 2003), self-report measures are based on information that is consciously available to the individual. Although Welburn and colleagues (2002) argue that the person is likely to have some awareness of his or her schemas, as they significantly impact the person's life, it is reasonable to assume that some aspects of EMSs are not sufficiently measured by self-report questionnaires.

A third area of limitations pertains to the overlap of concepts and unclear causal direction between the variables in the current study. As discussed in Section 2.3.1, *EMSs and Anxiety*, there are several similarities between EMSs and diagnostic features of anxiety disorders. Thus, it is possible that the EMS-endorsement at the beginning of treatment does not reflect the presence of maladaptive schemas developed in childhood and adolescence, but rather reflects pathoplastic effects (Widiger & Smith, 2010). That is, elevated EMSs may reflect current symptoms and the subsequent decline may thus be a reflection of symptom-reduction and not schema-healing. In the study of Wang and colleagues (2010), currently depressed individuals scored higher than previously depressed individuals on 14/15 EMSs as measured by YSQ. This supports the pathoplastic theory. However, they also found that

YSQ-scores were higher in previously depressed than never-depressed individuals, indicating that EMSs measured by YSQ, are not fully reducible to pathoplastic effects of current pathology. Another potentially confounding overlap in the current study is that of the two cognitive variables. There is a potential problem of multicollinearity as YSQ and MCQ are highly correlated. The correlation was high for both the between-person ( $r = .76$ ), and for the within-person ( $r = .57$ ) effect. However, when tested for multicollinearity, the tolerance values for all predictors were satisfactory at above .43. Thus, multicollinearity may not pose a significant limitation. The significant effects of symptoms on cognitive variables when examining reversed causation severely challenges the interpretation of causality in the current study. Finding that BAI-scores explained similar amounts of variance in MCQ and YSQ as the reversed, indicates that inferences regarding causality should at best be viewed as tentative. The results of the prospective analyses pose especially severe challenges to the idea that changes in cognitions precede and cause changes in symptoms, as BAI-change was a better predictor for MCQ and YSQ from post-treatment to follow-up than the reversed.

A fourth limitation relates to the lack of control over therapy in the time between post-treatment and follow-up. This can impact the interpretations regarding the differences in effects between MCT and CBT. At follow-up, 67.8% of the sample (73% CBT and 62% MCT) reported that they had received treatment in the follow-up period (Johnson et al., 2016). Of those receiving additional treatment, 33% received CBT, while the majority (49%) of participants could not tell what kind of treatment they were given (Johnson et al., 2016). Additional treatment after discharge can be expected to have a differential impact on continued improvement depending on whether it concurs with the treatment given while admitted. The effects of MCT could be reversed due to therapy that counteracts the way of dealing with thoughts encouraged by MCT. Wells (2009, p. 257) claims that certain interventions in for example cognitive therapy encourages the patient to engage in the CAS-processes of worry and rumination. Such therapy could possibly weaken the improvement gained through the eight weeks of MCT. Patients in the CBT-group were more likely to receive treatment after discharge that concurred with the treatment they received while admitted. Therefore, it is possible that the larger treatment gains from post-treatment to follow-up were a result of continued concurring therapy, rather than representing a delayed effect of the 8-week inpatient CBT treatment. As there is no control of subsequent therapy, the generalizability of treatment condition-effects are limited.



## 5.4 Conclusion

The current study is part of a larger psychotherapy research project aimed at increasing the knowledge of MCT and CBT for complex anxiety disorders, in a naturalistic clinical setting. Overall, the results in this study indicate that EMSs are related to anxiety symptoms in complex anxiety disorders, thus supporting the main hypothesis. Concurrently, there are significant between- and within person relationships between schemas and anxiety symptoms. The hypothesized decrease in EMSs over time was confirmed, supporting previous findings of EMS-decline in non-schema therapy. The hypothesis regarding the prospective effect of schema-change on subsequent symptoms was only partially supported. It appears that degree of schema-change affects the level of, but not change in, subsequent symptoms. However, the lack of effect of schema-change on symptom-change can be a consequence of limited change in symptoms from post-treatment to follow-up.

The hypothesis based on “hard” metacognitive theory of larger declines in EMSs in MCT was not consistently supported. Larger declines were found in the analyses during treatment, but not across all three time-points. The comparative analysis of the relationship between EMSs and symptoms, and metacognition and symptom showed that both EMSs and metacognitions are substantially related to psychopathology. Neither form of cognition can be said to be unequivocally supported as contenders for core cognition in the current study. It rather seems that both types of cognition are potentially beneficial focuses for change in psychotherapy for complex anxiety disorders. Thus, effecting within-person changes in EMSs and metacognitions is likely to effect changes in anxiety symptoms.

## 5.5 Future Directions

Future research should attempt to circumvent the methodological limitations of this study. It is considered important that future studies include more measurement-occasions as more within-person observations would allow for using time-lagged analyses with disaggregated variables. This gives the opportunity to investigate how within-person changes in the predictor relate to subsequent outcome-scores over time. Several observations of the within-person relationship will reduce the risk of randomly occurring lagged relationships. Furthermore, it is central that future studies make efforts to reduce the potential influence of shared method variance and the other limitations caused by relying exclusively on information from self-report questionnaires. Data regarding the predictor and outcome variables should be gathered by different methods and at different times in order to reduce the

potential confounding effect of shared-method variance. For example, EMSs may be better measured by tests within the Stroop-paradigm or other tests that can give better indications of unconscious automatic processes.

Future research would also benefit from closer control over therapy in the follow-up period. Methodologically, it would be best if the patients did not receive therapy from other therapists in the follow-up period. However, it would be highly unethical to deny people treatment in the name of finding clearer long-term treatment-effects. Therefore, one should rather attempt to ensure some continuity in treatment focus in subsequent therapy by engaging in collaboration with the patients' therapists. When the patients return for the 1-year follow-up, more detailed enquiries as to the content of the therapy in the follow-up period should be made. Doing so could help answer the question of whether the reduction in treatment gains for the MCT-group can be caused by contradictory subsequent therapy, as is proposed by Wells.

Future research and theorizing should also elaborate on whether and under what conditions changes in metacognitions and EMSs are necessary and sufficient to cause lasting symptom-relief. The current results show the need for further specification, as symptoms were found to increase while EMSs decreased in the MCT-condition, and symptoms decreased significantly while there was little decline in metacognitions in the CBT-condition. These observations do not concur well with schema theory and metacognitive theory, respectively. A possible point of investigation is the role of the patients' degree of belief in the models proposed in treatment, as this can impact what variables the patient associates with symptom-relief.

Based on the trend towards larger change of EMSs in MCT, future research should look more closely into what processes cause schema-change in MCT. Does MCT affect EMSs through schema-healing processes defined by Young and colleagues (2003)? Or is schema-change a by-product of change in metacognition as proposed in hard metacognitive theory? The inclusion of process-measures of both types of change would be useful in further investigation of the different conceptualisations of the role of schemas.

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# Appendix A – Multilevel Model Equations

## Base model – comparison model

The score of person  $i$  at time  $t$  is a function of the person's time-specific deviation ( $r_{it}$ ) from the grand mean, that is the average of all persons in the sample across all time points ( $\beta_0$ ).

This is expressed in the Level 1 equation.

Level 1 equation:

$$y_{it} = \beta_0 + r_{it}$$

## Model with random intercept

The score of person  $i$  at time  $t$  is a function of the person's time-specific deviation ( $r_{it}$ ) from the person's mean over all time-points ( $\beta_{0i}$ ). This is expressed in the Level 1 equation. Each person's mean is a function of the grand mean, and the person-specific mean deviation from the grand mean. This is expressed in the Level 2 equation.

Level 1 equation:

$$y_{it} = \beta_{0i} + r_{it}$$

Level 2 equation:

$$\beta_{0i} = \gamma_{00} + u_{0i}$$

## Model with random slope

The score of person  $i$  at time  $t$  is a function of the person's score when the predictor equals zero ( $\beta_{0i}$ ), the person's growth over time ( $\beta_{1i}$ ) and the person's time-specific deviation from the score predicted by the intercept and slope parameters ( $r_{it}$ ). This is expressed in the Level 1 equation. Each person's mean is a function of the grand mean, and the person-specific mean deviation from the grand mean. And each person's slope is a function of the average slope and a person-specific deviation from this slope. This is expressed in the Level 2 equations.

Level 1 equation:

$$y_{it} = \beta_{0i} + \beta_{1i} \text{Time}_{it} + r_{it}$$

Level 2 equation:

$$\beta_{0i} = \gamma_{00} + u_{0i}$$

$$\beta_{1i} = \gamma_{10} + u_{1i}$$

Reduced form equation:

$$y_{it} = \gamma_{00} + \gamma_{10} \text{Time}_{it} + u_{0i} + u_{1i} \text{Time}_{it} + r_{it}$$

# Appendix B – Equations for Hypotheses

## Hypothesis 1 – Unconditional linear model with random intercept

Level 1 equation:

$$YSQ_{it} = \beta_{0i} + \beta_{10} \text{Time}_{it} + r_{it}$$

Level 2 equations:

$$\beta_{0i} = \gamma_{00} + u_{0i}$$

$$\beta_{10} = \gamma_{10}$$

Reduced form equation

$$YSQ_{it} = \gamma_{00} + \gamma_{10} \text{Time}_{it} + u_{0i} + r_{it}$$

## Hypothesis 2 – Conditional intercept and time-varying covariate

BP-YSQ = YSQ total person mean (between-person effect), WP-YSQ= YSQ total person mean centred score (within-person effect).

Level 1 equation

$$BAI_{it} = \beta_{0i} + \beta_{10} \text{WP-YSQ}_{it} + r_{it}$$

Level 2 equations

$$\beta_{0i} = \gamma_{00} + \gamma_{01} \text{BP-YSQ}_i + u_{0i}$$

$$\beta_{10} = \gamma_{10}$$

Reduced form equation

$$BAI_{it} = \gamma_{00} + \gamma_{01} \text{BP-YSQ}_i + \gamma_{10} \text{WP-YSQ}_{it} + u_{0i} + r_{it}$$

### *Pseudo-R<sup>2</sup>-calculation*

Level 1 Residual Variance ( $\sigma^2$ )  $\sim R^2 = (\sigma^2_{\text{base}} - \sigma^2_{\text{current}}) / \sigma^2_{\text{base}}$

Level 2 Intercept Variance ( $\tau_0$ )  $\sim R^2 = (\tau_{0\text{base}} - \tau_{0\text{current}}) / \tau_{0\text{base}}$

## Hypothesis 3 – Conditional linear model

Level 1 equation

$$BAI_{it} = \beta_{0i} + \beta_{10} \text{Time}_{it} + r_{it}$$

Level 2 equations

$$\beta_{0i} = \gamma_{00} + \gamma_{01} \text{YSQchange}_i + u_{0i}$$

$$\beta_{10} = \gamma_{10} + \gamma_{11} \text{YSQchange}_i$$



Reduced form equation

$$BAI_{it} = \gamma_{00} + \gamma_{01} YSQchange_i + \gamma_{10} Time_{it} + \gamma_{11} YSQchange_i Time_{it} + u_{0i} + r_{it}$$

### **Exploratory 1 – Comparison of conceptualisations**

Models in the exploratory analyses were based on the equations from Hypothesis 3 and 4, with BP- and WP-MCQ and BP- and WP-YSQ as covariates, respectively. The comparison – model was calculated based on the following reduced form equations:

Comparison of concurrent between- and within person effects of MCQ and YSQ:

$$BAI_{it} = \gamma_{00} + \gamma_{01} BP-YSQ_i + \gamma_{02} BP-MCQ_i + \gamma_{10} WP-YSQ_{it} + \gamma_{20} WP-MCQ_{it} + u_{0i} + r_{it}$$

Comparison of prospective change-effects of MCQ and YSQ:

$$BAI_{it} = \gamma_{00} + \gamma_{01} YSQ-change_i + \gamma_{02} MCQ-change_i + \gamma_{10} Time_{it} + \gamma_{11} YSQ-change_i Time_{it} + \gamma_{12} MCQ-change_i Time_{it} + u_{0i} + r_{it}$$

### **Exploratory 2 – Treatment condition effects**

First, main effects of time and treatment condition (TC) on each dependent variable (MCQ, BAI and YSQ) were investigated first during treatment, then across treatment and follow-up for each variable by the following equation:

$$y_{it} = \gamma_{00} + \gamma_{01} TC_i + \gamma_{10} Time_{it} + u_{0i} + r_{it}$$

Then, six separate analyses were made, two for each dependent variable. One analysis of change during treatment, and one of change across all three time-points.

$$y_{it} = \gamma_{00} + \gamma_{01} TC_i + \gamma_{10} Time_{it} + \gamma_{11} TC_i Time_{it} + u_{0i} + r_{it}$$

# Appendix C – Syntax Commands

## Hypothesis 1

```
MIXED YSQ_score WITH Time_log
  /CRITERIA=CIN(95) MXITER(1000) MXSTEP(100) SCORING(1) SINGULAR(0.000000000001)
  HCONVERGE(0, ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
  /FIXED=Time_log | SSTYPE(3)
  /METHOD=ML
  /PRINT=G SOLUTION TESTCOV
  /RANDOM=INTERCEPT | SUBJECT(ID) COVTYPE(VC).
  /SAVE=RESID.
```

## Hypothesis 2

```
MIXED BAI_score WITH BP_YSQ WP_YSQ
  /CRITERIA=CIN(95) MXITER(1000) MXSTEP(100) SCORING(1) SINGULAR(0.000000000001)
  HCONVERGE(0, ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
  /FIXED=BP_YSQ WP_YSQ | SSTYPE(3)
  /METHOD=ML
  /PRINT=G SOLUTION TESTCOV
  /RANDOM=INTERCEPT | SUBJECT(ID) COVTYPE(VC).
  /SAVE=RESID.
```

## Hypothesis 3

```
1COMPUTE filter_$=(Time=2 | Time=14).
VARIABLE LABELS filter_$ 'Time=2 | Time=14 (FILTER)'.
VALUE LABELS filter_$ 0 'Not Selected' 1 'Selected'.
FORMATS filter_$ (f1.0).
FILTER BY filter_$.
EXECUTE.
```

```
MIXED BAI_score WITH Time_log YSQ_change_02
  /CRITERIA=CIN(95) MXITER(1000) MXSTEP(100) SCORING(1) SINGULAR(0.000000000001)
  HCONVERGE(0, ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
  /FIXED= Time_log YSQ_change_02 Time_log*YSQ_change_02 | SSTYPE(3)
  /METHOD=ML
  /PRINT=SOLUTION TESTCOV
  /RANDOM=INTERCEPT | SUBJECT(ID) COVTYPE(VC)
  /SAVE=RESID.
```

## Exploratory 1 – Comparison of conceptualisations

Same syntax commands as for Hypothesis 3 and 4 with YSQ-effects and MCQ-effects separately. Syntax for comparison models:

---

<sup>1</sup> From now on denoted by “Data selected” followed by relevant time-points.

**Concurrent** (Data selected: All cases)

```
MIXED BAI_score WITH BP_MCQ WP_MCQ BP_YSQ WP_YSQ
/CRITERIA=CIN(95) MXITER(1000) MXSTEP(100) SCORING(1) SINGULAR(0.000000000001)
HCONVERGE(0, ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
/FIXED=BP_MCQ WP_MCQ BP_YSQ WP_YSQ | SSTYPE(3)
/METHOD=ML
/PRINT=G SOLUTION TESTCOV
/RANDOM=INTERCEPT | SUBJECT(ID) COVTYPE(VC).
/SAVE=RESID.
```

**Prospective** (Data selected: Time=2 | Time=14).

```
MIXED BAI_score WITH Time_log YSQ_change_02 MCQ_change_02
/CRITERIA=CIN(95) MXITER(1000) MXSTEP(100) SCORING(1) SINGULAR(0.000000000001)
HCONVERGE(0, ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
/FIXED= Time_log YSQ_change_02 Time_log*YSQ_change_02 MCQ_change_02
Time_log*MCQ_change_02 | SSTYPE(3)
/METHOD=ML
/PRINT=SOLUTION TESTCOV
/RANDOM=INTERCEPT | SUBJECT(ID) COVTYPE(VC)
/SAVE=RESID.
```

**Exploratory 2 – Treatment condition effects**

**During treatment** (Data selected: Time=0 | Time=2).

```
MIXED MCQ_score WITH Time_log Group
/CRITERIA=CIN(95) MXITER(1000) MXSTEP(100) SCORING(1) SINGULAR(0.000000000001)
HCONVERGE(0, ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
/FIXED= Time_log Group Time_log*Group | SSTYPE(3)
/METHOD=ML
/PRINT=SOLUTION TESTCOV
/RANDOM=INTERCEPT | SUBJECT(ID) COVTYPE(VC)
/SAVE=RESID.
```

```
MIXED BAI_score WITH Time_log Group
/CRITERIA=CIN(95) MXITER(1000) MXSTEP(100) SCORING(1) SINGULAR(0.000000000001)
HCONVERGE(0, ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
/FIXED= Time_log Group Time_log*Group | SSTYPE(3)
/METHOD=ML
/PRINT=SOLUTION TESTCOV
/RANDOM=INTERCEPT | SUBJECT(ID) COVTYPE(VC)
/SAVE=RESID.
```

```
MIXED YSQ_score WITH Time_log Group
/CRITERIA=CIN(95) MXITER(1000) MXSTEP(100) SCORING(1) SINGULAR(0.000000000001)
HCONVERGE(0, ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
/FIXED= Time_log Group Time_log*Group | SSTYPE(3)
/METHOD=ML
/PRINT=SOLUTION TESTCOV
/RANDOM=INTERCEPT | SUBJECT(ID) COVTYPE(VC)
/SAVE=RESID.
```

**Across all three time points:** Same syntax as above, with the exception of: (Data selected: All cases)

### **Analyses of reversed causation**

**Concurrent** (Data selected: All cases)

```
MIXED MCQ_score WITH BP_BAI WP_BAI
/CRITERIA=CIN(95) MXITER(1000) MXSTEP(100) SCORING(1) SINGULAR(0.000000000001)
HCONVERGE(0, ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
/FIXED=BP_BAI WP_BAI | SSTYPE(3)
/METHOD=ML
/PRINT=SOLUTION TESTCOV
/RANDOM=INTERCEPT | SUBJECT(ID) COVTYPE(VC)
/SAVE=RESID.
```

```
MIXED YSQ_score WITH BP_BAI WP_BAI
/CRITERIA=CIN(95) MXITER(1000) MXSTEP(100) SCORING(1) SINGULAR(0.000000000001)
HCONVERGE(0, ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
/FIXED=BP_BAI WP_BAI | SSTYPE(3)
/METHOD=ML
/PRINT=SOLUTION TESTCOV
/RANDOM=INTERCEPT | SUBJECT(ID) COVTYPE(VC)
/SAVE=RESID.
```

**Prospective** (Data selected: Time=2 | Time=14)

```
MIXED MCQ_score WITH Time_log BAI_change_02
/CRITERIA=CIN(95) MXITER(1000) MXSTEP(100) SCORING(1) SINGULAR(0.000000000001)
HCONVERGE(0, ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
/FIXED= Time_log BAI_change_02 Time_log*BAI_change_02 | SSTYPE(3)
/METHOD=ML
/PRINT=SOLUTION TESTCOV
/RANDOM=INTERCEPT | SUBJECT(ID) COVTYPE(VC)
/SAVE=RESID.
```

```
MIXED YSQ_score WITH Time_log BAI_change_02
/CRITERIA=CIN(95) MXITER(1000) MXSTEP(100) SCORING(1) SINGULAR(0.000000000001)
HCONVERGE(0, ABSOLUTE) LCONVERGE(0, ABSOLUTE) PCONVERGE(0.000001, ABSOLUTE)
/FIXED= Time_log BAI_change_02 Time_log*BAI_change_02 | SSTYPE(3)
/METHOD=ML
/PRINT=SOLUTION TESTCOV
/RANDOM=INTERCEPT | SUBJECT(ID) COVTYPE(VC)
/SAVE=RESID.
```