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Affective symptoms' associations with metacognitive beliefs, self-reported and performance-measured executive function

Graduate thesis in Clinical psychology

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Abstract

Executive control and metacognitive beliefs are viewed as key contributors to emotional distress and disorder in the theoretical basis for metacognitive therapy, the self-regulatory executive function model. The present study aimed to investigate relative, shared and interacting contributions of these to levels of affective symptoms in a non-clinical sample. The study is the first to include both a self- and performance measure of executive function together with assessments of metacognitive beliefs and affective symptoms. Data were collected from 59 subjects recruited mainly from the NTNU campus, who completed the self-report forms Metacognitions Questionnaire-30, the Behaviour Rating Inventory of Executive Function-Adult, the Hospital Anxiety & Depression Scale, and three rounds of the neuropsychological test Conners Continuous Performance Test 3. Analyses with hierarchical multiple regression supported a moderate associations between metacognitive beliefs and affective symptoms. Performance test variables and self-reported executive function were initially found to significantly account for variance in symptoms, but did not display unique contributions in the final model. One possible interpretation of these results in line the with self-regulatory executive function model is that metacognitive beliefs take precedence in the relationship between executive function and effective symptoms.

Keywords: Metacognitions; Executive function; Cognitive control; Metacognitive therapy; Anxiety; Depression

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Introduction

Cognitive behavioural therapies have long been the standard first line of treatment for most affective disorders (Hollon & Beck, 2013), and have proven effective for many patients, but far from all (Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012; Lambert, 2013). Low to moderate treatment effects, non-adherence, and missing evidence for central theoretical claims about cognitive therapy's effective elements has prompted the development of a third wave of evidence based therapies (Kahl, Winter, & Schweiger, 2012). These therapies emphasise psychological processes and their integration instead of content such as thoughts and schemas, linking clinical practice to evidence about basic processes believed to be involved with the development and maintenance of psychological dysfunction (Kahl et al., 2012; Wells, 2002). One category of these processes is *metacognition*, which refers to: a) beliefs and knowledge about cognition and cognitive processes; b) the regulation of cognition to attain goals through strategies, control, monitoring and appraisal; c) the relationship between a. and b. (A. L. Brown, 1987; Flavell, 1979; Nelson & Narens, 1994). Part of *Metacognitive Therapy* (MCT; Wells, 2002, 2011) is evaluating and targeting metacognitive beliefs such as “my worrying is dangerous” and linked metacognitive processing plans such as tendencies toward threat monitoring and worry. Metacognition is conceptually related to, and overlaps with, *executive functions*: An umbrella term used mostly in cognitive psychology and neuropsychology for functions that guide goal-directed behaviour by controlling and coordinating lower-level processes (such as memory, attention and motor function), usually requiring some mental effort (Banich, 2009; Goldstein, Naglieri, Princiotta, & Otero, 2014). Both maladaptive metacognition and problems with executive functioning have by themselves been linked to affective disorder and psychological distress both empirically and theoretically (Kurtz & Gerraty, 2009; Rock, Roiser, Riedel, & Blackwell, 2014; Shin, Lee, Kim, & Kwon, 2014; Sun, Zhu, & So, 2017). The empirical relationships between executive function and metacognition has nevertheless received little attention, as have their potential interactions and/or relative overlap with psychological problems. The few studies available indicate some correlation between aspects of executive functions and metacognition, but uses either only self-report or (in one case) only performance measures of executive functioning (Fernie, McKenzie, Nikčević, Caselli, & Spada, 2016; Kraft,

Jonassen, Stiles, & Landrø, 2017; Spada, Georgiou, & Wells, 2010; Spada & Roarty, 2015). The present study seeks to further explore distinctions in the relationship between metacognition, executive functioning and emotional distress by employing both a self-report and a test measure of executive functioning.

Metacognition

Building on the concepts of metacognition, schema theory and findings on the relationship between attention and emotion, Matthews and Wells (2014/1994) proposes the *Self-Regulatory Executive Function* (S-REF) model as an account of metacognitive factors involved in the aetiology of emotional distress and affective disorders. *Metacognitions* in S-REF refer to both implicit plans for cognitive processing derived from metacognitive knowledge, and declarative beliefs linked to these, stored in long-term memory (Matthews & Wells, 2014/1994; Wells, 2002; Wells & Matthews, 1996). These plans for coping strategies, judgement heuristics, attention and memory searches are activated more or less deliberately in the service of self-regulation in response to *intrusions*: Perceived self-relevant threats/discrepancies (of sensory or internal origin) from the lower *automatic processing level*. This *self-regulatory processing* takes place as deliberate online processing amendable to conscious control and is, therefore dependent on attentional resources (Matthews & Wells, 2014/1994).

Processing plans have embedded self-regulation goals, which specify the modification of existing cognitive and metacognitive beliefs depending on the success of implementation. S-REF processing also affects lower-level activity by biasing the activation of networks related to particular threats (Wells, 2002). S-REF processing terminates when goals defined by process plans are met, or is suspended when attention is re-routed to other processes. If goals remain unmet and un-modified, S-REF processing will be re-initiated under the same circumstances as it was initiated before distraction (Matthews & Wells, 2014/1994). Wells (2002) theorises that goal-attainment dictates the person's emotional state, with depression being linked to existing - and anxiety to anticipated, failure. Anxiety or depression is produced by S-REF processing and terminates with distraction when goals are attained, or alternative processing plans are implemented.

Ordinarily, S-REF processing is brief and dynamic, i.e. metacognitions are selected, adapted and successfully support coping strategies and belief modification to accomplish self-

regulation. *Cognitive Attentional Syndrome* (CAS) is used by Wells (2002) as a blanket term for dysfunctional processing strategies that leads to pervasive self-regulatory processing, and therefore sustained negative affect and limitations on concentration and attentional flexibility. Wells (2002) proposes that it is variations of CAS that causes psychological distress such as in anxiety and depression, not the content of thoughts and that CAS is caused by maladaptive metacognitions. These are metacognitive beliefs that prompt repeated negative appraisals, threat monitoring, unhelpful plans for coping (e.g. avoidance, thought suppression) and unrealistic or rigid goals that hinder S-REF termination by belief modification or goal attainment/adjustment. Beliefs about the danger and uncontrollability of worry paired with beliefs about the need to control thoughts are indicated as especially influential in producing anxiety and a “cognitive gridlock” in response to intrusions (Spada, Nikčević, Moneta, & Wells, 2008).

CAS as a theory about the relationship between negative affect, heightened self-focus and metacognition defines concrete principles for therapeutic interventions across disorders: The goal in MCT is to terminate CAS, change metacognitive beliefs enabling CAS, and to learn more adaptive coping strategies for self-regulation (Wells, 2002, 2011). Differences in affective disorder-syndromes are explained by concrete top-down metacognitive and bottom-up cognitive vulnerabilities interacting with CAS. For example, *rumination* and *worry* is believed to be separately related to respectively depression and anxiety (Wells, 2002), and are both characterised by self-regulation attempts involving passive and repetitive focus on possible causes and symptoms of distress (Cartwright-Hatton & Wells, 1997; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Watkins, Moulds, & Mackintosh, 2005). Worry and rumination has been repeatedly indicated to produce negative affect, produce more intrusions, and to impede on problem solving, memory and attention (Borkovec, Robinson, Pruzinsky, & DePree, 1983; Gana, Martin, & Canouet, 2001; S. Hayes, Hirsch, & Mathews, 2008; Krebs, Hirsch, & Mathews, 2010; Nolen-Hoeksema et al., 2008; Ruscio & Borkovec, 2004). According to Matthews and Wells (2014/1994) rumination, threat monitoring, and negative affect, all contribute to limiting the capacity for deliberate attention necessary for adaptive S-REF activity, through capacity limitations and motivational effects. This limitation perpetuates CAS, limiting cognitive resources through increased self-focused and inflexible attention.

In addition to metacognitions interacting with rumination and worry, vulnerabilities in bottom-up cognitive processing may explain why some people are vulnerable to affective disorder and others not. S-REF theory focuses on the vulnerability caused by *attentional bias*, an oversensitivity to threatening, personally relevant information (Matthews & Wells, 2014/1994). Attentional bias in emotional disorder can most likely be attributed to both automatic processing (such as pre-attentive bias and deficient executive functioning) and persevering effects of strategies for self-regulation such as threat monitoring (Matthews & Wells, 2014/1994). Matthews and Wells (2014/1994) remark that regardless of cause, strategic processes might be used to modify bias in lower-order processing. This modification of bias is the basis for the *Attention Training Technique* (ATT; Wells, 2007), an MCT intervention aimed at increasing attention flexibility, freeing up attentional resources, thereby attenuating CAS and indirectly modifying metacognitive beliefs (for example about worry being uncontrollable) (Papageorgiou & Wells, 2000; Wells, 2007). Standalone ATT has so far shown promise as a treatment for anxiety and depression in small scale RCTs (Knowles, Foden, El-Deredy, & Wells, 2016).

MCT also targets metacognition through psychoeducation, mindfulness exercises and exposure (Wells, 2002, 2011). For example, the therapist and patient might try to challenge the metacognitive belief “Worrying can drive me insane” by actively trying to go insane by worrying. Metacognitive beliefs can be measured during assessment/therapy, or for research purposes, using the *Metacognitions Questionnaire* (MCQ-65; Cartwright-Hatton & Wells, 1997), or its’ short-form MCQ-30 and five subscales (Wells & Cartwright-Hatton, 2004). In addition to associations with emotional distress in non-clinical samples (Spada, Mohiyeddini, & Wells, 2008), prospective studies have found overall metacognitions to predict residual changes in anxiety (BAI; Ryum et al., 2017; Yılmaz, Gençöz, & Wells, 2011) and depression (BDI; Yılmaz et al., 2011). In a meta-study by Normann, van Emmerik, and Morina (2014) MCT was found to be effective in the treatment of anxiety and depression and to produce substantial differences in metacognitions that persisted at treatment follow-up. This effect supports the association between symptoms and metacognition, but not the temporal precedence of change to one or the other, or change in metacognitions as the effective component of therapy. Stronger evidence for the mediating role of metacognitions comes from a study on patients treated for obsessive-

compulsive disorder (Solem, Håland, Vogel, Hansen, & Wells, 2009), where metacognitions predicted 22% percent of post-treatment symptom variance.

Executive function

The deliberate online processing described in S-REF theory (Matthews & Wells, 2014/1994; Wells, 2002; Wells & Matthews, 1996) is closely related to the notion of executive functions as used in cognitive psychology and neuropsychology: An umbrella term for functions that guide goal-directed behaviour by controlling and coordinating lower-level processes, usually requiring some mental effort (Banich, 2009; Goldstein et al., 2014). Interest in functions that govern the use of basic processes has roots back to the study of patients with frontal lobe damage, who in spite of average performance on various tests demonstrated pervasive difficulties with among others strategic thinking, emotions, and general everyday functioning (Hanks, Rapport, Millis, & Deshpande, 1999; Szczepanski & Knight, 2014). Pinpointing executive functions is complicated by inconsistency in referring to attention and working memory-functions as executive functions, and by different fields applying their terminology. For instance, “cognitive control” is commonly used in cognitive neuroscience with working definitions synonymously with those for executive functions (Goldstein et al., 2014; Mackie & Fan, 2017).

Neuropsychological tests have been the standard for researching differences in control functions that have been sorted as executive functions (Banich, 2009; Duncan Roger Johnson Michaela Swales Charles Freer, 1997). These are functions like inhibiting an automated response, sequencing and initiating behaviour, creating and maintaining a relevant mental set and switching between goals and sub-goals (Banich, 2009; Goldstein et al., 2014). A common conceptualization of executive functions, based on factor analysis of neuropsychological tests, separates them into the three highly correlated but separate facets: a) *Inhibition*, withholding proponent/dominant/automatic responses); b) *shifting*, flexibility in shifting between operations, tasks and mental sets; c) *updating*, monitoring, coding and revision of working memory representations (Friedman & Miyake, 2004; Friedman et al., 2008; Miyake et al., 2000). This separation and unity of test measured executive functioning is reflected in lesion and imaging studies indicating multiple overlapping networks and substrates to be involved (Chung, Weyandt, & Swentosky, 2014; Mackie & Fan, 2017; Szczepanski & Knight, 2014). Inhibition has later been substituted for a *common executive function factor* accounting for some of the

variance in shifting and updating, and almost all variance in inhibition (Friedman, Miyake, Robinson, & Hewitt, 2011; Friedman et al., 2008). This factor is suggested to reflect the ability to maintain and manage task goals, and use these to bias basic processes – requirements which inhibition-like tasks may be especially sensitive to (Friedman & Miyake, 2017; Friedman et al., 2008; Miyake & Friedman, 2012).

The *Conners Continuous Performance test 3'd edition* (CCPT-3; Conners, 2014) is included in the study as a performance measure indicating general (common factor) executive functioning. Results from the CCPT-3 are believed well suited for this, as the test measures both the inhibition of prepotent responses, and requires the maintenance and adjusting of task sets to efficiently coordinate responding (Ballard, 2001; Riccio, Reynolds, Lowe, & Moore, 2002; Soreni, Crosbie, Ickowicz, & Schachar, 2009). CCPT-3 is a test in the *Continuous Performance Test* (CPT) family, whose main protocol consists in subjects responding quickly and accurately to infrequent targets, with the addition of a not-X (do not respond) condition in CCPT. Extensive reviews (Riccio & Reynolds, 2001; Riccio et al., 2002) conclude CPT type tests to be sensitive to general damage and dysfunction, and to be associated with neural substrates for attention and executive functioning. CCPT task activity has been demonstrated as reliably associated with BOLD activity in brain structures associated with executive functioning in healthy participants (Dosenbach et al., 2006; Ogg et al., 2008; Olsen et al., 2013).

Performance-based measures are structured with well-defined goals and limitations, which have raised concerns about these tests' ability to assess real-world problem-solving, especially in novel and unstructured situation (Toplak, West, & Stanovich, 2013). These concerns are one of the reasons why the present study utilises both a self-report and rating-scale measure. The Behaviour Rating Inventory of Executive Function, Adult (BRIEF-A) was designed to be a more ecologically valid measure of everyday problems related to compromised executive functions (Roth, Isquith, & Gioia, 2005). In support of BRIEF-A's relevance to measuring everyday functioning, scores have been demonstrated to correlate significantly with daily life functioning (Garcia-Molina, Tormos, Bernabeu, Junque, & Roig-Rovira, 2012) and have been shown to converge well with assessments of executive functioning problems in neuropsychological reports (Matheson, 2010). On the other hand, correlations between results on BRIEF-A and neuropsychological test are mostly insubstantial (Donders, Oh, & Gable, 2015;

Finnanger et al., 2015; Garcia-Molina et al., 2012; Løvstad et al., 2012; Løvstad et al., 2016), and BRIEF-A scores seem to be more closely related to measures of emotional distress than with performance measures or injury (Løvstad et al., 2012; Løvstad et al., 2016; Schiehser et al., 2011). The reason behind this may be that performance measures and behavioural rating scales measure different, somewhat independent features, of executive functioning: Respectively goal pursuit in unstructured environments versus processing efficiency in structured environments (Toplak et al., 2013). While the prior is assessed by BRIEF-A in this study, individual variation in the latter is measured with CCPT-3.

Compromised executive functioning is featured in a range of psychiatric conditions (Snyder, Miyake, & Hankin, 2015), and has been clearly linked to major depressive disorder (Rock et al., 2014; Snyder, 2013), obsessive-compulsive disorder (Shin et al., 2014), and bipolar disorder (Kurtz & Gerraty, 2009). For all these, light to moderate problems across domains appears in clinical states together with other cognitive symptoms (Kurtz & Gerraty, 2009; Rock et al., 2014; Shin et al., 2014; Snyder, 2013). Domain profiles of executive dysfunction differ somewhat between disorders, but methodological issues and high rates of comorbidity complicates the interpretation of these (Kessler, Chiu, Demler, & Walters, 2005; Snyder et al., 2015). Evidence for a relationship between anxiety syndromes other than OCD and executive functioning is mostly indirect or theoretical, such as trait anxiety and worry in non-clinical samples being related to problems inhibiting competing responses (Bishop, 2009; Eysenck & Derakshan, 2011; Snyder et al., 2010; Snyder et al., 2015).

Low scores on executive functioning tests have been demonstrated to predicts both worry (Crowe, Matthews, & Walkenhorst, 2007; Snyder et al., 2010), rumination (De Lissnyder et al., 2012; Demeyer, De Lissnyder, Koster, & De Raedt, 2012; Whitmer & Banich, 2007; Zetsche, D'Avanzato, & Joormann, 2012) and problems with utilizing healthy coping strategies (Andreotti et al., 2013; McRae, Jacobs, Ray, John, & Gross, 2012), which are all risk factors for psychopathology (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Gana et al., 2001; McLaughlin & Nolen-Hoeksema, 2011; Ruscio et al., 2007; Yang, Cao, Shields, Teng, & Liu, 2017). General executive function limitations across multiple disorders and its connections to common risk factors have been suggested to reflect executive dysfunction as a transdiagnostic cognitive vulnerability (Goschke, 2014; Nolen-Hoeksema & Watkins, 2011). A major caveat when

considering this, is that still little is known about the exact role of executive functioning in developing psychiatric difficulties (Goschke, 2014; Snyder et al., 2015); the present study seeks to make clearer whether metacognitions may be involved in such a role.

Executive function and metacognition

A transdiagnostic model (Nolen-Hoeksema & Watkins, 2011) illustrates how general executive functioning deficits and metacognitive beliefs could interact in paths toward psychiatric difficulties. The most apparent intersection between executive functioning and metacognition as understood in MCT and S-REF-theory is that both describe deliberate processes that bias “lower levels” in order to obtain self-regulation goals. Whereas the literature on executive functioning focuses on general and specific abilities to coordinate and control basic processes, and ways to measure these, S-REF-theory is more concerned with how knowledge and strategy affect this regulation. This difference in theoretical focus is reflected in that while Wells (Matthews & Wells, 2014/1994; Wells, 2011) attributes control over attention in affective disorder as secondary to CAS, authors like Snyder et al. (2015) proposes executive functioning as an important possible risk-factor. Regardless of causality, it seems theoretically plausible that interactions between phenomena described as executive functioning and metacognition will affect mood and functioning, contributing to the force and persisting of affective symptoms.

One of these interactions is the proposed effect of CAS on attentional resources, which in turn may diminish the person’s capacity to regain functional S-REF processing, creating the “cognitive gridlock” that is the target of attention training in MCT (Knowles et al., 2016; Matthews & Wells, 2014/1994; Spada, Nikčević, et al., 2008; Wells, 2011). Kraft et al. (2017) found a significant relationship between metacognitive beliefs about the danger and uncontrollability of worry and lower scores on neuropsychological tests of shifting ability, and offer the interpretation that decreased shifting ability may lead to repeated failures to stop rumination, which in turn feed into beliefs about uncontrollability.

The combination of decreased ability to effectively modulate lower-level processing with dysfunctional metacognitions could make a person especially prone toward worry and rumination, which as mentioned are both related to executive functioning and metacognition. Executive functioning could also affect the person’s ability to execute more adaptive sub-

dominant processing plans for self-regulation (Andreotti et al., 2013; Gotlib & Joormann, 2010; McRae et al., 2012).

Studies looking at relationships between scores on MCQ-30 and the self-reported *Attentional Control Scale* (designed to measure the ability to voluntarily control attention by inhibiting unfavorable dominant responses; Derryberry & Reed, 2002) have found somewhat different patterns of small to moderate correlations with metacognitive beliefs (Ferne et al., 2016; O'Carroll & Fisher, 2013; Spada et al., 2010; Spada & Roarty, 2015). In the studies by Spada et al. (2010) and O'Carroll and Fisher (2013), attentional control and metacognitive beliefs also yielded separate contributions to predicting state anxiety, demonstrating that anxiety relates uniquely to the two. Support for an interaction between the contributions of metacognition and executive functioning was found by Fergus, Bardeen, and Orcutt (2012) in a study utilising self-report measures of CAS-activity, attentional control, stress, anxiety and depression in a non-clinical sample. Interactions between attentional control and the CAS measure explained additional variance for both stress, anxiety and depression scores, but were interpreted cautiously by the authors because of methodological issues. Together, these studies support the relevance of interactions between executive functioning and metacognitions but suffer the general shortcomings of being cross-sectional, using convenience sampling and relying solely on self-report (Ferne et al., 2016; Spada et al., 2010; Spada & Roarty, 2015).

Aim of the study

The present study examines the relationship between individual levels of metacognitive beliefs as conceptualised in S-REF theory, self-reported executive functioning, and a performance measure of executive functioning, with individual levels of self-reported affective symptoms in a non-clinical sample of adults. The study is the first to utilise both self- and performance-reported executive functioning together with a measure of metacognitive beliefs. The following hypothesis is tested: Self- and performance measured executive functioning, and metacognitive beliefs contribute both uniquely and through moderation effects in accounting for variance in affective symptoms. Examining the relative contributions and combined effects of executive functioning and metacognitive beliefs on affective symptoms could be relevant to revising metacognitive models and therapies for affective disorder, for instance on the

importance of including the ATT module depending on a patients profile of difficulties, or on the suitability of metacognitive versus cognitive therapy for certain patients.

Methods

This thesis uses data from the SLEEPIC study (2017) led by NTNU associate professors Ingvild Saksvik-Lehouillier and Alexander Olsen. The project was approved by the Regional Ethical Committee (REK) 06.03.2017, with case number 2017/85. SLEEPIC follows a multiple baseline design, where several instruments are applied multiple times throughout data collection.

Sample

Recruitment and criteria. Participants were recruited through social media, the university intranet, appeals in lectures and with posters on the NTNU campus. An outline of the persons NEO-PI-3 profile and an actigraphy data printout was offered as incentives when recruiting. Prerequisites for participation were: Norwegian language proficiency enough to comprehend and fill out the questionnaires used, being 18-35 year old, and not meeting any of the criteria for exclusion presented in an initial email to those interested in joining. These criteria for exclusion were: Psychiatric, neurological and somatic conditions like substance abuse, personality disorder, severe developmental disorder, acquired brain damage, progressive neurological disorder, respiratory disease or other states that might severely impact functioning.

Recruited sample. The total sample for data collection rounds 1 through 5 was composed of 59 subjects (female = 47; male = 12). Subject age ranged from 19 to 33 ($M = 22,61$; $SD = 2,97$).

Instruments

Metacognitions Questionnaire-30 (MCQ-30). The short form of the *Metacognitions Questionnaire* (MCQ; Cartwright-Hatton & Wells, 1997), *Metacognitions Questionnaire-30* (MCQ-30; Wells & Cartwright-Hatton, 2004), is a self-report inventory designed to measure individual differences in metacognitive beliefs, monitoring tendencies and evaluations (going onward referred to as “metacognitive beliefs”). The inventory consists of 30 four-point Likert-scale items (running from 1 = “do not agree” to 4 = “agree very much”), selected from the MCQ’s 65 to preserve the same five subscales as in the original. These subscales are derived

from factor analysis of MCQ-scores by Cartwright-Hatton and Wells (1997), and include: 1) *Positive beliefs about worry* (PBW; e.g. “Worrying helps me cope”); 2) *Negative beliefs about worrying being uncontrollable and dangerous* (NBW; e.g. “When I start worrying I cannot stop”); 3) *Cognitive confidence* (CC; e.g. “I have poor memory”); 4) *Negative beliefs about thoughts in general* (SPR; e.g. “Not being able to control my thoughts is a sign of weakness”); 5) *Cognitive self-consciousness* (CSC; e.g. “I think a lot about my thoughts”). Negative beliefs about thoughts in general (SPR) was renamed *Beliefs about the need to control thought* (NC) in MCQ-30 (Wells & Cartwright-Hatton, 2004). The five subscales are scored from the raw scores of six items exclusive to each scale so that summarising them provides a metacognitive beliefs total score.

Validation of the MCQ-30 (Wells & Cartwright-Hatton, 2004) with a community sample (N=182) generally confirms the original five-factor structure, reveal intercorrelations matching those of MCQ-65, and show good internal consistency for all scales ($\alpha = 0.72$ to 0.93). Test-retest reliability (M days = 35; SD= 19) was found to be high (total score = $.75$; scales ranging from $.59$ to $.79$), indicating metacognitive beliefs to be relatively stable individual traits (Wells & Cartwright-Hatton, 2004). Convergent and predictive validity is demonstrated by correlations between MCQ-30 scales and measures of depression, anxiety and obsessions (Spada et al., 2010; Spada, Mohiyeddini, et al., 2008; Wells & Cartwright-Hatton, 2004); this especially for NBWs correlations with pathological worry, trait anxiety (Wells & Cartwright-Hatton, 2004), HADS-D and HADS-A (Spada, Mohiyeddini, et al., 2008) and state anxiety (Spada et al., 2010). Normative means from community samples are available from Wells and Cartwright-Hatton (2004) and Spada, Mohiyeddini, et al. (2008), but not for age strata; age and score are negatively correlated for all factors except CC (Spada, Mohiyeddini, et al., 2008).

Only the MCQ-30 total is included for analysis in the present study. This decision was based on considerations about the number of parameters to include to reduce the risk of type 2 error and model overfitting when inferring from a small sample (Field, 2018), and the assumption that meaningful differences in levels of metacognitive beliefs can be picked up by a general factor. Analyses by Fergus and Bardeen (2017) supports the usefulness and measurement invariance across genders of a general MCQ-30 metacognitions factor (scale total), demonstrated to account for 88% of total score variance.

Conners Continuous Performance Test 3 (CCPT-3). *Conners' Continuous Performance Test, third edition* (CCPT-3; Conners, 2014) is a computer-administered Go/NoGo neuropsychological test designed to measure aspects of attention. The respondent is instructed to press the spacebar key as promptly and accurately as possible at any time a target stimulus (bold letters A-Z) appears on the screen, but to do nothing when an "X" is displayed instead. Letters appear for 250 milliseconds and are presented in six blocks, with three 30-letter sub-blocks each. Each sub-block has different inter-stimulus-intervals (ISI); either 1, 2, 3 or 4 seconds. ISI ordering is randomized across blocks. Altogether 232 target stimuli and 36 non-targets are displayed. A high signal-to-noise ratio especially challenges subjects to continuously uphold correct responses, and inhibit the wrong (infrequent) response (Egeland, 2010). Before the 14 minutes long main test, respondents complete a one minute trial round after being instructed according to the technical manual. The trial round can be repeated if considered necessary.

CCPT-3 validation studies demonstrates very strong internal consistency (pooled median $\alpha = .92$) and good test-retest-reliability (pooled median $r = .67$; Conners, 2014). CCPT-3 norms are taken from a representative sample (N=1400) of the US population (Conners, 2014). Results are given as raw scores and as age- and gender-adjusted T-scores. Responses produce several output scores for speed, accuracy and variability in responses, which have been linked to functional domains of attention and executive functions (Conners, 2014; Riccio et al., 2002).

CCPT-3 is included in the present study as a performance measure of general (common factor) executive functioning, as it involves demands related to the maintenance and management of task goals, long term maintenance and management of task-sets biasing basic processes (regulating efficient task-relevant activation under different stimulus presentation conditions), and the short term reactive correction of this biasing (inhibiting proponent response to X) (Friedman & Miyake, 2017; Olsen et al., 2013; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Due to the same methodological considerations as mentioned above for metacognitive beliefs, only a limited number of output scores were included for analysis: *Commission* (COM; responses to non-targets); *omissions* (OMI, non-responses to targets); *mean hit reaction-time* (HRT; Mean time in milliseconds between target presentation and correct response); *Hit Reaction Time Standard Deviation* (HRTSD; Consistency of RT for entire administration). These are the variables most often used to measure response efficiency and

accuracy with CCPT (Homack & Riccio, 2006), and are all considerable contributors to CPT discriminant validity for attentional and executive functioning problems (Conners, 2014; Homack & Riccio, 2006). The variables included corresponds to those used by another study using CCPT-2 as a measure of cognitive control (Wohlwend, Olsen, Håberg, & Palmer, 2017), and are expected to be sensitive to performance in a non-clinical sample (Conners, 2014).

Scores were calculated from the means of primary score t-scores from three separate administrations; all primary score means were weighted equally. Subjects indicated as applying a very liberal or conservative (+/- 3 SD) response style (C; a measure of speed/accuracy trade-off), or a high rate of random/anticipatory responses (+ 3 SD) were excluded from analysis to counteract bias from strategic responding.

Behaviour Rating Inventory of Executive Function, Adult (BRIEF-A). The BRIEF-A (Roth et al., 2005) self-report questionnaire is composed of 75 statements relating to executive function problems in daily life. The surveyee indicates on a three-point scale whether problems have occurred 1: *Never*, 2: *Sometimes* or 3: *Often*, the last six months (Roth et al., 2005). A higher global score indicates a higher experienced burden of executive function-related problems. Raw scores are converted into age-corrected T-scores, where scores over 65 are considered in the clinical range. BRIEF-A offers nine statistically and theoretically derived subscales related to executive function domains: 1. *Inhibit*; 2. *Shift*; 3. *Emotional control*; 4. *Self-Monitor*; 5. *Initiate*; 6. *Working memory*; 7. *Plan/organize*; 8. *Task monitor*; 8. *Organization of materials*. Negativity and inconsistency validity-scales are also included. Responders scoring above the threshold for an invalid response on these were excluded. T-scores are based on Roth et al. (2005) from a normative sample of 1200 informant- and 1050 self-reports. Non-clinical sample Norwegians are demonstrated to score 0.5 to 0.75 SD below the U.S. norm (Løvstad et al., 2016).

Scoring produces a *Global Executive Composite* (GEC; subscales 1-9), in addition to a *Behavioural Regulation Index* (BRI; subscales 1-4) and a *Metacognition Index* (MI; subscales 5-9). The factors are highly correlated but are likely to reflect meaningful functional and anatomical distinctions in executive function (Roth, Lance, Isquith, Fischer, & Giancola, 2013). Still, only the inhibit and Working memory subscales closely parallel lexically similar performance-based test (Toplak et al., 2013), and only *working memory* has been linked to

specific anatomical abnormalities (Garlinghouse, Roth, Isquith, Flashman, & Saykin, 2010). Note also that the “metacognitive” in MCI differs from this words meaning in MCQ-30, referring more to the planning and execution of problem-solving. Only the GEC T-score is included for analysis in the present study, due to the same methodological issues as regarding the MCQ-30, high intercorrelations between factors, and the assumption that a global scale is better suited to pick up variance in a non-clinical population.

In support of BRIEF-A’s validity, clinical populations with known executive function challenges often score higher than controls (Finnanger et al., 2015; Løvstad et al., 2012; Olsen et al., 2014; Roth et al., 2013), self- and informant-report show moderate to high correlations (Donders et al., 2015; Garcia-Molina et al., 2012; Løvstad et al., 2016; Matheson, 2010; Roth et al., 2005), and high convergence with other questionnaires measuring executive function problems has been demonstrated (Roth et al., 2005). BRIEF-A demonstrates good internal consistency (Roth et al., 2013; Waid-Ebbs, Wen, Heaton, Donovan, & Velozo, 2012) and good four-week test-retest reliability (Roth et al., 2005).

Hospital Anxiety and Depression Scale (HADS). The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) is a self-report questionnaire originally designed to measure psychological distress in patients treated for somatic conditions. The questionnaire consists of 14 Likert-scale items relating to symptoms of anxiety and depression over the past two weeks, which are rated on four points from zero (no symptoms) to three (high symptom load). Items focus on psychological symptoms over somatic ones to avoid false positives. With the exception of “panic”, no item explicitly relates to psychiatric symptoms. Responses go into three scales: Total score (HADS-T; item 1-14), depression (HADS-D; item 2, 4, 6, 8, 10, 12 and 14), and anxiety (HADS-A; item 1, 3, 5, 7, 9, 11 and 13) (Zigmond & Snaith, 1983). Five of seven items for HADS-D are related to anhedonia; items for HADS-D are mainly related to symptoms of generalized anxiety (tenseness and worry) (Mykletun, Stordal, & Dahl, 2001). Cutoffs for HADS-A or HADS-D are, following original norms, commonly set to ≥ 8 to indicate probable mild symptoms and the need for further evaluation (Leiknes, Dalsbø, & Siqueland, 2016; Zigmond & Snaith, 1983). In a meta-analysis by Brennan, Worrall-Davies, McMillan, Gilbody, and House (2010) a cutoff of ≥ 8 gave a pooled positive likelihood ratio 4.98 (3.52–7.04) for any depressive disorder and 3.03 (2.02–4.54) for Generalized anxiety disorder.

Norwegian norms are not available, and cutoffs for HADS-T have not been used systematically (Leiknes et al., 2016).

HADS is mostly saturated by a general distress factor explaining around 70% of item variance (Norton, Cosco, Doyle, Done, & Sacker, 2013), and lacks validity in discriminating between depression and anxiety in clinical settings (Brennan et al., 2010; Norton et al., 2013). Factor structure seems largely dependent on sample and method of analysis (Cosco, Doyle, Ward, & McGee, 2012), but is most commonly a hierarchical three-factor solution corresponding to the original scales, which may still be interesting for research purposes (Cosco et al., 2012; Norton et al., 2013). As a general screening instrument, HADS performs with satisfactory sensitivity, but it is not recommended as a case-finding tool in the community populations (Brennan et al., 2010; Leiknes et al., 2016). In a review on the psychometric properties of the Norwegian version of HADS (Leiknes et al., 2016) internal consistency is summarised as high (mean $\alpha < .70$) for all scales. Due to HADS' lacking validity in discriminating between depression and anxiety, only the total raw score was used in analyses, as a measure of participants' general level of affective symptoms.

Data collection

Five periods of data collection was carried out. Round one through three took place between March 13. and May 4. 2017; round four and five between February 19. and March 15. 2018. There was a limit of 15 subjects attending each round. Each round of data collection lasted 11 successive days; only baseline measures from test day 1, 2 and 3 are used in the present study.

Tests were administered in five sessions throughout the 11 days: Day 1 (first), 4 (second) and 8 (third) with usual sleep; day 9 (fourth) and 11 (fifth) after partial sleep deprivation. Participant's attendance times were set to between 07:30 and 10:30. Subjects were asked to abstain from caffeinated drinks during and up to two hours before testing. Variables such as location, test instructors manner, and time spent were standardised as much as possible. Test instructors followed a written script detailing the administration of tests and participation information and were trained by the project's leaders. Test instructors were all 5'th year students or research fellows in psychology.

Test day 1 (day 1). Subjects were presented information about the study and their participation in it, signed an informed consent agreement and answered demographic questions. After this, subjects completed CCPT-3, PANAS, two short 1-10 scales on pain and fatigue answered both before and after CCPT-3, and a 1-10 rating of their effort and performance on the CCPT-3. Next, subjects filled out a questionnaire battery, containing among other instruments HADS, BRIEF-A, MCQ-30 and NEO-PI-3. Subjects were then distributed their sleep diary and Actiwatch Spectrum Pro © and viewed a video recorded instruction for use.

Test day 2 and 3 (day 4 and 8). Subjects completed CCPT-3, the same short rating scales as on day 1, and PANAS.

Data analyses

Data editing and analyses were performed using IBS SPSS 25 and the PROCESS macro version 3.3 (A. F. Hayes, 2017). Variables and methods were selected for further analysis based on theoretical inference, compatibility with statistical assumptions, and from relevant bivariate correlations.

Omission (OMI) were excluded from regression analysis as there was close to zero variance in the measure ($SD = 0.3$ for raw scores compared to 3.9 in normative data; Conners, 2014). The remaining CCPT-3 parameters were investigated for internal consistency and found unsuitable for analysing as a composite measure ($\alpha = .531$); factor reduction using principal component analysis was judged as unadvisable due to small sample size (Mundfrom, Shaw, & Ke, 2005). Remaining CCPT-3 variables were therefore entered into analysis separately.

Simple linear regression was performed to further investigate a bivariate correlation between hit reaction time and metacognitive beliefs. Mediation analysis with HADS scores as mediator was run in PROCESS to investigate whether a known association between lowered speed and affective symptoms could account for the correlation (Conners, 2014).

Ordinary least squares hierarchical multiple regression was performed to investigate to what degree CCPT-3-parameters, metacognitive beliefs (MCQ-30) and self-reported executive function (BRIEF-A) contributes individually or collectively to the prediction of affective symptoms (HADS). Analyses were executed as forced entry in three blocks, adding CCPT-3 parameters at step one to be able to see if they collectively predict affect scores. Three separate

analyses were performed with respectively unedited, winsorized and square root-transformed data, to mitigate possible problems with statistical assumptions. Winsorization and transformation produced only small changes to regression estimates. No standard (i.e. non-crossover) moderation effects were tested due to metacognitive beliefs being the only significant predictor in the final model. Possible crossover moderations were inspected visually (see Appendix A); none was included in the analysis due to insufficient statistical power to reveal small effects. Mediation analysis was executed in PROCESS, to further quantify the relative contribution of metacognitive beliefs and self-reported executive function in the prediction of negative affect scores.

Preliminary data inspection and data editing.

Missing and re-computed values. Two missing BRIEF-A items were replaced by group means before computing totals. Five participants had their CCPT-3 scores computed from two, not three administrations due to absence. BRIEF-A and HADS scores were re-computed to correspond to clinically meaningful values comparable with other research.

Excluded cases. Two participants were excluded from analysis out of validity concerns, in line with recommendations in Connors (2014): One subject with a perservations (anticipatory/random responses) z-score of 5.7; one subject with a response style z-score of -3.58, corresponding to an extreme “speed at the cost of accuracy” strategy.

Data distribution. An overview of q-q plots and distribution statistics (Shapiro-Wilkins) showed some deviations from normality for all scores except BRIEF-A and HRT. These were mainly due to positive skew from flooring effects and positive kurtosis. HRTSD and COM scores trended towards one main and one smaller distribution. HADS and MCQ-30 scores were centred around the median, but with an even (not tapering off) distribution towards higher scores, and a small cluster at higher scores. These departures from normality were judged as natural concerning the concepts being measured and considering sample properties, and not as incompatible with OLS regression. HRT seemed to perform well as a sensitive measure of individual differences in attention and executive function, as displayed by low deviation from normality and acceptable variance.

One borderline outlier ($z = 3.18$) and four extreme values ($z = 1.87 - 2.01$) were identified in COM scores. Six observations ($z = 1.75 - 2.3$) were flagged as extreme values in HRTSD scores. One extreme value ($z = 2.9$) was observed in HRT scores. Two extreme values ($z = 2.57$) were identified in HADS scores. One borderline outlier ($z = 3.03$), was identified in MCQ-30 scores. Outliers were inspected manually and found likely to represent true values. All scores are in a range expected to be observed in a normal population (Conners, 2014; Roth et al., 2005; Wells & Cartwright-Hatton, 2004), but some were extreme compared to the relatively low-dispersion study sample.

Compatibility with statistical assumptions.

Hit reaction time and metacognitive beliefs. Inspection of the scree plot depicted in Figure 1 supported linearity, but Cooks distances and DFBetas showed the correlation to be dependent on three influential cases related to extreme MCQ-30 scores (MCQ-30 total > 80 ; $z = 2.44 - 3.03$). 80% winsorization or removal of any one of these cases cancels a significant correlation. Inspections of predicted versus expected residuals did not indicate obvious problems with heteroscedasticity, non-linearity or independence of errors. The Kolmogorov-Smirnov test indicated acceptable normality (.062) of residuals, but Q-Q plot inspection showed unexpectedly many large residuals at z scores over 2 and under - 1.3.

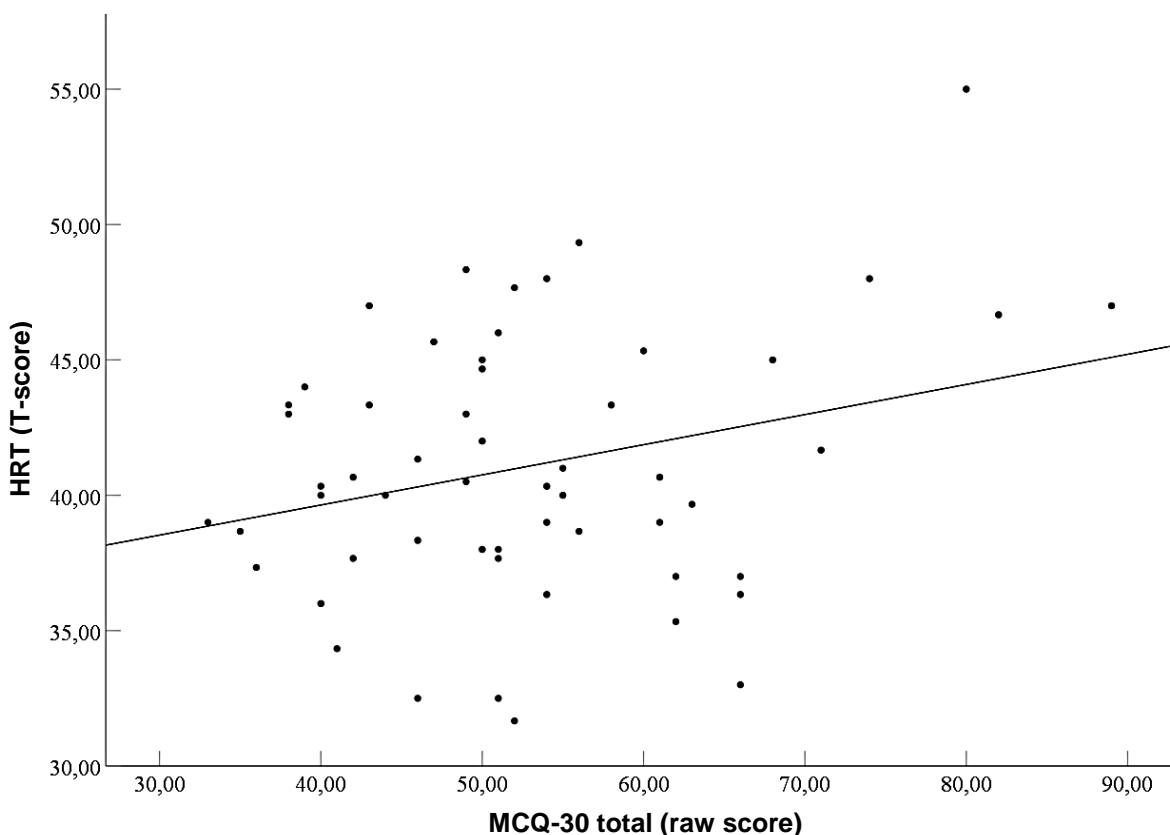


Figure 1. Relationship between CCPT-3 hit reaction time T-scores and scores on the Metacognitions-30 questionnaire. $r = .277$.

Prediction of variance in HADS scores from CCPT-3, BRIEF-A and MCQ-30-scores.

Outliers and influential cases. Inspection of deleted residuals and leverage statistics identified seven influential cases. These cases are summarised in Table A1 in Appendix B. One case was identified as an outlier based Field (2018), and to exert an especially large influence on several betas and on residual distribution normality.

Analysis was performed again with an 80% winsorized sample (all variables) to decrease estimation bias from influential cases; results are presented in Table 3. The same observation was identified as an outlier and was excluded from both analyses due to small sample size, following recommendations in Barnett and Lewis (1994). Outlier removal decreased BRIEF-A GEC beta and p-value away from bordering to significant in the final model, marginally increased the MCQ-30 total beta, and increased R^2 by 15% for the final model.

Collinearity. The model includes no predictors with large simple correlations (see Table 2). Inspection of VIF values and tolerance statistics did not imply problems with multicollinearity, following recommended criteria in Field (2018) of VIF values above 10, and tolerance values below 0.1. Also in line with Field, looking at collinearity diagnostics for condition indexes over 30 implied moderate collinearity between commissions and hit reaction time (condition index 49,7); other condition indexes were acceptable.

Heteroscedasticity. Normality tests and Q-Q plot inspection indicated acceptably normally distributed residuals. Screening of the residuals vs predicted values plot indicated some problems with heteroscedasticity when including MCQ-30-scores as a predictor. The Breusch-Pagan and Koenker test for heteroscedasticity was significant ($p = .003$), indicating non-homogenous residuals (Pryce, 2002). Regression with non-transformed data was therefore computed using wild bootstrapping re-sampled from model residuals, as recommended in MacKinnon (2006) and Astivia and Zumbo (2019) for independent but heteroscedastic errors. Bootstrapping produced marginally lowered standard errors and p values.

Linearity and additivity. The Durbin-Watson test (1,772) indicated acceptable independence of errors (Durbin & Watson, 1951). Visual inspection of individual scatterplots indicated a linear relationship between predictors and HADS scores, and curve estimation from MCQ-30 or BRIEF-A scores did not indicate non-linear functions to be better suited. Visual inspection of residuals vs predicted values plots showed the model to be less accurate for higher predicted values and as possibly indicating problems with linearity.

Square root transformation. Due to possible problems with linearity, unequal variance and influential observations from positive skew, regression was also ran with square root transformed HADS and MCQ-30 data, in line with recommendations in Field (2018). Transformation did not markedly impact model estimates (see Table 3), but improved residual normality, linearity and equality of variance. Analysis of transformed data was run using simple bootstrapping.

Mediation.

Mediation analysis was performed using the PROCESS 3.3 macro (A. F. Hayes, 2017), with MCQ-30 total as the mediator, BRIEF-A GEC as the predictor, and CPT-variables as covariates.

Results are reported for unwinsorized data; analysis with transformed or winsorized data yielded comparable results. As wild bootstrapping was not available for the macro, the HC4 heteroscedasticity-consistent standard error estimator recommended for smaller samples with high leverage cases (Cribari-Neto, 2004; A. F. Hayes & Cai, 2007), was used.

Results

Descriptives

Table 1 presents descriptive for all variables included in analysis. Mean and median HADS total scores corresponded to those from other non-clinical populations (Leiknes et al., 2016). Five participants reported HADS scores in line with mild -and two reported scores in line with moderate affective symptoms. Mean and median MCQ-30 total scores and standard deviation were consistent with those from validation-studies with non-clinical samples (Wells & Cartwright-Hatton, 2004). Mean, and median BRIEF-A global executive composite scores (GEC T-score) were in line with those expected in a Norwegian sample but demonstrated much higher dispersion than in normative data (Løvstad et al., 2016; Roth et al., 2005). CCPT-3 adjusted T-scores showed performance on speed and consistency above average for the sample as a whole, and no scores below average performance (low scores are positive). There was generally high homogeneity in the sample, as expressed by low values on measures of dispersion, except for in BRIEF-A scores.

Table 1.

Descriptives for included CPT parameters and MCQ-30, HADS and BRIEF-A totals. CPT-3 and BRIEF-A scores are given as T-scores, others as raw scores (N = 56).

| Variable | M (95% bootstrapped CI) | SD | 5% trimmed mean | Median | Range /Interquartile range |
|----------|-------------------------|-------|-----------------|--------|----------------------------|
| COM | 49.98 (47.89-52.06) | 7.87 | 49.49 | 48.33 | 38.67 – 75 / 10.08 |
| HRT | 41.09 (39.82-42.35) | 4.77 | 41.03 | 40.5 | 31.67 – 55 / 7 |
| HRTSD | 39.12 (39.82-42.35) | 4.87 | 39 | 38.7 | 31 – 50.33 / 4.8 |
| MCQ-30 | 52.77 (49.60 – 55.95) | 11.96 | 52.06 | 51 | 33 - 89 / 17 |
| BRIEF-A | 42.06 (37.06 – 47.00) | 18.73 | 41.84 | 41 | 5 - 82 / 25 |
| HADS | 8.39 (6.96 – 9.77) | 5.30 | 8.00 | 7 | 1 - 22 / 7.5 |

COM = CCPT-3 commissions T-score; HRT = CCPT-3 hit reaction time T-score; HRTSD = CCPT-3 hit reaction time standard deviation T-score; MCQ-30 = Metacognitions questionnaire-30 raw score total; BRIEF-A = Behaviour Rating Inventory of Executive Function-Adult Global Executive Composite T-score; HADS = Hospital Anxiety and Depression Scale raw score total.

Bivariate correlations. Table 2 presents bivariate correlations for all variables included for analysis. BRIEF-A, MCQ-30 and HADS total scores were found to be moderately correlated with each other ($p < .01$). Correlations found between CPT-scores are in line with those found in normative data (Conners, 2014). No CPT-variables were found to in themselves be correlated with HADS totals, but a significant ($p < .05$) correlation was found between HRT and MCQ-30 totals.

Table 2.

Bivariate correlations for outcome and independent variables (N = 56).

| Variable | 1 | 2 | 3 | 4 | 5 | 6 |
|-------------------|------------------|------------------|----------------|-------------------|----------------|---|
| 1. MCQ-30 | - | | | | | |
| 2. BRIEF-A | .630** (.000) | - | | | | |
| 3. HADS | .652** (.000) | .564** (.000) | - | | | |
| 4. COM | .138 (.306) | .114 (.397) | .114 (.400) | - | | |
| 5. HRT | .277* (.035) | .241 (.241) | .174 (.194) | -.468** (.000) | - | |
| 6. HRTSD | .157 (.244) | -.005 (.971) | .025 (.861) | .388** (.003) | .231 (.084) | - |

* $p < .05$; ** $p < .01$

MCQ-30 = Metacognitions questionnaire-30 total raw score; BRIEF-A = Behaviour Rating Inventory of Executive Function-Adult Global Executive Composite T-score; HADS = Hospital Anxiety and Depression Scale raw score total; COM = CCPT-3 commissions T-score; HRTS = CCPT-3 hit reaction time standard deviation T-score; HRT = CCPT-3 hit reaction time T-score.

Hit reaction time and metacognitive beliefs

Simple linear regression was performed to further investigate the relationship between hit reaction time (HRT) and metacognitive beliefs (MCQ-30 total). Figure 1 presents the relationship as a scatterplot.

Linear regression. Simple linear regression with all observations included showed a significant relationship ($r = .277$, $p < .05$) between hit reaction time and metacognitive beliefs. A Pearson coefficient of .277 suggests that 7.7% more variance in metacognitive beliefs can be explained in a model by adding hit reaction time as a predictor.

Mediation. Mediation analysis with affective symptoms (HADS total) as the mediator demonstrated a significant indirect relationship ($b = 0.108$, 95% BCa CI [-0.095, 0.306]), suggesting that 39.27% of the relationship between reaction time and metacognitive beliefs is shared with affect scores, with a non-significant unique relationship between hit reaction time and metacognitive beliefs in the model.

Predicting affective symptoms from performance- and test measured executive functioning and metacognitive beliefs

Multiple regression. Table 3 displays results from hierarchical multiple regression with unwinsorized, winsorized and square root transformed data; reported findings applies to all data conditions unless otherwise specified. Commissions (COM) and reaction time (HRT) scores from CCPT-3 were significant individual predictors of affective symptoms (HADS total) at step 1 ($p < .01$ for untransformed data; $p < .05$ for transformed data), but non-significant together with hit reaction time variability (HRTSD) in collectively accounting for variance in affective symptoms ($R^2 = .11 - .13$, $p > .05$). At step 2, self-reported executive function (BRIEF-A GEC) together with CCPT-3 variables was found to account for 32-34% of the variance in affective symptoms ($p < .001$), equalling a significant R^2 change of 20-23%. CCPT-3 variables lost significance as predictors at step 2 when partialling out variance explained by self-reported executive function. Adding metacognitive beliefs as a predictor at step 3 increased explained variance in affective symptoms over and above model 2, with the final model accounting for 54-55% of the variance in affective symptoms ($p < .001$), equalling an R^2 change of 20-23%. Self-reported executive function lost significance as a predictor when partialling out variance explained by metacognitive beliefs (MCQ-30 total) at step 3.

Table 3.

Summary of hierarchical multiple regression for variables predicting affect scores (HADS total raw score) for unwinsorized, winsorized, and square root transformed data (N = 56). 95% bias-corrected confidence intervals and standard errors based on 2000 bootstrap samples.

| <u>Unwinsorized data</u> | | | | | | | | | | | | |
|--|---------------------|-----------------|---------|------|---------------------|------------------|---------|------|---------------------|------------------|---------|------|
| Variable | <u>Step 1</u> | | | | <u>Step 2</u> | | | | <u>Step 3</u> | | | |
| | B | SE | β | p | B | SE | B | p | B | SE | B | p |
| COM | 0.27 (0.09, 0.47) | 0.08 | .42 | .001 | 0.11 (-0.06, 0.27) | 0.08 | .17 | .205 | 0.06 (-0.10, 0.23) | 0.08 | .10 | .501 |
| HRTSD | -0.16 (-0.39, 0.01) | 0.12 | -.17 | .189 | -0.02 (-0.24, 0.20) | 0.12 | -.02 | .873 | -0.05 (-0.27, 0.17) | 0.12 | -.05 | .714 |
| HRT | 0.44 (0.13, 0.80) | 0.14 | .42 | .004 | 0.15 (-0.13, 0.46) | 0.15 | .15 | .321 | 0.04 (-0.24, 0.31) | 0.14 | .04 | .809 |
| BRIEF-A | | | | | 0.13 (0.08, 0.20) | 0.03 | .50 | .000 | 0.03 (-0.02, 0.09) | 0.03 | .13 | .274 |
| MCQ-30 | | | | | | | | | 0.26 (0.16, 0.36) | 0.05 | .62 | .000 |
| $R^2 (\Delta R^2)$ | | .13 | | | | .33 (.20) | | | | .54 (.21) | | |
| <i>F</i> change | | 2.55 (p = .065) | | | | 14.09 (p = .000) | | | | 22.93 (p = .000) | | |
| <u>80% winsorized data (all variables)</u> | | | | | | | | | | | | |
| Variable | <u>Step 1</u> | | | | <u>Step 2</u> | | | | <u>Step 3</u> | | | |
| | B | SE | β | p | B | SE | β | p | B | SE | β | P |
| COM | 0.31 (0.06, 0.56) | 0.12 | .44 | .000 | 0.13 (-0.05, 0.27) | 0.12 | .18 | .204 | 0.06 (-0.10, 0.23) | 0.09 | .08 | .290 |
| HRTSD | -0.17 (-0.50, 0.16) | 0.16 | -.17 | .146 | -0.01 (-0.24, 0.20) | 0.15 | -.01 | .965 | -0.05 (-0.27, 0.17) | 0.12 | -.06 | .698 |
| HRT | 0.31 (0.01, 0.62) | 0.15 | .35 | .005 | 0.07 (-0.13, 0.46) | 0.15 | .08 | .553 | 0.04 (-0.24, 0.33) | 0.14 | .04 | .727 |
| BREIF-A | | | | | 0.13 (0.08, 0.18) | 0.03 | .50 | .000 | 0.04 (-0.01, 0.09) | 0.03 | .16 | .155 |
| MCQ-30 | | | | | | | | | 0.27 (0.19, 0.35) | 0.05 | .60 | .000 |
| $R^2(\Delta R^2)$ | | .12 | | | | .32 (.20) | | | | .55 (.23) | | |
| <i>F</i> change | | 2.33 (p = .084) | | | | 14.93 (p = .000) | | | | 26.02 (p = .000) | | |

Square root transformed HADS and MCQ-30 data

| Variable | Step 1 | | Step 2 | | Step 3 | |
|-------------------|----------------|------|-----------------|------|-----------------|------|
| | β | p | β | p | β | P |
| COM | .38 | .040 | .11 | .505 | .04 | .788 |
| HRTSD | -.11 | .523 | .05 | .724 | .02 | .848 |
| HRT | .36 | .040 | .07 | .669 | -.04 | .769 |
| BRIEF-A | | | .53 | .000 | .17 | .202 |
| MCQ-30 | | | | | .62 | .000 |
| $R^2(\Delta R^2)$ | .11 | | .34 (.23) | | .54 (.20) | |
| F change | 2.1 (p = .109) | | 17.4 (p = .000) | | 22.6 (p = .000) | |

COM = CCPT-3 commissions T-score; HRTSD = CCPT-3 hit reaction time standard deviation T-score; HRT = CCPT-3 hit reaction time T-score; BRIEF-A = Behaviour Rating Inventory of Executive Function-Adult Global Executive Composite T-score; MCQ-30 = Metacognitions questionnaire-30 raw score total.

Mediation. The indirect relationship between self-reported executive function (BRIEF-A GEC) and negative affect (HADS total) through metacognitive beliefs (MCQ-30 total), with CCPT-3 variables as covariates, equalled $b = 0.10$, 95% BCa CI [0.05, 0.15]. The mediator accounted for about 74% of the total effect. There was no significant direct relationship between self-reported executive function and negative affect. Variance in affect scores explained by self-reported executive functioning was thus mostly shared with metacognitive beliefs. There was no significant effect of covariates. Results for mediation with untransformed data are summarised in Figure 2.

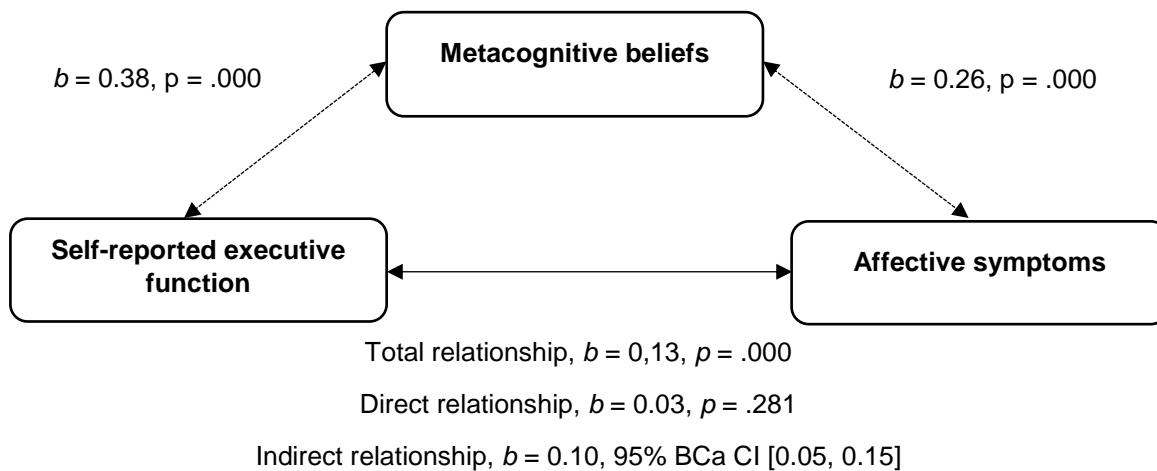


Figure 2. Regression coefficients for the relationship between self-reported executive function (BRIEF-A GEC) and affective symptoms (HADS total) mediated by metacognitive beliefs (MCQ-30 total). Covariates (CPT-parameters) are not included in the figure.

Discussion

The present study sought out to examine the relative and combined contributions of metacognitive beliefs and self- and performance rated executive function in the prediction of affective symptoms in a non-clinical sample of adults. Executive function, metacognitive beliefs and their interactions are implicated both empirically and theoretically in the development and maintenance of affective disorder and emotional distress. The hypothesis tested was: Self- and performance measured executive function and metacognitive beliefs contribute both uniquely and through moderation effects in accounting for variance in affective symptoms.

Simple bivariate correlations showed large associations between affective symptoms (HADS total) and metacognitive beliefs (MCQ-30 total), between affective symptoms and self-reported executive functioning (BRIEF-A Global Executive Composite), and between metacognitive beliefs and self-reported executive function. There was also a small significant correlation between metacognitive beliefs and reaction time (CCPT-3 hit reaction time), and small to moderate associations between CCPT-3 parameters as expected from normative data.

Mediation analysis demonstrated the association between reaction time and metacognitive beliefs to be mediated 39% by affective symptoms, with the direct correlation turning out non-significant. The association was also found to be reliant on three extreme values; two of which were HADS scores in the range of moderate clinical affective symptoms.

Hierarchical multiple linear regression at step 1 demonstrated the performance-measure executive function variables commissions and reaction time (CCPT-3) to be significant ($p < .05$) predictors of affective symptoms when entered together with reaction time variability, but performance-measured executive function as a whole to be non-significant in accounting for variance in affect scores (R^2 ranging from .11 to .13 between data conditions; $p > .05$). Adding self-reported executive function (BRIEF-A Global Executive Composite) at step 2 contributed a

small significant increase (R^2 change ranging from .20 to .23 between data conditions; $p < .001$) in explained variance beyond performance measure variables. Performance measure variables were non-significant as predictors at step 2. Adding metacognitive beliefs (MCQ-30 total) at step 3 contributed a small significant increase (R^2 change ranging from .20 to .23 between data conditions; $p < .001$) in explained variance beyond self-reported executive function. All variables besides metacognitive beliefs were non-significant as predictors after partialling out variance also accounted for by metacognitive beliefs at step 3. The final model was found to account for about half the variance in affective symptoms ($p < .001$), compared to 2/5 for metacognitive beliefs alone ($p < .001$).

Mediation analysis demonstrated the relationship between self-reported executive function and affective symptoms to be mediated 74% by metacognitive beliefs, with the direct association turning out non-significant. This means that most variance shared between self-reported executive functioning and affective symptoms were also shared with metacognitive beliefs, with a non-significant portion of unique variance accounted for by self-reported executive function. No interaction terms were tested due to metacognitive beliefs being the only significant predictor in the final model. Potential crossover interactions were inspected visually and were assessed as too small to be tested due to low statistical power

Interpretation of main findings

Relationship between metacognitive beliefs and affective symptoms. Previous studies with non-clinical samples have demonstrated similar associations as in the present study, with metacognitive beliefs (MCQ-30 total) predicting respectively 61% and 31% of anxiety and depression items measured with HADS (Spada, Mohiyeddini, et al., 2008), and a beta (.34, $p > .001$) similar to the one found in the present study (Spada, Nikčević, et al., 2008). Bivariate correlations between metacognitive beliefs and affective symptoms measured with HADS found in these and in the present study were higher than correlations found in studies of cancer patients undergoing chemotherapy, with mean total HADS scores about four points higher ($SD \approx 7.5$; M. Quattropani, Lenzo, & Filastro, 2017; M. C. Quattropani, Lenzo, Mucciardi, & Toffle, 2016). This difference is likely partially attributable to a different ratio of depression to anxiety symptoms, but could also point towards a non-linear relationship between emotional distress and metacognitive beliefs. Still, a non-linear relationship is hard to reconcile with robust findings on

the association between metacognitive beliefs and affective disorders (Sun et al., 2017) and prompts the question of what the relationship between MCQ-30 and HADS totals in a non-clinical sample represents. Three hypothetical accounts are discussed below:

Measurement overlap. One reason for the strong association between scores on the MCQ-30 and HADS may have to do with measurements being sensitive to similar underlying concepts, i.e. aspects of emotional distress. Tests of MCQ-30 and MCQ-65 convergent validity has focused on relations to measures of trait-anxiety, worry, rumination and obsessive-compulsive symptoms (Wells & Cartwright-Hatton, 2004) – all which are also moderately to strongly associated with HADS scores (Lisspers, Nygren, & Söderman, 1997; McEvoy, Watson, Watkins, & Nathan, 2013; Snorrason, Smári, & Olafsson, 2011; Spada, Hiou, & Nikcevic, 2006). HADS is designed to measure emotional distress in somatic patient populations and has for this reason focused on non-somatic symptoms of anhedonia related to depression, and non-somatic symptoms of generalized anxiety with several items related to worry and restlessness (Zigmond & Snaith, 1983). As seen in Spada, Nikčević, et al. (2008) and Spada, Mohiyeddini, et al. (2008), associations with HADS scores for symptoms of anxiety are twice as large as those for depression. Spada, Mohiyeddini, et al. (2008) attributes this to poor HADS depression scale content validity; another possible explanation is that the observed associations are less indicative of a conceptual relationship, but may reflect similar sensitivities of items related to worry (HADS does not include any items related to rumination, and asks more about anhedonia than about negative affect). A large degree of measurement overlap may be more the result resolution loss by using only scale totals, than of poor MCQ-30 discriminant validity, as subscales have demonstrated meaningful and specific relationships with external measures as predicted from S-REF theory distinct from those for negative affect (Maher-Edwards, Fernie, Murphy, Wells, & Spada, 2011; Solem et al., 2009; Spada et al., 2006; Spada & Wells, 2005).

Measurement overlap could also be the reason why variance in affective symptoms explained by BRIEF-A scores turns out non-significant after partialling out MCQ-30 scores. Løvstad et al. (2016) found scores on BRIEF-A to be highly correlated with emotional distress, and non-significantly with test measures of executive function, in both clinical and non-clinical groups. With this in mind, the shared explained variance in affective symptoms between MCQ-30 and BRIEF-A scores could reflect similar measurement sensitivity to emotional distress. The

variance accounted for over and above by MCQ-30 scores might then simply reflect a higher sensitivity to non-somatic questionnaire items (e.g. worry, anxious apprehension), while items on the BRIEF-A are more likely to overlap with depressive symptoms such as emotional dysregulation, subjective working memory problems and initiating action (Scott, Strong, Gorter, & Donders, 2016), that there are less of on the HADS. Conversely, measures of collinearity indicated the measures to predict variance with acceptable independence, meaning that BRIEF-A and MCQ-30 are most likely not directly measuring the same phenomenon.

Spurious relationship. Another possible reason for the strong correlation between MCQ-30 and HADS scores could be the presence of unaccounted for third variables. Neuroticism and trait anxiety has been demonstrated to have strong associations with both negative affect (Bados, Gómez-Benito, & Balaguer, 2010; T. A. Brown, Chorpita, & Barlow, 1998) and metacognitive beliefs (van der Heiden et al., 2010; Wells & Cartwright-Hatton, 2004). van der Heiden et al. (2010) proposes metacognitive beliefs as a second-order vulnerability factor mediating between neuroticism and affective disorder, based on shared variance between MCQ-30 and neuroticism scores in the prediction of anxiety symptoms, but the cross-sectional design of their study means that the data might also reflect spurious mediation (Fiedler, Schott, & Meiser, 2011). A spurious relationship could, for instance, be the expression of the parallel but not intertwined development of dysfunctional metacognitive beliefs and negative affectivity, or of MCQ-30 items being sensitive to trait properties without reflecting between-level causal processes.

Conceptual association. High overlap with the concepts mentioned above is nonetheless expected for metacognitive beliefs, given their proposed role as second-order mediators between traits and symptoms (Dragan & Dragan, 2014; Dragan, Dragan, Kononowicz, & Wells, 2012). Indirect support for such a role comes from prospective and longitudinal research on worry and rumination: Worry and rumination is demonstrated to causally increase the risk emotional distress (Gana et al., 2001; Huffziger, Reinhard, & Kuehner, 2009; Nolen-Hoeksema et al., 2008), to be affected by high levels of neuroticism (McCullough, Bellah, Kilpatrick, & Johnson, 2001; Ormel & Wohlfarth, 1991; Trapnell & Campbell, 1999), and to explain residual variance in negative affect after controlling for neuroticism (Eisma et al., 2015; Hale III, Klimstra, & Meeus, 2010). Kingston (2013) found metacognitive beliefs about instrumental usefulness to account for residual variance in worry and rumination after controlling for neuroticism and

affective symptoms in a 6-8 week prospective study with a non-clinical sample. These findings strengthen the interpretation of metacognitive beliefs as more than a reflection of neuroticism.

As accounted for in initial sections, the association between MCQ-30- and HADS-scores might also reflect the role of maladaptive metacognitive beliefs as predisposing for negative affect and affective symptoms through CAS-activity, as proposed in S-REF theory (Wells, 2002; Wells & Matthews, 1996). A causal role is suggested by longitudinal and prospective studies having demonstrated metacognitive beliefs to predict symptoms of depression and anxiety (Ruiz & Odriozola-González, 2015; Ryum et al., 2017; Weber & Exner, 2013; Yilmaz et al., 2011). There is also evidence that they can moderate the impact of life stress (Palmier-Claus, Dunn, Morrison, & Lewis, 2011; Ramos-Cejudo & Salguero, 2017; Yilmaz et al., 2011), catastrophic misinterpretation (Bailey & Wells, 2016) and uncertainty intolerance (Ruggiero et al., 2012) on symptoms. Clinical trials have also indicated significant symptom reduction attributable to targeting metacognitive beliefs, after controlling for other predictors (McEvoy, Erceg-Hurn, Anderson, Campbell, & Nathan, 2015; Solem et al., 2009). This change is likely to be mediated by a reduction in patterns of repetitive negative thought, i.e. rumination/worry. These results – suspending notions about metacognitive beliefs as simply a marker for other traits – convincingly indicates metacognitive beliefs as a causal mechanism in the development and sustaining of affective symptoms. In light of this, the current results further support a moderate relationship between general metacognitive beliefs and affective symptoms in non-clinical populations. How this relationship could be integrated with executive functioning is discussed in a separate section.

Relationship between executive function and affective symptoms. As introduced, executive function has been linked to affective symptoms, has been demonstrated to predict worry and rumination, and has thus been proposed as a cognitive risk factor for emotional distress and psychopathology. The following section discusses this role in light of study findings.

Self-reported executive functioning (BRIEF-A). Studies measuring both metacognitive beliefs and self-reported cognitive control (in most of the literature used synonymously with executive function) measured with the Attentional Control Scale (ACS) have demonstrated unique explained variance in performance-related state anxiety with non-clinical samples (O'Carroll & Fisher, 2013; Spada et al., 2010). Two substantial differences here is the use of a

different self-report scale, and measurements being taken at a point where an increased toll on executive function and increased S-REF activity is expected. The ACS has been subject to the same evaluations as BRIEF-A regarding lacking correlations with test measures, and probably being mostly saturated by affective symptoms (Quigley, Wright, Dobson, & Sears, 2017), which supports comparisons with results using BRIEF-A.

The same provisions are relevant in interpreting the correlation between self-reported executive function and affective symptoms as when interpreting the correlation between affective symptoms and metacognitive beliefs. The association between BRIEF-A scores and emotional distress is already mentioned. Additionally, neuroticism has also been demonstrated to correlate with aspects of self-reported executive function (Buchanan, 2016; Murdock, Oddi, & Bridgett, 2013; Robison, Gath, & Unsworth, 2017). One interpretation of this associations is that executive function ability interacts with temperamental negative affectivity by those high on the latter having a greater load of intrusions in daily living (control failure X concerns model; McVay & Kane, 2013). According to *Attentional control theory* (Eysenck, Derakshan, Santos, & Calvo, 2007) neuroticism will primarily affect executive function when meeting complex and/or stressful task demands, as those high in neuroticism are more likely to worry and engage in task-irrelevant rumination/worry that negatively impacts inhibition, switching and updating by competing for cognitive resources. The lack of pressing task-demands when filling out HADS and BRIEF-A might thus be the reason why the current study failed to find unique shared variance between executive function and affective symptoms, while O'Carroll and Fisher (2013) and Spada et al. (2010) did.

The overlap between BRIEF-A- and MCQ-30-scores could also partially be the expression of BREF-A measuring *beliefs* about executive function more than actual performance – as evidenced by the missing correlation also in the present study between test and self-reported executive function. People with higher levels of affective symptoms tend to make biased evaluations about their abilities in multiple domains, including cognitive abilities (Chambless & Gillis, 1993; Hermans et al., 2008), and it is possible that the association between BRIEF-A and HADS scores is more an expression of this than of overlapping measurement sensitivity. The MCQ-30 scales cognitive confidence contains several subjective appraisals of memory, as does BRIEF-A. Again, acceptable collinearity indicates that the same thing is not being measured, but

the measures could be affected differently by a general tendency to monitor and make biased evaluations about cognitive functioning: The MCQ-30 asks about cognitive self-consciousness, and is thus explicitly measuring the same phenomenon that might be influencing BRIEF-A-scores. Response effects such as these are likely to be especially prominent in the current non-clinical sample, consisting of subjects with a presumably low rate of disruptive symptoms. One could, therefore, speculate that the scale would explain unique variance in affective symptoms in another sample. Possible mediation of the relationship between executive function and negative affect is discussed further in its own section.

Test measured executive function (CCPT-3). Other studies using versions of CPT have failed to find substantial correlations between test parameters and self-reported anxiety and depression scores (Hill, Smitherman, Pella, O'Jile, & Gouvier, 2008; Robertson, Kutcher, & Lagace, 2003). The present study found commissions and hit reaction time to be significant predictors of affective symptoms, but these estimates may be somewhat affected by moderate collinearity between the two measures. The combination of slower reaction times and higher rates of false hits (commissions) indicates actual problems with task-relevant mobilisation of cognitive resources, as supposed to strategy effects, e.g. more careful responding. Impacted reaction time and ability to withhold proponent responses (commissions) can both be seen as manifestations of more or less endogenous common factor executive functioning ability, i.e. the ability to maintain and manage task goals, and use these to bias basic processes, i.e. task-relevant activation (Friedman & Miyake, 2017). This interpretation is weakened by the non-significant contribution of reaction time variability (across time and conditions), as problems with response coordination would be thought to result in problems adjusting to new stimulus presentation intervals. Alternative interpretations are discussed below.

One possibility that could account for invariability across conditions is that higher reaction time and more commission could be the expression of known associations between affective disorder and somewhat reduced psychomotor speed, especially for depression (Gualtieri & Morgan, 2008; Marvel & Paradiso, 2004). With regards to commissions, this could be partly attributable to variance made up by the psychomotor component of the go/nogo signals, in a sample that overall scores well above average. A related possibility is that the association is an expression of minor neurobiological deficits underlying both negative affect and reduced

executive function (Etkin, Gyurak, & O'Hara, 2013), or is the result of subtle degenerative effects from periods of depression or anxiety disorder (Lupien, McEwen, Gunnar, & Heim, 2009; McEwen, 2003) flagged by residual symptoms. These interpretations assume linear effects across clinical and non-clinical groups, which is not necessarily the case. Several studies have found an inverted U-relationship between trait anxiety and performance on neuropsychological tasks in non-clinical populations (Bierman, Comijs, Jonker, & Beekman, 2005; De Visser et al., 2010; Salthouse, 2012), while the effect of depressive symptoms appear to be linear (Salthouse, 2012). It should also be noted that medications for the treatment of anxiety and mood disorders could be responsible for the link between (residual) symptoms and performance (Ballard, 1996).

Symptoms of anxiety and depression could relate differently to performance in additional ways. For instance, those with depression or anxiety symptoms may perform worse on tasks demanding high precision because of respectively suppressed or elevated baseline levels of autonomic arousal (T. A. Brown et al., 1998), making it harder to obtain optimal task arousal levels (Mirsky, Anthony, Duncan, Ahearn, & Kellam, 1991). It should be noted that findings on the relationship between arousal and CPT task performance are variable across measures and studies (Ballard, 1996). Motivational effects may also better explain the association with affective symptoms more than ability. People with a higher load of depressive symptoms might engage less to produce fast and correct responding (Treadway, Buckholtz, Schwartzman, Lambert, & Zald, 2009), or have performance on the included parameters affected by the suitability of predominantly approach or avoidance based motivation (Trew, 2011). Individual high on depressive symptoms will also frequently have to expand more effort to mobilize task performance than those low on symptoms (Paulus, 2015), making motivation even more important.

As supposed to BRIEF-A, CCPT-3 data are taken from a situation posing complex task demands, and could, therefore, be more suspect to the indirect effects of underlying neuroticism (Eysenck et al., 2007). The effects of distracting non-task-relevant processes could also be expected to be more linear across conditions than problems with biasing lower processes to reach optimal task-activation. If the correlation between BRIEF-A data and affective symptoms is partly due to third-variable-associations with neuroticism, this might explain why BRIEF-A accounts for all variance in affective symptoms explained by CCPT parameters. The correlation

between worse reaction time and commissions-scores with affective symptoms might also be indicative of vulnerability to CAS activity that interplays with the ability to efficiently coordinate and control task-relevant and irrelevant processes – this is discussed further in the section about possible moderation effects.

Mediation and moderation.

Mediation of the relationship between executive functioning and affective symptoms by metacognitive beliefs. One interpretation of the overlap in explained variance between measures of executive function and metacognitive beliefs is that the association between executive functioning and affective symptoms is mediated by metacognitive processes (rumination, worry, threat monitoring) indicated by answers about metacognitive beliefs. This conceptualisation emphasises the influence of strategy and knowledge over the influence of endogenous cognitive abilities, in line with the focus in S-REF-theory (Matthews & Wells, 2014/1994; Wells, 2011).

Mediation by metacognitive beliefs could come in the form of protracted CAS activity that further exhausts cognitive resources, capacity limitations from ongoing repetitive negative thought (Beckwé, Deroost, Koster, De Lissnyder, & De Raedt, 2014; Connolly et al., 2014; S. Hayes et al., 2008; Wells, 2011), or impeded use of more optimal attention and coping strategies (Dugas, Freeston, & Ladouceur, 1997; Nolen-Hoeksema et al., 2008; van Randenborgh, de Jong-Meyer, & Hüffmeier, 2010). The betas in Figure 2 implies unique variance shared between MCQ-30 and BRIEF-A scores in the present study, which means that the association between them is most likely not only there because of shared sensitivity to emotional distress. This relationship might be an expression of subjective executive function complaints persisting outside periods of heightened affective symptoms, as part of a complicated relationship between metacognitive beliefs and self-perceived executive function.

A person could be vulnerable to executive functioning limitations because of metacognitive beliefs that enable CAS activity (Beckwé et al., 2014), thereby having affective symptoms exacerbated, which could lead to increased executive functioning problems (Snyder et al., 2015). This, in turn, could confirm and elaborate metacognitive beliefs, e.g. negative beliefs about the danger/uncontrollability of worry, or cognitive confidence beliefs about memory deficits (Kraft et al., 2017; Wells & Matthews, 1996). These elaborated metacognitive beliefs could then increase monitoring, worry, rumination and negative judgements of executive

functioning outside periods of increased affective symptoms, and thus act as a vulnerability factor for repeated episodes (Wells, 2011; Wells & Matthews, 1996). For instance, reduced subjective belief in one's memory together with positive beliefs about monitoring to compensate for perceived memory problems could lead to increased internal focus, which again could lead to an increase in intrusions and a decrease in perceiving disconfirming external cues, leading to increased/protracted CAS activity. Subjective evaluations of executive functioning could, in this way, become a self-fulfilling prophecy that also affects test-measured scores.

Moderation of the relationship between executive functioning and affective symptoms by metacognitive beliefs. The above explanation has a somewhat different starting point from an account where people's initial ability to perform executive control interacts with metacognitive beliefs and processes to worsen/uphold affective symptoms, seeing both concepts as individually contributing risk factors. This could for instance happen by individual levels of executive functioning affecting the ability to disengage from CAS activity (Koster, De Lissnyder, Derakshan, & De Raedt, 2011; Kraft et al., 2017; Matthews & Wells, 2014/1994) or the ability to engage in more adaptive subdominant processing and re-appraisal (Andreotti et al., 2013; Gotlib & Joormann, 2010; McRae et al., 2012). Similarly, individual levels of executive functioning could affect the rate of intrusions that enable CAS processing through problems with upholding efficient task focus (McVay & Kane, 2010), or by executive function failures acting as intrusions in their own right. These interactions could then trigger cascades similar to if metacognitive beliefs and subjective cognitive problems or neuroticism was the starting point, making the relationship between subjective and objective executive functioning and metacognitive beliefs even more complicated. One would, in any case, expect to find a linear moderation effect of test measured executive function on negative affect regardless of previous syndromal episodes (Matthews & Wells, 2014/1994). The mentioned study by Fergus et al. (2012) found a weak moderation effect of self-reported cognitive control on the relationship between CAS activity and depressive and anxiety symptoms in a non-clinical sample. The present study had too low statistical power to detect weak interaction effects, but visual inspection (appendix A) speculatively suggests moderation from both test measured and self-reported executive function. Still, caution should be taken in the interpretation of CPT-scores as indicative of stable executive functioning, as they might to a more considerable degree reflect functioning during structured high-demand situations.

Relationship between reaction time and metacognitive beliefs. Only one other study has – to the author's knowledge – investigated associations between test measured executive function and metacognitive beliefs: Kraft et al. (2017) found correlations between shifting ability and beliefs about needing to control thoughts and beliefs about the uncontrollability and danger of worry, after partialling out affective symptoms. The correlations between reaction time and metacognitive beliefs found in the present study were found to be insignificant after partialling out variance shared with affective symptoms, and to be dependent on three influential cases (two of which were HADS scores in the range of moderate clinical affective symptoms). Deliberations about group differences become speculative given the current study's methodology, but one possible interpretation is that the correlation reflects the effect of syndromal CAS activity on affect and executive function performance. A vulnerability to syndromal CAS activity could again be mediated through individual levels of metacognitive beliefs (Matthews & Wells, 2014/1994; Wells, 2011). The association might also reflect confounders, e.g. neuroticism or any other of the previously proposed influences of negative affect on executive function that also has ties to metacognitive beliefs.

Implications

Results from the present study support previous findings on the association between metacognitive beliefs and emotional distress, and further outlines possible mediation effects of metacognitive beliefs on the relationship between executive function and affect. These mechanisms between executive function, metacognitive beliefs and affective symptoms have possible implications for therapy.

The substantial overlap in variance between metacognitive beliefs, executive function and affective symptoms, together with previous studies pointing towards causality (Kingston, 2013; McEvoy et al., 2015; Solem et al., 2009), indicates metacognitive beliefs and executive functioning as valuable targets in the prevention and treatment of affective disorders. Preliminary studies suggest metacognitive therapy to produce clinically significant and long-term changes in treatment-resistant depression (H. M. Nordahl, 2009; Papageorgiou & Wells, 2015; Wells et al., 2009, 2012) and PTSD (Wells, Walton, Lovell, & Proctor, 2015; Wells et al., 2008), compared directly or indirectly with cognitive behavioural therapy as treatment as usual. Including treatment targets such as metacognitive beliefs, metacognitive processes and executive

functioning might be what is needed for the high number of people not helped by current treatment alternatives.

Regarding specific implications for metacognitive therapy, study results underscore the soundness of validating known influences on executive functioning during CAS activity, while also informing about possibly biased evaluations outside of these episodes that might work as self-fulfilling prophecies. This distinction could be especially important in the context of newly occurring affective disorder, and for individuals otherwise at risk for developing concurrent maladaptive metacognitive beliefs and subjective executive function problems. This awareness about the role of subjective cognitive difficulties interacting with metacognitions and affects is already an integral part of metacognitive therapy, which might be part of the reason why it shows promise as a therapy for treatment-resistant occurrences. Future clinical research could assess whether people with different levels of self-reported or performance-measured executive function capacity might better take advantage of metacognitive versus cognitive therapy.

Although executive functions are believed to have a considerable genetic component (Friedman et al., 2011; Friedman et al., 2008), and results from efforts to train executive functions in affective disorder are variable (Bowie, Gupta, & Holshausen, 2013; Motter et al., 2016), there are indications that metacognitive therapy can influence executive function performance. Groves et al. (2015) found moderate ($p = .03$) improvement on the *Groton Maze Learning Task* for the MCT but not the CBT group in a treatment study, independent of changes in affective symptoms, which the authors attributed to the inclusion of the attention training module. Although, these improvements could be attributable to the task being chiefly a measure of working memory, which has demonstrated much better “trainability” than executive function (Bowie et al., 2013). It is also uncertain whether results like these mainly reflect bottom-up neurocognitive change (quantitatively easier to regulate) or the use of more adaptive strategies (easier to regulate because of better strategies). Nonetheless, results from the current study imply that care should be taken, recognising the relative contribution of self-reported versus test-measured executive function, not to substantiate biased self-evaluations by introducing exercises such as the attention training technique in such a way that it could be misunderstood as a cognitive remediation technique.

Methodological strengths and limitations

Design and statistical procedures. Using a cross-sectional design precludes the opportunity to make assumptions about causality, which includes true moderation-effect (Fiedler et al., 2011; Field, 2018). The use and reporting of multiple data conditions with adjustments for deviations from statistical assumptions can be seen as a general strength of the study, seeing as statistics were largely homogenous across conditions and therefore less likely to reflect biased estimates. It is of course possible that the corrections used were not appropriate, and should be replaced with for instance robust regression.

Power. With a sample size of $N = 56$ and five predictors, multiple regression cannot be expected to demonstrate small effects even with low error variance across measures (Kelley, 2013). A weakness of the study design is, therefore, the inability to produce reliable null results, especially considering that effect sizes in similar studies often are found in the range of small to moderate (e.g., Kraft et al., 2017; H. Nordahl & Wells, 2017; O'Carroll & Fisher, 2013).

Instruments and variables. A general limitation across the included measures is the use scale totals where subscales are available, for statistical reasons. Using totals exclusively has both complicated comparisons with studies reporting results for only subscales, has limited the possibility to examine more specific predictions, and may have averaged out important differences within measurements and associations across measures.

MCQ-30. The previously cited study by Fergus and Bardeen (2017) advises continued use of MCQ-30 subscales, as they add (a smaller portion of) reliable variance independent of a general factor. MCQ-30 subscales are also specifically tied to predictions about executive functioning in S-REF theory and relates differently empirically to meaningful outcomes (Ferne et al., 2016; H. Nordahl & Wells, 2017; Solem et al., 2009; Spada & Wells, 2005; Wells, 2011).

HADS. While HADS subscales add little discriminative validity in clinical use, they can still add meaningful distinctions used in research (Leiknes et al., 2016). The anxiety and depression subscales have as discussed demonstrated very different correlations with metacognitive beliefs, and an averaging could wash out meaningful associations. The averaging of anxiety and depression symptoms will also mask discrete associations with executive

functioning, where clear ties to depression and bipolar disorder are much better established than for anxiety (Paulus, 2015; Rock et al., 2014).

CCPT-3. CPT measures are not usually sorted under executive function tests (Strauss, Sherman, & Spreen, 2006), and should mainly be expected to measure aspects of executive functioning related to the control of attention (Conners, 2014). Considering that the role of cognitive control functions proposed in S-REF theory in large refers to attentional flexibility (Matthews & Wells, 2014/1994), this might not be a problem. On the other hand, residual correlations between metacognitive beliefs and executive functions measured with CANTAB in Kraft et al. (2017) were only found for shifting ability, which also relates conceptually to disengagement from CAS activity (Koster et al., 2011; Matthews & Wells, 2014/1994). Further, the inhibition component of CCPT-3 scores might be attenuated compared to in research using CCPT-2, due to a slightly different ratio of targets to non-targets (20% CCPT-2; 10% CCPT-3), which could be seen as further removing the CPT from a valid measure of executive functions. A strength of the CPT parameters included is that they are taken from three averaged administrations, and therefore are more likely to reflect true ability scores and not contextual or state influences.

BRIEF-A. A further limitation to comparability with similar research was the use of BRIEF-A instead of the Attentional Control Scale (ACS). Although they can be argued to likely be broadly comparable (Quigley et al., 2017), ACS focuses on symptoms of attentional focusing and attentional shifting, while BRIEF-A items assess a broader spectrum of executive function. The only study available using both tests (Healy, Treadwell, & Reagan, 2011) found moderate shared variance between ACS totals and the two BRIEF-A subscales Behavioural Regulation Index ($r = .40, p < .001$) and Metacognition Index ($r = .50, p < .001$), but without information about if this shared variance was unique. The current study obtained comparable simple correlations to studies using the ACS, but further interpretations of this finding suffers under the exclusive use of the global composite score.

Sample characteristics, model fit and generalizability. Implications drawn from the present study about useful targets for therapeutic intervention builds on the supposition of a linear relationship between study variables across clinical and non-clinical groups. HADS scores

were as mentioned not normally distributed, showing a deviation from elsewhere positively skewed data that possibly reflected affective syndromes. The size of this cluster still implied a lower prevalence of affective disorder than expected from population prevalence studies (Mykletun, Knudsen, & Mathiesen, 2009), which points toward possible representativeness-issues with the current sample.

Statistics showed only marginal differences across the three data-condition, increasing confidence in that influential data points did not inflate values. Nonetheless, the opposite effect of “washing out” possibly significant effects at the population level could be the result of over-zealously controlling for influential points (Barnett & Lewis, 1994). Especially the removal of one observation that had opposite-signed correlations between HADS and BRIEF-A and MCQ-30 scores markedly increased model fit, but had the effect of moving the BRIEF-A beta away from near significant in the final model, and could have removed a trend that would have become more obvious in a larger sample instead of being an outlier.

Heteroscedasticity between HADS and MCQ-30 scores could be the expression of flooring effects, with a “plateau” of MCQ-30 scores for most people, and increased variability depending on affective symptom aetiology. Effects on error terms were adjusted with bootstrapping, but this could also further point toward a problem with interpreting effects as linear across levels of affective symptoms.

MCQ-30 and HADS scores were used with no adjustment, despite possible age effects (Leiknes et al., 2016; Spada, Mohiyeddini, et al., 2008), but participants’ age ranged only from 19 to 33. While CCPT-3 and BRIEF-A T-scores were age- and gender-adjusted, no measures were adjusted for known effects of education on outcomes (Connors, 2014; Roth et al., 2005). This effect might impact study generalizability, as most study participants attended higher education, and one solution could be to include education and other demographic variables as controls. However, partialling out variance from multiple potential sources of confounders in such a small sample would further underpower analyses to the point of rendering them nonsensical (Kelley, 2013).

Conclusion

The present study further supports metacognitive beliefs to be associated with affective symptoms by demonstrating moderate correlations between total scores on the Metacognitions Questionnaire-30 and the Hospital Anxiety & Depression Scale.

Contrary to study predictions, no unique contributions of executive function were found, as all variance in affective symptoms could be accounted for by metacognitive beliefs in the final model. However, these results should not be taken as a true null finding, as the study was underpowered to detect small effects. The lack of unique contributions from executive function might be the expression the primacy of metacognitive beliefs in the relationship between the three phenomena, as proposed in S-REF theory, but could also reflect confounders or validity problems with the instruments included.

An account where metacognitive beliefs mediate the association between self-assessed executive function and affective symptoms, as supposed to interacting with executive function ability as a separate risk factor, has implications for therapy, and for metacognitive therapy specifically. Biased evaluations of executive function ability, and possible self-fulfilling effects of these evaluations in transactions with metacognitive beliefs in periods of increased cognitive attentional syndrome activity, might contribute to the development and maintenance of affective symptoms. Awareness of these transactions might be the reason why metacognitive therapy shows promise for treatment-resistant occurrences of depression and anxiety disorders.

Research with larger samples, the ability to assess causality, and multiple well-validated measures of executive function performance would be needed to accurately discern the relationship between subjective and objective ability, subscales of metacognitive beliefs and affective symptoms.

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Appendix A

Values at different levels of metacognitive beliefs

Figure A1 through A4 illustrates the relationship between affective symptoms (HADS) and other study variables dependent on different levels of metacognitive beliefs (MCQ-30 totals stratified into three equally large groups: low = $z < -0.43$; high = $z > 0.38$).

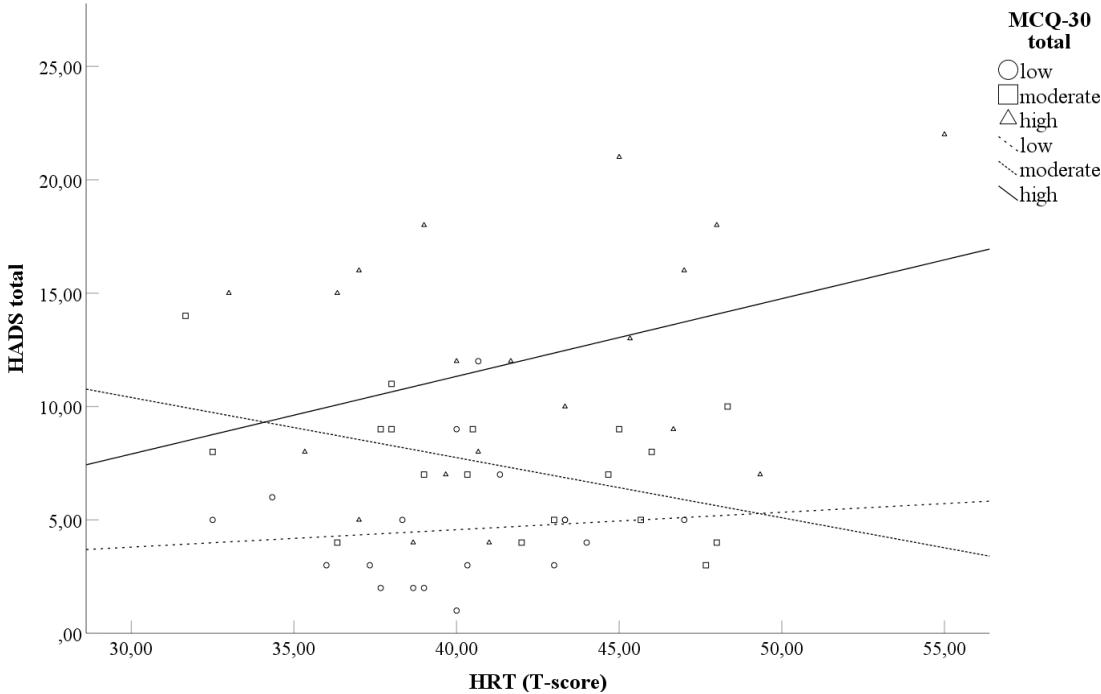


Figure A1. Relationship between CPT-3 hit reaction time T-scores and HADS totals for different levels of MCQ-30-scores.

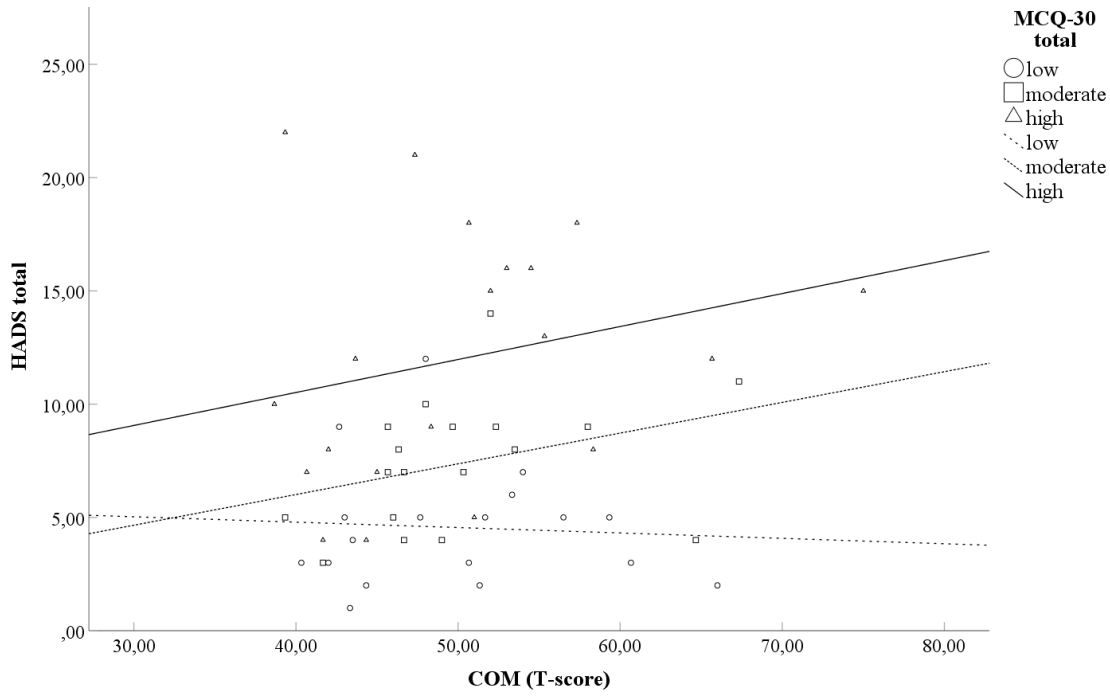


Figure A2. Relationship between CPT-3 commissions T-scores and HADS totals for different levels of MCQ-30-scores.

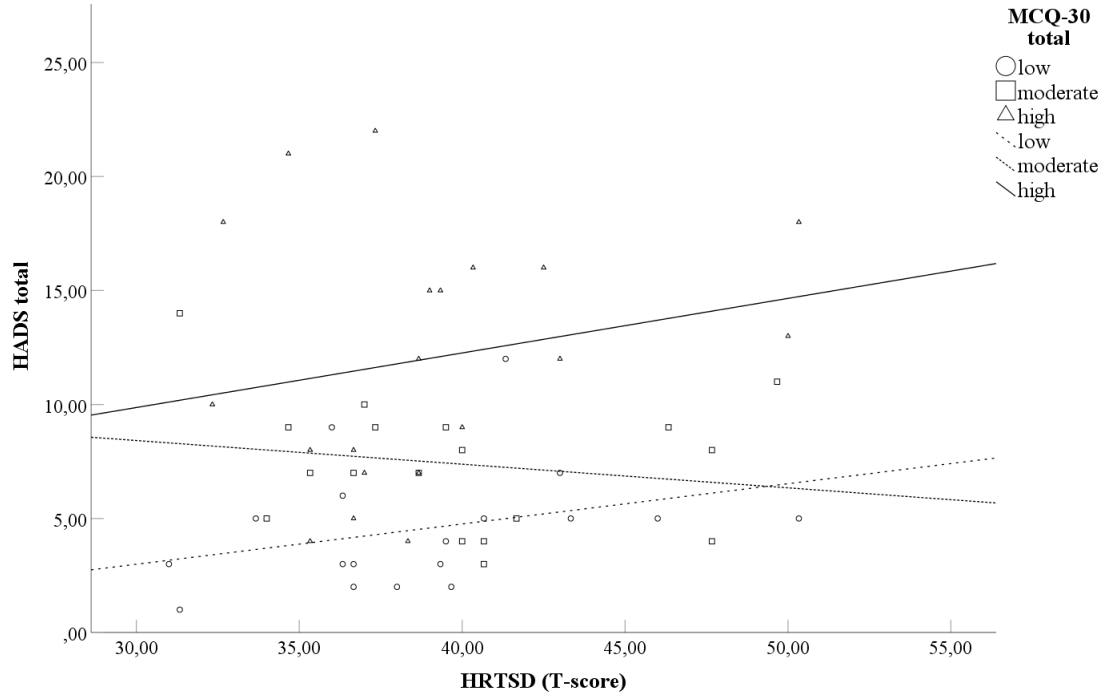


Figure A3. Relationship between CPT-3 hit reaction time standard deviations T-scores and HADS totals for different levels of MCQ-30-scores.

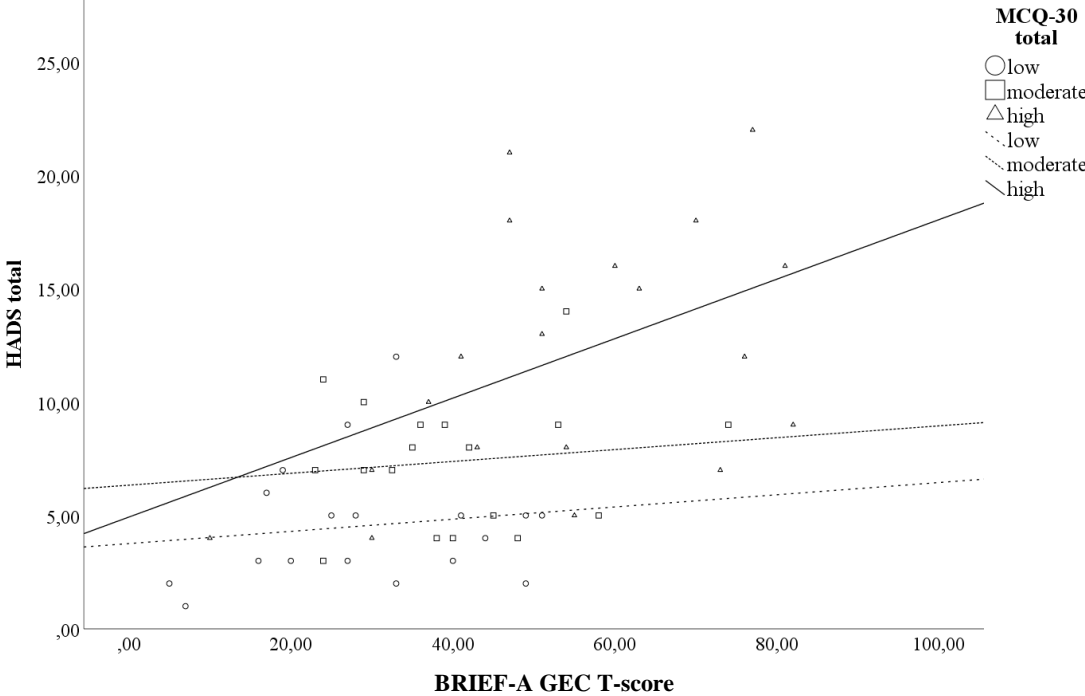


Figure A4. Relationship between BREIF-A Global Executive Composite T-scores and HADS totals for different levels of MCQ-30-scores.

Appendix B

Leverage statistics for multiple regressions

Table B1 presents mean values, leverage statistics and studentized deleted residuals for multiple regression after data inspection and data editing. The criteria used when flagging cases as influential were: DFBetas > 0.25 (Cohen, Cohen, West, & Aiken, 2014); Cooks distances $> .88$ (Cohen et al., 2014); Mahalanobis distances > 17.45 (Barnett & Lewis, 1994); studentized deleted residuals > 3.29 (Field, 2018).

Table B1.

Values and leverage statistics for influential cases

| Case | HADS total | Value / Standardized DFBeta | | | | | Cooks distances | Mahalanobis distances | Studentized del. residual |
|----------------|---------------|--------------------------------|-------------|------------|------------|-------------|--------------------|--------------------------|------------------------------|
| | | MCQ | BREIF | COM | HRT | HRTSD | | | |
| | | -30 | -A | | | | | | |
| a ^a | 22 | 47 / | 63 / | 43.67 / | 41.67 / | 33 / | .22 | 4.7 | 3.7 |
| | | -.70 | .92 | -.20 | -.01 | -.31 | | | |
| b | 9 | 74 / | 47 / | 50.67 / | 48 / | 50.33 / | .14 | 6.9 | -2.1 |
| | | -.47 | -.26 | .12 | -.04 | -.01 | | | |
| c | 22 | 80 / | 77 / | 39.33 / | 55 / | 37.33 / | .11 | 12.5 | 1.3 |
| | | .26 | .02 | .05 | .45 | -.24 | | | |
| d | 21 | 68 / | 47 / | 47.33 / | 45 / | 34.67 / | .09 | 4.9 | 2.1 |
| | | .47 | -.35 | .25 | .36 | -.50 | | | |
| e | 18 | 74 / | 47 / | 50.67 / | 48 / | 50.33 / | .08 | 8.9 | 1.4 |
| | | .35 | -.16 | -.13 | .02 | .38 | | | |
| f | 7 | 56 / | 73 / | 40.57 / | 49.33 / | 38.67 / | .03 | 6.7 | -1 |
| | | .17 | -.27 | .08 | -.12 | -.02 | | | |
| g | 15 | 66 / | 51 / | 75 / | 33 / | 39 / | .02 | 13.2 | 0.6 |
| | | .09 | -.07 | .27 | .04 | -.15 | | | |

Note. Values exceeding recommended cutoff scores in bold.; ^a Excluded from further analysis

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