Volume 1

RESEARCH VOLUME

UNDERSTANDING AND TREATING DEPRESSIVE RUMINATION

by

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Thesis Overview

This thesis was completed as partial requirement for the award of a Clinical Psychology Doctorate.

The first volume contains the research component of the thesis. The research compared individuals diagnosed with depression and individuals without any psychiatric diagnosis on the extent that they engaged in depressive rumination and on their ability to manipulate the contents of their short-term memory. The research findings suggest that individuals with a diagnosis of depression and who ruminated, experience difficulty altering the contents of their working memory when presented with neutrally valenced stimuli. This deficit in the cognitive control of working memory in depressed participants who ruminate could explain why they have difficulty stopping themselves from engaging in depressive rumination, a condition that exacerbates both the symptoms as well as the duration of a depressive episode.

A literature review of the current treatments of depressive rumination identified that current treatments are underpinned by the intention either to reappraise cognitive content or to break the association between behaviours and emotional states, omitting to investigate the potential benefits that improving the ability to control short-term memory may have on reducing the symptom of depressive rumination.

The second volume contains five clinical practice reports. The first clinical practice report demonstrates how a cognitive behaviour formulation and a systemic formulation can be utilised to explain the presented difficulties of an individual with learning disabilities. The second clinical practice report shows the findings of a service evaluation investigating the usefulness of multi-disciplinary team meetings in a learning disability service. The third clinical practice report demonstrates how the single-case design can inform the measurement of the effectiveness of an intervention aiming to improve the symptoms of anxiety in an 'older' adult. The fourth clinical practice report is a case study. It describes the therapeutic process with an individual with dual diagnosis in a secure setting. Finally, clinical practice report five is a summary of an orally delivered case study of a looked after child referred for attention deficit hyperactivity disorder.

Dedicated to my parents

Γιώργος Ζυγούρης   &

Κατερίνα Ζυγούρη

for their unconditional love and support.

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Introduction

Depressive rumination is a type of repetitive thought with deleterious effect on mental health. Depressive rumination is associated with deficits in the control of working memory and has been shown to be a robust predictor of onset and severity of a depressive episode. Consequently, a number of therapeutic approaches for depression endeavour to remediate depressive rumination either directly or by proxy in an effort to alleviate depressed mood and to prevent relapse. Maintenance of depressed mood has been argued to be the result of a deficit in the ability to regulate depressiogenic cognitive structures. This review investigates which of these structures are targeted by current psychotherapeutic interventions.

Method

A computerised search using key words was conducted on a number of academic databases to identify peer-reviewed articles documenting the efficacy of treatments of depressive rumination. Additional references were obtained through the references section of relevant articles and chapters. The resulting articles were arranged thematically under their respective intervention within two broad groups: interventions aimed at restructuring cognitive content and behavioural interventions.

Results

Results identified evidence for both cognitive interventions, such as cognitive therapy, metacognitive therapy and mindfulness therapy, and for behavioural interventions, such as distraction, aversion behavioural activation and graded exposure.

Discussion

Current therapeutic treatments of depressive rumination appear to privilege interventions targeting the reappraisal of cognitive content and the disruption of activation of mood congruent cognitions, whilst omitting to directly remediate the structural deficit in cognitive inhibition of depressiogenic thoughts. Treatments such as cognitive remediation therapy can target such structural cognitive deficits. The article proposes that future research needs to investigate the effectiveness of such interventions for the treatment of depressive rumination and depression.

Keywords: cognitive inhibition, depression, intervention, response styles theory, rumination, treatment.

Response Styles Theory, Cognitive Inhibition and Treatments of Depressive Rumination: A literature review

Helpful and Unhelpful Repetitive Thinking

Repetitive thought is defined as the "process of thinking attentively, repetitively or frequently about one's self and one's world" (Segerstorm, Stanton, Alden & Shortbridge, 2003, p. 909). Depressive rumination is a form of repetitive thought that may be differentiated from other forms of repetitive thoughts by (a) its negative emotional valence, (b) its relatively fixed and invariant content of cognition, (c) its focus upon internal representations rather than on-going experience and (d) because the ruminative process does not aid in problem-solving by producing novel interpretations, evaluations or strategies with respect to the ruminative content. Repetitive and recurrent thinking is a common experience in the absence of psychopathology. In fact, it is argued that repetitive thought is an everyday mental process engaged by all people (Harvey, Watkins, Mansell & Shafran, 2004).

Repetitive thoughts may have both helpful and unhelpful consequences, whereas depressive rumination has only unconstructive sequelae. A helpful repetitive thought can take the form of problem-solving, planning and mental simulation, for example. Problem-solving involves a number of stages, such as definition of the problem, generation of alternative solutions, implementation of the chosen solution and evaluation of its

effectiveness (D'Zurilla & Goldfried, 1971). All stages of problem-solving may involve repetitive thinking. Mental simulation also involves the repeated mental rehearsal of a situation where the desired outcome is achieved. Mental simulation can form an important coping strategy whereby rehearsal of possible events can lead to mastery of optimal behaviours for those events (Pham & Taylor, 1999). Another example of constructive repetitive thought is counterfactual thinking, whereby one generates imagined mental representations of alternative versions of the past (Roese, 1997), thus increasing the likelihood in overcoming similar difficulties in the future. Defensive pessimism is also an example of constructive repetitive thoughts. It is characterised by setting low expectations for future outcomes whilst thinking of the worst case scenario and planning ahead on how to overcome it (Spencer & Norem, 1996). Helpful repetitive thought patterns can aid emotion regulation and are associated with reduced psychopathology.

In the literature on depressive rumination, repetitive thought is more commonly associated with unconstructive thinking. Depressive rumination is only one construct associated with unconstructive thinking, worrying being another seemingly similar construct associated with psychopathology. Although there can be considerable overlap between these constructs, it is generally accepted that they lead to separate expressions of psychopathology. For example, worrying has generally been defined as directing one's attention to "a chain of thoughts and images,

negatively affect-laden and relatively uncontrollable", in anticipation of a future event, which contains the possibility of one or more negative outcomes, with the intention of proactively problem-solving that issue (Borkovec, Robinson, Pruzinsky & Depree, 1983, p. 10). Depressive rumination is differentiated from worrying in that depressive rumination is not characterised by an intention to problem-solve. Instead, it appears to act as an emotion regulation strategy, a "mode of responding to distress that involves the repeated and passive direction of attentional resources on memories of symptoms of distress and on the possible causes and consequences of these symptoms" (Nolen-Hoeksema, Wisco, Lyubomirsky, 2008, p. 400).

The cognitive content of both worrying and depressive rumination is characterised by negative emotional valence. Worrying tends to be associated with repetitive thoughts about the anticipation of an uncertain future event and the experience of symptoms of anxiety, whereas depressive rumination tends to be associated with repetitive thoughts about the past and symptoms of dysphoria (Nolen-Heeksema, Wisco, Lyubomirsky, 2008).

Depressive Rumination

Cross-sectional studies (Riso, du Toit, Blandino, Penna, Dacey, Duin et al., 2003) using the Ruminative Response Scale (RRS; Nolen-Hoeksma, 1991a, Nolen-Hoeksma, 1991b) show that depressive rumination is

elevated for both currently depressed participants as well as participants who have been previously depressed but were in remission at the time of investigation. Depressive rumination has also been found to be associated with symptoms of depression in children, adolescents and adults (Abela, Vanderbilt & Rochon, 2004; Ito, Tornita, Hasui, Otsuka, Katayama, Kawarmura, et al., 2003; Kuyken, Watkind, Holden & Cook, 2006). Using the same measure, prospective longitudinal studies have found that depressive rumination predicts depressive symptoms in patients with clinical depression, after controlling for baseline depression (Rhoan, Sigmon & Dorhofer, 2003) and that it predicts depressive symptoms across a range of follow-up periods in initially non-depressed participants (e.g. Nolen-Hoeksema, Stice, Wade & Bohon, 2007; Sakamoto, Kambara & Tanno, 2001), in addition to the future onset of a major depressive episode across a range of follow-up periods in initially non-depressed participants (e.g. Nolen-Hoeksema, 2000; Spasojevic & Alloy, 2001).

The Response Styles Theory

The response styles theory (RST; Nolen-Hoeksema, 1991a) has received considerable attention within the literature on depressive rumination (Abela, Brozina & Haigh, 2002; Just & Alloy, 1997; Kuehner & Weber, 1999; Nolen-Hoeksema, 2004; Nolen-Hoeksema, Morrow & Fredrickson, 1993; Nolen-Hoeksema, Wisco & Lyubomirsky, 2008; Sarin, Abela & Auerbach, 2005; Strauss, Muday, McNall & Wong, 1997). The response

styles theory postulates that rumination is a trait-like style of responding to low mood. This unhelpful response style exacerbates and prolongs distress, in particular the symptoms of depressive disorder. It is argued that it does this through four mechanisms.

First, rumination enhances the effects of depressed mood on thinking. Consequently, it is more likely that people will utilise the negative memories and thoughts activated by their low mood to make sense of their current circumstances. Teasdale and colleagues (Clark & Teasdale, 1985; Teasdale & Fogarty, 1979; Teasdale, Taylor, & Fogarty, 1980; Watkins & Teasdale, 2001; Watkins, Teasdale, Williams, 2000) have clearly demonstrated the effect of depressed mood on thinking by the selective priming of mood-congruent information and memories, beliefs and expectations. The mood congruent priming of depressiogenic mental representation serves to further intensify negative mood, producing a self-perpetuating cycle of negative cognitive content and dysphoric mood. A considerable body of research supports the idea that self-focus can amplify the effect of negative mood on thinking (e.g. Pyszczynki & Greenberg, 1987, Nolen-Hoeksma & Murrow, 1993; Mor & Winquist, 2002).

Secondly, rumination exacerbates and prolongs depressed mood by generating fatalistic and pessimistic biases when the individual engages in problem-solving. It appears that the combination of low mood and

rumination is depressiogenic. Studies on individuals who have recently experienced low mood due to naturally occurring stress or traumatic life events, who routinely ruminate, experience more severe and longer periods of low mood than individuals who use an alternative emotion regulation strategy (Morrow & Nolen-Hoeksema, 1990; Watkins & Teasdale, 2001). Experimental studies which use rumination induction to dysphoric participants demonstrate a negative bias and distorted interpretations of imaginary life events such as overgeneralising failures whilst dismissing successes (Lyubomisky & Nolen-Hoeksema, 1995; Greenberg, Pyszczynki, Burling & Tibbs, 1992). In addition, similar methodologies demonstrate that ruminators experience less agency and make more negative self-evaluations (Lyubomirksy, Tucker, Caldwell & Berg, 1999).

Thirdly, rumination hinders motivation and promotes engaging in unhelpful behaviours leading to increases in stressful circumstances. For example, when some individuals experience low or dysphoric mood, they may avoid social contact, brood about their problems, dwell upon the consequences and causes of their depressed symptoms, whilst not taking any action to proactively solve their problems (Lyubormirsky, Tucker, Caldwell & Berg, 1999; Nolen-Hoeksema, 1996). In a field study, women who employed a ruminative style experienced higher distress upon discovering symptoms of potential physical illness, which led them to delay seeking appropriate medical advice (Lyubomirsky, Kasri & Chang, 2003).

Finally, depressive rumination could hinder or eliminate protective and repairing strategies that might moderate the effects of depressed mood. For example, social support has been widely demonstrated to moderate the effects of depressed mood (Brown, Andrews, Harris, Adler, & Bridge, 2009; George, Blazer, Hughes, & Fowler, 1989). There is evidence that chronic ruminators are perceived unfavourably by others (Schwartz & McCombs Thomas, 1995) and tend to have less social support available to them (Nolen-Hoeksema & Davis, 1999). This could be there result of experiencing heightened feelings of vengeance (McCullough, Bellah, Kilpatrick & Johnson, 2001), responding with increased aggression when provoked (Collins & Bell, 1997) and tending to experience dependency towards other people (Spasojevic & Alloy, 2001).

Cognitive Inhibition and Emotion Regulation in Depression The response styles theory argues a trait-like quality to depressive rumination. Convergent findings from experimental cognitive psychology (e.g. Mayr & Keele, 2000) as well as neuroimaging (see Banich, Mackiewicz, Cepue, Whitmer, Miller & Heller, 2009) have highlighted the role of cognitive inhibition of working memory in the aetiology of rumination.

Working memory is a limited capacity memory system that provides temporary access to a set of mental representations in the service of on-going cognitive processing (Miyake & Shah, 1999). Since the capacity of

working memory is limited, its contents must be frequently updated if the representations are to be sustained over time. Similarly, mental representations must be "switched" into and out of working memory according to the particular task demands operant at that time.

In order to switch between representations in working memory, the no longer relevant information must be depotentiated or inhibited and the newly attended representations must be potentiated. Inhibition of no longer relevant information may be achieved automatically through competitive inhibition or by self-directed attempts to reallocate attentional resources away from the no longer relevant representations.

Impairment of self-directed inhibitory mechanisms would result in reduced intentional control of working memory representations and in working memory become potentiated by non-goal relevant stimuli (e.g., attention being "grabbed" by the emotional content of the stimuli). Furthermore, the decrease in intentional control of working memory may impede ability to re-evaluate or reappraise automatic reaction to the stimulus (in that the attentional capture by the immediate properties of the stimuli cannot be over-ridden by intentional inhibition). Failure to switch attention away from the ruminative theme could result in maintenance of depressive mood via selective priming of mood-congruent semantic representations becoming more available for the appraisal of on-going experience.

The model presented by Joorman (2010; Figure 1) suggests that negative events trigger mood congruent retrieval of negatively toned cognitions and memories with which to interpret on-going experience. With impairment of the inhibition in working memory the individual will experience greater difficulty switching from negative toned cognitions and memories, resulting in increased rumination on depressiogenic themes, decreased reappraisal of on-going experience and thus further reinforcing the accessibility of negative themed memories and appraisals. Overtime, this process increases long-term retrieval of negative toned material, and reinforces the axiomatic acceptance of the content of depressiogenic rumination. Indeed, the ability to control working memory has been shown to be predictive of recovery from depressive episodes (Joorman & Gotlib, 2010).

From this model, it follows that successful psychological interventions should target one or more of the following modalities:

- Reduce the mood valence and activation of negative cognitions (segment A of the model in Figure 1).
- Remediate the deficits in cognitive inhibition and improve control of working memory (segment B of the model in Figure 1).

- Address the failure of reappraisal and its consequences for the subjective acceptability of the depressiogenic interpretation of on-going experience (segment C of the model in Figure 1).

Negative
Event

A

Negative
Mood
→
Activation of
Negative
←
Cognitions

B

Deficits in
Cognitive
Inhibition

Access of
Negative Material
To WM

Inability to Expel
Negative Material
From WM

C

Rumination ⇧ Reappraisal ⇩ Accessibility of
Mood-Incongruent ⇩
Material

Enhanced Long-Term
Memory for Negative
Material
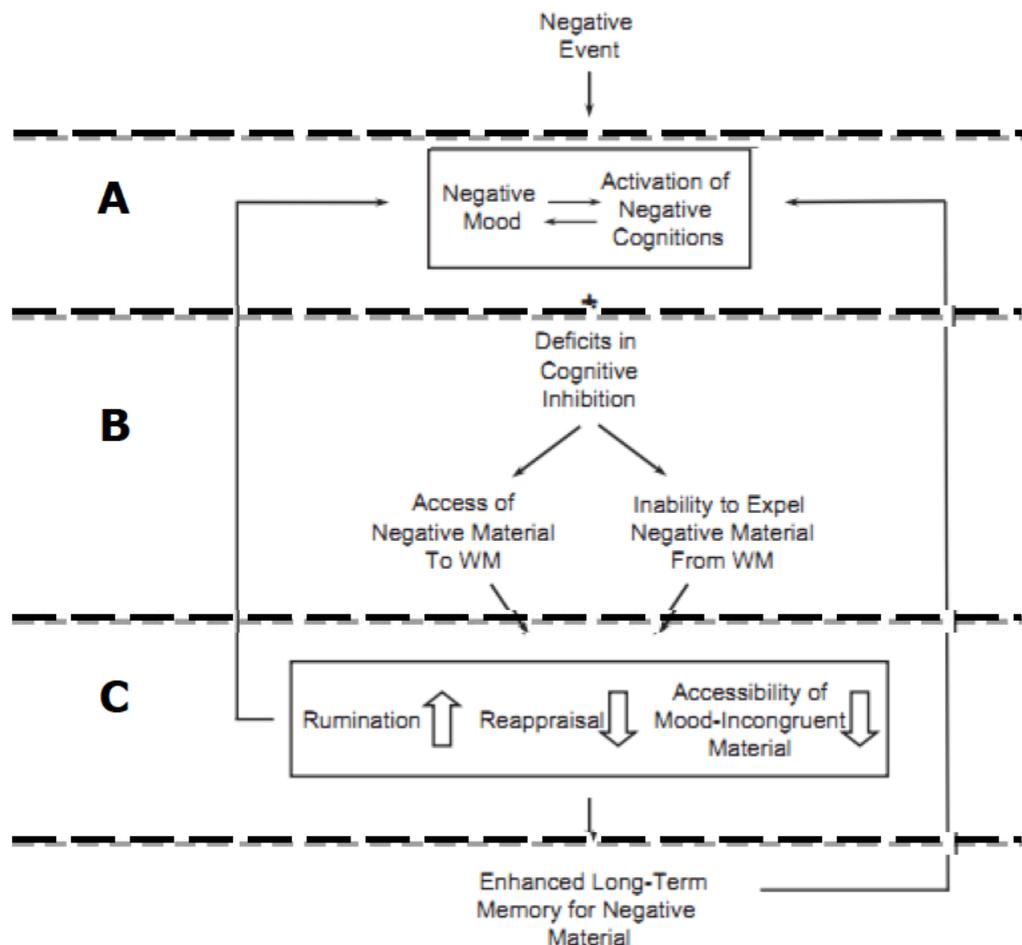
Figure 1. Schematic representation of the Joorman (2010) model linking cognitive inhibition with emotion regulation in depression. Segments A, B and C highlight separate therapeutic objectives of psychological interventions.

The following sections of this article detail a conceptual narrative review aiming to identify the predominant treatment modalities used by current interventions targeting the symptoms of depressive rumination. The review is not an exhaustive attempt to evaluate the effectiveness of the various treatments on depressive rumination. Rather, it tries to identify the driving principles of the existing interventions and suggest future directions drawing form empirical conceptualisations of depressive rumination. Through this review, it becomes apparent that psychological interventions underutilise techniques that intent to remediate the deficit in cognitive inhibition directly. This conceptual narrative review article argues that interventions remediating the ability of cognitive inhibition may prove a successful approach to address this deficit directly and break the cognitive maintenance cycle of depression and or prevent against relapse. The review suggests that further research is required in the use of cognitive remediation training for the treatment for depressive rumination and depression.

Methods

A computerised search using keyword terms was conducted to identify publications investigating treatments of rumination for depression, included the following terms (using wild cards, such as *depres** for *depression, depressed and depressive*). The terms were split into two lists (Table 1), the first list contained references to depression and the second

list contained references to rumination. Each term from the first list was combined with each term from the second list using the Boolean term 'AND'. The terms were entered into a number of academic databases (i.e. PsycINFO, Web of Science, MEDLINE) from the beginning point of that database until April 2012. In addition, relevant chapters (e.g. Papageorgiou & Wells, 2003b) and the reference lists of the obtained articles were reviewed for relevant articles.

Table 1

Terms used for the identification of relevant studies.

List 1	List 2
Depres* (-ed, -ing, -ive)	Ruminat* (-ion, -ive, -ing)
Low mood	Reflect* (-ion, -ive, -ing)
Dysthymi* (-ic, -ia)	Worr* (-y, -ing)
Mental Ill* (-, -ness)	Rehears* (-al, -ing)
Hopeless* (-, -ness)	Chew over
	Contemplat* (-e, -ive, -ion)
	Regurg* (-itate, -itaton)
	Repetit* (-ion, -ive)
	Think (-, -ing)
	Muse

The search strategy resulted in 1043 papers. Of those papers, studies were excluded if they were not peer-reviewed and did not report the outcome of a psychological intervention with explicit reference to depressive rumination. In addition, studies were excluded from this review if the intervention was a combination of treatment approaches already investigated separately (e.g. Cognitive Behavioural Therapy). Since the current review attempts to document the breadth of treatments for the symptom of depressive rumination, there were no specific exclusion criteria regarding a minimum methodological quality. The studies were grouped into two broad thematic categories: interventions aimed at restructuring cognitive content and behavioural interventions.

Interventions Aimed at Restructuring Cognitive Content Three interventions in particular target cognitive content directly: cognitive therapy, metacognitive therapy and mindfulness based cognitive therapy. Cognitive therapy targets beliefs about the self, others and the 'world'. Metacognitive therapy targets beliefs about the usefulness of rumination and the function of emotions. Mindfulness-based cognitive therapy promotes a disengagement from positive, negative and neutral judgements of internal and external experiences.

Cognitive Therapy

Cognitive therapy for depression (Beck, Rush, Shaw & Emery, 1979) has been widely researched and evaluated. Numerous reviews and meta-analyses suggest that it is an effective treatment for mild to moderate depression (e.g. Dobson, 1989; DeRubeis & Crits-Christoph, 1998).

Traditional cognitive therapy attempts to alleviate repetitive distressing cognitive content by either inferential challenge (i.e., manipulation of the subjective beliefs regarding the probability of an adverse event occurring) or evaluative challenge (i.e. manipulation of the subjective beliefs regarding the consequences of an event). In both cases, the challenge is at the level of cognitive content, that is, the emphasis is upon the semantic aspects of the cognition rather than the procedural or algorithmic aspects which may underlie the experience of the specific cognitive content.

Nevertheless, cognitive therapy has been shown to be effective in reducing the length of a depressive episode (Butler, Chapman, Forman, & Beck, 2006) and preventing relapse (DeRubeis & Crits-Christoph, 1998). In contrast, depressive rumination has been linked with prolonged and exacerbated symptoms of depression (Nolen-Hoeksema, 1991a). Therefore, it is possible that cognitive therapy may also promote an indirect reduction in depressive rumination. Indeed, cognitive therapy techniques have been associated with a reduction in depressive

rumination (Watkins, Scott, Wingrove, Rimes, Bathurst, Steiner et al., 2007).

From the perspective of Joorman's (2010) model, traditional cognitive therapy would promote reappraisal of the cognitive content, reducing its emotional valence and thus reducing the attentional capturing effects of the cognition.

Metacognitive Therapy

In contrast to cognitive therapy, metacognitive therapy does not try to challenge the content of depressive thoughts directly. Instead, metacognitive therapy attempts to alter the meaning the depressed individual assigns to the act of ruminating. Metacognitive therapy for depression is a theory driven approach based on the Wells and Matthews' Self-Regulatory Executive Function model (S-REF) of emotional disorders (Wells, 2000; Wells & Matthews, 1994). Wells and Papageorgiou (2004) define four main goals in metacognitive therapy for depression: (1) to introduce the individual to the idea that rumination and selective attention to threat are the core of the problem, (2) to help the individual abandon ruminative thinking and support them to develop flexible control over their attention, (3) to challenge an individual's beliefs that rumination is useful, and (4) to modify the unhelpful beliefs that the individual maintains about experiencing emotions, which contribute to fear and relapse.

Metacognitive therapy has only recently been formalised as a therapeutic approach and as such the empirical evidence for its use in depressive rumination is limited. However, Papageorgiou and Wells (2003a) provide some initial empirical validation for attention training treatment in clinical populations. This is a technique used within the psychological formulation framework of metacognitive therapy (Papageorgiou & Wells, 2000). The only other study exploring the effectiveness of metacognitive therapy (Wells, Fischer, Myers, Wheatley, Patel & Brewin, 2009), has provided some encouraging findings too, however the limited sample size (four patients only) and the lack of a control group limit the generalisation of these results.

Metacognitive therapy, compared to cognitive therapy, moves the focus of the intervention from the content of the depressiogenic beliefs that maintain the negative affect to the beliefs about the ruminative process per se. From the perspective of Joorman's (2010) model, metacognitive therapy intervenes at the level of cognitive content (at the level of inferences and evaluations regarding mental processes). In addition, metacognitive therapy also incorporates strategies to reduce individuals' sustained attentional focus upon the ruminative content. This might include challenging beliefs about the usefulness of rumination as well as simple behaviour strategies to redirect the attention away from the ruminative content. This approach should be differentiated from

interventions aiming at strengthening directly the underlying executive components of working memory.

Mindfulness

Mindfulness involves maintaining receptive attention and non-evaluative moment-to-moment awareness of mental processes and states (Brown, Ryan & Creswell, 2007a). From this perspective, all pleasant as well as unpleasant feelings and thoughts are openly accepted as transient internal states. It is suggested that a purely observant mindful stance allows for unprejudiced information processing (Brown, Ryan & Creswell, 2007b), that is, the individual is encouraged to focus upon on-going experience without engaging in evaluation of the depressiogenic content.

Mindfulness interventions teach breathing and relaxation exercises and promote an openness and acceptance to emotional experiences irrespective of their valence (Zylowska, Ackerman, Yang, Futrell, Horton, Hale, et al., 2008). Mindfulness is associated with higher emotional intelligence (attunement to others), improved self-control as well as reduced symptoms of psychopathology such as neuroticism, anxiety and depression (Brown et al., 2007b). Indeed, mindfulness-based cognitive therapy (MBCT; Segal, Teasdale & Williams, 2002), a protocol-based treatment for emotional disorders, has been shown to be an effective treatment for individuals who suffer with symptoms of depression and to compare favourably with treatment as usual for those who have

experienced three or more major depressive episodes (Ma & Teasdale, 2004). It is suggested that mindfulness interventions work by promoting an experiential self-focus, which reduces analytical self-focus (Watkins & Teasdale, 2001). By reducing analytical self-focus, a depressed individual could learn how to break the learnt association between depressed mood and patterns of negative thinking at various levels (Teasdale & Barnard, 1993).

Ramel, Goldin, Carmona and McQuaid (2004) found that mindfulness training reduced rumination in individuals with lifetime mood disorders (including depression). They postulated that the main change promoted by mindfulness training involved reduction in ruminative thinking. They found that mindfulness training lead to reductions in ruminative thinking, even after controlling for reductions in affective symptoms and dysfunctional beliefs. In addition, a follow-up study suggested that the more mindfulness meditation was reportedly practiced, the less rumination was experienced. Similar results have been reported in other studies (e.g. Deyo, Kimberly, Ong, Koopman, 2009).

Metacognitive therapy attempts to modify the inferential and evaluative aspects of the individual's response to depressiogenic content. By encouraging an "experiential focus", the learnt association between the depressiogenic cognitive content and the evoked emotional reaction is reduced. Once again, and in common with metacognitive therapy, this

mechanism of change does not intend to directly remediate the cognitive control of working memory, which Joorman (2010) suggested was the main vulnerability factor for the development of depressive rumination.

Summary of Cognitive Therapies

Within the Joorman (2010) model, the act of rumination blocks the positive reappraisal of the eliciting situation. Both Cognitive and Metacognitive Therapies seek to facilitate the reappraisal eliciting situation. Cognitive therapy attempts to do this by directly challenging the content of depressive rumination, whereas metacognitive therapy challenges beliefs and behaviours focussed upon the ruminative process itself. Mindfulness-based interventions also attempt to reduce the impact of depressive rumination upon reappraisal by encouraging the individual to take a non-evaluative position with regard to cognitive content.

Overall, it appears that all Cognitive Therapies evaluated are effective in manipulating different aspects of cognitive continent at the level of appraisal. Nevertheless, there is a distinction between cognitive therapy and 'third wave' interventions, such as mindfulness-based cognitive therapy and metacognitive therapy. Whereas cognitive therapy alters the inferences of the actual beliefs, 'third wave' interventions focus upon changing the individuals' thinking about their relationship with those beliefs.

Behavioural Interventions

Cognitive interventions have tended to focus on the reappraisal of depressiogenic cognitive content or evaluative beliefs regarding cognitive processes, behaviours and emotions. Within the context of Joorman's (2010) model, cognitive interventions have tended to predominantly focus upon the blocking of on-going processing of ruminative content and reducing the accessibility of mood incongruent material.

Behavioural interventions derive from classical and operant learning theory. Behavioural interventions tend to focus on abolishing the establishing associations between negative life events and depression related cognitions.

Distraction

Distractive coping is defined as "actively turning one's attention away from one's depressive symptoms on to pleasant or neutral thoughts and actions" (Huffziger & Kuehner, 2009, p. 224). The original response styles theory (Nolen-Hoeksema, 1991a) argued that positive distractions were an adaptive response to depression. Rumination was perceived in direct opposition to distraction. Distraction leads to improved mood through refocusing attention on positive aspects, in contrast to rumination which focuses attentional resources upon the depressiogenic content.

The evidence for the efficacy of distraction is equivocal. Some studies report a negative correlation between rumination and distraction in

clinically depressed populations (e.g. Bagby & Parker, 2001), while other studies report a positive correlation (e.g. Schmaling, Dimidjian, Katon & Sullivan, 2002) or no correlation (e.g. Kuehner& Webber, 1999).

Nolen-Hoeksema, Wisco and Lyubomirsky (2008) argue that these inconsistent findings might be attributable to a lack of standardisation in the cohort of participants with respect to variables such as gender, age and mental health status. They also argue that the way that distraction is operationalized in research studies may be insufficient. Studies investigating the relationship between rumination and distraction tend to utilise the Ruminative Response Scale (RRS, Nolen-Hoeksema, 1991b). The distraction sub-scale of the RRS is comprised of 11 activities which are generally pleasurable, for example, "do something you enjoy" and "go to a favourite place to get your mind off your feelings". These 11 activities are rated on a Likert scale. Individuals with a higher score are considered to have engaged more in distraction. This scale, however, lacks a measure of engagement with the distracting activity. For example, there could be depressed individuals who engage in a number of such activities, whilst still being distracted by their ruminative thoughts. There could also be other depressed individuals who engage in a limited number of distracting activities, but pour their full attention into it and so do not ruminate as much. This explanation would also be in line with laboratory studies which consistently demonstrate that inducing a positive focus on depressed

individuals leads to reductions in depressive affect (Mor & Winquist, 2002).

Accordingly, distraction based interventions attempt to reduce rumination by diminishing the established operations associated with the cognitions and moods that initiate the ruminative process.

Aversion

Thought stopping is a procedure where an aversive stimulus is introduced to produce a response different from the undesirable one (Wolpe, 1973). In this procedure, the therapist shouts "STOP!" or delivers another aversive stimulus such as a painful shock when the treated individual engages in depressive rumination. As the treatment progresses, the treated individual learns to implement the procedure themselves by saying or thinking "STOP!", usually followed by deliberate and positive self-talk. Many variations have emerged whereby the individual inflicts an aversive stimulus upon themselves, such slapping and elastic band on their wrist or pinching themselves. Accordingly, it is possible to view thought stopping as an extreme form of a distraction based intervention.

Thought stopping has a long tradition. It has been shown to be a successful treatment for a range of undesirable psychological conditions such as anger problems (Feindler & Ecton, 1986), obsessional ruminations (Emmelkamp & Kwee, 1977), post-traumatic stress disorder (Hickling & Blancard, 1999) and indeed depression (Peden, Rayens, Hall & Beebe,

2001; Paden, Rayens & Hall, 2005; Peden, Rayens, Hall & Grant, 2005). Nevertheless, thought stopping is considered by some as a treatment only to be used only under strict conditions and with potential adverse effects (Brown, O'Leary & Barlow, 1993; Ellis, 1998; Haddock, Slade, Bentall, Reid & Faragher, 1998).

More recently, some theorists and researchers have argued that it is an ineffective, even counterproductive, technique (e.g. Hannan & Tolin, 2005). For example, Beevers, Wenzlaff, Hayes and Scott (1999) argue that since depressed individuals are already very likely to use thought suppression to control negatively valenced thought, and this appears to worsen depressive mood, alternative techniques to thought suppression should be preferred instead. Such an interpretation of the research findings has been argued to be limited (Bakker, 2009), because depressed individuals may simply have more to suppress or they may be poorer at suppressing and may need additional coaching in this. In addition, evidence does not exist to suggest that pathological symptoms are exacerbated following thought stopping interventions (Bakker, 2009).

Nevertheless, drawing from the Jorrman (2010) model, aversion based interventions attempt to reduce rumination by reducing or abolishing the established operations associated with the cognitions and moods that initiate the ruminative process.

Behavioural activation

A large number of studies suggest that exercise is associated with mental health (Arent, Landers, & Etnier, 2000; Biddle, 2000; Lawlor, 2001). Stathopoulou, Powers, Berry, Smits and Otto (2006) conducted a meta-analysis for the use of exercise as a treatment for depression. The results are compelling and in favour of exercise irrespective of the modality of exercise (i.e. aerobic and resistance training) (e.g. Martinsen, Hoffart & Solberg, 1989). There was, however, a dose-response relationship. For example, Dunn, Trivedi, Kampert, Clark and Chambliss (2005) found that a five-time-a-week workout totalling an expenditure of 17.5 kcal/kg was more effective than a three-time-a-week totalling 7.0 kcal/kg or flexibility exercises.

Stathopoulou et al. (2006) argued that exercise improves well-being through a variety of mechanisms, such as neurochemical, by regulating sleep, by increasing action tendencies in people with depression as well as directly affecting cognitive factors. Craft (2005) studied whether a 9-week exercise programme of moderate-intensity could lead to a change in ruminative response style. All participants were females diagnosed with clinical depression. As anticipated, exercise reduced overall symptoms of depression by the third week, mirroring previous findings. In addition, all participants reported a reduced use of ruminative strategies across time. Similar studies involving male participants have not been conducted. In the light of the evidenced qualitative differences between genders both in

the intensity of depression and levels of rumination (Nolen-Hoeksema, 1991a), these findings may not be consistent between both men and women.

Once again, the mechanism of change associated with behavioural activation interventions is centred on controlling the activation of negative cognitions and negative mood, and thus inhibiting the ruminative cycle.

Graded Exposure and Habituation Training

Gradual exposure involves the controlled exposure to the upsetting stimulus. This leads to repeated activation of the fear structures with concurrent elimination of avoidance rituals and escape behaviours. This procedure tends to lead to a reduction in the anxiety levels surrounding the troubling stimulus. A graded hierarchy of experiences is usually developed collaboratively with the individual who is then exposed sequentially to them. The intervention concludes once there is only minimal anxious arousal in the presence of the troubling stimulus.

In vivo and imaginal gradual exposure has been used with success in a number of psychological disorders characterised by repetitive thought patterns such as Obsessive-Compulsive Disorder (e.g. Antony &Swinson, 2000) and Post-Traumatic Stress Disorder (Foa, Rothbaum, Riggs & Murdock, 1991).

Rachman (1980) and Teasdale (1999) speculated that principles of exposure may apply to treatment of symptoms of depression. Exposure-based cognitive therapy (EBCT; Hayes & Harris, 2000) combines exposure techniques with techniques from cognitive therapies. EBCT is a structured intervention developed around developing toleration of fear of sadness through expressive writing, by gradually increasing affective charge during therapeutic sessions over a period of 20 weeks. Outcome studies on the effectiveness of EBCT are currently limited albeit encouraging (Hayes, Feldman, Beevers, Laurenceau et al., 2007).

The mechanism of change associated with graded exposure and habituation training is focused upon inhibiting the learnt association between internal or external eliciting cognition (often negative cognitions) and negative mood, and thus inhibiting the ruminative cycle described by Joorman (2010).

Summary of Behavioural Interventions

When viewed from the perspective of the Joorman (2010) model, behavioural interventions for rumination predominantly target a reduction in the activation of negative cognitions and of negative mood (segment A of Joorman's model; see Figure 1) whereas Cognitive therapies focus upon the effects of rumination upon the appraisal system (segment C of Joorman's model; see Figure 1). "Traditional" cognitive therapy involves direct challenging of the content of depressive rumination, whereas

metacognitive therapy challenges the beliefs and behaviours regarding the ruminative process itself. Mindfulness-based interventions attempt to control the impact of depressive rumination by encouraging the individual to take a non-evaluative position with regard to the specific depressiogenic cognitive content.

Discussion

There is evidence for the efficacy of interventions aiming at restructuring cognitive content, such as cognitive therapy, metacognitive therapy and mindfulness based cognitive therapy. The mechanisms of change for these therapies appear to be predominately by modifying appraisals made by the ruminating individual. Equally, there is evidence for the efficacy of behavioural interventions. Behavioural interventions appear to work by stopping or disrupting the activation of mood congruent cognitions. These mechanisms of change are expressed within the model of cognitive inhibition and emotion regulation proposed by Joorman (2010). However, this model also predicts that interventions remediating deficits in the control of working memory and specifically in cognitive inhibition would be equally efficacious; however, there is currently no research in this area. Further, there may be clear benefits in terms of reducing vulnerabilities in future episodes of depression if trait-like vulnerabilities to rumination may be remediated.

Clearly this poses an area of omission in the current arsenal of intervention strategies since remediating the individual's 'ability' for cognitive inhibition (for both emotional and non-emotional content) could reduce the likelihood of descending into a spiral of continuous rumination on depressiogenic themes and consequently reduce the likelihood of further recurrence of depressed mood. Furthermore, it is a corollary of Joorman's model (2010) that improving cognitive inhibition skills would not necessarily act in competition to other interventions and may be implemented alongside other interventions to better equip the individual with the requisite skills to protect themselves from future episodes of depression (e.g. Watkins et al., 2007).

Cognitive remediation therapy focuses upon improving specific cognitive functions. It achieves this either by helping the individual exercise the area of poor cognitive function or by helping the individual exercise other compensatory cognitive functions. Cognitive remediation therapy focuses upon the underlying processes rather than the content of thought and perception. It promotes a functional reorganisation of cognitive operations that reduce the use of the deficient cognitive strategy and remedy the underlying neurocognitive deficits. In practice, it involves the engagement of individuals in stimulating activities that target the development of the deficient cognitive skills without the distress of focusing on the negative issues of their mental illness. It has been utilised successfully in people with brain injury (Rohling, Faust, Beverly, Demakis, 2009), dyslexia

(Broom & Doctor, 1995), schizophrenia (Demily & Nicolas, 2008) and anorexia nervosa (Baldock & Tchanturia, 2007).

Attention process training for example (Park, Proulx & Towers, 1999; Sohlberg, Johnson, Paule, Raskin & Mateer, 2001) is a widely used rehabilitation programme designed to remediate attention deficits in individuals with brain injury. The training is comprised of hierarchically organised tasks that exercise different components of attention such as sustained, alternating, divided and selective attention. This programme has been shown to place increased demands on both the attentional control and the working memory systems, indeed, the systems that according to Joorman (2010) are also deficient in individuals who suffer from depressive rumination and depression. There is convincing evidence of the effectiveness of training programmes such as this in individuals with traumatic brain injury (Cicerone, Dahlberg, Kalmar, Langenbahn, Malec, Bergquist, et al.; 2000; Cicerone, Dahlberg, Malec, Langenbahn, Felicetti, Kneipp, et al., 2005; Cicerone, Langenbahn, Braden, Malec, Kalmar, Fraas, et al., 2011), but the effectiveness of such training for the reduction of symptoms of depression remains untested.

In light of the presented evidence, depressive rumination has been argued to be a core and characteristic symptom of depression. Through this review, it has become apparent that current interventions used to treat these conditions focus primarily upon remediating the subjective meaning

that the individual holds about their current condition. By doing so, they place varied, but nevertheless limited, emphasis on increasing cognitive flexibility and remediating cognitive functioning of working memory. This article has highlighted an absence of research investigating the usefulness of attentional and memory remediating techniques on individuals with depression, and proposes that this direction could inform novel interventions and new therapeutic modalities for the treatment of depressive rumination and depression.

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Research Report

COGNITIVE CONTROL OF WORKING MEMORY: COMPARISON OF

DEPRESSED AND NON-DEPRESSED INDIVIDUALS

Abstract

Introduction

Previous research has shown that rumination underpins diverse psychological disorders. Depressive rumination has been associated with poor executive control of working memory when negative emotional valenced stimuli are involved. This leads to poor reappraisal of problematic cognitions and exacerbation of symptoms. Cognitive inhibition appears to be at the core of the dysfunction in the executive control of working memory when valenced stimuli are involved. However, little research exists on the deficits in the executive control of working memory in individuals with depression when presented with neutral stimuli.

Method

The n-back methodology was implemented to compare set inhibition and set switching cost of neutrally valenced information between thirty individuals diagnosed with depression and thirty healthy controls.

Results

Individuals diagnosed with depression experienced significant set switching cost, but not set inhibition cost. Furthermore, rumination was found to be unrelated to both set switching and set inhibition, whereas

depressive symptomatology was found to be associated with set switching cost only.

Discussion

This study suggests the existence of a general deficit in executive control of working memory in those with depression and that this is independent of the valence of presented stimuli. This moves the emphasis away for the emotional significance from the cognitive representations to the cognitive processes. Rumination is discussed in light of these findings, and limitations of the study are highlighted.

Keywords: rumination, depression, set inhibition, set switching memory.

Cognitive control of working memory:

Comparison of depressed and non-depressed individuals

Depressive rumination

Unconstructive repetitive thinking underlies a number of taxonomically diverse mental health problems (Nolen-Hoeksema & Watkins, 2011). Conditions as diverse as post-traumatic stress disorder, obsessive compulsive disorder, social anxiety, depression, dysthymia and schizophrenia include unconstructive repetitive thinking as a core clinical characteristic. Unconstructive repetitive thinking is independent of the degree of physiological arousal, in that it occurs in disorders associated with increased arousal (e.g., OCD, social anxiety, phobia) as well as reduced arousal (e.g., depression and dysthymia).

Depressive rumination has been associated with poor inhibitory control over working memory representations of emotionally valenced stimuli. Theories that are predominantly motivated by clinical considerations have tended to emphasise the attention capture associated with emotionally valenced information (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008; Papageorgiou & Siegle, 2003). This notion is in line with the conceptualisation of depressive rumination in the response styles theory (Nolen-Hoeksema, Wisco & Lyubomirsky, 2008), whereby depressive rumination is perceived as "a mode of responding to distress that involves

repetitively and passively focusing on symptoms of distress and on the possible causes and consequences of these symptoms. [Depressive] rumination does not lead to active problem solving to change [individuals'] circumstances surrounding these symptoms. Instead, people who are ruminating remain fixated on the problems and on their feelings about them without taking action" (Nolen-Hoeksema, Wisco & Lyubomirsky, 2008, p.400).

Alternatively, other models of rumination have postulated a failure of the cognitive control systems to inhibit emotional information that is no longer task relevant. The model presented by Joorman (2010) suggests that negative events trigger mood congruent retrieval of negatively toned cognitions and memories with which to interpret on-going experience. With impairment of the inhibition in working memory the individual will experience greater difficulty switching from negative toned cognitions and memories, resulting in increased rumination on depressiogenic themes, decreased reappraisal of on-going experience and further reinforcing of the accessibility of negative themed memories and appraisals. Overtime this process increases long-term retrieval of negative toned material, and reinforces the axiomatic acceptance of the content of depressiogenic rumination.

The Joorman (2010) model suggests two complementary processes in executive control of working memory. Firstly, the no longer relevant

representations in working memory require depotentiation either automatically through competitive inhibition from the activation of other mental representations or through the self-directed inhibition by the reallocation of attentional resources. Secondly, the current goal relevant representations require potentiation within working memory resources. Accordingly, problematic rumination might occur via a failure of the inhibitory or the potentiating subsystems. The failure to potentiate the current goal relevant representations would result in an inability to switch from the no longer relevant stimulus to the new stimulus. Alternatively, a failure of the inhibitory subsystem would result in increased competition between the no longer relevant stimulus and the goal relevant stimulus such that sustained attention is impeded and the individual finds themselves in constant competition between goal relevant stimuli and the ruminative theme.

Current Research on Cognitive Inhibition in Depression

Deficits in cognitive inhibition may be responsible for a lack of personal control of working memory representations (as suggested by the ruminative response styles theory and the Joorman (2010) model). However, a number of studies have failed to demonstrate these attentional biases in those with depression (MacLeod, Tata & Mathews, 1987; Mogg, Bradley, Williams & Mathews, 1993). Consequently, Williams, Watts, Macleod & Mathews (1997) proposed an alternative model whereby

depression did not affect attention functioning but instead functioned by biasing post-attentional elaboration. Therefore, according to this conceptualisation, depressive biases would be found in recall tasks, but not in selective attention tasks. However, there are some conditions where depressed individuals appear to be characterised by attentional bias. For example, recent dot probe task studies reported selective attention in depression under conditions of long stimuli exposures (Gotlib, Krasnoperova, Yue & Joorman, 2004) and under supraliminal conditions (Mogg et al., 1993). According to Bradley, Mogg and Lee (1997), these results suggest that an attentional bias for negative information exists among depressed individuals. Although depressed individuals may not actively direct their attention towards negative information, once negative information captures their attention, they may have more difficulty in disengaging from it.

It has been suggested that similar processes may underlie both selective retrieval from memory and selective attention in external stimuli (Anderson & Spellman, 1995). Accordingly, if depression leads to inhibitory dysfunction in attention, depression may also lead to inhibitory dysfunction in memory. This memory dysfunction would be particularly evident in tasks of intentional forgetting (Bjork, 1972). The idea behind studies in intentional forgetting is to instruct at some point the participants to forget items that have been presented and encoded. The instruction could be explicit, for example, by informing the participant that

the items will not be tested again. The instruction could also be implicit, whereby a sequential task requires the participant to "get rid" of the code just stored in working memory. To achieve intentional forgetting, the participant would have to utilise inhibitory processes.

A number of studies suggest differential forgetting effects between depressed and non-depressed participants in methodologies that implemented negatively valent stimuli. For example, Power, Dalgleish, Claudio, Tata and Kentish (2000) found that when dysphoric and non-dysphoric participants were asked to make valence judgements about presented words and to remember them at the same time, they both showed similar directed forgetting effects for both positive and negative words. However, when they were asked to rate the presented words in relation to self rather than simply provide pleasantness ratings, the demonstrated differential forgetting where non-dysphoric participants recalled more positive than negative words in the "forget" instruction. Finally, when depressed participants were asked to forget negative adjectives, they performed worse than anxious and normal controls. These findings suggest that when depressed or dysphoric participants are asked to forget negatively valence stimuli, they perform worse at inhibiting the information from their working memory than other participants.

More specifically, Joormann and Gotlib (2010) used a negative affective priming task to assess associations between inhibition and use of

rumination, reappraisal, and expressive suppression. Not only did depressed participants exhibit the predicted lack of inhibition when processing negative material, but reduced inhibition of negative material was associated with greater rumination, less use of reappraisal and more use of expressive suppression.

These findings are exemplary of a deficit in executive control of working memory and have been replicated in a number of studies (e.g. Hertel & Gerstle, 2003; Joorman, Hertel, Brozovich & Botlib, 2005).

Accordingly, there is converging evidence of deficits in executive control processes in individuals with depression. Whitmer and Banich (2007) examined the specific cognitive mechanisms underlying this inhibitory deficit, specifically whether rumination was associated with an inability to switch attention away from old to new information (set-switching cost) or with an inability to effectively inhibit the processing of previously relevant information (set-inhibition cost). Increased executive control of working memory would be indicated by low set-switching cost and high set-inhibition cost. Using the task-switching paradigm reported by Mayr and Keele (2000), Whitmer and Banich (2007) demonstrated that depressive rumination was associated with a deficit in inhibiting prior mental sets (set-inhibition cost) whereas angry and intellectual rumination was associated with difficulties in switching to a new task set (set-switching cost), but not with inhibition of a prior task set. However, doubt remains

as to the generalizability of this effect to individuals with clinical depression as Whitmer and Banich (2007) used a sample of 43 participants selected on the basis of being the top 10% (high ruminators) and bottom 10% (nonruminators) in a population of 776 psychology undergraduates. To date, this effect has not been replicated in individuals who have received a clinical diagnosis of depression.

Design

In the current study, a cross-sectional research design was utilized comparing set-inhibition cost and set-switching cost between individuals with a diagnosis of depression and individuals without a diagnosis.

We hypothesized that people with a diagnosis of depression would have difficulties in the executive control of their working memory. This would be evident by demonstrating difficulties in inhibiting previously relevant information (low set-inhibition cost) and/or by demonstrating difficulties in set-switching (high set-switching cost).

Method

Participants

Twenty-seven females and 35 males participated in this study. The control participants were recruited from undergraduate students participating at the Research Participation Scheme at the University of Birmingham. The

participants meeting inclusion criteria were required to complete both the screening questionnaires as well as the computerised assessment in the same session. The inclusion criteria for this recruitment route were that they were fluent in English and had no significant sensory difficulties and that they scored within the non-clinical range on all of the clinical subscales of the SCL-90-R.

A total number of 45 non-clinical participants were approached. After screening with the SCL-90-R, 14 participants were excluded from the control group as they showed clinically significant scores on one or more of the clinical subscales of the SCL-90-R.

The mental health participants were recruited from patients attending Primary Care Mental Health Teams in the West Midlands area. Potential participants were identified from patients attending community mental health services who were currently experiencing symptoms of depression. The inclusion criteria for the participants attending psychiatric services were that they were fluent in English and had no significant sensory difficulties. Inclusion to the mental health group was defined in terms of on-going receipt of mental health services and a diagnosis of depression.

Measures

The Ruminative Response Styles (RRS) questionnaire (Nolen-Hoeksema & Morrow, 1991) is a 22-item, four point Likert scale with anchors of 1 = 'Never' to 4 = ' Always'. This self-report questionnaire assesses how

frequently participants ruminate on their feelings of sadness or depression. Specifically, it assesses the respondents' degree of absorption with the symptoms and consequences of having a depressed mood. The RRS has good internal consistency (Cronback's a = .82), moderate to high test-retest reliability over one year (r = .47, p < .001) and has been shown to be predictive of depression (Just & Alloy, 1997; Kuehner & Webber, 1999; Nolan, Roberts & Gotlib, 1998; Nolen-Hoeksema, Parker & Larson, 1994; Nolen-Hoeksma, 2000; Spasojevic & Alloy, 2001).

Treynor, Gonzalez and Nolen-Hoeksema (2003) have argued that the RRS is comprised of three subscales, namely reflection, brooding and depression-related. 'Reflection' questions are related with problem-solving and coping, in contrast with 'brooding' questions that focus around self-criticism and the 'depression' related questions that describe general symptoms of depression.

Symptom Checklist 90 – Revised (SCL-90-R; Derogatis, 1994) is a multi-dimensional self-report questionnaire. It consists of 90 items covering eight dimensions of psychological distress: phobic anxiety, anxiety, depression, somatization, obsessive-compulsivity, distrust and interpersonal sensitivity, hostility and insomnia. The overall total score of the SCL-90-R is used to represent psychoneuroticism. Each item is reported on a five-point Likert scale with anchors from 1 – 'Not at all' to 5 = 'Extremely'. The participants are asked to report the extent to which the

symptoms were manifest during the week preceding the day of answering the questionnaire. The SCL-90-R has been shown to possess good construct (Derogatis & Cleary, 1977, Roskin & Dasberg, 1983) and discriminate (Roskin & Dasberg, 1983) validity.

Procedure

This study followed the specific procedures outlined in the task switching paradigms of Mayr and Keele (2000). Participants were required to identify the spatial location of a target item in a 2x2 matrix. On each trial the stimuli could vary according to (a) shape, (b) colour or (c) orientation. The participant was cued to the dimension that should be used to identify the target item by a word which appeared in the middle of the matrix prior to the presentation of the stimuli. For example, if the respondent was cued to "colour" then the top right quadrant would be the correct response for the slide shown in Figure 1. Alternatively, if the respondent was cued to "shape" then the top left quadrant would be the correct response.
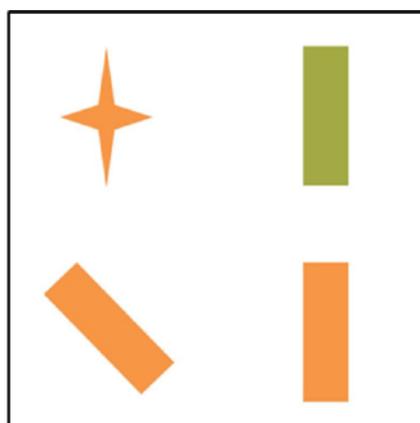

Figure 1: An example of the 2×2 stimulus matrix.

The sequencing of cues across trials allows for the separation of inhibition and switching components of working memory (see Figure 2).

Inhibition Trials are defined as those in which the cue is different from the cue in the immediately preceding trial (n-1), but the same as the cue two trials back (n-2; e.g., orientation-colour-orientation). Inhibitory trials require both set switching and n-2 inhibition. The sequence 'colour – shape – colour' requires that colour is the initial selected response. This then requires a set switch in order to respond to shape at the second trial and the inhibition of colour from the previous trial. On the third trial, response time is a function of switching from shape to colour and the continuing inhibition effect upon colour from the previous 'colour – shape' sequence. Accordingly, in the sequence 'colour – shape – colour', responding to the third component requires a set switch back to colour which may be impeded by its previous inhibition during the response to the second element of the sequence.

Trial Types	% of Total Trials
Trials Requiring a Set-Switch:	
Inhibitory (e.g., colour – shape – colour)	28.5%
Control (orientation – shape - colour)	28.25%
Unclassified (colour – colour – shape; orientation – orientation – colour)	20.0%
Trials Without a Set-Switch:	
Repeat Trials (e.g. colour – colour)	21.75%
Measures of Executive Function	
Set Inhibition Cost	
How Measured:	RT to Inhibitory Trials – RT to Control Trials
Relationship to Performance:	Larger Difference; Better Executive Ability
Set-Switching Cost	
How Measured:	Average of RT to Non-Inhibitory Trials – RT to Repeat Trials
Relationship to Performance:	Smaller Difference; Better Executive Ability

Figure 2. Trial Types and Measures of Executive Function. All calculations are based on reaction time.

Control Trials are defined as those trials in which the cue is different from the cue on the preceding two trials. This may take two forms; firstly, where all three cues are different (e.g., orientation – shape - colour); secondly, where the cue is different from a previously repeated trial (e.g., orientation – orientation - colour). Similar to Inhibition Trials, Control Trials require two set switches, but differ in that they do not require n-2 back inhibition.

Repeat Trials are defined as those trials that do not require a set switch (e.g. colour - colour or shape - shape).

Set Switching Cost is defined as the additional time it takes to respond to non-inhibitory trials that require the use of a different task set than used in the previous trial (e.g., change from colour to orientation as a task set) as compared with repeat trial, in which the same task sets are used on two trials (e.g. orientation followed by orientation).

Set Switching Cost is thought to reflect the time needed to reconfigure the cognitive processes involved in the representation of the to-be-used task set. Thus, if ruminators have switching difficulties, ruminative tendency should be associated with increased Set Switching Costs.

Inhibition Cost is defined as the extra time involved in switching back to a recently abandoned task set (e.g., orientation at the end of an orientation – colour - orientation sequence) compared with the cost of switching to a less recently abandoned task (e.g., orientation at the end of a shape –

colour - orientation sequence). This is considered a measure of set inhibition not confounded by switching abilities (as in both inhibition and control conditions the to-be-used cue had been preceded by at least two task switches).

When a respondent switches from one task to another, the first task set is thought to be inhibited to allow a faster and smoother transition to the second. Thus, if the participant returns to the inhibited task immediately afterward, it takes more time than switching to a less recently abandoned talk because of the extra time needed to overcome the inhibition of the prior task set's representation (Mayr & Keele, 2000). Hence, if ruminators cannot effectively inhibit prior task sets, they should have less inhibition to overcome than non-ruminators do and should therefore have smaller time costs (and therefore a faster response time) when reusing those representations.

Results

Overall Results

In order to identify outliers in the inhibition and set switch, a pairwise Euclidean distance matrix was calculated. The distances for each individual were summed and are presented in Appendix 1. As can be seen from these graphs, participants 11 and 47 showed marked dissimilarity to the rest of the participants (most probably the result of inappropriate test

taking behaviour) and were therefore eliminated from subsequent analyses. Additional data from 14 healthy control participants were removed from the analysis because they scored in the clinical range on one or more of the SCL-90-R subscales.

The final dataset consisted of 60 participants. Thirty participants suffered from no psychiatric distress (healthy controls) as measured by the SCL-90-R (Derogatis, 1994) and all of the mental health participants scored in the clinical range on the depression subscale of the SCL-90-R.

The average accuracy of the included participants' responses was 95.74% (SD=2.14, min = 89.5%, max = 95.74%). All inferential analyses were conduct only on runs for which the correct response was provided for each of the three trails within a sequence.

Differences between Depressed and Healthy Control Groups on the SCL-90-R and the Ruminative Response Scale

A comparison of the means between the healthy controls and the mental health group showed that the two groups had markedly different profiles. As can be seen in Table 1, the mental health group scored significantly higher in all of the subscales of the SCL-90-R.

Table 1

Differences in Psychopathology between Depressed and Healthy Control

Participants.

	Healthy Control Participants		Depressed Participants		*t*
	Mean	SD	Mean	SD	
Anxiety	46.17	8.09	66.93	11.48	-8.096*
Depression	51.07	7.75	71.4	9.28	-9.209*
Hostility	49.67	8.52	64.13	7.78	-6.866*
Interpersonal Sensitivity	49.50	8.89	68.03	9.55	-7.782*
Obsessive Compulsive	50.50	9.10	69.4	8.96	-8.105*
Paranoid Ideation	46.13	6.69	58.40	11.99	-4.892*
Phobic Anxiety	47.23	5.88	61.60	10.94	-6.336*
Psychoticism	48.03	6.62	61.77	10.75	-5.958*
Somatisation	47.87	8.43	65.87	10.41	-7.362*
Global Severity Index	49	7.30	68.57	9.03	-8.823*
Positive Symptom Total	48.90	8.81	67.4	6.55	-9.234*

* *p* < .001. *Note.* M = Mean. SD = Standard Deviation.

In addition, depressed participants scored significantly higher in the total score of the RRS as well as its subscales of depression and brooding, but not for the reflection subscale (see Table 2).

Table 2

Differences in Engagement and Quality of Rumination between Depressed and Healthy Control Participants.

	Depressed Participants		Healthy Control Participants		
	M	SD	M	SD	*t*-test
RRS Total	57.1	15.04	45.9	15.7	-2.82*
RRS Depression	32.13	8.28	25.5	8.07	-3.14*
RRS Brooding	13.47	4.04	10.3	3.68	-3.17*
RRS Reflection	11.08	3.85	10.1	4.67	-1.27

* $p < .0125$ (Bonferroni correction applied)

Note. M = Mean. SD = Standard Deviation.

Differences between Depressed and Healthy Control Groups between Trials

A 2 (Group: Healthy control vs. Depressed participants) by 4 (Trial Type: Inhibitions vs. Repeat vs. Unclassified vs. Control) repeat measures analysis of variance was calculated to assess reaction time across

conditions. A significant Mauchley Sphericity test was reported (W = 0.39; $X^2_5=53.54$; p< 0.01) therefore the Greenhouse-Geisser estimate of significance was employed in further analyses. A significant interaction effect for trial type by Group was observed ($F_{3,174}=14.44$; Epsilon = 0.60; Greenhouse-Geisser adjusted p < 0.01). This effect is presented in Figure 3.
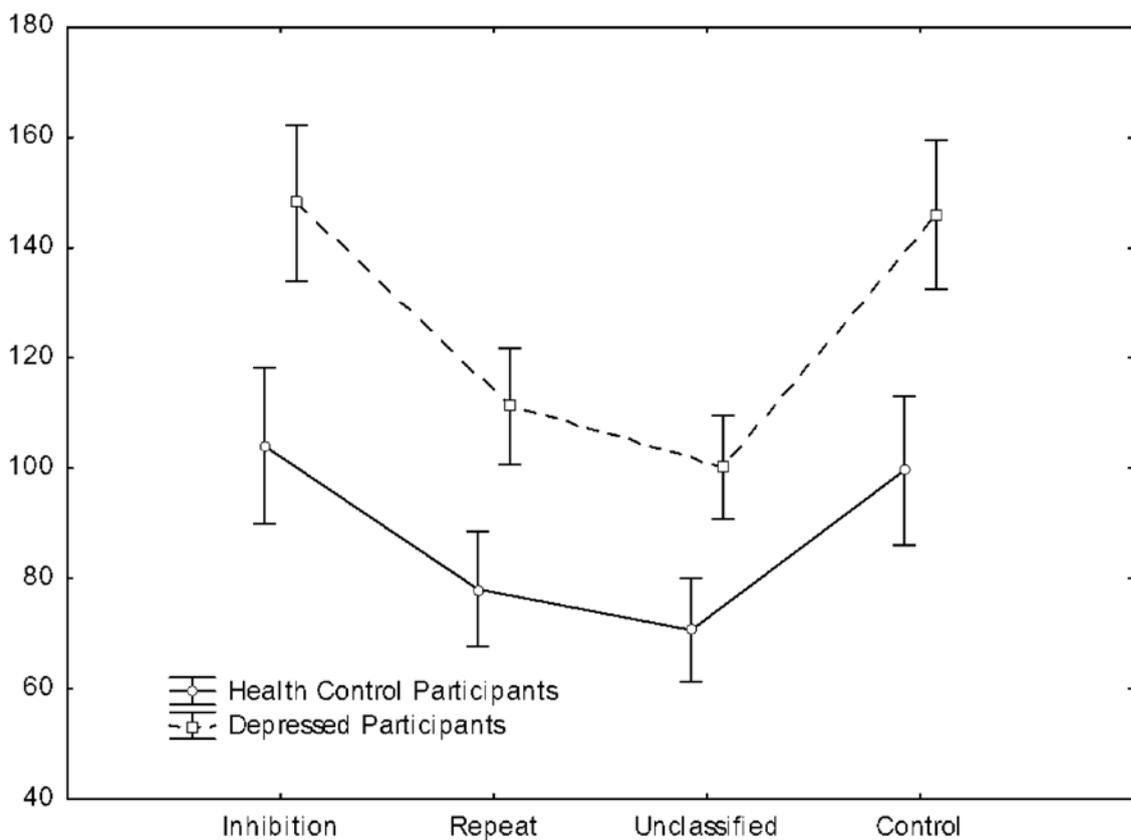

Figure 3: Differences between depressed and healthy control participants on inhibition, repeat, unclassified and control trials. The vertical axis represents reaction time in milliseconds, whereas the horizontal axis represents trial type.

In both the depressed and healthy control participants the control trials differed significantly from the repeat and unclassified trials but not from the inhibition trials (see Table 4, Appendix 2). For all trial types the depressed participants showed greater reaction times than did the healthy control participants: $t(58) = 104.01$, $p < .001$ for inhibition, $t(58) = 78.04$, $p < .01$ for repeat, $t(58) = 70.61$, $p < .05$ for unclassified, and $t(58) = 99.62$, $p < .001$.

Differences between Depressed and Non-Depressed Groups between Set Switching and Set Inhibition Cost

A mixed two-way ANOVA was calculated to assess differences in set switching and set inhibition reaction time between individuals with and without mental health difficulties. This analysis revealed a significant interaction effect (Wilks's Lambda = .918, $F(1, 58) = 5.19$, p = .026, partial eta squared =.082) between executive function (set inhibition vs. set switching cost) and mental health status (Healthy control vs. Participants with mental health difficulties). This statistically significant difference between the set inhibition and set-switching cost among participants who suffered from depression and those who did not suffer from any psychiatric illness is presented in Figure 4.
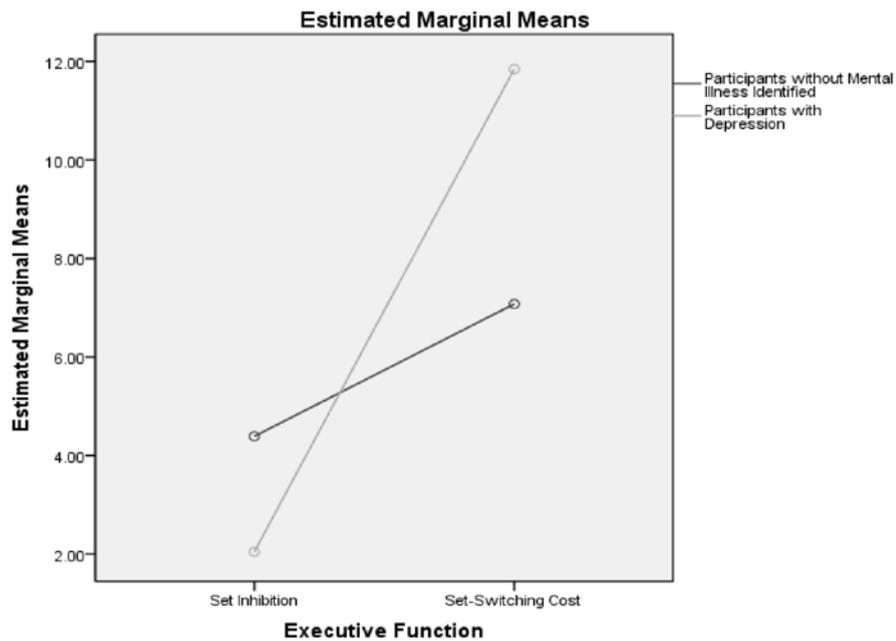
Figure 4. Estimated Marginal Means of participants with depression and participants without for each of the two executive functions. The vertical axis represents the average cost in milliseconds for each of set-inhibition and set-switching (horizontal axis). High set-inhibition cost and low set-switching cost reflect better the executive function.

Differences between Depressed and Non-Depressed Groups between Set Switching and Set Inhibition Cost: Post hoc analysis.

Depressed participants were found to be significantly slower ($t(44.48) = -2.37$, p = 0.02) when set switching ($M = 11.85$, $SD = 9.69$ vs. $M = 7.08$, $SD = 5.23$), but there was no significant difference ($t(58) = 1.13$, p = 0.26) in set inhibition between the two groups ($M = 2.05$, $SD = 7.91$ vs. $M = 4.39$, $SD = 8.14$). There was also a significant effect for the difference

between set inhibition and set switching in the control and mental health participants (t(58) = 2.27, p = 0.26) with the mental health group showing the greater disparity.

Correlation analysis of set switching cost, set inhibition and the depression sub-scale of SCL-90-R

There was no significant association between the depression sub-scale score of the SCL-90-R and set inhibition cost (r(58) = -0.1, p > .05) but there was a positive association between the depression sub-scale and the set switching cost (r(58) = 0.24, p < .04).

Table 3

Correlations Between Executive Control of Working Memory and Rumination

Measure	RRS Total	RRS Depression	RRS Brooding	RRS Reflection
Set Inhibition	-.12	-.16	-.14	.01
Set Switching	-.01	.004	.06	-.12

p > .05

Correlation analysis of set switching cost, set inhibition and RRS and its subscales.

RRS total score did not correlate significantly with either set inhibition or with set-switching cost. Similarly, neither of the RRS subscales for Depression, Brooding and Reflection correlated significantly with set inhibition and set-switching costs (Table 3).

Discussion

As anticipated, responses on the Ruminative Response Scale replicated previous findings and showed that individuals with depression experience more depressive rumination than those who do not experience distress from psychopathology symptoms. In particular, it replicated previous research (Burwell & Shirk, 2007; Lo, Ho & Hollon, 2008) indicating depressed individuals do not differ from healthy controls in their ability for reflective thinking, but that they significantly differ in how much they engage in depressiogenic rumination and brooding.

It is well established that depressed individuals exhibit a deficit in the control of their working memory when confronted with negative emotion valence material (Lyubomirsky & Nolen-Hoeksema, 1995; Nolen-Hoeksema, 2000; Watkins & Teasdale, 2001), suggesting a deficit in executive control of working memory. Given that the stimuli used in this

experiment were not emotionally valenced, our research provides further evidence of poor executive control of working memory in individuals who suffer from depression and suggests that the deficit in working memory exists irrespective of the emotional significance of materials in working memory. Specifically, depressed participants demonstrated difficulty in changing the content of their working memory rather than in forgetting. This finding is important, and provides a novel contribution to the existing literature, in that it demonstrates a general deficit of executive control of working memory that is independent of the specific content of the stimuli and provides support for Joormann and Gotlib's (2010) emphasis upon cognitive processes rather than the emotional significance of specific cognitive representations.

Previous research and theorists have proposed that a deficit in inhibitory control of working memory may be used to explain depressive rumination (Hester & Garavan, 2005; Linville, 1996; Ursin, 2005; Watkins & Brow, 2002; Witmer & Banich, 2007). These findings were largely based upon analogue studies using undergraduate students who were either selected on the basis of how much they ruminated or were subjected to mood induction procedures to manipulate the respondents' predilection to rumination. The association between such analogue studies of otherwise normally functioning individuals and those with clinical depression has not provided evidence of causality.

The results of the current study, which compares individuals with and without current mental health difficulties, does not support the emphasis on inhibitory control of working memory. Instead, the current study suggests that the deficits in executive control of working memory reflect problems of set-switching rather than set inhibition and may reflect general difficulties in cortical activation among depressed people. In an fMRI task that required depressed and non-depressed participants to complete an n-back working memory task, Harvey et al. (2005) notes a general "hypofrontality" in depressed participants that was associated with an impaired behavioural performance. In this study the depressed patients required greater activation within the lateral prefrontal cortex, anterior cingulate and parietal cortex to maintain a similar level of performance as controls during the working memory task.

Understandably, participants who had a diagnosis of depression also scored significantly higher on the depression sub-scale of the SCL-90-R when compared with healthy control participants. In addition, the analysis suggested the higher an individual scored on the depression sub-scale the more difficulty they had in set switching. However, there was no substantial association between the depressive sub-scale and set inhibition cost. This finding may provide evidence that the level of depression could be linked to the degree of deficit at the level of cognitive processing information already existing in working memory. A greater number of depressive symptoms may indicate a greater cognitive deficit.

A lack of association between the depression sub-scale and the RRS was unexpected, especially with regard to the brooding and the depression subscales of the RRS. This counter-intuitive discrepancy may be due to the self-report nature of both scales. Another reason for this discrepancy may be because a number of the participants recruited from mental health services were already in receipt of cognitive-behaviour therapy. It is possible that those participants were already engaging in problem-solving cognitive strategies which in turn counteracted the deleterious effect of depressive rumination causing this discrepancy.

A possible cognitive deficit in processing neutrally valenced information in working memory in people with a diagnosis of depression may be a characteristic symptom of their depressive illness rather than a deficit which exists among different diagnoses. Moritz, Hubner and Kluwe (2004), utilising a similar methodology with people who suffered from obsessive-compulsive disorder (OCD) concluded that researchers "should be prepared that executive impairments in OCD will not be found with neutral (i.e., concern irrelevant) material" (p. 681). In contrast to people with depression, there is evidence that verbal and non-verbal memory in people with OCD remains intact (Moritz, Kloss, Jahn, Schick & Hand, 2003) and this may also extend to executive function. Although the verdict from the research in executive function of individuals who suffer from different psychological disorders is still largely inconclusive, such differences provide support for disorder-unique deficits. Such differences

could prove helpful in defining the assessment, differential diagnosis and the development of targeted deficit-specific treatments.

This study also indicates that interventions targeting deficits in the executive control of working memory directly, such as cognitive remediation therapy may be beneficial for people suffering with depressive rumination. The predominant advantage of such an approach would be that it could be self-administered using a computer, either as a stand-alone intervention or as a homework task to be undertaken as part of another therapeutic approach. Furthermore, cognitive remediation tasks could be incorporated in computer games, which could make this intervention accessible to children, adolescents and individuals who would not engage in talking therapies or pharmaceutical treatments. It is acknowledged, however, that people who suffer from depressive rumination are also likely to experience reduce motivation and may only engage in interventions, such as cognitive remediation as part of a relapse prevention plan only.

A number of factors need to be taken into consideration in future research in this area. It is possible that depressed participants who have been engaging in treatment for a while have developed strategies that counterbalance their depressive ruminative predisposition. Further research in this area could take into account the number amount and type (e.g., medication, psychotherapy) of support each participant has had.

Furthermore, there is evidence that much of the subjective therapeutic progress takes place during the first few sessions (Duncan, Miller & Hubble, 1999). If self-reported questionnaires are administered during that time, responses may express individuals' hopes and expectations of therapy rather than their current symptomatology. In these cases self-report measures could be used in combination with other, more objective measures. Finally, a matched design with regard to age and gender could control for extraneous factors such as gender and dexterity accounted by age alone in between groups comparisons.

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PUBLIC DISSEMINATION DOCUMENT

Understanding and Treating Depressive Rumination

Public Dissemination Document

What was the study about?

This study investigated how people with depression use their short-term memory. People with depression tend to ruminate, that is, they go repeatedly over negative thoughts in their mind. Rumination takes place in short-term memory and can be counterproductive for mental health. However, depressed people and people prone to depression find it difficult to stop themselves from ruminating negative thoughts about themselves, others or the 'world' in general.

As rumination occurs in the short-term memory, it is necessary to alter what is occupying the short-term memory in order to forget about these thoughts and stop ruminating. One can change what is occupying the short-term memory either by emptying the contents of it ('intentionally forgetting') or by replacing the contents of it (switching to thinking about something else).

Previous research has shown that people with depression find it more difficult to stop themselves from ruminating negative thoughts than individuals without depression. However, it has not been investigated

whether people with depression also find it more difficult to stop themselves from thinking about neutral thoughts too.

If people with depression find it difficult to make changes to their short-term memory from neutral thoughts too, when compared with non-depressed people, then this may suggest that depressed people have a difficulty in controlling their thoughts and the contents of their short-term memory in general, and that this may be contributing to the tendency for negative rumination in depression.

How did the study investigate the way in which individuals use their short-term memory?

Our Study compared the responses from thirty individuals who had a diagnosis of depression and thirty individuals who did not have any mental health problems. All participants filled in a questionnaire to tell us how much they ruminated. In addition, they performed a computerised task which assessed their short-term memory.

In this task, participants were asked to pick the 'odd one out' of four shapes they were shown on the screen. They were told to pick the one that was different in terms of colour, shape or orientation, by pressing the relevant key on the keyboard. For example, the following figure shows one of the slides used. If the participant was asked to pick the odd one out in

terms of colour, then the participant would have to press the key corresponding to the object on the top right corner. If they were asked to pick the odd one out by shape, then this would be the object on the top left hand corner. The computer automatically recorded how long it took for each participant to make a correct selection.
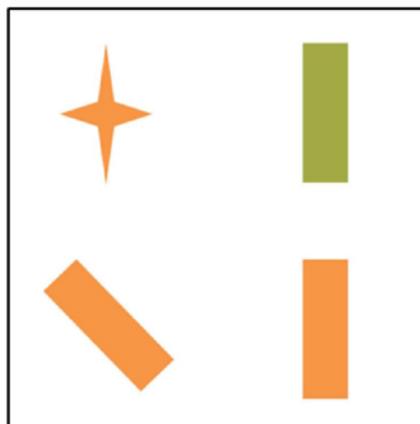

Figure 1. An example of the slide used for the computerised task.

All sixty participants had to complete the same sequence of 400 slides. We speculated that if depressed participants found it harder to empty their short-term memory, that is, they continued to ruminate on the previous slide, then they would find it harder to concentrate on making a choice on the next slide, and it would therefore take them longer to give a response.

Finally, the sequence of the slides was selected so that it would help us separate between the two different ways that we control the content of

our short-term memory: intentional forgetting and switching to another task.

What were the findings?

As anticipated, we found that individuals who suffer from depression also ruminated more than individuals without a diagnosis. In addition, we found that people with depression had difficulty switching to thinking about something else, but did not have difficulty in forgetting.

What are the implications of the findings?

These findings are important because they show a general difficulty in the ability of depressed individuals to control the contents of their short-term memory. Furthermore, this difficulty appears to be independent of whether the presented information is of an emotional nature or not. This finding is important because it suggests that there may be an underlying cognitive (thinking) problem in people who experience depression. These findings may help shape future interventions for rumination and depression.

What do these findings mean for the existing treatments of depressive rumination and depression?

To answer this question we will first describe the broad principles that current psychological treatments help individuals to ruminate less.

How do current psychological interventions work to help an individual ruminate less?

Our review of the literature identified two broad categories that encompass psychological interventions for depressed people who ruminate. Both categories included a number of distinctive therapeutic approaches and interventions.

The first category includes three therapeutic approaches: (a) cognitive therapy, (b) metacognitive therapy and (c) mindfulness based cognitive therapy. All three therapeutic approaches try to help the depressed individual re-evaluate some unhelpful beliefs that are common in individuals who experience depression. For example, cognitive therapy focuses upon helping the depressed individual to identify whether what they ruminate upon holds true or not. Metacognitive therapy and mindfulness-based cognitive therapy shift the focus of the intervention towards exploring the usefulness of the act of ruminating and help the individual move away from ruminating, either by challenging the belief that an individual has no control over their rumination or by promoting an

experiential and non-judgemental attitude towards ruminating. These interventions have been shown to reduce ruminating and improve low mood.

The second category includes a series of interventions that are based on the understanding of how people associate different experiences. Through continuous practice, depressed people have learnt that ruminating and feeling low go 'hand in hand'. In fact, they have learnt it so well, that starting to ruminate will generate low mood, similar to salivating when thinking about tasty food. Interventions in this category try to help the depressed individual engage in behaviours that have an opposite effect. An example of an intervention that comes under this category is distraction. If, a depressed individual distracts themselves from ruminating, by focusing on pleasant or neutral thoughts, then they will experience less or no low mood. Similarly, behavioural activation, another form of distraction, involves engaging in pleasurable or neutral physical activities instead of ruminating. Such interventions have been shown to reduce ruminating and improve depressed mood.

What does this research suggest?

Our research suggests that individuals who suffer from depression and who ruminate may have difficulty in their ability to control a cognitive function; that is, to manipulate the contents of their short-term memory

and ruminating is the result of this deficit. Furthermore, ruminating makes symptoms of depression worse.

Interventions aiming to help individuals develop more control over their short-term memory do exist and have been used successfully in treating other mental health conditions. However, there is no research on the application of these interventions in people who suffer with depression. We propose that future research needs to investigate the effectiveness of such interventions in people who suffer from rumination and depression.

Where can I get more information about this research?

This research is in the public domain and may be accessed by making a request to the Main Library at the University of Birmingham using the following contact details:

Main Library

University of Birmingham

Edgbaston

Birmingham

B15 2TT

Tel.: 0121 414 5828

Appendix 1

APPENDICES OF RESEARCH REPORT

Appendix 1

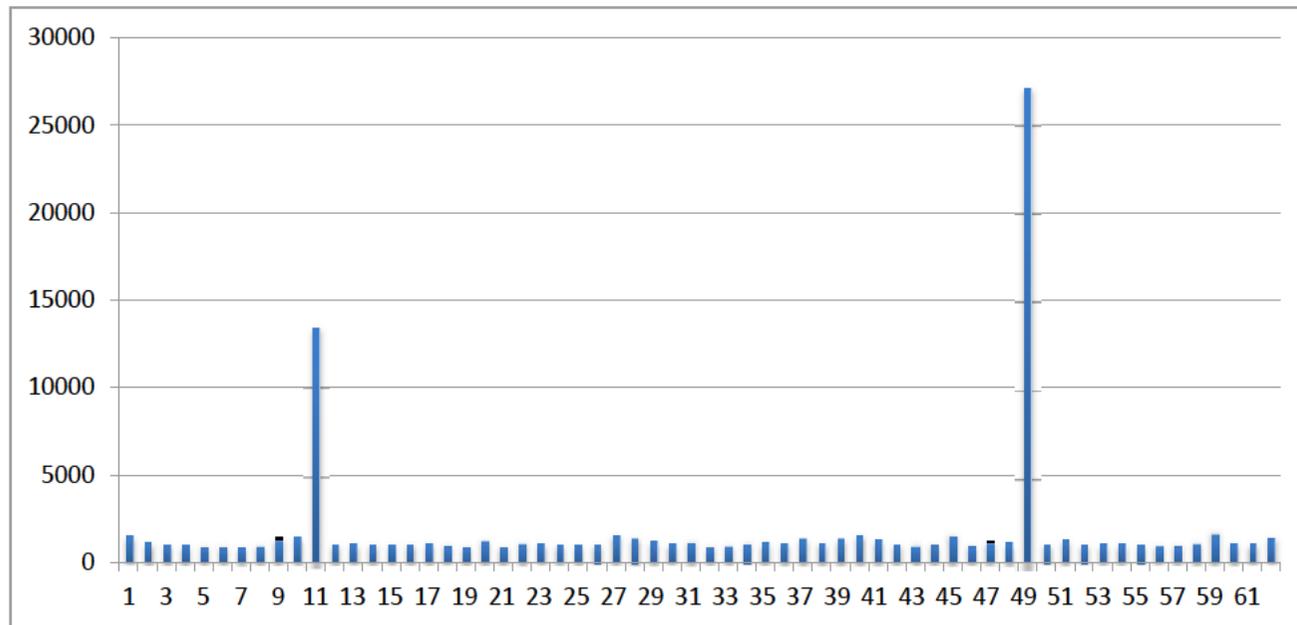Comparison between the pairwise Euclidean distance for each participant.

Figure 5. Sum of the Euclidian distance between all participants' Set Inhibition.
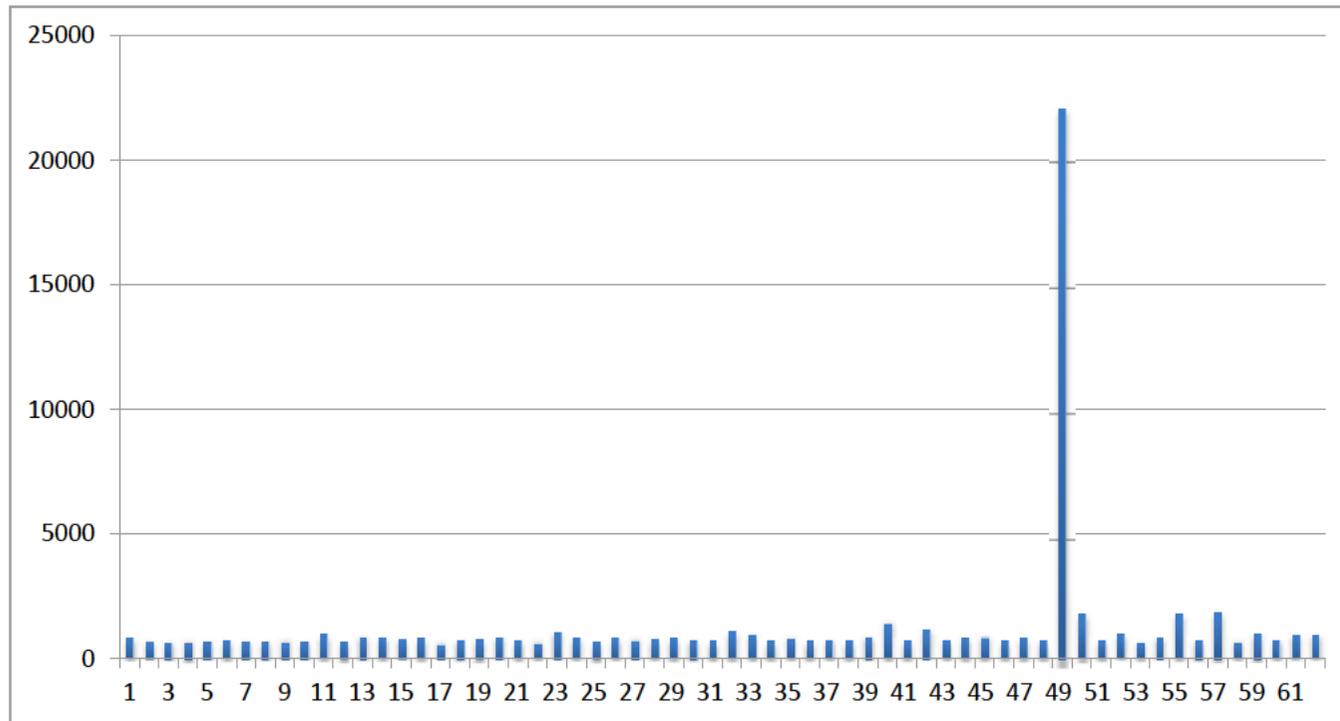
Figure 6. Sum of the Euclidian distance between all participants' Set Switching Cost.

Appendix 2

Table 4.

Post-hoc analyses between Groups and Trials.

Tukey HSD test: Approximate Probabilities for Post Hoc Tests Error: Between; Within; Pooled MSE = 1087.6, df = 63.999

			1	2	3	4	5	6	7	8
		t-test	**104.01**	**78.036**	**70.609**	**99.616**	**148.10**	**111.24**	**100.13**	**146.05**
1	Healthy Control	Inhibition		0.000032	0.000032	0.462118	0.000181	0.989318	0.999811	0.000265
2	Healthy Control	Repeat	0.000032		0.013896	0.000032	0.000127	0.005499	0.177271	0.000127
3	Healthy Control	Unclassified	0.000032	0.013896		0.000032	0.000127	0.000386	0.020168	0.000127

#	Group	Subtype								
4	Healthy Control	Control	0.462118	0.000032	0.000032		0.000134	0.869166	1.000000	0.000145
5	Depressed	Inhibition	0.000181	0.000127	0.000127	0.000134		0.000032	0.000032	0.981549
6	Depressed	Repeat	0.989318	0.005499	0.000386	0.869166	0.000032		0.000039	0.000032
7	Depressed	Unclassified	0.999811	0.177271	0.020168	1.000000	0.000032	0.000039		0.000032
8	Depressed	Control	0.000265	0.000127	0.000127	0.000145	0.981549	0.000032	0.000032	

Note: The *t* value for each comparison is reported in bold across the top row. The *p* value for each comparison is reported in its respective cell.

Appendix 2

MEASURES USED FOR RESEARCH REPORT

Rumination Scale

People think and do many different things when they feel depressed. Please read each of the items bellow and indicate whether you almost never, sometimes, often, or almost always think or do each one when your feel down, sad or depressed. Please indicate what you *generally* do, not what you think you should do.

1 almost never   2 sometimes   3 often   4 almost always

1. Think about how alone you feel.

2. Think "I won't be able to do my job if I don't snap out of this".

3. Think about your feelings of fatigue and achiness.

4. Think about how hard it is to concentrate.

5. Think "What am I doing to deserve this?"

6. Think about how passive and unmotivated you feel.

7. Analyse recent events to try to understand why you are depressed.

8. Think about how you don't seem to feel anything any more.

9. Think "Why can't I get going?".

10. Think "Why do I always react this way?"

11. Go away by yourself and think about why you feel like this.

12. Write down what you are thinking about and analyse it.

13. Think about a recent situation, wishing it had gone better.

14. Think "I won't be able to concentrate if I keep feeling this way".

15. Think "Why do I have problems other people do not have?"

16. Think "Why can't I handle things better?"

17. Think about how sad you feel.

18. Think about all your shortcomings, failings, faults and mistakes.

19. Think about how you don't feel up to doing anything.

20. Analyse your personality to try to understand why you are depressed.

21. Go someplace alone to think about your feelings.

22. Think about how angry you are with yourself.

Appendix 3

ETHICAL APPROVAL FOR RESEARCH

Appendix 4

PARTICIPANT'S INFORMATION SHEETS

Study Number: RG 11-140

Participant Identification Number for this trial:

INFORMATION ABOUT THE RESEARCH

Clinical Participants

(Version 1.1, 1st November 2011)

Title of Project: Cognitive Control of Working Memory in Depressed and Non-Depressed Individuals.

Name of Researcher: Nikolaos Zygouris

We would like to invite you to take part in our research study. Before you decide we would like you to understand why the research is being done and what it would involve for you. One of our team will go through the information sheet with you and answer any questions you have. We'd suggest this should take a few minutes.

Talk to others about the study if you wish.

Part 1 tells you the purpose of this study and what will happen to you if you take part.

Part 2 gives you more detailed information about the conduct of the study

Ask us if there is anything that is not clear.

PART 1 – Purpose of the study

1. *What is the purpose of the study?*

   Rumination is a way of responding to distress that involves repeatedly focusing on one's worries and on possible causes and consequences. Rumination is a crucial component of depression. When someone is ruminating, they occupy their memory by passively chewing over the same information time and time again; and sometimes they find it hard to stop. It is not surprising then, that rumination has been associated with difficulties in controlling what is in our memory. In fact, some researchers suggest that people ruminate *because they find it hard to ignore* what is on their mind when doing other activities. Since people with depression tend to ruminate more, this research attempts to find out if they also find it harder to stop ruminating when compared with people who are not depressed.

2. *Why have I been invited?*

   Your clinician has identified you as a candidate for this study
   because you are suffering from depression.

3. *Do I have to take part?*

   It is up to you to decide to join the study. We will describe the study
   and go through this information sheet. If you agree to take part, we
   will then ask you to sign a consent form. You are free to withdraw at
   any time, without giving a reason. This would not affect the
   standard of care you receive.

4. *What will happen to me if I take part?*

   If you decide to take part, we will contact you to arrange for a time
   and place to meet. We can meet at your home, the University of
   Birmingham or where you normally meet with your clinician (subject
   to room availability). We will try to accommodate the day and time
   to suit you. Participation to this study is separate to any treatment
   you may receive at present and treatment you may require in the
   future. Your treatment will remain unaffected no matter you
   decision.

5. *What if I incur expenses?*

If you are an NHS patient who makes a trip only for the purpose of this study (e.g. if we meet at the University of Birmingham), a total of £5 will be given to you to contribute towards your travel expenses.

6. *What will I have to do?*

First, we will ask you to complete two questionnaires. This will normally take about 15 minutes. Then, you will complete a computer-based task, where you will be shown some pictures while trying to remember some of the information we will give you. The computer-based task will take about 40 minutes.

7. *What are the possible disadvantages and risks of taking part?*

There are no short-term and long-term side-effects as a result of participating in this study.

8. *What are the possible benefits of taking part?*

We cannot promise the study will help you directly, but the information we get from this study could help improve the treatment of people with depression in the future.

9. *What if there is a problem?*

Any complaint about the way you have been dealt with during the study or any possible harm you might suffer will be addressed. The detailed information on this is given in Part 2.

10.   *Will my taking part in the study be kept confidential?*

Yes. We will follow ethical and legal practice and all information about you will be handled in confidence. The details are included in Part 2.

This completes Part 1. If the information has interested you and you are considering participation, please read the additional information in Part 2 before making a decision.

Part 2 – Conduct of the study

11.   *What if relevant new information becomes available?*

Sometimes we get new information about the issue being studied. If this new information suggests that the study may have an adverse effect on you, we will explain this to you before you start. At this point, we will either seek your consent again before continuing or terminate the study. Your safety is paramount.

12.   *What will happen if I don't want to carry on with the study?*

If you withdraw from the study we will destroy all identifiable information, but may need to use the data collected up to your withdrawal.

13. *What if there is a problem?*

If you have a concern about any aspect of this study, you should ask to speak to the researchers who will do their best to answer your questions on ⬜⬜⬜⬜⬜. If you remain unhappy and wish to complain formally, you can do this by contacting your local Patient Advisory Liaison Service (PALS) on ⬜⬜⬜⬜⬜.

In the event that something does go wrong and you are harmed during the research and this is due to someone's negligence then you may have grounds for a legal action for compensation against The University of Birmingham, but you may have to pay your legal costs. In addition, the normal University of Birmingham complaints mechanisms will still be available to you (if appropriate).

14. *Will my taking part in this study be kept confidential?*

All information which is collected about you during the course of the research will be kept strictly confidential, and any information about you which leaves the hospital/clinic will have your name and address removed so that you cannot be recognised.

15.   Will my GP be informed of my participation?

Your GP will normally <u>not</u> be informed of your participation. We may have to contact your GP if you voluntarily disclose information that clearly indicates that you are risk to yourself or someone else. We will let you know if we intend to contact your GP.

16.   What will happen to any information I give?

All data collected will be anonymised and coded before stored in an electronic format and analysed. All hardcopies will be kept in a locked cabinet. Data will be destroyed after approximately six years following the completion of the study. This amount of time is standard practice for studies reported in academic journals.

17.   What will happen to the results of the research study?

We intend to publish the anonymous results of this study to an academic journal. Similarly, a public dissemination document will also be created and you are welcome to receive copy. To give us your permission to send you a copy of the public dissemination document, please provide us with either your email address or your postal address in the space provided at the back of the consent form.

18.   *Who is organising and funding the research?*

The University of Birmingham organises and funds this study. This study is undertaken by the principal investigator towards partial fulfilment of the requirements of a doctoral course in Clinical Psychology at the University of Birmingham.

19.   *Who has reviewed this study?*

All research with participants from the NHS is looked at by independent group of people, called a Research Ethics Committee, to protect your interests. This study has been reviewed and given favourable opinion by the National Research Ethics Service Committee West Midlands – South Birmingham.

20.   *Further information and contact details:*

- For general information about clinically relevant research, please see:

  http://www.nhs.uk/Conditions/Clinical-trials/Pages/Introduction.aspx

- For specific information about this research project or to discuss any concerns, please contact either Nikos Zygouris (Primary Investigator) or Dr Christopher Jones (Academic Tutor) on ⬛⬛⬛⬛⬛⬛.

- If you feel that you need advice as to whether you should participate. Please contact your GP.

- If you are unhappy with the study please contact Nikos Zygouris or Dr Christopher Jones on ▆▆▆▆▆▆▆. If we cannot address your concerns, please contact your local Patient Advisory Liaison Service (PALS) on ▆▆▆▆▆▆▆. You can find more information about PALS at: http://www.pals.nhs.uk/

- If you find that this study raises any issues of concern to you, please discuss your concerns with the researcher, your doctor, or if you are a student control, you may also wish to seek advice from the University Counselling Service. Their telephone contact number is ▆▆▆▆▆▆▆.

Participant Identification Number for this trial:

INFORMATION ABOUT THE RESEARCH

Non-Clinical Participants

(Version 1.1, 1st November 2011)

Title of Project: Cognitive Control of Working Memory in Depressed and Non-Depressed Individuals.

Name of Researcher: Nikolaos Zygouris

We would like to invite you to take part in our research study. Before you decide we would like you to understand why the research is being done and what it would involve for you. One of our team will go through the information sheet with you and answer any questions you have. We'd suggest this should take a few minutes.

Talk to others about the study if you wish.

Part 1 tells you the purpose of this study and what will happen to you if you take part.

Part 2 gives you more detailed information about the conduct of the study

Ask us if there is anything that is not clear.

PART 1 – Purpose of the study

21. *What is the purpose of the study?*

    Rumination is a way of responding to distress that involves repeatedly focusing on one's worries and on possible causes and consequences. Rumination is a crucial component of depression. When someone is ruminating, they occupy their memory by passively chewing over the same information time and time again; and sometimes they find it hard to stop. It is not surprising then, that rumination has been associated with difficulties in controlling what is in our memory. In fact, some researchers suggest that people ruminate *because they find it hard to ignore* what is on their mind when doing other activities. Since people with depression tend to ruminate more, this research attempts to find out if they also find it harder to stop ruminating when compared with people who are not depressed.

22.   *Why have I been invited?*

If you do not receive support for any mental health problems, your are a potential candidate for this study.

23.   *Do I have to take part?*

It is up to you to decide to join the study. We will describe the study and go through this information sheet. If you agree to take part, we will then ask you to sign a consent form. You are free to withdraw at any time, without giving a reason.

24.   *What will happen to me if I take part?*

If you decide to take part, we will contact you to arrange for a time and place to meet. We will normally meet at the University of Birmingham. We will try to accommodate the day and time to suit you.

25.   *What if I incur expenses?*

There is no provision for payment of any expenses you incur, but you will be accredited RPS credits if you sign up through that scheme.

26.   *What will I have to do?*

First, we will ask you to complete two questionnaires. This will normally take about 15 minutes. Then, you will complete a computer-based task, where you will be shown some pictures while trying to remember some of the information we will give you. The computer-based task will take about 40 minutes.

27.   *What are the possible disadvantages and risks of taking part?*
There are no short-term and long-term side-effects as a result of participating in this study.

28.   *What are the possible benefits of taking part?*
We cannot promise the study will help you directly, but the information we get from this study could help improve the treatment of people with depression in the future.

29.   *What if there is a problem?*
Any complaint about the way you have been dealt with during the study or any possible harm you might suffer will be addressed. The detailed information on this is given in Part 2.

30.   *Will my taking part in the study be kept confidential?*

Yes. We will follow ethical and legal practice and all information about you will be handled in confidence. The details are included in Part 2.

This completes Part 1. If the information has interested you and you are considering participation, please read the additional information in Part 2 before making a decision.

Part 2 – Conduct of the study

31.   *What if relevant new information becomes available?*

Sometimes we get new information about the issue being studied. If this new information suggests that the study may have an adverse effect on you, we will explain this to you before you start. At this point, we will either seek your consent again before continuing or terminate the study. Your safety is paramount.

32.   *What will happen if I don't want to carry on with the study?*

If you withdraw from the study we will destroy all identifiable information, but may need to use the data collected up to your withdrawal.

33.   *What if there is a problem?*

If you have a concern about any aspect of this study, you should ask to speak to the researchers who will do their best to answer your questions on ▮▮▮▮▮▮▮▮. If you remain unhappy and wish to complain formally, you can do this by contacting the Director of the Clinical Psychology Doctorate by writing to Dr Jan Oyebode at ▮▮▮▮▮▮▮▮▮▮

In the event that something does go wrong and you are harmed during the research and this is due to someone's negligence then

you may have grounds for a legal action for compensation against The University of Birmingham, but you may have to pay your legal costs. In addition, the normal University of Birmingham complaints mechanisms will still be available to you (if appropriate).

34.   *Will my taking part in this study be kept confidential?*

All information which is collected about you during the course of the research will be kept strictly confidential.

35.   *Will my GP be informed of my participation?*

Your GP will normally <u>not</u> be informed of your participation.

36.   *What will happen to any information I give?*

All data collected will be anonymised and coded before stored in an electronic format and analysed. All hardcopies will be kept in a locked cabinet. Data will be destroyed after approximately six years following the completion of the study. This amount of time is standard practice for studies reported in academic journals.

37.   *What will happen to the results of the research study?*

We intend to publish the anonymous results of this study to an academic journal. Similarly, a public dissemination document will also be created and you are welcome to receive copy. To give us

your permission to send you a copy of the public dissemination document, please provide us with either your email address or your postal address in the space provided at the back of the consent form.

38.   *Who is organising and funding the research?*

The University of Birmingham organises and funds this study. This study is undertaken by the principal investigator towards partial fulfilment of the requirements of a doctoral course in Clinical Psychology at the University of Birmingham.

39.   *Who has reviewed this study?*

All research with participants from the NHS is looked at by independent group of people, called a Research Ethics Committee, to protect your interests. This study has been reviewed and given favourable opinion by the National Research Ethics Service Committee West Midlands – South Birmingham.

40.   *Further information and contact details:*

- For general information about clinically relevant research, please see:

  http://www.nhs.uk/Conditions/Clinical-

  trials/Pages/Introduction.aspx

- For specific information about this research project or to discuss any concerns, please contact either Nikos Zygouris (Primary Investigator) or Dr Christopher Jones (Academic Tutor) on ▮▮▮▮▮▮▮▮

- If you feel that you need advice as to whether you should participate. Please contact your GP.

- If you are unhappy with the study please contact Nikos Zygouris or Dr Christopher Jones on ▮▮▮▮▮▮▮. If we cannot address your concerns, please contact Dr Jan Oyebode (Director of the Clinical Psychology Doctorate) at ▮▮▮▮▮▮▮▮▮.

- If you find that this study raises any issues of concern to you, please discuss your concerns with the researcher, your doctor, or if you are a student control, you may also wish to seek advice from the University Counselling Service. Their telephone contact number is ▮▮▮▮▮▮▮.

Appendix 5

'INSTRUCTION FOR AUTHORS' FOR NOMINATED JOURNAL

AMERICAN PSYCHOLOGICAL SOCIETY'S 'EMOTION'