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UNIVERSITY OF MIAMI

COGNITIVE MECHANISMS OF ANGER REGULATION: THE ROLE OF EXECUTIVE FUNCTION

BY

Katherine G. Denny

A DISSERTATION

Submitted to the Faculty
of the University of Miami
in partial fulfillment of the requirements for
the degree of Doctor of Philosophy

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COGNITIVE MECHANISMS OF ANGER REGULATION: THE ROLE OF EXECUTIVE FUNCTION

Katherine G. Denny

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Anger is a prevalent and powerful emotion, arising when another person has thwarted a self-relevant goal or event, and increased effort is required for goal achievement. While the majority of research indicates that anger is an affective precursor to aggression, recent research implies that anger may also have beneficial consequences – at least for some people in some situations. With the negative cost of anger often being severe, and yet the potential that anger utilization can lead to enhanced performance, it is important to understand individual differences in the ability to regulate and utilize anger. To explore this area of research, this study applied the current understanding of the connection between executive functions (EF) and emotion regulation (ER) to an examination of anger and anger regulation. Cognitive ER (e.g., reappraisal) is an effective way to regulate emotions such as anger, and has been examined at the level of daily functioning. It has been demonstrated that general cognitive control is related to ER, however only minimal research has examined the role of specific aspects of EF on ER, and this has not been examined specifically in the context of anger regulation. In light of this lack of information regarding the role of EFs in anger regulation, undergraduate participants (n=101) completed behavioral and physiological measures to

examine the influence of distinct components of EF (Inhibition and Shifting) on the ability to regulate induced anger and perform on a frustrating visual motor task.

My results begin to establish a connection between EF and anger regulation. This relationship is complex but it is identified through three main findings, 1) biased attention (vigilance) to anger can affect ER; improving it under some circumstances, diminishing it in others, 2) a subset of Shifting and Inhibition results support the existence of a role of cognitive ER strategies in anger regulation, 3) EF with happy stimuli is associated with lower self-report anger, but worse performance on a frustrating task. Overall, these findings indicate a potentially important role of EF in emotion regulation, but highlight the complexity of this relationship. They provide a refined focus for future studies. Furthermore, this study has important implications for identifying neural components that promote successful utilization, as well as for understanding neurological deficits associated with an inability to regulate anger, a common sequela of traumatic brain injury (TBI). With this information I hope to increase our ability to identify, prevent, and treat the detrimental emotional disturbances caused by TBI in military personnel and athletes.

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Chapter 1. Introduction

Anger is a strong and important emotion which, when left unchecked, can lead to aggression and detrimental outcomes. However, when anger is appropriately regulated, it can help individuals harness their motivation to overcome an obstacle standing between them and an important personal goal. The key component here is the effective regulation of anger. In this study, I will focus on top-down cognitive regulation of anger through executive function (EF) and working memory.

Anger regulation through EF is essential for daily wellbeing in non-clinical populations, but it may be particularly significant in populations of military personnel and athletes participating in contact sports. These populations are required to perform complex tasks in an environment that promotes increased anger. Additionally, they are at an elevated risk for traumatic brain injury (TBI), which has been shown to disrupt the physical structures and pathways that aid top-down regulation of anger. This damage may be evidenced by the increased irritability and aggression, and decreased emotional control that is associated with brain injury.

This project focuses on understanding the role of EFs in anger regulation in a healthy undergraduate population, and the research is designed to provide insight into anger and its regulation in the overall healthy population. However this study is also intended as a first step toward an understanding of TBIs in military personnel and athletes, in particular of their ability to regulate anger for the sake of sustained performance, and of possible neural mechanisms that are disrupted through TBI. I hope to increase our ability to identify, prevent, and treat the detrimental emotional disturbances caused by TBI in these populations.

Anger

Emotions such as anger are typically conceptualized as self-relevant, multi-faceted reactions that include changes in subjective experience, behavior, and central and peripheral responding (Gross & Thompson, 2007; Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). Emotions are considered to be responses to our perceptions of relevant events or stimuli, and are thought to play an important role in preparing a behavioral response, facilitating interpersonal interactions, and increasing memory for relevant events (Gross & Thompson, 2007). Emotions are generated in the subcortical limbic system (i.e., amygdala, hippocampus; Williams, Suchy, & Rau, 2009).

Anger is a prevalent and easily recognizable emotion. Most people report feeling angry from several times a week to several times a day (Averill, 1982). Anger has a strong ability to capture attention; angry facial expressions are recognized quickly and accurately, and angry individuals are usually perceived as competent, dominant, and threatening (Lerner & Tiedens, 2006).

From an appraisal theory perspective on emotions, anger is characterized as a negatively valenced emotion, which typically arises when a self-relevant goal has been thwarted by another person and is often associated with a sense of confidence that, through increased effort, one will be able to attain his or her goal (Lerner & Tiedens, 2006). It has also been suggested that anger can occur without these prerequisite appraisals; increased facial expression of anger has been demonstrated with physical restraint in infants (Stenberg, Campos, & Emde, 1983) and uncomfortable temperatures have been shown to lead to increased self-reports of anger (Anderson, Deuser, & DeNeve, 1995). Anger has been shown to narrow one's focus of attention, causing one to

take in less information, which directly contrasts with positive emotions, which broaden one's intake of information (Fredrickson & Branigan, 2005). Anger is associated with changes in both central (Carver & Harmon-Jones, 2009; Cox & Harrison, 2008) and peripheral physiology (e.g., cardiovascular reactivity; Cox & Harrison, 2008; Stemmler, Heldmann, Pauls, & Scherer, 2001). Together, these affective, cognitive, and physiological changes ready an individual to change the situation, remove the problem, and re-establish the circumstances that existed prior to the negative event (Lerner & Tiedens, 2006).

While anger plays a beneficial role in preparing one for action, it can also lead to detrimental consequences. Anger is an affective precursor to aggression, it is a core feature of many externalizing disorders (e.g., oppositional defiant disorder; American Psychiatric Association, 2000), and it may increase one's risk for developing cardiovascular disease (Brosschot & Thayer, 1998). Thus, whether anger is beneficial or detrimental depends on how it is regulated.

Anger may prove useful in situations which require increased motivation or the confrontation of an obstacle. In this light, anger has been linked to the behavioral approach system, a motivational system that is predominantly associated with positive emotions (Carver & Harmon-Jones, 2009). This notion is supported by research using a variety of methodological approaches including self-report measures of affect (e.g., PANAS; Harmon-Jones, Harmon-Jones, Abramson, & Peterson, 2009) and motivation orientation (BIS/BAS, Study 1 and 2, Carver, 2004), as well as measurement of neural activation (Harmon-Jones, 2007). These findings are in line with the idea that anger is associated with increased effort to achieve a goal, and they highlight functional aspects of

anger. These findings are complemented by recent research taking a functional approach to ER, which indicates that individuals may aim to increase useful emotions regardless of valence. Research taking this utilization approach has shown that in certain situations individuals will choose to enhance negative emotion and forego positive emotion if they believe that this will help them achieve a long term goal (Tamir, 2005). This is in contrast to the hedonic approach where the goal of ER is to increase positive, and decrease negative, affect. Research supporting these ideas has shown that when anticipating a confrontational task, individuals preferred activities that would increase their level of anger, and anger actually improved performance in the confrontational task (Tamir, Mitchell, & Gross, 2008). It has also been demonstrated that highly resilient individuals utilize the motivational implications of anger to persevere on a frustrating task (Denny & Siemer, 2011).

Anecdotally, this slight up-regulation of anger may be utilized by, and helpful for, some athletes as they prepare for an important game. Prior to big events, many athletes can be found listening to aggressive music and getting themselves focused and energized for competition. Even during events, certain athletes (e.g., tennis player John McEnroe) would purposefully get angry (e.g., at the referee or their opponent). In watching McEnroe compete, it was noticeable that anger would elevate his game and he would perform better in this highly affective state.

Thus, while the majority of traditional research indicates that anger may be harmful, more recent research implies that anger may also have beneficial consequences – at least for some people in some situations. The negative cost of anger is often severe, but the potential exists for anger utilization to lead to enhanced performance. In light of

this tradeoff, my overarching research question is: What distinguishes individuals who are able to utilize and regulate anger successfully from those who cannot? I investigated this question by assessing three specific aims. First, I examined the relation between individual differences in EF and the ability to down regulate affective and physiological aspects of anger during a frustrating task. Second, I assessed whether these differences in EF are associated with performance on a frustrating task when angry. Third, I investigated the influence of physiological responding to anger and anger regulation on performance on a frustrating task.

To begin to address these objectives, I will review the current understanding of cognitive ER and EF in the examination of anger regulation.

Cognitive emotion regulation

Emotion regulation is a set of processes that allow for the altering of emotional experience or expression (Gross & Thompson, 2007). ER strategies can include avoiding situations that are likely to cause negative emotions (situation selection) or taking steps to make the situation less emotional (situation modification). If one is unable to avoid a particular situation all together, one can purposefully select what he or she is paying attention to (attentional deployment) such as choosing to ignore emotional aspects of a situation and instead attending to more pleasant thoughts or stimuli (e.g., distraction).

A related means of ER is affect-biased attention, or vigilance, towards particular types of stimuli (Todd, Cunningham, Anderson, & Thompson, 2012). Affect-biased attention refers to the selective attention processes that modify attention to favor certain categories of emotionally relevant stimuli (Todd et al., 2012). This biased attention primes the visual system so that it privileges incoming information based on experience

of what is motivationally relevant in a particular context, which creates a predisposition to attend to certain categories over others. When affect biased attention becomes habitual, it may modulate emotional responses to stressful events. For example, if one is pre-tuned to see more happy faces relative to angry faces in a crowd, one may be less likely to experience feelings of negative affect and heightened physiological arousal in a stressful situation than if one's attention is habitually biased toward negative expressions (Todd et al, 2012). It could also be argued that being selectively tuned in to happy faces means that one may be more disrupted and negatively affected when forced to deal with stressful situations. The terms affect-biased attention and vigilance will be used interchangeably throughout this paper.

Individuals can also regulate their emotions by changing how they view the situation (cognitive change), or altering the meaning of a situation so that it is more aligned with the emotions that they would like to experience or that they find more beneficial (cognitive reappraisal). Reappraisal is the cognitive reinterpretation of the meaning or self-relevance of an event, which subsequently alters its emotional significance (Gross & Thompson, 2007; Ray et al., 2008). It alters the evaluation of an emotional stimulus early in the emotion generative process, so that the emotional stimulus may have a different impact (e.g. viewing an interview as a chance to show one's strengths rather than an opportunity to fail). This regulation strategy recruits cognitive control processes and can involve strategies such as viewing a situation from a different perspective (Goldin, McRae, Ramel, & Gross, 2008; Ray et al., 2008). Rumination is a thought process associated with an inability to effectively cognitively reappraise a situation. Rumination involves attending to and "recycling" negative

thoughts, and has been shown to increase or maintain a sad or angry mood (Ray, Wilhelm, & Gross, 2008). An additional method of ER, suppression of expression, is implemented following the emotion onset with the goal of hiding facial expressions associated with an emotion.

Cognitive ER strategies (e.g., reappraisal) are often generated in our frontal lobes. Through strong neural connections to limbic brain regions, the frontal lobes exert top-down control over emotions and thus provide the basis for cognitive ER. Reappraisal is associated with increased activity in cognitive control prefrontal cortex regions and decreased limbic system response (Goldin et al., 2008). Given its reliance on cognitive control, reappraisal is particularly relevant for this study.

Ample research has demonstrated that cognitive reappraisal is an effective form of ER. Cognitive reappraisal has been shown to decrease the subjective experience of emotions without substantial physiological costs (Mauss, Cook, Cheng, & Gross, 2007). This strategy is associated with fewer symptoms of depression, increased positive emotion, better interpersonal functioning, and more adaptive peripheral physiological responding (Ray et al., 2008). Regular use of reappraisal has been associated with increased ability to regulate emotions, enhanced interpersonal functioning, and physical and psychological well-being (Goldin et al., 2008). For example, following an anger induction, individuals who report regular and successful use of reappraisal indicate less anger and negative emotions and more positive emotions (Mauss et al., 2007).

Reappraisal is linked with the effective recruitment of cognitive control processes, which in turn promotes efficient ER (Goldin et al., 2008). In contrast, rumination is associated with poor cognitive control (e.g., poor inhibition of negative thoughts) and is

characterized by the repetitive thinking of negative events or stimuli. Rumination is associated with reduced EF; in particular, reduced inhibition of negative material has been associated with greater rumination in a depressed population (Joormann & Gotlib, 2010). Particularly relevant for this study, rumination about angry events may lead to increased anger. Given this information, deficits in the ability to cognitively regulate anger may result in increased anger rumination and, thus, in increased anger.

The role of anger regulation is unquestionably important in healthy populations in everyday life. The importance of anger regulation is made even more potent when examining clinical populations that present with a decreased ability to regulate anger, as demonstrated by increased irritability and aggression. These are both common symptoms of traumatic brain injury (TBI). TBI is becoming increasingly prevalent in the general population, and is particularly relevant in military personnel and athletes competing in high level contact sports, two populations that regularly put themselves in situations where they are at an increased risk for TBI. TBI research indicates that structural and axonal damage caused by TBI is associated with cognitive and neuropsychological deficits such as poor cognitive regulation of prepotent emotional and behavioral responses such as anger and aggression (Williams et al., 2009). Examining ER in populations with deficits in top down control further heightens the awareness of the critical role of efficient ER

Research has examined the role of cognitive control in ER and the impact it has on daily functioning (e.g., interpersonal functioning, Gross & John, 2003), and recent research has examined areas of neural activation during reappraisal (Goldin et al., 2008). It is thought that EFs (described below) provide the basic building blocks for cognitive

control and top down self-regulation, however very little work has studied cognitive control at this more refined level of individual EFs and how it relates to specific ER strategies such as reappraisal (see Gyurak et al., 2011 for an exception).

Executive Function

EFs are defined as basic cognitive control mechanisms that contribute to more complex cognitive functioning and thus help regulate dynamic human cognition (Miyake et al., 2000). They are considered to be volitional and effortful (Williams et al., 2009) and provide the building blocks for more complex cognitive tasks (Miyake et al., 2000). EFs help to override prepotent and automatic responses. This control makes it possible for an individual to engage in goal-directed and future-oriented behaviors such as generating goals, modifying behaviors based on situational demands, and executing actions necessary for goal achievement (Williams et al., 2009). EFs are centrally located in the frontal lobes and maintain tight connections throughout the brain. Established patterns of connectivity indicate that the frontal lobes may organize behavior but rely heavily on other regions (e.g., the limbic system) for input and successful functioning (Jurado & Rosselli, 2007). The limbic system is particularly relevant for this study because of its prominent role in emotional processing (Williams et al., 2009). The connection between the frontal lobes and the limbic system, and thus the integration of cognitive control and emotional reactivity, plays an important role in the generation of informed solutions and adaptive behavior (Williams et al., 2009).

These brain regions (frontal lobes and limbic system), and their role in the top down regulation of emotion, are of particular clinical relevance for individuals who have suffered from TBI (Hartikainen et al., 2010). The frontal lobes and the white matter tracts

connecting frontal lobes and the subcortical limbic system are regions at high risk for damage by TBI (Mayer et al., 2010). Damage to these areas is often associated with the symptoms of poor ER (e.g., irritability, aggression) following head injury (Miller, 1994).

Despite the basic understanding of where EFs are generally located, there is some debate about which factors should be considered an EF. This debate includes the question of whether working memory (WM) should be considered an EF, or whether EFs are instead a component of working memory. The view I adopt for this study is described in the next section.

In a well-known conceptualization of WM, Baddeley and colleagues (Baddeley, 1992) have endorsed a three part system of WM with two so called slave systems for storing visual and phonological information (which have been conceptualized as the working memory capacity (WMC) component of WM) and a central executive that operates as a gating system, delegating which information can be allowed into the storage systems. In a more recent review, Baddeley (2003) emphasized the fractionation between the central executive and the two storage systems, creating a clear separation between central executive and storage systems while still associating them as parts of working memory.

The central executive is the most complex component of WM. Based on Baddeley's three part system, Miyake and colleagues (2000) sought to refine our understanding of the central executive. They examined the organization of EFs and identified three independent aspects of EFs that share an underlying commonality: (1) shifting of mental sets, (2) inhibition of prepotent responses and (3) updating of WM (Miyake et al., 2000). The components of EF that I examine in this study (shifting of

mental sets and inhibition of prepotent responses, see below) can be viewed as specific components of the central executive, and therefore as components of WM separate from the storage systems. By focusing on these components, I can explore the underlying mechanisms of cognitive control.

Based on the fact that they have been previously implicated as being involved in ER (De Lissnyder, Koster, Derakshan, & De Raedt, 2010; Denny & Siemer, 2011), I have elected to focus on two EF components: shifting of mental sets and inhibition of prepotent responses.

The shifting of mental sets, or **shifting**, is based on the ability to shift back and forth between mental sets or operations. The ability to shift requires the disengagement of a previous set which is no longer relevant, and an active engagement to a new, relevant set (Miyake et al., 2000). An example of a task that requires shifting is the neuropsychological test, Trails Making - B (Arbuthnott & Frank, 2000). In this task, participants are asked to connect circles that contain either a number (from 1 to 13) or a letter (from A to L) in alternating ascending order (e.g., 1 – A – 2 – B – 3) until they reach the number 13. This requires that they shift back and forth from counting by numbers to moving to the next letter of the alphabet.

The inhibition of prepotent responses, or **inhibition**, involves the purposeful inhibition of a prepotent or automatic response when necessary (Miyake et al., 2000). The Stroop task is a measure of inhibition (Miyake et al., 2000). In this task, participants are asked to read out loud the color of ink in which a color word is printed. For example, the correct response for the word RED printed in green ink is green. This requires that

participants inhibit the tendency to produce a more dominant or automatic response (i.e., to read the word).

Many studies have focused on these areas of EF because they are lower level functions that can be defined in a fairly precise and circumscribed manner (Huizinga, Dolan, & van der Molen, 2006), and they are likely to be involved in the performance of more complex EF tasks (Miyake et al., 2000).

These EFs also contribute to performance on complex working memory span tasks designed from the perspective of the three part system of WM (Baddeley, 1992; Baddeley, 2003). Based on this relationship and its relevance in previous studies of ER (Schmeichel, Volokhov, & Demaree, 2008; Schmeichel & Demaree, 2010), I will include in this study a combined measure of storage capacity and EF measure (automated Ospan; Unsworth, Heitz, Schrock, & Engle, 2005). The operation span task was designed to measure information storage (WMC) while simultaneously processing additional information (requiring EF), thus tapping both the storage and processing aspects of WM (Conway et al., 2005). The operation span task combines the presentation of to-be-remembered stimuli (letters) and a secondary task requiring participants to verify the validity of a basic mathematic equation (2 x 4 - 3). The score is quantified in two ways. One, it is quantified by the sum of all perfectly recalled sets, and two, by the total number of letters recalled in the correct position (Unsworth et al., 2005). WM span tasks have been shown to predict complex cognitive behaviors including reasoning and problem solving. Complex WM tasks are contrasted with simple WM tasks (e.g., digit span) where, for example, participants are asked to repeat a sequence of digits. Digit span tests assess storage capacity (WMC) but not EF.

Despite substantial research examining the role of cognitive control in ER at a level of daily functioning, there has been minimal research looking at individual differences in more detailed cognitive control, as measured by EFs, and how these differences influence the ability to regulate emotions. There has been even less research examining specific EFs associated with the regulation of anger. Therefore, this study will focus on the role of EFs (shifting, inhibition) as building blocks for cognitive ER.

In the next section I will discuss the literature supporting the association between EF, WM, and ER**,** and will then apply these relationships to understanding the role of EF in anger regulation.

Executive Function and Emotion Regulation

There is limited research that has explored the relationship between cognitive control and ER. The majority of this research has not focused on EFs in particular, but rather on working memory, either through complex WMC tasks, which incorporate EF (OSpan: Jha, Stanley, Kiyonaga, Wong, & Gelfand, 2010; Schmeichel & Demaree, 2010; Schmeichel et al., 2008), or through simple WMC tasks, which measure pure capacity (digit span; Gyurak et al., 2009; Gyurak, Goodkind, Kramer, Miller, & Levenson, 2011). In particular, research has focused on how emotions influence WMC. Therefore, I will explain WMC research before transitioning to the more limited body of work on EF (Gotlib & Joormann, 2010; Gyurak et al., 2009, 2011; Johnson, 2009).

Working memory capacity. In general, WMC studies have shown that negative emotions and related experiences (e.g., stress) have a detrimental effect on WMC. For example, one study showed that anxiety and stress reduced complex WMC in a military cohort during predeployment training (Jha et al., 2010).

There is less research examining how WMC influences ER. One set of studies demonstrated that in a college age sample, higher complex WMC was associated with more successful down regulation of affective facial expressions and better reappraisal of negative stimuli as indicated by experiencing less negative emotions (Schmeichel et al., 2008). A follow up to this study (Schmeichel & Demaree, 2010) was conducted to examine a possible alternative hypothesis for the relationship between WMC and ER: that higher WMC was associated with better ER due to the ability to adhere to instructions. Results supported the initial hypotheses, further confirming that individuals with higher WMC more effectively engaged in spontaneous ER after receiving negative feedback. Schmeichel and colleagues interpreted these results to mean that higher WMC is directly associated with an enhanced ability to spontaneously regulate emotions.

WMC is highlighted in the cognitive control and depression literature in that having a larger WMC, and having the most relevant information taking up spots in limited capacity of working memory, is beneficial. First, a larger WMC allows an individual to keep more information in the front of his or her mind. This is helpful for successful ER because it helps individuals to, for example, recognize how they are feeling, know that they want to change the way they are feeling, and to employ efficient means of ER (e.g., to reappraise a situation in order to view a different perspective to reduce negative affect). Second, having the most up to date information in WM can help an individual to 'live in the moment' and move on from negative situations. For example, if a person experiences a negative event (e.g., receives a bad grade on an exam), he or she can keep this information in WM beyond the point where it is relevant (e.g., still thinking about the bad grade while going to the movies with friends maintains a negative mood) or

they can update what they are thinking about to reflect their current situation and thus change their mood (e.g., enjoy spending time with friends and be happy). Thus, WM and WMC are important for ER because they help an individual to keep a relevant goal in mind and disengage from irrelevant information, thus promoting successful ER. This may be particularly relevant for the onset and maintenance of negative mood in depression (Gotlib & Joormann, 2010).

Executive function. Research with clinical populations has also shown that deficits in WMC and EFs (in particular, deficits in inhibition) may play an integral role in the onset and maintenance of mood disorders (Gotlib & Joormann, 2010). Depression is characterized by sustained negative mood, which can occur for a number of reasons (e.g., constantly paying attention to negative stimuli in the environment; interpreting neutral stimuli as negative). Gotlib and Joormann (2010) have reviewed the role of cognitive deficits in the ability to disengage from negative thoughts and have found that EFs, in particular inhibition, are in charge of disengaging from, and inhibiting access of, irrelevant material that may be associated with problems in letting go of negative material (Gotlib & Joormann, 2010). Thus a decreased ability to inhibit irrelevant information may be a key component to the dysregulation of negative emotions in people suffering from depression.

Recent research on the role of executive functions in ER has used a variety of cognitive tasks. One set of studies used a task shifting paradigm with emotional and neutral stimuli to measure the ability to control attention in an affective context (Johnson, 2009). This study found that individuals with less effective emotional attention control showed greater difficulty in disengaging from emotional stimuli and engaging with the

neutral stimuli than in shifting from neutral to emotional cues. In particular, individuals higher in trait anxiety (characterized by a decreased ability to manage the intrusive worrisome thoughts that promote high levels of anxiety) showed lower emotional attention control than less anxious individuals as demonstrated by increased costs of shifting away from emotional stimuli. A second study linked emotional attentional control to effective ER (Johnson, 2009). Results suggested that increased costs of shifting away from emotional stimuli (indicating difficulty disengaging with emotional—and engaging with neutral—stimuli) were associated with increased frustration during a challenging anagram task (Johnson, 2009). These findings provide indirect support for the role of set-shifting to affective stimuli in ER.

Another set of studies distinguished between different aspects of EFs (inhibition, task shifting, cognitive flexibility) and their relationship to the ability to regulate negative emotions (Gyurak et al., 2009; Gyurak et al., 2011). These studies examined a wide range of EF abilities by studying participants with neurodegenerative disorders (e.g., frontotemporal lobar degeneration, Alzheimer's disease) and healthy controls. Cognitive flexibility (as measured by verbal fluency) was related to better down- (Gyurak et al., 2009) and up- (Gyurak et al., 2011) regulation of emotions. No other measures of EF were associated with ability to regulate emotion. Verbal fluency was measured by the individual's ability to generate a list of words or objects that fit a specified criterion (i.e., starts with the letter F) which has implications for ER because of the need to remember rules of appropriate emotion expression and behavior and to efficiently carry out these actions.

While the studies conducted by Gyurak and colleagues diverge from our current definition of EF by examining more complex cognitive aspects as measured by neuropsychological assessment, the inclusion of these findings is still integral to the current study. Along with studies by Gotlib and Joormann (2010) and Johnson (2009) which assessed specific aspects of EF, Gyurak and colleagues have conducted one of few studies that distinguish between different aspects of EF and how they contribute to ER. By identifying specific measures of EF as well as neuropsychological measures associated with ER, these studies help us to focus our attention on specific EFs.

Together, these independent lines of research begin to establish a relationship between specific aspects of EF and ER; however, this relationship has yet to be examined with anger regulation.

Executive Function and Anger Regulation

While no one has explicitly examined the relationship between EF and anger *regulation*, limited research has examined the relationship between EF and anger, which provides indirect evidence for this study. For example, Wilkowski and Robinson (2007) have shown that individuals low (versus high) in trait anger demonstrate increased recruitment of cognitive control resources when faced with hostile stimuli. Inasmuch as low trait anger is the result of habitual successful anger regulation, this finding provides evidence that cognitive control is important for controlling anger. Another set of studies examined the relationship between cognitive control and aggression, a behavioral outcome of anger (Denson, Pedersen, Friese, Hahm, & Roberts, 2011). Denson and colleagues found that, after provocation, participants showed decreased self-control on an unpleasant task. Furthermore, they found that provocation led to increased anger

rumination, which depleted cognitive control capacity, which in turn led to increased aggression (Denson et al., 2011). I interpret their findings this way: Cognitive control is involved, and depleted, in the control of anger and aggression. Two studies examining outcomes associated with different forms of ER (reappraisal and rumination) found that following provocation, participants instructed to reappraise (which relied on effective cognitive control) showed less subjective anger, cognitive perseveration, and sympathetic nervous system activation than participants instructed to ruminate about the situation (Ray et al., 2008). These results indicate that the cognitive representation of an event influences the emotional experience when holding relatively constant the duration and subject of a person's thoughts.

Together, these studies highlighted the importance of cognitive control for anger regulation, however they did not assess independent aspects of EF, nor did they directly examine the regulation of anger. Therefore, I aim to examine the role of two specific aspects of EF (set shifting and inhibition) in anger regulation, as well as how these aspects are related to performance. Mental set shifting is thought to be crucial for successful cognitive ER because processes such as reappraisal involve the reinterpretation of the meaning of a situation. Likewise, the inhibition of a prepotent response is an important component of anger regulation because the inhibition of behavioral impulses associated with anger is a crucial aspect of anger regulation. In addition to set shifting and inhibition, I will include a measure of WMC, a more complex (and ambiguous) measure of cognitive control that has been previously associated with ER (e.g., Schmeichel, Volokhov, & Demaree, 2008). Based on this information, this study assessed the EF profile associated with the ability to maintain or down regulate

anger during a frustrating task as measured by self-report affect and physiological measures.

Measurement of Emotion Regulation

The effectiveness of ER has been assessed using a combination of methods, including self-report measures and psychophysiological and neurological activity assessments. Studies using self-report measures usually assess self-reported affect prior to and following regulation; a change in self-reported mood following regulation is an indicator of successful regulation (Aim 1, see below). These measures rely on an awareness of, and an ability to accurately report, one's experienced emotions. Self-report measures are potentially susceptible to bias and demand effects. For this reason, studies have supplemented self-report with biological measures.

Numerous studies have identified physiological patterns that are associated with emotional responding and ER. From an evolutionary perspective, physiological changes associated with emotions contribute to an individual's ability to efficiently carry out necessary actions (e.g., increased arousal leads to the enhanced ability to run from the bear). Heart Rate (HR) and variation in HR (heart rate variability, HRV) are useful physiological markers of emotional responding and regulation.

HR is a marker of sympathetic activation. In response to a stressor, HR increases, peaks, and then decreases during recovery from the stressor. This pattern allows for HR to be used as an additional indicator of emotion experience and regulation. Studies examining cardiac response to stressors have consistently indicated that HR increases in response to provocation and returns to baseline levels upon the removal of anger-inducing stimuli (Herrald & Tomaka, 2002; Mauss et al., 2007; Vögele, Sorg, Studtmann,

& Weber, 2010). Influence of different ER strategies on HR have yielded mixed results; some reports indicate a significant decrease in HR in reappraisal compared to suppression conditions (Denson et al., 2011), while others have found no relation between HR and the use of different regulation strategies (Mauss et al., 2007).

In addition to HR itself, heart rate variability (HRV) is a biological indicator of emotional responding and regulation. HRV measures beat-to-beat variation in heart-beat intervals. This study will focus on HRV in the high frequency band of respiration (0.12 - 0.4 Hz) which is called Respiratory Sinus Arrhythmia (RSA). RSA is a marker of the flexibility of the interplay between the two divisions of the autonomic nervous system, the sympathetic and parasympathetic nervous systems, which both receive vagal nerve influence. The parasympathetic nervous system (PNS) is associated with the maintenance of low level arousal (Denson et al., 2011). During safe situations, increased exertion of the vagal nerve (or the "vagal brake") increases parasympathetic influence and thereby decreases HR, which allows for increased ability to attend to one's environment. The vagal brake is associated with rest and recovery. Influence of the vagal brake leads to acute increases in RSA (Gyurak & Ayduk, 2008). High frequency RSA, as measured in this study, is thus thought to mostly reflect PNS influence. In contrast, the sympathetic nervous system (SNS) is associated with increased arousal in response to stress. During challenging situations, the vagal brake is withdrawn, which leads to increased influence by the SNS, which in turn prepares one's body to effectively respond to a stressor through fight or flight. Withdrawal of the vagal brake is associated with acute decreases in RSA (Gyurak & Ayduk, 2008). As a result of the interplay between these two autonomic systems, resting RSA is a measure of variability at rest and is considered to be

a marker of chronic efficiency and flexibility of the central-peripheral feedback system (Gyurak & Ayduk, 2008). Changes in RSA can also reflect event- or action-based changes in the interplay between PNS and SNS. Change in RSA is measured by the difference between resting RSA and RSA during an interaction or engagement with one's environment (e.g., regulating one's emotions). This study focused on changes in RSA associated with anger regulation.

Our HR naturally speeds up on inhalation and slows down on exhalation, and respiration rate and depth through the breathing cycle influence HRV. Therefore it is imperative to take respiration into account in order to achieve a measure of HRV that is more purely associated with differences in vagal tone and thus adaptive emotional responding (Butler, Wilhelm, & Gross, 2006). When measuring resting RSA, it is possible to physically limit variability in respiration across participants by inducing paced breathing (e.g., Butler, Wilhelm, & Gross, 2006). During paced breathing, participants are instructed to breath in rhythm with the rise and fall of a tone. This approach allows the researchers to consider HR, respiration rate, and respiration volume under controlled conditions. Results from Butler et al. (2006) demonstrated that higher resting RSA, above and beyond respiration rate and volume, was associated with more emotional reactivity during a negative conversation which represents efficient responding to one's situation (Butler, Wilhelm, & Gross, 2006). This finding highlights the importance of accounting for respiration when measuring RSA. The paced breathing procedure is less feasible when measuring changes in RSA in response to a stressor, however. Therefore, in the present study, we will continuously measure respiration rate throughout the study and

control the influence of respiration rate on HRV statistically (e.g., Rottenberg, Wilhelm, Gross, & Gotlib, 2003).

High resting RSA is associated with efficient responding to environmental changes compared to low RSA, which is associated with poor ER (Denson, Grisham, Moulds, 2011). It is thought that high resting RSA is a marker of one's capacity to flexibly and efficiently regulate affect and behavior, and may protect against difficulties controlling negative affect (Gyurak & Ayduk, 2008). Resting RSA is associated with the down regulation of negative emotional responding under high stress conditions (more so than low to moderate levels of stress), and it may also be related to the ability to up regulate or experience strong emotions when necessary (Gyurak & Ayduk, 2008).

In line with the association between high RSA and effective ER, studies examining change in RSA have found that increased RSA is associated with the adaptive strategy of reappraisal more so than the less adaptive strategy of suppression (Denson et al., 2011). One study had participants view a disturbing film and then discuss the film with another participant while reappraising, suppressing, or responding as they normally would. Participants in the reappraisal condition showed the largest increase in RSA followed by participants in the suppression condition (Butler, Wilhelm, & Gross, 2006).

Another connection between reappraisal and high RSA is their mutual association with increased frontal lobe activation and cognitive control (Denson et al., 2011). Reappraisal calls on cognitive resources early in the emotional process, before the emotion is fully formed, which requires fewer overall self-regulatory resources. It has also been demonstrated that high RSA is associated with increased prefrontal cortical activity (dorsolateral prefrontal cortex), better EF (e.g., working memory, sustained

attention; Hansen, Johnsen, & Thayer, 2003), and enhanced prefrontal regulation of emotion. This highlights the association between cognitive control and the regulation of emotional and physiological arousal.

Additionally, a few studies have examined RSA in the context of anger regulation. One study examined the relationship of RSA as an index of ER, and the perception of other-blame as a way of inducing anger (León, Hernández, Rodríguez, & Vila, 2009). This study found that a high level of resting RSA was associated with lower other-blame after a highly intentional interpersonal transgression. The authors interpret these findings to indicate that RSA influenced cognitive regulation of emotional responding (León et al., 2009). A similar study examined the effects of instructed reappraisal or suppression of RSA in response to anger provocation. Participants were instructed to watch a controversial video, and were assigned to reappraise, suppress, or view normally (Denson, Grisham, & Moulds, 2011). Results indicated that RSA change was greater in the reappraisal condition versus control, and that RSA change in the suppression condition was not significantly different from the reappraisal or control conditions. Denson et al. (2011) find their results to be consistent with the idea that RSA is associated with ER. These findings highlight the importance of RSA in representing cognitive regulation of emotion.

In light of the discussion above, I used increase in RSA and decrease in HR as indicators of effective anger regulation (Aim 1). In addition to using physiological responding to anger as a dependent variable representing regulation, I also used these measures as independent variables to predict performance on a visual motor task (Aim 3, see below).

Emotion regulation to maintain performance

ER is essential for the smooth management of daily ups and downs, but in light of the instrumental approach to ER, emotions can also be regulated in order to put individuals in a position where they can perform to the best of their abilities. Being able to achieve an ideal level of affective and physiological activation is important for achieving optimal performance on tasks requiring physiological activation as well as fine motor skills and concentration. This can be described most tangibly in athletic contexts. In these environments, it is often essential to be physically activated and motivated but still calm enough to carry out fine motor skills (e.g. hitting a baseball). Announcers of athletic events regularly talk about individual athletes "getting into their zone", "getting fired up", or "taking a deep breath" before shooting a crucial free throw. Coaches or teammates may also encourage a player to "brush it off" or to "get their head back in the game" after making a mistake. These are all ways in which athletes regulate emotions in order to perform to the best of their abilities. The nature of athletics is also well designed to encourage anger; an opposing individual is an obstacle to a goal, and increased motivation and persistence can help one reach this goal. With this in mind, it is imperative to successfully regulate anger and its strong physiological component in order to perform the skilled actions required to successfully reach one's goal. An examination of the EF components that contribute to the regulation of the affective and physiological components of anger is important for performance and identification of which individuals are able to utilize anger. In light of these ideas, this study examined cognitive (Aim 2, see below) and physiological (Aim 3) predictors of performance on a frustrating fine motor task.

Aims and Hypotheses

Aim 1: The first aim of this study is to investigate the effects of individual differences in inhibition and set shifting on the ability to down regulate anger during a frustrating task. To do so, I examined the roles of shifting and inhibition on the ability to regulate anger as indicated by 1) lower self-report negative affect, and 2) increased RSA and decreased HR during a frustrating task. Based on the fact that the existing literature highlights the importance of EF in ER, I hypothesize that better abilities in both inhibition and set shifting will be associated with better anger regulation. Given the minimal research on specific EF and ER, I will not state formal hypotheses regarding the differential contribution of the two aspects of EF to anger regulation.

Aim 2: The second aim of this study is to assess individual differences in inhibition and set shifting associated with maintained or improved performance on a frustrating task when angry. To answer this question, I examined the effects of distinct aspects of EF (inhibition, shifting) on performance as measured by accuracy and perseverance. Again, given the paucity of literature in this area, I will not state differential hypotheses but predict that enhanced abilities in each area of EF will be associated with better physiological regulation and maintained performance.

Aim 3: My final aim in this study is to examine the influence of physiological responding to anger and anger regulation on performance of a frustrating task. I hypothesize that increased RSA and decreased HR during a frustrating task will predict better performance as measured by accuracy and perseverance.

This study examined the role of individual differences in specific measures of EF (inhibition and set shifting) on the ability to regulate affective and physiological aspects

of anger and to perform on a fine motor task when angry. This study also examined regulation of physiological activation as a predictor of performance on a fine motor task. While this study examined these questions in an undergraduate population that is not characterized by marked head trauma, it provides a logical first step towards recognizing and treating individuals who have experienced TBI.

Chapter 2. Methods

Overview

This study was completed in two sessions. When participants signed up for the study they were directed to a link where they provided waived consent and completed the online survey. Following completion of this survey, participants came into the laboratory for the behavioral component of the study.

After undergoing the informed consent procedure, undergraduate participants were attached to electrodes for psychophysiological measurement and seated in front of the computer. Following a brief resting period (5 minutes) that allowed them to become acquainted with the physiological sensors, they completed tasks to measure EF (Inhibition and Shifting) and complex WMC. They then completed the first trial of a behavioral distress tolerance measure, which required them to perform a fine motor task while receiving frustrating negative feedback; this provided a baseline measure for this task. After watching a neutral video to restore baseline levels of mood, they underwent an angry mood induction that included writing about a recent angry memory while listening to angry music. They were then given instructions to regulate any strong emotions that they were experiencing. While in an angry mood, they completed a second trial of the distress tolerance task. Lastly, they completed a self-report measure of regulation strategy and success. Peripheral physiology (HRV, HR, respiration) was recorded throughout the session; self-report mood was assessed at various points.

Participants.

Participants were 101 undergraduates (60 females, 41 males) at the University of Miami who participated for partial course credit. Mean age was 18.88 and ranged from

17-22. Racial breakdown of this sample was as follows: Causasian (53), Hispanic (19), Asian (13), African American (4), Middle Eastern (2), Other (2), and 8 people did not complete the online demographics form where race was indicated.

Measures

Shifting and inhibition. These EFs were assessed by an affective task shifting paradigm (De Lissnyder et al., 2010; Mayr & Keele, 2000). This task was designed to separate two different components of task switching: Shifting and Inhibition. In this task, subjects were asked identify the spatial location of a deviant object. Each stimulus display presented four face stimuli arranged in a 2 x 2 matrix. The stimuli could vary by emotional expression, background color, or gender. Immediately before the stimulus presentation, a centrally presented cue identified which dimension to use to identify the location of the deviant stimulus.

In the study by De Lissnyder et al. (2010), faces were chosen as stimuli material instead of words or objects because they provided ecologically valid interpersonal stimuli and have been used in studies of depression and rumination (Joormann, Dkane, & Gotlib, 2006; Raes, Hermans, & Williams, 2006). Stimuli were chosen from the NimStim Face Stimulus Set (Tottenham et al., 2009). This stimulus set is racially diverse, and includes facial expressions from Asian-American, African-American, European-American, and Latino-American actors (Tottenham et al., 2009). A total of 12 angry and 12 happy faces were selected; closed mouth expressions were used. In a validation study, angry faces (Mean proportion correct = 0.84, SD = 0.17) and happy faces (Mean proportion correct = 0.92, SD = 0.07) were recognized with good validity. The same person was used for both

angry and happy facial expressions. Images were adjusted to the same size (180 x 232 pixels).

Angry faces were selected to investigate whether individual differences in response to angry stimuli were particularly critical in predicting one's ability to regulate anger. This assumption is in line with recent findings that have shown that it is specifically the recruitment of executive control resources in response to anger stimuli that predicts individual differences in trait anger (Wilkowski & Robinson 2007, 2008) and aggression (Denny & Siemer, 2011). Happy faces were chosen to contrast angry faces because they share similar motivational implications; both emotions have been linked to the motivational approach system (Carver & Harmon-Jones, 2009). Affective stimuli contribute to an emotional context that is important for this study; however, the investigation of affect-specific effects is exploratory.

In this task, the cue word (emotion, gender, color) was presented for 500 ms and signaled the task-relevant feature for the stimulus grid that was presented. Subsequently, four faces were presented in a 2 x 2 grid. Each face differed on three dimensions: emotional facial expression (angry or happy), gender (male or female), and background color (dark grey or light grey). Participants were instructed to identify the face that differs from the others as fast and as accurately as possible. The stimuli remained on the computer screen until the participant responded. A response was registered by pressing a key on the keyboard that corresponded to the appropriate stimulus location. The four keys were arranged in the same spatial pattern as the 2 x 2 layout. Following a response, a black screen was presented for 100 ms before the cue for the next trial appeared.

Each trial type consisted of a sequence of two or three cued dimensions/trials. The order of the cued dimensions was pseudo-random depending on the trial type. A trial type was determined by the relationship of the final cued dimension and the one or two preceding trials. This task was composed of four trial types:

1. *Control.* Control trials were defined as trials in which the rules in both of the two preceding trials differed from the current trial (e.g., gender, emotion, background color). These trials were contrasted with inhibitory trials to obtain a measure of inhibition and with repeat trials to compute a measure of Shifting.

2. *Inhibitory.* Inhibitory trials were defined as trials where the present rule differed from the rule in the preceding trial (n-1), but was identical to the rule in the trial before the preceding trial (n-2; e.g., gender, emotion, gender). Inhibition and control trials were both shift trials. The important difference is that inhibition trials required the subject to shift to a rule that had to be inhibited in the previous trial. When a subject shifted from one rule to another, the previous rule or mental set must be inhibited to allow for a shift to the present set. If this inhibited set needed to be re-activated in the next trial, it would take additional time to overcome this inhibition. Thus, the extra time that it took to re-activate the rule reflected the degree to which the rule was inhibited in the previous trial.

3. *Unclassified*. Unclassified trials consisted of a repeat and then a shift trial (e.g., gender – gender – facial expression). These trials were combined with control trials to calculate a measure of set shifting costs.

4. *Repeat*. Repeat trials occurred when the same task set was used on consecutive trials (e.g., emotion – emotion) and provided a measure of reaction time when shifting was not necessary.

Reaction time (RT) differences were used to obtain measures for Inhibition and Shifting based on comparisons between specific trial types. Shifting costs were defined as the additional time that it took to respond to a trial in which the rule changed (e.g., change from facial expression to gender) compared to repetition trials in which the rule did not change from the previous trial (e.g., gender to gender). Shifting costs were calculated by the difference between the mean RT of control and unclassified trials and RT to repeat trials [(RT to control + RT to unclassified)/2 – RT to repeat trials]. Shifting costs reflect the time needed to shift to a new rule; a smaller difference indicates better executive ability. Therefore, difficulties with set shifting imply increased set shifting costs.

The ability to inhibit an irrelevant rule or response set was measured by the difference in RTs between inhibitory and control trials [RT to inhibitory trials – RT to control trials]. High inhibition scores reflect good executive ability.

This task consists of four blocks. Per block, each of the first three trial types occurred equally often (24 per type) and 36 trials were repeat trials. All calculations were based on the responses to the last trial in the sequence. The cued dimension (facial expression, gender, color) of the last trial was presented equally often.

This task was initially used to examine whether individuals with subclinical depression or depressive rumination showed associated deficits in inhibition and set shifting to affective stimuli (De Lissnyder et al., 2010). Results from this study indicated that set-shifting impairments were present only in moderately to severely depressed participants. Results also showed that rumination was related to impaired set shifting without valence-specific impairment (valence refers to affect of stimuli, angry or happy),

and to inhibition impairments specifically when processing negative stimuli (De Lissnyder et al., 2010). These results indicated that this task is capable of assessing the relationship between differences in EF to affective stimuli and the ability to regulate negative information.

Working memory. Complex WM, which tapped into both the storage and processing aspects of WM, was assessed by the automated Operation Span task (Aospan; Unsworth et al., 2005). In this task, a short list of items to be remembered were presented, interspersed with an unrelated processing task requiring a quick response. The to-be-remembered items were 3 to 7 capitalized letters from a pool of 12 letters, which were presented individually for 800 ms each. In the memory portion of this task, all 12 letters were shown, and subjects indicated in order which letters were presented. This part of the task was untimed. The processing portion of this task required that participants verify a simple mathematic equation involving a multiplication or division and then addition or subtraction. The processing stimuli were presented for an adjusted amount of time based on individual performance during practice trials. Participants were encouraged to keep their math accuracy at or above 85% at all times. The WM score was measured by the total number of items, across trials, which were recalled in their correct order.

Mirror Tracing Persistence Task. The Mirror Tracing Persistence Task (MTPT) requires that a participant use the computer mouse to trace the outline of a star displayed on the computer screen (Daughters et al., 2005). The marker, indicating the location of the mouse on the outline of the star, moves in the opposite direction of the mouse and requires precise and continuous movement. If the marker goes off the line or stops for too long, the participant hears a harsh noise and the marker goes back to the beginning. The

task ends after five minutes or when the participant presses Enter. Outcome variables include total time, total distance, maximum distance, number of errors, and errors per second.

Mood induction procedure. The mood induction procedure was a combined music and autobiographical recall procedure (e.g., Eich, Ng, Macaulay, Percy, & Grebneva, 2007; Siemer, 2001). For the angry autobiographical recall procedure, participants were asked to remember a time in the past month in which they had experienced intense anger and write about the event in as much detail as possible (Bodenhausen, Sheppard, & Kramer, 1994; Rusting & Nolen-Hoeksema, 1998). Throughout the autobiographical mood induction, each participant listened to angry music, an interpretation of heavy metal songs by a cello quartet (Apocalyptica, 1998; Siemer, 2005) through closed headphones.

Peripheral Physiology. A MindWare Bionex data acquisition system was used to record Electrocardiograph (ECG) and Respiration Frequency (RF). Physiological data was collected continuously from the start of the baseline period prior to the tests of EF through the end of the recovery period. Three disposable ECG electrodes (93-0100) were attached in a lead II configuration with ECG minus on upper right collarbone, ECG positive on lower left rib, and ground on the lower right rib. Respiration frequency was measured using a respiration effort transducer belt (50-4504) worn around the chest above the rib-cage. ECG and respiratory signals were acquired using Mindware's Biolab data acquisition software, sampled at 1,000 Hz, and digitized with a 16-bit analog-to-digital converter. High Frequency Respiration Sinus Arrhythmia (HF-RSA), a measure of heart rate variability in the high frequency band of respiration between 0.12 -0.4 Hz, was utilized as a marker of parasympathetic influence via the vagal nerve. HF-RSA was

calculated using MindWare HRV3.0.18 software using the following procedure. Beat-to-beat interval series were obtained from the ECG and converted into a time series of instantaneous beat-to-beat intervals with a resolution of 4 Hz. Spectral analysis was used to determine the power spectral density in the frequency band between 0.12 and 0.4 Hz. This value was then log-transformed to provide an index of HF-RSA. Prior to calculating HF-RSA, R-wave markers in the ECG signal were visually inspected for artifacts. The MAD/MED artifact detection (Berntson, Quigley, Jang, & Boysen, 1990) algorithm in MindWare software (MindWare Heart Rate Variability Application, Version 3.0.18, Mindware Technologies Ltd., Gahanna, OH) was utilized to correct artifacts. Current guidelines for calculating HF-RSA were followed (Task Force of The European Society of Cardiology and The North American Society of Pacing and Electrophysiology, 1996). RSA and HR values were calculated for one minute segments throughout the baseline (5 min) and MTPT (5 min) periods of the study.

Demographics. Participants completed a questionnaire assessing demographic characteristics, including age, gender, race and ethnicity, education background, income, occupation, marital status, and children.

Health Questionnaire. Participants provided information regarding current medical conditions (particularly diabetes, heart disease, hypertension, and health conditions affecting the central nervous system), current medication use (particularly selective serotonin reuptake inhibitors, tricyclic antidepressants, antipsychotic drugs, antihistamines, and beta-blockers), caffeine consumption, recent exercise, and sleep, which have been shown in prior studies to affect RSA levels (see review by Rottenberg,

2007). Participants also indicated number of previous concussions (with or without loss of consciousness).

Affect Ratings. Affect ratings were assessed at several points prior to and subsequent the mood induction. Subjects were asked to rate on a scale from 1 (not at all) to 9 (extremely) how much they experienced each of the following emotions: angry, frustrated, calm, sad, guilty, amused, and happy.

Emotion Regulation Questionnaire. Following completion of all behavioral tasks, participants will complete a questionnaire that will assess ER strategies used during the MTPT and how successful the strategies were.

Trait Measures. Participants completed a number of trait measures. To assess the extent that they reported controlling anger, anxiety and depressed mood, participants completed the Courtauld Emotional Control Scale (CECS; Watson & Greer, 1983). Participants responded on a scale from 1 (almost never) to 4 (almost always) to 21 prompts such as "When I feel angry… I smother my feelings" and "When I feel unhappy… I put on a bold face."

To assess trait anger and aggression (a behavioral correlate of anger), participants completed two self-report measures: the State-Trait Anger Expression Inventory – 2 (STAXI-2) and the Aggression Questionnaire (AQ; Buss & Perry, 1992). The STAXI-2 is comprised of 57 questions that create the following subscales: State anger (Feeling angry, Feeling like verbally expressing anger, Feeling like physically expressing anger), Trait anger (Angry temperament, Angry reaction), Anger expression (Anger-in, Anger-out), and Anger control (Control of anger-in, Control of anger-out), and an Anger Total score. Participants responded to statements such as, "I feel like swearing" and "I get

angry when I'm slowed down by others' mistakes," on a scale from 1 (not at all) to 4 (very much so). The AQ comprised 29 questions that created four subscales: Anger, Physical aggression, Hostility, and Verbal aggression, as well as a total score. Participants responded to statements such as, "I have become so mad that I have broken things" and "I have trouble controlling my temper" on a scale from 1 (extremely uncharacteristic of me to 7 (extremely characteristic of me).

Participants also completed the Behavioral Inhibition System and Behavioral Activation System (BISBAS; Carver & White, 1994) that measured the two general motivation systems that underlie behavior (inhibition and activation systems). The BISBAS comprised 20 questions such as "I feel pretty worried or upset when I think or know that someone is angry at me" that created two main subscales, BIS and BAS. The BAS was further sectioned into subscales of Drive, Fun Seeking, and Reward Responsiveness. Participants responded to each question on a scale from 1 (very true for me) to 4 (very false for me).

Participants also completed the Center for Epidemiologic Studies of Depression Scale (CES-D; Radloff, 1977). The CES-D comprised 20 questions such as, "I felt that everything I did was an effort," that inquired about how the participant felt in the past week. The sum of responses created a total score.

Procedure

Participants were run individually and were seated before a computer. The experimenter introduced the study as an investigation on the effects of music on cognitive tasks. Participants first completed the measures of EF and WMC, followed by their first trial of the behavioral measure (MTPT). They then watched a neutral video to allow for

recovery from resource depletion (*Coral Sea Dreaming*, Hannan, 1999). Participants then began the autobiographical and music portions of the mood induction; they continued to listen to angry music as they completed the second trial of the behavioral measure (MTPT). Prior to starting the MTPT, each participant was instructed to down regulate his or her anger in order to perform to the best of his or her ability; it was suggested that they viewed the task as a challenge and an opportunity to demonstrate their skill. All tasks were done on computer.

Power analyses.

A sample size of 100 participants was based on a conservative effect size estimate that was informed by previous studies of these constructs. *WMC and Emotion Regulation.* Schmeichel et al. (2008) demonstrated that higher WMC was associated with better ability to regulate the subjective experience of emotion with an effect size of $R^2 = 0.17$. *Verbal Fluency and Emotion Regulation.* Gyurak et al. (2011) showed that in an elder population (both controls and individuals showing cognitive decline), verbal fluency was related to several measures of down regulation of negative emotion. Verbal fluency was associated with down-regulation of emotional experience with an effect size of $R^2 = 0.11$ (controlling for age and general cognitive function). *Physiology and Emotion Regulation.* Gyurak et al. (2011) also found that verbal fluency was associated with decreased HR during down-regulation with an effect size of $R^2 = 0.13$. Given that the only known study examining individual aspects of EF and anger regulation was conducted with a sample showing deficits of EF, I also looked at studies with anger experience and regulation using a general population. These studies found significance with ER and physiological measures of anger with sample sizes ranging from 42 (Mauss et al., 2006) to 111 (Mauss

et al., 2007). Further indirect support was found by Memedovic, Grisham, Denson, and Moulds (2010), who demonstrated that trait reappraisal predicted reduced diastolic and systolic blood pressure following provocation; effect sizes of $R^2 = 0.61$ and $R^2 = 0.52$ indicated necessary sample sizes of 14 and 20 respectively. Based on these previous studies I used nQuery Advisor Version 7.0 (Elashoff, 2007) to estimate the required sample size to detect the significance of a single predictor in a four predictor regression model that explains a variance proportion of $R^2 = 0.1$ in the population (the smallest reported effect size was $R^2 = 0.11$). This number was $N = 97$.

Chapter 3. Results

Self-report affect

Mood composite. Factor analysis with individual items from the four mood rating scales (Affective Rating Scale, ARS) yielded 3 primary affective composites: Happy (cheerful, happy, pleased, energetic), Angry (irritable, angry, frustrated, grouchy), and Sad (sad, down, depressed, gloomy). These composites were the primary affective ratings subsequently used in all analyses involving self-report mood.

Mood across time. Participants completed the ARS at four time points following: a neutral movie clip (baseline, Time 1), Mirror Tracing Persistence Task #1 (MTPT1, Time 2), mood induction procedure (MIP, Time 3), and Mirror Tracing Persistence Task #2 (MTPT2, Time 4, see Figure 3.1). A 3 (Mood: happy, angry, sad) x 4 (Time point) repeated measures ANOVA assessed changes in mood across time (see Table 3.1).

There were significant effects of both mood and time, and of their interaction. To assess change in mood across the study, I conducted paired sample *t*-tests. MTPT1 increased negative and decreased positive affect. Compared to baseline (Time 1), mood after the MTPT1 (Time 2) showed significantly increased anger ($t(81) = 11.43$, $r^2 = 0.62$, $p < .001$) and sadness, ($t(81) = 2.67$, $r^2 = 0.08$, $p < .01$), and significantly decreased happiness ($t(81) = 7.55$, $r^2 = 0.41$, $p < .001$). Compared to baseline, mood after the mood induction (Time 3) showed significantly increased anger ($t(81) = 8.77$, $r^2 = 0.49$, $p < .001$) and sadness ($t(81) = 3.83$, $r^2 = 0.15$, $p < .001$), and significantly decreased happiness ($t(81) = 8.85$, $r^2 = 0.49$, $p < .001$). Compared to baseline, mood after MTPT2 (Time 4) showed significantly increased anger ($t(81) = 11.35$, $r^2 = 0.62$, $p < .001$) and

sadness ($t(81) = 3.05$, $r^2 = 0.10$, $p < .01$), and significantly decreased happiness ($t(81) = 7.90$, $r^2 = 0.44$, $p < .001$).

This study focused on the experience and regulation of anger, and therefore it was important to show a selective increase in anger rather than sadness (see Figure 3.1 and Table 3.2). There was no difference between anger and sadness at baseline (Time 1; $t(82) = 1.18$, *ns*). Anger was significantly higher than sadness following MTPT1 (Time 2; $t(84) = 12.08$, $p < .001$), the mood induction (Time 3; $t(84) = 8.57$, $p < .001$), and MTPT2 ($t(84) = 12.11$, $p < .001$). Thus, anger, more so than sadness, increased following MTPT1, the mood induction, and MTPT2. It is also interesting to note that anger was significantly higher following both of the MTPTs than following the angry mood induction, MTPT1 ($t(83) = 3.69$, $r^2 = 0.14$, $p < .001$) and MTPT2 ($t(83) = 3.15$, $r^2 = 0.11$, $p < .01$). Anger, after the mood induction and prior to starting MTPT2, was still significantly elevated compared to baseline ($t(81) = 8.77$, $r^2 = 0.49$, $p < .001$) and was therefore sufficient to test the ability to work on a frustrating task while angry.

Physiology across time. Peripheral physiology, including heart rate (HR), respiratory sinus arrhythmia (RSA), and respiration, was measured across time. Compared to baseline, HR increased significantly during MTPT1 ($t(84) = 2.87$, $r^2 = 0.09$, $p < .01$), MTPT2 ($t(82) = 3.90$, $r^2 = 0.1$, $p < .001$), and recovery ($t(83) = 7.56$, $r^2 = 0.44$, $p < .001$; see Figure 3.2). Compared to baseline, RSA did not significantly change during MTPT1 ($t(84) = 0.88$, $r^2 = 0.01$, *ns*), or MTPT2 ($t(82) = 0.06$, $r^2 = 0.00$, *ns*). RSA during recovery was significantly higher than baseline ($t(83) = 2.67$, $r^2 = 0.08$, $p < .01$; see Figure 3.3). Compared to baseline, respiration did not significantly change during MTPT1 ($t(84) = 1.45$, $r^2 = 0.02$, *ns*), or MTPT2 ($t(82) = 0.74$, $r^2 = 0.01$, *ns*). Respiration

during recovery was significantly lower than baseline ($t(83) = 3.45$, $r^2 = 0.13$, $p < .001$; see Figure 3.4).

Inhibition and Shifting Task

Block comparison. Paired sample *t*-tests were conducted to compare blocks one (B1) and two (B2) of the Inhibition and Shifting task. *T*-tests were conducted on each pair of cue (Color, Emotion, Gender) and procedure type (control, inhibitory, repeat, unclassified) resulting in 12 comparisons. All comparisons were significant, with faster reaction times on the second block. Combined trials from both blocks were included in further analyses.

Accuracy and reaction time cutoffs. Shifting data were not obtained from three participants due to technical error. Average accuracy for individual trial responses (not triplets, maximum possible trials = 1152) of the total sample was 85% (sd = 17.6, range = 16-99, mean = 976 trials). To set a lower limit for overall task accuracy, participants with accuracy lower than 60% (n = 11) were excluded from further analyses. Average accuracy of remaining participants (n = 87) was 90% (sd = 0.09%). To reduce the influence of outliers, any trial (t1 - t3) with a reaction time greater than 3500 ms was excluded. The 3500 ms cutoff retained 76.64% (sd = 18.1) of the total individual trials, and 89.85% (sd = 8.19%) of accurate trials for the remaining analyses. Only trials where all responses were correct were included in subsequent analyses. See Table 3.3 for the mean percentages of trials that were included [(included trials/total number of trials) * 100], when trials were excluded if they did not meet criteria for 100% trial accuracy, and reaction times smaller than 3500 ms. All further analyses were based on reaction time to last or third trial.

Initial Statistical Analyses. Initial statistical analyses were imperative for understanding the basics of the Inhibition and Shifting task, and I have conducted all basic analyses following De Lissnyder et al. (2010). However, because these analyses are not an integral component in the assessment of the aims proposed in this dissertation, I have placed them in the appendix (see Appendix D). The variables presented in the remainder of this results section are specific variables derived from those analyses. The cued dimension of Color was excluded at time points two and three in remaining analyses due to its RT's being significantly faster than RT's for Gender and Emotion (see De Lissnyder et al., 2010).

Cued dimensions and Valence specific affect. Given the focus of this study on ER and affective stimuli, the remaining analyses will highlight two important aspects of each Shifting and Inhibition variable. First is whether the cued dimension is affective (Emotion) or neutral (Gender, Color). It is important to note whether the particular variable is shifting to or from an affective or neutral cue. Furthermore, I will examine *valence specific* Inhibition and Shifting results. Valence refers to the emotional expression (Angry or Happy) of the face of the correct picture in a specified trial (trial1 (t1), trial 2 (t2), or trial 3 (t3)). These variables are labeled in reference to the two emotions in the order in which they are presented (see Table 3.4).

Inhibition.

Inhibition composites were computed as the difference between reaction times during inhibitory and control trials to all cued dimensions (the cued dimension of Color was not included at t3 because of significantly faster RTs). Taking longer to respond when a cue was just inhibited (compared to control trials)—that is, getting over the initial

inhibition—indicates better inhibition, a marker of better EF. Therefore, higher inhibition scores indicate better EF.

Inhibition – Valence specific composites. Valence specific Inhibition composites were created according to the valence of the correct stimulus from the first and third trials of inhibitory trials, and the valence of the final correct stimulus of the control trials. The valence of the last trial was the same for both trial types.

To examine the influence of valence at t1 and t3 on inhibition composite scores, I conducted a 2 (t1 valence: Happy, Angry) x 2 (t3 valence: Happy, Angry) repeated measures ANOVA. Results indicate that there was no main effect of valence at trial 1 ($F(1,86) = 1.125$, *ns*) or at trial 3 ($F(1,86) = 3.20$, $p = .077$), and there was no interaction effect ($F(1,86) = .12$, *ns*). Although not significant, the pattern of results suggests that Angry valence at t3 yielded smaller Inhibition scores, compared to when Happy was the correct valence at t3 (see Table 3.5). This is similar to the findings of De Lissnyder et al. (2010).

In summary, there were no significant valence based differences for inhibition trials or Inhibition composites. However, the pattern of results for the Inhibition composites indicates that changing to Happy at t3, rather than to Angry, tended to yield higher composite scores that indicated better Inhibition. That participants respond more quickly when the target stimulus is Angry may indicate a vigilance to Angry stimuli, even when required to inhibit this cued dimension.

Inhibition to Emotion cues – Valence specific composites. Next, I examined valence specific Inhibition costs when inhibiting the cued dimension of Emotion or Gender only. To examine the ability to inhibit the cued dimension of Emotion, I included

only trials where the cued dimension at t1 and t3 was Emotion, and at t2 the cued dimension was Gender. Trials were indexed by valence at t1 and t3. I conducted paired sample *t*-tests to compare the effect of valence on RT to emotion inhibitory and control trials, and to the Inhibition composites. There were no significant differences for inhibitory or control trials. Inhibition_A_H was significantly higher than from Inhibition_A_A ($t(69) = 2.19$, $p < .05$, see Table 3.6[1]).

Inhibition to Gender cues – Valence specific composites. To examine the ability to inhibit the cued dimension of Gender, I included only trials where the cued dimension at t1 and t3 was Gender, and t2 cued dimension was Emotion. Trials were indexed by valence at t1 and t3. There were no significant differences between composite scores (see Table 3.7).

Set shifting

Set shifting composites comprised the average of the shifting trials (control, unclassified) minus repeat trials (see Appendix C). This composite represents the additional time that it takes to switch between cued dimensions compared to repeat trials. A smaller difference represents better ability to shift between different rules and indicates better EF.

Set shifting - Valence specific trial types. To examine valence specific shifting costs, I included all combinations of Happy and Angry at t2 and t3, for repeat, unclassified, and control trials. I examined trials that included switching to and switching

[1] Different N values are due to decreased number of trials when applying constraints to both cued dimensions and valence of correct stimuli. Participants without correct trials of each type were not included.

from Emotion. For additional information regarding trial types for the below composites, please see Appendix C.

Set shifting - Valence specific composites. Valence specific Shifting composites were created according to valence of the t2 and t3 for shifting trial types (control, unclassified) and the t1 and t2 for repeat trial types. The valence of the last trial was the same for all three trial types.

Gender to Emotion – Composite. To examine the influence of valence at trial 2 and valence at trial 3 on the Shifting composite, I conducted a 2 (t2 emotion: Happy, Angry) x 2 (t3 emotion: Happy, Angry) repeated measures ANOVA (see Table 3.8). There was a main effect of valence at trial 3. The Shifting composite was significantly higher when t3 valence was Happy compared to when t3 was Angry. There was no main effect of valence at t2, and there was no significant interaction effect. I conducted paired sample *t*-tests to compare influence of different valence switches within the Shifting composites (see Table 3.9). There was a significant difference when comparing H_H to A_H ($t(83) = 2.69$, $p < .01$). The difference between H_A versus A_A approached significance, ($t(83) = 1.87$, $p = .065$). All other comparisons were not statistically significant.

In summary, Shifting composites that did not require a change of valence (i.e., H_H, A_A) tended to have higher set shifting scores than set shifting composites that did require a valence change. However, the respective comparisons were not all significant.

Emotion to Gender – Composite. To examine the influence of t2 and t3 valence on set shifting composite scores when switching from Emotion to Gender, I conducted a 2 (t2 valence: Happy, Angry) x 2 (t3 valence: Happy, Angry) repeated measures

ANOVA. There was no main effect of valence at t2 or at t3, and there was no interaction effect. I conducted paired sample *t*-tests to compare influence of different valence changes within the Shifting composites (see Table 3.10). Shifting H_A had a significantly higher shifting score than Shifting A_A, $t(86) = 2.31$, $p < .03$. All other comparisons were not significant.

Relationship between variables. Shifting negatively correlates with Inhibition ($r(87) = -3.17$, $p < .01$) which indicates that better Shifting is associated with better Inhibition. Both Shifting ($r(85) = -.50$, *ns*) and Inhibition ($r(85) = .09$, *ns*) are not significantly correlated with working memory.

Physiological and performance outcome variables were minimally correlated. Respiration (difference score) was significantly correlated with errors on MTPT2 ($r(75) = .28$, $p < .02$) and total time on the MTPT2 ($r(75) = .33$, $p < .01$). HR and RSA were not significantly correlated with any of the MTPT2 outcome variables.

Hypothesis Testing

In the introduction, I hypothesized that better executive functioning at the most basic level (lower composite scores for Shifting, higher for Inhibition) would predict outcome variables (self-report anger, physiological reactivity, performance on a frustrating task). In conducting the analyses, it became clear that the message of the results appeared in the details of the composite scores, therefore I have classified each Shifting and Inhibition composite on two additional layers. The first is cued dimension – are participants switching to or from a neutral cue (i.e., Gender, Color) or an affective cue (Emotion)? The second layer is the valence of the correct picture in relevant trials– is the target stimulus portraying a Happy or an Angry face? This creates two cue types for each

of Shifting and Inhibition composites, plus a combined Shifting composite that includes shifting to and from both Gender and Emotion (see #3 in Table 3.11). Each of these cue types includes the four different valence combinations (see Table 3.11). The inclusion of these layers provides a more nuanced understanding of the role of affective and neutral cues, and, thereby, of how people attend to negative (Angry) and positive (Happy) stimuli. I use each numbered variable (#1-5) at the level of cued dimension (affective/neutral) as the set of predictors in its own regression.

Aim #1.

My first aim was to examine the effects of individual differences in Inhibition and Shifting on the ability to down regulate or control anger during a frustrating task. I conducted separate regression models to investigate the predictive value of each set of Shifting and Inhibition composites (Table 3.11, #1-5) on the ability to down regulate anger during a frustrating task after an anger mood induction. Different ways of measuring the ability to regulate anger were included as different dependent variables: self-report anger following the stressful task (Regression 1.1a), and HR (Regression 1.1b) and RSA (Regression 1.1c) during the stressful task. HR and RSA were difference scores between physiological values measured during MTPT2 and at baseline. I hypothesized that better abilities in Shifting (smaller number) and Inhibition (larger number) would predict better anger regulation as evidenced by lower self-report anger (Regressions 1.1a-c), lower HR (Regressions 1.2a-c) and higher RSA during the MTPT (Regression 1.3).

Regression 1.1a-b: DV: Self-report anger. I conducted stepwise regression analyses with self-report anger after the second MTPT (Time 4) as the dependent variable: baseline anger and working memory (OSpan) were included as control

variables, and valence specific Shifting from Gender to Emotion (Regression 1.1a) and Shifting from Emotion to Gender (Regression 1.1b) as independent variables. Regression 1.1a results indicate that baseline anger, Shifting H_A, and Shifting A_A were significant predictors of self-report anger after a frustrating fine motor task (see Tables 3.12-3.13).

Next, I examined valence-specific Shifting effects when shifting from Emotion to Gender. The model was significant; Shifting A_H and baseline anger were significant predictors of self-report anger after MTPT2 (see Tables 3.14-3.15).

Regression 1.1c. I entered valence specific Inhibition composites (to Emotion, and in a separate regression, to Gender) in the second step instead of the valence specific Shifting composites. None of these variables were significant predictors of self-report anger following the second MTPT.

Controlling for different baseline anger (Regression 1.1a-b). To examine the influence of anger after the mood induction on anger following MTPT2, I ran additional regression models controlling for anger after the mood induction. Anger after the mood induction was a more significant predictor of anger after MTPT2 than anger after MTPT1 (step 2 $t = 8.34$ versus $t = 6.75$). The originally significant shifting variables (H_A and A_A) remained significant. This indicates that perceived anger after the mood induction provided a control similar to self-report anger following the baseline. The same pattern holds true for Regression 1.1b (IV: Shifting to gender, DV: Self-report anger).

Regression 1.2a-c: DV: Heart rate. I conducted the same step-wise regressions as above, but with HR as the dependent variable. Shifting H_H (from Gender to Emotion; Regression 1.2a, see Tables 3.16-3.17) and Inhibition H_A (with both cued dimensions of Gender and Emotion) were significant negative predictors of HR (Regression 1.2b, see

Tables 3.18-3.19). Shifting A_A (from Emotion to Gender) was a significant positive predictor (Regression 1.2c, see Tables 3.20-3.21). None of the overall models were significant. There were no other significant predictors.

Regression 1.3: DV: RSA. I conducted the same regression analyses with RSA as the dependent variable, and respiration as an additional control (included in step 1). There were no significant predictors and the models were not significant.

Aim #1 Summary. These results partially support my hypothesis that better EF is associated with lower subjective anger and physiological activity. Better performance on Shifting and Inhibition variables that required a change in valence supported my hypothesis. Specifically, better Shifting H_A (to Emotion) and Shifting A_H (to Gender) were associated with lower self-report anger; better Inhibition H_A (both) was associated with lower HR during the stressful task.

Better performance on Shifting and Inhibition variables that did not require a valence change (Angry or Happy) predicted higher anger or HR and therefore did not support my hypothesis. Specifically, better Shifting A_A (to Emotion) is associated with higher anger, and better Shifting H_H (to Emotion) and Shifting A_A (to Gender) were associated with higher HR.

Aim #2.

My second aim was to assess individual differences in Inhibition and Shifting associated with maintained or improved performance on a frustrating visual motor task when angry (MTPT2). Performance on MTPT2 was measured in a number of ways, and each outcome variable is included as the dependent variable of a separate regression. I hypothesized that better ability in Shifting and Inhibition would predict better

performance on three dimensions: Errors per second (fewer errors per second), maximum distance (larger distance) and total time (higher total time). These regressions controlled for baseline anger, working memory (Ospan). If the DV is time on MTPT2, then time on MTPT1 was also included as a control. Shifting and Inhibition variables were included as predictors in separate regressions.

Regression 2.1a-b: Errors per second. Shifting H_H (to Emotion) was a significant predictor of errors per second (see Tables 3.22-3.23). The overall model was significant and errors per second on MTPT1 and working memory were also significant predictors.

Inhibition A_H (to Emotion) was a significant predictor of errors per second (see Tables 3.24-3.25). The model was significant and errors per second on MTPT1 and working memory were significant predictors. None of the Shifting or Inhibition variables were significant predictors of total time spent on the second MTPT.

Aim #2 Summary. Overall, these results did not confirm my hypothesis; better Shifting and Inhibition scores did not predict better performance on MTPT2. Better performance on Shifting and Inhibition variables that required a response to a Happy target stimulus on the last trial (from either Happy or Angry) predicted higher errors per second. Specifically, better performance on Shifting H_H (to Emotion) and Inhibition A_H (to Emotion) were associated with more errors per second on MTPT2. Conversely, working memory was a significant negative predictor, indicating that higher working memory (better) was associated with fewer errors per second.

Aim #3.

Aim 3 examined the relationship between physiological response and performance on the second MTPT. I hypothesized that decreased HR and increased RSA during the MTPT2 (difference from the baseline) would be better predictors of MTPT2 performance. To assess this hypothesis, I conducted regression analyses to investigate the independent effects of HR (Regression 3.1a) and RSA (Regression 3.1b) on MTPT2 performance (total time).

Regression 3.1a-b: Total Time (MTPT2). In the first regression (Regression 3.1a, see Tables 3.26-3.27), HR was a significant negative predictor, and time spent on MTPT1 was a significant positive predictor, of time spent on MTPT2. The relationship between HR and performance on MTPT2 indicates that higher HR during MTPT2 is associated with less time spent on MTPT2.

RSA was the predictor in the second regression, controlling for respiration and time spent on MTPT1 (Regression 3.1b, see Tables 3.28-3.29). Time spent on the first MTPT and respiration were significant predictors of time spent on MTPT2. MTPT1 and respiration are positively correlated with time spent on MTPT2, indicating that better initial performance and faster breathing rate are associated with longer time spent on MTPT2. RSA was not a significant predictor. Adding baseline anger as an additional control did not change these results.

Regressions 3.2: Other MTPT2 Performance Measures. Additional regressions were run to assess the influence of physiology on other MTPT performance measures. Lower HR was associated with increased errors (see Tables 3.30-3.31). All other predictors were not significant.

Aim #3 Summary. These results partially confirmed my hypothesis. HR was a significant negative predictor of time spent on MTPT2: lower HR was associated with working longer, which confirmed my hypothesis. Similarly, respiration was a significant predictor of total time spent on MTPT2 with faster respiration being associated with working longer. Conversely, RSA was not a significant predictor of time on MTPT2, which did not support my hypothesis.

Chapter 4. Discussion

In the present study, I examined whether specific aspects of executive function were associated with anger regulation and performance when angry. I also examined the role of physiology as a marker of emotional response and regulation on the ability to perform on a frustrating fine motor task. To these ends, I proposed three aims. The first aim was to examine the influence of individual differences in inhibition and shifting on the ability to down regulate anger during a frustrating task. The second aim was to investigate the role of these same individual differences in the performance on a frustrating fine motor task. The third aim was to examine the influence of physiological response to anger and anger regulation, again on performance on a frustrating task.

To assess these aims I administered an affective task shifting paradigm to a sample of undergraduate students. Additionally, participants completed a frustrating fine motor task (Mirror Tracing Persistence Task - MTPT) both prior to and following an angry mood induction. Physiological data (HR, RSA, respiration) were collected throughout the duration of the study.

In line with De Lissnyder et al. (2010), I calculated composite scores for Shifting and Inhibition based on comparisons between trial types. Comparison of trial types were similar in order of reaction time to those found by De Lissnyder et al. (2010) and are explained in the appendix (Appendix D).

In order to more carefully examine the role of affective versus neutral cues, I created Shifting and Inhibition composites that accounted for shifting to or from the cued dimension of Emotion, or inhibiting either neutral (Color, Gender) or affective (Emotion) cued dimensions. The cued dimension of Color was not included in the last two trials

because it was significantly faster than the other dimensions (De Lissnyder, 2010). To more carefully examine the role of affect, Shifting and Inhibition composites specified the valence (Angry/Happy) of the target stimuli, proving information as to whether subjects were required to change valence and in which direction. These composites, reflecting cued dimensions and valence, were analyzed on their own and ultimately used to examine specified aims.

Inhibition and Shifting composites

Comparison of the Inhibition and Shifting composites show that, although there were only a few comparisons that reached significance, two classes of results can be discerned:

(1) *Inhibition costs: less effective EF is associated with valence change to Anger.* For the Inhibition composites where the cued dimension of Emotion was inhibited, changing to Angry was associated with smaller Inhibition costs than changing to Happy. Smaller Inhibition costs are indicative of less effective EF, which indicates that it is harder to inhibit an emotional cue when the last emotion is Anger (i.e., you are faster to respond to the Angry cue). This may be due to a heightened vigilance to anger, which overrides the previous inhibition of the cued Emotion dimension. Similarly, when inhibiting the cued dimension of Gender, changing away from Happy yielded lower composite scores indicative of less effective executive function.

Some Inhibition costs were negative indicating that, for these specific cases, the inhibitory trials were faster than the control trials. Given the number of layers involved in understanding the cause, and influence, of these negative composites, I will discuss them on a case-by-case basis as they come up in the hypothesis testing.

(2) *Shifting cost: less effective EF is associated with valence changes.* Shifting costs to Emotion were significantly higher when they did not require a valence change compared to Shifting costs that did involve a change in valence. Shifting costs to Gender were significantly higher when changing from Happy to Angry compared to seeing two Angry stimuli. No other patterns emerged from Shifting costs to Gender. Negative Shifting costs indicate that the repeat trials were slower than the shifting trials; these significant predictors will be explained below.

Hypothesis Testing – with Shifting variables as predictors

As a means of simplifying the interpretation on my results, I conceptualize the results that include the shifting variables (Aims #1 and #2) as falling into four sets based on valence change and whether or not the outcome is consistent with my hypotheses. These categories have two functions: (1) they make it clear as to whether each result is consistent with my hypotheses, and (2) they provide a framework for discussing the nuances of the data. The conceptual sets are as follows (see Table 4.1):

I. Better performance on Inhibition and Shifting variables that require a change from Happy to Angry are associated with better outcome (i.e., lower self-report anger and lower HR). These findings support my hypotheses.
II. Better performance on Shifting variables where Angry stimuli are presented at both time points is associated with mixed results – half of the results are consistent with my hypotheses and half are not.
III. Better performance on Inhibition and Shifting variables that require a change from Angry to Happy present mixed results – half of the results are consistent with my hypotheses and half are not.

IV. Better performance on Shifting variables where Happy stimuli are presented at both time points are associated with worse outcomes (i.e., higher heart rate, more errors). These results are not consistent my hypotheses.

For each individual Shifting and Inhibition variable there is an interpretation based on affective/neutral cues and valence changes that potentially explains the relationship with the outcome variables. Based on this conceptualization, I will use each set as a starting point for an individual section of the discussion, and will explore the individual findings that it comprises.

I. Happy to Angry: Consistent with hypotheses. The first conceptual set presents two explanations for the relationship between executive function, valence change, and indicators of emotion regulation. The first explanation supports the idea that better inhibition of a happy face (as evidenced by Inhibition H_A) is associated with lower heart rate. The second explanation supports the idea that being faster to locate an angry face (evidenced by Shifting H_A) is associated with lower self-report anger. Both outcomes imply better emotion regulation.

Results indicate that better ability on Inhibition H_A is predictive of a smaller increase in heart rate during a stressful task. Better scores represent better inhibition when finding the Happy target and ignoring the Angry faces. Inasmuch as participants were able to effectively do so, they were slowed down when required to switch to trials with Angry target stimuli. This can be interpreted as showing that a better ability to inhibit is associated with smaller change in heart rate during a stressful task. This relationship creates an association between better inhibition of affective stimuli and lower sympathetic activation during a frustrating task. These Inhibition scores include cued

dimensions of Gender and Emotion at both time points, so do not provide information regarding switching to or from Emotional cues. This finding supports my hypothesis that better executive function is associated with better physiological aspects of ER.

The next finding also confirms my hypothesis. Better ability with Shifting H_A is associated with lower self-report anger following a frustrating task. Better shifting scores consist of shifting trials from a Happy face (neutral cue: Gender) to an Angry face (affective cue: Emotion) that are closer in latency to repeat trials with the same valence change. This implies that participants were able to quickly shift to Angry stimuli, which may indicate augmented attention towards locating Angry stimuli. This affective-biased attention, or vigilance, to angry stimuli was associated with lower anger following a frustrating task. This suggests that vigilance towards anger provides the ability to quickly locate and deal with angry stimuli, thus allowing time for adequate emotion regulation, which promotes lower self-report anger. While the hypotheses from the first conceptual set are not a priori intended to address the question of vigilance, they nonetheless provide valuable information that can be interpreted in this context.

The anxiety literature examines vigilance to negative affective cues and its relationship with the ability to efficiently regulate emotions. This research has yielded mixed results regarding whether anxious individuals pay more or less attention to anxiety provoking stimuli – results vary depending on duration of stimulus presentation, type of stimulus, and whether stimulus avoidance leads to increased or decreased uncertainty (Mansell et al, 1999). My results regarding the ability to quickly locate angry or happy faces, provide additional information regarding affect bias and its association with successful emotion regulation.

The significant predictor Shifting H_A presents an additional piece of information in that its mean score is negative. A negative Shifting cost indicates that the shifting trials were faster than the repeat trials. More specifically, participants were able to more quickly shift from Happy to Angry stimuli when presented with different cues, than from Happy to Angry stimuli when presented with the same cues. Since it would be expected that participants would respond more quickly to the same cue twice in a row (as seen in the repetition trials) rather than having to change cues, this equally fast response to shifting and repetition trials could indicate that participants' reaction times were more influenced by the Angry valence of the target stimuli than they were by the cued dimension. This further supports a possible role for the vigilance hypothesis – a target with an Angry face overrides the salience and direction of other cues.

The findings in this set confirm my hypothesis that better executive function is associated with better emotion regulation as evidenced by lower anger. But they do raise questions at the affective level – why would being able to quickly locate and change to anger be associated with lower perceived anger? This question suggests two possible interpretations regarding how sensitivity to finding angry faces influences emotion regulation: Does increased sensitivity to angry stimuli indicate vigilance and a concomitant increase in time to effectively regulate? Or does decreased sensitivity indicate a better emotion regulation strategy? The next two findings provide additional evidence on the topic.

II. Angry to Angry: Partially consistent with hypotheses. These next two results involve Shifting composites where both shifting and repeat trial types have Angry target stimuli for both trials. The lack of valence change distinguishes this category from the

previous one. This category presents mixed findings both with regard to hypothesis testing and understanding on an intuitive level.

Better Shifting A_A (cues: Emotion to Gender) is associated with lower heart rate. This fits with the previous interpretation that better vigilance to Angry stimuli (e.g., shift trials with valence change from Happy to Angry) provides adequate time and attention to allow for effective regulation of negative emotions when frustrated. This specific relationship shows an association with a smaller increase in HR during a frustrating task.

This finding supports my hypothesis that better executive function is associated with decreased sympathetic activation. It also bolsters the vigilance hypothesis – changing quickly to an Angry target stimulus is associated with having enough time for effective emotion regulation. It extends this relationship to include two Angry stimuli, indicating that it is not just the valence change from Happy to Angry that is associated with increased vigilance to Angry stimuli.

The two previous findings indicate that shifting to Angry is associated with lower anger and lower heart rate. The next predictor deviates from these findings in cued dimension, valence, and relationship to vigilance, and helps to refine the predictors of successful emotion regulation.

This finding indicates that better ability in Shifting A_A (cues: Gender to Emotion) is predictive of increased anger. This suggests that higher vigilance to Angry stimuli is associated with increased anger, a different perspective on vigilance than that offered by the prior findings. This predictor is different from the other predictors as follows. It is different from Shifting A_A (cue: Emotion to Gender) because it requires a

shift in cued dimensions from Gender to Emotion. This means that when there are two back-to-back Angry stimuli, when the last cue requires finding an affective cue, this is associated with increased self-report anger. It is different from Shifting H_A because there is no valence change, but both trials have a shift in cued dimension from Gender to Emotion.

This suggests that, for participants who experience more anger after the frustrating task, focusing on an Angry target even if the cue is neutral (Gender) facilitates responses to an Angry target when the cue is affective (Emotion). In other words, responding quickly to Angry stimuli at multiple time points, regardless of the cue is associated with higher anger. An additional explanation is that the small difference between shifting and repeat trials with two Angry trials may indicate that when a participant is presented with back-to-back Angry stimuli they ignore the switch in cued dimensions, and just search for the Angry stimuli.

While these findings do not definitively indicate whether vigilance to Angry stimuli is beneficial or detrimental to efficient emotion regulation, they begin to provide specific parameters (cue, valence) that should be incorporated when examining this question.

III. Angry to Happy: Partially consistent with hypotheses. I next discuss results with variables that include a valence change from Angry to Happy. The first result indicates that better Inhibition A_H is associated with more errors on the frustrating task. Higher inhibition scores represent a more complete inhibition of Angry emotional cues, which is demonstrated by a subsequent slower response to Happy stimuli, and in this case is associated with more errors. One possible way to explain this relationship is that one's

ability and tendency to inhibit angry cues on this task is representative of one's tendency to inhibit anger in the real world. Inhibiting anger may take considerable effort and detract from a task at hand, resulting in the higher number of errors on the frustrating task after an angry mood induction. This finding is in line with research that has found that cognitive control is critical for, and depleted by, controlling anger and aggression (Denson, 2011).

Better Shifting A_H costs were associated with lower self-report anger after a frustrating task. This indicates that a better ability to shift from an Angry stimulus with an affective cue to a Happy stimulus with a neutral cue is associated with lower perceived anger. This both confirms my hypothesis and indicates that being able to quickly locate a happy face is associated with lower anger. Furthermore, this supports the idea of executive function contributing the reappraisal, the ability to shift one's focus away from an undesired to a desired stimuli.

This finding leads nicely into the section examining Shifting scores with Happy stimuli on both trials, which allows us to compare changing from Angry to Happy or seeing two Happy stimuli. These three predictors create an intriguing combination – better Shifting to Happy predicts lower anger, higher heart rate, and more errors (see next section for details on heart rate and errors). This demonstrates that faster/more efficient responding to Happy images is indeed associated with less anger but is also accompanied by worse physiological responding and performance during a frustrating task.

*IV. Happy to Happy: Not consistent with hypotheses***.** Better Shifting H_H was associated with higher heart rate and more errors per second, outcomes that are not consistent with my hypotheses. This result can be interpreted to say that people who are

quick to focus on the happy stimuli are more disrupted when asked to perform in an angry state. The rapid focus on the happy stimuli and corresponding negative performance may indicate that this happy state is not conducive for the fine motor performance required for this frustrating task. The increased sympathetic changes (increased heart rate) may further indicate a discomfort with being angry and performing an increasingly frustrating task. These findings support research indicating that experiencing some anger is beneficial for performance on certain tasks (Tamir, 2005).

Importantly, results also indicate that better working memory is associated with fewer errors per second. This is important because it demonstrates that while a neutral working memory task predicts better performance, a more fine-grained executive function measure with affective stimuli supports the opposite relationship. This further endorses the importance of including emotional stimuli when examining executive function.

Hypothesis Testing – Physiological variables as predictors

In addition to examining the role of executive function in perceived anger, physiological response, and performance on a frustrating fine motor task, I assessed the influence of physiological state on the ability to perform the fine motor task.

Results examining the role of heart rate on performance on the frustrating task indicate that a smaller increase in heart rate during the frustrating fine motor task is associated with longer time spent on the task. This indicates that lower sympathetic activity (reflecting less activation of the fight or flight system) is associated with being able and/or willing to work longer on a fine motor task designed to increase frustration. This implies that being able to remain ‘cool under pressure’ is associated with increased

willingness to keep working. However, this same smaller increase in HR was associated with more errors on the task. So while these participants were willing to work longer on this task, their performance was impaired.

This is in line with physiological research. Heart rate is a marker of sympathetic activation, which prepares the body for fight or flight. Lower heart rate is indicative of decreased sympathetic activity, which is associated with periods of rest and exploration. Increases in heart rate are associated with preparation for managing a stressor. The smaller increases that were associated with longer duration may represent a person's willingness to "explore" the challenging task, but a physiological state that is not congruent with being able to step up to a challenge.

Respiratory Sinus Arrythmia (RSA) was not a significant predictor of performance. RSA represents parasympathetic activation; increased RSA has been associated with successful emotion regulation and indicates a relaxed state. This finding indicates that parasympathetic activation was not associated with performance on the frustrating task. However, this same analysis showed that faster respiration was associated with longer duration on the task. In the emotion regulation literature, respiration is usually included primarily as a control for RSA (either physically or statistically) but is not discussed as an indicator or predictor of emotion experience or regulation (Butler, et al., 2006). It has however been shown that quicker and slightly deeper than normal breathing rates is a physiological marker of anger (Philippot, Chapelle, & Blairy, 2002). This would make sense given subjects had just undergone an anger induction procedure and were working on a task designed to increase frustration. That increased respiration is associated with working longer may indicate that

experiencing some physiological symptoms of anger may motivate a person to work longer.

General discussion.

This study combines and builds upon a number of different lines of research. Anger is traditionally associated with negative affect and negative outcomes, however more recently researchers are starting to look at anger in a more positive and beneficial light (Carver, 2009; Tamir, 2008). This contrast between the detriments and benefits of anger leads us back to this study's main research question: what distinguishes individuals who are able to utilize and regulate anger successfully from those who are not? In an attempt to answer this question, I focused on the role of cognitive emotion regulation and drew from various lines of related literature including the role of broad cognition (working memory capacity) and specific executive function in enhanced ability to regulate emotions and in psychopathology, as well as the role of cognition in trait anger. With this question in mind, and knowledge of these connecting literatures, this study is the first, to my knowledge, to examine the contribution of specific measures of executive function to affective stimuli to anger regulation, physiological responses to anger and anger regulation, and performance on a frustrating task.

Results from this study provide a number of insights into the role of executive function in the ability to regulate anger. I will first touch on the complexity and then elaborate on the relationships revealed here, including, 1) vigilance to anger can be beneficial or detrimental, depending on the circumstances, 2) executive function can be associated with reappraisal and rumination with anger, and 3) better executive function in

conjunction with happy stimuli is associated with reduced anger but impaired performance.

These results indicate that there is a relationship between executive function and anger regulation, and highlight the complexity of this connection. This in turn calls attention to the complex methodology that is required to first examine executive function with affective stimuli, and then to relate these components to anger regulation. My results stress the importance of examining both neutral and affective cues, as well as the valence of relevant stimuli.

When examining my results on a more detailed level, a few pertinent themes are evident. First, increased vigilance to angry stimuli can either be beneficial or detrimental for emotion regulation or performance when angry. This is in line with the idea that anger has the opportunity to be both harmful and beneficial, depending on how it is managed. It also provides preliminary evidence indicating that differences in executive function with affective stimuli may contribute to either effective regulation or dysregulation of anger. When a person shows a biased response towards anger, consideration of both the cued dimension and stimulus valence are required to predict their response. This level of complexity is congruent with that found in the attention bias and anxiety literature where type of anxiety, type of stimuli, and duration of stimulus presentation are all relevant to understanding attention bias.

Second, select Shifting findings indicate that better ability to shift away from Angry to Happy was associated with lower perceived anger. This supports the idea that better executive function in the form of set shifting supports effective regulation of anger; this executive function has been shown to contribute to the ability to reappraise.

However, successful inhibition of anger was also associated with decreased performance. This has implications for understanding the role of executive function (specifically inhibition) for preventing rumination, as well as possible depletion of executive function when asked to inhibit a reaction to angry stimuli. In total, these results indicate that there is a relationship between executive function and anger regulation, but that further research is required to understand the nuance of this relationship.

Third, better executive function performance with happy stimuli is associated with the subjective experience of less anger, but predicts worse performance on a frustrating task. This implies that individuals who quickly attend to happy stimuli are more disrupted during a frustrating task when they are already angry. This finding corroborates recent findings that anger may be beneficial for performance on certain tasks (Tamir, 2008).

Limitations. As with all studies, this study has its limitations. To my knowledge, this is only the second study that has used the Affective Shifting task. I used different stimuli than the original study in order to include multi-racial stimuli that were congruent with the racial make-up of the sample population. Perhaps as a result, the data from this task did not cleanly replicate previous findings for the basic cued dimensions and trial types (De Lissnyder, et al., 2010, Whitmer & Banich, 2007). Most noticeably, the repeat trials were not significantly faster than the shifting trials (control, unclassified, inhibitory) across all cued dimensions. This created some disruption in the Shifting composites; the shifting cost partially dependent on the repeat trials showed the fastest reaction time of the trial types. This may indicate that negative shifting scores more accurately indicate an affective vigilance rather than a shifting cost; these were described as such on a case-by-

case basis. This may have been in part due to difficulty distinguishing emotion expression and gender for some of the stimuli. These differences limited my ability to fully utilize individual differences in shifting/inhibition to explore their role in anger regulation. An additional limitation is that the reported results were not included in my hypotheses. These findings that included valence and cues were exploratory and were conducted post-hoc.

Strengths. Despite not being able to exactly replicate previous findings for the affective shifting task, the use of affective stimuli in a cognitive task remains a strength of this study. This task allows us to begin to examine how affective stimuli influence cognitive abilities, and in turn how cognition influences the ability to regulate emotions. The use of anger is relevant for a number of reasons. Anger is extremely noticeable and potent and has repeatedly proven that it can be destructive. But it can also be helpful – this study takes a step towards answering the question of what differentiates helpful versus harmful in the world of anger regulation.

An additional, and unforeseen, strength of this study was that the MTPT proved to be incredibly frustrating. It actually increased self-report anger more than the angry mood induction. This supports the use of the MTPT for increasing both anger and frustration while simultaneously providing objective measures of participants' ability to maintain prolonged focus and utilize fine motor skills.

Implications. These results emphasize the complexity of examining affective executive function in anger and anger regulation, and in turn stress the importance of improved methodology in future studies. Follow up studies should aim to clearly tease apart cued dimensions (affective/neutral) and change in valence. This may require

examining executive function as a whole, rather than trying to distinguish between Shifting and Inhibition, in order to focus on the effects of affective versus neutral cues. With more clear findings, further studies could better inform attention bias theory as it pertains to anger.

Moreover, further research elaborating on these findings may eventually provide clinical implications for populations who have suffered repeated traumatic brain injury (TBI, see Appendix E). One symptom of repeated TBI is a decreased ability to successfully regulate strong negative emotions, which can be associated with increased aggression, depression, and, at its worst, suicide. The long-term effects of repeated TBI can be devastating and irreversible, and to date there is limited ability to decide when a person has had too many concussions which has return-to-play implications for contact sport athletes, and return-to-battle implications for military personnel. Therefore, an enhanced understanding of the role of anger regulation following TBI, and the contribution of EF to this relationship, could prove to be an effective tool for the early identification of chronic problems due to TBI. This information could allow for implementation of recovery and rehabilitation strategies.

Summary. I have linked specific measures of executive function to measures of successful anger regulation in an effort to answer the following question: What distinguishes individuals who are able to utilize and regulate anger successfully from those who cannot? No simple answer is apparent. My results suggest that there are indeed connections between executive function and anger regulation, but that these connections are complex. This experiment emphasizes the detail required in the study of executive function and emotion regulation, and indicates areas that would benefit from increased

clarity. It highlights the idea that vigilance to anger can lead to beneficial or detrimental outcomes depending on the context, it provides a connection between EF and anger that supports the idea of reappraisal and rumination, and offers increased support for the idea that EF abilities with happy stimuli do not aid performance in frustrating situations. While there is still considerable work to be done before we can answer the question of what leads to successful anger regulation, this study provides insight and focus for future research. Furthermore, this line of research has important clinical implications; it may provide information essential to improving prevention, identification, and treatment of affective disorders associated with TBI and emotion dysregulation.

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Figure 3.1. *Mood by time*

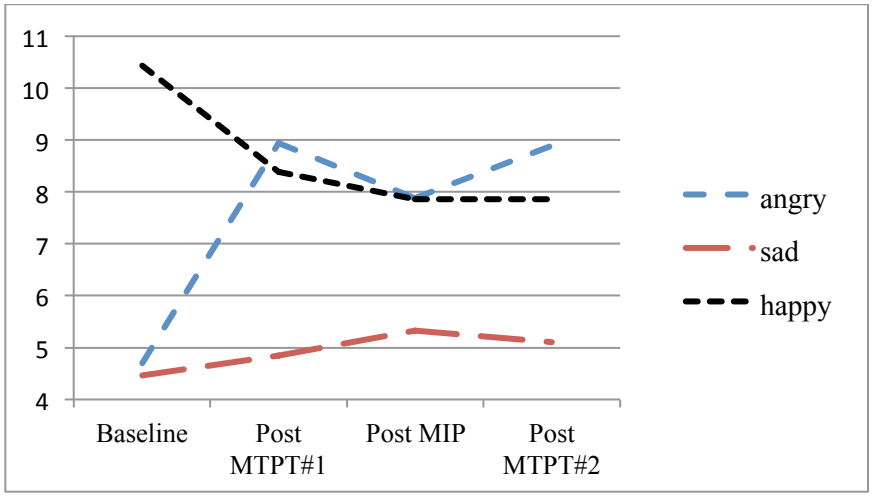

Figure 3.2 . Heart rate over time

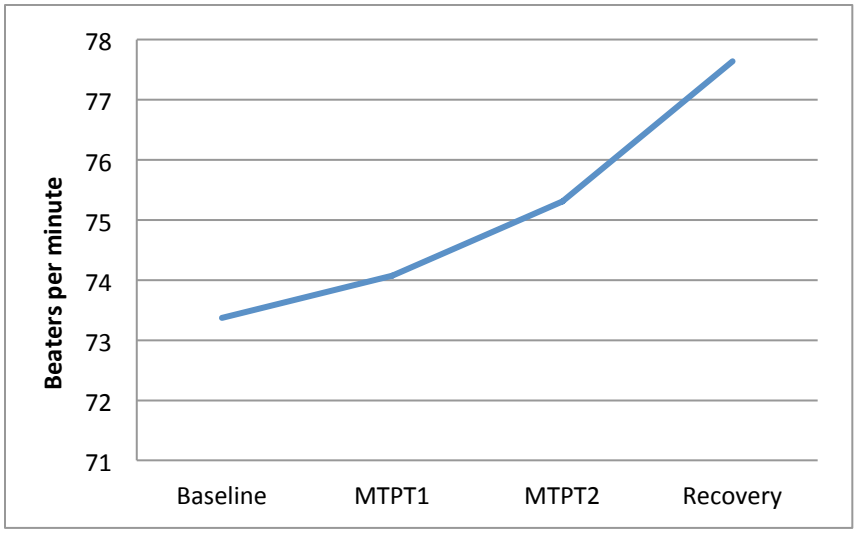

Figure 3.3. RSA over time

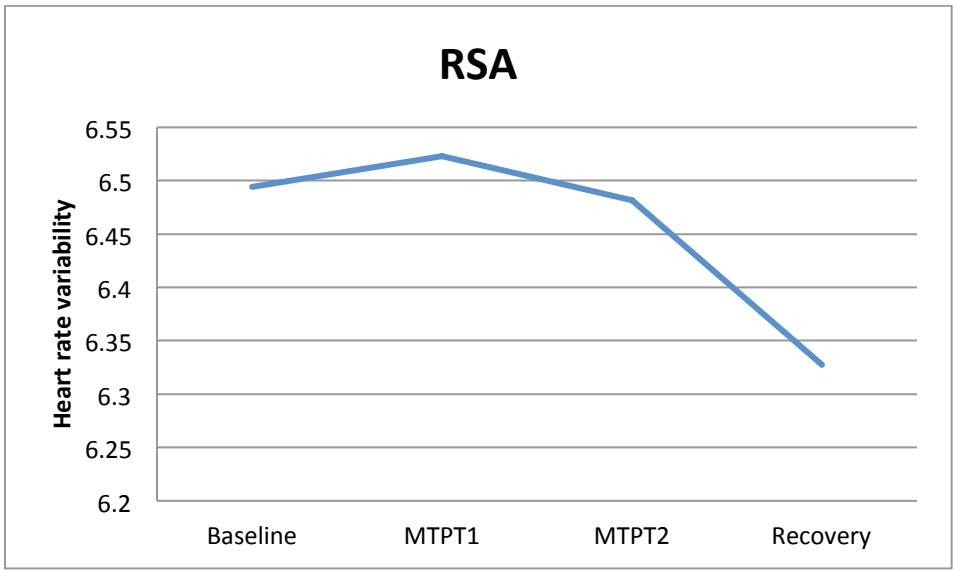

Figure 3.4. Respiration over time

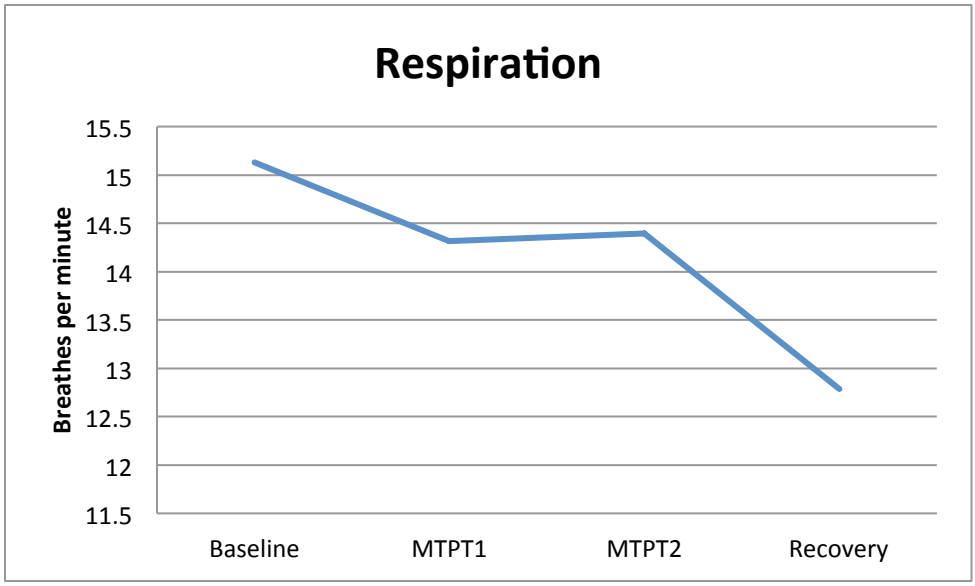

Table 3.1. *Repeated measures ANOVA for Mood by Time*

Source	df	SS	MS	*F*
Mood	(2,158)	2339.73	1169.86	44.16***
Time	(3,237)	104.64	34.88	18.00***
Mood*Time	(6,474)	1243.2	207.2	57.14***

Significance: ***$p < .001$

Table 3.2. *Means and standard deviations (SD) for mood across time points.*

	Baseline		Post MTPT1		Post MIP		Post MTPT2	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Angry	4.70	1.81	8.94	3.24	7.88	3.43	8.89	3.58
Sad	4.46	1.48	4.84	1.75	5.33	2.14	5.1	2.33
Happy	10.44	3.79	8.39	3.47	7.86	3.51	7.86	3.89

Table 3.3. *Trials included when restricting based on accuracy and RT cutoff of 3500ms.*

Trial type		N	Mean % Retained	SD
	Control	87	72.06	16.54
	Inhibitory	87	71.58	16.35
	Repeat	87	78.35	12.42
	Unclassified	87	73.67	15.61
Cued dimension				
	Color	87	81.47	14.55
	Emotion	87	66.42	17.04
	Gender	87	75.33	14.64

Table 3.4. *Key to valence specific shifting and inhibition trials*

Code	t1/t2	t3
H_H	Happy	Happy
H_A	Happy	Angry
A_A	Angry	Angry
A_H	Angry	Happy

Table 3.5. *Means and Standard Deviations (SD) for Inhibition Composite scores*

Inhibition Composite	N	Mean	SD
Happy to Happy	87	29.58	189.79
Happy to Angry	87	-9.34	222.98
Angry to Happy	87	56.00	239.58
Angry to Angry	87	2.74	201.90

Table 3.6. *Means and Standard Deviations (SD) for Inhibition to Emotion Composites*

Inhibition Variable	N	Mean	SD
Happy to Happy	84	4.15	525.94
Happy to Angry	80	-56.22	484.06
Angry to Happy	78	113.1	521.89
Angry to Angry	76	-37.28	384.2

Table 3.7. *Means and Standard Deviations (SD) for Inhibition to Gender Composites by valence*

Inhibition Variable	N	Mean	SD
Happy to Happy	83	-11.03	526.98
Happy to Angry	83	-32.19	534.66
Angry to Happy	85	54.79	504.26
Angry to Angry	81	118.82	604.34

Table 3.8. *Repeated measures ANOVA for Shifting to Emotion: trial 1 x trial 3 valence*

Source	df	SS	MS	*F*
Trial 2 Emotion	(1, 81)	157,369	157,369	1.01
Trial 3 Emotion	(1, 81)	1,825,200	1,825,200	11.46**
T2 Emotion * T3 Emotion	(1, 81)	109,930	109,930	0.62

Significance: ***p* < .01*

Table 3.9. *Means and Standard Deviations (SD) for Set shifting composite - Gender to Emotion*

Set shifting composite by valence	N	Mean	Std. Dev.
Happy to Happy	85	65.52	429.95
Happy to Angry	84	-39.47	442.92
Angry to Angry	86	55.23	343.3
Angry to Happy	85	-128.17	452.52

Table 3.10. *Means and Standard Deviations (SD) for Set shifting composite - Emotion to Gender*

Set shifting composite by valence	N	Mean	SD
Happy to Happy	86	0.69	390.04
Happy to Angry	87	52.67	412.73
Angry to Angry	87	-68.27	341.89
Angry to Happy	86	17.13	367.73

Table 3.11. *Layers of set-shifting and inhibition variables*

Cue level *Affective or Neutral*	Valence *Happy or Angry*
Set shifting	
1. Emotion to gender	H_H, H_A, A_H, A_A
2. Gender to emotion	H_H, H_A, A_H, A_A
3. Both combined	H_H, H_A, A_H, A_A
Inhibition	
4. Emotion	H_H, H_A, A_H, A_A
5. Gender	H_H, H_A, A_H, A_A

Table 3.12. *Regression 1.1a IV. Shifting – Gender to Emotion. DV. Self-report anger after MTPT2.*

		β	*t*	R^2	$R^2\Delta$	*F*	df
Step 1.				0.12	0.12**	5.11**	(2,75)
	Baseline Anger	0.31	2.78**				
	Working Memory	-0.13	-1.14				
Step 2.				0.22	0.10	3.26**	(6,75)
	Baseline Anger	0.31	2.78*				
	Working Memory	-0.14	-1.28				
	Shifting (to Emotion) H_H	0.15	1.36				
	Shifting (to Emotion) H_A	0.24	2.13*				
	Shifting (to Emotion) A_A	-0.23	-2.10*				
	Shifting (to Emotion) A_H	0.10	0.87				

* $p < .05$, ** $p < .01$

Table 3.13. *Zero-order correlations for Regression 1.1a. IV. Shifting – Gender to Emotion. DV. Self-report anger after MTPT2.*

	1	2	3	4	5	6
1. Anger - post-MTPT2						
2. Anger - baseline	.33					
3. Working Memory	-.17	-.15				
4, Shifting (to Emotion) H_H	.04	-.21	.001			
5. Shifting (to Emotion) H_A	.21	.13	.04	.02		
6. Shifting (to Emotion) A_A	-.16	-.03	-.04	.23	.19	
7. Shifting (to Emotion) A_H	.02	-.12	-.08	-.04	-.19	-.01

*$p < .05$, **$p < .01$

Table 3.14. *Regression 1.1b. IV. Shifting – Emotion to Gender. DV. Self-report anger on MTPT2*

	β	*t*	R^2	$R^2\Delta$	*F*	df
Step 1.			0.13	0.13**	5.74**	(2,79)
Baseline Anger	0.32	2.94**				
Working Memory	-0.13	-1.21				
Step 2.			0.20	0.07	3.05**	(6,79)
Baseline Anger	0.31	2.87**				
Working Memory	-0.12	-1.10				
Shifting (to Gender) H_H	-0.02	-0.19				
Shifting (to Gender) H_A	-0.07	-0.63				
Shifting (to Gender) A_A	-0.22	-0.20				
Shifting (to Gender) A_H	0.24	2.23*				

Significance: *$p < .05$, **$p < .01$

Table 3.15. *Zero-order correlations for Regression 1.1b. IV. Shifting – Emotion to Gender. DV. Self-report anger after MTPT2.*

	1	2	3	4	5	6
1. Anger - post-MTPT2						
2. Anger - baseline	.34***					
3. Working Memory	-.18~	-.15				
4. Shifting (to Gender) H_H	-.08	-.17	-.13			
5. Shifting (to Gender) H_A	-.12	.02	.09	.13		
6. Shifting (to Gender) A_A	-.04	.10	.08	-.01	.18~	
7. Shifting (to Gender) A_H	.27**	.02	-.03	-.05	-.18~	-.11

Significance: ~p<0.06, *$p < .05$, **p < .01, ***$p < .001$

Table 3.16. *Regression 1.2a. IV. Shifting – Gender to Emotion. DV. Heart rate.*

		β	*t*	R^2	$R^2\Delta$	*F*	df
Step 1.				0.01	0.01	0.31	(2,73)
	Baseline Anger	-0.02	-0.19				
	Working Memory	-0.094	-0.78				
Step 2.				0.11	0.11	1.43	(6,73)
	Baseline Anger	-0.06	-0.52				
	Working Memory	-0.09	-0.75				
	Shifting Composite H_H	-0.30	-2.44*				
	Shifting Composite H_A	-0.04	-0.28				
	Shifting Composite A_A	0.06	0.46				
	Shifting Composite A_H	0.13	1.12				

Significance: *$p < .05$

Table 3.17. *Zero-order correlations for Regression 1.2a. IV. Shifting – Gender to Emotion. DV. Heart rate.*

	1	2	3	4	5	6
1. Heart rate						
2. Baseline Anger	-.01					
3. Working Memory	-.09	-.16				
4. Shifting (to Gender) H_H	-.28**	-.22*	-.01			
5. Shifting (to Gender) H_A	-.08	.14	.05	.04		
6. Shifting (to Gender) A_A	-.02	-.02	-.05	.25*	.23*	
7. Shifting (to Gender) A_H	.16	-.12	-.07	-.03	-.22*	-.01

Significance: *$p < .05$, **$p < .01$,

Table 3.18. *Regression 1.2b. IV. Inhibition – both Gender and Emotion. DV. Heart rate.*

		β	*t*	R^2	$R^2\Delta$	*F*	df
Step 1.				0.002	0.002	0.09	(2,78)
	Baseline Anger	0.02	0.20				
	Working Memory	-0.04	-0.33				
Step 2.				0.08	0.80	1.07	(6,78)
	Baseline Anger	0.01	0.11				
	Working Memory	-0.09	-0.77				
	Inhibition (both) H_H	0.08	0.62				
	Inhibition (both) H_A	-0.27	-2.24*				
	Inhibition (both) A_H	0.02	0.18				
	Inhibition (both) A_A	0.13	1.05				

Significance: * $p < .05$

Table 3.19. *Zero-order correlations for Regression 1.2b. IV. Inhibition – both Gender and Emotion. DV. Heart rate.*

	1	2	3	4	5	6
1. Heart rate						
2. Baseline Anger	.03					
3. Working Memory	-.04	-.19*				
4. Inhibition (both) H_H	.10	.05	.25**			
5. Inhibition (both) H_A	-.23*	-.13	-.11	-.13		
6. Inhibition (both) A_H	.04	.04	.07	.27**	-.01	
7. Inhibition (both) A_A	.05	-.30**	.03	.06	.31**	-.02

Significance: *$p < .05$, **$p < .01$

Table 3.20. *Regression 1.2c. IV. Shifting – Emotion to Gender. DV. Heart rate.*

	β	*t*	R^2	$R^2\Delta$	*F*	df
Step 1.			0.002	0.002	0.07	(2,77)
Baseline Anger	-.03	-0.24				
Working Memory	-.04	-0.31				
Step 2.			0.097	0.096	1.28	(6,77)
Baseline Anger	-.10	-0.83				
Working Memory	-.09	-0.79				
Shifting (to Gender) H_H	-.12	-1.05				
Shifting (to Gender) H_A	.01	0.10				
Shifting (to Gender) A_A	.29	2.49**				
Shifting (to Gender) A_H	.09	0.77				

Significance: **$p < .01$

Table 3.21. *Zero-order correlations for Regression 1.2c. IV. Shifting – Emotion to Gender. DV. Heart rate.*

	1	2	3	4	5	6
1. Heart rate						
2. Baseline Anger	-.02					
3. Working Memory	-.03	-.16				
4. Shifting (to Gender) H_H	-.09	-.17	-.14			
5. Shifting (to Gender) H_A	.03	.01	.08	.12		
6. Shifting (to Gender) A_A	.26**	.12	.10	-.01	.20*	
7. Shifting (to Gender) A_H	.04	.03	-.01	-.001	-.18	-.15

Significance: *$p < .05$, **$p < .01$

Table 3.22. *Regression 2.1a. IV. Shifting – Gender to Emotion. DV. Errors per Second (MTPT2).*

		β	*t*	R^2	$R^2\Delta$	*F*	df
Step 1.				0.59	0.59***	31.93***	(3,69)
	Anger - post MIP	-0.08	-1.03				
	Working Memory	-0.17	-2.18**				
	Errors per Second	0.76	9.59***				
Step 2.				0.64	0.05	15.81***	(7,69)
	Anger - post MIP	-0.09	-1.19				
	Working Memory	-0.18	-2.29**				
	Errors per Second	0.77	10.07***				
	Shifting (to Emot) H_H	-0.21	-2.61**				
	Shifting (to Emot) H_A	0.09	1.12				
	Shifting (to Emot) A_A	-0.01	-0.09				
	Shifting (to Emot) A_H	0.01	0.13				

Significance: ** $p < .01$. *** $p < .001$

Table 3.23. *Zero-order correlations for Regression 2.1a. IV. Shifting – Gender to Emotion. DV. Errors per Second (MTPT2).*

	1	2	3	4	5	6	7
1. Errors per Second - MTPT2							
2. Anger - post MIP	-.10						
3. Working Memory	-.15	-.14					
4. Errors per Second - MTPT1	.75***	.06	.01				
5. Shifting (to Emotion) H_H	-.14	.03	.002	.08			
6. Shifting (to Emotion) H_A	.01	.17	.03	-.05	.06		
7. Shifting (to Emotion) A_A	-.09	-.02	-.09	-.08	.27**	.21*	
8. Shifting (to Emotion) A_H	.05	-.03	-.07	.06	-.01	-.20*	.02

Significance: *$p < .05$, **$p < .01$, ***$p < .001$

Table 3.24. *Regression 2.1b. IV. Inhibition – Emotion. DV. Errors per Second (MTPT2)*

	β	*t*	R^2	$R^2\Delta$	*F*	df
Step 1.			0.56	0.56***	22.44***	(3,55)
Anger - post MIP	-.09	-0.97				
Working Memory	-.18	-1.94***				
Errors per Second	.71	7.63***				
Step 2.			0.65	.08*	12.65***	(7,55)
Anger - post MIP	-.09	-0.97				
Working Memory	-.21	-2.28**				
Errors per Second	.75	8.48***				
Inhibition (to Emot) H_H	-.05	-0.51				
Inhibition (to Emot) H_A	-.15	-1.63				
Inhibition (to Emot) A_A	-.10	-1.11				
Inhibition (to Emot) A_H	.22	2.38*				

Significance: *$p < .05$, **$p < .01$, ***$p < .001$

Table 3.25. *Zero-order correlations for Regression 2.1b. IV. Inhibition – Emotion. DV. Errors per Second (MTPT2)*

	1	2	3	4	5	6	7
1. Errors per Second - MTPT2							
2. Anger - post MIP	-.04						
3. Working Memory	-.26*	-.15					
4. Errors per Second - MTPT1	.73***	.03	-.14				
5. Inhibition (to Emotion) H_H	-.09	-.01	.23*	-.04			
6. Inhibition (to Emotion) H_A	-.03	.11	-.13	.21	.06		
7. Inhibition (to Emotion) A_H	.18	.07	.06	-.02	.27*	-.13	
8. Inhibition (to Emotion) A_A	.06	.08	-.12	.18	.14	.26*	.23*

Significance: *$p < .05$, **$p < .01$, ***$p < .001$

Table 3.26. *Regression 3.1a. IV. Heart rate. DV. Total time (MTPT2)*

	β	*t*	R^2	$R^2\Delta$	*F*	df
Step 1.			0.653	0.65***	137.43***	(1,74)
Total time - MTPT1	0.81	11.72***				
Step 2.			0.673	0.20*	74.09***	(2,74)
Total time - MTPT1	0.79	11.71***				
Heart Rate	-0.14	-2.09*				

Significance: *$p < .05$, ***$p < .001$

Table 3.27. *Zero-order correlations for Regression 3.1a*

	1	2
1. Total time - MTPT2		
2. Total time - MTPT1	.81***	
3. Heart Rate	-.22**	-.10

Significance: **$p < .01$, ***$p < .001$

Table 3.28. *Regression 3.1b. IV. RSA. DV. Total time (MTPT2)*

		β	*t*	R^2	$R^2\Delta$	*F*	df
Step 1.				0.674	0.674***	74.56***	(2,74)
	Total time - MTPT1	0.15	2.17*				
	Respiration	0.773	11.18***				
Step 2.				0.676	0.001	49.31***	(3,74)
	Total time - MTPT1	0.15	2.21*				
	Respiration	0.77	11.11***				
	RSA	0.04	0.53				

Significance: *$p < .05$, ***$p < .001$

Table 3.29. *Zero-order correlations for Regression 3.1b*

	1	2	3
1. Total time – MTPT2			
2. Respiration	.33**		
3. Total time – MTPT1	81***	.23*	
4. RSA	.01	-.11	-.01

Significance: * $p < .05$, ** $p < .01$, *** $p < .001$

Table 3.30. *Regression 3.2. IV. Heart rate. DV. Errors per Second (MTPT2)*

		β	*t*	R^2	$R^2\Delta$	*F*	df
Step 1.				0.29	0.29***	13.99***	(2,71)
	Errors per Second - MTPT1	0.53	5.17***				
	Anger - baseline	-0.01	-0.14				
Step 2.				0.34	.049*	11.58***	(3,71)
	Errors per Second - MTPT1	0.55	5.49***				
	Anger - baseline	-.003	-0.023				
	Heart rate	-.22	-2.25*				

Significance: *$p < .05$, ***$p < .001$

Table 3.31. *Zero-order correlations for Regression 3.1c.Regression 3.1c. IV. Heart rate. DV. Errors per Second (MTPT2)*

	1	2	3
1. Errors per Second - MTPT2			
4. Errors per Second - MTPT1	.54***		
2. Anger - baseline	-.11	-.19	
4. Heart rate	-.18	.08	.04

Significance: ~$p < 0.07$ ***$p < .001$

Table 4.1. *Conceptual sets for valence and hypotheses - Outline of discussion.*

I. Happy to Angry *(consistent with hypotheses)*	II. Angry to Angry *50/50*	III. Angry to Happy *50/50*	IV. Happy to Happy *(not consistent with hypotheses)*
Better Inhibition H_A *Lower HR*	Better Shifting A_A *Lower HR*	Better Shifting A_H *Lower Anger*	Better Shifting H_H *Higher HR*
Better Shifting H_A *Lower Anger*	Better Shifting A_A *Higher Anger*	Better Inhibition A_H *Higher Errors*	Better Shifting H_H *Higher Errors*

Table D.1. *Means and Standard Errors (SE) for cue and trial type*

Cue	Mean	Std. Error
Color	1036.13	27.36
Emotion	2152.37	25.01
Gender	1725.56	27.71

Table D.2. *Means and Standard Errors (SE) for cue and trial type*

Trial type	Mean	Std. Error
Control	1639.84	23.48
Inhibitory	1683.50	24.12
Repeat	1614.74	23.35
Unclassified	1614.00	24.14

Table D.3. *Means and Standard Errors (SE) for the interaction between cue * trial type*

Cue	Trial type	Mean	SE	Trial type	Mean	SE	Trial type	Mean	SE
Color				Emotion			Gender		
	Control	1046.57	31.74	Control	2148.16	25.60	Control	1724.79	30.59
	Inhibit	1091.51	31.95	Inhibit	2186.74	27.35	Inhibit	1772.26	29.51
	Repeat	974.86	24.82	Repeat	2152.43	29.59	Repeat	1716.94	30.08
	Unclass	1031.59	29.57	Unclass	2122.16	30.37	Unclass	1688.25	29.43

Table D.4. *Inhibition Composites: Variables included, mean composite score and SD*

Composite	Variables included in Composite	Mean	SD
Total	All inhibition and control trials	21.82	123.31
Color	All inhibition and control trials to Color cue	44.95	184.86
Emotion	All inhibition and control trials to Emotion cue	38.59	196.64
Gender	All inhibition and control trials to Gender cue	47.47	184.15
Angry	All cued dimensions and trial types – Valence of the correct picture on the last trial was Angry.	-2.74	173.3
Happy	All cued dimensions and trial types – Valence of the correct picture on the last trial was Happy.	44.72	172.18

Table D.5. *Shifting Composites: Variables included, mean composite score and SD*

Composite	Variables included in Composite	Mean	SD
Total	All control, unclassified, repeat trials	41.83	110.52
Color	All control, unclassified, repeat trials to Color cue	64.22	175.91
Emotion	All control, unclassified, repeat trials to Emotion cue	-17.27	171.25
Gender	All control, unclassified, repeat trials to Gender cue	-10.42	143.04
Angry	All cued dimensions and trial types – Valence of the correct picture on the last trial was Angry.	36.99	135.03
Happy	All cued dimensions and trial types – Valence of the correct picture on the last trial was Happy	48.39	153.23

Table D.6. *Valence specific Shifting Composites: Variables included, mean composite score and SD*

	Variables included in Composite			
Shifting Composite	Trial 2	Trial 3	Mean	SD
Emotion to Gender	Emotion	Gender	-4.91	159.33
Gender to Emotion	Gender	Emotion	-19.03	200.51
Gender to Emotion-Angry	Gender	Emotion - Angry	-32.87	235.71
Gender to Emotion-Happy	Emotion	Emotion - Happy	-5.19	268.03
Emotion to Color	Color	Color	790.72	218.79
Color to Emotion	Color	Emotion	-2.89	170.9
Color to Emotion-Angry	Color	Emotion - Angry	-28.18	211.54
Color to Emotion-Happy	Color	Emotion - Happy	23.13	248.09

Table D.7. *Repeat and shifting trials to Emotion cues by valence*

Valence	Repeat trials		Shifting trials	
	Mean	SD	Mean	SD
Happy to Happy	2099.46	336.45	2173.26	386.68
Happy to Angry	2179.35	332.37	2140.58	432.18
Angry to Happy	2220.16	329.30	2100.95	390.12
Angry to Angry	2073.26	306.83	2139.87	333.81

Table D.8. *Repeat and shifting trials to Gender cues by valence*

Valence	Repeat trials		Shifting trials	
	Mean	SD	Mean	SD
Happy to Happy	1657.41	316.57	1658.10	414.50
Happy to Angry	1748.77	336.71	1799.12	450.03
Angry to Happy	1679.09	324.68	1696.22	373.37
Angry to Angry	1753.15	326.75	1690.83	355.69

Appendix A: Affect Rating Survey

We would now like to ask you a few questions. Please answer all the questions below using the following scale:

1	*2*	*3*	*4*	*5*
not at all	*a little*	*moderately*	*quite a bit*	*extremely*

How strongly do you currently feel:

____ curious	____ angry
____ sad	____ anxious
____ thoughtful	____ alert
____ irritable	____ happy
____ focused	____ tired
____ cheerful	____ frustrated
____ down	____ depressed
____ relaxed	____ worried
____ up	____ grouchy
____ confused	____ energetic
____ active	____ pleased
____ distracted	____ gloomy

Appendix B: Emotion Regulation Questionnaire

1. Did the **first** mirror tracing task make you feel frustrated?

1	2	3	4	5	6	7
Not at all	Very little		A medium amount		A lot	Quite a lot

2. Did you press Enter to terminate the first task before it ended? YES NO
3. If yes, why did you decide to terminate the **first** mirror tracing task?

_____
_____
_____
_____

4. How much anger did you experience in the **first** mirror tracing task?

1	2	3	4	5	6	7
No anger at all	Very little anger		Medium anger		A lot of anger	The most anger

5. How well were you able to concentrate on the **first** mirror tracing task?

1	2	3	4	5	6	7
Could not concentrate at all	Could not concentrate well		Medium concentration		Could concentrate well	Could concentrate very well

6. Did the **second** mirror tracing task make you feel frustrated?

1	2	3	4	5	6	7
Not at all	Very little		A medium amount		A lot	Quite a lot

7. Did you press Enter to terminate the second task before it ended? YES NO
8. If yes, why did you decide to terminate the **second** mirror tracing task?

_____
_____

9. How much anger did you experience in the **second** mirror tracing task?

1	2	3	4	5	6	7
No anger at all	Very little anger		Medium anger		A lot of anger	The most anger

10. How well were you able to concentrate on the **second** mirror tracing task?

1	2	3	4	5	6	7
Could not concentrate at all	Could not concentrate well		Medium concentration		Could concentrate well	Could concentrate very well

11. Do you think that feeling angry helped your accuracy on the **second** mirror tracing task

1	2	3	4	5	6	7
Not at all	Very little		A medium amount		A lot	Quite a lot

12. Do you think that feeling angry hurt your accuracy on the **second** mirror tracing task

1	2	3	4	5	6	7
Not at all	Very little		A medium amount		A lot	Quite a lot

13. Do you think that feeling angry motivated you to work longer on the **second** mirror tracing task

1	2	3	4	5	6	7
Not at all	Very little		A medium amount		A lot	Quite a lot

14. Do you think that feeling angry motivated you stop sooner on the **second** mirror tracing task

1	2	3	4	5	6	7
Not at all	Very little		A medium amount		A lot	Quite a lot

15. Do you think that feeling angry distracted you during the **second** mirror tracing task

1	2	3	4	5	6	7
Not at all	Very little		A medium amount		A lot	Quite a lot

16. Did you use any strategies (e.g., channel anger to keep trying, try not to think about the angry memory) to keep working on the **second** mirror tracing task? YES NO

17. If yes, please describe _____

_____

_____

_____

_____

Appendix C: Trial types and measures for executive function

Trial types	Cue trial (trial 1)	Cue trial (trial 2)	Cue trial (trial 3)
Trials requiring a set shift			
Inhibitory trials (a-b-a)	emotion	gender or color	emotion
	gender	emotion or color	gender
	color	emotion or gender	color
Control trials (c-b-a)	gender (color)	color (gender)	emotion
	color (emotion)	emotion (color)	gender
	gender (emotion)	emotion (gender)	color
Unclassified trials (b-b-a)	gender (color)	gender (color)	emotion
	emotion (color)	emotion (color)	gender
	emotion (gender	emotion (gender)	color
Trials without a set shift			
Repeat trials (a-a)		emotion	emotion
		gender	gender
		color	color

Measuring of executive function

Inhibition

Indexed: RT to Inhibitory trials - RT to control trials

Interpretation: High inhibition scores reflect good executive ability

Set shifting costs

Indexed: Average of RT to non-inhibitory switch trials – repetition trials

[RT to Control + RT to Unclassified]/2 - RT to Repeat trials

Interpretation: Smaller difference is associated with better executive function

Table adapted from De Lissnyder et al., 2010

Appendix D: Preliminary Results

Overall inhibition and shifting results. I conducted 3 (cue: Emotion, Gender, Color) x 4 (trial type: inhibitory, control, unclassified, repeat) repeated measures ANOVA to assess for overall task performance. Analyses revealed a main effect of cue, $F(2, 172) = 1088.35$, $p < .001$. Response on the cued dimension Emotion was significantly slower than response on the cued dimension of Gender ($t(86) = 24.09$, $p < .001$) and Color ($t(86) = 42.57$, $p < .001$). Response on the cued dimension of Gender was significantly slower than cued dimension of Color ($t(86) = 27.45$, $p < .001$). See Table D.1 for means and standard errors.

Results indicated a main effect of trial type, $F(3, 258) = 17.50$, $p < .001$ (see Table D.2). Response on inhibitory trials were significantly slower than unclassified ($t(86) = 3.89$, $p < .001$) and repeat trials ($t(86) = 5.81$, $p < .001$). Inhibitory trials were slower than control trials, but this difference was not significant ($t(86) = 1.65$, *ns*). Unclassified trials were significantly faster than control trials, $t(86) = 2.45$, $p = .016$. Repeat trials were faster than control trials ($t(86) = 4.24$, $p < .001$) and unclassified trials ($t(86) = 2.16$, $p < .05$). The main effects of cue and trial type present a similar pattern to that found by De Lissnyder et al. (2010).

Results showed a significant interaction of cue by trial type, $F(6, 516) = 2.65$, $p < .02$ (see Table D.3). For the cued dimension of Color, repeat trials yielded the fastest reaction time followed by unclassified then control trials. Repeat trials were faster than unclassified trials ($t(86) = 2.93$, $p < .01$) control trials ($t(86) = 3.38$, $p < .001$) and inhibitory trials ($t(86) = 6.28$, $p < .001$). Results for the cued dimension of Color are similar to results found by De Lissnyder et al. (2010). For the cued dimension of Gender,

unclassified trials were fastest followed by repeat, then control trials. Unclassified trials were faster than control trials ($t(86) = 2.23$, $p < .03$) and inhibitory trials ($t(86) = 4.73$, $p < .001$). Inhibitory trials were slower than control trials, ($t(86) = 2.40$, $p < .02$) and repeat trials ($t(86) = 2.62$, $p < .01$). For the Emotion dimension, unclassified trials were fastest followed by control, then repeat trials. Inhibitory trials were slower than unclassified trials, ($t(86) = 2.85$, $p < .01$). Inhibitory trials were the slowest for all three cued dimensions. Results for the cued dimensions of Gender and Emotion are different than results found by De Lissnyder et al. (2010) because the repeat trials were not the fastest trial type.

Inhibition

Inhibition – Trial type. To examine performance on trial types to be included in the Inhibition composite I conducted a 3 (cue: Emotion, Gender, Color) by 2 (trial type: inhibitory, control) repeated measures ANOVA. Results indicated a main effect of cue, $F(2,172) = 863.79$, $p < .001$. Emotion trials were slower than Gender trials ($t(86) = 24.09$, $p < .001$). Both Emotion ($t(86) = 42.57$, $p < .001$) and Gender ($t(86) = 27.45$, $p < .001$) trials were slower than Color trials. Analyses revealed a main effect of trial type, $F(1,86) = 14.49$, $p < .001$; inhibitory trials were significantly slower than control trials. That inhibitory trials were slower than control trials is imperative for creating an Inhibition composite that reflects the cost of inhibiting a previous stimuli. Follow up paired sample t-tests indicate that inhibitory trials are significantly slower than control trials for the cued dimension of Color ($t(86) = 2.27$, $p < .05$) and Gender ($t(86) = 2.40$, $p < .02$). This similar relationship for the cued dimension of Emotion trends towards significance. There was not a significant interaction between cue and trial type.

Inhibition - Composite. Inhibition composites were computed as the difference between reaction times to inhibitory and control trials. Taking longer to respond when a cue was just inhibited (i.e., getting over the initial inhibition) indicates better inhibition, a marker of better EF. Subtracting reaction time on control trials accounts for individual differences. Higher Inhibition scores indicate better EF (see Table D.4).

Paired sample *t*-tests showed no significant differences between the all Inhibition composites. However, the differences between Total and Angry ($t(86) = 1.92$, $p = .058$), Total and Happy, ($t(86) = 1.73$, $p = .08$) and between Angry and Happy ($t(86) = 1.84$, $p = .07$) approached significance.

Shifting

Shifting – Trial type. I ran a 3 (cue: Emotion, Gender, Color) by 3 (trial type: control, repeat, unclassified) repeated measures ANOVA to obtain an index of trials included for the set-shifting composite. Results showed a main effect of cue, $F(2,172) = 1037.86$, $p < .001$. Follow up t-tests indicate that mean reaction time to Emotion cues is slower than Gender cues ($t(86) = 24.09$, $p < .001$), and both Emotion ($t(86) = 42.57$, $p < .001$) and Gender ($t(86) = 27.45$, $p < .001$) are slower than Color cues. Results showed a main effect of trial type, $F(2, 172) = 4.03$, $p < .02$. Control trials are faster than repeat trials ($t(86) = 4.24$, $p < .01$) and unclassified trials ($t(86) = 2.45$, $p < .02$). Repeat trials are faster than unclassified trials ($t(86) = 2.16$, $p < .05$).

Results showed an interaction effect, $F(4,344) = 3.72$, $p < .01$. For the cued dimension of Color, repeat trials are faster than control and unclassified trials. For cued dimension of Gender, repeat trials are faster than control trials but slower than unclassified trials. For cued dimension of Emotion, repeat trials are slower than both

control and unclassified trials. The cued dimension of Color follows the same results pattern as De Lissnyder et al (2010). For the cued dimensions of Gender and Emotion, repeat trials were not the fastest trials, which goes against expectations.

Shifting – Composites. Shifting composites were calculated by the difference between the average of shifting trials (control, unclassified) and repeat trials. Smaller shifting scores indicate better executive ability (see Table D.5). I examined differences in shifting composites to the three cued dimensions (Color, Emotion, Gender). Paired sample *t*-tests indicate a significant difference when comparing Color with the cued dimensions of Emotion ($t(86) = 2.89$, $p < .01$) and Gender ($t(86) = 2.95$, $p < .01$). Color showed significantly larger shifting score than Emotion and Gender cues. There was no difference between Emotion and Gender cues ($t(86) = 0.298$, *ns*).

Shifting – Affective composites. To examine ability to shift to and from emotional and non-emotional material I created shifting composites that included control and unclassified trials that shift from Emotion (trial 2: Angry, Happy) to non-affective cues (Gender/Color; trial 3), and vice versa (see Table D.6). Paired sample *t*-tests were conducted to examine differences between cued dimensions. Results indicated that there was not a significant difference when shifting from Gender to Emotion versus Emotion to Gender ($t(86) = 0.55$, *ns*). Furthermore, there was not a significant difference when shifting from Gender to Emotion (Angry), or from Gender to Emotion (Happy; $t(86) = 0.84$, *ns*). There was a significant difference between shifting from Emotion to Color versus Color to Emotion ($t(86) = 26.56$, $p < .001$). There was no difference when shifting from Color to Emotion (Angry) versus Emotion (Happy; $t(86) = 1.55$, *ns*).

Set shifting – Gender to Emotion. To examine the ability to shift from a non-affective cue (Gender) to an affective cue (Emotion) I included trials where the second to last cued dimension was Gender and the last cued dimension was Emotion[2]. Examining valence is particularly important for the repeat variables. Half of the repeat trials did not require a valence change (H_H, A_A), half of the repeat trials did require a valence change (H_A, A_H). A required change in valence ostensibly created another shift (via the valence change) within the repeat trials. I took the mean of unclassified and control trials to create an overall shifting trial (see Table D.7 for means).

To examine whether there were RT differences for trial type due to valence, I ran a 2 (trial type: repeat, shifting) x 4 (valence: H_H, H_A, A_A, A_H) repeated measures ANOVA. Results indicate no main effect of valence, $F(3,243) = 1.39$, *ns*, or of trial type, $F(1,81) = 0.35$, *ns*. There was an interaction effect, $F(3, 243) = 4.26$, $p < .01$.

For repeat trials, valence order H_H was fastest, followed by A_A, A_H, and H_A (see Table D.7). Repeat trials H_H was significantly faster than A_H ($t(85) = 3.76$, $p < .001$), and H_A ($t(85) = 2.91$, $p < .01$), but was not significantly faster than A_A ($t(85) = 0.43$, *ns*). It is important to note that trials that did not require a valence switch (e.g., H_H) were faster than trials that required a valence switch (e.g., H_A). For shifting trials, the order of valence (from fastest to slowest) was A_H, A_A, H_A, H_H. Paired sample *t*-tests indicated that there were no significant differences between these valences for shifting trials to Emotion.

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[2] Restricting the last cued dimension to emotion, and the second to last to emotion and gender limits the number of trials included in creating means (mean number of trials included=62.74, 19% of trials excluded for low accuracy and RT in excess of 3500 ms), which is reflected in the degrees of freedom.

Set shifting – Emotion to Gender. To examine the ability to switch away from an affective cue (Emotion) to a neutral cue (Gender) I included trials where the cued dimension at t2 is Emotion and the cued dimension at t3 is Gender. I took the mean of control and unclassified trials to create an overall shifting trial (see Table D.8 for means). To examine whether there were reaction time differences for trial type due to valence, I ran a 2 (trial type: repeat, shifting) x 4 (valence: H_H, H_A, A_A, A_H) repeated measures ANOVA. Results indicate a main effect of valence, $F(3,255) = 6.22$, $p < .001$. From fastest to slowest, valence reaction times fell in the order of H_H, A_H, A_A, H_A (see Table D.8). There was no main effect of trial type, ($F(1, 85) = 0.005$, *ns*) and there was no interaction effect, $F(3, 255) = 1.35$, *ns*.

To compare the effect of valence switch on reaction time for repeat trials, I conducted paired sample *t*-tests. H_H was significantly faster than H_A ($t(86) = 3.21$, $p < .01$) and A_A ($t(86) = 3.46$, $p < .001$). A_H was significantly faster than A_A ($t(86) = 2.45$, $p < .02$) and H_A ($t(86)=2.21$, $p < .03$). To compare the effect of valence switch on reaction time for shifting trials, I conducted paired sample *t*-tests. H_H was significantly faster than H_A ($t(86) = 2.61$, $p < .02$), and A_A was faster than H_A ($t(86) = 2.17$, $p < .05$)

Appendix E. Clinical Implications: TBI

An enhanced understanding of the cognitive regulation of anger, may prove particularly beneficial in clinical treatment of individuals who have experienced TBI, in particular, repeated mild TBI (mTBI; see appendix for criteria).

TBI, and its deleterious short and long-term effects, is becoming more widely recognized as a public health problem. TBI is becoming increasingly more prevalent in the general population, as well as military and athlete populations. The effects of TBI are pervasive and can be devastating. Additionally, TBI in football, at the levels of high school, college, and the NFL, has received a recent surge of media attention putting this important topic at the forefront of the minds of athletes, clinicians, researchers, and funding agencies.

TBI of all severities has been shown to cause focal damage in specific areas of the brain, as well as leading to impaired connectivity between brain regions. In particular, the frontal and anterior temporal lobes are susceptible to contusion based on their position within the skull (Kraus et al., 2007). Disruption of function is also caused by diffuse damage to white tracts due to their susceptibility to shearing forces during TBI (Kraus et al., 2007). Structural and axonal damage is associated with many of the cognitive and neuropsychological sequelae of TBI. These deficits include disruption of EFs (based in the frontal lobes; Levin &Hanten, 2005), which not only can lead to problems with reasoning and problem solving, but can also hinder top down regulation of pre-potent emotional and behavioral responses such as anger and aggression (frontal limbic connectivity; Williams et al., 2009). While mTBI is associated with minimal decline in IQ, which allows for a normal presentation following the injury, the slight decline in EF

caused by the injury may lead to difficulties with behavioral disinhibition and modulation of emotional responses. For example, the effects of TBI may become clear as patients show difficulties facing daily challenges and have a hard time coping with stressors (Williams et al., 2009). This taps directly into this studies examination of EF with affective stimuli, an angry mood induction, and use of a frustrating task.

Recovery from mTBI is usually rapid with acute symptoms resolving in hours and most individuals reporting being symptom free in 10 days. However, some individuals show more persistent symptoms, which are captured as a part of the postconcussive syndrome (PCS). PCS is composed of psychiatric, cognitive, and somatic complaints such as depression, anxiety, irritability, and difficulty concentrating (Williams et al., 2009; Williams, Potter, & Ryland, 2010). One theory of PCS is that it is an integration of both structural and axonal damage resulting from the TBI and a predisposition for psychological symptoms. In support of this third option, the authors state that "while MTBI may set the conditions for PCS to occur, there does appear to be a role for psychological mechanisms in persistence of symptoms" (Williams et al., 2010). A predisposition for mood disorders may be exacerbated by TBI. Psychological symptoms may maintain PCS, as well as being exacerbated by TBI in the first place. Understanding the cognitive mechanisms associated with function and dysfunction of affect regulation can enhance our understanding of clinical disorders (e.g., Joormann & Gotlib, 2010) and inform clinical treatments. Additional information on how these cognitive mechanisms are disrupted by TBI is important for understanding the emotional disturbances following TBI (Williams et al., 2010) and for informing preventative and therapeutic measures.

Thus far, I have only discussed the influence of a single TBI. The long term effects of repeated TBIs are worse. One of the devastating consequences of chronic repeated TBI is chronic traumatic encephalopathy (CTE). CTE is found in individuals who have sustained repeated TBI's. Symptoms over time include decreased attention and memory, lack of judgment, poor insight, and overt dementia, as well as emotional symptoms of irritability, outbursts of aggressive behavior, cognitive decline, unsteadiness, headaches, slurred speech, and parkinsonism(McKee et al., 2009). The symptoms of CTE are described as insidious, progressive, and do not permit reversibility, even after retiring from contact sports. While researchers are able to identify characteristics associated with CTE, the only current means to diagnose CTE is through autopsy.

A seminal paper on CTE (McKee et al., 2009) focused on 51 neuropathologically confirmed cases of CTE in which 46 (90%) were athletes. Relevant features of this paper include the high level of mood disturbance (depression) and anger outbursts, as well as a number of suicidal fatalities. These findings are particularly relevant given the increase in high profile suicides of former high level athletes (primarily NFL, but also collegiate football). Researchers are currently working to better understand how to diagnose CTE while individuals are still alive, and may draw on neuropsychological measures to aid their search.

The results found in this study indicate that we have a long way to go before these ideas are applied to clinical populations, they do however start to identify the role of specific EFs in anger regulation.