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Personal Cognition and the Affect Regulation Process: Affect Reactivity, Affect Regulation Ability, and Responses to Cognitive Errors.

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PERSONAL COGNITION AND THE AFFECT REGULATION PROCESS: AFFECT REACTIVITY, AFFECT REGULATION ABILITY AND RESPONSES TO COGNITIVE ERRORS

by

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Abstract

A growing body of research examines relationships between cognitive tendencies and a number of personality and affective (i.e. emotional) traits. While several mechanisms have been suggested to explain these links, the exact reasons for the observed effects remain unclear in a number of circumstances. The current research examines the potential underlying mechanisms of observed links between cognitive error reactivity and various components of the affect regulation process; those individuals who make errors in strings on standard cognitive tasks are higher in trait negative affect, react more strongly to negative daily events, and may show deficits in self-regulation ability (Compton, Robinson, Ode, Quandt, Fineman, & Carp, 2008). The current study tests whether observed links between cognitive error reactivity and affective traits/processes are due to affect reactivity (Larsen, & Ketelaar, 1989) or affect regulation ability (Hemenover, Augustine, Shulman, Tran, & Barlett, 2008). Participants completed measures of both personality and error reactivity and then underwent an anxiety induction followed by one of three affect regulation tasks. Results reveal that neither affect reactivity (i.e., reaction to the anxiety induction) nor affect regulation ability (i.e., affective change due to the regulation task) adequately explain links between error reactivity and personality. The implications of these findings for both personality and cognitive psychology are discussed.

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Personal Cognition and the Affect Regulation Process: Affect Reactivity, Affect Regulation Ability, and Responses to Cognitive Errors.

Recent research reveals links between more stable and "cold" cognitive tendencies, such as working memory and error reactivity, and a number of personality and affective (i.e. emotional) traits (e.g., Schmeichel, Volokhov, & Demaree, 2008; Compton, Robinson, Ode, Quandt, Fineman, & Carp, 2008; Augustine & Larsen, in press). While several mechanisms have been suggested to explain these links, the exact reasons for the observed effects remain unclear in a number of circumstances. Of primary interest for the current research are the observed links between cognitive error reactivity and various components of the affect regulation process; those individuals who make errors in strings on standard cognitive tasks are higher in trait negative affect, react more strongly to negative daily events, and may show deficits in self-regulation ability (Compton, et al., 2008). The goal of the current research is to examine whether observed links between cognitive error reactivity and affective traits/processes are due to affect reactivity (Larsen, & Ketelaar, 1989) or affect regulation ability (Hemenover, Augustine, Shulman, Tran, & Barlett, 2008). First, I review the extant literature on the links between cognitive and personality variables. Second, I examine the function of affect reactivity and affect regulation ability with regards to the process of affect regulation. Third, I review individual differences in affect reactivity and the ways in which cognitive error reactivity may relate to the process of affect reactivity. Finally, I examine individual differences in affect regulation ability and the potential relationship between cognitive error reactivity to these individual differences.

Personal Cognition

Whereas social cognition examines the links between cognitive tendencies and social behavior, a growing body of research examines links between cognitive tendencies and personality processes, or personal cognition. The majority of the findings to date suggest that these relationships may be largely observed for those personality traits with an affective (i.e., emotional) component. This is consistent with established links between temporary affective states and cognition. The interplay between temporary affective states and cognition has received a massive amount of research attention across several distinct literature areas. Indeed, mounting evidence suggests that temporary affect and cognition are, in the least, dependent on one another (Storbeck & Clore, 2007), or at most, not treated differently by the brain (Duncan & Feldman Barrett, 2007). Given the wide links between affective states and cognition, it should not be surprising that affective traits are also related to cognition. Negative affect laden traits such as neuroticism, positive affect laden traits such as extraversion, and various components of the affect regulation process are related to a number of stable cognitive tendencies, such as judgment and decision making style (Hilbig, 2008; Hirsh, Morisano, & Peterson, 2008; Lauriola & Levin, 2001; Davis, Patte, Tweed, & Curtis, 2007; Augustine & Larsen, in press), working memory capacity (Hofmann, Gschwendner, Friese, Wiers, & Schmitt, 2008; Schmeichel, et al., 2008; Mikels, Reuter-Lorenz, Beyer, & Fredrickson, 2008), and error reactivity (Robinson, Meier, Wilkowski, & Ode, 2007; Robinson, Ode, Wilkowski, & Amodio, 2007; Compton et al., 2008; Augustine & Larsen, 2010). In this section I review the literature on personal cognition and describe the primary topic of the current research - error reactivity and trait affect.

Personality and Decision Making

As individuals seek to process their surroundings and make decisions, affect serves as a constant source of information, especially when deliberative thinking is either not possible or not appropriate. As Slovic and Peters (2006) stated, " Although analysis is certainly important in some decision-making circumstances, reliance on affect is generally a quicker, easier, and more efficient way to navigate in a complex, uncertain, and sometimes dangerous world (pp 322)." State affect leads to the use of certain heuristics and the exhibition of certain biases in a variety of decision making tasks. For instance, when estimating yearly accident fatalities, those experiencing positive affect provide higher estimates than those experiencing negative affect (Johnson & Tversky, 1983). In addition, those experiencing negative affect are more likely to reject offers in the ultimatum game (Harle & Sanfey, 2007). Finally, both naturally occurring and induced happiness predict selective attention to rewarding stimuli (Tamir & Robinson, 2007).

Just as state affect may serve as a temporary source of quick and (usually) efficient information, trait affect may serve as a constant source of information. In other words, trait affect may inform decision making in a stable and global manner. Recent evidence suggests that this is the case for four different decision making realms: heuristic usage, risky decision making, performance deficits, and susceptibility to irrelevant information.

First, affective traits are predictive of the degree to which individuals rely on certain heuristics when making decisions. Neuroticism, a negative affect laden trait, is predictive of the use of the recognition heuristic. When asked to estimate city population

size, those higher in neuroticism are more likely to base their judgments on their familiarity with the city, rather than actual population information (Hilbig, 2008). Affective traits are also predictive of the degree to which individuals choose smaller rewards today rather than larger rewards in the future, or temporal discounting (for a review of temporal discounting, see Frederick, Loewenstein, & O'Donoghue, 2002). Those high in extraversion or neuroticism and lower in cognitive ability show a tendency towards greater temporal discounting (Hirsh, et al., 2008). Second, neuroticism predicts the types of risks individuals are willing to take. Those higher in neuroticism are apt to make less risky decisions to achieve gains, but more risky decisions to avoid losses (Lauriola & Levin, 2001). Third, those higher in impulsivity and sensitivity to reward (positive affect laden traits) as well as sensitivity to punishment (a negative affect laden trait) show more performance deficits on the Iowa Gambling Task (Davis, et al., 2007). Finally, extraversion and neuroticism interact with the valence of primes seen before making probability judgments. Those higher in extraversion make higher probability judgments in response to positive primes while those higher in neuroticism make lower probability judgments in response to negative primes, even when state affect is controlled (Augustine & Larsen, in press).

In sum, trait affect may inform judgment and decision making at a broad and global level just as state or temporary affect informs judgment and decision making at a temporary level. Thus, a first link between stable affective traits and stable cognitive tendencies (i.e. decision making styles) is observed. However, evidence for the existence of personal cognition exists beyond the judgment and decision making literature. Working memory span is also related to stable affective tendencies.

Working Memory and Affect Regulation

Individuals differ in the degree to which they are able to regulate their behavior and affect. Several studies suggest that these differences in regulation ability may be due, in part, to differences in working memory capacity. Those with larger working memory capacities are better able to regulate their automatic behavioral and affective responses (Hofmann, et al., 2008). In other words, larger working memory capacities may facilitate self-regulation behavior. In line with this, working memory capacity predicts individuals' ability to engage in affect regulation, and these increased abilities lead to improved regulatory consequences (i.e. more positive and less negative affect, Schmeichel, et al., 2008).

Affective information takes up space in working memory and affect regulation may, in part, function by interrupting the maintenance of this information. With a larger working memory capacity, more cognitive resources would be available for the deployment of a regulation attempt, despite the resources occupied by affective information. In other words, those with smaller working memory capacities could have such a large portion of their cognitive resources depleted by affective experience itself, that no resources are available for regulation. Working memory may also possess a uniquely affective component. Any individual with deficits in the affective components of working memory may show a decreased ability to repair affect (Mikels, et al., 2008).

Regardless of any specific causal links between working memory capacity and self-regulation ability, these findings indicate yet another link between "cold" cognitive tendencies and affective traits (i.e., regulation ability). The final area of personal

cognition to be discussed herein concerns error reactivity, and it is this cognitive tendency that is directly relevant to the current research.

Error Reactivity

At a broad level, trait negative affect is related to cognitive control, such that those higher in negative affect show lessened cognitive control, or a lessened ability to inhibit dominant responses (i.e. attend to negative stimuli; Moriya & Tanno, 2008). The effects of this lack of control on affect regulation behavior can be observed by measuring individuals' reaction to errors. There are two primary ways of encapsulating cognitive error reactivity: post-error slowing and the tendency to make errors in strings.

In standard cognitive tasks (i.e., the Stroop task), some individuals show greater post-error slowing, or the tendency to slow down on the trial following a trial on which an error was committed. This slowing tendency is thought to represent the ability to detect performance threats (i.e., mistakes) as they occur. While this slowing tendency or threat detection ability does not directly relate to affective traits, affective traits interact with post-error slowing to predict affective reactions. Post-error slowing interacts with extraversion to predict displayed levels of anxiety, such that those low in extraversion and high in slowing tendency display more anxiety (Robinson, Meier, Wilkowski, & Ode, 2007). Post-error slowing also interacts with neuroticism, such that those high in neuroticism and high in post-error slowing show lower levels of daily distress (Robinson, Ode, Wilkowski, & Amodio, 2007). While post-error slowing may not directly relate to these affective traits, the second error-reactivity measure, the tendency to make errors in strings, is directly related to neuroticism.

Some individuals show a tendency to make errors in strings in standard cognitive tasks; they are likely to make more than one error in a row. This tendency (as measured with the Stroop, AX-CPT, and N-back tasks) is directly related to neuroticism (Compton et al., 2008; Augustine & Larsen, 2010); those who make errors in strings are higher in neuroticism. While generally related to trait negative affect, this tendency may be specifically relevant for anxiety related behavior. The tendency to make errors in strings is related more strongly to the anxiety subfacet of neuroticism (Augustine & Larsen, 2010) and moderates the relationships between daily stress and anxiety, such that those who are more likely to make errors in strings show a stronger relationship between daily stress and anxiety (Compton et al., 2008). Genetic and neurological evidence also support the links between error-reactivity and affective traits.

At a genetic level of analysis, the same gene alleles that regulate negative affective functioning may also play a role in general cognitive functioning. A polymorphism in the control region (5-HTTLPR) of the serotonin transporter gene 5-HT has been consistently linked to trait negative affect. Those who possess the short allele in this region show lower serotonin uptake (for a review, see Lesch & Canli, 2006). The neurotransmitter serotonin is a well established determinant/marker of a variety of negative affect disorders and traits. Those possessing the short 5-HTTLPR allele do show higher levels of trait negative affect in the form of depression (Canli et al, 2006), anxiety, and neuroticism (Lesch et al, 1996). At a cognitive level, this allele length also predicts differential brain activation in response to neutral stimuli, indicating that the 5-HTTLPR region may play a role in broader cognitive functions (Canli et al., 2005). In

other words, the same genetic structure is related to both affective and cognitive functioning.

At a neurological level, error regulation and affective functioning are associated with activation in the same brain regions. The anterior cingulate cortex is thought to be involved in emotion regulation (Ochsner & Gross, 2005; Hajcak & Foti, 2008). This region has also been shown to be involved in error avoidance and monitoring (Brown & Braver, 2005). In addition, this brain region shows a response when errors are made, the degree of this response predicts the magnitude of negative reaction to the error, and this response is modulated by affective variables (Hajcak & Foti, 2008). Thus, not only is the link between cognition and trait affect observable at a genetic level, the link between error reactivity and trait affect is also observable at a neurological level.

In sum, broad associations between a variety of cognitive processes and trait affective variables suggest that we should consider individual differences in cognition in our efforts to understand affect regulation. Of particular interest is the error reactivity process and, more specifically, the tendency to make errors in strings. This tendency is associated with negative affective traits and anxiety in particular, and moderates individuals' reactions to aversive events.

There are two potential explanations for the observed links between the tendency to make errors in strings and the affect regulation process. One is that this tendency captures or explains individual differences in affect reactivity (i.e. Larsen & Ketelaar, 1989), or the degree of an individual's reaction to an affective stimulus. That is, the stronger an individual's typical negative affective reactions, the more likely they will be to make errors in strings. The other explanation is that this tendency to make errors in

strings captures or explains individual differences in the ability to repair negative affect (i.e. Hemenover et al., 2008). That is, the lower a person's ability to regulate or repair negative affect, the more likely they will be to make errors in strings. In the next section, I review the role of affect reactivity and regulation ability in the affect regulation process. I then review individual differences in affect reactivity and regulation ability and describe how the tendency to make errors in strings may relate to each of these constructs.

The Affect Regulation System

Gross (1998) defines emotion regulation as a set of "processes by which individuals influence which emotions they have, when they have them, and how they experience and express these emotions (pg. 275)." One might attempt to decrease, increase, or maintain their current emotions. For instance, when in a particularly unpleasant mood, one might attempt to decrease their experience of negative affect and increase their experience of positive affect. When in a good mood, one might attempt to maintain that good mood. Individuals also attempt to alter the frequency of certain affective experiences. Some people try to avoid situations that increase negative affect and seek out situations that promote positive affect. Additionally, individuals attempt to alter the manner in which they express emotion. Someone might make a "happy face" to show that they identify with another person's positive experiences. Three major models (Gross, 1998; Bonnano, 2001; Larsen, 2000) have been proposed that describe the processes that individuals engage in when attempting to regulate their affective experiences.

In Gross'(1998) model, regulatory efforts are either response focused or antecedent focused. That is, people either regulate emotion while they are experiencing

an emotional episode (response focused) or attempt to control the emotions that they may experience in the future (antecedent focused). These two goals are accomplished through a set of five specific regulation efforts. First, individuals engage in situation selection; they choose environments that are consistent with, or might produce, their desired affective state. When in an affectively relevant situation, one engages the second regulation effort, situation modification. In situation modification, one attempts to alter components of the situation that are inconsistent with the desired affective state. Third, attention deployment is used to attend to stimuli that are consistent with their regulatory goals. When a situation is not consistent with regulatory goals, a cognitive change (the fourth regulation effort) may be necessary to interpret the meaning of the situation in a manner consistent with regulatory goals. Finally, one may engage in response modulation, whereby their emotional response tendencies are influenced once they have been elicited (Gross, 1998).

Bonanno (2001) offers another model by which individuals purposely alter their affective experiences. Central to this model is the idea of emotional homeostasis, a process by which an individual engages in efforts to achieve and maintain a desired emotional state. Similar to cellular homeostasis, the process of emotional homeostasis focuses on eliminating affects not in-line with the desired emotional state and increasing or maintaining affects consistent with the desired emotional state. To achieve or maintain this emotional homeostasis, one engages in a set of three regulation efforts; control regulation, anticipatory regulation, and exploratory regulation. If one is currently experiencing an emotional response not in-line with their homeostasis point, they engage in control regulation, which includes automatic and instrumental behaviors used for the

purpose of immediate affect regulation. When the homeostasis point is achieved, one then begins anticipatory regulation. In anticipatory regulation, one attempts to eliminate future experiences that would direct them away from homeostasis and encourage experiences that would maintain homeostasis. Finally, if control and anticipatory regulation efforts are successful, one can then attempt to develop new skills, knowledge, or resources that will aid in future regulatory efforts through a process known as exploratory regulation (Bonanno, 2001). In this model, any experience that gives one information pertaining to future regulation is classified as exploratory regulation.

The idea of an emotional homeostasis point is not unique to Bonanno's (2001) model of affect regulation. Although called the affective set point in this model, Larsen (2000) also proposes a model of affect regulation in which individual's attempt to reach an "affective norm." In Larsen's (2000) model, individuals are aware (consciously or not) of their desired affective set point and go through a continual process in which they compare their current affective state to this set point. Should an inconsistency exist between their desired set point and their current affective state, a regulation process is engaged to address the discrepancy.

In all of these models, individuals engage in some type of regulatory effort when their current affective experience is inconsistent with their desired affective experience. However, the degree of inconsistency between desired and current affective experience is largely dependent on affect reactivity. If individuals react to affective stimuli in a differential manner, then the frequency and efficacy of regulation required to maintain a desired state would be based on these reactions. Indeed, individual differences exist in

affect reactivity, and these differences in affect reactivity are related to neuroticism in the same manner as are differences in error reactivity.

Affect Reactivity

Steeped in animal learning theory and the effects of anxiolytic (anti-anxiety) medications, the work of Jeffrey Gray (1971, 1981, 1987; Gray & McNaughton, 2000; for a review, see Fowles, 2006) was the first to examine potential differences in the way individuals respond to affective stimuli. Gray's theory of personality is based on two separate neurological systems that govern sensitivity to environmental cues of reward and punishment. These systems, which he termed the behavioral activation (BAS) and behavioral inhibition systems (BIS) regulate behavior in the presence of cues for reward and punishment, respectively. Gray thought that these systems exhibited trait-like properties; stable individual differences exist in the sensitivity of these two systems. In other words, one could have a habitually sensitive (or insensitive) BAS or BIS. Individuals with habitually sensitive (or more active/reactive) behavioral activation systems would be more likely to react to signals of reward and those with active behavioral inhibition systems would be more likely to react to signals of punishment. The output of reactions to these signals comes in the form of affect, with reaction to reward creating positive and reaction to punishment creating negative affect.

The research to date has generally supported Gray's initial notions regarding the mechanism of affect reactivity. In experimental tests of the theory, participants report their affective state, undergo an affect induction procedure, and then report their affective state a second time. The degree to which affectively relevant personality variables predict participant's reactions to the affect induction are then examined. Individuals with

high trait positive affect exhibit a larger positive affect reaction to positive affect inductions and those with high trait negative affect exhibit a larger negative affect reaction to negative affect inductions.

In the first study to experimentally examine affect reactivity, Larsen and Ketelaar (1991; see also Rusting & Larsen, 1997, 1999; Zelenski & Larsen, 2000) demonstrated that extraversion and neuroticism predict reactions to positive and negative affect stimuli, respectively. The existence of reactivity differences centering around extraversion and neuroticism has been replicated using a variety of affect induction procedures such as guided imagery (Larsen & Ketelaar, 1991), false performance feedback (Larsen & Ketelaar, 1989), viewing affective images (Zelenski & Larsen, 2000), and viewing affective films (Hemenover, 2003; Hemenover, Augustine, Shulman, Tran, & Barlett, 2008),

The affect reactivity findings for neuroticism have also been extended into non-laboratory settings using experience sampling designs. Neurotics experience more negative health symptoms (Larsen & Kasimatis, 1991), undesirable events (David, Green, Martin, & Suls, 1997), and general problems (Suls & Martin, 2005) than do their emotionally stable counterparts. They also react more strongly when these daily troubles arise. For instance, neuroticism predicts greater reactivity to interpersonal conflicts (Suls, Martin, & David, 1998). Neurotics also react with more distress to negative stress appraisals (Cimbolic Gunthert, Cohen, & Armeli, 1999).

All of the above studies rely on self-reported levels of affect to establish the occurrence of an affect reactivity process. These processes may also be observable at a physiological level. This would be an appropriate finding given Gray's focus on the

physiological basis of the reactivity/sensitivity process. Indeed, several findings suggest that individuals do experience physiological reactivity to signals of reward and punishment. Extraversion and neuroticism predict the magnitude of facial-emotional responses to positive and negative stimuli, respectively (Berenbaum & Williams, 1995). In addition, neuroticism predicts greater skin conductance reactivity (as well as a more prolonged response) to negative stimuli (Norris, Larsen, & Cacioppo, 2007). Neuroticism also predicts heightened, non-specific electrodermal activity, suggesting hyperactivity of the sympathetic nervous system on the part of those higher in neuroticism (Larsen & Cruz, 1995). At a neural level, extraversion and neuroticism predict brain activation in response to positive and negative stimuli, respectively; these effects are observable across a number of different brain regions (Canli, Zhao, Desmond, Kang, Gross, & Gabrieli, 2001). Finally, the brain asymmetries associated with extraversion and neuroticism are also predictive of differential reactions to positive and negative affect, respectively (Wheeler, Davidson, & Tomarken, 1993).

In sum, individuals differ in the degree to which they react to affective stimuli. This process of affect reactivity has been consistently linked to neuroticism in both the laboratory and field, and with both behavioral and physiological measures. The links between neuroticism and affect reactivity are directly relevant for findings regarding error reactivity.

Error Reactivity and Affect Reactivity

The existence of a reactivity process has been consistently demonstrated using affective stimuli. However, individuals show differential reactions to non-affective stimuli as well, particularly to markers of reward and punishment. Those higher in BAS

react with more positive affect to signals of reward and those higher in BIS react with more negative affect to signals of punishment (Gable, Reis, & Elliot, 2000). In addition, those high in BAS experience more positive affect when seeking reward and those high in BIS experience more negative affect when placed under stress (Heponiemi, Keltikangas-Jarvinen, Puttonen, & Ravaja, 2003). Finally, extraversion predicts positive affect reactivity to monetary gain whereas neuroticism predicts negative affect reactivity to monetary losses (Gomez, Cooper, & Gomez, 2000).

Making an error in a standard cognitive task may also be a form of punishment. In some cases this is explicit, as when feedback is given for errors. In easier tasks, no feedback is necessary as a mistake in the task is quite obvious. If neuroticism predicts responses to more overt forms of punishment (i.e. monetary loss), it may also predict responses to mistakes and signals of punishment in these cognitive tasks. These responses to errors in standard cognitive tasks would come in the form of error reactivity. If an individual reacted more strongly to an error on a trial, that reaction would disrupt responses on the ensuing trial. Any disruption of response would increase the likelihood of a second error. Thus, with a stronger reaction to a mistake, an individual prone to more intense reactions to punishment (i.e., a neurotic) would be more likely to make errors on ensuing trials. It is in this manner that findings linking neuroticism and anxiety to the tendency to make errors in string (Compton et al., 2008) may be explained by individual differences in affect reactivity.

While affect reactivity may explain the observed findings linking error reactivity and affective traits/processes, it is also possible that self-regulation ability provides an alternative explanation for these effects. Given the affective nature of the variables

associated with the tendency to make errors in strings, affect regulation (rather than some broader form of self-regulation) is of primary interest in attempting to explain these findings.

Affect Regulation

The efficacy of any attempt to regulate affective experience is dependent on two factors: the type of regulation strategy used and the ability to use that strategy. If individuals use more effective strategies, then the affect regulation attempt is more successful. If individuals are higher in affect regulation ability, then any regulation attempt, regardless of the type of strategy employed, should be more effective. In this section, I review difference in affect regulation strategy effectiveness and affect regulation ability.

In general, people want to feel happy; we wish to experience mild positive affect most of the time (Rusting & Larsen, 1995; Larsen, 2000; Augustine, Hemenover, Larsen, & Shulman, 2010). Given this, most affect regulation efforts are aimed at affect repair, or efforts to decrease levels of negative affect and increase or maintain levels of positive affect. Thus, the efficacy of any affect regulation strategy is usually defined by how well that strategy allows one to repair a negative affective state. However, not all affect regulation strategies are created equal. Certain strategies have been demonstrated to be more effective means of affect repair than others.

Regardless of popular claims to the contrary, catharsis (venting), is ineffective at reducing negative affect and actually increases bad feelings (Geen & Quanty, 1977; Bushman, 2002). These findings suggest that, if you hit a pillow because you are angry at your boss, your anger levels would likely increase rather than decrease. Yet another

strategy found to be ineffective at reducing negative affect and increasing positive affect is suppression (i.e., hiding one's feelings). Although those using suppression report less negative affect, physiological measures show that they are actually experiencing higher levels of emotional arousal than before the repair effort began (Gross & John, 2003). Thus, attempting to suppress the urge to cry will actually produce physiological arousal patterns more intense than if you had just allowed yourself to cry. Catharsis and suppression are well known examples of frequently employed strategies that produce results opposite of what is intended.

Despite these examples, numerous effective strategies for the repair of affect do exist. Downward social comparison can be an effective regulation strategy; merely thinking about those who "have it worse" has positive affective consequences (Aspinwall & Taylor, 1993). Attempting to avoid the future experience of negative affect is also an effective method of affect regulation. If one simply avoids encountering stimuli that are known to have negative affect inducing properties, then less negative affect will be experienced (Larsen & Prizmic, 2004). Reappraisal has definite benefits; not only does this strategy aid in reducing negative affect, it also has physical, immune, and psychological benefits; those who consistently use reappraisal have a lower level of physiological-emotional arousal, a decreased probability of future health problems, and an increase in subjective well-being (Gross & John, 2003). Distraction has also been shown to be an effective method for affect repair in most situations, having been found to decrease depressed mood even in naturally depressed patients (Nolen-Hoeksema & Morrow, 1993). Finally, an effective way to reduce negative affect and increase positive

affect is engaging in rewarding, pleasant activities (Fichman, Koestner, Zuroff, & Gordon, 1999).

In a meta-analysis of the effectiveness of affect regulation strategies, Augustine and Hemenover (2009) found that there were wide differences in the effectiveness of both specific affect regulation strategies and categories of affect regulation strategies (e.g. Parkinson & Totterdell, 1999). At a broad level, strategies involving distraction and reappraisal were the most effective strategies for affect repair. However, within each category of affect repair strategies, broad differences existed between the effectiveness of repair attempts in different studies. Thus, there are wide differences in the effectiveness of affect regulation strategies.

Given the differences in affect regulation strategy effectiveness, the degree to which one uses a given strategy could explain any individual differences in a broad measure of personal affect regulation success. As such, the type of affect regulation strategy used is controlled in the current research. However, differences in affect regulation effectiveness still exist even when controlling the type of strategy used.

Those high in extraversion, negative mood regulation expectancies, mood monitoring, and mood labeling, and those low in neuroticism demonstrate better affect regulation ability (Shulman, Augustine, & Hemenover, 2006; Hemenover, Augustine, Shulman, Tran, & Barlett, 2008). When randomly assigned to engage in an affect regulation strategy (after a negative affect induction) participants who possessed more positive (and less negative) affective profiles were better able to use the assigned strategy for affect regulation. This means that, given the same stimuli and conditions for a

regulation task, and even if motivated to engage in an effective strategy, certain individuals are less able to regulate their affective experiences.

Additional evidence supports the existence of individual differences in the ability to repair affect. For instance, those high in agreeableness put more effort into their regulation attempts (Meier, Robinson, & Wilkowski, 2006). This effortful and often automatic regulation may be partially facilitated by the automatic activation of regulation mechanisms in the brain (Haas, Omura, Constable, & Canli, 2007). Consistent users of reappraisal may also gain a similar ability boost, as this strategy also engages early neural mechanisms that dampen further negative affect (Hajcak & Nieuwenhuis, 2006). In addition to possessing superior repair abilities, extraverts (vs. introverts) are also better able to engage in positive mood maintenance (Lischetzke & Eid, 2006) and engage in effective affect regulation in even the most benign (i.e. control conditions) settings (Augustine & Hemenover, 2008). Thus, individual differences exist in the ability to regulate affect, regardless of the type of strategy employed. These differences may account for the observed links between the tendency to make errors in strings and affective traits/processes.

Error Reactivity and Affect Regulation Ability

Just as expanding the definition of punishment (i.e. error on a cognitive task) allows the use of affect reactivity as an explanation for findings linking the tendency to make errors in strings, expanding the definition of an error allows the use of affect regulation ability as a mechanism for explaining these findings. The tendency to make errors in strings could be thought of as the tendency to let errors go uncorrected or a tendency to let one's reaction to an error interfere with subsequent behavior (i.e., a

breakdown in self-regulation). This tendency becomes relevant for affect regulation ability when one views negative affect as a mistake. In general, people do not want to experience negative affect (Augustine et al, 2010). Thus, the experience of negative affect is, in a sense, an error. As such, the tendency to let any error (i.e., in a Stroop task) go uncorrected may be related to the tendency to let an affective error (i.e., the experience of negative affect) go uncorrected.

A failure to regulate behavior following a cognitive error leads to additional errors; the mistakes made are not corrected. The failure to regulate affect, or failure to expend enough effort and regulate affect in an appropriate manner, leads to less effective affect regulation. As was discussed previously, a number of findings suggest that affective and cognitive regulation (particularly error reactivity) both rely on the same neurological substrates (i.e., the anterior cingulate cortex, Brown & Braver ,2005; Ochsner & Gross, 2005; Hajcak & Foti, 2008). It is possible that all self-regulation (cognitive and affective) relies on the same cognitive and neural control systems (Compton et al., 2008). If all regulation shares common systems, then deficits in one type of regulation ability may be related to deficits in another. Regulation of one's affective response to an error in a cognitive task would, in all likelihood, not take place. The speed of the tasks would preclude the possibility of engaging in a conscious affect regulation attempt. However, any observed relationships between the tendency to make errors in strings and affective variables could be due to common underlying regulation systems and a general failure of self-regulation.

Overview of the Current Research

A growing body of research regarding personal cognition suggests that personality is related to a number of "cold" cognitive tendencies, such as decision making, working memory, and error reactivity. However, mechanisms that explain these findings are lacking in a number of areas. Of interest to the current research is the process of error reactivity and, in particular, the tendency to make errors in strings. The tendency to make errors in strings has been found to predict neuroticism, anxiety, and anxiety responses to stressors. Two different aspects of the affect regulation process may explain these findings: affect reactivity or affect regulation ability.

Affect reactivity, or the magnitude of reaction to affective stimuli, is related to affective traits in a manner similar to that of the tendency to make errors in strings. In addition, differences in affect reactivity have been found for non-affective stimuli. These findings suggest that individuals may make errors in strings because a stronger reaction to a mistake on one trial disrupts responses on ensuing trials. Alternatively, affect regulation ability may explain relationships between affective traits and the tendency to make errors in strings. Given shared systems between cognitive and affective regulation, it is possible that all errors, including affective errors (i.e. undesired negative affect), are monitored in a similar manner. Thus, a failure to regulate behavior following an error on a cognitive task would be indicative of consistent self-regulation failures in a variety of domains, including affect regulation. The goal of the current research is to test these two alternative hypotheses.

These two alternative hypotheses will be tested by examining individual differences in anxiety reactivity and anxiety regulation ability. There are three reasons

for choosing anxiety, rather than global negative affect, as the target of the research. First, the process of affect reactivity was initially based on anxiety reactions (see Fowles, 2006). Second, the tendency to make errors in strings shows the strongest relationships with the anxiety subfacet of neuroticism (Augustine & Larsen, 2010). Finally, this tendency moderates daily relationships between stress and anxiety (Compton et al., 2008).

To test these two alternative hypotheses, individuals will undergo an anxiety induction and then engage in one of three affect regulation strategies: reappraisal, distraction, or control. These different affect regulation strategies will be used to allow for individual differences in affect regulation ability to emerge. Reappraisal and distraction are two highly effective strategies which should allow for wide individual differences in ability (Augustine & Hemenover, 2009; Hemenover et al., 2008). These two strategies also differ in the amount of cognitive resources required for implementation, with reappraisal consuming more cognitive resources than distraction (Sheppes & Meiran, 2008). Given the influence of stable levels of cognitive resources on the affect regulation process (Schmeichel et al., 2008), it is possible that effects could emerge only for those strategies requiring few cognitive resources (i.e. distraction). The use of a control condition will also allow for the observance of individual differences in affect regulation ability. Despite the label, "control" conditions are actually highly effective means of affect regulation (Augustine and Hemenover, 2009) and do allow for the emergence of individual differences in affect regulation ability (Augustine & Hemenover, 2008).

Using an anxiety induction and three effective affect regulation manipulations, the current study examines the potential underlying mechanisms of the observed relationships between the tendency to make errors in strings and affective traits. If this tendency is related to participants' reaction to the anxiety induction (but not affect regulation ability) then links between error reactivity and affective traits are a product of affect reactivity. On the other hand, if the tendency to make errors in strings is related to the degree to which participants decrease in anxiety following the regulation manipulation (but not affect reactivity), then links between error reactivity and affective traits are a product of affect regulation ability.

The Current Research

Method

Participants

All participants were recruited from a psychology students participant pool at a large, private, Midwestern university and received partial course credit for participation (Age: M = 19.13, SD = 1.19; 76.9% female; 66.3 % Caucasian, 23.6% Asian, 6.3% African-American, 1.9% Hispanic). A total of 208 participants were recruited for this study (conditions: control n = 70, distraction n = 70, reappraisal n = 68). Assuming effect sizes of r = .25, *power* = .96.

Materials

Affect Induction

Anxiety was induced using a memory recall procedure. Participants were instructed to, "Think of a recent experience in which you felt very anxious. We would like you to write about that experience for the next five minutes. Please describe exactly

what made you feel anxious. Describe why this made you feel anxious. Also describe what it felt like to be anxious: what were you thinking, what types of bodily sensations did you experience, etc. Please be as specific and detailed as possible." Participants wrote about this anxiety provoking experience for five minutes.

Affect Regulation Manipulation

Participants were randomly assigned to engage in one of three affect regulation tasks: control, reappraisal, or distraction. Instructions for the control condition (adapted from Hemenover et al., 2008) were, "For the next 5 minutes we would like you to list your thoughts as they occur to you. On the lines below, list whatever thoughts are going through your mind. List any thought that occurs to you." Instructions for the reappraisal condition (adapted from Hemenover et al., 2008) were, "Sometimes when bad things happen, they can also have some positive consequences. For the next 5 minutes we would like you to list some good things that occurred as a result of the anxiety experience you just wrote about. In other words, what are some positive consequences of your anxiety provoking experience?" Finally, instructions for the distraction condition (adapted from Rusting & Nolen-Hoeksema 1998) were, "For the next five minutes we would like you to focus your thoughts and attention on things that are in no way relevant to the anxiety provoking experience you described earlier. You may write about anything you like, as long as it is in no way related to the anxiety experience you just described."

An independent coder reviewed each participant's writing to determine if the participant followed the instructions (2 participants in the reappraisal condition did not follow instructions and were removed from the dataset). Additionally, to determine how

much effort was exerted during these tasks, the number of words each participant wrote was calculated.

Error Reactivity Measures

The tendency to make errors in strings and post-error slowing was assessed using two different cognitive tasks, the AX-CPT and the Stroop. In the AX-CPT (Braver, Cohen, & Barch, 2002), participants are presented with two letters in succession and asked to make one response if the letters are an A followed by an X and another response if any other combination of letters appears. Each letter was presented for 125 milliseconds (ms) and participants were given 400ms to respond on each of the 120 trials. A warning was presented if a response was not made during the 400ms response window.

The Stroop task (Stroop, 1935) was administered in a similar manner as was used by Compton and colleagues (2008). On each trial, individuals indicated the color of the font used for one of six color words (red, orange, yellow, green, blue, or purple). The task began with 24 practice trials that gave performance feedback. Participants then completed four blocks of 80 trials (25 trials where the word and font color were congruent and 55 trials where the word and font color were incongruent). Between blocks, participants were reminded of the correct response mapping (i.e. respond to font color, not the word itself). To ensure task difficulty, responses were speeded, with the stimulus presented for 150ms and a response window of 1.0 seconds allowed.

Questionnaires

Affect. State affect was assessed using three adjective scales. These scales measured negative (unhappy, miserable, sad, grouchy, gloomy, blue, distressed, annoyed, fearful, upset, hostile, angry; T1 α = .86, T2 α = .90, T3 α = .87), positive (enthusiastic,

elated, excited, euphoric, lively, peppy, happy, delighted, glad, cheerful, warmhearted, pleased; T1 α = .94, T2 α = .93, T3 α = .94), and anxious (nervous, jittery, anxious, tense, uneasy, worried, restless, on edge, anxiety, panicky; T1 α = .86, T2 α = .89, T3 α = .89) affect. Participants indicated the extent to which they were experiencing each affective state using a 5-point, very slightly or not at all – extremely, Likert-type scale.

Personality. Neuroticism was assessed using the full neuroticism subfacet scale from the International Personality Item Pool (IPIP; Goldberg, Johnson, Eber, Hogan, Ashton, Cloninger, & Gough, 2006)**.** The IPIP neuroticism scale is a 60 item scale that assesses neuroticism (*M* = 2.62, *SD* = .51, α = .94)as well as six subfacets of neuroticism (anxiety, *M* = 2.91, *SD* = .72, α = .84; anger, *M* = 2.47, *SD* = .70, α = .86; depression, *M* = 2.13, *SD* = .75, α = .88; self-consciousness, *M* = 2.75, *SD* = .75, α = .84; immoderation, *M* = 2.95, *SD* = .65, α = .77; vulnerability, *M* = 2.52, *SD* = .69, α = .86) using a 5-point, very inaccurate – very accurate, Likert-type scale.

Procedure

Participants came into the lab in small groups for a study entitled "Personality and Affective Memory." They provided consent and then completed all personality and error reactivity measures, as well as a baseline affect measurement (affect T1). They then underwent the affect induction procedure and completed a second affect measurement (affect T2). Finally, participants were randomly assigned to complete one of the three affect regulation tasks (distraction, reappraisal, or control), completed the final affect measurement (affect T3), and were debriefed and released (see Figure 1).

Results

Manipulation Checks

To ensure that the affect induction increased anxiety, and did so equally across conditions, a 2 (Time: Affect T1, T2) x 3 (Condition: control, reappraisal, distraction) mixed model analysis of variance was conducted for each affect measure (see Table 1 for all affect data). For anxiety, results indicate a significant effect of time (F (1, 205) = 21.12, $p < .001$, $d = .27$) that did not differ based on repair condition (time x condition interaction, F (2, 205) = 1.64, $p = .20$). For negative affect, results indicate a significant effect of time (F (1, 205) = 5.75, $p < .02$, $d = .14$) that did not differ based on repair condition (time x condition interaction, F (2, 205) = .99, $p = .37$). Finally, for positive affect, results indicate a significant effect of time (F (1, 205) = 37.92, $p < .001$, $d = -.27$) that did not differ based on repair condition (time x condition interaction, F (2, 205) = 1.05, $p = .35$). Thus, as intended, the anxiety induction resulted in significant increases anxiety and negative affect, and significant decreases in positive affect, and these effects were not dependent on repair condition.

To examine the cross-task reliability of the error reactivity measures (Stroop and AXCPT), error rates were compared across tasks. Examining raw scores, the number of repeated errors ($r = .10$, $p = .16$) and the proportion of repeated errors to total errors ($r = .11$, $p = .13$) were not significantly related across tasks, although the total number of errors were modestly related ($r = .25$, $p < .001$). Further, if the tendency to make errors in strings is calculated in a manner consistent with previous research (accuracy following errors – accuracy following correct responses: Compton et al., 2008), the relationship between tasks remains non-significant ($r = .09$, $p = .17$). In addition, although the

relationships between neuroticism (r = .16, p < .05) and trait anxiety (r = .17, p < .01) and the tendency to make repeated errors (accuracy following errors – accuracy following correct responses) is significant for the AXCPT, it is not for the Stroop (neuroticism r = .08, p = .19; trait anxiety r = .01, p = .90). For the tendency to slow down following an error (reaction time on a correct trial following an error trial minus reaction time on a correct trial following a correct trial; Compton et al., 2008), measures between tasks were again unrelated (r = -.06, p = .39). As with the measure of the tendency to make errors in strings, post-error slowing was related to neuroticism and trait anxiety for the AX-CPT (Neuroticism r = -.20, p < .01; Anxiety r = -.14, p < .05), but not for the Stroop (Neuroticism r = .02, p = .75; Anxiety r = .08, p = .19). Thus, assuming that it is possible to reliably measure the tendency to make errors in strings, these two tasks seem to be tapping different components of the error reactivity process. As such, rather than creating a conglomerate measure, each task will be examined separately in ensuing analyses.

Repair Strategy Effectiveness

To determine if affect repair effectiveness differed based on condition, several ANCOVAs were conducted with affect time 3 as the dependent variable, affect time 2 entered as a covariate, and condition entered as the independent variable. Results for anxiety (F (2, 204) = 11.62, p < .001), negative affect (F (2, 204) = 3.48, p < .05), and positive affect (F (2, 204) = 10.11, p < .001) all indicate that affect repair effectiveness differed based on condition. Follow-up analyses using Student's t tests indicates that participants in the reappraisal condition experienced larger decreases in anxious (t (136) = 2.65, p < .05, d = -.45) and negative affect (t (136) = 2.29, p < .05, d = -.39), and larger increases in positive affect (t (136) = -2.65, p < .05, d = .45), than did those in the control

condition. Those in the distraction condition experienced larger decreases in anxious affect (t (138) = 3.83, $p < .05$, d = -.65) and larger increases in positive affect (t (138) = -3.97, $p < .05$, d = .67) than did those in the control condition. No other differences were observed between conditions (ts = -1.76 – 1.81, *ns*). Thus, affect repair effectiveness differed based on condition, with the reappraisal and distraction conditions leading to the most effective affect repair.

To examine if participants' effort during the repair task differed based on condition, an analysis of variance was conducted on the number of words each participant wrote during the repair task. Results indicate that effort did differ based on condition (F (2, 202) = 14.05, $p < .05$), with those in the control condition writing the most (M = 134.87, SD = 36.43), followed by those in the distraction (M = 122.76, SD = 29.30) and reappraisal conditions (M = 106.15, SD = 28.12). Post-hoc comparisons (using a Bonferroni adjustment) indicate that participants in the control (*Mean Dif* = 28.72, SE = 5.43, $p < .05$, d = .88) and distraction (*Mean* Dif = 16.61, SE = 5.45, $p < .05$, d = .58) conditions wrote more words than did those in the reappraisal condition. Given the differences in word counts between conditions, it is possible that effort would be a determinant of repair effectiveness. To examine this possibility, several hierarchical multiple regression analyses were conducted with affect time 3 as the dependent variable, affect time 2 entered on step 1 as a covariate, and word count entered on step 2 as the independent variable. Results for anxiety (β = .02, t (202) = .35, p = .73), negative affect (β = -.07, t (202) = -1.36, p = .17), and positive affect (β = .02, t (202) = .40, p = .69) all indicate that effort did not predict repair effectiveness. Given these results, effort will not

be considered as a possible moderator/covariate for any ensuing analyses of the predictors of repair effectiveness.

Affect Reactivity

The following analyses were conducted to determine the degree to which neuroticism, trait anxiety, and error reactivity predict participants' reactions to the anxiety induction. To accomplish this, a number of hierarchical multiple regression analysis were conducted with affect time 2 entered as the dependent variable, affect time 1 entered on step 1 as a covariate, and the relevant individual difference variable (neuroticism, trait anxiety, or error reactivity) entered on step 2 as the independent variable. As would be expected based on prior research, neuroticism and trait anxiety predicted the degree to which participants reacted to the induction, with those higher in this traits showing larger increases in anxious (neuroticism β = .11, t (205) = 1.94, p < .05; anxiety β = .10, t (205) = 1.87, p < .065) and negative affect (neuroticism β = .10, t (205) = 1.83, p < .07; anxiety β = .11, t (205) = 2.12, p < .05), and larger decreases in positive affect (neuroticism β = -.15, t (205) = -3.35, p < .001; anxiety β = -.15, t (205) = -3.23, p < .001).

For the tendency to make errors in strings (calculated as accuracy following errors – accuracy following correct responses: Compton et al., 2008), error reactivity on the Stroop task did not predict anxiety reactivity (β = -.05, t (205) = -.91, p = .36), negative affect reactivity (β = -.04, t (205) = -.71, p = .48), or positive affect reactivity (β = .01, t (205) = .19, p = .85). Similarly, error reactivity on the AXCPT did not predict anxiety reactivity (β = -.08, t (205) = -1.41, p = .16), negative affect reactivity (β = -.09, t (205) = -1.72, p = .09), or positive affect reactivity (β = -.04, t (205) = -.89, p = .38).

For post error slowing, error reactivity on the Stroop task did not predict anxiety reactivity (β = -.10, t (205) = -1.88, p = .07), negative affect reactivity (β = -.02, t (205) = -.41, p = .68), or positive affect reactivity (β = .00, t (205) = .00, p = .99). Similarly, error reactivity on the AXCPT did not predict anxiety reactivity (β = -.03, t (205) = -.53, p = .60), negative affect reactivity (β = -.06, t (205) = -1.19, p = .24), or positive affect reactivity (β = .08, t (205) = 1.84, p = .07). Thus, although neuroticism and trait anxiety predicted affect reactivity, neither measure of error reactivity predicted affect reactivity.

Affect Regulation Ability

The following analyses were conducted to determine the degree to which neuroticism, trait anxiety, and error reactivity predict participants' affect regulation ability (i.e., degree to which participants altered their affective states due to the repair task). To accomplish this, a number of hierarchical multiple regression analyses were conducted in which affect time 3 was entered as the dependent variable, affect time 2 was entered on step 1 as a covariate (to control the effects of affect reactivity), the relevant individual difference variable (personality variables were centered) and the condition effect were entered on step 2, and the condition x individual difference variable interaction was entered on step 3. The results of these analyses can be viewed in Tables 2 - 4.

Results indicate that, consistent with prior research (Shulman et al., 2006), neuroticism predicts negative affect repair. However, neuroticism does not significantly predict anxiety or positive affect repair, nor are any of the effects interacting with condition. Trait anxiety does not significantly predict the repair of any type of affect and trait anxiety does not interact with condition. Finally, neither the tendency to make errors

in strings (accuracy following errors – accuracy following correct responses) nor post-error slowing predict, or interact with condition to predict, the repair of any type of affect.

Although none of the condition x individual difference variable effects were significant, it is still possible that one of the individual difference variables may have an effect on affect repair in a particular condition. To examine this, simple slopes follow-ups were conducted (Cohen, Cohen, West, & Aiken, 2003). As seen in Table 5, although there is a main effect of neuroticism for negative affect repair, this effect is particularly strong in the reappraisal condition. The results also reveal that both measures of the tendency to make errors in strings are related to anxiety repair in the control condition. Those who are more likely to make errors in strings experienced less anxious affect following the control condition manipulation. Additionally, this tendency, as measured by the Stroop, predicts positive affect repair in the distraction condition, such that those who are more likely to make errors in strings were lower in positive affect following the distraction condition manipulation. Post-error slowing on the Stroop predicts anxiety repair in the control condition and positive affect repair in the distraction condition, such that those with a greater tendency to slow down following an error displayed higher levels of anxiety and positive affect. Additionally, post-error slowing on the AX-CPT predicts predicts the repair of all affect types in the control condition, such that those with a greater tendency to slow down following an error displayed higher levels of anxiety and negative affect, and lower levels of positive affect. Finally, post-error slowing on the AX-CPT predicts positive affect repair in the distraction condition, such that those with a greater tendency to slow down following an error were lower in positive affect.

Discussion

In this study, I sought to examine the potential mechanisms underlying previously observed associations between cognitive error reactivity and various parameters of affective experience. Prior research suggests two competing mechanisms. First, the tendency to make errors in string may be related to neuroticism and stress reactivity (Compton et al., 2008) due to the process of affect reactivity. If individuals react more strongly to environmental signals of negative affect, they may react in a similar manner to the negative cue of making an error on a cognitive task. This relatively large reaction to making an error would disrupt responding on ensuing trials and thus, lead to a greater tendency to make an error following an error. Second, these associations may be due to shared systems that underlie all regulation attempts, be they cognitive or affective. Following this explanation, any individual with deficits in one regulatory realm (i.e., affect regulation) would show, due to shared systems, regulatory deficits in another realm (i.e., cognitive error regulation). To test these two alternative hypotheses, participants in this study first underwent an anxiety induction and then were randomly assigned to complete one of three affect repair tasks (reappraisal, suppression, or control condition). Neither the affect reactivity, nor the affect regulation ability, hypothesis were supported in this study.

Personality and Affective Responses to Manipulations

The majority of research on both affect reactivity and regulation has focused on global negative affect, and not specific affects, such as anxiety (Augustine & Hemenover, 2009). As such, the design of this study allowed for an replication of several effects known in the literature and an extension of those effects into the realm of anxiety

reactivity and regulation. First, in terms of the effectiveness of the different strategies for the repair of anxiety, results indicate that affect repair effectiveness did differ based on condition. Participants in the reappraisal and distraction conditions experienced more effective affect repair than those in the control condition. Moreover, while effort did differ based on condition, such that those in the distraction and control conditions wrote more words than those in the reappraisal condition, effort did not predict affect repair effectiveness. Thus, consistent with other research regarding the effectiveness of distraction and reappraisal for the repair of global negative affect (Augustine & Hemenover, 2009), these strategies were highly effective for the elimination of anxious affect in this study. In addition, there is something unique to the cognition underlying the implementation of these strategies, as the effort involved in implementation does not predict the degree to which one is successful at using these affect regulation strategies.

These data also extended research on affect reactivity into the realm of anxious affect. Consistent with past research on reactivity to global negative affect (e.g., Larsen & Ketelaar, 1989), both neuroticism and trait anxiety predicted affect reactivity to the anxiety induction, such that those higher in these traits experienced larger increases in negative and anxious affect, and larger decreases in positive affect. Thus, the reactivity processes which are a hallmark of the highly neurotic individual can also be observed when examining specific affects, such as anxiety.

Thus, this study replicated known effects; personality predicts the effects of affect inductions and reappraisal and distraction are effective affect repair strategies. In addition, this study also allowed for the first examination of the potential predictors of anxiety repair ability. In a series of studies by Hemenover and colleagues (2008), it was

found that neuroticism (and other negative affect traits) predicted the ability to repair both global negative affect and sadness. In line with these findings, my results indicate that neuroticism predicts the ability to repair negative affect. Further analyses indicate that the effect of neuroticism on negative affect repair is particularly strong in the reappraisal condition. While I did observe a main effect of neuroticism on the repair of negative affect, there were no effects of neuroticism on the repair of positive or anxious affect. Anxiety is highly related to neuroticism, forming a subfacet of this broad personality trait. There are several reasons why those relatively high in neuroticism would be less able to repair negative affect, but similar effects were not observed for anxiety.

First, in factor analytic studies, results consistently demonstrate that affective experience can be best described using two broad and orthogonal factors pertaining to negative and positive affect (i.e., Watson, Clark, & Tellegen, 1988). It may be that, by parsing out a unique portion of the negative affectivity factor and thus, loosing the structural cohesion of negative affect, the ability to detect individual differences in anxious affect is reduced. This possibility also relates to a second potential explanation, with relatively strong cohesion amongst negative affects, it may be that the increased anxiety of the neurotic individual is a by product of a decreased ability to repair other negative affects. As fluctuations in one type of negative affect are often accompanied by fluctuations in another, then a failure to regulate other affects, such as sadness, would be accompanied by increases (or relatively smaller decreases) in anxiety at a mean level. Finally, it may be that, while the neurotic individual does show deficits in the ability to repair other types of negative affects, they may be equally adept at repair anxiety as are

emotionally stable individuals. If this were the case, then the increased anxiety of the neurotic individual would be the results of other processes, not regulatory ability. Indeed, neuroticism did predict an increased reaction to the anxiety induction. While this reactivity was controlled in order to examine repair ability, the increased reactivity has real world implications. While they may be able to decrease anxious affect to the same degree as someone low in neuroticism, the highly neurotic individual would have a higher level of anxiety at the beginning of that attempt (due to increased reactivity) and thus, the same magnitude of affect repair in those low and high in neuroticism would still result in a higher level of anxiety for the neurotic. In other words, the neurotic starts a repair attempt with more anxiety than an emotionally stable individual and, given the same efficacy of repair, the neurotic still has more anxious affect than the stable individual following repair.

Error Reactivity

In this study, I utilized two different tasks, the Stroop and AX-CPT, to obtain measures of error reactivity. Both tasks yielded a measure of the tendency to make errors in strings and a measure of the tendency to slow down following an error. Unfortunately, the tendency to make errors in strings did not correlate between tasks; the same result was obtained for the measure of post-error slowing. This is unusual considering that similar results have been obtained with error reactivity measures from both the Stroop (Compton et al, 2008) and AX-CPT (Augustine & Larsen, 2010). However, this is not unusual when reaction time based measures are considered in a broader context. While obviously not widely publicized due to a lack of significant findings, many cognitive tasks that are purported to tap the same underlying psychological constructs are not

correlated (Larsen, Augustine, Prizmic, & Bono, 2010). Despite this measurement issue, these tasks still predict important outcomes, predict outcomes in similar manners, and are widely used in a number of literature areas. Thus, despite the seeming disconnect between my measures of error reactivity, it is still possible that they would each predict the affective tendencies measured in this study, and do so in the same manner.

In line with this, the tendency to make errors in strings, as assessed using the AX-CPT, was related to both neuroticism and trait anxiety, such that those higher in these traits were more likely to make errors in strings. The tendency to make errors in strings, as assessed using the Stroop, was not related to either personality variable. In addition, post-error slowing, as assessed using the AX-CPT, was related to both neuroticism and trait anxiety, such that those higher in these traits were less likely to slow down following an error. These effects were not observed for the Stroop. The disconnect between correlates of measures stemming from the AX-CPT and Stroop is possibly due to the lack of a relationship between measures from these tasks. It may be that the AX-CPT is reliably measuring error-reactivity processes while the Stroop is not. However, past research and theory has indicated that, while the tendency to make errors in strings should be related to neuroticism, post-error slowing should only show an indirect relationship with neuroticism (Compton et al., 2008; Augustine & Larsen, 2010). It may be that this indirect relationship between post-error slowing and neuroticism was observable in a direct manner due to the large sample size of this study. In any case, measures of error reactivity taken from the AX-CPT are displaying relationships with other study variables that are consistent with past research.

Mechanisms Underlying Personality and Error Reactivity Relationships

To test whether associations between error reactivity and neuroticism are due to reactivity processes, I examined the degree to which error reactivity predicted participants' reaction to the anxiety induction. Neither the tendency to make errors in strings nor post-error slowing predicted affect reactivity for anxious, negative, or positive affect. Thus, affect reactivity processes do not seem to be driving the relationship between neuroticism and the tendency to make errors in strings.

To examine the possibility that shared regulatory systems are driving relationships between neuroticism and error reactivity, I examined the degree to which error reactivity predicted participants' ability to repair the anxious affect that resulted from the affect induction. No main effects on the repair of any type of affect (anxious, negative, or positive) and no condition interactions were observed for the tendency to make errors in strings or post-error slowing. Despite this, and in an attempt to further understand the nature (or lack thereof) of the results, the degree to which each variable predicted affect repair in each condition was examined.

Both measures (AX-CPT and Stroop) of the tendency to make errors in strings were related to anxiety repair in the control condition. However, this effect is in the opposite direction as would be expected based on past research; those who are more likely to make errors in strings experienced less anxious affect following the control condition manipulation. Additionally, this tendency, as measured by the Stroop, predicts positive affect repair in the distraction condition, such that those who are more likely to make errors in strings were lower in positive affect following the distraction condition manipulation. This effect is consistent with the notion that those with a greater tendency

to make errors in strings would be less able to engage in effective affect repair. Thus, while the tendency to make errors in strings does seem to relate to affect repair ability, the effects are not consistent in direction, nor are the effects totally consistent with past research.

For post-error slowing as assessed with the Stroop, the tendency to slow down following an error predicts anxiety repair in the control condition and positive affect repair in the distraction condition, such that those with a greater tendency to slow down following an error displayed higher levels of anxiety and positive affect. Additionally, post-error slowing on the AX-CPT predicts predicts the repair of all affect types in the control condition, such that those with a greater tendency to slow down following an error displayed higher levels of anxiety and negative affect, and lower levels of positive affect. Finally, post-error slowing on the AX-CPT predicts positive affect repair in the distraction condition, such that those with a greater tendency to slow down following an error were lower in positive affect. Those with a greater tendency to slow down following an error should, in theory, display greater affect repair ability. Post-error slowing interacts with neuroticism, such that those high in neuroticism and high in post-error slowing show lower levels of daily distress (Robinson, et al., 2007). In other words, when one slows down following an aversive stimulus (like an error, or signal of negative affect), one has more time to engage regulatory mechanisms, be they cognitive or affective, on an ensuing task/trial. However, the majority of the within condition findings for post-error slowing indicate the opposite tendency, these individuals are less able to effectively engage in affect repair. Thus, as was seen with the results for the tendency to

make errors in strings, while post error slowing does seem to be related to affect regulation ability, these effects are not in the expected direction.

There are several potential explanations for the disconnect between previous research and the findings of this study. The first possible explanation is that our manipulations were unsuccessful. However, the anxiety induction successfully increased anxious and negative affect, and decreased positive affect. In addition, the study replicated known effects for the effectiveness of different types of affect regulation strategies. Thus, both the induction and repair task were successfully implemented. The second possible explanation is that the design of our study did not allow for the emergence of individual difference effects. Again, this is not the case. The finding that neuroticism predicted both anxiety reactivity and repair ability suggest that the study was indeed able to detect individual differences in these processes. Third, it is possible that there is simply no link between error reactivity and the parameters of affective responding. Given the lack of effects for affect reactivity and the inconsistent nature of effects for affect regulation ability, this is certainly a possibility. However, this study did replicate the relationship between neuroticism/trait anxiety and the tendency to make errors in strings. In addition, numerous studies have found associations between error reactivity and affective processes (Robinson, et al., 2007; Compton et al., 2008; Augustine & Larsen, 2010). However, the nature of this past research points to yet another possible explanation for the lack of and/or inconsistent nature of the findings in this study. All previous research on the connection between error reactivity and affective processes has relied on field studies, in the form of intensive time sampling (i.e., daily diary, experience sampling, ecological momentary assessment). It may be that, by taking

the reactivity and regulatory processes out of the real world and into a relatively contrived laboratory setting, some level of complexity was lost. The complexity inherent in naturalistic settings, as well as the aggregation of experience through repeated measurement, may be required to observe these associations. The specific components of a natural setting that would allow the emergence of effects are unknown, and I am hesitant to venture a guess as to what exactly might explain the dissociation between research settings. However, given the number of findings linking error reactivity and affective experience, and especially considering that some effects were replicated in this study, it is somewhat unlikely that there is simply no link between error reactivity and the parameters of affective responding. With a shortage of other explanations, the change of research setting seems the most likely explanation for the lack of and/or inconsistency of the findings of this study.

Implications and Future Directions

The findings of this study have several implications for both the existing literature and future research regarding affect reactivity and regulation. First, if the lack of findings in this study is related to a removal of these processes from a naturalistic setting, then this should be verified. It may be possible to test both a reactivity and a regulation ability explanation using intensive time sampling. By using event contingent as well as random time sampling, one could potentially tease apart these two explanations. In other words, if participants were randomly sampled for emotions to provide baselines and covariates, and participants recorded their reactions when they encountered affective events (event-contingent recording), then one could potentially examine these hypotheses. However, the design and conductance of this type of study would be extremely complicated and,

perhaps, unfeasible. As such, with the possibility that the exact nature of error reactivity - affect relationships can not be determined through laboratory based research, the mechanisms underlying these associations may remain unclear.

The results of this study also have implications for the study of affect regulation in general. My results revealed that, as with global negative affect, reappraisal and distraction were the most effect means for repairing anxious affect. There is a dearth of research examining the efficacy of affect regulation strategies for the repair of specific types of affect. This study provides one of the first attempts to extend research on global affect regulation into the study of specific affects. While these results were consistent with effects for global affect, it is possible that other specific negative affects (i.e., disgust, anger, etc.) may be better alleviated using other types of affect regulation strategies (i.e., distancing, social support, etc.). Future research should continue to examine the effects of different types of affect regulation strategies on specific affects.

By looking at anxiety in particular, these results also shed light on the individual difference variables that drive the ability to use affect regulation strategies. I found that neuroticism predicted the ability to repair negative, but not anxious affect following an anxiety induction. This deviates from the relatively simple picture that one might construct of the highly neurotic individual; they are not simply higher in reactivity and lower in repair ability. While the neurotic may show deficits in the ability to repair global negative affect, they do not show deficits in the ability to repair anxious affect. This is in line with other findings concerning the implementation of affect regulation attempts that show that the neurotic also engages in regulation during periods of high negative affect (Augustine & Larsen, in press). Thus, the picture for the highly neurotic

individual is not all bad. While they may show certain affective deficits, there are also instances in which the neurotic individual seems to show a level of healthy affective functioning on par with their emotionally stable counterparts. As research on the affective behaviors associated with neuroticism continues, more efforts should be made to determine, not only those instances wherein the neurotic individual shows deficits, but also those instances wherein the neurotic is relatively successful in managing their affective states.

Finally, the results of this study have implications for the use of a number of computerized tasks. The lack of a relationship between a number of tasks that are supposed to be measuring the same underlying constructs should be of major concern to researchers using these tasks. If viewed from a measurement perspective, it does not matter if these tasks are predicting outcomes in a similar or even consistent manner. If they are intended to measure the same construct then they should be at least marginally correlated; the move from questionnaire based responding to a reaction time or error based measurement does not excuse these tasks from the basic psychometric requirements of valid measurement. With relatively low test-retest values and few significant correlations amongst these tasks, it may be necessary to go back to the proverbial drawing board. Although the study of "button-pushing psychology" continues to grow at a rapid pace, a step back to examine the measures and tasks used may be required.

In sum, error reactivity and affective experience do seem to be related. Although the tendency to make errors in strings has been consistently related to neuroticism and to real world affective responses in past research, this study failed to determine whether two

specific mechanisms underlie these associations. Hopefully, continued research into the relationships between affective and cognitive response tendencies will eventually yield an explanation for these effects.

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Table 1

Affect data

	Across Conditions	Distraction Condition	Reappraisal Condition	Control Condition
NA Time 1	1.40 (.45)	1.38 (.42)	1.41 (.51)	1.43 (.43)
NA Time 2	1.47 (.54)	1.44 (.56)	1.53 (.66)	1.45 (.38)
NA Time 3	1.28 (.41)	1.21 (.39)	1.29 (.47)	1.35 (.34)
Anxiety Time 1	1.83 (.62)	1.85 (.63)	1.86 (.65)	1.80 (.61)
Anxiety Time 2	2.02 (.76)	2.11 (.81)	1.94 (.78)	2.02 (.68)
Anxiety Time 3	1.71 (.63)	1.59 (.57)	1.62 (.59)	1.92 (.68)
PA Time 1	1.93 (.80)	1.93 (.81)	1.93 (.84)	1.95 (.75)
PA Time 2	1.72 (.78)	1.78 (.89)	1.69 (.80)	1.68 (.66)
PA Time 3	1.99 (.86)	2.24 (.94)	1.98 (.87)	1.75 (.70)

Note: N = 208; NA = negative affect, PA = positive affect; all data are presented as Mean (SD)

Table 2

Neuroticism, Trait Anxiety, and Repair Effectiveness

		Neuroticism			Trait Anxiety		
Level		Anxiety	NA	PA	Anxiety	NA	PA
1	Affect T2	.64 (12.02*)	.67 (12.94*)	.76 (16.97*)	.64 (12.02*)	.67 (12.94*)	.76 (16.97*)
2	ID	.02 (.37)	.11 (-2.01*)	.05 (.95)	-.03 (-.43)	.01 (.16)	.06 (1.37)
	Condition	-.24 (-4.66*)	-.13 (-2.46*)	.19 (4.51*)	-.24 (-4.69*)	-.13 (-2.49*)	.19 (4.49*)
3	ID x Condition	-.19 (-1.42)	-.15 (-1.09)	.05 (.42)	-.02 (-.15)	-.01 (-.06)	.01 (.07)

Note: $N = 208$; data presented as β (t); *$p < .05$; ID = individual difference predictor, NA = negative affect, PA = positive affect

Table 3

The Tendency to make Errors in Strings and Repair Effectiveness

		AXCPT Repeated Errors			Stroop Repeated Errors		
Level		Anxiety	NA	PA	Anxiety	NA	PA
1	Affect T2	.64 (11.78*)	.67 (12.80*)	.76 (16.79*)	.64 (11.78*)	.67 (12.80*)	.76 (16.79*)
2	ID	-.02 (-.40)	-.03 (-.60)	-.02 (-.34)	-.08 (-1.58)	-.02 (-.28)	-.06 (-1.29)
	Condition	-.23 (-4.42*)	-.13 (-2.42)	.20 (4.53*)	-.23 (-4.48*)	-.13 (-2.46*)	.20 (4.55*)
3	ID x Condition	-.09 (-.39)	-.14 (-.65)	.29 (1.59)	.19 (.58)	-.02 (-.08)	-.14 (-.51)

Note: $N = 208$; data presented as β (t); *$p < .05$; ID = individual difference predictor, NA = negative affect, PA = positive affect

Table 4

Post-Error Slowing and Repair Effectiveness

		AXCPT Post-Error Slowing			Stroop Post-Error Slowing		
Level		Anxiety	NA	PA	Anxiety	NA	PA
1	Affect T2	.64 (11.98*)	.67 (12.85*)	.77 (17.02*)	.64 (11.98*)	.67 (12.85*)	.77 (17.02*)
2	ID	.04 (.76)	.04 (.82)	-.05 (-1.05)	.02 (.36)	.02 (.37)	.02 (.45)
	Condition	-.23 (-4.42*)	-.13 (-2.45*)	.20 (4.52*)	-.24 (-4.60*)	-.13 (-2.48*)	.19 (4.46*)
3	ID x Condition	.04 (.31)	-.08 (-.58)	.09 (.76)	-.01 (-.09)	-.14 (-.98)	.07 (.58)

Note: $N = 208$; data presented as β (t); *$p < .05$; ID = individual difference predictor, NA = negative affect, PA = positive affect

Table 5

Predictors of Affect Repair Effectiveness in each Condition

ID	Affect	Control	Reappraisal	Distraction
Neuroticism	Anxiety	.08 (1.43)	-.01 (-.24)	-.01 (-.23)
	NA	.08 (1.56)	.11 (1.97*)	.01 (.12)
	PA	.01 (.26)	.03 (.73)	.03 (.60)
Trait Anxiety	Anxiety	-.01 (-.08)	-.03 (-.55)	.01 (.02)
	NA	-.01 (-.16)	.03 (.63)	-.01 (-.12)
	PA	.03 (.71)	.06 (1.36)	.02 (.52)
Stroop Repeated Errors	Anxiety	-.39 (-2.56*)	-.19 (-1.28)	-.12 (-.75)
	NA	-.13 (-.83)	-.01 (-.07)	.02 (.12)
	PA	-.05 (-.36)	-.18 (-1.37)	-.27 (-2.10*)
AXCPT Repeated Errors	Anxiety	-.18 (-2.13*)	-.02 (-.26)	.07 (.81)
	NA	-.14 (-1.64)	-.02 (-.20)	-.01 (-.12)
	PA	.10 (1.35)	-.02 (-.32)	-.10 (-1.34)
Stroop Error Slowing	Anxiety	.13 (2.32*)	.00 (.02)	-.10 (-1.72)
	NA	.09 (1.68)	.01 (.22)	-.07 (-1.30)
	PA	-.08 (-1.68)	.02 (.31)	.12 (2.57*)
AXCPT Error Slowing	Anxiety	.13 (2.41*)	.02 (.36)	-.04 (-.71)
	NA	.12 (2.29*)	.00 (.02)	-.01 (-.21)
	PA	-.11 (-2.49*)	-.08 (-1.66)	.09 (1.96*)

Note: $N = 208$; data presented as β (t); *$p < .05$; ID = individual difference predictor, NA = negative affect, PA = positive affect

Figure 1

Experimental Procedure