THE INFLUENCE OF TRAIT APPROACH & AVOIDANCE MOTIVATION ON THE COURSE OF DEPRESSION AND ANXIETY

BY

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DISSERTATION

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ABSTRACT

Trait approach and avoidance motivation are higher-order individual differences that are related to personality, emotional temperament, and basic drives (i.e., sensitivity to pain and pleasure). Previous research has shown that approach and avoidance motivation are related to depression and anxiety, but the question of how trait motivation affects these dimensions of psychopathology has yet to be answered. The present study aimed to begin to answer this question by identifying potential neural mechanisms that could explain this relationship. Dimensional measures of depression (i.e., depressive loss of interest, depressive low positive affect) and anxiety (i.e., anxious arousal, anxious apprehension) were gathered at two time-points. Neural data and measures of trait approach and avoidance were gathered at the first time point. Trait avoidance motivation was associated with increases in both dimensions of depression and anxious arousal, and trait approach motivation was associated with decreases in depressive low positive affect. An adaptive balance between approach and avoidance motivation (i.e., more approach relative to avoidance) was generally associated with decreases in both dimensions of depression and anxious arousal. Neural activity during the anticipation of punishments and the receipt of disappointing feedback mediated the relationship between this adaptive balance and changes in anxious arousal and depressive low positive affect, respectively. Regions that mediated changes in anxious arousal were part of neural networks associated with self-referential processing, inhibition, and the integration of emotional information with goals (e.g., default mode network). Regions that mediated changes in depressive low positive affect were associated with processing the somatic aspects of emotion. Results suggest that those with an adaptive balance between trait approach and avoidance motivation engage with negative or disappointing information and that this engagement is protective against worsening symptoms of depressive low positive affect and anxious arousal. These findings are in line with theory undergirding therapeutic approaches that encourage engaging with feared or unpleasant information as opposed to avoiding it. Furthermore, these findings show that trait approach and avoidance motivation are associated with a broad network of brain regions related to important aspects of emotional experience and that these networks may be fruitful targets for future mechanistic and therapeutic research.

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INTRODUCTION

Trait approach and avoidance motivation

The constructs of approach and avoidance have been theorized to be the basic drivers of behavior (e.g., Bentham, 1779/1879; Freud, 1915; James, 1890) and have more recently been referred to as temperaments (Elliot, 1999; Elliot, 2006; Elliot & Thrash, 2002, 2010) or as individual differences in motivational tendencies (Spielberg et al., 2011a, b, 2012). The present project follows Spielberg and colleagues (2011a, b, 2012) and conceptualizes these factors as trait-level individual differences in motivational tendency. Conceptually, approach motivation is characterized by sensitivity to, vigilance for, affective reactivity to, and behavior directed toward appetitive stimuli. Avoidance motivation is characterized by a similar sensitivity to, vigilance for, and affective reactivity to aversive stimuli, with behavior directed away from such stimuli (e.g., Elliot, 2006; Elliot & Thrash, 2002, 2010, cf. Harmon-Jones, Harmon-Jones, & Price, 2013). For example, someone with predominantly trait approach motivation would be more reactive to stimuli associated with positive outcomes and would be motivated by the pursuit of positive outcomes. According to theory, trait approach motivation can include the experience of negative emotions (e.g., frustrated non-reward), but would make the experience of positive emotions more likely (Elliot & Thrash, 2002).

These motivational dispositions are hypothesized to arise early in life, and while they are thought to be stable (Bates, 1987; Buss & Plomin, 1984; Elliot & Thrash, 2010, Spielberg et al., 2011a) and heritable (for a review, see Clark & Watson, 1991) they are also influenced by maturation and experience (Henderson & Wachs, 2007; Roberts & Jackson, 2008; Rothbart & Bates, 1998). Given the hypothesized impact that approach and avoidance may have on psychological well-being across development, much research has been devoted to investigating the role of these motivational factors in the etiology of psychopathology, and specifically, depression and anxiety (Barlow, 2002; Beck, 1967; Caspi, Moffitt, Newman, & Silva, 1996; Clark et al., 1994; Henriques & Davidson, 2000; Krueger et al., 1996; Rothbart & Bates, 2008; Spielberg et al., 2011; Trew, 2011; Whittle, Allen, Lubman, & Yucel, 2006; and for reviews, see Barlow et al., 2014; Clark, Watson, & Mineka, 1994; Nigg, 2000, 2006).

Dimensional conceptualization of depression and anxiety

Depression and anxiety are two of the most common (Kessler et al., 2005) and costly (Kessler, DuPont, Berglund, & Wittchen, 1999; Simon, Ormel, VonKorff, & Barlow, 1995) manifestations of psychopathology. However, research is still clarifying the individual difference factors that can affect and maintain their course. The goal of this project is to understand whether trait approach and avoidance motivation serve as risk factors that affect the course of depression and anxiety. Research on the cross-sectional and longitudinal relationships between motivation and these two types of psychopathology has revealed inconsistent findings, especially in the domain of depression (Dickson & MacLeod, 2004; Henriques & Davidson, 2000; Layne et al., 1982). Reasons for this inconsistency may be the heterogeneous nature of both depression and anxiety as well as the difficulty of measuring trait approach and avoidance motivation (Elliot, 2006; Elliot & Thrash, 2002; Davidson et al., 1999; Heller et al., 1997; Nitschke et al., 1999a, 2001). The present project aims to clarify this inconsistency by using dimensional, empirically-supported operationalizations of anxiety and depression as well as multivariate estimates of trait approach and avoidance motivation that can better account for error in measurement of these constructs. If these measurements are indeed more precise estimates of these constructs, it will allow for more accurate estimates of the relationships among them and a clearer test of whether aspects of trait motivation act as risk-factors that affect the course of depression and anxiety. If successful, this project will provide insights into the mechanisms of the course and maintenance of depression and anxiety, which may influence theory, treatment planning, and treatment development.

In this project, anhedonic depression is operationalized as the dimensions of low positive affect and depressive loss of interest. Studies examining the construct validity of measures assessing these dimensions of depression support low positive affect as specific to depression and distinct from anxiety (Clark & Watson, 1991; Nitschke et al., 2001). Depressive loss of interest has been found to be strongly associated with measures of negative affect and trait avoidance motivation (Nitschke et al., 2001; Spielberg et al., 2011a), making it less specific to depression. However, scales measuring this construct comprise symptoms relevant to the diagnosis of depression (Bredemeier et al., 2010) and have been used productively in research with trait approach and avoidance motivation (Spielberg et al., 2011a). Factor analyses has found that scales measuring depression (i.e., Mood and Anxiety Symptom Questionnaire – Anhedonic Depression scale [MASQ-AD] can be separated into distinct subscales associated with low positive affect and depressive loss of interest (Nitschke et al., 1999). The present project will therefore use these two dimensions of depression to test hypotheses about the relationship between trait approach and avoidance motivation and symptoms specific to depression (i.e., low positive affect) and symptoms that may be shared with other manifestations of psychopathology (i.e., high negative affect).

Similarly to depression, anxiety can be separated into two dimensions: future-oriented worry (anxious apprehension; Andrews & Borkovec, 1988) and somatic hyperarousal and tension (anxious arousal; Watson et al., 1995), each of which has been demonstrated to be trait-like in their temporal stability within individuals (Sharp, Miller, & Heller, 2015). This dimensional conceptualization of anxious apprehension and anxious arousal is supported by psychometric factor analysis showing distinct item loadings (Nitschke et al., 1999), and by psychophysiological methods showing differential patterns of lateralized neural activity – with anxious apprehension associated with left prefrontal cortex (PFC)activity and anxious arousal with right parietotemporal activity (Engels et al., 2010; Heller & Nitschke, 1998; Heller et al., 1997, Nitschke et al., 1999). Differences in neural lateralization have also been associated with trait approach and avoidance motivation – with approach motivation associated with left PFC activity and avoidance motivation associated with right PFC activity (Spielberg et al., 2011b; and for reviews see Davidson & Irwin, 1999; Heller, 1993). The present project examined the relationship between trait motivation and these two empirically-supported dimensions of anxiety in an effort to better understand the impact of trait motivation on the course of anxiety.

Operationalization of approach and avoidance motivation and their relationship to psychopathology

Many methods have been used to measure the constructs of trait approach and avoidance motivation (Elliot & Thrash, 2002). For example, researchers interested in personality have used extraversion and neuroticism scales (NEO-Five Factor Inventory, McCrae & Costa, 2004). In contrast, those researching basic motivational drives have examined the behavioral activation and inhibition system scales (BIS-BAS; Carver & White, 1994; Gray, 1982). In turn, work on stable experience and expression of emotion has employed positive and negative temperament scales (General Temperament Survey, Watson & Clark, 1993). Using structural equation modeling (SEM), Elliot and Thrash (2002) demonstrated that the common core of these three scales could be represented by trait approach and avoidance motivation. The present project also uses SEM as it provides estimates of trait motivation while effectively controlling for errors in measurement of these constructs.

The inconsistencies in the relationship between motivation and psychopathology are most prominent for depression. For instance, depression has consistently been associated with low approach motivation (Davidson, 1998; Dickson & MacLeod, 2004; Henriques & Davidson, 2000; Layne et al., 1982; Meehl, 1975, Shankman et al., 2007, but see Layne, Gross, & Buckley, 1980). However, depression-related biases toward negative information (e.g., Gotlib & Joormann, 2010) have been suggested to potentially arise from decreases in trait approach motivation, increases in avoidance motivation, or both (Henriques & Davidson, 2000; Tomarken & Keener, 1998). Research investigating these possibilities has produced mixed findings with studies noting associations between depression and either increased avoidance motivation (Layne et al., 1982), decreased avoidance motivation (Henriques & Davidson, 2000), or no relationship between depression and avoidance (Dickson & MacLeod, 2004). One potential explanation offered in the literature for the inconsistent relationship between avoidance and depression is a lack of accounting for co-occurring anxiety (Clark & Watson, 1991; Engels et al., 2007).

Research on the contributions of trait approach and avoidance motivation to anxiety disorders is more consistent. Many anxiety disorders are theorized to have in common the fundamental characteristics of vigilance for, (Davidson, 2002, and for a review, see Barlow et al., 2014) and active or passive avoidance of, unpleasant stimuli (Mineka & Zinbarg, 2006). As is suggested by the tripartite model of depression and anxiety (Clark & Watson, 1991), it is possible that the relationship between depression and avoidance motivation is the result of the negative affect common to anxiety and depression. This model further suggests that low approach motivation is the most important risk factor for the development of depression (e.g., Davidson, 2002) and that approach motivation is likely unrelated to anxiety. Elucidating the neural processes associated with trait approach and avoidance and how these processes affect the course of depression and anxiety over time can clarify the role these motivational dispositions play as risk factors.

Despite a wealth of research describing the relationship between approach and avoidance and depression and anxiety, few studies have offered an account of these relationships that include both neural and longitudinal data (cf. Bress et al., 2013, McFarland et al., 2006). What follows is a review of the literature exploring the relationship among these constructs as well as the identification of psychological and neural processes that may explain this relationship.

It should be noted that approach and avoidance are similar to other constructs that have been related to the experience of emotion (e.g., promotion and prevention, positive affect and negative affect). Although research has shown these variables to have complex relationships among themselves and differential relationships with outcome variables (e.g., Eddington et al., 2007; Smits & Boeck, 2006, and for a review, see Corwell, Franks, & Higgins, 2014), theoretical support and empirical evidence exists suggesting that these labels represent overlapping constructs with similar effects on the course of depression and anxiety (Barlow et al., 2014; Brown, 2007; Brown & Barlow, 2009; Campbell-Sills, Liverant, & Brown, 2004; Chorpita, & Barlow, 1998; Elliot & Thrash, 2002, 2010; Gershuny & Sher, 1998; Griffith et al., 2010; Kasch et al., 2002; Shankman & Klein, 2003; Watson, Clark, & Carey, 1988). For the purpose of this review, the conceptual relationship between trait approach motivation and constructs used in other studies will be specified and the relationship between these constructs and outcome variables will be assumed to be similar for trait approach and avoidance motivation.

Cross-sectional relationships between trait motivation and depression and anxiety

It has been hypothesized that low levels of approach combined with high levels of avoidance are related to the experience of depression (Aldoa, Nolen-Hoeksema, & Schweizer, 2010; Davidson, 1998; Ferster, 1973; Fowles, 1988; Moulds et al., 2007; Pinto-Meza et al., 2006; Trew, 2011) while high levels of avoidance are related to the experience of anxiety (Clark & Watson, 1991; Davidson, 2002; Dickson & MacLeod, 2004). In support of the relationships between motivation, and depression and anxiety, a study using structural equation modeling found evidence of a positive relationship between trait avoidance motivation and depression, anxious apprehension and anxious arousal, while trait approach motivation was observed to have a negative relationship with depression (Spielberg et al., 2011a). It was suggested that the shared contribution of trait avoidance motivation to both disorders may explain, in part, the high degree of co-occurrence of depression and anxiety in the population (e.g., Clark & Watson, 1991; Kessler, 2004).

In another study using factor analysis, a large sample of patients diagnosed with mood and anxiety disorders filled out a series of questionnaires designed to capture variance associated with these disorders as well as with positive affect, negative affect, and autonomic arousal (Brown, Chorpita, & Barlow, 1998). A hierarchical structure was identified in the questionnaire data showing two higher-order factors, similar to trait approach and avoidance motivation (termed extraversion and neuroticism), above the lower-order factors indicated by symptoms of mood and anxiety disorders. Significant paths were observed between neuroticism (avoidance motivation) and all mood and anxiety disorders, including those characterized by anxious apprehension (generalized anxiety disorder) and anxious arousal (panic disorder), while extraversion (trait approach) was significantly related only to unipolar depression and social anxiety disorder (Brown, Chorpita, & Barlow, 1998). These data support the hypothesis that approach and avoidance represent higher-order individual difference factors (Elliot & Thrash, 2002, 2010) that significantly predict symptoms of psychopathology broadly and depression and dimensions of anxiety specifically.

Two constructs closely related to approach and avoidance motivation that have been thoroughly studied in the literature are the behavioral activation system (BAS) and behavioral inhibition system (BIS; Elliot & Thrash, 2010; Gray, 1982; Gray & McNaughton, 1996). The BIS/BAS scale (Carver & White, 1994) is one of the most common tools used to assess these constructs and has been found to be related to symptoms of depression and anxiety. In line with research on trait approach motivation and its relationship to depression (e.g., Henriques & Davidson, 2000), lower levels of the BAS have been found to be related to the severity of depressive symptoms in samples of depressed patients (Pinto-Meza et al., 2006) and predicted greater length of depressive episodes (Kasch et al., 2002, McFarland et al., 2006).

There is less evidence linking the BAS to anxiety (Clark & Watson, 1991); however, the BIS has been shown to be related to both depression and anxiety. Specifically, higher scores on the BIS have been found in those with depression (Johnson et al., 2003; Kasch et al., 2002) and anxiety (Johnson, Turner, & Iwata, 2003). Together, these studies and those reviewed above suggest that the relationships between trait motivation and depression and anxiety generally parallel the predictions made by the tripartite model (Clark & Watson, 1991) and Davidson and colleagues (e.g., Davison, 2002; Henriques & Davidson, 2000). Though the studies referenced above provide data on the relevance of these motivation-related constructs for the cross-sectional experience of anxiety and depression, longitudinal data are necessary to understand the temporal relationship among these variables. The present project aims to address this need by building on the literature describing the course of depression and anxiety as a function of trait approach/avoidance motivation.

Longitudinal relationships between approach/avoidance and depression and anxiety

Several studies have investigated the relationship between trait approach/avoidance and depression/anxiety using longitudinal techniques (De Beurs et al., 2000; Grasbeck et al., 1993; Kendler et al., 1993; Roberts & Kendler, 1999). Results generally support the tripartite model of depression and anxiety (Clark & Watson, 1991), with some exceptions (Gershuny & Sher, 1998; Joiner & Lonigan, 2000) and highlight a strong role for trait avoidance motivation in predicting internalizing disorders generally (for reviews, see Barlow et al., 2014; Nigg, 2006). Focusing first on depression, a study of the effects of maternal depression comparing children with "difficult temperaments" (i.e., high avoidance motivation) to children with "easy temperaments" (i.e., low avoidance motivation, high approach motivation) found a higher incidence of future depression in the former group when both groups had depressed mothers (Radke-Yarrow, 1998). The influence of parenting on the development of depression was also found in a study comparing children with low positive emotionality (i.e., low approach motivation) with high positive emotionality (i.e., high approach motivation). In this study, Lengua and colleagues (2000) found that parental rejection led to depression only in children with low levels of approach motivation. Similarly, low levels of the BAS have been found to be related to less spontaneous remission of depressive symptoms six (McFarland et al., 2006) and eight months after initial assessment (Kasch et al., 2002). These prospective studies suggest that high avoidance or low approach motivation, especially when combined with environmental stressors, are important factors in the development and maintenance of depression.

A similar relationship between trait avoidance motivation and environmental stressors has been found to exist for anxiety generally; however, few if any prospective studies have decomposed anxiety into anxious apprehension and anxious arousal. In a series of studies by Kagan and colleagues (Kagan, 1994; Schwartz, Snidman, & Kagan, 1999), it was found that 30% of subjects who were behaviorally inhibited (i.e., high levels on the BIS) as children went on to develop anxiety later in life. While high levels on the BIS did confer risk on its own, environmental factors exacerbated this risk. Behaviorally inhibited third to sixth graders were found to have a greater risk for developing anxiety if they experienced more stress in their daily lives (Brozina & Abela, 2006) and if their parents also struggled with anxiety (Biederman et al., 2001).

One potentially fruitful prediction emerging from the tripartite model posits that the interaction between trait approach and avoidance motivation will have a differential relationship with depression and anxiety. Specifically, as described above, high avoidance and low approach motivation will lead to depression, whereas high avoidance motivation alone is likely to lead to anxiety. Using measures of positive and negative affect, Joiner and Lonigan (2000) found that the interaction of these two individual differences predicted symptoms of depression but not anxiety at two-month follow-up. This study found no main effects of individual differences on the development of depression or anxiety. Another study using measures of extraversion and neuroticism found the interaction of these factors to predict both depression and anxiety at three-year follow-up (Gershuny & Sher, 1998). This work provides partial support for the tripartite model with regard to depression, but not anxiety. It also adds to the inconsistency of the data regarding the relationship between approach/avoidance motivation and depression and anxiety and points to a need to better characterize this relationship. Furthermore, though the studies reviewed above provide evidence of an effect of trait approach and avoidance motivation on the course of depression and anxiety over time, they do not clarify the specific neural/psychological processes that may be involved in this relationship. The ultimate goal of the present project is to identify such processes and clarify the inconsistencies present in the literature.

Neural and psychological relationships between approach/avoidance motivation and depression and anxiety

PFC involvement in trait approach motivation and depression

A great deal of cognitive and neuroscience research has explored the link between trait approach motivation and depression. Davidson (1993) and others (for a review, see Heller, 1993) have proposed a theoretical model of trait approach/avoidance motivation that describes the relationship between these individual differences and prefrontal cortex organization. Specifically, the right PFC has been implicated in the avoidance of undesirable outcomes, threat monitoring, and the generation of negative affect involved in depression and anxiety. The left PFC has been implicated in approach to desirable outcomes, and in the generation of positive affect that, when low, is involved in depression (Davidson, 2002, and for a review, see Davidson & Irwin, 1999). Supporting the relationship between approach motivation-related left PFC activity and depression, lesions in this area of the brain produce amotivational behavior and negative mood similar to depression (e.g., loss of initiative, indecision; pathological crying; sadness; Gainotti, 1972; Henriques & Davidson, 1991; Luria, 1973). Trait hypoactivation in the left PFC has also been hypothesized to contribute to the likelihood of experiencing emotions associated with approach motivation deficits (e.g., sadness, depression, decreased responsivity to reward; Trew, 2011). In line with these findings, depression has been found to be associated with lower levels of resting-state electroencephalographic (EEG) activity in left PFC (Henriques & Davidson, 1991). These data suggest a role for activity of left PFC in approach motivation and implicate dysfunction in this system in depression.

However, there is still debate about the nature of EEG asymmetry in the PFC and its relationship to both depression and trait approach motivation (Debener et al., 2000; Miller et al., 2013; Tomarken & Zald, 2009). For instance, a study investigating predictors of the course of depression found a measure of trait approach motivation (measured via the BAS scale), but not EEG asymmetry, to be predictive of a worse course of depression at six-month follow-up (McFarland et al., 2006). It is possible that EEG asymmetry is relevant to the current experience and symptoms of depression, but not to its development or progression. There is also a growing consensus in the literature that the PFC is too heterogeneous to be adequately characterized by a method comparing the gross activity of one hemisphere to the other (for a review, see Miller et al., 2013).

Given the documented longitudinal associations between trait approach motivation and depression (e.g., Kasch et al., 2002), it would be valuable to identify more specific neural processes that may represent vulnerabilities to the exacerbation of symptoms of depression. To this end, lower sensitivity to reward indexed by the amplitude of an EEG component referred to as the feedback negativity (FN) was found to be predictive of the development of depression at two-year follow-up in a sample of never-depressed adolescent girls (Bress et al., 2013). This relationship held even after controlling for baseline levels of depressive symptoms and neuroticism, which is closely related to trait avoidance motivation (Elliot & Thrash, 2002, 2010).

Although trait approach motivation was not measured directly in this study, sensitivity and responsivity to rewards is a central component of trait approach motivation (e.g., Elliot & Thrash, 2002). This lower sensitivity to reward in depression has been documented in a number of studies (Hundt et al., 2007; Shankman et al., 2007) and has also been shown to affect the ability of depressed individuals to adjust their behavior in response to rewards (Henriques & Davidson, 2000). Results of these studies suggest that disruptions in neural systems related to trait approach motivation may contribute to the development of depression independently of trait avoidance motivation.

Altered sensitivity to reward is not the only information processing bias related to trait approach motivation and depression (for a review, see Gotlib & Joorman, 2010). Measures of approach motivation (i.e., BAS sensitivity and extraversion) have been related to sensitivity to positive information more generally (Noguchi, Gohm, & Dalsky, 2006). Disruptions in neural systems involved in processing positive information may lead to the observed difficulty for those with depression to process such information (e.g., subtle positive facial expressions, Joormann & Gotlib, 2006) and recall positive memories (Bradley, Mogg, & Williams, 1995), resulting in less exposure to, or opportunities for, positive emotion and thus representing either a risk or maintenance factor for depression. In order to identify the specific neural systems involved in these biases and determine whether they act as risk or maintenance factors for depression, a neuroimaging technique with higher spatial resolution is needed. Functional magnetic resonance imaging (fMRI) is such a technique and has been used successfully to explore the neural relationship between trait approach motivation and depression (e.g., Spielberg et al., 2011b).

For instance, in support of Davidson and colleagues' observations that the brain is lateralized with respect to emotion processing (e.g., Davidson et al., 1990; Henriques &

Davidson, 1991), an area of left dorsolateral prefrontal cortex (DLPFC) was found to be sensitive to positive words using fMRI and direct tests of laterality of brain activity (Herrington et al., 2010). Extending this work more directly to trait motivation, Spielberg and colleagues (2011b) found activity related to engaging in an executive functioning task (i.e., color-word Stroop) in left DLPFC to be positively correlated with trait approach motivation. The authors concluded that this region of DLPFC is involved in biasing lower-order processes (e.g., paying attention to task-relevant information), and that this biasing is affected by motivational traits (Spielberg et al., 2011b). For example, someone with strong trait approach motivation may be driven to perform well by the desire to succeed and would think in terms of the presence or absence of success (as opposed to the presence or absence of failure, as in trait avoidance motivation; Elliot & Thrash, 2002). However, there are more regions involved in processing positive or pleasant information than the PFC.

OFC and Ventral Striatal involvement in trait approach motivation and depression

The orbital frontal cortex (OFC) and subcortical systems involved in Pavlovian and operant learning (e.g., ventral striatum) have also been associated with processing positive information, as well as with depression and trait approach motivation. Studies have shown the OFC to be sensitive to the perceived value of stimuli (Kringelbach et al., 2003; O'Doherty, 2004, 2007) and to updating value representations in novel situations (Schoenbaum & Esber, 2010). The ventral striatum has similarly been shown to respond to stimulus value and plays a critical role in learning from rewards (Delgado, 2007, and for a review, see Daniel & Pollman, 2014). Illustrating the importance of trait approach motivation in learning, positive correlations have been found between this trait and activity in both the OFC and the ventral striatum during the receipt of reward (Pizzagalli et al., 2009; Simon et al., 2010). Depression has been associated with lower OFC activation to happy faces (Fitzgerald et al., 2009) and lower levels of activity (Dougherty & Rauch, 1997; Drevets, 2001) and tissue volume (Drevets, 2001; Pizzagalli et al., 2009) in the ventral striatum. Further, using a large community sample of adolescents, Stringaris and colleagues (2015) found that decreased ventral striatal activity during the anticipation of reward was associated with current depression and predicted increased symptoms of depression at two-year follow-up. This evidence suggests activity in regions of the brain sensitive to the value of stimuli varies as a function of trait approach motivation and plays a role in the course of depression.

However, despite the potential for the relationship between trait avoidance motivation and brain activity to be a marker for the risk of developing depression, no study to date has related measures of trait approach motivation to activity in specific brain regions to predict changes in depressive symptoms. This project aims to investigate this relationship for the purpose of identifying areas of the brain that may be relevant to mechanisms that contribute to the course of depression.

PFC involvement in trait avoidance motivation and depression

Trait avoidance motivation and depression have been found to overlap in the way they affect information processing. Research has found trait avoidance motivation to be related to sensitivity to negative information (Noguchi, Gohm, & Dalsky, 2006) and difficulty disengaging from negative stimuli (Derryberry & Reed, 1994). Depression has also been shown to increase the salience of, and difficulty in, disengaging from negative material (for a review, see Gotlib & Joormann, 2010). However, this line of research has found these biases to be more common at the level of elaboration and retrieval of such material (Isen, 1984; Bower & Forgas, 2000) and to be not as common at the level of stimulus sensitivity (Mogg & Bradley, 2005; Gotlib, Yue, & Joorman, 2005). The presence of these biases has also been detected after remission of depression (Joorman & Gotlib, 2007), which suggests they may represent an enduring risk factor for the maintenance and relapse of depression.

Research has also identified overlap in the neural correlates of trait avoidance motivation and depression. Evidence exists that supports the role of the right PFC in trait avoidance motivation-related processing (e.g., Spielberg et al., 2011b). However, revealing the relationship between right PFC and depression has required more rigorous analytic techniques. For instance, using statistical tests of laterality on fMRI data, a region of right PFC was found to be more sensitive to negative words in a group of participants with high levels of depressive symptoms and low levels of anxiety (Herrington et al., 2010). A study using similar methods and analyses extended these findings and found greater right DLPFC activity to negative words as a function of depressive symptoms, but only when symptoms of anxious apprehension were low and symptoms of anxious arousal were high (Engels et al., 2010). However, in that same study, lower activity in the right inferior frontal gyrus (IFG) was found under the same conditions. The authors interpreted these findings to mean that depression increased the salience of the task-irrelevant negative information (greater right DLPFC activity) while interrupting processes normally involved in biasing processing toward task-relevant goals (lower right IFG activity; Aron et al., 2003; Aron, Robbins, & Poldrack, 2004). This interpretation has a number of implications; (1) depression and trait avoidance motivation may operate similarly in terms of over-valuing negative information, (2) the functional architecture of the PFC is complex and cannot be organized only according to valence (for a review, see Miller et al., 2013), and (3) in order to fully understand the neural correlates of depression, one must take co-occurring anxiety into account (Engels et al., 2010).

Amygdala involvement in trait avoidance motivation and depression

Other regions of the brain have also been implicated in both trait avoidance motivation and depression. One such region is the amygdala[1], a subcortical structure related to emotional processing and detecting salient (e.g., novel, threatening) stimuli in the environment relevant for the goals and motivations of the organism (e.g., Cain & LeDoux, 2008; Ernst & Fudge, 2009, and for a review, see Cunningham & Brosch, 2012). Both amygdala volume (Davidson, Pizzagalli, & Nitschke, 2002; Davidson et al., 2002) and increased amygdala activity have been associated with depression (Drevets, 2001; Krishnan & Nestler, 2008). It is possible this increase in amygdala activity results in biased estimates of the salience of emotional stimuli and leads to either a preference to avoid such stimuli or an increase in the perceived significance of these stimuli. Either manifestation of this biased processing has been hypothesized to contribute to depression by increasing felt experiences of negative events, increasing memories of those events, and motivating withdrawal behavior (Davidson, Pizzagalli, & Nitschke, 2002; Davidson, Pizzagalli, Nitschke, & Putnam, 2002; Drevets, 2001; Thase, 2009).

Involvement of attentional bias in trait avoidance motivation and dimensions of anxiety

As there is less empirical evidence and theoretical support for the link between trait approach motivation and anxiety (e.g., Davidson, 2002; Spielberg et al., 2011a), only the links

[1]The OFC has also been implicated in depression (e.g, Fitzgerald et al., 2008; Spielberg et al., 2014), but less direct evidence supports its role in avoidance motivation-related processing (Spielberg et al., 2012; but see Spielberg et al., 2011b).

between trait avoidance motivation and anxiety will be reviewed. Supporting theories based on the tripartite model of depression and anxiety (Clark & Watson, 1991), trait avoidance motivation was found to predict both anxious apprehension and anxious arousal (Spielberg et al., 2011a). Further, this relationship was strongest for anxious apprehension, highlighting the central role of avoidance in this dimension of anxiety (Andrews & Borkovec, 1988). This relationship may be explained by theories linking both trait avoidance motivation and anxiety to the vigilance for, and preferential processing of, threatening stimuli (Bradley et al., 1995; Elliot, 2006; Elliot & Thrash, 2002; Matthews et al., 1996).

These theories are supported by a large body of experimental evidence using a variety of tasks examining attention bias. For instance, trait anxiety, a construct closely related to both anxious apprehension (Heller et al., 1997) and trait avoidance motivation (Barlow et al., 2014), has been found to increase the likelihood that neutral stimuli are judged as threatening (Mogg & Bradley, 1998). Further, individuals high in trait anxiety (as well as those diagnosed with anxiety disorders) demonstrate an attentional bias toward threatening stimuli presented subliminally (Mogg et al., 1993) and supraliminally (for a review, see Zinbarg & Yoon, 2008), followed by efforts to direct attention away from such stimuli (MacLeod & Matthews, 2012; Matthews & MacLeod, 2005). These attentional processes have been found to recruit regions of the brain implicated in salience and top-down attentional control that have also been associated with trait avoidance motivation (Spielberg et al., 2014; and for a review, see Aupperle & Paulus, 2010).

Amygdala and PFC involvement in trait avoidance motivation and dimensions of anxiety

The amygdala has been shown to be involved in processing the salience of external stimuli (e.g., Gottfried, O'Doherty, & Dolan, 2002; Pessoa & Adolphs, 2010) and its tonic and phasic activity has been strongly associated with trait avoidance motivation (Davidson, 2002; Spielberg et al., 2012). This association, coupled with amygdala hyper-reactivity observed in disorders involving anxious apprehension (i.e., GAD, Nitschke et al., 2009) and anxious arousal (i.e., panic, Pfleiderer et al., 2007), suggests that the trait avoidance motivation-related increases in amygdala function may be related to the development of anxiety. However, no studies to date have examined whether trait avoidance motivation-related amygdala activity predicts change in anxiety over time.

Studies have also shown anatomical and functional connections between the amygdala and the PFC (for a review, see Davidson, 2002), and it has been proposed that the PFC can bias activity in this subcortical region to be in line with avoidance-related goals in the context of anxious arousal (Spielberg et al., 2012, 2014). In a study examining the level of functional connectivity between a region of DLPFC previously associated with trait avoidance motivation (Spielberg et al., 2012) and the amygdala, Spielberg and colleagues (2014) observed greater connectivity between these regions as a function of anxious arousal. This finding supports the hypothesis that DLPFC biases the amygdala to interpret potentially goal-irrelevant stimuli as salient and/or threating in the context of high anxious arousal. However, the authors acknowledge the correlational nature of this relationship. For example, it is possible that activity in the amygdala in response to salient cues increases activity in right DLPFC in a bottom-up fashion and correlations with trait avoidance motivation are driven by sensitivity of this subcortical region. Given the observation made by Davidson (2002) regarding the relationship between amygdala reactivity and measures of trait avoidance motivation, this is a likely hypothesis, which will be further explored in the present project.

Despite the relationship between anxious apprehension and trait avoidance motivation (Spielberg et al., 2011a), no association between anxious apprehension and DLPFC-amygdala connectivity was found (Spielberg et al., 2014). While seemingly contradictory, this finding is in line with the theory of anxiety-related brain activity put forward by Heller and colleagues (Heller, Etienne, & Miller, 1995; Heller & Nitschke, 1998; Heller, et al., 1997; Nitschke et al., 1999; Sharp, Heller, & Miller, 2015). According to this theory, anxious arousal should be associated with increased right PFC activity and anxious apprehension should be associated with increased left PFC activity. This theory has found empirical support (Engels et al., 2007; Isotani et al., 2001) and further studies have reconciled its seeming inconsistency with the theories associating trait approach/avoidance motivation with leftward and rightward lateralized neural activity. In particular, an fMRI study using an emotion Stroop paradigm was able to identify two separable regions in left PFC corresponding to anxious apprehension and positive affect, one inferior and the other superior, respectively (Engels et al., 2007). The authors attributed the anxious apprehension-related increase of activity in left inferior PFC (similar to Broca's area) to verbal rehearsal tendencies typically associated with this form of anxiety. This study adds to the literature supporting the hypothesis that the PFC comprises a functionally diverse set of regions (e.g., Engels et al., 2010), and suggests that trait motivational processes may be instantiated in more dorsal regions of the PFC (Spielberg et al., 2013).

AIMS AND HYPOTHESES

Though the research reviewed above illustrates the relationship between measures of trait approach/avoidance motivation and depression/anxiety, few studies have examined the interrelationship among these variables in the context of identifying risk for increasing symptoms of these disorders (cf., Bress et al., 2013). This represents a gap in the literature that this project intends to address using measures of psychopathology and fMRI data gathered at baseline (time 1) and measures of psychopathology gathered two to three years later (time 2). The present project has three primary aims.

Aim one is to extend previous work on approach and avoidance motivation (e.g., Elliot & Thrash, 2002; Spielberg et al., 2011a) and to identify whether these traits are risk-factors for increases in symptoms of depression and anxiety over time. Broadly, it is hypothesized that trait approach and avoidance motivation would influence the course of depression and anxiety in ways similar to those predicted by the tripartite model (Clark & Watson, 1991) and previous research (e.g., Spielberg et al. 2011a). Specifically, it is predicted that: higher trait avoidance motivation will lead to increases in anxious apprehension, anxious arousal, depressive loss of interest, and depressive low positive affect over a period of two to three years ($M = 2.4$); and lower trait approach motivation will lead to increases in depressive low positive affect over a period of two to three years. This time period was chosen to allow for potentially meaningful change to occur and to allow for a follow-up assessment before a majority of the participants (initially tested in their first year of college) graduated. Furthermore, follow-up assessment during this phase of life, which is characterized by transition (e.g., graduation from college) and increases in psychopathology (Kessler & Walters, 1998; Reinherz et al., 1999; Schulenburg & Zarret, 2006), increased the likelihood of detecting changes in depression. Given the contribution of avoidance motivation to depression, the interaction between the two motivational variables of low approach/high avoidance motivation should lead to the largest increases in depressive low positive affect (i.e., e.g., Gershuny & Sher, 1998).

As reviewed above, although approach and avoidance are hypothesized to operate independently, one process tends to predominate (Elliot & Thrash, 2002). It appears that the relative contribution of approach to avoidance motivation may capture the nature of anxiety and depression and is predictive of their course better than either process acting independently (Elliot, 2006, Elliot & Thrash, 2002, 2010). Therefore, in addition to deriving scores for approach and avoidance, their difference (approach minus avoidance) was calculated to produce a measure of how much one process dominates over the other. Given previous research implicating high approach and low avoidance as an adaptive combination (Elliot & Thrash, 2002; Spielberg et al., 2011a), this measure is referred to as the adaptivity score in the present study. This score may more effectively account for a situation in which strong avoidance processes may be balanced by strong approach processes resulting in null effects on psychopathology. Positive values of this score suggest the predominance of approach processes, whereas negative values suggest the predominance of avoidance processes. The higher the value of either a positive or a negative score, the stronger the predominance of one process over the other. Therefore, another specific prediction within aim one was that this adaptivity score will be negatively related to increases in anxious apprehension, anxious arousal, depressive loss of interest, and depressive low positive affect over a period of two to three years.

Aim two of this project was to examine whether trait approach and avoidance motivation affect performance on, and neural activity associated with, a monetary incentive-delay (MID) task which contained motivation-related anticipatory cues (i.e., possible monetary rewards and punishments), emotional stimuli (i.e., positively and negatively valenced as well as neutral words), and motivation-related consummatory cues (i.e., actual monetary rewards or punishments).

Hypotheses within aim two were focused on the relationships between approach and avoidance, and brain activity measured using fMRI. It was predicted that brain regions sensitive to valenced emotional and motivational stimuli would correlate with scores on approach and avoidance constructs. Specifically, it was predicted that trait approach motivation and the adaptivity score will be positively associated with neural activity during the anticipation and receipt of monetary rewards in salience and value processing regions (e.g., ventral striatum and OFC). It was also hypothesized that trait avoidance motivation will be positively associated with neural activity during the anticipation and receipt of monetary punishments. Specifically, increased reactivity of salience and value-processing regions (e.g., amygdala and OFC) during anticipation of monetary punishment will be positively associated with higher trait avoidance motivation and negatively related to the adaptivity score.

A further prediction within aim two was that blunted reactivity to reward in feedback-processing regions (e.g., ventral striatum) during both anticipatory and consummatory task periods will be associated with lower approach motivation and lower adaptivity scores. It was also predicted that trait approach/avoidance motivation will be correlated with lateralized activity in regions of the DLPFC during the emotional word period when the DLPFC is potentially involved in biasing activity in the regions specified above. In line with theories put forth by Davidson and Spielberg (Henriques & Davidson, 1991; Spielberg et al., 2012, 2014; Sutton & Davidson, 2000), left DLPFC activity will be associated with trait approach motivation and high adaptivity scores, and right DLPFC activity will be associated with higher avoidance motivation and lower adaptivity scores (see Engels et al., 2007).

As the neural correlates of trait approach/avoidance motivation rarely overlap given their hypothesized independence (e.g., Elliot & Thrash, 2002, 2010; Sutton & Davidson, 2000), the final prediction of aim two was that the only region that will correspond to their interaction is one identified as related to both types of motivation by Spielberg and colleagues (2011b) in the left DLPFC.

Alternatively, it is possible that patterns of brain activity described above associated with avoidance (and low adaptivity scores) will reflect a general disengagement with negative material. This disengagement has been observed in clinical literature in conditions such as social anxiety disorder and post-traumatic stress disorder (Badour et al., 2012; Goldin & Gross, 2010; and see Chawla & Ostafin, 2007 for a review). Further, as many treatments for such conditions involve increasing engagement with negative stimuli (i.e., increasing approach behavior), another alternative hypothesis is that higher trait approach motivation (and high adaptivity scores) will be associated with patterns of brain activity that suggest increased engagement with negative material (e.g., Badour et al., 2012).

Aim three of the present project was to determine whether neural activity at time 1 that varied as a function of approach and avoidance motivation mediated the relationship between trait motivation and changes in symptoms of psychopathology at time 2. If increased activity of salience and value-processing regions (e.g., OFC, ventral striatum, amygdala) during anticipatory task periods is observed as a function of avoidance or the approach-avoidance ratio, it was hypothesized that this activity will, in turn, predict greater increases in symptoms of both dimensions of depression and dimensions of anxiety. If approach-related activity is observed in right DLPFC during the emotion-word period, it was hypothesized that this activity will predict increases in symptoms of anxious arousal, but not anxious apprehension. If low approach motivation-related blunting of reward-related neural activity (e.g., ventral striatum, OFC) is observed, it was hypothesized that this activity will predict greater increases in both dimensions of depression. If avoidance-related activity is observed in right DLPFC during the emotion-word period, it was hypothesized that this relationship will predict increases in dimensions of depression. Lastly, if the interaction of approach and avoidance motivation is related to activity in left DLPFC during the emotion-word period, it was hypothesized that this activity will predict the greatest increases in dimensions of depression.

The general pattern described above is that high trait avoidance and low adaptivity scores will be related to the processing of negative material and that this relationship will lead to increases in psychopathology. In addition, low trait approach will be uniquely related to blunted activity in reward-processing regions and that this will in turn lead to increases in depressive low positive affect. An alternative set of hypotheses is that high avoidance and low adaptivity scores will be associated with decreased activity in value- and emotion-processing regions for negative material and that this relationship will result in increases in depressive loss of interest, anxious apprehension, and anxious arousal (e.g., Chawla & Ostafin, 2007). Furthermore, high approach and high adaptivity will be associated with increased activity in value- and emotion-processing regions for negative material and that this relationship will result in decreased depressive loss of interest (e.g., Badour et al., 2012).

The results of the present project could begin to reveal potential mechanisms by which approach and avoidance motivation affect cognition and the course of depression and anxiety. Furthermore, the results will provide evidence on whether the theories that inform present hypotheses are well-suited to explain the relationships among the constructs, as measured, of trait approach and avoidance motivation, depression and anxiety, and brain activity.

METHODS: RELATIONSHIP BETWEEN TRAIT MOTIVATION AND CHANGE IN PSYCHOPATHOLOGY

Subjects

Subjects were undergraduates recruited via the University of Illinois at Urbana-Champaign subject pool who gave written consent and completed a series of questionnaires, including the Positive Affect (PA) and Negative Affect (NA) subscales of the Positive and Negative Affect Schedule (PANAS; Watson et al., 1988) as partial fulfillment of enrollment in a psychology course. In order to be eligible for the present study, they had to either (1) score at or above the 80th percentile (≥ 29) on the NA subscale of the PANAS and at or below the 50th percentile (≤ 34) on the PA subscale; (2) score at or above the 80th percentile (≥ 41) on the PA subscale and at or below the 50th percentile (≤ 22) on the NA subscale; or (3) score at or below the 50th percentile on the NA and PA subscales (≤ 22 on the NA subscale and ≤ 34 on the PA subscale). Percentile cutoff scores were determined using a large sample of college students (N = 600). Group membership was not a factor in the present study and all analyses used continuous data. Individuals who gave written informed consent to participate were also given the option to be contacted for a follow-up study.

Those subjects who gave consent to be contacted for a follow-up study were reached via email or phone. The final sample used in analyses examining the relationship between psychopathology and trait motivation were those of the original sample who agreed to return, and had usable fMRI data from time 1 (T1, N = 43). Subjects were nearly equally split by gender (46% female), predominantly Caucasian (79%), with a mean age of 22 (SD = 0.95). The amount of temporal distance between time 1 and time 2 (T2) ranged from two to three years (M = 2.4, SD = 0.5). The study was approved by the University of Illinois at Urbana-Champaign IRB.

Subjects who still lived in the area or who were willing to travel came in to the laboratory (N = 33, 51% female) to complete a neuropsychological battery, a structured clinical interview for DSM-IV disorders (SCID-IV), and a series of online questionnaires. The remaining subjects (N = 10, 50% female) completed questionnaires and SCID-IV interviews online and via phone, respectively, and consented to research using an online procedure approved by the IRB. As this project was primarily concerned with dimensional manifestations of depression and anxiety, only scores from the questionnaires were used.

T1 Questionnaires

The research presented here is part of a larger study that asked subjects to complete a battery of questionnaires. Questionnaires of relevance to the present project, described below, include those used in Elliott and Thrash (2002, 2010) and Spielberg et al. (2011a, b) to obtain latent measures of trait approach and avoidance motivation. Specifically, subjects completed the 12-item Extraversion (NEO-E) and the 12-item Neuroticism (NEO-N) subscales from the NEO-Five Factor Inventory (NEO-FFI, McCrae & Costa, 2004). On each subscale, subjects rated how a series of descriptive statements was characteristic of themselves on a scale from 1 (strongly disagree) to 5 (strongly agree). The NEO-E scale consists of statements such as "I really enjoy talking to people," and the NEO-N scale consists of statements such as "I often feel tense and jittery."

Subjects were also asked to complete the Behavioral Inhibition System/Behavioral Activation System scale (BIS/BAS). Like the NEO-FFI, each item asks subjects how a series of descriptive statements is characteristic of themselves on a scale from 1 (very true for me) to 4 (very false for me). The 7-item BIS subscale consists of items such as "I feel pretty worried or upset when I think or know somebody is angry at me." The 13-item BAS subscale includes statements such as "When I want something I usually go all-out to get it."

The Positive Temperament (GTS-PT) and Negative Temperament (GTS-NT) subscales of the General Temperament Survey (GTS) were included as the final indicators of trait approach and avoidance motivation. This questionnaire asked subjects to indicate how true or untrue each item was of them (1 = true or mostly true, 2 = false or mostly false). The 27-item GTS-PT consisted of statements such as "I get excited when I think about the future." The 28-item GTS-NT consisted of statements such as "I frequently find myself worrying about things."

In order to obtain the two latent factor measures of trait approach and avoidance motivation, the standardized factor weights from a larger sample of undergraduates (N = 1,114; Spielberg et al., 2011a) were used (see Table 1). The raw scores from each questionnaire were centered and then multiplied by the standardized factor weight for that questionnaire (DiStefano, Zhu, & Mindrila, 2009). These values were then summed in accordance with the factor structure used in Spielberg and colleagues (2011a). BAS, NEO-E, and GTS-PT were used as primary indicators for approach temperament, with BIS and GTS-NT added to increase model stability (Spielberg et al., 2011a). BIS, NEO-N, and GTS-NT were used as primary indicators for avoidance temperament, with BAS added to increase model stability (Spielberg et al., 2011a). Similar to the relationship between trait approach and avoidance motivation observed by Spielberg and colleagues (2011a), present latent factors were negatively correlated ($r = -0.31$, $p < 0.001$). The difference score measuring the balance between approach and avoidance was calculated by subtracting the latent factor scores of avoidance from approach, such that higher scores on this difference measure reflect high approach and low avoidance. This relationship is illustrated by the negative correlation of this difference score with avoidance ($r = -0.86$, $p < 0.001$) and its positive correlation with approach ($r = 0.75$, $p < 0.001$).

To measure symptoms of depression and anxiety, a 39-item subset of the Mood and Anxiety Symptom Questionnaire (MASQ) and the 16-item Penn State Worry Questionnaire (PSWQ) were administered. The MASQ asked subjects to rate the frequency with which they experienced a symptom over the past week on a scale from 1 (not at all) to 5 (extremely). The MASQ was decomposed into subscales that measured depression and anxiety. Specifically, the 17-item anxious arousal scale, containing items such as "hands were cold or sweaty", the 8-item depressive loss of interest scale, containing items such as "felt like nothing was very enjoyable", and the 14-item depressive low positive affect scale, containing items such as "felt like I had a lot to look forward to" (reverse scored). The PSWQ asked subjects to rate how typical or characteristic each item was of them on a scale from 1 (not at all typical) to 5 (very typical). This scale contained items such as "I've been a worrier all my life".

T2 Questionnaires

In order to measure change in symptoms of depression and anxiety from T1 to T2, subjects completed the 39-item subset of the MASQ and the PSWQ. Questionnaires were identical in terms of item content and order at both T1 and T2. The only difference between the two time-points was the method of administration, with T1 administered via pencil and paper and T2 administered online (see Table 2 for correlations among all measures from T1 and T2).

Questionnaire analysis

Changes in symptoms of psychopathology were measured using scores from T1 as independent variables in the first step of a multiple regression analysis with scores from T2 as dependent variables. Trait approach and avoidance scores were entered in the second step and their interaction was entered at the third step. In this way, the variance of baseline symptom levels is controlled for and the analysis is a more valid representation of change over time. This approach provides an appropriate estimate of the relationship between change over time and trait motivation because it controls for variance in T1 symptoms of psychopathology that are likely related to trait motivation, and allows for the examination of rank-order change, which can capture change mean-level change misses (Roberts, Caspi, & Moffitt, 2001). This analysis was used to determine whether trait approach and avoidance, or an interaction of these factors, affected the course of depression and anxiety over time.

RESULTS: RELATIONSHIP BETWEEN TRAIT MOTIVATION AND CHANGE IN PSYCHOPATHOLOGY

Multiple linear regression analyses using approach and avoidance, their interaction, and adaptivity scores revealed that these trait scores predicted symptom increases in both depression and anxiety.

Depressive low positive affect: The equation with T2 depressive low positive affect as the dependent variable and T1 depressive low positive affect, approach, avoidance, and their interaction as the independent variables was significant (F(4, 41) = 16.34, $p = 0.00$, $f^2 = 1.63$), with an R^2 of 0.62. The interaction of approach and avoidance was not a significant predictor of change in depressive low positive affect ($\beta = -0.17$, $p = 0.11$). Higher levels of approach motivation predicted decreases in depressive low positive affect ($\beta = -0.29$, $p = 0.05$), and higher levels of avoidance motivation predicted increases in depressive low positive affect ($\beta = 0.48$, $p = 0.00$). The multiple regression equation with the adaptivity score as the independent variable and depressive low positive affect as the dependent variable was also significant (F(2, 43) = 30.81, $p = 0.00$, $f^2 = 1.44$), with an R^2 of 0.59. A higher adaptivity score predicted significant decreases in depressive low positive affect ($\beta = -0.66$, $p = 0.00$).

Depressive loss of interest: The multiple linear regression analysis with T1 depressive loss of interest and trait motivation scores as the independent variables and T2 depressive loss of interest as the dependent variable was significant (F(4, 41) = 5.47, $p = 0.00$, $f^2 = 0.54$), with an R^2 of 0.35. The interaction of approach and avoidance was not a significant predictor of change in depressive loss of interest ($\beta = -0.05$, $p = 0.74$). Higher levels of avoidance motivation predicted increases in depressive loss of interest ($\beta = 0.53$, $p = 0.00$), but approach motivation did not predict change in depressive low positive affect (β = 0.06, p = 0.69). The multiple regression equation with the adaptivity score as the independent variable and depressive loss of interest as the dependent variable was also significant (F(2, 43) = 7.67, p = 0.00, f^2 = 0.35), with an R^2 of 0.26. A higher adaptivity score predicted significant decreases in depressive loss of interest (β = -0.40, p = 0.04).

Anxious arousal: Multiple linear regression analysis using T1 anxious arousal and trait motivation scores as independent variables and T2 anxious arousal as the dependent variable was significant (F(4, 41) = 7.54, p = 0.00, f^2 = 0.72), with an R^2 of 0.42. As in the analyses above, the interaction of approach and avoidance was not significant (β = -0.17, p = 0.17). Higher levels of avoidance motivation predicted increases in anxious arousal (β = 0.33, p = 0.02), but approach motivation did not predict change in anxious arousal (β = -0.02, p = 0.88). The regression equation with the adaptivity score entered as an independent variable instead of approach and avoidance separately also significantly predicted changes in anxious arousal (F(2, 43) = 13.13, p = 0.00, f^2 = 0.61), with an R^2 of 0.38. A higher adaptivity score predicted significant decreases in anxious arousal (β = -0.26, p = 0.04).

Anxious apprehension: The multiple regression equation with T1 anxious apprehension and trait motivation scores as independent variables and T2 anxious apprehension as the dependent variable was significant (F(4, 41) = 19.43, p = 0.00, f^2 = 1.94), with an R^2 of 0.66. However, only T1 anxious apprehension was a significant predictor of T2 anxious apprehension (β = 0.64, p = 0.00). Neither the interaction of approach and avoidance (β = 0.00, p = 0.98), approach (β = 0.09, p = 0.37), nor avoidance (β = 0.22, p = 0.25) significantly predicted change in psychopathology. The equation using the adaptivity score as the independent variable along with T1 anxious apprehension also significantly predicted change in anxious apprehension (F(2,

43) = 38.15, p = 0.00, f^2 = 1.78), with an R^2 of 0.64. As above, only T1 anxious apprehension predicted T2 anxious apprehension (β = 0.80, p = 0.00), and the adaptivity score was unrelated (β = -0.01, p = 0.97) to changes in psychopathology.

As predicted, trait approach, avoidance, and the adaptive balance between the two predicted changes in dimensions of depressive low positive affect, depressive loss of interest, and anxious arousal[2]. Specifically, avoidance was positively related to worsening symptoms across all three dimensions and approach was negatively related to worsening depressive low positive affect. The adaptivity score showed negative relationships with worsening dimensions of depression and anxiety, with the strongest relationship observed for depressive low positive affect, which highlights the contribution of both forms of motivation to changes in this dimension of psychopathology. The next part of the project was designed to uncover the mechanisms by which the relationships between trait motivation and psychopathology observed above operate. To accomplish this, fMRI was used to measure neural responses in a paradigm that evoked the experiences of anticipating and receiving monetary feedback, as well as processing emotional material. Each of these processes has been hypothesized to be affected by trait motivation (Elliot, 1999; 2006; Elliot & Thrash, 2002; 2010; Spielberg et al., 2011b) and is relevant to the course of psychopathology (e.g., Davidson, 2002; Henriques & Davidson, 1991; 2000; Leventhal, 2008).

[2] As mentioned above, anxious apprehension showed a high degree of stability from T1 to T2, and thus no relationships were observed between this dimension of anxiety and trait motivation.

METHODS: FMRI IMAGING AND MEDIATION ANALYSES

fMRI Subjects

Before they were able to participate in the fMRI study, subjects were screened for a history of serious brain injury, abnormal hearing or vision, claustrophobia, left-handedness, metal in their body, pregnancy, and nonnative English-speaking. A total of 98 subjects completed the fMRI protocol. Criteria for exclusion were (1) movement of more than one voxel (2.13 mm) between adjacent fMRI volumes; (2) committing errors on 13% or more of the trials; or (3) poor structural MRI and fMRI registration. Thirteen subjects were excluded by these criteria and the final sample (n = 85) was predominantly Caucasian (93% non-Hispanic/Latino) with nearly equal numbers of male and female participants in the sample (49% female) and a mean age of 19.24 years (SD = 1.41).

fMRI Task

During fMRI acquisition, subjects completed a locally developed version of the monetary incentive delay task (see Figure 1) that consisted of a practice block of 24 trials followed by 3 blocks of 48 trials each, resulting in a total of 144 task trials. Each trial began with a cue signaling one of four potential monetary outcomes: (1) potential reward or punishment, (2) potential punishment only (3), potential reward only, or (4) neither reward nor punishment possible (see Figure 1). Cue phase probed the experience of the anticipation of future events.

A fixation dot followed the cue for a variable interstimulus interval (ISI) before a target emotion word (positive, neutral, or negative) appeared on screen and changed color after a variable amount of time. Subjects were told that outcome depended on whether they were able to push a button before the word changed color. The amount of time before the word changed color varied adaptively for each participant and trial block in a way that guaranteed a nearly equal number of successful and failed trails. The word phase probed the experience of emotional material. After another variable ISI, feedback was displayed and indicated to subjects whether they had won or lost money, there was no money change, or they had made an error (Burdwood et al., in press; Infantalino et al., in press; Figure 2). The feedback phase probed the experience of the receipt of performance-dependent monetary outcomes.

Cues did not indicate reward/punishment magnitudes, only the potential for reward or punishment. Rewards for successful trials ranged from $1.80 to $2.35 (mean: $2.08), and punishment for unsuccessful performance was associated with a monetary loss with the same range of values. Motivation on trials where there was no money at stake was maintained by informing subjects they could receive a bonus task block that would result in only monetary gain at the end of the three task blocks if their performance reached a certain threshold.

Duration of ISI, intertrial interval (ITI), and trial order was optimized with a locally written algorithm (based in part on a genetic algorithm, Wager & Nichols, 2003), which maximized signal detection for each contrast. Optimization was performed in four stages, with the first three stages optimizing the order of stimuli during each period and the fourth stage optimizing timing. For instance, the first stage was devoted to determining the optimal order of the cue period while holding timing and the other two periods constant. Optimal order was determined by the order of stimuli that maximized the mean design efficiency for contrasts possible during that period (e.g., reward vs. punishment). The next two stages proceeded the same way, and the fourth stage optimized the ISI and ITI for each trial. The optimization procedure was constrained to hold the transition properties between stimulus type equal (e.g., the probability of reward/punishment trials preceding no reward/punishment trials was equal to their preceding reward/no punishment trials). This optimization procedure ensured a balance between being able to deconvolve the blood-oxygen-level-dependent (BOLD) hemodynamic responses to each period in the task and balancing the psychological expectation of participants regarding stimulus order.

The 144 emotion target words were selected from the Affective Norms for English Words (ANEW) set (see Table 3; Bradley & Lang, 1999). Forty-eight positive (e.g., joy), 48 neutral (e.g., butter), and 48 negative (e.g., afraid) words were selected on the basis of established norms for arousal, valence, word length, and frequency of use in the English language (Bradley & Lang, 1999). Positive and negative words had equivalent arousal levels, $t(47) = .24$, $p = .81$, and each of these arousal levels was significantly higher than the arousal level of neutral words, $ts > 23$, $ps < .001$. Stimuli presentation and behavioral measurement were controlled by locally developed MATLAB code (version 2009a, The MathWorks, Natick, MA), using Psychophysics Toolbox (version 2.54; Brainard, 1997; Pelli, 1997).

Behavioral Data Analysis Plan

In order to measure the effects of trait motivation on behavior, average reaction time (RT) was calculated for negative words, positive words, and neutral words. Valence and arousal contrasts were created for the RT data by subtracting RTs to positive words from negative words, and the average of RTs to emotional words from neutral words, respectively. Trait approach and avoidance scores motivation were regressed onto these measures to elucidate whether they impacted processing of valenced or arousing stimuli. In order to understand whether brain activity is related to behavior, RTs were correlated with brain activity within significant clusters identified in the analyses described below.

fMRI Data Acquisition

MRI data were acquired using a Siemens Magnetom Trio 3T scanner. Two MPRAGE structural sequences were acquired (192 axial slices with isotropic spatial extent of 0.9 mm) for registering each participant's functional data to standard space. Gradient field maps were then collected to correct for geometric distortions in the functional data caused by magnetic field inhomogeneities (Jezzard & Balaban, 1995). A set of 331 functional imaging volumes were collected during each of the three task blocks (TR 3000 ms, TE 25 ms, flip angle 90°, FOV = 256 mm x 256mm) for a total of 993 functional images. Each image consisted of 50 oblique axial slices (slice thickness 2.40 mm, in-plane voxel size: 2.13 x 2.13 mm) acquired parallel to the plane containing the anterior and posterior commissures. Three volumes at the beginning of each task block were discarded to allow the scanner to reach steady state.

fMRI Data Reduction and Analysis Plan

MRI processing and statistical analyses were implemented using the FSL analysis package (http://fsl.fmrib.ox.ac.uk/fsl/fslwiki/). Functional data for each participant were motion-corrected in MCFLIRT (Jenkinson et al., 2002), temporally filtered with a 1/90 Hz high-pass filter, spatially smoothed using a 3-D Gaussian kernel (5 mm FWHM), slice-time-corrected, and fieldmap-corrected. Level 1 regression analyses were then performed for each block of each participant's preprocessed functional time series data using FILM (Woolrich et al., 2001). Statistical maps were generated via multiple regression computed for each intracerebral voxel. A separate predictor was entered for each cue type (resulting in 4 predictors), each emotion-word type crossed with the preceding cue type (resulting in 12 predictors), and each feedback type (success and failure) crossed with preceding cue type (resulting in 8 predictors). Three predictors of no interest were included to account for performance errors, one modeling each period of the trial (cue, word, feedback). Each predictor was convolved with a gamma function to approximate the temporal course of the blood-oxygen-level-dependent (BOLD) hemodynamic response function. A per-voxel effect-size parameter estimate (β) map representing the magnitude of activation was created for each predictor. Functional activation maps for each participant were then warped into a common stereotaxic space (the 2009 Montreal Neurological Institute 152 symmetrical 1 x 1 x 1 mm template, resampled to 2 x 2 x 2 mm; Fonov et al., 2009) using FNIRT (Andersson, Jenkinson, & Smith, 2007).

Thirteen contrasts were created to examine the response to the cue, word, and feedback period of the task. A reward contrast (only reward possible vs. neither reward nor punishment possible) and a punishment contrast (only punishment possible vs. neither reward nor punishment possible) were created to investigate the anticipation of monetary gain and loss. A motivational contrast was created to compare all cues with motivational value (the possibility of winning or losing money) to those without such value to assess the impact of motivational information. A valence contrast (positive words vs. negative words) and an arousal contrast (positive and negative words vs. neutral words) were created to investigate the effect of emotion. A contrast comparing all words preceded by rewarding cues to those not preceded by such cues was created to investigate how cueing approach-related processes would affect the word period. Similarly, a contrast comparing all words preceded by punishing cues to those not preceded by punishing cues was created. Two affect contrasts were created – a positive affect (positive words compared to neutral words) and a negative affect (negative words compared to neutral words) contrasts – to understand the effect of trait motivation on these two independent emotional dimensions. Finally, four outcome contrasts were created to investigate the receipt of monetary gain and loss. The first compared reward versus no change, which could include frustrative non-reward (being fast enough on a trial without the possibility of reward [i.e., both potential outcomes were neutral]) and relief (being fast enough on a trial with the possibility of punishment or neutral feedback). This contrast isolated the effects of experiencing rewarding feedback compared to neutral, non-punishing feedback. Similar to the first, the second compared punishment versus no change. The third directly compared the experience of reward to punishment, which will investigate how trait motivation affects this traditional gain vs. loss contrast. The last contrast compared all trials where participants received the best possible feedback (including neutral) to those trials where they received the worst possible feedback (including neutral). This contrast expands the definition of reward and punishment beyond monetary gain and loss and may capture a fuller psychological experience of these two constructs.

Level 2 fixed-effects analyses were performed to obtain an average of each contrast across the three task blocks within each participant. Level 3 statistical analyses were carried out using FLAME to compute the mean activation for each comparison across subjects. The effect of trait approach and avoidance motivation was assessed at each period of the task using level 4 statistical analyses that include these individual difference measures as continuous predictors within each contrast described above.

Two masks based on the Harvard-Oxford probabilistic atlas available in FSL were used to limit the number of voxels under consideration in order to help control family-wise error rate. First, a mask isolating a priori regions of interest including the PFC, OFC, anterior and posterior ACC, and basal ganglia regions was used to isolate areas of the brain that have been found to be associated with motivation, executive function, and salience/feedback processing. Second, a whole brain gray-matter mask was used for exploratory analyses to reveal what other brain regions may contribute to the relationship between psychopathology and trait motivation (e.g., posterior regions previously implicated in executive attention; e.g., Banich et al., 2000). Two-tailed t-tests were conducted on each β map and then converted to z-scores to determine the significance of the βs compared to zero. To correct for multiple comparisons, non-parametric thresholding of the β maps using the Monte Carlo permutation tool available in FSL (Randomise; Winkler et al., 2014) was implemented using 5000 permutations. The threshold-free cluster-enhancement (TFCE) statistic was used, which returns voxel-wise statistics that also takes into account cluster size (Smith & Nichols, 2009). This tool estimated the appropriate threshold for the TFCE statistic to set an overall family-wise error rate of 0.05 and an individual voxel Z-threshold of 2.32. Z-values for each significant cluster were averaged across all voxels to create a single score for each cluster for each participant. These data were then extracted and used in correlation analyses in SPSS 24. Labeling of brain regions comprising significant clusters was done using the Harvard-Oxford probabilistic structural atlas included in FSL. Brodmann's Areas were defined using the Yale BioImage Suite Package (Lacadie et al., 2008).

Mediation analysis: relationship between T1 brain activity, trait motivation, and change in psychopathology

Patterns of brain activity within each trial period (cue, word, and feedback) that significantly correlated with trait approach and avoidance motivation were used as mediator variables in analyses using the smaller follow-up sample. Brain activity was chosen as the mediator in these equations because it represents a meaningful but more costly estimate of the effect of motivation on psychopathology. If a relationship exists between present measures of motivation, brain activity, and changes in psychopathology that can be explained through a logical mechanistic framework, then results could begin to build an argument that these measures of motivation are cost-effective estimates of specific neural mechanisms. Chance of type I error was controlled by further limiting brain regions used in mediation analyses to those that displayed a relationship with change in symptoms of depression and anxiety. Rank order changes in depression and anxiety were used as dependent variables, and approach and avoidance as well as the adaptivity score were used as independent variables.

Significant clusters from both a priori and exploratory analyses were considered in mediation analyses. Any brain region identified in both *a priori* and exploratory analyses was only used in mediation analyses once. Furthermore, two types of mediation analyses were considered; 1) analyses in which activity from a single brain region was entered as a mediator between trait motivation and change in psychopathology, 2) analyses in which correlated brain regions that were each associated with change in the same type of psychopathology were entered into exploratory multiple mediation analyses. These exploratory analyses were performed to test whether activity in correlated regions jointly can account for a greater proportion of the indirect effects of trait motivation on changes in psychopathology than activity in each of these regions separately. All these analyses were intended to clarify how trait approach and avoidance motivation affect changes in depression and anxiety.

Brain activity was extracted from significant clusters using the fslmeants command in FSL and imported into SPSS. Analyses were performed in SPSS (Version 24, IBM, 2017) using the PROCESS macro (Hayes, 2008). The statistical significance of indirect effects (c-paths) was assessed using a bias-corrected bootstrap confidence interval based on 10,000 samples. The statistical significance of direct effects (c'-paths), relationships between independent variables and moderators (a-paths), and the relationships between moderators and dependent variables (b-paths) were assessed using standard 95% confidence intervals.

RESULTS: FMRI IMAGING AND MEDIATION ANALYSES

Reaction time and trait motivation

Examining the impact of approach and avoidance motivation scores on RT revealed that responses to arousing stimuli (the average of both positive and negatively valenced words contrasted against neutral words) were faster as a function of the adaptivity score ($r = 0.25$, $p = 0.015$) and avoidance motivation scores ($r = -0.22$, $p = 0.028$). No other relationship between RT, approach, avoidance, and the adaptivity score emerged.

Planned analyses: PFC plus basal ganglia and amygdala mask

Analyses using the adaptivity score as higher-level regressors were focused on hypothesized areas of interest within the prefrontal cortex, basal ganglia, and amygdala. Significant relationships were observed during the cue phase of the task. Specifically, those with higher trait approach than avoidance motivation showed more neural activity to cues containing the possibility of punishment than to cues lacking the possibility of punishment in right posterior cingulate, right orbital frontal cortex/frontal medial cortex, left paracingulate, left superior frontal gyrus, left frontal pole, and right frontal pole (Table 4).

Significant relationships were also observed during the word phase of the task for two different contrasts. Within the valence contrast, those with higher approach relative to avoidance motivation showed stronger neural responses to negative words relative to positive words in the right supplementary motor area. Within the positive affect contrast, those with higher approach relative to avoidance motivation showed stronger neural responses to neutral words than positive words in left primary motor cortex. No relationship between the adaptivity score and feedback was observed in these regions of interest.

When approach, avoidance, and their interaction were entered as higher-level predictors, several relationships were observed in the cue and word phases, but not the feedback phase, in the prefrontal cortex, basal ganglia, and amygdala. Activity unique to avoidance motivation was higher to cues that did not signal punishment as compared to those that did signal punishment in ventromedial PFC/medial OFC and the left frontal pole. Avoidance motivation was also significantly associated with neural activity correlated with processing positively valenced words compared to negatively valenced words in two regions within the left precentral gyrus and one region in left primary motor cortex (Table 4).

Relationship between RT and brain data

As the arousal score was the only RT contrast related to approach and avoidance scores (avoidance score: $r = -0.22$, $p = 0.028$; adaptivity score: $r = 0.25$, $p = 0.015$), only this contrast was correlated with brain data. No relationships between the arousal contrast and brain data during the word phase (i.e., the only phase during which RT was possible) in *a priori* regions of interest were found.

Neural activity at T1 as the mediator between trait motivation and change in psychopathology

Mediation analyses were performed with activity from regions of interest extracted from planned analyses focusing on PFC, basal ganglia, and the amygdala. The adaptivity score was the only measure used as an independent variable because it was the only one significantly associated with brain regions that was also associated with change in psychopathology. Brain data from right posterior cingulate cortex, left paracingulate cortex, and left superior frontal gyrus were used as mediator variables in three separate mediation analyses. As these regions were all strongly correlated with one another (see Table 5), they were considered together in a multiple mediation analysis.

Brain data were extracted from contrasts that highlighted processing the anticipation of punishment, which was the only contrast associated with the adaptivity score that showed a relationship with change in psychopathology. Change in anxious arousal was used as the dependent variable, as this was the only measure of change in psychopathology that showed a relationship with brain data in *a priori* regions of interest (see Table 4).

Results from analyses using posterior cingulate cortex as the mediator (see Figure 3) indicated that the indirect effect of the adaptivity score on change in anxious arousal was negative (-0.23, boot-strap 95% CI: -0.58 to -0.03). The path from the adaptivity score to posterior cingulate cortex was positive (4.12, 95% CI: 1.80 to 6.44) whereas the path from posterior cingulate cortex activity to change in anxious arousal was negative but non-significant (-0.06, 95% CI: -0.12 to 0.008). This result suggests that having more approach relative to avoidance increases activity in the posterior cingulate during trials that signal the possibility of punishment. In turn, this increased activity decreased the likelihood of worsening symptoms of anxious arousal. The direct effect of the adaptivity score on change in anxious arousal taking into account brain activity was not significant (c' = -0.26, 95% CI: -0.77 to 0.26).

When the paracingulate was used as the mediator (see figure 4), the indirect effect of the adaptivity score on change in anxious arousal was negative (-0.31, boot-strap 95% CI: -0.63 to -0.06). The path from the adaptivity score to paracingulate activity was positive (4.50, 95% CI: 2.33 to 6.66) and the path from paracingulate activity to change in anxious arousal was negative (-0.07, 95% CI: -0.14 to -0.002). Similarly to the relationship observed above, this result suggests that having more approach relative to avoidance increases activity in the posterior cingulate during trials that signal the possibility of punishment. In turn, this increased activity decreases the likelihood of worsening symptoms of anxious arousal. The direct effect of the adaptivity score on symptom change taking into account brain activity was not significant (c' = -0.17, 95% CI: -0.70 to 0.36).

Results indicate that the superior frontal gyrus did not mediate the relationship between the adaptivity score and change in anxious arousal (see figure 5, c = -0.25, boot-strap 95% CI: -0.69 to 0.03). The direct effect of the adaptivity score controlling for brain activity was also non-significant (c' = -0.23, 95% CI: -0.75 to 0.29).

A multiple mediation model with posterior cingulate cortex, paracingulate cortex, and superior frontal cortex as the mediators (see figure 6) revealed a significant negative total indirect effect of adaptivity score on change in anxious arousal (-0.42, boot-strap 95% CI: -0.84 to -0.05). All paths from the adaptivity score to brain region mediators were positive (all 95% CIs did not include zero) and all paths from brain region mediators to change in anxious arousal were negative (though all 95% CIs did include zero). This result suggests that the adaptivity score increases activity in this network of regions during the anticipation of punishment, which reduces the likelihood of worsening symptoms of anxious arousal over time. The direct effect of the adaptivity score on symptom change taking into account brain activity was not significant (c' = -0.07, 95% CI: -0.63 to 0.50).

Exploratory analyses: whole-brain gray matter mask

Whole-brain analyses were intended to reveal what other brain regions may contribute to the relationship between psychopathology and trait motivation (e.g., posterior regions previously implicated in executive function and attention that may be affected by differences in trait motivation; Banich, 2000). The adaptivity score was significantly associated with activity during all phases of the task. The adaptivity score was positively correlated with neural responses to cues signaling punishment as compared to those free of punishment signals in the right posterior cingulate cortex, bilateral frontal pole, and left paracingulate cortex. In the word phase, the adaptivity was related to the positive affect contrast such that a higher adaptivity score was associated with greater neural activity to neutral compared to positive words in the left precuneus, the left primary motor cortex, and the left primary somatosensory cortex. Within the feedback phase, the adaptivity score was related to stronger responses to feedback indicating no change than to feedback indicating reward in the three regions of primary somatosensory cortex: a left superior region, a left inferior region, and a right region; as well as in right superior frontal gyrus (see Table 6).

When approach, avoidance, and their interaction were entered as higher-level predictors, relationships emerged between the unique effects of avoidance in the cue and word phases. Avoidance was associated with increased activity to cues signaling the absence of punishment compared to cues signaling punishment in the ventromedial PFC/medial OFC and left frontal pole. Avoidance was also associated with positively valenced as compared to negatively valenced words in the right superior parietal lobule and left precentral gyrus (see Table 6).

Relationship between RT and brain data

As above, the arousal score was the only RT contrast related to approach and avoidance scores (avoidance score: $r = -0.22$, $p < 0.05$; adaptivity score: $r = 0.25$, $p < 0.05$), and only this contrast was then correlated with brain data. No relationships between the arousal contrast and brain data in exploratory regions of interest were found.

Neural activity at T1 as the mediator between trait motivation and change in psychopathology

As above, mediation analyses were performed with activity from regions of interest extracted from exploratory whole-brain analyses. Again, the adaptivity score was the only measure used as an independent variable because it was the only one significantly associated with brain regions also associated with change in psychopathology. Brain data from right primary somatosensory cortex, left primary somatosensory cortex, and right superior frontal gyrus were used as mediator variables in four separate mediation analyses. The right primary somatosensory cortex showed a relationship with anxious arousal, and all of these regions showed a relationship with depressive low positive affect (see Table 6). As the regions associated with depressive low positive affect were all strongly associated with one another (see Table 7), they were considered together in a multiple mediation analysis. Brain data were extracted from contrasts that highlighted processing the neutral feedback compared to rewarding feedback. The neutral condition includes the experience of frustrated non-reward as well as relief from not being punished. This was the only contrast associated with the adaptivity score that showed a relationship with change in psychopathology in the exploratory analyses that had not already been submitted to mediation analysis.

Results from analyses using right primary somatosensory cortex as the mediator (see Figure 7) indicates that the indirect effect of the adaptivity score on change in anxious arousal was negative (-0.27, boot-strap 95% CI: -0.72 to -0.02). The path from the adaptivity score to right primary somatosensory cortex was positive (10.76, 95% CI: 4.88 to 12.40) whereas the path from right primary somatosensory cortex activity to change in anxious arousal was negative (-

0.03, 95% CI: -0.05 to -0.001). These results suggests that having more approach relative to avoidance increases activity in the right primary somatosensory cortex during the processing of neutral feedback compared to rewarding feedback. In turn, this increased activity decreases the likelihood of worsening symptoms of anxious arousal. The direct effect of the adaptivity score on change in anxious arousal taking into account brain activity was not significant (c' = -0.20, 95% CI: -0.71 to 0.32).

When the right primary somatosensory cortex was used as the mediator (see figure 8) and change in depressive low positive affect as the dependent variable, the indirect effect of the adaptivity score on symptom change was negative (-0.41, boot-strap 95% CI: -0.99 to -0.01). The path from adaptivity score to primary somatosensory cortex activity was positive (10.76, 95% CI: 4.88 to 12.40) and the path from primary somatosensory cortex activity to change in depressive low positive affect was negative (-0.04, 95% CI: -0.07 to -0.003). This result suggests that having more approach relative to avoidance increases activity in the primary somatosensory cortex during the processing of neutral feedback compared to rewarding feedback. In turn, this increased activity decreases the likelihood of worsening symptoms of depressive low positive affect. The direct effect of adaptivity score on depressive low positive affect taking into account brain activity was significant (c' = -1.79, 95% CI: -2.76 to -0.82). Thus, the adaptivity score influences symptom change in depressive low positive affect both via its effects on brain activity and through other pathways not captured in right primary somatosensory cortex activity.

Results from analyses using left primary somatosensory cortex as the mediator (see figure 9) indicated that the indirect effect of the adaptivity score on change in depressive low positive affect was negative (-0.29, boot-strap 95% CI: -0.79 to -0.05). The path from the adaptivity score to left primary somatosensory cortex was positive (9.41, 95% CI: 2.72 to 16.11) whereas the path from left primary somatosensory cortex activity to change in depressive low positive affect was negative (-0.03, 95% CI: -0.06 to -0.0003). This result suggests that having more approach relative to avoidance motivation increases activity in the left primary somatosensory cortex during the processing of neutral feedback compared to rewarding feedback. In turn, this increased activity decreases the likelihood of worsening symptoms of depressive low positive affect. The direct effect of the adaptivity score on change in depressive low positive affect taking into account brain activity was significant (c' = -1.85, 95% CI: -2.81 to -0.89). This result suggests the adaptivity score affects symptom change indirectly via left primary somatosensory cortex activity and directly through other pathways not identified in this analysis.

Using superior frontal gyrus as the mediator (see figure 10) reveals a non-significant negative indirect effect of the adaptivity score on change in depressive low positive affect = -0.22, Boot 95% CI: -0.71 to 0.21). The direct effect of the adaptivity score on change in depressive low positive affect taking into account brain activity was significant (c' = -1.77, 95% CI: -2.86 to -0.69). This result suggests that the adaptivity score leads to lower levels of depressive low positive affect independently of superior frontal gyrus activity.

A multiple mediation model with right primary somatosensory cortex, left primary somatosensory cortex, and superior frontal gyrus as mediators (see figure 11) revealed a non-significant negative total indirect effect of the adaptivity score on change in depressive low positive affect (-0.42, boot-strap 95% CI: -1.04 to 0.09). The direct effect of the adaptivity score on symptom change taking into account brain activity was significant (c' = -1.80, 95% CI: -2.90 to -0.74), suggesting this group of brain regions considered together did not account for the effect of the adaptivity score on change in depressive low positive affect.

DISCUSSION

Relationship between trait motivation and change in psychopathology

Extending previous work and in line with nearly all proposed hypotheses, present results show positive relationships between worsening depressive low positive affect, depressive loss of interest, anxious arousal, and trait avoidance motivation; and negative relationships between worsening depressive loss of interest and trait approach motivation (Clark & Watson, 1991; Elliot & Thrash, 2002; Spielberg et al., 2011a). Counter to hypotheses, no relationship was observed between changes in anxious apprehension and avoidance motivation. However, this measure of anxiety showed strong stability from T1 to T2, which is in line with the theory that describes this dimension of anxiety as trait-like and less likely to vary over time (Sharp, Miller, & Heller, 2015).

Present results extend previously observed cross-sectional relationships observed between trait motivation and depressive low positive affect as measured by the MASQ (Nitschke et al., 2001; Spielberg et al., 2011a; Watson et al., 1995) by showing that trait avoidance motivation predicts increases, and trait approach motivation predicts decreases, in this dimension of depression, even after controlling for the relationship between motivation and T1 depression. Unlike previous research using cross-sectional data (Spielberg et al., 2011a), present results show that trait avoidance motivation had a stronger relationship ($\beta = 0.48$) to changes in depressive low positive affect than to trait approach motivation ($\beta = -0.29$). These results suggest that the unique effects of avoidance motivation are more potent risk-factors for worsening symptoms of this dimension of depression than approach motivation. However, it is clear that approach motivation still plays a role in the worsening course of depressive low positive affect by providing a countervailing influence (Clark & Watson, 1991). Similarly, the adaptivity score demonstrated a strong negative relationship with changes in depressive low positive affect (β = -0.66). The strength of this relationship suggests that individuals at the greatest risk for worsening symptoms of this dimension of depression are those with a predominance of avoidance motivation and low approach motivation.

Change in depressive loss of interest showed a strong relationship with trait avoidance motivation (β = 0.53) and no relationship with trait approach motivation (β = 0.06). This is consistent with past cross-sectional research (Clark et al., 1994; Nitschke et al., 2001; Spielberg et al., 2011a). The relationship between avoidance motivation and the MASQ depressive loss of interest scale (AD8) may be explained by the relationship between this scale and the general distress factor that was identified in a factor analysis performed on the items of the MASQ (Watson et al., 1995). This general distress factor is conceptually similar to the broad negative affect factor described in the tripartite model of depression and anxiety (Clark & Watson, 1991), and to the higher-order neuroticism factor identified by Zingbarg and colleagues (2016). The present results highlight the role of trait avoidance motivation as a risk-factor for the exacerbation of depressive loss of interest. Indeed, previous research has hypothesized that low approach motivation may be a risk-factor for the first onset of depression (Shankman et al., 2007) whereas avoidance motivation acts as a maintenance factor (Spielberg et al., 2011a). This study provides some support to the latter argument since avoidance motivation was strongly predictive of worsening symptoms of both depressive low positive affect and depressive loss of interest.

The adaptivity score showed a slightly weaker relationship with depressive loss of interest (β = -0.40) than depressive low positive affect, which is expected given the lower impact of approach motivation on this dimension of depression. However, as above, individuals with a preponderance of avoidance motivation and low approach motivation were at the greatest risk for worsening symptoms of depressive loss of interest.

As predicted, changes in anxious arousal were related to trait avoidance motivation (β = 0.33) and not approach motivation (β = -0.02). These results were in line with earlier work and theories that connected anxiety with avoidance motivation (Davidson, 2002; Spielberg et al., 2011a). Some hypotheses based on the tripartite model were not supported. Specifically, this model posits that anxious arousal is more closely related to somatic hyperarousal and not as closely related to negative affect and avoidance motivation (Clark & Watson, 1991). However, the hypothesis that this dimension of anxiety would not be related to approach motivation was supported (Clark & Watson, 1991). The primary contribution of avoidance motivation to changes in anxious apprehension was also reflected in the relatively weak relationship between this dimension of anxiety and the adaptivity score (β = -0.26). Several theories support the role of avoidance behaviors in the maintenance and exacerbation of anxiety (e.g., Leventhal, 2008), and this project offers empirical support for this relationship.

The results discussed here contribute to the literature on the relationship between trait motivation and depression and anxiety by extending the current understanding of these clinical constructs beyond cross-sectional relationships. However, these results do not clarify how trait approach and avoidance motivation confer risk for a worsening course of depression and anxiety. Specifically, behavioral effects alone could not, by definition, identify neural mechanisms that mediate the relationship between motivation and psychopathology. To this end, the present fMRI paradigm was designed to identify brain regions that varied as a function of both motivation and of the experience of the anticipation/experience of rewards/punishments and emotional processing.

fMRI and Mediation analyses

Patterns of brain activity that covaried with trait motivation were examined in a modified monetary incentive delay task. The fMRI task contained three phases examining the neurocognitive events associated with 1) the cue phase that probed the anticipation of future outcomes, 2) the word phase that probed the processing of positive, negative and neutral material, and 3) the feedback phase that probed the experience of monetary outcomes based on performance.

Patterns of neural activity associated with approach and avoidance motivation were not in line with hypotheses formulated on the basis of existing theories of motivational traits, but were in line with the alternative hypotheses based on treatment literature (e.g., Badour et al., 2012). Early biobehavioral theories of trait motivation based on observations of animal models and human behavior posited that approach motivation would lead to greater attention to positive aspects of the environment and avoidance motivation would lead to greater attention to negative aspects of the environment (Elliot, 2000). Present results suggest that the opposite was true in our sample and that the behavioral patterns associated with avoidance observed in studies related to the etiology, maintenance, and treatment of psychopathology explained the present data better (Badour et al., 2012; Goldin & Gross, 2010; and see Chawla & Ostafin, 2007 for a review).

Generally, higher approach motivation was associated with neural reactivity to more negative stimuli (e.g., neutral relative to positive), whereas avoidance was associated with stronger neural reactivity to more positive stimuli (e.g., neutral relative to negative). This would suggest that those with high approach motivation engage with emotionally negative material in the environment and those with high avoidance motivation are more likely to engage with positive material.

Cue phase of task (anticipation phase)

Consideration of *a priori* regions[3] of interest involved in the cue phase of the task suggested that only avoidance and the adaptivity score affected processing of the anticipation of punishment. Specifically, avoidance motivation was associated with greater reactivity to neutral cues as compared to punishing cues within the vmPFC/OFC and the left frontal pole. The vmPFC has been strongly associated with emotional processes (e.g., reward, fear; de la Vega et al., 2016) and the OFC has been associated with subjective valuation that informs decision-making (Montague & Berns, 2002; Padoa-Schioppa & Assad, 2008; for a review see Stalnaker, Cooch, & Schoenbaum, 2015). It is possible that as avoidance motivation increases, the processing of these emotion- and value-related regions is biased away from the anticipation of negative information (i.e., cues that signal punishment) in favor of comparatively less negative information (i.e., cues that signal no loss of money). However, this biasing was not observed to mediate any part of the relationship between trait motivation and changes in psychopathology.

The avoidance-related frontal pole region of interest (ROI) identified in the present study overlaps with the dorsal frontal pole cluster identified in a large-scale parcellation of this region using diffusion tensor imaging (DTI) tractography and cluster analysis (Orr, Smolker, & Banich, 2015). Using DTI to observe anatomical connections and patterns of co-activation to identify functional connections, it was observed that this dorsal region of the frontal pole was part of a

[3] Nearly identical peak coordinates were identified in exploratory analyses characterizing the impact of avoidance motivation in the cue phase. ROIs differed only in extent, with the smaller mask isolating *a priori* regions yielding larger clusters than the whole-brain mask. Given this similarity in peak coordinates, only ROIs from *a priori* analyses will be discussed for this phase of the task.

network of regions involved in top-down goals, such as cognitive control and maintaining action plans (Orr, Smolker, & Banich, 2015). In the present project, positive associations between left frontal pole activity and avoidance motivation during the processing of neutral cues over punishing cues may indicate that focusing on comparatively less negative information may be in line with top-down goals. Once again, the activity in this brain region was not observed to mediate changes between trait motivation and changes in psychopathology.

The adaptivity score was positively associated with greater neural reactivity to punishment cues as compared to neutral cues. The peak voxels for the vmPFC/OFC and the left frontal pole sensitive to the adaptivity score were nearly identical to the peak voxel of these regions sensitive to avoidance motivation, although the extent of each ROI was different. The adaptivity score necessarily involves both approach and avoidance processes, and is negatively related to avoidance motivation. Therefore, the processes captured by the relationship between the adaptivity score and the punishment > neutral cue contrast in these two ROIs are likely the same as those captured by the relationship between avoidance and the neutral > punishment cue contrast. In addition to these two regions, neural activity sensitive to the adaptivity score during this contrast was also observed in the right frontal pole, the left paracingulate, the left superior frontal gyrus, and the right posterior cingulate cortex[4]. Activity in these regions reflects the balance between approach and avoidance motivation and is likely associated with the adaptive nature of this score.

Similar patterns of anatomical connectivity and functional co-activation have been observed for left and right frontal pole (Orr, Smolker, & Banich, 2015). Therefore, the positive

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[4] Each of these regions was observed in *a priori* and exploratory analyses. Peak voxels for each region were nearly identical between the two analyses and ROIs only differed in extent. However, more ROIs were revealed using the smaller *a priori* mask. Therefore, only those regions in *a priori* analyses will be discussed.

associations between right frontal pole activity and the adaptivity score during the processing of punishing cues over neutral cues likely indicates that focusing on comparatively more negative information may be in line with top-down goals. As with the left frontal pole, the activity in right frontal pole was not observed to mediate the relationship between trait motivation and changes in psychopathology.

The paracingulate ROI positively related to the adaptivity score identified in the present study corresponded to a region of midcingulate cortex (Vogt, 2016) that was functionally related to a network of brain regions involved in cognitive control and in the processing of negative affect (de la Vega et al., 2016). This activity is line with the adaptive control hypothesis that posits this region to be involved in integrating emotional signals with top-down cognitive control goals to facilitate adaptive behavior in the context of uncertainty (Shackman et al., 2011; Cavanagh & Shackman, 2015). In the context of the present study, positive correlations between this region and adaptivity scores during the anticipation of punishment relative to neutral outcomes may reflect a willingness to engage with the potential for negative outcomes in those with a higher adaptivity score. Indeed, processing within this region also mediated the relationship between the adaptivity score and changes in anxious arousal. Specifically, as adaptivity scores increased, activity within this region also increased, which in turn led to decreases in anxious arousal over time.

Adaptivity scores also correlated with activity in the left superior frontal gyrus, which showed spatial overlap with a region labeled as the dorsal medial PFC (dmPFC) defined using a meta-analytic database of nearly 10,000 studies and multivariate classification (de la Vega, 2016). This classification was based on patterns of co-activation across a wide range of designs and subsequent identification of psychological constructs most strongly associated with each identified region using the Neurosynth database (Yarkoni et al., 2011). The dmPFC was shown to have relationships with a network of brain regions involved in processes including self-referential thought and theory of mind (Baumgartner et al., 2012; Denny et al., 2012; Mitchell et al., 2005), and work from our lab has linked this region with inhibition (e.g., Spielberg et al., 2011b). These associations suggest the left superior frontal gyrus plays a role in the consideration of consequences. As this region was active during the anticipation of punishment and correlated with an adaptive balance between approach and avoidance in the present study, it is possible the superior frontal gyrus is part of the process of engaging with negative information and inhibiting the desire to avoid it. However, activity within the superior frontal gyrus did not mediate the relationship between the adaptivity score and change in psychopathology, and these claims need to be evaluated further in future research.

The right posterior cingulate cortex was the last ROI observed to correlate with adaptivity scores during the anticipation of punishment. This ventral region of the posterior cingulate has been characterized as a central hub of the default mode network (DMN; Greicius et al., 2009; Margulies et al., 2009; Leech et al., 2011, and see Leech & Sharp, 2014 for a review). Although the DMN is often referred to as a task-negative network (Fox et al., 2005), shifts between deactivation and activation are noted within this network in the context of tasks when individuals direct attention inward, such as during episodic memory retrieval, daydreaming, and planning for the future (Burdwood et al., 2016; Spreng, 2012). The upregulation of this node of the DMN observed in the present study during the anticipation of punishment may reflect participants engaging in planning for the possibility of loss or recalling past experiences of loss. Present mediation analyses provided evidence for this upregulation being protective. Posterior cingulate cortex activity accounted for the relationship between the adaptivity score and changes in anxious arousal such that increased adaptivity scores predicted greater posterior cingulate activity that, in turn, reduced the likelihood of worsening symptoms of anxious arousal (and see Burdwood et al., 2016 for similar associations between DMN and anxious arousal). This process may be protective in that it could represent participants engaging with negative material in ways that are adaptive as opposed to avoiding it, which has been implicated in a number of psychiatric conditions (e.g., social anxiety, post-traumatic stress disorder [PTSD]; Badour et al., 2012; Goldin & Gross, 2010; and see Chawla & Ostafin, 2007 for a review).

Despite the superior frontal gyrus not acting as a mediator between motivation and changes in psychopathology, activity in all three regions (i.e., paracingulate gyrus, superior frontal gyrus, and posterior cingulate gyrus) was correlated and they each showed relationships with psychopathology (see Table 4). Multiple mediation analyses using the paracingulate gyrus, superior frontal gyrus, and posterior cingulate gyrus support the role for all three of these brain regions working in concert to protect against worsening symptoms of anxious arousal. The activity in these brain regions correlated during the anticipation of punishment; however, whether these ROIs are indeed part of a network is unclear. Multivariate work identifying functional networks involving posterior cingulate cortex showed connections between this region and superior frontal gyrus, but not paracingulate gyrus (Leech, Braga, & Sharp, 2012). It may be that these regions are each involved in the adaptive functioning described above, and are doing so as part of different networks (e.g., DMN, fronto-parietal attention networks). Together, the present findings from the cue phase highlight the role of trait motivation in affecting anticipatory brain activity, and begin to elucidate the complexity of coordinated brain activity that can lead to either adaptive or maladaptive coping styles with implications for the course of psychopathology (i.e., anxious arousal).

Word phase of task (emotional phase)

The unique effects of avoidance on the processing of emotional valence recruited left primary motor cortex and two regions in left precentral gyrus in *a priori* analyses, and a region of right superior parietal lobule in exploratory analyses[5]. In keeping with what has been stated above, these relationships were counter to our hypotheses built on existing theories in that all avoidance-related regions were more reactive to positively than to negatively valenced words.

Left primary motor cortex and left precentral gyrus activity likely reflects the action of the right hand during performance of the reaction time task (e.g., Porro et al., 1996). The relationship of activity in these regions with avoidance motivation during the processing of positive words over negative words may indicate that individuals high on avoidance motivation engage motor regions to a greater degree in the presence of positive than negative emotions. However, neither RT data nor changes in psychopathology were related to the activity of this region. Therefore, this interpretation is only speculative.

A parcellation of the superior parietal lobule based on multimodal imaging data (i.e., DTI, co-activation patterns, and resting-state data) showed that this region can be separated into anatomically and functionally distinct sub-regions (Wang et al., 2016). The right superior parietal lobule active during emotionally valenced words and correlated with avoidance motivation in the present study overlapped with a region associated with processes including vision, motion, and working memory (Wang et al., 2016, see also Wolpert, Goodbody, & Husain, 1998). It is possible that avoidance-related activity in this region corresponds to prioritizing the integration of the various sensory features of positively valenced words over

[5] The region of left precentral gyrus identified in exploratory analyses had a nearly identical peak voxel as the one identified in *a priori* analyses. Only the precentral ROI identified in *a priori* analyses will be discussed.

negatively valenced words as well as the potential outcomes of performance (e.g., reward vs. punishment). Once again, no relationships were found between this region and RT or change in psychopathology. Therefore, the consequences of this neural activity are still unclear.

The adaptivity score was associated with similar motor-related regions as avoidance motivation during the valence contrast (i.e., right supplementary motor area). As above, this region lacked relationships with RT and psychopathology. This score was also related to the processing of positive affect (positive words contrasted against neutral words) in left primary motor cortex[6] in *a priori* analyses and in left primary somatosensory cortex and left precuneus in exploratory analyses. All relationships were in the direction of increased processing of neutral words relative to positive words, suggesting relatively greater importance of comparatively less positive information for individuals with a predominance of approach motivation.

Primary somatosensory cortex activity has been implicated in word reading (Peeva et al., 2010) as well as "feeling" emotions as body-states (i.e., the somatic marker hypothesis, Bechara & Damasio, 2005; Damasio, Tranel, & Damasio, 1991; Poppa & Bechara, 2018). The somatic marker hypothesis states that the physical sensations and processes that contribute to the experience of emotions (e.g., endocrine release, changes in heart rate, etc.) are processed in networks of the brain associated with physical sensation (e.g., insula, somatosensory cortex), and that this information can affect higher order cognition and decision-making (Damasio, Tranel, & Damasio, 1991; see Poppa & Bechara, 2018 for an updated review of this hypothesis). The observed somatosensory activity may reflect greater verbal and emotional engagement with the neutral words themselves as a function of higher approach relative to avoidance motivation. The region of precuneus observed in the present study has been associated with mental imagery

[6] A left primary motor cortex ROI emerged in both *a priori* and exploratory analyses. These ROIs were nearly identical in their peak coordinates, but differed in extent. Only the ROI from *a priori* analyses will be discussed.

associated with movement (Zhang & Li, 2012), and may be related to increased preparations to respond to neutral relative to positive stimuli in individuals with more approach than avoidance motivation. However, as no relationships were observed between activity in either of these ROIs and RT or change in psychopathology, more research is needed to understand the role of trait motivation in these regions of the brain.

Feedback phase of task (consummatory phase)

Adaptivity scores were positively associated with the experience of receiving neutral feedback (i.e., neither a loss nor gain of money) as compared to rewarding feedback in two ROIs in left somatosensory cortex (one superior and one inferior), one ROI in right primary somatosensory cortex, and one ROI in right superior frontal gyrus in exploratory analyses[7]. This contrast can be generally interpreted as receiving a disappointing result, and also includes components of frustrated non-reward (a relatively negative outcome) compared to a positive result.

The involvement of the somatosensory cortex in the receipt of this form of feedback once again aligns with the somatic marker hypothesis (e.g., Damasio, Tranel, & Damasio, 1991). Specifically, it may be that individuals with higher approach than avoidance motivation "feel" the receipt of this comparatively less positive (and on certain trials, negative) outcome more than individuals with the opposite trait motivational pattern. An example of how this may be protective using a situation in daily life would be receiving news that you have not been considered for a promotion. The ability to engage with, instead of disengage from, this information would be adaptive for future behavior (e.g., asking for feedback on interview style).

[7] No ROIs were observed to feedback in *a priori* analyses.

Indeed, engaging with less positive stimuli appears to be protective as activity in left superior and right somatosensory cortices separately account for much of the variance in the relationship between the adaptivity score and changes in depressive low positive affect. Furthermore, the right somatosensory cortex also accounted for a significant amount of variance in the relationship between the adaptivity score and changes in anxious arousal.

As reviewed above, the left superior frontal gyrus seems to play a role in the consideration of consequences. Given that this region was active during the receipt of less positive (and at times negative) feedback and correlated with an adaptive balance between approach and avoidance, it is likely the superior frontal gyrus is involved in the process of engaging with less desirable information. However, as this ROI was not found to mediate the relationship between adaptivity scores and changes in psychopathology, it seems adaptivity scores lead to lower levels of depressive low positive affect independently of superior frontal gyrus activity.

Despite the fact that the superior frontal gyrus did not mediate the relationship between trait motivation and change in psychopathology, activity from the three brain regions used in mediation analyses was correlated and they were all related to change in depressive low positive affect (see Table 6). Multiple mediation analyses revealed that the direct effect of adaptivity scores on changes in depressive low positive affect was still significant after considering all these regions simultaneously. This suggests that the indirect effects of the left superior and right somatosensory cortices account for a significant amount of the variance in the protective relationship between adaptivity scores and depressive low positive affect. However, it does not appear that these regions act in concert with the superior frontal gyrus.

Conclusions: Integration of behavioral and neural evidence

The purpose of the present study was to further understanding of the relationship between trait approach and avoidance motivation and dimensions of anxiety and depression (Elliot & Thrash, 2002; Spielberg et al., 2011a, b, 2012), and to begin to identify potential neural mechanisms that explain relationships between these clinical constructs. Self-report and imaging data were used to characterize these relationships in order to arrive at a complex picture of the dynamics between neural activity and clinical phenomenology.

Predicted relationships were observed between self-report measures of approach and avoidance motivation and changes in dimensions of depression and anxiety in support of existing theories of these pathological states[8] (e.g., Clark & Watson, 1991; Spielberg et al., 2011a). However, neural predictions did not fall in line with hypotheses based on existing theories of how approach and avoidance affect neural processing (e.g., Davidson et al., 2000; Spielberg et al., 2011b, 2012). Instead, across the three phases of the fMRI task, trait avoidance motivation was associated with reactivity to stimuli that were comparatively less negative and/or positive, and higher approach relative to avoidance motivation was associated with the reverse. Although this pattern was not expected, mediation analyses revealed that it fits with theories about the development and maintenance of psychopathology in the treatment literature. Reducing avoidance behavior and increasing approach behavior is the central task in many therapies for anxiety- and depression-related conditions (Ainsworth et al., 1978; Barlow, Allen, & Choate, 2004; Barlow, Chorpita, & Turovsky, 1996, Craske & Barlow, 2000; Leventhal, 2008). Therefore, and as suggested by present results, successful treatment involves engaging with

[8] Hypotheses were not confirmed for anxious apprehension, which did not show enough variability from T1 to T2 for change in this dimension of anxiety to be explained by trait motivation.

potentially aversive information so that one can process it and move on as opposed to avoiding it.

The present results also illustrate that adaptive, trait-motivation-related stimulus processing is not a straightforward valence-bound process. This complexity is made clear by the multiple, functionally diverse regions that mediated the relationship between the adaptive balance between approach and avoidance motivation and dimensions of depression and anxiety. For example, adaptivity score-related regions that mediated changes in anxious arousal were associated with self-referential thought, inhibition, and the integration of emotional material with goals in the context of uncertainty (e.g., Burdwood et al., 2016). Each of these regions was active during the anticipation of potentially negative outcomes, and it stands to reason that being able to engage these functions adaptively would be protective against worsening anxiety (e.g., Leventhal, 2008). The brain regions that mediated the relationship between the adaptivity score and depressive low positive affect also constituted a complex neural system involved in processing the "feeling" associated with the receipt of less-positive feedback (e.g., Poppa & Bechara, 2018). Disengagement from such relatively negative experiences (e.g., emotions associated with disappointment) has been observed to impact the ability to cope with such experiences and to contribute to the maintenance of depressive symptoms (Craske & Barlow, 2000; Leventhal, 2008). In addition, it was the predomination of approach relative to avoidance that resulted in the richest set of brain-motivation associations underscoring the utility of this measure to capture adaptive functioning and its relationship to brain function (Elliot & Thrash, 2002; Spielberg et al., 2011a).

As discussed above, cue-related activity mediated changes in anxiety and feedback-related activity mediated changes in depression. These patterns highlight two important observations: anticipatory cognitions seem to have the biggest impact on anxiety, and the emotional engagement with present experience of feedback seems to have the biggest impact on depression. These observations fall in line with theories on the future-oriented nature of anxiety (e.g., Andrews & Borkovec, 1988) and on deficits in feedback processing observed in depression (e.g., Davidson et al., 1990, 1999; Henriques & Davidson, 2000).

The present study thus broadens the scope of processes affected by trait motivation to include anticipatory and consummatory experiences. As mentioned above, current results align with theories linking anxiety with expectations about the future and depression with feedback processing. These data represent a promising avenue of future research into how motivation affects psychopathology, considered in the context of its temporal antecedents, i.e., future-oriented worry of anxiety and present-oriented feedback processing deficits of depression (Andrews & Borkovec, 1988; Davidson et al., 1990, 1999; Henriques & Davidson, 2000; Sharp, Miller, & Heller, 2015).

The results of the present study need to be considered in the context of its limitations. As this was a multi-time point design, it was possible to observe changes in depression and anxiety. However, more than two time points would have made it possible to observe more complex dynamics between psychopathology and trait motivation. Furthermore, trait motivation and brain activity were only measured at T1, which did not make it possible to test the stability of these individual measures and relationships among them. Also, although the current sample size was large for an fMRI study (N = 85), the follow-up sample was much smaller (N = 43) and may have lowered the ability to detect certain complex effects (e.g., interactions). The fMRI task included an emotional phase (i.e., the word phase); however, the salience of emotion during this phase was limited given the nature of the speeded reaction time task. Therefore, the amount of engagement of emotion-processing regions may have been lower than for other tasks that place more emphasis on emotional experience (e.g., emotion-word Stroop, Engels et al., 2010; Herrington et al., 2010).

Nonetheless, the present study had several methodological strengths that made it well-suited to explore how motivation affects psychopathology. For instance, the present study employed an established latent factor estimation procedure (Elliot & Thrash, 2002, 2010; Spielberg et al., 2011a, 2011b, 2012) that accounted for errors in measurement and aggregated a number of different self-report measures of trait approach and avoidance motivation. This approach to calculating trait motivation better reflects the complex cognitive processes that are captured by this construct. Furthermore, neural data used in mediation analyses were calculated based on the larger sample, which provides greater stability of error terms and allows for greater confidence in observed findings. The results of this study contribute new insights to the literature on the relationship between trait motivation and psychopathology and suggest that engaging with both the anticipation and receipt of negative information is adaptive as it relates to depression and anxiety. One further possible conclusion from these findings in terms of future therapeutic interventions is the need to help patients fully engage the wide range of cognitive processes found to be associated with the adaptive balance between approach and avoidance motivation, and to do so at multiple stages of experience (e.g., anticipation, consummation). In doing so, treatment of depression and anxiety becomes less about decreasing avoidance and increasing approach, and more about learning a variety of cognitive skills focused on different aspects of coping with challenging experiences.

TABLES

	BIS	BAS	GTS-NT	GTS-PT	NEO-N	NEO-E
Approach	0.25	0.62	0.13	0.83	--	0.84
Avoidance	0.83	0.18	0.91	--	0.89	--

Table 1. Factor weights from Spielberg et al., 2011a that were used to define present trait approach and avoidance latent factor variables. BIS = BIS-BAS Behavioral Inhibition System, BAS = BIS-BAS Behavioral Activation System, GTS-NT = General Temperament Survey Negative Temperament, GTS-PT = General Temperament Survey Positive Temperament, NEO-N = NEO-Five Factor Inventory Neuroticism, NEO-E = NEO-Five Factor Inventory Extraversion.

Descriptive Statistics **Correlations**

	Mean	Std. Dev.	BIS	BAS	GTS-NT	GTS-PT	NEO-N	NEO-E	AP	AV	Adapt.	T1 Worry	T1 AA	T1 LI	T1 Low PA	T2 Worry	T2 AA	T2 LI	T2 Low PA
BIS	20.97	4.51	—																
BAS	40.94	5.60	-0.16	—															
GTS-NT	11.42	8.15	.713**	-.176*	—														
GTS-PT	17.62	6.54	-.423**	.551**	-.511**	—													
NEO-N	31.46	10.80	.735**	-.277**	.873**	-.575**	—												
NEO-E	42.18	8.87	-.321**	.535**	-.512**	.813**	-.575**	—											
AP	-0.02	1.88	-.203*	.775**	-.344**	.892**	-.445**	.901**	—										
AV	0.01	2.40	.880**	-0.15	.941**	-.513**	.938**	-.480**	-.308**	—									
Adapt.	-0.02	3.47	-.717**	.522**	-.835**	.837**	-.889**	.819**	.754**	-.857**	—								
T1 Worry	47.59	14.71	.782**	-.202*	.823**	-.387**	.841**	-.389**	-.250**	.879**	-.745**	—							
T1 AA	24.84	6.68	.329**	-.213*	.467**	-.209*	.458**	-.191*	-0.17	.444**	-.399**	.294**	—						
T1 LI	16.28	5.05	.435**	-.319**	.660**	-.649**	.692**	-.588**	-.545**	.635**	-.734**	.520**	.506**	—					
T1 Low PA	36.78	11.77	.442**	-.404**	.584**	-.670**	.669**	-.702**	-.638**	.595**	-.756**	.530**	.228*	.725**	—				
T2 Worry	44.19	14.09	.671**	-.290*	.664**	-.384**	.689**	-0.22	-0.24	.741**	-.615**	.800**	0.18	.440**	.360*	—			
T2 AA	22.60	5.38	.322*	-0.01	.392**	-.338*	.459**	-0.07	-0.13	.439**	-.358*	.315*	.562**	.399**	0.24	.417**	—		
T2 LI	14.77	4.57	.403**	-0.01	.504**	-.337*	.540**	-0.21	-0.18	.543**	-.452**	.417**	.546**	.429**	.323*	.374**	.702**	—	
T2 Low PA	38.45	9.42	.449**	-0.26	.624**	-.569**	.684**	-.625**	-.554**	.642**	-.713**	.583**	0.09	.621**	.640**	.387**	.354*	.545**	—

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Table 2. Descriptive statistics and correlations among all self-report variables used in time 1 (T1) and time 2 (T2). BIS = BIS-BAS Behavioral Inhibition System, BAS = BIS-BAS Behavioral Activation System, GTS-NT = General Temperament Survey Negative Temperament, GTS-PT = General Temperament Survey Positive Temperament, NEO-N = NEO-Five Factor Inventory Neuroticism, NEO-E = NEO-Five Factor Inventory Extraversion, AP = Trait approach motivation (latent factor), AV = Trait avoidance motivaiton (latent factor), Adapt. = Adaptivity score (Approach minus Avoidance), Worry = Anxious apprehension, AA = Anxious arousal, LI = Depressive loss of interest, Low PA = Depressive low positive affect.

	Positive Words	Neutral words	Negative words
Average arousal	6.59	3.73	6.56
Average valence	7.80	5.23	2.49
Average frequency	51.50	51.81	51.98
Average word length	5.78	5.33	5.38

Table 3: Arousal and valence data from the ANEW set are measured on a scale ranging from 1 to 9, with 9 corresponding to the most arousing and pleasant ratings, respectively. Frequency information was obtained from Toglia and Battig (1978).

a priori Regions of Interest		Brain Region	MNI Coordinates of Peak Voxel (*mm*)			Volume (mm^3)	Relationship with Change in Psychopathology (β)		
Unique Effect of Avoidance			X	Y	Z		*AA*	*Loss of Interest*	*Low PA*
Cue Phase of Task									
	Neutral > Punishment Cues	L. Frontal Pole (BA10)	-19	57	33	320	0.15	-0.12	0.08
		R. vmPFC/mOFC (BA11)	1	43	-17	1,784	0.19	0.06	0.05
Word Phase of Task									
	Positive > Negative Words	L. Primary Motor Cortex (BA4)	-25	-23	71	2,448	-0.05	-0.22	0.06
		L. Precentral Gyrus (BA6)	-7	-19	63	2,248	-0.01	-0.21	-0.01
		L. Precentral Gyrus (BA6)	-31	-9	57	704	-0.07	-0.21	0.03
Feedback Phase of Task									
	No significant effects								
Unique Effect of Approach									
No Significant Results									
Interaction of Approach and Avoidance									
No Significant Results									
Adaptivity Score									
Cue Phase of Task									
	Punishment > Neutral Cues	L. Paracingulate (BA8)	-1	31	39	192	**-0.37****	-0.27	-0.18
		L. Superior Frontal Gyrus (BA9)	-3	45	35	152	**-0.34****	-0.07	-0.10
		L. Frontal Pole (BA9)	-15	57	35	1,256	-0.13	0.22	-0.04
		R. Frontal Pole (BA9)	5	61	33	560	-0.23	0.15	-0.00
		R. Posterior Cingulate Cortex (BA30)	3	-43	15	392	**-0.34***	-0.25	-0.20
		R. vmPFC/mOFC (BA11)	1	43	-17	24	-0.25	-0.11	-0.16
Word Phase of Task									
	Negative > Positive Words	R. Supplementary Motor Area (BA6)	1	-9	55	1,144	0.04	0.29	0.00
	Neutral > Positive Words	L. Primary Motor Cortex (BA4)	-39	-17	41	16	0.04	0.04	-0.10
Feedback Phase of Task									
	No significant effects								

** = $p > 0.05$ ** = $p > 0.01$*

Table 4. Significant brain regions revealed in *a priori* analyses are organized by dimension of motivation and contrast. Relationship with change psychopathology was calculated using regression. Extracted brain data and T1 psychopathology were used as IVs and T2 psychopathology was used as the DV. AA = Anxious arousal, Low PA = Depressive low positive affect, L. = Left, R. = Right, BA = Brodmann's Area, vmPFC = ventromedial prefrontal cortex, mOFC = medial orbital frontal cortex.

	PCC	Paracingulate	SFG
PCC	—		
Paracingulate	0.518**	—	
SFG	0.450**	0.609**	—

Table 5. Correlation among brain regions sensitive to the anticipation of punishment and the adaptivity score. PCC = Posterior cingulate cortex, SFG = Superior frontal gyrus. N = 85, ** = p < 0.001.

Exploratory Analyses		Brain Region	MNI Coordinates of Peak Voxel (*mm*)			Volume (*mm*^{3})	Relationship with Change in Psychopathology (β)		
Unique Effect of Avoidance			X	Y	Z		*AA*	*Loss of interest*	*Low PA*
Cue Phase of Task									
	Neutral > Punishment Cues	L. Frontal Pole (BA10)	-17	57	33	56	0.18	-0.06	0.14
		R. vmPFC/mOFC (BA11)	1	43	-17	448	0.19	0.09	0.06
Word Phase of Task									
	Positive > Negative Words	R. Superior Parietal Lobule (BA7)	19	-45	67	408	0.09	-0.04	0.03
		L. Precentral Gyrus (BA6)	-7	-17	63	1,168	0.01	-0.20	-0.01
Feedback Phase of Task									
	No significant effects								
Unique Effect of Approach									
No Significant Results									
Interaction of Approach and Avoidance									
Cue Phase of Task									
	No significant effects								
Word Phase of Task									
	No significant effects								
Feedback Phase of Task									
	No significant effects								
Adaptivity Score									
Cue Phase of Task									
	Punishment > Neutral Cues	L. Paracingulate (BA8)	-1	31	39	184	**-0.37****	-0.27	-0.18
		L. Frontal Pole (BA9)	-15	57	35	2,512	-0.13	0.22	-0.04
		R. Frontal Pole (BA9)	3	61	31	1,214	-0.23	0.15	-0.00
		R. Posterior Cingulate Cortex (BA30)	3	-45	15	72	**-0.35***	-0.19	-0.23
Word Phase of Task									
	Neutral > Positive Words	L. Precuneus (BA7)	-5	-71	55	760	0.04	0.18	-0.1
		L. Primary Motor Cortex (BA4)	-39	-17	43	800	-0.15	0.01	-0.15
		L. Primary Somatosensory Cortex (BA1)	-57	-11	21	352	-0.04	-0.14	-0.22
Feedback Phase of Task									
	Neutral > Rewarding Feedback	L. Primary Somatosensory Cortex (BA1)	-37	-41	57	2,864	-0.18	-0.02	**-0.30***
		R. Superior Frontal Gyrus (BA6)	21	3	57	160	-0.19	-0.19	**-0.30***
		R. Primary Somatosensory Cortex (BA1)	45	-23	45	248	**-0.36****	-0.21	**-0.36***
		L. Primary Somatosensory Cortex (BA1)	-43	-21	41	128	-0.25	0.04	-0.25

Table 6. Significant brain regions revealed in *a priori* analyses are organized by dimension of motivation and contrast. Relationship with change psychopathology was calculated using regression. Extracted brain data and T1 psychopathology were used as IVs and T2 psychopathology was used as the DV. AA = Anxious arousal, Low PA = Depressive low positive affect, L. = Left, R. = Right, BA = Brodmann's Area, vmPFC = ventromedial prefrontal cortex, mOFC = medial orbital frontal cortex. * = > *0.05* ** = > *0.01*

	R. Somatosensory	L. Somatosensory	Superior Frontal
R. Somatosensory	—		
L. Somatosensory	0.650**	—	
Superior Frontal	0.511**	0.623**	—

Table 7. Correlation among brain regions sensitive to the receipt of less positive/disappointing feedback and the adaptivity score. R = Right, L = Left. N = 85, ** = $p < 0.001$.

FIGURES

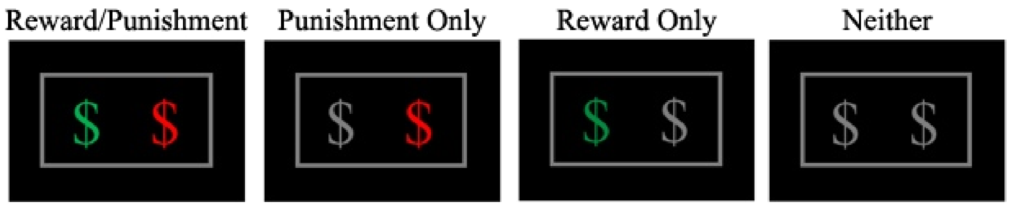

Figure 1. Cues signaling the potential outcome of each trial. Each cue was represented equally across the three blocks of the task.

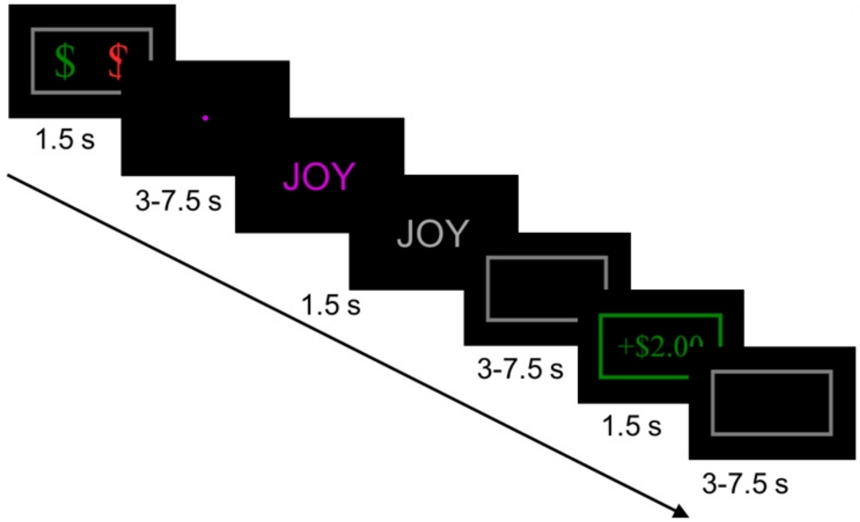

Figure 2. Monetary Incentive Delay trial sequence.

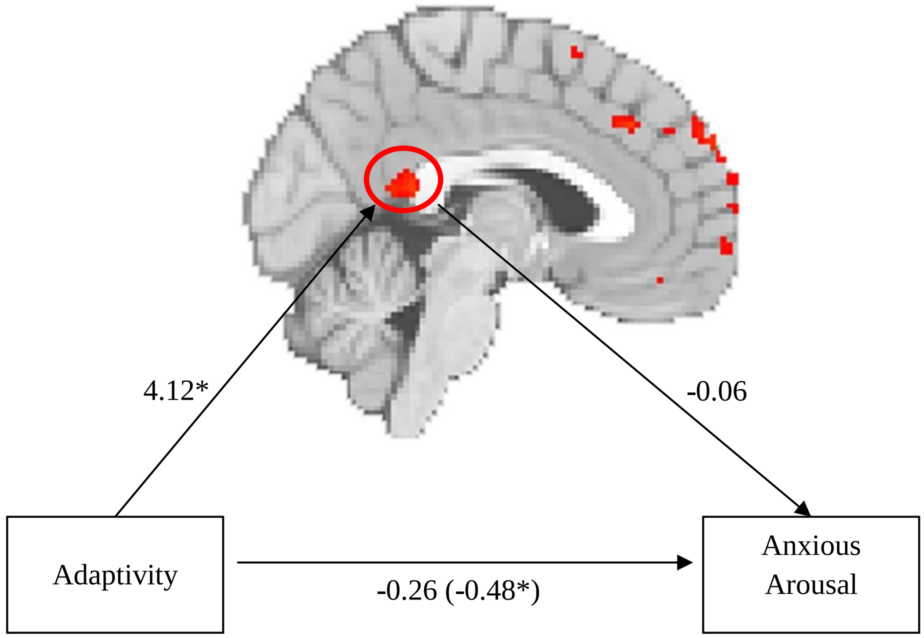

Figure 3. Mediation model from *a priori* analysis with right posterior cingulate cortex (PCC) activity associated with the punishment > neutral cue contrast as the mediating variable between the adaptivity score and changes in anxious arousal. Neural data is thresholded at a family-wise error rate of 0.05. MNI coordinates of peak voxel (X, Y, Z) = (3, -43, 15). * = $p < 0.05$.

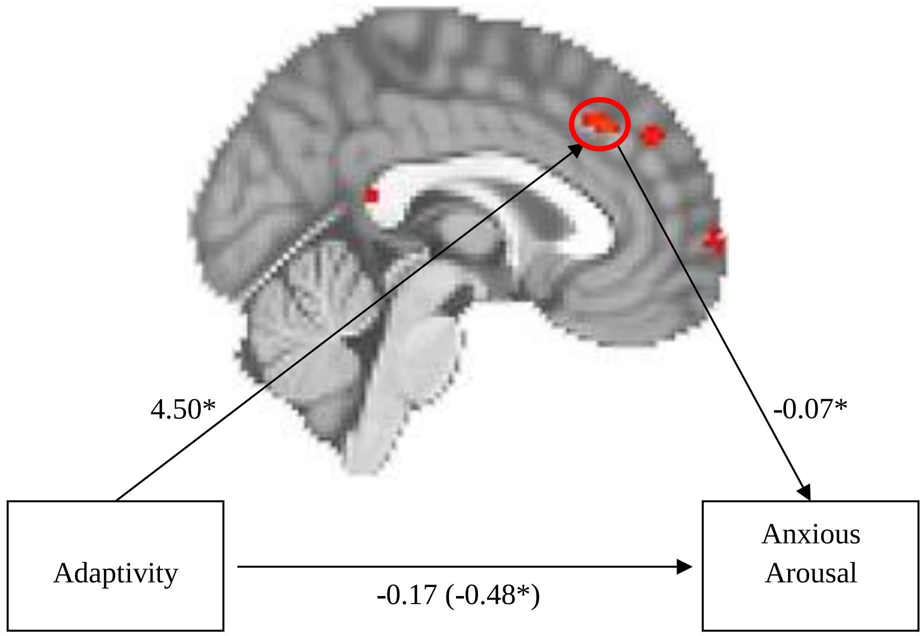

Figure 4. Mediation model with left paracingulate cortex activity associated with the punishment > neutral cue contrast as the mediating variable between the adaptivity score and changes in anxious arousal. Neural data is thresholded at a family-wise error rate of 0.05. MNI coordinates of peak voxel (X, Y, Z) = (-1, 31, 39). * = $p < 0.05$.

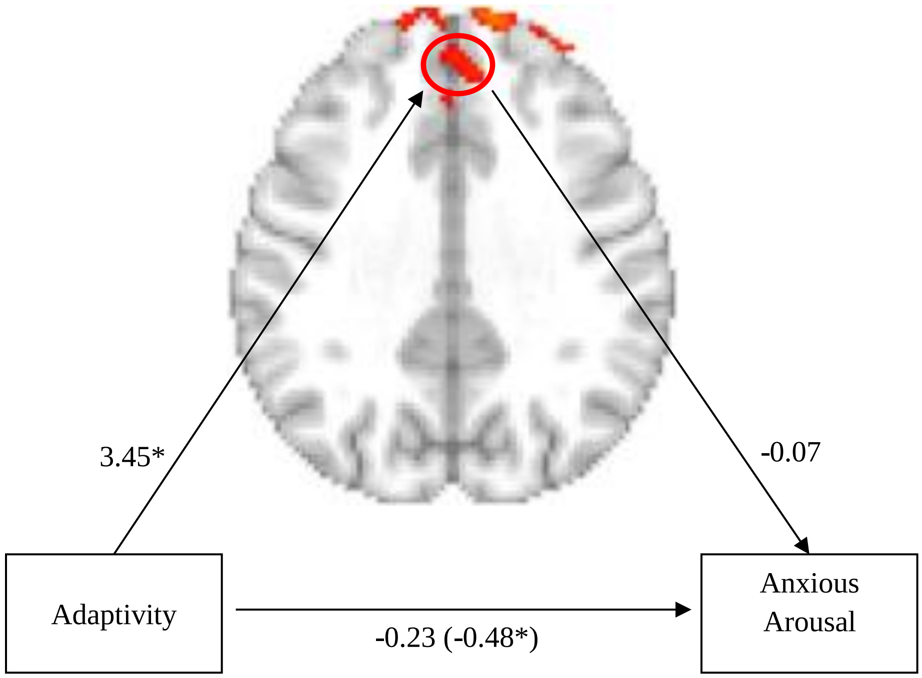

Figure 5. Mediation model with left superior frontal gyrus activity associated with the punishment > neutral cue contrast as the mediating variable between the adaptivity score and changes in anxious arousal. Neural data is thresholded at a family-wise error rate of 0.05. MNI coordinates of peak voxel (X, Y, Z) = (-3, 45, 35). * = $p < 0.05$.

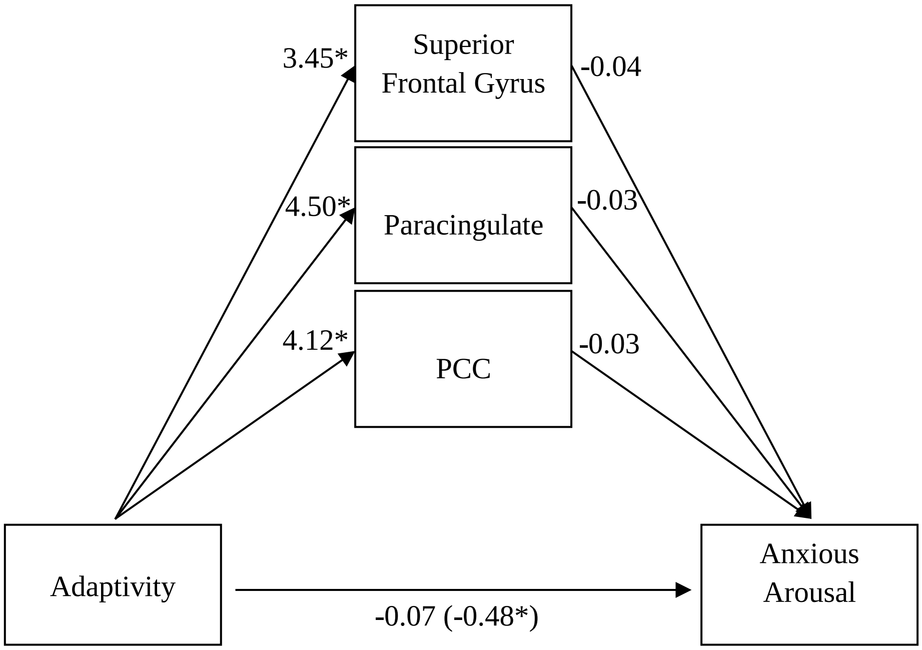

Figure 6. Multiple mediation model with posterior cingulate cortex (PCC), paracingulate, and superior frontal gyrus activity associated with the punishment > neutral cue contrast as the mediating variables between the adaptivity score and changes in anxious arousal. * = $p < 0.05$.

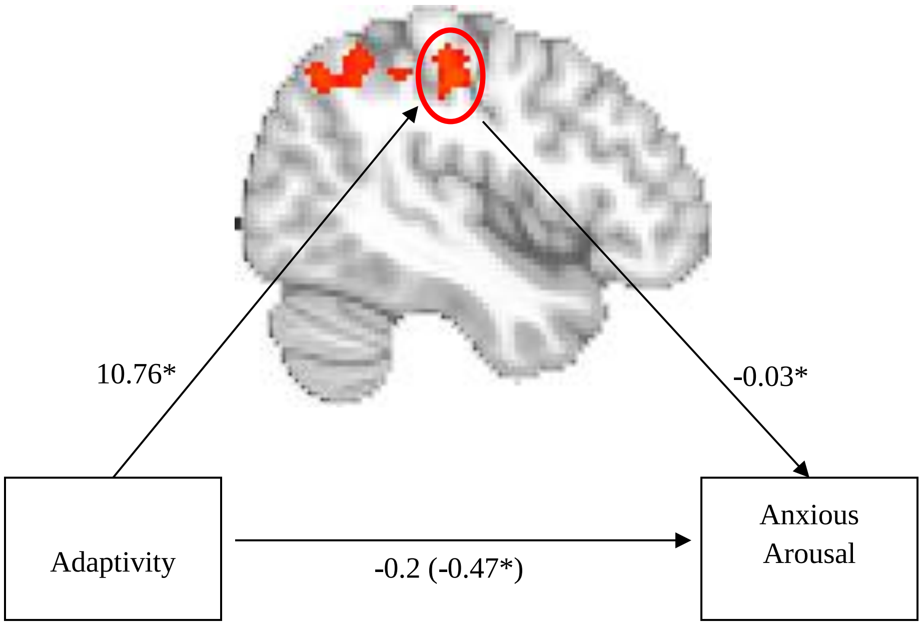

Figure 7. Mediation model with right somatosensory cortex activity associated with the neutral > positive feedback contrast as the mediating variable between the adaptivity score and changes in anxious arousal. Neural data is thresholded at a family-wise error rate of 0.05. MNI coordinates of peak voxel (X, Y, Z) = (45, -23, 45). * = $p < 0.05$.

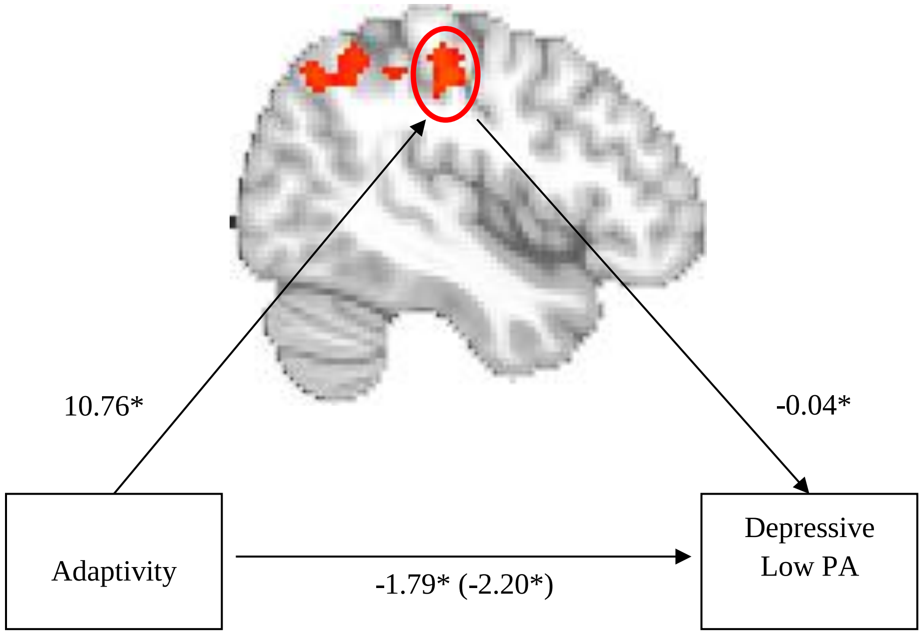

Figure 8. Mediation model with right somatosensory cortex activity associated with the neutral > positive feedback contrast as the mediating variable between the adaptivity score and changes in depressive low positive affect. Neural data is thresholded at a family-wise error rate of 0.05. MNI coordinates of peak voxel (X, Y, Z) = (45, -23, 45). Depressive low PA = Depressive low positive affect, * = $p < 0.05$.

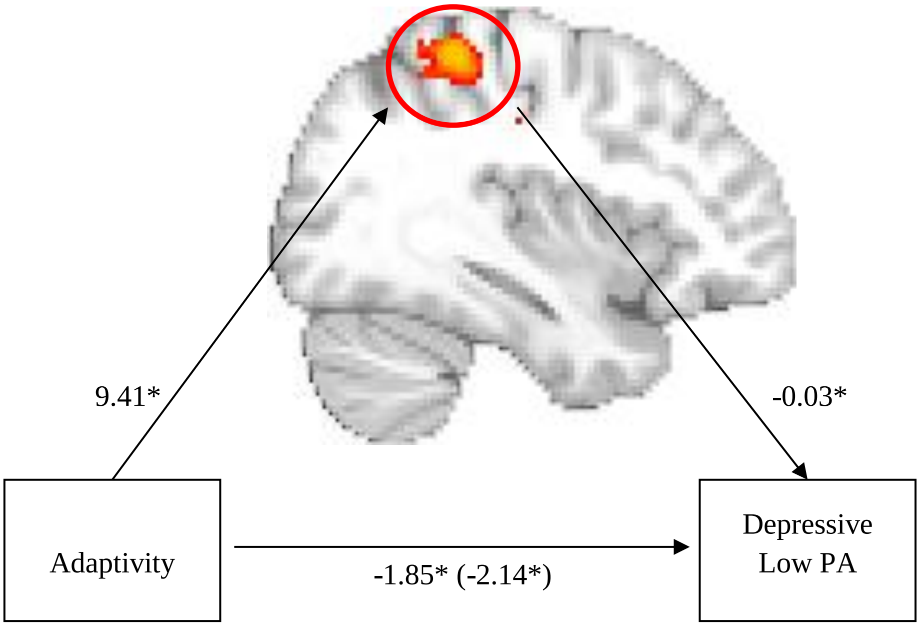

Figure 9. Mediation model with left somatosensory cortex activity associated with the neutral > positive feedback contrast as the mediating variable between the adaptivity score and changes in depressive low positive affect. Neural data is thresholded at a family-wise error rate of 0.05. MNI coordinates of peak voxel (X, Y, Z) = (-37, -41, 57). L = left, Depressive low PA = Depressive low positive affect, * = $p < 0.05$.

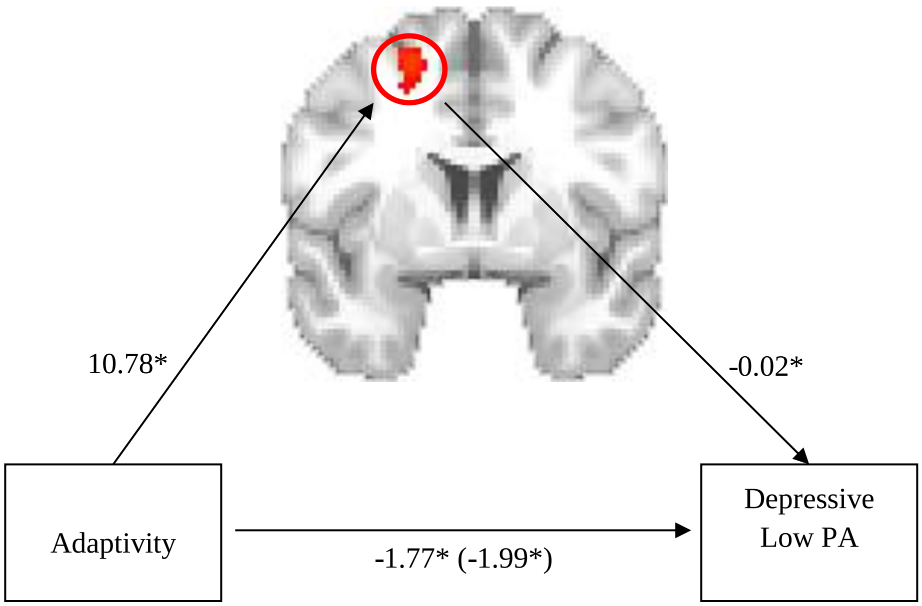

Figure 10. Mediation model with superior frontal gyrus activity associated with the neutral > positive feedback contrast as the mediating variable between the adaptivity score and changes in depressive low positive affect. Neural data is thresholded at a family-wise error rate of 0.05. MNI coordinates of peak voxel (X, Y, Z) = (21, 3, 57). Depressive low PA = Depressive low positive affect, * = $p < 0.05$.

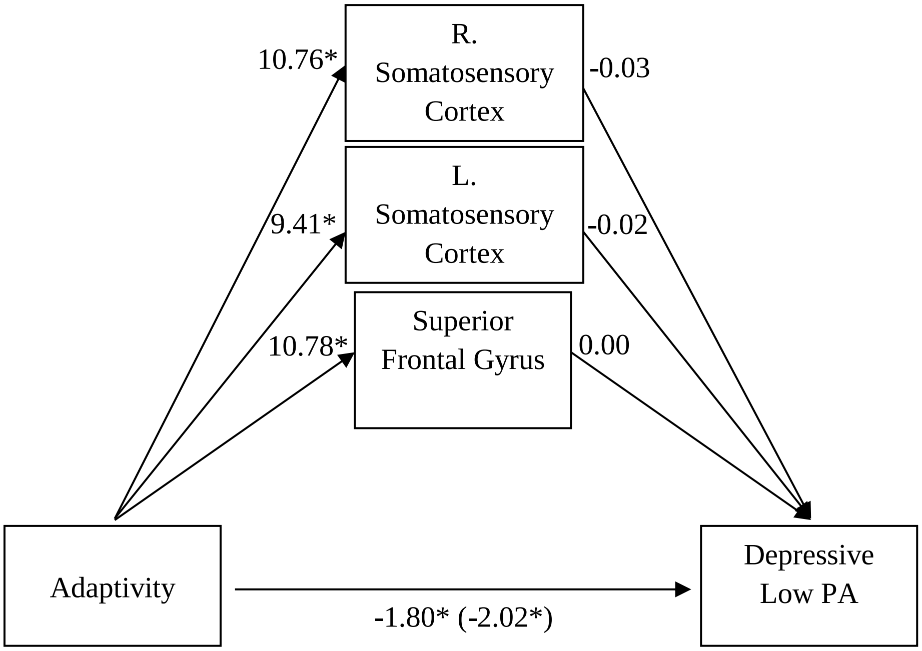

Figure 11. Multiple mediation model with right somatosensory cortex, left somatosensory cortex, and superior frontal gyrus activity associated with the neutral > positive feedback contrast as the mediating variables between the adaptivity score and changes in depressive low positive affect. R = Right, L= Left, Depressive low PA = Depressive low positive affect, * = $p < 0.05$.

REFERENCES

Aldoa, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review, 30*, 217-237.

Andersson, J. L. R., Jenkinson M., Smith, S. (2010). Non-linear registration, aka spatial normalisation. FMRIB technical report TR07JA2.

Andrews, V. H., & Borkovec, T. D. (1988). The differential effects of inductions of worry, somatic anxiety, and depression on emotional experience. *Journal of Behavior Therapy and Experimental Psychiatry*, *19*(1), 21-26. doi:10.1016/0005-7916(88)90006-7.

Aron, A. R., Fletcher, P. C., Bullmore, E. T., Sahakian, B. J., & Robbins, T. W. (2003). Stop-signal inhibition disrupted by damage to right inferior frontal gyrus in humans. *Nature neuroscience*, *6*(2), 115-116.

Aron, A. R., Robbins, T. W., & Poldrack, R. A. (2004). Inhibition and the right inferior frontal cortex. *Trends in cognitive sciences*, *8*(4), 170-177.

Aupperle Robin, L., & Martin, P. P. (2010). Neural systems underlying approach and avoidance in anxiety disorders. *Dialogues Clin Neurosci*, *12*(4), 517-531.

Badour, C. L., Blonigen, D. M., Boden, M. T., Feldner, M. T., & Bonn-Miller, M. O. (2012). A longitudinal test of the bi-directional relations between avoidance coping and PTSD severity during and after PTSD treatment. *Behaviour research and therapy, 50(10)*, 610-616. doi: 10.1016/j.brat.2012.06.006

Barlow, D. H. (2002). *Anxiety and its disorders: The nature and treatment of anxiety and panic* (2nd ed.). New York, NY: Guilford Press

Barlow, D. H., Sauer-Zavala, S., Carl, J. R., Bullis, J. R., & Ellard, K. K. (2014). The nature, diagnosis, and treatment of neuroticism back to the future. *Clinical Psychological Science, 2*(3), 344-365. doi: 10.1177/2167702613505532.

Bates, J. E. (1987). Temperament in infancy. In J. Osofsky (Ed.), *Handbook of infant development* (2nd ed., pp. 1101–1149). New York: Wiley Press

Bechara, A., & Damasio, A. R. (2005). The somatic marker hypothesis: A neural theory of economic decision. *Games and economic behavior, 52(2)*, 336-372.

Beck, A. T. (1967). *Depression: Clinical, experimental, and theoretical aspects*. University of Pennsylvania Press.

Bentham, J. (1779/1879). Introduction to the principles of morals and legislation. Oxford: Clarendon Press.

Biederman, J., Hirshfeld-Becker, D. R., Rosenbaum, J. F., Hérot, C., Friedman, D., Snidman, N., Kagan, J., & Faraone, S. V. (2001). Further evidence of association between behavioral inhibition and social anxiety in children, *American journal of Psychiatry*, *158*(10), 1673-1679.

Bower, G. H., & Forgas, J. P. (2000). Affect, memory, and social cognition.

Bradley, B. P., Mogg, K., Millar, N., & White, J. (1995). Selective processing of negative information: effects of clinical anxiety, concurrent depression, and awareness. *Journal of abnormal psychology*, *104*(3), 532.

Bradley, B. P., Mogg, K., & Williams, R. (1995). Implicit and explicit memory for emotion-congruent information in clinical depression and anxiety. *Behaviour research and therapy*, *33*(7), 755-770.

Bradley, M. M., & Lang, P. J. (1999). *Affective norms for English words (ANEW): Instruction manual and affective ratings* (pp. 1-45). Technical report C-1, the center for research in psychophysiology, University of Florida.

Brainard, D. H. (1997). The psychophysics toolbox. *Spatial vision*, *10*, 433-436.

Bredemeier, K., Spielberg, J. M., Silton, R. L., Berenbaum, H., Heller, W., & Miller, G. A. (2010). Screening for depressive disorders using the Mood and Anxiety Symptoms Questionnaire Anhedonic Depression Scale: a receiver-operating characteristic analysis. *Psychological assessment, 22(3)*, 702-710.

Bress, J. N., Foti, D., Kotov, R., Klein, D. N., & Hajcak, G. (2013). Blunted neural response to rewards prospectively predicts depression in adolescent girls. *Psychophysiology*, *50*(1), 74-81. doi: 10.1111/j.1469-8986.2012.01485.x.

Brown, T. A. (2007). Temporal course and structural relationships among dimensions of temperament and DSM–IV anxiety and mood disorder constructs. *Journal of Abnormal Psychology*, *116*, 313–328.

Brown, T. A., & Barlow, D. H. (2009). A proposal for a dimensional classification system based on the shared features of the DSM-IV anxiety and mood disorders: implications for assessment and treatment.*Psychological assessment*, *21*(3), 256.

Brown, T. A., Chorpita, B. F., & Barlow, D. H. (1998). Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal.*Journal of abnormal psychology*, *107*(2), 179. doi: 10.1037/0021-843X.107.2.179.

Brozina, K., & Abela, J. R. (2006). Behavioural inhibition, anxious symptoms, and depressive symptoms: A short-term prospective examination of a diathesis-stress model. *Behaviour Research and Therapy, 44*(9), 1337-1346.

Buss, A., & Plomin, R. (1984). *Temperament: Early developing personality traits*. Hillsdale, NJ: Erlbaum

Cain, C. K., & LeDoux, J. E. (2008). Emotional processing and motivation: in search of brain mechanisms. *Handbook of approach and avoidance motivation*, 17-34.

Campbell-Sills, L., Liverant, G. I., & Brown, T. A. (2004). Psychometric evaluation of the behavioral inhibition/behavioral activation scales in a large sample of outpatients with anxiety and mood disorders. *Psychological Assessment, 16*, 244–254.

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: the BIS/BAS scales. *Journal of personality and social psychology, 67*(2), 319.

Caspi, A., Moffitt, T. E., Newman, D. L., & Silva, P. A. (1996). Behavioral observations at age 3 years predict adult psychiatric disorders: Longitudinal evidence from a birth cohort. *Archives of general psychiatry, 53*(11), 1033-1039. doi: 10.1037/0022-006X.64.3.552.

Chawla, N., & Ostafin, B. (2007). Experiential Avoidance as a Functional Dimensional Approach to Psychopathology: An Empirical Review. *Journal of Clinical Psychology, Vol. 63(9)*, 871–890. doi: 10.1002/jclp.20400

Chorpita, B. F., & Barlow, D. H. (1998). The development of anxiety: the role of control in the early environment. *Psychological bulletin*, *124*(1), 3. doi: 10.1037/0033-2909.124.1.3.

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: psychometric evidence and taxonomic implications. *Journal of abnormal psychology*, *100*(3), 316. doi: 10.1037/0021-843X.100.3.316.

Clark, L. A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of abnormal psychology*, *103*(1), 103. doi:10.1037/0021-843X.103.1.103.

Cornwell, J. F., Franks, B., & Higgins, E. T. (2014). Truth, control, and value motivations: the "what," "how," and "why" of approach and avoidance. *Frontiers in systems neuroscience*, *8*, 194. doi:10.3389/fnsys.2014.00194.

Cunningham, W. A., & Brosch, T. (2012). Motivational salience amygdala tuning from traits, needs, values, and goals. *Current Directions in Psychological Science*, *21*(1), 54-59. doi: 10.1177/0963721411430832.

Daniel, R., & Pollmann, S. (2014). A universal role of the ventral striatum in reward-based learning: Evidence from human studies. *Neurobiology of learning and memory*, *114*, 90-100.

Davidson, R. J. (1993). Cerebral asymmetry and emotion: Conceptual and methodological conundrums. *Cognition and Emotion, 7*, 115–138.

Davidson, R. J. (1998). Anterior electrophysiological asymmetries, emotion, and depression: Conceptual and methodological conundrums. *Psychophysiology*, *35*(05), 607-614.

Davidson, R. J. (2002). Anxiety and affective style: role of prefrontal cortex and amygdala. *Biological psychiatry*, *51*(1), 68-80. doi: 10.1016/S0006-3223(01)01328-2.

Davidson, R. J., Ekman, P., Saron, C. D., Senulis, J. A., & Friesen, W. V. (1990). Approach-withdrawal and cerebral asymmetry: Emotional expression and brain physiology: I. *Journal of personality and social psychology*, *58*(2), 330.

Davidson, R. J., & Irwin, W. (1999). The functional neuroanatomy of emotion and affective style. *Trends in cognitive sciences*, *3*(1), 11-21.

Davidson, R. J., Abercrombie, H., Nitschke, J. B., & Putnam, K. (1999). Regional brain function, emotion and disorders of emotion. *Current opinion in neurobiology*, *9*(2), 228-234. doi:10.1016/S0959-4388(99)80032-4.

Davidson, R. J., Pizzagalli, D., Nitschke, J. B., & Putnam, K. (2002). Depression: perspectives from affective neuroscience. *Annual review of psychology*, *53*(1), 545-574. doi: 10.1146/annurev.psych.53.100901.135148.

Debener, S., Beauducel, A. E., Nessler, D., Brocke, B., Heilemann, H., & Kayser, J. U. R. (2000). Is resting anterior EEG alpha asymmetry a trait marker for depression?. *Neuropsychobiology*, *41*(1), 31-37.

De Beurs, E., Beekman, A. T. F., Deeg, D. J. H., Van Dyck, R., & Van Tilburg, W. (2000). Predictors of change in anxiety symptoms of older persons: results from the Longitudinal Aging Study Amsterdam. *Psychological Medicine*, *30*(03), 515-527.

Delgado, M. R. (2007). Reward- related responses in the human striatum. *Annals of the New York Academy of Sciences*, *1104*(1), 70-88.

Derryberry, D., & Reed, M. A. (1998). Anxiety and attentional focusing: Trait, state and hemispheric influences. *Personality and Individual differences*, *25*(4), 745-761.

Dickson, J., & MacLeod, A. (2004). Brief Report Anxiety, depression and approach and avoidance goals. *Cognition and Emotion*, *18*(3), 423-430. doi: 10.1080/02699930341000013.

DiStefano, C., Zhu, M., & Mindrila, D. (2009). Understanding and using factor scores: Considerations for the applied researcher. *Practical Assessment, Research & Evaluation*, *14*(20), 1-11.

Dougherty, D., & Rauch, S. L. (1997). Neuroimaging and neurobiological models of depression. *Harvard Review of Psychiatry*, *5*(3), 138-159.

Drevets, W. C. (2001). Neuroimaging and neuropathological studies of depression: implications for the cognitive-emotional features of mood disorders. *Current opinion in neurobiology*, *11*(2), 240-249.

Eddington, K. M., Dolcos, F., Cabeza, R., Krishnan, K. R. R., & Strauman, T. J. (2007). Neural correlates of promotion and prevention goal activation: an fMRI study using an idiographic approach. *Journal of Cognitive Neuroscience*, *19*(7), 1152-1162. doi:10.1162/jocn.2007.19.7.1152.

Elliot, A. J. (1999). Approach and avoidance motivation and achievement goals. *Educational psychologist*, *34*(3), 169-189. doi: 10.1207/s15326985ep3403_3.

Elliot, A. J. (2006). The hierarchical model of approach-avoidance motivation. *Motivation and emotion*, *30*(2), 111-116. doi: 10.1007/s11031-006-9028-7.

Elliot, A. J., & Thrash, T. M. (2002). Approach-avoidance motivation in personality: approach and avoidance temperaments and goals. *Journal of personality and social psychology*, *82*(5), 804.

Elliot, A. J., & Thrash, T. M. (2010). Approach and avoidance temperament as basic dimensions of personality. *Journal of personality*, *78*(3), 865-906. doi: 10.1111/j.1467-6494.2010.00636.x.

Engels, A. S., Heller, W., Mohanty, A., Herrington, J. D., Banich, M. T., Webb, A. G., & Miller, G. A. (2007). Specificity of regional brain activity in anxiety types during emotion processing. *Psychophysiology, 44*(3), 352-363. doi: 10.1111/j.1469-8986.2007.00518.x.

Engels, A. S., Heller, W., Spielberg, J. M., Warren, S. L., Sutton, B. P., Banich, M. T., & Miller, G. A. (2010). Co-occurring anxiety influences patterns of brain activity in depression. *Cognitive, Affective, & Behavioral Neuroscience*, *10*(1), 141-156. doi:10.3758/CABN.10.1.141.

Ernst, M., & Fudge, J. L. (2009). A developmental neurobiological model of motivated behavior: anatomy, connectivity and ontogeny of the triadic nodes. *Neuroscience & Biobehavioral Reviews*, *33*(3), 367-382.

Ferster, C. (1973). A functional analysis of depression. *American psychologist*, *28*(10), 857.

Fitzgerald, P. B., Laird, A. R., Maller, J., & Daskalakis, Z. J. (2008). A meta-analytic study of changes in brain activation in depression. *Human brain mapping*, *29*(6), 683-695. doi: 10.1002/hbm.20426.

Fonov, V. S., Evans, A. C., McKinstry, R. C., Almli, C. R., & Collins, D. L. (2009). Unbiased nonlinear average age-appropriate brain templates from birth to adulthood. *NeuroImage*, *47*, S102.

Fowles, D. C. (1988). Psychophysiology and psychopathology: A motivational approach. Psychophysiology, 25(4), 373-391. doi: 10.1111/j.1469-8986.1988.tb01873.x.

Freud, S. (1915). Repression. In the standard edition of *Complete Psychological*

Works of Sigmund Freud, vol. XIV. London: Hogarth, 1957.

Gainotti, G. (1972). Emotional behavior and hemispheric side of the lesion. *Cortex*, *8*(1), 41-55.

Gershuny, B. S., & Sher, K. J. (1998). The relation between personality and anxiety: findings from a 3-year prospective study. *Journal of abnormal psychology*, *107*(2), 252. doi:10.1037/0021-843X.107.2.252.

Goldin, P. R., & Gross, J. J. (2010). Effects of mindfulness-based stress reduction (MBSR) on emotion regulation in social anxiety disorder. *Emotion, 10(1)*, 83. doi: 10.1037/a0018441.

Gotlib, I. H., & Joormann, J. (2010). Cognition and depression: current status and future directions. *Annual review of clinical psychology*, *6*, 285. doi:10.1146/annurev.clinpsy.121208.131305.

Gotlib, I. H., Yue, D. N., & Joormann, J. (2005). Selective attention in dysphoric individuals: the role of affective interference and inhibition. *Cognitive Therapy and Research*, *29*(4), 417-432.

Gottfried, J. A., O'Doherty, J., & Dolan, R. J. (2002). Appetitive and aversive olfactory learning in humans studied using event-related functional magnetic resonance imaging. *The Journal of Neuroscience*, *22*(24), 10829-10837.

Gräsbeck, A., Hagnell, O., Otterbeck, L., & Rorsman, B. (1993). Anxiety in the Lundby Study: re-evaluation according to DSM-III-R, incidence and risk. *Neuropsychobiology*, *27*(1), 1-8.

Gray, J. A. (1982). On mapping anxiety. *Behavioral and Brain Sciences*, *5*(03), 506-534.

Gray, J. A., & McNaughton, N. (1996). The neuropsychology of anxiety: Reprise. In *Nebraska symposium on motivation* (Vol. 43, pp. 61-134). University of Nebraska Press.

Griffith, J. W., Zinbarg, R. E., Craske, M. G., Mineka, S., Rose, R. D., Waters, A. M., & Sutton, J. M. (2010). Neuroticism as a common dimension in the internalizing disorders. *Psychological Medicine*, *40*, 1125–1136.

Harmon-Jones, E., Harmon-Jones, C., & Price, T. F. (2013). What is approach motivation?. *Emotion Review*, *5*(3), 291-295. doi: 10.1177/1754073913477509.

Heller, W. (1993). Neuropsychological mechanisms of individual differences in emotion, personality, and arousal. *Neuropsychology*, *7*(4), 476. doi:10.1037/0894-4105.7.4.476.

Heller, W., Etienne, M. A., & Miller, G. A. (1995). Patterns of perceptual asymmetry in depression and anxiety: implications for neuropsychological models of emotion and psychopathology. *Journal of abnormal psychology*,*104*(2), 327. doi: 10.1037/0021-843X.104.2.327.

Heller, W., Nitschke, J. B., Etienne, M. A., & Miller, G. A. (1997). Patterns of regional brain activity differentiate types of anxiety. *Journal of Abnormal Psychology*, *106*(3), 376. doi: 10.1037/0021-843X.106.3.376.

Henderson, H. A., & Wachs, T. D. (2007). Temperament theory and the study of cognition–emotion interactions across development. *Developmental Review*, *27*(3), 396-427. doi:10.1016/j.dr.2007.06.004.

Henriques, J. B., & Davidson, R. J. (1991). Left frontal hypoactivation in depression. *Journal of abnormal psychology*, *100*(4), 535. doi: 10.1037/0021-843X.100.4.535.

Henriques, J. B., & Davidson, R. J. (2000). Decreased responsiveness to reward in depression. *Cognition & Emotion*, *14*(5), 711-724. doi: 10.1080/02699930050117684.

Herrington, J. D., Heller, W., Mohanty, A., Engels, A. S., Banich, M. T., Webb, A. G., & Miller, G. A. (2010). Localization of asymmetric brain function in emotion and depression. *Psychophysiology*, *47*(3), 442-454. doi:10.1111/j.1469-8986.2009.00958.x.

Hundt, N. E., Nelson-Gray, R. O., Kimbrel, N. A., Mitchell, J. T., & Kwapil, T. R. (2007). The interaction of reinforcement sensitivity and life events in the prediction of anhedonic depression and mixed anxiety-depression symptoms. *Personality and Individual Differences*, *43*(5), 1001-1012.

Isen, A. M. (1984). Toward understanding the role of affect in cognition. *Handbook of social cognition*, *3*, 179-236.

Isotani, T., Tanaka, H., Lehmann, D., Pascual-Marqui, R. D., Kochi, K., Saito, N., Yagyu, H., Kinoshita, T., & Sasada, K. (2001). Source localization of EEG activity during hypnotically induced anxiety and relaxation. *International Journal of Psychophysiology*, *41*(2), 143-153.

James, W. (1890). *The principles of psychology* (vol. 2). NY: Henry Holt & Co.

Jenkinson, M., Bannister, P., Brady, M., & Smith, S. (2002). Improved optimization for the robust and accurate linear registration and motion correction of brain images. *Neuroimage*, *17*(2), 825-841.

Jezzard, P., & Balaban, R. S. (1995). Correction for geometric distortion in echo planar images from B0 field variations. *Magnetic resonance in medicine*, *34*(1), 65-73.

Johnson, S. L., Turner, R. J., & Iwata, N. (2003). BIS/BAS levels and psychiatric disorder: An epidemiological study. *Journal of Psychopathology and Behavioral Assessment*, *25*(1), 25-36. doi: 10.1023/A:1022247919288.

Joiner, T. E., & Lonigan, C. J. (2000). Tripartite model of depression and anxiety in youth psychiatric inpatients: Relations with diagnostic status and future symptoms. *Journal of Clinical Child Psychology*, *29*(3), 372-382.

Joormann, J., & Gotlib, I. H. (2006). Is this happiness I see? Biases in the identification of emotional facial expressions in depression and social phobia. *Journal of abnormal psychology*, *115*(4), 705.

Joormann, J., & Gotlib, I. H. (2007). Selective attention to emotional faces following recovery from depression. *Journal of abnormal psychology*, *116*(1), 80.

Kagan, J. (1994). *Galen's prophecy.* New York: Basic Books.

Kasch, K. L., Rottenberg, J., Arnow, B. A., & Gotlib, I. H. (2002). Behavioral activation and inhibition systems and the severity and course of depression. *Journal of abnormal psychology*, *111*(4), 589. doi: 10.1037/0021-843X.111.4.589.

Kendler, K. S., Neale, M. C., Kessler, R. C., Heath, A. C., & Eaves, L. J. (1993). Panic disorder in women: a population-based twin study. *Psychological medicine*, *23*(02), 397-406.

Kessler, R. C. (2004). The epidemiology of dual diagnosis. *Biological psychiatry*, *56*(10), 730-737.

Kessler, R., Berglund, P., Demler, O., Jin, R., Merikangas, K., Walters, E., et al. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, *62*(6), 593-603.

Kessler, R., DuPont, R., Berglund, P., & Wittchen, H. (1999). Impairment in pure and comorbid generalized anxiety disorder and major depression at 12 months in two national surveys. *American Journal of Psychiatry*, *156*, 1915-1923. doi:10.1002/da.20019.

Kessler, R. C., & Walters, E. E. (1998). Epidemiology of DSM-III-R major depression and minor depression among adolescents and young adults in the National Comorbidity Survey. *Depression and Anxiety, 7*, 3-14.

Kringelbach, M. L., O'Doherty, J., Rolls, E. T., & Andrews, C. (2003). Activation of the human orbitofrontal cortex to a liquid food stimulus is correlated with its subjective pleasantness. *Cerebral cortex*, *13*(10), 1064-1071.

Krishnan, V., & Nestler, E. J. (2008). The molecular neurobiology of depression. *Nature*, *455*(7215), 894-902.

Krueger, R. F., Caspi, A., Moffitt, T. E., Silva, P. A., & McGee, R. (1996). Personality traits are differentially linked to mental disorders: a multitrait-multidiagnosis study of an adolescent birth cohort. *Journal of abnormal psychology, 105(3),* 299.

Layne, C., Gross, R. S., & Buckley, M. F. (1980). Ratio scales of the reward values and punisher aversions of depressed undergraduates. *Journal of Clinical Psychology*, *36*(3), 640-646.

Layne, C., Merry, J., Christian, J., & Ginn, P. (1982). Motivational deficit in depression. *Cognitive Therapy and Research*, *6*(3), 259-273. doi: 10.1007/BF01173575.

Legon, W., Sato, T. F., Opitz, A., Mueller, J., Barbour, A., Williams, A., & Tyler, W. J. (2014). Transcranial focused ultrasound modulates the activity of primary somatosensory cortex in humans. *Nature Neuroscience, 17(2),* 322-329. DOI: 10.1038/nn.3620

Lengua, L. J., Wolchik, S. A., Sandler, I. N., & West, S. G. (2000). The additive and interactive effects of parenting and temperament in predicting adjustment problems of children of divorce. *Journal of Clinical Child Psychology*, *29*(2), 232-244.

Luria, A. R. (1973). The frontal lobes and the regulation of behavior. *Psychophysiology of the frontal lobes, 332*, 3-26.

MacLeod, C., & Mathews, A. (2012). Cognitive bias modification approaches to anxiety. *Annual Review of Clinical Psychology*, *8*, 189-217.

Mathews, A., & MacLeod, C. (2005). Cognitive vulnerability to emotional disorders. *Annu. Rev. Clin. Psychol.*, *1*, 167-195.

Mathews, A., Ridgeway, V., & Williamson, D. A. (1996). Evidence for attention to threatening stimuli in depression. *Behaviour research and therapy*, *34*(9), 695-705.

McCrae, R. R., & Costa, P. T. (2004). A contemplated revision of the NEO Five-Factor Inventory. *Personality and individual differences*, *36*(3), 587-596. doi: 10.1016/S0191-8869(03)00118-1.

McFarland, B. R., Shankman, S. A., Tenke, C. E., Bruder, G. E., & Klein, D. N. (2006). Behavioral activation system deficits predict the six-month course of depression. *Journal of affective disorders*, *91*(2), 229-234. doi: 10.1016/j.jad.2006.01.012.

Meehl, P. E. (1975). Hedonic capacity: Some conjectures. *Bulletin of the Menninger Clinic*, *39*(4), 295.

Miller, G. A., Crocker, L. D., Spielberg, J. M., Infantolino, Z. P., & Heller, W. (2013). Issues in localization of brain function: the case of lateralized frontal cortex in cognition, emotion, and psychopathology. *Frontiers in Integrative Neuroscience, 7, 2.* doi:10.3389/fnint.2013.00002.

Mineka, S., & Zinbarg, R. (2006). A contemporary learning theory perspective on the etiology of anxiety disorders: it's not what you thought it was. *American psychologist*, *61*(1), 10. doi: 10.1037/0003-066X.61.1.10.

Mogg, K., & Bradley, B. P. (1998). A cognitive-motivational analysis of anxiety. *Behaviour research and therapy*, *36*(9), 809-848.

Mogg, K., & Bradley, B. P. (2005). Attentional bias in generalized anxiety disorder versus depressive disorder. *Cognitive therapy and research*, *29*(1), 29-45.

Mogg, K, Bradley, B. P., Williams, R., & Mathews, A. (1993). Subliminal processing of emotional information in anxiety and depression. *Journal of Abnormal Psychology, 102*, 304–311.

Montague P. R., & Berns G. S. (2002). Neural economics and the biological substrates of valuation. *Neuron, 36*, 265–284.

Moulds, M. L., Kandris, E., Starr, S., & Wong, A. C. (2007). The relationship between rumination, avoidance and depression in a non-clinical sample. *Behaviour research and therapy*, *45*(2), 251-261.

Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology: views from cognitive and personality psychology and a working inhibition taxonomy. *Psychological bulletin*, *126*(2), 220. doi:10.1037/0033-2909.126.2.220.

Nigg, J. T. (2006). Temperament and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, *47*(3-4), 395-422. doi:10.1111/j.1469-7610.2006.01612.x.

Nitschke, J. B. (1998). The puzzle of regional brain activity in and anxiety: the importance of subtypes and comorbidity. *Cognition & Emotion*,*12*(3), 421-447. doi:10.1080/026999398379664.

Nitschke, J. B., Heller, W., Imig, J. C., McDonald, R. P., & Miller, G. A. (2001). Distinguishing dimensions of anxiety and depression. *Cognitive Therapy and Research*, *25*(1), 1-22. doi: 10.1023/A:1026485530405.

Nitschke, J. B., Heller, W., Palmieri, P.A., & Miller, G.A. (1999). Contrasting patterns of brain activity in anxious apprehension and anxious arousal. *Psychophysiology, 36,* 628-637.

Nitschke, J. B., Sarinopoulos, I., Oathes, D. J., Johnstone, T., Whalen, P. J., Davidson, R. J., & Kalin, N. H. (2009). Anticipatory activation in the amygdala and anterior cingulate in generalized anxiety disorder and prediction of treatment response. *American Journal of Psychiatry*, 166(3), 302-310.

Noguchi, K., Gohm, C. L., & Dalsky, D. J. (2006). Cognitive tendencies of focusing on positive and negative information. *Journal of Research in Personality*, *40*(6), 891-910. doi:10.1016/j.jrp.2005.09.008.

O'Doherty, J. P. (2004). Reward representations and reward-related learning in the human brain: insights from neuroimaging. *Current opinion in neurobiology*, *14*(6), 769-776.

O'Doherty, J. P. (2007). Lights, camembert, action! The role of human orbitofrontal cortex in encoding stimuli, rewards, and choices. *Annals of the New York Academy of Sciences*, *1121*(1), 254-272.

Orr J. M., Smolker H. R., & Banich M. T. (2015). Organization of the human frontal pole revealed by large-scale DTI-based connectivity: Implications for control of behavior. *PLoS ONE 10(5).* 1-23.

Padoa-Schioppa C., & Assad J. A. (2006). Neurons in orbitofrontal cortex encode economic value. *Nature, 441*. 223–226.

Peeva, M. G., Guenther, F. H., Tourville, J. A., Nieto-Castanon, A., Anton, J.-L., Nazarian, B., & Alario, F.-X. (2010). Distinct representations of phonemes, syllables, and supra-syllabic sequences in the speech production network. *NeuroImage*, *50(2)*, 626–638. http://doi.org/10.1016/j.neuroimage.2009.12.065

Pelli, D. G. (1997). The VideoToolbox software for visual psychophysics: Transforming numbers into movies. *Spatial vision, 10*(4), 437-442.

Pessoa, L., & Adolphs, R. (2010). Emotion processing and the amygdala: from a'low road'to'many roads' of evaluating biological significance. *Nature reviews neuroscience*, *11*(11), 773-783.

Pfleiderer, B., Zinkirciran, S., Arolt, V., Heindel, W., Deckert, J., & Domschke, K. (2007). fMRI amygdala activation during a spontaneous panic attack in a patient with panic disorder. *The World Journal of Biological Psychiatry*, *8*(4), 269-272.

Pinto-Meza, A., Caseras, X., Soler, J., Puigdemont, D., Pérez, V., & Torrubia, R. (2006). Behavioural inhibition and behavioural activation systems in current and recovered major depression participants. Personality and Individual Differences, 40(2), 215-226. doi:10.1016/j.paid.2005.06.021.

Pizzagalli, D. A., Holmes, A. J., Dillon, D. G., Goetz, E. L., Birk, J. L., Bogdan, R., ... & Fava, M. (2009). Reduced caudate and nucleus accumbens response to rewards in unmedicated individuals with major depressive disorder. *American Journal of Psychiatry*, *166*(6), 702-710. doi: 10.1176/appi.ajp.2008.08081201.

Porro, C. A., Francescato, M. P., Cettolo, V., Diamond, M. E., Baraldi, P., Zuiani, C., Bazzocchi, M., & Di Prampero, P. E. (1996). Primary motor and sensory cortex activation during motor performance and motor imagery: a functional magnetic resonance imaging study. *Journal of Neuroscience, 16(23)*, 7688-7698.

Radke-Yarrow, M. (1998). *Children of depressed mothers: From early childhood to maturity*. Cambridge University Press.

Reinherz, H. Z., Giaconia, R. M., Carmola, A. M., Wasserman, M. S., & Silverman, A. B. (1999). Major depression in the transition to adulthood: Risks and impairments. *Journal of Abnormal Psychology, 108*, 500-510.

Roberts, B. W., & Jackson, J. J. (2008). Sociogenomic personality psychology. *Journal of personality*, *76*(6), 1523-1544. doi: 10.1111/j.1467-6494.2008.00530.x.

Roberts, B. W., Caspi, A., & Moffitt, T. E. (2001). The kids are alright: growth and stability in personality development from adolescence to adulthood. *Journal of personality and social psychology*, *81*(4), 670. doi: 10.1037/0022-3514.81.4.670.

Roberts, S. B., & Kendler, K. S. (1999). Neuroticism and self-esteem as indices of the vulnerability to major depression in women. *Psychological medicine*, *29*(05), 1101-1109.

Rothbart, M. K., & Bates, J. E. (1998). Temperament. In W. Damon (Editor-In-Chief) & N. Eisenberg (Vol. Ed.), *Handbook of child psychology: Vol. 3. Social, emotional, and personality development* (5th ed., pp. 105–176). New York: Wiley.

Rothbart M. K., & Bates, J. E. (2008). Temperament. In Damon, W., Lerner, R. M. (Eds.), *Child and Adolescent Development: An Advanced Course* (pp 54-92). Hoboken, NJ: John Wiley & Sons.

Schoenbaum, G., & Esber, G. R. (2010). How do you (estimate you will) like them apples? Integration as a defining trait of orbitofrontal function. *Current opinion in neurobiology*, *20*(2), 205-211.

Schulenberg, J. E., & Zarrett, N. R. (2006). Mental health during emerging adulthood: continuity and discontinuity in courses, causes, and functions. In J. J. Arnett & J. L. Tanner (Eds.), Emerging adults in America: Coming of age in the 21st century (pp. 135-172). Washington, DC, US: American Psychological Association.

Schwartz, C. E., Snidman, N., & Kagan, J. (1999). Adolescent social anxiety as an outcome of inhibited temperament in childhood. *Journal of the American Academy of Child & Adolescent Psychiatry*, *38*(8), 1008-1015.

Shankman, S. A., & Klein, D. N. (2003). The relation between depression and anxiety: an evaluation of the tripartite, approach-withdrawal and valence-arousal models. *Clinical Psychology Review*, *23*(4), 605-637. doi: 10.1016/S0272-7358(03)00038-2.

Shankman, S. A., Klein, D. N., Tenke, C. E., & Bruder, G. E. (2007). Reward sensitivity in depression: a biobehavioral study. *Journal of abnormal psychology*, *116*(1), 95. doi: 10.1037/0021-843X.116.1.95.

Sharp, P. B., Miller, G. A., & Heller, W. (2015). Transdiagnostic dimensions of anxiety: neural mechanisms, executive functions, and new directions. *International Journal of Psychophysiology*, *98*(2), 365-377. doi: 10.1016/j.ijpsycho.2015.07.001.

Simon, G., Ormel, J., VonKorff, M., & Barlow, W. (1995). Health care costs associated with depressive and anxiety disorders in primary care. *American Journal of Psychiatry*, *152*(3), 352-357. doi:10.1176/ajp.152.3.352.

Simon, J. J., Walther, S., Fiebach, C. J., Friederich, H. C., Stippich, C., Weisbrod, M., & Kaiser, S. (2010). Neural reward processing is modulated by approach-and avoidance-related personality traits. *Neuroimage*, *49*(2), 1868-1874. doi:10.1016/j.neuroimage.2009.09.016.

Smith, S. M., & Nichols, T. E. (2009). Threshold-free cluster enhancement: addressing problems of smoothing, threshold dependence and localisation in cluster inference. *Neuroimage*, *44*(1), 83-98. doi: 10.1016/j.neuroimage.2008.03.061.

Smits, D. J., & Boeck, P. D. (2006). From BIS/BAS to the big five. *European Journal of Personality*, *20*(4), 255-270. doi: 10.1002/per.58.

Spielberg, J. M., Heller, W., & Miller, G. A. (2013). Hierarchical brain networks active in approach and avoidance goal pursuit. doi: 10.3389/fnhum.2013.00284.

Spielberg, J. M., Heller, W., Silton, R. L., Stewart, J. L., & Miller, G. A. (2011). Approach and avoidance profiles distinguish dimensions of anxiety and depression. *Cognitive Therapy and Research*, *35*(4), 359-371. doi: 10.1007/s10608-011-9364-0.

Spielberg, J. M., Miller, G. A., Engels, A. S., Herrington, J. D., Sutton, B. P., Banich, M. T., & Heller, W. (2011). Trait approach and avoidance motivation: lateralized neural activity associated with executive function. *Neuroimage*,*54*(1), 661-670. doi:10.1016/j.neuroimage.2010.08.037.

Spielberg, J. M., Miller, G. A., Warren, S. L., Engels, A. S., Crocker, L. D., Sutton, B. P., & Heller, W. (2012). Trait motivation moderates neural activation associated with goal pursuit. *Cognitive, Affective, & Behavioral Neuroscience*, *12*(2), 308-322. doi: 10.3758/s13415-012-0088-8.

Spielberg, J. M., Miller, G. A., Warren, S. L., Sutton, B. P., Banich, M., & Heller, W. (2014). Transdiagnostic dimensions of anxiety and depression moderate motivation-related brain networks during goal maintenance. *Depression and anxiety*, *31*(10), 805-813. doi: 10.1002/da.22271.

Stalnaker, T. A., Cooch, N. K., & Schoenbaum, G. (2015). What the orbital frontal cortex does not do. *Nature Neuroscience, 18(5)*, 620-627.

Stringaris, A., Vidal-Ribas Belil, P., Artiges, E., Lemaitre, H., Gollier-Briant, F., Wolke, S., ... & Fadai, T. (2015). The brain’s response to reward anticipation and depression in adolescence: Dimensionality, specificity, and longitudinal predictions in a community-based sample. *American Journal of Psychiatry*, *172*(12), 1215-1223. doi: 10.1176/appi.ajp.2015.14101298.

Toglia, M. P., & Battig, W. F. (1978). *Handbook of semantic word norms*. Lawrence Erlbaum.

Tomarken, A. J., & Zald, D. H. (2009). Conceptual, methodological, and empirical ambiguities in the linkage between anger and approach: comment on Carver and Harmon-Jones (2009).

Tomarken, A. J., & Keener, A. D. (1998). Frontal brain asymmetry and depression: A self-regulatory perspective. *Cognition & Emotion*, *12*(3), 387-420.

Trew, J. L. (2011). Exploring the roles of approach and avoidance in depression: an integrative model. *Clinical psychology review*, *31*(7), 1156-1168. doi:10.1016/j.cpr.2011.07.007.

Wager, T. D., & Nichols, T. E. (2003). Optimization of experimental design in fMRI: a general framework using a genetic algorithm. *Neuroimage*, *18*(2), 293-309. doi:10.1016/S1053-8119(02)00046-0.

Wang, J., Yang, Y., Fan, L., Xu, J., Li, C., Liu, Y., Fox, P. T., Eickhoff, S. B., Yu, C., & Jiang, T. (2015). Convergent functional architecture of the superior parietal lobule unraveled with multimodal neuroimaging approaches. *Human brain mapping*, *36(1)*, 238-257.

Watson, D., & Clark, L. A. (1993). Behavioral disinhibition versus constraint: A dispositional perspective.

Watson, D., Clark, L. A., & Carey, G. (1988). Positive and negative affectivity and their relation to anxiety and depressive disorders. *Journal of abnormal psychology*, *97*(3), 346. doi:10.1037/0021-843X.97.3.346.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of personality and social psychology*, *54*(6), 1063.

Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of abnormal psychology*, *104*(1), 3-14.

Whittle, S., Allen, N. B., Lubman, D. I., & Yücel, M. (2006). The neurobiological basis of temperament: towards a better understanding of psychopathology. *Neuroscience & Biobehavioral Reviews*, *30*(4), 511-525. doi: 10.1016/j.neubiorev.2005.09.003.

Winkler, A. M., Ridgway, G. R., Webster, M. A., Smith, S. M., & Nichols, T. E. (2014). Permutation inference for the general linear model. *Neuroimage,92*, 381-397. doi:10.1016/j.neuroimage.2014.01.060.

Wolpert, D. M., Goodbody, S. J., & Husain, M. (1998). Maintaining internal representations: the role of the human superior parietal lobe. *Nature Neuroscience*, *1(6),* 529-533.

Woolrich, M. W., Ripley, B. D., Brady, M., & Smith, S. M. (2001). Temporal autocorrelation in univariate linear modeling of FMRI data. *Neuroimage,14*(6), 1370-1386.

Zinbarg, R. E., & Yoon, K. L. (2008). RST and clinical disorders: Anxiety and depression. *The reinforcement sensitivity theory of personality*, 360-397.