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Positive Emotion and Impulsivity in Individuals with Bipolar I Disorder

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POSITIVE EMOTION AND IMPULSIVITY IN INDIVIDUALS WITH BIPOLAR I DISORDER

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Impulsivity as a symptom during mania is well documented, but less is known about the driving factors behind these impulsive acts. Perhaps this is because impulsivity is a term that encompasses multiple facets. There is evidence for a type of impulsivity in which extreme sensitivity to positive affective states can influence cognitive processes, such as error detection and identification of threatening cues. This can ultimately lead to engagement in impulsive actions during elevated mood states. Among people who are vulnerable to mania, positive moods have larger or more pervasive effects than they have on other people. While there is more conclusive evidence of impulsiveness among persons with BD (Bipolar Disorder) during mania, as compared to euthymia, it is less clear what specific mechanisms or mania symptoms perpetuate this effect. This study examined the possibility that positive emotion creates a greater processing bias among persons with BD than among other persons, such that neutral and negative stimuli are perceived less negatively. This study utilized multiple methods to measure the emotional response to threat--the motivational component with measurement of the affect-modulated eyeblink startle response, the arousal component with measurement of skin conductance levels, and the explicit appraisal component of emotional experience with self-report mood ratings. Twenty-two participants with BD and 25 control participants

viewed a series of valenced pictures (negative, positive, neutral), underwent a cognitive speed task to induce a positive mood, and then viewed another series of valenced pictures. Acoustic startle probes were paired with the pictures at 2500ms and 4500ms. Skin conductance tonic levels, eyeblink startle amplitude, and eyeblink latency times were measured continuously through the laboratory session. BD participants had lower SCL tonic levels to the negative stimuli after the mood induction, compared to control participants. There was no group difference in eyeblink amplitude or in eyeblink latency. Several limitations, including issues with study methodology, recruitment bias, and validity of the IAPS stimuli, were reviewed. Future directions for research and clinical implications were discussed.

Keywords: bipolar disorder, impulsivity, startle reflex, positive mood

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

Chapter 1: Introduction

Background

> When you're high it's tremendous. The ideas and feelings are fast and frequent like shooting stars, and you follow them until you find better and brighter ones…. But, somewhere, this changes…credit cards revoked, bounced checks to cover, explanations due at work, apologies to make, intermittent memories, friendships gone or drained, a ruined marriage. And always, when will it happen again?
>
> Kay Redfield Jamison, An Unquiet Mind, p g. 67-68

The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR) reports that Bipolar I disorder (BD I) has a 1.3 to 1.5 percent prevalence rate (American Psychiatric Association, 2000). This disorder has been associated with financial and social impairments (Michalak et al., 2005), the highest rates for comorbidity of alcohol or substance abuse for any Axis I disorder (Chen & Dilsaver, 1996; McIntyre & Keck, 2006), and high rates of episode relapse and hospitalizations (Fawcett, 2008). The DSM-IV-TR identified three major subtypes of BD: bipolar I disorder, bipolar II disorder, and cyclothymia (American Psychiatric Association, 2000). This study will focus on BD I, since severe dysfunctions in mania occur only within this subtype.

BD I is diagnosed on the basis of a single manic or mixed episode, that requires three (with elevated mood) or four (with irritable mood) of the following symptoms: increased self-confidence, decreased need for sleep, pressured speech, flight of ideas and/or racing

thoughts, an increase in goal-directed activity or psychomotor agitation, and increased involvement in activities which have potential for high risk or negative consequences (APA, 2000). One common symptom of mania is engagement in high-risk and impulsive acts. The DSM-IV-TR (2000) lists one of the defining symptom of mania as "excessive involvement in pleasurable activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments)." For example, one study indicated that manic episodes involving high-risk sexual behavior are a risk factor for contracting HIV (Meade, Graff, Griffin, & Weiss, 2008). Often the most disastrous consequences of a manic episode are the job losses and interpersonal disruptions stemming from the episode's characteristically high risk and impulsive acts (Dougherty et al., 2004; Swann et al. 2004; Swann et al. 2005).

Impulsivity as a manifestation of BD I is well documented, but less is known about the driving factors behind these impulsive acts. In this section, I will briefly discuss several conceptualizations of impulsivity before focusing on a type of impulsivity that occurs in response to emotions. After reviewing literature bearing on the role of impulsiveness in mania risk, and evidence of impulsiveness in persons with BD who are both euthymic and manic, I will interpret that evidence in terms of impulsive responses to emotion. Last, I will provide a rationale for investigating positive mood and threat detection in BD in this study.

Various Constructs of Impulsivity

Impulsivity is a broad concept, with different hypothesized causes and different manifestations. Impulsivity is associated with several disorders including externalizing disorders, substance use, borderline personality disorder, and mania. Whereas impulsivity

has been characterized as a symptom in various disorders, the exact nature of impulsivity is less well understood. More specifically, impulsivity is a term that encompasses multiple facets. Some have defined impulsivity as "an inability to delay rewards or an inability to withhold a response" (Moeller et al., 2001, pg. 1783-1784). Others have defined impulsivity as "a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others" (Dougherty et al., 2009 pg. 64) or "responding quickly without adequate assessment of the context or consequences" (Swann et al., 2005). While research has been conducted to examine factors that may encompass impulsivity, less has been done to examine the processes that may drive impulsive behavior. Some mechanisms could be purely cognitive, such as "the inability to maintain attention to complete a particular task rather than becoming distracted to an alternative task" (Strakowski et al., 2010, pg. 286) or the tendency to engage in behaviors without thought to future consequences (Damasio et al., 1990). Some other research suggests that affect may be a factor in engagement in impulsive actions (Cyders & Smith, 2007; Whiteside & Lynam, 2003). In the following section, I will review evidence for a type of impulsivity in which extreme sensitivity to affective states can influence cognitive processes such as error detection and identification of threatening cues. This can ultimately lead to engagement in impulsive actions during elevated mood states.

Impulsivity in Response to Emotions

Intense negative and positive mood may both be related to high levels of impulsivity, yet the mechanisms by which these kinds of affect influence impulsive behaviors may differ. In this section, I will first review how negative affect can influence

reactivity to stimuli and engagement in impulsive behaviors. Then I will turn to effects of positive affect on impulsivity.

Negative emotion-driven impulsivity. People vary in how behaviorally responsive they are to intense emotions. Broadly described, emotions are brief, discrete states, and often are coordinated responses that involve subjective, physiological, facially expressive and behavioral components (Humrichouse et al., 2007). Moods are defined as longer in duration, and generally fluctuate less than emotions. Emotions and mood interact in a reciprocal manner in which emotions and lead to certain moods, and certain moods are more likely to trigger specific emotions (Davidson, 1994).

Experiencing intense negative affect may motivate individuals to engage in rash behaviors during this affective state (Davidson, 2003). It may be that the needed cognitive effort to regulate one's negative mood state may induce less rational (or less thorough) processing of the environment, which may facilitate acting in a rash manner. The Urgency subscale from the UPPS (Whiteside & Lynam, 2001) was developed to identify individual differences in the tendency to engage in rash actions or behaviors with negative consequences in the face of intense negative emotions. The development of the UPPS was an attempt by Whiteside and Lynam to consolidate the previous literature by identifying and separating distinct personality facets that have been previously lumped together under the term, *impulsivity*. The individual with high urgency reacts quickly, spontaneously, and rashly in an intense negative mood, even in the face of negative consequences (e.g., ''When I am upset I often act without thinking"). Behaviors such as impulsive aggression (Whiteside et al., 2005), heavy alcohol consumption (Cyders & Smith, 2008), and binge eating (Settles et al., in press) are related to negative urgency.

One study explored whether a negative mood induction can lead to an increase in risky decision-making. Leith and Baumeister (1996) had 33 participants run in place for 3 minutes (to induce high physiological arousal). Then participants rated their mood from -10 (lowest bad mood) to 10 (highest positive mood). Afterwards, participants were offered a choice between Choice A (70% chance of winning $2--high chance of winning, but low reward) or Choice B (2% chance of winning $25—low chance, high reward). Losing in either choice entailed listening to a 3-minute tape of fingernails scratching across a blackboard. Participants who reported high arousal and negative emotions were the most likely to choose the riskier option (Choice B). Arousal alone was not the explanation, however, because people who were aroused without feeling bad at the same time did not display this pattern. Thus, high arousal plus negative affective valence may induce less rational processing and cause an individual to engage in a bigger risk than he or she intended.

In sum, research suggests that some individuals react to negative emotions in a rash manner, even at the cost of longer-term goals. Evidence on how negative emotions induce impulsivity suggests that negative urgency is a desire to engage in rash actions in order to reduce the negative emotions, while disregarding other contingencies. Other findings indicate that negative emotions are most likely to increase risky decision-making if they are combined with high arousal.

Positive emotion-driven impulsivity. Parallel to negative urgency, there is also evidence that positive emotions may increase engagement in rash actions, or greater risk-taking. It may be that positive emotions may lead to biased risk-taking, which may result in negative outcomes or consequences. One way of measuring this possible risk factor, in

which one engages in a rash manner, is with a self-report questionnaire, the Positive Urgency Measure (PUM; Cyders & Smith, 2007). Cyders et al. (2007) developed this scale to capture the tendency to react rashly when experiencing positive mood (e.g. "When I get really happy about something, I tend to do things that can have bad consequences" and "When I am very happy, I feel like it is OK to give in to cravings or overindulge"). The basis for developing this measure was a variety of empirical evidence of tendencies toward rash action on the part of some people in response to positive emotions. For example, positive mood has been identified as a risk factor to relapse of a gambling episode among individuals meeting criteria for gambling disorder (Holub, Hodgins, & Peden, 2005).

One study by Cyders et al. (2007) investigated positive urgency as a risk factor among people with drinking problems. Cyders et al. (2007) assessed the motive to drink in order to enhance an existing positive mood, as measured by the Drinking Motives questionnaire (DMQ; Cooper, 1994). The DMQ is a 20-item scale that reflects four motivations for drinking: coping motives (drinking to cope or deal with negative affect), enhancement motives (drinking to enhance positive affect), social motives (drinking to increase socialization), and conformity motives (drinking to fit in with a group). Alcohol consumption was assessed in this study by the Drinking Styles Questionnaire (DSQ; Smith, McCarthy, & Goldman, 1995), which includes quantity and frequency of consumption. Cyders et al. (2007) found that positive urgency was related to binge drinking for those who drink to enhance a positive mood (ß = .43, p <.001), but not for those who do not drink to enhance a positive mood (ß = .16, *ns*).

In a year-long longitudinal study (Zapolski et al., 2009) with 407 college-aged participants, positive urgency predicted engagement in high-risk activities, such as unprotected sex and substance use, while controlling for baseline engagement of behaviors at the beginning of the study and for other factors such as sensation seeking, negative urgency, lack of planning and lack of perseverance. Thus, this study highlighted that high positive urgency predicted willingness to engage in rash behaviors without thought to consequences when in a good mood.

In a 2010 study by Cyders et al., 104 college-age participants completed the PUM, then underwent two positive mood induction procedures in order to induce a positive mood, and then completed a laboratory procedure on risk-taking. First, participants were asked to imagine and be fully involved in the emotional experience while listening to an audiotaped recording of an individual recounting positive events that had occurred. Then participants were asked to re-imagine a personal past experience that induced an extremely positive mood, and were asked to write down their past experience. Then, participants completed the Balloon Analogue Risk Task (BART; Lejuez et al., 2002), a laboratory measure of risk taking. In this laboratory task, participants inflate a computer simulated balloon, and can choose how many times to pump up the balloon, with the participant earning money for each pump. However, if the pump is inflated too many times, it will burst and the participant loses the money earned from that balloon. Thus, participants can choose to stop pumping the balloon and take the money earned up to that point, or continue to attempt to earn more money. Performance on the BART task has been correlated with engagement in substance use, gambling disorders, impulse control disorders, and unprotected sex (for review, please see Hunt et al., 2005). In the

2010 study by Cyders et al., participants with higher PUM scores had a greater tendency to engage in risk-taking behavior on the BART, as compared to participants with lower PUM scores. Thus, this was the first study address the issue of self-reporting bias, and was able to experimentally induce a positive mood in participants, and investigate with laboratory tasks that those with higher PUM scores were more likely to engage in riskier decision making during a positive mood.

According to appraisal theories of emotion (Lerner & Keltner, 2000; Lerner & Keltner, 2001), an existing positive emotion can lead people to interpret a neutral situation as more positive than it would otherwise seem. In an early study, undergraduates were given false performance feedback to induce either a positive or negative mood. Then they were asked to rate the valence of slides. The authors found that experimentally induced positive moods led individuals to interpret neutral pictures as more positive (Isen & Shalker, 1982). Thus, in ambiguous situations, a positive mood could lead an individual to interpret a neutral situation as more positive than it is. This bias could then apply more generally to assessing the environment and responding appropriately.

Is this biasing effect of positive mood restricted to neutral stimuli? Perhaps not. There is also evidence that in risky or threatening situations, a positive mood can lead to underestimation of cues of threat. In another early study by Johnson and Tversky (1983), healthy participants first read newspaper articles about death rates, and then experienced a positive mood induction. Those who had a positive mood induction recalled lower death rates than the actual death rate in the story they had read. Thus, in healthy

participants, a positive mood predicts a biasing effect on the perception of mood-incongruent stimuli.

How can a positive mood influence impulsiveness? To the extent that rash action is inhibited by perception of threat in the environment, the failure to perceive threat accurately could result in a failure to inhibit action appropriately. Some studies suggest that being in a positive mood can contribute to reacting in rash ways in two ways. First, positive mood may increase engagement in rash behaviors without thought to negative consequences. Second, during a positive mood, misinterpreting situations as more positive than they are may prevent individuals from accurately evaluating the risks or consequences of a situation. Perceiving less risk of adverse consequences, they are less likely to restrain themselves from acting. Both of these mechanisms can lead to impulsive actions when in a positive mood.

Summary of emotion-based impulsivity. In sum, a negative mood can induce rash behaviors in an effort to decrease distress; a positive mood can induce rash behaviors in an effort to sustain the mood. A positive mood can also yield positive interpretations of neutral stimuli and contribute to underestimation of cues of threat. With this information as background, I will next turn to positive mood in BD.

Emotion Processing in BD and Neurobiological Systems

There is a large body of evidence suggesting that the amygdala and the ventral lateral prefrontal cortex are involved in emotional functioning (Adolphs, 2010; Davis & Whalen, 2001; Davidson, 2001; Scheider et al., 2012; Strakowski et al., 2012; Townsend & Altshuler, 2012). The amygdala plays a key role in emotion perception and signaling the emotional salience of stimuli to other areas of the brain (Strakowski et al., 2012).

Specifically, the amygdala plays a role in the projections to the brainstem or the hypothalamus to stimulate the autonomic nervous system or the hypothalamic pituitary adrenal axis (Adolphs, 2010; Davis & Whalen, 2001; Davidson, 2001). The ventral lateral prefrontal (VLPFC) cortex integrates emotional information from external stimuli, regulates the intensity of internal emotional reactions, and plays a role in inhibitory actions (Scheider et al., 2012; Strakowski et al., 2012; Townsend & Altshuler, 2012). These two parts of the brain then work together in an iterative feedback loop. Underactivation of the VLPFC can then lead to lack of inhibitory modulation of the amygdala (Strakowski et al., 2012). Broadly, findings from fMRI studies with manic BD participants suggest left amygdala over-activation, and decreased activation in the VLPFC (Strakowski et al., 2012). Concurrent irregular functioning of these two brain areas has been suggested as a mechanism that would produce the emotional dysregulation in BD (Blond et al., 2012; Strakowski et al., 2012). There have been mixed findings in fMRI studies during BD depression states with some findings indicating an underactivation of the frontal lobe (Malhi et al., 2004), while others suggesting no significant differences between depressed and euthymic patients during behavioral tasks designed to elicit emotional processing. Findings from studies with euthymic BD participants have indicated no consistent difference in amygdala functioning between euthymic BD participants and controls. However, some findings suggest decreased activation in the VLPFC (Townsend & Altshuler, 2012) during times of euthymia. In sum, there is more conclusive evidence of the amygdala and the VLPFC's roles during times of mania, while findings are more mixed for times during BD depression and euthymia.

Positive Affect and Mania: Theoretical Considerations

Intense positive mood, such as euphoria, is one cardinal symptom of mania (APA, 2000). This suggests that the mechanisms just discussed may be relevant to mania. One theory on a relationship between risk for mania and positive mood is based upon the Behavioral Activation Model (Johnson et al., 2000; Johnson et al., 2012; Urošević et al., 2008; Urošević et al., 2010). This model appears to be consistent with both mechanisms just discussed by which positive feelings may lead to rash behavior. This model rests on broader statements about motivation.

Several theorists have argued that two general motivational systems underlie behavior, the behavioral inhibition system (BIS) and the behavioral activation system (BAS) (Depue & Iacono, 1989). The BAS is believed to regulate appetitive motives. It is a neurobiological system that influences one to behaviorally approach reward-related goals. At the neurobiological level, BAS involves dopaminergic (DA) projections to the frontal cortex, amygdala, nucleus accumbens, ventral pallidum, septum, and hippocampus (Davidson, 1994). According to the BAS dysregulation model of bipolar disorder, persons vulnerable to BD I have an overly sensitive BAS (Johnson et al., 2012). BAS sensitivity can influence the speed of learning, when rewarding stimuli are present, and can influence an individual's ability to shift learned responses (Johnson et al., 2012). It is theorized that an initial success can lead to increased confidence and amplified willingness to engage in goals in people with an overreactive BAS (Johnson, 2005).

Indeed, in a 2008 study by Harmon-Jones et al., 41 participants at-risk for BD (those with a cutoff off score greater than 13 on the General Behavior Inventory-Hypomania Biphasic Scale (GBI;Depue et al., 1989)) or BD II, and 53 control

participants were measured by electroencephalography while asked to solve anagrams with varying levels of difficulty. Participants in this study could either earn money if they solved the anagram or lose money if they did not solve the anagram. BD spectrum participants were found to have greater activation in the left frontal cortex during the reward trials, but not the medium or low reward trials, nor for the punishment trials. That is, those at-risk or BD spectrum participants were more engaged and willing to sustain more effort for the more difficult rewards, compared to controls.

Consistent with this view, behavioral approach sensitivity, as measured by the Behavioral Activation Scales (Carver & White, 1994), is related to mania in BD I in both cross-sectional and prospective studies (Alloy, Bender, et al., 2011; Alloy, Urosevic, et al., 2011; Johnson et al., 2000; Meyer et al., 2001). In a longitudinal study (Alloy, Bender, et al., 2011), participants with a high total BAS score (cutpoint $\geq$ 43) exhibited a shorter time to onset of bipolar spectrum disorders over a 12.8 month period of time, than those with a moderate BAS score (37-39).

One specific BAS subscale is more closely linked to impulsivity than the others, the Fun-Seeking subscale. The Fun Seeking subscale captures the tendency to seek out novel experiences with the expectation that these experiences will increase positive emotion. The subscale has been correlated with extraversion (Eysenck & Eysenck, 1985), low harm avoidance, and novelty seeking (Carver & White, 1994). The Fun-Seeking subscale of the BIS/BAS Scale was correlated with a lifetime bipolar spectrum disorder in two longitudinal studies (Alloy et al., 2006; Jones & Day, 2008), and predicted a greater likelihood of conversion from Bipolar II to Bipolar I disorder (Alloy, Urosevic, et al., 2011).

Impulsivity Findings in BD I

What is the broader scope of evidence concerning impulsivity and BD? Much of the research in this area has utilized self-report scales and tasks that focus on facets of impulsivity found in other psychiatric disorders, and do not take into account the role positive emotion plays in BD. I will first discuss the relationship between risk for BD and impulsivity. Then I will briefly review findings from laboratory tasks in euthymic participants.

Prospective relationship of impulsivity and BD I. One study has reported that elevated impulsivity scores help predict a BD I diagnosis. This was a 13-year longitudinal study examining the Hypomanic Personality Scale (HPS; Eckblad & Chapman, 1986), along with self-reports of impulsivity. The latter was assessed by the Impulsive-Nonconformity Scale, which assesses difficulty in delaying (e.g., "If I want something, delays are unbearable"; Chapman et al., 1984). The two scales together were better predictors of onset of bipolar disorder than high HPS scores without elevated impulsivity scores (Kwapil et al., 2000). In a 4.5 year longitudinal study following college-aged participants at-risk for BD, higher scores on the Impulsive-Nonconformity Scale predicted progression to Bipolar I disorder (Alloy, Urosevic, et al., 2011). Taken together, these two findings suggest that impulsivity occurs early in the disorder and is related to the disorder, rather than being a consequence of the illness.

Impulsivity during euthymia. Studies have reported mixed findings regarding impulsivity in euthymic participants with BD. Some studies investigating impulsivity using self-reported questionnaires in cross-sectional studies with clinical populations have reported euthymic participants scoring higher than healthy controls (Peluso et al.,

2006; Swann, Anderson, Dougherty, and Moeller, 2001; Swann, Lijffijt, Lane, Steinberg, and Moeller, 2009) while other studies have not (Christodoulou et al., 2006).

Impulsivity findings using laboratory tasks during euthymia. Laboratory paradigms attempting to test underlying cognitive mechanisms and behavioral actions have reported a similar pattern of mixed findings in BD in adolescents and adults (Clark, Iversen, and Goodwin, 2002; Leibenluft et al., 2007).

Behavioral inhibition. The Go/No-Go task (Donders, 1969) is designed to measure the ability to inhibit a motor response. Participants must learn when to behaviorally respond (Go) to cues (letters or pictures) that have been paired with rewards, and learn to withhold a response (No Go) to cues that have been paired with punishment or non-reward. That is, words appear on a computer screen, and subjects are initially instructed to respond for certain letters or pictures (Go), but not to other letters or pictures (No Go). After blocks requiring responses to (Go) words, the instructions are reversed so that the participant is now instructed to respond to the previously ignored words and ignore the previously rewarded words.

In a study investigating behavioral inhibition and BD, 27 euthymic BD I and 25 control participants completed the Go/No-Go task while undergoing fMRI scanning (Kaladjian et al., 2009). Despite no group differences on performance on the Go/No-Go, fMRI findings suggested that BD participants exhibited less activation in the left frontopolar cortex and less activation in the amygdala, as compared to control participants. Another study had 32 euthymic and 30 control participants complete the Go/No-Go task while undergoing fMRI scanning. Findings from this study also indicated no group differences on accuracy and reaction times on the Go/No-Go task. While the

inferior frontal cortex (IFC), which is involved in the modulation or inhibition of behaviors, was activated in both groups, it was less activated in the BD group, as compared to the control group (Townsend, Bookheimer, et al., 2012). Taken together, findings from these studies may suggest that while there are no behavioral differences between BD and control participants, there seems to be a neurological difference of an underactivation of areas related to inhibition or modulation of actions, even during times of euthymia for BD participants.

Reward responsivity and decision-making. Riskiness in decision-making can be thought of as a kind of impulsiveness, but one that is more complex than others. The Iowa Gambling Task (IGT; Bechara, Damasio, Tranel & Anderson, 1994) measures how responsive one is to immediate rewards relative to punishments. This task is designed to distinguish people who are so attuned to the size of the possible reward that they disregard possible negative consequences of choosing it. The IGT task involves four decks of cards called "A" "B" "C" and "D". Choosing an "A" or "B" card is followed by an immediate large gain of money, but also a potentially larger loss of money. Choosing a "C" or "D" card gives a small gain, without the same risk of loss. Choosing an A or B card thus represents a risky or rash action.

A recent 2011 study by Martino et al. asked 85 BD (48 euthymic BD I, 37 BD II), and 34 control participants to complete the IGT. There were no group differences between the euthymic BD participants and controls on the IGT. However, one interesting finding was that BD participants with a history of suicide attempt selected more risky cards, as compared to BD participants without a history of suicide. Another 2011 study by Mallory-Diniz et al. asked 95 euthymic and 94 control participants to complete the

IGT and also geneotyped the participants for the 5-HTTLPR. The short form of the 5-HTTLPR and suicidal behavior has been previously established (Neves et al., 2008). BD participants had worse performance on later blocks of the IGT, as compared to control participants. Similarly, BD participants with a history of suicide attempted performed more poorly, as compared to BD participants without a history of suicide and also compared to control participants. However, there was no reported difference in the genotype between BD and control participants, or between BD participants with or without a history of suicide. While there are mixed findings in euthymia in which some studies showed elevated risk taking in BD participants (Malloy-Diniz et al., 2011), other studies did not (Clark et al., 2002; Martino et al., 2011; Yechiam et al., 2008). One interesting finding was the relationship between a history of suicide attempts and decision-making on the IGT. However, the IGT may not offer a conclusive picture of impulsivity and BD.

Impulsivity during mania. Findings with the same laboratory tasks described above with manic BD participants indicate more consistent results. In this section, I will review relevant studies that examined people with BD in manic or hypomanic states.

Impulsivity findings using self-report questionnaires during mania. Researchers have used self-report questionnaires such as the Barratt Impulsiveness Scale (BIS-11; Barratt and Patton, 1983) to measure deficits in attention, motor-control and planning. Some studies have found that elevated scores are related to current mania, when compared to controls (Peluso et al., 2007). Other studies have found no differences in euthymic (n = 22) and manic bipolar (n = 12), compared to controls (Swann et al., 2003). However, the latter authors note that in this study, 3 out of the 12 manic participants were

in mixed states and many of the euthymic participants endorsed subsyndromal depressive symptoms. Thus, it is unclear if mood symptoms were related to the lack of difference between the manic and euthymic group. In another study, Swann et al. (2008) correlated the BIS-11 subscales with positive and negative affective symptoms of participants. The Motor Impulsiveness subscale related to mania symptoms (Swann et al., 2008). The Non-planning and Attentional Control subscales related only to depression symptoms.

Impulsivity findings using laboratory tasks during mania. Findings from behavioral lab tasks among persons with BD I during mania suggest that there are unique cognitive and emotional differences in mania compared to euthymia. In this section, I describe laboratory findings bearing on the ability to inhibit behaviors, the ability to detect negative facial expressions, and reward responsivity.

Behavioral inhibition. The affective Go/No-Go task yields the same measure of inhibition as the Go/No-Go task, but the substitution of affective stimuli for the neutral stimuli also permits analysis of performance in response to cues of different emotional valences (e.g., happy versus sad). The affective Go/No-Go task comprises eight test blocks of 18 stimuli each (nine positively-valenced words and nine negatively-valenced words). The task requires subjects to attend and respond to relevant targets while inhibiting responses to stimuli of the competing affective category. As in the basic task, the responses shift from one block to another, so that the person must respond not to happy but to sad targets, and withhold not from sad but from happy targets. Thus, the task not only provides a measure of behavioral inhibition, but also indicates the modulation of this inhibition by the emotional content of the stimuli (Drevets & Raichle, 1998).

Murphy et al. (1999) investigated motor inhibition in 18 manic participants, 40 healthy controls, and 28 depressed patients using this task. Manic patients were impaired in their ability to inhibit inappropriate responses overall. Both patient groups exhibited attentional biases only for emotional stimuli congruent with their current mood. That is, depressed participants were faster to respond to sad targets, but not happy ones, compared to manic and healthy control participants. The manic participants were quicker to respond to the happy targets, but not the sad ones, as compared to depressed and healthy control participants.

One limitation of the reviewed studies has been the cross-sectional, between-subjects design. A recent study by Strakowski et al. (2010) used a within-subjects design, measuring 108 bipolar I manic or mixed and 48 control participants on three tasks: a stop signal task, a delayed reward task, and a continuous performance task. The BD participants were then followed for up to one year; if they developed either depression or euthymia, they were reassessed with the same measures. The control participants were also assessed with the same instruments three and six months after the initial assessment. At initial testing, the manic participants exhibited difficulty inhibiting a response on the stop signal task, made more impulsive responses on the delayed reward task, and had lower sensitivity on the continuous performance task, as compared to control participants. After recovering from mania, bipolar participants exhibited no significant differences from healthy subjects on any behavioral task. However, it should be noted that self-ratings scores of impulsivity from the BIS-11 remained elevated, and so one possibility is that Strakowski and colleagues may not have measured critical aspects of impulsivity with the three behavioral tasks used, such as emotion-based impulsivity.

Detection of negative facial expressions. If persons with BD are attuned to positive stimuli, does this mean that they are less sensitive to the detection of negative stimuli? There is some suggestion that mania decreases sensitivity to facial expressions. Poorer recognition of emotional facial expressions suggests a deficit in the detection of social cues and the associated social information that assist in decision-making and information processing (Cole, Teti, & Zahn-Waxler, 2003). There have been mixed findings for detection of negative facial expressions in studies with euthymic participants (Venn et al., 2004; Yurgelun-Todd et al., 2000), while studies with manic participants suggest decreased identification of negative expressions compared with positive expressions.

In a key study, Lembke and Ketter (2002) asked adult manic BD I and healthy control participants to match affective words to facial expressions. Manic participants had difficulty recognizing all four expressions of negative emotion (e.g. fear, disgust, anger and sadness) compared to healthy control participants (Lembke & Ketter, 2002), though they had no trouble identifying happy expressions. One interpretation of these findings is that persons who are manic experience difficulties in the recognition of some forms of threatening cues.

Reward responsivity and decision-making. Findings of BD manic participants and risk taking during the IGT have been more conclusive. In one study (Yechiam et al., 2008) twenty-eight bipolar patients (14 manic and 14 remitted) and 25 controls were tested using the IGT with 60 cards in each trial block. Manic participants made more risky choices over the course of 5 trial blocks compared to the euthymic BD and control participants. However, other studies have indicated both state and trait based responding

on the IGT. In another study (Adida et al. 2011), 45 manic, 32 bipolar depressed, 90 euthymic BD participants and 150 age, IQ, and gender matched control participants completed the IGT. The manic, depressed and euthymic BD participants chose cards from the risky deck more frequently than control participants. Post-hoc pairwise comparisons revealed no significant difference in risk-taking between manic and depressed, manic and euthymic, or depressed and euthymic participants.

Elevated mood episodes in BD can be comprised of either manic or mixed episodes. A mixed episode meets both the DSM -IV criteria for a manic episode and the criteria for a major depressive episode (MDE; with a duration of one week) (APA, 2000). While both these types of elevated mood episodes are prevalent within BD, comparing manic versus mixed states may allow researchers to pinpoint whether the unique aspect of intense positive emotion found in mania is related to impulsivity.

In a recent study by Strakowski et al. (2009), 50 manic, 20 mixed, and 34 control participants completed a battery of laboratory tasks. Only manic, and not mixed BD I patients, were significantly more likely to make high-risk choices, as compared to healthy subjects. Strakowski et al. noted that task performance was not significantly correlated with the severity of symptoms. That is, the authors suggested that the difference between a manic state, compared to a mixed state, was related to risky decision-making, but they could not relate this difference on IGT performance to any specific symptom.

The inconclusive and mixed findings may point to the overall issue of construct validity. More specifically, mixed findings may suggest that the various self-report measures and laboratory tasks may be measuring different constructs of impulsivity. In attempting to address this issue, Cyders and Coskunpinar (2012) investigated if there was

a relationship among self-report and behavioral lab task measures. Specifically, they explored how the various constructs of impulsivity (i.e., negative urgency, lack of planning, lack of perseverance, sensation seeking, and positive urgency) from self-report measures were related to laboratory paradigms (i.e. IMT/DMT, GoStop [Dougherty et al., 2005], two choice [TCIP; Dougherty et al., 2005], single key impulsivity paradigm [SKIP; Dougherty et al., 2005], the Brown-Peterson task [BPT; Kane & Engle, 2000], and the TIME paradigm [Dougherty et al., 2005]). Researchers found small to medium effect sizes for some of the relationships between constructs of impulsivity and laboratory tasks, and generally suggest that the self-report measures and lab tasks may capture different aspects of impulsivity. In summary, impulsivity findings from laboratory tasks with BD participants indicated elevated impulsive findings in mania as compared to euthymia.

While there is more conclusive evidence of impulsiveness among persons with BD during mania, as compared to euthymia, it is less clear what specific mechanisms or manic symptoms perpetuate this effect, and if researchers are studying the same construct of impulsivity. Indeed, the recent review by Cyders and Coskunpinar (2012) suggest that self-report constructs of impulsivity are not at all correlated to behavioral laboratory tasks. There is also no clear agreement on which measurements to capture mania severity. Thus, it remains unclear whether it is positive mood or other manic symptoms that are the drivers of these findings.

Mechanisms of Positive Mood in BD I

Reviewed in the preceding section was evidence of behavioral impulsivity increasing in mania, as compared to euthymia, within BD. Also described earlier was

evidence that positive moods induce a bias in processing among people in general. A plausible line of argument is that among people who are vulnerable to mania, positive moods have larger or more pervasive effects than they have on other people. There is some evidence that fits that view.

In this section, I will review evidence of the role positive mood plays in impulsivity in BD. Parallel to the process of negative rumination, the repetitive focus on content, antecedents and consequences of one's affective state that can contribute to the onset and maintenance of a depressive episode (Nolen-Hoeksema & Morrow, 1994), researchers posit that positive rumination, the active processing during a positive mood state, may be related to hypomanic symptoms and mood episodes in BD (Feldman et al., 2008). One study reported that individuals at-risk for BD were more likely than healthy controls to engage in cognitive strategies to sustain their positive mood (Feldman et al., 2008). In a 2008 study by Johnson et al., 28 individuals diagnosed with BD, 35 individuals diagnosed with major depressive disorder (MDD), and 44 control participants were asked to complete self-report measures on rumination styles of negative emotions with the Ruminative Response Scale (Nolen-Hoeksema & Morrow, 1991) and of positive emotions with the Response to Positive Affect Questionnaire (Feldman et al., 2008). While participants with MDD and BD both endorsed rumination of negative emotion, as compared to controls, only the BD group endorsed rumination of positive emotions (Johnson et al., 2008).

As well as cognitive strategies to sustain their positive emotions, participants with BD may have difficulties in down-regulating their emotions. Emerging studies using laboratory paradigms have shown continued engagement in emotional responding in

individuals with BD. In a 2011 study by Gruber, Eidelman, et al., participants with BD self-reported engaging in rumination strategies for both negative and positive emotions, compared to control participants. Indeed, participants with BD reported engaging in more effort to regulate their emotions, but being less successful than their control participants when viewing negative, positive, and neutral emotionally eliciting film clips (Gruber et al., 2012). Finding from these studies suggest an enduring pattern of heightened positive emotionality in BD (Gruber et al., 2008; Gruber, Harvey, & Purcell, 2011; Gruber, 2012).

In euthymic persons with BD I, positive mood increases risky decision-making (Johnson, Ruggero, & Carver, 2003; Murphy, Rubinstein, Rogers, Robbins, Paykel, & Sahakian, 2001), dysfunctional assumptions about risk-taking (Lam, Wright, & Smith, 2004), and elevated goal expectations (Ruggero & Johnson, 2006) to a greater extent than occur in other persons. A study by Roiser et al. (2009) investigated the effects of positive mood on signal detection and decision-making. BD I and control participants first underwent a positive mood induction (false positive feedback) and then completed the Affective Go/No-go task. Following mood induction, BD participants had a higher rate of incorrectly responding to happy words during sad word target blocks, compared to controls. Perhaps then, persons with BD I have more difficulty processing cues to withhold action when in a positive mood.

Reactivity to positive mood. There is additional evidence of elevated reactivity to positive mood, both in euthymic BD I participants and in those at risk for mania. In two daily diary studies, those at-risk for BD reported greater trait positive affect (Hofmann & Meyer, 2006), and also greater variability within the day between negative

and positive affect to daily life events, as compared to those at risk for unipolar depression and control participants (Lovejoy & Steuerwald, 1995). Findings from these two early studies exploring daily affect variability and risk for BD suggest that one risk marker for BD may be the greater experience of positive affect, and also mood fluctuations throughout the day to daily life events. Recent findings on positive emotion and BD suggest sustained positive emotions for individuals with BD. In one study, BD participants after a positive mood induction study were reported to have a continued elevated positive mood, while control participants had returned to baseline levels at the study end (Famer et al., 2006). Thus, the positive mood induction procedure can be applied as a mild simulation of the hypomanic mood and may be able to reveal the shifts in decision-making in response to the emotion that are some of the cognitive shifts occur in day to day life outside of episode. Therefore, examining milder mood shifts is important.

Beyond diary studies, self-report measures capturing the willingness to engage in rash behaviors during a positive mood have also been related to BD. In a recent study by Johnson et al. (under review), 79 euthymic BD I and 80 healthy controls completed a battery of self–report questionnaires on impulsivity. Of the measures of impulsivity, the PUM was more correlated with BD I; other commonly utilized measures, such as the BIS-11, were not as strongly correlated. The PUM specifies positive emotion as an antecedent for impulsive behavior (Cyders et al., 2007), and as such, this finding thus may suggest that over-reactivity to positive emotion plays a key role in the mania risk for BD I.

An analog study by Johnson and colleagues (2007) allowed for confirmatory and exploratory modeling of the latent variable, impulsivity, to provide statistical evidence of positive mood relating to impulsivity within BD. Over 2,400 University students were screened with the Hypomanic Personality Scale (HPS; Eckblad & Chapman, 1986) and the Inventory to Diagnose Depression—Lifetime Version (IDD–L; Zimmerman & Coryell, 1987). Ninety-three students who met the established cut-off for the HPS completed a battery of questionnaires. Two factors emerged from preliminary factor analyses of these scales. One factor was composed of the BAS Fun-Seeking subscale (Carver & White, 1994) and the PUM (Cyders et al., 2007). It was a significant predictor of tendencies toward mania and alcohol abuse. The second factor was characterized by measures of aggression (physical, verbal) and anger from the Buss-Perry Aggression Questionnaire (Buss & Perry, 1992). This factor did not relate to tendencies toward mania. Thus, this study provides some support for a dimension of impulsivity underlying self-report inventories on positive affect. Broadly, the results of this study provide support for the importance of considering impulsivity within the context of positive and negative emotions.

In sum, some previous findings suggest a pervasive experience of positive affect in daily life for those at-risk for BD, and a key role of positive mood in impulsive actions among persons with BD. Perhaps the effects occur because the positive mood creates a processing bias that screens out threats. Another possibility is that the effects occur because the positive mood creates a stronger desire to sustain the positive mood. However, many of the relevant findings are ambiguous about the mechanisms. More work is needed to test these potential mechanisms and how they relate to BD.

This study explored the latter possibility by examining whether positive emotion creates a greater processing bias among persons with BD than among other persons, such that neutral stimuli and negative stimuli are perceived less negatively and positive stimuli are perceived more positively.

Measures of Emotional States

Emotional reactions are not a singular event, but a multi-component process made up of feelings, facial expressions, appraisals, and physiological reactions (Mauss et al., 2005). Thus, there are several methods to measure emotional experiences. One method would be to examine the biological mechanisms of emotional functioning; another is to examine measures of psychological reactions (e.g., self-report of emotion states). Emotional states are often viewed as having at least two components, valence and activation (Kring, 1999; Larsen & Diener, 1992). Valence refers to the pleasant/unpleasant or positive/negative hedonic property of the emotion. Activation refers to the overall intensity or arousal level of a response (high/low), but in itself lacks directionality (Patrick et al., 1993). On the other hand, when a valence is specified, activation generally means the emotion is more intense. Two measures, the skin conductance level (SCL) and the acoustic startle response, have been historically used to capture the arousal and valence dimensions, respectively, of the emotional response. In the following section, I will review SCL and the acoustic startle response as indices of emotional responding.

Skin conductance level (SCL). One measure of sympathetic activation is the SCL, in which sweat glands are activated and moisture is secreted onto the surface skin. There are multiple levels of electrodermal responding, but SCL reactivity to stimuli have

been related to the ventromedial prefrontal cortex and the limbic system in the brain (Sequeira & Roy, 1993). Emotional reactivity has been measured by two main parameters, skin conductance response (SCR), and SCL tonic level. SCL has been historically used as a measure of electrodermal activity of longer-lasting stimuli such as attending or engaging in a task. Tonic level is the mean value computed across two points of time. Higher tonic levels are taken as an indicator of greater emotional intensity, while reduced tonic levels suggests diminished emotional reaction (Elliott, 1992). SCL acts as a nonspecific measure of sympathetic activation and captures overall arousal, but does not distinguish between positive and negative valence of the emotion (Lang, Greenwald, Bradley, & Hamm, 1993; Witvliet & Vrana, 1995).

The acoustic startle response. A psychophysiological measure related to the hedonic properties of emotion is the startle blink response. The eyeblink startle reflex is captured by the contraction of the orbicularis oculi—the stretching of the skin under the left eye when one blinks (Blumenthal et al., 2005). The startle reflex is an automatic, defensive eyeblink response triggered by an abrupt and loud noise (or a puff of air) that is presented unexpectedly.

Two distinct neurobiological systems have been associated with the startle response. Findings suggest that activation of the central amygdala to negative stimuli, which is responsive to cue specific fear, potentiates the startle response, while activation of the nucleus accumbens to positive stimuli, attenuates the startle response (Davis et al., 1997).

The startle response is captured by two main variables, latency to blink and startle amplitude. Latency is the time required to blink after presentation of the stimulus (Cook,

Hawk, Davis & Stevenson, 1991; Hawk, Stevenson & Cook, 1992). Common response onset latency windows include 21–120ms for acoustically elicited blinks (suggested by Balaban, Losito, Simons, & Graham, 1986). Startle amplitude is the peak size of the blink response (Herpertz et al., 2001). These variable definitions follow suggested guidelines (Blumethal et al., 2005). Amplitude and blink latency have been moderately correlated -.68 (fear-neutral) (Vrana et al., 1996). That is, a participant with a large response tends to initiate responses more quickly. This pattern of response is reflexive, but the pattern of responding can alter when presented with affective stimuli. Researchers have described this pattern of responding as affect-modulated startle reactivity (Lang, 1995).

In studies exploring affect-modulated startle reactivity, participants view a series of valenced pictures, from pleasant (e.g. romance, athletes, food, and pictures of babies), to unpleasant (e.g. pictures of guns, knives, and injury), and neutral (e.g. pictures of utensils and mushrooms). Viewing of these valenced pictures leads to activation of either an appetitive (for pleasant) or defensive (for unpleasant) motivational state (Lang, 1995; Cuthbert et al., 1996). During viewing of the pictures, short noise bursts (e.g. 95-130dB) are presented occasionally. In healthy control participants, the acoustic startle eyeblink reflex is larger when viewing unpleasant pictures, as compared to neutral pictures, and attenuated during pleasant pictures, as compared to neutral pictures (Vrana et al., 1988). Effect sizes ranged from .2 to .41 in studies investigating group differences in phobia (Hamm et al., 1997; Smith et al., 2006), and psychopathy (Benning et al., 2005; Levenston et al., 2000).

Interpreting these variables (i.e., latency and startle amplitude) is more complex. Latency has been found to be dependent on both level of arousal and valence. In one

study by Bradley et al. (2006), pictures rated as high in arousal and high in unpleasant valence prompted faster time to blinks (*M* = 36.9ms) than pictures rated as high in arousal and high in pleasant valence (*M* = 40.6ms). However, latency rates for both the pleasant (*M* = 39.5ms) and unpleasant (*M* = 38.2ms) pictures rated low in arousal did not differ in onset latency, and both were slower than latency times than when presented with the high arousal high unpleasant stimuli.

Amplitude appears to depend on the arousal level and the affective state of the subject, such that the reflex is facilitated by aversive motivational states and attenuated by appetitive states (Grillon & Baas, 2003; Lang et al., 1998; Vrana et al., 1988). This was illustrated by a 1995 study by Lang et al. that directly manipulated affective states by virtue of picture content. Participants were presented with stimuli that were high-arousal negative-valenced (threat, mutilation, sickness, grief), low-arousal negative valenced (pollution), high-arousal positive-valenced (erotica, romance), low-arousal positive-valenced (food and families), and neutral (household items and mushrooms). Startle potentiation was greatest for the high arousal, negative-valenced pictures, whereas startle reactivity was least for the high-arousal, positive-valenced pictures. This finding was consistent with the notion that pictures that most strongly activate the defensive motivational system result in the strongest eyeblink response, while pictures that most strongly activate the appetitive motional system result in the least startle response (See Figure 1).

In an elegant study by Anders et al. (2004), 16 healthy participants without any history of neurologic or psychiatric disorders, viewed 40 IAPS pictures while they were presented with acoustic probes (the dB was not stated in the article). Skin conductance

responses (SCR) and eyeblink startle were measured while participants underwent fMRI scanning. In this study, startle amplitude responses were associated with activity in the amgydala while SCR responses were associated with activity in the thalamus and frontomedial cortex. Findings from this study suggest that these two measurements capture activation from stimuli and processing of affective material in different areas of the brain.

The startle response has also been employed to investigate individual differences in negative affect reactivity as an underlying vulnerability factor for psychopathology. For instance, high-fear participants in one study showed larger startle blinks and slower blink latencies while viewing aversive compared with neutral pictures, whereas low fear participants showed no potentiation for aversive scenes (Cook et al., 1992). In this study, the startle response was able to distinguish between those who are vulnerable to the negative affect that the stimulus elicited compared to those who were not. These results suggest that the aversive startle reflex varies as a function of individual differences in experienced fear.

These indices of emotional response could be another method of assessing individual differences in positive reactivity to stimuli. To my knowledge, only two studies have tested the affect-modulated startle response among adult persons with BD. Giakoumaki and colleagues (2010) asked 21 euthymic BD I participants, 19 unaffected siblings, and 42 controls to view 18 pleasant, 18 unpleasant (sadness, threat, and disgust), and 18 neutral pictures. Acoustic probes (104 dB) were presented during 12 of 18 pictures in each affective category at 300, 3000, and 4500ms after picture onset. Baseline startle, assessed during blank screens, was lower in BD participants and sibling groups,

compared to controls. Whereas the control group displayed increases in startle magnitude to negative pictures at 4500ms, as compared to baseline, both the BD I and sibling participants failed to show any change in startle reactivity from their baseline levels for any of the pictures. Results from this study suggest possible blunted reactivity among persons with BD.

Another study was able to compare BD with another group with known struggles with affect reactivity, MDD. In a 2005 study by Forbes et al., 38 participants with a history of childhood onset of unipolar depression, 38 participants with childhood onset of bipolar spectrum disorder, and 60 control participants were asked to view 12 neutral, 12 pleasant (e.g. ice cream cone), and 12 negative (e.g. mutilation) for 6 seconds with 100dB acoustic probes presented at 3, 4 or 5 seconds after picture onset. While there were no reported significant differences among the unipolar, bipolar, and control participants in eyeblink amplitude *during* presentation of the valenced stimuli, there were differences in eyeblink amplitude *after* presentation of the stimuli. Specifically, while both unipolar and control participants exhibited increased eyeblink amplitude after presentation of the negative valenced pictures, as compared to during presentation of the negative pictures, the bipolar group did not. This lack of reactivity was argued by Forbes et al. as insensitivity to negative stimuli for BD participants. That is, it was suggested that BD participants were less reactive to the negative stimuli. However, it should be noted that symptom severity only assessed depression symptoms with the Beck Depression Inventory (BDI; Beck et al., 1988a) and the Beck Anxiety Inventory (BAI; Beck et al., 1988b), and no mania scales were utilized to assess for mania/hypomania symptoms.

In sum, I have reviewed evidence that some psychophysiological measures vary with valence and arousal intensity. That is, SCL vary with the rated arousal levels of the stimuli, and gauges intensity of reactions. The startle eyeblink response varies with both valence and arousal level, in a continuum from intensely negative (greatest amplitude) to intensely positive (lowest amplitude). Within BD, there have been two studies with adults with BD investigating startle reactivity. One study found an overall blunted startle reactivity to aversive stimuli in individuals with BD and the unaffected family members. However, that study is marred by methodological problems, including (for present interests) the absence of a mood manipulation condition. While the second study also reported possible lack of reactivity to negative stimuli, there was no assessment of mania symptoms to gauge if subsyndromal mania symptoms were related to the lack of reactivity to negative stimuli.

Overall Summary

That persons with BD engage in impulsive or rash actions is well known. However, less is known about the mechanisms that may induce these rash actions. This may be partly due to failing to distinguish among the multiple facets of impulsivity. Theoretical models suggest that those with BD have stronger approach tendencies towards novel stimuli and dispositional tendencies towards impulsive actions when in a positive mood.

There is evidence of impulsivity during mania from self-report questionnaires and laboratory tasks. Some evidence up to this point suggests that the manipulation of positive emotional states has larger effects on individuals with BD as compared to controls. One possibility is that people with BD have stronger approach tendencies than

other people. Whereas the positive experience crests and then fades for other people, for people with BD it continues to build. Perhaps this is because they are less attuned to detection of cues of threat.

Taken together, these pieces of evidence suggest that a major characteristic of BD is heightened positive emotional reactivity. However, there are few studies investigating the physiological mechanisms underlying emotional reactivity in BD. Previous studies using only self-reports of emotional responses capture only conscious, reportable feelings, and ignore the fast changing reactions that are difficult to express.

Present Study

In this study, I investigated the role of positive mood in emotional reactivity to pleasant, unpleasant, and neutral stimuli. More specifically, I examined physiological responses to affective stimuli in the form of SCL and the startle response. I recruited persons with BD I, who were currently euthymic, and control participants. Data collection of SCL and startle eyeblink responses began with the first set of six neutral pictures to acclimate the participants to the acoustic startle probe. Participants were then presented with a series of two sets of six pictures each of valenced (pleasant and unpleasant) stimuli, and three sets of six pictures each of neutral stimuli. This provided a test of whether the groups differed in their responses to emotional stimuli in the absence of a mood manipulation.

Next, all participants underwent a positive mood manipulation. Participants were presented with two sets of six pictures each of valenced (pleasant and unpleasant) stimuli, and three sets of six pictures each of neutral stimuli. This second series of trials allowed

a test of how the groups responded to the mood manipulation (please see Figure 2 for order of procedure).

Hypotheses

1. *Physiological response to IAPS stimuli prior to mood induction.* I predict that BD I participants will not exhibit lower SCL or more attenuated startle responses, compared to controls in the neutral block or in the initial valenced series (i.e., pleasant, unpleasant, or neutral), prior to the mood manipulation.

2. *Mood and physiological response to IAPS stimuli after the mood induction.* After the positive mood manipulation, both the BD and control participants will report a greater positive mood, as compared to their mood at the beginning of the mood manipulation. After the mood induction, I predict that those with a reported increased positive mood will have a greater processing bias, such that among persons with BD, negative and neutral stimuli are perceived less negatively and positive stimuli are perceived more positively, compared to control participants. Thus, during the second set of trials, BD participants will display lower SCL to the threatening and neutral picture stimuli, as compared to healthy controls. BD participants will also exhibit lower startle amplitude and slower latency to the threatening and neutral picture stimulus, as compared to controls.

With repeated presentation of positive stimuli, the startle response is attenuated in healthy controls. Since individuals with BD may perceive positive stimuli more positively, I hypothesize that BD participants will have slower latency and lower startle amplitude, when compared to healthy controls, during presentation of positive stimuli.

However, since SCL is a measure of arousal, I hypothesize that individuals with BD will display greater SCL tonic to the pleasant stimuli, than compared to healthy controls.

3. *Mechanism of eyeblink startle response to acoustic probe.* Based on previous findings, I expect startle eyeblink response to be strongest when paired with the acoustic probe presented later in the viewing interval of the IAPS presentation (i.e., 4500ms) compared to the earlier presentation (i.e., 2500ms) among control participants for the threatening picture set, as a reflection of the affect-modulated startle response . I expect no pattern of increase in potentiation in later viewing of the threatening picture set for the BD participant group

Chapter 2: Method

Participants

Interested participants were recruited from the community in South Florida through community mental health centers, support groups, fliers, and print and Internet advertising (see Appendix A and B). Trained graduate students then conducted forty-five minute phone interviews to determine if individuals were eligible to participate in the study (see Appendix C). Roughly three participants were screened for every eligible participant. All phone screens were immediately shredded if the ineligible participant wished for his/her information to be shredded. All eligible participants were then asked to come in for the study session at the University of Miami, Coral Gables campus. If there was an extended period of time of greater than one week from the phone screen to the scheduled session, participants were screened for mood symptoms two days prior to their scheduled session in order to determine if they were still eligible. Upon arriving for the study session, all interested potential participants were asked to read and review the informed consent form and the researcher answered any questions concerning the protocol (see Appendix D). For participants who completed the informed consent, demographic information was collected, and diagnostic and symptom rating interviews were administered to determine study eligibility (See Appendix E – L). A clinical psychology graduate student who was trained and supervised by a clinical psychologist conducted the diagnostic interviews. Diagnostic eligibility was determined by a sole interviewer. This interviewer was a trained graduate-student in clinical psychology who had received extensive training in SCID procedures, trained by Drs. Sheri Johnson and

Ann Kring, and had previous experience administering SCIDs in multiple studies. She had also been a part of a study team with established inter-rater reliability = 1.0 for the depression and mania modules (please see Johnson, Carver & Gotlib, 2012).

Thirty-two eligible BD individuals were between 18 and 65 years of age, and English speaking. Thirty-two age-and gender-matched control individuals without a diagnosis of BDMDD, or Psychotic Disorder (SCID; First, Spitzer, Gibbon, & Williams, 1996) were eligible to participate in the study.

Exclusion criteria included the following: Mental disorders due to a general medical condition, alcohol or drug abuse or dependence within the past six months as diagnosed with the SCID, or clinically significant scores on the interview-administered mania and depression scales (i.e., scores greater than 10 on the Bech-Rafaelson Mania Scale [MAS] (see Bech et al., 2002) and 9 on the Modified Hamilton Rating Scale for Depression [MHRSD] (see Miller et al., 1985)). Amounts of caffeine and nicotine were recorded to control for hyporeactivity on SCL and the startle response (Flaten, Aasli, & Blumenthal, 2003).

Study Procedures

After completing the consent form and the demographic form, each participant completed the symptom severity measures and then met with the interviewer to complete the SCID. If each participant met inclusion criteria either for BD or control participants, the participant was asked to wash his/her hands using a gentle, non-alcohol based soap provided by the researcher. Afterwards, the participant was prepped for sensor placement. Alcohol prep pads were used gently on the forehead and under the left eye for eyeblink

startle sensors. Next, the skin was gently abraded with Mavidon "lemon prep" skin preparation. After wiping the forehead area and the skin under the left eye clean, another alcohol prep pad was used to clean off any "lemon prep" residue. Two miniature silver/silver chloride electrodes filled with electrolyte paste .8 cm were attached below the pupil and the outer canthus of the left eye, and one sensor was attached at the forehead as the grounding sensor (see Figure 3a). Palm sensors from the participants non-dominant hand was applied with two one square centimeter disposable silver/silver chloride (Ag/AgCl) electrodes, with an additional drop of Biopac 101-isotonic recording electrical gel to ensure a good electrical connection between the electrode and the skin (see Figure 3b). Leads were then attached to all electrodes, and the participants were instructed to rest their hand, palm face up, in order to minimize arm movement (see Appendix M for protocol).

Then each participant was familiarized on how to complete the Self-Assessment Manikin (SAM; Bradley & Lang, 1994; see Figure 4) rating procedure, which involves ratings of pleasure and arousal, and the Mood Grid. Then participants were asked to complete the SAM and Mood Grid. Individuals were asked to quietly sit in a dimly lit enclosed room for five minutes viewing a white slide on a 32" display with psychophysiology sensors attached. At the start of the acquisition period (after 5 minutes have elapsed), continuous psychophysiology data was collected (SCL and eyeblink response). Mood ratings were collected at multiple time points. Then participants were instructed, "You will now view a series of pictures. Watch each picture for the entire time it is on the screen. Occasionally, noise may be presented over the headphones; you should ignore the noise" (Greenwald, Bradley, Cuthbert, & Lang, 1998). The neutral trial block

consisted of viewing six different neutral slides from the International Affective Picture System (IAPS; National Institute of Mental Health [NIMH], 1998), each shown for five seconds, with an inter-picture interval of five seconds, on a 32" screen. Each neutral picture was paired with a startle acoustic probe, 95dB, a 40-msburst of broad-band white noise, either at 2500 or 4500 ms after picture onset. This stimulus was generated by a Coulbourn S81-02 white-noise generator and presented over Sennheiser HS 202 headphones.

Then the first series of target pictures (pleasant, unpleasant, and neutral) were administered. The target series were comprised of two blocks of 12 pictures each, balanced for valence and arousal levels from the IAPS, with one block consisting entirely of six all neutral pictures. The first block consisted of six unpleasant valenced pictures presented in succession and then six neutral pictures. Each picture was shown for five seconds, followed by a five second inter-picture interval with a white screen. The second block consisted of six neutral picture stimuli (each shown for five seconds, followed by a five second inter-picture interval). The third block consisted of six positive valenced pictures presented in succession and then six neutral pictures (each shown for five seconds, followed by a five second inter-picture interval). There was a 30 second inter-block interval after each of the blocks.

Sixty-six percent of the slides in each block were paired with a startle acoustic probe, 95 dB, a 40-ms burst, of broad-band white noise, at 2500 or 4500ms after picture onset. That is, two of the six slides in each content domain (pleasant, unpleasant) and two out of the six neutral slides were not presented with a startle stimulus. Four out of the

six slides in the all-neutral blocks were paired with a startle acoustic probe, 95 dB, a 40ms burst, of broad-band white noise, at 2500 or 4500ms after picture onset.

After presentation of the full three blocks, participants then viewed a white screen for one minute. Then participants completed a procedure to induce a positive mood (Pronin & Wegner, 2006). During this task, participants were asked to read 40 fast positive statements on a computer screen (described in more detail below). After the mood manipulation, the second target series of pictures were presented with the acoustic startle probe, using the same procedures as used in the first series.

Ratings of subjective affect using the Self-Assessment Manikin (SAM; Bradley & Lang, 1994) were completed at the end of the first block of neutral pictures, at the end of each of the blocks within the first target series, after the mood manipulation, and at the end of each of the blocks within the second target series. The Mood Grid was completed prior to the presentation of the first neutral block, and at the end of the entire IAPS presentation, that is the positive minus the neutral block after the positive mood induction. Additional self-report measures were completed 8 minutes after the end of the task to assess factors related to impulsivity. After this, participants were debriefed and paid $25 (please see Figure 2 for order of procedure).

Measures

Study measures included questionnaires, self-report mood measures, picture stimuli, a cognitive speed task, and physiological assessments. In the following section, I will discuss the validity of the study measures.

Questionnaires

Demographics. All participants completed a form concerning personal background. This included information on racial and ethnic background, age, gender, marital status, number of children, occupation, years of education, and income (see Appendix F). Medications and medication dosages were asked of all participants (see Appendix G).

Diagnostic and symptom measures. Several studies have reported that severity of mood symptoms can impact responding to stimuli during impulsivity studies (Dougherty et al., 2001; Forbes et al., 2005; Murphy et al., 1999; Strakowski et al., 2008; Swann et al., 2003). Previous studies on BD have varied with regard to symptoms of participants; some studies have not accounted for possible mood state effects due to psychiatric symptoms. Thus, in this study, symptom severity measures were captured by the measures below to assess current mood state.

There was one interviewer to assess for symptom severity and to administer the Structured Clinical Interview (First, Spitzer, Gibbon, & Williams, 1996). This interviewer was trained to reliability prior to the start of this study.

Structured Clinical Interview for DSM-IV (SCID). The SCID (First, Spitzer, Gibbon, & Williams, 1996) is a semi-structured interview administered to determine DSM -IV Axis I diagnoses. Participants completed the lifetime and current depression, mania, substance abuse and dependence, anxiety and psychosis disorders modules of the SCID. The SCID has been used to determine DSM-based mental health diagnoses in research studies for over twenty years. This diagnostic instrument has been reliable in

diagnoses for bipolar disorders and for mania symptoms (alpha = .94; e.g., Johnson, Winett, Meyer, Greenhouse, & Miller, 1999).

Modified Hamilton Rating Scale for Depression (MHRSD). The MHRSD (Miller, Bishop, Norman, & Maddever, 1985) is a 17-item interviewer-administered scale designed to assess symptoms of current depression such as depressed mood, guilt, and appetite loss (see Appendix J). This modified version correlates highly with the original HRSD (r = .84), and high interrater reliability has been observed with an interclass correlation (alpha = .93; Johnson et al., 1999). Internal consistency was at alpha = .78 in the current sample.

Bech-Rafaelson Mania Scale (MAS). The MAS (Bech, Bolwig, Kramp, & Rafaelsen, 1979) contains 11 items that assess symptoms such as elevated mood, sleep disturbance, and activity level. Each item is rated on a 5-point scale, of 0 (not present) to 4 (severe) (see Appendix I). The MAS was used to assess symptoms of current mania among the participants in the current study. The scale is widely used to assess manic symptoms and has demonstrated high interrater reliability (alpha = .92) and is also highly sensitive to changes in mania (Johnson, Winett, Meyer, Greenhouse, & Miller, 1999). Internal consistency was alpha = .69 in this sample.

Self-report symptom scales. Previous studies have shown that self-report symptoms and clinician ratings on mood symptoms have differed (Altman et al., 1997). Therefore, incorporation of self-rating scales can further validate mood states and symptom ratings.

The Altman Self-Rating Mania Scale (ASRM). The ASRM (Altman, Hedeker, Peterson & Davis, 1997) is a self-report questionnaire used to assess current mania

severity (see Appendix K). This 5-item scale was designed to quickly assess mania severity. This scale has been used to assess manic and hypomanic symptoms and has shown acceptable interrater reliability (alpha = .70; Meyer, Beevers, & Johnson, 2004). Internal consistency was at alpha = .77 in the current sample.

Mood and Anxiety Symptoms Questionnaire (MASQ). The MASQ-D30 (Wardenaar, van Veen, Giltay, de Beurs, Penninx, & Zitman, 2010) is a shortened thirty-item version questionnaire of the MASQ (Watson, Clark, Weber, Assenheimer, Strauss, & McCormick, 1995) which measures feelings, sensations, problems and experiences related to mood and anxiety in the past week (see Appendix L). Participants are asked to rate responses on a 5-point scale ranging from "not at all" to "extremely". Participants are asked to rate the extent to which they experienced each symptom "in the past week, including today." Subscales include: symptoms of anxiety and depression and anhedonia anxious arousal, and high positive affect. The scale has high internal consistency (alpha = .87 - .93), and the MASQ- D30 correlates with other measures of such as the Beck Depression Inventory-II (r = .79), Beck Anxiety Inventory (r = .76), and the Montgomery Asberg Depression Rating Scale (r = .70). This scale has strong internal consistency (alpha = .83 in the current sample).

Mood Grid. This grid asks participants to self-report their current level of happiness, confidence, relaxation, irritability, excitement, and talkativeness. Each of the six items is rated on a seven-point scale ranging from one "not at all" to seven "extremely", with four representing a neutral mood. The grid is comprised of items from several other scales, including the Positive and Negative Affect Schedule (PANAS; Watson & Clark, 1994), the Affect Valuation Inventory (AVI; Tsai, Knutson, & Fung,

2006), and a brief mood grid for assessing valence/arousal (Jefferies, Smilek, Eich, & Enns, 2008). The average alpha across administration for all six items was = .73.

The Self-Assessment Manikin (SAM). After presentation of the first series of picture sets, participants were asked to self-rate their mood and then were presented with the Pronin mood induction in which they were asked to read out loud fast-paced statements on reward, positive mood, and goal-achievement. Participants were again asked to rate their mood after the mood induction.

Participants rated their emotional experience using a computerized version of the Self-Assessment Manikin (SAM; Bradley and Lang, 1994). The SAM uses manikin figures on a 9-point scale for each of the two dimensions (see Figure 4). On the valence dimension, the SAM figures range from happy, smiling figure (1, very pleasant) to an unhappy, frowning figure (9, very unpleasant) (see Figure 4) . A change in mood was defined as a point change in the SAM manikin in mood (Lang et al., 1997; Cyders et al, 2010; Forbes et al., 2005). On the arousal dimension, the SAM figures range from an excited figure with eyes wide open and an active body (1, very aroused) to a calm figure with closed eyes and an inactive body (9, very calm). Previous research has demonstrated that the SAM valence and arousal dimensions reliably covary with physiological reactions associated with emotional arousal. Internal consistency for SAM Mood ratings was alpha = .88 in this sample. Internal consistency for SAM Arousal ratings was alpha = .89 in this sample. Participants read a script developed by Bradley & Lang (1994).

> You will see 2 sets of 5 figures, each arranged along a continuum. We call this set of figures SAM, and you will be using these figures to rate how you felt while viewing the pictures. SAM shows two different kinds of feelings: Happy vs. Unhappy, and Excited vs. Calm. You can see that each SAM figure varies along each scale. In this illustration, the first SAM scale is the happy-unhappy scale, which ranges from a smile to a frown.

> At one extreme of the happy vs. unhappy scale, you felt happy, pleased, satisfied, contented, hopeful. If you felt completely happy you can indicate this by placing an "X" over the figure at the left, like this (demonstrate with SAM1). The other end of the scale is when you felt completely, unhappy, annoyed, unsatisfied, melancholic, despaired, bored. You can indicate feeling completely unhappy by placing an "X" on the figure at the right, like this (demonstrate with SAM2). The figures also allow you to describe intermediate feelings of pleasure, by placing an "X" over any of the other pictures. If you felt completely neutral, neither happy nor sad, place an "X" over the figure in the middle. If, in your judgment, your feeling of pleasure or displeasure falls between two of the pictures, then place an "X" between the figures, like this (demonstrate with SAM3). This permits you to make more finely graded ratings of how you feel. The excited vs. calm dimension is the second type of feeling displayed here. At one extreme of the scale you felt stimulated, excited, frenzied, jittery, wide-awake, aroused. On the other hand, at the other end of the scale, you felt completely relaxed, calm, sluggish, dull, sleepy, unaroused. You can indicate you felt completely calm by placing an "X" over the figure at the right of the row, like this (demonstrate with SAM5). As with the happy-unhappy scale, you can represent intermediate levels by placing an "X" over any of the other figures. If you are not at all excited nor at all calm, place an "X" over the figure in the middle of the row.

Laboratory Tasks

Cognitive speed task. I used an empirically-tested procedure (Pronin & Wegner, 2006) to induce a positive mood. That is, before undergoing the mood induction, participants were given instructions at the beginning of the task:

> Once you get started, you will see a series of statements presented one word at a time on the screen. Read each word of each sentence aloud as it appears. Don't worry if it takes you a few sentences before you get used to it. When you're ready to begin, click the mouse once, and the study will begin. And remember, as soon as words start to come up on the screen, you should be reading them.

First participants were asked to read 5 neutral statements to help them learn the task. Then, participants were asked to read sixty statements such as, "My thinking is clear

and rapid" on a computer screen presented at a speed of 40ms per letter and 320 ms between slides. The fast statements are roughly twice as fast as normal reading speeds (Pronin & Wegner, 2006).

In a 2008 study, Pronin et al. asked undergraduate participants to read statements at a fast or a slow rate to determine if speed correlated with specific mood states. In the fast condition, statements were presented at a speed of 40ms per letter and 320 ms between slides. In the slow condition, statements appeared at 170 milliseconds per letter (with an additional 4,000 milliseconds between slides). Pronin et al. (2008) reported that participants reported significantly more positive affect after the fast condition than they did at baseline (*Ms* = 4.59 vs. 3.63, *SD*s = 1.52 and 1.30). Only the group randomized to the fast condition also reported pressured speech and increased feelings of grandiosity. The reverse was true for participants in the slow condition: They reported less positive affect after participating in the slow condition compared to baseline (*Ms* = 3.70 vs. 4.28, *SD*s = 1.43 and 1.01), $F(1, 15) = 7.17$, $p = .02$.

In another study (Chandler & Pronin, 2012) participants were asked to read a series of fast- or slow- paced statements, and afterwards, participants completed the BART task (Lejuez et al., 2002). Participants in the fast-paced group reported faster thought speed, took more risks on the BART task, and reported greater positive mood than those in the slow speed. Thus, studies manipulating the speed task have indicated increased positive affect, faster thought speed, feelings of grandiosity and an increased willingness to take risks for participants. Many of these outcomes are similar to symptoms of mood elevation. As racing thoughts are identified as a cardinal prodrome of

mania (Lam & Wong, 1997), the effect of the mood induction may be analogous to the subtle mood shifts experienced by individuals with BD.

International Picture System (IAPS). The IAPS (Center for the Study of Emotion and Attention, 1999; Lang, Bradley, & Cuthbert, 1999; Lang, Bradley, & Cuthbert, 2001) is a picture set of over 1000 pictures that have been rated for emotional valence and arousal. To assess the two dimensions of pleasure, and arousal, the Self-Assessment Manikin (SAM), an affective rating system devised by Lang (1980) was used. The mean ratings of valence and arousal for these materials are highly internally consistent. The split-half coefficients for the valence and arousal dimensions were highly reliable ($p < .001$; $r's = .94$ and .93, respectively for 21 pictures). These pictures have been viewed by over 3000 university student participants (Bradley & Lang, 2007) and are routinely used as a paradigm to elicit psychophysiology measures such as skin conductance (e.g., Lang et al., 2003; Greenwald et al., 1989), and startle response (e.g., Giakoumaki et al., 2010; Patrick et al., 1993; Smith et al., 2006;). As well, IAPS has been used to study emotional response in various clinical populations: neurological disorders (e.g. Siebert, Markowitsch & Bartel, 2003), PTSD (e.g., Amdur, Larsen & Liberzon, 2000), depression (e.g., Dichter, Tomarken & Baucom, 2002), and schizophrenia (e.g. Quirk & Strauss, 2001). A collection of 42 neutral, 12 threat-related and 12 positive pictures presented in previous studies investigating threat and fear were shown for 5 seconds each (Bradley et al., 2001; Greenwald et al., 1998; Lang et al., 1990; Smith et al., 2005; Vrana et al. 1988).

The study slide deck (see Appendices A-C for individual picture valence and arousal levels) has been utilized in previous studies and matched to arousal levels for the

unpleasant and pleasant slides (Bradley et al., 2001; Greenwald et al., 1998; Lang et al., 1990; Smith et al., 2005; Vrana et al. 1988). The themes of the threat-related pictures consisted of: animal attack, human attack, accident, mutilation and aimed pictures of guns. The themes of the neutral pictures consisted of mushrooms, utensils and buildings. The positive themed pictures consisted of: athletes, money and adventure.

Physiological Measures

Physiological data were collected between March 2011 and July 2011. Startle eyeblink reactivity data was analyzed using Mindware Technologies Electromyography (EMG 3.0.15) Analysis Software. SCL data was analyzed using Mindware Technologies Electrodermal Activity Skin Conductance (EDA 3.0.15) Analysis Software.

Startle response. The eyeblink component of the startle reflex was indexed by recording activity of the orbicularis oculi muscle directly beneath the left eye, by positioning two miniature silver/silver chloride electrodes filled with electrolyte paste .8 cm below the pupil and the outer canthus of the left eye. The following startle measures were examined: startle amplitude and latency.

Following parameters set by Vaidyanathan et al., (2009), trials were identified as unstable if blink onset occurred earlier than 20ms or if the startle response overlapped with a preceding spontaneous eyeblink. Zero amplitude response trials were defined as trials in which no discernible blink response occurred within the 21–120ms peak window after acoustic startle presentation. Of the entire study sample, 8% of the trials were identified with a zero response for eyeblink amplitude; 2 BD and 2 control participant's eyeblink amplitude data was excluded from analyses since they had over 30% of zero response trials. Startle amplitude was calculated by averaging responses for each valence

(unpleasant, pleasant and neutral) and by timing (2500ms, 4500ms) of stimuli for each block.

Latency is the time required to blink after presentation of the stimulus (Cook, Hawk, Davis & Stevenson, 1991; Hawk, Stevenson & Cook, 1992). Common response onset latency windows include 21 to 120ms for acoustically elicited blinks (suggested by Balaban, Losito, Simons, & Graham, 1986). Unstable responses were identified as responses not occurring within the 21 to 120ms window. 13% of the trials were identified as unstable responses; 3 BD and 3 control participant's eyeblink latency data was excluded from analysis since over 30% of their trials were unstable.

Data was collected using BIOPAC bioamplifiers. Measures were A/D converted, sampled at 1000 Hz, and processed using AcqKnowledge and MindWare softwares. The Coulbourn S81-02 white-noise generator was used to generate the acoustic startle stimuli. I verified volume levels at exactly 95 dB using an acoustic decibel tester, the Reliability Direct AR824 Multi-Range Sound Level Meter.

Skin conductance level (SCL). SCL was assessed with the placement of two Ag/AgCl electrodes (within 1-cm² contact area) filled with 0.05-mol NaCl unibase electrode paste (Fowes et al., 1981) on the medial phalanx surfaces of the middle and index fingers of the nondominant hand. SCL data was captured with Biopac Systems Inc., model mp 100A linked to a PC by means of a computer software program, Mindware. After the experimenter places the sensor on the participant's fingers, participants were given at least 5 minutes to acclimate to the equipment. The following variables were measured within the suggested typical values (Dawson et al., 2000): tonic level with typical values between 2-20 microsiemens. Potential confounds, including medications,

nicotine, caffeine, and medical conditions were assessed. A room temperature gauge was checked prior to each session to maintain a room temperature of 23°C-27°C (73.4°F-80.6°). SCL data was analyzed using Mindware Technologies Electrodermal Activity Skin Conductance (EDA) Analysis Software. SCL tonic level data was examined separately by valence and averaged timing effects (2500ms, 4500ms) of stimuli in order to investigate electrodermal reactivity to stimuli. Data values that were not within the range of 2-20 microsiemens were excluded from analyses. Of the entire study sample, 16% of the trials were not within range; three control participants and four BD participant's SCL data was excluded from analyses.

Chapter 3: Results

Planned Analyses

Analyses for the first hypothesis consisted of repeated-measures ANOVAs with group (bipolar, control) as the between-subjects variable, and valence and timing (2500ms, 4500ms) as the within-subjects variables for only the blocks prior to the mood induction. Analyses for the second hypothesis consisted of repeated-measures ANOVAs with group (bipolar, control) as the between-subjects variable, and valence and timing (2500ms, 4500ms) as the within-subjects variables for only the blocks after the mood induction. Then, in order to test the feasibility of combining the pre and post mood induction data, repeated-measures ANOVAs were conducted with group as the between-subjects variable, and timing (2500ms, 4500ms) and block (block 1[neutral stimuli following the block of negative stimuli], block 2 [all neutral block], block 3 [neutral stimuli following the block of positive stimuli]) as the two within-subjects factors, for the neutral blocks before and after the mood induction. Then, the neutral blocks were averaged by timing, and I conducted repeated-measures ANOVAs comparing findings before and after the mood induction. The pre-post analyses included, group (BD, control) as a between-subjects factor, and pre-post (before the mood induction, after the mood induction), valence (negative, neutral, positive), and time (2500ms, 4500ms) as the three within-subjects factors.

Preliminary Analyses

Before examining the hypotheses, preliminary analyses investigated whether demographic, symptom ratings, and self-report variables (please see Table 2a) and psychophysiological variables (please see Table 3a-c) were normally distributed. Having

a kurtosis or skew outside of an acceptable range of +/-2 standard errors (Cramer & Howitt, 2004) or a statistically significant Shapiro-Wilk result suggests a non-normal distribution. Analyses indicated that the MASQ Anhedonic Depression subscale score was skewed and had a leptokurtic distribution (skew = 1.76, *SE* = .34; kurtosis = 2.65, *SE* = .67; Shapiro-Wilk = 0.76, $p < .001$), the MASQ Somatic Anxiety subscale was skewed and had a leptokurtic distribution (skew = 2.02, *SE* = .34; kurtosis = 4.62, *SE* = .67; Shapiro-Wilk = 0.77, $p < .001$), the Reverse Digit Span (RDS) score was skewed (skew = .77, *SE* = .34; kurtosis = .46, *SE* = .67; Shapiro-Wilk = .93, $p = .001$), and the total BRMS score was skewed (skew = 1.31, *SE* = .34, kurtosis = -1.41, *SE* = .67; Shapiro-Wilk = .84, $p < .001$). Logarithmic transformation of the data reduced the skew of the MASQ Anhedonic subscale to 0.57 (*SE* = .36), reduced kurtosis to -0.15 (*SE* = .70), which resulted in a non-significant Shapiro-Wilk statistic, D(58) = 0.76, *ns*. Logarithmic transformation of the data reduced the skew of the MASQ Somatic Anxiety subscale to .62 (*SE* = .36), reduced kurtosis to -.54 (*SE* = .70), which resulted in a non-significant Shapiro-Wilk statistic, D(58) = 0.65, *ns*. Analyses on both variables were conducted using the log-transformed data. Square-root transformation of the RDS reduced skew to .04 (*SE* = .36), reduced kurtosis to .30 (*SE* = .70), which resulted in a non-significant Shapiro-Wilk statistic, D(58) = .96, *ns*. Log transformation of the BMRS reduced skew to -.42 (*SE* = .36).

In reviewing the psychophysiological data, there was one participant with data that systematically was not within range for the two startle response variables, and one other participant with erratic readings. Eyeblink response values out of range suggest poor sensor placement. The one participant with erratic values was noted to be chewing

gum during the session. One participant also had SCL tonic range values which suggested poor sensor placement, and one person was noted to be moving his hand during the session. Thus, data from these four individuals were excluded from analyses due to invalid values. I then assessed for kurtosis and skew in the physiological variables. Eyeblink latency and SCL tonic level data were within acceptable ranges for skew and kurtosis (Cramer & Howitt, 2004) (please see Tables 4b-c); however, eyeblink amplitude data were not within acceptable ranges. Thus, eyeblink amplitude data were square-root transformed. The transformed values are within the accepted -/+2 SE range (please see Table 4a).

Characteristics of Sample

Demographic characteristics of the resulting sample (n = 56) can be found on Table 1a. I explored between-group differences in the following continuous variables: demographics (age, years of education), symptom characteristics (mania severity score, depression severity score, self-reported anxiety symptom score), temperature of room, and time of day. I tested for group differences in the following categorical variables: percentage of gender, anxiety disorder history, alcohol or substance abuse history, use of nicotine/caffeine. There were no group differences in gender, age, self-reported mania symptoms, temperature, time of day, or use of nicotine/caffeine. The BD I group also self-reported higher Anhedonic Depression symptoms from the MASQ, ASRM scores, and Somatic Anxiety symptoms from the MASQ, as compared to control participants. BD I participants were rated with higher HAM-D depression scores, as compared to control participants, but the group mean for BD I participants was below the cut-off for a moderate-severity range based upon the HAM-D (Angst et al., 1993), and no participant

was above the exclusion criterion. There were also group differences in the percent of people who endorsed a lifetime history of anxiety Disorders and substance or alcohol abuse. A greater percentage of BD participants endorsed either generalized anxiety disorder (GAD) or panic disorder, and also lifetime alcohol and/or substance abuse disorder, as compared to the control participants.

Attention check. Participants periodically were presented with a question on the computer screen asking them to recall if a certain picture item (i.e. wallet) has been presented in the previous picture block. Participants were asked to respond with a 1 "yes" or 2 "no". These attentional checks were presented after the negative-neutral and positive-neutral blocks, for a total of 4 times during the entire session. Participants could not continue with the study session until the question was answered. Four simple *t*-tests determined that the percentage of accurate recognition of the pictures used in the study did not differ between groups, and all participants had greater than 80% correct recognition in the sample set. No records were removed from the dataset because of low recognition scores.

Test of Hypotheses

Physiological response to IAPS stimuli before mood induction. The first hypothesis was that there would be no group difference in SCL (i.e. tonic level) and startle eyeblink response (i.e. latency and startle amplitude) in the first block of neutral pictures or the valenced picture sets before the mood induction. Only significant findings will be discussed in this section, I will refer to relevant tables for non-significant findings.

Initial neutral block. In analyses of the first hypothesis, I utilized a series of ANOVAs with group (bipolar, control) as the between-subjects factor, and timing

(2500ms, 4500ms) as the within-subjects factor, in the initial neutral block. There were no significant group main effects, main effects or interaction effects for SCL, eyeblink amplitude, or eyeblink latency (see Table 7).

Negative versus neutral block. In the next series of ANOVAs, I investigated group differences in physiological variables comparing the first negative block of IAPS stimuli to the neutral block of IAPS immediately following the negative set, with group (bipolar, control) as a between-subjects factor, and timing (2500ms, 4500ms) and valence (negative, neutral) as two within-subjects factors (see Table 8).

SCL tonic levels. In the first ANOVA, I tested for group difference in SCL. There was no between-group difference. There was a significant valence effect, $F(1,50) = 41.14$, $p < .001$, Cohen's $d = .18$, such that SCL tonic level was greater during presentation of the negative IAPS stimuli ($M = 7.59$, $SD = 4.03$) than in the neutral stimuli that followed ($M = 6.88$, $SD = 3.97$). There was a significant timing effect, $F(1,50) = 26.55$, $p < .001$, Cohen's $d = .06$, such that SCL tonic levels were greater when the acoustic probe was presented at 2500ms ($M = 7.36$, $SD = 4.01$), than when presented at 4500ms ($M = 7.11$, $SD = 3.96$). In sum, SCL tonic level was greater during the negative set, as compared to the neutral set, and when the acoustic probe was presented at 2500ms, as compared to at 4500ms. However, there was no between-group difference.

Eyeblink amplitude. In the next ANOVA, I examined eyeblink amplitude. There was a significant valence effect, $F(1,50) = 9.91$, $p = .003$, Cohen's $d = .33$, such that eyeblink amplitude was greater during the negative IAPS set ($M = 5.28$, $SD = 1.19$), as compared to the neutral set ($M = 4.93$, $SD = .95$). These results suggest that eyeblink

amplitude did increase when the stimuli were negative, but that the BD and control group did not responded differently from one another.

Eyeblink latency. In the next ANOVA, I tested for group difference in eyeblink latency. There was a significant timing effect, $F(1,52) = 8.35$, $p = .006$, Cohen's $d = .44$, such that blinks were faster when the acoustic probe was presented at 2500ms ($M = .72$, $SD = .28$), as compared to at 4500ms ($M = .85$, $SD = .31$). Blinks were faster to the 2500ms acoustic probe, as compared to the 4500ms probe. However, there was no between-group difference.

Neutral-only block. In the next series of ANOVAs, I tested for group difference in the all neutral IAPS set, with group (bipolar, control) as a between-subjects factor, and timing (2500ms, 4500ms) as the within-subjects factor. There were no significant group main effects, main effects or interaction effects for SCL, eyeblink amplitude or eyeblink latency (see Table 9).

Positive versus neutral block. In the next series of ANOVAs, I tested for group difference in physiological variables in the first positive set of IAPS stimuli and the subsequent neutral set of IAPS, with group (bipolar, control) as a between-subjects factor, and timing (2500ms, 4500ms) and valence (positive, neutral) as two within-subjects factors (see Table 10).

SCL tonic levels. In one ANOVA, I examined group difference in SCL. There was a significant valence effect, $F(1,48) = 10.88$, $p = .002$, Cohen's $d = .07$, such that SCL tonic level was greater during the positive IAPS set ($M = 6.44$, $SD = 4.09$), as compared to the neutral set ($M = 6.16$, $SD = 4.05$). There was a significant timing effect, $F(1,48) = 7.56$, $p = .008$, Cohen's $d = .04$, such that SCL tonic level was greater when

the acoustic probe was presented at 2500ms (M = 6.47, SD = 4.07), as compared to when presented at 4500ms (M = 6.30, SD = 4.01). Thus, the SCL results during the positive-neutral IAPS set indicate that SCL tonic levels were greater during presentation of the positive stimuli, as compared to neutral, and greater during presentation of the 2500ms acoustic probe, as compared to 4500ms, but that there were no group differences.

Eyeblink amplitude. Next, I explored group difference in eyeblink amplitude. There was a significant interaction among valence, timing, and group, $F(1,52) = 5.02$, $p = .029$. In order to partition the significant three-way interaction, I ran two separate repeated-measures ANOVAs with group as a between-subjects factor, and timing (2500ms, 4500ms) as the within-subjects factor, for each valence. In the first ANOVA, I investigated difference in eyeblink amplitude in the negative valence. There was no significant main group effect, or significant interaction between timing and group. In the second ANOVA, I examined eyeblink amplitude in the neutral set. There was no significant group main effect, or timing effect. However, there was a significant interaction effect between timing and group, $F(1,52) = 6.46$, $p = .01$. Next, I conducted follow-up analyses using two paired-sample t-tests with a Bonferroni p-value set at.05/2. I tested whether: 1) there was a difference in eyeblink amplitude for BD participants as a function of timing (2500ms, 4500ms) in the neutral set and 2) there was a difference in eyeblink amplitude for control participants as a function of timing (2500ms, 4500ms)[1]. In my first follow-up question, analyses indicated that there was a significant difference in eyeblink amplitude in timing, such that BD participants displayed greater amplitude in the neutral set when presented with the 4500ms acoustic probe (M = 5.18, SD = 1.28),

[1] I conducted independent t-tests at each of the times, 2500ms and 4500ms, within the neutral set and found no significant group difference, p's > .05.

compared to when presented with the 2500ms probe (M = 4.59, SD = 1.01), $t(25) = -2.53$, $p = .013$. In my second follow-up question, analyses indicated that there was no significant difference for control participants, $t(27) = 1.17$, *ns*, when comparing eyeblink amplitude when the acoustic probe was presented at 2500ms (M = 4.89, SD = 1.02) or at 4500ms (M = 4.59, SD = 1.13). In sum, BD participants displayed greater eyeblink amplitude when presented with the 4500ms probe, as compared to when presented with the 2500ms probe. Control participants did not display a difference in eyeblink amplitude during the neutral set.

Eyeblink latency. In the next ANOVA, I tested for group difference in eyeblink latency. There were no significant group main effect, main effects or interaction effects.

In sum, there were no group difference in SCL or eyeblink latency in any of the blocks. However, BD participants displayed greater eyeblink amplitude in the neutral set with the later timed acoustic probe. This finding was contrary to predictions.

Mood and physiological response to IAPS stimuli after the mood induction. After the positive mood manipulation, it was expected that both the BD and control participants would report a higher positive mood, as compared to their mood at the beginning of the mood manipulation. However, it was expected that there would be no between-subjects group difference in mood measures pre-mood manipulation and post-mood manipulation. Because arousal could be indicative of either negative or positive valenced emotions, only the SAM mood self-report measure was used as a measure of mood change.

Test of mood induction. To test this hypothesis, a two-way repeated-measures ANOVA with group (bipolar, healthy control) as a between-subjects factor, and pre-post

as the within-subjects variable (SAM Mood prior to the mood induction, SAM Mood after the mood induction), was conducted. There was no significant group main effect, $F(1,54) = 0.11$, *ns*. However, the main effect of pre-post was significant, $F(1,54) = 16.39$, $p < .001$, Cohen's $d = .57$. Participants rated their mood as happier (SAM mood rating $M = 6.63$, $SD = 1.36$) after the mood induction than before the mood induction (SAM mood rating $M = 5.89$, $SD = 1.21$). The pre-post by group interaction effect was not significant, $F(1,54) = 0.11$, *ns*, which indicates that there were no differences in reported mood change between the two groups of participants (see Tables 5a and 5b for mean and standard deviation values for SAM Mood and Arousal ratings for all study participants with physiological values).

Subset of participants who endorsed positive mood. Further examination of the data identified a subset of participants who had reported a positive mood change and another subset who had not. Of the 27 BD I participants: 22 reported at least a one point positive mood change or rated their mood as positive (within the range of "6-9") before and after the mood induction; five reported a negative change of mood or rated their mood as negative ("1-4") before and after the mood induction. Of the 29 control participants: 25 reported at least a one point positive mood change or rated their mood as positive before and after the mood induction; four reported a negative change of mood or a negative mood before and after the mood induction. That is, 29% of the BD participants, and 14% of the control participants did not report a positive mood after the mood induction. These proportions did not differ significantly from each other, $X^2(5,56) = 3.82$, *ns*.

Because the second and third hypotheses of the study are based upon a successful induction of positive mood, subsequent analyses only included the subset of participants (BD: $n = 22$; control: $n = 25$) who either reported a one-point positive mood change after the induction or reported a sustained positive mood before and after the mood induction.

Subset comparisons and re-analyses. Overall, there were no significant differences in demographics or symptom characteristics between BD participants who did report positive mood induction as compared to BD participants who did not. There were no significant differences in demographics between controls participants who reported a mood change as compared to control participants who did not (please see Table 20). However, there was a difference in symptom characteristics for control participants who reported a positive mood as compared to those who did not. Control participants who did not report experiencing a positive mood also had greater MASQ-Anhedonia Depression self-report scores ($N = 4$; $M = 1.06$, $SD = .23$) than control participants who reported experiencing a positive mood ($N = 25$; $M = .91$, $SD = .08$). In testing for a difference between these two groups using the Kruskal-Wallis test, findings indicated a difference in median scores, $X^2(1, N = 29) = 4.07$, $p = .044$. As initial preliminary analyses showed data with large skew and kurtosis ranges, subsequent analyses used log transformed data for the MASQ-Anhedonic Depression and MASQ-Somatic Anxiety and the transformed physiological values.

I then repeated the analysis of pre-post mood scores with only the reduced subset, using a repeated-measures ANOVA with group (bipolar, control) as a between-subjects factor, and pre-post (pre and post SAM mood ratings) as the within-subjects factor. The group main effect was not significant, $F(1,45) = 0.44$, *ns*. However, the repeated

measures main effect of pre-post was significant, $F(1,45) = 42.69$, $p < .001$, Cohen's $d = .90$. Overall, participants rated their mood as happier (SAM mood rating $M = 7.07$, $SD = 1.16$) after the mood induction than before the mood induction (SAM mood rating $M = 5.93$, $SD = 1.36$). The SAM mood by group interaction effect was not significant, $F(1,45) = 0.09$, *ns*, which indicates that there were no differences in reported mood change between the two groups of participants (see Tables 6a and 6b for mean and standard deviation values for SAM Mood and Arousal ratings for this subset of participants). I also conducted a repeated-measures ANOVA with the pre and post SAM arousal rating as the within-subjects factor. There was no significant group main effect, $F(1,44) = 1.46$, *ns*. There was a significant pre-post effect, $F(1,44) = 21.68$, $p < .001$, such that participants rated their arousal levels higher before the mood induction ($M = 3.57$, $SD = 1.62$) as compared to after the mood induction, ($M = 4.87$, $SD = 1.72$).

Analysis of subset for blocks prior to mood induction. To ensure that the results reported thus far remain after reducing the subject sample, I repeated all of the analyses described above under Hypothesis 1 to investigate physiological responding prior to the mood induction for this sample subset. Findings were nearly identical, and only one finding differed from the entire sample regarding Hypothesis 1.

I investigated difference in eyeblink amplitude in the first positive block, with an ANOVA with group (bipolar, control) as a between-subjects factor, and timing (2500ms, 4500ms) and valence (positive, neutral) as the two within-subjects factors. This analysis now yielded no significant interaction among timing, valance and block, $F(1,40) = 2.73$, *ns*.

Physiological responses after the mood induction. In the next series of ANOVAs, I tested differences in physiological variables after the positive mood induction. I predicted that BD participants would perceive the negative and neutral stimuli less negatively, as compared to control participants, after the mood induction. Specifically, BD participants would display lower SCL, lower eyeblink amplitude, and longer eyeblink latency, as compared to control participants, when viewing negative and neutral stimuli. Moreover, I predicted that BD participants would perceive positive stimuli more positively, as compared to control participants, after the mood induction. As such, BD participants would display lower eyeblink amplitude, longer eyeblink latency, but greater SCL tonic levels, as compared to control participants, when viewing positive stimuli.

Negative versus neutral block. I started by comparing the second negative block of IAPS stimuli to the neutral block of IAPS that immediately followed that negative set, with group (bipolar, control) as a between-subjects factor, and timing (2500ms, 4500ms) and valence (negative, neutral) as the two within-subjects factors (see Table 11).

SCL tonic levels. With the first ANOVA, I examined SCL. There was a significant valence effect (negative, neutral set following the negative set), $F(1,38) = 29.05$, $p < .001$, Cohen's d = .20, such that SCL tonic levels were greater during the negative IAPS set ($M = 8.34$, $SD = 4.67$), as compared to during the neutral set ($M = 7.45$, $SD = 4.28$).

There was also a significant interaction between valence and group, $F(1,38) = 4.04$, $p = .05$. Post-hoc analyses on this interaction were conducted with independent *t*-tests using a Bonferroni correction set at a significant *p*-value of .05/3. In the first

comparison, I investigated group differences in the negative IAPS set. In the second comparison, I investigated differences in SCL tonic level as a function of valence among only control participants. In my third comparison, I investigated SCL tonic level as a function of valence among only BD participants. In reviewing the SCL means of the groups in both the negative (BD: $M = 6.92$, $SD = 3.84$; control: $M = 9.51$, $SD = 4.94$) and neutral IAPS sets (BD: $M = 6.39$, $SD = 3.90$; control: $M = 8.31$, $SD = 4.47$), all means were similar except for the mean for control participants in the negative IAPS set ($M = 9.51$, $SD = 4.94$).

Findings from the first post-hoc comparison suggest no statistically significant group difference, $t(38) = 1.82$, *ns*, in SCL tonic levels between BD ($M = 6.92$, $SD = 3.84$) and control participants ($M = 9.51$, $SD = 4.94$) in the negative IAPS set. The second post-hoc comparison, indicated that there was a significant difference in SCL tonic levels, $t(21) = 5.22$, $p < .001$, such that SCL was greater in the negative IAPS set ($M = 9.51$, $SD = 4.94$) as compared to the neutral ($M = 8.31$, $SD = 4.47$) IAPS set among control participants. The third post-hoc comparison indicated that while SCL tended to be greater in the negative set ($M = 6.92$, $SD = 3.84$), as compared to the neutral set ($M = 6.39$, $SD = 3.90$) among BD participants, the difference was not statistically significant, $t(17) = 2.24$, *ns*. That is, BD participants after the mood induction are less reactive to negative stimuli after a positive mood. As there were no other group differences over other blocks, this finding may point to the unique role of positive mood in the lack of detection of negative stimuli.

In addition to the effects just described, there was a significant timing effect, $F(1,38) = 9.33$, $p = .004$, Cohen's $d = .05$, such that SCL tonic levels were greater when

presented with acoustic probes at the 2500ms time (M = 8.00, SD = 4.42), than when presented at 4500ms (M = 7.79, SD = 4.43), when collapsed across the negative and neutral sets.

In conducting post-hoc analyses on the interaction between valence and timing, I used three paired samples *t*-tests with a Bonferroni correction significance *p*-value set at .05/3. I 1) used one *t*-test to determine if there was a significant difference as a function of timing in the negative IAPS set, then 2) investigated if there was a difference in timing in the neutral set, and then 3) investigated differences in valence when the acoustic probe was presented at 4500ms. The first comparison found that SCL tonic levels were greater, $t(39) = 3.44$, $p = .001$, when the acoustic probe was presented at 2500ms (M = 8.51, SD = 4.65), as compared to at 4500ms (M = 8.18, SD = 4.60), in the negative IAPS set. The second *t*-test indicated that while SCL tended to be greater when the probe was presented at 2500ms (M = 7.49, SD = 4.27), as compared to at 4500ms (M = 7.40, SD = 4.31) in the neutral set, this difference was not significant, $t(39) = 1.16$, *ns*. Results from the third post-hoc indicated that SCL was greater in the negative set (M = 8.18, SD = 4.59), as compared to the neutral set (M = 7.40, SD = 4.31), when the probe was presented at 4500ms, $t(39) = 4.93$, $p < .001$.

Eyeblink amplitude. I then conducted an ANOVA on eyeblink amplitude. There was a significant valence effect, $F(1,40) = 4.06$, $p = .05$, Cohen's $d = .06$, such that eyeblink amplitude was greater during the negative IAPS set (M = 7.36, SD = 5.36), as compared to the neutral set (M = 7.06, SD = 5.09).

Eyeblink latency. In the next ANOVA, I examined eyeblink latency. There were no significant group main effect, no main effects or interaction effects.

Neutral-only block. The next series of ANOVAs tested for group difference in the all-neutral picture stimulus, with group (bipolar, control) as a between-subjects factor, and timing (2500ms, 4500ms) as the within-subjects factor. There was no significant group main effect, main effect or interaction effect in SCL, eyeblink amplitude or eyelink latency (see Table 12).

Positive versus neutral block. For the next series of ANOVAs, I tested for difference during presentation of the second block of positive stimuli, with group (bipolar, control) as a between subjects factor, and timing (2500ms, 4500ms) and valence (positive, neutral) as the two within subjects factors (see Table 13).

SCL tonic levels. In the first ANOVA, I examined SCL tonic levels. There was a significant valence effect, $F(1,38) = 11.69$, $p = .002$, Cohen's $d = .08$, in which SCL tonic levels were greater during the positive IAPS set ($M = 7.01$, $SD = 4.38$) as compared to the neutral set ($M = 6.67$, $SD = 4.19$). There was a significant timing effect, $F(1,38) = 5.61$, $p = .023$, in which SCL tonic level was greater when the acoustic probe was presented at 2500ms ($M = 6.88$, $SD = 4.28$), as compared to at 4500ms ($M = 6.78$, $SD = 4.27$). Thus, while there were no group differences, SCL tonic levels were greater during presentation of the positive stimuli, when compared to the neutral stimuli, and were greater when the probe was presented sooner rather than later.

Eyeblink amplitude. In the next ANOVA, I examined eyeblink amplitude. There was no significant group main effect, or main effect, or interaction effects.

Eyeblink latency. In the next ANOVA, I examined eyeblink latency. There was a significant timing effect, $F(1,37) = 3.96$, $p = .05$, Cohen's $d = .43$, such that eyeblink

latency was longer when the acoustic probe was presented at 2500ms (*M* = .90, *SD* = .35), compared to 4500ms (*M* = .76, *SD* = .30).

In sum, there were no group differences in eyeblink amplitude or eyeblink latency after the mood induction. In support of my hypothesis, while control participants displayed greater SCL levels when presented with negative stimuli after the mood induction, compared to when presented with neutral stimuli, BD participants did any difference in SCL when presented with the negative stimuli after the mood induction.

Combining pre-manipulation and post-manipulation data. A complete picture of the effect of the mood manipulation requires examining differences between responses before the manipulation and responses after the manipulation. The full data set is quite complicated, however, involving repeated assessment of responses to neutral stimuli, in addition to valenced stimuli. In order to reduce the size of the design, it would be desirable to combine responses to all neutral stimuli before and after the manipulation. If this could be done, the full data set could be treated as a 2 (group) by 2 (pre, post) by 3 (positive, neutral, and negative) design.

To test the feasibility of doing this, a series of analyses was conducted to assess the similarity of responses to neutral stimuli before and (separately) after the manipulation. These analyses included group as a between subjects factor, to allow for the possibility that variation in reaction might differ by group; and timing (2500ms, 4500ms) and block (block 1[neutral stimuli following the initial block of negative stimuli], block 2 [all neutral block], block 3 [neutral stimuli following the initial block of positive stimuli]) as the two within-subjects factors.

Blocks 1 to 3 prior to mood induction. In the first series of ANOVAs, I examined reactions to the neutral picture stimuli in blocks 1 to 3, the blocks before the mood induction (see Table 14).

SCL tonic levels. In one ANOVA, I tested for difference in SCL. There was a significant main effect of block, $F(2,72) = 6.05$, $p = .004$. I conducted post-hoc analyses to explore differences between the blocks with paired samples *t*-tests. Results yielded no significant difference between block 1 ($M = 7.23$, $SD = 4.04$) and block 2 ($M = 6.88$, $SD = 3.15$), $t(38) = 1.16$, *ns*. However, SCL tonic level was greater in block 1 ($M = 7.23$, $SD = 4.04$), as compared to block 3 ($M = 6.31$, $SD = 4.11$), $t(37) = 3.67$, $p = .001$. While SCL tended to be greater in block 2 ($M = 6.88$, $SD = 3.15$), as compared to block 3 ($M = 6.31$, $SD = 4.11$), this difference was not significant, $t(37) = 1.99$, *ns*.

Eyeblink amplitude. In the following ANOVA, I tested for difference in eyeblink amplitude. There was a significant main effect of block, $F(2,74) = 4.00$, $p = .022$. I conducted post-hoc analyses on the block effect using three paired samples *t*-tests with Bonferroni correction significance *p*-values set at .05/3. Analyses indicated that eyeblink amplitude was greater in block 1 ($M = 4.94$, $SD = .94$), as compared to block 2 ($M = 4.60$, $SD = 1.01$), $t(39) = 3.04$, $p = .004$, but not significant when comparing block 1 ($M = 4.94$, $SD = .94$) to block 3, ($M = 4.82$, $SD = .92$) , $t(38) = 1.01$, *ns*. There was no significant difference when comparing eyeblink amplitude in block 2 ($M = 4.60$, $SD = 1.01$) to block 3 ($M = 4.82$, $SD = .92$), $t(39) = -1.83$, *ns*.

Eyeblink latency. In the next ANOVA, I examined eyeblink latency. There were no significant group main effect, main or interaction effects.

Blocks 4 to 6 after the mood induction. The second series of ANOVAs examined reactions to the neutral picture stimuli in blocks 4 to 6, the blocks after the mood induction (see Table 15).

SCL tonic levels. In testing for difference in SCL tonic levels, there was a significant block effect, $F(2,76) = 10.50$, $p < .001$. Next, I conducted post-hoc analyses on the block effect using three paired samples *t*-tests with a Bonferroni correction *p*-value set at .05/3 to test for differences between the blocks. Findings indicated no significant difference between block 4 ($M = 7.45$, $SD = 4.28$) and block 5 ($M = 7.36$, $SD = 4.28$), $t(39) = 0.42$, *ns.* SCL tonic level was greater in block 4 ($M = 7.45$, $SD = 4.28$), as compared to block 6 ($M = 6.64$, $SD = 4.19$), $t(39) = 4.07$, $p < .001$, and was also greater in block 5 ($M = 7.36$, $SD = 4.28$) as compared to block 6 ($M = 6.64$, $SD = 4.19$), $t(39) = 4.34$, $p < .001$.

Eyeblink amplitude. In testing for difference in eyeblink amplitude, an ANOVA revealed a block effect, $F(2,78) = 6.14$, $p = .003$. Follow-up analyses on the block effect used three paired-samples *t*-tests to compare the blocks with a Bonferroni *p*-value set at .05/3. In comparing: block 4 ($M = 5.09$, $SD = 1.15$) to block 5 ($M = 4.97$, $SD = 1.16$), there was no significant difference, $t(40) = 0.94$, *ns*; block 4 to block 6 ($M = 4.87$, $SD = .98$), there was no statistically significant difference, $t(41) = 1.68$, *ns*; in block 5 to block 6, there was no significant difference, $t(41) = 0.43$, *ns*.

Eyeblink latency. With the next ANOVA, I examined eyeblink latency. There was no significant group main effect, main or interaction effects.

In sum, analyses into the pattern of physiological responding for the neutral IAPS sets in blocks 1-3 and blocks 4-6 suggest that there are a few differences among

responses to neutral stimulus sets, but the differences were not very systematic. Keeping these differences in mind, I proceeded to examine the pre-manipulation and post-manipulation data together.

Examining pre-manipulation and post-manipulation data together. For these analyses, all three neutral blocks pre-manipulation were averaged by time, and all three neutral blocks post-manipulation were averaged by time. These analyses included, group (BD, control) as a between-subjects factor, to allow for the possibility that variation in reaction might differ by group; and pre-post (before the mood induction, after the mood induction), valence (negative, neutral, positive), and time (2500ms, 4500ms) as the three within-subjects factors.

SCL tonic levels. In the first repeated measures ANOVA, I investigated difference in SCL tonic levels (see Table 16). There was a significant valence effect, $F(2,72) = 22.85$ $p < .001$, but no significant interaction between valence and group, $F(2,72) = 0.16$, *ns*. There was a significant time effect, $F(1,36) = 10.11$, $p = .003$, such that SCL was greater when the acoustic probe was presented at 2500ms ($M = 7.41$, $SD = 3.84$) than when presented at 4500ms ($M = 7.30$, $SD = 4.08$). In conducting post-hoc analyses on the main effect of valence, I used three paired-samples *t*-tests with a *p*-value set at .05/3. SCL tonic levels were greater in the negative set ($M = 8.34$, $SD = 4.18$), as compared to the neutral set ($M = 7.12$, $SD = 3.85$), $t(37) = 6.38$, $p < .001$, and greater in the negative set ($M = 8.34$, $SD = 4.18$), as compared to the positive set ($M = 7.07$, $SD = 4.25$), $t(38) = 4.84$, $p < .001$. There was no significant difference in SCL tonic levels when comparing the neutral set and the positive set, $t(37) = 0.35$, *ns*.

There was a significant interaction among pre-post, valence, and group, $F(2,72) = 3.77$, $p = .028$. To partition the significant three-way interaction (Cohen, 2007), I first ran a between-within ANOVA with group as a between-subjects factor, and valence measures before to the mood induction (negative, neutral, positive) as the within-subjects factors. There was no significant group main effect, $F(1,36) = 0.69$, *ns*. There was a significant valence effect, $F(2,72) = 12.46$, $p < .001$, but no significant interaction between valence and group, $F(2,72) = 1.19$, *ns*. Next, I ran a one-way ANOVA with group as a between-subjects factor, and valence measures after to the mood induction (negative, neutral, positive) as the within-subjects factors. There was no significant group main effect, $F(1,38) = 2.50$, *ns*. There was a valence effect, $F(2,76) = 23.45$, $p < .001$, but no significant interaction between valence and group, $F(2,76) = 1.76$, *ns*.

As both the ANOVAs revealed valence effects, which mirrored the main valence effect. In reviewing the means, I conducted follow-up comparisons with twelve paired sample *t*-tests with a Bonferroni p-value set at .05/12, comparing valence blocks before and after the mood induction first only for the control participants and then only for the BD participants. I tested whether for control, and then BD, participants if there was a difference in SCL for: 1) the negative set before the mood induction, when compared to the neutral set before the mood induction, 2) the negative set before the mood induction, when compared to the positive set before the mood induction, 3) the positive set before the mood induction, as compared to the neutral set before the mood induction, 4) the negative set after the mood induction, as compared to the neutral set after the mood induction, 5) the negative set after the mood induction, as compared to the positive set after the mood induction, and 6) the positive set after the mood induction, as compared to

the neutral set after the mood induction. I will only discuss significant findings, and highlight differences between the two groups.

One comparison found a significant difference, $t(21) = 4.56$, $p < .001$, such that SCL was greater in the negative set ($M = 8.32$, $SD = 4.15$), as compared to the neutral set prior to the mood induction ($M = 7.32$, $SD = 3.80$), for control participants. There was no significant difference, $t(15) = 3.26$, ns, when comparing the negative set ($M = 7.54$, $SD = 4.17$) before the mood induction to the neutral set ($M = 6.21$, $SD = 3.44$) before the mood induction for BD participants.

Another comparison found a significant difference, $t(21) = 4.49$, $p < .001$, such that SCL was greater in the negative set after the mood induction ($M = 9.50$, $SD = 4.94$) as compared to the neutral set after the mood induction ($M = 7.99$, $SD = 4.44$) for control participants. There was no significant difference, $t(17) = 3.31$, $p = .004$, when comparing the negative set after the mood induction ($M = 6.92$, $SD = 3.84$) and the neutral set after the mood induction ($M = 6.12$, $SD = 3.74$).

Another comparison found a significant difference, $t(21) = 3.76$, such that SCL was greater in the negative set after the mood induction, as compared to the positive set after the mood induction ($M = 7.89$, $SD = 4.83$) for control participants. As well, there was also a significant difference, $t(17) = 3.53$, $p = .003$, such that SCL was greater during the negative set ($M = 6.92$, $SD = 3.84$), as compared to the positive set ($M = 5.34$, $SD = 3.61$) for BD participants.

Findings from this set of post-hoc analyses suggest that while there were differences in SCL tonic levels when comparing the negative and neutral set before and after the mood induction for control participants, there was no such difference for BD

participants. That is, while control participants displayed greater SCL tonic levels during presentation of the negative IAPS set, as compared to when presented with the neutral set, this pattern of response did not occur for BD participants.

There was a significant interaction between valence and time, $F(2,72) = 4.30$, $p = .017$. In conducting post-hoc analyses on the interaction between valence and time, I used seven paired samples *t*-tests to determine if 1) there was a significant difference as a function of timing in the negative IAPS set, then 2) investigated if there was difference in timing in the neutral set, and then 3) investigated if there was a difference in timing in the positive set, and then 4) tested if there was a difference in valence comparing the negative and neutral sets when the acoustic probe was presented at 2500ms, and then 5) tested if there was a difference in valence comparing the negative and neutral sets when the acoustic probe was presented at 4500ms, and then 6) tested if there was a difference in valence comparing the positive and neutral sets when the acoustic probe was presented at 2500ms, and last 7) tested if there was a difference in valence comparing the positive and neutral sets when the acoustic probe was presented at 4500ms. The first comparison found that SCL tonic levels were greater, $t(38) = 4.57$, $p < .001$, when the acoustic probe was presented at 2500ms ($M = 8.38$, $SD = 4.19$), as compared to when presented at 4500ms ($M = 8.09$, $SD = 4.17$), in the negative set. The second comparison found no significant difference, $t(37) = -0.04$, *ns*, in SCL levels when the acoustic probe was presented at 2500ms ($M = 7.12$, $SD = 3.66$) or at 4500ms ($M = 7.12$, $SD = 4.05$) in the neutral set. The third comparison found no difference, $t(39) = 3.14$, *ns*, when the probe was presented at 2500ms ($M = 6.96$, $SD = 4.30$) or at 4500 ($M = 6.72$, $SD = 4.24$) in the positive set. The fourth comparison found that SCL was greater, $t(37) = 6.30$, $p < .001$, in

the negative, as compared to the neutral set, when the probe was presented at 2500ms. The fifth comparison found that SCL was greater, $t(37) = 6.00$, $p < .001$, in the negative set, as compared to the neutral set, when the probe was presented at 4500ms. The sixth comparison found no difference, $t(37) = -0.39$, *ns*, in SCL when comparing the positive set to the neutral set, when the probe was presented at 2500ms. The seventh comparison found no difference, $t(37) = 1.66$, *ns*, when comparing the positive set to the neutral set, when the probe was presented at 4500ms.

Eyeblink amplitude. I then explored eyeblink amplitude (see Table 17). There was a significant pre-post effect, $F(1,36) = 6.07$, $p = .019$, such that eyeblink amplitude was greater in the blocks after the mood induction ($M = 5.16$, $SD = .95$), compared to the blocks before the mood induction ($M = 4.97$, $SD = .85$). There was a significant valence effect, $F(2,72) = 13.61$, $p < .001$. In conducting post-hoc analysis on the valence effect, I used three paired samples *t*-tests, with a Bonferroni correction *p*-value set at .05/3. Eyeblink amplitude was greater in the negative blocks ($M = 5.31$, $SD = 1.01$), compared to the neutral blocks ($M = 4.97$, $SD = .82$), $t(37) = 4.40$, $p < .001$, and greater in the negative blocks, as compared to the positive blocks ($M = 4.80$, $SD = .99$), $t(42) = 4.79$, $p < .001$. While eyeblink amplitude tended to be greater in the neutral block, as compared to the positive block, this difference was not statistically significant, $t(37) = 0.42$, *ns*.

Eyeblink latency. In the third repeated measures ANOVA test, I examined eyeblink latency. There was no significant group main effect, main or interaction effect (see Table 18).

Supplemental Analyses. I also investigated if severity of BD was related to eyeblink amplitude or latency for the different valenced IAPS stimuli. The severity of the

illness was operationalized as the number of depressive or manic episodes within a participant's lifetime. I also investigated if years of illness were related to the eyeblink amplitude or latency for the different valenced IAPS stimuli. Years of illness was operationalized as age of last episode (mania or depression) minus age of onset (mania or depression).

Illness factors. For BD participants, a greater number of manic episodes was correlated with a slower eyeblink latency reaction time during the positive IAPS set after the mood induction when presented with the 2500ms probe, $r(19) = 0.52$, $p = .022$. For BD participants, a greater number of episodes of depression correlated with both a lower eyeblink amplitude during the first all-neutral set when the acoustic probe was presented at 2500ms, $r(16) = -0.51$, $p = .044$, and in the neutral block after the second positive IAPS set (block 6) when the probe was presented at 4500, $r(17) = -0.56$, $p = .02$. A greater number of episodes of depression correlated with a slower eyeblink latency time during the all-neutral set after the mood induction when the probe was presented at 4500ms, $r(19) = 0.48$, $p = .04$, and in second positive IAPS set when the probe was presented at 2500ms, $r(19) = 0.50$, $p = .028$.

Current symptom severity. Next, I investigated if current mania or depression symptom severity (i.e., MASQ-Anhedonia Depression, MASQ-Somatic Anxiety, ASRM, BRMS, Ham-D) was related to eyeblink amplitude or eyeblink latency during presentation of the different valenced IAPS stimuli. These analyses included only the BD sample. For BD participants, a greater number of depressive symptoms on the MASQ-Anhedonia Depression subscale correlated with lower eyeblink amplitude in: the first neutral block after the negative set when the probe was presented at 2500ms, $r(18) = -$

0.57, $p = .013$, in the first positive block when the startle probe was presented at 4500ms, $r(19) = -0.50$, $p = .028$, and in the second positive IAPS set when the acoustic probe was presented at 4500ms, $r(19) = -0.47$, $p = .045$. A greater number of depressive symptoms on the MASQ-Anhedonia Depression subscale correlated with greater eyeblink latency in the first negative set when the probe was presented at 4500ms, $r(20) = 0.70$, $p < .001$, and in the all-neutral block after the mood induction when the probe was presented at 2500ms, $r(20) = 0.56$, $p = .011$.

For BD participants, a greater number of anxiety symptoms on the MASQ-Somatic Anxiety subscale was correlated with greater eyeblink latency in the all-neutral set after the mood induction, $r(20) = 0.49$, $p = .03$.

A greater number of mania symptoms on the BRMS was correlated with greater eyeblink amplitude both in the first all-neutral block when the acoustic probe was presented at 2500ms, $r(18) = 0.56$, $p = .015$, and in the all-neutral set after the mood induction when the probe was presented at 2500ms, $r(19) = 0.59$, $p = .008$, and at 4500ms, $r(19) = 0.48$, $p = .038$. There was also a significant negative correlation between a higher score on the BRMS and eyeblink latency, $r(20) = -0.73$, $p < .001$, such that those with a greater number of mania symptoms displayed shorter eyeblink latency time on the neutral set after the first positive set when the probe was presented at 4500ms..

Self-report mood measures. Next, I investigated the relationship between self-report SAM mood measures and physiological responses in startle eyeblink response and SCL tonic level. There were no significant relationships for BD participants. For control participants, those with a greater self-reported positive mood on the SAM displayed less eyeblink amplitude on the first negative set when the probe was presented at 2500ms,

$r(24) = -0.47$, $p = .019$, and on the all neutral set, block 2, prior to the mood induction, during the 2500ms probe, $r(24) = -0.45$, $p = .029$.

Testing the mechanism of eyeblink startle response to acoustic probe. Previous studies have reported greater eyeblink startle amplitude response when paired with later presentations of the acoustic startle probe, while viewing negative valenced pictures (Smith et al., 2005). I predicted that control participants would display greater eyeblink amplitude in the negative IAPS sets with later presentations of the acoustic probe, as compared to earlier presentations of the acoustic probe. I expected no pattern of increasing eyeblink amplitude for the BD participants. In a series of ANOVAs, I tested difference in eyeblink amplitude and latency, with timing of probe (2500ms, 4500ms) and pre-post (negative block before mood induction, negative block after mood induction) as the two within-subjects factors, and group (bipolar, control) as the between-subjects factor. There were no significant group main effect, main or interaction effects for eyeblink amplitude or eyeblink latency (see Table 19).

In further exploring a lack of linear responding pattern for control participants, I investigated differences in self-reported SAM Mood and Arousal ratings in the first and second negative blocks. When comparing to overall negative arousal mean rating ($M = 6.00$, $SD = 2.21$) for the negative stimuli used in this study (please see Appendix A), control participants reported experiencing less arousal in the first negative block, $t(24) = -2.59$, $p = .016$, and also reported experiencing less arousal in the second negative block after the mood induction, $t(24) = -3.84$, $p = .002$. When comparing the overall mean rating for the negative stimuli ($M = 2.83$, $SD = 1.69$), control participants also reported feeling less negative mood in the first negative block, $t(24) = 8.21$, $p < .001$, and in the

second negative block, $t(24) = 11.13$, $p < .001$. Engagement in the affective-modulated startle response is dependent upon experiencing both high valence *and* high arousal (Bradley et al., 2006; Bradley & Lang, 2007; Cuthbert et al., 1996; Lang, 1990), and a significant difference in self-reported mood and arousal ratings may point to issues with the stimuli to elicit a level of mood and arousal for a response.

Chapter 4: Discussion

The goal of the current study was to test if there was a difference between individuals with BD and control participants in physiological responses to valenced stimuli both before and after a mood induction. To address this aim, I gathered diagnostic, self-report mood ratings and physiological data on BD and control participants. Participants viewed a series of pictures (negative, positive, and neutral), then underwent a cognitive speed task validated to increase positive mood, and then viewed another series of valenced pictures. An acoustic probe was presented at varying times (2500ms, 4500ms) during the picture presentation to assess affective modulation of the startle response by emotionally salient stimuli. In the following section, I will explore the study findings, discuss study limitations and strengths, and conclude with clinical implications and future directions.

I will first explore whether the paradigm showed anticipated effects for timing and valence for SCL tonic level, eyeblink startle amplitude, and eyeblink latency. As predicted, there was a significant effect for valence such that SCL tonic levels were significantly greater during presentation of the negative block, when compared to both the positive or neutral IAPS blocks, both before and after the mood induction. In addition, SCL tonic levels were greater in the positive set, as compared neutral IAPS set, both before and after the mood induction. After the positive mood induction, SCL tonic levels were greater overall during presentation of all of the blocks, as compared to the blocks prior to the mood induction. There were also timing differences, such that SCL tonic levels were greater when the probe was presented at 2500ms, compared to at 4500ms, in both the negative and positive IAPS sets. When comparing the interaction

effects of valence on the timing of the acoustic probe, SCL tonic levels were greater in the negative set, compared to the neutral set, when acoustic probes were presented at both the 2500ms and 4500ms times. These findings suggest that SCL tonic levels reflect overall arousal reactivity to high valenced stimuli (Bradley et al., 1988; Bradley & Lang, 2000; Codispotti et al., 2001; Gomez and Danuser, 2004).

Current theory suggests that viewing affective stimuli will engage the motivational system. When the acoustic probe is presented in conjunction with an unpleasant foreground, the affect-modulated startle reflex is amplified. Conversely, when the acoustic tone is paired with a pleasant foreground, the affect-modulated startle reflex is attenuated (Lang, 1995; Lang et al., 1990). In this study, eyeblink amplitude was greater after the positive mood induction. When exploring eyeblink amplitude differences among the valences, eyeblink amplitude was greater during presentation of the negative stimuli and attenuated during the presentation of the positive and neutral stimuli.

Normative prediction would be for a linear pattern of responding for the startle response across valences (greater eyeblink amplitude with later acoustic presentations in the negative set, when compared to the neutral set, and greater eyeblink amplitude response with later acoustic presentations in the neutral set, when compared to the positive set). This pattern was found in previous studies (Giakoumaki et al., 2010; Greenwald et al., 1989; Lang et al., 2003; Smith et al., 2006), but it was not found in control participants for this study.

It was also anticipated that control participants would display a faster blink time with later presentations of the acoustic probe when paired with a negative valenced picture, and a slower blink time with later presentations of the acoustic probe when

paired with a positive valenced picture (Lang et al., 1990; Smith et al., 2005; Vrana et al., 1988). In contrast to this, participants displayed slower blinks to the acoustic probe at 4500ms in the negative set, as compared to the neutral set. As well, participants displayed faster blink time to the acoustic probe at 4500ms in the second positive set, as compared to at the 2500ms time. These response patterns are contrary to my predictions and to previous theoretical conceptualizations of the affect-modulated startle response in healthy control studies.

Lack of the expected linear pattern of response may be due to a failure of the IAPS stimuli to elicit an adequate amount of hedonic activation and arousal in the participant. Specifically, the affect-modulated startle response is based upon elicitation of both high valence *and* high arousal in participants. Control participants in this study reported feeling less aroused and less negative mood from the picture set, as compared to participants from the original validation study (Lang et al., 1999). Indeed, there have been findings of high coherence among measures of self-report of emotional experience, facial behavior, and physiological response to highly valenced emotions, but low coherence among measures for low valenced and low arousal emotions (Maus et al., 2005). Although the response pattern to negative stimuli has been widely replicated, there have been fewer robust findings reporting a pattern of theorized response to positive stimuli. Several studies have reported no difference in eyeblink startle response to positive stimuli, when compared to neutral stimuli (Cook et al., 1991; Jackson et al., 2000; Jansen & Frijda, 1994; Witvliet & Vrana, 2000). Interestingly, there is recent evidence which suggests that interpretation can influence the affect-modulated startle response. Findings from one study indicated that eyeblink amplitude increased during

self-relevant positive imagery, as compared to standard IAPS positive stimuli and also compared to non-relevant standard positive imagery (Miller et al., 2002).

In sum, the different SCL findings from valence and timing effects suggest that the paradigm was able to invoke high arousal in participants. Differences in eyeblink amplitude with the presentation of valenced stimuli suggest hedonic activation in participants. However, there was a lack of linear startle response with the timing of the acoustic probe. Overall, some findings from this study do suggest that this paradigm was able to successfully elicit appropriate physiological responses to affective stimuli. In the following section, I will discuss group difference between BD and control participants on physiological responses to valenced stimuli before and after the mood induction.

Hypothesis 1

My first hypothesis was that there would be no group difference in physiological responses (SCL tonic levels, eyeblink amplitude, eyeblink latency) and self-report mood measures (Mood Grid, SAM mood measures) before a mood induction. Analyses indicated no difference between groups on the initial Mood Grid (confidence, irritability, talkativeness) or SAM Mood measures. There was no reported difference in SAM mood ratings between groups after any block of presentation of the valenced IAPS sets. This finding, which was anticipated for this study, adds to the body of literature confirming that BD and control participants self-report similar ratings of emotional experience of affective stimuli (Gruber, Eidelman, et al., 2001; Gruber, Harvey et al., 2011)

When exploring physiological reactivity prior to the mood induction, there was no group difference in SCL tonic level for any of the valenced stimuli or timed effects of the acoustic startle probe. Participants with BD displayed greater eyeblink amplitude in the

neutral IAPS set in block 3 (positive then neutral) at the 4500ms acoustic probe, as compared to presentations of the acoustic probe at 2500ms. There was no difference in eyeblink amplitude in control participants when comparing timing effects in the neutral IAPS set in block 3. Our results are consistent with findings in one study in which BD participants displayed greater eyeblink amplitude during presentation of the neutral stimuli, when compared to controls (M'Bailara et al., 2009). There was no group difference in eyeblink latency. As there are only currently two studies on the affect-modulated startle response in euthymic BD participants, these findings should be interpreted with care.

Hypothesis 2

The second study hypothesis rested upon the success of the positive mood induction for study participants. However, a substantial percentage of participants reported no positive mood change. When investigating differences between those who reported a mood change as compared to those who did not report a mood change, participants from the control group who reported no positive mood or a negative mood from the mood induction were more likely to endorse a greater number of self-reported depression symptoms on the MASQ Anhedonia Depression subscale than control participants who reported a positive mood. It should be noted that no control participant met criteria for current or a past MDE. There was no significant difference in any demographic or symptom severity variables when comparing BD participants who reported a positive mood change as compared to BD participants who did not report a mood change. It may be that the more symptomatic participants had difficulty engaging in a positive mood induction. Because of this failure of the mood induction, analyses of

post-induction data were conducted including only those participants who reported a positive mood post-induction.

I predicted that participants with BD would be less reactive than control participants --with lower eyeblink amplitude, slower eyeblink latency, and lower SCL tonic levels-- to the negative and neutral IAPS stimuli after the positive mood induction. Moreover, I hypothesized that participants with BD would exhibit lower startle amplitude, slower latency, and greater SCL tonic level, when compared to control participants, during presentation of positive stimuli.

As anticipated, BD participants had lower SCL tonic levels to the negative stimuli after the mood induction, compared to control participants. Contrary to my hypothesis, however, there was no group difference in eyeblink amplitude or in eyeblink latency. In sum, while there were group differences in SCL tonic levels, there was no group difference in eyeblink startle reactivity.

In my supplemental analyses, I explored if other factors such as illness severity and psychiatric symptoms were related to physiological response to threat. I first explored the relationship between the number of mood episodes (mania, depression, anxiety) and startle eyeblink reactivity. For BD participants, a greater number of depressive episodes correlated with a lower eyeblink amplitude in the all-neutral set after the mood induction, and greater eyeblink latency in both the all-neutral set and positive IAPS set after the mood induction. Taken together, the lower eyeblink amplitude and slower eyeblink time suggests that BD participants with a greater number of depressive episodes appeared to perceive neutral stimuli more positively.

The two studies of startle response with euthymic BD samples on emotional reactivity to valenced stimuli have reported mixed results. Findings from one study suggested that BD participants displayed an overall blunted response both to positive and negative stimuli, when compared to control participants (Giakoumaki et al., 2010). Findings from the second study indicated an attenuated response only to negative stimuli for BD participants, when compared to MDD and control participants (Forbes et al., 2005). It has been suggested that a blunted response may be influenced more by depression symptoms—which parallel a blunted response pattern found in startle reactivity in unipolar depressed individuals (Gruber, 2011; Rottenberg et al., 2002).

BD participants who reported a greater number of manic episodes had slower eyeblink latency during the positive set following the mood induction. The slower eyeblink latency suggests that the BD participants with a greater number of manic episodes were more reactive to the positive stimuli than BD participants with less number of manic episodes. When applying this patterned response to other paradigms, our results are consistent with findings in a 2007 study by Putman et al. that investigated automatic response to threatening stimuli in a facial recognition task with those at-risk for BD (i.e., high scores on the GBI) and healthy controls. The authors reported that at-risk participants displayed less orienting to fearful faces, and increased orienting to happy faces, while healthy controls displayed the normal pattern of increasing attention to threatening faces. The selective attention to positive stimuli for those at-risk may be related to the activation of the appetitive motivational state for rewarding cues, at the disregard of threatening cues.

When exploring the relationship between current symptoms and emotional response in my sample of participants with BD, a greater number of depressive symptoms on the MASQ Anhedonia Depression subscale were correlated to lower eyeblink amplitude to the neutral block after the first negative set and to the neutral set following the first positive set. Depression symptoms also correlated with a longer latency in the first negative set and in the all-neutral set after the mood induction. These findings parallel the extensive literature on depression symptoms in MDD and the eyeblink startle reflex, and the broader evidence which suggests depression is characterized by dysfunctions in responses to affective stimuli. Studies with MDD participants suggest that a greater number of depression symptoms were related to overall blunted startle reactivity to both positive and negative stimuli in pictures (Allen et al.,1999; Sloan & Sandt, 2010) and in affective film clips (Kaviani et al., 2004).

For BD participants in this study, a greater number of mania symptoms on the BRMS correlated with greater amplitude in the all-neutral set before and after the mood induction. Interestingly, a greater number of mania symptoms were correlated with a shorter reaction time in the neutral set after the first positive IAPS set. The greater eyeblink amplitude response in the neutral set after the second negative IAPS set suggests that BD participants with a greater number of mania symptoms may have difficulties down-regulating negative emotions.

While BD participants in this study were euthymic, a significant correlation between physiological response and history of mood episodes suggests that there is a patterned relationship between emotional response to positive emotions and the course of illness in BD. Findings from a few recent studies suggest that participants with BD may

exhibit continued responses to emotional stimuli, even after the stimuli is no longer present (Forbes et al., 2005). Forbes et al. argued that participants with BD may engage in strategies to maintain the affective experience of the valenced stimuli, which may influence the later startle response. Indeed, BD participants in another study were found to have sustained positive emotions that extended across negative and non-valenced contexts (Gruber, Harvey, et al., 2011). This pattern of heightened peak arousal with an attenuated return to baseline over time may point to the larger picture of an inability to modulate positive emotion. In relating continued responding to emotional stimuli to BD, BD participants who were more likely to ruminate about negative and positive emotions, as compared to control participants, reported a greater number of manic episodes (Gruber, Eidelman, et al., 2011).

Moreover, within this group, a greater number of anxiety symptoms from the MASQ Somatic Anxiety subscale correlated with greater eyeblink latency in the all-neutral set after the mood induction. Endorsement of anxiety symptoms was not surprising, given the high rates of co-morbidity between anxiety disorders and BD (Bellani et al., 2012). It is important to note, however, that no study has directly investigated the role of anxiety in physiological response in BD, and thus this study's findings on anxiety in BD and physiological response to emotional stimuli are exploratory.

Limitations

The limitations in my study include issues with study methodology, recruitment bias, and validity of the IAPS stimuli. One study limitation was the percentage of participants who had an increase in positive mood after the mood induction procedure---

which raises concerns about whether there was sufficient power to detect a difference between groups in startle reactivity after the mood induction. I therefore conducted a power analysis with the program *G*Power* (Faul et al., 2007) to find out whether my study had enough statistical power to detect group differences in eyeblink startle response during the negative valenced set after the mood induction. With a target effect size at .20 (i.e., a small size effect, according to Cohen's 1977 effect size conventions), the power to detect a group effect based upon the existing sample size was determined to be 0.16.

Issues with power may also be related to a percentage of participants who reported no change in mood, or even a negative mood, after the mood induction. For some BD participants, there may be one explanation for the failure of the mood induction. There is growing evidence that some individuals with BD are attempting to down-regulate positive emotions in order to prevent the emergence of mania symptoms (Feldman et al., 2008). However, as I did not assess for this strategy in this study, it is unclear if BD participants were engaging in this strategy in order to help disengage from affective stimuli so as to circumvent a heightened emotional experience. The four control participants who reported a negative mood or no change from an initial negative mood after the mood induction also differed in the number of symptoms endorsed on the MASQ Anhedonia Depression subscale. It may be that those participants were experiencing depression symptoms which made it difficult to induce a positive mood (Grusser et al., 2007). Another alternative explanation may be that the Pronin task may have had an undesirable secondary effect of eliciting a negative or mixed response due to the verbal fluency and processing speed that the task required. The mood induction was validated on a college-aged sample without any risk for psychopathology from a

university where participants' processing speed and verbal fluency may have differed from this study's sample. In Pronin and Wegner's 2010 study on the mood induction, the authors reported that the main effect of positive mood was mediated by the participant's subjective experience of the thought speed. Therefore, it may be that participants needed to identify the fast speed of reading the statements as a positive and enjoyable experience. In this study, the small subset of the sample that reported a greater negative mood after the mood induction may have found the mood induction a challenging and negative experience.

Another limitation that should be noted is the potential recruitment bias. It was challenging to recruit euthymic participants with stable sub-clinical symptoms, even though there are numerous studies reporting that inter-episode symptoms endure (Swann et al., 2001) and that subsyndromal mania and hypomania symptoms are three times more likely to occur than symptoms at the clinical threshold for an episode of mania (Judd et al., 2002). However, findings suggest that euthymic individuals with BD display heightened reactivity to positive mood (Farmer et al., 2006; Gruber et al., 2008), engage in risky decision-making (Johnson, Ruggero, & Carver, 2005; Murphy et al., 2001), and engage in cognitive strategies to sustain a positive mood (Feldman et al., 2008); thus, investigating mild mood shifts is crucial to understanding traits based reactivity to threat in BD. There were also concerns surrounding the validity of euthymia status, medication regimen, and co-morbidities, since symptoms were self-reported without verification from a secondary source (e.g., a family member or a treatment provider). However, mania, depression, and anxiety symptoms were assessed via both clinician and self-report scales in order to address this issue. A second source of potential bias was that all

diagnostic assessments were completed by one interviewer, which raises the concern of biases in diagnosis. A third and final source of potential bias was that BD and control groups were not matched on current psychiatric symptoms and diagnostic history. BD participants endorsed more anxiety symptoms on the MASQ Somatic Anxiety subscale, and there were a greater percentage of BD participants, than control participants, who endorsed a lifetime history of anxiety disorders, specifically, GAD and panic disorder, and alcohol or substance abuse. Thus, null findings on group differences could be attributed to a control group that was less symptomatic and reported a less co-morbid psychiatric history.

The inconclusive findings from this study may be due to the failure of the IAPS stimuli to elicit an appropriate hedonic and arousal response in participants. Control participants reported less arousal and less negative mood when presented with the negative stimuli, as compared with participants in the original IAPS validation study. However, every attempt had been made to select a set of validated negative IAPS pictures that had elicited significant effects in startle response in previous studies (Lang et al, 1993; Smith et al., 2005; Vrana, 1996). I chose pictures from a deck of pictures in the moderate range for arousal and valence. Findings from studies using IAPS stimuli suggest that that electrodermal activity varies with emotional intensity and arousal levels, with larger responses elicited in highly arousing context, either unpleasant or pleasant (Balconi et al., 2011; Bernat et al., 2006; Bradley & Lang, 2000; Cuthbert et al., 1996; Lang et al, 1993; Miller et al., 2002). In addition, while the images used in this study were standardized and validated in other studies as eliciting emotional responses, the findings may not be entirely generalizable to threat detection in real-life situations.

The affect-modulated startle response is based upon the pairing of an aversive acoustic startle to an affective foreground. Depending on if one is in a defensive or appetitive motivational state, the aversive acoustic startle will either amplify or inhibit a startle response. However, the acoustic probe must be viewed as aversive in order to facilitate the affect-modulated startle response. Generally, a more intense tone is seen as more aversive. The acoustic startle probe utilized in this study was in the 95 dB range---which could have impacted detectable differences in startle reactivity. Most studies with healthy control participants reported positive effects when the acoustic startle probe was paired with IAPS stimuli in the 103-110 dB range (Bernat et al., 2006; Bradley et al., 2006; Vaidyanathan et al., 2009). Studies using acoustic probes with pediatric BD participants have presented acoustic probes at 95dB (Rich et al., 2005), while studies with adult BD participants have ranged in presentation of probes from 95dB (Carroll et al., 2007) to 100dB (Forbes et al., 2005) and 104dB (Giakoumaki et al., 2010; Iacono et al., 1984). In a study investigating the role of acoustic probe intensity, Cuthbert et al. (1996) reported that more intense probes were related to greater eyeblink amplitude responses during presentation of the negative pictures. However, Cuthbert et al. did report that for each probe intensity (80dB, 95dB, 105dB), eyeblink amplitude still increased when the probe was paired with high-arousal, high-valenced pictures.

A final limitation is that the process of habituation may have influenced physiological findings. To address the issue of habituation to the IAPS pictures, participants were presented with non-repeating stimuli within similar reported arousal and valence ratings. Other researchers have attempted to further explore the issue of habituation to valenced pictures. In a 2000 study by Larson et al., participants were asked

either to view the same set of IAPS stimuli 4 weeks apart, or view two different sets of IAPS stimuli, with similar valence and arousal ratings, 4 weeks apart, in order to investigate the test-retest stability of the affect-modulated startle response. Findings indicated that there was moderate stability for the negative, compared to neutral, and positive, as compared to neutral, responses only for those participants who viewed two separate sets of IAPS stimuli.

Strengths of the Study

This study had multiple strengths. There has been no other study specifically investigating physiological reactivity to threat after a positive mood induction---a mechanism that could parallel a similar process of emotion-based impulsivity during mania for individuals with BD. Furthermore, many studies have recruited at-risk, bipolar spectrum, and BD II participants to investigate responding in BD. In this study, I was able to recruit individuals who met criteria for BD I.

A second strength of this study was that while the Pronin mood induction had been shown to increase mood in a university sample, it had not yet been validated with a clinical sample of individuals diagnosed with BD. My study was able to show that this mood induction increased mood in this vulnerable population group, without unsafe consequences. The literature on the success of positive mood inductions has been mixed, with some studies reporting success in inducing positive mood in BD or at-risk for BD participants with false feedback (Farmer et al., 2006; Roiser et al., 2008; Trevisani et al., 2008; Wright et al., 2005), while other studies have reported unsuccessful positive mood inductions (Gruber et al., 2011; Mansell & Lam, 2006). Interestingly, some studies show a profile in which BD participants continue to sustain a positive mood, even over

negative and neutral stimuli, whereas control participants return to their baseline levels (Farmer et al., 2006; Gruber et al., 2009, 2011). However, BD participants in this study did not differ from control participants in self-report of mood reactivity to the mood induction, nor was there a difference between the two groups in mood levels at the end of the study session.

Future Directions

Most studies, including this study, have used a cross-sectional design to assess for threat reactivity. A longitudinal study could assess whether threat reactivity changes over time with changes in mood state and symptoms, or the course of illness (age, number of episodes, medication classes). In addition, integrating other factors such as co-morbidities with anxiety, alcohol, or substance abuse, may help elucidate the interaction between symptomatology and threat reactivity in BD as well. The two studies on the startle response with BD participants did not assess for how alcohol or substance co-morbidities may have impacted startle response in their studies. Studies on the startle response for those who meet criteria for alcohol abuse indicated that alcohol may reduce the overall levels of arousal, but not affect the pattern of responding to valenced pictures (Curtin et al., 1998; Grillon et al., 1994). With a high co-morbidity rate of alcohol and substances in BD, future studies may be able to further understand threat detection within BD by including those with and without alcohol and substance disorders in startle response studies.

While this study had a non-psychiatric control group, future studies may be able to further disentangle the unique aspects of BD by recruiting participants with disorders known to also encounter difficulties with emotional processing such as MDD and

borderline personality disorder. Moreover, medications have been shown to impact neurological responses to emotional stimuli (Yurgelun-Todd et al., 2000). Since an unmedicated BD sample is unethical and unfeasible, future studies should randomly assign participants based upon medication class. Also, it may be the case that by selecting broadly defined euthymic BD participants, this study selected out the most severely symptomatic and chronic participants-- in whom threat detection may be the greatest risk factor (Carpenter & Hittner, 1997; Lebowitz et. al., 2001; Swann 2001; van Gorp et al., 1998). Thus, future studies should follow individuals longitudinally in order to assess for changes in physiological reactivity based upon mood state or other characteristics, including individuals with complex co-morbidities and in elevated and depressed states in order to further tease apart state, as compared to trait, effects of threat detection.

Incorporating other paradigms such as fMRI studies, and genetic studies will allow researchers to further understand how other methods of investigation may assist in the assessment and treatment of impulsivity during mania in BD. Indeed, there is growing evidence of the role of the amygdala in emotion perception and regulation, the ventral lateral prefrontal cortex modulating external emotional stimuli, and the ventromedial cortex modulating internal emotional states (Strakowski et al., 2012). In consequence, future fMRI studies could incorporate laboratory tasks designed to elicit specific positive emotions (e.g., achievement, joy, pride, excitement), which are related to specific patterns of emotional response in BD (Chen & Johnson, 2012; Gruber & Johnson, 2009). Genetics studies may help better elucidate vulnerability and risk factors that play into impulsivity. One study indicated that unafflicted family members and individuals with BD had a blunted startle response when presented with IAPS stimuli, as compared to

control participants (Giakoumaki et al., 2010). This response pattern could be a heritable marker suggesting a genetic influence in threat responsivity.

As well as investigating other paradigms related to impulsivity, exploring other mechanisms related to affective processing in BD may help explain the role of emotion-based impulsivity in BD. There is growing evidence of the major role that positive emotion plays in BD (Gruber et al., 2010). Based on clinical observation, and the literature surrounding BD and impulsivity, we know that some individuals with BD can become engaged in high risk activities when manic. Several pieces of research suggest that positive urgency may be an underlying vulnerability trait factor for decreased threat detection and impulsivity in BD. For example, while not a part of this study, participants with BD who rated themselves higher on the PUM, revealing a tendency to be responsive to positive mood, also displayed both greater startle reactivity to the acoustic probe, and slower response latency during the positive block after the mood induction. That is, participants who were more likely to engage in rash behaviors while in a positive mood were more reactive and slower to react during the positive IAPS presentation after the mood induction. In participants with BD, the PUM has accounted for 14% of the variance in a study on quality of life and BD (Victor et al., 2011). The willingness to engage in high-risk activities during a positive mood can lead to disastrous consequences and poor life choices. Thus, a pivotal goal would be to identify the behavioral and cognitive strategies that allow individuals to regulate their positive mood and increase sensitivity to threat during a positive mood.

Clinical implications

Investigation of threat detection has many important clinical implications for the assessment and treatment of BD. Indeed, impulsivity in an elevated mood has been associated with high risk for suicide, financial difficulties, risky sexual behaviors, and alcohol and substance abuse. Moreover, there are several pieces of evidence that suggest that individuals with BD who exhibit difficulties regulating positive emotions may experience a more severe course of the illness. In a 2009 study, participants with BD who endorsed experiencing more positive emotions experienced increased symptoms of mania 6 months afterwards (Gruber & Johnson, 2009). It could be that difficulties with emotional regulation in BD are explained by the maintenance of, or positive rumination on, positive emotions, regardless of environmental cues (Gruber, 2011).

Clinicians may be able to intervene at critical junctures as an individual with BD begins to experience symptoms of mood elevation in order to mitigate engagement in rash actions and behaviors. Studies have reported that individuals with BD are able to accurately report experiencing mania prodromal symptoms (Lam & Wong, 2005). Therefore, one clinical intervention may be the use of implementation interventions (i.e., if-then planning) in order to strategically avoid risk-taking behaviors (Webb et al., 2010). Another clinical intervention may be the use of cognitive reappraisals to modify the subjective emotional intensity of an emotional experience (Ochsner et al., 2004; Urry, 2009). Incorporation psychotherapy interventions such as cognitive behavioral therapy (Basco & Rush, 2005), family therapy (Miklowitz et al., 2003), life events and social rhythms therapy (Frank et al., 2005), psychoeducation on mania in bipolar disorders (Johnson & Fulford, 2009), and dialectical behavioral therapy (Linehan et al., 2008) may

address how to identify an elevated mood state, regulate emotions, and incorporate behavioral and cognitive interventions for threat detection. As well, there needs to be further research into the efficacy of adjunctive pharmacological interventions (Braquehais et al., 2010) in conjunction with psychological interventions specifically targeting impulsivity in BD. In sum, research may inform how clinicians can focus on the identification and integration of intervention that can target risk factors for impulsivity during mania such as threat detection and emotion regulation of positive emotions.

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Tables

Table 1a

Characteristics of Bipolar I and control group

		Bipolar	Controls	*p*-value
		(n = 27)	(n = 29)	
Age		36.43 +/- 9.35	35.22 +/- 10.02	.14
Ethnicity		69% Caucasian	64% Caucasian	.45
		23% African American	24% African American	
		7% other/bi-racial	12% other/bi-racial	
MDE onset		18.4 +/- 6.4		
Number of episodes of MDE		9.2 +/- 4.76		
Mania onset		22.4 +/- 4.9		
Number of episodes of Mania		7.92 +/- 11.01		
Anxiety History		48%	21%	.001
Alcohol/Substance Abuse History		55%	3%	.001
Female		70%	54%	.34
Years of Education		14.74 +/- 1.47	14.95+/- 2.16	.68
BRMS		2.32 +/- 2.12	1.34 +/- 1.29	.03
Altman		10.26 +/- 3.62	10.44 +/- 4.19	.86
Ham-D		4.48 +/- 2.86	2.43 +/- 2.20	.002
MASQ Anhedonic Depression		14.10 +/- 6.63	9.06 +/- 3.43	<.001
MASQ Somatic Anxiety		22.16 +/- 8.16	16.16 +/- 5.07	.001
Reverse Digit Span		6.96 +/- 1.72	6.60 +/- 2.62	.56
Mood Stabilizer		40%	0%	
Lithium		15%	0%	
SSRI		65%	18%	
Antipsychotic		20%	0%	
Benzodiazepines		44%	18%	

Table 2a
Values of variables

	Mean	SD	Min	Max	Skew (se)	Kurtosis (se)
Age	35.45	10.89	20	57	.19(.34)	--.59(.67)
Years of Education	14.82	1.83	11	19	.56(.34)	-.61(.67)
BRMS	1.83	1.80	0	5	1.31(.34)	-.41(.67)
Altman	10.35	3.89	5	19	.42(.32)	-.78(.67)
Ham-D	3.44	2.73	0	7	.33(.34)	-.96(.67)
MASQ Anhedonic Depression	11.54	5.80	7	25	1.76(.34)	2.65(.67)
MASQ Somatic Anxiety	19.11	7.36	13	47	2.02(.34)	4.62(.67)
Reverse Digit Span	6.77	2.22	3	13	.77(.34)	.46(.67)

Table 3a
Untransformed Eyeblink Amplitude Data

			M	SD	Min	Max	Skew	SE	Kurtosis	SE
	Neutral	2500	38.16	23.03	10.83	111.21	1.32	0.33	1.18	0.65
		4500	34.95	19.61	6.13	94.65	1.32	0.33	1.94	0.65
Bl 1	Negative	2500	32.38	18.32	5.87	94.33	0.96	0.33	1.29	0.65
		4500	30.77	15.70	8.40	86.49	1.05	0.33	1.97	0.65
	Neutral	2500	26.72	15.58	8.25	90.80	1.82	0.33	4.67	0.65
		4500	29.33	17.32	9.26	101.54	1.88	0.33	4.51	0.65
Bl 2	Neutral	2500	25.07	16.54	3.25	79.91	1.18	0.33	1.31	0.65
		4500	23.92	12.80	4.09	68.85	1.29	0.33	2.50	0.65
Bl 3	Positive	2500	24.44	12.99	6.37	68.58	1.40	0.33	2.19	0.65
		4500	27.23	19.21	6.33	117.96	2.39	0.33	7.80	0.65
	Neutral	2500	25.25	12.34	9.06	68.54	1.24	0.33	1.99	0.65
		4500	26.75	14.75	9.04	77.01	1.18	0.33	1.33	0.65
Bl 4	Negative	2500	31.88	17.92	7.00	98.55	1.45	0.33	2.39	0.65
		4500	33.94	22.02	3.73	138.70	2.13.	0.33	8.04	0.65
	Neutral	2500	30.86	16.36	9.54	90.12	1.07	0.33	1.59	0.65
		4500	28.91	17.19	4.86	99.32	1.66	0.33	4.19	0.65
Bl 5	Neutral	2500	27.40	13.50	3.81	56.50	0.35	0.33	-0.68	0.65
		4500	26.05	15.62	6.16	80.64	1.32	0.33	1.96	0.65
Bl 6	Positive	2500	27.30	15.83	9.50	79.57	1.34	0.33	1.72	0.65
		4500	30.76	22.66	10.83	123.92	2.21	0.33	5.69	0.65
	Neutral	2500	28.78	15.80	7.73	75.86	1.29	0.33	1.26	0.65
		4500	26.05	16.29	5.79	87.73	1.38	0.33	2.81	0.65

Table 3b
Untransformed Eyeblink Latency Data

			M	SD	Max	Min	Skew	SE	Kurtosis	SE
	Neutral	2500	0.85	0.38	1.65	0.19	0.01	0.32	-0.72	0.63
		4500	0.80	0.37	1.81	0.13	0.44	0.32	-0.17	0.63
Bl 1	Negative	2500	0.77	0.45	1.92	0.10	0.38	0.32	-0.61	0.63
		4500	0.87	0.39	1.80	0.03	0.09	0.32	-0.05	0.63
	Neutral	2500	0.71	0.42	1.74	0.01	0.63	0.32	-0.23	0.63
		4500	0.85	0.45	1.79	0.15	0.19	0.32	-1.00	0.63
Bl 2	Neutral	2500	0.79	0.63	4.08	0.10	0.79	0.32	-0.19	0.63
		4500	0.77	0.41	1.85	0.00	0.53	0.32	0.16	0.63
Bl 3	Positive	2500	0.87	0.42	1.85	0.03	0.18	0.32	-0.46	0.63
		4500	0.86	0.39	1.70	0.29	0.24	0.32	-0.80	0.63
	Neutral	2500	0.73	0.35	1.51	0.10	-0.04	0.32	-0.68	0.63
		4500	0.81	0.45	2.12	0.01	0.78	0.32	0.30	0.63
Bl 4	Negative	2500	0.90	0.47	2.38	0.10	0.73	0.32	0.53	0.63
		4500	0.78	0.47	2.57	0.14	0.73	0.32	0.04	0.63
	Neutral	2500	0.78	0.42	1.65	0.02	0.19	0.32	-1.03	0.63
		4500	0.87	0.49	2.50	0.05	0.17	0.32	-0.57	0.63
Bl 5	Neutral	2500	0.81	0.51	2.82	0.03	0.20	0.32	-0.81	0.63
		4500	0.86	0.50	2.73	0.06	0.58	0.32	-0.44	0.63
Bl 6	Positive	2500	0.89	0.51	2.99	0.05	0.58	0.32	-0.11	0.63
		4500	0.82	0.44	1.69	0.00	0.28	0.32	-0.86	0.63
	Neutral	2500	0.91	0.46	1.75	0.05	0.00	0.32	-0.97	0.63
		4500	0.70	0.42	1.96	0.10	0.72	0.32	0.37	0.63

Table 3c
Untransformed SCL Tonic Level Data

			M	SD	Min	Max	Skew	SE	Kurtosis	SE
	Neutral	2500	8.89	4.71	2.18	19.06	0.50	0.33	-0.84	0.66
		4500	8.98	4.92	2.08	20.28	0.53	0.33	-0.87	0.66
Bl 1	Negative	2500	8.00	4.23	2.55	16.88	0.50	0.33	-1.00	0.66
		4500	7.80	4.32	2.51	16.73	0.61	0.33	-0.87	0.66
	Neutral	2500	7.29	4.23	2.20	16.16	0.69	0.33	-0.83	0.66
		4500	7.00	4.21	2.11	16.18	0.76	0.33	-0.79	0.66
Bl 2	Neutral	2500	6.76	3.04	2.26	15.59	0.88	0.33	0.25	0.66
		4500	7.19	4.62	2.05	21.51	1.03	0.33	0.36	0.66
Bl 3	Positive	2500	7.01	4.95	2.05	25.35	1.35	0.33	2.04	0.66
		4500	6.89	5.00	2.03	26.11	1.51	0.33	2.78	0.66
	Neutral	2500	6.82	5.08	1.81	25.63	1.56	0.33	2.57	0.66
		4500	6.65	4.88	1.59	24.04	1.45	0.33	2.01	0.66
Bl 4	Negative	2500	8.94	5.11	2.52	26.42	1.00	0.33	1.28	0.66
		4500	8.51	5.03	2.26	24.24	0.91	0.33	0.58	0.66
	Neutral	2500	7.93	4.55	2.20	19.29	0.73	0.33	-0.20	0.66
		4500	7.84	4.68	2.14	20.88	0.85	0.33	0.03	0.66
Bl 5	Neutral	2500	7.80	4.64	1.69	20.55	0.75	0.33	0.15	0.66
		4500	7.77	4.58	1.65	19.89	0.71	0.33	-0.10	0.66
Bl 6	Positive	2500	7.43	4.70	1.52	19.36	0.90	0.33	0.16	0.66
		4500	7.28	4.61	1.48	18.56	0.88	0.33	-0.06	0.66
	Neutral	2500	7.05	4.50	1.41	18.96	0.92	0.33	0.07	0.66
		4500	7.12	4.62	1.37	20.20	0.90	0.33	0.14	0.66

Table 4a
Square-root Transformed Eyeblink Amplitude Data

			M	SD	Min	Max	Skew	SE	Kurtosis	SE
	Neutral	2500	5.74	1.51	3.29	9.63	0.76	0.34	0.42	0.67
		4500	5.52	1.36	2.48	9.39	0.14	0.34	0.01	0.67
Bl 1	Negative	2500	5.31	1.45	2.42	8.29	0.00	0.34	-0.68	0.67
		4500	5.22	1.20	2.90	7.44	-0.20	0.34	-1.00	0.67
	Neutral	2500	4.79	1.10	2.87	7.86	0.24	0.34	-0.08	0.67
		4500	5.04	1.16	3.04	8.50	0.76	0.34	0.51	0.67
Bl 2	Neutral	2500	4.57	1.37	1.80	7.64	0.27	0.34	-0.86	0.67
		4500	4.58	1.08	2.02	6.81	-0.05	0.34	-0.67	0.67
Bl 3	Positive	2500	4.63	1.02	2.52	6.91	0.29	0.34	-0.62	0.67
		4500	4.73	1.19	2.52	7.84	0.38	0.34	0.09	0.67
	Neutral	2500	4.79	1.06	3.01	7.07	0.28	0.34	-0.59	0.67
		4500	4.86	1.23	3.01	8.78	0.73	0.34	0.42	0.67
Bl 4	Negative	2500	5.26	1.25	2.65	8.11	0.48	0.34	-0.58	0.67
		4500	5.38	1.51	1.93	8.94	0.22	0.34	-0.85	0.67
	Neutral	2500	5.22	1.27	3.09	7.95	0.14	0.34	-0.71	0.67
		4500	4.98	1.26	2.20	7.68	0.00	0.34	0.20	0.67
Bl 5	Neutral	2500	4.94	1.25	1.95	7.42	-0.25	0.34	-0.49	0.67
		4500	4.75	1.33	2.48	8.98	0.66	0.34	1.20	0.67
Bl 6	Positive	2500	4.92	1.30	3.08	8.92	0.74	0.34	0.34	0.67
		4500	4.91	1.12	3.29	7.30	0.28	0.34	-1.10	0.67
	Neutral	2500	4.99	1.17	2.78	8.41	0.74	0.34	1.20	0.67
		4500	4.68	1.14	2.41	7.11	0.46	0.34	-0.55	0.67

Table 4b
Eyeblink Latency Data

			M	SD	Min	Max	Skew	SE	Kurtosis	SE
	Neutral	2500	0.82	0.36	0.19	1.55	-0.02	0.33	-0.70	0.64
		4500	0.80	0.37	0.13	1.81	0.48	0.33	-0.09	0.64
Bl 1	Negative	2500	0.76	0.45	0.10	1.92	0.47	0.33	-0.45	0.64
		4500	0.87	0.39	0.03	1.80	0.07	0.33	-0.16	0.64
	Neutral	2500	0.69	0.40	0.01	1.74	0.61	0.33	-0.24	0.64
		4500	0.84	0.44	0.15	1.79	0.27	0.33	-0.91	0.64
Bl 2	Neutral	2500	0.72	0.43	0.10	1.91	0.53	0.33	0.01	0.64
		4500	0.75	0.39	0.00	1.82	0.40	0.33	0.08	0.64
Bl 3	Positive	2500	0.87	0.41	0.03	1.68	0.03	0.33	-0.68	0.64
		4500	0.85	0.38	0.29	1.70	0.18	0.33	-0.81	0.64
	Neutral	2500	0.75	0.35	0.10	1.51	-0.10	0.33	-0.62	0.64
		4500	0.82	0.46	0.01	2.12	0.59	0.33	0.20	0.64
Bl 4	Negative	2500	0.87	0.46	0.10	2.38	0.82	0.33	0.88	0.64
		4500	0.74	0.40	0.14	1.89	0.45	0.33	0.09	0.64
	Neutral	2500	0.76	0.41	0.02	1.54	0.21	0.33	-1.06	0.64
		4500	0.82	0.45	0.05	1.87	0.25	0.33	-0.51	0.64
Bl 5	Neutral	2500	0.77	0.44	0.03	1.75	0.21	0.33	-0.76	0.64
		4500	0.82	0.42	0.06	1.84	0.53	0.33	-0.35	0.64
Bl 6	Positive	2500	0.87	0.43	0.05	1.98	0.57	0.33	-0.14	0.64
		4500	0.80	0.44	0.00	1.69	0.31	0.33	-0.82	0.64
	Neutral	2500	0.92	0.46	0.05	1.75	-0.02	0.33	-0.96	0.64
		4500	0.69	0.42	0.10	1.96	0.60	0.33	0.45	0.64

Table 4c
SCL Tonic Level Data

		M	SD	Min	Max	Skew	SE	Kurtosis	SE
	Neutral	8.65	4.63	2.18	19.06	0.61	0.34	-0.61	0.67
		8.70	4.79	2.08	20.28	0.64	0.34	-0.60	0.67
Bl 1	Negative	7.72	4.04	2.55	16.69	0.55	0.34	-0.90	0.67
		7.46	4.03	2.51	16.73	0.63	0.34	-0.78	0.67
	Neutral	6.99	4.02	2.20	16.10	0.56	0.34	-0.60	0.67
		6.68	3.95	2.11	15.87	0.63	0.34	-0.58	0.67
Bl 2	Neutral	6.46	2.65	2.26	13.01	0.64	0.34	-0.40	0.67
		6.74	4.04	2.05	15.95	0.62	0.34	-0.55	0.67
Bl 3	Positive	6.48	4.12	2.05	17.71	0.65	0.34	-0.35	0.67
		6.34	4.08	2.03	17.11	0.42	0.34	-0.16	0.67
	Neutral	6.22	4.10	1.81	16.95	0.51	0.34	0.22	0.67
		6.10	4.00	1.59	15.95	0.59	0.34	0.09	0.67
Bl 4	Negative	8.42	4.37	2.52	19.31	0.53	0.34	-0.36	0.67
		8.01	4.38	2.26	19.17	0.59	0.34	-0.40	0.67
	Neutral	7.51	4.06	2.20	17.30	0.58	0.34	-0.51	0.67
		7.39	4.12	2.14	17.30	0.64	0.34	-0.52	0.67
Bl 5	Neutral	7.34	4.06	1.69	16.39	0.43	0.34	-0.64	0.67
		7.32	4.03	1.65	16.28	0.45	0.34	-0.73	0.67
Bl 6	Positive	6.98	4.18	1.52	19.04	0.59	0.34	0.41	0.67
		6.86	4.16	1.48	18.17	0.65	0.34	0.06	0.67
	Neutral	6.62	3.97	1.41	16.06	0.58	0.34	-0.49	0.67
		6.66	4.04	1.37	15.79	0.60	0.34	-0.45	0.67

Table 5a
SAM Mood Ratings for Study Sample with Physiological Measures

SAM Self-report ratings	Bipolar (n = 27)		Control (n = 29)	
	M	SD	M	SD
After Neutral Block	5.74	1.83	5.91	1.29
After Block 1 (Negative-Neutral)	5.11	1.69	4.90	1.35
After Neutral Block 2	5.30	1.20	5.41	.78
After Block 3 (Positive-Neutral)	5.74	1.40	5.97	1.02
SAM Post-mood Induction	6.44	1.53	6.76	1.18
After Block 4 (Negative-Neutral)	5.52	1.31	5.72	1.22
After Neutral Block 5	5.78	1.12	5.41	.91
After Block 6 (Positive-Neutral)	5.52	1.60	5.72	1.22

Table 5b
SAM Arousal Ratings for Entire Study Sample with Physiological Measures

SAM Self-report ratings	Bipolar (n = 27)		Control (n = 29)	
	M	SD	M	SD
After Neutral Block	5.30	1.88	5.37	1.61
After Block 1 (Negative-Neutral)	4.70	1.38	5.10	1.05
After Neutral Block 2	4.93	1.54	4.55	1.27
After Block 3 (Positive-Neutral)	5.48	1.70	4.83	1.39
SAM Post-mood Induction	6.48	1.78	6.27	1.23
After Block 4 (Negative-Neutral)	4.93	1.71	4.90	1.23
After Neutral Block 5	5.11	1.72	4.38	1.42
After Block 6 (Positive-Neutral)	4.74	1.95	4.90	1.37

Table 6a

SAM Mood Ratings for Participants Who Reported a Successful Positive Mood Change

SAM Self-report ratings	Bipolar (n = 22)		Control (n = 25)	
	M	SD	M	SD
Neutral Block	5.64	1.94	6.04	1.37
Block 1 (Negative-Neutral)	5.09	1.80	5.20	1.44
Neutral Block 2	5.23	1.27	5.56	1.08
Block 3 (Positive-Neutral)	5.77	1.51	6.04	1.21
SAM Post-mood induction	6.95	1.21	7.12	1.13
Block 4 (Negative-Neutral)	5.59	1.40	5.40	1.16
Neutral Block 5	5.82	1.18	5.44	.92
Block 6 (Positive-Neutral)	5.55	1.74	5.72	1.21

Table 6b

SAM Arousal Ratings for Participants Who Reported a Successful Positive Mood Change

SAM Self-report ratings	Bipolar (n = 22)		Control (n = 25)	
	M	SD	M	SD
Neutral Block	5.55	1.95	5.48	1.64
Block 1 (Negative-Neutral)	4.77	1.38	5.32	1.31
Neutral Block 2	4.95	1.50	4.68	1.58
Block 3 (Positive-Neutral)	5.50	1.79	4.96	1.62
SAM Post-mood induction	6.73	1.78	6.32	1.73
Block 4 (Negative-Neutral)	4.91	1.80	4.92	1.55
Neutral Block 5	5.09	1.82	4.56	1.69
Block 6 (Positive-Neutral)	4.77	2.00	5.08	1.58

Table 7

Initial Neutral Block

	df	F	*p*
	SCL		
Group	1	0.07	.774
Time	1	0.07	.793
Time X Group	1	0.54	.467
Error(Time)	51		
	Eyeblink Amplitude		
Group	1	0.55	.565
Time	1	2.06	.274
Time X Group	1	0.07	.976
Error(TIME)	53		
	Eyeblink Latency		
Group	1	0.18	.675
Time	1	0.25	.621
Time X Group	1	0.59	.446
Error(Time)	52		

Table 8

Block 1 (Negative -Neutral)

	df	F	*p*
	SCL		
Group	1	0.05	.818
Time	1	26.55	.000
Time X Group	1	2.53	.118
Error(Time)	50		
Valence	1	41.14	.000
Valence X Group	1	0.47	.497
Error(Valence)	50		
Time X Valence	1	0.25	.618
Time X Valence X Group	1	1.56	.217
Error(Time X Valence)	50		
	Eyeblink Amplitude		
Group	1	0.55	.463
Time	1	0.54	.467
Time X Group	1	0.51	.478
Error(Time)	50		
Valence	1	9.91	.003
Valence X Group	1	1.73	.194
Error(Valence)	50		
Time X Group	1	1.74	.193
Time X Valence X Group	1	0.11	.738
Error(Time X Valence)	50		
	Eyeblink Latency		
Group	1	0.10	.753
Time	1	8.35	.006
Time X Group	1	0.40	.532
Error(Time)	52		
Valence	1	0.79	.378
Valence X Group	1	2.30	.136
Error(Valence)	52		
Time X Valence	1	0.09	.768
Time X Valence X Group	1	0.10	.755
Error(Time X Valence)	52		

Table 9

Block 2 (All-neutral)

	df	F	*p*
SCL			
Group	1	0.43	.337
Time	1	0.80	.375
Time X Group	1	0.43	.514
Error(Time)	49		
Eyeblink Amplitude			
Group	1	0.86	.358
Time	1	0.06	.805
Time X Group	1	0.01	.917
Error(Time)	51		
Eyeblink Latency			
Group	1	0.25	.146
Time	1	0.27	.603
Time X Group	1	0.25	.620
Error(Time)	52		

Table 10

Block 3 (Positive-Neutral)

	df	F	*p*
	SCL		
Group	1	0.14	.712
Time	1	7.56	.008
Time X Group	1	0.44	.509
Error(Time)	48		
Valence	1	10.88	.002
Valence X Group	1	1.74	.194
Error(Valence)	48		
Time X Valence	1	0.07	.395
Time X Valence X Group	1	1.03	.316
Error(Time X Valence)	48		
	Eyeblink Latency		
Group	1	0.18	.669
Time	1	0.21	.648
Time X Group	1	2.37	.130
Error(Time)	51		
Valence	1	1.61	.211
Valence X Group	1	1.27	.265
Error(Valence)	51		
Time X Valcne	1	0.75	.390
Time X Valence X Group	1	1.69	.199
Error(Time X Valence)	51		
	Eyeblink Amplitude		
Group	1	1.27	.266
Time	1	1.32	.256
Time X Group	1	3.37	.072
Error(Time)	52		
Valence	1	0.25	.618
Valence X Group	1	0.55	.460
Error(Valence)	52		
Time X Valcne	1	0.02	.898
Time X Valence X Group	1	5.02	.029
Error(Time X Valence)	52		

Table 11

Block 4 (Negative-Neutral)

	df	F	*p*
	SCL		
Group	1	2.68	.110
Time	1	9.33	.004
Time X Group	1	0.25	.618
Error(Time)	38		
Valence	1	27.05	.000
Valence X Group	1	4.03	.052
Error(Valence)	38		
Time X Valence	1	5.36	.026
Time X Valence X Group	1	0.63	.432
Error(Time X Valence)	38		
	Eyeblink Amplitude		
Group	1	1.855	.181
Time	1	.005	.943
Time X Group	1	.329	.570
Error(Time)	40		
Valence	1	4.063	.051
Valence X Group	1	.005	.942
Error(Valence)	40		
Time X Valence	1	3.658	.063
Time X Valence X Group	1	.288	.594
Error(Time X Valence)	40		
	Eyeblink Latency		
Group	1	1.34	.255
Time	1	0.12	.732
Time X Group	1	0.80	.377
Error(Time)	37		
Valence	1	0.88	.355
Valence X Group	1	2.16	.150
Error(Valence)	37		
Time X Valence	1	2.45	.126
Time X Valence X Group	1	0.14	.706
Error(Time X Valence)	37		

Table 12

Block 5 (All-neutral)

	df	*F*	*p*
	SCL		
Group	1	2.04	.161
Time	1	1.21	.278
Time X Group	1	1.56	.219
Error(Time)	38		
	Eyeblink Amplitude		
Group	1	1.20	.280
Time	1	0.12	.726
Time X Group	1	0.57	.455
Error(Time)	40		
	Eyeblink Latency		
Group	1	.076	.784
Time	1	.196	.661
Time X Group	1	1.102	.301
Error(Time)	37		

Table 13

Block 6 (Positive-Neutral)

	df	*F*	*p*
SCL			
Group	1	1.94	.172
Time	1	5.61	.023
Time X Group	1	1.39	.246
Error(Time)	38		
Valence	1	11.69	.002
Valence X Group	1	0.66	.420
Error(Valence)	38		
Time X Valence	1	1.86	.181
Time X Valence X Group	1	0.06	.807
Error(Time X Valence)	38		
Eyeblink Amplitude			
Group	1	2.14	.151
Time	1	2.31	.136
Time X Group	1	1.10	.300
Error(Time)	41		
Valence	1	0.20	.656
Valence X Group	1	1.30	.260
Error(Valence)	41		
Time X Valence	1	3.66	.063
Time X Valence X Group	1	0.17	.684
Error(Time X Valence)	41		
Eyeblink Latency			
Group	1	0.51	.478
Time	1	3.96	.054
Time X Group	1	1.93	.173
Error(Time)	37		
Valence	1	2.24	.143
Valence X Group	1	0.35	.555
Error(Valence)	37		
Time X Valence	1	0.30	.590
Time X Valence X Group	1	0.75	.393
Error(Time X Valence)	37		

Table 14

Neutral Blocks 1 to 3

	df	F	*p*
	SCL		
Group	1	0.85	.362
Time	1	0.16	.688
Time X Group	1	0.35	.559
Error(Time)	36		
Block	2	6.05	.004
Block X Group	2	0.15	.624
Error(Block)	72	0.04	.843
Time X Block	2	1.98	.132
Time X Block X Group	2	0.53	.397
Error(Time X Block)	72		
	Eyeblink Amplitude		
Group	1	0.85	.362
Time	1	2.03	.163
Time X Group	1	2.44	.127
Error(Time)	37		
Block	2	4.00	.022
Block X Group	2	0.73	.487
Error(Block)	74		
Time X Block	2	0.36	.702
Time X Block X Group	2	0.84	.436
Error(Time X Block)	74		
	Eyeblink Latency		
Group	1	0.41	.528
Time	1	0.86	.361
Time X Group	1	2.25	.142
Error(Time)	37		
Block	2	0.57	.570
Block X Group	2	1.46	.240
Error(Block)	74		
Time X Block	2	0.75	.475
Time X Block X Group	2	0.72	.490
Error(Time X Block)	74		

Table 15

Neutral Blocks 4 to 6

	df	*F*	*p*
	SCL		
Group	1	2.03	.163
Time	1	1.28	.264
Time X Group	1	0.33	.567
Error(Time)	38		
Block	2	10.50	.000
Block X Group	2	0.08	.920
Error(Block)	76		
Time X Block	2	0.29	.750
Time X Block X Group	2	1.23	.300
Error(Time X Block)	76		
	Eyeblink Amplitude		
Group	1	2.045	.161
Time	1	0.17	.686
Time X Group	1	0.00	.998
Error(Time)	39		
Block	2	6.14	.003
Block X Group	2	0.47	.627
Error(Block)	78		
Time X Block	2	2.45	.359
Time X Block X Group	2	0.17	.845
Error(Time X Block)	78		
	Eyeblink Latency		
Group	1	1.34	.254
Time	1	1.30	.262
Time X Group	1	0.43	.517
Error(Time)	37		
Block	2	0.51	.601
Block X Group	2	1.47	.237
Error(Block)	74		
Time X Block	2	1.56	.216
Time X Block X Group	2	0.51	.603
Error(Time X Block)	74		

Table 16

Combined data: SCL

	df	F	*p*
	SCL		
Group	1	1.11	.299
Pre-post	1	2.41	.129
Pre-post X Group	1	1.59	.215
Error(Pre-post)	36		
Valence	2	22.85	.000
Valence X Group	2	0.16	.852
Error(Valence)	72		
Time	1	10.11	.003
Time X Group	1	0.00	.975
Error(Time)	36		
Pre-post X Valence	2	0.17	.841
Pre-post X Valence X Group	2	3.78	.028
Error(Pre-post X Valence)	72		
Pre-post X Time	1	0.41	.526
Pre-post X Time X Group	1	0.62	.438
Error(Pre-post X Time)	36		
Valence X Time	2	4.30	.017
Valence X Time X Group	2	0.32	.727
Error(Valence X Time)	72		
Pre-post X Valence X Time	2	0.39	.679
Pre-post X Valence X Time X Group	2	0.96	.388
Error(Pre-post X Valence X Time)	72		

Table 17

Combined data: Eyeblink Amplitude

	df	F	*p*
Eyeblink Amplitude			
Group	1	0.81	0.374
Pre-post	1	6.07	0.019
Pre-post X Group	1	0.17	0.684
Error(Pre-post)	36		
Valence	2	13.61	.000
Valence X Group	2	0.87	0.422
Error(Valence)	72		
Time	1	0.78	0.384
Time X Group	1	2.93	0.096
Error(Time)	36		
Pre-post X Valence	2	0.03	0.972
Pre-post X Valence X Group	2	1.00	0.374
Error(Pre-post X Valence)	72		
Pre-post X Time	1	0.12	0.735
Pre-post X Time X Group	1	1.76	0.193
Error(Pre-post X Time)	36		
Valence X Time	2	0.59	0.559
Valence X Time X Group	2	0.27	0.766
Error(Valence X Time)	72		
Pre-post X Valence X Time	2	2.47	0.1
Pre-post X Valence X Time X Group	2	1.26	0.289
Error(Pre-post X Valence X Time)	72		

Table 18

Combined data: Eyeblink Latency

	df	F	*p*
	Eyeblink Latency		
Group	1	0.78	0.382
Pre-post	1	1.97	0.168
Pre-post X Group	1	0.00	0.992
Error(Pre-post)	37		
Valence	2	2.62	0.079
Valence X Group	2	0.94	0.397
Error(Valence)	74		
Time	1	1.00	0.325
Time X Group	1	1.19	0.283
Error(Time)	37		
Pre-post X Valence	2	0.10	0.906
Pre-post X Valence X Group	2	0.06	0.943
Error(Pre-post X Valence)	74		
Pre-post X Time	1	2.37	0.132
Pre-post X Time X Group	1	0.01	0.905
Error(Pre-post X Time)	37		
Valence X Time	2	0.76	0.469
Valence X Time X Group	2	1.22	0.301
Error(Valence X Time)	74		
Pre-post X Valence X Time	2	0.56	0.573
Pre-post X Valence X Time X Group	2	1.51	0.229
Error(Pre-post X Valence X Time)	74		

Table 19

Hypothesis 3

	df	F	*p*
Eyeblink Amplitude			
Group	1	1.07	.308
Time	1	0.48	.492
Time X Group	1	0.14	.713
Error(Time)	41		
Pre-post	1	0.82	.371
Pre-post X Group	1	0.00	.965
Error(Pre-post)	41		
Time X Pre-post	1	1.86	.180
Time X Pre-post X Group	1	1.02	.318
Error(Time X Pre-post)	41		
Eyeblink Latency			
Group	1	0.02	.904
Time	1	0.01	.938
Time X Group	1	0.30	.585
Error(Time)	37		
Pre-post	1	0.87	.356
Pre-post X Group	1	0.05	.832
Error(Pre-post)	37		
Time X Pre-post	1	2.15	.151
Time X Pre-post X Group	1	0.44	.510
Error(Time X Pre-post)	37		

Table 20.

Characteristics of Bipolar I and control group who reported successful mood induction

	Bipolar	Controls	*p*-value
	(n = 22)	(n = 25)	
Age	35.8 +/- 11.75	36.86 +/- 11.27	.75
Ethnicity	68% Caucasian	60% Caucasian	.58
	23% Af Am	20% Af Am	
	9% other/bi-racial	20% other/bi-racial	
MDE onset	20.5 +/- 6.36		
episodes of MDE	8.75 +/- 4.81		
Mania onset	23.2 +/- 4.24		
episodes of Mania	9.01 +/- 12.72		
Anxiety History	50%	20%	.001
Alcohol/Substance	32%	4%	.001
Female	80%	64%	.17
Years of Education	14.83 +/- 1.54	15.08 +/- 2.02	.65
BRMS	2.27 +/- 2.16	1.36 +/- 1.32	.08
Altman	10.91 +/- 3.78	9.64 +/- 3.99	.27
Ham-D	5.00 +/- 2.65	2.25 +/- 1.96	<.001
MASQ Dep	14.68 +/- 6.83	8.24 +/- 1.83	<.001
MASQ Anx	23.05 +/- 8.73	15.52 +/- 3.10	.001
Reverse Digit Span	6.74 +/- 2.05	6.76 +/- 2.57	.97
Mood Stabilizer	41%	0%	
Lithium	23%	0%	
SSRI	73%	20%	
Antipsychotic	18%	0%	
Benzodiazepines	45%	16%	

Figures

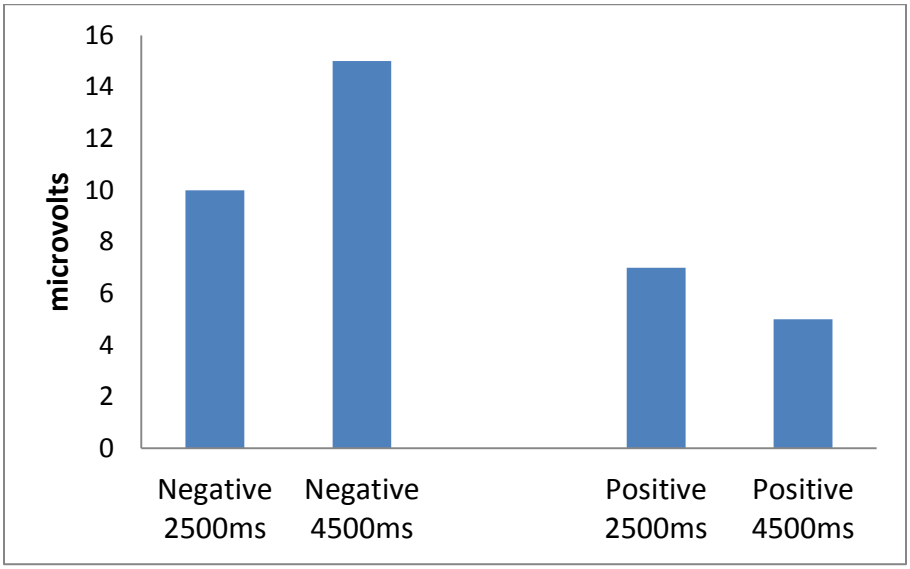


Figure 1. Affect modulated Startle Response

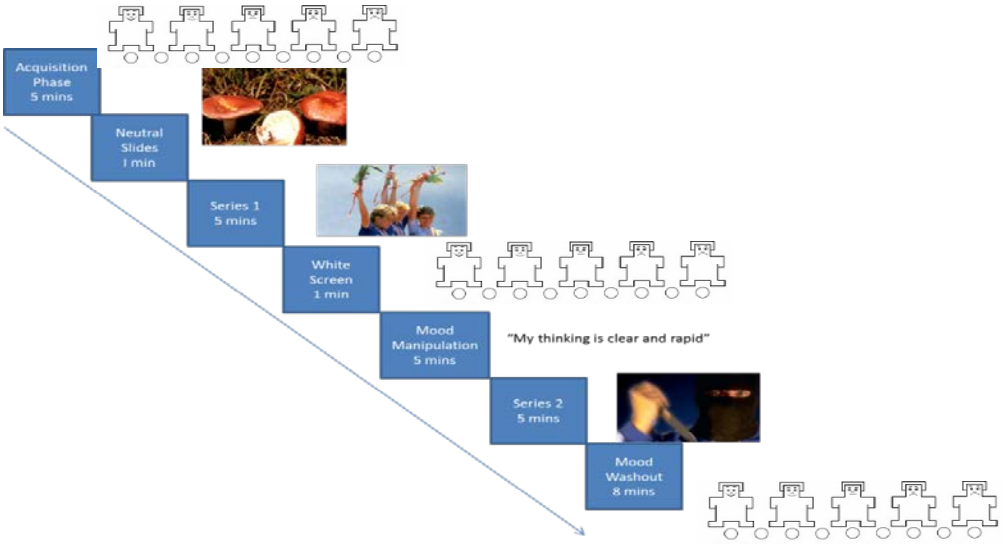


Figure 2. Order of Procedure. SAM Mood ratings were asked after each block of IAPS presentations, as well as after the mood induction.

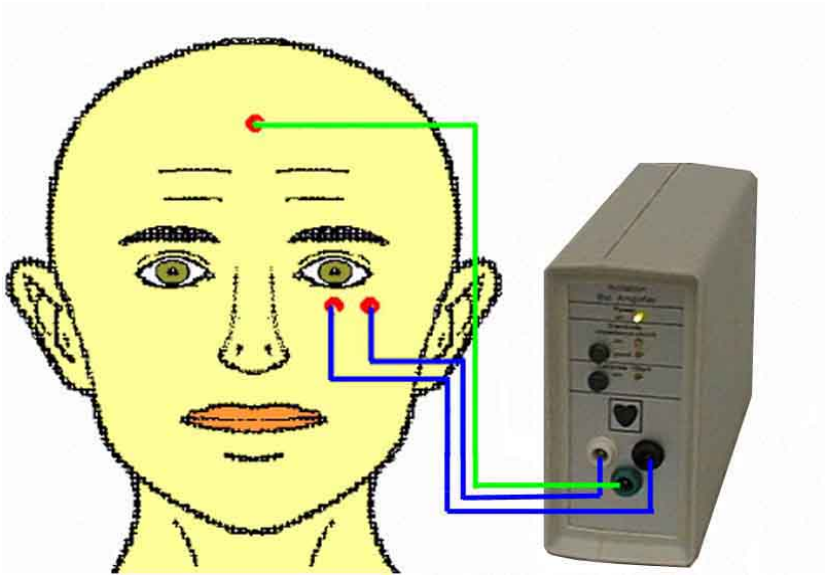


Figure 3a. Placement of Eyeblink Sensors.

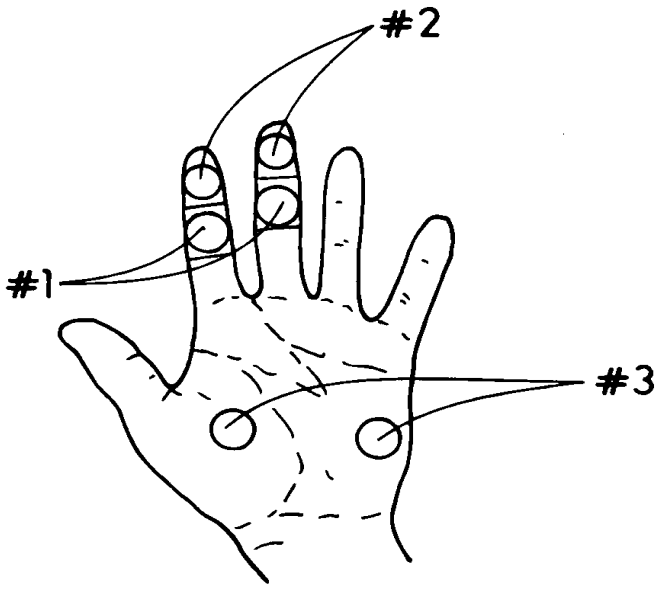


Figure 3. Three electrode placements for recording electrodermal activity. Placement #1 involves volar surfaces on medial phalanges, placement #2 involves volar surfaces of distal phalanges, and placement #3 involves thenar and hypothenar eminences of palms.

Figure 3b. Placement of SCL Sensors.

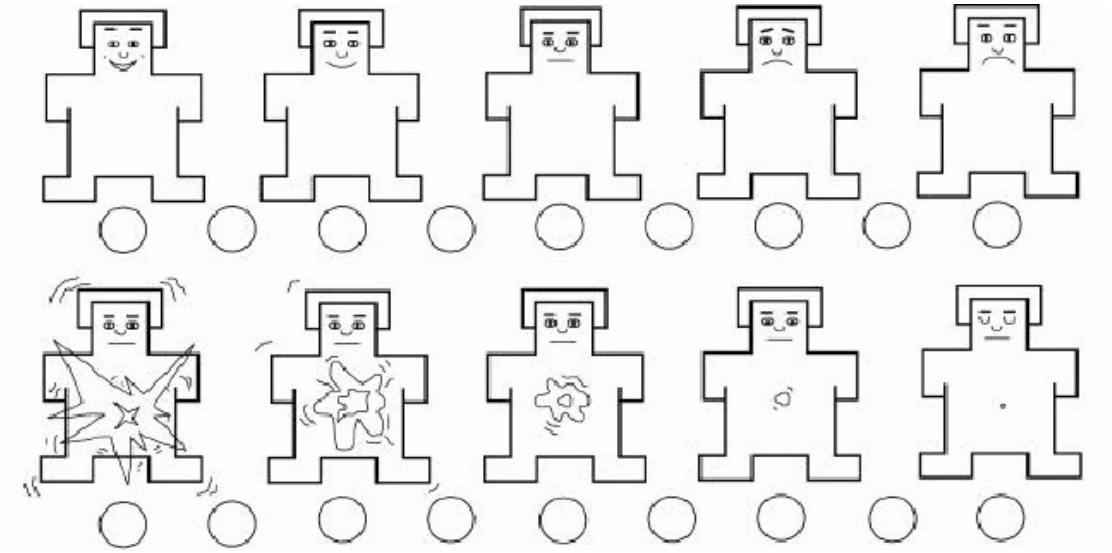


Figure 4. Self-Assessment Manikin

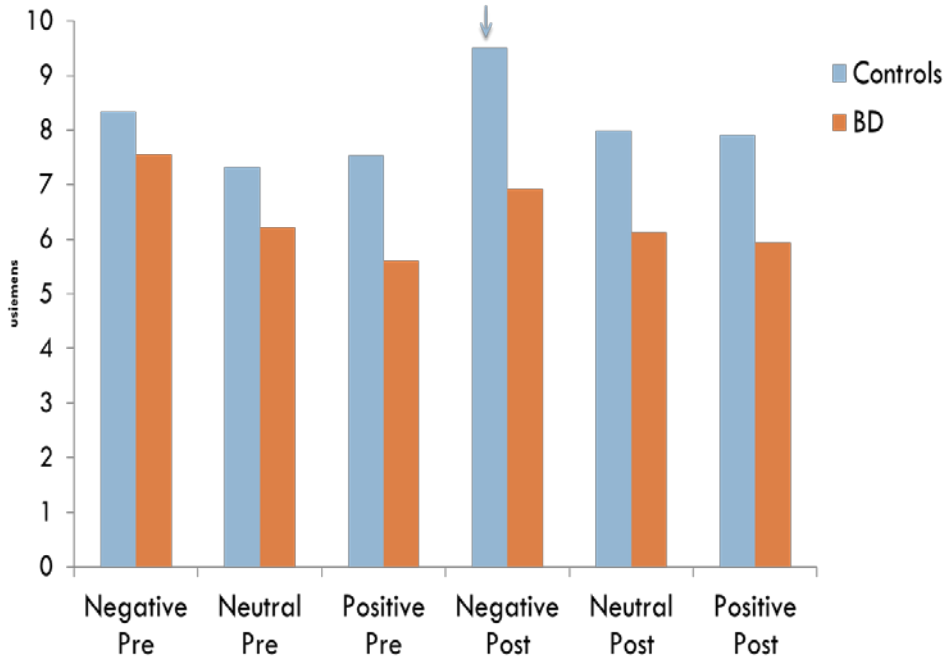


Figure 5. Three-way Interaction among pre-post, valence and group.

Appendices

Appendix A: Advertisement for BD participants

Have you been told that you have:
MANIA,
MANIC-DEPRESSION, or
BIPOLAR DISORDER?
The University of Miami is conducting a study to look at how moods relate to thinking.
The study involves an interview about mood changes and different computer tasks.

Participants will be paid $25 per hour.

To learn more please contact:
The Positive Moods Study at 305-284-2307

Appendix B: Advertisement for control participants

How do moods influence thinking?

We are conducting a study which involves an interview about mood changes, and computer games and tasks.

Participants must be native English speakers and between the ages of 18 and 65.
Participants will be paid $25 per hour.

To learn more please contact:

The Positive Moods Study at 305-284-2307

Appendix C: Phone screen

Positive Moods

Potential Participant's FIRST Name: _____ Date: ___ 2011

Phone Number: _____ Screened by: _____

Recommendations/Questions about eligibility:

1.HAVE THEY PREVIOUSLY BEEN ENROLLED IN COGGIE?

YES / NO

a. **GET SCID FILE ON DX CONFIRMATION- YES / NO**

b. **HC-GO OVER MODULES (SUB ABUSE); AND BRMS AND HAM-D**

c. **BIPOLAR-GO OVER SUB ABUSE MODULE, AND BRMS AND HAM-D**

EXCLUSION FOR BD (THEN SET UP CALL BACK LATER FOR SYMPTOMS)

GREATER than 10 on the BRMS SCORE_____

GREATER THAN 9 on the HAM-D
SCORE _____

Inclusion Criteria for BP I:
BP I / not currently in episode (but we will call to check status if so until we can schedule them)

Inclusion criteria for Healthy controls:
no lifetime mania or major depression. If you suspect subsyndromal mood disorders, better to rule out

Inclusion criteria for both:

1. age 18-65 . . . . .
2. **only native English speakers**
3. **no substance abuse in the past six months . .**
4. no primary psychotic disorder
5. no indications of impaired mental status or
6. developmental disabilities that would interfere
7. with task completion
8. no mood episodes secondary to general medical
9. conditions
10. no ECT within the past 18 months
11. no brain injury or medical conditions of the CNS
12. not color blind or dyslexic . . .
13. no HIV . . . . .

Hi, this is ____, calling from the University of Miami. Can I please speak with ____.

[If person's not available: OK, would it be possible for me to leave a message for him/her? Could you ask them to call me at 305-284-2307? [leave good times to reach]. I'm calling about the Positive Moods Study at UM (that you expressed interest in). Sound familiar? May I take a few minutes now to describe our study? [If no, reschedule at a convenient time].

Great, we really appreciate your interest in helping our research. This is a study designed to help us understand more about how mood changes are related to the way people think about positive and negative information. We aren't studying treatment as part of this study. Rather, this study just involves viewing pictures, computer tasks, interviews, and questionnaires, and does not involve or change treatment in any way.

Does that sound interesting to you? Would you like to hear more about how it works? The study has a few parts.

First, we would conduct a phone interview with you to see if the study is a good fit. This could take anywhere from 5 to 20 minutes. We will be asking you a series of questions on fairly sensitive matters including psychiatric history/symptoms, substance abuse and HIV/AIDS status. You have the right to refuse to answer any of our questions. Since we will be asking questions of a sensitive nature, we suggest that you are in a private place during the phone screen because we may ask you to provide us with more information on some of the issues above.

Is this okay with you?

If you are eligible, we would invite you in to UM to complete a more detailed interview and some questionnaires. That session can last up to two hours. During that session, you might listen to audiotapes, see pictures on the computer, fill in questionnaires, or complete other interviews.

Do you have any questions?

During the time that you spend here at UM, we pay $25 per hour. [we don't pay for phone interview time nor for transportation.] [We split into two sessions because we worry people will be exhausted, but if they want to schedule one of those sessions in the morning, and come back in the afternoon after a lunch break, or some such thing, that is no problem.]

The study does not involve any stressors, shocks, or things like that. In fact, you don't have to answer anything you don't want to. The toughest part is that we ask a lot of personal questions about mood changes, drug use, and things like that. Anything you don't want to answer, though, you don't have to. We are really thankful that you are interested in helping us, and so we want to make sure you felt comfortable with anything that we ask.

Do you have time for the brief interview now? It will take about half an hour. I would also like to note that, even if you are ineligible for this particular study, we are conducting other similar studies for which you may be eligible. If it is okay with you, we will use this phone screen to help determine eligibility for those other studies as well. Does that sound good to you?

A. HOW DID YOU HEAR ABOUT THIS STUDY? :

_____

- Be sure to indicate how they heard about the study as precisely as possible

1. Sex: (should be apparent) Male Female
2. What is your date of birth? _____/_____/_____ Age:_____

***NOTE**: Participants should be 18-65.

3. What is your race/ethnicity? (place check marks)

Ethnicity: ____ Hispanic/Latino _____ NOT Hispanic/Latino

Race: ____American Indian/Alaskan Native _____Asian _____Native Haiwaiian/Pacific Isl.

____Black African American ____White

3. Are you a native English speaker: YES NO

If NO: How long have you spoken English? _____

***NOTE**: If less than 10 years, **EXCLUDE.**

Ok, great. Now, I'm going to ask you several questions that are more sensitive and personal than the ones I've just asked. There are no right or wrong answers to any of these questions. Again, everything you say will be kept strictly confidential. There are a few legal limits to this confidentiality. If I thought you were going to hurt yourself, or hurt someone else, especially a child or elderly person, I might be required to report this information to the appropriate agency. You may choose to skip questions if you do not wish to answer them. Is it OK to continue?

I am now going to ask you some general health questions.

1. Have you ever received an injury or trauma to your head?
   YES NO
   Details:
   IF YES: ask when it occurred, and duration of Loss of Consciousness
   IF NO: ask separately about any accident that resulted in a Loss of Consciousness; get details
   INELIGIBLE if LOC > 5 min w/in past 12 months or LOC > 1 hr if beyond 1 year
2. Have you ever been diagnosed with any neurological disorder, such as Alzheimer's Disease, Parkinson's Disease, Huntington's Disease?
   YES NO
   Details:
   IF YES: what kind and duration:
3. Have you ever had a stroke, hemorrhage, or brain tumor?
   YES NO
   Details:
4. Have you ever had brain/neural surgery or brain radiation treatment (e.g. for brain tumor)?
YES NO
   Details:

5. Do you have seizures or Epilepsy?
   YES NO
   Details:
   IF YES: Ask about severity, frequency and medication
6. Do you have Dyslexia?
   YES NO
   - IF YES AND LIKELY BIPOLAR: RULE OUT OF STUDY
7. Are you color blind?
   YES NO
   - IF YES AND LIKELY BIPOLAR: RULE OUT OF STUDY
8. Do you have HIV or AIDS?
   YES NO
   - IF YES AND LIKELY BIPOLAR: RULE OUT OF STUDY

*****NOTE MEDICAL RULE OUTS ON FRONT OF INTERVIEW**
IF INELIGIBLE STOP INTERVIEW HERE.

9. In the past 18 months, have you had any electroconvulsive or shock treatments?

YES NO

*****IF YES: RULE OUT OF STUDY**

Lifetime (current or past) Manic Episode (pages 12 thru 14)

1. Feeling good or "high"

? 1 2 3

elevated, expansive mood ___ irritable mood___

if 1 go to NEXT MODULE

2. spent time ? 1 2 3 increase in activity ___ psychomotor agitation ___

2. How long

? 1 2 3

If 1 go to NEXT MODULE

4. how did you feel	?	1	2	3
5. less sleep	?	1	2	3
6. more talkative	?	1	2	3
7. thoughts racing	?	1	2	3
8. easily distracted	?	1	2	3
9. caused trouble	?	1	2	3

10. At least 3 "B" sxs are coded "3" (4 if mood only irritable) 1 3

If 1, ask number 11; If 3, go to number 12

11. any other time?___ if yes, return to past manic episode and inquire about worst episode; If no, go to past hypomania (below)

12. serious problems at home or work, or went to hospital 1 3

If 1, Any other episodes?

13. physically ill, or drugs/medications 1 2 3

IF MANIA HISTORY PRESENT: continue as potential bipolar

- **If mania criteria met in past month, arrange to follow up** in a few weeks so that we can catch them when they are euthymic

IF NO MANIA HISTORY: continue as potential control

Psychotic and Associated Symptoms

1 people talking about you	?	1	2	3
2 anyone giving hard time, or trying to hurt you	?	1	2	3
3 feeling especially important or with special powers	?	1	2	3
4 feeling something wrong physically	?	1	2	3
5a unusual religious experiences	?	1	2	3

5b delusions of guilt____ 5c control ____ 5d read mind_____ 5e broadcasting_____

Other delusions_____

AUDITORY HALLUCINATIONS

7. hearing things others couldn't hear	?	1	2	3
If 1, go to visual hallucinations				
8. comment on what you were doing or thinking	?	1	2	3
9. how many voices? Talking to each other?	?	1	2	3
(2 or more voices = 3)				

VISUAL HALLUCINATIONS

10. visual hallucinations	?	1	2	3
11. tactile hallucinations	?	1	2	3
12. other hallucinations	?	1	2	3

gustatory___ olfactory___

DIFFERENTIAL DIAGNOSIS OF PSYCHOTIC DISORDERS

1. Psychotic sxs more than 2 weeks at times other than during depressed/ manic episodes

? 1 2 3

IF 3, RULE OUT OF ALL STUDIES

LIMIT THE FOLLOWING TO THE PAST YEAR

SCREENING MODULE

P1.	Had >= 5 drinks during past year?	1	2	3
P2.	Used street drugs during past year?	1	2	3
P3.	Hooked on medicine during past year?	1	2	3

Substance Use Disorders- Alcohol Use Disorders (pages 23 thru 26)

If screening ques. #1 is "no" skip to non- alcohol substance use disorders ___

1a) What have drinking habits been like during this past year?

_____

When in the past year were you drinking the most? Record date, describe pattern.

_____

During that time, how often were you drinking, what, and how much?

_____

1b) During that time, did our drinking cause problems for you?_____

1c) Did anyone object to your drinking?_____

If no incidents of excessive drinking, go to non- alcohol substance use disorders.

Past year Alcohol Abuse

2. missed work or school because intoxicated or hung over	?	1	2	3
3. drinking in a dangerous situation	?	1	2	3
4. drinking has gotten you in trouble with law	?	1	2	3
5. drinking caused problems with other people	?	1	2	3

E6 At least one "A" item coded "3" 1 3

IF 3, RULE OUT, ASK ABOUT PREVIOUS 6 MONTHS FOR DISSERTATIONS

Past six months Alcohol Abuse

2b. missed work or school because intoxicated or hung over	?	1	2 3
3b. drinking in a dangerous situation	?	1	2 3
4b. drinking has gotten you in trouble with law	?	1	2 3
5b. drinking caused problems with other people	?	1	2 3
E6b. At least one "B" item coded "3"		1	3

IF 3, RULE OUT OF DISSERTATION STUDIES AS WELL AND DISCONTINUE SCREEN

If bipolar and alcohol dependence seem likely then refer to Dr. Salloum's team: let them know they can call 305-243-1298 for a treatment study on bipolar disorder and substance use

NON-ALCOHOL SUBSTANCE ABUSE DISORDERS
*****PAST SIX MONTHS ONLY*****

Screening ques. #2 yes___ no___ : screen Ques #3 yes___ no___. If no to both, skip module

code "1" if drug never used, used only once., or if prescribed drug used as directed
code "2" if used at least twice, but less than 10x in any month
code "3" if used at least 10x in any month or if possible dependent on prescribed drug

1. Ever taken any to get high /sleep better /lose weight /mood?

sedatives- hypnotics- anxiolytics	?	1	2	3
cannabis	?	1	2	3
stimulants	?	1	2	3
opioids	?	1	2	3
cocaine	?	1	2	3
hallucinogens/PCP	?	1	2	3
other	?	1	2	3
any drug groups coded "2" or "3"		1		3

2. period using many drugs, regardless of type, to get high	1	2	3

If 3, use poly drug column. If no drugs coded "2" or "3" then screen is complete; go to next page

Past year substance abuse

3. missed work or school because high or recovering ? 1 2 3
4. using in a dangerous situation ? 1 2 3
5. using/possessing has gotten you in trouble with law ? 1 2 3
6. using caused problems with other people ? 1 2 3

7. At least one "A" item coded "3"
1 3

If bipolar and cocaine dependence seem likely then refer to Dr. Salloum's team: let them know they can call 305-243-1298 for a treatment study on bipolar disorder and substance use

IF PREVIOUSLY DID SCID, GET MOOD SYMPTOMS. IF CAN RATE BASED UPON SCID INTERVIEW, DO IT.

BRMS

(11-item version, Bech, Bolwig, Kramp, & Rafaelsen, 1979)

1. **Activity (motor)**: *Over the past week, have there been times when it was hard for you to complete tasks or conversations because you were too restless? Did you have a lot of energy and felt very lively?*
    0. Normal motor activity, adequate facial expression.
    1. Slightly increased motor activity, lively facial expression.
    2. Somewhat excessive motor activity, lively gestures.
    3. Outright excessive motor activity, on the move most of the time.
    4. Constantly active, restless energetic. Even if urged, patient cannot sit still.
2. **Activity (verbal)**: *Have you had more to say or been any more talkative than usual? Have other people noticed? Have there been times when it was hard for someone to get a word in edgewise?*
    0. Normal verbal activity.
    1. Somewhat talkative.
    2. Very talkative, no spontaneous intervals in the conversation.
    3. Difficult to interrupt.
    4. Impossible to interrupt, dominates completely the conversation.

3. **Flight of thoughts**: *Have your thoughts been racing? Were there times when it was difficult for others to follow your conversation because your thoughts seemed to be racing? Have you found yourself jumping from topic to topic during conversation? Have you found yourself making rhymes or puns in your conversation?*
    0. Cohesive speech, no flight of thoughts.
    1. Lively descriptions, explanations and elaborations without losing connection with the topic of conversation. The speech is thus still cohesive.
    2. Now and again it is difficult for the patient to stick to the topic, as the patient is distracted by random associations (example: often rhymes, clangs, puns, pieces of verse or music).
    3. The line of thought is regularly disrupted by diversionary associations.
    4. It is difficult to impossible to follow the patient's line of thought, as the patient constantly jumps from one topic subject to another.

4. **Voice/Noise level**: *Have you been talking any more loudly? Were there any times when you were making more noise than usual? Have you been singing or shouting at all during this week?*
    0. Natural volume of voice.
    1. Speaks loudly without being noisy.
    2. Voice discernible at a distance, and somewhat noisy.
    3. Vociferous, voice discernible at a long distance, is noisy, singing.
    4. Shouting, screaming, or using other sources of noise due to hoarseness.

5. **Hostility/Destructiveness**: *Have you been any more impatient or irritable during this week? Have you had any arguments or conflicts with people? How bad was the argument? (Did it become heated enough for shouting, threats, or even physical conflict?)*
    0. No signs of impatience or hostility.
    1. Somewhat impatient or irritable, but control is maintained.
    2. Markedly impatient or irritable. Provocation badly tolerated.
    3. Provocative, makes threats, but can be calmed down.
    4. Overt physical violence. Physically destructive.

6. **Mood (feelings of well-being)**: *Would you say that over the past week you were in a very good mood? Were you more happy or cheerful than usual? Have there been times when you seemed to be in a better mood than the circumstances or than people around you? Have you been laughing or joking more than usual?*
    0. Neutral mood.
    1. Slightly elevated mood, optimistic, but still adapted to situation.
    2. Moderately elevated mood, joking, laughing.
    3. Markedly elevated mood, exuberant both in manner and speech?
    4. Extremely elevated mood, quite irrelevant to situation. Mood was expressed behaviorally.

7. **Self-esteem**: *How have you been feeling about yourself? Would you say you felt more self-confident? Did you feel that you had any special powers or abilities?*
    0. Normal self-esteem.
    1. Slightly increased self-esteem, slightly boasting.
    2. Moderately increased self-esteem, boasting. Frequent use of superlatives.
    3. Bragging, unrealistic ideas.
    4. Grandiose ideas which cannot be corrected.

8. **Contact**: *Have you found yourself involved in any one else's concerns, work, or relationships? Have you been any more likely to give others advice?*
    0. Normal contact.
    1. Slightly meddling, putting his oar in.
    2. Moderately meddling and arguing.
    3. Dominating, arranging, directing, but still in context with the setting.
    4. Extremely dominating and manipulating, without context with the setting.

9. **Sleep (average of last 3 nights)**: *How much have you been sleeping in the last 3 nights? Did you still feel rested, despite getting very little sleep?*
    0. Habitual duration of sleep.
    1. Duration of sleep reduced by 25%.
    2. Duration of sleep reduced by 50%.
    3. Duration of sleep reduced by 75%.
    4. No sleep.

10. **Sexual interest**: *Have you been thinking more about sex or more interested in sex?*
    0. Habitual sexual interest and activity.
    1. Slight increase in sexual interest and activity.
    2. Moderate increase in sexual interest and activity.
    3. Marked increase in sexual interest and activity, as shown in manner and speech.
    4. Completely and inadequately occupied by sexuality.

11. **Work**: *Have you been working over the past week? How about having to do daily tasks or chores, like making your bed, keeping your room clean, or participating in ward activities? Have there been any difficulties with these jobs? Have you been able to accomplish the tasks required? Have you had difficulties with concentration or attention while trying to complete your jobs? Have you had a lot of projects going at once?* [DO NOT RATE WORK IMPAIRMENT DUE TO DEPRESSION]
    0. Normal work activity.
    1. Slightly increased drive, but work quality is slightly reduced, as motivation is changing, and the patient is somewhat distractible
    2. Increased drive, but motivation clearly fluctuating. The patient has difficulties in judging own work quality and the quality is indeed lowered. Often quarrels at work.

3. Work capacity clearly reduced, and from time to time the patient loses control. Has to stop work and be sick listed. If the patient is hospitalized, he can participate for some hours per day in ward activities.
4. The patient is (or ought to be) hospitalized and unable to participate in ward activities.

TOTAL_____

MODIFIED HAMILTON RATING SCALE for DEPRESSION

---

1. ** DEPRESSED MOOD

How have you been feeling recently? Have you felt low in spirits, gloomy, or depressed? What percentage of time over the past week have you felt this way?

0. Absent
1. Mild-gloomy attitude, *may* be accompanied by infrequent weeping spells, sad, blue, waning of interests
2. Moderate-may be accompanied by feelings of inadequacy, self-pity, worrying, decrease in social interests and activity level, pessimism, "locked in", occasional weeping, apathy, decrease in experience of pleasure
3. Severe-may be characterized by hopelessness, greater tendency to withdraw socially, near absence of interest or participation in other than essential activities, hardly anything produces pleasure, weeping may be frequent (or beyond tears)
4. Extreme symptoms-complete withdrawal

9. Can't rate

6. ** GUILT

Are you critical of yourself for your weaknesses or mistakes? Do you blame yourself for things that go wrong around you even if others seem to think that you didn't have anything to do with them? Do you think your present illness is some type of punishment for something? Do you hear voices threatening or accusing you?

0. Absent
1. Feelings of self-reproach, self-blame, specific instance of lapse
2. Thoughts that negative events or reactions were caused by oneself; general or many instances or lapses for which one feels guilty; stronger convictions of one's guilt
3. Belief that illness might be a punishment, possibly delusional guilt
4. Delusional guilt, *with* hallucinations

9. Can't rate

***9. SUICIDE**

Do you feel that life is worth living?

Do you wish you were dead?

Do you have thoughts of committing suicide?

Have you tried to kill yourself?

0. Absent
1. Feels life is not worth living
2. Wishes he were dead or any thoughts of possible death to himself
3. Suicidal ideas, gestures, or plans
4. Attempted suicide (any serious attempt rated 4)

9. Can't rate

10. INSOMNIA

Do you have trouble sleeping?

*** a. Early**

Do you have trouble falling asleep? How long does it take you to fall asleep? How often?

0. Absent
1. Occasional (fewer than 3 days a week), mild, trivial (less than 1 hour delay)
2. Frequent (3 or more times per week) and severe (1 hour or more delay)
9. Can't rate

*** b. Middle**

Once you get to sleep do you wake up during the night? What do you do when you wake up? Can you get back to sleep?

0. Absent
1. Occasional (fewer than 3 days a week), mild (less than 1 hour delay in returning to sleep)
2. Frequent (several times per night with difficulty returning to sleep, 3 or more times per week) and severe (1 hour or more to return to sleep)
9. Can't rate

*** c. Late**

Do you wake up earlier than your usual time (before onset of depression) in the morning? Can you go back to sleep?

0. Absent
1. Occasional (fewer than 3 days a week), mild (less than 1 hour early)
2. Frequent (3 or more days per week) and severe (1 hour or more early)
9. Can't rate

11. APPETITE AND WEIGHT

*** a. Loss of appetite**

How is your appetite compared to the way it usually is? Do you have trouble with constipation or other problems with your stomach or bowels?

0. Absent

1. Loss of appetite, mild or occasional
2. Loss of appetite, severe or constant constipation
9. Can't rate

*** b. Loss of weight**

Over the *past month*, when not dieting, have you lost any weight?

0. Absent
1. One or 2 pounds or more over the past month
2. Three pounds or more over the past month
9. Can't rate

*** 12. a. LOSS OF ENERGY**

Have you had less energy than usual, or have you been getting tired more easily? How has this affected your work or other activities? Have your back, head, or limbs felt heavy or ached?

0. No loss of energy
1. Subjective loss of energy or feelings of tiredness
2. Marked interference with functioning (decrease in work and activities) OR feelings of heaviness or achiness
9. Can't rate

*** c. WORK AND ACTIVITIES**

Do you find that you have trouble doing things you really need to do (e.g. job, housework, studies)? How has this decreased interest in things and/or people affected your life?

0. Absent
1. Somewhat decreased efficiency, effortfulness;and/or decreased interest in or gets less pleasure from hobbies, interests, social contacts
2. Decreased performance, neglects or delays some things; withdraws from unnecessary activity, decreased participation in hobbies, social events
3. Considerably diminished performances of work or routine activities, more things are neglected or postponed indefinitely, virtually unproductive; avoids social contacts, nothing seems pleasurable, no interests
4. Unable to work, nonproductive, completely immobilized
9. Can't rate

*** d. LOSS OF LIBIDO**

Over the past *month* has there been any change in your interest in sex? Does this represent a change from the way you usually feel about sex?

0. No change
1. Some loss of interest and performance
2. Almost total loss of interest and sexual activity
9. Can't rate

*** 13. ANXIETY**

a. Psychic anxiety - anxious, tense, jittery, nervous, restless, "up tight", apprehensive, frightened, scared, irritable, worrying

Have there been times lately that you felt very anxious or frightened? Are these feelings fleeting, do they occur for a while and then go, or are they continuous? What percentage of time over the past week would you say that you have felt this way? What kinds of situations do you feel anxious in? Have you been in any situation where you were so anxious that you simply had to get out, run, or do something else about it?

0. Absent
1. Transient tension, occasional irritability, mild exaggeration of worrying
2. Fairly constant tension, more frequent irritability, somewhat "hyper" or jittery.
3. Pervasive apprehension, tension, irritability, constant ruminative worrying
4. Panic attacks; phobias restrict activity

9. Can't rate

*** b. Somatic anxiety**

Symptoms are rated on the basis of the report of symptoms in the following systems:
(a) respiratory: labored breathing, shortness of breath, smothering or choking feelings, etc.;
(b) cardiovascular, flushing, accelerated heart rate, palpitations, faintness, chest pain or discomfort, etc.;
(c) gastrointestinal: indigestion, stomach upset, heartburn, stomach cramps, diarrhea, etc.
(d) genito-urinary frequency;
(e) sweating;
(f) giddiness, blurred vision, tinnitus;
(g) neuromuscular, trembling or shaking, headaches, muscle tension, dizziness, tingling, etc.

When you felt anxious, what was it like?
Did you notice your heart beating faster?
(Ask about other somatic symptoms noted above-circle each symptom that is present)
Have these bodily changes hindered your performance in any way?

0. Absent
1. Mild-one or more symptoms, complains of some discomfort but continues to participate in daily activities.
2. Moderate-e.g., symptoms from more than 1 system, occasionally patient can't take part in activities because of *bodily discomfort*
3. Severe-symptoms so uncomfortable that patient frequently has trouble taking part in activities
4. Extreme-multiple symptoms that are incapacitating, i.e., bodily discomfort precludes taking part in any activities

9. Can't rate

*** 14. HYPOCHONDRIASIS**

How is your physical health?

Do you tend to worry about your health?

Are you so concerned with your health that you find it hard to think about other things? (Important: Interviewer is evaluating the extent to which the patient focuses on physical health to the exclusion of others symptoms)

0. Absent
1. Preoccupation with health, bodily, function, trivial or doubtful symptoms
2. Much preoccupation with physical symptoms, thoughts of organic disease
3. Strong conviction of presence of physical disease, querulous attitude
4. Hypochondriacal delusions and hallucinations, e.g. rotting, blockages, etc.
9. Can't rate

*** 15. INSIGHT** (patient background should be taken into account)

Do you think there is anything the matter with you? What do you think it is? Could it be that you have emotional problems?

0. Acknowledges being depressed or ill
1. Acknowledges illness but attributes cause to unlikely factors, e.g. bad food, climate, overwork, etc.
2. Denies being ill
9. Can't rate

*** 16. RETARDATION (direct observation)**

0. Absent
1. Slight retardation at interview; flattening of affect and fixity of expression
2. Obvious retardation at interview; monotonous voice; delay in answering, motionless
3. Interview difficult, prolonged
4. Complete stupor
9. Can't rate

*** 17. AGITATION (direct observation)**

0. Absent
1. Low level of agitation, fidgeting, obvious restlessness (e.g. picking at hands or clothing, leg movements) for large proportion of interview
2. High level of agitation, includes fidgeting, obvious restlessness *as well as* the patient getting up during the interview, pacing, etc.
9. Can't rate

TOTAL SCORE: _____

For those who are **ELIGIBLE** but who have **MOOD SYMPTOMS THAT WOULD INTERFERE WITH COMPLETING A SCID:**

Thank you very much for your time and for answering these questions. We are interested in working with you.
We would like to invite you to come in to meet with a trained graduate student to complete an interview.
Because you are describing being a bit [high/low/unfocused/whatever their words are], we'd also like to stay in touch with you to see how you are feeling over the next few months until you are having fewer symptoms

Are you currently being treated by a doctor for the way that you have been feeling?
YES NO
Has your doctor made any changes to your treatment regimen recently? YES
NO

Schedule a time to talk with them about how they are feeling.

For people **NOT ELIGIBLE:**

Thank you very much for your time and for answering these questions. Based on what you told me, it does not seem like this study is a good fit for you. If, however, you are interested in research in general, we would be happy to keep your name and number for future studies done here at UM. Would that be okay with you? You have been so great at answering these questions, that if we have a good study, I want to give you a call.

If they ask for feedback: say, it is not really based on any one issue-- we look at a profile of different issues.

For people who are **ELIGIBLE**

Thank you very much for your time and for answering these questions. We would love to schedule you to come in for the first session of the study. This session will last about ONE TO ONE HALF hours and involves an interview and filling out some questionnaires. We also ask that you bring with you a list of all medications that you are taking along with the dosage. What times might work for you?

DIRECTIONS
Do you need directions to the campus? If so, how will you be coming?

Flipse Building
5665 Ponce De Leon Drive
Coral Gables, FL 33146-0751

If you plan to take the Metrorail:
Our stop is University Station. After you get off the train, cross the street in front of you, which is Ponce de Leon. There is a shuttle stop that will take you to the Ponce Garage. If you choose to walk from the Metrorail, turn left after crossing the street. The Ponce de Leon parking garage is the first building on your right hand side.

If you plan to drive:
From the North: Take I-95 South to US1. Stay on US1 until South Alhambra Circle. Make a right. You will be facing the Ponce Garage. The Flipse Building is attached to the garage.
From the South: Take US1 North to South Alhambra Circle. Make a left. You will be facing the Ponce Garage. The Flipse Building is attached to the garage.

CANNOT COMPENSATE FOR PARKING. ONLY NEED TO PAY METERS BEFORE 4PM!

CALL 305-284-2307 IF RUNNING BEHIND OR IF THEY GOT LOST.

Appendix D: Consent

Positive Moods

PURPOSE:

The purpose of this study is to understand how people with and without bipolar disorder think when in a positive mood. We would like to interview you about your moods and mood symptoms. We would also like to have you fill out some questionnaires and do some simple computer tasks.

PROCEDURES:

The interview about your mood will take up to an hour and a half to complete. The interview will involve asking you questions about your moods and experiences in the past, including how your sleep, energy, and thoughts change with moods. It will also include questions about your experiences of anxiety, alcohol and drug use, and other related topics. Based on this interview, we will decide whether you are eligible for other parts of the study.

Depending on your responses, we may continue with the next part of the study, or we may want to wait until you are in a different mood state. If we wait, we will contact you once a month by phone for up to one year to see how you are feeling, and when your mood is in a certain range, we will invite you to continue with the study. These phone interviews can take between 5 and 20 minutes.

As soon as your mood is in our target range, we will schedule you to come to the University of Miami for a testing session that involves a computer task. At the start of this session, we will interview you about how you are feeling. Then, we will ask you to look at different pictures on the screen. Some of these pictures may be distressing to you, and you have the right to stop this task if viewing the pictures becomes too distressing to you. During this task, you will be videotaped, and we will measure your eyeblinks and skin conductance. Last, we will ask you to complete some self-report questionnaires about your mood. The session at the University will take approximately 1 to 1.5 hours.

We will audiotape your initial interview and one of the computer tasks, and we will videotape the picture viewing task. If you ask us to erase your audiotapes or videotapes, we will do so. We will erase all of the audiotapes in this study within 5 years, when we will have finished the study.

RISKS:

There may be some risk from these procedures. We will ask questions about your feelings and behaviors. Some of the questions cover experiences that may be very

personal or stressful experiences, such as some questions about suicidal thoughts. You have the right at any point during the study to skip questions you do not want to answer or stop the interview entirely. If you are distressed, we will help you to find treatment options in the Miami area that are affordable for you. You will also be asked to view some pictures that may be distressing to you. If you feel uncomfortable viewing the pictures, you can ask to skip them and you may continue with the study without penalty.

BENFITS:
No direct benefit can be promised to you for taking part in this study.

COMPENSATION:
We will pay you $25 per hour for your time at the University of Miami.

ALTERNATIVES:
You have the alternative not to participate in this study. While you are being interviewed, viewing the pictures or engaging in any other tasks for the study, you can decide to stop at any time. Nothing bad will happen to you if you choose not to complete the study.

CONFIDENTIALITY:
The investigators and their assistants will consider your records confidential to the extent permitted by law, except that if any information collected indicates the immediate potential for harm to you, your child, or others, the investigators and or their staff or associates may be required to disclose such information. If it seems that you might be likely to hurt yourself or others, study staff will coordinate contact with emergency services, calling the police if necessary. The U.S. Department of Health and Human Services (DHHS) may request to review and obtain copies of your records. Your records may also be reviewed for audit purposes by authorized University employees or other agents who will be bound by the same provisions of confidentiality. Study files will be maintained in a locked office, and electronic files will be maintained on a secure server with password protection. The videotapes will be password protected in a locked office and will be destroyed at the end of this study.

RIGHT TO WITHDRAW:
You have the right to withdraw at any time. Your desire not to participate, or your request to withdraw from the study, will not affect your standing with the University.
OTHER PERTINENT INFORMATION:
You may ask and will receive answers to any questions during the course of the study. If you have any questions about this study, please contact Charles Carver, PhD at 305-

284-2817. If you have questions about your rights as a research participant you may contact the Human Subjects Research Office, at (305) 243-3195.

We will give you a copy of this form for your records. If you are willing to be in this study, please sign below.

_____	_____
Signature	Date
_____	_____
Person Obtaining Consent	Date
_____	_____
Print Name of Person Obtaining Consent	Date

Principal Investigator: Charles Carver, PhD
Telephone: 305-284-2817

Appendix E: Checklist

Subject ID: _____ Date: _____
EXPERIMENTER:_____

TIME ARRIVED: _____

TIME ENDED:_____

Left handed/ Right handed

TIME STARTED PSYCHOPHYSIOLOGICAL STUDY: _____

1A. BRMS #ON PHONE
1B. HAM-D # ON PHONE
TOTAL_____ DATE_____
TOTAL_____ DATE_____
SCHEDULED SESSION DATE:

SESSION:
1. Call Carmen, 305.284.7345, an hour before session and let her know that a participant will be showing up. Meet participant in 2nd floor reception area.
2. Walk them up to the 4th floor, room 467.
3. Give them a copy of the consent form to review and read over. Answer any questions, then sign and collect the forms. IMPORTANT: Store the consent forms separately from all other forms and make sure the participant number is on all forms (except consent form).
4. Have them complete W-9 form.
5. Ask participant to turn off their cell phone.
6. Complete SCID (if needed), BRMS, HAM-D, and Medication Screener.
7. Have participant complete ARMS, and MINI-MASQ.
8. Make sure under mood symptom cut offs.

FORMS:

1. CONSENTED		**YES**	**NO**
2. GIVE EXTRA COPY OF CONSENT		**YES**	**NO**
3. SIGN W-9 FORM		**YES**	**NO**
4A. BRMS # SESSION	TOTAL_____		
4B. HAM-D # SESSION	TOTAL_____		
4C. ARMS# SESSION			

4D. MASQ # SESSION

TOTAL_____ GD_____ AA_____

AD_____

4. **SCID Screening Questions YES NO**
5. **SCID + DEMOG FILE YES NO—**

INELIGIBLE or On FILE

6. **MEDICATION SCREEN YES NO**
7. **PSYCHOPHYSIOLOGICAL STUDY**

- **(saved EPRIME FILE WITH PT # and moved to data file)**
  - **YES NO**
- **(saved PSYCHOPHYS FILE WITH PT #) YES NO**

8. **SELF-REPORT PACKET (CONFIRM ALL FORMS COMPLETED)**

YES NO

(have #1-6)

9. **PAYMENT (round to 15 mins) YES NO**
10. **Slipped PAYMENT FORM under Jen's door YES NO**
11. **PUT PARTICIPANT FILE FOLDER IN CABINET YES NO**
    (put CONSENT form, W-9 form in separate folders)
12. **CALL PARTICIPANT NEXT DAY TO CHECK IN YES NO**

TIME CALLED PARTICIPANT_____

EXPERIMENTER _____

Appendix F: Demographic Form

Age_____

Sex

1. male 2. female

Place of Birth _____

Primary language _____

Bilingual? Y N

Age moved to U.S.? _____

Number of years in U.S.? _____

Marital Status _____

1 single
2 cohabitating
3 married
4 remarried
5 divorced
6 widowed
7 separated

Number of Years for Current Marital Status _____

Education _____

12 high school graduate
16 college graduate
18 advanced degree

Occupation _____

Hollingshead occupational code _____

1 higher executives
2 business managers
3 administrative personnel
4 clerical and sales workers
5 skilled manual employees
6 machine operators
7 unskilled employees

Employment _____

1 Full time
2 Part time
3 homemaker
4 student
5 laid off
6 disability
7 retired
8 leave of absence
9 unemployed

Ethnicity

1 hispanic/latino
2 not hispanic/latino

Race: _____

1 american indian or alaskan native
2 asian
3 native haiwaiin or other pacific islander
4 black or african american
5 white

Appendix G: Medication List

CURRENT MEDICATION SCREEN

Are you currently taking any medications? YES NO

(Mark below any they endorse, and be sure to ask about each medication individually *even if they say no*.)

Trade name	**Generic names**	**DOSAGE (LIST # OF TIMES/DAY)**	**TAKING SINCE (MONTH/YEAR)**	**% MISSED**
SSRIs or Antidepressants				
Second/third generation antipsychotics				
Clozaril	clozapine			
Zyprexa	olanzapine			
Risperdal	risperidone			
Seroquel	quetiapine			
Geodon	ziprasidone			
Abilify	aripiprazole			
First-generation antipsychotics				
Haldol	haloperidol			
Thorazine	chlorpromazine			
Prolixin	fluphenazine			
Trilafon	perphenazine			
Mellaril	thioridazine			
Are you taking any other medications? If the participant names other drugs beyond those coded above, list them below:				

Benzo or anti-anxiety			

OTHER SUBSTANCES	AMOUNT (CUPS/BOTTLES)	LAST TAKEN	HOW OFTEN PER WEEK/ HOW LONG?
CAFFEINE			
NICOTINE			
ALCOHOL			
COLD MEDICATIONS			
Any other:			

Appendix H: Reverse Digit Span

Start: Trial 1 of Item 1
Discontinue: After a score of 0 on ***both*** trials of any item
Experimenter Guidelines: Say items one digit per second dropping voice inflection slightly on the last digit of the sequence. Then pause to allow response. If a subject asks you to repeat an item say: **Just take your best guess.**
Instructions

Say: **Now I am going to say some numbers. When I stop, I want you to say them backward. For example, if I say 7-1-9, what would you say?**

If the examinee responds correctly (9-1-7) say: **That's right**

Proceed to Trial 1 of Item 1. However if the examinee responds incorrectly, provide the correct response and say:

No, you would say 9-1-7. I said 7-1-9, so to say it backward, you would say 9-1-7. Now try these numbers. Remember, you are to say them backwards: 3-4-8.

Digits Backward Trial Item/Response		Trial Score	Item Score (0, 1, or 2)
1	1 2-4		
	2 5-7		
2	1 6-2-9		
	2 4-1-5		
3	1 3-2-7-9		
	2 4-9-6-8		
4	1 1-5-2-8-6		
	2 6-1-8-4-3		
5	1 5-3-9-4-1-8		
	2 7-2-4-8-5-6		
6	1 8-1-2-9-3-6-5		
	2 4-7-3-9-1-2-8		
7	1 9-4-3-7-6-2-5-8		
	2 7-2-8-1-9-6-5-3		
		Digits Backward Total Score (Maximum = 14)	

Do not provide any assistance on this example or any of the items. Whether or not the examinee responds correctly (i.e., 8-4-3), proceed to Trial 1 of Item 1.

Score each item 0, 1, or 2 points as follows:

- 2 points if the examinee passes both trials
- 1 point if the examinee passes only one trial
- 0 points if the examinee fails both trials

Appendix K: The Altman Self-Rating Mania Scale

On this questionnaire are groups of 5 statements. Read each statement carefully. Choose the one statement in each group that best describes how you have been feeling for the **past week.**

Please note.
The word '**occasionally**' here means once or twice;
'**often**' means several times or more;
'**frequently**' means most of the time.

1. A. I do not feel happier or more cheerful than usual.
   B. I occasionally feel happier or more cheerful than usual.
   C. I often feel happier or more cheerful than usual.
   D. I feel happier or more cheerful than usual most of the time.
   E. I feel happier or more cheerful than usual all of the time.

2. A. I do not feel more self-confident than usual.
   B. I occasionally feel more self-confident than usual.
   C. I often feel more self-confident than usual.
   D. I feel more self-confident than usual most of the time.
   E. I feel more self-confident than usual all of the time.

3. A. I do not need less sleep than usual.
   B. I occasionally need less sleep than usual.
   C. I often need less sleep than usual.
   D. I frequently need less sleep than usual.
   E. I can go all day and night without any sleep and still not feel tired.

4. A. I do not talk more than usual.
   B. I occasionally talk more than usual.
   C. I often talk more than usual.
   D. I frequently talk more than usual.
   E. I talk constantly and cannot be interrupted.

5. A. I have not been more active (either socially, sexually, at work, home, or school) than usual.
   B. I have occasionally been more active than usual.
   C. I have often been more active than usual.
   D. I have frequently been more active than usual.
   E. I am constantly active or on the go all the time.

Appendix L: Bech-Rafaelson Mania Scale

BRMS

(11-item version, Bech, Bolwig, Kramp, & Rafaelsen, 1979)

1. **Activity (motor)**: *Over the past week, have there been times when it was hard for you to complete tasks or conversations because you were too restless? Did you have a lot of energy and felt very lively?*
    0. Normal motor activity, adequate facial expression.
    1. Slightly increased motor activity, lively facial expression.
    2. Somewhat excessive motor activity, lively gestures.
    3. Outright excessive motor activity, on the move most of the time.
    4. Constantly active, restless energetic. Even if urged, patient cannot sit still.

2. **Activity (verbal)**: *Have you had more to say or been any more talkative than usual? Have other people noticed? Have there been times when it was hard for someone to get a word in edgewise?*
    0. Normal verbal activity.
    1. Somewhat talkative.
    2. Very talkative, no spontaneous intervals in the conversation.
    3. Difficult to interrupt.
    4. Impossible to interrupt, dominates completely the conversation.

3. **Flight of thoughts**: *Have your thoughts been racing? Were there times when it was difficult for others to follow your conversation because your thoughts seemed to be racing? Have you found yourself jumping from topic to topic during conversation? Have you found yourself making rhymes or puns in your conversation?*
    0. Cohesive speech, no flight of thoughts.
    1. Lively descriptions, explanations and elaborations without losing connection with the topic of conversation. The speech is thus still cohesive.
    2. Now and again it is difficult for the patient to stick to the topic, as the patient is distracted by random associations (example: often rhymes, clangs, puns, pieces of verse or music).
    3. The line of thought is regularly disrupted by diversionary associations.
    4. It is difficult to impossible to follow the patient's line of thought, as the patient constantly jumps from one topic subject to another.

4. **Voice/Noise level**: *Have you been talking any more loudly? Were there any times when you were making more noise than usual? Have you been singing or shouting at all during this week?*
    0. Natural volume of voice.
    1. Speaks loudly without being noisy.
    2. Voice discernible at a distance, and somewhat noisy.
    3. Vociferous, voice discernible at a long distance, is noisy, singing.

4. Shouting, screaming, or using other sources of noise due to hoarseness.

5. **Hostility/Destructiveness**: *Have you been any more impatient or irritable during this week? Have you had any arguments or conflicts with people? How bad was the argument? (Did it become heated enough for shouting, threats, or even physical conflict?)*
    0. No signs of impatience or hostility.
    1. Somewhat impatient or irritable, but control is maintained.
    2. Markedly impatient or irritable. Provocation badly tolerated.
    3. Provocative, makes threats, but can be calmed down.
    4. Overt physical violence. Physically destructive.

6. **Mood (feelings of well-being)**: *Would you say that over the past week you were in a very good mood? Were you more happy or cheerful than usual? Have there been times when you seemed to be in a better mood than the circumstances or than people around you? Have you been laughing or joking more than usual?*
    0. Neutral mood.
    1. Slightly elevated mood, optimistic, but still adapted to situation.
    2. Moderately elevated mood, joking, laughing.
    3. Markedly elevated mood, exuberant both in manner and speech?
    4. Extremely elevated mood, quite irrelevant to situation. Mood was expressed behaviorally

7. **Self-esteem**: *How have you been feeling about yourself? Would you say you felt more self-confident? Did you feel that you had any special powers or abilities?*
    0. Normal self-esteem.
    1. Slightly increased self-esteem, slightly boasting.
    2. Moderately increased self-esteem, boasting. Frequent use of superlatives.
    3. Bragging, unrealistic ideas.
    4. Grandiose ideas which cannot be corrected.

8. **Contact**: *Have you found yourself involved in any one else's concerns, work, or relationships? Have you been any more likely to give others advice?*
    0. Normal contact.
    1. Slightly meddling, putting his oar in.
    2. Moderately meddling and arguing.
    3. Dominating, arranging, directing, but still in context with the setting.
    4. Extremely dominating and manipulating, without context with the setting.

9. **Sleep (average of last 3 nights)**: *How much have you been sleeping in the last 3 nights? Did you still feel rested, despite getting very little sleep?*
    0. Habitual duration of sleep.
    1. Duration of sleep reduced by 25%.
    2. Duration of sleep reduced by 50%.
    3. Duration of sleep reduced by 75%.
    4. No sleep.

10. **Sexual interest**: *Have you been thinking more about sex or more interested in sex?*
    0. Habitual sexual interest and activity.
    1. Slight increase in sexual interest and activity.
    2. Moderate increase in sexual interest and activity.
    3. Marked increase in sexual interest and activity, as shown in manner and speech.
    4. Completely and inadequately occupied by sexuality.

11. **Work**: *Have you been working over the past week? How about having to do daily tasks or chores, like making your bed, keeping your room clean, or participating in ward activities? Have there been any difficulties with these jobs? Have you been able to accomplish the tasks required? Have you had difficulties with concentration or attention while trying to complete your jobs? Have you had a lot of projects going at once?* [DO NOT RATE WORK IMPAIRMENT DUE TO DEPRESSION]
    0. Normal work activity.
    1. Slightly increased drive, but work quality is slightly reduced, as motivation is changing, and the patient is somewhat distractible
    2. Increased drive, but motivation clearly fluctuating. The patient has difficulties in judging own work quality and the quality is indeed lowered. Often quarrels at work.
    3. Work capacity clearly reduced, and from time to time the patient loses control. Has to stop work and be sick listed. If the patient is hospitalized, he can participate for some hours per day in ward activities.
    4. The patient is (or ought to be) hospitalized and unable to participate in ward activities.

TOTAL_____

Appendix M: Mood and Anxiety Symptoms Questionnaire

Below is a list of feelings, sensations, problems, and experiences that people sometimes have. Read each item and then mark the appropriate choice. Use the choice that best describes how much you have felt or experienced things this way **this past week, including today**. Use this scale when answering:

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

1.	Felt really happy	1	2	3	4	5
2.	Felt tense or “high strung”	1	2	3	4	5
3.	Felt depressed	1	2	3	4	5
4.	Was short of breath	1	2	3	4	5
5.	Felt withdrawn from other people	1	2	3	4	5
6.	Felt dizzy or lightheaded	1	2	3	4	5
7.	Felt hopeless	1	2	3	4	5
8.	Hands were cold or sweaty	1	2	3	4	5
9.	Felt like I had a lot to look forward to	1	2	3	4	5
10.	Hands were shaky	1	2	3	4	5
11.	Felt like nothing was very enjoyable	1	2	3	4	5
12.	Felt keyed up, “on edge”	1	2	3	4	5
13.	Felt worthless	1	2	3	4	5
14.	Had trouble swallowing	1	2	3	4	5
15.	Felt like I had a lot of interesting things to do	1	2	3	4	5
16.	Had hot or cold spells	1	2	3	4	5
17.	Felt like a failure	1	2	3	4	5
18.	Felt like I was choking	1	2	3	4	5
19.	Felt really lively, or “up”	1	2	3	4	5
20.	Felt uneasy	1	2	3	4	5
21.	Felt discouraged	1	2	3	4	5
22.	Muscles twitched or trembled	1	2	3	4	5
23.	Felt like I had a lot of energy	1	2	3	4	5
24.	Was trembling or shaking	1	2	3	4	5
25.	Felt like I was having a lot of fun	1	2	3	4	5
26.	Had a very dry mouth	1	2	3	4	5

Appendix J: Modified Hamilton Rating Scale for Depression

2. ** DEPRESSED MOOD

How have you been feeling recently? Have you felt low in spirits, gloomy, or depressed? What percentage of time over the past week have you felt this way?

0. Absent
1. Mild-gloomy attitude, *may* be accompanied by infrequent weeping spells, sad, blue, waning of interests
2. Moderate-may be accompanied by feelings of inadequacy, self-pity, worrying, decrease in social interests and activity level, pessimism, "locked in", occasional weeping, apathy, decrease in experience of pleasure
3. Severe-may be characterized by hopelessness, greater tendency to withdraw socially, near absence of interest or participation in other than essential activities, hardly anything produces pleasure, weeping may be frequent (or beyond tears)
4. Extreme symptoms-complete withdrawal

9. Can't rate

7. ** GUILT

Are you critical of yourself for your weaknesses or mistakes? Do you blame yourself for things that go wrong around you even if others seem to think that you didn't have anything to do with them? Do you think your present illness is some type of punishment for something? Do you hear voices threatening or accusing you?

0. Absent
1. Feelings of self-reproach, self-blame, specific instance of lapse
2. Thoughts that negative events or reactions were caused by oneself; general or many instances or lapses for which one feels guilty; stronger convictions of one's guilt
3. Belief that illness might be a punishment, possibly delusional guilt
4. Delusional guilt, *with* hallucinations

9. Can't rate

***9. SUICIDE**

Do you feel that life is worth living?

Do you wish you were dead?

Do you have thoughts of committing suicide?

Have you tried to kill yourself?

0. Absent
1. Feels life is not worth living
2. Wishes he were dead or any thoughts of possible death to himself
3. Suicidal ideas, gestures, or plans
4. Attempted suicide (any serious attempt rated 4)

9. Can't rate

10. INSOMNIA

Do you have trouble sleeping?

*** a. Early**

Do you have trouble falling asleep? How long does it take you to fall asleep? How often?

0. Absent
1. Occasional (fewer than 3 days a week), mild, trivial (less than 1 hour delay)
2. Frequent (3 or more times per week) and severe (1 hour or more delay)
9. Can't rate

*** b. Middle**

Once you get to sleep do you wake up during the night? What do you do when you wake up? Can you get back to sleep?

0. Absent
1. Occasional (fewer than 3 days a week), mild (less than 1 hour delay in returning to sleep)
2. Frequent (several times per night with difficulty returning to sleep, 3 or more times per week) and severe (1 hour or more to return to sleep)
9. Can't rate

*** c. Late**

Do you wake up earlier than your usual time (before onset of depression) in the morning? Can you go back to sleep?

3. Absent
4. Occasional (fewer than 3 days a week), mild (less than 1 hour early)
5. Frequent (3 or more days per week) and severe (1 hour or more early)
10. Can't rate

11. APPETITE AND WEIGHT

*** a. Loss of appetite**

How is your appetite compared to the way it usually is? Do you have trouble with constipation or other problems with your stomach or bowels?

0. Absent
1. Loss of appetite, mild or occasional
2. Loss of appetite, severe or constant constipation
9. Can't rate

*** b. Loss of weight**

Over the *past month*, when not dieting, have you lost any weight?

0. Absent
1. One or 2 pounds or more over the past month
2. Three pounds or more over the past month
9. Can't rate

*** 12. a. LOSS OF ENERGY**

Have you had less energy than usual, or have you been getting tired more easily? How has this affected your work or other activities? Have your back, head, or limbs felt heavy or ached?

0. No loss of energy
1. Subjective loss of energy or feelings of tiredness
2. Marked interference with functioning (decrease in work and activities) OR feelings of heaviness or achiness
9. Can't rate

*** c. WORK AND ACTIVITIES**

Do you find that you have trouble doing things you really need to do (e.g. job, housework, studies)? How has this decreased interest in things and/or people affected your life?

0. Absent
1. Somewhat decreased efficiency, effortfulness;and/or decreased interest in or gets less pleasure from hobbies, interests, social contacts
2. Decreased performance, neglects or delays some things; withdraws from unnecessary activity, decreased participation in hobbies, social events
3. Considerably diminished performances of work or routine activities, more things are neglected or postponed indefinitely, virtually unproductive; avoids social contacts, nothing seems pleasurable, no interests
4. Unable to work, nonproductive, completely immobilized
9. Can't rate

*** d. LOSS OF LIBIDO**

Over the past *month* has there been any change in your interest in sex? Does this represent a change from the way you usually feel about sex?

0. No change
1. Some loss of interest and performance
2. Almost total loss of interest and sexual activity
9. Can't rate

*** 13. ANXIETY**

a. Psychic anxiety - anxious, tense, jittery, nervous, restless, "up tight", apprehensive, frightened, scared, irritable, worrying

Have there been times lately that you felt very anxious or frightened? Are these feelings fleeting, do they occur for a while and then go, or are they continuous? What percentage of time over the past week would you say that you have felt this way? What kinds of

situations do you feel anxious in? Have you been in any situation where you were so anxious that you simply had to get out, run, or do something else about it?

0. Absent
1. Transient tension, occasional irritability, mild exaggeration of worrying
2. Fairly constant tension, more frequent irritability, somewhat "hyper" or jittery.
3. Pervasive apprehension, tension, irritability, constant ruminative worrying
4. Panic attacks; phobias restrict activity

9. Can't rate

*** b. Somatic anxiety**

Symptoms are rated on the basis of the report of symptoms in the following systems:
(a) respiratory: labored breathing, shortness of breath, smothering or choking feelings, etc.;
(b) cardiovascular, flushing, accelerated heart rate, palpitations, faintness, chest pain or discomfort, etc.;
(c) gastrointestinal: indigestion, stomach upset, heartburn, stomach cramps, diarrhea, etc.
(d) genito-urinary frequency;
(e) sweating;
(f) giddiness, blurred vision, tinnitus;
(g) neuromuscular, trembling or shaking, headaches, muscle tension, dizziness, tingling, etc.

When you felt anxious, what was it like?
Did you notice your heart beating faster?
(Ask about other somatic symptoms noted above-circle each symptom that is present)
Have these bodily changes hindered your performance in any way?

0. Absent
1. Mild-one or more symptoms, complains of some discomfort but continues to participate in daily activities.
2. Moderate-e.g., symptoms from more than 1 system, occasionally patient can't take part in activities because of *bodily discomfort*
3. Severe-symptoms so uncomfortable that patient frequently has trouble taking part in activities
4. Extreme-multiple symptoms that are incapacitating, i.e., bodily discomfort precludes taking part in any activities
9. Can't rate

*** 14. HYPOCHONDRIASIS**

How is your physical health?
Do you tend to worry about your health?
Are you so concerned with your health that you find it hard to think about other things?
(Important: Interviewer is evaluating the extent to which the patient focuses on physical health to the exclusion of others symptoms)

5. Absent
6. Preoccupation with health, bodily, function, trivial or doubtful symptoms
7. Much preoccupation with physical symptoms, thoughts of organic disease
8. Strong conviction of presence of physical disease, querulous attitude
9. Hypochondriacal delusions and hallucinations, e.g. rotting, blockages, etc.
9. Can't rate

*** 15. INSIGHT** (patient background should be taken into account)

Do you think there is anything the matter with you? What do you think it is? Could it be that you have emotional problems?

0. Acknowledges being depressed or ill
1. Acknowledges illness but attributes cause to unlikely factors, e.g. bad food,
2. climate, overwork, etc.
3. Denies being ill
9. Can't rate

*** 16. RETARDATION (direct observation)**

0. Absent
1. Slight retardation at interview; flattening of affect and fixity of expression
2. Obvious retardation at interview; monotonous voice; delay in answering, motionless
3. Interview difficult, prolonged
4. Complete stupor
9. Can't rate

*** 17. AGITATION (direct observation)**

0. Absent
1. Low level of agitation, fidgeting, obvious restlessness (e.g. picking at hands or clothing, leg movements) for large proportion of interview
2. High level of agitation, includes fidgeting, obvious restlessness *as well as* the patient getting up during the interview, pacing, etc.
9. Can't rate

TOTAL SCORE: _____

Appendix M: Protocol

Positive Moods Psychophys PROTOCOL

1. Check with Jen if PT showed-she will be in either rm 467 or 440
2. If pt showed: Place "do not disturb" sign on the lab door
3. Set up all the equipment needed (see below)
4. Remember to wear the white lab coat so that the participant can identify you.

To start:

1. The participant ID will be on the folder. Also, confirm that the ID is the same one in the white protocol binder. Sign in on the white binder—ALSO confirm that you are running the pt in GMAIL calendar.
    a. **0-100** will be healthy controls
    b. **101-199** will be bipolar participants
2. Check that computers 1 AND 2 are on.

3. **General →Make sure the following items are available in the room or in the POSITIVE MOOD BAGGIE:**
    - Alcohol pads
    - Kleenex / tissues
    - 2 GREEN sensors for GSR
    - 3 BLACK sensors for EMG
    - Needle/Plunger
    - Signa GEL (green bottle)
    - 3 Sensor collars (white with blue tabs)
    - 2 GSR sensor collars (rectangle in GSR baggie)
    - Tape
    - The "CODE_OF_PARTICIPANT CHECKLIST"
    - Headphones (will be in top drawer)
    - Key Board cover
    - Remote control for the display in the eye tracking room
        - Change the source on the computer so that the screen in the eye-tracking room shows the Computer1 image (HDMI 4) if not, display that image by pressing the Source button on the remote.

COMPUTER 1 (EPRIME):

1. Open the folder, "**POSITIVE MOODS**", on computer 1.

2. First open the file, **"Movie"** but do not run it. Just keep it open so that you can play it after the eprime file is done running.

3. There should be the eprime file, **POSITIVE MOOD**.

4. Open the file. Click on the **generate** icon (a white script). Then, click on the **purple running** icon to run.

5. Enter in Subject Number = Participant ID NUMBER

6. Session Number = 1 ; click OK

7. **WAIT,** do not click on the Summary of Startup Button until study begins. If you need to escape, hit CANCEL or control, alt, delete to exit out of the program. Then, start over.

COMPUTER 2 (PSYCHOPHYS):

A. Open "BioLab 3.0A_1" program.

B. Make sure that the next Channels are "ON" with the adequate Filter Type selected:
   - Ch 4, GSC (Filter Type: EDA),
   - Ch 7, Bio Potential (Filter Type: Band Pass)

C. Press "ACQUIRE (green button)"

D. Create a new file and call it "**CODE_OF_PARTICIPANT_PosMood**" (e.g., 76_PosMood)

E. Save file under "**Positive Moods**" folder on the desktop.

F. **Open up Internet Explorer**—the homepage will be the camera screen. Move the cursors to get the camera recording so that you can see their face (it should take up the whole screen).

NOW SET UP SENSORS TO HOOK UP PARTICIPANT

Sensor Preparation and Placement for EMG and Skin Conductance

For psychophys make sure you do the following before the participant arrives:

EMG → 3, 4mm sensors to the three EMG leads (all BLACK)→ this hooks to BLACK dot

SCR → 2 GSR sensors to the two GREEN SCR leads-→ this hooks to GREEN dot

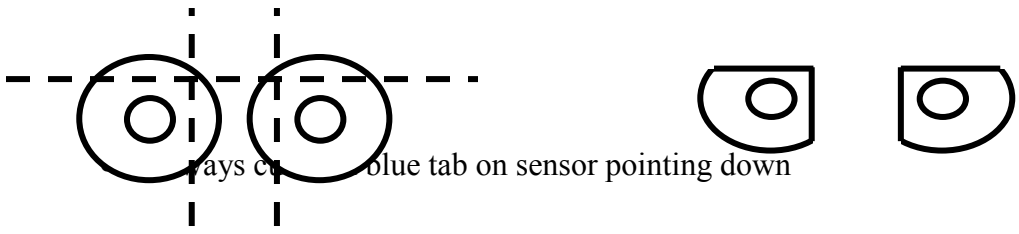
Pre-participant preparation

For all measures, prior to the arrival of the participant you should:

I. **Cut sensor collars, if needed (there should be pre-cut sensors in the POSITIVE MOOD BAGGIE)**

The EMG sensor collars are cut in order to allow them to be placed closer together and closer to the site of EMG activity.

1. For obicularis, we cut both the top and insides of the collars to allow the sensors to be placed close together on the **LEFT** eye (for obicularis)



ays cu blue tab on sensor pointing down

- CUT first over the third of the top
- Then cut parallel to the blue tab
- The first one with the blue tabs pointing down is left alone and will be used as the ground sensor

II. Gently pull off one side of the collar with the sensor still attached.
1. **Next attach the sensor collars to the sensors, with the sensors directly over the hole in the collars, with the wires hanging down and the blue tab pointed down.**
2. If the collar falls off, get another collar from a fresh batch with the same side cut off. Make certain that the collar is securely attached by rubbing firmly with your thumb.

III. **Fill "gel applicator" with gel**
1. Locate a gel applicator body and applicator tip (should be extra in the 2nd drawer of the red file cabinet). They need to be clean-old gel in the needle is NOT good.
2. Prior to attaching the tip, pull out the plunger.
3. Fill green SIGNA GEL up to 1 level in the syringe.
4. Next, attach the needle tip to the syringe. BE CAREFUL!
5. Slowly reattach the plunger.
6. Fill the EMG sensors just to the edge of the collar. **It is VERY IMPORTANT that you do not touch the sensor cup surface with the gel applicator tip**. This will destroy the Ag-AgCl coating.
7. Wipe off the excess with a tissue
8. Keep sensors laid out with collars facing up to keep the gel in the sensor
9. (after the session is done, there are instructions below on how to clean the sensors. NEVER leave gel in the sensors, we have to throw them out then and any residue weakens the signal)

After participant has arrived:
Record the Session Start Time and room temperature

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9. **WASH HANDS:** Ask the participant if he/she needs to use the restroom or drink some water. Ask participant to go to the restroom and wash their hands with soap – even if they do not need to use the restroom. Take the participant to the restroom.

10. Ask participant to **take off any necklaces, watches, bracelets, and/or earrings** that might become uncomfortable with the headphones on.

11. **Place headphones on participant**.

ATTACH PSYCHOPHYS: Attach psychophysiology equipment outside eye tracking room in purple chair.

First connect the EMG sensors (see Picture at end)

1. Prior to attaching these sensors inform the participant that the sensors will feel a bit funny under their eye and may make their eye blink and water for a few moments. Tell them that they will get used to the feeling within a few minutes.
    a. Make certain that you have the participant close their eyes if you are anywhere near the eye.
    b. Use alcohol swaps to clean the areas (middle of forehead, and below the left eye. Ask them to close their eyes during this. Tell them what you will be doing to prepare them. DO not swipe anywhere near the eye) – wait a second after cleaning so that the cleaned area is not wet anymore. This helps the sensors to stick.
    c. Next use a little bit of lemon prep to abrade the skin.
    d. Last, use a tissue to wipe off excess and then use another alcohol swap to clean off the lemon prep residue.

Ground sensor placement (standard sensor in middle socket)

a. You will attach a ground sensor in the center of the participant's forehead-ask to scrunch their forehead, and place it on the smooth section in the middle. Attach this sensor such that the sensor lead and the BLUE TAB is pointing up and back toward the participants ear to avoid the lead dangling in the participants face.
b. Tape the wire to the top of the headphone so that the wire is not in the person's face.
    - **Then Place the BLACK sensors for the eyes**
    - Tell the participant to look directly forward and up. Place the first sensor under the eye and directly in line with the pupil. You should place the sensor such that the collar top fits right into the crease under the eyelid. Ask them to blink and ask if it's distracting or it hurts. If so, remove and start placement again.

    - Place the second sensor directly next to the first and to the outside (and slightly higher following their lower eyelid if possible) of the first sensor such that the collars just touch. Again, this should place the top of the collar right into the crease under the eye.

    - Be sure that both eye cables are pointing down toward the ground at their attachment point to the sensor.

    e. Plug end of sensor leads into the MIDDLE ground channel on the headbox. Tape the ground sensor to the top of the headphones. Plug the two eye sensors into the BLACK socket.

f. Make sure that neither eye sensor is attached to the middle socket (this is used for the ground placement)

Skin Conductance placement (rectangle sensors with GREEN wires)

a. Connect the SCR sensors to the palms of the participant's non-dominant hand. **Do NOT use alcohol swabs. Use tape to hold the cables in place. (see picture at end)**

b. You will place the SCR RECTANGLE (from the GSR baggie) sensors on the fleshy part of the palm under the pinky. Place the two sensors vertically on the palm with the outer edges of the collars just touching.
c. You will place each GREEN sensor directly on top of these collars.
d. If the sensor appears to be insecurely attached, you can wrap tape around the sensor cup to secure more firmly to the palm.
g. Have Participant walk over to eyetracking room and sit down.
h. Adjust the cable so that they don't sit on them or roll over them.
i. CLIP the sensors to a shirt area. LET the participant know you will do this.
j. Next have the participant put on the head rest.
k. Next place cushion and Keyboard on lap. Check that the number key is on!
l. Place the Key board cover over the keyboard and position it so that only the number key is visible.
m. Tell them,

"We will be asking you to enter in numbers and press the ENTER KEY in the study."

j. Attach the cables to the psychophys box on the right – GREEN (SCR) goes to GREEN sticker, EMG goes to BLACK STICKER
k. REMIND PARTICIPANTS NOT TO USE LEFT HAND (NON-DOMINANT)—Tell them:

"Please keep you left hand still"

l. Hook up the headphones to the speaker (double check that it's the input for the headphones, not the microphone)

<u>COMPUTER 2 (PSYCHOPHYS)</u>

DO NOT PRESS START UNTIL THE PARTICIPANT IS HOOKED UP

1. Press "**START/STOP (Enter)".**
2. **Record Psychophys START Time**

<u>COMPUTER 1 (EPRIME)</u>

NOW Press "YES" on the Summary of Startup Info screen on COMPUTER 1

1. Only press "Exit (Esc)" once the eprime protocol stops
2. After the end of eprime task, maximize the movie clip, "**MOVIE**" and play for the duration.

What if Eprime crashes?

A. Make note of it in their binder, where it crashed, and reassign with the next PT Number and restart the protocol again.
B. Email Jen to let her know and log it in the white binder

Sensor Removal and Cleaning

1. Disconnect each sensor lead from the headbox and drape to the side of the participant so that you do not step on the leads.
2. Have a tissue in your hand and Carefully remove the electrodes from the participant. For the facial EMG sensors, it is often best to place one finger on the participants face directly next to the sensor to avoid the skin pulling while removing the sensor. It is very important that you do not pull on the sensor leads. Pull on the sensor collar tabs to remove.

Self-Rated Questionnaires

- First hang the cable on the side wall and walk the Participant up to 467 to complete the battery of self-rated questionnaires.
- Remind them to complete it and not to skip any questions.
- Tell them you will check on them periodically. (it should take 20-30 minutes to complete the self-rated questionnaires).
- MAKE SURE THEY DO NOT LEAVE THE ROOM TO MAKE CALLS OR LEAVE. RESTROOM AND WATER BREAKS ARE OKAY.

1. Come back to the lab room and bring all of the sensors (and the gel applicator) to the sink in the rest room.
2. Remove sensor collars from each sensor GENTLY. Again, do not pull on the leads. Hold the sensor cup and the tab on the sensor collar.
3. Place each sensor cup directly under the water stream from the facet. Make certain that the end of the sensor leads do not get wet. Hold the sensor close to the bottom of the sink to avoid splashing. Move the orientation of the sensor around relative to the jet stream to allow the water to hit the sensor cup from different angles. Do this for 10-15 seconds
4. Remove the sensor from the water and blow firmly on the cup to remove the water.
5. Repeat steps 5-6 until there is absolutely no residual gel on the sensor cup surface. It is very important that the surface is completely clean.
6. Clean the gel applicator and tip.
    a. Squirt excess gel into the garbage can
    b. Remove the plunger and fill the applicator with water, and squire though the applicator.
    Repeat a few times.
    c. Disconnect the applicator tip and blow through it
    d. Inspect the tip and make certain that you can see through it with no residual gel.
    e. Clean the applicator body
    f. Put the applicator body and tip back into the baggie
7. Hang the BLACK electrodes from the sensor rack to air dry.
8. Check in with participant. See if they are done.
9. Ask if they have any questions.
10. Note time completed.
11. Confirm that W-4 Form is completed.
12. Get check from Jen.

13. Debrief them.
14. Remind them that we will make a follow-up phone call the next day. Ask when it is a good time to call and check-in.

DEBREIFING SCRIPT:

"Thank you for participating. Do you have any questions about the study? [probe for attempts to figure out hypotheses] Let me tell you a little bit more about what we are doing in this project. In this study we are looking at how different people may react differently while viewing various types of pictures. To look at difference in reactions, we attached sensors under your eye and on your fingers. The sounds you sometimes heard along with the pictures make you blink. People blink either a lot or a little, depending on the type of picture they are looking at. But different people may blink more or less, as well. In the middle of the session we had you read a series of statements at a fast rate. This procedure has been shown in other studies to create a positive mood. We are also looking at whether the positive mood influences different people to different degrees when they look at similar pictures after having the mood induced."

"Do you have any questions?"

Address any questions the participant might have.

! If they indicate any distress or any sign of inappropriate positive mood, inquire about their feelings. Seek supervision with Dr. Carver or Dr. Marker if a person has concerns about any aspect of the study or the deception. Remind them that we will make a follow-up phone call the next day. Ask when it is a good time to call and check-in.

CONTACTS:

JEN

DR. MARKER 305-600-3032

DR. CARVER 305-284-2817

Appendix N: Negative Valence IAPS

Negative Valence					
Number	**Theme**	**Valence**	**SD**	**Arousal**	**SD**
1050	animal attack	3.46	2.15	6.87	1.68
1112	animal attack	4.71	1.7	4.6	2.4
1300	animal attack	3.55	1.78	6.79	1.84
1930	animal attack	3.79	1.92	6.42	2.07
3530	human attack indirect	1.8	1.32	6.82	2.09
6360	human attack indirect	2.23	1.73	6.33	2.51
6510	human attack direct	2.46	1.58	6.96	2.09
6360	human attack indirect	2.23	1.73	6.33	2.51
6410	human attack direct	3.49	2.07	5.89	2.28
9610	accident	2.89	1.43	5.23	2.14
9600	accident	2.48	1.62	6.46	2.31
9910	accident	2.06	1.26	6.2	2.56
9920	accident	2.5	1.52	5.76	1.96
3016	mutilation	1.9	1.31	5.82	2.44
3017	mutilation	2.45	1.35	5.34	2.39
3062	mutilation	1.87	1.31	5.78	2.57
3061	mutilation	2.32	1.61	5.28	2.6
6190	aimed at gun	3.57	1.84	5.64	2.03
6200	aimed at gun	3.2	1.62	5.82	1.99
6210	aimed at gun	3.57	2.95	5.64	1.83
AVERAGE		**2.83**	**1.69**	**6.00**	**2.21**

Appendix O: Positive Valence IAPS

Positive Valence					
Number	**Theme**	**Valence**	**SD**	**Arousal**	**SD**
8465	runner	5.24	1.29	2.82	1.93
8080	sailing	7.73	1.25	7.12	1.95
8490	rollercoaster	6.85	2.36	6.25	1.96
8600	mascot	6.25	1.65	4.18	2.27
5621	skydive	7.57	1.42	6.99	1.95
5623	athlete	7.19	1.44	5.67	2.32
5626	flying	6.71	2.06	6.1	2.19
8400	rafting	7.43	1.4	7	1.56
8540	athlete	7.28	1.59	4.96	2.34
8186	sky diving	7.22	1.38	6.98	2.05
8170	boat	7.63	1.34	6.12	2.3
8185	skydiving	7.32	1.58	7.06	2.09
8190	skiing	8.1	1.39	6.28	2.57
8200	water skiing	7.54	1.37	6.35	1.98
8350	athlete	7.18	1.56	5.18	2.28
8370	rafting	7.77	1.29	6.73	2.24
8501	money	8.14	1.24	6.86	2
8502	money	7.33	1.63	5.48	2.41
8503	money	6.93	1.81	6.86	2
AVERAGE		**7.23**	**1.53**	**6.05**	**2.13**

Appendix P: Neutral IAPS

Neutral Number	Theme	Valence	SD	Arousal	SD
5395	boat	5.34	1.21	4.23	2.03
5500	mushroom	5.42	1.58	3	2.42
5510	mushroom	5.15	1.43	3	2.42
5520	mushroom	5.33	1.49	2.95	2.42
5530	mushroom	5.38	1.49	2.95	2.42
5531	mushroom	5.15	1.45	3.69	2.11
5532	mushroom	5.19	1.69	3.79	2.2
5533	mushroom	5.31	1.17	3.12	1.9
5534	mushroom	4.84	1.4	3.14	2.03
5731	door	5.39	4	2.74	1.95
6150	light socket	5.08	1.58	3.22	2.02
7000	rolling pin	5	1.17	2.42	1.79
7002	towel	4.97	0.84	3.16	2
7006	bowl	4.88	0.97	2.33	1.67
7009	blue cap	4.93	0.99	3.01	1.97
7030	utensil	4.69	1	2.99	2.09
7031	shoes	4.52	1.04	2.03	1.51
7040	utensil	4.69	1.11	2.69	1.93
7050	hair dryer	4.93	1.09	2.75	1.8
7060	trash can	4.43	0.81	2.55	1.77
7078	bucket	3.52	1.16	3.73	1.78
7080	utensil	5.27	1.44	2.32	1.84
7090	book	5.19	1.09	2.61	2.03
7100	fire hydrant	5.24	1.46	2.89	1.7
7140	bus	5.5	1.2	2.92	2.38
7150	umbrella	4.72	1.42	2.61	1.76
7170	light bulb	5.33	1	3.27	2.22
7179	lamp	4.95	1.49	1.87	1.48
7190	clock	5.59	0.8	3.8	2.14
7175	lamp	4.87	1.27	1.72	1.26
7211	clock	4.81	1	4.2	2.4
7235	chair	4.96	1.78	2.83	2
7491	building side	4.82	1.18	2.39	1.9
7242	building	5.35	1.03	3.56	2.18
7052	clothes pin	5.24	1.39	2.57	1.86
7045	zipper	4.88	0.88	3.26	1.88
7061	puzzle	5.42	1.33	3.65	2.03

Appendix P

Neutral Number	Theme	Valence	SD	Arousal	SD
7026	picnic table	5.41	1.33	2.43	1.73
7038	shoes	4.97	0.93	2.92	1.84
7055	light bulb	4.92	0.77	2.82	1.83
7096	car	5.7	1.2	4.04	1.8
7130	truck	4.79	1.14	3.54	2.01
7192	vase	5.69	1.22	3.65	1.84
7224	file cabinet	4.38	1.49	2.55	1.86
7233	plate	5.01	1.21	2.51	1.74
7500	building side	5.33	1.44	3.26	2.18
7510	building side	6.05	1.6	4.52	2.35
AVERAGE		**5.08**	**1.29**	**3.03**	**1.97**