

Measuring Distinct Reward Processes in Depression and Mania

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Abstract

Major Depressive Disorder (MDD) and Bipolar Disorder (BPD) are mental illnesses that cause immense burdens both at personal and societal levels. Symptoms of both conditions are increasingly being understood as aberrations in reward processing, with “anhedonia” (one of two hallmark symptoms of depression) seen as hypoactive anticipatory reward processing, and clinical mania increasingly associated with hyperactive dopaminergic pathways. The current study observed 36 adults and used EEG while participants engaged in a reward-based time-estimation task. Analyses focused on four event related potentials (ERPs) looking at various features of reward processing: the Stimulus Preceding Negativity (SPN), linked to anticipation of reward-related feedback, the Negative 2 (N2), linked to exertion of cognitive control during goal-oriented tasks, the Feedback-Locked P3 (FB-P3), linked to mental appraisal of how salient a reward is, and the Late Positive Potential (LPP), linked to integration of reward-related feedback into behavior. Participants also completed a series of measures asking about depressive and hypomanic symptoms. We predicted that higher levels of depressive symptoms would show negative correlation with all four ERPs, while higher levels of hypomanic symptoms would correlate positively with SPN, N2, and FB-P3 and negatively with LPP. Contrary to hypotheses, no significant correlations were observed between SPN, N2, FB-P3, or LPP potentials and depressive and hypomanic symptoms. Implications of our understanding of reward processes in MDD and BPD as well as limitations of the current study and directions for future research are discussed.

Introduction

Overview

Major Depressive Disorder (MDD) and Bipolar Disorder (BPD) are mental illnesses that are immensely burdensome at both a personal and a societal level. Some estimate that MDD affects up to 350,000,000 people worldwide, and the World Health Organization cites MDD, whose symptoms include depressed mood, loss of interest in activities, problems eating and sleeping regularly, trouble concentrating, and feelings of worthlessness or guilt, as the leading cause of disability worldwide, largely because it impairs people's abilities to work productively and form functional relationships (Sullivan, Neale, & Kendler, 2000). Experts estimate that around 10-15% of Americans experience some form of depression, and the disease is especially burdensome because as many as 60% of those who experience symptoms either do not realize they have a problem or choose not to see clinicians for diagnosis or treatment (Sullivan et al., 2000). BPD also provides a huge personal and societal burden, with patients at high risk for substance abuse and suicide, with a high rate of misdiagnosis, and with some studies showing it to be the most expensive behavioral healthcare diagnosis (Fajutrao, Locklear, Priaulx, & Heyes, 2009; Peele, Xu, & Kupfer, 2003; Simon & Unützer, 1999). Because MDD and BPD are so burdensome and affect so many people, better understanding the etiology and maintenance of these illnesses could be crucial in designing effective, accessible treatment options and diagnostic measures.

Recently, symptoms of MDD and BPD have been associated with deficits in reward processing. Research on reward systems in the brain has suggested that we have distinct processes for anticipating reward before we receive it (which some conceptualize as "wanting," related to approaching rewarding stimuli) and actually consuming a reward (which some

conceptualize as “liking,” related to the actual hedonic experience of receiving a reward).

Limited research has suggested that patients with MDD may show deficits in their anticipation-based reward systems or with integrating their experience consuming reward into future behavior (McFarland & Klein, 2009; Pizzagalli, Iosifescu, Hallett, Ratner, & Fava, 2008). Research on reward systems in BPD is also limited, but some studies suggest that BPD is related to hyperactive reward systems (Berpohl et al., 2010). Using EEG, which has high enough temporal resolution to separately measure the anticipation-based and consumption-based reward systems in the brain, this study looks at how depressive and manic symptoms are related to two anticipation-based event related potentials (ERPs) and consumption-based reward ERPs to better understand the deficits in reward systems associated with MDD and BPD. We anticipate that depressive symptoms will correlate negatively with anticipatory ERPs and the consumption-based ERP associated with integrating feedback into behavior, while we anticipate that hypomanic symptoms will associate positively with both anticipatory and consumption-based ERPs.

Depression and Reward Processing

Ever since Feighner published the first diagnostic criteria for depression in 1972, anhedonia, traditionally conceptualized as a loss of pleasure, has been considered one of depression’s core symptoms (Feighner et al., 1972; Gorwood, 2008). However, recent studies have suggested that in experimental settings, depressed individuals only display “loss of pleasure” inconsistently, and many have highlighted that hedonic processing might not be a singular construct. One test used to test hedonic capacity is the “sweet taste test,” in which participants consume mixtures with different concentrations of sugar and rate the pleasantness of each. In a review of four separate studies administering the “sweet taste test” to individuals with

depression, Treadway and Zald have found that those with depression reported the same hedonic intensity (that is, liking the sugar mixtures the same) as controls (Treadway & Zald, 2011).

These studies and others have suggested that anhedonia may be more complex than just not “liking” rewards and positive stimuli, and may be more related to disruptions in motivational processes (i.e., “wanting”). In 1998, Berridge and Robinson conducted experiments on dopamine-depleted mice and controls, feeding them either sucrose (sweet) or quinine (bitter), and observing patterns of affective taste reactivity. Reactions were classified as “hedonic” (i.e., more licking), “aversive” (i.e., gapes, face-washing, chin rubs), or “neutral” (i.e., neither hedonic nor aversive, or quick sequences involving both). They found that dopamine-depleted mice and controls displayed the same hedonic reactions to the sweet and bitter solutions, but found that dopamine-depleted mice did not eat or drink voluntarily as often as controls, and did not show as much goal-directed behavior towards sucrose than did control mice (Berridge & Robinson, 1998). This experiment established a distinction between “wanting,” or reward anticipation, and “liking,” or reward outcome processes in the brain, and in the decades since, this distinction has been thoroughly established in humans as well (Pool, Sennwald, Delplanque, Brosch, & Sander, 2016). Because “loss of pleasure” (that is, a deficit in “liking”) evidently does not explain anhedonia, in recent years, many recent studies have approached it by focusing on the motivation to seek reward and the ability to modulate behavior based on reinforced reward-related feedback.

A number of studies have suggested that people with depression show differences in reward-related behavior than healthy people do. In a study published in 2005, Pizzagalli et al. recruited non-clinical undergraduates to perform a signal-detection task in which they were briefly shown cartoon faces with either short or long mouths, and were then asked to identify whether the face had a short or long mouth with a monetary reward for correct response. As a

manipulation, they assigned half of participants to a reward schedule in which correctly identifying the short mouth would provide thrice as much money as correctly choosing long mouth, and the other half to the opposite (that is, correctly identifying long would provide thrice as much as correctly identifying short). They found that participants exhibited a “response bias” by which they frequently responded correctly to the stimulus with a higher reward and frequently responded incorrectly to the stimulus with a lower reward, but found that in participants with elevated depressive symptoms demonstrated no response bias (Pizzagalli, Jahn, & O’Shea, 2005). Pizzagalli et al. corroborated these results in a similar study with participants diagnosed with MDD, finding that depressed patients showed heavily diminished response bias compared to controls (Pizzagalli et al., 2008). These studies and others suggest that anhedonia (and other depressive symptoms it may underlie) can be conceptualized as a reduced ability to adapt behavior in response to salient rewards; participants with many depressive symptoms seem to have difficulty integrating a history of reinforcement into their reward-seeking behavior. Together, these results also predict that those with higher levels of anhedonia (or other depressive symptoms) would exhibit blunted reward anticipation.

Research looking at depression and anticipation-based reward systems have generally revealed that people with depression experience blunted affect when anticipating reward. McFarland and Klein studied participants who were either currently depressed, previously depressed, or never depressed (control), asking them to fill out a series of state-emotion questionnaires before and after completing a number of puzzles. As a manipulation, they sorted participants into four conditions: anticipated reward (winning a cash reward for solving a certain percent of the puzzles), anticipated punishment (being punished for getting too many wrong), non-reward, and avoidance (told they were close to being punished, but then not being

punished)s. They found that the changes in state emotion during the anticipated punishment, non-reward, and avoidance conditions were not significantly different among the three groups, but found that when they knew that they were about to win money (the anticipated reward condition), those who were currently depressed exhibited significantly diminished increases in positive affect than the other two groups (McFarland & Klein, 2009). It is worth noting that the post-trial questionnaires were completed before the reward or punishment was actually administered, so the results indicate that depressed individuals experienced blunted affect when anticipating reward.

Overall, research on depression and reward has generally revealed that people with depression may experience blunted affect while anticipating reward, and may have difficulty incorporating reward-related feedback (i.e., receiving a reward and enjoying it) into future reward-related behavior (i.e., repeating the same action in the same situation later). One of the current study's aims is to clarify which aspects of anticipation and consumption of reward are deficient in depression.

Mania and Reward Processing

Mania and BPD have also been linked to changes in reward processing in the brain, and particularly, to hyperactive anticipatory reward processes. Anticipatory reward processes are generally linked to dopaminergic pathways in the brain (Berpohl et al., 2010). In 1986, Jacobs and Silverstone found that administering Dextroamphetamine, a dopamine agonist, to non-clinical individuals induced a mania-like phenotype (including excessive energy, irritability, overly good mood, erratic talking, inability to concentrate, and feelings of power). This gave rise to the idea that dopamine hyperactivity or hypersensitivity could be a mechanism for mania and BPD (Jacobs & Silverstone, 1986). In more recent years, studies of the ventral striatum and the

ventral prefrontal cortex, both considered to be a part of a larger corticolimbic reward circuit (Haber & Knutson, 2010), have found increased activity in these regions in individuals with mania (Berpohl et al., 2010; Caseras, Lawrence, Murphy, Wise, & Phillips, 2013). In an experiment by Berpohl et al., healthy controls and participants with mania participated in a monetary incentive delay task, in which they anticipated to win or lose different amounts of money, while monitored using fMRI. The study found that manic patients exhibited increased activation in the left lateral orbitofrontal cortex when expecting increasing gains and decreased activity in the left IOFC when expecting increasing losses, which was the inverse of the effect observed in healthy controls (Berpohl et al., 2010). This finding was consistent with the idea that manic symptoms are associated with hyperactive anticipatory reward signals, and also suggests that those with mania or BPD might have difficulty integrating loss-related feedback and reinforcement into behavior.

Similarly, in an experiment by Caseras et al., healthy controls, euthymic bipolar I patients (euthymic means currently in neutral mood state, BPD-I is characterized by severe mood episodes alternating between mania and depression), euthymic bipolar II patients (BPD-II is characterized by milder mania but just as severe depressive episodes) engaged in a task in which they could either gain or avoid losing money by correctly guessing the number of a card, all while being observed using fMRI and structural scanning (Caseras et al., 2013). Caseras et al. designed the experiments such that participants had to wait in anticipation before their answer was deemed either correct or incorrect, so the brain scans were able to pick up activity for both the anticipation and the receipt of a reward or punishment. They found that patients with BPD-II (that is, patients diagnosed with alternating episodes of hypomania and severe depression) showed significantly higher ventral striatal activity during anticipation than did patients with

BPD-I or controls, and also found that elevated ventral striatal activity was related to reward consumption (Caseras et al., 2013). Together, studies of manic symptoms and dopaminergic systems suggest that hypomania (rather than full blown mania) may be related to increased dopaminergic activity in the ventral striatum, and also predicts that hypomanic symptoms might positively correlate not just to anticipation reward processes, but to outcome reward processes as well. Additionally, hypomanic symptoms are expected in people at risk for later BPD, and are also sometimes considered symptoms of premorbid BPD, so studying hypomania could give insight into how to treat the onset of BPD before it begins (Eckblad & Chapman, 1986). Additionally, BPD (in both types) consists not just of manic or hypomanic symptoms, but of depressive symptoms as well; studying these two sets of symptoms in a non-clinical sample could allow us to separate the two and understand them better individually.

Measuring Reward Processing: EEG and ERPs

While this paper has focused so far on behavioral and neuroimaging approaches to measuring anticipation-based and outcome-based reward systems, electroencephalography (EEG) is a method for visualizing brain activity that is advantageous because of its relatively high temporal resolution (i.e., it can distinguish temporally between anticipation and consumption of reward), its relatively simple experimental setup, and low cost compared to other methods (e.g., MRI or fMRI). EEG is an imaging method that measures electrical activity on the scalp. By observing a participant asked to close their eyes and relax, we can isolate and localize (since EEG uses a number of electrode spaced out around the head and scalp) various types of resting brain waves. Additionally, by instructing participants to use certain patterns of thinking (e.g., rumination, worry) or engage in certain types of tasks (e.g., risk evaluation, reinforcement learning), we can visualize the resulting brainwaves as well. EEGs are especially useful because

by asking a participant to repeatedly engage in a specific task over many trials, we can average the wave forms from each iteration of the task. By this process, noise between iterations evens out (random positive noise generally cancels out random negative noise), and we can isolate and localize waveforms associated with the task itself; this is called an event related potential, or ERP (Teplan, 2002). Using ERPs for specific reward-related tasks, we can isolate several types of waves that represent activity at various stages of the hedonic process.

Two ERPs studied in reward-anticipation are the Stimulus Preceding Negativity (SPN), a negative, fronto-central waveform, and the N2, another negative, fronto-central waveform that appears around 200-400ms after a reward cue; both of these components appear after goal-oriented behavior but before receipt of feedback (Brunia, Hackley, van Boxtel, Kotani, & Ohgami, 2011; Potts, 2011). In their review of SPN, Brunia et al. describe a body of research suggesting that this ERP is more negative when subjects are anticipating reward-related feedback (as opposed to just feedback in general). The pre-feedback SPN grows drastically as the reward becomes more imminent, and it is generally understood to represent activity in the insula, and it has been established as a reliable representation of reward-anticipation in reward-related tasks (Brunia et al., 2011). Additionally, the N2 is speculated to either vary based on whether an event is accordant or discordant with expectations, with a greater amplitude when an event occurs according to expectations, or to represent cognitive-control, with higher amplitudes indicating greater exertion of cognitive control (Folstein & Van Petten, 2008; Potts, 2011).

Two ERPs, the Feedback-Locked P3 (FB-P3) and the Late-Positive Potential (LPP), have been shown to represent aspects of neural reward-outcome processes. The FB-P3, a positive component localized to centro-parietal portions of the brain, is most prominent around 250-450ms after feedback has been administered. It has been theorized as an indicator of feedback's

motivational salience, with amplitude increasing as rewards increase (San Martin, 2012). In an experiment by Gu et al., participants were monitored using EEG equipment and asked to complete a task in which they were asked to respond in one of two ways, one of which led to a reward and one of which led to a punishment. Ultimately, they found that the FB-P3 increased in amplitude as the magnitude of the reward increased. Thus, the FB-P3 can be considered as representing the reward-evaluation process, judging the relative importance of a reward (Gu, Wu, Jiang, & Luo, 2011). Similarly, the LPP is also a positive centro-parietal component, although it lasts longer than the FB-P3 does (Schupp, Flaisch, Stockburger, & Junghofer, 2006). The LPP has not always been considered implicated in reward processing, but has been shown to represent prolonged cognitive processing of high-arousal motivational stimuli and high-arousal emotional stimuli. Additionally, a handful of recent studies have suggested that LPP activity predicts behavioral adjustment in subsequent trials to optimize reward-outcome. It is worth noting that FB-P3 is seen as influenced by reward-evaluation (the prospective motivation to learn the outcome of a response in relation to a possible reward), while it is unclear whether LPP is influenced by reward-evaluation or by performance-evaluation (the retrospective assessment of whether a completed action was good or bad at obtaining a reward) (Pornpattananangkul & Nusslock, 2015).

Pornpattananangkul et al. recruited non-clinical participants to complete a reward time-estimation task while monitored using EEG, with the ultimate goal of determining relationships between reward-anticipation ERPs and reward-outcome ERPs. In their task, participants were asked to press a button 3.5s after a cue, and were provided feedback; in half of the trials, participants were cued beforehand that they would receive no reward, and received no reward regardless of their performance; in the other half, they were cued that they could receive a

reward for accurate time estimation, and were rewarded accordingly. No-reward trials were used as controls. Additionally, to control for individual differences in time-estimation ability, if a participant correctly responded in one trial, on the subsequent trial, the time-window for correct responses was shortened by 20ms; if a participant incorrectly responded, the opposite occurred. Experimenters measured SPN, N2, FB-P3, and LPP ERPs among others. One of their significant findings was that SPN activity strongly predicted LPP activity (Pornpattananangkul & Nusslock, 2015). The findings from this study provide some relationship between anticipatory and outcome-based reward systems, but they do so without taking into account depressive or manic symptomatology.

Reward-Based ERPs and Depressive and Manic Symptoms

Studies looking at depressive using EEG have suggested that depression is linked to depressed dopaminergic pathways in the left prefrontal cortex. In one study looking at neural correlates of reward sensitivity in people with depression, Shankman et al. had healthy controls and patients with MDD complete a task in which they played on a “slot machine” while monitored with EEG. Half of the time, there was no incentive to play (i.e., no possible reward), and the other half of the time, winning could provide a reward (Shankman, Klein, Tenke, & Bruder, 2007). Thus, the reward conditions were intended to elicit approach motivation and anticipatory responses in the brain, while the no-reward conditions served as a control. They ultimately found that in patients with MDD, the left hemisphere’s prefrontal cortex had significantly depressed activity, which seems to corroborate prior fMRI evidence of decreased left prefrontal activity (Berpohl et al., 2010; Shankman et al., 2007). This study confirms observations of deficient dopaminergic pathways, but is limited in that it does not elucidate the

relationship between reward-anticipation, reward-outcome, and subjective experience of reward in people exhibiting depressive symptoms.

Similarly, studies using EEG to look at reward in patients with BPD have suggested that BPD is related to increased dopaminergic activity in the left PFC. In one such study, conducted by Harmon-Jones et al., healthy controls and patients with bipolar disorder were monitored by EEG as they attempted to solve easy, medium, or hard anagrams (they were cued as to which it would be) to either earn money or avoid losing money. This study found that participants with BPD showed greater relative left frontal cortical activity when preparing for hard puzzles in the “earning” condition (Harmon-Jones et al., 2008). This result corroborates prior fMRI findings that in patients with mania, left hemispheric frontal regions are especially active in reward tasks, but does not clarify the relationship between reward-anticipation, reward-outcome, and subjective experiences of reward in those experiencing manic or hypomanic symptoms (Berpohl et al., 2010; Harmon-Jones et al., 2008).

The Current Study

Few studies have looked at the differences in reward systems based on depressive and manic symptoms in the same sample. Additionally, few studies have looked at reward using tasks that can discriminate between anticipatory and consumption-based reward processes. Thus, the current study aims to use EEG and self-report measures to better understand the relationship between anticipatory reward processes, outcome reward processes, and subjective experiences of reward processing, as they relate to depressive and hypomanic symptoms. We will observe two anticipation-based ERPs, the SPN and the N2, as well as two consumption-based ERPs, the FB-P3 and the LPP, to see how specific reward processes are related to depressive and hypomanic

symptoms. We predict that higher depressive symptoms will be negatively correlated with SPN, N2, and LPP ERPs, while higher hypomanic symptoms will be positively correlated with the SPN, N2, and FB-P3 but negatively with the LPP.

Methods

Participants

Thirty-six adults (12 male, 24 female), ages ranging from 18-55 ($M=22.13$, $SD=3.85$), participated in the current study. The majority of the sample identified as Caucasian (52.1%), while 14.9% identified as Asian or Asian American, 12.8% identified as Hispanic or Latino, 10.6% identified as African American, and 9.6% identified as “other.” Exclusion criteria for participation included left-handedness, history of head injuries, and current use of acne medication, all in accordance with guidelines from prior studies measuring ERPs. They were paid \$10 per hour for their participation.

Task

The current study used a modification of the reward time-estimation task, and a schematic representation of the task is shown in Figure 1 (Pornpattananangkul & Nusslock, 2015). The task is designed to show differences in anticipation and consumption reward processes both when winning or not winning reward, and both when reward is possible or not. Participants were first connected to EEG equipment prior to engaging in the task. In each trial, participants were first presented with a cue indicating that there either would or would not be the possibility of winning a monetary reward on that trial. In “reward” trials, participants were informed that for successfully completing the task, they would receive \$0.20, while in “no-reward” trials, they were informed that they would not receive monetary reward regardless of

performance. Cues comprised of either circle or square shapes, counterbalanced across the sample, and controlled for top-down processes like contrast, luminance, and spatial frequency using the SHINE toolbox developed by Willenbockel et al. (Willenbockel et al.). Similar to the previously used task, participants were asked to press a button 3.5s after the initial cue, and responses were considered accurate if responses occurred during the correct time window (which were tailored to individuals' response times during practice trials to make overall accuracy rates around 50%) (Kotani et al., 2003). Two seconds after the participant pressed the button, the screen displayed one line of feedback indicating the participant's performance on that estimation trial, including an "=" for a response within the time window, "<2" for a response of less than 2s after the cue, "<3.5" for a response between 2s and the targeted 3.5s, ">3.5" for a response between the targeted 3.5s and 5s, and ">5" for a response slower than 5s. Below that line, the screen displayed whether the participant had won money for the trial, with either a "\$" indicating the participant won \$0.20 (only for good performance in reward trials) or "0" indicating the participant won no money for the trial (for bad performances on reward trials or any performance on a no-reward trial). Feedback stayed on the screen for one second. The task consisted of six blocks, each with 36 trials (half "reward" and half "no-reward" within each block).

Procedure

After researchers obtained informed consent, participants were led through the reward time-estimation task described above. Participants were then administered a variety of questionnaires gauging demographics, as well as a battery for depressive symptoms (Beck Depression Inventory II, BDI-II) and a battery for hypomanic symptoms (Hypomanic Personality Scale, HPS), as well as others not used in the current study. All of these batteries have been tested for validity and consistency in previous literature. All data were collected by Michael

Vanderlind, a PhD candidate in clinical psychology at Yale, while he studied at Northwestern University. Data collection was completed in March 2015.

EEG Equipment

EEG data were collected from inside an electro-magnetically shield booth, and were sampled continuously at 500 Hz (DC to 100Hz on-line, Neuroscan Inc.) from thirty-two Ag/AgCl scalp electrodes. Impedance was kept below $5k\Omega$ for the scalp and $10k\Omega$ for the eye electrodes. EEG recordings were referenced both offline (to linked mastoids) and online (to the left mastoid). For offline analyses, Horizontal electrooculogram (HEOG) and vertical electrooculogram (VEOG), which can contaminate EEG data, were recorded using four eye electrodes, and were corrected for using PCA algorithms in the NeuroScan EDIT (Neuroscan Inc.). EEG artifacts related to head movement were corrected for manually, and epochs with artifacts ($\pm 75 \mu\text{V}$) were rejected. Recordings were offline bandpass-filtered at .01-30 Hz.

Data Analysis

For ERP analysis, “<2” or “>5” trials (that is, very fast or very slow responses) were not included, and ERPs were measured during each trial’s reward cue and feedback stages. The current study looked at the SPN, N2, FB-P3, and LPP ERP components, with specific time windows and electrode sites for each component were used based on previous studies. The SPN component’s mean amplitude was measured between 2600 and 3000ms after participant response (button press), during a 400ms time interval during which the feedback was on the screen, at the EEG cap’s Cz site (C = central, z = midline of the head). The N2 component’s mean amplitude was measured between 200 and 350ms at the EEG’s Pz site (P = parietal) (Potts, 2011). The FB-P3 and the LPP, which are both feedback-locked, were epoched from -100 to 1000ms in reference to the onset of feedback, and feedback stimuli were coded based on if

participants responded accurately or inaccurately. The FB-P3 component's mean amplitude was measured between 350 and 500ms at the EEG cap's Pz site (Yeung & Sanfey, 2004). The LPP component's mean amplitude was measured between 450 and 950ms at the CPz site (CP = centroparietal, z = midline of the head) (Schupp et al., 2004).

For SPN and N2, the two anticipatory ERPs, differences in amplitude between reward trials and no-reward trials (“ Δ SPN,” “ Δ N2”) were calculated to determine how anticipatory potentials differed when there was a possible reward during the trial. For the FB-P3 and the LPP, the same differences were calculated, but were categorized by whether the participant accurately or inaccurately completed the task (“ Δ FB-P3-good,” “ Δ FB-P3-bad,” “ Δ LPP-good,” and “ Δ LPP-bad”). This was so that we could observe consumption-based reward processes both when the participant actually received a reward and when the participant failed to earn a reward when a reward was possible.

Results

Paired *t*-tests were conducted on N2 and SPN amplitudes across reward-cue conditions to determine whether these ERPs significantly differed between trials. The amplitude of N2 was significantly less negative in reward trials ($M=-3.52$, $SD=4.33$) than in no-reward trials ($M=-4.91$, $SD=3.76$), $t(35)=3.08$, $p<.01$. The amplitude of SPN was significantly more negative in reward trials ($M=-5.10$, $SD=2.57$) than in no-reward trials ($M=-3.20$, $SD=2.62$), $t(35)=-7.26$, $p<.001$.

Then, a 2 (cue: reward, no-reward) x 2 (outcome: good performance, bad performance) repeated-measure ANOVA was conducted for FB-P3 and LPP, the two outcome-related ERPs, to see if there were main effects for these components across cue type and outcome type. For FB-

P3, results revealed a main effect of cue type, $F(1, 35)=104.2, p<.001, \eta^2_p=.75$, as well as a main effect of outcome, $F(1, 35)=4.42, p=.043, \eta^2_p=.11$. The amplitude of FB-P3 was significantly greater in the reward trials ($M=16.86, SD=.96$) than in no-reward trials ($M=12.18, SD=.91$), and also significantly greater after good performance ($M=15.07, SD=.94$) than after bad performance ($M=13.97, SD=.95$). For LPP, results revealed a main effect of cue type, $F(1, 35)=103.75, p<.001, \eta^2_p=.75$, as well as a main effect of outcome, $F(1, 35)=47.38, p<.001, \eta^2_p=.58$. The amplitude of LPP was significantly greater on reward trials ($M=9.70, SD=.57$) than on no-reward trials ($M=6.97, SD=.54$), and was also significantly greater after bad performance ($M=9.68, SD=.60$) than after good performance ($M=6.99, SD=.55$).

Depressive symptoms, measured by the BDI-II, ranged from 0 to 28 ($M=8.53, SD=6.72$), while hypomanic symptoms, measured by the HPS, ranged from 3 to 34 ($M=16.47, SD=7.90$). We examined the relation between depressive symptoms, manic symptoms, and SPN, FB-P3, and LPP amplitudes through zero-order correlations. Bivariate correlation analysis were conducted between depressive symptoms, hypomanic symptoms, Δ SPN ($M=-1.90, SD=1.57$), Δ N2 ($M=1.40, SD=2.72$), Δ FB-P3-good ($M=5.20, SD=3.11$), Δ FB-P3-bad ($M=4.15, SD=3.22$), Δ LPP-good ($M=2.54, SD=2.03$), and Δ LPP-bad ($M=2.92, SD=2.42$). No significant results correlations between symptoms and ERP components were found, and results are summarized in Table 1.

Discussion

The current study investigated the relationship between anticipation and consumption of reward and depressive and manic symptoms. We aimed to learn more about whether depressive symptoms are associated with hypoactive reward processing and hypomanic symptoms with

hyperactive processing. Our results ultimately found no relationship between the four ERPs we studied and depressive and hypomanic symptoms, but did validate the task we used and provide insight on future directions.

Our results validated the time-estimation reward task created by Pornpattananangkul and Nusslock, with both anticipation-based ERPs showing significant differences between trial types, and with both consumption-based ERPs showing main effects for both cue type and outcome type (Pornpattananangkul & Nusslock, 2015). As predicted, the SPN was significantly more negative during reward trials than non-reward trials, confirming prior research suggesting that the SPN's magnitude increases through anticipation of reward (Kotani et al., 2003). Similarly, the FB-P3 had significantly higher amplitude during reward trials than non-reward trials, and during good performances than bad ones, confirming prior research arguing that the FB-P3 increases as reward salience increases (Gu et al., 2011). The LPP was also significantly greater during reward trials than during no-reward trials, and was significantly greater on bad performances than on good ones. Given the uncertainty regarding the exact role of LPP in reward, these results could mean one of many things; perhaps, the LPP was greater in reward trials and bad performance trials because these were more emotionally arousing than their counterparts, or perhaps these two trial types induced more reinforcement learning (reward trials would be more salient, and bad performances might cause participants to modify behavior). The N2, however, was significantly smaller in magnitude in reward trials than in no-reward trials, contrary to hypotheses; prior literature on the N2 suggests that higher levels of cognitive control yield larger amplitudes, and we would expect participants to exhibit higher levels of cognitive control when there was a possibility of reward (Folstein & Van Petten, 2008). Further research into the factors modulating N2 could help clarify our results. Overall, this task could continue to

be used in future studies to better understand the relationships between anticipation-based and consumption-based reward processes.

We found no zero-order correlations between any of the ERPs we measured and either depressive or hypomanic symptoms, contrary to our hypotheses. Our study was limited in many ways that could have affected our results. For one, our sample was relatively small, with only 36 total participants, and the sample was generally very heterogeneous in terms of symptomatology, with BDI-II scores ranging from 0 to 28 and HPS scores ranging from 3 to 34. Because our range of symptoms was relatively small, and because almost all participants fell in sub-clinical range for depressive and hypomanic symptoms, our data may have been influenced heavily by floor effects in symptomatology, making any sort of relationship between symptoms and ERPs tough to illuminate with high resolution. Future studies looking with larger sample sizes could split participants into groups based on symptomatology (for example, <13 vs >13 on the BDI-II) and compare group differences, or could just use clinical MDD and PD samples to gain a better idea of how symptoms are related to ERPs. Additionally, the current study was limited in that it was cross-sectional; future research could track participants over time, perhaps observing whether indices of reward changed as levels of symptomatology increased or decreased.

The lack of correlations between LPP and our depressive or hypomanic symptoms also illuminates how little is understood about the LPP waveform itself. Some studies have suggested that the LPP is modulated by emotional arousal, while others argue that the LPP is related to integrating reward-related feedback into behavior (Kisley, Wood, & Burrows, 2007; Pornpattananangkul & Nusslock, 2015). Thus, our results might just suggest that our subjects generally did not experience significant emotional arousal during our task, rather than suggesting that depressive and manic symptoms are not associated with deficits in reinforced learning.

Further research about the LPP's precise role in reward processing might elucidate our findings, and future studies might consider looking at another reward-based ERP related to reinforced learning.

The current study may have been limited in the measures it used. The Hypomanic Personality Scale gauges personality traits related to hypomania, but does not actually gauge for symptoms. Some recent research has criticized the HPS because it has largely been tested only on non-clinical populations, and that when administered with clinical populations, it appears to be highly confounded with hypomanic and manic symptoms (Parker, Fletcher, McCraw, & Hong, 2014). Our study aimed to use the HPS to measure hypomanic symptomatology, and this measure may not have been the ideal tool to measure that. Future studies could consider using tools like the Minnesota Multiphasic Personality Inventory or other measures to get a more direct estimate of hypomanic and manic symptomatology.

Overall, this study sought to illuminate a gap in current literature and to better understand the deficits associated with MDD and BPD at specific points in reward processing. While the current study was limited in a number of ways, future studies using EEG and time-estimation reward tasks with larger, more symptomatically diverse samples could help shed some light on this area of research with more resolution and detail.

Acknowledgements

First and foremost, I want to thank Professor Jutta Joormann for her guidance and her generosity, and the Affect Regulation and Cognition Lab for providing me the resources and support to complete this project. I would like to specifically thank Michael Vanderlind, Libby Lewis, and Vera Vine for their unwavering support of my studies in the ARC Lab, and for

dedicating time and resources to helping me hone in on my areas of interest within the field of clinical psychology. Finally, I want to thank Professor Joshua Knobe, Dr. Mark Sheskin, and the rest of the Cognitive Science program for providing mentorship and institutional support over the course of my undergraduate studies at Yale. Without all of these people, this project would have been impossible.

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Table 1. Zero-order correlations between current depressive symptoms, current hypomanic symptoms, and electrocortical indices of reward anticipation (SPN) and consumption (FB-P3, LPP)

	1.	2.	3.	4.	5.	6.	7.
1. BDI-II	---						
2. HPS	.45**	---					
3. SPN	.21	-.03	---				
4. ΔFB-P3-good	-.04	-.10	-.41**	---			
5. ΔFB-P3-bad	-.12	-.20	-.67**	.51**	---		
6. ΔLPP-good	-.19	.05	-.18	.38*	.03	---	
7. ΔLPP-bad	-.13	-.16	-.70**	.43**	.77**	.04	---

Note. “Good” in this case refers to the amplitude of the ERP in trials where participants accurately completed the task and received reward, while “bad” in this case refers to the amplitude of ERP in trials where participants inaccurately responded and did not receive money.

** denotes $p < .05$ while ** indicates $p < .01$*

Figure 1. Schematic representation of time-estimation reward task (Pornpattananankul & Nusslock, 2015)

