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Individual differences in the time course of reward processing: Stage-specific links with depression and impulsivity

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ABSTRACT

Reward dysfunction has been implicated in a wide range of psychological disorders, including internalizing and externalizing psychopathology. Basic neuroscience research has shown that reward is a multistage process, yet it is unclear how specific stages relate to individual differences in reward sensitivity. The current study utilized event-related potentials elicited during a monetary incentive task to parse sub-stages within anticipatory and consummatory reward processing. Effects of depressive symptoms and trait impulsivity were examined at each sub-stage (N = 92). Reward anticipation modulated neural activity across three sub-stages: cue detection (cue-P3), approach behavior (contingent negative variation, CNV), and outcome anticipation (stimulus preceding negativity). Reward delivery modulated activity across two sub-stages: initial evaluation (reward positivity, RewP), and allocation of attention (feedback-P3). Sensation seeking predicted faster reaction times, as well as cue-P3 and RewP amplitudes. Depression and lack of premeditation interacted to predict CNV and RewP amplitudes. Results demonstrate that individual differences in reward functioning are stage-specific.

1. Introduction

Reward has well-characterized neural circuitry, with converging evidence across animal and human samples. In particular, the mesolimbic circuit is thought to be critically involved in reward processing and includes the ventral tegmental area, the nucleus accumbens, and medial and orbital frontal cortices (Berridge & Kringelbach, 2008; Liu, Hairston, Schrier, & Fan, 2011). Broadly, reward processing can be broken down into three core components of 'liking' (hedonic impact), 'wanting' (incentive salience), and 'learning' (predictive associations and cognitions), each of which can be mapped onto distinct neuroanatomical and neurochemical systems (Berridge, Robinson, & Aldridge, 2009).

In line with this componential framework, reward is best understood not as a unitary phenomenon but as a dynamic set of processes that unfold over time. Neuroimaging studies distinguish broadly between anticipatory and consummatory reward processing, with overlapping neural networks involved in each stage (Liu et al., 2011). While fMRI provides excellent spatial resolution for identifying relevant reward-related neural circuitry, it lacks the temporal resolution to precisely characterize the time course of activity therein. For example, the broad stage of reward "anticipation" in fMRI studies often spans initial cue detection, motor preparation, target processing, and outcome anticipation; these represent functionally distinct anticipatory sub-stages, yet they cannot be isolated with fMRI alone. Event-related potentials (ERPs), on the other hand, have millisecond temporal resolution and are well-suited to dissecting the temporal dynamics of reward processing in a fine-grained manner.

The overarching goals of the present study are to (a) isolate a comprehensive set of reward sub-stages using ERPs (i.e., sub-stages within each of the broader stages of "anticipatory" and "consummatory" processing) and (b) test for individual differences in reward processing using this stage-wise approach. We focus here on measures of depressive symptom and trait impulsivity, both of which have been related to abnormalities in reward processing but which have not yet been systematically examined together. By combining measures of depression and impulsivity within in a relatively large, unselected sample, we intend to establish a foundation for future research in relevant clinical populations.

1.1. Stages of reward processing

In a previous study, we examined the temporal dynamics of reward using an adapted version of the monetary incentive delay (MID) task (Novak & Foti, 2015). The MID task is designed to sepa-

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rate anticipatory from consummatory reward processing, and it has been widely used in fMRI studies, both in basic research (Knutson, Fong, Adams, Varner, & Hommer, 2001; Knutson, Westdorp, Kaiser, & Hommer, 2000) and clinical applications (Knutson, Bhanji, Cooney, Atlas, & Gotlib, 2008; Strohle et al., 2008). The typical trial structure of the MID task is as follows: a cue signals the contingency for that trial (e.g., monetary incentive, neutral), followed by a target stimulus that requires a button press. On incentive trials, fast responses to the target result in reward feedback, whereas slow responses result in punishment. In fMRI studies, the "anticipation" period typically spans the presentation of the cue, anticipation of the target, motor response, and anticipation of the feedback (Knutson et al., 2000).

In our ERP design, we further divided reward anticipation into two specific sub-stages: cue detection and target anticipation (i.e., approach behavior). The allocation of attention to reward-predicting cues is captured by the cue-P3, an ERP that peaks between 300 and 500 ms post-stimulus at parietal sites. The P3 is generally more positive for salient, task-relevant, or unexpected stimuli (Polich & Kok, 1995), and in the MID task it is increased for incentive versus neutral cues (Novak & Foti, 2015). The transition from cue processing to motivated approach behavior is captured by the contingent negative variation (CNV), a negative-going slow wave associated with cued motor preparation (Brunia, van Boxtel, & Böcker, 2012). On the MID task, the CNV elicited in anticipation of the target stimulus is larger for incentive versus neutral trials (Novak & Foti, 2015). A third sub-stage within reward "anticipation" is the period after the motor response and in anticipation of feedback delivery, which ought to elicit a stimulus-preceding negativity (SPN). While we did not consider the SPN in our previous study, other research has shown that it is also sensitive to reward contingencies (Foti & Hajcak, 2012; Kotani et al., 2003; Ohgami, Kotani, Hiraku, Aihara, & Ishii, 2004; Ohgami et al., 2006). We expected here that the SPN would be similarly increased in anticipation of feedback on incentive versus neutral trials.

Consummatory neural activity, meanwhile, was divided into two sub-stages: initial evaluation and allocation of attention (Novak & Foti, 2015). Initial evaluation is captured by the reward positivity (RewP; also known as the feedback negativity [FN], feedback-related negativity [FRN], and medial frontal negativity [MFN]), an ERP that represents the early, binary differentiation of favorable versus unfavorable outcomes (Hajcak, Moser, Holroyd, & Simons, 2006). It peaks 250–300 ms after feedback presentation and manifests as a relative negativity to monetary losses and as a relative positivity to monetary gains.[1] While traditionally conceptualized as a neural signal tracking the occurrence of unfavorable outcomes (i.e., errors or monetary loss) (Gehring & Willoughby, 2002; Holroyd, Coles, & Nieuwenhuis, 2002; Miltner, Braun, & Coles, 1997), recent research has indicated that RewP amplitude is largely driven by reward delivery (Foti, Weinberg, Dien, & Hajcak, 2011; Holroyd, Krigolson, & Lee, 2011; Holroyd, Pakzad-Vaezi, & Krigolson, 2008; Proudfit, 2015), with a larger RewP associated with self-reported and behavioral reward sensitivity (Bress & Hajcak, 2013) and reward-related activity in the striatum and medial frontal cortex (Becker, Nitsch, Miltner, & Straube, 2014; Carlson, Foti, Mujica-Parodi, Harmon-Jones, & Hajcak, 2011; Foti, Carlson, Sauder, & Proudfit, 2014). Immediately following the RewP, the allocation of attention to uncertain outcomes is captured by the feedback-P3 (fb-P3). On the MID task, the fb-P3 is primarily sensitive to outcome salience rather than valence; it is increased for both wins and losses on incentive trials versus break-even feedback on neutral trials (Novak & Foti, 2015)

1.2. Individual differences in reward processing

In our previous study the cue-P3, CNV, RewP, and fb-P3 were all highly sensitive to reward contingencies, yet the degree of reward-related modulation was only modestly correlated *across* ERPs (Novak & Foti, 2015). For example, the cue-P3 and fb-P3—while having similar morphologies—were not significantly correlated with one another, indicating the unique allocation of attention toward reward cues versus reward outcomes, respectively. These results suggest that a stage-wise approach, combining information from multiple ERPs, will be fruitful for characterizing reward dynamics, allowing for a more fine-grained characterization of reward processing.

A key gap in the literature is how these stages may uniquely map onto individual differences in reward sensitivity. For example, a substantial literature has examined reward dysfunction in major depression (Pizzagalli, 2014). Depression is typified by anhedonia, the significant loss of interest and pleasure in normally enjoyable activities. In experimental studies, depressed individuals fail to exhibit reward-related speeding in reaction time (Henriques & Davidson, 2000; Henriques, Glowacki, & Davidson, 1994; Pizzagalli, Jahn, & O'Shea, 2005) and are behaviorally insensitive to reward contingencies (Henriques & Davidson, 2000; Pizzagalli, Iosifescu, Hallett, Ratner, & Fava, 2008). fMRI studies, meanwhile, have linked depression with reduced reward-related neural activity throughout the mesolimbic circuit, particularly in the ventral striatum (Elliott, Sahakian, Michael, Paykel, & Dolan, 1998; Epstein et al., 2006; Keedwell, Andrew, Williams, Brammer, & Phillips, 2005; Knutson et al., 2008; Pizzagalli et al., 2009; Tremblay et al., 2005).

Complementing these data, recent ERP studies have linked depression with reduced neural sensitivity to reward outcomes (Proudfit, 2015). In both clinical (Foti et al., 2014; Liu et al., 2014) and non-clinical samples (Bress, Meyer, & Hajcak, 2013; Bress, Smith, Foti, Klein, & Hajcak, 2012; Foti & Hajcak, 2009), symptoms of depression have been associated with a blunted RewP. This may represent a neurobiological mechanism of risk (Foti, Hajcak, Kotov, & Klein, 2011), such that a blunted RewP has been shown to prospectively predict the first episode depression onset (Bress, Foti, Kotov, Klein, & Hajcak, 2013). Other studies have linked depression with abnormal EEG activity in the alpha frequency band when anticipating the possibility of reward, which is interpreted to reflect an underactive approach system (Nelson, Shankman, & Proudfit, 2014; Shankman, Klein, Tenke, & Bruder, 2007; Shankman et al., 2013).

Separate from this research on reward dysfunction in depression, other studies have examined the impact of trait impulsivity on reward processing. Impulsivity is featured in every major model of personality (Cloninger, Przybeck, & Svrakic, 1991; Cloninger, Svrakic, & Przybeck, 1993; Eysenck & Eysenck, 1985; Tellegen, 1982, 1985; Whiteside & Lynam, 2001) and is a consistent correlate of a variety of problematic behaviors in clinical samples (Anestis et al., 2009; Black et al., 2013; Dawe & Loxton, 2004). Like reward, however, impulsivity is not a unitary phenomenon. Research over the last 15 years suggests that impulsivity may be an "artificial umbrella term" that references relatively distinct and separable "impulsigenic" traits (Cyders & Smith, 2008; Sharma, Kohl, Morgan, & Clark, 2013; Whiteside & Lynam, 2001). Five such traits appear in the UPPS model (Whiteside & Lynam, 2001): Negative/Positive Urgency, (lack of) Premeditation, (lack of) Perseverance, and Sensation Seeking. High levels of these impulsigenic traits predict a

[1] In previous FN/FRN/MFN studies, this ERP has been scored as the loss minus win difference, yielding a prominent negativity at frontocentral electrodes. In light of findings linking this ERP to reward sensitivity, it has been proposed that it may be more appropriate to instead take the win minus loss difference (Proudfit, 2015), which yields a positivity at frontocentral electrodes. The magnitude of the valence effect (win vs. loss) is the same in each case, although the sign is the opposite: emphasizing loss-related activity in the former and gain-related activity in the latter.