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Pre-ejection Period Reactivity to Reward is Associated with Anhedonic Symptoms of Depression among Adolescents

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Abstract

Pre-ejection period (PEP) reactivity to reward has been posited as a specific index of behavioral approach and incentive motivation, suggesting it might be uniquely associated with the affective and motivational deficits of anhedonia. This study evaluated PEP reactivity to a reward task as a predictor of depressive symptoms among adolescents, examining global depressive symptoms as well as specific anhedonic and nonanhedonic symptoms clusters. Participants included 76 adolescents, ages 11–15 years (52% female). This study found marginal support for an association between PEP reactivity to reward and concurrent anhedonia symptoms, but no association with nonanhedonic or the global scale. Findings are discussed in terms of potential associations between peripheral psychophysiological measures and dopaminergic functioning and also the utility of this measure for future research on anhedonia.

Anhedonia refers to a decrease in interest or pleasure in response to previously rewarding stimuli (American Psychiatric Association, 2013), impaired motivation, reinforcement learning, and reward-based decision making (Pizzagalli, 2014). Anhedonia and its associated motivational deficits may be present with or without the negative valence components of depression (Chen, Eaton, Gallo, Nestadt, 2000). Approximately one-third of depressed individuals experience clinically significant levels of anhedonia (Pelizza & Ferrari, 2009) which is associated with elevated risk for future depressive symptoms (Hundt et al., 2007), prolonged time to recovery (McFarland et al., 2006), depression chronicity over 10 years (Moos & Cronkite, 1999), and suicidal ideation and attempts over a 2-year period (Spijker et al., 2010).

Anhedonia is characterized by deficits in positive affect and motivation, and as such may be associated specifically with deficits in reward processing. Research supports a strong link between anhedonia and deficits in anticipatory responding to incentives in the mesolimbic dopamine pathway (Zisner & Beauchaine, in press). Pre-ejection period (PEP) reactivity, a cardiac measure of sympathetic nervous system responding, has been associated with reward motivation, behavioral approach, and mesolimbic dopamine responding to rewards (Richter & Gendolla, 2009; Zisner & Beauchaine, 2016). Recently, researchers have utilized PEP reactivity to reward and stress to examine the symptoms observed in depression (Brinkmann

& Franzen, 2013; Franzen & Brinkman, 2015; Silvia, Nusbaum, Eddington, Beaty, & Kwapil, 2014). However, these studies have examined depression globally and not anhedonia specifically, limiting the inferences that may be made about the pathophysiology of anhedonia. The present study examines blunted pre-ejection period reactivity to reward as a biomarker specific to anhedonic symptoms and not the negative affective and disrupted eating and sleeping symptoms of depression.

Depression and Blunted Physiology

Blunted physiological responses to both stress and reward paradigms are globally associated with and predictive of depression (Brinkmann, Franzen, Rossier, & Gendolla, 2014; Carroll, Phillips, & Der, 2008; de Rooij, Schene, Phillips, & Roseboom, 2010; Franzen & Brinkmann, 2015; Harkness, Stewart, Wynne-Edwards, 2011; Phillips, Hunt, Der, & Carroll, 2011; Pizzagalli et al., 2009; Rottenberg, Clift, Bolden, & Salomon, 2007; Salomon et al., 2009; Schwerdtfeger & Rosenkaimer, 2011; York, Hassan, Li, Li, Fillingim, & Sheps, 2007). Despite the uniformity in these findings, one limitation to the current literature may be that showing overall blunted physiological responding does not progress our understanding of the pathophysiological mechanisms that underlie blunted peripheral reactivity in anhedonia symptoms specifically (Phillips, Ginty, & Hughes, 2013). For example, much of the physiological research evaluates depressive symptoms globally (e.g. Brinkmann et al., 2009); however, the specific symptom cluster of anhedonia is related to deficient motivation and reward responding, which may be present independent of negative affect components of depression (Chen, Eaton, Gallo, Nestadt, 2000), and thus may be characterized by disruptions in central nervous system substrates associated with these positive valence system components. Research may require more nuanced questions to identify the precise physiological mechanisms underlying anhedonia. One potential correlate of anhedonia may be mesolimbic dopamine dysfunction, which has extensive evidence implicating it as a risk factor for mental health problems marked by reward-based impairments (Franken, Booij, & Brink, 2005; Heinz, 2002; Pizzagalli, 2014; Wise, 2008; 2009).

Blunted Central Dopamine Function and Anhedonia

The mesolimbic dopaminergic (DA) pathway is implicated in reward processing and reinforcement learning and has become a central neurological substrate associated with major depressive disorder (Forbes et al., 2009; Forbes & Dahl, 2012; Pizzagalli, 2014). Moreover, mesolimbic DA transmission is theorized to contribute specifically to anhedonia and reduced motivation (Nestler & Carlezon, 2006). Mesolimbic dopamine is involved in incentive motivation (Berridge, 2007) and behavioral approach (Brenner, Beauchaine, & Sylvers, 2005; Gatzke-Kopp et al., 2009), and several neuroimaging and dopamine depletion studies implicate compromised dopamine transmission in reward and incentive insensitivity observed in major depressive disorder (see Pizzagalli, 2014). Thus, it may be that individual differences in mesolimbic dopamine levels may manifest behaviorally as self-report anhedonia and peripherally as blunted physiological responding to reward tasks.

Pre-ejection Period and Central Dopamine Reactivity

Cardiac pre-ejection period (PEP) refers to the duration of time spanning left-ventricular depolarization to the ejection of blood into the aorta (Sherwood, Allen, Fahrenberg, Kelsey, Lovallo, & van Doormen, 1990). Although basal PEP is subject to influence from multiple autonomic and central nervous system sources, PEP reactivity is almost exclusively determined by sympathetic (β -adrenergic) influences (Sherwood, Allen, Obrist, & Langer, 1986) such that increases in SNS arousal correspond to shortening PEP. Researchers have frequently used PEP reactivity as a broad-stroke measure of SNS reactivity to examine the physiological effects of stress, fear, and anxiety (Beauchaine, 2001; Boyce et al., 2001; Bubier & Drabick, 2008). Although diminished PEP reactivity (i.e. smaller PEP decreases in response to stimuli) may reflect a general biological hypo-responsivity to stimuli (see Ginty, 2013), inferences about specific central nervous system processes cannot be made without considering stimulus conditions (Phillips et al., 2013; Zisner & Beauchaine, 2016).

As outlined elsewhere, extensive theory and research suggest that PEP reactivity to carefully selected reward-based stimuli serves as a peripheral marker of mesolimbic DA reactivity to incentives (Beauchaine et al., 2007, 2013; Beauchaine & Gatzke-Kopp, 2012; Brenner & Beauchaine, 2011; Brenner et al., 2005; Zisner & Beauchaine, 2016). The summary of their research indicates the SNS facilitates the metabolic demands of approach motivated behaviors through changes in cardiac output via increases in contractile force of the left ventricle (Sherwood et al., 1986, 1990). Further, changes in SNS are modulated by dopamine (Mannelli et al., 1999) and direct infusions of dopamine agonists into the mesolimbic dopamine system increase cardiac output via the SNS (van den Buuse, 1998), similar to that observed among healthy participants administered reward tasks (Brenner et al., 2005; Richter & Gendolla, 2009).

Several studies within the externalizing literature provide empirical support implicating blunted PEP and DA responding to reward in the etiology of psychopathology (for a review see Beauchaine & Gatzke-Kopp, 2013). Given the comorbidity between externalizing disorders and depression (Hink et al., 2013) and neuroimaging studies suggesting similar disturbances in mesolimbic dopamine functioning across these disorders (Forbes & Dahl, 2012; Gatzke-Kopp et al., 2009; Zisner & Beauchaine, in press), it stands to reason that similar PEP reactivity to reward may be observed in depressed individuals reporting anhedonic symptoms. As reviewed above, the loss of incentive motivation and behavioral approach observed in anhedonia is theoretically tied to dysfunction in dopaminergic pathways (Forbes & Dahl, 2012; Pizzagalli, 2014). Research that attempts to examine the integrity of these pathways must utilize refined assessments of the constructs purportedly involved. Given the heterogeneity in depression (Chen, Eaton, Gallo, Nestadt, 2000), it may be that the effects of impaired central dopamine function, and by extension, reduced PEP reactivity to reward, may manifest only in theoretically expected symptom clusters such as anhedonia.

PEP Reactivity and Depression

Several studies have examined PEP responses to both stress and reward and their associations with depressive symptoms and, as expected, most find that attenuated PEP reactivity is associated with greater symptoms (Brinkmann et al., 2009; Brinkmann et al., 2014; Brinkmann & Franzen, 2013; Franzen & Brinkmann, 2015; Salomon, Bylsma, White, Panaite, & Rottenberg, 2013; Silvia et al., 2014; although see Salomon, Clift, Karlsdóttir, & Rottenberg, 2009). Although the pattern of significant attenuated PEP reactivity is consistent across these studies, PEP changes to stress are likely induced via brain systems that subserve other motivational states (Brenner & Beauchaine, 2011). Additionally, all but one of the above studies fails to measure anhedonia specifically. Rather, these studies utilize global scales of common depression self-report measures, which limits the anhedonic-specific inferences that can be made with regard to reward responding and central DA functioning.

The Current Study

Although several studies have used PEP reactivity to reward to evaluate depression symptoms, none have evaluated the differential associations of physiological responses to reward and anhedonia specific clusters of symptoms. The goal of the current study was to evaluate the relationships between PEP reactivity to reward and global depressive symptoms and specific symptoms clusters such as anhedonia. We hypothesized that PEP response to reward would be specifically associated with anhedonic symptoms.

Method

Participants

Participants were 76 (52% female) adolescents aged 11–15 years ($M = 13.29$, $SD = 0.78$) enrolled in public schools in the Pacific Northwest. Approximately 79% were Caucasian; 8% were Asian-American; 1% were African-American; 12% identified as biracial or other. Youth were invited to participate in the school-based screening if they were (1) 10 to 14 years old; (2) in 5th to 8th grades; and (3) if they and one parent were sufficiently fluent in English to complete study questionnaires. Parents provided consent and youth provided assent for screening. Because the purpose of the broader study was to identify prospective pathways to adolescent-onset depression, youth were invited to the laboratory visit only if they reported depressive symptoms below the clinical cutoff (i.e. total score of 13 or lower) on the Children's Depressive Inventory – 2nd Edition (CDI-2; Kovacs, 2010) at the screening visit. At screening, 88% of youth were below the clinical cutoff and therefore eligible to participate.

Procedure

All procedures were IRB approved, and prior to each visit both parent/guardian and youth provided consent. Youth were invited to laboratory visit between 4–6 months after screening. At this visit, youth completed self-report measures and laboratory activities.

Approximately six months after the T1 visit, youth were invited to the laboratory visit where they completed self-reported measures on a desktop computer and then participated in a 3-

minute baseline period in which they viewed neutral nature scenes. Following this, participants were presented with a delayed-matching-to-sample task based on the task used by Richter and Gendolla (2009). In this task, each trial began with an image of randomly placed black points on a white background displayed for 440 ms. Following this, a new image appeared for 2400 ms with some of the black points changed to grey. The first image was presented again for 800 ms, followed by a final image which appeared for 1600 ms in which multiple black points were again changed to grey. Participants were then shown a screen asking if the two images that contained grey points were different or the same. In half the trials, the target images were identical, and on the other half, the second image had a single grey point that differed from the first image. Participants were instructed that the computer would randomly decide a performance expectation to determine if they would earn an additional \$10 gift card following their visit. After each correct response, a screen informed the subjects they had responded correctly and that they had received points toward the performance cutoff. This reward task and incentive have been shown to reliably produce PEP responses in nondepressed individuals (Richter & Gendolla, 2009). In accordance with research suggesting effort-based tasks be unfixed in difficulty (Wright et al., 2002), each task was presented until the participant made a response. Participants were further cued to try to gain the highest score possible to increase their likelihood of obtaining the card. Regardless of the participant's performance, following the task, the computer informed the participant they had eclipsed the benchmark and would receive the \$10 gift card. Youth were paid a total of \$20 for the visit and parents were paid \$25.

A total of 141 participants completed the initial laboratory visit. However, the administration of the reward task at the 6-month follow-up was piloted to only a random subsample of the original 141 participants ($N = 96$). The 45 participants that were not administered the reward task did not differ from those who did complete the reward task in terms of demographic, predictor, or outcome variables. Due to excessive movement artifacts or technical problems (e.g. loosening leads), PEP data were not usable for 19 youth and these participants were not included in the analyses. Finally, one participant was excluded from analyses due to insufficient self-report data, yielding a final sample of 76 adolescents.

Measures

Pre-ejection Period—Cardiac PEP was derived using electrocardiography and impedance cardiography to determine the time interval between left-ventricular depolarization and ejection of blood into the aorta. Electrocardiograph data were acquired using a BIOPAC MP150 Data Acquisition Unit and thoracic impedance was acquired using a BIOPAC NICO100C Noninvasive Cardiac Output Module (Goleta, CA), sampled at 1 kHz, and processed offline using MindWare Technologies IMP 3.0.10 analysis program (Gahanna, OH). Data were visually inspected for incorrect placement of markers by the automated scoring algorithm and corrected as needed by trained research assistants. PEP was ensemble averaged using 30-second epochs. The average PEP value across the three minutes of the vanilla baseline was used to create a single basal PEP score. PEP reactivity to the reward paradigm was determined by first averaging participants PEP across the three minute reward task. Second, change scores were computed by subtracting basal PEP from PEP across reward tasks. Thus, positive change scores reflect a decrease in sympathetic arousal (i.e.,

lengthening PEP) and negative change values reflect an increase in sympathetic arousal (i.e., shortening PEP).

Depressive Symptoms—Youth depressive symptoms were assessed with the Children's Depression Inventory – 2nd Edition (CDI-2; Kovacs, 2010). The CDI-2 is a 28-item self-report inventory that inquires about the presence of depressive symptoms within the past two weeks; it is normed for use with youth aged 8 to 17. Each item contains three statements; participants were asked to select the statement that best described them in the previous two weeks. Total scores on the CDI can range from 0 to 54, with higher scores indicating more severe depressive symptoms. The CDI has repeatedly demonstrated excellent internal consistency (alpha reliability ranges from .80 to .87), test–retest reliability, and predictive and construct validity, especially in community samples (Blumberg & Izard, 1986; Kovacs, 1981, 1985). The CDI-2 was administered at screening and youth with scores greater than 14 were not eligible for the follow-up lab visit. Children were re-administered the CDI-2 at the first and second lab visit. The internal consistency of the CDI-2 at screening was excellent (.88) and at the laboratory visits were adequate (T1 = .79; T2 = .77).

To examine the different symptom clusters of depression, separate scores were created to reflect anhedonia symptoms and negative emotionality/other depression symptoms. Although the CDI factor structure does not specify a specific anhedonia scale (Kovacs, 2010), previous work indicates specific CDI items may be selected to create an anhedonia and low positive affect symptom scale (Chorpita et al., 1998; Hankin, 2008; Logan et al., 2013; Wetter & Hankin, 2009). In the present study, the six CDI items (numbers 4, 12, 15, 20, 21, & 22; Chorpita et al., 1998) were summed to create an anhedonia scale. The greatest level of endorsement for each of these items was: *I do not want to be with people at all; I feel alone all the time; I never have fun at school; I don't have any friends; I have to push myself all the time to do my schoolwork; Nothing is fun*. To calculate a score containing the remaining depressive symptoms, the total anhedonia scale was subtracted from the overall CDI score to yield a nonanhedonic depressive symptom score. This symptom cluster consisted of items reflecting negative mood, sleep impairment, and appetite disruptions.

Results

Variable correlations and descriptives are presented in Table 1. One PEP reactivity outlier was identified and this change score was winsorized. To examine our first hypothesis, data were analyzed using linear regression in SPSS 23.0. The first set of analyses tested PEP reactivity to reward as a predictor of CDI total symptom scores at T1 and at T2, controlling for sex. This was conducted using two separate linear regression analyses with total CDI scores at each visit as the dependent variable. The results for both analyses suggested that PEP reactivity to reward was not associated with total depressive symptoms at either time point (see Table 2).

To examine the differential relationships of PEP reactivity with depressive symptoms clusters, we performed a repeated measures multivariate analysis of covariance in SPSS 23.0 placing anhedonic and nonanhedonic symptoms at both T1 and T2 as dependent variables, PEP reactivity to reward at T2 as the predictor, and sex as a control. Of note, sex was

associated with anhedonic symptoms at T1 indicating that females reported greater anhedonia symptoms than males at T1.

The analysis revealed that PEP reactivity to reward was associated with T2 anhedonic symptoms such that individuals with smaller PEP changes to reward reported greater concurrent anhedonia. In contrast, PEP reactivity to reward was not associated with the remaining T2 nonanxious depressive symptoms cluster variable, suggesting the relationship between PEP reactivity to reward is specific to anhedonic symptoms and not global depressive symptoms. PEP reactivity to reward at T2 was not associated with T1 anhedonic symptoms or T1 nonanxious depression symptoms (see Table 3).

Discussion

The motivational and reward processing deficits of depression have been increasingly associated with attenuated PEP reactivity to laboratory tasks. In effort to identify the motivational deficits of depression, most researchers have utilized PEP reactivity to stressors and rewards as predictors of global depression scores. The aim of the current study was to extend this research by examining the specificity of PEP reactivity to a reward task and its relationship with different symptoms clusters of depression. Following from extant research we first tested the association between PEP reactivity to reward and global depressive symptoms. Contrary to what previous adult research has suggested, PEP reactivity to reward was not associated with global depressive symptoms. We further tested whether PEP reactivity to reward was uniquely associated with anhedonic versus nonanxious symptoms of depression. As expected, diminished PEP reactivity to our monetary incentive task was associated with concurrent anhedonia symptoms, but not associated with concurrent nonanxious symptoms. PEP reactivity to reward was not associated with anhedonic or nonanxious symptoms 6 months prior.

The findings of this study extend the research on PEP reactivity and depression in a couple of ways. First, this study extends our understanding of potential pathophysiological determinants of anhedonia by demonstrating that diminished PEP reactivity to reward was specifically related to anhedonic symptoms of depression. As briefly presented above and reviewed elsewhere (Beauchaine & Gatzke-Kopp, 2012; Brenner & Beauchaine, 2011; Brenner et al., 2005), PEP reactivity to incentives marks mesolimbic dopamine responding to the same tasks, and impaired mesolimbic dopamine functioning is implicated in development of anhedonia symptoms. Thus, the findings of the current study provide evidence that support greater inferences of the pathophysiology of anhedonia and depression. That is, it may be that diminished PEP reactivity to incentives specifically may be used as an index of central DA dysfunction and as a biomarker of the effort, motivation, and reward-processing deficits of anhedonia.

The results of this study also suggest a phasic relationship between PEP reactivity to reward and anhedonia. The analysis revealed PEP reactivity to reward was associated with anhedonic symptoms concurrently, but was not associated with anhedonic symptoms 6 months prior. Although we observed a strong correlation between anhedonia symptoms across the six months, temporal fluctuations in depressive symptoms are common even

among those persistently experiencing symptoms (Angst, Gamma, Rössler, Ajdacic, & Klein, 2009; Keller, Neale, & Kendler, 2007), and it may be that PEP reactivity to reward is sensitive to more proximally assessed anhedonic symptoms. Future research can evaluate whether PEP reactivity to reward is a state correlate of anhedonia by examining the stability of PEP reactivity to reward across visits in conjunction with anhedonia symptoms.

A further direction for future research is to examine potential factors that may impact PEP reactivity to reward such as adversity and stress (Zisner & Beauchaine, in press). Given the well-established link between stress and depressive symptoms (Brown & Harris, 1978; Hammen, 2005), future research may benefit from examining the effect of stress on anhedonia and PEP reactivity to reward. Notably, Keller and colleagues (2007) have found different categories of stressful life events predicted fluctuations of different depressive symptom profiles, including anhedonia. This may yield further support given animal research linking chronic stress to decreased motivated behavior, reward sensitivity, and dopaminergic pathways functioning (see Pizzgalli, 2014 for review). Furthermore, integrated cognitive vulnerability-stress models of depression suggest that individual differences in maladaptive cognitive styles and ruminative tendencies place some at increased risk for depression when confronted with stressful life events (Abela, Aydin, & Auerbach, 2006; Mezulis, Hyde, & Abramson, 2006). Thus, future research may further examine the effects of stress on PEP reactivity to reward and anhedonia as a function of individual differences in cognitions, behaviors, or regulation strategies.

Additionally, to our knowledge this is the first study to examine the relationships between PEP reactivity to reward and depressive symptoms specifically in children and adolescents. Although previous researchers have examined PEP reactivity to reward in children, these are typically associated with externalizing psychopathology (see Beauchaine et al., 2007) or undifferentiated internalizing symptoms (e.g. anxiety and depression; Nederhof et al., 2014). Consequently, this study extends the developing literature of reward reactivity and depression to childhood and adolescence.

One of the outstanding questions of the current findings is the failure to find any relationship between PEP reactivity and global depressive symptoms. Several of the adult studies evaluating PEP reactivity to reward separate participants into dysphoric and nondysphoric groups based on membership into the highest and lowest quartiles, respectively, of a depressive symptom measures (Brinkmann et al., 2009; Brinkmann et al., 2014; Brinkmann & Franzen, 2013; Franzen & Brinkmann, 2015). Such a split may have therefore included those that were elevated on anhedonia in addition to other symptoms of depression, which may have increased their ability to detect differences between these groups but reduced the interpretability of their findings in terms of symptoms.

The current study extends the literature by improving upon the functional inferences that can be made about PEP response to rewards and depression. Specifically, the results point to disrupted mesolimbic DA responding in adolescents reporting higher levels of anhedonia. Although this aids in our understanding of the deficits and substrates associated with depression, PEP reactivity to reward is only a proxy for central DA. Despite this limitation, PEP reactivity to reward is still a much cheaper way to evaluate risk for anhedonia and other

reward deficiencies and may yet be of use for future researchers. It is also necessary to note the borderline nature of our findings. The bivariate association between T2 anhedonia and PEP reactivity to reward trend toward significance and becomes significant only in the model with covariates. Furthermore, this finding would not remain significant if we adjusted for multiple comparisons and therefore should be interpreted with caution pending replication in other studies.

Limitations aside, this study highlights the importance of specificity in construct measurement and in stimulus and physiological variable selection. Previous research has illustrated the value of using psychophysiological variables to examine depression and at the same time has called for a better understanding of the mechanisms underlying these relationships (Phillips et al., 2013). Anhedonia is one of many possible symptoms of depression and prior research has provided specific hypotheses about the neural substrates and manifest behaviors. Here we have shown that PEP reactivity to reward is a particularly sensitive measure of anhedonic symptoms, which offers many opportunities to explore the etiological mechanisms of anhedonia and may ultimately assist in providing useful insights into the treatment of depression.

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Table 1

Means, Standard Deviations, and Correlations

	1	2	3	4	5	6	7	8	9
1. Sex	-								
2. CDI total (T1)	-.26*	-							
3. Anhedonia (T1)	-.24*	.68**	-						
4. Non Anhedonia (T1)	-.21	.96**	.48**	-					
5. CDI total (T2)	-.15	.67**	.44**	.68**	-				
6. Anhedonia (T2)	-.15	.46**	.45**	.42**	.73**	-			
7. Non Anhedonia (T2)	-.10	.68**	.40**	.71**	.95**	.52**	-		
8. PEP Baseline (T2)	.08	.22	.13	.23*	.21	.23*	.19	-	
9. PEP Reactivity - Reward (T2)	.03	-.05	-.02	-.05	.07	.22†	.01	.19	-
<i>M</i>	-	4.30	0.82	3.49	4.28	1.00	3.28	101.66	-1.11
<i>SD</i>	-	4.26	1.14	3.42	4.23	1.89	3.53	7.22	3.01
Range	-	0.00	0.00	0.00	0.00	0.00	0.00	79.00	-8.67
Min	-	0.00	0.00	0.00	0.00	0.00	0.00	79.00	-8.67
Max	-	19.00	5.00	15.00	20.00	5.00	17.00	115.67	5.00

Note. Sex coded Female = 0, Males = 1.

† $p < .06$,

* $p < .05$,

** $p < .001$

Table 2
Linear regression models predicting total depressive symptoms from PEP reactivity to reward tasks.

Variable	T1 Depressive Symptoms			T2 Depressive Symptoms		
	<i>b</i>	<i>a</i> (SE)	<i>t</i>	<i>p</i>	<i>b</i>	<i>a</i> (SE)
PEP change reward (T2)	-.04	(0.04)	-.35	.725	.07	(0.04)
Sex	-.25	(0.23)	-2.24	.028	-.15	(0.23)

Note. PEP = Pre-ejection period.

^aStandardized regression coefficients.

Table 3

Regression model predicting anhedonic and nonanhedonic symptom clusters from PEP reactivity to reward task.

	<i>b</i> ^a (<i>SE</i>)	<i>t</i>	<i>p</i>
T1 Anhedonia			
PEP reactivity	-.01 (.04)	-0.11	.916
Sex	-.24 (.23)	-2.09	.041
T1 Nonanhedonia			
PEP reactivity	-.05 (.04)	-0.41	.685
Sex	-.21 (.23)	-1.82	.073
T2 Anhedonia			
PEP reactivity	.23 (.04)	2.00	.049
Sex	-.16 (.22)	-1.39	.169
T2 Nonanhedonia			
PEP reactivity	.02 (.04)	0.14	.886
Sex	-.10 (.23)	-0.90	.374

Note. PEP = Pre-ejection period.

^aStandardized regression coefficients.