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A Preliminary Examination of the Relation between Neural Sensitivity to Reward and History of Alcohol Use Disorder among Adults with Internalizing Psychopathologies

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Abstract

Decreased reward responsiveness, as demonstrated utilizing the event-related potential (ERP) component the reward positivity (RewP), is an established correlate of internalizing psychopathologies (IPs), such as depressive and anxiety disorders. Although IPs are highly comorbid with alcohol use disorders (AUDs) and despite evidence that AUDs are also characterized by aberrant reward processing styles, no studies have examined how AUD history impacts the RewP among adults with IPs. The current preliminary study sought to examine this question in a sample of 65 adults with 1) current IPs (i.e., depression, social anxiety, and/or generalized anxiety, 2) current IPs with a history of an AUD (IP + Past AUD), and 3) no history of a DSM-IV disorder. Participants completed a guessing reward task while electroencephalogram (EEG) was recorded. Results indicated that participants in the IP group exhibited a more attenuated RewP relative to + Past UD and healthy control individuals. Findings from this study highlight the importance of examining diagnostic subgroups among adults with anxiety and depressive disorders, and suggest that a history of AUD may enhance reward reactivity at the neural level in individuals with IPs.

Keywords

reward positivity; alcoholism; internalizing disorders; anxiety; depression; EEG; ERP

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Declaration of Interest

Conflicts of interest: None

1. Introduction

Internalizing psychopathologies (IPs), such as anxiety and depressive disorders, are characterized by deficits in reward sensitivity across multiple units of analysis (for reviews, see [1,2]). Initial responsiveness to reward attainment is one construct identified within the positive valence system of the National Institute of Mental Health's Research Domain Criteria (RDoC) that has been studied extensively in relation to IPs. To capture deficits in this construct at the neural level, researchers have utilized the reward positivity (RewP), an event-related potential (ERP) component assessed via electroencephalography (EEG). The RewP, also often referred to as the feedback negativity (FN) or feedback related negativity (FRN), appears as a relative positivity approximately 250–350 ms following reward feedback; this positivity is thought to indicate response to receiving a reward [3]. Importantly, the RewP exhibits both high retest and internal reliability [4], and research indicative of its validity includes correlation with self-reported reward sensitivity and reward learning behavior [5], positive emotionality [6], and activation in the ventral striatum and medial prefrontal cortex [7,8].

Evidence across studies highlights a blunted RewP among adults and children with depression, and research also supports a blunted RewP as a predictor of future depression onset [3,9,10,11]. Negative relationship between RewP amplitude and severity of anhedonia and other depressive symptoms has also been found [12]. There is less research examining the RewP in anxious individuals and the existing literature is somewhat mixed (see [13,14,15]). Recent data from our laboratory offering a two-factor model for anxiety suggest that conflicting findings may be due to variation among specific anxiety disorders such that anxious misery, but not fear-based anxiety, is associated with a blunted RewP [16].

Taken together, prior studies suggest that a reduced RewP is robustly related to depression and to a lesser extent anxiety. It remains unknown, however, how various subgroups of IPs may influence reward responsiveness. Notably, studies have documented high comorbidity rates between IPs and alcohol use disorders (AUDs; encompassing alcohol abuse and dependence as defined by the DSM-IV). Studies in the general population show that individuals with IPs have a 2- to 3-fold increased risk of alcohol use disorders [17,18,19]. Heavy drinking is also known to increase the risk of onset of depressive episodes and severe anxiety [20]. Despite documented high comorbidity rates, no studies to date have examined how AUD history may impact the RewP among individuals with IPs. Moreover, no studies to date have examined the RewP in an AUD sample. A better understanding of the relation between reward responsiveness and alcohol use patterns among individuals with IPs could likely shed light on potential mechanisms implicated in these comorbid disorders.

Consistent with evidence for a blunted RewP in depression and anxiety, some argue that AUD can be defined in terms of a blunted response to reward. For instance, Blum et al.'s [21] reward deficiency syndrome (RDS) hypothesis posits that individuals who display chronic hyporeactivity to rewards are more likely to engage in risky behaviors, including excessive alcohol use to compensate for insufficient natural reinforcement and underactive dopamine neurotransmission. Both human and non-human research has further shown that exposure to heavy alcohol use results in neuroadaptations in the brain which potentiate

negative affect and blunt reactivity to natural, non-alcohol related rewards [22]. At the same time, there is a parallel literature suggesting that AUD is also characterized by enhanced reward responsiveness. Specifically, several studies show that greater reward sensitivity is associated with greater alcohol intake in nonclinical populations [23,24,25] and with early-onset drinking, a known risk factor for a future AUD [26]. Evidence from functional magnetic resonance imaging (fMRI) studies also indicates that neural regions implicated in reward (i.e., caudate, striatum) are enhanced among individuals at high risk for developing an AUD [27,28,29] and among patients with alcohol dependence [30]. Therefore, AUD has been linked to both blunted and enhanced reward responding, though no study has examined the RewP (specifically) in AUD samples. In addition, despite the high comorbidity patterns between AUD and IPs, no study to date has directly examined whether history of AUD modulates the association between IPs and RewP. This is noteworthy given that the RewP is widely considered as a potential biomarker for IP, especially depression [3].

Therefore, the objective of the current preliminary study was to examine differences in the RewP during reward processing among three groups: 1) adults with current IPs (i.e., depression, social anxiety disorder, and/or generalized anxiety); 2) adults with current IPs and a history of an AUD (IP + Past AUD); and 3) adults with no history of any psychological disorder. Anxiety disorders were collapsed across social anxiety and generalized anxiety due to high comorbidity, and therefore we were unable to test the aforementioned two-factor model of anxiety [16]. Consistent with previous studies [3,9,10,11,12,13,14,16], we expected that adults with current IPs would exhibit an attenuated RewP relative to healthy controls (HCs). Based on previous behavioral and fMRI studies involving current drinking behavior/diagnoses [21,22,23,24,25,26,27,28,29,30], we predicted that IP + Past AUD adults would exhibit an altered RewP—either enhanced or blunted—relative to both HCs and individuals with IP but no history of AUD.

2. Method

Participants were part of a larger study examining response to cognitive behavioral therapy (CBT). As alcohol or substance abuse or dependence within six months was exclusionary for the study, this paper represents a secondary exploratory analysis. The study was approved by the University of Illinois at Chicago (UIC) Institutional Review Board, and informed consent was obtained from all participants. Participants were interviewed by Master's- or Doctorate-level clinicians using the Structured Clinical Interview for DSM-IV (SCID-IV) [31] to assess current and lifetime diagnoses of Axis I disorders. At baseline, 90 adults completed the Guessing Reward Task. Of these 90 adults, 65 had usable EEG data (defined as at least 12 trials in each condition for the EEG task). The 25 participants excluded for poor quality EEG data did not differ statistically from included participants on demographic or relevant study variables (i.e., age, sex, anxiety/depressive symptoms, diagnoses) (lowest $p = .09$). Of these 65 adults, 24 had no history of any DSM-IV psychological disorder, and 41 met criteria for a current anxiety or depressive disorder and were not taking any psychotropic medications. Out of these 41 individuals, 15 also had a past history of an AUD (as assessed by the SCID; see Table 1 for demographics and clinical characteristics of sample). The average length of AUD remission for the IP + Past AUD group was 1.95 years. After the screening evaluation, a consensus panel of at least three study staff or trained

clinicians determined participant eligibility, whether or not there were co-occurring disorders, and which was the principal disorder warranting treatment. Additionally, all participants passed a drug test before completing the EEG portion of the study.

Participants completed an adaptation of a guessing reward task based on work by Forbes et al. [32] (Figure 1). The task comprised 60 trials (15 win, 15 loss, 15 no-win, and 15 no-loss), each consisting of a decision, anticipation, and outcome period, separated by an intertrial interval ranging between four seconds and seven seconds. During the decision period, participants were presented with a question mark (four seconds) and pressed a button in order to guess whether a computer-selected number was greater than or less than five. For the anticipation phase, participants viewed a circle with the numbers one through nine and a yellow arrow indicating the range of the actual number (i.e., whether the participant was correct or incorrect; presented for six seconds). Participants were informed that a correct response indicates the possibility of winning one dollar or breaking even, whereas an incorrect response indicates the possibility of losing 50 cents or breaking even. During the outcome period, participants were presented with the “actual” number for 500 ms and received feedback for 500 ms in the form of a happy face for winning, sad face for losses, and neutral face for breaking even (i.e., no-win, no-loss). Participants saw their total earnings every 20 trials (\$2.50, \$4.50, \$7.50). Participants were told that they would receive the total amount of their winnings, but in fact, each participant received ten dollars.

Continuous EEG was recorded using a 34-channel cap (32-channel setup based on 10 out of 20 systems with the addition of FCz and Iz) and the BioSemi system (BioSemi, Amsterdam, the Netherlands). Electrodes were placed on the left and right mastoids, and the electroculogram was recorded from four facial electrodes. The data were digitized at 24-bit resolution with a Least Significant Bit value of 31.25 nV and a sampling rate of 1024 Hz. The voltage from each active electrode was referenced online with respect to a common mode sense active electrode.

Data were processed offline using Brain Vision Analyzer software (Brain Products, Gilching, Germany) and converted to a linked mastoid reference, filtered with high- and low-pass filters of 0.1 Hz and 30 Hz, respectively. Continuous EEG data were segmented beginning 100 ms before stimulus onset and continuing for the 500 ms after onset. Eyeblinks were corrected using the method by Gratton et al. [33]. The mean number of artifact free-trials across conditions was 14 ($SD = 1.58$). Data were baseline corrected using the 100 ms interval prior to feedback and averaged across trials for each condition (win, no-win, loss, and no-loss). The RewP was scored as the mean amplitude 230–300 ms following reward feedback at a pooling of frontal electrode sites (AF3, AF4, Fz), where the responses were maximal; analyses focused on the win minus no-win (breaking even) difference score (RewP) with more positive values for the difference score indicating greater reactivity to reward.

2.1 Analytical plan

We first examined whether the three groups (HC, IP, IP + Past AUD) differed in regard to demographic and clinical characteristics utilizing a series of one-way ANOVA tests. To examine whether the two groups (IP, IP + Past AUD) differed in regard to primary and

comorbid diagnoses, a series of chi-square tests were conducted. To examine whether the three groups differed in neural sensitivity to reward, a separate one-way ANOVA was conducted with the RewP serving as the dependent variable and diagnostic group (HC, IP, IP + Past AUD) serving as the independent variable. Post-hoc Bonferroni comparisons were conducted for significant effects.

3. Results

Demographic and clinical characteristics of the sample separated by diagnostic group are provided in Table 1. As expected, adults with IP and IP + Past AUD exhibited greater clinician-rated and self-reported depressive and anxiety symptoms, relative to the HC adults. In addition, the IP and IP + Past AUD groups did not differ in regard to primary or comorbid anxiety, depressive, or substance use disorder diagnoses, nor did the two groups differ in depression and anxiety symptoms. The three groups did not differ in regard to age, sex, race, or ethnicity.

Next, we examined group differences in the RewP. Across all participants, the response to wins was significantly more positive compared to the response to breaking even, $t(64) = 2.54, p < .05$ (win: $M = 5.11 \mu V, SD = 3.93 \mu V$; breaking even: $M = 3.85 \mu V, SD = 4.92 \mu V$), providing justification for utilizing the difference score (RewP). Results revealed a main effect of group, $F(2,65) = 6.55, p < .01, \eta^2 = .17$. Pairwise comparisons revealed that the IP group ($M = -0.41, SE = 0.75$) exhibited a more attenuated RewP response, relative to the HC ($M = 1.62, SE = 0.75, p = .05$) and IP + Past AUD groups ($M = 3.83, SE = .94, p = .001$). The difference in the RewP between the IP + Past AUD and HC groups was a nonsignificant trend ($p = .07$). The waveforms and scalp topographies depicting these findings are presented in Figure 2.

4. Discussion

The current preliminary study sought to examine the relationship between reward responsiveness and AUD history utilizing the RewP during reward processing in a sample of HCs and individuals with current IPs. Consistent with previous studies [3,9,11,12,13,16], adults with a current anxiety or depressive disorder exhibited an attenuated RewP, relative to HCs. Importantly, we also found evidence for IP adults with a past AUD exhibiting an enhanced RewP, but only compared with IP adults with no AUD history. These preliminary findings suggest that within individuals with IPs, history of AUD is associated with enhanced reward responsiveness at the neural level. Although prior studies have consistently documented an attenuated RewP response among youth and adults with depression [3,9,11,12,13,16], the current findings suggest that AUD history influences the directionality of this relationship. As such, these results underscore the general importance of examining diagnostic subgroups and patterns of comorbidity among adults with depressive and anxiety disorders when exploring reward sensitivity patterns.

As previously discussed, AUD has been linked to both blunted [21,22] and enhanced [23,24,25,26,27,28,29,30] reward responding. The current pattern of findings is partially consistent with prior research on AUD and reward reactivity suggesting AUD is associated

with enhanced reward reactivity, and, consequently, in contrast with the reward deficiency syndrome (RDS) hypothesis. Other studies have shown that individuals at-risk and with current AUDs report greater reward pleasure and responsiveness [34] and display greater striatal responses to natural rewards, relative to individuals without AUD [27,29]. Our results show a similar directional pattern such that individuals with IP + Past AUD exhibited an enhanced RewP, an ERP component generated in the striatum, relative to individuals with IP only. Notably, the difference in magnitude of the RewP between the HC and IP + Past AUD group was a non-significant trend; therefore, it will be important for future studies to replicate this effect of enhanced reward responding with a larger sample to determine if this is an electrophysiological marker that characterizes AUD, or is simply a marker that differentiates IP from IP + AUD patients.

This hyperactive reward responsivity observed among individuals with current IPs in remission from AUD may be a persistent or stable risk factor for AUD that may influence vulnerability to relapse among individuals with IPs. Specifically, it is well known that individuals with a past history of AUD are at elevated risk for developing problematic drinking behaviors in the future [35]. One possibility is that individuals who are sensitive to rewards may find alcohol to be positively reinforcing, which drives excessive consumption. This possibility is supported by research showing that heightened sensitivity to reward at the self-report level is related to cue-elicited urge to drink among drinkers [36]. In addition, a separate study found an association between drinking for enhancement reasons and attentional bias to reward, and these findings were related to frequent alcohol use as well [37]. Future longitudinal studies are needed to determine whether this enhanced reward responsivity at the neural level may be one mechanism placing individuals with IPs and a past AUD at future risk for developing problematic drinking behaviors over time.

An alternative explanation for the current findings is that the enhanced reactivity to reward in the IP + Past AUD sample may be an acquired consequence of prolonged alcohol exposure. Specifically, there is some evidence to suggest that reward reactivity is diminished in currently active AUD, including reactivity to natural rewards such as money, food, and sex. For instance, one study showed that alcohol significantly activates the nucleus accumbens among social drinkers but not heavy drinkers [38]. Due to the design of the present study, we cannot rule out the possibility that AUD in remission enhances the RewP via a rebound effect after a period of heavy drinking. Future, longitudinal studies are needed to directly test this possibility.

There were strengths of the current study including being the first to suggest that examining diagnostic subgroups within IPs may influence the direction of reward responding at the neural level. The use of ERPs to examine this question is also a strength of the current study given the established psychometric properties of the RewP [4,5,6,7,8,39]. However, there were limitations to the current study that should be addressed. First, the current study was preliminary in nature and as a result, the sample size was relatively small and included mostly young adults. The small sample precluded our ability to explore differences between anxiety and depressive disorders, examine potential moderators of interest (i.e., sex, age), and perhaps detect significant effects. Similarly, while the IP and IP + AUD groups did not significantly differ in depression rates, percentages of primary MDD diagnoses were slightly

higher in the IP (53.8%) versus IP + AUD (33.3%) group. Thus, future studies with larger sample sizes and more balanced diagnostic profiles are needed to test moderators of interest and replicate the observed group differences. Finally, the current study did not include a group of participants with a history of AUD but no IP history, nor explore family history of AUD among participants. It will be important for future studies to explore whether adults with AUD (but no IP) and/or a positive family history of AUD also exhibit an enhanced RewP, or whether this response is only observed among adults with AUD and current internalizing psychopathology.

Given that a blunted RewP has been more consistently observed in depression, future studies are needed to determine if these findings are specific for anxiety and/or depressive disorders. In samples with less comorbidity among anxiety disorders, the previously mentioned two-factor model of anxiety should be tested [16]. Next, the current study did not include a measure of degree of current alcohol use. Thus, it will be important for future research to explore if there are differences in the RewP among individuals with varying severity of alcohol use disorder, as well as individuals with a history of alcohol abuse versus dependence. Finally, the current study focused on IP adults with a past AUD, which confirmed the importance of exploring diagnostic subgroups when exploring reward responsiveness. However, it will be important for future studies to examine whether the current findings are replicated in adults with a current AUD, including those with and without comorbid IP.

In summary, the current preliminary study suggests that AUD history may enhance reward responsiveness at the neural level among individuals with IPs. Although prior research has documented an attenuated RewP as a biological marker of internalizing disorders, the current findings suggest that AUD history may impact the directionality of this reward response. Together, findings emphasize the general importance of examining diagnostic subgroups among adults with depressive and anxiety disorders when examining risk factors.

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Highlights

- Findings of an attenuated reward positivity in internalizing disorders are supported.
- IP adults exhibited a blunted reward positivity, relative to healthy controls (HCs).
- The reward positivity was increased in IP + Past AUD adults.
- Subgroups of internalizing disorders may exhibit differential reward reactivity.

Figure 1.

Design of the guessing reward task. The task comprised 60 trials (15 win, 15 loss, 15 no-win, and 15 no-loss), each consisting of a decision period, anticipation period, and outcome period, separated by an intertrial interval ranging between 4 seconds and 7 seconds.

Figure 2.

On the top, topographic maps of activity (win minus breaking even) for three groups: healthy controls (HC), internalizing psychopathology (IP), and internalizing psychopathology with a past history of alcohol use disorder (IP + Past AUD). On the bottom, response-locked ERP waveforms (pooling of AF3, AF4, Fz) for win and breaking even trials, as well as the difference waves (reward positivity; RewP) for the three groups. The RewP was scored as the mean amplitude 230–300 ms following reward feedback, as indicated by the grey box outlining the focal point of the waveforms. HC = healthy controls; IP = internalizing psychopathology diagnosis; IP + Past AUD = current internalizing psychopathology diagnosis with history of an alcohol use disorder; RewP = reward positivity.

Table 1.

Demographics and clinical characteristics of the sample by group.

	HC (n=24)	IP (n=26)	IP + Past AUD (n=15)
<u>Demographics</u>			
Age (years)	23.22 (4.54) _a	21.88 (2.37) _a	24.13 (3.66) _a
Sex (% female)	62.5% _a	73.1% _a	80.0% _a
Race/Ethnicity			
Caucasian	37.5% _a	69.2% _a	66.7% _a
African American	12.5% _a	3.8% _a	6.7% _a
Hispanic	29.2% _a	46.2% _a	40.0% _a
Asian	29.2% _a	19.2% _a	6.7% _a
Other/Biracial	16.7% _a	7.7% _a	20.0% _a
<u>Clinical Characteristics</u>			
HAM-D Symptoms	.71 (1.23) _a	13.15 (6.22) _b	12.4 (5.97) _b
HAM-A Symptoms	1.0 (1.50) _a	18.46 (8.62) _b	18.27 (7.49) _b
DASS Depression	.17 (.48) _a	10.31 (4.77) _b	10.47 (4.96) _b
DASS Anxiety	.21 (.51) _a	7.85 (5.13) _b	6.33 (4.91) _b
<u>Current Primary Diagnosis</u>			
GAD	-	11.5% _a	13.3% _a
MDD	-	53.8% _a	33.3% _a
SAD	-	34.6% _a	53.3% _a
<u>Current Comorbid Diagnosis</u>			
GAD	-	7.7% _a	20.0% _a
MDD	-	38.5% _a	26.7% _a
SAD	-	42.3% _a	26.7% _a
Panic Disorder	-	15.4% _a	26.7% _a
PTSD	-	15.4% _a	26.7% _a
Dysthymia	-	7.7% _a	20.0% _a
<u>Past Substance Disorder Diagnosis</u>			
Past Substance Abuse		11.5% _a	6.7% _a
Past Substance Dependence		7.7% _a	13.3% _a

Note: DASS = depression and anxiety stress scales; GAD = generalized anxiety disorder; HAM-Hamilton anxiety rating scale; HAM-D = Hamilton depression rating scale; HC = healthy controls; IP = internalizing psychopathology diagnosis; IP + Past AUD = current internalizing psychopathology diagnosis with history of an alcohol use disorder; MDD = major depressive disorder; PTSD = post-traumatic stress disorder; SAD = social anxiety disorder; Groups with the same subscript do not significantly differ from one another.