

# **An event-related potential study of decision making and feedback utilization in female college students who binge drink**

Eun-Chan Na, Kyoung-Mi Jang, Myung-Sun Kim\*

Department of Psychology, Sungshin Women's University, Seoul, Republic of Korea

\* [kimms@sungshin.ac.kr](mailto:kimms@sungshin.ac.kr) (MSK)

# Abstract

This study investigated the ability to use feedback for decision making in female college students who binge drink (BD) using the Iowa Gambling Task (IGT) and event-related potentials (ERPs). Twenty-seven binge drinkers and 23 non-binge drinkers (non-BD) were identified based on scores on the Korean version of the Alcohol Use Disorder Test and the Alcohol Use Questionnaire. The IGT consists of four cards, including two cards that result in a net loss, with large immediate gains but greater losses in the long term, and two cards that result in a net gain, with small immediate gains but reduced losses in the long term. Participants were required to choose one card at a time to maximize profit until the end of the task while avoiding losses. The BD group showed a significantly lower total net score than the non-BD group, indicating that the BD group chose more disadvantageous cards. The BD group showed significantly smaller  $\Delta$ FRN amplitudes (difference in amplitudes of feedback-related negativity [FRN] between gain and loss feedback) except in P3. Additionally,  $\Delta$ FRN amplitudes in the fronto-central area were positively correlated with the total net score and net scores for sectors 4 and 5. Thus, total net scores and later performance on the IGT increased as  $\Delta$ FRN amplitudes from the fronto-central area increased. FRN is known to reflect early feedback evaluation employing a bottom-up mechanism, whereas P3 is known to reflect late feedback processing and allocation of attentional resources using a top-down mechanism. These results indicate that college students who binge drink have deficits in early evaluation of positive or negative feedback and that this deficit may be related to decision making deficits.

Keyword: Binge drinking, Decision making, Feedback-related negativity, Feedback utilization, Event-related potentials, P3

# Introduction

Binge drinking (BD) is defined as a repeated pattern of excessive alcohol consumption and abstinence over a short period of time [1-3]. BD is most prevalent among young adults, especially college students [3,4,5], and is associated with various problems including assault, drunk driving, unguided or unsafe sexual behavior, and academic underachievement [3,6-8]. Additionally, binge drinkers exhibit similar structural and functional brain abnormalities and neuropsychological deficits to patients with alcohol use disorder (AUD) [1,9-13], and BD predicts the development of AUD in the future [11,14-16].

Patients with AUD cannot stop drinking alcohol even though they suffer from its negative consequences [17,18]. Such behaviors reflect inefficient decision making among patients with AUD, as they continue to seek immediate rewards and ignore future consequences [19,20]. In other words, they not only underestimate the negative consequences of alcohol consumption [21] but also emphasize immediate rewards over long-term consequences [22,23]. Decision making deficits have been observed in patients with AUD [19,24-28] and in binge drinkers [29-33].

Decision making is defined as a process of forming a preference for an option, making a choice based on the preference, executing the choice, and evaluating the consequences of the choice [34]. Decision making is a complex process including both cognitive and non-cognitive processes (i.e., emotions) [35], and various brain areas, such as the orbitofrontal, ventromedial prefrontal, anterior cingulate cortices, and amygdala, are involved in decision making [36-40].

The Iowa Gambling Task (IGT) is widely used to evaluate decision making ability [41,42]. Participants are asked to choose one of four cards on every trial to maximize profit while avoiding loss. The chosen card results in gains on every trial, but also results in intermittent losses. The cards differ in feedback magnitude and probability. Two cards (A and B) result in large immediate gains but greater losses, causing a net loss (disadvantageous cards), whereas the other two cards (C and D) lead

to small immediate gains and smaller losses, resulting in a net gain (advantageous cards). Participants must evaluate feedback such as valence (gain or loss), magnitude (large or small), and the probability of encountering losses to learn the contingency between the card and its consequences [43,44].

Studies investigating decision making ability in patients with AUD using the IGT found that patients with AUD performed poorly compared to normal controls, choosing significantly more disadvantageous cards and significantly fewer advantageous cards compared with the controls [19, 25,26,28,45]. Additionally, positive correlations were observed between IGT performance and grey matter volume in the dorsal and ventromedial prefrontal cortices, which are crucial for decision making [46]. Poor IGT performance has also been observed in individuals with BD [29,30,33,47]. For example, adolescents [47] and college students with BD [33] performed significantly worse on the IGT than did non-BD group.

Feedback utilization, a process of identifying whether an action induces positive or negative consequences and evaluating those consequences, is crucial to making efficient decisions [48]. Considerable improvement in our understanding of the neurological basis of feedback utilization has revealed that the orbitofrontal, ventromedial prefrontal, and anterior cingulate cortices as well as the ventral striatum are involved in feedback utilization [49-53]. The ventral striatum is involved in prediction errors, i.e., how actual feedback differs from personal expectations, whereas the orbitofrontal cortex is involved in evaluating feedback based on prediction errors [54-57]. Additionally, the anterior cingulate cortex evaluates rewards in situations where contingencies are uncertain and then relays the evaluation of the reward to motor areas for response execution [37].

Studies using event-related potentials (ERPs) suggest two components, feedback-related negativity and P3, as the electrophysiological indices of feedback utilization [48,58]. Gehring and Willoughby [58] used a simple gambling task to observe a negative peak approximately 265 ms post feedback whose amplitude was larger in response to negative than to positive feedback. This peak is known as feedback-related negativity (FRN) or outcome-related negativity [59]. FRN is sensitive to

feedback valence (gain or loss) [60] and is associated with activation of the midbrain dopaminergic system [61]. Additionally, reinforcement-learning theory suggests that FRN reflects prediction errors, i.e., the difference between actual feedback and personal expectation [48,62,63]

P3, another ERP component related to feedback utilization, is a positive peak observed in central-parietal areas at 275-700 ms post feedback [48,59]. P3 is known to be sensitive not only to feedback valence but also to feedback magnitude and probability [60,63-66]. It has been suggested that P3 reflects activation of the locus coeruleus-norepinephrine system and processing of task-relevant information to maximize decision making efficiency [67]. In other words, P3 reflects, unlike FRN, a top-down mechanism that processes and evaluates feedback-related information in detail [48,63].

Alcohol consumption affects feedback utilization. A study that used a gambling task and measured ERPs found that the alcohol consumption group exhibited significantly lower FRN amplitudes in response to both gain and loss feedback, especially to loss feedback, than did a placebo group, indicating that alcohol consumption affects feedback utilization [68]. Deficits in feedback utilization are also observed in patients with AUD. For example, Fein and Chang [69] using the Balloon Analogue Risk Task, observed that patients with AUD and a family history of AUD exhibited significantly smaller FRN amplitudes than did those without a family history. Kamarajan et al. [70] used a gambling task and reported that patients with AUD exhibited lower P3 amplitudes in response to both gain and loss feedback and smaller FRN amplitudes to loss feedback than did normal controls. Additionally, they observed increased activation in primary sensory and motor areas during the FRN time window and decreased activation in the cingulate gyrus during the P3 window in patients with AUD relative to normal controls. These results indicate that the sensory and motor areas of patients with AUD are hyper-excited during early feedback evaluation, and areas involved in feedback evaluation are hypo-activated compared to normal controls [70].

To our knowledge, only one study has investigated feedback utilization deficits in binge

drinkers using ERPs. That study, which used the IGT, found that the BD group tended to exhibit smaller FRN amplitudes ( $p = .06$ ) than the non-BD group [71]. However, that study used the original computerized IGT [41,42], which had two limitations: First, the original IGT consisted of 100 trials, which is not suitable for an ERP study where a sufficient number of trials is needed [72]. Second, the original IGT displays gains in every trial and subsequently displays losses according to each card's probability. When multiple stimuli are displayed in succession, the ERPs to loss feedback might be contaminated by previous gain feedback.

The present study investigated feedback utilization ability during decision making in BD female college students using the IGT and ERP. Specifically, this study examined whether decision making deficits in BD female students are related to feedback utilization deficits and, if so, how they are reflected in feedback-related ERP components, FRN and P3. Based on previous findings, we hypothesized that the BD group would perform significantly worse than the non-BD group on the IGT; that the BD group would show significantly smaller FRN and P3 amplitudes than the non-BD group; and that IGT performance and feedback-related ERPs would be positively correlated. As gender differences are observed in BD [73-75], decision making [76], and ERP amplitudes [77], only female college students were included in this study.

## Materials and methods

### Participants

The details of the participant screening procedures have been described in previous studies by our research group [33,78]. The Korean version of the Alcohol Use Disorder Identification Test (AUDIT-K) [79,80], Alcohol Use Questionnaire (AUQ) [81], and a questionnaire inquiring about binge drinking episodes in the last 2 weeks were administered to 435 female college students. The

BD and non-BD groups were defined based on 1) alcohol-related problems and drinking habits, 2) the number of BD episodes, and 3) drinking speed. The BD group included those who 1) scored at least 12 but less than 26 on the AUDIT-K, 2) had consumed four or more glasses at one sitting in the last 2 weeks, and 3) drank two or more glasses per hour. Although the World Health Organization (WHO) recommends using a score  $> 8$  as the cutoff point for problem drinking [79], the cutoff score of 12 was applied because a cutoff point of 8 includes those who do not have apparent drinking problems but may display problem drinking in the future [82,83]. In contrast, those who received scores  $> 26$  on the AUDIT-K were also excluded, as AUD was suspected. The non-BD group included those who 1) scored less than 8 on the AUDIT-K, 2) had not drunk four or more glasses in one sitting in the last 2 weeks, and 3) drank 1 glass or less per hour.

The Structured Clinical Interview of the Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition (SCID-NP) [84] was administered to ensure that no participants had a psychiatric disorder. Additionally, the Self-rating Depression Scale (SDS) [85], the State-Trait Anxiety Inventory (STAI) [86], and Barratt Impulsivity Scale (BIS) [87] were administered to evaluate depression, anxiety, and impulsivity, respectively. To control for the influence of alcohol-related genes and family history, the Korean version of the Children of Alcoholics Screening Test (CAST-K) [88,89] was administered, and those who scored 6 or more were excluded. Last, those who were left-handed or ambidextrous were also excluded to control for the effect of brain lateralization.

In the end, 50 students participated in this study (27 in the BD group and 23 in the non-BD group). This study was approved by Sungshin Women's University Institutional Review Board (SSWUIRB 2017-040). The participants provided written informed consent after receiving a description of the study, and they were paid for their participation.

## **The Korean version of the Alcohol Use Disorder Identification**

## Test (AUDIT-K)

The AUDIT [79], a self-administered questionnaire designed to measure the presence of AUD and drinking problems, consists of 10 items. The total score ranges from 0 to 40. Three items inquire about frequency and quantity of alcohol consumption, three about symptoms related to alcohol dependence, and four about psychosocial problems related to alcohol consumption. The Korean version was administered in this study [80].

## Alcohol Use Questionnaire (AUQ)

The AUQ [81] is a self-administered questionnaire measuring drinking patterns. Items 10, 11, and 12 evaluate drinking speed, frequency of being drunk within the last 6 months, and the rate of being drunk when consuming alcohol, respectively. These three items were used to calculate a BD score [90]. The binge score was calculated using the following equation:

$$\text{AUQ Binge score} = [\text{Item 10} \times 4 + \text{Item 11} + \text{Item 12} \times 0.2]$$

## The Iowa Gambling Task (IGT)

This study employed a modified version of the original computerized IGT [41] to make the task suitable for measuring ERPs (Fig 1A). Four cards were displayed on a computer monitor, and participants were asked to maximize profits until the end of the game by choosing a card during each trial. Gain or loss feedback was displayed after each choice, with gain feedback consisting of a green smiling emoticon with points earned and loss feedback consisting of a red crying emoticon with the points lost (Fig 1B).



**Fig 1. The modified IGT.** A) A fixation point will be displayed for 1,000ms then four cards will be displayed till the participants make their choice. At 700ms after a card is chosen, feedbacks will be displayed for 1,000ms. B) The feedback stimuli consists of gain conditions and loss conditions. In gain conditions, green smiling emoticon and the earned points will be displayed whereas red crying emoticon and the lost points will be displayed in loss conditions.

The magnitude and probability of gain and loss for each card were set as for the original computerized IGT [41]. The cards consisted of two disadvantageous cards (A and B), which provided large gains and larger losses, resulting in a net loss, and two advantageous cards (C and D), which provided small gains but smaller losses, resulting in a net gain. Cards A and C each had a 50% chance of causing losses, whereas cards B and D had a 10% chance of causing losses.

The task consisted of three blocks; the locations of the cards were changed at the beginning of each block to keep participants motivated. Each block comprised 100 trials; a total of 320 trials, including 20 practice trials, were administered. Decision making ability was measured by the net score, which was calculated by subtracting the frequency of choosing the disadvantageous cards (A and B) from the frequency of choosing the advantageous ones (C and D)

E-Prime software (version 2.0; Psychological Software Tools, Inc., Sharpsburg, PA, USA) was used to administer the modified IGT. A fixation point (+) was displayed for 1,000 ms, and the cards were then displayed until the participants made their choice by pressing a button. The feedback, either a gain or loss, was displayed for 1,000 ms at 700ms after a card was chosen.

## Electrophysiological recording procedure

Electroencephalography (EEG) was measured using a 64-channel Geodesic sensor net connected to a 64-channel, high-input impedance amplifier (Net Amp 300; Electrical Geodesics,

Eugene, OR, USA) in a shielded and soundproofed room. All electrodes were referenced to Cz, and impedance was maintained at 50 K $\Omega$  or less [91]. EEG activity was recorded continuously using a 0.3 - 100 Hz bandpass filter at a sampling rate of 500 Hz. The recorded EEG data were digitally filtered using a 0.3 - 30 Hz bandpass and re-referenced to the average reference. The continuous EEG was then segmented into 800 ms epochs (from 100 ms pre- to 700 ms post-feedback). Additionally, epochs contaminated by artifacts such as eye blinks were removed based on the threshold of a peak-to-peak amplitude of  $\pm 70$   $\mu$ V from the eye channels. The remaining data were averaged according to feedback valence, i.e., gain and loss feedback.

## Statistical analysis

Demographic variables were analyzed with independent *t*-tests. The total net scores on the modified IGT were analyzed with independent *t*-tests. Additionally, each block was subdivided into five sectors, and scores for each sector were averaged across the three blocks to calculate sector net scores to measure performance improvement across trials. The sector net scores were analyzed with mixed-design analysis of variance (ANOVA), where group (BD or non-BD) was a between-subjects factor, and sector (1 - 5) was a within-subject factor.

ERP components and time windows were determined based on grand averaged ERPs and individual ERP waveforms. FRN was defined as the most negative peak observed at 200 - 275 ms after feedback-onset, and P3 was defined as the most positive peak followed by FRN, i.e., observed 275 - 600 ms after feedback. Because the FRN and P3 time windows overlapped and because the FRN is a negative and P3 is a positive peak, it is possible that latent components representing FRN and P3 independently might be distorted on the ERP waveforms due to the overlapping windows where the amplitudes and latencies do not clearly represent the differences by feedback valence [92]. To overcome this problem, it is necessary to isolate ERP components; difference waves have been

recommended for this purpose [92]. Therefore,  $\Delta$ FRN (FRN effect) and  $\Delta$ P3 (P3 effect) were defined as the amplitude difference between gain and loss feedback [64,66,93-96].

Amplitudes and latencies of each component were analyzed by mixed ANOVA. Electrode site (FC3, FCz, FC4, C3, Cz, C4, P3, Pz, and P4) and valence (gain or loss) were within-subject factors, and group was a between-subjects factor. The electrode sites for  $\Delta$ FRN and  $\Delta$ P3 were a within-subject factor, and group was a between-subjects factor. Greenhouse-Geisser corrections were used in cases of violation of sphericity, and corrected  $p$ -values are reported when appropriate. The mean numbers of trials included in the FRN/P3 analysis for the BD and non-BD groups were 105.57 (gain = 161.89, loss = 51.26) and 111.33 (gain = 17.48, loss = 52.17), respectively. The two groups did not differ in terms of trials for averaging FRN/P3 in the gain feedback ( $F[1,48] = .62, p = .44$ ), the loss feedback ( $F[1,48] = .04, p = .85$ ) or both feedbacks ( $F[1,48] = .57, p = .46$ ). The relationships of the  $\Delta$ FRN and  $\Delta$ P3 amplitudes with performance on the IGT, i.e., total net scores and sector net scores, were analyzed using Pearson's correlation coefficient analysis. A  $p$ -value  $< .05$  was considered significant.

## Results

### Demographic characteristics

The BD and non-BD groups did not differ in terms of age ( $t[48] = -1.08, p = .29$ ), educational level ( $t[48] = -1.07, p = .29$ ), SDS ( $t[48] = .80, p = .43$ ), or trait anxiety on the STAI ( $t[48] = 1.05, p = .30$ ). However, the BD group exhibited significantly higher state anxiety on the STAI ( $t[48] = 5.49, p < .001$ ), BIS ( $t[48] = 6.92, p < .001$ ), AUDIT-K total score ( $t[48] = 16.81, p < .001$ ), drinking speed ( $t[48] = 12.56, p < .001$ ), frequency of being drunk within the last 6 months ( $t[48] = 5.63, p < .001$ ), percentage of being drunk when consuming alcohol ( $t[48] = 3.73, p < .01$ ),

and AUQ binge score ( $t[48] = 9.94, p < .001$ ) compared to the non-BD group. The demographic characteristics of the BD and non-BD groups are presented in Table 1.

**Table 1. Demographic characteristics of the non-BD and BD groups.**

	Non-BD ( <i>n</i> = 23)	BD ( <i>n</i> = 27)	<i>t</i>
	Mean (SD)	Mean(SD)	
Age (years)	22.04(1.92)	21.44(1.99)	-1.08
Education (years)	15.09(1.08)	14.74(1.20)	-1.07
SDS	39.61(5.39)	41.15(7.83)	.80***
STAI state	38.57(8.10)	56.93(14.16)	5.49**
STAI trait	38.70(7.56)	41.26(9.37)	1.05
BIS	63.48(10.95)	83.26(9.29)	6.92**
AUDIT-K	2.39(1.80)	17.37(4.20)	16.81***
Speed of drinking (drinks/hour)	.65(.57)	4.22(1.34)	12.56***
Times drunk in the last 6 months	.13(.34)	5.07(4.55)	5.63***
Percentage of times became drunk when drinking (%)	11.87(23.37)	39.44(28.83)	3.73**
AUQ binge drinking score	5.11(5.17)	29.85(11.66)	9.94***

**Notes:** \*\* $p < .01$  \*\*\* $p < .001$ .

**Abbreviations:** SDS, Self-Rating Depression Scale; STAI, Spielberger's State-Trait Anxiety Inventory; BIS, Barratt Impulsivity Scale; AUDIT-K, The Korean version of Alcohol Use Disorder Identify Test; AUQ, Alcohol Use Questionnaire.

As significant differences in state anxiety and impulsivity were detected, mixed analysis of covariance was performed with state anxiety and impulsivity as covariates to control their effect on the IGT and ERP components. However, the analysis revealed that state anxiety as a covariate was not significantly associated with the IGT ( $p = .086$ ), FRN ( $p = .565$ ), or P3 ( $p = .634$ ) and that impulsivity as a covariate was not significantly associated with the IGT ( $p = .464$ ), FRN ( $p = .295$ ), or P3 ( $p = .631$ ).

## The modified Iowa Gambling Task (IGT)

The BD group exhibited a significantly lower total net score than the non-BD group ( $t[48] = -2.61, p < .05$ ). In terms of sector net scores, a main effect of sector was observed ( $F[4,192] = 2.45, p < .05$ ). A further post hoc analysis revealed a trend toward a lower net score for sector 2 than for sector 4 ( $p = .09$ ). Additionally, a main effect of group was observed ( $F[1,48] = 7.28, p < .05$ ), with the BD group exhibiting significantly lower sector net scores than the non-BD group. However, the sector  $\times$  group interaction was not significant ( $F[4,192] = 1.226, p = .30$ ). Mean total and sector net scores of the BD and non-BD groups are presented in Table 2 and Fig 2.

**Table 2. Performance of the modified IGT in the non-BD and BD groups.**

	Non-BD ( $n=23$ )	BD ( $n=27$ )
	Mean (SD)	Mean (SD)
Sector 1	-.03 (4.16)	-1.73 (3.04)
Sector 2	.72 (5.73)	-3.04 (4.14)
Sector 3	.93 (5.53)	-1.63 (3.49)
Sector 4	2.23 (5.75)	-1.41 (4.35)
Sector 5	1.25 (5.14)	-1.93 (4.08)
Total	5.10 (23.43)	-9.73 (15.11)

**Fig 2. Performance of the modified IGT.** Sector net scores (left) and total net scores (right) of the modified IGT in non-binge drinking and binge drinking groups.

## Electrophysiological measures

The grand-averaged ERPs elicited by gain and loss feedback at fronto-central (FCz), central (Cz), and parietal midlines (Pz) for the BD and non-BD groups are displayed in Fig 3. The BD and non-BD groups exhibited the largest FRN and P3 amplitudes at Cz. The topographical distribution of FRN and P3 measured at all electrodes when the largest FRN and P3 amplitudes were observed are displayed in Figs 4 and 5, respectively.

**Fig 3. The grand-averaged ERPs.** The grand-averaged ERPs elicited by gain and loss feedback at FCz, Cz and Pz for non-binge drinking and binge drinking groups.

**Fig 4. Topographical distribution of FRN.** The topographical distribution of FRN measured at all electrodes when the maximum FRN amplitudes were observed.

**Fig 5. Topographical distribution of P3.** The topographical distribution of P3 measured at all electrodes when the maximum P3 amplitudes were observed.

Main effects of valence ( $F[1,48] = 62.17, p < .001$ ) and electrode site ( $F[8,384] = 18.52, p < .001$ ) were observed in terms of FRN amplitudes. FRN amplitudes in response to loss feedback were significantly larger than those in response to gain feedback, and the largest and smallest FRN amplitudes were observed at Cz and FC4, respectively. Additionally, a valence  $\times$  group interaction was observed ( $F[1,48] = 8.06, p < .01$ ). A simple effect analysis revealed that while both groups exhibited larger FRN in response to loss than to gain feedback, the difference in the FRN amplitudes

between the gain and loss feedback was larger in the non-BD group (mean difference = 2.32,  $p < .001$ ) than in the BD group (mean difference: 1.09,  $p < .01$ ). In addition, an electrode site  $\times$  valence interaction was observed ( $F[8,384] = 12.32, p < .001$ ) such that FRN amplitudes in response to the loss feedback were larger than those to the gain feedback in all electrodes except FC3. The main effect of group was not significant ( $F[1,48] = .08, p = .78$ ). The mean FRN amplitudes of the BD and non-BD groups are presented in Table 3.

**Table 3. Mean FRN amplitudes ( $\mu V$ ) in the non-BD and BD groups.**

	Non-BD ( $n = 23$ )		BD ( $n = 27$ )	
	Gain	Loss	Gain	Loss
FC3	3.41(2.27)	2.79(2.75)	3.44(2.02)	3.43(2.54)
FCz	4.94(3.50)	1.53(3.84)	4.51(2.59)	3.13(3.50)
FC4	3.32(2.35)	.62(2.56)	3.38(1.88)	1.87(2.68)
C3	3.98(2.60)	2.80(2.73)	3.73(1.88)	3.40(1.97)
Cz	7.11(4.07)	3.72(4.24)	6.88(2.65)	4.67(3.37)
C4	4.03(2.42)	1.51(2.48)	3.48(1.87)	1.78(2.46)
P3	4.49(2.50)	2.96(2.68)	3.39(1.88)	3.12(1.82)
Pz	7.04(3.45)	4.18(3.73)	6.04(2.65)	5.33(2.90)
P4	4.30(2.56)	1.63(3.11)	3.50(2.18)	1.77(3.08)

**Notes:** () standard deviation.

Main effects of group ( $F[1,48] = 6.67, p < .05$ ) and electrode site ( $F[8,384] = 12.32, p < .001$ ) were observed for  $\Delta$ FRN. The BD group exhibited a significantly smaller  $\Delta$ FRN compared to the non-BD group. The greatest  $\Delta$ FRN amplitude was observed at Cz, and the smallest was detected at FC3. The electrode site  $\times$  group interaction was not significant ( $F[8,384] = .70, p = .70$ ).

Main effects of valence ( $F[1,48] = 12.85, p < .01$ ) and electrode site ( $F[8,384] = 3.46, p < .05$ ) were observed in terms of FRN latencies. Thus, FRN latencies in response to gain feedback were shorter than those in response to loss feedback ( $p < .01$ ). In addition, the shortest latency was observed at Pz, and the longest was observed at P4. The valence  $\times$  electrode site interaction was also significant ( $F[8,384] = 10.25, p < .001$ ). The latencies in response to gain feedback were significantly shorter than those in response to the loss feedback at FCz, FC3, FC4, Cz, C3, and C4 but not at the other electrode sites. The valence  $\times$  group interaction was not significant ( $F[1,48] = 2.75, p = .10$ ). Mean FRN latencies of the BD and non-BD groups are presented in Table 4.

**Table 4. Mean FRN latencies (ms) in non-BD and BD groups.**

	Non-BD ( $n = 23$ )		BD ( $n = 27$ )	
	Gain	Loss	Gain	Loss
FC3	218.96(22.23)	244.17(22.19)	228.07(28.63)	235.93(23.10)
FCz	218.70(23.68)	244.17(17.18)	229.19(28.19)	240.07(19.77)
FC4	226.61(20.22)	236.70(19.58)	228.07(21.99)	238.30(8.53)
C3	216.96(16.48)	234.52(23.45)	227.85(24.95)	228.89(23.09)
Cz	218.35(20.64)	238.09(17.71)	225.41(28.07)	234.67(17.59)
C4	228.96(21.11)	235.39(17.95)	230.52(19.55)	234.15(17.51)
P3	230.26(25.20)	228.96(24.55)	221.85(18.68)	218.59(20.49)
Pz	228.26(23.96)	220.43(17.44)	224.15(19.99)	223.11(21.48)
P4	236.17(21.23)	231.48(18.67)	236.22(16.14)	230.96(15.77)

**Notes:** () standard deviation.

Main effects of valence ( $F[1,384] = 180.72, p < .001$ ) and electrode site ( $F[8,384] = 35.58, p < .001$ ) were observed in the P3 amplitudes. The P3 amplitudes in response to loss feedback were larger than those in response to gain feedback, and the largest and smallest P3 amplitudes were



observed at Cz and C3, respectively. A valence  $\times$  electrode site interaction was also observed ( $F[8,384] = 84.46, p < .001$ ), with the largest difference in P3 amplitudes between the gain and loss feedback at Cz and the smallest at P4. However, the main effect of group ( $F[1,48] = .64, p = .43$ ), the interaction effect of valence  $\times$  group ( $F[1,48] = .15, p = .70$ ), and the electrode site  $\times$  group interaction ( $F[8,384] = .59, p = .79$ ) were not significant. The mean P3 amplitudes of the BD and non-BD groups are presented in Table 5.

**Table 5. Mean P3 amplitudes ( $\mu V$ ) in the non-BD and BD groups.**

	Non-BD ( $n = 23$ )		BD ( $n = 27$ )	
	Gain	Loss	Gain	Loss
FC3	5.86(2.90)	10.49(4.87)	5.44(2.40)	10.11(3.46)
FCz	8.4(4.49)	16.65(7.60)	6.74(2.90)	14.63(5.00)
FC4	7.19(3.47)	10.92(5.53)	6.42(2.10)	9.97(4.16)
C3	6.02(2.71)	10.14(4.43)	5.95(2.25)	10.05(3.27)
Cz	9.08(4.68)	19.10(7.69)	8.18(3.25)	17.80(5.43)
C4	7.95(3.42)	10.75(4.58)	7.14(2.10)	10.14(3.58)
P3	6.80(3.03)	10.14(3.65)	6.71(2.22)	9.98(3.13)
Pz	8.71(4.04)	13.93(4.22)	8.96(2.70)	12.67(4.67)
P4	8.42(3.43)	10.62(4.17)	7.62(2.45)	10.01(4.04)

**Notes:** () standard deviation.

A main effect of electrode site was observed in  $\Delta P3$  ( $F[8,384] = 73.33, p < .001$ ). The largest  $\Delta P3$  amplitude was observed at Cz, and the smallest at P4. No main effect of group ( $F[1,48] = .23, p = .63$ ) or group  $\times$  electrode site interaction ( $F[8,384] = 1.08, p = .38$ ) was observed.

Main effects of valence ( $F[1,48] = 51.89, p < .001$ ) and electrode site ( $F[8,384] = 11.65, p < .001$ ) were observed for the P3 latencies. The P3 latencies in response to loss feedback were significantly shorter than those in response to gain feedback ( $p < .001$ ); the shortest latency was observed at Pz, and the longest at FC4. An interaction effect of valence  $\times$  electrode site was also significant ( $F[8,384] = 7.49, p < .001$ ). P3 latencies elicited by loss feedback were shorter than those by gain feedback at all electrode sites except FCz and Cz. The group  $\times$  valence interaction was not significant ( $F[1,48] = 2.93, p = .09$ ). The mean P3 latencies of the BD and non-BD groups are presented in Table 6.

**Table 6. Mean P3 latencies (ms) in the non-BD and BD groups.**

	Non- BD ( <i>n</i> = 23)		BD ( <i>n</i> = 27)	
	Gain	Loss	Gain	Loss
FC3	336.70(35.14)	324.17(22.20)	334.89(28.16)	315.93(23.10)
FCz	329.57(32.75)	324.17(17.18)	328.00(30.47)	320.07(19.77)
FC4	334.70(31.24)	316.70(19.58)	342.30(25.59)	318.30(18.53)
C3	339.39(35.19)	314.52(23.45)	346.30(30.74)	308.89(23.09)
Cz	317.83(32.79)	318.09(17.71)	324.30(36.81)	314.67(17.59)
C4	336.43(29.55)	315.39(17.95)	346.37(24.51)	314.15(17.51)
P3	334.26(33.27)	308.96(24.55)	337.26(33.62)	298.59(20.49)
Pz	311.13(29.54)	300.43(17.44)	316.37(35.65)	303.11(21.48)
P4	329.48(29.67)	311.48(18.67)	348.74(27.94)	310.96(15.77)

**Notes:** () standard deviation.

**Correlations between performance on the modified IGT and  $\Delta$ FRN/ $\Delta$ P3 amplitudes**

Positive correlations were observed between  $\Delta$ FRN amplitudes at FCz and total net scores ( $r = .298, p < .05$ ), sector 4 net scores ( $r = .333, p < .05$ ), and sector 5 net scores ( $r = .357, p < .05$ ) of the IGT. Thus, larger  $\Delta$ FRN amplitudes at FCz were associated with better IGT performance, especially in the later sectors of the IGT. On the other hand, no significant association was detected between the  $\Delta$ P3 amplitudes and IGT performance.

## Discussion

This study investigated feedback utilization ability for decision making in BD college students using the modified IGT and ERP data. The BD group exhibited significantly lower total net IGT scores and lower  $\Delta$ FRN amplitudes than did the non-BD group. Additionally, the  $\Delta$ FRN amplitude at the fronto-central area was positively correlated with the total net scores, sector 4 net scores, and sector 5 net scores on the IGT.

The BD group exhibited significantly lower total net scores than the non-BD group did, and performance of the non-BD group tended to increase as the task progressed (mean sector 1 = -.03; sector 2 = .73; sector 3 = .93; Sector 4 = 2.23; sector 5 = 1.25), whereas the BD group persistently chose disadvantageous cards over advantageous ones (mean sector 1 = -1.73; sector 2 = -3.04; sector 3 = -1.63; sector 4 = -1.41; sector 5 = -1.93). These results are consistent with those of previous studies [30-33] and suggest that individuals with BD have deficits in decision making. To maximize gains on the IGT, one must choose more advantageous cards that provide small initial gains but result in a net gain over disadvantageous cards that provide a large initial gain but result in a net loss. Johnson et al. [30] suggested that poor performance on the IGT in individuals with BD reflects their failure to consider consequences, i.e., tendency to pursue immediate rewards, disregarding the larger potential risk.

The statistical analyses of FRN, one of the ERP components elicited by feedback, revealed

that the BD group exhibited significantly lower  $\Delta$ FRN amplitudes than the non-BD group did. The non-BD group exhibited larger FRN amplitudes in response to loss feedback than to gain feedback, whereas the FRN amplitude differences in the BD group between gain and loss feedback were significantly smaller than those in the non-BD group. These results are consistent with previous studies on patients with AUD and male BD college students [69-71]. The present study also revealed that both groups exhibited larger FRN amplitudes in response to loss feedback than to gain feedback, which is consistent with many previous studies [58,60,97-99] and suggests that FRN is sensitive to feedback valence. FRN is known to reflect an early evaluation of feedback provided by the environment [48,60,98]. For example, Yeung and Sanfey [60] suggested that FRN and P3 reflect early and late stages of feedback processing, respectively. Gu et al. [98] reported that FRN reflects early feedback evaluation based on the salience of the feedback information.

Insensitivity to future consequences (IFC) in patients with AUD and substance use disorder (SUD) has been consistently reported [21,100,101]. For example, Cantrell et al. [101], using a modified version of the IGT, measured the preference for larger versus smaller rewards (PLvS), the difference between frequencies of choosing the cards that provide large gains and cards with small gains, and IFC, the difference between frequencies of choosing cards that result in a net loss and cards that result in a net gain in patients with AUD. The results showed that although patients with AUD did not exhibit significantly different PLvS scores, they exhibited significantly higher IFC scores than the control group. Additionally, a study of patients with SUD, including AUD, using the IGT and the Prospect Valence Model analysis observed a consistent lack of sensitivity to losses in patients with SUD [102]. Therefore, significantly smaller  $\Delta$ FRN amplitudes in the BD group compared to the non-BD group observed in the present study suggest that the BD group has deficits in early feedback evaluation and that they are less sensitive to loss feedback than are members of the non-BD group.

In this study, no significant difference in the P3 amplitudes was observed between the BD and non-BD groups, which was not consistent with previous studies reporting reduced P3 amplitudes

in patients with AUD [70,103]. The generators of P3 are known to be located in the temporo-parietal junction or locus coeruleus-norepinephrine system [67]. On the other hand, alcohol is known to affect frontal areas of the brain [68,104]. For example, those who consume alcohol exhibit reduced N450 amplitudes in frontal areas, whereas P3 amplitudes in the parietal and occipital areas are not affected by alcohol consumption [104]. Nelson et al. [68] also reported that alcohol consumption reduces both theta and delta band activities, which are known major components of FRN and P3, respectively, affecting theta band activity more severely. Whereas alcohol consumption affects the frontal area, overall grey and white matter volume reductions, including those in frontal areas, are observed in patients with AUD [46,105,106]. For example, one study observed reduced whole-brain network cluster coefficients in patients with AUD and reported that longer AUD duration was associated with a global decrease in the efficiency of the brain network [107]. These results suggest that alcohol consumption affects frontal areas first, and then spreads over the whole area as drinking duration increases. Taking together, our results imply that BD of relatively short duration (the mean drinking duration in the BD group was 33.33 months) may affect later feedback evaluation and attentional resource allocation relatively less severely than does BD with a long drinking history.

Both groups exhibited larger P3 amplitudes with loss feedback than with gain feedback. Studies on feedback-related ERPs using tasks other than the IGT have reported larger P3 amplitudes in response to gain feedback than to loss feedback [63,64,108,109], whereas studies using the IGT observed larger P3 amplitudes in response to loss feedback than to gain feedback [93,110]. Feedback-related P3 is known to be sensitive to different feedback information, not just to feedback valence but also to feedback magnitude and probabilities as well [60,63,64,66,97,98,111]. This suggests that P3 reflects feedback processing with a top-down mechanism that allocates attentional resources to the information relevant to the task at hand [67,98]. The loss magnitudes of each card must be understood to maximize profit on the IGT. Thus, participants need to understand that disadvantageous cards (A and B) result in large gains, but losses will soon accumulate over gains, and thus shift their preference or attention progressively toward advantageous cards (C and D) [44]. These results suggest that both

groups allocated their attentional resources to feedback valence, especially to loss feedback, while taking the modified IGT.

Although the importance of feedback utilization for decision making has been emphasized [34,48], only one study has investigated the association between IGT performance and feedback-related ERPs [93]. Carlson et al. [93] investigated how children responded to gain/loss feedback using P3 and evaluated how anticipation prior to the response was related to behavioral adjustment using stimulus-preceding negativity (SPN). That study found that the difference in children's SPN amplitude between advantageous and disadvantageous decks was positively correlated with behavioral adjustment. In the present study,  $\Delta$ FRN amplitudes at FCz were positively correlated with total net scores and sectors 4 and 5 net scores on the IGT. Thus, larger differences between FRN amplitudes with gain and loss feedback were associated with improved performance on the modified IGT. No previous study has reported an association between FRN and IGT performance; studies using the reversal learning task have reported associations between FRN and behavioral adjustments [108,112]. For example, Frank et al. [112] compared negative learners, who learn stimulus-result contingencies by avoiding negative feedback, with positive learners, who learn these contingencies by pursuing positive feedback; they found significant positive correlations between the tendency to avoid negative feedback and error-related negativity (ERN) amplitudes, which is the ERP component known to share some neural sources with FRN [94]. To perform successfully on the IGT, participants must learn the contingencies between the cards and their consequences implicitly during the task (e.g., gain or loss feedback) [113]. Therefore, these results suggest that early feedback evaluation in the fronto-central area is associated with the implicit learning process during decision making.

No significant associations between  $\Delta$ P3 amplitudes and IGT performance were observed in this study. Previous results for P3 amplitudes and behavioral adjustments using the reversal learning task are inconsistent [112,114]. For example, Frank et al. [112] reported that FRN amplitudes, not P3 amplitudes, predicted behavioral adjustment, whereas Chase et al. [114] reported that P3 amplitudes,

not FRN amplitudes, predicted behavioral adjustment. The differences between these two studies lay in the task instructions. Frank et al. [112] did not provide any information regarding contingency shifts during the task and requested that participants make decisions based on their internal judgment, whereas Chase et al. [114] told the participants that the contingency would shift during the task and requested that participants adjust their responses when they were certain that the contingency had shifted. Thus, the latter study reflected decision making based more on a set of rules provided prior to the task than on the actual feedback during the task. San Martin [48] suggested that the importance of FRN and P3 in behavioral adjustment varies depending on which information is more important when performing the given task. In our study, participants were only instructed regarding the goal and process of the task. Therefore, the rules of the task (probability of loss and magnitude of cards) must be learned solely through feedbacks. Such a task design is closer to the study by Frank et al. [112]. These results suggest that both the BD and non-BD groups relied more on early feedback evaluation of valence than on late evaluation with a top-down mechanism as they performed the modified IGT.

This study has several limitations. First, the feedback evaluation investigated here focused mainly on feedback valence. The magnitudes of the cards on the modified IGT increased, as was the case in the original IGT [41]. Although this may keep participants motivated, the increasing magnitude forbids examination of how feedback-related ERPs respond differently to feedback of small or large magnitude. Additionally, the probabilities of encountering losses from cards B and D were too low (10%) to secure enough trials to investigate how ERPs differ based on the probability of losses. Second, this study measured feedback utilization using time-based ERPs. However, the time windows for FRN and P3 are close to each other, and they may distort each other in ERP waveforms. Difference waves were measured to isolate ERP components and prevent such distortion, but other techniques, such as spectral analysis or functional connectivity analysis, may reveal more detailed information, such as how different neural waves interact and communicate during feedback processing.

In conclusion, the BD group exhibited significantly lower total net scores on the modified IGT and significantly lower  $\Delta$ FRN amplitudes. On the other hand, no differences were observed in  $\Delta$ P3 or P3 amplitudes between the groups. Additionally, positive correlations were observed between  $\Delta$ FRN amplitudes in the fronto-central area and IGT performance. These results imply that the BD group had deficits in decision making and early feedback evaluation, with a tendency to pursue immediate large gains even at greater potential risks, revealing deficits in early evaluation regarding feedback valence.

## Author Contributions

**Conceptualization:** Myung-Sun Kim.

**Formal analysis:** Eun-Chan Na, Kyoung-Mi Jang.

**Funding acquisition:** Myung-Sun Kim.

**Methodology:** Eun-Chan Na, Kyoung-Mi Jang.

**Supervision:** Myung-Sun Kim.

**Writing – review & editing:** Eun-Chan Na, Kyoung-Mi Jang, Myung-Sun Kim.



# References

1. Maurage P, Bestelmeyer PE, Rouger J, Charest I, Belin P. Binge drinking influences the cerebral processing of vocal affective bursts in young adults. *Neuroimage Clin.* 2013;3:218-225.<https://doi.org/10.1016/j.nicl.2013.08.010> PMID: 24179866
2. Parada M, Corral M, Mota N, Crego A, Rodriguez Holguin S, Cadaveira F. Executive functioning and alcohol binge drinking in university students. *Addict Behav.* 2012;37(2):167-172.<https://doi.org/10.1016/j.addbeh.2011.09.015> PMID: 21996093
3. Wechsler H, Nelson TF. Binge drinking and the American college student: what's five drinks? *Psychology of addictive behaviors : journal of the Society of Psychologists in Addictive Behaviors.* 2001;15(4):287-291. PMID: 11767258
4. Chun S, Sohn A, Song CH, Lee JY, Kim SK. Health and behavioral consequences of binge drinking in college: A national survey of students at 60 campuses. *Journal of Korean Alcohol Science.* 2003;4(2):119-135.
5. Stephens DN, Duka T. Review. Cognitive and emotional consequences of binge drinking: role of amygdala and prefrontal cortex. *Philosophical transactions of the Royal Society of London Series B, Biological sciences.* 2008;363(1507):3169-3179.<https://doi.org/10.1098/rstb.2008.0097> PMID: 18640918
6. Cha D. Understanding binge-drinking: A test of the theory of planned behavior. *J Korean Journal of Journalism Communication Studies.* 2005;49(3):346-390.
7. Chun S. Analysis of college student binge drinking and alcohol-related problems. *J Korean Alcohol Sci.* 2002;3(2):221-233.
8. Naimi TS, Brewer RD, Mokdad A, Denny C, Serdula MK, Marks JS. Binge drinking among US adults. *Jama.* 2003;289(1):70-75. PMID: 12503979
9. Campanella S, Peigneux P, Petit G, Lallemand F, Saeremans M, Noel X, et al. Increased cortical activity in binge drinkers during working memory task: a preliminary assessment

- through a functional magnetic resonance imaging study. PloS one. 2013;8(4):e62260.<https://doi.org/10.1371/journal.pone.0062260> PMID: 23638017
10. Crego A, Rodriguez-Holguin S, Parada M, Mota N, Corral M, Cadaveira F. Reduced anterior prefrontal cortex activation in young binge drinkers during a visual working memory task. Drug and alcohol dependence. 2010;109(1-3):45-56.<https://doi.org/10.1016/j.drugalcdep.2009.11.020> PMID: 20079980
11. Kanny D, Liu Y, Brewer RD, Lu H. Binge drinking - United States, 2011. MMWR supplements. 2013;62(3):77-80. PMID: 24264494
12. Lopez-Caneda E, Cadaveira F, Crego A, Gomez-Suarez A, Corral M, Parada M, et al. Hyperactivation of right inferior frontal cortex in young binge drinkers during response inhibition: a follow-up study. Addiction (Abingdon, England). 2012;107(10):1796-1808.<https://doi.org/10.1111/j.1360-0443.2012.03908.x> PMID: 22487028
13. Mota N, Parada M, Crego A, Doallo S, Caamano-Isorna F, Rodriguez Holguin S, et al. Binge drinking trajectory and neuropsychological functioning among university students: a longitudinal study. Drug and alcohol dependence. 2013;133(1):108-114.<https://doi.org/10.1016/j.drugalcdep.2013.05.024> PMID: 23791027
14. Jennison KM. The short-term effects and unintended long-term consequences of binge drinking in college: a 10-year follow-up study. The American journal of drug and alcohol abuse. 2004;30(3):659-684. PMID: 15540499
15. O'Neill SE, Parra GR, Sher KJ. Clinical relevance of heavy drinking during the college years: cross-sectional and prospective perspectives. Psychology of addictive behaviors : journal of the Society of Psychologists in Addictive Behaviors. 2001;15(4):350-359. PMID: 11767268
16. Tucker JS, Orlando M, Ellickson PL. Patterns and correlates of binge drinking trajectories from early adolescence to young adulthood. Health psychology : official journal of the Division of Health Psychology, American Psychological Association. 2003;22(1):79-87.

PMID: 12558205

17. Association AP. Diagnostic and statistical manual of mental disorders (DSM-IV). Washington DC: American Psychiatric Association; 1994.
18. Association AP. Diagnostic and statistical manual of mental disorders (DSM-5®). Washington DC: American Psychiatric Association; 2013.
19. Bechara A, Dolan S, Denburg N, Hindes A, Anderson SW, Nathan PE. Decision-making deficits, linked to a dysfunctional ventromedial prefrontal cortex, revealed in alcohol and stimulant abusers. *Neuropsychologia*. 2001;39(4):376-389. PMID: 11164876
20. Mazas CA, Finn PR, Steinmetz JE. Decision-making biases, antisocial personality, and early-onset alcoholism. *Alcoholism, clinical and experimental research*. 2000;24(7):1036-1040. PMID: 10924007
21. Mallett KA, Lee CM, Neighbors C, Larimer ME, Turrissi R. Do we learn from our mistakes? An examination of the impact of negative alcohol-related consequences on college students' drinking patterns and perceptions. *Journal of studies on alcohol*. 2006;67(2):269-276. PMID: 16562409
22. MacKillop J, Miranda R, Jr., Monti PM, Ray LA, Murphy JG, Rohsenow DJ, et al. Alcohol demand, delayed reward discounting, and craving in relation to drinking and alcohol use disorders. *Journal of abnormal psychology*. 2010;119(1):106-114. <https://doi.org/10.1037/a0017513> PMID: 20141247
23. Amlung M, Sweet LH, Acker J, Brown CL, MacKillop J. Dissociable brain signatures of choice conflict and immediate reward preferences in alcohol use disorders. *Addiction biology*. 2014;19(4):743-753. <https://doi.org/10.1111/adb.12017> PMID: 23231650
24. Bechara A. Risky business: emotion, decision-making, and addiction. *Journal of gambling studies*. 2003;19(1):23-51. PMID: 12635539
25. Fein G, Klein L, Finn P. Impairment on a simulated gambling task in long-term abstinent alcoholics. *Alcoholism, clinical and experimental research*. 2004;28(10):1487-1491. PMID:

15597080

26. Goudriaan AE, Oosterlaan J, de Beurs E, van den Brink W. Decision making in pathological gambling: a comparison between pathological gamblers, alcohol dependents, persons with Tourette syndrome, and normal controls. *Brain research Cognitive brain research*. 2005;23(1):137-151. <https://doi.org/10.1016/j.cogbrainres.2005.01.017> PMID: 15795140
27. Mitchell JM, Fields HL, D'Esposito M, Boettiger CA. Impulsive responding in alcoholics. *Alcoholism, clinical and experimental research*. 2005;29(12):2158-2169. PMID: 16385186
28. Noel X, Bechara A, Dan B, Hanak C, Verbanck P. Response inhibition deficit is involved in poor decision making under risk in nonamnesic individuals with alcoholism. *Neuropsychology*. 2007;21(6):778-786. <https://doi.org/10.1037/0894-4105.21.6.778> PMID: 17983291
29. Goudriaan AE, Grekin ER, Sher KJ. Decision making and binge drinking: a longitudinal study. *Alcoholism, clinical and experimental research*. 2007;31(6):928-938. <https://doi.org/10.1111/j.1530-0277.2007.00378.x> PMID: 17403069
30. Johnson CA, Xiao L, Palmer P, Sun P, Wang Q, Wei Y, et al. Affective decision-making deficits, linked to a dysfunctional ventromedial prefrontal cortex, revealed in 10th grade Chinese adolescent binge drinkers. *Neuropsychologia*. 2008;46(2):714-726. <https://doi.org/10.1016/j.neuropsychologia.2007.09.012> PMID: 17996909
31. Xiao L, Bechara A, Grenard LJ, Stacy WA, Palmer P, Wei Y, et al. Affective decision-making predictive of Chinese adolescent drinking behaviors. *Journal of the International Neuropsychological Society : JINS*. 2009;15(4):547-557. <https://doi.org/10.1017/s1355617709090808> PMID: 19573273
32. Xiao L, Bechara A, Gong Q, Huang X, Li X, Xue G, et al. Abnormal affective decision making revealed in adolescent binge drinkers using a functional magnetic resonance imaging study. *Psychology of addictive behaviors : journal of the Society of Psychologists in Addictive Behaviors*. 2013;27(2):443-454. <https://doi.org/10.1037/a0027892> PMID:

- 22486330
33. Yoo JY, Kim MS. Deficits in Decision-Making and reversal learning in college students who participate in Binge drinking. *J Neuropsychiatry*. 2016;6(6):321-330.
34. Ernst M, Paulus MP. Neurobiology of decision making: a selective review from a neurocognitive and clinical perspective. *Biological psychiatry*. 2005;58(8):597-604.<https://doi.org/10.1016/j.biopsych.2005.06.004> PMID: 16095567
35. Bechara A, Damasio H, Damasio AR, Lee GP. Different contributions of the human amygdala and ventromedial prefrontal cortex to decision-making. *The Journal of neuroscience : the official journal of the Society for Neuroscience*. 1999;19(13):5473-5481. PMID: 10377356
36. Bechara A, Damasio H, Damasio AR. Emotion, decision making and the orbitofrontal cortex. *Cerebral cortex (New York, NY : 1991)*. 2000;10(3):295-307. PMID: 10731224
37. Bush G, Vogt BA, Holmes J, Dale AM, Greve D, Jenike MA, et al. Dorsal anterior cingulate cortex: a role in reward-based decision making. *Proceedings of the National Academy of Sciences of the United States of America*. 2002;99(1):523-528.<https://doi.org/10.1073/pnas.012470999> PMID: 11756669
38. Ernst M, Bolla K, Mouratidis M, Contoreggi C, Matochik JA, Kurian V, et al. Decision-making in a risk-taking task: a PET study. *Neuropsychopharmacology : official publication of the American College of Neuropsychopharmacology*. 2002;26(5):682-691.[https://doi.org/10.1016/s0893-133x\(01\)00414-6](https://doi.org/10.1016/s0893-133x(01)00414-6) PMID: 11927193
39. Kennerley SW, Walton ME, Behrens TE, Buckley MJ, Rushworth MF. Optimal decision making and the anterior cingulate cortex. *Nature neuroscience*. 2006;9(7):940-947.<https://doi.org/10.1038/nn1724> PMID: 16783368
40. Wallis JD. Orbitofrontal cortex and its contribution to decision-making. *Annual review of neuroscience*. 2007;30:31-56.<https://doi.org/10.1146/annurev.neuro.30.051606.094334> PMID: 17417936

41. Buelow MT, Suhr JA. Construct validity of the Iowa Gambling Task. *Neuropsychology review*. 2009;19(1):102-114.<https://doi.org/10.1007/s11065-009-9083-4> PMID: 19194801
42. Bechara A, Damasio AR, Damasio H, Anderson SW. Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition*. 1994;50(1-3):7-15. PMID: 8039375
43. Dunn BD, Dalgleish T, Lawrence AD. The somatic marker hypothesis: a critical evaluation. *Neuroscience and biobehavioral reviews*. 2006;30(2):239-271.<https://doi.org/10.1016/j.neubiorev.2005.07.001> PMID: 16197997
44. Webb CA, DelDonno S, Killgore WD. The role of cognitive versus emotional intelligence in Iowa Gambling Task performance: What's emotion got to do with it? *Intelligence*. 2014;44:112-119.<https://doi.org/10.1016/j.intell.2014.03.008> PMID: 25635149
45. Dom G, De Wilde B, Hulstijn W, van den Brink W, Sabbe B. Decision-making deficits in alcohol-dependent patients with and without comorbid personality disorder. *Alcoholism, clinical and experimental research*. 2006;30(10):1670-1677.<https://doi.org/10.1111/j.1530-0277.2006.00202.x> PMID: 17010134
46. Le Berre AP, Rauchs G, La Joie R, Mezenge F, Boudehent C, Vabret F, et al. Impaired decision-making and brain shrinkage in alcoholism. *European psychiatry : the journal of the Association of European Psychiatrists*. 2014;29(3):125-133.<https://doi.org/10.1016/j.eurpsy.2012.10.002> PMID: 23182846
47. Moreno M, Estevez AF, Zaldivar F, Montes JM, Gutierrez-Ferre VE, Esteban L, et al. Impulsivity differences in recreational cannabis users and binge drinkers in a university population. *Drug and alcohol dependence*. 2012;124(3):355-362.<https://doi.org/10.1016/j.drugalcdep.2012.02.011> PMID: 22425410
48. San Martin R. Event-related potential studies of outcome processing and feedback-guided learning. *Frontiers in human neuroscience*. 2012;6:304.<https://doi.org/10.3389/fnhum.2012.00304> PMID: 23162451

49. Delgado MR, Nystrom LE, Fissell C, Noll DC, Fiez JA. Tracking the hemodynamic responses to reward and punishment in the striatum. *Journal of neurophysiology*. 2000;84(6):3072-3077.<https://doi.org/10.1152/jn.2000.84.6.3072> PMID: 11110834
50. Elliott R, Friston KJ, Dolan RJ. Dissociable neural responses in human reward systems. *The Journal of neuroscience : the official journal of the Society for Neuroscience*. 2000;20(16):6159-6165. PMID: 10934265
51. Knutson B, Westdorp A, Kaiser E, Hommer D. FMRI visualization of brain activity during a monetary incentive delay task. *NeuroImage*. 2000;12(1):20-27.<https://doi.org/10.1006/nimg.2000.0593> PMID: 10875899
52. O'Doherty J, Kringelbach ML, Rolls ET, Hornak J, Andrews C. Abstract reward and punishment representations in the human orbitofrontal cortex. *Nature neuroscience*. 2001;4(1):95-102.<https://doi.org/10.1038/82959> PMID: 11135651
53. Rogers RD, Ramnani N, Mackay C, Wilson JL, Jezzard P, Carter CS, et al. Distinct portions of anterior cingulate cortex and medial prefrontal cortex are activated by reward processing in separable phases of decision-making cognition. *Biological psychiatry*. 2004;55(6):594-602.<https://doi.org/10.1016/j.biopsych.2003.11.012> PMID: 15013828
54. O'Doherty JP, Dayan P, Friston K, Critchley H, Dolan RJ. Temporal difference models and reward-related learning in the human brain. *Neuron*. 2003;38(2):329-337. PMID: 12718865
55. Elliott R, Dolan RJ, Frith CD. Dissociable functions in the medial and lateral orbitofrontal cortex: evidence from human neuroimaging studies. *Cerebral cortex (New York, NY : 1991)*. 2000;10(3):308-317. PMID: 10731225
56. McClure SM, Berns GS, Montague PR. Temporal prediction errors in a passive learning task activate human striatum. *Neuron*. 2003;38(2):339-346. PMID: 12718866
57. Pagnoni G, Zink CF, Montague PR, Berns GS. Activity in human ventral striatum locked to errors of reward prediction. *Nature neuroscience*. 2002;5(2):97-98.<https://doi.org/10.1038/nn802> PMID: 11802175



58. Gehring WJ, Willoughby AR. The medial frontal cortex and the rapid processing of monetary gains and losses. *Science (New York, NY)*. 2002;295(5563):2279-2282.<https://doi.org/10.1126/science.1066893> PMID: 11910116
59. Kamarajan C, Porjesz B, Rangaswamy M, Tang Y, Chorlian DB, Padmanabhapillai A, et al. Brain signatures of monetary loss and gain: outcome-related potentials in a single outcome gambling task. *Behavioural brain research*. 2009;197(1):62-76.<https://doi.org/10.1016/j.bbr.2008.08.011> PMID: 18775749
60. Yeung N, Sanfey AG. Independent coding of reward magnitude and valence in the human brain. *The Journal of neuroscience : the official journal of the Society for Neuroscience*. 2004;24(28):6258-6264.<https://doi.org/10.1523/jneurosci.4537-03.2004> PMID: 15254080
61. Tobler PN, Fiorillo CD, Schultz W. Adaptive coding of reward value by dopamine neurons. *Science (New York, NY)*. 2005;307(5715):1642-1645.<https://doi.org/10.1126/science.1105370> PMID: 15761155
62. Bellebaum C, Poleszki D, Daum I. It is less than you expected: the feedback-related negativity reflects violations of reward magnitude expectations. *Neuropsychologia*. 2010;48(11):3343-3350.<https://doi.org/10.1016/j.neuropsychologia.2010.07.023> PMID: 20655319
63. Wu Y, Zhou X. The P300 and reward valence, magnitude, and expectancy in outcome evaluation. *Brain research*. 2009;1286:114-122.<https://doi.org/10.1016/j.brainres.2009.06.032> PMID: 19539614
64. Hajcak G, Moser JS, Holroyd CB, Simons RF. It's worse than you thought: the feedback negativity and violations of reward prediction in gambling tasks. *Psychophysiology*. 2007;44(6):905-912.<https://doi.org/10.1111/j.1469-8986.2007.00567.x> PMID: 17666029
65. Poleszki D, Sartori G, Rumiati R, Vidotto G, Daum I. Brain correlates of risky decision-making. *NeuroImage*. 2010;49(2):1886-1894.<https://doi.org/10.1016/j.neuroimage.2009.08.068> PMID: 19761850



66. Xu Q, Shen Q, Chen P, Ma Q, Sun D, Pan Y. How an uncertain cue modulates subsequent monetary outcome evaluation: an ERP study. *Neuroscience letters*. 2011;505(2):200-204.<https://doi.org/10.1016/j.neulet.2011.10.024> PMID: 22027182
67. Nieuwenhuis S, Aston-Jones G, Cohen JD. Decision making, the P3, and the locus coeruleus-norepinephrine system. *Psychological bulletin*. 2005;131(4):510-532.<https://doi.org/10.1037/0033-2909.131.4.510> PMID: 16060800
68. Nelson LD, Patrick CJ, Collins P, Lang AR, Bernat EM. Alcohol impairs brain reactivity to explicit loss feedback. *Psychopharmacology*. 2011;218(2):419-428.<https://doi.org/10.1007/s00213-011-2323-3> PMID: 21559803
69. Fein G, Chang M. Smaller feedback ERN amplitudes during the BART are associated with a greater family history density of alcohol problems in treatment-naïve alcoholics. *Drug and alcohol dependence*. 2008;92(1-3):141-148.<https://doi.org/10.1016/j.drugalcdep.2007.07.017> PMID: 17869027
70. Kamarajan C, Rangaswamy M, Tang Y, Chorlian DB, Pandey AK, Roopesh BN, et al. Dysfunctional reward processing in male alcoholics: an ERP study during a gambling task. *Journal of psychiatric research*. 2010;44(9):576-590.<https://doi.org/10.1016/j.jpsychires.2009.11.019> PMID: 20035952
71. Wahlstrom LC. Feedback-related negativity, decision-making, and college binge drinking. Dissertation, Lincoln, NE: The University of Nebraska-Lincoln; 2013.
72. Schuermann B, Kathmann N, Stiglmayr C, Renneberg B, Endrass T. Impaired decision making and feedback evaluation in borderline personality disorder. *Psychological medicine*. 2011;41(9):1917-1927.<https://doi.org/10.1017/s003329171000262x> PMID: 21262034
73. O'Malley PM, Johnston LD. Epidemiology of alcohol and other drug use among American college students. *Journal of studies on alcohol Supplement*. 2002(14):23-39. PMID: 12022728
74. Wechsler H, Lee JE, Kuo M, Seibring M, Nelson TF, Lee H. Trends in college binge

- drinking during a period of increased prevention efforts. Findings from 4 Harvard School of Public Health College Alcohol Study surveys: 1993-2001. Journal of American college health : J of ACH. 2002;50(5):203-217.<https://doi.org/10.1080/07448480209595713> PMID: 11990979
75. Weitzman ER, Nelson TF, Wechsler H. Taking up binge drinking in college: the influences of person, social group, and environment. The Journal of adolescent health : official publication of the Society for Adolescent Medicine. 2003;32(1):26-35. PMID: 12507798
76. Bolla KI, Eldreth DA, Matochik JA, Cadet JL. Sex-related differences in a gambling task and its neurological correlates. Cerebral cortex (New York, NY : 1991). 2004;14(11):1226-1232.<https://doi.org/10.1093/cercor/bhh083> PMID: 15142963
77. Larson MJ, South M, Clayson PE. Sex differences in error-related performance monitoring. Neuroreport. 2011;22(1):44-48.<https://doi.org/10.1097/WNR.0b013e3283427403> PMID: 21304441
78. Park S, Kim MS. An event-related potential study of spatial working memory in binge drinking college students. PloS one. 2018;13(9):e0203696.<https://doi.org/10.1371/journal.pone.0203696> PMID: 30199547
79. Barbor T, La Fuente J, Saunders J, Grant M. AUDIT: the alcohol use disorders identification test: guidelines for use in primary health care. Geneva: World Health Organization; 1992.
80. Lee B, Lee C, Lee P, Choi M, Namkoong K. Development of Korean version of alcohol use disorders identification test (AUDIT-K): Its reliability and validity. J Korean Acad Addict Psychiatry. 2000;4(2):83-92. PMID: 18640538
81. Mehrabian A, Russell JA. A questionnaire measure of habitual alcohol use. Psychological reports. 1978;43(3):803-806. PMID: 740823
82. Kim JS, Oh MK, Park BK, Lee MK, Kim GJ. Screening criteria of alcoholism by alcohol use disorders identification test (AUDIT) in Korea. Journal of the Korean Academy of Family Medicine. 1999;20(9):1152-1159.

83. Conigrave KM, Hall WD, Saunders JB. The AUDIT questionnaire: choosing a cut-off score. Alcohol Use Disorder Identification Test. Addiction (Abingdon, England). 1995;90(10):1349-1356. PMID: 8616463
84. First MB, Spitzer RL, Gibbon M, Williams JB. Structured clinical interview for DSM-IV axis I disorders-Patient edition (SCID-I/P, Version 2.0) New York. NY: Biometrics Research Department, New York State Psychiatric Institute; 1995.
85. Zung WW, Richards CB, Short MJ. Self-rating depression scale in an outpatient clinic. Further validation of the SDS. Archives of general psychiatry. 1965;13(6):508-515. PMID: 4378854
86. Spielberger C, Gorsuch RL, Lushene R, Vagg P, Jacobs G. Manual for the state-trait anxiety inventory. Palo Alto, CA: Consulting Psychologists; 1983.
87. Patton JH, Stanford MS, Barratt ES. Factor structure of the Barratt impulsiveness scale. Journal of clinical psychology. 1995;51(6):768-774. PMID: 8778124
88. Jones J. The Children of Alcoholics Screening Test: Test Manual (Chicago, Camelot Unlimited). Chicago: Camelot Unlimited; 1983.
89. Kim M, Chang H, Kim K. Development of the Korean version of the children of alcoholics screening test (CAST-K): a reliability and validity study. J Korean Neuropsychiatr Assoc. 1995;34(4):1182-1193.
90. Townshend JM, Duka T. Patterns of alcohol drinking in a population of young social drinkers: a comparison of questionnaire and diary measures. Alcohol and alcoholism (Oxford, Oxfordshire). 2002;37(2):187-192. PMID: 11912076
91. Tucker DM. Spatial sampling of head electrical fields: the geodesic sensor net. Electroencephalography and clinical neurophysiology. 1993;87(3):154-163. PMID: 7691542
92. Luck SJ. An introduction to the event-related potential technique. Cambridge, MA: MIT press; 2014.
93. Carlson SM, Zayas V, Guthormsen A. Neural correlates of decision making on a gambling

- task. Child development. 2009;80(4):1076-1096.<https://doi.org/10.1111/j.1467-8624.2009.01318.x> PMID: 19630895
94. Holroyd C. A note on the oddball N200 and the feedback ERN. Neurophysiology. 2004;78:447-455.
95. Holroyd CB, Krigolson OE, Baker R, Lee S, Gibson J. When is an error not a prediction error? An electrophysiological investigation. Cognitive, Affective, Behavioral Neuroscience. 2009;9(1):59-70. PMID: 19246327
96. Walsh MM, Anderson JR. Modulation of the feedback-related negativity by instruction and experience. Proceedings of the National Academy of Sciences of the United States of America. 2011;108(47):19048-19053.<https://doi.org/10.1073/pnas.1117189108> PMID: 22065792
97. Goyer JP, Woldorff MG, Huettel SA. Rapid electrophysiological brain responses are influenced by both valence and magnitude of monetary rewards. Journal of cognitive neuroscience. 2008;20(11):2058-2069.<https://doi.org/10.1162/jocn.2008.20134> PMID: 18416673
98. Gu R, Lei Z, Broster L, Wu T, Jiang Y, Luo YJ. Beyond valence and magnitude: a flexible evaluative coding system in the brain. Neuropsychologia. 2011;49(14):3891-3897.<https://doi.org/10.1016/j.neuropsychologia.2011.10.006> PMID: 22019775
99. Holroyd CB, Coles MGH. The neural basis of human error processing: reinforcement learning, dopamine, and the error-related negativity. Psychological review. 2002;109(4):679-709.<https://doi.org/10.1037/0033-295x.109.4.679> PMID: 12374324
100. Bechara A, Damasio H. Decision-making and addiction (part I): impaired activation of somatic states in substance dependent individuals when pondering decisions with negative future consequences. Neuropsychologia. 2002;40(10):1675-1689. PMID: 11992656
101. Cantrell H, Finn PR, Rickert ME, Lucas J. Decision making in alcohol dependence: insensitivity to future consequences and comorbid disinhibitory psychopathology.

- Alcoholism, clinical and experimental research. 2008;32(8):1398-1407.<https://doi.org/10.1111/j.1530-0277.2008.00714.x> PMID: 18565158
102. Baitz HA. Component processes of decision making in persons with substance use disorders. Dissertation, Burnaby (BC): Simon Fraser University; 2016.
103. Porjesz B, Begleiter H, Bihari B, Kissin B. Event-related brain potentials to high incentive stimuli in abstinent alcoholics. Alcohol (Fayetteville, NY). 1987;4(4):283-287. PMID: 3620097
104. Curtin JJ, Fairchild BA. Alcohol and cognitive control: implications for regulation of behavior during response conflict. Journal of abnormal psychology. 2003;112(3):424-436. PMID: 12943021
105. Buhler M, Mann K. Alcohol and the human brain: a systematic review of different neuroimaging methods. Alcoholism, clinical and experimental research. 2011;35(10):1771-1793.<https://doi.org/10.1111/j.1530-0277.2011.01540.x> PMID: 21777260
106. Fein G, Di Sclafani V, Cardenas VA, Goldmann H, Tolou-Shams M, Meyerhoff DJ. Cortical gray matter loss in treatment-naive alcohol dependent individuals. Alcoholism, clinical and experimental research. 2002;26(4):558-564. PMID: 11981133
107. Sjoerds Z, Stufflebeam SM, Veltman DJ, Van den Brink W, Penninx BW, Douw L. Loss of brain graph network efficiency in alcohol dependence. Addiction biology. 2017;22(2):523-534.<https://doi.org/10.1111/adb.12346> PMID: 26692359
108. Bellebaum C, Daum I. Learning-related changes in reward expectancy are reflected in the feedback-related negativity. The European journal of neuroscience. 2008;27(7):1823-1835.<https://doi.org/10.1111/j.1460-9568.2008.06138.x> PMID: 18380674
109. Toyomaki A, Murohashi H. The ERPs to feedback indicating monetary loss and gain on the game of modified “rock–paper–scissors”. International Congress Series. 2005;1278:381-384.
110. Cui JF, Chen YH, Wang Y, Shum DH, Chan RC. Neural correlates of uncertain decision making: ERP evidence from the Iowa Gambling Task. Frontiers in human neuroscience.

2013;7:776.<https://doi.org/10.3389/fnhum.2013.00776> PMID: 24298248

111.Hajcak G, Simons RF. Oops!.. I did it again: an ERP and behavioral study of double-errors.

Brain and cognition. 2008;68(1):15-21.<https://doi.org/10.1016/j.bandc.2008.02.118> PMID:

18442875

112.Frank MJ, Woroch BS, Curran T. Error-related negativity predicts reinforcement learning

and conflict biases. Neuron. 2005;47(4):495-

501.<https://doi.org/10.1016/j.neuron.2005.06.020> PMID: 16102533

113.Bechara A. The role of emotion in decision-making: evidence from neurological patients

with orbitofrontal damage. Brain and cognition. 2004;55(1):30-

40.<https://doi.org/10.1016/j.bandc.2003.04.001> PMID: 15134841

114.Chase HW, Swainson R, Durham L, Benham L, Cools R. Feedback-related negativity codes

prediction error but not behavioral adjustment during probabilistic reversal learning. Journal

of cognitive neuroscience. 2011;23(4):936-946.<https://doi.org/10.1162/jocn.2010.21456>

PMID: 20146610

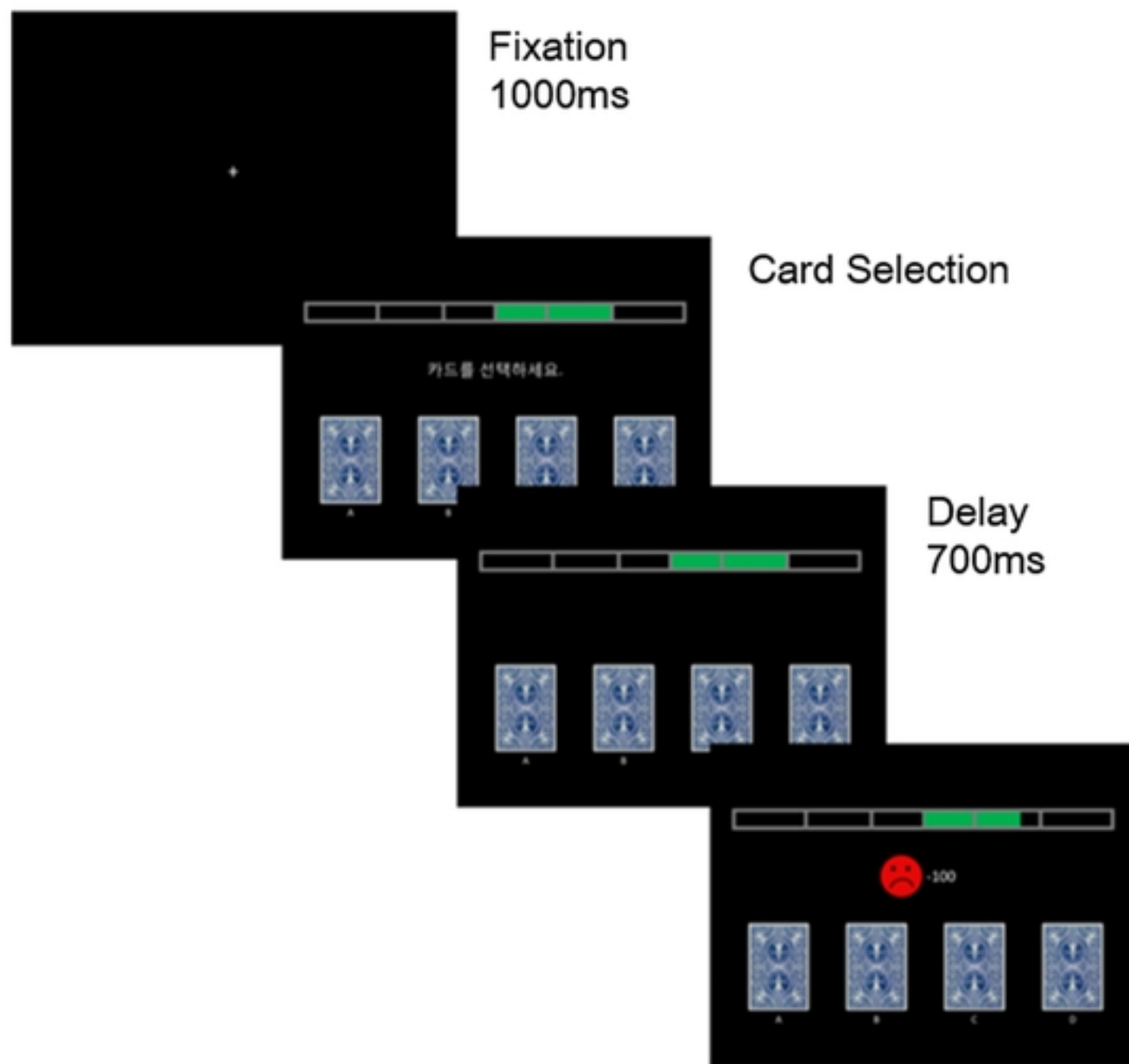
## Supporting information

**S1 File. Behavioral data.**

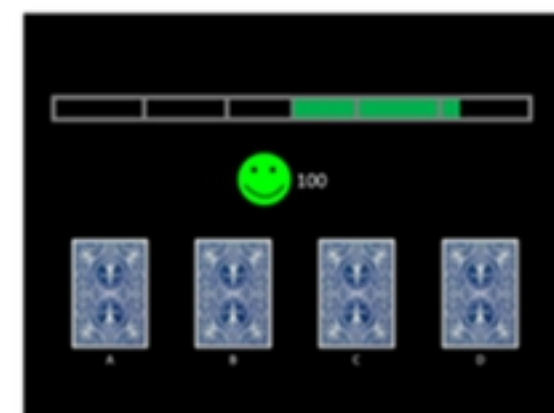
(XLSX)

**S2 File. ERP data.**

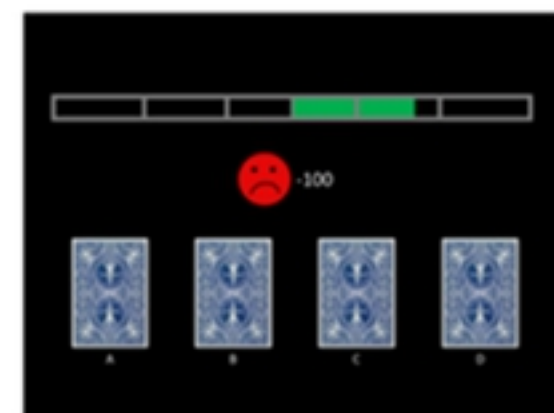
(XLSX)



A)



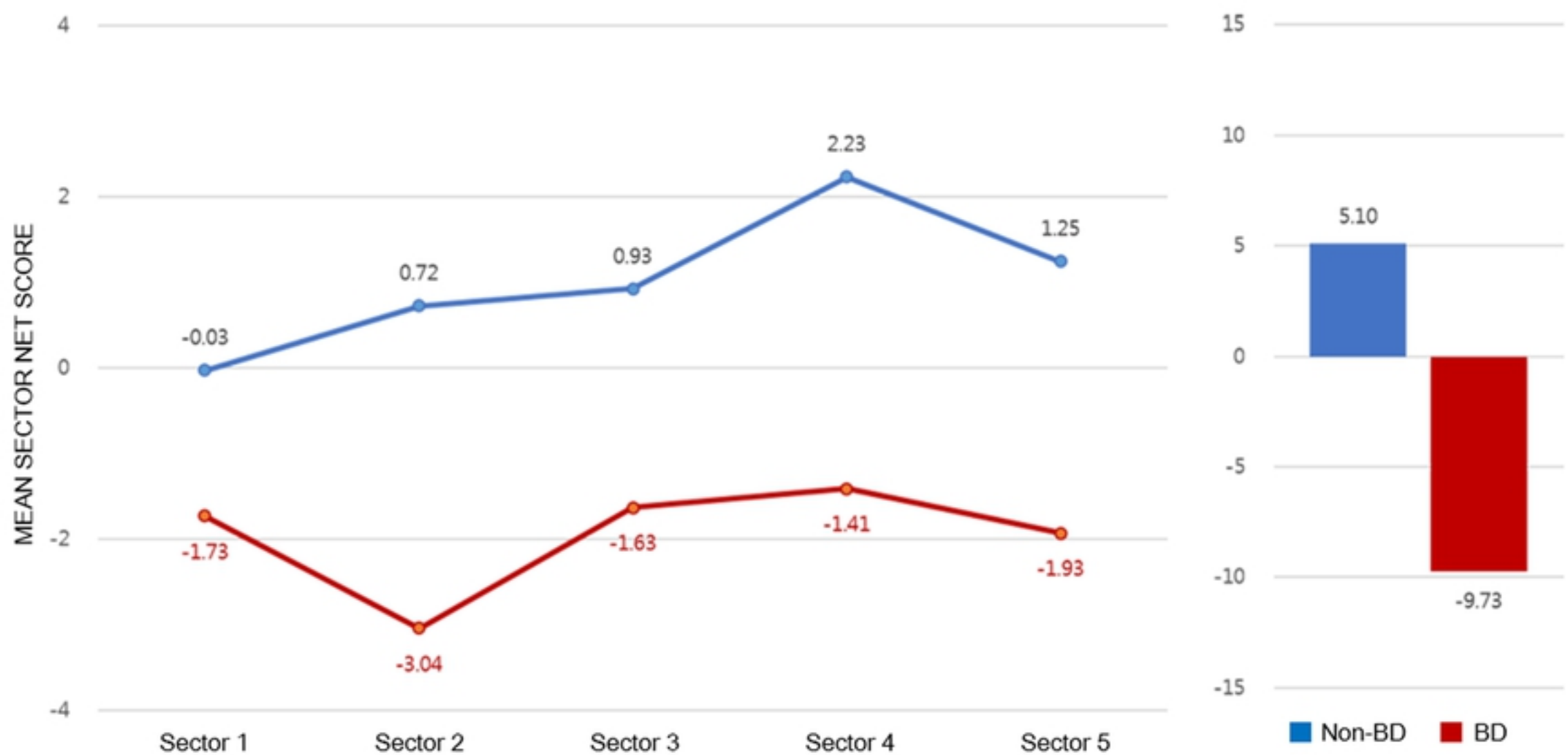
Gain



Loss

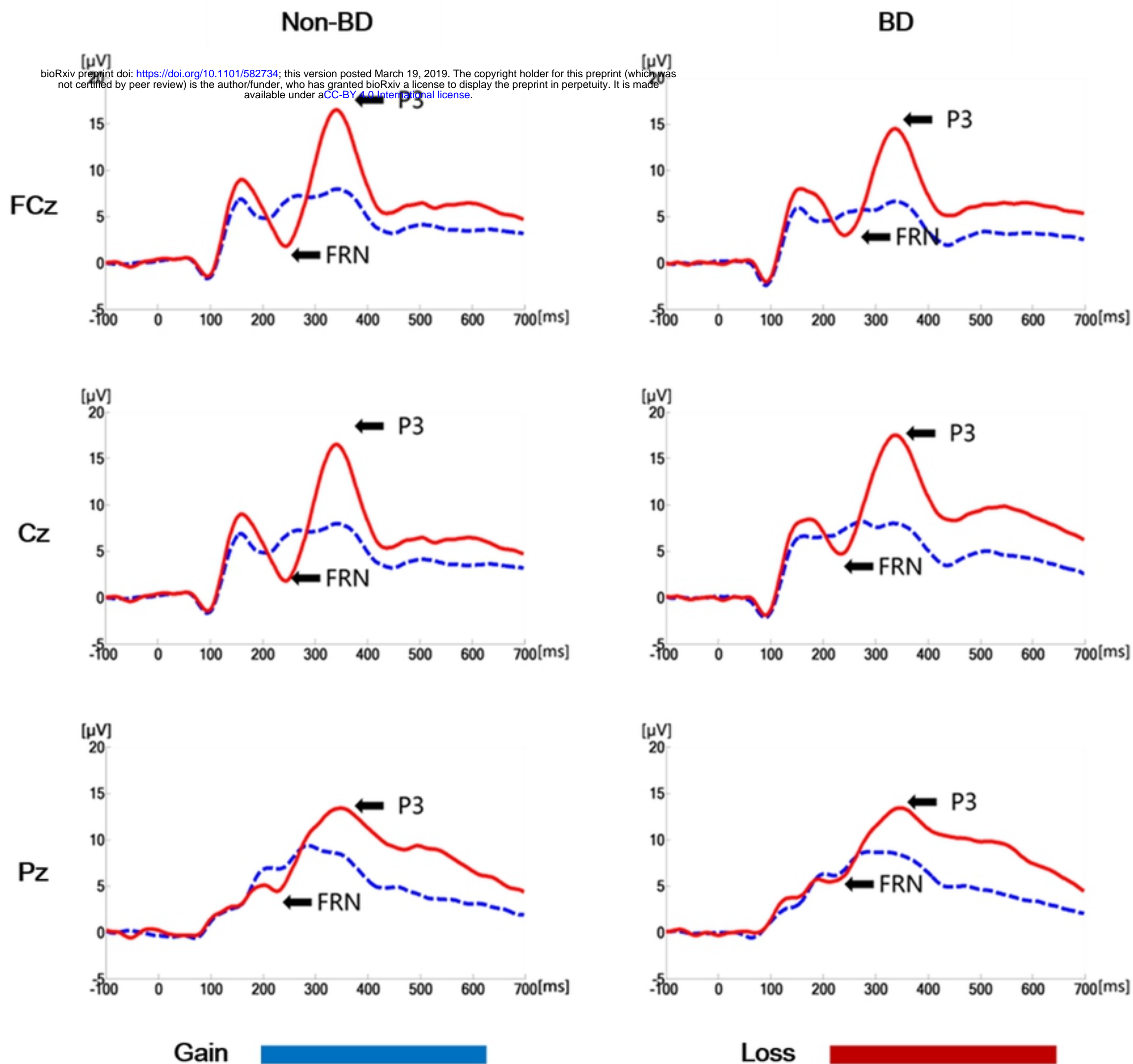
B)

Figure

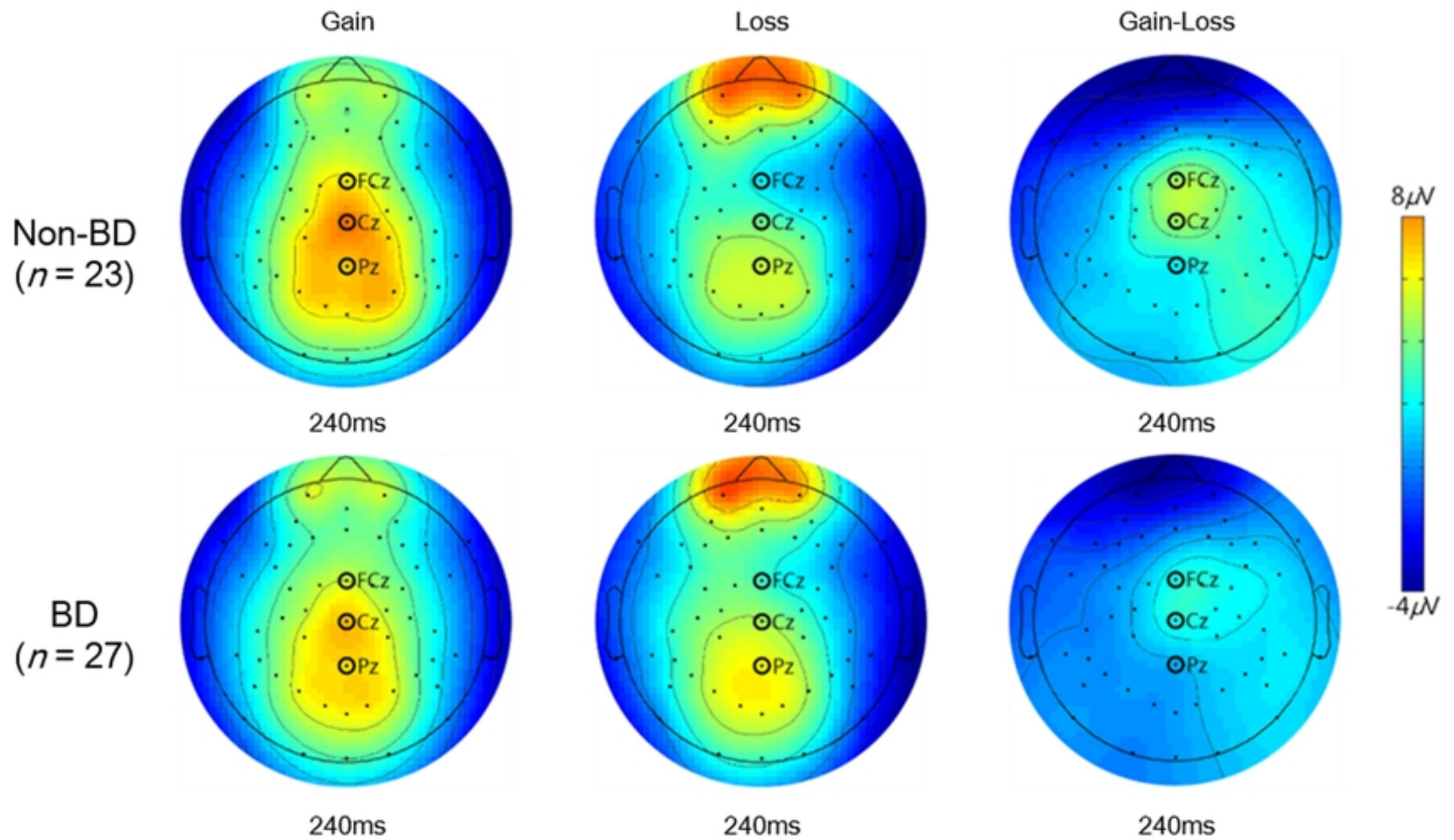


Figure

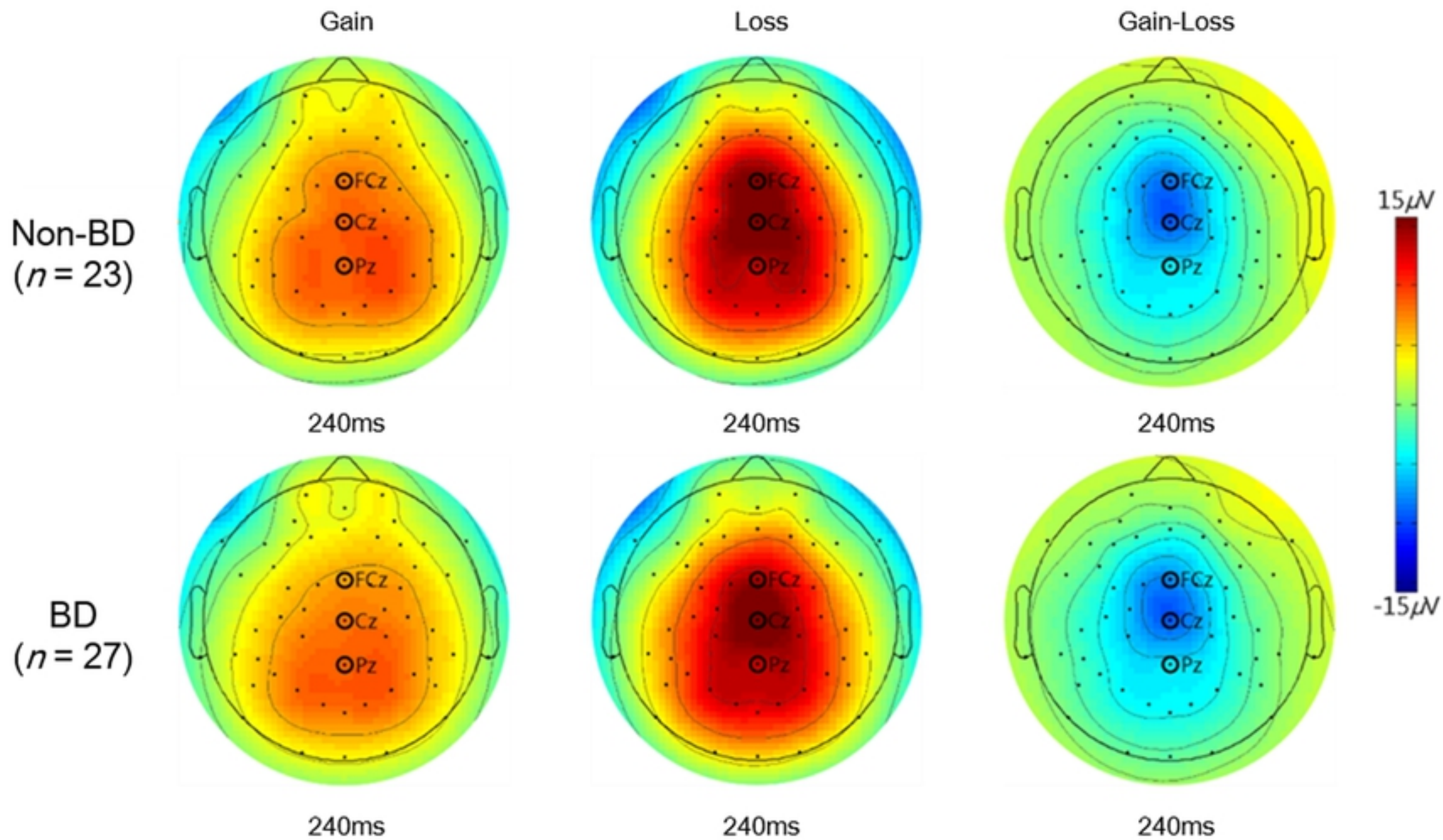




Figure



Figure



Figure