Hypersensitivity to Reward in Problem Gamblers

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Background: Recent research has begun to examine the neurophysiologic basis of pathological gambling. However, direct evidence of a behavioral deficit and an accompanying neurofunctional deviation in a realistic gambling context such as Black Jack has not yet been reported.

Methods: Electroencephalogram was recorded while 20 problem gamblers and 21 control participants played a computerized version of Black Jack. Participants were asked to decide at point scores between 11 and 21 whether they wanted to take another card ("hit") to arrive closer to 21 than the opponent (simulated by computer) or not to take another card ("sit") to avoid going over 21 ("bust").

Results: At a critical point score of 16, problem gamblers decided more often to hit despite losses due to a bust on the preceding trial, whereas control participants decided more often to sit under these conditions. Furthermore, problem gamblers showed more reward-related positive amplitudes in the event-related brain potential than control participants after successful hit decisions at 16.

Conclusions: Here we provide experimental evidence for high-risk taking behavior in gamblers and its correlate in event-related brain potentials. Our results suggest that high-risk-taking behavior in problem gamblers is associated with an increased reward-related neural response to infrequent successes of this behavior.

Key Words: Addiction, anterior cingulate cortex, Black Jack, decision making, problem gambling, risk

The pathological gambler is a high risk taker. Infrequent wins are accompanied by frequent losses, yet the pathological gambler persists despite negative consequences. In learning theory terms, such persistence may be attributed either to insensitivity to punishment associated with frequent losses or to hypersensitivity to the reward associated with infrequent wins. Accordingly, it has been suggested that the midbrain dopamine system, as a central reinforcement mechanism, plays an important role in pathological gambling (1,2,3–5).

The event-related brain potential (ERP) between 250 and 350 ms after a feedback stimulus is believed to be sensitive to the arrival of dopamine signals in the anterior cingulate cortex (6). Unexpected punishing events such as negative performance feedback and monetary losses lead to decreases in the activity of midbrain dopamine neurons (7), resulting in a negative potential at the scalp (6,8,9). In contrast, unexpected rewarding events such as positive performance feedback and monetary gains lead to increases in the activity of midbrain dopamine neurons, resulting in relatively positive scalp amplitudes (10). It has recently been proposed that the negative potential represents the default response and that this response is modulated by the positive deflection associated with rewarding events (11).

We used these brain potential measures to evaluate the response of 20 problem gamblers and 21 normal control participants to critical events in a computerized version of Black Jack (12,13). Participants referred to as "problem gamblers" could be classified as "pathological" according to two of the three measures used to define the gambling status (for details, see Supplement 1). The players started with a point score between 11 and 21 and then decided whether to "hit," that is, to take another card (value between 2 and 11) to approach a score of 21, or to "sit," that, is not to take another card in the hope of beating an opponent with the current score. If the additional card increases the score beyond 21, then the player loses ("busts"). Decisions to hit at increasing scores become increasingly risky as the probability of a bust increases. In previous research, we showed busts elicited more negative ERPs than "no-busts" and that high-risk hit decisions elicited negative ERPs and activated anterior cingulate cortex (12,13). We also showed that control participants hit on 50% of the trials with scores of 16. This behavior is high-risk because, for scores of 15 and higher, taking an additional card is associated with a lower expected value than sitting at the current score. Therefore, we focused on the decision making of problem gamblers and control participants at scores of 16 by computing the probability of hitting or sitting at this score. Furthermore, because gamblers tend to persist in gambling in the face of losses, this analysis evaluated these probabilities as a function of the outcome of the previous trial (bust or no-bust) with the same score. In addition, we evaluated the ERPs following hit decisions at scores of 16 as a function of the outcome (bust or no-bust).

Methods and Materials

Participants

Forty-three male participants were recruited from the student population of the Friedrich-Schiller-Universität and the University of Applied Sciences at Jena (21 problem gamblers; mean age: 23.00 years, SD ± 3.2 years; 22 control participants; mean age: 23.54 years, SD ± 4.5 years). Participants were initially selected from a group of 529 respondents on the basis of responses to the Short Questionnaire for Gambling (Kurzfragebogen zur Glücksspielsucht, 14). The 30 highest scorers were assigned to the provisional problem gambler group, and the lowest 30 scorers were assigned to the provisional control group. Final selection was based on a diagnostic interview (see Supplement 1 for more details). The problem gamblers (PGs) had to meet the DSM-IV-TR criteria for pathological gambling. Some also fulfilled criteria of specific phobia (three persons), social phobia (four persons), and alcohol abuse (three persons). Control participants

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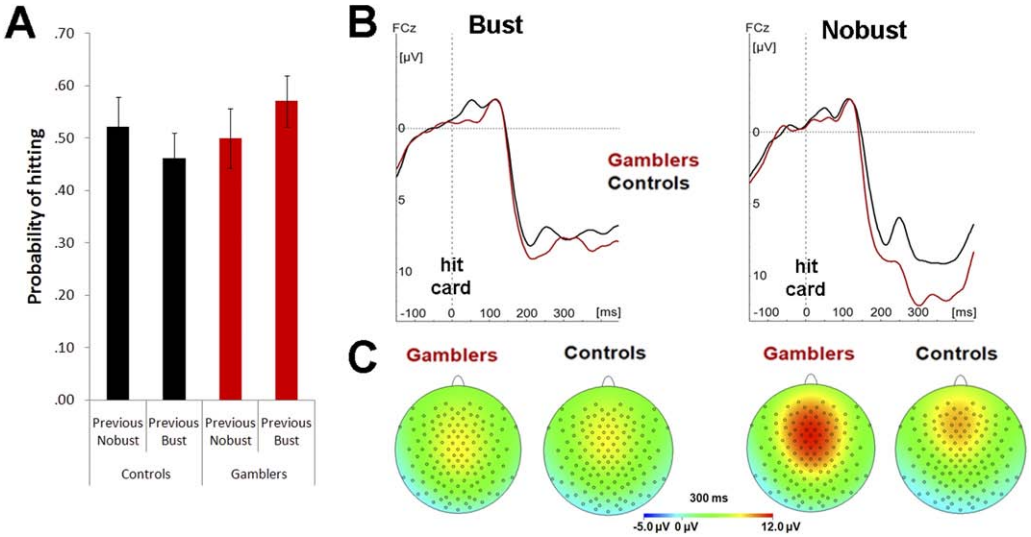

Figure 1. Behavior and electrophysiologic responses of problem gamblers (gamblers) and normal control participants (controls) in a Black Jack task. **(A)** Probability of hitting (taking another card) at a point score of 16 after a previous "bust" or "no-bust" trial for the two groups. **(B)** Event-related brain potential responses from electrode FCz for hit trials at a point score of 16, which led to either a bust or no-bust. Time 0 represents the time at which the hit card was presented. **(C)** Topography of the scalp-recorded activity at 300 ms after presentation of the hit card for the two groups (gamblers and control participants) and the two outcomes (Bust and No-bust).

were selected to match the PGs on age, sex, and handedness. They were free of any active Axis I disorder, except for two participants who had a specific phobia. After the experiment, both PGs (Mean 3.81) and control participants (Mean .88) completed a German version of the South Oaks Gambling Screen (15). The difference between the PGs and control participants was significant ($p < .001$), confirming the original group assignment.

Procedure

All participants provided written informed consent to take part in the study, which was approved by the local ethics board. After receiving verbal instruction on the rules of the game, participants performed the German version of Black Jack called Seventeen and Four (for details, see Supplement 1 and [13]). All participants were paid €6 per hour for participation, plus an extra bonus that varied between 0 and 27.50 EUR according to the outcome of their wagering. Participants received an average bonus of €8.37 (SD ± 7.0) for successful trials (no significant difference between groups; $p = .33$).

EEG Recording and Quantification

Electrodes were applied for the measurement of the electroencephalogram (EEG) and the electro-oculogram (EOG) using the Easy-Cap electrode system (Falk Minow Services, Munich, Germany) and a 128-channel AC amplifier (input impedance: 10 MΩ). In this cap, 125 electrodes are arranged according to the 10-10 system, with the remaining electrodes being located at equal distances between these electrodes. EEG waveforms were averaged separately for each participant, each electrode, and for busts and no-busts at a point score of 16. For each person and each condition, we computed mean amplitudes at FCz in a time window from 270 to 320 msec after presentation of an additional card that led to a bust or a no-bust. For the analysis of these ERP amplitudes, an analysis of variance of group (Gamblers, Control Participants) by outcome (Bust, No-bust) was used. The data of two participants were excluded because of missing data. For more details concerning the EEG methods, see (Supplement 1 and [13]).

Results

We first analyzed decision-making behavior at a point score of 16. After a previous no-bust trial, both problem gamblers and control participants hit at 16 with a probability of approximately .5 (Figure 1A). However, following a bust on the previous trial, the probability of hitting decreased for control participants (.45) but increased for gamblers (.57). The interaction of group by previous outcome was significant: $F(1,39) = 6.54$, $p = .015$. Thus, the gamblers made more risky decisions after a bust ($p < .05$), consistent with the "gambler's fallacy" that a trial following a bust is not likely to be another bust. In contrast, control participants became more conservative after a bust ($p < .05$).

To determine whether the gamblers' behavior could be attributed to insensitivity to losses (busts) or hypersensitivity to rewards (no-busts), we compared the ERPs of gamblers and control participants following busts and no-busts after a hit at a score of 16 (Figure 1B). The groups did not differ in response to busts. However, in comparison to control participants, the gamblers' response to the presentation of a card that led to a no-bust was characterized by a much more positive potential at 300 msec after the presentation of the card. The interaction of group by outcome was significant: $F(1,39) = 5.07$, $p = .030$. The difference between the groups with respect to the positive potential following no-busts was significant at $p < .05$. The topography of these effects, shown in Figure 1C, indicates that this positive potential has a frontocentral distribution (11) and can be modeled by a dipole in the anterior cingulate cortex (ACC; Supplement 1). The difference between bust and no-busts was significant for PGs ($p < .001$) but not for control participants ($p = .322$).

Finally, there was a significant correlation (.36, $p = .023$) between the level of risk taking (probability of hitting at point score of 16) and the difference in amplitude of the positive ERP component between no-busts and busts at a point score of 16.

Discussion

These data suggest that problem gamblers take risky decisions in the face of prior losses, a form of deviant behavior that resembles that exhibited by problem gamblers in real life. This deviant behavior is associated with a hypersensitive response to rewards (no-busts) rather than to an insensitivity to negative events (busts).

The hypersensitive neural response to unexpected rewards (not busting after "hitting" at a score of 16) is manifested in a positive ERP component with a latency of 300 msec. The frontocentral distribution of this component indicates that it is not the classic parietal maximum P3 or P300. This component has been associated with activity in the medial frontal cortex, specifically the ACC, and with phasic increases in dopamine (10,11,16). Furthermore, it has been proposed that this neural response is elicited when events are better than expected, and it has been linked to reinforcement learning (6,17) and decision making of the kind observed in our task (10,11). In this context, it is interesting that the dopaminergic system has been proposed to be related to gambling (2,4,18–20).

Given this association among dopamine, the positive ERP component, reward, and gambling, our results suggest that the problem gambler's persistent risk taking might be due to a hyperactive dopaminergic response to infrequent rewards and an associated overevaluation of such positive outcomes. In particular, the motivation for the high risk taking in problem gamblers may be attributed to the hyperactive response to successful risk taking.

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Supplementary material cited in this article is available online.

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