

Problem gamblers are hyposensitive to wins: An analysis of skin conductance responses during actual gambling on electronic gaming machines

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Abstract

Physiological arousal is purportedly a key determinant in the development and maintenance of gambling behaviors, with problem gambling conceptualized in terms of abnormal autonomic responses. Theoretical conceptualizations of problem gambling are discordant regarding the nature of deficit in this disorder; some accounts posit that problem gamblers are hypersensitive to reward, and others that they are hyposensitive to reward and/or punishment. Previous research examining phasic electrodermal responses in gamblers has been limited to laboratory settings, and reactions to real gaming situations need to be examined. Skin conductance responses (SCRs) to losses, wins, and losses disguised as wins (LDWs) were recorded from 15 problem gamblers (PGs) and 15 nonproblem gamblers (NPGs) while they wagered their own money during electronic gaming machine play. PGs demonstrated significantly reduced SCRs to reward. SCRs to losses and LDWs did not differ for either PGs or NPGs. This hyposensitivity to wins may reflect abnormalities in incentive processing, and may represent a potential biological marker for problem gambling.

Descriptors: Electronic gaming machine, Skin conductance response, Problem gambling, Loss disguised as win (LDW), Arousal, Gambling

Problem gambling (also known as disordered or pathological gambling) has been reclassified recently as an addictive disorder in the Diagnostic and Statistical Manual of Mental Disorders, 5th ed. (DSM-5; cf. DSM-IV-TR; American Psychiatric Association, 2013) due to its high comorbidity and many shared similarities with substance use disorders (Blaszczynski, Walker, Sharpe, & Nower, 2008). It is characterized by continued harmful patterns of gambling activity despite severe personal and interpersonal consequences, and is associated with high rates of depression and suicide (Raylu & Oei, 2002).

Several theories attempting to explain problem gambling behaviors highlight abnormal psychophysiological reactions to reward and/or punishment as a major determinant in the development and maintenance of this disorder (e.g., Blaszczynski & Nower, 2002; Blum et al., 1996, 2000; Damasio, 1994; Goldstein & Volkow, 2002; Sharpe & Tarrrier, 1993). Specifically, such theories propose that characteristic behaviors of problem gambling stem from either a hypersensitivity to reward, or a hyposensitivity to reward and/or to punishment. Behavioral (e.g., Brown, 1986;

McConaghy, 1980; Zuckerman, 1979) and cognitive-behavioral (e.g., Blaszczynski & Nower, 2002; Sharpe, 2002) models of gambling behavior implicate autonomic arousal, perceived as the excitement associated with gambling, as fundamentally appealing and a (possibly *the*) major reinforcer for the gambler. Sharpe and Tarrrier (1993) posit that problem gamblers cognitively appraise rewarding outcomes as more significant due to conditioning that occurred during previous encounters with gambling activity, and they should therefore demonstrate greater increases in physiological arousal following positively valenced (i.e., win) outcomes. The state of optimal functioning theory (McConaghy, 1980) postulates that problem gamblers are chronically hypoaroused and engage in harmful gambling behaviors in order to achieve a normal level of functioning. Biological hedonism models (e.g., Zuckerman, 1979) propose that individual differences in personality mediate the propensity to seek out reward or to avoid punishment. Further empirical research on how problem gamblers respond to positively and negatively valenced stimuli is required to ascertain which of these conceptualizations of problem gamblers are correct, and to ultimately determine the nature of deficit in this disorder. Electrodermal activity has proven to be a reliable indicator of autonomic and cortical arousal (Barry, 1996; Barry et al., 2004; Boucsein, 1992; Lykken & Venables, 1971; Raskin, 1973); however, most gambling studies have examined changes in heart rate (HR) as the primary index of arousal (e.g., Anderson & Brown,

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1984; Coventry & Hudson, 2001; Krueger, Schedlowski, & Meyer, 2005; Ladouceur, Sevigny, Blaszczynski, O'Connor, & Lavoie, 2003; Meyer et al., 2000, 2004), a variable shown to be a better indicator of vigilance and task performance (Barry, 2006; Tremayne & Barry, 2001).

To determine whether problem gamblers are abnormally sensitive to outcomes of varying incentive value, the responses that accompany instances of reward and punishment must be examined; however, the majority of previous autonomic research has examined the effects of gambling on tonic arousal levels over extended periods of time, either comparing levels before, during, and after play (e.g., Carroll & Huxley, 1994; Coulombe, Ladouceur, Desharnais, & Jobin, 1992; Griffiths, 1993; Meyer et al., 2000, 2004), or over long periods of gambling (e.g., Coventry & Norman, 1997; Dickerson, Hinchey, England, Fabre, & Cunningham, 1992; Sharpe, 2004). This type of tonic research has generally found that gambling activity increases arousal, particularly when winning (Coventry & Constable, 1999; Coventry & Hudson, 2001; Sharpe, 2004) and during gambling in naturalistic settings (Anderson & Brown, 1984; Diskin, Hodgins, & Skitch, 2003) for both problem and nonproblem gamblers. Although such research has provided valuable insights into the arousing nature of gambling activities, several problems with this approach are apparent. For example, because tonic levels are recorded over relatively long periods of gambling activity, the effect of individual win and loss events cannot be accurately determined, but are nevertheless likely to influence arousal. Furthermore, it remains unclear whether the increases in arousal during gambling reported in previous tonic research were due to the actuality of winning, or the excitement caused by gambling activities in general (and, by extension, the mere possibility of winning). Individual differences on tonic measures are more susceptible to a range of confounding influences, including social interactions, the consumption of legal (e.g., caffeine, nicotine, alcohol) and illegal drugs, physical movements, and the context in which the outcomes are experienced (e.g., the amount wagered, the presence of features on electronic gaming machines, i.e., bonus free spins/games, second screen games, scatters, or substitutes that generally result in larger amounts of money/credits being returned to players). Comparisons between groups are also difficult due to different behaviors and practices of problem gamblers, such as wagering larger amounts of money, consuming greater amounts of alcohol and cigarettes, and/or spending greater amounts of time gambling compared to nonproblem gamblers (Blaszczynski, Sharpe, & Walker, 2001).

In order to overcome these problems, and to allow an investigation of the theoretical conceptualizations of problem gambling, the current study sought to examine the phasic skin conductance responses (SCRs) immediately following individual win and loss outcomes in an ecologically valid setting. Although scarce, previous studies that have taken this phasic approach have demonstrated that the reactions to gambling outcomes are sufficiently robust to be reliably captured and quantified, and generally report greater responses following wins compared to losses (Dixon, Harrigan, Sandhu, Collins, & Fugelsang, 2010; Goudriaan, Oosterlaan, de Beurs, & Van den Brink, 2006; Lole, Gonsalvez, Blaszczynski, & Clarke, 2012; Wilkes, Gonsalvez, & Blaszczynski, 2009). Even fewer studies have examined these reactions in problem gamblers. [Goudriaan et al. \(2006\)](#) reported that, while healthy controls displayed decreased HR after losses and increased HR following wins, problem gamblers demonstrated a decrease in HR following both win and loss outcomes during play on the Iowa gambling task (Bechara, Damasio, Damasio, & Anderson, 1994). SCRs following

wins or losses were not found to differ for either problem gamblers or healthy controls. The authors of that study interpreted these findings as indicative of a reward hyposensitivity in problem gambling. However, these results require further validation, as studying gambling behavior in artificial laboratory environments can engender a number of problems (Anderson & Brown, 1984). By definition, gambling involves placing a wager on an unpredictable outcome, in which the result of the gamble reflects an element of chance (Bolen & Boyd, 1968). For ethical reasons, participants in laboratory-based studies are usually not permitted to gamble with their own money, and there is no (or, at best, limited) potential to win large amounts of money compared to when gambling in a casino or club setting. Similarly, if participants need to reach a certain threshold of credit amounts before they receive a reward, their level of excitement may be quite low if they know that they are unlikely to reach this threshold. Alternatively, if they believe that they can only catch up by taking risks they would not normally take, they may become unrealistically excited.

Another important factor for studies where participants wager freely assigned credits relates to the nature of negative outcomes; participants may perceive loss outcomes as merely nonrewarding, rather than punishing, since they are not actually losing their own money. Thus, motivations and the extent to which the task resembles real gambling activity are likely to influence responses (Anderson & Brown, 1984). In order to develop an ecologically valid account of problem gamblers' responses to reward and punishment, the current study investigated the physiological reactions that occur during actual gambling activity on electronic gaming machines (EGMs, also known as poker or slot machines) in licensed gaming venues when participants wagered their own money. EGM gambling is of particular clinical significance to this population, as a high proportion of individuals seeking treatment for gambling report addiction to this gambling medium (Dowling, Smith, & Thomas, 2001), and this form of gambling is associated with a faster progression of addiction (Breen & Zimmerman, 2002), as well as more severe symptoms ([Petry, 2003](#)).

In addition to wins and losses, losses disguised as wins (LDWs), outcomes in which the amount returned is less than that wagered, are a key outcome experienced during EGM gambling. These outcomes are accompanied by visual and auditory feedback similar to that triggered by wins (in contrast, flashing visual or auditory feedback are absent in response to a loss), and are estimated to constitute up to 18% of all outcomes (often outnumbering wins) ([Dixon et al., 2010](#)). Novice gamblers have been shown to display similar physiological reactions to LDWs as they do to true wins, suggesting that these outcomes are a design feature of EGMs that contribute to continued play despite overall loss of money ([Dixon et al., 2010](#); see also Clark, Lawrence, Astley-Jones, & Gray, 2009; Luo, Wang, & Qu, 2011; Qi, Ding, Song, & Yang, 2011).

The current study sought to investigate the immediate physiological responses of problem gamblers to EGM outcomes experienced while they gambled with their own money in an actual club environment, and whether these responses differ from the responses of experienced nonproblem gamblers. Following the findings of previous literature (Dixon et al., 2010; Goudriaan et al., 2006; Lole et al., 2012; Wilkes et al., 2009), we expected greater SCRs following wins compared to losses for nonproblem gamblers. Because theoretical conceptualizations regarding the significance of reward in problem gambling are conflicting (e.g., some support the notion of reward hyposensitivity, whereas others suggest a hypersensitive response to reward in these individuals),

the current study investigated which account of the nature of deficit in this disorder is supported by examining the psychophysiological responses of these individuals that occur during actual gambling activity. Based on the assumption that wins are more motivationally significant than losses, and that LDWs are perceived to be like wins (Dixon et al., 2010), we predicted that wins and LDWs would elicit greater SCRs than loss outcomes for experienced nonproblem gamblers. Moreover, because these outcomes have been suggested to contribute to the development and maintenance of problem gambling behaviors on EGMs (Clark et al., 2009; Dixon et al., 2010), we predicted that problem gamblers would be more responsive to LDWs than nonproblem gamblers.

Method

Participants

The current study is part of a larger ongoing program of research examining reactions to stimuli that occur during real EGM play between problem and nonproblem gamblers recruited from licensed gaming venues. Signs inviting patrons to participate in the study were posted in the venue. Individuals wishing to participate approached the researcher seated at a small table near the gaming area.

Data were recorded from 34 nonproblem gamblers (NPGs), and 22 problem gamblers (PGs). Of these participants, 11 NPGs and 7 PGs experienced a “feature” during the study. Preliminary analyses showed that experiencing a feature during the course of EGM play increases tonic arousal levels over an extended period of time. Features on EGMs are triggered by the attainment of a particularly desirable combination of symbols and typically involve the presentation of a series of free trials over a period of time lasting up to several minutes. The outcomes that occur within such periods are presented in rapid succession and are generally associated with auditory and visual stimuli similar to those that accompany wins. Such outcomes are automatically generated and occur independently of the gambler’s actions; thus, the win outcomes experienced within a free reel spin feature are not comparable to normal wins since they are not associated with betting activity. The attainment of a feature signals an increased likelihood of large wins and greater credits returned, since the wins that occur during such periods can be multiplied depending on the individual gaming machine (for example, winnings that occur on normal win trials can be doubled or tripled during a feature). Moreover, the nonwin trials that occur within features cannot be considered true loss outcomes since the player’s own credits are not actually used and lost (hence, free spin). Thus, participants who experienced features were excluded from the current analyses; their data will be analyzed and reported elsewhere once a sufficient sample is obtained. Eight of the individuals classified as NPGs experienced fewer than five epochs for at least one outcome type, and were also excluded from the final analysis. Accordingly, 15 problem gamblers (10 male, 5 female; $M_{age} = 34.17$ years, $SD = 13.40$; age range 18–70 years) and 15 nonproblem gamblers (9 male, 6 female; $M_{age} = 40.18$ years, $SD = 20.64$; age range 18–70 years) were included in the current analyses. All participants were of Caucasian European or Asian heritage. Written informed consent was obtained from all participants prior to their involvement in the study; they were advised that participation was entirely voluntary and that they could withdraw from the study at any time. The University of Wollongong Human Research Ethics Committee approved the research protocol.

Materials

Recording equipment. The Ambulatory Monitoring System (model AMS5fs; Groot, de Geus, & de Vries, 1998) was used to record electrodermal activity. Two sintered silver/silver-chloride (Ag/AgCl) electrodes (outer diameter: 1.5 cm, inner diameter: 0.8 cm) were filled with an inert 0.05 M NaCl electrolyte cream, and placed on the volar surface of the medial phalanx of the third and fourth digits of the nondominant hand, which were cleaned using 70% isopropyl alcohol wipes. Skin conductance was recorded at a constant voltage of 0.5 V, and sampled at 10 Hz (0.1 s intervals).

Measure of gambling behavior. The Problem Gambling Severity Inventory (PGSI) of the Canadian Problem Gambling Index (Ferris & Wynne, 2001) was designed to measure general population prevalence rates for problem gambling. Participants are required to answer nine questions that assess their ability to control their gambling behaviors (e.g., “Have you bet more than you could really afford to lose?”), and the frequency (i.e., *never, sometimes, most of the time, or almost always*) they experienced health-related, financial, and/or psychological problems (e.g., “Has gambling caused you any health problems, including stress or anxiety?”) in the previous 12 months as a result of their gambling activity. This measure was used in the current study to categorize individuals as problem gamblers (score of 8 or higher, to a total maximum score of 27) or nonproblem gamblers (score below 8). This cut-off score has been shown to reliably identify the gambler’s diagnostic status based on DSM-IV criteria and clinical assessment interviews (Ferris & Wynne, 2001). Since it provides a means of identifying problem gamblers in a quick, confidential, and anonymous manner (thus, avoiding the problem of symptom under-reporting commonly associated with socially undesirable behaviors, e.g., Sudman, 2001), this quantitative self-report measure was chosen for use in the current study.

Procedure

After providing informed consent, participants were fitted with the recording equipment. Apart from being asked to keep movement to a minimum, participants were instructed to play on an EGM of their choice as they typically would for as long as they desired. During play, the researcher stood behind them and, when each event (win, loss, LDW, start of a feature) occurred, discreetly pressed a remote event marker (four different buttons on a small oval pad, size of a car key) that inserted a mark at the appropriate place on the physiological recording. When wins and LDWs occur on EGMs, the machine gradually accumulates credits for that particular spin (separate auditory stimuli are also presented for as long as the credits “climb”); losses were identified by the researcher immediately after they occurred, whereas wins and near-wins could only be recorded as such once it was determined whether the amount returned exceeded the threshold of the amount bet (therefore, the physiological effects associated with experiencing these outcomes would be likely to start earlier than when the event was actually finally defined and marked by the researcher; see Figure 1B).

Ethics guidelines allowed the researcher to record but not to promote gambling in any manner (e.g., by setting a uniform start total or bet amount). Thus, participants were in total control of the amount of time and money spent during the session, and the amount bet on each trial (i.e., the amount wagered was not held constant). Participants determined completion of the session of

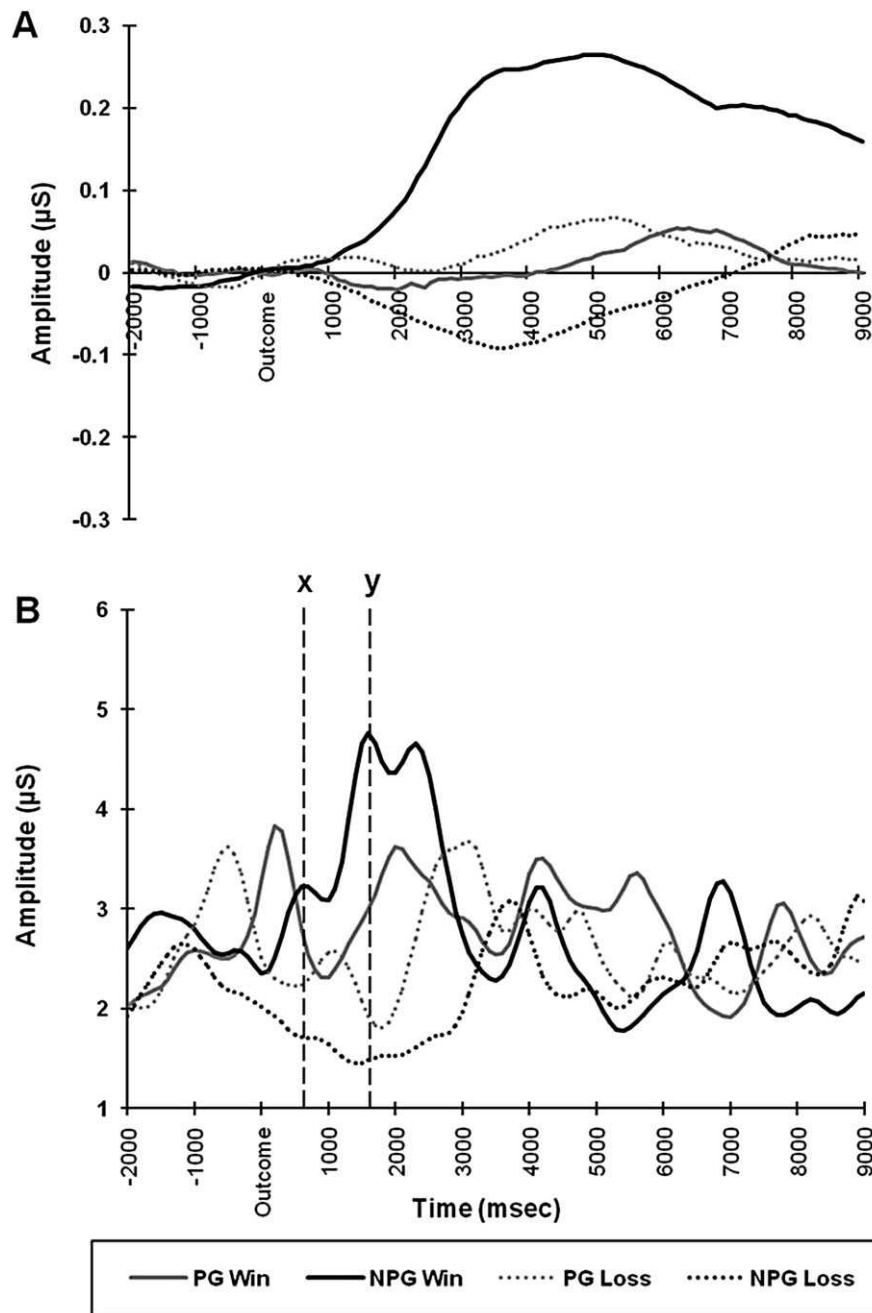


Figure 1. Mean skin conductance response (SCR) following wins and time-matched losses for the problem gambler (PG) and nonproblem gambler (NPG) groups. A: Raw grand-average SCR waveforms for the time-matched epochs for each outcome type. B: Driver data, as calculated by LedaLab, for each outcome type (note the different scales for panels A and B); the vertical dashed line, labeled “x,” indicates the response likely caused by the accumulation of credits before a threshold of recognition for a win, whereas line “y” most likely indicates the peak response elicited by processing the significance of the actual win.

play and, accordingly, advised the researcher of their intent to discontinue gambling or the testing phase. Upon completion of play, participants completed the PGSI and were given a bistro voucher (valued at 40 AUD) for use within the gaming venue in appreciation of their time.

Data Extraction and Analysis

The raw electrodermal data were epoched offline in order to isolate each individual outcome from the continuous data trace. These

epochs included a 2 s period pre-event and a 9 s period postevent. The time of occurrence for each outcome was adjusted by 1.0 s to compensate for the delay in the researcher’s reaction time, as estimated by a separate computer program. A similar procedure and correction has been employed in previous studies (Wilkes et al., 2009).

Since EGMs allow a rapid succession of bet placement (every 3 to 6 s), SCRs from consecutive events frequently overlapped. Traditional data extraction methods, which examine trough-to-peak differences, have been shown to underestimate SCR amplitudes in

paradigms with short interstimulus intervals due to distortion caused by the recovery slope of preceding responses (Boucsein, 1992). In order to overcome this problem, the data were analyzed using LedaLab software (version 2.10; Benedek & Kaernbach, 2010). Based on the assumption that sudomotor nerve bursts (which underlie skin conductance responses) are characterized by a distinct and compact period of activity and that, theoretically, the activity of these nerves cannot be negative, this program uses biexponential algorithms to decompose overlapping SCRs in a four-step process that is repeated a number of times (in this case, three times) to ensure the data are optimized and to increase the goodness of fit of the model. The discrete decomposition analysis performed on each individual trial calculates the amount of electrodermal activity caused by tonic skin conductance levels, the sudomotor nerve (i.e., the driver of the SCR), and the remainder signal (i.e., deviations from the standard SCR shape, proposed to be caused by pore opening). SCR amplitude and area under the curve (AUC) measures are then derived from the single, nonoverlapped response (derived by convolution of each impulse using an algorithm that estimates the underlying sudomotor nerve activity based on the shape of the SCR, and adding the remainder activity related to subsequent pore opening processes, if these data are available), and the original skin conductance (SC) data are reconstructed by adding the tonic component. The separation of SCRs from tonic skin conductance level also eliminates the need to adjust for decreasing baseline levels by de-trending individual epochs. This program was set to calculate the sum of all amplitudes, and the total AUC, for any response over 0.01 μS in the 1 to 3 s following each stimulus occurrence.

Once the overlapping SCRs had been processed, each win and each LDW incidence was time-matched with the previous loss outcome (i.e., that occur at comparable points in time). This matching procedure was performed in order to avoid the problem of falling tonic skin conductance levels over the course of the experiment and to give a more accurate representation of the responses to these outcomes. Although the number of epochs varied between individuals, an equal number of win and loss epochs and an equal number of LDW and loss epochs were included in the analyses (as mentioned above, each participant experienced at least five epochs of each outcome type). The amplitude and AUC data for each epoch were averaged together based on outcome type (win, loss, LDW) for each participant. Because a different number of win and LDW outcomes (as well as time-matched loss outcomes) were experienced by each participant, the data for these outcomes were subjected to two separate 2 Group (PG, NPG) \times 2 Outcome mixed-design analysis of variance (ANOVA). An independent samples *t* test was performed to assess whether the tonic skin conductance levels, as assessed by the LedaLab program, differed between problem and nonproblem gambler groups.

Results

Group Characteristics and Behavioral Data

The mean PGSI scores for the PG and NPG groups were 15.0 ($SD = 4.4$) and 1.2 ($SD = 1.4$), respectively. Scores for participants in the NPG group ranged from 0 to 3 (47% scored 0, 6% scored 1, 20% scored 2, and 27% scored 3), and scores for the PG group ranged from 8 to 24 out of a total possible score of 27. Independent samples *t* tests revealed that the PG and NPG groups did not significantly differ in the age ($p = .288$) or sex ($p = .716$) of participants. Loss outcomes were experienced most frequently (67.2%

Table 1. Mean (Standard Deviation) SCR Values (μS) of the PG and NPG Groups for Amplitude and Area Under the Curve Measures Following Wins, LDWs, and Losses

	PG (<i>M</i> , <i>SD</i>)	NPG (<i>M</i> , <i>SD</i>)
Group main effect		
Win vs. loss comparison		
Amplitude	.15 (.09)	.28 (.06)
Area	3.15 (1.15)	5.08 (1.10)
LDW vs. loss comparison		
Amplitude	.22 (.06)	.23 (.04)
Area	4.32 (.82)	3.98 (.79)
Outcome \times Group interaction		
Win vs. loss comparison		
Amplitude		
Wins	.14 (.09)	.38 (.08)
Losses	.16 (.06)	.18 (.05)
Area		
Wins	3.03 (1.35)	7.06 (1.43)
Losses	2.99 (1.09)	3.09 (1.08)
LDW vs. loss comparison		
Amplitude		
LDWs	.24 (.06)	.22 (.04)
Losses	.20 (.07)	.24 (.05)
Area		
LDWs	4.12 (.92)	3.84 (.85)
Losses	4.72 (1.95)	4.13 (.95)

SCR = skin conductance response; PG = problem gambler; NPG = nonproblem gambler; LDW = loss disguised as win.

of trials), followed by LDWs (17.7% of trials), and wins (15.1% of trials). On average, each participant experienced 78 losses ($SD = 49$; range = 26–209), 20 LDWs ($SD = 48$; range = 7–54), and 17 wins ($SD = 48$; range = 5–51) within the testing session.

Physiological Data

While tonic skin conductance levels of PGs ($M = 6.05 \mu\text{S}$, $SE = 3.83$) appeared to be slightly higher than NPGs ($M = 5.24 \mu\text{S}$, $SE = 3.42$), this difference was not significant ($p = .544$). Compared to losses, wins elicited significantly greater AUC ($M_{win} = 5.05 \mu\text{S}$, $SE = 1.18$; $M_{loss} = 3.04 \mu\text{S}$, $SE = 1.34$), $F(1,28) = 4.77$, $p = .037$, $\eta_p^2 = .10$, but the difference for SCR amplitudes ($M_{win} = .26 \mu\text{S}$, $SE = .08$; $M_{loss} = .17 \mu\text{S}$, $SE = .04$) failed to reach significance ($p = .057$). No main effect of group was found for either amplitude ($p = .428$) or area measures ($p = .498$). A significant Group \times Outcome interaction revealed that, in the NPG compared to the PG group, wins elicited greater AUC, $F(1,28) = 5.22$, $p = .030$, $\eta_p^2 = .15$, and SCR amplitudes, $F(1,28) = 5.63$, $p = .025$, $\eta_p^2 = .14$, but minimal differences following losses for both groups (Figure 1). The mean SCR values for the group and Group \times Outcome interaction following wins and losses can be seen in Table 1.

Electrodermal activity following LDW outcomes was found not to be significantly different from the activity following losses, in terms of SCR amplitude ($M_{LDW} = .23 \mu\text{S}$, $SE = .04$; $M_{loss} = .22 \mu\text{S}$, $SE = .05$; $p = .328$), or AUC ($M_{LDW} = 3.98 \mu\text{S}$, $SE = .86$; $M_{loss} = 4.42 \mu\text{S}$, $SE = 1.02$; $p = .234$). No main effect of group was found for either amplitude ($p = .229$), or area measures ($p = .189$). The Group \times Outcome interaction for the loss versus LDW comparison was not significant for SCR amplitude ($p = .189$) or AUC ($p = .269$) (Figure 2). The mean SCR values for the group and Group \times Outcome interaction following LDWs and losses can be seen in Table 1.

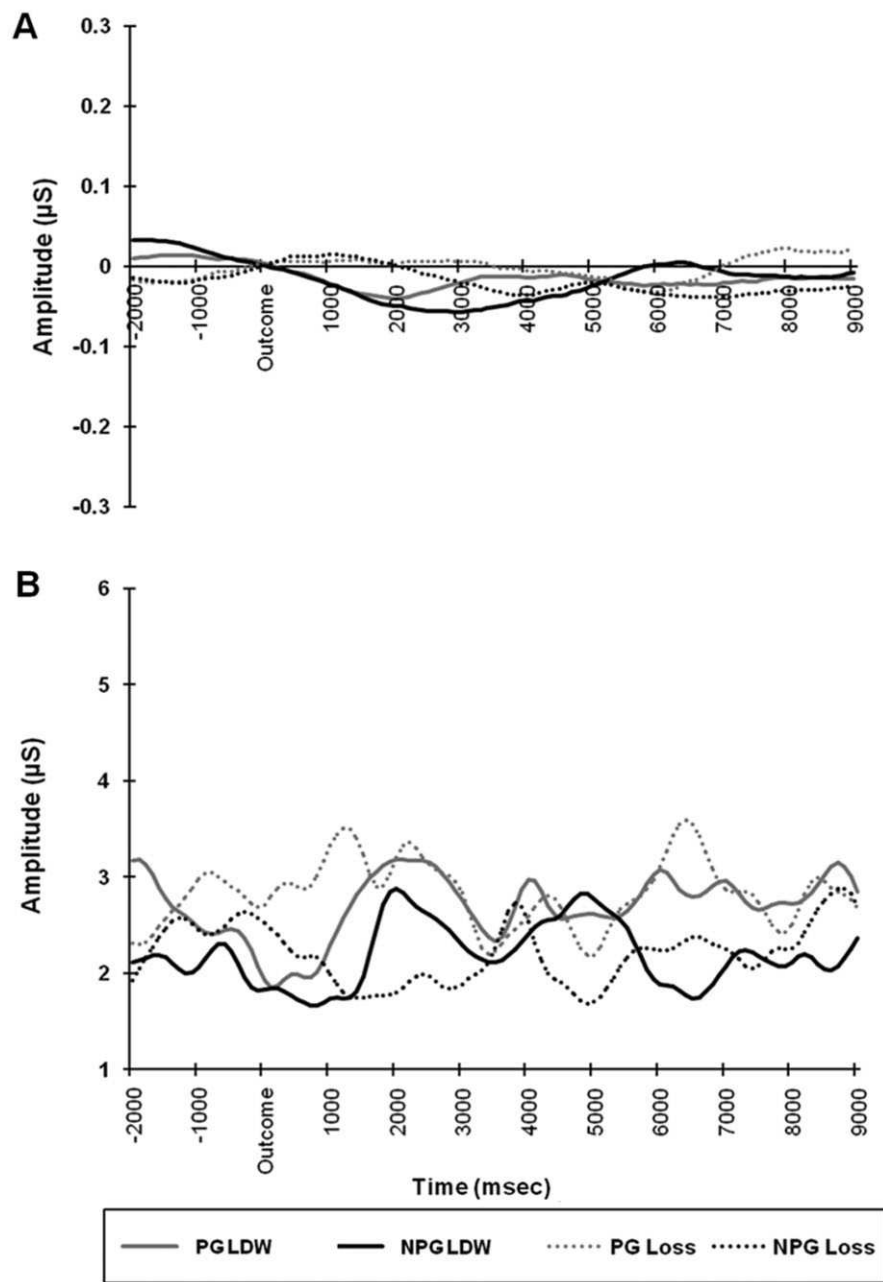


Figure 2. Mean skin conductance response (SCR) following loss disguised as win (LDW) and time-matched loss outcomes for the problem gambler (PG) and nonproblem gambler (NPG) groups. A: Waveforms representing the raw grand-average SCR for the time-matched epochs for each outcome type. B: Driver data, as calculated by LedaLab, for each outcome type (note the different scale for panels A and B).

Discussion

Problem gamblers demonstrated attenuated SCRs to win outcomes, suggesting a hyposensitive response to rewarding stimuli in affected individuals. This finding corroborates previous research using HR as an indicator of arousal (Goudriaan et al., 2006), and previous neuroimaging research showing evidence for reduced cortical activity in reward-related brain circuitry of problem gamblers after the experience of reward (de Ruiter et al., 2009; Reuter et al., 2005; cf. Miedl, Fehr, Meyer, & Herrmann, 2010; van Holst, Veltman, Büchel, van den Brink, & Goudriaan, 2012). Also

consistent with previous research (Dixon et al., 2010; Lole et al., 2012; Sharpe, 2004; Wilkes et al., 2009), wins were found to induce larger SCRs than losses in individuals familiar with gambling but who do not report gambling-related problems, highlighting the motivational significance of experiencing reward in EGM gambling. It is unlikely that the attenuated SCRs following losses observed in the current study are due to the frequent occurrence of these outcomes, as reduced responses were also observed following less frequent LDWs. Moreover, the main focus of the current study was to examine the between-group differences in responding during actual gambling activity, which would not be affected by the

frequency of occurrence of outcomes (i.e., problem gamblers and nonproblem gamblers would be expected to experience the same proportion of win and loss outcomes).

The finding of reduced reward sensitivity of problem gamblers corroborates theoretical interpretations given by several neurobiological accounts that implicate impaired reward processing as the basis of problem gambling behaviors (Blum et al., 2000; Damasio, 1994), and has important ramifications for conceptualizations of the nature of the deficit in this disorder. The apparent hyposensitivity to reward in problem gamblers may be caused by malfunctioning in the cortical regions associated with incentive value processing, such as the mesolimbic-dopaminergic reward system (de Ruiter et al., 2009; Holroyd & Coles, 2002; Volkow, Fowler, Wang, & Swanson, 2004), or areas of the brain associated with generating appropriate emotional responses to these outcomes (Damasio, 1994). This response pattern is consistent with previous electrophysiological and neuroimaging research on substance use disorder (e.g., Goldstein et al., 2007, 2008; Kamarajan et al., 2010; Porjesz, Begleiter, Bihari, & Kissin, 1987; for reviews, see Ditcher, Damiano, & Allen, 2012, and Volkow et al., 2004), and is particularly exciting as it suggests these disorders may reflect different manifestations of the same underlying deficit in the reward circuitry of the brain (Blum et al., 1996, 2000; Damasio, 1994). Specifically, reduced functioning/availability of the D2 dopamine (DRD2) minor (A1) allele receptors may lead to less efficient transmission of dopamine following drug ingestion for substance users and the experience of win outcomes for problem gamblers (see Nemoda, Szekely, & Sasvari-Szekely, 2011, and Noble, 2003, for reviews on previous research findings). Consequently, afflicted individuals are hypothesized to experience less pleasure following such rewarding stimuli, potentially explaining the maladaptive behaviors associated with these disorders, including increased reward-seeking behavior (Blum et al., 2000) and/or suboptimal decision making (Damasio, 1994).

The hyposensitive response to reward does not provide support for some arousal-based models of problem gambling; specifically, those that predict problem gamblers evaluate wins as more significant, and will thus show greater physiological reactions to these outcomes (e.g., Sharpe & Tarrier, 1993). This pattern of responding may corroborate other arousal-based theories that posit problem gamblers are hypoaroused and use gambling to achieve an optimal state of functioning (e.g., Brown, 1986; Jacobs, 1986; cf. Cocco, Sharpe, & Blaszczynski, 1992). Taken with our finding that the tonic arousal levels did not differ between problem and nonproblem gamblers on the day of gambling, this reward hyposensitivity could be an aspect of a general state of hypoarousal in this disorder. However, to confirm this, lower tonic baseline measures will need to be demonstrated among PGs on nongambling days.

The precise mechanisms underlying the attenuated response to reward exhibited by problem gamblers could not be determined in the current study and should be the focus of future research. It could be argued that PGs demonstrate attenuated SCRs to wins due to the effects of repeated exposure to gambling activity rather than an inherent hyposensitivity to reward. Specifically, because PGs gamble more frequently, they may have become more accustomed to wins compared to non-PGs and do not perceive them to be as salient as they once did. Further research on the lifetime trajectory of these responses is required in order to determine the extent that overexposure to gambling activity and genetic predispositions contribute to the development of problematic gambling behaviors.

Nevertheless, the attenuated SCRs to rewarding outcomes observed in the current study may be used as a marker for deficit in this disorder if further research verifies it as robust.

The current study was the first to examine the psychophysiological reactions to losses disguised as wins during actual gambling activity in problem and nonproblem gamblers. These outcomes were not found to elicit electrodermal responses that were significantly different to losses in either group. This finding is in contrast with results previously reported by Dixon et al. (2010), who found that SCRs and HR responses to LDW outcomes were comparable with those following wins, which were both significantly different from losses. This discrepancy is possibly due to the fact that their study examined responses only in novice undergraduate gamblers in a laboratory setting, and not experienced gamblers who may have become accustomed to such events. This notion is interesting, as it suggests that the experience of losses disguised as wins is important in the development, but not the maintenance, of gambling behaviors, and that this disorder may have a distinct lifetime trajectory. Specifically, when people gamble for the first few times, they may be more excited by both wins and LDWs, but, as time progresses, they may only be excited by true wins. As mentioned above, further research is required into the influence of repeated exposure to gambling activity on the developmental trajectory of this disorder; in particular, whether PGs have habituated to wins and losses disguised as wins (albeit at differential rates, i.e., responses to LDWs may be habituated to more quickly than wins). Unfortunately, the background gambling history and experience of participants in the study were not explicitly elicited and quantified. In order to minimize disruption to patrons and business within the gaming venue in which the data were collected, only the PGSI was administered, and not the full Canadian Problem Gambling Index. The latter would have given a clearer indication of the experience participants had with gambling. Nevertheless, since nearly half of the NPG group scored 2 or 3 on the PGSI, it is assumed that participants in the current study were more familiar with gambling activity than college students who merely participate in gambling research in return for course credits or other small reward (e.g., participants in the Dixon et al., 2010, study scored either 0 or 1 on this measure).

Since the recording device used in the current study allowed only four different event markers to be inserted into the physiological data record, an examination of psychophysiological responses following different-sized bets or different magnitude outcomes could not be conducted (although it should be noted that participants seldom changed the amount wagered on each trial within the testing session). Because small and large win types were averaged together, the results are likely to represent responses to more frequently experienced small wins. As mentioned above, it is possible that, while problem gamblers appear to be hyposensitive to small wins that occur on EGMs, they may be more responsive to the experience of significantly larger wins and/or bonus features, or, alternatively, they may need to experience larger magnitude wins to feel the same level of excitement as nonproblem gamblers feel toward small wins.

As previously mentioned, wins and LDWs encountered during EGM gambling are associated with the gradual accumulation of credits, whereas losses are quickly identified as such. Because such presentation of loss and nonloss outcomes are a genuine design feature of EGMs, the differential latencies associated with recording these events were not able to be avoided, but are nevertheless likely to influence responses. Future research may choose to

examine the effect that the anticipation of a potential win associated with accumulating credits (as opposed to the identification of the actual outcome) has on psychophysiological responses. Another limitation of the current research was the presence of researchers during the recording process, which possibly influenced participants to gamble differently from how they normally would; however, this effect was constant and unlikely to be responsible for between-event effects observed in the study. To overcome such problems, future research could use a video camera to record the participant gambling, and later match the occurrence of events with the resultant physiological reactions.

Finally, anecdotal evidence has suggested that problem gamblers may be motivated primarily by the experience of bonus features during play. Unfortunately, because not all participants in the original sample experienced these outcomes, there was not sufficient power to allow analysis of the influence of features on normal and abnormal gambling. Future examination of the physiological

responses that occur in response to these outcomes may help to further elucidate the motivations of gamblers on EGMs.

This study is the first to investigate the phasic physiological reactions to gambling outcomes while problem gamblers and nonproblem gamblers wager their own money on EGMs in a real gaming environment. Problem gamblers were found to exhibit attenuated responses to reward, whereas wins were found to elicit greater SCRs than losses for nonproblem gamblers. These findings suggest that a hyposensitivity to reward that may underlie the problematic behaviors characteristic of this disorder, such as gambling with larger amounts of money and for longer periods of time, presumably in order to experience the same excitement and satisfaction as nonproblem gamblers. Responses following LDW outcomes were not found to differ from losses in either group. While further research is necessary to validate these results, the current study highlights the potential value of this apparent hyposensitive response as a biological marker for this disorder.

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