

## Psychophysiological determinants and concomitants of deficient decision making in pathological gamblers

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### Abstract

Psychophysiological responses are considered to be a mediating factor in the development of pathological gambling (PG) and PG has been associated with differential arousal levels during gambling. Yet little is known about the specific psychophysiological responses to wins and losses in PG. This study investigated heart rate (HR) and skin conductance responses (SCRs) during the Iowa Gambling Task (IGT) in an adult PG group ( $n = 46$ ) and a normal control (NC) group ( $n = 47$ ). Anticipatory psychophysiological reactions to disadvantageous and advantageous choices during the IGT and psychophysiological responses to wins and losses were measured. The PG group performed worse than the NC group on the IGT and exhibited lower anticipatory SCRs and HR decreases when pondering choices of disadvantageous card decks during the IGT. The PG group showed a decrease in HR after losses and wins, whereas the NC group showed a decrease in HR after losses, but an increase in HR after wins. Reward and punishment sensitivity as measured by the self-report BIS/BAS scale influenced IGT performance and psychophysiological responses, but in general these effects were similar for the PG group and the NC group. Lower anticipatory psychophysiological responses to disadvantageous choices in PG suggest impaired risk assessment in this group. Absence of a HR increase after wins possibly implies that reward sensitivity is decreased in PG. Because levels of reward and punishment sensitivity were associated with differential anticipatory HR responses to advantageous and disadvantageous decks, it would be advisable to include this taxonomy in studies on psychophysiological responses to rewards and losses.

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### 1. Introduction

Alcohol and substance abuse and dependence, and pathological gambling (PG) have similarities on both the phenotypic and endophenotypic level. Both PG and alcohol and substance dependence are disorders characterized by a lack of self-regulation. Diminished self-regulation is displayed when an addicted person is not able to inhibit the urge for a desired drug or behavior, and to shift his or her behavior from the addictive reinforcement to a less self-destructive reinforcement. Although classified as an impulse control disorder, PG is regarded as

a ‘behavioral addiction’ by some researchers (Blanco et al., 2001; Marks, 1990), and several DSM-IV-TR criteria for PG resemble those of alcohol and drug dependence, such as loss of control, tolerance, withdrawal, and the experience of negative consequences due to the gambling related behavior (Diagnostic and Statistical Manual IV-Text Revision, American Psychiatric Association, 1994).

Moreover, diminished neurocognitive functions have been found in both substance dependence (Bechara et al., 2002b; Bechara and Damasio, 2002a; Fein et al., 2002; Giancola and Moss, 1998; Pau et al., 2002) and PG (Goudriaan et al., 2005; Petry, 2001a,b). Neurobiological similarities between PG and substance dependence have been clearly demonstrated (Bolla et al., 2003; Miguel-Hidalgo and Rajkowska, 2003; Paulus et al., 2002; Potenza et al., 2003; Reuter et al., 2005). However, until now, the study of similarities between addiction and PG with regard to psychophysiological parameters has been very

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limited, partly because a suitable model was not available. The introduction of the somatic marker model has greatly changed this situation on a theoretical level, whereas the development of the Iowa Gambling Task, which is based on the somatic marker model, has provided an ecologically valid measurement tool to be used in empirical studies.

The somatic marker hypothesis (Damasio, 1996) provides a relevant theoretical framework for studying decision making processes and the role of psychophysiological reactions when anticipating rewards or losses in PG. This hypothesis postulates that unconscious bodily states ('somatic markers') guide our behavior. Somatic markers develop initially due to the experience of pleasurable or aversive stimuli (primary inducers), which generate a somatic response, such as a heart rate (HR) or skin conductance response (SCR). After early learning experiences, these somatic markers can be induced by activations of neural pathways that developed due to these earlier experienced stimuli. These activations (secondary inducers) can be elicited in anticipation of a situation which activates the memory or neural activation pattern of the earlier pleasurable or aversive consequence. Thus, somatic markers function to enable automatic or intuitive decision making, which is especially relevant in complex situations, where somatic markers promote decisions, and prevent profligate rational reasoning (Damasio, 1996). The ventromedial prefrontal cortex is thought to play a crucial role for triggering somatic markers activated by secondary inducers (Bechara et al., 1999, 2002b).

The role of somatic markers in behavior has been investigated in a number of studies on decision making, using the Iowa Gambling Task (IGT; Bechara et al., 1994). In the IGT, participants play a computerized card test in which they have to learn to select cards from four decks, differing in long-term net wins. Participants have to learn over the course of the task, to choose the advantageous decks instead of the more risky, disadvantageous decks. Studies have shown that participants develop stronger somatic responses (somatic markers) before selecting cards from the disadvantageous decks, compared to responses before card selections from the advantageous decks. These somatic responses occur even before they have conscious knowledge of the difference between the decks. The development of these psychophysiological responses is thus argued to be essential to advantageous behavioral responses on this task (Bechara et al., 1994, 2000). Patients with ventromedial prefrontal lobe damage and substance dependent individuals show deficits in the generation of somatic markers in anticipation of disadvantageous choices, and perform worse on this task (Bechara and Damasio, 2002a; Bechara et al., 1994, 2001). However, some studies raised doubt on certain assumptions made in the somatic marker hypothesis. For instance, patients with dorsolateral prefrontal brain damage also performed worse on the IGT (Manes et al., 2002) and the assertion in the somatic marker hypothesis that somatic markers precede conscious knowledge in the IGT is challenged (Maia and McClelland, 2004). However, this last study did not investigate physiological responses, and thus, the role of somatic markers in the development of advantageous choices on the IGT is still undisputed.

The first research question focused on the role of somatic markers in the behavioral performance of pathological gamblers on the IGT. We hypothesized that pathological gamblers, over the course of the task, would develop diminished somatic responses preceding disadvantageous choices compared to normal controls, similar to the diminished somatic markers of patients with ventromedial prefrontal brain damage, and individuals with substance dependence.

In addition to the study of anticipatory somatic markers in PG, the study of psychophysiological reactions after experiencing wins or losses is relevant, because diminished psychophysiological responses to positive and/or negative consequences are proposed as factors contributing to the development and continuation of PG and alcohol/substance dependence (Błaszczynski and Nower, 2002; Sharpe, 2002; Clark and Robbins, 2002; Koob and Le Moal, 1997). Despite the importance given to psychophysiological processes in theories on PG, the number of studies on psychophysiological responses after separate wins or losses is relatively small. Psychophysiological reactions in pathological gamblers during actual gambling have been shown to be both higher and lower than those of normal controls (for a review, see: Goudriaan et al., 2004). Two studies examined psychophysiological reactions when imagining wins or losses in pathological gamblers. One of these studies indicated no differences in arousal when pathological gamblers imagined a win or a loss situation, whereas higher arousal was present in normal controls when imagining a win situation, in comparison to imagining losing (Sharpe, 2004). Another study reported higher psychophysiological reactions in pathological gamblers who imagined winning when watching a poker machine video compared to high and low frequency gambling normal control groups (Sharpe et al., 1995). No studies were found studying the separate effects of actual experienced wins and losses in a controlled experimental setting. Our second research question thus was whether the PG and NC group would differ in physiological reactions after wins or losses. The study of both anticipatory somatic markers and psychophysiological reactions after wins and losses in pathological gamblers could delineate whether somatic markers and/or psychophysiological reactions after wins and losses play a role in the diminished performance reported in the IGT studies in pathological gamblers.

Finally, reward and punishment sensitivity are known as risk factors in the development of alcohol and substance dependence (Dawe et al., 2004). These personality traits can be measured by self-report scales. Several studies indicate that reward and punishment sensitivity mediate the psychophysiological response to wins and losses (Knyazev et al., 2002; DePascalis et al., 1996; Kilzieh and Cloninger, 1993). Differences in reward and punishment sensitivity have also been reported in pathological gamblers compared to normal controls (Goudriaan et al., 2005; Petry, 2001a), and these factors could thus influence psychophysiological responses when performing the IGT. Therefore, exploratory analyses focused on the role of reward and punishment sensitivity traits on psychophysiological responses in the PG and normal control (NC) group. Fowles' psychophysiological application of Gray's reward and punishment sensitivity personality

theory (Fowles, 1980; Gray, 1987) refers to the influences of reward (influencing the behavioral approach system, BAS) and punishment (influencing the behavioral inhibition system, BIS) on general arousal, approach and avoidance behavior (Gray, 1987). Fowles' model states that HR variability is sensitive for cues for reward, influencing the sympathetic autonomic nervous system (Sturgis and Gramling, 1998; Fowles et al., 1982), whereas skin conductance level (SCL) variability is influenced by cues that signal aversive consequences, reflecting activity of the parasympathetic autonomic nervous system (Finn et al., 1994; Fowles, 1988, 1980). Our third research question thus examined whether reward and punishment sensitivity accounted for within group variance often encountered within clinical samples, and whether differences in these traits could also explain differences in psychophysiological responses between the PG and the NC group.

## 2. Methods

### 2.1. Recruitment and screening

The PG group ( $n = 46$ ) was recruited from outpatients of an addiction treatment center. PG diagnoses were made according to DSM-IV PG criteria, using the Dutch version of section T of the DSM-IV Diagnostic Interview Schedule (DIS; Robins et al., 1998). The Dutch version of the South Oaks Gambling Screen (SOGS, Lesieur and Blume, 1987) was administered to obtain a sensitive measure of gambling severity (Strong et al., 2003). The NC group ( $n = 47$ ) was recruited through local newspaper advertisements and from hospital staff. Telephone screening excluded NC subjects who had been treated for a mental disorder in the past 3 years. Exclusion criteria for the PG and the NC groups were: (a) co-morbid lifetime alcohol or substance abuse or dependence, diagnosed with the Dutch version of the Clinical International Diagnostic Inventory (CIDI, World Health Organisation, 1997); (b) a history of major psychiatric disorders such as psychotic or manic episodes, schizophrenia or hospitalization for psychiatric disorders; (c) current treatment for mental disorders other than PG; (d) physical conditions known to influence cognition or motor performance (e.g., multiple sclerosis, rheumatic disease); (e) the use of psychotropic medication which could not be discontinued; (f) a positive urine screen for alcohol, cannabis, or benzodiazepines.

The estimated IQ was based on two subtests of the Wechsler Adult Intelligence Scale (Vocabulary and Block Design), which correlate in the 90s with the full scale IQ (Groth-Marnat, 1997). A minimum estimated IQ of 80 was used as an inclusion criterion. The study design was approved by the Amsterdam Medical Center Local Ethical Committee and written informed consent was given by all participants before testing.

### 2.2. Iowa Gambling Task (IGT)

The IGT was used as described in Bechara et al. (1999). Amounts in dollars were replaced by amounts in euros, divided by two. Four decks of cards were presented on the computer screen. The goal of this task was to discover which of the four decks were advantageous in the long run, and to learn to select cards from the two advantageous decks instead of choosing from the two more risky, long-term disadvantageous decks. Unknown to the subject, two decks rendered high rewards, but also resulted in high net losses; these decks were disadvantageous in the long run. The other two advantageous decks gave lower rewards, but also gave lower net losses, and resulted in a net gain in the long run. Frequency of losses differed between the decks. Within the two advantageous decks one deck rendered more frequent lower losses and one deck rendered less frequent higher losses, but both of the advantageous decks resulted in the same net gain in the long run. Likewise, within the two disadvantageous decks one deck rendered more frequent lower losses, and one deck rendered less frequent higher losses. Participants were instructed to select a deck by pressing a button on a response box with four buttons, corresponding to the four decks. One

hundred card selections were made. After the response, the feedback screen was displayed for 3200 ms. Following this, a 4500–5000 ms inter-trial interval was introduced, in which no card selections could be made, in order to allow for a natural recovery of SCRs. For motivational purposes, participants were told before the start of the task that they would receive 1% of their winnings on the IGT. The dependent measure for general performance on the IGT was the number of cards picked from the advantageous decks during the five consecutive stages of the task. Each stage corresponded to 20-card selections. Reaction time data are presented in Goudriaan et al. (2005).

### 2.3. Self-report measures

**2.3.1. Reward and punishment sensitivity.** The Dutch version of the BIS/BAS self-report scale was administered (Carver and White, 1994; Putman et al., 2004). This scale measures affective responses to impending rewards (BAS) or punishments (BIS) and contains 20 items with a four-point Likert scale ranging from “strongly agree” to “strongly disagree”. The BAS items are divided in three subcategories: BAS drive, BAS reward sensitivity, and BAS fun seeking. The BIS subscale has no subcategories and measures punishment sensitivity. The BIS subscale (seven items) and BAS reward sensitivity subscale (five items) were used in this study, because our primary research goal was to measure the influence of reward and punishment sensitivity on psychophysiological responses. Adequate reliability for the BIS punishment sensitivity subscale (Cronbach's  $\alpha = 0.71$ ) and for the BAS reward sensitivity subscale (Cronbach's  $\alpha = 0.74$ ) was established in this study.

**2.3.2. Arousal self-report measure.** After performance of the IGT, participants indicated on a visual analogue scale how arousing the task was to them, by marking a 10 cm line, at their point of preference. To the left of the line “not at all” was printed, to the right of the line “very much” was printed.

### 2.4. Psychophysiological responses

The Vrije Universiteit Ambulatory Monitoring System-36 was used to measure the electrocardiogram (ECG) and SCL (Klaver et al., 1994). The ECG was measured via two active electrodes and one ground electrode. Ag/AgCl electrodes were used (10 mm, Ultratrace). One active electrode was placed at the jugular notch of the sternum, between the collar bones. The other active electrode was placed below the left breast, 4 cm beneath the nipple. The ground electrode was placed at the right side of the chest, between the lower two ribs. The ECG signal was led into a differential amplifier, with an input impedance higher than 1 M $\Omega$ . The amplified ECG was passed through a band pass filter at 17 Hz, and the filtered signal was used for R-peak triggering. At each R-peak, a millisecond counter was read and reset, yielding inter beat interval (IBI) series.

Preparatory HR responses were calculated by subtracting the IBI during the response (IBI 1) from the IBI before the response (IBI 0). HR responsiveness to wins and losses was calculated by subtracting the third IBI after the response (IBI 3) from the IBI during the response (IBI 1). Thus, positive values for IBI 0 minus IBI 1, or IBI 1 minus IBI 3, indicate heart rate decreases, and negative values indicate HR increases.

SCL was recorded through two 1 cm<sup>2</sup> Ag/AgCl electrodes attached with Velcro straps to the medial phalanx surfaces of the middle and index fingers of the non-dominant hand. A 0.5 V constant voltage procedure was used with a sample rate of 100 ms. Electrolyte gel (0.05 M NaCl) was applied to the two electrodes (Fowles et al., 1981). Anticipatory and reactive SCRs were calculated as the largest change in SCL during a 4000 ms window before or after a selection was made. A 4500–5000 ms stabilization period for SCRs was incorporated in each trial. Every response the participant made was synchronized with the sampling computer to the nearest millisecond.

### 2.5. Statistics

ANCOVAs were applied, with age as a covariate, because age is known to influence psychophysiological measures (Boutcher and Stocker, 1996; Garwood et al., 1982). Within both the advantageous and the disadvantageous decks, one deck resulted in frequent low losses and one deck resulted in infrequent

high losses. Because frequency of punishment could influence the groups differently, a preliminary analysis was performed to discover whether the groups differed on the number of cards they selected from decks with high and low punishment frequency. No significant effects of frequency of punishment, or interactions with other factors were found. Therefore, frequency of punishment was excluded from all subsequent analyses. The advantageous decks (A and B), and disadvantageous decks (C and D) were combined for all subsequent analyses, thus resulting in Group (NC and PG)  $\times$  Choice (advantageous and disadvantageous) ANCOVAs for the anticipatory psychophysiological analyses, and in Group  $\times$  Choice  $\times$  Gain (win and loss) ANCOVAs for the psychophysiological response after wins/losses analyses. The magnitude of wins and losses differed between the advantageous and disadvantageous decks, and the groups differed on the number of cards picked from the advantageous and disadvantageous decks. Therefore, choice (advantageous or disadvantageous) was also included as a within subject factor in all the analyses for responses after wins and losses. All post-hoc pair-wise comparisons were Bonferroni corrected. Missing data due to technical difficulties resulted in a smaller  $n$  for the HR and SCR analyses. Furthermore, data were excluded from the analyses when deviating more than three times the interquartile range from the 25th or 75th percentile. Data were also excluded due to artifacts in the SCL signal caused by movements. The number of excluded or missing data ranged from 0 to 5 in each group, resulting in 1–6 missing cases (6.5% of the total  $N$ ) for each analysis.

The exploratory BIS/BAS analyses were performed with three BIS/BAS subgroups, scoring either: (1) low on BIS and low on BAS reward sensitivity; (2) low on BIS and high on BAS reward sensitivity; or (3) high on BIS and high on BAS reward sensitivity. As only six participants were categorized as high on BIS and low on BAS, these participants were excluded from the BIS/BAS analyses. Participants were assigned to one of the BIS/BAS groups based on whether they scored higher or lower than the overall median scores. The ANCOVAs thus added one extra between factor (three levels) than the ANCOVAs mentioned in the paragraph above.

### 3. Results

Table 1 indicates that no differences existed between the PG and the NC group on demographical data, such as age (ANOVA), sex ( $\chi^2$  analysis), baseline HR and SCL, or estimated IQ (ANOVAs). The PG group had higher BIS punishment sensitivity scores than the NC group,  $F(1, 90) = 10.0, p < 0.01, \eta^2 = 0.10$ , and higher BAS reward sensitivity scores than the NC group,  $F(1, 90) = 13.4, p < 0.001, \eta^2 = 0.13$ . However, a  $\chi^2$

analysis indicated no differences in the overall number of participants from the PG group and the NC group in the BIS/BAS subgroup classification. This indicates that roughly the same number of participants from the PG and the NC group could be categorized in one of four BIS/BAS subgroups, despite the small differences in mean BIS/BAS scores between the PG and the NC group. No group differences were present for self-reported arousal (ANOVA).

#### 3.1. Behavioral performance on the IGT

A repeated measures ANCOVA was performed, with group as between subjects factor, stage (5 blocks of 20 trials) as a within subjects factor, and the number of cards picked from the advantageous decks as the dependent measure. This analysis revealed an effect for stage,  $F(4, 85) = 9.02, p < 0.001, \eta^2 = 0.29$ , and a group effect,  $F(1, 88) = 13.05, p < 0.01, \eta^2 = 0.12$ . The effect for group was qualified by a linear group by stage interaction,  $F(1, 88) = 13.84, p < 0.001, \eta^2 = 0.13$ . As is depicted in Fig. 1, the NC group learned to pick more cards from the advantageous decks during the consecutive stages of the task, whereas the PG group did not show an increase in the number of cards picked from the advantageous decks.

#### 3.2. Anticipatory psychophysiological responses

**3.2.1. Anticipatory HR variability.** An ANCOVA with group as a between subject factor, choice (advantageous or disadvantageous) as a within subject factor, and HR variability (IBI 0 minus IBI 1) as the dependent variable indicated no overall effect of choice difference. Yet, an interaction between group and choice was present,  $F(1, 91) = 5.80, p < 0.05, \eta^2 = 0.06$ , indicating that the PG group and NC group differed in anticipatory HR decrease before choosing from the advantageous and disadvantageous decks. As is depicted in Fig. 2, the NC group showed a larger HR decrease before a selection from the disadvantageous decks compared to a selection from the advantageous

Table 1  
Demographic, psychophysiological and self-report data of the pathological gambling group and the normal control group

	Pathological gamblers	Normal controls	Statistical tests
Number of participants	46	47	
Male/female $N$ (% male)	39/7 (85% Male)	34/13 (72% Male)	ns
Age (S.D.)	37.8 (9.1)	35.9 (11.5)	ns
Estimated Full Scale IQ <sup>a</sup> (S.D.)	117.7 (13.5)	121.6 (14.8)	ns
Mean SOGS score (S.D.)	14.4 (6.1)	–	–
Mean heart rate during IGT in bpm (S.D.)	70.7 (9.7)	69.5 (9.2)	ns
Mean skin conductance in $\mu$ S (S.D.)	17.4 (8.0)	17.0 (7.1)	ns
Low BIS Low BAS $N$ (%)	10 (21.7%)	20 (42.6%)	ns
Low BIS High BAS $N$ (%)	19 (41.3%)	13 (27.7%)	ns
High BIS Low BAS $N$ (%)	2 (4.3%)	4 (8.5%)	ns
High BIS High BAS $N$ (%)	15 (32.6%)	10 (21.3%)	ns
Mean BIS punishment sensitivity score (S.D.)	18.4 (2.8)	16.3 (3.4)	$F(1, 90) = 10.0, p < 0.01$
Mean BAS reward sensitivity score (S.D.)	17.8 (2.0)	16.0 (2.6)	$F(1, 90) = 13.4, p < 0.001$
Self-reported arousal (0–10)	5.03 (2.94)	5.10 (2.45)	ns

BAS = behavioral activation system; BIS = behavioral inhibition system; bpm = beats per minute; IGT = Iowa Gambling Task; IQ = intelligence quotient; SD = standard deviation; SOGS = South Oaks Gambling Screen.

<sup>a</sup> The estimated IQ was based on two subtests of the Wechsler Adult Intelligence Scale, see text.

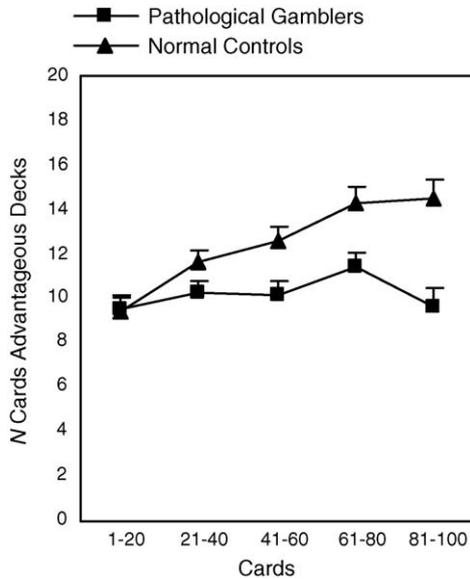


Fig. 1. Iowa Gambling Task performance of pathological gamblers and normal controls during the five consecutive stages of the task. Error bars are the standard errors of the means.

decks, whereas this effect was in the opposite direction in the PG group.

3.2.2. *Anticipatory SCRs.* An ANCOVA, with group as a between subject factor, choice (advantageous or disadvantageous) as a within subject factor, and SCR as the dependent variable, indicated no overall effect of choice. However, an interaction between group and choice was present,  $F(1, 91) = 4.75, p < 0.05, \eta^2 = 0.05$ , indicating that the groups differed in anticipatory SCRs for the advantageous and disadvantageous decks. As is depicted in Fig. 3, stronger anticipatory SCRs for the NC group were found when anticipating a selection from the disadvantageous decks, whereas for the PG group no differences were present in anticipatory SCRs between the advantageous and disadvantageous decks.

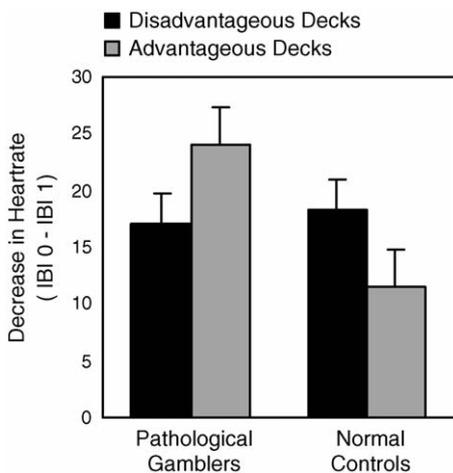


Fig. 2. Heart rate deceleration in beats per minute before choosing from advantageous or disadvantageous decks in pathological gamblers and normal controls. Error bars are the standard errors of the means.

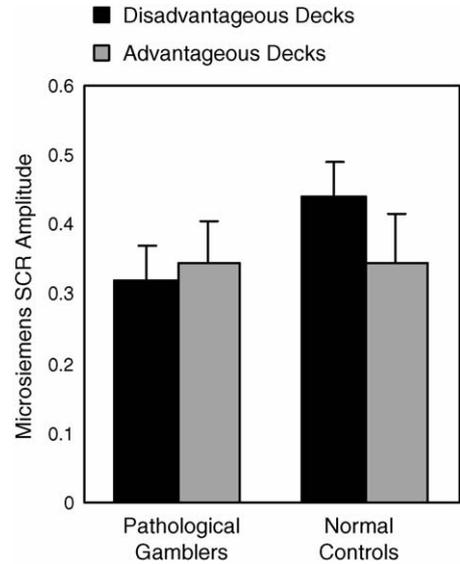


Fig. 3. Skin conductance response in pathological gamblers and normal controls before choosing from disadvantageous or advantageous decks on the Iowa Gambling Task. Error bars are the standard errors of the means.

3.3. Responses after wins and losses

3.3.1. *HR reactivity after wins and losses.* An ANCOVA with group as a between subjects factor, gain (win or loss) and choice (advantageous or disadvantageous) as within subjects factors, and IBI 1 minus IBI 3 as the dependent variable, was performed. An effect of gain was found,  $F(1, 82) = 38.24, p < 0.001, \eta^2 = 0.30$ , indicating that HR increased after wins, and decreased after losses. No effect of choice was present. An effect of group was present,  $F(1, 82) = 3.86, p < 0.05, \eta^2 = 0.05$ . The group effect was qualified by a group by gain interaction,  $F(1, 82) = 6.79, p < 0.05, \eta^2 = 0.07$ . Post-hoc pairwise analyses indicated a difference in HR reactivity after wins between the PG and the NC group ( $p < 0.05$ ), whereas no differences in HR reactivity were present between the groups after experiencing losses ( $p = 0.79$ ). As is depicted in Fig. 4, a slight HR decrease (positive value) after wins was present in the PG group, but a HR increase (negative value) was present in the NC group. However, HR in both the PG and the NC group decreased after losses (positive values).

3.3.2. *SCRs after wins and losses.* An ANCOVA was performed with group as a between subjects factor, gain (win or loss) and choice (advantageous or disadvantageous) as within subjects factors, and the largest difference in SCL during the 4000 ms interval after a card selection as the dependent variable. No group effects or interactions between group and factors were present, indicating that SCRs did not differ between the PG and the NC group after experiencing wins or losses.

3.4. Exploratory BIS/BAS analyses

BIS/BAS was inserted as a between subjects factor in all ANCOVAs. BIS/BAS influenced behavioral performance on the IGT,  $F(2, 80) = 5.37, p < 0.01, \eta^2 = 0.10$ . Post-hoc pair-wise comparisons indicated that the low BIS low BAS group per-

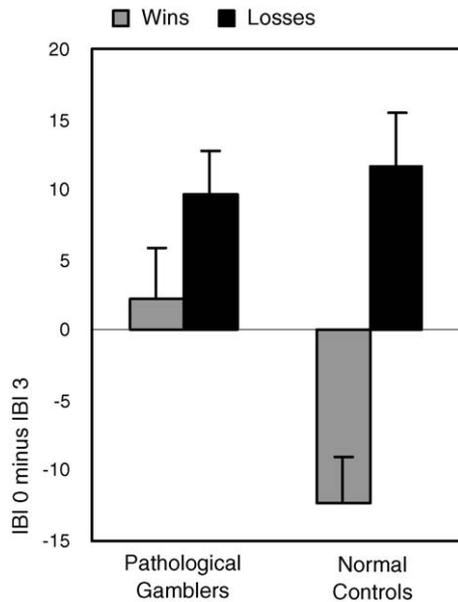


Fig. 4. Heart rate response in beats per minute after losses or rewards from the disadvantageous or advantageous decks on the Iowa Gambling Task. Negative values indicate HR acceleration, positive values indicate HR deceleration. Error bars are the standard errors of the means.

formed better than both the low BIS high BAS group ( $p < 0.05$ ) and the high BIS high BAS group ( $p < 0.001$ ). No group by BIS/BAS interaction was present, indicating that pathological gamblers and normal controls were influenced by BIS/BAS in a similar way.

For the preparatory HR reactions, an interaction was present between group and BIS/BAS,  $F(2, 85) = 6.20, p < 0.01, \eta^2 = 0.15$ , indicating that the effects of BIS/BAS on prepara-

tory HR differed between the PG and the NC group. Post-hoc pair-wise comparisons indicated an interaction between group and BIS/BAS for the low BIS/low BAS, compared to the low BIS/high BAS group,  $F(1, 49) = 16.78, p < 0.001, \eta^2 = 0.26$ . As can be seen in Fig. 5a, the low BIS/low BAS NC group showed larger differences in anticipatory HR between the advantageous and disadvantageous decks compared to the low BIS/low BAS PG group. Fig. 5b shows that in the low BIS/high BAS subgroups this effect was almost absent, and the difference in anticipatory HR between the PG and the NC groups before choosing from advantageous in comparison to disadvantageous decks was smaller.

No BIS/BAS group differences were present in preparatory SCRs, indicating that BIS/BAS effects did not have a significant influence on preparatory SCRs. Furthermore no differential effects of BIS/BAS on the PG group and the NC group were present.

No BIS/BAS effects or interactions between BIS/BAS and group were present for the psychophysiological responses after wins and losses, indicating that the HR responses and SCRs after wins and losses in the PG and the NC group were not differentially affected by BIS/BAS.

#### 4. Discussion

This study was the first to investigate differences in psychophysiological responses between pathological gamblers and normal controls when anticipating advantageous and disadvantageous choices, and when experiencing separate wins and losses. The findings of disadvantageous decision making on the IGT, in combination with a pattern of both abnormal anticipatory

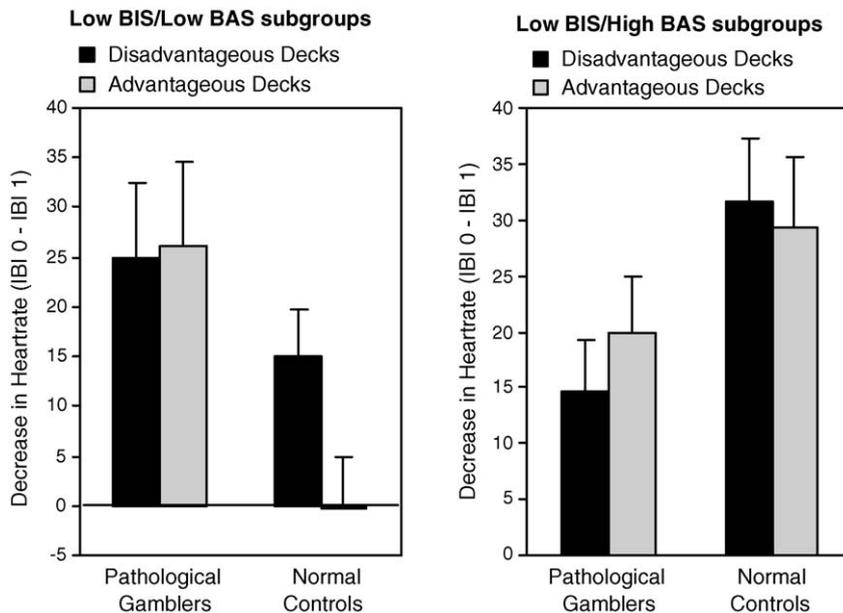


Fig. 5. (a) (left) Anticipatory heart rate responses on the Iowa Gambling Task when choosing from disadvantageous or advantageous decks for the low BIS/low BAS subgroups in the pathological gambling and the normal control group. (b) (right) Anticipatory heart rate responses on the Iowa Gambling Task when choosing from disadvantageous or advantageous decks for the low BIS/high BAS subgroups in the pathological gambling and the normal control group. Error bars are the standard errors of the means.

SCR and HR reactivity in pathological gamblers, are consistent with studies showing deficient behavioral decision making in substance abusing and dependent individuals (Bolla et al., 2003; Ernst et al., 2003; Bechara et al., 2001) and a study indicating lower somatic markers in substance dependent individuals (Bechara and Damasio, 2002a). The results indicate that in PG, the unfavorable behavioral performance on the IGT is related to deficient development of somatic markers.

The diminished risk sensitivity in PG may interfere with adequate decision making in real life. For instance, when starting to gamble, diminished psychophysiological responses to risky choices could result in a failure to inhibit the action tendency to take a bet, and gambling behavior more in general, and thus may have a role in the etiology and continuity of this disorder. More in general, pathological gamblers may have difficulty assessing risky situations, and evaluating the possible future negative consequences of their behavior.

The HR responsiveness of pathological gamblers after rewards was lower compared to the HR responsiveness of the NC group. Thus, the pathological gamblers may have needed larger wins to establish the same level of psychophysiological arousal as normal controls. Pre-existing low reward responsiveness due to low dopamine levels in the reward pathways of the brain has been proposed as a mediating factor in the development of substance dependence (Goldstein and Volkow, 2002) and PG (Blum et al., 1996, 2000). Our results do not answer the etiological question whether lower reward responsiveness existed before developing PG. However, the lower responsiveness in HR after rewards in the PG group fits in with the notion of lower reward responsiveness in PG and substance dependence in general. The finding of lower HR responsiveness to wins in PG converges with a recent neuroimaging study that found reduced activation of the mesolimbic reward system in pathological gamblers when experiencing monetary rewards (Reuter et al., 2005).

The orbitofrontal and dorsolateral prefrontal cortical circuits are activated during performance of the IGT and other decision making tasks (Bolla et al., 2003; Fukui et al., 2005). In addition, anticipatory autonomic activity, such as SCRs, have been shown to modulate the activity of the thalamus, anterior cingulate cortex, and the ventromedial as well as the dorsolateral prefrontal cortex (Critchley et al., 2001; Nagai et al., 2004). The findings of abnormal anticipatory HR and SCR responses in PG are therefore indirect evidence of abnormal functioning in these brain areas. The first neuroimaging studies in PG also indicate abnormal functioning of the ventromedial prefrontal cortex, basal ganglia, and thalamus (Potenza et al., 2003).

Despite the difference in somatic markers and HR responsiveness to rewards between the PG and the NC group, the subjectively reported arousal in the PG and the NC group did not differ from each other. This indicates that self-report data on arousal did not have a strong relation with psychophysiological reactivity in our study, a finding which has been reported previously (Coventry and Constable, 1999; Leary and Dickerson, 1985). A possible explanation for this discrepancy is that the subjective experience of arousal may relate to different psychophysiological

processes, such as overall HR or SCL. The similar HR and SCL found in the PG and NC groups could therefore account for the lack of differences between reported subjective arousal in PG and NC.

In our study, only anticipatory responses differed between advantageous and disadvantageous decks for the NC and the PG group. This suggests that only anticipatory reactions were related to IGT performance in both the PG and the NC group. Yet, whether both psychophysiological reactions before and reactions after deciding influence decision making on the IGT is open to debate. Some studies on the IGT reported differences between groups in anticipatory reactions on the IGT, but not in reactions after experiencing wins or losses (Bechara and Damasio, 2002a; Crone et al., 2004). However, a study on persons with Huntington's disease did imply that physiological responses after making decisions on the IGT can differentiate between good and bad performers (Campbell et al., 2004). Another study in healthy participants also reported a relationship between psychophysiological responses after experiencing wins and losses and performance on the IGT, but no relation between psychophysiological anticipatory responses and performance on the IGT (Suzuki et al., 2003). These studies thus suggest that psychophysiological reactions at different time points may influence performance on the IGT.

The moderating effect of reward and punishment sensitivity as measured by self-report on psychophysiological reactions in the PG group and the NC group was limited to anticipatory HR responses. Higher anticipatory HR reactivity in anticipation of disadvantageous decks compared to advantageous decks was present in the low BIS/ low BAS NC group, but not in the low BIS/low BAS PG group. For the low BIS/high BAS subgroups, this effect was absent. Yet, for the larger part no confounding effects of reward and punishment sensitivity on psychophysiological differences between the PG and the NC group were present. Although no differences between the NC and the PG groups were present when dividing the groups in high and low BIS/BAS scores, it should be noted that the PG group scored higher on both BIS and BAS measures. This implicates a somewhat higher sensitivity for immediate positive and negative consequences in the PG group. Furthermore, subgroups with lower punishment and reward sensitivity scores performed better on the IGT than the subgroup with high reward sensitivity, or the subgroup with both high punishment sensitivity and high reward sensitivity. Higher BIS and BAS scores are associated with a worse performance on the IGT. Studies in normal populations, investigating the effects of reward and punishment sensitivity on behavioral responses after rewards and losses are therefore warranted.

Some limitations should be noted. The PG group predominantly consisted of males, because PG more frequently occurs in males, and thus more male pathological gamblers were present and recruited in the treatment center. The small number of female pathological gamblers in our study restricted the possibility of separate analyses on female and male pathological gamblers. Research shows that physiological reactions to stressors differ between men and women (Burns, 1995; Wallbott and Scherer, 1991). We therefore recommend including more

women in future studies in addictive behaviors, allowing for the comparison of psychophysiological reactions in both sexes. Secondly, participants could win a proportion of their wins on the IGT, but they could not lose money when their net result at the end of the task was lower than the amount of money they started with. Real losses of private money were not deemed ethical in a study with pathological gamblers often in strained financial situations. This may have compromised the ecological validity of the encountered losses. Pathological gamblers and normal controls may have differentially evaluated the fictitious losses. The absence of a difference in psychophysiological responses after losses between the PG group and the NC group could therefore have resulted from the fictional character of losses in the IGT or from group differences in evaluation of the importance of these losses. Finally, our PG group excluded individuals with co-morbid alcohol or substance abuse or dependence. In this way, the effects of PG could be studied without the confounding effects of high alcohol or drug use. However, this also limits generalization of the results to a general PG population. There is evidence that alcohol and substance dependence are also related to diminished development of somatic markers (Bechara et al., 2001), and therefore it would be expected that in pathological gamblers with co-morbid alcohol or substance dependence, deficiencies in the development of somatic markers would be even higher.

In conclusion, the results from this study clearly indicate that in a carefully screened group of pathological gamblers without co-morbid disorders, abnormal psychophysiological antecedents of decision making were present across two psychophysiological measures: SCRs and HR variability. Deficiencies in developing psychophysiological reactions to behavior with negative consequences could play a role in the development and/or continuation of PG. The current study also showed that pathological gamblers exhibit a diminished HR response after rewards, compared to normal controls, implying a diminished reward sensitivity in PG. Longitudinal studies could shed light on the causal relation between abnormal psychophysiological reactions and PG, for instance by including a sub-clinical PG group, and a group of social gamblers who do not exhibit gambling problems, and assess whether they develop PG or alcohol or substance dependence over time. Reward and punishment sensitivity did influence behavioral performance on the IGT. For the larger part, the BIS/BAS taxonomy was not related to differential psychophysiological responses in the PG group and the NC group.

The findings of this study could have relevance for clinical practice, such as in the application of biofeedback training (Naisberg, 2002; Trudeau, 2000), because psychophysiological reactions to imagination of gambling situations in PG can be influenced by cognitive behavioral treatment (Freidenberg et al., 2002). However, whether somatic markers and psychophysiological responses to positive and negative consequences can be 'trained' or whether these phenomena are fundamentally non-remediable remains an open question. At the moment, an applicable intervention could therefore involve psycho-education aimed at abstinence for pathological gamblers with lowered psychophysiological risk sensitivity.

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## References

- American Psychiatric Association, 1994. Diagnostic and Statistical Manual of Mental Disorders, 4th ed. American Psychiatric Press, Washington, DC.
- Bechara, A., Damasio, H., 2002a. Decision-making and addiction (part I): impaired activation of somatic states in substance dependent individuals when pondering decisions with negative future consequences. *Neuropsychologia* 40, 1675–1689.
- Bechara, A., Damasio, A.R., Damasio, H., Anderson, S.W., 1994. Insensitivity to future consequences following damage to human prefrontal cortex. *Cognition* 50, 7–15.
- Bechara, A., Damasio, H., Damasio, A.R., Lee, G.P., 1999. Different contributions of the human amygdala and ventromedial prefrontal cortex to decision-making. *J. Neurosci.* 19, 5473–5481.
- Bechara, A., Tranel, D., Damasio, H., 2000. Characterization of the decision-making deficit of patients with ventromedial prefrontal cortex lesions. *Brain* 123 (Pt 11), 2189–2202.
- Bechara, A., Dolan, S., Denburg, N., Hindes, A., Anderson, S.W., Nathan, P.E., 2001. Decision-making deficits, linked to a dysfunctional ventromedial prefrontal cortex, revealed in alcohol and stimulant abusers. *Neuropsychologia* 39, 376–389.
- Bechara, A., Dolan, S., Hindes, A., 2002b. Decision-making and addiction (part II): myopia for the future or hypersensitivity to reward? *Neuropsychologia* 40, 1690–1705.
- Blanco, C., Moreyra, P., Nunes, E.V., Saiz-Ruiz, J., Ibanez, A., 2001. Pathological gambling: addiction or compulsion? *Semin. Clin. Neuropsychiatry* 6, 167–176.
- Blaszczynski, A., Nower, L., 2002. A pathways model of problem and pathological gambling. *Addiction* 97, 487–499.
- Blum, K., Braverman, E.R., Holder, J.M., Lubar, J.F., Monastra, V.J., Miller, D., Lubar, J.O., Chen, T.J., Comings, D.E., 2000. Reward deficiency syndrome: a biogenetic model for the diagnosis and treatment of impulsive, addictive, and compulsive behaviors. *J. Psychoactive Drugs* 32 (Suppl.), 1–112.
- Blum, K., Sheridan, P.J., Wood, R.C., Braverman, E.R., Chen, T.J., Cull, J.G., Comings, D.E., 1996. The D2 dopamine receptor gene as a determinant of reward deficiency syndrome. *J. Royal Soc. Med.* 89, 396–400.
- Bolla, K.I., Eldredge, D.A., London, E.D., Kiehl, K.A., Mouratidis, M., Contoreggi, C., Matochik, J.A., Kurian, V., Cadet, J.L., Kimes, A.S., Funderburk, F.R., Ernst, M., 2003. Orbitofrontal cortex dysfunction in abstinent cocaine abusers performing a decision-making task. *NeuroImage* 19, 1085–1094.
- Boutcher, S.H., Stocker, D., 1996. Cardiovascular response of young and older males to mental challenge. *J. Gerontol. B Psychol. Sci. Soc. Sci.* 51, 261–267.
- Burns, J.W., 1995. Interactive effects of traits, states, and gender on cardiovascular reactivity during different situations. *J. Behav. Med.* 18, 279–303.
- Campbell, M.C., Stout, J.C., Finn, P.R., 2004. Reduced autonomic responsiveness to gambling task losses in Huntington's disease. *J. Int. Neuropsychol. Soc.* 10, 239–245.
- Carver, C.S., White, T.L., 1994. Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: the BIS/BAS scales. *J. Pers. Soc. Psychol.* 67, 319–333.
- Clark, L., Robbins, T., 2002. Decision-making deficits in drug addiction. *Trends Cognit. Sci.* 6, 361.
- Coventry, K.R., Constable, B., 1999. Physiological arousal and sensation-seeking in female fruit machine gamblers. *Addiction* 94, 425–430.
- Critchley, H.D., Mathias, C.J., Dolan, R.J., 2001. Neural activity in the human brain relating to uncertainty and arousal during anticipation. *Neuron* 29, 537–545.
- Crone, E.A., Somsen, R.J., Beek, B.V., van der Molen, M.W., 2004. Heart rate and skin conductance analysis of antecedents and consequences of decision making. *Psychophysiology* 41, 531–540.

- Damasio, A.R., 1996. The somatic marker hypothesis and the possible functions of the prefrontal cortex. *Philos. Trans. Royal Soc. Lond. B* 351, 1413–1420.
- Dawe, S., Gullo, M.J., Loxton, N.J., 2004. Reward drive and rash impulsiveness as dimensions of impulsivity: implications for substance misuse. *Addict. Behav.* 29, 1389–1405.
- DePascalis, V., Fiore, A.D., Sparita, A., 1996. Personality, event-related potential (ERP) and heart rate (HR): an investigation of Gray's theory. *Pers. Individ. Differences* 20, 733–746.
- Ernst, M., Grant, S.J., London, E.D., Contoreggi, C.S., Kimes, A.S., Spurgeon, L., 2003. Decision making in adolescents with behavior disorders and adults with substance abuse. *Am. J. Psychiatry* 160, 33–40.
- Fein, G., Di Sclafani, V., Meyerhoff, D.J., 2002. Prefrontal cortical volume reduction associated with frontal cortex function deficit in 6-week abstinent crack-cocaine dependent men. *Drug Alcohol Depend.* 68, 87–93.
- Finn, P.R., Kessler, D.N., Hussong, A.M., 1994. Risk for alcoholism and classical conditioning to signals for punishment: evidence for a weak behavioral inhibition system? *J. Abnorm. Psychol.* 103, 293–301.
- Fowles, D.C., 1980. The three arousal model: implications of Gray's two-factor learning theory for heart rate, electrodermal activity, and psychopathy. *Psychophysiology* 17, 87–104.
- Fowles, D.C., 1988. Psychophysiology and psychopathology: a motivational approach. *Psychophysiology* 25, 373–391.
- Fowles, D.C., Christie, M.J., Edelberg, R., Grings, W.W., Lykken, D.T., Venables, P.H., 1981. Committee report. Publication recommendations for electrodermal measurements. *Psychophysiology* 18, 232–239.
- Fowles, D.C., Fisher, A.E., Tranel, D.T., 1982. The heart beats to reward: the effect of monetary incentive on heart rate. *Psychophysiology* 19, 506–513.
- Freidenberg, B.M., Blanchard, E.B., Wulfert, E., Malta, L.S., 2002. Changes in physiological arousal to gambling cues among participants in motivationally enhanced cognitive-behavior therapy for pathological gambling: a preliminary study. *Appl. Psychophysiol. Biofeedback* 27, 251–260.
- Fukui, H., Murai, T., Fukuyama, H., Hayashi, T., Hanakawa, T., 2005. Functional activity related to risk anticipation during performance of the Iowa Gambling Task. *NeuroImage* 24, 253–259.
- Garwood, M., Engel, B.T., Capriotti, R., 1982. Autonomic nervous system function and aging: Response specificity. *Psychophysiology* 19, 378–385.
- Giancola, P.R., Moss, H.B., 1998. Executive cognitive functioning in alcohol use disorders. *Recent Dev. Alcohol Res.* 14, 227–251.
- Goldstein, R.Z., Volkow, N.D., 2002. Drug addiction and its underlying neurobiological basis: neuroimaging evidence for the involvement of the frontal cortex. *Am. J. Psychiatry* 159, 1642–1652.
- Goudriaan, A.E., Oosterlaan, J., de Beurs, E., van den Brink, W., 2004. Pathological gambling: a comprehensive review of biobehavioral findings. *Neurosci. Biobehav. Rev.* 28, 123–141.
- Goudriaan, A.E., Oosterlaan, J., de Beurs, E., van den Brink, W., 2005. Decision making in pathological gambling: a comparison between pathological gamblers, alcohol dependents, persons with Tourette syndrome, and normal controls. *Cognit. Brain Res.* 23, 137–151.
- Gray, J.A., 1987. *The Psychology of Fear and Stress*. Cambridge University Press, New York.
- Groth-Marnat, G., 1997. *Handbook of Psychological Assessment*, 3rd ed. John Wiley and Sons, New York.
- Kilzieh, N., Cloninger, C.R., 1993. Psychophysiological antecedents of personality. *J. Pers. Disorders* 7, 100–117.
- Klaver, C.H.A.M., de Geus, E.J.C., de Vries, J., 1994. Ambulatory monitoring system. In: Maarse, F.J. (Ed.), *Applications, Methods and Instrumentations*. Swets and Zeitlinger, Lisse, pp. 254–268.
- Knyazev, G.G., Slobodskaya, H.R., Wilson, G.D., 2002. Psychophysiological correlates of behavioural inhibition and activation. *Pers. Individ. Differences* 33, 647–660.
- Koob, G.F., Le Moal, M., 1997. Drug abuse: hedonic homeostatic dysregulation. *Science* 278, 52–58.
- Leary, K., Dickerson, M., 1985. Levels of arousal in high- and low-frequency gamblers. *Behav. Res. Ther.* 23, 635–640.
- Lesieur, H.R., Blume, S.B., 1987. The South Oaks Gambling Screen (SOGS): a new instrument for the identification of pathological gamblers. *Am. J. Psychiatry* 144, 1184–1188.
- Maia, T.V., McClelland, J.L., 2004. A reexamination of the evidence for the somatic marker hypothesis: what participants really know in the Iowa gambling task. *Proc. Natl. Acad. Sci. USA* 101, 16075–16080.
- Manes, F., Sahakian, B., Clark, L., Rogers, R., Antoun, N., Aitken, M., Robbins, T., 2002. Decision-making processes following damage to the prefrontal cortex. *Brain* 125, 624–639.
- Marks, I., 1990. Behavioural (non-chemical) addictions. *Br. J. Addict.* 85, 1389–1394.
- Miguel-Hidalgo, J.J., Rajkowska, G., 2003. Comparison of prefrontal cell pathology between depression and alcohol dependence. *J. Psychiat. Res.* 37, 411–420.
- Nagai, Y., Critchley, H.D., Featherstone, E., Trimble, M.R., Dolan, R.J., 2004. Activity in ventromedial prefrontal cortex covaries with sympathetic skin conductance level: A physiological account of a “default mode” of brain function. *NeuroImage* 22, 243–251.
- Naisberg, Y., 2002. Long-term biophysical synchronization for prevention of addiction spectrum formation in high risk children and adolescents: Theory and practice. *Med. Hypotheses* 58, 433–438.
- Pau, C.W., Lee, T.M., Chan, S.F., 2002. The impact of heroin on frontal executive functions. *Arch. Clin. Neuropsychol.* 17, 663–670.
- Paulus, M.P., Hozack, N.E., Zauscher, B.E., Frank, L., Brown, G.G., Braff, D.L., Schuckit, M.A., 2002. Behavioral and functional neuroimaging evidence for prefrontal dysfunction in methamphetamine-dependent subjects. *Neuropsychopharmacology* 26, 53–63.
- Petry, N.M., 2001a. Pathological gamblers, with and without substance use disorders, discount delayed rewards at high rates. *J. Abnorm. Psychol.* 110, 482–487.
- Petry, N.M., 2001b. Substance abuse, pathological gambling, and impulsiveness. *Drug Alcohol Depend.* 63, 29–38.
- Potenza, M.N., Leung, H.C., Blumberg, H.P., Peterson, B.S., Fulbright, R.K., Lacadie, C.M., Skudlarski, P., Gore, J.C., 2003. An FMRI Stroop task study of ventromedial prefrontal cortical function in pathological gamblers. *Am. J. Psychiat.* 160, 1990–1994.
- Putman, P., Hermans, E., van Honk, J., 2004. Emotional stroop performance for masked angry faces: it's BAS, not BIS. *Emotion* 4, 305–311.
- Reuter, J., Raedler, T., Rose, M., Hand, I., Glascher, J., Buchel, C., 2005. Pathological gambling is linked to reduced activation of the mesolimbic reward system. *Nature Neurosci.* 8, 147–148.
- Robins, L., Cottler, L., Bucholz, K., Compton, W., 1998. *Diagnostic Interview Schedule for DSM-IV (DIS-IV-Revision 11 Sep 1998)*. Washington University, School of Medicine, Department of Psychiatry, St. Louis.
- Sharpe, L., 2002. A reformulated cognitive-behavioral model of problem gambling. A biopsychosocial perspective. *Clin. Psychol. Rev.* 22, 1–25.
- Sharpe, L., 2004. Patterns of autonomic arousal in imaginal situations of winning and losing in problem gambling. *J. Gambling Stud.* 20, 95–104.
- Sharpe, L., Tarrar, N., Schotte, D., Spence, S.H., 1995. The role of autonomic arousal in problem gambling. *Addiction* 90, 1529–1540.
- Strong, D.R., Breen, R.B., Lesieur, H.R., Lejuez, C.W., 2003. Using the Rasch model to evaluate the South Oaks Gambling Screen for use with nonpathological gamblers. *Addict. Behav.* 28, 1465–1472.
- Sturgis, E.T., Gramling, S.E., 1998. Psychophysiological assessment. In: Bellack, A.S., Hersen, M. (Eds.), *Behavioral Assessment: A Practical Handbook*, 4th ed. Allyn and Bacon, Needham Heights, MA, pp. 126–157.
- Suzuki, A., Hirota, A., Takasawa, N., Shigemasa, K., 2003. Application of the somatic marker hypothesis to individual differences in decision making. *Biol. Psychol.* 65, 81–88.
- Trudeau, D.L., 2000. The treatment of addictive disorders by brain wave biofeedback: a review and suggestions for future research. *Clin. Electroencephalogr.* 31, 13–22.
- Wallbott, H.G., Scherer, K.R., 1991. Stress specificities: differential effects of coping style, gender, and type of stressor on autonomic arousal, facial expression, and subjective feeling. *J. Pers. Soc. Psychol.* 61, 147–156.
- World Health Organisation, 1997. *Composite International Diagnostic Interview—Version 2.1*. World Health Organization, Geneva.